Final

Human Health Risk Assessment for the Coeur d'Alene Basin Extending from Harrison to Mullan on the Coeur d'Alene River and Tributaries
Remedial Investigation/Feasibility Study

Prepared for
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TABLE OF CONTENTS

ABBREVIATIONS AND ACRONYMS xx

EXECUTIVE SUMMARY ES-1

1.0 INTRODUCTION 1-1
1.1 SITE LOCATION AND BACKGROUND 1-1
1.1.1 Community and Industrial Development 1-1
1.1.2 Public Health Concerns 1-3
1.2 STUDY POPULATIONS AND STUDY AREA 1-6
1.3 PURPOSE AND OBJECTIVES 1-6
1.4 SCOPE AND LIMITATIONS 1-7
1.5 REPORT STRUCTURE 1-7

2.0 DATA EVALUATION 2-1
2.1 BASIN GEOGRAPHICAL AREAS 2-1
2.1.1 CSM Unit 1 2-2
2.1.2 CSM Unit 2 2-2
2.1.3 CSM Unit 3 2-3
2.1.4 CSM Unit 4 2-3
2.1.5 CSM Unit 5 2-3
2.2 SELECTION OF DATA USED IN HHRA 2-4
2.2.1 Environmental Data 2-4
2.2.2 Biological Data 2-13
2.3 DATA QUALITY 2-14
2.3.1 Data Quality Objectives/Data Quality Assessment 2-14
2.3.2 Data Usability 2-15
2.3.3 Evaluation of Sample Quantitation Limits 2-16
2.4 SELECTION OF CHEMICALS OF POTENTIAL CONCERN 2-16
2.4.1 COPC Screening Process 2-17
2.4.2 Comparison to Background Concentrations 2-18
2.4.3 Essential Nutrients 2-19
2.4.4 Frequency of Detection 2-20
2.4.5 Comparison to Screening Values and Risk-Based Preliminary Remediation Goals 2-20
2.5 RESULTS OF COPC SCREENING 2-21
2.5.1 Soil/Sediment 2-21
2.5.2 Tap Water 2-23
2.5.3 Surface Water 2-24
2.5.4 Groundwater 2-25
2.5.5 House Dust 2-26

2.5.6 Air 2-26
2.5.7 Fish 2-27
2.5.8 Homegrown Vegetables 2-27
2.5.9 Water Potatoes 2-27
2.6 SUMMARY OF CHEMICALS OF POTENTIAL CONCERN 2-27

3.0 EXPOSURE ASSESSMENT 3-1
3.1 CHARACTERIZATION OF EXPOSURE SETTING 3-1
3.1.1 Physical Setting 3-1
3.1.2 Coeur d'Alene Basin Demographics 3-3
3.1.3 Human Health Exposure Areas 3-14
3.1.4 Characterization of Potentially Exposed Populations 3-15
3.1.5 Populations of Potential Concern 3-19
3.2 IDENTIFICATION OF EXPOSURE PATHWAYS 3-22
3.2.1 Sources, Fate and Transport, and Affected Media 3-23
3.2.2 Potentially Complete Pathways Excluded From Quantification 3-24
3.2.3 Complete Pathways Selected for Quantification 3-26
3.2.4 Potential Coeur d'Alene Tribal Pathways Selected for Quantification 3-28
3.2.5 Complete Exposure Pathways Quantified by Area 3-34
3.3 QUANTIFICATION OF EXPOSURE TO NON-LEAD CHEMICALS 3-34
3.3.1 Exposure Point Concentration 3-34
3.3.2 Estimation of Chemical Intakes 3-41
3.3.3 Exposure Factors 3-46
3.4 QUANTIFICATION OF LEAD EXPOSURE 3-56
3.4.1 Child Lead Model Overview 3-56
3.4.2 Adult Lead Model Overview 3-57
3.5 SUMMARY OF EXPOSURE ASSESSMENT 3-58

4.0 HUMAN HEALTH TOXICITY ASSESSMENT 4-1
4.1 ORAL TOXICITY CRITERIA 4-1
4.1.1 Cancer Effects 4-1
4.1.2 Noncancer Effects 4-2
4.2 DERMAL TOXICITY CRITERIA 4-2
4.3 CHEMICAL PROFILES 4-3
4.3.1 Antimony 4-3
4.3.2 Arsenic 4-4
4.3.3 Cadmium 4-7
4.3.4 Iron 4-9
4.3.5 Lead 4-11
4.3.6 Manganese 4-14
4.3.7 Mercury 4-15
4.3.8 Zinc 4-17

5.0 RISK CHARACTERIZATION FOR NON-LEAD CHEMICALS 5-1
5.1 METHODOLOGY FOR ASSESSING NONCANCER HAZARD 5-1
5.2 METHODOLOGY FOR ASSESSING CANCER RISK 5-2
5.3 RISK CHARACTERIZATION RESULTS 5-2
5.3.1 Noncancer Hazard 5-3
5.3.2 Cancer Risks 5-6
5.3.3 Hazards/Cancer Risks for Combinations of Scenarios 5-7
5.3.4 Subsistence Risks and Hazards 5-9
5.4 SUMMARY OF RISK CHARACTERIZATION 5-11
5.5 POTENTIAL PRELIMINARY REMEDIATION GOALS FOR ARSENIC . . . 5-12

6.0 CHARACTERIZATION OF LEAD HEALTH RISK 6-1
6.1 INTRODUCTION/METHODOLOGY 6-1
6.1.1 Lead Health Risk Assessment for the Resident Population 6-2
6.1.2 Lead Health Risk Assessment for the Coeur d'Alene Tribe 6-5
6.2 OBSERVED BLOOD LEAD LEVELS 6-7
6.2.1 Blood Lead Health Criteria 6-7
6.2.2 State of Idaho / Panhandle Health District Children's Blood Lead Survey Results 6-10
6.2.3 Summary Results for Follow-up Investigations of High Blood Lead Children 6-14
6.2.4 Adult Blood Lead Survey Results 6-17
6.2.5 Coeur d'Alene Tribe Blood Lead Levels. 6-17
6.3 LEAD EXPOSURE PATHWAYS 6-18
6.3.1 Dietary Lead Sources 6-18
6.3.2 Lead in Water 6-18
6.3.3 Lead in Air 6-19
6.3.4 Lead in Paint 6-19
6.3.5 Lead in Soils and Dusts 6-20
6.4 SITE-SPECIFIC BLOOD LEAD AND ENVIRONMENTAL EXPOSURE ANALYSIS 6-22
6.4.1 Correlation Analysis 6-22
6.4.2 Regression Analysis 6-23
6.4.3 Summary of Site-Specific Lead Health Analysis 6-28
6.5 BASELINE AND INCREMENTAL LEAD EXPOSURE INTAKE RATES . . 6-30
6.5.1 Exposure Routes Considered 6-30
6.5.2 Developing Baseline Intake Rates for the Resident Population 6-32
6.5.3 Developing Incremental Intake Rates for the Resident Population . . . 6-34
6.5.4 Developing Intake Rates for the Coeur d'Alene Tribe 6-40
6.6 ESTIMATED BASELINE BLOOD LEAD LEVELS 6-43
6.6.1 Childhood Baseline Blood Lead Levels 6-44
6.6.2 Resident Children's Incremental Blood Lead Levels 6-47

6.6.3 Adult Model Blood Lead Estimates 6-50
6.6.4 Native American Blood Lead Levels 6-51
6.7 RISK CHARACTERIZATION 6-52
6.7.1 Overview and Summary 6-52
6.7.2 Indices of Lead Health Risk 6-53
6.7.3 Observed Blood Lead Levels 6-53
6.7.4 Site-specific Analysis of Paired Blood Lead and Environmental Source Observations 6-54
6.7.5 Predicted Blood Lead Levels 6-54
6.7.6 Potential Lead Health Risk Reduction Strategies 6-58

7.0 UNCERTAINTIES IN RISK ASSESSMENT 7-1
7.1 FACTORS LEADING TO POSSIBLE OVERESTIMATION OF RISK 7-3
7.1.1 Data Collection and Evaluation 7-3
7.1.2 Exposure Assessment 7-5
7.1.3 Toxicity Assessment and Risk Calculations 7-7
7.2 FACTORS LEADING TO POSSIBLE UNDERESTIMATION OF RISK 7-10
7.2.1 Data Collection and Evaluation 7-10
7.2.2 Exposure Assessment 7-12
7.2.3 Toxicity Assessment and Risk Calculations 7-15
7.3 FACTORS LEADING TO POSSIBLE UNDERESTIMATION OR OVERESTIMATION OF RISK 7-18
7.3.1 Data Collection and Evaluation 7-18
7.3.2 Exposure Assessment 7-22
7.3.3 Toxicity Assessment 7-23
7.3.4 Risk Calculations 7-26
7.4 UNCERTAINTIES IN LEAD RISK ASSESSMENT 7-26
7.4.1 Uncertainty in the Use of Observed Blood Lead Levels 7-26
7.4.2 Uncertainty in Data Collection and Evaluation 7-32
7.4.3 Uncertainty in Exposure Assessments 7-39
7.4.4 Uncertainty in Blood Lead Level Modeling 7-44
7.4.5 Uncertainty Regarding Candidate Risk Reduction Activities 7-49

8.0 SUMMARY AND CONCLUSIONS 8-1
8.1 PURPOSE 8-1
8.2 STUDY AREA 8-2
8.3 RESIDENT POPULATION, LAND-USE, ECONOMY AND HOUSING 8-3
8.4 DATA USED IN THE HHRA 8-4
8.5 CHEMICALS OF POTENTIAL CONCERN 8-6
8.6 TOXICITY ASSESSMENT 8-7
8.7 EXPOSURE SUBAREAS 8-7
8.8 POPULATIONS OF POTENTIAL CONCERN 8-8

8.9 RECEPTORS, EXPOSURE PATHWAYS AND SELECTED SCENARIOS . 8-10
8.10 HUMAN HEALTH RISK CHARACTERIZATION FOR METALS OTHER THAN LEAD 8-12
8.10.1 Non-carcinogenic Risk 8-12
8.10.2 Arsenic Carcinogenic Risks 8-13
8.10.3 Non-lead RME Residential and Neighborhood Risks and Hazards . . 8-15
8.10.4 Non-lead RME Public Recreational Risks and Hazards 8-16
8.10.5 Non-lead RME Occupational Risks and Hazards (Construction Worker) 8-16
8.10.6 Non-lead RME Modern and Traditional Subsistence Exposure Scenarios (Tribal Members) 8-16
8.10.7 Risks and Hazards for Combined Non-lead Exposures 8-17
8.10.8 Potential Preliminary Remediation Goals for Arsenic 8-17
8.11 HUMAN HEALTH RISK CHARACTERIZATION FOR LEAD 8-18
8.11.1 Observed Blood Lead Levels 8-18
8.11.2 Representativeness of the Surveys 8-19
8.11.3 Follow-up of Children with High Blood Lead Levels 8-19
8.11.4 Site-specific Analysis of Paired Blood and Environmental Lead Data 8-20
8.11.5 Biokinetic Predictions of Resident Children's Blood Lead Levels . . . 8-20
8.11.6 Lead Health Risks from Exposures Outside the Residential Environment 8-22
8.11.7 Native American Blood Lead Levels 8-23
8.11.8 Lead Health Risk Reduction Strategies 8-23
8.11.9 Biokinetic Blood Lead Modeling for Residential Cleanup Levels . . . 8-24
8.11.10 Lead Health Risk Reduction for Childhood Recreational Activities . . 8-26
8.11.11 Lead Health Risk Reduction for Childhood Consumption of Local Foodstuff 8-26
8.11.12 Lead Health Risk Reduction for Adult Occupational Activities 8-26
8.11.13 Lead Health Risk Reduction for Adult Recreational Activities 8-26
8.11.14 Lead Health Risk Reduction for Adult Consumption of Local Foodstuff 8-26
8.11.15 Lead Health Risk Reduction for Native American Subsistence Activities 8-27
8.12 RESPONSE TO COMMENTS - PUBLIC REVIEW DRAFT 8-27
8.12.1 General and Specific Responses to Public Comments 8-27
8.12.2 Additional Analysis and Appendices in Responses to Public Comments 8-27
8.12.3 Clarification of USEPA Policy Regarding Human Health Risk Assessment for Lead 8-28

9.0 REFERENCES 9-1

APPENDICES

Appendix A	Risk Assessment Guidance for Superfund (RAGS), Volume 1, Part D, Tables
Appendix B	Expedited Screening Level Risk Assessment for Common Use Areas, Coeur d'Alene River Basin
Appendix C	Summary Table of Potential Receptor Groups
Appendix D	Graphs of Concentration Distributions of Mercury, Thallium, and Manganese
Appendix E	Data Set Used to Calculate Human Health Exposure Point Concentrations
Appendix F	Distribution Checks and Statistical Summaries of Data by Geographical Area and Medium
Appendix G	95 Percent Upper Confidence Limit Statistical Summary Results by Geographical Area and Medium
Appendix H	Toxicity Profiles for Chemicals of Potential Concern
Appendix I	Graphs and Statistical Analysis of House Dust Concentrations
Appendix J	Potential Dose from Consumption of Beef
Appendix K	Summary Statistics for Antimony, Arsenic, Cadmium, Iron, Manganese, and Zinc for the Fall 1999 Coeur d'Alene Basin Sampling Event
Appendix L	Summary Statistics for Antimony, Arsenic, Cadmium, Iron, Manganese, and Zinc for the Summer 1999 Coeur d'Alene Basin Sampling Event
Appendix M	QA/QC Memoranda for the Summer and Fall 1999 Coeur d'Alene Basin Sampling Events
Appendix N	Proposed Geographic Subdivisions and Archived Reanalysis Results for the Coeur d'Alene Basin Human Health Risk Assessment
Appendix O	Selected EPA Guidance Documents for Lead
Appendix P	Baseline Intakes for the EPA Default and Box Model Scenarios by Area and by Age Level
Appendix Q	Development of the IEUBK Bunker Hill Superfund Site Models and Bioavailability Estimates
Appendix R	Observed and Predicted Blood Lead Levels by Age, Area, Dust Concentration and Action Criteria
Appendix S	Heavy Metals Concentrations in Water Potatoes in the Coeur d'Alene Basin, Idaho
Appendix T	Masked Basin HHRA Lead Data
Appendix U	Cancer Rate Analysis in Shoshone County
Appendix V	IEUBK Input Parameters
Appendix W	Comments and Responses on Public Review Draft, July 2000

FIGURES

ES-1 Coeur d'Alene Basin
ES-2 Arithmetic and Geometric Mean Blood Lead Concentrations by Geographic Area- 9 Month through 9 Year Old Children (1996-1999 Combined)
ES-3 Basin Mean Blood Lead Levels by Age (1996-1999 combined)
ES-4 Percent to Exceed Blood Lead Concentrations by Geographic Area-9 Month through 9 Year old Children (1996-1999 Combined)
ES-5 Percent of Children to Exceed Critical Toxicity Levels by Age (Basin-wide 1996-1999 Combined)
ES-6 Observed and Predicted Geomean Blood Lead Levels for 0-84 Month Old Children Only-IEUBK Batch Mode
ES-7 Observed and Predicted Percent to Exceed 10 Fg/dl for 0-84 Month Old Children-IEUBK Batch Mode

1-1 Site Location Map

2-1 Stream Segments in Eastern Portion of Coeur d'Alene River Basin
2-2 Stream Segments in Western Portion of Coeur d'Alene River Basin

3-1a Basin Study Regions
3-1b Census Block Groups Overlaid Onto Basin Study Areas
3-2 Coeur d'Alene River Basin School District Enrollment 1990 to 2000
3-3 Lower Basin Conceptual Site Model
3-4 Kingston Conceptual Site Model
3-5 Side Gulches Conceptual Site Model
3-6 Osburn Conceptual Site Model
3-7 Silverton Conceptual Site Model
3-8 Wallace Conceptual Site Model
3-9 Ninemile Conceptual Site Model
3-10 Mullan Conceptual Site Model
3-11 Blackwell Island Conceptual Site Model
3-12 Baseline Risk Assessment for Area 5 Lower Basin Area "Southwestern Section"
3-13 Baseline Risk Assessment for Area 5 Lower Basin Area "Central Section"
3-14 Baseline Risk Assessment for Area 5 Lower Basin Area "Northeastern Section"
3-15 Baseline Risk Assessment for Area 4 Kingston Area
3-16 Baseline Risk Assessment for Area 3 Side Gulches Area
3-17 Baseline Risk Assessment for Area 7 Silverton Area
3-18 Baseline Risk Assessment for Area 8 Wallace Area
3-19 Baseline Risk Assessment for Osburn, Silverton and Wallace Areas Combined, Soil/Sediment Sampling Locations
3-20 Baseline Risk Assessment for Osburn, Silverton and Wallace Areas Combined, Surface Water Sampling Locations

3-21 Baseline Risk Assessment for Area 2 Burke/Ninemile Area, Soil/Sediment Sampling Locations
3-22 Baseline Risk Assessment for Area 2 Burke/Ninemile Area, Surface Water Sampling Locations
3-23 Baseline Risk Assessment for Area 2 Burke/Ninemile Area, Groundwater Sampling Locations
3-24 Baseline Risk Assessment for Area 1 Mullan Area "West Section"
3-25 Baseline Risk Assessment for Area 1 Mullan Area "East Section"
3-26 Baseline Risk Assessment for Blackwell Island Area

5-1 Summary of Total RME Noncancer Hazard
5-2 Total RME Noncancer Hazard—Residential (Child 0 - 6), All Chemicals
5-3 Total RME Noncancer Hazard—Residential (Child/Adult), All Chemicals
5-4 RME Noncancer Hazard—Residential (Child 0 - 6), by Chemical
5-5 RME Noncancer Hazard—Residential (Child/Adult), by Chemical
5-6 RME Noncancer Hazard—Future Residential, Burke/Ninemile
5-7 Total RME Noncancer Hazard—Neighborhood Recreational (Child 4 - 11), All Chemicals
5-8 RME Noncancer Hazard—Neighborhood Recreational (Child 4 - 11), by Chemical
5-9 Total RME Noncancer Hazard—Public Recreational (Child 0 - 6), All Chemicals
5-10 RME Noncancer Hazard—Public Recreational (Child 0 - 6), by Chemical
5-11 RME Noncancer Hazard—Occupational (Adult), by Chemical
5-12a RME Noncancer Hazard—Recreational Fish Consumption (Adult)
5-12b RME Noncancer Hazard—Residential Vegetable Consumption
5-13 Summary of RME Cancer Risk
5-14 RME Cancer Risk—Residential
5-15 RME Cancer Risk—Neighborhood Recreational (Child 4 - 11)
5-16 RME Cancer Risk—Public Recreational
5-17 RME Cancer Risk—Occupational (Adult)
5-18 RME Noncancer Hazard—Residential Plus Neighborhood Recreational, All Chemicals
5-19 RME Cancer Risk—Residential Plus Neighborhood Recreational
5-20 Total RME Noncancer Hazard—Modern and Traditional Subsistence Exposure Scenarios, All Chemicals (Child Age 0 to 6 years)
5-21 Total RME Noncancer Hazard—Modern and Traditional Subsistence Exposure Scenarios, All Chemicals (Adult/Child)
5-22 Total RME Cancer Risk—Modern and Traditional Subsistence Exposure Scenarios (Adult/Child)

6-1a Arithmetic and Geometric Mean Blood Lead Concentrations by Geographic Area - 9 Month Through 9 Year Old Children (1996 - 1999 combined)
6-1b Percent to Exceed Blood Lead Concentrations by Geographic Area - 9 Month Through 9 Year old Children (1996 - 1999 combined)
6-2 Basin Mean Blood Lead Levels by Age (1996 - 1999 combined)

6-3 Percent of Children to Exceed Critical Toxicity Levels by Age (Basin-wide 1996 - 1999 combined)
6-4 Geometric Mean Adult Blood Lead Levels by Age and Geographic Area
6-5 Geometric Mean and Maximum Blood Lead Levels for Reproductive Aged Females (17-45 Years Old) by Geographic Area
6-6 Environmental Pathways for Lead Exposure for the Bunker Hill Site
6-7a Geometric Mean Interior Paint Lead Loading by Geographic Area
6-7b Geometric Mean Exterior Paint Lead Loading by Geographic Area
6-8a Arithmetic Mean Soil and House Dust Lead Concentrations by Geographic Area
6-8b Arithmetic Mean Soil and House Dust Lead Concentrations by Geographic Area
6-8c Geometric Mean Soil and House Dust Lead Concentrations by Geographic Area
6-9a Geometric Mean Dust Mat Lead Concentration by Geographic Area
6-9b Geometric Mean Dust Loading Rate by Geographic Area
6-9c Geometric Mean Dust Mat Lead Loading Rate by Geographic Area
6-10a Scatterplots for Blood Lead Concentration and Environmental Source Variables
6-10b Scatterplots for Dust Lead and Environmental Source Variables
6-10c Scatterplots for Dust Lead Loading and Environmental Source Variables
6-10d Scatterplots for Vacuum Dust Concentration and Environmental Variables
6-11 Percentage of Blood Lead Observations ≥10 ug/dl Associated with an Interior Lead Paint Hazard
6-12a Comparison of Geometric Mean Environmental Lead Levels Between High and Low Blood Lead Levels in Children (1-9 yrs.) Exposed to an Interior Lead Paint Hazard
6-12b Comparison of Geometric Mean Environmental Lead Levels Between High and Low Blood Lead Levels in Children (1-9 yrs.) Not Exposed to an Interior Lead Paint Hazard
6-13 Geometric Mean Dust Mat and Vacuum Bag Dust Lead Concentration by Geographic Area
6-14a Estimated Lead Intake for Four-year-old Children by Geographic Area - EPA Default
6-14b Estimated Lead Intake for Four-year-old Children by Geographic Area - Box Model
6-15a Occupational CT Lead Intake Rates Compared to Baseline
6-15b Occupational RME Lead Intake Rates Compared to Baseline
6-16a Summary of Children's Recreational Potential Incremental Lead Intakes - CT
6-16b Summary of Children's Recreational Potential Incremental Lead Intakes - RME
6-16c Summary of Adult Recreational Potential Incremental Lead Intakes -CT
6-16d Summary of Adult Recreational Potential Incremental Lead Intakes - RME
6-17a Estimated CT Tribal Children Lead Intake Rates
6-17b Estimated RME Tribal Children Lead Intake Rates
6-18a Estimated CT Tribal Adult Lead Intake Rates
6-18b Estimated RME Tribal Adult Lead Intake Rates
6-19a Observed and Predicted Geomean Blood Lead Levels for 9-84 Month Old Children Only-IEUBK Batch Mode
6-19b Observed and Predicted Blood Lead Levels for 9-60 Month Old Children-IEUBK Batch Mode

6-19c Observed and Predicted Blood Lead Levels for 9-24 Month Old Children-IEUBK Batch Mode
6-20a Observed and Predicted Percent to Exceed 10 Fg/dl for 9-84 Month Old Children-IEUBK Batch Mode
6-20b Observed and Predicted Percent to Exceed Levels for 9-60 Month Old Children-IEUBK Batch Mode
6-20c Observed and Predicted Percent to Exceed Levels for 9-24 Month Old Children-IEUBK Batch Mode
6-21a Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Mullan
6-21b Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Burke/Nine Mile
6-21c Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Wallace
6-21d Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Silverton
6-21e Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Osburn
6-21f Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Side Gulches
6-21g Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Kingston
6-21h Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Lower Basin
6-22a Estimated Percent of Children to Exceed 10 µg/dl Blood Lead Associated with Various Yard Soil Cleanup Action Criteria Using Different Dust Concentrations - Wallace
6-22b Estimated Percent of Children to Exceed 10 µg/dl Blood Lead Associated with Various Yard Soil Cleanup Action Criteria Using Different Dust Concentrations - Lower Basin
6-23a Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Mullan
6-23b Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Burke/Nine Mile
6-23c Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Wallace
6-23d Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Silverton
6-23e Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Osburn
6-23f Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Side Gulches
6-23g Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Kingston

6-23h Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Lower Basin
6-24a Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Mullan
6-24b Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Burke/Nine Mile
6-24c Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Wallace
6-24d Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Silverton
6-24e Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Osburn
6-24f Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Side Gulches
6-24g Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Kingston
6-24h Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Lower Basin

7-1 Cadmium Concentrations in All Vegetables and Soil
7-2 Cadmium Concentrations in Leafy Vegetables and Soil
7-3 Cadmium Concentrations in Root Vegetables and Soil

8-1 Coeur d'Alene Basin
8-2 Percent to Exceed Blood Lead Concentrations by Geographic Area - 9 Month through 9 Year old Children (1996 - 1999 Combined)
8-3 Arithmetic and Geometric Mean Blood Lead Concentrations by Geographic Area - 9 Month through 9 Year Old Children (1996 - 1999 Combined)
8-4 Basin Mean Blood Lead Levels by Age (1996 - 1999 combined)
8-5 Percent of Children to Exceed Critical Toxicity Levels by Age (Basin-wide 1996 - 1999 Combined)
8-6 Observed and Predicted Geomean Blood Lead Levels for 0-84 Month Old Children Only - IEUBK Batch Mode
8-7 Observed and Predicted Percent to Exceed 10 µg/dl for 0-84 Month Old Children-IEUBK Batch Mode
8-8a Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Mullan
8-8b Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Burke/Nine Mile
8-8c Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Wallace

8-8d Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Silverton
8-8e Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Osburn
8-8f Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Side Gulches
8-8g Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Kingston
8-8h Predicted Percentage of 0-84 Month Old Children to Exceed 10 µg/dl Blood Lead for Various Yard Soil Action Levels - Lower Basin

TABLES

ES-1 Selected Chemicals of Potential Concern in Each Medium
ES-2 RME Hazard Quotients and Cancer Risks for Arsenic for Modern and Traditional Future Subsistence Exposure Scenarios
ES-3 RME Hazard Quotients and Cancer Risks for Residential and Neighborhood Scenarios
ES-4 RME Hazard Quotients and Cancer Risks for Public Recreational Scenario
ES-5 RME Hazard Quotients and Cancer Risks for Occupational Scenario

2-1 Stream Segments and Beach Sites in CSM Units, 1, 2, 3, and 4
2-2 Chemicals With Sample Quantitation Limits Exceeding Screening Values
2-3 Potential Background Concentrations for Soil, Surface Water, and Groundwater
2-4 Soil Screening Values for Air Pathway
2-5 Summary of Analytical Results for Chemicals in Soil/Sediment With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples
2-6 Summary of Analytical Results for Chemicals in First-Run Tap Water With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples
2-7 Summary of Analytical Results for Chemicals in Flushed-Line Tap Water With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples
2-8 Summary of Analytical Results for Chemicals in Surface Water With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples
2-9 Summary of Analytical Results for Chemicals in Groundwater With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples
2-10 Summary of Analytical Results for Chemicals in House Dust With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples
2-11 Summary of Analytical Results for Chemicals in Air With Concentrations Exceeding Screening Values in Any Sample
2-12 COPCs Selected for Each Medium

3-1a Summary of Basin Geographic Areas and Population (Source: 1990 Census Data)
3-1b Survey Subareas Included Within Proposed HHRA Geographic Subdivision

3-2 Basin Demographics Over Time (Source: County Profiles of Idaho, IDOC and 1990 Census)
3-3 Census Block Groups Falling Partially Outside of Basin Study Area Boundaries (Source: 1990 Census)
3-4 Summary Population Characteristics
3-5 Household Characteristics (Source: 1990 Census)
3-6 Housing Characteristics (Source 1990 Census)
3-7 Comparison of Median Values of Housing (Source: 1990 Census)
3-8 Student Population and Educational Attainment (Source: 1990 Census)
3-9 Basin Area Household Income (Source: 1990 Census)
3-10 Shoshone County Profile (Source: Profile of Rural Idaho, IDOC)
3-11 Kids Count Data as Presented in Yearly Reports (Source: Idaho KIDS COUNT: Profiles of Child Well-Being 1996-2000)
3-12 1999-2000 Kids Count Data - Economic Well Being (Source: Idaho KIDS COUNT: Profiles of Child Well-Being 1999-2000 and School District Data)
3-13 1999-2000 Kids Count Data - Child Population Change (Source: Idaho KIDS COUNT: Profiles of child Well-being 1999-2000)
3-14 School District Data (Source: School Districts #391, 392, and 393)
3-15 School District Enrollment by Grade (Source: School Districts #391, 392, and 393)
3-16 Estimated Child Population and Sample Population (Sources: 1990 Census, Idaho KIDS COUNT, and School District Data)
3-17 Public and Private Sewer and Water Hookups (Source: 1990 Census and 1999 Sewer District (SD) data)
3-18 Estimated Number of Housing Units by Basin Area (Source: 1990 Census and 1999 Sewer District Data)
3-19a Summary of Exposure Pathways Quantified in HHRA
3-19b Tribal Exposure Routes To Be Considered
3-20 Summary of Exposure Point Concentrations for Reasonable Maximum Exposure
3-21 Number of Samples Used to Calculate Exposure Point Concentrations
3-22 Residential Exposure Factors
3-23 Neighborhood Recreational Exposure Factors
3-24 Public Recreational Exposure Factors
3-25 Occupational Exposure Factors
3-26a Traditional Subsistence Scenario Exposure Factors
3-26b Modern Subsistence Scenario Exposure Factors

4-1 Oral Toxicity Criteria
4-2 Lowest-Observed-Effect Levels in Children Exposed to Lead
4-3 Lowest-Observed-Effect Levels in Adults Exposed to Lead

5-1 Summary of Hazard/Risk Estimates and Risk Drivers
5-2 Chemicals With Hazard Indices Greater Than or Equal to 1

5-3 Summary of Hazard/Risk Estimates for Combined Child/Adult Residential and Neighborhood Recreational Scenarios
5-4 Summary of RME Hazard/Risk Estimates and Risk Drivers for Modern Subsistence Exposure Scenario
5-5 Summary of RME Hazard/Risk Estimates and Risk Drivers for Traditional Subsistence Exposures
5-6 Potential Preliminary Remediation Goals for Arsenic
5-7 Summary of the Percent of Basin Residences with 95 Percent UCL Arsenic Concentrations Exceeding Selected Potential PRGs

6-1a Summary of Basin Blood Lead Level Observations by Year (9 Month to 9 Year Old Children)
6-1b Summary of the Number of Blood Lead Observations and Repeat Children
6-2 Annual Blood Lead Summary Data by Geographic Subarea for Children (Fg/dl) (9 Months Through 9 Years)
6-3 Geographic Subarea Blood Lead Summary Data for All Children (1 year through 9 years old) For All Years (Fg/dl)
6-4a Basin Blood Lead Levels for 1-9 Year Old Children, All Years Combined (Fg/dl)
6-4b Basin Blood Lead Levels for 1-6 Year Old Children, All Years Combined (Fg/dl)
6-4c Basin Blood Lead Levels for 1-7 Year Old Children, All Years Combined (Fg/dl)
6-5 Basin Blood Lead Levels by Geographic Area and Age (µg/dl)
6-6a Basin-wide Blood Lead Level Summary for 1 Through 6 Year Old Children by Area (Survey Results-All Years)
6-6b Basin-wide Blood Lead Level Summary for 1-7 Year Old Children by Area (Survey Results-All Years)
6-7 Comparison of Coeur d'Alene and BHSS Results to National and State-Wide Blood Lead Levels
6-8a Summary of Adult Blood Lead Levels by Age and Geographic Subarea (Fg/dl)
6-8b Observed Blood Lead Levels for Reproductive Aged Females (Fg/dl) by Geographic Subarea
6-9 Dietary Lead Summary from IEUBK Model for Children
6-10 Summary of Garden, Fish, and Riparian Vegetation Lead Levels
6-11a Mullan: Summary Lead Concentration Data for Environmental Media (mg/kg) and Percent of Soil/Dust Samples by Concentration Category (mg/kg)
6-11b Burke/Nine Mile: Summary Lead Concentration Data for Environmental Media (mg/kg) and Percent of Soil/Dust Samples by Concentration Category (mg/kg)
6-11c Wallace: Summary Lead Concentration Data for Environmental Media (mg/kg) and Percent of Soil/Dust Samples by Concentration Category (mg/kg)
6-11d Silverton: Summary Lead Concentration Data for Environmental Media (mg/kg) and Percent of Soil/Dust Samples by Concentration Category (mg/kg)
6-11e Osburn: Summary Lead Concentration Data for Environmental Media (mg/kg) and Percent of Soil/Dust Samples by Concentration Category (mg/kg)

6-11f Side Gulches: Summary Lead Concentration Data for Environmental Media (mg/kg) and Percent of Soil/Dust Samples by Concentration Category (mg/kg)
6-11g Kingston: Summary Lead Concentration Data for Environmental Media (mg/kg) and Percent of Soil/Dust Samples by Concentration Category (mg/kg)
6-11h Lower Basin: Summary Lead Concentration Data for Environmental Media (mg/kg) and Percent of Soil/Dust Samples by Concentration Category (mg/kg)
6-11i Harrison: Summary Lead Concentration Data for Environmental Media (mg/kg) and Percent of Soil/Dust Samples by Concentration Category (mg/kg)
6-11j Rocky Point: Summary Lead Concentration Data for Environmental Media (mg/kg) and Percent of Soil/Dust Samples by Concentration Category (mg/kg)
6-12a Summary of CUA Surface Water Lead Concentration (Disturbed Samples)(Fg/L)
6-12b Summary of CUA Surface Water Lead Concentration (Undisturbed Samples)(Fg/L)
6-13 Paint Lead Loadings by Geographic Area
6-14 Summary of Neighborhood Stream Sediment Lead Levels by Geographic Subarea
6-15a Summary of Surface Soil Lead Levels for Common Use Areas (mg/kg)
6-15b Summary of Sediment Lead Levels for Common Use Areas (mg/kg)
6-16 Summary of Lead Concentration for Waste Pile Soils (mg/kg)
6-17 Mat Dust Lead Concentration, Dust Loading, and Lead Loading for the Coeur d'Alene Basin
6-18 Summary of Lead Concentrations for Sediment and Surface Soil in the Lower Basin
6-19 Correlation Matrix for Blood Lead Levels and Environmental Source Variables
6-20 Correlation Matrix for House Dust Lead Levels and Environmental Source Variables
6-21 General Linear Model and Regression Coefficients for Blood Lead and Environmental Sources
6-22a General Linear Model and Regression Coefficients for Entryway Mat Lead Concentration and Environmental Sources
6-22b General Linear Model and Regression Coefficients for Entryway Mat Lead Concentration and Environmental Sources
6-22c General Linear Model and Regression Coefficients for Entryway Mat Lead Concentration and Environmental Sources (Final Model)
6-22d General Linear Model and Regression Coefficients for Dust Mat Lead Loading and Environmental Sources
6-23 General Linear Model and Regression Coefficients for Vacuum Bag Lead Concentration and Environmental Sources
6-24a IEUBK Default Inhalation and Ingestion Parameters
6-24b EPA Adult Model Default Ingestion Parameters
6-25a Four Year Old Children's Baseline Lead Intake Rates for Ingestion of Soil and Dust - EPA Default
6-25b Four Year Old Children's Baseline Lead Intake Rates for Ingestion of Soil and Dust - Box Model
6-26 Adult Baseline Lead Intake Rates for Ingestion of Soil and Dust
6-27a Adult Occupational Exposure Factors for Non-lead Soil Related Activities Ingestion

6-27b Adult Occupational Exposure Factors for Lead Soil Related Activities Ingestion
6-27c EPA Adult Lead Default Values for Non-Soil Related Activities
6-28a Estimated Occupational Intake Rates Associated with Community Soils for Geographic Subareas Using Non-Lead Ingestion Rates
6-28b Estimated Occupational Intake Rates Associated with Community Soils for Geographic Subareas Using EPA Adult Model Ingestion Rates
6-29a Estimated Occupational Incremental Lead Intake Rates for Potential Soil Lead Concentration Levels using Non-lead Ingestion Rates
6-29b Estimated Occupational Incremental Lead Intake Rates for Potential Soil Lead Concentration Levels using Lead Soil Related Ingestion Rates
6-30 Occupational Lead Intake Rates for Adult Occupational Scenarios
6-30a Percentile Lead Concentration for Occupational Soils
6-30b Percentile Lead Intake Rates For Occupational Soils
6-30c Central Tendency and Reasonable Maximum Exposure Lead Intakes Compared to Baseline
6-31 Recreational Exposure Factors
6-32a Incremental Sediment/Soil Ingestion Lead Intake for Children-Central Tendency (CT)
6-32b Incremental Sediment/Soil Ingestion Lead Intake for Children- Reasonable Maximum Exposure (RME)
6-32c Incremental Sediment/Soil Ingestion Intake for Adults-Central Tendency (CT) and Reasonable Maximum Exposure (RME)
6-33 Recreational Surface Water Exposure Factors
6-34a Incremental Recreational Surface Water Ingestion Lead Intake Rates for Children
6-34b Incremental Recreational Surface Water Ingestion Lead Intake Rates for Adults
6-35 Upland Parks Incremental Intakes for Children
6-35a Percentile Surface Soil Lead Concentrations
6-35b Lead Intakes Associated with Concentration Percentiles
6-35c Incremental Central Tendency and Reasonable Maximum Exposure Intake Rates Compared to Baseline
6-36 Neighborhood Stream Incremental Intakes for Children 4 Years Through 11 Years Old
6-36a Percentile Lead Concentration for Sediment and Surface Water
6-36b Percentile Lead Intake Rates for Sediment and Surface Water
6-36c Central Tendency and Reasonable Maximum Exposure Intake Estimates Compared to Baseline
6-37 CUA Public Beach Incremental Intakes for Children 0 Through 6 Years Old
6-37a Percentile Lead Concentrations for Sediments and Surface Water
6-37b Percentile Lead Intake Rates by Media
6-37c Central Tendency and Reasonable Maximum Exposure Lead Intake Rates Compared to Baseline
6-38 Incremental Lead Intake Rates for Waste Piles for Children 4 through 11 Years Old
6-38a Percentile Lead Concentrations for Waste Pile Soils
6-38b Percentile Lead Intake Rates for Waste Piles

6-38c Central Tendency and Reasonable Maximum Exposure Incremental Lead Intakes Compared to Baseline
6-39 Local Foodstuff Exposure Factors for the Resident Populations
6-40a Children's Incremental Lead Intake Rate from Home Grown Produce
6-40b Adults' Incremental Lead Intake Rate from Home Grown Foodstuff
6-41a Recreational Fish Incremental Intakes for Children
6-41b Recreational Fish Incremental Intakes for Adults
6-42a Summary of Children's Potential Incremental Intakes (CT)
6-42b Summary of Children's Potential Incremental Intakes (RME)
6-43a Summary of Adult's Potential Incremental Intakes (CT)
6-43b Summary of Adult's Potential Incremental Intakes (RME)
6-44a Traditional Tribal Lead Intake Rate Estimates for Children by Quantile Percentages
6-44b Traditional Tribal Lead Intake Rate Estimates for Adults by Quantile Percentages
6-45 Modern Tribal Lead Intake Rate Estimates for Children by Quantile Percentages
6-46 Modern Tribal Lead Intake Rate Estimates for Adults by Quantile Percentages
6-47a Predicted and Observed Baseline Blood Lead Levels Community Mode IEUBK- 9-84 Months
6-47b Predicted and Observed Baseline Blood Lead Levels Community Mode IEUBK- 9-24 Months
6-47c Predicted and Observed Baseline Blood Lead Levels Community Mode IEUBK- 9-60 Months
6-48 Summary of Blood Lead, Yard Soil, and Vacuum Dust for IEUBK Batch Mode Input
6-49 IEUBK Batch Mode Overall Observed v. Predicted Blood Lead by Age
6-50a IEUBK Batch Mode Overall Observed v. Predicted Blood Lead by Geographic Area, Ages 9-84 Months: Default and 40:30:30
6-50b IEUBK Batch Mode Observed v. Predicted Blood Lead by Geographic Area, Age 9-60 Months: Default and 40:30:30
6-50c IEUBK Batch Mode Observed v. Predicted Blood Lead by Geographic Area, Age 9-24 Months: Default and 40:30:30
6-51a Upland Parks Estimated Recreational Blood Lead Increments for 0-84 Month Old Children (CT)
6-51b Upland Parks Estimated Recreational Blood Lead Increments for 0-84 Month Old Children (RME)
6-52a Neighborhood Stream Recreational Blood Lead Increment for 0-84 Month Old children (CT)
6-52b Neighborhood Stream Recreational Blood Lead Increment for 0-84 Month Old Children (RME)
6-53a Public Beach Recreational Blood Lead Increment for Children 0-84 Month Old (CT)
6-53b Public Beach Recreational Blood Lead Increment for Children 0-84 Month Old (RME)
6-54a Waste Pile Recreational Blood Lead Increment for 0-84 Month Old Children (CT)
6-54b Waste Pile Recreational Blood Lead Increment for 0-84 Month Old Children (RME)
6-55a Home Grown Vegetable Central Tendency (CT) Intakes for Children (0-84 mos.)

6-55b Home Grown Vegetable Reasonable Maximum Exposure (RME) Intakes for Children (0-84 mos.)
6-56a Recreational Fish Ingestion Central Tendency (CT) Intakes for Children (0-84 mos.)
6-56b Recreational Fish Ingestion Reasonable Maximum Exposure (RME) Intakes for Children (0-84 mos.)
6-57 Calculations of Preliminary Remediation Goals (PRGs) using the Occupational CT Values
6-58 Calculations of Preliminary Remediation Goals (PRGs) using the Occupational RME Values
6-59a Calculations of Preliminary Remediation Goals (PRGs) using the Upland Parks CT Values
6-59b Calculations of Preliminary Remediation Goals (PRGs) using the CUAs CT Values
6-60a Calculations of Preliminary Remediation Goals (PRGs) using the Upland Parks RME Values
6-60b Calculations of Preliminary Remediation Goals (PRGs) using the CUA RME Values
6-61a Estimated Post-Remedial Blood Lead Levels and Probability to Exceed Health Criteria by Community and Individuals for a 2000 mg/kg Soil Action Level - EPA Default Model
6-61b Estimated Post-Remedial Blood Lead Level and Probability to Exceed Health Criteria by Community and Individuals for a 1500 mg/kg Soil Action Level - EPA Default Model
6-61c Estimated Post-Remedial Blood Lead Level and Probability to Exceed Health Criteria by Community and Individuals for a 1000 mg/kg Soil Action Level - EPA Default Model
6-61d Estimated Post-Remedial Blood Lead Level and Probability to Exceed Health Criteria by Community and Individuals for a 800 mg/kg Soil Action Level - EPA Default Model
6-61e Estimated Post-Remedial Blood Lead Level and Probability to Exceed Health Criteria by Community and Individuals for a 600 mg/kg Soil Action Level - EPA Default Model
6-61f Estimated Post-Remedial Blood Lead Level and Probability to Exceed Health Criteria by Community and Individuals for a 400 mg/kg Soil Action Level - EPA Default Model
6-62a Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 2000 mg/kg Soil Action Level-Box Model
6-62b Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 1500 mg/kg Soil Action Level-Box Model
6-62c Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 1000 mg/kg Soil Action Level-Box Model
6-62d Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 800 mg/kg Soil Action Level-Box Model
6-62e Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 600 mg/kg Soil Action Level-Box Model
6-62f Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 400 mg/kg Soil Action Level-Box Model

7-1 Effect of Reduced Dermal Surface Areas (SAs) on Current Total RME Risks and Hazards for the Neighborhood Recreational Scenario
7-2 Summary of Geometric Means and Ratios of Chemical Concentrations in House Dust and Yard Soil
7-3 Cadmium Concentrations in Vegetables and Soil
7-4 Generic and Basin-Specific Elements of Uncertainty in Blood Lead (Pb-B) Data Gathering
7-5 Elements of Uncertainty in Environmental Lead Data Gathering and Assessment in The Basin
7-6 Elements of Uncertainty in Lead Exposure Data in the Basin
7-7 Elements of Uncertainty in IEUBK Modeling of Blood Lead (Pb-B) Levels in the Basin
7-8 Summary Statistics for Environmental Variables for All Data and the Subset of Data Paired with Blood Lead Observations

8-1 Selected Chemicals of Potential Concern in Each Medium
8-2 Health Effects of Exposure to Chemicals of Potential Concern
8-3 RME Hazard Quotients and Cancer Risks for Residential and Neighborhood Scenarios
8-4 RME Hazard Quotients and Cancer Risks for Public Recreational Scenario
8-5 RME Hazard Quotients and Cancer Risks for Occupational Scenario
8-6 RME Hazard Quotients and Cancer Risks for Arsenic for Modern and Traditional Future Subsistence Exposure Scenarios

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ABBREVIATIONS AND ACRONYMS

ALA	ä-aminolevulenic acid
ALAD	ä-aminolevulinic acid dehydrase
ARAR	applicable or relevant and appropriate requirement
ATSDR	Agency for Toxic Substances Disease Registry
AWQC	Ambient Water Quality Criteria
bgs	below ground surface
BHSS	Bunker Hill Superfund Site
CDAB	Coeur d' Alene River Basin
CLP	Contract Laboratory Program
cm	centimeter
cm^2	square centimeter
COPC	chemical of potential concern
CSM	conceptual site model
CT	central tendency
CTE	central tendency estimate
CUA	common use areas
Ecology	Washington State Department of Ecology
EP	erythrocyteprotoporphyrin
EPC	exposure point concentration
FDA	Food and Drug Administration
FS	feasibility study
FSPA	field sampling plan addendum
g	gram
g/kg-day	grams per kilogram per day
GSD	geometric standard deviation
HDL	high-density lipoprotein
HEAST	Health Effects Assessment Summary Tables
HHRA	human health risk assessment
IEUBK	Integrated Exposure Uptake Biokinetic
IRIS	Integrated Risk Information System
kg	kilogram
kg/g	kilogram per gram
kg/mg	kilogram per milligram
L	liter
L/kg	liter per kilogram
LDL	low-density lipoprotein
LHIP	Lead Health Intervention Program
LOAEL	lowest-observed-adverse-effect level
LOEL	lowest-observed-effect level
MCL	Maximum Contaminant Level

µg/dl	microgram per deciliter
µg/L	microgram per liter
$\mu g/m^3$	microgram per cubic meter
µm	micrometer
mg	milligram
mg/kg	milligram per kilogram
mg/kg-day	milligram per kilogram per day
mg/m^3	milligram per cubic meter
mg/µg	milligram per microgram
mL	milliliter
MLE	maximum likelihood estimate
MRL	Minimum Risk Level
MTCA	Model Toxics Control Act
NAAQC	National Ambient Air Quality Criteria
NAS	National Academy of Sciences
NFCS	Nationwide Food Consumption Survey
NHANES	National Health and Nutrition Examination Survey
NHANES II	second National Health and Nutrition Examination Survey
NHANES III	third National Health and Nutrition Examination Survey
NOAEL	no-observed-adverse-effect level
NRC	National Research Council
NS confluence	confluence of the North Fork and the South Fork
PET	particulate emission factor
PHD	Panhandle Health District
ppb	parts per billion
ppm	parts per million
PRG	preliminary remediation goal
RAO	Remedial Action Objectives
RDA	recommended daily allowance
RfC	reference concentration
RfD	reference dose
RI	remedial investigation
RME	reasonable maximum exposure
ROD	Records of Decision
ROW	Rights-of-way
SF	slope factor
SV	screening value
UCL_{95}	95 percent upper confidence limit
USDA	U.S. Department of Agriculture
USEPA	United States Environmental Protection Agency
WIC	Woman Infant and Children
XRF	X-ray fluorescent

The Human Health Risk Assessment (HHRA) for the Coeur d'Alene Basin Extending from Harrison to Mullan on the Coeur d'Alene River and Tributaries.

EXECUTIVE SUMMARY

This ***EXECUTIVE SUMMARY*** includes the main findings and a brief description of the Coeur d'Alene Basin (CDAB) HHRA. A more complete synopsis is found in the ***SUMMARY and CONCLUSIONS*** document that is also Section 8 of the HHRA. That document parallels the larger HHRA report and is intended as a complete review for the general public. Those readers requiring additional detail should consult the full HHRA and the Appendices that are provided on an attached CD. These documents are available for review at the local information repositories, the Idaho Department of Environmental Quality (DEQ) office in Kellogg and the U.S. Environmental Protection Agency (USEPA) office in Coeur d'Alene.

Study Area: The CDAB, including Lake Coeur d'Alene and the Coeur d'Alene River drainage, is the ancestral home of the Coeur d'Alene Indian Tribe. Since the late 19th century, this area has been the center of one of the most productive mining districts in the world. During most of the last century, substantial quantities of industrial wastes were directly discharged into the environment from mining, mineral processing, and smelting activities. Public health investigations in the 1970s to 1980s resulted in the designation, in 1983, of a 21 square mile area called the Bunker Hill Superfund Site (BHSS), or "the Box," surrounding the former smelter complex near Kellogg. Remedial activities and public health response activities have been ongoing in the BHSS for two decades.

RI/FS: A Remedial Investigation and Feasibility Study (RI/FS) is currently being undertaken to characterize the degree and extent of the contaminant release in the CDAB outside "the Box". Concurrent with the RI/FS, this baseline HHRA addresses potential human health risks associated with residual heavy metals contamination for areas east of Harrison upstream from the mouth of the Coeur d'Alene River. A screening level HHRA was previously completed for Coeur d'Alene Lake beach areas, and a similar screening level HHRA is being completed for the Spokane River that drains Lake Coeur d'Alene into the State of Washington.

Baseline HHRA: The baseline risk assessment is an evaluation of the potential threats to public health from site contaminants in the absence of any remedial action. The primary tasks accomplished in performing the HHRA included data collection, data evaluation, exposure assessment, toxicity assessment, and risk characterization. The main purpose of this HHRA is to determine the extent of heavy metal contamination in environmental media that current or future residents and visitors to the CDAB may come in contact with, to evaluate the potential human health risks associated with exposure to those contaminated media, and to provide information for risk managers to evaluate the need for remedial action and development of associated clean-up criteria. Figure ES-1 shows various features of the CDAB.

Geographic Subareas: The Basin was divided into eight principal HHRA geographic subareas based on existing communities, identified routes of potential human exposure, public use patterns, and the results of environmental lead health surveys in each area. Those geographic subareas shown on Figure ES-1 are: the Lower Basin (the floodplain of the lower Coeur d'Alene River from Harrison to, and including Cataldo), Kingston (including the area of the Basin between the BHSS and Cataldo), the Side Gulches (including residences in the side canyons along streams draining into the South Fork between the BHSS and Silverton), Osburn, Silverton, Wallace, Burke/Nine Mile (including Nine Mile Creek and Canyon Creek), and Mullan.

Data Used in the HHRA: In addition to traditional geographic, climatic, and demographic information, two basic data sources were used in the HHRA. Those data either i) originated in investigations associated with the RI/FS or the Natural Resource Damage Assessment (NRDA) being conducted by federal and Tribal trustees, or ii) obtained in health surveys conducted by the State Department of Health and Welfare and allied local and federal health agencies. The principal source of the latter data was a comprehensive blood lead and environmental exposure study conducted in 1996, and follow-up blood lead surveys conducted in 1997-1999.

Special Health Concerns: Chemicals of potential concern (COPCs) were identified using a decision process that included a comparison of detected chemical concentrations with health-based screening values (SV). In total, eight metals were selected for assessment including antimony, arsenic, cadmium, iron, lead, manganese, mercury and zinc. The principal health concerns are associated with lead and its potential to cause neurological developmental effects in children; and arsenic for its potential to cause cancers of the skin, bladder, kidney, lung, and liver, and various pre-cancer and noncancer effects in skin by ingestion. Table ES-1 summarizes the COPCs for each media evaluated.

Populations of Potential Concern: Certain population groups in the Basin could be more sensitive to contamination, or more likely to be subjected to greater exposure than the typical individual. These populations include *infants, children, pregnant women* as they represent the *fetus,* and individuals with *subsistence lifestyles,* including some Coeur d'Alene Tribe members.

Lead Health Surveys: The greatest health concern is lead poisoning and excess lead absorption noted in health surveys of the resident population. Lead health risks are assessed by comparing blood lead levels to current Centers for Disease Control (CDC) criteria: excessive prevalence of blood lead levels in the 10 µg/dl -14 µg/dl range are indicative of excess exposure, levels of 15 µg/dl or greater are indicative of increased risk to individuals, levels exceeding 20 µg/dl call for clinical management, and levels of 45 µg/dl require immediate medical intervention.

Observed Blood Lead Levels: Figure ES-2 summarizes observed blood lead data by geographic subarea for 9 month through 9 year old children in the Basin. Figure ES-3 shows mean blood lead levels by age. The highest blood lead levels are observed in the youngest age groups. One and two year old children have arithmetic mean blood lead levels of 7.0 µg/dl and 8.0 µg/dl, respectively, and geometric

mean concentrations of 6.2 µg/dl to 6.3 µg/dl. Geometric mean levels then decrease with age from 5.2 µg/dl at age 3 to 3.0 µg/dl at age 8.

Children at-risk of Adverse Health Effects: Figures ES-4 and ES-5 summarize the percent of children to exceed critical toxicity levels. The results differ markedly with age. In the lowest age groups, 9 months through 3 years, 19% to 26% of children Basin-wide exceed 10 µg/dl. The rate is highest in 2 year old children with 17% of this group exceeding 15 µg/dl. Among preschool children, 16% have blood lead levels exceeding 10 µg/dl. For 9 month through 9 year old children, 10% of observed blood lead levels exceed 10 µg/dl. The public health criteria adopted for the BHSS, based on federal health advisories, recommends that no more than 5% of children exceed 10 µg/dl with less than 1% greater than 15 µg/dl.

Representativeness of the Surveys: Approximately 25% of eligible children participated in the surveys. Participation was lowest among younger children. There are divergent opinions as to how well the health surveys represent non-participants from throughout the Basin. Although there are no apparent differences in environmental exposure, selection bias related to individual family decisions to participate may have occurred and current representativeness is unknown. One argument suggests that the incidence of lead poisoning is likely greater among non-participants, as families that did have their children tested are more attentive to lead poisoning and have benefitted from the local health department's efforts to assist parents in reducing exposures. A counter argument suggests that paying each child $40 as an incentive favored low-income participation. Because potentially high exposures are associated with poverty-related factors, this argument suggests higher blood lead levels would be expected among the participants.

Related Socio-economic Problems: Lower socio-economic status indicated by the 31% of Shoshone County children living in poverty, the percentage of births paid for by medicaid, subsidized school lunch programs, high welfare payments, low-rents, and high unemployment rates are associated with greater risk of lead poisoning. The substantial decrease in young children in the population indicates young families are continuing to leave the area to look for work. Increased welfare payments to the remaining homes with children may indicate the area is attracting and retaining economically disadvantaged families.

Poverty-related Risk Co-factors: Although children from more affluent areas or higher socio-economic categories are no less susceptible to contaminant exposures and the resulting effects, poverty and lead poisoning interact in several ways to put poor children at greater risk. Less affluent families may have lowered nutritional status and live in poorly maintained housing. Parents may experience more difficulties in managing the home and children, and are less able to provide a stimulating and healthy home environment. Home and child hygiene and behavioral risk co-factors can lead to increased ingestion rates of soils and dusts. Yard soils and house dust can be more contaminated due to deteriorating lead paint, proximity to industrial sources, and lesser quality maintenance of the home, yard, and local infrastructure. The age of housing in the Basin is problematic due to lead paint and accumulation of contaminated dusts throughout the last century. Risk managers may wish to consider socio-economic conditions in the area when developing risk reduction strategies.

Follow-up Investigations: Follow-up investigations were completed by the local health department for 50 of 58 children whose blood lead levels exceeded 10 µg/dl. Twenty-five investigations involving 21 individual children were conducted for observed blood lead levels exceeding 15 µg/dl. Risk profiles indicate excess absorption associated with high soil and dust concentrations at homes in the Burke/Nine Mile subarea. Older children's risk profiles in this area indicate recreational exposures in neighborhood areas contaminated by tailings. High blood lead levels in Wallace are indicated in younger children and are possibly associated with paint and remodeling problems, high soil lead levels in play areas, and dusty or difficult to clean homes. Both Mullan and Osburn had no children greater than the 15 µg/dl blood lead criteria, and children's blood lead levels in the 10 µg/dl to 14 µg/dl range were associated with high residential soil and dust concentrations or play in contaminated areas. West of the BHSS, excess absorption was associated with either soils and sediments in homes that had been flooded in 1996, or extended recreational activities in the river or lateral lakes areas of the Lower Basin.

Site-specific Analysis of Paired Blood and Environmental Lead Data: Site-specific regression analysis of the relationship between blood lead levels and environmental variables indicate that contaminated soils, house dust, and lead based paint are all related to excess absorption. The overall results suggest complex exposure pathways, with blood lead levels most related to dust lead loading in the home, followed by independent effects of yard soil lead, interior paint lead condition, and exterior paint lead content. The dust lead pathway is most influenced by outdoor soils, augmented by paint contributions in older homes, especially those in poor condition. The overall effect is exacerbated by dusty conditions in Burke/Nine Mile and to a lesser extent in Wallace. The Lower Basin is a notable exception. High blood lead levels are observed, although little problem is indicated with respect to residential soils, dustiness or house dust lead concentrations in the Lower Basin. This indicates exposures outside the home environment.

Biokinetic Predictions of Resident Children's Blood Lead Levels: Current federal guidance requires the use of a biokinetic model for lead health risk assessment. The Integrated Exposure Uptake Biokinetic (IEUBK) model was used to estimate residential baseline (everyday home life) blood lead predictions. Both the EPA Default Model (using national assumptions for soil and dust ingestion rates and bioavailability) and the Box Model (derived specifically for the BHSS cleanup) were employed. The Box Model uses a lower bioavailability estimate and includes a community-wide component for soil/dust exposure that is not included in the EPA Default Model.

Predicted and Observed Blood Lead Levels: Figures ES-6 and ES-7 show observed and predicted blood lead levels and percent of children to exceed 10 µg/dl for the EPA Default and Box Models for those children tested in health surveys. East of the BHSS, the baseline Box Model is a better predictor of observed mean blood lead levels. In these areas, the EPA Default baseline model significantly over-predicts both observed concentrations and the percent of children to experience excess absorption. West of the BHSS, and particularly in the Lower Basin, both models are ineffective in describing the observed high blood lead levels. Several possible reasons for the differences in predictions should be considered in the development of risk management strategies, including assumed bioavailabilities, relative contributions of soil and dust sources, the effect of intervention efforts in reducing blood lead levels, and the representativeness of blood lead surveys.

Predicted Need for Residential Cleanup: The EPA Default version of the IEUBK Model Batch Mode application predicts a greater than 5% exceedance of the 10 µg/dl health criteria, *associated with baseline residential exposures*, for all geographic areas. The Box Model predicts exceedance greater than 5% for Mullan, Burke/Nine Mile, Wallace, Silverton, and the Lower Basin. The areas adjoining the BHSS including Kingston, Osburn, and the Side Gulches are projected at less than 5% exceedance for baseline residential exposures by the Box Model.

Preliminary Residential Soil Action Levels: Preliminary analysis, using the Box Model, suggests that a cleanup threshold for soils of 800 mg/kg to 1000 mg/kg is necessary to achieve risk levels in the upper Basin comparable to those established for the BHSS. The EPA Default Model suggests cleanup levels for soils below 400 mg/kg are required to achieve similar risk criteria. Both models indicate that lead paint stabilization will be required in combination with soil remediation to reduce house dust lead concentrations to protective levels. Potential paint stabilization would apply to the approximately 20% of housing units that currently have lead paint in poorly maintained condition. These measures will not resolve excessive blood lead levels observed in the Lower Basin and Kingston.

EPA Policy Regarding Individual Risk: Current USEPA policy addresses individual risks for those children left at the highest exposure levels and recommends that the probability of experiencing a blood lead level of 10 µg/dl or greater, *at any residence*, be less than 5%. Box Model estimates of individual risks indicate this criteria is considerably more stringent than that applied at the BHSS and would require a soil cleanup in the 600 mg/kg to 800 mg/kg range. Using the EPA Default Model to calculate a residential soil cleanup level protective of risk to individuals results in a soil level below the EPA residential soil screening level of 400 mg/kg. This is caused by elevated levels of lead in house dust in portions of the Basin. As a result, risk managers, public health officials and community representatives will need to assess the applicability of this criteria to the Basin population and alternative risk reduction techniques that might provide the necessary level of protectiveness.

Lead Health Risks from Exposures Outside the Residential Environment: Potentially significant recreational exposures are noted for children engaged in certain activities in particular areas of the Basin. Recreational activities can result in significant exposures in the more contaminated areas of the upper Basin and throughout the floodplain areas west of the BHSS. This is a possible explanation for the higher than predicted blood lead levels observed among Lower Basin children. Additionally, swimming and water sport activities in disturbed sediment-laden surface water can result in substantial increases in intake and lead absorption. Potential exposures are of particular concern to neighborhood stream sediments in Burke/Nine Mile, and at public swimming areas in the Side Gulches and the Lower Basin. Potentially significant increases in blood lead levels could also result from consumption of home grown vegetables.

Action Levels for Other Media: Discussion and development of candidate action levels for children's incremental recreational activities and fish and local produce consumption cannot be addressed in this document. Appropriate risk reduction methods and action levels will have to be evaluated by risk managers after fundamental approaches to reducing baseline blood lead levels have been determined.

Assessing whether these actions would be sufficient to reduce non-lead risks and hazards to acceptable levels must also be accomplished in relation to actions addressing cumulative risks to lead.

Coeur d'Alene Tribal Scenarios: The ***subsistence scenario*** pertains to children and adults engaged in traditional (aboriginal) or modern subsistence lifestyles in the floodplain of the lower Coeur d'Alene River. These are future scenarios, as subsistence lifestyles are not known to be currently practiced in the floodplain. Exposure pathways quantified for subsistence lifestyles are similar to those evaluated for residential and recreational receptors. Adequate fish tissue metals concentrations are available for the Lower Basin and the Spokane River. Results for fish in this HHRA should not be extended to the Lake Coeur d'Alene fishery.

Native American Blood Lead Levels: No blood lead data are available for Coeur d'Alene Tribe members practicing subsistence lifestyles. Blood lead levels were not predicted for either the traditional or modern subsistence scenarios because extremely high estimated intake rates coupled with cultural-specific dietary and behavioral considerations invalidate current blood lead models. Nevertheless, projected intake rates are sufficiently high to indicate that blood lead levels associated with subsistence activities in the floodplain of the Lower Basin would exceed any current health criteria for children or adults in either scenario.

Potential Native American Lead Intake Rates: It is important to note that the high lead intake rates are associated with several media. Soil and sediment intakes, fish fillet and peeled water potato, and ingestion of disturbed surface water during swimming and bathing activities would each individually result in excessive lead intake. Consumption of whole fish from the Spokane River or un-peeled water potatoes from the Lower Basin would present especially dangerous intake levels. It is likely that background or pristine environmental concentrations would be required for all media to safely support Native American subsistence activities.

Cancer Risk due to Arsenic for the Coeur d'Alene Tribal Scenarios: The highest cancer risks are associated with Coeur d'Alene Tribal subsistence lifestyles. Table ES-2 shows the Reasonable Maximum Exposure (RME or 95th percentile) cancer risk for arsenic for the modern and traditional subsistence exposure scenarios for the combined adult/child age group. RME cancer risks exceeded EPA's acceptable 10^{-4} to 10^{-6} risk criteria in all exposure pathways, with cancer risks ranging from approximately 1×10^{-5} to 1×10^{-3}. Total RME cancer risk for the modern subsistence is approximately 7×10^{-4} and 3×10^{-3} for the traditional scenario, suggesting unacceptable cancer risks from exposure to arsenic through all media and pathways.

Cancer Risk due to Arsenic for the Resident Population: For the resident population, cancer risks were evaluated for children and adults (age 0 to 30 years). As shown in Table ES-3, total RME cancer risk for each scenario was in the range of 10^{-6} to 10^{-4}, except for the residential scenario at the Side Gulches where the RME cancer risk was 3×10^{-4} due to drinking water exposures at a private well.

Non-carcinogenic Risk for the Coeur d 'Alene Tribal Scenarios: Noncancer risks are expressed as a hazard quotient. If the value is less than or equal to 1, no adverse health effects are anticipated. Hazard quotients greater than 1 may be associated with adverse health effects. Summary hazard results for non-carcinogenic effects are also provided in Table ES-2. Risks and hazards for the Coeur d'Alene Tribe traditional subsistence scenario were the highest of any population. Modern and traditional subsistence exposures were evaluated only as future scenarios because subsistence lifestyles are not known to be currently practiced in the floodplain. Risks and hazards for the modern subsistence scenario were similar to those for the highest residential areas. For both subsistence scenarios, arsenic and iron in soil and sediment were the greatest contributors to noncancer hazard. Hazards from fish ingestion are likely underestimated for subsistence exposures because the whole fish may be eaten. Hazards are estimated using data for fish fillets that have substantially lower metals concentrations (e.g., an order of magnitude) than whole fish.

Non-carcinogenic Risk for the Resident Population: For typical CT (50th percentile) exposures to the resident population, potential unacceptable hazards occur only for future resident children and future child/adult residents of the Burke/Nine Mile area, if they were to use groundwater as a domestic supply. In general, the hazards and risks calculated for typical exposures were lower by approximately an order of magnitude compared to those calculated for RME (95th percentile) conditions. All other excess hazard quotients discussed are for RME conditions.

Non-lead RME Residential and Neighborhood Risks and Hazards: Under current conditions, the Side Gulches had the highest risks and hazards for the 0-6-year age group and the combined children and adults age group (Table ES-3). The Lower Basin had the second highest risks and hazards for these age groups. The Lower Basin had the highest concentrations of arsenic and iron in soil and sediment (except for waste piles). The higher risks and hazards in the Side Gulches were due to high concentrations of arsenic in water in one private well. The Burke/Nine Mile area had the highest neighborhood risks and hazards because of the waste pile exposures evaluated for this area. Waste piles had the highest concentrations of non-lead metals.

Non-lead RME Public Recreational Risks and Hazards: Of the 8 geographical areas evaluated, five had publicly developed recreational areas with sampling results. Hazards from the use of these areas exceeded 1 for the 0- to 6-year age group only along the lower Coeur d'Alene River from the confluence of the North Fork and the South Fork downstream to Harrison (Table ES-4). Cancer risks were highest for this area as well.

Non-lead RME Occupational Risks and Hazards (Construction Worker): Of the 8 subareas, five were evaluated for risks and hazards to construction workers actively engaged in work that involves soil disturbance. As with the other receptor groups evaluated, risks and hazards were highest in the Lower Basin, and the Lower Basin is the only area where combined hazards exceeded 1, with a hazard quotient of 0.5 for arsenic and 0.7 for iron (Table ES-5 and Table 5-1).

Non-carcinogenic Chronic Effects of Lead: The hazard quotients developed for non-lead metals should be considered as potentially underestimating noncancer risks due to additional exposures to lead. Lead is known to have adverse effects to many of the same organ systems of concern in the development of the hazard indices. Potential lead effects are not accounted for in these risk estimates, although substantial lead intake rates are anticipated for these populations.

Background Arsenic Levels: Cancer risks and non-carcinogenic hazards were calculated on the basis of total arsenic concentrations in each area. However, some of the arsenic is naturally present (pre-mining background concentration) and may be contributing significantly to the total arsenic concentration in soil and sediment. As a result, background levels may account for a significant percentage of the risk due to arsenic in some areas. In other cases, background does not add significantly to total arsenic risk.

Potential Preliminary Remediation Goals (PRGs) for Arsenic: PRGs provide useful targets when evaluating remedial alternatives, and are calculated by defining a target risk goal and then solving the basic risk equations for concentrations rather than solving for risk. Arsenic PRGs were calculated based on either cancer or non-cancer health effects because arsenic is the COPC that is most consistently a risk driver for all non-lead risk assessment scenarios. Potential residential PRGs based on exposure to arsenic in soil by ingestion and dermal exposure are: 35 mg/kg for children aged 0 - 6 years (non-cancer), 64 mg/kg for children and adults combined (cancer risk of 1 x 10-4), and 123 mg/kg for combined child and adult non-cancer exposures.

Responses to Comments - Public Review Draft **:** Numerous comments were received from several reviewers on the Public Review Draft of the HHRA released in July 2000. Appendix W contains a compendium of public comments received and the authors' responses. Also included as part of the response to comments are an independent peer evaluation of the comments received and the USEPA *Technical Review Workgroup for Lead (TRW)* evaluation of the HHRA. Enhanced discussion and additional analyses have been provided in several areas of the document.

Revised Dermal Exposure Estimates: Alternate exposure parameters were employed in assessing the dermal exposure route for arsenic and cadmium included in Section 5.0. These analyses resulted in a 15-20% reduction in estimated exposures to these metals by skin absorption.

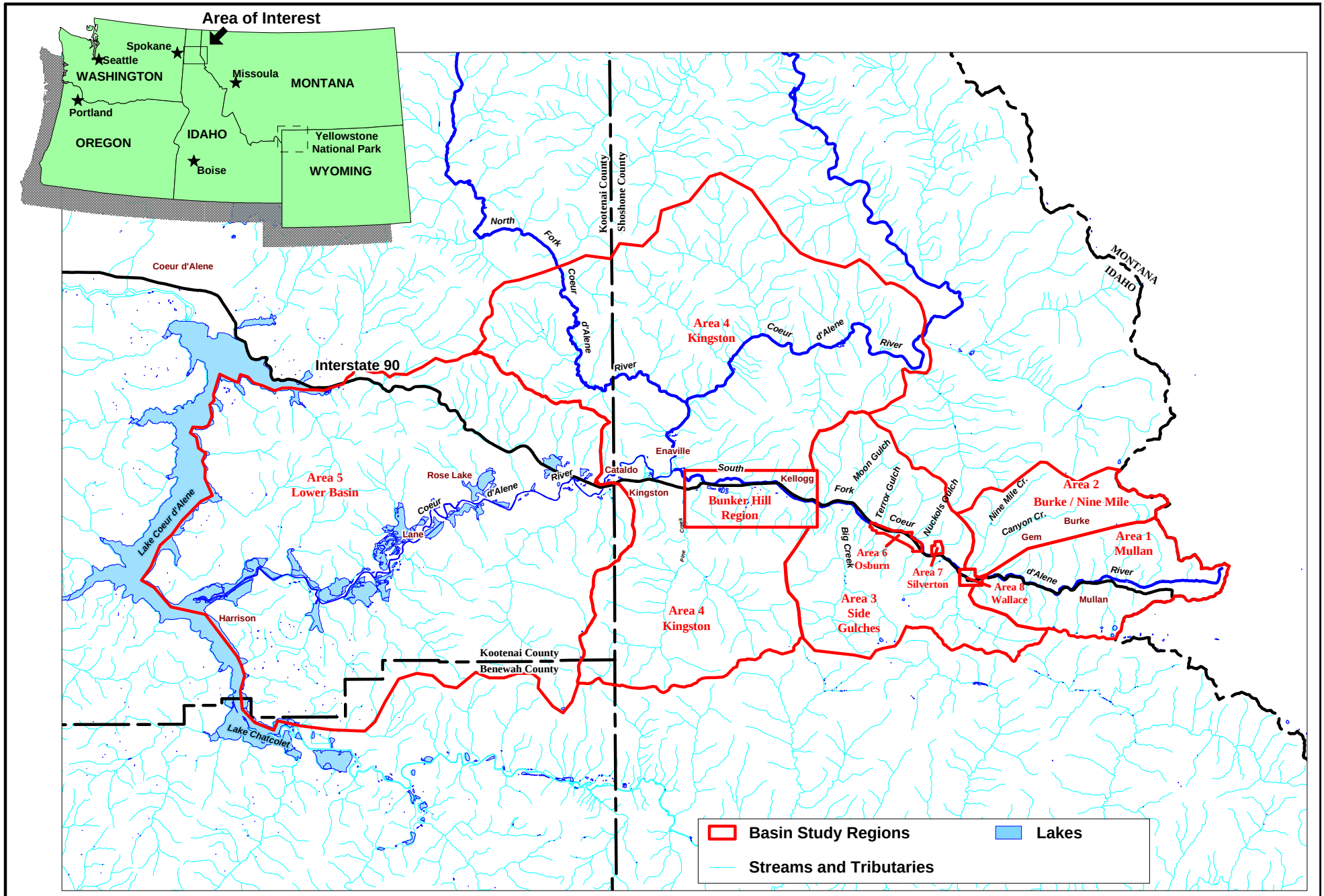
Revised Drinking Water Exposures: Drinking water lead exposures were revised to separate purged tap water samples (1997-1999 surveys) from home well source samples (1996 survey).

Additional Site-specific Blood Lead Data: Additional information regarding the blood lead data base used in the site-specific analysis was provided in Section 6.2.2. Data were provided indicating the number of repeat blood lead observations, the number of households participating in the surveys, and characteristic blood lead levels for those children providing more than one sample.

Lead-based Paint Hazard Analysis: Additional analysis of the lead paint hazard associated with observed high blood lead levels is presented in Section 6.4.2. About 30% of children exhibiting high

blood lead levels, for which this index was available, were from homes with a potential interior lead paint hazard. About 70% of the high blood lead children came from homes classified as having no interior lead paint hazard.

Clarification of USEPA Policy: Additional clarification of USEPA policy regarding the use of environmental media and observed blood lead data in human health risk assessment is provided in Section 8.12.



TerraGraphics Environmental Engineering, Inc.	FILE 1999-07-13 Basin RA / es-1	REQUESTOR L.Wichers	Coeur d'Alene Basin Human Health Risk Assessment	Figure ES-1
	DATE 07JUL2000	DESIGNER B. Bailey		Coeur d'Alene Basin
	PROJECT NO. 980111	CARTOGRAPHER B. Bailey		
		APPROVED L.Wichers		

Figure ES-2 Arithmetic and Geometric Mean Blood Lead Concentrations by Geographic Area - 9 Month through 9 Year Old Children (1996 - 1999 Combined)

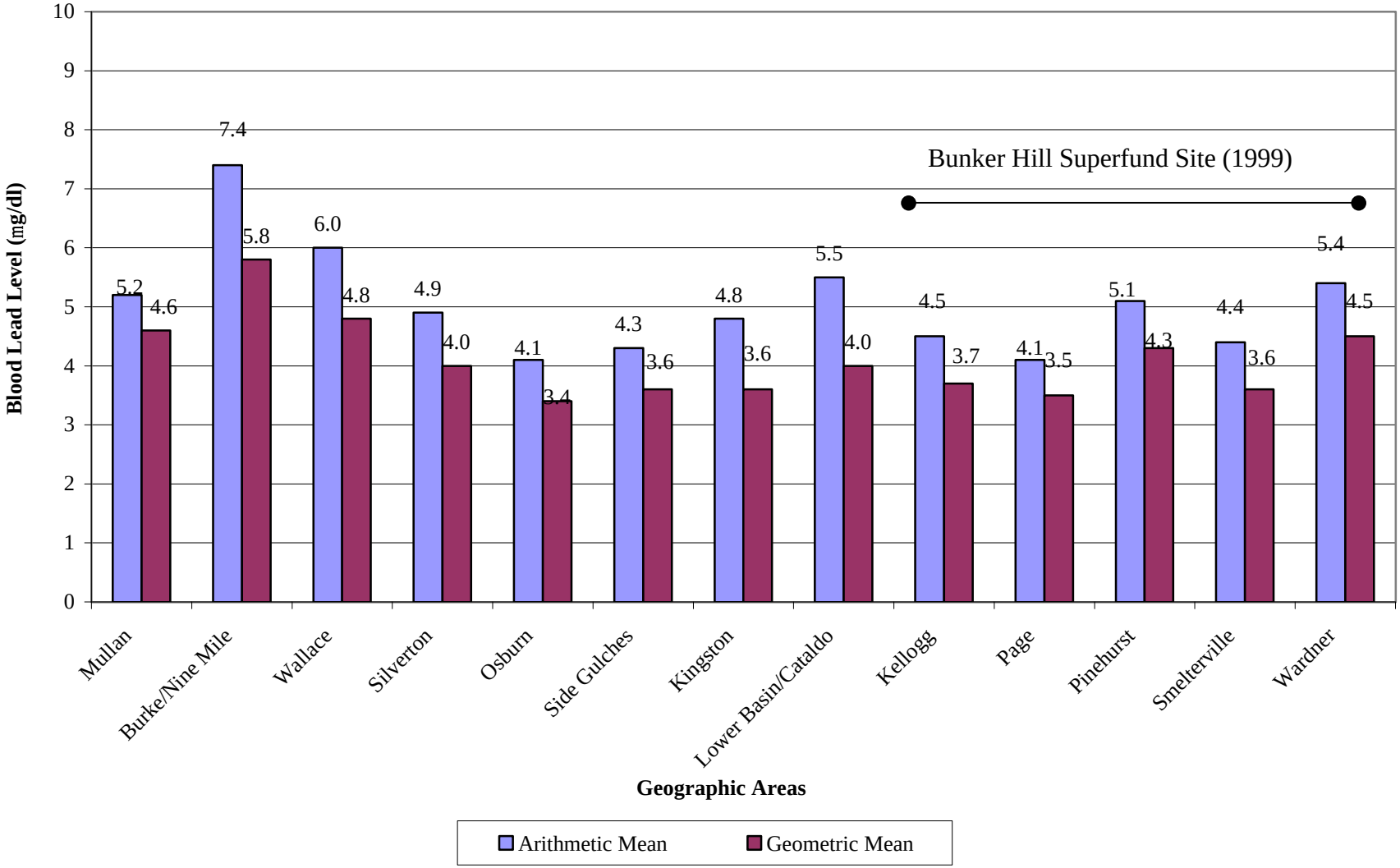


Figure ES-3 Basin Mean Blood Lead Levels by Age
(1996 - 1999 combined)

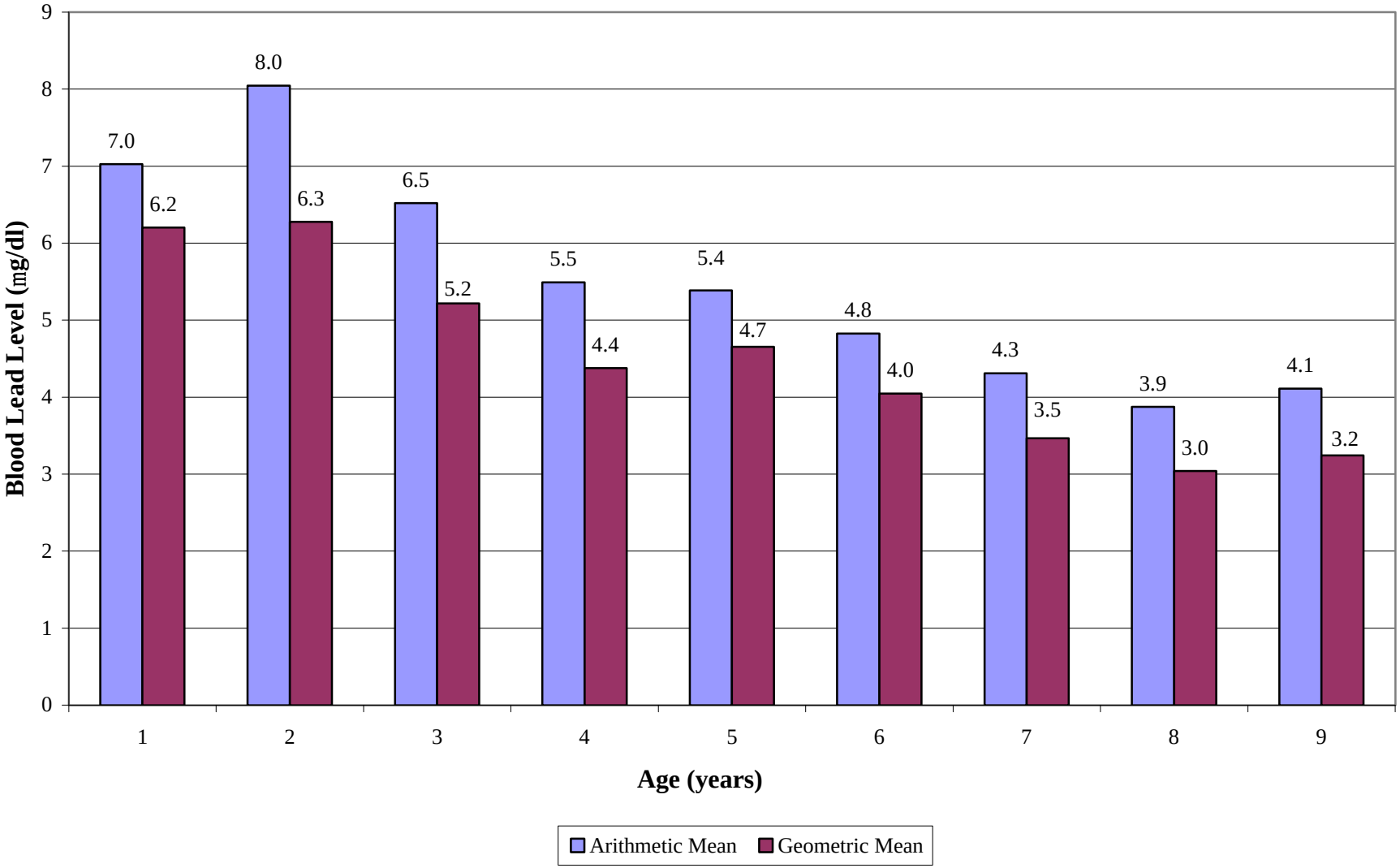


Figure ES-4 Percent to Exceed Blood Lead Concentrations by Geographic Area - 9 Month through 9 Year old Children (1996 - 1999 Combined)

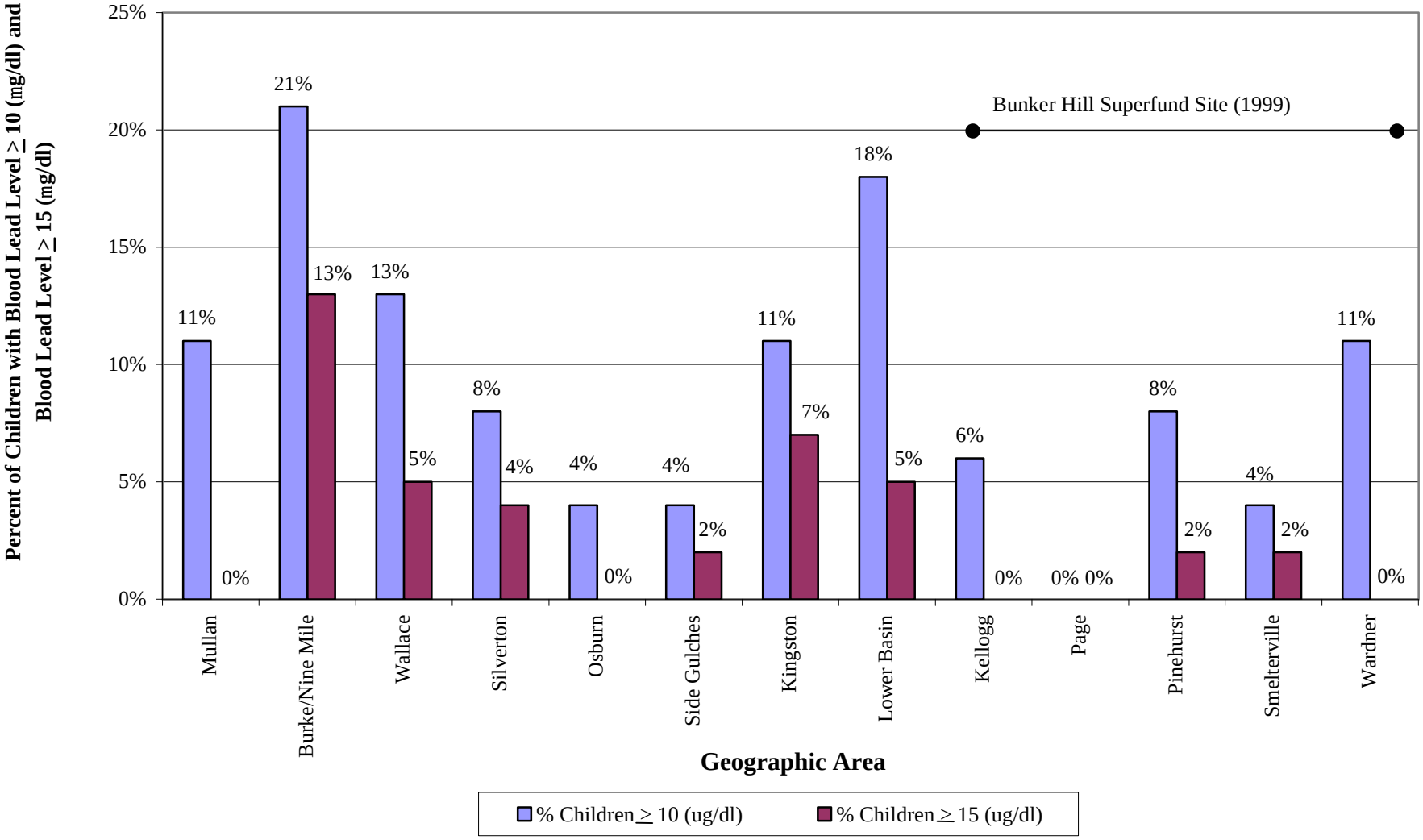


Figure ES-5 Percent of Children to Exceed Critical Toxicity Levels by Age (Basin-wide 1996 - 1999 Combined)

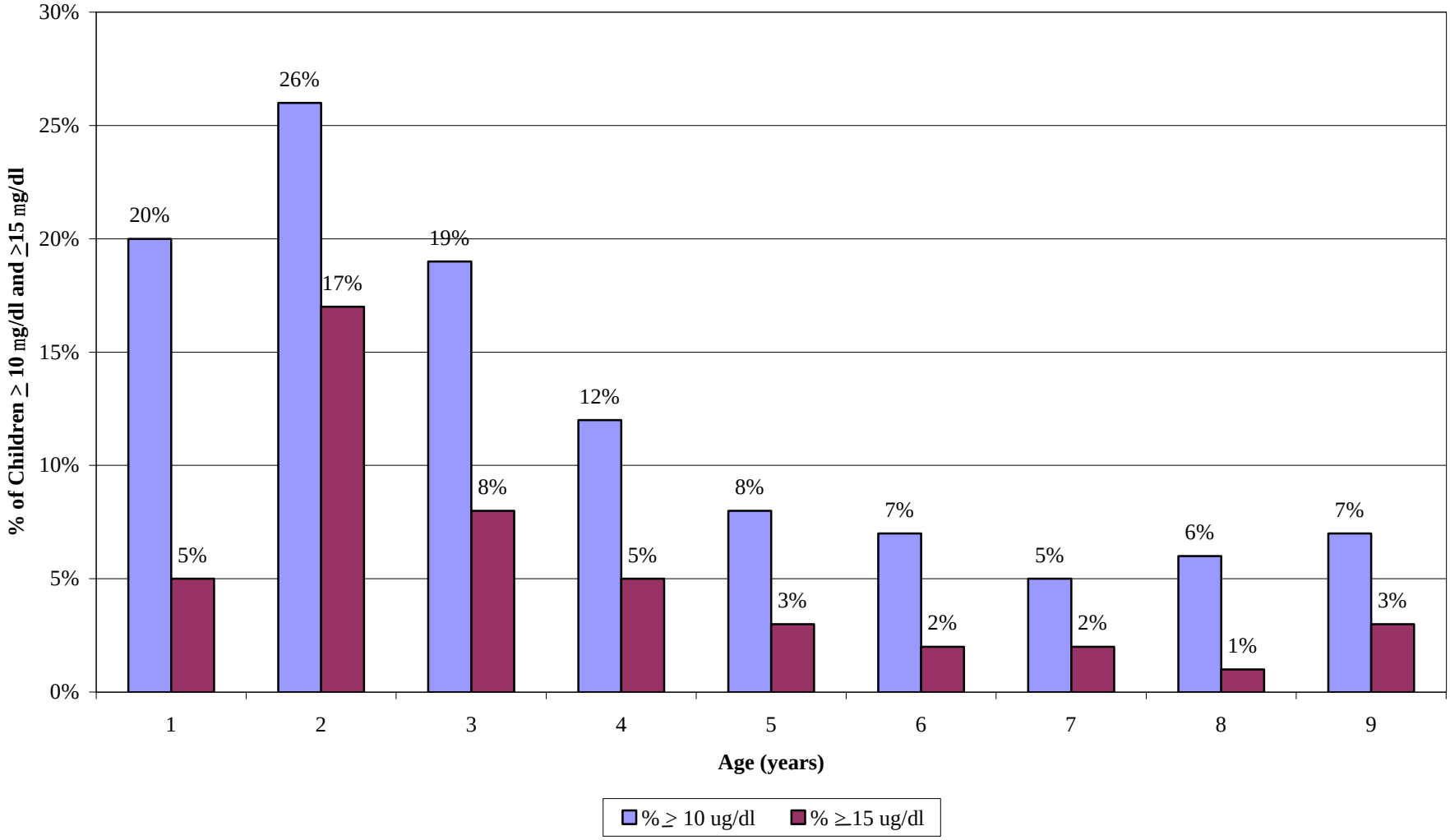


Figure ES-6 Observed and Predicted Geomean Blood Lead Levels for 0-84 Month Old Children Only - IEUBK Batch Mode

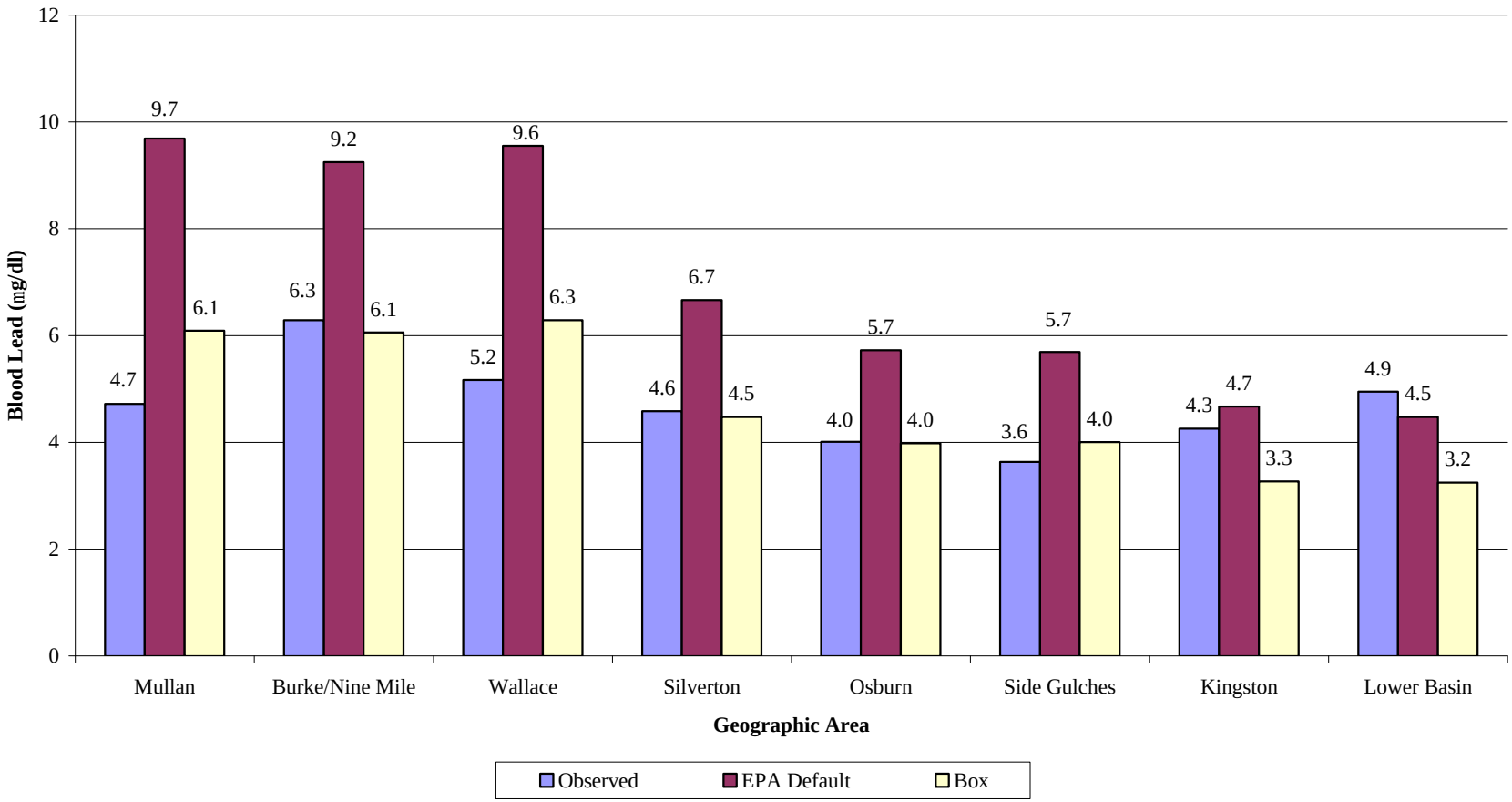


Figure ES-7 Observed and Predicted Percent to Exceed 10 mg/dl for 0-84 Month Old Children - IEUBK Batch Mode

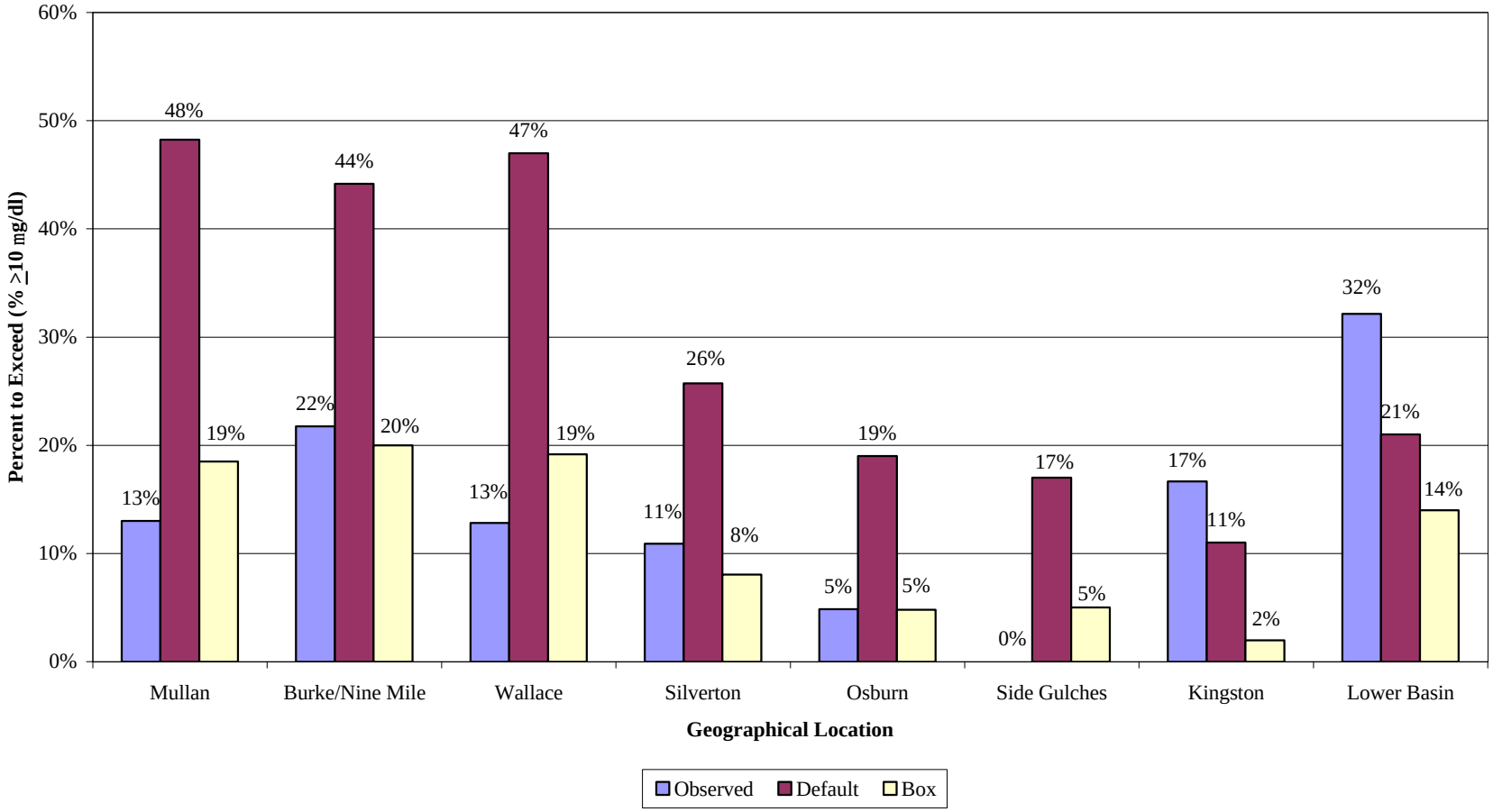


Table ES-1
Selected Chemicals of Potential Concern in Each Medium

Chemical	Soil/ Sediment	House Dust	Tap Water	Surface Water	Groundwater	Air	Fish	Vegetables
Antimony	X	X			X			
Arsenic	X	X	X	X	X			X
Cadmium	X	X		X	X		X	X
Iron	X	X						
Lead	X	X	X	X	X	X	X	X
Manganese	X	X		X				
Mercury				X			X	
Zinc	X	X			X			

Table ES-2
RME Hazard Quotients and Cancer Risks for Arsenic for Modern and Traditional Future Subsistence Exposure Scenarios

Age Group	Modern		Traditional	
	Hazard Quotient	Cancer Risk	Hazard Quotient	Cancer Risk
Adult	3	No arsenic data	10	No arsenic data
Child	9	Not evaluated	43	Not evaluated
Child/adult	4	7×10^{-4}	19	3×10^{-3}

Table ES-3
RME Hazard Quotients and Cancer Risks for Residential and Neighborhood Scenarios

Geographical Area	Children Age 0 to 6 Years[a]	Children Age 4 to 11 Years (Risks/Hazards From Neighborhood)		Children and Adults Age 0 to 30 Years	
	Hazard Quotient	Hazard Quotient	Cancer Risk	Hazard Quotient	Cancer Risk
Lower Basin	4	1	2×10^{-5}	1	1×10^{-4}
Kingston	2	1	3×10^{-5}	0.7	5×10^{-5}
Side Gulches	6	2	2×10^{-5}	2	3×10^{-4}
Osburn	3	0.3	5×10^{-6}	0.9	8×10^{-5}
Silverton	2	0.5	9×10^{-6}	0.7	5×10^{-5}
Wallace	3	0.6	8×10^{-6}	0.8	5×10^{-5}
Nine Mile, current conditions	3	1	4×10^{-5}	1	8×10^{-5}
Nine Mile, future conditions	22	Same as current	Same as current	12	3×10^{-5}
Mullan	3	0.4	4×10^{-6}	1	7×10^{-5}
Vegetables (all areas)	2	Not evaluated	Not evaluated	2	8×10^{-5}

[a]Cancer risks were not evaluated for the 0- to 6-year age group.

Table ES-4
RME Hazard Quotients and Cancer Risks for Public Recreational Scenario

Geographical Area	Children Age 0 to 6 Years[a]	Children and Adults Age 0 to 30 Years	
	Hazard Quotient	Hazard Quotient	Cancer Risk
Blackwell Island	0.7	0.2	1×10^{-5}
Lower Basin Soil/water risks/hazards	2	0.5	3×10^{-5}
Fishing in lateral lakes	Not evaluated	0.9	No arsenic data
Kingston (confluence of the North Fork and South Fork)	2	0.6	4×10^{-5}
Side Gulches	No public areas evaluated	No public areas evaluated	No public areas evaluated
Osburn	No public areas evaluated	No public areas evaluated	No public areas evaluated
Silverton	0.3	0.09	6×10^{-6}
Wallace	0.5	0.1	6×10^{-6}
Nine Mile (current conditions)	No public areas evaluated	No public areas evaluated	No public areas evaluated
Mullan	No public areas evaluated	No public areas evaluated	No public areas evaluated

[a]Cancer risks were not evaluated for the 0- to 6-year age group.

Table ES-5
RME Hazard Quotients and Cancer Risks for Occupational Scenario

Geographical Area	Adults (25 Years of Exposure)	
	Hazard Quotient	Cancer Risk
Lower Basin	2	8×10^{-5}
Kingston	0.5	3×10^{-5}
Osburn, Silverton, Wallace areas combined	0.4	2×10^{-5}
Nine Mile	0.4	2×10^{-5}
Mullan	0.5	2×10^{-5}

1.0 INTRODUCTION

The Coeur d'Alene Basin (CDAB) in northern Idaho has long been known to be contaminated by historical mining and smelting activity. Public health investigations in the 1970s to 1980s resulted in the designation of the 21 square mile area called the Bunker Hill Superfund Site (BHSS), or "the Box," surrounding the former ore refining complex near Kellogg. Recently, the United States Environmental Protection Agency (USEPA) has extended a Remedial Investigation and Feasibility Study (RI/FS) to include the larger area of contaminant release in the CDAB. This expansion resulted from the review of previous studies indicating areas outside of the original BHSS boundaries present a potential threat to human health and the environment. The RI is currently being undertaken to characterize the degree and extent of the contaminant release and the FS was initiated to select the most appropriate remedial action based on site-specific applicability, effectiveness, ability to implement , and relative cost. Concurrent with the RI/FS, the Human Health Risk Assessment (HHRA) is being conducted to determine potential health risks associated with residual heavy metals contamination in the CDAB. This document provides the HHRA for the areas east of Harrison upstream from the mouth of the Coeur d'Alene River.

1.1 SITE LOCATION AND BACKGROUND

1.1.1 Community and Industrial Development

The CDAB is a vast hydrologic drainage network of over 3700 square miles located in Shoshone and Kootenai Counties in Northern Idaho (Figure 1-1). The Coeur d'Alene (CDA) River flows west through the Basin for approximately 53 miles from the Idaho/Montana state border to Lake Coeur d'Alene which then drains to the Spokane River. It is estimated that as many as 10,000 people live in over 20 incorporated and unincorporated communities in the CDA Basin area (excluding the Bunker Hill Superfund Site and the city of Coeur d'Alene). Most of the communities included in the present study have developed at or near old mine portals and ore milling sites, or are adjacent to large mine waste (tailings) or contaminated alluvial deposits. Communities west of, and including, the city of Coeur d'Alene are not included in the present study as explained in Section 2.1, and more specifically in 2.1.5.

The Coeur d'Alene Basin including Lake Coeur d'Alene and the St. Joe, Spokane, and Coeur d'Alene River Basins was the ancestral home of the Coeur d'Alene Indian nation for centuries prior to the coming of European immigrants in the mid to late 1800s. Agricultural settlements developed around the Jesuit missions in the area during the mid 19th century. The existence of significant deposits of gold, silver, and lead was first reported in the Coeur d'Alene Mining District in 1882 (Day 1963). Subsequently, several mining towns developed in the upper Basin and these areas were extensively mined and became one of the largest and most productive lead, silver, and zinc producing areas in the United States, namely northern Idaho's Silver Valley. The Bunker Hill and Sunshine mines, near Kellogg, Idaho, were among the largest silver and lead producers in the United States, and several

mining companies are still active in the area today. Over 100 years of past mine waste and over 60 years of smelter emissions have been discharged into the CDAB.

The Upper Basin, for the purposes of the HHRA, is contained in a steep mountain canyon of the South Fork of the Coeur d'Alene River and adjacent tributary gulches. The Upper Basin contains 11 residential cities or unincorporated areas, about half of which are located within the BHSS. This area is the heart of the world famous Coeur d'Alene mining district that was a major producer of silver, lead, zinc and other metals throughout the last century.

The 21-square mile Bunker Hill Superfund Site (BHSS) (National Priority Listing - September 8, 1983) is located on the South Fork of the CDA River near the center of the upper Basin. The communities of Kellogg, Smelterville, Wardner, Page, and Pinehurst are located within the site boundaries (Figure 1-1). Two Records of Decision (RODs) have been completed for the BHSS (one for the populated areas in August 1991 and a second for the non-populated areas in September 1992) (USEPA 1991c, 1992b). When the site was in full operation, it included: a milling and concentrating operation, a lead smelter, a silver refinery, a cadmium plant, an electrolytic zinc plant, a phosphoric acid and phosphate fertilizer plant, two sulfuric acid plants, a 160-acre tailings impoundment, and several hundred acres of heavy metals contaminated soils (JEG 1988, TerraGraphics 2000a). Major products of the Bunker Hill operation were lead, zinc and zinc alloys, zinc oxide, cadmium, silver, hard (antimonial-arsenical) lead, gold ore, copper matte, cobalt, sulfuric acid, nickel, phosphoric acid, and four grades of fertilizer (BCI 1992). During the 1960s and 70s, Bunker Hill supplied approximately 25% of the primary lead refined in the United States with a daily production capacity of over 300 tons of metallic lead. Extensive remedial actions and lead health assessments have been accomplished at the BHSS. The recent Five Year Review of the BHSS Project provides a detailed summary of those activities (TerraGraphics 2000a).

The Lower Basin area includes 11 lateral chain lakes and extensive wetlands, located adjacent to the main channel and within the CDA River's floodplain. These marshes and lakes provide an extensive recreational area between the town of Cataldo and Lake Coeur d'Alene. Camping, fishing, boating, swimming, hunting, and wildlife photography/observation are popular activities throughout the lower CDAB. There are no incorporated cities between Cataldo and Harrison at the mouth of main River. However, there are a few small unincorporated village areas and several rural residences.

Cataldo Mission Flats, located near the historic Cataldo Mission, was originally a Tribal farm consisting of native hay meadows and pasture. Mine tailings, and effluent from other sources, were deposited in the river bed adjacent to the flats as the mining industry developed in the upstream basin. Sediments eventually inhibited river boat navigation to the Mission, and were subsequently dredged and deposited on the flats. Such dredging ceased in 1930, when river boat navigation was discontinued. During the early twentieth century, reworking of the metal-rich sediments near Cataldo resulted in significant disturbance of the CDA River Basin floodplain (USGS 1990).

By the 1950s, mine tailings piped from the river covered 2,000 acres of the Cataldo Mission Flats to an average depth of twenty-five to thirty feet. Sediment dredging, pumping 7000 gallons of water per minute, and excavating some 500 tons of contaminated river sediments per day, continued until 1968. Approximately 72 million tons of this sediment contaminated with mine tailings have been discharged into the CDA River (Krieger 1990, Weston 1989).

Approximately forty acres of the sediments deposited on the Cataldo Flats have not developed a stable vegetation cover. These barren areas, typified by milled ore deposits, are subject to drying during the summer months. Off-road vehicle enthusiasts use the barren areas for recreation, further retarding vegetation and increasing their own risk of heavy metals exposure. Under these conditions, winds entrain the highly mobile fine materials to such a degree as to occasionally reduce driving visibility.

The materials deposited on the flats are a heterogeneous mix ranging from milled ore, high in zinc, lead, and cadmium, to typical river sand and gravel. Alluvial sediments throughout the CDAB have extremely high concentrations of arsenic, lead, cadmium, zinc, and other trace elements (Krieger 1990). The sediments act as both a sink and a source of contamination, and their transport affects the concentrations of heavy metals in the soil, biota, surface water, and groundwater. High water flows, especially during winter and spring, scour sediments from the banks and bottom of the river and transport elevated quantities of metals downstream. When the flows decrease, the metals accumulate in the slow-moving sections of the river, in the lateral lakes, and downstream in Lake Coeur d'Alene. Sediments deposited on the floodplain become contaminated soils. Heavy metals contamination extends throughout the river system, including approximately 30 miles of the lower CDAB, the lateral chain lakes, and the northern two-thirds of Lake Coeur d'Alene (Haness 1991).

1.1.2 Public Health Concerns

The Bunker Hill Company mining and smelting complex closed in 1981. The site was added to the National Priorities List (NPL) in 1983, and the subsequent Lead Health Study was conducted jointly by state, federal, and local health agencies the same year (PHD 1986). This comprehensive survey of lead poisoning and exposures in the community showed elevated levels of lead in blood among area children, including those born after the smelter closure. Since the early 1970s, the exposure pathways and human health impacts associated with exposure to heavy metals have been studied extensively at the BHSS. Over the past 15 years, over 4,000 children living within the site have been tested for blood lead levels. Up to 75 percent of the preschool children tested (throughout the 1970s and early 1980s) had elevated blood lead levels ($10 Fg/dl) (JEG 1988). Blood lead levels of BHSS children in the early 1980s averaged around 15 Fg/dl, while today, the average blood lead level of children is near 4 Fg/dl. About 6% of children have blood lead levels $\geq$10 Fg/dl (TerraGraphics 2000a).

Residual contamination in community soils and dusts was identified as the primary source of lead exposure to children. The primary route of exposure has been identified as incidental ingestion of soils and dusts by ordinary hand-to-mouth and play activities (TerraGraphics 2000a). These same exposure

pathways potentially exist for individuals throughout the CDAB. Soil lead values near the river downstream of the Bunker Hill site typically range from 2,000 to 12,000 mg/kg while those in the upper Basin range from 500 to 25,000 mg/kg. Generally, soil samples average 2500-2800 mg/kg lead throughout the CDA River Valley (Neufeld 1987, Haness 1991, Lustig 1991).

In 1991, the Center for Disease Control's (CDC's) intervention level of 25 Fg/dl of lead in blood for children was revised downward to 10 Fg/dl. In response to reductions in the health intervention level in children, the geographic area of human health concern surrounding the Bunker Hill Site has continued to expand. However, minimal testing of residents upstream and downstream from the site for lead and other heavy metals had been done. Children tested in the early 1970's, living outside the boundaries of the BHSS, often exhibited blood lead levels of 40 to 50 Fg/dl. No organized screening occurred beyond the site boundaries from 1975 until 1996.

In addition to CDC's concern about blood lead levels in children, the Agency for Toxic Substances and Disease Registry (ATSDR) expressed concerns as part of a Health Consultation done on the Coeur d'Alene River in June, 1991. That health consultation indicated that contamination within the Basin was not well characterized and that it may represent a threat to public health. It was recommended that periodic monitoring of soil and sediments occur where human contact is likely, that persons who have frequent contact with contaminated soils and sediments be identified, and that the use of untreated drinking water from the river or a shallow aquifer be discouraged.

In 1996, the State of Idaho, the Panhandle Health District (PHD) and ATSDR began consideration of the entire CDA River Basin for health-related concerns similar to those of the BHSS. The reason for this concern was based upon known historical mining practices in the BHSS site, the CDCs blood lead action level, and the Health Consultation accomplished by ATSDR. Additionally, fate and transport studies at the BHSS site indicated that metals contamination had spread from the site along the Coeur d'Alene River, into the chain lakes area, into Lake Coeur d'Alene, and possibly into the Spokane River (SAIC 1990). A large-scale, multimedia sampling study within the Basin was performed in 1996 by the State of Idaho, USEPA, and ATSDR (IDHW 1999).

To better define the nature and extent of the contamination in the Basin, EPA Region X began additional RI/FS data collection activities that are currently ongoing through the federal contractors URS Greiner and CH2M Hill. The expansion of the region of concern from the BHSS to the greater Coeur d'Alene Basin requires that a Human Health Risk Assessment (HHRA) be performed for the Basin. It is important that the HHRA be conducted comprehensively, within the context of the history of lead health problems in the region, and be consistent with ongoing health intervention actions at the BHSS. The State of Idaho, in conjunction with Region X, has performed the HHRA for the Basin, while Region X will coordinate other RI/FS activities for the Basin. It is the desire of the State to coordinate all HHRA activities with ongoing RI/FS activities, in a manner consistent with USEPA Risk Assessment Guidance.

The ATSDR and the Washington Department of Health have independently conducted both Health Consultations and issued Health Advisories for the Basin area in the last two years. The Idaho Department of Health and Welfare, Division of Health (IDOH) requested technical assistance from ATSDR in 1998 to evaluate the likelihood that the levels of lead, mercury, and cadmium detected in fish caught in the Coeur d'Alene lateral lakes would result in adverse health effects if these fish were eaten (ATSDR 1998). Additionally, the USEPA requested ATSDR to conduct an independent review of the environmental sampling data from 47 Common Use Areas (CUAs) and 80 residential properties in the Coeur d'Alene Basin in the Spring of 2000. The purpose of the health consultations was to assess the potential for public health hazards based on environmental data. A public health hazard is defined by ATSDR as sites that pose a public health hazard due to the existence of long-term exposures (> 1 year) to hazardous substances or conditions that could result in adverse health effects (ATSDR 2000a).

In evaluating the fillet fish data for northern pike, bullhead catfish, and yellow perch, ATSDR concluded that adverse health effects are not likely to occur from ingestion of fish from the lateral chain lakes and other sources of lead are present in the Coeur d'Alene Basin that may affect residents if chronic exposures occur (ATSDR 1998).

Specifically, ATSDR was asked to assess whether Early Action Levels (EALs) proposed by the USEPA to remediate certain homes and CUAs were protective of public health. Based on the information available for the 47 CUAs east of Harrison, ATSDR found that the EALs proposed for antimony, arsenic, cadmium, and lead may not provide an adequate margin of safety for area residents. ATSDR also concluded that recreational visitors to the CUAs are not likely to experience adverse health effects from metals below the EALs. The recommendation that ATSDR provided in the health consultation was to notify area residents of the hazards posed by recreational activities at the CUAs along the Coeur d'Alene River (ATSDR 2000b).

The health hazards from lead contamination in soil, indoor dust, and tap water to children using the residential homes data were evaluated differently than the CUAs. The potential health threat was evaluated three ways, by 1) calculating an estimated daily intake (dose) and comparison to an Intake of Concern for the population (IOC), 2) estimating expected blood lead levels through use of the EPA's Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead, and 3) estimating blood lead levels through an ATSDR integrated exposure regression analysis model for use at lead sites. Based on the three methodologies utilized in the health consultation and data from the residential homes, a public heath hazard may exist for children living at more than half of the residences sampled by the USEPA in 1998 and 1999. Children in approximately 50 homes had estimated lead exposures twice the IOC and/or estimated blood lead levels in excess of the CDC action level of 10 µg/dl. Results of the evaluation suggest that children one to two years old may be the population of concern for elevated blood lead levels. Increased hazard may also be likely if other routes of exposure unaccounted for in the calculations, such as lead based paint, consumption of biota, and recreational activities in the Basin, are a significant route of exposure to lead. ATSDR made recommendations including differing

intervention strategies based on the level of risk that included continuation or initiation of blood lead monitoring and the current intervention program (ATSDR 2000a).

In addition to the published ATSDR health consultations for the Coeur d'Alene Basin, the Spokane Regional Health District, Washington State Department of Health, and the Washington State Department of Ecology recently issued a health advisory for consumption of Spokane River fish (June 2000). The advisory concluded that:

- all of the fish sampled in the Spokane River had elevated lead concentrations (rainbow trout, mountain whitefish, and large-scale sucker),
- lead levels in whole fish were significantly higher than levels found in fillets,
- no increased risk exists for most people who eat fillets from fish caught in the Spokane River,
- children should not eat whole fish or any meals prepared using whole fish, and
- adults (in particular, pregnant women) should limit the number of whole fish eaten.

1.2 STUDY POPULATIONS AND STUDY AREA

The study area is defined as the CDA Basin, which includes the South Fork of the Coeur d'Alene River and tributaries, and the main stem of the CDA River for approximately 53 miles from the Idaho/Montana border to Lake Coeur d'Alene, excluding the 21 square-mile Bunker Hill Superfund Site. The population includes those individuals living throughout the defined study area, but is limited to those individuals living within the CDAB at the time health and environmental surveys were performed (i.e., 1996-1999). For the blood lead surveys, children are defined as individuals older than 6 months, but less than or equal to 9 years of age. Sub-categories of this age group, (up to 84 months of age) including infants, preschoolers and toddlers are used throughout the HHRA to assess risks to children using the EPA Lead Model (USEPA 1994d; USEPA 1998f). Adult females are defined as women of reproductive age between the ages of 17 years and 49 years. The remainder of the population is comprised of all individuals that do not fit these categories.

It is estimated that approximately 10,000 people reside in about 5000 homes located in the defined area. A comprehensive review of the population and demographics of the CDAB is discussed in Section 3.0.

1.3 PURPOSE AND OBJECTIVES

The main purpose of this HHRA is to determine the extent of heavy metal contamination in environmental media that may expose current or future residents or visitors to the CDAB, to evaluate the potential human health risks associated with exposure to those contaminated media, and to provide information for risk managers to evaluate need for remedial action and development of associated clean-up criteria.

The specific objectives of this document are:

1. To review and summarize health and environmental data available for the CDAB,
2. To select the chemicals of potential concern (COPC),
3. To identify potentially exposed residents and visitors, exposure pathways and populations of concern,
4. To summarize toxicity information for the COPCs,
5. To characterize human health risk related to lead and other metal exposures,
6. To evaluate potential risk-based clean-up action levels, and
7. To identify uncertainties associated with the entire HHRA process.

1.4 SCOPE AND LIMITATIONS

The baseline risk assessment is an evaluation of the potential threats to public health from site contaminants in the absence of any remedial action (the no action alternative), as defined and required by Section 300.430(e)(6) of the National Oil and Hazardous Substances Pollution Contingency Plan (NCP). The results of the baseline risk assessment are used to determine the need for remedial action and to establish risk-based remediation goals.

The primary tasks in performing a risk assessment include data collection, data evaluation, exposure assessment, toxicity assessment and risk characterization. In addition, scoping and development of conceptual site models (CSMs) were performed so that the ultimate goal of risk assessment, assessing the current situation, also provides baseline information for instituting risk management measures to protect human health.

For the purposes of this HHRA, the CDAB is defined to be the area from the Idaho-Montana border to Harrison. The 21 square-mile BHSS is excluded from this assessment. Other areas, such as regions south of Harrison, Blackwell Island, Corbin Park beaches, and other areas identified by the State, EPA and the Coeur d'Alene Tribe, are also included as part of this HHRA.

1.5 REPORT STRUCTURE

This report is structured to follow the general Human Health Risk Assessment process.

Section 1.0 Introduction provides a brief description of community and industrial development in the Basin area, a short history of health concerns related to mining pollution, the study area, and the purpose, objectives, and limitations of the report.

Section 2.0 Data Evaluation discusses the organization of the RI/FS process and the data that is used throughout the HHRA. The data are organized by geographic area and media. Data quality evaluations are provided to assess the reliability of the information used in the assessment. Chemicals of potential

health concern (COPCs) are identified through a screening process comparing available media-specific concentrations to health related criteria. Eight metals were selected for further evaluation in this process. Those metals are antimony (Sb), arsenic (As), cadmium (Cd), iron (Fe), lead (Pb), manganese (Mn), mercury (Hg), and zinc (Zn).

Section 3.0 Exposure Assessment identifies those population groups that are of special concern for potential human health effects associated with the COPCs . Generally, longtime local residents are of concern with respect to carcinogenic and chronic non-carcinogenic health effects due to most of the metals. Women of reproductive age, as they represent the fetus, and children, are of most concern for lead. Exposure pathways, or the routes and mechanisms through which people contact and take these metals into the body are identified. Formulae are developed to quantify these intakes by identifying and selecting appropriate values for exposure factors, such as how much time children spend in particular play areas and how much soil they consume during those activities. Exposure point concentrations (EPCs) are developed that estimate the concentration of each contaminant in each media that people contact. These results are then combined to estimate characteristic intakes or how much of each contaminant is taken into the body by members of the sensitive population groups. Intake calculations are developed for the non-lead chemicals, or those with chronic or long-term health concerns in this section. Sub-chronic intake rates for lead are developed in Section 6.0.

Section 4.0 Human Health Toxicity Assessment discusses the health concerns associated with each of the COPCs and the routes of exposure and toxicological mechanisms that can lead to adverse health effects. Critical toxicity criteria such as no observable effect or “safe” levels are identified and discussed.

Section 5.0 Risk Characterization for Non-Lead Chemicals assesses the health risks associated with the characteristic intakes developed in Section 3.0 by comparison to the toxicity criteria identified in Section 4.0. This is accomplished for both carcinogenic (cancer causing) and non-carcinogenic health effects and for the combined effects of metals with common health effects endpoints. Risks are evaluated for both the resident population, those that currently live in the area, and for the Native American population that traditionally, or may in the future, practice subsistence lifestyle in the Lower Basin.

Section 6.0 Characterization of Lead Health Risks is accomplished separately from the other contaminants of concern for a number of reasons. These factors are discussed and observed blood lead levels from health department surveys conducted throughout the Basin are summarized with the results of special follow-up investigations of lead poisoned children. Site-specific quantitative analysis of exposure survey data relating observed blood lead levels to measured environmental variables at individual residences is accomplished. Lead exposure pathways are identified and quantified through route-specific exposure factors and intake routes are quantified. Predictive blood lead modeling, using both USEPA recommended and site-specific exposure factors, is accomplished and compared to observed results. These models are then used to project blood lead levels for current and future use

scenarios. Native American subsistence intake rates are also developed and compared to similar rates in other populations.

Section 7.0 Uncertainties in Risk Assessment discusses the significance of the results of the risk characterization and those factors that may lead to possible overestimation or underestimation of risk. Uncertainties are discussed relative to development and use of the HHRA database, the non-lead estimates of risk developed in Section 5 and the observed lead health problems and risk calculations developed for lead in Section 6. Concerns that interest groups may have and those factors that risk managers may want to consider in the development of risk reduction strategies are included in this section.

Section 8.0 Summary and Conclusions is developed as a stand alone chapter that is available for public review. This section summarizes the entire document, repeats important findings from previous sections, and discusses the results of conclusions of the risk assessment.
Section 8 is also being distributed publically as an abbreviated form of the risk assessment for those less interested in the details of the document. An **Executive Summary** is also available that briefly summarizes the major findings of the risk assessment.

Appendices The Appendices for this document are voluminous and are produced only in electronic format on CD. The CD is attached to the back cover of the report.

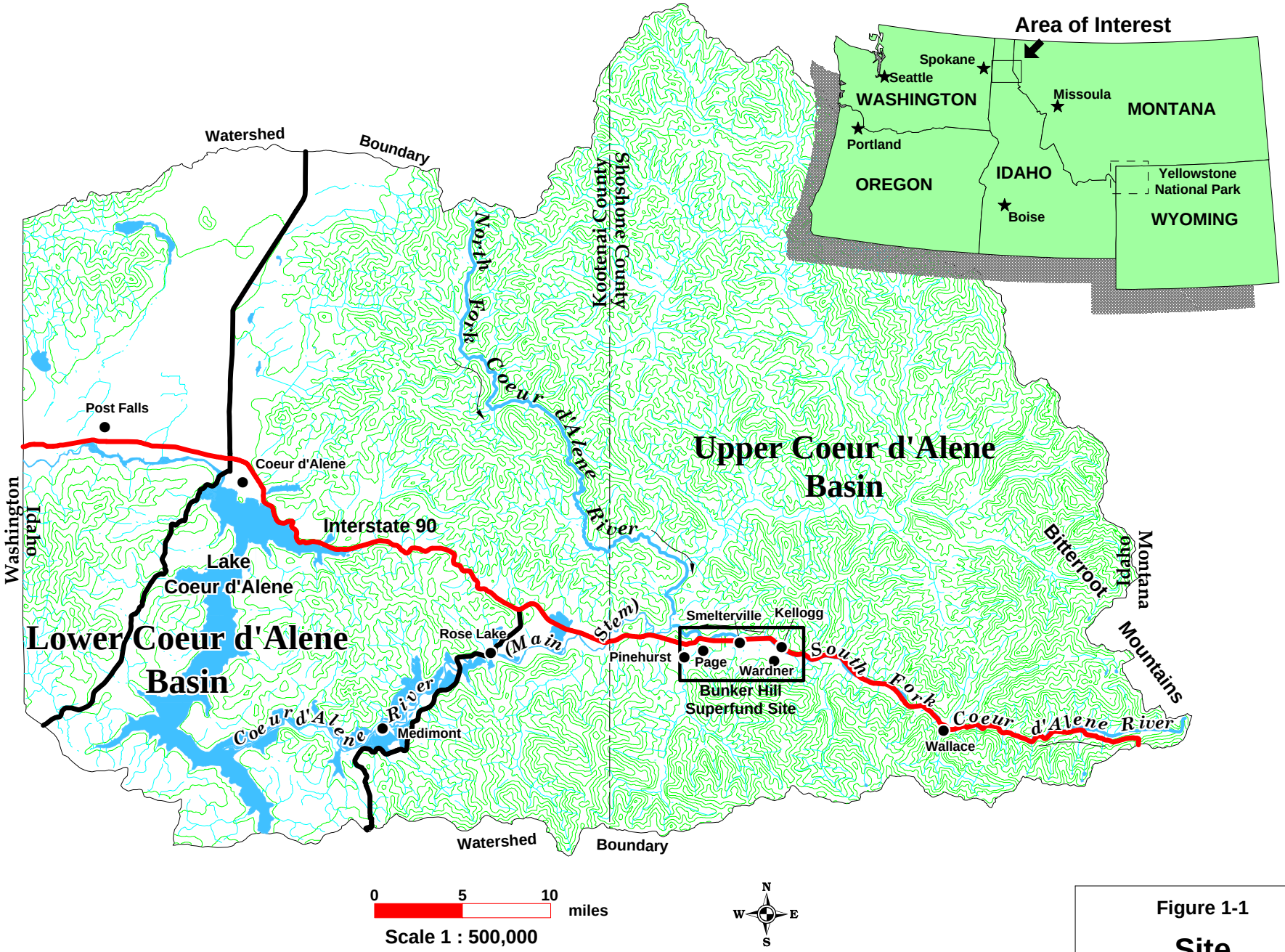


Figure 1-1

Site Location Map

TerraGraphics Environmental Engineering, Inc.

- Towns
- Interstate Highways
- Major Highways
- Rivers and Streams
- Elevation Contours

2.0 DATA EVALUATION

Samples of soil, house dust, tap water, groundwater, homegrown vegetables, sediment, surface water, fish, and plants (i.e., water potatoes) have been collected in the Coeur d'Alene River Basin. Because of the large quantity of analytical data available, the data were organized into a form appropriate for the baseline human health risk assessment (HHRA) according to the following procedure:

1. The data were sorted by medium and geographical area.
2. A baseline HHRA data set was developed.
3. The methods used for sample analysis were evaluated.
4. The data quality was evaluated with respect to sample quantitation limits, qualifiers and codes, and blanks.
5. Chemicals of Potential Concern (COPCs) were identified using a decision process that included a comparison of detected chemical concentrations with screening values (SV).

2.1 BASIN GEOGRAPHICAL AREAS

For the purpose of the remedial investigation/feasibility study (RI/FS), the Coeur d'Alene Basin has been divided into five geographical areas, called conceptual site model (CSM) units. Because these divisions were developed for use in the RI and Eco RA, and not for the HHRA, not all of these divisions are applicable to the HHRA. Therefore, these divisions were further broken down into geographic subareas to more clearly reflect human exposure areas. The following section discusses each of the five CSM Units and briefly describes the geographic subareas associated with each CSM Unit. These geographic subareas and the specific human exposure areas evaluated within each subarea are discussed in further detail in Section 3 and Appendix N. The purpose of this section is to provide consistency between the HHRA, the Eco RA, and the RI.

The Coeur d'Alene Basin has been divided into the following five CSM Units:

- CSM Unit 1: Upper Watersheds (including Beaver Creek, Big Creek, Canyon Creek, Moon Creek, Nine Mile Creek, Pine Creek, Prichard Creek, and Upper South Fork Coeur d'Alene River [from here on referred to as Upper South Fork]),
- CSM Unit 2: Midgradient Watersheds (including South Fork Coeur d'Alene River [from here on referred to as the South Fork], North Fork Coeur d'Alene River [from here on referred to as the North Fork], and Coeur d'Alene River),
- CSM Unit 3: Lower Coeur d'Alene River and Flood plain,
- CSM Unit 4: Coeur d'Alene Lake, and
- CSM Unit 5: Spokane River.

These CSM units were defined as part of a Basinwide CSM developed by the Ecological Risk Assessment Work Group (CH2M HILL 1998). Each CSM unit is further broken down into stream

segments based on stream drainage areas and morphology. Not all watersheds and/or stream segments have human populations, nor do they all have significant contamination. CSM Units 1, 2, and 3 were selected as applicable to this baseline HHRA. Human health concerns in CSM Unit 4 were addressed in the *Expedited Screening Level Risk Assessment for Common Use Areas* (provided as Appendix B), with the exception of Blackwell Island, which is included in this baseline HHRA, and Harrison Beach, which is included in the discussion of CSM Unit 3 (Section 2.1.3). CSM Unit 5 (Spokane River) has been evaluated separately. Maps showing the stream segments in CSM Units 1, 2, and 3 are provided in Figures 2-1 and 2-2. Because much of the sample data for the Basin have been segregated by watershed, the initial evaluation of available data is discussed within the context of the CSM units for the purposes of selecting data and COPCs. In Section 3, new geographical areas are selected that are more applicable to human health.

Appendix C provides an evaluation of potential exposure to residents for each stream segment. Table 2-1 provides a summary of the CSM units, geographical subareas, and segments, and indicates whether relevant data have been collected and included in the baseline HHRA for that segment. The following sections discuss each of the CSM units in more detail.

2.1.1 CSM Unit 1

Twelve of the 24 stream segments in CSM Unit 1 have not been included in the baseline HHRA. The following excluded stream segments include:

- Those minimally impacted by mining activities (as determined by the number of source areas within and upstream of the segment) and thus likely to have low contaminant concentrations (segments listed in Table 2-1 as “relatively uncontaminated”),
- Those at which there are no residential populations and where little or no routine recreational use is anticipated, and
- Those relatively inaccessible to humans (e.g., limited roads and difficult terrain).

No EPA data were available for 10 of the 12 excluded segments. These segments are in areas that fit the exclusion criteria listed above (i.e., they have been minimally impacted by mining and have minimal residential or recreational populations); therefore, the lack of data does not constitute a data gap for the HHRA. There are non-EPA data available for many of these segments; however, these data are not applicable to the HHRA for the reasons stated. Figure 2-1 shows the stream segments located in CSM Unit 1. The segments excluded from the baseline HHRA are shown with the segment name underlined. The data collected in CSM Unit 1 that is included in this HHRA falls within the geographic areas of Mullan, Burke/Nine Mile, and portions of the Side Gulches and Kingston. These areas are further defined in Section 3.

2.1.2 CSM Unit 2

Two of the four stream segments in CSM Unit 2 have been included in the baseline HHRA. MidGradSeg03 was excluded because it comprises the North Fork and is minimally impacted by mining. MidGradSeg02 was excluded because it consists mainly of the 21-square-mile area referred to as the Bunker Hill Superfund site, which is being addressed in another investigation. (See Appendix C for a detailed summary of the segments.) The geographic subareas that are encompassed in CSM Unit 2, as outlined in Section 3, are Wallace, Silverton, Osburn, and part of the Side Gulches and Kingston.

2.1.3 CSM Unit 3

All six segments located in CSM Unit 3 have been included in the baseline HHRA. Harrison Beach (considered part of CSM Unit 4 in Appendix B) has been included with CSM Unit 3 in the baseline HHRA because it is located near the mouth of the lower Coeur d'Alene River, in LCDRSeg06 (Figure 2-2), and the metal concentrations in sediment at Harrison Beach are similar to those at the other locations in CSM Unit 3. The data from CSM Unit 3 is considered in the Lower Basin subarea.

2.1.4 CSM Unit 4

The expedited screening level risk assessment for common use areas (Appendix B) examined beaches around Coeur d'Alene Lake that are used by the public. None of the beaches had metal concentrations greater than the levels of potential concern for human health, with the exception of Blackwell Island. Concentrations of metals in sediment on Blackwell Island warranted further evaluation of this area in the baseline HHRA. All data from Blackwell Island were previously screened in the expedited risk assessment (Appendix B); therefore, data from Blackwell Island are not included in the selection of COPCs in this section. The COPCs selected in Section 2.5 are evaluated at Blackwell Island in subsequent sections of the report (Section 3) and are grouped in the Lower Basin geographic subarea. Although, Lake Coeur d'Alene sites were screened out as an area of concern for the general population, there may be a concern with tribal populations consuming fish. Concern for potential health threats associated with tribal fish consumption are warranted for the following reasons:

- Tribal consumption rates of fish caught from Lake Coeur d'Alene are expected to be higher than other groups fishing the Lake.
- Tribal members traditionally consumed whole fish which can have concentrations of metals approximately an order of magnitude greater than filleted fish.
- The Washington Department of Health has advised against consuming whole fish from the upper Spokane River.

However, data are not available for tribal exposures to fish from the Lake. Therefore, whole fish from Lake Coeur d'Alene is a data gap that has been identified as a future requirement.

2.1.5 CSM Unit 5

CSM Unit 5 is comprised of the Spokane River. Human health concerns for the Spokane River were evaluated separately in the *Draft Final Screening Level Human Health Risk Assessment for Nonresidential Receptors* (USEPA 2000d). In 1999, whole-body and fillet fish samples were collected from the Spokane River by the Washington State Department of Ecology for use in the ecological risk assessment. Whole body fish data from CSM Unit 5 was used in the baseline human health risk assessment to estimate tribal exposures to lead through consumption of whole fish caught from Lake Coeur d'Alene (See Section 2.2.1). However, the fish ingestion pathway was not evaluated in the *Draft Final Screening Level Human Health Risk Assessment for Nonresidential Receptors*.

2.2 SELECTION OF DATA USED IN HHRA

2.2.1 Environmental Data

In the extensive sampling efforts that have occurred in the Coeur d'Alene River Basin, samples have been collected from a variety of environmental media, including soil, house dust, tap water, groundwater, sediment, surface water, and various plant and animal tissues (including homegrown vegetables, fish, and water potatoes). The locations of nonresidential soil, sediment, surface water, and groundwater samples used in the risk calculations are shown in Figures 3-12 through 3-26 in Section 3. Residential data are not shown on these figures because of confidentiality agreements with homeowners; however, residential data are included in the risk calculations for the COPCs selected in Section 2.4.

Data collected by investigators other than the EPA have generally not been included in the baseline HHRA. Because the EPA has conducted sampling investigations specifically for purposes of assessing human health risks, these data were preferentially selected over historical data collected by others and for potentially different purposes. However, data from the Coeur d'Alene Tribe and the State of Idaho were used where applicable.

As of the summer of 1999, the EPA had completed 11 sampling events for various media in the Coeur d'Alene River Basin. These sampling events are referred to in this report by their field sampling plan addendum (FSPA) numbers (e.g., FSPA No.1). Eight of these sampling events produced data applicable to the baseline HHRA.

A summary of each sampling event is included in the RI report for the Coeur d'Alene Basin. Data resulting from the implementation of FSPA Nos. 2,3,4,5,6,7,8, and 12 have been included in the baseline HHRA. The following provides a brief summary of the exposure areas for the FSPAs used in baseline HHRA. Section 3.1.4 further characterizes exposure area by exposure scenario and geographical subarea.

- The sampling for FSPA Nos. 2, 3, and 4 was not planned for human health purposes. The samples were collected for purposes of site characterization, not specifically for human health risk assessment. Consequently, the sampling locations and methods differ somewhat from

those used during the implementation of FSPA Nos. 5, 6, 7, 8, and 12, which were designed to meet HHRA requirements. Therefore, no defined exposure area exists for these FSPAs because the sampling results were not intended for use in the baseline HHRA. This data was used to provide some generic information on potential risks from surface water and sediment exposures in the Osburn, Silverton and Wallace combined, Nine Mile, Canyon Creek, and Mullan areas. This information was used to evaluate neighborhood risks and, in general, sample locations were within a relatively short distance (approximately 2 miles) of at least one residence. The use of this data is further discussed in the uncertainty section, Section 7.

- FSPA No. 5 was the sampling of common use areas and included the sampling of upland parks in Silverton and Wallace in CSM Unit 2, the common use areas along the lower Coeur d'Alene River in CSM Unit 3 (Lower Basin), and beach common use areas around Lake Coeur d'Alene (CSM Unit 4). The beaches around the Lake were previously evaluated in the expedited screening level risk assessment for common use areas (Appendix B), as mentioned in Section 2.1.4. Blackwell Island and Harrison Beach are the only common use areas from the screening level risk assessment that are evaluated in the HHRA because they are the only beaches that had metals concentrations above the screening level, warranting further investigation in the HHRA. Because Harrison Beach is located near the mouth of the Lower Coeur d'Alene River, it was evaluated along with the other CUAs in the Lower Basin subarea. Blackwell Island, however, was evaluated as its own geographical subarea. The exposure areas for the CUAs were defined as the area that people use (i.e., ball fields, parks, etc.) Therefore, the actual exposure areas varied on a site by site basis. As an example of how the exposure areas were defined, refer the representative photographs and sampling location maps for Blackwell Island and Harrison Beach in Appendices A and B of the expedited screening level risk assessment (Appendix B).
- The FSPA Nos. 6, 7, and 12 sampling events were the residential samplings. The exposure areas for these FSPAs varied from residence to residence and were defined by the property boundaries of the yard area.
- FSPA No. 8 included the sampling of five waste piles and groundwater in source areas. The waste piles were sampled as part of the evaluation of neighborhood recreational exposures because local children were known to play on the sampled waste piles located near their homes. Waste piles were sampled purposively to provide generic information on potential waste piles exposures.

Because of the large extent of the Basin study area, it was not possible to sample every single location where exposures might occur. Exposure areas were grouped into geographical subareas in order to give an idea of what typical exposures might be. Even though risks and hazards are evaluated by geographical subarea (See Section 5), risk management decisions will be made on a site by site basis. No risk management decisions will be made in the absence of data.

Data resulting from the implementation of FSPA Nos. 1, 9, 10, and 11 have not been included. FSPA No. 1 involved sediment coring from transects in the lateral lakes; however, samples collected from adjacent locations during FSPA No. 5 consisted of surface sediments along the shoreline and were,

therefore, more appropriate to the evaluation of human exposure. FSPA No. 9 entailed hyperspectral-imaging work. FSPA No. 10 sampling efforts are applicable only to the ecological risk assessment. FSPA No. 11 consisted of data gap sampling not applicable to the risk assessment.

In the summer of 1996, the Idaho Department of Health and Welfare (IDHW) conducted a study (IDHW 2000) that characterized both environmental contamination and biological indices of human exposure within the Basin. During this study, data from 843 residential homes were systematically obtained within the Basin. The data obtained from the IDHW study included yard soil, house mat dust, house vacuum dust, lead-based paint measurements, and tap water. All samples collected were originally analyzed for lead and cadmium. Additional analyses of a subset of the soil data for other metals (e.g., arsenic, mercury, and zinc) have been completed for approximately 80 homes and these were included in the risk calculations for the non-lead metals in addition to the EPA residential data.

In July of 1999, a strategy was adopted to augment the existing database with new information sufficient to support site-specific analysis and provide the risk assessment effort with appropriate information to characterize lead exposure in the Basin. Those public areas, communities, and specific media for which little data were available were sampled in the summer of 1999 by the State of Idaho. A supplemental survey was also conducted by the State of Idaho in November of 1999, that collected environmental samples and survey data from the homes of those children providing blood lead results that had not previously been sampled. Of the 132 homes that were not included in previous efforts, approximately 90 of those homes were sampled in the fall 1999 survey.

The samples from the IDHW study, all EPA residential data, and additional residential data collected by the State of Idaho in the summer and fall of 1999 are included in the lead risk assessment section. The methodology and justification for combining the data sets for the site-specific lead analysis is summarized in the Yard Soil section; the technical memorandum is provided in Appendix N.

Data from fish tissue collected by the State of Idaho and whole fish collected by the State of Washington for the Spokane River RI/FS have been included in the baseline HHRA. Data from water potato samples collected by the Coeur d'Alene Tribe have also been included for application to the traditional and modern tribal scenarios. Water potato data collection methodology is included in Appendix S.

Data from adits were not included because human exposure to water from adits is anticipated to be minimal. Adits are generally located in areas with limited potential for human access and most of them are fenced to prevent human entry. Any unfenced adits will be fenced as part of remediation activities, further limiting exposure.

Yard Soil

Prior to combining the existing data sets for use in the risk assessment, statistical analysis were performed to determine if the data were compatible. Due to the similarities in field sampling protocol of

the FSPA06, FSPA07, and FSPA12 surveys, these data sets were combined by the USEPA. However, the 1996 IDHW/ATSDR study was conducted under a different protocol than that used in the three USEPA surveys. These protocols differ in two major aspects, the sampling methodologies employed and selection of homes. Homes were self-identified based upon a voluntary call-in basis in the USEPA surveys, whereas the IDHW/ATSDR study homes were selected randomly, although the service was offered to everyone.

To evaluate whether the field sampling and analytical techniques used in the surveys produce similar results, surface soil lead and cadmium concentrations from 23 homes common to both the IDHW/ATSDR and the USEPA surveys were compared. The results suggest a strong correlation between the two survey results, but lead concentrations determined by the USEPA protocols may be higher than that observed in IDHW/ATSDR survey. However, this difference, was not apparent for cadmium and the magnitude of the increase is likely not indicative of significant methodological differences between the two protocols with respect to exposure point concentrations and risk calculations for other metals. As a result, surface soil results from the two surveys were combined for additional analysis for metals other than lead. Appendix N provides the methodology and justification for combining the data sets for the site-specific lead analysis.

Several subareas were under-represented in the USEPA surveys. As a result, 89 IDHW/ATSDR samples were retrieved from archives and submitted for re-analysis. Two groups were re-analyzed; 24 samples were analyzed through the EPA CLP and 65 samples were re-analyzed at a private laboratory under contract with the State of Idaho. Statistical evaluations suggested that analytical results were reproducible for lead and cadmium, and that it was appropriate to use the new archive results to characterize other metals concentrations in soils for the risk assessment.

Non-Lead Metals. Soil samples were collected from 191 residential yards in the Coeur d'Alene Basin and analyzed for 23 inorganics. Eighty of these homes were sampled by the State of Idaho, the other 111 homes were sampled by the EPA during work under FSPA Nos. 6, 7, and 12. Both surface and shallow subsurface soil samples were collected. Under the EPA sampling protocol, a minimum of 20 composite samples were collected from five areas at each property. This methodology provided individual sampling locations representing an area in the yard, rather than a single point, thus reducing the potential for anomalous low or high outliers. In addition, discrete areas of the yard with potentially high exposures, i.e., vegetable gardens, children's play areas, gravel driveways, and downspouts were also sampled.

Prior to analysis, soil and house dust samples were sieved through an 80-mesh sieve to capture the fraction less than 175-µm in diameter following American Society for Testing and Materials (ASTM) Method D-422 and the portion that passed through the sieve was analyzed for total metals. The samples were sieved to produce particles of the size expected to adhere to skin (Kissel, Richter, and Fenske 1996a; Duggan and Inskip1985; Que Hee et al. 1985). The size fraction of 175 µm was selected as the most appropriate for evaluating human health exposures for the following reasons:

- Humans receive their greatest exposure to sediments from inadvertent soil ingestion via hand-to-mouth activity resulting from soil adhered to skin (and possibly clothing and objects such as toys).
- A review of scientific literature has identified an upper cut-off size range for dermal particle adherence of 150 to 250 µm (USEPA 2000c).
- The 175-µm size fraction has been used in health risk analyses in the Coeur d'Alene Basin. Using the 175-µm fraction provides comparability with comprehensive soil data collected from upstream mining and smelting sources.
- The 175-µm size fraction has been used in geochemical-exploration studies conducted by the USGS to define naturally occurring levels of metals in soil and rock. Using the 175-µm fraction provides comparability with comprehensive soil and mineral data collected by Gott and Cathrall (1980).
- The 175-µm size fraction is compatible (and protective) for use in the IEUBK Model. The model was validated and calibrated using soil concentration inputs based on the fraction less 250 µm (Hogan et al. 1998).
- Empirical data for determining soil bioavailability for lead for the IEUBK Model is based on studies using the less than 250-µm size fraction (USEPA 2000c, Maddaloni et al. 1998; Casteel et al. 1997).

Lead. Approximately 1020 homes throughout the Basin from the 1996 IDHW, FSPA Nos. 6, 7, and 12, and the 1999 State of Idaho surveys had yard soil data analyzed for lead. In addition, discrete areas of the yard with potentially high exposures, i.e., vegetable gardens, children's play areas, gravel driveways, and downspouts were also sampled. All yard soil samples were sieved to retain particles of less than 175-µm diameter, as discussed above.

House Dust

Non-Lead Metals. Dust samples from floor mats and vacuum cleaner bags were collected from residences throughout the Coeur d'Alene Basin. Eighty-three mat samples and 77 vacuum bag samples were collected and analyzed for 23 inorganics. For 16 of the mat samples from FSPA Nos. 6, 7, and 12, strict adherence to mat handling protocols did not occur. These data are included in the risk assessment for lead and the implications of mishandling are further discussed in Section 7. Generally, the data from the mat samples could underestimate the dust loading because homeowners cleaned the mats, rolled them up, or held them up vertically.

Lead. Dust mats were placed and collected from about 500 homes throughout the Basin and vacuum bag samples were collected from approximately 320 homes for lead analysis during the 1996 IDHW, FSPA Nos. 6, 7, and 12, and the 1999 State of Idaho surveys. Vacuum bag samples give a general representation of lead concentration in the home, while dust mats provide both concentration and a dust loading rate (i.e., grams of dust/m^2/day), and lead loading rate (i.e., mg of lead/m^2/day). Two of the 24 dust mat samples from the 1999 State of Idaho survey were qualified because mat handling protocols by the residents were not followed. Therefore, only 2 mats are known to have been tampered with by

residents. Because 1996 was the first year dust mat sampling was performed, difficulties in quantifying the number of mats tampered with by the residents were encountered.

Tap Water

Non-Lead Metals. Tap water samples were collected from 100 homes and analyzed for 23 inorganic constituents. Forty of the homes were supplied with water from a private source (i.e., a well, a seep or a spring, or surface water). The other 60 homes were supplied with water from a public water supply system. Samples included both first-run and flushed-line tap water. The first-run tap water sample was collected at the beginning of the day, before water had been flushed through the pipes. The flushed-line samples were collected after water had been allowed to run for 10 minutes. Flushing allows metals that might have leached out of the water pipes in a home to be flushed from the water system. Tap water samples were collected under FSPA Nos. 6 and 12.

Lead. A total of about 200 first-run and about 425 flushed-line tap water samples were analyzed for lead throughout the Basin from homes using both a private source and homes using a public water supply system. These samples were collected in the 1996 IDHW, FSPA Nos. 6 and 12, and the 1999 State of Idaho surveys.

Lead-Based Paint

XRF measurements on approximately 415 homes were collected in the 1996 IDHW survey. Lead concentration in interior and exterior surfaces were collected. These data were used to determine if a relationship existed with dust and blood lead levels. These data were not used in the non-lead portion of the analysis.

Groundwater

Eighty groundwater samples were collected from 27 monitoring wells surrounding selected source areas near Nine Mile and Canyon Creeks as part of FSPA No. 8. The groundwater samples were analyzed for 23 inorganics. The groundwater is not currently being used as drinking water, which is obtained from shallow wells up in the side canyons of the two creeks; however, the data were used to quantify a future drinking water use scenario. The use of groundwater as drinking water was the only future scenario evaluated, because all other land use conditions are assumed to remain the same.

Homegrown Vegetables

During work under FSPA No. 6, vegetables were opportunistically collected from 24 residential gardens, after permission was granted by the homeowners. Samples included aboveground produce such as lettuce, basil, cauliflower, cabbage, rhubarb, corn, kohlrabi, and spearmint and root produce such as potatoes, carrots, beets, radishes, and onions. Lettuce, carrots, and potatoes were the most

frequently sampled vegetables. All produce samples were analyzed for arsenic, cadmium, and lead, because these metals were presumed to be good indicator chemicals for health risks from produce. Arsenic and lead were selected because of their toxicity and concentrations in soil. Cadmium was selected because of its toxicity and ability to bioaccumulate in plants.

Upland Soil

Parks, Schools, and Day Care Centers. More than 900 soil samples were collected at 13 upland parks, schools, and day care centers in Silverton and Wallace and analyzed for use in the risk calculations during work under FSPA No. 5. The parks and facilities in Silverton were the following:

- Satner Field, Silverton School District,
- Huggie Bear Day Care,
- Silverton Ball Field near Huggie Bear,
- Silverton T-ball/Wellman Field,
- Silverton T-ball/Wellman Field Park, and
- Silverton T-ball/Wellman & Satner Fields parking lot.

The parks and facilities in Wallace were the following:

- Small city park near schools,
- Wallace Library,
- Wallace City Park (monument),
- Wallace Depot,
- Canyon Avenue Park,
- Wallace Visitors Center & parking lot, and
- Wallace High School & Grammar School playground.

Soil samples were collected from a depth of 0 to 1 inch. In grassy areas, sod was removed and soil was collected both from the root zone and down to the 1-inch depth. Prior to analysis, the soil samples were sieved (Kissel, Richter, and Fenske 1996a).

Waste Piles. Many waste piles are located throughout the Coeur d'Alene Basin. As stated in USEPA 1989, "contamination may be unevenly distributed across a site, resulting in hot spots (areas of high contamination relative to other areas of the site). If a hot spot is located near an area which, because of site or population characteristics, is visited or used more frequently, exposure to the hot spot should be addressed separately." These waste piles are considered to be hot spots as described above and young children have been observed playing on these piles or were reported as having played on them; therefore, piles were sampled "purposively" in order to estimate risks resulting from exposure to waste piles. Purposive sampling is that in which the investigator chooses the sampling location, whereas random and systematic sampling are independent of the investigator and are considered unbiased. While purposive sampling is generally not recommended for risk assessment evaluations, it is

considered an acceptable sampling method for "evaluation of visually obvious contamination" (USEPA 1989), such as waste piles.

Twenty-seven surface soil samples were collected from five waste piles associated with particular mines in the Canyon Creek, Nine Mile Creek, and Mullan areas during FSPA No. 8. The mines Tamarack #7 and Tiger Poorman are located along Canyon Creek, Rex Reach/Reach #2 and Success are along Nine Mile Creek, and the Morning Mine waste dump is on the outskirts of Mullan. Given the large number of waste piles in the Basin, these five were sampled to give a representative understanding of metals concentrations in waste piles. Many waste piles that are accessible on the valley floor and some of the side canyons do receive heavy use by teenagers and adults, although the pile may not be easily accessible by younger children. Therefore, many of the more remote piles do provide exposure and will be evaluated during the remedial design phase although the risk assessment did not quantitatively evaluate these piles.

Because of the rocky nature of the waste piles, not enough fines could be collected from the 0- to 1-inch-depth interval for sieving. Therefore, rather than collecting samples from both the 0- to 1-inch and 1- to 6-inch-depth intervals, samples were collected only from the 0- to 6-inch interval and sieved prior to analysis. The assumption is that the 0- to 6-inch interval is representative of the concentration in the top inch.

Floodplain Soil and Sediment

Approximately 480 samples of Flood plain soil and/or sediment were collected from various locations throughout the Coeur d'Alene Basin during FSPA Nos. 3 and 5. Sediment samples were collected under FSPA No. 3 over a variety of depths to 4 feet below ground surface (bgs). Data from surface sediment samples were selected for inclusion in the baseline HHRA. The results of sediment sampling under FSPA No. 3 (South Fork, Canyon Creek, and Nine Mile Creek) are based on bulk, rather than sieved, samples and sampling locations were not based on human use patterns, because this sampling effort was not initially intended for use in the human health portion of the risk assessment. This data was used because no more human health data was available. Because the samples were not sieved prior to analysis, it likely under-estimates concentrations at these three river segments.

Work under FSPA No. 5 included sampling of soil and sediment in the Flood plain of the lower Coeur d'Alene River. Samples were collected from areas known to be used by the public. Soil/sediment in areas where only surficial play was expected were collected (generally picnic areas a short distance up from the water) from a depth of 0 to 1 inch. Dry sediment along beaches was sampled to a depth of 12 inches. Sediment in the wading portion of the beach (waterline to a water depth of 3 feet) was sampled, at a depth of 0 to 6 inches. Samples were sieved prior to analysis.

Surface Water

Surface water samples were collected from the South Fork and the majority of the large tributary streams: Canyon Creek, Nine Mile Creek, Big Creek, Moon Creek, Beaver Creek, and Pine Creek under FSPA Nos. 2 and 4. These sample collection locations were selected for the purpose of site characterization, not for the evaluation of human health risks.

Subsequently, "disturbed" surface water samples under FSPA No. 5 were collected from the lower Coeur d'Alene River specifically for the HHRA. Disturbed surface water is surface water that contains suspended sediment due to active disturbance of the water by the sampler. These disturbed samples were collected at shallow-water beach locations with a low or moderate slope. Steeply sloped beaches were not sampled based on the assumption that individuals are unlikely to attempt to wade on steeply sloped beaches. Before collecting the surface water samples, the field crew disturbed the sediments (i.e., kicked up sediments into the water column with their feet) in an effort to mimic surface water conditions during water play activities; thus, water samples were randomly collected from the area of dirty water and contained large amounts of suspended sediments. In contrast to previous surface water sampling in other locations, the sampling locations for the human health risk assessment were based on human use patterns of the water bodies.

For subsistence lifestyle exposure scenarios, in addition to disturbed water samples, undisturbed surface water samples were collected in the Lower Basin. The assumption is that tribal members collect undisturbed surface water as their drinking water source. Undisturbed surface water samples were analyzed for 23 metals.

Surface water data for total metals (rather than dissolved metals) were selected for use in the baseline HHRA. Total metals were used in the HHRA for the following reasons:

- Human health toxicity criteria consider total exposure regardless of dissolved versus total form, and
- Humans are exposed via incidental ingestion of the total fraction in water.

Fish Tissue

From 1995 through 1997, the State of Idaho and the Coeur d'Alene Tribe collected fish samples, which were filleted prior to analysis. Data from 312 fish fillet samples from three different species (bullhead, perch, and northern pike) were used in the HHRA. The fish were collected from Killarney, Medicine, and Thompson Lakes, all of which are part of the lateral lakes area of the Coeur d'Alene Basin. Fish tissues were analyzed for mercury, lead, and cadmium. As discussed in Section 2.2.5 for vegetables, indicator chemicals in fish tissue were selected for analysis based on their toxicity and persistence (i.e., ability to bioaccumulate).

Tribal populations traditionally consumed whole fish caught from Lake Coeur d'Alene. Therefore, whole body fish samples represent the fish ingestion pathway for tribal members more accurately than the fillet tissue for the tribal scenarios used in this HHRA. However, as discussed in Section 2.1.4,

whole fish data from the lake is not available, but some whole-body data from the Spokane River is available for lead. In 1999, 67 fish were collected from the Spokane River by the Washington State Department of Ecology. Of the 67 fish, 54 were filleted and then analyzed and 13 whole body fish were analyzed for lead. The fish species collected were wild rainbow trout, hatchery rainbow trout, large scale sucker, mountain whitefish, and crayfish (Johnson 2000). The results from this effort were intended for the ecological portion of the Spokane River RI/FS, but the data are used in lead risk calculations in this human health risk assessment for tribal scenarios for consumption of whole fish. However, there is great uncertainty surrounding these risk and hazard estimates. Extrapolation of hazards and risks associated with fish in Lake Coeur d'Alene from lateral lakes fillet data and Spokane whole fish data is not recommended for a number of reasons. While species with similar feeding habits were compared between the two environments, their exposure pathways to contaminated sediment are different. For example, because the dynamic river environment offers a greater variety of feeding habitats, a bottom feeding river fish is less likely to ingest contaminated sediments than a bottom feeding fish in the uniform depositional environment of the lateral lakes. Similarly, although they share similar feeding habits, the prey base for trout in the river environment is less exposed overall to depositional environments than that for perch in the lakes. In addition, different species bioaccumulate metals at different rates. Therefore, human exposure to metals through consumption of fish may vary with species being consumed. Thus, neither fish data from the lateral lakes nor fish data from Spokane River are likely representative of fish in Lake Coeur d'Alene.

Concern for potential health threats associated with tribal fish consumption is warranted for the following reasons:

- Tribal consumption rates of fish caught from Lake Coeur d'Alene are expected to be higher than other groups fishing the Lake.
- Tribal members traditionally consumed whole fish which can have concentrations of metals approximately an order of magnitude greater than filleted fish.
- The Washington Department of Health has advised against consuming whole fish from the upper Spokane River.

However, data are not available for tribal exposures to fish from the Lake. Therefore, whole fish from Lake Coeur d'Alene is a data gap that has been identified as a future requirement.

Water Potatoes

The Coeur d'Alene Tribe traditionally eats water potatoes harvested from the lower Coeur d'Alene River. In 1994, the tribe collected 95 samples of water potatoes from the lower Coeur d'Alene River and analyzed them for metals, both with skin and without skin. The report regarding collection of water potato data is included as Appendix S. The available data include concentrations of the following detected metals in water potatoes: aluminum, cadmium, lead, manganese, iron, and zinc.

2.2.2 Biological Data

The Idaho Department of Health and Welfare (IDHW) and the Agency for Toxic Substances and Disease Registry (ATSDR) and the Panhandle Health District (PHD) have conducted fixed site blood lead screening in upper and lower basin communities from 1996-1999. The IDHW/ATSDR study was undertaken in the summer of 1996 and the three lead health surveys were conducted by the local PHD in 1997, 1998, and 1999. The IDHW/ATSDR study included blood draws for lead analysis and urine cadmium analysis for both adults and children; the PHD surveys were voluntary child blood lead screenings. A total of 524 children aged 9 months through 9 years have provided venous blood lead samples over the four years. Confidentiality of these data are protected under Idaho State law. However, a redacted blood lead data set is available in Appendix T. The data from these surveys are further discussed in Section 6.

2.3 DATA QUALITY

2.3.1 Data Quality Objectives/Data Quality Assessment

EPA has standard procedures for how data quality shall be assessed in Human Health Risk Assessments. These procedures are outlined in EPA's Data Quality Objectives (DQO) Process Guidance (USEPA 1994d) and Data Quality Assessment (DQA) guidance (USEPA 1998e). These guidance documents were followed and are discussed in varying degrees in the individual FSPAs and the RI/FS. The following is a brief summary of how these guidance documents were applied to the Baseline HHRA. See Section 4.2 of Part 1 of the RI for more a detailed discussion.

The purpose of the 1994 DQO process guidance is to provide general guidance to organizations on developing data quality criteria and performance specifications for decision making. The DQO process is a strategic planning approach that provides a systematic procedure for defining the criteria that a data collection design should satisfy, including when, where, and how many samples to collect and the tolerable level of decision errors for the study. The seven step DQO process was considered and documented in the Draft Technical Work Plan (URS Greiner and CH2M Hill 1998), and considered further and documented in varying degrees in the individual FSPAs developed from 1997 through 2000. Each FSPA and USGS task was developed to address specific data gaps identified after reviewing available historical data and results of previous sampling and analysis efforts. The purpose of each data collection effort was to investigate areas potentially impacted by mining related activities. Due to the large geographic extent of the study area, it was not possible to fully characterize all areas. As all data gaps were not addressed, subsequent studies of specific areas identified for remedial actions may be needed to support remedial design efforts.

The DQA process is a comparison of the implemented sampling approach and the resulting analytical data against the sampling and data quality requirements specified by the DQOs. Results of the DQA are used to determine whether data are of adequate quality and quantity to support the decision-making process. The data quality assessment performed for this study includes evaluation of the quality of the analytical data generated for each of the field sampling efforts and evaluation of the adequacy of the data collected during this study, samples were submitted to commercial laboratories or to EPA for

analysis using the EAPs contract laboratory program (CLP) methods or the EPA SW-846 methods. High quality is maintained in these programs through the use of on-site audits, performance evaluation samples, quarterly performance reports, fraud detection mechanisms, performance based scheduling and continuous inspection of laboratory data. Additionally, all analytical data were validated according to EPA data validation guidance (USEPA 1994e). Following validations, the data set was further reviewed for proper application of data qualifiers. Data identified during validation as being unacceptable for project uses were not carried forward in the assessment.

The sampling plans were designed to provide data to decide if areas are impacted with a high degree of certainty. Since data can only estimate what the true condition of an area is, decisions that are based on measurement data could be in error. Risk assessment requires a high degree of certainty in the supporting data; therefore, field sampling and analysis plans developed to collect data specifically to support the human health risk assessment included sample collection designs with a known confidence level (95 or 99 percent) for the majority of common use area sampling. Specifying confidence levels in advance of sampling is important when defining sites where it is difficult to determine if an area has been impacted by contamination (average concentration close to screening values). Where historical information clearly indicates areas are contaminated (average concentration much greater than screening value), specifying confidence intervals prior to sampling is unnecessary because the chances of falsely characterizing the area are very low. For example, historical data have demonstrated that the waste piles in the Basin contain contaminated soil. Subsequent sampling events have confirmed the initial assumption.

2.3.2 Data Usability

Optimizing data useability in baseline human health risk assessments reduces uncertainty in environmental data used in risk assessment. The 1992, Final Guidance for Data Usabiltiy in Risk Assessment (USEPA 1992e) provides practical guidance on how to obtain an appropriate level of quality of all environmental analytical data required for Superfund human health risk assessments. The following section briefly describes the Data Usability guidance and how it was applied to the HHRA.

The four data application questions requiring an answer for risk assessment from the 1992 guidance are as follows:

1. What contamination is present, and at what levels? - The extent of contamination in the Basin is addressed in Section 2.2 which describes sample collection methods, data analysis procedures, and notes where samples were collected specifically for human health needs versus other uses. The vast majority of the data used in the HHRA was collected based on human health considerations and fulfills the requirements of risk assessment guidance as described in USEPA 1989 and USEPA 1992e. For the relatively small amount of data used that was not collected for HHRA use (sediment and surface water data in the South Fork, Canyon Creek, and Nine Mile Creek), the uncertainties associated with these data are discussed in both Section 2 and in Section 7. Other than the data noted above and the special case of waste piles, all samples

were collected using a randomized or systematic sample design appropriate for risk assessment evaluations.

2. Are site concentrations different from background? - Background samples provide baseline measurements to determine the degree of contamination. Background samples are collected and analyzed for each medium of concern in the same manner as other site samples and require the same degree of quality control and data review. Section 2.4.2 presents background concentrations for all applicable media, except groundwater, for comparisons with site concentrations as part of the COPC screening process. The results of the COPC screening are presented in Section 2.5.
3. Are all exposure pathways and areas identified and examined? - The nature of the exposure pathways and areas to be examined is critical to the selection of a sampling design and analytical methods. If the pathways and areas are not identified properly, the resulting characterization may be inappropriate. Exposure pathways are identified and discussed in Section 3.2 in detail. In addition, exposure pathways are illustrated in the conceptual site model diagrams in Figures 3-3 through 3-11.
4. Are all exposure areas fully characterized? - For all exposure areas to be fully characterized, sampling must be representative and must satisfy performance objectives determined during the planning process. Human health exposure areas are discussed and characterized by FSPA in Section 2.2.1 and by exposure scenario and geographical area in Section 3.1.4. However, they were not explicitly defined in many cases due to the large and complex area of the Basin. Additional data may be required to support remedial design and remedial action activities on a site-by-site basis for individual subareas of the Basin.

2.3.3 Evaluation of Sample Quantitation Limits

Some chemicals for which samples were analyzed were not detected in a sample. These “nondetected” chemicals may be present at a concentration just below the reported sample quantitation limit, or they may not be present in the sample at all. In determining the concentrations most representative of potential exposures at the site, the detected concentrations of a chemical were considered together with one-half of the sample quantitation limit for nondetections.

The adequacy of quantitation limits was evaluated by comparing the sample quantitation limits for each chemical in each environmental medium to risk-based screening values. If a sample quantitation limit was less than the risk-based screening value, it was considered adequate. If sample quantitation limit is greater than the SV, risk may be either overestimated or underestimated. Chemicals with sample quantitation limits greater than the corresponding SV were antimony, arsenic, mercury, and thallium. The number of nondetected values greater than SVs and the total number of nondetected samples for these four chemicals are listed in Table 2-2. Sample quantitation limits greater than SVs may be a concern for chemicals that are not selected because those chemicals might be present elsewhere at levels that warrant a health concern. If a chemical, whose SV is less than the sample quantitation limit, is not selected as a COPC, then one would not know whether or not the chemical is present in concentrations above the level of health concern in future analysis. Antimony, arsenic, and mercury

were selected as COPCs; thallium was not. Potential underestimation of health risks due to not selecting thallium is further discussed in Section 7.

2.4 SELECTION OF CHEMICALS OF POTENTIAL CONCERN

Typically, not all chemicals present at a site pose health risks or contribute significantly to overall site risks. EPA guidelines recommend the selection of a group of COPCs based on their inherent toxicity, their concentration at the site, and their behavior in the environment (USEPA 1989).

The occurrence, distribution, and selection of COPCs are presented in EPA's required format (USEPA 1998c), Risk Assessment Guidance, Part D, Table 2 series (Appendix A). The purpose of these tables is to provide the following:

- Information useful for evaluating the detected chemicals, e.g., frequency of detection and maximum concentration,
- Chemical screening levels, and
- Rationale for selection of COPCs.

The EPA, Part D, Table 2 series (Appendix A) also includes information on potentially applicable or relevant and appropriate requirements (ARARs) for comparison purposes. Although COPCs are primarily selected based on human toxicity, in some cases, ARARs may influence the selection of COPCs. The selected ARARs for groundwater and tap water are the EPA Maximum Contaminant Levels (MCLs), which are the legal limits of chemical concentrations allowed in drinking water. The selected ARARs for surface water are the MCLs and the Ambient Water Quality Criteria (AWQC). AWQC are standards used as the basis for controlling discharges or releases of pollutants (USEPA 1998d). AWQC values that are protective of humans consuming fish are provided in the tables as more applicable than AWQC values that are protective of humans who both consume fish and drink the surface water. Surface water bodies in the Coeur d'Alene Basin are not used as a source of drinking water; however, individuals may inadvertently ingest surface water during recreational activities (e.g., wading or swimming). No ARARs are available for soil or sediment. No ARARs are available for air, except for the National Ambient Air Quality Criterion (NAAQC) for lead.

2.4.1 COPC Screening Process

COPCs were selected for soil/sediment, tap water, surface water, groundwater, house dust, and air. For other media, including homegrown vegetables, fish, and water potatoes, all detected metals were considered COPCs and were evaluated quantitatively in the baseline HHRA.

For purposes of COPC selection, all soil and sediment data were combined, including yard soil, upland soil, waste piles, and stream and river sediments.

The screening process consisted of the following steps:

1. Comparison of chemical concentrations in soil/sediment, tap water, surface water, and groundwater to background concentrations,
2. Identification of chemicals that are essential nutrients and/or nontoxic to humans,
3. Determination of the frequency of chemical detection,
4. For noncarcinogens, comparison of the maximum detected chemical concentration in a particular medium with an SV of 0.1 of the risk-based preliminary remediation goal (PRG); for carcinogens (only arsenic), comparison of the maximum detected concentration with a SV, which is equal to the PRG,
5. If the maximum detected concentration exceeds the SV, evaluation of the frequency and magnitude of the exceedance,
6. Comparison of the maximum detected chemical concentration with the PRG, and
7. If the maximum detected chemical concentration exceeds the PRG, evaluation of the frequency and magnitude of the exceedance.

These steps are described in more detail in the following sections.

2.4.2 Comparison to Background Concentrations

The term background is used here to refer to chemical concentrations that would be expected in the Coeur d'Alene Basin in the absence of historical and ongoing emissions from local mining, smelting, and other ore processing operations. The EPA defines background for inorganic chemicals as "…the concentration of inorganics found in soils or sediments surrounding a waste site, but which are not influenced by site activities or releases" (USEPA 1995c). The potential background concentrations provided in the following subsections for soil/sediment, surface water, and groundwater should be considered preliminary. Background concentrations have been calculated for the RI/FS of the Coeur d'Alene Basin. These background concentrations are discussed below for soil and surface water.

The background concentrations discussed in this section and summarized in Table 2-3 were used for purposes of comparison only. The selected COPCs all exceeded background throughout the entire Basin or a major portion of the Basin (i.e., an entire CSM unit).

Background Concentrations in Soil and Sediment

The primary source used for soil and sediment background concentrations in most previous health risk assessments for the 21-square-mile area commonly referred to as the Bunker Hill Superfund Site (BHSS) and the Coeur d'Alene Basin is the Gott and Cathrall study (Gott and Cathrall 1980). This study analyzed the less than 175 micron fraction of 8,700 soil samples in a 300-square-mile area centered on the Coeur d'Alene Basin. Table 2-3 provides the 90th percentile values of the data for all geologic formations in the area. These levels are considered representative of undisturbed pre-mining soils in the entire Coeur d'Alene Basin. These values were used for comparison with site soil and

sediment concentrations in the baseline HHRA, with the exception of lead and cadmium. Lead and cadmium background concentrations are changed slightly from the values presented in Gott and Cathrall (1980). They are based on a more rigorous statistical analysis of Gott and Cathrall's data by Le Jeune and Cacela (1999). The Le Jeune and Cacela cadmium and lead background concentrations are being used in the Basin RI/FS.

Separate background concentrations were developed for sediments around Coeur d'Alene Lake, as reported in the expedited screening level risk assessment for common use areas, (Appendix B). Derivation of those values followed recent state of California guidelines (CalEPA 1992). National guidance detailing procedures for the selection of background values in soil is not available. Although the background sediment concentrations reported in the expedited screening level risk assessment were very similar to the Gott and Cathrall 90th percentile values, the Gott and Cathrall values were selected for the baseline HHRA because they are considered more representative of the study area.

Background Concentrations in Surface Water

Background concentrations of metals in surface water in the Coeur d'Alene Basin were calculated using the approach described in Appendix C of Maest et al. (1999). Using this approach, EPA accounted for differences in mineralization and watershed properties to determine "baseline" concentrations of total cadmium, lead, and zinc in four portions of the Coeur d'Alene Basin: the Upper South Fork, the Page-Galena mineral belt, the Pine Creek drainage, and the entire South Fork Coeur d'Alene River basin. Surface water background concentrations were developed as part of the RI for the Basin and are presented in Table 2-3.

Background Concentrations in Groundwater

Sufficient data to estimate groundwater background concentrations are not available. Total metals concentrations are used in the HHRA and totals were not available for groundwater background concentrations. Only limited dissolved background concentrations were available and these are discussed here for comparison purposes only. Groundwater has been sampled at 116 locations north of the Palouse River in Idaho (Parliman, Seitz, and Jones 1980). Groundwater was sampled at only seven locations in the Coeur d'Alene River valley upstream from Cataldo. Six of the seven locations were potentially impacted by mining activities, whereas the seventh location, on the North Fork, may be indicative of background concentrations. However, these samples were analyzed for dissolved inorganics rather than total inorganics. Concentrations of mercury, molybdenum, selenium, and vanadium were determined to be "negligible"; however, the sample quantitation limits were not reported. The results of these analyses for dissolved arsenic, cadmium, chromium, copper, iron, lead, and zinc for the North Fork monitoring well are reported in Table 2-3. The potential background concentration for arsenic in groundwater from Parliman, Seitz, and Jones (1980) is discussed in Section 2.5.2. However, because these concentrations are dissolved, they are not directly comparable to the concentrations of total metals presented in the Part D, Table 2 series (Appendix A). Therefore, these groundwater sampling data (Parliman, Seitz, and Jones 1980) are not included in the Part D tables.

2.4.3 Essential Nutrients

Under normal circumstances, the following chemicals are not associated with toxicity to humans: aluminum, calcium, iron, magnesium, potassium, and sodium (USEPA 1998e). With the exception of iron, these chemicals are not considered for inclusion as COPCs. Iron screening values were calculated using a provisional reference dose (RfD) derived from a no-observed-adverse-effect level (NOAEL) based on iron levels in the U.S. population from the second National Health and Nutrition Examination Survey (NHANES II) database (USEPA 1999e). Iron was included as a COPC because of the magnitude and number of exceedances over screening levels (see Section 2.5).

Like iron, aluminum also has a provisional RfD and a calculated soil PRG in the EPA Region 9 PRG tables. However, aluminum was excluded as a COPC. Aluminum was excluded from consideration as a COPC for two main reasons: (1) no concentrations exceed EPA Region 9 PRGs (USEPA 1999c), and (2) concentrations are likely within background for northern Idaho (Shacklett and Boerngen 1984). The impacts of excluding aluminum from the risk assessment are discussed further in the uncertainty section of this report (Section 7).

2.4.4 Frequency of Detection

EPA guidance allows the elimination of chemicals from the quantitative evaluation if they are detected infrequently and the magnitude of exceedance is not a concern (USEPA 1989). In this assessment, a frequency of detection of 5 percent was used as a criterion for the elimination of chemicals as COPCs. In other words, if a chemical was detected in fewer than 5 percent of the samples for a particular medium, it was eliminated as a COPC if the magnitude of exceedance (of the PRG) was not a concern. In all cases, chemicals were detected in greater than 5 percent of the samples.

2.4.5 Comparison to Screening Values and Risk-Based Preliminary Remediation Goals

Soil, Sediment, Tap Water, and Groundwater

Maximum detected concentrations of chemicals in each medium were compared to SVs and risk-based PRGs. PRGs are defined as the residential values listed in EPA Region 9 PRG tables (USEPA 1999c); they represent concentrations in soil, air, and tap water that correspond to a 1 in 1,000,000 (1×10^{-6}) cancer risk (for carcinogenic chemicals) or a noncancer hazard quotient of 1.0 (for noncarcinogenic chemicals). Residential soil PRGs are protective of the ingestion, dermal contact, and inhalation exposure pathways. Residential tap water PRGs are protective of the ingestion pathway. SVs for carcinogens are the same as the corresponding PRG. For chemicals with noncarcinogenic toxicity, however, SVs are defined as 0.1 of the corresponding PRG. The use of 0.1 of EPA Region 9 PRGs for noncarcinogens allows for a more protective screening process by accounting for the additive toxicity of multiple contaminants and follows EPA Region 10 guidelines (USEPA 1998e). Soil PRGs and SVs were used for screening soil and sediment data; tap water PRGs and SVs were used for screening tap water, groundwater, and surface water data.

If the maximum detected concentration of a chemical in a particular medium (soil/sediment, tap water, surface water, or groundwater) did not exceed the SV, the chemical was eliminated as a COPC in that medium. If the maximum detected concentration exceeded the SV, then the frequency and magnitude of exceedance were evaluated. Chemicals with less than 10 percent of the data exceeding the SV and no exceedances of the PRG were eliminated as COPCs. Chemicals with more than 10 percent of their data exceeding the SV were further evaluated by considering the distribution of the concentrations and the frequency of exceedances over the PRG.

Surface Water

Because there are no applicable SVs for surface water that is not used as drinking water, MCLs and AWQC were selected as appropriate SVs. AWQC were preferentially used, if available, as they are closer to approximating the human use of the water body (the selected AWQC are protective against eating fish from the surface water). If a chemical had no AWQC (only 7 of the 23 inorganics have an AWQC), it was screened against the MCL.

Air

Although the residential PRGs for soil include an inhalation component, a separate SV for air was calculated for comparison to the soil and sediment data in the absence of site-specific air data. The SVs for the air pathway were estimated using a default particulate emission factor (PEF), as recommended by the EPA (USEPA 1996b). The PEF relates the chemical concentration in soil with the concentration of dust particles in the air due to fugitive dust emissions from surface-contaminated sites. Particulate emissions are caused by wind erosion and, therefore, depend on the erodibility of the surface material. The EPA used default assumptions for wind speed and the percent vegetation to calculate a PEF value. The default PEF value of 1.32×10^9 m^3/kg (USEPA 1996b) was used to calculate the air pathway SVs for all metals that had inhalation toxicity criteria. The default value was used as a protective approach to screening. Calculations for the air SVs are found in Table 2-4. Chemicals with less than 10 percent of the data exceeding the SV and no exceedances of the PRG were eliminated as COPCs. Chemicals with more than 10 percent of their data exceeding the SV were further evaluated by considering the distribution of the concentrations and the frequency of exceedances over the PRG.

2.5 RESULTS OF COPC SCREENING

The following subsections describe the results of the screening process for each medium, including the rationale for selecting or eliminating chemicals as COPCs. Tables 2-5 through 2-10 show all chemicals detected at concentrations greater than the SVs in more than 10 percent of the samples.

2.5.1 Soil/Sediment

Approximately 4,000 soil and sediment samples were collected within the study area and analyzed for 23 inorganics (Tables 2.1.1, 2.2.1, and 2.3.1 in Appendix A). The tables in Appendix A separate samples by CSM unit for soil/sediment, tap water, and surface water to show differences between CSM units; however, differences in chemical concentration were relatively minimal. Samples were collected from residential yards, common use areas (i.e., public areas), waste rock piles, and stream/river sediments. Thirteen metals were detected at least once at a concentration greater than the SV. The metals with at least one sample but less than 10 percent of the samples exceeding the SV were barium, copper, silver, and vanadium. These chemicals had frequencies of exceedance of 2.4, 3.8, 2.6, and 0.8 percent, respectively. However, concentrations of only nine of these metals exceeded the SV in more than 10 percent of the samples (Table 2-5): antimony, arsenic, cadmium, iron, lead, manganese, mercury, thallium, and zinc.

Seven metals were selected as COPCs in soil/sediment:

- Antimony,
- Arsenic,
- Cadmium,
- Iron,
- Lead,
- Manganese, and
- Zinc.

Six metals (antimony, arsenic, cadmium, iron, lead, and manganese) were selected as COPCs in soil and sediment based on the magnitude and frequency of their exceedances over their respective SVs and PRGs. Iron and manganese concentrations exceeded their 90th percentile background concentrations of 65,000 mg/kg and 3,600 mg/kg in only 9 percent and 11 percent of all soil/sediment samples, respectively. Approximately 10 percent of the data would be expected to exceed the 90th percentile value. Although iron and manganese concentrations in CSM Units 1 and 2 may be within the range of natural background, 68 percent of the iron concentrations and 77 percent of manganese concentrations in CSM Unit 3 exceeded their background concentrations (65,000 mg/kg and 3,600 mg/kg, respectively). Therefore, iron and manganese were selected as COPCs based on exceedances of SVs in all CSM units and exceedances of background in CSM Unit 3.

Three metals (mercury, thallium, and zinc) had relatively low frequencies of exceedance over their respective SVs (13 percent to 15 percent) and very low frequencies of exceedance over their PRGs (less than 1 percent). However, zinc was selected as a COPC because of its historical association with mining activities in the Basin and its prominence in the ecological risk assessment being conducted concurrently with the HHRA. Nevertheless, zinc concentrations are unlikely to contribute significantly to human health risks when compared with the other selected metals. Mercury and thallium were not selected as COPCs in soil and sediment for the following reasons:

- The concentrations of these metals exceeded their respective PRGs with a low frequency less than one percent.
- Histograms of their concentration distribution (Appendix D) indicate the majority of the data are well below the SV; thus, exposures to these metals would not contribute significantly to health risks (hazard quotients of 0.1 or less) in comparison to the other metals.
- Mercury was excluded as a COPC in soil and sediment because exposure to mercury through direct ingestion of soil is not an apparent health concern. However, mercury has the ability to bioconcentrate up the food chain. Therefore, human exposures to mercury may occur through consumption of other organisms (i.e., fish) which are exposed to mercury in soil and sediment. This issue was addressed by selecting mercury as a COPC in the fish ingestion pathway (Section 2.5.7).

The potential underestimation of health risks due to the exclusion of mercury and thallium is discussed in Section 7. In general, floodplain soils/sediments in CSM Unit 3 had the highest concentrations of metals, with the exception of waste piles.

2.5.2 Tap Water

One hundred and two samples of first-run tap water were collected from 100 homes; 100 samples of flushed-line tap water were collected from 100 homes (Tables 2.1.2, 2.1.3, 2.2.2, 2.2.3, 2.3.2, and 2.3.3 in Appendix A). The first-run samples were expected to produce the highest concentration of metals that might leach from water pipes and solder in pipe joints. With the exception of lead, human exposure to chemicals from home plumbing is outside the scope of this risk assessment.

In first-run samples, nine metals were detected at least once at a concentration greater than the SV: antimony, arsenic, cadmium, copper, iron, lead, nickel, thallium, and zinc. In flushed-line tap water samples nine metals were detected at least once at a concentration greater than the SV: antimony, arsenic, cadmium, copper, iron, lead, manganese, thallium, and zinc. In the first-run samples, concentrations of four of the metals exceeded the SV in more than 10 percent of the samples: arsenic, cadmium, copper, and lead (Table 2-6). In the flushed-line sample, concentrations of one metal (arsenic), exceeded the SV in more than 10 percent of the samples (Table 2-7). MCLs are also provided in the tables for discussion purposes because the water samples are from home taps currently being used for drinking water. MCLs are the legal limits for chemicals in drinking water.

Two metals were selected as COPCs in tap water:

- Arsenic, and
- Lead.

The sample quantitation limit for arsenic was greater than the SV in 100 percent of the nondetected samples collected from tap water because the SV is not technically achievable. The lowest sample

quantitation limit was 0.2 µg/L, four times the SV (Table 2-2). Therefore, wherever arsenic was detected, it was detected above the SV and it is uncertain whether the arsenic concentrations in nondetected samples are greater than or less than the SV. Arsenic was detected in approximately 44 percent of the samples.

In the Parliman, Seitz, and Jones study (1980), one sample (from a water supply well in the town of Mullan possibly unimpacted by mining) out of seven samples had dissolved fractions of arsenic detected at a concentration of 1 µg/L. Analytical results for a water sample collected from a well on the North Fork, also likely unimpacted by mining, indicated an arsenic concentration of "0" and the detection limit was not reported. Another sample collected from a well within the Bunker Hill Superfund site (likely mining-impacted) had a dissolved arsenic concentration of 8 µg/L. Arsenic was not detected in the other four wells and the detection limits were not reported. Dissolved fraction concentrations should be lower than total concentrations indicating that arsenic in tap water from a groundwater source is potentially at background; however, the background sample size is extremely small. In general, arsenic in municipally supplied water (groundwater source) was detected at concentrations less than 0.6 µg/L (total concentration), although some detection limits for the nondetected samples were as high as 1.7 µg/L. Arsenic concentrations, on average, were higher in privately supplied water, with detected concentrations ranging from 0.3 µg/L to 9.2 µg/L.

Although arsenic concentrations are below the MCL (the legal limit for drinking water) and potentially at background levels, arsenic was selected as a COPC in tap water because of its exceedance of the SV and the uncertainties surrounding its detection limit.

Risk assessment procedures for lead consider all the lead an individual might encounter in his or her environment regardless of the source. Therefore, all measurements of lead in tap water are evaluated as part of the lead risk assessment regardless of the water source or whether the concentrations exceeded an SV. The Guidance Manual for the IEUBK Model recommends using an average of first draw and flushed tap water samples (USEPA 1994a). Both first draw and flushed tap water samples were collected in FSPA Nos. 6, 7, and 12 and the 1999 State of Idaho Surveys regardless of the source of water. However, in the 1996 IDHW survey, only well water samples were collected from a point as close to the well head as possible and preferably before any holding tanks or filter systems.

Copper was excluded as a COPC because exceedances of the SV were limited to first-run samples, indicating a plumbing issue rather than a mining-related issue. Copper concentrations in only 4 percent of the first-run samples exceeded the PRG and the concentration in only one flushed-line sample exceeded the SV (0.1 percent).

Cadmium was excluded as a COPC because of its relatively low frequency of exceedance of the SV (12 percent) and the PRG (0.1 percent, only one sample) in first-run samples and because exceedances of the SV in flushed-line samples were only 7 percent.

2.5.3 Surface Water

Up to 379 surface water samples were collected and analyzed for 23 inorganics (Tables 2.1.4, 2.2.4, and 2.3.4 in Appendix A). These samples were collected from stream/river locations throughout CSM Units 1, 2, and 3. Of the 379 samples, 130 consisted of "disturbed" surface water containing actively stirred up sediment, and the rest were collected without active sediment disturbance.

Fifteen metals were detected at least once at a concentration greater than the SV. Nine of these metals had frequencies of exceedance of the SV of less than 2 percent. Concentrations of five of these metals exceeded the SV in more than 10 percent of the samples (Table 2-8). All five were selected as COPCs in surface water for both "disturbed" and "undisturbed" water conditions:

- Arsenic,
- Cadmium,
- Lead,
- Manganese, and
- Mercury.

For the metals that were not selected, the frequencies of exceedance of the SVs were less than 2 percent. Concentrations of antimony and zinc did exceed the MCL in more than 10 percent of the samples; however, the AWQC when available are more applicable SVs for water that is not used as drinking water (Section 2.4.5).

Mercury in nondisturbed water samples (all locations except the lower Coeur d'Alene River), was detected in only 3 percent of the samples at concentrations less than the SV. Because sediments could be stirred up during water play activities (i.e., water becomes "disturbed"), mercury was selected as a COPC in surface water for all locations.

2.5.4 Groundwater

Approximately 84 groundwater samples were collected and analyzed for 23 inorganics (Table 2.1.5 in Appendix A). These samples were collected from monitoring wells surrounding selected source areas in CSM Unit 1. Groundwater results are included for screening for the purpose of a future tap water use scenario, because the groundwater is not currently being used as drinking water (groundwater used as municipal supply water is from a different aquifer). Eleven metals were detected at least once at a concentration greater than the SV and concentrations of six of the metals exceeded the SV in 10 percent or more of the samples (Table 2-9). Five of these metals were selected as COPCs (all but manganese):

- Antimony,
- Arsenic,
- Cadmium,

- Lead, and
- Zinc.

Concentrations of manganese exceeded the SV in 16 percent of the samples and exceeded the PRG in 4 percent of the samples. However, manganese was not selected as a COPC because of the low frequency of exceedance of the SV (indicating the majority of the data are below the SV) and the PRG (less than 3 percent). It should be noted that manganese concentrations exceeded a secondary MCL in 25 percent of the samples, that could be an issue if the groundwater was ever developed as a drinking water source. The secondary MCL for manganese is less than the SV. Secondary MCLs are not health based. The potential underestimation of risk due to the exclusion of manganese as a COPC in groundwater is discussed in the uncertainty section (Section 7).

2.5.5 House Dust

The chemicals selected as COPCs in outdoor soil were automatically selected as COPCs in house dust because, with the exception of lead, the source of mining-related chemicals in house dust is assumed to be outdoor soil (Table 2.4.1 in Appendix A). To ensure that no additional COPCs should be selected for house dust and to verify that the concentrations of chemicals selected in outdoor soil also exceeded the SVs for indoor dust, house dust concentrations were screened using soil SVs. There were 160 samples of house dust from floor mats and vacuum bags, representing the 83 homes available for screening.

The detected chemicals and frequencies of exceedances of the SVs in dust are similar to those seen for soil. Fourteen metals were detected in dust at least once at a concentration greater than the SV. Thirteen of these metals were the same ones that exceeded the SVs in soil. The additional metal in dust (nickel) was detected at a concentration exceeding the SV in only one sample, a frequency well below 10 percent. In dust, as in soil, the concentrations of nine metals exceeded the SV in more than 10 percent of the samples (Table 2-10). Eight of the nine metals in dust were the same as those in soil: antimony, arsenic, cadmium, iron, lead, manganese, mercury, and zinc. The ninth metal was copper.

Seven metals were selected as COPCs in house dust:

- Antimony,
- Arsenic,
- Cadmium,
- Iron,
- Lead,
- Manganese, and
- Zinc.

Copper and mercury had relatively low frequencies of exceedance (15 and 14 percent, respectively) of the SVs and no exceedances of the PRGs; consequently, they were not selected as COPCs in house dust according to the same rationale described for soil in Section 2.5.1.

Lead in house dust is evaluated in Section 6.0. Vacuum bag dust lead is used in the IEUBK Model analysis. Dust mat samples were not used as input to the model. In the case of a missing vacuum bag dust sample, the average dust lead concentration for that community was used. Dust mat samples have not typically been evaluated in the IEUBK Model.

2.5.6 Air

All soil and sediment samples collected within CSM Units 1, 2, and 3 were combined to evaluate the air pathway (Table 2.4.2 in Appendix A). Approximately 4,200 soil and sediment samples were collected and analyzed for 23 inorganics. These samples were collected from residential homes, common use areas, waste rock piles, and stream/river sediments. Arsenic and manganese were the two chemicals detected at a concentration greater than the SV in at least one sample; however, the frequency of the exceedances were less than 10 percent (Table 2-11). Due to the low percentage of exceedances, these two chemicals were excluded as COPCs for the air pathway. Regardless of its concentrations, lead was selected as a COPC in air to be quantitatively evaluated in the lead model.

2.5.7 Fish

No COPC screening was performed for fish tissue data because no appropriate SVs were available (Table 2.4.4 in Appendix A). All chemicals analyzed in fish were considered COPCs:

- Cadmium,
- Lead, and
- Mercury.

2.5.8 Homegrown Vegetables

No COPC screening was performed for homegrown vegetables. All produce samples were analyzed for arsenic, cadmium, and lead (Table 2.4.3 in Appendix A). The COPCs selected for homegrown vegetables to be evaluated quantitatively in the baseline HHRA are the following.

- Arsenic,
- Cadmium, and
- Lead.

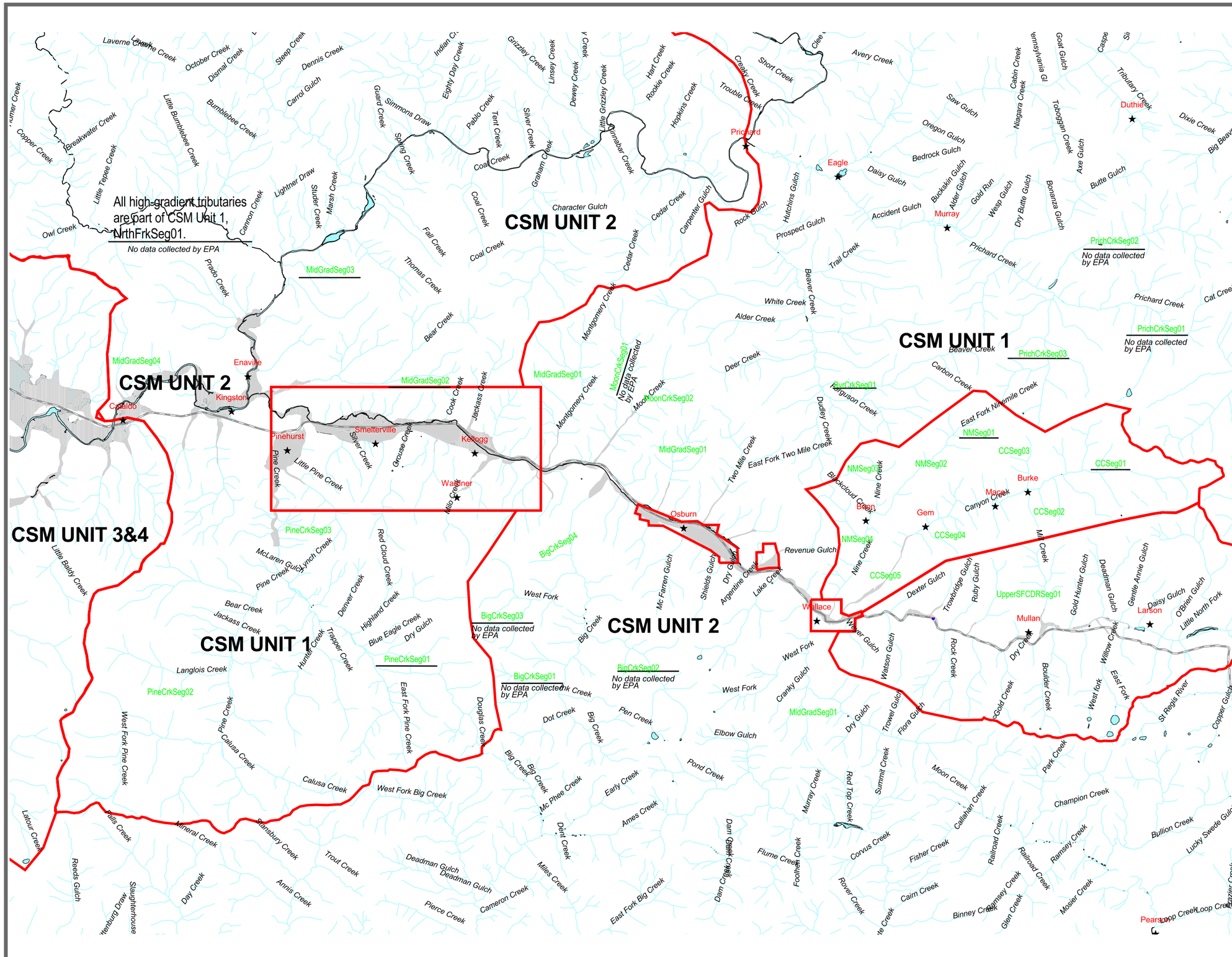
2.5.9 Water Potatoes

No comparison of detected chemicals to SVs was performed for water potatoes because of a lack of appropriate SVs. Cadmium and lead were selected as indicator COPCs to be consistent with the other food chain samples, vegetables and fish (Table 2.3.5 in Appendix A). The other metals selected as COPCs in soil for which there are analytical data in water potatoes (manganese, iron, and zinc) are unlikely to contribute significantly to health risks due to water potato ingestion in comparison to direct ingestion of soil and sediment.

2.6 SUMMARY OF CHEMICALS OF POTENTIAL CONCERN

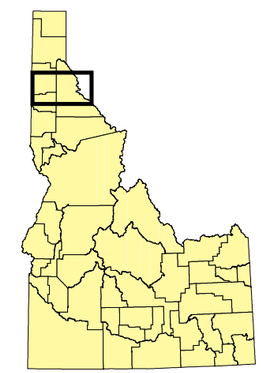
Chemicals were selected as COPCs if they exceeded screening values that were based on residential exposures. For the general population, residential exposures would be the highest that would be expected for any media. However, the risk assessment also evaluated subsistence exposures. The special subsistence population could potentially have exposures to media greater than residential populations. Thus some chemicals, which were screened out, might present a health risk for the subsistence populations. This issue is further discussed in Section 7.2.1. Table 2-12 summarizes the COPCs selected in each medium for quantitative evaluation in the baseline HHRA.

Figure 2-1
Stream Segments in Eastern Portion of Coeur d'Alene River Basin



LEGEND

- MidGradSeg03 — Segment Excluded from HHRA
- City
- Interstate 90
- Stream
- River
- Floodplain
- Basin Study Regions



LOCATION MAP

NOTES

1) Map data were obtained from the Bureau of Land Management, CH2M HILL, and URS Greiner, Inc.

2) This map was created for planning purposes for the Coeur d'Alene River Basin Study.

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SCALE 1:160,000

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027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

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View: EastHalf Segments
Layout:East Half
3/06/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983

Date of Plot: MARCH 6, 2001

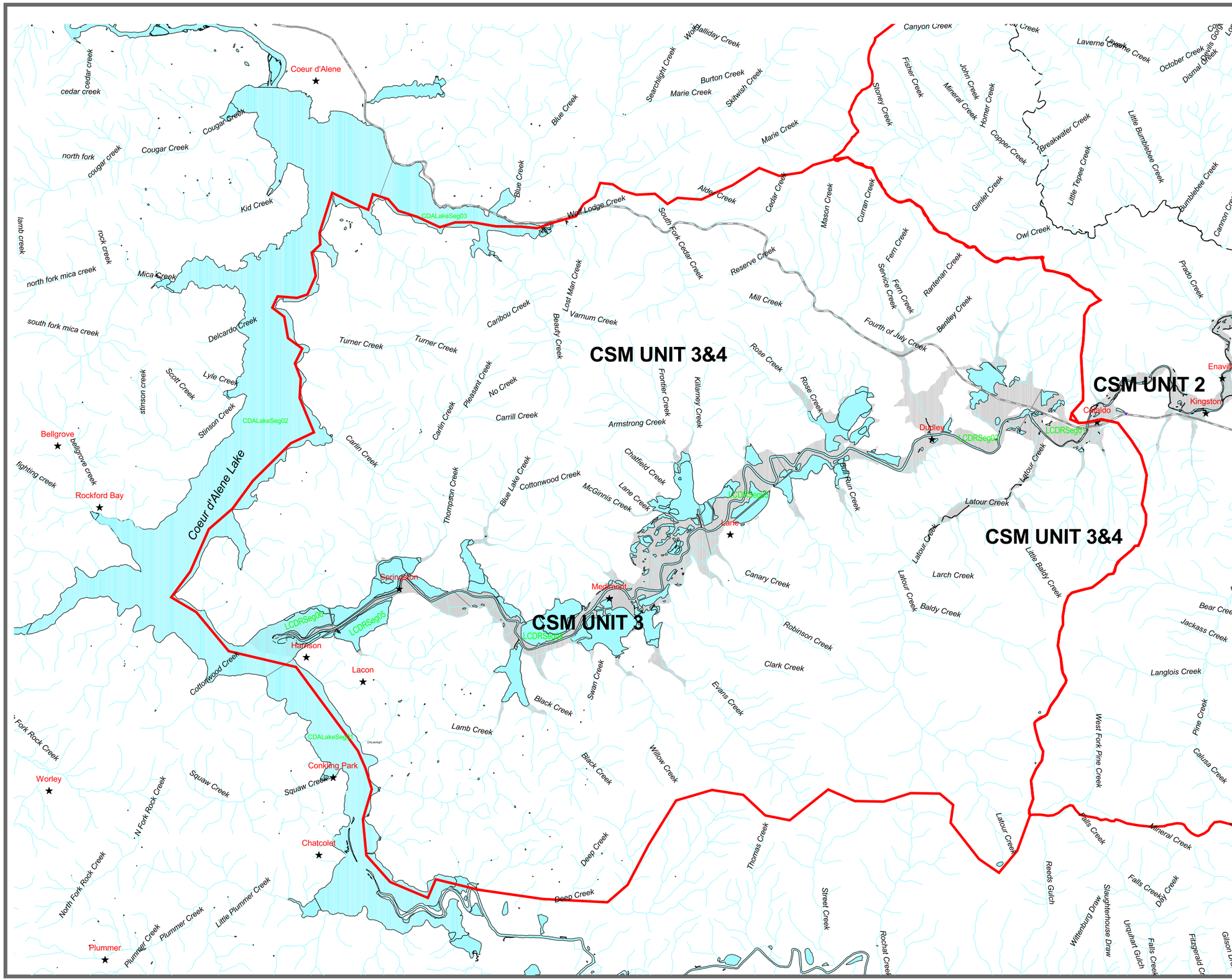
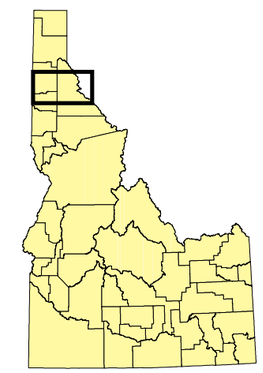


Figure 2-2
Stream Segments in Western Portion of Coeur d'Alene River Basin

LEGEND

- City
- Interstate 90
- Stream
- River/Lake
- Floodplain
- Basin Study Regions



LOCATION MAP

NOTES

1) Map data were obtained from the Bureau of Land Management, CH2M HILL, and URS Greiner, Inc.

2) This map was created for planning purposes for the Coeur d'Alene River Basin Study.

SCALE 1:160,000

1 0 1 Miles

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027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

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LAYOUT:-L-West Half (11x17)
03/06/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983

Date of Plot: MARCH 6, 2001

Table 2-1
Stream Segments and Beach Sites in CSM Units, 1, 2, 3, and 4

Exposure Scenario	Segment	Data Included in HHRA?
CSM Unit 1: Upper Watersheds ***Geographical Subareas: Mullan, Nine Mile, Side Gulches, and Kingston***		
Residential only	BigCrkSeg04	Yes
Residential and recreational (water play)	NMSeg03	Yes
Residential and recreational (water play and waste piles)	CCSeg02	Yes
	CCSeg03	Yes
	CCSeg04	Yes
	CCSeg05	Yes
	NMSeg02	Yes
	NMSeg04	Yes
	PineCrkSeg02	Yes
	PineCrkSeg03	Yes
Residential and recreational (water play, waste piles, and upland parks/schools)	UpperSFCDRSeg01	Yes
Residential and recreational (waste piles)	MoonCrkSeg02	Yes
Recreational (water play and upland parks)	BvrCrkSeg01	No[a]
Recreational (waste piles)	PineCrkSeg01	No[b]
Minimal human use	BigCrkSeg01	No[c,d]
	BigCrkSeg02	No[c,d]
	BigCrkSeg03	No[c,d]
	CCSeg01	No[b,c,d]
	MoonCrkSeg01	No[b,c,d]
	NrthFrkSeg01	No[b,c,d]
	NMSeg01	No[b,c,d]
	PrichCrkSeg01	No[b,c,d]
	PrichCrkSeg02	No[b,c,d]
	PrichCrkSeg03	No[b,c,d]
CSM Unit 2: Midgradient Watersheds ***Geographical Subareas: Wallace, Silverton, Osburn, Side Gulches, and Kingston***		
Residential and recreational (water play, waste piles, and upland parks/schools)	MidGradSeg01	Yes
Residential and recreational (water play and upland parks)	MidGradSeg04	Yes

Table 2-1 (Continued)
Stream Segments and Beach Sites in CSM Units, 1, 2, 3, and 4

Exposure Scenario	Segment	Data Included in HHRA?
Minimal human use	MidGradSeg02	No[c,e]
	MidGradSeg03	No[c]
CSM Unit 3: Lower Coeur d'Alene River and Floodplain ***Geographical Subarea: Lower Basin***		
Residential and recreational (water play, beaches, and upland parks)	LCDRSeg01	Yes
	LCDRSeg02	Yes
	LCDRSeg03	Yes
	LCDRSeg04	Yes
	LCDRSeg05	Yes
	LCDRSeg06	Yes
CSM Unit 4: Coeur d'Alene Lake ***Geographical Subarea: Blackwell Island***		
Recreational (water play and beaches)	Blackwell Island	Yes
	22 Beach Sites	No[f]

[a]Area will be addressed by the U.S. Forest Service. No soil data available.
[b]Minimal human access to area.
[c]Area is relatively uncontaminated
[d]No EPA data available for this segment.
[e]Consists of the 21-square-mile area commonly referred to as the Bunker Hill Superfund site, which is being investigated separately.
[f]Previously evaluated (see Appendix B).

Note:
Locations of segments are shown in Figures 2-1 and 2-2.

Table 2-2
Chemicals With Sample Quantitation Limits Exceeding Screening Values

Medium	Chemical	Range of Sample Quantitation Limits	Screening Value	Exceedance		
				No. of Nondetections	No. of Nondetections Exceeding SV	Frequency of Exceedance (%)
Soil/sediment	Antimony	0.2 - 30 mg/kg	3 mg/kg	1,063	162	15.2
	Arsenic	0.18 - 15.2 mg/kg	0.38 mg/kg	22	21	95.5
	Thallium	0.38 - 9.8 mg/kg	0.52 mg/kg	2,958	2,484	84
Tap water (first-run)	Arsenic	0.2 - 1.7 µg/L	0.045 µg/L	57	57	100
	Thallium	0.03 - 0.4 µg/L	0.26 µg/L	97	2	2.1
Tap water (flushed-line)	Arsenic	0.2 - 0.7 µg/L	0.045 µg/L	55	55	100
	Thallium	0.03 - 0.38 µg/L	0.26 µg/L	94	1	1.1
Groundwater	Arsenic	0.2 - 1.0 µg/L	0.045 µg/L	64	64	100
	Thallium	0.03 - 1.0 µg/L	0.26 µg/L	73	32	43.8
Surface water	Arsenic	0.16 - 2.0 µg/L	0.14 µg/L[a]	149	149	100
	Mercury	0.1 - 0.5 µg/L	0.051 µg/L[a]	256	256	100

[a]Ambient Water Quality Criterion

Note:
SV - screening value

Table 2-3
Potential Background Concentrations for Soil, Surface Water, and Groundwater

Chemical	Soil[a] (mg/kg)	North Fork Surface Water[b] (µg/L)	Groundwater[c] (µg/L)
Antimony	5.8	0.51	NA
Arsenic	22	0.65	"0"
Barium	1,109	NA	NA
Beryllium	2.1	NA	NA
Cadmium	2.86	0.09	3
Calcium	1	NA	NA
Total chromium	64	NA	"0"
Cobalt	20	NA	NA
Copper	53	1.21	67
Iron	65,000	113	30
Lead	175	1.46	7
Magnesium	1.1	NA	NA
Manganese	3,600	8.28	NA
Mercury	0.3	0.09	"0"
Nickel	38	NA	NA
Silver	1.1	0.12	NA
Sodium	NA	NA	NA
Thallium	NA	NA	NA
Vanadium	154	NA	"0"
Zinc	280	20.71	20

[a]90th percentile values from Gott and Cathrall 1980, except for lead and cadmium, which are 95th percentile values from Le Jeune and Cacela 1999
[b]Surface water background values as calculated by URSG, March 2000
[c]From Parliman, Seitz, and Jones 1980

Notes:
Soil background concentrations were used for sediment.
NA - not available
"0" - laboratory sample quantitation limit not available (nondetection)

Table 2-4
Soil Screening Values for Air Pathway

Chemical	CAS No.	Unit Risk Factor $(\mu g/m^3)^{-1}$	Reference Concentration (mg/m^3)	Reference Dose for Inhalation (mg/kg-d)	Screening Value (mg/kg)
Aluminum	7429-90-5	NA	NA	NA	—
Antimony (trioxide)	7440-36-0	—	2.00E-04[a]	—	27,500
Arsenic	7440-38-2	4.30E-03[a]	—	—	747
Barium	7440-39-3	—	5.00E-04[b]	1.40E-04[c]	68,800
Beryllium	7440-41-7	2.40E-03[a]	—	—	1,340
Cadmium	7440-43-9	1.80E-03[a]	—	—	1,780
Calcium	7440-70-2	NA	NA	NA	—
Chromium[d]	7440-47-3	1.20E-02[a]	1.00E-04[a]	—	268
Cobalt	7440-48-4	—	2.00E-05[b]	5.70E-06[e]	2,750
Copper	7440-50-8	NA	NA	NA	—
Iron	7439-89-6	NA	NA	NA	—
Lead	7439-92-1	NA	NA	NA	—
Magnesium	7439-95-4	NA	NA	NA	—
Manganese	7439-96-5	—	5.00E-05[a]	—	6,880
Mercury[f]	7487-94-7	—	3.90E-04[a]	—	41,300
Nickel[g]	7440-02-0	2.40E-04[a]	—	—	13,400
Potassium	7440-09-7	NA	NA	NA	—
Selenium	7782-49-2	NA	NA	NA	—
Silver	7440-22-4	NA	NA	NA	—
Sodium	7440-23-5	NA	NA	NA	—
Thallium	1314-32-5	NA	NA	NA	—
Vanadium	7440-62-2	NA	NA	NA	—
Zinc	7440-66-6	NA	NA	NA	—

$$\text{Screening value for carcinogens (mg/kg)} = \frac{TR \times ATc \times 365 \text{ days/year}}{URF \times 1000\ \mu g/mg \times EF \times ED \times 1/PEF}$$

$$\text{Screening value for noncarcinogens (mg/kg)} = \frac{THQ \times AT_{nc} \times 365 \text{ days/year}}{EF \times ED \times (1/RfC \times 1/PEF)}$$

Where:

TR (target risk) = 1.00E-06 (unitless)
ATc (averaging time [carcinogen]) = 70 years
URF (unit risk factor) = chemical-specific $(\mu g/m^3)^{-1}$
EF (exposure frequency) = 350 days/year
ED (exposure duration) = 30 years
PEF (particulate emission factor) = 1.32E+09 m^3/kg
THQ (target hazard quotient) = 0.1 unitless
AT_{nc} (averaging time for noncarcinogen) = 30 years

Table 2-4 (Continued)
Soil Screening Values for Air Pathway

RfC (reference concentration) = chemical-specific mg/m^3

[a]USEPA 1999a
[b]RfC = Reference dose for inhalation x 70 kg x $(20\ m^3/day)^{-1}$
[c]USEPA 1997c
[d]Screening level based on carcinogenic effects of chromium RfC from inhalation of chromium VI particulates.
[e]USEPA 1998g
[f]RfC based on inhalation of elemental mercury vapor.
[g]URF based on inhalation of nickel refinery dust.

Notes:
— - not applicable
NA - not available

Table 2-5
Summary of Analytical Results for Chemicals in Soil/Sediment With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples

Chemical	No. of Detections	No. of Samples	Maximum Conc. (mg/kg)	Screening Value (mg/kg)	No. of Detections Exceeding SV	Percentage of Samples Exceeding SV	PRG (mg/kg)	No. of Detections Exceeding PRG	Percentage of Samples Exceeding PRG	Background Conc.[a] (mg/kg)	No. of Detections Exceeding Background Conc.	Retained as COPC?
Antimony	2,966	4,029	623	3	1,766	43.8	30	313	7.77	5.8	1,239	Yes
Arsenic[b]	4,186	4,208	3,610	0.38	4,186	99.5	0.38	4,186	99.48	22	1,346	Yes
Cadmium	3,939	4,208	194	3.7	1,923	45.7	37	184	4.37	2.86	2,290	Yes
Iron	3,980	3,980	256,000	2,200	3,980	100	22,000	1,527	38.37	65,000	369	Yes
Lead	4,208	4,208	67,100	400	1,336	31.7	400	1,336	31.75	175	3,065	Yes
Manganese	4,002	4,002	26,400	310	3,878	96.9	3,100	500	12.49	3,600	450	Yes
Mercury	3,570	4,208	47.3	2.2	534	12.7	22	6	0.14	0.3	2,226	No
Thallium	633	3,898	14.4	0.52	537	13.8	5.2	31	0.80	NA	—	No
Zinc	4,208	4,208	25,800	2,200	610	14.5	22,000	3	0.07	280	2,806	Yes

[a]90th Percentile values from Gott and Cathrall 1980
[b]Carcinogen; SV and PRG are protective of cancer health effects

Notes:
Chemicals shown in ***bold italic type*** were selected as COPCs.
COPC - chemical of potential concern
NA - not available
PRG - preliminary remediation goal (from tables in EPA Region 9 Web site at http://www.epa.gov/region09/waste/sfund/prg)
SV - screening value (0.1 times EPA Region 9 PRGs for noncarcinogens and same as PRGs for carcinogens)

Table 2-6
Summary of Analytical Results for Chemicals in First-Run Tap Water With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples

Chemical	No. of Detections	No. of Samples	Maximum Conc. (µg/L)	Screening Value (µg/L)	No. of Detections Exceeding SV	Percentage of Samples Exceeding SV	PRG (µg/L)	No. of Detections Exceeding PRG	Percentage of Samples Exceeding PRG	MCL (µg/L)	No. of Detections Exceeding MCL	Retained as COPC?
Arsenic[a]	45	102	7.6	0.045	45	44.1	0.045	45	44.1	50	0	Yes
Cadmium	45	102	33.6	1.8	12	11.8	18	1	1.0	5	5	No
Copper	98	102	2,620	140	27	26.5	1,400	4	3.9	1,300	4	No
Lead	101	102	78.5	4	36	35.3	4	36	35.3	15	11	Yes

[a]Carcinogen; SV and PRG are protective of cancer health effects

Notes:
Chemicals shown in ***bold italic type*** were selected as COPCs.
COPC - chemical of potential concern
MCL - Maximum Contaminant Level
PRG - preliminary remediation goal (from tables in EPA Region 9 Web site at http://www.epa.gov/region09/waste/sfund/prg)
SV - screening value (0.1 times EPA Region 9 PRGs for noncarcinogens and same as PRGs for carcinogens)

Table 2-7
Summary of Analytical Results for Chemicals in Flushed-Line Tap Water With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples

Chemical	No. of Detections	No. of Samples	Maximum Conc. (µg/L)	Screening Value (µg/L)	No. of Detections Exceeding SV	Percentage of Samples Exceeding SV	PRG (µg/L)	No. of Detections Exceeding PRG	Percentage of Samples Exceeding PRG	MCL (µg/L)	No. of Detections Exceeding MCL	Retained as COPC?
Arsenic[a]	45	100	9.2	0.045	45	45.0	0.045	45	45.0	50	0	Yes
Lead	83	100	9.5	4	2	2.0	4	2	2.0	15	0	Yes

[a]Carcinogen; SV and PRG are protective of cancer health effects

Notes:
Chemicals shown in ***bold italic type*** were selected as COPCs.
COPC - chemical of potential concern
MCL - Maximum Contaminant Level
PRG - preliminary remediation goal (from tables in EPA Region 9 Web site at http://www.epa.gov/region09/waste/sfund/prg)
SV - screening value (0.1 times EPA Region 9 PRGs for noncarcinogens and same as PRGs for carcinogens)

Table 2-8
Summary of Analytical Results for Chemicals in Surface Water With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples

Chemical	No. of Detections	No. of Samples	Maximum Conc. (µg/L)	Screenin g Value (µg/L)	MCL (µg/L)	No. of Detections Exceeding MCL	Percentage of Samples Exceeding MCL	AWQC (µg/L)	No. of Detections Exceeding AWQC	Percentage of Samples Exceeding AWQC	Back-ground Conc. (µg/L)	No. of Detects Exceeding Background Conc.	Retained as COPC?
Antimony	220	379	39.5	NA	6	93	24.5	4,300	0	0	0.51	201	No
Arsenic	230	379	600	NA	50	59	15.6	0.14	228	60.2	0.65	162	Yes
Cadmium	274	379	1,810	NA	5	184	48.5	NA	--	--	0.09	274	Yes
Lead	359	379	81,500	NA	15	227	59.9	NA	--	--	1.46	287	Yes
Manganese	316	379	84,900	NA	50 (SMCL)	184	48.5	100	154	40.6	8.28	270	Yes
Mercury	122	379	43.9	NA	2	60	15.8	0.051	122	30.6	0.09	122	Yes
Zinc	307	379	540,000	NA	5000 (SMCL)	75	19.8	69,000	3	0.3	20.71	237	No

Notes:
Chemicals shown in ***bold italic type*** were selected as COPCs.
— - not applicable
AWQC - Ambient Water Quality Criteria Human Health Consumption of 'Organism Only' (USEPA 1998d)
COPC - chemical of potential concern
MCL - Maximum Contaminant Level
NA - not available
PRG - preliminary remediation goal (from tables Region 9 Web site at http://www.epa.gov/region09/waste/sfund/prg)
SMCL - secondary maximum contaminant level
SV - screening value (0.1 times EPA Region 9 PRGs for noncarcinogens and same as PRGs for carcinogens)

Table 2-9
Summary of Analytical Results for Chemicals in Groundwater With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples

Chemical	No. of Detections	No. of Samples	Maximum Conc. (µg/L)	Screening Value (µg/L)	No. of Detections Exceeding SV	Percentage of Samples Exceeding SV	PRG (µg/L)	No. of Detections Exceeding PRG	Percentage of Samples Exceeding PRG	MCL (µg/L)	No. of Detections Exceeding MCL	Retained as COPC?
Antimony	32	84	18	1.5	27	32.1	15	1	1.2	6	10	Yes
Arsenic[a]	20	84	16.1	0.045	20	23.8	0.045	20	23.8	50	0	Yes
Cadmium	71	84	996	1.8	61	72.6	18	39	46.4	5	51	Yes
Lead	71	84	3,170	4	51	60.7	40	51	60.7	15	29	Yes
Manganese	51	84	8,030	170	13	15.5	1,700	3	3.6	50 (SMCL)	21	No
Zinc	83	84	145,000	1,100	51	60.7	11,000	20	23.8	5,000 (SMCL)	31	Yes

[a]Carcinogen; SV and PRG are protective of cancer health effects

Notes:
Chemicals shown in ***bold italic type*** were selected as COPCs.
COPC - chemical of potential concern
MCL - Maximum Contaminant Level
PRG - preliminary remediation goal (from tables in Region 9 Web site at http://www.epa.gov/region09/waste/sfund/prg)
SMCL - secondary maximum contaminant level
SV - screening value (0.1 times EPA Region 9 PRGs for noncarcinogens and same as PRGs for carcinogens)

Table 2-10
Summary of Analytical Results for Chemicals in House Dust With Concentrations Exceeding Screening Values in More Than 10 Percent of Samples

Chemical	No. of Detections	No. of Samples	Maximum Conc. (mg/kg)	Screening Value (mg/kg)	No. of Detections Exceeding SV	Percentage of Detections Exceeding SV	PRG (mg/kg)	No. of Detections Exceeding PRG	Percentage of Detections Exceeding PRG	Retained as COPC?
Antimony	160	160	318	3	142	88.8	30	29	18.1	Yes
Arsenic[a]	160	160	635	0.38	160	100.0	0.38	160	100.0	Yes
Cadmium	159	160	375	3.7	146	91.3	37	5	3.1	Yes
Copper	160	160	1,040	280	24	15.0	2,800	0	0.0	No
Iron	160	160	60,800	2,200	158	98.8	22,000	115	71.9	Yes
Lead	160	160	59,500	400	133	83.1	400	133	83.1	Yes
Manganese	160	160	5,460	310	153	95.6	3,100	3	1.9	Yes
Mercury	160	160	21.5	2.2	21	13.1	22	0	0.0	No
Zinc	160	160	57,500	2,200	24	15.0	22,000	2	1.3	Yes

[a]Carcinogen; SV and PRG are protective of cancer health effects.

Notes:
There are no background values available for house dust.
Chemicals shown in ***bold italic type*** were selected as COPCs.
COPC - chemical of potential concern
NA - not available
PRG - preliminary remediation goal (from tables in Region 9 Web site at http://www.epa.gov/region09/waste/sfund/prg)
SV - screening value (0.1 times EPA Region 9 PRGs for noncarcinogens and same as PRGs for carcinogens)

Table 2-11
Summary of Analytical Results for Chemicals in Air With Concentrations Exceeding Screening Values in Any Sample

Chemical	No. of Detections	No. of Samples	Maximum Conc. (mg/kg)	Screening Value (mg/kg)	No. of Detections Exceeding SV	Percentage of Detections Exceeding SV	PRG	No. of Detections Exceeding PRG	Percentage of Detections Exceeding PRG	ARAR (mg/m^3)	No. of Detections Exceeding ARAR	Retained as COPC?
Arsenic[a]	4,186	4,208	3,610	747	5	0.1	NA	--	--	NA	--	No
Lead	4,208	4,208	67,100	NA	--	--	NA	--	--	1.5 (NAAQC)	--	Yes
Manganese	4,002	4,002	26,400	6,880	290	7.2	NA	--	--	NA	--	No

[a]Carcinogen; SV and PRG are protective of cancer health effects.

Notes:
Chemicals shown in ***bold italic type*** were selected as COPCs.
— - not applicable
COPC - chemical of potential concern
NA - not available
NAAQC - National Ambient Air Quality Criteria
PRG - preliminary remediation goal (from tables in Region 9 Web site at http://www.epa.gov/region09/waste/sfund/prg)
SV - screening value (0.1 times EPA Region 9 PRGs for noncarcinogens and same as PRGs for carcinogens)

Table 2-12
COPCs Selected for Each Medium

Chemical	Soil/ Sediment	House Dust	Tap Water	Surface Water	Groundwater	Air	Fish	Homegrown Vegetable	Water Potatoes
Antimony	X	X			X				
Arsenic	X	X	X	X	X			X	
Cadmium	X	X		X	X		X	X	X
Iron	X	X							
Lead	X	X	X	X	X	X	X	X	X
Manganese	X	X		X					
Mercury				X			X		
Zinc	X	X			X				

3.0 EXPOSURE ASSESSMENT

3.1 CHARACTERIZATION OF EXPOSURE SETTING

3.1.1 Physical Setting

Site Location

The Coeur d'Alene Basin is located in the Panhandle region of northern Idaho and lies within Kootenai and Shoshone Counties. The Basin is part of the Bitterroot Mountain Range and the Coeur d'Alene Mountains. Much of the area is rural and contains a wide variety of landscape types rich in natural resources including floodplain, steep mountain canyons, and river valley.

Topography and landscape vary in the Basin from relatively open, flat, floodplain areas of the Coeur d'Alene River in the western portion of the Basin to steep, narrow canyons in the eastern portion of the Basin. The floor of the valley near the boundary between Kootenai and Shoshone Counties is roughly 1 mile wide and narrows significantly eastward toward Shoshone County. Valley areas near Wallace are only 0.25 mile wide.

The Purcell Trench, which includes the Rathdrum Prairie, forms the western side of the Basin from Coeur d'Alene Lake to Athol. With the exception of the Rathdrum Prairie, stream channels in the Basin store more unconsolidated, alluvial soil and rock material in the stream bottoms and along the toe slopes than most other areas in the Idaho Panhandle region. These materials are very susceptible to movement (IPNF 1998). The upper North Fork has shallower and weakly weathered, rocky soils. Soils in the lower Coeur d'Alene River area tend to be more highly weathered and contain less rock fragments in the soil profile, making them susceptible to subsoil and substratum erosion (IPNF 1998).

In the mountainous terrain of the eastern portion of the Coeur d'Alene Basin, soils are typically poorly developed, apparently due to the steady erosion of the soil cover on the steep slopes of the canyons. Many areas lack vegetation and consist of loose rock fragments.

Climate

The climate in northern Idaho is influenced primarily by prevailing westerly winds that carry maritime air masses from the North Pacific across the northern Rockies during the winter and spring. This weather pattern persists from the Selkirk Mountains in British Columbia south to the Clearwater National Forest and is characterized by precipitation occurring as long gentle rains, deep snow accumulations at higher elevations, cloudiness, and high humidity. Changes in the position of the jet stream can push inland maritime airflow north causing significant drought in northern Idaho.

Elevation is also a major influence on local climate. The lowest elevations (approximately 2000 feet), which lie in the western portion of the Basin, are generally the warmest and driest. Areas with higher

elevation (approximately 5500 feet) in the eastern portion of the Basin generally are cooler and have greater annual precipitation.

Summers in the area are generally hot and dry with only about 12 percent of the annual precipitation occurring between July and September. Approximately 50 percent of the annual precipitation occurs between November and February. The remaining precipitation takes place in the spring. Winter temperatures are 15 to 25 degrees higher than those in continental locations of similar latitude. These weather patterns make the Basin one of the highest precipitation areas of the upper Columbia River Basin and result in the potential for frequent high water events.

Local Communities and Area Use

Much of the Basin is rural, undeveloped land. Approximately 32 percent of Kootenai County and 75 percent of Shoshone County consist of federally managed lands, primarily National Forest Lands (IPNF 1998). These areas are rich in natural resources including forests, wildlife, and a number of tributaries and streams that support a variety of aquatic organisms. However, many of these areas are inaccessible due to the lack of roads and the difficult terrain. Interstate 90 (I-90) has provided limited access to the otherwise rural area.

Tourism related to the use of these natural resource areas for recreational purposes has increased significantly over the last two decades and is one of the fastest growing contributors to the local economy. Recreational use of the area's abundant natural resource areas include riding off-road vehicles, snowmobiling, berry picking, fishing and floating the Coeur d'Alene River, and cross-country and downhill skiing.

Despite the recent economic growth, the lack of development in the Basin has resulted in many small rural communities, primarily along the Coeur d'Alene River and its tributaries. The majority of the population of the Basin live in the cities of Coeur d'Alene and Post Falls, which have populations exceeding 10,000 people. All the other communities in the Basin have populations below 6500, and in both Kootenai and Shoshone Counties, more than 38 percent of the total population live in rural areas outside of major cities, including members of the Coeur d'Alene Tribe (IPNF 1998). The Coeur d'Alene Tribe is dependent on the waters of Lake Coeur d'Alene, the surrounding lateral lakes, and the Coeur d'Alene River and closely interacts with the natural environment and uses the natural resources. The Coeur d'Alenes' historic use of the Basin is explained in greater detail in Section 3.1.5, *Subsistence Lifestyles*, while current uses are described in Section 2.1.4.

Communities along the upper Coeur d'Alene River and its tributaries were established and supported in the past by the mining and timber industries, agriculture, and related activities. Mining activities have occurred in the area for more than 100 years and between 1880 and 1965, over 400 sawmills opened and closed in the Basin (IPNF 1998).

The CSM units and stream segment subdivisions described in Section 2.1 were originally established for the ecological risk assessment. Consequently, these divisions generally do not reflect human exposure patterns in the Basin. Geographic subareas defined for human health risk assessment are discussed in Section 3.1.3.

3.1.2 Coeur d'Alene Basin Demographics

Introduction

The purpose of this section is to describe the population and regional characteristics of the Coeur d'Alene Basin HHRA study area. Emphasis is placed on data specific to children, the primary population of concern for lead exposure, and risk co-factors, such as parental income, education, and socioeconomic status, considered to influence the risk of lead poisoning. In general, demographic data are presented according to geographic divisions and data sources. Geographic divisions include HHRA geographic subareas as well as counties, cities, and school districts within the Basin.

The primary source for Basin area demographics is 1990 census data. A discussion of the applicability of 1990 data to current conditions and boundary conflicts between Basin study areas and census tracts is included in following section *Geographic Areas, Data Sources, and Assumptions*. The 1990 census data was used because the census geographic divisions of blocks and block groups could be more appropriately applied to HHRA geographic study areas than county and city boundaries. More current data is presented at a county level, primarily for Shoshone County that is assumed to be representative of the Basin area. Most of the data presented for Shoshone County is referenced from Idaho Department of Commerce (IDOC) reports and documents. Additional data specific to children in Shoshone County and the State of Idaho is referenced from "Idaho Kids Count: Profiles of Well-Being" annual reports. School district annual enrollment data is the final source of demographic information described in this report. School district data is considered the best and most recent data available for determining the actual numbers of children currently living in the Basin area. Data for most geographic divisions and sources is presented in a comparison format so that Basin area/county/school district demographics can be viewed in relation to the State of Idaho.

Geographic Areas, Data Sources, and Assumptions

Geographic Areas. Demographic data is presented according to the following geographic areas: the Basin, Shoshone County, the cities of Mullan, Osburn, and Wallace, and School Districts #391, 392, and 393 (Table 3-1a). The Basin Area consists of the eight subareas defined for the Coeur d'Alene Basin Study listed in Table 3-1b and shown in Figure 3-1a. All references to the Basin in this report include summary information for the population living within the outer boundaries formed by the eight study areas.

Small portions of both Benewah and Kootenai Counties, as well as a large part of Shoshone County are included in the Basin Area. However, data at a county level is only presented for Shoshone

County. Shoshone County was selected as being representative because 73% of the Basin Area population and 51% of the land mass is in Shoshone County. Additionally, the demographic characterization of Shoshone County is considered more typical of the Basin Area than either Kootenai or Benewah Counties. Kootenai County encompasses the city of Coeur d'Alene, that is not inside of the Basin Study Area boundaries and is more urban in character. Kootenai County summary demographics would strongly reflect the influence of the relatively large population and strong economy found in Coeur d'Alene. Benewah County is more rural in character than Kootenai County, however, only a small percentage of the land in Benewah County is included within the Basin Area (3%). In addition, while most of the economies of the communities in the Basin Area are traditionally based in mining, production of forest and wood products has traditionally served as the foundation for the local economy in Benewah County.

Only a small portion of the demographic data presented in this report is for cities within the Basin Area. There are no major cities (i.e., population greater than 20,000) within the Study Area boundaries and the amount of data available for the smaller communities is limited. Data is presented for three cities within Shoshone county (Mullan, Osburn, and Wallace) because all three are defined as study areas in the Basin and are incorporated. No data specific to Silverton were available, although it is also a defined study area within the Basin.

Three school districts are included in the Basin Study Area. These are the Kellogg School District (#391), the Mullan School District (#392), and the Osburn-Wallace School District (#393). A significant portion of the Kellogg School District includes students residing within the Bunker Hill Superfund Site (BHSS). Data from previous studies done on the BHSS were used to determine the approximate percentage of students within the BHSS boundaries and these students were then excluded from counts of the Basin Area student population.

Data Sources. The primary data sources for Basin demographics are listed below.

1. 1990 Census (CensusCD +Maps V.2.0)
2. Idaho Department of Commerce reports, documents, and Internet accessible data including,
    - Idaho Facts: Information and Statistics About Idaho's People and Economy
    - Profile of Rural Idaho, A look at economic and social trends affecting rural Idaho
    - County Profiles of Idaho
    - Idaho Community Profiles (www.idoc.state.id.us)
3. "Idaho Kids Count: Profiles of Well-Being" annual reports, 1996-2000
4. School District Data as provided by School Districts 391, 392, and 393

Data from the 1990 census was the primary source for Basin Area demographics. Census data was used because data were available for small geographic units, referred to as blocks and block groups,

that could be overlaid on Basin Area maps to develop demographic information specific to defined study areas. Two areas of concern regarding the use of 1990 census data include the changes that may have occurred in the last ten years and the potential for census geographic grouping boundaries to be different than Basin Study Area boundaries. The applicability of 1990 census data to current conditions is considered throughout the report when corresponding data is available from both the 1990 census and more recent years.

Several factors that have changed over time, as presented in "County Profiles" (IDOC) for Shoshone County, are described in Table 3-2. Some census data are also included. As indicated in Table 3-2, the population of Shoshone County and the cities within the County have shown a significant decrease in population since the 1970s and 1980s. Shoshone County had a population of 19,718 in 1970 and by 1990, that number had decreased by almost 30% to 13,931. However, since 1990, the population has shown comparatively little change with a minor (0.4%) decrease from 1990 to 1998. The cities within the County show similar trends. Between 1990 and 1996, the populations in Mullan and Wallace decreased by 3.2% and 5.5%, respectively. The population of Osburn showed a slight increase of 0.9% for the same time period. By comparison, the State of Idaho showed a 20% increase in population since between 1990 and 1998.

Also shown in Table 3-2 are the changes in economic indicators for Shoshone County such as total employment, mining employment, and unemployment. Data from 1990 is presented from two sources for these factors because census employment data did not match data presented in IDOC reports. This may be due to the time of year the data was reported. IDOC data, rather than census data, are used in the following comparisons because the 1970 and 1980 data are referenced from the "County Profiles" report. Similar to population changes, all three factors showed a significant change from 1980 to 1990. Total employment decreased by 30% and mining employment decreased by 37%. Correspondingly, the unemployment rate increased from 6.7% to 9.9%. Between 1990 and 1996, total employment increased slightly (4%), while mining employment continued to decrease significantly (58%). Tourism and recreation appear to be growth sectors, replacing some of the mining jobs. Unemployment showed a slight increase (0.3%) from 1990 to 1998.

In general, the greatest changes in the Basin Area demographics occurred between 1970 and 1990. Since 1990, changes in population and economic indicators, with the exception of mining jobs, have remained fairly constant indicating relatively little growth or decline. In summary, while 1990 census data is not completely accurate in describing the current population in the Basin Area, it does reflect the current economic and demographic status of the Basin Area.

Geographic boundaries formed by the census groupings, and a potential for a mis-match with Basin Area boundaries, was the second concern in using census data. Census data is available in units referred to as census blocks and census block groups. Census blocks are defined as small areas bounded on all sides by visible features such as streets, roads, streams, and railroad tracks, and by institutional boundaries such as city, town, township, and county limits, property lines, and short, imaginary extensions of streets and roads. Census blocks are the smallest geographic units for which

basic demographics are available. Census block groups are made up of census blocks and are the smallest geographic units for which detailed demographics are available. Because of the greater amount of data available for census block groups, these were the preferred regional divisions to use for Basin Area data and are the primary source for demographic summaries presented in this report.

Census block group boundaries were overlaid onto the Basin Study area map on GIS. Block groups falling within Basin Areas were then related to the appropriate study area. Corresponding block group data was then linked to the individual study areas. Figure 3-1b shows the census block groups overlaying Basin study area boundaries. Block group boundaries were similar along the southern edge of the study area, but differed in the northern section. Three block groups fell only partially within the study boundaries. The population and area of block groups with boundaries outside of the study area are listed in Table 3-3.

The block groups that have only a small portion of their entire area within the Basin Areas boundaries make up 30% of the total Basin Area. However, they account for only 11% of the total population. In addition, the land portions of these groups outside of the study area boundaries are in highly forested and mountainous areas likely to have small populations. It is assumed that most of the people living in these block groups are located within or close to the study area boundaries which are relatively proximal to population centers. Based on this assumption, it was determined appropriate to include these groups in the summary demographics.

Sources other than the 1990 census are presented at State, county, city, and school district levels. The primary assumption in using this data is that the information presented for Shoshone County, Mullan, Osburn, Wallace, and the school districts can be considered representative of the Basin as a whole.

General Basin Demographics

The Coeur d'Alene Basin Study Area encompasses approximately 880 square miles of the northern Panhandle region of the State of Idaho, or 1.1% of the total area of Idaho. The study area includes small portions of both Benewah and Kootenai Counties, as well as a large portion of Shoshone County, excluding Lake Coeur d'Alene and the Spokane River. Benewah County and Kootenai County make up 3% and 46% of the Basin Area, respectively. Shoshone County accounts for the remaining 51%.

The Basin Area is considered rural without major cities (i.e., population of 20,000 or more), and higher education facilities or regional medical centers. Approximately 10,500 people, or 1% of the total population of Idaho reside within the study area. Typical of most rural areas in Idaho, the population density is relatively low, with 5.3 persons per square mile living in Shoshone County. Comparatively, the State of Idaho averages 14.8 persons per square mile, while rural areas in Idaho average 6.1 persons per square mile (IDOC 1999). The low population density in Shoshone County is attributable to the fact that approximately 96.2% of the land is forested and is primarily owned by the federal government. Most of the federal land is either national forest or held by the Bureau of Land Management (BLM).

The economy of the region, traditionally based in mining, has declined over the last 10 to 20 years due to mine closures and layoffs and a lack of other industry to replace them. Table 3-2 shows the total mining employment in 1980, 1990, and 1996. Between 1980 and 1996, total mining employment decreased by 74%. As a result, the total population has also showed a declining trend as people move outside of the area seeking jobs. The population of Shoshone County decreased by 29% between 1970 and 1990. Between 1990 and 1998, the population has remained relatively unchanged, with only a slight decreasing trend (0.4%). From 1997 to 1998, Shoshone County is noted as one of only fifteen counties in Idaho to lose population. The population loss in all fifteen counties was attributed to downturns in agriculture, timber, and mining (IDOC 1999).

As the younger generation is forced to move outside of the area to find employment, the population of the Shoshone County is also becoming older as indicated in Table 3-2. The median age of residents in Shoshone County in 1970 was 27.3 years. Since then, the median age has been increasing and in 1998 the median age of residents was estimated at 39.6. Comparatively, the median age of residents statewide in 1998 was 33.5. In 1997, only nine counties in Idaho, including Shoshone, had greater than 15% of their population aged 65 and over. The percent of the population aged 65 and over in Shoshone County in 1997 was 15.7%. In 1970, the percent was less than half that at 7.1%.

The following sections provide additional and more detailed demographic data of the Basin Area according to 1990 census data, as well as a comparison of Shoshone County demographics with other rural counties and statewide data for Idaho. Information specific to the child population as presented in "Idaho Kids Count: Profiles of Well-Being" annual reports and as determined by school district data are also summarized. Finally, the total number of children currently living within the Basin Area (excluding the BHSS) and the number of housing units within the Basin Area were estimated based on the available data.

Basin Area Demographics based on 1990 Census Data

Summary population characteristics for the Basin Area are presented in Table 3-4. The Basin Area makes up approximately 1.0% of the total population of Idaho. The breakdown of the percentage of males, females, and minorities in the Basin Area is similar to statewide data with a 51/49 ratio of male to female and the majority of persons being white (98%). However, there is a higher population of Hispanic persons statewide (5.1%) compared to the Basin Areas (2.1%). The Basin Area population is relatively older with a higher percentage of persons over age 35 (55%) when compared with the overall state of Idaho percent (44%) and correspondingly, a smaller percentage of persons under age 35, at 45% for the Basin Area and 56% statewide. The relatively older population of Shoshone County is attributed to the decline in mining jobs, and the subsequent migration of workers and their families outside of the area in search of employment.

Household characteristics of the Basin population and the State of Idaho are presented in Table 3-5. The total number of households in the Basin Area is 4215 or 1.2% of the total number of households in Idaho. The breakdown of family type is similar for both the Basin Areas and the State of Idaho, with

the majority of the families being made up of married couples either with or without children. The percentage of single parent families for both Idaho and the Basin Areas is similar at 7% and 6%, respectively.

Table 3-6 summarizes the housing characteristics of the Basin Area and the State of Idaho. The total number of housing units in the Basin Area is 5651 or 1.4% of the total number of housing units in Idaho. The percentage of occupied housing units in the Basin Area (74%) is lower than the statewide percentage (87%). This may be due to a high number of seasonal units in some of the Basin Area census groups. Both the Lower Basin study area and the Kingston Area contain census block groups with a high percentage of seasonal units, up to 61% of total housing units in a Lower Basin block group near Lake Coeur d'Alene. Block groups in the Kingston Area (Shoshone County) with a high percentage of seasonal units are located near the North Fork of the Coeur d'Alene River. The percentage of renter occupied units is lower in the Basin Area than statewide at 23% and 30%, respectively. The statewide average is likely influenced by a higher number of renters in urban areas.

The breakdown of the number of units in housing for both the Basin Area and the State are similar, with the majority, 73% and 71% respectively, being single unit dwellings. The percentage of 2-9 unit dwellings (duplexes to small apartment complexes) and 10+ unit dwellings (large apartment complexes) is smaller in the Basin Area (7%) than statewide (14%), while the percentage of mobile/trailer homes in the Basin is slightly higher (19%) than statewide (14%). The total number of 2+ unit dwellings in the Basin Area as estimated from the 1990 census is 376. Mobile/trailer homes are often reflective of socio-economic status and are unlikely to be a source of lead-based paint. The total number of mobile/trailer homes in the Basin Area as estimated from the 1990 census is 1053 units.

Housing units in the Basin Area are typically older than that reported statewide. Housing age was found to a significant factors influencing lead loadings in a study done on housing units in North Idaho (Spalinger et al. 2000), with older houses (built before 1960) showing a higher loading than newer housing units (built after 1960). This was attributed to two factors, lead paint and a longer exposure period to lead in dust and soils. The use of lead paint in residential homes declined in the 1960s and was banned by 1978. Forty-eight percent of the housing units in the Basin Area were built before 1960, and over half (60%) of those were built before 1940. Statewide, only 37% were built before 1960 and less than half of those (44%) were built before 1940.

Since 1980, the percentage of houses built in the Basin Area is also lower than statewide, at 12% and 18%, respectively. Current data show a similar lag in housing growth for Shoshone County. From 1990 to 1997 housing growth in Shoshone County was 5.6% which fell well behind statewide growth of 21.6% (Profile of Rural Idaho, IDOC).

In addition to being older homes, residents of the Basin Area have also lived in their houses for longer periods of time, as seen in Table 3-6. Almost half (45%) of the housing units at the time of the 1990 census were occupied by persons living in them for a minimum of ten years (i.e., moved in before 1980). Statewide, a higher percentage (65%) moved in to housing units after 1980 and only 34%

moved in before 1980. Studies done on the BHSS found that housing units where residents had lived in the unit for 5 years or more showed a lower lead loading rate than units with shorter term residents. Specifically, rentals with a highly mobile population showed higher lead loadings (TerraGraphics 2000a). Of the 4205 occupied housing units in the Basin Area, 954 (23%) were renter occupied.

Median values for housing characteristics for individual block groups within the Basin Areas are listed in Table 3-7. State of Idaho median values for housing characteristics are also included in Table 3-7. The three Basin Areas defined as cities, Wallace, Osburn, and Silverton, do not have census block groups associated with them because none of the block groups are completely or mostly contained within the city Basin Areas (Figure 3-1b). The demographic data for block groups that are partially within City Study Areas are included in the Side Gulches Study Area. Where available, City information is also included in the tables but not in the summary descriptions. As indicated in Table 3-7, the median year built for housing in Idaho is 1970. Basin Area housing is typically older than the overall State housing with twelve of the eighteen block groups in the Basin Area (67%) having a median year built prior to 1970. The median year built for housing in all of the block groups located within the Lower Basin Area (3) was greater than 1970. All three are in Kootenai County. The other three block groups with a median year built after 1970 were in Shoshone County in the Kingston Study Area (2) and the Side Gulches Study Area (1). Housing in the Mullan Basin Area and the City of Wallace are the oldest with a median year built of 1939.

The median value of housing units in 1990 in the State of Idaho as presented in Table 3-7 was $58,000. Basin Area housing values were typically lower than the State median with fourteen of the eighteen block groups included in the Basin Area (78%) having median housing values less than the statewide median. The four block groups with median values greater than $58,000 are located within the Kingston (2) and Lower Basin Study Areas (2). The majority of median rent values in the Basin Areas are also lower than the State median rent value of $330. Of the eighteen block groups in the Basin Area, only one (located in the Kingston Study Area) has a higher median rent value.

Student population and educational attainment data for the Basin Areas and the State of Idaho are presented in Table 3-8. Of the entire student population in Idaho, including pre-primary, elementary, high school, and college students, 2416 or 0.8% of the student population live within the Basin Area. The percentage of the population over age 25 without high school diplomas is greater in the Basin Areas (27%) than in the State (20%). Correspondingly, the percentage of the population over age 25 with high school diplomas (73%) is less than the State percentage of 80%. The percentage of the population that attended college is also slightly lower for the Basin Areas at 19%, than the statewide percentage of 24%. The percentage of college graduates and the percentage of the population obtaining a Masters Degree or higher is lower for the Basin Areas than for the State, with combined percentages of 19% and 30%, respectively.

Household income for the Basin Areas and the State area presented in Table 3-9. A slightly higher percentage of the Basin Area population (54%) had incomes in the lower bracket (less than $25,000) than the statewide percentage (49%) and correspondingly, a slightly lower percentage (11%) than the statewide percentage (16%) had incomes greater than $50,000. The median household income from 1990 is presented in Table 3-9 for block groups located within the Basin Areas and the State of Idaho. The three Basin Areas defined for cities, Wallace, Osburn, and Silverton, do not have census block groups associated with them because none of the block groups are completely or mostly contained within the city Basin Areas (Figure 3-1b). The demographic data for block groups that are partially within City Study Areas are included in the Side Gulches Study Area. Where available, City information is also included in the tables but not in the summary descriptions. The median household income for the State of Idaho in 1990 was $25,257. Thirteen of the eighteen block groups within the Basin Area (72%) had lower median household incomes that the state median value. Block groups with median incomes exceeding the statewide median were located in the Kingston Study Area, the Lower Basin Study Area, and the Side Gulches Study Area.

Shoshone County Profile -- Current Data

Table 3-10 summarizes data as presented in "Profile of Rural Idaho," (Idaho Department of Commerce). The table presents a comparison of data from Shoshone County, the State of Idaho, and urban and rural areas in Idaho. In general, Shoshone County falls behind the state, and both urban and rural Idaho for all economic indicators and income and poverty levels. The unemployment rate is significantly higher in Shoshone County than state, rural, and urban levels at almost twice the percentage of the other areas (12.8%) in 1997. Business growth from 1990 to 1996 fell well behind even statewide rural levels for both total and retail growth, and housing growth from 1990-1997 was significantly behind Idaho's statewide, urban, and rural areas at 5.6% compared to 21.6%, 27.1%, and 13.6%, respectively.

Per capita personal income in 1996 is the only category in which Shoshone County is higher than rural Idaho, and that is only slightly at $16,938 compared to $16,513 for rural Idaho. The statewide and urban values were $19,865 and $21,773, respectively. The median household income for Shoshone County in 1995 was $6,000 to $10,000 lower than Idaho statewide, urban, and rural values. The percent of persons in poverty in Shoshone county in 1995 was significantly higher than all other Idaho areas, with a high percentage (31.2%) of children living in poverty. This data is similar to the data presented from the Kids Count reports in Table 3-11 for 1990, 1994, and 1996, with the percentage of children in poverty increasing for each of the years. Welfare payments per capita were also significantly higher in Shoshone County in 1998 at $771 compared to $378, $452, and $248 statewide, in urban, and in rural Idaho, respectively.

The percentage of housing units built before 1939 and after 1970 are also presented in Table 3-10. Interestingly, almost the same percentage of housing units in Shoshone County were built before 1939 (34.4%) as were built after 1970 (32.5%). In contrast, the statewide, urban, and rural percentages all indicate a significantly higher percentage of housing units built after 1970. Basin-wide, the percentage

of housing units built before 1940, as reported in the 1990 census (Table 3-6) was similar to Shoshone County at 29%. Older housing units are likely to have a higher lead loading rate when compared with newer housing units due to the presence of lead paint and a longer exposure period to lead soils and dusts.

Kids Count Data

Data as presented in "Idaho Kids Count 1999-2000: Profiles of Well-Being" annual reports from 1996 to 1999-2000 are summarized in Table 3-11 (Annie E. Cassie Foundation 2000). Kids Count reports are published annually and are intended to provide reliable data to inform citizens and policy makers about the status of Idaho's children and to improve their well-being. Data in Table 3-11 are presented according to Report Years. Annual reports contain a compilation of data from several different years. The actual years that presented data are referenced from are footnoted below the table. For example, population data in the 1996 annual report are based on 1994 estimates. The four annual reports summarized here present a range of data from 1990 to 1998.

Data from Shoshone County is compared to the State of Idaho for several factors affecting the child population in Table 3-11. Between 1994 and 1998, the child population under 18 in Shoshone County showed a decrease of 6%, while the total population remained fairly constant. Statewide, the child population showed a slight increase (3%) between 1994 and 1998, along with the total population that increased by 8%. The decreasing child population is likely due to families moving outside of the area in search of employment. Data for children living in Shoshone County showed higher than statewide percentages of child poverty, single parent families, infant mortality, low birth weight babies, school dropouts, teen births, and teen violent deaths for all years included. As an example, the percentage of children in poverty in Shoshone County increased from 23.7% to 31.2% from 1990 to 1996, while the percentage of children in poverty statewide remained relatively constant at approximately 16% to 17%. Many of these poor social indicators are often associated with a depressed economy.

Economic well being data from the 1999-2000 Kids Count Annual Report are summarized in Table 3-12. Again, Shoshone County exceeds the statewide percentage for adverse economic factors. The percentage of school children receiving free or reduced price lunch in Shoshone County in the 1997-1998 school year was 50%. The percentage increased to 54% in the 1998 to 1999 school year. Statewide, the percentages were lower at 41% and 42% for the 1997-1998 and 1998-1999 school years, respectively. Data as reported by the Kellogg School District (#391) and the Osburn-Wallace School District (#393) in the current school year (1999-2000) indicated that 44% of all students enrolled in the Kellogg district and 40% of all students enrolled in the Osburn-Wallace District received free or reduced price lunch. The Kids Count data was for elementary students only, and indicate that a higher percentage of elementary students receive free or reduced price lunch than secondary (high school) students.

Births paid for by medicaid showed a decrease in Shoshone County from 1997 to 1998 (55% to 42%), however, the percentage remained higher than statewide numbers of 33% and 28% for 1997 and 1998, respectively.

The socio-economic status of children in Shoshone County seems to have decreased over the last decade as illustrated in Tables 3-11 and 3-12. Table 3-13 summarizes the change in the child population, also a decrease, as presented in the 1999-2000 annual report. From 1990 to 1998, the number of children under the age of 5 in Shoshone County decreased by 12.1%. Statewide, the number of children under age 5 increased by 12.2%. The number of children between the ages of 5 and 17 in Shoshone County also decreased by 7.7% while statewide the number increased by 14%. Overall, the total number of children under 18 in Shoshone County decreased by 8.7% and increased in statewide by 13.5%. While the child population in Shoshone County appears to be decreasing, negative factors affecting children continuing to live in the area seem to increase (Table 3-11). Socioeconomic status of families has been noted to be a significant factor affecting children's blood lead levels and environmental media in numerous studies (Pirkle et al. 1998, Brody et al. 1994, Clark et al. 1985, Bornschein et al. 1985) .

School District Data

Tables 3-14 and 3-15 summarize school district enrollment data for the Kellogg School District (#391), the Mullan School District (#392), and the Osburn-Wallace School District (#393). School district data was obtained with the help of the three districts involved. The Kellogg School District has the highest enrollment numbers over the last decade followed by the Osburn-Wallace District and finally, the Mullan School District. Overall, enrollment in all three districts have shown a consistent decrease in the total number of students since the early 1990s. Total enrollment in the three districts decreased by 13% from 1992 to 2000. Enrollment data is shown graphically in Figure 3-2.

A significant portion of the Kellogg School District (#391) includes students residing within the Bunker Hill Superfund Site (BHSS). However, the BHSS is not included in the current Basin Study Area. In order to exclude the student population residing within the BHSS, the number of students residing in the BHSS was estimated using a study done in 1999 which showed that approximately 68% of the students enrolled in the Kellogg School District lived within the BHSS (TerraGraphics 2000a). Therefore, the total enrollment of students in the Kellogg District living outside of the BHSS was determined by subtracting 68% of the total number of students given for the Kellogg District. The adjusted data are shown in Table 3-14. Table 3-15 shows the breakdown of student enrollment data by grade. The percentage of students in each grade is similar (all between 7-9%).

Estimation of the Child Population

Children eligible for the blood lead sampling program conducted as part of Basin risk assessment studies are defined as children between the ages of 9 months through 9 years. In order to determine the percent of the population sampled in this program, the total number of children in the Basin Area

between the ages of 9 months and 9 years was estimated. Three sources were considered in determining this estimate. The first was the 1990 census data. Census estimates are available for both Shoshone County (a major portion (73%) of the total Basin Population) and the Basin Area as a whole. The second source is the school district enrollment data. Not all children living in the Basin attend one of the three districts, for example, some attend private schools or are home schooled. Based on Basin Area census estimates, children attending private school comprise approximately 5% of the entire student population. The third data source is "Kids Count" estimates of the child population in Shoshone County. All three sources and the sample populations based on these sources are summarized in Table 3-16. School District enrollment totals do not include students within the BHSS, but were increased by 5% to account for students enrolled in private schools. Although it was observed in the BHSS that the number of children in younger age brackets decreased in recent years, it was difficult to obtain estimates of the number of children in different age brackets in the Basin, a large area crossing county boundaries and school districts (TerraGraphics 2000a). The following method estimates the sample population for 0-9 year olds, assuming an even distribution of children in each age group (Table 3-15).

1. The total number of children from a certain age range as given in a source (e.g., 2484 children in the Basin Area between 0-17 years, as referenced from the 1990 census data) was divided by the total number of years in the age range (18). The result is the number of children in each age group.
2. Children eligible for the sample program span a total of 9 1/3 years. Therefore, the number obtained in step 1 (children in each age group) was then multiplied by 9.33 years (9 years + 3 months) to determine the estimated sample population.

The results from Basin Area 1990 census data as well as the Shoshone County estimates are slightly higher than the school district data. The reason for the discrepancy is likely a small number of children living outside of the three school districts and portions of Shoshone County not included in the Basin Study Area. The sample population estimates range from 1025 (2000 school district data) to 1288 (1990 Basin Area Census data). Assuming a 13% decrease in enrollment since 1990 yields an estimated 9 month to 9 year old childhood population of approximately 1120 children based on the 1990 census data. The estimated childhood population for the eight geographic subareas of the Basin is then 1025-1120 individuals based on the school district and 1990 census data, respectively.

Estimation of the Total Number of Housing Units and Yards

Basin Area sampling has included soil samples from yards in Basin Area housing units. In order to determine the percentage of yards that have been sampled it is necessary to know the total number of yards in the Basin Area. This was estimated by two methods. The first estimate was based on housing unit data from the 1990 census and the second was based on a combination of the current number of sewer hook-ups in the area and 1990 census data.

Census data were used to estimated the number of housing units by assuming that each housing unit counted in the 1990 census corresponded with one yard. However, housing unit, as defined by the

census, is a house, an apartment, a mobile home, a group of rooms, or a single room that is occupied (or if vacant, intended for occupancy) as separate living quarters. Therefore, there is not necessarily a yard associated with each unit (e.g., an apartment) and the estimate of yards based on census housing units would likely be an overestimate. The percentage of total housing units in the Basin Area with two or more units in the building, however is small (7%), with the majority of all units being either single unit dwellings or mobile/trailer homes. The total number of housing units in the Basin Area based on 1990 census data, with and without the Lower Basin Area included, is 5651 and 3740, respectively (Table 3-17). Both numbers exclude housing units within the BHSS. The number of yards in the Basin Area was then estimated by subtracting 7% of the total number of housing units. The resulting estimate of the total number of yards in the Basin Area based on 1990 census data is 5255. The number of yards in the Basin Area (excluding the BHSS and the Lower Basin Study Area) was estimated the same way at 3728. The Lower Basin Area was excluded in the second estimate for future comparison with sewer district data.

The second method used to estimate the total number of yards was done by combining current information on the number of sewer hookups in the Basin Area (with the exception of the Lower Basin) with 1990 Census data. Data was used from two sewer districts, the South Fork Coeur d'Alene River Sewer District and the Kingston-Cataldo Sewer District. Sewer district data for the Lower Basin Area was not readily available and was not included. It was possible with sewer district data to separate apartment buildings and their individual units so that only one yard was associated with each building, however, some units are not on public sewer (e.g., units using septic tanks) and therefore the housing unit estimate based on sewer data alone would be low. Data on the number of septic tanks in the Basin Area was not available because permitting was not required until the 1970s and septic tanks installed before then would not be counted. In addition, between the 1970 and 1990, many of the permits issued were not available on record.

In order to account for housing units not on public sewer, the percentage of housing units from the 1990 census data not on public sewer was assumed to be similar to current percentages.
Basin Area public water and sewer data are summarized in Table 3-17. Data for the Basin Area excluding the Lower Basin is shown for both the 1990 census and data collected by the sewer districts in the Basin Area in 1999. The 1990 census data indicated that approximately 72% (2873) of the housing units in the Basin Area w/o the Lower Basin were serviced by public sewer. The number of public sewer hook-ups in the same area in 1999 was estimated by the sewer district at 3065. The total number of housing units in the Basin Area w/o the Lower Basin in 1999 (italicized) was then estimated by assuming that 3065 units make-up 72% of the total number of housing units. The resulting estimate of the total number of housing units in the Basin Area without the Lower Basin is 4257.

After determining the total number of housing units using sewer district data, buildings with multiple housing units but only one yard (apartments and duplexes) were counted as one yard and the remaining number of units was subtracted from the housing unit total to estimate the total number of yards. It was assumed that housing units with septic tanks were not multi-unit dwellings. The estimated number of yards as shown in Table 17 is 3570. This number does not include yards in the Lower Basin. The

number of yards as estimated strictly by census data was 3728 excluding the Lower Basin and 5255 including the Lower Basin.

Housing unit estimates and corresponding yard estimates separated according to Basin Study Area are summarized in Table 3-18. The sewer hook-up data shown in Table 3-18 was obtained from 1999 sewer district data. The estimates are based on the percentage of public sewer hook-ups being 72% of the total number of housing units as described above.

3.1.3 Human Health Exposure Areas

It was necessary to establish geographical areas on the basis of potential human exposure. Within the CSM units, nine geographic areas were identified as human health exposure areas according to the route of human exposure evaluated and the public use patterns in each area (Figure 3-1). As noted in Section 2.2, only portions of the large areas identified on Figure 3-1 have been sampled. Evaluation of human exposure is applicable only to the sampled public areas shown on Figures 3-12 to 3-26, and to sampled residences. Risk management decisions will not be made for unsampled areas in the absence of data. The nine geographical areas are listed below.

- Lower Basin,
- Kingston,
- Side Gulches,
- Osburn,
- Silverton,
- Wallace,
- Nine Mile,
- Mullan, and
- Blackwell Island.

The Lower Basin includes all of the Coeur d'Alene River west of Cataldo (CSM Unit 3). Human health concerns in Coeur d'Alene Lake (CSM Unit 4) from exposure to metals through surface soil, sediment, and surface water were evaluated in the expedited screening level risk assessment for common use areas (Appendix B). In that assessment, all sites except Harrison Beach and a recreational area on Blackwell Island passed the screening process and, therefore, required no further evaluation. Harrison Beach has been evaluated as part of the Lower Basin. The Spokane River (CSM Unit 5) has been evaluated separately.

3.1.4 Characterization of Potentially Exposed Populations

Residents and visitors to the Basin could be exposed to affected media during their normal daily activities including home life, recreation, and work. However, exposure of individuals to affected media will not be the same across the Basin because of differences in the following factors:

- Location of their home,
- Affected media in the areas in which they spend time,
- Frequency of use of the local recreation areas, and
- Availability and use of public services (i.e., drinking water).

For example, concentrations of metals in media within the Basin are not uniform, indicating that some residents will be exposed to lower concentrations in their home than others. Some individuals may have private gardens or collect, grow, and/or eat local vegetables, fruit, livestock, fish, and wildlife, while others may not. Some residences and businesses have private drinking water supplies, while others use a municipal source. Because individuals in the Basin live in different areas, under different conditions, and in different lifestyles, exposures from place to place and person to person can differ significantly.

However, evaluation of exposure for each individual is neither practical nor useful in determining appropriate remedial action. Therefore, individuals have been combined into groups of major receptor types that have similar exposure to affected media in terms of the type and extent of exposure. Although, remedial decisions will be made on a site by site basis. No remedial actions will take place in the absence of site specific data. The following subsection describes the major receptor types, as well as cases in which multiple exposures may occur as a result of participation in multiple activities within the Basin.

Major Exposure Scenarios and Potential Receptors

Residential. Both children and adults who reside in the Basin could be exposed to several of the affected media while living in their homes. Residential exposure scenarios are based on judgements about activities that might be undertaken by Basin residents but may not necessarily result in exposure. Many daily activities that could result in exposure can occur both within and outside the residence. Regular use and maintenance of the home and yard as well as leisure activities create the potential for exposure to affected media. In addition, residents may grow vegetables or eat locally grown produce and thus be subjected to exposures via the food chain.

Neighborhood Recreational. In addition to exposure in their homes and yards, residents might have other opportunities to be exposed to affected media within their local neighborhoods. Residents who live very near affected creeks or rivers and their shores, near local parks or schools with affected media, or near waste piles of rock or tailings might also be exposed to affected media during leisure and recreational activities near their home.

Public Recreational. Recreational exposure in neighborhoods is likely similar to that in public recreational areas. However, individuals from outside the Basin might use public recreational areas while not being exposed to affected media in their homes. Similarly, residents of the Basin might travel to public recreational areas in different locations within the Basin. For example, a resident of a rural upriver community might choose to travel to recreational areas in the Lower Basin, which are unavailable in the steep canyons upriver. Public recreational exposure was quantified separately from

residential and neighborhood recreational exposure because of the potential for cross-Basin travel and the possibility that visitors from outside the Basin will use public recreational areas within the Basin.

Occupational. Individuals in the Basin may come in contact with affected media while performing their daily work activities. In general, occupational exposures are less significant than residential and recreational exposures because of limited contact with affected media. However, for some workers, such as individuals who have intensive contact with soil during excavation work, exposure might be relatively high for short periods, depending on the work location. Therefore, intensive occupational exposure to affected surface and subsurface soil was evaluated for a "construction worker." Other potential on-the-job exposures, such as drinking tap water or coming in contact with surface water or sediment, were not evaluated because no exposure or infrequent, minor exposure is expected to occur or because the type of exposure was already being evaluated for another major receptor type.

Subsistence. Coeur d'Alene Tribal resident activities extended throughout the Basin. Site-specific media contaminant levels are used in the HHRA for available data collected near the mouth of the Coeur d'Alene River and the Chain Lakes area. Traditionally, tribal members occupied many areas of the Basin and utilized the resources that each area had to offer, especially the water bodies and waterways. For the purposes of the HHRA, Coeur d'Alene Tribal authorities have requested that two specific tribal exposure scenarios be investigated, developed, and utilized. Those scenarios are the Traditional Tribal Subsistence Lifestyle and the Current Subsistence Lifestyle. The Traditional Subsistence scenario considers the aboriginal riparian resident lifestyle traditionally practiced by the Tribe. The Current Subsistence scenario considers those tribal members that continue to practice a subsistence lifestyle today.

Traditional Subsistence activities were carried out in numerous locations throughout the Coeur d'Alene Basin and included diverse locations. Current contaminant concentrations vary widely by media and geography throughout this area. The two exposure scenarios utilize the available site-specific sediment, soil, and tissue exposure point concentrations data obtained from the mouth of the Coeur d'Alene River and for the Lower Basin area, including the Chain Lakes. This area corresponds with one of the main Tribal units resident locations in traditional times, and a potential future harvest area for the current subsistence scenario.

Multiple Exposures. Individuals in the Basin might fit into more than one receptor type and therefore be exposed to affected media in more than one way. Some examples include the following:

- Local residents who are also exposed during neighborhood recreation (including exposures via the food chain),
- Local residents who also use recreation areas in the Basin (including exposures via the food chain), and
- Local residents who also work locally as construction workers.

The related increases in incremental risk due to multiple exposures are discussed in Section 5.

Potential Exposed Residents by Area

Potential human receptors who might be located in each of the human health exposure areas are described in the following subsections and are summarized in Table 3-19a.

Lower Basin. The Lower Basin includes the lower Coeur d'Alene River and its associated floodplain. Residential population density is sparse throughout the area in Cataldo, Dudley, Rose Lake, Lane, and Medimont. Both adult and child residents in the Lower Basin could be exposed to affected media. Residents who live near the lower Coeur d'Alene River might also be exposed to affected media in their neighborhood while engaging in recreational activities. Although both adults and children could be exposed, exposure is likely greater and more frequent for children who play for longer periods of time, such as children between 4 and 11 years old. The ample available natural resources and public recreation in the Lower Basin also provide an opportunity for visitors to the area to be exposed to affected media in public recreation areas including the lower Coeur d'Alene River and associated beaches and picnic areas. It is also possible that construction and excavation workers in the Lower Basin might be exposed to affected soil.

Kingston. Within the Kingston area are the confluence of the North Fork and the South Fork (NS confluence), Pine Creek, and their extensive tributaries. The upper portions of Pine Creek were considered relatively uncontaminated and, therefore, were not evaluated in the HHRA. Residences in the Kingston area are sparse throughout the area in the town of Kingston, as well as along Pine Creek, and in Enaville. (The portion of Pine Creek that is within the boundary of the 21-square-mile Bunker Hill Superfund Site has been excluded from this evaluation.) Both adult and child residents in the Kingston area could be exposed to affected media. Residents who live near the NS confluence or Pine Creek might also be exposed to affected media in their neighborhood while engaging in recreational activities. Although both adults and children could be exposed, exposure is likely greater for children between 4 and 11 years old. Although less accessible than those in the Lower Basin, the public recreation areas in the Kingston area, specifically the NS confluence, also provide an opportunity for visitors to be exposed to affected media in beach and picnic areas. It is also possible that construction and excavation workers in the Kingston area might be exposed to affected soil.

Side Gulches. The Side Gulches include Moon Creek, Two Mile Creek, Terror Gulch, Montgomery Gulch, and Nuckols Gulch, Sunny Slopes, the lower portion of Big Creek, and Elk Creek Pond and its surrounding area. Residences in the Side Gulches area are located primarily along Big Creek with sparse households along the other creeks and gulches. Both adult and child residents in the Side Gulches area near Big Creek could be exposed to affected media. Residents who live near Elk Creek Pond and its surrounding area might also be exposed to affected media in their neighborhood while engaging in recreational activities. Although both adults and children could be exposed, exposure is likely greater for children between 4 and 11 years old.

Osburn. Both child and adult residents of the town of Osburn could be exposed to affected media in their homes and yards. Residents who live near the South Fork could also be exposed to affected

media while playing in their neighborhood. It is also possible that construction and excavation workers in the town might be exposed to affected soil.

Silverton. Both child and adult residents of the town of Silverton could be exposed to affected media in their homes and yards. Residents who live near the South Fork could also be exposed to affected media while playing in their neighborhood. Children who play at neighborhood parks and schools with affected media could be exposed. Visitors to public parks and schools in the area might also be exposed to affected media in these areas. (See Section 2.2 for a list of parks and schools.) It is also possible that construction and excavation workers in the town might be exposed to affected soil.

Wallace. Both child and adult residents of the town of Wallace could be exposed to affected media in their homes and yards. Residents who live near the South Fork of the Coeur d'Alene River could also be exposed to affected media while playing in their neighborhood. Children who play at neighborhood parks and schools with affected media could be exposed. (See Section 2.2 for a list of parks and schools.) Visitors to public parks and schools in the area might also be exposed to affected media in these areas. It is also possible that construction and excavation workers in the town might be exposed to affected soil.

Burke/Nine Mile. The Burke/Nine Mile area includes the residents in the communities of Mace, Burke, Gem, Blackcloud, and Woodland Park, which are located along Nine Mile and Canyon Creeks. Both child and adult residents of these small rural communities could be exposed to affected media in their homes and yards. Residents who live near Nine Mile and Canyon Creeks could also be exposed to affected media while playing in their neighborhood. Children who play on waste piles in their neighborhood could be exposed to the highest concentrations of metals (see list of waste piles in Section 2). It is also possible that construction and excavation workers in these communities might be exposed to affected soil.

Mullan. The Mullan area includes the area in and around the town of Mullan and the uppermost portion of the South Fork. Both child and adult residents of this community could be exposed to affected media in their homes and yards. Residents who live near the South Fork, which includes most of the residents in the area, could also be exposed to affected media while playing in their neighborhood. Children who play on waste piles in their neighborhood could be exposed to the highest concentrations of metals. Waste pile exposures are quantified and evaluated for Morning Mine Dump in the outskirts of Mullan. It is also possible that construction and excavation workers in these communities might be exposed to affected soil.

Blackwell Island. On Blackwell Island, there is a recreation area with beach and picnic areas that can be accessed by the public. Visitors to this area could be exposed to affected media on and around the island.

3.1.5 Populations of Potential Concern

Certain populations in the Basin could be more sensitive to contamination or more likely to be subjected to greater exposure than the average individual in each of the receptor groups. These populations include infants and children and individuals with subsistence lifestyles, including some members of the Coeur d'Alene Tribe. The following subsections briefly describe these populations in terms of the characteristics that either make them more sensitive or more likely to have greater exposure. Section 4 and Appendix H provide greater detail of the effects of each chemical on the populations of concern.

Infants and Children

Because of their physical vulnerability and small body size, infants and children are often assumed to be more susceptible to the potential toxic effects of chemicals in the environment. Studies have shown that susceptibility clearly depends on the chemical and on the exposure situation. Although these differences are chemical-specific, infants and children are a unique population that needs to be considered in risk assessments (Guzelian, Henry, and Olin 1992). Their risks may differ qualitatively and quantitatively from those of adults for a variety of reasons including differences in behavior (i.e., frequent hand-to-mouth behavior), physiology, metabolism, pharmacokinetics, diet, and exposure environment.

Some of the COPCs evaluated in this risk assessment are known developmental toxicants. Because chemicals can cross the transplacental barrier, pregnant women and women of child bearing age are also considered a sensitive population in order to protect the developing fetus. The toxicity profiles in Appendix H contain further chemical specific details regarding the effects of developmental toxicants.

Aspects of physiology that differ between children and adults include differences in intake per unit of body weight of air, food, and water (and associated chemicals). These differences are related to differences in rates of respiration and circulation and cell proliferation rates in many organs, which are often greater in children than in adults.

Similarly, dermal, intestinal, and respiratory absorption may be greater or lesser in children depending on the chemical and the exposure scenario. During times of rapid growth, the amount of food ingested per unit of body weight may be greater for children (Plunkett, Turnbull, and Rodricks 1992).

There are major metabolic differences between children and adults that can significantly affect their ability to respond to chemical exposure. Some metabolic systems are more efficient in childhood than during adulthood (such as cytochrome P-450 activity) while others are less efficient. Chemical-specific metabolic differences between children and adults are also evident (Guzelian, Henry, and Olin 1992).

Pharmacokinetics, including the absorption, distribution, and excretion of various chemicals, differs between children and adults on a chemical-specific basis. These differences are sometimes a result of developmental changes in membrane permeability and in the binding and storage of chemicals (Guzelian, Henry, and Olin 1992; Plunkett, Turnbull, and Rodricks 1992; Faustman et al. 2000; Goldstein 1990; Rodier 1995).

The diet of a child is often quite different from that of adults. Dietary differences, such as the amount of vegetables, fruit, fish, or red meat consumed, can have an effect on the amount of chemical ingested in food items. In addition, nutritional status has a profound effect on toxicity response.

One of the most obvious differences between adults and children is the difference in physical environment and living habits. For instance, children are generally closer to the floor, carpet, and ground; their daily activities and hand-to-mouth behavior significantly influence the amount of chemical exposure that occurs. Secondary exposures to children from materials brought home from the parents work areas (particularly construction sites) may also affect the residential dust exposure scenario.

As a result of these influential differences, infants and children often receive a different effective dose of a chemical than adults, even when chemical concentrations in affected media are the same. Therefore, in this assessment, children were considered a sensitive population for all COPCs and were evaluated appropriately. Specifically, both children (0 to 6 years of age) and adults were evaluated for residential and public recreational exposure types. Neighborhood recreational exposures were evaluated for children between the ages of 4 and 11 because maximum exposure was anticipated in this age group as a result of their play habits and their interaction with the physical environment.

Subsistence Lifestyles

The traditional economy of the Coeur d'Alene Tribe was characterized by a complex and highly structured system of food source production, distribution, and consumption. The Plateau people generally practiced a seasonally based transhumance, an annual cycle of utilization of specific economic sources. This travel involves the return annually to well known camps for root digging, fishing, hunting, and high elevation hunting and berry picking. The basic winter village in the Basin is the center of the cycle and is never fully abandoned by certain individuals of the society, especially the elderly and children too young to travel on their own but too heavy to be carried (Sprague 1999).

The following paragraph from Teit compares the Coeur d'Alene to the rest of the Plateau Culture Area (including the Umatilla Tribe):

> At certain seasons considerable numbers of people congregated at famous camas and other root-digging grounds. They also went to the Spokane for salmon fishing, trading, and sports. These journeys were made on foot, for there were no water routes leading to these places. On the whole the people were fairly sedentary, and most of them lived the greater part of the year on their home grounds, although they had no permanent houses or villages, unless the long communal dance houses of the larger villages may be so called. Being a semisedentary people, and living in a country where wood, bark, and vegetal materials of many kinds abounded, the Coeur d'Alene developed the arts of fishing, canoe making, and textile work in weaving of mats, bags, and baskets, probably to a greater degree than any of the neighboring tribes (Teit 1930:151).

There were "three, possibly four, units corresponding to divisions of the tribe." These were (1) Coeur d'Alene Lake and/or Spokane River; (2) Coeur d'Alene River; and (3) St. Joe River (Teit 1930). Because of their proximity to the fishing grounds at Spokane Falls the first group tended to be more permanent. The second group was located around the mouth of the Coeur d'Alene River and upstream near Harrison, Medimont, and between Killarney Lake and Robinson Creek near what is now Lane. The third group resided at the mouth of the St. Joe River and west of Mission Point on Lake Coeur d'Alene; the six villages in this group extended upriver as far as modern St. Maries (Teit 1930:38-39).

The Coeur d'Alene were largely dependent upon Lake Coeur d'Alene and its tributaries; perhaps more than any other Plateau group. Water played a central role in all aspects of life, from birth to death and was included in all major cultural events. Individuals spent a great portion of their time in the water; generally through fishing, hunting, gathering, bathing, recreating, and other various activities. The special emphasis on water and its ritual importance is demonstrated in a statement by Father Point in 1843 that the sign of the cross was made at all important events but especially always before smoking the pipe or drinking water (Point 1967:94).

The Coeur d'Alene were also involved in harvesting, consuming, and utilizing primarily riparian resources. Much of the raw material used in the manufacture of various necessary items was obtained primarily from within the riparian environment (Nugent 1997). The areas of manufacturing that the Coeur d'Alene were especially noted were in the use of bark and mats. Mats were made of the cat-tail and tule plants, both of which grow in water. Mats were made by weaving and sewing and were used as lodge covers. The preparation of hides involved soaking in water for long durations to loosen the hair. The finished product was often painted green from an algae collected from stagnant pools. For stone-boiling the Coeur d'Alene used woven baskets like their neighbors but unlike several others, they also used bark containers (Ray 1942:136).

Seasonal round activities were also intimately associated with tribal social, political, and religious organization. Many aspects of traditional Coeur d'Alene tribal organization were based upon gender and age, with strong incentives for conformance. Men were mainly involved with hunting and fishing, but also participated in gathering. Women were mainly involved with gathering, fishing, and food processing and preparation. The division of labor was distributed evenly among tribal members. Children also helped with the work, particularly gathering and fishing.

The division of the harvest took place according to a certain political structure. The individuals responsible were given a large portion of the kill, while the particular chief involved with the hunt received a portion for distribution to needy individuals within the tribe (Nugent 1997).

All of these activities are undertaken collectively in family or tribal groups and involve children and women of reproductive age, that are considered the population at greatest risk. These activities also result in substantially greater potential exposures associated with consumption rates of resident fish and riparian vegetation, and soil and sediment contact rates associated with typical residence and harvest practices for both ingestion and dermal routes. Due to the Tribe's dependence on water from Lake

Coeur d'Alene, the surrounding lateral lakes, and the Coeur d'Alene River and close interaction with the natural environment, maximum exposures were assumed.

3.2 IDENTIFICATION OF EXPOSURE PATHWAYS

An exposure pathway represents the course a chemical takes from its source to the exposed individual. An exposure pathway consists of four parts: (1) a source and mechanism of chemical release, (2) a retention or transport medium, (3) a point of potential human contact with the affected medium, and (4) an exposure route at the contact point (USEPA 1989). Without all of these four parts, a potential exposure pathway is not complete and, therefore, not a risk to human health.

Exposure pathways that were considered in this HHRA include standard pathways common to most HHRAs at hazardous waste sites. A few less common pathways relevant to exposure in the Coeur d'Alene River Basin were also considered. The following points of potential human contact and the potential exposure routes for each of the receptor types were considered and are discussed in the subsequent sections:

- Ingestion of and dermal contact with soil and house dust (residential, neighborhood recreational, public recreational, occupational, and subsistence residents),
- Inhalation of dust from soil (residential and subsistence residents),
- Ingestion of and dermal contact with tap water (residential residents),
- Ingestion of and dermal contact with surface water (neighborhood recreational, public recreational, and subsistence residents),
- Ingestion of and dermal contact with sediment (neighborhood recreational, public recreational, and subsistence residents),
- Ingestion of homegrown vegetables (residential residents),
- Ingestion of locally grown beef (residential residents),
- Ingestion of fish (public recreational and subsistence residents),
- Ingestion of wild game (public recreational and subsistence residents), and
- Ingestion of wild plants (residential and subsistence residents).

All the pathways discussed in the following sections apply to both current and future conditions except future use of groundwater (Nine Mile area only) and future tribal use of the floodplain of the lower Coeur d'Alene River (Lower Basin). Section 3.2.1 describes the sources, fate and transport mechanisms, and affected media in the Coeur d'Alene Basin. Section 3.2.2 presents the potentially complete exposure pathways that were not selected for quantification in the HHRA, as well as the rationale for exclusion. Section 3.2.3 presents the potentially complete exposure pathways that were quantified in the HHRA by exposure area. The potential exposure pathways that were considered for the Coeur d'Alene Tribe traditional and current subsistence are described in Section 3.2.4.

3.2.1 Sources, Fate and Transport, and Affected Media

Diagrams of potential sources, fate and transport mechanisms, and affected media are shown in Figures 3-3 through 3-11 for each geographic area. These figures also show the potential receptor types and exposure media that are discussed in Sections 3.2.2 and 3.2.3. A general description of possible sources, transport mechanisms, and media is provided in the following text; however, not all of these sources, mechanisms, and media are present in each area.

As a result of mining activities in the Basin, surface and subsurface soils containing high concentrations of metals have been disturbed and redistributed throughout the Basin to construct roads and railroad beds, to supplement agricultural areas, and to be stored in waste piles. Mining activities have caused releases of concentrate, mill tailings, and waste rock to contaminate surface and subsurface soils with high concentrations of metals. This material has subsequently been eroded and transported along the Coeur d'Alene River. It has also been used as fill in construction of roads, Interstate 90, and the Union Pacific Railroad. When soils with high concentrations of metals are exposed to air, a natural geochemical process can occur in which the sulfide form of the metals is oxidized. These oxidized metals can adsorb to soil particles and be transported from the landscape with the soil via surface runoff and wind and water erosion to nearby creeks and rivers. Many of the metals in the creeks and rivers are adsorbed to transported soil particles as well as cobble, particulate matter suspended in the water column, and sediment. Some metals are released into the water column via dissolution. As a result of the frequent flood events that occur in the Basin and the natural migration of the river channels, this sediment and other organic material have been resuspended, carried, and redeposited in many downstream locations within the floodplain, into Coeur d'Alene Lake and beyond. This redeposited sediment can cover upland areas or remain in downstream channels as bedload sediment.

Metals adsorbed to soil particles can also be released via dissolution and become soluble in rainwater and soil pore water that eventually percolate through the soil layers to groundwater. Likewise, metals adsorbed to soil particles can be transported via wind erosion as fugitive dusts that eventually settle on surrounding areas.

As a result of these processes, high metals concentrations originating in subsurface soil deposits have been transported to and detected in adjacent surface soil, groundwater, surface water, sediment, upland soil, and dust.

3.2.2 Potentially Complete Pathways Excluded From Quantification

Dermal Contact With Tap Water and Surface Water

The uptake of inorganic chemicals through the skin from water is primarily limited to compounds dissolved in water. While water soluble metals are absorbed at higher rates than insoluble ones, the penetration rate of water through the skin is slow (0.001 cm/hour) (USEPA 1992a). Several investigators have also shown that electrolytes in dilute solution penetrate the skin poorly (USEPA

1992a). Absorption rates similar to that of water have been observed for the chloride salts of zinc, cadmium, and mercury, and for sodium chromate and silver nitrate (Wahlberg 1968; Skog and Wahlberg 1964). The recommended dermal permeability factor for metals is quite low at 0.001 cm/hour (USEPA 1998b) and it applies only to the dissolved fraction. Therefore, dermal contact with metals in tap water and surface water, although a complete pathway, was not quantified in the HHRA.

Inhalation of Fugitive Dust

Inhalation of dust has been evaluated in previous risk assessments in the Basin and was determined not to be a primary contributor to exposure and risk (Weston 1989), although inhalation was identified as a risk at the Bunker Hill Superfund Site (USEPA 1990a). Changes that have occurred at the Bunker Hill Superfund Site since the risk assessment have resulted in decreased dust concentrations from metal source areas at the site. The screening process used to select COPCs examined available soil data and screened out all chemicals in this pathway because measured concentrations were less than the SVs. The SVs were estimated using the PEF, as discussed in Section 2.4.1, which relates the chemical concentration in soil with the concentration of dust particles in the air. In addition, the results of ongoing dust monitoring indicate that this exposure pathway is being sufficiently controlled to preclude significant exposures. See Section 7.2.2 for further discussion of this pathway.

Ingestion of Beef Cattle Grazing in Floodplain

Cattle graze in the floodplain meadows along the lower reaches of the Coeur d'Alene River. There are four known ranching operations in the floodplain (USFWS 1999). Because the meadows are periodically inundated with floodwaters, the meadow soils are contaminated with mining-related chemicals. Therefore, grasses in these meadows may take up inorganic chemicals, which may then be eaten by grazing cattle. Furthermore, cattle directly ingest soil on and around the floodplain grasses. Therefore, the consumption of local beef may result in the ingestion of some inorganic chemicals.

Ingestion of metals in beef could occur. However, because of the absence of beef tissue data, this pathway was not quantified in the HHRA. The consequences of excluding this pathway are discussed in Section 7.

Ingestion of Waterfowl and Large Game From Hunting

Waterfowl and large game that feed, live, and/or breed in the area are exposed to inorganic chemicals via the consumption of potentially affected plants and the incidental ingestion of affected soil. Dermal contact with soil and ingestion of and dermal contact with contaminated water are also routes that might affect metal concentrations in waterfowl and large game. Previous investigations have shown that lead and cadmium accumulate in blood and some tissues of small mammals and birds in the Basin (Szumski 1999; Audet et al. 1999). Waterfowl deaths due to acute lead toxicity have been documented (Audet et al. 1999).

Both residents and nonresidents might hunt, capture, and eat waterfowl and large game in the area, thus being exposed indirectly to inorganic chemicals. Although this pathway is complete, it is not expected to result in a significant exposure for the majority of the population, and, therefore, it was not evaluated quantitatively for metals other than lead. Exclusion of this pathway for waterfowl is supported by previous Basin studies that investigated tissue metal concentrations in waterfowl (Weston 1989). Results indicate that although metals tend to accumulate in kidneys of ducks collected within the Coeur d'Alene Wildlife Management Area, the concentrations are not high enough to pose a health threat due to the consumption of other tissues (Weston 1989). A study conducted by the Idaho Department of Fish and Game in August 1986 found that cadmium and lead were not detected in most duck breast tissue sampled even though both metals were detected in significant concentrations in kidney, liver, and bone. Similarly, zinc was detected in breast tissue at concentrations 50 to 90 percent lower than those in kidney, liver, and bone (Krieger 1990). Therefore, this pathway was not quantified in the HHRA.

For the subset of the population who hunt big game, there is a potential for metals exposure, particularly from white-tail deer who may spend an appreciable portion of their lives grazing in the floodplain of the lower Coeur d'Alene River. Unlike waterfowl, tissue data and other supporting studies are not available for any big game animals (one sample of white-tail deer muscle is all that is available). Two considerations may mitigate metals exposure from game: 1) divalent metals such as cadmium, lead and zinc are generally transported within the animal's body via the blood and stored or retained primarily in the spleen, kidney, bone, and liver, and 2) the big game catch limit of one or two animals per year would limit the amount of organ meat any one family could eat. Tissues normally eaten by hunters, primarily muscle and fat, contain lower concentrations of metal than the bones and organs (Benson et al.1976). However, "lower concentrations in tissue" may still have health impacts depending on the actual concentrations. In the absence of data, this pathway has not been quantified in the risk assessment and remains a source of uncertainty. This pathway is further discussed in Section 7.

Ingestion of Wild Plants Harvested From Floodplain

Three main types of plants are reportedly gathered from the floodplain area: berries, wild rice, and water potatoes. Uptake of metals by plants is minimal, indicating exposures due to their consumption would also be low. Except for cadmium, these metals do not tend to bioaccumulate in plant tissue (Nwosu, Harding, and Linder 1995). For example, the recommended root uptake factors (ratio of concentration in plant tissue to the concentration in the soil) for arsenic, cadmium, lead, and mercury are 0.0004, 0.04, 0.002, and 0.05, respectively (CalEPA 1996). However, other studies have shown that concentrations of cadmium in some plant tissues are higher than those in surrounding soils (Nwosu, Harding, and Linder 1995). Because the general population is not expected to eat a significant quantity of these plants, this pathway was not quantified for the general population in the HHRA. The ingestion of water potatoes was quantified for subsistence exposures.

Concentrations of metals in wild plants will likely be small in comparison to concentrations in soil and/or sediments that might be directly ingested and when compared with other homegrown food items. The direct ingestion of soil and sediment and the ingestion of homegrown vegetables were quantitatively

evaluated and risks from eating wild plants will be insignificant when compared to the risks from these pathways.

3.2.3 Complete Pathways Selected for Quantification

Ingestion of and Dermal Contact With Soil

Affected soil is the primary medium with which residents, neighborhood recreational residents, public recreational visitors, and workers are likely to come into contact. Therefore, exposure by incidental ingestion is likely and was quantified in the HHRA. This same affected soil is a primary source of indoor dust in residences. The contribution of indoor dust to risk was evaluated and is discussed qualitatively in this assessment for the non-lead metals. Indoor dust exposures are quantified for lead.

In general, metals in soil are strongly adsorbed and will not leach except under strongly acidic conditions. Soil-water partition coefficients (ratio of concentration in soil to concentration in water when both are available) for metals in the Basin range from 25 to 900, indicating that the metals are strongly adsorbed to soil (RAIS 1999). Therefore, absorption of metals through the skin is probably very slow.

Although dermal absorption of metals in soil through the skin is a complete pathway, available data indicate that the contribution of dermal soil exposure to overall risk is typically small (USEPA 1995b, 1996a). Furthermore, data on the amount of metals in soil absorbed through the skin is extremely limited (USEPA 1992a). However, dermal absorption of metals through the skin was quantified for arsenic and cadmium only, for which limited skin absorption data exist (USEPA 1998e, 1999c). In addition, these metals are more mobile in the environment than other metals because cadmium is soluble in water and arsenic has multiple oxidation states (USEPA 1985).

Ingestion of Tap Water

Metals have been detected in tap water at several locations. Ingestion of tap water is a significant source of intake into the body, so this pathway was included wherever residents or recreational visitors may have access to water containing mining-related chemicals. Only arsenic exceeded conservative screening criteria protective of human health. Therefore, of the non-lead metals, only arsenic was evaluated for the tap water ingestion pathway. Lead in tap water was evaluated quantitatively because the risk assessment for lead utilized the EPA IEUBK lead model which attempts to account for all sources of lead in the environment, not just sources with concentrations that exceed SVs.

Ingestion of Surface Water

Surface water in the Basin has been found to contain mining-related chemicals. Contact with surface water may result in exposure via incidental ingestion and dermal absorption (qualitatively evaluated). This is most likely to occur at sites where access to water is easy and attractive, such as developed or

undeveloped beaches along the Coeur d'Alene River west of Cataldo. At these sites, recreational visitors or local residents may be exposed to contaminants in surface water while playing in the water. This is of particular concern where play in shallow water may disturb sediments and suspend them in the water column. In such cases, incidental ingestion of surface water containing suspended sediments may result in a significant dose. This pathway was quantified in the HHRA.

There are residents with ready access to affected surface water within neighborhoods that lack beaches or other amenities to attract visitors from beyond the immediate area. For example, children living close to Canyon Creek or Nine Mile Creek may play in the water near their homes. This may produce significant incremental doses; therefore, this pathway was included in the residential scenario at some locations (see Table 3-19a).

Ingestion of and Dermal Contact With Sediment

Ingestion of and dermal contact with sediment is likely to occur in the same situations as ingestion of surface water, as discussed in previous subsections. Sediment is defined as material transported by surface water and deposited along the banks of surface water bodies. Sediment includes the sand found at beaches, which may be above or below the water line. Sediments below the water line were evaluated in the surface water pathways described in the previous subsection, since they may be resuspended in the water column. Sediment above the water line may produce exposure through contact-intensive activities such as digging in beach sand. At beaches, the combination of exposed skin surface area and wet media may cause stronger adherence of sediments to the skin (Kissel et al. 1996a, 1996b, 1998; Holmes et al. 1999). This may lead to higher rates of incidental ingestion and prolonged dermal contact, so these pathways were quantified in the HHRA. As with soil, dermal contact was evaluated only for arsenic and cadmium (USEPA 1998e, 1999c). Exposure pathways for sediment were evaluated for the public recreational individual type and for appropriate residential neighborhood recreational receptor types.

Ingestion of Homegrown Vegetables

Consumption of garden vegetables was evaluated quantitatively. Currently, residents in the Basin grow and consume garden vegetables from yards that may contain mining-related chemicals. If either leafy (e.g., lettuce or cabbage) or below ground (e.g., radishes, beets, or carrots) produce was present at homes with vegetable gardens, a sample of the plant tissue was collected and analyzed for metals. These vegetable data were used to quantitatively evaluate exposures to metals via the ingestion of homegrown produce.

3.2.4 Potential Coeur d'Alene Tribal Pathways Selected for Quantification

Soil/sediment

The Coeur d'Alene made their homes largely in the floodplain in structures with soil floors covered with natural materials such as animal skins or woven mats. As a result, intense contact with soil and sediment would have occurred throughout the year in nearly all activities. The mat lodge, the basic structure for lodging, was rarely found anyplace but in the Lake Basin (Sprague 1999). Additionally, a variety of cultural activities would have resulted in increased soil and sediment exposure. These activities include children's games, vision quests, burials, and dances.

Children played various games involving strength and skill such as running, throwing balls and sticks, and shooting toy bows and arrows.

The vision quest involves long trips into remote areas. The activities of a person on a vision quest were to show evidence of being at a specific quest site and to tire oneself to become more susceptible to the spirit. The vision quest still has religious importance today.

"The Coeur d'Alene disposed of their dead by burial in the earth or in rock slides" (Ray 1942:237). The modern, Plateau-wide practice of each tribal member throwing in a shovel or hand-full of dirt into a new grave is reported as early as 1853.

Dances were largely for preparing for war or upon returning from war, spirit dances of individuals during the winter ceremonies, and social dances involving courtship (Teit 1930:186; Ray 1942:253). All of this activity normally took place in the floodplain of the Basin.

Surface Water

For the Coeur d'Alene, the number of activities taking place in the water are almost as extensive as the time spent on land, so the incidental ingestion of surface water could contribute substantially to exposure.

While fishing, a good portion of time was spent in the water. The following standard fishing techniques were employed by the Coeur d'Alene (Teit 1930:105-107; Ray 1942:104-116; for illustrations Walker 1967): the spear (harpoon and leister types), gaff, dip net, and hook and line. Other fishing techniques more akin to gathering were traps, weirs, dams, and nets. The weirs were a community effort and various ceremonies accompanied the construction. It involved long periods of time in shoulder high water; women bore the major brunt of the time in the water.

The Coeur d'Alene were well known for killing large groups of deer in Coeur d'Alene Lake. If one trait in hunting separates the Coeur d'Alene from the other Plateau groups it is their ability to drive deer into the Lake and dispatch them in the Lake from canoes by spearing, shooting with arrows, clubbing, or drowning by holding the head underwater by the antlers or with a crooked stick. The Coeur d'Alene would also do the same to elk, moose, and bear (Teit 1930:101).

Beavers were hunted by destroying the dam or swimming into the beaver house and then clubbing them to death. Attacking the beaver by way of the water was limited in the southern Plateau to only the Coeur d'Alene (Ray 1942:119).

One water plant important to the Coeur d'Alene and not found among many other Plateau peoples, was the water potato. It grows in soft mud underwater and is collected by several methods, the most common being digging underwater with the hands or a forked stick.

A pregnant mother was encouraged to bathe frequently for an easy birth. At birth, the newborn was bathed immediately (Ray 1942:194, 196).

The specific importance of water in puberty was expressed: "The old people made boys and girls bathe in cold water every day. This was to make them strong, hardy, and able to endure cold. It was also believed to make them healthy, immune from colds and sickness, and able to recover quickly from wounds" (Teit 1930:168-169). Vision quest sites were often near bodies of water because diving in water was expected to help tire the individual (Ray 1942:236).

The Coeur d'Alene disposed of their dead by burial. Prior to burial, they would wash the body and wrap it in a robe, skins, or especially tule mats. People involved in the burial process were purified by fasting and would "bathe themselves in running water" (Teit 1930:174); their clothes were also purified by being "immersed in stream or lake overnight" (Ray 1942:220-221).

Games of athletic skill, especially swimming and canoe racing were popular (Ray 1942:185). All people could swim; the arms were worked in a dog-like fashion, and they struck out with the right foot. Nearly all the men could dive, and some men could dive across St. Joe River (Teit 1930:134). The Coeur d'Alene also participated in the popular sports of canoe racing and tipping (Kowrach and Connolly 1990:56, 198).

Curing by shamans involved water and included the sequence of hand washing, blowing on the water, throwing the used water out, obtaining more, and then sprinkling it on the patient. The patients hands were also immersed in water as part of the cure. The use of water in Coeur d'Alene curing exceeds that of all other Plateau groups (Ray 1942:242-243).

Groundwater Seeps/Spring Water

Because spring and small stream water was universally preferred to major river and lake water for drinking by Plateau peoples; this would be the expected source for drinking water. According to Ray, water was consumed prior to eating any food at feasts (Ray 1942:133). The numerous activities discussed under the surface water pathway could, under some conditions, be practiced in seep and spring water and result in the ingestion of water.

Steam (sweating)

Although only the dermal and inhalation pathways are considered in the sweat bath discussion, sweat bathing was done immediately prior to plunging in cold water, either surface water or spring water.

Sweat houses were the typical Plateau domed type made of willows covered with sod. They tended to be small by Plateau standards, holding only one or two people or up to six (Ray 1942:181; George 1938). They were always located near fresh water that provided the source for the cold water plunge.

Sweating was for cleanliness, curing, and purification such as before hunting, after a burial, or after a battle (Ray 1942:123, 127). Children were expected, under threat of whipping, to sweat and plunge in cold water every day of the year. The daily sweat could be individual but it often was also an important social activity where the two sexes were separated and could discuss the events of the day. The Coeur d'Alene are also unusual in that they have both the sweat house and a pool of water heated with rocks, a combination of traits not common in the Plateau (Ray 1942:181-182). The sweat bath is still very much a part of Coeur d'Alene culture.

Biota - Fish

The importance of various food types for the Coeur d'Alene was the same as for other Plateau groups, with fish and roots as the primary source and meat from hunting as the third source. Meat and fish were cooked by roasting when fresh and usually boiled if dried. Not only was fish tissue consumed, but other parts of the fish including skin, scales, bones, viscera, eggs, and head. Some Native Americans have traditionally eaten the bones and organs as well (Walker and Pritchard 1999:50).

Fish caught in Coeur d'Alene Lake or the rivers were dried and smoked in the normal Plateau manner. Fish would be collected locally or brought back from fishing or trading from all of the Plateau fishing centers including Kettle Falls, The Dalles-Celilo, and lesser known locations such as the Upper reaches of the North Fork of the Clearwater River and the Snake River at the mouth of the Palouse River at Palus Village. These trading adventures were more typical of young men and were as much a social activity as they were for fish. Such trading also gave the Coeur d'Alene a wider range of fish varieties, especially for some of the salmon varieties that were richer in oil. Dried fish were traded largely from the Spokane and to a lesser extent from the Palus (Teit 1930:112-113).

The Coeur d'Alene fishing territory extended from the North Fork of the Clearwater River to the southern margin to Lake Pend Oreille and included Coeur d'Alene Lake and its major tributaries - the Coeur d'Alene, St. Joe and St. Maries Rivers - the upper portion of the Spokane River to Spokane Falls, Latah (Hangman) Creek, and the headwaters of the Palouse River (Scholz et al. 1985:40).

Other sources of food from the water included the pond turtle, frogs, and crayfish. All of these are still used today. The fish in the diet of modern Coeur d'Alene is extensive but a large portion of the fish comes from the Columbia River system.

Biota - Water Potato

As previously mentioned, the water potato was an important plant to the Coeur d'Alene. Also known as the wapato on the lower Columbia River, it was collected the last week of October through November along the shore of Chatcolet, Hayden, and Harrison lakes; it was also historically collected in the floodplain of the Coeur d'Alene River Basin (Palmer 1993:17). The Tribe continues to harvest water potatoes during the last week of October through November in the wetlands of Benewah and Chatcolet. In a specific study of the Coeur d'Alene water potato, it was reportedly collected at 91 different sites (Frey 1995). One Coeur d'Alene elder described the loss of this plant food due to the rising of Coeur d'Alene Lake and the subsequent pollution as one of the major psychological losses to the Coeur d'Alene people (Aripa pers. com. 1994). In preparing the water potato, the dark skin layer is often removed prior to boiling or baking; the narrow segment of the taproot (tail) is left attached if possible for added flavor (Frey pers. com. 2000).

Biota - Other Vegetation

The Coeur d'Alene Tribe utilized almost forty different species of plants from their territory for food, and others for their fibers or as dyes or medicines. Within traditional society, roughly one-third of the diet was comprised of roots and berries (Nugent 1997).

Roots harvested included 16 species with camas (*Camassia quamash*), bitterroot (*Lewisia redivia*), and kouse (*Lomatium* sp.) as the most important. In the early spring (April through May), the Coeur d'Alene moved to higher elevations to the berry, camas, and kouse areas. The area of Chatcolet Lake was once an excellent source of camas before it was inundated through the construction of the Post Falls dam. "The plant [camas] was so plentiful in many places that it is no exaggeration to say that in the upper St. Maries Basin more than one-half of the total herbaceous vegetation in the lowlands was composed of this one species" (Leiberg 1897:37).

Of the approximate 22 species of berries that were utilized, all but one of which grew in Coeur d'Alene territory, most grew in the mountains to the east (Teit 1930:90). Two species of lichens *Bryoria fremontii* and *Alectoria sarmentosa,* were collected and eaten by the Coeur d'Alene. Both are extremely plentiful at all elevations.

Stocks, seeds, and berries were collected, some berries could be shaken off, and others were combed. Roots were dug with digging sticks of the typical Plateau design, a slightly curved stick about four feet in length with a transverse elk antler handle less than a foot in length. Digging stick was used for all kinds of digging including graves and cooking ovens. After contact with settlers, the digging stick was iron (Teit 1930:91; Ray 1942:145). Berries were dried and strung or made into cakes. Root crops were cooked in earth ovens, usually without adding water for steam.

Biota - Animal Protein

The most important game animals were deer and elk in Coeur d'Alene territory. Other game of minor importance were moose, mountain goat, big horn sheep, bear, and beaver. Hunting of game included

all of the usual Plateau techniques such as ambushes, blinds, screens, deer fences, driving, chasing, running with dogs, and using any number of techniques in the water (as mentioned previously) or combinations of these (Point 1967:178, 180).

Bears were also killed with dead falls. The hunting of grizzly bears is rarely mentioned in historical sources, but Kowrach and Connolly devoted several pages to a discussion of this dangerous activity (Kowrach and Connolly 1990:37-40). The dangerous method of thrusting a bi-pointed bone or stick in the open mouth of a bear was known to the Coeur d'Alene (Ray 1942:123).

Biota - Upland Birds/Water Fowl

Snares were used for upland, large birds such as grouse (Ray 1942:120). Doves were plentiful and shot or snared. Important small game included numerous local and migratory water fowl. Ducks were caught with hook and line baited with fish. Water fowl were shot from blinds either on land or canoes (Ray 1942:122). Small game also included "eagles, hawks, and woodpeckers for their feather" (Teit 1930:96). Eagles were caught in the typical fashion of hiding under a screen, often in wet areas, and reaching up and grabbing the eagle by the feet (Ray 1942:121).

Biota - Breast Milk

There are no data on breast feeding for the traditional Coeur d'Alene. The leading ethnologist for the Spokane, a Salish group very close culturally to the Coeur d'Alene, states that "at 1½ to 2½-years-of-age a child was weaned and encourage to walk" (Ross pers. com. 2000). For the Flathead, also a Salish speaking group to the east, Turney-High says that "children were not weaned until they were three years old. Spoiled children might not be weaned even at that time" (Turney-High 1937:72). The same author says that Kuteani "children were weaned at two at the very limit" (Turney-High 1941:114).

Traditional and Current Subsistence Exposure Routes

Table 3-19b shows those exposure routes that will be considered either quantitatively or qualitatively in the HHRA for the Tribal pathways.

Fully addressing the Native American exposures within the CDAB requires consideration of additional chemicals and routes of exposure not included in other scenarios in the HHRA. The resident riparian lifestyle in the Traditional Subsistence scenario and the harvest techniques employed throughout tribal history suggest that dermal exposure routes may be more significant than those applied to recreational scenarios for other populations. The tribal riparian lifestyle has the potential for significant prolonged dermal exposures to both sediment and water. One example of this type could be the harvest of the

water potato (*Sagittaria spp.*) within the mouth of the Coeur d'Alene River. These activities also involve women of reproductive age accompanied by small children for extended periods of time.

Tribal consumption rates show that traditionally as much as one-third of the overall diet was resident fish. This substantial contribution to the diet requires reexamination of mercury levels in resident fish in assessing the Traditional Subsistence scenario.

The same exposure routes will be evaluated for the Traditional Subsistence scenario and the Current Subsistence scenario. For evaluation of the human health risks associated with each scenario, the exposure factors will reflect the difference for each exposure route. Generally, the exposure frequency, i.e. a shorter period of time, for the Current Subsistence are reduced from the values used in the Traditional Subsistence scenario.

The exposure factors to be quantified in the tribal scenarios for the human health risk assessment are:

- incidental ingestion of soil/sediment,
- dermal contact with soil/sediment,
- incidental ingestion of surface water during recreational activities,
- ingestion of surface water as the main drinking water source,
- consumption of fish, and
- consumption of water potatoes.

The remainder of the tribal pathways discussed in Section 3.2.4 will not be quantified in this risk assessment, due to limitations on available data or relative significance to contribute to the overall risk to human health. Data associated with certain exposure routes may be available, but it is considered insufficient to characterize media contaminant levels. Therefore, hazards and risks displayed in Sections 5.3.4 and 6.6.4 are judged to be underestimated and not reflective of all possible exposure factors. Example calculations also showed these pathways do not significantly contribute to human health risk when compared to those pathways being quantified.

3.2.5 Complete Exposure Pathways Quantified by Area

Figures 3-3 through 3-11 present the potentially complete exposure pathways that were considered in this baseline HHRA. Potentially complete exposure pathways that were quantified are noted with a closed circle while those that were potentially complete but not quantified are noted with an open circle. Blank cells indicate that the pathway was not complete or that the receptor type does not exist in the particular area. Table 3-19a summarizes the exposure pathways that have been quantified for each exposure area.

3.3 QUANTIFICATION OF EXPOSURE TO NON-LEAD CHEMICALS

This section defines the magnitude, frequency, and duration of exposure for the populations and exposure pathways selected for quantitative evaluation. This evaluation is conducted in two stages: (1) estimation of exposure point concentrations (EPCs), and (2) quantification of pathway-specific

intakes. Intakes are calculated for both the reasonable maximum exposure (RME) and a central tendency (CT) exposure.

The RME is the maximum exposure that is reasonably expected to occur at a site. Intake parameter values have been selected so that the combination of all parameters results in an estimate of the RME for a particular exposure pathway. By design, the estimated RME is higher than that expected to be experienced by most of the exposed population.

As recommended in EPA's *Guidance for Risk Characterization* (USEPA 1995d), CT exposure estimates represent the central estimates of exposure or dose. The CT exposure estimate is intended to be more representative of likely human exposures.

3.3.1 Exposure Point Concentration

To calculate a cancer risk or a noncancer hazard, an estimate must be made of the chemical concentration to which an individual may be exposed. According to the EPA (USEPA 1991a, 1992c), the concentration term (or EPC) should be an estimate of the average concentration to which an individual would be exposed over a significant portion of a lifetime. Because of the uncertainty associated with estimating the true average concentration at a site, the EPA recommends the use of the 95 percent upper confidence limit (UCL_{95}) of the mean as the appropriate estimate of the average site concentration for an RME scenario (USEPA 1991a, 1992d, 1993c). At the UCL_{95}, the probability of underestimating the true mean is less than 5 percent. The UCL_{95} can address the uncertainties surrounding a distribution average due to limited sampling data. The locations of nonresidential soil, sediment, surface water, and groundwater sampling used in calculating EPCs are shown in Figures 3-12 through 3-26. These Figures display the sampling locations within the areas defined in Figure 3-1a. Residential sampling locations are not shown on these figures because of confidentiality agreements with homeowners.

A UCL_{95} was calculated if the number of samples evaluated was greater than or equal to 10 for a particular chemical. Sampling data from Superfund sites have shown that data sets with fewer than 10 samples per exposure area provide poor estimates of the mean concentration. For sites with limited amounts of data (i.e., less than 10 samples) the maximum value can be used as the concentration term (USEPA 1992d). Therefore, in data sets with fewer than 10 samples, the maximum concentration was selected as a health-protective estimate of exposure (Note: only eleven out of 49 data sets had fewer than 10 samples.) Table 3-21 lists the number of samples used to calculate all EPCs except the tribal EPCs. The data used to calculate the tribal EPCs is included in Appendix E. (For the screening level risk assessment for Coeur d'Alene Lake (Appendix B), UCL_{95} values were calculated for sample sizes of seven and greater. Seven samples were considered sufficient for each location based on the assumption that the sampled medium was relatively homogenous, a reasonable assumption for beach sediment deposited from upstream sources. The EPCs calculated for the HHRA were not all for relatively homogenous materials from the same source; consequently, calculations of UCL_{95} were only completed for sample populations of 10 or more.)

For the concentration term under the CT scenario, the mean of the sample data was used to represent an EPC (USEPA 1992d).

A complete listing (including soil depth interval and sampling location) of non-residential data used to calculate EPCs is presented in Appendix E. Appendix F contains residential data, which has been decoded to protect home owner privacy, used in calculating the residential EPCs. For nondetections, concentrations of half the detection limit were used in the EPC calculations. The Part D Table 3 series in Appendix A provides a summary of data including the EPCs for each exposure medium; detailed statistics are included in Appendices F and G for each medium and geographical location. Appendix F provides a detailed summary of the statistical results from the MTCA*Stat v2.1* applications (e.g., distribution tests, arithmetic means, maximum and minimum concentrations, and histograms) and includes the data used to calculate each EPC. All the data summarized in the Part D Table 3 series are from the information in Appendix F, with the exception of the UCL_{95} values for data lognormal distribution and data with neither a lognormal nor a normal distribution. Appendix G provides the SYSTAT *v.9* bootstrap exports used to calculate the RME UCL_{95} values for data with a lognormal distribution and data that were neither lognormal nor normal.

Calculation of RME Exposure Point Concentrations

The formula used to calculate a UCL_{95} depends on the distribution of the data, i.e., the "shape" of the curve (USEPA 1992d). EPA experience shows that most environmental contaminant data sets have a lognormal distribution (USEPA 1992d). However, in cases where the distribution is questionable or unknown, the EPA recommends (1) performing a statistical test to determine the best distribution assumption for the data set, and (2) graphing the data. Statistical tests or graphs (only for data sets with greater than 500 samples) were used to determine the distribution for all data sets with 10 or more samples. The results of the statistical tests and the graphs are included in Appendix F.

The Model Toxics Control Act (MTCA) statistical add-in to Microsoft Excel (MTCA*Stat v2.1*) provided by the Washington State Department of Ecology (Ecology) was used to determine distributions and calculate corresponding UCL_{95} values. The Shapiro-Wilk W-test for sample sizes less than or equal to 50, or D'Agostino's test (D-test) for sample sizes greater than 50, was performed on each data set. These tests determine if the data set best matches a normal, lognormal, or neither distribution (WDOE 1992; USEPA 1992d). The W-test and D-test are described in further detail in *Statistical Methods for Environmental Pollution Monitoring* (Gilbert 1987) and in *Statistical Guidance for Ecology Site Managers* (WDOE 1992).

If the result of a distribution test indicated a normally distributed data set, a normal UCL_{95} was calculated using MTCA*Stat v2.1* with an equation reflecting a Student's t-distribution as described in EPA guidance (USEPA 1992d). If the MTCA*Stat v2.1* results indicated a lognormal distribution of the data set, a one-sided UCL_{95} was calculated using the bootstrap method as recommended by the EPA (USEPA 1997b) based on CV, skewness, and sample size, as described in the following

paragraph. The bootstrap exports are included in Appendix F. This particular method also applied to data sets where both the normal and lognormal assumptions of the distribution were rejected.

The bootstrap method is a nonparametric statistical technique, which can construct approximate confidence intervals for the population mean. This approach makes no assumptions regarding the distribution for the underlying population. EPA's technical issue paper recommending the bootstrap procedure under certain circumstances (USEPA 1997b) focused primarily on the problems associated with calculating a UCL_{95} when the distribution of the contaminant concentration appears to be highly skewed. Positively skewed distributions are usually modeled by the lognormal distribution. However, this skewness is possibly due to biased sampling, multiple populations, or outliers and is not necessarily due to lognormally distributed data (USEPA 1997b). Statisticians showed that incorrectly assuming a lognormal distribution may lead to erroneous results (Gilbert 1993; Stewart 1994). After presenting several simulated examples in its issue paper (USEPA 1997b), the EPA concluded that the use of several other methods (e.g., jackknife, bootstrap, and the Central Limit Theorem) is more accurate than the H-statistic UCL_{95} (lognormal UCL_{95} calculation previously recommended by USEPA 1992d). Therefore, the bootstrap method was chosen. The bootstrap procedure is discussed in further detail in *The Jackknife, the Bootstrap, and Other Resampling Plans* (Efron 1982).

Using SYSTAT *v.9* software, the bootstrap procedure involves drawing repeated samples of size *n* with replacement from the given set of data. The process is repeated many times and each time an estimate of the sample mean is calculated. For this baseline HHRA, the process was repeated 250 to 500 times depending on the sample size *n*. Even with 2,000 repetitions of the process, the estimate of the UCL_{95} was reasonably similar to the UCL_{95} calculated with fewer repetitions. (Test runs were conducted using 2,000, 1,000, 500, and 250 repetitions and the resulting UCL_{95} estimates were not substantially different from each other.) Subsequently, the bootstrapped estimates of the mean are ranked, the ranks are converted to percentiles, and the first estimate of the mean closest to the 95th percentile is used as the UCL_{95} (the RME Medium EPC Value–see Appendix A). Results of the bootstrap estimates are provided in Appendix G. Uncertainties associated with this procedure are discussed in Section 7.

Calculation of CT Exposure Point Concentrations

To calculate a central tendency exposure point concentration, USEPA 1992d recommends the use of the arithmetic mean no matter what the underlying distribution of the data set. However, more recent EPA studies state that for lognormally distributed data sets, an adjusted geometric mean is a more appropriate estimator of CT especially when the coefficient of variation is greater than one (USEPA 1997b). Most of the lognormally distributed data sets used to calculate EPCs in this HHRA had coefficients of variation larger than one (see Appendix F). Therefore, the geometric mean (from MTCA*Stat* export in Appendix F) multiplied by a "bias factor" is used to calculate the CT when the data is lognormally distributed (as recommended by Gilbert 1987 and Singh et al. 1997) and the arithmetic mean (the mean from MTCA*Stat* export in Appendix F) is used to calculate CT estimate of the EPC when the underlying distribution variance unbiased estimator (MVUE) of the mean, is

calculated by the formula: lognormal mean = exp [geometric mean + (standard deviation^2/2)] (Gilbert 1987). The Table 3 series in Appendix A lists the distribution of the data and the CT values used in the HHRA as well as the arithmetic means of the data sets.

Exposure Point Concentrations by Medium

Table 3-20 summarizes the RME EPCs by medium (presented in detail in the Part D Table 3 series in Appendix A). The following subsections provide details on the data sets selected for each EPC, any data excluded from the calculations, and any unusual features of the particular data set. Table 3-21 summarizes the numbers of samples used for each EPC calculation in each human health exposure area.

Yard Soil EPCs. For the soil EPC, all soil samples collected in the 0- to 1-inch-depth interval of the homes in a particular geographical area were used. A separate residential soil EPC was calculated for each geographical area and Table 3-21 presents the numbers of homes and soil samples collected at the 0- to 1-inch depth for each of the eight residential areas. For example, the Kingston residential soil EPC is based on 71 samples collected from 22 homes.

Residential exposure to chemicals in soil may occur outside in the yard and inside the house since soil from the yard may be carried into the house. The default assumption in EPA's lead model is that, on average, 45 percent of a child's total intake of soil and dust is derived from outdoor soil and 55 percent is derived from indoor house dust (USEPA 1994a). Although some house dust concentrations of metals other than lead are available, the house dust data are insufficient to include directly in the EPC calculations for the non-lead metals. Of the 191 homes with soil data, only 83 homes had floor mat data (44 percent) and 74 homes had vacuum bag data (39 percent) for the nonlead metals. Therefore, only the data from yard soil were used to develop the residential soil EPCs, and yard soil concentrations served as surrogates for house dust concentrations. (Tables 3.1.1, 3.2.1, 3.3.1, 3.4.1, 3.5.1, 3.6.1, 3.8.1, and 3.9.1 in Appendix A provide the yard soil EPCs for each geographical area.) Using soil concentrations as surrogates for house dust concentrations has the potential to either underestimate or overestimate human health risks. The available house dust data is discussed in the following subsection and is further evaluated in Section 7.

House Dust Data. Two different types of house dust samples were collected : (1) floor mats placed just inside outer doors in the home, and (2) samples from vacuum cleaner bags. However, for the non-lead metals not every home that was sampled for soil was sampled for dust. Dust samples were collected only from homes that were volunteered for sampling. Therefore, there is a lack of sufficient dust data for each of the geographical subregions. Although soil is assumed to be a major contributor to indoor concentrations of chemicals in dust, and it could be thought a good predictor of indoor dust concentrations for those homes where dust data is lacking, graphs of dust versus yard soil concentrations for the non-lead metals did not indicate a strong correlation (see Appendix I). The reasons for the lack of clear trends are uncertain. Yard soil concentrations may be good predictors of some, but not all chemicals in dust. For example, additional sources of chemicals in dust (e.g., lead from paint as an additional lead source in dust) as well as differences in grain size between soil and dust

may exist, resulting in enrichment or dilution of various chemicals in dust relative to soil. Therefore, soil samples were selected as a surrogate measure of dust concentrations for the non-lead metals for the following reasons:

- There is a lack of sufficient dust data for the non-lead metals for each of the geographical subregions (there is a great deal more dust data available for lead, see Sections 2.2.1 and 2.5.5).
- Because of the lack of paired yard soil and house dust data, the relationship between soil and dust for the non-lead metals is undefined, making a quantitative prediction of dust concentrations where there is no dust data highly uncertain. Note- paired yard soil and dust data for lead are available for all homes; thus, for the lead risk assessment, no predictions of concentrations are necessary.

Section 7.3.1 discusses in greater detail the uncertainty surrounding the relationship between house dust and yard soil chemical concentrations, as well as the uncertainty regarding the use of yard soil as a surrogate for house dust concentrations.

Tap Water EPCs. The average of first-run and flushed-line tap water samples was calculated to arrive at one chemical concentration per home. The assumption that 50 percent of the water ingested is from the first-run sample is the default assumption in EPA's lead model (USEPA 1994a). The only non-lead COPC in tap water is arsenic; the arsenic concentrations in first-run and flushed-line samples were very similar. Fewer than 10 tap water samples were collected in five of the eight residential exposure areas (Table 3-21). In these areas, the maximum concentration (after averaging first-run and flushed-line) was selected as the EPC. (Tables 3.1.2, 3.2.2, 3.3.2, 3.4.2, 3.5.2, 3.6.2, 3.8.2, and 3.9.2 in Appendix A provide the tap water EPCs for each geographical area.)

Total metal concentrations, rather than dissolved metal concentrations, were used for all water exposures, i.e., tap water, groundwater, and surface water.

Groundwater EPCs. All groundwater samples from wells drilled during source investigations in the Nine Mile/Canyon Creek area were used to calculate future drinking water EPCs. (Table 3.8.3 in Appendix A provides the groundwater EPCs for Nine Mile/Canyon Creek.)

Homegrown Vegetable EPCs. Homegrown produce data were collected from a wide variety of vegetables. Because the ingestion rates selected for vegetable consumption are based on total vegetable intake rather than categories of vegetables, all the vegetable data were pooled before calculating EPCs (see Table 3.11.1 in Appendix A).

Concentrations of metals in vegetables were reported as dry weight concentrations and the percent moisture content of the sample was reported separately. The wet weight concentration was used to calculate EPCs for the vegetable ingestion pathway because people generally do not remove the moisture from their produce before eating. The wet weight conversion from dry weight is as follows:

wet weight = (dry weight value)(100 ! % moisture/100)

Upland Soil EPCs. Sampled waste piles are present in the Nine Mile/Canyon Creek area and in the Mullan area. Waste piles are present in many other areas of the upper Coeur d'Alene Basin; however, in many cases the piles are not adjacent to residential homes (as is the case in the Nine Mile and Mullan areas) and sieved surface soil samples are not available because there were not enough fines in the top one inch of soil due to the rocky nature of waste piles. The available waste pile data are considered representative of this type of exposure throughout the Basin. (Tables 3.8.4 and 3.9.4 in Appendix A provide the waste pile EPCs for Nine Mile and Mullan.)

The public recreational soil areas are located in developed parks and ball fields in the upper Basin. The developed parks are located in the towns of Wallace and Silverton. Although samples up to a depth of 24 inches were collected in the upland parks, only the 0- to 1-inch-depth interval was used in the public recreational EPC calculations because surface soil has the greatest impact on exposure at these locations. (Tables 3.5.3 and 3.6.3 in Appendix A provide the upland parks EPCs for Silverton and Wallace.)

As described for public surface water EPCs, public areas of exposure to floodplain soil/sediment are all located along the lower Coeur d'Alene River. Exposed populations include neighborhood recreational groups as well as the general public.

Floodplain Soil/Sediment EPCs. Floodplain soil/sediments include the loose alluvial material along the beaches of the creeks and rivers to a depth of 12 inches, the depth to which a child could be expected to dig, and the soils in the upland areas around the creeks and rivers, where children might be exposed to surface soils (a depth of 1 inch) during camping and picnicking. All of these materials were combined to calculate EPCs for the Coeur d'Alene River areas. (Tables 3.1.3, 3.1.5, 3.2.3, 3.2.5, 3.3.3, 3.3.4, 3.4.3, 3.7.1, 3.8.5, 3.9.4, and 3.10.1 in Appendix A provide the floodplain soil/sediment EPCs.).

Even though the chemical concentrations in the upland area soils and the beach area sediments may be statistically different for some chemicals at some sites, any differences that may exist are not relevant for all exposure areas, except for perhaps Blackwell Island, because of the following exposure assumptions used in calculating risk:

- The upland areas and the beach areas are in close proximity to one another.
- The upland areas have all been impacted by previous flood events and therefore experienced a mixing of soil and sediment materials. Therefore, it can be assumed that beach sediments and upland soils are relatively homogenous.
- A receptor is presumed to spend an equal amount of time in upland areas as in beach areas. In addition, it is assumed that receptors will have an equal probability of visiting one CUA in a geographical area as another.

In the case of Blackwell Island, the upland areas and beach areas are more spatially separated than the other exposure areas. Therefore, a receptor may not spend an equal amount of time in upland and beach areas. In addition. Blackwell Island may not have experienced the mixing of upland soil and beach sediment as the other CUAs, because it may not have been impacted by flood events. If soil/sediment concentrations from Blackwell Island exceed target risk and hazard goals (See Section 5), Blackwell Island will be further reviewed before remedial decisions are made.

As described for public surface water EPCs, public areas of exposure to floodplain soil/sediment are all located along the lower Coeur d'Alene River. Exposed populations include neighborhood recreational groups as well as the general public.

Surface Water EPCs. Neighborhood recreational surface water exposures generally occur in the creeks, in the side canyons, and in the upper South Fork where there are no developed public park areas but residences are located adjacent to the water. (The South Fork has very few depositional areas within the river channel. Material in the channel consists mostly of gravel, pebbles, and cobbles. Thus, exposure to sediments containing mining-related metals is limited.) Surface water samples from various sampling events were segregated by geographical area. Table 3-3 presents the numbers of samples per area available for the EPC calculations. (Tables 3.1.4, 3.2.4, 3.2.6, 3.3.5, 3.7.2, 3.8.6, 3.9.5, and 3.10.2 in Appendix A provide the surface water EPCs.)

The water samples for neighborhood recreational exposures were collected without stirring up the sediment prior to sampling (as was done for public recreational water samples collected in the lower Coeur d'Alene River (Section 2.2.8). Therefore, water samples collected in the neighborhood areas did not contain a high amount of suspended solids. Surface water samples from the lower Coeur d'Alene River, which were collected after purposeful disturbance of the water, contained suspended sediments.

Fish Tissue EPCs. Fish fillet data (wet weight concentrations) are available from three species of fish (perch, bullhead, and pike) from three of the lateral lakes along the lower Coeur d'Alene River. Separate EPCs were calculated for each species after pooling the data from the three lateral lakes (Medicine, Killarney, and Thompson) (see Table 3.11.2 in Appendix A). All fish fillet data are from one geographical area, the Lower Basin. The upper Basin has a very limited fish population and little fishing occurs.

As was previously discussed in Section 2.2.1, whole fish tissue data is not available for use in this human health risk assessment for the tribal scenarios. Whole body metal concentrations are usually higher than fillet concentrations; thus, use of fillet data for populations which consume whole fish (tribal subsistence scenarios) likely underestimates the chemical dose from fish. Whole body and fillet tissue samples were collected and analyzed for lead from the Spokane River for three species of fish (large-scale sucker, rainbow trout, and mountain whitefish) (Johnson 2000). EPCs were calculated for the Spokane River fillet data. If the same concentration increases apply to the species sampled in the

lateral lakes, risk from fish ingestion would likely significantly increase if whole bodies are eaten rather than fillets.

Construction Site Soil EPCs. Soil EPCs should estimate average exposure (with an adequate margin of safety) for a specific activity; therefore, all soil data from all depth intervals in each applicable geographical area were combined for the occupational EPC calculations for the construction worker scenario, with the exception of data from the waste piles. For occupational soil exposures in the Lower Basin, sediment data were also included because of the large floodplain area and the lack of easy differentiation between sediment and soil in the floodplain. The data were combined because of the nature of a typical construction worker's activity. For instance, construction workers could be moving "dirt" from one location to another and in the process be exposed to a variety of depth intervals. Thus, combining the data results in occupational EPCs that best represent a construction worker's typical soil exposure. (See Tables 3.1.7, 3.2.7, 3.7.3, 3.8.7, and 3.9.6 in Appendix A for construction site soil EPCs for each applicable geographical area.)

Water Potato EPCs. Separate water potato EPCs were calculated from peeled potatoes and from unpeeled potatoes. The Coeur d'Alene Tribe collected and analyzed water potatoes. Water potatoes were evaluated for non-lead metals (Table 3.1.6 in Appendix A) and lead (see Section 3.4).

3.3.2 Estimation of Chemical Intakes

As part of the exposure assessment, chemical intakes were determined for each pathway and population included in the quantitative risk assessment. In general, the intake of a chemical is estimated from exposure models that combine various exposure factors related to behavior and physiology, such as exposure frequency and duration, contact rate, chemical concentration, body weight, and averaging time. Chemical intake equations for each exposure pathway and medium are presented below. The equations and the selected intake parameters are presented in the Part D, Table 4 series, in Appendix A and are summarized in Tables 3-22 through 3-26. Exposure scenarios by geographical area are summarized in Table 3-19a.

Soil and Sediment

Two exposure pathways were evaluated for soil and sediment: incidental ingestion and dermal contact. Intake of chemicals through the incidental ingestion of soil or sediment was calculated as follows (see also Appendix A, Tables 4.1, 4.4, 4.5, 4.6, 4.8, 4.9, and 4.12):

$$\text{Chemical Intake} = C_s \times SIF_{s\text{-}ing} \quad (1)$$

$$SIF_{s\text{-}ing} = (IR \times EF \times ED \times CF \times ABS) / (BW \times AT) \quad (2)$$

where

C_s	=	Chemical concentration in soil or sediment (mg/kg)
$SIF_{s\text{-}ing}$	=	Summary intake factor for soil/sediment ingestion (kg/kg-day)
IR	=	Ingestion rate (mg/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (kg/mg)
ABS	=	Gastrointestinal absorption
BW	=	Body weight (kg)
AT	=	Averaging time (days)

It was assumed that 100 percent of the soil and sediment ingested is from potentially contaminated areas of the site.

For residential and public recreational soil/sediment ingestion, risks were evaluated for both a child (age 0 to 6) and an integrated child/adult scenario (child age 0 to 6, adult age 7 to 30). The integrated child/adult scenario for soil/sediment ingestion was calculated as follows:

$$SIF_{s\text{-}ing} = [(IR_{ch} \times ED_{ch})/BW_{ch} + (IR_a \times ED_a) / BW_a] \times EF \times CF \times ABS / AT \qquad (3)$$

where

$SIF_{s\text{-}ing}$	=	Summary intake factor for soil/sediment ingestion (kg/kg-day)
IR_{ch}	=	Child soil/sediment ingestion rate (g/day)
ED_{ch}	=	Child exposure duration (years)
BW_{ch}	=	Child body weight (kg)
IR_a	=	Adult soil/sediment ingestion rate (g/day)
ED_a	=	Adult exposure duration (years)
BW_a	=	Adult body weight (kg)
EF	=	Exposure frequency (days/year)
CF	=	Conversion factor (kg/mg)
ABS	=	Gastrointestinal absorption
AT	=	Averaging time (days)

Intake of chemicals through dermal contact with soil or sediment was calculated as follows:

$$\text{Absorbed Dose (mg/kg-day)} = C_s \times SIF_{s\text{-}derm} \qquad (4)$$

$$SIF_{s\text{-}derm} = SA \times AF \times ABS \times EF \times ED \times CF / (BW \times AT) \qquad (5)$$

where

C_s	=	Chemical concentration in soil/sediment (mg/kg)
$SIF_{s\text{-}derm}$	=	Summary intake factor for dermal contact with soil/sediment (kg/kg-day)
SA	=	Skin surface area available for contact (cm^2)
AF	=	Soil-to-skin adherence factor (mg/cm^2-event)
ABS	=	Chemical-specific dermal absorption factor (unitless)
EF	=	Exposure frequency (events/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (kg/mg)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

For residential and public recreational dermal contact scenarios, risks were evaluated for both a child (age 0 to 6) and an integrated child/adult scenario. The integrated child/adult scenario for dermal contact with soil/sediment was calculated as follows:

$$SIF_{s\text{-}derm} = [(SA_{ch} \times AF_{ch} \times ED_{ch}) / BW_{ch} + (SA_a \times AF_a \times ED_a) / BW_a] \times ABS \times EF \times CF / AT \quad (6)$$

where

$SIF_{s\text{-}derm}$	=	Summary intake factor for dermal contact with soil/sediment (kg/kg-day)
SA_{ch}	=	Child skin surface area available for contact (cm^2)
AF_{ch}	=	Child soil-to-skin adherence factor (mg/cm^2-event)
ED_{ch}	=	Child exposure duration (years)
BW_{ch}	=	Child body weight (kg)
SA_a	=	Adult skin surface area available for contact (cm^2)
AF_a	=	Adult soil-to-skin adherence factor (mg/cm^2-event)
ED_a	=	Adult exposure duration (years)
BW_a	=	Adult body weight (kg)
ABS	=	Chemical-specific dermal absorption factor (unitless)
EF	=	Exposure frequency (events/year)
CF	=	Conversion factor (kg/mg)
AT	=	Averaging time (days)

Groundwater/Tap Water

Intake of chemicals through the ingestion of groundwater as a drinking water source was calculated as follows (see also Appendix A, Table 4.2):

$$\text{Chemical Intake (mg/kg-day)} = C_w \times SIF_w \quad (7)$$

$$SIF_w = IR_w \times EF \times ED \times CF / (BW \times AT) \quad (8)$$

where

C_w	=	Chemical concentration in groundwater/tap water (µg/L)
SIF_w	=	Summary intake factor for ingestion of tap water (L/kg-day)
IR_w	=	Ingestion rate of tap water (L/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (mg/µg)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

Risks from tap water ingestion were evaluated for both a child (age 0 to 6) and an integrated child/adult scenario. The integrated child/adult scenario for tap water ingestion was calculated as follows:

$$SIF_w = [(IR_{ch} \times ED_{ch}) / BW_{ch} + (IR_a \times ED_a) / BW_a] \times EF \times CF / AT \quad (9)$$

where

SIF_w	=	Summary intake factor for ingestion of tap water (L/kg-day)
IR_{ch}	=	Child tap water ingestion rate (L/day)
ED_{ch}	=	Child exposure duration (years)
BW_{ch}	=	Child body weight (kg)
IR_a	=	Adult tap water ingestion rate (L/day)
ED_a	=	Adult exposure duration (years)
BW_a	=	Adult body weight (kg)
EF	=	Exposure frequency (days/year)
CF	=	Conversion factor (mg/µg)
AT	=	Averaging time (days)

Surface Water

Intake of chemicals through the incidental ingestion of surface water during swimming or wading was evaluated using the following equation (see also Appendix A, Tables 4.7 and 4.10):

$$\text{Chemical Intake (mg/kg-day)} = C_w \times SIF_{sw} \quad (10)$$

$$SIF_{sw} = IR_{sw} \times ET \times EF \times ED \times CF1 \times CF2 / (BW \times AT) \quad (11)$$

where

C_w	=	Chemical concentration in surface water (µg/L)
SIF_{sw}	=	Summary intake factor for incidental ingestion of surface water (mg-L/µg-kg-day)
IR_{sw}	=	Ingestion rate of surface water during swimming or wading (mL/hour)
ET	=	Exposure time (hours/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF1	=	Conversion factor (mg/µg)
CF2	=	Conversion factor (L/mL)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

Risks from incidental surface water ingestion were evaluated for both a child (age 0 to 6) and an integrated child/adult scenario. The integrated child/adult scenario for surface water ingestion was calculated as follows:

$$SIF_{sw} = [(IR_{ch} \times ED_{ch}) / BW_{ch} + (IR_a \times ED_a) / BW_a] \times ET \times EF \times CF1 \times CF2 / AT \qquad (12)$$

where

SIF_{sw}	=	Summary intake factor for incidental ingestion of surface water (L/kg-day)
IR_{ch}	=	Child surface water ingestion rate (mL/hour)
ED_{ch}	=	Child exposure duration (years)
BW_{ch}	=	Child body weight (kg)
IR_a	=	Adult surface water ingestion rate (mL/hour)
ED_a	=	Adult exposure duration (years)
BW_a	=	Adult body weight (kg)
ET	=	Exposure time (hours/day)
EF	=	Exposure frequency (days/year)
CF1	=	Conversion factor (mg/µg)
CF2	=	Conversion factor (L/mL)
AT	=	Averaging time (days)

Homegrown Vegetables

Intake of chemicals through the ingestion of homegrown vegetables was calculated as follows (see also Appendix A, Table 4.3):

$$\text{Chemical Intake (mg/kg-day)} = C_{veg} \times SIF_{veg} \qquad (13)$$

$$SIF_{veg} = IR_{veg} \times EF \times ED \times CF / AT \qquad (14)$$

where

C_{veg}	=	Chemical concentration in homegrown vegetables (mg/kg)
SIF_{veg}	=	Summary intake factor for ingestion of homegrown vegetables (kg/kg-day)
IR_{veg}	=	Intake rate of homegrown vegetables (g/kg-day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (kg/g)
AT	=	Averaging time (days)

Because the intake rate for homegrown vegetables is already normalized to body weight (i.e., the intake rate is presented as g/day per kg body weight), a separate intake calculation was not performed for children.

Fish

Intake of chemicals through the ingestion of recreationally caught fish was calculated for adults only (see Appendix A, Tables 4.11 and 4.13). The following equation was used:

$$\text{Chemical Intake (mg/kg-day)} = C_f \times SIF_f \quad (15)$$

$$SIF_f = IR_f \times EF \times ED \times CF / (BW \times AT) \quad (16)$$

where

C_f	=	Chemical concentration in fish tissue (mg/kg)
SIF_f	=	Summary intake factor for ingestion of fish (kg/kg-day)
IR_f	=	Intake rate of fish (g/kg-day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CF	=	Conversion factor (kg/g)
BW	=	Body weight (kg)
AT	=	Averaging time (days)

3.3.3 Exposure Factors

Exposure factors were identified for the RME and CT cases as described in the following subsections. The exposure factors were selected after a review of historical risk assessments associated with the Bunker Hill Superfund Site, EPA guidance documents, site-specific health studies, and professional judgement. The exposure factors used in this baseline HHRA are summarized in Tables 3-22 through 3-26.

General Exposure Factors

The following exposure factors are common to all the scenarios evaluated in this baseline HHRA.

Body Weight. An adult body weight of 70 kg was assumed. This is the average body weight for adult men and women combined, rounded to 70 kg (USEPA 1991a, 1991b). For children ages 0 to 6, an average body weight of 15 kg was assumed (USEPA 1991a, 1991b). For children ages 4 to 11, a value of 28 kg was used; this is the 50th percentile body weight for boys and girls combined (USEPA 1997a). Average body weights were used for both the RME and CT cases, because when combined with the other variables in the intake equation, it is believed to result in the most reasonable estimate of intake (USEPA 1989). For example, it would not be reasonable to assume that the smallest person would have the highest intake.

Exposure Duration. For RME residential and recreational exposures, an exposure duration of 30 years was assumed. This represents the 90th percentile for time spent at one residence (USEPA 1991a). Of the 30 years total exposure duration, it was assumed that a 6-year period (ages 0 to 6) accounts for the period of highest soil ingestion and lowest body weight. A 24-year duration was assessed for older children and adults (USEPA 1991a). For children ages 4 to 11, an exposure duration of 7 years (to cover the entire age range) was used. A construction worker was conservatively assumed to work for 25 years in the same area; this represents the 95th percentile for length of time that employees work in the same location, according to the Bureau of Labor Statistics (as cited in USEPA 1991a).

For the CT residential and recreational cases, an exposure duration of 9 years was assumed; this represents the 50th percentile for residence time at one location (USEPA 1991b). Of the 9 years total duration, 2 years was assumed for ages 0 to 6; an exposure duration of 7 years was assumed for older children and adults (USEPA 1993c). For children ages 4 to 11, a CT exposure duration of 2 years was assumed. For the construction worker, a CT exposure duration of 6.6 years was selected. This represents the median length of time a worker spends at one job (USEPA 1997a).

Averaging Time. For carcinogens, an averaging time of 70 years (equivalent to a lifetime) was used (USEPA 1989). For noncarcinogens, an averaging time equal to the exposure duration was used (USEPA 1989).

Dermal Absorption Factor. The dermal absorption factor represents the fraction of a chemical that is absorbed through the skin via contact with soil or sediment. Dermal absorption factors of 0.03 and 0.001 were assumed for arsenic and cadmium, respectively (USEPA 1998e).

Gastrointestinal Absorption Factor. The dose calculated by the exposure assessment is considered an “administered” or “applied” dose unless it is corrected for the extent of systemic absorption into the blood stream (“absorbed” dose). In general, when assessing exposure by a given route, the amount of absorption of chemicals should be adjusted if absorption for the population at risk differs from the

population (human or laboratory animals) used to develop the relevant toxicity criteria (see Section 4). This discrepancy may result from differences in the administered form of the toxicant or from differences in physiological processes. A correction for gastrointestinal absorption via soil ingestion was considered appropriate only for arsenic, as discussed in the following paragraph.

Gastrointestinal absorption of ingested arsenic varies greatly with the water solubility of the arsenic compound and the physical form administered (USEPA 1984). For example, absorption of arsenic trioxide is reported to be 30 to 40 percent for the compound in suspension, but as high as 95 percent or greater for the compound in solution (Ariyoshi and Ikeda 1974; USEPA 1984). Because the toxicity criterion is based on inorganic arsenic dissolved in drinking water, an absorption correction should be considered for the differences between arsenic absorption from soil versus from drinking water. There is uncertainty associated with the bioavailability of arsenic in soil, differences in soil types, and the lack of human absorption data.

The correction factor used in this risk assessment is the EPA Region 10 default relative bioavailability factor of 60 percent (USEPA 2000b). This value was derived from the EPA Region 10 animal study in which immature swine were dosed with residential soil (80 percent) and slag dust (42 percent) from the smelter-impacted Ruston/North Tacoma Superfund site in Washington (USEPA 1996d). A site-specific gastrointestinal absorption factor for arsenic was derived from this study and the lower 95 percent confidence limit of that value has become the Region 10 default bioavailability factor; 60 percent (USEPA 2000b). The lower 95 percent confidence limit was used because it was assumed that the relative arsenic bioavailability would be lower for mining impacted soils than for smelter impacted soils due to the differences in arsenic particle size and arsenic species (USEPA 2000b). For a more detailed discussion of the bioavailability rate of arsenic, see Section 7.

Residential Exposure Factors

The residential exposure factors apply to exposures to yard soil, house dust, tap water, and homegrown vegetables by children (ages 0 to 6) and adults living in the human health exposure areas.

Soil and House Dust Ingestion Rate. For the RME case, a residential soil ingestion rate of 100 mg/day was selected for adults and a soil ingestion rate of 200 mg/day was selected for children (USEPA 1991a). These ingestion rates account for ingestion of both outdoor soil and indoor dust and are believed to represent upperbound values for soil and dust ingestion; they are EPA's RME default values for residential soil ingestion (USEPA 1991a). For the CT case, a soil ingestion rate of 50 mg/day was assumed for adults who do not engage in activities with a lot of soil or dust contact on a regular basis (USEPA 1993c). For children, a soil ingestion rate of 100 mg/day was selected; this value was deemed to be reasonable based on results using tracer elements and is nearly identical to the ingestion rate for this age group based on age-specific values utilized in support of the NAAQC for lead and the lead biokinetic uptake model (USEPA 1993c).

Tap Water Ingestion Rate. An adult RME drinking water ingestion rate of 2 L/day was selected. This value is currently used by the EPA Office of Water in setting drinking water standards and is EPA's default RME adult drinking water ingestion rate (USEPA 1991a). For the CT case, an adult water ingestion rate of 1.4 L/day is based on the average intake observed from five studies as summarized in the *Exposure Factors Handbook* (USEPA 1997a) and is EPA's recommended value (USEPA 1991b, 1993c). For children, a drinking water ingestion rate of 1 L/day was used for both the RME and CT cases (USEPA 1999c).

Homegrown Vegetable Ingestion Rate. A wet-weight vegetable ingestion rate of 5.04 g per kg body weight per day (g/kg-day) for the RME case and 0.492 g/kg-day for the CT case were selected, based on the U.S. Department of Agriculture (USDA) Nationwide Food Consumption Survey (NFCS). The most recent data (1987-88) were used to generate intake rates for home-produced foods because they are believed to be reflective of current consumption patterns among the U.S. population. The NFCS collected information over a 7-day period on the socioeconomic and demographic characteristics of households, and the types, amounts, value, and sources of foods consumed by the household (USEPA 1997a). The sample size for the 1987-88 survey was approximately 4,300 households, consisting of over 10,000 individuals. The NFCS tabulated the percentage of total intake of each home-produced food item group consumed during the survey period. The percentage of homegrown vegetable intake was presented as the ratio of total intake of the homegrown vegetable to the total intake of all forms of food.

Percentiles of average daily intake derived from short-term intervals (e.g., 7 days) are generally not reflective of long-term patterns. For example, homegrown vegetables have a strong seasonal component associated with their use. The consumption rates are generally influenced by the following factors (USEPA 1997a):

- Size of garden,
- Yield,
- Quality of produce,
- Types of vegetables grown,
- Length of growing season, and
- Climate.

Therefore, an approach was developed that attempted to account for seasonal variability (e.g., climate and length of growing season) in consumption. Seasonally adjusted percentile distributions for a given region were calculated by averaging the corresponding percentiles of each of the four seasonal distributions of the region. When using regional seasonally adjusted distributions to approximate regional long-term distributions, it was assumed that each individual consumes at the same regional percentile level for each season and consumes at a constant weekly rate throughout a given season (USEPA 1997a).

For consumers in the West Region, the recommended seasonally adjusted 50th and 95th percentile vegetable intake rates are 0.492 g/kg-day and 5.04 g/kg-day, respectively (USEPA 1997a, Table 13-33). The use of these values in calculating chemical intake does not require the body weight factor to be included in the denominator of the daily intake equation. The reason is that the total survey population included children as well as adults and in addition to adult body weight and adult intake rates, child body weight and child intake rates were factored in to the overall ingestion rate. The average body weight for all participating age groups was approximately 60 kg.

In addition, it was assumed that the seasonally adjusted intake rates also include consumption of canned and frozen homegrown vegetables throughout the year. The intake rates were not adjusted for preparation losses (e.g., removal of skin, peel, core, caps, stems, and defects, or draining of liquids from canned or frozen forms (USEPA 1997a). Therefore, the homegrown vegetable ingestion rates are assumed to be health protective.

Exposure Frequency. The EPA default residential exposure frequency of 350 days/year was used for the RME residential case (USEPA 1991a). For CT exposures to yard soil, a residential exposure frequency of 260 days/year was used. This is based on the 1997 Exposure Frequency Handbook, Table 15A-3, "Time Spent in Various Microenvironments." The table lists the mean time spent at home for men and women as 62% and 71%, respectively. The more conservative 71% (260 days per year) time spent at home was selected as the CT exposure frequency for yard soil. For CT exposures to tap water, an exposure frequency of 234 days/year was assumed based on being away from home one-third of the time; this is the CT default value listed in USEPA 1993c. Because the vegetable ingestion rate is an average daily consumption rate, the appropriate exposure frequency for both the RME and the CT case is 365 days/year.

Skin Surface Area. For adults exposed to yard soil, a skin surface area of 2500 cm^2 was selected; this represents an adult wearing a short-sleeved shirt, long pants, and shoes, with exposed skin surface limited to the face, hands, and forearms (USEPA 1998e). For child exposure to yard soil, a skin surface area of 2200 cm^2 was selected; this represents the exposed area on a child wearing short-sleeved shirt and shorts, but no shoes; exposure is to head, hands, forearms, lower legs, and feet (USEPA 1998e).

Adherence Factor. Soil adherence factors of 0.1 and 0.2 were assumed for adults and children, respectively. These values are based on data reported by Kissel et al. (1996a, 1996b, 1998) and Holmes et al. (1999).

Neighborhood Recreational Exposure Factors

The neighborhood recreational exposure factors apply to local residents adjacent to mining-impacted rivers, creeks, waste piles, and parks/schools. The population of greatest concern for these exposures is assumed to be children ages 4 to 11; therefore, this is the receptor group that was evaluated for neighborhood recreational exposures.

Soil and Soil/Sediment Ingestion Rates. An RME soil/sediment ingestion rate of 300 mg/day was selected for children ages 4 to 11. This value represents the 90th percentile intake based on a soil and feces tracer study (van Wijnen et al. 1990, as cited in USEPA 1997a) that measured ingestion rates for 78 children at campgrounds. The "campground" intake rate is now considered by EPA Region 10 to be the appropriate ingestion rate for intermittent recreational exposures (USEPA 1999d). The mean value of soil intake from the same study, 120 mg/day, was selected as the soil and sediment ingestion rate for the CT case.

Surface Water Ingestion Rate. An incidental surface water ingestion rate of 30 mL/hour was selected; this value was used by the EPA to derive AWQC for human health (USEPA 1998d).

Exposure Time for Incidental Ingestion of Surface Water. An exposure time of 1 hour per event for incidental ingestion of water while swimming was assumed (USEPA 1997a, Table 15-176). This represents the 50th percentile value for swimming duration in a freshwater swimming pool. It is also EPA's recommended value for assessing swimming activities (USEPA 1997a).

Exposure Frequency. For neighborhood recreational exposure to waste pile soil, it was assumed that children ages 4 to 11 would visit a waste pile once per week for 34 weeks (April through November) each year. The assumption of 34 weeks is based on professional judgement in consideration of weather conditions in the Basin and the historical divisions of the year from previous HHRAs conducted in the Bunker Hill Superfund Site. Historically, 17 weeks of summer and 17 of spring and fall combined have been assumed for recreational exposures (Jacobs Engineering et al. 1989). In addition, it was assumed that the weekly exposure would occur over a 7-hour period; this is EPA's recommended value for children's weekend exposure outdoor time (USEPA 1997a).

For neighborhood recreational exposures to upland parks and schools, as well as upland soils in the Elk Creek area, it was assumed that children ages 4 to 11 would visit these locations two times per week for 34 weeks (April through November) each year.

For neighborhood recreational exposures to floodplain soil/sediment and surface water, an exposure frequency of four times per week for 24 weeks (May through mid-October) was assumed. In addition, it was assumed that exposures would occur for 3 hours per day for soil/sediment and 1 hour per day for surface water; these are the upper and lower percentiles of the average time, respectively, spent at most outdoor activities (USEPA 1997a). These values are similar to the 2 hours/day and 3 hours/day "other" outdoor exposures used to represent summer exposures for age groups 2 through 6 years old and 7 through 12 years old, respectively (Jacobs Engineering et al. 1989). Hours per day were normalized to days per year assuming 14 waking hours per day (see Table 3.23).

Skin Surface Area. Because the creeks are generally too small for swimming, the body surface area available for contact with sediment at areas with no public access was assumed to be 5080 cm^2 (assuming shorts, bare feet, and short-sleeved shirt for children 4 to 11 years old) (USEPA 1997a). For neighborhood exposures in the lower Coeur d'Alene River and the lateral lakes (i.e., Lower Basin

and Kingston [NS confluence]) where swimming is possible, a skin surface area of 7960 cm^2 was assumed. This value is based on the assumption that of the 24 weeks per year of exposure, swimming would occur during the warmest 16 weeks of the year (with a corresponding skin surface area of 9400 cm^2, the median skin surface area for male children ages 4 to 11), whereas wading and playing along the shoreline (without swimming) would occur during 8 weeks of the year (with a corresponding skin surface area of 5080 cm^2) (USEPA 1997a).

For exposure to soil at upland parks and schools, as well as in the vicinity of Elk Creek, a skin surface are of 5080 cm^2 was selected. This represents shorts, bare feet, and short-sleeved shirts for children 4 to 11 years old (USEPA 1997a).

Adherence Factor. A soil adherence factor of 0.2 was assumed for neighborhood children engaged in recreational activities. This value is based on data reported by Kissel et al. (1996a, 1996b, 1998) and Holmes et al. (1999).

Public Recreational Exposure Factors

The public recreational exposure factors apply to children ages 0 to 6 and adults involved in recreational activities at public parks/schools and beaches.

Soil and Soil/Sediment Ingestion Rates. A soil/sediment ingestion rate of 300 mg/day was selected for children based on the study previously discussed for neighborhood soil/sediment ingestion rates (van Wijnen et al. 1990, as cited in USEPA 1997a). The adult residential default of 100 mg/day ingestion rate for soil/sediment was selected for public recreational exposures (USEPA 1991a).

Surface Water Ingestion Rate. An incidental surface water ingestion rate of 30 mL/hour was selected; this value was used by the EPA to derive AWQC for human health (USEPA 1998d).

Exposure Time for Incidental Ingestion of Surface Water. An exposure time of 1 hour per event for incidental ingestion of water while swimming was assumed (USEPA 1997a, Table 15-176). This represents the 50th percentile value for swimming duration in a freshwater swimming pool.

Ingestion Rate of Fish. A fish ingestion rate of 46 g/day was selected based on national fish portion sizes (USEPA 1997a) and information from a local fish consumption survey (ATSDR 1989). The Agency for Toxic Substances Disease Registry (ATSDR) performed a study in 1989 examining fish consumption in the Coeur d'Alene Basin. The survey encompassed three populations: members of the Coeur d'Alene Tribe, fishing license holders, and volunteers. Individuals were asked about the amount and type of locally caught fish they consumed. They also provided blood and urine samples for analysis of lead and cadmium. The survey results were reported as the number of meals with fish per week. Participants' answers were placed into the following categories: 0 meals per week; less than 1 meal per week; 1 to 2 meals per week; 3 to 6 meals per week; and 7 to 14 meals per week.

The largest percentage of volunteer and fishing license populations (approximately 70 percent and 74 percent, respectively) reported that they ate less than one fish meal per week. The second largest category (1 to 2 fish meals per week) consisted of 20 percent of volunteers and 16 percent of individuals with a fishing license. Approximately 78 percent of the tribal population reported that they ate 0 fish meals per week, while 12 percent reported eating 1 to 2 fish meals per week. The tribal population was the only group with a significant percentage (7 percent) in the highest category (7 to 14 meals per week).

In order to estimate a fish ingestion rate from the ATSDR survey data, an assumption was made about the amount of fish consumed per meal. The study reported the amount of fish eaten per meal and estimated portion size. The average portion size based on a dressed 8-inch trout for adult males and females was 252 grams.

Alternatively, the EPA (USEPA 1997a) recommends a 50th percentile fish portion size per meal of 129 g and a 95th percentile portion size of 326 g per the general population. With one fish meal per week for the surveyed population and the 95th percentile portion size, the fish ingestion rate would be approximately 46 g/day (one meal per week x 326 g per meal/7 days per week). This value was used to represent the RME recreational fish consumption rate. The value of 25 g/day was used as the CT fish consumption rate. EPA's recommended ingestion rate for freshwater anglers is 25 g/day (USEPA 1997a).

Exposure Frequency. For upland soil exposures, the selected exposure frequency was twice per week for 8 months (April through November), or 34 weeks per year. Exposure was assumed to occur over a 7-hour period for each event, based on the recommended children's weekend outdoor exposure time (USEPA 1997a). Hours per day were normalized to day per year assuming 14 waking hours (see Table 3.24).

Recreational exposure frequencies of 32 days per year for beach sediment and surface water exposures were assumed for public access beaches, based on 2 days per week from June through September. This exposure frequency was also selected for the expedited screening level risk assessment for common use areas surrounding Coeur d'Alene Lake, with input from state, local, and tribal agencies (Appendix B).

Skin Surface Area. For adults exposed to soil at upland parks and schools, a skin surface area of 2500 cm^2 was selected; this represents an adult wearing a short-sleeved shirt, with exposed skin surface limited to face, hands, and forearms (USEPA 1998e). For child exposure to soil at upland parks and schools, a skin surface area of 2200 cm^2 was selected; this represents a child (age 0 to 6) wearing a short-sleeved shirt and shorts, but no shoes; exposure is to head, hands, forearms, lower legs, and feet (USEPA 1998e).

For exposures to soil and sediment at public beaches, exposure of the entire body was assumed; this corresponds to 18,000 cm^2 for adults and 6500 cm^2 for children ages 0 to 6 (USEPA 1998e).

Adherence Factor. Soil/sediment adherence factors of 0.1 and 0.2 were assumed for adults and children, respectively. These values are based on data reported by Kissel et al. (1996a, 1996b, 1998) and Holmes et al. (1999).

Occupational Exposure Factors

Occupational exposure factors apply to construction workers and other workers who have more exposure than the average adult.

Soil Ingestion Rate. An RME soil ingestion rate of 300 mg/day for a construction worker was selected. This value is for an adult occupational exposure scenario with extensive soil contact; it is based primarily on the work of van Wijnen et al. (1990). However, the van Wijnen et al. study involved a 3- to 5-day exposure of children staying at campgrounds with the objective of maximizing the possibility of direct contact with soil. According to recent EPA Region 10 guidance (USEPA 1999d), adults in activities involving direct contact with soil would be unlikely to have soil ingestion rates greater than those of the children in this short-duration study. A recent work by Stanek et al. (1991), provides a 95th percentile ingestion rate of about 300 mg/day for adults over a 4-week period while engaged in routine day-to-day activities. This estimate, as stated by the authors, is highly uncertain due to the small size of the study. Although the estimate is uncertain, the Stanek report provides evidence that adults can have soil ingestion rates in the magnitude of 300 mg/day. Adults in activities involving direct contact with soil would be unlikely to have soil ingestion rates lower than the upper percentile shown in this long-term study of adults in day-to-day activities, which may include occasional activities requiring direct soil contact (USEPA 1999d).

For the CT case, a soil ingestion rate of 200 mg/day was assumed. This represents the recommended value for adult occupational soil ingestion in a soil contact exposure scenario.

Exposure Frequency. An RME exposure frequency of 195 working days per year (5 days per week for 39 weeks) was assumed for the construction worker. This is equivalent to a 9-month construction season (March to November, excluding 3 months for snow cover). For the CT case, an exposure frequency of 43 days per year was assumed (5 days per week for 8.7 weeks). This is equivalent to a 2-month construction project, the assumed length of time for soil contact-intensive activities related to the construction of a housing subdivision or commercial structure. Hours per day were normalized to days per year assuming 14 waking hours (See Table 3-24).

Skin Surface Area. Exposed skin surface area for a construction worker was assumed to be 2500 cm^2; this corresponds to exposure to face, forearms, and hands (USEPA 1998e).

Adherence Factor. A soil adherence factor of 0.1 was assumed for construction workers. This value is based on data for gardeners reported by Kissel et al. (1996a, 1996b, and 1998) and Holmes et al. (1999).

Traditional Tribal Subsistence Exposure Factors

Both the traditional subsistence and current subsistence lifestyles have been characterized, and appropriate tribal exposure factors and consumption rates have been developed. Generally, the various tribal exposure pathways and consumption rates included for use by the Coeur d'Alene Tribe were originally developed from research conducted with the Umatilla Tribe in Northeastern Oregon (Harris and Harper 1997). The results of that research were subsequently utilized within the Hanford Screening Assessment under a subsistence resident scenario (CRCIA).

Seasonality. The research described in Section 3.2.4 suggests that a seasonal difference in potential aboriginal exposure to a contaminated environment exists. Present-day tribal members practicing strict adherence to a traditional way of life and consuming fresh food would be subject to a potentially large seasonal variation in exposure. However, because the traditional native diet for all members of the tribe was comprised of roughly one-third resident fish, one-third large mammals, and the remaining one-third of roots and berries; and because the preservation of food for later consumption is known to have occurred; it is reasonable to assume that diet and exposure remained constant throughout the year regardless of gender.

Gender. Due to the shared division of labor between men and women, there would have been little variation in exposure due to gender-specific traditional activity. Tribal men, women, and children would have been exposed to contaminated surface water and sediment through the traditional methods of fishing and gathering riparian vegetation (including the water potato). Surface water was also used for bathing and cooking that would have resulted in additional exposure to all tribal members.

Potential exposure factors that are inclusive of the most conservative exposure pathways and consumption rates for all tribal individuals are presented in Table 3-26a. The Traditional Subsistence exposure factors presented have been developed under the assumption that the traditional subsistence lifestyle involved residing within the floodplain almost the entire year.

The justification for the values assigned for each exposure factor are largely derived from the Stuart Harris and Barbara Harper article titled "A Native American Exposure Scenario" that was published in *Risk Analysis*; the article provided representative values for the Umatilla Tribe that were generated through interviews with tribal members. The soil and sediment ingestion rate used for adults and children is 300 milligrams (mg) per day, derived from a campground study cited in the 1999 EPA Region 10 Supplemental Guidance for Soil Ingestion Rates (van Wijnen et al. 1990). EPA also recommends that 300 mg per day ingestion rate be used for a "soil contact intense" adult exposure scenario. The dermal contact rate for soil that will be used in the risk calculations is 0.8 mg/cm^2. This value represents a midrange adherence rate, based on studies by Kissel et al. and Holmes et al. The skin surface area of 2500 cm^2 used for the soil exposure route represents the face, hands, and forearms for adults as defined by EPA; for children aged 0 to 6, the value used is 2200 cm^2 and represents the head, hands, forearms, lower legs, and feet. For exposure to sediment, the skin surface area used for adults is 18,000 cm^2 and 6500 cm^2 for children, representing full body exposure. Exposure frequency

for tribal members to sediment and surface water (incidental ingestion pathway) is 210 days per year, the equivalent of 7 months when weather constraints are considered. The exposure frequency for all other routes and pathways is 365 days per year. Surface water ingestion of 3 liters per day for adults represents greater consumption of water compared to the EPA suggested value of two liters per day for average total fluid intake; the child ingestion rate is 1.5 liters per day is also equivalent to 150% of the EPA suggested value. It is assumed that the Coeur d'Alenes consume more water based on their active lifestyle and the climate in the Coeur d'Alene Basin. The fish consumption value was derived from interviews conducted by Harris and Harper; the diet for the Umatilla Tribe largely consists of fresh and dried salmon and resident fish from the Columbia River. Similarly, the Coeur d'Alenes are dependent upon fish from the Coeur d'Alene River, and the rate of 540 grams per day is used in this risk assessment. The 574 grams per day for a 70 kilogram adult as a consumption rate for the water potato is the EPA recommended intake for fruits and vegetables by urban and suburban Native American. This value is equivalent to 8.2 grams per kilogram per day for the traditional scenario. Some studies on the traditional subsistence diet have shown higher consumption rates, but the interviews conducted by Harris and Harper indicated that the Umatilla ingestion rate is closest to the EPA value.

Current Subsistence Scenario

The Current Subsistence exposure factors requested by the Coeur d'Alene Tribe are intended to represent current tribal members utilizing traditional hunting and gathering activities and a subsistence diet only. As suggested in characterizing traditional activities, potential exposure factors that are inclusive of the most significant pathways are included in Table 3-26b. These estimates reflect conservative values for potential exposure pathways and consumption rates for all tribal individuals.

The exposure duration for current subsistence activities is reduced to 61 days per year for exposure to surface water, sediment, and soil. This value represents the warmest two months of the year for a permanent residence in the floodplain. A fish ingestion rate of 170 grams per day for 365 days per year is assumed to represent the 95th percentile consumption rate. This value was derived from a study of four Columbia River tribes and is outlined in EPA's Exposure Factors Handbook (USEPA 1997a). The skin surface area for the current subsistence scenario is increased to 5700 cm^2 for adults and 2800 cm^2 for children to represent more dermal exposure during the summer months. These values are EPA's suggested values for outdoor exposure. The ingestion rate for wild plants harvested in the floodplain is 1.6 grams per kilogram per day, equal to 20% of the traditional subsistence ingestion rate of 8.2 grams per kilogram per day.

3.4 QUANTIFICATION OF LEAD EXPOSURE

3.4.1 Child Lead Model Overview

The current EPA risk assessment method for evaluating lead uses a mathematical model designed to predict the probable blood lead (PbB) concentrations for children between 6 months and 84 months of

age who have been exposed to lead through environmental media (air, water, soil, dust, and diet). The EPA model is referred to as the Integrated Exposure Uptake Biokinetic (IEUBK) Model (USEPA 1994a). The model has the following four functional components:

- **Exposure Component:** The exposure component uses environmental media-specific consumption rates and lead concentrations to estimate media-specific lead intake rates for air, water, soil, dust, and diet.
- **Uptake Component:** The uptake component uses media-specific fractional uptake and lead intake into the lungs or digestive tract to estimate the amount of lead absorbed into the child's blood.
- **Biokinetic Component:** The biokinetic component accounts for the transfer of lead between blood and other body tissues, or the elimination of lead from the body altogether.
- **Probability Distribution Component:** The probability distribution component shows a probability of a certain outcome (e.g., a PbB concentration greater than 10 µg/dl of blood in an exposed child based on the parameters used in the model).

The IEUBK Model combines assumptions about lead exposure and uptake with assumptions on how lead behaves in the body to predict a central tendency estimate (CT) PbB concentration for a child between 6 months and 84 months of age. Children in this age group are considered by the EPA to be sensitive age group for lead exposure because, compared to older children, they ingest more soil, absorb more lead from the gastrointestinal tract, and are more sensitive to the effects of lead. Within the 6 to 84 month age group, children between 24 and 36 months are especially sensitive to lead health effects because blood lead levels tend to peak at this time when children are especially vulnerable to neurological effects at this stage in their neurological development (Rodier 1995).

The IEUBK's estimated risk of elevated blood lead levels corresponds to cumulative exposure to a multimedia set of environmental lead levels, generally at and around a residence, with which a child or group of children would have contact while living there. This estimated risk is intended to describe the potential for elevated blood lead for any children who would have similar exposure, not just the current residents (Hogan et al. 1998).

In addition to predicting a CT PbB concentration, an estimation of variation in PbB is applied to the CTE to predict the probability of an individual child's PbB concentration exceeding a given PbB level. The EPA and the Centers for Disease Control and Prevention (CDC) have determined that childhood PbB concentrations at or above 10 µg/dl present risks to the child's health. Therefore, the target (acceptable) distribution of childhood PbB concentrations is one in which there is a probability of no more than 5 percent that the PbB concentration of a typical child will exceed 10 µg/dl (USEPA 1994a).

3.4.2 Adult Lead Model Overview

The EPA has developed an adult lead model (USEPA 1996c), which is designed to protect adult women of child-bearing age such that the 95th percentile value for fetal, PbB concentration is no more than 10 µg/dl. The developing fetus is the most sensitive population for adult worker exposure. Inputs to the model should be CT values, rather than RME values, because potential variability is accounted for using a geometric standard deviation (GSD) to approximate a potential distribution of PbB concentration.

The model estimates a PbB concentration using the following formula:

$$PbB_{maternal,\ central} = PbB_0 + [(BKSF \times SC \times IR_s \times AF_s \times EF_s) / AT] \qquad (17)$$

where

$PbB_{maternal,\ central}$	=	Maternal geometric mean blood lead concentration (µg/dl)
PbB_0	=	Baseline maternal blood lead concentration (µg/dl)
BKSF	=	Biokinetic slope factor (µg/dl per µg/day)
SC	=	Soil concentration at the site (mg/kg)
IR_s	=	Ingestion rate (g/day)
AF_s	=	Absorption fraction for soil
EF_s	=	Exposure frequency for contact with site soils
AT	=	Averaging time (the total period during which soil contact may occur, i.e., 365 days/year)

and

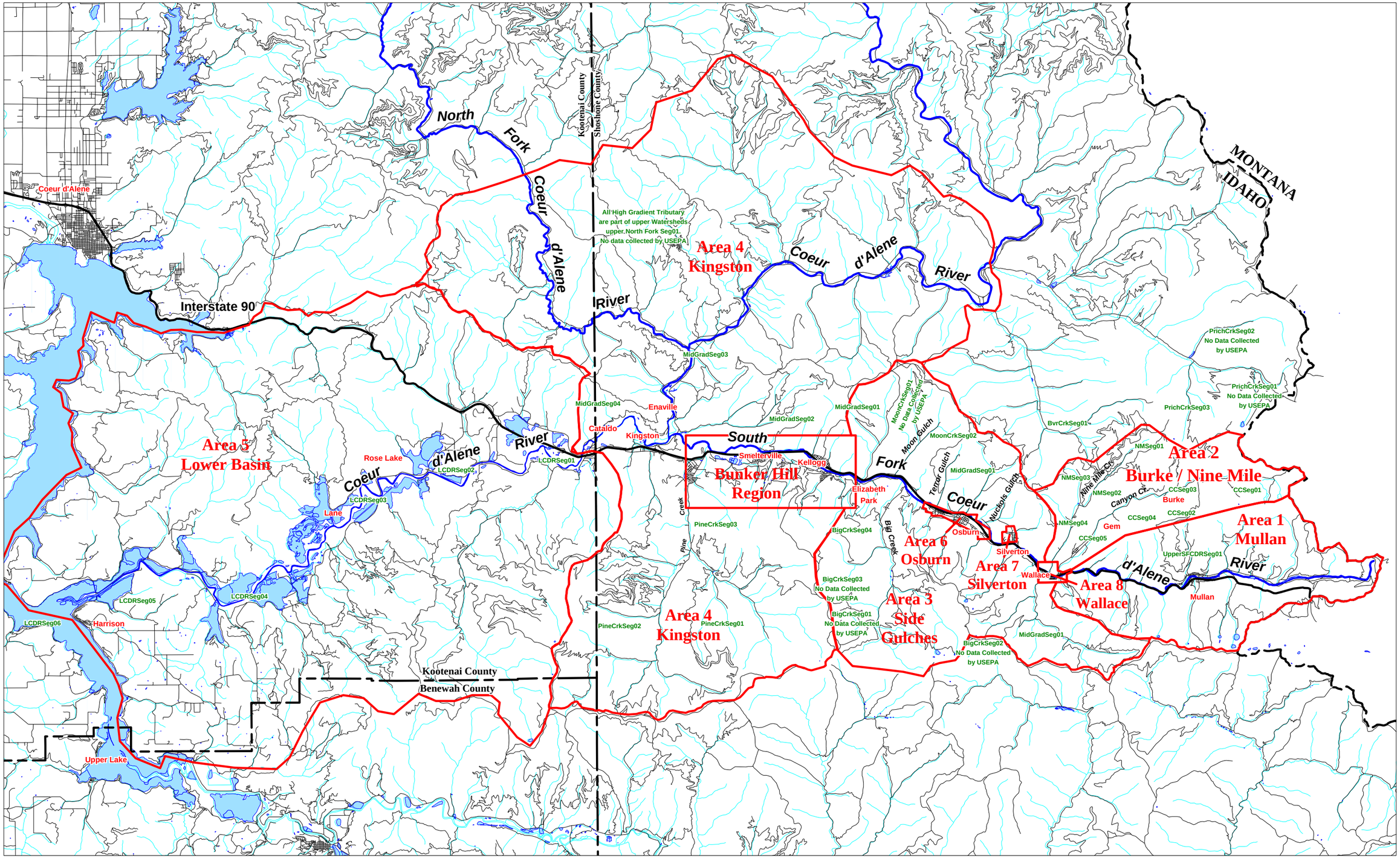
$$PbB_{maternal,\ goal} = \frac{PbB_{fetal,\ 0.95}}{(GSD^{1.645} \times R)}$$

where

$PbB_{maternal,\ goal}$	=	Goal for the mean blood lead concentration for pregnant women
$PbB_{fetal,\ 0.95}$	=	Goal for the 95th percentile blood lead concentration for fetuses born to women having been exposed to lead in site soils (10 µg/dl)
GSD	=	Geometric standard deviation for adults
1.645	=	Distribution variable related to percent above geometric mean blood lead concentration; i.e., student t value for $t_{0.95}$
R	=	Constant of proportionality between fetal blood lead concentration at birth and maternal blood lead concentration; the EPA default value (0.9) was used in this assessment

3.5 SUMMARY OF EXPOSURE ASSESSMENT

Chemical intakes were calculated as described in previous sections and are provided in Appendix A. The chemical intakes have been combined with the toxicity information presented in Section 4 to calculate the cancer risk and the noncancer hazard for the non-lead metals for each exposure scenario (Section 5). The results of the risk analysis for lead are presented in Section 6.



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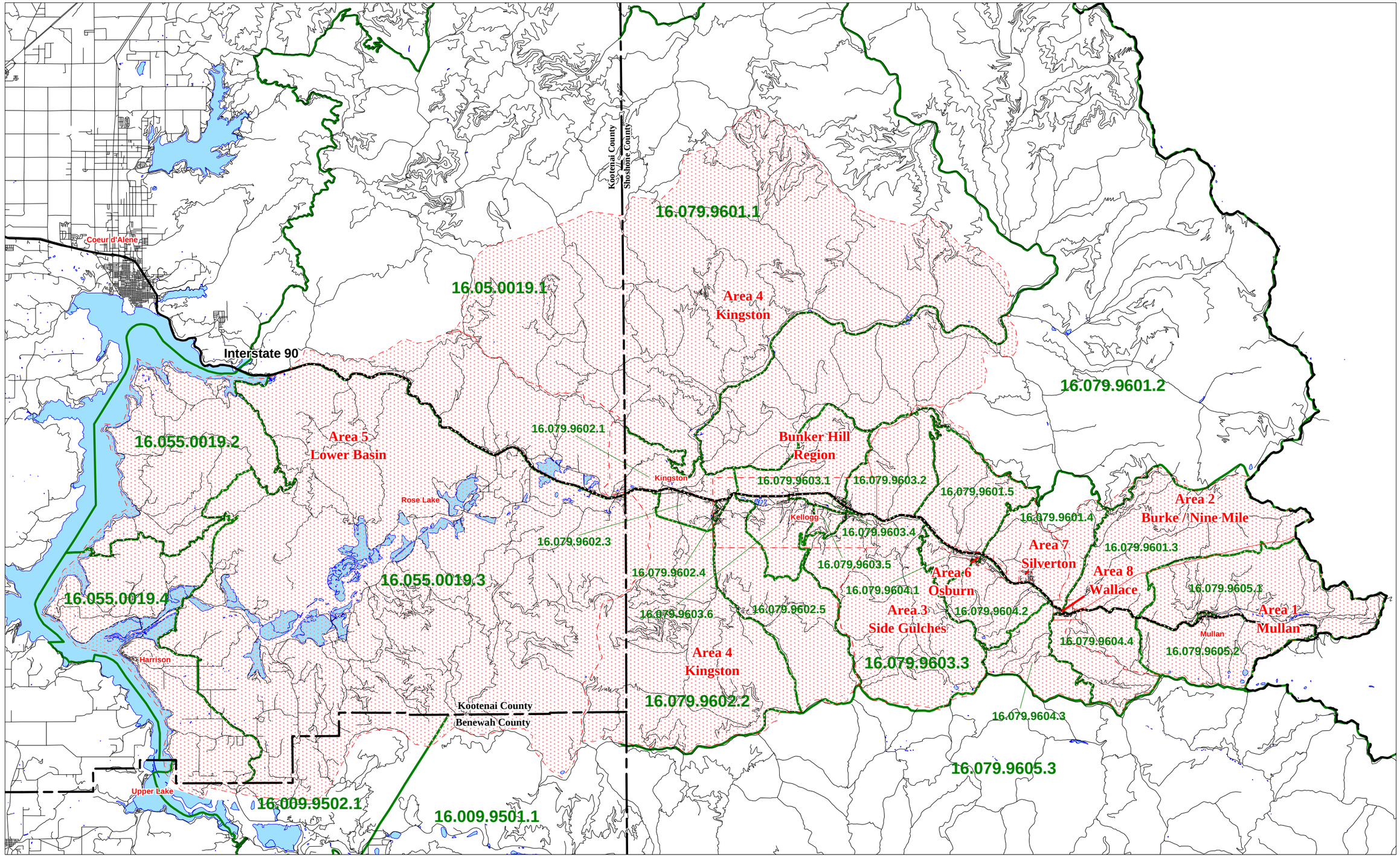
TerraGraphics Environmental Engineering, Inc.	FILE 1999-07-13 Basin RA / figure3-1	REQUESTOR L.Wichers	Coeur d'Alene Basin Human Health Risk Assessment	Basin Study Regions; Streams and Tributaries; Lakes; Roads
	DATE 26OCT99	DESIGNER B. Bailey		
	PROJECT NO 980111	CARTOGRAPHER B. Bailey		
		APPROVED S. Spalinger		

Figure 3-1a

Basin Study Regions



TerraGraphics
Environmental Engineering, Inc.

File	1999-07-13 Basin RA / figure3-1b	Requestor	A. Brooks
Date	26OCT99	Designer	S. Morgan, B. Bailey
Project No.	980111	Cartographer	B. Bailey
		Approved	

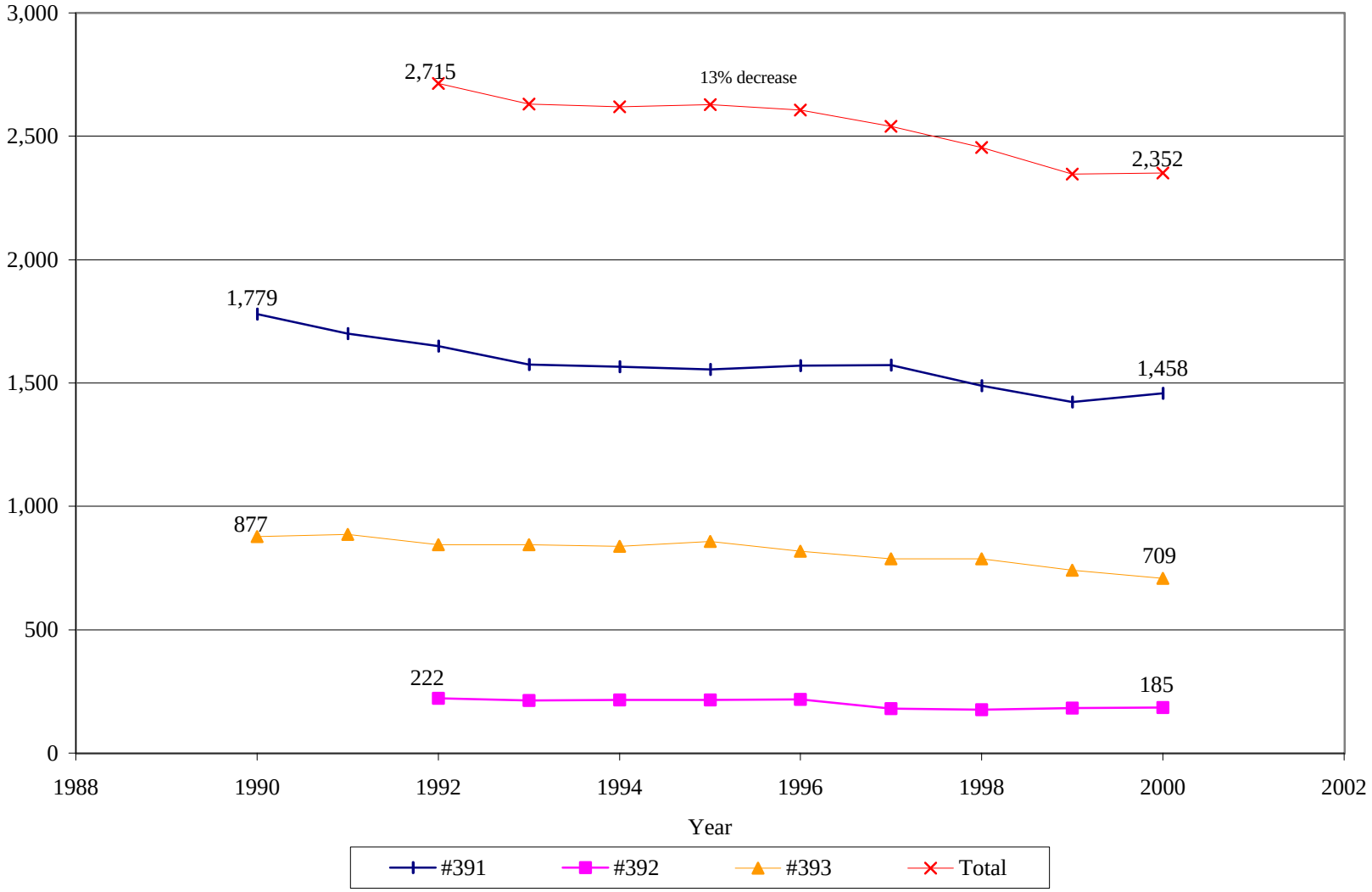
Coeur d'Alene Basin
Human Health Risk Assessment

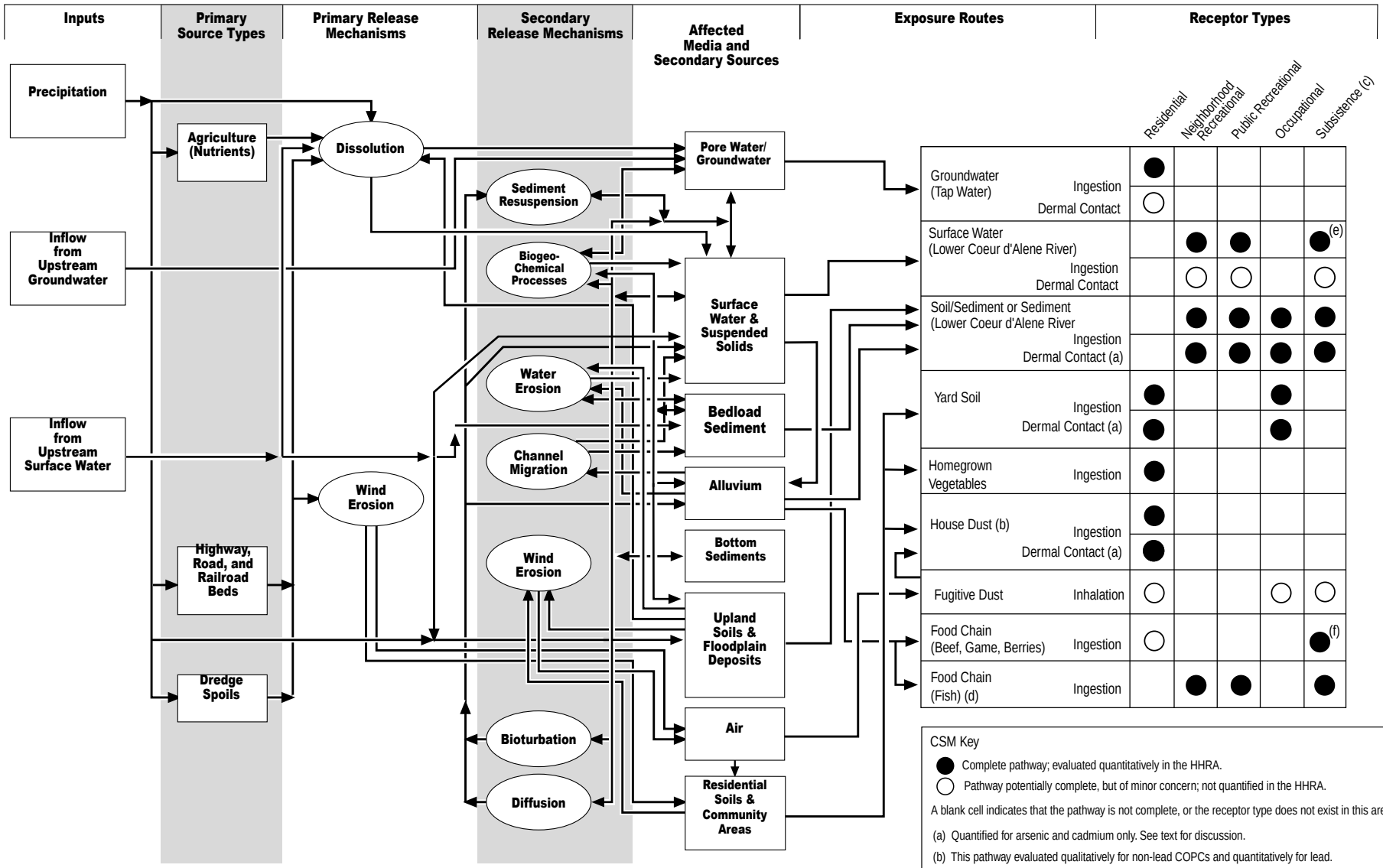
Basin Study Regions
Roads
Census Blocks

Figure 3-1b

Census Block Groups Overlaid Onto Basin Study Areas

Figure 3-2 Coeur d'Alene River Basin School District Enrollment 1990 to 2000





Exposure Route		Residential	Neighborhood Recreational	Public Recreational	Occupational	Subsistence (c)
Groundwater (Tap Water)	Ingestion	●				
	Dermal Contact	○				
Surface Water (Lower Coeur d'Alene River)			●	●		●(e)
	Ingestion		○	○		○
	Dermal Contact					
Soil/Sediment or Sediment (Lower Coeur d'Alene River			●	●	●	●
	Ingestion		●	●	●	●
	Dermal Contact (a)					
Yard Soil	Ingestion	●			●	
	Dermal Contact (a)	●			●	
Homegrown Vegetables	Ingestion	●				
House Dust (b)	Ingestion	●				
	Dermal Contact (a)	●				
Fugitive Dust	Inhalation	○			○	○
Food Chain (Beef, Game, Berries)	Ingestion	○				●(f)
Food Chain (Fish) (d)	Ingestion		●	●		●

CSM Key

● Complete pathway; evaluated quantitatively in the HHRA.

○ Pathway potentially complete, but of minor concern; not quantified in the HHRA.

A blank cell indicates that the pathway is not complete, or the receptor type does not exist in this area.

(a) Quantified for arsenic and cadmium only. See text for discussion.

(b) This pathway evaluated qualitatively for non-lead COPCs and quantitatively for lead.

(c) Both traditional and modern subsistence tribal member exposure scenarios quantitatively evaluated.

(d) Although presented for Lower Basin, exposure to recreationally caught fish could occur for residents of other areas.

(e) Exposures quantified for both "disturbed" and "undisturbed" water. See text.

(f) Water potatoes only.

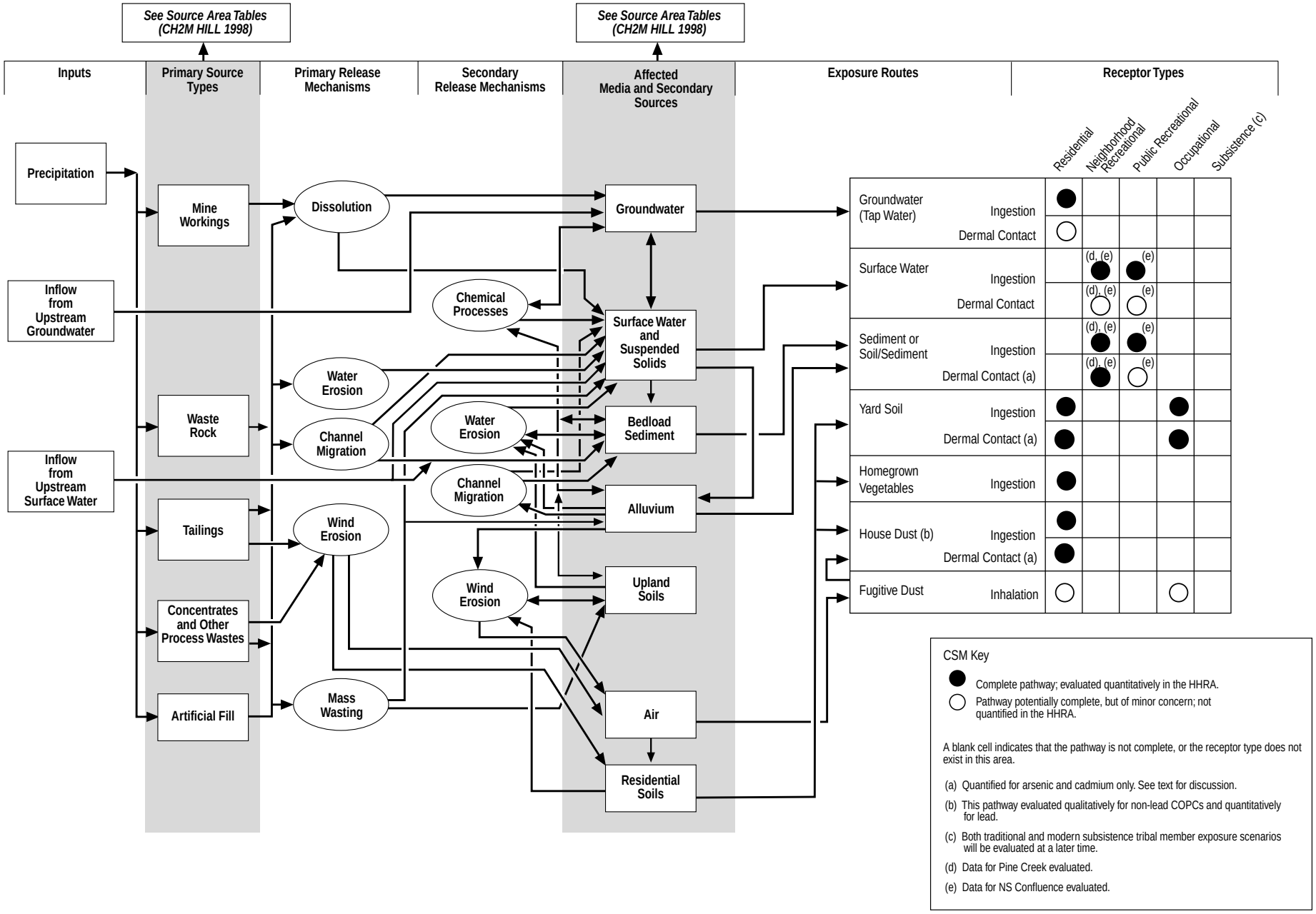
EPA REGION 10

027-RI-CQ-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc. Control:
Generation: 1

BHHRA-4
03/01/01

Figure 3-3
Lower Basin Conceptual Site Model



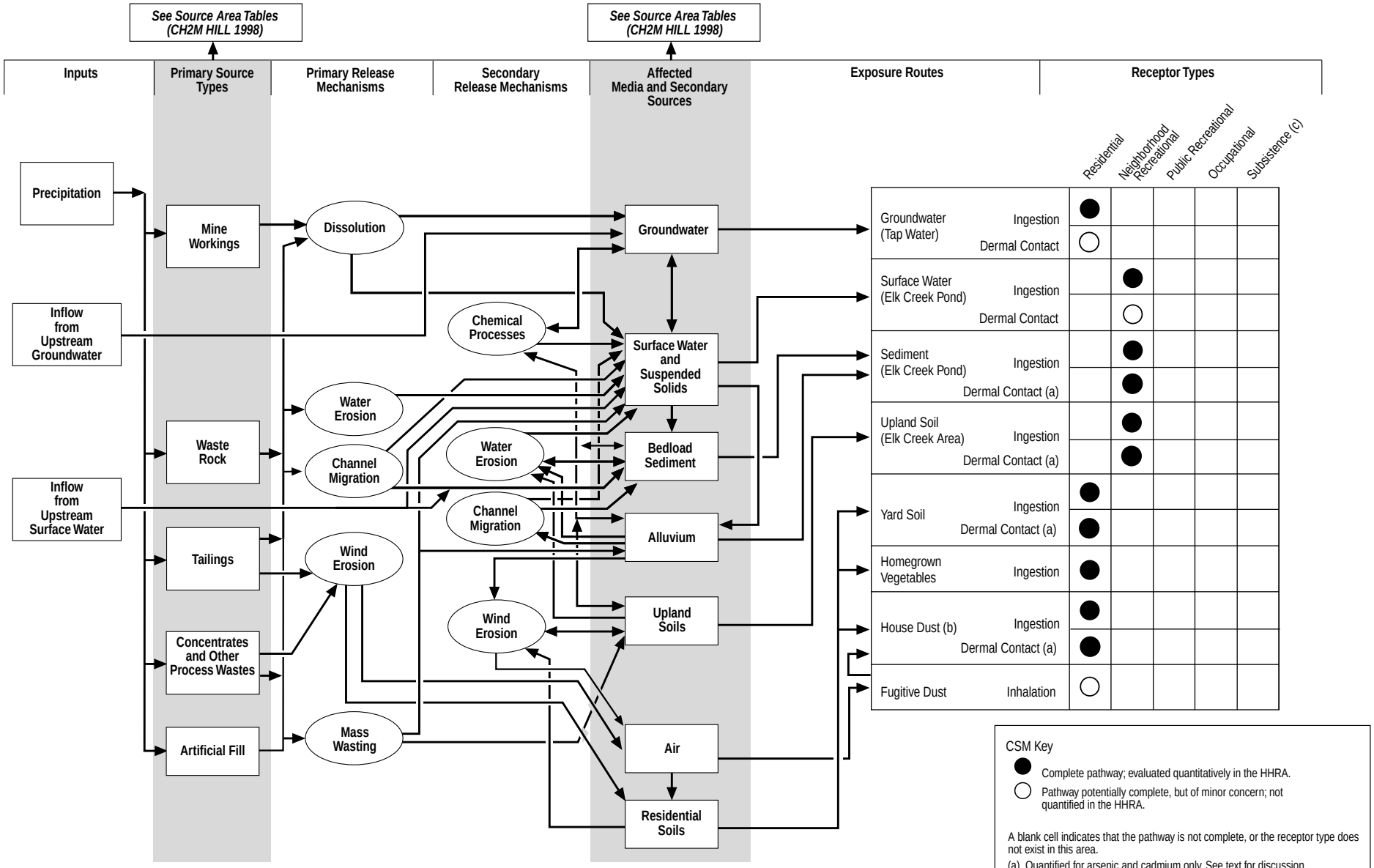
EPA REGION 10

027-RI-CQ-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc. Control:
Generation: 1

BHHRA-4
03/01/01

Figure 3-4
Kingston Conceptual Site Model



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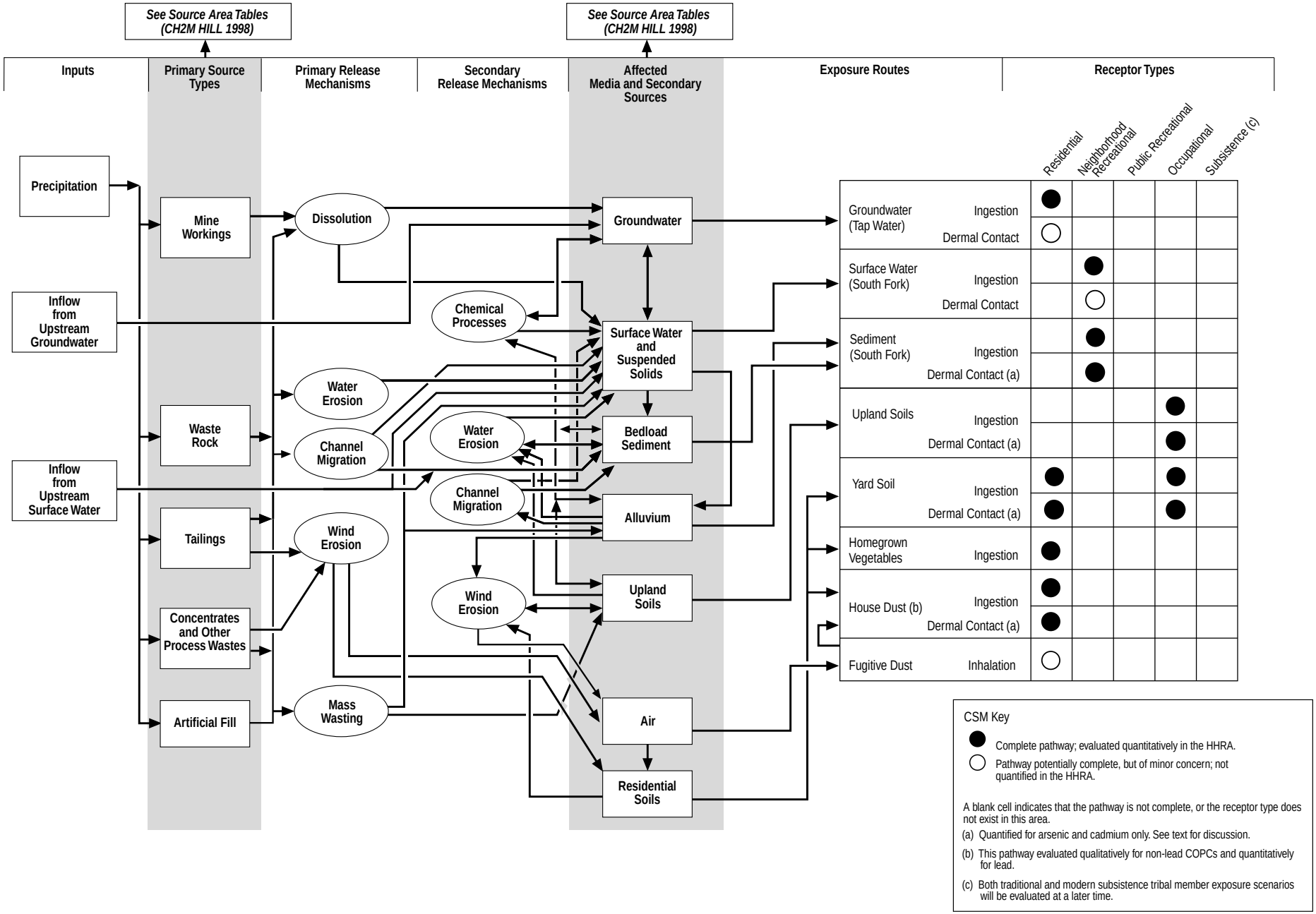
027-RI-CQ-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc. Control:
Generation: 1

BHHRA-4
03/01/01

Figure 3-5
Side Gulches Conceptual Site Model



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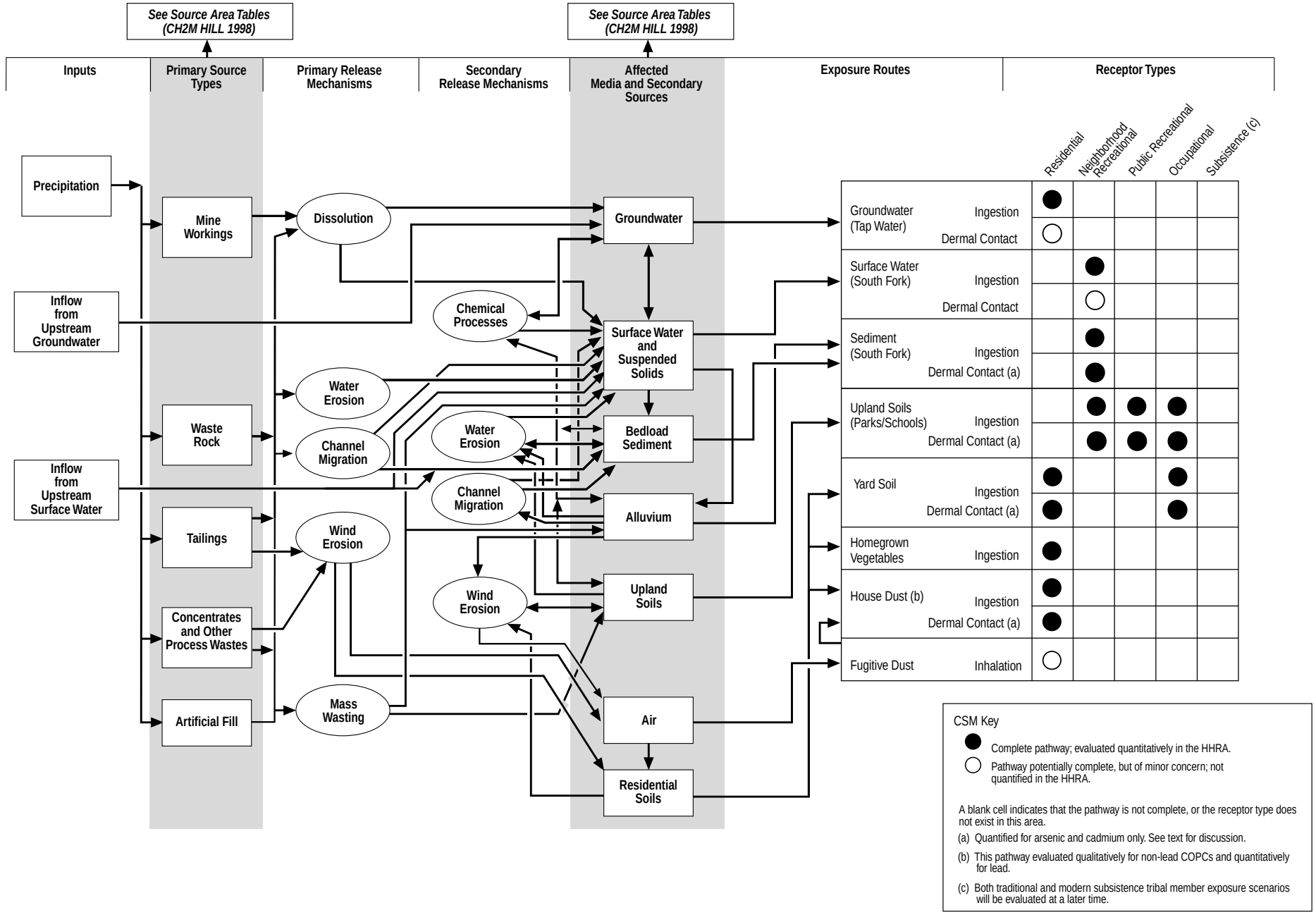
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc. Control:
Generation: 1

BHHRA-4
03/01/01

Figure 3-6
Osburn Conceptual Site Model



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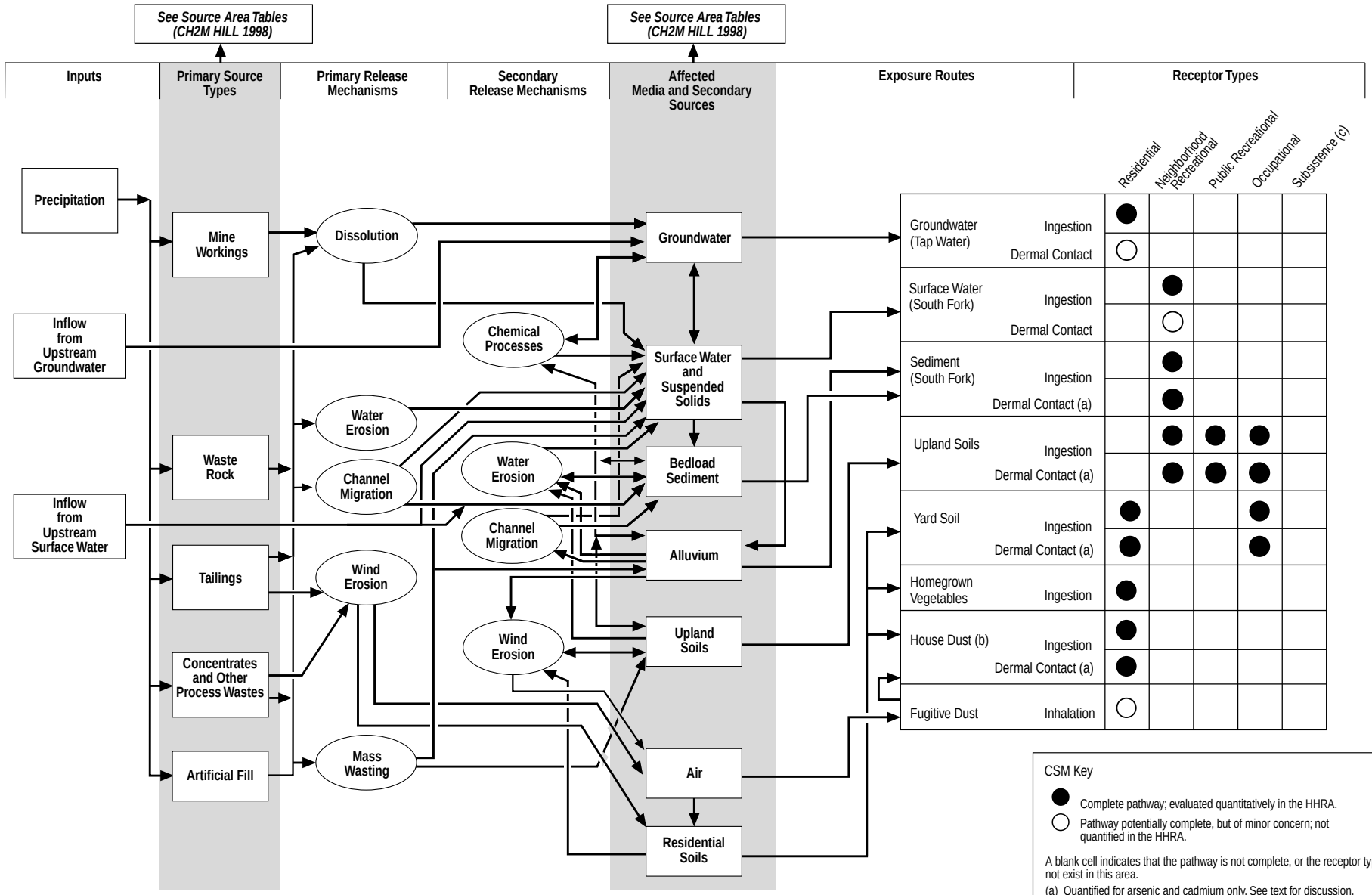
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc. Control:
Generation: 1

BHHRA-4
03/01/01

Figure 3-7
Silverton Conceptual Site Model



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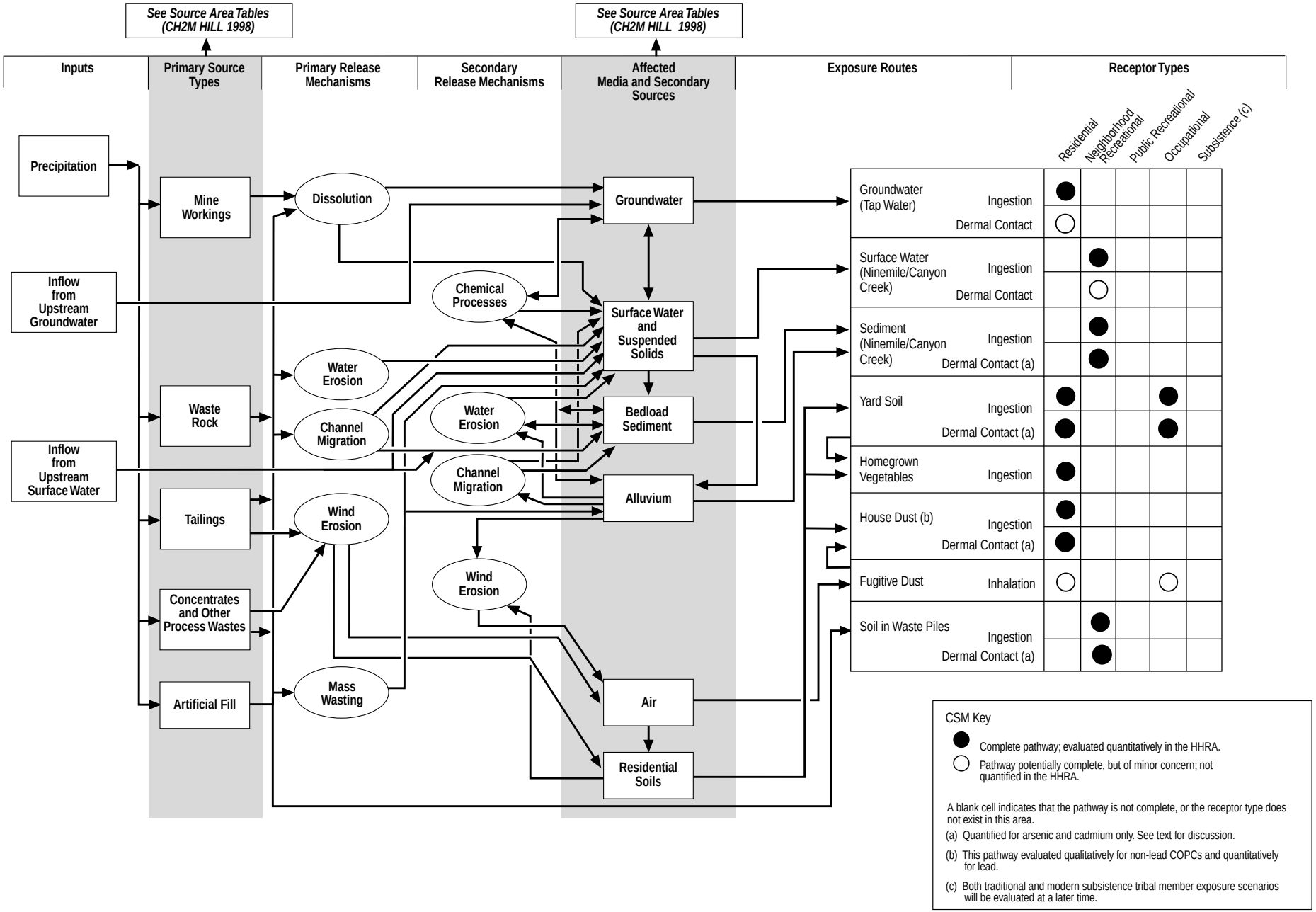
027-RI-CQ-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc. Control:
Generation: 1

BHHRA-4
03/01/01

Figure 3-8
Wallace Conceptual Site Model



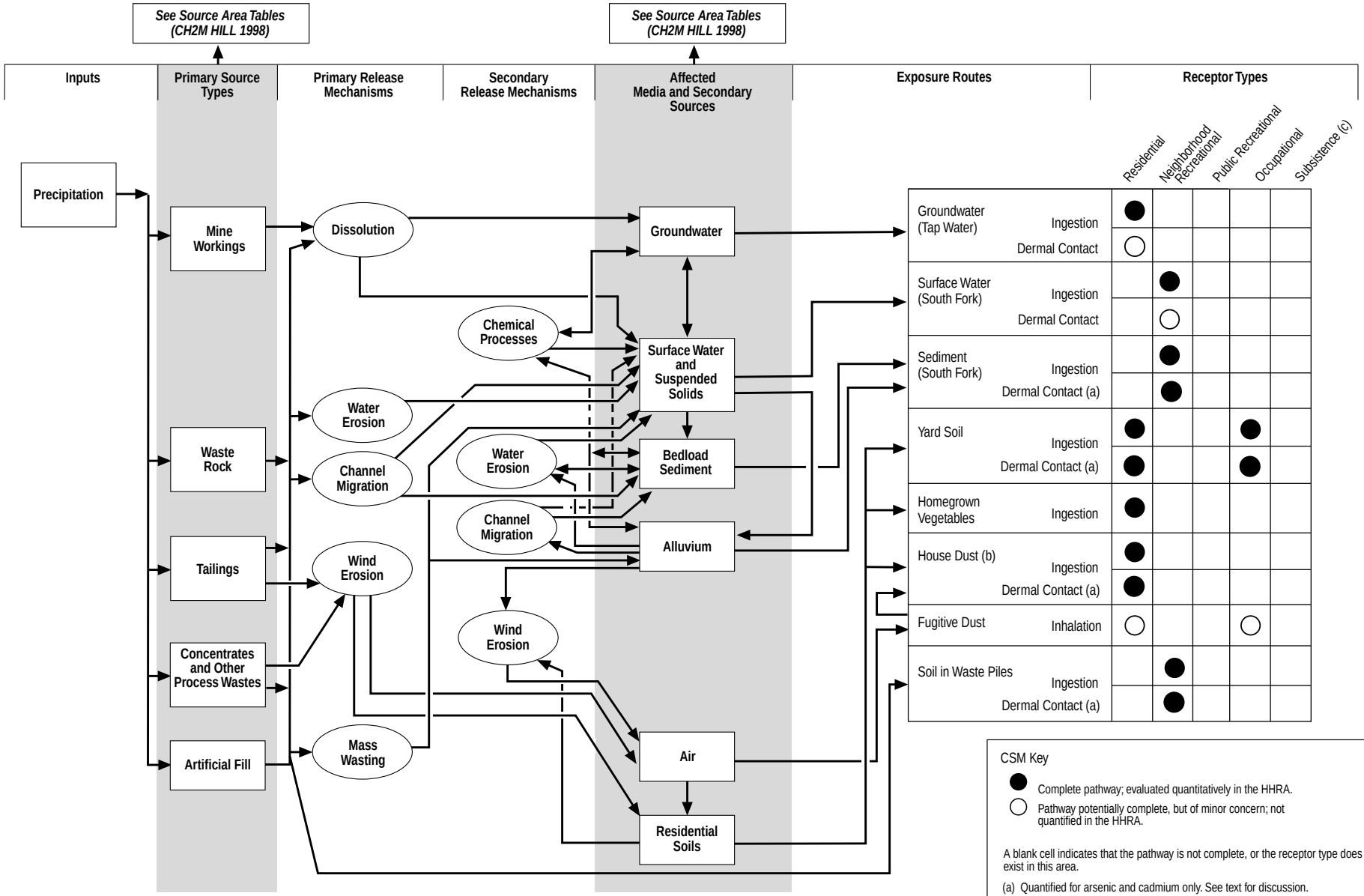
EPA REGION 10

027-RI-CQ-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc. Control:
Generation: 1

BHHRA-4
03/01/01

Figure 3-9
Ninemile Conceptual Site Model



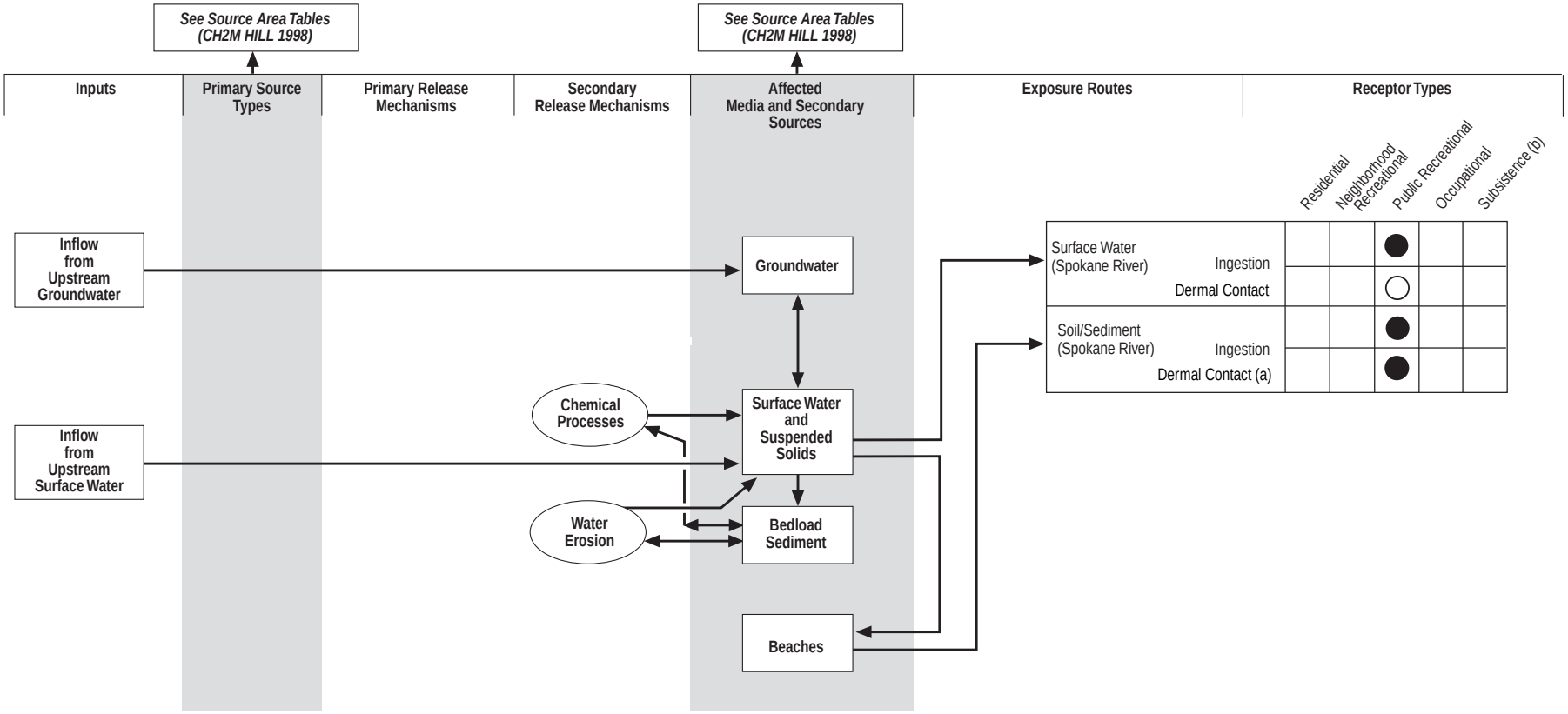
EPA REGION 10

027-RI-CQ-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc. Control:
Generation: 1

BHHRA-4
03/01/01

Figure 3-10
Mullan Conceptual Site Model



CSM Key

● Complete pathway; evaluated quantitatively in the HHRA.

○ Pathway potentially complete, but of minor concern; not quantified in the HHRA.

(a) Quantified for arsenic and cadmium only. See text for discussion.

(b) Both traditional and modern subsistence tribal member exposure scenarios will be evaluated at a later time.

A blank cell indicates that the pathway is not complete, or the receptor type does not exist in this area.

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027-RI-CQ-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc. Control:
Generation: 1

BHHRA-4
03/01/01

Figure 3-11
Blackwell Island Conceptual Site Model

Area 5 Lower Basin

Figure 3-12
Baseline Risk Assessment for
Area 5 Lower Basin "Southwestern Area"

Thompson Lake
CUA038
SD:5
SS:5
SW:5

RM135 Long Beach/Springston
CUA039
SD:10
SS:5
SW:5

West of Blue Lake
CUA041
SD:5
SS:5
SW:5

Medimont Boat Ramp
CUA045
SD:5
SS:5
SW:5

West Beach Near Medimont
CUA043
SS:5

RM 145
CUA048
SD:10
SS:5
SW:5

Across river from Springston
CUA036
SD:10
SS:5
SW:5

Medimont Hill Camping Area
CUA044
SS:5

Springston Beach Site
CUA035
SD:10
SS:5
SW:5

Harrison Beach
CUA018
SD:26
SW:7

Trestle area next to Route 97
CUA033
SD:5
SS:5

Rainy Hill Fishing Area
CUA046
SS:5

Rainy Hill Picnic Area
CA047
SD:2
SS:5
SW:5

Area 5 Lower Basin

Matchline for "Central Section and Southwestern Section"

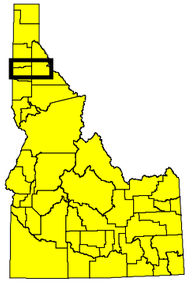
CSM Unit 3-4

Carlin Cr
Blue Lake Creek
Chatfield Creek
Killarne Lake
Lane Creek
Thompson Lake
Blue Lake
Springston
LCDRSeg05
Coeur D'Alene River
Medimont
LCDRSeg04
Swan Lake
Cave Lake
Coeur d'Alene Lake
LCDRSeg06
Anderson Lake
Harrison
Lacon
Black Lake
Black Creek
Lamb Creek
Conkling Park

LEGEND

Site Name
Site Number
Sample Type: # of Samples
SD (Sediment)
SS (Surface Soil)
SW (Surface Water)

- Common Use Area Sampling Location
- Stream
- Road
- City
- Basin Study Regions
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc.Technical Data Management database as of 3/29/00.

SCALE 1:60,000

0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\basin_risk_for_terragraphics.apr
VIEW: Blackwell Island
EXTENT:SW Lower Basin
LAYOUT: Lower Basin Southwestern Section
3/6/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

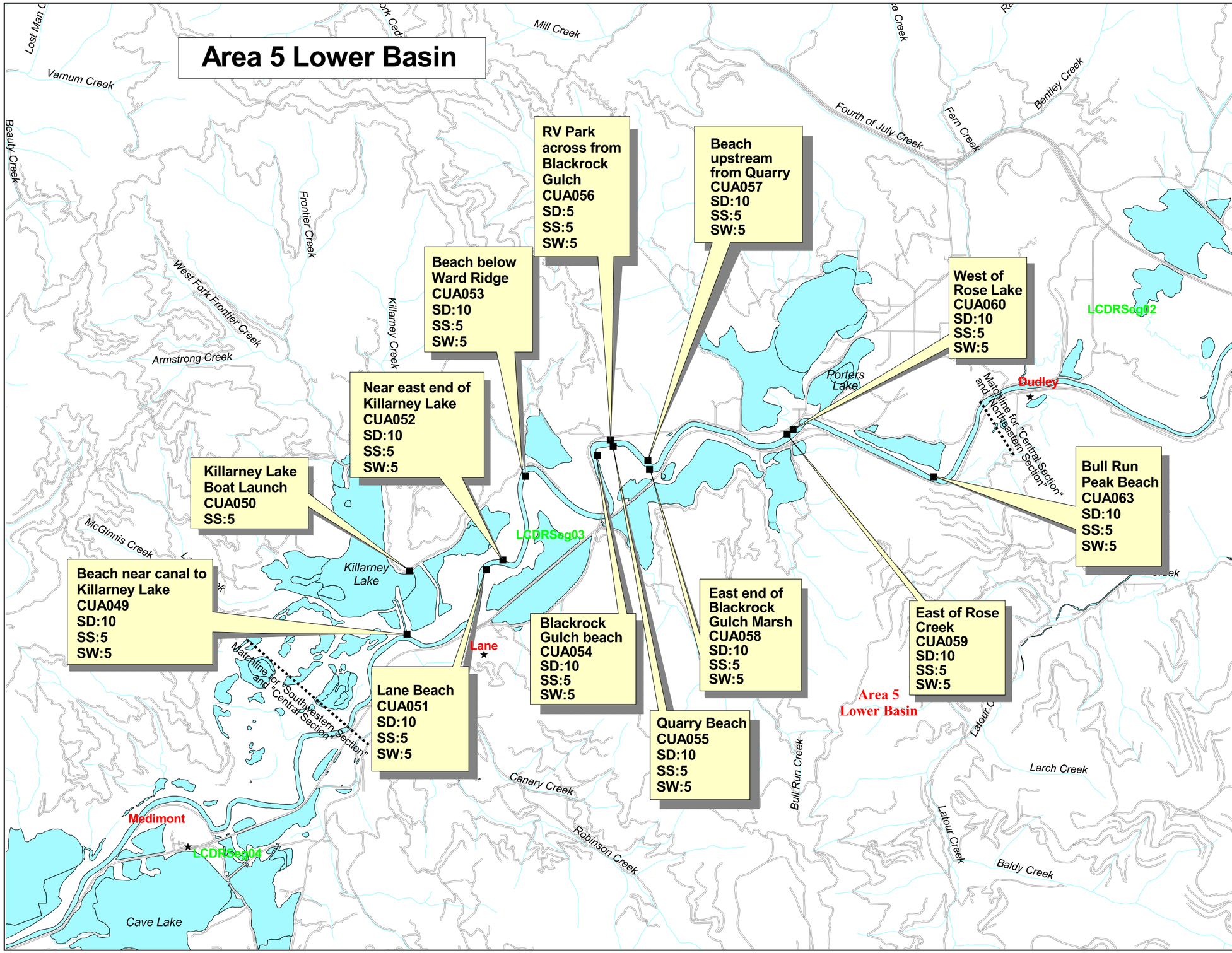
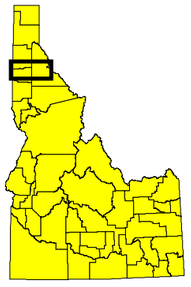


Figure 3-13
Baseline Risk Assessment for Area 5 Lower Basin "Central Section"

LEGEND

Site Name
Site Number
Sample Type:# of Samples
SD (Sediment)
SS (Surface Soil)
SW (Surface Water)

- Common Use Area Sampling Location
- Stream
- Road
- City
- CSM Unit Boundary
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 3/29/00.

SCALE 1:60,000

0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\basin_risk_for_terragraphics.apr
VIEW: killarney to rose lake
EXTENT:killarney to rose
LAYOUT: Lower Basin Area Central Section
3/6/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

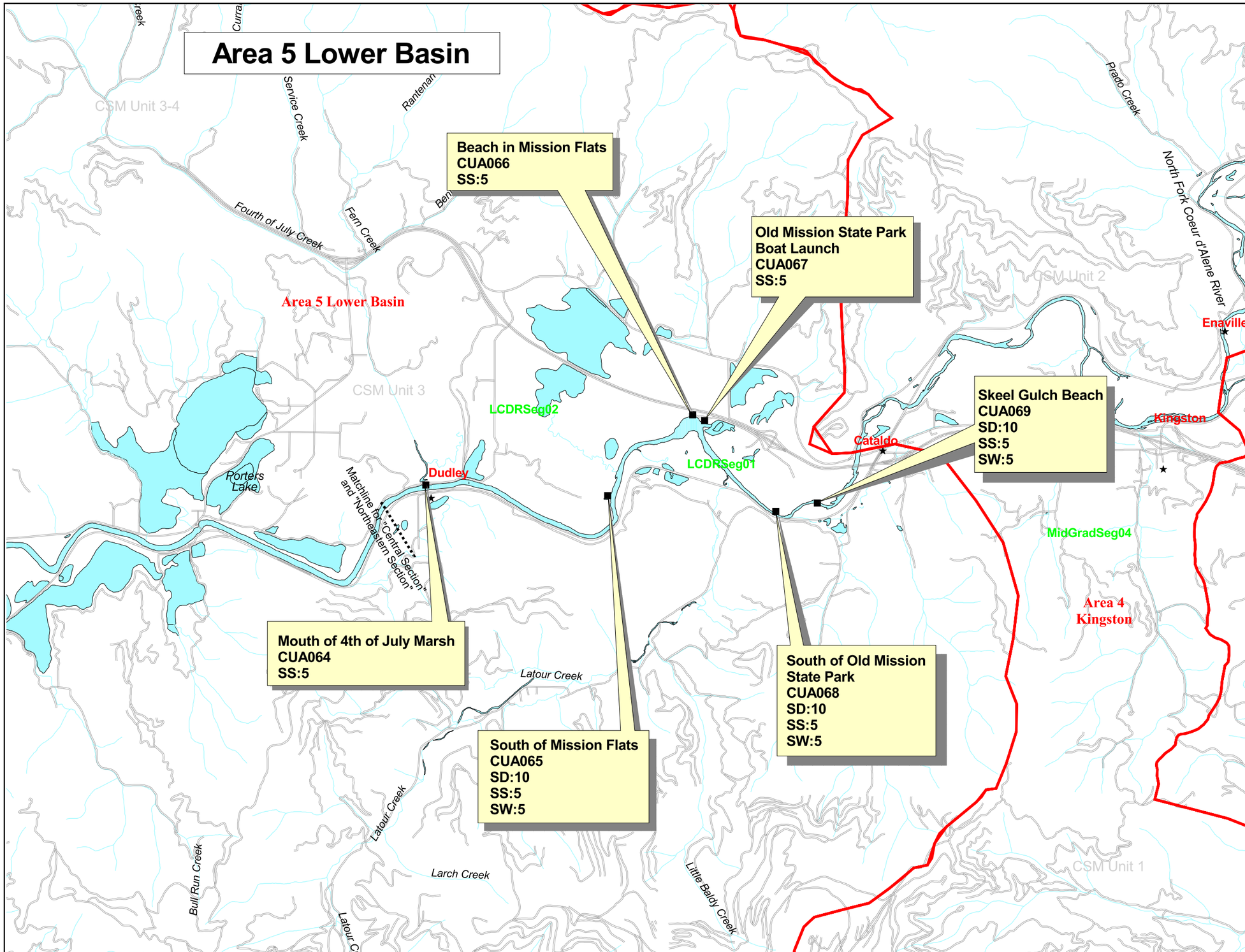
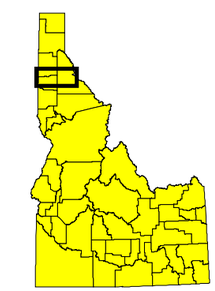


Figure 3-14
Baseline Risk Assessment for
Area 5 Lower Basin "Northeastern Section"

LEGEND

Site Name
Site Number
Sample Type:# of Samples
SD (Sediment)
SS (Surface Soil)
SW (Surface Water)

- Common Use Area Sampling Location
- Stream
- Road
- City
- Basin Study Region
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 3/29/00.

SCALE 1:60,000

0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\basin_risk_for_terragraphics.apr
VIEW: 4th of July to Skeel
EXTENT:NE Lower Basin
LAYOUT:lower basin NE section
3/6/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

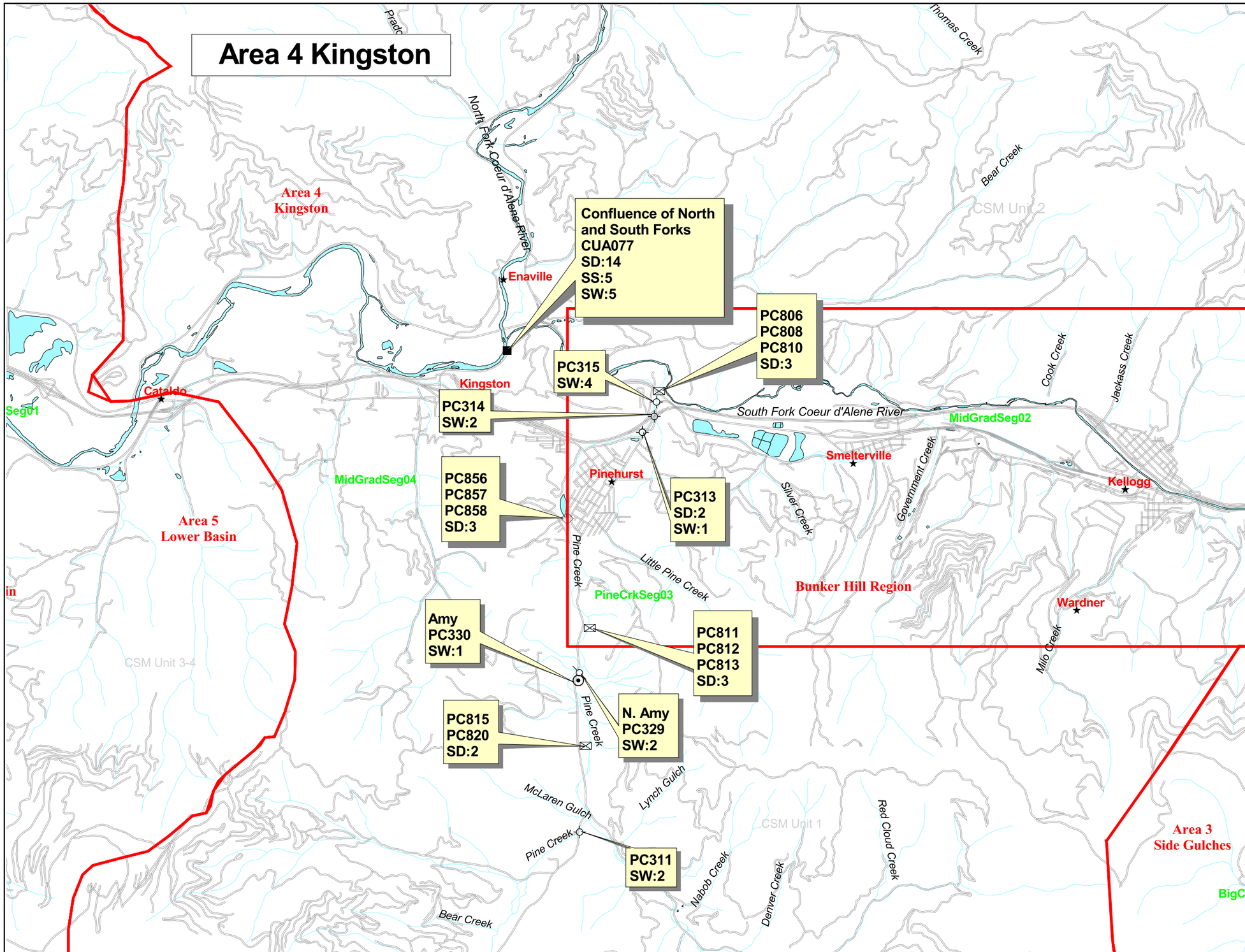
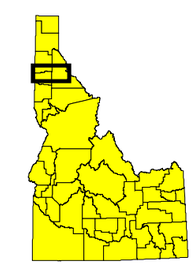


Figure 3-15
Baseline Risk Assessment for Area 4 Kingston

LEGEND

Site Name (Only CUAs and seeps are named)
Site Number
Sample Type: # of Samples
SD (Sediment)
SS (Surface Soil)
SW (Surface Water)

- Common Use Area Sampling Location
- River Sampling Location
- Adit Sampling Location
- Hand Auger Sampling Location
- Seep Sampling Location
- Test Pit Sampling Location
- Stream
- Road
- City
- Basin Study Region
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 3/29/00.

SCALE 1:60,000

0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\basin_risk_for_terragraphics.apr
VIEW: Kingston
EXTENT:Kingston
LAYOUT: Kingston
3/6/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

Area 3 Side Gulches

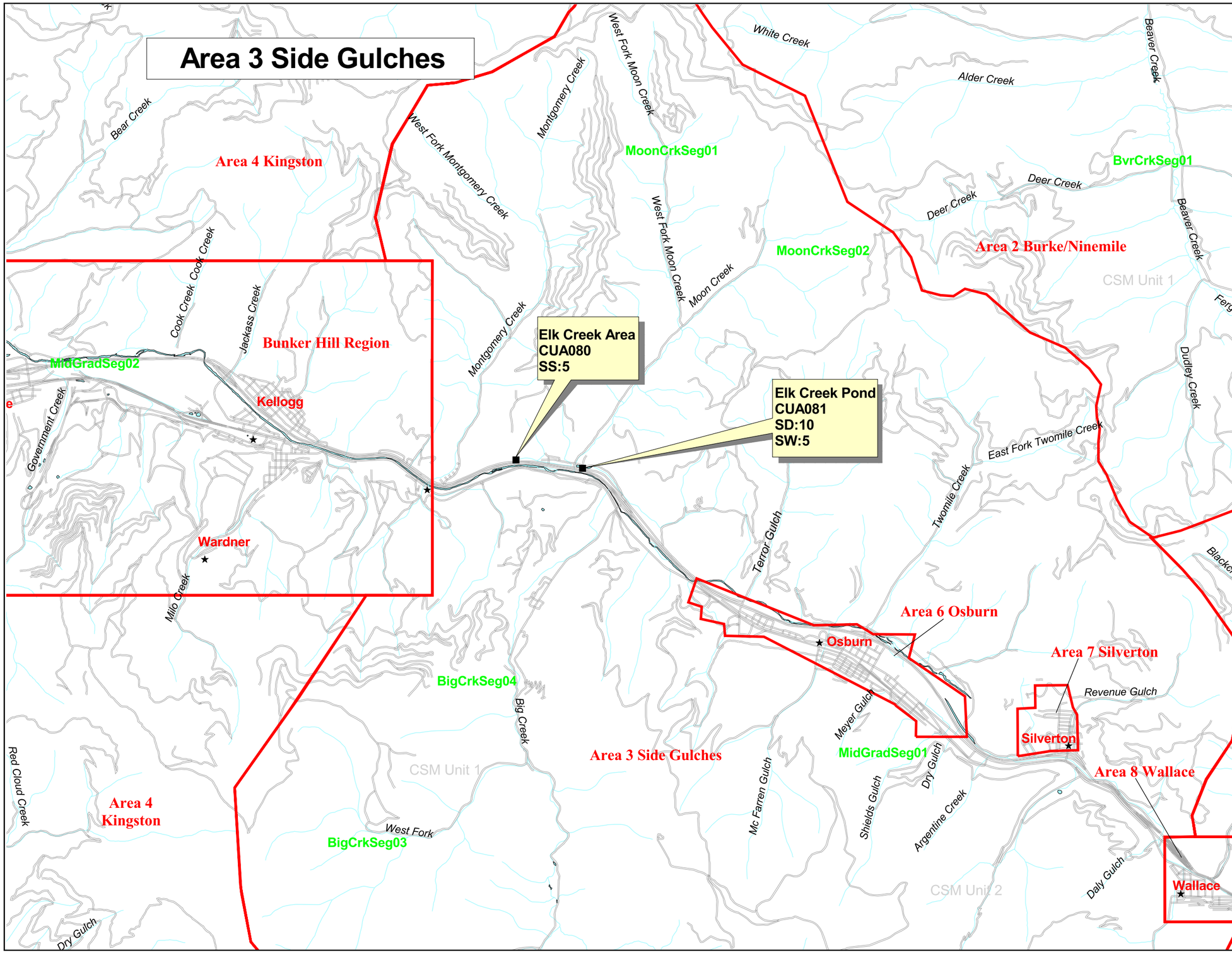
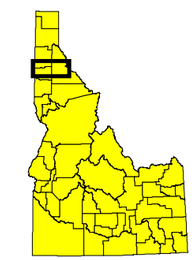


Figure 3-16
Baseline Risk Assessment for Area 3 Side Gulches

LEGEND

- Site Name
- Site Number
- Sample Type: # of Samples
- SD (Sediment)
- SS (Surface Soil)
- SW (Surface Water)

- Common Use Area Site Location
- Stream
- Road
- City
- Basin Study Regions
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 3/29/00.

SCALE 1:60,000

0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\basin_risk_for_terragraphics.apr
VIEW: Side Gulches
EXTENT:Side Gulches
LAYOUT: Side Gulches
3/6/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

Area 7 Silverton

Satner Field
CUA095
SS:39
SB:116

Silverton Ballfield next to Huggy Bear Day Care
CUA094
SS:19
SB:59

Wellman & Satner Field Parking Lot
CUA091
SS:19
SB:57

Wellman Field
CUA 089
SS:19
SB:58

Wellman Field Park
CUA090
SS:19
SB:58

Silverton School/Huggy Bear Day Care
CUA092
SS:36
SB:116

Area 6 Osburn
Area 7 Silverton
Area 2 Burke/Ninemile
Area 3 Side Gulches
Area 1 Mullan
Area 8 Wallace

Osburn
Silverton
Wallace
Bunn
Gem

BigCrkSeg04
MidGradSeg01
NMSeg02
NMSeg03
CCSeg04
CCSeg05
BigCrkSeg01
BigCrkSeg02

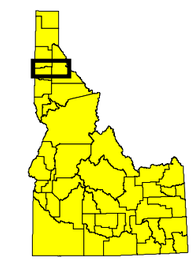
Montgomery Creek
Moon Creek
Ferguson Creek
Dudley Creek
East Fork Ninemile Cr
East Fork Twomile Creek
Twomile Creek
Terror Gulch
Ninemile Creek
Blackcloud Creek
Canyon Creek
Revenue Gulch
Meyer Gulch
Big Creek
Dry Gulch
Mc Farren Gulch
Argentine Creek
Shields Gulch
Trowbridge Gulch
Ruby Gulch
Dexter Gulch
Grouse Cr
Lake Creek
St Joe Creek
Rock Creek
Watson Gulch
East Fork
West Fork
Cranky Gulch
Placer Creek
Red Oak Gulch
Hord Gulch
Trowel Gulch
Flora Gulch
Dot Creek
Pen Creek
Elbow Gulch
CSM Unit 1

Figure 3-17
Baseline Risk Assessment for Area 7 Silverton

LEGEND

Site Name
Site Number
Sample Type: # of Samples
SS (Surface Soil)
SB (Subsurface Soil)

- Common Use Area Sampling Location
- Stream
- Road
- City
- Basin Study Regions
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc.Technical Data Management database as of 4/13/00.

SCALE 1:60,000

0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

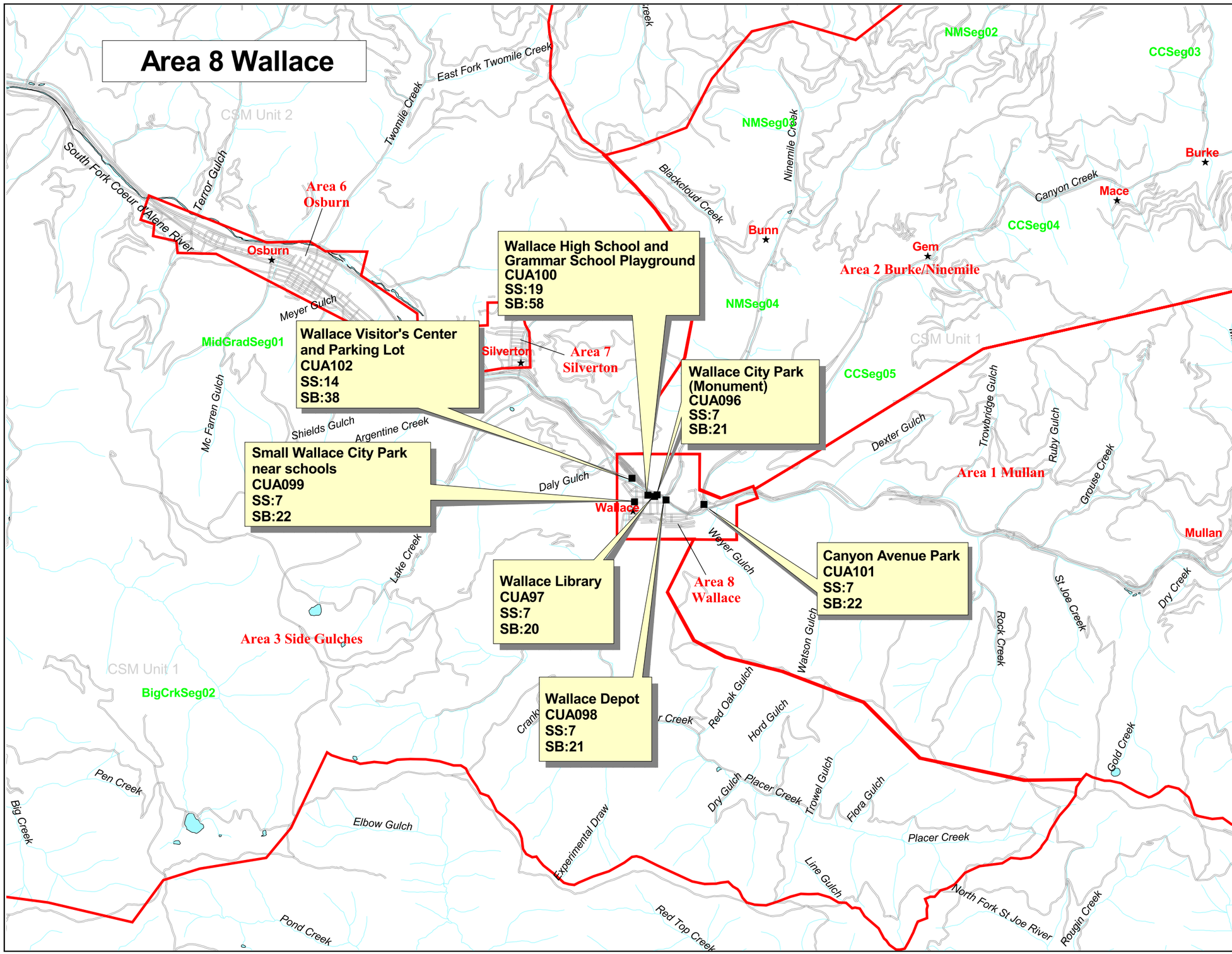
Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\basin_risk_for_terragraphics.apr
VIEW: Silverton
EXTENT:Silverton
LAYOUT: Silverton
3/6//2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

Area 8 Wallace

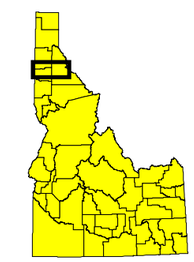
Figure 3-18
Baseline Risk Assessment for
Area 8 Wallace



LEGEND

Site Name
Site Number
Sample Type: # of Samples
SS (Surface Soil)
SB (Subsurface Soil)

- Common Use Area Sampling Location
- Stream
- Road
- City
- River Segment
- Basin Study Regions
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2MHILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 3/29/00.

SCALE 1:60,000

0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\basin_risk_for_
terragraphics.apr
VIEW: Wallace
EXTENT:Wallace
LAYOUT: Wallace
3/6/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

Areas 6 - 8 Osburn, Silverton and Wallace Combined

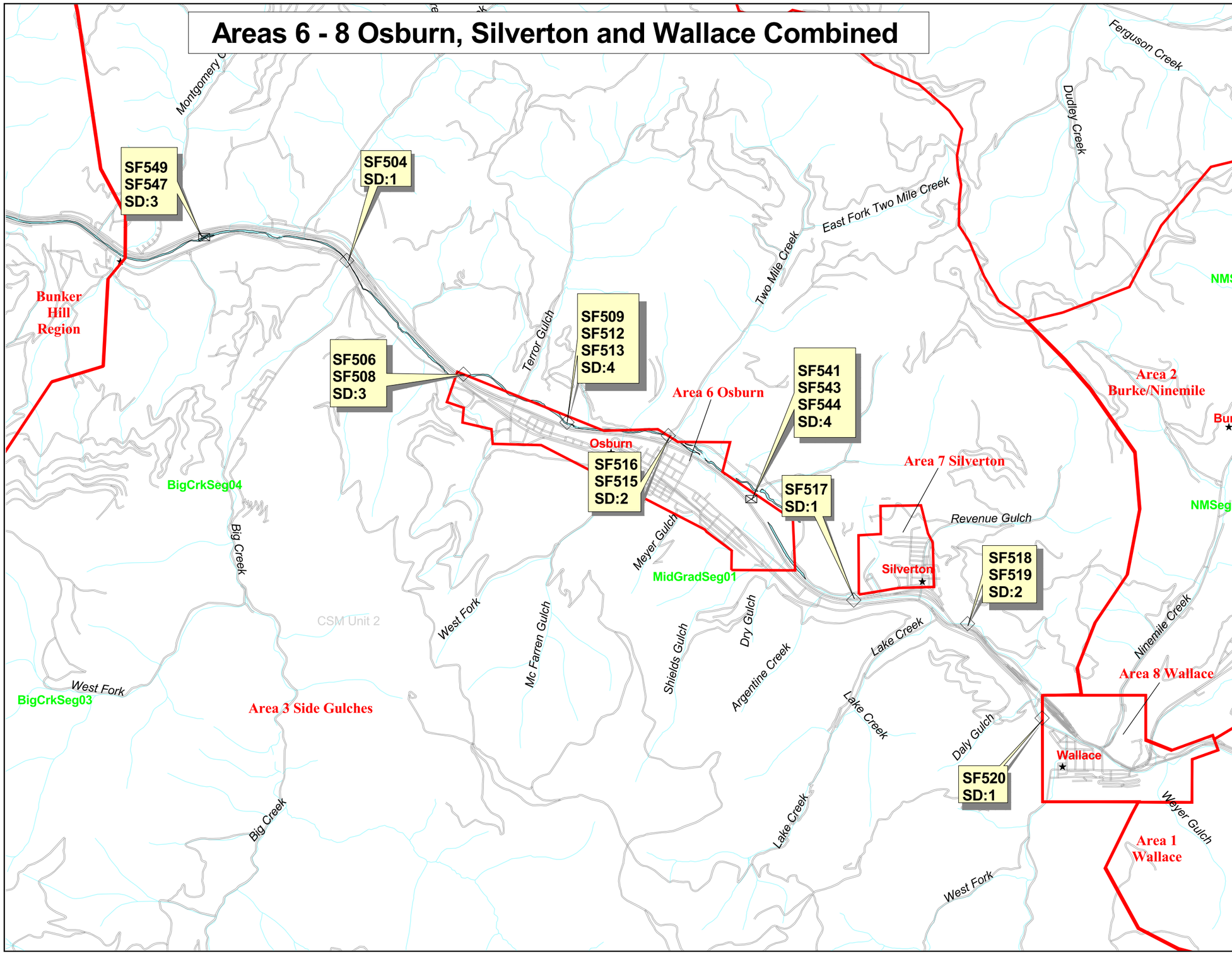
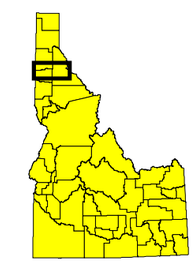


Figure 3-19
Baseline Risk Assessment for Osburn, Silverton and Wallace Areas Combined
Soil/Sediment Sampling Locations

LEGEND

Site Number
Sample Type: # of Samples
SD (Sediment)

- Test Pit Sampling Location
- Hand Auger Sampling Location
- Stream
- Road
- City
- Basin Study Regions
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 03/29/00.

SCALE 1:48,000

0 0.5 1 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\baseline_risk_for_terragraphics.apr
V: OSW SD
E:
L: OSW SD
3/6/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

Areas 6 - 8 Osburn, Silverton and Wallace Combined

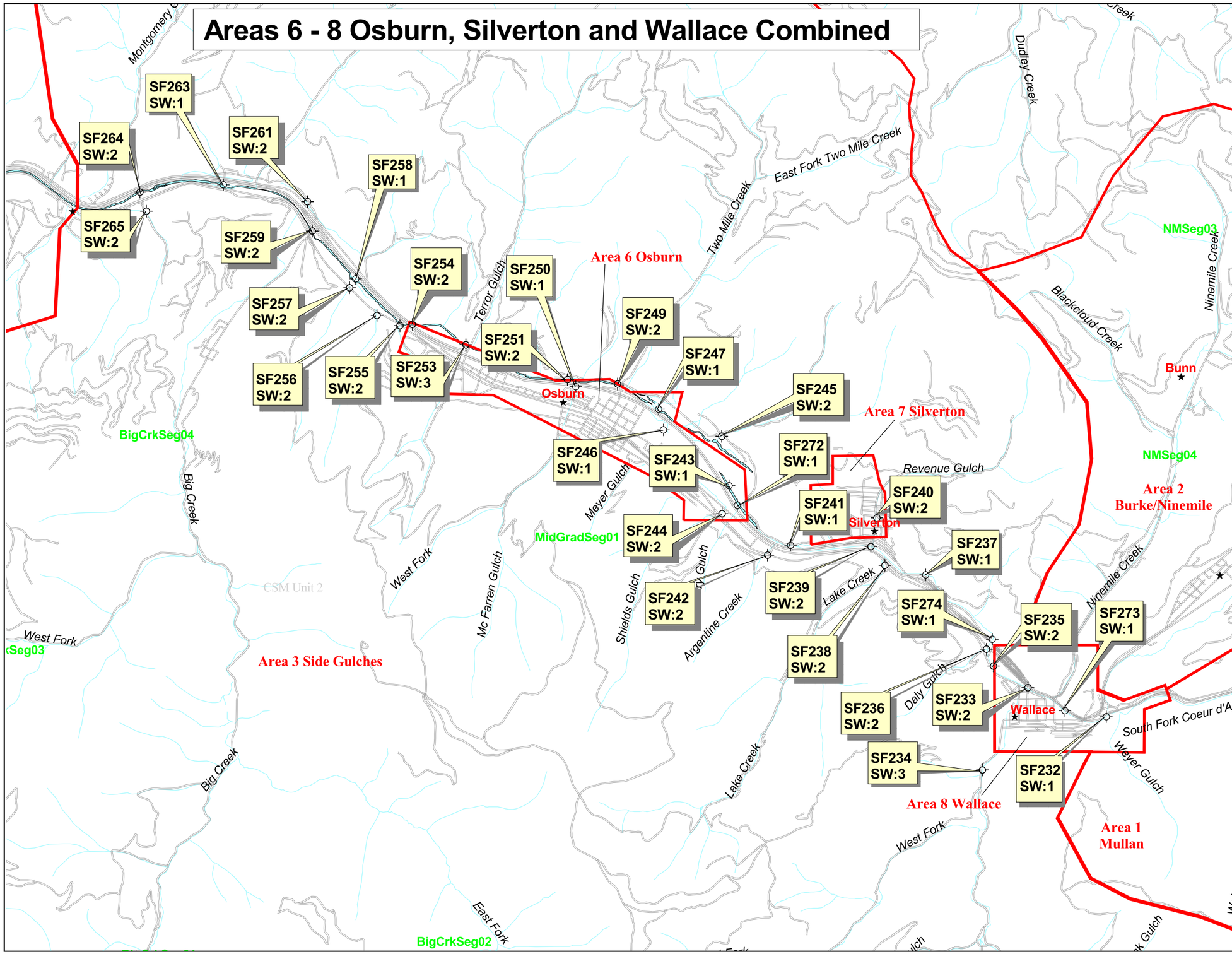
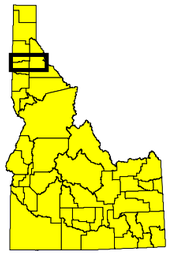


Figure 3-20
Baseline Risk Assessment for Osburn, Silverton and Wallace Areas Combined Surface Water Sampling Locations

LEGEND

Site Number
Sample Type: # of Samples
SW (Surface Water)

- River/Stream Sampling Location
- Stream
- Road
- City
- CSM Unit Boundary
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 03/29/00.

SCALE 1:48,000

0 0.5 1 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

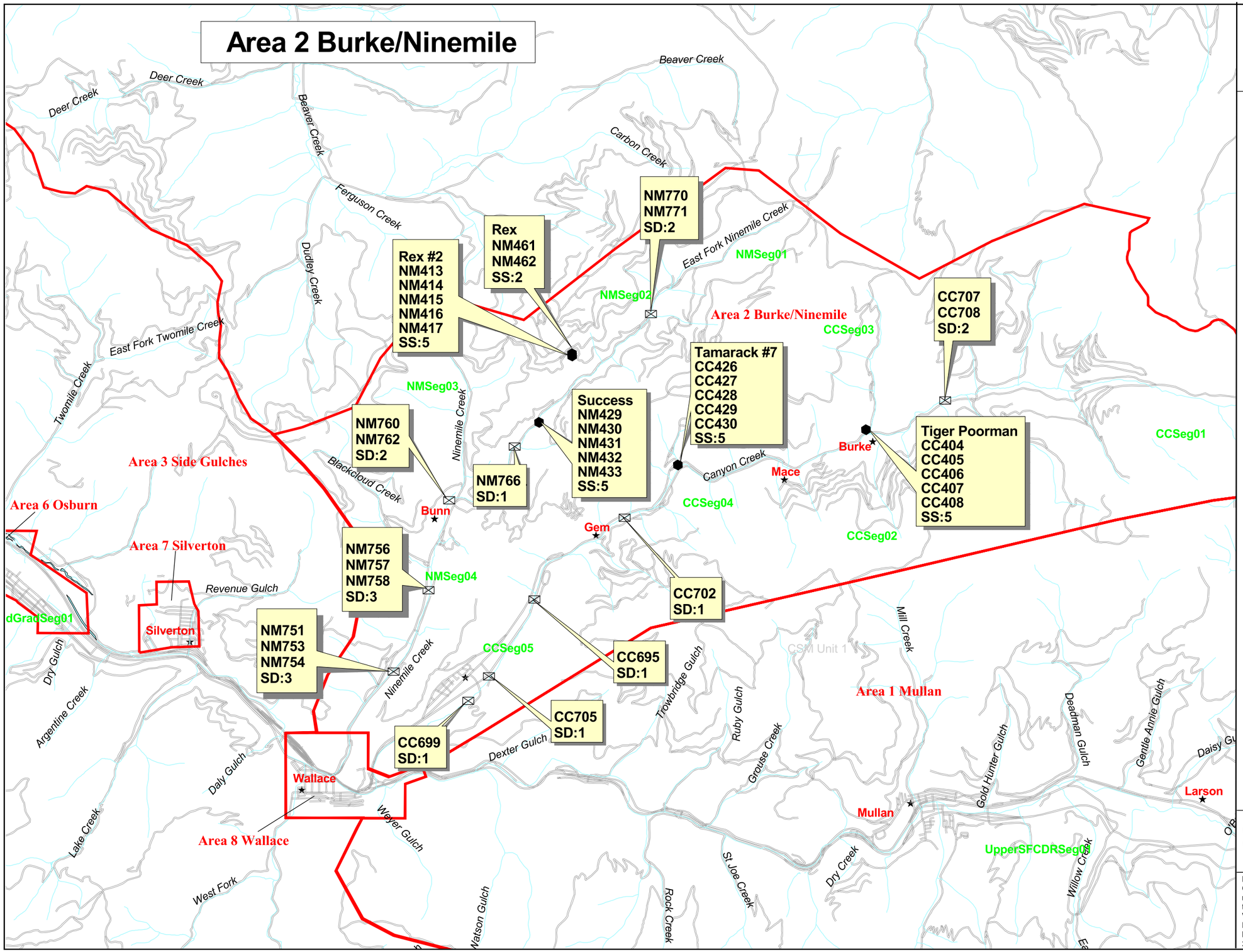
Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\baseline_risk_for_terragraphics.apr
V: OSW SW
E:Surface Water
L: OSW SW
3/06/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

Area 2 Burke/Ninemile

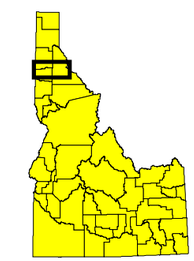
Figure 3-21
Baseline Risk Assessment for
Area 2 Burke/Ninemile
Soil/Sediment Sampling Locations



LEGEND

Site Name (Only waste piles are named)
Site Number
Sample Type: # of Samples
SD (Sediment)
SS (Surface Soil)

- Soil Sampling Location
- Test Pit Sampling Location
- Stream
- Road
- City
- Basin Study Regions
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 03/29/00.

SCALE 1:60,000

0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

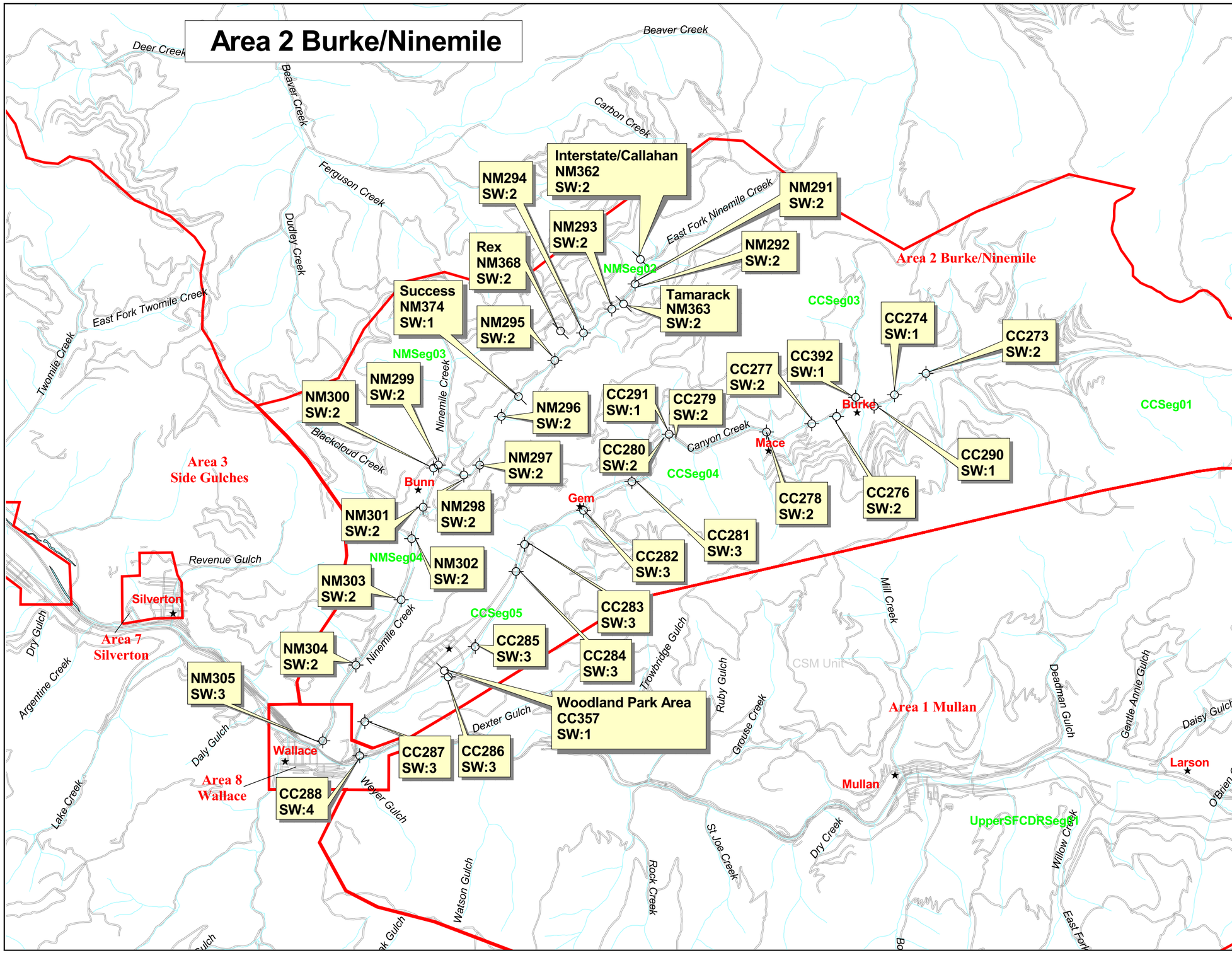
Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\basin_risk_for_terragraphics.apr
VIEW: Ninemile Soil
EXTENT:Ninemile Soil
LAYOUT: Ninemile Soil
3/06/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

Area 2 Burke/Ninemile

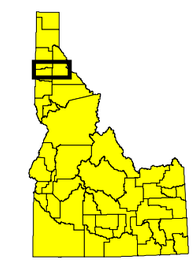
Figure 3-22
Baseline Risk Assessment for
Area 2 Burke/Ninemile
Surface Water Sampling Locations



LEGEND

Site Name (Only seeps are named)
Site Number
Sample Type: # of Samples
SW (Surface Water)

- River/Stream Sampling Location
- Seep Sampling Location
- Stream
- Road
- City
- Basin Study Regions
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 03/29/00.

SCALE 1:60,000
0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

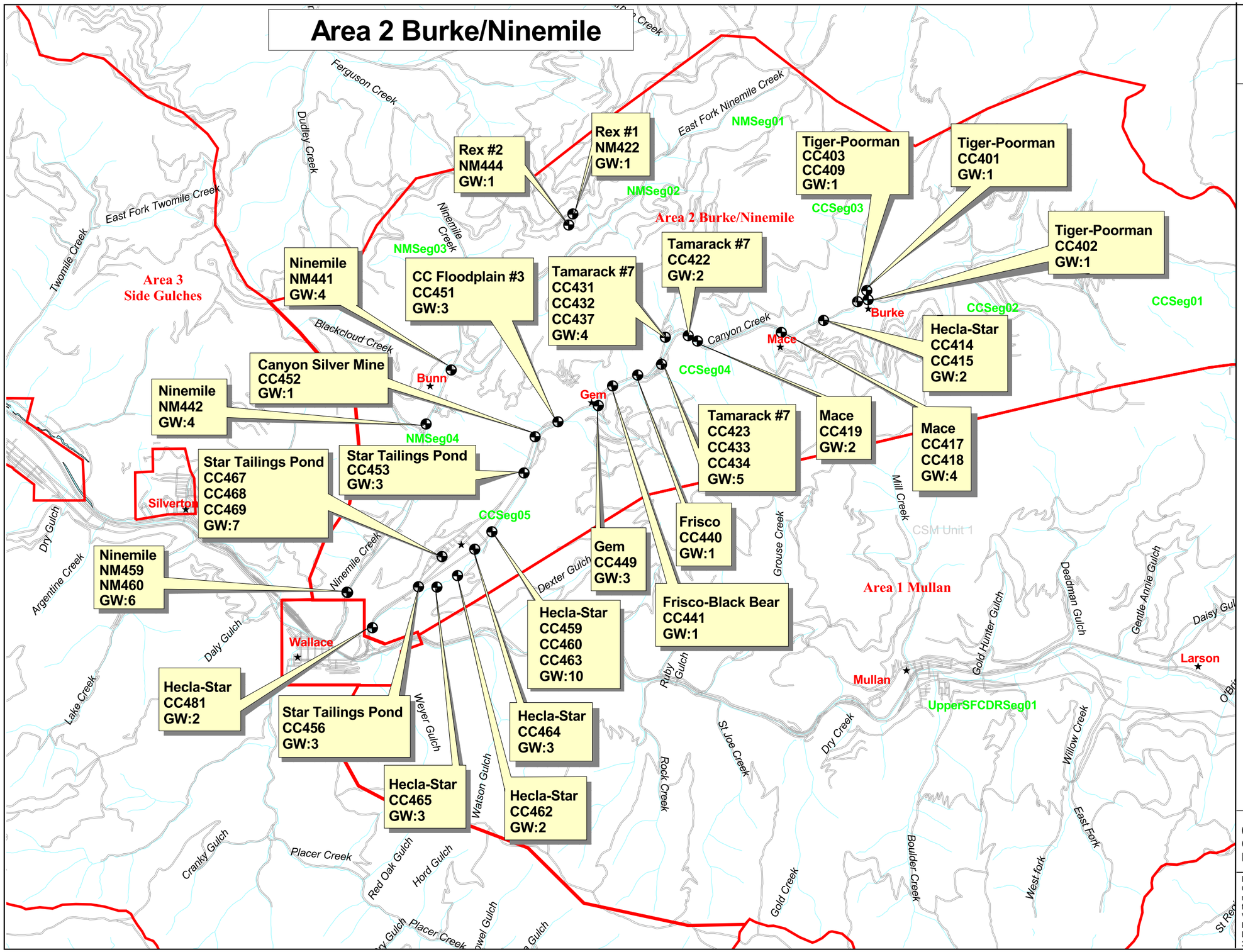
Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\basin_risk_for_terragraphics.apr
VIEW: Ninemile SW
EXTENT:Ninemile SW
LAYOUT: Ninemile SW
3/06/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

Area 2 Burke/Ninemile

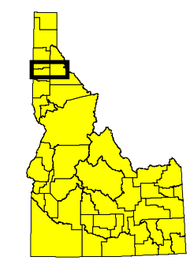
Figure 3-23
Baseline Risk Assessment for
Area 2 Burke/Ninemile
Groundwater Sampling Locations



LEGEND

Site Name
Site Number
Sample Type: # of Samples
GW (Groundwater)

- Monitor Well Sampling Location
- Stream
- Road
- City
- River Segment
- Basin Study Regions
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 03/29/00.

SCALE 1:60,000

0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\basin_risk_for_terragraphics.apr
VIEW: Ninemile Groundwater
EXTENT:Ninemile
LAYOUT: Ninemile Groundwater
3/06/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

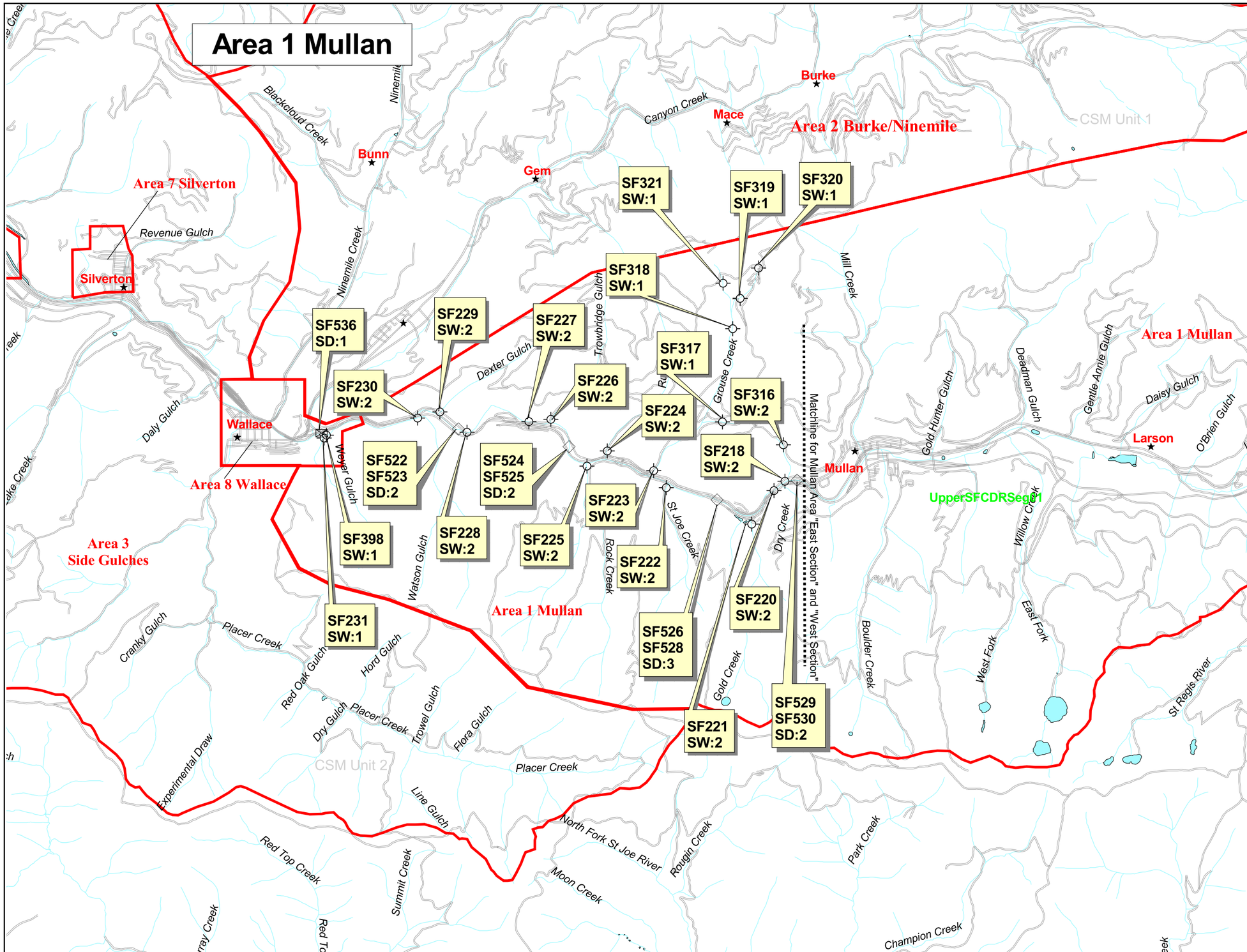
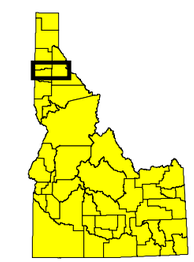


Figure 3-24
Baseline Risk Assessment for Area 1 Mullan "West Section"

LEGEND

Site Number
Sample Type: # of Samples
SD (Sediment)
SW (Surface Water)

- Seep Sampling Location
- River/Stream Sampling Location
- Test Pit Sampling Location
- Hand Auger Sampling Location
- Stream
- Road
- City
- Basin Study Regions
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 03/29/00.

SCALE 1:60,000
0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\basin_risk_for_terragraphics.apr
VIEW: Mullan
EXTENT:Mullan West
LAYOUT: Mullan
3/06/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

Area 1 Mullan

Burke
Gem
Area 2 Burke/Ninemile
Area 1 Mullan
Area 3
Side Gulches
Mullan
Larson
CSM Unit 1
Matchline for Mullan Area "East Section" and "West Section"
UpperSFCDRSeg01

Mullan Waste Pile
SF401
SF402
SF403
SF404
SF405
SS:5

SF216 SW:1
SF275 SW:1
SF531 SD:1
SF209 SW:3
SF206 SW:2
SF205 SW:2
SF539 SD:1
SF210 SW:2
SF211 SW:2
SF207 SW:2
SF213 SW:2
SF215 SW:2
SF214 SW:2
SF212 SW:2
SF208 SD:2 SW:2
SF204 SW:2
SF202 SW:2
SF201 SW:2

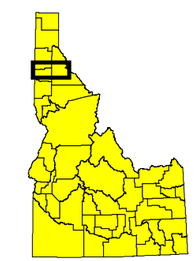
Dexter Gulch
Trowbridge Gulch
Ruby Gulch
Grouse Creek
Mill Creek
Gold Hunter Gulch
Deadman Gulch
Gentle Annie Gulch
Daisy Gulch
O'Brien Gulch
Little North Fork
South Fork Coeur d'Alene River
Summit Creek
Chipmunk Creek Chipmonk Creek
Spokane Creek
Spokane C
Dry Creek
St Joe Creek
Rock Creek
Willow Creek
East Fork
Boulder Creek
West fork
Gold Creek
Flora Gulch
Placer Creek
North Fork St Joe River
Rougin Creek
Moon Creek
Park Creek
Champion Creek
Callahan Creek
Creek
Wonderful Creek
Bullion Creek
St Regis River
Copper Gulch
Borax Creek
Haun Creek
Brimstone Creek
Chippy creek
Shamrock Gulch
Hanaker Creek
Denna Mora Creek
Hilda Creek
Rainy Cr

Figure 3-25
Baseline Risk Assessment for Area 1 Mullan "East Section"

LEGEND

Site Name (Only waste piles are named)
Site Number
Sample Type: # of Samples
SD (Sediment)
SS (Surface Soil)
SW (Surface Water)

- Soil Sampling Location
- River/Stream Sampling Location
- Test Pit Sampling Location
- Hand Auger Sampling Location
- Stream
- Road
- City
- Basin Study Regions
- Lake/River/Floodplain



Location Map

NOTES

1) Base map coverages obtained from the Coeur d'Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner, Inc. Technical Data Management database as of 03/29/00.

SCALE 1:60,000

0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

Doc Control 4162500.5791.04.L
Generation 1
n:\Projects\CUAs\baseline_risk_for_terragraphics.apr
VIEW: Mullan East
EXTENT:Mullan East
LAYOUT: Mullan East
3/06/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

Blackwell Island Area

Mikelson Canal
Hy Line Ditch
Windy Creek
Harrison Creek
SpokaneRSeg01
Spokane River
Blackwell Island
CUA021
SD:14
SS:7
SW:7
CSM Unit 5
Cedar Creek
Coeur d'Alene
Fernan Lake
CDALakeSeg02
Coeur d'Alene Lake
Cougar Creek
North Fork
Kid Creek
CSM Unit 4
Area 5
Lower Basin
CDALakeSeg03

Figure 3-26
Baseline Risk Assessment for Blackwell Island Area

LEGEND

Site Name
Site Number
Sample Type: # of Samples
SD (Sediment)
SS (Surface Soil)
SW (Surface Water)

- Common Use Area Sampling Location
- Stream
- Road
- City
- CSM Unit Boundary
- River/Floodplain/Lake

Location Map

NOTES

1) Base map coverages obtained from the Coeur d' Alene Tribe, URS Greiner, Inc., CH2M HILL, and the Bureau of Land Management.

2) Sampling locations obtained from URS Greiner Inc. Technical Data Management database as of 03/29/00.

SCALE 1:60,000

0.5 0 0.5 Miles

N

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

EPA REGION 10

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VIEW: SpknR\CDA I
EXTENT:Blackwell Island
LAYOUT: Blackwell Island
3/06/2001

This map is based on Idaho State Plane Coordinates West Zone, North American Datum 1983.

Date of Plot: MARCH 6, 2001

Table 3-1a Summary of Basin Geographic Areas and Population (Source: 1990 Census Data)

	Total Area (mi^2)	Portion of Area in Basin (mi^2)	Basin %[1]	Total Population	Portion of Pop in Basin	Basin %[1]
Counties						
Benewah County	782	22	3%	7,937	--	--
Shoshone County	2,628	427	51%	13,931	7,683	73%
Kootenai County	1,312	386	46%	69,795	2,813	27%
Total	**4,722**	**835**	**100%**	**91,663**	**10,496**	**100%**
Basin Study Areas						
Burke/Nine Mile	33	--	4%	676	--	6%
Kingston	293	--	35%	2,849	--	27%
Lower Basin	362	--	43%	2,112	--	20%
Mullan	50	--	6%	985	--	9%
Osburn	1.3	--	0.2%	1,579	--	15%
Side Gulches[2]	95	--	11%	1,285	--	12%
Silverton	0.3	--	0.04%	--	--	--
Wallace	0.9	--	0.1%	1,010	--	10%
Total	835	100%	100%	10,496	100%	100%

[1]The percentage each county makes up of the Basin Area

[2]Silverton population estimate is included in the Side Gulches Study Area population estimate

Table 3-1b Survey Sub-Areas Included within Proposed HHRA Geographic Sub-Divisions

Proposed Geographic Subdivisions	Proposed Area Name	Areas included in IDHW/ATSDR Database	Areas included in USEPA FSPA06/07 Database	Areas included in USEPA FSPA12 Database
Mullan	Mullan	Mullan	Mullan	Mullan
Burke/Nine Mile	Nine Mile	Burke, Nine Mile	Burke, Gem, Black Cloud, Woodland Park	Burke, Black Cloud, Woodland Park
Wallace	Wallace	Wallace	Wallace	Wallace
Silverton	Silverton	Silverton	Silverton	Silverton
Osburn	Osburn	Osburn	Osburn	Osburn
Side Gulches	Side Gulches	Big Creek, Elk Creek, Montgomery Gulch, Moon Gulch, Nuckols Gulch, Sunny Slopes, Terror Gulch, Two Mile	Elk Creek, Moon Gulch, Nuckols Gulch, Terror Gulch	Nuckols Gulch
Kingston	Kingston	Kingston, Pine Creek	Kingston, Pinehurst	Kingston
Lower Basin/Cataldo	Lower Basin	Lower Basin	Cataldo	Cataldo

Table 3-2 Basin Demographics Over Time (Source: County Profiles of Idaho, IDOC, and 1990 Census)

	1970	1980	Census 1990	County Profile 1990	1996	1997	1998
Population							
Shoshone County	19,718	19,226	13,931	13,931	--	13,982	13,870
Mullan	1,279	1,269	821	821	795	--	--
Osburn	2,248	2,220	1,579	1,579	1,593	--	--
Wallace	2,206	1,736	1,010	1,010	954	--	918
Idaho	--	--	1,006,749	1,006,749	--	--	1,210,232
Shoshone County							
Median Age	27.3	28.8	--	37.3	--	--	--
65+ Years (%)	7.1	10.3	--	16.7	--	--	--
Total Employment	--	9,126	5,310	6,422	6,663	--	--
Mining Employment	--	2,465	1,282	1,542	642	--	--
% Labor Force Unemployed	--	6.7	--	9.9	--	--	10.2

Table 3-3 Census Block Groups Falling Partially Outside of Basin Study Area Boundaries (Source: 1990 Census)

Block Group Key ID	Basin Area	County	Total Area (mi^2)	Portion of Area in Basin (mi^2)	Basin %[1]	Population Number	Population Basin %[1]
16.055.0019.1	Kingston	Kootenai	264	80.9	10%	701	6.7%
16.079.9601.1	Kingston	Shoshone	228	122.9	15%	162	1.5%
16.079.9601.2	Kingston	Shoshone	465	49.2	6%	257	2.4%
Block Group Total			957	253	**30%**	1,120	**10.7%**
Basin Area Total			835	835	100%	10,496	100%

[1]The percentage each block group makes up of the Basin Area

Table 3-4 Summary Population Characteristics

	Basin Areas		State of Idaho	
	Number	**Percent**	**Number**	**Percent**
Total Population[1]	10,496	1.0%	1,006,749	
Total Family Households[1]	3,071	1.2%	265,597	
Total Households[1]	4,215	1.2%	361,432	
Sex and Ethnicity				
Male	5,330	51%	501,548	50%
Female	5,166	49%	505,201	50%
White	10,331	98%	950,802	94%
Black	6	0.1%	3,653	0.4%
American Indian	100	1.0%	14,677	1.5%
Asian and Pacific	10	0.1%	9,096	0.9%
Other Ethnicity	49	0.5%	28,521	2.8%
Hispanics of All Races	219	2.1%	51,679	5.1%
Ages				
Age 0 to 4	587	5.6%	80,046	8.0%
Age 5 to 14	1,594	15.2%	180,265	17.9%
Age 15 to 24	1,145	10.9%	145,146	14.4%
Age 25 to 34	1,400	13.3%	154,087	15.3%
Age 35 to 44	1,655	15.8%	149,338	14.8%
Age 45 to 54	1,347	12.8%	98,910	9.8%
Age 55 to 64	1,102	10.5%	77,819	7.7%
Age over 65	1,666	15.9%	121,138	12.0%

[1]Percent of Idaho Total

Table 3-5 Household Characteristics (Source: 1990 Census)

	Basin Areas		State of Idaho	
	Number	**Percent**	**Number**	**Percent**
Total Households[1]	4,215	1.2%	361,432	
Family Households	3,071	73%	265,597	73%
Non-Family Households	1,144	27%	95,835	27%
Family/Household Type				
One Person Households	1,028	24%	80,364	22%
Married with Child Families	1,117	27%	113,964	32%
Married no Child Families	1,552	37%	113,901	32%
Single with Child Families	242	6%	24,359	7%
Single no Child Families	160	4%	13,373	4%

[1]Percent of Idaho Total

Table 3-6 Housing Characteristics (Source: 1990 Census)

	Basin Areas/ Block Groups		State of Idaho	
	Number	Percent	Number	Percent
Total Housing Units[1]	5,651	1.4%	413,327	
Occupied Housing Units	4,205	74%	360,723	87%
Owner Occupied Units	3,251	77%	252,687	70%
Renter Occupied Units	954	23%	108,036	30%
Moved in 1980+	2,286	54%	235,825	65%
Moved in 1960-1979	1,409	34%	97,345	27%
Moved in Before 1960	456	11%	26,218	7%
One Unit	4,132	73%	294,458	71%
2-9 Units	264	5%	41,497	10%
10+ Units	112	2%	17,412	4%
Mobile/Trailer Home	1,053	19%	56,625	14%
Built before 1940	1,628	29%	65,682	16%
Built 1940-1959	1,071	19%	85,130	21%
Built 1960-1979	2,269	40%	188,061	45%
Built after 1980	683	12%	74,454	18%

[1]Percent of Idaho Total

Table 3-7 Comparison of Median Values of Housing (Source: 1990 Census)

Basin Area	Census Block Group Key ID	n	Median Year Built		Median Value of Units		Median Rent	
			Basin Areas	State of Idaho	Basin Areas	State of Idaho	Basin Areas	State of Idaho
Burke/Nine Mile	16.079.9601 .3	333	1950	1970	$ 18,600	$ 58,000	$ 275	$ 330
Kingston	16.055.0019 .1	269	1965		$ 60,900		$ 280	
	16.079.9601 .1	183	1972		$ 67,500		$ 375	
	16.079.9601 .2	186	1968		$ 29,400		$ 215	
	16.079.9602 .1	161	1966		$ 42,100		$ 221	
	16.079.9602 .2	197	1970		$ 26,100		$ 269	
	16.079.9602 .5	304	1967		$ 40,500		$ 247	
Lower Basin	16.055.0019 .2	567	1977		$ 111,700		$ 245	
	16.055.0019 .3	631	1972		$ 45,400		$ 307	
	16.055.0019 .4	444	1971		$ 58,800		$ 288	
Mullan	16.079.9605 .1	317	1939		$ 21,900		$ 223	
	16.079.9605 .2	188	1939		$ 22,600		$ 189	
Side Gulches	16.079.9601 .4	354	1954		$ 41,600		$ 310	
	16.079.9601 .5	153	1972		$ 31,500		$ 294	
	16.079.9604 .1	306	1959		$ 27,100		$ 258	
	16.079.9604 .2	459	1957		$ 37,200		$ 285	
	16.079.9604 .4	300	1939		$ 32,900		$ 247	
	16.079.9604 .3	299	1939		$ 24,300		$ 201	
Wallace[1]	NA	597	1939		$ 28,200		$ 221	
Osburn[1]	NA	744	1958		$ 35,300		$ 272	
Silverton[1]	NA	--	--		--		--	
# of Block Groups less than State Median			12	67%	14	78%	17	94%

[1]None of the census block groups lie entirely or mostly within the City study group areas (Figure1). Block groups partially within the City study groups are included in the Side Gulches Study Area.

Table 3-8 Student Population and Educational Attainment (Source: 1990 Census)

	Basin Areas/ Block Groups		State of Idaho	
	Number	Percent	Number	Percent
Preprimary Students	115	5%	18,745	6%
Elementary and High School Students	1,979	82%	210,095	71%
College Students	322	13%	66,798	23%
Total Student Population	2,416	100%	295,638	100%
Population age 25+	7,170		601,292	
No High School Diploma	1,949	27%	121,787	20%
High School Graduates	5,221	73%	479,505	80%
Attended College	1,386	19%	145,291	24%
College Graduates	1,097	15%	151,322	25%
Degree, Masters+	269	4%	31,692	5%

Table 3-9 Basin Area Household Income (Source: 1990 Census)

	Block Group Key ID	N	Basin Areas/ Block Groups		State of Idaho	
			Number	Percent	Number	Percent
Total Number of Households			4,215		361,432	
Income Under $15,000			1,343	32%	98,404	27%
Income $15-25,000			920	22%	80,355	22%
Income $25-35,000			774	18%	65,633	18%
Income $35-50,000			675	16%	61,608	17%
Income $50-75,000			357	8%	38,506	11%
Income over $75,000			146	3%	16,926	5%
Children in Poverty (under age 18)			577	15%	49,159	14%
People in Poverty (all ages)			1,517	10%	130,588	13%
Median Household Income					$ 25,257	
Burke/Nine Mile	16.079.9601 .3	282	$ 22,875			
Kingston	16.055.0019 .1	245	$ 28,029			
	16.079.9601 .1	71	$ 17,159			
	16.079.9601 .2	92	$ 17,500			
	16.079.9602 .1	158	$ 24,531			
	16.079.9602 .2	178	$ 19,000			
	16.079.9602 .5	292	$ 26,176			
Lower Basin	16.055.0019 .2	213	$ 31,484			
	16.055.0019 .3	391	$ 21,964			
	16.055.0019 .4	243	$ 21,726			
Mullan	16.079.9605 .1	238	$ 22,895			
	16.079.9605 .2	168	$ 24,444			
Side Gulches	16.079.9601 .4	339	$ 26,607			
	16.079.9601 .5	143	$ 23,750			
	16.079.9604 .1	287	$ 18,650			
	16.079.9604 .2	404	$ 26,719			
	16.079.9604 .4	228	$ 23,650			
	16.079.9604 .3	243	$ 15,500			
Wallace	NA		$ 19,750			
Osburn	NA		$ 21,604			
Silverton[1]	NA		--			
# of Groups less than State Median			13	72%		

[1]None of the census block groups lie entirely or mostly within the City study group areas (Figure1). Block groups partially within the City study groups are included in the Side Gulches Study Area.

Table 3-10 Shoshone County Profile (Source: Profile of Rural Idaho, IDOC)

	Shoshone County	State of Idaho	State of Idaho	
			Urban	Rural
Economic Indicators				
Unemployment Rate 1997	12.8%	5.3%	4.8%	6.8%
1990-96 Business Growth				
Total	17.6%	29.6%	30.9%	26.9%
Retail	6.3%	19.2%	20.8%	16.1%
Housing Growth 1990-97	5.6%	21.6%	27.1%	13.6%
Income and Poverty				
Per Capita Personal Income 1996	$16,938	$19,865	$21,773	$16,513
Median Household Income 1995	$24,541	$32,003	$34,946	$30,075
Percent Persons Below Poverty 1995				
All Ages	21.4%	12.6%	11.2%	14.5%
Under 18	31.2%	16.5%	15.6%	18.8%
Over 18	17.9%	10.7%	9.5%	12.7%
Welfare Payments Per Capita 1998	$771	$378	$452	$248
Housing				
Percent Housing Units Built				
Before 1939	34.4%	13.1%	10.6%	17.1%
Since 1970	32.5%	59.3%	61.4%	55.7%

Table 3-11 Kids Count Data as Presented in Yearly Reports (Source: Idaho KIDS COUNT: Profiles of Child Well-Being 1996-2000)

	Shoshone County (Report Year)				State of Idaho (Report Year)			
	1996	1997	1998	1999-2000	1996	1997	1998	1999-2000
Total Population [1]	13,871	14,024	13,982	13,870	1,133,054	1,189,251	1,210,232	1,228,684
Child Population Under 18 [1]	3,530	3,521	3,480	3,313	339,478	348,509	351,352	351,158
Children Under 18 in Poverty [2]	23.7	23.7	27.8	31.2	16.2	16.2	15.5	16.5
Single-Parent Families with Children Under Age 18 [3]	21.3	23.7	23.7	23.7	14.7	17.6	17.6	17.6
Infant Mortality [4]	9.1	9.4	7.9	NR	6.7	6.8	6.8	7.2
Low Birth Weight Babies (% of births) [4]	7.1	6.9	6.7	7.1	5.6	5.7	6.0	6.0
Babies Born to Mothers without Adequate Prenatal Care (% of births) [4]	23.3	25.0	25.3	34.9	28.1	28.6	29.1	30.3
Babies Born to Teen Mothers without Adequate Prenatal Care (% of teen births) [4]	0.0	0.9	1.9	42.1	1.3	1.3	1.4	39.8
Child Deaths Ages 1-14 (annual rate per 100,000 children) [5]	25.9	25.9	39.6	NR	32.8	31.8	33.8	32.4
School Dropouts Ages 16-19 [3]	14.6	14.6	14.6	14.6	10.4	10.4	10.4	10.4
Teen Births Ages 15-17 (annualized rate per 1,000 females ages 15-17) [4]	31.6	27.2	25.4	22.3	28.2	27.5	25.6	24.7
Juvenile Violent Crime Arrests Ages 10-17 (annualized rate per 1,000 youths) [4]	3.2	2.6	1.9	3.5	2.7	2.5	2.3	2.2
Teens Not in school and Not Working Ages 16-19 [3]	7.7	14.6	14.6	14.6	5.3	8.0	8.0	8.0
Teen Violent Deaths Ages 15-19 (annualized rate per 100,000 teens) [5]	90.9	144.9	172.3	140.0	76.9	76.6	74.9	73.6

1 1996 report presented1994 data, 1997 report presented1996 data, 1998 report presented1997 data, 1999-2000 report presented1998 data

2 1996 and 1997 reports presented1990 data, 1998 report presented1994 data, 1999-2000 report presented1996 data

3 All reports presented1990 data

4 1996 report presented1993-1995 data, 1997 report presented1994-1996 data, 1998 report presented1995-1997 data, 1999-2000 report presented1996-1998 data

5 1996 report presented1993-1995 data, 1997 report presented1994-1996 data, 1998 report presented1995-1997 data, 1999-2000 report presented1994-1998 data

Table 3-12 1999-2000 Kids Count Data - Economic Well Being (Source: Idaho KIDS COUNT: Profiles of Child Well-Being 1999-2000 and School District Data)

	Shoshone County		State of Idaho	
	Total	Percent	Total	Percent
Kids Count				
Elementary school children receiving free or reduced price lunch (1997-98)	415	50.1%	48,059	40.9%
Elementary school children receiving free or reduced price lunch (1998-99)	443	54.0%	46,947	42.0%
School District				
All school children (District #391) receiving free or reduced price lunch (1999-2000)	642	44.0%	--	--
All school children (District #393) receiving free or reduced price lunch (1999-2000)	283	40.0%	--	--
Births paid for by Medicaid (1997)	92	55.4%	6,046	32.6%
Births paid for by Medicaid (1998)	69	41.6%	5,419	28.0%

Table 3-13 1999-2000 Kids Count Data - Child Population Change
(Source: Idaho KIDS COUNT: Profiles of Child Well-Being 1999-2000)

	Shoshone County			State of Idaho		
	1990	1998	% Change	1990	1998	% Change
Children under age 5	815	716	-12.1%	81,546	91,467	12.2%
Children age 5-17	2,814	2,597	-7.7%	227,848	259,691	14.0%
Total Under 18	3,629	3,313	-8.7%	309,394	351,158	13.5%

Table 3-14 School District Data (Source: School Districts #391, 392, and 393)

School District	Kellogg # 391	Kellogg w/o BHSS	Mullan # 392	Wallace/Osburn # 393	Total	Total w/o BHSS
Total Student Population (K-12)						
1990	1,779	569		877		
1991	1,701	544		887		
1992	1,649	528	222	844	2,715	1594
1993	1,574	504	213	844	2,631	1561
1994	1,566	501	215	839	2,620	1555
1995	1,556	498	216	857	2,629	1571
1996	1,571	503	218	818	2,607	1539
1997	1,573	503	180	787	2,540	1470
1998	1,490	477	176	788	2,454	1441
1999	1,422	455	183	741	2,346	1379
2000	1,458	467	185	709	2,352	1361
Decrease in School District Enrollment (1992-2000)					363	233
					13%	15%

Table 3-15 School District Enrollment by Grade
(Source: School Districts #391, 392, and 393)

School District	Kellogg # 391	Mullan # 392	Wallace/Osburn # 393	Total	%
Kindergarten	116	9	54	179	8%
1st Grade	110	15	46	171	8%
2nd Grade	114	8	45	167	8%
3rd Grade	124	14	51	189	9%
4th Grade	115	12	39	166	8%
5th Grade	122	19	54	195	9%
6th Grade	111	15	45	171	8%
7th Grade	117		50	167	8%
8th Grade	119		45	164	7%
9th Grade	113		51	164	7%
10th Grade	97		58	155	7%
11th Grade	116		52	168	8%
12th Grade	89		56	145	7%
Total				2201	100%

Table 3-16 Estimated Child Population and Sample Population (Sources: 1990 Census, Idaho KIDS COUNT, and School District Data)

Geographic Area	Source	Year	Total Population Age Range	Total Population Years	Total Population Number[1,2]	Population per each year of age	Sample Population Estimate (*9.33)
Basin Area	1990 Census	1990	0-17	18	2,484	138	1,288
Basin Area	School District	1992	5-18	13	1,673	129	1,201
		1993	5-18	13	1,639	126	1,176
		1994	5-18	13	1,633	126	1,172
		1995	5-18	13	1,649	127	1,184
		1996	5-18	13	1,616	124	1,160
		1997	5-18	13	1,544	119	1,108
		1998	5-18	13	1,513	116	1,086
		1999	5-18	13	1,448	111	1,039
		2000	5-18	13	1,429	110	1,025
Shoshone County	1990 Census	1990	0-17	18	2,384	132	1,236
	Kids Count	1990	0-17	18	2,416	134	1,252
		1994	0-17	18	2,462	137	1,276
		1996	0-17	18	2,449	136	1,269
		1997	0-17	18	2,407	134	1,248
		1998	0-17	18	2,297	128	1,191

[1]Adjusted to omit the student population within the Bunker Hill Superfund Site

[2]School district data was increased by 5% to include students enrolled in private school or home schooling

Table 3-17 Public and Private Sewer and Water Hook-ups (Source: 1990 Census and 1999 Sewer District (SD) data)

	Basin Areas (Census)[1]		Basin Area w/o Lower Basin (Census)[1]		Basin Area w/o Lower Basin (SD-1999)[1]	
	Number	**Percent**	**Number**	**Percent**	**Number**	**Percent**
Total Housing Units	5,651		4,009		*4,257*	
Estimated # of Yards	5,255		3,728		*3,570*	
Water from a Public Source	3,209	57%	2,850	71%	--	--
Water from a Private Source	2,442	43%	1159	29%	--	--
Sewer with a Public Utility	3,035	54%	2,873	72%	3,065	*72%*
Sewer by Private Means	2,616	46%	1136	28%	*1,192*	*28%*

[1]Adjusted to omit BHSS Housing Units

Table 3-18 Estimated Number of Housing Units by Basin Area (Source: 1990 Census and 1999 Sewer District Data)

Basin Area	Housing Units 1990 Census	Sewer District 1999 Sewer Hook-Ups[3]	Housing Units 1999 Estimate[2,3]	Est. # of Yards[3]
Burke/Ninemile	333	191	265	245
Kingston	1,300	724	1006	1006
Lower Basin	1,642	--	--	--
Mullan	505	398	553	548
Side Gulches[1]	530	461	640	624
Wallace	597	552	767	649
Osburn	744	739	1026	847
Silverton	--	271	376	360
Total	5,651	3,065	4,257	3,919
Total w/o Lower Basin	4,009	3,065	4,257	3,919

[1]Includes the housing units in Silverton

[2]Determined by assuming 72% of all housing units in Shoshone County have sewer with a public utility

[3]Lower Basin not included

Table 3-19a
Summary of Exposure Pathways Quantified in HHRA

Geographical Area	Timeframe	Scenario	Receptor	Receptor Age (years)	Exposure Medium	Exposure Point	Exposure Route
Lower Basin	Current and future	Residential[a]	Resident	Child 0 - 6	Groundwater	Tap water	Ingestion
				Child 0 - 6/adult 7 - 30	Groundwater	Tap water	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Dermal
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Dermal
	Current and future	Neighborhood recreational	Neighborhood resident	Child 4 - 11	Disturbed surface water	Lower Coeur d'Alene River	Ingestion
				Child 4 - 11	Soil/sediment	Lower Coeur d'Alene River	Ingestion
				Child 4 - 11	Soil/sediment	Lower Coeur d'Alene River	Dermal
	Current and future	Public recreational	Visitor	Child 0 - 6	Disturbed surface water	Lower Coeur d'Alene River	Ingestion
				Child 0 - 6/adult 7 - 30	Disturbed surface water	Lower Coeur d'Alene River	Ingestion
				Child 0 - 6	Soil/sediment	Lower Coeur d'Alene River	Ingestion
				Child 0 - 6/adult 7 - 30	Soil/sediment	Lower Coeur d'Alene River	Ingestion
				Child 0 - 6	Soil/sediment	Lower Coeur d'Alene River	Dermal
				Child 0 - 6/adult 7 - 30	Soil/sediment	Lower Coeur d'Alene River	Dermal
	Current and future	Occupational	Construction worker	Adult	Surface/subsurface soil	Construction site soil	Ingestion
				Adult	Surface/subsurface soil	Construction site soil	Dermal
Kingston	Current and future	Residential	Resident	Child 0 - 6	Groundwater	Tap water	Ingestion
				Child 0 - 6/adult 7 - 30	Groundwater	Tap water	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Dermal
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Dermal

Table 3-19a (Continued)
Summary of Exposure Pathways Quantified in HHRA

Geographical Area	Timeframe	Scenario	Receptor	Receptor Age (years)	Exposure Medium	Exposure Point	Exposure Route
Kingston (Continued)	Current and future	Neighborhood recreational	Neighborhood resident	Child 4 - 11	Sediment	Pine Creek	Ingestion
				Child 4 - 11	Sediment	Pine Creek	Dermal
				Child 4 - 11	Soil/sediment	NS confluence	Ingestion
				Child 4 - 11	Soil/sediment	NS confluence	Dermal
				Child 4 - 11	Surface water	Pine Creek	Ingestion
				Child 4 - 11	Surface water	NS confluence	Ingestion
	Current and future	Public recreational	Visitor	Child 0 - 6	Soil/sediment	NS confluence	Ingestion
				Child 0 - 6/adult 7 - 30	Soil/sediment	NS confluence	Ingestion
				Child 0 - 6	Soil/sediment	NS confluence	Dermal
				Child 0 - 6/adult 7 - 30	Soil/sediment	NS confluence	Dermal
				Child 0 - 6	Surface water	NS confluence	Ingestion
				Child 0 - 6/adult 7 - 30	Surface water	NS confluence	Ingestion
	Current and future	Occupational	Construction worker	Adult	Surface/subsurface soil	Construction site soil	Ingestion
				Adult	Surface/subsurface soil	Construction site soil	Dermal
Side Gulches	Current and future	Residential	Resident	Child 0 - 6	Groundwater	Tap water	Ingestion
				Child 0 - 6/adult 7 - 30	Groundwater	Tap water	Ingestion
	Current and future	Residential	Resident	Child 0 - 6	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Dermal
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Dermal

Table 3-19a (Continued)
Summary of Exposure Pathways Quantified in HHRA

Geographical Area	Timeframe	Scenario	Receptor	Receptor Age (years)	Exposure Medium	Exposure Point	Exposure Route
Side Gulches (Continued)	Current and future	Neighborhood recreational	Neighborhood resident	Child 4 - 11	Sediment	Elk Creek Pond	Ingestion
				Child 4 - 11	Sediment	Elk Creek Pond	Dermal
				Child 4 - 11	Surface soil	Elk Creek Area	Ingestion
				Child 4 - 11	Surface soil	Elk Creek Area	Dermal
				Child 4 - 11	Surface water	Elk Creek Pond	Ingestion
Osburn	Current and future	Residential	Resident	Child 0 - 6	Groundwater	Tap water	Ingestion
				Child 0 - 6/adult 7 - 30	Groundwater	Tap water	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Dermal
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Dermal
Silverton	Current and future	Residential	Resident	Child 0 - 6	Groundwater	Tap water	Ingestion
				Child 0 - 6/adult 7 - 30	Groundwater	Tap water	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Dermal
	Current and future	Neighborhood recreational	Neighborhood resident	Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Dermal
				Child 4 - 11	Surface soil	Upland parks/schools	Ingestion
				Child 4 - 11	Surface soil	Upland parks/schools	Dermal

Table 3-19a (Continued)
Summary of Exposure Pathways Quantified in HHRA

Geographical Area	Timeframe	Scenario	Receptor	Receptor Age (years)	Exposure Medium	Exposure Point	Exposure Route
Silverton (Continued)	Current and future	Public recreational	Visitor	Child 0 - 6	Surface soil	Upland parks/schools	Ingestion
				Child 0 - 6/adult 7 - 30	Surface soil	Upland parks/schools	Ingestion
				Child 0 - 6	Surface soil	Upland parks/schools	Dermal
				Child 0 - 6/adult 7 - 30	Surface soil	Upland parks/schools	Dermal
Wallace	Current and future	Residential	Resident	Child 0 - 6	Groundwater	Tap water	Ingestion
				Child 0 - 6/adult 7 - 30	Groundwater	Tap water	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Dermal
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Dermal
	Current and future	Neighborhood recreational	Neighborhood resident	Child 4 - 11	Surface soil	Upland parks/schools	Ingestion
				Child 4 - 11	Surface soil	Upland parks/schools	Dermal
	Current and future	Public recreational	Visitor	Child 0 - 6	Surface soil	Upland parks/schools	Ingestion
				Child 0 - 6/adult 7 - 30	Surface soil	Upland parks/schools	Ingestion
				Child 0 - 6	Surface soil	Upland parks/schools	Dermal
				Child 0 - 6/adult 7 - 30	Surface soil	Upland parks/schools	Dermal
MidGradSeg01[b]	Current and future	Neighborhood recreational[c]	Neighborhood resident	Child 4 - 11	Sediment	South Fork Coeur d'Alene River	Ingestion
				Child 4 - 11	Sediment	South Fork Coeur d'Alene River	Dermal
				Child 4 - 11	Surface water	South Fork Coeur d'Alene River	Ingestion
	Current and future	Occupational	Construction worker	Adult	Surface/subsurface soil	Construction site soil	Ingestion
				Adult	Surface/subsurface soil	Construction site soil	Dermal

Table 3-19a (Continued)
Summary of Exposure Pathways Quantified in HHRA

Geographical Area	Timeframe	Scenario	Receptor	Receptor Age (years)	Exposure Medium	Exposure Point	Exposure Route
Nine Mile	Current and future	Residential	Resident	Child 0 - 6	Groundwater	Tap water	Ingestion
				Child 0 - 6/adult 7 - 30	Groundwater	Tap water	Ingestion
	Future	Residential	Resident	Child 0 - 6	Groundwater	Tap water	Ingestion
				Child 0 - 6/adult 7 - 30	Groundwater	Tap water	Ingestion
	Current and future	Residential	Resident	Child 0 - 6	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Dermal
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Dermal
	Current and future	Neighborhood recreational	Neighborhood resident	Child 4 - 11	Sediment	Ninemile/Canyon Creeks	Ingestion
				Child 4 - 11	Sediment	Ninemile/Canyon Creeks	Dermal
				Child 4 - 11	Surface soil	Waste piles	Ingestion
				Child 4 - 11	Surface soil	Waste piles	Dermal
				Child 4 - 11	Surface water	Ninemile/Canyon Creeks	Ingestion
	Current and future	Occupational	Construction worker	Adult	Surface/subsurface soil	Construction site soil	Ingestion
				Adult	Surface/subsurface soil	Construction site soil	Dermal
Mullan	Current and future	Residential	Resident	Child 0 - 6	Groundwater	Tap water	Ingestion
				Child 0 - 6/adult 7 - 30	Groundwater	Tap water	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Ingestion
				Child 0 - 6	Surface soil	Yard soil/house dust	Dermal
				Child 0 - 6/adult 7 - 30	Surface soil	Yard soil/house dust	Dermal

Table 3-19a (Continued)
Summary of Exposure Pathways Quantified in HHRA

Geographical Area	Timeframe	Scenario	Receptor	Receptor Age (years)	Exposure Medium	Exposure Point	Exposure Route
Mullan (Continued)	Current and future	Neighborhood recreational	Neighborhood resident	Child 4 - 11	Sediment	South Fork Coeur d'Alene River	Ingestion
				Child 4 - 11	Sediment	South Fork Coeur d'Alene River	Dermal
				Child 4 - 11	Surface soil	Waste piles	Ingestion
				Child 4 - 11	Surface soil	Waste piles	Dermal
				Child 4 - 11	Surface water	South Fork Coeur d'Alene River	Ingestion
	Current and future	Occupational	Construction worker	Adult	Surface/subsurface soil	Construction site soil	Ingestion
				Adult	Surface/subsurface soil	Construction site soil	Dermal
Blackwell Island	Current and future	Public recreational	Visitor	Child 0 - 6	Soil/sediment	Spokane River	Ingestion
				Child 0 - 6/adult 7 - 30	Soil/sediment	Spokane River	Ingestion
				Child 0 - 6	Soil/sediment	Spokane River	Dermal
				Child 0 - 6/adult 7 - 30	Soil/sediment	Spokane River	Dermal
	Current and future	Public recreational	Visitor	Child 0 - 6	Surface water	Spokane River	Ingestion
				Child 0 - 6/adult 7 - 30	Surface water	Spokane River	Ingestion
All Areas	Current and future	Residential	Resident	Child 0 - 6	Plant tissue	Homegrown vegetables	Ingestion
				Child 0 - 6/adult 7 - 30	Plant tissue	Homegrown vegetables	Ingestion
	Current and future	Public recreational	Visitor	Adult	Animal tissue	Fish in lower Coeur d'Alene River	Ingestion

[a]Vegetables and fish are listed separately under "All Areas."
[b]Shown in Figure 3-1
[c]Exposures for children living in Osburn, Silverton, and Wallace

Note:
NS confluence - confluence of North Fork Coeur d'Alene River and South Fork Coeur d'Alene River

Table 3-19b Tribal Exposure Routes To Be Considered

Traditional Subsistence Scenario

Media	Exposure Route	Quantified or qualified?	Reasoning if not quantified
Soil	Ingestion	Quantified	
	Dermal	Quantified	
	Inhalation	Qualified	insignificant pathway
Sediment	Ingestion	Quantified	
	Dermal	Quantified	
	Inhalation	Qualified	insignificant pathway
Air	Inhalation	Qualified	insignificant pathway
Seep/Spring Water	Ingestion	Qualified	limited by available data
	Dermal	Qualified	limited by available data
	Inhalation	Qualified	insignificant pathway
Surface Water	Ingestion	Quantified	
	Dermal	Qualified	insignificant pathway
	Inhalation	Qualified	insignificant pathway
	Recreational ingestion	Quantified	
Biota	Fish	Quantified	for lateral lakes only
	Water potatoes	Quantified	
	Animal protein	Qualified	limited by available data
	Other organs	Qualified	limited by available data
	Upland birds	Qualified	limited by available data
	Waterfowl	Qualified	limited by available data
	Breast milk	Qualified	insignificant pathway
Steam (sweating)	Dermal	Qualified	insignificant pathway
	Inhalation	Qualified	insignificant pathway

Modern Subsistence Scenario

Media	Exposure Route	Quantified or qualified?	Reasoning if not quantified
Soil	Ingestion	Quantified	
	Dermal	Quantified	
	Inhalation	Qualified	insignificant pathway
Sediment	Ingestion	Quantified	
	Dermal	Quantified	
	Inhalation	Qualified	insignificant pathway
Air	Inhalation	Qualified	insignificant pathway
Seep/Spring Water	Ingestion	Qualified	limited by available data
	Dermal	Qualified	limited by available data
	Inhalation	Qualified	insignificant pathway
Surface Water	Ingestion	Quantified	
	Dermal	Qualified	insignificant pathway
	Inhalation	Qualified	insignificant pathway
Biota	Fish	Quantified	for lateral lakes only
	Water potatoes	Quantified	
	Animal protein	Qualified	limited by available data
	Other organs	Qualified	limited by available data
	Upland birds	Qualified	limited by available data
	Waterfowl	Qualified	limited by available data
	Breast milk	Qualified	insignificant pathway
Steam (sweating)	Dermal	Qualified	insignificant pathway
	Inhalation	Qualified	insignificant pathway

Table 3-20
Summary of Exposure Point Concentrations for Reasonable Maximum Exposure

Geographical Area	Exposure Medium	EPC Units	Antimony	Arsenic	Cadmium	Iron	Manganese	Mercury	Zinc
Lower Basin	Yard soil	mg/kg	8.25	48.53	6.87	37,703	2,292	—	1,199
	Tap water	µg/L	—	0.97	—	—	—	—	—
	Soil/sediment at lower Coeur d'Alene River	mg/kg	23.22	119.45	35.05	105,451	9,886	—	5,666
	Surface water at lower Coeur d'Alene River	µg/L	—	79.61	72.9	—	16,651	4.5	—
	Construction site soil	mg/kg	21.08	108.91	30.71	97,012	8,919	—	4,998
Kingston	Yard soil	mg/kg	4.06	25.04	3.92	21,971	1,085	—	719
	Tap water	µg/L	—	0.405	—	—	—	—	—
	Sediment at Pine Creek	mg/kg	12.83	22.35	0.92	16,300	451.62	—	320.31
	Surface water at Pine Creek	µg/L	—	0.73	1.08	—	38.46	0.09	—
	Soil/sediment at NS confluence	mg/kg	39.78	163.2	26.58	100,621	8,585	–	3,744
	Surface water at NS confluence	µg/L	–	134	83.8	–	9,470	4.6	–
	Construction site soil	mg/kg	6.86	34.6	5.1	29,003	1,693	—	1,057
Side Gulches	Yard soil	mg/kg	15.94	50.74	9.62	27,190	1,367	—	1,320
	Tap water	µg/L	—	8.4	—	—	—	—	—
	Surface soil at Elk Creek	mg/kg	54.9	98.5	64.5	99,928	9,280	—	6,130
	Sediment at Elk Creek	mg/kg	27.84	77.74	38.38	35,970	2,223	—	5,031
	Surface water at Elk Creek	µg/L	—	32	60.3	—	8,570	3.5	—
Osburn	Yard soil	mg/kg	12.54	46.74	5.25	22,488	1,199	—	597
	Tap water	µg/L	—	0.28	—	—	—	—	—
Silverton	Yard soil	mg/kg	8.9	21.46	5.44	20,198	1,068	—	816
	Tap water	µg/L	—	0.545	—	—	—	—	—
	Surface soil at upland parks/schools	mg/kg	6.94	28.44	4.57	20,025	1,090	—	739

Table 3-20 (Continued)
Summary of Exposure Point Concentrations for Reasonable Maximum Exposure

Geographical Area	Exposure Medium	EPC Units	Antimony	Arsenic	Cadmium	Iron	Manganese	Mercury	Zinc
Wallace	Yard soil	mg/kg	13.7	22.57	7.63	22,240	989	—	967
	Tap water	µg/L	—	0.494	—	—	—	—	—
	Surface soil at upland parks/schools	mg/kg	18.22	27.65	10.72	28,639	1,502	—	1,921
MidGradSeg01[a]	Sediment at South Fork Coeur d'Alene River[b]	mg/kg	38.11	52.64	18.89	41,314	3,755	—	3,397
	Surface water at South Fork Coeur d'Alene River[b]	µg/L	—	1.096	4.029	—	32.04	0.122	—
	Construction site soil	mg/kg	9.26	27.31	5.21	21,860	1,129	—	752
Nine Mile	Yard soil	mg/kg	11.94	41.62	6.52	23,311	1,250	—	1,159
	Tap water	µg/L	—	0.74	—	—	—	—	—
	Future tap water	µg/L	2.1	1.25	130.85	—	—	—	19,756
	Waste piles	mg/kg	42.34	518.49	31.67	43,032	1,721	—	6,196
	Sediment at Nine Mile/Canyon Creeks	mg/kg	71.25	50.62	59.2	36,476	2,626	—	8,883
	Surface water at Nine Mile/Canyon Creeks	µg/L	—	1.1	111.02	—	1,699	0.31	—
	Construction site soil	mg/kg	10.61	24.97	6.54	21,976	1,135	—	1,070
Mullan	Yard soil	mg/kg	20.05	39.04	6.39	24,742	1,628	—	1,375
	Tap water	µg/L	—	0.25	—	—	—	—	—
	Waste piles	mg/kg	20.3	42.1	13.8	33,200	2,750	—	2,880
	Sediment at South Fork Coeur d'Alene River	mg/kg	4.46	15.89	19.83	47,300	5,233	—	4,017
	Surface water at South Fork Coeur d'Alene River	µg/L	—	0.6	1.24	—	18.96	0.086	—
	Construction site soil	mg/kg	18.68	31.66	5.04	26,912	1,941	—	953
Blackwell Island	Soil/sediment at Spokane River	mg/kg	3.195	50	13.4	48,881	3,666	—	1,800
	Surface water at Spokane River	µg/L	—	20.8	47.9	—	6,980	0.1	—

Table 3-20 (Continued)
Summary of Exposure Point Concentrations for Reasonable Maximum Exposure

Geographical Area	Exposure Medium	EPC Units	Antimony	Arsenic	Cadmium	Iron	Manganese	Mercury	Zinc
All geographical areas	Homegrown vegetables	mg/kg	—	0.025	0.319	—	—	—	—
	Fish from lower Coeur d'Alene River								
	Bullhead	mg/kg	—	—	0.008	—	—	0.052	—
	Northern pike	mg/kg	—	—	0.006	—	—	0.133	—
	Perch	mg/kg	—	—	0.037	—	—	0.089	—

[a]Shown in Figure 3-1
[b]Exposures for children living in Osburn, Silverton, and Wallace

Notes:
— - not a COPC for this medium
NS confluence - confluence of North Fork Coeur d'Alene River and South Fork Coeur d'Alene River

Table 3-21
Number of Samples Used to Calculate Exposure Point Concentrations

Geographical Area	Residential						Occupational	Public Recreational[f]				Neighborhood Recreational		
	No. of Homes	SS	Vacuum Bag Dust[a]	Floor Mat Dust[a]	TW	GW[b]	SS and SB	SS	SB	SW	SD	Waste Pile	SW	SD
Lower Basin	13	28	1	2	4	—	457	155	—	122	233	—	—	—
Kingston	22	71	6	6	7	—	226	5	—	5	14	—	12	13
Side Gulches	26	81	5	6	8	—	—	—	—	—	—	5[d]	5	10
Osburn	42	309	25	27	32	—	— [c]	—	—	—	—	—	—	—
Silverton	20	55	4	4	4	—	— [c]	151	464	—	—	—	—	—
Wallace	14	82	8	8	11	—	— [c]	68	202	—	—	—	—	—
MidGradSeg01	—[e]	—	—	—	—	—	2,312	—	—	—	—	—	56	21
Nine Mile	33	222	18	23	26	80	677	—	—	—	—	22	80	17
Mullan	21	81	7	7	8	—	230	—	—	—	—	5	67	14
Blackwell Island	0	—	—	—	—	—	—	7	—	7	14	—	—	—

[a]Vacuum cleaner bag and floor mat data were not used directly in the calculations of exposure point concentrations (see text Section 3.3.1 and 7.3.1).
[b]Groundwater data are used to calculate a "future drinking water risk" for hypothetical residents in Nine Mile area.
[c]The data from these areas are combined under MidGradSeg01 for occupational exposures.
[d]These are surface soil samples from Elk Creek area, rather than waste pile data.
[e]Residential data from this segment are shown for the individual towns, i.e., Osburn, Wallace, and Silverton.
[F]Surface soil and sediment were combined for calculation of exposure pint concentrations.
Notes:
— - no data selected for use in risk assessment (data were either not available or not applicable)
GW - groundwater
SB - subsurface soil collected from more than 1 inch below ground surface
SD - Sediment
SS - surface soil collected from 0 to 1 inch below ground surface
SW - surface water
TW - tap water

Table 3-22
Residential Exposure Factors

Exposure Parameter	RME Value	Reference	CT Value	Reference
Exposure Assumptions for Ingestion of Chemicals in Yard Soil				
Ingestion rate - adult (mg/day)	100	USEPA 1991a	50	USEPA 1993c
Ingestion rate - child (mg/day)	200	USEPA 1991a	100	USEPA 1993c
Exposure frequency (days/year)	350	USEPA 1991a	260	A
Exposure duration - adult (years)	24	USEPA 1991a	7	USEPA 1993c
Exposure duration - child (years)	6	USEPA 1991a	2	USEPA 1993c
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Body weight - adult (kg)	70	USEPA 1991a	70	USEPA 1991a
Body weight - child (kg)	15	USEPA 1991a	15	USEPA 1991a
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer, child/adult (days)	10,950	USEPA 1989	3,285	USEPA 1989
Averaging time - noncancer, child (days)	2,190	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Dermal Contact With Chemicals in Yard Soil				
Skin surface area - adult (cm^2)	2,500	USEPA 1998e	2,500	USEPA 1998e
Skin surface area - child (cm^2)	2,200	USEPA 1998e	2,200	USEPA 1998e
Adherence factor - adult (mg/cm^2-event)	0.1	USEPA 1998e	0.1	USEPA 1998e
Adherence factor - child (mg/cm^2-event)	0.2	USEPA 1998e	0.2	USEPA 1998e
Exposure frequency (events/year)	350	USEPA 1991a	260	A
Exposure duration - adult (years)	24	USEPA 1991a	7	USEPA 1993c
Exposure duration - child (years)	6	USEPA 1991a	2	USEPA 1993c
Body weight - adult (kg)	70	USEPA 1991a	70	USEPA 1991a
Body weight - child (kg)	15	USEPA 1991a	15	USEPA 1991a
Dermal absorption factor (unitless)	Chem. specific	USEPA 1998e	Chem. specific	USEPA 1998e
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer, child/adult (days)	10,950	USEPA 1989	3,285	USEPA 1989
Averaging time - noncancer, child (days)	2,190	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Ingestion of Tap Water				
Ingestion rate - adult (L/day)	2	USEPA 1991a	1.4	USEPA 1993c
Ingestion rate - child (L/day)	1	USEPA 1999c	1	USEPA 1999a
Exposure duration - adult (years)	24	B	7	USEPA 1993c
Exposure duration - child (years)	6	B	2	USEPA 1993c
Body weight - adult (kg)	70	USEPA 1991a	70	USEPA 1991a
Body weight - child (kg)	15	USEPA 1991a	15	USEPA 1991a
Exposure frequency (days/year)	350	USEPA 1991a	234	USEPA 1993c
Conversion factor (mg/µg)	1.0E-03	NA	1.0E-03	NA
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer, child/adult (days)	10,950	USEPA 1989	3,285	USEPA 1989
Averaging time - noncancer, child (days)	2,190	USEPA 1989	730	USEPA 1989

Table 3-22 (Continued)
Residential Exposure Factors

Exposure Parameter	RME Value	Reference	CT Value	Reference
Exposure Assumptions for Ingestion of Homegrown Vegetables				
Intake rate of homegrown vegetables (g/kg-day)	5.04	C	0.492	C
Exposure frequency (days/year)	365	D	365	D
Exposure duration (years)	30	USEPA 1991a	9	USEPA 1993c
Conversion factor (kg/g)	1.0E-03	NA	1.0E-03	NA
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time -noncancer (days)	10,950	USEPA 1989	3,285	USEPA 1989

Notes:
A - Exposure frequency was based on 3 months limited soil exposure due to snow-covered or frozen ground.
B - USEPA 1991a recommends an adult/child exposure duration of 24/6 years for ingestion of soil; for consistency, an exposure duration of 24/6 years was selected for ingestion of tap water.
C - Ingestion rate is seasonally adjusted and incorporates the body weights of all participants in the study (children and adults) from USEPA 1997a.
D - Ingestion rate of vegetables is an average daily consumption rate; therefore, 365 days/year was selected as the frequency of exposure for both the RME and CT cases.

N/A - not applicable

Table 3-23
Neighborhood Recreational Exposure Factors

Exposure Parameter	RME Value	Reference	CT Value	Reference
Exposure Assumptions for Ingestion of Chemicals in Waste Pile Soil				
Ingestion rate (mg/day)	300	A	120	A
Exposure frequency (days/year)	17	B	8.5	B
Exposure duration (years)	7	C	2	USEPA 1993c
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Body weight (kg)	28	D	28	D
Averaging time - cancer (days)	25,500	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer (days)	2,555	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Dermal Contact With Chemicals in Waste Pile Soil				
Skin surface area (cm^2)	5,080	USEPA 1997a	5,080	USEPA 1997a
Soil to skin adherence factor (mg/cm2-event)	0.2	USEPA 1998e	0.2	USEPA 1998e
Dermal absorption factor (unitless)	Chem. specific	USEPA 1998e	Chem. specific	USEPA 1998e
Exposure frequency (events/year)	34	E	17	E
Exposure duration (years)	7	C	2	USEPA 1993c
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Body weight (kg)	28	D	28	D
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer (days)	2,555	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Ingestion of Chemicals in Upland Soil (Parks/Schools/Elk Creek Area)				
Ingestion rate (mg/day)	300	A	120	A
Exposure frequency (days/year)	34	F	17	F
Exposure duration (years)	7	C	2	USEPA 1993c
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Body weight (kg)	28	D	28	D
Averaging time - cancer (days)	25,500	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer (days)	2,555	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Dermal Contact With Chemicals in Upland Soil (Parks/Schools/Elk Creek Area)				
Skin surface area (cm^2)	5,080	USEPA 1997a	5,080	USEPA 1997a
Soil to skin adherence factor (mg/cm^2-event)	0.2	USEPA 1998e	0.2	USEPA 1998e
Dermal absorption factor (unitless)	Chem. specific	USEPA 1998e	Chem. specific	USEPA 1998e
Exposure frequency (events/year)	68	G	34	G
Exposure duration (years)	7	C	2	USEPA 1993c
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Body weight (kg)	28	D	28	D
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer (days)	2,555	USEPA 1989	730	USEPA 1989

Table 3-23 (Continued)
Neighborhood Recreational Exposure Factors

Exposure Parameter	RME Value	Reference	CT Value	Reference
Exposure Assumptions for Ingestion of Chemicals in Floodplain Soil/Sediment				
Ingestion rate (mg/day)	300	A	120	A
Exposure frequency (days/year)	21	H	10	H
Exposure duration (years)	7	C	2	USEPA 1993c
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Body weight (kg)	28	D	28	D
Averaging time - cancer (days)	25,500	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer (days)	2,555	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Dermal Contact With Chemicals in Floodplain Soil/Sediment				
Skin surface area (cm^2)	5,080	I	5,080	I
Soil to skin adherence factor (mg/cm^2-event)	0.2	USEPA 1998e	0.2	USEPA 1998e
Dermal absorption factor (unitless)	Chem. specific	USEPA 1998e	Chem. specific	USEPA 1998e
Exposure frequency (events/year)	96	J	48	J
Exposure duration (years)	7	C	2	USEPA 1993c
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Body weight (kg)	28	D	28	D
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer (days)	2,555	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Ingestion of Surface Water				
Ingestion rate (mL/hour)	30	USEPA 1998d	30	USEPA 1998d
Exposure time (hours/day)	1	USEPA 1997a	1	USEPA 1997a
Exposure frequency (days/year)	96	I	I	
Exposure duration (years)	7	C	2	USEPA 1993c
Conversion factor (mg/µg)	0.001	NA	0.001	NA
Conversion factor (L/mL)	0.001	NA	0.001	NA
Body weight (kg)	28	D	28	D
Averaging time - cancer (days)	2.6E+04	USEPA 1989	2.6E+04	USEPA 1989
Averaging time - noncancer (days)	2,555	USEPA 1989	730	USEPA 1989

Notes:
A - The RME value of 300 mg/day is the 90th percentile soil intake from van Wijnen (1990); the CT value of 120 mg/day is the mean soil intake from the same study, as cited in USEPA 1999d.
B - Exposure frequency for the RME is calculated as 34 weeks/year x 7 hours/day x 1 day/week / 14 hours/day = 17 days/year; for the CT as 34 weeks/year x 7 hours/day x once every other week, 0.5 / 14 hours/day = 8.5 days/year.
C - Neighborhood exposure assumes children ages 4 to 11 playing in the waste piles.
D - Value is the 50th percentile for boys and girls, ages 4 to 11.
E - Exposure frequency for the RME is calculated as 34 weeks/year x 1 event/week = 34 events/year; for the CT as 34

Table 3-23 (Continued)
Neighborhood Recreational Exposure Factors

weeks/year x once every other week = 17 events/year.
F - Exposure frequency for the RME is calculated as 34 weeks/year x 7 hours/day x 2 days/week / 14 hours/day = 34 days/year, assuming weekend outdoor exposure. Exposure frequency for the CT is calculated as 34 weeks/year x 7 hours/day x 1 day/week / 14 hours/day = 17 days/year.
G - Exposure frequency for the RME is calculated as 34 weeks/year x 2 events/week = 68 events/year; for the CT as 34 weeks/year x 1 event/week = 34 events/year.
H - Exposure frequency for the RME is calculated as 24 weeks/year x 3 hours/day x 4 days/week / 14 hours/day = 21 days/year (3 hours/day is the high end of the 50th percentile range (1 to 3 hours/day) from USEPA 1997a). Exposure frequency for the CT is calculated as 24 weeks/year x 3 hours/day x 2 days/week / 14 hours/day = 10 days/year.
I - Exposure frequency for the RME is calculated as 24 weeks/year x 4 events/week = 96 events/year; for the CT as 24 weeks/year x 2 events/week = 48 events/year.
J - At the Lower Basin and Kingston (NS confluence), a skin surface area of 7,960 cm^2 was used to reflect the possibility that swimming and, therefore, exposure of the entire body to contaminants in sediment could occur at these locations. It was assumed that swimming would occur during 16 weeks of the year (the warmest months), while wading and playing along the shoreline without swimming would occur during 8 weeks of the year. The median skin surface area for male children ages 4 to 11 is 9,400 cm^2 (USEPA 1997a). The skin surface area was calculated as follows: ((16 weeks x 9,400 cm^2) + (8 weeks x 5,080 cm^2)) / 24 weeks = 7,960 cm^2.

NA - not available

Table 3-24
Public Recreational Exposure Factors

Exposure Parameter	RME Value	Reference	CT Value	Reference
Exposure Assumptions for Ingestion of Chemicals in Upland Soil (Parks/Schools)				
Ingestion rate - adult (mg/day)	100	USEPA 1991a	50	USEPA 1993c
Ingestion rate - child (mg/day)	300	A	120	A
Exposure frequency - adult (days/year)	30	B	15	B
Exposure frequency - child (days/year)	34	B	17	B
Exposure duration - adult (years)	24	USEPA 1991a	7	USEPA 1993c
Exposure duration - child (years)	6	USEPA 1991a	2	USEPA 1993c
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Body weight - adult (kg)	70	USEPA 1991a	70	USEPA 1991a
Body weight - child (kg)	15	USEPA 1991a	15	USEPA 1991a
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer, child/adult (days)	10,950	USEPA 1989	3,285	USEPA 1989
Averaging time - noncancer, child (days)	2,190	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Dermal Contact With Chemicals in Upland Soil (Parks/Schools)				
Skin surface area - adult (cm^2)	2,500	USEPA 1998e	2,500	USEPA 1998e
Skin surface area - child (cm^2)	2,200	USEPA 1998e	2,200	USEPA 1998e
Adherence factor - adult (mg/cm^2-event)	0.1	USEPA 1998e	0.1	USEPA 1998e
Adherence factor - child (mg/cm^2-event)	0.2	USEPA 1998e	0.2	USEPA 1998e
Exposure frequency (events/year)	68	C	34	C
Exposure duration - adult (years)	24	USEPA 1991a	7	USEPA 1993c
Exposure duration - child (years)	6	USEPA 1991a	2	USEPA 1993c
Body weight - adult (kg)	70	USEPA 1991a	70	USEPA 1991a
Body weight - child (kg)	15	USEPA 1991a	15	USEPA 1991a
Dermal absorption factor (unitless)	Chem. specific	USEPA 1998e	Chem. specific	USEPA 1998e
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer, child/adult (days)	10,950	USEPA 1989	3,285	USEPA 1989
Averaging time - noncancer, child (days)	2,190	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Ingestion of Chemicals in Floodplain Soil/Sediment				
Ingestion rate - adult (mg/day)	100	USEPA 1991a	50	USEPA 1993c
Ingestion rate - child (mg/day)	300	A	120	A
Exposure frequency (days/year)	32	D	16	D
Exposure duration - adult (years)	24	USEPA 1991a	7	USEPA 1993c
Exposure duration - child (years)	6	USEPA 1991a	2	USEPA 1993c
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Body weight - adult (kg)	70	USEPA 1991a	70	USEPA 1991a
Body weight - child (kg)	15	USEPA 1991a	15	USEPA 1991a
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer, child/adult (days)	10,950	USEPA 1989	3,285	USEPA 1989

Table 3-24 (Continued)
Public Recreational Exposure Factors

Exposure Parameter	RME Value	Reference	CT Value	Reference
Averaging time - noncancer, child (days)	2,190	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Dermal Contact With Chemicals in Floodplain Soil/Sediment				
Skin surface area - adult (cm^2)	18,000	USEPA 1998e	18,000	USEPA 1998e
Skin surface area - child (cm^2)	6,500	USEPA 1998e	6,500	USEPA 1998e
Adherence factor - adult (mg/cm^2-event)	0.1	USEPA 1998e	0.1	USEPA 1998e
Adherence factor - child (mg/cm^2-event)	0.2	USEPA 1998e	0.2	USEPA 1998e
Exposure frequency (events/year)	32	D	16	D
Exposure duration - adult (years)	24	USEPA 1991a	7	USEPA 1993c
Exposure duration - child (years)	6	USEPA 1991a	2	USEPA 1993c
Body weight - adult (kg)	70	USEPA 1991a	70	USEPA 1991a
Body weight - child (kg)	15	USEPA 1991a	15	USEPA 1991a
Dermal absorption factor (unitless)	Chem. specific	USEPA 1998e	Chem. specific	USEPA 1998e
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer, child/adult (days)	10,950	USEPA 1989	3,285	USEPA 1989
Averaging time - noncancer, child (days)	2,190	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Ingestion of Surface Water				
Ingestion rate (mL/hour)	30	USEPA 1998d	30	USEPA 1998d
Exposure time (hours/day)	1	USEPA 1997a	1	USEPA 1997a
Exposure duration - adult (years)	24	E	7	USEPA 1993c
Exposure duration - child (years)	6	E	2	USEPA 1993
Body weight - adult (kg)	70	USEPA 1991a	70	USEPA 1991a
Body weight - child (kg)	15	USEPA 1991a	15	USEPA 1991a
Exposure frequency (days/year)	32	D	16	D
Conversion factor (mg/µg)	1.0E-03	NA	1.0E-03	NA
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer, child/adult (days)	10,950	USEPA 1989	3,285	USEPA 1989
Averaging time - noncancer, child (days)	2,190	USEPA 1989	730	USEPA 1989
Exposure Assumptions for Ingestion of Fish				
Ingestion rate of fish (g/day)	46	ATSDR 1989	25	USEPA 1997a
Exposure frequency (days/year)	365	F	365	F
Exposure duration (years)	30	USEPA 1991a	9	USEPA 1993c
Conversion factor (kg/g)	1.0E-03	NA	1.0E-03	NA
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time -noncancer (days)	10,950	USEPA 1989	3,285	USEPA 1989

Notes:
A - The RME value of 300 mg/day is the 90th percentile soil intake from van Wijnen (1990); the CT value of 120 mg/day is the mean soil intake from the same study, as cited in USEPA 1999d.

Table 3-24 (Continued)
Public Recreational Exposure Factors

B - RME exposure frequency for adult: 34 weeks/year x 7 hours/day x 2 days/week / 16 hours/day = 30 days/year; for child: 34 weeks/year x 7 hours/day x 2 days/week / 14 hours/day = 34 days/year. Two days/week assumes weekend outdoor exposure. The CT exposure frequency for adults is: 34 weeks/year x 7 hours/day x 1 day/week / 16 hours/day = 15 days/year; for a child, 34 weeks/year x 7 hours/day x 1 day/week / 14 hours/day = 17 days/year.
C - Exposure frequency for the RME is calculated as 34 weeks/year x 2 events/week = 68 events/year; for the CT as 34 weeks/year x 1 event/week = 34 events/year.
D - Professional judgement
E - USEPA 1991a recommends an adult/child exposure duration of 24/6 years for ingestion of soil; for consistency, an exposure duration of 24/6 years was selected for ingestion of tap water.
F - Ingestion rate of fish is an average daily consumption rate; therefore, 365 days/year was selected as the frequency of exposure for both the RME and CT cases.

NA - not available

Table 3-25
Occupational Exposure Factors

Exposure Parameter	RME Value	Reference	CT Value	Reference
Exposure Assumptions for Ingestion of Chemicals in Construction Site Soil				
Ingestion rate (mg/day)	300	USEPA 1999d	200	USEPA 1999d
Exposure frequency (days/year)	195	A	43	A
Exposure duration (years)	25	USEPA 1991a	6.6	USEPA 1997a
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Body weight (kg)	70	USEPA 1991a	70	USEPA 1991a
Averaging time - cancer (days)	25,500	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer (days)	9,125	USEPA 1989	2,409	USEPA 1989
Exposure Assumptions for Dermal Contact With Chemicals in Construction Site Soil				
Skin surface area (cm^2)	2,500	USEPA 1998e	2,500	USEPA 1998e
Soil to skin adherence factor (mg/cm^2-event)	0.1	USEPA 1998e	0.1	USEPA 1998e
Dermal absorption factor (unitless)	Chem. specific	USEPA 1998e	Chem. specific	USEPA 1998e
Exposure frequency (events/year)	195	A	43	A
Exposure duration (years)	25	USEPA 1991a	6.6	USEPA 1997a
Conversion factor (kg/mg)	1.0E-06	NA	1.0E-06	NA
Body weight (kg)	70	USEPA 1991a	70	USEPA 1991a
Averaging time - cancer (days)	25,550	USEPA 1989	25,550	USEPA 1989
Averaging time - noncancer (days)	2,555	USEPA 1989	730	USEPA 1989

Notes:
A - professional judgement
NA - not available

Table 3-26a Traditional Subsistence Scenario Exposure Factors

Media	Exposure Route	Age Group	Intake/ Contact Rate (per day)	Exposure Frequency (days/year)	Exposure Duration (years)	Other Parameters	Other Parameters Definitions	Comments
Soil	Ingestion	Child	300 mg	365	6	-	-	Ingestion rate is derived from a study of children at campgrounds as cited in EPA Region 10 Guidance (1999c).
	Ingestion	Adult	300 mg	365	64	-	-	Ingestion rate is derived from a study of children at campgrounds as cited in EPA Region 10 Guidance (1999c). EPA also recommends this value for a "soil contact
	Dermal	Child	0.8 mg/cm^2	365	6	2200 cm^2	Skin surface area	Contact value is average surface area of head, forearms, hands, lower legs, and feet of 0-6 year old children.
	Dermal	Adult	0.8 mg/cm^2	365	64	2500 cm^2	Skin surface area	Contact value is average surface area of face, hands, and forearms for adults.
Sediment	Ingestion	Child	300 mg	210	6	-	-	Child ingestion rate derived from studies at campgrounds. Assumes 214 days per year based on 7 months (April through October) due to weather constraints.
	Ingestion	Adult	300 mg	210	64	-	-	EPA Region 10 recommends 300 mg/day for a "soil contact intense" adult exposure scenario. Assumes 214 days per year based on 7 months (April through October) due to weather constraints.
	Dermal	Child	0.8 mg/cm^2	210	6	6500 cm^2	Skin surface area	Contact value is derived from a study conducted by Kissel et al (Ref by EPA 1997a) . 6500 cm^2 assumes full body exposure for children. Assumes 214 days per year based on 7 months (April through October) due to weather constraints.
	Dermal	Adult	0.8 mg/cm^2	210	64	18,000 cm^2	Skin surface area	Contact value is derived from a study conducted by Kissel et al (Ref by EPA 1997a). 18000 cm^2 assumes full body exposure for adults. Assumes 214 days per year based on 7 months (April through October) due to weather constraints.

Table 3-26a Traditional Subsistence Scenario Exposure Factors (continued)

Media	Exposure Route	Age Group	Intake/ Contact Rate (per day)	Exposure Frequency (days/year)	Exposure Duration (years)	Other Parameters	Other Parameters Definitions	Comments
Surface Water	Ingestion	Child	1.5 L	365	6	-	-	Assumes 1.5 L of undiluted/unfiltered surface water is consumed daily.
	Ingestion	Adult	3 L	365	64	-	-	Assumes 3 L of undiluted/unfiltered surface water is consumed daily.
	Recreational Ingestion	Child/Adult	30 mL/hr	210	70	-	-	30 mL/hour is the EPA default value for incidental ingestion of surface water.
Biota	Fish	Child/Adult	540 g (total wet weight)	365	70	-	-	540 g represents a reasonable maximum intake based upon several sources/surveys. 540 g is equivalent to approximately 1/3 of the adult daily diet (800-1000 kcal/day depending upon species).
	Fruit and Vegetation (includes *Sagittaria spp.*)	Child/Adult	8.2 g/kg	365	70	-	-	574 grams per day for a 70 kg adult based on Umatilla estimate (CRCRA 1996); equivalent to 8.2 grams per kilogram per day.

mg: milligram
mg/cm^2: milligrams per square centimeter
m^3: cubic meter
L: liter
hr: hour
g: gram
kg: kilogram
mL/day: milliliter per day
cm^2: square centimeter
$\mu g/m^3$: micrograms per cubic meter
cm^3: cubic centimeter
L/m^3: liter per cubic meter
kcal: kilocalorie

Table 3-26b Modern Subsistence Scenario Exposure Factors

Pathways		Exposure Parameters						
Media	Exposure Route	Age Group	Intake/ Contact Rate (per day)	Exposure Frequency (days/year)	Exposure Duration (years)	Other Parameters	Other Parameter Definitions	Comments
Soil	Ingestion	Child	300 mg	61	6	-	-	Ingestion rate is derived from a study of children at campgrounds as cited in EPA Region 10 Guidance. Limited to 122 day annual exposure during the warmest 4 months of the year (June through
	Ingestion	Adult	300 mg	61	64	-	-	Ingestion rate is derived from a study of children at campgrounds as cited in EPA Region 10 Guidance (1999c). EPA also recommends this value for a "soil contact intense" adult exposure scenario. Limited to 122 day annual exposure during the warmest 4 months of the year (June through September).
	Dermal	Child	0.8 mg/cm^2	61	6	2800 cm^2	Skin surface area	Contact value is average surface area of head, forearms, hands, lower legs, and feet of 0-6 year old children. Limited to 122 day annual exposure during the warmest 4 months of the year (June through September).
	Dermal	Adult	0.8 mg/cm^2	61	64	5700 cm^2	Skin surface area	Contact value is derived from a study conducted by Kissel et al (Ref by EPA 1997a). 5000 cm2 represents 25% of the total available skin surface area. Limited to 122 day annual exposure during the warmest 4 months of the year (June through September).
Sediment	Ingestion	Child	300 mg	61	6	-	-	Child ingestion rate derived from studies at campgrounds. Assumes 122 days per year based on 4 months due to weather constraints.
	Ingestion	Adult	300 mg	61	64	-	-	EPA Region 10 recommends 300 mg/day for a "soil contact intense" adult exposure scenario. Limited to 122 day annual exposure during the warmest 4 months of the year (June through September).
	Dermal	Child	0.8 mg/cm^2	61	6	6500 cm^2	Skin surface area	Contact value is derived from a study conducted by Kissel et al (Ref by EPA 1997a). 6500 cm^2 assumes full body exposure for children. Assumes 122 days per year based on 4 months due to weather constraints.
	Dermal	Adult	0.8 mg/cm^2	61	64	18,000 cm^2	Skin surface area	Contact value is derived from a study conducted by Kissel et al (Ref by EPA 1997a). 18000 cm^2 assumes full body exposure for adults. Assumes 122 days per year based on 4 months due to weather constraints.

Table 3-26b Modern Subsistence Scenario Exposure Factors (continued)

Pathways		Exposure Parameters						
Media	**Exposure Route**	**Age Group**	**Intake/ Contact Rate (per day)**	**Exposure Frequency (days/year)**	**Exposure Duration (years)**	**Other Parameters**	**Other Parameter Definitions**	**Comments**
Surface Water	Ingestion	Child	1.5 L	61	6	-	-	Assumes 1.5 L of undiluted/unfiltered surface water is consumed daily while in the field. Limited to 122 day annual exposure during the warmest 4 months of the year (June through September).
	Ingestion	Adult	3 L	61	64	-	-	Assumes 3 L of undiluted/unfiltered surface water is consumed daily while in the field. Limited to 122 day annual exposure during the warmest 4 months of the year (June through September).
	Recreational Ingestion	Child/Adult	30 mL/hr	61	70	-	-	30 mL/hour is the EPA default value for incidental ingestion of surface water.
Biota	Fish	Child/Adult	170 g (total wet weight)	365	70	-	-	95th percentile consumption rate for 4 Columbia River tribes, as presented in EPA's Exposure Factors Handbook
	Fruit and Vegetation (includes *Sagittaria spp.*)	Child/Adult	1.6 g/kg	365	70	-	-	Value represents 20% of the traditional subsistence ingestion rate of 8.2 g/kg-day.

mg: milligram
mg/cm^2: milligrams per square centimeter
m^3: cubic meter
L: liter
hr: hour
g: gram
kg: kilogram
mL/day: milliliter per day
cm^2: square centimeter
$\mu g/m^3$: micrograms per cubic meter
cm^3: cubic centimeter
L/m^3: liter per cubic meter
kcal: kilocalorie

4.0 HUMAN HEALTH TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to weigh the available and relevant evidence regarding the potential for chemicals to cause adverse health effects to exposed individuals, and to provide a quantitative estimate of the relationship between the magnitude of exposure and the likelihood of adverse effects (USEPA 1989). This section summarizes the potential toxic effects of each chemical of concern as well as the relevant toxicity criteria that are used to assess the risks associated with the dose of the COPCs. A fundamental principle of toxicology is that the dose determines the severity of the effect. Accordingly, the toxicity criteria describe the quantitative relationship between the dose of a chemical and the type and incidence of the toxic effect. This relationship is referred to as the dose-response. The types of toxicity criteria are described below followed by brief discussions of specific criteria and associated health effects for each COPC. More detailed discussions of toxicity criteria for each metal are provided in Appendix H. Table 4-1 and Tables 5.1, 5.2, 6.1 and 6.2 in Appendix A summarize the toxicity criteria used in this assessment.

4.1 ORAL TOXICITY CRITERIA

A dose-response evaluation is the process of quantitatively evaluating toxicity information and characterizing the relationship between the dose of the chemical and the incidence of adverse health effects in the exposed population. From this quantitative dose-response relationship, toxicity criteria are derived that can be used to estimate the potential for adverse health effects as a function of exposure to the chemical. Toxicity values are combined with the summary intake factors calculated in Section 3 and are used to calculate human risks for various exposure scenarios. Exposure to chemicals can result in cancer or noncancer effects, which are characterized separately. Essential dose-response criteria are the EPA slope factor (SF) values for assessing cancer risks and the EPA-verified RfD values for evaluating noncancer effects. These criteria are from the EPA's online database, Integrated Risk Information System (IRIS) (USEPA 2000a). Where IRIS criteria were not available (only iron), other EPA sources of toxicity criteria were investigated.

4.1.1 Cancer Effects

The cancer SF (in units of $(mg/kg\text{-}day)^{-1}$) expresses excess cancer risk as a function of dose. The dose-response model is based on high- to low-dose extrapolation, and assumes that there is no lower threshold for the initiation of toxic effects. Specifically, cancer effects observed at high doses in laboratory animals or from occupational or epidemiological studies are extrapolated, using mathematical models, to low doses common to environmental exposures. These models are essentially linear at low doses, such that no dose is without some risk of cancer. SFs have been developed by the EPA for both the oral (ingestion) and inhalation routes of exposure. Only oral SFs were used in the HHRA Report.

The SF for arsenic, the only established human carcinogen evaluated in this risk assessment, is based on human epidemiologic studies and real environmental exposures. The EPA has classified arsenic as a

proven human carcinogen. Some of the other metals of concern are classified as a probable or possible human carcinogen by EPA, but human data are limited or inadequate to classify them as a known (or proven) human carcinogen. Therefore, there are no corresponding Cancer Slope Factors for these COPCs, and a quantitative evaluation of possible associated cancers risk is not possible.

4.1.2 Noncancer Effects

Chronic RfDs are defined as an estimate of a daily exposure level for the human population, including sensitive populations, that is likely to be without appreciable risk of noncancer effects during a lifetime of exposure (USEPA 1989). Chronic RfDs are specifically developed to be protective for long-term exposure to a chemical and are generally used to evaluate the potential noncancer effects associated with exposure periods of 7 years to a lifetime. RfDs are expressed as mg/kg-day and are calculated using lifetime average body weight and intake assumptions.

RfD values are derived from experimental data on the no-observed-adverse-effect level (NOAEL) or lowest-observed-adverse-effect level (LOAEL) in animals or humans. The NOAEL is the highest tested chemical dose given to animals or humans that has not been associated with any adverse health effects. The LOAEL is the lowest chemical dose at which health effects have been reported. RfDs are calculated by dividing the NOAEL or LOAEL by a total uncertainty factor, which represents a combination of individual factors for various sources of uncertainty associated with the database for a particular chemical or with the extrapolation of animal data to humans. RfDs and associated uncertainty factors for each chemical are discussed in Section 4.3. IRIS also assigns a level of confidence in the RfD. The level of confidence is rated as either high, medium, or low based on confidence in the study and in the database. RfDs have been developed by the EPA for both the oral (ingestion) and inhalation routes of exposure. Only oral RfDs were used in this HHRA.

4.2 DERMAL TOXICITY CRITERIA

Only arsenic and cadmium were evaluated for dermal toxicity in this risk assessment because scientific support for dermal toxicity for the other metals is inadequate (USEPA 1999c). There are no available RfDs or SFs specifically for dermal exposures. Risks and hazards associated with dermal exposure are evaluated using an oral toxicity factor corrected for percutaneous absorption. This route-to-route extrapolation assumes that on the basis of absorbed (as opposed to administered) dose, the toxicity of a hazardous constituent is the same once it enters the blood, regardless of the actual route of exposure. The administered dose is the amount that is presented to a person's "exchange surfaces" or points of contact with the external world, including the mouth, skin, and nose. The absorbed dose is the fraction of the administered dose that actually enters the body's general circulation. Because the skin forms an effective barrier to many chemicals, only a fraction of the dose administered on the skin's surface will be absorbed through the skin into the bloodstream.

The chronic RfD for arsenic was not adjusted from an administered dose to an absorbed dose because the RfD is based on the NOAEL for skin effects from a study involving arsenic exposures to more than

40,000 people in Taiwan. These people were exposed for a significant portion of their lifetime to arsenic-contaminated groundwater used as drinking water. Because most arsenic ingested in water is absorbed through the gastrointestinal tract, the administered RfD is a good approximation of the orally absorbed dose (USEPA 2000a). For cadmium, the administered oral RfD of 0.001 mg/kg-day (food) was multiplied by a gastrointestinal fraction of 2.5 percent to derive the dermal RfD of 0.000025 mg/kg-day (USEPA 2000a).

4.3 CHEMICAL PROFILES

Toxic effects of the chemicals of concern are summarized in the following subsections along with the toxicity criteria for assessing noncancer and cancer effects. In general, the information has been summarized from the latest available ATSDR profile for each chemical and the information is provided in Appendix H.

4.3.1 Antimony

Antimony is found at low concentrations in soil, generally 1 mg/kg or less. The geochemical properties of antimony are similar to those of arsenic (i.e., antimony has +3 and +5 valence states). As with arsenic, antimony may be associated with nonferrous ore deposits and, therefore, can be a pollutant in industrial environments. Antimony is a constituent in alloys with nonferrous metals such as tin, lead, and copper. Sulfides are used in the production of rubber and pyrotechnics. Chlorides are used as coloring agents and catalysts.

Antimony is poorly absorbed from the gastrointestinal tract. Acute exposure by ingestion is irritating to the gastrointestinal tract. Long-term ingestion exposure in laboratory animals has been associated with changes in blood chemistry, including increased serum cholesterol and decreased nonfasting serum glucose levels. The issue of bioavailability of antimony in soil is important because antimony often exists, at least in part, as a poorly soluble salt and may also occur in particles of inert or insoluble material. These factors all tend to reduce the bioavailability of antimony.

Inhalation of antimony compounds has been reported to be toxic to smelter workers, producing effects in both the respiratory and gastrointestinal tract. Inhalation exposure in workers may also be associated with effects on the cardiovascular system (elevated blood pressure) and pneumoconiosis, including interstitial inflammation leading to fibrosis of the lung and altered pulmonary function.

There is inconclusive evidence of a relationship between the inhalation of antimony trioxide and excess risk of lung cancer and reproductive disorders. Cancer evidence from studies in human populations is very limited, and carcinogenicity studies in laboratory animals provide conflicting results.

The oral RfD for antimony of 0.0004 mg/kg-day is based on decreases in nonfasting blood glucose levels, altered cholesterol levels, and decreased longevity in rats administered antimony in drinking water at a concentration of 5 µg/L for life. The RfC of 0.0002 mg/m^3 has been developed specifically

for antimony trioxide and is based on the occurrence of chronic interstitial inflammation in the lungs and reduced clearance of inhaled particulates in rats exposed by inhalation for 1 year.

4.3.2 Arsenic

Arsenic trioxide is the most commercially important form of arsenic and is produced primarily from flue dust that is generated at copper and lead smelters. The principal use of arsenic (as arsenic trioxide) is in wood preservatives and a smaller proportion is used in the production of agricultural chemicals such as insecticides, herbicides, algaecides, and growth stimulants for plants and animals. The use of many arsenical pesticides has been phased out because of concerns about human health risks during production or use. Arsenic trioxide is no longer produced in the United States. Smaller amounts of arsenic are used in the production of glass and nonferrous alloys and in the semiconductor industry.

Arsenic has been shown to be toxic to human populations in areas of the world where it is present at naturally elevated concentrations in groundwater and to occupationally exposed workers in copper smelters and chemical plants. There is strong evidence that arsenic is carcinogenic in humans by both oral and inhalation routes. Arsenic occurs in soil and rock along with other minerals such as copper, lead, iron, and nickel. It is typically found in soil in the form of an insoluble sulfide. Naturally occurring arsenic concentrations in soil range from 1 to 40 mg/kg, with a mean concentration of approximately 5 mg/kg. Naturally occurring arsenic concentrations in groundwater average around 1 to 2 µg/L, except for some western states with geological features that have naturally elevated concentrations of arsenic. Concentrations in groundwater in these areas range from 5 to more than 500 µg/L. In the United States, over 350,000 people may drink water containing arsenic concentrations higher than the current MCL of 50 µg/L. USEPA has promulgated a new MCL for arsenic of 10 µg/L and estimated over 12 million people may be drinking water containing arsenic at concentrations above 10 µg/L (Federal Register 2001). The USGS estimates that 40% of both large and small water supplies have arsenic concentrations greater than 1 µg/L (Welch et al. 1999).

Inorganic arsenic (the form typically found in soil or water) is often in a form that is readily absorbed either by ingestion or by inhalation. Following absorption, it is distributed throughout the body. Studies with laboratory animals suggest that the bioavailability of arsenic in soil may be lower than that of arsenic ingested in solution. The issue of arsenic bioavailability is especially important at mining, milling, and smelting sites because the arsenic at these sites often exists, at least in part, as a poorly soluble sulfide and may also occur in particles of inert or insoluble material. These factors all tend to reduce the bioavailability of arsenic (See discussion for arsenic's gastrointestinal absorption factor in Section 3.3.3).

Arsenic is partly metabolized in the liver by methylation (the metabolic addition of methyl groups to inorganic arsenic ions), converting inorganic arsenic into methyl- and dimethylarsenic compounds. Absorbed organic and inorganic arsenic compounds are principally excreted in the urine. Methylation followed by urinary excretion has been considered a detoxification mechanism for inorganic arsenic. However, a recent study by Mass et al. (2001) found that methylated trivalent arsenic added to human

peripheral lymphocytes produced direct DNA damage. These findings indicate that biomethylation of absorbed inorganic arsenic is not solely a detoxification pathway (see also a further discussion in the uncertainty section).

Several organic arsenicals have been found to accumulate in fish and shellfish. These derivatives (mainly arsenobetaine and arsenocholine, also referred to as “fish arsenic”) have been studied by several researchers and have been found to be essentially nontoxic.

Arsenic at high levels of exposure is irritating to the gastrointestinal tract. Common symptoms in humans after acute high-dose ingestion of inorganic arsenic compounds are nausea, vomiting, and diarrhea. Signs of peripheral neuropathy have been noted in individuals who have ingested inorganic arsenic. The neuropathy is detected as numbness in the hands and feet, progressing to a painful “pins and needles” sensation. Acute lethality from arsenic ingestion is usually attributed to cardiopulmonary collapse.

Evidence of reproductive or developmental toxicity in humans is limited. However, a recent study (Hopenhayn-Rich et al.2000) found, in a retrospective analysis of a Chilean city with formerly high water arsenic levels, that there were significant associations between late fetal mortality rates, neonatal mortality rates, and postnatal mortality rates and the concurrent water arsenic concentrations. These data support a role for water arsenic levels increasing late fetal and infant mortality (Hopenhayn-Rich et al.2000). Studies in laboratory animals suggest that arsenic produces developmental toxicity (reduced birth weight, fetal malformations, and increased fetal mortality) at high levels of exposure. The data suggest that inorganic arsenic does not pose a significant risk of developmental toxicity except at levels that would cause toxic effects on the mother (i.e., maternally toxic doses) (Holson et al. 2000, ATSDR 1993).

Arsenic has been associated with adverse effects on human populations in different parts of the world, which were exposed to levels in drinking water exceeding 300 µg/L over a long period of time. Two recent studies (Kurtio et al. 1999, Chiou et al. 2001) found a statistically significant increased risk of bladder cancer at drinking water levels well below 300 µg/L at 0.5 µg/L and 10-50 µg/L for the Finnish and Taiwan studies, respectively (see also uncertainty section).

The distinguishing adverse effects associated with chronic ingestion of arsenic are skin lesions (hyperkeratoses and hyperpigmentation) and skin cancer. Other adverse effects due to ingestion exposure include cancer of the internal organs (prostate, liver, bladder, and kidney) and a vascular disease known as “blackfoot disease” (Blackfoot disease has been observed only in an area of Taiwan where there are naturally elevated arsenic concentrations in drinking water). Occupational exposure (principally copper smelter workers) has been associated with an increased incidence of lung cancer. The EPA has given arsenic a carcinogenicity weight-of-evidence classification of a Group-A (human carcinogen) based on sufficient evidence of cancer mortality from both ingestion and inhalation exposures in human populations. The International Agency for Research on Cancer classifies arsenic as a proven human carcinogen.

Some information about human populations that may be sensitive to arsenic exposure has been identified. Individuals with impaired liver function or poor nutritional status may not detoxify arsenic efficiently and may be at greater risk of adverse effects from arsenic exposure. In addition according to current data, children are sensitive to arsenic for two reasons. First, two studies have shown that children do not biomethylate arsenic as well as adults (although this may make them less sensitive to cancer effects noted in the recent Mass et al. study described above), i.e., they are at higher risk for noncancer effects and to some extent cancer effects from the higher net fraction of inorganic arsenic (Kurttio et al. 1998; Concha, Nermell, and Vahter 1998a). Second, there has been a recent finding that children appear to be more sensitive for response when one looks at biomarkers that are specific for certain carcinogens. Tang et al. (1999) reported that compared to adults, children have higher circulating levels of a key biomarker for carcinogenic substances from environmental tobacco smoke. Pregnant women have also been identified as a sensitive population. It has been shown that arsenic crosses the placental barrier (Concha et al. 1998b, NRC 1999), and in pregnant women exposed to arsenic, blood arsenic levels in the newborns are almost as high as the level in cord blood. Food and drinking water are the largest sources of arsenic exposure. Studies in laboratory animals suggest that low levels of dietary arsenic may be beneficial or essential. However, there is no known specific biochemical mechanism by which arsenic could exert a beneficial effect. If arsenic is beneficial to humans, then the daily requirement is probably met by normal dietary intake.

The EPA has promulgated a new MCL for arsenic based on recent epidemiological findings associating arsenic exposure with an increase in internal organ cancers. At the request of the EPA, the National Academy of Sciences (NAS) reviewed the current state of science for estimating risks associated with arsenic in drinking water. In its review, completed in 1999, the NAS recommended lowering the MCL from the current interim drinking water standard of 50 µg/L. This recommendation is based on NAS's assessments of the risks of skin, lung, and bladder cancer from drinking water containing inorganic arsenic (NRC 1999). The EPA published a final rule lowering the MCL from 50 µg/L to 10 µg/L (Federal Register 2001). In addition to information from the NRC's report, the EPA also considered a recent epidemiological study in Utah (Lewis et al. 1999) when proposing the new MCL. The Utah study found a significant increase in hypertensive heart disease among males and females, although no dose-response trend was noted between the low, medium, and high exposure groups for this disease end point. Cardiovascular effects of arsenic have been documented in a number of other studies at higher arsenic concentrations than the 4 Fg/L to 620 Fg/L reported in the Utah study (median exposure concentration <200 Fg/L). The Utah study also found a statistically significant increase in prostate cancer and nephritis/nephrosis among study males. Prostate cancer has not previously been associated with arsenic; however, other studies have noted kidney problems, see further discussion in the uncertainty section. While the Utah population is likely not representative of the United States population in general, the EPA considers this study to provide further weight to concerns about arsenic health effects in drinking water at concentrations below the current MCL. The Utah study is of particular interest because of the relatively low range of arsenic water concentrations in contrast to other epidemiologic studies which generally had average arsenic exposure in the several hundreds µg/L range. In addition, the Utah study suggested that cardiovascular effects can occur at lower levels than those seen in the studies reviewed by NRC. The Utah study is further discussed in section 7.

The oral RfD for arsenic is based on the occurrence of hyperpigmentation and hyperkeratosis and vascular complications observed in the Taiwanese population ingesting elevated levels of arsenic in drinking water. The NOAEL was calculated to be 0.0008 mg/kg-day. An uncertainty factor of 3 is applied to account for both (1) the lack of data to preclude reproductive toxicity as a critical effect, and (2) some uncertainty pertaining to whether the NOAEL of the critical study accounts for all sensitive individuals. The oral RfD for arsenic is 0.0003 mg/kg-day. According to the EPA, strong scientific arguments can be made for various values within a factor of 2 or 3 of the currently recommended RfD value, i.e., 0.0001 to 0.0008 mg/kg-day. An inhalation RfD or reference concentration (RfC) has not been estimated for arsenic (USEPA 2000a).

The oral unit risk factor for estimating excess lifetime cancer risks is based on the incidence of skin cancer observed in the Taiwanese population ingesting elevated levels of arsenic in drinking water. Doses were converted to equivalent doses for males and females in the United States based on differences in body weights and differences in water consumption. It was assumed that skin cancer risk in the U.S. population would be similar to that in the Taiwanese population. The maximum likelihood estimate (MLE) of skin cancer risk for a 70-kg person drinking 2 L of water per day ranged from 1×10^{-3} to 2×10^{-3} for an arsenic intake of 1 µg/kg-day. Expressed as a single value, the cancer unit risk for drinking water is 5×10^{-5} L/µg. Details of the assessment are in USEPA (1988) (USEPA 2000a). Using the assumptions of 2 L/day drinking water consumption and 70-kg body weight, this unit risk factor converts to an oral SF of 1.5 $(\text{mg/kg-day})^{-1}$. It should be noted that the EPA's assessment is based on Taiwanese data on the prevalence of skin cancer from the IRIS database. However, arsenic has also been associated with internal organ cancers, particularly lung and bladder cancer (NRC 1999, Federal Register 2001). Recent epidemiological data from South America indicate that risks based on fatal internal cancer could be an order of magnitude higher than risks based on skin cancer. Thus, risks calculated from IRIS could be underestimated. See Section 7 for a more detailed discussion. NRC 1999 estimated that the combined risk for bladder and lung cancer could be as high as 1 in 100 at arsenic's previous MCL of 50 ppb.

4.3.3 Cadmium

Cadmium is obtained mainly as a by-product during the processing of zinc-bearing ores and also from the refining of lead and copper from sulfide ores. Cadmium is used primarily for the production of nickel-cadmium batteries, in metal plating, and for the production of pigments, plastics, synthetics and metallic alloys. Cadmium has been shown to be toxic to human populations from occupational inhalation exposure and accidental ingestion of cadmium-contaminated food. Inhalation of cadmium dust in certain occupational settings may be associated with an increased incidence of lung cancer. Ingestion of elevated levels of cadmium has resulted in toxicity to the kidney and skeletal system and may be associated with an elevated incidence of hypertension and cardiovascular disease.

Cadmium is poorly absorbed from the lung, gastrointestinal tract, and skin. Individuals with dietary deficiencies of iron, calcium, or protein exhibit higher absorption of ingested cadmium. The issue of cadmium bioavailability is especially important at mining, milling, and smelting sites because the

cadmium at these sites often exists, at least in part, as a poorly soluble sulfide and may also occur in particles of inert or insoluble material. These factors all tend to reduce the bioavailability of cadmium in soil. Cadmium in the body binds readily to certain sulfur-containing proteins, such as metallothionein. Binding to metallothionein is thought to reduce the toxicity of cadmium. Following ingestion, fecal excretion is high due to poor gastrointestinal absorption. Most cadmium that has been absorbed, however, is excreted very slowly, with fecal and urinary excretion being about equal. Urinary cadmium levels are an indicator of body burden, i.e., chronic exposure.

Much of the understanding about cadmium toxicity in humans is based on epidemiological studies of human populations. Humans consuming cadmium-contaminated rice in Japan developed kidney and skeletal system effects. Inhalation of cadmium in occupational settings has also been associated with kidney toxicity. There is conflicting evidence as to whether or not cadmium exposure produces cardiovascular effects or hypertension in humans; factors such as cigarette smoking are confounders in determining the relationship between cadmium exposure and cardiovascular effects. Excessive cadmium ingestion exposure in combination with a low dietary intake of iron may be associated with anemia.

Ingested cadmium is not known to be carcinogenic in humans. Studies in laboratory animals generally do not indicate that cadmium is carcinogenic by ingestion. Inhaled cadmium is carcinogenic to laboratory animals. However, epidemiological studies of cadmium-exposed workers have been inconclusive in demonstrating the carcinogenicity of inhaled cadmium. The EPA has classified cadmium as a probable human carcinogen by inhalation (Group B1) based on limited evidence in humans and sufficient evidence in laboratory animals.

Populations potentially sensitive to cadmium have not been studied systematically; however, it is possible to infer potential sensitivities based on the available data. Individuals with poor nutritional status, particularly in terms of iron and calcium, may absorb more cadmium from the gastrointestinal tract. Individuals with preexisting kidney damage may experience kidney toxicity at cadmium doses lower than the dose that would be toxic for normal individuals.

The EPA recently conducted a toxicological review of cadmium and compounds in support of a proposed revision of the toxicity factors currently listed in IRIS. However, the report is currently undergoing external review and the proposed toxicity factors have not been finalized.

The current EPA recommendation consists of two oral RfDs for cadmium, one for cadmium exposure from food and one for cadmium exposure from water. Both RfDs recognize that a concentration of 200 µg/g (wet weight) in the human kidney cortex is the highest renal level not associated with significant proteinuria. A toxicokinetic model was used by the EPA to determine the level of chronic human oral exposure (NOAEL) that results in the critical concentration of cadmium in the kidney of 200 µg/g; the model assumes that 0.01 percent of the cadmium body burden is eliminated per day (USEPA 1985). Assuming 2.5 percent absorption of cadmium from food or 5 percent from water, the toxicokinetic model predicts that the NOAEL for chronic cadmium exposure is 0.005 and 0.01 mg/kg-

day from water and food, respectively (i.e., the doses corresponding to the 200 µg/g critical kidney concentration). An uncertainty factor of 10 to account for intrahuman variability was applied to these NOAELs to obtain an RfD of 0.0005 mg/kg-day (water) and an RfD of 0.001 mg/kg-day (food) (USEPA 2000a). No inhalation RfD or RfC is currently listed for cadmium. A dermal RfD of 0.001 mg/kg-day multiplied by 2.5 percent (0.000025 mg/kg-day) was selected for use in the calculations.

The critical toxic effect proposed for both the oral RfD and inhalation RfC is renal dysfunction, as indicated by minimal proteinuria/enzymuria. This critical effect is supported by the results of several cross-sectional population studies, especially the CadmiBel population study of Buchet et al. (1990). The CadmiBel study authors (Lauwerys et al., 1993) found that the critical kidney cortex level of cadmium in the general population was 50 ppm, four-fold lower than that found in mainly healthy workers, 200 ppm. This difference is not unexpected, in that general population data include more of a range of inter-individual health status including those with poor health. A toxicokinetic model was used with the data in this study to calculate both a daily oral intake and a continuous air concentration of cadmium that would result in a 10 percent occurrence of minimal enzymuria (the critical effect) in the population at the age of 70. A representative level of dietary cadmium intake was integrated into the toxicokinetic model. The net oral intake (model result minus diet) of 0.0007 mg/kg-day was designated the oral RfD. USEPA (1999f) has proposed that one RfD be used for oral exposures to all media (i.e., separate RfDs were not proposed for ingestion of cadmium in food or water). The modeled concentration of cadmium inhaled concomitant with this same representative dietary intake was designated as the inhalation RfC of 0.0007 mg/m^3. For both the RfD and the RfC, alternate contributions of intake from background (and therefore different RfDs and RfCs) are described in EPA's toxicological review (USEPA 1999f).

4.3.4 Iron

Iron is a major constituent in rocks and soil. In combination with carbon, manganese, chromium, nickel, and other elements, it is used in the manufacture of steel. Iron is an essential element in human nutrition; however, there is the potential for adverse health effects principally from excessive ingestion exposure.

The absorption of iron and its distribution in the body are closely regulated to maintain homeostasis. Absorption of iron from the diet ranges between 2 and 15 percent, with increased absorption during times of greater need, such as childhood, pregnancy, or following blood loss. Iron is found mostly in hemoglobin in red blood cells; however, it can also be stored in the liver and spleen. Excretion of iron from the body is fairly limited. Excess iron is bound to proteins and stored primarily in the liver.

The issue of iron bioavailability is especially important when considering soil exposure pathways because iron in soil can exist, at least in part, as poorly soluble salts and may also occur in particles of inert or insoluble material. These factors all tend to reduce the bioavailability of iron.

Severe acute toxicity has resulted from the accidental ingestion of iron-containing medications, principally by children eating ferrous sulfate tablets with candy-like coatings. Signs of overexposure include ulceration of the gastrointestinal tract with vomiting (including blood), black stools, damage to the liver and kidneys, and metabolic acidosis. Death from exposure to iron is thought to occur from renal failure and cirrhosis of the liver.

Chronic overexposure (also known as iron overload) may occur as a result of excessive dietary consumption of iron or from a condition known as idiopathic hemochromatosis. Chronic overexposure results in excess iron accumulation in the liver, spleen, pancreas, endocrine organs, and the heart. Adverse effects may include disturbance of liver function, diabetes mellitus, disturbance of endocrine function, and cardiovascular effects. On a cellular level, increased lipid peroxidation occurs, resulting in damage to the membranes of cell organelles. Although there are no known sensitive populations for exposure to iron, idiopathic hemochromatosis is thought to have a genetic component.

Years of inhalation of iron oxide fumes or dust causes a benign pneumoconiosis in miners and metal workers referred to as siderosis, which generally does not result in reduced pulmonary function. An increased incidence of lung cancer has been observed among hematite miners and iron workers who have been exposed to iron oxide. However, there may be other factors to explain the observed cancer incidence, including exposure to other carcinogens such as cigarette smoke, radon, and polycyclic aromatic hydrocarbons, or exposure to silica dust.

While iron generally is not considered to be carcinogenic or mutagenic, excess iron can result in lipid peroxidation, which may result in genotoxic effects, such as damage to DNA or chromosomes. In studies with laboratory animals, iron overload may potentiate the effects of other carcinogens. Elevated exposure to iron is not considered to be associated with reproductive or developmental toxicity.

The EPA's IRIS database does not currently provide an RfD, cancer SF, or other toxicological information for iron (USEPA 2000a). The EPA Superfund Technical Support Center has developed a provisional oral RfD for iron. The EPA notes that iron is an essential nutrient and that deriving a risk assessment value for it poses special problems in that the dose-response curve is U-shaped (i.e., there is a range of doses necessary to maintain health; doses both above and below that range can result in adverse effects). Thus, the provisional RfD must be protective against deficiency as well as toxicity. A NOAEL for chronic iron overload has been estimated using the values for dietary intake and iron status indices taken from the NHANES II database (USEPA 1999e). Looker et al. (1988) compared dietary iron intakes with biochemical indices of iron status using data from NHANES II. The average intakes of iron ranged from 0.15 to 0.27 mg/kg-day. The serum ferritin levels and percent serum transferritin saturation (both indicators of iron overload) were within the normal range. Thus, iron intake levels of 0.15 to 0.27 mg/kg-day are considered both sufficient to protect against iron deficiency and insufficient to cause the toxic effects of iron overload.

Using the NOAEL of 0.27 mg/kg-day (representing the upper bound value in the range of mean dietary iron intakes, dietary plus supplemental, taken from the NHANES II database) and dividing by an

uncertainty factor of 1 yields the provisional chronic oral RfD of 0.3 mg/kg-day. An uncertainty factor of 1 is supported by the fact that iron is an essential nutrient. In addition, the oral RfD for iron was derived from intake data from over 20,000 individuals aged 6 months to 74 years and humans exert an efficient homeostatic control over iron such that body burdens are kept constant with normal variations in diet. This RfD supplies adequate levels of iron to meet the nutritional requirements of adults and adolescents. It does not supply the recommended dietary allowance (RDA) for members of the population with greater requirements for shorter-than-lifetime durations, including children and pregnant women. Further, this RfD may not be protective of individuals with inherited disorders of iron metabolism and could be conservative if applied to exposure scenarios involving forms of iron with low bioavailability (USEPA 1999e).

4.3.5 Lead

Lead is a soft, bluish-gray metal. Lead acetate and lead nitrate are soluble in water; lead chloride is slightly soluble; and lead sulfide, lead phosphate, and lead oxides are not soluble in water. Some primary uses of lead in the United States are in lead-acid storage batteries, ammunition, bearing metals, brass, bronze, cable covering, extruded products, sheet lead, solder, ceramics, type metal, ballast or weights, tubes or containers, oxides, and gasoline additives.

Substantial quantities of both human and animal data are available regarding the toxicity of lead. This toxicity profile relies primarily on human data. Adverse effects of lead in humans are most often related to the blood lead level as an indicator of internal lead dose. Therefore, whenever possible, this text relates adverse effects to blood lead levels rather than to external exposure. The Centers for Disease Control (CDC) have based policy on primary and secondary childhood lead prevention activities on the association of certain adverse health effects with different blood lead levels. Tables 4-2 and 4-3 summarize the lowest-observed-effect levels (LOELs) (expressed as PbB levels) for key lead health effects in children and adults, based on information in NRC and CDC (1993;1991). Section 6.2.1 describe the CDC primary and secondary prevention guidelines regarding childhood blood lead levels.

Lead absorption is influenced by the route of exposure, the exposure medium, speciation and physiochemical characteristics of lead, and the age and physiological state of the exposed individual. Approximately 30 to 50 percent of airborne particulate lead is absorbed. Children 2 weeks to 8 years of age absorb about 40 to 50 percent of ingested lead. A study using Bunker Hill soils found nonfasted adults absorbed 2.5 percent of lead ingested in soil and fasted adults absorbed 26.2% of lead ingested in soil (Maddaloni et al. 1998). The amount of lead absorbed from the skin in humans is unknown.

Lead is absorbed into blood, where about 99 percent of it is located in red blood cells. Lead in blood rapidly exchanges with lead in other soft tissues. Bone contains about 94 percent and 73 percent of total lead body burden in adults and children, respectively. The average half-life for lead is 28 to 36 days in blood, about 40 days in soft tissues, and about 27 years in bone. Lead in bone can be mobilized into maternal blood during pregnancy and lactation. Lead in maternal blood is efficiently transported to the fetus, and breast milk can be a significant source of lead for breast-feeding infants.

Lead in the gastrointestinal tract that is not absorbed is eliminated in the feces. Absorbed lead that is not retained is eliminated in the urine or excreted in the feces following biliary secretion into the gastrointestinal tract.

Death from encephalopathy has been reported in children and adults with very high blood lead levels (e.g., 80-100 Fg/dl). There is conflicting evidence in occupational mortality studies of chronic lead exposure. IQ decrements, fine-motor dysfunction, altered behavior, peripheral neuropathy, and reduced motor nerve conduction have been reported in children. A threshold below which lead does not affect IQ in children has not been identified. Decreased hearing thresholds and alterations in the electrical activity of the brain have also been observed in children. Lead can also induce neurotoxicity in adults, including encephalopathy, overt neurological signs, decreased scores on neurobehavioral tests, and decreased motor nerve conduction.

Lead interferes with heme synthesis. Reduction of the heme body pool can lead to adverse effects in several physiological systems. Anemia can result from decreased hemoglobin production and increased red blood cell destruction. Lead-induced inhibition of heme synthesis can interfere with the conversion of vitamin D to its hormonal form, 1,25-dihydroxyvitamin D. There is no apparent threshold for indicators of decreased heme synthesis.

Acute, generally reversible, nephropathy can occur during the early stages of high exposure to lead. Chronic (irreversible) nephropathy can also occur. Acute exposures to high levels of lead can produce cardiac lesions, electrocardiographic abnormalities, and hemolytic anemia in children and adults. There is conflicting evidence regarding the potential effects of blood lead levels on blood pressure in adults. Colic is a relatively late symptom of severe or clinical lead poisoning generally observed at blood lead levels greater than 50 Fg/dl.

Women with occupational exposures to lead during pregnancy have an increased rate of miscarriages and stillbirths. There is no evidence of teratogenic effects in humans or animals due to exposure to low levels of lead. There is conflicting information regarding the potential effects of lead on birth weight, gestational age, and growth in children. There is conflicting evidence regarding the potential effects of lead on human chromosomes. In men with occupational exposures some reproductive effects (e.g., decreased sperm count, abnormal sperm morphology, decreased sperm mobility, and hormonal changes) can occur at blood lead levels of 40 Fg/dl or greater.

Although lead is considered to be carcinogenic in animals with the endpoint being renal cancer, evidence of its carcinogenicity in humans is generally considered to be inadequate. EPA's IRIS database classifies lead as a probable human carcinogen (Group B2), based on sufficient evidence in animals, but inadequate evidence in humans. Lead carcinogenicity will not be evaluated quantitatively in this risk assessment.

Sensitive members of the population can include developing embryos/fetuses/neonates, young children, women, and individuals with chronic neurological dysfunction or kidney disease. Older adults are at

risk for lead-associated hypertension (NRC 1993). The embryo/fetus/neonate may be at increased risk due to the effects of lead because of a developing nervous system that is more sensitive to the effects of lead and the transfer of maternal lead during pregnancy and lactation. Young children may be especially at risk because compared to adults they absorb more lead from the gastrointestinal tract; retain more absorbed lead; have a greater prevalence of nutritional deficiencies (e.g., calcium, iron, and zinc), which can increase both the absorption and the toxic effects of lead; have an incompletely developed blood-brain barrier; have a developing nervous system that is more sensitive to the effects of lead; ingest much more soil/dust per kg body weight, ingest more water per kg body weight; and inhale more air per kg body weight. Women who are pregnant, are lactating, or have osteoporosis may be themselves at greater risk due to lead because each of these conditions may intensify the mobilization of lead from bone.

Blood lead level is the easiest and most widely used index of lead exposure and toxicity. Blood lead primarily reflects recent exposure for lead but can also reflect, to a lesser extent, the body burden of lead, which is more related to long-term exposure. For children and fetuses, 10 µg/dl is generally considered a blood lead level of concern (CDC 1997; CDC 1991). There is less agreement on a single blood lead level of concern for male adults and nonpregnant female adults, but estimates fall within the range of 25 to 40 µg/dl. However, analysis of U.S. NHANES II epidemiological data (NAS 1993) shows hypertensive effects in the form of elevated systolic and diastolic blood pressure in older adults at blood lead values well below this range.

A number of pharmacokinetic models for lead are available to predict blood lead levels based on lead intake in various exposure media (USEPA 1994a, 1996c; CalEPA 1992, 1996; O'Flaherty 1998; Leggett 1993; Bowers, Beck, and Karam 1994; ATSDR 1999b). The EPA models (USEPA 1994a, 1996c) are typically used at Superfund sites to evaluate risk to adults or children from exposure to environmental lead.

The toxic effects of lead are generally considered to be similar regardless of the route of entry. Most adverse effects of lead have been related to lead in blood and (to a lesser extent) tooth dentin (Tables 4-2 and 4-3). There are relatively few data relating human health effects to exposure-route specific external exposure (e.g., mg/kg-day or m^3/day).

Ingestion is the primary route of exposure for children and other non-occupationally exposed individuals. However, dose-response data based on external ingestion dose (mg/kg-day) in humans were limited. Hematological effects were observed in adult humans who ingested 0.02 to 0.03 mg lead acetate/kg-day for 14 days or 0.01 to 0.02 mg lead acetate/kg-day for 3 to 7 weeks.

Inhalation is an important route of exposure for adults at work. However, very little dose-response data in workers using lead air concentrations (mg/m^3) were located. A 47 percent decrease in ALAD activity was observed in men inhaling lead at a concentration of 0.011 mg/m^3 for 18 weeks.

ATSDR (1999b) reported that no studies were located regarding toxicity of lead in humans or animals specifically from dermal exposure. Dermally applied lead nitrate is rapidly absorbed by the skin, but the toxicology significance is unknown.

4.3.6 Manganese

Manganese is an essential element in human nutrition, serving as a cofactor in several enzymatic reactions. When ingested, manganese is considered to be among the least toxic of the trace elements. The adverse health effects from manganese are principally associated with inhalation exposure in the workplace. Acute inhalation exposure can produce irritation of the respiratory tract. Chronic inhalation exposure can produce a central nervous system disorder resembling Parkinsonism, known as manganism.

Daily intake of manganese ranges from 2 to 9 mg/day. Manganese is poorly absorbed following oral exposure, and reports of human intoxication following ingestion exposures are not common. However, some studies suggest that neurological effects may be associated with the consumption of drinking water with elevated levels of manganese. Although ingestion exposure studies suggest that manganese may be weakly carcinogenic in laboratory animals, these data are inadequate to support a classification as carcinogenic by the EPA. The EPA has categorized manganese as “not classifiable with regard to human carcinogenicity” (Group D).

Several studies have shown that inhalation of manganese in occupational settings is associated with neurological effects. The principal signs of manganism include tremors, weakness in the legs, staggering gait, behavioral disorders, slurred speech, and a fixed facial expression. There is no evidence indicating that inhalation exposure to manganese is carcinogenic in humans; however, there is some evidence of male reproductive effects.

Development of the oral RfD for manganese recognizes that disease states in humans have been associated with both deficiencies and excessive intakes of manganese. The oral RfD for manganese is set at 10 mg/day (0.14 mg/kg-day) and is based on the upper end of the normal dietary intake rate. This value is considered a NOAEL for dietary intake and has not been adjusted by an uncertainty factor. The EPA emphasizes that individual requirements for, as well as adverse reactions to, manganese may be highly variable. The RfD is estimated to be an intake for the general population that is not associated with adverse health effects; this is not meant to imply that intakes above the RfD are necessarily associated with toxicity (USEPA 2000a).

The oral RfD for manganese was evaluated further in other media (drinking water or soil) based on an epidemiological study of manganese in drinking water (USEPA 2000a). Whereas the results from this study do not allow a quantitative evaluation of dose-response, they raise concerns about possible adverse neurological effects at doses not far from the range of essential concentrations. For assessing exposure to manganese from drinking water or soil, USEPA (2000a) recommends adjustment by an uncertainty factor of 3, yielding an oral RfD of 0.047 mg/kg-day. Four reasons are provided for the

use of an uncertainty factor to adjust the oral RfD for soil and water exposure: (1) in fasted individuals, there may be increased uptake of manganese from water; (2) the study raises some concern regarding possible adverse health effects associated with a lifetime consumption of drinking water with manganese concentration of about 2 mg/L; (3) because infant formula typically has a much higher concentration of manganese than that of human milk, manganese in the water could represent an additional source of intake for infants; and (4) neonates may absorb more manganese from the gastrointestinal tract and may be less able to excrete absorbed manganese, and more absorbed manganese may cross their blood-brain barrier.

For this HHRA, an oral RfD of 0.14 mg/kg-day was used to evaluate occupational exposures to manganese in soil. For all other manganese exposures, an oral RfD of 0.047 mg/kg-day was used.

The oral RfDs of 0.047 to 0.14 mg/kg-day and the inhalation RfD of 0.000014 mg/kg-day for manganese (USEPA 2000a) suggest that inhaled manganese may be much more toxic than ingested manganese. Differences in absorption between the two routes cannot alone account for this large difference. The EPA reports that after absorption into blood via the respiratory tract, manganese is transported through the blood stream directly to the brain, bypassing the initial clearance effects of the liver. They state that this pathway from the respiratory tract to the brain is the primary reason for the differential toxicity between inhaled and ingested manganese. In addition, recent studies in animals have shown that manganese has a unique ability among metals to be taken up in the brain via olfactory pathways (Tjalve and Henriksson 1997). This process involves direct diffusion of manganese from the nasal cavity to the central nervous system without entering blood, therefore bypassing both the initial clearance effects of the liver and the blood-brain barrier (Tjalve and Henriksson 1997). This direct pathway to the central nervous system might account in part for the higher toxicity of inhaled manganese.

4.3.7 Mercury

Elemental mercury is a silvery metallic liquid that is volatile at room temperature. Mercury is found in soil and rocks typically as an ore known as cinnabar, consisting of insoluble mercuric sulfide. Concentrations in soil and rock average 0.5 mg/kg, though actual concentrations vary considerably depending upon location. Much of the mercury produced in the United States comes from secondary sources, such as recycling. The largest use of mercury is in the electrolytic production of chlorine and caustic soda. Other uses include electrical devices, switches and batteries, measuring and control instruments, medical and dental applications, and electric lighting.

Mercury has been shown to be toxic to human populations as a result of occupational exposure and accidental ingestion of mercury-contaminated food. The nature of mercury toxicity depends on its chemical form. Accidental ingestion exposure to high levels of organic mercury compounds has produced developmental toxicity in humans.

Ingestion of inorganic mercury, the form most likely to be found in soil, has been associated with kidney toxicity in laboratory animals. The adverse effect of concern associated with soil exposure scenarios, therefore, is likely to be kidney toxicity. Ingestion studies with inorganic mercury suggest cancer effects in laboratory animals. The EPA has classified mercuric chloride and methylmercury as possible human carcinogens (Group C), based on the absence of data in humans and limited evidence of carcinogenicity in animals.

The issue of mercury bioavailability is especially important at mining, milling, and smelting sites because the mercury at these sites often exists, at least in part, as a poorly soluble sulfide and may also occur in particles of inert or insoluble material. These factors all tend to reduce the bioavailability of mercury from soil.

Occupational inhalation exposure to metallic mercury vapor or organic mercury vapor has resulted in neurological effects and kidney toxicity. Toxicity due to inhalation of inorganic mercury salts, the form most likely to be found in soil, has not been studied.

Children are considered a sensitive population for exposure to mercury. Potential differences in sensitivity between children and adults are primarily due to differences in routes of exposure and rates of intake (for example exposure of infants via ingestion of breast milk), greater permeability of the blood-brain barrier in fetuses and infants, and the importance of developmental milestones during childhood exposure periods (such as language or cognitive development). Children also appear to have different patterns of tissue distribution of mercury and methylmercury (i.e., biokinetic patterns) that are different from those of adults.

More recently, the EPA has developed the Mercury Research Strategy to address key scientific questions in order to reduce uncertainties currently limiting its ability to assess and manage mercury and methylmercury risks. This strategy will include evaluations to link toxicity to exposure using a biokinetic model, assessment of sensitive populations, evaluation of recent epidemiological studies, and evaluation of immunological effects.

The EPA has published chronic oral RfDs for mercuric chloride and methyl mercury on its IRIS database (USEPA 2000a). The most sensitive adverse effect for mercuric chloride is reported to be the formation of mercury-induced autoimmune glomerulonephritis. Based on weight of evidence from three subchronic feeding and/or subcutaneous studies in rats, the oral RfD for mercuric chloride is 0.0003 mg/kg-day. All treatment groups exhibited a toxic effect; therefore, a NOAEL was not reported. An uncertainty factor of 1,000 was applied for extrapolations from LOAEL to NOAEL endpoints, subchronic to chronic exposures, and animal to human populations. The EPA reported a high confidence in the oral RfD for mercuric chloride. A subchronic oral RfD of 0.003 mg/kg-day is provided in the Health Effects Assessment Summary Tables (HEAST) for mercuric chloride, based on autoimmune effects observed in rats after subcutaneous injection (USEPA 1997c).

EPA's chronic oral RfD for methyl mercury of 0.0001 mg/kg-day was used to evaluate exposures to mercury in fish (USEPA 2000a). Methyl mercury can be more toxic than mercuric chloride and is likely to be present in fish tissue. Exposures to mercury in all other media were evaluated using the oral RfD for mercuric chloride. Methyl mercury's oral RfD is based on developmental neurologic abnormalities in human infants as determined by epidemiologic studies. An uncertainty factor of 10 has been assigned to this RfD and EPA's confidence in this RfD is medium. A committee of the NAS (NRC 2000) has recently reported its analyses of current human and experimental animal data for methylmercury and has also, as a result, endorsed EPA's methylmercury RfD value of 0.1 µg/dl in its report "Toxicological Effects of Methylmercury" (NRC 2000).

No cancer SFs have been developed for mercury compounds. However, the EPA has classified both mercuric chloride and methylmercury as possible human carcinogens (Group C), based on the absence of data in humans and limited evidence of carcinogenicity in animals, whereas elemental mercury is in Group D (not classifiable due to inadequate data) (USEPA 2000a).

4.3.8 Zinc

Zinc is used in a wide variety of industrial, agricultural, and consumer products. It is found in all human tissues and all body fluids and is essential for growth, development and reproduction. The RDA for zinc is 15 mg, with a slightly higher requirement for pregnant women. Individuals with adequate nutritional levels of zinc absorb approximately 20 to 30 percent of all ingested zinc.

Zinc is usually present in tap water at concentrations less than 0.2 mg/L, although drinking water in galvanized pipes can contain up to 2 to 5 mg/L. Typically, concentrations are much less than the secondary MCL of 5 mg/L, which is based on the threshold for metallic taste in water. An estimate of daily intake of zinc for the adult U.S. population in food is 10 to 20 mg/day.

Gastrointestinal distress is a common symptom following acute oral exposure to zinc compounds. Accidental poisonings have occurred as a result of the use of zinc supplements and from food contamination caused by the use of zinc-galvanized containers. Symptoms develop within 24 hours and include nausea, vomiting, diarrhea, and abdominal cramps. Anemia also may occur in severe cases of acute exposure or in high-dose exposures of longer duration. Inhalation exposure to high concentrations of some zinc compounds (zinc oxide fume) has been associated with "metal fume fever." Attacks of metal fume fever are characterized by chills and fever, weakness, and sweating. Recovery usually occurs within 24 to 48 hours. Zinc chloride, a corrosive inorganic salt, is more damaging to the respiratory tract than zinc oxide. Zinc chloride is a primary ingredient in smoke bombs, and serious respiratory injury has been reported to result from accidental inhalation of smoke from these bombs.

Developmental or reproductive toxicity has been reported in laboratory animals with relatively high levels of exposure to zinc. There is only one unconfirmed report documenting adverse reproductive effects in pregnant women provided zinc supplementation. Other studies in humans conclude there have been no adverse reproductive or developmental effects from exposure to zinc. Genotoxicity

studies have provided very limited evidence of mutagenicity and of weak effects on chromosomes. Available epidemiological studies of human populations and toxicity studies in laboratory animals do not indicate that zinc is carcinogenic. The EPA has given zinc a carcinogenicity weight-of-evidence classification of D (not classifiable as to human carcinogenicity), based on inadequate evidence in humans and laboratory animals.

Zinc interacts with other trace metals and has a protective effect against toxicity from exposure to lead and cadmium. Excessive dietary zinc produces a copper deficiency in laboratory animals. Similar findings have been observed in humans receiving long-term treatment with zinc. No specific data regarding human populations that are unusually susceptible to the toxic effects of zinc have been identified; however, individuals who are malnourished or have a marginal copper status may be more susceptible to the effects of excessive zinc exposure.

The oral RfD is based on a clinical study that investigated the effects of oral zinc supplements on copper and iron balance. A 10-week study of zinc supplementation in 18 healthy women given zinc gluconate supplements twice daily (50 mg zinc/day, or 1.0 mg/kg-day) resulted in a decrease in erythrocyte superoxide dismutase activity. There was a general decline in the mean serum high-density lipoprotein (HDL)-cholesterol in a higher-dose group (receiving 75 mg/day). The EPA has reported that while it is not absolutely certain that the zinc supplementation of 50 mg/day (1.0 mg/kg-day) represents a clearly biologically significant endpoint, this level, when viewed collectively with other studies investigating effects on HDL-cholesterol, may signify the beginning of the dose-response trend (USEPA 2000a). The significance of this change is unknown in light of an absence of increase in low-density lipoproteins (LDLs). An intake of 1.0 mg/kg-day was identified as LOAEL for zinc effects. An uncertainty factor of 3 was used, based on a minimal LOAEL from a moderate-duration study of the most sensitive humans and consideration of a substance that is an essential dietary nutrient. The oral RfD for zinc is 0.3 mg/kg-day (USEPA 2000a).

An RfC or inhalation RfD has not been developed for zinc (USEPA 2000a).

Table 4-1
Oral Toxicity Criteria

Chemical	Cancer: Slope Factor $(mg/kg\text{-}day)^{-1}$	Noncancer: Reference Dose (mg/kg-day)	Toxicity Endpoint	Uncertainty Factor/Level of Confidence[a]	Reference
Antimony	None	0.0004	Reduced lifespan, altered cholesterol levels	1,000/Low confidence	USEPA 2000a
Arsenic	1.5 EPA Group A carcinogen[c]	0.0003	Skin cancer (SF), hyper- pigmentation and hyperkeratosis of the skin (RfD)	3/Medium confidence	USEPA 2000a
Cadmium	None	0.001 (Food); 0.0005 (water); 0.000025 (dermal)	Kidney proteinuria	10/High confidence	USEPA 2000a
Lead[b]	None	None	Neurological effects	None	See text
Iron	None	0.3	Hematological effects	Not rated	USEPA 1999e
Manganese	None	0.14 (Soil/food); 0.047 (water)	Central nervous system effects	3/Medium confidence	USEPA 2000a
Mercury	None	0.0003	Kidney damage	1,000/High confidence	USEPA 2000a
Methyl-mercury	None	0.0001	Prenatal developmental effects	10/Medium confidence	USEPA 2000
Zinc	None	0.3	Anemia	3/Medium confidence	USEPA 2000a

[a]Applies only to reference doses.
[b]Toxicity criteria not applicable for lead; see text Section 4.3.5.
[c]EPA's Weight-of-Evidence Classification System:

Group A - human carcinogen (sufficient evidence in humans)
Group B1 - probable human carcinogen (limited human data available)
Group B2 - probable human carcinogen (sufficient evidence in animals, inadequate or no evidence in humans)
Group C - possible human carcinogen (limited evidence in animals)

Notes:
RfD - reference dose
SF - slope factor

Table 4-2
Lowest-Observed-Effect Levels in Children Exposed to Lead

Blood Lead LOEL (µg/dl)	Neurologic Effects	Heme-Synthesis Effects	Other Effects
< 10* - 15	Deficits in neurocognitive and neurobehavioral development, electrophysiologic changes, and lower IQ	ALA-D inhibition	Reduced gestational age and birthweight; reduced size up to age 7 to 8 years
15 - 20		Erythrocyte protoporphyrin increase	Impaired vitamin D metabolism, pyrimide-5'-nucleotidase inhibition
< 25	Longer reaction time	Reduced hematocrit	
30	Slower nerve conduction		
40		Increasing CP-U and ALA-U	
70	Peripheral neuropathies	Frank anemia	
80 - 100	Encephalopathy		Colic, other gastrointestinal effects, kidney effects

Note: LOEL - lowest-observed-effect level
Source: NRC 1993.
*10 µg/dl is not a NOEL - no-observed-effect level

Table 4-3
Lowest-Observed-Effect Levels in Adults Exposed to Lead

Blood Lead LOEL (µg/dl)	Heme-Synthesis Effects	Neurologic Effects	Renal Effects	Reproductive Effects	Cardiovascular Effects
< 10	ALA-D inhibition				
10 - 15					Increased blood pressure
15 - 20	Erythrocyte protoporphyrin increase in females				
25 - 30	Erythrocyte protoporphyrin increase in males				
40	Increasing CP-U and ALA-U	Peripheral nerve dysfunction (slower nerve conduction)			
50				Altered testicular function	
60				Female reproductive effects	
80	Frank anemia				
100 - 120		Encephalopathic signs and symptoms	Chronic nephropathy		

Note: LOEL - lowest-observed-effect level
Source: NRC 1993.

5.0 RISK CHARACTERIZATION FOR NON-LEAD CHEMICALS

Risk characterization is the summarizing step of risk assessment (USEPA 1995d). In the risk characterization, the toxicity values (RfDs and SFs) are applied in conjunction with the concentrations of COPCs and intake assumptions to estimate cancer risks and health hazards other than cancer.

Noncancer health hazards and cancer risk were calculated for both the reasonable maximum exposure (RME) and central tendency (CT) exposure conditions. RME hazard/risk estimates are based on the maximum exposure that is reasonably expected to occur at a site. Intake parameter values were selected so that the combination of all parameters resulted in an estimate of the RME for a particular exposure pathway. By design, the estimated RME is higher than that expected to be experienced by most of the exposed population. As recommended in EPA's *Guidance for Risk Characterization* (USEPA 1995d), CT exposure estimates reflect the central estimates of exposure or dose. The CT exposure estimate is intended to be more representative of average exposures. The purpose of evaluating both CT and RME estimates of exposure is to attempt to bound the true exposure for a particular pathway.

5.1 METHODOLOGY FOR ASSESSING NONCANCER HAZARD

The potential for adverse health effects other than cancer (noncancer effects) was characterized by dividing estimated chemical intakes (Appendix A, Table 7 through 10 series) by chemical-specific RfDs (Appendix A, Table 5 series). The resulting ratio is the hazard quotient (HQ), derived as follows:

$$HQ = \frac{\text{Chemical Intake (mg/kg-day)}}{\text{RfD (mg/kg-day)}}$$

Use of the RfD assumes that there is a level of intake (the RfD) below which it is unlikely that even sensitive individuals (e.g., senior citizens and children), will experience adverse health effects over a lifetime of exposure. EPA Region 10 recommends that, if available, the child-specific RfD be used when calculating child hazards. Otherwise, use of the chronic RfD along with the average child chemical intake is appropriate (USEPA 1999g). If the average daily intake exceeds the RfD (that is, if the HQ exceeds 1), there may be cause for concern regarding noncancer effects (USEPA 1989).

The EPA risk assessment guidelines (USEPA 1989) consider the additive effects associated with simultaneous exposure to several chemicals by first specifying that all hazard quotients be summed across exposure pathways and chemicals to estimate the total hazard index (HI). This summation conservatively assumes that the toxic effects of all chemicals would be additive, or in other words, that all chemicals cause the same toxic effect and act by the same mechanism (USEPA 1986).

If the total hazard index is less than or equal to 1, multiple-pathway exposures to COPCs at the site are considered unlikely to result in an adverse effect. If the total hazard index is greater than 1, further evaluation of exposure assumptions and toxicity, including consideration of the specific affected target

organs and the mechanisms of toxic actions of COPCs, is warranted to ascertain whether the cumulative exposure would in fact be likely to harm exposed individuals.

5.2 METHODOLOGY FOR ASSESSING CANCER RISK

The potential for carcinogenic effects is evaluated by estimating the probability of developing cancer over a lifetime based on exposure assumptions and chemical specific toxicity criteria. The increased likelihood of cancer due to exposure to a particular chemical is defined as the excess cancer risk (i.e., in excess of a background cancer risk of 1 in 100 or 1×10^{-2}). Excess lifetime cancer risk is estimated by multiplying the estimated chemical intake by the cancer SF, as follows:

$$\text{Cancer Risk} = \text{Chemical Intake (mg/kg-day)} \times \text{SF (mg/kg-day)}^{-1}$$

This formula applies to cancer risks lower than 1×10^{-2} (1 in 100). All cancer risks in this assessment were lower than 1×10^{-2}.

The risks resulting from exposure to multiple carcinogens are assumed to be additive. The total cancer risk is estimated by adding together the estimated risk for each COPC and for each exposure pathway (Appendix A, Table 8 through 10 series). However, in this instance, arsenic is the only carcinogen that was evaluated. The EPA's target acceptable excess cancer risk range is 10^{-6} to 10^{-4}. Cancer risks below 10^{-6} are considered acceptable and will not be evaluated further. Cancer risks above 10^{-4} are generally considered unnaccpetable and generally warrant remedial action. Cancer risks which fall within this range will be further evaluated in the Feasibility Study where risk management decisions will be considered and remedial actions may or may not be warranted (USEPA 1991c).

5.3 RISK CHARACTERIZATION RESULTS

The results of the risk calculations are provided in Appendix A, Table 7 through 10 series. Total CT and RME hazard and risk was estimated for each exposure scenario in each exposure area by combining the individual hazards and risks calculated for each media and pathway, except for the vegetable and fish ingestion pathways. These two pathways were evaluated separately. Hazards and risks were not combined across exposure scenarios, except qualitatively as discussed later in this section (Section 5.3.3). Total hazards and risks are evaluated in order to identify potential areas of concern for further evaluation. Where total hazards and risks are elevated above EPA's acceptable range, the risk drivers (whether chemical specific or media specific, or both) are identified and then individual hazards and risks are evaluated. The following media and pathways were included in total risks and hazards for each exposure scenario:

- Residential – Ingestion of and dermal contact with yard soil; ingestion of groundwater currently used as a drinking water source (tap water).

- Future Residential – Ingestion of shallow groundwater near metal source areas (groundwater is not currently used as drinking water). Future residents would also have the same yard soil contact as listed above for residential.
- Neighborhood recreational – Ingestion of and dermal contact with floodplain soil/sediment; ingestion of surface water. For Nine Mile and Mullan, ingestion of and dermal contact with waste pile soil was also included. For Side Gulches, Silverton and Wallace, ingestion of and dermal contact with soil from upland parks and schools was also included.
- Public recreational (visitors) – Ingestion of and dermal contact with floodplain soil/sediment; ingestion of surface water. For Silverton and Wallace, only ingestion of and dermal contact with soil from upland parks and schools were included in the risks and hazards.
- Construction workers – Ingestion of and dermal contact with surface and subsurface soils.

ž Subsistence – Ingestion of and dermal contact with surface soil and floodplain soil/sediment; ingestion of surface water; ingestion of fish; and ingestion of water potatoes.

Table 5-1 provides CT and RME values for total hazard index and cancer risk for each exposure scenario. In addition, the table provides information regarding key chemicals and media contributing to RME hazard index and cancer risk values. Figures 5-1 to 5-20 also provide information regarding key chemicals and media contributing to the RME hazard index and cancer risk. USEPA (1989) risk assessment guidance recommends that final hazard and risk results be presented as one significant figure only. Therefore, on the Tables at the end of this section, a hazard quotient of 1 could be any value between 0.95 and 1.4. For values less than 0.95, a hazard is again rounded to one significant figure, only the reported value is a decimal, rather than a whole number. (For example, for a value of 0.85, a hazard of 0.9 is reported.) Similarly, a risk of 2×10^{-5} could be any value between 1.5×10^{-5} and 2.4×10^{-5}. Hazards that round to less than one are presented as less than one, with one significant figure, i.e. 0.4. The Appendix A Tables 7 through 10 series, and the figures at the end of this section report risk and hazard results to two significant figures. The following subsections discuss noncancer hazards, cancer risks, and hazard/risks for scenario combinations.

5.3.1 Noncancer Hazard

This section discusses total hazard indices for chemicals and pathways in each exposure area, and chemical-specific hazard indices for risk drivers summed across pathways in each exposure area. Noncancer hazards were evaluated for four age groups: child residential and public recreational (child visitor), 0 to 6 years; child neighborhood recreational, age 4 to 11; child/adults, age 0 to 30; and occupational adults (construction workers), 25 years of exposure. In all cases, the greatest hazards were for children, age 0 to 6.

Total Hazard Indices

As shown in Table 5-1, total CT hazard indices were less than or equal to 1, with the exception of the future residential scenario in Burke/Nine Mile (CT hazard index = 10 and 5 for child and child/adult, respectively). Total RME hazard indices exceeded 1 for the 0- to 6-year age group in all of the

geographical areas where residential hazards were evaluated: the Lower Basin, Kingston, the Side Gulches, Osburn, Silverton, Wallace, Mullan, and Burke/Nine Mile. Total RME hazard indices also exceeded 1 for a number of other scenarios: the future residential scenario in Burke/Nine Mile; the child public recreational scenario in the Lower Basin and Kingston; the child/adult residential scenario in the Side Gulches; the child neighborhood recreational scenario in the Side Gulches; the occupational scenario in the Lower Basin (Figure 5-1); the future child/adult residential scenario in Burke/Nine Mile; and the vegetable garden pathway.

- For residential scenarios, the key medium contributing to total RME hazard indices was yard soil. One exception was tap water in the Side Gulches, which also contributed significantly to the total RME hazard indices (Figures 5-2 and 5-3).
- For residential scenarios, arsenic and iron were the key chemicals contributing to total RME hazard indices (Figures 5-4 and 5-5).
- In the special case of future use of groundwater by residents in the Burke/Nine Mile area, cadmium and zinc were the key chemicals contributing to the total RME hazard indices (Figure 5-6).
- Depending on the exposure area, one or more of various media (upland surface soil, soil/sediments, surface water, and waste piles) were key contributors to total RME hazard index for recreational scenarios (Figures 5-7 and 5-9).
- For recreational scenarios, arsenic and iron were the key chemicals contributing to total RME hazard index (Figures 5-8 and 5-10).
- For occupational scenarios, arsenic and iron were the key chemicals contributing to total RME hazard index (Figure 5-11).
- For homegrown vegetables, cadmium was the key chemical contributing to the total RME hazard index, whereas for fish, mercury was the key chemical (Figures 5-12a-b).

The total RME hazard indices suggest that several of the exposure scenarios listed could pose a threat of noncancer health effects, assuming that the effects from the key COPCs (i.e., arsenic and iron or cadmium and zinc) are additive. As discussed in the following section, this is a protective assumption that probably overestimates the hazard index at the site.

Chemical-Specific Hazard Indices

Risk drivers in each of the current scenarios were either (1) arsenic and/or iron or (2) cadmium and/or zinc. There is no evidence that noncancer effects from arsenic plus iron or cadmium plus zinc are additive. On the contrary, it is more likely that the interactions of these two pairs of chemicals are antagonistic (protective), rather than additive. Iron is known to interfere with the absorption of ingested metals (e.g., calcium, lead, and cadmium); (whether iron affects the absorption of ingested arsenic is unknown). Oral zinc supplementation decreases the absorption of orally administered cadmium in humans (ATSDR 1999a). Dietary zinc reduces cadmium-induced testicular damage, hypertension, pulmonary damage, and developmental effects in animals (USEPA 1994c). The mechanism of zinc's reduction of cadmium toxicity may be (1) a zinc-induced decrease in the bioavailability of ingested

cadmium and/or (2) a decrease in cadmium/zinc ratio in target tissues (USEPA 1994c). Chemical interactions are further discussed in Section 7.

Because of the uncertainties surrounding chemical interactions, chemical specific hazard indices were also evaluated and are discussed below. Table 5-2 lists scenarios for which hazard indices for specific chemicals (added across pathways) were equal to or greater than 1.

For chemical-specific RME hazard indices:

- Other than the special cases (discussed in the last two bullets below), the only specific chemicals with RME hazard indices that exceeded 1 were arsenic and, in one case only (residential exposures for children 0 to 6 years in the Lower Basin), iron.
- Of the nine exposure areas, four (Kingston, Silverton, Wallace, and Blackwell Island) had no cases in which specific chemicals had RME hazard indices that were equal to or greater than 1.
- Only residential scenarios had RME hazard indices that exceeded 1. In no cases did the neighborhood recreational, public recreational, or occupational scenarios have chemicals with RME hazard indices that exceeded 1.
- Tap water in the Side Gulches was the only medium other than soil for which an HQ exceeded 1, with the exception of the special cases described in the last two bullets.
- In the special case of future use of groundwater in the Burke/Nine Mile area, RME hazard indices for cadmium and zinc each exceeded 1 for the residential child and residential child/adult scenarios.
- In the special case of homegrown produce, RME hazard indices for cadmium exceeded 1 for the residential child/adult scenarios (vegetable hazards were not calculated separately for the child age group because ingestion rates for this parameter were age adjusted and apply equally to both age groups).

The hazard index for iron exceeded 1 only in the Lower Basin, and the Lower Basin is the only area where iron concentrations are likely to be greater than background concentrations. Therefore, iron is a concern only for the 0- to 6-year age group in the Lower Basin. While the majority of arsenic concentrations did not result in hazard indices greater than 1 for a particular scenario, arsenic hazard indices were equal to 1 for young children for most residential scenarios (See Table 5-2), as well as for some neighborhood and public scenarios. Because arsenic hazards are at their allowed maximum for residential children, any additional exposures could potentially increase the hazards above acceptable levels; therefore, arsenic is the significant noncancer chemical besides lead in the entire Basin.

Hazards in the Side Gulches from the tap water are due to arsenic concentrations of 8 µg/L in one private well. In general, arsenic concentrations in private sources from all areas are the major contributors to drinking water hazards. Most private water sources had concentrations ranging from 1 to 4 µg/L, with the Side Gulches well containing the highest concentration in the Basin. The new MCL for arsenic is 10 µg/L, and arsenic concentrations in some private water sources in the Basin may

exceed the new MCL. In general, arsenic concentrations in public water systems ranged from nondetected to 0.6 µg/L.

The results of the noncancer evaluation for RME cases suggest that the following exposure scenarios could pose an unacceptable threat of noncancer effects if current conditions remain the same:

- Arsenic and iron (primarily in yard soil) for child residents in the Lower Basin,
- Arsenic (primarily in yard soil and tap water) for child and child/adult residents in the Side Gulches (note: some arsenic water concentrations from private sources throughout the Basin may exceed the new MCL),
- Arsenic (primarily in yard soil) for child residents in Osburn and Mullan, and arsenic in soil and groundwater in Burke/Nine Mile (this groundwater is not currently used as a drinking water source),
- Cadmium and zinc in groundwater for child and child/adult residents in Burke/Nine Mile, and
- Cadmium in homegrown vegetables for child/adult residents.

For the CT cases, potential unacceptable exposures occur only for child residents in the Side Gulches when all exposure routes are combined and for future child and future child/adult residents of Burke/Nine Mile from ingestion of tap water.

5.3.2 Cancer Risks

Cancer risks were evaluated for two age groups: child/adult, age 0 to 30, and occupational adult, 25 years of exposure.

As shown in Table 5-1 and Figure 5-13, total RME cancer risk for each scenario was in the range of 10^{-6} to 10^{-4}. CT cancer risk for each scenario was also in or below the range of 10^{-6} to 10^{-4} (Table 5-1 and Figures 5-16 through 5-18). The risk values presented in the tables and the text are rounded to one significant figure as recommended by the EPA (USEPA 1989). The unrounded values are shown in the figures and in Appendix A, Table 8 through 10 series. Only two areas had cancer risks in the 10^{-4} range, residents in the Lower Basin and the Side Gulches with cancer risks of 1×10^{-4} and 3×10^{-4}, respectively. All other risks were either above 1×10^{-6} or in the 10^{-6} to 10^{-5} range.

Arsenic was the only carcinogenic chemical evaluated at the site. For the residential scenarios, exposure to arsenic in yard surface soil contributed most of the total RME cancer risk (Figure 5-14). However, arsenic in tap water contributed significantly to total RME cancer risk for residents at the Side Gulches (see Figure 5-14 and discussion in previous section). Although tap water was not the primary contributor to cancer risk for the residential scenarios, RME cancer risk for tap water exceeded 1×10^{-6} in all exposure areas (Figure 5-14).

For the special case future residential scenario at Burke/Nine Mile, the total RME cancer risk from groundwater was 3×10^{-5} (Table 5-1). Arsenic risks in surface/subsurface soil for construction workers ranged from 2×10^{-5} to 8×10^{-45} (Figure 5-17). For recreational scenarios in each exposure area, the following media contributed to most or all of RME cancer risk due to arsenic (Figures 5-15 through 5-16):

- Soil/sediment in the lower Coeur d'Alene River for the Lower Basin (highest concentrations of arsenic in the entire Basin with the exception of waste piles),
- Soil/sediment at the North-South confluence in Kingston,
- Upland surface soil from the Elk Creek area and sediment from Elk Creek Pond in the Side Gulches (Elk Creek area soil and sediment had the second highest arsenic concentrations in the entire Basin after floodplain soil/sediments in the Lower Basin),
- Sediment in the South Fork (Osburn, Wallace, and Silverton neighborhood exposures),
- Surface soil from waste piles in Burke/Nine Mile,
- Soil in waste piles and sediment in the South Fork in Mullan, and
- Soil/sediment from the Spokane River on Blackwell Island.

Although surface water was never the primary contributor to RME cancer risk, cancer risk estimates exceeded 1×10^{-6} for surface water in the Lower Basin (neighborhood and public recreational), Kingston (public recreational NS confluence exposures), and the Side Gulches (neighborhood recreational Elk Creek Pond exposures) (Figures 5-15 and 5-16). The samples from these areas consisted of "disturbed" surface water (see Section 2.8), that is, surface water that contained suspended sediments due to disturbance by the sampler. Therefore, surface water risks in the other water bodies could have been as high as those seen for the "disturbed" water bodies if the sampling methods had been the same for all water bodies.

5.3.3 Hazards/Cancer Risks for Combinations of Scenarios

The hazards/cancer risks for individual scenarios discussed in the previous sections do not consider the potential for the same individual to be exposed via more than one exposure scenario. For example, it is possible that children and adults exposed to yard soil and tap water at their home could also be exposed to other media (soil, sediment, waste piles, or surface water) during recreational use of schools, parks, creeks, and ponds in the neighborhood. Other combinations of scenarios are also possible such as residents who also visit public recreational areas, catch and eat locally caught fish or residents who also eat homegrown vegetables, and so on.

Child/Adult Residential Plus Neighborhood Recreational

Table 5-3 and Figures 5-18 and 5-19 provide total RME hazard index and cancer risk for the combined residential child/adult and neighborhood recreational scenarios. In addition, the table provides information regarding key chemicals and media contributing to the hazard index and cancer risk values. Hazards for the youngest age group (0-6 year-olds) were not added to neighborhood

hazards (4-11 year-olds) because the age range is not the same. The 4-11 year old age group is included in the integrated child/adult which covers ages 0-30 years.

As shown in Table 5-3, total RME hazard indices for the combined residential child/adult and neighborhood recreational scenarios exceeded 1 in the Lower Basin, Kingston, the Side Gulches, and Burke/Nine Mile. Risk drivers were arsenic and iron in yard soil and in various media (e.g., soil, sediments, and waste piles) in neighborhood recreational areas. In addition, arsenic in tap water contributed significantly to the RME hazard index for the Side Gulches.

The total hazard indices suggest that child/adult residents in these 4 areas who are also exposed to recreational media in the neighborhood might have an unacceptable threat of noncancer health effects assuming that effects from the key chemicals (i.e., arsenic and iron) are additive. However, there is no evidence that noncancer effects from exposure to arsenic plus iron are additive. Therefore, chemical-specific hazard indices were estimated for the combined residential child/adult and neighborhood recreational scenarios. The only chemical-specific hazard indices that exceeded 1 were for arsenic (added across pathways) in the Side Gulches and arsenic hazards were equal to one in the Burke/Nine Mile area. Therefore, the results of the noncancer evaluation suggest that the following exposure scenarios could pose an unacceptable threat of noncancer effects for the combined child/adult residential and neighborhood recreational:

- Arsenic (primarily in yard soil, tap water, and soil/sediments) in the Side Gulches, and
- Arsenic (primarily in yard soil and waste piles) in Burke/Nine Mile.

As shown in Table 5-3, the RME cancer risks due to arsenic for the combined residential child/adult and neighborhood recreational scenarios were all in the range of 10^{-6} to 10^{-4}. Lower Basin, Side Gulches and Burke/Nine Mile had the highest total cancer risks of 1×10^{-4}, 3×10^{-4}, and 1×10^{-4}, respectively. Most of the area's total RME cancer risk can be attributed to yard surface soil concentrations (Figure 5-14). Tap water also contributed significantly to the total RME cancer risk in the Side Gulches (Figure 5-14), and various recreational media (soil, sediments, and waste piles) also contributed significantly to the total RME cancer risks in Kingston, Silverton, and Burke/Nine Mile.

Other Combinations of Exposure Scenarios

For several exposure areas, the total RME hazard index and cancer risk for the child/adult residential scenario were slightly less than or equal to 1 and 10^{-4}, respectively. For some of these exposure areas, adding neighborhood recreational exposure to the child/adult residential scenario increased the total RME hazard index and cancer risk to levels greater than 1 and 10^{-4}, respectively. The addition of other types of exposure to the child/adult residential scenario (e.g., ingestion of homegrown vegetables or fish, or visiting public recreational areas) might also in some cases increase the RME hazard index and cancer risk to unacceptable levels. Noncancer hazards from sport fishing were slightly less than 1 (0.9); however, the noncancer hazard from eating cadmium in vegetables was 2, and cancer risk from arsenic in vegetables was 8×10^{-5}. Cadmium in vegetables is further discussed in Section 7.

5.3.4 Subsistence Risks and Hazards

This section discusses total hazard indices and cancer risk for both modern and traditional subsistence exposure scenarios. For both scenarios, RME noncancer hazards were evaluated for three age groups (child of age 0 to 6 years; child/adult of age 0 to 70, and adult), and RME cancer risks were evaluated for the adult/child age group for arsenic. Tables 5-4 and 5-5 summarize the hazards and risks for the two subsistence scenarios and Figures 5-20 through 5-22 display the risks and hazards by medium for all chemicals. (See also Appendix A, Table 7 through 10 series.)

Modern Subsistence Exposure Scenario

As shown in Table 5-4, total RME hazard indices were greater than 1 for each age group in the modern subsistence exposure scenario. The age group having the greatest total hazard index was the child age group with 9, followed by child/adult with 4, and then adult with 3. This suggests that exposure to metals through all pathways presents an unacceptable health hazard to all age groups for noncancer health effects, with children having the greatest risk.

- For the child age group, the key pathways contributing to total RME hazards are ingestion of arsenic and iron from surface soil, sediment, and arsenic in undisturbed surface water (Figure 5-21).
- For the adult/child age group, when evaluating hazards across the individual media, none of the hazard indices exceeded 1. When evaluating total hazards by chemical (i.e., adding the hazards from each pathway for a particular chemical), only the total hazard index for arsenic exceeded 1 (Table 5-4).
- Ingestion of fish was the only pathway evaluated for the adult only age group. The total hazard index for fish ingestion exceeded 1, with mercury in northern pike being the greatest risk driver (Table 5-4). As was previously discussed in Section 2.2.1, whole fish tissue data is not available for use in this human health risk assessment for the tribal scenarios. Whole body metal concentrations are usually higher than fillet concentrations; thus, use of fillet data for populations which consume whole fish (tribal subsistence scenarios) likely underestimates the chemical dose from fish.

Also shown in Table 5-4 are the RME cancer risks for arsenic for the modern subsistence exposure scenario. RME cancer risks exceed 10^{-6} in all exposure pathways with cancer risks ranging from approximately 1×10^{-5} to 2×10^{-4}. Total RME cancer risk is approximately 7×10^{-4}. Exposure to arsenic in sediments through dermal absorption plus ingestion contributed most of the total RME cancer risk (Figure 5-22). Ingestion of undisturbed surface water also contributed significantly to total risk.

Traditional Subsistence Exposure Scenario

As shown in Table 5-5, total RME hazard indices were greater than 1 for each age group in the traditional subsistence exposure scenario, the child having the greatest hazard with a hazard index of 43. The total noncancer hazards for the adult and adult/child age groups were 10 and 19, respectively.

- For children, exposure to metals through all exposure pathways, except the ingestion of disturbed surface water, represents potential unacceptable risk for noncancer health effects (Figure 5-20). Ingestion of surface soil and ingestion of sediment contribute most to the total RME hazard index for the traditional subsistence exposure scenario with hazard indices of 17 and 11, respectively.
- For the combined adult/child age group, the total hazard index exceeded 1 for each exposure pathway except dermal absorption from surface soil and ingestion of disturbed surface water. Ingestion of surface soil is the greatest risk driver for this age group. Ingestion of water potatoes, sediment and undisturbed surface water each contribute hazard indices of 4, 3 and 4, respectively to the total RME hazard index (Figure 5-21). The key metals contributing to the total RME hazard are arsenic, cadmium and iron.
- Ingestion of fish was the only pathway evaluated for the adult age group. The total hazard index for fish ingestion exceeded 1, with mercury in northern pike being the most significant risk driver. As was previously discussed in Section 2.2.1, whole fish tissue data is not available for use in this human health risk assessment for the tribal scenarios. Whole body metal concentrations are usually higher than fillet concentrations; thus, use of fillet data for populations which consume whole fish (tribal subsistence scenarios) likely underestimates the chemical dose from fish.

Also shown in Table 5-5 are the RME cancer risk for arsenic for the traditional subsistence exposure scenario for the combined adult/child age group. RME cancer risks exceeded 10^{-6} in all exposure pathways, with cancer risks ranging from approximately 4×10^{-5} to 1×10^{-3} (Figure 5-22). Total RME cancer risk is approximately 3×10^{-3}, suggesting potentially unacceptable cancer risks from exposure to arsenic through all media and pathways.

The hazards from eating fish are underestimated for subsistence residents because hazard estimates are based on concentrations in fish fillets. The subsistence tribal members eat the whole fish, not just the fillets, and concentrations of metals in whole fish are greater than those in fillets. In addition, fish fillet data are from the lateral lakes, not Coeur d'Alene Lake. Sufficient fish tissue data were not available from Coeur d'Alene Lake to characterize health risks; however, tribal populations do eat fish from the lake. Therefore, tribal health hazards from eating fish from Coeur d'Alene Lake are unknown.

5.4 SUMMARY OF RISK CHARACTERIZATION

The results of the risk characterization indicate that some exposure areas could pose an unacceptable threat of noncancer effects for some individuals and exposure media under the RME condition. These include (1) young children exposed to arsenic in yard soil in the Lower Basin, the Side Gulches, Osburn, Mullan, and Burke/Nine Mile, (2) young children exposed to iron in yard soil in the Lower Basin, (3) children/adults exposed to arsenic in yard soil and tap water in the Side Gulches, (4) young children and children/adults ingesting cadmium and zinc in groundwater in Burke/Nine Mile in the future (groundwater at Burke/Nine Mile is not currently used as a drinking water source), (5) young children and children/adults ingesting cadmium in homegrown vegetables, and (6) all residents and pathways for subsistence lifestyles.

Cancer risk estimates exceeded 1×10^{-6} for all individuals in all exposure areas under the RME condition. Over half of the areas also had cancer risk estimates exceeding 1×10^{-6} for all individuals under the CT condition. Only one scenario (RME condition for residents in the Side Gulches) had a cancer risk exceeding 1×10^{-4}.

Arsenic was the only carcinogenic COPC evaluated at the site. For residential scenarios, yard surface soil contributed the most to cancer risk. For residents in the Side Gulches, tap water also contributed significantly to cancer risk. Although tap water was not the primary contributor to cancer risk for residential scenarios, RME cancer risk estimates for tap water exceeded 1×10^{-6} in all exposure areas. The risk is almost entirely due to selected high concentrations of arsenic in scattered private wells.

Depending on the exposure area, one or more of various media (upland surface soil, soil/sediments, sediments, or waste piles) contributed the most to cancer risk for recreational visitors. Although surface water was never the primary contributor to cancer risk, RME cancer risk estimates for "disturbed" surface water exceeded 1×10^{-6} for recreational scenarios in several exposure areas. Surface/subsurface soil contributed all of the cancer risk for construction workers.

Surface soil and sediment contributed the most to hazards and cancer risks for the subsistence scenarios. The modern subsistence scenario had similar hazards to those found for the highest residential child exposures. Cancer risks were higher for the modern subsistence scenario, but close to those for the highest residential exposures. Hazards and risks for the traditional subsistence scenario were higher than those for the residential scenario by an order of magnitude. For the modern subsistence scenario, arsenic and iron were the only chemicals with hazard quotients greater than 1 (also similar to residential hazards). For the traditional scenario, mercury in fish, manganese in soil and sediment, and cadmium in water potatoes also had hazard quotients greater than 1 in addition to arsenic and iron. Hazards from mercury in fish are likely underestimated for subsistence tribal members because they eat the whole fish, not just fillets.

Combinations of the exposure scenarios described above (e.g., child/adult residential plus neighborhood recreational) would result in hazard/risk estimates that are higher than those discussed in this summary. However, combining the risk and hazard numerical results from the scenarios probably overestimates the total numerical hazard/risk for actual residents. For example, child/adult residents are

assumed to spend 24 hours/day, 350 days/year at the residence. Assuming that they also regularly spend several hours/day at a neighborhood or public recreational area or are occupationally exposed results in "double counting" (exposure for more than 24 hours/day), which will overestimate hazard/risk. However, it is clear that many of these additional exposure pathways could result in higher total risks than those shown for residential individuals.

Although risks and hazards have been evaluated by geographical subarea, residential remedial actions will be made on a home by home basis and will not occur without sampling, if there is no data. Common use areas, neighborhood, and waste pile remedial activities will be determined on a site by site basis and will involve the local communities. In addition, as with residential remedial activities, no remediation will take place in the absence of data.

5.5 POTENTIAL PRELIMINARY REMEDIATION GOALS FOR ARSENIC

This section evaluates potential health-based preliminary remediation goals (PRGs) in site soils under current and probable future conditions. These calculated PRGs provide targets for developing and evaluating response action alternatives and are considered along with current U.S. EPA guidelines on remediation and risk management . They are considered preliminary during the RI/FS stage; they do not become final action levels until agreement regarding the removal action is reached and documented in the Remedial Action Objectives document. In this section, potential PRGs were developed for residential, public recreational and neighborhood recreational exposures to arsenic in soil.

Chemical-specific PRGs are concentration goals for individual chemicals for specific media and exposure scenario combinations (USEPA 1991e). Potential risk-based PRGs were only developed for arsenic because it is the COPC that was the most consistent risk driver in the various scenarios. Potential PRGs were developed in order to provide an estimate of the number of basin homes that could require remedial activity based on concentrations in the Basin.

Potential risk-based preliminary remediation goals (PRGs) were calculated by defining a target risk goal and then solving the basic risk assessment equations for concentration rather than solving for risk (USEPA 1991e). Target risk goals and equations differ for carcinogenic and noncarcinogenic health effects, as described in Sections 5.1 and 5.2.

The target goal for noncancer hazards is typically a hazard index of 1. A hazard index of 1 is the point at which the estimated dose equals the reference dose associated with no adverse health effects. RME hazard indices for exposure to soil ranged from 2 to 4 for child residential (age 0-6 years) exposures to yard soil; 0.7 to 2 for child public recreational (age 0-6 years) exposures to soil/sediment; and 0.2 to 0.9 and 0.1 to 0.9 for child neighborhood recreational (age 4-11 years) exposures to waste piles and soil/sediment, respectively. Arsenic is the COPC that was most consistently a risk driver for these scenarios. Therefore, potential PRGs were calculated for exposure to arsenic in soil by these receptors, with the idea that remediation of the soil to levels appropriate for reduction of health effects resulting from exposure to arsenic will sufficiently lower total hazards. Potential PRGs for noncancer

health effects associated with exposure to arsenic via the oral and dermal exposure pathways were calculated using the summary intake factors (SIFs) as previously derived in Section 3.3.2 in the following equation.

$$PRG_{NC}\ (mg/kg) = THQ \times RfD / (SIF_{ing} + SIF_{derm})$$

where,

THQ	= Target hazard quotient (unitless)
RfD	= Reference dose (mg/kg-day)
SIF_{ing}	= Ingestion summary intake factor (kg/kg-day)
SIF_{derm}	= Dermal summary intake factor (kg/kg-day)

Target cancer risk goals set by U.S. EPA are defined over a range of 10^{-6} to 10^{-4} (USEPA 1991d). RME cancer risks from exposure to arsenic in soil ranged from 4×10^{-5} to 8×10^{-5} for residential exposures to yard soil; 1×10^{-5} to 4×10^{-5} for public recreational exposures to soil/sediment; and 3×10^{-6} to 3×10^{-5} and 2×10^{-6} to 2×10^{-5} for neighborhood recreational exposure to waste piles and soil/sediment, respectively. The following equation was used to calculate the cancer risk PRG:

$$PRG_{CA} = TR / (SF \times (SIF_{ing} + SIF_{derm}))$$

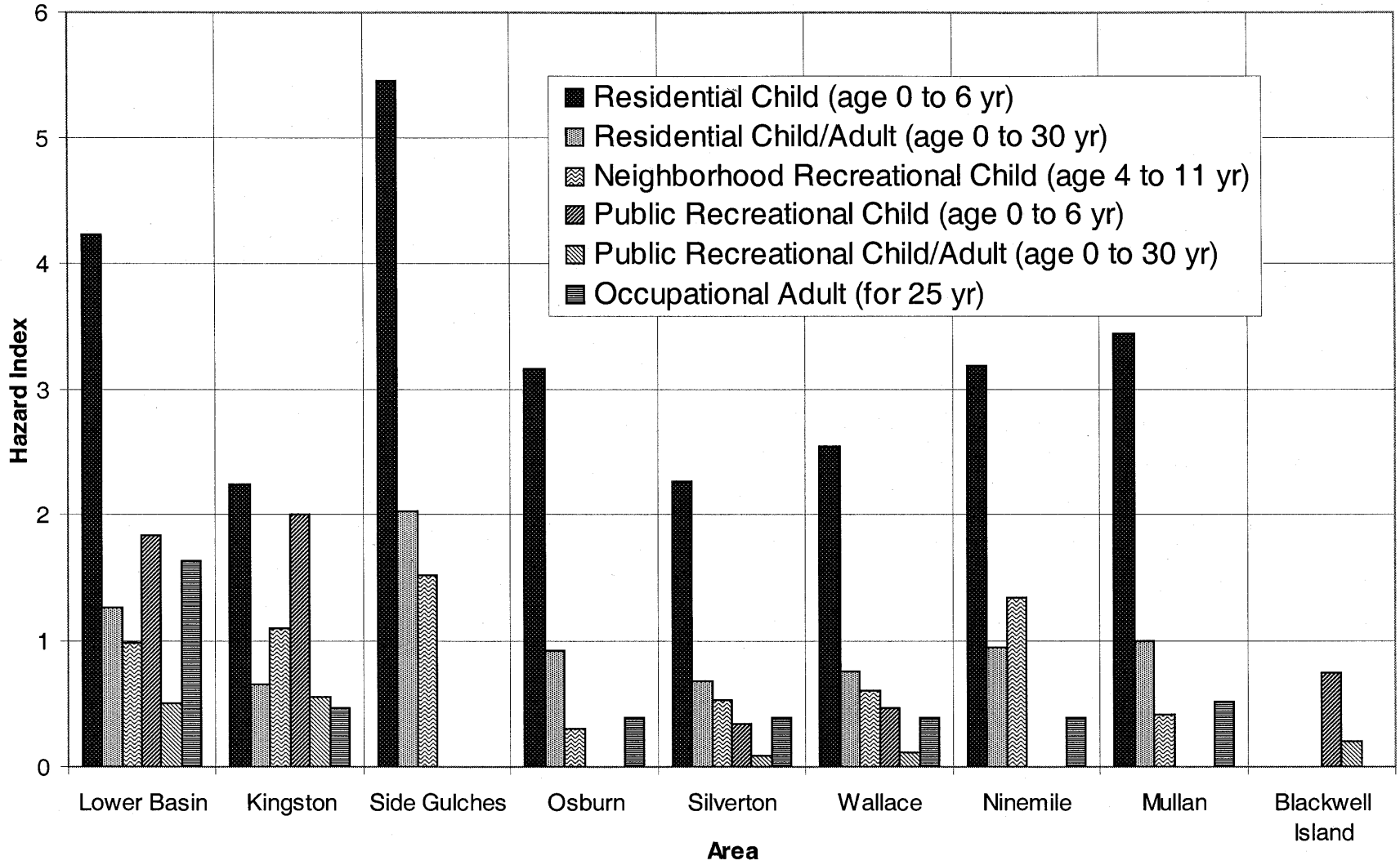
Where,

TR	= Target risk (unitless)
SF	= Cancer slope factor ($(mg/kg\text{-}day)^{-1}$)
SIF_{ing}	= Ingestion summary intake factor (kg/kg-day)
SIF_{derm}	= Dermal summary intake factor (kg/kg-day)

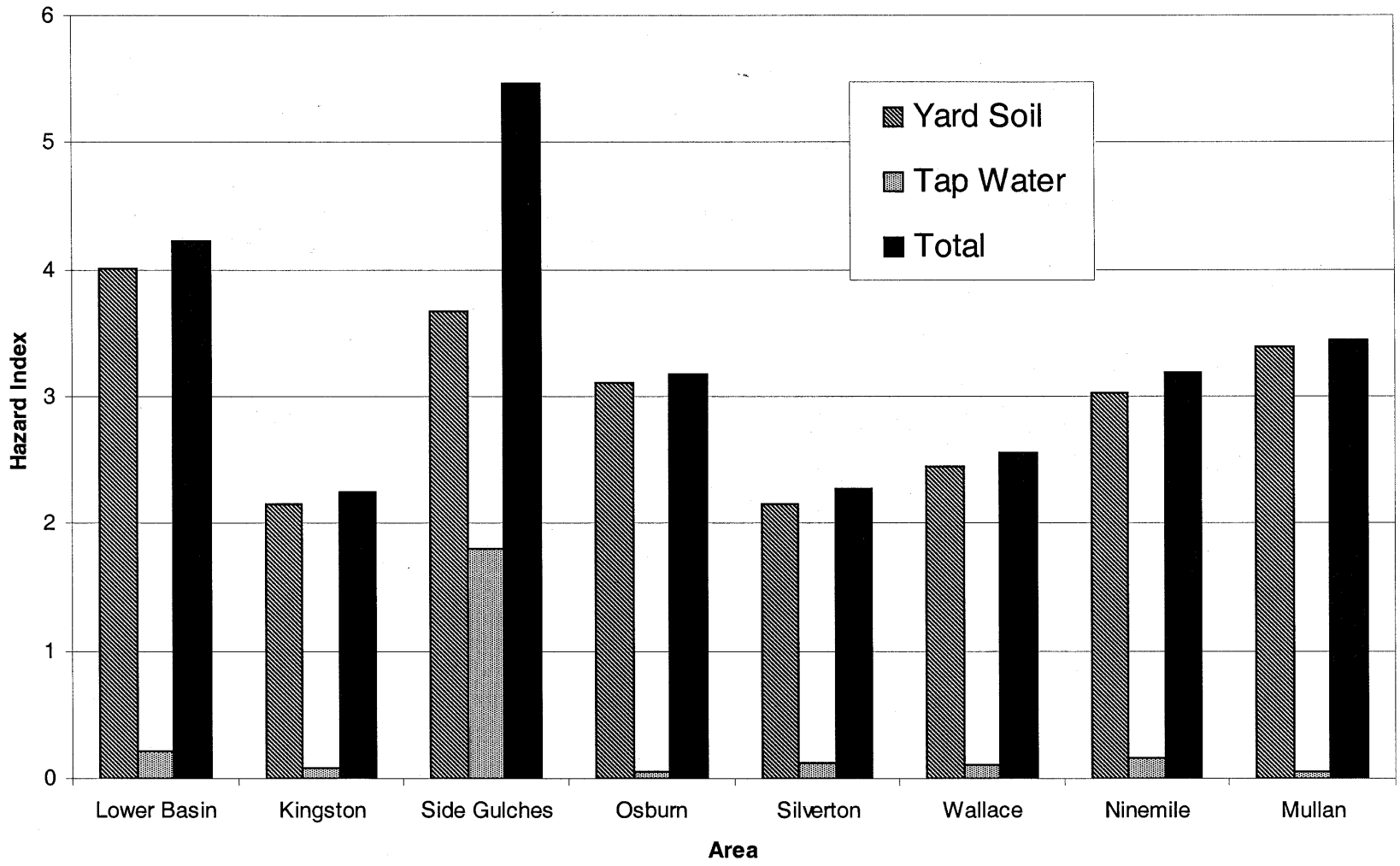
Table 5-6 summarizes the potential residential, public recreational, and neighborhood recreational PRGs. The PRGs will likely be selected based on noncancer health effects to young children because for both the residential and public recreational scenarios, the calculated child (0-6) noncancer PRG for the younger age groups is less than the child/adult cancer PRG based on a cancer risk of 10^{-4}. Likewise, for the neighborhood recreational scenario, the child (4-11) noncancer PRG is less than the child (4-11) cancer PRG based on a 10^{-4} cancer risk. However, as noted in Section 5.2, actions may be taken to address cancer risks within the 10^{-6} to 10^{-4} range, depending on site-specific conditions. Nearly all arsenic risks are within this range. PRGs are considered along with EPA guidelines and other risk management issues and do not become final action levels until agreement regarding the removal action is reached and documented in the Remedial Action Objectives memorandum.

Arsenic 95 percent UCLs were calculated for each residential property in the basin with available yard soil data for preliminary comparison with some of the potential PRGs listed on Table 5-6. Comparisons were made between the residential 95 percent UCLs and 35 mg/kg, 64 mg/kg, and 123 mg/kg (the child residential noncancer PRG, the child/adult residential cancer PRG based on 10^{-4} risk,

and the child/adult residential noncancer PRG, respectively). Table 5-7 summarizes the results of this preliminary comparison by geographical area. A total of 242 homes in the Basin have been sampled for yard soil to date. Of these 242 homes, as many as 31% may potentially need remedial activities if the child residential soil noncancer PRG of 35 mg/kg is selected, with 42% and 44% of the homes in the Side Gulches and Osburn, respectively exceeding 35 mg/kg and only 14% and 17% of the homes in Kingston and Silverton, respectively exceeding 35 mg/kg. Conversely, as little as 6% of the homes in the Basin will require remedial action if the child/adult residential soil noncancer PRG of 123 mg/kg is selected, with 15% of the homes in Osburn exceeding 123 mg/kg and none of the homes in Lower Basin and Kingston exceeding 123 mg/kg. However, as previously mentioned, these are preliminary comparisons in order to estimate the amount of remediation that might be needed throughout the Basin. Many other risk management issues, data analysis, and additional sampling may be needed before remedial goals are established.



EPA REGION 10	027-RI-CO-102Q Coeur d'Alene Basin RI/FS BASELINE HHRA	Doc Control: Generation: 1 BHHRA Series 3/12/01	**Figure 5-1** **Summary of Total RME Noncancer Hazard**



EPA REGION 10	027-RI-CO-102Q Coeur d'Alene Basin RI/FS BASELINE HHRA	Doc Control: Generation: 1 BHHRA Series 3/12/01	**Figure 5-2** **Total RME Noncancer Hazard - Residential (Child 0 - 6), All Chemicals**

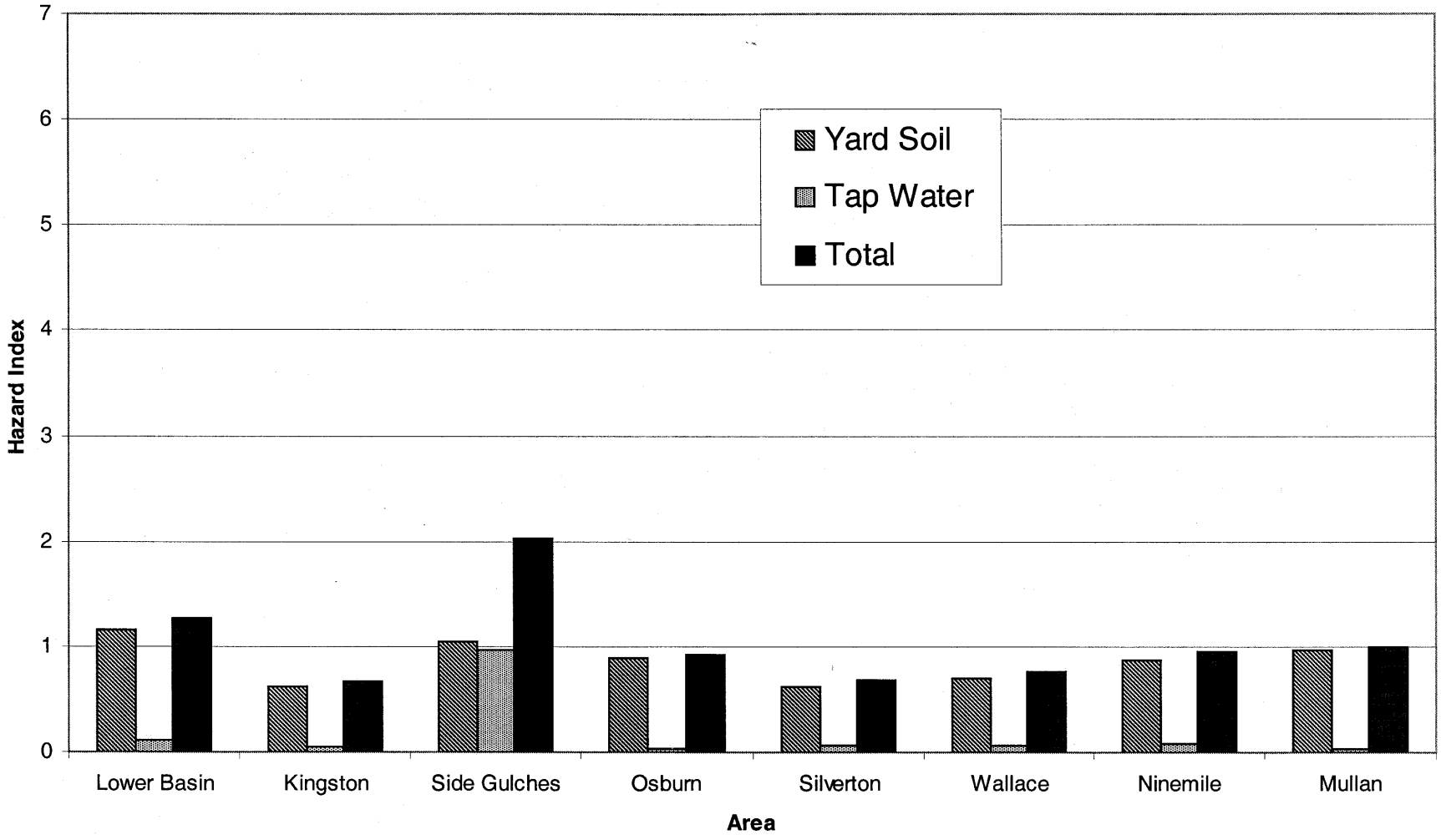


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Figure 5-3
Total RME Noncancer Hazard - Residential (Child /Adult), All Chemicals

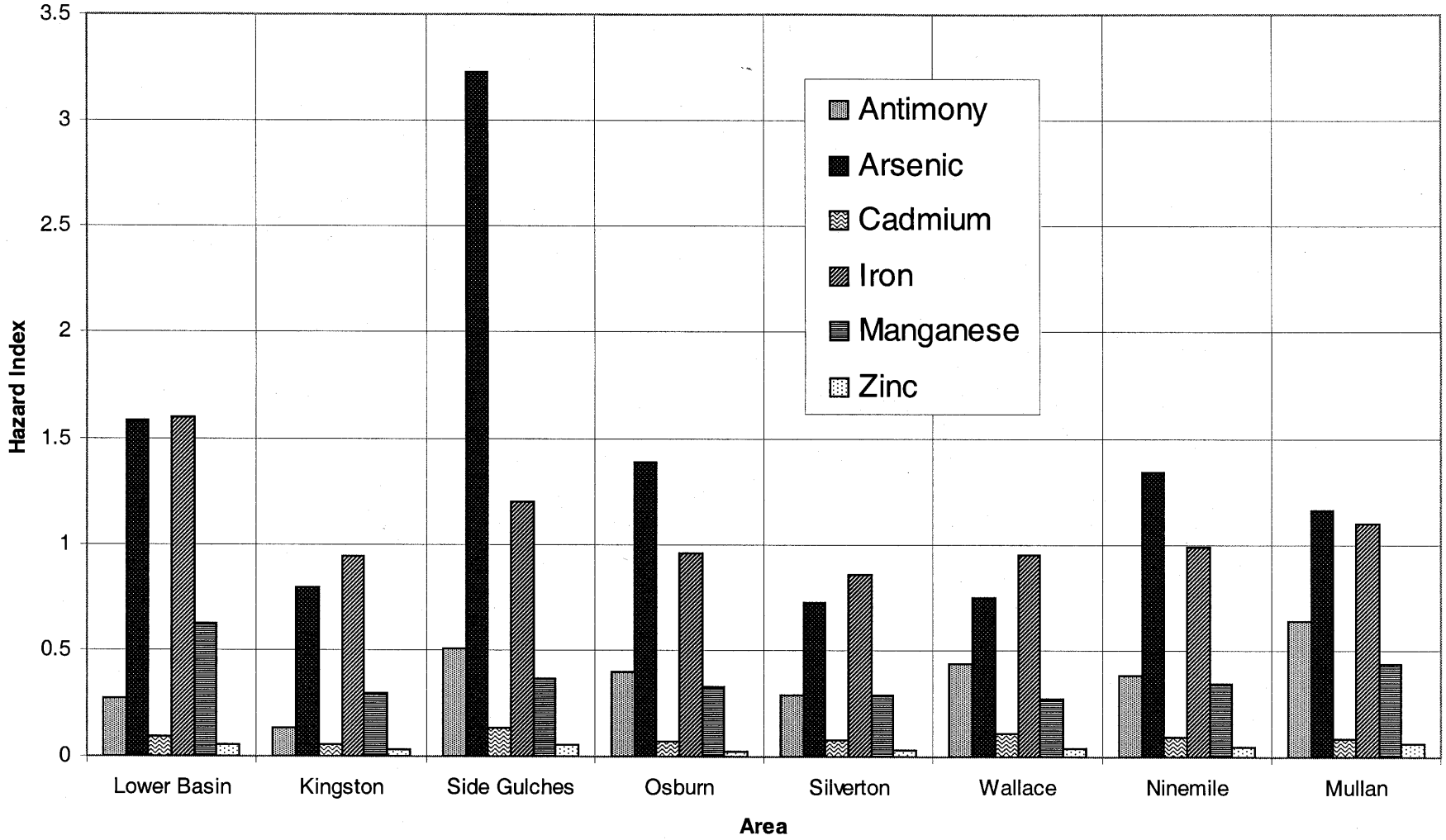

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Figure 5-4
RME Noncancer Hazard - Residential (Child 0 - 6), by Chemical

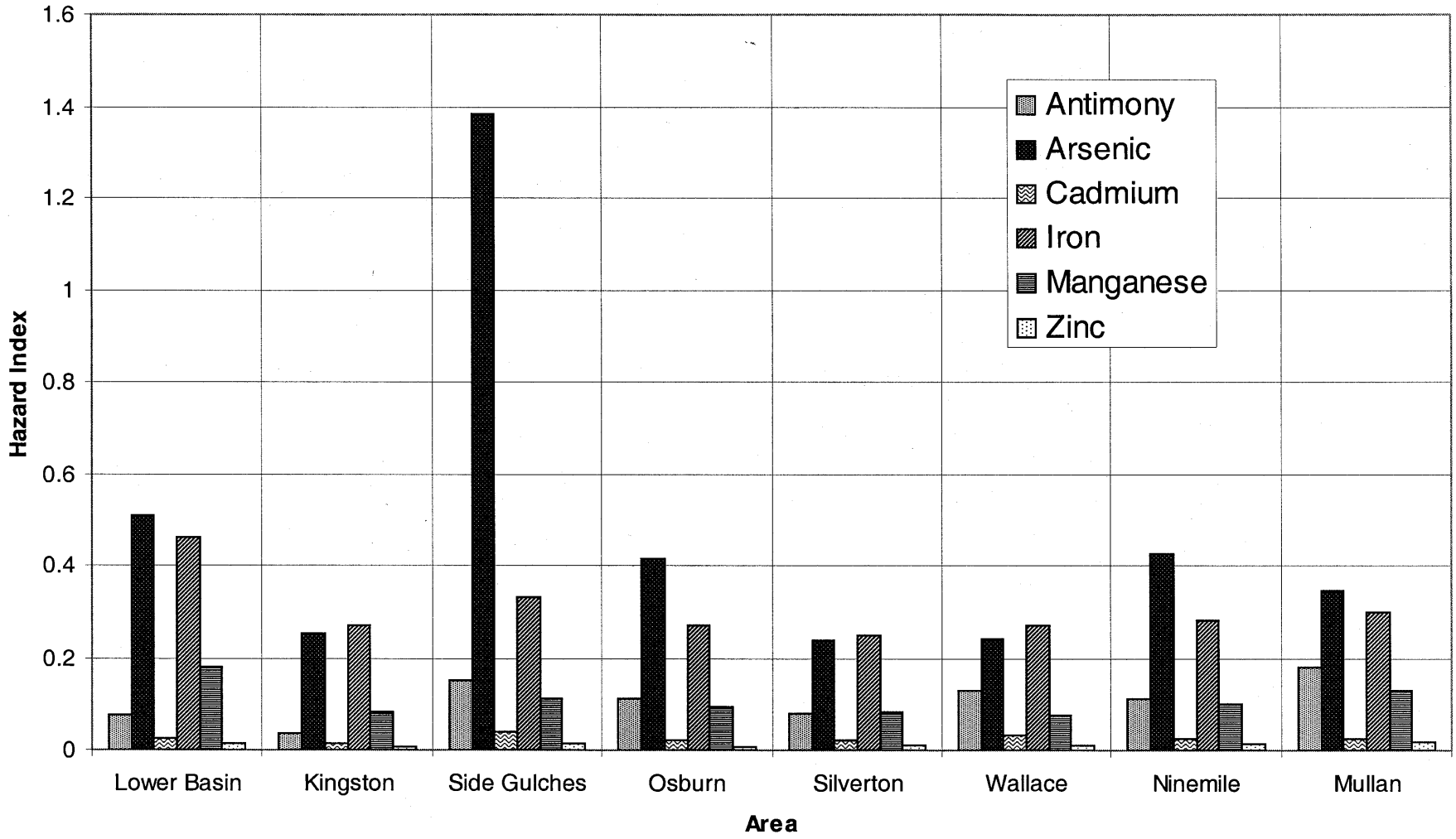

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Figure 5-5
RME Noncancer Hazard - Residential (Child/Adult), by Chemical

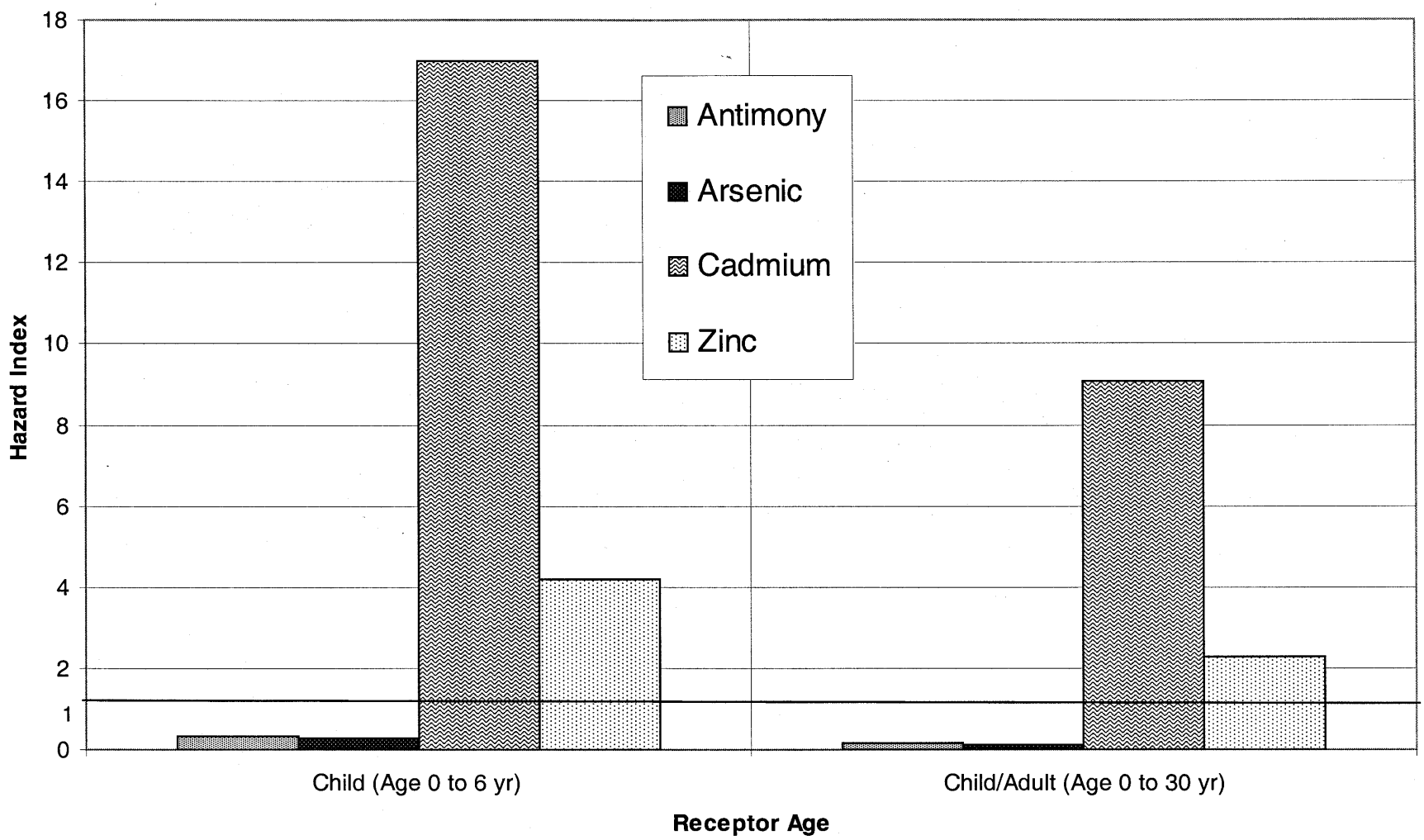

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Figure 5-6
RME Noncancer Hazard - Future Residential, Ninemile

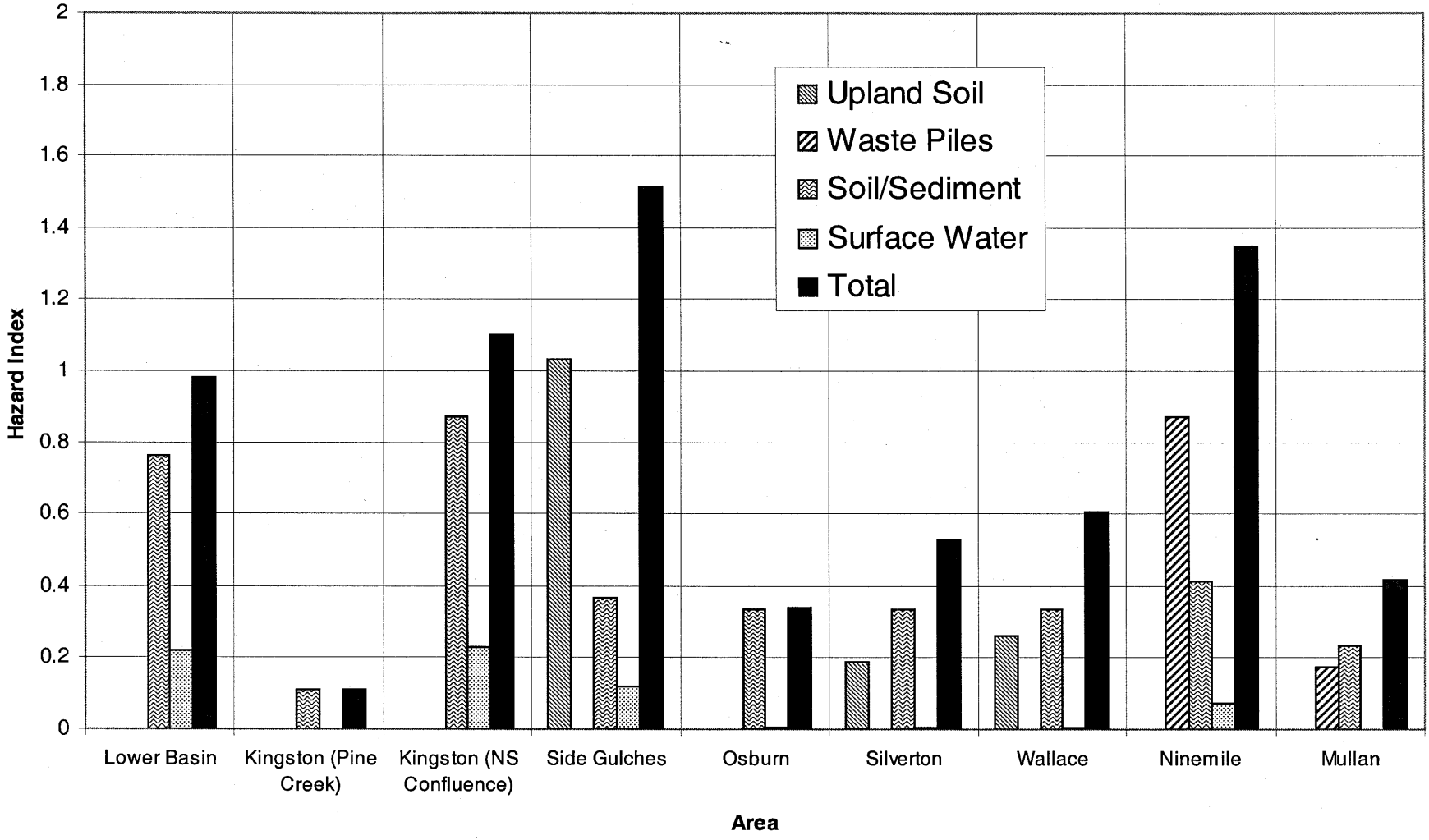

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Figure 5-7
Total RME Noncancer Hazard - Neighborhood Recreational (Child 4-11), All Chemicals

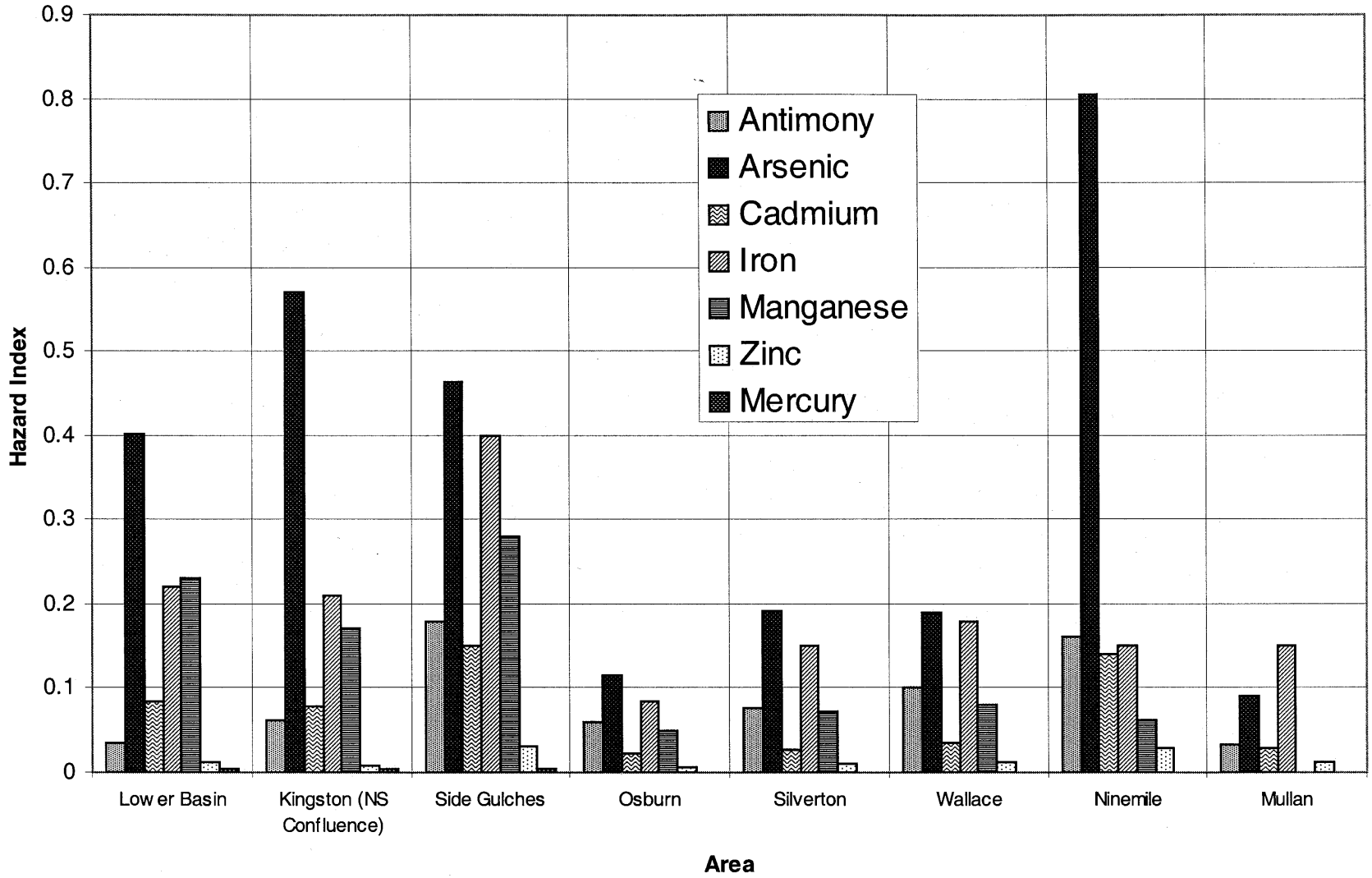

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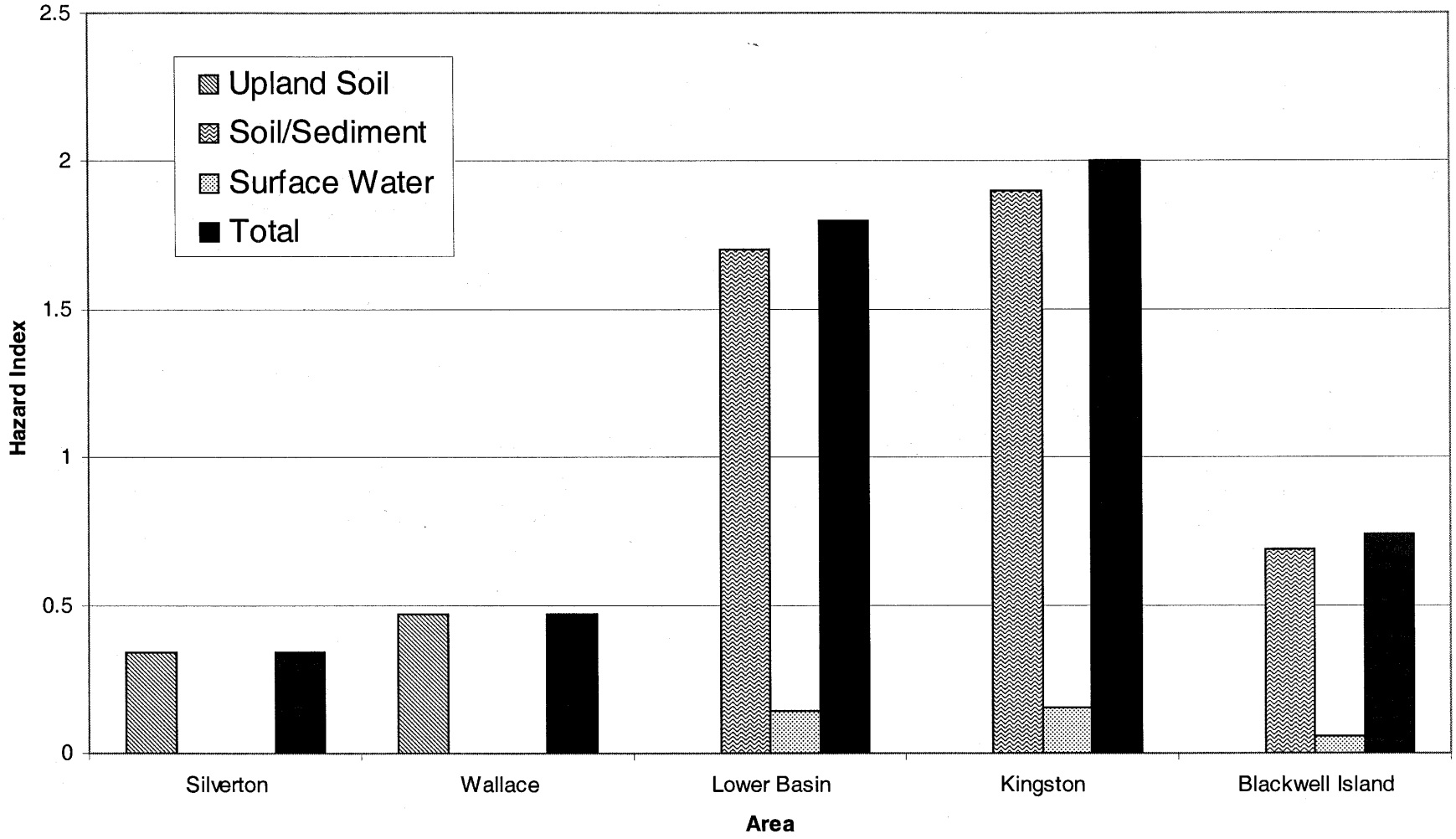
Figure 5-8
RME Noncancer Hazard - Neighborhood Recreational (Child 4-11), by Chemical



EPA REGION 10	027-RI-CO-102Q Coeur d'Alene Basin RI/FS BASELINE HHRA	Doc Control: Generation: 1 BHHRA Series 3/12/01	**Figure 5-9** **Total RME Noncancer Hazard - Public Recreational (Child 0 - 6), All Chemicals**

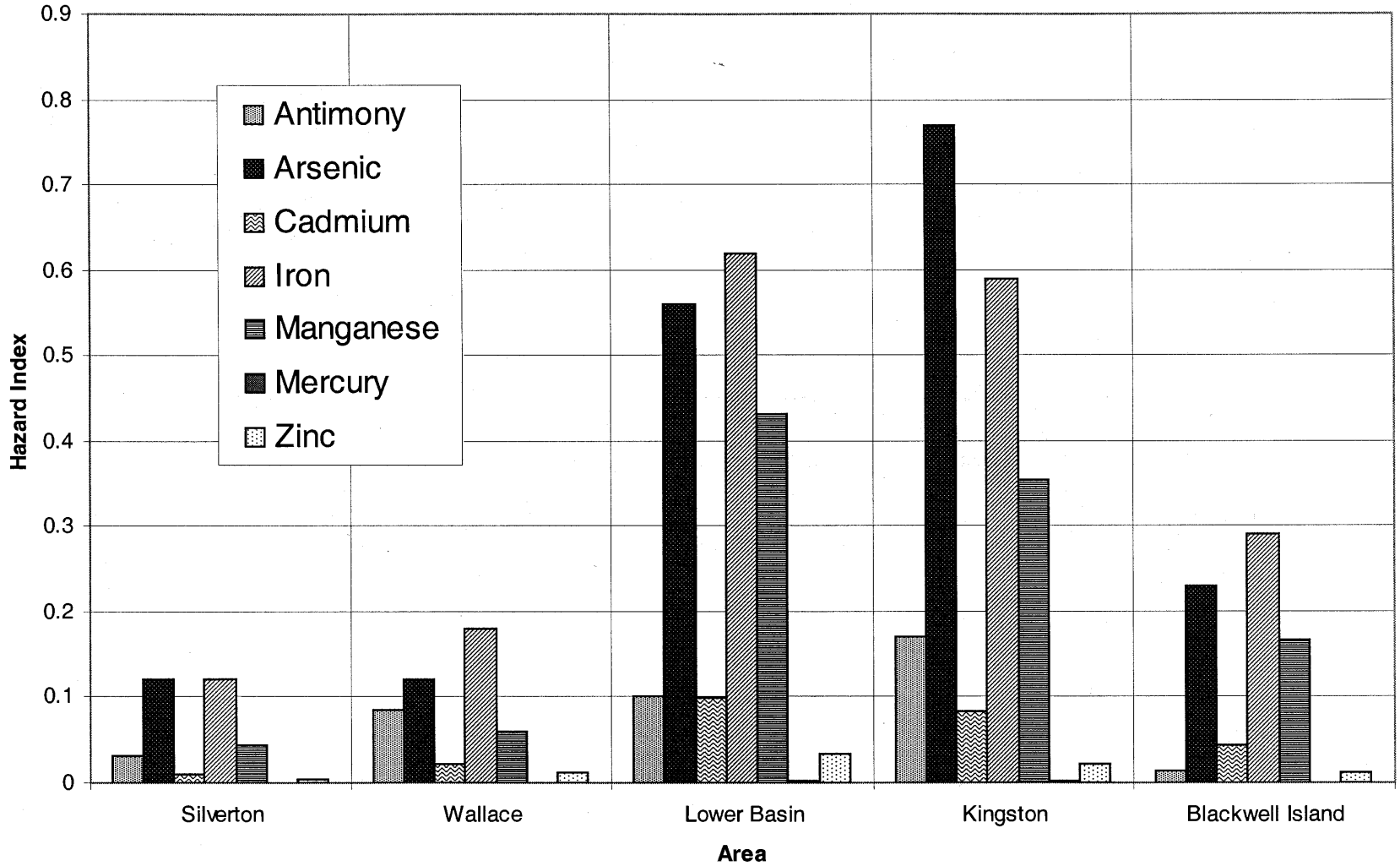


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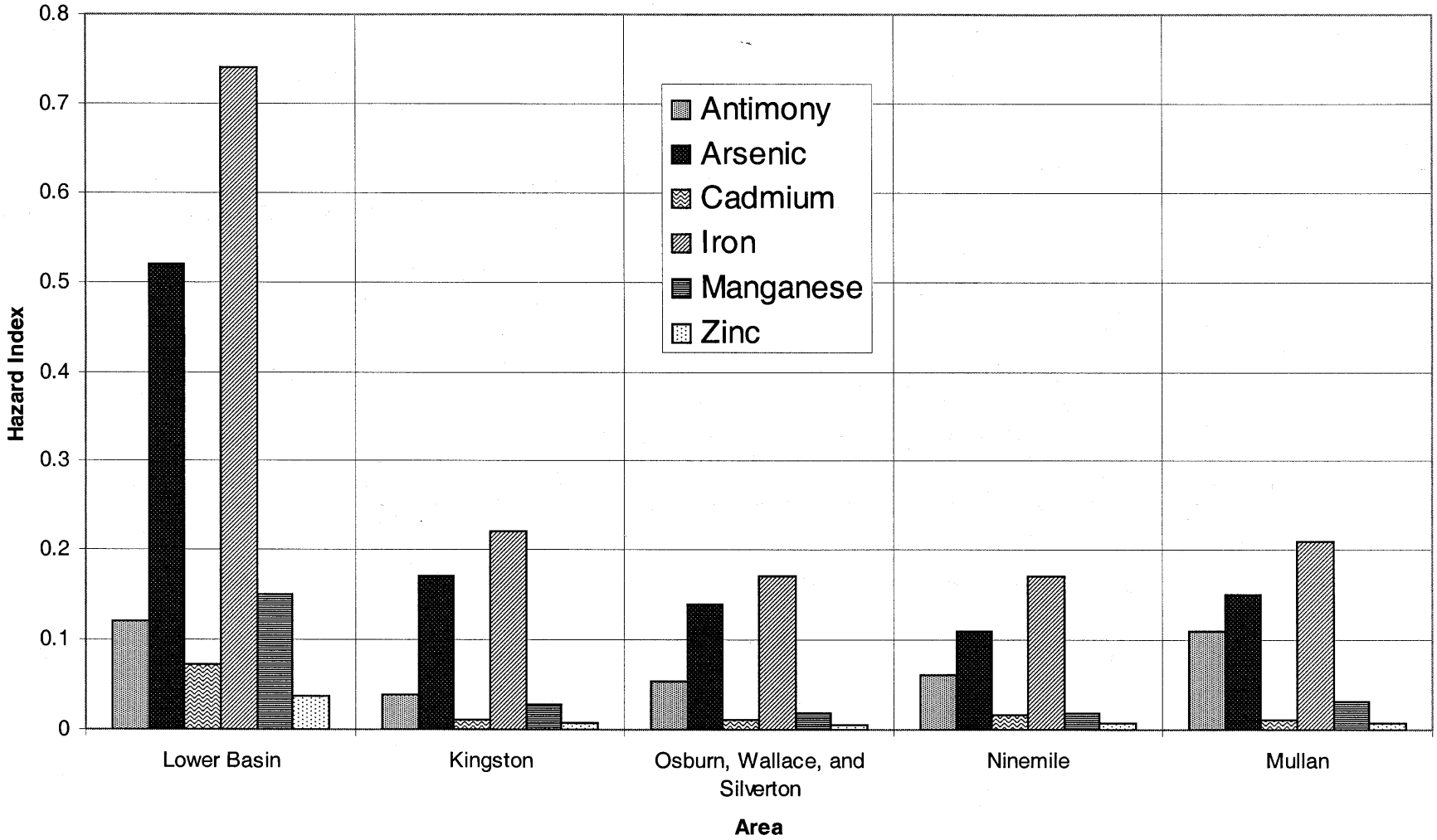
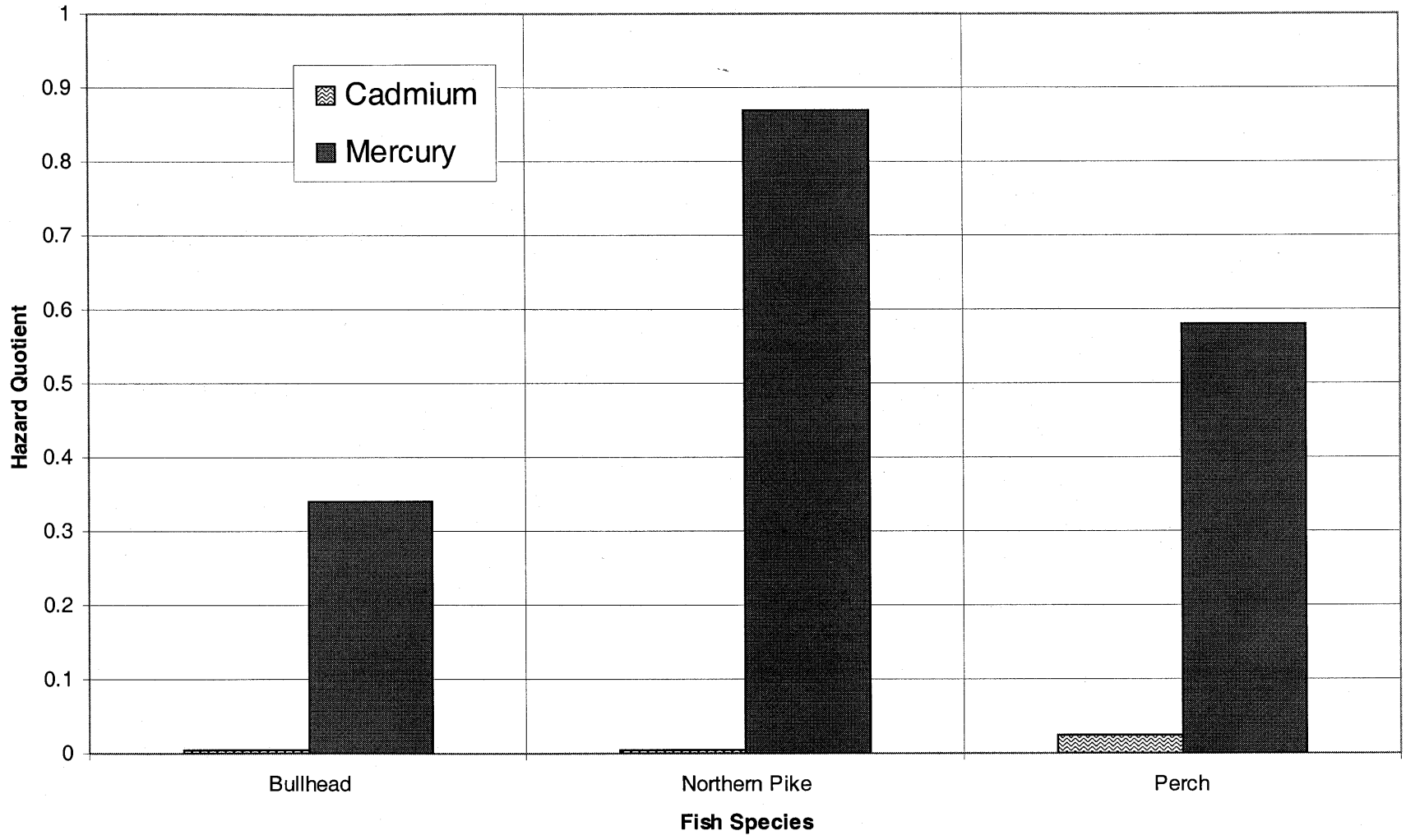
Figure 5-10
RME Noncancer Hazard - Public Recreational (Child 0 - 6), by Chemical



EPA REGION 10	027-RI-CO-102Q Coeur d'Alene Basin RI/FS BASELINE HHRA	Doc Control: Generation: 1 BHHRA Series 3/12/01	**Figure 5-11** **RME Noncancer Hazard - Occupational (Adult), by Chemical**



EPA REGION 10	027-RI-CO-102Q Coeur d'Alene Basin RI/FS BASELINE HHRA	Doc Control: Generation: 1 BHHRA Series 3/12/01	**Figure 5-12a** **RME Noncancer Hazard - Recreational Fish Consumption (Adult)**

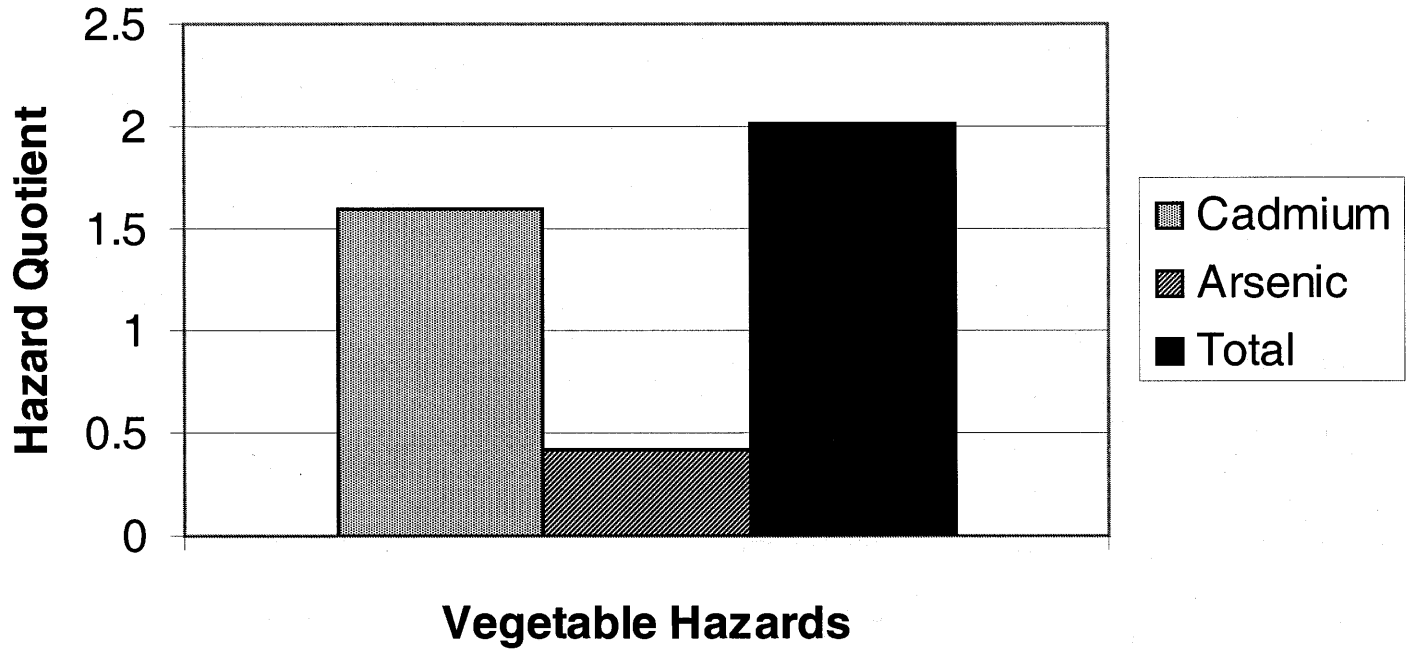


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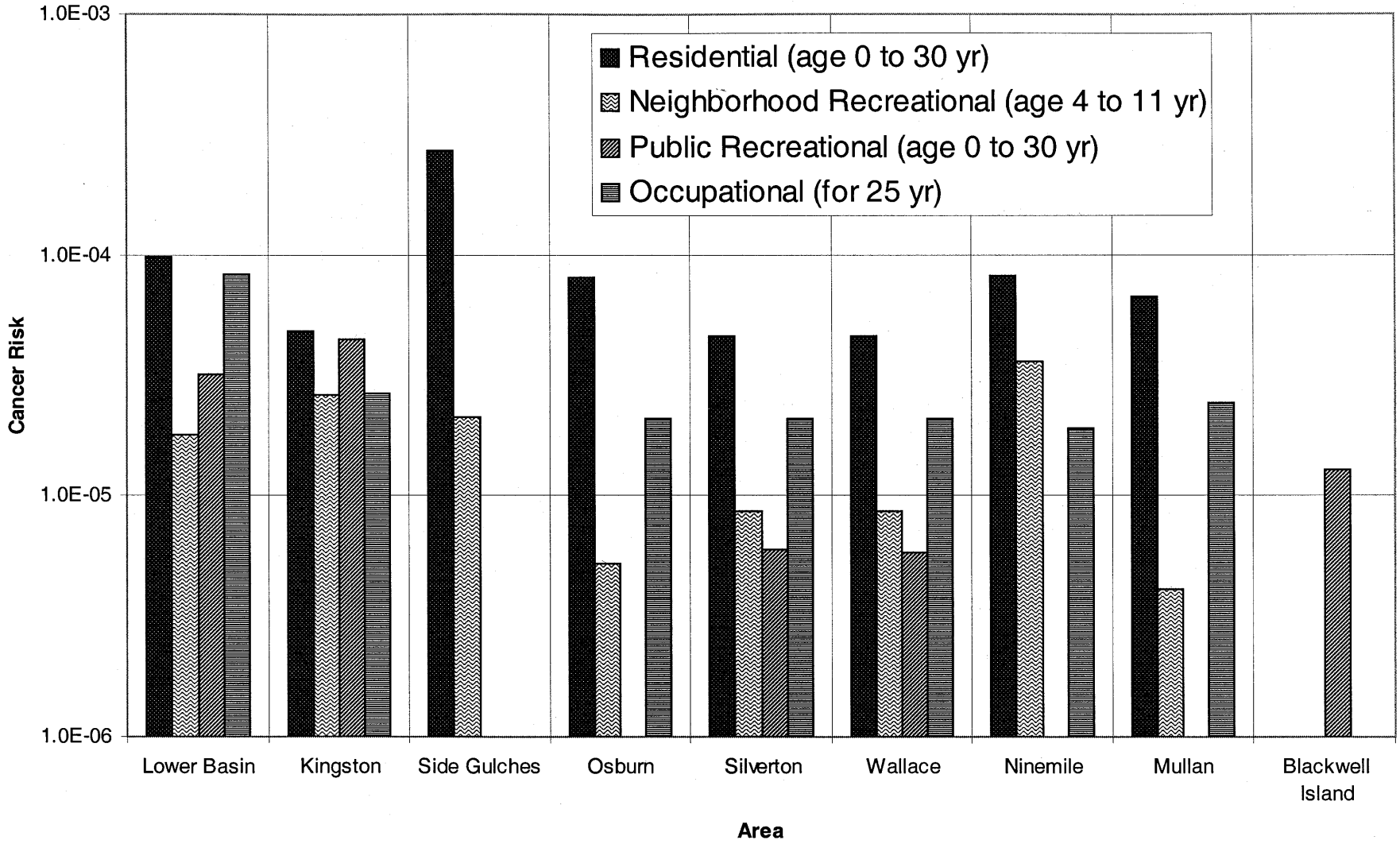
Figure 5-12b
RME Noncancer Hazard - Residential Vegetable Consumption



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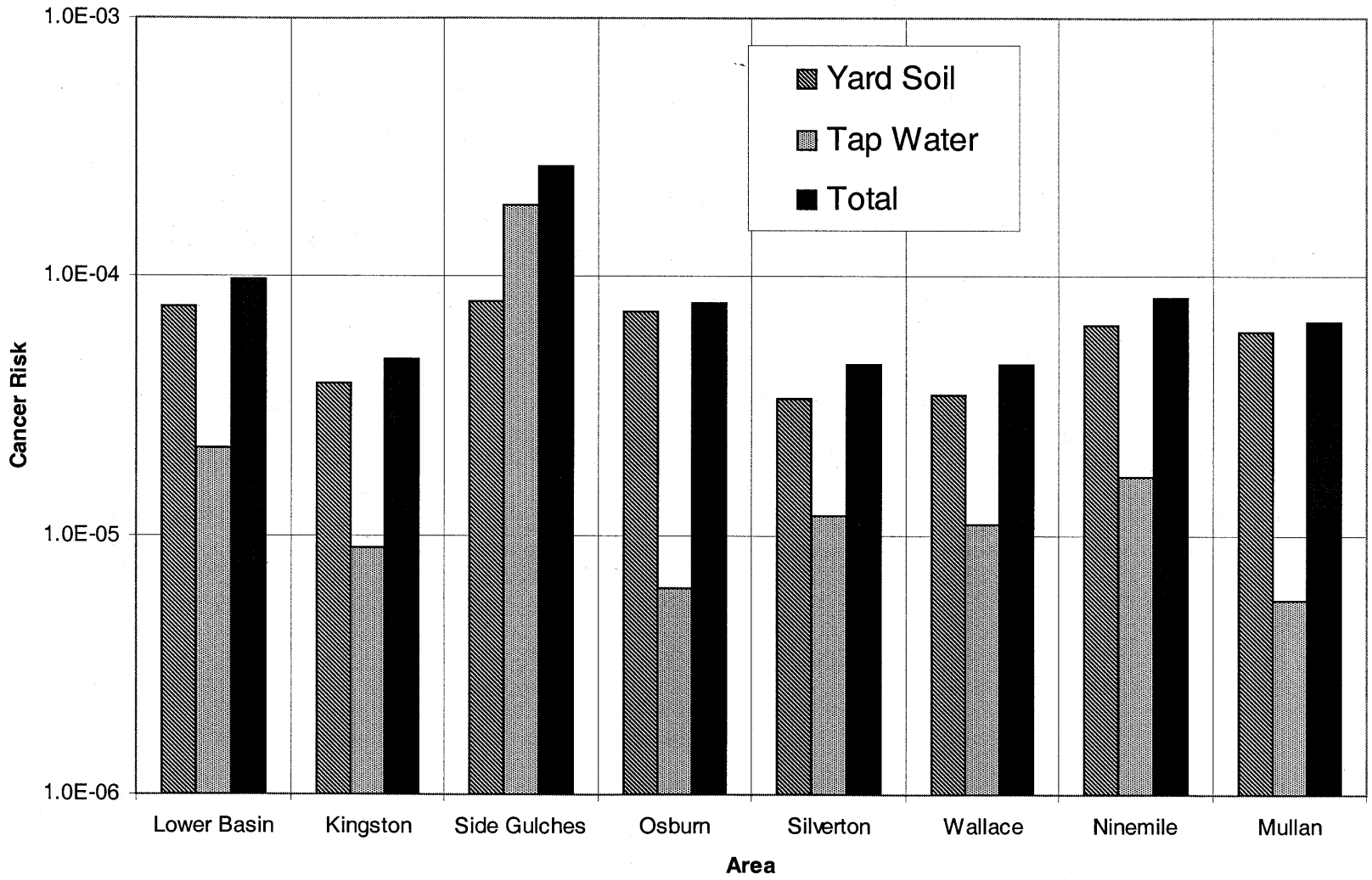
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc Control:
Generation: 1

BHHRA Series
3/12/01

Figure 5-13
Summary of RME Cancer Risk



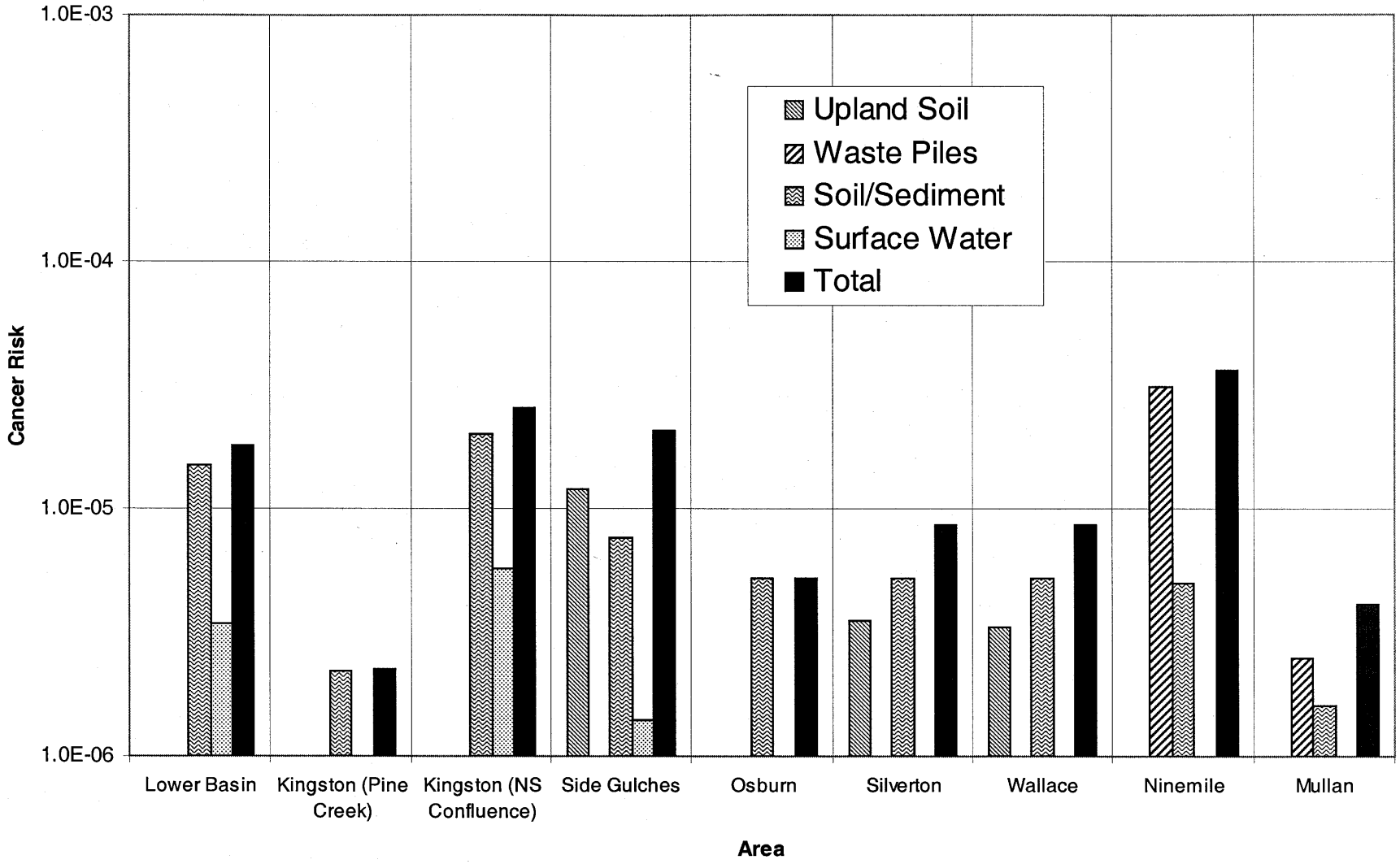
EPA REGION 10

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

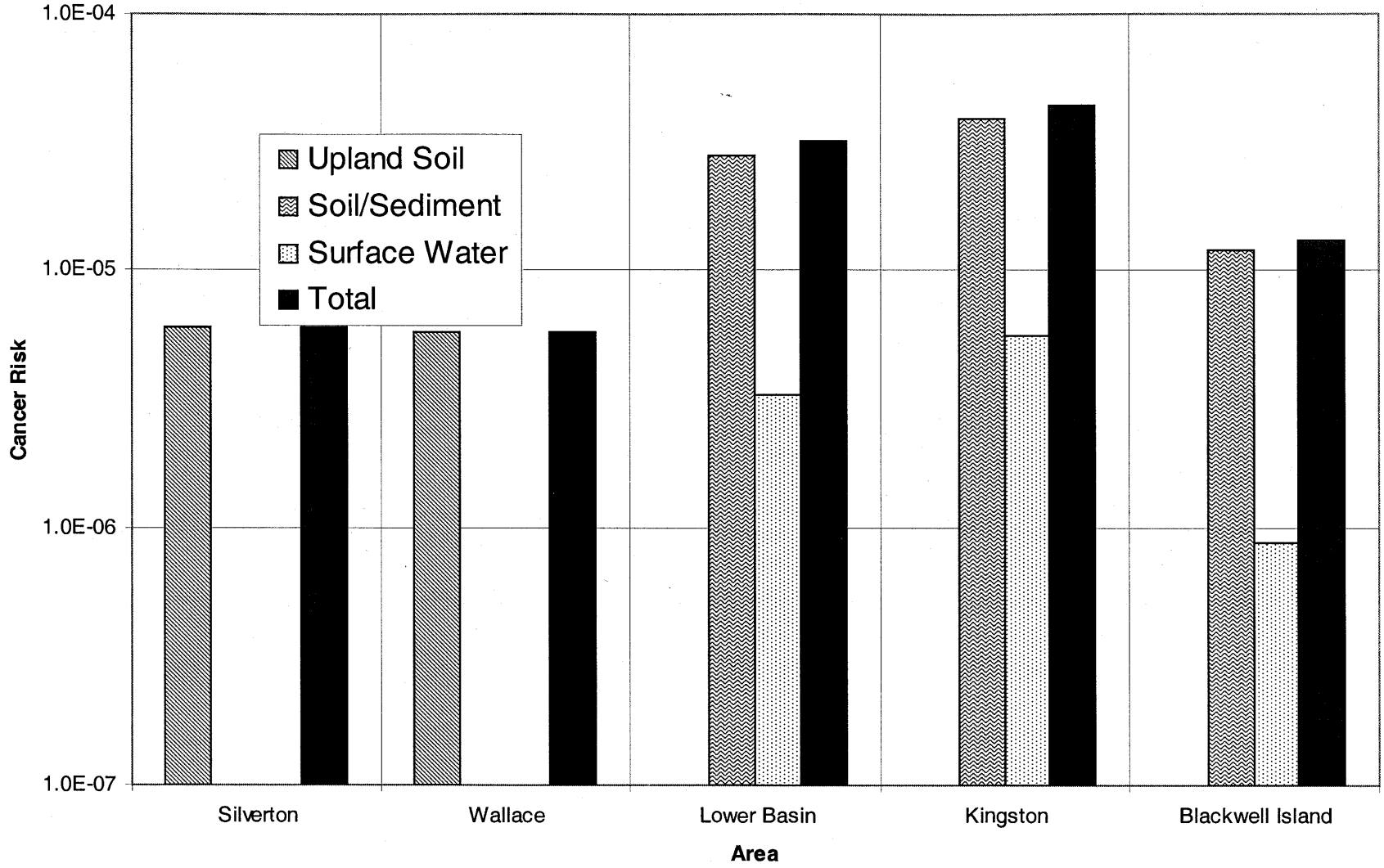
Doc Control:
Generation: 1

BHHRA Series
3/12/01

Figure 5-14
RME Cancer Risk - Residential



EPA REGION 10	027-RI-CO-102Q Coeur d'Alene Basin RI/FS BASELINE HHRA	Doc Control: Generation: 1 BHHRA Series 3/12/01	**Figure 5-15** **RME Cancer Risk - Neighborhood Recreational (Child 4-11)**



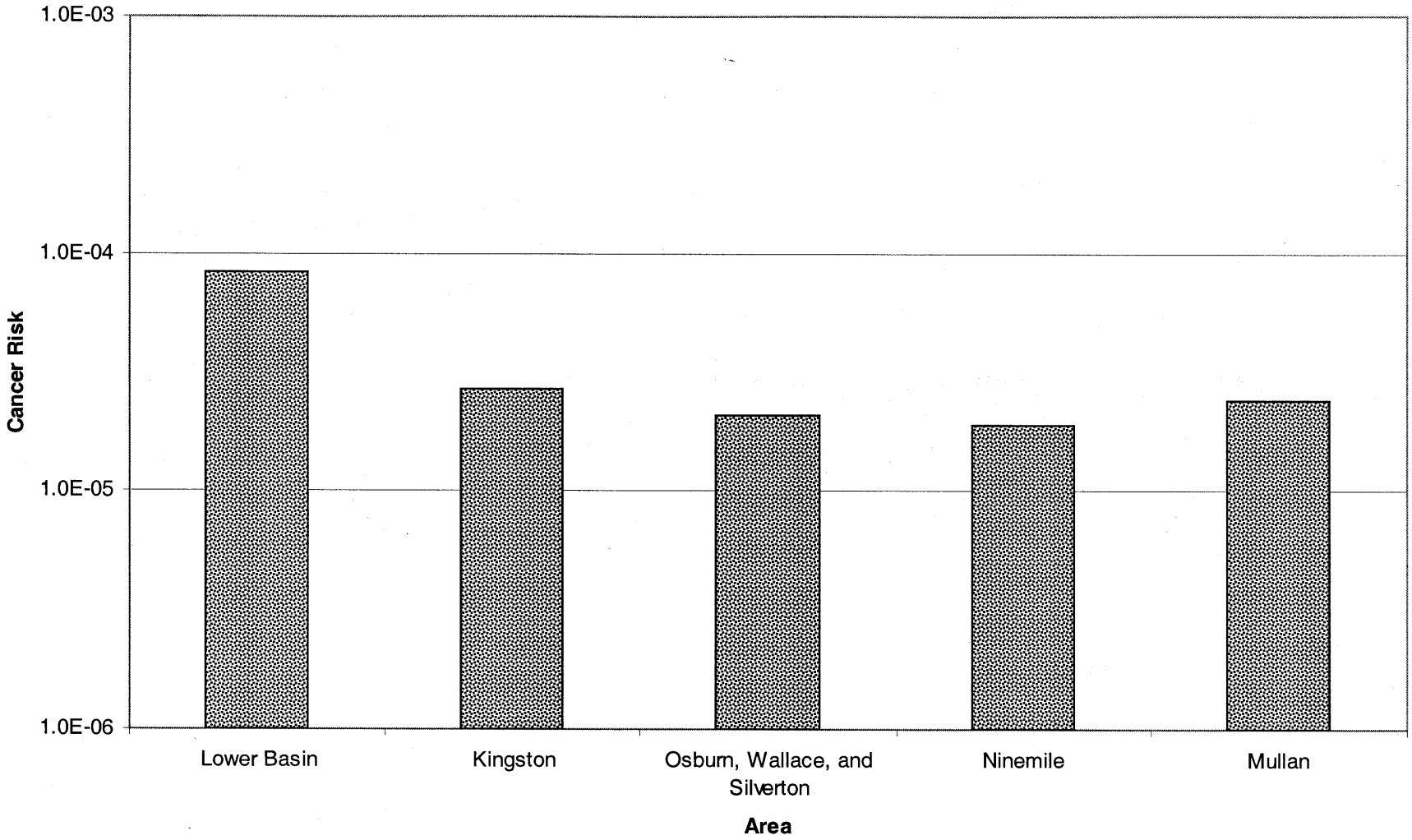
EPA REGION 10

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc Control:
Generation: 1

BHHRA Series
3/12/01

Figure 5-16
RME Cancer Risk - Public Recreational



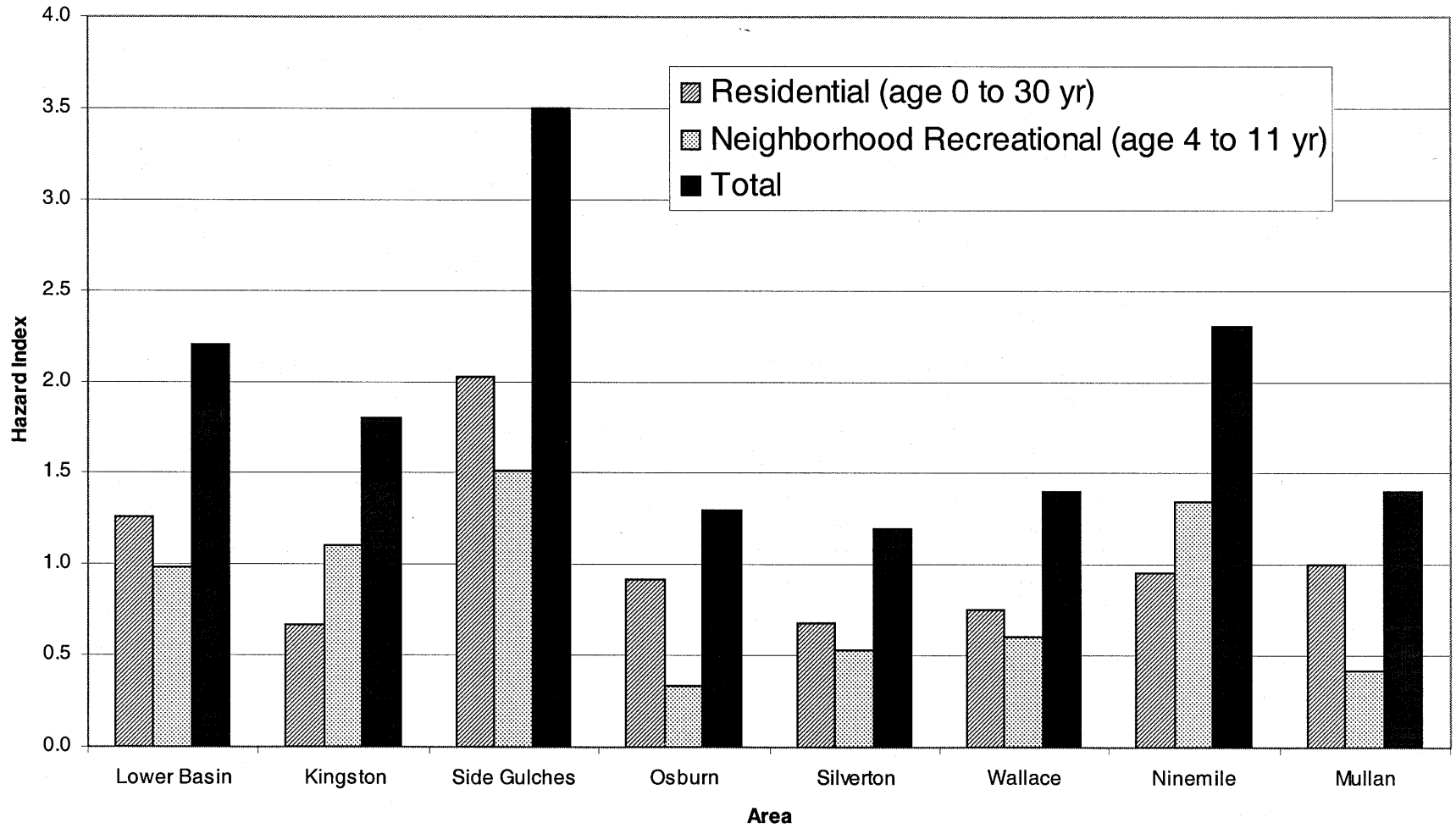
EPA REGION 10

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

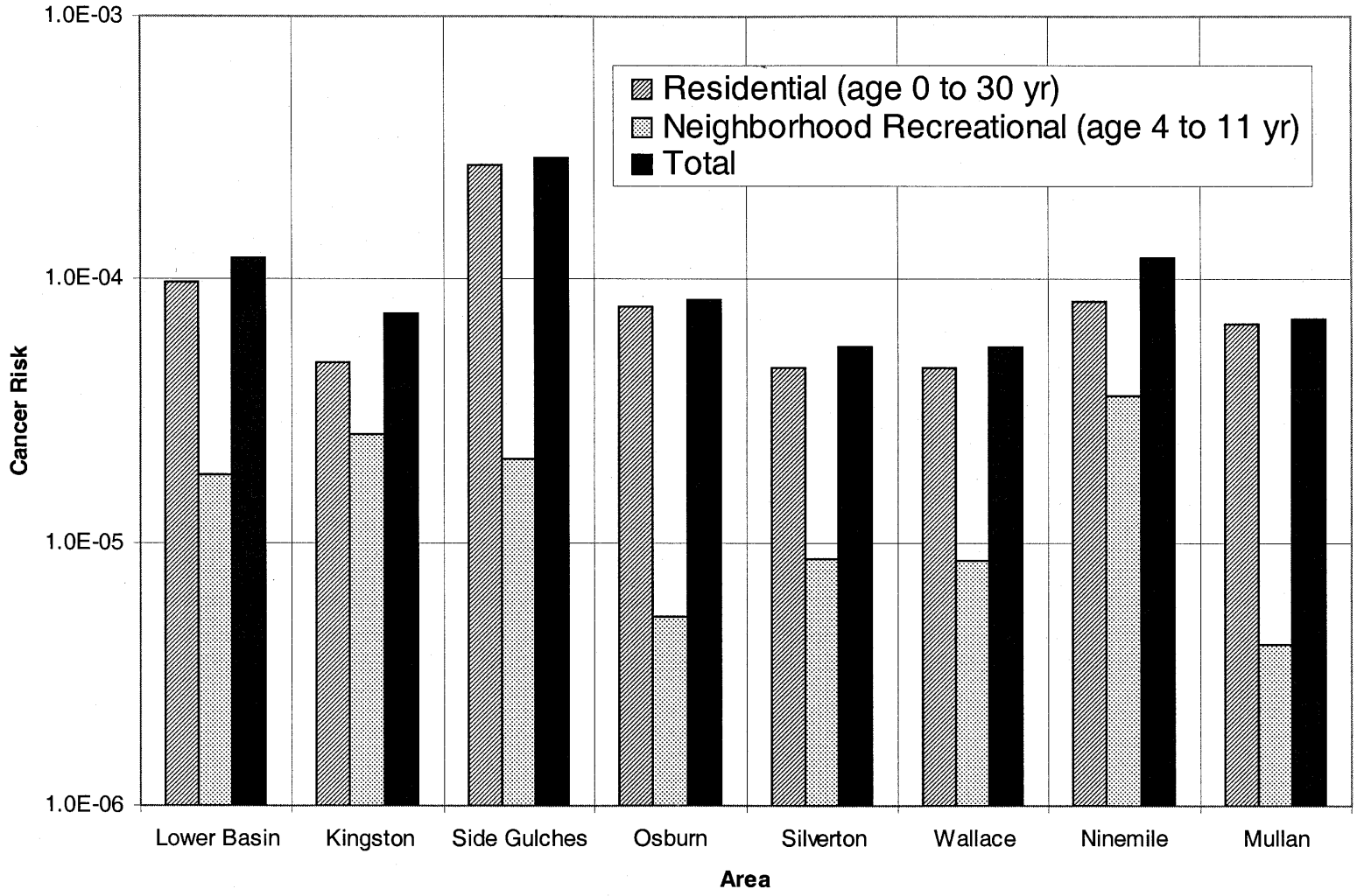
Doc Control:
Generation: 1

BHHRA Series
3/12/01

Figure 5-17
RME Cancer Risk - Occupational (Adult)



EPA REGION 10	027-RI-CO-102Q Coeur d'Alene Basin RI/FS BASELINE HHRA	Doc Control: Generation: 1 BHHRA Series 3/12/01	**Figure 5-18** **RME Noncancer Hazard - Residential Plus Neighborhood Recreational, All Chemicals**



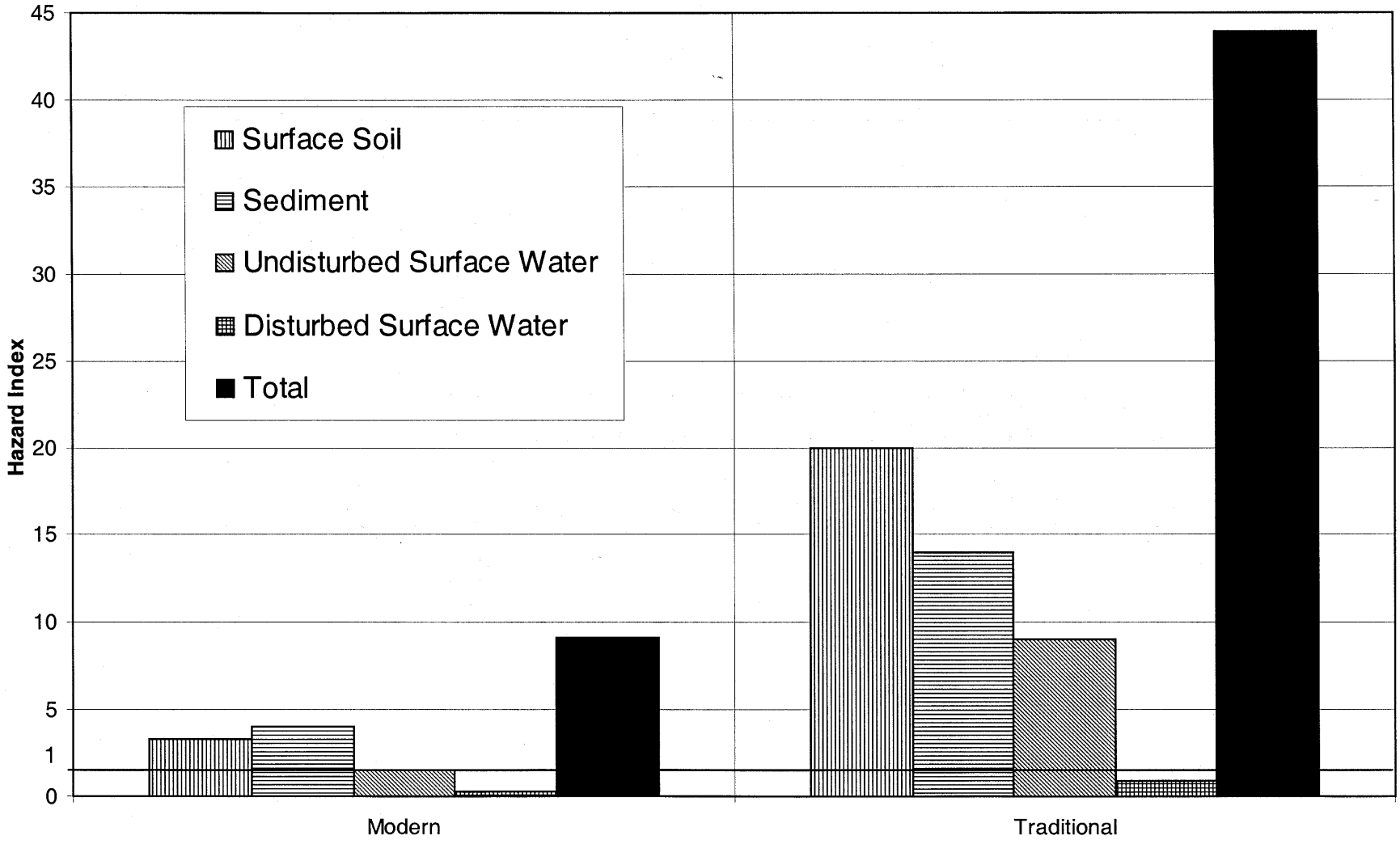
EPA REGION 10

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc Control:
Generation: 1

BHHRA Series
3/12/01

Figure 5-19
RME Cancer Risk - Residential Plus Neighborhood Recreational



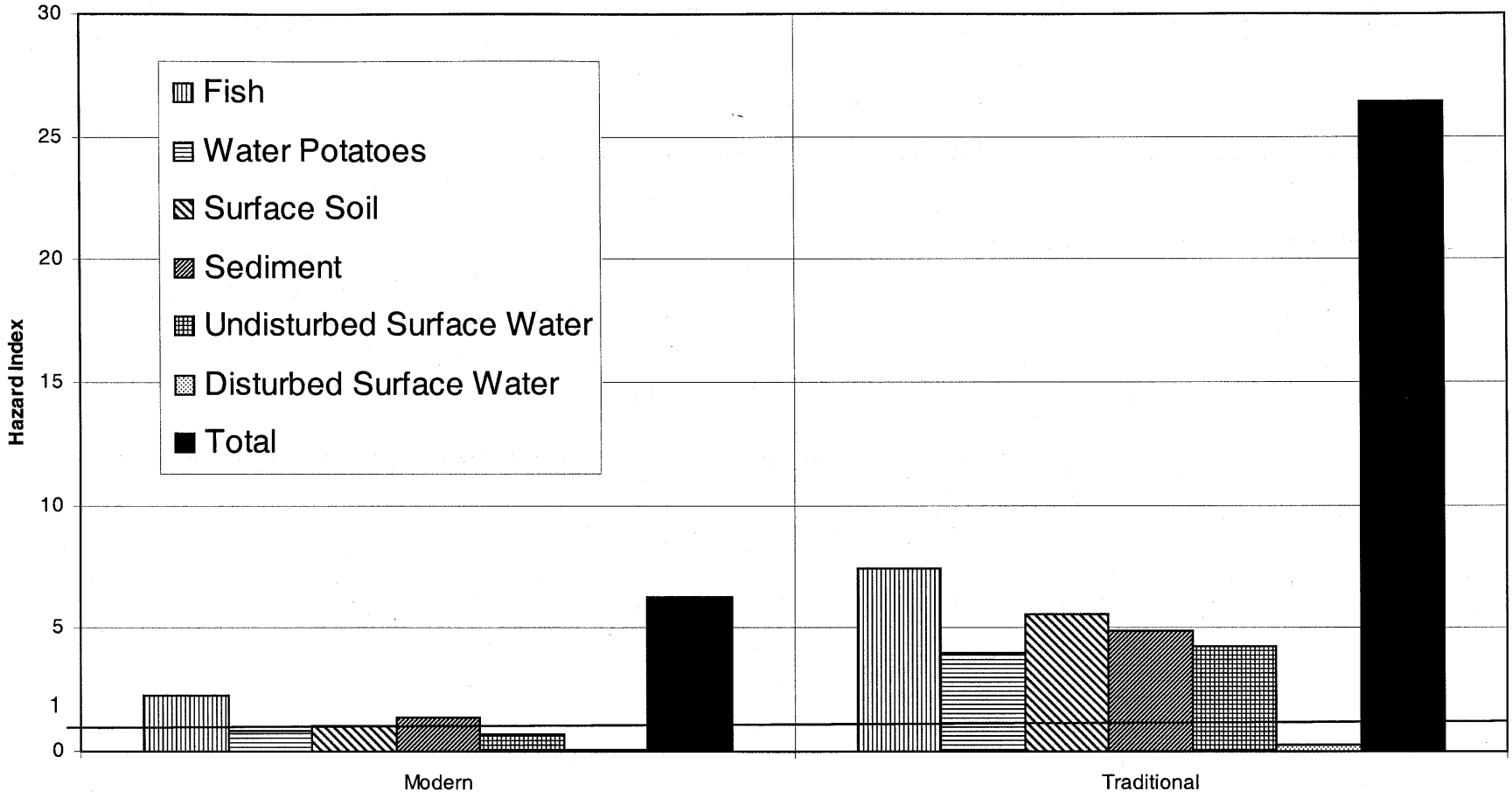
EPA REGION 10

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc Control:
Generation: 1

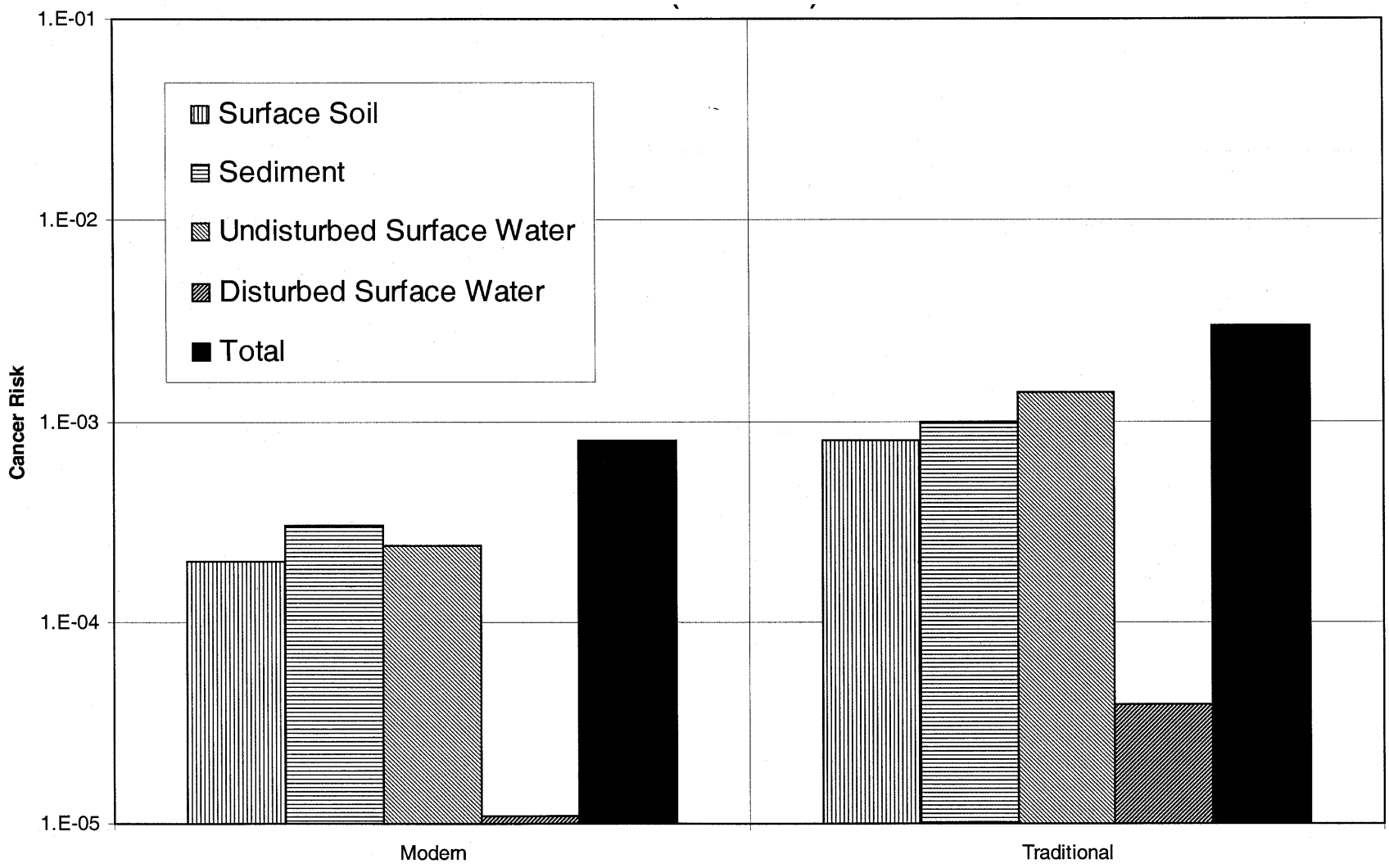
BHHRA Series
3/12/01

Figure 5-20
Total RME Noncancer Hazard - Modern and Traditional Subsistence Exposure Scenarios
All Chemicals (Child Age 0 to 6 years)



Note: The fish ingestion pathway was evaluated for the Adult only receptor age group, all other pathways were evaluated for the combined Adult/Child receptor age group.

EPA REGION 10	027-RI-CO-102Q Coeur d'Alene Basin RI/FS BASELINE HHRA	Doc Control: Generation: 1 BHHRA Series 3/12/01	**Figure 5-21** **Total RME Noncancer Hazard - Modern and Traditional Subsistence Exposure Scenarios** **All Chemicals (Adult/Child)**



EPA REGION 10

027-RI-CO-102Q
Coeur d'Alene Basin RI/FS
BASELINE HHRA

Doc Control:
Generation: 1

BHHRA Series
3/12/01

Figure 5-22

Total RME Cancer Risk - Modern and Traditional Subsistence Exposure Scenarios (Adult/Child)

Table 5-1
Summary of Hazard/Risk Estimates and Risk Drivers

Exposure Scenario/ Receptors	Total Hazard/Risk				RME Risk Drivers				
	CT		RME		Arsenic		Iron	Cadmium	Zinc
	HI	CR	HI	CR	HI (Media)	CR (Media)	HI (Media)	HI (Media)	HI (Media)
Lower Basin									
Child residents	1	NC	**4**	NC	1 (yard soil)	NC	**2 (yard soil)**	—	—
Child/adult residents	0.4	**9E-06**	1	**1E-04**	—	**7E-05 (yard soil)**	—	—	—
Child neighborhood recreational	0.3	**2E-06**	1	**2E-05**	—	**1E-05 (soil/sediment)**	—	—	—
Child visitors[a]	0.4	NC	**2**	NC	0.5 (soil/sediment)	NC	0.6 (soil/sediment)	—	—
Child/adult visitors[a]	0.1	**3E-06**	0.5	**3E-05**	—	**3E-05 (soil/sediment)**	—	—	—
Construction workers	0.2	**3E-06**	**2**	**8E-05**	0.5 (subsurface/ surface soil)	**8E-05 (subsurface/ surface soil)**	0.7 (subsurface/ surface soil)	—	—

[a]Visitors refer to public recreational receptors.
[b]Based on sediment and surface water in the South Fork for Osburn, Wallace, and Silverton combined.
[c]Based on sediment and surface water in the South Fork for Osburn, Wallace, and Silverton combined and surface soil in upland parks and schools in Silverton.
[d]Based on sediment and surface water in the South Fork for Osburn, Wallace, and Silverton combined and surface soil in upland parks and schools in Wallace.
[e]MidGradSeg0l includes the towns of Osburn, Wallace, and Silverton. Risk and hazard estimates are applicable to construction workers on projects in any of the towns.
[f]Individual pathway not included in the total risk and hazard estimates for the exposure scenarios and receptors. See text discussion.
[g]Based on Northern Pike, the species with the highest concentrations.
[h]Based on sediment, surface water, and wastepiles.
Notes:
Bold value indicates HI exceeds 1 or CR exceeds 1E-06.
— - not a risk driver
CR - cancer risk CT - central tendancy
HI - hazard index NC - not calculated
RME - reasonable maximum exposure

Table 5-1 (Continued)
Summary of Hazard/Risk Estimates and Risk Drivers

Kingston									
Child residents	0.7	NC	**2**	NC	0.6 (yard soil)	NC	1 (yard soil)	—	—
Child/adult residents	0.2	**5E-06**	0.7	**5E-05**	—	**4E-05 (yard soil)**	—	—	—
Child neighborhood recreational	0.3	**2E-06**	1	**3E-05**	—	**2E-05 (soil/sediment)**	—	—	—
Child visitors[a]	0.4	NC	**2**	NC	0.7 (soil/sediment)	NC	0.6 (soil/sediment)	—	—
Child/adult visitors[a]	0.1	**3E-06**	0.6	**4E-05**	—	**4E-05 (soil/sediment)**	—	—	—
Construction workers	0.06	9E-07	0.5	**3E-05**	—	**3E-05 (subsurface/ surface soil)**	—	—	—
Side Gulches									
Child residents	1	NC	**6**	NC	**3 (yard soil/ tap water)**	NC	1 (yard soil)	—	—
Child/adult residents	0.5	**2E-05**	**2**	**3E-04**	1 (yard soil/ tap water)	**3E-04 (yard soil/ tap water)**	0.3 (yard soil)	—	—
Child neighborhood recreational	0.3	1E-06	**2**	**2E-05**	0.4 (soil/sediment)	**2E-05 (Elk Creek soil/Elk Creek Pond sediment)**	—	—	—
Osburn									
Child residents	1	NC	**3**	NC	1 (yard soil)	NC	1 (yard soil)	—	—
Child/adult residents	0.3	**9E-06**	0.9	**8E-05**	—	**7E-05 (yard soil)**	—	—	—
Child neighborhood recreational[b]	0.07	3E-07	0.3	**5E-06**	—	**5E-06 (sediment)**	—	—	—

Table 5-1 (Continued)
Summary of Hazard/Risk Estimates and Risk Drivers

Silverton									
Child residents	0.7	NC	**2**	NC	0.6 (yard soil)	NC	0.9 (yard soil)	—	—
Child/adult residents	0.2	**5E-06**	0.7	**5E-05**	—	**3E-05 (yard soil)**	—	—	—
Child neighborhood recreational[c]	0.1	5E-07	0.5	**9E-06**	—	**8E-06 (surface soil/sediment)**	—		
Child visitors[a]	0.06	NC	0.3	NC	—	NC	—	—	—
Child/adult visitors[a]	0.02	3E-07	0.09	**6E-06**	—	**6E-06 (surface soil)**	—	—	—
Wallace									
Child residents	0.9	NC	**3**	NC	0.6 (yard soil)	NC	0.9 (yard soil)	—	—
Child/adult residents	0.3	**6E-06**	0.8	**5E-05**	—	**4E-05 (yard soil)**	—	—	—
Child neighborhood recreational[d]	0.1	6E-07	0.6	**8E-06**	—	**8E-06 (surface soil/sediment)**	—		
Child visitors[a]	0.09	NC	0.5	NC	—	NC	—	—	—
Child/adult visitors[a]	0.03	4E-07	0.1	**6E-06**	—	**6E-06 (surface soil)**	—	—	—
MidGradSeg0l[e]									
Construction workers	0.05	7E-07	0.4	**2E-05**	—	**2E-05 (subsurface/ surface soil)**	—	—	—
Mullan									
Child residents	1	NC	**3**	NC	1 (yard soil)	NC	1 (yard soil)	—	—
Child/adult residents	0.3	**6E-06**	1	**7E-05**	—	**6E-05 (yard soil)**	—	—	—
Child neighborhood recreational[h]	0.06	2E-07	0.4	**4E-06**	—	**4E-06 (waste piles/sediment)**	—	—	—
Construction workers	0.07	8E-07	0.5	**2E-05**	—	**2E-05 (subsurface/ surface soil)**	—	—	—

Table 5-1 (Continued)
Summary of Hazard/Risk Estimates and Risk Drivers

Nine Mile									
Child residents	1	NC	**3**	NC	1 (yard soil)	NC	1 (yard soil)	—	—
Future child residents	**10**	NC	**22**	NC	—	NC	—	**17 (groundwater)**	**4 (groundwater)**
Child/adult residents	0.3	**8E-06**	1	**8E-05**	—	**7E-05 (yard soil)**	—	—	—
Future child/adult residents	**5**	**3E-06**	**12**	**3E-05**	—	**3E-05 (groundwater)**	—	**9 (groundwater)**	**2 (groundwater)**
Child neighborhood recreational[h]	0.1	7E-07	1	**4E-05**	0.7 (waste piles)	**3E-05 (waste piles)**	—	—	—
Construction workers	0.05	6E-07	0.4	**2E-05**	—	**2E-05 (subsurface/ surface soil)**	—	—	—
Blackwell Island									
Child visitors[a]	0.1	NC	0.7	NC	—	NC	—	—	—
Child/adult visitors[a]	0.05	1E-06	0.2	**1E-05**	—	**1E-05 (soil/sediment)**	—	—	—
Homegrown Vegetables[f]									
Child residents	0.1	NC	**2**	NC	—	NC	—	2	—
Child/adult residents	0.1	**2E-06**	**2**	**8E-05**	—	**8.00E-05**	—	2	—
Fish[g]									
Adult visitors[a]	0.4	NC	0.9	NC	—	NC	—	—	—

Table 5-2
Chemicals With Hazard Indices Greater Than or Equal to 1

Exposure Scenario/Receptors	Chemical	Hazard Index
Lower Basin		
RME residential child	Arsenic, iron	2, 2
Side Gulches		
RME residential child	Arsenic, iron	3, 1
RME residential child/adult	Arsenic	1
Osburn		
RME residential child	Arsenic	1
Burke/Nine Mile		
RME current/future residential child	Arsenic	1
RME future residential child (groundwater only)	Cadmium, zinc	17, 4
RME future residential child/adult (groundwater only)	Cadmium, zinc	9, 2
Mullan		
RME residential child	Arsenic, iron	1, 1
Homegrown Vegetables		
RME residential child	Cadmium	2
RME residential child/adult	Cadmium	2

Notes:
Individual pathway not combined with hazards from other exposure scenarios or receptors. See text discussion.
See also Figures 5-4 and 5-5.

Table 5-3
Summary of Hazard/Risk Estimates for Combined Child/Adult Residential and Neighborhood Recreational Scenarios

Total Hazard/Risk		RME Risk Drivers		
RME		Arsenic		Iron
HI	CR	HI (Media)	CR (Media)	HI (Media)
Lower Basin				
2	**1E-04**	0.7 (Yard soil/soil/sediment)	**8E-05 (Yard soil)**	0.7 (Yard soil/soil/sediment)
Kingston				
2	**8E-05**	0.6 (Yard soil/soil/sediment)	**6E-05 (Yard soil/soil/sediment)**	1 (Yard soil/soil/sediment)
Side Gulches				
4	**3E-04**	**2 (Yard soil/tap water/upland surface soil)**	**3E-04 (Yard soil/tap water)**	0.7 (Yard soil/soil)
Osburn				
1	**9E-05**	—	**7E-05 (Yard soil)**	—
Silverton				
1	**6E-05**	—	**4E-05 (Yard soil/upland surface soil/sediment)**	—
Wallace				
1	**6E-05**	—	**4E-05 (Yard soil)**	—
Mullan				
1	**7E-05**	—	**6E-05 (Yard soil)**	—
Burke/Nine Mile				
2	**1E-04**	1 (Yard soil/waste piles)	**1E-04 (Yard soil/tap water/waste piles)**	0.4 (Yard soil)

Notes:
Bold value indicates HI exceeds 1 or CR exceeds 1E-06.
— - not a risk driver
CR - cancer risk
HI - hazard index
RME - reasonable maximum exposure

Table 5-4 Summary of RME Hazard/Risk Extimates and Risk Drivers for Modern Subsistence Exposure Scenario

Exposure Medium	Exposure Pathway	Receptor Age Group	Total Hazard / Risk		RME Risk Drivers					
			HI	CR	Arsenic		Cadmium	Iron	Manganese	Mercury
					HI	CR	HI	HI	HI	HI
Fish										
Bullhead	Ingestion	Adult	1	--	--	--	--	--	--	1
Northern Pike			**3**	--	--	--	--	--	--	**3**
Perch			**2**	--	--	--	--	--	--	**2**
Water Potato[a]	Ingestion	Child/Adult	0.8	--	--	--	0.8	--	--	--
Surface Soil	Dermal	Child	0.3	--	0.3	--	--	--	--	--
		Child/Adult	0.2	**6.E-05**	0.1	**6.E-05**	--	--	--	--
	Ingestion	Child	**3**	--	0.8	--	--	1	0.6	--
		Child/Adult	0.8	**1.E-04**	0.2	**1.E-04**	--	0.3	--	--
Sediment	Dermal	Child	0.8	--	0.7	--	--	--	--	--
		Child/Adult	0.5	**2.E-04**	0.4	**2.E-04**	--	--	--	--
	Ingestion	Child	**3**	--	0.8	--	--	1	0.7	--
		Child/Adult	0.9	**1.E-04**	0.2	**1.E-04**	--	0.4	--	--
Undisturbed Surface Water	Ingestion	Child	1	--	1	--	--	--	--	--
		Child/Adult	0.7	**2.E-04**	0.5	**2.E-04**	--	--	--	--
Disturbed Surface Water	Ingestion	Child	0.3	--	--	--	--	--	--	--
		Child/Adult	0.1	**1.E-05**	--	**1.E-05**	--	--	--	--
Total		Adult[b]	**3**	--	--	--	0.1	--	--	**3**
		Child	**9**	--	**4**	--	0.5	**2**	**2**	0.1
		Child/Adult	**4**	**7.E-04**	**2**	**7.E-04**	1	1	0.5	0.1

[a]The water potato hazard listed in the table is for unpeeled water potatoes. The hazard for peeled water potatoes is 0.5.

[b]Total hazard for the Adult only age group is based on Nothern Pike. The species with the highest concentration.

Notes:
Bold value indicates HI exceeds 1 or CR exceeds 1E-06.
-- - Either not calculated or not a risk driver.
CR - cancer risk
HI - hazard index
RME - reasonable maximum exposure

Table 5-5 Summary of RME Hazard/Risk Estimates and Risk Drivers for Traditional Subsistence Exposures

Exposure Medium	Exposure Pathway	Receptor Age Group	Total Hazard/Risk		RME Risk Drivers						
					Antimony	Arsenic		Cadmium	Iron	Manganese	Mercury
			HI	CR	HI	HI	CR	HI	HI	HI	HI
Fish											
Bullhead	Ingestion	Adult	**4**	--	--	--	--	--	--	--	**4**
Northern Pike			**10**	--	--	--	--	--	--	--	**10**
Perch			**7**	--	--	--	--	--	--	--	**7**
Water Potato[a]	Ingestion	Child/Adult	**4**	--	--	--	--	**4**	--	--	--
Surface Soil	Dermal	Child	**2**	--	--	**2**	--	--	--	--	--
		Child/Adult	0.5	**2.E-04**	--	--	**2.E-04**	--	--	--	--
	Ingestion	Child	**17**	--	1	**5**	--	0.6	**7**	**4**	--
		Child/Adult	**5**	**6.E-04**	--	**1**	**6.E-04**	--	**2**	1	--
Sediment	Dermal	Child	**3**	--	--	**2**	--	--	--	--	--
		Child/Adult	**2**	**7.E-04**	--	**2**	**7.E-04**	--	--	--	--
	Ingestion	Child	**11**	--	0.7	**3**	--	0.5	**4**	**3**	--
		Child/Adult	**3**	**4.E-04**	--	1	**4.E-04**	--	1	0.7	--
Undisturbed Surface Water	Ingestion	Child	**9**	--	--	**7**	--	0.7	--	0.8	0.8
		Child/Adult	**4**	**1.E-03**	--	**3**	**1.E-03**	--	--	--	0.4
Disturbed Surface Water	Ingestion	Child	0.9	--	--	0.3	--	--	--	0.4	--
		Child/Adult	0.2	**4.E-05**	--	--	**4.E-05**	--	--	--	--
Total		Adult[b]	**10**	--	--	--	--	0.4	--	--	**10**
		Child	**43**	--	**2**	**19**	--	**2**	**11**	**8**	0.8
		Child/Adult	**19**	**3.E-03**	0.5	**7**	**3.E-03**	**5**	**3**	**2**	0.4

[a]The water potato hazard listed in the table is for unpeeled water potatoes. The hazard for peeled water potatoes is 2.

[b]Total hazard for the Adult only age group is based on Nothern Pike. The species with the highest concentration.

Notes:
Bold value indicates HI exceeds 1 or CR exceeds 1E-06.
-- - Either not calculated or not a risk driver.
CR - cancer risk
HI - hazard index
RME - reasonable maximum exposure

Table 5-6 Potential Preliminary Remediation Goals for Arsenic

	Residential Soil Ing. and Dermal (child 0-6) mg/kg	Residential Soil Ing. and Dermal (child/adult) mg/kg	Public Recreational Soil/Sed Ing. and Dermal (child 0-6) mg/kg	Public Recreational Soil/Sed Ing. and Dermal (child/adult) mg/kg	Neighborhood Recreational Waste Pile Ing. And Dermal (child 4-11) mg/kg	Neighborhood Recreational Soil/Sed Ing. And Dermal (child 4-11)- Lower Basin and Kingston mg/kg	Neighborhood Recreational Soil/Sed Ing. And Dermal (child 4-11)- All other areas mg/kg
Arsenic - Cancer (10^{-4} risk)		64		420	1663	815	1016
Arsenic - Cancer (10^{-5} risk)		6		42	166	81	102
Arsenic - Cancer (10^{-6} risk)		1		4	17	8	10
Arsenic - Noncancer	35	123	234	810	748	367	457

Table 5-7 Summary of the Percent of Basin Residences with 95 Percent UCL Arsenic Concentrations Exceeding Selected Potential PRGs

	Total Number of homes sampled for yard soil*	Percent of homes ≥ 35 (mg/kg)	Percent of homes ≥ 64 (mg/kg)	Percent of homes ≥ 123 (mg/kg)
Lower Basin	12	25%	17%	0%
Kingston	28	14%	4%	0%
Side Gulches	31	42%	23%	6%
Osburn	52	44%	21%	15%
Silverton	23	17%	9%	4%
Wallace	36	36%	6%	3%
Nine Mile	34	29%	12%	6%
Mullan	26	23%	15%	4%
CdA Basin	242	31%	14%	6%

* Subsequent residential sampling efforts have taken place since the generation of residential EPCs used in this Baseline HHRA. The total number of homes is based upon the total number of homes sampled in the Basin for which yard soil data is available to date, including these subsequent residential samplings.

6.0 CHARACTERIZATION OF LEAD HEALTH RISK

6.1 INTRODUCTION/METHODOLOGY

The approach to human health risk assessment for lead differs from that of other metals in several ways. It is important to note those differences and how the methodologies employed relate to the Coeur d'Alene Basin population. Among the important considerations are the nature of the health effects, the behavior of lead in the body, measurements of biological effects, indices of risk, how risks are quantified, availability of data (both site-specific and in the national experience), and the relationships between absorption levels and environmental media.

The lead health effects of greatest concern, at the environmental and blood lead levels observed in the Basin, are subtle and sub-chronic in nature. That is, the adverse effects of low-level lead poisoning can result from relatively short-term exposures on the order of months, as opposed to periods of years to lifetime for other metals. Similarly the effects of lead poisoning are less likely to be diagnosed in a clinical setting and often go undetected.

The adverse health effects of lead have been related to blood lead concentration or micrograms of lead per deciliter of whole blood (µg/dl). As a result, blood lead levels have evolved as indices of health criteria. Currently, 10 µg/dl has been identified as the national level of concern for young children and pregnant women. The health effects observed at a blood lead level of 10 µg/dl are sub-clinical, meaning that, generally, these effects cannot be diagnosed in an individual child. Establishment of these sub-clinical health effects of lead was based on numerous scientific studies involving comparisons of large groups of children (NRC 1993, ATSDR 1999b, and CDC 1997).

Lead health risk assessment evaluates potential for these adverse effects to occur by comparison of observed or predicted blood lead levels to these standards. Risks to population groups are assessed by determining the expected or observed percentage of the population to exceed those criteria. Risk to individuals is often expressed as the probability that the subject's blood lead level will exceed the specified level (i.e., 10 µg/dl).

Public health authorities have developed policies indicating the acceptability of certain probabilities or percentages of populations exhibiting blood lead levels in excess of the criteria. Those policies are presented in this Section 6.2. For example, the U.S. Environmental Protection Agency's current remediation goal, with respect to children, is that no child in an identifiable population has a greater than 5% probability of a blood lead level of 10 µg/dl or greater.

As a result, lead health risk assessment involves measuring and modeling blood lead levels for relatively short-term exposures and relating those to national criteria. Pharmaco-kinetic models have been developed to predict blood lead levels resulting from different (lead) intake scenarios. These models have evolved to assist risk assessors in estimating population blood lead levels for differing environmental situations (USEPA 1994d, 1998f) and are attached as Appendix O. Detailed descriptions can be found on-line at http://www.epa.gov/superfund/programs/lead/prods.htm#guidance.

These models are especially useful in multiple source situations, such as the Basin, where individuals can be exposed to lead in many aspects of their lives. Coupled with site-specific measurements of blood lead levels and environmental exposure levels and the experience of the nearby BHSS, these models can be useful tools in aiding the understanding of the complex mechanisms involved in lead poisoning. The same findings can then be used to devise and implement response strategies to reduce risks and minimize the potential for lead absorption among the local population.

This type of analysis is performed in this Section for the resident population. Results from site-specific lead exposure studies, surveys of lead absorption and follow-up results for local children, and special environmental sampling have been combined with modeling efforts to provide a comprehensive analysis of lead health risks for the resident population in the Basin.
However, this approach was not possible with respect to the lead health risk concerns for the Coeur d'Alene Tribe. There are no site-specific human health biological data to evaluate. Tribal members abandoned traditional, historic subsistence practices in the River environment a century ago because of high levels of contamination. Currently available blood lead models were developed for suburban/urban applications and have not been tested nor applied to Native American traditional activities. As a result, risk assessment for potential subsistence activities by Tribal members is limited to estimating intake rates and qualitatively comparing those, to rates of other populations. However, it is clear that a subsistence-based lifestyle requires environmental lead levels orders of magnitude lower than those measured throughout the floodplain of the Coeur d'Alene River.

6.1.1 Lead Health Risk Assessment for the Resident Population

Two approaches to the lead Human Health Risk Assessment (HHRA) have been conducted to assist risk managers, the local communities, and public health officials in developing health protective strategies to minimize the future incidence of lead poisoning among the resident population in the Basin. These two methods are generally referred to in this document as ***conventional*** (e.g., a predictive, mechanistic approaches to blood lead modeling) and ***site-specific*** analysis (e.g., a descriptive, empirical approach). The conventional Integrated Exposure Uptake Bio-kinetic Model (IEUBK) approach is intended to be predictive of future, potential blood lead levels associated with a site. Empirical comparisons have documented a reasonable concordance between IEUBK Model predictions and observed blood lead levels (Hogan et al. 1998). Other studies have been critical of the IEUBK's predictive capabilities (Carroll and Galindo 1998, Biesada and Hubicki 1999, Griffin et al. 1999). This model has been effectively utilized to establish cleanup criteria and monitor remedial effectiveness at the Bunker Hill Superfund Site (BHSS) for more than a decade (TerraGraphics 2000a) (See Appendix Q). The site-specific method more accurately describes past blood lead trends; its predictive value for future blood leads may be contingent on continuing public health intervention activities to monitor blood lead levels and reduce exposure. Both of these methods attempt to relate potential lead poisoning, as measured by blood lead content, to levels of lead in the environment.

In any assessment of human health risk in a community setting, basic steps are undertaken to characterize and evaluate the potential health problems. Those individuals and groups that are most sensitive, or most at-risk, to lead poisoning are identified. In this case, young children and women of reproductive age (as they might expose the unborn child) are the ***sensitive population***. The suspect ***sources of lead*** are identified. In the Basin, the principal sources are mining industry wastes and lead-based paint used on both the inside and outside of homes. Less prevalent sources include the historic use of leaded gasoline, plumbing, and other consumer goods that contain lead. Over the years, these sources have contaminated other ***environmental media*** that can now be contacted by children and women of reproductive age. In the Basin, ***soils and house dust*** are contaminated by historic discharges of mining industry waste, deteriorating paint, or past use of leaded gasoline. Soils may include home yards, roadsides and driveways, schoolyards, play grounds, recreational locations, the river floodplain, and other contaminated areas. ***Drinking water*** may be contaminated by mine wastes or use of lead solder in plumbing. Contaminated dust in the ***air*** can contribute to lead intake through inhalation. Soils and dust can also contaminate ***food*** from local gardens. Supermarket food may also contain lead.

Once these sources are known, the ***routes of exposure*** or pathways by which children or adults may contact and ingest this lead are identified. The routes of exposure of greatest concern in the Basin are children consuming soil and dust in their everyday activities, contaminated food, and drinking water. A variety of soil and dust media may be involved, and the lead in the soils and dust may come from different sources. Children consume interior dust in their homes, schools or day cares; and soils from home yards and play areas. Adults consume soil in both their home, and recreational and occupational environments. The lead in these soils and dusts can originate from paint, mine waste piles, tailings in the river flood plain, leaded gasoline combustion, deposition of airborne dust, and other potential sources. Lead exposure pathways are identified and media concentration data are summarized in Section 6.3.

In ***conventional risk assessment***, estimates are developed of how much lead is ingested (or taken into the body) through soils, dust, water, and food. This is accomplished by multiplying the amount of soil, dust, water, and food, etc., that children and adults consume, by the lead concentration in these media. This value is usually calculated in units of micrograms of lead ingested per day (µg/day) and is called the ***lead intake.*** The IEUBK Model for lead is used to estimate the mean (i.e., geometric mean) ***blood lead level*** expected for a typical child consuming that amount of lead. However, all children do not respond in the same way to the same intake of lead; some have higher blood lead levels and others have lower. The IEUBK Model also predicts the percentage of children likely to exceed a given blood lead level. The exceedance predictions are calculated using an empirically derived measure of blood lead variability (i.e., geometric standard deviation) for a group of similarly exposed children. Intake rates are calculated and presented in Section 6.5. Blood lead estimates are developed in Section 6.6.

The United States Centers for Disease Control (CDC) determined that blood lead levels greater than 10 µg/dl present an undue risk of damaging health effects; the USEPA has established a national goal of

no more than 5% of children in any community exceeding a 10 µg/dl blood lead level. The EPA guidance refers to both limiting exposure and determining soil lead concentrations so that "a typical (or hypothetical) child or group of children would have an estimated risk of no more than 5% of exceeding a blood lead of 10 µg/dl" (USEPA 1998f, USEPA 1994d). The IEUBK Model predicts that percentage of children (6-84 months) expected to exceed the 10 µg/dl blood lead health standard. If the estimated risk of children having blood lead levels greater than or equal to 10 µg/dl exceeds 5%, then corrective measures should be undertaken to reduce that risk. Lead health risks are characterized in Section 6.7.

Site-specific analysis of risk in this report refers to the collection of blood lead data from the resident population and ***relating the observed blood lead levels to measured concentrations in environmental media*** collected during the time of the study. These studies seek to establish that percentage of the population that is actually experiencing lead poisoning and characterize the direct link between lead in blood and the various media. This is generally accomplished by conducting well controlled investigations that collect both blood lead and environmental source data and relating those through statistical techniques.

In the site-specific approach, an actual measurement of the percent of children to exceed 10 µg/dl is obtained and relationships between blood lead and soil, dust, paint, water, and socioeconomic factors can be evaluated. Generally, these types of studies are cross-sectional and designed to describe relationships between environmental lead levels and levels of lead measured in blood for the population, at a particular point in time. Both the conventional and site-specific approaches have been used at Superfund sites throughout the country and are consistent with USEPA guidance (USEPA 1989, 1994a). This risk assessment employs both methodologies. However, USEPA guidance requires that any site-specific analysis be based on compelling scientific evidence, collected in controlled investigations that are representative of the population of concern, the contaminated media, and the routes and pathways of lead exposure that are, or could be, occurring in the future.

The majority of blood lead data available for the Coeur d'Alene Basin are observational and opportunistic based on voluntary participation in health response programs and were not solicited for experimental or survey purposes. Blood lead levels are not, nor were ever, intended to be randomized, and the blood lead database is limited to less than 1/3 of the 9 month to 9 year old population and less than 20% of pre-school children. As a result, the representativeness of this self-selected population to all the children in the Basin is unknown, and the true prevalence of blood lead levels exceeding 10 µg/dl cannot be determined with these data. Because there is not a representative database for blood lead levels, this variable cannot be used to quantitatively assess the risk or probability of exceeding blood lead criteria in the overall population, especially for young children.

Although the blood lead database does not represent the overall population, it is can be considered representative of those children that were sampled. Additionally, a representative sample of environmental media was obtained in 1996 and has been substantially supplemented to near 50% of all

homes in the Basin. These data can be used to develop a hybrid risk assessment technique that uses the site-specific analysis to develop input parameters for the IEUBK Model that are particular to this population in the Coeur d'Alene Basin. As a result, several analyses of the available data to support site-specific amendments to the IEUBK model are presented in this report. The latter approach was accomplished at the BHSS in 1990 and the "Box Model"was used to develop the cleanup strategy employed there over the last decade. A detailed explanation of the technical basis for the Box Model is attached as Appendix Q. Existing blood lead data are summarized in Section 6.2, and site-specific quantitative analyses are presented in Section 6.4. Strict application of current EPA policy requires applying the default mode of the IEUBK in addition to any site-specific analysis. Both the default and site-specific IEUBK analyses are presented in Section 6.6.

6.1.2 Lead Health Risk Assessment for the Coeur d'Alene Tribe

For the purposes of this assessment, Coeur d'Alene Tribal authorities have requested that two specific tribal exposure scenarios be investigated, developed, and utilized within the Coeur d'Alene Basin (CDAB) Human Health Risk Assessment (HHRA). Those scenarios are the ***Traditional Tribal Subsistence Lifestyle,*** representing the aboriginal existence, and the ***Modern Subsistence Lifestyle***, representing tribal members practicing modern subsistence activities. Both lifestyles were characterized in Section 3.1.3, and appropriate tribal exposure factors and consumption rates were developed. In large part, the various tribal exposure pathways and consumption rates included for use by the Coeur d'Alene Tribe were initially developed from research conducted with the Umatilla Tribe in Northeastern Oregon (Harris and Harper 1997). The results of that research were subsequently used within the Hanford Screening Assessment under a subsistence resident scenario (CRCIA 1996).

Research specific to the Coeur d'Alene Tribe has been conducted to evaluate the appropriateness of the Umatilla Tribal exposure factors in the HHRA. Adjusted exposure parameters extrapolated from the Hanford Screening Assessment are presented in Tables 3-26a-b. These exposure parameters are consistent with standard approaches to human health risk assessment, and are intended to be protective of traditional Coeur d'Alene Tribal riparian zone subsistence activities. The Traditional Tribal Subsistence exposure factors presented were developed assuming that the lifestyle involved residence within the floodplain for almost the entire year. The Coeur d'Alene Tribe has also requested that exposure factors and consumption rate information be developed and considered for modern tribal members utilizing traditional hunting/gathering activities and a subsistence diet in today's environment (Modern Subsistence Scenario). The Modern Subsistence Scenario does not assume permanent residence within the floodplain.

Tribal exposure pathways are discussed in Sections 3.2.4 and 6.5.1. Lead intake for select pathways are developed in Section 6.5.4. Potential lead absorption is discussed in Section 6.6.4 and risks are discussed in Section 6.7.5.

6.2 OBSERVED BLOOD LEAD LEVELS

6.2.1 Blood Lead Health Criteria

Centers for Disease Control Advisory Regarding Children's Blood Lead Levels

The Centers for Disease Control have outlined three major areas for development of policies and activities related to childhood lead poisoning prevention. These areas are primary prevention activities, secondary prevention activities, and monitoring (surveillance). Primary prevention activities include evaluation and control of residential lead-based paint hazards, public lead education, professional lead education and training, anticipatory guidance by child health-care providers, and identification and control of sources of lead exposure other than lead-based paint. Primary prevention activities are aimed to prevent children from being exposed to lead (CDC 1997).

The Centers for Disease Control have the following secondary prevention guidelines regarding child blood lead levels (adapted from CDC 1997, Table 4.3):

C	blood lead concentration <10 µg/dl	Re-assess or re-screen in one year. No additional action is necessary unless exposure sources change.
C	blood lead concentration 10-14 µg/dl	Provide family lead education. Provide follow-up testing. Refer for social services, if necessary.
C	blood lead concentration 15-19 µg/dl	Provide family lead education. Provide follow-up testing. Refer for social services, if necessary. If blood lead levels persist or worsen (i.e., 2 venous blood lead levels in this range at least three months apart), proceed according to actions for blood lead concentrations in the 20-44 µg/dl range.
C	blood lead concentration 20-44 µg/dl	Provide coordination of care (case management), clinical management, environmental investigation, and lead- hazard control.
C	blood lead concentration 45-69 µg/dl	Within 48 hours, begin coordination of care (case management), clinical management,

		environmental investigation, and lead- hazard control.
C	blood lead concentration ≥70 µg/dl	Hospitalize child and begin medical treatment immediately. Begin coordination of care (case management), clinical management, environmental investigation, and lead-hazard control immediately.

U.S. Environmental Protection Agency Policy regarding Children's Blood Lead Levels

At lead contaminated residential sites, EPA seeks assurance that the health of the most susceptible population (children and women of child bearing age) is protected and promotes a program that pro-actively assesses and prevents unacceptable exposures to lead. EPA believes that predictive tools should be used to evaluate the risk of lead exposure, and that cleanup actions should be designed to address both current and potential future risk. For this reason, cleanup decisions can be made on IEUBK predicted blood lead levels alone. Blood lead monitoring provides useful and complementary data to Model results. Blood lead monitoring data is invaluable to initiate treatment and intervention for children with elevated lead levels, but is of limited use in developing remedial action criteria. This EPA policy is attached as Appendix O (USEPA 1994d, USEPA 1998f). The policy is also available online at http://www.epa.gov/superfund/programs/lead/prods.htm#guidance.

To meet these objectives, EPA seeks actions that limit exposure to soil lead levels such that a typical child or group of similarly exposed children would have an estimated risk of no more than 5% exceeding a blood lead level of 10 µg/dl.

EPA emphasizes the use of the IEUBK Model for estimating risks for childhood lead exposure from a number of media, such as soils, dust, air, water, and other sources to predict blood lead levels in children 6 months through 84 months old. EPA recommends that the IEUBK Model be used as the primary tool to generate risk-based soil cleanup levels at lead sites for current or future residential land use. Response actions can be taken using IEUBK Model predictions alone; blood lead studies are not required.

Blood lead studies and surveys are useful tools at lead sites and can be used to identify key site-specific exposure pathways and to direct health professionals to individuals needing immediate assistance in minimizing lead exposure; however, EPA recommends that blood lead studies not be used for establishing long-term remedial or non-time-critical removal cleanup levels at lead sites.

It is recommended that risk assessments conducted at lead-contaminated residential sites use the individual residence as the primary exposure unit of concern. This does not mean

that a risk assessment should be conducted for every home yard, rather that the soil lead concentration data from yards and other residential media (for example, interior dust and drinking water) should be input into the IEUBK Model to provide a preliminary remediation goal (PRG) for the residential setting. When applicable, potential exposure to accessible site-related lead sources outside the residential setting should also be evaluated to understand how these other potential exposures contribute to the overall risk to children, and to suggest appropriate cleanup measures for those areas (USEPA 1998f).

Bunker Hill Superfund Site Remedial Action Objectives

Site wide Remedial Action Objectives (RAOs) are defined in the Populated and Non-populated Records of Decision (RODs) for the Bunker Hill Superfund Site (BHSS) (USEPA 1991c, USEPA 1992b). The blood lead RAOs were defined in the 1991 Populated Areas ROD and were based on site-specific blood lead levels among children and environmental media lead concentrations at the site; the RAOs defined for the BHSS were developed prior to publication of the 1994 EPA guidance that outlines the current blood lead criteria (USEPA 1994d). The blood lead RAOs at the BHSS seek to reduce the incidence of lead poisoning in the community to the following levels:

- less than 5% of children with blood lead levels of 10 µg/dl or greater, and
- no individual child exceeding 15 µg/dl (nominally, <1% of the population).

These objectives are to be achieved by a strategy that includes:

i) Remediating all residential yards, commercial properties, and Rights-of-way (ROWs) that have lead concentrations greater than 1000 milligrams of lead per kilogram of soil (mg/kg);
ii) Achieving a geometric mean yard soil concentration of less than 350 mg/kg for each community in the site;
iii) Controlling fugitive dust and stabilizing and covering contaminated soils throughout the site; and
iv) Achieving a geometric mean interior house dust lead concentrations of less than 500 mg/kg for each community in the site.

The success of the BHSS strategy depends on reduction of interior house dust lead levels to concentrations comparable to post-remedial area soils. If house dust lead levels remain elevated, within the BHSS, homes with concentrations greater than 1000 mg/kg will be considered for interior remediation. Differences between the BHSS RAOs and contemporary EPA lead policies are the result of guidance issued after the BHSS ROD (USEPA 1994d, 1998f). Key distinctions are:

i) Protection against the risk of an elevated blood lead to an individual at the 10 µg/dl level (instead of 15 µg/dl on BHSS);

ii) Focus prevention efforts and risk criteria on younger children (6 to 84 months of age instead of up to 9 years);

iii) The percentage of children exceeding 10 µg/dl can be estimated from blood lead surveys in the BHSS as participation rates exceed 50% and reasons for failure to participate are known for the majority of the remaining resident children (TerraGraphics 2000a).

6.2.2 State of Idaho / Panhandle Health District Children's Blood Lead Survey Results

Sources of Site-specific Blood Lead Observations

Surveys of blood lead levels in the Basin have been conducted in each of the four summers from 1996 to 2000. Results from the 2000 survey are not included in this document. The initial survey was conducted in association with the *Coeur d'Alene River Basin Environmental Health Exposure Assessment* (IDHW 2000). Participants in the 1996 study were solicited at their homes for both blood lead samples and an environmental survey of the residence. Most of those families contacted, consented to the environmental survey and samples were collected from 843 homes. A total of 667 adults and 98 children aged 9 months through 9 years provided blood lead samples. These data were analyzed and discussed in the parent document (IDHW 2000).

Subsequent fixed-site blood lead screening was offered in both 1997 and 1998. In 1997, 26 children aged 9 months through 9 years responded. Eleven of these children had provided samples in the previous year. In 1998, parents of 128 children opted to participate in the program. In both years children were offered $20 to provide a sample. No renumeration was offered in 1996. Despite the increased turnout, the relatively small number of participants caused concern among public health authorities that several children with high blood lead levels among the Basin wide population were going undetected, and that the results may not be representative of non-participants. A more aggressive solicitation was accomplished in 1999. Government and mining industry officials agreed to jointly support a fixed-site screening and each participant was offered $40 to provide a blood sample. Turnout was considerably larger as a result of the increased solicitation efforts and incentives. A total of 272 children in the target age group provided samples in 1999. This represents approximately 24% to 27% of the estimated 1025 to 1120 children in the Basin study area. However, participation continued to be age-biased and several children were repeats from earlier years.

The turnout for children aged 0-5 was less than 20% as compared to near 30% for 6-9 year old children. Although a total of 524 blood lead observations were compiled in the four surveys from 1996 to 1999, some children had repeat blood draws. There were a total of 424 individual children from 247 households from the four surveys (Table 6-1b). Eighty-one (81) children from 57 homes were tested more than once. Sixty-five (65) of those children were tested twice, 13 were tested three times and 3 were tested in each of the four years. Of those children tested more than once, 11 had levels greater than 10 µg/dl and received intervention services from the local public health program. Seven (7)

of these children had lower blood lead levels in subsequent testing, 1 had the same level, and 3 had higher levels. The children tested more than once tended to have lower than average levels for children in their age group on the first test and similar levels on subsequent testing. Of the 81 children tested more than once, 21 had higher than average blood lead levels for their age group, 51 had lower, 9 had average levels. These results would indicate that some observations used in the risk analysis had lower blood lead levels than might be obtained in a random sampling of the population.

Blood lead summary data for children 9 months through 9 years old, by year and geographic subareas, and the numbers of children targeted for follow-up health response services are presented in Tables 6-1a and 6-2. Figures 6-1a-b show mean blood lead levels and the percent of children to exceed the 10 µg/dl and 15 µg/dl health criteria by geographic area for both the Basin and the BHSS.

Site-specific Blood Lead Results for Children

Table 6-1a shows Basin wide summary results for each of the four surveys. The overall geometric mean blood lead level for the 1997 to 1999 surveys was 4.2 µg/dl compared to 4.0 µg/dl in the 1996 survey, indicating consistency in the overall level of absorption in recent years. The percentage of children with levels greater than or equal to 10 µg/dl ranged from 9% to 15%, and averaged 10% for the four year period, also indicating consistency across the four years. About 5% of children have had levels 15 µg/dl or greater, and between 1% and 2% of children exhibited blood lead concentrations greater than 20 µg/dl. These latter results also seem consistent across the four year period despite the variation in turnout.

Follow-up services include counseling by a public health nurse and an environmental exposure assessment of the child's residence. Of the 58 children exhibiting blood lead levels greater than 10 µg/dl, 50 follow-up investigations were successfully completed and the findings are summarized in Section 6.2.3.

Blood Lead Levels and Percent to Exceed Toxicity Criteria by Geographic Subarea

Mean blood lead levels and the percent of children to exceed 10 µg/dl differ by Basin subarea. Mean blood lead levels and the number and percentage to exceed toxicity criteria for subareas are shown in Tables 6-2 and 6-3. The highest levels are observed in the upper Silver Valley or easternmost portions of the study area. Arithmetic mean concentrations for the four year period are 7.4 µg/dl, 6.0 µg/dl and 5.2 µg/dl for Burke/Nine Mile, Wallace, and Mullan respectively. Arithmetic mean levels range from 4.1 µg/dl to 4.9 µg/dl in the intermediate areas from Silverton downstream to Kingston. The arithmetic mean concentration for the Lower Basin/Cataldo subarea is 5.5 µg/dl. Geometric mean blood lead levels follow a similar pattern.

The percent of children to exceed toxicity criteria shown in Table 6-3 also follows a similar pattern to mean concentration. The highest toxicity rates are observed in Burke/Nine Mile at 21% exceeding 10

µg/dl, 13% exceeding 15 µg/dl, and 4% with levels of 20 µg/dl or greater. The Lower Basin/Cataldo subarea showed the next highest toxicity rate with 18% exceeding 10 µg/dl and 5% greater than the 15 µg/dl criteria. No children were in the 20 µg/dl range in the Lower Basin. Wallace, Mullan and Silverton, respectively, showed 13%, 11% and 8% of children with levels of 10 µg/dl or greater. From 4% to 5% of children tested in Silverton and Wallace exhibited blood lead levels exceeding the 15 µg/dl criteria, and 1% in Silverton and 3% in Wallace exceeded 20 µg/dl. Osburn and the Side Gulches area both showed 4% of children exceeding 10 µg/dl and only one child in all four years exceeded 15 µg/dl in the Side Gulches. Kingston showed 11% greater than or equal to 10 µg/dl and 7% exceeded the 15 µg/dl criteria.

Blood Lead Levels and Percent to Exceed Toxicity Criteria by Age Group

Tables 6-4a-c and 6-5 and Figure 6-2 show the blood lead distribution by age for all children for all years. The highest blood lead levels are observed in the youngest age groups as shown in Tables 6-4a-c. One and two year old children have arithmetic mean blood lead levels of 7.0 µg/dl and 8.0 µg/dl, respectively, and geometric mean concentrations of 6.2 µg/dl to 6.3 µg/dl. Geometric mean levels then decrease with age from 5.2 µg/dl at age 3 to 3.0 µg/dl at age 8. There is a slight increase in mean concentrations for 9 year olds.

The percent of children to exceed critical toxicity levels differs markedly with age, as shown in Tables 6-4a-c and Figure 6-3. In the lowest age groups, 9 months to 3 years, 19% to 26% exceed the 10 µg/dl criteria. The rate is highest in 2 year old children with 17% of this group equal to or exceeding 15 µg/dl. For 4 year old children 12% exceed the 10 µg/dl criteria and 5% exceed 15 µg/dl. In older children the percent to exceed 10 µg/dl ranges from 5% to 8%, and 1% to 3% greater than or equal to 15 µg/dl.

Age-adjusted Blood Lead and Toxicity Estimates

It is important to note the relative rate of participation for children among the different age groups. Fewer children participated in the youngest age groups. Children in the 1 and 2 year old categories provided 40 and 46 samples, respectively, over the last 4 years, as opposed to 72 and 91 children, respectively in the oldest age groups. As a result, overall statistics provided for these results are possibly biased toward higher age groups and lower blood lead levels and incidence of toxicity. Tables 6-6a-b show observed and estimated mean blood lead levels for 1-6 (9-72 months) and 1-7 (9-84 months) year old children for each Basin subarea, respectively. The age-adjusted percent to exceed the 10 µg/dl criteria for these populations is 16.2% for 1-6 year old children (9-72 months) and 14.8% for 1-7 year old children (9-84 months), as shown in Tables 6-4b-c.

Comparison to National and State wide Lead Absorption Databases

Comparison of blood lead data for the Coeur d'Alene Basin to other sites and national or State wide surveys is problematic. There is a divergence of opinions regarding the appropriate comparisons. The blood lead data used in these analyses should be considered observational and opportunistic. Blood lead samples were collected from children participating in voluntary health intervention efforts. This was not a survey or experiment designed to obtain a representative sampling of the population. The data represent the population that elected to participate. There are age biases and repeat measurements included in the data. In previous memoranda, the State has offered the following description of blood lead levels observed within the BHSS relative to State and national surveys (edited to reflect Basin blood lead levels):

> Basin wide, 10% of 9 month through 9 year old children tested in 1999 showed blood lead levels of 10 µg/dl or greater and 5% exceeded 15 µg/dl. In the Superfund Site, 6.2% of children tested in 1999 showed blood lead levels of 10 µg/dl or greater and 0.8% exceeded 15 µg/dl. For 1-6 year old children in the Basin, the percent to exceed 10 µg/dl was 16%, and 8% exceeded in the Superfund Site in 1999. These results can be compared with a State wide survey conducted in 1997 that found pre-school children living in pre-1970 housing exhibited 4.2% exceedance of the 10 µg/dl criteria (IDHW 1998). Nationally, the 1991-1994 National Health and Nutrition Evaluation Survey (NHANES III) reports that 5.6% and 1.4% of 1-5 year old children in pre-1946 and 1946-1973 aged housing, respectively, exceed the 10 µg/dl blood lead criteria among similarly aged white non-Hispanic children (Pirkle et al. 1998).
>
> The 1999 Basin wide geometric mean blood lead level for 9-month to 9-year old children was 4.2 µg/dl and ranged from 3.3 µg/dl in Osburn to 5.6 µg/dl in Burke/Nine Mile. In the Superfund Site, the 1999 geometric mean blood lead was 3.9 µg/dl, down from 4.0 µg/dl in 1998 and 4.5 µg/dl in 1997. As summarized in Table 6-7, for 1-6 year old children, the mean Basin wide blood lead level was 5.2 µg/dl compared to 4.2 µg/dl in the Superfund Site. State wide, the average blood lead level for 1-6 year old children was 3.7 µg/dl in older housing. The majority of housing in the Basin is in the pre-1970 category. Nationally, comparable levels in 1991-1994 for the non-Hispanic population were 3.3 µg/dl and 2.4 µg/dl in pre- and post-1946 housing, respectively (Pirkle et al. 1998).

Comparisons of these results with elevated blood lead prevalence rates from Idaho wide or nation wide surveys is problematic and should be done with caution. Such large data sets, for various technical reasons, cannot be used to compare and draw conclusions about the relative degree of health hazard existing for children in the Basin communities.

Scientific designs of the NHANES surveys, are constructed in a way that does not allow simple comparisons with results of blood lead distributions for a single community. NHANES data provide a current snapshot for numerous *national* subsets or strata, that may not be appropriate for any single community. An explicit warning on technical grounds against making such comparison is in the

Executive Summary of ATSDR's 1988 report to Congress on childhood lead poisoning in America (ATSDR 1988).

Additionally, the purpose and design of the Basin surveys were conducted in a manner that does not match the organization of the various demographic and socioeconomic strata in the NHANES III survey reports (e.g., race/ethnicity, income, housing age).

Table 6-7 shows comparisons of blood lead levels from the various studies.

6.2.3 Summary Results for Follow-up Investigations of High Blood Lead Children

Follow-up Services

Follow-up investigations were conducted for 50 of the 58 children exhibiting high blood lead levels in the Basin. Follow-up lead health counseling consists of a public health nurse and/or a senior environmental health specialist employed by PHD contacting the parents of each child with an elevated blood lead level. The health specialist and nurse provide counseling and written information on how to identify sources of lead and reduce the child's exposure. A questionnaire is completed and educational materials are provided to the parents of children with a blood lead equal to or greater than intervention levels. Nutritional counseling and multiple vitamins with iron are also provided. A follow-up blood screen is offered 3-4 months later, and it is recommended that the child's blood lead information be shared with the family physician and that the child participate in the following Summer Screening Programs.

The environmental survey includes:

- A records search of environmental data collected from the residence.
- Sampling of soil, dust, paint, water, etc., as appropriate.
- Counseling regarding the avoidance of locally grown produce.
- Education regarding play activities, including those not associated with the primary residence.
- Evaluation of sources of exposure associated with parental occupations, hobbies, and other household activities.
- Evaluation of past or planned home remodeling activities.
- Recommendation for those without vacuum cleaners to use one of the high efficiency vacuums available through the Lead Health Intervention Program (LHIP).

A public health nurse and a senior environmental health specialist are available for consultations regarding sources of exposure to lead and the management of exposure pathways. A variety of locally developed and commercial fact sheets, brochures, coloring books, and videos are available regarding lead and children and exposure to lead during pregnancy.

Lead health information has been integrated into existing programs offered by the local health district. This information has been added to the Well Child Program, Immunization Clinics, Woman Infant and Children (WIC) Clinics, and pregnancy screening and prenatal clinics offered by the PHD. Prenatal blood lead screening is available for all pregnant women in the area through the LHIP. Pregnant women are offered blood lead testing and nutritional counseling during the first and third trimesters and are advised to provide their blood lead and exposure history to their private physicians. It is also recommended that a cord blood sample be collected when the child is born.
Each year, a public health nurse visits area grade schools. Classes are conducted for students in kindergarten through the third grade, and the nurse is available for presentations to classes through the 12th grade. Various methods are used including a puppet show and doll house to teach the concepts. The presentation covers the students' role in identification and management of exposure pathways that may affect them or their siblings. The program is presented in May so children can be reminded of the hazards of lead in soil and dust prior to summer vacation, when they are at the greatest risk of exposure.

A physician awareness program has been developed to keep local physicians apprized of program activities and the services that are available. Reference materials and a resource manual regarding lead and other heavy metals have been provided to area physicians and the local hospital. Upon request, additional follow-up activities and sampling can be conducted on behalf of physicians with special concerns regarding a patient with an elevated blood lead level.

Follow-up Results

A total of 25 follow-up investigations over the last four years were for children with blood lead levels greater than 15 µg/dl. These investigations concerned 21 individual children in 19 families. A small number of children were followed for more than one year. Children who were provided with follow-up services ranged in age from 1 to 9 years. One child was 1 year old, 6 children were 2 years old, 3 children were 3 years old, 4 were 4 years old, 3 were 5 years old, 1 was 7 years old, 1 child was 8 years old, and 2 children were 9 years old. Several siblings of these children also had blood lead levels in the 10-14 µg/dl range.

Eleven children, or more than 50% of those with blood lead levels above the 15 µg/dl standard, were from Burke/Nine Mile or Wallace. Three of these children were from Silverton and the Side Gulches, and 7 were from Kingston, Cataldo and the Lower Basin. No children with blood lead levels exceeding 15 µg/dl were from Mullan or Osburn.

Of the 8 children from Burke/Nine Mile, 3 were two years old and the remainder were 4 to 9 years old. These children's exposure profiles were characterized by high soil and dust concentrations, generally exceeding 2000 mg/kg; access to contaminated tailings in local play areas; and one home with possible paint problems.

In Wallace, 4 children, all less than or equal to 4 years of age, exhibited blood lead levels exceeding the 15 µg/dl criteria. Each child's exposure profile indicated possible paint or remodeling difficulties, the homes were noted as dusty and difficult to keep clean, and contaminated play areas were indicated for some children. Investigations of the 3 children in the Silverton/Side Gulches areas indicated possible play area exposures and potential difficulties with former residences in other subareas of the Basin.

There were 7 children with blood lead levels greater than 15 µg/dl in the Kingston and Lower Basin areas. There were 2 two year olds, 2 three year olds, and 2 five year olds and 1 four year old children. All of the children's profiles either indicated extremely high soil and dust lead levels (> 4000 mg/kg) associated with flooding, or extended time in contaminated recreational areas in the Lower Basin.

The remaining follow-up investigations were conducted on children in the 10-14 µg/dl blood lead range. Many of these children were siblings of those discussed above. Of 6 additional follow-up surveys completed in the Kingston/Cataldo/Lower Basin subareas, most were in Cataldo and involved homes contaminated by flooding or older children recreating in the river or lateral lake areas.

Seven additional follow-up investigations were conducted in the Osburn, Side Gulches, and Silverton subareas. These children were from seven different age groups. No paint or remodel problems were indicated. One child lived in a home with soil and driveway lead levels exceeding 2000-3000 mg/kg, and 4 children were noted to play in contaminated recreation areas.

Of 5 additional children followed in Wallace in the 10-14 µg/dl category, 2 children were 1 year old, 1 child was 2 years old, 3 were 3 years old, and 2 children were 4 years old. Three children's risk profiles indicated possible paint or remodeling problems, and one child spent extended time in a Lower Basin campground with high soil and sediment lead levels.

Five additional children were followed in the Burke/Nine Mile area. One child was 2 years old, 1 was 3 years old, 1 was 5 years old, 1 was 6 years old, and the remaining child was 7 years of age. These children's profiles indicated high soil and dust concentrations and exposure to tailings during recreational activities. Two children, ages 4 and 8 years, were followed in Mullan. High soil and dust concentrations and playing in mine waste areas were indicated in the risk profiles.

Summary of Follow-up Findings

In summary, the follow-up risk profiles indicate excess absorption associated with high soil and dust concentrations at homes in the Burke/Nine Mile subarea. Older children's risk profiles in this area indicate recreational exposures in neighborhood areas contaminated by tailings and old mill sites. High blood lead levels in Wallace are indicated in younger children and are possibly associated with paint and remodeling problems, high soil lead levels in play areas, and dusty or difficult to clean homes. Both Mullan and Osburn had no children in the greater than 15 µg/dl blood lead criteria and children's blood lead levels in the 10-14 µg/dl range were associated with high residential soil and dust concentrations or

play in contaminated areas. West of the BHSS, excess absorption was associated with either homes that had been flooded or with extended recreational activities in the river or lateral lake areas.

6.2.4 Adult Blood Lead Survey Results

Adult Blood Lead Levels by Age and Subarea

Table 6-8a summarizes adult blood lead levels collected in the 1996 Basin wide survey by geographic subarea. As opposed to the childhood blood lead observations, adult data are likely representative of the overall Basin population. The 1996 Study was census based; participant households were representative of non-participant households and 667 adults provided blood lead samples. Demographic comparisons between participating and non-participating households demonstrated similar house age, education, and income. Study participants were slightly more likely to own their home compared to non-participants. Figure 6-4 shows geometric mean blood lead levels by area and age group for the entire adult population. Figure 6-5 shows geometric mean and maximum blood lead levels for reproductive aged females (17-45 years) by geographic subarea. For the overall population, mean blood lead levels increase with age. Residents older than 50 years show mean blood lead levels ranging from 30% to 90% higher than the 10-25 year old category. Geomean blood lead levels in the younger group range from 1.9 µg/dl in Silverton to 3.2 µg/dl in the Burke/Nine Mile area. Generally, adult blood lead levels show a pattern similar to children with the highest levels occurring in the areas east of Wallace and lower levels in Osburn and the Lower Basin. Exceedance of the 10 µg/dl health criteria was uncommon among younger individuals with the majority of high levels occurring in older individuals in the upper Basin. The highest exceedance rate was in the Burke/Nine Mile area with 7 of 66 adults tested showing levels of 10 µg/dl or greater.

Blood Lead Levels for Reproductive Aged Females

Table 6-8b and Figure 6-5 summarize results for reproductive aged females. Among reproductive aged women, 2 of 151 women tested in the 17 to 45 year age group showed levels greater than or equal to 10 µg/dl. The highest level observed was 16 µg/dl. Geometric mean blood lead levels among reproductive aged females were 2.0 µg/dl or less in all areas except Burke/Nine Mile (2.4 µg/dl) and Wallace (2.6 µg/dl). The USEPA has recommended using a baseline blood lead estimate of 1.7 µg/dl to characterize non-Hispanic, white, rural adult female populations in the United States. Blood lead levels among reproductive aged individuals in the Basin are slightly higher, with some increase noted in the Wallace and Burke/Nine Mile subareas.

6.2.5 Coeur d'Alene Tribe Blood Lead Levels

No observed blood lead data are available for Coeur d'Alene Tribe members other than those included in the resident population. No measurements of biological response to lead exposure are available for Tribal members that engage in subsistence lifestyles.

6.3 LEAD EXPOSURE PATHWAYS

Children and adults are exposed to lead from a variety of sources in their everyday environment. Figure 6-6 shows the exposure routes evaluated in formulating the clean-up strategy and in subsequent IEUBK Model analyses of blood lead response for the BHSS. This figure shows that children are exposed to multiple contaminated environmental media including diet, drinking water, air, soils and dusts, and other consumer products including lead paint in the home.

6.3.1 Dietary Lead Sources

Market Basket Foods

Table 6-9 summarizes national Market Basket lead intake rates used for the resident population in this risk assessment. These values are obtained from the IEUBK Model for lead (USEPA 1994b).

Local Produce/Riparian Zone Produce/Fish

Table 6-10 summarizes media concentration data for garden produce (collected by URS Greiner in 1998 under FSPA 06), riparian vegetation (collected by the Coeur d'Alene Tribe in 1994), fish fillets from the lateral lakes (collected by State of Idaho from 1995-1997) and whole fish (collected by Washington State Department of Ecology) from the Spokane River.

The fish ingestion pathway evaluation for the tribal scenarios is based on filleted tissue metals data from a limited number of species from the lateral lakes and whole fish from the Spokane River. These results are likely not representative of fish from Coeur d'Alene Lake and extrapolation of hazards and risks to the Coeur d'Alene Lake fishery is not recommended.

6.3.2 Lead in Water

Drinking Water Sources

Tables 6-11a-j includes private well source and both first draw and purged tap water samples from 1997-1999 surveys for the resident population. For intake and IEUBK analysis, purged tap water and well source concentrations were used. Well source samples were collected from locations nearest to the well and do not reflect plumbing contribution to lead intake. Use of purged tap water results fails to account for lead leaching into and accumulating in water lines during low use periods. Current IEUBK Guidance suggests using an average of first draw and purged water (USEPA 1994a). These considerations indicate that drinking water concentrations may be underestimated for these individuals. However, it should also be noted that the laboratory used in the 1996 study did not report concentrations below the current drinking water source standard of 5 µg/l. These values were estimated at 2.5 µg/l in the original study and this report. Of the total 222 wells sampled in 1996, 183 reported

levels below 5 µg/l. These observations were assigned values of 2.5 µg/l. Subsequent analysis of samples collected in later years indicate that the geometric mean concentration of samples reported below 5 Fg/l was 0.75 µg/l. This suggests that use of the 2.5 µg/l estimate for drinking water lead concentrations may be an overestimate.

Surface Water Sources

Tables 6-12a-b summarize surface water lead levels for both disturbed and undisturbed samples, respectively.

6.3.3 Lead in Air

Lead in air data are not available for the Basin. However (for the purposes of this report), lead in air is assumed to be a minimal contributor to overall exposure and IEUBK Model default assumptions are applied when applicable (See Section 3.2.2 and Appendix V).

6.3.4 Lead in Paint

Paint lead was measured in the upper Basin in the large epidemiologic surveys conducted in 1974-75. The highest levels of lead paint in those surveys were noted in the communities east of Wallace (IDHW 1976). Because the housing stock is predominantly older than 1960, lead paint is prevalent and has been investigated on a case-by-case basis in the follow-up of children with high lead levels. Occasional problems with lead paint have been noted in individual situations, but health officials do not believe this problem to be widespread. The majority of interior paint lead impact is believed to be manifested through house dust.

Lead in paint was measured by XRF techniques in the *Coeur d'Alene River Basin Environmental Health Exposure Assessment* (IDHW 2000). Those data were analyzed in a semi-quantitative manner in the cited report. Those data have since been reanalyzed following the methods suggested by Bornschein (pers. comm. 2000). For the purposes of this risk assessment, XRF results were compiled on a house-by-house basis from the 1996 survey. All XRF readings and appropriate notations and surface condition variables were entered into a computer database. These results were then summarized to determine minimum, maximum, median and mean loadings and condition results for interior, exterior and entryway surfaces. Table 6-13 shows summary statistics for lead-based paint by geographic subarea. Figures 6-7a-b show mean interior and exterior paint lead loadings by geographic area.

This methodology results in numerous descriptors of paint lead content and condition. All of these variables are subsequently applied without bias in quantitative analysis procedures. For purposes of defining lead-based paint, the standard established by HUD is a XRF reading of 1.0 mg/cm^2 or higher was used (HUD 1995, 1997). For this report, paint measured as 1.0 mg/cm^2 and above is considered

lead-based, and paint measured less than 1.0 mg/cm^2, or below the instrument detection limit is not considered lead-based.

6.3.5 Lead in Soils and Dusts

The soils and dust pathways are among the most complex and, generally, greatest contributors to childhood lead levels. Available soil and dust data suggest that house dust contains a complex combination of lead from several sources moderated by environmental conditions and social, economic, and cultural factors that influence home maintenance and behavior. Soils from home yards, neighborhoods, and throughout a community all seem to contribute significantly to house dust (TerraGraphics 2000a). Except children that eat paint chips, lead paint exposure predominantly comes from peeling and chalking paints incorporated into house dusts or outdoor soils. Local produce is most often contaminated by soils or dusts adhering to plant tissue despite washing efforts. Airborne contamination presents its greatest hazard as it settles and contaminates surface dusts that are ingested by children.

These factors suggest that children are exposed by both direct contact with contaminated soils or house dusts and indirect exposure to contaminated soils through house dusts which originate from outdoor soils, paint, and possibly, residual dust particulate in structures (TerraGraphics 2000a, Succop et al. 1998).

Yard Soil Lead Levels

Yard soil, play area, garden, driveway and rights-of-way (ROW) soil lead levels are summarized by geographic area in Tables 6-11a-j. These tables also contain percentage distributions for relative contaminant levels by subarea. Figures 6-8a-c show mean yard soil and house dust lead levels for the Basin and other areas in North Idaho (Spalinger et al. 2001). These results have been aggregated from several surveys conducted over a four year period in the Basin. Appendix N summarizes the methods used to combine these data for analysis.

Community and Neighborhood Soil and Sediment Lead Levels

Table 6-14 summarizes neighborhood stream sediment lead concentration by geographic subarea.

Common Use Area Soil and Sediment Lead Levels

Tables 6-15a-b summarize soil and sediment lead levels, respectively, for common use areas by geographic subarea.

Waste Piles

Table 6-16 summarizes available lead contamination levels for waste piles.

House Dust Lead Levels

House dust lead levels for vacuum bag and dust mat samples collected in the Basin are summarized by geographic subarea in Tables 6-11a-j and Figures 6-8a-c. Dust and lead loading rate data from 1996 entryway mats are summarized in Table 6-17 and Figures 6-9a-c.

Riparian Zone Soil and Sediment Lead Levels

Table 6-18 summarizes soil and sediment lead concentration levels in the Lower Basin floodplain from Rose Lake to Harrison.

6.4 SITE-SPECIFIC BLOOD LEAD AND ENVIRONMENTAL EXPOSURE ANALYSIS

6.4.1 Correlation Analysis

Blood and Environmental Lead Level Correlations

General correlation matrices were developed for the blood and dust database for all geographic areas combined. These matrices were examined to assess the linear association between the pairs of variables and to preliminarily identify the best predictors of blood lead levels and house dust lead concentrations for multiple regression model analysis. Tables 6-19 and 6-20 show the overall correlation matrix for all geographic areas for blood and dust lead concentrations. Most of the correlations in Tables 6-19 and 6-20 are highly significant (p#0.001). In reviewing these results, it should be noted that the paint lead methodology results in numerous descriptors of paint lead content and condition. In contrast, the house dust and yard soil data used in the quantitative analysis are composites and aliquot concentrations were not available from the parent studies. As a result, several paint lead variables expressing the same general relationship may appear in the tables.

Examination of the correlation matrix for blood lead levels (Table 6-19) shows that blood lead level is significantly correlated with age (inversely), dust mat lead loading rate, yard soil lead concentration, community geometric mean soil concentration, and paint lead condition and XRF loading. The highest correlations are with the dust mat lead loading rate (r=0.63, p#0.001), followed by the mean interior paint condition (r=0.48, p#0.001). Other high correlations with blood lead levels are the median exterior lead XRF reading (r=0.41, p#0.001), the median exterior paint condition (r=0.40, p#0.001), the median interior lead XRF reading (r=0.34, p#0.001), mean interior paint condition code (r=0.48, p≤0.001), mat lead concentration (r=0.31, p#0.001), yard soil lead (r=0.16, p#0.001), and the community geometric mean soils (r=0.12, p#0.05). Scatterplots for select variable pairs are presented in Figure 6-10a.

The correlation matrix for the house dust mat data (Table 6-20) shows that log transformed dust mat lead concentration is significantly correlated with yard soils, community geometric mean soils, and paint lead loading. The highest correlations are with the log transformed yard soil lead (r=0.65, p#0.001), followed by the community geometric mean soils (r=0.56, p#0.001) and non-log-transformed paint variables; the maximum interior lead XRF reading (r=0.30, p#0.001), the arithmetic mean exterior lead XRF reading (r=0.29, p#0.001), and the maximum interior paint condition (r=0.13, p#0.05). Scatterplots for select variable pairs are presented in Figure 6-10b.

The house dust mat data, (Table 6-20) shows that log transformed lead loading rate is significantly correlated with yard soils, community geometric mean soils, and paint lead levels. The highest correlations are with the log transformed yard soil lead (r=0.53, p#0.001), followed by the community geometric mean soils (r=0.44, p#0.001) and non-log-transformed paint variables; the maximum interior

lead XRF reading (r=0.29, p#0.001), the arithmetic mean exterior lead XRF reading (r=0.26, p#0.001), the mean interior paint condition (r=0.25, p#0.001), and the mean exterior paint condition (r=0.25, p#0.001). Scatterplots for select variable pairs are presented in Figure 6-10c.

Examination of the correlation matrix for the house dust vacuum data (Table 6-20) shows that log transformed vacuum cleaner bag lead concentration is significantly correlated with dust mat lead content, yard soils, community geometric mean soils, and paint lead loading. The highest correlations are with the log transformed dust mat lead concentration (r=0.72, p#0.001), followed by the log transformed yard soil lead (r=0.52, p#0.001), the community geometric mean soils (r=0.45, p#0.001) and non-log-transformed paint variables, the maximum interior lead XRF reading (r=0.30, p#0.05), and the arithmetic mean exterior lead XRF reading (r=0.25, p#0.05). Scatterplots for select variable pairs are presented in Figure 6-10d.

6.4.2 Regression Analysis

Blood Lead and Environmental Exposure Factors

Stepwise regression analysis was employed to identify variables for the best model describing the blood lead and environmental exposure relationship. The blood lead data used in these analyses should be considered observational and opportunistic. Blood lead samples were collected from children participating in voluntary health intervention efforts. This was not a survey or experiment designed to obtain a representative sampling of the population. The data represent the population that elected to participate. The single variable model step identified lead loading rate on entryway mats as describing most of the variation in blood lead levels. Yard soil lead concentrations were identified as the next most significant variable. These variables were followed by the median exterior paint loading, as measured by XRF, the minimum interior paint condition, and an inverse relationship with age. No other environmental variables met significance criteria of p=0.05 in the presence of these factors.

Although the residuals of the blood lead model are normally distributed and basic statistical assumptions are met, variables included in the model are distributed log-normally. This model form was also investigated using log transformations for both independent and dependent variables and alternate statistics (mean, maximum, minimum, median) for the paint related measurements. No improvements in model significance, R-square statistic or residual distributions, were noted with variable transformations. The R-square statistic was diminished with the dependent variable transformation. No other paint variables were found to increase model significance or the R-square value. Interior paint loading and exterior condition were not significant in any measurement.

Table 6-21 shows the best model selected to describe blood lead levels. Five variables are included that explain about 60% (R-squared = 0.60) of the variation in children's blood lead levels. This is a strong relationship for a study of this type. In previous analysis of blood lead and environmental exposures in the Basin (including the BHSS) typical R-squared statistics ranged from 0.20 to 0.75 with

the lower range more common in recent years (TerraGraphics 2000a, Yankel et al. 1977). The variables selected include: i) Age of the Child, ii) Yard Soil Lead Level, iii) Dust Mat Lead Loading Rate, iv) Median Exterior Paint Lead Loading, and v) Minimum Interior Paint Condition.

All five variables show similar standardized regression coefficients. The coefficients for age and yard soil lead are similar to those noted at the BHSS, indicating a consistent dose-response relationship. According to the model, typical blood lead levels decrease by about 0.34 µg/dl per year of age. Blood lead levels increase by an average of 0.7 µg/dl per 1000 mg/kg lead in home yard soil. Compared to the BHSS, where blood lead levels increase by an average of 2 µg/dl per 1000 mg/kg lead in home yard soil (using the simple multiple regression model) and other reported literature values of 0.6-12.6 µg/dl per 1000 mg/kg lead in yard soil, the Basin yard soil coefficient falls into the lower end of reported slope coefficients (TerraGraphics 2000a, Xintaras 1992).

The strong mat dust lead loading relationship was noted in the *Coeur d'Alene River Basin Environmental Health Exposure Assessment* (IDHW 2000). Blood lead levels increase by about 0.16 µg/dl per mg/m^2/day of lead loading to the home. At a constant typical dust loading rate of 1 g/day, this increase is about 0.5 µg/dl per 1000 mg/kg. Geometric mean dust loading rates range from 1.04 g/m^2/day in Osburn to 2.31 g/m^2/day in Burke/Nine Mile. Geometric mean dust mat lead concentrations range from 318 mg/kg in Lower Basin/Cataldo to 1781 mg/kg in Burke/Nine Mile. These two variables combine to yield dust lead loading rates ranging from a geometric mean of 0.48 mg/m^2/day in the Lower Basin to 4.28 mg/m^2/day in Burke/Nine Mile (Table 6-17).

The significance of the paint variables adds additional information. The Median Exterior Paint Loading is the median XRF reading for all exterior paint lead measurements collected at a home. This variable may be indicative of an additional lead source to children. However, its significance in the presence of yard soil and house dust lead loading suggests a separate pathway may be present or the variable may be a surrogate for house age and community-specific effects. The effect of this variable is about 0.52 µg/dl per mg/cm^2. Geometric mean exterior paint loadings range from 0.03 mg/cm^2 in Kingston and the Side Gulches to 0.22 mg/cm^2 in Wallace (Figure 6-7b).

The significance of Minimum Interior Paint Condition is also interesting. This value represents the best single paint condition noted for a home. Most homes (79%) have a Category 1 value, indicating at least the painted surface in one room was in good condition. However, there are some number of homes (19%) where the best paint condition noted was Category 2 (chipping and peeling on a few surfaces in all rooms), or Category 3 (indicating all paint was in chipping, peeling, and chalking condition on most surfaces) (2%). These data suggest that children living in homes with extraordinarily poor paint condition exhibit significantly higher blood lead levels. Children in homes in Minimum Category 2 would exhibit about a 2 µg/dl greater response than those in Category 1. Those in Category 3 would show about 2 µg/dl greater response than Category 2. This variable reflects a potential source of lead from which children could consume paint particles directly or through incorporation in the house dust, and may also be a surrogate for home hygiene and socioeconomic and housing quality status.

Bivariate Analysis of Blood Lead Levels and Lead-Based Paint Hazard

A bivariate analysis was performed on the blood lead data to further examine the relationship with lead-based paint. An interior lead-based paint hazard was assigned home-by-home using the Minimum Interior Paint Condition (INTCCMIN) variable determined significant in the blood lead regression analysis and the Maximum Interior Paint Lead Loading (INTMAX) determined significant in the dust mat lead loading regression analysis in the following section. These two paint variables were then used to define an interior paint lead hazard. If the INTMAX variable was below 1.0 mg/cm^2, or was not detected, then there was no interior lead paint hazard. If the INTMAX variable was 1.0 mg/cm^2 or higher, but the INTCCMIN variable showed the paint was in good condition (Category 1), there was no interior lead paint hazard. There was an interior lead paint hazard if lead-based paint existed ($ 1.0 mg/cm^2) and the condition of the paint was Category 2 (chipping and peeling on a few surfaces in all rooms), or Category 3 (indicating all paint was in chipping and peeling condition on most surfaces).

Of the total 524 blood lead observations available in the database, 216 lived in a house that had been tested for lead based paint by XRF. Of those 524 blood lead observations, 58 (11%) had blood lead levels greater than or equal to 10 Fg/dl. Of the 216 observations with an associated lead paint measurement, a similar percentage, 9.2% (or 20 observations), had blood lead levels greater than or equal to 10 Fg/dl. Of those 216 observations with an associated lead paint measurement, 23 (or 11%) indicated an interior lead paint hazard and 193 (89%) showed no hazard.

Figure 6-11 shows that 70% (14/20) of the children with high blood leads were not associated with an interior lead paint hazard, while 30% (6/20) were associated with a lead paint hazard. One hundred ninety-six (196) blood lead observations below 10 Fg/dl were from homes that had been tested for lead based paint. Figures 6-12a and 6-12b represent environmental variables analyzed for high and low blood lead levels in children exposed and not exposed to an interior lead paint hazard. Vacuum and mat dust, and yard soil lead concentrations were almost all significantly different among children with high blood lead levels and children with blood leads less than 10 Fg/dl for both those exposed and not exposed to a lead paint hazard. The children exposed to a lead paint hazard saw increased concentrations and loading rates compared to children not exposed to a lead paint hazard, although not enough observations exist to compare vacuum dust lead levels. Yard soil lead levels in those exposed to a lead paint hazard, although not statistically different (p=0.06), show an increased mean for those children with high blood leads compared to those with blood lead levels less than 10 Fg/dl.

This simple bivariate analysis suggests similar conclusions to those discussed in the blood lead regression analysis. Children with blood lead levels greater than or equal to 10 Fg/dl are exposed to significantly higher soil, dust, and dust lead loading levels than children with blood leads less than 10 Fg/dl. Lead-based paint plays a minor, but significant, role in affecting these blood lead levels. Children living in houses with an interior lead paint hazard are exposed to increased soil and dust lead concentrations and dust lead loading rates compared to those not exposed to a lead paint hazard. A disproportionate number of children with high blood lead levels come from homes with an identified

lead paint hazard (i.e., 30% of children with high blood lead levels come from the 11% of homes identified with an interior lead paint hazard). However, the majority of children with high blood lead levels (70%) come from homes with no identified lead paint hazard.

These results were obtained using the two interior lead paint measurements significant in the regression analysis, INTCCMIN and INTMAX, to determine whether an interior lead paint hazard existed. Other paint variables were considered. Using the median concentration and condition code as an average paint variable for each house results in 15% (3/20) of the high blood lead observations associated with an interior lead paint hazard. Examining the maximum concentration and condition code for each house shows that a possible 45% (9/20) of the high blood leads would be associated with an interior lead paint hazard. The worst case scenario would be if the house had both an interior and exterior lead paint hazard. The interior and exterior maximum concentration and condition code for each house would provide this estimate and shows that 30% (6/20) of the blood lead observations greater than or equal to 10 Fg/dl had both an interior and exterior lead paint hazard. In any case, despite the importance of lead paint for some individuals, the majority of children exhibiting high blood lead levels come from homes not exhibiting a lead paint hazard.

Dust, Soil, and Paint Lead Relationships

Stepwise regression analyses, similar to the blood lead regression analyses, were conducted for dust mat lead concentration, lead loading rate, and vacuum bag lead concentration using dust lead, soil lead, and paint lead source variables. For these analyses, soil and dust lead concentrations were log transformed to meet the appropriate statistical assumptions underlying regression analysis.

Tables 6-22a-b show two candidate regression models for mat lead concentration following stepwise selection of candidate independent variables. In Table 6-22a the first model explains 44% of the variation in log mat lead concentration using four variables: i) log of the yard soil lead concentration, ii) the maximum interior lead XRF reading, iii) the minimum interior paint condition, and iv) exterior median lead XRF loading. Three of these variables were also significantly related to blood lead levels in the preceding analysis. Interior paint lead loading was not significant in the blood lead regression model. These model results suggest that both soil and paint are potential sources of lead in house dust. The relative sums-of-square, F-statistic and standardized regression coefficients suggest that soil is the largest contributor with both interior and exterior paint having similar, but lesser, significance than soils. Homes with extraordinarily poor paint condition also show increased mat dust lead concentrations.

Table 6-22b shows the same model with the community geometric mean soil concentration variable added. The inclusion of this variable in the model results in loss of significance for both interior and exterior paint lead concentration variables. Table 6-22c shows the final model form excluding the non-significant variables. This model explains 47% of the variation in dust mat concentrations as opposed to 44% in the previous form including paint lead loadings. These results show that yard soil continues to be the most significant contributor to entryway dust lead followed by soils from the community at large.

These results suggest that community mean soil lead concentrations and paint lead loading co-vary as a function of housing and community age and that any major effect of paint lead on dust lead concentration is manifested through soils. Older homes may have more lead paint, higher soil lead content, accumulated more lead dust from historic industrial operations and fugitive emissions from various sources, and be located closer to mineral industry activities.

These results may also suggest another pathway where lead dusts may be brought in from the larger community where a worker (construction, etc.) brings those dusts and soils home. Recent studies reviewed by Roscoe et al. 2000 indicate higher blood lead levels were consistently reported among children of workers that may bring lead home from the workplace.

The model in Table 6-22c suggests about a 0.8 mg/kg decrease in dust mat lead concentration per mg/kg decrease in yard soil concentration at typical values. This is similar to the effect of soil lead on house dust lead levels noted in the BHSS (TerraGraphics 2000a). The same model suggests that dust mat lead levels in homes with poor minimum paint condition would have dust lead levels typically 300 mg/kg greater per condition code category, and that dust lead concentration reductions would parallel decreases in community mean soil lead concentration.

Table 6-22d shows the select regression model form for dependent variable log (dust mat lead loading rate). Four variables are significant in describing 36% of the variation in lead loading rate. The log of yard soil lead concentration was again the most significant variable followed by the interior paint minimum condition, the community mean soil concentration, and the interior paint maximum lead loading by XRF. In this case, the maximum interior lead XRF reading remained significant at $p=0.02$ in the presence of community soil. Exterior paint lead content was not significant.

Table 6-23 shows the select regression model for dependent variable log vacuum bag lead concentration. This analysis is limited by the number of observations containing both vacuum bag and paint XRF readings; two observations were identified as outliers and were excluded from the statistical analysis. Only 68 observations are available for this analysis. About 55% of the variation in vacuum bag lead concentration is explained by log transformed yard soil and dust mat lead concentrations, and the maximum interior paint lead loading shown in Table 6-23. This analysis suggests that vacuum bag lead concentration is largely related to dust mat lead concentrations, with yard soil lead content and the maximum interior paint XRF reading contributing at the $p=0.01$ and 0.03 significance level, respectively. The empirical models in Table 6-22c and 6-23 are used in Section 6.7.6 to project post soil remediation dust lead concentrations.

6.4.3 Summary of Site-Specific Lead Health Analysis

These site-specific analyses add considerable insight into the blood lead and environmental source relationships ongoing in the Basin. Blood lead levels are strongly related to dust lead loading, yard soil lead, and exterior paint loadings. Interior paint condition is a significant risk co-factor that is also related

to dust mat lead concentration. Although lead-based paint is important for some individuals (30% of children with elevated blood lead levels), it is not the most significant factor affecting the majority of the children (the remaining 70% of children) with high blood lead levels. The slope of the blood lead to yard soil relationship of 0.7 µg/dl per 1000 mg/kg soil lead is similar to that observed at the BHSS and other mining related sites. Succop et al. (1998) using simple regression techniques estimated a yard soil slope coefficient of 1.5 µg/dl per 1000 mg/kg soil lead; however, by using more sophisticated statistical techniques a yard soil slope coefficient of 2.2 µg/dl per 1000 mg/kg soil lead was observed. The relationship with dust lead loading rate was previously noted in the *Coeur d'Alene River Basin Environmental Health Exposure Assessment* (IDHW 2000). However, this measurement has not been reported in dose-response analysis at other sites, and was not utilized at the BHSS until remediation was largely complete in Smelterville.

Most of the effect of soil lead on blood lead is likely manifested through house dust. Dust lead loading rate is the most significant variable in describing blood lead levels, explaining more than 40% of the variation in a single variable model. The variable is made up of two components. Those are dust loading rate, or the amount of dust that collects on an entryway mat per day, and the lead concentration of that dust. Examination of the database suggests that both components are important in exposure assessment for the Basin. Figures 6-9a-c show geometric means for both components and the product lead loading rate for each geographic subarea.

Figure 6-9b shows that the 1996 dust loading rate, or amount of dust entering a home, is highest in the Burke/Nine Mile area at 2.31 $g/m^2/day$, followed by Wallace and Mullan near 1.40 $g/m^2/day$, Osburn and Silverton in the 1.00 to 1.30 $g/m^2/day$ range, Side Gulches and Kingston at 1.44 $g/m^2/day$ and 1.21 $g/m^2/day$, respectively, and the Lower Basin at 1.54 $g/m^2/day$. A similar pattern is also noted in the lead concentration of these dusts, shown in Figure 6-9a. Mean dust mat lead concentration exceeds 1200 mg/kg and 1700 mg/kg, respectively, in Mullan, Burke/Nine Mile, and Wallace, and is near 900 mg/kg in the remainder of the Basin above the BHSS, and is near 600 mg/kg in Kingston and 300 mg/kg in the Lower Basin.

The combination of these effects results in extremely high lead loadings in Burke/Nine Mile area, nearly five times that in the Silverton to Kingston reach. Wallace also shows especially elevated lead loading rates, nearly twice the majority of the other geographic subareas. The remainder of the areas show similar lead loading rates, except the Lower Basin, with rates about 40% less than the Kingston to Silverton reach. Lead loading rate (from 1996) is shown in Table 6-17.

Mean blood lead levels and percent of children to exceed critical toxicity criteria follow a similar pattern with highest levels in Burke/Nine Mile followed by Wallace. Mullan, Silverton, Osburn and Kingston that all have similar levels. The exception to this trend is the Lower Basin where lead loading is notably lower, but blood lead levels and percent of children with high blood lead levels are similar to Wallace.

These results suggest that the combination of dusty conditions and high lead content of dusts is the primary determinant of blood lead absorption in the upper Basin. Lead loading rate, in turn, is most related to outdoor yard soil lead content with this variable alone explaining 42% (of the 47%) of the variation in mat lead concentration. The slope of the mat dust to yard soil lead concentration relationship is about 0.8. Interior paint condition code is also significant with respect to dust mat lead concentration, indicating as much as 300 mg/kg increase in poor condition homes. Exterior and interior paint loading is significant when added to the previous variables in regression analysis, but becomes non-significant in the presence of community mean soil concentrations. This relationship is difficult to interpret because both paint concentration and community wide contamination levels are higher in older communities. However, this relationship is potentially important as the model indicates house dust lead levels are sensitive to community wide soil concentrations, as was noted at the BHSS and other sites. Elevated blood lead levels in the Lower Basin and follow-up activities initiated by the Panhandle Health District suggest that pathways or sources beyond the immediate residence are associated with recreational activities in the floodplain.

The same variables, yard and community mean soil concentration and interior paint condition remain significant with respect to lead loading rate. Yard soil lead concentration is the strongest determinant in lead loading followed by community mean soils and interior paint condition. Maximum interior paint lead loading also becomes a weaker, but significant, determinant of lead loading.

Finally, vacuum bag dust lead levels are dependent on dust mat lead concentration, outdoor yard soil lead content, and indoor paint lead loading. Generally, vacuum bag dust lead concentration ranges from about 60% to 80% of dust mat lead content as shown in Figure 6-13. These overall results suggest a complex pathway of exposure. Blood lead levels are most related to lead loading in the home, followed by independent effects of yard soil lead, and paint lead condition and exterior lead content. The dust lead pathway is most influenced by outdoor soils, but is augmented by paint contributions particularly in older homes in poor condition. The overall effect is exacerbated by dusty conditions in Burke/Nine Mile and to a lesser extent in Wallace. Much less of a problem is noted with respect to dustiness or dust concentrations in the Lower Basin.

6.5 BASELINE AND INCREMENTAL LEAD EXPOSURE INTAKE RATES

6.5.1 Exposure Routes Considered

Lead intake rates are developed for the identified pathways and exposure routes in a format compatible with input to the IEUBK Model and USEPA Adult Model for lead that predict geometric mean blood lead levels. Intake rates were calculated using the Central Tendency (CT) exposure factor values for input into both models. CT values represent a typical or average case (or 50% percentile). Exposure routes and intake estimates are developed for Suburban/Rural community scenarios for the local resident population and for Native American scenarios for Coeur d'Alene Tribe members practicing traditional or modern subsistence activities.

Current Resident Population

Intake rates for the resident population are developed in both baseline and incremental exposure format. Baseline exposure refers to typical everyday activities associated with residential life in the Basin. Incremental exposures are associated with specific activities (e.g., recreational or occupational) that can add to the baseline exposure.

Baseline exposure routes for lead in the residential environment for both children and adults include:

- incidental ingestion of soils/sediment/sub-surface soils,
- incidental ingestion of house dusts,
- ingestion of drinking water,
- inhalation of dust in air, and
- consumption of market basket foods.

Incremental exposures are addressed in four major categories: i) occupational, ii) recreational, iii) local foodstuff, and iv) consumer goods.

Intake rates for adults in occupational settings outside the mineral processing industry include:

- incidental ingestion of soils, and
- inhalation of dust in air.

Recreational intake rates for children and adults include:

- incidental ingestion of soils/sediments,
- incidental ingestion of surface waters,
- ingestion of drinking water, and
- inhalation of dust in air.

Intake rates associated with local foodstuffs for children and adults include:

- consumption of fish from the lateral lakes and Spokane River for Tribal Scenarios,
- consumption of locally grown vegetables.

Lead intake rates for consumer goods include:

- ingestion of lead-based paint other than that manifested through house dust for young children.

Native American Scenarios

Coeur d'Alene Tribal practices are such that the risk assessment is not easily sub-divided into baseline and incremental activities. Tribal lifestyle and exposure pathways are discussed in Section 3.0. The resident riparian lifestyle in the Traditional Subsistence scenario and the harvest techniques employed throughout tribal history represent holistic practices that encompass all activities in an overall lifestyle. Fully addressing potential Native American lead exposures within the Basin requires consideration of routes of exposure not included in other scenarios in the HHRA. The tribal riparian lifestyle has the potential for significant prolonged exposures to both sediment and water. One example could be the harvest of the water potato (*Sagittaria spp.*) at the mouth of the Coeur d'Alene River. These activities also involve women of reproductive age accompanied by small children for extended periods of time.

The same exposure routes are evaluated for both the Traditional Subsistence scenario and the Modern Subsistence scenario. For evaluation of the human health risks associated with each scenario, the exposure factors will reflect the difference for each exposure route. Generally, the exposure frequencies for the Modern Subsistence are reduced from the values used in the Traditional Subsistence scenario.

Intake rates developed for the tribal scenarios are:

- incidental ingestion of soil/sediment,
- dermal contact with soil/sediment (non-lead only),
- ingestion of surface water as drinking water,
- incidental ingestion of surface water during recreational activities,
- consumption of fish, and
- consumption of water potatoes.

The remainder of the tribal pathways, discussed in Section 3.2.4, are not quantified in this risk assessment, due to limitations on available data or relatively insignificant contribution to the overall risk to human health. Data associated with certain exposure routes may be available, but are considered

insufficient to characterize media contaminant levels. Example calculations also showed some pathways do not significantly contribute to human health risk when compared to others being quantified.

Table 3-19b shows those Native American exposure routes that will be considered either quantitatively or qualitatively in the HHRA.

6.5.2 Developing Baseline Intake Rates for the Resident Population

Children's Baseline Intake Rates

Baseline intake rates estimate the amount of lead taken into the body through everyday, normal activities undertaken by the general population. These rates apply to everyone. These are calculated by multiplying the amount of soil, dust, food, and water ingested by the amount of lead contained in each medium.

Baseline Ingestion and Inhalation Rates

Baseline lead intake rates are calculated using the ingestion rates developed within the IEUBK Model and USEPA Adult Model for lead. These rates will correspond with the default ingestion, consumption, and inhalation parameters indicated in current USEPA Guidance. Using default assumptions indicates that values that characterize children and adults throughout the United States are applicable to estimate the amount of soil, dust, food, and water consumed in typical activities for the resident population in the Basin. Tables 6-24a-b, respectively, show default ingestion and inhalation rates for children from the IEUBK Model guidance and default parameters for adults from the USEPA Adult Model for Lead.

Soil and Dust Ingestion

Both the IEUBK Model and USEPA Adult models combine soil and dust ingestion rates into a single input variable. The relative contribution is partitioned, or divided, among the various sources of soil and dust available to the population. Two initial partitions were developed for application to the IEUBK Model analysis. The first scenario is the USEPA guidance default partition of 55% house dust : 45% yard soil using the individual home yard as the soil exposure unit. This application is referred to as the "EPA Default Model". The second scenario corresponds to the 40% house dust : 30% yard soil : 30% community soil partition utilized at the Bunker Hill Superfund Site (BHSS), referred to as the "Box Model" or "40:30:30 Model". The Box Model is described in more detail in Appendix Q.

Drinking Water Ingestion

Default drinking water concentrations are included in both the EPA Default Model and Box Model for Community Mean applications. Observed concentrations are employed in the Batch Mode application

with the community geometric mean concentration (calculated from purged tap water and 1996 well water data presented in Tables 6-11) substituted for missing observations.

Inhalation of Dust in Air

Default values will be used for inhalation of dusts. Support calculations demonstrate that the relative contribution of this route does not justify additional characterization. Inhalation intakes assume similar RME exposure factors and are estimated using methods from EPA Exposure Factors Handbook (USEPA 1990c). The inhalation exposure for airborne contaminants is estimated by the following formula:

$$I_{(inhal)} = C \times IR \times EF \times ED \times EP$$

Where:

$I_{(inhal)}$	=	Inhalation intake (µg/year)
C	=	Concentration of contaminant (µg/m^3)
IR	=	Inhalation rate (2.1 m^3/hr)
EF	=	Event frequency (5 days/wk)
ED	=	Event duration (39 weeks/yr)
EP	=	Event period (8 hrs/day)

Assuming 0.1 µg/m^3 air lead concentration consistent with BHSS observations and a moderate ventilation rate for average adults, yields a ventilation intake of 328 µg. Assuming 50% retention and absorption in the lungs results in 164 µg of lead absorption per nine month construction season. This represents approximately 4% of the total baseline intake rate for lead in the Lower Basin, compared to 1% in Wallace. Neither dermal nor inhalation exposure routes are considered in the remaining analyses.

Dietary Lead

Default dietary intake rates representing the typical US market basket are included in both model forms. National default values are used for baseline dietary intake rates throughout these analyses.

Summary Baseline Intake Rates

Tables 6-25a-b summarize estimated mean typical daily lead intake rates for each subarea in the Basin for both the EPA Default and Box Model scenarios. Tables 6-25a-b and Figures 6-14a-b show example intake rates for 4 year old children. Appendix P contains similar intake rates for other ages. Estimated Baseline lead intake for 4 year old children based on geometric mean conditions in each

subarea range from a low of 30 µg/day in the Lower Basin to 99 µg/day in Wallace for the EPA Default scenario. Generally, the Box Model intake rates are slightly lower ranging from 27 µg/day to 95 µg/day for the same subareas, respectively. Estimated mean intake rates are considerably higher in the upper Basin ranging from 85 µg/day to 99 µg/day east of and including Wallace, 52 µg/day to 64 µg/day in the Silverton to the BHSS reach, 43 µg/day to 46 µg/day in Kingston and 27 µg/day to 30 µg/day in the Lower Basin.
All of the Baseline intake rates are dominated by the soil and dust component. Air, drinking water and dietary lead intake for the typical child total about 8.5 µg/day or about 10% of the total baseline intake in the upper Basin and about 30% for the Lower Basin. In the EPA Default scenario, house dust contributes about 50% to 60% of the total intake in all areas, ranging from 17 µg/day in the Lower Basin to 55 µg/day in the upper Basin. Yard soil is estimated to contribute only 5 µg/day typically in the Lower Basin to 35 µg/day in Wallace in the EPA Default scenario. In this scenario, yard soil accounts for 17% of the total estimated intake in the Lower Basin to 35% in Wallace.

The percentage contribution to total intake differs somewhat for the Box Model scenario. In this scenario, house dust contributes slightly less than in the EPA Default scenario ranging from 37% to 47% of the total lead intake throughout the Basin. Soils contribute from 26% in the Lower Basin to 48% in the upper Basin. However, in the Box Model the combined total of soils from the individual home yard and the greater community contribute a similar amount to overall intake.

Adult Baseline Intake Rates

Baseline intake rates for adults are shown in Table 6-26 based on a typical soil and dust ingestion rate of 50 mg/day comprised of ½ soil, ½ dust. However, these results are not used in the subsequent Adult Model for Lead analyses and are presented to identify potential adult lead intakes. Soil and dust ingestion rates are directly input to the Adult Model for Lead using values of 50 mg/day, 100 mg/day, and 200 mg/day (Table 6-27b-c and Tables 6-28a-b). Total baseline intake rates for lead from soils and dust range from a mean of 10 µg/day in the Lower Basin to 44 µg/day in Wallace or about half that determined for children. Estimates range from 18 µg/day to 23 µg/day in the areas near the Box (Kingston to Silverton), and 39 µg/day to 40 µg/day in the upper Basin. In all cases the majority of baseline lead intake is from house dust.

6.5.3 Developing Incremental Intake Rates for the Resident Population

Incremental lead intake rates refer to the amount of lead taken into the body during activities in which only certain members of the population engage. These individuals either consume more soil, dust, water, food, than the general population or those media have higher lead content.

Incremental lead intake rates are determined for a variety of potential activities that could significantly add to the amount of lead taken into the body. These rates are developed on an activity specific basis for both a typical or average case called the Central Tendency (CT) or a worst case called the

Reasonable Maximum Exposure (RME) estimate. Initial soil, dust, food, and water ingestion and inhalation values used for these intake calculations correspond to those developed for the non-lead risk assessment. Specific adjustments have been made to accommodate lead-specific factors or input from reviewers, the public or other interested parties.

Estimated incremental intakes are presented in tabular form including both the absolute intake value and the percent increment above baseline. In this manner, risk managers can evaluate the relative effect that incremental activities could have on baseline intake rates.

Occupational Intake Rates

Potentially significant occupational activities have been classified according to the likelihood of encountering soil and dust exposures in the normal course of employment. Three classification levels are proposed: nominal, medium (CT), and intensive (RME) corresponding to the following qualitative evaluation. Nominal exposures are consistent with typical residential behaviors and activities, or those occupations that have no special or particular relationship with contaminated soils. Nominal occupational exposures are not evaluated in this risk assessment. The medium classification corresponds to those individuals whose jobs involve periodic exposure to soil sources, such as public property maintenance, typical construction workers, or laborers (equivalent to the CT).

Intensive occupational exposure refers to individuals whose employment specifically involves exposures to soils such as landscapers; farmers and agricultural workers; remediation workers; construction workers routinely involved in excavation, demolition, or site development; or utility or road workers (equivalent to the RME). Intensive occupational scenarios also have a higher likelihood of high take-home lead dusts, or greater potential for secondary child exposures of workers' children, as more frequent contact with soils and dusts occurs in this occupational scenario. Mineral industry workers are specifically excluded in the occupational scenario, as exposure to lead is specifically regulated by occupational health authorities. Although individuals working in the mining industry are not evaluated in this HHRA for lead exposure in the workplace, they are considered in the residential scenario.

Soil and dust ingestion and inhalation rates, exposure frequencies, and contact times for these activities do not correspond with those developed for the non-lead portion of the risk assessment. Recommended ingestion rates from USEPA Adult Model for lead ingestion rates are employed for all adult lead health risk calculations. Comparison to non-lead intake parameters are shown in Tables 6-27 a-c. Lead intake rates for medium classification are represented by the CT estimates, with the RME representing the more intense exposure category. No estimates have been developed for nominally exposed populations. Table 6-27a shows intake parameters used for the non-lead portions of the risk assessment for the RME (or intensive) and CT (or medium) estimate. For lead risk assessment, alternate values corresponding to the EPA Adult model recommendations of 200 mg/day RME and

100 mg/day CT are used as noted in Table 6-27b. Table 6-28a shows typical RME and CT lead intakes respectively, for community mean soils by geographic subarea. Included in these tables are the estimated baseline intake rates assuming a 50 mg/day soil/dust ingestion rate. These results suggest that lead intake for an adult employed in medium intensity occupational activities involving typical residential soils would increase from 25% to 43% above baseline using the non-lead parameters. For the intense contact or RME activities lead intake would increase by 1.8 to 2.9 times. Table 6-28b and Figures 6-15a-b show similar lead intake estimates using the EPA Adult Model recommended ingestion parameters. These results suggest about 20% over baseline for CT and one to two times baseline for RME ingestion rates, respectively.

Tables 6-29a-b show estimated occupational intake rates associated with different potential soil concentration intervals. Incremental intake rates are shown both seasonally, based on the RME and CT outdoor work duration, and averaged annually. Seasonally adjusted intake rates for unprotected work in 2000 mg/kg soils, for example in Table 6-29a, are 246 µg/day for medium exposure and 419 µg/day for intensively exposed occupations. Averaged over the year (12 months) the respective values are 47 µg/day and 321 µg/day. Table 6-29a corresponds to non-lead ingestion rates. Table 6-29b shows adult occupational exposure for lead ingestion rates.

Tables 6-11a-j through 6-16 show typical soils concentrations for each residential area, waste piles in the upper Basin and soils/sediments in the upper Basin and Lower Basin floodplain. The appropriate estimated intake rates for occupational activities associated with these soils have been included in Table 6-30b. For example, the estimated annual average 50th percentile soil concentration CT intake rate for a median intensive occupation in the Lower Basin floodplain is 37 µg/day.

This baseline intake rate is also included in Table 6-30c. The estimated baseline intake rate for the Lower Basin is 10 µg/day. The combined baseline and incremental rate for an intensively exposed worker in the Lower Basin floodplain is 47 µg/day. This rate is 366% or 3.7 times increase over baseline for this subarea. Other examples in Tables 6-30b-c show the incremental and combined intake estimates and percent increases for occupational exposures to various soils throughout the Basin.

Recreational Activities Intake Rates

Proposed classifications of recreational activities are similar to the occupational categories. However, additional potential exposure routes, at-risk population factors, and exposure frequencies are considered.

Recreational activities have been classified according to the frequency of encountering contaminated environmental media and the amount of media contacted. With regard to soils and dusts, these activities can also be considered nominal, medium (CT) or intense (RME). Nominal activities are considered consistent with baseline residential activities and will not be developed as incremental exposures. Nominal exposures to particular sources within a community (e.g., a neighborhood

playground) are considered to be included in the soil partition for the baseline exposure. Medium intensity activities include picnicking, hiking, fishing, exploring, etc. (equivalent to the CT). Intense recreational activities include such practices as dirt biking, fort building, beach activities, four-wheeling, gardening, landscaping, etc., that involve deliberate and continued contact with soils (equivalent to the RME).

Ingestion of surface waters is not considered as a substitute source for typical drinking water during recreational activities. Surface waters are addressed as an incidental source of ingestion of suspended sediments during swimming and beach activities. Inhalation of contaminated dusts are considered incidental to soil/dust ingestion and are not developed as a separate pathway.

Intake rates are developed for sub-populations including young children and reproductive-aged women. Three exposure frequency categories are considered; incidental, seasonal, and year-round. Because significant increases in blood lead levels can be associated with short-term exposures on the order of a few weeks or months, only incidental and seasonal estimates are developed.

Soil and dust and surface water ingestion rates, exposure frequencies and contact times for these activities correspond with those developed for the non-lead portion of the risk assessment. No estimates are developed for nominally exposed populations as these exposures are incidental to the baseline.

Exposure point concentrations for recreational activities are developed at successive 500 mg/kg concentration intervals for soils and dusts. This results in classification of particular recreational areas according to lead content of the environmental media and estimated incremental intakes for both medium and highly exposed recreational groups.

Common Use Areas (CUAs) and other recreational locations are also classified by types of recreational activity supported. Factors determining type of activity include the following criteria:

- Intensity of use - nominal, medium, intensive.
- Sensitive population - frequented by young children, older children or adults only.
- Frequency of use - incidental or seasonal.

From this classification scheme, incremental intake estimates associated with particular recreational activities and locations can be obtained by applying the site-specific soil lead concentration and activity characteristics.

Incremental Soil Lead Intake. Table 6-31 summarizes the CT intake parameters for soil and dust media for the non-lead portion of the risk assessment. CT estimates are used for the medium exposure category and RME estimates for the intensive recreational activities.

Tables 6-32a-c summarize incremental intake estimates due to recreational activities for soils and sediments at successive potential concentration levels. Incremental intake rates are shown both seasonally, based on the number of weeks for each recreational activity, and averaged annually. Tables 6-32a-b show, respectively, medium (CT) and intensive (RME) intake rates for children. Seasonally adjusted intake rates for 1500 mg/kg soils in Upland Park areas, for example, are 13 µg/day for medium exposure and 64 µg/day for intensively exposed children's recreational activities. Averaged over the year these rates are 8 µg/day and 42 µg/day, respectively.

Similar values for adults are found in Table 6-32c. For 1500 mg/kg lead soils in Upland Parks, the seasonally adjusted rates for adults are 5 µg/day and 19 µg/day, for CT and RME conditions, respectively. Annual rates are 3 µg/day and 12 µg/day, respectively.

Incremental Surface Water Intakes. Table 6-33 shows intake factors for incidental ingestion of surface water in recreational scenarios. Tables 6-34a-b show incremental lead intake rates associated with potential surface water lead concentrations for disturbed sediment sites, respectively, for children and adults. These tables show seasonally adjusted and annual average incremental lead intakes associated with potential surface water concentrations at Common Use Areas and neighborhood streams for children. For example, a site with a characteristic surface water concentration of 5000 µg/l lead concentration would result 21 µg/day seasonal and 7 µg/day annual average lead intake for the CT recreational situation for children. Corresponding RME incremental intakes are 43 µg/day and 13 µg/day, respectively. For the neighborhood scenario at the same concentration, CT estimates are 43 µg/day seasonal and 20 µg/day annual. RME estimated intakes are 86 µg/day and 39 µg/day, respectively.

Table 6-12a shows typical concentration of lead in water and suspended sediment at public beaches in the Basin and those for local residential areas. Tables 6-14 through 6-16 show typical soil and sediment concentrations for neighborhood stream sediments, CUAs, and waste piles in the upper Basin.

Incremental Intake Rates for Upland Park CUAs. The appropriate estimated annual average CT intake rates for recreational activities associated with these soils have been included in Tables 6-35a-c for medium exposed 0-6 year old children. Table 6-35a also shows percentile surface soil lead concentrations for Upland Parks in the Mullan area, the mid-reach of the upper Basin from Wallace to the BHSS, and the Lower Basin from Kingston to Harrison. There are no Upland Park CUAs for the Burke/Nine Mile Canyon sub-unit. Incremental recreational increases for this geographic subarea are discussed under neighborhood sediments below.

Table 6-35b shows incremental lead intake rates associated with the respective percentile soil concentrations. Table 6-35c shows medium (CT) and reasonable maximum exposure (RME) intake rates (i.e., 50th and 95th percentile) compared to baseline intake rates for each geographic subarea. CT and RME annual intake rates are calculated using CT soil ingestion rates from Table 6-31. The

RME intake value in Table 6-35c refers to the 95th percentile media concentration calculated for the geographic area. Only CT ingestion rate estimates are developed for inclusion in IEUBK Model blood lead predictions in Section 6.6. Children practicing RME soil ingestion rates would have considerably higher lead intake rates than those indicated in Tables 6-35b-c.

The results in Table 6-35c can be used to assess relative intake rates. For example, the estimated annual intake rate for a medium (CT) child recreation in the Lower Basin floodplain (mean soil concentration 3415 mg/kg lead) is 19 µg/day. The baseline intake rates are also included in Tables 6-35b-c with the incremental intake rates. The combined baseline and incremental rate for a medium exposed child in the Lower Basin floodplain is 49 µg/day (19 µg/day incremental +30 µg/day baseline) using CT values. This rate is a 63% increase over baseline intake for the typical four year old child playing in 50th percentile soil lead levels in the Lower Basin.

Other examples in Tables 6-35b-c show the incremental and combined soil and dust lead intake estimates and percent increases over baseline for recreational exposures throughout the Basin. Generally, CT or typical incremental intake rates at 50th percentile concentrations are modest (5 to 7 µg/day) east of the BHSS. This represents a less than 10% increase over baseline for these areas. In the Lower Basin, incremental CT intakes are larger (19 µg/day), representing approximately a 40% to 60% increase over baseline rates.

RME results for 95th percentile concentrations in the upper Basin, however, are substantial in the mid-reach area, as a small number of CUAs are severely contaminated. RME (95th percentile concentration) upland park CUA intakes represent about 70% to 125% increase over baseline in these areas. RME incremental intakes in the Lower Basin are about 50% greater than CT increments, and are about twice the baseline.

Incremental Intake Rates for Neighborhood Sediments and Surface Water. Potential lead intake rates associated with recreational activities in or near streams in the immediate neighborhood of residences are shown in Tables 6-36a-c in the same manner as upland parks in Tables 6-35a-c. Percentile lead concentrations are shown in Table 6-36a for both surface water and sediment and corresponding lead intake rates are calculated in Table 6-36b. CT and RME intake rates are compared to baseline by geographic area in Table 6-36c. Generally, neighborhood streams contribute little to overall intake, as suspended sediment concentrations are relatively low compared to CUAs. Principal incremental intakes in this scenario are due to stream sediments and are most significant in the Burke/Nine Mile subarea (13 µg/day annual average) and the mid-reach areas (8 µg/day). These constitute almost a 15% increase over baseline for the CT situation. For the RME, substantial contributions to overall exposure are noted for Burke/Nine Mile (221 µg/day or nearly tripling baseline) and Mullan (45 µg/day), a 50% increase over baseline.

Neighborhood sediment and surface water contributions for the Lower Basin are low because these types of exposures generally occur at CUAs (discussed above) or public beaches (discussed below), rather than neighborhood streams as defined for the upper Basin.

Incremental Intake Rates for Public Beaches. Combined sediment and surface water lead intakes for public beaches included among the CUAs are shown in Tables 6-37a-c for children age 0-6 years. As opposed to neighborhood streams, these sites represent public access areas frequented by both Basin residents and visitors. As a result, the data have been combined and are uniform increments for all geographic subareas. The CT incremental lead intake for these sites is 19 µg/day for both sediment and surface water ingestion. The RME sediment intake rate is 30 µg/day lead and the corresponding surface water incidental ingestion rate is 42 µg/day, respectively. The combined CT rate adds 40% to 45% to the baseline intake for an upper Basin child, 60% to 70% for a mid-reach child east of the BHSS, 84% in Kingston, and 128% in the Lower Basin. The RME is a substantial contribution, nearly doubling baseline rates east of Wallace, more than doubling baseline in the mid-reach, and tripling baseline intake in the Lower Basin.

Incremental Intake Rates for Waste Piles. There are reported incidents of children playing on mining industry waste piles in the Burke/Nine Mile and Mullan subareas. Tables 6-38a-c show percentile lead concentration data for waste piles sampled in these areas, associated intake estimates and comparison of CT and RME intake to baseline. These results show that the assumed contact rates result in little CT incremental exposure in Mullan, although at 95th percentile concentrations there is a 14% increase over baseline in Mullan. Potential exposures in the Burke/Nine Mile area, however are significant, ranging from a 13% increase over baseline at the 50th percentile concentration to a 159% increase for the 95th percentile.

Lead Intake Rates from Local Foodstuff. Ingestion of lead associated with local foodstuff includes consumption of garden vegetables and fish from the lateral lakes area. Recommended USEPA typical and RME consumption rates for fish fillets are used for adults; child consumption rates were derived from the adult values by calculating a consumption rate in units of grams per kilogram per day then multiplying by the child body weight (15 kg). Lead intake estimates for consumption of local garden produce have also been developed using recommended USEPA default rates. Table 6-39 summarizes the intake parameters for garden produce and sport fish ingestion consistent with the non-lead portion of the risk assessment. Table 6-40a shows typical CT and RME child incremental lead intake rates for homegrown vegetables. The CT and RME estimates are based on the 50th and 95th percentile lead concentrations for fresh weight vegetables developed in Table 6-10. Both estimated CT and RME incremental intake rates can add substantially to baseline intakes. Consumption of 50th percentile concentration homegrown vegetables on a regular basis could increase total estimated intake by 24% in Wallace to 79% in the Lower Basin. Intake rates from 95th percentile produce are extreme. Table 6-40b shows typical CT and RME adult incremental lead intake rates for homegrown vegetables.

Tables 6-41a-b show similar results for sport fishery intake rates for children and adults. For adults typical incremental intake rates are modest. For children typical local fish consumption would increase total baseline intake by 1% to 12%.

Summary of Incremental Lead Intake Rates. Tables 6-42a-b and 6-43a-b and Figures 6-16a-d summarize relative intake rates for various incremental activities for children and adults, respectively.

6.5.4 Developing Intake Rates for the Coeur d'Alene Tribe

Tables 3-26a-b, respectively, show exposure factors for the Traditional and Modern Subsistence scenarios for the Coeur d'Alene Tribe. Figures 6-17a-b summarize estimated Tribal intake rates for children for each scenario, respectively. Adult intakes are summarized in Figures 6-18a-b.

Traditional Subsistence Intake Rates

Tables 6-44a-b show estimated lead intake rates, respectively, for children and adults for traditional subsistence pathways and percentiles for media concentration levels. Tribal intakes are not appropriately characterized as baseline or incremental, as the Tribal lifestyle is cumulative and encompasses all pathways simultaneously. As a result, Table 6-44a-b entries are arranged by media and percentile and are summed for corresponding percentile levels. This yields an estimated total intake rate for the sum of all like-percentile estimates. That is, all 50th percentile intakes from various media are combined for the 50th percentile total intake estimate. This method is likely appropriate for the CT estimate. However, the RME or any minimal intake estimate (e.g., 10th percentile) likely overestimates the upper confidence limit (UCL) and underestimates the lower limits, as the extreme rates for each pathway are less likely to coincide.

Total estimated intake rates are extremely high for both adults and children for all but the lowest concentration percentile categories. Intake rates for the CT and greater media concentrations typically exceed 1000 µg/day for children and 2000 µg/day for adults. These intake rates are an order of magnitude higher than observed intake rates in the residential population or pre-remedial estimates at the BHSS. High intakes of lead from dietary sources may present a greater hazard than lead in soil and dust because of potentially greater absorption of lead from dietary sources compared to lead in dusts, soils, or tailings, particularly in times of famine. Conversely, increased dietary absorption of lead may be mitigated by reduced lead absorption associated with a full stomach (Maddaloni, *et al.* 1998).

Tables 6-44a-b show estimated intake rates for both non-food and foodstuff for children and adults, respectively. Each table is presented in two forms. The upper portion of the table shows intake calculations using whole fish data from the Spokane River and unpeeled water potatoes. The lower portion shows fish fillet and peeled water potato lead content. The upper portion represents a worst

case situation where fish would be consumed whole and unpeeled water potatoes are a surrogate for the entire vegetable matter portion of the diet. Such assumptions are not unreasonable, according to Tribal sources, as these practices may have occurred in times of famine during traditional existence.

The lower portion of the table uses fish fillet and peeled water potato data. Total intake rates are reduced by 50% to 80% (as compared to whole fish and unpeeled water potato), but these values are not typical of a true, traditional scenario of whole fish and unpeeled water potatoes. However, regardless of this significant adjustment, estimated CT intake rates exceed 1300 µg/day for children and adults and are nearly double for the RME situation. These intake rates are also extremely high, about five to ten times pre-remedial levels at the BHSS.

Modern Subsistence Intake Rates

Figures 6-17a-b and 6-18a-b contrast estimated Traditional and Modern Subsistence intake rates. Modern Subsistence lead intake rates are summarized in Tables 6-45 and 6-46, which are arranged in a format similar to the Traditional intake estimate tables. Seasonal lead intake rates for the CT estimate are 258 µg/day for children and 311 µg/day for adults assuming only fish fillet and peeled water potatoes are consumed. RME intake rates are approximately 2 times CT estimates. These rates are similar to pre-remedial total intake rates at the BHSS. However, a greater percentage of total intake for subsistence pathways is from dietary sources that would be more bioavailable.

For modern subsistence families, the largest portion of intake is from ingestion of soils and sediments while engaging in subsistence activities on land and in disturbed surface waters. Consumption of whole fish or unpeeled water potatoes by modern subsistence families would result in dangerously high intake rates for both children and adults.

6.6 ESTIMATED BASELINE BLOOD LEAD LEVELS

6.6.1 Childhood Baseline Blood Lead Levels

The estimated Baseline intake rates for children developed in Section 6.5.2 were input to the IEUBK Model for both the EPA Default and Box Model scenarios. Estimates of mean blood lead levels and the percent of children to exceed 10 µg/dl were obtained from these applications in both the community and batch mode of the IEUBK Model. The community mode models predict blood lead levels based on geometric mean soil and dust concentrations for each geographic subarea. The model predictions are then compared to community mean blood lead observations. The following comparisons should be interpreted with caution and consideration of the uncertainties associated with both predicted and observed blood lead levels (Mushak 1998). The batch mode utilizes all paired blood lead data, or only those observations that have accompanying soil and dust lead measurements. Mean blood lead levels are then projected for each individual observation and the results are aggregated to develop community means that are then compared to the observed blood lead results. As such, the observed mean blood lead levels are not necessarily the same for both model applications. Some of the blood lead observations included in the overall community mean calculations for the community mode do not have corresponding environmental data and are not included in the batch mode runs. Exposure scenarios and corresponding input assumptions and data used for the IEUBK Model are summarized in Appendix V.

Community mode IEUBK Model Estimates

The community mode application of the IEUBK Model predicts outcome blood lead distributions from community mean exposures. In this mode, the default estimates of baseline dietary, air, and drinking water intakes were used and geometric mean soil and dust concentrations for each subarea were applied. Tables 6-11a-h summarize the soil and dust input database for this application.

The community mode application results in an estimated mean blood lead level for each age group. The percent of children to exceed the 10 µg/dl health criteria are calculated by applying a pre-determined geometric standard deviation to the mean. Tables 6-47a-c show predicted and observed mean blood lead levels and percent to exceed estimates for each subarea for various age groups.

Results for both the EPA Default and Box Model are shown. These models differ principally in the partition of soil and dust sources to overall intake and in the assumed bioavailability of soils and dusts. The differences in the soil and dust partition is discussed in Section 6.5.2. The EPA Default Model uses a 30% bioavailability estimate for soils and dusts, whereas the Box Model uses an 18% site-specific estimate derived in past analysis of BHSS blood lead monitoring results (USEPA 1994a, TerraGraphics 2000a). Appendix Q contains details regarding the development and parameters included in the Box Model.

Table 6-47a shows observed and predicted blood lead levels for 9-84 month old children using the community mode. For the EPA Default Model, predicted mean blood lead levels exceed the observed geometric mean for all subareas except in the Lower Basin, where observed geometric mean blood lead levels are 25% greater than EPA Default model predictions. In the upper Basin, EPA Default predictions are 1.4 to 1.9 times greater than the observed concentrations.

For the community mode application of the Box Model, an opposite pattern is noted. The Baseline Box Model under-predicts blood lead levels in the Lower Basin by a factor of 1.8 for the geometric mean, or the model predicts about 55% the observed mean blood lead concentration in this subarea. In Kingston, the observed means are equal to the prediction. Upstream from the BHSS, the predicted concentrations range from 1.0 to 1.3 times the observed geometric means, with Burke/Nine Mile blood lead levels slightly under-predicted and the Side Gulches and Mullan showing similar over-prediction.

Comparisons of predicted and observed percentages of children to exceed the 10 µg/dl health criteria follow a similar pattern, also shown in Table 6-47a. The Baseline EPA Default scenario tends to over predict the observed percentage by a factor of 1.8 to 4.3 in the areas east of Wallace. In the three easternmost geographic subareas, this model predicts 40% to 48% of children will exceed 10 µg/dl, whereas 10% of Mullan and 19 % of Wallace and 22% of Burke/Nine Mile children tested showed excessive levels. For the middle reach from Silverton to the BHSS the model predicts 15% to 21% exceedance where 0% to 11% of children were observed with greater than 10 µg/dl blood lead levels. The EPA default model under-predicts observed percent exceedance in the areas west of the BHSS predicting near 9% for Kingston and less than 2% for the Lower Basin, whereas 14% and 25% of children were observed with levels of 10 µg/dl or greater.

The community mode Box Model tends to predict the percentage of 9-84 month old children exceeding 10 µg/dl east of the BHSS fairly well. Estimates range from 15% to 19% east of, and including Wallace, compared to 10% to 22% observed. From 3% to 5% of children in the Silverton to BHSS reach were predicted to exceed the 10 µg/dl criteria and 0 to 11% were observed. Downstream from the BHSS, the Box Model greatly under-predicts observed toxicity projecting less than 5% exceedance compared to 14% to 25% of children in this age group exhibiting levels of 10 µg/dl or greater.

With younger age groups, similar patterns of over- and under-prediction are noted, though the magnitude of difference is less (Tables 6-47b-c). The EPA Default Model continues to significantly over-predict observed blood lead levels in the area east of Wallace. The Box Model effectively predicts both mean blood lead levels and percent to exceed 10 µg/dl in this area. Mean blood lead predictions and observations are within 1.2 µg/dl (i.e., 1-20%). Percent to exceed 10 µg/dl estimates range from 25% to 32% compared to 17% to 46% observed for less than 2 year old children (Table 6-47b). Respective predicted and observed estimates for 1 to 5 year old children (9-60 months) range from 20% to 25% percent predicted and 17% to 26 % observed (Table 6-47c).

In the reach from Silverton to the BHSS, the Box Model over-predicts observed 1-5 year old mean levels and under-predicts 2 year old levels by 0.3 to 1.3 µg/dl or generally within 10% to 20%. Percent to exceed the 10 µg/dl criteria predicted for two year olds ranged from 8% to 11% compared to the high of 19% observed in Silverton. For 1 to 5 year old children, the respective predictions were 5% to 8% compared to 0% to 13% observed. The EPA Default model over-predicts observed levels in the BHSS to Silverton reach by 1.2 to 2.0 times, or 25% to 97%.

West of the BHSS, both models significantly under-predict observed blood lead levels in the Lower Basin. Observed mean blood lead levels are more than twice those predicted by either model in the community mode. Similarly, neither of these models effectively captures the percent of children to exceed the 10 µg/dl health criteria in this geographic subarea. In the Kingston subarea, the EPA Default model over-predicts and the Box Model under-predicts observed levels. However, both models under-predict the percent to exceed criterion.

Batch mode IEUBK Model Estimates

Generally, when paired site-specific environmental and blood lead data are available, the batch mode application of the IEUBK Model more appropriately describes the distribution of blood lead levels. In contrast to the community mode, batch mode for comparative analysis was applied only to those homes with observed blood lead observations. The community mode included all homes for which soil and dust lead data were available. In the batch mode, each observation is sequentially applied to the model and the results are aggregated for the entire population. The batch mode was applied to the entire Basin population in a single run. Table 6-48 summarizes the characteristics of the input data set. A total of 445 observations were included in these model runs.

Some modifications were made to the data set to maximize the number of observations available. If either the soil lead or vacuum dust lead level was missing for any observation, the geometric mean value for that geographic subarea was substituted and the observation was retained. If both values were missing the observation was deleted. In cases where the tap water value was missing, a community geometric mean lead concentration (using the 1996 well water and purged tap water) was used.

A small number of children with high blood levels included in these analyses have been noted to have exposures outside the home. This is particularly true in the Lower Basin where recreational exposures were suspected in health department followup investigations. The model will tend to under-predict blood lead levels for these children. Some children are included in the analysis more than once as described in Section 6.2 2. Children tested more than once tended to have lower than average levels for children in their age group on the first test and similar levels on subsequent testing. These results would indicate that the model might over-predict concentrations for these children.

Table 6-49 summarizes the observed and predicted blood lead levels and percent of children to exceed the 10 µg/dl criteria Basin wide for three age groups. Overall, the batch mode of the EPA Default

Model over-predicts mean blood lead levels by 27% for 9-24 moth olds, 41% for 9-60 month olds and 43% for children age 9-84 months. The Box Model performs more effectively in this mode predicting 5.5 µg/dl versus 6.2 µg/dl observed for 9-24 month olds, 4.9 µg/dl versus 5.1 µg/dl for 9-60 month olds, and 4.5 µg/dl versus 4.6 µg/dl observed for children age 9-84 months. However, the EPA Default Model consistently over-predicts, and the Box Model under-predicts the percent of children to exceed the 10 µg/dl criteria.

Tables 6-50a-c and Figures 6-19a-c and Figures 6-20a-c show the results by geographic subarea. The observed mean blood lead levels, although similar, differ from those reported in the previous section due to differing number of observations included in the analysis. Results are reported for both the EPA Default and Box Model (40:30:30). The results are similar to the community mode model runs. Both arithmetic and geometric mean blood lead levels are over-predicted by the EPA Default model throughout the upper Basin. Predicted levels are more than two times observed concentrations in Mullan and Wallace and are approximately 50% higher in Burke/Nine Mile, Silverton, Osburn, and the Side Gulches. West of the BHSS, mean blood lead levels are better predicted by the EPA Default model. For Kingston, the arithmetic mean prediction is 5.0 µg/dl versus 5.7 µg/dl observed and 4.7 µg/dl predicted versus 4.3 µg/dl observed for the geometric mean. In the Lower Basin, the predicted and observed arithmetic means are 7.0 µg/dl and 6.9 µg/dl, respectively, and geometric means are 4.5 µg/dl predicted and 4.9 µg/dl observed.

In the upper Basin, the Box Model is an effective predictor of mean blood lead levels, generally over-predicting geometric means and predicting near or slightly below arithmetic means. The degree of over-prediction is greater from Wallace east and is minimal from Silverton to the BHSS. West of the BHSS, the Box Model tends to under-predict mean blood lead concentrations by approximately 10% to 50% in the batch mode application.

A similar pattern is noted with percent toxicity predictions. Generally, east of Wallace, the batch mode EPA Default Model over-predicts toxicity or the percent to exceed the 10 µg/dl health criteria, projecting 44% to 48% exceedance versus 13% to 22% observed. The Box Model projects 19 % and 20% exceedance for these communities. From Silverton to the BHSS, the Box Model also accurately predicts observed percent toxicity, projecting 5% to 8% to exceed versus 0% to 11% observed. The EPA default model predicts 17% to 26% exceedance in this reach.

West of the BHSS, both models underpredict the percentage of children greater than or equal to 10 µg/dl. The EPA Default version estimates 11% and 21% exceedance, respectively, for Kingston and the Lower Basin, as opposed to 17% and 32% observed. The Box Model predicts 2% and 14%, respectively, for these areas.

Tables 6-50b-c, respectively, show similar predicted and observed batch mode results for the 9-month through 24-month and 9-month through 60-month old children's age groups. Although the number of observations become scarce for some age/geographic area groups, the results tend to parallel the

community mode applications. Figures 6-19a-c show predicted and observed mean blood lead levels for each age group. These figures illustrate that, in general, the Box Model provides a better prediction of observed levels in the upper Basin, while the EPA Default Model performs better in the lower Basin.

Discussion of Baseline IEUBK Model Results

The results of the Baseline IEUBK Model applications suggest that there are potentially three different exposure situations ongoing in the Basin with respect to the residential soil and dust lead. East of the BHSS, the Baseline Box Model is a better predictor of observed blood lead levels. In these areas, the EPA Default Baseline model significantly over-predicts both observed concentrations and the percent of children to experience excess absorption. Immediately east of the BHSS in Osburn, the Side Gulches and Silverton, the Baseline Box Model fairly-well describes both observed mean blood lead levels and the percent of children exceeding the health criteria.

West of the BHSS and particularly in the Lower Basin the Box Model is ineffective, under-predicting both mean blood lead levels and percent exceedance. The EPA Default Model describes mean blood lead levels fairly well, but fails to capture the percent of children to exceed health criterion.

The magnitude of the variance in observed population blood lead levels, particularly in the Lower Basin, suggests a large variation in exposure. This is especially true relative to the Baseline intake estimates. A small number of children are exhibiting much greater blood lead levels than expected under any scenario from the Baseline intakes. Potential sources of these additional exposures are addressed in the incremental exposure analysis.

6.6.2 Resident Children's Incremental Blood Lead Levels

Recreational Exposures Blood Lead Increments

Incremental lead intake rates associated with children's recreational activities were developed in Section 6.5. Tables 6-32 through 6-41 show incremental intake rates associated with potential media concentrations for soils in upland park areas, neighborhood sediments and surface waters, public beaches, and waste piles. Tables 6-35 through 6-41 show typical percentile concentrations for these media for each geographic subarea and developed specific intake estimates using CT ingestion rates and CT (50th percentile) and RME (95th percentile) media concentrations for those areas.

Table 6-51a develops estimates of blood lead increments associated with the CT recreational intake estimates for upland parks from Table 6-35. This is accomplished by adding the estimated annual average intake in µg/day to the baseline model for each subarea through the *Other Source* option of the IEUBK Model. For example, in Table 6-51a, the 50th percentile recreational intake estimate for upland park areas in Mullan is 7 µg/day. Entering this rate through the *Other Source* route in the

Community Mode IEUBK EPA Default baseline model (Table 6-47a), results in an estimated blood lead level of 9.8 µg/dl for 9-84 month old children. This compares to the baseline estimate of 9.3 µg/dl from Table 6-47a, or a 0.5 µg/dl increase. This represents about a 5% predicted increase in mean blood lead level associated with the 7% increase in intake for the CT upland park media concentration of 1189 mg/kg from Table 6-35a.

Table 6-51b shows the addition of the 95th percentile recreational intake increment. This represents a child (9-84 months) playing at the most contaminated upland park CUA (e.g., 3270 mg/kg from Table 6-35a for Mullan). This 20% increment in intake results in a mean blood lead estimate of 10.6 µg/dl, an increment of 1.3 µg/dl, or a 14% increase in blood lead level.

Tables 6-51a-b also show the estimated percent of children to exceed 10 µg/dl if they were to engage in these play activities at the upland park locations. For the Mullan example, the EPA Default Model predicts that 43% of 9-84 month old children will exceed 10 µg/dl due to the baseline exposure (See Table 6-47a). This value can be interpreted as the probability that a child would experience an excessive blood lead level associated with baseline exposures in Mullan according to EPA Default Model estimates. Tables 6-51a-b, respectively, show that this probability increases to 45% for the CT exposure and 53% for the RME.

Tables 6-51a-b also show corresponding estimates developed using the Box Model to predict baseline and incremental blood lead levels. Similar results are shown for neighborhood streams and sediments in Tables 6-52a-b, public beaches in Tables 6-53a-b, and waste piles in Tables 6-54a-b.

Discussion of Children's Recreational Incremental IEUBK Model Results

Figures 6-19a-h summarize the incremental blood lead results for each geographic area. These are discussed in the following four sub-sections. *Upland park activities* refer to general recreational practices in contact with soils in non-water related activities. Swimming and intense contact with waters and sediments are discussed in the *public beaches* section. Wading and stream exploration-type activities in the general residential area near homes are discussed under *neighborhood sediments and surface water*. Contact with *waste piles* is discussed in the final sub-section below.

Upland Park Activities. Figures 6-21a-h show the observed mean and estimated baseline and baseline plus incremental blood lead levels associated with upland park recreational activities for each geographic subarea. These results suggest that there are relatively modest potential increases in blood lead levels associated with typical upland park activities in the upper Basin in comparison to baseline levels. For the 50th percentile (or CT) CUA soil concentration typical incremental intake and blood lead increases are less than 10% of baseline east of the BHSS. For the RME, however, there are substantial increases projected for extremely contaminated play areas in the upper Basin. No upland park CUAs were identified for the Burke/Nine Mile area. Recreational activities for this subarea are discussed under neighborhood sediments and surface water and waste piles.

West of the BHSS, conversely, there are substantial potential increments for these activities in typical (CT) recreational activities in the Kingston and Lower Basin areas relative to baseline estimates. Inclusion of potential recreational intakes in these areas results in near 50% increases above baseline intake rates and projected blood lead levels.

Neighborhood Sediments and Surface Water. Figures 6-21a-h show the observed mean and estimated baseline and incremental blood lead levels associated with neighborhood sediments and surface water recreational activities for each geographic area. For the typical or CT media concentration levels, minimal intake and blood lead increments are projected for contact with local sediments and stream water in Mullan and the lower Basin. In this case, exposures in the lower Basin are limited to the Pine Creek area. Most water contact exposures for the lower Basin are discussed as public beach activities. Near 10% increases are predicted in both intake and blood lead for the remainder of the upper Basin, with expected levels somewhat higher in Burke/Nine Mile. For the RME, however, the most contaminated streams result in 20% to 35% increases in intake for the Wallace to BHSS reach, a 50% increase in Mullan, and nearly a tripling of baseline intake in Burke/Nine Mile (271% increase).

Public Beaches. Figures 6-21a-h show for each geographic area the observed mean and estimated baseline and incremental blood lead levels associated with public beach recreational activities. Public swimming areas are assessed throughout the Basin and are not subdivided by geographic subarea. However, for this analysis the incremental exposure is added to baseline for each subarea. Substantial incremental intake and estimated blood lead increments are predicted for sediment and disturbed surface water ingestion at public beaches throughout the upper and Lower Basin for typical or CT concentrations. Estimated incremental blood lead levels range from 13.2 µg/dl for Wallace children to 6.6 µg/dl in the Lower Basin. Respectively, these are 33% and 36% potential increases in blood lead level associated with public beach activities at 50th percentile concentrations. Engaging in these activities at RME (95th percentile) sites can result in more than doubling annual intake rates and substantial blood lead increases for children throughout the Basin.

Waste Piles. Figures 6-21a-h show for each geographic area the observed mean and estimated baseline and incremental blood lead levels associated with play on waste piles in the upper Basin areas of Mullan and Burke/Nine Mile. For the 50th percentile (CT) concentrations incremental exposures are minimal in Mullan and an 8% increase over baseline blood lead levels in Burke/Nine Mile. At the RME (95th percentile) concentration, a 14% increase in intake is projected for Mullan and a near doubling of total intake is predicted for Burke/Nine Mile.

Summary of Children's Recreational Incremental Exposures. Potentially significant recreational exposures are noted for certain activities in particular areas of the Basin. Upland park type recreation can result in significant exposures in the more contaminated areas of the upper Basin and throughout the areas west of the BHSS. Potential recreational exposures in the lower Basin are more significant because of both higher soil concentrations and lower baseline residential exposures. This can result in

higher dose-response rates to incremental exposures at lower blood lead levels. This is a possible explanation for the higher than predicted blood lead levels observed among lower Basin children.

Additionally, swimming and water sport activities that could result in ingestion of disturbed sediment laden surface water can result in substantial increases in intake and lead absorption. Potential exposures are of particular concern to neighborhood stream sediments in Burke/Nine Mile, and at public swimming areas in the Side Gulches and the Lower Basin.

Incremental Blood Lead Levels Associated with Local Foodstuff

Homegrown Produce. Tables 6-55a-b show estimated CT and RME blood lead levels, respectively, associated with consuming local produce. Potentially significant increases in blood lead levels could result from consumption of home grown vegetables. Increased intake from foodstuff can result in higher blood lead levels due to the high bioavailability of dietary lead.

Sport Fishery. Tables 6-56a-b show similar results for local fish consumption. Figures 6-21e-h show baseline and estimated incremental blood lead levels by geographic area including, local foodstuff.

6.6.3 Adult Model Blood Lead Estimates

Adult Occupational Blood Lead Level Estimates

Tables 6-57 and 6-58 show the USEPA Adult Model for lead applications for occupational Preliminary Remediation Goals (PRGs) for the parameters and intakes shown in Tables 6-27 through 6-30, respectively, for CT and RME scenarios. The EPA Adult Model was used to determine the concentration value for soils that would result in an upper confidence limit (95th percentile) blood lead level of 10 µg/dl for women of child-bearing age. The results suggest that for CT or medium intensive soil contact occupations, the probability for blood lead levels to exceed 10 µg/dl is greater than the 5% criteria at soil concentrations ranging from 2800 mg/kg to 4500 mg/kg. For intensively exposed or RME occupational activities, corresponding values range from 300 mg/kg to 500 mg/kg.

Adult Recreational Blood Lead Level Estimates

Tables 6-59a-b and 6-60a-b show CT and RME values with PRG calculations for upland park and CUA recreational activities for adults. For intense soil contact recreational practices such as dirt biking, beach activities, four-wheeling, gardening, landscaping, etc., that involve deliberate and continued contact with soils, the probability for blood lead levels to exceed 10 µg/dl is greater than the 5% criteria at concentrations ranging from 3700 mg/kg to 6400 mg/kg lead. These values correspond to the 90th to 95th percentile concentrations in upper Basin recreational areas and 50th to 95th percentiles among Lower Basin CUAs.

6.6.4 Native American Blood Lead Levels

Blood lead levels were not estimated for either the traditional or modern subsistence scenarios because estimated lead intake levels ranged from 300-1000 µg/day. These high rates coupled with cultural-specific dietary and behavioral considerations precluded estimating all RME and CT scenarios. Estimated lead intake rates for these scenarios are too high to predict blood lead levels with confidence. Predictions for blood lead levels associated with subsistence activities in the floodplain of the lower Basin would significantly exceed all health criteria for children or adults in either scenario.

6.7 RISK CHARACTERIZATION

6.7.1 Overview and Summary

The risk of lead poisoning among adults and children has been assessed in several ways in the preceding sections. Observed blood lead levels indicate a significant and consistent incidence of elevated blood lead levels among the children in the Basin who have been tested for lead. The observed blood lead levels and rates of elevated blood lead have changed little from 1996 through 2000 despite varying levels of participation in the Basin during these years. The consistency of the blood lead levels increases the confidence in the representativeness of the blood lead observations. Quantitative site-specific analysis of paired blood and environmental variables indicate excess absorption is associated with a combination of contaminated soils, house dust and paint sources. Lead intake estimates developed for various pathways indicate potentially significant rates associated with both baseline (everyday home life common to the entire population) and incremental exposures (certain activities practiced by part of the population).

Predictions of blood lead levels and percent of resident children to experience excess absorption was accomplished with two IEUBK Models, both applied in the community and batch modes. The EPA Default version predicts higher blood lead levels and percent of children to exceed 10 µg/dl than the site-specific model employed at the neighboring BHSS. Both models predict excess absorption rates associated with baseline residential exposures for the upper Basin east of Silverton. The models provide conflicting results in Osburn and the Side Gulches. The EPA Default model suggests greater than 5% risk of exceeding the 10 µg/dl criteria, and the Box Model projects 5% exceedance. Using *baseline residential exposures*, neither model predicts the degree of excessive absorption observed in Kingston or the Lower Basin. Both models predict potentially significant incremental blood lead levels associated with particular recreational activities in contaminated areas, and consumption of local foodstuff. These incremental exposures may explain the higher than expected blood lead levels observed in the Lower Basin.

Observed blood lead levels among adults are significantly lower, particularly among reproductive aged females. Older individuals (≥50 years) tend to have higher blood lead levels in the adult population. Intake and Adult Model blood lead predictions indicate potentially significant risks associated with occupational and recreational activities in certain areas, and consumption of local foodstuff.

Evaluation of potential Native American lifestyle practices in the Lower Basin indicates severe lead intake rates for both children and adults in either the traditional existence or modern subsistence scenarios. Both non-food (soil, sediment, and water), and fish and riparian foodstuff exposure routes result in unacceptably high lead intake levels.

6.7.2 **Indices of Lead Health Risk** (adapted from CDC, 1997)

The risks associated with elevated blood lead levels are characterized by comparison to the CDC criteria: excessive prevalence of blood lead levels in the 10-14 µg/dl range are indicative of excess exposure to the individual; levels of 15-19 µg/dl are indicative of excessive lead absorption and require education and nutritional intervention and more frequent screening; levels of 20-44 µg/dl require medical and environmental intervention and perhaps chelation; levels of 45 µg/dl and higher (45-69) require environmental and medical intervention with chelation therapy. Children with blood lead levels at or above 70 µg/dl require immediate hospitalization and chelation therapy, along with immediate environmental management.

USEPA policy seeks actions that limit exposure to soil lead levels such that a typical (or hypothetical) child or group of similarly exposed children would have an estimated risk of no more than 5% likelihood of exceeding a 10 µg/dl blood lead level (USEPA 1994d, USEPA 1998f - Attached as Appendix O). The USEPA also recommends the use of the individual residence as the primary exposure unit of concern and that the IEUBK Model be used as the primary tool to estimate risk. This policy requires that the probability of the typical 9-84 month old child at any residence experiencing a blood lead level of 10 µg/dl or greater be no more than 5%. This differs from the RAO adopted at the adjacent BHSS that requires that no more than 5% of the community wide population of nine month to nine year old children have levels of 10 µg/dl or greater and that less than 1% of children have levels of 15 µg/dl or greater. Comparisons of predicted blood lead levels to both criteria are accomplished below.

6.7.3 **Observed Blood Lead Levels**

The highest prevalence of elevated blood lead levels among 9 month through 9 year old children is observed in Burke/Nine Mile at 21% exceeding 10 µg/dl, 13% exceeding 15 µg/dl and 4% with levels of 20 µg/dl or greater. The Lower Basin subarea showed the next highest toxicity rate with 18% exceeding 10 µg/dl and 5% greater than 15 µg/dl. No children were in the 20 µg/dl range in the Lower Basin. Wallace, Mullan and Silverton, respectively, showed 13%, 11% and 8% of children with levels of 10 µg/dl, or greater. From 4% to 5% of children tested in Wallace and Silverton exhibited blood lead levels exceeding 15 µg/dl and 1% to 3% exceeded 20 µg/dl, respectively. Osburn and the Side Gulches area both showed 4% of children exceeding 10 µg/dl and only one child (in the Side Gulches) in four years exceeded 15 µg/dl. Kingston showed 11% greater than or equal to 10 µg/dl and 7% exceeded the 15 µg/dl.

The highest blood lead levels are observed in the youngest age groups. One and two year old children have arithmetic mean blood lead levels of 7.0 µg/dl and 8.0 µg/dl , respectively, and geometric mean concentrations of 6.2 µg/dl to 6.3 µg/dl. Geometric mean levels then decrease with age from 5.2 µg/dl at age 3 to 3.0 µg/dl at age 8.

The percent of children to exceed critical toxicity levels differs markedly with age. In the lowest age groups, 9 months to 36 months, 19% to 26% exceed 10 µg/dl. The rate is highest in 2 year old children with 17% of this group exceeding 15 µg/dl. For four year old children, 12% exceed 10 µg/dl and 5% exceed 15 µg/dl. In older children, the percent to exceed 10 µg/dl ranges from 5% to 8%, and 1% to 3% exceed 15 µg/dl.

Follow-up investigations were completed by the local health department for 50 of 58 children whose blood lead levels exceeded 10 µg/dl. Twenty-six investigations involving 21 individual children were for observed blood lead levels exceeding 15 µg/dl. Risk profiles indicate excess absorption associated with high soil and dust concentrations at homes in the Burke/Nine Mile subarea. Older children's risk profiles in this area indicate recreational exposures in neighborhood areas contaminated by tailings. High blood lead levels in Wallace are indicated in younger children and are possibly associated with paint and remodeling problems, high soil lead levels in play areas, and dusty or difficult to clean homes. Both Mullan and Osburn had no children with blood lead levels above 15 µg/dl and children's blood lead levels in the 10-14 µg/dl range were associated with high residential soil and dust concentrations or play in contaminated areas. West of the BHSS, excess absorption was associated with either homes that had been flooded or extended recreational activities in the river or lateral lake areas of the Lower Basin.

6.7.4 Site-specific Analysis of Paired Blood Lead and Environmental Source Observations

Site-specific quantitative analysis of the relationship between blood lead levels and environmental variables indicate that contaminated soils, house dust and lead based paint are all related to excess absorption. The overall results suggest complex exposure pathways, with lead absorption levels most related to dust lead loading rate in the home, followed by independent effects of yard soil lead, interior paint lead condition and exterior paint lead content. The dust lead pathway is most influenced by outdoor soils, augmented by paint contributions in older homes, especially those in poor condition. The overall effect is exacerbated by extremely dusty conditions in Burke/Nine Mile and to a lesser extent in Wallace. The Lower Basin is a notable exception. High blood lead levels are observed, although little problem is indicated with respect to dustiness or dust lead concentrations in the Lower Basin.

Quantitative models relating blood lead levels to soil, house dust, and paint lead levels and house dust levels to soil and paint sources were developed. These are used below to quantify baseline exposures and project risk reductions that might be achieved through source modifications.

6.7.5 Predicted Blood Lead Levels

The USEPA recommends the use of the IEUBK Model for estimating risks for childhood lead exposure from a number of sources, such as soils, dust, air, water, and other sources to predict blood lead levels in children 9 months to 84 months old. EPA recommends that the IEUBK Model be used as the primary tool to generate risk-based soil cleanup levels at lead sites for current or future residential

land use. Response actions can be taken using IEUBK Model predictions alone; blood lead studies are not required (USEPA 1994d, 1998f).

USEPA policy also recommends that risk assessments use the individual residence as the primary exposure unit of concern. This does not mean that a risk assessment should be conducted for every yard, rather that the soil lead contamination data from yards and other residential media (for example, interior dust and drinking water) should be input into the IEUBK Model to provide a preliminary remediation goal (PRG) for the residential setting. When applicable, potential exposure to accessible site-related lead sources outside the residential setting should also be evaluated to understand how these other potential exposures contribute to the overall risk to children, and to suggest appropriate cleanup measures for those areas (USEPA 1994d, 1998f).

This policy has been addressed in this assessment. Lead health risks in the residential setting are projected through ***baseline residential exposures*** and those outside the residential setting are assessed through ***incremental exposures***.

Residential Baseline Blood Lead Level Predictions

Residential baseline (everyday home life) blood lead predictions were accomplished using four different applications of the IEUBK Model. Both the EPA Default Model (using national assumptions for soil and dust ingestion rates and bioavailability) and the Box Model derived specifically for the BHSS were employed. Both models were applied in both the community and batch modes. The results suggest that there are potentially three different exposure situations ongoing in the Basin with respect to the residential soil and dust lead.

East of Wallace, the baseline Box Model is a better predictor of observed mean blood lead levels, although the percentage of younger children above the 10 µg/dl criteria was under predicted in Wallace, Silverton, Osburn, Kingston, and the Lower Basin (See Figure 6-20c). In these areas, the EPA Default baseline model significantly over-predicts both observed concentrations and the percent of children to experience excess absorption. In the community mode, both models predict more than 5% of 9-84 month old children will exceed the 10 µg/dl criteria in Mullan, Wallace, and Burke/Nine Mile. The EPA Default Model predicts 40% to 48% exceedance in these areas, the Box Model predicts 15% to 19% above the criteria. Observed exceedance in these areas ranged from 10% to 22%.

Immediately east of the BHSS in Osburn, the Side Gulches and Silverton, the batch mode Baseline Box Model fairly-well describes both observed mean blood lead levels and the percent of children exceeding the health criteria. Observed exceedance of the 10 µg/dl criteria for 9-84 month old children ranged from 0% to 11% in this reach. The EPA Default Model predicts 17% to 26% exceedance ***associated with baseline residential exposures*** for these areas, as opposed to the Box Model 5% to 8% projection.

West of the BHSS and particularly in the Lower Basin the Box Model is ineffective, under-predicting both mean blood lead levels and percent exceedance. The EPA Default Model fairly-well describes mean blood lead levels, but underestimates percent of children to exceed the health criteria. Both the EPA Default and Box Models failed to predict these high blood lead levels. The community mode estimates for Kingston (14% observed greater than 10 µg/dl) were 9% and 2%, respectively, for the EPA Default and Box models. For the Lower Basin (25% observed greater than 10 µg/dl), the respective community mode predictions were 2% and 0%. Batch mode estimates for Kingston were 11% for the EPA Default Model and 2% for the Box Model. Batch mode estimates for the Lower Basin were better, 21% and 14%, respectively, but also under-predicted the 32% observed.

There are several possible factors that could contribute to the difference in exposures and blood lead levels among these areas of the Basin. There could be physical and chemical differences in the soil and dust contaminants. Differences in chemical form, particle size and matrix effects could result in different physical accessability and bioavailability to children. These differences could be attributable to the original source of the lead from mine, mill or smelter wastes, or from the degree of weathering and secondary mineralization that has occurred while in the environment.

The degree of dustiness, vegetative cover, and snow cover in these communities could be a factor, as the larger communities have curbs and gutters and other infrastructure that is not available in the smaller villages. The size of yards, use of lead paint, age of the communities and proximity to industrial or transportation sources could all impact this relationship. The habits and behavior of children, particularly as they move about neighborhoods and select favorite play areas and activities may present important differences in the larger cities, small residential areas or rural homes.

The Batch mode more accurately reflects variance in exposures and is a better predictor than the community mode of the IEUBK Model. The EPA Default version of the IEUBK Model Batch Mode application predicts a greater than 5% exceedance of the 10 µg/dl health criteria, ***associated with baseline residential exposures***, for all geographic areas. The Box Model predicts exceedance greater than 5% for Mullan, Burke/Nine Mile, Wallace, Silverton and the Lower Basin. The areas adjoining the BHSS including Kingston, Osburn and the Side Gulches are projected at or less than 5% exceedance for baseline residential exposures by the Box Model (see Figure 6-20a).

Incremental Exposure Blood Lead Predictions

Potentially significant recreational exposures are noted for certain activities in particular areas of the Basin. Upland park type recreation can result in significant exposures in the more contaminated areas of the upper Basin and throughout the areas west of the BHSS. Recreational exposures in the Lower Basin are more significant because of both higher soil concentrations and lower baseline residential exposures. This can result in higher dose response rates to incremental exposures at lower blood lead levels. This is a possible explanation for the higher than predicted blood lead levels observed among Lower Basin children.

Swimming and water sport activities that could result in ingestion of disturbed sediment-laden surface water can result in substantial increases in intake and lead absorption. Potential exposures are of particular concern to neighborhood stream sediments in Burke/Nine Mile, and at public swimming areas in the Side Gulches and the Lower Basin.

Potentially significant increases in blood lead levels could also result from consumption of home grown vegetables. Increased intake from foodstuff can result in higher blood lead levels due to the high bioavailability of dietary lead.

Adult Resident Population Blood Lead Predictions

Occupational. Adult blood lead model estimates were developed for medium intensity (CT) soil contact occupations or jobs involving periodic exposure to soil sources, such as public property maintenance, typical construction workers, or laborers. These results suggest that exposures to soils ranging in lead concentration from 2800 mg/kg to 4500 mg/kg could result in more than a 5% probability of blood lead greater than 10 µg/dl. Few soil concentrations in this range are observed in residential areas of the Basin. In upland park CUAs these values correspond to the 90th to 95th percentile of sites. In the Lower Basin floodplain 50% to 95% of soils exceed these levels.

Intensive or RME exposure refers to individuals whose employment specifically involves exposures to soils such as landscapers; farmers and agricultural workers; remediation workers; construction workers routinely involved in excavation, demolition, or site development; or utility or road workers. Mineral industry workers are excluded from the occupational scenario, as exposure to lead is specifically regulated by occupational health authorities. Although individuals working in the mining industry are not evaluated in this HHRA for lead exposure in the workplace, they are considered in the residential scenario. For these workers, soils near 500 mg/kg could result in more than a 5% probability of having a blood lead level greater than 10 µg/dl.

Recreational. For typical (CT) adult recreational activities, less than 5% probability of exceeding 10 µg/dl is predicted for all recreational area soil concentrations observed in the Basin. For intense (RME) soil contact recreational practices such as dirt biking, beach activities, four-wheeling, gardening, landscaping, etc., that involve deliberate and continued contact with soils, 95th percentile blood lead estimates exceed 10 µg/dl at concentrations ranging from 3700 mg/kg to 6400 mg/kg lead. These values generally represent the 90th to 95th percentile concentrations in upper Basin recreational areas and 50th to 95th percentiles among lower Basin CUAs.

Native American Blood Lead Levels

Blood lead levels were not predicted for either the traditional or modern subsistence scenarios because extremely high intake rates coupled with cultural-specific dietary and behavioral considerations exceed the capabilities of current blood lead models. Nevertheless, projected intake rates are sufficiently high

to suggest that blood lead levels associated with subsistence activities in the floodplain of the Lower Basin would significantly exceed all health criteria for children or adults in either scenario.

The high lead intake rates are associated with several media. Soil and sediment intakes, fish fillet and peeled water potato, and ingestion of disturbed surface water during swimming and bathing activities would each individually result in excessive lead intake. Consumption of whole fish from the Spokane River or un-peeled water potatoes from the Lower Basin would result in especially dangerous intake levels.

6.7.6 Potential Lead Health Risk Reduction Strategies

The results suggest complex pathways of exposure are ongoing in the Basin. Resident children's blood lead levels are most related to dust lead loading in the home, followed by independent effects of yard soil lead, paint lead condition, and exterior lead paint content. The dust lead pathway is most influenced by outdoor soils, but is augmented by paint contributions particularly in older homes in poor condition. The overall effect is exacerbated by dusty conditions in Burke/Nine Mile and to a lesser extent in Wallace. Fewer problems are noted with respect to dustiness or dust concentrations in the Lower Basin. West of the BHSS, excess absorption was associated with either homes that had been flooded or extended recreational activities in the river or lateral lake areas.

Significant recreational exposures are noted for certain activities in particular areas of the Basin and from consumption of home grown vegetables. Excessive occupational exposures could occur with particular unprotected jobs in highly contaminated areas. Subsistence Native American practices in the Lower Basin are dangerous, particularly if whole fish or unpeeled water potatoes made up a substantial portion of the diet.

An integrated approach to risk reduction is needed to reduce multiple sources of lead exposure. Baseline residential exposures could be reduced through cleanup of excessive soil contamination coupled with paint stabilization to simultaneously reduce direct exposure to these media and house dust lead concentrations. Targeted cleanups of recreational areas, coupled with access limitations or appropriate warnings, could be used to prevent excessive incremental exposures. Provision of clean gardening media could reduce incremental exposure to local produce. Worker safety protocols could be developed to protect adults while employed in contaminated soil related jobs. Native Americans should continue to refrain from food harvest and subsistence activities in the Lower Basin until substantial improvements are made. In the interim, individual children's exposures could be reduced by removal actions and continuing and enhancing current health intervention activities until final remedial determinations aimed at primary prevention are completed.

For the residents, children's baseline blood lead levels are likely to be the determining factor in establishing media-specific remediation goals or concentration action levels. The baseline blood lead

levels then become a critical determinant in developing required risk reduction strategies for incremental, or away from home, activities.

Discussion and development of candidate action levels for children's incremental recreational activities and fish and local produce consumption ultimately depend upon risk management decisions that are forthcoming and cannot be addressed by the risk assessment. Comprehensive risk reduction methods and action levels will be evaluated by risk managers after fundamental approaches to reducing baseline blood lead levels have been identified.

Resident Childhood Population Baseline Risk Reduction

Community Mode IEUBK Model Blood Lead Projections for Various Cleanup Action Levels.

The community mode of the IEUBK model was used to conduct an abbreviated sensitivity analysis regarding potential residential soil cleanup remedies. Select input parameters were varied in the IEUBK to evaluate those most likely to influence outcome blood lead levels in soil lead reduction scenarios. Only soil and dust concentration variables were modified in this analysis. The soil and dust partition and bioavailability assumptions inherent in the EPA Default and Box Model applications were retained in these analyses. Outcome dust lead concentrations resulting from soil and paint lead remediation efforts were found to be the most important determinant of post-remedial blood lead levels. The key to evaluating this strategy is estimating the effect any soil or paint remediation efforts will have on dust lead concentrations. Figures 6-22a-b demonstrate, respectively for Wallace and the Lower Basin, the sensitivity of the predicted percent of 9-84 month old children to exceed the 10 µg/dl health criteria for various soil and dust lead concentration reduction scenarios. Tables 2 and 3 and Figures 1 through 3 in Appendix R show results for all subareas in tabular and graphic format.

Figures 6-22a-b show results for a remediation strategy addressing home yards with soil lead concentrations exceeding action levels varying from 2000 mg/kg to 400 mg/kg. In each case it is assumed that soils exceeding the action level are replaced with soils of less than 100 mg/kg. The value of the resulting community mean lead level is then recalculated using these substitute values for remediated yards. Tables 2 and 3 in Appendix R show the resulting community mean soil lead level. This value is input to the community mode IEUBK Model and outcome blood lead levels are estimated for both the EPA Default and the Box Model.

Two potential effects representing upper and lower bounds of any dust lead reduction are assessed in Figures 6-22a-b. Those bounds are i) dust lead levels remain unchanged, or ii) dust lead levels reduce to the geometric mean soil lead concentration. Examination of the results in Figure 6-22a lead to the following conclusions:

Dust lead levels in the upper Basin are critical determinants of the efficacy of any cleanup strategy.

To achieve acceptable blood lead levels, a substantial reduction of house dust lead levels in the upper Basin will be necessary.

Figure 6-22a shows that if Wallace dust lead levels remain unchanged, then the 5% community exceedance goal is unattainable in the EPA Default analysis and only marginally attainable for yard soil cleanup of 600 mg/kg, or less, under the Box Model assumptions. Under the assumption that dust concentrations decrease to concurrent soil lead projections, a 5% community exceedance of 10 µg/dl is achieved at the 1500 mg/kg level for the Box Model and 1000 mg/kg for the EPA Default Model. Reducing risks to individuals, as required by current EPA guidance, would require more protective soil levels.

In the Lower Basin (shown in Figure 6-22b), and to a lesser extent in the Kingston subarea, yard soil and house dust lead concentration reductions are unlikely to be effective in reducing observed high blood lead levels, unless these homes are located within the floodplain. Residential soil and dust lead concentrations in these areas are generally low and baseline intake rates do not suggest an absorption problem. For these areas, excepting some individual situations, development of strategies addressing incremental exposures outside the home environment are more likely to be effective in reducing risk of lead poisoning. Because residential soil and house dust concentrations are generally low in the Lower Basin, it is likely that children residing on properties located outside of the floodplain are receiving much of their exposure outside of the home, including recreational areas.

Estimating Post-remedial Soil and Dust Lead Concentrations. In assessing risks from potential action levels for soils and dusts, there are substantial differences between the EPA Default and Box Model projections. Use of the EPA Default model would require substantially lower action levels than the Box Model projections. The EPA Default Model predicts future levels based on nationally derived assumptions of soil/dust intake and typical bioavailability. The Box Model assumes that past relationships between environmental lead and blood lead levels observed in the BHSS are predictive of future exposures and behavior patterns in the Basin. Although calibration of the lead model with blood lead data can accurately describe past relationships, its predictive value depends on sustaining patterns of behavior and levels of awareness that modify levels of exposure. Behavior modification has not yet been proven effective as a long-term approach to preventing lead hazards.

In either model format, the key to evaluating risk is estimating the dust lead concentration resulting from differing levels of soil or paint remediation. The site-specific analysis was conducted to preliminarily assess the efficacy of any strategy adopted to reduce dust lead levels. This analysis suggests that blood lead levels are highly dependent on dust lead loading rates, yard soil contamination levels, and paint lead, particularly in poorly maintained housing. Dust lead loading rates, in turn, are dependent on both dust loading or dustiness in a community and the lead content of that dust. Outdoor soils both in the

yard and the community are the primary determinant in dust mat lead concentrations augmented by interior paint lead levels, again in poor quality housing.

The quantitative relationships developed in the site-specific analysis can be used to predict the effects of risk reduction strategies. Figures 6-23a-h show, for each subarea, projected community soil, dust mat and vacuum bag dust lead levels. The latter values are predicted by substitution of the appropriate soil concentrations into the regression model equations developed in Tables 6-22c and 6-23. This was a two step process, first estimating mat lead concentration in equation 6-22c from yard and community mean soil lead levels assuming good minimum paint condition. The resultant mat concentration estimate was then substituted with soil and mean, or typical, interior paint concentration in equation 6-23 to estimate the vacuum bag dust lead concentration. Both projections assume paint stabilization has been implemented in the poorest quality housing.

Batch Mode IEUBK Model Blood Lead Projections for Various Cleanup Action Levels.
IEUBK Model batch mode estimates are made using the entire 994 home data base assembled for the Basin. Mean blood lead levels are estimated for 9 through 84 month old children at every home in the Basin using the observed yard soil lead concentration and estimated dust lead levels shown in Figures 6-23a-h. Progressive remediation schemes are evaluated by reducing all yard soil concentrations greater than the suggested action level to 100 mg/kg lead and recalculating the community mean soil lead and dust lead values. This was accomplished at potential action levels of 2000 mg/kg, 1500 mg/kg, 1000 mg/kg, 800 mg/kg, 600 mg/kg and 400 mg/kg.

Tables 6-61a-f and 6-62a-f summarize the results for the EPA Default and Box Model versions, respectively. Figures 6-24a-h show the results for both models for both community wide and individual risks for 9 through 84 month children to exceed 10 µg/dl and 15 µg/dl health criteria. For example, for the 1000 mg/kg action level in Figure 6-24h, the community wide probability to exceed 10 µg/dl is 3% for the EPA Default estimate and 0% for the Box Model in the Lower Basin. In Wallace (Figure 6-24c) the estimate is 21% for the EPA Default Model and 5% for the Box model. Estimated percentages of children to exceed 15 µg/dl are found in the inset to these figures. For the 1000 mg/kg action level for Wallace, 7% are expected to exceed 15 µg/dl for the EPA Default Model, and 1% are expected for the Box Model.

USEPA policy requires that the probability of the typical 9-84 month old child at any residence experiencing a blood lead level of 10 µg/dl or greater, be no more than 5% (USEPA 1994d, 1998f). Figures 6-24a-h also show (in parentheses) the individual risk for 9-84 month old children associated with candidate action levels. These estimates are developed using the action level for the yard soil lead concentration and the estimated community mean dust concentration. As a result, there may be some underestimation of blood lead due to the dust lead level. The individual probability of exceeding 10 µg/dl for the 1000 mg/kg action level for the Lower Basin is 38% by the EPA Default Model, and 7% by the Box Model (Figure 6-24h). For Wallace, corresponding values are 46% and 12%, respectively (Figure 6-24e). For the 1000 mg/kg action level example above the most exposed children in the

Lower Basin would have a 14% probability of exceeding 15 µg/dl in the Lower Basin according to the EPA Default Model and a 1% chance by the Box Model. Corresponding probabilities for Wallace are 19% and 2%.

There are two major considerations in assessing these results. First, the risk of exceeding the health criteria projected in this analysis only accounts for baseline (or home residential) exposures after paint stabilization. Consequently, incremental exposures that might occur in addition to home exposure are not addressed by this analysis. Second, USEPA policy and the RAOs applied at the BHSS require consideration of individual risks for those children left at the highest exposure levels.

The disparity in risk allocation across a post-remedial community results from the nature of a yard soil cleanup implemented on a yard-by-yard basis. Remediated yards have levels near background (40 mg/kg to 100 mg/kg lead), while other children are exposed to concentrations 4 to 50 times greater depending on the action level. At the BHSS, this disparity was addressed through the 15 µg/dl RAO. The 1000 mg/kg action level at the BHSS was addressed in the RAO requiring that less than 1% of children exceed the 15 µg/dl criteria. The cleanup action level necessary to meet the site wide goal of having less than 5% of the children exceeding 10 µg/dl at the BHSS was approximately 1500 mg/kg. However, this level was rejected in favor of the more protective 15 µg/dl RAO. Maximum post-remedial individual risks at the BHSS are estimated at between 10% and 15% for the 10 µg/dl health criteria and from 1% to 2% for the 15 µg/dl RAO. Comparable risk levels could be achieved in the Basin with a 400-600 mg/kg action level by the EPA Default model or an 800-1000 mg/kg action level by the Box Model. The acceptable level of risk to individuals will be a critical determinant in developing risk management alternatives for the Basin.

Individual risks for those children in homes with yard soil lead concentrations near the action level are shown in Tables 6-61a-f through 6-62a-f, for each of the candidate action levels. For example, for the 1000 mg/kg action level in Table 6-61c, the columns headed *Homes 800-1000 mg/kg* show the risk of exceeding 10 µg/dl ranges from 36% to 46% for children living in these homes according to the EPA Default Model. The corresponding value for the Box Model in Table 6-62c is 7% to 12%.

These tables also indicate the number and percent of the homes sampled in each subarea, the number and percent of those homes that would require remediation under the 1000 mg/kg action level, and how many homes are in the 800 mg/kg to 1000 mg/kg range in each geographic subarea. For example, 42 (or 38%) homes in Wallace were above the 1000 mg/kg action level and eighteen (or 16%) additional homes are in the 800 mg/kg to 1000 mg/kg range.

The subsequent remediation Table 6-61d shows the outcome if those homes in the 800 mg/kg to 1000 mg/kg range were remediated. Overall community wide risk would remain about the same in Kingston and the Lower Basin, but would drop from 21% to 14% in Wallace, with intermediate drops in other areas. Those at highest risk are now in the 600 mg/kg to 800 mg/kg range and have about a 10% lower probability of experiencing a high blood lead level, than those in the previous table.

For the EPA Default Model in the batch mode, 5% probabilities are not achieved even at the 400 mg/kg cleanup level. At an action level of 400 mg/kg, probabilities of exceeding 10 µg/dl remain at 6% for several areas.

Results for the Box Model shown in Tables 6-62a-f are much different. Less than 5% community wide probabilities of exceeding 10 µg/dl are indicated at the 2000 µg/dl action level in Kingston and the Lower Basin, and at the 1500 mg/kg level in Silverton, the Side Gulches, Osburn, and Mullan. All areas project less than 5% of children greater than or equal to 10 µg/dl at the 1000 mg/kg action level.

For the 1000 mg/kg Box Model action level scenario (Table 6-62c), risks to children in the 800 mg/kg to 1000 mg/kg yard soil lead concentration range are from 7% to 12%. The probability of exceeding 15 µg/dl ranges from 1% to 2% for these children. These risk probabilities are similar to the RAOs for the BHSS. Under this cleanup scenario approximately 33% to 43% of homes in areas east of and including Wallace, and 8% to 13% of homes in the remainder of the Basin would require remediation.

For the 800 mg/kg Box Model action level (Table 6-62d), from 40% to 55% of homes east of and including Wallace and 13% to 17% of homes in the remainder of the Basin would require remediation. The probability of exceeding 10 µg/dl for the highest individuals drops to 5% to 7% with less than 1% projected to exceed 15 µg/dl. Figures 6-24a-h summarize these results. The values in parentheses show the average individual probability of exceeding 10 µg/dl for children living in homes with the highest post-remedial soil concentrations. The inset table shows the community wide and individual probability of exceeding the 15 µg/dl health criteria.

Yard Soil Risk Reduction Conclusions

Based on residential exposures, achieving a remedial action goal of no more than 5% of children in a community having blood lead levels of 10 µg/dl or greater requires a cleanup level of 400 mg/kg to 1000 mg/kg based on Default and Box IEUBK Model runs, respectively. To achieve an acceptable risk, of no more than 5% probability that an individual child has a blood lead of 10 µg/dl, would require similar yard soil cleanup levels of 400 to 800 mg/kg. Consideration of incremental exposures would require lower levels of lead in soils. These analyses assume that paint lead stabilization has been achieved and that lead levels in house dust will decline as yard soils are remediated. No other risk reduction activities have been considered in these analyses.

Incremental Exposure Risk Reduction

Childhood Recreational. Substantial increases in blood lead levels are predicted for particular play activities in contaminated areas of the Basin. Blood lead increments to existing baseline conditions were developed for this report. For most activities, appropriate action levels will likely vary between the 25th and 95th percentile concentration levels for the specific media. However, determination of appropriate risk reduction action levels for soil and sediments must be made in conjunction with concurrent

decreases in baseline, or residential, risk levels. These analyses are not possible until appropriate baseline levels are available.

Childhood Consumption of Local Foodstuff. Similarly, the significance of local produce and fish from the lateral lakes area depends on the relative baseline blood lead level. In this case, a determination of allowable dietary intake based on baseline blood lead levels will be required. These can be compared to incremental fish and local produce intake tables relating intake to media contaminant levels.

Adult Occupational. Tables 6-57 and 6-58 show estimated blood lead levels associated with potential soil and dust concentration levels in occupational activities. These results suggest that in order to maintain 95% of reproductive aged women's blood lead levels below 10 µg/dl, protective measures should be taken for typical workers when in contact with soils exceeding 2800 mg/kg to 4500 mg/kg lead. For those workers engaged in heavy contact with soils for extended periods of time, the corresponding level of concern is 500 mg/kg lead. Consideration should also be given to secondary take-home exposures of workers' children for those workers engaged in heavy contact with contaminated soils.

Adult Recreational. Tables 6-59a-b and 6-60a-b show similar results for upland park, land-based, and CUA recreational activities. These results suggest protective measures should be employed for adults engaging in intense soil-related recreational practices with soils exceeding 3700 mg/kg.

Adult Consumption of Local Foodstuff. Some local vegetable garden produce shows high lead content that could substantially increase total intake to levels of concern among pregnant women. Adult consumption of local fish adds minimally to total intake at typical fish fillet lead concentrations. However, at maximum concentrations and consumption rates, the increased intake could be of concern, although it is unlikely that the species of fish providing the samples would be consumed in large amounts.

Native American Subsistence Activities. Native American subsistence practices in the Lower Coeur d'Alene River would be ill-advised. Soil and sediment ingestion rates associated with residence in the floodplain and food harvest practices are extremely high. Near background level concentrations would be required to achieve acceptable intake rates for soils and sediments. Additionally, two critical elements of the native diet, fish and water potatoes, contain unsafe levels of lead when aboriginal consumption rates are applied. Lead levels in these food sources may also likely need to be in equilibrium with background soil and water conditions to assure acceptable intake rates. However, appropriate background lead levels for biological media are unknown other than water potatoes in an adjacent drainage that show below detection levels for lead.

Figure 6-1a Arithmetic and Geometric Mean Blood Lead Concentrations by Geographic Area - 9 Month through 9 Year Old Children (1996 - 1999 Combined)

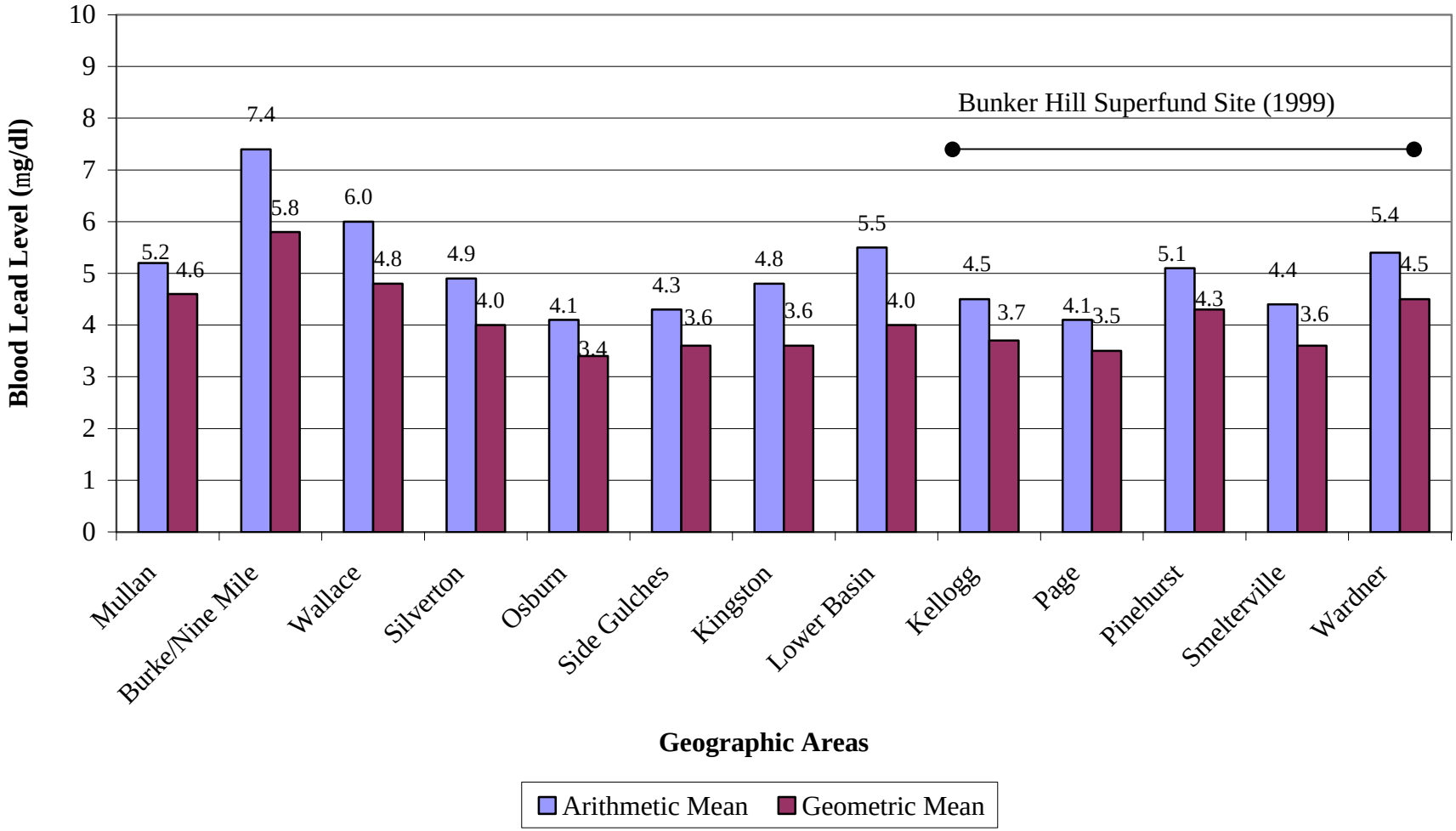


Figure 6-1b Percent to Exceed Blood Lead Concentrations by Geographic Area - 9 Month through 9 Year old Children (1996 - 1999 Combined)

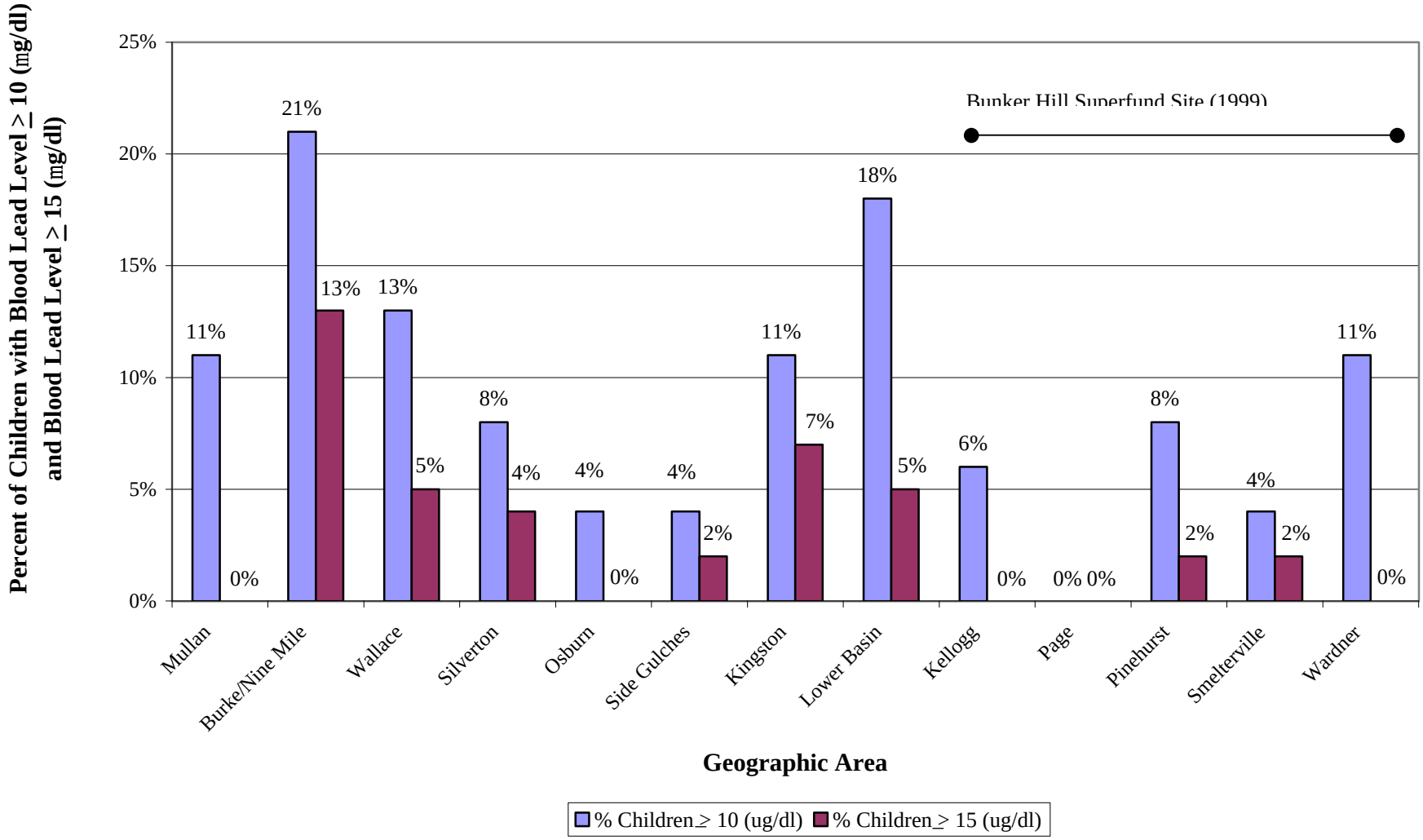


Figure 6-2 Basin Mean Blood Lead Levels by Age (1996 - 1999 combined)

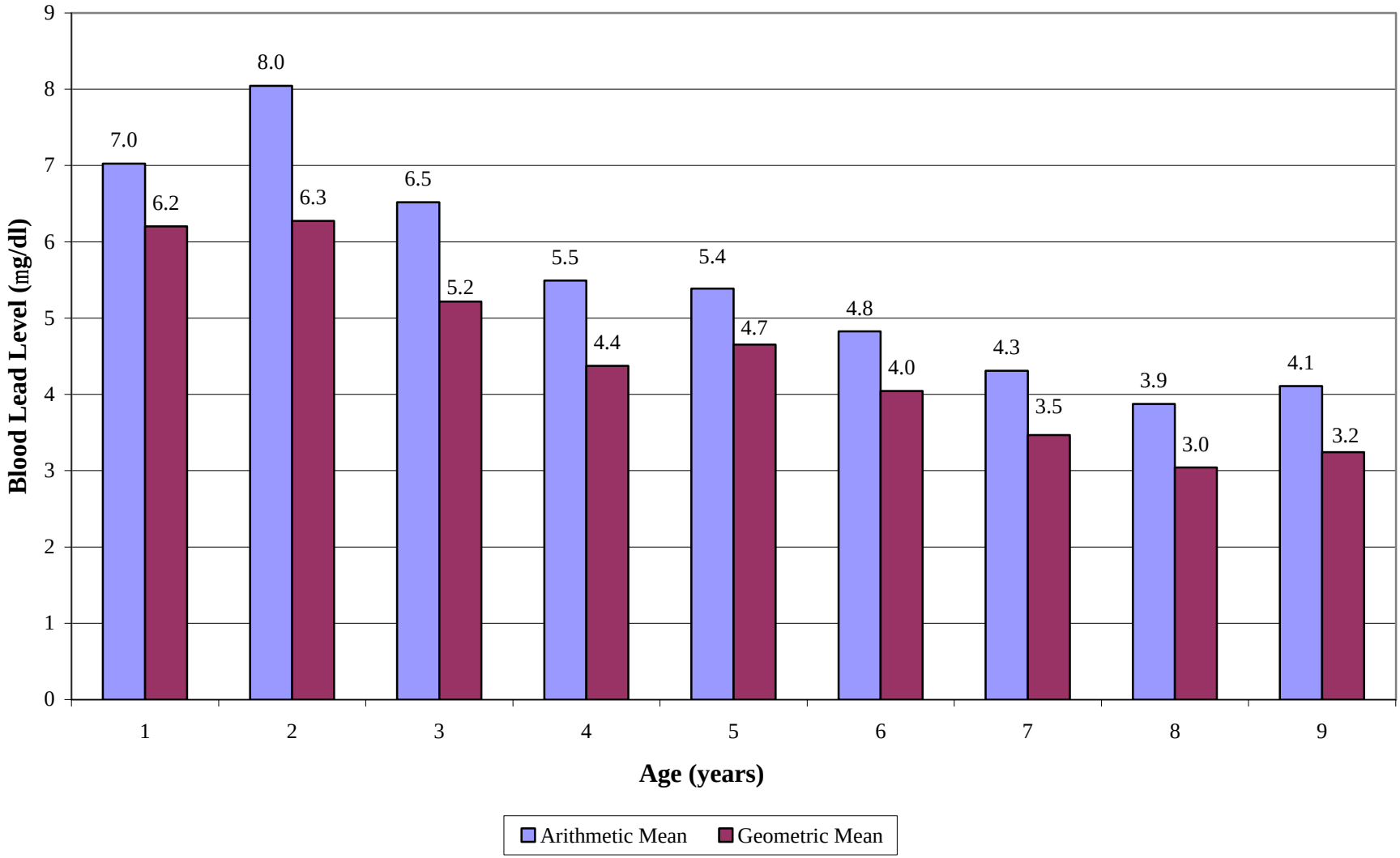


Figure 6-3 Percent of Children to Exceed Critical Toxicity Levels by Age (Basin-wide 1996 - 1999 combined)

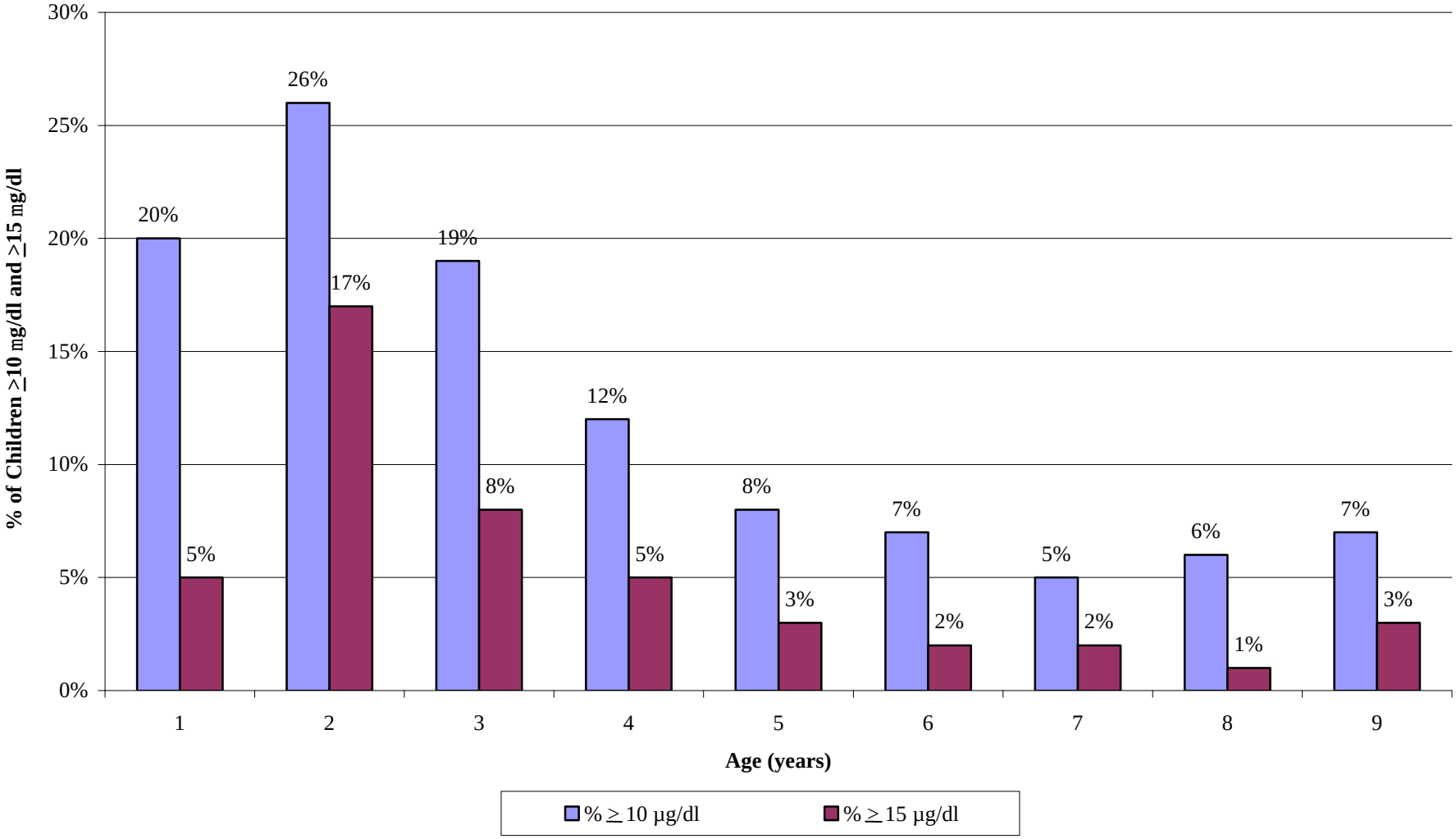


Figure 6-4 Geometric Mean Adult Blood Lead Levels by Age and Geographic Area

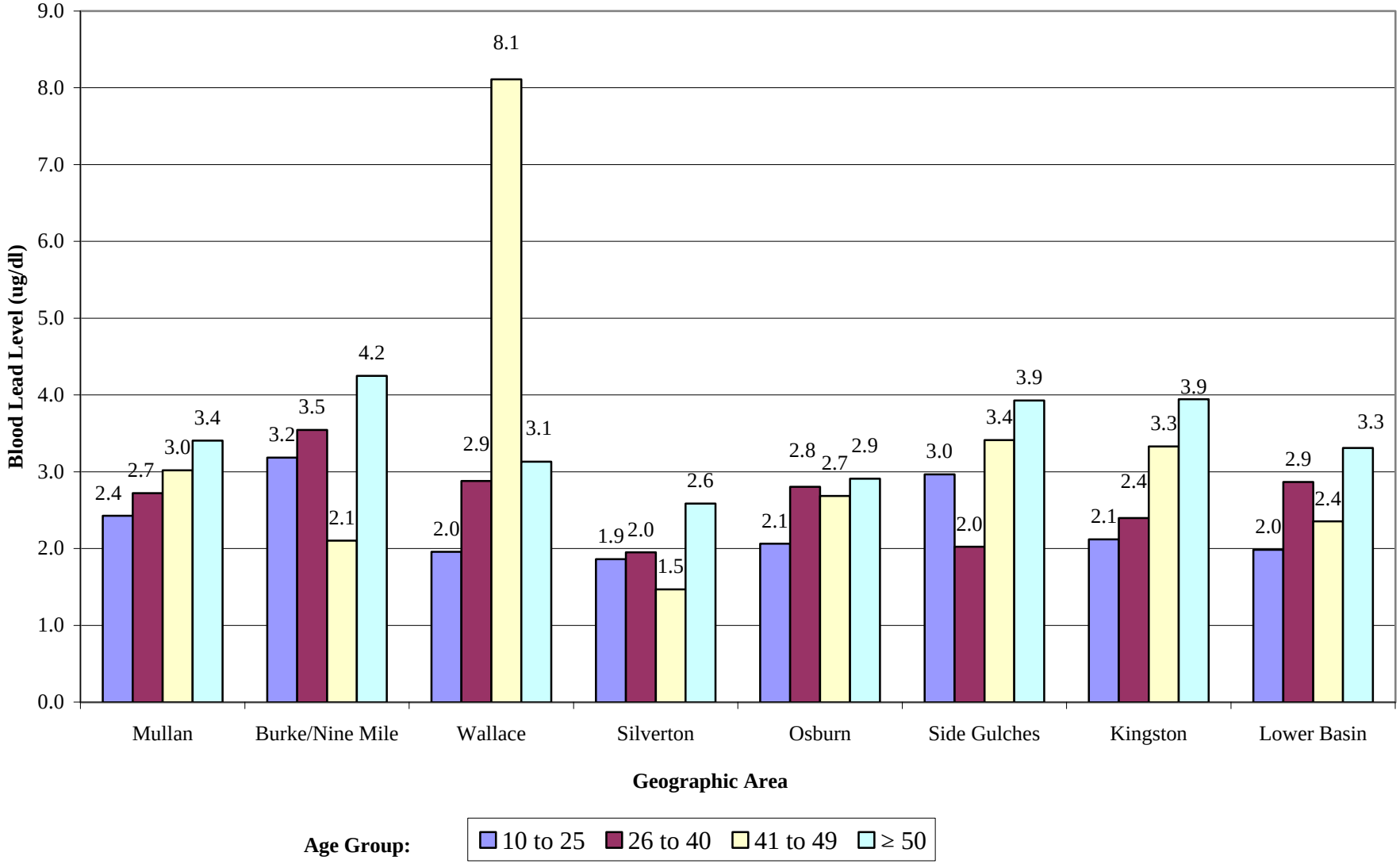


Figure 6-5 Geometric Mean and Maximum Blood Lead Levels for Reproductive Aged Females (17-45 Years Old) by Geographic Area

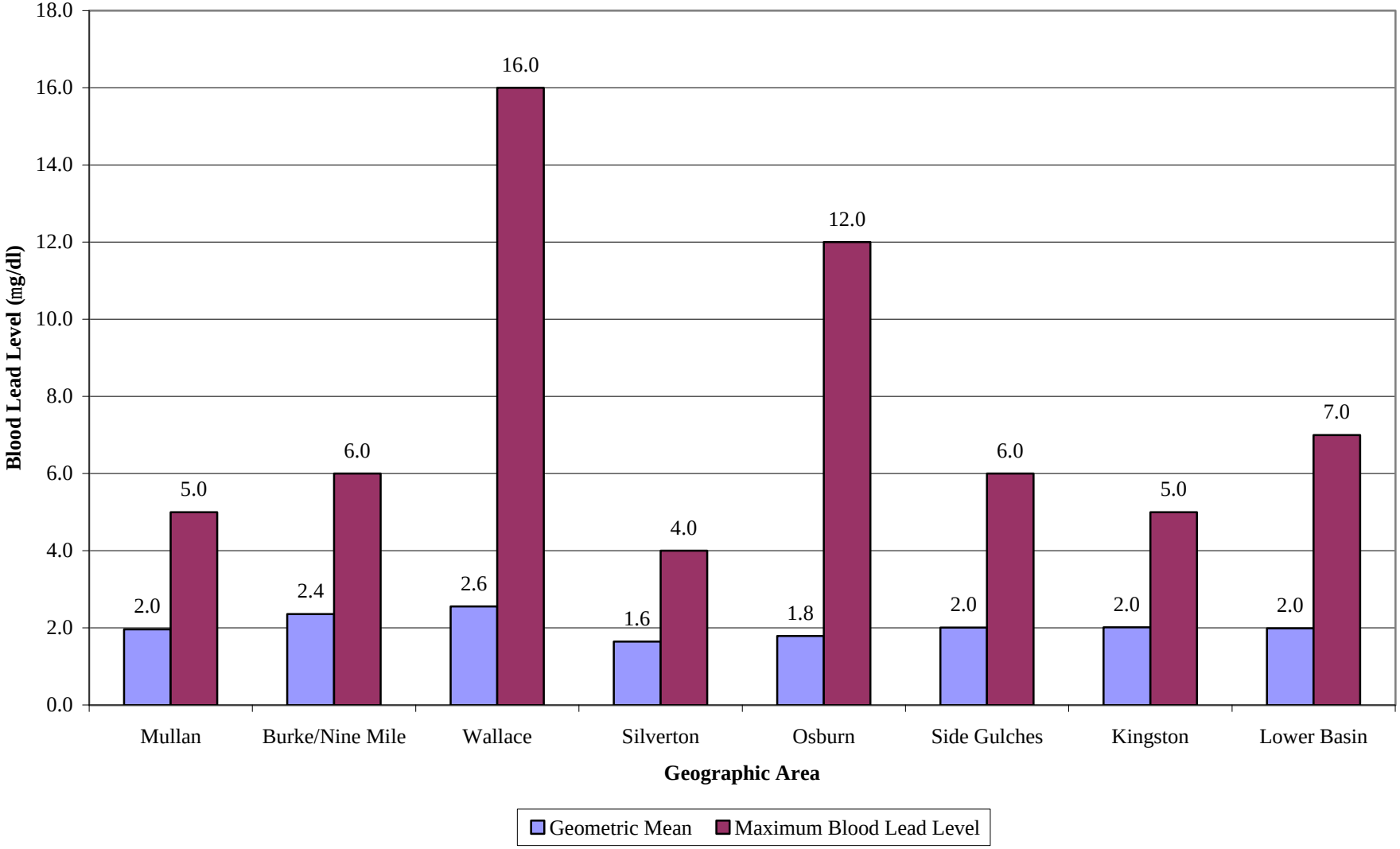


Figure 6-6

ENVIRONMENTAL PATHWAYS FOR LEAD EXPOSURE FOR THE BUNKER HILL SITE

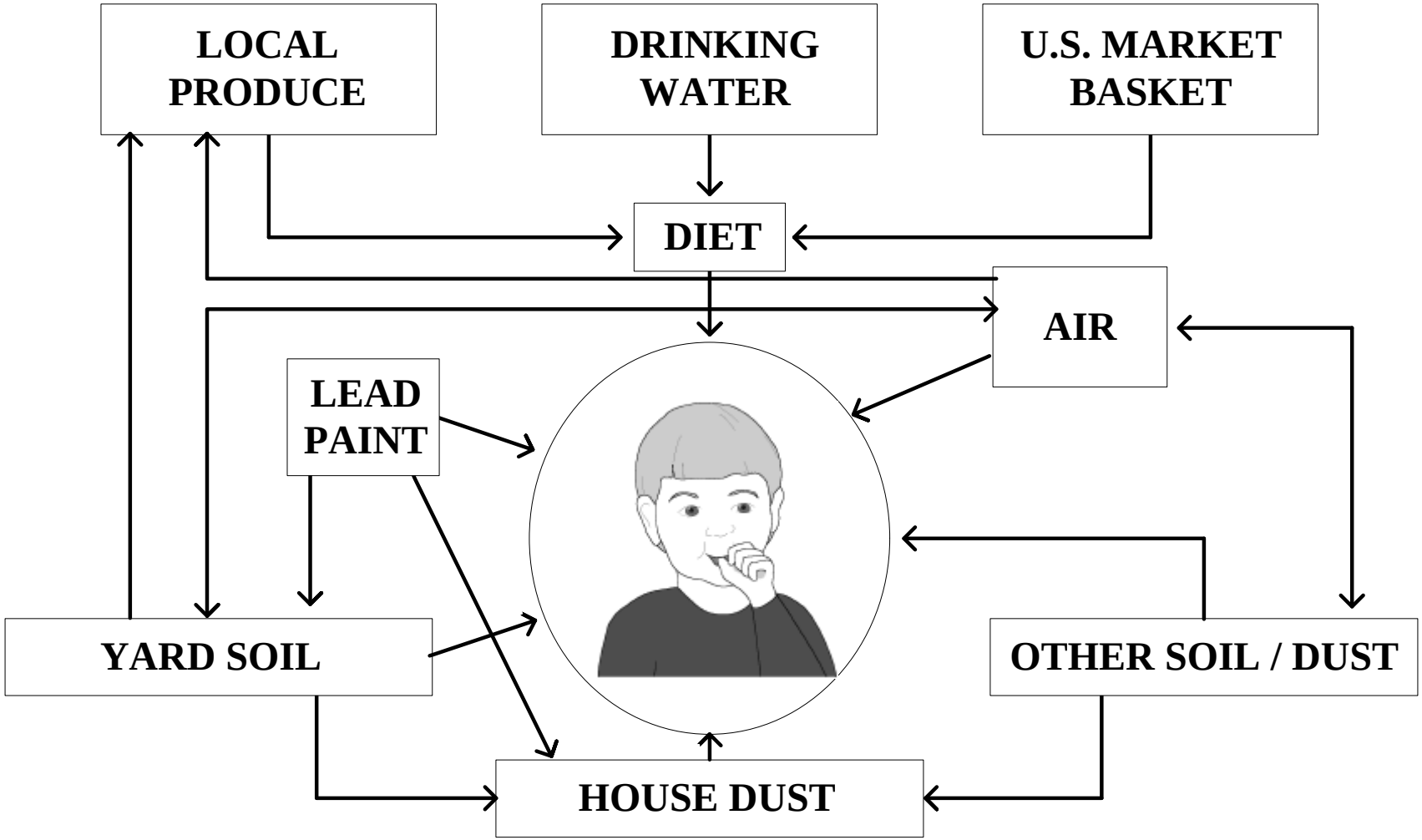


Figure 6-7a Geometric Mean Interior Paint Lead Loading by Geographic Area

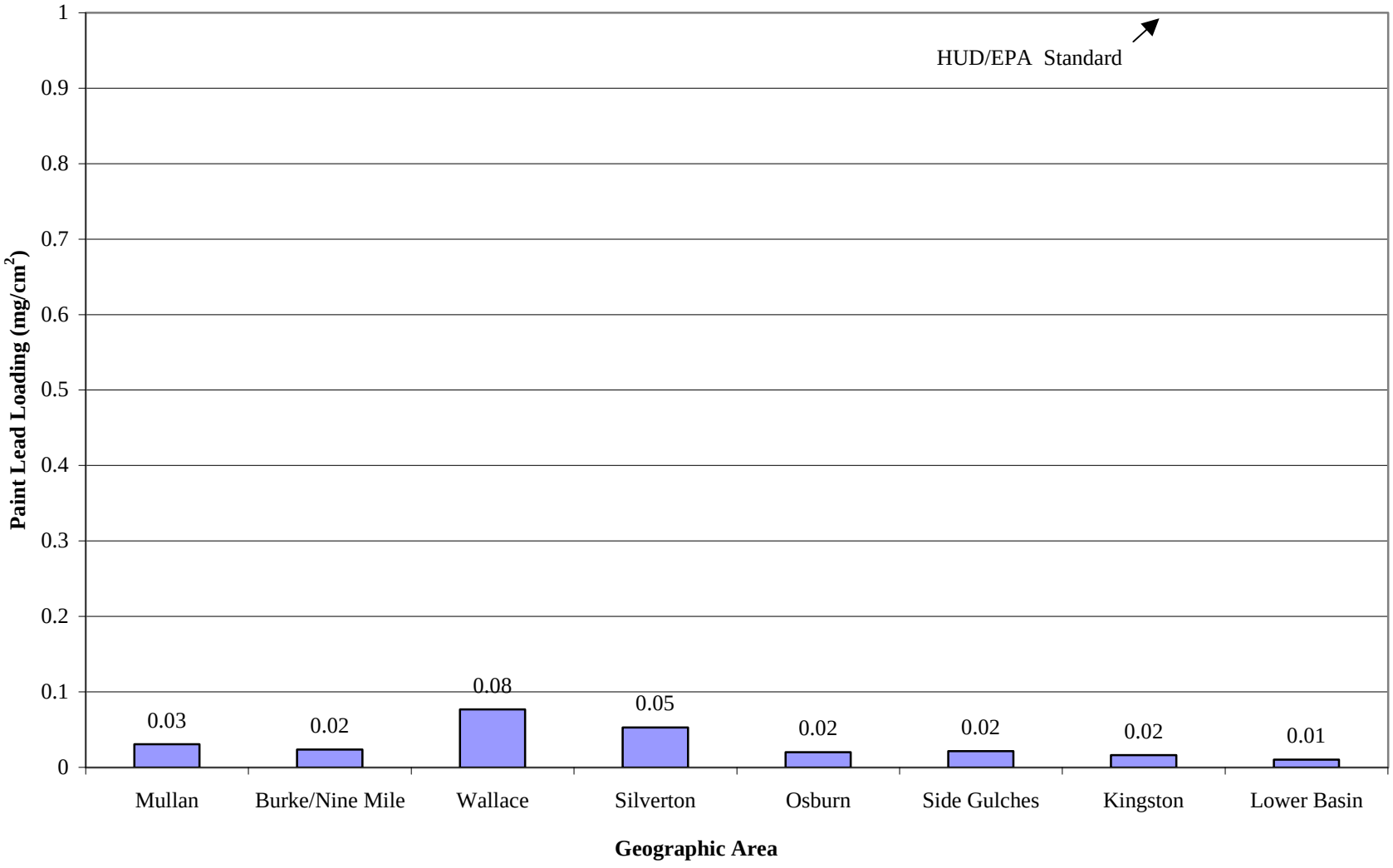


Figure 6-7b Geometric Mean Exterior Paint Lead Loading by Geographic Area

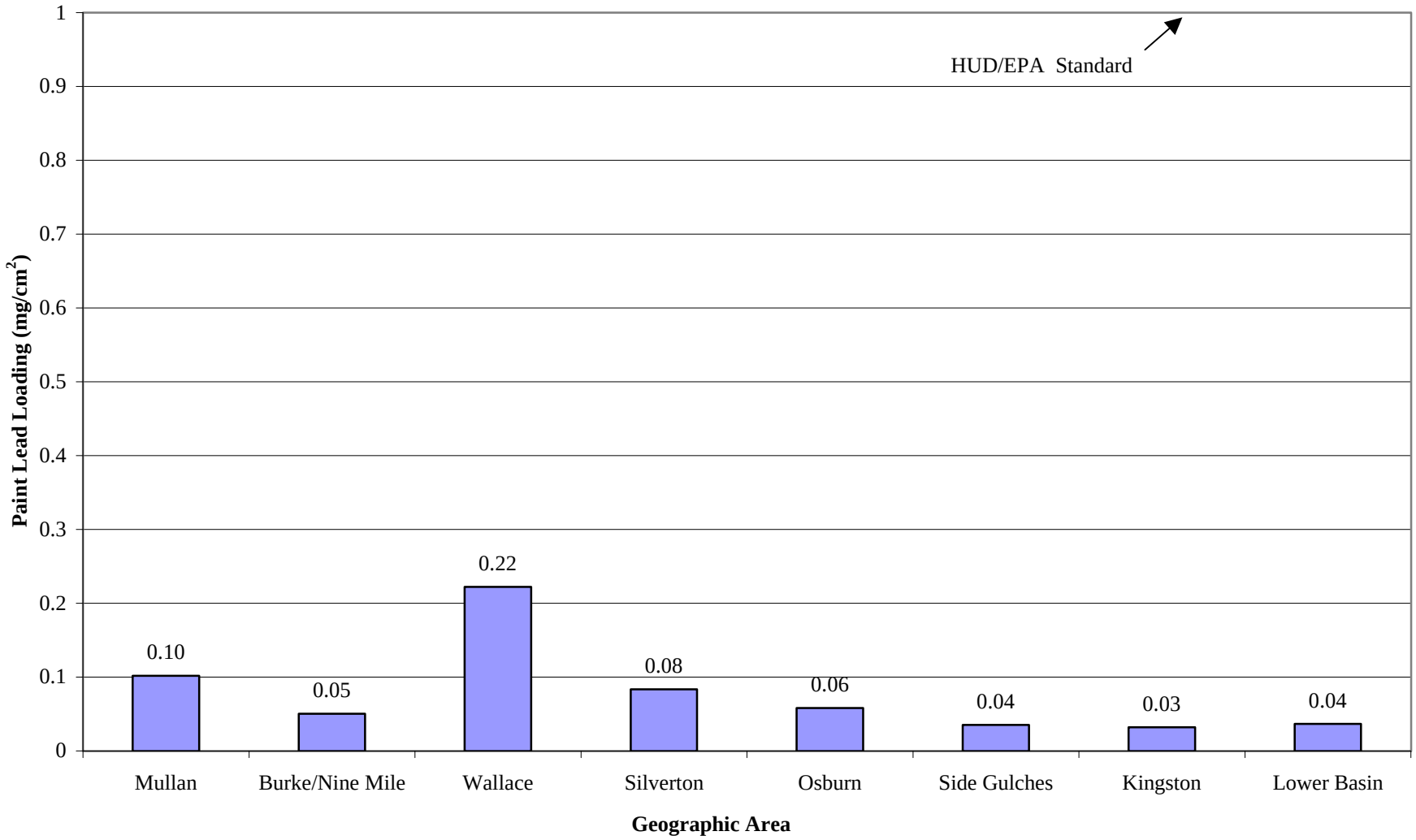


Figure 6-8a Arithmetic Mean Soil and House Dust Lead Concentrations by Geographic Area

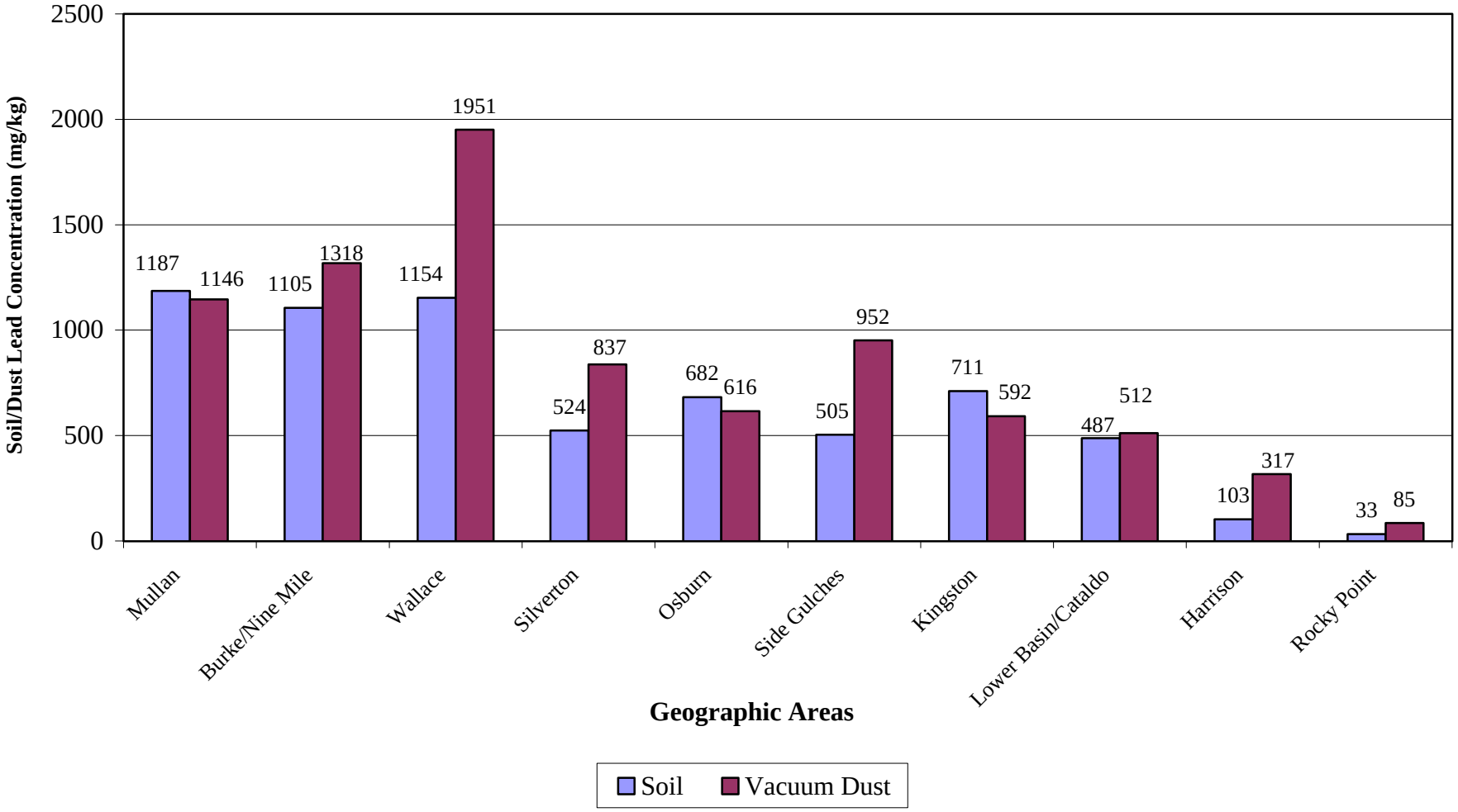


Figure 6-8b Arithmetic Mean Soil and House Dust Lead Concentrations by Geographic Area

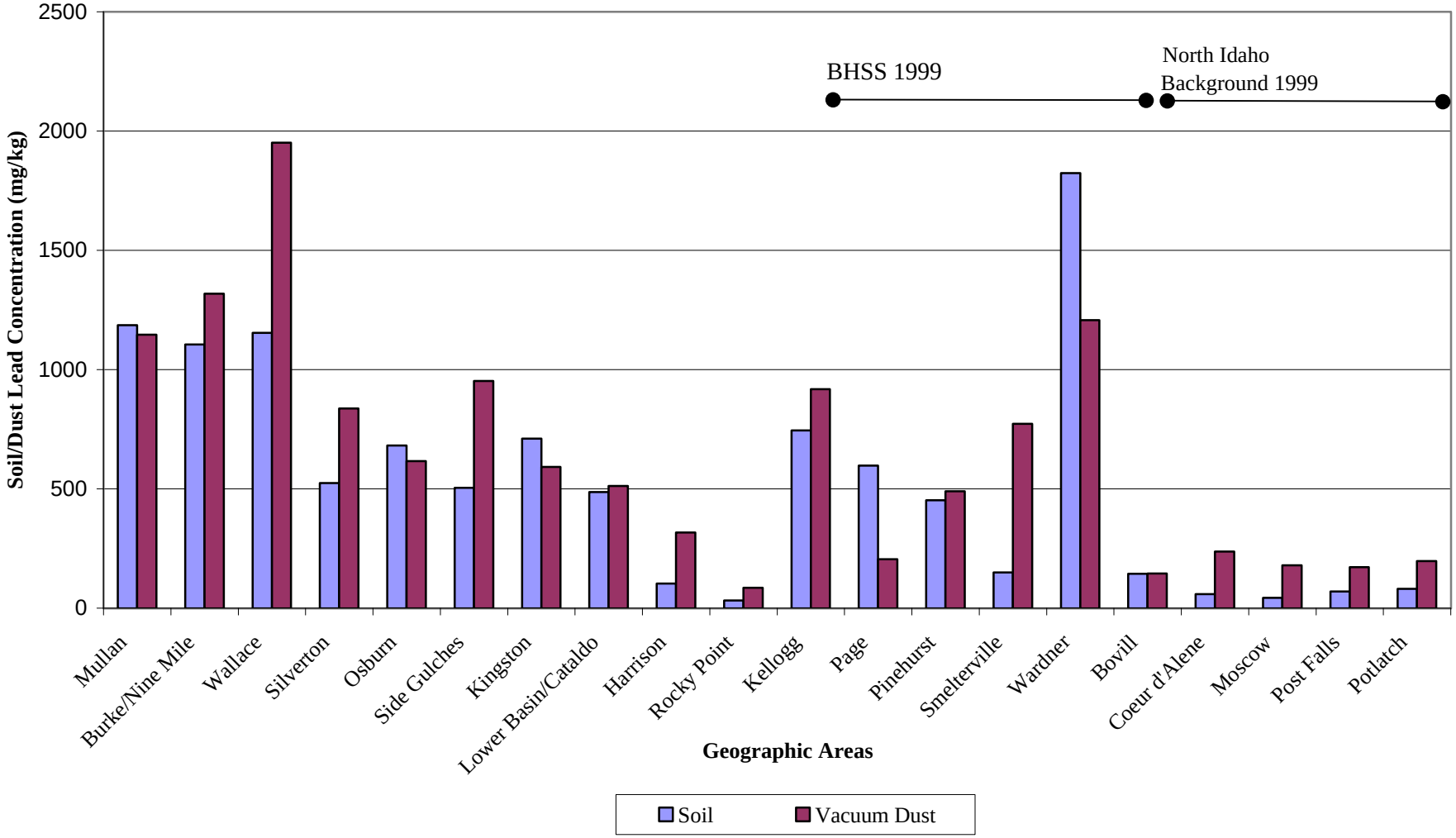


Figure 6-8c Geometric Mean Soil and House Dust Lead Concentrations by Geographic Area

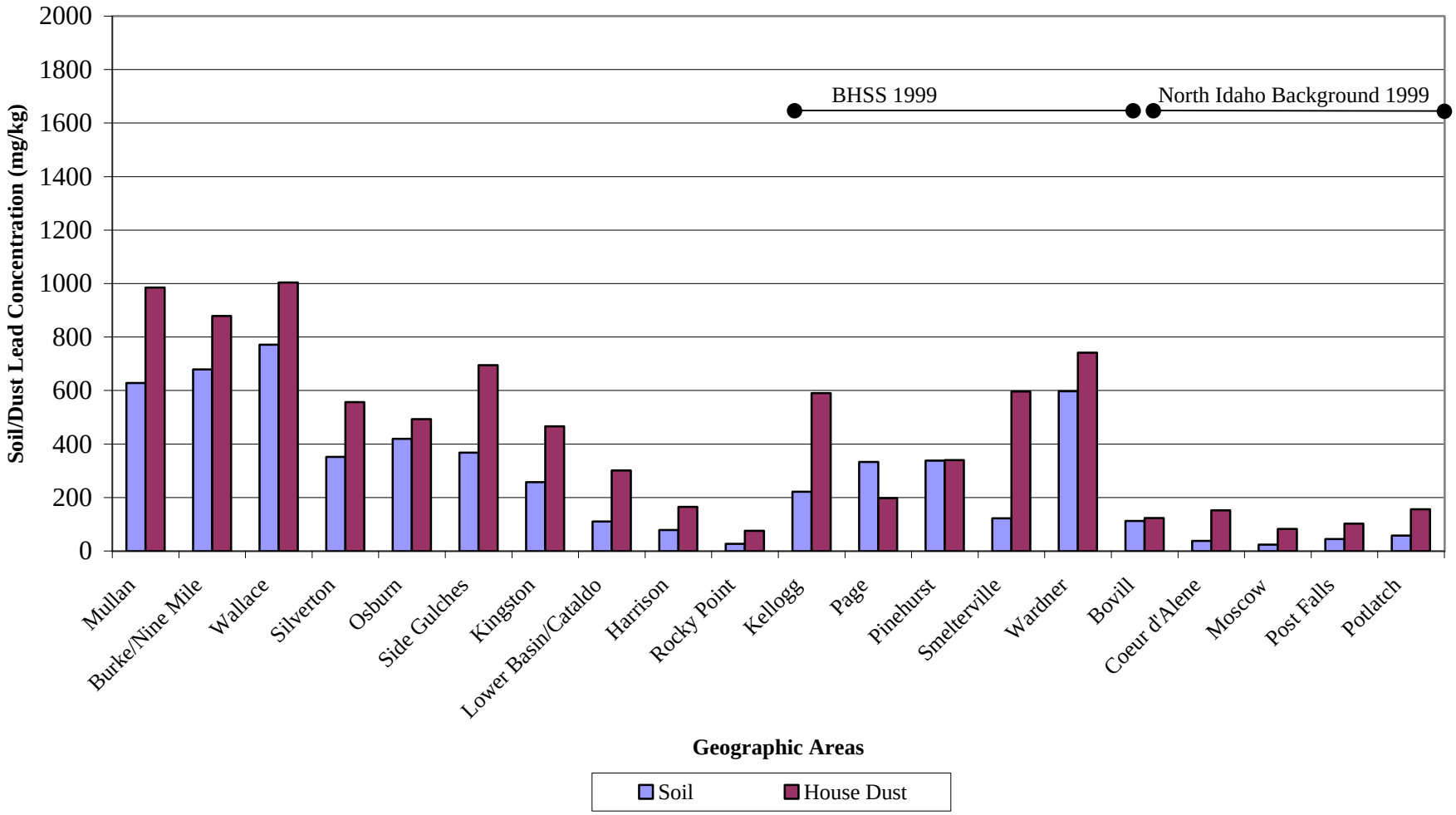


Figure 6-9a Geometric Mean Dust Mat Lead Concentration by Geographic Area

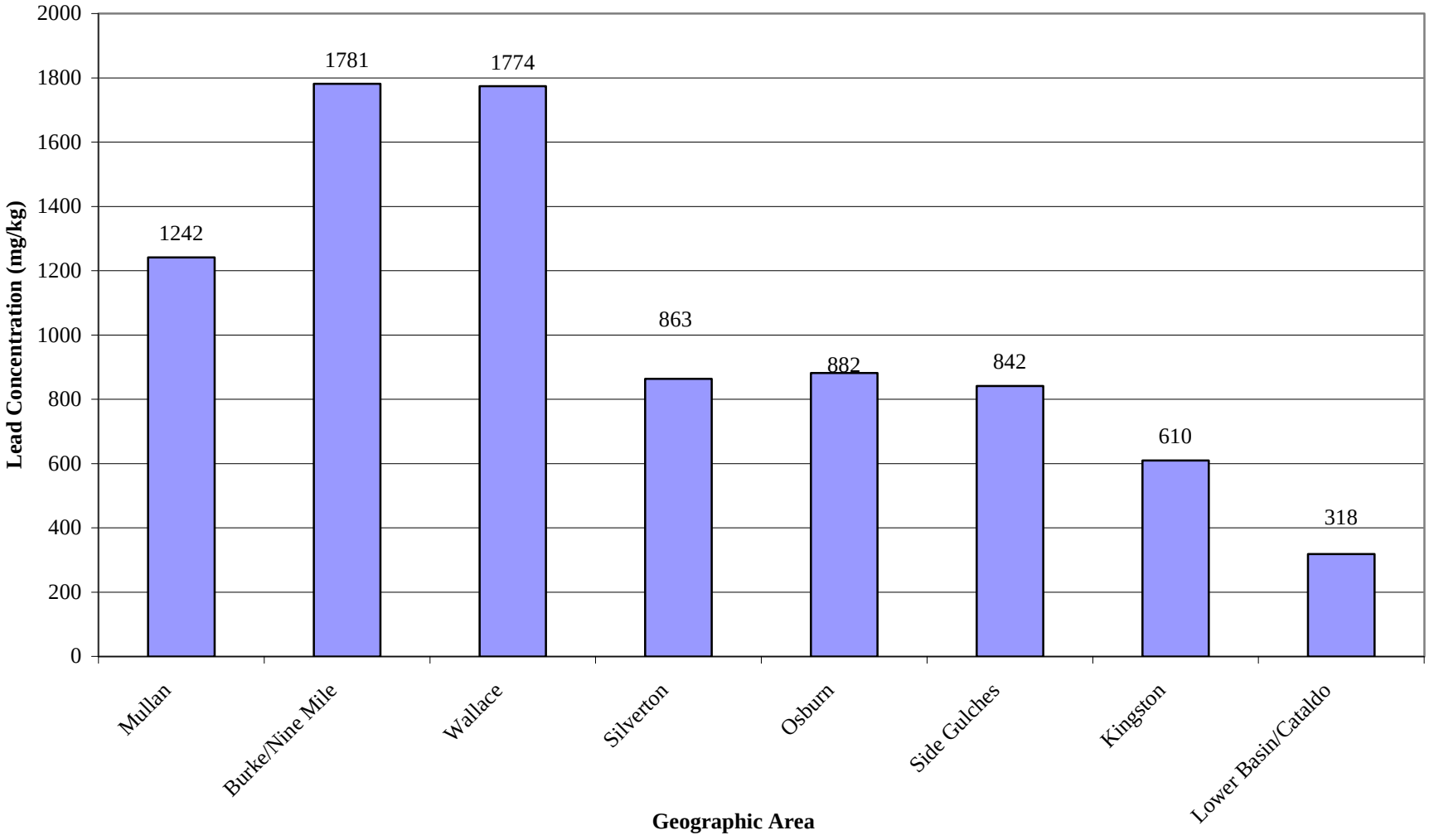


Figure 6-9b Geometric Mean Dust Loading Rate by Geographic Area

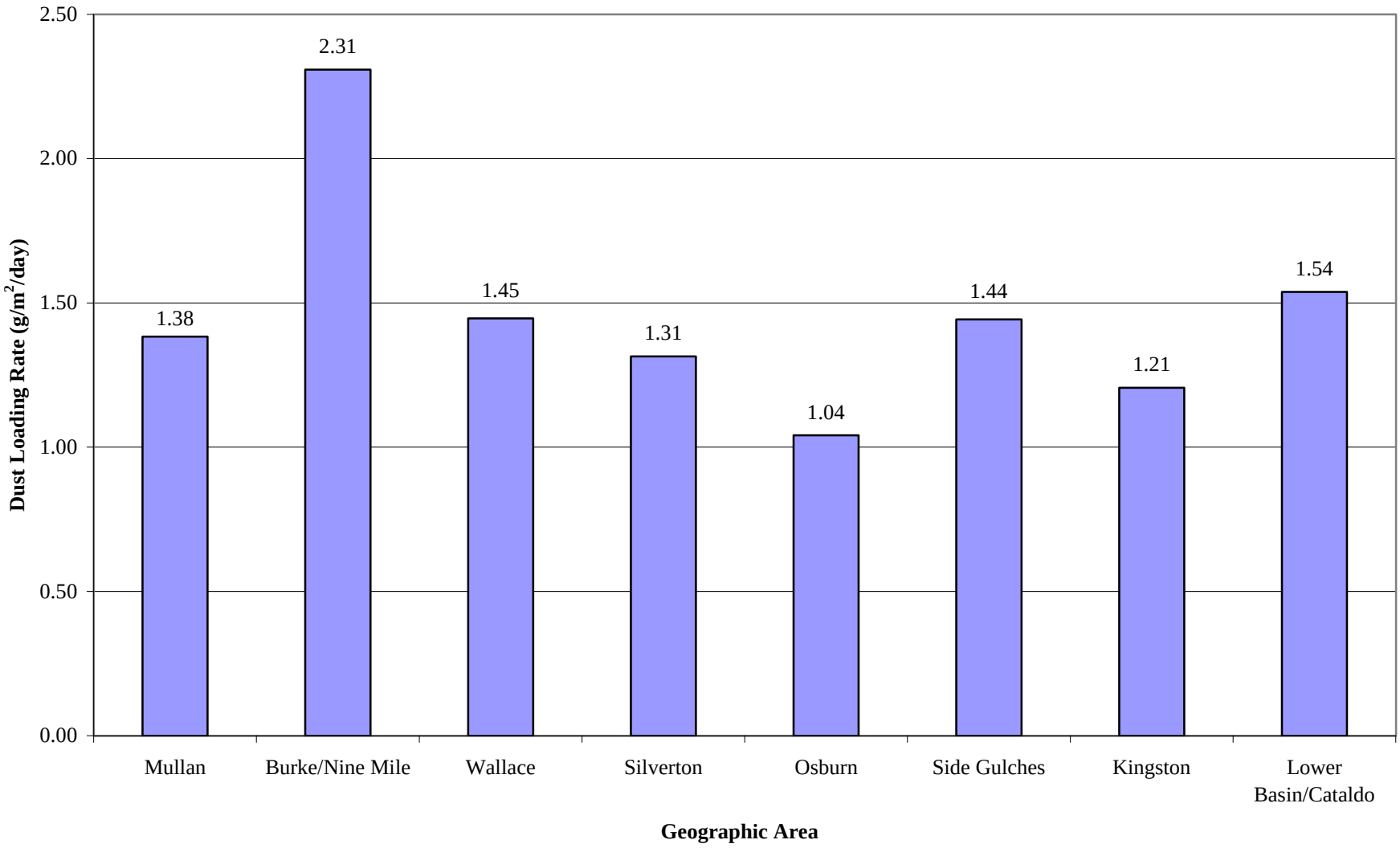


Figure 6-9c Geometric Mean Dust Mat Lead Loading Rate by Geographic Area

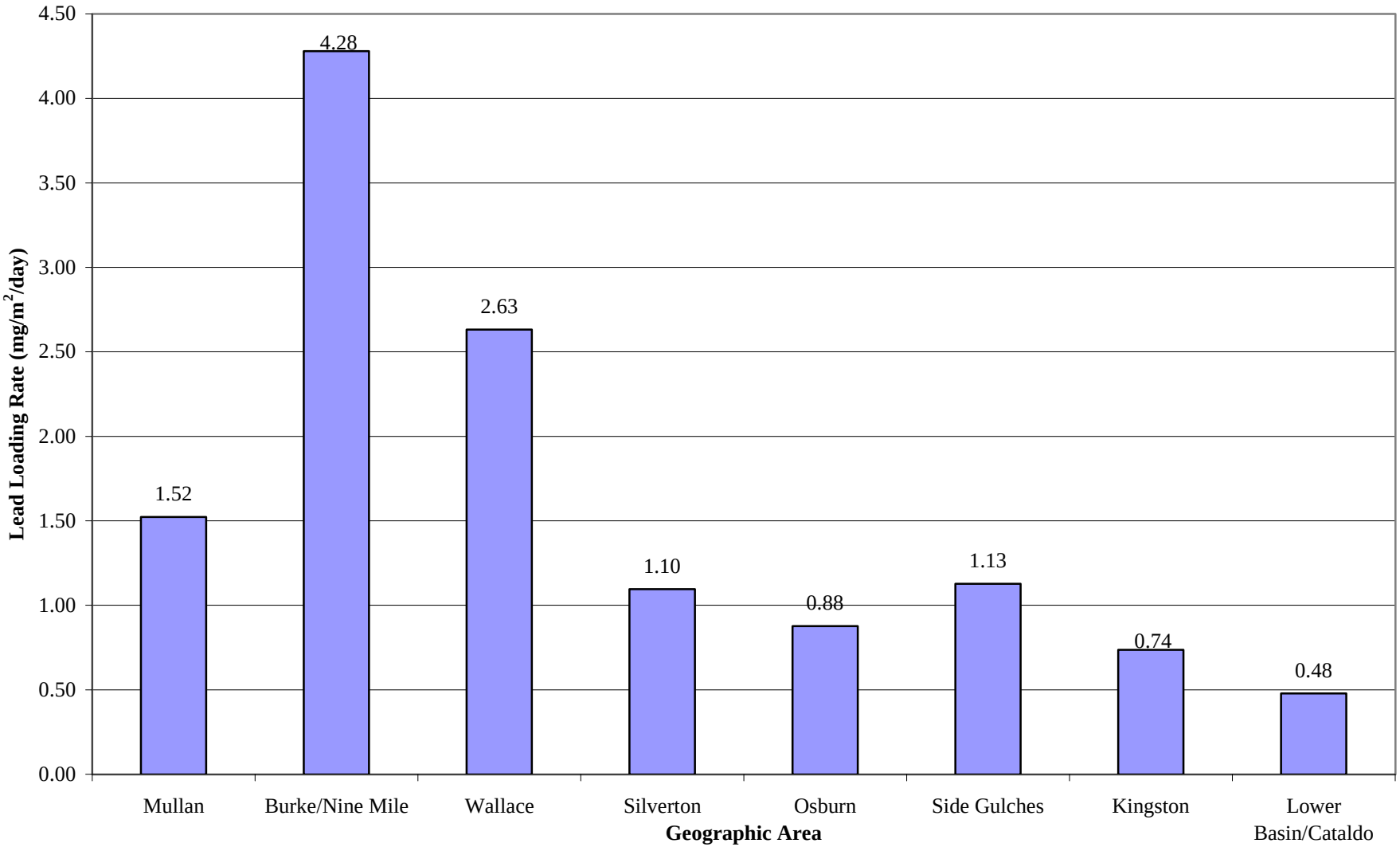
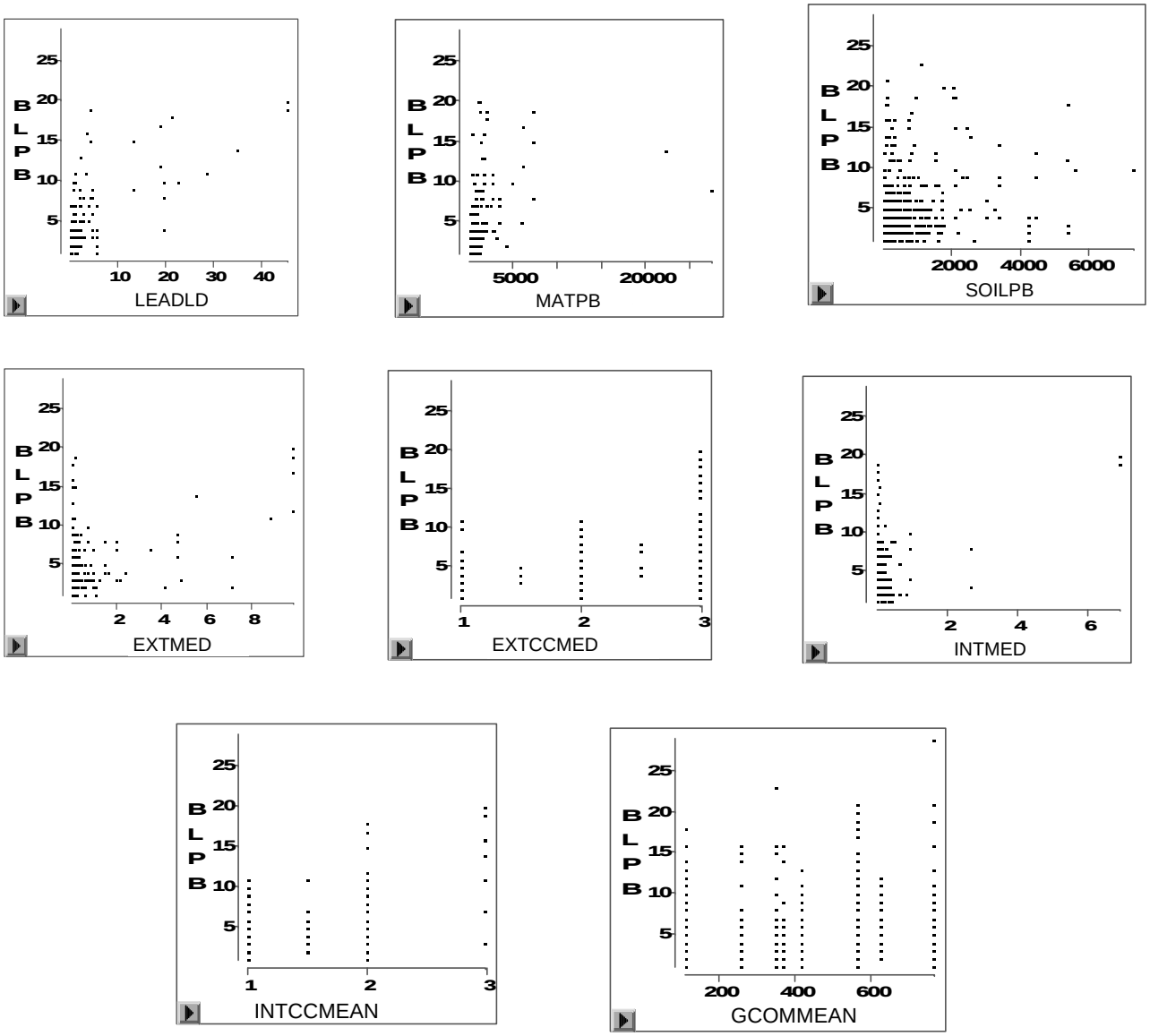
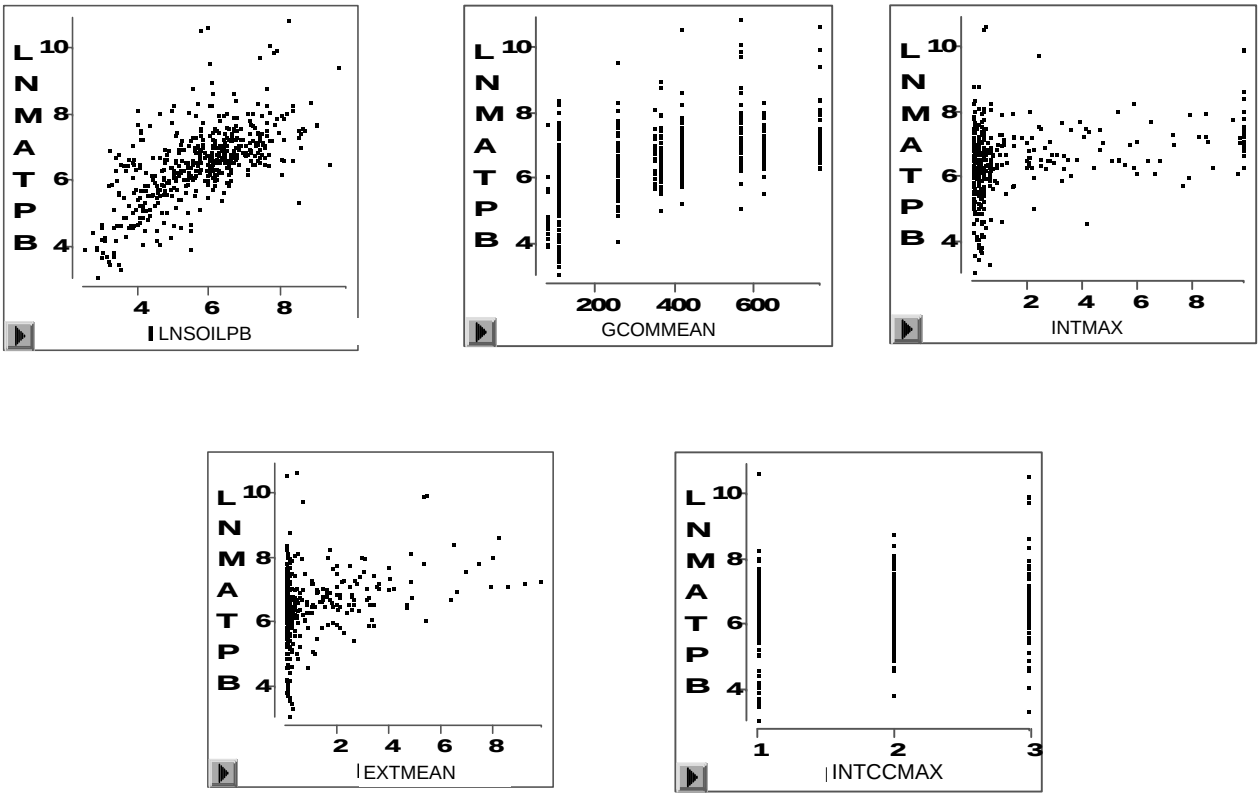


Figure 6-10a Scatterplots for Blood Lead Concentration and Environmental Source Variables



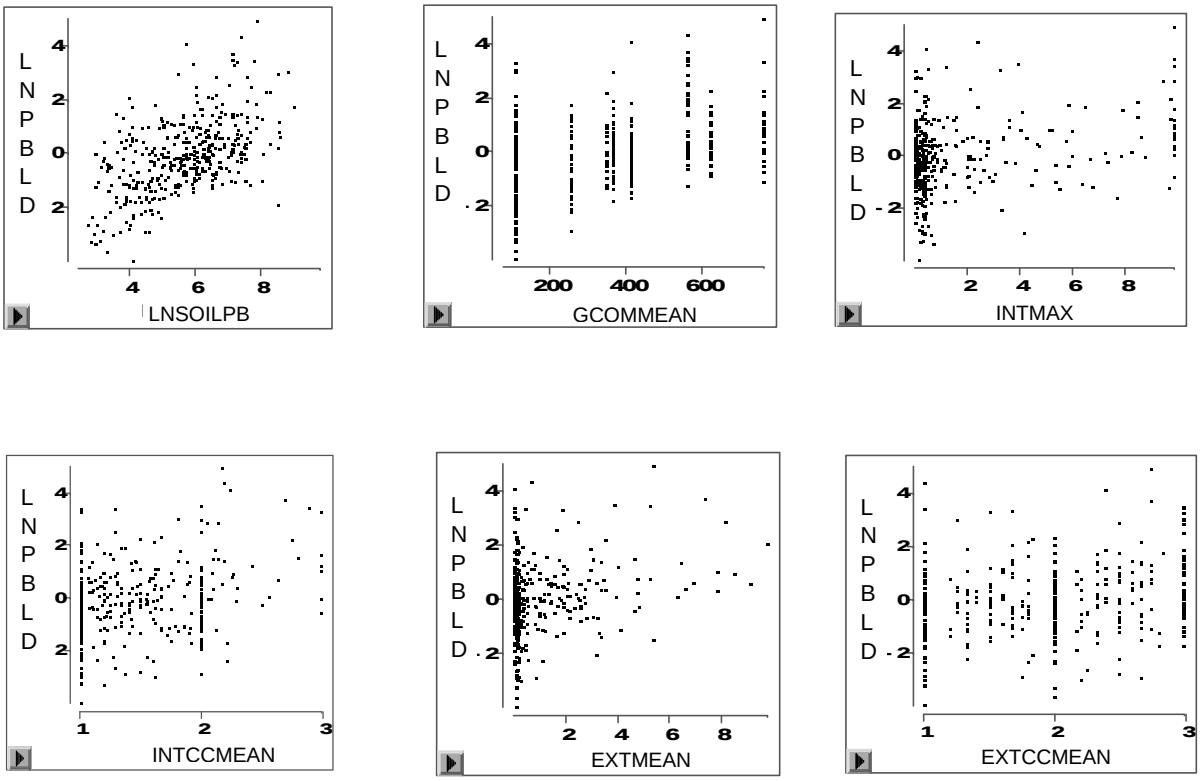
BLPB	Blood lead levels (ug/dl)
MATPB	Mat dust lead concentration (mg/kg)
LEADLD	Lead loading rate (mg/m^2/day)
SOILPB	Yard soil lead concentration (mg/kg)
INTMED	Median interior paint lead loading (mg/cm^2)
INTCCMED	Median interior paint condition (unitless)
EXTMED	Median exterior paint lead loading (mg/cm^2)
EXTCCMED	Median exterior paint condition (unitless)

Figure 6-10b Scatterplots for Dust Lead and Environmental Source Variables



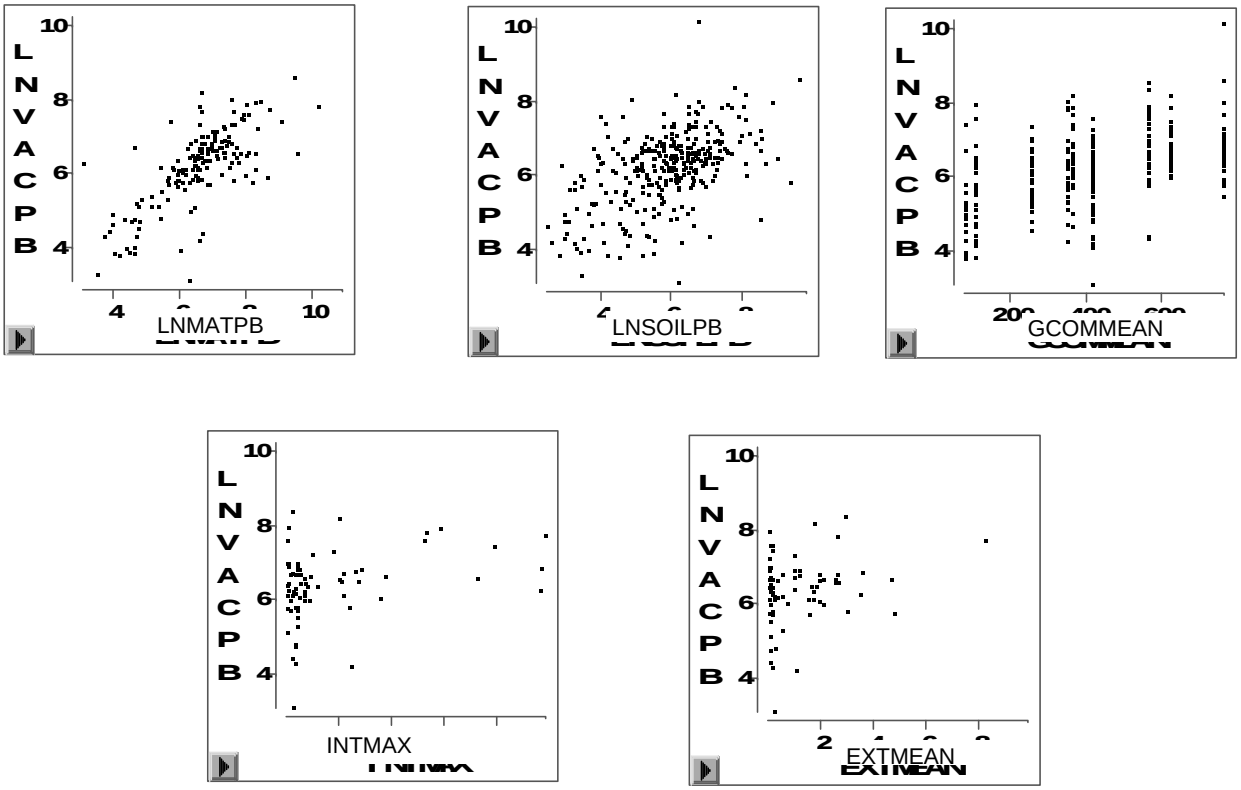
LNMATPB	Ln Mat dust lead concentration (mg/kg)
LNSOILPB	Ln Yard soil lead concentration (mg/kg)
GCOMMEAN	Community geometric mean soil lead concentration (mg/kg)
INTMAX	Maximum interior paint lead loading (mg/cm^2)
INTCCMAX	Maximum interior paint condition (unitless)
EXTMEAN	Arithmetic mean exterior paint lead loading (mg/cm^2)
GCOMMEAN	Community geometric mean soil lead concentration (mg/kg)

Figure 6-10c Scatterplots for Dust Lead Loading and Environmental Source Variables



LNPBLD	Ln Lead loading rate (mg/m^2/day)
LNSOILPB	Ln Yard soil lead concentration (mg/kg)
GCOMMEAN	Community geometric mean soil lead concentration (mg/kg)
INTMAX	Maximum interior paint lead loading (mg/kg)
INCCMEAN	Arithmetic mean interior paint condition (unitless)
EXTMEAN	Arithmetic mean exterior paint lead loading (mg/kg)
EXCCMEAN	Arithmetic mean exterior paint condition (unitless)

Figure 6-10d Scatterplots for Vacuum Dust Concentration and Environmental Variables



LNVACPB	Ln Vacuum cleaner dust lead concentration (mg/kg)
LNMATPB	Ln Mat dust lead concentration (mg/kg)
LNSOILPB	Ln Yard soil lead concentration (mg/kg)
GCOMMEAN	Community geometric mean soil lead concentration
INTMAX	Maximum interior paint lead loading (mg/cm^2)
EXTMEAN	Arithmetic mean exterior paint lead loading (mg/cm^2)

Figure 6-11
Percentage of Blood Lead Observations ≥ 10 ug/dl Associated With an Interior Lead Paint Hazard

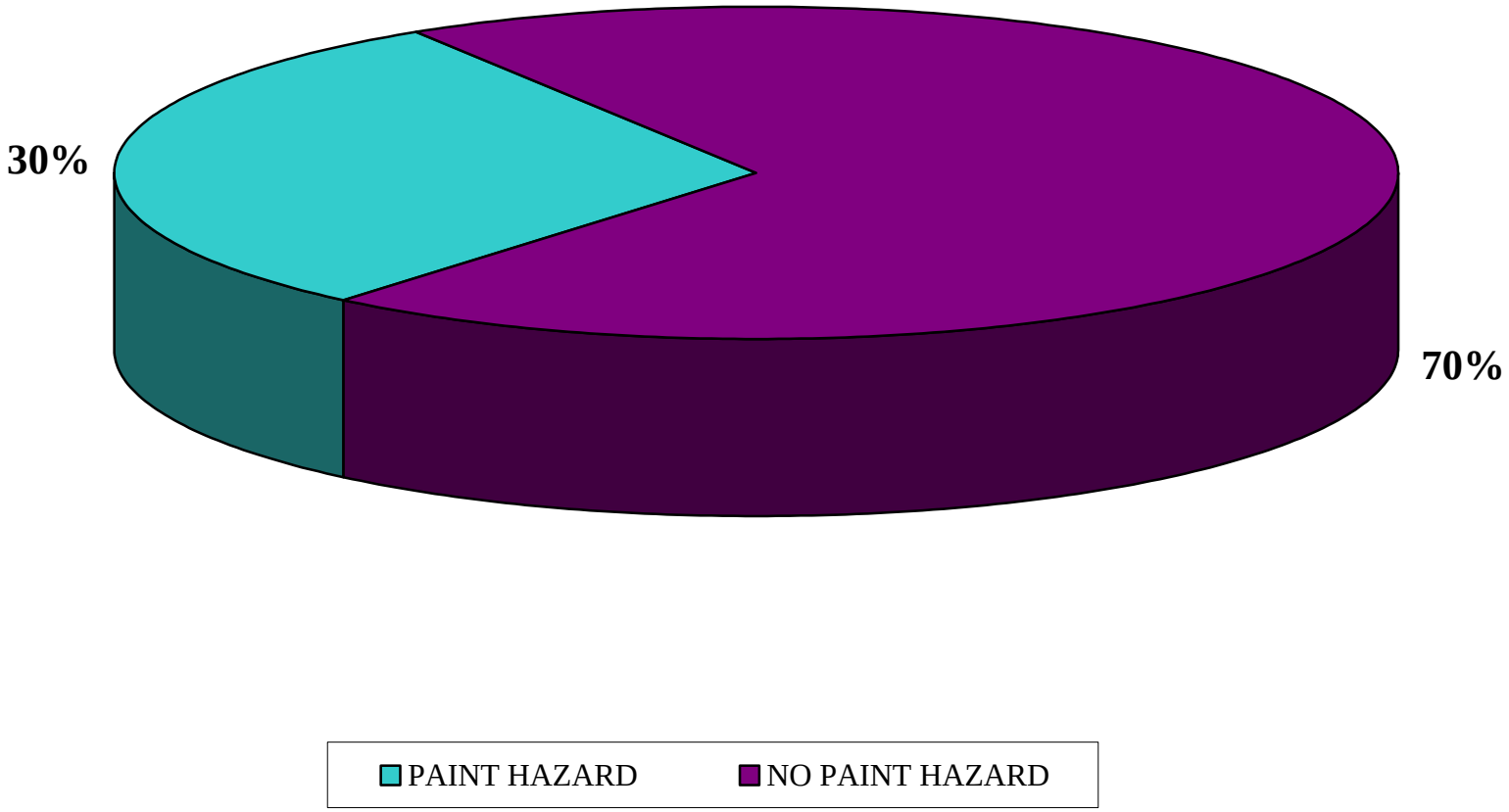


Figure 6-12a Comparison of Geometric Mean Environmental Lead Levels Between High and Low Blood Lead Levels in Children (1-9 yrs.) Exposed to an Interior Lead Paint Hazard

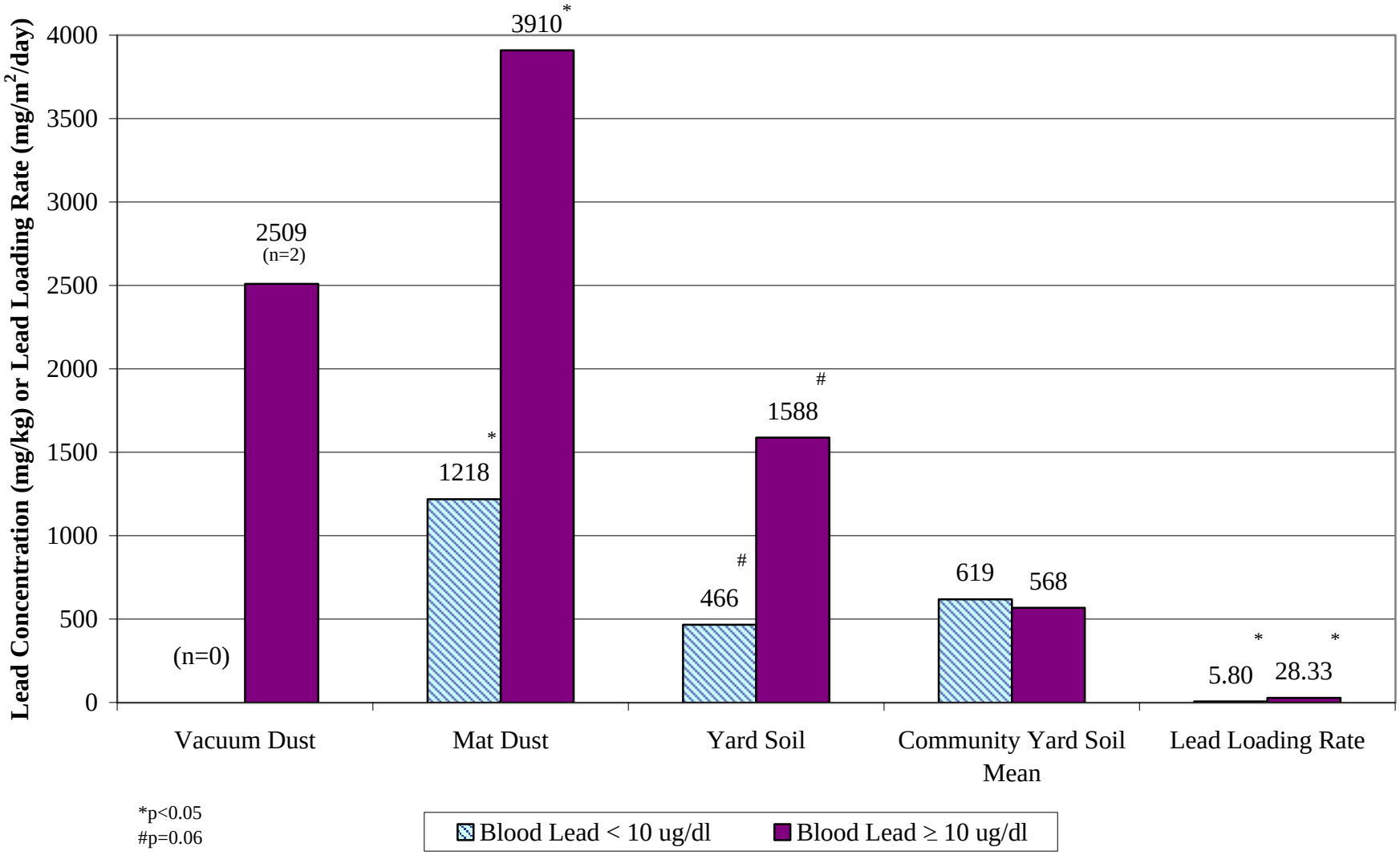


Figure 6-12b Comparison of Geometric Mean Environmental Lead Levels Between High and Low Blood Lead Levels in Children (1-9 yrs.) Not Exposed to an Interior Lead Paint Hazard

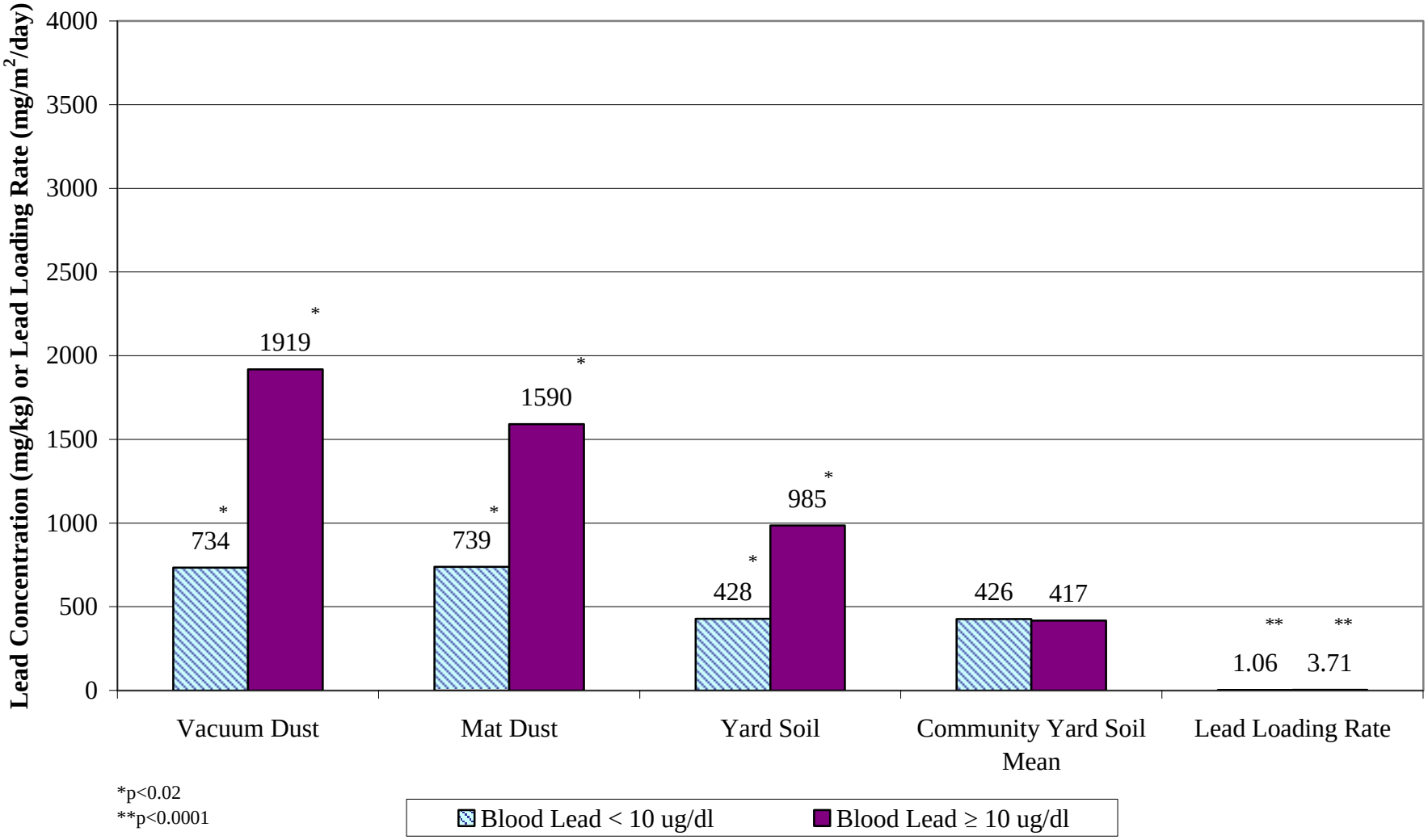


Figure 6-13 Geometric Mean Dust Mat and Vacuum Bag Dust Lead Concentration by Geographic Area

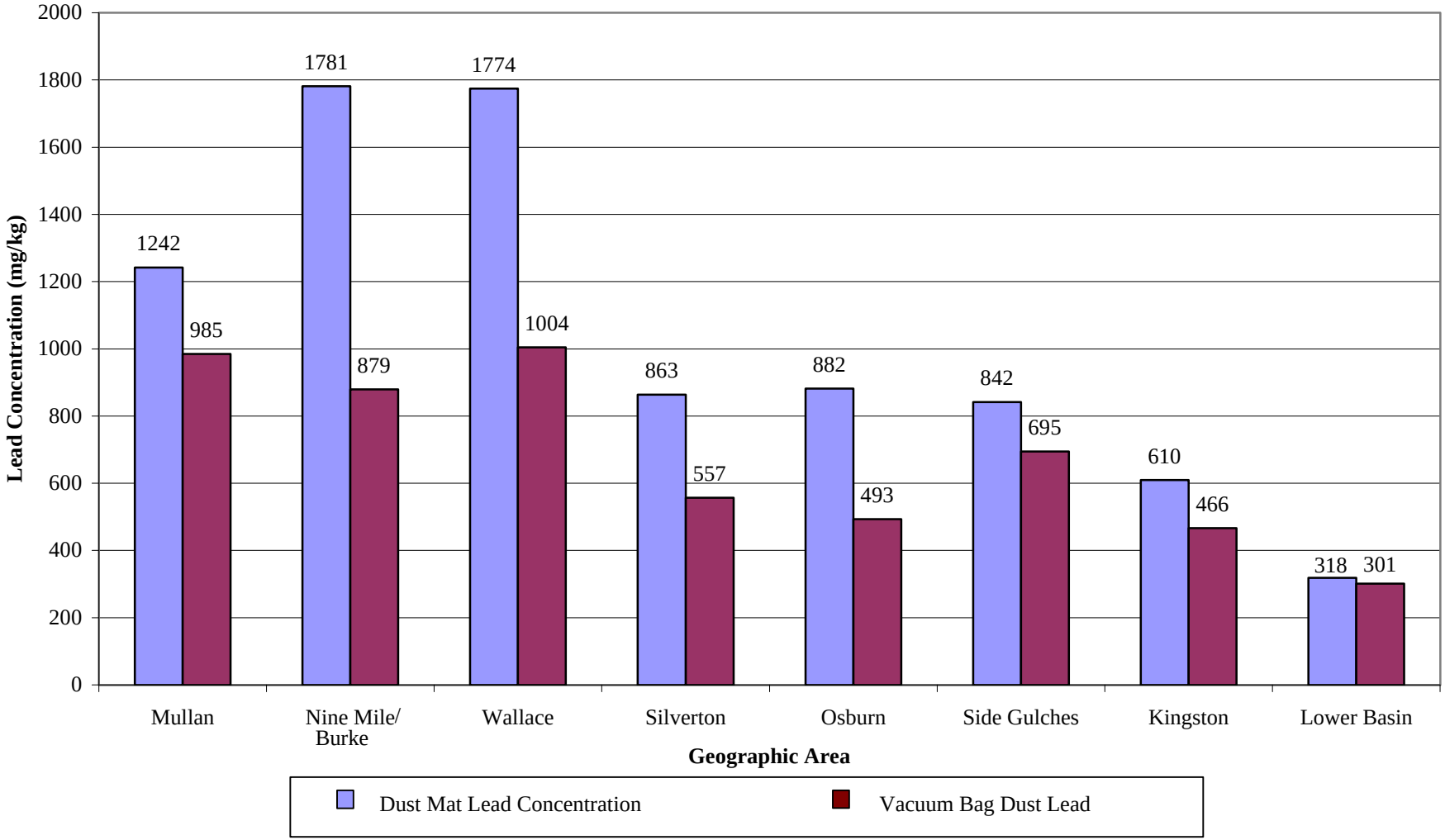


Figure 6-14a Estimated Lead Intake for Four-year-old Children by Geographic Area - EPA Default

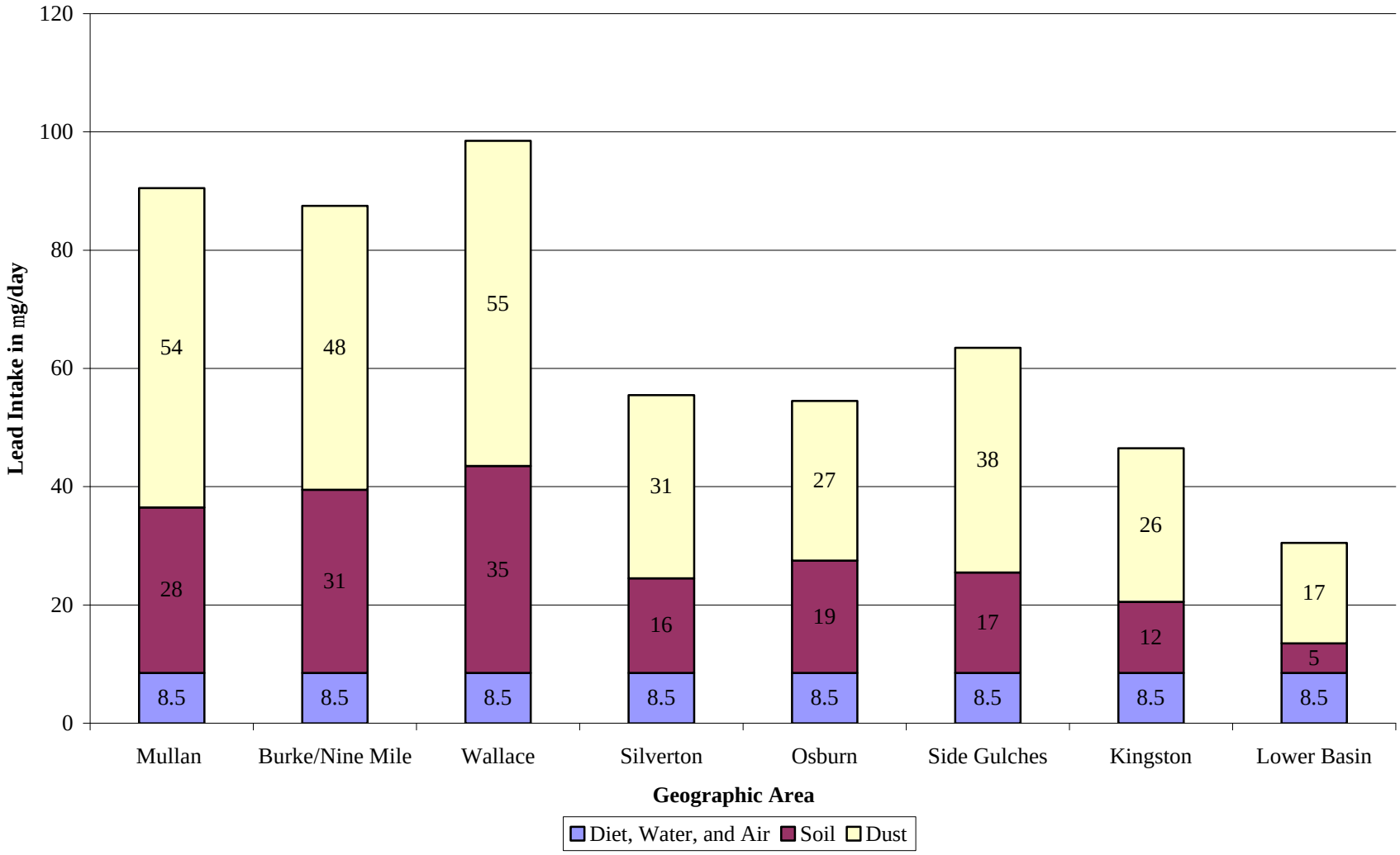


Figure 6-14b Estimated Lead Intake for Four-year-old Children by Geographic Area - Box Model

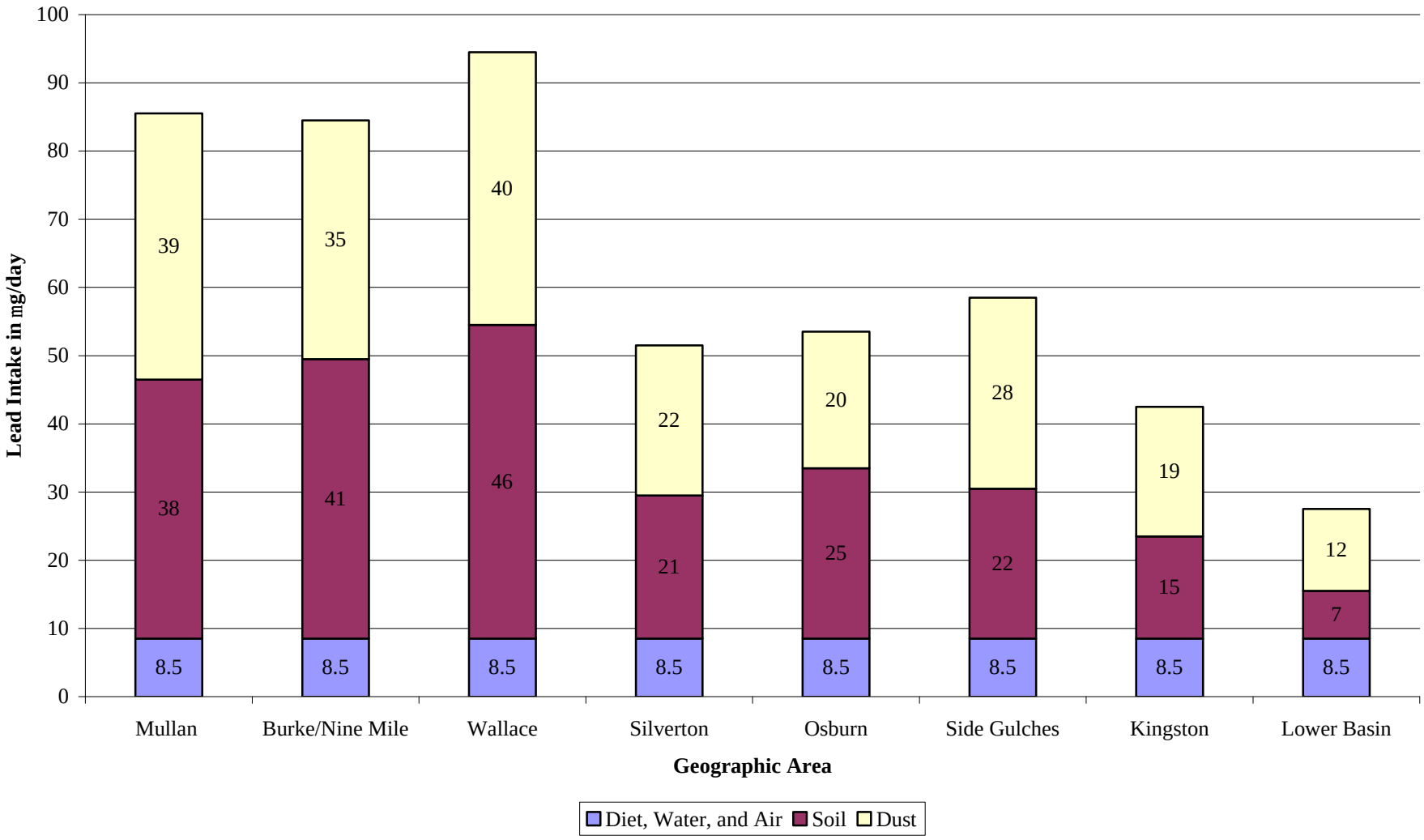


Figure 6-15a Occupational CT Lead Intake Rates Compared to Baseline

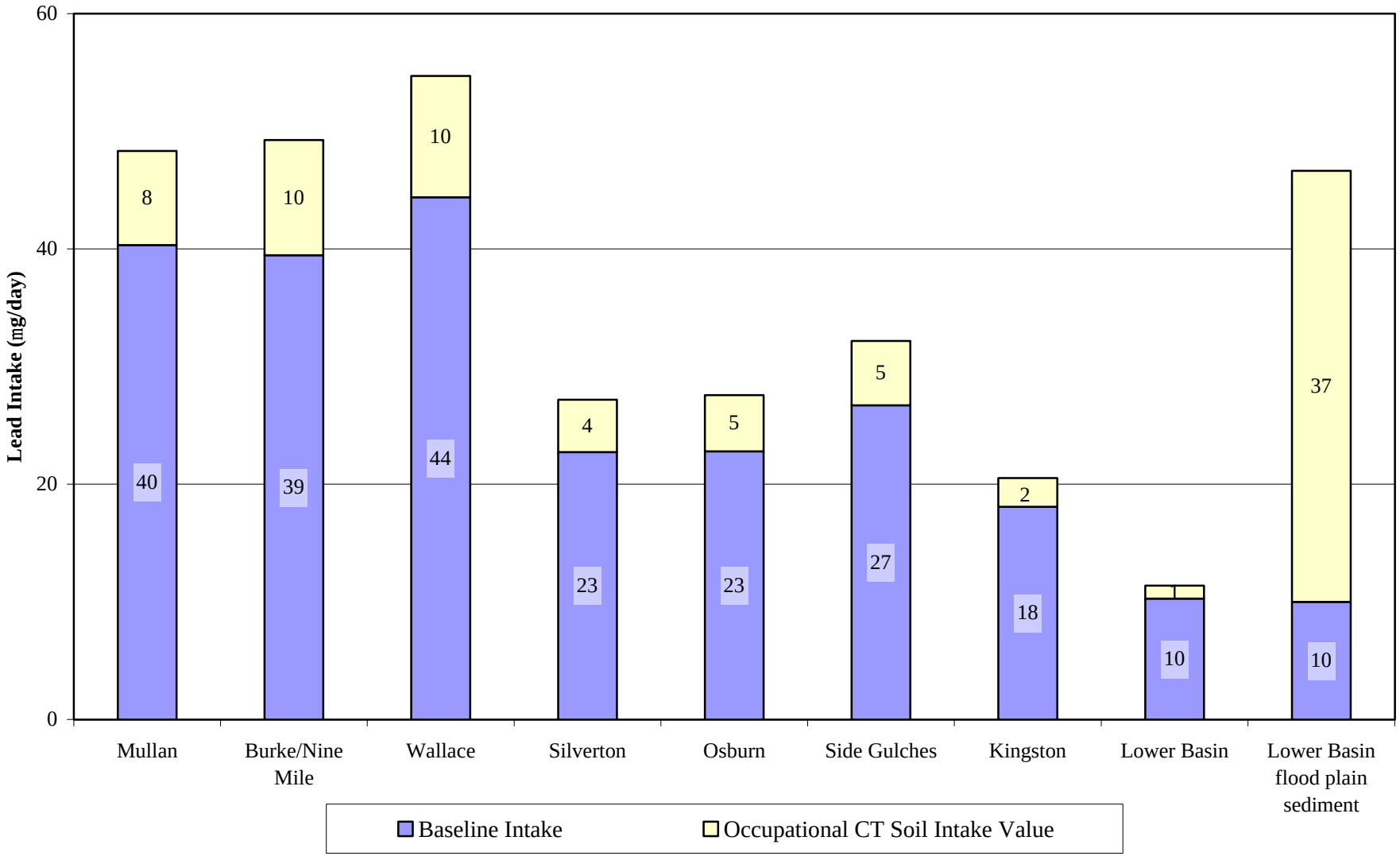


Figure 6-15b Occupational RME Lead Intake Rates Compared to Baseline

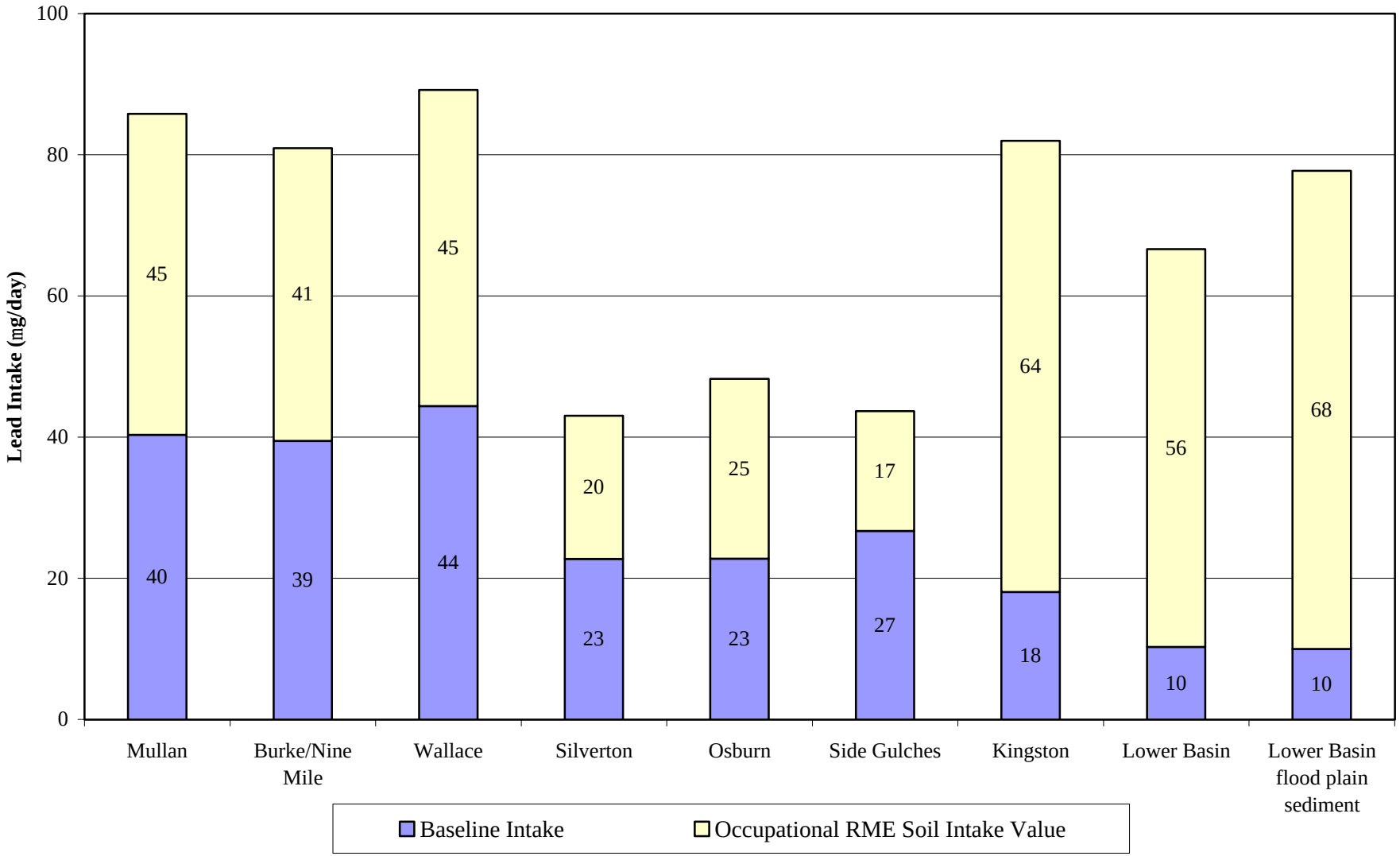


Figure 6-16a Summary of Children's Recreational Potential Incremental Lead Intakes - CT

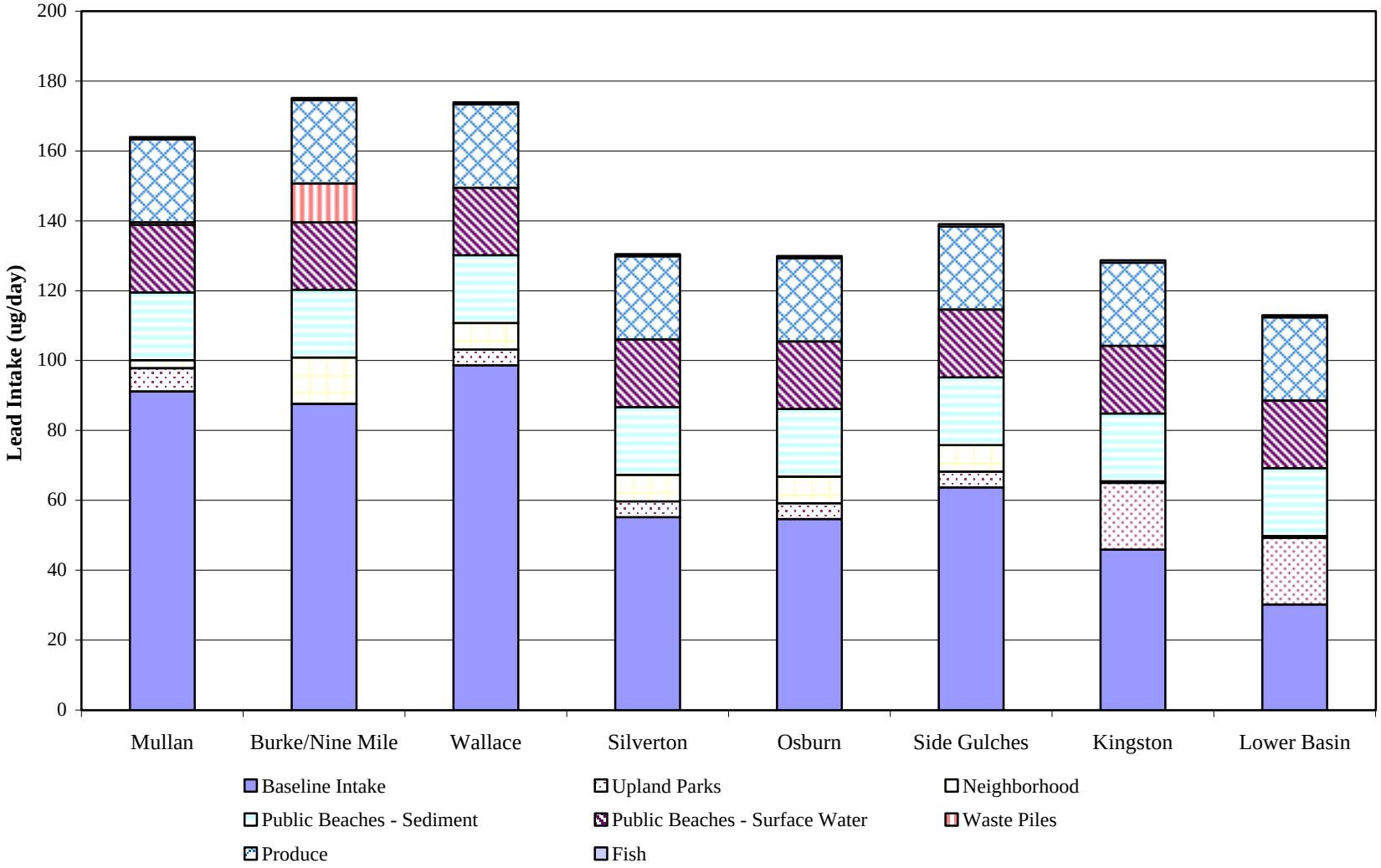


Figure 6-16b Summary of Children's Recreational Potential Incremental Lead Intakes - RME

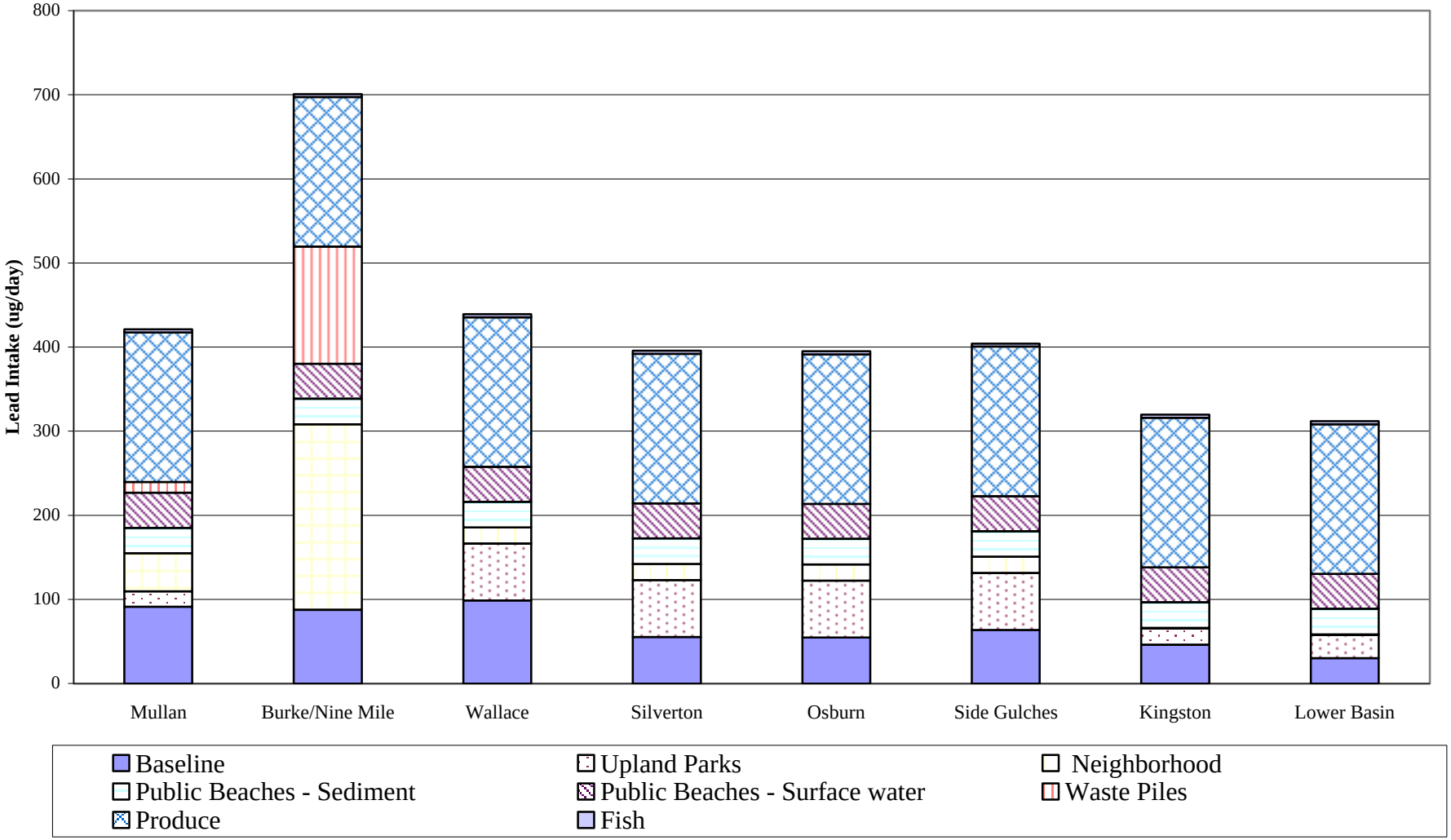


Figure 6-16c Summary of Adult Recreational Potential Incremental Lead Intakes - CT

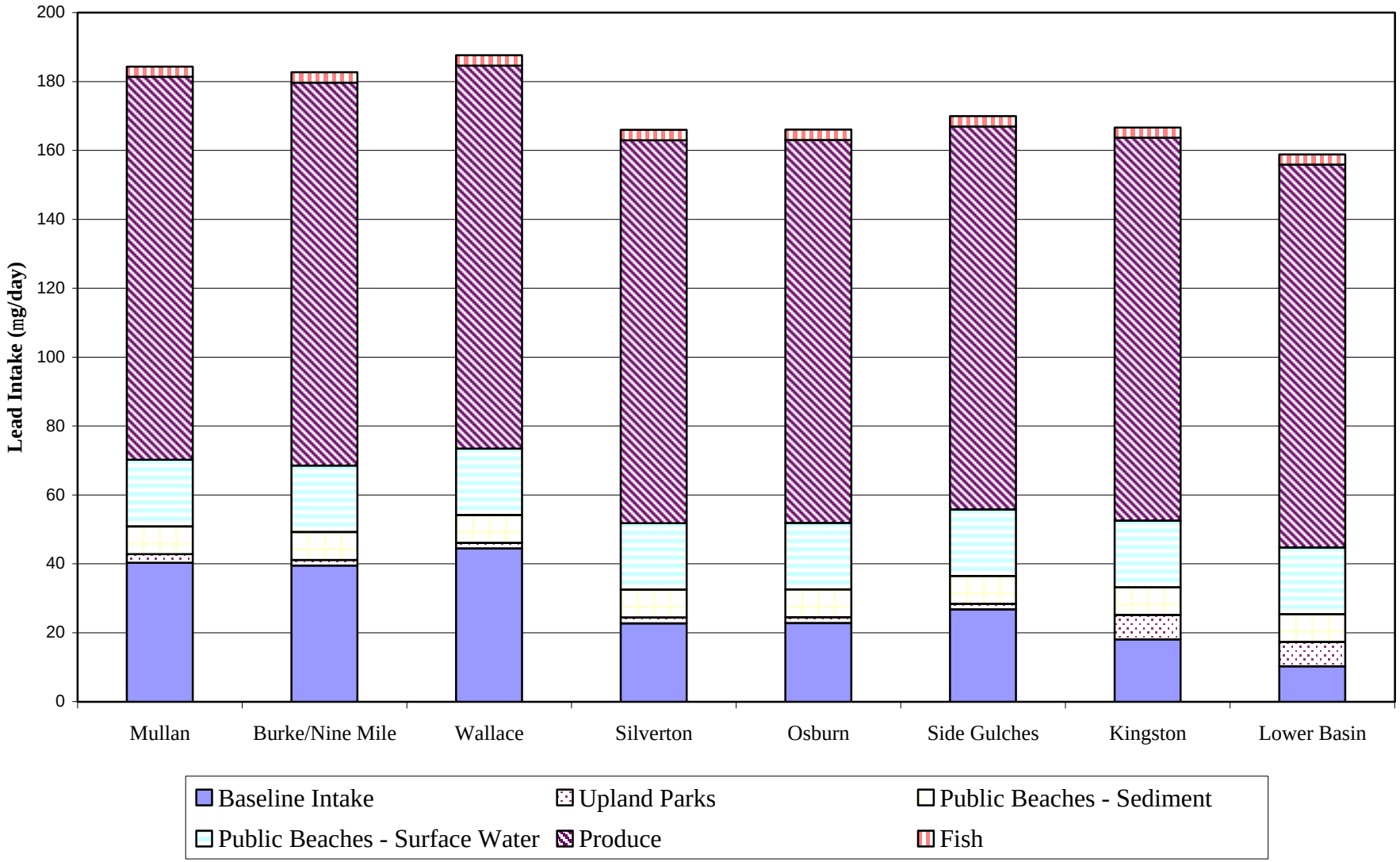


Figure 6-16d Summary of Adult Recreational Potential Incremental Lead Intakes - RME

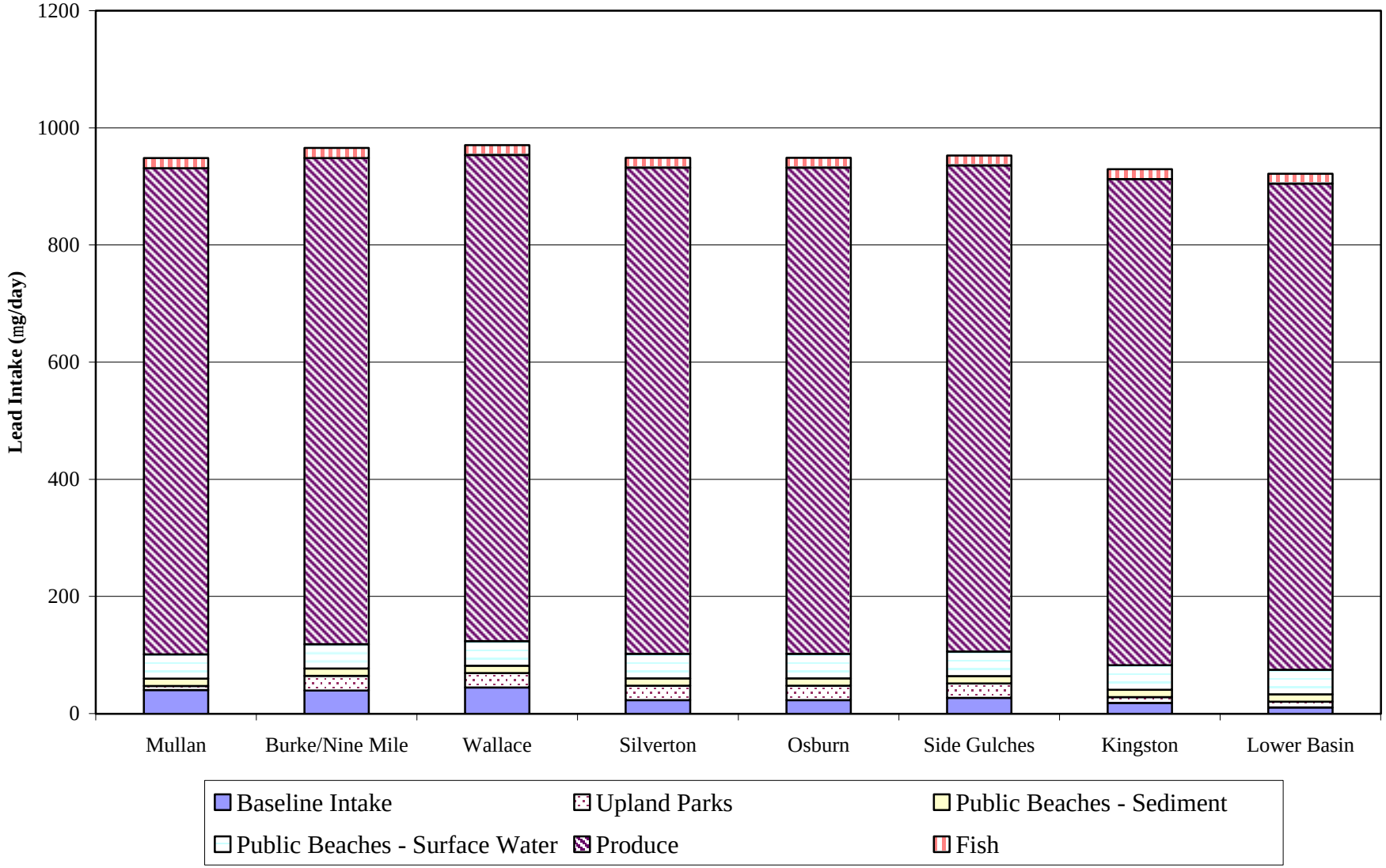


Figure 6-17a Estimated CT Tribal Children Lead Intake Rates

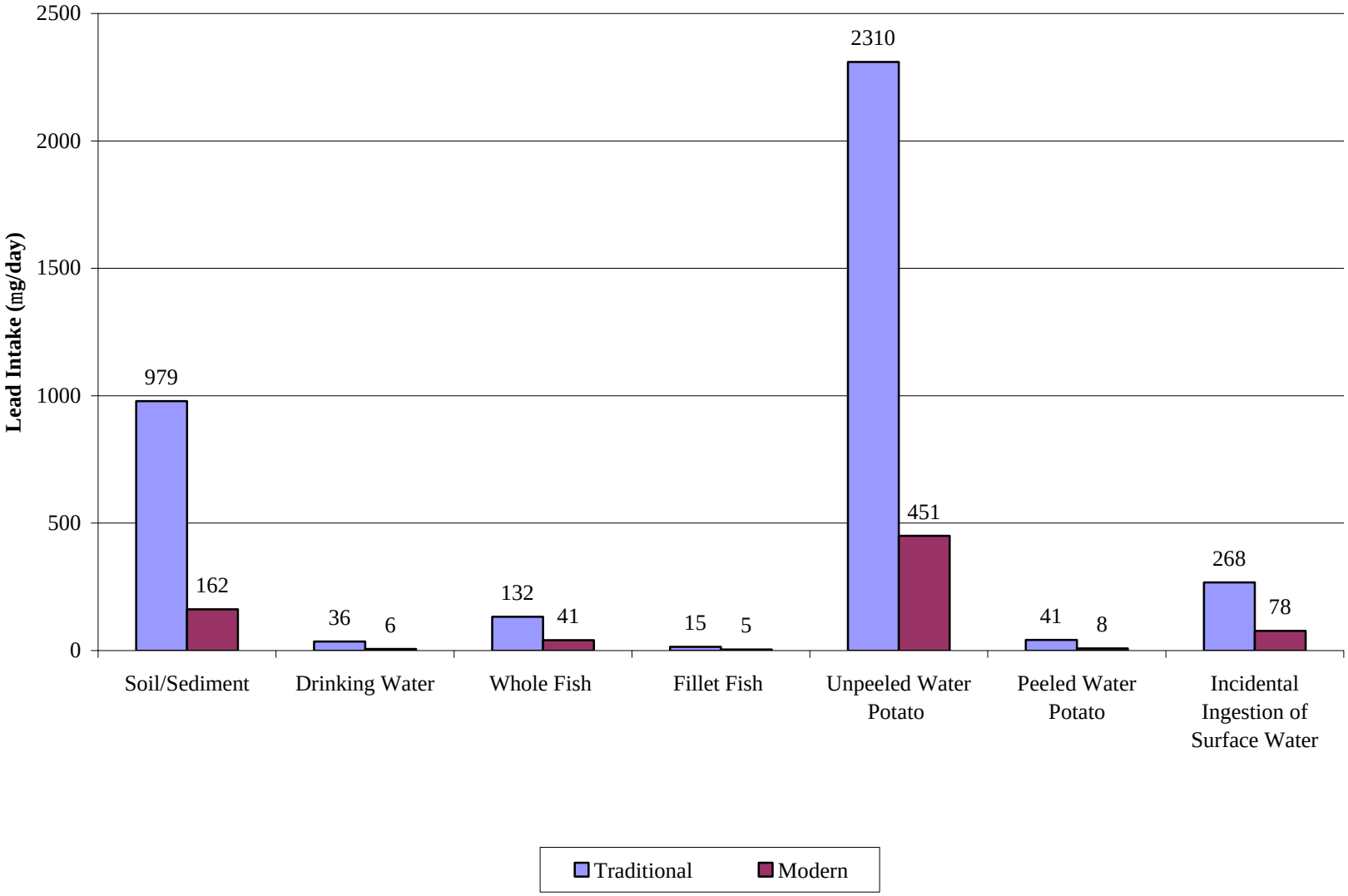


Figure 6-17b Estimated RME Tribal Children Lead Intake Rates

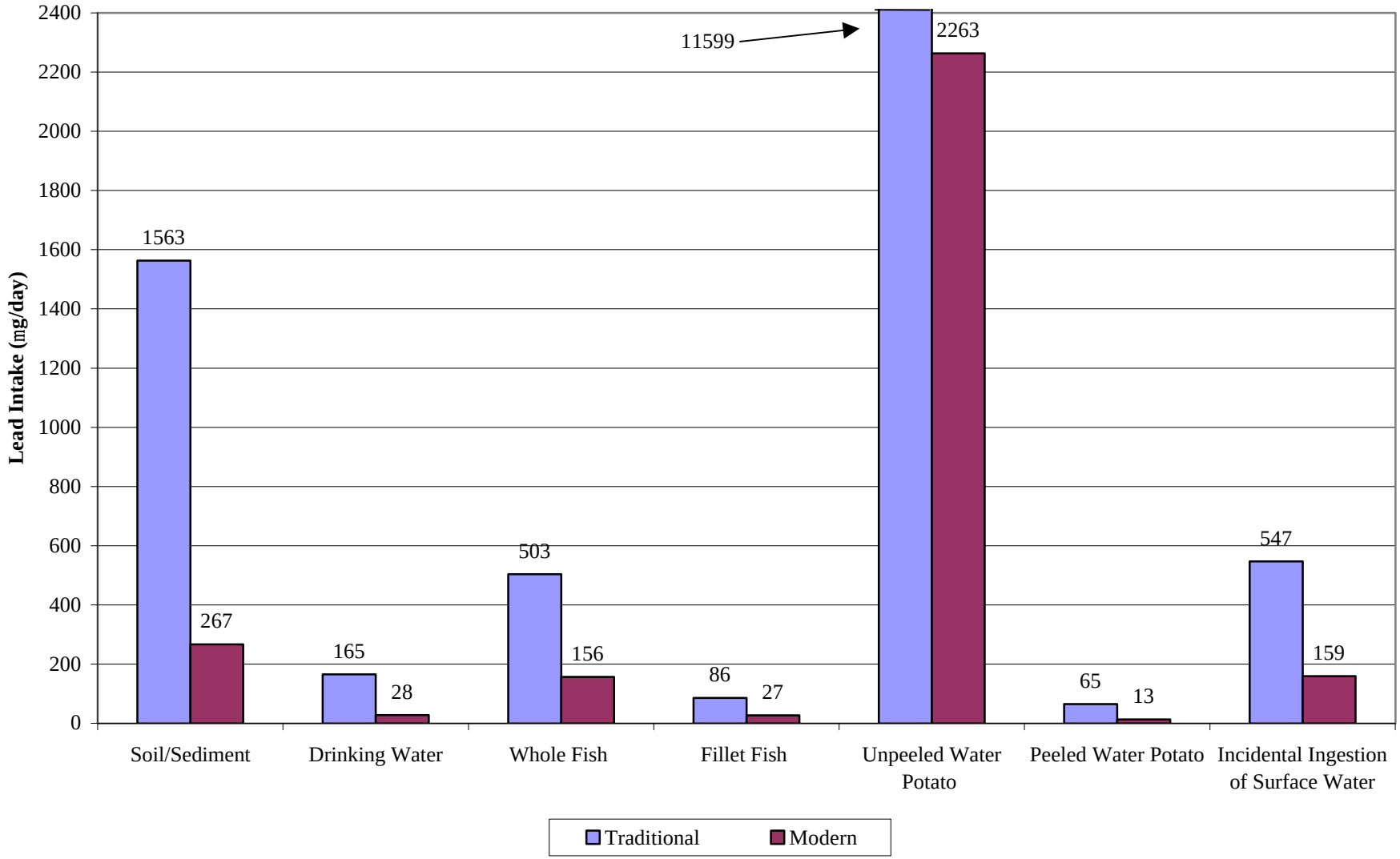


Figure 6-18a Estimated CT Tribal Adult Lead Intake Rates

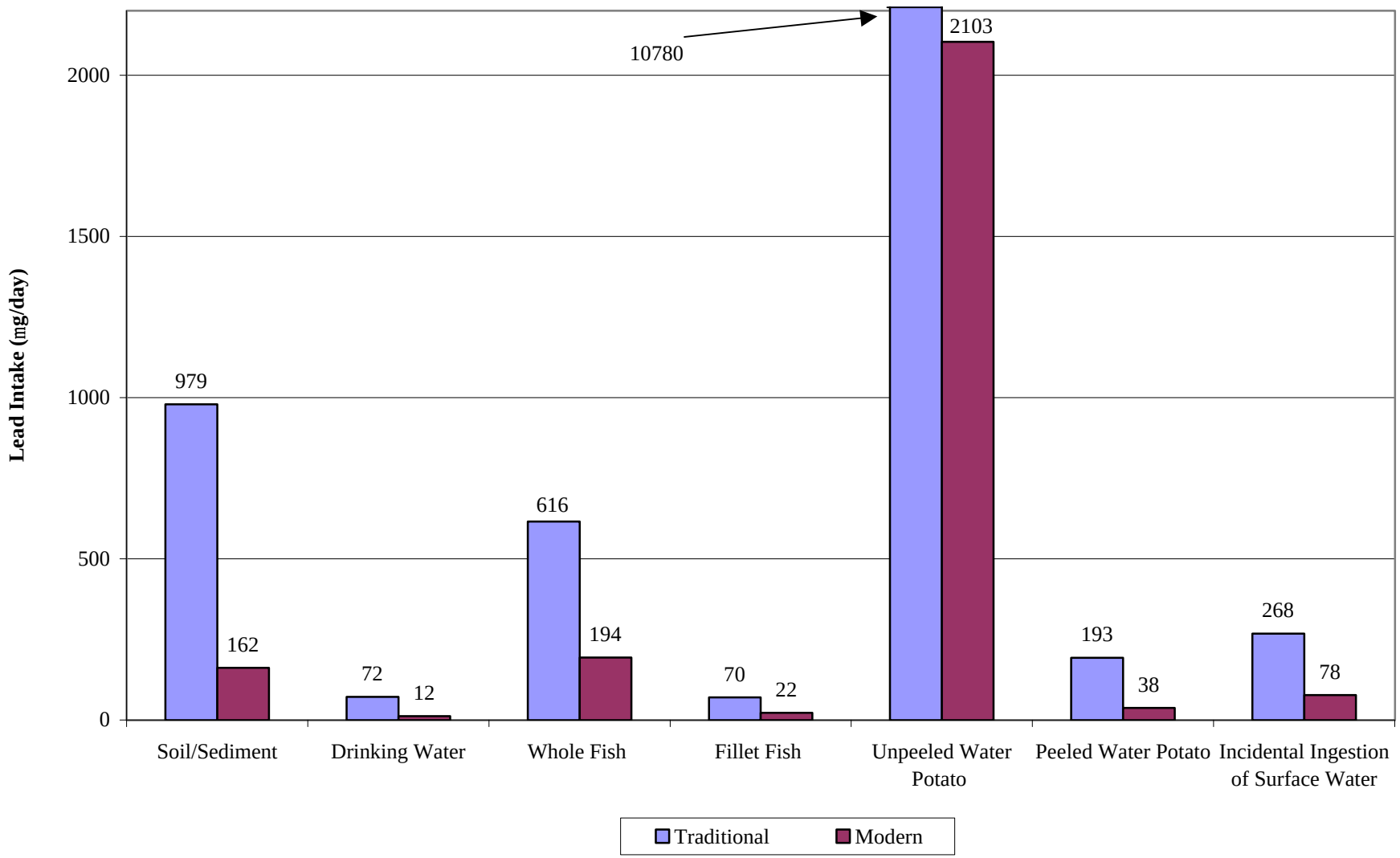


Figure 6-18b Estimated RME Tribal Adult Lead Intake Rates

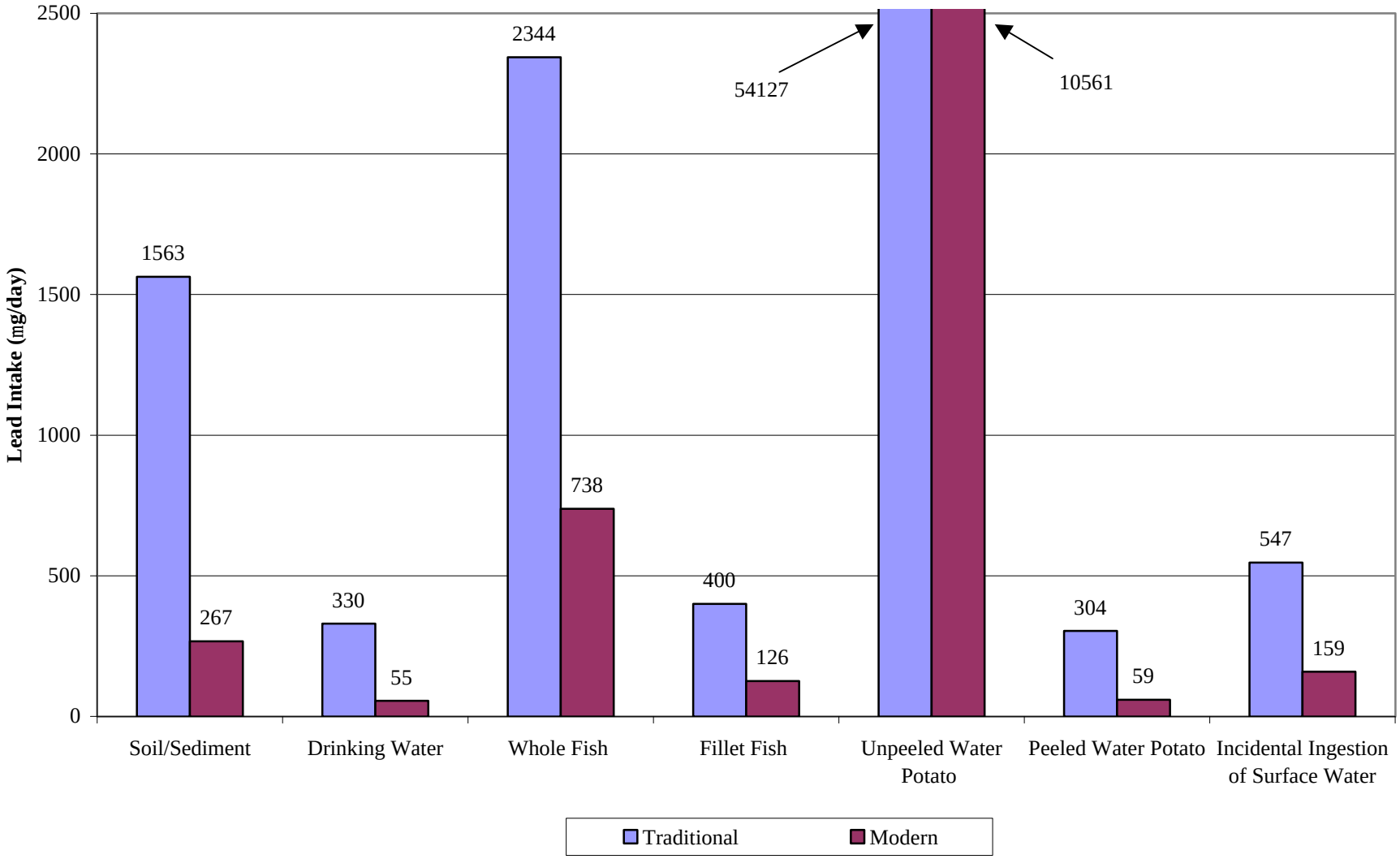


Figure 6-19a Observed and Predicted Geomean Blood Lead Levels for 9-84 Month Old Children Only - IEUBK Batch Mode

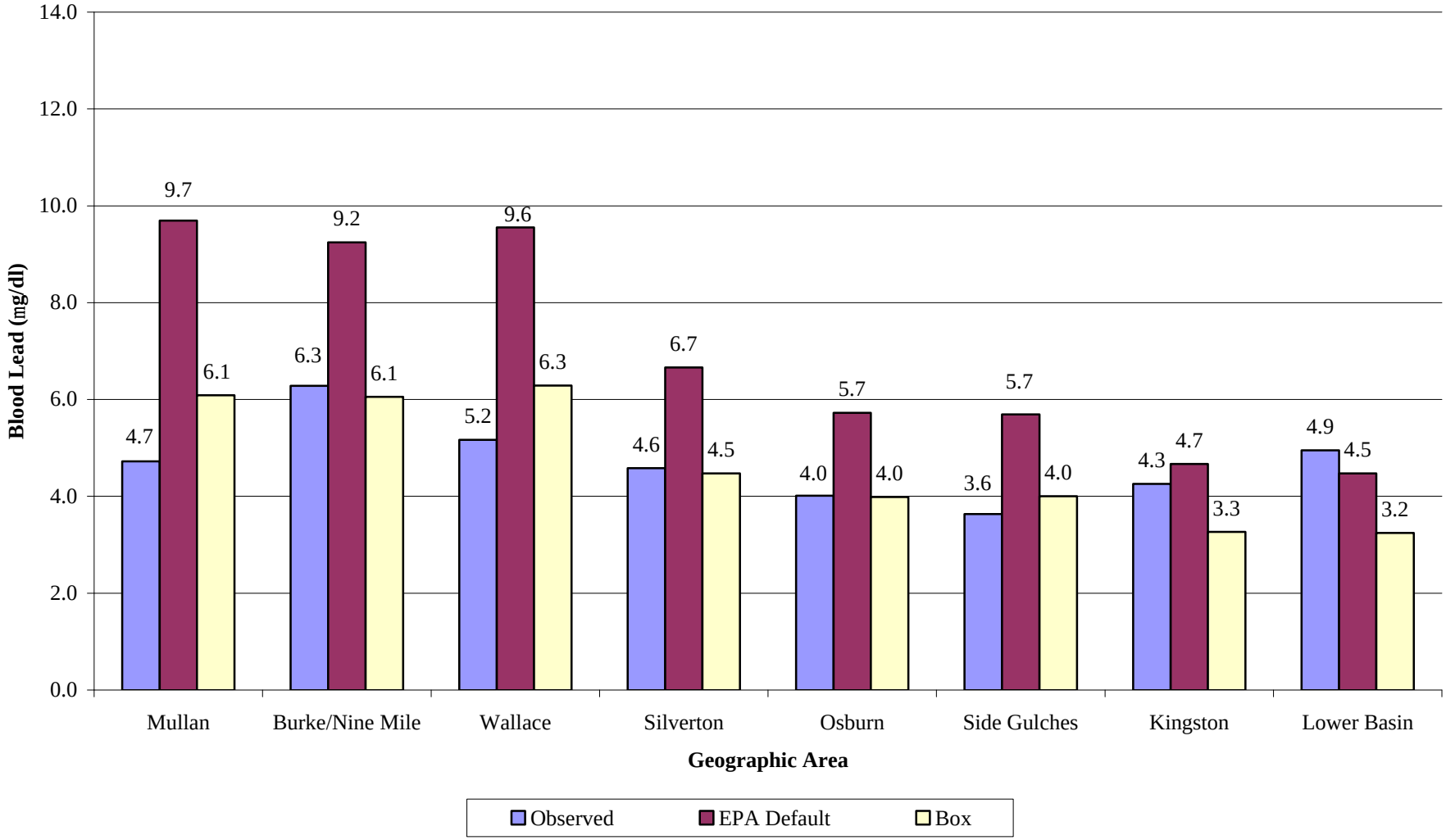


Figure 6-19b Observed and Predicted Blood Lead Levels for 9-60 Month Old Children-IEUBK Batch Mode

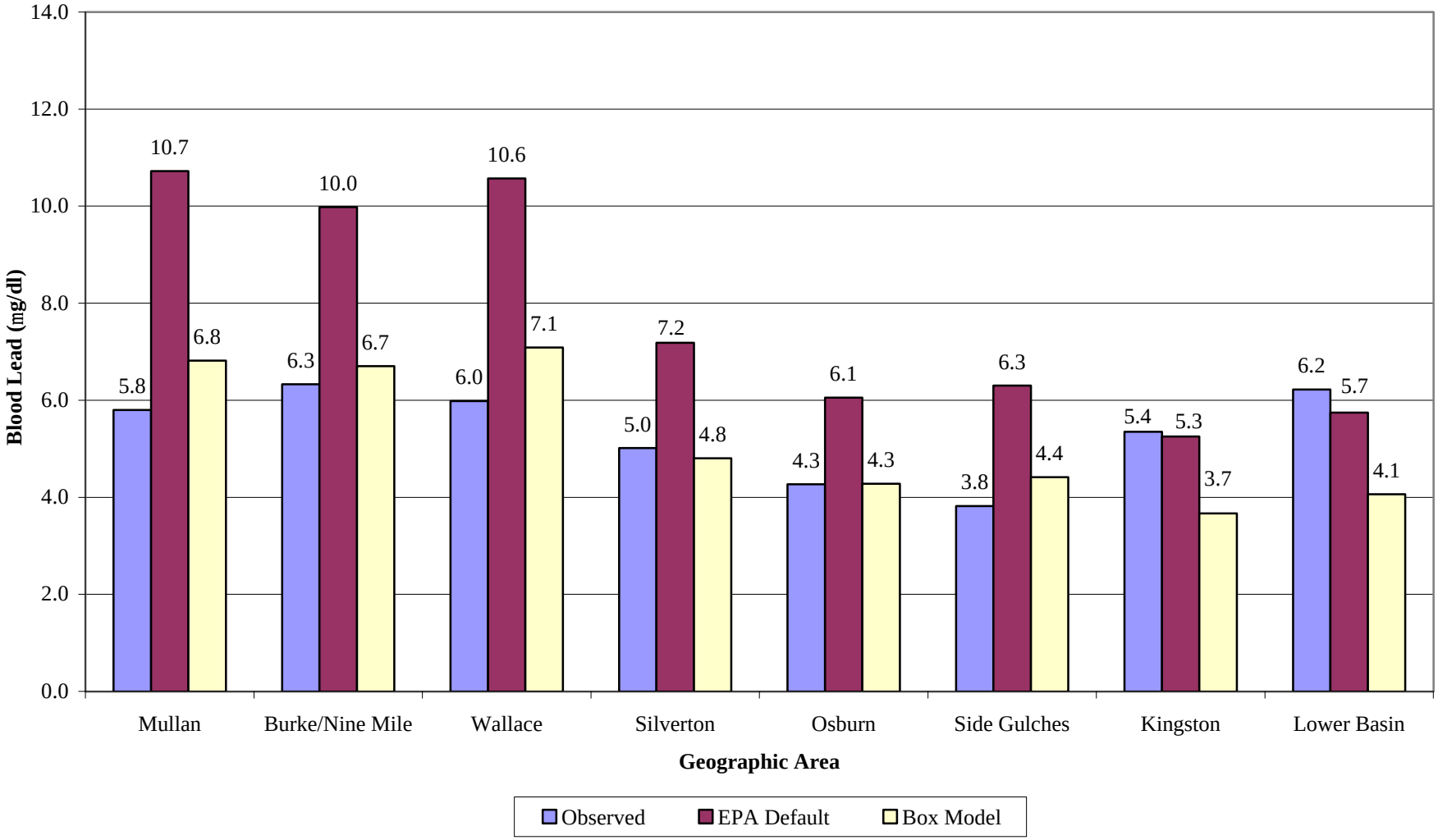


Figure 6-19c Observed and Predicted Blood Lead Levels for 9-24 Month Old Children-IEUBK Batch Mode

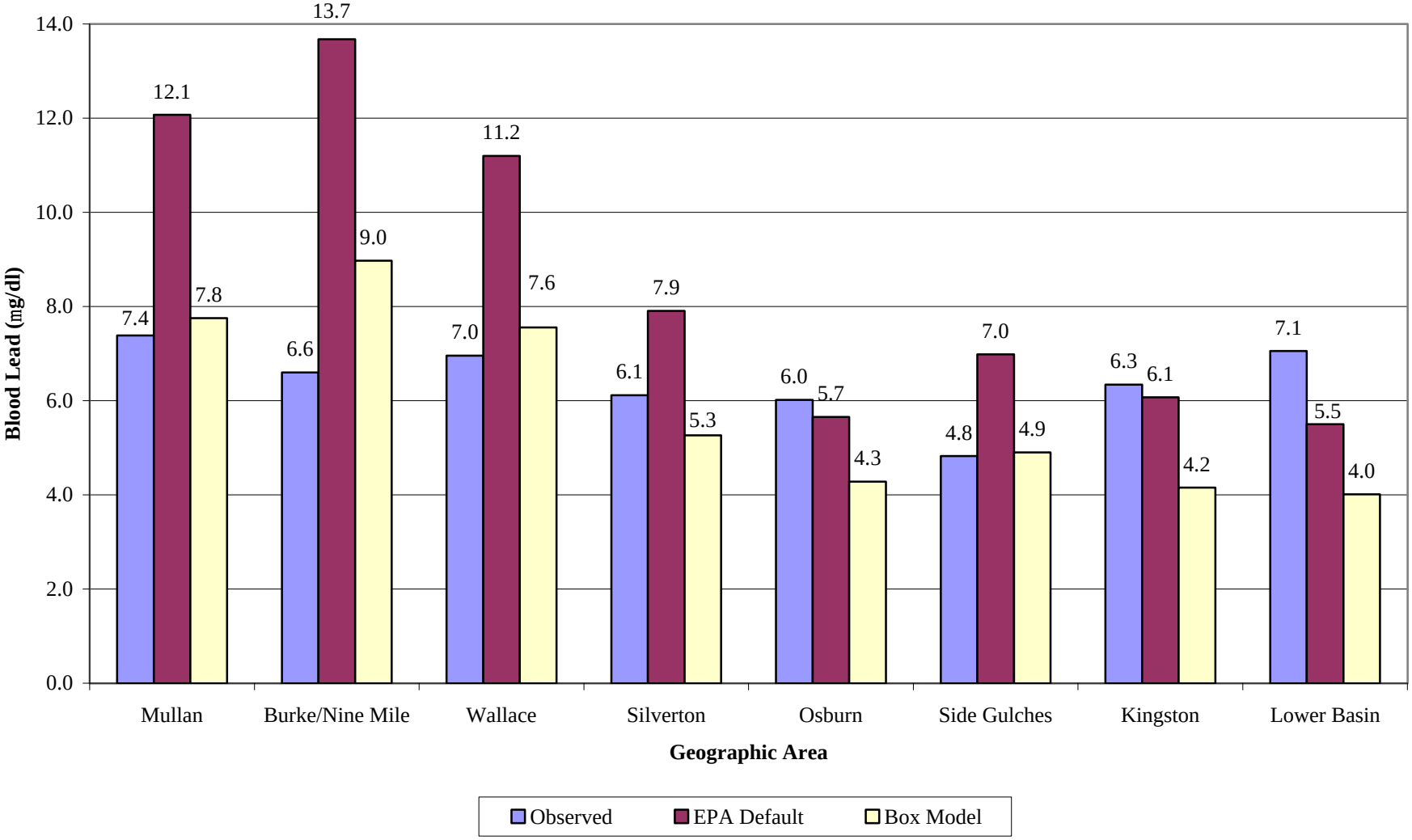


Figure 6-20a Observed and Predicted Percent to Exceed 10 mg/dl for 9-84 Month Old Children - IEUBK Batch Mode

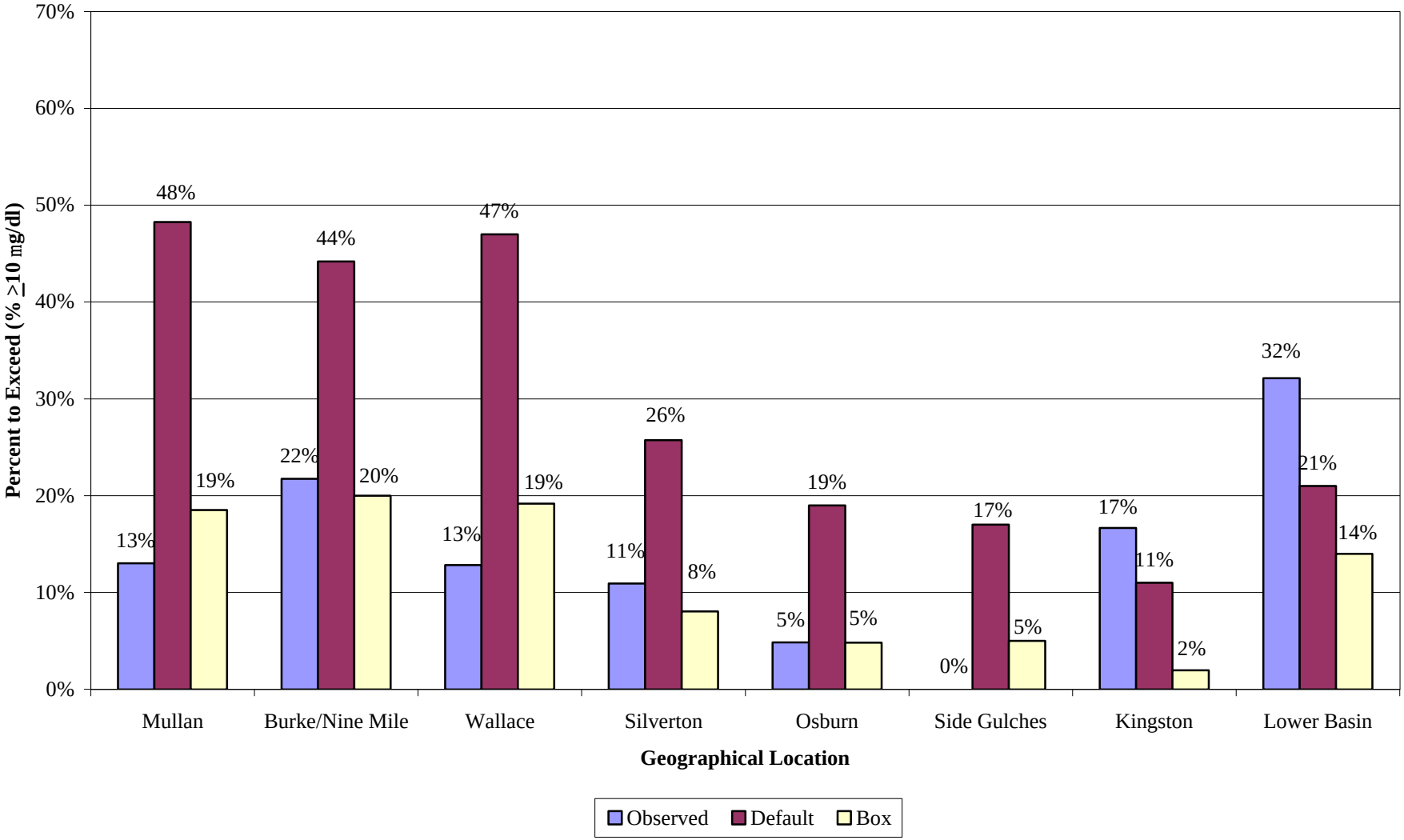


Figure 6-20b Observed and Predicted Percent to Exceed Levels for 9-60 Month Old Children-IEUBK Batch Mode

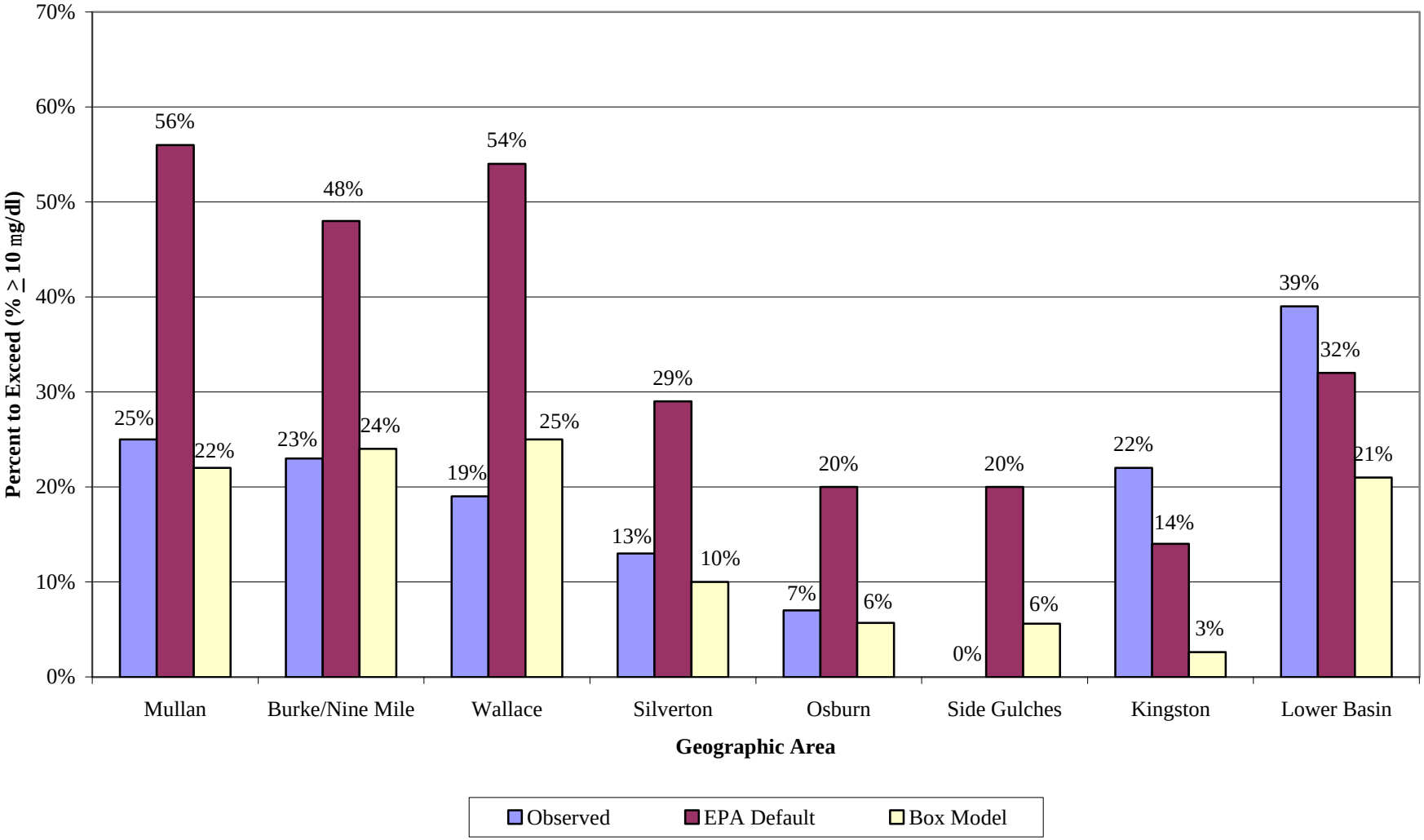


Figure 6-20c Observed and Predicted Percent to Exceed Levels for 9-24 Month Old Children-IEUBK Batch Mode

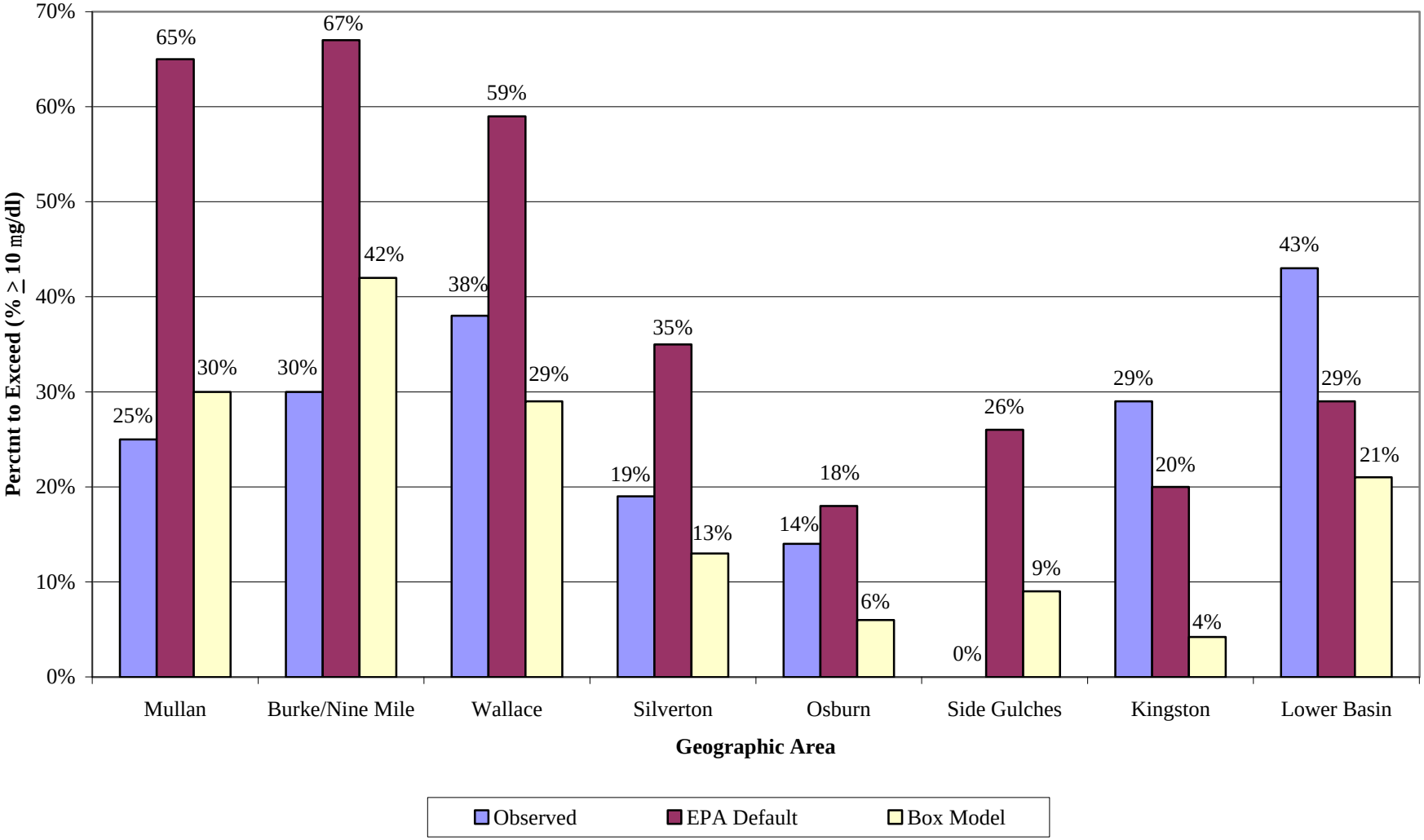


Figure 6-21a Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Mullan

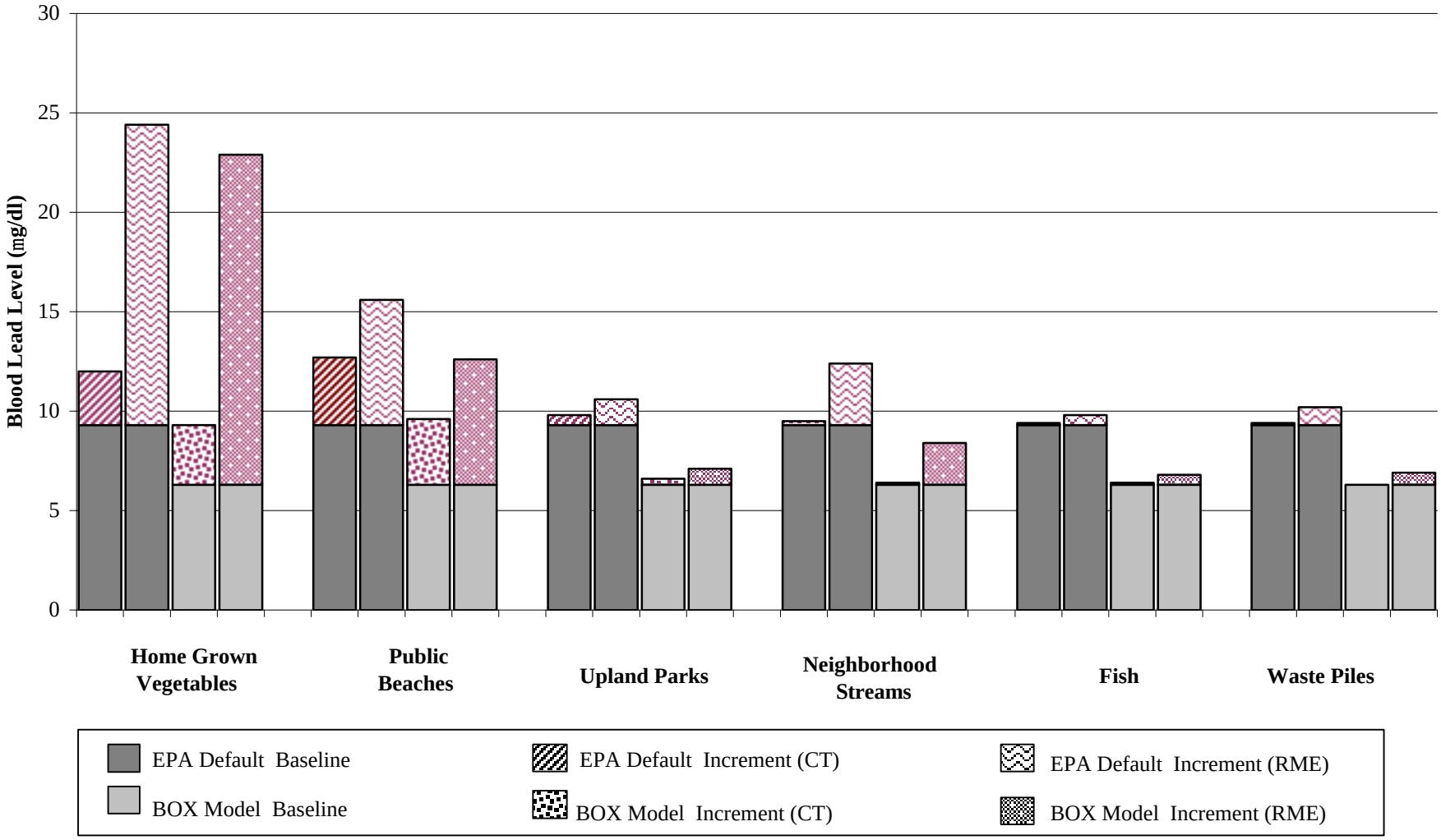


Figure 6-21b Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Burke/Nine Mile

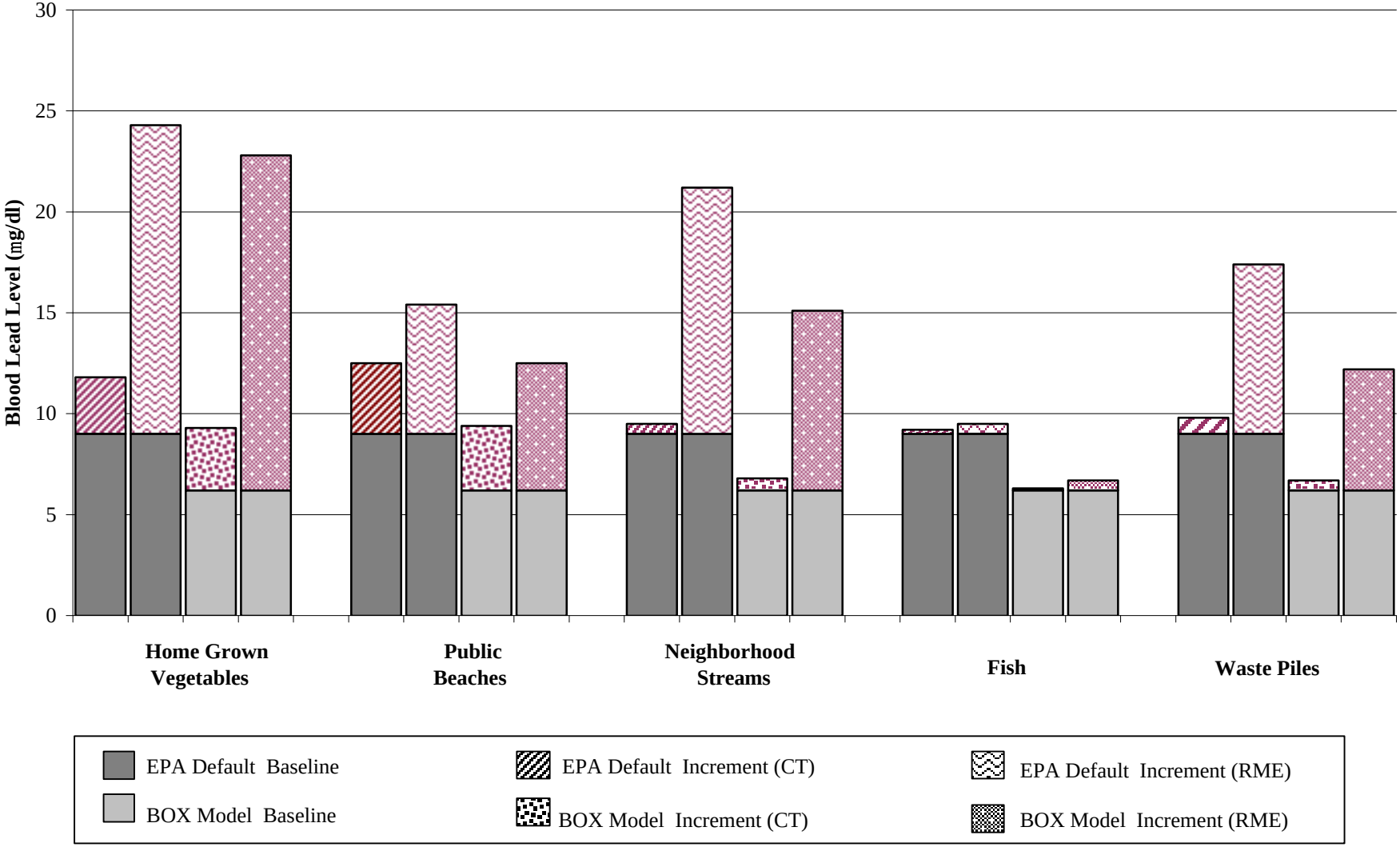


Figure 6-21c Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Wallace

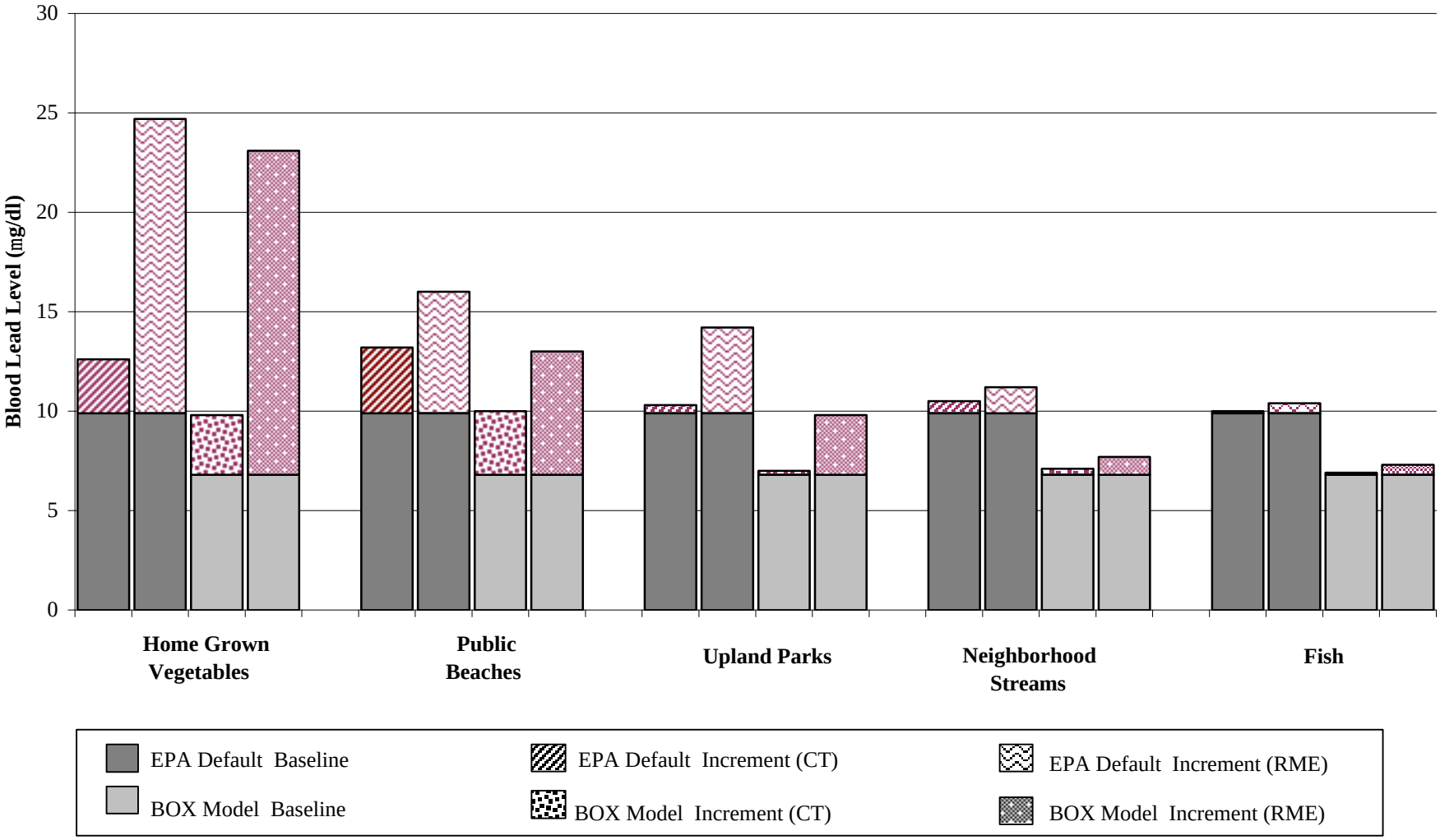


Figure 6-21d Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Silverton

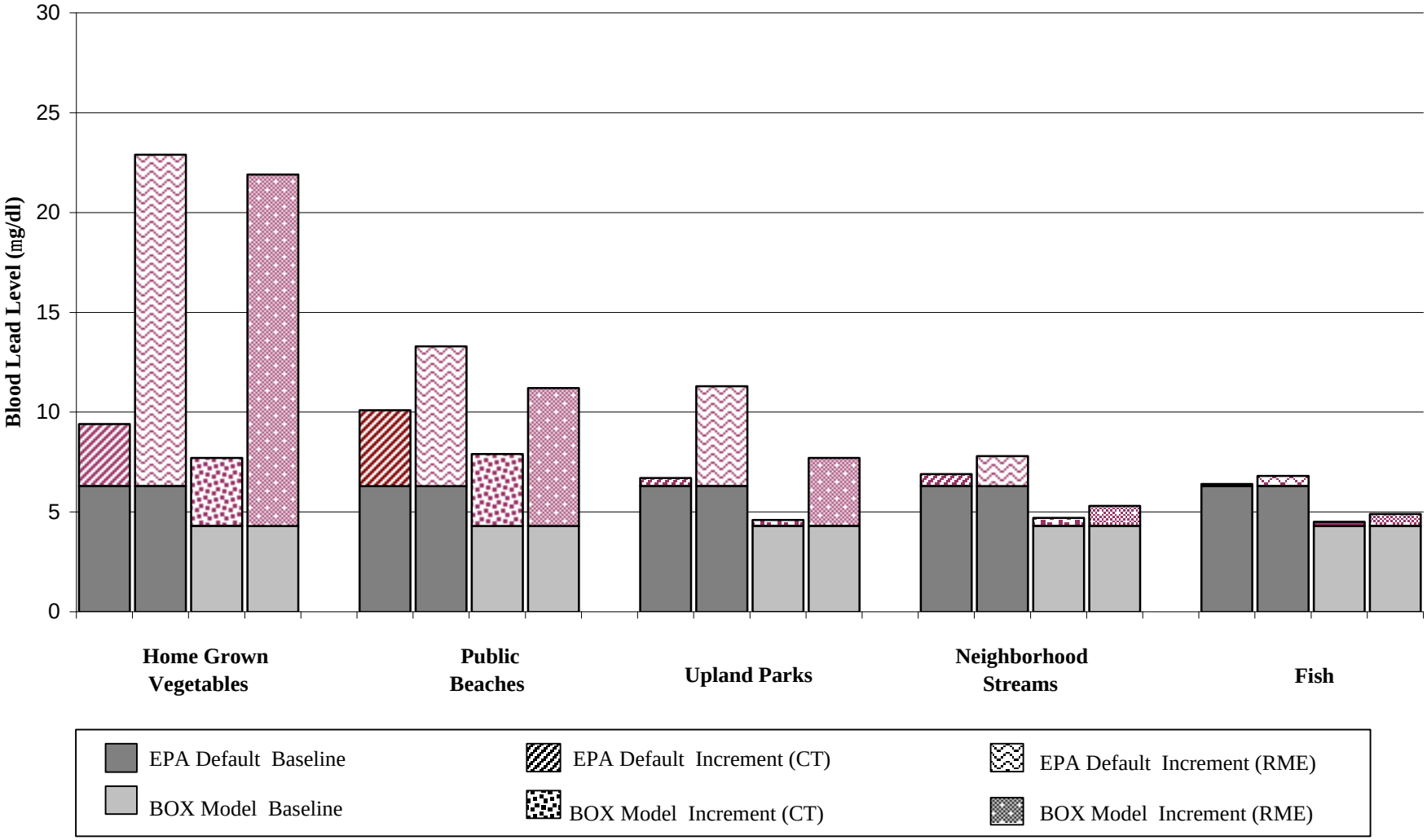


Figure 6-21e Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Osburn

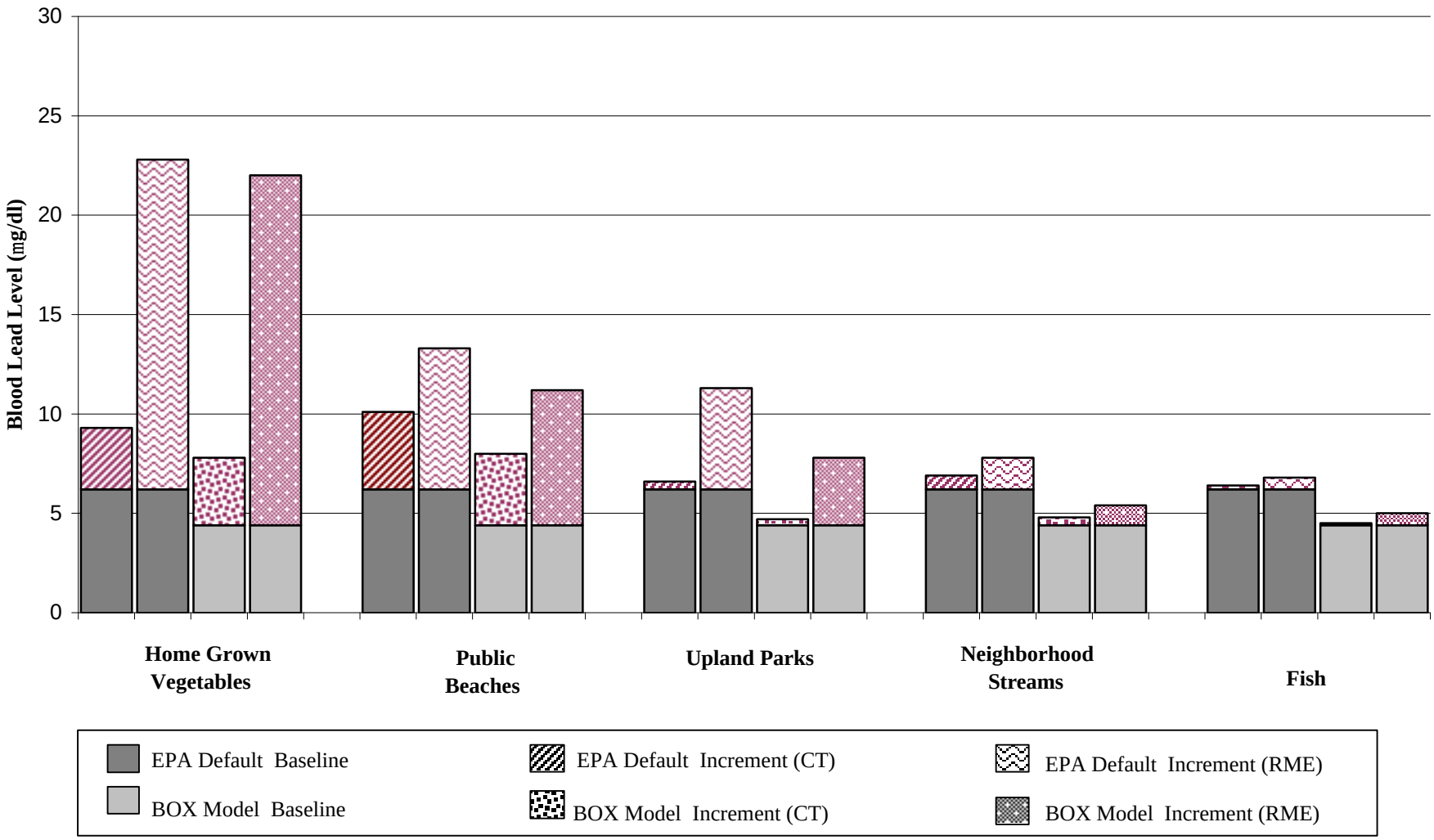


Figure 6-21f Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Side Gulches

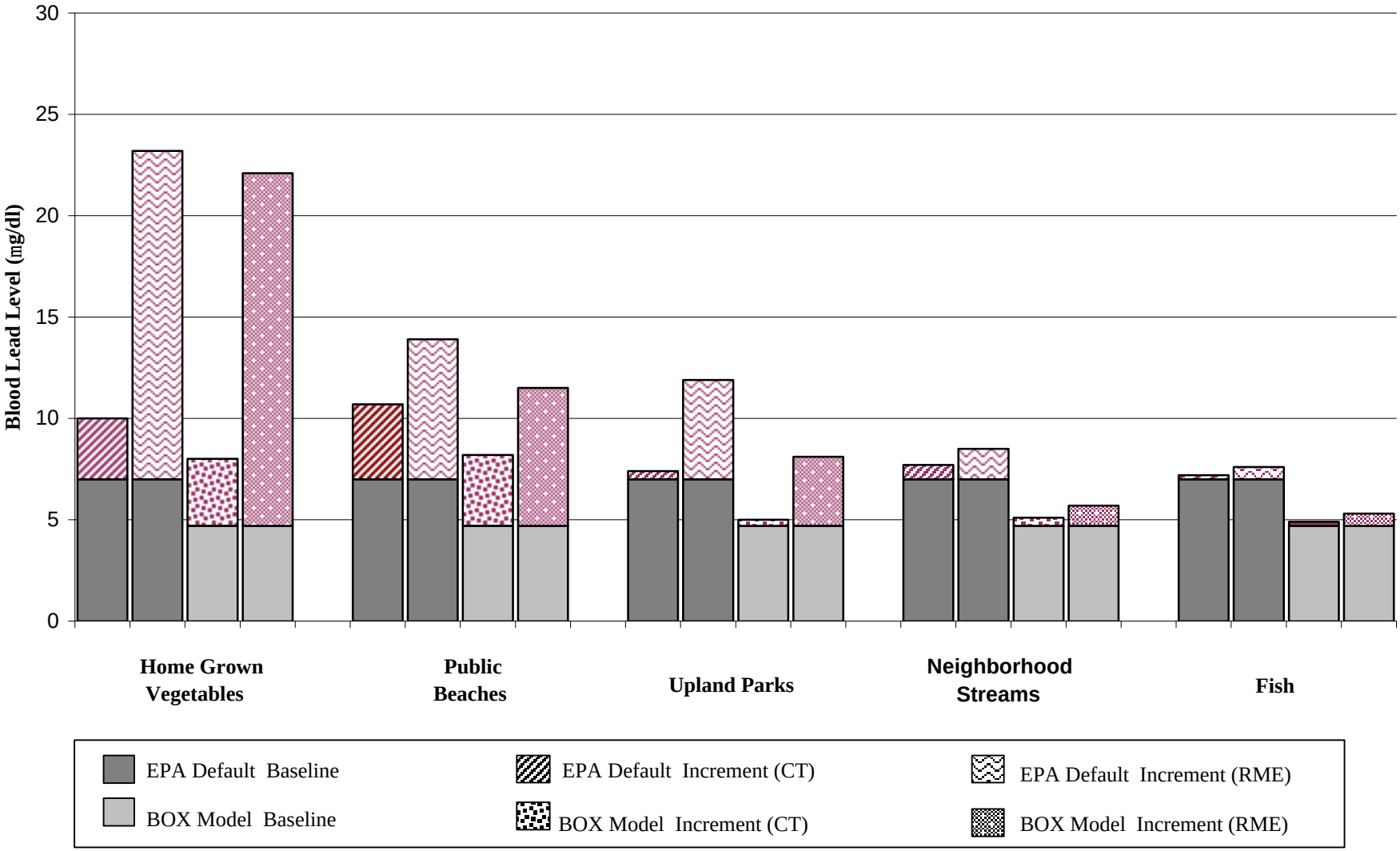


Figure 6-21g Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Kingston

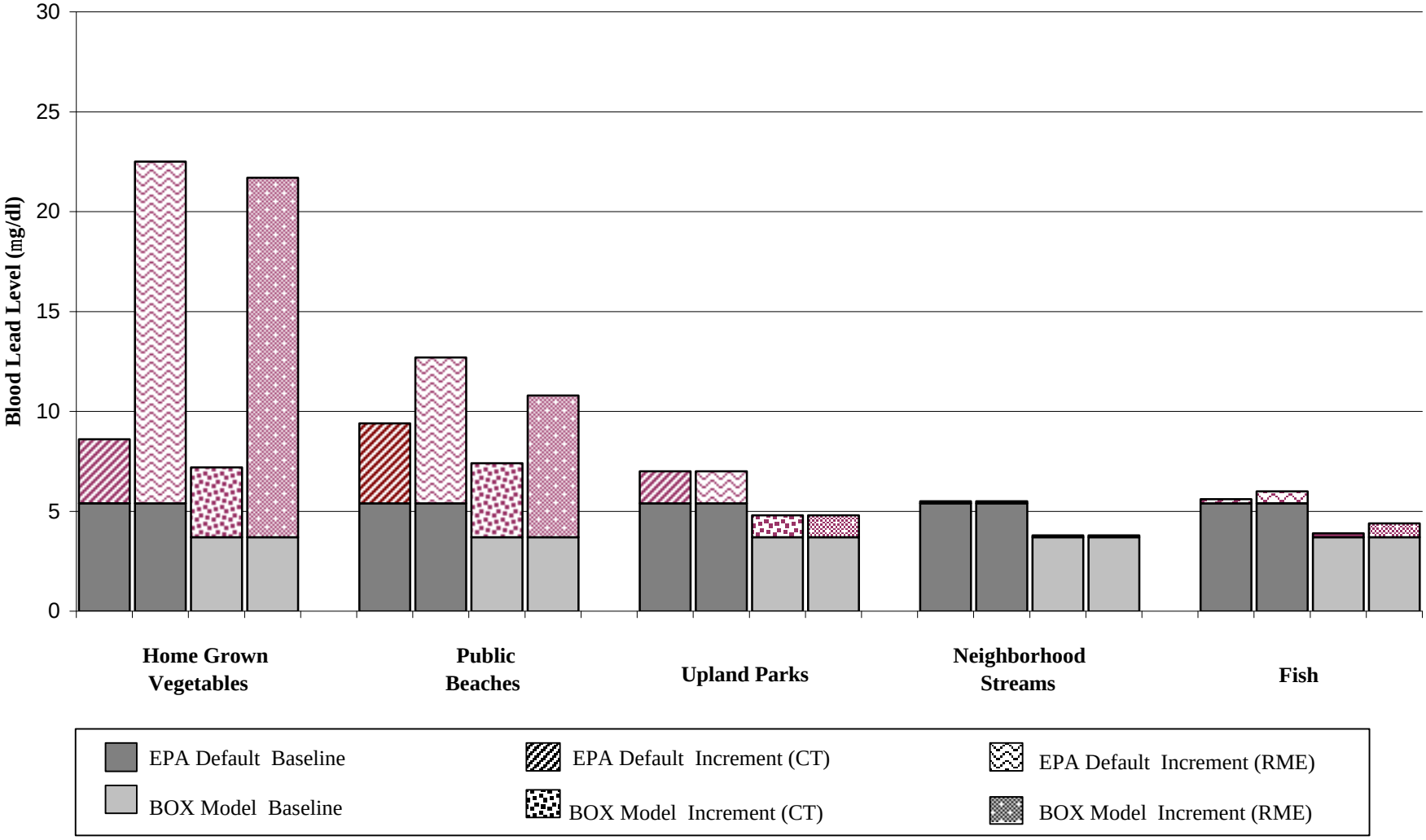


Figure 6-21h Incremental Blood Lead Estimates for 0-9 Year-Old Children by Recreational Activity and Local Foodstuff - Lower Basin

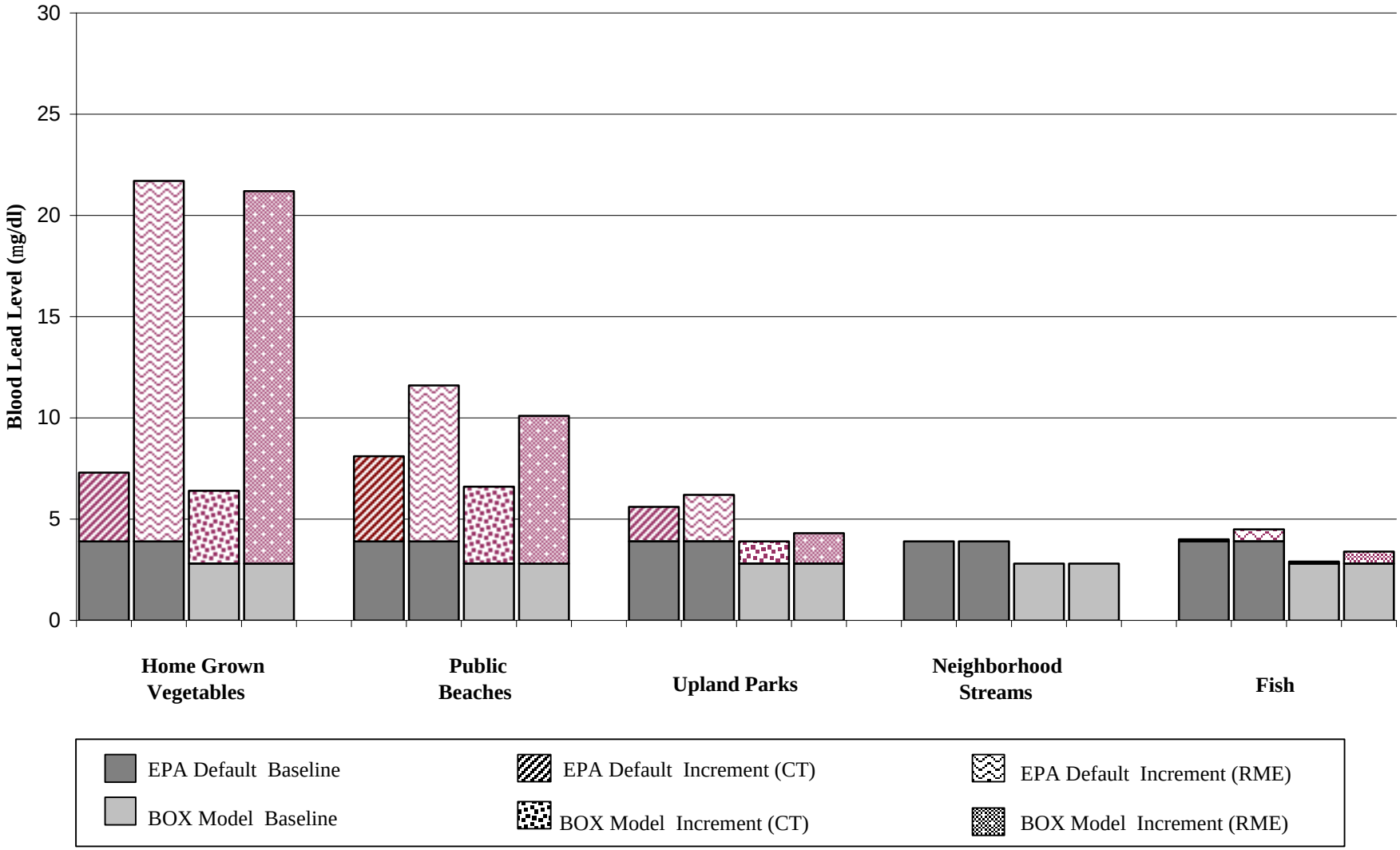


Figure 6-22a Estimated Percent of Children to Exceed 10 mg/dl Blood Lead Associated with Various Yard Soil Cleanup Action Criteria Using Different Dust Concentrations - Wallace

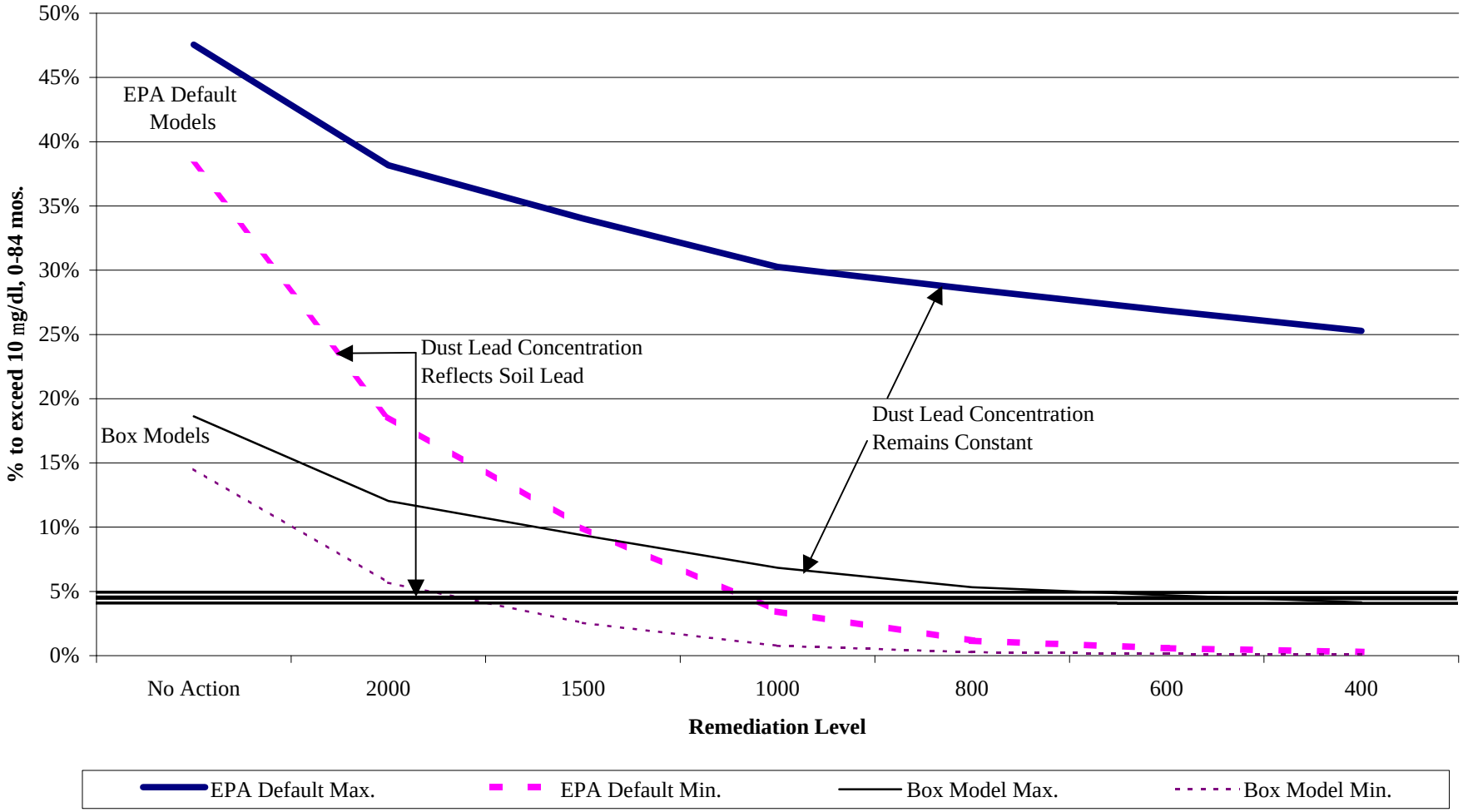


Figure 6-22b Estimated Percent of Children to Exceed 10 mg/dl Blood Lead Associated with Various Yard Soil Cleanup Action Criteria Using Different Dust Concentrations - Lower Basin

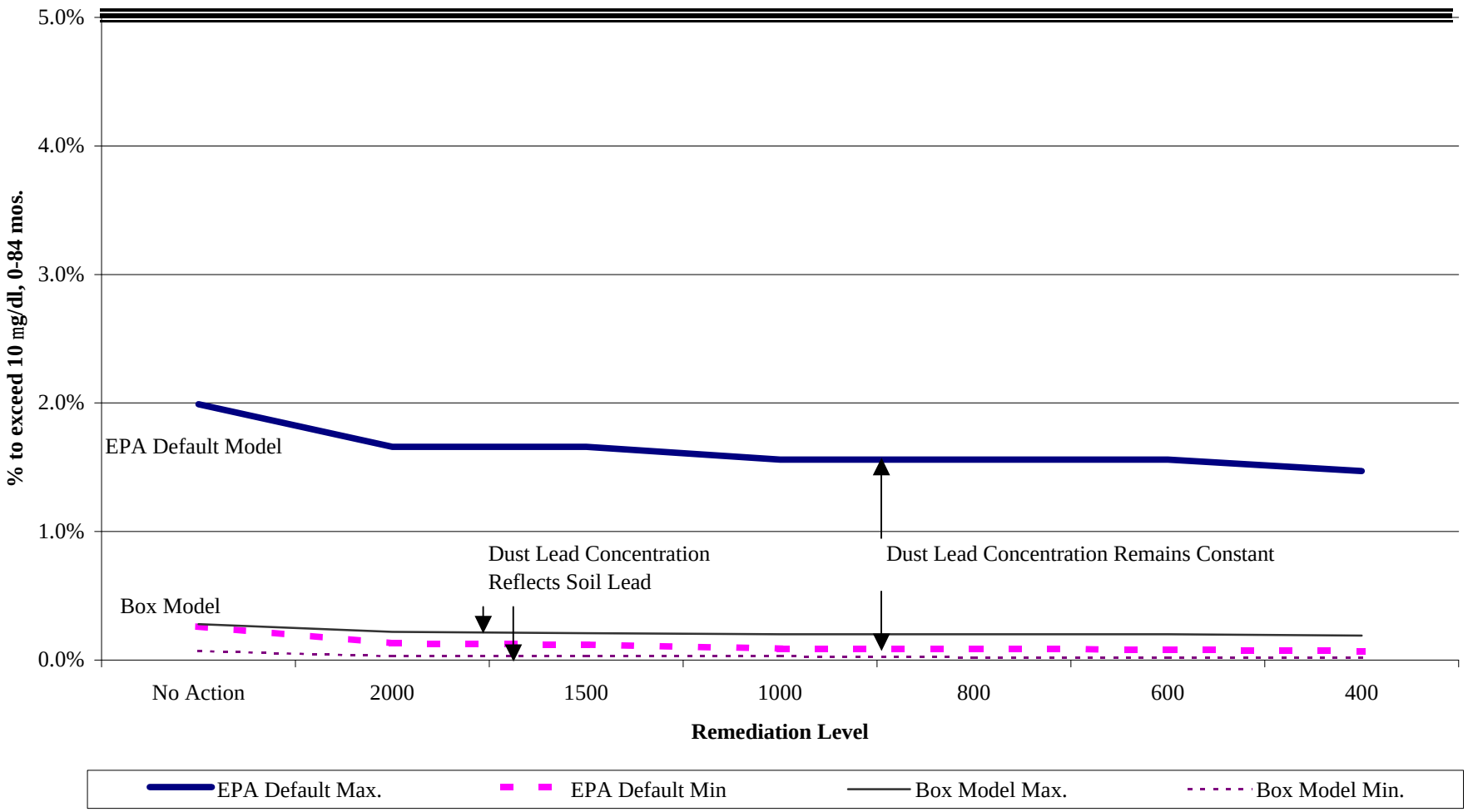


Figure 6-23a Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Mullan

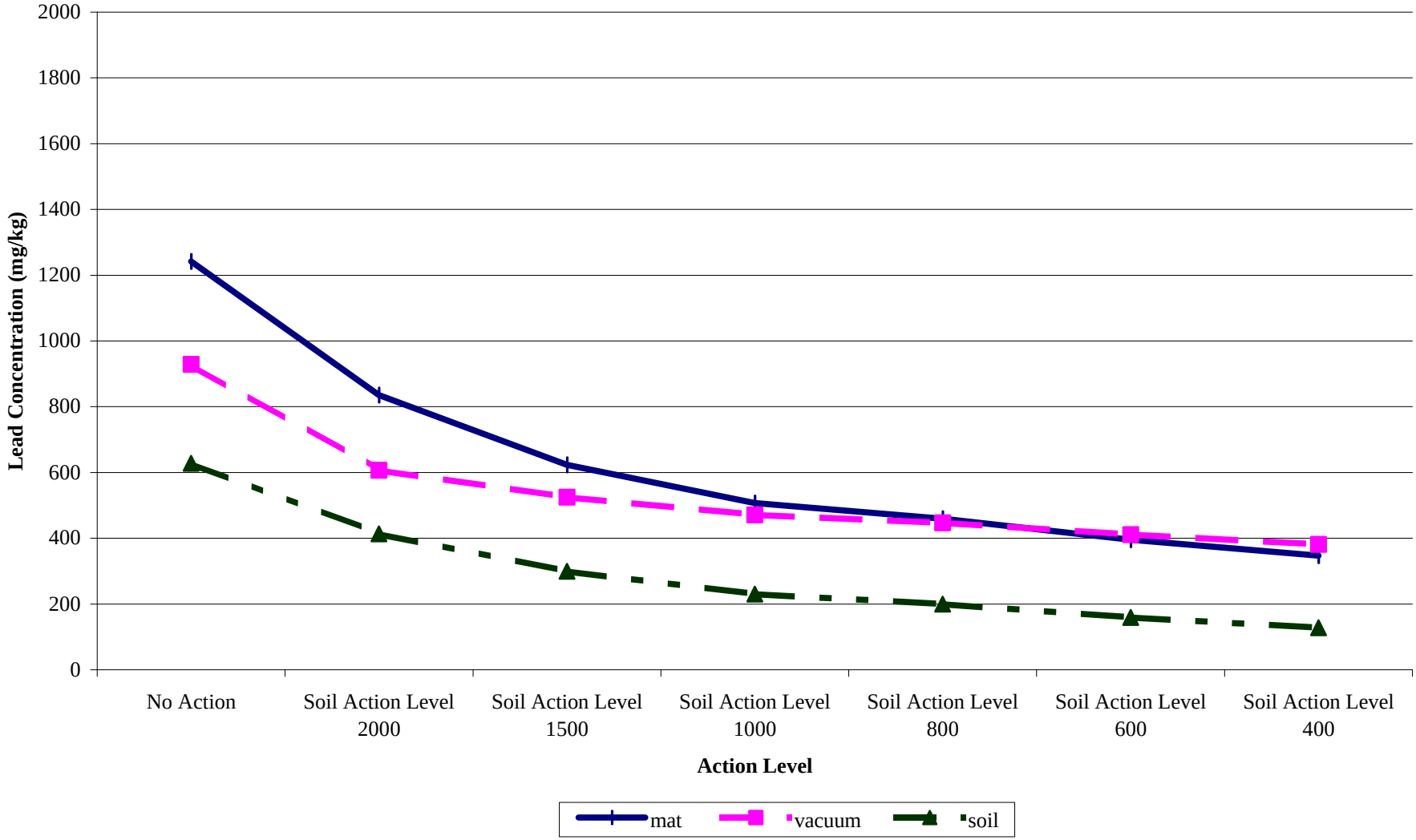


Figure 6-23b Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Burke/Nine Mile

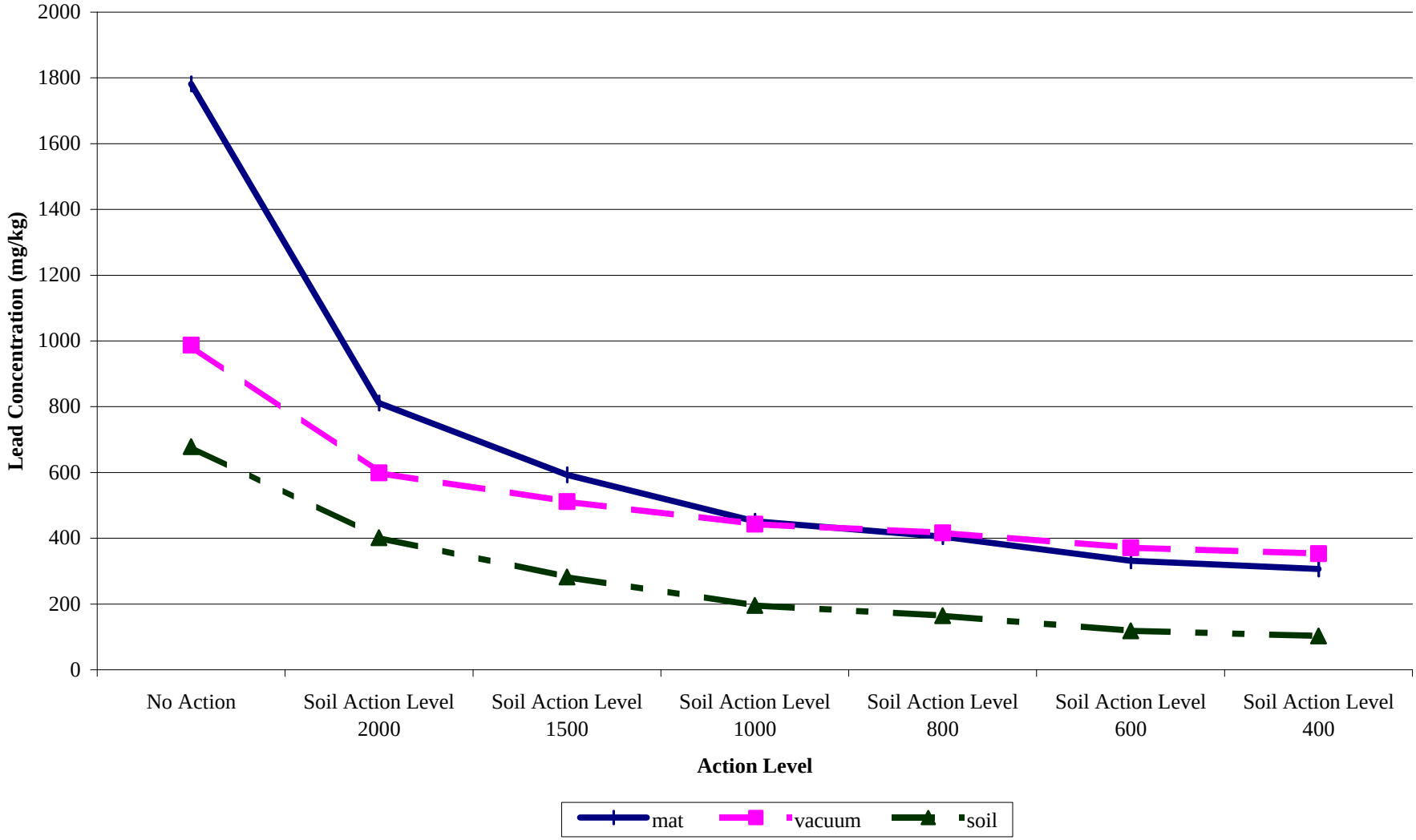


Figure 6-23c Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Wallace

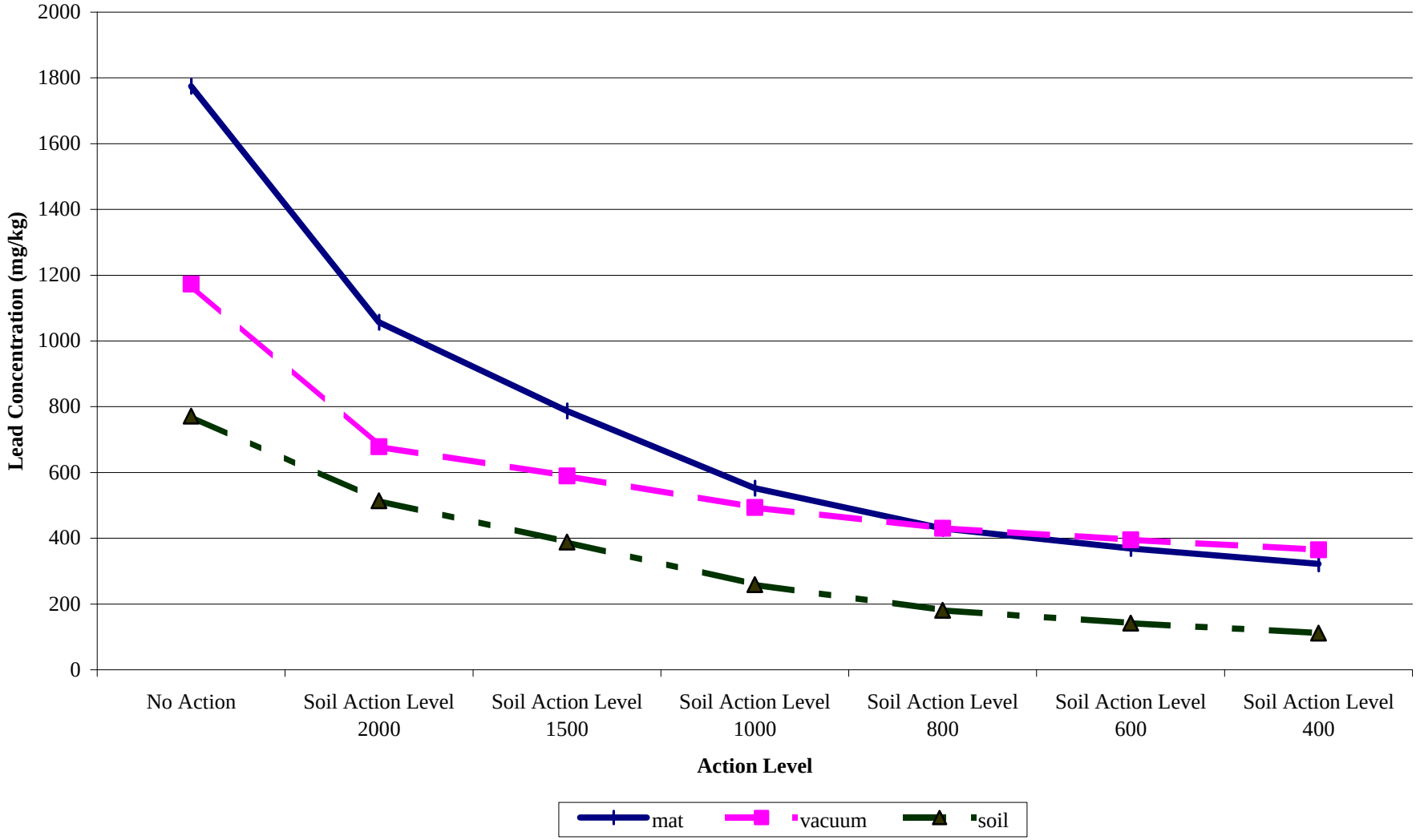


Figure 6-23d Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Silverton

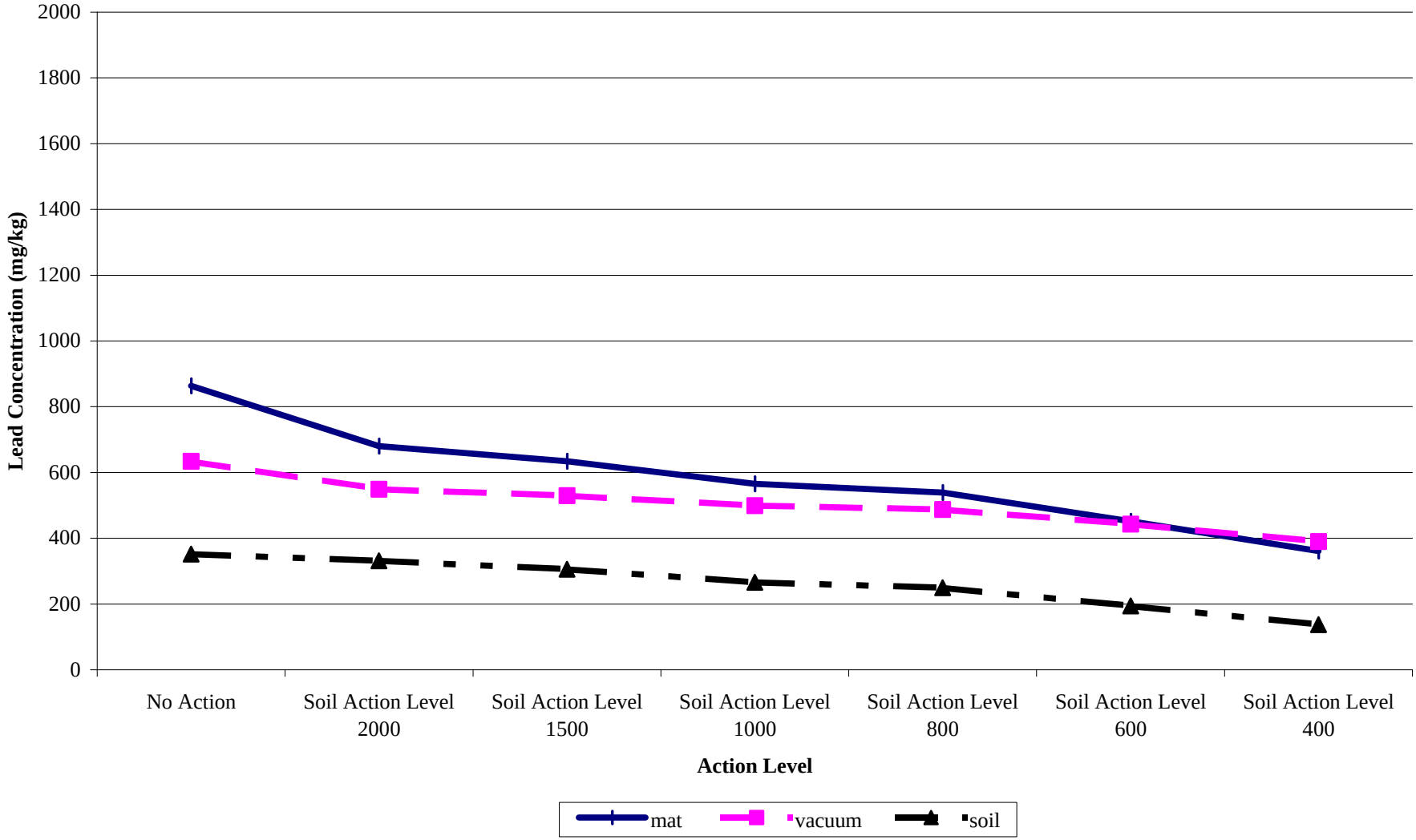


Figure 6-23e Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Osburn

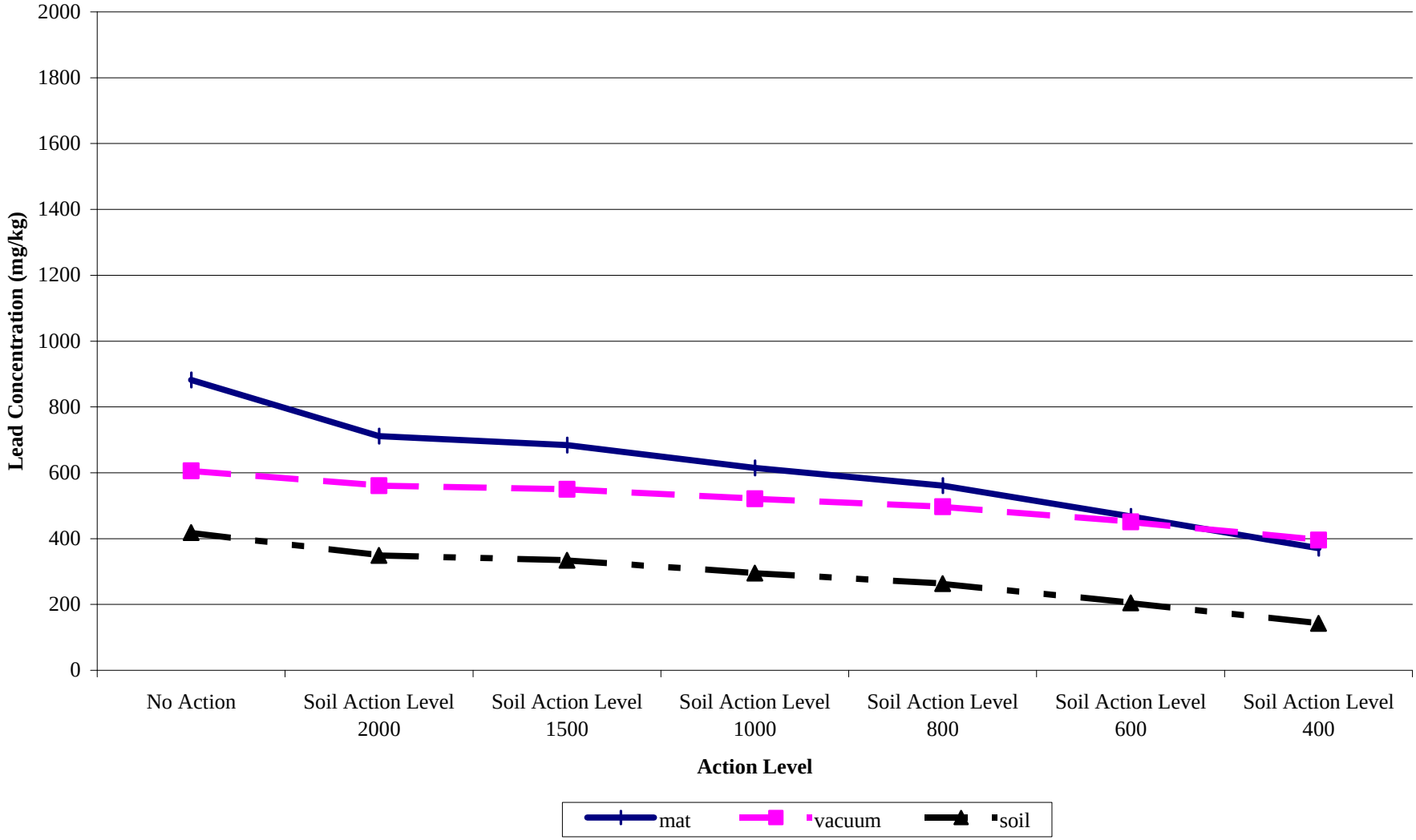


Figure 6-23f Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Side Gulches

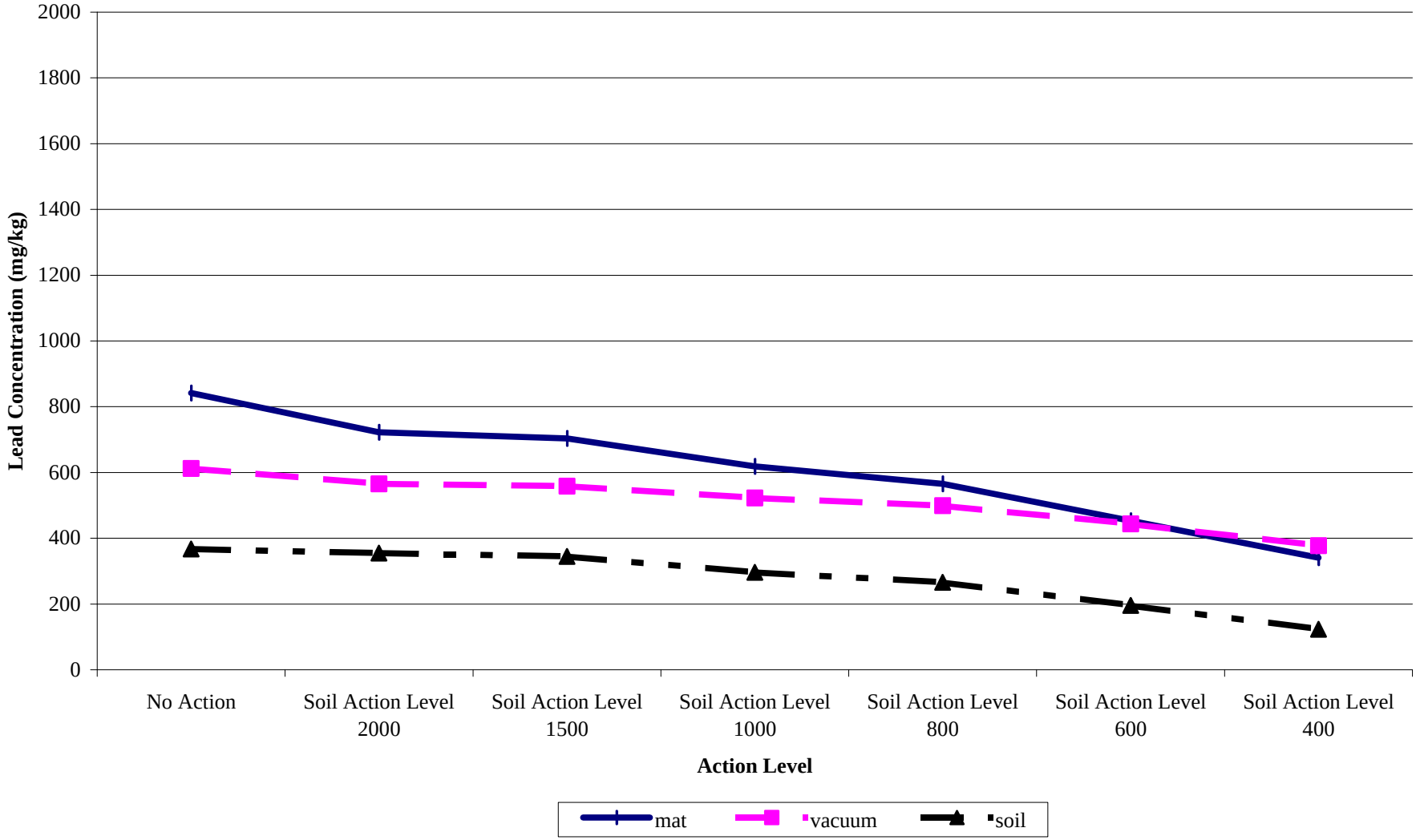


Figure 6-23g Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Kingston

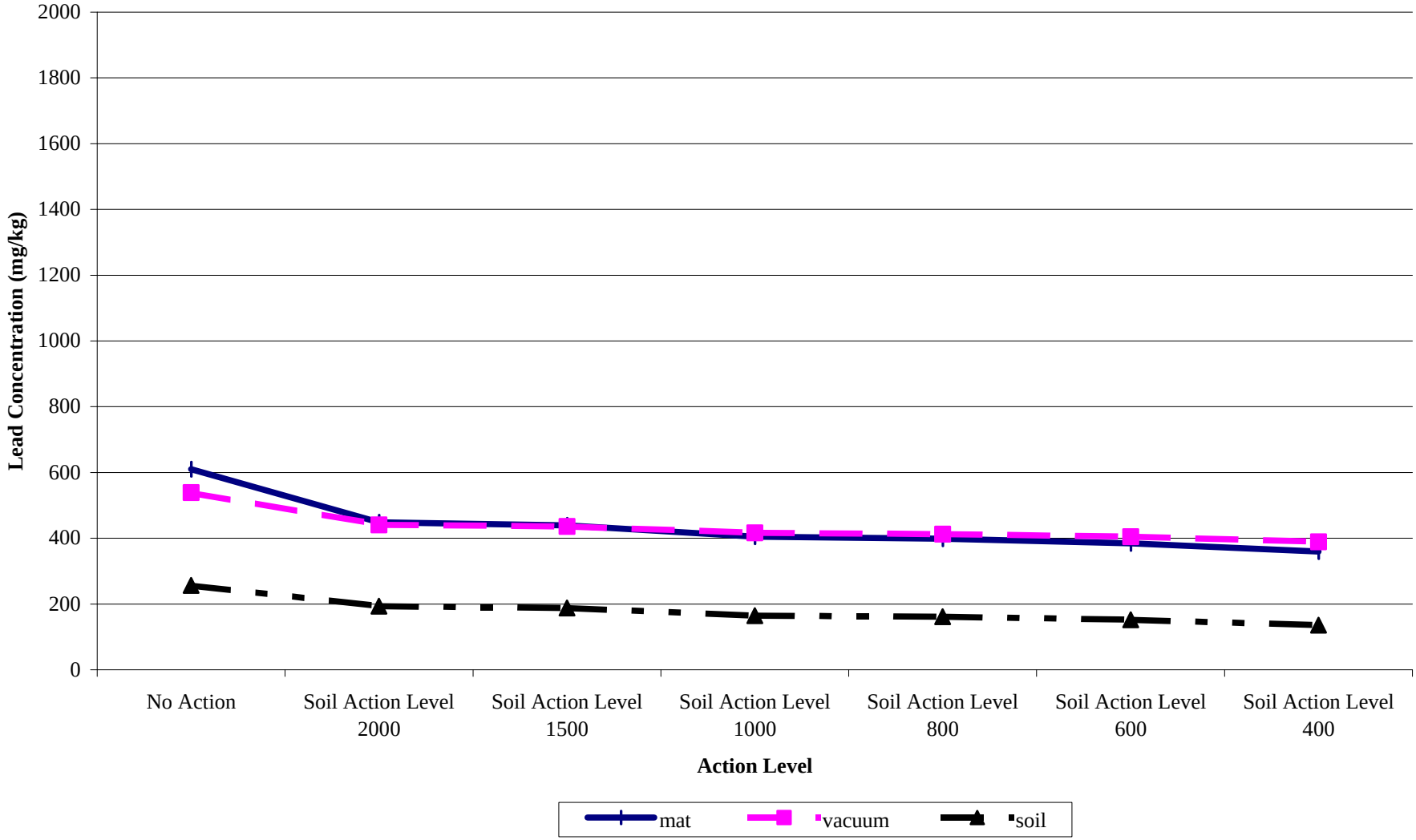


Figure 6-23h Estimated Community Geometric Mean Dust Mat, Vacuum, and Soil Lead Concentrations for Various Yard Soil Action Levels - Lower Basin

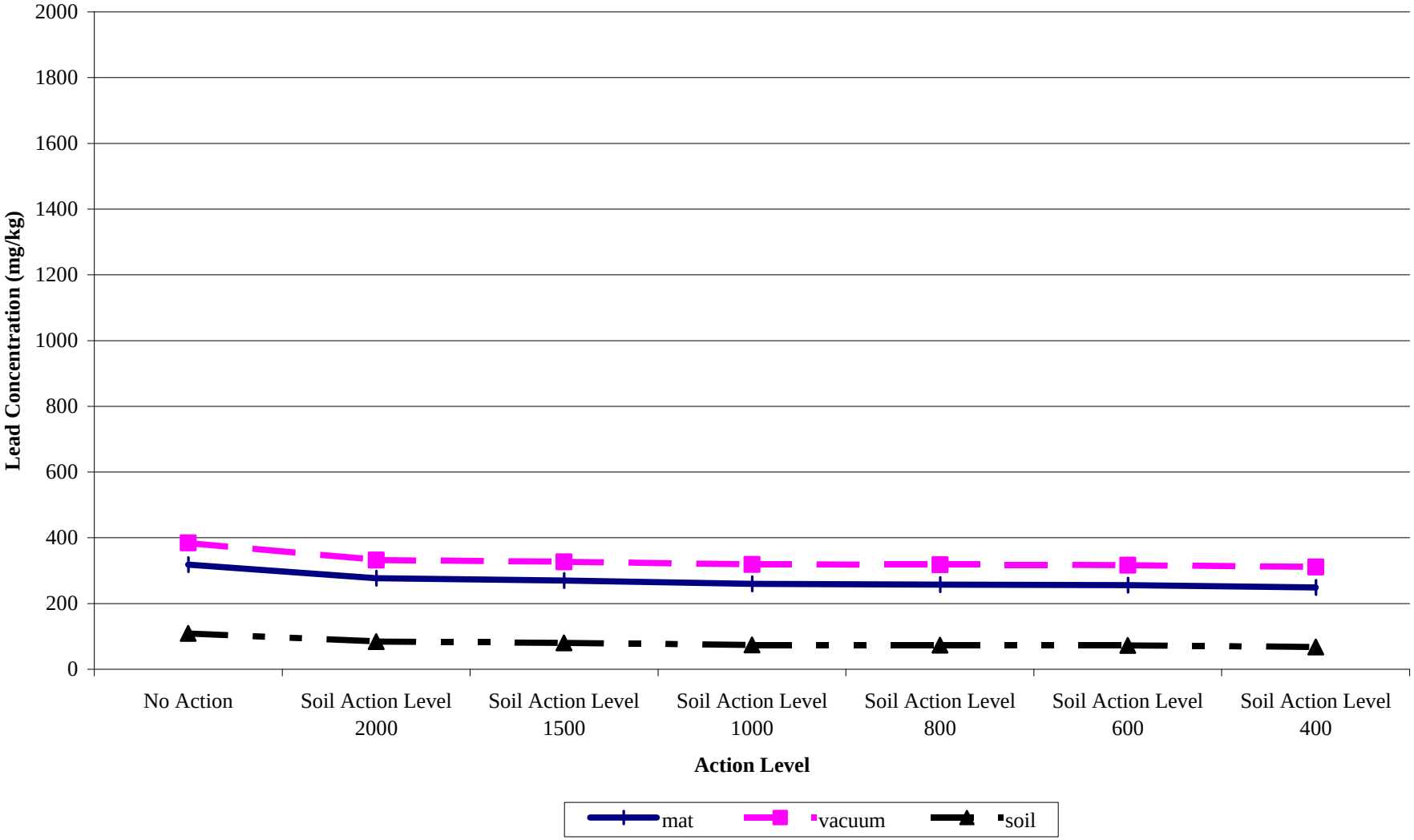
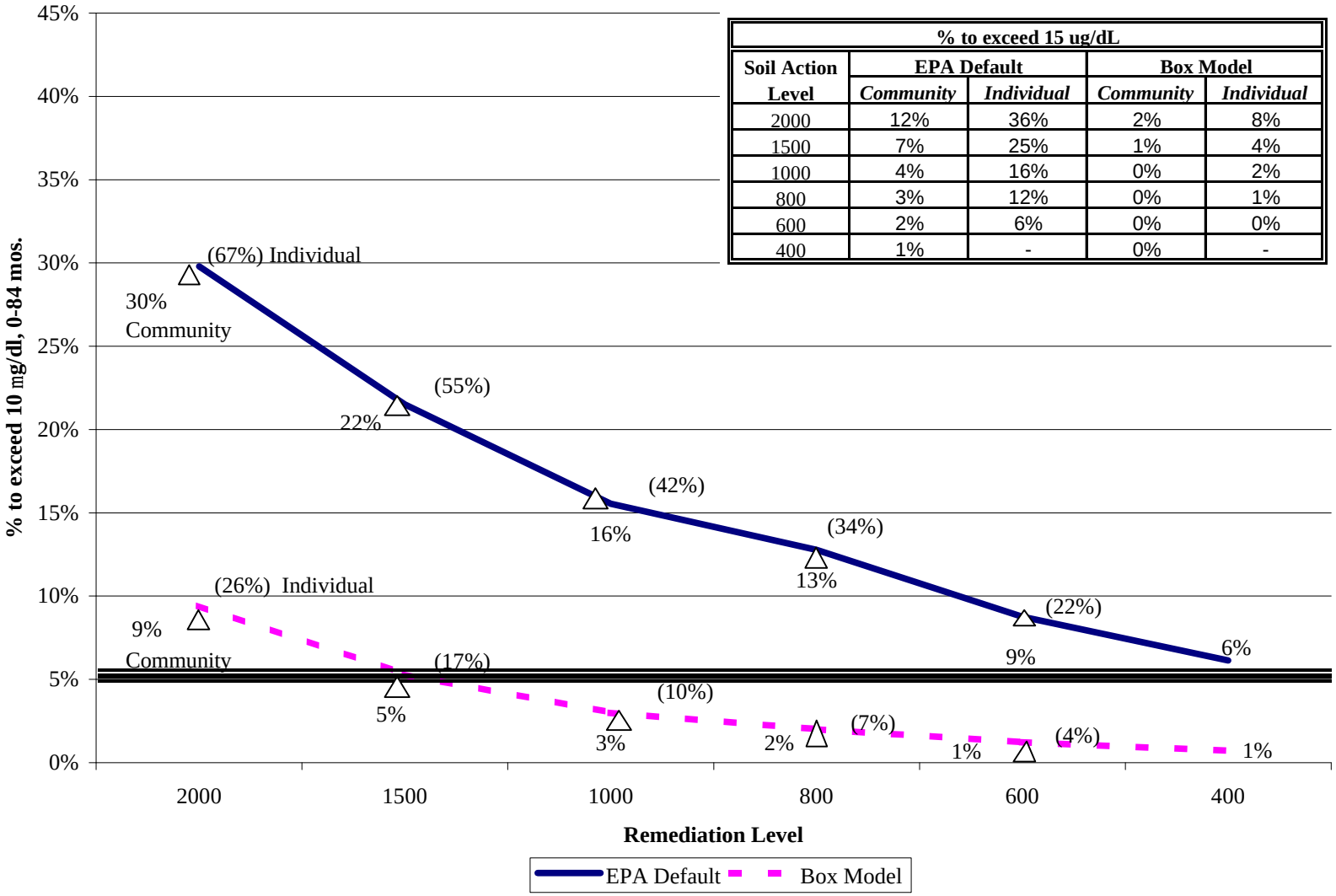
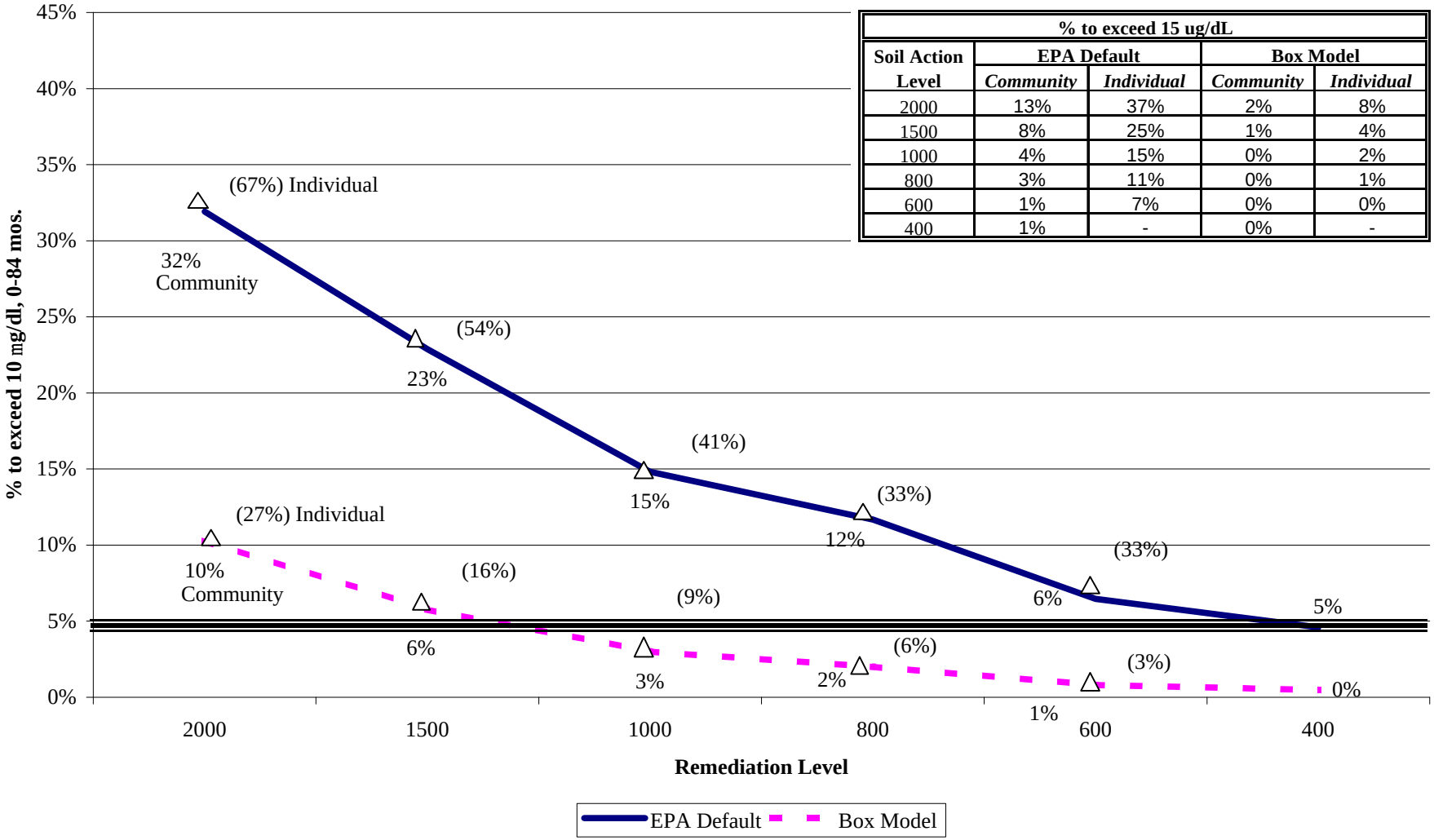


Figure 6-24a Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Mullan



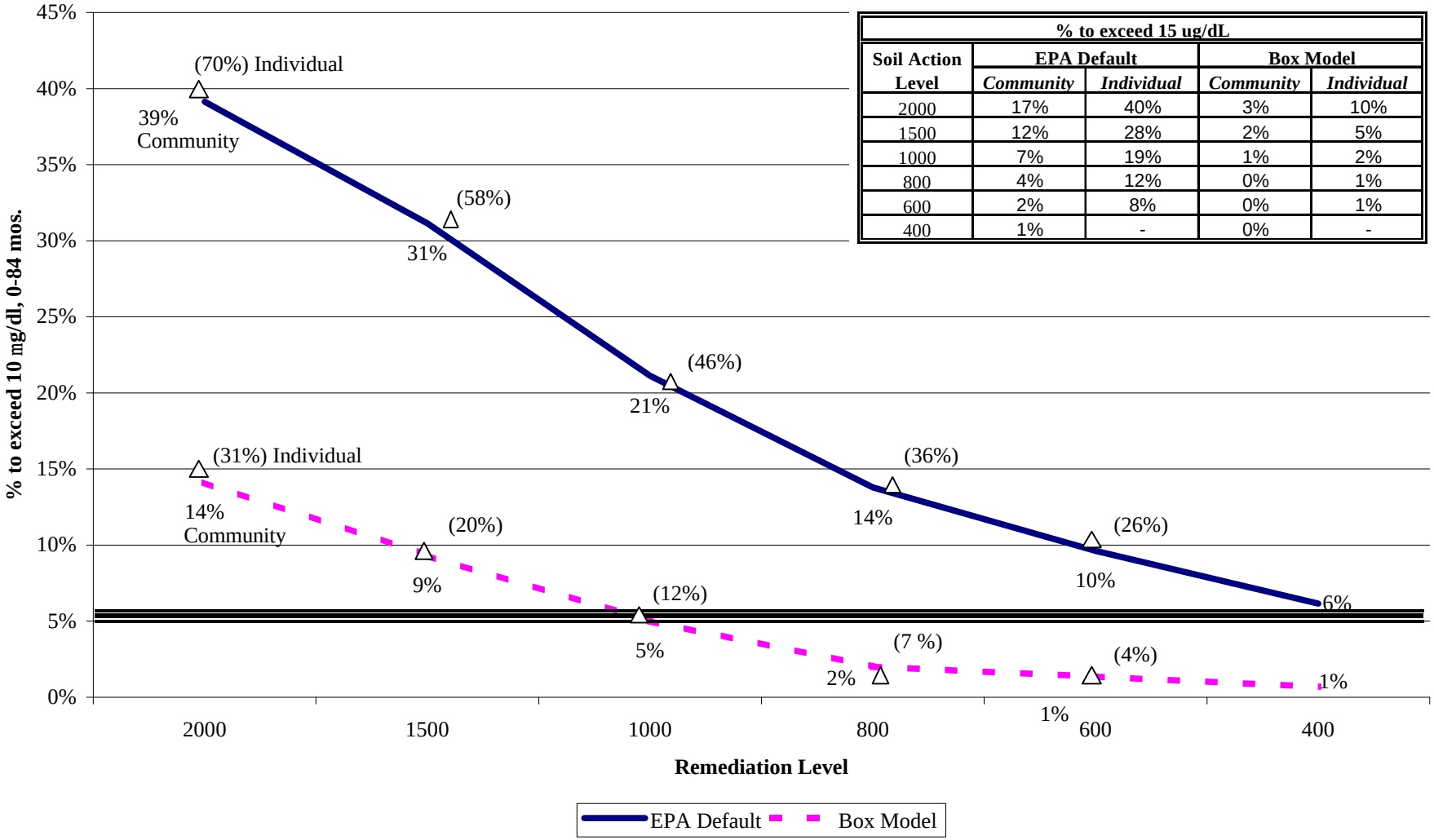
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	12%	36%	2%	8%
1500	7%	25%	1%	4%
1000	4%	16%	0%	2%
800	3%	12%	0%	1%
600	2%	6%	0%	0%
400	1%	-	0%	-

Figure 6-24b Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Burke/Nine Mile



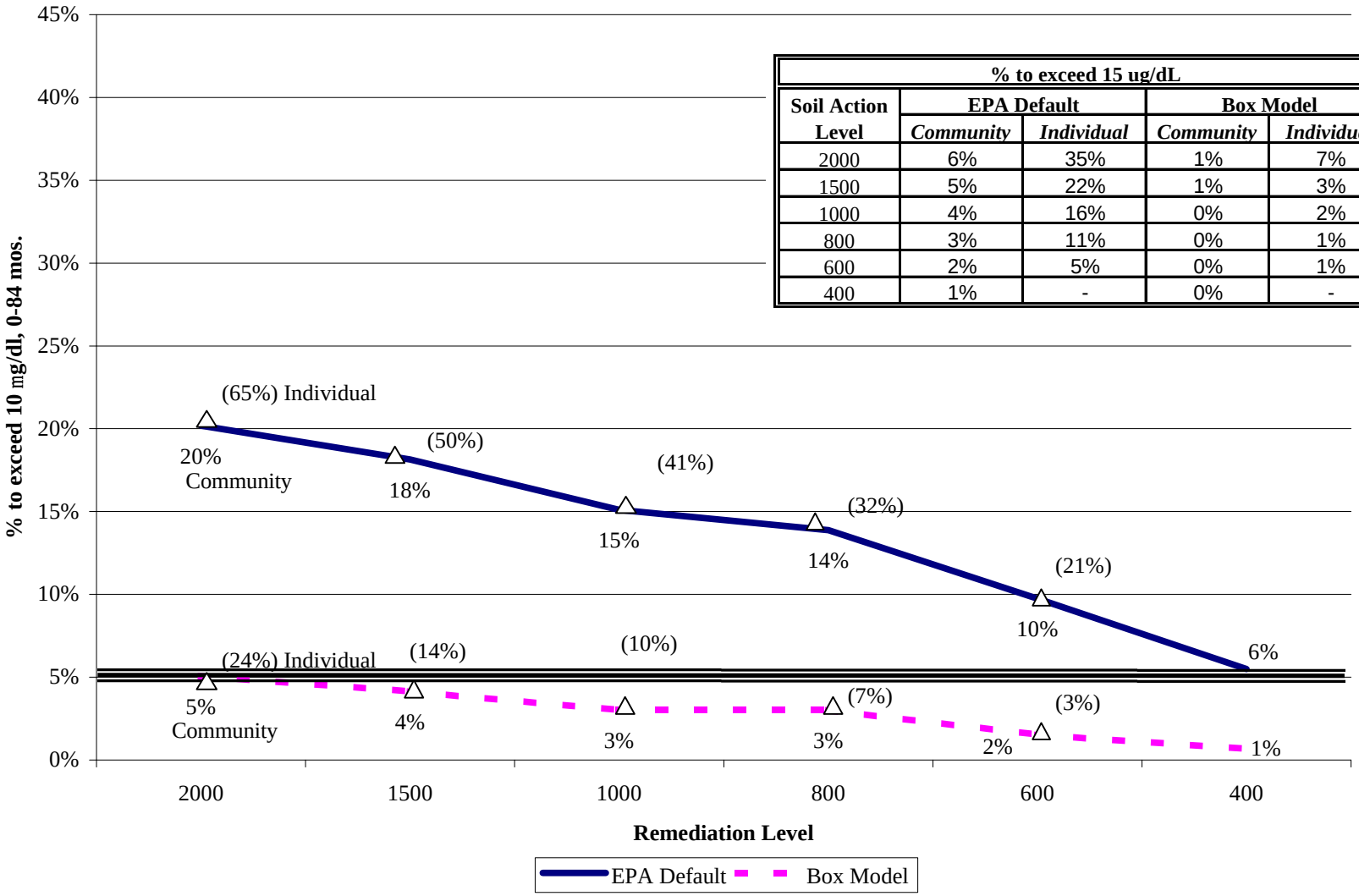
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	13%	37%	2%	8%
1500	8%	25%	1%	4%
1000	4%	15%	0%	2%
800	3%	11%	0%	1%
600	1%	7%	0%	0%
400	1%	-	0%	-

Figure 6-24c Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Wallace



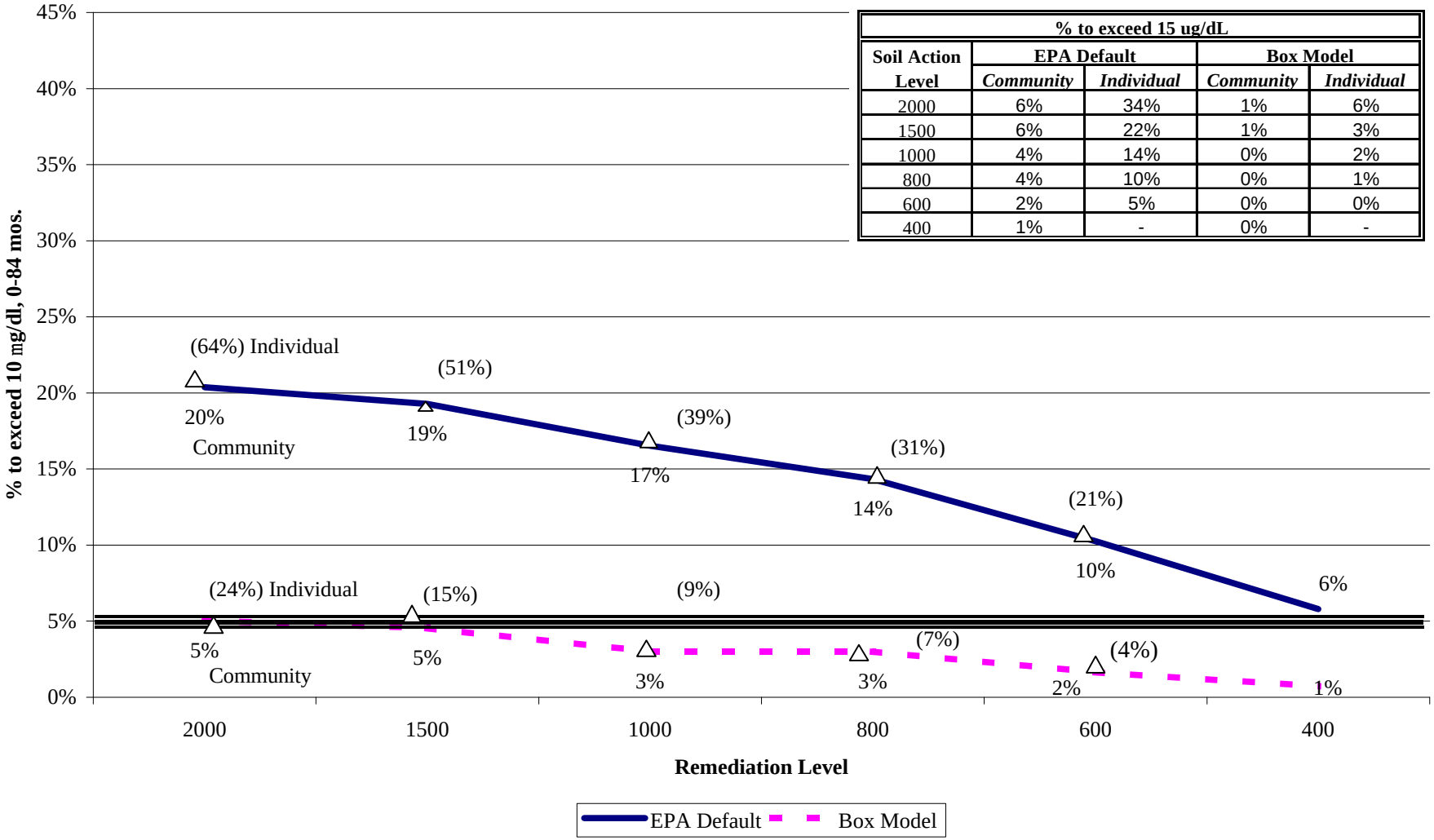
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	17%	40%	3%	10%
1500	12%	28%	2%	5%
1000	7%	19%	1%	2%
800	4%	12%	0%	1%
600	2%	8%	0%	1%
400	1%	-	0%	-

Figure 6-24d Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Silverton



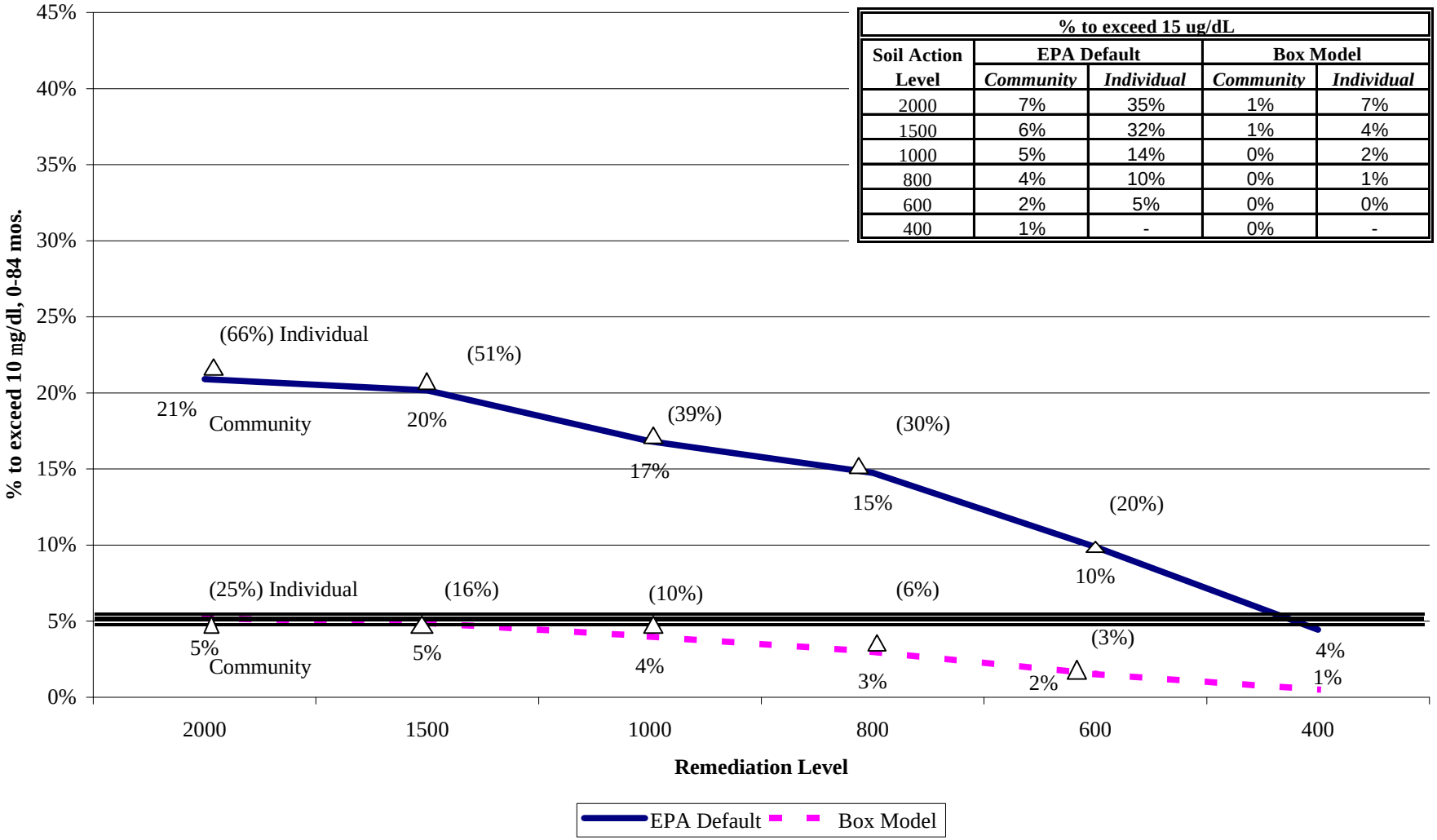
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	6%	35%	1%	7%
1500	5%	22%	1%	3%
1000	4%	16%	0%	2%
800	3%	11%	0%	1%
600	2%	5%	0%	1%
400	1%	-	0%	-

Figure 6-24e Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Osburn



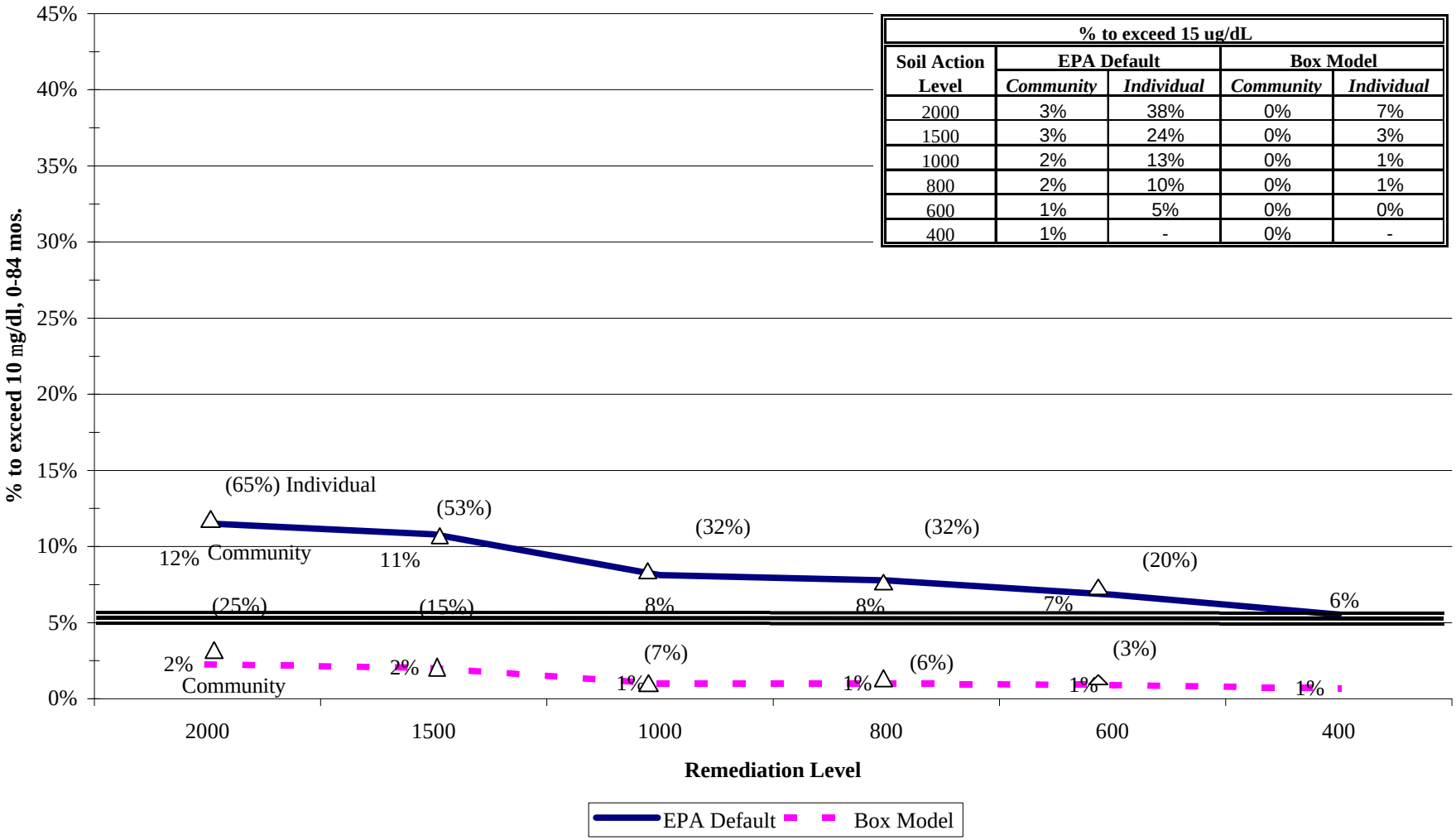
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	6%	34%	1%	6%
1500	6%	22%	1%	3%
1000	4%	14%	0%	2%
800	4%	10%	0%	1%
600	2%	5%	0%	0%
400	1%	-	0%	-

Figure 6-24f Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Side Gulches



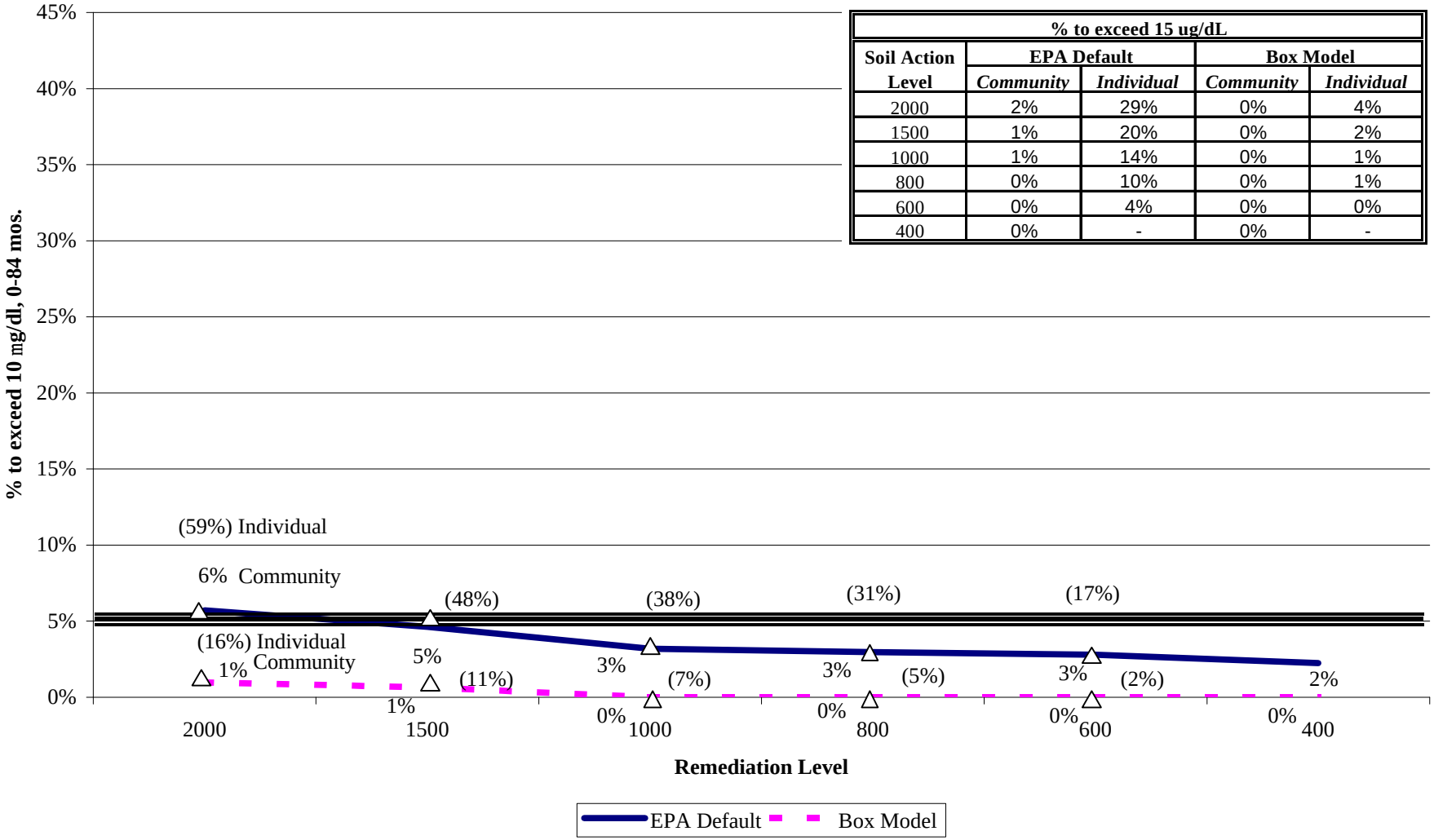
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	7%	35%	1%	7%
1500	6%	32%	1%	4%
1000	5%	14%	0%	2%
800	4%	10%	0%	1%
600	2%	5%	0%	0%
400	1%	-	0%	-

Figure 6-24g Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Kingston



% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	3%	38%	0%	7%
1500	3%	24%	0%	3%
1000	2%	13%	0%	1%
800	2%	10%	0%	1%
600	1%	5%	0%	0%
400	1%	-	0%	-

Figure 6-24h Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Lower Basin



% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	2%	29%	0%	4%
1500	1%	20%	0%	2%
1000	1%	14%	0%	1%
800	0%	10%	0%	1%
600	0%	4%	0%	0%
400	0%	-	0%	-

Table 6-1a
Summary of Basin Blood Lead Level Observations by Year
(9 Month Through 9 Year Old Children)

Year	N	Minimum (mg/dl)	Maximum (µg/dl)	Arithmetic Mean (µg/dl)	Geometric Mean (µg/dl)	# ≥ 10 mg/dl	% ≥ 10 mg/dl	# ≥ 15 mg/dl	% ≥ 15 mg/dl	# ≥ 20 mg/dl	% ≥ 20 mg/dl	# ≥ 25 mg/dl	% ≥ 25 mg/dl	Number of Followups Completed
1996	98	1	18	5.2	4.0	15	15.3%	6	6.1%	0	0.0%	0	0.0%	12
1997	26	1	19	5.5	4.2	4	15.4%	2	7.7%	0	0.0%	0	0.0%	3
1998	128	1	21	5.1	4.2	11	8.7%	4	3.1%	2	1.6%	0	0.0%	10
1999	272	1	29	5.3	4.2	28	10.3%	13	4.8%	4	1.5%	1	0.4%	25

Table 6-1b
Summary of the Number of Blood Lead Observations and Repeat Children

Total No. of Blood Lead Observations	524
Total No. of Individual Child Observations	424
Total No. of Households	247
Total No. of Children Tested More Than Once	81
Total No. of Households for Repeated Children	57
Total No. of Children Tested Twice	65
Total No. of Children Tested Three Times	13
Total No. of Children Tested All Four Years	3
Total No. of Blood Lead Observations for Repeated Children with Blood Leads ≥ 10 µg/dl	11

Table 6-2
Annual Blood Lead Summary Data by Geographic Subarea for Children (mg/dl)
(9 Months Through 9 Years)

Year	Number of Observations				Arithmetic Mean	Standard Deviation	Geometric Mean	Minimum	Maximum
	Total	≥10mg/dl	≥15mg/dl	≥20mg/dl					
Mullan Area									
1996	11	0	0	0	3.7	1.6	3.4	2	7
1997	-	-	-	-	-	-	-	-	-
1998	5	1	0	0	7.6	2.1	7.4	6	11
1999	22	3	0	0	5.3	2.9	4.7	2	12
Total	38	4	0	0	5.2	2.7	4.6	2	12
Burke/Nine Mile Area									
1996	17	6	3	0	8.3	4.9	6.6	1	17
1997	8	3	2	0	8.2	7.4	5.6	2	19
1998	18	4	3	2	7.5	6.4	5.5	2	21
1999	33	3	2	1	6.6	4.0	5.6	1	20
Total	76	16	10	3	7.4	5.2	5.8	1	21
Wallace Area									
1996	14	1	0	0	4.2	2.8	3.6	2	11
1997	1	-	-	-	2.0	-	-	2	2
1998	28	4	1	0	5.9	3.6	4.9	1	16
1999	34	5	3	2	6.8	5.8	5.4	2	29
Total	77	10	4	2	6.0	4.6	4.8	1	29
Silverton Area									
1996	14	2	1	0	5.5	3.8	4.6	2	16
1997	5	0	0	0	4.4	2.6	3.8	2	8
1998	26	0	0	0	4.1	1.8	3.7	1	8
1999	28	4	2	1	5.4	4.9	4.0	1	23
Total	73	6	3	1	4.9	3.7	4.0	1	23
Osburn Area									
1996	15	1	0	0	4.0	2.9	3.4	1	13
1997	7	0	0	0	3.8	1.9	3.4	1	7
1998	22	0	0	0	4.0	1.9	3.6	1	8
1999	56	3	0	0	4.0	2.5	3.3	1	11
Total	100	4	0	0	4.1	2.4	3.4	1	13
Side Gulches Area									
1996	8	0	0	0	2.7	1.0	2.5	1	4
1997	-	-	-	-	-	-	-	-	-
1998	12	1	0	0	5.0	3.1	4.5	3	14
1999	31	1	1	0	4.3	2.9	3.6	1	16
Total	51	2	1	0	4.3	2.8	3.6	1	16

Table 6-2 (continued)
Annual Blood Lead Summary Data by Geographic Subarea for Children (mg/dl)
(9 Months Through 9 Years)

Year	Number of Observations				Arithmetic Mean	Standard Deviation	Geometric Mean	Minimum	Maximum
	Total	≥10mg/dl	≥15mg/dl	≥20mg/dl					
Kingston Area									
1996	7	1	1	0	6.4	4.7	5.2	2	16
1997	-	-	-	-	-	-	-	-	-
1998	8	0	0	0	2.8	1.9	2.4	1	7
1999	39	5	3	0	4.8	4.2	3.6	1	16
Total	54	6	4	0	4.8	4.0	3.6	1	16
Lower Basin/Cataldo Area									
1996	12	4	1	0	5.2	5.6	3.1	1	18
1997	5	1	0	0	5.4	4.0	4.4	2	12
1998	9	1	0	0	3.5	3.8	2.6	1	13
1999	29	4	2	0	6.2	4.2	5.0	1	18
Total	55	10	3	0	5.5	4.4	4.0	1	18

Table 6-3 Geographic Subarea Blood Lead Summary Data for All Children (1 year through 9 years old) For All Years (mg/dl)

Area	Total	# ≥10mg/dl	% ≥10mg/dl	# ≥15mg/dl	% ≥15mg/dl	# ≥20mg/dl	% ≥20mg/dl	Arithmetic Mean	Geometric Mean	Standard Deviation	Geometric Standard Deviation	Minimum	Maximum
Mullan	38	4	11%	-	-	-	-	5.2	4.6	2.7	1.7	2	12
Burke/Nine Mile	76	16	21%	10	13%	3	4%	7.4	5.8	5.2	2.1	1	21
Wallace	77	10	13%	4	5%	2	3%	6.0	4.8	4.6	1.9	1	29
Silverton	73	6	8%	3	4%	1	1%	4.9	4.0	3.7	1.9	1	23
Osburn	100	4	4%	-	-	-	-	4.1	3.4	2.4	1.8	1	13
Side Gulches	51	2	4%	1	2%	-	-	4.3	3.6	2.8	1.8	1	16
Kingston	54	6	11%	4	7%	-	-	4.8	3.6	4.0	2.2	1	16
Lower Basin/ Cataldo	55	10	18%	3	5%	-	-	5.5	4.0	4.4	2.3	1	18
Total	524	58	11%	25	5%	6	1%	-	-	-	-	-	-

Table 6-4a Basin Blood Lead Levels for 1-9 Year Old Children, All Years Combined (mg/dl)

Age (years)	N	Percent Participation	Cumulative Percent	Mininum	Maximum	Arithmetic Mean	Standard Deviation	Geometric Mean	# ≥ 10 mg/dl	% ≥ 10	# ≥ 15 mg/dl	% ≥ 15 mg/dl
1	40	7.6%	7.6%	2	16	7.0	3.6	6.2	8	20%	2	5%
2	46	8.8%	16.4%	2	29	8.0	6.3	6.3	12	26%	8	17%
3	52	9.9%	26.3%	1	21	6.5	4.5	5.2	10	19%	4	8%
4	57	10.9%	37.2%	1	21	5.5	4.2	4.4	7	12%	3	5%
5	62	11.8%	49.0%	1	16	5.4	3.0	4.7	5	8%	2	3%
6	46	8.8%	57.8%	1	20	4.8	3.3	4.0	3	7%	1	2%
7	58	11.1%	68.9%	1	15	4.3	2.8	3.5	3	5%	1	2%
8	72	13.7%	82.6%	1	17	3.9	3.1	3.0	4	6%	1	1%
9	91	17.4%	100.0%	1	19	4.1	3.4	3.2	6	7%	3	3%
All	524	100%		1	29	5.3	4.0	4.2	58	11%	25	5%

Table 6-4b Basin Blood Lead Levels for 1-6 Year Old Children, All Years Combined (mg/dl)

Age (years)	N	Percent Participation	Cumulative Percent	Mininum	Maximum	Arithmetic Mean	Standard Deviation	Geometric Mean	# ≥ 10 mg/dl	% ≥ 10	Age-Adjusted % ≥ 10	# ≥ 15 mg/dl	% ≥ 15 mg/dl	Age-Adjusted % > 15
1	40	13.2%	13.2%	2	16	7.0	3.6	6.2	8	20.0%	25.3%	2	5.0%	6.3%
2	46	15.2%	28.4%	2	29	8.0	6.3	6.3	12	26.1%	28.6%	8	17.4%	19.1%
3	52	17.2%	45.5%	1	21	6.5	4.5	5.2	10	19.2%	18.7%	4	7.7%	7.5%
4	57	18.8%	64.4%	1	21	5.5	4.2	4.4	7	12.3%	10.9%	3	5.3%	4.7%
5	62	20.5%	84.8%	1	16	5.4	3.0	4.7	5	8.1%	6.6%	2	3.2%	2.6%
6	46	15.2%	100.0%	1	20	4.8	3.3	4.0	3	6.5%	7.2%	1	2.2%	2.4%
All	303	100%		1	29	5.3	4.0	4.2	45	14.9%	16.2%	20	6.6%	7.1%

Table 6-4c Basin Blood Lead Levels for 1-7 Year Old Children, All Years Combined (mg/dl)

Age (years)	N	Percent Participation	Cumulative Percent	Mininum	Maximum	Arithmetic Mean	Standard Deviation	Geometric Mean	# ≥ 10 mg/dl	% ≥ 10	Age-Adjusted % ≥ 10	# ≥ 15 mg/dl	% ≥ 15 mg/dl	Age-Adjusted % > 15
1	40	11.1%	11.1%	2	16	7.0	3.6	6.2	8	20.0%	25.8%	2	5.0%	6.4%
2	46	12.7%	23.8%	2	29	8.0	6.3	6.3	12	26.1%	29.2%	8	17.4%	19.5%
3	52	14.4%	38.2%	1	21	6.5	4.5	5.2	10	19.2%	19.1%	4	7.7%	7.6%
4	57	15.8%	54.0%	1	21	5.5	4.2	4.4	7	12.3%	11.1%	3	5.3%	4.8%
5	62	17.2%	71.2%	1	16	5.4	3.0	4.7	5	8.1%	6.7%	2	3.2%	2.7%
6	46	12.7%	83.9%	1	20	4.8	3.3	4.0	3	6.5%	7.3%	1	2.2%	2.4%
7	58	16.1%	100.0%	1	15	4.3	2.8	3.5	3	5.2%	4.6%	1	2.0%	1.8%
All	361	100.0%		1	29	5.3	4.0	4.2	45	12.5%	14.8%	20	5.5%	6.5%

Table 6-5 Basin Blood Lead Levels by Geographic Area and Age (mg/dl)

	Kingston	Lower Basin	Mullan	Burke/ Nine Mile	Osburn	Side Gulches	Silverton	Wallace	Basin wide
Age 1									
N	3	5	4	4	9	3	6	6	40
Minimum	2	4	5	2	4	2	4	8	2
Maximum	5	14	9	9	11	7	16	16	16
Arithmetic Mean	3.7	7.0	6.5	5.3	6.6	4.3	7.7	11.7	7.0
Standard Deviation	1.53	4.12	1.73	2.87	2.19	2.52	4.41	2.66	3.6
Geometric Mean	3.4	6.2	6.3	4.6	6.2	3.8	6.8	11.4	6.2
GSD	1.61	1.67	1.28	1.86	1.39	1.87	1.65	1.26	1.7
# ≥ 10	0	1	0	0	1	0	1	5	8.0
% ≥ 10	0%	20%	0%	0%	11%	0%	17%	83%	20%
# ≥ 15	0	0	0	0	0	0	1	1	2.0
% ≥ 15	0%	0%	0%	0%	0%	0%	17%	17%	5%
Age 2									
N	5	4	2	6	5	7	10	7	46
Minimum	2	3	5	2	2	4	3	2	2
Maximum	15	18	11	20	10	9	23	29	29
Arithmetic Mean	9.0	9.8	8.0	11.7	6.4	5.9	7.4	7.6	8.0
Standard Deviation	5.43	6.65	4.24	8.50	3.05	1.86	6.55	9.52	6.3
Geometric Mean	7.3	7.9	7.4	8.4	5.6	5.6	5.7	5.1	6.3
GSD	2.25	2.20	1.75	2.67	1.88	1.36	2.01	2.32	2.0
# ≥ 10	2	2	1	3	1	0	2	1	12.0
% ≥ 10	40%	50%	50%	50%	20%	0%	20%	14%	26%
# ≥ 15	1	1	0	3	0	0	2	1	8.0
% ≥ 15	20%	25%	0%	50%	0%	0%	20%	14%	17%
Age 3									
N	9	10	1	4	4	7	9	8	52
Minimum	1	1	5	6	2	1	2	3	1
Maximum	16	18	5	21	6	7	10	19	21
Arithmetic Mean	6.4	7.1	5.0	12.3	3.8	3.9	5.7	7.9	6.5
Standard Deviation	4.50	5.00	0.00	6.50	2.06	1.77	2.96	4.97	4.5
Geometric Mean	5.1	5.5	5.0	11.0	3.3	3.4	4.9	6.8	5.2
GSD	2.20	2.32	1.00	1.70	1.80	1.82	1.81	1.73	2.0
# ≥ 10	2	2	0	2	0	0	2	2	10.0
% ≥ 10	22%	20%	0%	50%	0%	0%	22%	25%	19%
# ≥ 15	1	1	0	1	0	0	0	1	4.0
% ≥ 15	11%	10%	0%	25%	0%	0%	0%	13%	8%
Age 4									
N	11	5	1	5	12	4	12	7	57
Minimum	1	2	12	3	1	3	2	2	1
Maximum	16	13	12	18	11	5	8	21	21
Arithmetic Mean	4.4	6.2	12.0	8.0	3.6	3.8	5.0	9.1	5.5
Standard Deviation	4.15	4.32	0.00	6.12	2.54	0.96	1.71	6.15	4.2
Geometric Mean	3.3	5.0	12.0	6.4	3.0	3.7	4.7	7.5	4.4
GSD	2.04	2.09	1.00	2.14	1.79	1.28	1.54	2.07	2.0
# ≥ 10	1	1	1	1	1	0	0	2	7.0
% ≥ 10	9%	20%	100%	20%	8%	0%	0%	29%	12%
# ≥ 15	1	0	0	1	0	0	0	1	3.0
% ≥ 15	9%	0%	0%	20%	0%	0%	0%	14%	5%
Age 5									
N	3	4	4	13	13	4	10	11	62
Minimum	4	3	2	2	1	2	2	2	1
Maximum	16	16	6	10	8	3	12	9	16
Arithmetic Mean	9.3	9.5	4.3	5.7	4.9	2.8	4.6	5.1	5.4
Standard Deviation	6.11	5.32	2.06	2.25	1.98	0.50	3.10	1.97	3.0
Geometric Mean	8.0	8.1	3.8	5.3	4.4	2.7	3.9	4.7	4.7
GSD	2.00	2.03	1.72	1.54	1.78	1.22	1.83	1.52	1.7
# ≥ 10	1	2	0	1	0	0	1	0	5.0
% ≥ 10	33%	50%	0%	8%	0%	0%	10%	0%	8%
# ≥ 15	1	1	0	0	0	0	0	0	2.0
% ≥ 15	33%	25%	0%	0%	0%	0%	0%	0%	3%

Table 6-5 Basin Blood Levels by Geographic Area and Age mg/dl (continued)

	Kingston	Lower Basin	Mullan	Burke/ Nine Mile	Osburn	Side Gulches	Silverton	Wallace	Basin wide
Age 6									
N	3	7	5	7	9	7	4	4	46
Minimum	1	3	2	4	2	1	2	2	1
Maximum	5	11	6	20	6	6	8	8	20
Arithmetic Mean	2.7	4.7	3.8	8.6	3.9	3.9	4.0	6.0	4.8
Standard Deviation	2.08	2.98	1.64	5.86	1.36	1.86	2.71	2.83	3.3
Geometric Mean	2.2	4.2	3.5	7.3	3.7	3.4	3.5	5.3	4.0
GSD	2.24	1.65	1.55	1.79	1.44	1.87	1.80	1.93	1.8
# ≥ 10	0	1	0	2	0	0	0	0	3.0
% ≥ 10	0%	14%	0%	29%	0%	0%	0%	0%	7%
# ≥ 15	0	0	0	1	0	0	0	0	1.0
% ≥ 15	0%	0%	0%	14%	0%	0%	0%	0.00	2%
Age 7									
N	9	5	4	11	10	3	5	11	58
Minimum	1	1	4	1	1	2	2	1	1
Maximum	5	10	7	15	9	4	4	7	15
Arithmetic Mean	2.3	3.8	5.5	7.2	3.9	3.3	2.6	4.3	4.3
Standard Deviation	1.41	3.83	1.73	3.82	2.28	1.15	0.89	1.62	2.8
Geometric Mean	2.0	2.5	5.3	6.0	3.3	3.2	2.5	3.9	3.5
GSD	1.83	2.76	1.38	2.06	1.87	1.49	1.37	1.68	2.0
# ≥ 10	0	1	0	2	0	0	0	0	3.0
% ≥ 10	0%	20%	0%	18%	0%	0%	0%	0%	5%
# ≥ 15	0	0	0	1	0	0	0	0	1.0
% ≥ 15	0%	0%	0.00	9%	0%	0%	0%	0%	2%
Age 8									
N	5	7	10	9	17	6	8	10	72
Minimum	1	1	2	1	1	1	1	1	1
Maximum	8	5	11	17	13	14	8	9	17
Arithmetic Mean	3.6	2.1	4.5	4.9	3.8	4.7	3.0	4.0	3.9
Standard Deviation	2.70	1.46	2.72	5.01	2.74	4.68	2.27	2.45	3.1
Geometric Mean	2.9	1.8	3.9	3.4	3.2	3.4	2.4	3.3	3.0
GSD	2.17	1.87	1.70	2.35	1.88	2.33	2.01	1.96	2.0
# ≥ 10	0	0	1	1	1	1	0	0	4.0
% ≥ 10	0%	0%	10%	11%	6%	17%	0%	0%	6%
# ≥ 15	0	0	0	1	0	0	0	0	1.0
% ≥ 15	0%	0%	0%	11%	0%	0%	0%	0%	1%
Age 9									
N	6	8	7	17	21	10	9	13	91
Minimum	2	1	2	2	1	1	1	2	1
Maximum	6	4	10	19	5	16	8	7	19
Arithmetic Mean	3.5	2.6	5.0	7.3	2.5	4.6	3.4	3.3	4.1
Standard Deviation	1.38	1.19	3.06	5.06	1.21	4.35	2.30	1.32	3.4
Geometric Mean	3.3	2.4	4.3	5.7	2.2	3.4	2.8	3.1	3.2
GSD	1.44	1.63	1.83	2.11	1.71	2.18	2.04	1.41	2.0
# ≥ 10	0	0	1	4	0	1	0	0	6.0
% ≥ 10	0%	0%	14%	24%	0%	10%	0%	0%	7%
# ≥ 15	0	0	0	2	0	1	0	0	3.0
% ≥ 15	0%	0%	0%	12%	0%	10%	0%	0%	3%

Table 6-6a Basin-wide Blood Lead Level Summary for 1 Through 6 Year Old Children by Area (Survey Results-All Years)

Area	N	Minimum	Maximum	Arithmetic Mean	Standard Deviation	Geometric Mean	Geometric Standard Deviation	# ≥10 mg/dl	% ≥10 mg/dl	# ≥15 mg/dl	% ≥15 mg/dl
Mullan	17	2	12	5.6	2.9	4.9	1.7	2	12%	0	0%
Burke/Nine Mile	39	2	21	8.1	5.6	6.5	1.9	9	23%	6	15%
Wallace	43	2	29	7.7	5.4	6.3	1.9	10	23%	4	9%
Silverton	51	2	23	5.7	3.9	4.8	1.8	6	12%	3	6%
Osburn	52	1	11	4.8	2.4	4.2	1.7	3	6%	0	0%
Side Gulches	32	1	9	4.2	1.9	3.8	1.7	0	0%	0	0%
Kingston	34	1	16	5.8	4.6	4.4	2.2	6	18%	4	12%
Lower Basin	35	1	18	7.1	4.6	5.7	2.0	9	26%	3	9%
Total	303	1	29	6.1	4.3	5.0	1.9	45	15%	20	7%

Table 6-6b Basin-wide Blood Lead Level Summary for 1-7 Year Old Children by Area (Survey Results-All Years)

Area	N	Minimum	Maximum	Arithmetic Mean	Standard Deviation	Geometric Mean	Geometric Standard Deviation	# ≥10 mg/dl	% ≥10 mg/dl	# ≥15 mg/dl	% ≥15 mg/dl
Mullan	21	2	12	5.6	2.6	5.0	1.6	2	10%	0	0%
Burke/Nine Mile	50	1	21	7.9	5.2	6.4	1.9	11	22%	7	14%
Wallace	54	1	29	7.0	5.1	5.7	1.9	10	19%	4	7%
Silverton	56	2	23	5.5	3.9	5.4	1.8	6	11%	3	5%
Osburn	62	1	11	4.6	2.4	4.0	1.8	3	5%	0	0%
Side Gulches	35	1	9	4.1	1.8	3.7	1.6	0	0%	0	0%
Kingston	43	1	16	5.1	4.4	3.7	2.3	6	14%	4	9%
Lower Basin	40	1	18	6.7	4.6	5.1	2.2	10	25%	3	8%
Total	361	1	29	5.8	4.2	4.7	1.9	48	13%	21	6%

Table 6-7 Comparison of Coeur d'Alene Basin and BHSS Results to National and State-Wide Blood Lead Levels*

		Blood Lead Concentrations		Percent
		Arithmetic Mean (mg/dl)	Geometric Mean (mg/dl)	≥ 10 mg/dl
Basin 1999	**(1-6 year olds)**	6.4	5.2	16.0%
BHSS 1999	**(1-6 year olds)**	5.0	4.2	7.8%
State-wide (1997)	**(1-6 year olds in high risk housing)**	3.7	NR	4.2%
National (1991-1994)				
Low Income	**Pre-1946 Housing**	NR	5.5	16.4%
	1946-1973 Housing	NR	3.6	7.3%
	Post 1973 Housing	NR	3.0	4.3%
Middle Income	**Pre-1946 Housing**	NR	2.9	4.1%
	1946-1973 Housing	NR	2.4	2.0%
	Post 1973 Housing	NR	1.9	0.4%
White (Non-Hispanic)	**Pre-1946 Housing**	NR	3.3	5.6%
	1946-1973 Housing	NR	2.4	1.4%
	Post 1973 Housing	NR	1.8	1.5%

*Source: Morbidity and Mortality, *Weekly Reports*, February 21, 1999; Pirkle et al. 1998; IDHW 1998
NR = not reported

Table 6-8a Summary of Adult Blood Lead Levels by Age and Geographic Subarea (mg/dl)

	BOTH MALES AND FEMALES			
	10 to 25	26-40	41-49	Age >50
Mullan				
N	12	16	9	31
Minimum	1	1	2	1
Maximum	9	13	9	14
Arithmetic Mean	2.8	3.6	3.6	4.6
Standard Deviation	2.08	3.07	2.46	3.56
Geometric Mean	2.4	2.7	3.0	3.4
Geometric Standard Deviation	1.70	2.18	1.77	2.25
# ≥10 mg/dl	0	1	0	4
% ≥10 mg/dl	0%	6%	0%	13%
# ≥15 mg/dl	0	0	0	0
% ≥15 mg/dl	0%	0%	0%	0%
Burke/Nine Mile				
N	13	17	8	28
Minimum	1	1	1	1
Maximum	10	11	4	33
Arithmetic Mean	3.7	4.7	2.4	6.0
Standard Deviation	2.25	3.31	1.19	6.30
Geometric Mean	3.2	3.5	2.1	4.2
Geometric Standard Deviation	1.77	2.29	1.72	2.32
# ≥10 mg/dl	1	3	0	3
% ≥10 mg/dl	8%	18%	0%	11%
# ≥15 mg/dl	0	0	0	2
% ≥15 mg/dl	0%	0%	0%	7%
Wallace				
N	19	18	7	24
Minimum	1	1	1	1
Maximum	5	16	33	14
Arithmetic Mean	2.2	4.1	14.4	3.7
Standard Deviation	1.08	3.99	13.45	2.61
Geometric Mean	2.0	2.9	8.1	3.1
Geometric Standard Deviation	1.68	2.24	3.71	1.79
# ≥10 mg/dl	0	2	3	1
% ≥10 mg/dl	0%	11%	43%	4%
# ≥15 mg/dl	0	1	3	0
% ≥15 mg/dl	0%	6%	43%	0%
Silverton				
N	4	17	6	11
Minimum	1	1	1	1
Maximum	3	7	5	10
Arithmetic Mean	2.0	2.4	1.8	3.1
Standard Deviation	0.82	1.62	1.60	2.43
Geometric Mean	1.9	2.0	1.5	2.6
Geometric Standard Deviation	1.58	1.85	1.94	1.78
# ≥10 mg/dl	0	0	0	1
% ≥10 mg/dl	0%	0%	0%	9%
# ≥15 mg/dl	0	0	0	0
% ≥15 mg/dl	0%	0%	0%	0%

Table 6-8a Summary of Adult Blood Lead Levels by Age and Geographic Subarea (mg/dl) (continued)

	BOTH MALES AND FEMALES			
	10 to 25	26-40	41-49	Age >50
Osburn				
N	25	22	24	72
Minimum	1	1	1	1
Maximum	6	20	14	20
Arithmetic Mean	2.4	3.9	3.8	4.1
Standard Deviation	1.32	4.19	3.49	3.72
Geometric Mean	2.1	2.8	2.7	2.9
Geometric Standard Deviation	1.78	2.17	2.31	2.27
# ≥10 mg/dl	0	1	2	6
% ≥10 mg/dl	0%	5%	8%	8%
# ≥15 mg/dl	0	1	0	2
% ≥15 mg/dl	0%	5%	0%	3%
Side Gulches				
N	10	13	8	32
Minimum	1.0	1.0	2.0	1.0
Maximum	9.0	6.0	8.0	22.0
Arithmetic Mean	3.6	2.6	3.9	4.9
Standard Deviation	2.37	1.89	2.17	3.97
Geometric Mean	3.0	2.0	3.4	3.9
Geometric Standard Deviation	1.98	2.11	1.70	1.91
# ≥10 mg/dl	0	0	0	2
% ≥10 mg/dl	0%	0%	0%	6%
# ≥15 mg/dl	0	0	0	1
% ≥15 mg/dl	0%	0%	0%	3%
Kingston				
N	7	12	9	22
Minimum	1	1	2	3
Maximum	8	5	7	10
Arithmetic Mean	2.9	2.8	3.7	4.3
Standard Deviation	2.54	1.59	1.73	2.19
Geometric Mean	2.1	2.4	3.3	3.9
Geometric Standard Deviation	2.26	1.88	1.59	1.50
# ≥10 mg/dl	0	0	0	1
% ≥10 mg/dl	0%	0%	0%	5%
# ≥15 mg/dl	0	0	0	0
% ≥15 mg/dl	0%	0%	0%	0%
Lower Basin				
N	40	23	50	58
Minimum	1	1	1	1
Maximum	8	10	7	14
Arithmetic Mean	2.4	3.3	2.8	4.1
Standard Deviation	1.58	2.01	1.66	2.86
Geometric Mean	2.0	2.9	2.4	3.3
Geometric Standard Deviation	1.81	1.78	1.77	1.91
# ≥10 mg/dl	0	1	0	3
% ≥10 mg/dl	0%	4%	0%	5%
# ≥15 mg/dl	0	0	0	0
% ≥15 mg/dl	0%	0%	0%	0%

Table 6-8b Observed Blood Lead Levels for Reproductive Aged Females (mg/dl) by Geographic Subarea

	Kingston	Lower Basin	Mullan	Burke/Nine Mile	Osburn	Side Gulches	Silverton	Wallace
N	16	41	13	19	23	13	12	14
Minimum	1	1	1	1	1	1	1	1
Maximum	5	7	5	6	12	6	4	16
Arithmetic Mean	2.4	2.3	2.3	2.8	2.3	2.5	1.8	3.6
Standard Deviation	1.45	1.31	1.44	1.69	2.28	1.66	0.94	4.09
Geometric Mean	2.0	2.0	2.0	2.4	1.8	2.0	1.6	2.6
Geometric Standard Deviation	1.81	1.76	1.79	1.82	1.84	1.94	1.62	2.21
# ≥10 mg/dl	0	0	0	0	1	0	0	1
% ≥10 mg/dl	0%	0%	0%	0%	4%	0%	0%	7%
# ≥15 mg/dl	0	0	0	0	0	0	0	1
% ≥15 mg/dl	0%	0%	0%	0%	0%	0%	0%	7%

Table 6-9 Dietary Lead Summary from IEUBK Model for Children

Age Range (years)	0 to 1	1 to 2	2 to 3	3 to 4	4 to 5	5 to 6	6 to 7
Dietary Intake (mg/day)	5.53	5.78	6.49	6.24	6.01	6.34	7.00

Table 6-10 Summary of Garden, Fish, and Riparian Vegetation Lead Levels

Area (Home Grown Vegetables)	**N**	**Minimum (mg/kg)**	**Maximum (mg/kg)**	**Arithmetic Mean (mg/kg)**	**Standard Deviation (mg/kg)**	**Geometric Mean (mg/kg)**	**GSD (mg/kg)**
Mullan	1	10.90	10.90	10.90	0.00	N/A	N/A
Burke/Nine Mile	4	2.79	12.76	7.72	4.08	N/A	N/A
Wallace	1	2.00	2.00	2.00	0.00	N/A	N/A
Silverton	2	1.00	18.82	9.91	12.60	N/A	N/A
Osburn	12	0.48	48.60	9.68	14.20	N/A	N/A
Side Gulches	2	2.64	3.44	3.04	0.56	N/A	N/A
Kingston	2	1.15	1.18	1.16	0.21	N/A	N/A
Lower Basin	0	-	-	-	-	N/A	N/A
All Areas	24	0.48	48.60	7.84	10.74	N/A	N/A
Water Potatoes							
Water Potatoes without Skin	93	0.25	1.98	0.37	0.21	0.35	1.32
Water Potatoes with Skin	95	0.33	126.79	29.34	29.18	14.92	4.02
Fish Fillet Lead Data							
Bullhead (Lateral Lakes)	126	0.03	0.69	0.14	0.121	0.10	2.097
Northern Pike (Lateral Lakes)	63	0.03	0.15	0.04	0.032	0.03	1.777
Perch (Lateral Lakes)	123	0.09	2.41	0.45	0.410	0.34	2.091
Wild Rainbow Trout (Spokane River)	19	0.03	0.48	0.15	0.117	0.12	2.128
Hatchery Rainbow Trout (Spokane R.)	5	0.02	0.23	0.14	0.090	0.11	2.784
Large Scale Sucker (Spokane River)	20	0.02	0.28	0.09	0.063	0.07	1.877
Mountain Whitefish (Spokane River)	10	0.02	0.07	0.03	0.015	0.03	1.514
Whole Fish Lead Data - Spokane River							
Wild Rainbow Trout	3	0.60	1.14	0.82	0.282	0.79	1.389
Hatchery Rainbow Trout	1	1.59	1.59	1.59	N/A	N/A	N/A
Large Scale Sucker	4	1.77	4.34	2.76	1.229	2.56	1.551
Mountain Whitefish	2	0.56	0.65	0.61	0.064	0.60	1.111
Crayfish	3	0.37	1.34	0.87	0.485	0.76	1.930

N/A Geometric means not calculated due to sparse data

Mullan

Table 6-11a Summary Lead Concentration Data for Environmental Media (mg/kg)

	N	Min	Max	Arith. Mean	St. Dev.	Geo Mean	Geo Stdev
Yard Soil	105	41	20218	1187	2230	628	2.91
Vacuum	32	429	4060	1146	754	985	1.70
Dust Mat	47	278	4460	1459	880	1242	1.78
ROW	12	336	8110	2887	2554	1841	2.93
Driveway	12	352	50700	8676	15254	2893	4.36
Play Area	4	81	6210	1698	3009	419	6.54
Garden	3	210	1370	887	604	677	2.78
Water (FD)*	17	0.28	16.00	5.01	5.08	2.74	3.53
Water (P)*	16	0.04	9.50	1.58	2.27	0.65	4.82
Well Water*	9	2.50	7.00	3.39	1.78	3.09	1.52

*water units are in µg/L, FD=First Draw, P=Purged, Well Water from 1996 survey

Percent of Soil/Dust Samples by Concentration Category (mg/kg)

	Percent						
	< 500 mg/kg	500-1000 mg/kg	1000-1500 mg/kg	1500-2000 mg/kg	2000-2500 mg/kg	2500-3000 mg/kg	> 3000 mg/kg
Yard Soil	44%	23%	10%	11%	4%	1%	7%
Vacuum	6%	56%	13%	19%	0%	3%	3%
Dust Mat	6%	32%	26%	13%	13%	2%	9%
ROW	17%	17%	8%	8%	8%	0%	42%
Driveway	8%	17%	17%	8%	8%	0%	42%
Play Area	75%	0%	0%	0%	0%	0%	25%
Garden	33%	0%	67%	0%	0%	0%	0%

Burke/Nine Mile

Table 6-11b Summary Lead Concentration Data for Environmental Media (mg/kg)

	N	Min	Max	Arith. Mean	St. Dev.	Geo Mean	Geo Stdev
Yard Soil	88	32	5410	1105	973	679	3.25
Vacuum	35	83	5800	1318	1263	879	2.63
Dust Mat	54	173	59500	4048	9232	1781	2.86
ROW	10	800	6200	2974	1937	2362	2.12
Driveway	27	46	36000	3524	6684	1690	3.74
Play Area	14	83	12100	1331	3107	543	3.07
Garden	10	146	11800	2127	3755	720	4.24
Water (FD)*	34	0.50	78.50	6.32	14.67	2.31	3.31
Water (P)*	34	0.04	2.50	0.85	0.70	0.57	2.67
Well Water*	18	2.50	6.00	2.69	0.83	2.62	1.23

*water units are in µg/L, FD=First Draw, P=Purged, Well Water from 1996 survey

Percent of Soil/Dust Samples by Concentration Category (mg/kg)

	Percent						
	< 500 mg/kg	500-1000 mg/kg	1000-1500 mg/kg	1500-2000 mg/kg	2000-2500 mg/kg	2500-3000 mg/kg	> 3000 mg/kg
Yard Soil	28%	28%	15%	13%	7%	5%	5%
Vacuum	31%	20%	17%	11%	6%	9%	6%
Dust Mat	4%	24%	26%	11%	7%	7%	20%
ROW	0%	20%	20%	0%	10%	10%	40%
Driveway	11%	11%	7%	11%	22%	7%	30%
Play Area	14%	79%	0%	0%	0%	0%	7%
Garden	50%	20%	10%	0%	0%	0%	20%

Wallace

Table 6-11c Summary Lead Concentration Data for Environmental Media (mg/kg)

	N	Min	Max	Arith. Mean	St. Dev.	Geo Mean	Geo Stdev
Yard Soil	110	54	16026	1154	1628	771	2.47
Vacuum	35	259	29725	1951	4944	1004	2.33
Dust Mat	42	604	47626	3616	7987	1774	2.54
ROW	9	706	10400	2364	3063	1585	2.25
Driveway	4	70	2140	1070	1001	556	4.86
Play Area	3	613	1260	998	340	953	1.47
Garden	4	257	1200	899	434	770	2.09
Water (FD)*	19	0.67	30.10	6.53	8.70	3.19	3.33
Water (P)*	19	0.12	2.00	0.89	0.65	0.65	2.43
Well Water*	8	2.50	25.00	5.31	7.96	3.33	2.26

*water units are in µg/L, FD=First Draw, P=Purged, Well Water from 1996 survey

Percent of Soil/Dust Samples by Concentration Category (mg/kg)

	Percent						
	< 500 mg/kg	500-1000 mg/kg	1000-1500 mg/kg	1500-2000 mg/kg	2000-2500 mg/kg	2500-3000 mg/kg	> 3000 mg/kg
Yard Soil	27%	35%	16%	10%	5%	3%	5%
Vacuum	11%	51%	23%	3%	3%	0%	9%
Dust Mat	0%	26%	31%	14%	2%	2%	24%
ROW	0%	44%	11%	11%	22%	0%	11%
Driveway	50%	0%	0%	25%	25%	0%	0%
Play Area	0%	33%	67%	0%	0%	0%	0%
Garden	25%	0%	75%	0%	0%	0%	0%

Silverton

Table 6-11d Summary Lead Concentration Data for Environmental Media (mg/kg)

	N	Min	Max	Arith. Mean	St. Dev.	Geo Mean	Geo Stdev
Yard Soil	70	94	6098	524	763	352	2.25
Vacuum	26	75	3390	837	869	557	2.52
Dust Mat	22	326	3658	1064	765	863	1.93
ROW	6	321	1650	899	515	759	1.97
Driveway	9	329	4500	1331	1292	955	2.32
Play Area	3	155	576	416	228	358	2.07
Garden	5	56	1000	329	384	204	2.91
Water (FD)*	17	0.47	45.90	5.51	10.51	3.03	2.54
Water (P)*	17	0.04	2.00	1.34	0.65	1.05	2.66
Well Water*	7	2.50	16.00	4.43	5.10	3.26	2.02

*water units are in µg/L, FD=First Draw, P=Purged, Well Water from 1996 survey

Percent of Soil/Dust Samples by Concentration Category (mg/kg)

	Percent						
	< 500 mg/kg	500-1000 mg/kg	1000-1500 mg/kg	1500-2000 mg/kg	2000-2500 mg/kg	2500-3000 mg/kg	> 3000 mg/kg
Yard Soil	70%	20%	6%	3%	0%	0%	1%
Vacuum	38%	46%	4%	0%	0%	8%	4%
Dust Mat	27%	32%	23%	9%	5%	0%	5%
ROW	33%	17%	33%	17%	0%	0%	0%
Driveway	22%	22%	33%	11%	0%	0%	11%
Play Area	33%	67%	0%	0%	0%	0%	0%
Garden	80%	20%	0%	0%	0%	0%	0%

Osburn

Table 6-11e Summary Lead Concentration Data for Environmental Media (mg/kg)

	N	Min	Max	Arith. Mean	St. Dev.	Geo Mean	Geo Stdev
Yard Soil	262	33	12884	682	1195	419	2.45
Vacuum	84	23	2192	616	366	493	2.17
Dust Mat	98	202	42044	1423	4219	882	1.94
ROW	10	427	10200	3276	3210	2150	2.68
Driveway	40	33	59400	3261	9368	1069	4
Play Area	6	58	5090	990	2011	255	4.96
Garden	24	76	6605	729	1350	368	2.82
Water (FD)*	49	0.16	56.10	7.88	12.18	3.72	3.30
Water (P)*	51	0.04	2.20	0.93	0.69	0.62	2.91
Well Water*	29	2.50	310.00	13.10	57.10	2.95	2.45

*water units are in µg/L, FD=First Draw, P=Purged, Well Water from 1996 survey

Percent of Soil/Dust Samples by Concentration Category (mg/kg)

	Percent						
	< 500 mg/kg	500-1000 mg/kg	1000-1500 mg/kg	1500-2000 mg/kg	2000-2500 mg/kg	2500-3000 mg/kg	> 3000 mg/kg
Yard Soil	61%	28%	5%	2%	2%	0.4%	3%
Vacuum	40%	49%	8%	1%	1%	0%	0%
Dust Mat	12%	58%	14%	8%	2%	2%	3%
ROW	10%	10%	10%	30%	0%	10%	30%
Driveway	25%	15%	10%	20%	10%	5%	15%
Play Area	83%	0%	0%	0%	0%	0%	17%
Garden	75%	8%	8%	0%	4%	0%	4%

Side Gulches

Table 6-11f Summary Lead Concentration Data for Environmental Media (mg/kg)

	N	Min	Max	Arith. Mean	St. Dev.	Geo Mean	Geo Stdev
Yard Soil	100	25	3356	505	437	368	2.38
Vacuum	26	116	3929	952	890	695	2.21
Dust Mat	53	167	8840	1196	1504	842	2.11
ROW	3	40	2300	1016	1161	402	8.07
Driveway	13	43	117000	11021	32242	855	8.44
Play Area	4	39	316	164	126	123	2.56
Garden	4	37	1010	374	448	184	4.39
Water (FD)*	14	0.28	7.20	2.23	2.19	1.44	2.77
Water (P)*	14	0.04	4.60	1.39	1.35	0.64	5.24
Well Water*	37	2.50	2.50	2.50	0.00	2.50	1.00

*water units are in µg/L, FD=First Draw, P=Purged, Well Water from 1996 survey

Percent of Soil/Dust Samples by Concentration Category (mg/kg)

	Percent						
	< 500 mg/kg	500-1000 mg/kg	1000-1500 mg/kg	1500-2000 mg/kg	2000-2500 mg/kg	2500-3000 mg/kg	> 3000 mg/kg
Yard Soil	59%	33%	6%	1%	0%	0%	1%
Vacuum	31%	42%	8%	8%	4%	4%	4%
Dust Mat	19%	47%	15%	8%	4%	0%	8%
ROW	33%	33%	0%	0%	33%	0%	0%
Driveway	31%	31%	15%	8%	0%	0%	15%
Play Area	100%	0%	0%	0%	0%	0%	0%
Garden	75%	0%	25%	0%	0%	0%	0%

Kingston

Table 6-11g Summary Lead Concentration Data for Environmental Media (mg/kg)

	N	Min	Max	Arith. Mean	St. Dev.	Geo Mean	Geo Stdev
Yard Soil	99	22	9228	711	1622	257	3.34
Vacuum	30	102	1750	592	409	466	2.07
Dust Mat	48	63	15500	1151	2296	610	2.69
ROW	11	36	1960	618	624	330	3.73
Driveway	21	12	13200	1420	2866	412	5.57
Play Area	5	49	1120	360	456	178	3.81
Garden	11	64	1010	219	279	142	2.36
Water (FD)*	22	0.18	13.10	3.29	3.44	2.18	2.60
Water (P)*	23	0.04	4.50	1.26	0.95	0.87	2.90
Well Water*	10	2.50	31.00	6.90	9.77	3.92	2.60

*water units are in µg/L, FD=First Draw, P=Purged, Well Water from 1996 survey

Percent of Soil/Dust Samples by Concentration Category (mg/kg)

	Percent						
	< 500 mg/kg	500-1000 mg/kg	1000-1500 mg/kg	1500-2000 mg/kg	2000-2500 mg/kg	2500-3000 mg/kg	> 3000 mg/kg
Yard Soil	81%	6%	5%	1%	1%	0%	6%
Vacuum	50%	33%	13%	3%	0%	0%	0%
Dust Mat	46%	29%	8%	4%	4%	2%	6%
ROW	55%	18%	18%	9%	0%	0%	0%
Driveway	57%	14%	0%	0%	19%	0%	10%
Play Area	80%	0%	20%	0%	0%	0%	0%
Garden	91%	0%	9%	0%	0%	0%	0%

Lower Basin

Table 6-11h Summary Lead Concentration Data for Environmental Media (mg/kg)

	N	Min	Max	Arith. Mean	St. Dev.	Geo Mean	Geo Stdev
Yard Soil	160	15	7350	487	1251	110	4.29
Vacuum	31	49	3140	512	646	301	2.81
Dust Mat	110	22	4805	623	866	318	3.26
ROW	10	15	1430	230	449	71	4.11
Driveway	8	21	1280	263	466	66	5.19
Play Area	3	26	11300	5985	5665	1242	29.16
Garden	2	15	24	19	6	19	1.38
Water (FD)*	8	1.10	7.30	3.36	2.07	2.87	1.82
Water (P)*	8	0.44	2.00	1.33	0.65	1.16	1.84
Well Water*	104	2.50	66.00	7.21	11.89	4.05	2.39

*water units are in µg/L, FD=First Draw, P=Purged, Well Water from 1996 survey

Percent of Soil/Dust Samples by Concentration Category (mg/kg)

	Percent						
	< 500 mg/kg	500-1000 mg/kg	1000-1500 mg/kg	1500-2000 mg/kg	2000-2500 mg/kg	2500-3000 mg/kg	> 3000 mg/kg
Yard Soil	86%	2%	3%	2%	1%	1%	6%
Vacuum	65%	23%	6%	0%	3%	0%	3%
Dust Mat	65%	18%	5%	5%	2%	0%	4%
ROW	80%	10%	10%	0%	0%	0%	0%
Driveway	75%	13%	13%	0%	0%	0%	0%
Play Area	33%	0%	0%	0%	0%	0%	67%
Garden	100%	0%	0%	0%	0%	0%	0%

Harrison

Table 6-11i Summary Lead Concentration Data for Environmental Media (mg/kg)

	N	Min	Max	Arith. Mean	St. Dev.	Geo Mean	Geo Stdev
Yard Soil	18	17	249	103	77	79	2.15
Vacuum	18	47	1830	317	458	165	3.01
Dust Mat	17	53	2390	278	555	143	2.53
ROW	17	17	689	103	163	56	2.70
Driveway	14	15	152	65	42	53	2.00
Play Area	0	N/A	N/A	N/A	N/A	N/A	N/A
Garden	8	19	516	154	164	96	2.90
Water (FD)*	18	1.10	62.40	11.79	18.36	4.43	3.98
Water (P)*	19	1.10	4.60	1.57	0.90	1.42	1.52
Well Water*	0	0.00	0.00	--	--	--	--

*water units are in µg/L, FD=First Draw, P=Purged, Well Water from 1996 survey

Percent of Soil/Dust Samples by Concentration Category (mg/kg)

	Percent						
	< 500 mg/kg	500-1000 mg/kg	1000-1500 mg/kg	1500-2000 mg/kg	2000-2500 mg/kg	2500-3000 mg/kg	> 3000 mg/kg
Yard Soil	100%	0%	0%	0%	0%	0%	0%
Vacuum	83%	11%	0%	6%	0%	0%	0%
Dust Mat	94%	0%	0%	0%	6%	0%	0%
ROW	94%	6%	0%	0%	0%	0%	0%
Driveway	100%	0%	0%	0%	0%	0%	0%
Play Area	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Garden	88%	13%	0%	0%	0%	0%	0%

Rocky Point

Table 6-11j Summary Lead Concentration Data for Environmental Media (mg/kg)

	N	Min	Max	Arith. Mean	St. Dev.	Geo Mean	Geo Stdev
Yard Soil	7	12	66	33	21	27	1.95
Vacuum	6	28	126	85	38	76	1.77
Dust Mat	6	34	123	81	33	75	1.62
ROW	5	18	50	30	13	28	1.50
Driveway	4	13	222	75	100	38	3.81
Play Area	0	N/A	N/A	N/A	N/A	N/A	N/A
Garden	1	24	24	24	N/A	24	N/A
Water (FD)*	5	1.80	158.00	35.66	68.51	8.11	6.08
Water (P)*	7	1.10	3.40	1.80	0.93	1.63	1.60
Well Water*	0	0.00	0.00	--	--	--	--

*water units are in µg/L, FD=First Draw, P=Purged, Well Water from 1996 survey

Percent of Soil/Dust Samples by Concentration Category (mg/kg)

	Percent						
	< 500 mg/kg	500-1000 mg/kg	1000-1500 mg/kg	1500-2000 mg/kg	2000-2500 mg/kg	2500-3000 mg/kg	> 3000 mg/kg
Yard Soil	100%	0%	0%	0%	0%	0%	0%
Vacuum	100%	0%	0%	0%	0%	0%	0%
Dust Mat	100%	0%	0%	0%	0%	0%	0%
ROW	100%	0%	0%	0%	0%	0%	0%
Driveway	100%	0%	0%	0%	0%	0%	0%
Play Area	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Garden	100%	0%	0%	0%	0%	0%	0%

Table 6-12a Summary of CUA Surface Water Lead Concentration (Disturbed Samples) (mg/L)

Site	Minimum Detected Value	Maximum Detected Value	Average Exposure Concentration*	95% Upper Confidence Limit	Reasonable Maximum Exposure
CUA018 - Harrison Beach	117	469	267	**N/A**	**N/A**
CUA035 - Springston Beach Site	661	2810	1670	2480	2480
CUA036 - Across river from Springston	6310	56500	18300	38700	38700
CUA038 - Thompson Lake	166	876	373	660	660
CUA039 - Long Beach RM135/Springston	7190	38800	22900	36900	36900
CUA041 - West of Blue Lake	2710	15600	9390	13900	13900
CUA045 - Medimont Boat Ramp	776	6520	3010	5690	5690
CUA047 - Rainy Hill picnic area	452	3280	1580	2750	2750
CUA048 - RM 145	16600	81500	41300	64900	64900
CUA049 - Beach near canal to Killarney Lake	8090	23100	14700	19800	19800
CUA051 - Lane Beach	21300	52300	31700	43100	43100
CUA052 - Near East end of Killarney Lake	11300	39600	25300	35800	35800
CUA053 - Beach below Ward Ridge	24600	46300	30800	39300	39300
CUA054 - Black Rock Gulch Beach	6300	29300	16300	25800	25800
CUA055 - Quarry Beach	7520	31000	18800	27500	27500
CUA056 - RV Park across from Black Rock Gulch	3240	25800	9690	18700	18700
CUA057 - Beach upstream from Quarry	13700	38800	28900	37900	37900
CUA058 - East end of Blackrock Gulch Marsh	7640	59400	29100	47600	47600
CUA059 - East of Rose Creek	2940	72500	25300	52300	52300
CUA060 - West of Rose Lake	3790	40300	21500	36000	36000
CUA063 - Bull Run Peak Beach	2310	11800	5280	8900	8900
CUA065 - Just South of Mission Falls	1980	8430	4250	6740	6740
CUA068 - South of Old Mission Park	251	1690	1180	1730	1690
CUA069 - Skeel Gulch Beach	141	1370	658	1110	1110
Total Lower Basin	117	81500	7530	N/A	N/A
CUA077 - Confluence with Coeur D'Alene River (Kingston)	85	12500	3270	8300	8300
CUA081 - Elk Creek Pond (Side Gulches)	35	7180	1630	4600	4600

* This is the arithmetic average by CUA, but for all CUAs in the Lower Basin, this value is the geometric mean.

Table 6-12b Summary of CUA Surface Water Lead Concentration (Undisturbed Samples) (mg/L)

Site	N	Minimum Detected Value	Maximum Detected Value	Mean	Geometric Mean	Standard Deviation	Geometric Standard Deviation
Lower Basin	93	2	430	38.0	3.13	64.0	0.89

Table 6-13 Paint Lead Loadings by Geographic Area

	Arithmetic Mean Interior Paint Lead Loadings (mg/cm^2)	Geometric Mean Interior Paint Lead Loadings (mg/cm^2)	Maximum Interior Paint Lead Loadings (mg/cm^2)	Median Interior Paint Lead Loadings (mg/cm^2)	Arithmetic Mean Interior Paint Condition Code*	Maximum Interior Paint Condition Code*	Minimum Interior Paint Condition Code*	Median Interior Paint Condition Code*
Mullan								
N	43	43	43	43	41	41	41	41
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	3.039	0.724	9.900	1.500	1.8	3.0	1.0	2.0
Mean	0.563	0.031	2.912	0.166	1.3	1.8	1.0	1.2
Stdev	0.721	6.688	3.597	0.258	0.2	0.6	0.0	0.4
Burke/Nine Mile								
N	38	38	38	38	35	35	35	35
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	5.504	2.141	9.900	6.975	3.0	3.0	3.0	3.0
Mean	0.882	0.023	3.099	0.535	1.6	2.1	1.4	1.6
Stdev	1.448	10.151	3.852	1.509	0.6	0.8	0.6	0.7
Wallace								
N	37	37	37	37	37	37	37	37
Min	0.009	0.001	0.100	0.001	1.0	1.0	1.0	1.0
Max	5.546	1.229	9.900	8.600	2.6	3.0	2.0	3.0
Mean	1.311	0.077	4.420	0.940	1.6	2.2	1.3	1.6
Stdev	1.705	5.256	4.175	1.944	0.5	0.7	0.5	0.5
Silverton								
N	23	23	23	23	21	21	21	21
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	1.129	0.572	9.900	0.850	1.6	3.0	1.0	2.0
Mean	0.336	0.053	1.696	0.201	1.2	1.8	1.0	1.3
Stdev	0.294	6.559	2.474	0.192	0.3	0.7	0.0	0.4
Osburn								
N	81	81	81	81	70	70	70	70
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	2.656	0.351	9.850	0.800	2.5	3.0	2.0	2.5
Mean	0.243	0.020	1.113	0.115	1.4	1.9	1.2	1.4
Stdev	0.361	6.375	1.899	0.137	0.4	0.7	0.4	0.5
Side Gulches								
N	52	52	52	52	47	47	47	47
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	1.091	0.340	6.400	0.400	3.0	3.0	3.0	3.0
Mean	0.200	0.022	0.905	0.117	1.4	1.9	1.2	1.4
Stdev	0.219	7.828	1.288	0.122	0.4	0.7	0.5	0.5
Kingston								
N	37	37	37	37	36	36	36	36
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	1.981	0.571	9.900	1.325	3.0	3.0	3.0	3.0
Mean	0.313	0.016	1.312	0.124	1.6	1.9	1.4	1.6
Stdev	0.525	7.233	2.324	0.240	0.5	0.7	0.5	0.6
Lower Basin								
N	104	104	104	104	96	96	96	96
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	7.850	7.850	9.900	7.850	3.0	3.0	3.0	3.0
Mean	0.298	0.010	0.863	0.231	1.5	1.8	1.3	1.4
Stdev	0.997	6.283	1.830	1.099	0.5	0.7	0.5	0.6

* 1- New Paint and/or excellent condition
2- Worn, chipped, or scraped paint
3- Peeling, cracked, flaking paint, and/or chalking

Table 6-13 Paint Lead Loadings by Geographic Area (continued)

	Arithmetic Mean Exterior Paint Lead Loadings (mg/cm^2)	Geometric Mean Interior Paint Lead Loadings (mg/cm^2)	Maximum Exterior Paint Lead Loadings (mg/cm^2)	Median Exterior Paint Lead Loadings (mg/cm^2)	Arithmetic Mean Exterior Paint Condition Code*	Maximum Exterior Paint Condition Code*	Minimum Exterior Paint Condition Code*	Median Exterior Paint Condition Code*
Mullan								
N	43	43	43	43	43	43	43	43
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	4.901	2.828	9.900	4.851	3.0	3.0	3.0	3.0
Mean	1.359	0.102	3.613	0.746	1.7	2.2	1.2	1.7
Stdev	1.510	7.028	3.852	1.350	0.6	0.8	0.5	0.7
Burke/Nine Mile								
N	39	39	39	39	38	38	38	38
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	8.258	4.700	9.900	9.900	3.0	3.0	3.0	3.0
Mean	1.598	0.050	3.617	1.216	2.2	2.5	1.9	2.2
Stdev	2.248	12.916	4.441	2.606	0.8	0.8	0.8	0.8
Wallace								
N	37	37	37	37	37	37	37	37
Min	0.076	0.006	0.150	0.001	1.0	1.0	1.0	1.0
Max	9.900	9.900	9.900	9.900	3.0	3.0	3.0	3.0
Mean	3.114	0.222	6.815	2.459	2.3	2.7	1.9	2.4
Stdev	2.618	7.887	3.994	3.456	0.5	0.5	0.7	0.6
Silverton								
N	24	24	24	24	23	23	23	23
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	2.600	1.835	8.200	2.800	3.0	3.0	3.0	3.0
Mean	0.691	0.084	1.646	0.429	1.7	2.1	1.4	1.6
Stdev	0.749	8.240	1.905	0.614	0.7	0.8	0.7	0.8
Osburn								
N	79	79	79	79	72	72	72	72
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	7.950	4.283	9.900	9.900	3.0	3.0	3.0	3.0
Mean	0.907	0.058	2.304	0.597	1.9	2.3	1.6	1.8
Stdev	1.411	8.873	3.341	1.429	0.6	0.7	0.7	0.7
Side Gulches								
N	53	53	53	53	48	48	48	48
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	2.200	1.668	4.450	2.350	3.0	3.0	3.0	3.0
Mean	0.285	0.035	0.671	0.213	1.7	2.1	1.4	1.7
Stdev	0.435	7.192	1.042	0.395	0.5	0.7	0.5	0.6
Kingston								
N	40	40	40	40	39	39	39	39
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	8.600	8.600	9.900	8.650	3.0	3.0	3.0	3.0
Mean	0.834	0.032	1.482	0.730	2.1	2.3	1.8	2.1
Stdev	1.797	15.649	2.854	1.921	0.7	0.7	0.7	0.8
Lower Basin								
N	102	102	102	102	97	97	97	97
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	3.200	0.930	9.900	2.776	3.0	3.0	3.0	3.0
Mean	0.358	0.037	0.981	0.212	1.9	2.3	1.6	1.9
Stdev	0.627	5.120	2.005	0.412	0.7	0.8	0.7	0.7

* 1- New Paint and/or excellent condition
2- Worn, chipped, or scraped paint
3- Peeling, cracked, flaking paint, and/or chalking

Table 6-13 Paint Lead Loadings by Geographic Area (continued)

	Arithmetic Mean Mat Location Paint Lead Loadings (mg/cm^2)	Geometric Mean Mat Location Paint Lead Loadings (mg/cm^2)	Maximum Mat Location Paint Lead Loadings (mg/cm^2)	Median Mat Location Paint Lead Loadings (mg/cm^2)	Arithmetic Mean Mat Location Paint Condition Code*	Maximum Mat Location Paint Condition Code*	Minimum Mat Location Paint Condition Code*	Median Mat Location Paint Condition Code*
Mullan								
N	40	40	40	40	34	34	34	34
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	3.600	3.600	6.100	3.600	3.0	3.0	3.0	3.0
Mean	0.355	0.028	0.500	0.293	1.4	1.5	1.4	1.4
Stdev	0.729	16.150	1.153	0.664	0.7	0.7	0.7	0.6
Burke/Nine Mile								
N	32	32	32	32	19	19	19	19
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	8.850	8.850	9.900	8.850	3.0	3.0	3.0	3.0
Mean	0.635	0.011	0.999	0.569	2.2	2.3	2.2	2.3
Stdev	1.877	19.830	2.806	1.882	0.8	0.7	0.8	0.8
Wallace								
N	30	30	30	30	22	22	22	22
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	9.900	9.900	9.900	9.900	3.0	3.0	3.0	3.0
Mean	1.226	0.050	1.277	1.219	1.8	2.0	1.7	1.8
Stdev	2.975	20.922	2.958	2.977	0.6	0.7	0.6	0.7
Silverton								
N	22	22	22	22	20	20	20	20
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	3.150	3.150	9.900	3.150	3.0	3.0	3.0	3.0
Mean	0.471	0.087	0.955	0.328	1.3	1.5	1.3	1.3
Stdev	0.845	8.017	2.140	0.655	0.6	0.7	0.6	0.5
Osburn								
N	68	68	68	68	54	54	54	54
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	2.475	1.250	7.250	1.476	3.0	3.0	3.0	3.0
Mean	0.237	0.025	0.421	0.210	1.5	1.6	1.4	1.5
Stdev	0.395	11.140	0.992	0.318	0.5	0.6	0.6	0.6
Side Gulches								
N	47	47	47	47	36	36	36	36
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	1.750	1.750	1.750	1.750	3.0	3.0	3.0	3.0
Mean	0.192	0.024	0.248	0.187	1.5	1.6	1.4	1.5
Stdev	0.300	12.960	0.342	0.300	0.5	0.6	0.6	0.5
Kingston								
N	31	31	31	31	24	24	24	24
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	2.200	2.200	3.550	2.200	3.0	3.0	3.0	3.0
Mean	0.298	0.015	0.391	0.276	1.7	1.7	1.6	1.6
Stdev	0.565	16.789	0.794	0.544	0.7	0.8	0.7	0.7
Lower Basin								
N	99	99	99	99	81	81	81	81
Min	0.001	0.001	0.001	0.001	1.0	1.0	1.0	1.0
Max	3.525	2.900	6.800	3.525	3.0	3.0	3.0	3.0
Mean	0.283	0.026	0.385	0.262	1.6	1.7	1.6	1.6
Stdev	0.576	14.743	0.942	0.562	0.6	0.7	0.7	0.7

* 1- New Paint and/or excellent condition
2- Worn, chipped, or scraped paint
3- Peeling, cracked, flaking paint, and/or chalking

Table 6-14 Summary of Neighborhood Stream Sediment Lead Levels by Geographic Subarea

Area	N	Minimum	Maximum	Arithmetic Mean	Standard Deviation	Geometric Mean	Geometric Standard Deviation
	Sediment (mg/kg)						
Mullan	14	31	13800	2204	3710	771	4.9
Burke/Nine Mile	17	88	67100	8756	16025	2803	5.6
Kingston	12	25	249	155	67	135	1.9
Osburn, Wallace, Silverton	22	738	6160	2889	1756	2395	1.9
All Areas	65	25	67100	3771	8824	1150	5.2
	Surface Water (mg/L)						
Mullan	65	0	44	4.3	7.7	1.2	5.8
Burke/Nine Mile	79	0	1650	87.1	236.8	20.6	6.7
Kingston	12	0	38	4.7	10.9	1.0	5.0
Osburn, Wallace, Silverton	66	0	49	11.4	13.6	3.0	6.8
All Areas	222	0	1650	35.9	146.0	4.3	9.1

Table 6-15a Summary of Surface Soil Lead Levels for Common Use Areas (mg/kg)

Site	Minimum Detected Value (mg/kg)	Maximum Detected Value (mg/kg)	Mean Soil Concentration	95% Upper Confidence Limit	Reasonable Maximum Exposure
CUA033 - Trestle area next to Route	1500	2470	2060	2440	2440
CUA035 - Springston Beach Site	2140	2710	2440	2670	2670
CUA036 - across river from Springston	2840	7250	4390	6110	6110
CUA038 - Thompson Lake	250	2420	818	1690	1690
CUA039 - Long Beach RM135/Springston	2830	3580	3290	3600	3580
CUA041 - West of Blue lake	382	2150	1070	1800	1800
CUA043 - West beach near Medimont	3540	4210	3930	4190	4190
CUA044 - Medimont Hill Camping Area	2160	3730	2660	3260	3260
CUA045 - Medimont Boat Ramp	2840	4690	3510	4210	4210
CUA046 - Rainy Hill Fishing Area	3770	4540	4150	4420	4420
CUA047 - Rainy Hill picnic area	1060	3290	1930	2750	2750
CUA048 - RM 145	3000	3680	3500	3770	3680
CUA049 - Beach near canal to Killarney Lake	3740	4900	4240	4750	4750
CUA050 - Killarney	15.3	271	73	179	179
CUA051 - Lane Beach	3910	4850	4340	4710	4710
CUA052 - Near East end of Killarney Lake	4040	4560	4250	4460	4460
CUA053 - Beach below Ward Ridge	4270	5330	4710	5110	5110
CUA054 - Black Rock Gulch Beach	3580	5120	4500	5040	5040
CUA055 - Quarry Beach	4100	4910	4370	4680	4680
CUA056 - RV Park across from Black Rock Gulch	3690	4850	4370	4820	4820
CUA057 - Beach upstream from Quarry	3190	5140	4350	5070	5070
CUA058 - East end of Blackrock Gulch Marsh	3910	6080	5180	6090	6080
CUA059 - East of Rose Creek	2970	6010	4610	5770	5770
CUA060 - West of Rose Lake	4420	5580	4900	5370	5370
CUA063 - Bull Run Peak Beach	4040	6390	4790	5700	5700
CUA064 - Mouth of 4th of July Marsh	3360	3920	3610	3820	3820
CUA065 - Just South of Mission Falls	3060	3530	3350	3530	3530
CUA066 - Beach in Mission Flats	2670	3520	3130	3470	3470
CUA067 - Old Mission State Park Boat Launch	123	1270	521	944	944
CUA068 - South of Old Mission Park	2190	3740	2910	3540	3540
CUA069 - Skeel Gulch Beach	2430	3690	3180	3710	3690
Canyon Elementary - Play Areas[1]			63		
Canyon Elementary - Ballfields[1]			56		
Canyon Elementary - Basketball Court[1]			25		
Total Lower Basin[2]	15	7250	1939		
CUA077 - Confluence with Coeur D'Alene River	2660	4020	3480	3950	3950
Silver Meadow Adventist School - Play Areas[1]			17		
Silver Meadow Adventist School - Ballfield[1]			136		
Silver Meadow Adventist School - Gravel Drive[1]			61		
Total Kingston[2]			149		
CUA080 - Elk Dreek Frontage roak/county Road	7870	15400	12100	15100	15100
CUA089 - Silverton T-ball/Wellman ballfield	179	726	309	360	360
CUA090 - Silverton T-ball/surrounding park at Wellman Ballfield	151	522	293	334	334
CUA091 - Silverton T-ball /Wellman & HS parking lot	362	3130	1330	1680	1680
CUA092 - Silverton School/Huggy Bear Day Care	66	4930	665	918	918
CUA094 - Silverton ballfield next to Huggy Bear Day Care	2604	6550	689	1290	1290
CUA095 - Silverton School District Satner Field	105	11600	1570	2310	2310
Total Silverton[2]	66	11600	665		
CUA096 - Wallace City Park (Monument)	1450	7222	3170	4630	4630
CUA097 - Wallace Library	1140	4710	2160	2940	2940
CUA098 - Wallace Depot	179	685	464	608	608
CUA099 - Small Wallace City Park mear schools	51.7	596	181	327	327
CUA100 - Wallace High School	64.6	6900	1430	2050	2050
CUA101 - Canyon Avenue Park	76.8	673	198	355	355
CUA102 - Wallace Visitor's Center	29.8	2670	927	1420	1420
Total Wallace[2]	29.8	7222	763		
Mullan Elementary[1]			3270		
Mullan High School - Play Area[1]			588		
Mullan High School - Public Area[1]			1790		
Mullan High School - Athletic Pavilion Public Area[1]			359		
Total Mullan[2]			1054		

[1]Data was taken from "Candidates for Early Removal Actions and/or Intervention Strategies among Schools and Daycares of Shoshone Kootenai Counties, Idaho." Raw data was not given.

[2]Totals for Lower Basin, Kingston, Silverton, Wallace, and Mullan are the geometric mean of the CUA's and schools in those geographic areas

Table 6-15b Summary of Sediment Lead Levels for Common Use Areas (mg/kg)

Site	Minimum Detected Value	Maximum Detected Value	Average Exposure Concentration*	95% Upper Confidence Limit	Reasonable Maximum Exposure
CUA18 - Harrison Beach	21	12100	1253	**N/A**	**N/A**
CUA033 - Trestle area next to Route	2290	2800	2610	2810	2800
CUA035 - Springston Beach Site	1460	2600	2210	2390	2390
CUA036 - across river from Springston	2560	3580	3020	3220	3220
CUA038 - Thompson Lake	18	117	55.6	99	99
CUA039 - Long Beach RM135/Springston	2520	7480	3540	4410	4410
CUA041 - West of Blue lake	39	3420	980	2320	2320
CUA045 - Medimont Boat Ramp	2620	3790	3110	3560	3560
CUA047 - Rainy Hill picnic area	2180	3590	2890	7340	3590
CUA048 - RM 145	3010	5000	3990	4340	4340
CUA049 - Beach near canal to Killarney Lake	2960	4670	3610	3910	3910
CUA051 - Lane Beach	3410	4450	3830	4030	4030
CUA052 - Near East end of Killarney Lake	3900	5960	4370	4740	4740
CUA053 - Beach below Ward Ridge	3900	15000	5750	7670	7670
CUA054 - Black Rock Gulch Beach	3580	7120	4840	5490	5490
CUA055 - Quarry Beach	4330	5410	4810	5000	5000
CUA056 - RV Park across from Black Rock Gulch	1750	5000	4070	5330	5000
CUA057 - Beach upstream from Quarry	4010	6080	5000	5380	5380
CUA058 - East end of Blackrock Gulch Marsh	4010	5300	4650	4920	4920
CUA059 - East of Rose Creek	282	6120	3780	4860	4860
CUA060 - West of Rose Lake	34	5910	3430	4530	4530
CUA063 - Bull Run Peak Beach	2830	29200	9560	14100	14100
CUA065 - Just South of Mission Falls	2420	4810	3770	4230	4230
CUA068 - South of Old Mission Park	1950	2660	2430	2550	2550
CUA069 - Skeel Gulch Beach	2060	3600	2720	2980	2980
Total Lower Basin	18	29200	2898	N/A	N/A
CUA077 - Confluence with Coeur D'Alene River (Kingston)	3010	6360	4410	4900	4900
CUA081 - Elk Creek Pond (Side Gulches)	45	14800	3630	6370	6370

* This is the arithmetic average by CUA, except in the Lower Basin, this value is the geometric mean.

Table 6-16 Summary of Lead Concentration for Waste Pile Soils (mg/kg)

	Burke/Nine Mile					Mullan	All Areas
	Tiger Poorman	Tamarack #7	Rex #2	Success	Rex	Mullan L.F.	All Waste Piles
N	5	5	5	5	2	5	27
Minimum	1510	104	2050	83.4	4610	228	83
Maximum	49800	63700	4500	6210	16100	4570	63700
Arithmetic Mean	12500	13838	3394	3625	10355	1871	7291
Standard Deviation	20884	27943	1118	2258	8125	2247	14726
Geometric Mean	5159	1246	3231	1985	8615	754	2217

Table 6-17 Mat Dust Lead Concentration, Dust Loading, and Lead Loading for the Coeur d'Alene Basin

Area	Total Number of Observations	Minimum	Maximum	Arithmetic Mean	Standard Deviation	Geometric Mean	Geometric Standard Deviation
	Dust Mat Lead Concentration (mg/kg)						
Mullan	47	278	4460	1459	880	1242	1.78
Burke/Nine Mile	54	173	59500	4048	9232	1781	2.86
Wallace	42	604	47626	3616	7987	1774	2.54
Silverton	22	326	3658	1064	765	863	1.93
Osburn	98	202	42044	1423	4219	882	1.94
Side Gulches	53	167	8840	1196	1504	842	2.11
Kingston	48	63	15500	1151	2296	610	2.69
Lower Basin/Cataldo	110	22	4805	623	866	318	3.26
	Dust Loading ($g/m^2/day$)						
Mullan	40	0.7	7.4	1.6	1.2	1.38	1.73
Burke/Nine Mile	37	0.5	23.5	4.0	5.1	2.31	2.79
Wallace	33	0.3	6.9	1.9	1.6	1.45	2.08
Silverton	19	0.5	17.5	2.2	3.8	1.31	2.31
Osburn	73	0.5	6.9	1.2	0.9	1.04	1.69
Side Gulches	47	0.5	6.8	1.8	1.3	1.44	1.83
Kingston	42	0.4	7.6	1.7	1.7	1.21	2.21
Lower Basin/Cataldo	109	0.3	17.0	2.5	3.0	1.54	2.61
	Lead Loading ($mg/m^2/day$)						
Mullan	40	0.4	10.5	2.0	2.0	1.52	2.04
Burke/Nine Mile	37	0.3	87.2	11.5	17.4	4.28	4.43
Wallace	33	0.3	158.3	8.4	27.5	2.63	3.13
Silverton	19	0.3	9.5	1.7	2.0	1.10	2.42
Osburn	73	0.2	66.2	2.0	7.7	0.87	2.49
Side Gulches	47	0.2	21.4	1.9	3.3	1.13	2.55
Kingston	42	0.1	6.3	1.4	1.4	0.74	3.25
Lower Basin/Cataldo	109	0.0	29.7	1.6	4.1	0.48	4.41

*loading data are for 1996 only; concentration data also include some mats corrected by EPA in 1997-1999, loading data for these were not available at this time.

Table 6-18 Summary of Lead Concentrations for Sediment and Surface Soil in the Lower Basin

Media	Minimum (mg/kg)	Maximum (mg/kg)	Arithmetic Mean Concentration (mg/kg)	Standard Deviation (mg/kg)	Geometric Mean Concentration (mg/kg)	GSD (mg/kg)
Sediment	56	9560	3642	1778	2945	2.53
Surface Soil	17	5180	2868	1741	1480	5.55

Table 6-19 Correlation Matrix for Blood Lead Levels and Environmental Source Variables

VARIABLE	BLPB	
	N	r
AGE	524	-0.305**
MATPB	169	0.314**
LEADLD	140	0.634**
VACPB	205	0.098
SOILPB	444	0.158**
GCOMMEAN	524	0.116*
INTMEAN	216	0.304**
INTMAX	216	0.177*
INTMED	216	0.341**
INCCMEAN	205	0.478**
INTCCMAX	205	0.324**
INTCCMIN	205	0.426**
INTCCMED	205	0.421**
EXTMEAN	217	0.327**
EXTMAX	217	0.185*
EXTMED	217	0.407**
EXCCMEAN	208	0.355**
EXTCCMAX	208	0.267**
EXTCCMIN	208	0.237**
EXTCCMED	208	0.395**

** $P \leq 0.001$

* $P \leq 0.05$

BLPB	Blood lead levels (µg/dl)
AGE	Child age (years)
MATPB	Mat dust lead concentration (mg/kg)
LEADLD	Lead loading rate ($mg/m^2/day$)
VACPB	Vacuum cleaner dust lead concentration (mg/kg)
SOILPB	Yard soil lead concentration (mg/kg)
GCOMMEAN	Community geometric mean soil lead concentration (mg/kg)
INTMEAN	Arithmetic mean interior paint lead loading(mg/cm^2)
INTMAX	Maximum interior paint lead loading (mg/cm^2)
INTMED	Median interior paint lead loading (unitless)
INCCMEAN	Arithmetic mean interior paint condition (unitless)
INTCCMAX	Maximum interior paint condition (unitless)
INTCCMIN	Minimum interior paint condition (unitless)
INTCCMED	Median interior paint condition (unitless)
EXTMEAN	Arithmetic mean exterior paint lead loading (mg/cm^2)
EXTMAX	Maximum exterior paint lead loading (mg/cm^2)
EXTMED	Median exterior paint lead loading (mg/cm^2)
EXCCMEAN	Arithmetic mean exterior paint condition (unitless)
EXTCCMAX	Maximum exterior paint condition (unitless)
EXTCCMIN	Minimum exterior paint condition (unitless)
EXTCCMED	Median exterior paint condition (unitless)

Table 6-20 Correlation Matrix for House Dust Lead Levels and Environmental Source Variables

VARIABLE	LNMATPB		LNPBLD		LNVACPB	
	N	r	N	r	N	r
LNMATPB	498	1	400	0.804**	163	0.721**
LNPBLD	400	0.804**	400	1	73	0.510**
LNVACPB	163	0.721**	73	0.510**	324	1
LNSOILPB	490	0.646**	392	0.534**	318	0.515**
GCOMMEAN	491	0.556**	400	0.439**	317	0.453**
INTMEAN	374	0.233**	374	0.226**	74	0.219
INTMAX	374	0.301**	374	0.285**	74	0.298*
INTMED	374	0.130*	374	0.153*	74	0.083
INCCMEAN	346	0.129*	346	0.252**	70	0.012
INTCCMAX	346	0.132*	346	0.222**	70	0.107
INTCCMIN	346	0.084	346	0.187**	70	0.041
INTCCMED	346	0.089	346	0.211**	70	-0.116
EXTMEAN	377	0.294**	377	0.264**	73	0.245*
EXTMAX	377	0.289**	377	0.241**	73	0.215
EXTMED	377	0.257**	377	0.243**	73	0.204
EXCCMEAN	359	0.198**	359	0.252**	70	0.153
EXTCCMAX	359	0.174**	359	0.199**	70	0.096
EXTCCMIN	359	0.132*	359	0.211**	70	0.125
EXTCCMED	359	0.199**	359	0.248**	70	0.197
MLMEAN	335	0.112*	335	0.115*	69	0.098
MLMAX	335	0.152*	335	0.131*	69	0.183
MLMED	335	0.089	335	0.095	69	0.020
MLCCMEAN	264	0.099	264	0.190*	57	0.156
MLCCMAX	264	0.119	264	0.199*	57	0.115
MLCCMIN	264	0.072	264	0.168*	57	0.165
MLCCMED	264	0.099	264	0.187*	57	0.175

** $P \leq 0.001$

* $P \leq 0.05$

LNMATPB	Log mat dust lead concentration (mg/kg)
LNPBLD	Log lead loading rate (mg/m^2/day)
LNVACPB	Log vacuum cleaner dust lead concentration (mg/kg)
LNSOILPB	Yard soil lead concentration (mg/kg)
GCOMMEAN	Community geomean soil lead concentration (mg/kg)
INTMEAN	Arithmetic mean interior paint lead loading (mg/cm^2)
INTMAX	Maximum interior paint lead loading (mg/cm^2)
INTMED	Median interior paint lead loading (mg/cm^2)
INCCMEAN	Arithmetic mean interior paint condition (unitless)
INTCCMAX	Maximum interior paint condition (unitless)
INTCCMIN	Minimum interior paint condition (unitless)
INTCCMED	Median interior paint condition (unitless)
EXTMEAN	Arithmetic mean exterior paint lead loading (mg/cm^2)
EXTMAX	Maximum exterior paint lead loading (mg/cm^2)
EXTMED	Median exterior paint lead loading (mg/cm^2)
EXCCMEAN	Arithmetic mean exterior paint condition (unitless)
EXTCCMAX	Maximum exterior paint condition (unitless)
EXTCCMIN	Minimum exterior paint condition (unitless)
EXTCCMED	Median exterior paint condition (unitless)
MLMEAN	Arithmetic mean mat location paint lead loading (mg/cm^2)
MLMAX	Maximum mat location paint lead loading (mg/cm^2)
MLMED	Median mat location paint lead loading (mg/cm^2)
MLCCMEAN	Arithmetic mean mat location paint condition (unitless)
MLCCMAX	Maximum mat location paint condition (unitless)
MLCCMIN	Minimum mat location paint condition (unitless)
MLCCMED	Median mat location paint condition (unitless)

Table 6-21 General Linear Model and Regression Coefficients for Blood Lead and Environmental Sources

Dependent Variable: BLPB
R-Square=0.597 (P<0.0001)
N=126

Variable	Estimate	Pr>F	Standardized Estimate
Intercept	2.8644	0.0032	0.0000
AGE	-0.3351	0.0007	-0.2056
SOILPB	0.0007	0.0012	0.2249
LEADLD	0.1638	0.0006	0.3212
EXTMED	0.5176	0.0005	0.2742
INTCCMIN	1.9230	0.0008	0.2313

Table 6-22a General Linear Model and Regression Coefficients for Entryway Mat Lead Concentration and Environmental Sources

Dependent Variable: LNMATPB
R-Square=0.444 (P<0.0001)
N=332

Variable	Estimate	Pr>F	Standardized Estimate
Intercept	3.5549	0.0001	0.0000
LNSOILPB	0.4664	0.0001	0.5868
INTMAX	0.0416	0.0174	0.1089
INTCCMIN	0.1960	0.0489	0.0818
EXTMED	0.0572	0.0269	0.0996

BLPB	Blood lead levels (µg/dl)
SOILPB	Yard soil lead concentration (mg/kg)
LEADLD	Lead loading rate ($mg/m^2/day$)
EXTMED	Median exterior paint lead loading (mg/cm^2)
INTCCMIN	Minimum interior paint condition (unitless)
LNMATPB	Log transformed mat dust lead concentration (mg/kg)
LNSOILPB	Log transformed yard soil lead concentration (mg/kg)
INTMAX	Maximum interior paint lead loading (mg/cm^2)

Table 6-22b General Linear Model and Regression Coefficients for Entryway Mat Lead Concentration and Environmental Sources

Dependent Variable: LNMATPB
R-Square=0.484 (P<0.0001)
N=332

Variable	Estimate	Pr>F	Standardized Estimate
Intercept	3.6096	0.0001	0.0000
LNSOILPB	0.3764	0.0001	0.4735
INTMAX	0.0264	0.1218	0.0692
INTCCMIN	0.2260	0.0189	0.0944
EXTMED	0.0347	0.1697	0.0605
GCOMMEAN	0.0013	0.0001	0.2488

Table 6-22c General Linear Model and Regression Coefficients for Entryway Mat Lead Concentration and Environmental Sources (Final Model)

Dependent Variable: LNMATPB
R-Square=0.466 (P<0.0001)
N=339

Variable	Estimate	Pr>F	Standardized Estimate
Intercept	3.5641	0.0001	0.0000
LNSOILPB	0.3838	0.0001	0.4818
INTCCMIN	0.2321	0.0168	0.0961
GCOMMEAN	0.0015	0.0001	0.2855

LNMATPB	Log transformed mat dust lead concentration (mg/kg)
LNSOILPB	Log transformed yard soil lead concentration (mg/kg)
INTMAX	Maximum interior paint lead loading (mg/cm^2)
INTCCMIN	Minimum interior paint condition (unitless)
EXTMED	Median exterior paint lead loading (mg/cm^2)
GCOMMEAN	Community geometric mean soil lead concentration (mg/kg)

Table 6-22d General Linear Model and Regression Coefficients for Dust Mat Lead Loading and Environmental Sources

Dependent Variable: LNPBLD
R-Square=0.361 (P<0.0001)
N=339

Variable	Estimate	Pr>F	Standardized Estimate
Intercept	-3.5216	0.0001	0.0000
LNSOILPB	0.4062	0.0001	0.4042
INTCCMIN	0.5489	0.0001	0.1801
INTMAX	0.0532	0.0203	0.1103
GCOMMEAN	0.0012	0.0005	0.1879

Table 6-23 General Linear Model and Regression Coefficients for Vacuum Bag Lead Concentration and Environmental Sources

Dependent Variable: LNVACPB
R-Square=0.546 (P<0.0001)
N=68

Variable	Estimate	Pr>F	Standardized Estimate
Intercept	3.2686	0.0001	0.0000
LNSOILPB	0.1438	0.0104	0.2702
LNMATPB	0.3380	0.0001	0.4568
INTMAX	0.0697	0.0296	0.2012

LNPBLD	Log transformed lead loading rate ($mg/m^2/day$)
LNSOILPB	Log transformed yard soil lead concentration (mg/kg)
INTCCMIN	Minimum interior paint condition (unitless)
INTMAX	Maximum interior paint lead loading (mg/cm^2)
GCOMMEAN	Community geometric mean soil lead concentration
LNMATPB	Log transformed mat dust lead concentration

Table 6-24a IEUBK Default Inhalation and Ingestion Parameters

Age (years)	Inhalation			Ingestion		
	Hours spent outdoors (hr/day)	Ventilation Rate (m^3/day)	Lung Absorption (%)	Total Dust and Soil Intake (g/day)	Diet Intake (mg/day)	Water (L/day)
0-1	1	2	32	0.085	5.53	0.20
1-2	2	3	32	0.135	5.78	0.50
2-3	3	5	32	0.135	6.49	0.52
3-4	4	5	32	0.135	6.24	0.53
4-5	4	5	32	0.100	6.01	0.55
5-6	4	7	32	0.090	6.34	0.58
6-7	4	7	32	0.085	7.00	0.59

Table 6-24b EPA Adult Model Default Ingestion Parameters

Parameter	Value	Unit	Definition
$PbB_{fetal,\ 0.95,\ goal}$	10	µg/dl	Goal for the 95th percentile blood lead concentration (µg/dl) in fetuses born to women having exposures to the specified site soil concentrations.
$GSD_{i,adult}$	1.8	-	Individual standard deviation among adults. A value of 1.8 is recommended for a homogeneous population and 2.1 is recommended for a more heterogeneous population
	2.1	-	
$R_{fetal/maternal}$	0.9	-	Fetal/maternal PbB ratio
$PbB_{adult,\ 0}$	1.7-2.2	µg/dl	Typical blood lead concentration in adults in the absence of exposures to the site that is being assessed
BKSF	0.4	µg/dl per µg/day	Biokenetic slope factor relating increase in typical adult blood lead concentration to average daily lead uptake
IR_s	0.05	g/day	Intake rate of soil, including both outdoor soil and indoor soil-derived dust
EF_s	219	day/yr	Exposure frequency for contact with assessed soils and/or dust derived in part from these soils.
AF_s	0.12	-	Absolute gastrointestinal absorption fraction for ingested lead in soil and lead in dust derived from soil

Table 6-25a Four Year Old Children's Baseline Lead Intake Rates for Ingestion of Soil and Dust - EPA Default

Area	Community Average Soil Conc. (mg/kg)	Soil Lead Intake (mg/day)	Community Average Dust Conc. (mg/kg)	Dust Lead Intake (mg/day)	Air Lead Intake (mg/day)	Water Lead Intake (mg/day)	Market Basket Lead Intake (mg/day)	Total Lead Intake (mg/day)
Mullan	628	28	985	54	0.5	2	6	91
Burke/Nine Mile	679	31	879	48	0.5	2	6	88
Wallace	771	35	1004	55	0.5	2	6	99
Silverton	352	16	557	31	0.5	2	6	55
Osburn	418	19	493	27	0.5	2	6	55
Side Gulches	373	17	695	38	0.5	2	6	64
Kingston	257	12	466	26	0.5	2	6	46
Lower Basin	110	5	301	17	0.5	2	6	30

Table 6-25b Four Year Old Children's Baseline Lead Intake Rates for Ingestion of Soil and Dust - Box Model

Area	Community Average Soil Conc. (mg/kg)	Soil Lead Intake (mg/day)	Community Average Dust Conc. (mg/kg)	Dust Lead Intake (mg/day)	Air Lead Intake (mg/day)	Water Lead Intake (mg/day)	Market Basket Lead Intake (mg/day)	Total Lead Intake (mg/day)
Mullan	628	38	985	39	0.5	2	6	86
Burke/Nine Mile	679	41	879	35	0.5	2	6	85
Wallace	771	46	1004	40	0.5	2	6	95
Silverton	352	21	557	22	0.5	2	6	52
Osburn	418	25	493	20	0.5	2	6	54
Side Gulches	373	22	695	28	0.5	2	6	59
Kingston	257	15	466	19	0.5	2	6	43
Lower Basin	110	7	301	12	0.5	2	6	27

Table 6-26 Adult Baseline Lead Intake Rates for Ingestion of Soil and Dust

	Soil Concentration (mg/kg)	Soil Intake (mg/day)	Dust Concentration (mg/kg)	Dust Intake (mg/day)	Total Intake (mg/day)
Mullan	628	14	985	27	40
Burke/Nine Mile	679	15	879	24	39
Wallace	771	17	1004	28	44
Silverton	352	8	557	15	23
Osburn	418	9	493	14	23
Side Gulches	373	8	695	19	27
Kingston	257	6	466	13	18
Lower Basin	110	2	301	8	10

Table 6-27a Adult Occupational Exposure Factors for Non-lead Soil Related Activities Ingestion

CT Values			RME Values		
Ingestion Rate (mg/day)	Exposure Frequency (days/year)	Averaging Time (days/year)	Ingestion Rate (mg/day)	Exposure Frequency (days/year)	Averaging Time (days/year)
200	43[a]	365	300	195[b]	365

[a]Equal to 8.7 weeks/year x 5 days/week; assumes a two month construction project.

[b]Equal to 39 weeks/year x 5 days/week; assumes a nine month construction season.

Table 6-27b Adult Occupational Exposure Factors for Lead Soil Related Activities Ingestion

CT Values			RME Values		
Ingestion Rate (mg/day)	Exposure Frequency (days/year)	Averaging Time (days/year)	Ingestion Rate (mg/day)	Exposure Frequency (days/year)	Averaging Time (days/year)
100	43[a]	365	200	195[b]	365

[a]Equal to 8.7 weeks/year x 5 days/week; assumes a two month construction project.

[b]Equal to 39 weeks/year x 5 days/week; assumes a nine month construction season.

Table 6-27c EPA Adult Lead Default Values for Non-soil Related Activities

EPA Adult Model Default Values		
Ingestion Rate (mg/day)	Exposure Frequency (days/year)	Averaging Time (days/year)
50	219	365

Intake (ug/day) = C_{soil} x IR_{soil} x EF x CF x 1/AT

C_{soil} = soil lead concentration (mg/kg)

IR_{soil} = soil ingestion rate (mg/day)

EF = exposure frequency (days/year)

CF = unit correction factor

AT = averaging time (days/year)

Table 6-28a Estimated Occupational Intake Rates Associated with Community Soils for Geographic Subareas Using Non-Lead Ingestion Rates

	Community Average Soil Concentration (mg/kg)	Baseline Intake (mg/day)	Central Tendency (CT)			Reasonable Maximum Exposure (RME)		
			Soil Intake Averaged Over the Year (mg/day)	Total Intake (mg/day)	% Intake Increase from Baseline	Soil Intake Averaged Over the Year (mg/day)	Total Intake (mg/day)	% Intake Increase from Baseline
Mullan	628	40	15	55	37%	101	141	250%
Burke/Nine Mile	679	39	16	55	41%	109	148	276%
Wallace	771	44	18	63	41%	124	168	279%
Silverton	352	23	8	31	36%	56	79	248%
Osburn	418	23	10	33	43%	67	90	294%
Side Gulches	373	27	9	36	33%	60	87	224%
Kingston	257	18	6	24	33%	41	59	228%
Lower Basin	110	10	3	13	25%	18	28	171%

Table 6-28b Estimated Occupational Intake Rates Associated with Community Soils for Geographic Subareas Using EPA Adult Model Ingestion Rates

	Community Average Soil Concentration (mg/kg)	Baseline Intake (mg/day)	Central Tendency (CT)			Reasonable Maximum Exposure (RME)		
			Soil Intake Averaged Over the Year (mg/day)	Total Intake (mg/day)	% Intake Increase from Baseline	Soil Intake Averaged Over the Year (mg/day)	Total Intake (mg/day)	% Intake Increase from Baseline
Mullan	628	40	7	48	18%	67	107	166%
Burke/Nine Mile	679	39	8	47	20%	73	112	184%
Wallace	771	44	9	53	20%	82	127	186%
Silverton	352	23	4	27	18%	38	60	165%
Osburn	418	23	5	28	22%	45	67	196%
Side Gulches	373	27	4	31	16%	40	67	149%
Kingston	257	18	3	21	17%	27	46	152%
Lower Basin	110	10	1	12	13%	12	22	114%

Table 6-29a Estimated Occupational Incremental Lead Intake Rates for Potential Soil Lead Concentration Levels using Non-lead Ingestion Rates

Soil Concentration (mg/kg)	Central Tendency (CT)		Reasonable Maximum Exposure (RME)	
	Soil Intake (mg/day)	Soil Intake Averaged Over the Year (mg/day)	Soil Intake (mg/day)	Soil Intake Averaged Over the Year (mg/day)
500	61	12	105	80
1000	123	24	210	160
1500	184	35	315	240
2000	246	47	419	321
2500	307	59	524	401
3000	369	71	629	481
3500	430	82	734	561
4000	491	94	839	641
4500	553	106	944	721
5000	614	118	1048	801
5500	676	130	1153	882
6000	737	141	1258	962

Table 6-29b Estimated Occupational Incremental Lead Intake Rates for Potential Soil Lead Concentration Levels using Lead Soil Related Ingestion Rates

Soil Concentration (mg/kg)	Central Tendency (CT)		Reasonable Maximum Exposure (RME)	
	Soil Intake (mg/day)	Soil Intake Averaged Over the Year (mg/day)	Soil Intake (mg/day)	Soil Intake Averaged Over the Year (mg/day)
500	31	6	70	53
1000	61	12	140	107
1500	92	18	210	160
2000	123	24	280	214
2500	154	29	349	267
3000	184	35	419	321
3500	215	41	489	374
4000	246	47	559	427
4500	276	53	629	481
5000	307	59	699	534
5500	338	65	769	588
6000	369	71	839	641

Table 6-30 Occupational Lead Intake Rates for Adult Occupational Scenarios

6-30a Percentile Lead Concentrations for Occupational Soils

Area	10% Surface Soil Concentration (mg/kg)	25% Surface Soil Concentration (mg/kg)	50% Surface Soil Concentration (mg/kg)	75% Surface Soil Concentration (mg/kg)	90% Surface Soil Concentration (mg/kg)	95% Surface Soil Concentration (mg/kg)
Mullan	176	320	678	1516	2804	3860
Burke/Nine Mile	121	500	833	1609	2558	3520
Wallace	271	527	876	1525	2829	3803
Silverton	124	193	378	636	1003	1724
Osburn	134	264	406	692	1338	2163
Side Gulches	143	256	463	649	930	1441
Kingston	76	131	208	468	1392	5427
Lower Basin	26	46	94	940	3930	4783
Lower Basin floodplain sediment	0	980	3110	4070	4840	5750

6-30b Percentile Lead Intake Rates for Occupational Soils

Area	Baseline Intake (mg/day)	10% Intake Value (mg/day)	25% Intake Value (mg/day)	50% Intake Value (mg/day)	75% Intake Value (mg/day)	90% Intake Value (mg/day)	95% Intake Value (mg/day)
Mullan	40	2	4	8	18	33	45
Burke/Nine Mile	39	1	6	10	19	30	41
Wallace	44	3	6	10	18	33	45
Silverton	23	1	2	4	7	12	20
Osburn	23	2	3	5	8	16	25
Side Gulches	27	2	3	5	8	11	17
Kingston	18	1	2	2	6	16	64
Lower Basin	10	0	1	1	11	46	56
Lower Basin floodplain sediment	10	0	12	37	48	57	68

6-30c Central Tendency and Reasonable Maximum Exposure Lead Intakes Compared to Baseline

Area	Baseline Intake (mg/day)	Central Tendency (CT)			Reasonable Maximum Exposure (RME)		
		50% Soil Intake Value (mg/day)	Total Intake (mg/day)	% Intake Increase from Baseline	95% Soil Intake Value (mg/day)	Total Intake (mg/day)	% Intake Increase from Baseline
Mullan	40	8	48	20%	45	86	113%
Burke/Nine Mile	39	10	49	25%	41	81	105%
Wallace	44	10	55	23%	45	89	101%
Silverton	23	4	27	20%	20	43	89%
Osburn	23	5	28	21%	25	48	112%
Side Gulches	27	5	32	20%	17	44	64%
Kingston	18	2	21	14%	64	82	354%
Lower Basin	10	1	11	11%	56	67	549%
Lower Basin floodplain sediment	10	37	47	366%	68	78	677%

Table 6-31 Recreational Exposure Factors

Scenario	Population	CT Values			RME Values		
		Ingestion Rate (mg/day)	Exposure Frequency[a] (days/year)	Averaging Time[b] (days/year)	Ingestion Rate (mg/day)	Exposure Frequency[a] (days/year)	Averaging Time[b] (days/year)
Upland Parks Soil	Adults	50	15	238[c]	100	30	238[c]
	Children (0-6)	120	17	238[c]	300	34	238[c]
Beach Sediment	Adults	50	16	224[d]	100	32	224[d]
(Public Beaches)	Children (0-6)	120	16	224[d]	300	32	224[d]
Waste Pile Soil	Children (4-11)	120	9	238[c]	300	17	238[c]
Neighborhood Sediment	Children (4-11)	120	10	168[e]	300	21	168[e]

[a]Exposure Frequency = $\frac{\text{\# weeks per year x \# waking hours at scenario per day x \# days per week}}{\text{\# waking hours per day}}$

Number of waking hours per day for adults is 16 hours; for children it is 14 hours.

Examples:

17 days/year = $\frac{\text{34 weeks/year x 7 hours/day x 1 day/week}}{\text{14 hours/day}}$

16 events/year = 16 weeks/year x 1 full day event/week

8.5 days/year = $\frac{\text{34 weeks/year x 7 hours/day x 0.5 day/week}}{\text{14 hours/day}}$

10 days/year = $\frac{\text{24 weeks/year x 3 hours/day x 2 days/week}}{\text{14 hours/day}}$

[b]Effective exposure duration is 365 days/year in the IEUBK model, whereas expected exposure is seasonal (as shown in the CT and RME values).

[c]Averaging time is equal to 34 weeks/year x 7 days/week = 238 days/year.

[d]Averaging time is equal to 32 weeks/year x 7 days/week = 224 days/year.

[e]Averaging time is equal to 24 weeks/year x 7 days/week = 168 days/year.

Intake (ug/day) = C_{soil} x IR_{soil} x EF x CF x 1/AT

C_{soil} = soil lead concentration (mg/kg)

IR_{soil} = soil ingestion rate (mg/day)

EF = exposure frequency (days/year)

CF = unit correction factor

AT = averaging time (days/year)

* Reference Tables 3-24 for rationale behind recreational exposure factors

Table 6-32a Incremental Sediment/Soil Ingestion Lead Intake for Children - Central Tendency (CT)

Concentration (mg/kg)	Upland Parks		Beach Sediment		Waste Piles		Neighborhood Sediment	
	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)
50	0	0	0	0	0	0	0	0
100	1	1	1	1	0	0	1	0
200	2	1	2	1	1	1	1	1
400	3	2	3	2	2	1	3	1
500	4	3	4	3	2	1	4	2
1000	9	6	9	5	4	3	7	3
1500	13	8	13	8	6	4	11	5
2000	17	11	17	11	9	6	14	7
2500	21	14	21	13	11	7	18	8
3000	26	17	26	16	13	8	21	10
3500	30	20	30	18	15	10	25	12
4000	34	22	34	21	17	11	29	13
4500	39	25	39	24	19	13	32	15
5000	43	28	43	26	21	14	36	16
5500	47	31	47	29	24	15	39	18
6000	51	34	51	32	26	17	43	20

Table 6-32b Incremental Sediment/Soil Ingestion Lead Intake for Children - Reasonable Maximum Exposure (RME)

Concentration (mg/kg)	Upland Parks		Beach Sediment		Waste Piles		Neighborhood Sediment	
	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)
50	2	1	2	1	1	1	2	1
100	4	3	4	3	2	1	4	2
200	9	6	9	5	4	3	8	3
400	17	11	17	11	9	6	15	7
500	21	14	21	13	11	7	19	9
1000	43	28	43	26	21	14	38	17
1500	64	42	64	39	32	21	56	26
2000	86	56	86	53	43	28	75	35
2500	107	70	107	66	54	35	94	43
3000	129	84	129	79	64	42	113	52
3500	150	98	150	92	75	49	131	60
4000	171	112	171	105	86	56	150	69
4500	193	126	193	118	96	63	169	78
5000	214	140	214	132	107	70	188	86
5500	236	154	236	145	118	77	206	95
6000	257	168	257	158	129	84	225	104

Table 6-32c Incremental Sediment/Soil Ingestion Intake for Adults - Central Tendency (CT) and Reasonable Maximum Exposure (RME)

Concentration (mg/kg)	Central Tendency (CT)				Reasonable Maximum Exposure (RME)			
	Upland Parks		Beach Sediment		Upland Parks		Beach Sediment	
	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)
50	0	0	0	0	1	0	1	0
100	0	0	0	0	1	1	1	1
500	2	1	2	1	6	4	7	4
1000	3	2	4	2	13	8	14	9
1500	5	3	5	3	19	12	21	13
2000	6	4	7	4	25	16	29	18
2500	8	5	9	5	32	21	36	22
3000	9	6	11	7	38	25	43	26
3500	11	7	13	8	44	29	50	31
4000	13	8	14	9	50	33	57	35
4500	14	9	16	10	57	37	64	39
5000	16	10	18	11	63	41	71	44
5500	17	11	20	12	69	45	79	48
6000	19	12	21	13	76	49	86	53

Table 6-33 Recreational Surface Water Exposure Factors

Scenario	Population	CT Values			RME Values		
		Ingestion Rate (ml/hour)	Exposure Frequency (events/year)	Averaging Time (days/year)	Ingestion Rate (ml/hour)	Exposure Frequency[a] (events/year)	Averaging Time[b] (days/year)
Recreational (Public Beaches)	Adults	30	16	112[c]	30	32	112[c]
	Children	30	16	112[c]	30	32	112[c]
Neighborhood Sediment	Children	30	48	168[d]	30	96	168[d]

[a]Exposure Frequency = # events per week x # weeks per year, where one event is a full day at the beach.

Examples:
32 events/year = 2 events/week x 16 weeks/year
96 events/year = 4 events/week x 24 weeks/year
16 events/year = 1 event/week x 16 weeks/year
48 events/year = 2 events/week x 24 weeks/year

[b]Effective exposure duration is 365 days/year in the IEUBK model, whereas expected exposure is seasonal (as shown in the CT and RME values).

[c]Averaging time is equal to 16 weeks/year x 7 days/week = 112 days/year.

[d]Averaging time is equal to 24 weeks/year x 7 days/week = 168 days/year.

Intake (ug/day) = C_{water} x IR_{water} x EF x CF x 1/AT

C_{water} = water lead concentration (ug/L)

IR_{water} = water ingestion rate (ml/hour)

EF = exposure frequency (hourly events/year)

CF = unit correction factor

AT = averaging time (days/year)

Table 6-34a Incremental Recreational Surface Water Ingestion Lead Intake Rates for Children

Concentration (mg/L)	Central Tendency (CT)				Reasonable Maximum Exposure (RME)			
	Recreational		Neighborhood		Recreational		Neighborhood	
	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)
50	0	0	0	0	0	0	1	0
200	1	0	2	1	2	1	3	2
500	2	1	4	2	4	1	9	4
1000	4	1	9	4	9	3	17	8
5000	21	7	43	20	43	13	86	39
10000	43	13	86	39	86	26	171	79
15000	64	20	129	59	129	39	257	118
20000	86	26	171	79	171	53	343	158
25000	107	33	214	99	214	66	429	197
50000	214	66	429	197	429	131	857	395
55000	236	72	471	217	471	145	943	434
60000	257	79	514	237	514	158	1029	473
65000	279	85	557	256	557	171	1114	513

Table 6-34b Incremental Recreational Surface Water Ingestion Lead Intake Rates for Adults

Concentration (mg/L)	Central Tendency (CT)		Reasonable Maximum Exposure (RME)	
	Intake (mg/day)	Intake Averaged over the Year (mg/day)	Intake (mg/day)	Intake Averaged over the Year (mg/day)
50	0	0	0	0
200	1	0	2	1
500	2	1	4	1
1000	4	1	9	3
5000	21	7	43	13
10000	43	13	86	26
15000	64	20	129	39
20000	86	26	171	53
25000	107	33	214	66
50000	214	66	429	131
55000	236	72	471	145
60000	257	79	514	158
65000	279	85	557	171

Table 6-35 Upland Parks Incremental Intakes for Children

6-35a Percentile Surface Soil Lead Concentrations

Upland Park Area	10% Surface Soil Concentration (mg/kg)	25% Surface Soil Concentration (mg/kg)	50% Surface Soil Concentration (mg/kg)	75% Surface Soil Concentration (mg/kg)	90% Surface Soil Concentration (mg/kg)	95% Surface Soil Concentration (mg/kg)
Mullan	359	474	1189	2530	3270	3270
Mid Reach[a]	198	309	808	1570	3170	12100
Lower Basin[b]	61	1070	3415	4350	4710	4900

[a] Mid Reach includes Silverton, Osburn, Wallace, and the Side Gulches.

[b] Lower Basin includes Kingston.

6-35b Lead Intakes Associated with Concentration Percentiles

	Baseline Intake (mg/day)	10% Intake Value (mg/day)	25% Intake Value (mg/day)	50% Intake Value (mg/day)	75% Intake Value (mg/day)	90% Intake Value (mg/day)	95% Intake Value (mg/day)
Mullan	91	2	3	7	14	18	18
Wallace	99	1	2	5	9	18	68
Silverton	55	1	2	5	9	18	68
Osburn	55	1	2	5	9	18	68
Side Gulches	64	1	2	5	9	18	68
Kingston	46	0	6	19	24	26	27
Lower Basin	30	0	6	19	24	26	27

6-35c Incremental Central Tendency and Reasonable Maximum Exposure Intake Rates Compared to Baseline

		Central Tendency (CT)			Reasonable Maximum Exposure (RME)		
	Baseline Intake (mg/day)	50% Intake Value (mg/day)	Total Intake (mg/day)	% Intake Increase from Baseline	95% Intake Value (mg/day)	Total Intake (mg/day)	% Intake Increase from Baseline
Mullan	91	7	98	7%	18	109	20%
Wallace	99	5	103	5%	68	166	69%
Silverton	55	5	60	8%	68	123	123%
Osburn	55	5	59	8%	68	122	124%
Side Gulches	64	5	68	7%	68	131	106%
Kingston	46	19	65	42%	27	73	59%
Lower Basin	30	19	49	63%	27	58	91%

Table 6-36 Neighborhood Stream Incremental Intakes for Children 4 Years Through 11 Years Old

6-36a Percentile Lead Concentration for Sediment and Surface Water

Area	10% Sediment Concentration (mg/kg)	10% Surface Water Concentration (µg/L)	25% Sediment Concentration (mg/kg)	25% Surface Water Concentration (µg/L)	50% Sediment Soil Concentration (mg/kg)	50% Surface Water Concentration (µg/L)	75% Sediment Concentration (mg/kg)	75% Surface Water Concentration (µg/L)	90% Sediment Concentration (mg/kg)	90% Surface Water Concentration (µg/L)	95% Sediment Concentration (mg/kg)	95% Surface Water Concentration (µg/L)
Mullan	114	0.13	361	0.3	696	1.3	1850	5.0	5270	11	13800	19
Burke/Nine Mile	160	1.0	858	6.4	4030	36	6930	74	20100	129	67100	296
Kingston*	83.9	0.2	110	0.4	153	0.65	218	1.7	228	12	249	38
Osburn, Wallace, Silverton, Side Gulches	1010	0.3	1490	0.5	2305	1.65	4450	21.9	5720	31.2	5880	36.8

*The Lower Basin subarea is included with Kingston.

6-36b Percentile Lead Intake Rates for Sediment and Surface Water

	Baseline Intake (µg/day)	10% Sediment Intake Value (µg/day)	10% Surface Water Intake Value (µg/day)	25% Sediment Intake Value (µg/day)	25% Surface Water Intake Value (mg/day)	50% Sediment Intake Value (mg/day)	50% Surface Water Intake Value (mg/day)	75% Sediment Intake Value (µg/day)	75% Surface Water Intake Value (mg/day)	90% Sediment Intake Value (µg/day)	90% Surface Water Intake Value (µg/day)	95% Sediment Intake Value (µg/day)	95% Surface Water Intake Value (mg/day)
Mullan	91	0	0	1	0	2	0	6	0	17	0	45	0
Burke/Nine Mile	88	1	0	3	0	13	0	23	0	66	1	221	1
Wallace	99	3	0	5	0	8	0	15	0	19	0	19	0
Silverton	55	3	0	5	0	8	0	15	0	19	0	19	0
Osburn	55	3	0	5	0	8	0	15	0	19	0	19	0
Side Gulches	64	3	0	5	0	8	0	15	0	19	0	19	0
Kingston	46	0	0	0	0	1	0	1	0	1	0	1	0
Lower Basin	30	0	0	0	0	1	0	1	0	1	0	1	0

6-36c Central Tendency and Reasonable Maximum Exposure Intake Estimates Compared to Baseline

		Central Tendency (CT)				Reasonable Maximum Exposure (RME)			
	Baseline Intake (µg/day)	50% Sediment Intake Value (µg/day)	50% Surface Water Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	95% Sediment Intake Value (µg/day)	95% Surface Water Intake Value (mg/day)	Total Intake (µg/day)	% Intake Increase from Baseline
Mullan	91	2	0	93	3%	45	0	137	50%
Burke/Nine Mile	88	13	0	101	15%	221	1	309	253%
Wallace	99	8	0	106	8%	19	0	118	20%
Silverton	55	8	0	63	14%	19	0	75	35%
Osburn	55	8	0	62	14%	19	0	74	36%
Side Gulches	64	8	0	71	12%	19	0	83	31%
Kingston	46	1	0	46	1%	1	0	47	2%
Lower Basin	30	1	0	31	2%	1	0	31	3%

Table 6-37 CUA Public Beach Incremental Intakes for Children 0 Through 6 Years Old

6-37a Percentile Lead Concentrations for Sediments and Surface Water

Area	10% Sediment Concentration (mg/kg)	10% Surface Water Concentration (µg/L)	25% Sediment Concentration (mg/kg)	25% Surface Water Concentration (µg/L)	50% Sediment Soil Concentration (mg/kg)	50% Surface Water Concentration (µg/L)	75% Sediment Concentration (mg/kg)	75% Surface Water Concentration (µg/L)	90% Sediment Concentration (mg/kg)	90% Surface Water Concentration (µg/L)	95% Sediment Concentration (mg/kg)	95% Surface Water Concentration (µg/L)
Lower Basin[a]	1250	658	2720	3010	3690	14700	4410	25300	5000	30800	5750	31700

[a] Lower Basin includes Kingston.

6-37b Percentile Lead Intake Rates by Media

	Baseline Intake (µg/day)	10% Sediment Intake Value (µg/day)	10% Surface Water Intake Value (µg/day)	25% Sediment Intake Value (µg/day)	25% Surface Water Intake Value (µg/day)	50% Sediment Intake Value (µg/day)	50% Surface Water Intake Value (µg/day)	75% Sediment Intake Value (µg/day)	75% Surface Water Intake Value (µg/day)	90% Sediment Intake Value (µg/day)	90% Surface Water Intake Value (µg/day)	95% Sediment Intake Value (µg/day)	95% Surface Water Intake Value (µg/day)
Mullan	91	7	1	14	4	19	19	23	33	26	41	30	42
Burke/Nine Mile	88	7	1	14	4	19	19	23	33	26	41	30	42
Wallace	99	7	1	14	4	19	19	23	33	26	41	30	42
Silverton	55	7	1	14	4	19	19	23	33	26	41	30	42
Osburn	55	7	1	14	4	19	19	23	33	26	41	30	42
Side Gulches	64	7	1	14	4	19	19	23	33	26	41	30	42
Kingston	46	7	1	14	4	19	19	23	33	26	41	30	42
Lower Basin	30	7	1	14	4	19	19	23	33	26	41	30	42

6-37c Central Tendency and Reasonable Maximum Exposure Lead Intake Rates Compared to Baseline

		Central Tendency (CT)				Reasonable Maximum Exposure (RME)			
	Baseline Intake (µg/day)	50% Sediment Intake Value (µg/day)	50% Surface Water Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	95% Sediment Intake Value (µg/day)	95% Surface Water Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline
Mullan	91	19	19	130	43%	30	42	163	79%
Burke/Nine Mile	88	19	19	126	44%	30	42	160	82%
Wallace	99	19	19	137	39%	30	42	171	73%
Silverton	55	19	19	94	70%	30	42	127	130%
Osburn	55	19	19	93	71%	30	42	127	132%
Side Gulches	64	19	19	102	61%	30	42	136	113%
Kingston	46	19	19	85	84%	30	42	118	157%
Lower Basin	30	19	19	69	128%	30	42	102	238%

Table 6-38 Incremental Lead Intake Rates for Waste Piles for Children 4 Through 11 Years Old

6-38a Percentile Lead Concentrations for Waste Pile Soils

Area	10% Surface Soil Concentration (mg/kg)	25% Surface Soil Concentration (mg/kg)	50% Surface Soil Concentration (mg/kg)	75% Surface Soil Concentration (mg/kg)	90% Surface Soil Concentration (mg/kg)	95% Surface Soil Concentration (mg/kg)
Mullan	228	234	245	4080	4570	4570
Burke/Nine Mile	306	2050	3970	4750	16100	49800

6-38b Percentile Lead Intake Rates for Waste Piles

	Baseline Intake (µg/day)	10% Intake Value (µg/day)	25% Intake Value (µg/day)	50% Intake Value (µg/day)	75%Intake Value (µg/day)	90% Intake Value (µg/day)	95% Intake Value (µg/day)
Mullan	91	1	1	1	11	13	13
Burke/Nine Mile	88	1	6	11	13	45	139

6-38c Central Tendency and Reasonable Maximum Exposure Incremental Lead Intakes Compared to Baseline

		Central Tendency (CT)			Reasonable Maximum Exposure (RME)		
	Baseline Intake (µg/day)	50% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	95% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline
Mullan	91	1	92	1%	13	104	14%
Burke/Nine Mile	88	11	99	13%	139	227	159%

Table 6-39 Local Foodstuff Exposure Factors for the Resident Populations

Scenario	Population	CT Values			RME Values		
		Ingestion Rate	Exposure Frequency (days/year)	Averaging Time (days/year)	Ingestion Rate	Exposure Frequency (days/year)	Averaging Time (days/year)
Recreational fishermen	Adults/Children	0.357 g/kg-day[a]	365	365	0.657 g/kg-day[a]	365	365
Home grown vegetables	Adults/Children	0.492 g/kg-day[b]	365	365	5.04 g/kg-day[b,c]	365	365

[a]Fish ingestion rate is calculated using the EPA suggested CT value of 25 g/day for adults and the ATSDR RME value of 46 g/day for adults. The ingestion rate becomes 0.357 g/kg-day when the adult body weight, 70 kg, is factored out. Ingestion rates become g/day when 0.357 g/kg-day is multiplied by body weight (70 kg for adults and 15 kg for children).

Examples: 5.4 g/day = 0.357 g/kg-day x 15 kg child
25 g/day = 0.357 g/kg-day x 70 kg adult

[b]Ingestion rate is seasonally adjusted and incorporates the body weights of all participants in the study (children and adults).

[c]Ingestion rate units become g/day when 0.492 g/kg-day is multiplied by body weight (70 kg for adults and 15 kg for children).

Examples: 7.4 g/day = 0.492 g/kg-day x 15 kg child
34 g/day = 0.492 g/kg-day x 70 kg adult

Fish intake (ug/day) = C_{fish} x IR_{fish} x EF x CF x 1/AT
C_{fish} = fish tissue lead concentration (mg/kg)
IR_{fish} = food ingestion rate for fish (g/day)
EF = exposure frequency (days/year)
CF = unit correction factor
AT = averaging time (days/year)

Vegetable intake (ug/day) = C_{veg} x IR_{veg} x EF x CF x BW x 1/AT
C_{veg} = wet weight vegetable lead concentration (mg/kg)
IR_{veg} = food ingestion rate for vegetables (g/kg-day)
EF = exposure frequency (days/year)
CF = unit correction factor
BW = body weight (kg)
AT = averaging time (days/year)

Table 6-40a Children's Incremental Lead Intake Rate from Home Grown Produce

Area	10% Wet Weight Vegetable Concentration (mg/kg)	25% Wet Weight Vegetable Concentration (mg/kg)	50% Wet Weight Vegetable Concentration (mg/kg)	75% Wet Weight Vegetable Concentration (mg/kg)	90% Wet Weight Vegetable Concentration (mg/kg)	95% Wet Weight Vegetable Concentration (mg/kg)
Total Basin	0.6	1.2	3.2	12	19	24

Area	10% Intake Value (µg/day)	25% Intake Value (µg/day)	50% Intake Value (µg/day)	75% Intake Value (µg/day)	90% Intake Value (µg/day)	95% Intake Value (µg/day)
Total Basin	4	9	24	85	139	178

		Central Tendency (CT)			Reasonable Maximum Exposure (RME)		
	Baseline Intake (µg/day)	50% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	95% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline
Mullan	91	24	115	26%	178	269	195%
Burke/Nine Mile	88	24	111	27%	178	265	203%
Wallace	99	24	122	24%	178	277	180%
Silverton	55	24	79	43%	178	233	322%
Osburn	55	24	78	44%	178	233	325%
Side Gulches	64	24	88	37%	178	242	279%
Kingston	46	24	70	52%	178	224	388%
Lower Basin	30	24	54	79%	178	208	589%

Table 6-40b Adults' Incremental Lead Intake Rate from Home Grown Foodstuff

Area	10% Wet Weight Vegetable Concentration (mg/kg)	25% Wet Weight Vegetable Concentration (mg/kg)	50% Wet Weight Vegetable Concentration (mg/kg)	75% Wet Weight Vegetable Concentration (mg/kg)	90% Wet Weight Vegetable Concentration (mg/kg)	95% Wet Weight Vegetable Concentration (mg/kg)
Total Basin	0.6	1.2	3.2	12	19	24

Area	10% Intake Value (µg/day)	25% Intake Value (µg/day)	50% Intake Value (µg/day)	75% Intake Value (µg/day)	90% Intake Value (µg/day)	95% Intake Value (µg/day)
Total Basin	20	40	111	399	648	830

		Central Tendency (CT)			Reasonable Maximum Exposure (RME)		
	Baseline Intake (µg/day)	50% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	95% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline
Mullan	40	111	151	276%	830	870	2058%
Burke/Nine Mile	39	111	150	285%	830	869	2131%
Wallace	44	111	156	250%	830	874	1870%
Silverton	23	111	134	489%	830	853	3653%
Osburn	23	111	134	488%	830	853	3644%
Side Gulches	27	111	138	416%	830	857	3108%
Kingston	18	111	129	615%	830	848	4593%
Lower Basin	10	111	121	1083%	830	840	8087%

Table 6-41a Recreational Fish Incremental Intakes for Children

Area	10% Fish Fillet Concentration (mg/kg)	25% Fish Fillet Concentration (mg/kg)	50% Fish Fillet Concentration (mg/kg)	75% Fish Fillet Concentration (mg/kg)	90% Fish Fillet Concentration (mg/kg)	95% Fish Fillet Concentration (mg/kg)
Total Basin	0.03	0.06	0.12	0.27	0.51	0.68

Area	10% Intake Value (µg/day)	25% Intake Value (µg/day)	50% Intake Value (µg/day)	75% Intake Value (µg/day)	90% Intake Value (µg/day)	95% Intake Value (µg/day)
Total Basin	0	0	1	1	3	4

	Baseline Intake (µg/day)	Central Tendency (CT)			Reasonable Maximum Exposure (RME)		
		50% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	95% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline
Mullan	91	1	92	1%	4	95	4%
Burke/Nine Mile	88	1	88	1%	4	91	4%
Wallace	99	1	99	1%	4	102	4%
Silverton	55	1	56	1%	4	59	7%
Osburn	55	1	55	1%	4	58	7%
Side Gulches	64	1	64	1%	4	67	6%
Kingston	46	1	47	1%	4	50	8%
Lower Basin	30	1	31	2%	4	34	12%

Table 6-41b Recreational Fish Incremental Intakes for Adults

Area	10% Fish Fillet Concentration (mg/kg)	25% Fish Fillet Concentration (mg/kg)	50% Fish Fillet Concentration (mg/kg)	75% Fish Fillet Concentration (mg/kg)	90% Fish Fillet Concentration (mg/kg)	95% Fish Fillet Concentration (mg/kg)
Total Basin	0.03	0.06	0.12	0.27	0.51	0.68

Area	10% Intake Value (µg/day)	25% Intake Value (µg/day)	50% Intake Value (µg/day)	75% Intake Value (µg/day)	90% Intake Value (µg/day)	95% Intake Value (µg/day)
Total Basin	1	2	3	7	13	17

		Central Tendency (CT)			Reasonable Maximum Exposure (RME)		
	Baseline Intake (µg/day)	50% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	95% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline
Mullan	40	3	43	7%	17	57	42%
Burke/Nine Mile	39	3	42	8%	17	56	43%
Wallace	44	3	47	7%	17	61	38%
Silverton	23	3	26	13%	17	40	75%
Osburn	23	3	26	13%	17	40	75%
Side Gulches	27	3	30	11%	17	44	64%
Kingston	18	3	21	17%	17	35	94%
Lower Basin	10	3	13	29%	17	27	166%

Table 6-42a Summary of Children's Potential Incremental Intakes (CT)

		Upland Parks	Neighborhood Streams		Public Beaches		Waste Piles	Produce	Fish	Total
	Baseline Intake (mg/day)	50% Intake Value (mg/day)	50% Sediment Intake Value (mg/day)	50% Surface Water Intake Value (mg/day)	50% Sediment Intake Value (mg/day)	50% Surface Water Intake Value (mg/day)	50% Intake Value (mg/day)	50% Intake Value (mg/day)	50% Intake Value (mg/day)	50% Intake Value (mg/day)
Mullan	91	7	2	0	19	19	1	24	1	164
Burke/Nine Mile	88	-	13	0	19	19	11	24	1	175
Wallace	99	5	8	0	19	19	-	24	1	174
Silverton	55	5	8	0	19	19	-	24	1	130
Osburn	55	5	8	0	19	19	-	24	1	130
Side Gulches	64	5	8	0	19	19	-	24	1	139
Kingston	46	19	1	0	19	19	-	24	1	129
Lower Basin	30	19	1	0	19	19	-	24	1	113

Table 6-42b Summary of Children's Potential Incremental Intakes (RME)

		Upland Parks	Neighborhood Streams		Public Beaches		Waste Piles	Produce	Fish	Total
	Baseline Intake (mg/day)	95% Intake Value (mg/day)	95% Sediment Intake Value (mg/day)	95% Surface Water Intake Value (mg/day)	95% Sediment Intake Value (mg/day)	95% Surface Water Intake Value (mg/day)	95% Intake Value (mg/day)	95% Intake Value (mg/day)	95% Intake Value (mg/day)	95% Intake Value (mg/day)
Mullan	91	18	45	0	30	42	13	178	4	421
Burke/Nine Mile	88	-	221	1	30	42	139	178	4	702
Wallace	99	68	19	0	30	42	-	178	4	439
Silverton	55	68	19	0	30	42	-	178	4	396
Osburn	55	68	19	0	30	42	-	178	4	395
Side Gulches	64	68	19	0	30	42	-	178	4	404
Kingston	46	19	1	0	30	42	-	178	4	320
Lower Basin	30	27	1	0	30	42	-	178	4	312

Table 6-43a Summary of Adult's Potential Incremental Intakes (CT)

		Upland Parks	Public Beaches		Produce	Fish	Total
	Baseline Intake (mg/day)	50% Intake Value (mg/day)	50% Sediment Intake Value (mg/day)	50% Surface Water Intake Value (mg/day)	50% Intake Value (mg/day)	50% Intake Value (mg/day)	Intake Value (mg/day)
Mullan	40	2	8	19	111	3	184
Burke/Nine Mile	39	2	8	19	111	3	183
Wallace	44	2	8	19	111	3	188
Silverton	23	2	8	19	111	3	166
Osburn	23	2	8	19	111	3	166
Side Gulches	27	2	8	19	111	3	170
Kingston	18	7	8	19	111	3	167
Lower Basin	10	7	8	19	111	3	159

Table 6-43b Summary of Adult's Potential Incremental Intakes (RME)

		Upland Parks	Public Beaches		Produce	Fish	Total
	Baseline Intake (mg/day)	95% Intake Value (mg/day)	95% Sediment Intake Value (mg/day)	95% Surface Water Intake Value (mg/day)	95% Intake Value (mg/day)	95% Intake Value (mg/day)	Intake Value (mg/day)
Mullan	40	7	13	42	830	17	948
Burke/Nine Mile	39	25	13	42	830	17	966
Wallace	44	25	13	42	830	17	971
Silverton	23	25	13	42	830	17	949
Osburn	23	25	13	42	830	17	949
Side Gulches	27	25	13	42	830	17	953
Kingston	18	10	13	42	830	17	929
Lower Basin	10	10	13	42	830	17	922

Table 6-44a Traditional Tribal Lead Intake Rate Estimates for Children by Quantile Percentages

Intake Rate Estimates With Whole Fish and Unpeeled Water Potato

Quantile Percent Concentration	Soil Concentration (mg/kg)	Sediment Concentration (mg/kg)	Soil/Sediment Weighted Concentration (mg/kg)	Soil/Sediment Intake (µg/day)	Undisturbed Surface Water Concentration (µg/L)	Water Intake as Drinking Water (µg/day)	Whole Fish Concentration[1] (mg/kg)	Whole Fish Intake (µg/day)	Wet Weight Water Potato Concentration[2] (mg/kg)	Water Potato Intake (µg/day)	Surface Water Concentration (µg/L)	Incidental Ingestion of Surface Water (µg/day)	Total Intake (µg/day)
10%	61	0	39	12	9	13.5	0.56	65	2	236	658	11	338
25%	1070	980	1037	311	12	18	0.65	75	6	786	2340	40	1231
50%	3350	3110	3262	979	24	36	1.14	132	19	2310	15500	268	3724
75%	4350	4070	4248	1274	32	48	1.77	205	46	5610	25300	437	7574
90%	4710	4840	4757	1427	56	84	3.12	362	74	9092	30800	532	11497
95%	4900	5750	5210	1563	110	165	4.34	503	94	11599	31700	547	14377

[1]The fish ingestion pathway evaluation is based on whole fish metals data collected from the Spokane River; interpolation of hazards and risks associated with Lake Coeur d'Alene fish from these data is not recommended.

[2]The wet weight data are for unpeeled water potatoes.

Intake Rate Estimates With Fish Fillet and Peeled Water Potato

Quantile Percent Concentration	Soil Concentration (mg/kg)	Sediment Concentration (mg/kg)	Soil/Sediment Weighted Concentration (mg/kg)	Soil/Sediment Intake (µg/day)	Undisturbed Surface Water Concentration (µg/L)	Water Intake as Drinking Water (µg/day)	Fish Fillet Concentration[3] (mg/kg)	Fish Fillet Intake (µg/day)	Peeled Wet Weight Water Potato Concentration[4] (mg/kg)	Water Potato Intake (µg/day)	Surface Water Concentration (µg/L)	Incidental Ingestion of Surface Water (µg/day)	Total Intake (µg/day)
10%	61	0	39	12	9	13.5	0.03	3	0.29	36	658	11	75
25%	1070	980	1037	311	12	18	0.07	8	0.31	38	2340	40	416
50%	3350	3110	3262	979	24	36	0.13	15	0.34	41	15500	268	1339
75%	4350	4070	4248	1274	32	48	0.32	37	0.37	45	25300	437	1841
90%	4710	4840	4757	1427	56	84	0.56	65	0.41	50	30800	532	2158
95%	4900	5750	5210	1563	110	165	0.74	86	0.53	65	31700	547	2426

[3]The fish ingestion pathway evaluation is based on filleted fish metals data collected from the Lateral Lakes; interpolation of hazards and risks associated with Lake Coeur d'Alene fish from these data is not recommended.

[4]The wet weight data are for peeled water potatoes.

Table 6-44b Traditional Tribal Lead Intake Rate Estimates for Adults by Quantile Percentages

Intake Rate Estimates With Whole Fish and Unpeeled Water Potato

Quantile Percent Concentration	Soil Concentration (mg/kg)	Sediment Concentration (mg/kg)	Soil/Sediment Weighted Concentration (mg/kg)	Soil/Sediment Intake (µg/day)	Undisturbed Surface Water Concentration (µg/L)	Water Intake as Drinking Water (µg/day)	Whole Fish Concentration[1] (mg/kg)	Fish Intake (µg/day)	Wet Weight Water Potato Concentration[2] (mg/kg)	Water Potato Intake (µg/day)	Surface Water Concentration (µg/L)	Incidental Ingestion of Surface Water (µg/day)	Total Intake (µg/day)
10%	61	0	39	12	9	27	0.56	302	2	1102	658	11	1454
25%	1070	980	1037	311	12	36	0.65	351	6	3667	2340	40	4405
50%	3350	3110	3262	979	24	72	1.14	616	19	10780	15500	268	12714
75%	4350	4070	4248	1274	32	96	1.77	956	46	26180	25300	437	28943
90%	4710	4840	4757	1427	56	168	3.12	1685	74	42430	30800	532	46242
95%	4900	5750	5210	1563	110	330	4.34	2344	94	54127	31700	547	58910

[1]The fish ingestion pathway evaluation is based on whole fish metals data collected from the Spokane River; interpolation of hazards and risks associated with Lake Coeur d'Alene fish from these data is not recommended.

[2]The wet weight data are for unpeeled water potatoes.

Intake Rate Estimates With Fish Fillet and Peeled Water Potato

Quantile Percent Concentration	Soil Concentration (mg/kg)	Sediment Concentration (mg/kg)	Soil/Sediment Weighted Concentration (mg/kg)	Soil/Sediment Intake (µg/day)	Undisturbed Surface Water Concentration (µg/L)	Water Intake as Drinking Water (µg/day)	Fish Fillet Concentration[3] (mg/kg)	Fish Intake (µg/day)	Peeled Wet Weight Water Potato Concentration[4] (mg/kg)	Water Potato Intake (µg/day)	Surface Water Concentration (µg/L)	Incidental Ingestion of Surface Water (µg/day)	Total Intake (µg/day)
10%	61	0	39	12	9	27	0.03	14	0.29	167	658	11	231
25%	1070	980	1037	311	12	36	0.07	38	0.31	178	2340	40	603
50%	3350	3110	3262	979	24	72	0.13	70	0.34	193	15500	268	1582
75%	4350	4070	4248	1274	32	96	0.32	170	0.37	211	25300	437	2188
90%	4710	4840	4757	1427	56	168	0.56	302	0.41	234	30800	532	2663
95%	4900	5750	5210	1563	110	330	0.74	400	0.53	304	31700	547	3144

[3]The fish ingestion pathway evaluation is based on filleted fish metals data collected from the Lateral Lakes; interpolation of hazards and risks associated with Lake Coeur d'Alene fish from these data is not recommended.

[4]The wet weight data are for peeled water potatoes.

Table 6-45 Modern Tribal Lead Intake Rate Estimates for Children by Quantile Percentages

Intake Rate Estimates With Whole Fish and Unpeeled Water Potato

Quantile Percent Concentration	Soil Concentration (mg/kg)	Sediment Concentration (mg/kg)	Soil/Sediment Weighted Concentration (mg/kg)	Soil/Sediment Intake (µg/day)	Undisturbed Surface Water Concentration (µg/L)	Water Intake as Drinking Water (µg/day)	Whole Fish Concentration[1] (mg/kg)	Whole Fish Intake (µg/day)	Wet Weight Water Potato Concentration[2] (mg/kg)	Water Potato Intake (µg/day)	Surface Water Concentration (µg/L)	Incidental Ingestion of Surface Water (µg/day)	Total Intake (µg/day)
10%	61	0	31	2	9	2	0.56	20	2	46	658	3	73
25%	1070	980	1025	51	12	3	0.65	23	6	153	2340	12	243
50%	3350	3110	3230	162	24	6	1.14	41	19	451	15500	78	737
75%	4350	4070	4210	211	32	8	1.77	64	46	1095	25300	127	1504
90%	4710	4840	4775	239	56	14	3.12	112	74	1774	30800	154	2294
95%	4900	5750	5325	267	110	28	4.34	156	94	2263	31700	159	2873

[1]The fish ingestion pathway evaluation is based on whole fish metals data collected from the Spokane River; interpolation of hazards and risks associated with Lake Coeur d'Alene fish from these data is not recommended.

[2]The wet weight data are for unpeeled water potatoes.

Intake Rate Estimates With Fish Fillet and Peeled Water Potato

Quantile Percent Concentration	Soil Concentration (mg/kg)	Sediment Concentration (mg/kg)	Soil/Sediment Weighted Concentration (mg/kg)	Soil/Sediment Intake (µg/day)	Undisturbed Surface Water Concentration (µg/L)	Water Intake as Drinking Water (µg/day)	Fish Fillet Concentration[3] (mg/kg)	Fish Fillet Intake (µg/day)	Peeled Wet Weight Water Potato Concentration[4] (mg/kg)	Water Potato Intake (µg/day)	Surface Water Concentration (µg/L)	Incidental Ingestion of Surface Water (µg/day)	Total Intake (µg/day)
10%	61	0	31	2	9	2	0.03	1	0.29	7	658	3	15
25%	1070	980	1025	51	12	3	0.07	3	0.31	7	2340	12	76
50%	3350	3110	3230	162	24	6	0.13	5	0.34	8	15500	78	258
75%	4350	4070	4210	211	32	8	0.32	11	0.37	9	25300	127	366
90%	4710	4840	4775	239	56	14	0.56	20	0.41	10	30800	154	438
95%	4900	5750	5325	267	110	28	0.74	27	0.53	13	31700	159	493

[3]The fish ingestion pathway evaluation is based on filleted fish metals data collected from the Lateral Lakes; interpolation of hazards and risks associated with Lake Coeur d'Alene fish from these data is not recommended.

[4]The wet weight data are for peeled water potatoes.

Table 6-46 Modern Tribal Lead Intake Rate Estimates for Adults by Quantile Percentages

Intake Rate Estimates With Whole Fish and Unpeeled Water Potato

Quantile Percent Concentration	Soil Concentration (mg/kg)	Sediment Concentration (mg/kg)	Soil/Sediment Weighted Concentration (mg/kg)	Soil/Sediment Intake (µg/day)	Undisturbed Surface Water Concentration (µg/L)	Water Intake as Drinking Water (µg/day)	Whole Fish Concentration[1] (mg/kg)	Whole Fish Intake (µg/day)	Wet Weight Water Potato Concentration[2] (mg/kg)	Water Potato Intake (µg/day)	Surface Water Concentration (µg/L)	Incidental Ingestion of Surface Water (µg/day)	Total Intake (µg/day)
10%	61	0	31	2	9	5	0.56	95	2	215	658	3	320
25%	1070	980	1025	51	12	6	0.65	111	6	715	2340	12	895
50%	3350	3110	3230	162	24	12	1.14	194	19	2103	15500	78	2549
75%	4350	4070	4210	211	32	16	1.77	301	46	5108	25300	127	5763
90%	4710	4840	4775	239	56	28	3.12	530	74	8279	30800	154	9231
95%	4900	5750	5325	267	110	55	4.34	738	94	10561	31700	159	11780

[1]The fish ingestion pathway evaluation is based on whole fish metals data collected from the Spokane River; interpolation of hazards and risks associated with Lake Coeur d'Alene fish from these data is not recommended.
[2]The wet weight data are for unpeeled water potatoes.

Intake Rate Estimates With Fish Fillet and Peeled Water Potato

Quantile Percent Concentration	Soil Concentration (mg/kg)	Sediment Concentration (mg/kg)	Soil/Sediment Weighted Concentration (mg/kg)	Soil/Sediment Intake (µg/day)	Undisturbed Surface Water Concentration (µg/L)	Water Intake as Drinking Water (µg/day)	Fish Fillet Concentration[3] (mg/kg)	Fish Fillet Intake (µg/day)	Peeled Wet Weight Water Potato Concentration[4] (mg/kg)	Water Potato Intake (µg/day)	Surface Water Concentration (µg/L)	Incidental Ingestion of Surface Water (µg/day)	Total Intake (µg/day)
10%	61	0	31	2	9	5	0.03	4	0.29	33	658	3	46
25%	1070	980	1025	51	12	6	0.07	12	0.31	35	2340	12	116
50%	3350	3110	3230	162	24	12	0.13	22	0.34	38	15500	78	311
75%	4350	4070	4210	211	32	16	0.32	54	0.37	41	25300	127	449
90%	4710	4840	4775	239	56	28	0.56	95	0.41	46	30800	154	563
95%	4900	5750	5325	267	110	55	0.74	126	0.53	59	31700	159	666

[3]The fish ingestion pathway evaluation is based on filleted fish metals data collected from the Lateral Lakes; interpolation of hazards and risks associated with Lake Coeur d'Alene fish from these data is not recommended.
[4]The wet weight data are for peeled water potatoes.

Table 6-47a Predicted and Observed Baseline Blood Lead Levels
Community Mode IEUBK- 9-84 Months

Area	9-84 Months					
	Observed* Geometric Mean (µg/dl)	Observed* % to exceed 10 µg/dl	Default Predicted Geometric Mean (µg/dl)	Default Predicted % to exceed 10 µg/dl	40:30:30 Predicted Geometric Mean (µg/dl)	40:30:30 Predicted % to exceed 10 µg/dl
Mullan	5.0	10	9.3	43	6.3	15
Burke/Nine Mile	6.4	22	9	40	6.2	15
Wallace	5.7	19	9.9	48	6.8	19
Silverton	4.5	11	6.3	15	4.3	3
Osburn	4.0	5	6.2	15	4.4	4
Side Gulches	3.7	0	7	21	4.7	5
Kingston	3.7	14	5.4	9	3.7	2
Lower Basin	5.1	25	3.9	2	2.8	0

* observed levels correspond to children 9 months through 9 years of age

Table 6-47b Predicted and Observed Baseline Blood Lead Levels
Community Mode IEUBK- 9-24 Months

Area	9-24 Months					
	Observed* Geometric Mean (µg/dl)	Observed* % to exceed 10 µg/dl	Default Predicted Geometric Mean (µg/dl)	Default Predicted % to exceed 10 µg/dl	40:30:30 Predicted Geometric Mean (µg/dl)	40:30:30 Predicted % to exceed 10 µg/dl
Mullan	6.7	17	11.3	58	7.6	27
Burke/Nine Mile	6.6	30	11.0	56	7.5	25
Wallace	7.4	46	12.0	61	8.2	32
Silverton	6.1	19	7.6	27	5.2	8
Osburn	6.0	14	7.6	26	5.3	8
Side Gulches	5.0	0	8.6	34	5.7	11
Kingston	5.5	25	6.6	18	4.5	4
Lower Basin	6.9	33	4.6	5	3.2	1

* observed levels only correspond to 9-24 months

Table 6-47c Predicted and Observed Baseline Blood Lead Levels
Community Mode IEUBK- 9-60 Months

Area	9-60 Months					
	Observed* Geometric Mean (µg/dl)	Observed* % to exceed 10 µg/dl	Default Predicted Geometric Mean (µg/dl)	Default Predicted % to exceed 10 µg/dl	40:30:30 Predicted Geometric Mean (µg/dl)	40:30:30 Predicted % to exceed 10 µg/dl
Mullan	5.7	17	10.2	50	6.9	20
Burke/Nine Mile	6.4	22	9.9	48	6.8	20
Wallace	6.4	26	10.8	53	7.4	25
Silverton	5.0	13	6.9	20	4.7	5
Osburn	4.3	7	6.9	20	4.8	6
Side Gulches	3.9	0	7.7	27	5.2	8
Kingston	4.7	19	5.9	13	4.1	3
Lower Basin	6.1	29	4.2	3	3	0

* observed levels only correspond to 9-60 months

Note: 40:30:30 refers to the "Box Model" or 40% house dust: 30% yard soil: 30% community soil

Table 6-48 Summary of Blood Lead, Yard Soil, and Vacuum Dust for IEUBK Batch Mode Input

Area	Total Number of Observations	Minimum	Maximum	Arithmetic Mean	Standard Deviation	Geometric Mean	Geometric Standard Deviation
	Blood Lead (mg/dl)						
Mullan	27	2	12	5.1	3.0	4.4	1.8
Burke/Nine Mile	70	1	21	7.4	5.4	5.7	2.1
Wallace	56	1	19	5.3	3.6	4.4	1.9
Silverton	69	1	23	5.0	3.7	4.1	1.9
Osburn	95	1	13	4.1	2.4	3.4	1.9
Side Gulches	45	1	16	4.3	2.9	3.6	1.8
Kingston	45	1	16	5.2	4.2	3.9	2.1
Lower Basin/Cataldo	38	1	18	5.7	5.0	3.9	2.5
	Yard Soil (mg/kg)						
Mullan	105	41	20218	1187	2230	628	2.91
Burke/Nine Mile	88	32	5410	1105	973	679	3.25
Wallace	110	54	16026	1154	1628	771	2.47
Silverton	70	94	6098	524	763	352	2.25
Osburn	262	33	12884	682	1195	419	2.45
Side Gulches	100	25	3356	505	437	368	2.38
Kingston	99	22	9228	711	1622	257	3.34
Lower Basin/Cataldo	160	15	7350	487	1251	110	4.29
	Vacuum Dust (mg/kg)						
Mullan	32	429	4060	1146	754	985	1.70
Burke/Nine Mile	35	83	5800	1318	1263	879	2.63
Wallace	35	259	29725	1951	4944	1004	2.33
Silverton	26	75	3390	837	869	557	2.52
Osburn	84	23	2192	616	366	493	2.17
Side Gulches	26	116	3929	952	890	695	2.21
Kingston	30	102	1750	592	409	466	2.07
Lower Basin/Cataldo	31	49	3140	512	646	301	2.81

Table 6-49 IEUBK Batch Mode Overall Observed v. Predicted Blood Lead by Age

	9-24 Months			9-60 Months			9-84 Months		
	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)
N	75	75	75	222	222	222	311	311	311
Minimum (µg/dl)	2	2	2	1	2	2	1	2	2
Maximum (µg/dl)	23	27	17	23	28	17	23	28	17
Arithmetic Mean (µg/dl)	7.5	9.2	6.1	6.3	8.2	5.4	5.7	7.6	5.0
Geometric Mean (µg/dl)	6.2	7.9	5.5	5.1	7.2	4.9	4.6	6.6	4.5
Geometric Standard Deviation	1.82	2.08	1.91	1.90	1.96	1.86	1.93	2.02	1.90
% $\geq$ 10 µg/dl	23%	37%	17%	16%	31%	13%	13%	28%	11%
% $\geq$ 15 µg/dl	12%	19%	6%	8%	14%	4%	7%	12%	3%

Note: observed levels are for children 9-84 months or 0-7 years old as opposed to community mode showing observed levels for 0-9 year olds.

Table 6-50a IEUBK Batch Mode Overall Observed v. Predicted Blood Lead by Geographic Area, Ages 9-84 Months: Default and 40:30:30

	Mullan			Burke/Nine Mile			Wallace			Silverton		
	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)
N	15	15	15	46	46	46	39	39	39	55	55	55
Minimum (µg/dl)	2	5	4	1	4	3	1	6	4	2	2	2
Maximum (µg/dl)	12	16	9	21	27	17	19	21	13	23	19	11
Arithmetic Mean (µg/dl)	5.5	10.2	6.4	7.8	10.2	6.6	6.1	10.0	6.5	5.5	7.3	4.7
Geometric Mean (µg/dl)	4.7	9.7	6.1	6.3	9.2	6.1	5.2	9.6	6.3	4.6	6.7	4.5
Geometric Standard Deviation	1.75	2.08	1.73	1.98	1.76	1.78	1.82	1.64	1.70	1.81	1.86	1.79
% ≥ 10 µg/dl	13%	48%	19%	22%	44%	20%	13%	47%	19%	11%	26%	8%
% ≥ 15 µg/dl	0%	22%	5%	15%	22%	7%	5%	20%	5%	5%	10%	2%

IEUBK Batch Mode Overall Observed v. Predicted Blood Lead by Geographic Area, Ages 9-84 Months: Default and 40:30:30 (continued)

	Osburn			Side Gulches			Kingston			Lower Basin			Total		
	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)
N	62	62	62	30	30	30	36	36	36	28	28	28	311	311	311
Minimum (µg/dl)	1	3	2	1	2	2	1	2	2	1	2	2	1	2	2
Maximum (µg/dl)	11	14	8	9	14	8	16	9	5	18	28	16	23	28	17
Arithmetic Mean (µg/dl)	4.6	6.2	4.2	4.0	6.1	4.2	5.7	5.0	3.4	6.9	7.0	4.5	5.7	7.6	5.0
Geometric Mean (µg/dl)	4.0	5.7	4.0	3.6	5.7	4.0	4.3	4.7	3.3	4.9	4.5	3.2	4.6	6.6	4.5
Geometric Standard Deviation	1.77	1.86	1.74	1.64	1.79	1.73	2.17	1.83	1.72	2.40	2.68	2.81	1.93	2.02	1.90
% ≥ 10 µg/dl	5%	19%	5%	0%	17%	5%	17%	11%	2%	32%	21%	14%	13%	28%	11%
% ≥ 15 µg/dl	0%	6%	1%	0%	5%	1%	17%	2%	0%	11%	16%	7%	7%	12%	3%

Note: observed levels are for children 9-84 months or 0-7 years old as opposed to community mode showing observed levels for 0-9 year olds.

Table 6-50b IEUBK Batch Mode Observed v. Predicted Blood Lead by Geographic Area, Age 9-60 Months: Default and 40:30:30

	Mullan			Burke/Nine Mile			Wallace			Silverton		
	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)
N	8	8	8	31	31	31	26	26	26	46	46	46
Minimum (µg/dl)	2	8	5	2	4	4	2	7	5	2	3	2
Maximum (µg/dl)	12	13	8	21	27	17	19	21	13	23	19	11
Arithmetic Mean (µg/dl)	6.8	10.9	6.9	7.9	11.1	7.2	7.0	10.9	7.2	6.0	7.8	5.0
Geometric Mean (µg/dl)	5.8	10.7	6.8	6.3	10.0	6.7	6.0	10.6	7.1	5.0	7.2	4.8
Geometric Standard Deviation	1.87	1.64	1.65	1.98	0.98	1.77	1.79	1.79	1.66	1.78	1.81	1.75
% ≥ 10 µg/dl	25%	56%	22%	23%	48%	24%	19%	54%	25%	13%	29%	10%
% ≥ 15 µg/dl	0%	25%	6%	16%	26%	9%	8%	25%	7%	7%	12%	2%

IEUBK Batch Mode Observed v. Predicted Blood Lead by Geographic Area, Age 9-60 Months: Default and 40:30:30 (continued)

	Osburn			Side Gulches			Kingston			Lower Basin			Total		
	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)
N	43	43	43	23	23	23	27	27	27	18	18	18	222	222	222
Minimum (µg/dl)	1	3	3	1	3	2	1	3	2	1	2	2	1	2	2
Maximum (µg/dl)	11	14	8	9	14	8	16	9	5	18	28	16	23	28	17
Arithmetic Mean (µg/dl)	5.0	6.5	4.4	4.2	6.6	4.5	6.7	5.6	3.8	8.2	9.3	5.7	6.3	8.2	5.4
Geometric Mean (µg/dl)	4.3	6.1	4.3	3.8	6.3	4.4	5.4	5.3	3.7	6.2	5.7	4.1	5.1	7.2	4.9
Geometric Standard Deviation	1.80	1.83	1.71	1.61	1.75	1.69	2.03	1.79	1.68	2.31	3.27	3.11	1.90	1.96	1.86
% ≥ 10 µg/dl	7%	20%	6%	0%	20%	6%	22%	14%	3%	39%	32%	21%	16%	31%	13%
% ≥ 15 µg/dl	0%	7%	1%	0%	6%	1%	15%	3%	0%	17%	25%	11%	8%	14%	4%

Note: observed levels are for children 9-60 months or 0-5 years old.

Table 6-50c IEUBK Batch Mode Observed v. Predicted Blood Lead by Geographic Area, Age 9-24 Months: Default and 40:30:30

	Mullan			Burke/Nine Mile			Wallace			Silverton		
	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)
N	4	4	4	10	10	10	8	8	8	16	16	16
Minimum (µg/dl)	5	10	7	2	8	6	3	8	6	3	4	3
Maximum (µg/dl)	11	13	8	20	27	17	16	15	9	23	17	10
Arithmetic Mean (µg/dl)	7.8	12.1	7.8	9.1	15.0	9.6	8.1	11.4	7.7	7.5	8.5	5.5
Geometric Mean (µg/dl)	7.4	12.1	7.8	6.6	13.7	9.0	7.0	11.2	7.6	6.1	7.9	5.3
Geometric Standard Deviation	1.44	1.61	1.61	2.39	2.00	1.70	1.83	1.64	1.64	1.86	1.85	1.74
% ≥ 10 µg/dl	25%	65%	30%	30%	67%	42%	38%	59%	29%	19%	35%	13%
% ≥ 15 µg/dl	0%	33%	8%	30%	44%	20%	13%	28%	8%	19%	15%	3%

IEUBK Batch Mode Observed v. Predicted Blood Lead by Geographic Area, Age 9-24 Months: Default and 40:30:30 (continued)

	Osburn			Side Gulches			Kingston			Lower Basin			Total		
	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)	Observed	Predicted (Default)	Predicted (40:30:30)
N	14	14	14	9	9	9	7	7	7	7	7	7	75	75	75
Minimum (µg/dl)	2	3	3	2	5	4	2	3	2	3	2	2	2	2	2
Maximum (µg/dl)	11	12	8	9	14	8	15	9	5	18	25	15	23	27	17
Arithmetic Mean (µg/dl)	6.5	6.2	4.5	5.2	7.4	5.0	7.7	6.5	4.3	8.7	8.9	5.6	7.5	9.2	6.1
Geometric Mean (µg/dl)	6.0	5.7	4.3	4.8	7.0	4.9	6.3	6.1	4.2	7.1	5.5	4.0	6.2	7.9	5.5
Geometric Standard Deviation	1.55	1.87	1.73	1.55	1.72	1.69	2.03	1.82	1.67	2.04	2.88	3.07	1.82	2.08	1.91
% ≥ 10 µg/dl	14%	18%	6%	0%	26%	9%	29%	20%	4%	43%	29%	21%	23%	37%	17%
% ≥ 15 µg/dl	0%	6%	1%	0%	9%	2%	14%	5%	1%	14%	23%	12%	12%	19%	6%

Note: observed levels are for children 9-24 months or 0-2 years old.

Table 6-51a Upland Parks Estimated Recreational Blood Lead Increments for 0-84 Month Old Children (CT)

Area	Baseline Intake (µg/day)	50% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood	IEUBK Box Model Baseline Geometric Mean Blood	EPA Default		Box Model	
							Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)	Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)
Mullan	91	7	98	7%	9.3	6.3	9.8	45%	6.6	18%
Wallace	99	5	103	5%	9.9	6.8	10.3	50%	7.0	21%
Silverton	55	5	60	8%	6.3	4.3	6.7	19%	4.6	5%
Osburn	55	5	59	8%	6.2	4.4	6.6	18%	4.7	5%
Side Gulches	64	5	68	7%	7.0	4.7	7.4	25%	5.0	6%
Kingston	46	19	65	42%	5.4	3.7	7.0	21%	4.8	5%
Lower Basin	30	19	49	63%	3.9	2.8	5.6	10%	3.9	2%

Table 6-51b Upland Parks Estimated Recreational Blood Lead Increments for 0-84 Month Old Children (RME)

Area	Baseline Intake (µg/day)	95% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood	IEUBK Box Model Baseline Geometric Mean Blood	EPA Defalt		Box Model	
							Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)	Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)
Mullan	91	18	109	20%	9.3	6.3	10.6	53%	7.1	22%
Wallace	99	68	166	69%	9.9	6.8	14.2	76%	9.8	45%
Silverton	55	68	123	123%	6.3	4.3	11.3	58%	7.7	27%
Osburn	55	68	122	124%	6.2	4.4	11.3	58%	7.8	29%
Side Gulches	64	68	131	106%	7.0	4.7	11.9	61%	8.1	30%
Kingston	46	19	65	42%	5.4	3.7	7.0	21%	4.8	5%
Lower Basin	30	27	58	91%	3.9	2.8	6.2	15%	4.3	3%

Table 6-52a Neighborhood Stream Recreational Blood Lead Increment for 0-84 Month Old children (CT)

	Baseline Intake (µg/day)	50% Sediment Intake Value (µg/day)	50% Surface Water Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood	IEUBK Box Model Baseline Geometric Mean Blood	EPA Default		Box Model	
								Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)	Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)
Mullan	91	2	0	93	3%	9.3	6.3	9.5	43%	6.4	15%
Burke/Nine Mile	88	13	0	101	15%	9.0	6.2	10.0	48%	6.8	20%
Wallace	99	8	0	106	8%	9.9	6.8	10.5	50%	7.1	22%
Silverton	55	8	0	63	14%	6.3	4.3	6.9	21%	4.7	5%
Osburn	55	8	0	62	14%	6.2	4.4	6.9	20%	4.8	6%
Side Gulches	64	8	0	71	12%	7.0	4.7	7.7	27%	5.1	7%
Kingston	46	1	0	46	1%	5.4	3.7	5.5	9%	3.8	2%
Lower Basin	30	1	0	31	2%	3.9	2.8	3.9	2%	2.8	0%

Table 6-52b Neighborhood Stream Recreational Blood Lead Increment for 0-84 Month Old Children (RME)

	Baseline Intake (µg/day)	95% Sediment Intake Value (µg/day)	95% Surface Water Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood	IEUBK Box Model Baseline Geometric Mean Blood	EPA Default		Box Model	
								Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)	Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)
Mullan	91	45	0	137	50%	9.3	6.3	12.4	64%	8.4	34%
Burke/Nine Mile	88	221	1	303	271%	9.0	6.2	21.2	93%	15.2	78%
Wallace	99	19	0	118	20%	9.9	6.8	11.2	56%	7.7	27%
Silverton	55	19	0	75	35%	6.3	4.3	7.8	29%	5.3	8%
Osburn	55	19	0	74	36%	6.2	4.4	7.8	29%	5.4	9%
Side Gulches	64	19	0	83	31%	7.0	4.7	8.5	34%	5.7	11%
Kingston	46	1	0	47	2%	5.4	3.7	5.5	9%	3.8	2%
Lower Basin	30	1	0	31	3%	3.9	2.8	3.9	2%	2.8	0%

Table 6-53a Public Beach Recreational Blood Lead Increment for Children 0-84 Month Old (CT)

	Baseline Intake (µg/day)	50% Sediment Intake Value (µg/day)	50% Surface Water Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood Lead (mg/dl)	IEUBK Box Model Baseline Geometric Mean Blood Lead (mg/dl)	EPA Default		Box Model	
								Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)	Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)
Mullan	91	19	19	130	43	9.3	6.3	12.7	67	9.6	43
Burke/Nine Mile	88	19	19	120	47	9.0	6.2	12.5	64	9.4	43
Wallace	99	19	19	137	39	9.9	6.8	13.2	70	10	48
Silverton	55	19	19	94	70	6.3	4.3	10.1	48	7.9	29
Osburn	55	19	19	93	71	6.2	4.4	10.1	48	8	30
Side Gulches	64	19	19	102	61	7.0	4.7	10.7	53	8.2	32
Kingston	46	19	19	85	84	5.4	3.7	9.4	43	7.4	25
Lower Basin	30	19	19	69	128	3.9	2.8	8.1	30	6.6	18

Table 6-53b Public Beach Recreational Blood Lead Increment for Children 0-84 Month Old (RME)

	Baseline Intake (µg/day)	95% Sediment Intake Value (µg/day)	95% Surface Water Intake Value	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood Lead (mg/dl)	IEUBK Box Model Baseline Geometric Mean Blood Lead (mg/dl)	EPA Default		Box Model	
								Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)	Geometric Mean (mg/dl)	% to Exceed 10 (mg/dl)
Mullan	91	30	42	163	79	9.3	6.3	15.6	81	12.6	67
Burke/Nine Mile	88	30	42	154	88	9.0	6.2	13.4	70	12.5	64
Wallace	99	30	42	171	73	9.9	6.8	16	81	13	67
Silverton	55	30	42	127	130	6.3	4.3	13.3	70	11.2	56
Osburn	55	30	42	127	132	6.2	4.4	13.3	70	11.2	56
Side Gulches	64	30	42	136	113	7.0	4.7	13.9	73	11.5	58
Kingston	46	30	42	118	157	5.4	3.7	12.7	67	10.8	53
Lower Basin	30	30	42	102	238	3.9	2.8	11.6	58	10.1	48

Table 6-54a Waste Pile Recreational Blood Lead Increment for 0-84 Month Old Children (CT)

	Baseline Intake (µg/day)	50% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood Lead (mg/dl)	IEUBK Box Model Baseline Geometric Mean Blood Lead (mg/dl)	EPA Default		Box Model	
							Geometric Mean (µg/dl)	% to Exceed 10 µg/dl	Geometric Mean (µg/dl)	% to Exceed 10 µg/dl
Mullan	91	1	92	1%	9.3	6.3	9.4	43	6.3	15
Burke/Nine Mile	88	11	99	13%	9.0	6.2	9.8	45	6.7	19

Table 6-54b Waste Pile Recreational Blood Lead Increment for 0-84 Month Old Children (RME)

	Baseline Intake (µg/day)	95% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood Lead (mg/dl)	IEUBK Box Model Baseline Geometric Mean Blood Lead (mg/dl)	EPA Default		Box Model	
							Geometric Mean (µg/dl)	% to Exceed 10 µg/dl	Geometric Mean (µg/dl)	% to Exceed 10 µg/dl
Mullan	91	13	104	14%	9.3	6.3	10.2	50	6.9	20
Burke/Nine Mile	88	139	221	170%	9.0	6.2	17.4	86	12.2	64

Table 6-55a Home Grown Vegetable Central Tendency (CT) Intakes for Children (0-84 mos.)

Area	Baseline Intake (µg/day)	50% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood	IEUBK Box Model Baseline Geometric Mean Blood	EPA Default		Box Model	
							Geometric Mean (µg/dl)	% to Exceed 10 µg/dl	Geometric Mean (µg/dl)	% to Exceed 10 µg/dl
Mullan	91	24	115	26%	9.3	6.3	12.0	61%	9.3	43%
Burke/Nine Mile	88	24	106	29%	9.0	6.2	11.8	61%	9.3	40%
Wallace	99	24	122	24%	9.9	6.8	12.6	67%	9.8	45%
Silverton	55	24	79	43%	6.3	4.3	9.4	43%	7.7	27%
Osburn	55	24	78	44%	6.2	4.4	9.3	43%	7.8	29%
Side Gulches	64	24	88	37%	7.0	4.7	10.0	48%	8.0	30%
Kingston	46	24	70	52%	5.4	3.7	8.6	36%	7.2	22%
Lower Basin	30	24	54	79%	3.9	2.8	7.3	24%	6.4	15%

Table 6-55b Home Grown Vegetable Reasonable Maximum Exposure (RME) Intakes for Children (0-84 mos.)

Area	Baseline Intake (µg/day)	95% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood	IEUBK Box Model Baseline Geometric Mean Blood	EPA Default		Box Model	
							Geometric Mean (µg/dl)	% to Exceed 10 µg/dl	Geometric Mean (µg/dl)	% to Exceed 10 µg/dl
Mullan	91	178	269	195%	9.3	6.3	24.4	96%	22.9	94%
Burke/Nine Mile	88	178	260	218%	9.0	6.2	24.3	96%	22.8	94%
Wallace	99	178	277	180%	9.9	6.8	24.7	96%	23.1	94%
Silverton	55	178	233	322%	6.3	4.3	22.9	94%	21.9	94%
Osburn	55	178	233	325%	6.2	4.4	22.8	94%	22.0	94%
Side Gulches	64	178	242	279%	7.0	4.7	23.2	94%	22.1	94%
Kingston	46	178	224	388%	5.4	3.7	22.5	94%	21.7	93%
Lower Basin	30	178	208	589%	3.9	2.8	21.7	94%	21.2	93%

Table 6-56a Recreational Fish Ingestion Central Tendency (CT) Intakes for Children (0-84 mos.)

Area	Baseline Intake (µg/day)	50% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood	IEUBK Box Model Baseline Geometric Mean Blood	EPA Default		Box Model	
							Geometric Mean (µg/dl)	% to Exceed 10 µg/dl	Geometric Mean (µg/dl)	% to Exceed 10 µg/dl
Mullan	91	1	92	1%	9.3	6.3	9.4	43%	6.4	16%
Burke/Nine Mile	88	1	89	1%	9.0	6.2	9.2	40%	6.3	15%
Wallace	99	1	100	1%	9.9	6.8	10.0	48%	6.9	20%
Silverton	55	1	56	2%	6.3	4.3	6.4	16%	4.5	4%
Osburn	55	1	56	2%	6.2	4.4	6.4	15%	4.5	4%
Side Gulches	64	1	65	2%	7.0	4.7	7.2	27%	4.9	6%
Kingston	46	1	47	2%	5.4	3.7	5.6	10%	3.9	2%
Lower Basin	30	1	31	3%	3.9	2.8	4.0	2%	2.9	0%

Table 6-56b Recreational Fish Ingestion Reasonable Maximum Exposure (RME) Intakes for Children (0-84 mos.)

Area	Baseline Intake (µg/day)	95% Intake Value (µg/day)	Total Intake (µg/day)	% Intake Increase from Baseline	IEUBK EPA Default Baseline Geometric Mean Blood	IEUBK Box Model Baseline Geometric Mean Blood	EPA Default		Box Model	
							Geometric Mean (µg/dl)	% to Exceed 10 µg/dl	Geometric Mean (µg/dl)	% to Exceed 10 µg/dl
Mullan	91	4	95	4%	9.3	6.3	9.8	45%	6.8	20%
Burke/Nine Mile	88	4	92	5%	9.0	6.2	9.5	43%	6.7	19%
Wallace	99	4	103	4%	9.9	6.8	10.4	50%	7.3	24%
Silverton	55	4	59	7%	6.3	4.3	6.8	20%	4.9	6%
Osburn	55	4	59	7%	6.2	4.4	6.8	19%	5.0	6%
Side Gulches	64	4	68	6%	7.0	4.7	7.6	25%	5.3	8%
Kingston	46	4	50	9%	5.4	3.7	6.0	13%	4.4	4%
Lower Basin	30	4	34	13%	3.9	2.8	4.5	4%	3.4	1%

Table 6-57 Calculations of Preliminary Remediation Goals (PRGs) using the Occupational CT Values

U.S. EPA Technical Review Workgroup for Lead, Adult Lead Committee

Exposure Variable	PRG Equation[1]	Description of Exposure Variable	Units	Values for Nonresidential Exposure Scenario Using Equation 1	
	1*			GSDi = 1.8	GSDi = 2.1
$PbB_{fetal, 0.95}$	X	95th percentile PbB in fetus	ug/dL	10	10
$R_{fetal/maternal}$	X	Fetal/maternal PbB ratio	--	0.9	0.9
BKSF	X	Biokinetic Slope Factor	ug/dL per ug/day	0.4	0.4
GSD_i	X	Geometric standard deviation PbB	--	1.8	2.1
PbB_0	X	Baseline PbB	ug/dL	1.7	1.7
IR_S	X	Soil ingestion rate (including soil-derived indoor dust)	g/day	0.100	0.100
IR_{S+D}		Total ingestion rate of outdoor soil and indoor dust	g/day	--	--
W_S		Weighting factor; fraction of IR_{S+D} ingested as outdoor soil	--	--	--
K_{SD}		Mass fraction of soil in dust	--	--	--
$AF_{S, D}$	X	Absorption fraction (same for soil and dust)	--	0.12	0.12
$EF_{S, D}$[2]	X	Exposure frequency (same for soil and dust)	days/yr	43	43
PRG		**Preliminary Remediation Goal**	**ppm**	**4,465**	**2,792**

[1] Equation 1 does not apportion exposure between soil and dust ingestion (excludes W_S, K_{SD}).
When $IR_S = IR_{S+D}$ and $W_S = 1.0$, the equations yield the same PRG.

[2] The averaging time (AT) is a fixed value of 365 days/yr.

***Equation 1, based on Eq. 4 in USEPA (1996).**

$$PRG = \frac{([PbB_{95}fetal/(R*(GSD_i^{1.645})])-PbB_0)*AT}{BKSF*(IR_S*AF_S*EF_S)}$$

Source: U.S. EPA (1996). Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil

Table 6-58 Calculations of Preliminary Remediation Goals (PRGs) using the Occupational RME Values

U.S. EPA Technical Review Workgroup for Lead, Adult Lead Committee

Exposure Variable	PRG Equation[1]	Description of Exposure Variable	Units	Values for Nonresidential Exposure Scenario Using Equation 1	
	1*			GSDi = 1.8	GSDi = 2.1
$PbB_{fetal, 0.95}$	X	95th percentile PbB in fetus	ug/dL	10	10
$R_{fetal/maternal}$	X	Fetal/maternal PbB ratio	--	0.9	0.9
BKSF	X	Biokinetic Slope Factor	ug/dL per ug/day	0.4	0.4
GSD_i	X	Geometric standard deviation PbB	--	1.8	2.1
PbB_0	X	Baseline PbB	ug/dL	1.7	1.7
IR_S	X	Soil ingestion rate (including soil-derived indoor dust)	g/day	0.200	0.200
IR_{S+D}		Total ingestion rate of outdoor soil and indoor dust	g/day	--	--
W_S		Weighting factor; fraction of IR_{S+D} ingested as outdoor soil	--	--	--
K_{SD}		Mass fraction of soil in dust	--	--	--
$AF_{S, D}$	X	Absorption fraction (same for soil and dust)	--	0.12	0.12
$EF_{S, D}$[2]	X	Exposure frequency (same for soil and dust)	days/yr	195	195
PRG		**Preliminary Remediation Goal**	**ppm**	**492**	**308**

[1] Equation 1 does not apportion exposure between soil and dust ingestion (excludes W_S, K_{SD}).
When $IR_S = IR_{S+D}$ and $W_S = 1.0$, the equations yield the same PRG.

[2] The averaging time (AT) is a fixed value of 365 days/yr.

***Equation 1, based on Eq. 4 in USEPA (1996).**

$$\textbf{PRG} = \frac{([PbB_{95}fetal/(R*(GSD_i^{1.645})])-PbB_0)*AT}{BKSF*(IR_S*AF_S*EF_S)}$$

Source: U.S. EPA (1996). Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil

Table 6-59a Calculations of Preliminary Remediation Goals (PRGs) using the Upland Parks CT Values

U.S. EPA Technical Review Workgroup for Lead, Adult Lead Committee

Exposure Variable	PRG Equation[1]	Description of Exposure Variable	Units	Values for Nonresidential Exposure Scenario Using Equation 1	
	1*			GSDi = 1.8	GSDi = 2.2
$PbB_{fetal, 0.95}$	X	95th percentile PbB in fetus	ug/dL	10	10
$R_{fetal/maternal}$	X	Fetal/maternal PbB ratio	--	0.9	0.9
BKSF	X	Biokinetic Slope Factor	ug/dL per ug/day	0.4	0.4
GSD_i	X	Geometric standard deviation PbB	--	1.8	2.1
PbB_0	X	Baseline PbB	ug/dL	1.7	1.7
IR_S	X	Soil ingestion rate (including soil-derived indoor dust)	g/day	0.050	0.050
IR_{S+D}		Total ingestion rate of outdoor soil and indoor dust	g/day	--	--
W_S		Weighting factor; fraction of IR_{S+D} ingested as outdoor soil	--	--	--
K_{SD}		Mass fraction of soil in dust	--	--	--
$AF_{S, D}$	X	Absorption fraction (same for soil and dust)	--	0.12	0.12
$EF_{S, D}$[2]	X	Exposure frequency (same for soil and dust)	days/yr	15	15
PRG		**Preliminary Remediation Goal**	**ppm**	**25,601**	**16,007**

[1] Equation 1 does not apportion exposure between soil and dust ingestion (excludes W_S, K_{SD}).
When $IR_S = IR_{S+D}$ and $W_S = 1.0$, the equations yield the same PRG.

[2] The averaging time (AT) is a fixed value of 365 days/yr.

***Equation 1, based on Eq. 4 in USEPA (1996).**

$$\textbf{PRG} = \frac{([PbB_{95}fetal/(R*(GSD_i^{1.645})])-PbB_0)*AT}{BKSF*(IR_S*AF_S*EF_S)}$$

Source: U.S. EPA (1996). Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil

Table 6-59b Calculations of Preliminary Remediation Goals (PRGs) using the CUAs CT Values

U.S. EPA Technical Review Workgroup for Lead, Adult Lead Committee

Exposure Variable	PRG Equation[1]	Description of Exposure Variable	Units	Values for Nonresidential Exposure Scenario Using Equation 1	
	1*			GSDi = 1.8	GSDi = 2.1
$PbB_{fetal, 0.95}$	X	95th percentile PbB in fetus	ug/dL	10	10
$R_{fetal/maternal}$	X	Fetal/maternal PbB ratio	--	0.9	0.9
BKSF	X	Biokinetic Slope Factor	ug/dL per ug/day	0.4	0.4
GSD_i	X	Geometric standard deviation PbB	--	1.8	2.1
PbB_0	X	Baseline PbB	ug/dL	1.7	1.7
IR_S	X	Soil ingestion rate (including soil-derived indoor dust)	g/day	0.050	0.050
IR_{S+D}		Total ingestion rate of outdoor soil and indoor dust	g/day	--	--
W_S		Weighting factor; fraction of IR_{S+D} ingested as outdoor soil	--	--	--
K_{SD}		Mass fraction of soil in dust	--	--	--
$AF_{S, D}$	X	Absorption fraction (same for soil and dust)	--	0.12	0.12
$EF_{S, D}$[2]	X	Exposure frequency (same for soil and dust)	days/yr	16	16
PRG		**Preliminary Remediation Goal**	**ppm**	**24,001**	**15,006**

[1] Equation 1 does not apportion exposure between soil and dust ingestion (excludes W_S, K_{SD}).
When $IR_S = IR_{S+D}$ and $W_S = 1.0$, the equations yield the same PRG.

[2] The averaging time (AT) is a fixed value of 365 days/yr.

***Equation 1, based on Eq. 4 in USEPA (1996).**

$$\textbf{PRG} = \frac{([PbB_{95}fetal/(R*(GSD_i^{1.645})])-PbB_0)*AT}{BKSF*(IR_S*AF_S*EF_S)}$$

Source: U.S. EPA (1996). Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil

Table 6-60a Calculations of Preliminary Remediation Goals (PRGs) using the Upland Parks RME Values

U.S. EPA Technical Review Workgroup for Lead, Adult Lead Committee

Exposure Variable	PRG Equation[1]	Description of Exposure Variable	Units	Values for Nonresidential Exposure Scenario	
				Using Equation 1	
	1*			GSDi = 1.8	GSDi = 2.2
$PbB_{fetal, 0.95}$	X	95th percentile PbB in fetus	ug/dL	10	10
$R_{fetal/maternal}$	X	Fetal/maternal PbB ratio	--	0.9	0.9
BKSF	X	Biokinetic Slope Factor	ug/dL per ug/day	0.4	0.4
GSD_i	X	Geometric standard deviation PbB	--	1.8	2.1
PbB_0	X	Baseline PbB	ug/dL	1.7	1.7
IR_S	X	Soil ingestion rate (including soil-derived indoor dust)	g/day	0.100	0.100
IR_{S+D}		Total ingestion rate of outdoor soil and indoor dust	g/day	--	--
W_S		Weighting factor; fraction of IR_{S+D} ingested as outdoor soil	--	--	--
K_{SD}		Mass fraction of soil in dust	--	--	--
$AF_{S, D}$	X	Absorption fraction (same for soil and dust)	--	0.12	0.12
$EF_{S, D}$[2]	X	Exposure frequency (same for soil and dust)	days/yr	30	30
PRG		**Preliminary Remediation Goal**	**ppm**	**6,400**	**4,002**

[1] Equation 1 does not apportion exposure between soil and dust ingestion (excludes W_S, K_{SD}).
When $IR_S = IR_{S+D}$ and $W_S = 1.0$, the equations yield the same PRG.

[2] The averaging time (AT) is a fixed value of 365 days/yr.

***Equation 1, based on Eq. 4 in USEPA (1996).**

$$\textbf{PRG} = \frac{([PbB_{95}fetal/(R*(GSD_i^{1.645})])-PbB_0)*AT}{BKSF*(IR_S*AF_S*EF_S)}$$

Source: U.S. EPA (1996). Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil

Table 6-60b Calculations of Preliminary Remediation Goals (PRGs) using the CUA RME Values

U.S. EPA Technical Review Workgroup for Lead, Adult Lead Committee

Exposure Variable	PRG Equation[1]	Description of Exposure Variable	Units	Values for Nonresidential Exposure Scenario	
				Using Equation 1	
	1*			GSDi = 1.8	GSDi = 2.2
$PbB_{fetal, 0.95}$	X	95th percentile PbB in fetus	ug/dL	10	10
$R_{fetal/maternal}$	X	Fetal/maternal PbB ratio	--	0.9	0.9
BKSF	X	Biokinetic Slope Factor	ug/dL per ug/day	0.4	0.4
GSD_i	X	Geometric standard deviation PbB	--	1.8	2.1
PbB_0	X	Baseline PbB	ug/dL	1.7	1.7
IR_S	X	Soil ingestion rate (including soil-derived indoor dust)	g/day	0.100	0.100
IR_{S+D}		Total ingestion rate of outdoor soil and indoor dust	g/day	--	--
W_S		Weighting factor; fraction of IR_{S+D} ingested as outdoor soil	--	--	--
K_{SD}		Mass fraction of soil in dust	--	--	--
$AF_{S, D}$	X	Absorption fraction (same for soil and dust)	--	0.12	0.12
$EF_{S, D}$[2]	X	Exposure frequency (same for soil and dust)	days/yr	32	32
PRG		**Preliminary Remediation Goal**	**ppm**	**6,000**	**3,752**

[1] Equation 1 does not apportion exposure between soil and dust ingestion (excludes W_S, K_{SD}).
When $IR_S = IR_{S+D}$ and $W_S = 1.0$, the equations yield the same PRG.

[2] The averaging time (AT) is a fixed value of 365 days/yr.

***Equation 1, based on Eq. 4 in USEPA (1996).**

$$PRG = \frac{([PbB_{95}fetal/(R*(GSD_i^{1.645})])-PbB_0)*AT}{BKSF*(IR_S*AF_S*EF_S)}$$

Source: U.S. EPA (1996). Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil

Table 6-61a Estimated Post-Remedial Blood Lead Levels and Probability to Exceed Health Criteria by Community and Individuals for a 2000 mg/kg Soil Action Level - EPA Default Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide		Homes 1500-2000 mg/kg			
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl	Number of Homes	% of Homes	Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	7.1	105	12	11%	30%	12%	12	11%	67%	36%
Burke/Nine Mile	7.2	88	14	16%	32%	13%	11	13%	67%	37%
Wallace	8.3	110	13	12%	39%	17%	11	10%	70%	40%
Silverton	6.1	70	1	1%	20%	6%	2	3%	65%	35%
Osburn	6.1	262	13	5%	20%	6%	4	2%	64%	34%
Side Gulches	6.2	100	1	1%	21%	7%	1	1%	66%	35%
Kingston	4.9	99	7	7%	12%	3%	1	1%	68%	38%
Lower Basin	3.7	160	11	7%	6%	2%	3	2%	59%	29%

Table 6-61b Estimated Post-Remedial Blood Lead Level and Probability to Exceed Health Criteria by Community and Individuals for a 1500 mg/kg Soil Action Level - EPA Default Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide		Homes 1000-1500 mg/kg			
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl	Number of Homes	% of Homes	Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	6.2	105	24	23%	22%	7%	11	10%	55%	25%
Burke/Nine Mile	6.1	88	25	28%	23%	8%	13	15%	54%	25%
Wallace	7.3	110	24	22%	31%	12%	18	16%	58%	28%
Silverton	5.9	70	3	4%	18%	5%	4	6%	50%	22%
Osburn	6.0	262	17	6%	19%	6%	13	5%	51%	22%
Side Gulches	6.1	100	2	2%	20%	6%	6	6%	51%	23%
Kingston	4.8	99	8	8%	11%	3%	5	5%	53%	24%
Lower Basin	3.6	160	14	9%	5%	1%	5	3%	48%	20%

Table 6-61c Estimated Post-Remedial Blood Lead Level and Probability to Exceed Health Criteria by Community and Individuals for a 1000 mg/kg Soil Action Level - EPA Default Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide		Homes 800-1000 mg/kg			
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl	Number of Homes	% of Homes	Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	5.5	105	35	33%	16%	4%	7	10%	42%	16%
Burke/Nine Mile	5.3	88	38	43%	15%	4%	7	15%	41%	15%
Wallace	6.1	110	42	38%	21%	7%	18	16%	46%	19%
Silverton	5.6	70	7	10%	15%	4%	2	6%	41%	16%
Osburn	5.7	262	30	11%	17%	4%	14	5%	39%	14%
Side Gulches	5.7	100	8	8%	17%	5%	5	6%	39%	14%
Kingston	4.6	99	13	13%	8%	2%	1	5%	36%	13%
Lower Basin	3.5	160	19	12%	3%	1%	1	3%	38%	14%

Table 6-61d Estimated Post-Remedial Blood Lead Level and Probability to Exceed Health Criteria by Community and Individuals for a 800 mg/kg Soil Action Level - EPA Default Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide		Homes 600-800 mg/kg			
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl	Number of Homes	% of Homes	Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	5.2	105	42	40%	13%	3%	13	10%	34%	12%
Burke/Nine Mile	4.9	88	45	51%	12%	3%	15	15%	33%	11%
Wallace	5.3	110	60	55%	14%	4%	14	16%	36%	12%
Silverton	5.4	70	9	13%	14%	3%	9	6%	32%	11%
Osburn	5.4	262	44	17%	14%	4%	34	5%	31%	10%
Side Gulches	5.4	100	13	13%	15%	4%	16	6%	30%	10%
Kingston	4.5	99	14	14%	8%	2%	3	5%	32%	10%
Lower Basin	3.5	160	20	13%	3%	0%	1	3%	31%	10%

Table 6-61e Estimated Post-Remedial Blood Lead Levels and Probability to Exceed Health Criteria by Community and Individuals for a 600 mg/kg Soil Action Level - EPA Default Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide		Homes 400-600 mg/kg			
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl	Number of Homes	% of Homes	Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	4.7	105	55	52%	9%	2%	14	10%	22%	6%
Burke/Nine Mile	4.3	88	60	68%	6%	1%	8	15%	24%	7%
Wallace	4.8	110	74	67%	10%	2%	17	16%	26%	8%
Silverton	4.9	70	18	26%	10%	2%	16	6%	21%	5%
Osburn	4.9	262	78	30%	10%	2%	60	5%	21%	6%
Side Gulches	4.8	100	29	29%	10%	2%	29	6%	20%	5%
Kingston	4.4	99	17	17%	7%	1%	7	5%	20%	5%
Lower Basin	3.4	160	21	13%	3%	0%	6	3%	17%	4%

Table 6-61f Estimated Post-Remedial Blood Lead Levels and Probability to Exceed Health Criteria by Community and Individuals for a 400 mg/kg Soil Action Level - EPA Default Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide	
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	4.4	105	69	66%	6%	1%
Burke/Nine Mile	4.1	88	68	77%	5%	1%
Wallace	4.4	110	91	83%	6%	1%
Silverton	4.3	70	34	49%	6%	1%
Osburn	4.3	262	138	53%	6%	1%
Side Gulches	4.0	100	58	58%	4%	1%
Kingston	4.2	99	24	24%	6%	1%
Lower Basin	3.4	160	27	17%	2%	0%

Table 6-62a Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 2000 mg/kg Soil Action Level - Box Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide		Homes 1500-2000 mg/kg			
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl	Number of Homes	% of Homes	Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	4.7	105	12	11%	9%	2%	12	11%	26%	8%
Burke/Nine Mile	4.7	88	14	16%	10%	2%	11	13%	27%	8%
Wallace	5.4	110	13	12%	14%	3%	11	10%	31%	10%
Silverton	4.0	70	1	1%	5%	1%	2	3%	24%	7%
Osburn	4.1	262	13	5%	5%	1%	4	2%	24%	6%
Side Gulches	4.1	100	1	1%	5%	1%	1	1%	25%	7%
Kingston	3.2	99	7	7%	2%	0%	1	1%	25%	7%
Lower Basin	2.4	160	11	7%	1%	0%	3	2%	16%	4%

Table 6-62b Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 1500 mg/kg Soil Action Level - Box Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide		Homes 1000-1500 mg/kg			
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl	Number of Homes	% of Homes	Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	4.0	105	24	23%	5%	1%	11	10%	17%	4%
Burke/Nine Mile	4.0	88	25	28%	6%	1%	13	15%	16%	4%
Wallace	4.7	110	24	22%	9%	2%	18	16%	20%	5%
Silverton	3.9	70	3	4%	4%	1%	4	6%	14%	3%
Osburn	4.0	262	17	6%	5%	1%	13	5%	15%	3%
Side Gulches	4.0	100	2	2%	5%	1%	6	6%	16%	4%
Kingston	3.2	99	8	8%	2%	0%	5	5%	15%	3%
Lower Basin	2.4	160	14	9%	1%	0%	5	3%	11%	2%

Table 6-62c Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 1000 mg/kg Soil Action Level - Box Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide		Homes 800-1000 mg/kg			
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl	Number of Homes	% of Homes	Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	3.6	105	35	33%	3%	0%	7	7%	10%	2%
Burke/Nine Mile	3.4	88	38	43%	3%	0%	7	8%	9%	2%
Wallace	3.9	110	42	38%	5%	1%	18	16%	12%	2%
Silverton	3.6	70	7	10%	3%	0%	2	3%	10%	2%
Osburn	3.8	262	30	11%	3%	0%	15	6%	9%	2%
Side Gulches	3.7	100	8	8%	4%	0%	5	5%	10%	2%
Kingston	3.0	99	13	13%	1%	0%	1	1%	7%	1%
Lower Basin	2.3	160	19	12%	0%	0%	1	1%	7%	1%

Table 6-62d Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 800 mg/kg Soil Action Level - Box Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide		Homes 600-800 mg/kg			
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl	Number of Homes	% of Homes	Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	3.3	105	42	40%	2%	0%	13	12%	7%	1%
Burke/Nine Mile	3.2	88	45	51%	2%	0%	15	17%	6%	1%
Wallace	3.4	110	60	55%	2%	0%	14	13%	7%	1%
Silverton	3.5	70	9	13%	3%	0%	9	13%	7%	1%
Osburn	3.6	262	45	17%	3%	0%	33	13%	7%	1%
Side Gulches	3.6	100	13	13%	3%	0%	16	16%	6%	1%
Kingston	3.0	99	14	14%	1%	0%	3	3%	6%	1%
Lower Basin	2.3	160	20	13%	0%	0%	1	1%	5%	1%

Table 6-62e Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 600 mg/kg Soil Action Level - Box Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide		Homes 400-600 mg/kg			
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl	Number of Homes	% of Homes	Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	3.1	105	55	52%	1%	0%	14	13%	4%	0%
Burke/Nine Mile	2.8	88	60	68%	1%	0%	9	10%	3%	0%
Wallace	3.1	110	74	67%	1%	0%	17	15%	4%	1%
Silverton	3.2	70	18	26%	2%	0%	16	23%	3%	0%
Osburn	3.2	262	78	30%	2%	0%	61	23%	4%	0%
Side Gulches	3.1	100	29	29%	2%	0%	29	29%	3%	0%
Kingston	2.9	99	17	17%	1%	0%	7	7%	3%	0%
Lower Basin	2.3	160	21	13%	0%	0%	6	4%	2%	0%

Table 6-62f Estimated Post-Remedial Blood Lead and Risk by Community and Individuals for a 400 mg/kg Soil Action Level - Box Model

Area	Predicted Geometric Mean Blood Lead (mg/dl)	Total Number of Homes	Number of Homes Remediated	% of Homes Remediated	Community Wide	
					Probability ≥ 10 mg/dl	Probability ≥ 15 mg/dl
Mullan	2.8	105	69	66%	1%	0%
Burke/Nine Mile	2.7	88	69	78%	0%	0%
Wallace	2.8	110	91	83%	1%	0%
Silverton	2.8	70	34	49%	1%	0%
Osburn	2.8	262	139	53%	1%	0%
Side Gulches	2.7	100	58	58%	1%	0%
Kingston	2.8	99	24	24%	1%	0%
Lower Basin	2.2	160	27	17%	0%	0%

7.0 UNCERTAINTIES IN RISK ASSESSMENT

The purpose of this baseline risk assessment is to identify areas and activities in the Coeur d'Alene River Basin with potential risks and hazards that are greater than the public health target goals established by the EPA. The findings of the risk assessment will be incorporated into the Feasibility Study (FS) in order to select the most appropriate human health remedies for the COPCs in areas where risks exceed target health goals. Estimating and evaluating health risks from exposure to environmental chemicals is a complex process with inherent uncertainties. Uncertainty reflects limitations in knowledge and simplifying assumptions that must be made in order to quantify health risks.

Uncertainty can be statistically classified into four types (Finkel 1990; Hattis and Burmaster 1994): parameter uncertainty, model uncertainty, decision-rule uncertainty, and variability. Of these, the first two often provide much of the overall uncertainty in risk assessment (in contrast to risk management), and provides much of the uncertainty for this HHRA.

Parameter uncertainty includes measurement errors and random and/or systematic errors arising from the inability to measure variables precisely and accurately (equipment and laboratory protocol problems), or because the quantity being measured varies spatially or temporally. Basic methodological (laboratory processing and equipment) errors were less a problem for the data sets included in the Basin HHRA, given the reliance on standardized CLP and other QA/QC-dictated criteria. The principal problems lie more with sampling, i.e., spatial and temporal error.

Spatial and temporal errors apply to both the lead and non-lead sections and to both environmental and exposure biomarker measurements such as blood lead. For brief illustration, the 1996 Basin ATSDR/IDHW study sampled exposure units, particularly residential soils, differently than did the later EPA field work. Geostatistical characterization of the various segments of the Basin was limited, due to the complex nature of the contaminant point sources. Consequently, point and block kriging or other geostatistical approaches to characterize distributions of contaminants are largely absent and limit the ability to quantify uncertainty in sampling.

Temporal errors are of differing type in the HHRA and include different times for environmental data gathering and blood lead sampling, along with differing sampling designs within separate blood survey efforts. Temporal errors also apply to the extent of any differences arising later, versus the present, in the absence of remedial measures: future land use changes, future exposure receptor demographic and socioeconomic characteristics, etc.

Model uncertainty can arise from use in any model of surrogate variables, from excluded variables that should have been included, abnormal conditions, incorrect model forms, etc. This is of special concern in lead risk assessment, as pathways of lead exposure have both direct effects (from contact with contaminated media) and secondary impacts such as the soil and paint contributions to house dust lead. Failure to correctly specify these variables can lead to uncertainties in interpreting quantitative results.

Abnormal conditions affecting model evaluation can be problematic when using a highly variable exposure measure, such as blood lead, to validate or calibrate exposure models using measured blood lead data. It is always important to characterize the validity of the blood lead data to assure the true reflection of steady-state lead exposures of children and other receptors, and not transitory or abrupt changes.

Decision-rule uncertainty, unlike the first two elements, is more important to the risk manager rather than the risk assessor. Examples include uncertainties within the process of evaluating competing or different priorities among societal and economic concerns when arriving at an acceptable level of measured or modeled risk.

"Variability" is often confused with "uncertainty," but the terms are different. Variability, according to currently accepted risk terminology and methodology, is taken as the underlying and relatively stable distribution of some parameter that can be empirically characterized in knowable biological, physical, bio-physicochemical, or chemical terms. Variability can be characterized empirically in an exposure population, but that does not eliminate its contribution to overall uncertainty. For example, in the case of lead and non-lead contaminants in yard soils, such factors as particle size distribution, chemical species, matrix effects, and so on can be characterized. However, this does not fully resolve uncertainty as to where to sample soil and what to sample.

Uncertainty can be assessed via a formal analysis or can be described qualitatively. The choice of qualitative or quantitative approaches depends on the completeness of the database and the purposes of the original risk analysis. In formal or quantitative analysis, the uncertainty with each parameter in the risk estimation process is first quantified. Uncertainty is described by inclusion of a standard error of means or probability density functions (relative probability for discrete parameter values). Then, numerical methods such as one- or two-dimensional Monte Carlo analyses can be used to develop a composite uncertainty distribution by merging all individual distributions. In this way, the risk or model equations undergo solution repetition using randomly sampled values from the specified distributions to calculate a distribution of risk values. For example, an exposure or risk level for lead can be selected that corresponds to the 95th percentile of the overall risk distribution rather than relying on a single point estimate of risk based on the 95th percentile measures of each parameter.

For the data sets used in this HHRA, "variability" has not been systematically assessed and some question of true representativeness of the sampled populations to the true populations remain within the point estimates. In addition, the overall data base borrows from several underlying studies conducted over a four year period. Given these limitations and the diversity of the data sets in the various Basin studies, uncertainty issues are addressed qualitatively in this HHRA.

Uncertainties reflect limitations in knowledge. In this assessment, uncertainties relate to (*i*) the development of media concentrations that people are exposed to, (*ii*) the assumptions about exposure and toxicity, and (*iii*) the characterization of health risks. Uncertainty in the development of media concentrations is due to the inability to sample every square inch of potentially impacted media at a site.

Instead, a limited number of samples must be obtained to represent the contaminant characteristics of a larger medium. The sampling strategies for non-lead contaminants in this assessment were, in general, designed to prevent underestimation of media concentrations, thus avoiding an underestimation of the risks to public health. In the risk assessment, uncertainties were handled conservatively (i.e., health protective choices were preferentially made). Media sampling for lead differed with some of the data sets and it is less likely that underestimations of media lead levels were systematically avoided.

There are uncertainties regarding the quantification of health risks in terms of a number of assumptions about both exposure and toxicity, including both site-specific and general uncertainties. Based on anticipation of uncertainty when quantifying exposure and toxicity, the health risks and hazards presented in this risk assessment are more likely to indicate that chemicals are exceeding target risk goals, although health risks may actually be negligible. Risk assessment methodology is less likely to indicate that chemicals are not a health risk when they actually are. This process is necessary to ensure the protection of public health.

Uncertainty in the risk assessment produces the potential for two kinds of errors. The first potential, or Type I, error is the identification of a specific chemical, area, or activity as a health concern when, in fact, it is not a concern (false positive conclusion). The second potential, or Type II error is the elimination of a chemical, area, or activity from further consideration when, in fact, there should be a concern (false negative conclusion). In the HHRA, uncertainties were handled conservatively (i.e., health protective choices were preferentially made). This strategy is more likely to produce false positive errors than false negative errors.

The following sections provide additional detail regarding uncertainties in the estimations of health risks.

7.1 FACTORS LEADING TO POSSIBLE OVERESTIMATION OF RISK

Assumptions in the risk assessment with the potential to overestimate risk are discussed in the following subsections. These protective assumptions compensate for uncertainties in the calculations or simplifications that might potentially underestimate risk (discussed in Section 7.2).

7.1.1 Data Collection and Evaluation

The data evaluation process addresses whether (1) chemicals are potentially present in various environmental media at levels of health concern, (2) site concentrations are different from background concentrations, and (3) sufficient samples have been collected to fully characterize each exposure pathway.

Soil and Sediment Sampling

Thousands of soil and sediment samples have been collected in the Coeur d'Alene Basin over many years and within a large area. The risk assessment for both lead and the non-lead metals used a subset

of the total data available. The HHRA data subset preferentially selected soil samples that were (1) sieved to represent the fine particles that stick to children's hands (less than 175-micron diameter), and (2) collected from places known to be used by people (primarily residential yards and areas with high public use). Where sieved data were unavailable, bulk data that met the risk assessment quality standards were used.

For non-lead metals, 191 homes were sampled and analyzed for non-lead metals. From these homes, 929 soil samples were used in the EPC calculations for residential exposure. The samples used for the residential soil data had all been sieved. All the homes from which soil samples were collected had been volunteered by the residents. It is unknown whether the volunteer aspect of the sampling resulted in an overestimation or underestimation of metal concentrations relative to residents who have not volunteered to have their properties sampled. For lead, however, nearly 1000 homes in the Basin were sampled (homes had been volunteered but a concerted effort was made to include as many homes as possible) and no significant differences could be found between the IDHW/ATSDR and EPA data sets for lead. Lead is collocated with the other mining-related metals, i.e., high lead concentrations indicate high concentrations of the other mining-related metals and vice versa (see Appendix F). As a result, the soil data for non-lead metals from the 191 homes are likely representative of the remainder of the Basin.

A total of 49 common use (or public) areas were sampled in the Basin. Common use areas were selected with local input to identify the areas with the highest public use. Of the 49 selected areas, 33 are located along the lower Coeur d'Alene River, 1 is located on Coeur d'Alene Lake (Blackwell Island), and 15 are upland parks, schools, and day care centers in the towns of Silverton and Wallace. This effort to evaluate public areas involved sampling nearly all of the commonly used areas throughout the Basin. A total of 647 soil and sediment samples were collected from public areas for use in the EPC calculations. Concentrations of metals in these areas ranged from relatively low to very high when compared to human health Screening Values (SVs). Therefore, exposure to metals during recreational activities in areas that have not been sampled is unlikely to be underestimated.

In addition to easily accessible public areas, the risk assessment also evaluated "neighborhood" exposure to soil and sediment at undeveloped areas adjacent to homes. As part of the effort to characterize neighborhood exposures, several waste piles near homes in Canyon Creek, Nine Mile Creek, and Mullan that could be accessed by children were sampled. A total of 27 samples were collected from five waste piles for use in the EPC calculations. Waste piles generally have the highest metal concentrations and including them as part of the neighborhood exposures likely resulted in overestimation rather than underestimation of these types of exposure.

Neighborhood sediment concentrations were evaluated in Canyon Creek, Nine Mile Creek, Elk Creek, Pine Creek, and the South Fork. A total of 75 sediment samples from the five water bodies were used in the EPC calculations. While there are a number of additional creeks in the upper Basin with homes adjacent to water, the other creeks are unlikely to have sediment concentrations greater than those in Canyon Creek or Nine Mile Creek, which have some of the largest mining impacts in the

area. Therefore, creeks that were not sampled and/or did not have data that were usable in the risk assessment are unlikely to have higher metals concentrations in sediments. In the absence of data, exposure to unsampled creeks should be assumed to be within the range of risks found in the risk assessment and potentially as great as the risks in Canyon Creek and Nine Mile Creek, where risks were greatest (Figures 5-1 and 5-2). However, neighborhood sediment concentrations may be underestimated because the samples were not sieved before analysis (see Section 7.2.1).

The concentrations of sediment and surface water for the five water bodies where sampling was not done for the purposes of human health should be considered. Sample locations were not selected based on human use patterns (see previous discussions in Section 2) and therefore, the risk results are of limited use for risk managers because specific exposure areas applicable to human health cannot be determined from this data. However, the risk results in Section 5 indicate that neighborhood exposures along Canyon and Nine Mile Creeks may be "risky" and therefore areas near homes may warrant further investigation. Many of the high concentration sediments in these two creeks are already slated for removal (or in some cases, removal actions have happened) because of ecological considerations.

Surface Water Sampling

Public exposures to surface water during recreational use were evaluated in the Lower Basin using “disturbed” samples, i.e., samples that were collected after the water had been stirred up. These water samples contained a large amount of suspended sediments. Because water in the lower Coeur d’Alene River typically has a lower suspended sediment load, metal concentrations in water have likely been overestimated. Concentration data from a total of 127 disturbed water samples were used in the EPC calculations.

7.1.2 Exposure Assessment

For estimating the RME, upper 90th percentile values or high-end estimates of national averages are generally used for exposure assumptions. As discussed by the EPA, the intent of the RME is to present risks as a range from CT to high-end risk (“above the 90th percentile of the population distribution”) (Habicht 1992). This descriptor is intended to “estimate the risks that are expected to occur in small but definable ‘high end’ segments of the subject population” (Habicht 1992). The EPA makes a distinction between scenarios that are possible, but highly improbable, and those that are conservative, but more likely to occur within a population, with the latter being favored in risk assessment. RME calculations thus overestimate risk for the majority of a hypothetical population even though all assumptions may not be at their maximum.

The following discussion summarizes exposure assumptions that potentially overestimate risk.

Ingestion of Soil and Sediment

The intake rates of soil and sediment included an assumption that the rates of ingestion of soil and sediment during recreational activities were 300 mg/day for young children, and 100 mg/day for older children and adults. This applied to all chemicals except lead, because different values for ingestion are used in the IEUBK Model for lead. The intake rate of 300 mg/kg day is the 90th percentile value from a study of the amount of soil ingested by children while camping (van Wijnen et al. 1990). The average value from this study was 120 mg/day. Use of the 90th percentile value likely overestimates soil ingestion for the majority of children.

The exposure rates may be exceeded by some individuals within a population. For example, a child on a given day may ingest more than 300 mg or the 200 mg/day assumed for residential exposures. A recent evaluation suggests that the 90th percentile level for the average daily soil ingestion rate may be as high as 1100 mg/day assuming the variability measured in warmer seasons can be extrapolated over a year (Calabrese and Stanek 1995). The 90th percentile of the average soil ingestion rate during the measurement period was about 180 mg/day (Calabrese and Stanek 1995). The soil ingestion rate is intended to be a daily average over the exposure period, rather than a maximum value, i.e., an actual child may ingest more than 200 or 300 mg one day but less than 200 or 300 mg on other days. Therefore, on average, over the years of the exposure periods, soil ingestion rates will be less than the 90th percentile values; therefore, ingestion will be overestimated for most children.

Averaging Time

The assumption of a 70-year averaging time used in EPA RME assumptions tends to overestimate cancer risks, which are prorated over the lifetime. The current life expectancy in the United States is actually 75.7 years (Bureau of the Census 1994). A 75.7-year averaging time is more consistent with the way the arsenic SF was developed and thus technically should be used in the risk calculations rather than the 70-year default value.

Adult Body Weight

The default value for adult body weight used in the risk calculations was 70 kg (USEPA 1991a, 1998e). The latest information presented in USEPA, 1997a, indicates that the average body weight for adults age 18 to 75 is currently closer to 71.8 kg. Therefore, use of 70 kg over-estimates risks and hazards for the adult population by about three percent. This indicates that the occupational exposures, which only include adults, are overestimated. However, in the combined adult/child risk and hazard calculations, the "adult" portion is assumed to include individuals of seven years to 30 years. Using a body weight of 70 kg for this age range may actually underestimate risks. In addition, cancer slope factors are calculated assuming 70 kg as the adult body weight and thus cancer risk calculations for arsenic correctly used 70 kg.

Skin Surface Area

The dermal surface areas used for the 4 to 11 year old age group in the neighborhood recreational scenarios were potentially overestimated for the assumed exposure period. The skin surface areas used to calculate risks and hazards assumed an exposure period of 24 weeks (May through mid-October) for exposure to floodplain soil/sediment along the river and 34 weeks (April through November) for exposure to upland parks and schools and waste pile soils. It was assumed that a child would be wearing shorts, shirt sleeves, and bare feet for the duration of the exposure period which corresponds to a skin surface area of 5080 cm^2. In addition, for floodplain soil/sediment exposure in the Lower Basin and Kingston, where swimming is a possibility, it was assumed that a child would be wearing a bathing suit for the warmest 16 weeks of the 24 weeks of exposure, corresponding to a time adjusted skin surface area of 7960 cm^2 (See Section 3.3.3).

Based on the climate and temperature patterns of the Basin, these skin surface areas may be overly protective, because it may not be reasonable to assume that a child would be barefoot, wearing shorts and shirt sleeves for all 34 weeks (April through November) for neighborhood exposure to waste piles and upland parks, nor would a child be swimming in the river for 16 weeks out of the 24 weeks per year of exposure to floodplain soil/sediments in the Lower Basin and Kingston areas. However, risks and hazards for the combined neighborhood exposures are not risk drivers and dermal exposures are a relatively low percentage of the total neighborhood risks, only 17% to 35% for arsenic. In addition, if skin surface areas are lowered to reflect more reasonable exposure assumptions, total RME risks and hazards will decrease by less than 10%. Table 7-1 summarizes the change in RME risk and hazard estimates if reduced skin surface areas are used in the calculations. These reduced skin surface areas assume that for dermal exposure to waste piles and upland parks a child will be wearing pants, shoes, and shirt sleeves for 10 weeks and shorts, shirt sleeves, and bare feet for the remaining 24 weeks of the 34 week exposure period, corresponding to a skin surface area of 2775 cm^2. For dermal exposure to floodplain soil/sediment it was assumed that a child would be wearing a swimsuit for only 8 weeks out of the 24 week exposure period (and shorts, shirt sleeves, and bare feet the other 16 weeks) in the Lower Basin and Kingston areas, and 4 weeks out of the 24 week exposure period (and shorts, shirt sleeves, and bare feet the other 20 weeks) in all other areas of the Basin, which corresponds to a skin surface area of 5175 cm^2 and 4240 cm^2, respectively. In summary, although the skin surface areas used in the calculations presented in Sections 3 and 5 of the report were likely overestimated, reducing skin surface areas to levels more appropriate for the local climate conditions results in a reduction of only 10% in the RME risks and hazards, and would not affect any of the conclusions reached.

7.1.3 Toxicity Assessment and Risk Calculations

Toxicity values have been developed by the EPA from the available toxicological data. These values frequently involve high- to low-dose extrapolations and are often derived from animal rather than human data. In addition, there may be few studies available for a particular chemical. As the unknowns increase, the uncertainty of the value increases. Uncertainty is addressed by reducing RfDs using

uncertainty factors and by deriving SFs using a conservative model. The greater the uncertainty, the greater the uncertainty factors and tendency to overestimate the toxicity.

The risk calculations combine uncertainties regarding the data evaluation, the exposure assessment, and the toxicity assessment.

Arsenic Toxicity Issues

For cancer effects, the conservative assumption is made that some finite risk is associated with exposure to even one atom of arsenic. EPA's SF for arsenic is based on a linear high- to low-dose extrapolation primarily from human studies. For some chemicals, detoxification reactions in the body may reduce the risk of cancer at low doses (Williams and Weisburger 1996). This was considered to be potentially true for arsenic because the human body changes inorganic arsenic to organic forms that were thought to be less toxic and are more readily excreted in the urine (Goyer 1996). Some inorganic arsenic is also directly excreted in the urine. The half-life of ingested arsenic in the body is about 4 days, with urine being the greatest pathway of elimination (NRC 1999).

A recent study has found that methylation of arsenic may not be a detoxification pathway. Mass et al. (2001) found that methylated trivalent arsenic added to human peripheral lymphocytes produced direct DNA damage. Genotoxicity at the DNA level was established using nicking and degradation assays to include use of the single-cell gel (SCG or "comet") assay. Dimethyltrivalent arsenic was 388 times more potent in inducing DNA damage than was inorganic arsenic in the study. The authors were careful to rule out artifactual occurrences as potential alternative explanations of their findings. They correctly noted that direct DNA damage may not be the only mechanism by which arsenic can impart carcinogenic effects. However, their findings show we cannot automatically assume that biomethylation of absorbed inorganic arsenic is solely a detoxification pathway. It may actually be a step required for carcinogenic action based on direct DNA damage.

The recent review of arsenic toxicity in drinking water by the National Research Council (NRC) found a wide variation in methylated forms of arsenic in the urine and recommended more study in this area before drawing conclusions or quantifying detoxification abilities and their effect on health endpoints (NRC 1999). The NRC (1999) also noted that more research is needed on possible differences in arsenic methylation abilities between children and adults.

Other potential sources of uncertainty regarding the arsenic SF are due to unresolved scientific issues regarding differences between the Taiwanese population (including, potentially, their ability to detoxify arsenic) and the U.S. population. The issues have been discussed in numerous reports, including a presentation of issues (Mushak and Crocetti 1995), a detailed response (Slayton et al. 1996), and a rebuttal (Mushak and Crocetti 1996). These differences in the Taiwanese population include their water intake, diet, hygiene, and exposure to other carcinogenic chemicals in drinking water as well as sources of arsenic other than drinking water (arsenic-contaminated water was used for agriculture and aquiculture). The current arsenic SF is based on skin cancer; however, the NRC report found

supporting evidence of internal organ cancers in populations in Argentina and Chile exposed to arsenic concentrations similar to those in Taiwan. In terms of diet, Argentina and Chile are more similar to the United States than Taiwan (this issue is discussed further in Section 7.2.3). The findings regarding internal organs support the current arsenic SF and indicate that it may even underestimate the cancer risks related to internal organs.

Skin cancer was not observed among 145 persons exposed to arsenic in drinking water in Millard County, Utah, at a concentration of 6 µg/kg-day (Southwick et al. 1983; Valberg et al. 1993). A number of studies did not report any skin cancer below this exposure level (Abernathy et al. 1989; USEPA 1988; ATSDR 1993). However, these studies did not have sufficient statistical power to detect effects if they existed and they did not consider internal organ cancers.

A recent study of arsenic exposure in the United States population has been published for an area of Utah where arsenic is naturally high in drinking water (Lewis et al. 1999). This study did not find an association between arsenic exposure and the arsenic cancer endpoints discussed above such as skin, lung, or bladder cancer. However, the study did find a positive association between arsenic exposure and increased cardiovascular effects. Vascular effects have been found to be associated with arsenic in other studies in the United States and elsewhere (Engel and Smith 1994, see also Section 4 and Appendix H). Because the population evaluated in the Lewis (1999) study was primarily of Mormon faith, the applicability of the results to the rest of the United States population should be interpreted with caution. The majority of the U.S. population is generally not as non-smoking, not as non-drinking, and not as healthy as the Mormon population. These factors can increase the susceptibility of individuals to disease.

Exposure Scenario Combinations

Combining exposure scenarios would result in higher hazard/risk estimates than presented for individual scenarios in this risk assessment. As an example, hazard/risk results were calculated for a combination of child/adult resident and neighborhood recreational scenarios in Section 5. Combining the two exposure scenarios resulted in more exposure areas with hazard indices higher than 1.0 and cancer risks higher than 1×10^{-4} than for individual scenarios. Therefore, calculating hazard/risk estimates for individual exposure scenarios could underestimate hazard/risk at the site in cases where combinations of exposures occur.

However, combining scenarios could also result in overestimates of hazard/risk for actual individuals. For example, child/adult residents are assumed to spend 24 hours/day, 350 days/year at their residence**.** Assuming that residents also regularly spend several hours each day at a neighborhood or public recreational area or also are exposed occupationally at the site results in “double counting” (exposure for more than 24 hours/day), which will overestimate hazard/risk. The combining of exposure scenarios conservatively assumes that the maximally exposed individual for one route and pathway is the same as the maximally exposed individual for all the other routes and pathways.

7.2 FACTORS LEADING TO POSSIBLE UNDERESTIMATION OF RISK

Potential underestimation of risk is always possible because sampling every square inch of a site is technically unfeasible, toxicity data are often incomplete, simplifying assumptions must be made, and all hypothetically possible conditions and pathways cannot be assessed. The protective assumptions presented in Section 7.1 are intended to balance factors that tend to underestimate risk. Some of the potential sources of underestimation are discussed in the following subsections.

7.2.1 Data Collection and Evaluation

Despite extensive sampling of soil, sediment, surface water, and groundwater throughout the Basin, some unsampled areas could have higher or lower concentrations than ones that were sampled.

Surface Water Sampling

Surface water samples were collected from the five water bodies noted in Section 7.1.1 (Elk Creek, Pine Creek, Canyon Creek, Nine Mile Creek, and the South Fork). These samples consisted of undisturbed water, i.e., no special effort was made to stir up the sediment prior to sample collection. Data from a total of 220 samples from these five water bodies were used in the EPC calculations. This data set includes water sample results from high-flow events when the water would be expected to carry an increased load of sediments (early spring runoff from storm events). However, it also includes data from samples collected at low flow. While most of these streams have limited amounts of sediments along the stream channels (unlike the lower Coeur d'Alene River) and recreational use would be expected to be minimal during high-flow events, additional exposure to metals in suspended sediments for individuals during water play in the creeks may be underestimated.

Sediment Sampling

The sediment data used to evaluate the neighborhood exposures for all geographical areas except the Lower Basin came from un-sieved samples, because the samples were not collected specifically for the purposes of the HHRA. Sediment data from sieved samples were not available for these areas. The smaller size fraction of sediment and soil generally has higher concentrations of metals than bulk samples. Therefore, using un-sieved data for neighborhood sediment exposure concentrations likely underestimates the magnitude of exposure for that pathway.

Chemicals Not Selected for Risk Assessment in Soil

Chemicals were selected for in-depth analysis in the risk assessment if they exceeded screening values that were based on residential exposures. For the general population, residential exposures would be the highest that would be expected for any media. However, the risk assessment also evaluated subsistence exposures. The special subsistence population could potentially have exposures to media greater than residential populations. Thus some chemicals, which were screened out, might present a

health risk for subsistence populations. Two things make the potential underestimation of COPCs for subsistence populations less of a concern:

1. The results of the risk calculations have identified this sub-population as having risks and hazards well in excess of target health goals for all media evaluated, so adding additional chemicals would not change the conclusions of either the risk assessment or potential risk management decisions.
2. The metals identified as a health concern are those associated with the mining history and are the most toxic to people. Therefore, they would be the risk drivers no matter how many additional chemicals were selected.

Three chemicals in soil and sediment had a handful of concentrations exceeding screening values based on residential exposures. Those three chemicals are discussed further below.

Mercury in soil and sediments was screened out from further evaluation in the data evaluation process because its exceedance over the SV would not contribute significantly to site hazards. Approximately 13 percent of the mercury concentrations exceeded the SV with less than 0.1 percent of the concentrations exceeding the residential PRG of 22 mg/kg (10 times the SV). In addition, the few mercury samples with concentrations greater than the residential PRG were all collocated with lead concentrations in excess of 2,000 mg/kg (6 samples out of 4,208 had concentrations greater than the PRG). Thus, soil remediation for lead would be expected to also remove any scattered mercury concentrations over the PRG. Appendix D includes a histogram of mercury concentrations in soil/sediment showing the majority of the concentrations at less than the SV. A UCL_{95} of the mean concentration was less than the SV, indicating that hazards due to mercury in soil would likely be 0.1 or less. Thus, mercury is not a significant contributor to site hazards and its exclusion from the risk assessment does not affect the conclusions of the risk assessment.

Thallium was also not selected as a COPC in soil and sediment. Thallium concentrations showed a pattern similar to that for mercury with concentrations in approximately 13 percent of the samples exceeding the SV and approximately 0.9 percent of the data exceeding the residential PRG. Thallium had the lowest frequency of detection of any metal (16 percent). Approximately 75 percent of the sample quantitation limits for thallium in soil exceeded the SV of 0.52 mg/kg and 0.1 percent (four samples) exceeded the PRG of 5.2 mg/kg. The majority of the sample quantitation limits were less than 2 mg/kg (approximately 80 percent). If thallium concentrations in the four samples with non-detections (sample quantitation limits over the PRG) were actually greater than the PRG, the hazard due to this chemical would not substantially increase. When thallium concentrations in soil and sediment are graphed using half the sample quantitation limit for the non-detected samples, the majority of the data are less than the SV and hazards would be approximately 0.1 or less for residential exposures and less than that for recreational and occupational exposures. If the concentrations in the samples with non-detections were closer to the sample quantitation limits, hazards due to thallium would still be unlikely to exceed 0.2 and, thus, would not have an impact on the results of risk assessment. Consequently, thallium is unlikely to contribute significantly to health hazards even if actual concentrations in the

samples with non-detections are just below the sample quantitation limit, rather than the default assumption of half the sample quantitation limit. Thus, the exclusion of thallium from the risk assessment does not affect the conclusions of the risk assessment. Appendix D includes a histogram showing the distribution of thallium concentrations in soil and sediment along with the mean and UCL_{95} of the mean.

Aluminum in soil and sediments was screened out from further evaluation because its concentrations are potentially at background for the area, and no concentrations exceeded the PRG. In addition, aluminum is likely present in a form that is less bioavailable than that used in deriving the provisional RfD. However, the majority of the data in CSM Units 1 and 2 (86 percent and 90 percent, respectively) exceeded the SV and 18 percent of the data exceeded the SV in CSM Unit 3. If aluminum had been included in the risk assessment, it would likely have resulted in HQs between 0.2 and 0.6 for residential soils, which could have an impact on the results of the risk assessment for some exposure scenarios. Aluminum concentrations are likely at or below the background concentrations for northern Idaho. The mean background concentration of aluminum for the western United States is estimated at 58,000 mg/kg, with a range of 5,000 to 100,000 mg/kg (Shacklett and Boerngen 1984). The maximum concentration in soil samples collected in the Basin was 52,000 mg/kg. Although quantifying aluminum exposures would increase hazard estimates, remediation efforts typically do not address chemicals that are present at background concentrations. Thus, including aluminum in the risk assessment would not affect ultimate risk management decisions.

7.2.2 Exposure Assessment

Exposure Factor

Estimates of exposure duration are based on best estimates of what exposures will be. If work or recreational patterns include more time in contaminated areas than the amount estimated, the current exposure estimates may underestimate actual exposure for the occupational and recreational scenario (exposures for the residential scenario assume all time is spent at the home).

Individuals within a population may have higher exposure rates than assumed by the separate exposure assumptions. However, the RME values used represent the maximum exposures that could reasonably be expected to occur in the population.

Under RME conditions, individuals are expected to be exposed to the Upper Confidence Limit of the mean concentration of chemicals for 30 years. Some individuals may live in the area for longer than 30 years.

Complete Pathways Excluded From Evaluation

Not all possible pathways were quantified. Potentially complete pathways that were not selected for quantification include inhalation; ingestion of beef, wild fowl, game, or berries from the lower floodplain; and dermal contact with water (all metals) and soil (all metals except arsenic and cadmium). Some of

the exposure pathways in the floodplain of the Lower Basin are discussed further in the paragraphs below. In general, excluding these pathways is assumed not to affect the conclusions of the risk assessment because where metals are the chemicals of concern, the soil ingestion pathway is always the main component determining site risk (Glass and SAIC 1992; USEPA 1995b; Weston 1989, 1996).

Ingestion of Local Beef. Local residents who eat locally raised beef are the population of concern for this pathway because beef purchased on the open market is typically not from only one source. Most individuals consume beef from cattle raised in a location other than their immediate community; thus, consumption of beef from local cattle will not contribute significantly to total exposure in the general population. Only 2.4 percent of the total meat consumed in the average U.S. household is from home-raised cattle and only 3.8 percent of the beef eaten in the average household is from home-raised cattle (USEPA 1997a). Because beef from home-raised cattle is consumed by only a small portion of the U.S. population and it constitutes a small percentage of the total diet, this pathway does not significantly contribute to total exposure for the general U.S. population.

Although metals taken up into plant tissue are available for ingestion by beef cattle and other herbivores, most metals are generally present in plant tissues at concentrations that are typically orders of magnitude less than the surrounding soil concentrations. Except for cadmium, these metals do not tend to bioaccumulate in plant tissue. For example, the recommended root uptake factors (ratio of concentration in plant tissue to the concentration in the soil) for arsenic, cadmium, lead, and mercury are 0.0004, 0.04, 0.002, and 0.05, respectively (CalEPA 1996). However, other studies have shown that concentrations of cadmium in some plant tissues are higher than those in surrounding soils (Nwosu, Harding, and Linder 1995), and cadmium was found to be a health hazard for vegetable consumption.

In addition, the amount of soil ingested by cattle while grazing is small, approximately 0.05 percent by weight of the pasture grasses ingested (CalEPA 1996). A study that investigated concentrations of metals in edible beef tissue in comparison to soil concentrations could not be located. However, a conservative calculation (see Appendix J) that assumes 100 percent contaminated cattle forage, 100 percent bioavailability, and no depuration, estimates that a concentration of 2 mg/kg arsenic in soil would result in a concentration of approximately 0.0033 mg of arsenic per kg of fresh edible beef tissue. Assuming conservatively that all meat consumed is contaminated beef, a 70 kg male who consumes 250 g of meat per day would have a dose of 0.000012 mg arsenic/kg body weight-day (CalEPA 1996; USEPA 1990c, 1998a). Calculations using the most health-protective assumptions (as described above) result in some health risks that exceed the target risk goals; however, actual metal concentrations in edible tissue are likely to be lower than the conservative estimate presented here. For example, individuals are unlikely to eat 250 mg/kg of local beef every day for 70 years and the bioavailability of arsenic in soil is unlikely to be 100 percent. If the uncertainty regarding this pathway is a concern, actual tissue samples should be collected from the edible portions of locally raised beef cattle.

Ingestion of Waterfowl and Large Game. Hunting of both waterfowl and large game in the Panhandle region is limited primarily to the fall and early winter (September to mid-December) and

takes are restricted depending on the species. Take of most large game is limited to one to two animals per year per permit holder. Take of waterfowl is higher, up to 8 and 14 per year for geese and ducks, respectively. Because of the difference in body size between large game and waterfowl, the total amount of metals consumed by humans is expected to be similar for game and waterfowl. These restrictions limit the amount of game that residents can ingest.

While wildfowl have been studied and are considered no to be a concern for human health (Weston 1989), big game has not been evaluated. Only one muscle tissue sample of white-tail deer from CSM Unit 2 is available (collected by the US Fish and Wildlife Service). While no conclusions can be reached from only one sample, the results of the sample and some conservative risk and hazard estimates are presented below for discussion purposes.

Wet weight concentrations of muscle tissue were calculated from dry weight concentrations assuming 70 percent moisture (default values of non-human mammalian moisture content range from 70 to 80 percent) and using the dry weight-to-wet weight formula presented in Section 3.3.1 for vegetables. Wet weight concentrations for four of the COPC metal are:

Arsenic	0.15 ppm
Cadmium	0.19 ppm
Mercury	0.03 ppm
Lead	1.17 ppm

To estimate risks and hazards we used an ingestion rate for game corrected for body weight of 0.446 g/kg-day, the 99th percentile rate from USEPA (1997a), and the same formulas used to calculate doses from vegetables (Table 4.3 in Appendix A). Under those assumptions, hazards from arsenic, cadmium, and methyl mercury are estimated at 0.2, 0.08, and 0.1, respectively. Arsenic risks are 4×10^{-5}, assuming all arsenic is in the inorganic form, a very conservative assumption. Thus, risks and hazards from this one sample do not appear to be a large concern and would not add significantly to the total residential risks and hazards in the Basin. However, one sample and a default exposure formula are insufficient to either include or exclude and exposure pathway. The deer was not collected from the area with the most contaminated soils (CSM Unit 3) and it is not known if the deer grazed in that area. Therefore, this pathway remains an uncertainty, is a potential source of metals for hunters, and may warrant further investigation.

Inhalation of Non-lead Metals in Dusts During High-dust Activities. Inhalation of metals in dusts was excluded as a pathway of concern for all metals but lead (see Section 2.4.5). The methodology used to evaluate dust inhalation risks makes an assumption that dust in the air comes primarily from soil being eroded by wind action. Health-protective assumptions are made regarding the "erosion potential" of the soil and how much dust will be in the air every day for 30 years. No non-lead chemicals were at high enough concentrations to present an inhalation health risk or hazard based on inhalation of wind-generated dust from soils; therefore, the inhalation pathway was not included in the risk assessment. However, some types of activities generate airborne dust in higher amounts than

would be produced by wind erosion such as recreational use of all terrain vehicles (ATVs) or certain types of construction activities. Workers engaged in soil disturbing activities in mining-impacted soils will need to take precautions to prevent exposure from a variety of pathways, including inhalation. Recreational exposures are typically infrequent and/or are of relatively short duration; therefore, exposures from ATV use are unlikely to pose a health risk if continuous exposure to dust under residential exposure conditions does not pose a risk. Nonetheless, frequent and lengthy use of ATVs on high-concentration soils such as waste piles could add significantly to exposures; thus, the lack of inclusion of this pathway could underestimate health risks for some individuals.

7.2.3 Toxicity Assessment and Risk Calculations

Bladder and Lung Cancer From Ingested Arsenic

After an extensive review of available literature, the NRC report evaluating health risks from arsenic in drinking water concluded that in addition to the previously documented risk of skin cancer, there is sufficient evidence to link ingested arsenic with bladder and lung cancer (NRC 1999). Increased rates of bladder and lung cancer have been observed in humans who ingest arsenic in drinking water containing several hundred µg/L. Two recent studies (discussed below), published since the NRC report, found an increased risk of bladder cancer at arsenic levels in drinking water well below previous reports. The NRC concluded that the evidence is insufficient to depart from the default assumption of a linear relationship (using EPA criteria in USEPA 1996e) and the two additional studies support that conclusion. The NRC report estimated that combined cancer risks due to drinking arsenic at the MCL of 50 µg/L might be as high as 1 in 100, an order of magnitude greater than the risk of skin cancer by itself (NRC 1999). The NRC acknowledges a number of problems with quantifying the dose response relationship for internal organ cancers; however, it recommends lowering the MCL as soon as possible because the current MCL likely does not meet EPA criterion for public health protection (i.e., risks falling within the range of 1×10^{-4} to 1×10^{-6}). Therefore, total cancer risks due to arsenic based on the skin cancer SF could be underestimated if risks from lung and bladder cancer were also considered.

Recently published findings (Chiou et al. 2001) for a large group (8000+) of arsenic-exposed northeast Taiwan residents found an association between arsenic in drinking water and an increased risk of bladder cancer at lower arsenic levels than have been noted in most other studies. The northeast Taiwan population is different than the heavily studied southwest Taiwan population in that each household had its own well; thus, enabling a more precise estimation of individual exposure than was possible in SW Taiwan where several households shared a well. Thousands of individual water arsenic samples (N=3901; 1/household, 85.1% of all households) were tested in this most recent investigation; consequently, dose characterization provided a better exposure reconstruction than was possible in the many, earlier SW Taiwan studies. Like the earlier studies, however, these subjects presented an overall increased risk of urinary tract cancers within a clear dose-response relationship. Chiou et al. showed the range of the dose-response relationship to begin at least at water arsenic levels below 50 ppb (MCL). In particular, they noted that there was a rough doubling of urinary cancer tract risk in the range of arsenic water levels of 10 to 50 ppb.

The Chiou et al. paper was preceded by the findings of Kurttio et al. (1999) showing increased bladder cancer rates for Finns imbibing well water arsenic levels below those linked to increased cancer risk and stratified as to dose-response in the Chiou et al. investigations. The arsenic levels reported in Kurttio et al. were quite low, the highest value being 64 ppb. An increased bladder cancer risk was observed in individuals with arsenic levels above 0.1 ppb and the finding was statistically significant at arsenic concentrations above 0.5 ppb. The authors note that their finding of a relative risk of bladder cancer above two at these low water concentrations is not consistent with other arsenic studies and note that the role of bias and chance in their findings needs to be carefully considered. The Finnish data do support linearity more than nonlinearity of the arsenic dose-response curve for internal cancers.

Chiou et al. (2001) juxtaposed the Finnish findings with their NE Taiwan finds, leading the authors to state:

> "A dose-response relation between arsenic and bladder cancer was also found in Finland...When the results of these two studies are considered together, arsenic appears to induce bladder cancer in a biological gradient at levels less than 50 Fg/liter (ppb)."

In addition to the strong evidence of lung and bladder cancer associated with ingested arsenic, there is additional evidence from the Taiwanese population of an association between arsenic in groundwater with cancer in other internal organs such as the kidney, liver, and colon (Chen et al. 1985, 1986). A dose-response relationship between arsenic in well water and cancer of the liver, nasal cavity, bladder, kidney, lung and prostate has been reported (Chen and Wang 1990). Studies in Argentina indicated an increase in kidney cancers in addition to skin, lung, and bladder cancers (Hopenhayn-Rich et al. 1996; Hopenhayn-Rich, Biggs, and Smith 1998). It is unknown whether the current SF for arsenic protects against these additional types of cancer.

Risks and Hazards Added to Residential Exposures

Risks and hazards due to the ingestion of locally grown vegetables and local fish were not combined with other risk/hazards in the risk and hazard totals presented in Section 5. The RME hazard indices for eating cadmium in fish ranged from 0.005 to 0.02 indicating that an increase in hazards from cadmium in fish is unlikely to affect the hazard totals or risk assessment conclusions if added to the residential scenarios. The RME hazard indices for eating mercury in fish were 0.3, 0.9, and 0.6 for bullhead, northern pike, and perch, respectively. If local fishermen eat only bullhead, total hazards for residents would not significantly be affected, if fish ingestion hazards due to methyl mercury were added to residential scenarios. However, for local fishermen who eat primarily northern pike, and secondarily perch, the hazards from eating fish containing methyl mercury could add significantly to residential scenarios at some locations. The mercury concentration in northern pike used in the risk calculations was 0.133 ppm and the value for perch was 0.089 ppm.

The ATSDR (1993) reports for mercury that the average concentration in most fish is less than 0.2 ppm and UCL_{95} mercury concentrations in fish tissue from the lateral lakes are below this value. The highest concentration of mercury in fish from the lateral lakes was a value of 0.48 ppm in northern pike.

The mercury concentrations used in the risk calculations are similar to mercury levels seen in uncontaminated areas and are similar to average values seen in fish across the country. The USGS sampled fish filets and sediments at 16 locations in Montana, Idaho, and Washington and analyzed the samples for metals, including mercury (Maret and Skinner 2000). The locations included Mullan and Pinehurst on the South Fork of the Coeur d'Alene River, and Enaville on the North Fork. They also sampled two locations along the St. Joe River, an area that could be considered to represent natural background for local fish tissue concentrations. The USGS report did not find any correlation between sediment concentrations and filet concentrations for any of the metals sampled, including mercury, cadmium, and lead, indicating that sediment concentrations did not appear to be a good predictor of filet concentrations (Maret and Skinner 2000). Researchers did not report that they considered the home range of the fish in their correlation estimates. Mercury levels in sediment need to be weighted to accurately reflect concentrations in the entire feeding range if fish range over a wider area for their food then is represented in the sediment sampling geostatistics. Researchers did not sample the same species as those evaluated in this risk assessment, the USGS sampled several species of trout, mountain whitefish, and largescale sucker. Samples were composited from up to 16 fish per location and one sample was sent to the laboratory for analysis. Pinehurst had the lowest mercury concentration in filet tissue although this location had the highest mercury concentrations in sediment (the filet concentration was approximately 0.07 ppm). Mercury concentrations in the two filet composites collected from the St. Joe River were approximately 0.13 ppm and 0.17 ppm. USEPA (1992e) studies of mercury concentrations in fish across the country found a median mercury concentration in filets of 0.18 ppm and mercury concentrations in the area of 0.18 ppm may be a concern. The St. Joe River concentrations are equal to or exceed the UCL95 values used in the risk assessment listed above.

The RME hazard index for homegrown vegetables was 2 for child and child/adult residents and the RME cancer risk was 8×10^{-5} for child/adult residents. These values are large enough to significantly affect the hazard/risk estimates for the residential scenario in several exposure areas and could impact the overall conclusions of the risk assessment. However, the vegetable data are considered semi-quantitative because of the non-systematic sampling of gardens and the small data set (e.g., vegetable data were not available for all exposure areas because of opportunistic sampling [see Section 2]). It is unknown how many homes in the Basin have vegetable gardens and whether the homes and vegetables sampled were representative. Cadmium concentrations in leafy and root vegetables in the limited data set appear to be higher by almost an order of magnitude than the national averages, and the concentrations in Basin vegetables are similar to those found in Pinehurst in 1983 (PHD et al. 1986).

Risks from eating vegetables containing arsenic were calculated assuming 100 percent of the arsenic in produce was inorganic (which is accepted as being more toxic than organic arsenic). The assumption of 100 percent is potentially an overestimate of inorganic arsenic in many foods, with some produce having as little as 25 percent of its arsenic content in the inorganic form (Schoof et al. 1999; Yost et al. 1998;

Mushak and Crocetti 1995). In the absence of site-specific speciated arsenic data and acknowledging that the types and amounts of produce consumed will vary between individuals, the 100 percent assumption is not unreasonable. However, the amount of inorganic arsenic in produce is another area of uncertainty, indicating the risk estimates from eating garden produce should be viewed with caution.

Not combining the results for vegetables with hazard/risk estimates for residents may have resulted in an underestimation of hazard/risk for some residents. The magnitude of the underestimation is unknown due to the uncertainty regarding the data set for homegrown vegetables and the uncertainty of the amount of inorganic arsenic ingested in produce.

7.3 FACTORS LEADING TO POSSIBLE UNDERESTIMATION OR OVERESTIMATION OF RISK

Several factors have the potential to overestimate or underestimate risk. This section discusses these factors and the likely effect of combining uncertainties in evaluating risk.

7.3.1 Data Collection and Evaluation

A risk assessment depends heavily on the quality and representativeness of the sampling data. Uncertainties contributing to sample variation may involve the heterogeneity of the sample matrix (e.g., particle sizes in soil), the number of samples collected in various locations, and the field or laboratory analytical techniques. These sampling uncertainties can underestimate or overestimate risk.

House Dust

The house dust data for the non-lead metals were not used in the EPC calculations for residential exposure primarily because of (1) the lack of sufficient dust data (in six of the eight residential areas fewer than 10 dust samples were available [see Table 3-~~3~~21]), and (2) the uncertainty associated with the prediction of a dust concentration on the basis of a yard soil concentration. Scatter plots of house dust versus soil (Appendix I) indicate no clear relationship between the concentrations in house dust and yard soil, i.e., as concentrations in soil go up, concentrations in dust do not necessarily increase also and vice versa. Because of the time spent in the house, especially by young children, a significant portion of a child's soil exposure occurs from dust in the home. In the EPA IEUBK Model for lead, the amount of time spent outdoors is assumed to represent 45 percent of the child's day, while 55 percent of the child's exposure occurs indoors (USEPA 1994a). As a result, metal concentrations in indoor dust that are significantly different from concentrations in outdoor soil could make a difference in the EPC and the estimate of risk or hazard.

Two types of house dust samples were collected: dust from floor mats and dust from vacuum cleaner bags. The data from vacuum cleaner bags are considered more representative of dust exposures in the home than data from floor mats, while floor mats provide a better estimate of the total amount of outside soil tracked into the home. Once the soil is in the home, other dust generated inside the home likely

dilutes the soil particles (unless there is an in-home source, such as with lead) (Trowbridge and Burmaster 1996). Examples of dust particles generated in the home are flaking skin scales, dust from carpet lint, organic material, and flakes of construction material (Trowbridge and Burmaster 1996). As a result, concentrations of metals in the vacuum cleaner bag samples are expected to be lower than those in outdoor soil because of the dilution of outdoor soil by indoor-generated dust (Trowbridge and Burmaster 1996). Concentrations of metals in floor mat samples are expected to be similar to those in outdoor soil. Iron and manganese were the only two metals that showed the expected pattern—concentrations in outdoor soil samples and floor mat samples were the same and concentrations in vacuum cleaner bag samples were about half those in outdoor soil.

Concentrations of antimony, arsenic, cadmium, and zinc all showed a statistically significant enrichment in floor mat dust when compared to outdoor soil. All but arsenic also showed an enrichment, though not as great, in vacuum bag dust. The results of the paired t-test performed on floor mat and vacuum cleaner bag concentrations versus outdoor soil concentrations are presented in Appendix I. The statistical comparison was performed after the removal of one outlier sample from each of the chemical-specific data sets, with the exception of antimony, which had no outliers. Data were tested for outliers using the studentized residual test performed in SYSTAT *v*.9. For the statistical comparison, the soil data were pooled for each chemical for all the homes with floor mat and vacuum cleaner data and were found to be lognormally distributed. Residential soil data for different geographical subareas presented in the Part D Table 3 series were often not lognormally distributed.

The statistical results indicate that concentration dilution is occurring in the home (vacuum cleaner bags) relative to entry into the home (floor mats); however, the concentrations of all metals, except arsenic and lead, in dust from vacuum cleaner bags exceeded the concentrations in outdoor soil. The reasons for the concentration enrichment are unknown. Possible explanations include the following:

- The mechanisms by which yard soil is being transported into the home preferentially bring in particles smaller than those (less than 175-micron diameter) collected in the yard soil samples. Because samples consisting of smaller particles have higher concentrations of metals, the concentrations in house dust are greater than those in outdoor soil.

- Bias may be introduced by the comparison of results from different sampling methods for house dust and soil.
- There may be sources of outdoor metals entering homes that are not from the yards.
- A potential indoor source of metals is being added to metals tracked in from the yard.

Further investigation is needed to explain the results (e.g. more data gathering, further analysis, etc.). Depending on the chemical and the dust data used, concentrations in house dust could either be the same as those in outdoor soil, less than those in outdoor soil, or greater than those in outdoor soil.

Ratios of chemical concentrations in soil to those in dust from floor mats and vacuum cleaner bags are presented in Table 7-2. Arsenic and iron are the chemicals contributing most to the noncancer hazard in

residential homes. Both their concentrations were generally lower in dust from vacuum cleaner bags than in outdoor soil, indicating that EPC concentrations could be overestimated for these metals; however, arsenic concentrations in the floor mat samples were higher than those in outdoor soil. If dust samples from vacuum bags represent the best measure of indoor dust, the use of soil concentrations as a surrogate for house dust concentrations for arsenic and iron likely over estimates health risks.

Vegetables

Vegetable samples were not collected in a systematic manner. As discussed previously, homes were sampled because the owners volunteered and only a small percentage of the homes had produce available for sampling. It is unknown how many homes in the Basin have vegetable gardens and whether the homes sampled were representative. Furthermore, the same produce was not sampled in every garden; the 24 sampled gardens did not have similar produce and a wide variety was sampled. Leafy vegetables and root vegetables were preferentially sampled from every garden, if available. For some types of produce only a few samples were collected and for some only one sample was collected. Because of the non-systematic sampling and the small data set, the vegetable data are considered semi-quantitative. It is unknown whether the data underestimated or overestimated the metal concentrations in homegrown produce. The estimated HQ associated with cadmium in vegetables is 2, which exceeds the target health goal of 1. Issues surrounding cadmium in vegetables are discussed further in the following text.

The database for cadmium concentrations in garden soil is much larger than that for cadmium in vegetables. When the concentrations of cadmium in vegetables are compared with the cadmium concentrations in collected garden soil, no trends are apparent. Figures 7-1 through 7-3 show scatter plots of cadmium concentrations in vegetables and soil for (1) all vegetables, 31 paired samples; (2) leafy vegetables, 11 paired samples; and (3) root vegetables, 19 paired samples. The plots do not indicate a relationship between cadmium concentrations in vegetables and soil (i.e., vegetable concentrations do not increase as soil concentrations increase). As a result, it is not possible to use cadmium concentrations in garden soil to determine whether the homes sampled for homegrown vegetables were representative of the entire site. The average concentration of cadmium in leafy vegetables is 0.42 mg/kg and in root vegetables it is 0.13 mg/kg. These concentrations are higher than those indicated by national survey data, which show the mean cadmium concentration in leafy vegetables as 0.033 mg/kg (ranging from of 0.016 to 0.142 mg/kg) and in root vegetables as 0.016 mg/kg (ranging from a trace to 0.028 mg/kg) (Gartrell et al. 1986). The average cadmium concentration in root vegetables in the Basin is higher than the highest value reported in ATSDR (1993). Recent surveys by the Food and Drug Administration (FDA) note that 80 percent of cadmium in the diet comes from lettuce, potatoes, and grains (Gunderson 1995).

Garden vegetables and garden soil were sampled at homes in the BHSS (PHD et al. 1986) in 1983. The dry-weight results for cadmium concentrations are compared with EPA's 1998 sampling results for the Coeur d'Alene Basin in Table 7-3. The sampling results are provided in Appendix B. Only dry-weight values were reported in the 1983 data; therefore, EPA dry weights are used for comparison.

Exposure to vegetables occurs to the wet product; therefore, wet-weight concentrations were used in the hazard calculations and wet-weight concentrations are shown in the figures. Wet-weight concentrations are much lower than dry-weight concentrations because vegetables contain a large percentage of water.

The 1983 and 1998 vegetable data are similar. Both indicate that leafy vegetables have higher concentrations of cadmium than root vegetables and that concentrations are a similar order of magnitude.

In conclusion, leafy vegetables, particularly lettuce, in the Coeur d'Alene Basin appear to have the highest concentrations of cadmium. This finding agrees with national survey information indicating that this vegetable type generally has the highest cadmium levels. (The latest FDA survey indicated that potatoes contain slightly higher cadmium concentrations than leafy vegetables [Gartrell et al. 1986]). Average cadmium concentrations in leafy vegetables and root vegetables, in the limited data set available for the Basin, appear to be almost an order of magnitude higher than the national averages, and Basin concentrations are similar to those found in Pinehurst in 1983 (PHD et al. 1986). Cadmium concentrations for vegetables in the Basin do seem elevated compared to the rest of the country.

Health risks due to cadmium in vegetables were estimated using a total vegetable ingestion rate for homegrown vegetables that was not vegetable specific. Actual health risks could be higher or lower, depending on the amount of leafy vegetables versus other types of vegetables consumed. On average, cadmium concentrations in leafy vegetables were three times the concentrations in root vegetables. Hazards due to cadmium in vegetables exceeded the target hazard goal, indicating that this may be an exposure pathway of some concern and potentially worth evaluating further to eliminate some of the uncertainty.

7.3.2 Exposure Assessment

Gastrointestinal Absorption of Arsenic From Soil

As recommended in EPA Region 10 guidance, a gastrointestinal absorption factor of 60 percent was used in exposure estimates for arsenic ingested in soil (USEPA 2000b). Confidence in the data set used to estimate 60% absorption is limited. Actual bioavailability is likely to vary across the site and would be higher or lower than 60%. In portions of the Basin, arsenic derived from residual smelter emissions may be more bioavailable than 60%. The EPA oral RfD and SF for arsenic were derived on the basis of an epidemiological study of a group of people who were exposed to high levels (e.g., intake of approximately 1,000 µg/day) of soluble arsenic in food and water. However, arsenic ingested in soil at mining sites is generally less well absorbed than soluble arsenic in food and water because (1) the arsenic form may be relatively insoluble (Davis et al. 1996), and (2) arsenic adsorbs to soil particles, making it less available for absorption (Valberg et al. 1997). Therefore, the gastrointestinal absorption factor of 60 percent was used to account for differences between the bioavailability of arsenic ingested in soil at

the site and the bioavailability of soluble arsenic ingested in water and the diet in the study used to derive the SF.

EPA Region 8 has used a generic gastrointestinal absorption factor for arsenic in soil of 80 percent for arsenic in smelter-derived waste and 50 percent for arsenic in mining-derived waste (USEPA 1993a). The assumption of lower bioavailability of arsenic in soil has been tentatively confirmed by results from animal studies indicating that soil-bound arsenic is not as bioavailable (8 to 48 percent) as arsenic in solution (Freeman et al. 1993, 1995; Groen et al. 1994; Yanez et al. 1993). However, the number of animals used in the study was small. In addition, a number of human studies have reported low urinary arsenic levels at sites with high arsenic concentrations in the soil, probably as a result of the low bioavailability of arsenic in soil (Butte-Silver Bow Dept. of Public Health and Cincinnati Dept. of Environmental Health 1992; Colorado Dept. of Health et al. 1990; Hewitt et al. 1995; Valberg et al. 1997). Site-specific gastrointestinal absorption factors for arsenic have been derived using soil from the Murray Smelter Superfund site in Utah (24 percent), and residential soil (80 percent) and slag dust (42 percent) from the Ruston/North Tacoma Superfund site in Washington (Weis, Henningsen, and Griffin 1996; USEPA 1993b, 1996d, 1997d).

The gastrointestinal absorption factor of 60 percent used in this risk assessment is within the range of values discussed. However, this is a source of uncertainty in the estimates of hazard/risk due to arsenic ingestion in soil.

Current Conditions Do Not Change

EPA residential exposure assumes that the current conditions remain the same over 30 years of exposure. Over this period, conditions may improve (e.g., many areas will be cleaned up after which cleaner sediments potentially will be deposited over the formerly contaminated areas) or, conversely, areas now contained could be disturbed (e.g., large flood events mobilizing new waste pile materials with high concentrations of metals). Any of these conditions could affect the actual amount of exposure.

7.3.3 Toxicity Assessment

Arsenic Toxicity Issues

There is uncertainty associated with the effects of arsenic at low exposure doses. Because of the controversy surrounding the carcinogenicity and toxicity of ingested arsenic, further revisions to the SF may occur in the future. Some of the outstanding issues previously discussed in this section include (1) a possible threshold dose (i.e., risks are less than predicted based on the linear model relationship between dose and cancer risk at low doses); (2) the increasing body of epidemiologic evidence linking internal cancers with ingested arsenic exposures, particularly of the bladder and lung; and (3) the differences in absorption between arsenic in drinking water versus soil.

A new study by Kaltreider et al. (2001) provides a mechanistic rationale for the genesis of diseases that are linked to glucocorticoid receptor (GR) disruption and simultaneously known to be associated with arsenic: vascular disease, diabetes (Type-2) from disruption of GR-mediated glucose metabolism, and molecular events leading to carcinogenesis. It is not yet clear how this new information might affect the numerical estimates of arsenic toxicity, but the study provides mechanistic support for some of the diseases that have been linked with arsenic exposure in epidemiologic studies.

Kaltreider et al. (2001) showed in rat studies that arsenic, used in the chemical form usually found in drinking water, interferes by disruption with a class of hormones called the glucocorticoids. These hormones serve multiple critical functions in the body, such as control of the blood vessels, blood glucose regulation, cell differentiation, and apoptosis, apoptosis being the process of controlled or programmed cell death. They function by connecting with a target site receptor (the GR).

The results say that arsenic interferes with glucocorticoids and their receptor function by way of altering in a newly found way how the hormone binds to its target tissues, thereby impairing glucocorticoid function. The glucocorticoid receptor, the target tissue site where the hormone interacts, is blocked by arsenic from responding in a way that puts it in a separate class of disrupter, i.e., disruption by glucocorticoid receptor-produced gene expression, i.e., genetic processes within the nucleus. Arsenic interferes with the hormone receptor in exposed cells to control the expression of target genes in the target cell nucleus.

The risks presented here for arsenic were calculated based on the total arsenic concentrations in each area. However, some of the arsenic is naturally present (pre-mining background concentration). For example, the 90th percentile soil background concentration for arsenic is estimated to be 22 mg/kg (Gott and Cathrall 1980). Of the 26 different arsenic EPCs for soil and sediment used in the RME risk and hazard calculations, only 4 were less than or equal to 22 mg/kg (Table 3-2). The arsenic concentrations used in the risk and hazard calculations are not 90th percentile values, but 95 percent upper confidence limits of the mean (UCL_{95}). The background UCL_{95} for arsenic is unknown and cannot be estimated from the Gott and Cathrall report where only the 75th and 90th percentile values for arsenic were provided. The 75th percentile concentration was 10 mg/kg and a background UCL_{95} for arsenic is thus potentially lower than 10 mg/kg. Therefore, all the arsenic soil EPCs are potentially greater than a UCL_{95} natural background value.

However, for many of the soil and sediment EPCs close to 22 mg/kg, background may be contributing significantly to the total arsenic concentration. Risk management activities typically take background concentrations into account for decisions about remediation. Thus, background may account for a large percentage of arsenic risks in some areas and may affect remedial decisions. When considering the contribution of background to risks, the bioavailability of arsenic in background soil as compared to arsenic in mining-impacted soil must be taken into consideration as well as the total background concentration. Another area of uncertainty is that background arsenic may have a different bioavailability than arsenic in mine wastes and thus risks would not be equal given equal soil concentrations. If bioavailability was equal, at an arsenic concentration of 10 mg/kg, risks for residential

exposures would be 3 x 10^{-5} (ingestion and dermal soil exposure, 60 percent gastrointestinal absorption rate). Cancer risks from arsenic due to residential soil exposure (highest risks) ranged from 5 x 10^{-5} to 1 x 10^{-4} for the eight residential areas evaluated (current conditions). Therefore, where risks from residential soil exposure are at the low end of the calculated risk range, below 6 x 10^{-5} (Kingston, Silverton, and Wallace), risks from a natural background concentration of 10 mg/kg could account for half the risk. For the five remaining residential areas which had risks from residential soil of 1 x 10^{-4} (Lower Basin, Side Gulches, Osburn, Mullan, and Nine Mile), the risk due to the background concentration of arsenic could account for approximately 30 percent of the risk. The incremental increases in risk above background are approximately 3 x 10^{-5} and 7 x 10^{-5} for the lower risk and higher risk areas, respectively. All increases above background are, therefore, greater than 10^{-6}, even if bioavailability is assumed to be the same and the background UCL_{95} is assumed to be 10 mg/kg. This same discussion would also apply to noncancer hazards due to arsenic.

Reference Dose for Iron

The RfD for iron is a provisional one; it has not gone through the peer review process necessary for its inclusion in the EPA on-line IRIS database. Therefore, the provisional RfD may be reevaluated and subsequently be raised or lowered. Such a change would increase or decrease the hazard estimates for iron in this report.

COPC Interactions

The potential toxicity-modifying interactions between COPCs in the Coeur d'Alene Basin are numerous and complex. Some of these interactions could be expected to increase toxic effects, some would reduce toxic effects. The following examples illustrate some of the complexities and potential interactions (TVA 1999):

- Several of the COPCs are required for nutritional adequacy in these concentrations (iron, manganese, and zinc) and have limited toxicity unless intake is excessive or through an inappropriate pathway. For example, an iron intake of 0.3 mg/kg body weight is recommended for nutritional adequacy, and an oral dose in excess of 40 mg/kg body weight is likely to be toxic. The RfD used in the hazard calculations for iron was 0.3 mg/kg and hazards slightly greater than 1 are, therefore, unlikely to be a health concern for a child who has an adequate nutrient intake.
- Some COPCs that are essential nutrients may have adverse effects by altering the physiological balance with another essential nutrient COPC. For example, iron deficiency increases the absorption of ingested manganese.
- The toxicity mechanisms of some of the COPCs are at least partially attributable to interference with normal metabolism of the COPCs that are essential nutrients. In some cases, toxicity may be ameliorated by providing what under normal circumstances might be considered an inordinate level of an essential nutrient. For example, cadmium may

cause anemia by interfering with normal iron metabolism and absorption. These symptoms can often be alleviated by zinc supplementation.

- Dietary status may influence an individual's response to some COPCs. For example, calcium deficiency and/or physiological states causing remobilization of calcium stores (e.g., pregnancy, lactation, and aging) may increase both the absorption of ingested lead and the concentration of lead in critical organs. Iron deficiency and/or physiological states associated with increased iron requirements (e.g., infancy) enhance lead absorption and promote lead toxicity. An inadequate dietary intake of protein, methionine, or choline may limit the ability of the liver to methylate arsenic, thereby limiting arsenic detoxification and excretion.

Interactions between COPCs may result in synergism or antagonism related to the toxic effect of individual COPCs and may even fundamentally alter the mechanism of the toxic effect in vivo. For example, rats exposed to mining waste that was contaminated primarily with arsenic, manganese, zinc, copper, and lead showed an apparent interaction between arsenic and manganese. The interaction appears to influence the bioaccumulation of arsenic and manganese in brain tissue and alters the release of the neurotransmitter dopamine in areas of the brain involved in motor activity, attention, and learning (Rodriguez et al. 1998).

The potential effects of exposure to several of the COPCs, especially those classified as essential nutrients, may also be modulated by homeostatic mechanisms, including the following:

- Limitation or enhancement of uptake from the gastrointestinal tract (e.g., iron)
- Alteration of the rate of excretion (e.g., manganese).

The magnitude of the effect of these homeostatic mechanisms would be expected to vary, depending on factors such as age and nutritional status (TVA 1999).

7.3.4 Risk Calculations

The effect of combining uncertainties associated with the various assumptions in the risk assessment is partially demonstrated by the difference between "typical" (average) and RME risk calculations. To address potential uncertainties, a number of conservative estimates were used for the RME calculations, which when combined could overestimate risk considerably for most individuals. As noted by Habicht (1992), maximizing all variables will result in an estimate that is above the actual values seen in the population in virtually all cases. This recent guidance, therefore, recommends (1) the use of near maximum values for one or a few variables with the majority being mean values, and (2) the evaluation of an average case for comparison.

7.4 UNCERTAINTIES IN LEAD RISK ASSESSMENT

Each of the parameters and assumptions made in the lead health risk analysis have some associated degree of uncertainty. The data utilized in developing quantitative estimates of risk variables and parameters, and the likelihood of the assumptions made regarding each parameter, should be considered in assessing the results of the risk calculations. Most of the data collected to represent exposure and response variables and to support the development of exposure factors, were collected by standard methodologies employed in the area for many years. These methods have been peer-reviewed and critiqued by various agencies prior to implementation. For many of the parameters, typical values have evolved from other studies and guidance and have been broadly applied in similar risk assessments. Other parameters are more site-specific in nature and subject to greater range of uncertainty. Additionally, the data used were a compendium of results from a number of studies that were designed and carried out for different objectives. As a result, some uncertainty is introduced from the combining of these studies.

There are five areas of uncertainty in the overall human health risk characterization for lead that are discussed in this section: i) observed blood lead concentrations and distributions in the various populations at increased risk for lead, ii) environmental data collection and evaluation including sampling and analysis protocols, iii) the broad range of exposure parameters linked to characterizing both baseline and incremental lead exposures, iv) the multiple areas of uncertainty and variability in the modeling of blood lead levels, and v) uncertainties associated with modeling results employed to derive risk-based soil lead cleanup levels.

7.4.1 Uncertainty in the Use of Observed Blood Lead Levels

Uncertainty in Blood Lead Measurements

All blood lead samples for both the Basin and the BHSS are venous samples drawn by a certified phlebotomist. Basin samples were drawn at fixed site screenings utilizing school buildings, and BHSS samples were drawn in a hospital setting. All samples have been analyzed at the same CDC certified laboratory for the last 15 years and are subject to all Public Health Service protocols and QA/QC procedures. More than 5000 blood lead samples have been collected and analyzed in the Silver Valley by this procedure. Risk managers should consider the measured blood lead levels to be reasonably accurate.

Uncertainty in Representativeness of Blood Lead Surveys

The blood lead surveys conducted by the State and local health departments were estimated to have sampled about 25% of the 9-month to 9-year old children living in the eight geographic Basin subareas. These data were used in the HHRA i) to characterize excess absorption levels among different age groups in the various geographic subareas, ii) as a dependent variable in quantitatively assessing dose-response relationships with independent environmental source variables, and iii) to compare to blood lead modeling estimates to assess the effectiveness of different model forms in describing current blood lead levels.

Review of these data indicate significant excess absorption is occurring, particularly among younger children, in several areas of the Basin. A strong quantitative relationship (R-square = 0.60, p=0.0001) between blood lead levels and soil, dust and paint lead concentrations was noted. This empirical relationship was subsequently used to predict the effect of exposure reductions on future environmental and blood lead levels. These findings and conclusions are applicable to the population studied. A site-specific form of the IEUBK Model for lead, the "Box Model" used for the BHSS provided reasonably accurate predictions of observed blood lead levels and percent to exceed toxicity criteria for areas east of the BHSS. The EPA Default Model tended to over-predict current blood lead levels in these areas. Both models under-predicted observed blood lead levels in the Lower Basin.

Each of these findings is based on an inherent assumption that those individuals providing blood samples are representative of the overall population of the Basin. The environmental source characteristics used in the site-specific analyses are compared in Table 7-8 and these results indicate that the source variables for the blood lead population are representative to that of all houses sampled. The sampled population was self-identified in response to solicitations from the health departments over a period of four years. In 1996, the area was systematically canvassed and a randomized sampling of 843 homes across the Basin was obtained. Although the participation rate for environmental sampling of these homes was adequate, few parents availed themselves of the opportunity to have their children tested. More than 660 adults agreed to provide blood samples. However, only 98 children were tested in the 1996 survey.

The latter three screenings were directed more toward health intervention efforts and showed different participation rates. Children from age 1-9 years were tested, although tighter age band sub-grouping results were presented. In 1999, extra solicitation efforts were applied including additional publicity, public and monetary support from the local mining industry, and a $40 per child financial incentive. This survey resulted in a turnout estimated near 25% of the eligible children in the Basin and a doubling of the total blood lead observations obtained. The overall response over the four years was biased toward older children, with about twice as many children in the 7-9 year category than 9-month to 36-month old children. The nature of this turnout raises questions regarding the reliability of using these data in the HHRA and subsequent remedial decision-making. The implications of the uncertainty in the representativeness of the observed blood lead database are discussed below for each major use of information.

Characterizing Excess Absorption in the Population. Review of the blood lead data indicate that about 10% of the 9-month to 9-year old children tested are experiencing excessive blood lead levels. The high blood lead levels are concentrated in younger children with 23% of 9-month to 36-month old children showing levels of 10 μg/dl or greater and 10% at 15 μg/dl or higher. A significant portion of these children (3%) are in the medical response category greater than 20 μg/dl. These results indicate an unacceptable level of absorption is ongoing among, at least, the younger children participating in the intervention program.

Blood lead screening has associated problems that can result in uncertainty and variability, as noted in Table 7-4. Much of the variability and uncertainty is related to the relative instability of this measure in young children. Blood lead levels may change with increases or decreases in lead intake. Questions leading to uncertainty in results include the extent to which those screenings with low turnout would be representative of those individuals, especially children, who did not participate.

One statistical problem noted in screenings of this type is participant selection bias. Two potential biases are important to consider. The announcement and implementation of the surveys and associated education efforts may have caused parents to undertake exposure reduction efforts with their children that result in lower blood lead levels. These same children may have been more likely to participate than those from uninformed families. The nature of the solicitation may have resulted in drawing from particular segments of the population that exhibit higher or lower blood lead levels.

It is not clear the extent to which the publicity and concerns raised about scheduled screenings would have affected blood lead in response to any abrupt lead exposure changes elicited by care giver concerns (Mushak 1998). Judging from the low participation rate in earlier screenings, there seems to be a differential attitude within the Basin as to the seriousness of the lead exposure problem. Many community members feel strongly that there are few health risks ongoing and resent the government intervention and investigation efforts. These factors could affect the relative seriousness of care-givers attempts to prevent exposure for their children among those who elect to participate. That is, those parents most concerned will willingly use the opportunity of a community screening to have their children tested. But they may also be those who extend their concerns about lead hazards to daily exposure prevention activities for their children as well. That is, those most likely to participate are those whose children represent a likely lower level of exposure than those care-givers who are indifferent to both needs for blood lead testing and children's activities in a lead-contaminated environment (Mushak 1998). This would suggest that blood lead levels among the non-participants would be expected to be greater than those that provided samples.

On the other hand, the use of money as an incentive would be expected to particularly favor low-income participation. Because potentially high exposures are associated with poverty-related factors, higher than average blood lead concentrations would be expected among the participants. More than 30% of young children in the Basin are classified a being in poverty and a majority of families with young children could possibly be considered low-income. As a result, it is unclear whether the observed blood lead levels noted in the health department surveys are higher or lower than those children not tested.

This question was examined in the BHSS where a door-to-door screening has been ongoing for over a decade. In the BHSS, recent turnout has been about 50% of the population down from more than 70% in the early years of the program. Of those that don’t participate, about 6% are not found in door-to-door solicitations despite repeated efforts, 4% indicate previous testing, 10% believe there is no problem, 8% give no reason, and samples are unobtainable from 10% (TerraGraphics 2000a). The only particular bias identifiable among non-participants was among those that were tested before with negative results. These families would be expected to have low blood lead levels. Coupled with the

monetary incentive that favors low-income participation, this factor would suggest that overall blood lead levels at the BHSS would show lower incidence of exceedance than observed among participants. No such information exists for the Basin population.

Comparison to Health Criteria and Other Populations. There are problems in comparing Basin blood lead levels to State and national surveys. The Basin-wide 1999 blood testing results of 10% exceeding the level of concern, cannot be directly compared with a State-wide survey conducted in 1997 that found that 4.2% of pre-school children living in pre-1970 housing had blood leads greater than the level of concern because i) all the children in the Basin do not live in pre-1970 housing, ii) children 9 months to 9 years of age cannot be compared to a population of pre-school age children, and iii) the 10% exceedance in the Basin may be the result of a bias in sampling of older children, and the influence of education and intervention activities; the second of these at least would clearly not have been present in the State-wide survey.

These problems can be somewhat mitigated by examining only pre-school results in the Basin. Sixteen percent of preschoolers showed blood lead levels greater than or equal to 10 µg/dl in the most recent survey. Housing data for the Basin indicate that about 48% of housing in the Basin has been built since 1960 compared to 37% statewide, although the housing age breakdown for the children providing blood lead samples is unknown. This question is also compounded by the age distribution of children in the Basin and the subset of the population providing samples. There has been a preferential loss of younger children in the population in the last decade. The percent of children under 5 years of age has decreased by 12% as compared to an 8% decrease for all children under age 18 in the last decade. The degree to which this difference affects population blood statistics or the demographic characteristics of the preschool children remaining in the valley is unknown. With regard to risk assessment considerations, future populations would see a repopulating of the lower age groups as economic conditions improve. These families would likely be new to the area, less acquainted with lead health risks, and less affected by intervention efforts. As a result, it is likely that the prevalence of excess absorption is substantially higher in the Basin than the in Idaho generally. Risk managers may want to consider that those newcomers to the area in the future may have higher dose response rates than the current population.

Comparisons of Basin blood lead levels to national statistics is also problematic as there is clear stratification in national blood lead levels with children's age, income, age of housing, ethnicity, and size of community. These factors interplay in complex ways. Living in older poorly maintained housing may introduce additional sources of lead. However, low income status may not be a direct cause of elevated blood lead levels, but rather reflect such co-factors as poor nutritional status and having to live in older homes because they are generally less expensive and the resulting higher exposures from greater dust and lead loading rates in older homes. Both a reduced nutritional status and increased dust exposures in older homes are arguments for greater risk reduction efforts.

It is not readily obvious how the Basin should be characterized relative to the national demographic considerations. The area is not ethnically diverse and observed blood lead levels were drawn from the largely white resident population. The Coeur d'Alene Tribe, for the greatest part, resides outside of the

area of contamination. No blood lead observations specific to Tribal members are available. Community size is small to rural and currently has a high incidence of poverty among young families. However, much of the poverty and low income status has been incurred in the last two decades associated with economic downturns in the mining industry. As a result, previously established community infrastructure, local government services, a stable older generation, and a tradition of community self-reliance in place prior to the economic downturn have helped to mitigate risk co-factors associated with poverty and comparison to other low income areas may not be justified.

Calibrating or Validating Predictive Models. It is important to note that neither the EPA Default or the Box Model were calibrated to observed blood lead levels in the Basin. The EPA Model was validated using blood lead levels from a variety of sites nationally and the parameter values are expected to provide representative estimates of blood lead levels based on health protective assumptions. The Box Model was developed through structural equation analysis of the last twelve years of blood lead and environmental exposure data collected through the course the BHSS cleanup. Both models were run "as is" and results were compared to observed levels in the Basin. No adjustment specific to observed Basin blood lead levels was made.

In comparing projected lead absorption results to observed blood lead levels, there is concern that observed values may not represent true baseline blood lead levels in the Basin. Blood lead levels may be biased high or low by selection factors discussed above. Additionally, observed levels may actually be reduced due to several factors, including the influence of a general community awareness of the lead problem, and as a result of general health education and specific intervention activities in the Basin. Analysis of the Lead Health Intervention Program developed for the BHSS indicate that community education and intervention activities have a substantial effect on reduction of blood lead levels in children. It is unclear how much the introduction of LHIP activities in the Basin in 1996, or the media and public attention to the potential problems have affected people's behavior with respect to lead exposures. However, any effect these efforts had likely resulted in lowering blood lead levels and particularly among those that provided samples. Many of the children whose families received follow-up counseling provided samples showing lowered lead levels in subsequent years. Siblings in these families and possibly acquaintances and neighbors would have similarly benefitted. As a result, risk managers might want to consider that model predictions based on default assumptions are likely to over-predict this population under current conditions.

Two general types of blood lead modeling were performed in the HHRA. These two methods are generally called conventional (e.g., a predictive, mechanistic approach to blood modeling) and site-specific analysis (e.g. a descriptive, empirical approach). The traditional IEUBK approach is intended to be predictive of future, potential blood lead levels associated with a site. The site-specific approach more accurately describes blood lead trends and current conditions; its predictive value for future blood leads may be contingent upon sustained efforts and efficacy of the Lead Health Intervention Programs to monitor blood lead levels and reduce exposure in perpetuity. This possibility, in addition to the self-selection bias toward children in the older age groups may result a significant reduction of observed exceedances of 10 µg/dl blood lead.

As a result, Box Model developed for the BHSS may inherently reflect some suppression of the dose-response relationship between blood lead levels and environmental exposures. Those same factors may be present in the Basin and partially account for the quantitative site-specific and the Box model's performance in describing observed concentrations and percent to exceed critical toxicity levels. There is a possibility that the relationships described in the site-specific quantitative models and the IEUBK Box Model may not apply to future generations and environmental conditions in the Basin. On the other hand, future improvement in the economic situation could mitigate those dose-response factors associated with poverty and the overall dose-response rate for the community could improve. However, particular families that remain poor would continue to be at increased risk. To err on the side of protectiveness, risk managers may want to consider the Box Model as a minimum expression of the dose-response relationship.

Screening for High Blood Lead Levels Intervention Activities. Turnout for the Basin-wide intervention program has been disappointingly low, although not atypical for fixed-site screenings. The primary purpose of the intervention screening is to identify children with high blood lead levels and to provide follow-up services that can help to reduce exposure and consequently, excess absorption. Review of the population statistics suggests that only one-in-four 9 month to 9 year old children is being sampled. The participating population is skewed toward 7-9 year old children, resulting in a substantially lower participation rate among 9-month to 36-month old children. Among the young children (9-month to 36-month olds) participating, 22% have blood lead levels exceeding 10 µg/dl and 10% exceed the 15 µg/dl criteria. As a result, it is likely that the majority of children experiencing excessive blood lead levels are unidentified and receiving only the general education benefits associated with the intervention program and no individualized health response. This finding suggests that increased surveillance and more aggressive intervention efforts may be required in the interim until permanent risk reduction actions can be put in place, and that the efficacy of long-term intervention as a health protective measure is in question.

7.4.2 Uncertainty in Data Collection and Evaluation

Qualitative statements of uncertainty for environmental data gathering within various segments of the Basin for lead and non-lead contaminants include sampling procedures and post-sampling measurement methodologies. The uncertainties associated with blood lead data gathering are summarized in Table 7-4. Three basic questions are addressed with respect to variability and uncertainty with the use of these data in the HHRA:

- Do lead and the non-lead contaminants potentially occur in various environmental media present in areas of the Basin at sufficient concentrations to produce levels of concern for health?

- Are the levels of lead and non-lead contaminants in site media different from background levels? That is, are levels of these substances higher than would be the case in the absence of historical mining, milling and smelting activities?
- Were the sampling plans, in terms of both numbers and types of sampling, adequate to characterize lead and non-lead contaminants in each exposure source and pathway?

Table 7-5 summarizes the elements of uncertainty associated with data gathering and evaluation with reference to environmental sampling and analysis for lead. The Table includes likely direction of bias to the media-specific lead measurements. Comparatively speaking, the level of uncertainty in media sampling for lead concentration or lead loading is arguably less overall than that residing in the parameters associated with lead exposures (exposure factors) and lead risks (measured and/or modeled blood lead levels).

An underlying assumption with environmental lead data collection, an assumption with its own uncertainty, is that the types of environmental media sampled adequately reflect all the media that would be encountered by human populations. This is especially of concern later in the process, during risk characterization, where all media at issue would or would not be entered into the applicable model.

A wealth of data exists to identify what would most certainly be those lead sources and pathways most relevant for the Basin. Expert consensus documents exist to identify and validate these for application to almost all human populations (NRC 1993; CDC 1991; ATSDR 1988, 1992, 1999b; USEPA 1986). Similarly, considerable data exists to specify likely environmental lead inputs to either ad-hoc, statistical models in the form of multi-regression models or mechanistic models such as EPA's Integrated Exposure-Uptake Biokinetic (IEUBK) and Adult Lead (ALM) models (USEPA 1994 a,b; USEPA 1996c).

Less obvious sources of environmental lead that may result in measurable intakes and some associated body lead burden may sometimes be difficult to identify and in theory add an element of uncertainty about all lead sources. In the case of the Basin, it is unlikely that such idiosyncratic lead sources comprise any significant increment of exposure. Of particular concern would be ethnic dietary components and folk medicines that are known to add sizeable amounts of lead to human intake. For example, imported canned foods may still have lead-seamed cans, while some preparations for traditional home treatments for pediatric ailments can contain high lead levels. The Basin population, however, is relatively homogenous demographically, such that few idiosyncratic sources of this type are likely significant.

General Soil and Sediment Sampling Plans

The risk assessment for both lead and non-lead contaminants incorporated only a portion of the many thousands of soil and sediment samples that have been collected throughout the Coeur d'Alene River Basin over the past 25 years. Two criteria for incorporation were that the soil samples had been sieved to a uniform fraction of particle sizes more reflective of likely human contact, and the samples had to

have been collected from sites along and within the Basin where some human contact would occur. This is not to say that exposures of and toxicity to ecological systems and populations would not occur where human contact is unlikely at present or in the future.

A matter of more concern in the above connection is the extent to which current or likely future human contact patterns are adequately addressed within the human residential or recreational areas addressed in this HHRA. This uncertainty cannot be totally resolved as projecting future land use practices involves a complex mix of economic and demographic/sociological factors.

Sampling for Yard Soil Lead in the Basin

Soil lead data available under the above criteria were gathered from the 1996 ATSDR/IDHW survey, two selective repeat screenings in 1999 and various EPA field samplings (notably the FSPA06, -07, and -12 samplings). More than a quarter of all homes in the Basin, and near 50% of those with suspect contamination, were sampled for lead in soil in the course of the above efforts. This amounts to about 1020 homes within both the 1996 and 1999 screenings by the State of Idaho and the FSPA06, -07, and -12 efforts.

How much uncertainty is associated with the merging of the various lead data sets from IDHW's 1996 and 1999 efforts and the EPA samplings? Any answer to that question is tempered by a trade off between the statistical and interpretive advantages gained for the Basin risk assessment through sizeable increases in the sample size through data merger, i.e., reduced uncertainty, versus the consequences of differences encountered in having different soil sampling protocols, ATSDR/IDHW vs. EPA, and differences in recruitment protocols.

Recruitment differed between the 1996 and the later EPA protocols, and some uncertainty as to the consequence of this for soil contaminant assessment exists. For lead and non-lead contaminants, homes in the Basin were self-identified through a voluntary call-in basis in the EPA screenings. For lead and cadmium, the IDHW/ATSDR homes were selected randomly from within the entire population of homes in the Basin. This began with a census of the study area for participants and non-participants. Data collection was preceded by public meetings and availability sessions about three weeks before actual sampling began. Household and individual questionnaires were used for participants.
Sampling protocols of yard soils differed between the State and EPA screenings. Neither technique conducted preliminary discrete core analyses to determine the in-yard variability of soil lead concentration. Because soil lead levels within a yard are likely log normally distributed, the more aliquots that are collected in composite sampling, the higher the probability of including a hot spot, and there are likely more typical aliquots to dilute the hot spot contribution. As a result, the more aliquots collected, the better the representation of true media concentration. It is typically the case that selected compositing protocols await the outcomes of a screening analysis using discrete samples. Both surveys relied on techniques developed at other sites to determine the compositing rate for yard soils.

The IDHW/ATSDR study collected a single composited core sample from a minimum of two and a maximum of 10 cores per yard per 500 ft^2, as being representative spatially of the likelihood of exposure population interactions with yard soils. This technique evolved from comprehensive studies conducted over a number years at the BHSS that has been successfully used to assess both dose-response relationships and characterize yards for remedial purposes.
The EPA surveys relied on techniques used at other mining sites in the western U.S. and sub-composited a number of subareas in each yard but did not collect all samples within sectors as discrete samples for analysis to permit assessment of contaminant level heterogeneity or distributions of contaminants spatially. Because the EPA's approach usually involved more aliquots, it had a higher probability of including a "hot spot" contribution. On balance, some uncertainty persists as to the representativeness of either protocol for a "true" sampling, and additional uncertainty exists as to the potential of the two different protocols to differentially depict an adequate expression of soil lead levels and their distributions within the Basin. This descriptor is uncertain as to how well it captures the full distribution of soil lead values in the Basin.

Analysis of twenty-three yards sampled independently, colleted by both methodologies shown in Appendix N, indicates little significant difference in overall lead levels, and none in cadmium concentrations. The difference noted in lead levels is associated with yards with extremely high concentrations that clearly represent excess risk by any analysis. With regard to the use of these data, both the predictive and empirical analysis relate the results, as obtained, to observed blood lead levels. Both models show a strong relationship that is effective in describing observed levels in those areas where residential soils are the largest contributor to exposure. It is likely, overall, that soil lead level data as merged for this risk assessment adequately represents typical or mean concentrations as used in subsequent analysis. However, these results may underestimate “hot spot” contributions, that may be significant in individual risk evaluations.

There is also uncertainty associated with soil sample preparation, specifically the sieve size used to segregate soil by particle size prior to laboratory analysis. All soil samples collected for health assessment or response actions in the Silver Valley for the last twenty-five years have been sieved to 175 micron particle size. Subsequent studies have shown that this size fraction is a reasonable expression of those particles that adhere to children’s hands and are most active in the hand-to-mouth pathway of soil and dust ingestion. Although this size fraction has been observed to contribute to the soil and dust exposure pathway, the validation for the IEUBK model was done using a 250 micron particle size. Use of the smaller particle size is likely to result in higher lead concentrations relative to the 250 micron particle size, which may result in an overestimation of risk.

According to the Technical Review Workgroup (TRW) for Lead Guidance Document from Short Sheet (http://www.epa.gov/superfund/programs/lead/products/sssiev.pdf) (EPA 2000c):

> Several studies indicate that the particle size fraction of soil and dust that sticks to hands is the fine fraction, and that reasonable high-end for this size fraction is 250 microns (µm) (Kissel et al. 1996a; Sheppard and Evenden 1994; Driver et al. 1989; Duggan and

Inskip 1985; Que Hee et al. 1985; Duggan 1983). This is also the particle size fraction that is most likely to accumulate in the indoor environment, as a result of deposition of wind-blown soil and transport of soil on clothes, shoes, pets, toys, and other objects. Lead concentration data for the fine (250 μm) fraction (Midvale data) were used in the calibration of the EPA Integrated Exposure Uptake Biokinetic (IEUBK Model for Lead in Children, and in the characterization of lead bioavailability in soil, using either *in vivo* or *in vitro* studies (Casteel et al. 1997; Maddaloni et al. 1998; Ruby et al. 1996)

Sampling of Other Soils for Lead

In contrast to yard soils, it is likely that lead in soils and sediments in various common use areas would somewhat overestimate the levels that would be relevant to human exposures. This arises from the highly conservative assumptions (most protective against health hazard) employed for these 49 public areas (Sample N=647).

Five waste piles (Sample N=27) were included in the "neighborhood" soil lead scenario. These waste piles have particularly high levels of lead and can often contain readily ingestible particles that also readily stick to hands. However, the likelihood of direct children's contact in most of these cases is quite low in the case of infants and toddlers, but increases with older, more mobile children. Therefore, sampling and measurements for lead likely overestimate direct contact, and the amount of overall lead contact infants and toddlers will encounter in the Basin. These may or may not overestimate exposures of older, mobile children roaming freely in and around waste piles. To the extent older children transfer into the home smaller, high lead particles on bikes, shoes, clothing, pets, and from waste piles for younger sibling exposures, an indirect added exposure of the siblings is an area of remaining uncertainty.

House Dust Lead

House dust lead characterized as lead concentration or lead loading per unit surface area is typically gathered by various methods, all of which depict different spatial segments of home surfaces and engender sampling uncertainty at several levels. Therefore, one level of uncertainty in this environmental parameter has to do with which type of dust sampling is employed and which approach more accurately expresses the lead content of interior dusts. A second area of uncertainty arises within each sampling method. Basin house dust samples were gathered as either vacuum bag collections (N=320) or by entry dust mats (N=500). The former provided lead as concentration, while the latter permitted quantification in terms of lead concentration or lead loading ($mg/m^2/day$).

Conventional vacuum cleaners collect a broad range of deposited dust particles. This range is further complicated by the state of repair and efficiency of the units and depicts a general but variable expression of interior dust lead level. Uncertainty has to do with how well the range of surfaces vacuumed coincide with the surfaces contacted by children, especially infants and

toddlers exploring their interior home environment. Dust particles largely adhere to children's hands, in contrast to soils where only the smaller fraction of soils are likely to do so. It is likely that some homemakers will vacuum thoroughly, including surfaces not encountered by children. Others would confine vacuuming to the areas in more common usage and that are likely to become more visibly dusty. The former vacuuming pattern may overestimate dust lead concentration with reference to children, while the direction of bias to measurement of more confined areas is not clear.

It is difficult to assess how well the mat dust sampling in this Basin study reflect general deposition of exterior dusts and soils outside of the short collecting protocol of the Basin studies. To the extent that household practice would be to more frequently vacuum entry way mats or carpet than the running collection time of mat loadings in this study, then mat lead measures overestimate actual likely persisting entry mat levels or loadings. To the extent that entry mat surface is largely ignored in vacuuming, then the short collection time in the Basin protocol underestimates likely dust lead content.

Lead-Based Paint

Considerable uncertainty attends the testing of lead-painted surfaces, simply because there are few methods that truly sample the content of lead in paint that infants and toddlers are likely to contact. Compounding the problem for Basin studies is the use of multiple measures for lead paint and the merging of this with expressions of painted surface condition. It is a problem in sampling statistics that the more expressions of concentration or loading for content of a substance, the more likely the finding that the substance has a more robust association with exposure than do those using other media. This is more likely to find statistical artifacts, rather than a true association.

Numerous expressions for lead in paint were employed in lead risk assessment, and these included use of the maximum level in the entire unit and the median level. Collectively, inclusion of these measures would likely overstate the level of lead in paint that is an accurate depiction of what children will encounter.

Groundwater

Groundwater sampling (N=80) was done from 27 monitoring wells sited near Nine Mile and Canyon Creeks. This water is not currently used as public drinking water and would only be an issue for future scenarios in land use where access to such waters might be required. In the latter case, extensive treatment might be required, limiting the extent to which lead levels would reflect human exposure levels. Uncertainty here is only for future likelihood of public consumption.

Surface Water Sampling

Measurement of lead and other contaminants in surface waters entailed a procedure in which sediments were stirred for suspension into the water column and then lead measured for the suspension samples. This would of course have produced a significant overestimation of what lead level would reside in either the water column itself or water with moderate amounts of sediment under less disturbed, non-flooding conditions.

Garden Vegetables

Vegetable sampling for lead is riddled with uncertainties of various types:

- What crops are grown by residents in the Basin and should these be preferentially sampled for lead?
- What is the range of vegetables which could be grown in the Basin, whatever the popularity of current crops, and should all crops be sampled equally for lead?
- In the absence of measurement data, what bioconcentration factor, linking lead in soil to metals in plant, should be used for estimates of plant lead uptake?
- Should results for washed or unwashed samples be used, given that part of garden crop lead content is from incorporated lead and part from external foliar, root or fruit surface contamination?
- Use of wet weight or dry weight concentrations?

Lead measurements in garden samples in the Basin were performed on all produce samples, along with testings of arsenic and cadmium. The sampling was opportunistic as described earlier. Available data for crop lead do not indicate whether actual lead samplings of garden produce would tend to bias the actual levels of lead relevant to human intakes upward or not. As examples, parameters affecting sampling results in terms of lead concentrations include garden size (how to sample areal segments), when during the growing season the samples were taken (net level of lead changes during growing season), rainfall during the growing season which affects growth rate, splash contamination of foliar and fruit surfaces with soil as well as frequency of dust wash-off from plant surfaces prior to testing. See also Section 7.3.

Fish

There a several insufficiencies in the fish tissue data. There is no accurate characterization of tissue levels available for the Coeur d'Alene Lake fishery, although this may be the source of most of the fish consumed in the area. Although the data collected for three species in the lateral lakes may represent that particular fishery, there is little information regarding fish consumption rates among either residents or the Coeur d'Alene Tribe. These data are for filleted fish that likely represent the favored preparation technique for sport fishery families, but may not apply to

Native American subsistence populations. Perch from the lateral lake fish tissue study carry the highest lead load compared to other fish fillet species. Perch are a highly desirable and sought after food fish in this area, and it is not uncommon for subsistence fishermen to take “buckets” of perch home and these small fish may be preserved whole.

One disappointing aspect of the lateral lakes fish sampling effort was the relative lack of larger bullheads and perch in the samples. The majority of the sampled perch were of a small size that would likely be tossed back by many fishermen. Whether this size factor impacted the metal loads in the tissue is unknown, but it is important to note that perch are predatory on other smaller fish so a food chain bio-accumulation might be a consideration. Comprehensive sampling of fish populations, appropriate tissues, harvest and consumption rates by both Tribal and sport fishery families, and comparable background concentrations in areas unaffected by the mining industry will be required to appropriately characterize risks associated with fish in the CDAB.

Water Potatoes

This plant is reportedly dominant in the Tribal subsistence activities and is prepared in a number of ways that can significantly affect lead content. The water potato is more highly coated with sediment because of its habitat, and that this surficial sediment in the Lower Coeur d’Alene contamination can overwhelm the risk analysis. Depending on how fish and water potatoes were (and are) traditionally prepared and cooked (e.g., unpeeled and boiled, etc.) can increase total lead intake rates 50% to 80%. Use of the water potato as a surrogate for all floodplain vegetation consumed in subsistence activities likely overestimates total intake from these sources, as other foods likely have a lower adhered sediment content.

According to tribal representatives, it may be more likely that a modern subsistence family would be eating fish fillets (rather than whole fish), but it is not likely that they would be peeling the water potatoes. This would continue to result in extremely high intake rates. There is data on background lead levels for water potatoes sampled from the St. Joe watershed wetlands at the extreme south end of Lake Coeur d’Alene (Campbell et al. 1999). These results for water potatoes in the St. Joe drainage show no detectable lead levels, compared to lead levels ranging from 0.33 to 127 mg/kg in the Lateral Lakes (wet weight detection limits of 0.04 - 1.8 mg/kg).

7.4.3 Uncertainty in Exposure Assessments

This section addresses lead intakes and associated uncertainties. Questions dealing with biokinetic issues (e.g., uptakes and dependence of uptakes on media lead content) are more appropriately noted in the next section, dealing with lead risk characterization. This division is consistent with EPA's preferred definition of exposure to mean media-specific intakes of lead. Elements of uncertainty for lead exposures in the Basin are summarized in Table 7-6. This portion of uncertainty analysis anticipates the use of exposure factors in either statistical (regression) or biokinetic models.

Intakes of lead are estimated at two rates. The central tendency (CT) intake measurement (for the main body of the population) is employed to derive baseline exposures and incremental exposures. The second uses a reasonable maximum exposure (RME) of intakes as the 95th percentile subset of the exposed populations. These individuals have increased, but not unlikely, incremental exposures compared to the bulk of individuals. CT and RME lead intake values are developed for upland parks, neighborhood stream sediments, public beach sediments, waste pile lead, and garden vegetable and fish consumption.

The principal areas of uncertainty in lead intakes that figure in predictive lead exposure model use for children and other risk populations include: ingested soil and dust, partitioning of intake into direct soil ingestion, and soil-generated dust, dietary lead intakes, inhalation of air lead or reentrained dust, ingestion of lead paint, dietary intakes, and incremental intakes of lead by (mainly) children in the Basin of garden vegetables, waste pile lead, neighborhood recreational and public use area lead intakes.

Uncertainty about intakes of media lead are compounded by the fact that there are demographically different populations: local residents and Coeur d'Alene tribal members. These groups stratify into demographically-defined risk groups based on the potential interactions with contaminated media. This HHRA distinguishes between these two populations through "subsistence" exposure scenarios that are largely theoretical. Neither traditional nor modern "subsistence" exposures to lead are known to be occurring in this area. Establishing the total absence of any subsistence activity with concomitant high lead exposures may be problematic. In any case, the sections below address, where appropriate, tribal vs. non-tribal exposure scenarios.

Soil Lead Intakes

As noted in Chapter 3, soil is the principal medium that Basin dwellers of various demographic type, under various lead exposure scenarios, are likely to encounter. In common with most assessments of mineral industry impacts, the soil exposure pathway is complete for both tribal and local residents, and for children and adults. Soil is also a main source of lead in exterior and home interior dusts. Ingestion of soil is the principal route of exposure, although dusts can be inhaled after reentrainment or in occupational activities.

Numerous variables affect soil lead intake and each has associated uncertainty. These have been described in detail by Mushak (1998) and EPA's IEUBK model user's manual (1994a). Some variables are generic and intrinsic, and some are Basin-specific. For example, the presence of Coeur d'Alene Tribe members with historically distinct practices within the lower Basin produces a set of soil lead exposure scenarios that differ markedly from the typical case for other residents. Other variables include age, the absence or presence of abnormal ingestion rates of soil (in children called pica), the fractional distribution of children's time and activities between

interior and exterior settings, the relative interactions with residential soils versus soils in the neighborhood, at daycare centers, kindergartens, parks, common use areas, and in the case of the lower Basin, beach and waterfront park contacts.

Considerable uncertainty surrounds the various attempts to quantify soil lead intakes, especially for the young child. This document assumes that soil intakes for adults and older children differs markedly relative to young children. At the same time, as noted by Mushak (1998, 1991) there is a relatively wide distribution of soil ingestion rates across groups of children and within a given period of time for an individual child.

Various studies have reported different soil ingestion rates for the infant and toddler. These differ with respect to study design protocol, the selection of which chemical tracers, the time intervals of the study and confounding variables, such as attributing to soil some tracers that co-occur in diet and may overstate the daily soil intake. The data of Calabrese and Stanek (1995) and Stanek and Calabrese (1995) as analyzed by Mushak (1998) indicate that soil ingestion rates are probably more variable across time and children, and within a given child's activities, than previously assumed. Even in the case of the Calabrese testing series, this group's later studies have continually refined their earlier studies with changes in estimates being required. At this time, there are improved data regarding ingestion rates of soil for infants and toddlers but no rigidly defined values, due to the considerable variability that occurs.

The EPA has extensively reviewed the various studies and the associated variability and uncertainty in observed ingestion rates. Overall, the current default selections for age-variable soil intakes for the most vulnerable subsets of children in the total age band 0-84 months in the IEUBK model, a range approximating 85 to 135 mg/day, are reasonable depictions of mean levels. For any adult lead exposure modeling, there is relatively little in the way of empirical data to assess uncertainty.

EPA's IEUBK model entails steady-state, i.e., stable, long-term distributions of lead among body compartments for body lead, derived in turn from stable intakes of lead. Factors that provoke abrupt changes in lead intakes from soils and other media, as might arise from heightened awareness and concerns about lead exposure and hazards to health of children by their parents and other care givers, can produce marked reductions in intake rates of dusts and soils. This can arise by such interventions as more attention paid to children's play behavior, limiting play to relatively clear areas, frequent washing of hands, etc. Such abrupt changes can also affect children's Pb-B levels and do so in a matter of days (Mushak 1998). Such abrupt changes cannot be modeled in a steady-state model, but rather require a physiologically-based pharmacokinetic (PB-PK) model. No such model, however, has been validated at extractive industry Western sites, unlike the extensive validation done for the IEUBK model.

There is some uncertainty in the Basin data about how soil lead ingestion rates for children can be estimated for scenarios beyond that of the residence: to neighborhood recreational soils, soils

in rights of way, beach common use and other soil scenarios. This expanded view of uncertainty differs with the ages of children. Infants and toddlers are assumed to be largely confined to home or sites outside the home equally represented by a residential soil intake scenario. It is for increasingly older children that soils external to the residence are at issue. However, working in opposition to this uncertainty is the fact that soil ingestion rates are comparatively lower in older individuals and are likely within a relatively tighter range.

Dusts and Soil/Dust Ratios

One area of considerable uncertainty deals with the interplay of soil lead ingestion rates and ingestion of soil-derived exterior and interior dusts. Soil lead makes a major contribution to exterior dusts and household dusts through a variety of physical and environmental mechanisms. This is usually addressed through inferential statistical analysis, where the overall level of uncertainty is generally higher than if systematic experimental studies were done. By use of a form of multiple regression analysis, structural equation modeling researchers have been able to show that at many sites including Western extractive industry sites, soil lead contributes to dust lead and these dust lead intakes also produce associations with blood lead. It is now largely accepted that it is interior dust that is the proximate environmental pathway medium for lead exposures of infants and toddlers (Mushak 1998; Succop et al. 1998; USEPA 1996c).

All of the soil ingestion rate studies conducted to date have not experimentally stratified total soil rates into outside soils in the yard, playgrounds, etc., and soil-derived dusts around the entry and in household interiors. These include the studies of Calabrese and coworkers (Calabrese and Stanek 1995; Stanek and Calabrese 1995). These experimental designs were such this was not determinable. Soil-derived interior dusts are not merely interior soils, toxicologically, physically, or biokinetically. Dust particles are much smaller and presumably would be more bioavailable, tend to have higher concentrations of lead per unit mass, and differ on the time scale in terms of their stability as an exposure measure. Unremediated soil lead levels change slowly, while dust lead as an exposure metric can vary greatly depending on such variables as household dust distributions, household cleaning practices, where children encounter dusts, etc.

EPA's IEUBK model attempts to address the inter-relationship of yard/community soils and interior dusts derived therefrom by considering these media as linked via a ratio of 55% dust, 45% soil. The HHRA employed soil/dust lead ratios in both the default 55/45 mode and as a more stratified ratio of 40-30-30 interior dust, yard soil and community soil. This partition was developed through the use of structural equations analysis at the BHSS. However, for the Basin, it is not clear that the latter introduces less uncertainty into the input, despite the fact that this particular partitioning produce a concordance with the observed data similar to that at the BHSS. In common with refining of other input parameters, it is assumed that those ratios of soil and dust that produce closer agreement between predicted and measured blood lead levels are more representative of true child exposures. That may be an erroneous or misleading assumption

where little assessment of the accuracy or reliability of the blood lead data has been done (Mushak 1998).

Food and Water Ingestion

Water lead levels are reasonably well known, given the preponderance of public supplies as water source for most of the Basin residents. Water volume intake rates are also reasonably well known as average values although climate in the Basin would indicate less water intake for much of the year than occurs in hotter climates. While there is some uncertainty about the range of water intakes and water lead levels owing to the presence or absence of lead-containing fittings, joint soldering, etc., overall lead intakes from water are comparatively less significant than those from soils and dusts. That's certainly the case for infants and toddlers, where oral exploratory activity greatly amplifies soil/dust lead intake.

A largely uniform, centralized food supply as any dietary lead source for the bulk of Basin residents constrains the uncertainty to variability in market basket lead testing results. In any case, background dietary lead for the base food supply is relatively low at the present time and relatively wide variability in this measure would contribute limited uncertainty to lead intakes from this pathway.

Garden crop lead intakes can be another matter. Garden crop data were gathered for the Basin since a number of residents had gardens. However, the use of garden crop intake parameters in this HHRA used very conservative EPA guidance values which served to likely overestimate to some extent lead intakes from garden crops. The garden crop lead intake scenario was employed in the HHRA in the context of incremental exposures, i.e., added intakes over the principal, baseline intakes of lead.

Lead intakes by Inhalation

Ambient air lead levels in the Basin at the time of measurements reported in this HHRA were quite low. Combined with ventilation rates appropriate for the different age bands of children, including infants and toddlers, one arrives at relatively low inhaled lead rates. Reentrained dusts in households where infants and toddlers spend most of their time would periodically occur with inside activity and presence of forced-air furnace units. However, this route's net input of lead relative to ingested interior dust lead would be relatively minor, even with considerable variability.

Lead Paint Intakes

Lead paint as a pathway, as is typical in most cases, can only be characterized in qualitative terms. Consequently, the amount of uncertainty and variability associated with lead intakes from this source in young children is principally inferred in comparative terms. That is, SEM or other

regression analysis techniques are generally used to calculate associations with paint exposure blood lead and compared to the relative contribution of soil lead, the latter modeled as working both directly and indirectly through dusts. The relative crudeness of indices for lead intake from paint rule out its ready use for input to any of the biokinetic models estimating blood lead, including the IEUBK model.

Incremental Exposures

The above pathways largely described the baseline lead exposures as defined by applications of the IEUBK model in the modeling subsections of Chapter 6 of the HHRA. Incremental lead intakes from non-residential soils away from the home can significantly add to overall exposure. These incremental exposures are largely assessed indirectly as to the degree of uncertainty in the parameters by observing how such incremental intakes alter agreement between estimated and measured blood lead levels. Again, this assumes that the blood lead data used for comparison are reasonably accurate and reliable.

Coeur d'Alene Tribal Subsistence Scenarios

Lead exposures for tribal members who do not engage in any traditional subsistence activities likely approximate those for the rest of the Basin residents. There is some uncertainty as to how much traditional patterns of interactions with Basin contamination zones exist, do not exist, are strongly discouraged, or are not strongly discouraged. No evidence exists to conclusively show that such traditional or modern tribal subsistence activities do not occur.

Intakes of lead by tribal members within the traditional and modern subsistence exposure scenarios are assumed to be associated with potential future uses, as tribal cultural practices were abandoned in this area a century ago. This means that there are few checks on the extent of uncertainty or variability for the intake factors derived solely for tribal practices within the Basin.

However, there is a good technical grounding to the assumptions that some tribal-specific pathways are huge compared to the remaining residents in the Basin. The intake rates for various media are similar to those described for other tribal scenarios and seem to be reasonable. It cannot be said they are excessively protective absent any current evidence of that.

7.4.4 Uncertainty in Blood Lead Level Modeling

IEUBK Models

Uncertainties associated with blood lead level modeling are summarized in Table 7-7.

Baseline Blood Lead Levels Estimates. The HHRA applied the IEUBK model extensively for two categories of risk: 1) the estimation of blood lead level means and blood lead level distributions for multiple areas within the Basin and within baseline and incremental lead exposure scenarios, and 2) calculations of blood lead level means and distributions at various yard soil cleanup concentrations. All model runs were accomplished using both the community and batch mode of the IEUBK and the EPA Default and Box Models. For reasons discussed below, risk managers should favor the batch mode application in their considerations. There are arguments favoring application of both the EPA Default and Box Models.

In both the EPA Default and Box Model versions, national default recommendations were used for all assumptions for which no site-specific information was available. The development of those assumptions and the associated uncertainties are described in detail in other publications and are not repeated in this discussion. For the EPA Default Model, only site-specific media concentration data were used. All other parameters are those suggested by current guidance.

The Box Model was applied as it was used in the BHSS Five Year Review document (TerraGraphics 2000a) and was not “adjusted”, or calibrated, to fit observed Basin blood lead data. The Box Model was developed through structural equation analysis of the last twelve years of blood lead and environmental exposure data collected through the course of BHSS cleanup. The Box Model does show a better concordance with observed data in the Basin and BHSS areas where residential exposures are dominant. The EPA Default Model significantly over-estimates blood lead levels in those portions of the Basin.

Community versus Batch Mode. All runs were accomplished in both the community and batch mode. The community lead exposure approach, where each of the areas in the Basin was characterized by geometric mean media-specific lead inputs to the model, gives a community-wide depiction of blood lead level mean and distribution. The second characterization of child exposures was the use of batch runs using available lead concentrations for each residential unit's media.

Use of the community mean input approach and subsequent estimation of community blood lead level means and blood lead level distributions is the least computationally and conceptually desirable of the various approaches that can be employed. The community approach subsumes too much uncertainty simply because it attenuates heterogeneity of lead exposures, and understates the most revealing depictions of blood lead distributions. For this reason, the IEUBK model's user manual (USEPA 1994a, b) discourages use of the model at this insensitive, gross level.

The batch run mode, where a blood lead level is calculated for each set of environmental input data for each residence, provides the most sensitive depiction of blood lead level and its distribution in the various communities. This approach also is in accord with user manual

guidance. Risk managers should consider the results of the batch mode runs as more reliable than those produced in the community mode.

Soil and Dust Partition. The Box Model's 40:30:30 house dust, yard soil, community soil partition was derived through structural equation analysis at the BHSS. It differs significantly from the EPA Default assumption in the inclusion of a soil contribution from outside the home yard. Other studies have noted that area-wide and neighborhood soils are significant contributors to blood lead levels in a community setting. At the BHSS, community-wide soils were identified as the greatest contributor to blood lead as a direct source and as a large component of house dust. It is likely that effective remediation efforts will require attention to community-wide soil levels, as was necessary at the BHSS. As a result, the EPA Default Model would not be expected to accurately describe observed blood lead levels, if there was a significant intake from community soils that differed from the yard lead exposure. The EPA Default Model, however, might better reflect the exposures of young children that seldom leave the home environment.

Bioavailability. Two choices of bioavailability were made for the IEUBK runs the default value of 30% and a lower figure of 18%, previously found to give good agreement with blood lead level data at the BHSS. The choice of 18% is potentially problematic, as it was borrowed from the specific set of conditions observed at a potentially different exposure area, even though that area was within the Basin. The 18% figure reflects those variables and conditions inherent in the BHSS screenings, that differ from the screenings accomplished in the Basin outside of the Superfund Site. The 30% bioavailability value is potentially problematic in that several studies at western mining sites suggest this figure may be high. Bioavailability is discussed in more detail in Appendix O and in the 1999 Five Year Review Report (TerraGraphics 2000a) and the 1999 Five Year Review Report Addendum and Extended Response to Technical Comments (TerraGraphics 2001).

It is important to note that the derivation of the 18% bioavailability estimate for the BHSS assumed that there was no suppression of intake rates associated with educational and intervention efforts. All of the reduced dose-response indicated at the site was attributed to reduced bioavailability and not to reduced intake rates. There is evidence at the BHSS to suggest that intervention efforts have contributed to lower blood lead levels, likely through reduced intakes. As a result, the 18% bioavailability should be considered a minimum as it is applied. Similar model results could be obtained from equally credible assumptions of higher bioavailability and lowered ingestion rates. This is particularly important for risk managers to consider when using the model to predict future levels associated with risk reduction efforts.

Changes made to IEUBK input parameters to match observed blood lead levels should be made and interpreted with caution. Comparisons of observed versus blood lead level predicted by the EPA Default Model, show that the BHSS initially over-predicted by a factor of 2 times in 1989, but gradually converged toward default levels over the next 10 years as exposures decreased

(TerraGraphics 2000a). An alternative interpretation of predicted versus observed blood lead trends at the BHSS is that the Box Model is more likely to over-predict blood leads for very high soil concentrations (> 2,000 mg/kg) and is less likely to over-predict blood leads for low soil concentrations (< 1,000 mg/kg) (Hogan et al. 1998). There is concern that applying the Box Model to the Basin may underestimate blood lead levels at lower concentrations. Since 1996, as lead concentrations dropped, the default bioavailability factor has accurately predicted blood lead levels at the BHSS (TerraGraphics 2000a). Conditions at Bunker Hill from 1996-1999 may more closely resemble the Basin than the period prior to 1996. Nevertheless, the Box Model does accurately describe mean blood lead levels and percent to exceed observations in those areas where community-wide residential soil lead concentrations are elevated.

The predictive value of the IEUBK Model depends on the representativeness of the environmental data to actual exposures (Hogan et al., 1998). This explanation relates to the results in the Lower Basin where residential lead levels are generally much lower than lead levels close to the Coeur d'Alene River. To the extent that children are exposed to elevated levels of lead remote from their homes, the Model will under predict their blood lead levels because it is under representing their exposure. Although this phenomenon seems most apparent in the Lower Basin (because homes are relatively cleaner than in other subareas), it may manifest throughout the site in the non-residential, incremental exposure scenarios. It may also be masked in areas where residences are moderately to severely contaminated with lead. If the remedy does not adequately address residential and incremental exposures, then remediated areas may exhibit the pattern apparent in the Lower Basin.

Geometric Standard Deviation (GSD). The GSD for blood lead distributions in human populations theoretically captures inter-individual variability in its physiological, behavioral, biokinetic and, to some extent, even exposure dimensions. A relatively small GSD adjustment can effect a considerable change in the upper tail of the log-normal blood lead distribution. No change in GSD from the EPA Default value was performed in these analyses for either model.

Incremental Blood Lead Levels. For inclusion of the incremental exposures in the IEUBK models, the same ingestion rates and exposure frequency and duration assumptions used for the non-lead metals were applied. As a result, the same uncertainty considerations apply. The most sensitive parameters in predicting blood lead increments among the models are ingestion rates, contact times, and bioavailabiltiy.

The means by which baseline residential scenario blood leads are first estimated and then used in combination with runs for incremental, non residential lead intakes in the IEUBK model runs can drive potential differences in the magnitude of the resulting incremental increase in children's blood lead levels and distributions. These are further dependent on methodology when there is remediation of the residential but not those community lead sources that provide the Pb-B increment. Incremental intakes of lead and any associated Pb-B increases are estimated by

looking at the difference in the estimates for combined residential/baseline plus additional (non-baseline) intakes, minus the residential baseline. However, the pre-remediation incremental amount of Pb-B derived in this way (total intake - background) can be different from, and in fact lower than, the incremental amount that would be estimated post-residential remediation. The overall impact of incremental sources outside the child's home can be demonstrably attenuated, that is, underestimated in the modeling, when one looks only at combined significant intakes before any clean-up actions.

The general biokinetic curve depicting Pb-B versus daily lead intake, or versus such other surrogate independent measures as level of lead in some medium such as soil, is curvilinear downward across a broad intake range (USEPA, 1986a, Ch. 10). As the intake increases, the relative incremental increase across the spectrum in Pb-B for an identical added intake becomes less and less. Because the model is constructed to reflect this curvilinearity, combining any high baseline lead intakes in the residential scenarios with an incremental intake, i.e., adding incremental lead intakes to those already far up the curve of Pb-B vs. Pb intake, would obviously underestimate what that incremental intake might produce in Pb-B if it had actually entered the simulations further down the curve of Pb-B versus total lead intake and in the steeper slope (more rectilinear) portion of the Pb-B vs. intake curve.

What this means, in essence, is that one cannot accurately quantify any residual risk to children after residences but not community lead sources are cleaned up if one estimates contributions of such lead to Pb-B by combining residential and non-residential contributions prior to remediation, especially if residence contamination is already significant. An alternate depiction of that "incremental" contribution to Pb-B and therefore remaining child health risk after residential remediation could be provided by adding that lower range of soil leads likely to encompass any clean-up level for the residence to whatever the incremental lead intakes are.

These comparisons could be depicted using several simple IEUBK runs for children 0-84 months of age. Risk managers may want to consider additional model applications in evaluating appropriate remedial strategies.

Site-specific Regression Models

Multiple regression analyses were run using various environmental inputs and calculating the robustness of statistical associations of each with measured blood lead level. There is uncertainty with how well the robustness of the associations were affected by how the environmental measures were gathered, and the appropriateness of the statistical models used. Interpretation of these results and reliance on the empirical relationships developed for future projections have inherent uncertainties.

For example, soil lead levels for use in either statistical or mechanistic, biokinetic models can have a direct impact on high-risk populations through contacting yard soils, and an indirect effect

through generation of interior house dust levels. Because soil-based interior dust lead is derived from soil, this pathway is not competitive with a direct soil contribution but additive to it, and this requires a model to maximally depict such pathway relationships.

Statistical modeling by ordinary regression analysis was used to assess dose-response and soil to dust slope factors. This type of analysis runs the risk of missing the pathway-contributing variables of interior dust. In regression models developed with blood lead as the dependent variable, dust lead loading rate shows the strongest relationship with blood lead levels followed by independent effects of yard soil and paint lead. The effect of soil likely reflects direct contact with soil lead and such behavioral factors as time spent playing in bare soils, playing in sand boxes, the frequency of hand to mouth activities, etc. The importance of the paint condition variable likely reflects factors of home hygiene and socioeconomic status as well as lead source considerations. The major impact of both yard soil and the paint source variables is likely captured in the dust loading variable, as these are the ultimate sources of the lead.

Those equations in which interior house dust lead concentration is the dependent variable indicates that soil and paint are both independent sources of lead to house dust. The models all show strong statistical significance and are likely reasonable quantitative indicators of the relative significance of these sources. However, extending these models to future predictions of the effect of source reductions through remedial action should be done with care as the slopes developed include factors that may be independent of, or influenced in peculiar ways by, the remedial action.

These various pathway relationships can be quantified using structural equation modeling, a form of multiple regression analysis used at the BHSS (Succop et al. 1998, TerraGraphics 2000a). However, this analysis was not attempted with this data base due to a scarcity of paired blood-soil-dust mat-vacuum bag results. As a result the predictive equations discussed in the next section should be viewed with caution.

Adult Model for Lead

The USEPA guidance requires use of baseline blood lead concentrations that reflect no site-related exposures. The method is primarily used to assess risks from lead contaminated sites for people who do not otherwise have excessive lead exposures. However, some residents in the Basin may also have ongoing exposures to lead elsewhere in the Basin. The use of national baseline blood lead levels may underestimate risk for people with baseline blood lead concentrations that are elevated due to other exposures.

Subsistence Blood Lead Levels

Modeling of subsistence blood lead levels was not accomplished because the intakes estimated for these potential activities would exceed the practical limitations of available models. Human

health risk assessments for lead among Basin residents entailed the use of annual blood lead screenings of children and adults and the use of the IEUBK model for descriptive estimates of blood lead averages and distributions. For tribal members, the model was not used nor are there any available blood lead screening data, notably for tribal children. Nevertheless, the exposures predicted for subsistence activities are great enough that blood lead levels exceeding 30 µg/dl can be expected for both children and adults.

7.4.5 Uncertainty Regarding Candidate Risk Reduction Activities

The risk reduction strategies discussed in Section 6 largely depend on the ability to accurately predict future environmental and blood lead levels based on projected remedial activities. The reliability of these estimates depends on the ability to describe current blood lead and environmental exposure levels, the applicability of those relationships in the future, and the effectiveness of the candidate remedial actions. The uncertainties in many of these considerations have been discussed above. These uncertainties include the representativeness of the observed blood lead surveys, the strength and applicability of the identified dose-response relationships, and the potential impacts of other sources either inadequately described or not quantified.

The overall analysis indicates that excessive levels of absorption are ongoing in both the upper and Lower Basin. Whether or not the available survey results overstate or understate the blood lead levels of non-participants, there is a clear need for risk reduction activities for, at least, those children with high blood lead levels. In the Upper Basin, house dust lead is the most important exposure source to children. House dust lead levels are influenced by soils and lead based paint. Analysis of the available data indicate both sources will require action to achieve acceptable house dust concentrations. Addressing only soil exposure will not resolve excessive intakes in homes with a significant lead paint risk. Conversely, lead paint stabilization will not sufficiently reduce house dust levels, as evidenced by homes with no lead paint problems having high dust lead concentrations.

Uncertainty in Risk Reduction Techniques

Intervention Programs. The first question with regard to the relative uncertainty is the specific type of risk reduction activity. The efficacy of intervention activities and long term reliance on methodologies to effect permanent behavioral changes are most uncertain. Reliance on education, and repeated warnings regarding parental attentiveness, as with personal and home hygiene, may be effective in the short term, but may lose their value as time goes on. The community as a whole has demonstrated little inclination for such programs by the failure to participate in screening surveys. Nearly one-third of children are currently in poverty. The coinciding socioeconomic problems that poor families deal with in raising their children, put a considerable burden on those families lacking in needed resources. These communities plan to revitalize their economies through recruitment of new industry. The continuing implementation of invasive public health intervention activities may deter investment in the area. This, in turn, will

not help to resolve the poverty-related risk co-factors, making exposure more difficult to address.

Access Restrictions and Institutional Controls. Remedial techniques to restrict access to contaminated media vary in uncertainty. Secure well-maintained fencing, physical barricades or protective barriers restricting access to contaminated sites can be successful in eliminating exposure. However, there are incumbent maintenance obligations that must be accepted and carried out and an appropriate enforcement mechanism must be established to ensure compliance. These institutional requirements introduce a level of uncertainty depending on the cooperation of the affected parties. Access restrictions through signage, education and public cooperation to maintain awareness are less certain; the public has indicated some reluctance to accept and maintain signs in CUAs.

Cleanup Actions. Elimination of contaminated media and replacement with suitable clean materials offers the most certainty. However, the question of how clean is clean always presents uncertainty. With regard to risk reduction measures involving cleanup of contaminated environmental media, the greatest unknown in effectively remediating the upper Basin is how much will soil cleanup and paint stabilization reduce house dust lead levels. In the Burke/Nine Mile area, these exposures are exacerbated by children accessing contaminated neighborhood areas and waste piles. There is considerable experience in remediating these types of sites in the BHSS that can be used to reduce uncertainty in their effectiveness.

In the Lower Basin, there is evidence that exposures to sources outside the home environment are contributing to, or are largely responsible for, excess exposure. In the Lower Basin, these exposures will not be significantly reduced by residential cleanup. Protecting homes from flooding and development of clean recreational areas are candidate actions to reduce excessive exposures in the Lower Basin. These remedial activities offer significant engineering challenges and are subject to continual re-contamination by flooding. The uncertainty in these approaches are related to technical feasibility and the public's willingness to use clean areas and avoid unremediated recreational sites.

Uncertainty in Blood Lead Projections

Comparisons between observed and predicted blood lead level must be judged at both portions of the distribution:

1) central tendency (geometric mean)
2) high-end (percentage of children above 10 μg/dl)

The protectiveness of remedial strategies considers both central tendency and high end risk estimates for current and future conditions. Since 1994, USEPA policy is to control risks to individuals from lead associated with discreet residences as well as the aggregate risks to entire

communities (USEPA 1994d, 1998f). A target proportion of 5 percent or less for a typical child exposed to a discreet residence above 10 μg/dl is the basis for determining target cleanup levels on Superfund Sites. Current USEPA Guidance recommends the use of IEUBK Model to best evaluate target cleanup levels for lead (USEPA 1994d, 1998f).

The analyses conducted in this assessment are consistent with that guidance. There is uncertainty, however, in the application of the models. All those factors discussed above should be considered including parameter uncertainty in the measurement of media contaminant levels and calculation of model inputs, in the selection of soil/dust partition and bioavailability, and in the comparisons to observed blood lead levels.

Aside from those many considerations, the brief sensitivity analysis conducted in Section 6 indicates the two most critical elements in projecting post-remedial blood lead levels. Those are 1) the selection of either the EPA Default or Box Model and 2) the projection of post-remedial house dust lead levels.

The analysis conducted in Section 6 suggests that the Box Model may be the more effective predictor of blood lead levels for the Basin. Results in the remedial program at the BHSS are testimony to the potential success of a similar approach in the Basin. However, this model relies on a reduced dose-response relationship developed from site-specific analysis of blood lead and environmental source relationships.

Adjusting the IEUBK Model based on paired environmental and blood data is a hybrid empirical approach to a mechanistic model. Empirical models describe direct and indirect relationships between measures of exposure and blood lead. By definition, empirical models describe observed relationships. They are descriptive of recent past conditions but may not be predictive of changed future conditions. The BHSS bioavailability value may not be reflective of baseline conditions to the extent that the 18% value reflects the sustained and intensive efforts of lead health intervention activities. Although the Default and Box modeling approaches differ, the differences in the predictions from the two models are not large, given uncertainties associated with both models, and it could be readily argued that actual risks fall within the range of predictions from the two models. Use of the Box Model to accurately predict blood lead concentration and select soil action levels in the Basin may be contingent upon a sustained effective Lead Health Intervention Program. The EPA Default Model results offer additional protectiveness, but that could come at a high price with little additional benefit if the Box Model assumptions are accurate.

The dust lead projections are empirically based on the site-specific regression analysis. The uncertainties associated with these models are discussed above. That the modeled relationships are similar to, and that the projections parallel, the results observed at the BHSS provides some comfort. Nevertheless, reducing house dust lead concentration is likely the most critical factor in bringing blood lead absorption to acceptable levels in the upper Basin.

Uncertainty Due to Unaccounted Multiple Exposures

In view of the importance of reducing house dust lead levels in any remedial strategy, it is important to consider other factors that may influence this media. Lead paint abatement, control of fugitive dust sources, stabilization of waste piles or tailings accumulations where tracking may occur, street cleaning and washing, greening programs, cleaning curbs and gutters and installing storm-water infrastructure, and interior cleaning are all measures that could help reduce dustiness and dust lead content.

Several potential exposure pathways were not quantified in the HHRA. These were discussed in previous sections for other metals. Similar considerations apply for lead. Such exposures include consumption of whole fish from Lake Coeur d'Alene, inhalation of fugitive dust, ingestion of waterfowl and big game, consumption of locally raised beef cattle, breast milk and floodplain vegetation used by subsistence populations. Any significant exposure associated with these media, although unlikely, would add to blood lead levels.

Uncertainty in Sub-population Group Protectiveness

Children in the infant/toddler age band clearly have higher mean blood lead levels and probability to exceed health criteria associated with various soil lead cleanup levels. Risk managers may want to consider age-specific responses in evaluating cleanup strategies.

Uncertainty in Future Use Scenarios

Some of the most metals-polluted soils, sediment and vegetation occur in the river floodplain below Cataldo. It is likely that this area will experience significant pressure for land use change in the future, including residential expansion (both year-round and seasonal) similar to that now occurring in the Coeur d'Alene/Spokane area. Although much of the floodplain is presumably unavailable to residential growth because it is protected by government ownership/management, there are thousands of acres of private lands in or near the floodplain where growth could occur.

Additionally, the State and Tribe are in the final stages of approving and building a major recreational trail (the UPRR Right-of-Way) through the heart of the river corridor. There is potential for real or perceived conflict in the eyes of the public associated with warnings and restricted access in some areas and concurrent invitations to the public to use these improvements. The Coeur d'Alene Tribe has indicated a desire for a cleanup strategy sufficiently protective to eventually support subsistence activities. Risk managers may want to consider the possibility of expanded use of this area for recreational, subsistence and residential development in developing cleanup and risk reduction strategies.

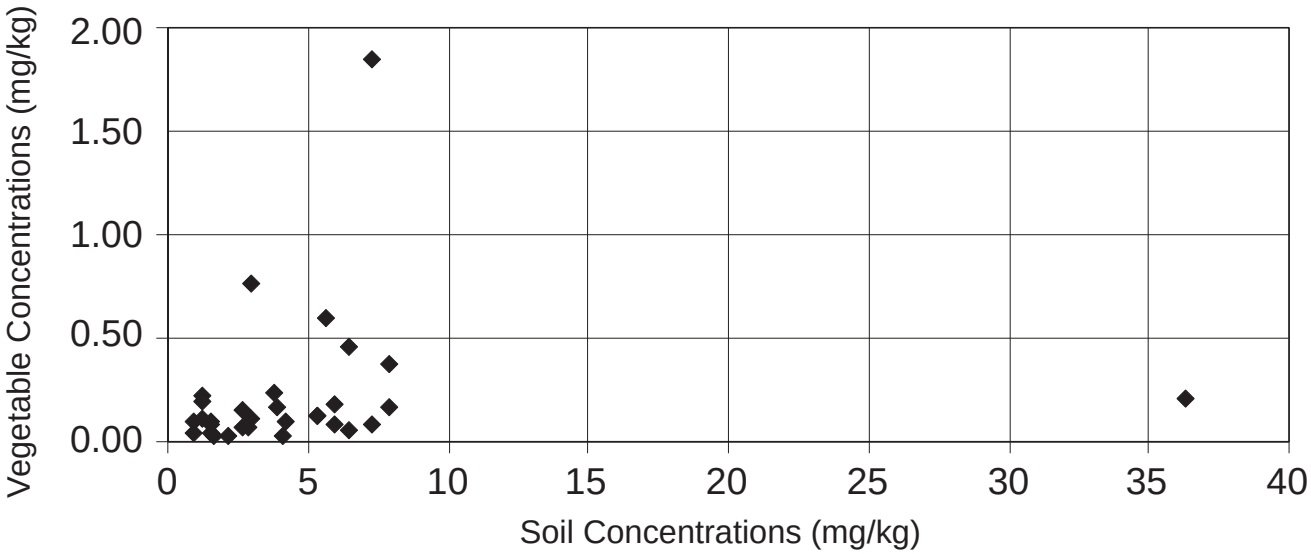
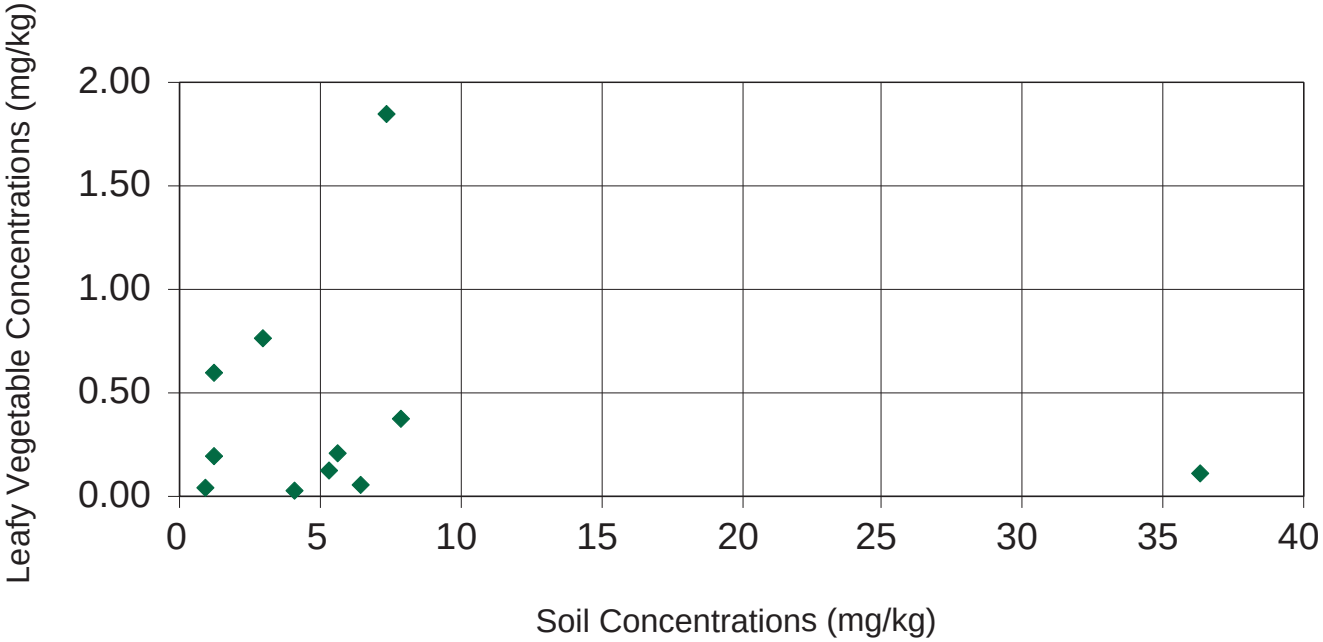


Figure 7-1
Cadmium Concentrations in All Vegetables and Soil



EPA REGION 10	027-RI-CQ-102Q Coeur d'Alene Basin RI/FS DRAFT BASELINE HHRA	Doc. Control: Generation: 1 BHHRA-1a 05/30/00	**Figure 7-2** **Cadmium Concentrations in Leafy Vegetables and Soil**

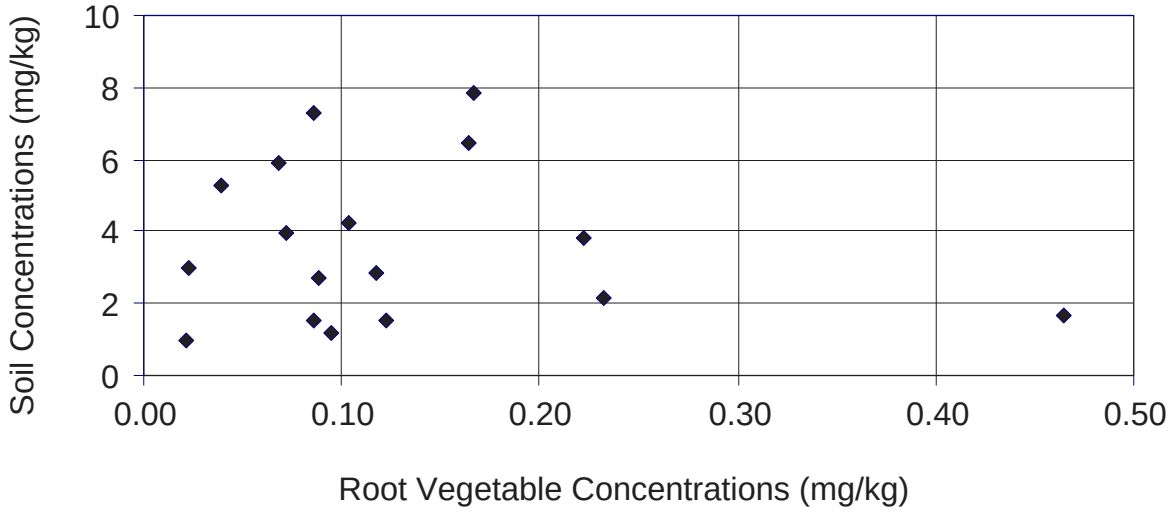


Figure 7-3
Cadmium Concentrations in Root Vegetables and Soil

Table 7-1
Effect of Reduced Dermal Surface Areas (SAs) on Current Total RME Risks and Hazards for the Neighborhood Recreational Scenario

	Curent Total RME (across all media & exposure routes)		Total RME with Reduced SAs (across all media & exposure routes)		Percent Decrease (1-Reduced/Current)	
	Hazard	Risk	Hazard	Risk	Hazard	Risk
Lower Basin	1.1	2.20E-05	1.01	2.0E-05	8%	11%
Kingston (NS Confluence)	1.2	3.20E-05	1.14	2.8E-05	5%	13%
Side Gulches	1.7	3.00E-05	1.66	2.8E-05	3%	8%
MidGradSeg01	0.38	7.20E-06	0.37	6.8E-06	2%	6%
Nine Mile	1.7	5.40E-05	1.64	5.0E-05	3%	8%
Mullan	0.46	6.00E-06	0.45	5.6E-06	3%	7%
Silverton	0.23	5.10E-06	0.22	4.7E-06	4%	7%
Wallace	0.3	5.00E-06	0.29	4.6E-06	2%	8%

* The current RME Skin Surface Areas used in these risk and hazard calculations are 7960, 5080, and 5080 cm^2 for Kingston and Lower Basin Soil/Sediment exposure, all other areas Soil/Sediment exposure, and Upland Soil and Waste Pile exposures, respectively. The reduced RME Skin Surface Areas used in these risk and hazard calculations are 5175, 4240, and 2775 cm^2 for Kingston and Lower Basin Soil/Sediment exposure, all other areas Soil/Sediment exposure, and Upland Soil and Waste Pile exposures, respectively.

Table 7-2
Summary of Geometric Means and Ratios of Chemical Concentrations in House Dust and Yard Soil

Chemical	Geometric Mean (mg/kg)				Ratio		
	Floor Mat Dust	Vacuum Cleaner Bag Dust	Surface Soil[a]	Surface Soil[b]	Mat Dust/SS	Vacuum Dust/SS	Mat Dust/ Vacuum Dust
Antimony	12.41	9.22	6.09	5.73	2.04	1.61	1.35
Arsenic	32.89	20.7	27.27	26.52	1.21	0.78	1.59
Cadmium	8.66	8.19	4.65	4.42	1.87	1.85	1.06
Iron	21,123.41	12,464.93	22,441.75	22,012.91	0.94	0.57	1.69
Manganese	1,040.96	558.76	1,073.99	1,045.1	0.97	0.53	1.86
Zinc	1,390.39	1,076.57	697.33	669.73	1.99	1.61	1.29

[a]Surface soil from yards with data from floor mat samples
[b]Surface soil from yards with data from vacuum cleaner bag samples

Notes:
Every home with a soil sample did not also have a mat sample and/or a vacuum sample.
Soil data were paired with mat data for all homes with mats and paired with vacuum data for all homes with vacuum data. Consequently, the means for surface soil paired with mats and surface soil paired with vacuums are different because the soil sample populations are different.

SS - surface soil

Table 7-3
Cadmium Concentrations in Vegetables and Soil

Sample Type	1983 PHD data, dry weight (mg/kg)[a]	1998 EPA data,[b] dry weight (mg/kg)
Carrots	19 Samples; mean = 4; range = 1 – 11	9 Samples; mean = 1.8; range = 0.3 – 4.7
Beets	7 Samples; mean = 6; range = 2 – 13	1 Sample; concentration = 0.6
Lettuce	10 Samples; mean = 12; range = 4 – 28	6 Samples; mean = 11; range = 3 – 28
Garden soil	20 Samples; mean = 7; range is 3 – 15	31 Samples; mean = 5; range is 1 – 36

[a]These results are from the Pinehurst area only (PHD et al. 1986). Smelterville and the Kellogg-Wardner-Page area were also sampled and generally had higher cadmium concentrations in soil and vegetables than Pinehurst. However, the Pinehurst samples were selected as potentially more representative of current conditions. The other two areas have been the subject of intensive remediation efforts.
[b]Data provided in Appendix F.

Table 7-4
Generic and Basin-Specific Elements of Uncertainty in Blood Lead (Pb-B) Data Gathering

Screening Event	Area of Uncertainty	Level of Uncertainty	Direction of Bias
Typical	Nature of screening: programmatic vs. health intervention, vs. single-shot	Potentially moderate to high absent programmatic structure	Typically lower Pb-Bs owing to attenuating effect of publicity, education, alarm, etc.
IDHW/ATSDR 1996	Reflection of Pb-B statistics in entire Basin	Moderate given level of participation	Potentially an underestimate absent statistical testings of screened vs. non-screened
Later 1997, 1998, 1999	Comparability of study or screening design with 1996 data	Moderate in comparison with 1996 results	Bias indeterminate over all 4 data sets

Table 7-5

Elements of Uncertainty in Environmental Lead Data Gathering and Assessment in The Basin

Pb Medium	Sampling	Main Areas of Uncertainty	Level of Uncertainty/ Bias
Yard soil	Variable across studies: single or subcomposite	Full reflection of high Pb areas and Pb distributions	Moderate/ underestimate
Dust	Personal vacuum cleaner bags or entry mats	Areas of home covered: relative inputs from yard soil	Moderate/ bias indeterminate
Community/other soils	Recreation, beach and waste pile sample testing	Relative contact times for variable age children	Moderate/ overestimate for infants and toddlers, likely overestimate for older children
Lead-based paint	Surface Pb by XRF + condition ranking: multiple measures	Largely qualitative testing; frequency of child contact vs. multiple Pb measures	Moderate/overestimate owing to multiple and/or maximum measures
Groundwater	Monitor well sampling	Uncertainty for eventual use by public	High for future use scenarios
Surface water	Stirred water with sediment suspension	Likely ingestion with sediment at this loading	Moderate/ overestimate
Vegetables	Current garden crop samples: growth period unknown	Sampling across yards and within yards	Moderate to high/ bias indeterminate

Table 7-6
Elements of Uncertainty in Lead Exposure Data in the Basin

Pb Medium	Variable	Areas of Uncertainty	Level of Uncertainty/ Bias
Yard soil ingestion	Soil ingestion rate and at which sites	How much soil ingested daily?	Moderate/ bias indeterminate
Soil-derived dusts	Soil-dust ratios	% of total soil = dust?	For model, moderate/ likely underestimate using "Box" 40:30:30 dust - yard soil - community soil vs. 55% -45% model default
Food	Current diet Pb intake	Centralized vs. local/ ethnic food in diet	Overall, low impact/ bias minimal
Tap water	Daily water volume and total Pb intake	% first-flush vs. full-run samples	Overall impact low/ bias minimal
Inhalation	Ambient air or reentrained dust inhalation	Inside/ outside time ratios, high for dust inhalation	Overall impact low/ bias indeterminate
Lead paint	Multiple measures incl. maximum reading and median of readings + condition ranking	Qualitative, not quantitative assessment; which of multiple measures most valid?	Moderate to high/ analyses favor overestimating
Multimedia incremental Pb intakes	Non-baseline intakes, largely away from home	Amounts of intake for quite different scenarios	Moderate/ overestimate
Multimedia Coeur d'Alene Tribal scenarios	Intakes of Pb media under "subsistence" scenarios using published data	Extent of variance with regular residents theoretical; question of level of use of subsistence practice	Moderate to high, given theoretical scenarios/ bias indeterminate, but high overestimate with practice avoidance

Table 7-7
Elements of Uncertainty in IEUBK Modeling of Blood Lead (Pb-B) Levels in The Basin

Model Parameter	Areas of Uncertainty	Level of Uncertainty	Direction of Bias
Bioavailability, 18% vs. default	1) True extent of bioavailability difference vs. default 2) At 18%, how much lack of agreement is uptake differences vs. intake differences?	Relevance of 18% bioavailability used in "Box" to rest of Basin for various reasons, including child group differences, somewhat questionable	Likely an underestimate using blood lead data as reference for calibration
Soil/Dust mass ratios	True extent of partitioning difference vs. default, if any; limited validating of 40:30:30 ratio selected	Potentially moderate vs. default of 55% dust/ 45% soil	Pb uptake likely an underestimate since soil Pb uptake likely less than dust Pb; if higher soil fraction, lower net uptake
Choice of population testing a) community b) batch run of residential unit media Pb vs. estimated Pb-Bs	a) Predicted Pb-Bs via community mode are uncertain b) Predicted Pb-Bs via batch runs maximizes site-specific data at less crude estimating level, lower uncertainty	Community runs less certain than batch runs	Community runs minimize high Pb areas; bias the overall Pb-B distribution low
Pb cleanup levels vs. child age bands at different soil Pb values	Levels of adequate protection and preservation of "not more than 5% $\geq$ 10 µg/dl" guidance	Protective use of cleanup criteria at EPA's policy of 0-84 mo. age band uncertain for most vulnerable age group: infants, toddlers	Tabulated estimates of exceedances of 10 µg/dl higher for 9-24 mos. vs. 0-84 mos.

Table 7-8

Summary Statistics for Environmental Variables for All Data and the Subset of Data Paired with Blood Lead Observations

Area	Stat	Mat Lead (mg/kg)		Lead Loading (ug/m^2/day)		Soil Lead (mg/kg)		Vacuum Lead (mg/kg)		Interior Mean Paint Lead (mg/cm^2)		Exterior Mean Paint Lead (mg/cm^2)	
		All Homes	All Children	All Homes	All Children	All Homes	All Children	All Homes	All Children	All Homes	All Children	All Homes	All Children
KINGSTON	N	48	14	42	10	99	44	30	25	37	7	40	10
	MIN	63	145	0.06	0.19	22	57	102	102	0.00	0.00	0.00	0.00
	MAX	15500	3505	6.31	3.78	9228	753	1750	1750	0.57	0.06	8.60	1.65
	GEOMEAN	610	660	0.74	0.96	257	207	466	326	0.02	0.02	0.03	0.18
	GSD	2.69	2.96	3.26	3.32	3.34	2.35	2.07	1.81	7.23	5.15	15.65	12.07
LOWER BASIN	N	110	18	109	18	160	38	31	15	104	23	102	22
	MIN	22	55	0.02	0.04	15	15	49	68	0.00	0.00	0.00	0.00
	MAX	4805	4805	29.75	22.52	7350	7350	3140	3140	7.85	0.12	0.93	0.21
	GEOMEAN	318	263	0.48	0.56	110	104	301	221	0.01	0.01	0.04	0.03
	GSD	3.26	3.24	4.41	6.47	4.29	6.04	2.81	3.59	6.28	3.29	5.12	4.99
MULLAN	N	47	10	40	9	105	27	32	14	43	13	43	13
	MIN	278	892	0.43	0.66	40	215	429	557	0.00	0.00	0.00	0.00
	MAX	4460	2800	10.47	4.79	20217	5620	4060	4060	0.72	0.27	2.83	2.83
	GEOMEAN	1242	1301	1.52	1.34	628	930	985	1385	0.03	0.03	0.10	0.04
	GSD	1.78	1.45	2.04	2.13	2.91	2.49	1.70	2.03	6.69	5.58	7.03	11.71
BURKE/ NINE MILE	N	54	33	37	27	88	70	35	33	38	38	39	38
	MIN	173	691	0.30	0.96	32	37	83	83	0.00	0.00	0.00	0.00
	MAX	59498	27601	87.17	45.70	5410	5410	5800	5800	2.14	2.14	4.70	4.70
	GEOMEAN	1781	2044	4.28	6.07	679	628	879	906	0.02	0.04	0.05	0.22
	GSD	2.86	2.60	4.43	3.81	3.25	4.01	2.63	2.72	10.15	11.87	12.92	11.67
OSBURN	N	98	35	73	27	262	95	84	48	81	46	79	45
	MIN	202	517	0.19	0.35	33	76	23	82	0.00	0.00	0.00	0.00
	MAX	42045	6020	66.16	3.91	12883	4251	2192	1340	0.35	0.28	4.28	0.51
	GEOMEAN	882	990	0.88	1.06	419	532	493	328	0.02	0.02	0.06	0.05
	GSD	1.94	1.81	2.49	1.91	2.45	2.34	2.17	2.26	6.38	6.51	8.87	6.45
SIDE GULCHES	N	53	19	47	16	100	45	26	14	52	28	53	28
	MIN	167	281	0.17	0.17	25	31	116	162	0.00	0.00	0.00	0.00
	MAX	8840	2103	21.37	5.73	3356	1200	3929	1646	0.34	0.25	1.67	1.67
	GEOMEAN	842	651	1.13	1.18	368	197	695	493	0.02	0.03	0.04	0.03
	GSD	2.11	1.78	2.55	4.06	2.38	2.65	2.21	1.78	7.83	7.67	7.19	8.08
SILVERTON	N	22	28	19	27	70	69	26	37	23	35	24	35
	MIN	326	374	0.28	0.42	94	94	75	75	0.00	0.00	0.00	0.00
	MAX	3658	1458	9.45	2.69	6098	1690	3390	3390	0.57	0.28	1.83	1.58
	GEOMEAN	863	859	1.10	1.21	352	356	557	660	0.05	0.11	0.08	0.16
	GSD	1.93	1.58	2.42	1.95	2.25	2.24	2.52	2.23	6.56	3.76	8.24	4.75
WALLACE	N	42	12	33	6	110	56	35	19	37	26	37	26
	MIN	604	716	0.35	1.17	54	65	259	681	0.00	0.01	0.01	0.01
	MAX	47624	3440	158.27	4.78	16027	3020	29724	3300	1.23	1.20	9.90	3.40
	GEOMEAN	1774	1404	2.63	2.31	771	866	1004	1059	0.08	0.08	0.22	0.12
	GSD	2.54	1.69	3.14	1.60	2.47	2.10	2.33	1.48	5.26	3.64	7.89	4.20

This table was originally created in M://Basin/Basin00/Comments/Table 1.xls

8.0 SUMMARY AND CONCLUSIONS

8.1 PURPOSE

The Coeur d'Alene Basin (CDAB) in northern Idaho includes Lake Coeur d'Alene and the St. Joe and Coeur d'Alene River drainages that are the ancestral home of the Coeur d'Alene Indian Tribe. Since the late 19th century, this area has been the center of one of the most productive mining districts in the world. Significant deposits of gold, silver, lead, zinc and associated metals have been mined and refined in the upper Basin for over a century. The area is known as Idaho's Silver Valley. During most of the last century, substantial quantities of industrial wastes were directly discharged to the environment from mining, mineral processing, and smelting activities, as was common practice at the time. Public health investigations in the 1970s to 1980s resulted in the designation, in 1983, of a 21 square mile area called the Bunker Hill Superfund Site (BHSS), or "the Box," surrounding the former smelter complex near Kellogg. Remedial activities and public health response activities have been ongoing in the BHSS for two decades.

A Remedial Investigation and Feasibility Study (RI/FS) is currently being undertaken to characterize the degree and extent of the contaminant release in the remainder of the CDAB. Concurrent with the RI/FS, this baseline Human Health Risk Assessment (HHRA) addresses potential health risks associated with residual heavy metals contamination in the CDAB for areas east of Harrison upstream from the mouth of the Coeur d'Alene River. A screening level HHRA was previously conducted for Coeur d'Alene Lake beach areas, and a similar screening level HHRA is being conducted for the Spokane River that drains Lake Coeur d'Alene into the State of Washington.

The baseline risk assessment is an evaluation of the potential threats to public health from site contaminants in the absence of any remedial action. The primary tasks accomplished in performing the HHRA included data collection, data evaluation, exposure assessment, toxicity assessment, and risk characterization. The purpose of this HHRA is to determine the extent of heavy metal contamination in environmental media that may expose current or future residents or visitors to the CDAB, to evaluate the potential human health risks associated with exposure to those contaminated media, and to provide information for risk managers to evaluate the need for remedial action and development of associated clean-up criteria.

Observed blood lead levels among area children collected by the Panhandle Health District (PHD) are considered in the HHRA. These data have been used to evaluate potential and active pathways, the degree and extent of excess absorption among the population, and the relationship between blood lead levels and environmental exposures. The participants in the PHD surveys were voluntary respondents to Basin-wide solicitations by the health department and it is not known if these individuals are representative of the overall population. To the extent that risk assessment or site-specific analyses rely, directly or indirectly, on observed blood lead data; baseline conditions reflect the ongoing PHD public health intervention efforts.

8.2 STUDY AREA

Figure 8-1 shows various features of the CDAB. The CDAB is located in the Panhandle region of northern Idaho and lies within Kootenai, Shoshone, and Benewah Counties. The Basin is on the west slope of the Bitterroot Mountain Range. Summers in the area are generally hot and dry with only about 12% of the annual precipitation occurring between July and September. Approximately 50% of the annual precipitation occurs between November and February. Winter temperatures are 15 to 25 degrees higher than those in continental locations of similar latitude. These weather patterns make the Basin one of the highest precipitation areas of the upper Columbia River Basin and result in the potential for frequent high water events. The remaining precipitation takes place in the spring.

Much of the area is rural and contains a wide variety of landscape types, rich in natural resources including floodplains, steep mountain canyons, and river valleys. Topography and landscape vary in the Basin from relatively open, flat floodplain areas of the Coeur d'Alene River in the western portion of the Basin to steep, narrow canyons to the east. The floor of the valley near the boundary between Kootenai and Shoshone Counties is roughly 1 mile wide and narrows significantly eastward toward Shoshone County. Valley areas near Wallace average 0.25 mile wide.

For the purposes of this HHRA, the study area is from the Idaho-Montana border in the east to Harrison in the west. The 21 square-mile BHSS is excluded from this assessment. Some additional areas, such as regions south of Harrison, Blackwell Island, and Corbin Park beaches, have been identified by the State, EPA, and the Coeur d'Alene Tribe, and are also included as part of this HHRA. Some subareas addressed in this HHRA are discussed as being located in the upper Basin, that is contained in the steep mountain canyon of the South Fork and adjacent tributary gulches east, or upstream, of the BHSS. The upper Basin contains 11 residential cities or unincorporated areas, about half of which are located within the BHSS. Most of the mines and industrial facilities that constitute the Coeur d'Alene mining district are, or were, located in the upper Basin.

Immediately west of the BHSS is the Kingston subarea that includes Kingston, Pine Creek, and the confluence of the North and South Forks of the Coeur d'Alene River. The Lower Basin area west of Cataldo includes 11 lateral chain lakes and extensive wetlands, located adjacent to the main channel and within the Coeur d'Alene River floodplain. These marshes and lakes provide an extensive recreational area between the town of Cataldo and Lake Coeur d'Alene. Camping, fishing, boating, swimming, hunting, and wildlife photography and observation are popular activities throughout the lower CDAB. There are no incorporated cities between Cataldo and Harrison at the mouth of the main Coeur d'Alene River. However, there are a few small unincorporated village areas and several rural residences.

8.3 RESIDENT POPULATION, LAND-USE, ECONOMY AND HOUSING

Much of the Basin is rural, undeveloped land. Approximately 32 percent of Kootenai County and 75 percent of Shoshone County consist of federally managed lands, primarily National Forests. These areas are rich in natural resources including forests, wildlife, and a number of tributaries and streams that support a variety of aquatic organisms. However, many of these areas are inaccessible due to the lack of roads, difficult terrain, or lack of services. Interstate 90 (I-90) provides limited access to the otherwise rural area.

Tourism related to the use of these natural resource areas for recreational purposes has increased significantly over the last two decades and is one of the fastest growing contributors to the local economy. Recreational use of the abundant natural resource areas include riding off-road vehicles, snowmobiling, berry picking, mountain biking, fishing and floating the Coeur d'Alene River, and cross-country and downhill skiing.

Approximately 10,500 people, or 1% of the total population of Idaho, reside within the study area. The economy of the region, traditionally based on mining, has declined over the last 10 to 20 years due to mine closures, layoffs, and a lack of other industry to replace the mineral-based economy. Between 1980 and 1996, total mining employment decreased by 74%. As a result, the total population shows a declining trend as people move outside of the area seeking jobs.

The population of Shoshone County decreased by 29% between 1970 and 1990. Between 1990 and 1998, the population remained relatively unchanged, with a slight decreasing trend (0.4%). Correspondingly, the unemployment rate increased from 6.7% to 9.9%. Between 1990 and 1996, total employment increased slightly (4%), while mining employment continued to decrease significantly (58%). Tourism and recreation appear to be growth sectors, replacing some of the mining jobs. Unemployment showed a slight increase (0.3%) from 1990 to 1998.

As the younger generation is forced to move outside of the area to find employment, the population of the Shoshone County is becoming older. The median age of residents in Shoshone County in 1970 was 27.3 years as compared to 39.6 in 1998. The percent of the population aged 65 and over in Shoshone County in 1997 was 15.7% compared to 7.1% in 1970. Between 1994 and 1998, the child population under age 18 in Shoshone County showed a decrease of 6%, while the total population remained fairly constant. Statewide, the child population showed a slight increase (3%) between 1994 and 1998, with a total population increase of 8%.

From 1990 to 1998, the number of children under the age of 5 years in Shoshone County decreased by 12.1%. Statewide, the number of children under age 5 increased by 12.2%. The number of children between the ages of 5 and 17 in Shoshone County decreased by 7.7%, while statewide, the number increased by 14%. Overall, the total number of children under 18 in Shoshone County decreased by 8.7% and increased statewide by 13.5%.

Socio-economic data for children living in Shoshone County showed higher than statewide percentages of child poverty, single parent families, infant mortality, low birth weight babies, school dropouts, teen births, and teen violent deaths for all years included. As an example, the percentage of children in poverty in Shoshone County increased from 23.7% to 31.2% from 1990 to 1996, while the percentage of children in poverty statewide remained relatively constant at approximately 16% to 17%. Births paid for by medicaid decreased in Shoshone County from
1997 to 1998 (55% to 42%); however, the percentage remained higher than statewide numbers of 33% and 28% for 1997 and 1998, respectively.

The total number of housing units in the Basin Area is 5651, or 1.4% of the total number of housing units in Idaho. The percentage of occupied housing units in the Basin Area (74%) is lower than the statewide percentage (87%) due to vacancies and a high number of seasonal units in some Lower Basin census blocks. The percentage of renter occupied units is lower in the Basin Area than statewide at 23% and 30%, respectively. The statewide average is likely influenced by a higher number of renters in urban areas.

Housing units in the Basin Area are typically older than those reported statewide. Forty-eight percent of the housing units in the Basin Area were built before 1960, and over half (60%) of those were built before 1940. Statewide, only 37% were built before 1960, and less than half of those (44%) were built before 1940. Since 1980, the percentage of houses built in the Basin Area has also been lower than statewide, at 12% and 18%, respectively. From 1990 to 1997 housing growth in Shoshone County was 5.6%, well below the statewide growth rate of 21.6%.

Basin Area housing values were typically lower than the State median with fourteen of the eighteen census block groups included in the Basin Area (78%) having median housing values less than the statewide median of $58,000. The four block groups with median values greater than $58,000 are located within the Kingston and Lower Basin subareas. The majority of median rent values in the Basin Areas are also lower than the State median rent value of $330. Of the eighteen block groups in the Basin Area, only one (located in the Kingston Study Area) has a higher median rent value.

8.4 DATA USED IN THE HHRA

In addition to traditional geographic, climatic, and demographic information, two basic data sources were used in the HHRA. Those data either i) originated in investigations associated with the RI/FS or the Natural Resource Damage Assessment (NRDA) being conducted under CERCLA by federal and Tribal trustees, or ii) were obtained in health surveys conducted by the State Department of Health and Welfare and allied local and federal health agencies. The principal source of the latter data was a comprehensive blood lead and environmental exposure study conducted in 1996, and follow-up blood lead surveys conducted in 1997-1999.

In 1996, the State of Idaho, the PHD, and the Agency for Toxic Substances and Disease Registry (ATSDR) conducted a large-scale, multimedia exposure study within the Basin. The investigation characterized both environmental contamination and biological indices of human exposure from 843 residential homes in the upper and Lower Basin. The data obtained included blood lead, urine cadmium, yard soil, house mat dust, home vacuum dust, lead-based paint measurements, and tap water. All samples collected were originally analyzed for lead and cadmium.

The 1996 study was followed by fixed-site blood lead surveys during the following three summers. Testing during all four summers produced a total of 524 children in the nine month through nine year old category and 667 adult blood lead observations (Adult blood leads were only collected during 1996). In addition, public health investigations were conducted at the homes of 50 children exhibiting blood lead levels greater than or equal to 10 μg/dl. In July of 1999, a strategy was adopted to augment the existing database with new information sufficient to support site-specific analysis and provide the risk assessment effort with appropriate information to characterize lead exposure in the Basin. This effort involved extensive development of the available paint lead and housing database that was not utilized in previous investigations, and targeted sampling of additional homes and potential exposure locations.

Those public areas, communities, and specific media for which little data were available were sampled in the summer of 1999 by the State of Idaho. A supplemental survey was also conducted by the State of Idaho in November of 1999 that collected environmental samples and survey data from the homes of those children providing blood lead results that had not previously been sampled. Of the 132 homes that were not included in previous efforts, approximately 90 of those homes were sampled in the Fall 1999 survey.

The combined sampling effort from the IDHW study, all EPA residential data, and additional residential data collected by the State of Idaho in the Summer and Fall of 1999 totals 1020 homes, and these are included in the lead risk assessment section.

Non-lead risk assessment data were largely obtained from the federal and Tribal investigations conducted in the course of the RI/FS and associated NRDA. Numerous samples of soil, house dust, tap water, groundwater, homegrown vegetables, sediment, surface water, fish, and plants (i.e., water potatoes) were collected in the CDAB. Because of the large quantity of analytical information available, the data were organized by medium and geographical area; and the methods used for sample analysis and data quality were evaluated. From this, a baseline data set to support the non-lead portions of the HHRA was developed.

Initially, all the available sampling data from the site was reviewed to identify chemicals that might contribute to risk based on concentration and toxicity. Much of the analytical data from these samples were applicable to human exposures. Others were not, primarily because of the sampling location (not a location people frequent) or the sampling methodology. The analytical data selected for use in the HHRA included:

- 524 blood lead observations from 424 individual children from 260 homes collected over four years,
- Yard soil from 191 homes for non-lead metals, 994 homes for lead,
- House dust from 83 homes for non-lead metals, 299 vacuum bag samples and 474 dust mat samples for lead,
- Groundwater from shallow wells in Burke/Nine Mile and Canyon Creeks (for a future scenario, this groundwater is not currently being used as a drinking water source),
- Tap water from 100 homes for non-lead metals, 398 homes for lead,
- Soil from 13 upland parks and schools located in the towns of Silverton and Wallace,
- Soil from five mining waste piles, two near Canyon Creek, two near Nine Mile Creek, and one near Mullan,
- Soil and sediment from 33 beach areas along the lower Coeur d'Alene River, and one beach area in Coeur d'Alene Lake (Blackwell Island),
- Surface water collected adjacent to 33 beach areas along the lower Coeur d'Alene River, and one beach area adjacent to Coeur d'Alene Lake (Blackwell Island),
- Sediment and surface water from Canyon Creek, Nine Mile Creek, Moon Creek, Big Creek, Beaver Creek, and Pine Creek,
- Fish fillet tissue from pike, perch, and bullhead (312 total samples from Medicine, Killarney, and Thompson Lakes) and whole fish tissue data from the Spokane River,
- Produce from 24 residential vegetable gardens, and
- XRF paint lead observations from 417 homes.

8.5 CHEMICALS OF POTENTIAL CONCERN

Chemicals of potential concern (COPCs) were identified using a decision process that included a comparison of detected chemical concentrations with screening values (SV). Additional analysis of a subset of the soil data for other metals (e.g., arsenic, mercury, and zinc) was completed for approximately 80 homes, and these were included in the risk calculations for the non-lead metals in addition to the EPA residential data.

Table 8-1 summarizes the COPCs for each media evaluated. For the solid media soil, sediment and house dust, seven metals including antimony, arsenic, cadmium, iron, lead, manganese and zinc were selected as COPCs. Only lead was selected as a COPC in air.

In water, five metals including arsenic, cadmium, lead, manganese, and mercury were selected as COPCs in surface water for both "disturbed" and "undisturbed" conditions. Five metals were selected as COPCs for groundwater including antimony, arsenic, cadmium, lead, and zinc. Only arsenic and lead were selected for tap water.

For dietary routes, all chemicals analyzed in fish including cadmium, lead, and mercury were considered COPCs. The COPCs selected for homegrown vegetables and water potatoes were arsenic, cadmium, and lead.

8.6 TOXICITY ASSESSMENT

A toxicity assessment was developed for each of the COPCs. The toxicity assessment identifies the adverse health effects associated with excess exposure to each metal. In particular, the relationship between the dose of a chemical and the occurrence of toxic effects is evaluated. Toxicity criteria for chemicals (that identify acceptable levels of contaminants) consider both cancer effects and adverse health effects other than cancer (noncancer effects). Generally, the acceptable levels of contaminant intake are based on reference doses related to specific effects for non-carcinogens and slope factors that estimate the potential incidence of cancers associated with the absorbed dose of the chemical.

The toxicity assessment for lead is based on its potential to cause neurocognitive and neurobehavioral effects in children. The toxicity criteria for lead is related to blood lead levels associated with these effects. The current level of concern is 10 μg/dl of lead in whole blood and is of greatest concern for children and pregnant women (as they represent the developing fetus). Arsenic, the primary COPC after lead, is assessed for its potential to cause skin cancer, frequently fatal cancers of the internal organs (e.g., bladder, kidney, lung, and liver), and various pre-cancer and noncancer effects in skin by ingestion. Arsenic was the only COPC evaluated for cancer effects. The other metals examined have various adverse health effects. Table 8-2 shows the principal health effects for each COPC.

8.7 EXPOSURE SUBAREAS

For the purposes of the ecological risk assessment and the RI/FS, the Coeur d'Alene Basin has been divided into exposure areas based on watersheds and drainage patterns. However, not all portions of the Basin are of concern with respect to human health considerations, and potential human exposures in a number of areas cross watershed boundaries. As a result, the Basin was divided into eight HHRA geographical subareas based on existing communities, identified routes of potential human exposure, public use patterns, and the results of environmental lead health surveys in each area. Those geographic subareas shown on Figure 8-1 are:

- Lower Basin (the floodplain of the lower Coeur d'Alene River from Harrison to, and including, Cataldo),
- Kingston (the area of the Basin between the 21-square-mile Bunker Hill Superfund Site and Cataldo; specifically, the town of Kingston, the confluence of the North Fork Coeur d'Alene River (North Fork) and the South Fork Coeur d'Alene River (South Fork), and residences near Pine Creek, but outside the Bunker Hill Superfund Site),

- Side Gulches (including residences in the side canyons along streams draining into the South Fork between the Bunker Hill Superfund Site and Mullan, with the exception of Nine Mile and Canyon Creeks),
- Osburn,
- Silverton,
- Wallace,
- Burke/Nine Mile (including Nine Mile Creek and Canyon Creek), and
- Mullan.

8.8 POPULATIONS OF POTENTIAL CONCERN

Certain population groups in the Basin could be more sensitive to contamination, or more likely to be subjected to greater exposure than the typical individual in each of the receptor groups. These populations include *infants, children* and *pregnant women* as they represent the *fetus*, and individuals with *subsistence lifestyles*, including some members of the Coeur d'Alene Tribe.

Because of their physical vulnerability and small body size, *infants and children* are often more susceptible to the potential toxic effects of chemicals in the environment. Their risks may differ qualitatively and quantitatively from those of adults for a variety of reasons including differences in behavior (e.g., frequent hand-to-mouth behavior), physiology, metabolism, pharmacokinetics, diet, and exposure environment.

Physiological differences include intake rates of air, food, and water (and associated chemicals) per unit of body weight. Similarly, dermal, intestinal, and respiratory absorption may be greater or lesser in children depending on the chemical and the exposure scenario. There are also major metabolic differences between children and adults that can significantly affect their ability to respond to chemical exposure. Pharmacokinetics, including the absorption, distribution, and excretion of various chemicals, differs between children and adults on a chemical-specific basis.

The diet of a child is often quite different from that of adults. Dietary differences, such as the amount of vegetables, fruit, fish, or red meat consumed, can have an effect on the amount of chemical ingested in food items. In addition, nutritional status has a profound effect on toxicity response. There are obvious differences between adults and children in the physical environment and living habits. For example, children are generally closer to the floor, carpet, and ground. Their daily activities, hand-to-mouth behavior, and lack of occupational exposure significantly influence the amount of soil and dust consumed, and chemical exposure that occurs. Although children may not have occupational exposures, they are susceptible to secondary exposures from take-home dusts brought in by a parent, other caregiver, or family member encountering contaminated media in the Basin through various routes of contact at work, e.g., working in local construction.

As a result, infants and children often receive a different effective dose of a chemical than adults, even when chemical concentrations in affected media are the same. Several poverty-related factors among the resident population cause additional concern for infants and children in this population. The high incidence of teenage pregnancies, infant mortality, low birth weights, and single parent families suggest possible nutritional deficiencies. Vitamin and essential nutrient deficits can contribute to higher metal absorption rates, particularly for lead as the body seeks calcium, and exacerbate adverse health effects for the fetus and infant.

Lower socio-economic status indicated by the 31% of children living in poverty, births paid for by medicaid, school lunch programs, high welfare payments, low-rents, and high unemployment rates are associated with greater risk of lead poisoning. The substantial decrease in young children in the population indicates young families are continuing to leave the area. Increase in welfare payments to the remaining homes with children may indicate the area is attracting and retaining economically disadvantaged families.

Poverty and lead poisoning interact in several ways. Children may have lowered nutritional status and live in poorer quality housing. Parents may experience more difficulties in managing the home and children, and are less able to provide a stimulating and healthy home environment. Home and child hygiene and behavioral risk co-factors can lead to increased ingestion rates of soils and dusts. Yard soils and house dust can be more contaminated due to deteriorating lead paint, proximity to industrial sources, and lesser quality maintenance of the home, yard, and local infrastructure. The age of housing in the Basin is problematic due to the frequent use of lead paint and accumulation of contaminated dusts throughout the last century.

As a result, poor children ingest more soil and dust that has a higher lead content. These children tend to absorb more of the ingested lead than those with a more nutritionally sound diet, resulting in higher blood lead levels. In addition, poor children are more vulnerable to adverse health effects resulting from their lower general health status, and reduced access to quality health care and early childhood educational opportunities. The increased risk of lead poisoning for children in lower socio-economic groups does not imply that other children in the Basin from areas with higher rent and housing values are not at-risk. Poor children are at relatively higher risk than those from more affluent families.

A second population of concern are fetuses, by virtue of maternal exposures to lead and certain non-lead contaminants such as arsenic. Lead crosses the human placental barrier and can expose fetal tissues at the most vulnerable periods of development. Another population of concern are elderly residents whose long-term exposures to lead may result in risk of hypertension or bone demineralizing disorders later in life that might release historically-accumulated bone lead to the blood stream.

Effective dose and routes of exposure can also differ markedly for those practicing *subsistence lifestyles*. The resident riparian lifestyle and harvest techniques employed throughout Coeur d'Alene tribal history represent holistic practices that encompass all activities in an overall lifestyle. Fully

addressing potential Native American exposures within the Basin requires consideration of routes of exposure not included in other scenarios in the HHRA. The tribal riparian lifestyle has the potential for significant prolonged exposures to both sediment and water and significant dietary intake. Examples are fishing, consumption of whole fish, and the harvest of the water potato (Sagittaria spp.) at the mouth of the Coeur d'Alene River. Vegetable consumption rates for the Tribe show a strong dependence on the water potato and, traditionally, as much as one-third of the overall diet was resident fish.

The traditional economy of the Coeur d'Alene Tribe was characterized by a complex and highly structured system of food source production, distribution, and consumption. The Plateau peoples generally practiced a seasonally based cycle of utilization of specific economic resources. This travel involves the return annually to well known camps for root digging, fishing, hunting, and high elevation hunting and berry picking.

The Tribe was largely dependent upon Lake Coeur d'Alene and its tributaries; perhaps more than any other Plateau group. Water played a central role in all aspects of life, from birth to death and was included in all major cultural events. Individuals spent a great portion of their time in the water; generally through fishing, hunting, gathering, bathing, recreating, and other various activities. The basic winter village in the Basin was the center of the cycle and was never fully abandoned by certain individuals of the society, especially the elderly and children too young to travel on their own but too heavy to be carried. The Coeur d'Alene were primarily involved in harvesting, consuming, and utilizing riparian resources. Much of the raw material used in the manufacture of various necessary items was obtained from within the riparian environment.

All of these activities were undertaken collectively in family or tribal groups and involve children and women of reproductive age, that are considered the population at greatest risk. These activities also result in substantially greater potential exposures associated with consumption rates of resident fish and riparian vegetation, and soil and sediment contact rates associated with typical residence and harvest practices for both ingestion and dermal routes.

Due to the Tribe's dependence on water from Lake Coeur d'Alene, the surrounding lateral lakes, the Coeur d'Alene River, and close interaction with the natural environment, maximum exposures were assumed. Additionally, it is not known how a subsistence diet affects metal absorption rates in the body. Nutritional factors, such as calcium or trace metal deficiencies, and periodic fasting associated with cultural activities or availability of foodstuff could lead to enhanced absorption rates, especially for growing infants and children, pregnant women and fetuses.

8.9 RECEPTORS, EXPOSURE PATHWAYS AND SELECTED SCENARIOS

In order for an adverse health effect to occur, a person must be exposed to the chemical of concern. Residents and visitors to the Basin could be exposed to affected media during their normal daily activities including home life, recreation, and work. However, across the Basin, exposures will not be

the same because of differences in activity patterns, e.g., people do not all use the same places for recreation and do not all eat homegrown vegetables. The amount of the chemical (the dose) a person encounters depends on the concentration of the chemical in the medium, the frequency of use of a particular area or medium, the length of time the person is exposed, the person's age, size, and intake of the medium.

Five major population groups were quantitatively evaluated in the risk assessment for a variety of exposure pathways. Inherent in these major groups are various pathways of exposure. For example, day care, school, deposition of wind-blown dusts and tracking of soils into the home are inclusive in the residential scenario. Different exposure pathways were also evaluated depending on the geographical area where they are encountered. For example, no waste pile exposures were evaluated in the Lower Basin because there are no waste piles present. The receptors and exposure pathways that were evaluated fall into one of five exposure scenarios.

The ***residential scenario*** pertains to children and adults who live in the Basin and could be exposed to affected media inside the home, in the yard, and the local community. Estimates of metals intake were developed for incidental ingestion of soil and house dust, dermal contact with soil, ingestion of drinking water, and ingestion of homegrown vegetables. A future drinking water scenario using shallow groundwater collected from metal source areas in Canyon Creek and Nine Mile Creek was also evaluated for hypothetical residents. Shallow groundwater is not widely used as a drinking water source for Basin residents.

The ***neighborhood recreational scenario*** pertains to children of ages 4 through 11 who would play in their neighborhood in or near creeks and on waste piles and for whom these exposures would be in addition to the residential scenario. In general, the areas evaluated for neighborhood exposures are undeveloped properties immediately adjacent to residences. Neighborhood recreational scenarios were quantitatively evaluated for exposures from incidental ingestion of sediments, dermal exposure to sediments, incidental ingestion of surface water during water play activities, and exposures to waste pile soils.

The ***public recreational scenario*** pertains to children and adults who use developed parks and playgrounds, and undeveloped recreation areas, whether they are local residents or visitors from outside the area. Public recreational exposures were quantified separately from residential and neighborhood recreational exposures because of the potential for cross-Basin travel and the possibility that visitors from outside the Basin could use the public areas. Public recreational scenarios were quantitatively evaluated for exposures from incidental ingestion of surface soil and sediments, dermal exposure to soils and sediments, incidental ingestion of surface water during water play activities, and ingestion of fish.

The ***occupational scenario*** pertains to adults who could come into contact with affected media in the course of their daily work activities. In general, work exposures would be less than residential and

recreational exposures because of more limited contact with the affected media. However, workers who have intensive contact with soils such as during construction activities, might have high exposures for short periods, depending on the work location. As a result, construction workers exposed to soils were quantitatively evaluated in the risk assessment.

The ***subsistence scenario*** pertains to children and adults engaged in traditional (aboriginal) or modern subsistence lifestyles in the floodplain of the lower Coeur d'Alene River. These are future scenarios, as subsistence lifestyles are not known to be currently practiced in the floodplain. Exposure pathways quantified for subsistence lifestyles are similar to those evaluated for residential and recreational receptors.

Typical (CT) and Reasonable Maximum Exposures (RME)

All scenarios were evaluated at two levels of probable contaminant intake. Those are called the typical, or Central Tendency (CT), and the Reasonable Maximum Exposures (RME) intake rates. The CT estimate is the most likely or typical amount of contaminant a member of the population will intake for each scenario. The RME represents the largest intake that can reasonably be expected for any individual member of the population. All risk calculations are performed at both levels of potential intake so that risk managers may consider the potential effects for both the bulk of the population and for those individuals most at risk. Generally, the CT estimate is for the 50th percentile of the population and the RME is calculated at the 95th percentile. The results can generally be interpreted to mean that the CT estimate applies to at least half of the population and the RME applies to the 5% of the population most exposed.

8.10 HUMAN HEALTH RISK CHARACTERIZATION FOR METALS OTHER THAN LEAD

8.10.1 Non-carcinogenic Risk

The exposure factors, media concentrations, and toxicity criteria are combined for the non-lead metals to calculate health risks. Health risks for chemicals that cause cancer are calculated differently from health risks for chemicals that cause noncancer health effects. For each non-carcinogenic chemical, there is a "threshold" dose. If a person is exposed to a chemical dose equal to or less than the threshold, no adverse health effects are expected. The chemical dose from the site is divided by the threshold dose to arrive at a "hazard quotient." If the hazard quotient is equal to or less than 1, no adverse health effects are anticipated. Hazard quotients greater than 1 may be associated with an adverse health effect. Noncancer health effects present age-specific concerns because young children are often more sensitive than adults. As a result, noncancer health risks were calculated separately for young children in addition to children and adults combined.

Summary hazard results for non-carcinogenic effects are provided in Tables 8-3 through 8-6 and are discussed by Exposure Scenario below. Risks and hazards for the traditional subsistence scenario were the highest of any receptor population. Modern and traditional subsistence exposures were evaluated only as future scenarios because subsistence lifestyles are not known to be currently practiced in the floodplain. For both exposure scenarios, hazard quotients were greater than 1 for each age group, with hazards for the traditional scenario being at least three times higher than hazards for the modern exposure scenario (Table 8-6). Risks and hazards for the modern subsistence scenario were similar to those for the highest residential areas.

For both the modern and traditional subsistence scenarios, arsenic and iron in soil and sediment were the greatest contributors to noncancer hazards. Hazards from fish ingestion are likely underestimated for subsistence exposures because the whole fish is consumed. Hazards are estimated using data on fish fillets, that have substantially lower metals concentrations (e.g., an order of magnitude) than whole fish.

For typical (CT) exposures to the resident population, potentially unacceptable hazards occur only for future child/adult residents of the Burke/Nine Mile area, if they were to use groundwater as a domestic supply. Hazards when all exposure routes are combined were equal to one (i.e., at the acceptable limit) for the resident children in the Lower Basin, the Side Gulches, Osburn, Burke/Nine Mile, and Mullan. In general, the hazards and risks calculated for typical (CT) exposures were lower by approximately an order of magnitude compared to those calculated for RME conditions. All other excess hazard quotients discussed are for RME conditions.

For the resident population, soil ingestion pathways contributed the most to risk and hazard totals, with soil ingestion generally contributing more than 90% of the noncancer risk. Hazards from arsenic contributed 30% to 60% of the total hazard for residents, and iron was the second largest contributor, accounting for 20% to 40% of the total residential hazard. However, in most cases the hazard due to iron did not exceed the target hazard quotient of 1.

In addition to arsenic, other chemicals exceeded a hazard quotient of 1 for a few age groups, pathways, and areas under RME conditions. Exposures to other chemicals with hazard quotients greater than 1 were the following:

- Cadmium hazards to residents from eating homegrown vegetables, and to traditional subsistence receptors from eating water potatoes,
- Iron hazards from ingesting soils and sediment in the Lower Basin (0- to 6-year age group for residential receptors and all ages for subsistence scenarios),
- For the hypothetical future scenario that includes drinking shallow groundwater in the Burke/Nine Mile area, cadmium and zinc hazards from drinking groundwater (0- to 6-year age group and 0- to 30-year age group), and

- For the modern subsistence scenario, mercury in fish and for the traditional subsistence scenario, mercury in fish, and manganese and iron in soils and sediments.

8.10.2 Arsenic Carcinogenic Risks

Cancer risks are calculated under the assumption that no level of the chemical is without some risk. Risk indices are presented as a probability of developing cancer, e.g., an increased risk of developing cancer of 1 person in 1,000,000 (a 1×10^{-6} cancer risk level). The U.S. Environmental Protection Agency (EPA) uses the general 10^{-4} to 10^{-6} risk range as a "target range" within which the Agency strives to manage risks as part of a Superfund cleanup. Once a decision has been made to take an action, the Agency has expressed a preference for cleanups achieving the more protective end of the range (i.e., 10^{-6}), although waste management strategies achieving reductions anywhere within the risk range may be deemed acceptable. Furthermore, the upper boundary of the risk range is not a discrete line at 1×10^{-4}, although EPA generally uses 1×10^{-4} in making risk management decisions. A specific estimate around 10^{-4} may be considered acceptable if justified based on site-specific conditions, including any remaining uncertainties on the nature and extent of contamination and associated risks. As a result, in certain cases EPA may consider risk estimates slightly greater than 1×10^{-4} to be protective (U.S. EPA 1991d).

For carcinogens, the greatest health concerns are (chronic) doses over the entire lifetime and cancer risks are calculated for children and adults combined, assuming exposure over a lifetime. Arsenic was the only carcinogen evaluated and the only chemical other than lead evaluated for the drinking water pathway (other chemicals and pathways were screened out because they did not pose a health risk).

The highest cancer risks are associated with subsistence lifestyles. RME cancer risks exceeded 10^{-6} in all exposure pathways, with cancer risks ranging from approximately 1×10^{-5} to 1×10^{-3}. Table 8-6 shows the RME cancer risk for arsenic for the traditional and modern subsistence exposure scenarios for the combined adult/child age group. Total RME cancer risk is approximately 3×10^{-3} for the traditional scenario, and 7×10^{-4} for the modern scenario. This suggests unacceptable cancer risks from exposure to arsenic through all media and pathways.

For the resident population, cancer risks were evaluated for two age groups: child/adult, age 0 to 30, and occupational adult, 25 years of exposure. As shown in Table 8-3, total RME cancer risk for each scenario was in the range of 10^{-6} to 10^{-4}, except for the residential scenario at the Side Gulches where the RME cancer risk was 3×10^{-4}. CT cancer risk for each scenario ranged from 10^{-6} to 10^{-4}.

For the residential scenarios, exposure to arsenic in yard surface soil contributed most of the total RME cancer risk. Arsenic in tap water also contributed significantly to total RME cancer risk for residents at the Side Gulches. Although tap water was not the primary contributor to cancer risk for the residential scenarios, RME cancer risk for tap water exceeded 10^{-6} in all exposure areas.

Arsenic risks in surface/subsurface soil for construction workers ranged from 2×10^{-5} to 8×10^{-5}. For recreational scenarios in each exposure area, the following media contributed to most or all of RME cancer risk due to arsenic:

- Soil/sediment in the lower Coeur d'Alene River for the Lower Basin (highest concentrations of arsenic in the entire Basin with the exception of waste piles),
- Soil/sediment at the North and South Fork confluence in Kingston,
- Upland surface soil from the Elk Creek area and sediment from Elk Creek Pond in the Side Gulches (Elk Creek area soil and sediment had the second highest arsenic concentrations in the entire Basin after floodplain soil/sediments in the Lower Basin),
- Sediment in the South Fork (Osburn, Wallace, and Silverton neighborhood exposures),
- Surface soil from waste piles in Burke/Nine Mile,
- Soil in waste piles and sediment in the South Fork in Mullan, and
- Soil/sediment from the Spokane River on Blackwell Island.

Cancer risks were calculated on the basis of total arsenic concentrations in each area. However, some of the arsenic is naturally present (pre-mining background concentration) and may be contributing significantly to the total arsenic concentration in soil and sediment. Risk management activities typically take background concentrations into account for decisions regarding remediation. As a result, background may account for a percentage of the risk due to arsenic in some areas and may affect remedial decisions.

8.10.3 Non-lead RME Residential and Neighborhood Risks and Hazards

Under current conditions, the Side Gulches had the highest risks and hazards for the 0- to 6-year age group and the combined children and adults age group (Table 8-3). The Lower Basin had the second highest risks and hazards for these age groups. The Lower Basin had the highest concentrations of arsenic and iron in soil and sediment (except for waste piles). The higher risks and hazards in the Side Gulches were due to high concentrations of arsenic in water in one private well. The Burke/Nine Mile area had the highest neighborhood risks and hazards because of the waste pile exposures evaluated for this area. Waste piles had the highest concentrations of non-lead metals.

Some additional hazards over target health goals for the 30-year period evaluated for child and adult residents exist for elementary-aged school children play in mining-affected media in their neighborhoods, particularly in the Side Gulches and Burke/Nine Mile areas. Additional risks and hazards for residents from arsenic and cadmium in vegetables are also a potential concern.

The hazard quotients in Table 8-3 represent the sum of hazards from all chemicals, and the majority of the hazards are due to arsenic and iron (60% to 100%). There is no evidence that the toxic effects of these two chemicals are additive: the noncancer hazard for arsenic is based on adverse effects on the skin, while the hazard for iron is based on adverse effects on the blood-forming system. For example,

the hazard quotient of 4 for the Lower Basin comprises an arsenic hazard quotient of 1.6 an iron hazard quotient of 1.6, and a hazard quotient of 0.6 for the other metals of concern.

8.10.4 Non-lead RME Public Recreational Risks and Hazards

Of the 8 geographic subareas evaluated for recreational exposure, five had public recreational areas with sampling results. Hazard Quotients from the use of these areas exceeded 1 for the 0- to 6-year age group only along the lower Coeur d'Alene River from the confluence of the North Fork and the South Fork downstream to Harrison (Table 8-4). Cancer risks were highest for this area as well.

8.10.5 Non-lead RME Occupational Risks and Hazards (Construction Worker)

Of the 8 geographic subareas, five were evaluated for risks and hazards to construction workers actively engaged in work that involves soil disturbance. As with the other populations evaluated, risks and hazards were highest in the Lower Basin, and the Lower Basin is the only area where the hazard quotient exceeded 1, with a hazard quotient of 0.5 for arsenic and 0.7 for iron (Table 8-5).

8.10.6 Non-lead RME Modern and Traditional Subsistence Exposure Scenarios (Tribal Members)

Risks and hazards for the traditional subsistence scenario were the highest of any receptor population. Cancer risks for both the modern and traditional exposure scenarios were greater than 10^{-6}. Total RME hazard indices for noncancer effects were greater than 1 for each age group in both the modern and the traditional subsistence exposure scenario with the child in the traditional scenario having the greatest hazard quotient of 43. The total noncancer hazards for the adult/child and adult age groups for the traditional subsistence scenario were 10 and 19, respectively.

- For subsistence children, exposure to metals through all exposure pathways, except the ingestion of disturbed surface water, represents potentially unacceptable risk for noncancer health effects. Ingestion of surface soil and ingestion of sediment contribute most to the total RME hazard index for the traditional subsistence exposure scenario with hazard indices of 17 and 11, respectively.
- For the combined subsistence adult/child age group, the total hazard index exceeded 1 for each exposure pathway except dermal absorption from surface soil and ingestion of disturbed surface water. Ingestion of water potatoes, ingestion of surface soil, and ingestion of undisturbed surface water are the greatest risk drivers for this age group. The key metals contributing to the total RME hazard are arsenic, cadmium and iron.

- Ingestion of fish was the only pathway evaluated for the subsistence adult age group. The total hazard index for fish ingestion exceeded 1, with mercury in fish being the most significant risk driver.

The hazards from eating fish are underestimated for subsistence populations because tissue concentration estimates are based on concentrations in fish fillets. Some tribal members eat the whole fish, not just the muscle tissue, and concentrations of metals in whole fish are greater than those in fillets. In addition, fish fillet data are from the lateral lakes, not Lake Coeur d'Alene. Sufficient fish tissue data were not available from Lake Coeur d'Alene to characterize health risks; however, tribal populations do eat fish from the lake. As a result, tribal health hazards due to fish consumption from Lake Coeur d'Alene are unknown.

8.10.7 Risks and Hazards for Combined Non-lead Exposures

Risks and hazards were not added across exposure scenarios because residential exposures assume people spend most of their time in the home environment. However, for example, if resident children were to play on a waste pile, eat homegrown vegetables, and recreate in the Lower Basin, their risks may be higher than those for residential children who spend the majority of their time at home. In contrast, if people spend significant amounts of time in areas with metal concentrations that are lower than those in their homes, overall risks would be lower.

The hazard quotients and risk estimates developed for non-lead metals should be considered as potentially underestimating noncancer risks for these populations due to additional exposures to lead. Lead is known to have adverse effects to many of the same organ systems of concern in the development of the hazard indices. Potential lead effects are not accounted for in these risk estimates, although substantial lead intake rates are anticipated for these populations. Lead risk assessment is addressed by a separate methodology below.

8.10.8 Potential Preliminary Remediation Goals for Arsenic

Health-based preliminary remediation goals (PRGs) can provide targets for developing and evaluating response action alternatives and are considered with other U.S. EPA guidelines when evaluating remediation options. PRGs are calculated by defining a target risk goal and then solving the basic risk equations for concentrations rather than solving for risk. Target risk goals and equations differ for carcinogenic and noncarcinogenic health effects. The target goal selected for noncancer hazards is a hazard index of 1, which is the point at which the estimated dose from the site equals the reference dose associated with no adverse health effects. As noted in Section 5.2, the highest target goal for carcinogens would generally be a cancer risk of 1×10^{-4} (increased risk of cancer of 1 in 10,000); however, target goals of 1×10^{-5} and 1×10^{-6} are sometimes considered if site-specific conditions warrant lower risk goals.

Arsenic is the COPC that was most consistently a risk driver for all non-lead risk assessment scenarios and soil was the largest contributor to risks. Thus, potential PRGs were calculated for exposure to arsenic in soil by ingestion and dermal exposure for residential, public, and neighborhood receptors. Residential children, aged 0 - 6 years have the lowest non-cancer PRG of 35 mg/kg. For residential children and adults combined, a cancer PRG with a target risk goal of 1×10^{-4} is 64 mg/kg, and the non-cancer PRG for children and adults combined is 123 mg/kg.

When arsenic concentrations from 242 homes (current number of homes that have been sampled for arsenic) are compared to PRGs of 35 mg/kg, 64 mg/kg, 100 mg/kg, and 123 mg/kg, concentrations in 31% of the sampled homes exceed the lowest PRG of 35 mg/kg. In contrast, 14% of sampled Basin homes exceed 64 mg/kg, 9% exceed 100 mg/kg, and only 6% exceed 123 mg/kg.

8.11 HUMAN HEALTH RISK CHARACTERIZATION FOR LEAD

8.11.1 Observed Blood Lead Levels

Lead health surveys conducted by State and local public health authorities note excessive levels of lead absorption in children throughout the Basin. Little problem is noted among adults, particularly in women of reproductive age, although specific data are not available for pregnant women. The risks associated with blood lead levels are characterized by comparison to current Centers for Disease Control (CDC) criteria: excessive prevalence of blood lead levels in the 10 µg/dl -14 µg/dl range are indicative of excess exposure in a community (Class IIA); levels of 15-19 µg/dl are indicative of excessive lead absorption and require education and nutritional intervention and more frequent screening (Class IIB). Levels of 20-44 µg/dl require medical and environmental intervention and perhaps chelation (Class III). Levels of 45 and higher (45-69) require environmental and medical intervention with chelation therapy (Class IV). Children with blood lead levels at or above 70 µg/dl require hospitalization and chelation therapy, along with immediate environmental management (Class V). Critical incidence criteria correspond to current Public Health Service recommendations of no more than 5% of children exceeding the 10 µg/dl level with no child greater than 15 µg/dl.

Figures 8-2 and 8-3 summarize observed blood lead data for children in the Basin combined for the years 1996 to 1999. The highest toxicity rates among nine month to nine year old children are observed in Burke/Nine Mile at 21% exceeding 10 µg/dl, 13% exceeding 15 µg/dl, and 4% with levels of 20 µg/dl or greater. The Lower Basin/Cataldo subarea showed the next highest toxicity rate with 18% exceeding 10 µg/dl and 5% greater than the 15 µg/dl criteria. No children were in the 20 µg/dl range in the Lower Basin. Wallace, Mullan and Silverton, respectively, showed 13%, 11%, and 8% of children with levels of 10 µg/dl, or greater. From 4% to 5% of children tested in Wallace and Silverton exhibited blood lead levels exceeding the 15 µg/dl criteria and 1% exceeded 20 µg/dl. Osburn and the Side Gulches area showed 4% of children exceeding 10 µg/dl and only one child in four years exceeded 15 µg/dl. Kingston showed 11% greater than or equal to 10 µg/dl and 7% exceeded the 15 µg/dl criteria.

The highest blood lead levels are observed in the youngest age groups. One and two year old children have arithmetic mean blood lead levels of 7.0 µg/dl and 8.0 µg/dl , respectively, and geometric mean concentrations of 6.2 µg/dl to 6.3 µg/dl. Geometric mean levels then decrease with age from 5.2 µg/dl at age 3 to 3.0 µg/dl at age 8 (Figure 8-4).

The percent of children to exceed critical toxicity levels differs markedly with age. In the lowest age groups, 9 months to 3 years, 19% to 26% of children Basin-wide exceed 10 µg/dl. The rate is highest in 2 year old children with 17% of this group exceeding 15 µg/dl. For four year old children, 12% exceed 10 µg/dl and 5% exceed 15 µg/dl. In older children, the percent to exceed 10 µg/dl ranges from 5% to 8%, and 1% to 3% exceed 15 µg/dl. Figures 8-4 and 8-5 summarize these results.

8.11.2 Representativeness of the Surveys

Approximately 25% of eligible children participated in the surveys. Participation was lowest among younger children. There are divergent opinions as to how well the health surveys represent non-participants from throughout the Basin. Selection bias may have occurred related to individual family decisions to participate and current representativeness is unknown. One argument suggests that the incidence of lead poisoning is likely greater among non-participants, as families that did have their children tested are more attentive to lead poisoning and have benefitted from the local health department's efforts to assist parents in reducing exposures. A counter argument suggests that paying each child $40 as an incentive in 1999 favored low-income participation. Because potentially high exposures are associated with poverty-related factors, this argument contends higher than average blood lead concentrations would be expected among the participants. There is also concern that younger children were under-represented in the surveys. Because young children typically have higher blood lead levels, overall population means and percent to exceed critical toxicity levels may be biased low. This could affect comparisons of model predictions to observed blood lead levels.

8.11.3 Follow-up of Children with High Blood Lead Levels

Follow-up investigations were completed by the local health department for 50 of 58 children whose blood lead levels exceeded 10 µg/dl. Twenty-five investigations involving 21 individual children were conducted for observed blood lead levels exceeding 15 µg/dl. Risk profiles indicate excess absorption associated with high soil and dust concentrations at homes in the Burke/Nine Mile subarea. Older children's risk profiles in this area also indicate recreational exposures in neighborhood areas contaminated by tailings. High blood lead levels in Wallace are indicated for younger children and are possibly associated with paint and remodeling problems, high soil lead levels in play areas, and dusty or difficult to clean homes. Both Mullan and Osburn had no children greater than the 15 µg/dl blood lead criteria and children's blood lead levels in the 10 µg/dl to 14 µg/dl range were associated with high residential soil and dust concentrations or play in contaminated areas. West of the BHSS, excess absorption was associated with either homes that had been flooded and were contaminated with

sediment and flood debris; or with extended recreational activities in the river or lateral lakes areas of the Lower Basin.

8.11.4 Site-specific Analysis of Paired Blood and Environmental Lead Data.

Site-specific quantitative analysis of the relationship between blood lead levels and environmental variables indicate that contaminated soils, house dust, and lead based paint are all related to excess absorption. The overall results suggest complex exposure pathways, with blood lead levels most related to dust lead loading in the home, followed by independent effects of yard soil lead, interior paint lead condition, and exterior paint lead content. The dust lead pathway is most influenced by outdoor soils, augmented by paint contributions in older homes, especially those in poor condition. The overall effect is exacerbated by dusty conditions in Burke/Nine Mile and to a lesser extent in Wallace. The Lower Basin is a notable exception. High blood lead levels are observed, although little problem is indicated with respect to dustiness or house dust lead concentrations in the Lower Basin. High blood lead levels in the Lower Basin have been associated with homes that were flooded in 1996 and recreational activities outside the home environment.

Quantitative models relating blood lead levels to soil, house dust, and paint lead levels and house dust levels to soil and paint sources were developed. These were used to quantify baseline exposures and project risk reductions that might be achieved through source modifications.

8.11.5 Biokinetic Predictions of Resident Children's Blood Lead Levels

The IEUBK model is used to estimate the average blood lead level expected for a typical child ingesting lead through soil, house dust, paint, and water, and also estimates the percentage of children predicted to exceed certain blood lead levels. Residential baseline (everyday home life) blood lead predictions were estimated using four different applications of the IEUBK Model. These applications included both the community and batch mode versions of the IEUBK. Both the EPA Default Model (using national assumptions for soil and dust ingestion rates and bioavailability) and the Box Model, derived specifically for the BHSS, were employed for each mode. The Box Model uses a lower bioavailability estimate and includes a community-wide component for soil/dust exposure that is not included in the EPA Default Model.

The community mode input includes the geometric mean media lead concentrations for all homes in a community and calculates an estimated mean blood lead level and percent of children to exceed 10 µg/dl. The batch mode was applied to only those homes for which an observed blood lead level was available. Similar results were obtained for both the community and batch mode applications. The EPA Default and Box Model versions, however, provided significantly different results. Batch mode results

were selected for evaluation and discussion as these results are more sensitive to variation in environmental exposure and can be directly compared to observed blood lead levels.

The EPA Default version of the IEUBK Model Batch Mode application predicts a greater than 5% exceedance of the 10 µg/dl health criteria, ***associated with baseline residential exposures***, for all geographic areas. The Box Model predicts exceedance greater than 5% for Mullan, Burke/Nine Mile, Wallace, Silverton, and the Lower Basin. The areas adjoining the BHSS including Kingston, Osburn, and the Side Gulches are projected at less than 5% exceedance for baseline residential exposures by the Box Model. Figures 8-6 and 8-7 show observed and predicted blood lead levels and percent of children to exceed 10 µg/dl for both the EPA Default and Box Models using the batch mode. The results suggest that there are potentially three different exposure situations ongoing in the Basin with respect to the residential soil and dust lead.

East of (and including) Wallace, the baseline Box Model is a better predictor of observed mean blood lead levels. In these areas, the EPA Default baseline model significantly over-predicts both observed concentrations and the percent of children to experience excess absorption. Both models predict more than 5% of 0-84 month old children will exceed the 10 µg/dl criteria in Mullan, Wallace, and Burke/Nine Mile. The EPA Default Model predicts 44% to 48% exceedance in these areas, and the Box Model predicts 19% to 20% above the criteria. Observed exceedance in these areas ranged from 13% to 22%.

Immediately east of the BHSS in Osburn, the Side Gulches, and Silverton, the baseline Box Model fairly-well describes both observed mean blood lead levels and the percent of children exceeding the health criteria. Observed exceedance of the 10 µg/dl criteria for 0-84 month old children ranged from 0% to 11% in this reach. The EPA Default Model predicts 17% to 26% exceedance ***associated with baseline residential exposures*** for these areas, as opposed to the Box Model 5% to 8% projection.

West of the BHSS, and particularly in the Lower Basin, the Box Model is ineffective in describing observed absorption, under-predicting both mean blood lead levels and percent exceedance. Both the EPA Default and Box Models failed to predict these high blood lead levels. The EPA Default Model fairly-well describes mean blood lead levels, but fails to capture the percent of children to exceed health criteria. The Batch mode estimates for Kingston (17% observed greater than 10 µg/dl) were 11% and 2%, respectively, for the EPA Default and Box models. For the Lower Basin (32% observed greater than 10 µg/dl), the respective batch mode predictions were 21% and 14%. This suggests that significant Lower Basin exposures may be occurring outside the immediate home environment.

There are several possible factors that could contribute to the difference in exposures and blood lead levels among these areas of the Basin. There could be physical and chemical differences in the soil and dust contaminants. Differences in chemical form, particle size and matrix effects could result in different physical accessability and bioavailability to children. These differences could be attributable to the

original source of the lead from mine, mill or smelter wastes, or from the degree of weathering and secondary mineralization that has occurred while in the environment.

The degree of dustiness and snow cover in these communities could be a factor, as the larger communities have curbs and gutters and other infrastructure that is not available in the smaller villages. The size of yards, use of lead paint, age of the communities and proximity to industrial or transportation sources could all impact this relationship. The habits and behavior of children, particularly as they move about neighborhoods and select favorite play areas and activities may present important differences in exposures between the larger cities, small residential areas or rural homes.

8.11.6 Lead Health Risks from Exposures Outside the Residential Environment

Lead exposures from sources or activities outside the home environment were evaluated by adding *incremental intake rates associated with the other exposure scenarios to the residential estimates*. Potentially significant recreational exposures are noted for children engaged in certain activities in particular areas of the Basin. Upland park type recreation can result in significant exposures in the more contaminated areas of the Upper Basin and throughout the floodplain areas west of the BHSS. Potential recreational exposures in the Lower Basin are more significant because of both higher soil concentrations and lower baseline residential exposures. This can result in higher dose response rates to incremental exposures at lower blood lead levels. This is a possible explanation for the higher than predicted blood lead levels observed among Lower Basin children.

Additionally, swimming and water sport activities that could result in ingestion of disturbed sediment-laden surface water can result in substantial increases in intake and lead absorption. Potential exposures to neighborhood stream sediments in Burke/Nine Mile, and at public swimming areas in the Side Gulches and the Lower Basin are of particular concern.

Potentially significant increases in blood lead levels could also result from consumption of home grown vegetables. Increased intake from foodstuff can result in higher blood lead levels due to the high bioavailability of dietary lead.

For typical adult recreational activities, less than 5% probability of exceeding 10 µg/dl is predicted for all recreational area soil concentrations observed in the Basin. For intense soil contact recreational practices such as dirt biking, beach activities, four-wheeling, gardening, landscaping, etc., that involve deliberate and continued contact with soils, 95th percentile blood lead estimates exceed 10 µg/dl at concentrations ranging from 3700 mg/kg to 6400 mg/kg lead. These values generally represent the 90th to 95th percentile concentrations in upper Basin recreational areas and 50th to 95th percentiles among Lower Basin common use areas.

Adult blood lead model estimates were developed for medium intensity soil contact occupations or jobs involving periodic exposure to soil sources, such as public property maintenance, typical construction

workers, or laborers. These results suggest that exposures to soils ranging in lead concentration from 2800 mg/kg to 4500 mg/kg could result in more than a 5% probability of blood lead greater than 10 µg/dl. Few soil concentrations in this range are observed in residential areas of the Basin. In Upland Park common use areas, these values correspond to the 90th to 95th percentile of sites. In the Lower Basin floodplain 50% to 95% of soils exceed these levels.

Intensive or RME exposure refers to individuals whose employment specifically involves exposures to soils such as landscapers; farmers and agricultural workers; remediation workers; construction workers routinely involved in excavation, demolition, or site development; or utility or road workers. For these workers, soils near 500 mg/kg could result in more than a 5% probability of having a blood lead level greater than 10 µg/dl. Mineral industry workers are specifically excluded as exposure to lead is specifically regulated by occupational health authorities. Although individuals are not evaluated in this HHRA for lead exposure in the workplace, they are considered in the residential scenario.

8.11.7 Native American Blood Lead Levels

Blood lead levels were not predicted for either the traditional or modern subsistence scenarios because extremely high estimated intake rates coupled with cultural-specific dietary and behavioral considerations invalidate current blood lead models. Nevertheless, projected intake rates are sufficiently high to indicate that blood lead levels associated with subsistence activities in the floodplain of the Lower Basin would exceed any current health criteria for children or adults in either scenario.

It is important to note that the high lead intake rates are associated with several media. Soil and sediment intakes, fish fillet and peeled water potato, and ingestion of disturbed surface water during swimming and bathing activities would each individually result in excessive lead intake. Consumption of whole fish from the Spokane River or un-peeled water potatoes from the Lower Basin would present especially dangerous intake levels. It is likely that background or pristine environmental concentrations would be required for all media to safely support Native American subsistence activities.

8.11.8 Lead Health Risk Reduction Strategies

These overall results suggest complex pathways of exposure are ongoing in the Basin. Resident children's blood lead levels are most related to dust lead loading in the home, followed by independent effects of yard soil lead, interior paint condition and exterior lead paint content. The dust lead pathway is most influenced by outdoor soils, but is augmented by paint contributions particularly in poorly maintained older homes. The overall effect is exacerbated by extremely dusty conditions in Burke/Nine Mile and to a lesser extent in Wallace. Significantly less problem is noted with respect to dustiness or dust concentrations in the Lower Basin. West of the BHSS, excess absorption was associated with either homes that had been flooded or extended recreational activities in the river or lateral lakes areas.

Potentially significant recreational exposures are noted for certain activities in particular areas of the Basin and from consumption of home grown vegetables. Excessive occupational exposures could occur with particular unprotected jobs in highly contaminated areas. Subsistence Native American practices in the Lower Basin would be dangerous, particularly if whole fish or unpeeled water potatoes contribute a substantial portion of the diet.

These pathways suggest an integrated approach to risk reduction may be advised. Baseline residential exposures could potentially be reduced through cleanup of excessive soil contamination coupled with paint stabilization to simultaneously reduce direct exposure to these media and subsequent house dust lead concentrations. Targeted cleanups of recreational areas, coupled with access limitations or appropriate warnings, could be used to prevent excessive incremental exposures. Provision of clean gardening media could reduce incremental exposure to local produce. Worker safety protocols could be developed to protect adults while employed in contaminated soil related jobs. Native Americans should continue to refrain from food harvest and subsistence activities in the Lower Basin until substantial improvements are made. Individual children's problems could be addressed by continuing and enhancing current health intervention activities until final remedial determinations are completed.

For the resident population, children's baseline blood lead levels are likely to be the determining factor in establishing media-specific remediation goals or concentration action levels. The baseline blood lead levels then become a critical determinant in developing required risk reduction strategies for incremental, or away from home, activities. As a result, it is possible to discuss preliminary potential cleanup levels for risk manager's consideration for children's baseline residential exposures and adult occupational and recreational activities.

However, discussion and development of candidate action levels for children's incremental recreational activities and fish and local produce consumption cannot be addressed in this document. Appropriate risk reduction methods and action levels will have to be evaluated by risk managers after fundamental approaches to reducing baseline blood lead levels have been made. Determining whether these actions would be sufficient to reduce non-lead risks to acceptable levels must also be accomplished in relation to actions addressing cumulative lead exposures.

8.11.9 Biokinetic Blood Lead Modeling for Residential Cleanup Levels

In the upper Basin, house dust lead levels are critical determinants of the efficacy of any cleanup strategy. Substantial reduction of upper Basin house dust lead levels will be necessary under any scenario to achieve acceptable blood lead levels.

Quantitative estimates of house dust and blood lead levels associated with proposed remedial activities were developed from the site-specific analysis. These analyses suggest that blood lead levels are highly dependent on dust lead loading rates, yard soil contamination levels, and paint lead, particularly in poorly maintained housing. Dust lead loading rates, in turn, are dependent on both dust loading, or

dustiness in a community, and the lead content of that dust. Outdoor soils, both in the yard and the community, are the primary determinant in dust mat lead concentrations augmented by interior paint lead levels, again in poorly maintained housing. Dust lead inside the home is dependent on dust mat lead, yard soil and interior paint concentrations.

Post-remedial dust lead concentrations for input to the IEUBK model were estimated by the regression model equation that quantitatively describe these pathways. This was accomplished by first estimating post-remedial soil concentrations based on replacing all home yards with soil lead levels exceeding the cleanup threshold with 100 mg/kg lead soils. These soil concentrations were then substituted into the model equations assuming a mean paint lead concentration and good paint condition. This implies that paint stabilization has been implemented. Mat and vacuum dust lead concentration were then successively estimated. The vacuum dust lead estimate was applied with the individual soil concentrations in the batch mode of the IEUBK for all ages of children and the results were aggregated for risk estimates. In this analysis, the entire housing database was utilized, as opposed to only those homes having blood lead observations used to evaluate dose-response relationships.

Preliminary analysis, using the Box Model, suggests that a cleanup threshold for soils of 800 mg/kg to 1000 mg/kg is necessary to achieve risk levels in the upper Basin comparable to those established for the BHSS. The EPA Default Model suggests cleanup levels for soils below 400 mg/kg are required to achieve similar risk criteria. These results are summarized in Figures 8-8a through 8-8h.

Both models indicate that lead paint stabilization will be required in combination with soil remediation to reduce house dust lead concentrations to protective levels. Potential paint stabilization would apply to the approximately 20% of housing units that currently have lead paint in poorly maintained condition. These measures will not resolve excessive blood lead levels observed in the Lower Basin.

In the Lower Basin, and to a lesser extent in the Kingston subarea, yard soil and house dust lead concentration reductions are likely to be less effective in reducing observed high blood lead levels. Residential soil and dust lead concentrations in these areas are generally low and projected residential intake rates do not suggest an excess absorption problem. For these areas, excepting some individual situations, development of strategies addressing incremental exposures outside the home environment are more likely to be effective in reducing risk of lead poisoning.

There are two major considerations in assessing these results. First, the risk of exceeding the health criteria projected in this analysis only accounts for baseline (or home residential) exposures after paint stabilization. Consequently, there is no safety margin allowing for incremental exposures that might occur in addition to home exposure. Second, current USEPA policy addresses individual risks for those children left at the highest exposure levels. Current policy recommends that the probability of the typical 0-84 month old child at any residence experiencing a blood lead level of 10 µg/dl or greater, be less than 5%. Box Model estimates of individual risks indicate this criteria is considerably more stringent than that applied at the BHSS and would require a soil cleanup in the 600

mg/kg to 800 mg/kg range. Using the EPA Default Model to calculate a residential soil cleanup level protective of risk to individuals results in a soil level below the EPA residential soil screening level of 400 mg/kg. This is caused be elevated levels of lead in house dust in portions of the Basin. As a result, risk managers, public health officials and community representatives will need to assess the applicability of this criteria to the Basin population and alternative risk reduction techniques that might provide the necessary level of protectiveness.

8.11.10 Lead Health Risk Reduction for Childhood Recreational Activities

Substantial increases in blood lead levels are predicted for particular play activities in contaminated areas of the Basin. Blood lead increments to existing baseline or residential conditions were developed for this report. However, determination of appropriate risk reduction action levels for soil and sediments in recreational areas cannot be accomplished until appropriate risk management strategies for residential sources have been identified.

8.11.11 Lead Health Risk Reduction for Childhood Consumption of Local Foodstuff

Similarly, the significance of local produce and fish from the lateral lakes area depends on the relative baseline residential blood lead level. In this case, a determination of allowable dietary intake based on baseline blood lead levels will be required. These can be compared to incremental fish and local produce intake tables relating intake to media contaminant levels.

8.11.12 Lead Health Risk Reduction for Adult Occupational Activities

Estimated blood lead levels associated with potential soil and dust concentration levels in occupational activities suggest that in order to maintain 95% of reproductive aged women's blood lead levels below 10 µg/dl, protective measures should be taken for typical workers when in contact with soils exceeding 2800 mg/kg to 4500 mg/kg lead. For those workers engaged in heavy contact with soils for extended periods of time working, the corresponding level of concern is 500 mg/kg lead.

8.11.13 Lead Health Risk Reduction for Adult Recreational Activities

Estimated blood lead responses for Upland Park or land-based recreational activities suggest protective measures should be employed for adults engaging in intense soil-related recreational practices with soils exceeding 3700 mg/kg.

8.11.14 Lead Health Risk Reduction for Adult Consumption of Local Foodstuff

Some local vegetable garden produce shows high lead content that could substantially increase total intake to levels of concern among pregnant women. Adult consumption of local fish adds minimally to total intake at typical fish fillet lead concentrations. However, at maximum concentrations and

consumption rates the increased intake could be of concern, although it is unlikely that the species of fish providing the samples would be consumed in large amounts.

8.11.15 Lead Health Risk Reduction for Native American Subsistence Activities

Native American subsistence practices in the Lower Coeur d'Alene Basin would be ill-advised. Soil and sediment ingestion rates associated with residence in the floodplain and food harvest practices are extremely high. Near background level concentrations would be required to achieve acceptable intake rates for soils and sediments. Additionally, two critical elements of the native diet, fish and water potatoes, contain unsafe levels of lead when aboriginal consumption rates are applied. Lead levels in these food sources may also likely need to be in equilibrium with background soil and water conditions to assure acceptable intake rates.

8.12 RESPONSE TO COMMENTS - PUBLIC REVIEW DRAFT

8.12.1 General and Specific Responses to Public Comments

Numerous comments were received from several reviewers of the Public Review Draft of the HHRA released in July 2000. Appendix W contains a compendium of public comments received and responses. The various written comments received and notes taken regarding discussions and questions during public meetings and presentations were entered into a database as specific comments and concerns. Individual responses to each of the specific written comments received from the public and interested parties are included in the data base. Those comments together with particular concerns expressed during meetings, conference calls, group discussions, and presentations were summarized in eleven general categories and a general response is provided for each comment category.

Also included as part of the response to comments are an independent peer evaluation of the comments received and the *U.S. EPA Technical Review Workgroup for Lead (TRW)* evaluation of the HHRA.

Some comments pointed out errors in presentation or data tables that have subsequently been corrected. Some analyses were modified. Enhanced discussion has been provided in several areas that were confusing to reviewers. Additional discussions regarding uncertainties in the analyses and conclusions were included in Section 7 to assist risk managers in decision making.

8.12.2 Additional Analysis and Appendices in Responses to Public Comments

Additional analysis and new information was added to the document in response to requests from the Public Reviewers. Analyses were amended and supplemental information was included in the text of the document in the following Sections.

- Tables and discussion regarding the potential adverse health effects associated with blood lead levels observed in the Basin were added to Section 6.0.
- A Table summarizing exposure and IEUBK input assumptions for the overall document was added as Appendix V.
- Alternate exposure parameters were employed in assessing the dermal exposure route for arsenic and cadmium included in Section 5.0. These analyses resulted in a 15-20% reduction in estimated exposures to these metals by skin absorption. These modification were made in response to public comments regarding the length of the recreational season in the Basin and the amount of clothing children would be expected to wear in engaging in these activities.
- Additional information regarding the blood lead data base used in the site-specific analysis was provided in Section 6.2.2. Data were provided indicating the number of repeat blood lead observations, the number of households participating in the surveys, and the characteristics of blood lead levels for those children providing more than one sample.
- Additional analysis of the lead paint hazard associated with observed high blood lead levels was presented in Section 6.4.2. This analysis classified homes as having a potential lead paint hazard if lead paint was present and the paint was not in good condition. About 20-25% of the homes tested in the Basin met this criteria. About 30% of children exhibiting high blood lead levels, for which this index was available, were from homes with a potential lead paint hazard. About 70% of the high blood lead children came from homes classified as having no lead paint hazard.
- Drinking water exposure to lead was reformatted in Section 6.0 to separate purged tap water samples collected from home surveys conducted in 1997-1999 from purged individual well source samples collected in the 1996 exposure survey. Previously, these data were averaged together to estimate characteristic purged drinking water lead concentration for various geographic subareas.
- Supplemental analysis of the incidence of cancer in Shoshone County has been included as Appendix U to the HHRA. This analysis, conducted by the Cancer Data Registry of Idaho concludes that the incidence of brain cancer in Shoshone County is not higher than expected, but does warrant periodic reanalysis.
- Appendices that have been added to the Document are Appendices S, T, U, V, and W. These appendices can be found on the attached CD.

8.12.3 Clarification of USEPA Policy Regarding Human Health Risk Assessment for Lead

Several reviewers indicated confusion and dissatisfaction with EPA policy regarding the use of environmental exposure and observed blood lead data to characterize risk to human health at CERCLA sites. USEPA policy requires risk to be characterized by environmental exposures. In the case of lead, the risk presented by the potential environmental exposure is evaluated by the percent of children expected to equal or exceed a 10 Fg/dl blood lead level, if that exposure occurs in the future.

Current USEPA policy addresses individual risks for those children left at the highest exposure levels and recommends that the probability of experiencing a blood lead level of 10 Fg/dl or greater, at any residence, be less than 5%.

Use of the IEUBK Model to Assess Risk: The approved method to estimate this risk is use of the IEUBK model for lead. Other predictive models may be used, if shown to be equivalent. No other published biokinetic models are currently considered equivalent in all their features in terms of specific required features for technical and regulatory application to risk assessment at CERCLA sites. Only the IEUBK is currently approved for use (USEPA 1994d, USEPA 1998d).

Default exposure and absorption parameters are to be used in the IEUBK analysis, unless there is compelling evidence to support the use of site-specific parameters. Default parameters are reflective of typical national conditions and application of these factors results in required cleanup criteria for soils and dusts near 400 mg/kg lead. This effectively serves as a default national cleanup standard that can be amended higher or lower based on site-specific information. Box Model estimates of individual risks indicate this criteria is considerably more stringent than that applied at the BHSS and would require a soil cleanup in the 600 mg/kg to 800 mg/kg range.

Since release of the Public Draft of the HHRA, the State has argued that there is sufficient site-specific evidence to support the use of reduced dose-response parameters for the Basin that could result in higher cleanup levels. This argument is based on the experience and success of using these same adjustments in risk management activities at the BHSS, and analysis of the existing blood lead and environmental database for the Basin. These results suggest that soil and dust cleanup criteria in the range of 800 mg/kg to 1000 mg/kg, applied in conjunction with alternative risk reduction techniques, could provide the necessary level of protectiveness, and would be in compliance with EPA guidance.

Use of Blood Lead Surveys to Assess Model Predications: The HHRA acknowledges, as many critiques have pointed out, that the paired blood lead/environmental exposure database available for the Basin is limited and has not been demonstrated to be representative of the overall population. Blood lead samples were not solicited for experimental or survey purposes. Blood lead samples are observational and opportunistic based on voluntary participation in health response programs. As a result, blood lead levels are not, nor were intended to be, randomized. The blood lead database is limited to less than 1/3 of the 9 month to 9 year old population and less than 20% of pre-school children.

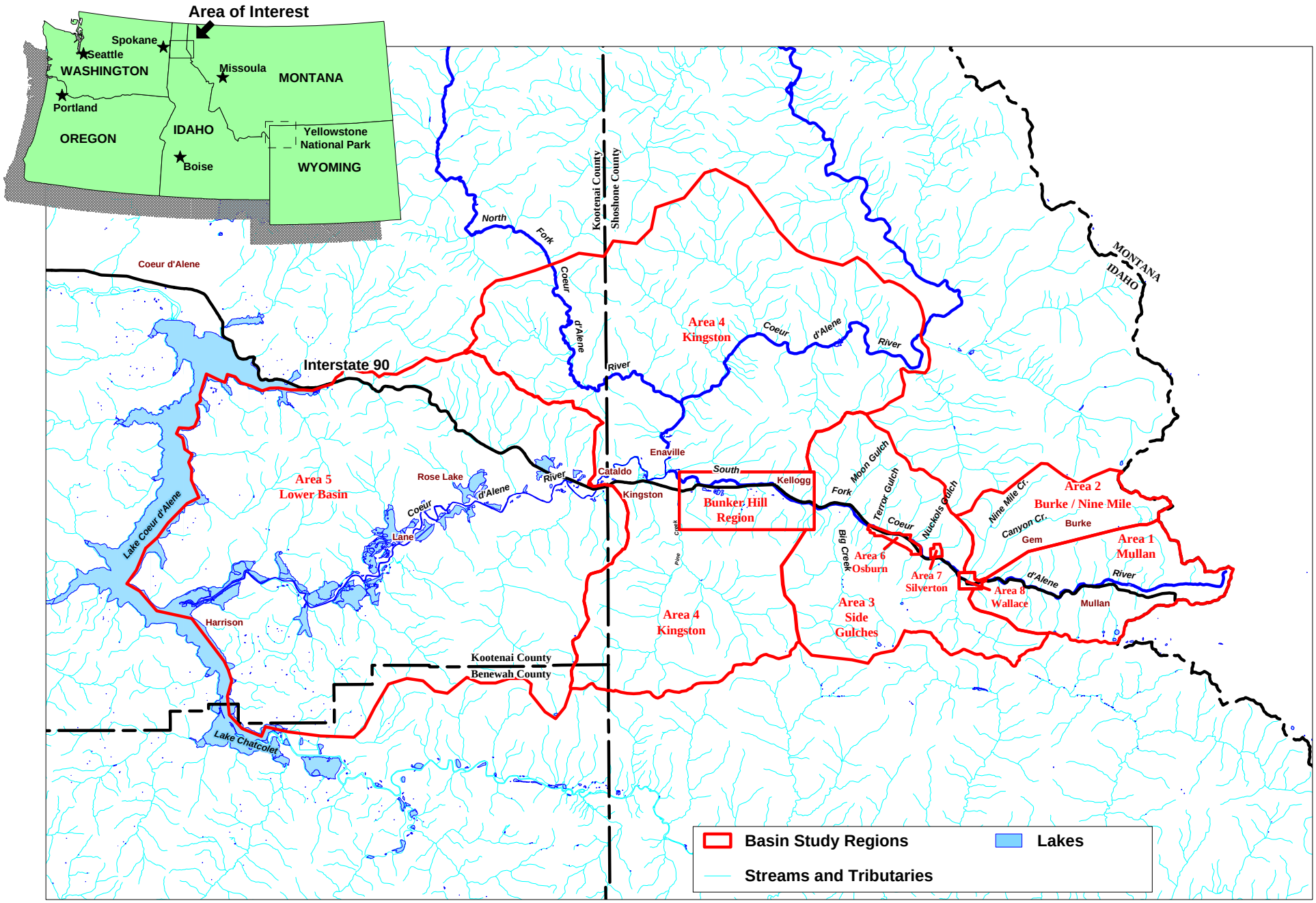
Because there is not a representative database for blood lead levels, survey results cannot be used to quantitatively assess the risk or probability of exceeding blood lead criteria in this population, especially for young children. This limitation was overcome in the BHSS, where participation rates exceed 50% and reasons for failure to participate are known for the majority of the remaining resident children (see Appendix Q). Turnout and identification of reasons for non-participation are insufficient in the Basin to assess the representativeness of the blood lead database.

Use of Site-specific Parameters in the IEUBK Model: Although less than 20% of young children have been tested for blood lead, a representative sample of environmental media was obtained in 1996 and has been substantially supplemented to near 50% of all homes in the Basin. As a result, several analyses of the available data to support site-specific amendments to the IEUBK model were presented. Strict application of current EPA policy requires applying the default mode of the IEUBK in addition to any site-specific analysis. The Default analysis was accomplished and, not surprisingly, results in cleanup threshold criteria near 400 mg/kg lead in soil and dust.

Although the blood lead database does not represent the overall population, it is can be considered representative of the population of children that were tested. Application of the IEUBK analysis to this sub-population, shows a response consistent with children in the BHSS. Moreover, comparison of the environmental exposures for those children that have provided blood lead samples to the overall environmental database shows no obvious differences in exposure media concentration. As a result, any suspected biases in the population providing blood lead levels are likely not media-concentration related, but could reflect behavioral associations with factors such as personal habits and socio-economic conditions.

Additionally, quantitative dose-response analysis of the sampled population shows slope factors for the blood lead to soil and dust concentrations similar to those obtained in the BHSS. Supplemental analysis shows an additional component related to lead-based paint for a small percentage of these children, that was not observed in the BHSS. Finally, application of the "Box" form of the IEUBK model shows conformance with the observed blood lead levels in the sampled population. The combination of these factors, support the State's argument that the "Box" cleanup criteria, augmented by paint abatement efforts and alternate risk reduction techniques, will sufficiently reduce risk for the sampled population and the remainder of Basin residents. It must be noted that the weight of evidence shows conclusively that the sampled population is at-risk of excessive blood lead levels. This is demonstrated both in the Default and Box applications of the IEUBK model, the quantitative analysis of the site-specific dose-response data and, most importantly, by direct blood lead measurements.

Responsible health agencies are compelled to act, at least, in the interest of the sampled population and those children exhibiting dangerous blood lead levels. Because the remainder of the population, that has not been tested, is exposed to similar community environmental concentrations and other children may exhibit similar behaviors and socio-economic status, current policies require they be protected as well. This policy applies unless compelling evidence to the contrary is provided. No such evidence exists.



TerraGraphics Environmental Engineering, Inc.	FILE 1999-07-13 Basin RA / fig8-1	REQUESTOR L.Wichers	Coeur d'Alene Basin Human Health Risk Assessment	Figure 8-1 Coeur d'Alene Basin
	DATE 07JUL2000	DESIGNER B. Bailey		
	PROJECT NO. 980111	CARTOGRAPHER B. Bailey		
		APPROVED L.Wichers		

Figure 8-2 Percent to Exceed Blood Lead Concentrations by Geographic Area - 9 Month through 9 Year old Children (1996 - 1999 Combined)

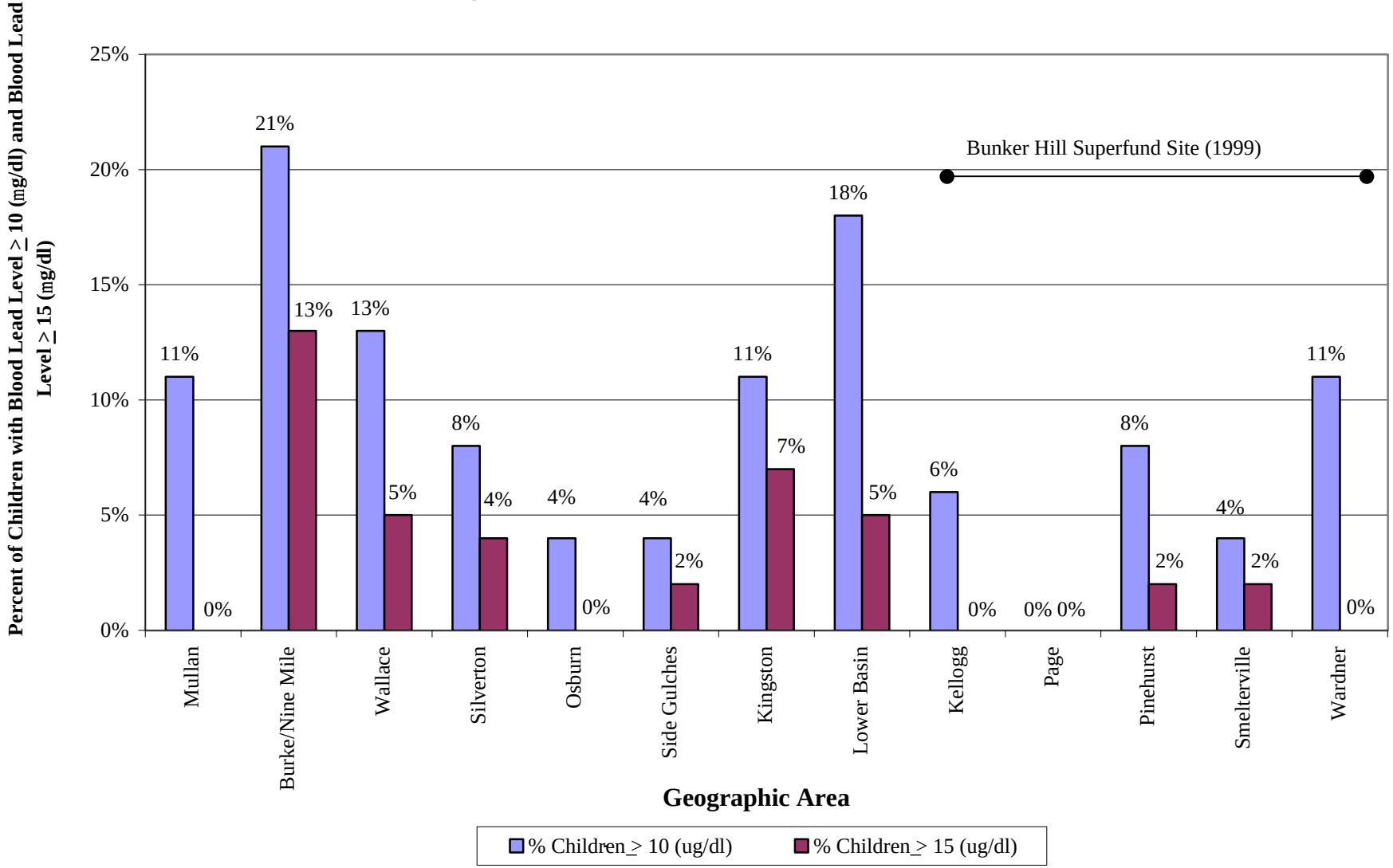


Figure 8-3 Arithmetic and Geometric Mean Blood Lead Concentrations by Geographic Area - 9 Month through 9 Year Old Children (1996 - 1999 Combined)

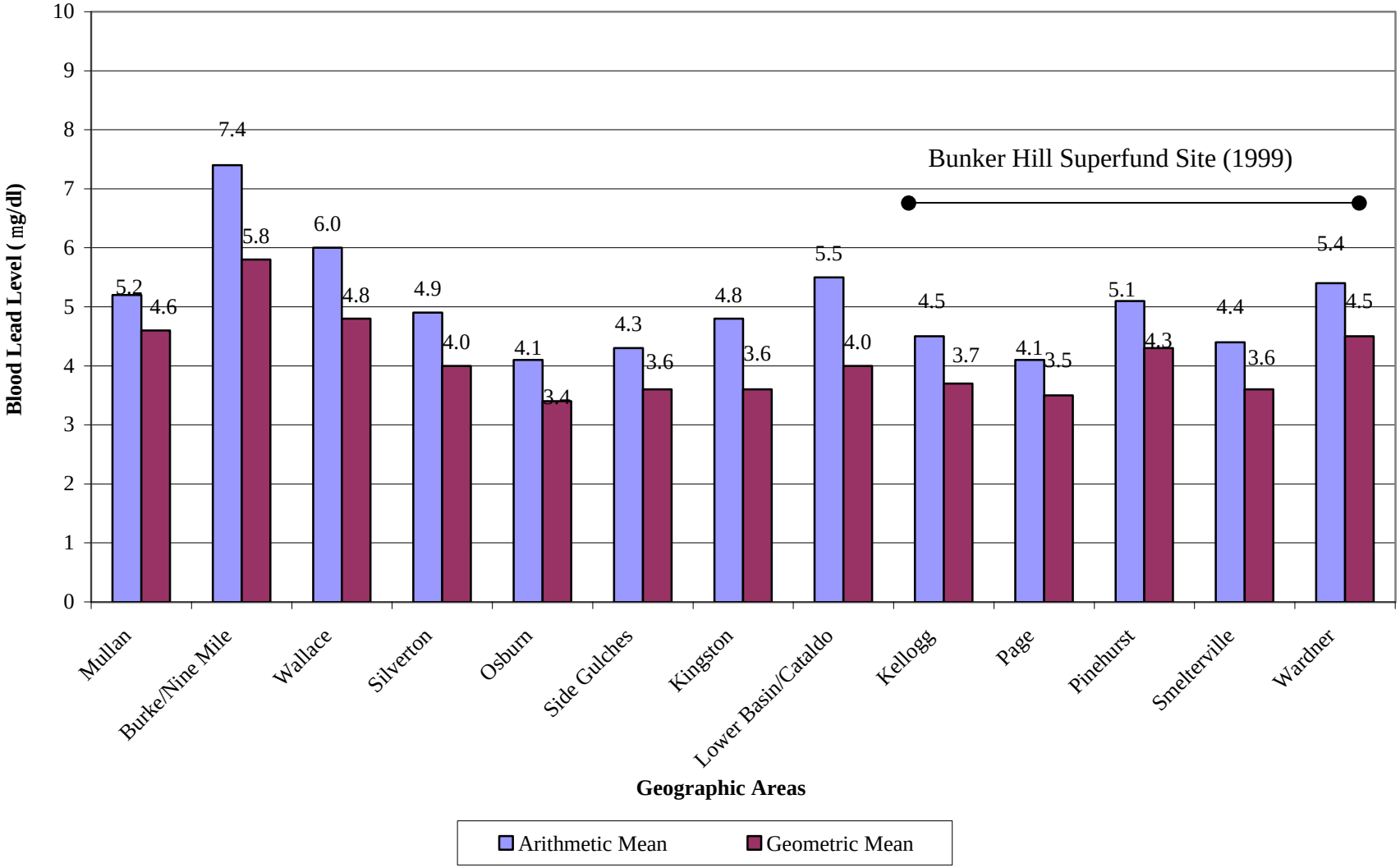


Figure 8-4 Basin Mean Blood Lead Levels by Age (1996 - 1999 combined)

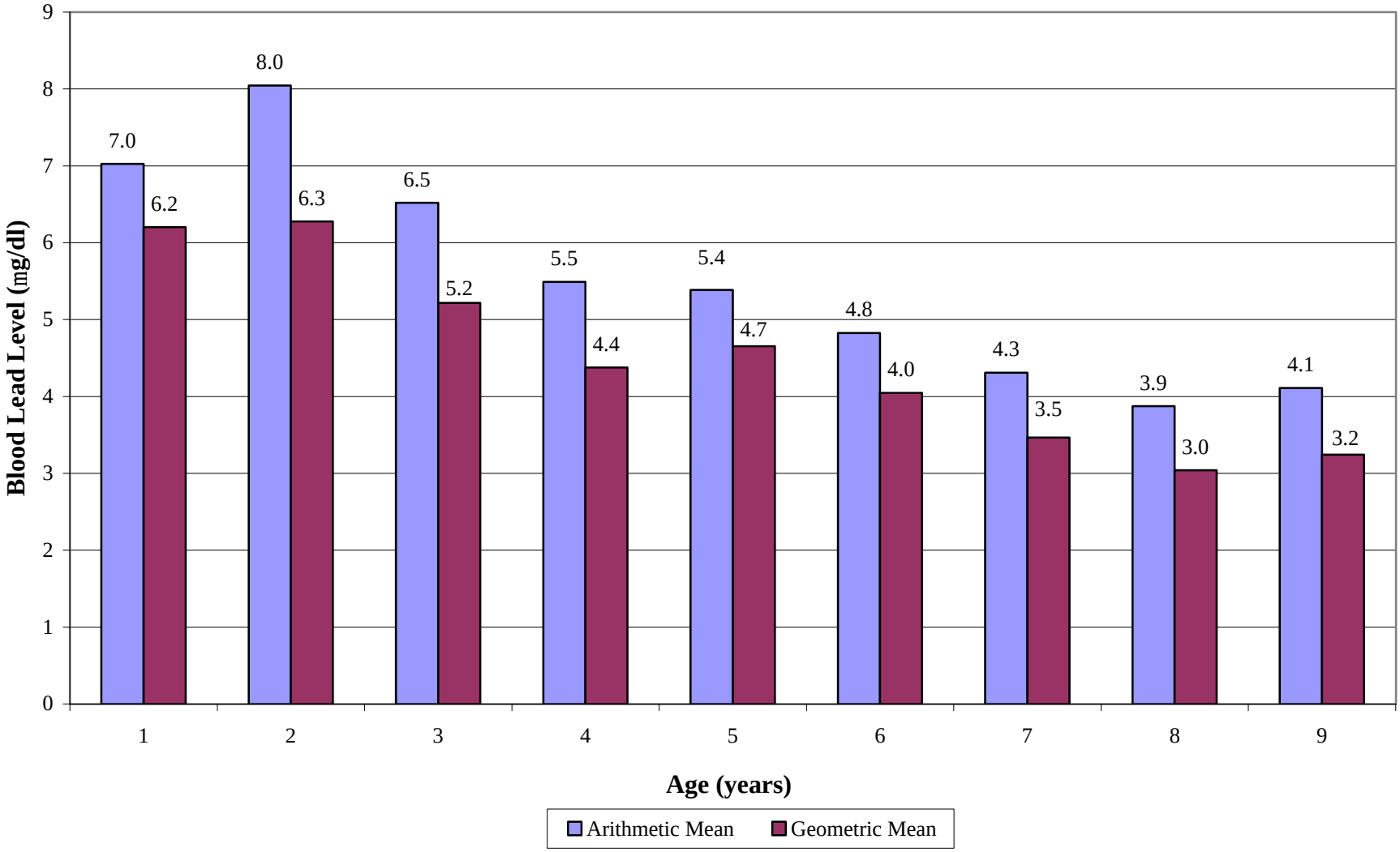


Figure 8-5 Percent of Children to Exceed Critical Toxicity Levels by Age (Basin-wide 1996 - 1999 Combined)

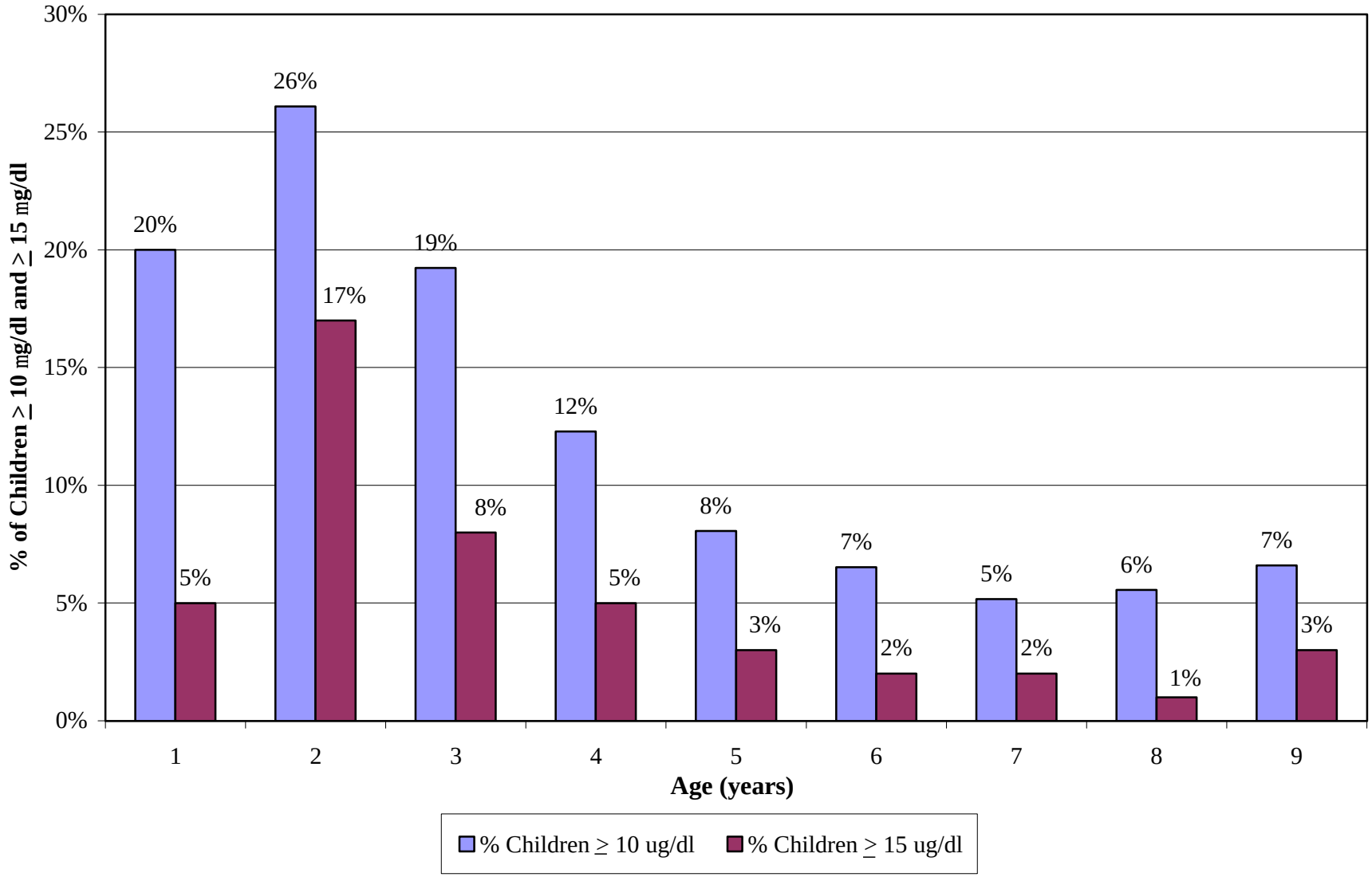


Figure 8-6 Observed and Predicted Geomean Blood Lead Levels for 0-84 Month Old Children Only - IEUBK Batch Mode

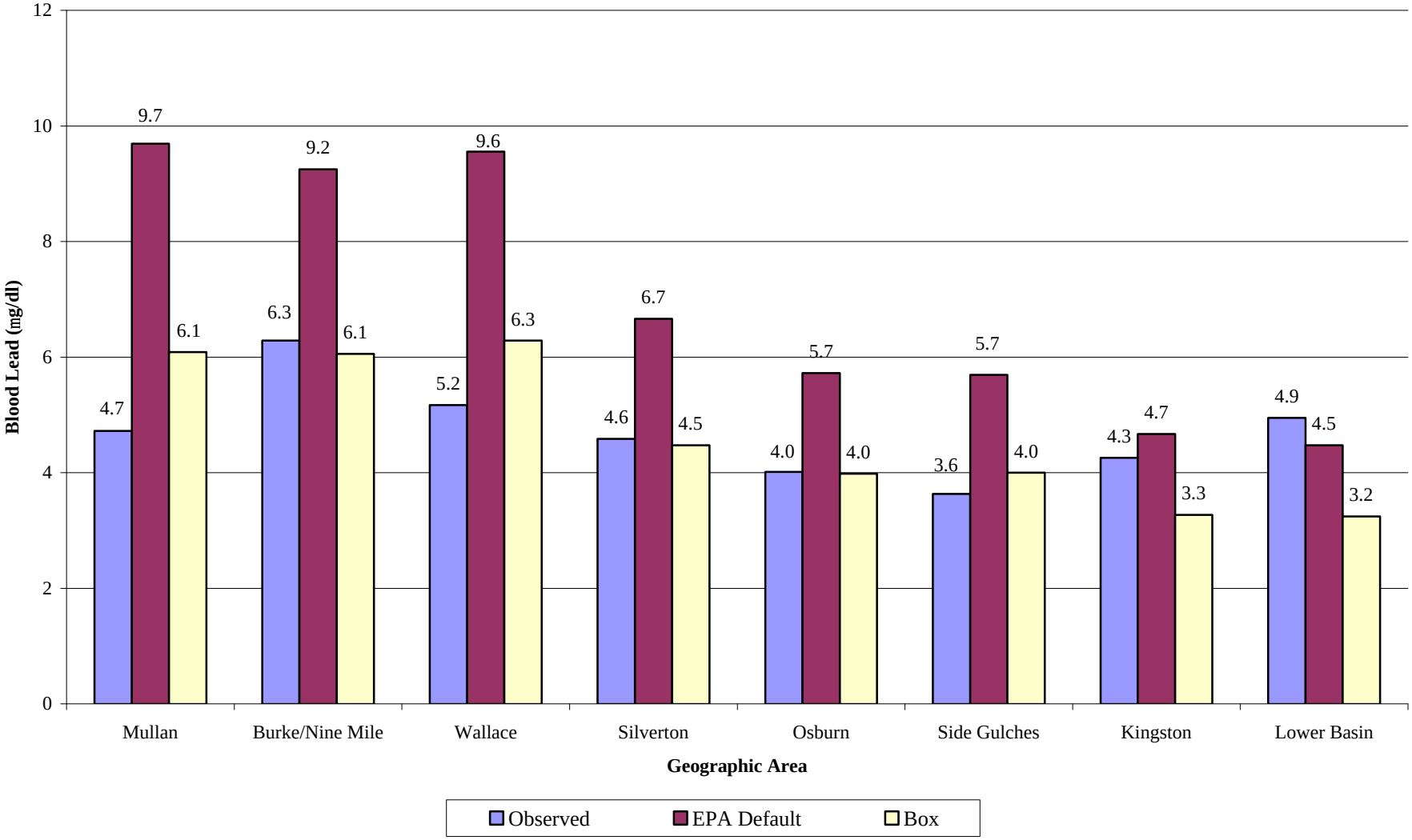


Figure 8-7 Observed and Predicted Percent to Exceed 10 mg/dl for 0-84 Month Old Children - IEUBK Batch Mode

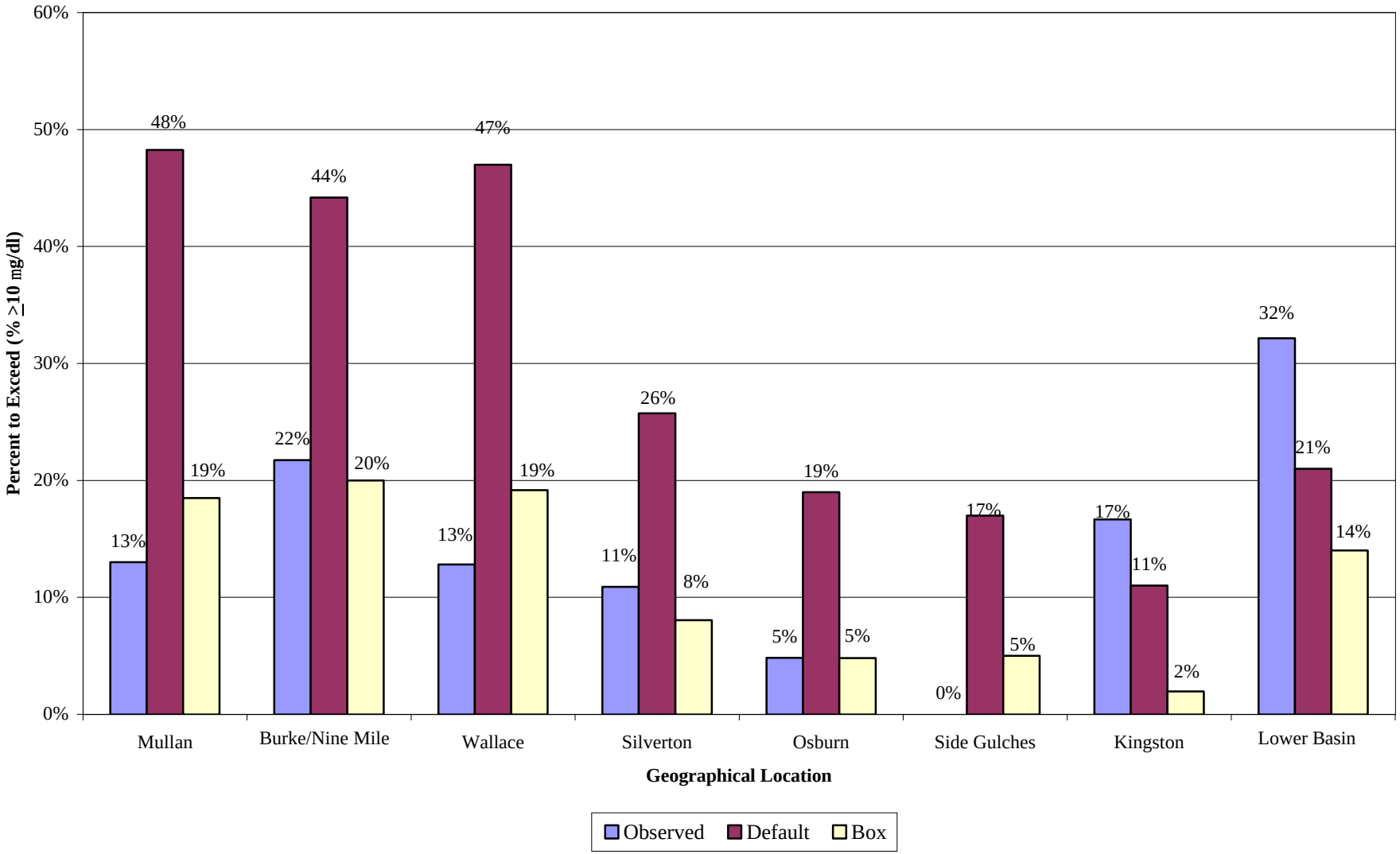
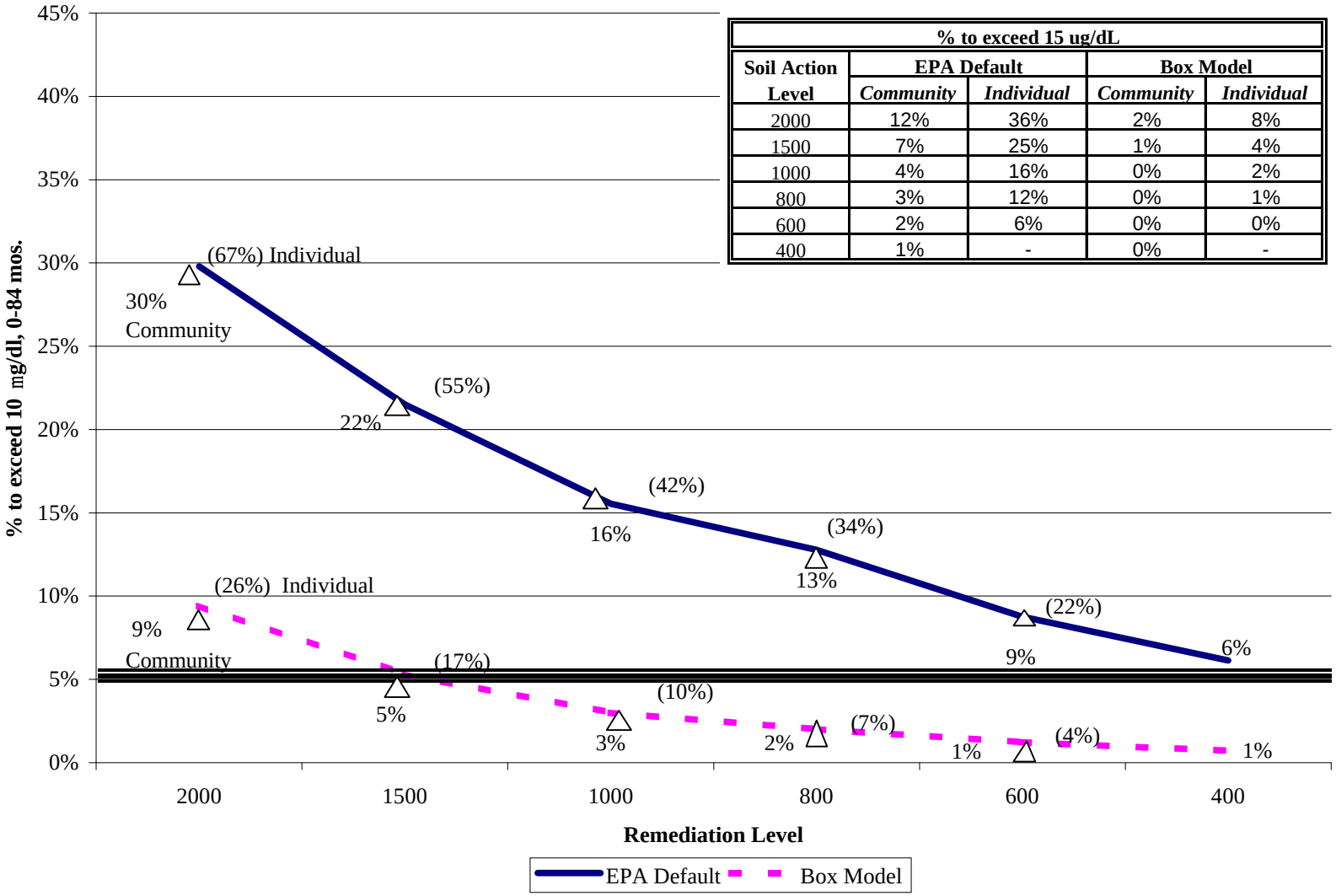
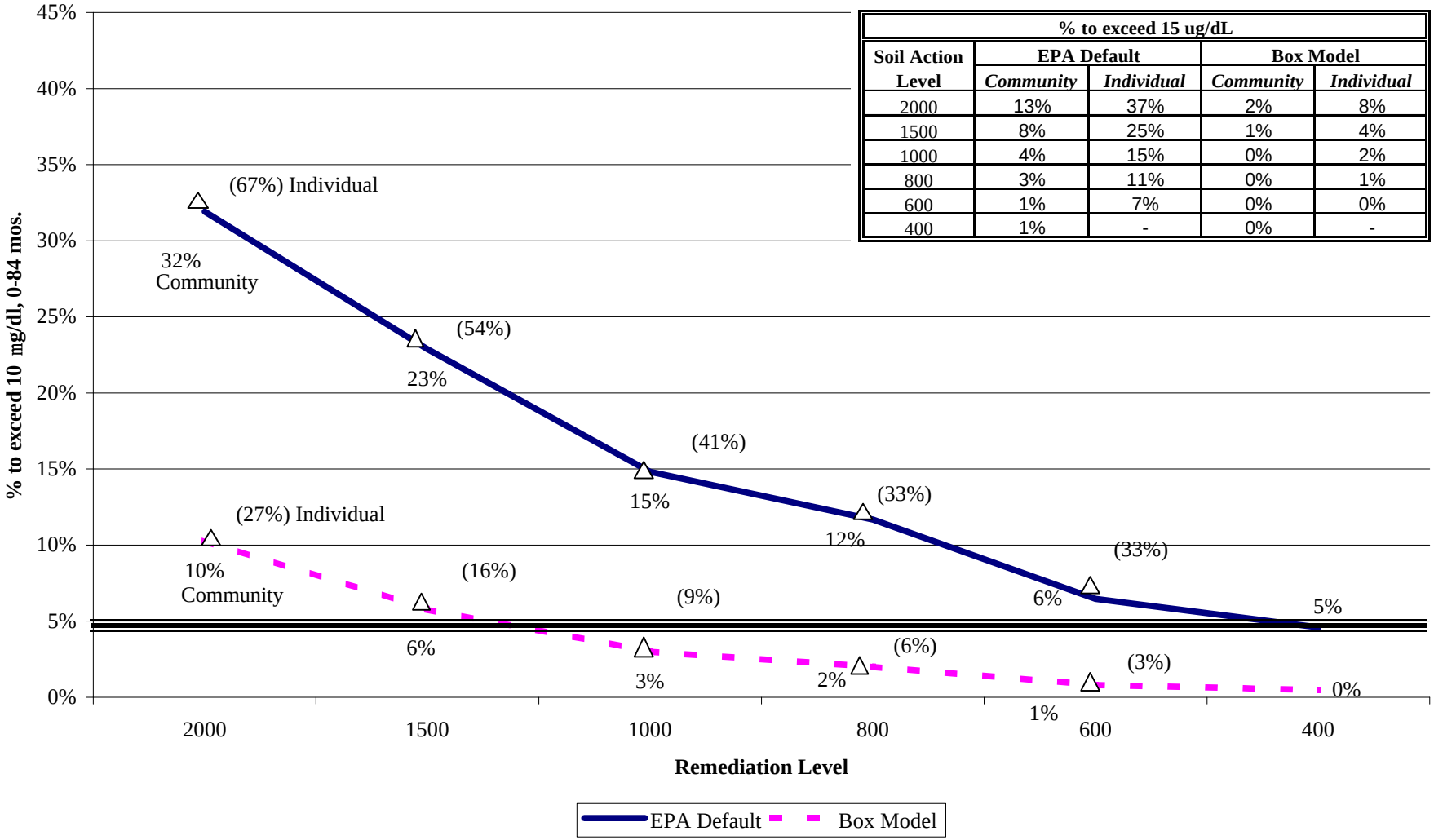


Figure 8-8a Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Mullan



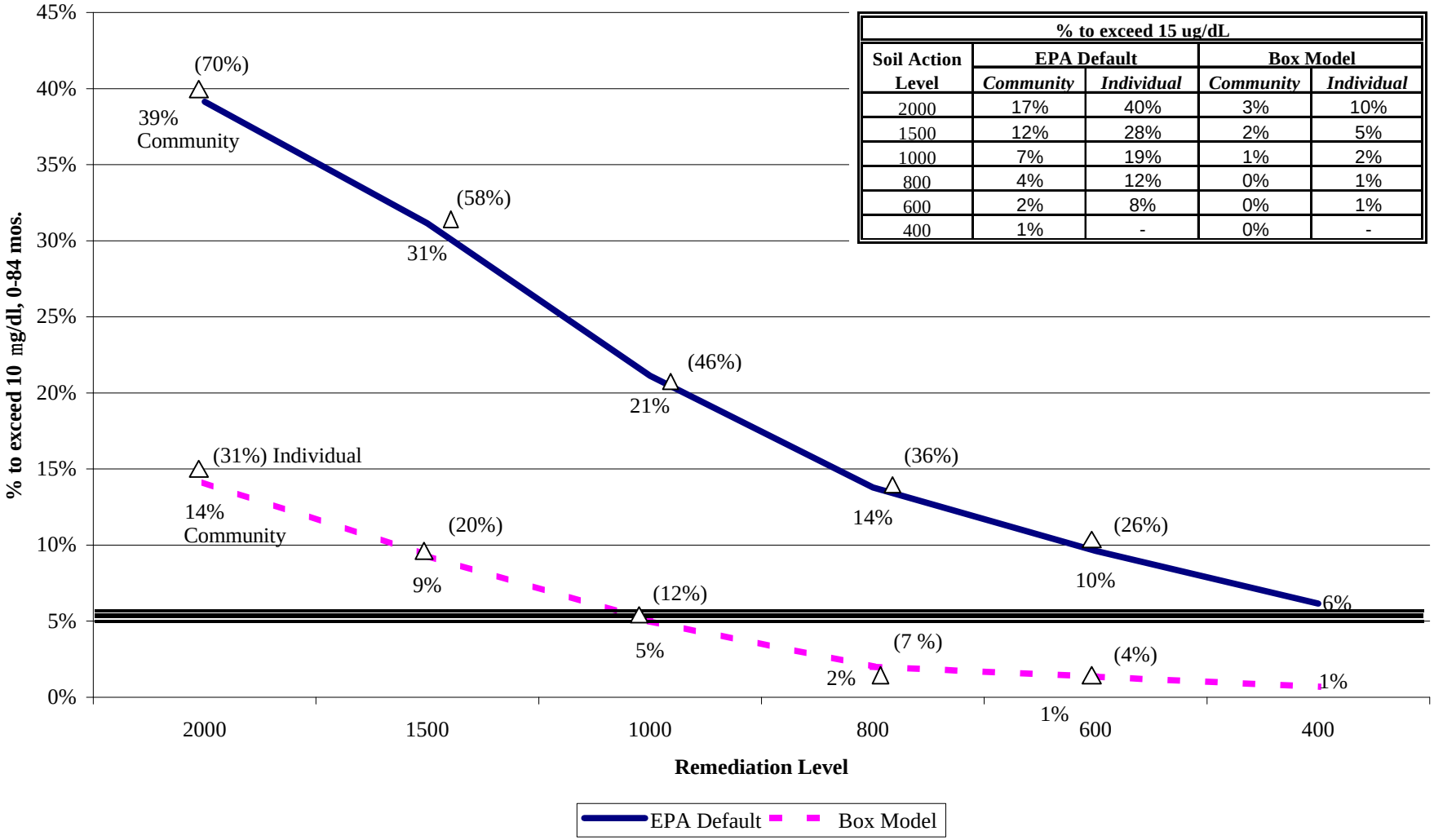
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	12%	36%	2%	8%
1500	7%	25%	1%	4%
1000	4%	16%	0%	2%
800	3%	12%	0%	1%
600	2%	6%	0%	0%
400	1%	-	0%	-

Figure 8-8b Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Burke/Nine Mile



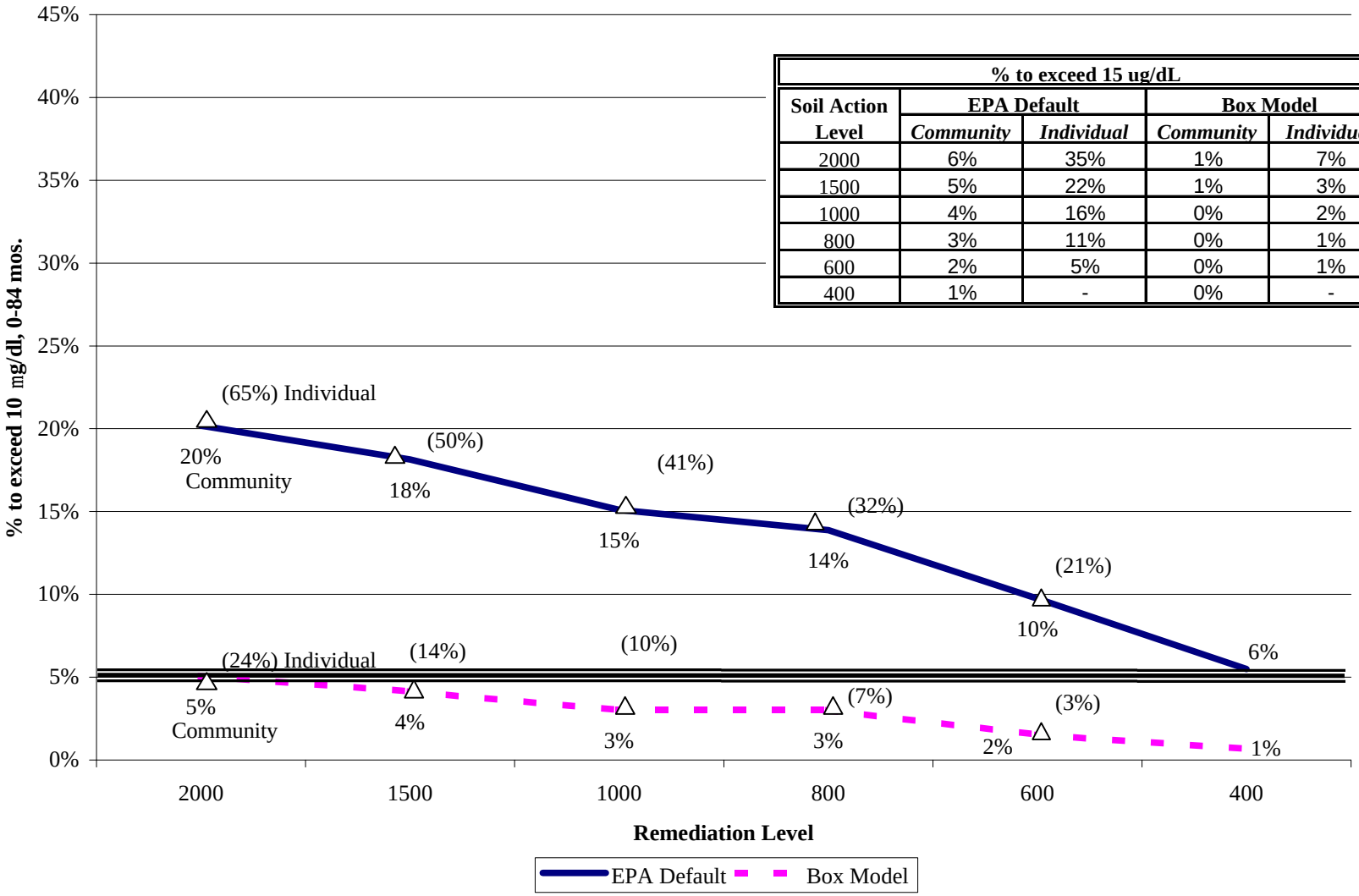
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	13%	37%	2%	8%
1500	8%	25%	1%	4%
1000	4%	15%	0%	2%
800	3%	11%	0%	1%
600	1%	7%	0%	0%
400	1%	-	0%	-

Figure 8-8c Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Wallace



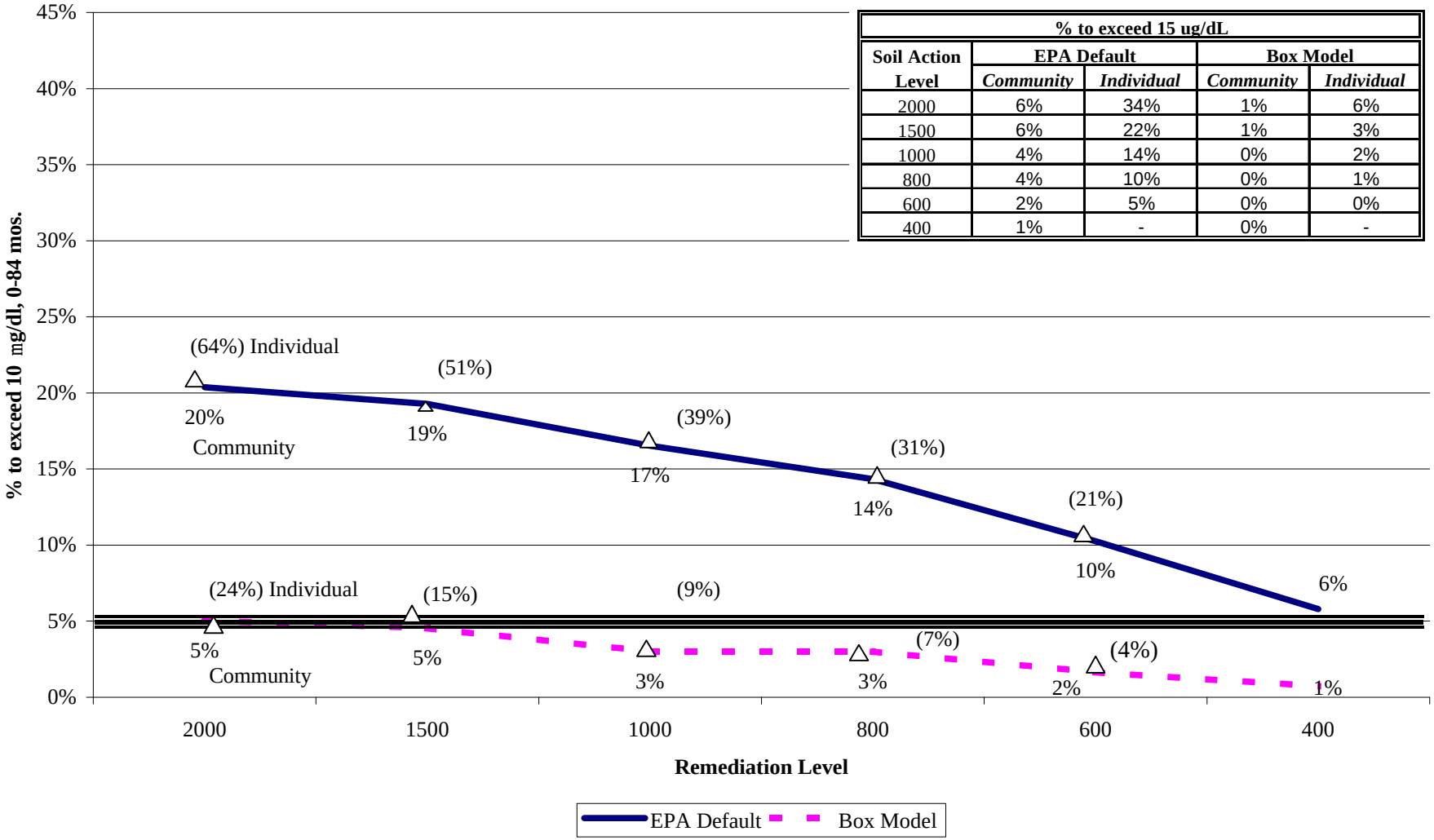
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	17%	40%	3%	10%
1500	12%	28%	2%	5%
1000	7%	19%	1%	2%
800	4%	12%	0%	1%
600	2%	8%	0%	1%
400	1%	-	0%	-

Figure 8-8d Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Silverton



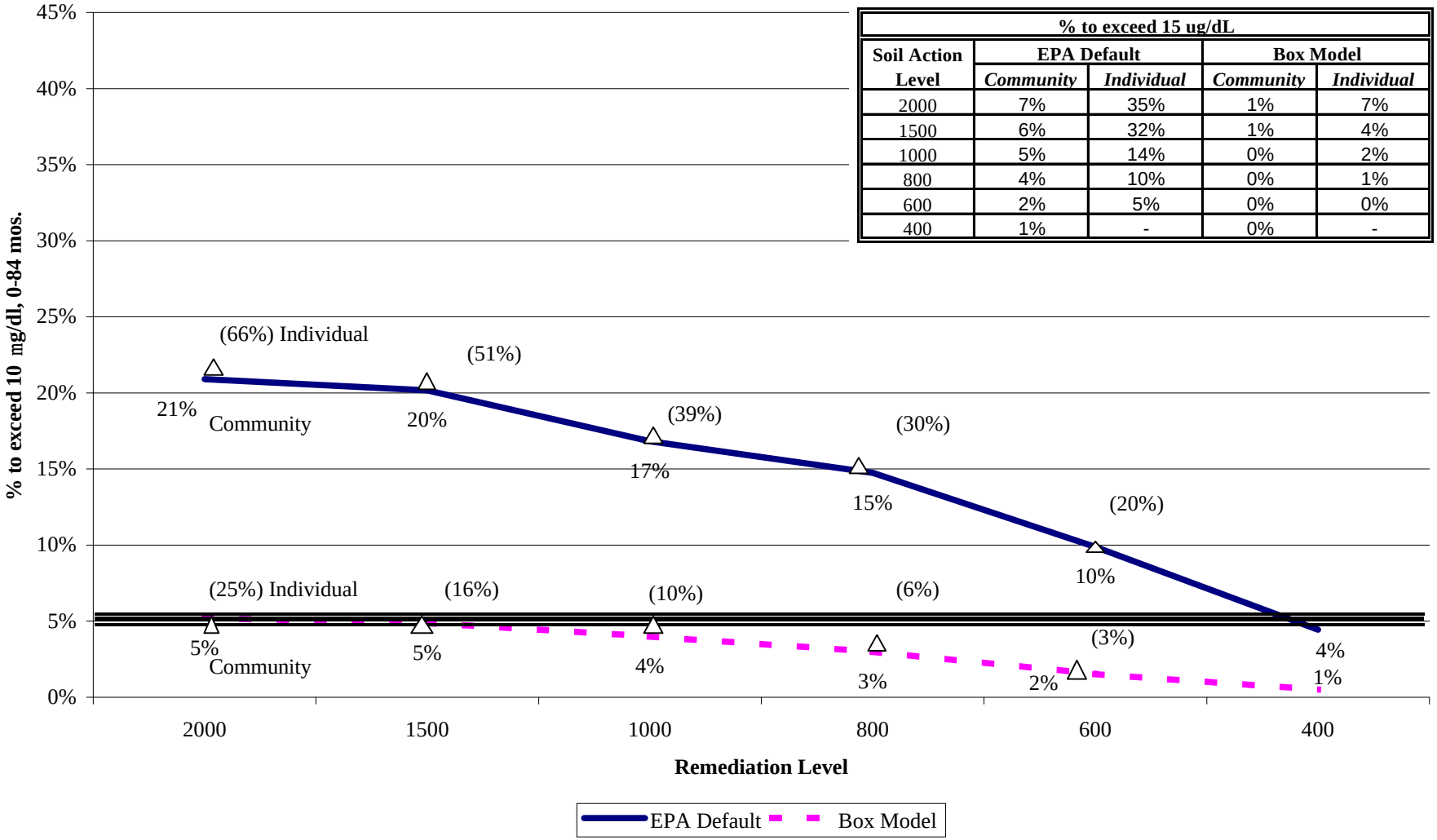
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	6%	35%	1%	7%
1500	5%	22%	1%	3%
1000	4%	16%	0%	2%
800	3%	11%	0%	1%
600	2%	5%	0%	1%
400	1%	-	0%	-

Figure 8-8e Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Osburn



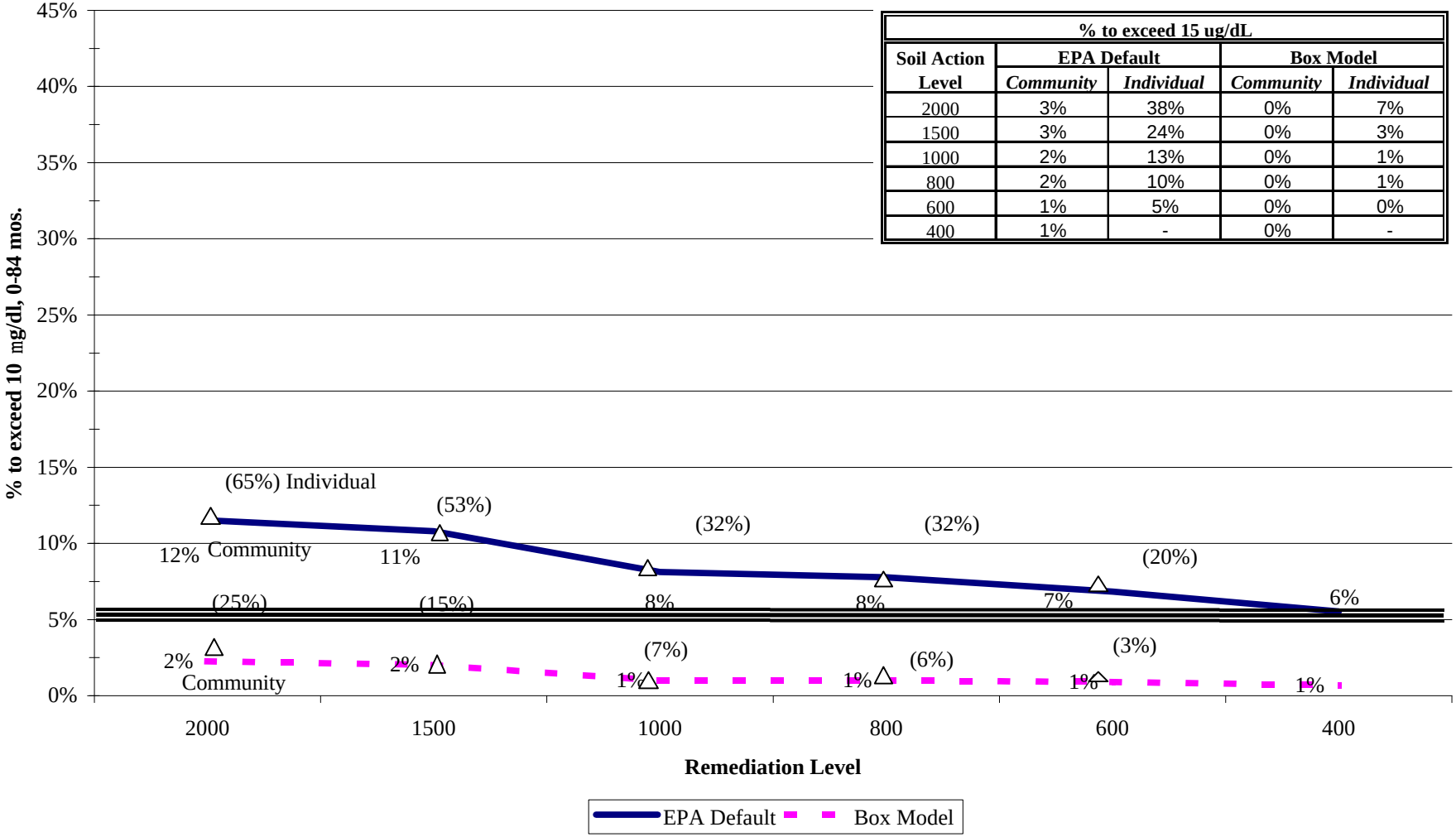
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	6%	34%	1%	6%
1500	6%	22%	1%	3%
1000	4%	14%	0%	2%
800	4%	10%	0%	1%
600	2%	5%	0%	0%
400	1%	-	0%	-

Figure 8-8f Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Side Gulches



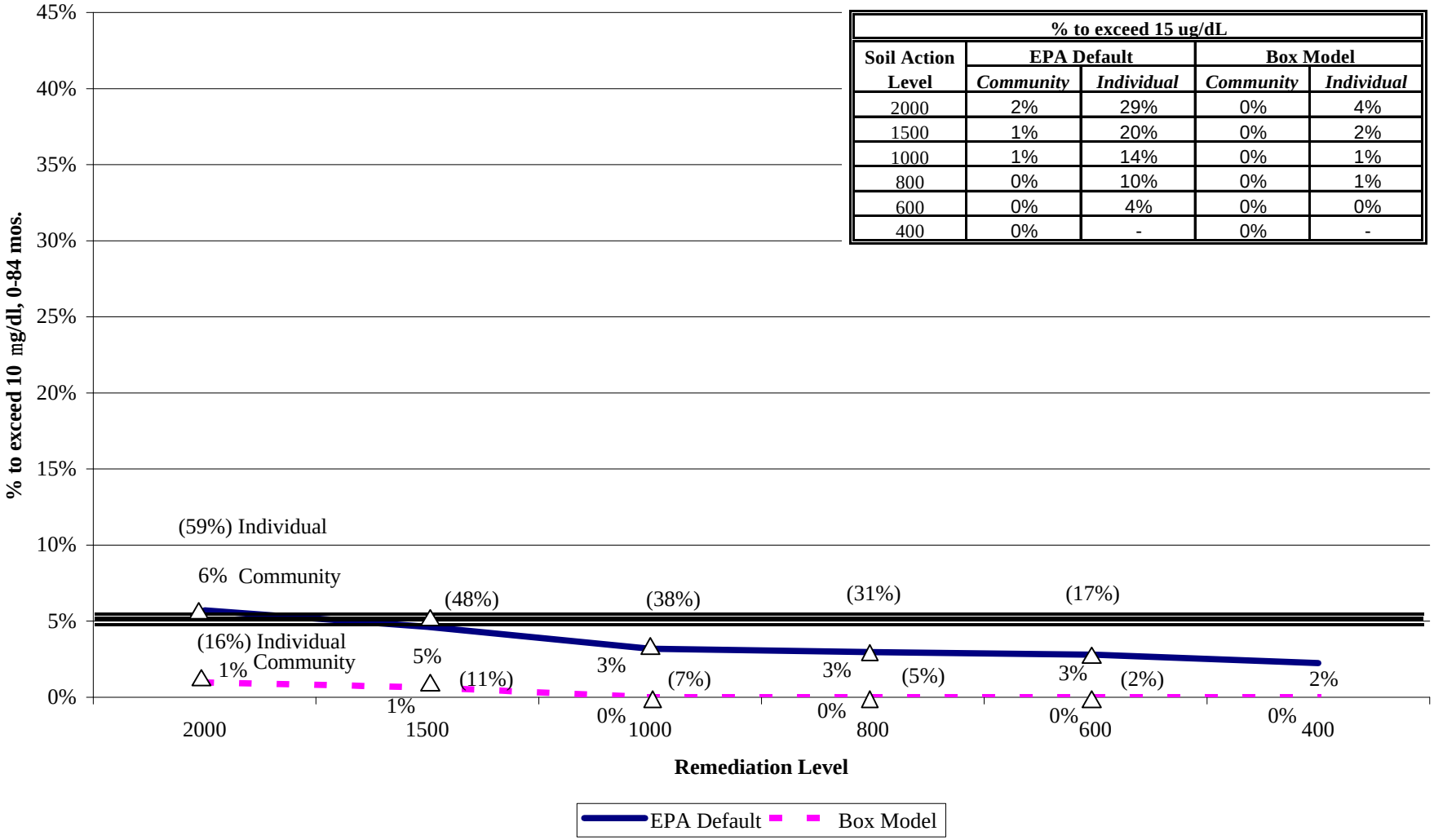
% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	7%	35%	1%	7%
1500	6%	32%	1%	4%
1000	5%	14%	0%	2%
800	4%	10%	0%	1%
600	2%	5%	0%	0%
400	1%	-	0%	-

Figure 8-8g Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Kingston



% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	3%	38%	0%	7%
1500	3%	24%	0%	3%
1000	2%	13%	0%	1%
800	2%	10%	0%	1%
600	1%	5%	0%	0%
400	1%	-	0%	-

Figure 8-8h Predicted Percentage of 0-84 Month Old Children to Exceed 10 mg/dl Blood Lead for Various Yard Soil Action Levels - Lower Basin



% to exceed 15 ug/dL				
Soil Action Level	EPA Default		Box Model	
	Community	*Individual*	*Community*	*Individual*
2000	2%	29%	0%	4%
1500	1%	20%	0%	2%
1000	1%	14%	0%	1%
800	0%	10%	0%	1%
600	0%	4%	0%	0%
400	0%	-	0%	-

Table 8-1
Selected Chemicals of Potential Concern in Each Medium

Chemical	Soil/ Sediment	House Dust	Tap Water	Surface Water	Groundwater	Air	Fish	Vegetables
Antimony	X	X			X			
Arsenic	X	X	X	X	X			X
Cadmium	X	X		X	X		X	X
Iron	X	X						
Lead	X	X	X	X	X	X	X	X
Manganese	X	X		X				
Mercury				X			X	
Zinc	X	X			X			

Table 8-2 Health Effects of Exposure to Chemicals of Potential Concern

Chemical	Health Effects	Carcinogenic Effects
Antimony	Respiratory effects, gastrointestinal effects, elevated blood pressure, fibrosis of the lungs, altered pulmonary function	
Arsenic	Gastrointestinal irritation, neuropathy, skin lesions, vascular disease, death due to cardiopulmonary collapse (acute dose)	Skin, liver, bladder, lung, kidney cancer. EPA Group A carcinogen[a].
Cadmium	Kidney disease, skeletal toxicity, cardiovascular disease, anemia, hypertension	Lung cancer. EPA Group B1 carcinogen[a].
Iron	Gastrointestinal ulcers, metabolic acidosis, kidney damage, liver damage, disturbance of endocrine function, diabetes mellitus, cardiovascular effects, siderosis, death due to renal failure or cirrhosis of the liver (acute dose), lipid peroxidation leading to genotoxic effects, hemachromatosis	
Lead	Neurocognitive, neurobehavioral, CNS effects, hematological, kidney effects (higher susceptiblity in young children)	Renal cancer. EPA Group B2 carcinogen[a].
Manganese	Respiratory tract irritaiton, CNS disorder resembling Parkinsonism (manganism)	EPA Group D carcinogen[a]
Mercury	Developmental toxicity, kidney toxicity, neurological effects	EPA Group C carcinogen[a] (mercuric chloride and methylmercury only), EPA Group D carcinogen (elemental mercury)
Zinc	Gastrointestinal irritation, anemia, fever-like symptoms, respiratory injury	EPA Group D carcinogen[a]

[a]EPA's Weight-of-Evidence Classification System:
Group A - human carcinogen (sufficient evidence in humans)
Group B1 - probable human carcinogen (limited human data available)
Group B2 - probable human carcinogen (sufficient evidence in animals, inadequate or no evidence in humans)
Group C - possible human carcinogen (limited evidence in animals)
Group D - not classifiable with regard to human carcinogenicity

Table 8-3
RME Hazard Quotients and Cancer Risks for Residential and Neighborhood Scenarios

Geographical Area	Children Age 0 to 6 Years[a]	Children Age 4 to 11 Years (Risks/Hazards From Neighborhood)		Children and Adults Age 0 to 30 Years	
	Hazard Quotient	Hazard Quotient	Cancer Risk	Hazard Quotient	Cancer Risk
Lower Basin	4	1	2×10^{-5}	1	1×10^{-4}
Kingston	2	1	3×10^{-5}	0.7	5×10^{-5}
Side Gulches	6	2	2×10^{-5}	2	3×10^{-4}
Osburn	3	0.3	5×10^{-6}	0.9	8×10^{-5}
Silverton	2	0.5	9×10^{-6}	0.7	5×10^{-5}
Wallace	3	0.6	8×10^{-6}	0.8	5×10^{-5}
Burke/Nine Mile, current conditions	3	1	4×10^{-5}	1	8×10^{-5}
Burke/Nine Mile, future conditions	22	Same as current	Same as current	12	3×10^{-5}
Mullan	3	0.4	4×10^{-6}	1	7×10^{-5}
Vegetables (all areas)	2	Not evaluated	Not evaluated	2	8×10^{-5}

[a]Cancer risks were not evaluated for the 0- to 6-year age group.

Table 8-4
RME Hazard Quotients and Cancer Risks for Public Recreational Scenario

Geographical Area	Children Age 0 to 6 Years[a]	Children and Adults Age 0 to 30 Years	
	Hazard Quotient	Hazard Quotient	Cancer Risk
Blackwell Island	0.7	0.2	1×10^{-5}
Lower Basin Soil/water risks/hazards	2	0.5	3×10^{-5}
Fishing in lateral lakes	Not evaluated	0.9	No arsenic data
Kingston (confluence of the North Fork and South Fork)	2	0.6	4×10^{-5}
Side Gulches	No public areas evaluated	No public areas evaluated	No public areas evaluated
Osburn	No public areas evaluated	No public areas evaluated	No public areas evaluated
Silverton	0.3	0.09	6×10^{-6}
Wallace	0.5	0.1	6×10^{-6}
Burke/Nine Mile (current conditions)	No public areas evaluated	No public areas evaluated	No public areas evaluated
Mullan	No public areas evaluated	No public areas evaluated	No public areas evaluated

[a]Cancer risks were not evaluated for the 0- to 6-year age group.

Table 8-5
RME Hazard Quotients and Cancer Risks for Occupational Scenario

Geographical Area	Adults (25 Years of Exposure)	
	Hazard Quotient	Cancer Risk
Lower Basin	2	8×10^{-5}
Kingston	0.5	3×10^{-5}
Osburn, Silverton, Wallace areas combined	0.4	2×10^{-5}
Burke/Nine Mile	0.4	2×10^{-5}
Mullan	0.5	2×10^{-5}

Table 8-6
RME Hazard Quotients and Cancer Risks for Arsenic for Modern and Traditional Future Subsistence Exposure Scenarios

Age Group	Modern		Traditional	
	Hazard Quotient	Cancer Risk	Hazard Quotient	Cancer Risk
Adult	3	No arsenic data	10	No arsenic data
Child	9	Not evaluated	43	Not evaluated
Child/adult	4	7×10^{-4}	19	3×10^{-3}

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APPENDICES

Appendix A	Risk Assessment Guidance for Superfund (RAGS), Volume 1, Part D, Tables
Appendix B	Expedited Screening Level Risk Assessment for Common Use Areas, Coeur d'Alene River Basin
Appendix C	Summary Table of Potential Receptor Groups
Appendix D	Graphs of Concentration Distributions of Mercury, Thallium, and Manganese
Appendix E	Data Set Used to Calculate Human Health Exposure Point Concentrations
Appendix F	Distribution Checks and Statistical Summaries of Data by Geographical Area and Medium
Appendix G	95 Percent Upper Confidence Limit Statistical Summary Results by Geographical Area and Medium
Appendix H	Toxicity Profiles for Chemicals of Potential Concern
Appendix I	Graphs and Statistical Analysis of House Dust Concentrations
Appendix J	Potential Dose from Consumption of Beef
Appendix K	Summary Statistics for Antimony, Arsenic, Cadmium, Iron, Manganese, and Zinc for the Fall 1999 Coeur d'Alene Basin Sampling Event
Appendix L	Summary Statistics for Antimony, Arsenic, Cadmium, Iron, Manganese, and Zinc for the Summer 1999 Coeur d'Alene Basin Sampling Event
Appendix M	QA/QC Memoranda for the Summer and Fall 1999 Coeur d'Alene Basin Sampling Events
Appendix N	Proposed Geographic Subdivisions and Archived Reanalysis Results for the Coeur d'Alene Basin Human Health Risk Assessment
Appendix O	Selected EPA Guidance Documents for Lead
Appendix P	Baseline Intakes for the EPA Default and Box Model Scenarios by Area and by Age Level
Appendix Q	Development of the IEUBK Bunker Hill Superfund Site Models and Bioavailability Estimates
Appendix R	Observed and Predicted Blood Lead Levels by Age, Area, Dust Concentration and Action Criteria
Appendix S	Heavy Metals Concentrations in Water Potatoes in the Coeur d'Alene Basin, Idaho
Appendix T	Masked Basin HHRA Lead Data
Appendix U	Cancer Rate Analysis in Shoshone County
Appendix V	IEUBK Input Parameters
Appendix W	Comments and Responses on Public Review Draft, July 2000

Appendix A

Risk Assessment Guidance for Superfund, Volume 1, Part D, Tables

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This appendix includes the Rags Part D Tables 1 through 10 series for the non-lead metals. The tables are organized by geographical areas first and second by scenario. The Table 2 series is organized first by CSM unit and second by medium. The summary table on the following page lists the order of the tables from the Table 3 series on. The order of the geographical areas is as follows: Lower Basin, Kingston, Side Gulches, Osburn, Silverton, Wallace, MidGradSeg02, Ninemile, Mullan, Blackwell Island, and all areas (fish and vegetables). Within each geographical area, the receptor order is as follows: residential, neighborhood recreational, public recreational, occupational, and subsistence modern and traditional (for Lower Basin).

Included in the appendix are the following:

1 Table 1 – Selection of Exposure Pathways
19 Table 2's – Occurrence, Distribution, and Selection of COPCs
49 Table 3's – Exposure Point Concentrations
24 Table 4's – Daily Intakes
2 Table 5's – Noncancer Toxicity Data
2 Table 6's – Cancer Toxicity Data
182 Table 7's – Noncancer Hazards
108 Table 8's – Cancer Risks
96 Table 9's – Summary of Noncancer and Cancer Results
61 Table 10's – Summary of Noncancer and Cancer Results for Chemicals and Pathways That Exceed Target Risk/Hazard Goals

Following the Tables 1 through 10 series are the data usability worksheets for field sampling plans (FSPs) 2 through 8 and FSP12. These worksheets provide information about the appropriateness and usability of the data used in the human health risk assessment.

Summary of Part D Table 3 Series by Geographical Area

Table 3 series number is listed in the second column for each geographical area.
The population exposed to a particular media is listed as well as the exposure medium because of the different combinations of data applicable to the different populations, see text (e.g., occupational exposures vs residential exposures).

Lower Basin

1)	3.1.1	Residential Surface Soil
2)	3.1.2	Residential Groundwater (Tap Water)
3)	3.1.3	Neighborhood and Public Recreational Soil/Sediment (Lower CDAR)
4)	3.1.4	Neighborhood and Public Recreational Surface Water (Lower CDAR)
5)	3.1.5	Subsistence Sediment
6)	3.1.6	Subsistence Plant Tissue (Water Potato)
7)	3.1.7	Occupational Soil
8)	3.1.8	Subsistence Surface Soil
9)	3.1.9	Subsistence Undisturbed Surface Water
10)	3.1.10	Subsistence Disturbed Surface Water

Kingston

1)	3.2.1	Residential Surface Soil
2)	3.2.2	Residential Groundwater (Tap Water)
3)	3.2.3	Neighborhood Recreational Sediment (Pine Creek)
4)	3.2.4	Neighborhood Recreational Surface Water (Pine Creek)
5)	3.2.5	Neighborhood and Public Recreational Soil/Sediment (NS Confluence)
6)	3.2.6	Neighborhood and Public Recreational Surface Water (NS Confluence)
7)	3.2.7	Occupational Soil

Side Gulches

1)	3.3.1	Residential Surface Soil
2)	3.3.2	Residential Groundwater (Tap Water)
3)	3.3.3	Neighborhood Recreational Surface Soil (Elk Creek Area)
4)	3.3.4	Neighborhood Recreational Sediment (Elk Creek Pond)
5)	3.3.5	Neighborhood Recreational Surface Water (Elk Creek Pond)

Osburn

1)	3.4.1	Residential Surface Soil
2)	3.4.2	Residential Groundwater (Tap Water)

Silverton

1)	3.5.1	Residential Surface Soil
2)	3.5.2	Residential Groundwater (Tap Water)
3)	3.5.3	Neighborhood and Public Recreational Surface Soil (Upland Parks/Schools)

Wallace

1)	3.6.1	Residential Surface Soil
2)	3.6.2	Residential Groundwater (Tap Water)
3)	3.6.3	Neighborhood and Public Recreational Surface Soil (Upland Parks/Schools)

Osburn, Wallace, and Silverton (MidGradSeg01)

1)	3.7.1	Neighborhood Recreational Sediment (South Fork CDAR)
2)	3.7.2	Neighborhood Recreational Surface Water (South Fork CDAR)
3)	3.7.3	Occupational Soil

Nine Mile

1)	3.8.1	Residential Surface Soil
2)	3.8.2	Residential Groundwater (Tap Water - Current/Future)
3)	3.8.3	Residential Groundwater (Tap Water - Future)
4)	3.8.4	Neighborhood Recreational Surface Soil (Waste Piles)
5)	3.8.5	Neighborhood Recreational Sediment (Nine Mile/Canyon Creek)
6)	3.8.6	Neighborhood Recreational Surface Water (Nine Mile/Canyon Creek)
7)	3.8.7	Occupational Soil

Mullan

1)	3.9.1	Residential Surface Soil
2)	3.9.2	Residential Groundwater (Tap Water)
3)	3.9.3	Neighborhood Recreational Surface Soil (Waste Piles)
4)	3.9.4	Neighborhood Recreational Sediment (South Fork CDAR)
5)	3.9.5	Neighborhood Recreational Surface Water (South Fork CDAR)
6)	3.9.6	Occupational Soil

Blackwell Island

1)	3.10.1	Public Recreational Soil/Sediment (Spokane River)
2)	3.10.2	Public Recreational Surface Water (Spokane River)

All Geographical Areas

1)	3.11.1	Residential Plant Tissue (Homegrown Vegetables)
2)	3.11.2	Recreational and Subsistence Animal Tissue (Fish From Lower CDAR)

DATA USABILITY WORKSHEET

Site: Field Sampling Plans 2 and 3

Medium: Surface water in Canyon, Ninemile, Big, Moon, Beaver and Pine Creeks and the South Fork of the CdA River.
Sediment in Canyon, Ninemile and the South Fork

Requirement	Comment
Field Sampling	
Discuss sampling problems and field conditions that affect data usability.	No substantial sampling problems or adverse filed conditions that might affect data usability were noted.
Are samples representative of receptor exposure for this medium (e.g. sample depth, grab vs composite, filtered vs unfiltered, low flow, etc.)?	Sample locations not selected based on human use patterns. Water sampling occurred at low flow, and samples were unfiltered. Sediment samples were bulk (not sieved), and were composited over depths as great as 4 feet.
Assess the effect of filed QC results on data usability.	Although small concentrations of several common analytes were found in some of the equipment rinseate and field blanks, the overall quality of the data were determined not to be adversely affected.
Summarize the effect of field sampling issues on the risk assessment, if applicable.	No appreciable field sampling issues were identified that might have any impact on the quality of the risk assessment.
Analytical Techniques	
Were the analytical methods appropriate for quantitative risk assessment?	Overall, the methods chosen were appropriate and adequate to provide useable data for a quantitative risk assessment.
Were detection limits adequate?	QAPP specified detection limit goals were met with the exception of some antimony, arsenic, and thallium results. These exceedances did not have a significant impact on the risk assessment.
Summarize the effect of analytical technique issues on the risk assessment , if applicable.	With the possible exception of thallium any changes in analytical technique would not have achieved any greater usability of better detection limits of the analytical data.

DATA USABILITY WORKSHEET (continued)

Site: FSPs 2 and 3

Medium: Surface water and sediment

Requirement	**Comment**
Data Quality Objectives	
Precision - How were duplicates handled?	Typically, one duplicate sample was collected for each group of 10 samples of a similar matrix type. Additional duplicate samples were collected for analyses as MS/MSDs to determine matrix effects. RPD values were calculated to evaluate precision.
Accuracy - How were split samples handled?	Accuracy was evaluated using calculated percent recoveries from surrogates and matrix spikes as applicable, specified in the analytical methods and the QAPP.
Representativeness - Indicate any problems associated with data representativeness (e.g., trip blank or rinseate blank contamination, COC problems, etc.).	Assessment of the quality control indicators for representativeness revealed no appreciable adverse impact on the data.
Completeness - Indicate any problems associated with data completeness (e.g., incorrect sample analysis, incomplete sample records, problems with field procedures, etc.).	Completeness goals were achieved by obtaining samples required at each location with a sufficient volume to complete the specified analyses. No problems with the QAPP specified goals were identified.
Comparability - Indicate any problems associated with data comparability.	As the Quality of the data from this study have been sufficiently established the data are well suited for comparison with previous or future data sets.
Were the DQOs specified in the QAPP satisfied?	Overall, the data set for this sampling event met the acceptance criteria and DQOs specified in the QAPP.
Summarize the effect of DQO issues on the risk assessment, if applicable.	In general, with the exception of a few detection limit exceedances for antimony, arsenic and thallium, all of the DQOs for this field sampling plan were met as specified in the QAPP. Therefore, the risk assessment was not adversely affected by any DQO issues.

DATA USABILITY WORKSHEET (continued)

Site: FSPs 2 and 3

Medium: Surface water and sediment

Requirement	Comment
Data Validation and Interpretation	
What are the data validation requirements for this region?	The data were validated using the technical guidelines and criteria specified in the individual analytical methods and the USEPA Functional Guidelines for Data Validation.
What method or guidance was used to validate the data?	The criteria set forth in the individual CLP, SW846, or water/waste water methods along with the USEPA Functional Guidelines for Data Validation were used, as applicable, to validate the data.
Was the data validation method consistent with regional guidance? Discuss any discrepancies.	The data validation was consistent with regional guidance and no discrepancies were noted.
Were all data qualifiers defined? Discuss those which were not.	All of the data qualifiers used were defined in the USEPA Functional Guidelines for Data Validation.
Which qualifiers represent usable data?	As specified in the USEPA Functional Guidelines, all data qualifiers, with the exception of "R", represent usable data.
Which qualifiers represent unusable data?	As specified in the USEPA Functional Guidelines, the data qualifier, "R", represents rejected and unusable data..
How are tentatively identified compounds handled?	None of the methods used to analyze this data set report tentatively identified compounds.

DATA USABILITY WORKSHEET (continued)

Site: FSPs 2 and 3

Medium: Surface water and sediment

Requirement	**Comment**
Summarize the effect of data validation and interpretation issues on the risk assessment, if applicable.	Thallium in sediment was not selected as a COPC and the majority of the detection levels exceeded the screening values; however, no impacts to risk assessment conclusions are anticipated, see Section 7.2 and Table 2-2 in the report.
Additional notes:	

Note: The purpose of this Worksheet is to succinctly summarize the data usability analysis and conclusions. Reference specific pages in the Risk Assessment text to further expand on the information presented here.

DATA USABILITY WORKSHEET

Site: Field Sampling Plan 4

Medium: Surface water in Canyon, Ninemile, Big, Moon, Beaver and Pine Creeks and the South Fork of the CdA River.

Requirement	Comment
Field Sampling	
Discuss sampling problems and field conditions that affect data usability.	No substantial sampling problems or adverse field conditions that might affect data usability were noted.
Are samples representative of receptor exposure for this medium (e.g. sample depth, grab vs composite, filtered vs unfiltered, low flow, etc.)?	Samples were collected of unfiltered water at high water flow. Sample locations were not selected based on human use patterns.
Assess the effect of filed QC results on data usability.	Although small concentrations of several common analytes were found in some of the equipment rinseate and field blanks, the overall quality of the data were determined not to be adversely affected.
Summarize the effect of field sampling issues on the risk assessment, if applicable.	No appreciable field sampling issues were identified that might have any impact on the quality of the risk assessment.
Analytical Techniques	
Were the analytical methods appropriate for quantitative risk assessment?	Overall, the methods chosen were appropriate and adequate to provide useable data for a quantitative risk assessment.
Were detection limits adequate?	QAPP specified detection limit goals were met with the exception of some antimony, arsenic, and thallium results. These exceedances did not have a significant impact on the risk assessment.
Summarize the effect of analytical technique issues on the risk assessment , if applicable.	With the possible exception of thallium any changes in analytical technique would not have achieved any greater usability of better detection limits of the analytical data.

DATA USABILITY WORKSHEET (continued)

Site: FSP 4

Medium: Surface water

Requirement	Comment
Data Quality Objectives	
Precision - How were duplicates handled?	Typically, one duplicate sample was collected for each group of 10 samples of a similar matrix type. Additional duplicate samples were collected for analyses as MS/MSDs to determine matrix effects. RPD values were calculated to evaluate precision.
Accuracy - How were split samples handled?	Accuracy was evaluated using calculated percent recoveries from surrogates and matrix spikes as applicable, specified in the analytical methods and the QAPP.
Representativeness - Indicate any problems associated with data representativeness (e.g., trip blank or rinseate blank contamination, COC problems, etc.).	Assessment of the quality control indicators for representativeness revealed no appreciable adverse impact on the data.
Completeness - Indicate any problems associated with data completeness (e.g., incorrect sample analysis, incomplete sample records, problems with field procedures, etc.).	Completeness goals were achieved by obtaining samples required at each location with a sufficient volume to complete the specified analyses. No problems with the QAPP specified goals were identified.
Comparability - Indicate any problems associated with data comparability.	As the Quality of the data from this study have been sufficiently established the data are well suited for comparison with previous or future data sets.
Were the DQOs specified in the QAPP satisfied?	Overall, the data set for this sampling event met the acceptance criteria and DQOs specified in the QAPP.
Summarize the effect of DQO issues on the risk assessment, if applicable.	In general, with the exception of a few detection limit exceedances for antimony, arsenic and thallium, all of the DQOs for this field sampling plan were met as specified in the QAPP. Therefore, the risk assessment was not adversely affected by any DQO issues.

DATA USABILITY WORKSHEET (continued)

Site: FSP 4

Medium: Surface water

Requirement	Comment
Data Validation and Interpretation	
What are the data validation requirements for this region?	The data were validated using the technical guidelines and criteria specified in the individual analytical methods and the USEPA Functional Guidelines for Data Validation.
What method or guidance was used to validate the data?	The criteria set forth in the individual CLP, SW846, or water/waste water methods along with the USEPA Functional Guidelines for Data Validation were used, as applicable, to validate the data.
Was the data validation method consistent with regional guidance? Discuss any discrepancies.	The data validation was consistent with regional guidance and no discrepancies were noted.
Were all data qualifiers defined? Discuss those which were not.	All of the data qualifiers used were defined in the USEPA Functional Guidelines for Data Validation.
Which qualifiers represent usable data?	As specified in the USEPA Functional Guidelines, all data qualifiers, with the exception of "R", represent usable data.
Which qualifiers represent unusable data?	As specified in the USEPA Functional Guidelines, the data qualifier, "R", represents rejected and unusable data..
How are tentatively identified compounds handled?	None of the methods used to analyze this data set report tentatively identified compounds.

DATA USABILITY WORKSHEET (continued)

Site: FSP 4

Medium: Surface water

Requirement	**Comment**
Summarize the effect of data validation and interpretation issues on the risk assessment, if applicable.	none
Additional notes:	

Note: The purpose of this Worksheet is to succinctly summarize the data usability analysis and conclusions. Reference specific pages in the Risk Assessment text to further expand on the information presented here.

DATA USABILITY WORKSHEET

Site: Field Sampling Plan 5

Medium: Soil, sediment, surface and drinking water, Common Use Areas, primarily beaches and upland parks

Requirement	Comment
Field Sampling	
Discuss sampling problems and field conditions that affect data usability.	No substantial sampling problems or adverse field conditions that might affect data usability were noted.
Are samples representative of receptor exposure for this medium (e.g. sample depth, grab vs composite, filtered vs unfiltered, low flow, etc.)?	Sample locations selected based on human use pattterns. Soil/sediment samples were surface composits and were sieved. Water samples were unfiltered and surface water samples were of "disturbed water" containing suspended sediments.
Assess the effect of filed QC results on data usability.	Although small concentrations of several common analytes were found in some of the equipment rinseate and field blanks, the overall quality of the data were determined not to be adversely affected.
Summarize the effect of field sampling issues on the risk assessment, if applicable.	No appreciable field sampling issues were identified that might have any impact on the quality of the risk assessment.
Analytical Techniques	
Were the analytical methods appropriate for quantitative risk assessment?	Overall, the methods chosen were appropriate and adequate to provide useable data for a quantitative risk assessment.
Were detection limits adequate?	QAPP specified detection limit goals were met with the exception of some antimony, arsenic, and thallium results. These exceedances did not have a significant impact on the risk assessment.
Summarize the effect of analytical technique issues on the risk assessment , if applicable.	With the possible exception of thallium any changes in analytical technique would not have achieved any greater usability of better detection limits of the analytical data.

DATA USABILITY WORKSHEET (continued)

Site: FSP 5

Medium: Soil, sediment, surface and drinking water

Requirement	Comment
Data Quality Objectives	
Precision - How were duplicates handled?	Typically, one duplicate sample was collected for each group of 10 samples of a similar matrix type. Additional duplicate samples were collected for analyses as MS/MSDs to determine matrix effects. RPD values were calculated to evaluate precision.
Accuracy - How were split samples handled?	Accuracy was evaluated using calculated percent recoveries from surrogates and matrix spikes as applicable, specified in the analytical methods and the QAPP.
Representativeness - Indicate any problems associated with data representativeness (e.g., trip blank or rinseate blank contamination, COC problems, etc.).	Assessment of the quality control indicators for representativeness revealed no appreciable adverse impact on the data.
Completeness - Indicate any problems associated with data completeness (e.g., incorrect sample analysis, incomplete sample records, problems with field procedures, etc.).	Completeness goals were achieved by obtaining samples required at each location with a sufficient volume to complete the specified analyses. No problems with the QAPP specified goals were identified.
Comparability - Indicate any problems associated with data comparability.	As the Quality of the data from this study have been sufficiently established the data are well suited for comparison with previous or future data sets.
Were the DQOs specified in the QAPP satisfied?	Overall, the data set for this sampling event met the acceptance criteria and DQOs specified in the QAPP.
Summarize the effect of DQO issues on the risk assessment, if applicable.	In general, with the exception of a few detection limit exceedances for antimony, arsenic and thallium, all of the DQOs for this field sampling plan were met as specified in the QAPP. Therefore, the risk assessment was not adversely affected by any DQO issues.

DATA USABILITY WORKSHEET (continued)

Site: FSP 5

Medium: Soil, sediment, surface and drinking water

Requirement	Comment
Data Validation and Interpretation	
What are the data validation requirements for this region?	The data were validated using the technical guidelines and criteria specified in the individual analytical methods and the USEPA Functional Guidelines for Data Validation.
What method or guidance was used to validate the data?	The criteria set forth in the individual CLP, SW846, or water/waste water methods along with the USEPA Functional Guidelines for Data Validation were used, as applicable, to validate the data.
Was the data validation method consistent with regional guidance? Discuss any discrepancies.	The data validation was consistent with regional guidance and no discrepancies were noted.
Were all data qualifiers defined? Discuss those which were not.	All of the data qualifiers used were defined in the USEPA Functional Guidelines for Data Validation.
Which qualifiers represent usable data?	As specified in the USEPA Functional Guidelines, all data qualifiers, with the exception of "R", represent usable data.
Which qualifiers represent unusable data?	As specified in the USEPA Functional Guidelines, the data qualifier, "R", represents rejected and unusable data..
How are tentatively identified compounds handled?	None of the methods used to analyze this data set report tentatively identified compounds.

DATA USABILITY WORKSHEET (continued)

Site: FSP 5

Medium: Soil, sediment, surface and drinking water

Requirement	**Comment**
Summarize the effect of data validation and interpretation issues on the risk assessment, if applicable.	Thallium in soil/sediment was not selected as a COPC and the majority of the samples were non-detects and the majority of the detection limits exceeded the screening values; however, no impacts to risk assessment conclusions are anticipated, see Section 7.2 and Table 2-2 in the report.
Additional notes:	

Note: The purpose of this Worksheet is to succinctly summarize the data usability analysis and conclusions. Reference specific pages in the Risk Assessment text to further expand on the information presented here.

DATA USABILITY WORKSHEET

Site: Field Sampling Plan 6 and 7 - Residential Sampling

Medium: Soil, house dust, tap water and garden vegetables

Requirement	Comment
Field Sampling	
Discuss sampling problems and field conditions that affect data usability.	No substantial sampling problems or adverse field conditions that might affect data usability were noted.
Are samples representative of receptor exposure for this medium (e.g. sample depth, grab vs composite, filtered vs unfiltered, low flow, etc.)?	Home yard soil: composites and discrete surface sampling. Samples sieved to <175 um. House dust sampled by mats and vacuum bags. Tap water: first draw and flushed, unfiltered.
Assess the effect of filed QC results on data usability.	Although small concentrations of several common analytes were found in some of the equipment rinseate and field blanks, the overall quality of the data were determined not to be adversely affected.
Summarize the effect of field sampling issues on the risk assessment, if applicable.	No appreciable field sampling issues were identified that might have any impact on the quality of the risk assessment.
Analytical Techniques	
Were the analytical methods appropriate for quantitative risk assessment?	Overall, the methods chosen were appropriate and adequate to provide useable data for a quantitative risk assessment.
Were detection limits adequate?	QAPP specified detection limit goals were met with the exception of some antimony, arsenic, and thallium results. These exceedances did not have a significant impact on the risk assessment.
Summarize the effect of analytical technique issues on the risk assessment , if applicable.	With the possible exception of thallium any changes in analytical technique would not have achieved any greater usability of better detection limits of the analytical data.

DATA USABILITY WORKSHEET (continued)

Site: FSPs 6 and 7

Medium: Soil, house dust, tap water and garden vegetables

Requirement	Comment
Data Quality Objectives	
Precision - How were duplicates handled?	Typically, one duplicate sample was collected for each group of 10 samples of a similar matrix type. Additional duplicate samples were collected for analyses as MS/MSDs to determine matrix effects. RPD values were calculated to evaluate precision.
Accuracy - How were split samples handled?	Accuracy was evaluated using calculated percent recoveries from surrogates and matrix spikes as applicable, specified in the analytical methods and the QAPP.
Representativeness - Indicate any problems associated with data representativeness (e.g., trip blank or rinseate blank contamination, COC problems, etc.).	Assessment of the quality control indicators for representativeness revealed no appreciable adverse impact on the data.
Completeness - Indicate any problems associated with data completeness (e.g., incorrect sample analysis, incomplete sample records, problems with field procedures, etc.).	Completeness goals were achieved by obtaining samples required at each location with a sufficient volume to complete the specified analyses. No problems with the QAPP specified goals were identified.
Comparability - Indicate any problems associated with data comparability.	As the Quality of the data from this study have been sufficiently established the data are well suited for comparison with previous or future data sets.
Were the DQOs specified in the QAPP satisfied?	Overall, the data set for this sampling event met the acceptance criteria and DQOs specified in the QAPP.
Summarize the effect of DQO issues on the risk assessment, if applicable.	In general, with the exception of a few detection limit exceedances for antimony, arsenic and thallium, all of the DQOs for this field sampling plan were met as specified in the QAPP. Therefore, the risk assessment was not adversely affected by any DQO issues.

DATA USABILITY WORKSHEET (continued)

Site: FSPs 6 and 7

Medium: Soil, house dust, tap water and garden vegetables

Requirement	Comment
Data Validation and Interpretation	
What are the data validation requirements for this region?	The data were validated using the technical guidelines and criteria specified in the individual analytical methods and the USEPA Functional Guidelines for Data Validation.
What method or guidance was used to validate the data?	The criteria set forth in the individual CLP, SW846, or water/waste water methods along with the USEPA Functional Guidelines for Data Validation were used, as applicable, to validate the data.
Was the data validation method consistent with regional guidance? Discuss any discrepancies.	The data validation was consistent with regional guidance and no discrepancies were noted.
Were all data qualifiers defined? Discuss those which were not.	All of the data qualifiers used were defined in the USEPA Functional Guidelines for Data Validation.
Which qualifiers represent usable data?	As specified in the USEPA Functional Guidelines, all data qualifiers, with the exception of "R", represent usable data.
Which qualifiers represent unusable data?	As specified in the USEPA Functional Guidelines, the data qualifier, "R", represents rejected and unusable data..
How are tentatively identified compounds handled?	None of the methods used to analyze this data set report tentatively identified compounds.

DATA USABILITY WORKSHEET (continued)

Site: FSPs 6 and 7

Medium: Soil, house dust, tap water and garden vegetables

Requirement	**Comment**
Summarize the effect of data validation and interpretation issues on the risk assessment, if applicable.	Thallium in soil was not selected as a COPC and the majority of the samples were non-detects and the majority of the detection limits exceeded the screening values; however, no impacts to risk assessment conclusions are anticipated, see Section 7.2 and Table 2-2 in the report. Arsenic in tap water was selected as a COPC, and about half the samples were non-detects. All the detection limits exceeded the screening value which is not technically feasible. Consequently, arsenic concentrations in tap water may be either under or overestimated depending on the actual concentration of the non-detected samples. No impacts to risk assessment conclusions are anticipated, see section 2.5 and Table 2-2 in the report.
Additional notes:	

Note: The purpose of this Worksheet is to succinctly summarize the data usability analysis and conclusions. Reference specific pages in the Risk Assessment text to further expand on the information presented here.

DATA USABILITY WORKSHEET

Site: Field Sampling Plan 8

Medium: Groundwater, waste piles (soil) in Canyon and Ninemile Creeks

Requirement	Comment
Field Sampling	
Discuss sampling problems and field conditions that affect data usability.	No substantial sampling problems or adverse field conditions that might affect data usability were noted.
Are samples representative of receptor exposure for this medium (e.g. sample depth, grab vs composite, filtered vs unfiltered, low flow, etc.)?	Groundwater collected from monitoring wells in shallow aquifer near source areas. Not used as a human water source. Soil: surface soil composites 0-12" depth.
Assess the effect of filed QC results on data usability.	Although small concentrations of several common analytes were found in some of the equipment rinseate and field blanks, the overall quality of the data were determined not to be adversely affected.
Summarize the effect of field sampling issues on the risk assessment, if applicable.	No appreciable field sampling issues were identified that might have any impact on the quality of the risk assessment.
Analytical Techniques	
Were the analytical methods appropriate for quantitative risk assessment?	Overall, the methods chosen were appropriate and adequate to provide useable data for a quantitative risk assessment.
Were detection limits adequate?	QAPP specified detection limit goals were met with the exception of some antimony, arsenic, and thallium results. These exceedances did not have a significant impact on the risk assessment.
Summarize the effect of analytical technique issues on the risk assessment , if applicable.	With the possible exception of thallium any changes in analytical technique would not have achieved any greater usability of better detection limits of the analytical data.

DATA USABILITY WORKSHEET (continued)

Site: FSP 8

Medium: groundwater, waste piles (soil)

Requirement	Comment
Data Quality Objectives	
Precision - How were duplicates handled?	Typically, one duplicate sample was collected for each group of 10 samples of a similar matrix type. Additional duplicate samples were collected for analyses as MS/MSDs to determine matrix effects. RPD values were calculated to evaluate precision.
Accuracy - How were split samples handled?	Accuracy was evaluated using calculated percent recoveries from surrogates and matrix spikes as applicable, specified in the analytical methods and the QAPP.
Representativeness - Indicate any problems associated with data representativeness (e.g., trip blank or rinseate blank contamination, COC problems, etc.).	Assessment of the quality control indicators for representativeness revealed no appreciable adverse impact on the data.
Completeness - Indicate any problems associated with data completeness (e.g., incorrect sample analysis, incomplete sample records, problems with field procedures, etc.).	Completeness goals were achieved by obtaining samples required at each location with a sufficient volume to complete the specified analyses. No problems with the QAPP specified goals were identified.
Comparability - Indicate any problems associated with data comparability.	As the Quality of the data from this study have been sufficiently established the data are well suited for comparison with previous or future data sets.
Were the DQOs specified in the QAPP satisfied?	Overall, the data set for this sampling event met the acceptance criteria and DQOs specified in the QAPP.
Summarize the effect of DQO issues on the risk assessment, if applicable.	In general, with the exception of a few detection limit exceedances for antimony, arsenic and thallium, all of the DQOs for this field sampling plan were met as specified in the QAPP. Therefore, the risk assessment was not adversely affected by any DQO issues.

DATA USABILITY WORKSHEET (continued)

Site: FSP 8

Medium: groundwater, waste piles (soil)

Requirement	Comment
Data Validation and Interpretation	
What are the data validation requirements for this region?	The data were validated using the technical guidelines and criteria specified in the individual analytical methods and the USEPA Functional Guidelines for Data Validation.
What method or guidance was used to validate the data?	The criteria set forth in the individual CLP, SW846, or water/waste water methods along with the USEPA Functional Guidelines for Data Validation were used, as applicable, to validate the data.
Was the data validation method consistent with regional guidance? Discuss any discrepancies.	The data validation was consistent with regional guidance and no discrepancies were noted.
Were all data qualifiers defined? Discuss those which were not.	All of the data qualifiers used were defined in the USEPA Functional Guidelines for Data Validation.
Which qualifiers represent usable data?	As specified in the USEPA Functional Guidelines, all data qualifiers, with the exception of "R", represent usable data.
Which qualifiers represent unusable data?	As specified in the USEPA Functional Guidelines, the data qualifier, "R", represents rejected and unusable data..
How are tentatively identified compounds handled?	None of the methods used to analyze this data set report tentatively identified compounds.

DATA USABILITY WORKSHEET (continued)

Site: FSP 8

Medium: groundwater, waste piles (soil)

Requirement	**Comment**
Summarize the effect of data validation and interpretation issues on the risk assessment, if applicable.	Thallium in soil was not selected as a COPC and the majority of the samples were non-detects and the majority of the detection limits exceeded the screening values; however, no impacts to risk assessment conclusions are anticipated, see Section 7.2 and Table 2-2 in the report.
Additional notes:	

Note: The purpose of this Worksheet is to succinctly summarize the data usability analysis and conclusions. Reference specific pages in the Risk Assessment text to further expand on the information presented here.

DATA USABILITY WORKSHEET

Site: Field Sampling Plan 12 - Residential Sampling

Medium: Soil, tap water

Requirement	Comment
Field Sampling	
Discuss sampling problems and field conditions that affect data usability.	No substantial sampling problems or adverse field conditions that might affect data usability were noted.
Are samples representative of receptor exposure for this medium (e.g. sample depth, grab vs composite, filtered vs unfiltered, low flow, etc.)?	Soil: composites and discrete surface sampling. Samples sieved to <175 um Tap water: first draw and flused, unfiltered, non-municipal supplies only.
Assess the effect of filed QC results on data usability.	Although small concentrations of several common analytes were found in some of the equipment rinseate and field blanks, the overall quality of the data were determined not to be adversely affected.
Summarize the effect of field sampling issues on the risk assessment, if applicable.	No appreciable field sampling issues were identified that might have any impact on the quality of the risk assessment.
Analytical Techniques	
Were the analytical methods appropriate for quantitative risk assessment?	Overall, the methods chosen were appropriate and adequate to provide useable data for a quantitative risk assessment.
Were detection limits adequate?	QAPP specified detection limit goals were met with the exception of some antimony, arsenic, and thallium results. These exceedances did not have a significant impact on the risk assessment.
Summarize the effect of analytical technique issues on the risk assessment , if applicable.	With the possible exception of thallium any changes in analytical technique would not have achieved any greater usability of better detection limits of the analytical data.

DATA USABILITY WORKSHEET (continued)

Site: FSP 12

Medium: Soil, tap water

Requirement	Comment
Data Quality Objectives	
Precision - How were duplicates handled?	Typically, one duplicate sample was collected for each group of 10 samples of a similar matrix type. Additional duplicate samples were collected for analyses as MS/MSDs to determine matrix effects. RPD values were calculated to evaluate precision.
Accuracy - How were split samples handled?	Accuracy was evaluated using calculated percent recoveries from surrogates and matrix spikes as applicable, specified in the analytical methods and the QAPP.
Representativeness - Indicate any problems associated with data representativeness (e.g., trip blank or rinseate blank contamination, COC problems, etc.).	Assessment of the quality control indicators for representativeness revealed no appreciable adverse impact on the data.
Completeness - Indicate any problems associated with data completeness (e.g., incorrect sample analysis, incomplete sample records, problems with field procedures, etc.).	Completeness goals were achieved by obtaining samples required at each locatior with a sufficient volume to complete the specified analyses. No problems with the QAPP specified goals were identified.
Comparability - Indicate any problems associated with data comparability.	As the Quality of the data from this study have been sufficiently established, the data are well suited for comparison with previous or future data sets.
Were the DQOs specified in the QAPP satisfied?	Overall, the data set for this sampling event met the acceptance criteria and DQOs specified in the QAPP.
Summarize the effect of DQO issues on the risk assessment, if applicable.	In general, with the exception of a few detection limit exceedances for antimony, arsenic and thallium, all of the DQOs for this field sampling plan were met as specified in the QAPP. Therefore, the risk assessment was not adversely affected by any DQO issues.

DATA USABILITY WORKSHEET (continued)

Site: FSP 12

Medium: Soil, tap water

Requirement	Comment
Data Validation and Interpretation	
What are the data validation requirements for this region?	The data were validated using the technical guidelines and criteria specified in the individual analytical methods and the USEPA Functional Guidelines for Data Validation.
What method or guidance was used to validate the data?	The criteria set forth in the individual CLP, SW846, or water/waste water methods along with the USEPA Functional Guidelines for Data Validation were used, as applicable, to validate the data.
Was the data validation method consistent with regional guidance? Discuss any discrepancies.	The data validation was consistent with regional guidance and no discrepancies were noted.
Were all data qualifiers defined? Discuss those which were not.	All of the data qualifiers used were defined in the USEPA Functional Guidelines for Data Validation.
Which qualifiers represent usable data?	As specified in the USEPA Functional Guidelines, all data qualifiers, with the exception of "R", represent usable data.
Which qualifiers represent unusable data?	As specified in the USEPA Functional Guidelines, the data qualifier, "R", represents rejected and unusable data..
How are tentatively identified compounds handled?	None of the methods used to analyze this data set report tentatively identified compounds.

DATA USABILITY WORKSHEET (continued)

Site: FSP 12

Medium: Soil, tap water

Requirement	**Comment**
Summarize the effect of data validation and interpretation issues on the risk assessment, if applicable.	Thallium in soil was not selected as a COPC and the majority of the samples were non-detects and the majority of the detection limits exceeded the screening values; however, no impacts to risk assessment conclusions are anticipated, see Section 7.2 and Table 2-2 in the report. Arsenic in tap water was selected as a COPC, and about half the samples were non-detects. All the detection limits exceeded the screening value which is not technically feasible. Consequently, arsenic concentrations in tap water may be either under or overestimated depending on the actual concentration of the non-detected samples. No impacts to risk assessment conclusions are anticipated, see section 2.5 and Table 2-2 in the report.
Additional notes:	

Note: The purpose of this Worksheet is to succinctly summarize the data usability analysis and conclusions. Reference specific pages in the Risk Assessment text to further expand on the information presented here.

TABLE 1
SELECTION OF EXPOSURE PATHWAYS
Coeur d'Alene River Basin

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current	Tailing Deposits and Slag Piles (Soil)	Surface Water (a)	Stream and River Water	Tribal Recreational	Child/Adult	Ingestion Dermal	NA NA	Quant. Qual.	Children/adults may be in direct contact with surface water during intermittent recreational activities. Undisturbed surface water is the main source of drinking water for tribal scenarios.
			Stream and River Sediment	Tribal Recreational	Child/Adult	Ingestion Dermal	NA NA	Quant. Quant.	Children/adults may be in contact with impacted sediments during intermittent recreational activities (swimming and beach play) and tribal activities (fishing, water potato collection, swimming, etc.).
			Native Plants *	Tribal	Child/Adult	Ingestion	NA	Quant.	Consumption of water potatoes growing in surface water and sediments are evaluated for tribal scenarios.
			Cattle (b) *	Residential	Child/Adult	Ingestion	NA	Qual.	Children and adults eat potentially affected cattle that graze on grasses growing in impacted sediment.
			Wild Fowl (b) *	Tribal Recreational	Child/Adult	Ingestion	NA	Qual.	Children and adults hunt and eat potentially affected wild fowl that are found in floodplain.
			Fish from Lower Basin and Spokane River (c)	Tribal Recreational	Child/Adult	Ingestion	NA	Quant.	Children and adults may collect fish that are potentially affected by impacted surface water and sediments; therefore, this pathway will be quantitatively evaluated. The Tribe depends on fish from Lake Coeur d'Alene as a main food source.
		Surface Soil (d)	Surface Soil	Residential Tribal Recreational Occupational	Child/Adult	Ingestion Dermal	NA NA	Quant. Quant.	Children and adults may potentially be in direct contact with impacted surface soils during outdoor activities at their homes, neighborhoods, or parks; therefore, the ingestion and dermal pathways will be quantitatively evaluated. Outdoor workers could be affected by contaminated soils in their day-to-day activities.
			Vegetables *	Residential	Child/Adult	Ingestion	NA	Quant.	Children and adults eat vegetables from gardens potentially containing impacted soils; therefore, this pathway will be evaluated quantitatively. Tribal populations collect native plants growing in impacted soils.
			Native Plants *	Tribal	Child/Adult	Ingestion	NA	Qual.	
			Game (f) *	Tribal Recreational	Child/Adult	Ingestion	NA	Qual.	Game animals (e.g., deer, beaver, and muskrats), except for water fowl, are unlikely to contain significant levels of metals, see text.
			House Dust	Residential	Child/Adult	Ingestion Inhalation	NA	Quant. Qual.	Fugitive dust containing significant levels of metals may be deposited inside housing units and accumulate on funiture, draperies, horizontal window blinds, carpet, and any non-vertical surface (e.g. window sills, shelves, etc.).
		Groundwater (e)	Tap Water	Residential	Child/Adult	Ingestion Dermal	NA NA	Quant. Qual.	Residents currently use groundwater for drinking and for household activities.
		Air	Resuspended Particulates from Surface Soils	Residential Tribal Recreational	Child/Adult	Inhalation	NA	Qual.	The inhalation pathway is likely negligible at the site as compared to the ingestion and dermal contact pathways for soil.

TABLE 1
SELECTION OF EXPOSURE PATHWAYS
Coeur d'Alene River Basin

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future	Tailing Deposits and Slag Piles (Soil)	Groundwater Surface Soil	Subsurface Soil	Residential	Child/Adult	Ingestion Dermal Inhalation	NA NA NA	Quant. Quant. Qual.	If affected soils below ground surface remain undisturbed, exposures are not likely to occur. However, residents do garden in their yards, potentially digging in the soil. Only the ingestion and dermal pathways will be evaluated quantitatively, since the inhalation pathway is considered negligible.
				Occupational	Adult	Ingestion Dermal Inhalation	NA NA NA	Quant. Qual. Qual.	If affected soils below ground surface remain undisturbed, occupational exposures are likely to be minimal. The occupational exposure pathway is addressed using subsurface and surface soil data for all geographic areas under an intensive soil contact scenario; sediment in the Lower Basin is also used.
		Surface Water (a)	Stream and River Water	Tribal Recreational	Child/Adult	Ingestion Dermal	NA NA	Quant. Qual.	Children/adults may be in direct contact with surface water during intermittent recreational activities. Undisturbed surface water is the main source of drinking water for tribal scenarios.
			Stream and River Sediment	Tribal Recreational	Child/Adult	Ingestion Dermal	NA NA	Quant. Quant.	Children/adults may be in contact with impacted sediments during intermittent recreational activities (swimming and beach play) and tribal activities (fishing, water potato collection, swimming, etc.).
			Fish from Lower Basin and Spokane River (c)	Tribal Recreational	Child/Adult	Ingestion	NA	Quant.	Children and adults may collect fish that are potentially affected by impacted surface water and sediments; therefore, this pathway will be quantitatively evaluated. The Tribe depends on fish from Lake Coeur d'Alene as a main food source.
		Surface Soil (d)	Surface Soil	Residential Tribal Recreational Occupational	Child/Adult	Ingestion Dermal	NA NA	Quant. Quant.	Children and adults may potentially be in direct contact with impacted surface soils during outdoor activities at their homes, neighborhoods, or parks; therefore, the ingestion and dermal pathways will be quantitatively evaluated. Outdoor workers could be affected by contaminated soils in their day-to-day activities.
			House Dust	Residential	Child/Adult	Ingestion Inhalation	NA	Quant. Qual.	Fugitive dust containing significant levels of metals may be deposited inside housing units and accumulate on funiture, draperies, horizontal window blinds, carpet, and any non-vertical surface (e.g. window sills, shelves, etc.).
		Groundwater (e)	Tap Water	Residential	Child/Adult	Ingestion Dermal	NA NA	Quant. Qual.	Groundwater for future scenario is not currently being used as a drinking water source; groundwater identified under the current scenario is being used and will continue to be used.
		Air	Resuspended Particulates from Surface Soils	Residential Tribal Recreational	Child/Adult	Inhalation	NA	Qual.	The inhalation pathway is likely negligible at the site as compared to the ingestion and dermal contact pathways for soil.

NOTES * Pathway will also be evaluated under a future exposure scenario

NA = Not applicable to CdA site; Quant. = quantitative anlaysis in the risk assessment; Qual. = qualitative analysis in the risk assessment; SW = surface water

a) In addition to impacts from surface soil erosion / stormwater runoff / impacted sediment, surface water is also affected by surface seepage of the groundwater.

b) Cattle graze in floodplain on grasses that grow in contaminated sediment. Wild fowl, also found in floodplain, are hunted and eaten by people.

c) In addition to impacts from contaminated surface water, fish are also affected by contaminated sediments.

d) In addition to direct contact with tailing deposits and waste piles, other soils have been impacted by depositions from water- and air- transported materials.

e) In addition to impacts from soil leachate, groundwater is also affected by surface water infiltration.

f) Limited samples have been collected from a variety of terrestrial game animals, e.g., muskrat, beavers, and deer.

TABLE 2.1.1

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN SOIL/SEDIMENT

CSM Unit 1

Scenario Timeframe:	Current
Medium:	Soil/Sediment
Exposure Medium:	Soil/Sediment
Exposure Point:	Soil/Sediment (all depths)

CAS Number	Chemical	Minimum Concentration (1)	Minimum Qualifier	Maximum Concentration (1)	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value (2)	Screening Toxicity Value (3)	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for Contaminant Deletion or Selection (4)
	Inorganics															
7429-90-5	Aluminum	1380		38500		mg/kg	R035	768/768	--	38500	na	7500	na	na	NO	NUT / IFE
7440-36-0	***Antimony***	0.28	J	623		mg/kg	SegBIG04	576/758	0.42 - 9.08	623	5.8	3	na	na	YES	ASL
7440-38-2	***Arsenic***	1.6	J	3610		mg/kg	CC/Tiger	788/800	5.2 - 13.9	3610	22	0.38 c	na	na	YES	ASL
7440-39-3	Barium	16.6	J	2350		mg/kg	R040	768/768	--	2350	1100	520	na	na	NO	IFE
7440-41-7	Beryllium	0.0784	J	1.9		mg/kg	R065	554/768	0.12 - 1.4	1.9	2.1	15	na	na	NO	BSL
7440-43-9	***Cadmium***	0.09		194	J	mg/kg	SegNM04	758/800	0.06 - 2.8	194	2.86	3.7	na	na	YES	ASL
7440-70-2	Calcium	232	J	99200		mg/kg	R035	768/768	--	99200	1	NE	na	na	NO	NUT
7440-47-3	Chromium (5)	0.124	J	198		mg/kg	R065	768/768	--	198	64	210 c	na	na	NO	BSL
7440-48-4	Cobalt	1.2		60.5		mg/kg	R053	767/768	8.5	60.5	20	330	na	na	NO	BSL
7440-50-8	Copper	7.6	J	1710		mg/kg	R065	800/800	--	1710	53	280	na	na	NO	IFE
7439-89-6	***Iron***	3190		147000		mg/kg	R074	768/768	--	147000	65000	2200	na	na	YES	ASL
7439-92-1	***Lead***	17.6		67100		mg/kg	SegCC05	800/800	--	67100	175	400	na	na	YES	ASL
7439-95-4	Magnesium	274	J	9930	J	mg/kg	R080	768/768	--	9930	1.1	NE	na	na	NO	NUT
7439-96-5	***Manganese***	70.8		20200		mg/kg	R086	768/768	--	20200	3600	310	na	na	YES	ASL
7487-94-7	Mercury	0.0474	J	47.3	J	mg/kg	R086	676/800	0.0478 - 0.37	47.3	0.3	2.2	na	na	NO	IFE
7440-02-0	Nickel	1.9		148		mg/kg	R065	751/768	2.7 - 23.9	148	38	150	na	na	NO	BSL
7440-09-7	Potassium	221	J	7170	J	mg/kg	R064	768/768	--	7170	na	NE	na	na	NO	NUT
7782-49-2	Selenium	0.34	J	6.1	J	mg/kg	R085	279/750	0.2 - 2.11	6.1	na	37	na	na	NO	BSL
7440-22-4	Silver	0.226	J	347	J	mg/kg	SegCC02	690/766	0.14 - 3.9	347	1.1	37	na	na	NO	IFE
7440-23-5	Sodium	27.2	J	5690		mg/kg	CC/Tamarack7	754/768	20.7 - 257	5690	na	NE	na	na	NO	NUT
1314-32-5	Thallium (6)	0.24	J	14.4	J	mg/kg	SegUG01	175/768	0.39 - 9.8	14.4	na	0.52	na	na	NO	IFE
7440-62-2	Vanadium	1.7	J	76.7		mg/kg	R053	768/768	--	76.7	154	52	na	na	NO	IFE/BKG
7440-66-6	***Zinc***	32.6		25800		mg/kg	CC/Tamarack7	800/800	--	25800	280	2200	na	na	YES	ASL

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration.

(2) Background concentrations are the 90th percentile values from "Geochemical-Exploration Studies in the Coeur d'Alene District, Idaho and Montana" (Gott and Cathrall 1980).

(3) Screening toxicity values are 0.1 times the EPA Region 9 Residential Soil PRGs (EPA 1998b) for noncarcinogens. Screening toxicity values for carcinogens, and for lead, are unchanged from the listed soil PRG.

(4) Rationale Codes

Selection Reason:	HIST:	Infrequent Detection but Associated Historically
	FD:	Frequent Detection
	TX:	Toxicity Information Available
	ASL:	Above Screening Levels
Deletion Reason:	IFD:	Infrequent Detection - less than 5 percent
	BKG:	Background Levels
	NTX:	No Toxicity Information
	NUT:	Essential Nutrient
	BSL:	Below Screening Level
	NHIST:	Not Historically Associated with Source
	IFE:	Infrequent Exceedance of STV - less than 10 percent; or Infrequent Exceedance of PRG

(5) STV listed for Total Chromium (1/6 ratio Cr VI/Cr III); a carcinogen.

(6) STV listed for Thallic Oxide (more conservative).

Definitions:

na = not available
-- Compound has a 100% detection frequency
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
NE = Not Established
J = Estimated Value
c = Cancer endpoint
N/A = Not Applicable
PRG = Preliminary Remediation Goal
STV = Screening Toxicity Value

TABLE 2.1.2

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN FIRST-RUN TAP WATER

CSM Unit 1

Scenario Timeframe:	Current
Medium:	Groundwater (varies)
Exposure Medium:	Groundwater
Exposure Point:	First-Run Tap Water

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background (2) Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	23.9		165		ug/L	R050	6/29	22 - 69.5	165	na	3700	50-200	SMCL	NO	BSL/NUT
7440-36-0	Antimony	2.1		7.5		ug/L	R039	3/29	0.049 - 0.82	7.5	na	1.5	6	MCL	NO	HWS / IFE
7440-38-2	***Arsenic***	0.24		7.6		ug/L	R085	10/29	0.2 - 0.79	7.6	na	0.045 c	50	MCL	YES	ASL
7440-39-3	Barium	20.5		71.9		ug/L	R105	10/29	7.2 - 30.5	71.9	na	260	2000	MCL	NO	BSL
7440-41-7	Beryllium	< 0.2	U	< 1	U	ug/L	N/A	0/29	0.2 - 1	< 1	na	7.3	4	MCL	NO	BSL/IFD
7440-43-9	Cadmium	0.1		1.8		ug/L	R042	13/29	0.079 - 1	1.8	na	1.8	5	MCL	NO	BSL/LMCL
7440-70-2	Calcium	4150		46700		ug/L	R105	29/29	--	46700	na	NE	NE	NE	NO	NUT
7440-47-3	Chromium (5)	0.1		0.58		ug/L	R065	13/29	0.058 - 5	0.58	na	18	100	MCL	NO	BSL
7440-48-4	Cobalt	0.12		0.64		ug/L	R037	5/29	0.037 - 5	0.64	na	220	NE	NE	NO	BSL
7440-50-8	Copper	5.7		610		ug/L	R054	29/29	--	610	na	140	1300 / 1000	MCL / SMCL	NO	LMCL/HWS
7439-89-6	Iron	31.4		761		ug/L	R042	8/29	8 - 116	761	na	1100	300	SMCL	NO	BSL/NUT
7439-92-1	***Lead***	0.28		12.4		ug/L	R042	29/29	--	12.4	na	4	15	MCL	YES	ASL
7439-95-4	Magnesium	602		19700		ug/L	R105	29/29	--	19700	na	NE	NE	NE	NO	NUT
7439-96-5	Manganese	1.5		10.7		ug/L	R050	5/29	1.3 - 10.3	10.7	na	170	50	SMCL	NO	BSL
7487-94-7	Mercury	< 0.1	U	< 0.2	U	ug/L	N/A	0/29	0.1 - 0.2	< 0.2	na	1.1	2	MCL	NO	BSL/IFD
7440-02-0	Nickel	6		6		ug/L	R054	1/29	4.4 - 5.7	6	na	73	140	MCL	NO	BSL/IFD
7440-09-7	Potassium	186		3520		ug/L	R105	28/29	115	3520	na	NE	NE	NE	NO	NUT
7782-49-2	Selenium	0.4		0.4		ug/L	R088	1/29	0.34 - 1	0.4	na	18	50	MCL	NO	BSL/IFD
7440-22-4	Silver	< 4.3	U	< 8	U	ug/L	N/A	0/29	4.3 - 8	< 8	na	18	100	SMCL	NO	BSL/IFD
7440-23-5	Sodium	403		22600		ug/L	R085	29/29	--	22600	na	NE	NE	NE	NO	NUT
1314-32-5	Thallium (6)	< 0.03	U	< 0.2	U	ug/L	N/A	0/29	0.03 - 0.2	< 0.2	na	0.26	2	MCL	NO	BSL/IFD
7440-62-2	Vanadium	0.13		0.13		ug/L	R050	1/29	0.053 - 5	0.13	na	26	NE	NE	NO	BSL/IFD
7440-66-6	Zinc	20.6		685		ug/L	R038	29/29	--	685	na	1100	5000	SMCL	NO	BSL

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration

(2) na - not available

(3) Screening toxicity values are 0.1 times the EPA Region 9 Tap Water PRGs (EPA 1998b) for noncarcinogens (except for lead).
Screening toxicity values for carcinogens are unchanged from the listed Tap Water PRG.

(4) Rationale Codes

Selection Reason:	HIST -	Infrequent Detection but Associated Historically
	FD -	Frequent Detection
	TX -	Toxicity Information Available
	ASL -	Above Screening Levels
Deletion Reason:	IFD -	Infrequent Detection - less than 5 percent
	BKG -	Background Levels
	NTX -	No Toxicity Information
	NUT -	Essential Nutrient
	BSL -	Below Screening Level
	NHIST -	Not Historically Associated with Source
	IFE -	Infrequent Exceedance - less than 10 percent of STV or infrequent exceedance of PRG
	LMCL -	Less Than MCL
	LSMCL -	Less Than Secondary MCL
	HWS -	Home Water Supply is currently being addressed

(5) Tap water STV listed for Chromium VI; no tap water PRG established for Total Chromium

(6) STV listed for Thallic Oxide (more conservative)

Definitions:

< - less than the laboratory method detection limit listed (non-detect)
N/A - Not Applicable
-- Compound has a 100% detection frequency
COPC - Chemical of Potential Concern
ARAR/TBC - Applicable or Relevant and Appropriate Requirement/To Be Considered
MCL - Federal Maximum Contaminant Level
SMCL - Secondary Maximum Contaminant Level
NE - Not Established
U - Compound was analyzed for, but not detected
J - Estimated Value
c - Cancer endpoint
PRG = Preliminary Remediation Goal
STV = Screening Toxicity Value

TABLE 2.1.3

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN FLUSHED TAP WATER

CSM Unit 1

Scenario Timeframe	Current
Medium:	Groundwater (varies)
Exposure Medium:	Groundwater
Exposure Point:	Flushed Tap Water

CAS Number	Chemical	Minimum Concentration (1)	Minimum Qualifier	Maximum Concentration (1)	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value (2)	Screening Toxicity Value (3)	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for Contaminant Deletion or Selection (4)
	Inorganics															
7429-90-5	Aluminum	< 22	U	< 96.1	U	ug/L	N/A	0/29	22 - 96.1	< 96.1	na	3700	50-200	SMCL	NO	NUT/BSL/IFD
7440-36-0	Antimony	1.3		7.9		ug/L	R039	3/29	0.049 - 0.99	7.9	na	1.5	6	MCL	NO	HWS / IFE
7440-38-2	***Arsenic***	0.21		9.2		ug/L	R085	10/29	0.2 - 0.69	9.2	na	0.045 c	50	MCL	YES	ASL
7440-39-3	Barium	20.9		71.2		ug/L	R105	10/29	7.6 - 29.6	71.2	na	260	2000	MCL	NO	BSL
7440-41-7	Beryllium	< 0.2	U	< 1	U	ug/L	N/A	0/29	0.2 - 1	< 1	na	7.3	4	MCL	NO	BSL / IFD
7440-43-9	Cadmium	0.1		0.72		ug/L	R088	5/29	0.079 - 1	0.72	na	1.8	5	MCL	NO	BSL
7440-70-2	Calcium	4150		46400		ug/L	R105	29/29	--	46400	na	NE	NE	NE	NO	NUT
7440-47-3	Chromium (5)	0.1		0.36		ug/L	R086	15/29	0.058 - 5	0.36	na	18	100	MCL	NO	BSL
7440-48-4	Cobalt	< 0.037	U	< 5	U	ug/L	N/A	0/29	0.037 - 5	< 5	na	220	NE	NE	NO	BSL / IFD
7440-50-8	Copper	1.3	J	46.5		ug/L	R054	25/29	0.087 - 5.7	46.5	na	140	1300 / 1000	MCL / SMCL	NO	BSL
7439-89-6	Iron	29.6		128		ug/L	R105	3/29	8 - 86.6	128	na	1100	300	SMCL	NO	BSL / NUT
7439-92-1	***Lead***	0.13		9.5		ug/L	R105	26/29	0.04	9.5	na	4	15	MCL	YES	ASL
7439-95-4	Magnesium	607		19600		ug/L	R105	29/29	--	19600	na	NE	NE	NE	NO	NUT
7439-96-5	Manganese	1.3		22.1		ug/L	R085	3/29	1.3 - 6.9	22.1	na	170	50	SMCL	NO	BSL
7487-94-7	Mercury	< 0.1	U	< 0.2	U	ug/L	N/A	0/29	0.1 - 0.2	< 0.2	na	1.1	2	MCL	NO	BSL / IFD
7440-02-0	Nickel	5.6		5.6		ug/L	R085	1/29	4.4 - 5	5.6	na	73	140	MCL	NO	BSL / IFD
7440-09-7	Potassium	148		3440		ug/L	R105	28/29	115	3440	na	NE	NE	NE	NO	NUT
7782-49-2	Selenium	0.33		2.6		ug/L	R086	2/29	0.36 - 1	2.6	na	18	50	MCL	NO	BSL
7440-22-4	Silver	< 4.3	U	< 6.1	U	ug/L	N/A	0/29	4.3 - 6.1	< 6.1	na	18	100	SMCL	NO	BSL / IFD
7440-23-5	Sodium	456		22100		ug/L	R085	29/29	--	22100	na	NE	NE	NE	NO	NUT
1314-32-5	Thallium (6)	0.3		2.3		ug/L	R074	4/29	0.03 - 0.13	2.3	na	0.26	2	MCL	NO	HWS / IFE
7440-62-2	Vanadium	< 0.053	U	< 5	U	ug/L	N/A	0/29	0.053 - 5	< 5	na	26	NE	NE	NO	BSL / IFD
7440-66-6	Zinc	5.4		214		ug/L	R088	28/29	8.2	214	na	1100	5000	SMCL	NO	BSL

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration

(2) na - not available

(3) Screening toxicity values are 0.1 times the EPA Region 9 Tap Water PRGs (EPA 1998b) for noncarcinogens (except for lead).
Screening toxicity values for carcinogens are unchanged from the listed Tap Water PRG.

(4) Rationale Codes

Selection Reason:	HIST -	Infrequent Detection but Associated Historically
	FD -	Frequent Detection
	TX -	Toxicity Information Available
	ASL -	Above Screening Levels
Deletion Reason:	IFD -	Infrequent Detection - less than 5 percent
	BKG -	Background Levels
	NTX -	No Toxicity Information
	NUT -	Essential Nutrient
	BSL -	Below Screening Level
	NHIST -	Not Historically Associated with Source
	IFE -	Infrequent Exceedance - less than 10 percent of STV or infrequent exceedance of PRG
	LMCL -	Less Than MCL
	LSMCL -	Less Than Secondary MCL
	HWS -	Home Water Supply is currently being addressed

(5) Tap water STV listed for Chromium VI, no PRG established for Total Chromium.

(6) STV listed for Thallic Oxide (more conservative).

Definitions:

< - less than the laboratory method detection limit listed (non-detect)
N/A - Not Applicable
-- Compound has a 100% detection frequency
COPC - Chemical of Potential Concern
ARAR/TBC - Applicable or Relevant and Appropriate Requirement/To Be Considered
MCL - Federal Maximum Contaminant Level
SMCL - Secondary Maximum Contaminant Level
NE - Not Established
U - Compound was analyzed for, but not detected
J - Estimated Value
c - Cancer endpoint
PRG = Preliminary Remediation Goal
STV = Screening Toxicity Value

TABLE 2.1.4

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN SURFACE WATER

CSM Unit 1

Scenario Timeframe:	Current
Medium:	Soil/Groundwater
Exposure Medium:	Surface Water
Exposure Point:	Rivers & Streams (2)

CAS Number	Chemical	Minimum (1) Conc.	Minimum Qualifier	Maximum (1) Conc.	Maximum Qualifier	Units	Location of Maximum Conc.	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background (3) Value	Screening (4) Toxicity Value	Potential ARAR/TBC Value (5)	Potential ARAR/TBC Source	COPC Flag	Rationale for (6) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	3.7		7580	J	ug/L	SegNM02	49/176	11.8 - 160	7580	na	NE	50 - 200	SMCL	NO	NUT
7440-36-0	Antimony	0.2		19		ug/L	SegUG01	72/176	0.032 - 2	19	0.51	NE	6 / 4300	MCL/AWQC	NO	LAWQC / IFE
7440-38-2	***Arsenic***	0.1		10.9		ug/L	SegNM02	59/176	0.16 - 2	10.9	0.65	NE	50 / 0.14	MCL/AWQC	YES	AAWQC
7440-39-3	Barium	1.3		370		ug/L	SegUG01	168/176	3 - 30.3	370	na	NE	2000	MCL	NO	LMCL
7440-41-7	Beryllium	0.39	J	2.4		ug/L	SegNM02	3/176	0.1 - 1	2.4	na	NE	4	MCL	NO	LMCL / IFD
7440-43-9	***Cadmium***	0.16	J	1810	J	ug/L	SegNM02	105/176	0.041 - 0.29	1810	0.09	NE	5	MCL	YES	AMCL
7440-70-2	Calcium	551	J	111000		ug/L	SegUG01	175/176	6650	111000	na	NE	na	na	NO	NUT
7440-47-3	Chromium	5		8.9	J	ug/L	SegNM02	7/176	0.4 - 6	8.9	na	NE	100	MCL	NO	LMCL / IFD
7440-48-4	Cobalt	0.023	J	178		ug/L	SegNM02	11/176	0.023 - 10	178	na	NE	na	na	NO	IFD
7440-50-8	Copper	0.16	J	340		ug/L	SegNM02	83/176	0.06 - 3	340	1.21	NE	1300	MCL	NO	LMCL
7439-89-6	Iron	6.7	J	16600		ug/L	SegNM02	107/176	5 - 106	16600	113	NE	300	SMCL	NO	NUT / IFE
7439-92-1	***Lead***	0.097	J	1650		ug/L	SegNM02	159/176	0.026 - 0.5	1650	1.46	NE	15	MCL	YES	AMCL
7439-95-4	Magnesium	125	J	72200		ug/L	SegUG01	176/176	--	72200	na	NE	na	na	NO	NUT
7439-96-5	***Manganese***	0.4		26800		ug/L	SegNM02	128/176	1 - 23.5	26800	8.28	NE	50 / 100	SMCL/AWQC	YES	ASMCL/AAWQC
7487-94-7	Mercury	0.13	J	0.32		ug/L	SegUG01	6/176	0.1 - 0.35	0.32	0.09	NE	2 / 0.051 (7)	MCL/AWQC	NO	LMCL / IFD
7440-02-0	Nickel	0.26	J	306		ug/L	SegNM02	17/176	0.22 - 20	306	na	NE	140 / 4600	MCL/AWQC	NO	LAWQC / IFE
7440-09-7	Potassium	193	J	8020		ug/L	SegNM02	112/176	200 - 2000	8020	na	NE	na	na	NO	NUT
7782-49-2	Selenium	0.22	J	13.5		ug/L	SegNM02	14/176	0.21 - 2	13.5	na	NE	50 / 11000	MCL / AWQC	NO	LMCL / LAWQC
7440-22-4	Silver	0.61		7.7		ug/L	SegUG01	5/176	0 - 0.35	7.7	0.12	NE	100	SMCL	NO	LSMCL / IFD
7440-23-5	Sodium	392	J	16200		ug/L	SegBIG04	176/176	--	16200	na	NE	na	na	NO	NUT
7440-28-0	Thallium	0.27	J	0.92	J	ug/L	SegUG01	3/176	0.005 - 0.47	0.92	na	NE	2 / 6.3	MCL/AWQC	NO	LMCL/LAWQC/IFD
7440-62-2	Vanadium	0.14		2.5		ug/L	SegUG01	9/174	0.1 - 10	2.5	na	NE	na	na	NO	IFD
7440-66-6	Zinc	0.94	J	540000		ug/L	SegNM02	121/176	5 - 31.8	540000	20.71	NE	5000 / 69000	SMCL/AWQC	NO	IFE

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration.

(2) Data from the Upper South Fork of the CdA River, Canyon Creek, Ninemile Creek, Big Creek, Moon Creek, Beaver Creek, and Pine Creek.

(3) Background values as calculated by URS, March 2000.

(4) Screening toxicity values not established (NE).

(5) Potential ARARs are the Ambient Water Quality Criteria, human health for consumption of organisms only. EPA 1998c (Vol.63, No.237)

(6) Rationale Codes

Selection Reason:

HIST:	Infrequent Detection but Associated Historically
FD:	Frequent Detection
TX:	Toxicity Information Available
ASL:	Above Screening Levels
AMCL:	Above MCL
ASMCL:	Above Secondary MCL
AAWQC:	Above Ambient Water Quality Criteria

Deletion Reason:

IFD:	Infrequent Detection - less than 10 percent
BKG:	Background Levels
NTX:	No Toxicity Information
NUT:	Essential Nutrient
BSL:	Below Screening Level
NHIST:	Not Historically Associated with Source
IFE:	Infrequent Exceedance - less than 10 percent of MCL/SMCL or infrequent exceedance of AWQC
LMCL:	Less Than MCL
LSMCL:	Less Than Secondary MCL
LAWQC:	Less Than Ambient Water Quality Criteria

Definitions:

< = less than the laboratory method detection limit listed (non-detect)
N/A = Not Applicable
-- Compound has a 100% detection frequency
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
AWQC = Ambient Water Quality Criteria Human Health Consumption of 'Organism Only'
J = Estimated Value
U - Compound was analyzed for, but not detected
c = Cancer endpoint
na = not available
MCL - Federal Maximum Contaminant Level
SMCL - Secondary Maximum Contaminant Level
STV = Screening Toxicity Value

(7) AWQC listed for elemental mercury

TABLE 2.1.5

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN GROUNDWATER

CSM Unit 1

Scenario Timeframe:	Future
Medium:	Groundwater *
Exposure Medium:	Groundwater
Exposure Point:	Tap Water

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background (2) Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	64.2		7680		ug/L	Tamarack # 7	23/84	50 - 68	7680	na	3700	50-200	SMCL	NO	NUT/IFE
7440-36-0	***Antimony***	0.81		18		ug/L	Tiger-Poorman	32/84	0.049 - 1.5	18	na	1.5	6	MCL	YES	ASL/AMCL
7440-38-2	***Arsenic***	0.21		16.1		ug/L	Star Tailings	20/84	0.2 - 1	16.1	na	0.045 c	50	MCL	YES	ASL
7440-39-3	Barium	8.5		435		ug/L	Tamarack # 7	79/84	8.5 - 14.9	435	na	260	2000	MCL	NO	LMCL/IFE
7440-41-7	Beryllium	0.3		1.5		ug/L	Tamarack # 7	5/84	0.28 - 1	1.5	na	7.3	4	MCL	NO	BSL/LMCL
7440-43-9	***Cadmium***	0.1	J	996	J	ug/L	Nine Mile	71/84	0.079 - 1	996	na	1.8	5	MCL	YES	ASL/AMCL
7440-70-2	Calcium	3660		229000		ug/L	Rex # 2	84/84	--	229000	na	NE	NE	NE	NO	NUT
7440-47-3	Chromium (5)	0.098		8		ug/L	Tamarack # 7	23/84	0.058 - 5	8	na	18	100	MCL	NO	BSL/LMCL
7440-48-4	Cobalt	0.12		33.9		ug/L	Nine Mile	28/84	0.037 - 5	33.9	na	220	NE	NE	NO	BSL
7440-50-8	Copper	0.46		573		ug/L	Nine Mile	38/84	0.087 - 5	573	na	140	1300 / 1000	MCL / SMCL	NO	LMCL/IFE
7439-89-6	Iron	7.1		5110		ug/L	Tamarack # 7	31/84	6.9 - 50	5110	na	1100	300	SMCL	NO	NUT/IFE
7439-92-1	***Lead***	0.44		3170		ug/L	Nine Mile	71/84	0.18 - 1	3170	na	4	15	MCL	YES	ASL/AMCL
7439-95-4	Magnesium	900		17600		ug/L	Hecla-Star	84/84	--	17600	na	NE	NE	NE	NO	NUT
7439-96-5	Manganese	1.8		8030		ug/L	Hecla-Star	51/84	1.5 - 5	8030	na	170	50	SMCL	NO	IFE
7487-94-7	Mercury	0.33		0.33		ug/L	Tiger-Poorman	1/84	0.2	0.33	na	1.1	2	MCL	NO	BSL/LMCL/IFD
7440-02-0	Nickel	5.8		19.4		ug/L	Rex # 2	21/84	5 - 5.7	19.4	na	73	140	MCL	NO	BSL/LMCL
7440-09-7	Potassium	451		36500		ug/L	Rex # 2	73/84	491 - 786	36500	na	NE	NE	NE	NO	NUT
7782-49-2	Selenium	0.3		5.8		ug/L	Nine Mile	21/84	0.36 - 1	5.8	na	18	50	MCL	NO	BSL/LMCL
7440-22-4	Silver	4.6		4.6		ug/L	Hecla-Star	1/84	4.5 - 5	4.6	na	18	100	SMCL	NO	BSL/LSMCL/IFD
7440-23-5	Sodium	872	J	18200		ug/L	Canyon Silver	84/84	--	18200	na	NE	NE	NE	NO	NUT
1314-32-5	Thallium (6)	0.11		0.28		ug/L	Tiger-Poorman	11/84	0.03 - 1	0.28	na	0.26	2	MCL	NO	LMCL/IFE
7440-62-2	Vanadium	3.3		4.2		ug/L	Tamarack # 7	2/84	3.2 - 5	4.2	na	26	NE	NE	NO	BSL/IFD
7440-66-6	***Zinc***	2.8		145000		ug/L	Nine Mile	83/84	5	145000	na	1100	5000	SMCL	YES	ASL

NOTES: * Groundwater collected at depths between 2 to 127 feet below ground surface.

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration

(2) na - not available

(3) Screening toxicity values are 0.1 times the EPA Region 9 Tap Water PRGs (EPA 1998b) for noncarcinogens (except for lead).
Screening toxicity values for carcinogens are unchanged from the listed Tap Water PRG.

(4) Rationale Codes

Selection Reason:	HIST -	Infrequent Detection but Associated Historically
	FD -	Frequent Detection
	TX -	Toxicity Information Available
	ASL -	Above Screening Levels
	AMCL:	Above MCL
	ASMCL:	Above Secondary MCL
Deletion Reason:	IFD -	Infrequent Detection - less than 10 percent
	BKG -	Background Levels
	NTX -	No Toxicity Information
	NUT -	Essential Nutrient
	BSL -	Below Screening Level
	NHIST -	Not Historically Associated with Source
	IFE -	Infrequent Exceedance - less than 10 percent of STV or infrequent exceedance of PRG
	LMCL -	Less Than MCL
	LSMCL -	Less Than Secondary MCL

(5) Tap water STV listed for Chromium VI, no PRG established for Total Chromium

(6) STV listed for Thallic Oxide (more conservative)

Definitions:

< - less than the laboratory method detection limit listed (non-detect)
N/A - Not Applicable
-- Compound has a 100% detection frequency
COPC - Chemical of Potential Concern
ARAR/TBC - Applicable or Relevant and Appropriate Requirement/To Be Considered
MCL - Federal Maximum Contaminant Level
SMCL - Secondary Maximum Contaminant Level
NE - Not Established
U - Compound was analyzed for, but not detected
J - Estimated Value
c - Cancer endpoint
PRG = Preliminary Remediation Goal
STV = Screening Toxicity Value

TABLE 2.2.1

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN SOIL/SEDIMENT

CSM Unit 2

Scenario Timeframe:	Current
Medium:	Soil/Sediment
Exposure Medium:	Soil/Sediment
Exposure Point:	Soil/Sediment (all depths)

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background (2) Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	1190	J	52000		mg/kg	CUA095	2777/2777	--	52000	na	7500	na	na	NO	NUT / IFE
7440-36-0	***Antimony***	0.21	J	299	J	mg/kg	R036	1978/2809	0.2 - 8.52	299	5.8	3	na	na	YES	ASL
7440-38-2	***Arsenic***	0.47	J	1060		mg/kg	CUA091	2929/2939	0.18 - 15.2	1060	22	0.38 c	na	na	YES	ASL
7440-39-3	Barium	23.3		4300		mg/kg	CUA100	2777/2777	--	4300	1100	520	na	na	NO	IFE
7440-41-7	Beryllium	0.1		5.1		mg/kg	R019	2274/2777	0.158 - 1.4	5.1	2.1	15	na	na	NO	BSL
7440-43-9	***Cadmium***	0.06		115		mg/kg	R120	2738/2939	0.06 - 3.6	115	2.86	3.7	na	na	YES	ASL
7440-70-2	Calcium	377		85200	J	mg/kg	R111	2777/2777	--	85200	1	NE	na	na	NO	NUT
7440-47-3	Chromium (5)	1.99	J	187		mg/kg	R104	2777/2777	--	187	64	210 c	na	na	NO	BSL
7440-48-4	Cobalt	2		69.1		mg/kg	CUA099	2777/2777	--	69.1	20	330	na	na	NO	BSL
7440-50-8	Copper	5.1		1390		mg/kg	R019	2939/2939	--	1390	53	280	na	na	NO	IFE
7439-89-6	***Iron***	4780		174000		mg/kg	CUA077	2777/2777	--	174000	65000	2200	na	na	YES	ASL
7439-92-1	***Lead***	9.9		56900		mg/kg	CUA096	2939/2939	--	56900	175	400	na	na	YES	ASL
7439-95-4	Magnesium	513	J	29700		mg/kg	R019	2777/2777	--	29700	1.1	NE	na	na	NO	NUT
7439-96-5	***Manganese***	45.6		18200		mg/kg	CUA096	2777/2777	--	18200	3600	310	na	na	YES	ASL
7487-94-7	Mercury	0.05		31.1		mg/kg	CUA096	2476/2939	0.05 - 0.5	31.1	0.3	2.2	na	na	NO	IFE
7440-02-0	Nickel	3.1		102		mg/kg	R061	2776/2777	9	102	38	150	na	na	NO	BSL
7440-09-7	Potassium	271		9870		mg/kg	R073	2776/2777	1110	9870	na	NE	na	na	NO	NUT
7782-49-2	Selenium	0.38	J	11.4		mg/kg	CUA077	837/2726	0.37 - 2.5	11.4	na	37	na	na	NO	BSL
7440-22-4	Silver	0.27		228		mg/kg	R120	2451/2777	0.38 - 3	228	1.1	37	na	na	NO	IFE
7440-23-5	Sodium	10.8	J	1930		mg/kg	CUA102	2682/2777	20.9 - 489	1930	na	NE	na	na	NO	NUT
1314-32-5	Thallium (6)	0.39	J	9.2		mg/kg	R019	384/2749	0.38 - 6.2	9.2	na	0.52	na	na	NO	IFE / NHIST
7440-62-2	Vanadium	1.7		91.4		mg/kg	R027	2774/2777	17.2 - 19.3	91.4	154	52	na	na	NO	BKG/IFE
7440-66-6	***Zinc***	19.8		24300		mg/kg	R016	2939/2939	--	24300	280	2200	na	na	YES	ASL / HIST

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration.

(2) Background concentrations are the 90th percentile values from "Geochemical-Exploration Studies in the Coeur d'Alene District, Idaho and Montana" (Gott and Cathrall 1980).

(3) Screening toxicity values are 0.1 times the EPA Region 9 Residential Soil PRGs (EPA 1998b) for noncarcinogens. Screening toxicity values for carcinogens, and for lead, are unchanged from the listed soil PRG.

(4) Rationale Codes

Selection Reason:	HIST:	Infrequent Detection but Associated Historically
	FD:	Frequent Detection
	TX:	Toxicity Information Available
	ASL:	Above Screening Levels
Deletion Reason:	IFD:	Infrequent Detection - less than 10 percent
	BKG:	Background Levels
	NTX:	No Toxicity Information
	NUT:	Essential Nutrient
	BSL:	Below Screening Level
	NHIST:	Not Historically Associated with Source
	IFE:	Infrequent Exceedance of STV - less than 10 percent; or Infrequent Exceedance of PRG

(5) STV listed for Total Chromium (1/6 ratio Cr VI/Cr III); a carcinogen.

(6) STV listed for Thallic Oxide (more conservative)

Definitions:

na = not available
-- Compound has a 100% detection frequency
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
NE = Not Established
J = Estimated Value
c = Cancer endpoint
N/A = Not Applicable
PRG = Preliminary Remediation Goal
STV = Screening Toxicity Value

TABLE 2.2.2

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN FIRST-RUN TAP WATER

CSM Unit 2

Scenario Timeframe:	Current
Medium:	Groundwater (varies)
Exposure Medium:	Groundwater
Exposure Point:	First-Run Tap Water

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background (2) Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	43.5		261		ug/L	R076	4/67	22 - 64.2	261	na	3700	50-200	SMCL	NO	NUT/BSL
7440-36-0	Antimony	0.4		3		ug/L	R032	10/67	0.049 - 1	3	na	1.5	6	MCL	NO	LMCL/HWS/IFE
7440-38-2	***Arsenic***	0.2	J	2.3		ug/L	R072	32/67	0.2 - 1.7	2.3	na	0.045 c	50	MCL	YES	ASL / HWS
7440-39-3	Barium	14.3		72		ug/L	R084	44/67	5.3 - 33.9	72	na	260	2000	MCL	NO	BSL
7440-41-7	Beryllium	0.3		0.3		ug/L	R029	1/67	0.2 - 1	0.3	na	7.3	4	MCL	NO	BSL/IFD
7440-43-9	Cadmium	0.13		33.6		ug/L	R143	29/67	0.079 - 1	33.6	na	1.8	5	MCL	NO	HWS
7440-70-2	Calcium	3840		28500		ug/L	R072	67/67	--	28500	na	NE	NE	NE	NO	NUT
7440-47-3	Chromium (5)	0.096		1.6		ug/L	R090	28/67	0.058 - 5	1.6	na	18	100	MCL	NO	BSL
7440-48-4	Cobalt	0.14		4.1		ug/L	R076	6/67	0.037 - 5	4.1	na	220	NE	NE	NO	BSL
7440-50-8	Copper	3.4	J	2620		ug/L	R145	65/67	0.087 - 0.34	2620	na	140	1300 / 1000	MCL / SMCL	NO	HWS
7439-89-6	Iron	9		6340		ug/L	R029	28/67	8 - 128	6340	na	1100	300	SMCL	NO	NUT/HWS
7439-92-1	***Lead***	0.16		78.5		ug/L	R143	66/67	0.5	78.5	na	4	15	MCL	YES	ASL / HWS
7439-95-4	Magnesium	1140		21100		ug/L	R066/R072	67/67	--	21100	na	NE	NE	NE	NO	NUT
7439-96-5	Manganese	8.8		167		ug/L	R057	12/67	1.3 - 14.3	167	na	170	50	SMCL	NO	BSL
7487-94-7	Mercury	0.12		0.24		ug/L	R076	2/67	0.1 - 0.21	0.24	na	1.1	2	MCL	NO	BSL/IFD
7440-02-0	Nickel	4.9		484		ug/L	R060	9/67	4.4 - 8.7	484	na	73	140	MCL	NO	HWS/IFE
7440-09-7	Potassium	213		1870		ug/L	R143	66/67	115	1870	na	NE	NE	NE	NO	NUT
7782-49-2	Selenium	0.32		0.43		ug/L	R032	5/67	0.34 - 1	0.43	na	18	50	MCL	NO	BSL
7440-22-4	Silver	< 4.3	U	< 7.6	U	ug/L	N/A	0/67	4.3 - 7.6	< 7.6	na	18	100	SMCL	NO	BSL/IFD
7440-23-5	Sodium	786		22800		ug/L	R066	67/67	--	22800	na	NE	NE	NE	NO	NUT
1314-32-5	Thallium (6)	0.1		3.9		ug/L	R024	5/67	0.03 - 0.2	3.9	na	0.26	2	MCL	NO	HWS/IFE
7440-62-2	Vanadium	2.6		2.7		ug/L	R047	2/67	0.053 - 5	2.7	na	26	NE	NE	NO	BSL/IFD
7440-66-6	Zinc	13.2		6630		ug/L	R143	67/67	--	6630	na	1100	5000	SMCL	NO	HWS/IFE

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration

(2) na - not available

(3) Screening toxicity values are 0.1 times the EPA Region 9 Tap Water PRGs (EPA 1998b) for noncarcinogens (except for lead).
Screening toxicity values for carcinogens are unchanged from the listed Tap Water PRG.

(4) Rationale Codes

Selection Reason:	HIST -	Infrequent Detection but Associated Historically
	FD -	Frequent Detection
	TX -	Toxicity Information Available
	ASL -	Above Screening Levels
Deletion Reason:	IFD -	Infrequent Detection - less than 5 percent
	BKG -	Background Levels
	NTX -	No Toxicity Information
	NUT -	Essential Nutrient
	BSL -	Below Screening Level
	NHIST -	Not Historically Associated with Source
	IFE -	Infrequent Exceedance - less than 10 percent of STV or infrequent exceedance of PRG
	LMCL -	Less Than MCL
	LSMCL -	Less than secondary MCL
	HWS -	Home Water Supply is currently being addressed

(5) Tap water STV listed for Chromium VI, no PRG established for Total Chromium.

(6) STV listed for Thallic Oxide (more conservative).

Definitions:

< - less than the laboratory method detection limit listed (non-detect)
N/A - Not Applicable
-- Compound has a 100% detection frequency
COPC - Chemical of Potential Concern
ARAR/TBC - Applicable or Relevant and Appropriate Requirement/To Be Considered
MCL - Federal Maximum Contaminant Level
SMCL - Secondary Maximum Contaminant Level
NE - Not Established
U - Compound was analyzed for, but not detected
J - Estimated Value
c - Cancer endpoint
PRG = Preliminary Remediation Goal
STV = Screening Toxicity Value

TABLE 2.2.3

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN FLUSHED TAP WATER

CSM Unit 2

Scenario Timeframe:	Current
Medium:	Groundwater (varies)
Exposure Medium:	Groundwater
Exposure Point:	Flushed Tap Water

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background (2) Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	28.7		164		ug/L	R072	3/67	22 - 67.8	164	na	3700	50-200	SMCL	NO	NUT/BSL/IFD
7440-36-0	Antimony	0.84		2.9		ug/L	R048	8/67	0.049 - 1.2	2.9	na	1.5	6	MCL	NO	HWS/LMCL/IFE
7440-38-2	***Arsenic***	0.2		3.2		ug/L	R066	34/67	0.2 - 0.7	3.2	na	0.045 c	50	MCL	NO	ASL/ HWS /LMCL
7440-39-3	Barium	15.7		66		ug/L	R060	39/67	7.6 - 33.7	66	na	260	2000	MCL	NO	BSL / LMCL
7440-41-7	Beryllium	< 0.2	U	< 1	U	ug/L	N/A	0/67	0.2 - 1	< 1	na	7.3	4	MCL	NO	BSL / IFD
7440-43-9	Cadmium	0.11		29		ug/L	R143	14/67	0.079 - 1	29	na	1.8	5	MCL	NO	ASL / HWS
7440-70-2	Calcium	3410		28400		ug/L	R072	67/67	--	28400	na	NE	NE	NE	NO	NUT
7440-47-3	Chromium (5)	0.096		0.88		ug/L	R084	31/67	0.058 - 5	0.88	na	18	100	MCL	NO	BSL / LMCL
7440-48-4	Cobalt	0.1		0.87		ug/L	R057	2/67	0.037 - 5	0.87	na	220	NE	NE	NO	BSL / IFD
7440-50-8	Copper	0.41	J	184	J	ug/L	R048	53/67	0.087 - 5.6	184	na	140	1300 / 1000	MCL / SMCL	NO	HWS/LMCL/IFE
7439-89-6	Iron	8.5		2170		ug/L	R057	13/67	8 - 102	2170	na	1100	300	SMCL	NO	NUT/IFE/HWS
7439-92-1	Lead	0.1	J	3.4		ug/L	R048	54/67	0.04 - 0.5	3.4	na	4	15	MCL	NO	BSL / LMCL
7439-95-4	Magnesium	1050		21100		ug/L	R072	67/67	--	21100	na	NE	NE	NE	NO	NUT
7439-96-5	Manganese	6.9		182		ug/L	R057	6/67	1.3 - 12.7	182	na	170	50	SMCL	NO	HWS / IFE
7487-94-7	Mercury	0.41		0.41		ug/L	R078	1/67	0.1 - 0.2	0.41	na	1.1	2	MCL	NO	BSL/IFD/LMCL
7440-02-0	Nickel	5.2		5.2		ug/L	R016	1/67	4.4 - 8.5	5.2	na	73	140	MCL	NO	BSL/IFD/LMCL
7440-09-7	Potassium	155		1810		ug/L	R143	67/67	--	1810	na	NE	NE	NE	NO	NUT
7782-49-2	Selenium	0.31		1.8		ug/L	R043	10/67	0.36 - 1	1.8	na	18	50	MCL	NO	BSL/LMCL
7440-22-4	Silver	< 4.3	U	< 6.8		ug/L	N/A	0/67	4.3 - 6.8	< 6.8	na	18	100	SMCL	NO	BSL/IFD/LMCL
7440-23-5	Sodium	768		22200		ug/L	R066	67/67	--	22200	na	NE	NE	NE	NO	NUT
1314-32-5	Thallium (6)	0.1		0.16		ug/L	R069	2/67	0.03 - 0.38	0.16	na	0.26	2	MCL	NO	BSL/IFD/LMCL
7440-62-2	Vanadium	3.1		3.1		ug/L	R024	1/67	0.053 - 5	3.1	na	26	NE	NE	NO	BSL/IFD
7440-66-6	Zinc	4.3		5530		ug/L	R143	63/67	5 - 5.9	5530	na	1100	5000	SMCL	NO	HWS/IFE

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration

(2) na - not available

(3) Screening toxicity values are 0.1 times the EPA Region 9 Tap Water PRGs (EPA 1998b) for noncarcinogens (except for lead).
Screening toxicity values for carcinogens are unchanged from the listed Tap Water PRG.

(4) Rationale Codes

Selection Reason:	HIST -	Infrequent Detection but Associated Historically
	FD -	Frequent Detection
	TX -	Toxicity Information Available
	ASL -	Above Screening Levels
Deletion Reason:	IFD -	Infrequent Detection - less than 5 percent
	BKG -	Background Levels
	NTX -	No Toxicity Information
	NUT -	Essential Nutrient
	BSL -	Below Screening Level
	NHIST -	Not Historically Associated with Source
	IFE -	Infrequent Exceedance - less than 10 percent of STV or infrequent exceedance of PRG
	LMCL -	Less Than MCL
	LSMCL -	Less than secondary MCL
	HWS -	Home Water Supply is currently being addressed

(5) Tap water STV listed for Chromium VI, no PRG established for Total Chromium.

(6) STV listed for Thallic Oxide (more conservative).

Definitions:

< - less than the laboratory method detection limit listed (non-detect)
N/A - Not Applicable
-- Compound has a 100% detection frequency
COPC - Chemical of Potential Concern
ARAR/TBC - Applicable or Relevant and Appropriate Requirement/To Be Considered
MCL - Federal Maximum Contaminant Level
SMCL - Secondary Maximum Contaminant Level
NE - Not Established
U - Compound was analyzed for, but not detected
J - Estimated Value
c - Cancer endpoint
PRG = Preliminary Remediation Goal
STV = Screening Toxicity Value

TABLE 2.2.4

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN SURFACE WATER

CSM Unit 2

Scenario Timeframe:	Current
Medium:	Soil/Groundwater
Exposure Medium:	Surface Water
Exposure Point:	South Fork of the CdA River and Elk Creek Pond

CAS Number	Chemical	Minimum (1) Conc.	Minimum Qualifier	Maximum (1) Conc.	Maximum Qualifier	Units	Location of Maximum Conc.	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value (3)	Screening (4) Toxicity Value	Potential ARAR/TBC Value (5)	Potential ARAR/TBC Source	COPC Flag	Rationale for (6) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	8.5	J	18900		ug/L	CUA081	27/74	0.69 - 56.6	18900	na	NE	50 - 200	SMCL	NO	NUT
7440-36-0	Antimony	0.2		36.7		ug/L	CUA077	48/74	0.032 - 7.2	36.7	0.51	NE	6 / 4300	MCL/AWQC	NO	LAWQC
7440-38-2	***Arsenic***	0.2		134	J	ug/L	CUA077	42/74	0.16 - 2	134	0.65	NE	50 / 0.14	MCL/AWQC	YES	AAWQC
7440-39-3	Barium	8.3		343		ug/L	CUA077	72/74	22.9 - 27.7	343	na	NE	2000	MCL	NO	LMCL
7440-41-7	Beryllium	< 0.11	U	< 1.3	U	ug/L	N/A	0/74	0.11 - 1.3	< 1.3	na	NE	4	MCL	NO	IFD/LMCL
7440-43-9	***Cadmium***	0.1	J	83.8		ug/L	CUA077	44/74	0.042 - 0.2	83.8	0.09	NE	5	MCL	YES	AMCL
7440-70-2	Calcium	4040		64300		ug/L	CUA081	74/74	--	64300	na	NE	na	na	NO	NUT
7440-47-3	Chromium	2.4		16.8		ug/L	CUA081	9/74	0.4 - 6	16.8	na	NE	100	MCL	NO	LMCL
7440-48-4	Cobalt	0.042		23.9		ug/L	CUA077	13/74	0.023 - 10	23.9	na	NE	na	na	NO	NTX
7440-50-8	Copper	0.2	J	236		ug/L	CUA077	50/74	0.06 - 3	236	1.21	NE	1300	MCL	NO	LMCL
7439-89-6	Iron	5.8	J	81100		ug/L	CUA077	56/74	5 - 76.8	81100	113	NE	300	SMCL	NO	NUT
7439-92-1	***Lead***	0.16	J	12500	J	ug/L	CUA077	71/74	0.27 - 0.5	12500	1.46	NE	15	MCL	YES	AMCL
7439-95-4	Magnesium	1410		19500		ug/L	CUA081	74/74	--	19500	na	NE	na	na	NO	NUT
7439-96-5	***Manganese***	0.83	J	9470		ug/L	CUA077	59/74	1 - 5	9470	8.28	NE	50 / 100	SMCL/AWQC	YES	AAWQC / ASMCL
7487-94-7	***Mercury***	0.18		4.6		ug/L	CUA077	8/74	0.1 - 0.5	4.6	0.09	NE	2 / 0.051 (7)	MCL/AWQC	YES	AMCL / AAWQC
7440-02-0	Nickel	0.3	J	34.3		ug/L	CUA081	6/74	0.22 - 20	34.3	na	NE	140 / 4600	MCL/AWQC	NO	LMCL / LAWQC
7440-09-7	Potassium	286	J	7040		ug/L	CUA081	52/74	200 - 2000	7040	na	NE	na	na	NO	NUT
7782-49-2	Selenium	0.29	J	2.3		ug/L	CUA077	11/74	0.21 - 2	2.3	na	NE	50 / 11000	MCL / AWQC	NO	LMCL / LAWQC
7440-22-4	Silver	18.5		26		ug/L	CUA081	2/74	0.042 - 5	26	0.12	NE	100	SMCL	NO	LSMCL / IFD
7440-23-5	Sodium	643		37600		ug/L	SegMG01	74/74	--	37600	na	NE	na	na	NO	NUT
7440-28-0	Thallium	0.16	J	0.22	J	ug/L	SegMG01	2/74	0.017 - 0.4	0.22	na	NE	2 / 6.3	MCL/AWQC	NO	IFD/LMCL/LAWQC
7440-62-2	Vanadium	0.12		40.7		ug/L	CUA081	11/74	0.11 - 10	40.7	na	NE	na	na	NO	NTX
7440-66-6	Zinc	1.8	J	9900		ug/L	CUA077	57/74	5 - 27.4	9900	20.71	NE	5000 / 69000	SMCL/AWQC	NO	IFE

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration.

(2) STV based on using surface water as a drinking water source.

(3) Background Values as calculated by URS, March 2000

(4) Screening toxicity values not established (NE).

(5) Potential ARARs are the Ambient Water Quality Criteria, human health for consumption of organisms only. EPA 1998c (Vol.63, No.237)

(6) Rationale Codes

Selection Reason:	HIST:	Infrequent Detection but Associated Historically
	FD:	Frequent Detection
	TX:	Toxicity Information Available
	ASL:	Above Screening Levels
	AMCL:	Above MCL
	ASMCL:	Above Secondary MCL
	AAWQC:	Above Ambient Water Quality Criteria
Deletion Reason:	IFD:	Infrequent Detection - less than 5 percent
	BKG:	Background Levels
	NTX:	No Toxicity Information
	NUT:	Essential Nutrient
	BSL:	Below Screening Level
	NHIST:	Not Historically Associated with Source
	IFE:	Infrequent Exceedance - less than 5 percent
	LMCL:	Less Than MCL
	LSMCL:	Less than secondary MCL
	LAWQC:	Less Than Ambient Water Quality Criteria

Definitions:

< = less than the laboratory method detection limit listed (non-detect)
N/A = Not Applicable
-- Compound has a 100% detection frequency
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
AWQC = Ambient Water Quality Criteria Human Health Consumption of 'Organism Only'
J = Estimated Value
U - Compound was analyzed for, but not detected
c = Cancer endpoint
na = not available
MCL - Federal Maximum Contaminant Level
SMCL - Secondary Maximum Contaminant Level
STV = Screening Toxicity Value

TABLE 2.3.1

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN SOIL/SEDIMENT

CSM Unit 3

Scenario Timeframe:	Current
Medium:	Soil/Sediment
Exposure Medium:	Soil/Sediment
Exposure Point:	Soil/Sediment (all depths)

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background (2) Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	1330		37300		mg/kg	CUA021	457/457	--	37300	na	7500	na	na	NO	NUT / IFE
7440-36-0	***Antimony***	0.43		73.7		mg/kg	CUA063	412/462	0.2 - 30	73.7	5.8	3	na	na	YES	ASL
7440-38-2	***Arsenic***	1.5		492	J	mg/kg	CUA066	469/469	--	492	22	0.38 c	na	na	YES	ASL
7440-39-3	Barium	30.1		632		mg/kg	CUA049	457/457	--	632	1100	520	na	na	NO	BKG/IFE
7440-41-7	Beryllium	0.11	J	9.7		mg/kg	CUA049	288/457	0.18 - 0.73	9.7	2.1	15	na	na	NO	BSL
7440-43-9	***Cadmium***	0.08		105		mg/kg	CUA057	443/469	0.08 - 0.7	105	2.86	3.7	na	na	YES	ASL
7440-70-2	Calcium	368		12600		mg/kg	CUA018	457/457	--	12600	1	NE	na	na	NO	NUT
7440-47-3	Chromium (5)	0.14	J	45.5		mg/kg	CUA049	415/457	0.14 - 3.9	45.5	64	210 c	na	na	NO	BSL/BKG
7440-48-4	Cobalt	1.1		106		mg/kg	CUA049	455/457	8.4 - 8.7	106	20	330	na	na	NO	BSL
7440-50-8	Copper	2.1		554		mg/kg	CUA063	469/469	--	554	53	280	na	na	NO	IFE
7439-89-6	***Iron***	4450		256000		mg/kg	CUA057	457/457	--	256000	65000	2200	na	na	YES	ASL
7439-92-1	***Lead***	10.9		29200		mg/kg	CUA063	469/469	--	29200	175	400	na	na	YES	ASL
7439-95-4	Magnesium	389		8310		mg/kg	CUA021	457/457	--	8310	1.1	NE	na	na	NO	NUT
7439-96-5	***Manganese***	92.3		26400		mg/kg	CUA058	457/457	--	26400	3600	310	na	na	YES	ASL
7487-94-7	Mercury	0.05		23		mg/kg	CUA063	418/469	0.05 - 0.4	23	0.3	2.2	na	na	NO	IFE
7440-02-0	Nickel	0.9		106		mg/kg	CUA049	456/457	5.8	106	38	150	na	na	NO	BSL
7440-09-7	Potassium	147		4510		mg/kg	CUA021	457/457	--	4510	na	NE	na	na	NO	NUT
7782-49-2	Selenium	0.44	J	6.3		mg/kg	CUA059	66/414	0.43 - 2.6	6.3	na	37	na	na	NO	BSL
7440-22-4	Silver	0.53		97.9		mg/kg	CUA063	400/457	0.39 - 5.7	97.9	1.1	37	na	na	NO	IFE
7440-23-5	Sodium	10.6	J	1430		mg/kg	CUA035	409/457	26.6 - 123	1430	na	NE	na	na	NO	NUT
1314-32-5	Thallium (6)	0.43	J	11.3	J	mg/kg	CUA066	74/381	0.38 - 3	11.3	na	0.52	na	na	NO	IFE
7440-62-2	Vanadium	0.39		106		mg/kg	CUA049	403/457	0.39 - 10.5	106	154	52	na	na	NO	BKG / IFE
7440-66-6	***Zinc***	14.3		21800		mg/kg	CUA057	469/469	--	21800	280	2200	na	na	YES	ASL

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration.

(2) Background concentrations are the 90th percentile values from "Geochemical-Exploration Studies in the Coeur d'Alene District, Idaho and Montana" (Gott and Cathrall 1980).

(3) Screening toxicity values are 0.1 times the EPA Region 9 Residential Soil PRGs (EPA 1998b) for noncarcinogens. Screening toxicity values for carcinogens, and for lead, are unchanged from the listed soil PRG.

(4) Rationale Codes

Selection Reason:	HIST:	Infrequent Detection but Associated Historically
	FD:	Frequent Detection
	TX:	Toxicity Information Available
	ASL:	Above Screening Levels
Deletion Reason:	IFD:	Infrequent Detection - less than 10 percent
	BKG:	Background Levels
	NTX:	No Toxicity Information
	NUT:	Essential Nutrient
	BSL:	Below Screening Level
	NHIST:	Not Historically Associated with Source
	IFE:	Infrequent Exceedance of STV - less than 10 percent or Infrequent Exceedance of PRG

(5) STV listed for Total Chromium (1/6 ratio Cr VI/Cr III); a carcinogen.

(6) STV listed for Thallic Oxide (more conservative).

Definitions:

-- Compound has a 100% detection frequency
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
NE = Not Established
J = Estimated Value
c = Cancer endpoint
na = not available
PRG = Preliminary Remediation Goal
STV = Screening Toxicity Value

TABLE 2.3.2

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN FIRST-RUN TAP WATER

CSM Unit 3

Scenario Timeframe:	Current
Medium:	Groundwater (varies)
Exposure Medium:	Groundwater
Exposure Point:	First-Run Tap Water

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background (2) Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	1740		1740		ug/L	R144	1/6	22 - 50	1740	na	3700	50-200	SMCL	NO	NUT / BSL
7440-36-0	Antimony	0.52		0.52		ug/L	R115	1/6	0.21 - 0.5	0.52	na	1.5	6	MCL	NO	BSL / LMCL
7440-38-2	***Arsenic***	0.26		4.3		ug/L	CUA050	3/6	0.34 - 1.7	4.3	na	0.045 c	50	MCL	YES	ASL / HWS
7440-39-3	Barium	45.4		80.3		ug/L	R144	4/6	5	80.3	na	260	2000	MCL	NO	BSL / LMCL
7440-41-7	Beryllium	< 0.2	U	< 1	U	ug/L	N/A	0/6	0.2 - 1	< 1	na	7.3	4	MCL	NO	BSL / LMCL / IFD
7440-43-9	Cadmium	0.18		4.2		ug/L	R115	3/6	1	4.2	na	1.8	5	MCL	NO	LMCL / HWS
7440-70-2	Calcium	9340		32800		ug/L	CUA067	6/6	--	32800	na	NE	NE	NE	NO	NUT
7440-47-3	Chromium (5)	0.14		0.14		ug/L	R007	1/6	0.11 - 5	0.14	na	18	100	MCL	NO	BSL / LMCL
7440-48-4	Cobalt	0.97		0.97		ug/L	R025	1/6	0.037 - 5	0.97	na	220	NE	NE	NO	BSL
7440-50-8	Copper	23.6		2430		ug/L	R025	4/6	5	2430	na	140	1300 / 1000	MCL / SMCL	NO	HWS
7439-89-6	Iron	106		2260		ug/L	R144	2/6	8 - 20.9	2260	na	1100	300	SMCL	NO	NUT
7439-92-1	***Lead***	2.2		17.2		ug/L	R144	6/6	--	17.2	na	4	15	MCL	YES	ASL / HWS
7439-95-4	Magnesium	3020		11700		ug/L	CUA067	6/6	--	11700	na	NE	NE	NE	NO	NUT
7439-96-5	Manganese	5.2		82		ug/L	R144	5/6	2.1	82	na	170	50	SMCL	NO	BSL
7487-94-7	Mercury	< 0.1	U	< 0.2	U	ug/L	N/A	0/6	0.1 - 0.2	< 0.2	na	1.1	2	MCL	NO	BSL / IFD / LMCL
7440-02-0	Nickel	51.9		51.9		ug/L	R025	1/6	4.4 - 5	51.9	na	73	140	MCL	NO	BSL / LMCL
7440-09-7	Potassium	755		1950		ug/L	R144	6/6	--	1950	na	NE	NE	NE	NO	NUT
7782-49-2	Selenium	< 0.34	U	< 1	U	ug/L	N/A	0/6	0.34 - 1	< 1	na	18	50	MCL	NO	BSL / IFD / LMCL
7440-22-4	Silver	< 4.3	U	< 5	U	ug/L	N/A	0/6	4.3 - 5	< 5	na	18	100	SMCL	NO	BSL / IFD / LSMCL
7440-23-5	Sodium	1940		18100		ug/L	CUA067	6/6	--	18100	na	NE	NE	NE	NO	NUT
1314-32-5	Thallium (6)	< 0.03	U	< 0.4	U	ug/L	N/A	0/6	0.03 - 0.4	< 0.4	na	0.26	2	MCL	NO	BSL / IFD / LMCL
7440-62-2	Vanadium	< 0.053	U	< 5	U	ug/L	N/A	0/6	0.053 - 5	< 5	na	26	NE	NE	NO	BSL / IFD
7440-66-6	Zinc	184		2810		ug/L	R144	6/6	--	2810	na	1100	5000	SMCL	NO	LSMCL / HWS / IFE

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration

(2) na - not available

(3) Screening toxicity values are 0.1 times the EPA Region 9 Tap Water PRGs (EPA 1998b) for noncarcinogens (except for lead).
Screening toxicity values for carcinogens are unchanged from the listed Tap Water PRG.

(4) Rationale Codes

Selection Reason:	HIST -	Infrequent Detection but Associated Historically
	FD -	Frequent Detection
	TX -	Toxicity Information Available
	ASL -	Above Screening Levels
Deletion Reason:	IFD -	Infrequent Detection - less than 5 percent
	BKG -	Background Levels
	NTX -	No Toxicity Information
	NUT -	Essential Nutrient
	BSL -	Below Screening Level
	NHIST -	Not Historically Associated with Source
	IFE -	Infrequent Exceedance - less than 10 percent of STV or Infrequent Exceedance of PRG
	LMCL -	Less Than MCL
	LSMCL -	Less Than Secondary MCL
	HWS -	Home Water Supply is currently being addressed

(5) Tap water STV listed for Chromium VI, no PRG established for Total Chromium.

(6) STV listed for Thallic Oxide (more conservative).

Definitions:

< - less than the laboratory method detection limit listed (non-detect)
N/A - Not Applicable
-- Compound has a 100% detection frequency
COPC - Chemical of Potential Concern
ARAR/TBC - Applicable or Relevant and Appropriate Requirement/To Be Considered
MCL - Federal Maximum Contaminant Level
SMCL - Secondary Maximum Contaminant Level
NE - Not Established
U - Compound was analyzed for, but not detected
J - Estimated Value
c - Cancer endpoint
PRG = Preliminary Remediation Goal
STV = Screening Toxicity Value

TABLE 2.3.3

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN FLUSHED TAP WATER

CSM Unit 3

Scenario Timeframe:	Current
Medium:	Groundwater (varies)
Exposure Medium:	Groundwater
Exposure Point:	Flushed Tap Water

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background (2) Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	2870		2870		ug/L	R144	1/4	22.3 - 50	2870	na	3700	50-200	SMCL	NO	NUT / BSL
7440-36-0	Antimony	2.8		2.8		ug/L	R144	1/4	0.049 - 0.5	2.8	na	1.5	6	MCL	NO	LMCL / HWS / IFE
7440-38-2	***Arsenic***	1.1		1.1		ug/L	R144	1/4	0.2 - 0.5	1.1	na	0.045 c	50	MCL	YES	ASL / HWS
7440-39-3	Barium	44.7		73.5		ug/L	R144	4/4	--	73.5	na	260	2000	MCL	NO	BSL / LMCL
7440-41-7	Beryllium	< 0.2	U	< 1	U	ug/L	N/A	0/4	0.2 - 1	< 1	na	7.3	4	MCL	NO	BSL / LMCL / IFD
7440-43-9	Cadmium	0.17		0.22		ug/L	R025	2/4	1	0.22	na	1.8	5	MCL	NO	BSL / LMCL
7440-70-2	Calcium	8910		13200		ug/L	R115	4/4	--	13200	na	NE	NE	NE	NO	NUT
7440-47-3	Chromium (5)	0.21		0.21		ug/L	R007	1/4	0.31 - 5	0.21	na	18	100	MCL	NO	BSL / LMCL
7440-48-4	Cobalt	< 0.037	U	< 5	U	ug/L	N/A	0/4	0.037 - 5	< 5	na	220	NE	NE	NO	BSL / IFD
7440-50-8	Copper	10.7		86		ug/L	R007	3/4	5	86	na	140	1300 / 1000	MCL / SMCL	NO	BSL / LMCL
7439-89-6	Iron	183		2280		ug/L	R144	2/4	8 - 20	2280	na	1100	300	SMCL	NO	NUT
7439-92-1	***Lead***	0.44		4.6		ug/L	R144	3/4	0.5	4.6	na	4	15	MCL	YES	ASL / HWS
7439-95-4	Magnesium	2940		6500		ug/L	R115	4/4	--	6500	na	NE	NE	NE	NO	NUT
7439-96-5	Manganese	24.2		65.4		ug/L	R144	3/4	2.7	65.4	na	170	50	SMCL	NO	BSL
7487-94-7	Mercury	< 0.1	U	< 0.2	U	ug/L	N/A	0/4	0.1 - 0.2	< 0.2	na	1.1	2	MCL	NO	BSL / IFD / LMCL
7440-02-0	Nickel	< 4.4	U	< 5.1	U	ug/L	N/A	0/4	4.4 - 5.1	< 5.1	na	73	140	MCL	NO	BSL / LMCL
7440-09-7	Potassium	583		2270		ug/L	R144	4/4	--	2270	na	NE	NE	NE	NO	NUT
7782-49-2	Selenium	< 0.36	U	< 1	U	ug/L	N/A	0/4	0.36 - 1	< 1	na	18	50	MCL	NO	BSL / IFD / LMCL
7440-22-4	Silver	< 4.3	U	< 5	U	ug/L	N/A	0/4	4.3 - 5	< 5	na	18	100	SMCL	NO	BSL / IFD / LSMCL
7440-23-5	Sodium	1850		9600		ug/L	R115	4/4	--	9600	na	NE	NE	NE	NO	NUT
1314-32-5	Thallium (6)	< 0.03	U	< 0.2	U	ug/L	N/A	0/4	0.03 - 0.2	< 0.2	na	0.26	2	MCL	NO	BSL / IFD / LMCL
7440-62-2	Vanadium	< 0.053	U	< 5	U	ug/L	N/A	0/4	0.053 - 5	< 5	na	26	NE	NE	NO	BSL / IFD
7440-66-6	Zinc	147		2410		ug/L	R144	4/4	--	2410	na	1100	5000	SMCL	NO	LSMCL/ HWS /IFE

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration

(2) na - not available

(3) Screening toxicity values are 0.1 times the EPA Region 9 Tap Water PRGs (EPA 1998b) for noncarcinogens (except for lead).
Screening toxicity values for carcinogens are unchanged from the listed Tap Water PRG.

(4) Rationale Codes

Selection Reason:	HIST -	Infrequent Detection but Associated Historically
	FD -	Frequent Detection
	TX -	Toxicity Information Available
	ASL -	Above Screening Levels
Deletion Reason:	IFD -	Infrequent Detection - less than 5 percent
	BKG -	Background Levels
	NTX -	No Toxicity Information
	NUT -	Essential Nutrient
	BSL -	Below Screening Level
	NHIST -	Not Historically Associated with Source
	IFE -	Infrequent Exceedance - less than 10 percent of STV or infrequent exceedance of PRG
	LMCL -	Less Than MCL
	LSMCL -	Less Than Secondary MCL
	HWS -	Home Water Supply is currently being addressed

(5) Tap water STV listed for Chromium VI, no PRG established for Total Chromium.

(6) STV listed for Thallic Oxide (more conservative).

Definitions:

< - less than the laboratory method detection limit listed (non-detect)
N/A - Not Applicable
-- Compound has a 100% detection frequency
COPC - Chemical of Potential Concern
ARAR/TBC - Applicable or Relevant and Appropriate Requirement/To Be Considered
MCL - Federal Maximum Contaminant Level
SMCL - Secondary Maximum Contaminant Level
NE - Not Established
U - Compound was analyzed for, but not detected
J - Estimated Value
c - Cancer endpoint
PRG = Preliminary Remediation Goal
STV = Screening Toxicity Value

TABLE 2.3.4

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN SURFACE WATER

CSM Unit 3

Scenario Timeframe:	Current
Medium:	Groundwater
Exposure Medium:	Surface Water
Exposure Point:	Coeur d'Alene River

CAS Number	Chemical	Minimum (1) Conc.	Minimum Qualifier	Maximum (1) Conc.	Maximum Qualifier	Units	Location of Maximum Conc.	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value (3)	Screening (4) Toxicity Value	Potential ARAR/TBC Value (5)	Potential ARAR/TBC Source	COPC Flag	Rationale for (6) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	268	J	123000		ug/L	CUA048	129/129	--	123000	na	NE	50 - 200	SMCL	NO	NUT/LSMCL
7440-36-0	Antimony	0.99		39.5	J	ug/L	CUA053	100/129	1.3 - 13.7	39.5	0.51	NE	6 / 4300	MCL / AWQC	NO	LAWQC
7440-38-2	***Arsenic***	2.9		600	J	ug/L	CUA048	129/129	--	600	0.65	NE	50 / 0.14	MCL / AWQC	YES	AMCL/AAWQC
7440-39-3	Barium	13		3280		ug/L	CUA048	129/129	--	3280	na	NE	2000	MCL	NO	IFE
7440-41-7	Beryllium	0.6		9.1		ug/L	CUA048	32/129	0.2 - 1.5	9.1	na	NE	4	MCL	NO	IFE
7440-43-9	***Cadmium***	1.3		1200	J	ug/L	CUA048	125/129	1	1200	0.09	NE	5	MCL	YES	AMCL
7440-70-2	Calcium	5680		50900		ug/L	CUA048	129/129	--	50900	na	NE	na	na	NO	NUT
7440-47-3	Chromium	0.82		151	J	ug/L	CUA048	99/129	0.53 - 5	151	na	NE	100	MCL	NO	IFE
7440-48-4	Cobalt	0.2	J	160	J	ug/L	CUA048	104/129	1.3 - 5	160	na	NE	na	na	NO	NTX
7440-50-8	Copper	3		2810		ug/L	CUA048	126/129	5	2810	1.21	NE	1300	MCL	NO	IFE
7439-89-6	Iron	1650	J	1000000		ug/L	CUA048	129/129	--	1000000	113	NE	300	SMCL	NO	NUT
7439-92-1	***Lead***	107		81500	J	ug/L	CUA048	129/129	--	81500	1.46	NE	15	MCL	YES	AMCL
7439-95-4	Magnesium	2120		62000		ug/L	CUA048	129/129	--	62000	na	NE	na	na	NO	NUT
7439-96-5	***Manganese***	124		84900		ug/L	CUA048	129/129	--	84900	8.28	NE	50 / 100	SMCL / AWQC	YES	ASMCL/AAWQC
7487-94-7	***Mercury***	0.12		43.9		ug/L	CUA048	108/128	0.1 - 0.49	43.9	0.09	NE	2 / 0.051 (7)	MCL / AWQC	YES	AMCL/AAWQC
7440-02-0	Nickel	3.6		219		ug/L	CUA048	80/129	4.4 - 20.4	219	na	NE	140 / 4600	MCL / AWQC	NO	LAWQC
7440-09-7	Potassium	624		8430		ug/L	CUA048	128/129	797	8430	na	NE	na	na	NO	NUT
7782-49-2	Selenium	0.36	J	13.5	J	ug/L	CUA048	54/129	0.32 - 1.1	13.5	na	NE	50 / 11000	MCL / AWQC	NO	LMCL/LAWQC
7440-22-4	Silver	4.4		308		ug/L	CUA048	72/129	4.3 - 5	308	0.12	NE	100	SMCL	NO	IFE
7440-23-5	Sodium	1310		3270		ug/L	CUA051	129/129	--	3270	na	NE	na	na	NO	NUT
7440-28-0	Thallium	0.12		54		ug/L	CUA057	15/129	0.03 - 0.76	54	na	NE	2 / 6.3	MCL / AWQC	NO	IFE
7440-62-2	Vanadium	2.8		172	J	ug/L	CUA048	65/129	2.6 - 25	172	na	NE	na	na	NO	NTX
7440-66-6	Zinc	114		116000		ug/L	CUA048	129/129	--	116000	20.71	NE	5000 / 69000	SMCL / AWQC	NO	IFE

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration.

(2) STV based on using surface water as a drinking water source.

(3) Background values as calculated by URS, March 2000.

(4) Screening toxicity values not established (NE).

(5) Potential ARARs are the Ambient Water Quality Criteria, human health for consumption of organisms only. EPA 1998c (Vol.63, No.237)

(6) Rationale Codes

Selection Reason:	HIST:	Infrequent Detection but Associated Historically
	FD:	Frequent Detection
	TX:	Toxicity Information Available
	ASL:	Above Screening Levels
	AMCL:	Above MCL
	ASMCL:	Above Secondary MCL
	AAWQC:	Above Ambient Water Quality Criteria
Deletion Reason:	IFD:	Infrequent Detection - less than 5 percent
	BKG:	Background Levels
	NTX:	No Toxicity Information
	NUT:	Essential Nutrient
	BSL:	Below Screening Level
	NHIST:	Not Historically Associated with Source
	IFE:	Infrequent Exceedance - less than 5 percent
	LMCL -	Less Than MCL
	LSMCL -	Less Than Secondary MCL
	LAWQC:	Less Than Ambient Water Quality Criteria

(7) AWQC listed for elemental mercury

Definitions:

< = less than the laboratory method detection limit listed (non-detect)
na = not available
-- Compound has a 100% detection frequency
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
AWQC = Ambient Water Quality Criteria Human Health Consumption of 'Organism Only'
NE = Not Established
J = Estimated Value
U - Compound was analyzed for, but not detected
c = Cancer endpoint
N/A = Not Applicable
MCL - Federal Maximum Contaminant Level
SMCL - Secondary Maximum Contaminant Level
STV = Screening Toxicity Value

TABLE 2.3.5
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN WATER POTATOES
CSM Unit 3

Scenario Timeframe:	Current
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Water Potatoes

CAS Number	Chemical	Minimum Concentration (1)	Minimum Qualifier	Maximum Concentration (1)	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value	Screening Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for Contaminant Deletion or Selection (2)
	Inorganics															
with skin																
7429-90-5	Aluminum	3.59		40.72		mg/kg	H1-T6U	94/95	2.30-4.67	40.72	na	na	na	na	NO	NUT
7440-43-9	***Cadmium***	0.07		3.71		mg/kg	MS5-U	87/95	0.05-0.12	3.71	na	na	na	na	YES	TX/FD
7439-89-6	Iron	62.45	J	3133	J	mg/kg	SM4-T11U	95/95	4.60-9.34	3133	na	na	na	na	NO	NUT
7439-92-1	***Lead***	1.48	J	127	J	mg/kg	MS5-U	89/95	0.46-1.87	127	na	na	na	na	YES	TX/FD
7439-96-5	Manganese	1.82	J	152	J	mg/kg	BL11-T9U	95/95	0.18-0.37	152	na	na	na	na	NO	NUT
7440-66-6	Zinc	8.41	J	147	J	mg/kg	MS5-U	95/95	0.46-0.93	147	na	na	na	na	NO	NUT
without skin																
7429-90-5	Aluminum	4.00	J	11.22		mg/kg	SM4-T3S	3/93	2.49-4.08	11.22	na	na	na	na	NO	NUT
7440-43-9	***Cadmium***	0.07	J	2.80		mg/kg	MS5-S	70/93	0.05-0.08	2.80	na	na	na	na	YES	TX/FD
7439-89-6	Iron	5.70		135		mg/kg	SM4-T12S	80/93	4.98-8.16	135	na	na	na	na	NO	NUT
7439-92-1	***Lead***	1.47		1.98		mg/kg	MS5-S	2/93	0.50-1.20	1.98	na	na	na	na	YES	TX/FD
7439-96-5	Manganese	1.43		60.65		mg/kg	MS2-S	93/93	0.20-0.33	60.65	na	na	na	na	NO	NUT
7440-66-6	Zinc	7.30	J	85.46		mg/kg	MS5-S	93/93	0.50-0.82	85.46	na	na	na	na	NO	NUT

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

na not available

(1) Minimum/maximum detected concentration.

(2) Rationale Codes

Selection Reason:
- HIST: Infrequent Detection but Associated Historically
- FD: Frequent Detection
- TX: Toxicity Information Available
- ASL: Above Screening Levels

Deletion Reason:
- IFD: Infrequent Detection - less than 10 percent
- BKG: Background Levels
- NTX: No Toxicity Information
- NUT: Essential Nutrient
- BSL: Below Screening Level
- NHIST: Not Historically Associated with Source
- IFE: Infrequent Exceedance - less than 10 percent

Definitions:
- N/A = Not Applicable
- -- Compound has a 100% detection frequency
- COPC = Chemical of Potential Concern
- ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
- NE = Not Established
- J = Estimated Value
- c = Cancer endpoint
- na = not available
- STV = Screening Toxicity Value

TABLE 2.3.6

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN UNDISTURBED SURFACE WATER

CSM Unit 3

Scenario Timeframe:	Future
Medium:	Groundwater
Exposure Medium:	Undisturbed Surface Water
Exposure Point:	Lower Coeur d'Alene River

CAS Number	Chemical	Minimum (1) Conc.	Minimum Qualifier	Maximum (1) Conc.	Maximum Qualifier	Units	Location of Maximum Conc.	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value (3)	Screening (4) Toxicity Value	Potential ARAR/TBC Value (5)	Potential ARAR/TBC Source	COPC Flag	Rationale for (6) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	<50	U	<68.7	U	ug/L	N/A	0/2	50-68.7	<68.7	na	NE	50 - 200	SMCL	NO	NUT/LSMCL
7440-36-0	Antimony	0.79		<1.3	U	ug/L	N/A	1/2	1.3	<1.3	0.51	NE	6 / 4300	MCL / AWQC	NO	LMCL/LAWQC
7440-38-2	***Arsenic***	7		20		ug/L	LC2919	4/9	.029-1	20	0.65	NE	50 / 0.14	MCL / AWQC	YES	AAWQC
7440-39-3	Barium	5		24.7		ug/L	LC3	3/5	4	24.7	na	NE	2000	MCL	NO	LMCL
7440-41-7	Beryllium	<0.35	U	<1	U	ug/L	N/A	0/2	0.35-1	<1	na	NE	4	MCL	NO	LMCL
7440-43-9	***Cadmium***	1		21		ug/L	LC2919	92/93	3	21	0.09	NE	5	MCL	YES	AMCL
7440-70-2	Calcium	3100		8070		ug/L	LC3	4/4	--	8070	na	NE	na	na	NO	NUT
7440-47-3	Chromium	17		17		ug/L	LC8257	1/5	6-13	17	na	NE	100	MCL	NO	LMCL
7440-48-4	Cobalt	<5	U	<5	U	ug/L	N/A	0/2	5-Jan	<5	na	NE	na	na	NO	NTX
7440-50-8	Copper	1.4	J	43		ug/L	LC8257	5/9	3-35	43	1.21	NE	1300	MCL	NO	LMCL
7439-89-6	Iron	18		3700		ug/L	LC60	4/7	12-65	3700	113	NE	300	SMCL	NO	NUT
7439-92-1	***Lead***	2		430		ug/L	LC60	91/93	15	430	1.46	NE	15	MCL	YES	AMCL
7439-95-4	Magnesium	1100		2820		ug/L	LC3	4/4	--	2820	na	NE	na	na	NO	NUT
7439-96-5	***Manganese***	6		380		ug/L	LC60	5/7	2	380	8.28	NE	50 / 100	SMCL / AWQC	YES	ASMCL/AAWQC
7487-94-7	***Mercury***	<1	U	<5	U	ug/L	N/A	0/5	0.1-5	<5	0.09	NE	2 / 0.051 (7)	MCL / AWQC	YES	AMCL/AAWQC
7440-02-0	Nickel	<5	U	<20	U	ug/L	N/A	0/5	5-20	<20	na	NE	140 / 4600	MCL / AWQC	NO	LMCL/LAWQC
7440-09-7	Potassium	466		712	J	ug/L	LC3	2/2	--	712	na	NE	na	na	NO	NUT
7782-49-2	Selenium	<0.21	U	<1	U	ug/L	N/A	0/2	0.21-1	<1	na	NE	50 / 11000	MCL / AWQC	NO	LMCL/LAWQC
7440-22-4	Silver	<0.22	U	<0.3	U	ug/L	N/A	0/2	0.22-0.3	<0.3	0.12	NE	100	SMCL	NO	LSMCL
7440-23-5	Sodium	850		2040		ug/L	LC3	4/4	--	2040	na	NE	na	na	NO	NUT
7440-28-0	Thallium	<0.1	U	<0.2	U	ug/L	N/A	0/2	0.1-0.2	<0.2	na	NE	2 / 6.3	MCL / AWQC	NO	LMCL/LAWQC
7440-62-2	Vanadium	<2	U	<5	U	ug/L	N/A	0/2	2-5	<5	na	NE	na	na	NO	NTX
7440-66-6	Zinc	6		690		ug/L	LC55	90/92	3	690	20.71	NE	5000 / 69000	SMCL / AWQC	NO	LSMCL/LAWQC

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration.

(2) STV based on using surface water as a drinking water source.

(3) Background values as calculated by URS, March 2000.

(4) Screening toxicity values not established (NE).

(5) Potential ARARs are the Ambient Water Quality Criteria, human health for consumption of organisms only. EPA 1998c (Vol.63, No.237)

(6) Rationale Codes

Selection Reason:	HIST:	Infrequent Detection but Associated Historically
	FD:	Frequent Detection
	TX:	Toxicity Information Available
	ASL:	Above Screening Levels
	AMCL:	Above MCL
	ASMCL:	Above Secondary MCL
	AAWQC:	Above Ambient Water Quality Criteria
Deletion Reason:	IFD:	Infrequent Detection - less than 5 percent
	BKG:	Background Levels
	NTX:	No Toxicity Information
	NUT:	Essential Nutrient
	BSL:	Below Screening Level
	NHIST:	Not Historically Associated with Source
	IFE:	Infrequent Exceedance - less than 5 percent
	LMCL -	Less Than MCL
	LSMCL -	Less Than Secondary MCL
	LAWQC:	Less Than Ambient Water Quality Criteria

(7) AWQC listed for elemental mercury

Definitions:

< = less than the laboratory method detection limit listed (non-detect)
na = not available
-- Compound has a 100% detection frequency
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
AWQC = Ambient Water Quality Criteria Human Health Consumption of 'Organism Only'
NE = Not Established
J = Estimated Value
U - Compound was analyzed for, but not detected
c = Cancer endpoint
N/A = Not Applicable
MCL - Federal Maximum Contaminant Level
SMCL - Secondary Maximum Contaminant Level
STV = Screening Toxicity Value

TABLE 2.4.1

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN HOUSE DUST

CSM Units 1, 2, and 3

Scenario Timeframe:	Current
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	House Dust

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration (7)	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background (2) Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	654		22600		mg/kg	R011 / 107	160/160	--	22600	na	7500	na	na	NO	NUT
7440-36-0	***Antimony***	0.56		318		mg/kg	R022 / 207	160/160	--	318	5.8	3	na	na	YES	ASL
7440-38-2	***Arsenic***	1.5		635		mg/kg	R027 / 107	160/160	--	635	22	0.38 c	na	na	YES	ASL
7440-39-3	Barium	19		1110		mg/kg	R042 / 207	145/160	138 - 323	1110	1100	520	na	na	NO	IFE
7440-41-7	Beryllium	1		4		mg/kg	R084 / 107	18/160	1 - 2	4	2.1	15	na	na	NO	BSL
7440-43-9	***Cadmium***	2		375	J	mg/kg	R054 / 107	159/160	1	375	2.86	3.7	na	na	YES	ASL
7440-70-2	Calcium	2210		291000		mg/kg	R010 / 107	160/160	--	291000	1	NE	na	na	NO	NUT
7440-47-3	Chromium (5)	4		145		mg/kg	R031 / 207	160/160	--	145	64	210 c	na	na	NO	BSL
7440-48-4	Cobalt	2		26		mg/kg	R042 / 107	156/160	2 - 4	26	20	330	na	na	NO	BSL
7440-50-8	Copper	18		1040		mg/kg	R008 / 207	160/160	--	1040	53	280	na	na	NO	IFE
7439-89-6	***Iron***	303		60800		mg/kg	R025 / 107	160/160	--	60800	65000	2200	na	na	YES	ASL
7439-92-1	***Lead***	23		59500		mg/kg	R054 / 107	160/160	--	59500	175	400	na	na	YES	ASL
7439-95-4	Magnesium	584		6880		mg/kg	R068 / 207	160/160	--	6880	1.1	NE	na	na	NO	NUT
7439-96-5	***Manganese***	30		5460		mg/kg	R025 / 107	160/160	--	5460	3600	310	na	na	YES	ASL
7487-94-7	Mercury	0.06		21.5	J	mg/kg	R054 / 107	160/160	--	21.5	0.3	2.2	na	na	NO	IFE
7440-02-0	Nickel	10		72		mg/kg	R073 / 207	151/160	10 - 20	72	38	150	na	na	NO	IFE
7440-09-7	Potassium	770		19900		mg/kg	R020 / 207	160/160	--	19900	na	NE	na	na	NO	NUT
7782-49-2	Selenium	0.5		21		mg/kg	R027 / 107	56/160	1 - 2	21	na	37	na	na	NO	BSL
7440-22-4	Silver	2		126		mg/kg	R054 / 107	146/160	2 - 4	126	1.1	37	na	na	NO	IFE
7440-23-5	Sodium	253		841000		mg/kg	R047 / 207	160/160	--	841000	na	NE	na	na	NO	NUT
1314-32-5	Thallium (6)	0.02		1.31		mg/kg	R041 / 107	160/160	--	1.31	na	0.52	na	na	NO	IFE
7440-62-2	Vanadium	2		40		mg/kg	R041 / 107	158/160	2	40	154	52	na	na	NO	IFE
7440-66-6	***Zinc***	60		57500	J	mg/kg	R054 / 107	160/160	--	57500	280	2200	na	na	YES	ASL

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration.

(2) Background values derived from "Geochemical-Exploration Studies in the Coeur d'Alene District, Idaho and Montana" (Gott and Cathrall, 1980).

(3) Screening toxicity values are 0.1 times the EPA Region 9 Residential Soil PRGs (EPA 1998b) for noncarcinogens. Screening toxicity values for carcinogens, and for lead, are unchanged from the listed soil PRG.

(4) Rationale Codes

Selection Reason:	HIST:	Infrequent Detection but Associated Historically
	FD:	Frequent Detection
	TX:	Toxicity Information Available
	ASL:	Above Screening Levels
Deletion Reason:	IFD:	Infrequent Detection - less than 10 percent
	BKG:	Background Levels
	NTX:	No Toxicity Information
	NUT:	Essential Nutrient
	BSL:	Below Screening Level
	NHIST:	Not Historically Associated with Source
	IFE:	Infrequent Exceedance - less than 10 percent

(5) STV listed for Total Chromium (1/6 ratio Cr VI/Cr III)

(6) STV listed for Thallic Oxide (more conservative)

(7) Location ID 107 = floor mat sample; Location ID 207 = vacuum bag sample

Definitions:

N/A = Not Applicable
-- Compound has a 100% detection frequency
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
NE = Not Established
J = Estimated Value
c = Cancer endpoint
na = not available
STV = Screening Toxicity Value

TABLE 2.4.2

OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN AIR

CSM Units 1, 2, and 3

Scenario Timeframe:	Current
Medium:	Soil/Sediment
Exposure Medium:	Soil/Sediment
Exposure Point:	Air

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background (2) Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value ug/m^3	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
	Inorganics															
7429-90-5	Aluminum	1190	J	52000		mg/kg	CUA095	4002/4002	--	52000	na	NE	na	na	NO	NUT
7440-36-0	Antimony	0.21	J	623		mg/kg	SegBIG04	2966/4029	0.2 - 30	623	5.8	27,500	na	na	NO	BSL
7440-38-2	Arsenic	0.47	J	3610		mg/kg	CC/Tiger	4186/4208	0.18 - 15.2	3610	22	747	na	na	NO	IFE
7440-39-3	Barium	16.6	J	4300		mg/kg	CUA100	4002/4002	--	4300	1100	68,800	na	na	NO	BSL
7440-41-7	Beryllium	0.0784	J	9.7		mg/kg	CUA049	3116/4002	0.12 - 1.4	9.7	2.1	1340	na	na	NO	BSL
7440-43-9	Cadmium	0.06		194		mg/kg	SegNM04	3939/4208	0.06 - 3.6	194	2.7	1780	na	na	NO	BSL
7440-70-2	Calcium	232		99200		mg/kg	R035	4002/4002	--	99200	1	NE	na	na	NO	NUT
7440-47-3	Chromium (5)	0.124	J	198		mg/kg	R065	3960/4002	0.14 - 3.9	198	64	268	na	na	NO	BSL
7440-48-4	Cobalt	1.1		106		mg/kg	CUA049	3999/4002	8.4 - 8.7	106	20	2,750	na	na	NO	BSL
7440-50-8	Copper	2.1		1710		mg/kg	R065	4208/4208	--	1710	53	NE	na	na	NO	NTX
7439-89-6	Iron	3190		256000		mg/kg	CUA057	4002/4002	--	256000	6.5	NE	na	na	NO	NUT
7439-92-1	***Lead***	9.9		67100		mg/kg	SegCC05	4208/4208	--	67100	171	NE	1.5	NAAQCs	YES	ASL
7439-95-4	Magnesium	274	J	29700		mg/kg	R019	4002/4002	--	29700	1.1	NE	na	na	NO	NUT
7439-96-5	Manganese	45.6		26400		mg/kg	CUA058	4002/4002	--	26400	3600	6,880	na	na	NO	IFE
7487-94-7	Mercury	0.0474	J	47.3		mg/kg	R086	3570/4208	0.0478 - 0.5	47.3	0.3	41,300	na	na	NO	BSL
7440-02-0	Nickel	0.9		148		mg/kg	R065	3983/4002	2.7 - 23.9	148	38	13,400	na	na	NO	BSL
7440-09-7	Potassium	147		9870		mg/kg	R073	4001/4002	1110	9870	na	NE	na	na	NO	NUT
7782-49-2	Selenium	0.34	J	11.4		mg/kg	CUA077	1182/3890	0.2 - 2.6	11.4	na	NE	na	na	NO	NTX
7440-22-4	Silver	0.226	J	347		mg/kg	SegCC02	3541/4000	0.14 - 5.7	347	1.1	NE	na	na	NO	NTX
7440-23-5	Sodium	10.6	J	5690		mg/kg	CC/Tamarack7	3845/4002	20.7 - 489	5690	na	NE	na	na	NO	NUT
1314-32-5	Thallium (6)	0.24	J	14.4		mg/kg	SegUG01	633/3898	0.38 - 9.8	14.4	na	NE	na	na	NO	NTX
7440-62-2	Vanadium	0.39		106		mg/kg	CUA049	3945/4002	0.39 - 19.3	106	154	NE	na	na	NO	BKG / NTX
7440-66-6	Zinc	14.3		25800		mg/kg	CC/Tamarack7	4208/4208	--	25800	280	NE	na	na	NO	NTX

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

(1) Minimum/maximum detected concentration.

(2) Background values derived from "Geochemical-Exploration Studies in the Coeur d'Alene District, Idaho and Montana" (Gott and Cathrall, 1980).

(3) Screening toxicity values were derived using the PEF formula as described in the Soil Screening Guidance (EPA 1996a), see text.

(4) Rationale Codes

Selection Reason:	HIST:	Infrequent Detection but Associated Historically
	FD:	Frequent Detection
	TX:	Toxicity Information Available
	ASL:	Above Screening Levels
Deletion Reason:	IFD:	Infrequent Detection - less than 10 percent
	BKG:	Background Levels
	NTX:	No Toxicity Information
	NUT:	Essential Nutrient
	BSL:	Below Screening Level
	NHIST:	Not Historically Associated with Source
	IFE:	Infrequent Exceedance - less than 10 percent

(5) STV listed for Total Chromium (1/6 ratio Cr VI/Cr III)

(6) STV listed for Thallic Oxide (more conservative)

Definitions:

N/A = Not Applicable
-- Compound has a 100% detection frequency
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
NE = Not Established; no inhalation toxicity criteria
J = Estimated Value
c = Cancer endpoint
na = not available
STV = Screening Toxicity Value

TABLE 2.4.3
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN HOME GROWN VEGETABLES
CSM Units 1, 2, and 3

Scenario Timeframe:	Current
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Homegrown Vegetables

CAS Number	Chemical	Minimum Concentration (1)	Minimum Qualifier	Maximum Concentration (1)	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value	Screening Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for Contaminant Deletion or Selection (2)
	Inorganics															
7440-38-2	***Arsenic***	0.01		0.11		mg/kg	R064	19/35	0.002-0.02	0.11	na	na	na	na	YES	TX/FD
7440-43-9	***Cadmium***	0.02		1.85		mg/kg	R062	35/35	--	1.85	na	na	na	na	YES	TX/FD
7439-92-1	***Lead***	0.01		3.71		mg/kg	R022	35/35	--	3.71	na	na	na	na	YES	TX/FD

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

na not available

(1) Minimum/maximum detected concentration.

(2) Rationale Codes

Selection Reason:
- HIST: Infrequent Detection but Associated Historically
- FD: Frequent Detection
- TX: Toxicity Information Available
- ASL: Above Screening Levels

Deletion Reason:
- IFD: Infrequent Detection - less than 10 percent
- BKG: Background Levels
- NTX: No Toxicity Information
- NUT: Essential Nutrient
- BSL: Below Screening Level
- NHIST: Not Historically Associated with Source
- IFE: Infrequent Exceedance - less than 10 percent

Definitions:
- N/A = Not Applicable
- -- Compound has a 100% detection frequency
- COPC = Chemical of Potential Concern
- ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
- NE = Not Established
- J = Estimated Value
- c = Cancer endpoint
- na = not available
- STV = Screening Toxicity Value

TABLE 2.4.4
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN IN FISH FROM COEUR D'ALENE RIVER
CSM Units 1, 2, and 3

Scenario Timeframe:	Current
Medium:	Surface Water and Sediment
Exposure Medium:	Fish
Exposure Point:	Fish from Coeur d'Alene River

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value	Screening Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (2) Contaminant Deletion or Selection
	Inorganics															
Bullhead																
7440-43-9	***Cadmium***	0.017		0.069		mg/kg	Killarney Lake	18/126	0.005	0.069	na	na	na	na	YES	TX/FD
7487-94-7	***Mercury***	0.05		0.21		mg/kg	Killarney Lake	63/126	0.025-0.03	0.21	na	na	na	na	YES	TX/FD
Northern Pike																
7440-43-9	***Cadmium***	0.011		0.02		mg/kg	Thompson Lake	6/63	0.005	0.02	na	na	na	na	YES	TX/FD
7487-94-7	***Mercury***	0.05		0.48		mg/kg	Thompson Lake	50/63	0.025-0.03	0.48	na	na	na	na	YES	TX/FD
Perch																
7440-43-9	***Cadmium***	0.01		0.169		mg/kg	Killarney Lake	91/123	0.005	0.169	na	na	na	na	YES	TX/FD
7487-94-7	***Mercury***	0.05		0.23		mg/kg	Medicine Lake	84/123	0.025-0.055	0.23	na	na	na	na	YES	TX/FD

NOTES:

Chemicals bolded and italicized exceeded their screening toxicity value.

na not available

(1) Minimum/maximum detected concentration.

(2) Rationale Codes

Selection Reason:
- HIST: Infrequent Detection but Associated Historically
- FD: Frequent Detection
- TX: Toxicity Information Available
- ASL: Above Screening Levels

Deletion Reason:
- IFD: Infrequent Detection - less than 10 percent
- BKG: Background Levels
- NTX: No Toxicity Information
- NUT: Essential Nutrient
- BSL: Below Screening Level
- NHIST: Not Historically Associated with Source
- IFE: Infrequent Exceedance - less than 10 percent

Definitions:
- N/A = Not Applicable
- -- Compound has a 100% detection frequency
- COPC = Chemical of Potential Concern
- ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
- NE = Not Established
- J = Estimated Value
- c = Cancer endpoint
- na = not available
- STV = Screening Toxicity Value

TABLE 3.1.1

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	5.62	8.17	25.6		mg/kg	8.25	95% UCL-NP	W-Test (1)	5.62	Mean -N	W-Test (1)
Arsenic	mg/kg	35.47	48.03	115		mg/kg	48.53	95% UCL-NP	W-Test (1)	35.47	Mean -N	W-Test (1)
Cadmium	mg/kg	4.97	6.88	15.5		mg/kg	6.87	95% UCL-NP	W-Test (1)	4.97	Mean -N	W-Test (1)
Iron	mg/kg	29992	38186	93000		mg/kg	37703	95% UCL-NP	W-Test (1)	29992	Mean -N	W-Test (1)
Manganese	mg/kg	1639	2342	6620	J	mg/kg	2292	95% UCL-NP	W-Test (1)	1639	Mean -N	W-Test (1)
Zinc	mg/kg	849	1185	2900		mg/kg	1199	95% UCL-NP	W-Test (1)	849	Mean -N	W-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatior

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

J: The associated value is an estimated quantity.

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W Test indicates data are neither normally nor lognormally distributed.

TABLE 3.1.2

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap Water

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	0.38	0.84	0.97		ug/L	0.97	Max	(1)	0.38	Mean-N	(2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

Static and purged tap water results were averaged.

Normal UCL values calculated using the formula: average + [t-statistic*(std deviation/count^0.5)]

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Distribution of the data could not be determined because less than 10 samples were available; therefore, the maximum concentration was used as the Reasonable Maximum Exposure EPC.

(2) Distribution of the data could not be determined because less than 10 samples were available; therefore, the arithmetic mean was used as the Central Tendency EPC.

TABLE 3.1.3

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Lower Basin - Neighborhood and Public Recreational Exposure Scenarios

Scenario Timeframe:	Current/Future
Medium:	Soil/Sediment
Exposure Medium:	Soil/Sediment
Exposure Point:	Soil/Sediment at Lower CDAR*

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	22.08	23.22	73.7		mg/kg	23.22	95% UCL-N	D-Test (2)	22.08	Mean-N	D-Test (2)
Arsenic	mg/kg	114.51	119.72	492	J	mg/kg	119.45	95% UCL-NP	D-Test (1)	114.51	Mean-N	D-Test (1)
Cadmium	mg/kg	33.14	35.13	105		mg/kg	35.05	95% UCL-NP	D-Test (1)	33.14	Mean-N	D-Test (1)
Iron	mg/kg	101280	105569	256000		mg/kg	105451	95% UCL-NP	D-Test (1)	101280	Mean-N	D-Test (1)
Manganese	mg/kg	9403	9886	26400		mg/kg	9886	95% UCL-N	D-Test (2)	9403	Mean-N	D-Test (2)
Zinc	mg/kg	5337	5672	21800		mg/kg	5666	95% UCL-NP	D-Test (1)	5337	Mean-N	D-Test (1)

NOTES:

* Floodplain soil and sediment near the Lower Coeur d'Alene River

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

J: The associated value is an estimated quantity.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gilbert, 1987).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

(2) D'Agostino's Test indicates data are normally distributed.

TABLE 3.1.4

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Lower Basin - Neighborhood and Public Recreational Exposure Scenarios

Scenario Timeframe:	Current/Future
Medium:	Surface Water
Exposure Medium:	Surface Water
Exposure Point:	Disturbed Surface Water at Lower CDAR

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	68.02	79.08	600	J	ug/L	79.61	95% UCL-NP	D-Test (1)	75.64	Mean-T	D-Test (1)
Cadmium	ug/L	54.82	72.84	1200	J	ug/L	72.9	95% UCL-NP	D-Test (2)	54.82	Mean-N	D-Test (2)
Manganese	ug/L	14678	16878	84900		ug/L	16651	95% UCL-NP	D-Test (1)	21269	Mean-T	D-Test (1)
Mercury	ug/L	3.62	4.5	43.9		ug/L	4.5	95% UCL-NP	D-Test (1)	4.51	Mean-T	D-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

J: The associated value is an estimated quantity.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring.Gilbert, 1987).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are lognormally distributed.

(2) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

TABLE 3.1.5

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Lower Basin - Subsistence Exposure Scenerio

Scenario Timeframe:	Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Sediment at Lower CDAR *

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	23.62	25.2	73.7		mg/kg	25.2	95% UCL-N	D-Test (1)	23.62	Mean-N	D-Test (1)
Arsenic	mg/kg	113.77	120.22	375		mg/kg	120.96	95% UCL-NP	D-Test (2)	113.77	Mean-N	D-Test (2)
Cadmium	mg/kg	36.49	39.33	105		mg/kg	39.33	95% UCL-N	D-Test (1)	36.49	Mean-N	D-Test (1)
Iron	mg/kg	107364	113412	256000		mg/kg	113073	95% UCL-NP	D-Test (2)	107364	Mean-N	D-Test (2)
Manganese	mg/kg	10082	10767	26400		mg/kg	10700	95% UCL-NP	D-Test (2)	10082	Mean-N	D-Test (2)
Zinc	mg/kg	5914	6398	21800		mg/kg	6400	95% UCL-NP	D-Test (2)	5914	Mean-N	D-Test (2)

NOTES:

* Sediment along the Lower Coeur d'Alene River

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatior

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring.Gilbert, 19

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are normally distributed.

(2) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

TABLE 3.1.6
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Lower Basin - Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface Water/Sediment
Exposure Medium:	Plant Tissue
Exposure Point:	Water Potatoes

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
With Skin												
Cadmium	mg/kg	0.39	0.48	3.7092		mg/kg	0.489	95% UCL-NP	D-Test (1)	0.38	Mean-T	D-Test (1)
Without Skin												
Cadmium	mg/kg	0.23	0.29	2.7978		mg/kg	0.291	95% UCL-NP	D-Test (1)	0.21	Mean-T	D-Test (1)

NOTES:
For ND - 1/2 the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.
D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and
Statistical Methods for Environmental Pollution Monitoring.Gilbert, 1987).
Normal UCL values calculated using MTCA Stat v*2.1* .
Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are lognormally distributed.

TABLE 3.1.7

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Lower Basin - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium: Soil	Soil
Exposure Medium:	Surface/Subsurface Soil
Exposure Point:	Construction Site Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	19.98	21.08	73.7		mg/kg	21.08	95% UCL-N	D-Test (1)	19.98	Mean -N	D-Test (1)
Arsenic	mg/kg	104.41	109.4	492	J	mg/kg	108.91	95% UCL-NP	D-Test (2)	104.41	Mean -N	D-Test (2)
Cadmium	mg/kg	29	30.87	105		mg/kg	30.71	95% UCL-NP	D-Test (2)	29	Mean -N	D-Test (2)
Iron	mg/kg	92917	97108	256000		mg/kg	97012	95% UCL-NP	D-Test (2)	92917	Mean -N	D-Test (2)
Manganese	mg/kg	8449	8919	26400		mg/kg	8919	95% UCL-N	D-Test (1)	8449	Mean -N	D-Test (1)
Zinc	mg/kg	4703	5013	21800		mg/kg	4998	95% UCL-NP	D-Test (2)	4703	Mean -N	D-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatio

J: The associated value is an estimated quantity.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring.Gilbert, 19

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Normal Data (Mean-N)
Mean of Log-transformed Data (Mean-T)

(1) D'Agostino's Test indicates data are normally distributed.

(2) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

TABLE 3.1.8

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Lower Basin - Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Lower CDAR

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	19.76	21.31	58.6	J	mg/kg	21.16	95% UCL-NP	D-Test (1)	19.76	Mean -N	D-Test (1)
Arsenic	mg/kg	115.62	124.35	492	J	mg/kg	124.44	95% UCL-NP	D-Test (1)	115.62	Mean -N	D-Test (1)
Cadmium	mg/kg	28.09	30.55	86.4		mg/kg	30.45	95% UCL-NP	D-Test (1)	28.09	Mean -N	D-Test (1)
Iron	mg/kg	92135	97647	222000		mg/kg	97440	95% UCL-NP	D-Test (1)	92135	Mean -N	D-Test (1)
Manganese	mg/kg	8382	8997	25200		mg/kg	8960	95% UCL-NP	D-Test (1)	8382	Mean -N	D-Test (1)
Zinc	mg/kg	4470	4860	14200	J	mg/kg	4856	95% UCL-NP	D-Test (1)	4470	Mean -N	D-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring.Gilbert, 1987).

J: The associated value is an estimated quantity.

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

TABLE 3.1.9

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Lower Basin - Subsistence Exposure Scenarios

Scenario Timeframe:	Current/Future
Medium:	Surface Water
Exposure Medium:	Surface Water
Exposure Point:	Undisturbed Surface Water at Lower CDAR

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	11.59	16.06	20		ug/L	20	Max	(2)	11.59	Mean-N	(3)
Cadmium	ug/L	2.66	3.15	21		ug/L	3.23	95% UCL-NP	D-Test (1)	2.66	Mean-N	D-Test (1)
Lead	ug/L	37.96	na	430		ug/L	na	na	D-Test (1)	na	na	D-Test (1)
Manganese	ug/L	109.47	214.05	380		ug/L	380	Max	(2)	109.47	Mean-N	(3)
Mercury	ug/L	1.53	2.8	2.5	U	ug/L	2.5	Max	(2)	1.53	Mean-N	(3)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

na - not applicable

U: The analyte was not detected above the reported sample detection limit.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring.Gilbert, 1987).

Shapiro-Wilks W Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGS: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

(2) Distribution of the data cannot be determined with accuracy because less than 10 samples were available; therefore, the maximum concentration was used as the Reasonable Maximum Exposure EPC.

(3) Distribution of the data cannot be determined with accuracy because less than 10 samples were available; therefore, the arithmetic mean was used as the Central Tendancy EPC.

TABLE 3.2.1

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	3.05	4.03	25.8	J	mg/kg	4.06	95% UCL-NP	D-Test (1)	3.05	Mean -N	D-Test (1)
Arsenic	mg/kg	20.35	24.76	105		mg/kg	25.04	95% UCL-NP	D-Test (1)	20.35	Mean -N	D-Test (1)
Cadmium	mg/kg	3.2	3.94	26		mg/kg	3.92	95% UCL-NP	D-Test (2)	3.59	Mean -T	D-Test (2)
Iron	mg/kg	19198	21585	96300		mg/kg	21971	95% UCL-NP	D-Test (1)	19198	Mean -N	D-Test (1)
Manganese	mg/kg	783	1042	9790		mg/kg	1085	95% UCL-NP	D-Test (1)	783	Mean -N	D-Test (1)
Zinc	mg/kg	506	700	8150		mg/kg	719	95% UCL-NP	D-Test (2)	449	Mean -T	D-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation

J: The associated value is an estimated quantity.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring.Gilbert, 19

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

(2) D'Agostino's Test indicates data are lognormally distributed.

TABLE 3.2.2

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap Water

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	0.21	0.29	0.405		ug/L	0.405	Max	(1)	0.21	Mean-N	(2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

Static and purged tap water results were averaged.
Normal UCL values calculated using MTCA Stat *v2.1* .

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Distribution of the data could not be determined because less than 10 samples were available; therefore, the maximum concentration was used as the Reasonable Maximum Exposure EPC.

(2) Distribution of the data could not be determined because less than 10 samples were available; therefore, the arithmetic mean was used as the Central Tendency EPC.

TABLE 3.2.3

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Sediment at Pine Creek

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	7.05	12.78	31.8	J	mg/kg	12.83	95% UCL-NP	W-Test (1)	7.05	Mean-N	W-Test (1)
Arsenic	mg/kg	15.34	23.23	60.6	J	mg/kg	22.35	95% UCL-NP	W-Test (2)	15.84	Mean-T	W-Test (2)
Cadmium	mg/kg	0.81	0.95	1.25	J	mg/kg	0.92	95% UCL-NP	W-Test (2)	0.82	Mean-T	W-Test (2)
Iron	mg/kg	14559	16678	24200	J	mg/kg	16300	95% UCL-NP	W-Test (2)	14622	Mean-T	W-Test (2)
Manganese	mg/kg	389.08	465.1	739	J	mg/kg	451.62	95% UCL-NP	W-Test (2)	392.21	Mean-T	W-Test (2)
Zinc	mg/kg	284.62	330.6	452	J	mg/kg	320.31	95% UCL-NP	W-Test (2)	285.89	Mean-T	W-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatior

J: The associated value is an estimated quantity.

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W Test indicates data are neither normally nor lognormally distributed.

(2) Shapiro-Wilk W Test indicates data are lognormally distributed.

TABLE 3.2.4

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface Water
Exposure Medium:	Surface Water
Exposure Point:	Surface Water at Pine Creek

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	0.57	0.78	1		ug/L	0.73	95% UCL-NP	W-Test (2)	0.57	Mean-N	W-Test (2)
Cadmium	ug/L	0.58	1.12	3.7		ug/L	1.08	95% UCL-NP	W-Test (1)	0.58	Mean-T	W-Test (1)
Manganese	ug/L	16.26	37.25	144		ug/L	38.46	95% UCL-NP	W-Test (2)	16.26	Mean-N	W-Test (2)
Mercury	ug/L	0.08	0.09	0.1	U	ug/L	0.09	95% UCL-NP	W-Test (2)	0.08	Mean-N	W-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calcula

U: The analyte was not detected above the reported sample detection limit.

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilk W Test indicates data are lognormally distributed.

(2) Shapiro-Wilks W Test indicates data are neither normally nor lognormally distributed.

TABLE 3.2.5

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Kingston - Neighborhood and Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/Sediment
Exposure Medium:	Soil/Sediment
Exposure Point:	Soil/Sediment at NS Confluence*

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	34.33	39.57	64.3		mg/kg	39.78	95% UCL-NP	W-Test (2)	34.47	Mean-T	W-Test (2)
Arsenic	mg/kg	145.15	163.27	266		mg/kg	163.2	95% UCL-NP	W-Test (2)	145.44	Mean-T	W-Test (2)
Cadmium	mg/kg	22.03	26.48	63.3		mg/kg	26.58	95% UCL-NP	W-Test (1)	22.03	Mean-N	W-Test (1)
Iron	mg/kg	89053	100738	174000		mg/kg	100621	95% UCL-NP	W-Test (2)	89354	Mean-T	W-Test (2)
Manganese	mg/kg	7431	8617	14800		mg/kg	8585	95% UCL-NP	W-Test (2)	7467	Mean-T	W-Test (2)
Zinc	mg/kg	3283	3727	6260		mg/kg	3744	95% UCL-NP	W-Test (2)	3286	Mean-T	W-Test (2)

NOTES:

* Surface soil and beach sediments near the confluence of the North and South Forks of the Coeur d'Alene River.

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatior

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics:
Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W-Test indicates data are neither normally nor lognormally distributed.

(2) Shapiro-Wilks W-Test indicates data are lognormally distributed.

TABLE 3.2.6
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Kingston - Neighborhood and Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface Water
Exposure Medium:	Surface Water
Exposure Point:	Surface Water at NS Confluence*

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	36.4	89.1	134	J	ug/L	134	Max	(1)	36.4	Mean-N	(1)
Cadmium	ug/L	29.5	58.97	83.8		ug/L	83.8	Max	(1)	29.5	Mean-N	(1)
Manganese	ug/L	3145	6613	9470		ug/L	9470	Max	(1)	3145	Mean-N	(1)
Mercury	ug/L	1.3	3.15	4.6		ug/L	4.6	Max	(1)	1.3	Mean-N	(1)

NOTES:

* Surface water at the confluence of the North and South Forks of the Coeur d'Alene River.

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation

J: The associated value is an estimated quantity.

Normal UCL values calculated using MTCA Stat *v2.1* .

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Distribution of the data could not be determinied because only 5 samples were available; therefore, the maximum concentration was used for the EPC.

TABLE 3.2.7
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Kingston - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/Subsurface Soil
Exposure Point:	Construction Site Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	5.51	6.78	64.3		mg/kg	6.86	95% UCL-NP	D-Test (1)	5.51	Mean -N	D-Test (1)
Arsenic	mg/kg	29.06	33.8	266		mg/kg	34.6	95% UCL-NP	D-Test (1)	29.06	Mean -N	D-Test (1)
Cadmium	mg/kg	4.24	5.01	63.3		mg/kg	5.1	95% UCL-NP	D-Test (2)	5.14	Mean -T	D-Test (2)
Iron	mg/kg	25811	28783	174000		mg/kg	29003	95% UCL-NP	D-Test (1)	25811	Mean -N	D-Test (1)
Manganese	mg/kg	1377	1666	14800		mg/kg	1693	95% UCL-NP	D-Test (1)	1377	Mean -N	D-Test (1)
Zinc	mg/kg	794	1018	24300		mg/kg	1057	95% UCL-NP	D-Test (1)	794	Mean -N	D-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring.Gilbert, 198

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

(2) D'Agostino's Test indicates data are lognormally distributed.

TABLE 3.3.1
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	10.26	16.41	248	J	mg/kg	15.94	95% UCL-NP	W-Test (1)	10.26	Mean - N	W-Test (1)
Arsenic	mg/kg	39.66	50.76	336		mg/kg	50.74	95% UCL-NP	W-Test (1)	39.66	Mean - N	W-Test (1)
Cadmium	mg/kg	6.95	9.45	115		mg/kg	9.62	95% UCL-NP	W-Test (1)	6.95	Mean - N	W-Test (1)
Iron	mg/kg	24797	27285	95700		mg/kg	27190	95% UCL-NP	W-Test (1)	24797	Mean - N	W-Test (1)
Manganese	mg/kg	1168	1383	7860		mg/kg	1367	95% UCL-NP	W-Test (1)	1168	Mean - N	W-Test (1)
Zinc	mg/kg	844	1281	20400	J	mg/kg	1320	95% UCL-NP	W-Test (2)	844	Mean - T	W-Test (2)

NOTES:

For ND - 1/2 the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.
W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).
Normal UCL values calculated using MTCA Stat *v2.1* .
Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W-Test indicates data are neither normally nor lognormally distributed.
(2) Shapiro-Wilk W-Test indicates data are lognormally distributed.

TABLE 3.3.2

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap Water

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	1.92	3.78	8.4		ug/L	8.4	Max	(1)	2.18	Mean-T	(2)

NOTES:

For nondetect results, 1/2 the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the higher concentration or the lower SQL was used in the calculation.

Static and purged tap water results were averaged.

Normal UCL value calculated using MTCA Stat *v2.1* .

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Distribution of the data could not be determined because less than 10 samples were available; therefore, the maximum concentration was used as the Reasonable Maximum Exposure EPC.

(2) Distribution of the data could not be determined because less than 10 samples were available; therefore, the lognormal mean was used as the Central Tendency EPC.

TABLE 3.3.3
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Surface Soil at Elk Creek Area

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	46.22	55.54	54.9	J	mg/kg	54.9	Max	(1)	46.22	Mean-N	(1)
Arsenic	mg/kg	79.96	91.9	98.5		mg/kg	98.5	Max	(1)	79.96	Mean-N	(1)
Cadmium	mg/kg	21.8	44.66	64.5		mg/kg	64.5	Max	(1)	21.8	Mean-N	(1)
Iron	mg/kg	85590	96951	99928		mg/kg	99928	Max	(1)	85590	Mean-N	(1)
Manganese	mg/kg	6786	9063	9280		mg/kg	9280	Max	(1)	6786	Mean-N	(1)
Zinc	mg/kg	3328	4919	6130		mg/kg	6130	Max	(1)	3328	Mean-N	(1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

J: The associated value is an estimated quantity.

Normal UCL values calculated using MTCA Stat *v2.1* .

Statistics:
Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Distribution of the data could not be determined because less than 10 samples were available; therefore, the maximum concentration was used for the EPC.

TABLE 3.3.4

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Sediment at Elk Creek Pond

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	17.68	27.84	47.1	J	mg/kg	27.84	95% UCL-N	W-Test (2)	17.68	Mean-N	W-Test (2)
Arsenic	mg/kg	62.28	79.5	113	J	mg/kg	77.74	95% UCL-NP	W-Test (3)	63.76	Mean-T	W-Test (3)
Cadmium	mg/kg	18.66	39.32	112		mg/kg	38.38	95% UCL-NP	W-Test (3)	42.77	Mean-T	W-Test (3)
Iron	mg/kg	29423	36226	52200		mg/kg	35970	95% UCL-NP	W-Test (1)	29423	Mean-N	W-Test (1)
Manganese	mg/kg	1468	2275	4410		mg/kg	2223	95% UCL-NP	W-Test (3)	1486	Mean-T	W-Test (3)
Zinc	mg/kg	2829	5099	10500		mg/kg	5031	95% UCL-NP	W-Test (3)	4222	Mean-T	W-Test (3)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

J: The associated value is an estimated quantity.

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W-Test indicates data are neither normally nor lognormally distributed.
(2) Shapiro-Wilks W-Test indicates data are normally distributed.
(3) Shapiro-Wilks W-Test indicates data are lognormally distributed.

TABLE 3.3.5
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface Water
Exposure Medium:	Surface Water
Exposure Point:	Surface Water at Elk Creek Pond

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	16.64	27.72	32		ug/L	32	Max	(1)	16.64	Mean-N	(1)
Cadmium	ug/L	26.44	46.96	60.3		ug/L	60.3	Max	(1)	26.44	Mean-N	(1)
Manganese	ug/L	2297	5690	8570		ug/L	8570	Max	(1)	2297	Mean-N	(1)
Mercury	ug/L	0.85	2.27	3.5		ug/L	3.5	Max	(1)	0.85	Mean-N	(1)

NOTES:
For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.
Normal UCL values calculated using MTCA Stat *v2.1* .

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Distribution of the data could not be determined because less than 10 samples were available; therefore, the maximum concentration was used for the EPC.

TABLE 3.4.1
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	10.06	12.63	248		mg/kg	12.54	95% UCL-NP	D-Test (1)	10.06	Mean-N	D-Test (1)
Arsenic	mg/kg	38.95	46.39	890		mg/kg	46.74	95% UCL-NP	D-Test (1)	38.95	Mean-N	D-Test (1)
Cadmium	mg/kg	4.81	5.23	26.2		mg/kg	5.25	95% UCL-NP	D-Test (1)	4.81	Mean-N	D-Test (1)
Iron	mg/kg	21454	22496	99200		mg/kg	22488	95% UCL-NP	D-Test (1)	21454	Mean-N	D-Test (1)
Manganese	mg/kg	1122	1202	5950		mg/kg	1199	95% UCL-NP	D-Test (1)	1122	Mean-N	D-Test (1)
Zinc	mg/kg	545	599	3560		mg/kg	597	95% UCL-NP	D-Test (2)	523	Mean-T	D-Test (2)

NOTES:

For ND - 1/2 the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gilbert 1987).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

(2) D'Agostino's Test indicates data is lognormally distributed.

TABLE 3.4.2

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap Water

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	0.23	0.28	0.9		ug/L	0.28	95% UCL-NP	W-Test (1)	0.23	Mean-N	W-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculati

Static and purged tap water results were averaged.

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W Test indicates data are neither normally nor lognormally distributed.

TABLE 3.5.1
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	5.82	8.72	82.5		mg/kg	8.9	95% UCL-NP	D-Test (1)	4.89	Mean - T	D-Test (1)
Arsenic	mg/kg	16.52	20.62	133		mg/kg	21.46	95% UCL-NP	D-Test (2)	16.52	Mean - N	D-Test (2)
Cadmium	mg/kg	4.4	5.31	23.8		mg/kg	5.44	95% UCL-NP	D-Test (1)	4.41	Mean - T	D-Test (1)
Iron	mg/kg	18361	20176	56700		mg/kg	20198	95% UCL-NP	D-Test (2)	18361	Mean - N	D-Test (2)
Manganese	mg/kg	928	1066	3440		mg/kg	1068	95% UCL-NP	D-Test (2)	928	Mean - N	D-Test (2)
Zinc	mg/kg	595	777	5000		mg/kg	816	95% UCL-NP	D-Test (1)	561	Mean - T	D-Test (1)

NOTES:

For ND - 1/2 the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gilbert 198

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are lognormally distributed.

(2) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

TABLE 3.5.2

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap Water

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	0.38	0.51	0.545		ug/L	0.545	Max	(1)	0.38	Mean-N	(2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

Static and purged tap water results were averaged.

Normal UCL values calculated using the formula: average + [t-statistic*(std deviation/count^0.5)]

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Distribution of the data could not be determined because less than 10 samples were available; therefore, the maximum concentration was used as the Reasonable Maximum Exposure EPC.

(2) Distribution of the data could not be determined because less than 10 samples were available; therefore, the arithmetic mean was used as the Central Tendency EPC.

TABLE 3.5.3
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Silverton - Neighborhood and Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Upland Parks/Schools

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	5.72	6.86	233		mg/kg	6.94	95% UCL-NP	(1)	4.28	Mean-T	(1)
Arsenic	mg/kg	23.18	27.9	1060		mg/kg	28.44	95% UCL-NP	(1)	16.88	Mean-T	(1)
Cadmium	mg/kg	3.99	4.53	90.8		mg/kg	4.57	95% UCL-NP	(1)	5.10	Mean-T	(1)
Iron	mg/kg	19535	20025	86000		mg/kg	20025	95% UCL-N	(2)	19535	Mean-N	(2)
Manganese	mg/kg	1039	1089	10000		mg/kg	1090	95% UCL-NP	(1)	1035	Mean-T	(1)
Zinc	mg/kg	633	731	18000		mg/kg	739	95% UCL-NP	(1)	537.23	Mean-T	(1)

NOTES:

For ND - 1/2 the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

Normal UCL values calculated using the formula: average + [t-statistic*(std deviation/count^0.5)]

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9*. This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics:
Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Sample size > 500; however, MTCA Stat checks distribution for sample sizes < 500. Therefore, data plotted on histograms using SYSTATv9. Distribution of the data appear lognormal.

(2) Sample size > 500; distribution of the iron data appear normal based on the histogram generated using SYSTATv9.

TABLE 3.6.1
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	8.81	13.35	161		mg/kg	13.7	95% UCL-NP	D-Test (1)	8.81	Mean - N	D-Test (1)
Arsenic	mg/kg	20.38	22.61	66.1		mg/kg	22.57	95% UCL-NP	D-Test (2)	20.32	Mean - T	D-Test (2)
Cadmium	mg/kg	6.46	7.68	46.8		mg/kg	7.63	95% UCL-NP	D-Test (1)	6.46	Mean - N	D-Test (1)
Iron	mg/kg	21149	22281	46700		mg/kg	22240	95% UCL-NP	D-Test (1)	21149	Mean - N	D-Test (1)
Manganese	mg/kg	895	990	3470		mg/kg	989	95% UCL-NP	D-Test (1)	895	Mean - N	D-Test (1)
Zinc	mg/kg	858	975	3150		mg/kg	967	95% UCL-NP	D-Test (2)	858	Mean - T	D-Test (2)

NOTES:

For ND - 1/2 the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gilbert 198

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

(2) D'Agostino's Test indicates data are lognormally distributed.

TABLE 3.6.2

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap Water

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	0.42	0.5	3.8		ug/L	0.494	95% UCL-NP	W-Test (1)	0.42	Mean-T	W-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatior

Static and purged tap water results were averaged.

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W-Test indicates data are lognormally distributed.

TABLE 3.6.3
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Wallace - Neighborhood and Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Upland Parks/Schools

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	16.03	18.74	261	J	mg/kg	18.22	95% UCL-NP	D-Test (2)	17.83	Mean-T	D-Test (2)
Arsenic	mg/kg	25.8	27.78	168		mg/kg	27.65	95% UCL-NP	D-Test (1)	25.8	Mean-N	D-Test (1)
Cadmium	mg/kg	9.64	10.73	64.1		mg/kg	10.72	95% UCL-NP	D-Test (1)	9.64	Mean-N	D-Test (1)
Iron	mg/kg	27036	28539	152000		mg/kg	28639	95% UCL-NP	D-Test (1)	27036	Mean-N	D-Test (1)
Manganese	mg/kg	1366	1515	18200		mg/kg	1502	95% UCL-NP	D-Test (1)	1366	Mean-N	D-Test (1)
Zinc	mg/kg	1681	1895	14655	J	mg/kg	1921	95% UCL-NP	D-Test (2)	1775	Mean-T	D-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatior

J: The associated value is an estimated quantity.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring.Gilbert, 19

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics:
Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are neither normally nor lognormally distributed.
(2) D'Agostino's Test indicates data are lognormally distributed.

TABLE 3.7.1

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Osburn, Wallace, and Silverton Combined - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Sediment at South Fork CDAR

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	22.03	35.86	174		mg/kg	38.11	95% UCL-NP	W-Test (1)	25.24	Mean-T	W-Test (1)
Arsenic	mg/kg	36.01	52.25	184	J	mg/kg	52.64	95% UCL-NP	W-Test (1)	36.81	Mean-T	W-Test (1)
Cadmium	mg/kg	14.16	19.16	64.4		mg/kg	18.89	95% UCL-NP	W-Test (2)	14.16	Mean-N	W-Test (2)
Iron	mg/kg	38100	42182	57200		mg/kg	41314	95% UCL-NP	W-Test (1)	38174	Mean-T	W-Test (1)
Manganese	mg/kg	3310	3816	6620	J	mg/kg	3755	95% UCL-NP	W-Test (1)	3314	Mean-T	W-Test (1)
Zinc	mg/kg	2551	3426	11500	J	mg/kg	3397	95% UCL-NP	W-Test (2)	2551	Mean-N	W-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

J: The associated value is an estimated quantity.

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W-Test indicates data are lognormally distributed.

(2) Shapiro-Wilks W-Test indicates data are neither normally nor lognormally distributed.

TABLE 3.7.2

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Osburn, Wallace, and Silverton Combined - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface Water
Exposure Medium:	Surface Water
Exposure Point:	Surface Water at South Fork CDAR

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	0.78	1.09	7.3		ug/L	1.096	95% UCL-NP	D-Test (2)	0.78	Mean-N	D-Test (2)
Cadmium	ug/L	3.19	3.99	12.5		ug/L	4.029	95% UCL-NP	D-Test (1)	10.49	Mean-T	D-Test (1)
Manganese	ug/L	25.98	32.31	174		ug/L	32.04	95% UCL-NP	D-Test (1)	39.36	Mean-T	D-Test (1)
Mercury	ug/L	0.1	0.12	0.84	J	ug/L	0.122	95% UCL-NP	D-Test (2)	0.1	Mean-N	D-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

J: The associated value is an estimated quantity.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gilbert 1987)

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are lognormally distributed.

(2) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

TABLE 3.7.3

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Osburn, Wallace, and Silverton Combined - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/Subsurface Soil
Exposure Point:	Construction Site Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	8.36	9.13	299		mg/kg	9.26	95% UCL-NP	(1)	6.69	Mean-T	(1)
Arsenic	mg/kg	25.6	27.55	1060		mg/kg	27.31	95% UCL-NP	(1)	21.87	Mean-T	(1)
Cadmium	mg/kg	4.95	5.2	95		mg/kg	5.21	95% UCL-NP	(1)	6.09	Mean-T	(1)
Iron	mg/kg	21538	21860	152000		mg/kg	21860	95% UCL-N	(2)	21538	Mean-N	(2)
Manganese	mg/kg	1097	1127	18200		mg/kg	1129	95% UCL-NP	(1)	1077	Mean-T	(1)
Zinc	mg/kg	713	754	18000		mg/kg	752	95% UCL-NP	(1)	666	Mean-T	(1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation

Normal UCL values calculated using the formula: average + [t-statistic*(std deviation/count^0.5)]

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Sample size > 500; however, MTCA Stat checks distribution for sample sizes < 500. Therefore, data plotted on histograms using SYSTATv9. Distribution of the data appear lognormal.

(2) Sample size > 500; distribution of the iron data appear normal based on the histogram generated using SYSTATv9.

TABLE 3.8.1
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	9.32	11.7	222		mg/kg	11.94	95% UCL-NP	D-Test (1)	8.22	Mean - T	D-Test (1)
Arsenic	mg/kg	31.4	41.7	1150		mg/kg	41.62	95% UCL-NP	D-Test (2)	31.4	Mean - N	D-Test (2)
Cadmium	mg/kg	5.98	6.53	27.1		mg/kg	6.52	95% UCL-NP	D-Test (2)	5.98	Mean - N	D-Test (2)
Iron	mg/kg	21819	23124	123000		mg/kg	23311	95% UCL-NP	D-Test (2)	21819	Mean - N	D-Test (2)
Manganese	mg/kg	1150	1243	7540		mg/kg	1250	95% UCL-NP	D-Test (2)	1150	Mean - N	D-Test (2)
Zinc	mg/kg	1061	1158	4420		mg/kg	1159	95% UCL-NP	D-Test (1)	1104	Mean - T	D-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gilbert 198

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are lognormally distributed.

(2) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

TABLE 3.8.2

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap Water

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	0.45	0.73	3.8		ug/L	0.74	95% UCL-NP	W-Test (1)	0.45	Mean-N	W-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculati

Static and purged tap water results were averaged.

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W-Test indicates data are neither normally nor lognormally distributed.

TABLE 3.8.3

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Nine Mile - Future Residential Exposure Scenario

Scenario Timeframe:	Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap Water

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	ug/L	1.6	2.14	18		ug/L	2.1	95% UCL-NP	D-Test (2)	1.67	Mean-T	D-Test (2)
Arsenic	ug/L	0.81	1.23	16.1		ug/L	1.25	95% UCL-NP	D-Test (1)	0.81	Mean-N	D-Test (1)
Cadmium	ug/L	91.79	127	996	J	ug/L	130.85	95% UCL-NP	D-Test (2)	91.79	Mean-N (3)	D-Test (2)
Zinc	ug/L	14239	19372	145000		ug/L	19756	95% UCL-NP	D-Test (2)	14239	Mean-N (3)	D-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

J: The associated value is an estimated quantity.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gilbert 1987).
Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics:
Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

(2) D'Agostino's Test indicates data are lognormally distributed.

(3) The Mean-T value exceeded the 95% UCL-NP for this chemical, likely due to the large variability in the data set (see Attachment B). Therefore, the arithmetic mean was selected as a more appropriate Central Tendency value.

TABLE 3.8.4
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Waste Piles

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	22.74	42.99	242	J	mg/kg	42.34	95% UCL-NP	W-Test (2)	19.81	Mean-T	W-Test (2)
Arsenic	mg/kg	200.19	479.99	3610		mg/kg	518.49	95% UCL-NP	W-Test (2)	95.57	Mean-T	W-Test (2)
Cadmium	mg/kg	19.39	30.79	146		mg/kg	31.67	95% UCL-NP	W-Test (1)	19.39	Mean-N	W-Test (1)
Iron	mg/kg	36673	43654	87900		mg/kg	43032	95% UCL-NP	W-Test (2)	36629	Mean-T	W-Test (2)
Manganese	mg/kg	1445	1772	3360		mg/kg	1721	95% UCL-NP	W-Test (2)	1478	Mean-T	W-Test (2)
Zinc	mg/kg	3983	5975	25800		mg/kg	6196	95% UCL-NP	W-Test (2)	5147	Mean-T	W-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatior

J: The associated value is an estimated quantity.

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics:
Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W-Test indicates data are neither normally nor lognormally distributed.
(2) Shapiro-Wilks W-Test indicates data are lognormally distributed.

TABLE 3.8.5

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Sediment at Nine Mile/Canyon Creek

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	39.39	72.03	288	J	mg/kg	71.25	95% UCL-NP	W-Test (1)	47.54	Mean-T	W-Test (1)
Arsenic	mg/kg	27.7	50.96	216	J	mg/kg	50.62	95% UCL-NP	W-Test (1)	23.12	Mean-T	W-Test (1)
Cadmium	mg/kg	37.35	60.1	194	J	mg/kg	59.2	95% UCL-NP	W-Test (1)	58.8	Mean-T	W-Test (1)
Iron	mg/kg	26882	35949	98700		mg/kg	36476	95% UCL-NP	W-Test (1)	26575	Mean-T	W-Test (1)
Manganese	mg/kg	1874	2720	6830		mg/kg	2626	95% UCL-NP	W-Test (1)	1856	Mean-T	W-Test (1)
Zinc	mg/kg	6037	8982	22400		mg/kg	8883	95% UCL-NP	W-Test (1)	8396	Mean-T	W-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatior

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W-Test indicates data are lognormally distributed.

TABLE 3.8.6

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface Water
Exposure Medium:	Surface Water
Exposure Point:	Surface Water at Nine Mile/Canyon Creek

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	0.77	1.05	10.9		ug/L	1.1	95% UCL-NP	D-Test (1)	0.7	Mean-T	D-Test (1)
Cadmium	ug/L	53.15	101.89	1810	J	ug/L	111.02	95% UCL-NP	D-Test (1)	58.92	Mean-T	D-Test (1)
Manganese	ug/L	748.82	1512	26800		ug/L	1699	95% UCL-NP	D-Test (2)	748.82	Mean-N	D-Test (2)
Mercury	ug/L	0.27	0.31	0.17	J	ug/L	0.31	95% UCL-NP	D-Test (2)	0.27	Mean-N	D-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

J: The associated value is an estimated quantity.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gilbert 198

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are lognormally distributed.

(2) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

TABLE 3.8.7
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Nine Mile - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/Subsurface Soil
Exposure Point:	Construction Site Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	9.46	11.15	256		mg/kg	10.61	95% UCL-NP	(1)	6.89	Mean - T	(1)
Arsenic	mg/kg	21.89	25.68	1150		mg/kg	24.97	95% UCL-NP	(1)	18.16	Mean - T	(1)
Cadmium	mg/kg	6.27	6.74	97		mg/kg	6.54	95% UCL-NP	(1)	7.64	Mean - T	(1)
Iron	mg/kg	21743	22474	123000		mg/kg	21976	95% UCL-NP	(1)	21308	Mean - T	(1)
Manganese	mg/kg	1107	1156	7540		mg/kg	1135	95% UCL-NP	(1)	1115	Mean - T	(1)
Zinc	mg/kg	1037	1100	8090		mg/kg	1070	95% UCL-NP	(1)	1030	Mean - T	(1)

NOTES:
For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation
Normal UCL values calculated using MTCA Stat *v2.1* .
Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Sample size > 500; however, MTCA Stat checks distribution for sample sizes < 500. Therefore, data plotted on histograms using SYSTATv9. Distribution of the data appear lognormal.

TABLE 3.9.1
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	13.22	18.64	201		mg/kg	20.05	95% UCL-NP	D-Test (1)	11.04	Mean-T	D-Test (1)
Arsenic	mg/kg	27.3	37.06	433		mg/kg	39.04	95% UCL-NP	D-Test (2)	27.3	Mean -N	D-Test (2)
Cadmium	mg/kg	5.07	6.38	42.5		mg/kg	6.39	95% UCL-NP	D-Test (2)	5.07	Mean -N	D-Test (2)
Iron	mg/kg	22709	24844	70400		mg/kg	24742	95% UCL-NP	D-Test (2)	22709	Mean -N	D-Test (2)
Manganese	mg/kg	1430	1614	4830		mg/kg	1628	95% UCL-NP	D-Test (1)	1412	Mean-T	D-Test (1)
Zinc	mg/kg	1082	1307	6450		mg/kg	1375	95% UCL-NP	D-Test (1)	1049	Mean-T	D-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gilbert 198

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are lognormally distributed.

(2) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

TABLE 3.9.2

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap Water

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	0.13	0.18	0.25		ug/L	0.25	Max	(1)	0.13	Mean-N	(2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

Static and purged tap water results were averaged.

Normal UCL values calculated using MTCA Stat *v2.1* .

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Distribution of the data could not be determined because less than 10 samples were available; therefore, the maximum concentration was used as the Reasonable Maximum Exposure EPC.

(2) Distribution of the data could not be determined because less than 10 samples were available; therefore, the arithmetic mean was used as the Central Tendency EPC.

TABLE 3.9.3
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Waste Piles

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	11.84	21.07	20.3		mg/kg	20.3	Max	(1)	11.84	Mean-N	(1)
Arsenic	mg/kg	23.6	39.5	42.1		mg/kg	42.1	Max	(1)	23.6	Mean-N	(1)
Cadmium	mg/kg	5.57	11.94	13.8		mg/kg	13.8	Max	(1)	5.57	Mean-N	(1)
Iron	mg/kg	22180	31200	33200		mg/kg	33200	Max	(1)	22180	Mean-N	(1)
Manganese	mg/kg	2103	2822	2750		mg/kg	2750	Max	(1)	2103	Mean-N	(1)
Zinc	mg/kg	1042	2239	2880		mg/kg	2880	Max	(1)	1042	Mean-N	(1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatior
Normal UCL values calculated using MTCA Stat *v2.1* .

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Distribution of the data could not be determined because less than 10 samples were available; therefore, the maximum concentration was used for the EPC.

TABLE 3.9.4
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Sediment at South Fork CDAR

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	2.72	4.45	14.7	J	mg/kg	4.46	95% UCL-NP	W-Test (1)	2.61	Mean-T	W-Test (1)
Arsenic	mg/kg	12.87	16.17	28.4	J	mg/kg	15.89	95% UCL-NP	W-Test (1)	13.21	Mean-T	W-Test (1)
Cadmium	mg/kg	9.9	19.91	81	J	mg/kg	19.83	95% UCL-NP	W-Test (1)	9.66	Mean-T	W-Test (1)
Iron	mg/kg	33569	47504	121000		mg/kg	47300	95% UCL-NP	W-Test (1)	34079	Mean-T	W-Test (1)
Manganese	mg/kg	3434	5270	15700		mg/kg	5233	95% UCL-NP	W-Test (1)	3513	Mean-T	W-Test (1)
Zinc	mg/kg	2334	4012	13700	J	mg/kg	4017	95% UCL-NP	W-Test (1)	2208	Mean-T	W-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatior

J: The associated value is an estimated quantity.

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W Test indicates data are lognormally distributed.

TABLE 3.9.5

MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY

Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface Water
Exposure Medium:	Surface Water
Exposure Point:	Surface Water at South Fork CDAR

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	0.51	0.59	1.8	J	ug/L	0.6	95% UCL-NP	D-Test (1)	0.54	Mean-T	D-Test (1)
Cadmium	ug/L	0.81	1.29	15.2		ug/L	1.24	95% UCL-NP	D-Test (2)	0.81	Mean-N	D-Test (2)
Manganese	ug/L	13.69	18.73	127		ug/L	18.96	95% UCL-NP	D-Test (1)	13.66	Mean-T	D-Test (1)
Mercury	ug/L	0.08	0.09	0.32		ug/L	0.086	95% UCL-NP	D-Test (2)	0.08	Mean-N	D-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

J: The associated value is an estimated quantity.

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gilbert 1987).
Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are lognormally distributed.

(2) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

TABLE 3.9.6
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Mullan - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/Subsurface Soil
Exposure Point:	Construction Site Soil

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	14.09	18.31	295		mg/kg	18.68	95% UCL-NP	D-Test (1)	14.09	Mean -N	D-Test (1)
Arsenic	mg/kg	26.48	31.81	433		mg/kg	31.66	95% UCL-NP	D-Test (1)	26.48	Mean -N	D-Test (1)
Cadmium	mg/kg	4.22	4.99	73.4		mg/kg	5.04	95% UCL-NP	D-Test (1)	4.22	Mean -N	D-Test (1)
Iron	mg/kg	24863	27083	147000		mg/kg	26912	95% UCL-NP	D-Test (1)	24863	Mean -N	D-Test (1)
Manganese	mg/kg	1688	1957	20200		mg/kg	1941	95% UCL-NP	D-Test (1)	1688	Mean -N	D-Test (1)
Zinc	mg/kg	833	947	9250		mg/kg	953	95% UCL-NP	D-Test (2)	823	Mean -T	D-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.
D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gilbert 198
Normal UCL values calculated using MTCA Stat *v2.1* .
Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics:

Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are neither normally nor lognormally distributed.
(2) D'Agostino's Test indicates data are lognormally distributed.

TABLE 3.10.1
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/Sediment
Exposure Medium:	Soil/Sediment
Exposure Point:	Soil/Sediment at Spokane River

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Antimony	mg/kg	2.75	3.22	5.5		mg/kg	3.195	95% UCL-NP	W-Test (1)	2.78	Mean-T	W-Test (1)
Arsenic	mg/kg	41.03	50	83.4		mg/kg	50	95% UCL-N	W-Test (2)	41.03	Mean-N	W-Test (2)
Cadmium	mg/kg	11.23	13.4	21.6		mg/kg	13.4	95% UCL-N	W-Test (2)	11.23	Mean-N	W-Test (2)
Iron	mg/kg	43133	49107	66800		mg/kg	48881	95% UCL-NP	W-Test (1)	43485	Mean-T	W-Test (1)
Manganese	mg/kg	2956	3641	7480		mg/kg	3666	95% UCL-NP	W-Test (1)	2996	Mean-T	W-Test (1)
Zinc	mg/kg	1575	1832	2460		mg/kg	1800	95% UCL-NP	W-Test (3)	1575	Mean-N	W-Test (3)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculation.

W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W-Test indicates data are lognormally distributed.
(2) Shapiro-Wilks W-Test indicates data are normally distributed.
(3) Shapiro-Wilks W-Test indicates data are neither normally nor lognormally distributed.

TABLE 3.10.2
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface Water
Exposure Medium:	Surface Water
Exposure Point:	Surface Water at Spokane River

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	ug/L	10.56	14.99	20.8		ug/L	20.8	Max	(1)	10.56	Mean-N	(2)
Cadmium	ug/L	20.01	33.34	47.9		ug/L	47.9	Max	(1)	20.01	Mean-N	(2)
Manganese	ug/L	2792	4781	6980		ug/L	6980	Max	(1)	2792	Mean-N	(2)
Mercury	ug/L	0.09	0.1	0.1	U	ug/L	0.1	Max	(1)	0.09	Mean-N	(2)

NOTES:
For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calculatic
U: The analyte was not detected above the reported sample detection limit. All values reported for this analyte were nondetect.
Normal UCL values calculated using MTCA Stat *v2.1* .

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Distribution of the data could not be determined because less than 10 samples were available; therefore, the maximum concentration was used as the Reasonable Maximum Exposure EPC.

TABLE 3.11.1
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
All Geographical Areas - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Plant Tissue
Exposure Point:	Homegrown Vegetables

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Arsenic	mg/kg	0.02	0.03	0.1128		mg/kg	0.025	95% UCL-NP	W-Test (1)	0.02	Mean-T	W-Test (1)
Cadmium	mg/kg	0.22	0.32	1.85		mg/kg	0.319	95% UCL-NP	W-Test (1)	0.21	Mean-T	W-Test (1)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the higher concentration or the lower SQL was used in the calculation.
W-Test: Developed by Shapiro and Wilk (Supplemental Guidance to RAGs: Calculating the Concentration Term. OSWER Directive 9285.7-081, May 1992).
Normal UCL values calculated using MTCA Stat *v2.1*.
Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9*. This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics: Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) Shapiro-Wilks W-Test indicates data are lognormally distributed.

TABLE 3.11.2
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
All Geographical Areas - Recreational and Subsistence Exposure Scenarios

Scenario Timeframe:	Current/Future
Medium:	Surface Water/Sediment
Exposure Medium:	Animal Tissue
Exposure Point:	Fish from Lower CDAR

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Bullhead												
Cadmium	mg/kg	0.01	0.01	0.069		mg/kg	0.008	95% UCL-NP	D-Test (1)	0.01	Mean-N	D-Test (1)
Mercury	mg/kg	0.05	0.05	0.21		mg/kg	0.052	95% UCL-NP	D-Test (1)	0.05	Mean-N	D-Test (1)
Northern Pike												
Cadmium	mg/kg	0.01	0.01	0.02		mg/kg	0.006	95% UCL-NP	D-Test (1)	0.01	Mean-N	D-Test (1)
Mercury	mg/kg	0.11	0.13	0.48		mg/kg	0.133	95% UCL-NP	D-Test (2)	0.12	Mean-T	D-Test (2)
Perch												
Cadmium	mg/kg	0.03	0.04	0.169		mg/kg	0.037	95% UCL-NP	D-Test (2)	0.03	Mean-T	D-Test (2)
Mercury	mg/kg	0.08	0.09	0.23		mg/kg	0.089	95% UCL-NP	D-Test (2)	0.08	Mean-T	D-Test (2)

NOTES:

For nondetect results, half the sample quantitation limit (SQL) was used as a proxy concentration. For duplicate sample results, the greater detected concentration or the lower SQL was used in the calc

D'Agostino's Test (WA State Dept. of Ecology Toxics Cleanup Program: Statistical Guidance for Ecology Site Managers. August 1992; and Statistical Methods for Environmental Pollution Monitoring. Gill

Normal UCL values calculated using MTCA Stat *v2.1* .

Non-parametric UCL values calculated using the Bootstrap method in SYSTAT *v9* . This method is discussed in "The Lognormal Distribution in Environmental Applications" (U.S. EPA 1997).

Statistics:
Maximum Detected Value (Max)
95% UCL of Normal Data (95% UCL-N)
95% non-parametric UCL (95% UCL-NP)
Mean of Log-transformed Data (Mean-T)
Mean of Normal Data (Mean-N)

(1) D'Agostino's Test indicates data are neither normally nor lognormally distributed.

(2) D'Agostino's Test indicates data are lognormally distributed.

TABLE 4.1
VALUES USED FOR DAILY INTAKE CALCULATIONS
Residential Exposure

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil
Exposure Point: Yard Soil
Receptor Population: Residents
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	Chronic Daily Intake (CDI) (mg/kg/day) =
	IRSa	Ingestion Rate of Soil (adult)	mg/day	100	USEPA 1991	50	USEPA 1993	
	IRSc	Ingestion Rate of Soil (child)	mg/day	200	USEPA 1991	100	USEPA 1993	For evaluating child exposure to noncarcinogens:
	EF	Exposure Frequency	days/year	350	USEPA 1991	260 (1)	USEPA 1993	= Csoil x IRS x EF x ED x AbsO x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	24	USEPA 1991	7	USEPA 1993	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	2	USEPA 1993	For evaluating combined adult and child exposures:
	AbsO	Gastrointestinal Absorption	unitless	chemical specific (2)	USEPA 2000	chemical specific (2)	USEPA 2000	
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	IngFadj =
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	70	USEPA 1991	[(EDc x IRSc) / BWc] + [(EDa x IRSa) / BWa]
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	15	USEPA 1991	Therefore,
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	CDIadj = Csoil x IngFadj x EF x AbsO x CF x 1/AT
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	
Dermal Contact	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	CDI (mg/kg-day) =
	SAa	Surface Area Available for Contact (adult)	cm^2	2,500	USEPA 1998a	2,500	USEPA 1998a	
	SAc	Surface Area Available for Contact (child)	cm^2	2,200	USEPA 1998a	2,200	USEPA 1998a	For evaluating child exposure to noncarcinogens :
	EF	Exposure Frequency	events/year	350	USEPA 1991	260 (1)	USEPA 1993	= Csoil x SA x EF x ED x AF x AbsD x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	24	USEPA 1991	7	USEPA 1993	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	2	USEPA 1993	For evaluating carcinogens (combined adult and child):
	AFa	Adherence Factor (adult)	mg/cm^2 -event	0.1	USEPA 1998a	0.1	USEPA 1998a	SFSadj =
	AFc	Adherence Factor (child)	mg/cm^2-event	0.2	USEPA 1998a	0.2	USEPA 1998a	= [(EDc x SAc x AFc) / BWc] + [(EDa x SAa x AFa) / BWa]
	AbsD	Dermal Absorption	unitless	As = 0.03; Cd = 0.001	USEPA 1998a	As = 0.03; Cd = 0.001	USEPA 1998a	Therefore,
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	CDIadj = Csoil x EF x CF x AbsD x SFSadj x 1/AT
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	70	USEPA 1991	
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	15	USEPA 1991	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	

NOTES: na: not applicable; -- not available

(1) Exposure frequency based on 3 months limited soil exposure due to snow-covered/frozen ground.

(2) Gastrointestinal absorption for arsenic is 60%, for all other chemicals it is assumed to be 100%.

TABLE 4.2
VALUES USED FOR DAILY INTAKE CALCULATIONS
Residential Exposure

Scenario Timeframe: Cuurent/Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Tap Water
Receptor Population: Residents
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	CW	Chemical Concentration in Water	µg/l	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	Chronic Daily Intake (CDI) (mg/kg/day) =
	IR-Wa	Ingestion Rate of Water (adult)	liters/day	2	USEPA 1991	1.4	USEPA 1993	
	IR-Wc	Ingestion Rate of Water (child)	liters/day	1	USEPA 1999a	1	USEPA 1999a	For evaluating child exposure to noncarcinogens:
	EF	Exposure Frequency	days/year	350	USEPA 1991	234	USEPA 1993	= CW x IR-W x EF x ED x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	24 (1)	USEPA 1991	7	USEPA 1993	
	EDc	Exposure Duration (child)	years	6 (1)	USEPA 1991	2	USEPA 1993	For evaluating combined adult and child exposures:
	CF	Conversion Factor	mg/µg	0.001	na	0.001	na	IngFadj =
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	70	USEPA 1991	= [(EDc x IR-Wc) / BWc] + [(EDa x IR-Wa) / BWa]
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	15	USEPA 1991	Therefore,
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	CDIadj = CW x EF x CF x IngFadj x 1/AT
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	

NOTES: na: not applicable; -- not available; * not quantitatively evaluated, see text

(1) USEPA 1991 recommends an adult/child exposure duration of 24/6 years for ingestion of soil; For consistency, an exposure duration of 24/6 years was selected for ingestion of tap water.

TABLE 4.3

VALUES USED FOR DAILY INTAKE CALCULATIONS

Residential Exposure

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Plant Tissue
Exposure Point: Homegrown Vegetables
Receptor Population: Residents
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Cveg	Chemical Concentration in Vegetables	mg/kg	See Table 3	See Table 3	See Table 3	See Table 3	Chronic Daily Intake (CDI)(mg/kg/day) =
	IR-veg	Ingestion Rate of Vegetables	g/kg-day	5.04 (1)	USEPA 1997	0.492	USEPA 1997	
	EF	Exposure Frequency	days/year	365 (2)	prof. judgement	365 (2)	prof. judgement	For evaluating exposure to noncarcinogens:
	ED	Exposure Duration	years	30	USEPA 1991	9	USEPA 1993	= Cveg x IR-veg x EF x ED x CF x 1/AT-N
	CF	Conversion Factor	kg/g	1.0E-03	na	1.0E-03	na	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	For evaluating exposures to carcinogens:
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	= Cveg x IR-veg x EF x ED x CF x 1/AT-C

NOTES: na: not applicable; -- not available

(1) Ingestion rate is seasonally adjusted and incorporates the body weights of all participants in the study (children + adults) from EPA 1997.

(2) Ingestion Rate of vegetables is an average daily consumption rate, therefore 365 days/year was selected as the frequency of exposure for both the RME and CT scenarios.

TABLE 4.4
VALUES USED FOR DAILY INTAKE CALCULATIONS
Neighborhood Recreational Exposure

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil
Exposure Point: Waste Piles
Receptor Population: Neighborhood Residents
Receptor Age: Children (4-11 years old)

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	--	--	Chronic Daily Intake (CDI) (mg/kg/day) (6) =
	IR-soil	Ingestion Rate of Soil	mg/day	300 (1)	USEPA 1999b	120 (1)	van Wijnen 1990	
	EF	Exposure Frequency	days/year	17 (2)	prof. judgement	8.5(2)	prof. judgement	= Csoil x IR-soil x EF x ED x AbsO x CF x 1/BW x 1/AT
	ED	Exposure Duration	years	7 (3)	prof. judgement	2	USEPA 1993	
	AbsO	Gastrointestinal Absorption	unitless	chemical specific (7)	USEPA 2000	chemical specific (7)	USEPA 2000	
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	
	BW	Body Weight	kilograms	28 (4)	USEPA 1997	28 (4)	USEPA 1997	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	
Dermal Contact	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	CDI (mg/kg-day) (6) =
	SA	Surface Area Available for Contact	cm^2	5,080	USEPA 1997	5,080	USEPA 1997	
	EF	Exposure Frequency	events/year	34 (5)	prof. judgement	17 (5)	prof. judgement	= Csoil x SA x EF x ED x AF x AbsD x CF x 1/BW x 1/AT
	ED	Exposure Duration	years	7 (3)	prof. judgement	2	USEPA 1993	
	AF	Adherence Factor (child)	mg/cm^2-event	0.2	USEPA 1998a	0.2	USEPA 1998a	
	AbsD	Dermal Absorption	unitless	As = 0.03; Cd = 0.001	USEPA 1998a	As = 0.03; Cd = 0.001	USEPA 1998a	
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	
	BW	Body Weight	kilograms	28 (4)	USEPA 1997	28 (4)	USEPA 1997	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	

NOTES: na: not applicable; -- not available

(1) 120 mg/day is the mean value of soil intake from study, whereas 300 mg/day is the RME value (90th percentile) from study.

(2) Exposure frequency calculated by: (34 weeks/year x 7 hours/day x 1 day/week) / 14 hours/day = 17 days/year for RME. The 7 hours/day assumes weekend outdoor exposure (EPA 1997).
For CT: (34 weeks/year x 7 hours/day x once every other week, 0.5) / 14 hours/day = 8.5 days/year.

(3) Neighborhood exposures assumes children between the ages of 4 through 11 are playing in the waste piles.

(4) Value is the average of boys and girls, ages 4 - 11, 50th percentile.

(5) Exposure frequency calculated by: 34 weeks/year x 1 event/week = 34 events/year for RME and 34 weeks/year, once every other week = 17 events/year for CT.

(6) The formula is not age-adjusted because adult exposures to this pathway are anticipated to be minimal, and the residential adult soil pathway will be protective of neighborhood exposures.

(7) Gastrointestinal absorption for arsenic is 60%, for all other chemicals it is assumed to be 100%.

TABLE 4.5
VALUES USED FOR DAILY INTAKE CALCULATIONS
Neighborhood Recreational Exposure

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil
Exposure Point: Upland Parks/Schools and Elk Creek Area
Receptor Population: Neighborhood Residents
Receptor Age: Children (4-11 years old)

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	Chronic Daily Intake (CDI) (mg/kg/day) (6) =
	IR-soil	Ingestion Rate of Soil	mg/day	300 (1)	USEPA 1999b	120 (1)	van Wijnen 1990	
	EF	Exposure Frequency	days/year	34 (2)	prof. judgement	17 (2a)	prof. judgement	For evaluating noncarcinogens (child only):
	ED	Exposure Duration	years	7 (3)	prof. judgement	2	EPA 1993	= Csoil x IR-soil x EF x ED x AbsO x CF x 1/BW x 1/AT-N
	AbsO	Gastrointestinal Absorption	unitless	chemical specific (7)	USEPA 2000	chemical specific (7)	USEPA 2000	
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	
	BW	Body Weight	kilograms	28 (4)	EPA 1997	28 (4)	EPA 1997	For evaluating carcinogens (child only) :
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989	25,550	EPA 1989	= Csoil x IR-soil x EF x ED x AbsO x CF x 1/BW x 1/AT-C
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	EPA 1989	ED x 365	EPA 1989	
Dermal Contact	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	CDI (mg/kg-day) (6) =
	SA	Surface Area Available for Contact	cm^2	5,080	EPA 1997	5,080	EPA 1997	
	EF	Exposure Frequency	events/year	68 (5)	prof. judgement	34 (5)	prof. judgement	= Csoil x SA x EF x ED x AF x AbsD x CFx 1/BW x 1/AT-N
	ED	Exposure Duration	years	7 (3)	prof. judgement	2	EPA 1993	
	AF	Adherence Factor (child)	mg/cm^2-event	0.2	EPA 1998a	0.2	EPA 1998a	For evaluating carcinogens (child only) :
	AbsD	Dermal Absorption	unitless	As = 0.03; Cd = 0.001	EPA 1998a	As = 0.03; Cd = 0.001	EPA 1998a	= Csoil x SA x EF x ED x AF x AbsD x CFx 1/BW x 1/AT-C
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	
	BW	Body Weight	kilograms	28 (4)	EPA 1997	28 (4)	EPA 1997	
	AT-C	Averaging Time (Cancer)	days	25,550	EPA 1989	25,550	EPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	EPA 1989	ED x 365	EPA 1989	

NOTES: na: not applicable; -- not available

(1) 120 mg/day is the mean value of soil intake from study, whereas 300 mg/day is the RME value (90th percentile) from study.

(2) RME Exposure frequency for **child**: (34 wks/yr x 7 hrs/dy x 2 dys/wk) / 14 hrs/dy = 34 dys/yr. The 7 hrs/dy for 2 dys/wk assumes weekend outdoor exposure (EPA 1997).

(2a) CT Exposure frequency for **child**: (34 wks/yr x 7 hrs/day x 1 day/wk) / 14 hrs/day = 17 days/year.

(3) Neighborhood exposures assumes children between the ages of 4 through 11 are playing at parks and schools.

(4) Value is the average of boys and girls, ages 4 - 11, 50th percentile.

(5) Exposure frequency calculated by: 34 weeks/year x 2 event/week = 68 events/year for RME and 34 weeks/year, 1 event/week = 34 events/year for CT.

(6) The formula is not age-adjusted because adult exposures to this pathway are anticipated to be minimal, and the residential adult soil pathway will be protective of neighborhood exposures.

(7) Gastrointestinal absorption for arsenic is 60%, for all other chemicals it is assumed to be 100%.

TABLE 4.6
VALUES USED FOR DAILY INTAKE CALCULATIONS
Neighborhood Recreational Exposures

Scenario Timeframe: Current/Future
Medium: Soil/Sediment
Exposure Medium: Soil/Sediment
Exposure Point: Soil/Sediment at Various Locations
Receptor Population: Neighborhood Residents
Receptor Age: Children (4-11 years old)

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Csed	Chemical Concentration in Sediments	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	Chronic Daily Intake (CDI) (mg/kg/day) (6) =
	IR-sed	Ingestion Rate of Sediment	mg/day	300 (1)	USEPA 1999b	120 (1)	van Wijnen 1990	
	EF	Exposure Frequency	days/year	21 (2)	prof. judgement	10 (2a)	prof. judgement	= Csed x IR-sed x EF x ED x AbsO x CF x 1/BW x 1/AT
	ED	Exposure Duration	years	7 (3)	prof. judgement	2	USEPA 1993	
	AbsO	Gastrointestinal Absorption	unitless	chemical specific (8)	USEPA 2000	chemical specific (8)	USEPA 2000	
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	
	BW	Body Weight	kilograms	28 (4)	USEPA 1997	28 (4)	USEPA 1997	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	
Dermal	Csed	Chemical Concentration in Sediments	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	CDI (mg/kg-day) (6) =
	SA	Surface Area Available for Contact (child)	cm^2	5080 (7)	EPA 1997	5080 (7)	EPA 1997	
	EF	Exposure Frequency	events/year	96 (5)	prof. judgement	48 (5)	prof. judgement	= Csed x SA x EF x ED x AF x AbsD x CF x 1/BW x 1/AT
	ED	Exposure Duration	years	7 (3)	prof. judgement	2	USEPA 1993	
	AF	Adherence Factor (child)	mg/cm^2-event	0.2	USEPA 1998a	0.2	USEPA 1998a	
	AbsD	Dermal Absorption	unitless	As = 0.03; Cd = 0.001	USEPA 1998a	As = 0.03; Cd = 0.001	USEPA 1998a	
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	
	BW	Body Weight	kilograms	28 (4)	USEPA 1997	28 (4)	USEPA 1997	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	

NOTES: na: not applicable; -- not available

(1) 120 mg/day is the mean value of soil intake from study, whereas 300 mg/day is the RME value (90th percentile) from study.

(2) RME Exposure Frequency calculated by: (24 weeks/year x 3 hours/day x 4 days/week) / 14 hours/day = 21 days/year. The 3 hours/day is the high end of the 50th percentile range (1-3 hours/day) from EPA 1997.

(2a) CT Exposure Frequency calculated by: (24 weeks/year x 3 hours/day x 2 days/week) / 14 hours/day = 10 days/year.

(3) Neighborhood exposures assumes children between the ages of 4 through 11 are playing with creek sediments.

(4) Value is the average of boys and girls, ages 4 - 11, 50th percentile (EPA 1997).

(5) Exposure frequency calculated by: 24 weeks/year x 4 events/week = 96 events/year for RME and 24 weeks/year x 2 events/week = 48 events/year for CT.

(6) The formula is not age-adjusted because adult exposures to this pathway are anticipated to be minimal, and the residential adult soil pathway will be protective of neighborhood exposures.

(7) At Lower Basin and Kingston (North-South Confluence), a skin surface area of 7,960 cm^2 was used to reflect the possibility that swimming and therefore exposiure of the entire body to contaminants in sediment could occur at these locations. It was assumed that swimming would occur during 16 weeks of the year (the warmest months of the year), while wading and playing along the shoreline without swimming would occur during 8 weeks of the year. The median skin surface area for male children age 4-11 is 9,400 cm^2 (USEPA 1997). The skin surface area was calculated as follows: ((16 weeks x 9,400 cm^2) + (8 weeks x 5,080 cm^2)) / 24 weeks = 7,960 cm^2.

(8) Gastrointestinal absorption for arsenic is 60%, for all other chemicals it is assumed to be 100%.

TABLE 4.7
VALUES USED FOR DAILY INTAKE CALCULATIONS
Neighborhood Recreational Exposure

Scenario Timeframe: Current/Future
Medium: Surface Water
Exposure Medium: Surface Water
Exposure Point: Surface Water at Various Locations
Receptor Population: Neighborhood Residents
Receptor Age: Children (4-11 years old)

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Incidental	CW	Chemical Concentration in Surface Water	µg/l	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	Chronic Daily Intake (CDI) (mg/kg/day) (4)=
Ingestion	IR-W	Ingestion Rate of Surface Water	mL/hour	30	USEPA 1998b	30	USEPA 1998b	
	EF	Exposure Frequency	events/year	96 (1)	prof. judgement	48 (1a)	prof. judgement	= CW x IR-W x EF x ET x ED x CF1 x CF2 x 1/BW x 1/AT
	ET	Exposure Time	hours/event	1	USEPA 1997	1	USEPA 1997	
	ED	Exposure Duration	years	7 (2)	prof. judgement	2	USEPA 1993	
	CF1	Conversion Factor 1	mg/µg	0.001	na	0.001	na	
	CF2	Conversion Factor 2	L/mL	0.001	na	0.001	na	
	BW	Body Weight	kilograms	28 (3)	USEPA 1997	28 (3)	USEPA 1997	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	

NOTES: na: not applicable; -- not available; * not quantitatively evaluated, see text

(1) RME Exposure Frequency calculated by: 24 weeks/year x 4 events/week= 96 events/year; assumes exposures occur from May through mid-October.

(1a) CT Exposure Frequency calculated by: 24 weeks/year x 2 events/week= 48 events/year; assumes exposures occur from May through mid-October.

(2) Neighborhood exposures assumes children between the ages of 4 through 11 are playing with creek surface water.

(3) Value is the average of boys and girls, ages 4 - 11, 50th percentile (EPA 1997).

(4) The formula is not age-adjusted because adult exposures to this pathway are anticipated to be minimal, and the residential adult soil pathway will be protective of neighborhood exposures.

TABLE 4.8
VALUES USED FOR DAILY INTAKE CALCULATIONS
Public Recreational Exposure

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface Soil
Exposure Point: Upland Parks/Schools
Receptor Population: Recreational Visitors
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	Chronic Daily Intake (CDI) (mg/kg/day) =
	IRSa	Ingestion Rate of Soil (adult)	mg/day	100	USEPA 1991	50	USEPA 1993	
	IRSc	Ingestion Rate of Soil (child)	mg/day	300	USEPA 1999b	120 (3)	van Wijnen 1990	For evaluating child exposure to noncarcinogens:
	EFa	Exposure Frequency (adult)	days/year	30 (1)	prof. judgement	15 (1a)	prof. judgement	= Csoil x IRS x EF x ED x AbsO x CF x 1/BW x 1/AT-N
	EFc	Exposure Frequency (child)	days/year	34 (1)	prof. judgement	17 (1a)	prof. judgement	
	EDa	Exposure Duration (adult)	years	24	USEPA 1991	7	USEPA 1993	For evaluating combined adult and child exposures:
	EDc	Exposure Duration (child)	years	6	USEPA 1991	2	USEPA 1993	IngFadj =
	AbsO	Gastrointestinal Absorption	unitless	chemical specific (4)	USEPA 2000	chemical specific (4)	USEPA 2000	
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	= (EFc x EDc x IRSc / BWc) + (EFa x EDa x IRSa / BWa)
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	70	USEPA 1991	Therefore,
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	15	USEPA 1991	CDIadj = Csoil x AbsO x CF x IngFadj x 1/AT
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	
Dermal Contact	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	CDI (mg/kg-day) =
	SAa	Surface Area Available for Contact (adult)	cm^2	2,500	USEPA 1998a	2,500	USEPA 1998a	
	SAc	Surface Area Available for Contact (child)	cm^2	2,200	USEPA 1998a	2,200	USEPA 1998a	For evaluating child exposure to noncarcinogens:
	EF	Exposure Frequency	events/year	68 (2)	prof. judgement	34 (2)	prof. judgement	= Csoil x SA x EF x ED x AF x AbsD x CFx 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	24	USEPA 1991	7	USEPA 1993	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	2	USEPA 1993	For evaluating combined adult and child exposures:
	AFa	Adherence Factor (adult)	mg/cm^2-event	0.1	USEPA 1998a	0.1	USEPA 1998a	SFSadj =
	AFc	Adherence Factor (child)	mg/cm^2-event	0.2	USEPA 1998a	0.2	USEPA 1998a	= [(EDc x SAc x AFc) / BWc] + [(EDa x SAa x AFa) / BWa]
	AbsD	Dermal Absorption	unitless	As = 0.03; Cd = 0.001	USEPA 1998a	As = 0.03; Cd = 0.001	USEPA 1998a	Therefore,
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	CDIadj = Csoil x EF x CF x AbsD x SFSadj x 1/AT
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	70	USEPA 1991	
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	15	USEPA 1991	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	

NOTES: na: not applicable; -- not available

(1) RME Exposure frequency for **adult**: (34 wks/yr x 7 hrs/dy x 2 dys/wk) / 16 hrs/dy = 30 dys/yr; for **child**: (34 wks/yr x 7 hrs/dy x 2 dys/wk) / 14 hrs/dy = 34 dys/yr. The 7 hrs/dy for 2 dys/wk assumes weekend outdoor exposure (EPA 1997).

(1a) CT Exposure frequency for **adult**: (34 wks/yr x 7 hrs/day x 1 day/wk) / 16 hrs/dy = 15 days/yr; for **child**: (34 wks/yr x 7 hrs/day x 1 day/wk) / 14 hrs/day = 17 days/year.

(2) Exposure frequency calculated by: 34 weeks/year x 2 events/week = 68 events/year for RME and 34 weeks/year x 1 event/week = 34 events/year for CT.

(3) 120 mg/day is the mean value of soil intake from study, whereas 300 mg/day is the RME value (90th percentile) from study.

TABLE 4.9
VALUES USED FOR DAILY INTAKE CALCULATIONS
Public Recreational Exposure

Scenario Timeframe: Current/Future
Medium: Soil/Sediment
Exposure Medium: Soil/Sediment
Exposure Point: Soil/Sediment at Various Locations
Receptor Population: Recreational Visitors
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Csed	Chemical Concentration in Sediments	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	Chronic Daily Intake (CDI) (mg/kg/day) =
	IRa-sed	Ingestion Rate of Sediment (adult)	mg/day	100	EPA 1991	50	EPA 1993	
	IRc-sed	Ingestion Rate of Sediment (child)	mg/day	300 (1)	USEPA 1999b	120 (1)	van Wijnen 1990	For evaluating child exposures to noncarcinogens :
	EF	Exposure Frequency	days/year	32	prof. judgement	16	prof. judgement	= Csed x IR-sed x EF x ED x AbsO x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	24	USEPA 1991	7	USEPA 1993	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	2	USEPA 1993	For evaluating combined adult and child exposures:
	AbsO	Gastrointestinal Absorption	unitless	chemical specific (2)	USEPA 2000	chemical specific (2)	USEPA 2000	
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	IngFadj = (IRc-sed x EDc / BWc) + (IRa-sed x EDa / BWa)
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	70	USEPA 1991	Therefore,
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	15	USEPA 1991	CDIadj = Csed x EF x AbsO x CF x IngFadj x 1/AT
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	
Dermal Contact	Csed	Chemical Concentration in Sediments	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	CDI (mg/kg-day) =
	SAa	Surface Area Available for Contact (adult)	cm^2	18,000	USEPA 1998a	18,000	USEPA 1998a	
	SAc	Surface Area Available for Contact (child)	cm^2	6,500	USEPA 1998a	6,500	USEPA 1998a	For evaluating child exposures to noncarcinogens :
	EF	Exposure Frequency	events/year	32	prof. judgement	16	prof. judgement	= Csed x SA x EF x ED x AF x AbsD x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	24	USEPA 1991	7	USEPA 1993	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	2	USEPA 1993	For evaluating combined adult and child exposures:
	AFa	Adherence Factor (adult)	mg/cm^2-event	0.1	USEPA 1998a	0.1	USEPA 1998a	SFSadj =
	AFc	Adherence Factor (child)	mg/cm^2-event	0.2	USEPA 1998a	0.2	USEPA 1998a	= [(EDc x SAc x AFc) / BWc] + [(EDa x SAa x AFa) / BWa]
	AbsD	Dermal Absorption	unitless	As = 0.03; Cd = 0.001	USEPA 1998a	As = 0.03; Cd = 0.001	USEPA 1998a	Therefore,
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	CDIadj = Csed x EF x CF x AbsD x SFSadj x 1/AT
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	70	USEPA 1991	
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	15	USEPA 1991	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	

NOTES: na: not applicable; -- not available

(1) 120 mg/day is the mean value of soil intake from study, whereas 300 mg/day is the RME value (90th percentile) from study.

(2) Gastrointestinal absorption for arsenic is 60%, for all other chemicals it was assumed to be 100%.

TABLE 4.10
VALUES USED FOR DAILY INTAKE CALCULATIONS
Public Recreational Exposure

Scenario Timeframe: Current/Future
Medium: Surface Water
Exposure Medium: Surface Water
Exposure Point: Surface Water at Various Locations
Receptor Population: Recreational Visitors
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Incidental	CW	Chemical Concentration in Surface Water	µg/l	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	Chronic Daily Intake (CDI) (mg/kg/day) =
Ingestion	IR-W	Ingestion Rate of Surface Water	mL/hour	30	USEPA 1998b	30	USEPA 1998b	
	EF	Exposure Frequency	days/year	32	prof. judgement	16	prof. judgement	For evaluating child exposure to noncarcinogens:
	ET	Exposure Time	hour/day	1	USEPA 1997	1	USEPA 1997	= CW x IR-W x EF x ET x ED x CF1 x CF2 x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	24 (1)	USEPA 1991	7	USEPA 1993	
	EDc	Exposure Duration (child)	years	6 (1)	USEPA 1991	2	USEPA 1993	For evaluating combined adult and child exposures:
	CF1	Conversion Factor 1	mg/µg	0.001	na	0.001	na	IngFadj = (EDc / BWc) + (EDa / BWa)
	CF2	Conversion Factor 2	L/mL	0.001	na	0.001	na	Therefore,
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	70	USEPA 1991	CDIadj = CW x IR-W x EF x ET x CF1 x CF2 x IngFadj x 1/AT
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	15	USEPA 1991	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	

NOTES: na: not applicable; -- not available; * not quantitatively evaluated, see text

(1) USEPA 1991 recommends an adult/child exposure duration of 24/6 years for ingestion of soil; For consistency, an exposure duration of 24/6 years was selected for ingestion of surface water.

TABLE 4.11

VALUES USED FOR DAILY INTAKE CALCULATIONS

Public Recreational Exposure

Scenario Timeframe: Current/Future
Medium: Surface Water/Sediment
Exposure Medium: Animal Tissue
Exposure Point: Fish from Lower Coeur d'Alene River
Receptor Population: Recreational Visitors
Receptor Age: Adults

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Cfish	Chemical Concentration in Fish	mg/kg	See Table 3	See Table 3	See Table 3	See Table 3	Chronic Daily Intake (CDI)(mg/kg/day) =
	IR-F	Ingestion Rate of Fish	g/day	46	USEPA 1997/ATSDR 1989	25	USEPA 1997	
	EF	Exposure Frequency	days/year	365 (1)	prof. judgement	365 (1)	prof. Judgement	For evaluating noncarcinogens:
	ED	Exposure Duration	years	30	USEPA 1991	9	USEPA 1993	= Cfish x IR-F x EF x ED x CF x 1/BW x 1/AT-N
	CF	Conversion Factor	kg/g	1.0E-03	na	1.0E-03	na	
	BW	Body Weight	kilograms	70	USEPA 1991	70	USEPA 1991	For evaluating carcinogens:
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	= Cfish x IR-F x EF x ED x CF x 1/BW x 1/AT-C
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	

NOTES: na: not applicable; -- not available

(1) Ingestion rate of fish is an average daily consumption rate, therefore 365 days/year was selected as the frequency of exposure for both the RME and CT scenarios.

TABLE 4.12
VALUES USED FOR DAILY INTAKE CALCULATIONS
Occupational Exposure

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface/Subsurface Soil
Exposure Point: Construction Site Soil
Receptor Population: Construction Workers
Receptor Age: Adults

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	Chronic Daily Intake (CDI) (mg/kg/day) =
	IR-soil	Ingestion Rate of Soil	mg/day	300	USEPA 1998c	200	USEPA 1998c	
	EF	Exposure Frequency	days/year	195 (1)	prof. judgement	43(1)	prof. Judgement	For evaluating noncarcinogens:
	ED	Exposure Duration	years	25	USEPA 1991	6.6	USEPA 1997	= Csoil x IR-soil x EF x ED x AbsO x CF x 1/BW x 1/AT-N
	AbsO	Gastrointestinal Absorption	unitless	chem-specific (2)	USEPA 2000	chem-specific (2)	USEPA 2000	
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.00E-06	na	
	BW	Body Weight	kilograms	70	USEPA 1991	70	USEPA 1991	For evaluating carcinogens:
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	= Csoil x IR-soil x EF x ED x AbsO x CF x 1/BW x 1/AT-C
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	
Dermal Contact	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	See Table Series 3	See Table Series 3	CDI (mg/kg-day) =
	SA	Surface Area Available for Contact	cm^2	2,500	USEPA 1998a	2500	USEPA 1998a	
	EF	Exposure Frequency	events/year	195 (1)	prof. judgement	43(1)	prof. Judgement	For evaluating noncarcinogens:
	ED	Exposure Duration	years	25	USEPA 1991	6.6	USEPA 1997	= Csoil x SA x EF x ED x AF x AbsD x CF x 1/BW x 1/AT-N
	AF	Adherence Factor	mg/cm^2-event	0.1	USEPA 1998a	0.1	USEPA 1998a	
	AbsD	Dermal Absorption	unitless	chem-specific	USEPA 1998d	chem-specific	USEPA 1998d	For evaluating carcinogens:
	CF	Conversion Factor	kg/mg	1.0E-06	na	1.0E-06	na	= Csoil x SA x EF x ED x AF x AbsD x CF x 1/BW x 1/AT-C
	BW	Body Weight	kilograms	70	USEPA 1991	70	USEPA 1991	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	25,550	USEPA 1989	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	ED x 365	USEPA 1989	

NOTES: na: not applicable; -- not available

(1) Exposure frequency is calculated assuming 9 months of construction work (excluding 3 months of snow cover) for the RME case: 39 weeks/year x 5 days/week = 195 days/year, assumes 2 months of construction work for the CT case.

(2) Gastrointestinal absorption for arsenic is 60%, for all other chemicals it is assumed to be 100%.

TABLE 4.13

VALUES USED FOR DAILY INTAKE CALCULATIONS

Modern Subsistence Exposure

Scenario Timeframe: Future

Medium: Surface Water/Sediment

Exposure Medium: Animal Tissue

Exposure Point: Fish from Lower Coeur d'Alene River

Receptor Population: Tribal Member

Receptor Age: Adults

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Cfish	Chemical Concentration in Fish	mg/kg	See Table 3	See Table 3	--	--	Chronic Daily Intake (CDI)(mg/kg/day) =
	IR-F	Ingestion Rate of Fish	g/day	170	USEPA 1997	--	--	
	EF	Exposure Frequency	days/year	365	na	--	--	For evaluating noncarcinogens:
	ED	Exposure Duration	years	70	Lifetime	--	--	= Cfish x IR-F x EF x ED x CF x 1/BW x 1/AT-N
	CF	Conversion Factor	kg/g	1.0E-03	na	--	--	
	BW	Body Weight	kilograms	70	USEPA 1991	--	--	For evaluating carcinogens:
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	--	--	= Cfish x IR-F x EF x ED x CF x 1/BW x 1/AT-C
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	--	--	

NOTES: na: not applicable

TABLE 4.14

VALUES USED FOR DAILY INTAKE CALCULATIONS

Modern Subsistence Exposure

Scenario Timeframe: Future

Medium: Surface Water/Sediment

Exposure Medium: Plant Tissue

Exposure Point: Water Potato from Lower Coeur d'Alene River

Receptor Population: Tribal Member

Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Cpotato	Chemical Concentration in Water Potato	mg/kg	See Table 3	See Table 3	--	--	Chronic Daily Intake (CDI)(mg/kg/day) =
	IR-P	Ingestion Rate of Water Potato	g/kg/day	8.2 (1)	Harris and Harper 1997	--	--	
	FI	Fraction Ingested (2)	unitless	0.2	prof. judgement	--	--	
	EF	Exposure Frequency	days/year	365	na	--	--	For evaluating noncarcinogens:
	ED	Exposure Duration	years	70	Lifetime	--	--	= Cpotato x IR-F x EF x FI x ED x CF x 1/AT-N
	CF	Conversion Factor	kg/g	1.0E-03	na	--	--	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	--	--	For evaluating carcinogens:
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	--	--	= Cpotato x IR-F x EF x ED x CF x 1/AT-C

NOTES: na: not applicable

(1) Ingestion rate incorporates the body weights of all participants in the study (children and adults) from Harris and Harper 1997.

(2) Assumes twenty percent of the ingested plant tissue is from the impacted floodplain area.

TABLE 4.15
VALUES USED FOR DAILY INTAKE CALCULATIONS
Modern Subsistence Exposure

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Surface Soil
Exposure Point: Surface Soil at Lower Coeur d'Alene River
Receptor Population: Tribal Member
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	na	na	Chronic Daily Intake (CDI) (mg/kg/day) =
	IRSa	Ingestion Rate of Soil (adult)	mg/day	300 (1)	USEPA 1999a	na	na	
	IRSc	Ingestion Rate of Soil (child)	mg/day	300 (1)	USEPA 1999a	na	na	For evaluating child exposure to noncarcinogens:
	EF	Exposure Frequency	days/year	60 (2)	Every day	na	na	= Csoil x IRS x EF x ED x AbsO x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating combined adult and child exposures:
	AbsO	Gastrointestinal Absorption	unitless	chemical specific (5)	USEPA 2000	na	na	
	CF	Conversion Factor	kg/mg	1.0E-06	na	na	na	IngFadj =
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	[(EDc x IRSc) / BWc] + [(EDa x IRSa) / BWa]
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	Therefore,
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	CDIadj = Csoil x IngFadj x EF x AbsO x CF x 1/AT
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	
Dermal Contact	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	na	na	CDI (mg/kg-day) =
	SAa	Surface Area Available for Contact (adult)	cm^2	5700 (3)	USEPA 1999a	na	na	
	SAc	Surface Area Available for Contact (child)	cm^2	2800 (3)	USEPA 1999a	na	na	For evaluating child exposure to noncarcinogens :
	EF	Exposure Frequency	events/year	60 (2)	professional judgement	na	na	= Csoil x SA x EF x ED x AF x AbsD x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating carcinogens (combined adult and child):
	AFa	Adherence Factor (adult)	mg/cm^2-event	0.8 (4)	USEPA 1997	na	na	SFSadj =
	AFc	Adherence Factor (child)	mg/cm^2-event	0.8 (4)	USEPA 1997	na	na	= [(EDc x SAc x AFc) / BWc] + [(EDa x SAa x AFa) / BWa]
	AbsD	Dermal Absorption	unitless	As = 0.03; Cd = 0.001	USEPA 1998a	na	na	Therefore,
	CF	Conversion Factor	kg/mg	1.0E-06	na	na	na	CDIadj = Csoil x EF x CF x AbsD x SFSadj x 1/AT
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	

NOTES: na: not applicable

(1) Soil ingestion rate based on a study of children at campgrounds.

(2) Assumes modern tribal members live in the floodplain during the two warmest months of the year (July and August).

(3) Skin surface area for adult represents head, hands, forearms, and lower legs; skin surface area for child represents head, hands, forearms, lower legs, and feet.

(4) Adherence factor for reed gatherers.

TABLE 4.16

VALUES USED FOR DAILY INTAKE CALCULATIONS

Modern Subsistence Exposure

Scenario Timeframe: Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Sediment at Lower Coeur d'Alene River
Receptor Population: Tribal Member
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Csed	Chemical Concentration in Sediments	mg/kg	See Table Series 3	See Table Series 3	na	na	Chronic Daily Intake (CDI) (mg/kg/day) =
	IRa-sed	Ingestion Rate of Sediment (adult)	mg/day	300 (1)	USEPA 1999a	na	na	
	IRc-sed	Ingestion Rate of Sediment (child)	mg/day	300 (1)	USEPA 1999a	na	na	For evaluating child exposures to noncarcinogens :
	EF	Exposure Frequency	days/year	60 (2)	prof. judgement	na	na	= Csed x IR-sed x EF x ED x AbsO x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating combined adult and child exposures:
	AbsO	Gastrointestinal Absorption	unitless	chemical specific (5)	USEPA 2000	na	na	
	CF	Conversion Factor	kg/mg	1.0E-06	na	na	na	IngFadj = (IRc-sed x EDc / BWc) + (IRa-sed x EDa / BWa)
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	Therefore,
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	CDIadj = Csed x EF x AbsO x CF x IngFadj x 1/AT
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	
Dermal Contact	Csed	Chemical Concentration in Sediments	mg/kg	See Table Series 3	See Table Series 3	na	na	CDI (mg/kg-day) =
	SAa	Surface Area Available for Contact (adult)	cm^2	18000 (3)	USEPA 1998a	na	na	
	SAc	Surface Area Available for Contact (child)	cm^2	6500 (3)	USEPA 1998a	na	na	For evaluating child exposures to noncarcinogens :
	EF	Exposure Frequency	events/year	60 (2)	prof. judgement	na	na	= Csed x SA x EF x ED x AF x AbsD x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating combined adult and child exposures:
	AFa	Adherence Factor (adult)	mg/cm^2-event	0.8 (4)	USEPA 1997	na	na	SFSadj =
	AFc	Adherence Factor (child)	mg/cm^2-event	0.8 (4)	USEPA 1997	na	na	= [(EDc x SAc x AFc) / BWc] + [(EDa x SAa x AFa) / BWa]
	AbsD	Dermal Absorption	unitless	As = 0.03; Cd = 0.001	USEPA 1998a	na	na	Therefore,
	CF	Conversion Factor	kg/mg	1.0E-06	na	na	na	CDIadj = Csed x EF x CF x AbsD x SFSadj x 1/AT
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	

NOTES: na: not applicable; -- not available

(1) Soil ingestion rate based on a study of children at campgrounds.

(2) Assumes modern subsistence lifestyle involves living near the Lower Coeur d'Alene River for two months of the year (July and August).

(3) Assumes full body exposure.

(4) Adherence factor for reed gatherers.

TABLE 4.17

VALUES USED FOR DAILY INTAKE CALCULATIONS

Modern Subsistence Exposure

Scenario Timeframe: Future
Medium: Surface Water
Exposure Medium: Undisturbed Surface Water
Exposure Point: Undisturbed Surface Water in Lower Coeur d'Alene River
Receptor Population: Tribal Member
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	CW	Chemical Concentration in Water	µg/l	See Table Series 3	See Table Series 3	na	na	Chronic Daily Intake (CDI) (mg/kg/day) =
	IR-Wa	Ingestion Rate of Water (adult)	liters/day	3	Harris and Harper 1997	na	na	
	IR-Wc	Ingestion Rate of Water (child)	liters/day	1.5 (1)	professional judgement	na	na	For evaluating child exposure to noncarcinogens:
	EF	Exposure Frequency	days/year	60 (2)	professional judgement	na	na	= CW x IR-W x EF x ED x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating combined adult and child exposures:
	CF	Conversion Factor	mg/µg	0.001	na	na	na	IngFadj =
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	= [(EDc x IR-Wc) / BWc] + [(EDa x IR-Wa) / BWa]
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	Therefore,
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	CDIadj = CW x EF x CF x IngFadj x 1/AT
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	

NOTES: na: not applicable

(1) Since the adult water ingestion rate is 150% of the default value, a child ingestion rate equivalent to 150% of the value used for child residential tap water consumption was used.

(2) Assumes modern subsistence lifestyle involves living near the Lower Coeur d'Alene River for two months of the year (July and August).

TABLE 4.18

VALUES USED FOR DAILY INTAKE CALCULATIONS

Modern Subsistence Exposure

Scenario Timeframe: Future
Medium: Surface Water
Exposure Medium: Disturbed Surface Water
Exposure Point: Disturbed Surface Water in Lower Coeur d'Alene River
Receptor Population: Tribal Member
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Incidental	CW	Chemical Concentration in Surface Water	µg/l	See Table Series 3	See Table Series 3	na	na	Chronic Daily Intake (CDI) (mg/kg/day) =
Ingestion	IR-W	Ingestion Rate of Surface Water	mL/hour	30	USEPA 1998b	na	na	
	EF	Exposure Frequency	days/year	60 (1)	prof. judgement	na	na	For evaluating child exposure to noncarcinogens:
	ET	Exposure Time	hour/day	1	USEPA 1997	na	na	= CW x IR-W x EF x ET x ED x CF1 x CF2 x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating combined adult and child exposures:
	CF1	Conversion Factor 1	mg/µg	0.001	na	na	na	IngFadj = (EDc / BWc) + (EDa / BWa)
	CF2	Conversion Factor 2	L/mL	0.001	na	na	na	Therefore,
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	CDIadj = CW x IR-W x EF x ET x CF1 x CF2 x IngFadj x 1/AT
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	

NOTES: na: not applicable

* not quantitatively evaluated, see text

(1) Assumes modern subsistence lifestyle involves living near the Lower Coeur d'Alene River for two months of the year (July and August).

TABLE 4.19

VALUES USED FOR DAILY INTAKE CALCULATIONS

Traditional Subsistence Exposure

Scenario Timeframe: Future

Medium: Surface Water/Sediment

Exposure Medium: Animal Tissue

Exposure Point: Fish from Lower Coeur d'Alene River

Receptor Population: Tribal Member

Receptor Age: Adults

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Cfish	Chemical Concentration in Fish	mg/kg	See Table 3	See Table 3	--	--	Chronic Daily Intake (CDI)(mg/kg/day) =
	IR-F	Ingestion Rate of Fish	g/day	540	Harris & Harper 1997	--	--	
	EF	Exposure Frequency	days/year	365	na	--	--	For evaluating noncarcinogens:
	ED	Exposure Duration	years	70	Lifetime	--	--	= Cfish x IR-F x EF x ED x CF x 1/BW x 1/AT-N
	CF	Conversion Factor	kg/g	1.0E-03	na	--	--	
	BW	Body Weight	kilograms	70	USEPA 1991	--	--	For evaluating carcinogens:
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	--	--	= Cfish x IR-F x EF x ED x CF x 1/BW x 1/AT-C
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	--	--	

NOTES: na: not applicable

TABLE 4.20

VALUES USED FOR DAILY INTAKE CALCULATIONS

Traditional Subsistence Exposure

Scenario Timeframe: Future

Medium: Surface Water/Sediment

Exposure Medium: Plant Tissue

Exposure Point: Water Potato from Lower Coeur d'Alene River

Receptor Population: Tribal Member

Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Cpotato	Chemical Concentration in Water Potato	mg/kg	See Table 3	See Table 3	--	--	Chronic Daily Intake (CDI)(mg/kg/day) =
	IR-F	Ingestion Rate of Water Potato	g/kg/day	8.2 (1)	Harris and Harper 1997	--	--	
	EF	Exposure Frequency	days/year	365	na	--	--	For evaluating noncarcinogens:
	ED	Exposure Duration	years	70	Lifetime	--	--	= Cpotato x IR-F x EF x ED x CF x 1/AT-N
	CF	Conversion Factor	kg/g	1.0E-03	na	--	--	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	--	--	For evaluating carcinogens:
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	--	--	= Cpotato x IR-F x EF x ED x CF x 1/AT-C

NOTES: na: not applicable

(1) Ingestion rate incorporates the body weights of all participants in the study (children and adults) from Harris and Harper 1997.

TABLE 4.21
VALUES USED FOR DAILY INTAKE CALCULATIONS
Traditional Subsistence Exposure

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Surface Soil
Exposure Point: Surface Soil at Lower Coeur d'Alene River
Receptor Population: Tribal Member
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	na	na	Chronic Daily Intake (CDI) (mg/kg/day) =
	IRSa	Ingestion Rate of Soil (adult)	mg/day	300 (1)	USEPA 1999a	na	na	
	IRSc	Ingestion Rate of Soil (child)	mg/day	300 (1)	USEPA 1999a	na	na	For evaluating child exposure to noncarcinogens:
	EF	Exposure Frequency	days/year	365 (2)	Every day	na	na	= Csoil x IRS x EF x ED xAbsO x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating combined adult and child exposures:
	AbsO	Gastrointestinal Absorption	unitless	chemical specific (5)	USEPA 2000	na	na	
	CF	Conversion Factor	kg/mg	1.0E-06	na	na	na	IngFadj =
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	[(EDc x IRSc) / BWc] + [(EDa x IRSa) / BWa]
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	Therefore,
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	CDIadj = Csoil x IngFadj x EF x AbsO x CF x 1/AT
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	
Dermal Contact	Csoil	Chemical Concentration in Soil	mg/kg	See Table Series 3	See Table Series 3	na	na	CDI (mg/kg-day) =
	SAa	Surface Area Available for Contact (adult)	cm^2	2500 (3)	USEPA 1998a	na	na	
	SAc	Surface Area Available for Contact (child)	cm^2	2200 (3)	USEPA 1998a	na	na	For evaluating child exposure to noncarcinogens :
	EF	Exposure Frequency	events/year	365	Every day	na	na	= Csoil x SA x EF x ED x AF x AbsD x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating carcinogens (combined adult and child):
	AFa	Adherence Factor (adult)	mg/cm^2-event	0.8 (4)	USEPA 1997	na	na	SFSadj =
	AFc	Adherence Factor (child)	mg/cm^2-event	0.8 (4)	USEPA 1997	na	na	= [(EDc x SAc x AFc) / BWc] + [(EDa x SAa x AFa) / BWa]
	AbsD	Dermal Absorption	unitless	As = 0.03; Cd = 0.001	USEPA 1998a	na	na	Therefore,
	CF	Conversion Factor	kg/mg	1.0E-06	na	na	na	CDIadj = Csoil x EF x CF x AbsD x SFSadj x 1/AT
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	

NOTES: na: not applicable
(1) Soil ingestion rate based on a study of children at campgrounds.
(2) Assumes daily exposure throughout lifetime.
(3) Skin surface area for adult represents face, hands, and forearms; skin surface area for child represents face, hands, forearms, lower legs, and feet.
(4) Adherence factor for reed gatherers.

TABLE 4.22

VALUES USED FOR DAILY INTAKE CALCULATIONS

Traditional Subsistence Exposure

Scenario Timeframe: Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Sediment at Lower Coeur d'Alene River
Receptor Population: Tribal Member
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	Csed	Chemical Concentration in Sediments	mg/kg	See Table Series 3	See Table Series 3	na	na	Chronic Daily Intake (CDI) (mg/kg/day) =
	IRa-sed	Ingestion Rate of Sediment (adult)	mg/day	300 (1)	USEPA 1999a	na	na	
	IRc-sed	Ingestion Rate of Sediment (child)	mg/day	300 (1)	USEPA 1999a	na	na	For evaluating child exposures to noncarcinogens :
	EF	Exposure Frequency	days/year	210 (2)	prof. judgement	na	na	= Csed x IR-sed x EF x ED x AbsO x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating combined adult and child exposures:
	AbsO	Gastrointestinal Absorption	unitless	chemical specific (5)	USEPA 2000	na	na	
	CF	Conversion Factor	kg/mg	1.0E-06	na	na	na	IngFadj = (IRc-sed x EDc / BWc) + (IRa-sed x EDa / BWa)
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	Therefore,
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	CDIadj = Csed x EF x AbsO x CF x IngFadj x 1/AT
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	
Dermal Contact	Csed	Chemical Concentration in Sediments	mg/kg	See Table Series 3	See Table Series 3	na	na	CDI (mg/kg-day) =
	SAa	Surface Area Available for Contact (adult)	cm^2	18000 (3)	USEPA 1998a	na	na	
	SAc	Surface Area Available for Contact (child)	cm^2	6500 (3)	USEPA 1998a	na	na	For evaluating child exposures to noncarcinogens :
	EF	Exposure Frequency	events/year	210 (2)	prof. judgement	na	na	= Csed x SA x EF x ED x AF x AbsD x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating combined adult and child exposures:
	AFa	Adherence Factor (adult)	mg/cm^2-event	0.8 (4)	USEPA 1997	na	na	SFSadj =
	AFc	Adherence Factor (child)	mg/cm^2-event	0.8 (4)	USEPA 1997	na	na	= [(EDc x SAc x AFc) / BWc] + [(EDa x SAa x AFa) / BWa]
	AbsD	Dermal Absorption	unitless	As = 0.03; Cd = 0.001	USEPA 1998a	na	na	Therefore,
	CF	Conversion Factor	kg/mg	1.0E-06	na	na	na	CDIadj = Csed x EF x CF x AbsD x SFSadj x 1/AT
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	

NOTES: na: not applicable; -- not available

(1) Soil ingestion rate based on a study of children at campgrounds.

(2) Assumes water potato harvesting or recreational activities associated with sediment/surface water in the Lower CdA River occurs 7 months of the year (April to October) due to weather constraints.

(3) Assumes full body exposure.

(4) Adherence factor for reed gatherers.

TABLE 4.23

VALUES USED FOR DAILY INTAKE CALCULATIONS

Traditional Subsistence Exposure

Scenario Timeframe: Future
Medium: Surface Water
Exposure Medium: Undisturbed Surface Water
Exposure Point: Undistrubed Surface Water in Lower Coeur d'Alene River
Receptor Population: Tribal Member
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	CW	Chemical Concentration in Water	µg/l	See Table Series 3	See Table Series 3	na	na	Chronic Daily Intake (CDI) (mg/kg/day) =
	IR-Wa	Ingestion Rate of Water (adult)	liters/day	3	Harris and Harper 1997	na	na	
	IR-Wc	Ingestion Rate of Water (child)	liters/day	1.5 (1)	professional judgement	na	na	For evaluating child exposure to noncarcinogens:
	EF	Exposure Frequency	days/year	365	Every day	na	na	= CW x IR-W x EF x ED x CF x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating combined adult and child exposures:
	CF	Conversion Factor	mg/µg	0.001	na	na	na	IngFadj =
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	= [(EDc x IR-Wc) / BWc] + [(EDa x IR-Wa) / BWa]
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	Therefore,
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	CDIadj = CW x EF x CF x IngFadj x 1/AT
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	

NOTES: na: not applicable

(1) Since the adult water ingestion rate is 150% of the default value, a child ingestion rate equivalent to 150% of the value used for child residential tap water consumption was used.

TABLE 4.24

VALUES USED FOR DAILY INTAKE CALCULATIONS

Traditional Subsistence Exposure

Scenario Timeframe: Future
Medium: Surface Water
Exposure Medium: Disturbed Surface Water
Exposure Point: Disturbed Surface Water in Lower Coeur d'Alene River
Receptor Population: Tribal Member
Receptor Age: Adults and Children

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Incidental	CW	Chemical Concentration in Surface Water	µg/l	See Table Series 3	See Table Series 3	na	na	Chronic Daily Intake (CDI) (mg/kg/day) =
Ingestion	IR-W	Ingestion Rate of Surface Water	mL/hour	30	USEPA 1998b	na	na	
	EF	Exposure Frequency	days/year	210 (1)	prof. judgement	na	na	For evaluating child exposure to noncarcinogens:
	ET	Exposure Time	hour/day	1	USEPA 1997	na	na	= CW x IR-W x EF x ET x ED x CF1 x CF2 x 1/BW x 1/AT-N
	EDa	Exposure Duration (adult)	years	64	Lifetime (70 years total)	na	na	
	EDc	Exposure Duration (child)	years	6	USEPA 1991	na	na	For evaluating combined adult and child exposures:
	CF1	Conversion Factor 1	mg/µg	0.001	na	na	na	IngFadj = (EDc / BWc) + (EDa / BWa)
	CF2	Conversion Factor 2	L/mL	0.001	na	na	na	Therefore,
	BWa	Body Weight (adult)	kilograms	70	USEPA 1991	na	na	CDIadj = CW x IR-W x EF x ET x CF1 x CF2 x IngFadj x 1/AT
	BWc	Body Weight (child)	kilograms	15	USEPA 1991	na	na	
	AT-C	Averaging Time (Cancer)	days	25,550	USEPA 1989	na	na	
	AT-N	Averaging Time (Non-cancer)	days	ED x 365	USEPA 1989	na	na	

NOTES: na: not applicable; -- not available; * not quantitatively evaluated, see text

(1) Assumes recreational activities associated with surface water in the Lower CdA River occurs only seven months of the year due to weather constraints.

Table 4 References

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TABLE 5.1

NON-CANCER TOXICITY DATA -- ORAL/DERMAL

Coeur d'Alene River Basin

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD Value	Oral RfD Units	Oral to Dermal Adjustment Factor (1)	Adjusted Dermal RfD (2)	Units	Endpoint/ Primary Target Organ	Combined Uncertainty/Modifying Factors	Sources of RfD/ Target Organ	Dates of RfD: Target Organ (MM/DD/YY)
Antimony	Chronic	4.00E-04	mg/kg-day	NA	NA	mg/kg-day	LOAEL/longevity, blood chemistry	1000	IRIS	10/25/99
Arsenic	Chronic	3.00E-04	mg/kg-day	NA	NA	mg/kg-day	NOAEL/skin pigmentation	3	IRIS	10/25/99
Cadmium (food)	Chronic	1.00E-03	mg/kg-day	0.025	2.50E-05	mg/kg-day	NOAEL/proteinuria	10	IRIS	10/25/99
Cadmium (water)	Chronic	5.00E-04	mg/kg-day	NA (3)	NA	NA	NOAEL/proteinuria	10	IRIS	10/25/99
Iron	NS	3.00E-01	mg/kg-day	NA	NA	mg/kg-day	NS	1	Region III RBCs & NCEA	10/25/99
Manganese (food)										
child and child/adult	Chronic	4.70E-02	mg/kg-day	NA	NA	mg/kg-day	NOAEL/Central Nervous System	1	IRIS	10/25/99
adult only	Chronic	1.40E-01	mg/kg-day	NA	NA	mg/kg-day	NOAEL/Central Nervous System	1	IRIS	10/25/99
Manganese (water)	Chronic	4.70E-02	mg/kg-day	NA (3)	NA	mg/kg-day	NOAEL/Central Nervous System	3	IRIS	10/25/99
Mercury	Chronic	3.00E-04	mg/kg-day	NA	NA	mg/kg-day	LOAEL/kidney damage, autoimmunity	1000	IRIS	10/25/99
Methylmercury (fish)	Chronic	1.00E-04	mg/kg-day	NA	NA	mg/kg-day	Benchmark Dose/develpmental neurological effects	10	IRIS	10/25/99
Zinc	Subchronic (10 weeks)	3.00E-01	mg/kg-day	NA	NA	mg/kg-day	LOAEL/enzyme-level effects	3	IRIS	10/25/99

N/A = Not Applicable

NS = Not Specified

NOAEL = No observed adverse effect level

LOAEL = Lowest obeserved adverse effect level

(1) The oral RfD will be adjusted for cadmium (food) only; for all other COPCs, the oral RfD will be used to evaluate dermal exposures (U.S.EPA, 1999, Region 9 PRG Tables).

(2) Adjusted Dermal RfD = oral RfD x Oral to Dermal Adjustment Factor

(3) The dermal pathway will not be evaluated quantitatively for COPCs in water

TABLE 5.2

NON-CANCER TOXICITY DATA -- INHALATION

Coeur d'Alene River Basin

Chemical of Potential Concern	Chronic/ Subchronic	Value Inhalation RfC	Units	Adjusted Inhalation RfD (1)	Units	Primary Target Organ	Combined Uncertainty/Modifying Factors	Sources of RfC:RfD: Target Organ	Dates (MM/DD/YY)
Antimony	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Arsenic	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Cadmium	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Iron	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Manganese	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Mercury	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Zinc	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

N/A = Not Applicable

TABLE 6.1

CANCER TOXICITY DATA -- ORAL/DERMAL

Coeur d'Alene River Basin

Chemical of Potential Concern	Oral Cancer Slope Factor	Oral to Dermal Adjustment Factor (1)	Adjusted Dermal Cancer Slope Factor (2)	Units	Weight of Evidence/ Cancer Guideline Description	Source Target Organ	Date (MM/DD/YY)
Antimony	--	--	--	--	NYR	IRIS	10/25/99
Arsenic	1.50E+00	NA	NA	$(mg/kg\text{-}d)^{-1}$	A	liver, kidney: IRIS	10/25/99
Cadmium	--	--	--	--	NYR	IRIS	10/25/99
Iron	--	--	--	--	NYR	IRIS	10/25/99
Manganese	--	--	--	--	D	IRIS	10/25/99
Mercury	--	--	--	--	C	IRIS	10/25/99
Zinc	--	--	--	--	D	IRIS	10/25/99

--' = no value available

IRIS = Integrated Risk Information System

NYR = not yet rated

NA = not applicable

(1) Gastrointestinal absorption factor (U.S.EPA, 1998)

(2) The oral slope factor will be used to evaluate dermal exposures (U.S.EPA Region 9 PRG Tables)

EPA Group:

A - Human carcinogen

B1 - Probable human carcinogen - indicates that limited human

B2 - Probable human carcinogen - indicates sufficient evidence inadequate or no evidence in humans

C - Possible human carcinogen

D - Not classifiable as a human carcinogen

E - Evidence of noncarcinogenicity

TABLE 6.2

CANCER TOXICITY DATA -- INHALATION

Coeur d'Alene River Basin

Chemical of Potential Concern	Unit Risk	Units	Adjustment (2)	Inhalation Cancer Slope Factor	Units	Weight of Evidence/ Cancer Guideline Description	Source	Date (1) (MM/DD/YY)
Antimony	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Arsenic	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Cadmium	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Iron	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Manganese	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Mercury	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Zinc	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

N/A = Not Applicable

TABLE 7.1.1.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	8.25	mg/kg	8.25	mg/kg	M	1.05E-04	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.6E-01
	Arsenic	48.53	mg/kg	48.53	mg/kg	M	3.72E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.2E+00
	Cadmium	6.87	mg/kg	6.87	mg/kg	M	8.78E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	8.8E-02
	Iron	37703	mg/kg	37703	mg/kg	M	4.82E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.6E+00
	Manganese	2292	mg/kg	2292	mg/kg	M	2.93E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	6.2E-01
	Zinc	1199	mg/kg	1199	mg/kg	M	1.53E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.1E-02
	(Total)												3.9E+00
Dermal	Arsenic	48.53	mg/kg	48.53	mg/kg	M	4.1E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.4E-01
	Cadmium	6.87	mg/kg	6.87	mg/kg	M	1.9E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	7.7E-03
	(Total)												1.4E-01
												Total Hazard Index Across All Exposure Routes/Pathways	4.0E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.1.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	5.62	mg/kg	5.62	mg/kg	M	2.67E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	6.7E-02
	Arsenic	35.47	mg/kg	35.47	mg/kg	M	1.01E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.4E-01
	Cadmium	4.97	mg/kg	4.97	mg/kg	M	2.36E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.4E-02
	Iron	29992	mg/kg	29992	mg/kg	M	1.42E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.7E-01
	Manganese	1639	mg/kg	1639	mg/kg	M	7.78E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.7E-01
	Zinc	849	mg/kg	849	mg/kg	M	4.03E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.3E-02
	(Total)												1.1E+00
Dermal	Arsenic	35.47	mg/kg	35.47	mg/kg	M	2.22E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.4E-02
	Cadmium	4.97	mg/kg	4.97	mg/kg	M	1.04E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	4.2E-03
	(Total)												7.8E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.2E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.2.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	8.25	mg/kg	8.25	mg/kg	M	3.01E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	7.5E-02
	Arsenic	48.53	mg/kg	48.53	mg/kg	M	1.06E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.5E-01
	Cadmium	6.87	mg/kg	6.87	mg/kg	M	2.51E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.5E-02
	Iron	37703	mg/kg	37703	mg/kg	M	1.38E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.6E-01
	Manganese	2292	mg/kg	2292	mg/kg	M	8.37E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.8E-01
	Zinc	1199	mg/kg	1199	mg/kg	M	4.38E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.5E-02
	(Total)												1.1E+00
Dermal	Arsenic	48.53	mg/kg	48.53	mg/kg	M	1.22E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.1E-02
	Cadmium	6.87	mg/kg	6.87	mg/kg	M	5.75E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.3E-03
	(Total)												4.3E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.1E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.2.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	5.62	mg/kg	5.62	mg/kg	M	8.15E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.0E-02
	Arsenic	35.47	mg/kg	35.47	mg/kg	M	3.09E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.0E-01
	Cadmium	4.97	mg/kg	4.97	mg/kg	M	7.21E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	7.2E-03
	Iron	29992	mg/kg	29992	mg/kg	M	4.35E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.5E-01
	Manganese	1639	mg/kg	1639	mg/kg	M	2.38E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	5.1E-02
	Zinc	849	mg/kg	849	mg/kg	M	1.23E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.1E-03
	(Total)												3.3E-01
Dermal	Arsenic	35.47	mg/kg	35.47	mg/kg	M	7.05E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.3E-02
	Cadmium	4.97	mg/kg	4.97	mg/kg	M	3.29E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.3E-03
	(Total)												2.5E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.6E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.3.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.97	ug/L	0.97	ug/L	M	6.20E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.1E-01
												Total Hazard Index Across All Exposure Routes/Pathways	2.1E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.3.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.38	ug/L	0.38	ug/L	M	1.62E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.4E-02
												Total Hazard Index Across All Exposure Routes/Pathways	5.4E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.4.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.97	ug/L	0.97	ug/L	M	3.37E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.1E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.4.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.38	ug/L	0.38	ug/L	M	7.40E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.5E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.5E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.5.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Lower CDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	23.22	mg/kg	23.22	mg/kg	M	1.43E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.6E-02
	Arsenic	119.45	mg/kg	119.45	mg/kg	M	4.42E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.5E-01
	Cadmium	35.05	mg/kg	35.05	mg/kg	M	2.16E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.2E-02
	Iron	105451	mg/kg	105451	mg/kg	M	6.50E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.2E-01
	Manganese	9886	mg/kg	9886	mg/kg	M	6.09E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.3E-01
	Zinc	5666	mg/kg	5666	mg/kg	M	3.49E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E-02
	(Total)												5.6E-01
Dermal	Arsenic	119.45	mg/kg	119.45	mg/kg	M	5.36E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.8E-01
	Cadmium	35.05	mg/kg	35.05	mg/kg	M	5.24E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.1E-02
	(Total)												2.0E-01
												Total Hazard Index Across All Exposure Routes/Pathways	7.6E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.5.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Lower CDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	22.08	mg/kg	22.08	mg/kg	M	2.59E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	6.5E-03
	Arsenic	114.51	mg/kg	114.51	mg/kg	M	8.07E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.7E-02
	Cadmium	33.14	mg/kg	33.14	mg/kg	M	3.89E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.9E-03
	Iron	101280	mg/kg	101280	mg/kg	M	1.19E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.0E-02
	Manganese	9403	mg/kg	9403	mg/kg	M	1.10E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.3E-02
	Zinc	5337	mg/kg	5337	mg/kg	M	6.27E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.1E-03
	(Total)												1.0E-01
Dermal	Arsenic	114.51	mg/kg	114.51	mg/kg	M	2.57E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.6E-02
	Cadmium	33.14	mg/kg	33.14	mg/kg	M	2.48E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	9.9E-03
	(Total)												9.6E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.0E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.6.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Disturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	79.61	ug/L	79.61	ug/L	M	2.24E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.5E-02
	Cadmium	72.9	ug/L	72.9	ug/L	M	2.05E-05	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	4.1E-02
	Manganese	16651	ug/L	16651	ug/L	M	4.69E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.0E-01
	Mercury	4.5	ug/L	4.5	ug/L	M	1.27E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.2E-03
												Total Hazard Index Across All Exposure Routes/Pathways	2.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.6.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Disturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	75.64	ug/L	75.64	ug/L	M	1.07E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.6E-02
	Cadmium	54.82	ug/L	54.82	ug/L	M	7.72E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	1.5E-02
	Manganese	21269	ug/L	21269	ug/L	M	3.00E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	6.4E-02
	Mercury	4.51	ug/L	4.51	ug/L	M	6.35E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.1E-03
												Total Hazard Index Across All Exposure Routes/Pathways	1.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.7.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Lower CDAR
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	23.22	mg/kg	23.22	mg/kg	M	4.07E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.0E-01
	Arsenic	119.45	mg/kg	119.45	mg/kg	M	1.26E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.2E-01
	Cadmium	35.05	mg/kg	35.05	mg/kg	M	6.15E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	6.1E-02
	Iron	105451	mg/kg	105451	mg/kg	M	1.85E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	6.2E-01
	Manganese	9886	mg/kg	9886	mg/kg	M	1.73E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.7E-01
	Zinc	5666	mg/kg	5666	mg/kg	M	9.93E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.3E-02
	(Total)												1.6E+00
Dermal	Arsenic	119.45	mg/kg	119.45	mg/kg	M	2.72E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	9.1E-02
	Cadmium	35.05	mg/kg	35.05	mg/kg	M	2.66E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.1E-02
	(Total)												1.0E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.7E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.7.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Lower CDAR
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	22.08	mg/kg	22.08	mg/kg	M	7.74E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.9E-02
	Arsenic	114.51	mg/kg	114.51	mg/kg	M	2.41E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.0E-02
	Cadmium	33.14	mg/kg	33.14	mg/kg	M	1.16E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.2E-02
	Iron	101280	mg/kg	101280	mg/kg	M	3.55E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E-01
	Manganese	9403	mg/kg	9403	mg/kg	M	3.30E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	7.0E-02
	Zinc	5337	mg/kg	5337	mg/kg	M	1.87E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	6.2E-03
	(Total)												3.1E-01
Dermal	Arsenic	114.51	mg/kg	114.51	mg/kg	M	1.31E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.4E-02
	Cadmium	33.14	mg/kg	33.14	mg/kg	M	1.26E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	5.0E-03
	(Total)												4.9E-02
												Total Hazard Index Across All Exposure Routes/Pathways	7.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.8.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Lower CDAR
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	23.22	mg/kg	23.22	mg/kg	M	1.05E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.6E-02
	Arsenic	119.45	mg/kg	119.45	mg/kg	M	3.23E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-01
	Cadmium	35.05	mg/kg	35.05	mg/kg	M	1.58E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.6E-02
	Iron	105451	mg/kg	105451	mg/kg	M	4.75E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.6E-01
	Manganese	9886	mg/kg	9886	mg/kg	M	4.46E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	9.5E-02
	Zinc	5666	mg/kg	5666	mg/kg	M	2.55E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.5E-03
	(Total)												4.1E-01
Dermal	Arsenic	119.45	mg/kg	119.45	mg/kg	M	1.19E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.0E-02
	Cadmium	35.05	mg/kg	35.05	mg/kg	M	1.16E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	4.7E-03
	(Total)												4.4E-02
												Total Hazard Index Across All Exposure Routes/Pathways	4.6E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.8.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Lower CDAR
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	22.08	mg/kg	22.08	mg/kg	M	2.26E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	5.6E-03
	Arsenic	114.51	mg/kg	114.51	mg/kg	M	7.03E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.3E-02
	Cadmium	33.14	mg/kg	33.14	mg/kg	M	3.39E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.4E-03
	Iron	101280	mg/kg	101280	mg/kg	M	1.04E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.5E-02
	Manganese	9403	mg/kg	9403	mg/kg	M	9.62E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.0E-02
	Zinc	5337	mg/kg	5337	mg/kg	M	5.46E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.8E-03
	(Total)												8.9E-02
Dermal	Arsenic	114.51	mg/kg	114.51	mg/kg	M	5.91E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.0E-02
	Cadmium	33.14	mg/kg	33.14	mg/kg	M	5.70E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.3E-03
	(Total)												2.2E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.1E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.9.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Disturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	79.61	ug/L	79.61	ug/L	M	1.40E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.7E-02
	Cadmium	72.9	ug/L	72.9	ug/L	M	1.28E-05	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	2.6E-02
	Manganese	16651	ug/L	16651	ug/L	M	2.92E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	6.2E-02
	Mercury	4.5	ug/L	4.5	ug/L	M	7.89E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.6E-03
												Total Hazard Index Across All Exposure Routes/Pathways	1.4E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.9.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Disturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	75.64	ug/L	75.64	ug/L	M	6.63E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.2E-02
	Cadmium	54.82	ug/L	54.82	ug/L	M	4.81E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	9.6E-03
	Manganese	21269	ug/L	21269	ug/L	M	1.86E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	4.0E-02
	Mercury	4.51	ug/L	4.51	ug/L	M	3.95E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.3E-03
												Total Hazard Index Across All Exposure Routes/Pathways	7.3E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.10.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Disturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	79.61	ug/L	79.61	ug/L	M	5.18E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.7E-02
	Cadmium	72.9	ug/L	72.9	ug/L	M	4.75E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	9.5E-03
	Manganese	16651	ug/L	16651	ug/L	M	1.08E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.3E-02
	Mercury	4.5	ug/L	4.5	ug/L	M	2.93E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	9.8E-04
												Total Hazard Index Across All Exposure Routes/Pathways	5.1E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.10.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Disturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	75.64	ug/L	75.64	ug/L	M	2.58E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.6E-03
	Cadmium	54.82	ug/L	54.82	ug/L	M	1.87E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	3.7E-03
	Manganese	21269	ug/L	21269	ug/L	M	7.25E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.5E-02
	Mercury	4.51	ug/L	4.51	ug/L	M	1.54E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.1E-04
												Total Hazard Index Across All Exposure Routes/Pathways	2.83E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.11.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	21.08	mg/kg	21.08	mg/kg	M	4.83E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.2E-01
	Arsenic	108.91	mg/kg	108.91	mg/kg	M	1.05E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.0E-01
	Cadmium	30.71	mg/kg	30.71	mg/kg	M	7.03E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	7.0E-02
	Iron	97012	mg/kg	97012	mg/kg	M	2.22E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.4E-01
	Manganese	8919	mg/kg	8919	mg/kg	M	2.04E-02	mg/kg-d	1.4E-01	mg/kg-d	N/A	N/A	1.5E-01
	Zinc	4998	mg/kg	4998	mg/kg	M	1.14E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.8E-02
	(Total)												1.6E+00
Dermal	Arsenic	108.91	mg/kg	108.91	mg/kg	M	6.23E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.1E-02
	Cadmium	30.71	mg/kg	30.71	mg/kg	M	5.86E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.3E-03
	(Total)												2.3E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.6E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.11.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	19.98	mg/kg	19.98	mg/kg	M	6.73E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.7E-02
	Arsenic	104.41	mg/kg	104.41	mg/kg	M	2.11E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.0E-02
	Cadmium	29	mg/kg	29	mg/kg	M	9.76E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	9.8E-03
	Iron	92917	mg/kg	92917	mg/kg	M	3.13E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.0E-01
	Manganese	8449	mg/kg	8449	mg/kg	M	2.84E-03	mg/kg-d	1.4E-01	mg/kg-d	N/A	N/A	2.0E-02
	Zinc	4703	mg/kg	4703	mg/kg	M	1.58E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.3E-03
	(Total)												2.3E-01
Dermal	Arsenic	104.41	mg/kg	104.41	mg/kg	M	1.32E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.4E-03
	Cadmium	29	mg/kg	29	mg/kg	M	1.22E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	4.9E-04
	(Total)												4.9E-03
												Total Hazard Index Across All Exposure Routes/Pathways	2.3E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.12 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface Water/Sediment
Exposure Medium:	Plant Tissue
Exposure Point:	Water Potatoes from the Lower CDAR
Receptor Population:	Tribal Members
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion													
(With Skin)	Cadmium	0.489	mg/kg	0.489	mg/kg	M	8.02E-04	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	8.0E-01
(Without Skin)	Cadmium	0.291	mg/kg	0.291	mg/kg	M	4.77E-04	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	4.8E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.13 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Members
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	21.16	mg/kg	21.16	mg/kg	M	6.96E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.7E-01
	Arsenic	124.44	mg/kg	124.44	mg/kg	M	2.45E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.2E-01
	Cadmium	30.45	mg/kg	30.45	mg/kg	M	1.00E-04	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.0E-01
	Iron	97440	mg/kg	97440	mg/kg	M	3.20E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.1E+00
	Manganese	8960	mg/kg	8960	mg/kg	M	2.95E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	6.3E-01
	Zinc	4856	mg/kg	4856	mg/kg	M	1.60E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.3E-02
	(Total)												2.8E+00
Dermal	Arsenic	124.44	mg/kg	124.44	mg/kg	M	9.16E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.1E-01
	Cadmium	30.45	mg/kg	30.45	mg/kg	M	7.47E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.0E-02
	(Total)												3.4E-01
												Total Hazard Index Across All Exposure Routes/Pathways	3.2E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.14 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Members
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	21.16	mg/kg	21.16	mg/kg	M	1.96E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	4.90E-02
	Arsenic	124.44	mg/kg	124.44	mg/kg	M	6.91E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.30E-01
	Cadmium	30.45	mg/kg	30.45	mg/kg	M	2.82E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.82E-02
	Iron	97440	mg/kg	97440	mg/kg	M	9.02E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.01E-01
	Manganese	8960	mg/kg	8960	mg/kg	M	8.30E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.8E-01
	Zinc	4856	mg/kg	4856	mg/kg	M	4.50E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.50E-02
	(Total)												8.0E-01
Dermal	Arsenic	124.44	mg/kg	124.44	mg/kg	M	4.44E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.5E-01
	Cadmium	30.45	mg/kg	30.45	mg/kg	M	3.62E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.4E-02
	(Total)												1.6E-01
												Total Hazard Index Across All Exposure Routes/Pathways	9.6E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.15 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	25.20	mg/kg	25.20	mg/kg	M	8.28E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.1E-01
	Arsenic	120.96	mg/kg	120.96	mg/kg	M	2.39E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.0E-01
	Cadmium	39.33	mg/kg	39.33	mg/kg	M	1.29E-04	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.3E-01
	Iron	113073	mg/kg	113073	mg/kg	M	3.72E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E+00
	Manganese	10700	mg/kg	10700	mg/kg	M	3.52E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	7.5E-01
	Zinc	6400	mg/kg	6400	mg/kg	M	2.10E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.0E-02
	(Total)												3.2E+00
Dermal	Arsenic	120.96	mg/kg	120.96	mg/kg	M	2.07E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.9E-01
	Cadmium	39.33	mg/kg	39.33	mg/kg	M	2.24E-06	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	9.0E-02
	(Total)												7.8E-01
												Total Hazard Index Across All Exposure Routes/Pathways	4.0E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.16 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	25.20	mg/kg	25.20	mg/kg	M	2.33E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	5.8E-02
	Arsenic	120.96	mg/kg	120.96	mg/kg	M	6.72E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.2E-01
	Cadmium	39.33	mg/kg	39.33	mg/kg	M	3.64E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.6E-02
	Iron	113073	mg/kg	113073	mg/kg	M	1.05E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.5E-01
	Manganese	10700	mg/kg	10700	mg/kg	M	9.91E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.1E-01
	Zinc	6400	mg/kg	6400	mg/kg	M	5.93E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.0E-02
	(Total)												9.0E-01
Dermal	Arsenic	120.96	mg/kg	120.96	mg/kg	M	1.30E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.3E-01
	Cadmium	39.33	mg/kg	39.33	mg/kg	M	1.41E-06	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	5.6E-02
	(Total)												4.9E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.4E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.17 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Undisturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	20.00	ug/L	20.00	ug/L	M	3.29E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E+00
	Cadmium	3.2	ug/L	3.2	ug/L	M	5.31E-05	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	1.1E-01
	Manganese	380	ug/L	380	ug/L	M	6.25E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.3E-01
	Mercury	2.5	ug/L	2.5	ug/L	M	4.11E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.4E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.5E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.18 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Undisturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	20.00	ug/L	20.00	ug/L	M	1.57E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.2E-01
	Cadmium	3.2	ug/L	3.2	ug/L	M	2.54E-05	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	5.1E-02
	Manganese	380	ug/L	380	ug/L	M	2.98E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	6.3E-02
	Mercury	2.5	ug/L	2.5	ug/L	M	1.96E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.5E-02
												Total Hazard Index Across All Exposure Routes/Pathways	7.0E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.19 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Disturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	79.61	ug/L	79.61	ug/L	M	2.62E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.7E-02
	Cadmium	72.9	ug/L	72.9	ug/L	M	2.40E-05	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	4.8E-02
	Manganese	16651	ug/L	16651	ug/L	M	5.47E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.2E-01
	Mercury	4.5	ug/L	4.5	ug/L	M	1.48E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.9E-03
												Total Hazard Index Across All Exposure Routes/Pathways	2.6E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.20 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Disturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	79.61	ug/L	79.61	ug/L	M	7.37E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.5E-02
	Cadmium	72.9	ug/L	72.9	ug/L	M	6.75E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	1.3E-02
	Manganese	16651	ug/L	16651	ug/L	M	1.54E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.3E-02
	Mercury	4.5	ug/L	4.5	ug/L	M	4.17E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.4E-03
												Total Hazard Index Across All Exposure Routes/Pathways	7.2E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.21 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface Water/Sediment
Exposure Medium:	Plant Tissue
Exposure Point:	Water Potatoes from the Lower CDAR
Receptor Population:	Tribal Members
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion													
(With Skin)	Cadmium	0.489	mg/kg	0.489	mg/kg	M	4.01E-03	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	4.0E+00
(Without Skin)	Cadmium	0.291	mg/kg	0.291	mg/kg	M	2.39E-03	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.4E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.22 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Members
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	21.16	mg/kg	21.16	mg/kg	M	4.23E-04	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.1E+00
	Arsenic	124.44	mg/kg	124.44	mg/kg	M	1.49E-03	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.0E+00
	Cadmium	30.45	mg/kg	30.45	mg/kg	M	6.09E-04	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	6.1E-01
	Iron	97440	mg/kg	97440	mg/kg	M	1.95E+00	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	6.5E+00
	Manganese	8960	mg/kg	8960	mg/kg	M	1.79E-01	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.8E+00
	Zinc	4856	mg/kg	4856	mg/kg	M	9.71E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.2E-01
	(Total)												1.7E+01
Dermal	Arsenic	124.44	mg/kg	124.44	mg/kg	M	4.38E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.50E+00
	Cadmium	30.45	mg/kg	30.45	mg/kg	M	3.57E-06	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.40E-01
	(Total)												1.6E+00
												Total Hazard Index Across All Exposure Routes/Pathways	1.9E+01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.23 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Members
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	21.16	mg/kg	21.16	mg/kg	M	1.19E-04	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.0E-01
	Arsenic	124.44	mg/kg	124.44	mg/kg	M	4.21E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.4E+00
	Cadmium	30.45	mg/kg	30.45	mg/kg	M	1.72E-04	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.7E-01
	Iron	97440	mg/kg	97440	mg/kg	M	5.49E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.8E+00
	Manganese	8960	mg/kg	8960	mg/kg	M	5.05E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.1E+00
	Zinc	4856	mg/kg	4856	mg/kg	M	2.74E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.1E-02
	(Total)												4.9E+00
Dermal	Arsenic	124.44	mg/kg	124.44	mg/kg	M	1.35E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.5E-01
	Cadmium	30.45	mg/kg	30.45	mg/kg	M	1.10E-06	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	4.4E-02
	(Total)												4.9E-01
												Total Hazard Index Across All Exposure Routes/Pathways	5.4E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.24 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	25.20	mg/kg	25.20	mg/kg	M	2.90E-04	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	7.2E-01
	Arsenic	120.96	mg/kg	120.96	mg/kg	M	8.35E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.8E+00
	Cadmium	39.33	mg/kg	39.33	mg/kg	M	4.53E-04	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	4.5E-01
	Iron	113073	mg/kg	113073	mg/kg	M	1.30E+00	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.3E+00
	Manganese	10700	mg/kg	10700	mg/kg	M	1.23E-01	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.6E+00
	Zinc	6400	mg/kg	6400	mg/kg	M	7.36E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.5E-01
	(Total)												1.1E+01
Dermal	Arsenic	120.96	mg/kg	120.96	mg/kg	M	7.24E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.4E+00
	Cadmium	39.33	mg/kg	39.33	mg/kg	M	7.84E-06	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.1E-01
	(Total)												2.7E+00
												Total Hazard Index Across All Exposure Routes/Pathways	1.4E+01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.25 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	25.20	mg/kg	25.20	mg/kg	M	8.17E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.0E-01
	Arsenic	120.96	mg/kg	120.96	mg/kg	M	2.35E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.8E-01
	Cadmium	39.33	mg/kg	39.33	mg/kg	M	1.27E-04	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.3E-01
	Iron	113073	mg/kg	113073	mg/kg	M	3.66E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E+00
	Manganese	10700	mg/kg	10700	mg/kg	M	3.47E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	7.4E-01
	Zinc	6400	mg/kg	6400	mg/kg	M	2.07E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	6.9E-02
	(Total)												3.1E+00
Dermal	Arsenic	120.96	mg/kg	120.96	mg/kg	M	4.55E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.5E+00
	Cadmium	39.33	mg/kg	39.33	mg/kg	M	4.93E-06	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.0E-01
	(Total)												1.7E+00
												Total Hazard Index Across All Exposure Routes/Pathways	4.8E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.26 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Undisturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	20.00	ug/L	20.00	ug/L	M	2.00E-03	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.7E+00
	Cadmium	3.2	ug/L	3.2	ug/L	M	3.23E-04	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	6.5E-01
	Manganese	380	ug/L	380	ug/L	M	3.80E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	8.1E-01
	Mercury	2.5	ug/L	2.5	ug/L	M	2.50E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.3E-01
												Total Hazard Index Across All Exposure Routes/Pathways	9.0E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.27 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Undisturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	20.00	ug/L	20.00	ug/L	M	9.55E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.2E+00
	Cadmium	3.2	ug/L	3.2	ug/L	M	1.54E-04	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	3.1E-01
	Manganese	380	ug/L	380	ug/L	M	1.81E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.9E-01
	Mercury	2.5	ug/L	2.5	ug/L	M	1.19E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.0E-01
												Total Hazard Index Across All Exposure Routes/Pathways	4.3E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.28 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Disturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	79.61	ug/L	79.61	ug/L	M	9.16E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.1E-01
	Cadmium	72.9	ug/L	72.9	ug/L	M	8.39E-05	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	1.7E-01
	Manganese	16651	ug/L	16651	ug/L	M	1.92E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	4.1E-01
	Mercury	4.5	ug/L	4.5	ug/L	M	5.18E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	9.0E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.1.29 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Disturbed Surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	79.61	ug/L	79.61	ug/L	M	2.58E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.6E-02
	Cadmium	72.9	ug/L	72.9	ug/L	M	2.36E-05	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	4.7E-02
	Manganese	16651	ug/L	16651	ug/L	M	5.40E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.1E-01
	Mercury	4.5	ug/L	4.5	ug/L	M	1.46E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.9E-03
												Total Hazard Index Across All Exposure Routes/Pathways	2.5E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.1.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	4.06	mg/kg	4.06	mg/kg	M	5.19E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.3E-01
	Arsenic	25.04	mg/kg	25.04	mg/kg	M	1.92E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.4E-01
	Cadmium	3.92	mg/kg	3.92	mg/kg	M	5.01E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	5.0E-02
	Iron	21971	mg/kg	21971	mg/kg	M	2.81E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.4E-01
	Manganese	1085	mg/kg	1085	mg/kg	M	1.39E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.0E-01
	Zinc	719	mg/kg	719	mg/kg	M	9.19E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.1E-02
	(Total)												2.1E+00
Dermal	Arsenic	25.04	mg/kg	25.04	mg/kg	M	2.11E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.0E-02
	Cadmium	3.92	mg/kg	3.92	mg/kg	M	1.10E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	4.4E-03
	(Total)												7.5E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.2E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.1.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	3.05	mg/kg	3.05	mg/kg	M	1.45E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.6E-02
	Arsenic	20.35	mg/kg	20.35	mg/kg	M	5.80E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.9E-01
	Cadmium	3.59	mg/kg	3.59	mg/kg	M	1.70E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.7E-02
	Iron	19198	mg/kg	19198	mg/kg	M	9.12E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.0E-01
	Manganese	783	mg/kg	783	mg/kg	M	3.72E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	7.9E-02
	Zinc	449	mg/kg	449	mg/kg	M	2.13E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.1E-03
	(Total)												6.37E-01
Dermal	Arsenic	20.35	mg/kg	20.35	mg/kg	M	1.28E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.3E-02
	Cadmium	3.59	mg/kg	3.59	mg/kg	M	7.50E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.0E-03
	(Total)												4.6E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.83E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.2.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	4.06	mg/kg	4.06	mg/kg	M	1.48E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.7E-02
	Arsenic	25.04	mg/kg	25.04	mg/kg	M	5.49E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.8E-01
	Cadmium	3.92	mg/kg	3.92	mg/kg	M	1.43E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.4E-02
	Iron	21971	mg/kg	21971	mg/kg	M	8.03E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.7E-01
	Manganese	1085	mg/kg	1085	mg/kg	M	3.96E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	8.4E-02
	Zinc	719	mg/kg	719	mg/kg	M	2.63E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.8E-03
	(Total)												5.9E-01
Dermal	Arsenic	25.04	mg/kg	25.04	mg/kg	M	6.28E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.1E-02
	Cadmium	3.92	mg/kg	3.92	mg/kg	M	3.28E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.3E-03
	(Total)												2.2E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.2.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	3.05	mg/kg	3.05	mg/kg	M	4.43E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.1E-02
	Arsenic	20.35	mg/kg	20.35	mg/kg	M	1.77E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.9E-02
	Cadmium	3.59	mg/kg	3.59	mg/kg	M	5.21E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	5.2E-03
	Iron	19198	mg/kg	19198	mg/kg	M	2.79E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.3E-02
	Manganese	783	mg/kg	783	mg/kg	M	1.14E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.4E-02
	Zinc	449	mg/kg	449	mg/kg	M	6.52E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.2E-03
	(Total)												1.9E-01
Dermal	Arsenic	20.35	mg/kg	20.35	mg/kg	M	4.04E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.3E-02
	Cadmium	3.59	mg/kg	3.59	mg/kg	M	2.38E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	9.5E-04
	(Total)												1.4E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.1E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.3.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.405	ug/L	0.405	ug/L	M	2.59E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.6E-02
												Total Hazard Index Across All Exposure Routes/Pathways	8.6E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.3.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.21	ug/L	0.21	ug/L	M	8.98E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.0E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.0E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.4.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.405	ug/L	0.405	ug/L	M	1.41E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	4.7E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.4.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.21	ug/L	0.21	ug/L	M	4.09E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.4E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.4E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.5.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Pine Creek
Receptor Population:	Neighboorhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	12.83	mg/kg	12.83	mg/kg	M	7.91E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.0E-02
	Arsenic	22.35	mg/kg	22.35	mg/kg	M	8.27E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.8E-02
	Cadmium	0.92	mg/kg	0.92	mg/kg	M	5.67E-07	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	5.7E-04
	Iron	16300	mg/kg	16300	mg/kg	M	1.00E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.3E-02
	Manganese	451.62	mg/kg	451.62	mg/kg	M	2.78E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	5.9E-03
	Zinc	320.31	mg/kg	320.31	mg/kg	M	1.97E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	6.6E-04
	(Total)												8.8E-02
Dermal	Arsenic	22.35	mg/kg	22.35	mg/kg	M	6.40E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.1E-02
	Cadmium	0.92	mg/kg	0.92	mg/kg	M	8.78E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.5E-04
	(Total)												2.2E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.1E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.5.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Pine Creek
Receptor Population:	Neighboorhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	7.05	mg/kg	7.05	mg/kg	M	8.28E-07	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.1E-03
	Arsenic	15.84	mg/kg	15.84	mg/kg	M	1.12E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.7E-03
	Cadmium	0.82	mg/kg	0.82	mg/kg	M	9.63E-08	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	9.6E-05
	Iron	14622	mg/kg	14622	mg/kg	M	1.72E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.7E-03
	Manganese	392.21	mg/kg	392.21	mg/kg	M	4.61E-05	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	9.8E-04
	Zinc	285.89	mg/kg	285.89	mg/kg	M	3.36E-05	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.1E-04
	(Total)												1.3E-02
Dermal	Arsenic	15.84	mg/kg	15.84	mg/kg	M	2.27E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.6E-03
	Cadmium	0.82	mg/kg	0.82	mg/kg	M	3.91E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.6E-04
	(Total)												7.7E-03
												Total Hazard Index Across All Exposure Routes/Pathways	2.0E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.6.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	NS Confluence
Receptor Population:	Neighboorhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	39.78	mg/kg	39.78	mg/kg	M	2.45E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	6.1E-02
	Arsenic	163.2	mg/kg	163.2	mg/kg	M	6.04E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.0E-01
	Cadmium	26.58	mg/kg	26.58	mg/kg	M	1.64E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.6E-02
	Iron	100621	mg/kg	100621	mg/kg	M	6.20E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.1E-01
	Manganese	8585	mg/kg	8585	mg/kg	M	5.29E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.1E-01
	Zinc	3744	mg/kg	3744	mg/kg	M	2.31E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.7E-03
	(Total)												6.1E-01
Dermal	Arsenic	163.2	mg/kg	163.2	mg/kg	M	7.32E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.4E-01
	Cadmium	26.58	mg/kg	26.58	mg/kg	M	3.97E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.6E-02
	(Total)												2.6E-01
												Total Hazard Index Across All Exposure Routes/Pathways	8.7E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.6.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	NS Confluence
Receptor Population:	Neighboorhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	34.47	mg/kg	34.47	mg/kg	M	4.05E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.0E-02
	Arsenic	145.44	mg/kg	145.44	mg/kg	M	1.02E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.4E-02
	Cadmium	22.03	mg/kg	22.03	mg/kg	M	2.59E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.6E-03
	Iron	89354	mg/kg	89354	mg/kg	M	1.05E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.5E-02
	Manganese	7467	mg/kg	7467	mg/kg	M	8.77E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.9E-02
	Zinc	3286	mg/kg	3286	mg/kg	M	3.86E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.3E-03
	(Total)												1.0E-01
Dermal	Arsenic	145.44	mg/kg	145.44	mg/kg	M	3.26E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-01
	Cadmium	22.03	mg/kg	22.03	mg/kg	M	1.65E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	6.6E-03
	(Total)												1.2E-01
												Total Hazard Index Across All Exposure Routes/Pathways	2.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.7.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Pine Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.73	ug/L	0.73	ug/L	M	2.06E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.9E-04
	Cadmium	1.08	ug/L	1.08	ug/L	M	3.04E-07	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	6.1E-04
	Manganese	38.46	ug/L	38.46	ug/L	M	1.08E-05	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.3E-04
	Mercury	0.09	ug/L	0.09	ug/L	M	2.54E-08	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.5E-05
												Total Hazard Index Across All Exposure Routes/Pathways	1.6E-03

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.7.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Pine Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.57	ug/L	0.57	ug/L	M	8.03E-08	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.7E-04
	Cadmium	0.58	ug/L	0.58	ug/L	M	8.17E-08	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	1.6E-04
	Manganese	16.26	ug/L	16.26	ug/L	M	2.29E-06	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	4.9E-05
	Mercury	0.08	ug/L	0.08	ug/L	M	1.13E-08	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.8E-05
												Total Hazard Index Across All Exposure Routes/Pathways	5.2E-04

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.8.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	NS Confluence
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	134	ug/L	134	ug/L	M	3.78E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.3E-01
	Cadmium	83.8	ug/L	83.8	ug/L	M	2.36E-05	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	4.7E-02
	Manganese	9470	ug/L	9470	ug/L	M	2.67E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	5.7E-02
	Mercury	4.6	ug/L	4.6	ug/L	M	1.30E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.3E-03
												Total Hazard Index Across All Exposure Routes/Pathways	2.3E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.8.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	NS Confluence
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	36.4	ug/L	36.4	ug/L	M	5.13E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.7E-02
	Cadmium	29.5	ug/L	29.5	ug/L	M	4.16E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	8.3E-03
	Manganese	3145	ug/L	3145	ug/L	M	4.43E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	9.4E-03
	Mercury	1.3	ug/L	1.3	ug/L	M	1.83E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.1E-04
												Total Hazard Index Across All Exposure Routes/Pathways	3.5E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.9.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	NS Confluence
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	39.78	mg/kg	39.78	mg/kg	M	6.98E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.7E-01
	Arsenic	163.2	mg/kg	163.2	mg/kg	M	1.72E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.7E-01
	Cadmium	26.58	mg/kg	26.58	mg/kg	M	4.66E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	4.7E-02
	Iron	100621	mg/kg	100621	mg/kg	M	1.76E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.9E-01
	Manganese	8585	mg/kg	8585	mg/kg	M	1.51E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.2E-01
	Zinc	3744	mg/kg	3744	mg/kg	M	6.56E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.2E-02
	(Total)												1.7E+00
Dermal	Arsenic	163.2	mg/kg	163.2	mg/kg	M	3.72E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.2E-01
	Cadmium	26.58	mg/kg	26.58	mg/kg	M	2.02E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	8.1E-03
	(Total)												1.3E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.9E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.9.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	NS Confluence
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	34.47	mg/kg	34.47	mg/kg	M	1.21E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.0E-02
	Arsenic	145.44	mg/kg	145.44	mg/kg	M	3.06E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.0E-01
	Cadmium	22.03	mg/kg	22.03	mg/kg	M	7.73E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	7.7E-03
	Iron	89354	mg/kg	89354	mg/kg	M	3.13E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.0E-01
	Manganese	7467	mg/kg	7467	mg/kg	M	2.62E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	5.6E-02
	Zinc	3286	mg/kg	3286	mg/kg	M	1.15E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.8E-03
	(Total)												3.0E-01
Dermal	Arsenic	145.44	mg/kg	145.44	mg/kg	M	1.66E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.5E-02
	Cadmium	22.03	mg/kg	22.03	mg/kg	M	8.37E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.3E-03
	(Total)												5.9E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.6E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.10.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	NS Confluence
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	39.78	mg/kg	39.78	mg/kg	M	1.79E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	4.5E-02
	Arsenic	163.2	mg/kg	163.2	mg/kg	M	4.42E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.5E-01
	Cadmium	26.58	mg/kg	26.58	mg/kg	M	1.20E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.2E-02
	Iron	100621	mg/kg	100621	mg/kg	M	4.54E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.5E-01
	Manganese	8585	mg/kg	8585	mg/kg	M	3.87E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	8.2E-02
	Zinc	3744	mg/kg	3744	mg/kg	M	1.69E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.6E-03
	(Total)												4.4E-01
Dermal	Arsenic	163.2	mg/kg	163.2	mg/kg	M	1.63E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.4E-01
	Cadmium	26.58	mg/kg	26.58	mg/kg	M	8.83E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.5E-03
	(Total)												5.8E-02
												Total Hazard Index Across All Exposure Routes/Pathways	5.0E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.10.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	NS Confluence
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	34.47	mg/kg	34.47	mg/kg	M	3.53E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	8.8E-03
	Arsenic	145.44	mg/kg	145.44	mg/kg	M	8.93E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.0E-02
	Cadmium	22.03	mg/kg	22.03	mg/kg	M	2.25E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.3E-03
	Iron	89354	mg/kg	89354	mg/kg	M	9.14E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.0E-02
	Manganese	7467	mg/kg	7467	mg/kg	M	7.64E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.6E-02
	Zinc	3286	mg/kg	3286	mg/kg	M	3.36E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.1E-03
	(Total)												8.9E-02
Dermal	Arsenic	145.44	mg/kg	145.44	mg/kg	M	7.51E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.5E-02
	Cadmium	22.03	mg/kg	22.03	mg/kg	M	3.79E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.5E-03
	(Total)												2.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.11.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	NS Confluence
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	134	ug/L	134	ug/L	M	2.35E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.8E-02
	Cadmium	83.8	ug/L	83.8	ug/L	M	1.47E-05	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	2.9E-02
	Manganese	9470	ug/L	9470	ug/L	M	1.66E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.5E-02
	Mercury	4.6	ug/L	4.6	ug/L	M	8.07E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.7E-03
												Total Hazard Index Across All Exposure Routes/Pathways	1.5E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.11.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	NS Confluence
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	36.4	ug/L	36.4	ug/L	M	3.19E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-02
	Cadmium	29.5	ug/L	29.5	ug/L	M	2.59E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	5.2E-03
	Manganese	3145	ug/L	3145	ug/L	M	2.76E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	5.9E-03
	Mercury	1.3	ug/L	1.3	ug/L	M	1.14E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.8E-04
												Total Hazard Index Across All Exposure Routes/Pathways	2.2E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.12.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	NS Confluence
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	134	ug/L	134	ug/L	M	8.73E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.9E-02
	Cadmium	83.8	ug/L	83.8	ug/L	M	5.46E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	1.1E-02
	Manganese	9470	ug/L	9470	ug/L	M	6.17E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.3E-02
	Mercury	4.6	ug/L	4.6	ug/L	M	3.00E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.0E-03
												Total Hazard Index Across All Exposure Routes/Pathways	5.4E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.12.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	NS Confluence
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	36.4	ug/L	36.4	ug/L	M	1.24E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.1E-03
	Cadmium	29.5	ug/L	29.5	ug/L	M	1.01E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	2.0E-03
	Manganese	3145	ug/L	3145	ug/L	M	1.07E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.3E-03
	Mercury	1.3	ug/L	1.3	ug/L	M	4.43E-08	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.5E-04
												Total Hazard Index Across All Exposure Routes/Pathways	8.6E-03

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.13.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Kingston - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	6.86	mg/kg	6.86	mg/kg	M	1.57E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.9E-02
	Arsenic	34.6	mg/kg	34.6	mg/kg	M	4.75E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.6E-01
	Cadmium	5.1	mg/kg	5.1	mg/kg	M	1.17E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.2E-02
	Iron	29003	mg/kg	29003	mg/kg	M	6.64E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.2E-01
	Manganese	1693	mg/kg	1693	mg/kg	M	3.88E-03	mg/kg-d	1.4E-01	mg/kg-d	N/A	N/A	2.8E-02
	Zinc	1057	mg/kg	1057	mg/kg	M	2.42E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.1E-03
	(Total)												4.7E-01
Dermal	Arsenic	34.6	mg/kg	34.6	mg/kg	M	1.98E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.6E-03
	Cadmium	5.1	mg/kg	5.1	mg/kg	M	9.73E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.9E-04
	(Total)												7.0E-03
												Total Hazard Index Across All Exposure Routes/Pathways	4.7E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.2.13.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Kingston - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	5.51	mg/kg	5.51	mg/kg	M	1.85E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	4.6E-03
	Arsenic	29.06	mg/kg	29.06	mg/kg	M	5.87E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.0E-02
	Cadmium	5.14	mg/kg	5.14	mg/kg	M	1.73E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.7E-03
	Iron	25811	mg/kg	25811	mg/kg	M	8.69E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.9E-02
	Manganese	1377	mg/kg	1377	mg/kg	M	4.63E-04	mg/kg-d	1.4E-01	mg/kg-d	N/A	N/A	3.3E-03
	Zinc	794	mg/kg	794	mg/kg	M	2.67E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.9E-04
	(Total)												5.9E-02
Dermal	Arsenic	29.06	mg/kg	29.06	mg/kg	M	3.67E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.2E-03
	Cadmium	5.14	mg/kg	5.14	mg/kg	M	2.16E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	8.7E-05
	(Total)												1.3E-03
												Total Hazard Index Across All Exposure Routes/Pathways	6.0E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.1.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	15.94	mg/kg	15.94	mg/kg	M	2.04E-04	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	5.1E-01
	Arsenic	50.74	mg/kg	50.74	mg/kg	M	3.89E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.3E+00
	Cadmium	9.62	mg/kg	9.62	mg/kg	M	1.23E-04	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.2E-01
	Iron	27190	mg/kg	27190	mg/kg	M	3.48E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E+00
	Manganese	1367	mg/kg	1367	mg/kg	M	1.75E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.7E-01
	Zinc	1320	mg/kg	1320	mg/kg	M	1.69E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.6E-02
	(Total)												3.5E+00
Dermal	Arsenic	50.74	mg/kg	50.74	mg/kg	M	4.28E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.4E-01
	Cadmium	9.62	mg/kg	9.62	mg/kg	M	2.71E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.1E-02
	(Total)												1.5E-01
												Total Hazard Index Across All Exposure Routes/Pathways	3.7E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.1.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	10.26	mg/kg	10.26	mg/kg	M	4.87E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.2E-01
	Arsenic	39.66	mg/kg	39.66	mg/kg	M	1.13E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.8E-01
	Cadmium	6.95	mg/kg	6.95	mg/kg	M	3.30E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.3E-02
	Iron	24797	mg/kg	24797	mg/kg	M	1.18E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.9E-01
	Manganese	1168	mg/kg	1168	mg/kg	M	5.55E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.2E-01
	Zinc	844	mg/kg	844	mg/kg	M	4.01E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.3E-02
	(Total)												1.1E+00
Dermal	Arsenic	39.66	mg/kg	39.66	mg/kg	M	2.49E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.3E-02
	Cadmium	6.95	mg/kg	6.95	mg/kg	M	1.45E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	5.8E-03
	(Total)												8.9E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.1E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.2.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	15.94	mg/kg	15.94	mg/kg	M	5.82E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.5E-01
	Arsenic	50.74	mg/kg	50.74	mg/kg	M	1.11E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.7E-01
	Cadmium	9.62	mg/kg	9.62	mg/kg	M	3.51E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.5E-02
	Iron	27190	mg/kg	27190	mg/kg	M	9.93E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.3E-01
	Manganese	1367	mg/kg	1367	mg/kg	M	4.99E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.1E-01
	Zinc	1320	mg/kg	1320	mg/kg	M	4.82E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.6E-02
	(Total)												1.0E+00
Dermal	Arsenic	50.74	mg/kg	50.74	mg/kg	M	1.27E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.2E-02
	Cadmium	9.62	mg/kg	9.62	mg/kg	M	8.05E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.2E-03
	(Total)												4.6E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.1E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.2.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	10.26	mg/kg	10.26	mg/kg	M	1.49E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.7E-02
	Arsenic	39.66	mg/kg	39.66	mg/kg	M	3.45E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.2E-01
	Cadmium	6.95	mg/kg	6.95	mg/kg	M	1.01E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.0E-02
	Iron	24797	mg/kg	24797	mg/kg	M	3.60E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E-01
	Manganese	1168	mg/kg	1168	mg/kg	M	1.69E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.6E-02
	Zinc	844	mg/kg	844	mg/kg	M	1.22E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.1E-03
	(Total)												3.2E-01
Dermal	Arsenic	39.66	mg/kg	39.66	mg/kg	M	7.88E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.6E-02
	Cadmium	6.95	mg/kg	6.95	mg/kg	M	4.60E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.8E-03
	(Total)												2.8E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.5E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.3.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	8.4	ug/L	8.4	ug/L	M	5.37E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.8E+00
												Total Hazard Index Across All Exposure Routes/Pathways	1.8E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.3.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	1.92	ug/L	1.92	ug/L	M	8.21E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.7E-01
												Total Hazard Index Across All Exposure Routes/Pathways	2.7E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.4.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	8.4	ug/L	8.4	ug/L	M	2.92E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	9.7E-01
												Total Hazard Index Across All Exposure Routes/Pathways	9.7E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.4.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	1.92	ug/L	1.92	ug/L	M	3.74E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.2E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.5.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Elk Creek Area
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	54.9	mg/kg	54.9	mg/kg	M	5.48E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.4E-01
	Arsenic	98.5	mg/kg	98.5	mg/kg	M	5.90E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.0E-01
	Cadmium	64.5	mg/kg	64.5	mg/kg	M	6.44E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	6.4E-02
	Iron	99928	mg/kg	99928	mg/kg	M	9.97E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.3E-01
	Manganese	9280	mg/kg	9280	mg/kg	M	9.26E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.0E-01
	Zinc	6130	mg/kg	6130	mg/kg	M	6.12E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.0E-02
	(Total)												9.5E-01
Dermal	Arsenic	98.5	mg/kg	98.5	mg/kg	M	2.00E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.7E-02
	Cadmium	64.5	mg/kg	64.5	mg/kg	M	4.36E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.7E-02
	(Total)												8.4E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.0E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.5.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Elk Creek Area
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	46.22	mg/kg	46.22	mg/kg	M	9.23E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.3E-02
	Arsenic	79.96	mg/kg	79.96	mg/kg	M	9.58E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.2E-02
	Cadmium	21.8	mg/kg	21.8	mg/kg	M	4.35E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	4.4E-03
	Iron	85590	mg/kg	85590	mg/kg	M	1.71E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.7E-02
	Manganese	6786	mg/kg	6786	mg/kg	M	1.35E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.9E-02
	Zinc	3328	mg/kg	3328	mg/kg	M	6.64E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.2E-03
	(Total)												1.5E-01
Dermal	Arsenic	79.96	mg/kg	79.96	mg/kg	M	8.11E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.7E-02
	Cadmium	21.8	mg/kg	21.8	mg/kg	M	7.37E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.9E-03
	(Total)												3.0E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.8E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.6.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Elk Creek Pond
Receptor Population:	Neighboorhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	27.84	mg/kg	27.84	mg/kg	M	1.72E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	4.3E-02
	Arsenic	77.74	mg/kg	77.74	mg/kg	M	2.88E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	9.6E-02
	Cadmium	38.38	mg/kg	38.38	mg/kg	M	2.37E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.4E-02
	Iron	35970	mg/kg	35970	mg/kg	M	2.22E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.4E-02
	Manganese	2223	mg/kg	2223	mg/kg	M	1.37E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.9E-02
	Zinc	5031	mg/kg	5031	mg/kg	M	3.10E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.0E-02
	(Total)												2.8E-01
Dermal	Arsenic	77.74	mg/kg	77.74	mg/kg	M	2.23E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.4E-02
	Cadmium	38.38	mg/kg	38.38	mg/kg	M	3.66E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.5E-02
	(Total)												8.9E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.6E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.6.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Elk Creek Pond
Receptor Population:	Neighboorhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	17.68	mg/kg	17.68	mg/kg	M	2.08E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	5.2E-03
	Arsenic	63.76	mg/kg	63.76	mg/kg	M	4.49E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.5E-02
	Cadmium	42.77	mg/kg	42.77	mg/kg	M	5.02E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	5.0E-03
	Iron	29423	mg/kg	29423	mg/kg	M	3.45E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E-02
	Manganese	1486	mg/kg	1486	mg/kg	M	1.74E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.7E-03
	Zinc	4222	mg/kg	4222	mg/kg	M	4.96E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.7E-03
	(Total)												4.2E-02
Dermal	Arsenic	63.76	mg/kg	63.76	mg/kg	M	9.13E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.0E-02
	Cadmium	42.77	mg/kg	42.77	mg/kg	M	2.04E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	8.2E-03
	(Total)												3.9E-02
												Total Hazard Index Across All Exposure Routes/Pathways	8.1E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.7.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Elk Creek Pond
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	32	ug/L	32	ug/L	M	9.02E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.0E-02
	Cadmium	60.3	ug/L	60.3	ug/L	M	1.70E-05	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	3.4E-02
	Manganese	8570	ug/L	8570	ug/L	M	2.42E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	5.1E-02
	Mercury	3.5	ug/L	3.5	ug/L	M	9.86E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.3E-03
												Total Hazard Index Across All Exposure Routes/Pathways	1.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.3.7.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Elk Creek Pond
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	16.64	ug/L	16.64	ug/L	M	2.34E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.8E-03
	Cadmium	26.44	ug/L	26.44	ug/L	M	3.73E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	7.5E-03
	Manganese	2297	ug/L	2297	ug/L	M	3.24E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	6.9E-03
	Mercury	0.85	ug/L	0.85	ug/L	M	1.20E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.0E-04
												Total Hazard Index Across All Exposure Routes/Pathways	2.3E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.4.1.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	12.54	mg/kg	12.54	mg/kg	M	1.60E-04	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	4.0E-01
	Arsenic	46.74	mg/kg	46.74	mg/kg	M	3.59E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.2E+00
	Cadmium	5.25	mg/kg	5.25	mg/kg	M	6.71E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	6.7E-02
	Iron	22488	mg/kg	22488	mg/kg	M	2.88E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.6E-01
	Manganese	1199	mg/kg	1199	mg/kg	M	1.53E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.3E-01
	Zinc	597	mg/kg	597	mg/kg	M	7.63E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.5E-02
	(Total)												3.0E+00
Dermal	Arsenic	46.74	mg/kg	46.74	mg/kg	M	3.94E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.3E-01
	Cadmium	5.25	mg/kg	5.25	mg/kg	M	1.48E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	5.9E-03
	(Total)												1.4E-01
												Total Hazard Index Across All Exposure Routes/Pathways	3.1E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.4.1.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	10.06	mg/kg	10.06	mg/kg	M	4.78E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.2E-01
	Arsenic	38.95	mg/kg	38.95	mg/kg	M	1.11E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.7E-01
	Cadmium	4.81	mg/kg	4.81	mg/kg	M	2.28E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.3E-02
	Iron	21454	mg/kg	21454	mg/kg	M	1.02E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.4E-01
	Manganese	1122	mg/kg	1122	mg/kg	M	5.33E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.1E-01
	Zinc	523	mg/kg	523	mg/kg	M	2.48E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.3E-03
	(Total)												9.7E-01
Dermal	Arsenic	38.95	mg/kg	38.95	mg/kg	M	2.44E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.1E-02
	Cadmium	4.81	mg/kg	4.81	mg/kg	M	1.01E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	4.0E-03
	(Total)												8.5E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.1E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.4.2.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	12.54	mg/kg	12.54	mg/kg	M	4.58E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.1E-01
	Arsenic	46.74	mg/kg	46.74	mg/kg	M	1.02E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.4E-01
	Cadmium	5.25	mg/kg	5.25	mg/kg	M	1.92E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.9E-02
	Iron	22488	mg/kg	22488	mg/kg	M	8.21E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.7E-01
	Manganese	1199	mg/kg	1199	mg/kg	M	4.38E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	9.3E-02
	Zinc	597	mg/kg	597	mg/kg	M	2.18E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.3E-03
	(Total)												8.5E-01
Dermal	Arsenic	46.74	mg/kg	46.74	mg/kg	M	1.17E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.9E-02
	Cadmium	5.25	mg/kg	5.25	mg/kg	M	4.39E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.8E-03
	(Total)												4.1E-02
												Total Hazard Index Across All Exposure Routes/Pathways	8.9E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.4.2.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	10.06	mg/kg	10.06	mg/kg	M	1.46E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.6E-02
	Arsenic	38.95	mg/kg	38.95	mg/kg	M	3.39E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-01
	Cadmium	4.81	mg/kg	4.81	mg/kg	M	6.98E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	7.0E-03
	Iron	21454	mg/kg	21454	mg/kg	M	3.11E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.0E-01
	Manganese	1122	mg/kg	1122	mg/kg	M	1.63E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.5E-02
	Zinc	523	mg/kg	523	mg/kg	M	7.59E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.5E-03
	(Total)												3.0E-01
Dermal	Arsenic	38.95	mg/kg	38.95	mg/kg	M	7.74E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.6E-02
	Cadmium	4.81	mg/kg	4.81	mg/kg	M	3.19E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.3E-03
	(Total)												2.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.4.3.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.28	ug/L	0.28	ug/L	M	1.79E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.0E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.0E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.4.3.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.23	ug/L	0.23	ug/L	M	9.83E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.3E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.3E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.4.4.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.28	ug/L	0.28	ug/L	M	9.72E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.2E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.2E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.4.4.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.23	ug/L	0.23	ug/L	M	4.48E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.5E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.5E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.1.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	8.9	mg/kg	8.9	mg/kg	M	1.14E-04	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.8E-01
	Arsenic	21.46	mg/kg	21.46	mg/kg	M	1.65E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.5E-01
	Cadmium	5.44	mg/kg	5.44	mg/kg	M	6.96E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	7.0E-02
	Iron	20198	mg/kg	20198	mg/kg	M	2.58E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.6E-01
	Manganese	1068	mg/kg	1068	mg/kg	M	1.37E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.9E-01
	Zinc	816	mg/kg	816	mg/kg	M	1.04E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.5E-02
	(Total)												2.1E+00
Dermal	Arsenic	21.46	mg/kg	21.46	mg/kg	M	1.81E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.0E-02
	Cadmium	5.44	mg/kg	5.44	mg/kg	M	1.53E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	6.1E-03
	(Total)												6.6E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.2E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.1.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	4.89	mg/kg	4.89	mg/kg	M	2.32E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	5.8E-02
	Arsenic	16.52	mg/kg	16.52	mg/kg	M	4.71E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.6E-01
	Cadmium	4.41	mg/kg	4.41	mg/kg	M	2.09E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.1E-02
	Iron	18361	mg/kg	18361	mg/kg	M	8.72E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.9E-01
	Manganese	928	mg/kg	928	mg/kg	M	4.41E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	9.4E-02
	Zinc	561	mg/kg	561	mg/kg	M	2.66E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.9E-03
	(Total)												6.3E-01
Dermal	Arsenic	16.52	mg/kg	16.52	mg/kg	M	1.04E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.5E-02
	Cadmium	4.41	mg/kg	4.41	mg/kg	M	9.21E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.7E-03
	(Total)												3.8E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.7E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.2.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	8.9	mg/kg	8.9	mg/kg	M	3.25E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	8.1E-02
	Arsenic	21.46	mg/kg	21.46	mg/kg	M	4.70E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.6E-01
	Cadmium	5.44	mg/kg	5.44	mg/kg	M	1.99E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.0E-02
	Iron	20198	mg/kg	20198	mg/kg	M	7.38E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.5E-01
	Manganese	1068	mg/kg	1068	mg/kg	M	3.90E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	8.3E-02
	Zinc	816	mg/kg	816	mg/kg	M	2.98E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.9E-03
	(Total)												6.0E-01
Dermal	Arsenic	21.46	mg/kg	21.46	mg/kg	M	5.39E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.8E-02
	Cadmium	5.44	mg/kg	5.44	mg/kg	M	4.55E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.8E-03
	(Total)												2.0E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.2.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	4.89	mg/kg	4.89	mg/kg	M	7.10E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.8E-02
	Arsenic	16.52	mg/kg	16.52	mg/kg	M	1.44E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.8E-02
	Cadmium	4.41	mg/kg	4.41	mg/kg	M	6.40E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	6.4E-03
	Iron	18361	mg/kg	18361	mg/kg	M	2.66E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.9E-02
	Manganese	928	mg/kg	928	mg/kg	M	1.35E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.9E-02
	Zinc	561	mg/kg	561	mg/kg	M	8.14E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.7E-03
	(Total)												1.9E-01
Dermal	Arsenic	16.52	mg/kg	16.52	mg/kg	M	3.28E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-02
	Cadmium	4.41	mg/kg	4.41	mg/kg	M	2.92E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.2E-03
	(Total)												1.2E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.0E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.3.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.545	ug/L	0.545	ug/L	M	3.48E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.2E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.3.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.38	ug/L	0.38	ug/L	M	1.62E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.4E-02
												Total Hazard Index Across All Exposure Routes/Pathways	5.4E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.4.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.545	ug/L	0.545	ug/L	M	1.89E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.3E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.3E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.4.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.38	ug/L	0.38	ug/L	M	7.40E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.5E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.5E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.5.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Silverton - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	6.94	mg/kg	6.94	mg/kg	M	6.93E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.7E-02
	Arsenic	28.44	mg/kg	28.44	mg/kg	M	1.70E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.7E-02
	Cadmium	4.57	mg/kg	4.57	mg/kg	M	4.56E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	4.6E-03
	Iron	20025	mg/kg	20025	mg/kg	M	2.00E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	6.7E-02
	Manganese	1090	mg/kg	1090	mg/kg	M	1.09E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.3E-02
	Zinc	739	mg/kg	739	mg/kg	M	7.38E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.5E-03
	(Total)												1.7E-01
Dermal	Arsenic	28.44	mg/kg	28.44	mg/kg	M	5.77E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.9E-02
	Cadmium	4.57	mg/kg	4.57	mg/kg	M	3.09E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.2E-03
	(Total)												2.0E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.9E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.5.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Silverton - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	4.28	mg/kg	4.28	mg/kg	M	8.54E-07	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.1E-03
	Arsenic	16.88	mg/kg	16.88	mg/kg	M	2.02E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.7E-03
	Cadmium	5.1	mg/kg	5.1	mg/kg	M	1.02E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.0E-03
	Iron	19535	mg/kg	19535	mg/kg	M	3.90E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.3E-02
	Manganese	1035	mg/kg	1035	mg/kg	M	2.07E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	4.4E-03
	Zinc	537.23	mg/kg	537.23	mg/kg	M	1.07E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.6E-04
	(Total)												2.8E-02
Dermal	Arsenic	16.88	mg/kg	16.88	mg/kg	M	1.71E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.7E-03
	Cadmium	5.1	mg/kg	5.1	mg/kg	M	1.72E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	6.9E-04
	(Total)												6.4E-03
												Total Hazard Index Across All Exposure Routes/Pathways	3.4E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.6.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Silverton - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	6.94	mg/kg	6.94	mg/kg	M	1.29E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.2E-02
	Arsenic	28.44	mg/kg	28.44	mg/kg	M	3.18E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-01
	Cadmium	4.57	mg/kg	4.57	mg/kg	M	8.51E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	8.5E-03
	Iron	20025	mg/kg	20025	mg/kg	M	3.73E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E-01
	Manganese	1090	mg/kg	1090	mg/kg	M	2.03E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	4.3E-02
	Zinc	739	mg/kg	739	mg/kg	M	1.38E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.6E-03
	(Total)												3.2E-01
Dermal	Arsenic	28.44	mg/kg	28.44	mg/kg	M	4.66E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.6E-02
	Cadmium	4.57	mg/kg	4.57	mg/kg	M	2.50E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.0E-03
	(Total)												1.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.4E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.6.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Silverton - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	4.28	mg/kg	4.28	mg/kg	M	1.59E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	4.0E-03
	Arsenic	16.88	mg/kg	16.88	mg/kg	M	3.77E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.3E-02
	Cadmium	5.1	mg/kg	5.1	mg/kg	M	1.90E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.9E-03
	Iron	19535	mg/kg	19535	mg/kg	M	7.28E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.4E-02
	Manganese	1035	mg/kg	1035	mg/kg	M	3.86E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	8.2E-03
	Zinc	537.23	mg/kg	537.23	mg/kg	M	2.00E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	6.7E-04
	(Total)												5.2E-02
Dermal	Arsenic	16.88	mg/kg	16.88	mg/kg	M	1.38E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.6E-03
	Cadmium	5.1	mg/kg	5.1	mg/kg	M	1.39E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	5.6E-04
	(Total)												5.2E-03
												Total Hazard Index Across All Exposure Routes/Pathways	5.7E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.7.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Silverton - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	6.94	mg/kg	6.94	mg/kg	M	3.24E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	8.1E-03
	Arsenic	28.44	mg/kg	28.44	mg/kg	M	7.96E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.7E-02
	Cadmium	4.57	mg/kg	4.57	mg/kg	M	2.13E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.1E-03
	Iron	20025	mg/kg	20025	mg/kg	M	9.34E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.1E-02
	Manganese	1090	mg/kg	1090	mg/kg	M	5.09E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.1E-02
	Zinc	739	mg/kg	739	mg/kg	M	3.45E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.1E-03
	(Total)												8.0E-02
Dermal	Arsenic	28.44	mg/kg	28.44	mg/kg	M	1.39E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.6E-03
	Cadmium	4.57	mg/kg	4.57	mg/kg	M	7.43E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.0E-04
	(Total)												4.9E-03
												Total Hazard Index Across All Exposure Routes/Pathways	8.5E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.5.7.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Silverton - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	4.28	mg/kg	4.28	mg/kg	M	4.52E-07	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.1E-03
	Arsenic	16.88	mg/kg	16.88	mg/kg	M	1.07E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.6E-03
	Cadmium	5.1	mg/kg	5.1	mg/kg	M	5.39E-07	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	5.4E-04
	Iron	19535	mg/kg	19535	mg/kg	M	2.06E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	6.9E-03
	Manganese	1035	mg/kg	1035	mg/kg	M	1.09E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.3E-03
	Zinc	537.23	mg/kg	537.23	mg/kg	M	5.67E-05	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.9E-04
	(Total)												1.5E-02
Dermal	Arsenic	16.88	mg/kg	16.88	mg/kg	M	4.39E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.5E-03
	Cadmium	5.1	mg/kg	5.1	mg/kg	M	4.42E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.8E-04
	(Total)												1.6E-03
												Total Hazard Index Across All Exposure Routes/Pathways	1.6E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.1.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	13.7	mg/kg	13.7	mg/kg	M	1.75E-04	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	4.4E-01
	Arsenic	22.57	mg/kg	22.57	mg/kg	M	1.73E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.8E-01
	Cadmium	7.63	mg/kg	7.63	mg/kg	M	9.76E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	9.8E-02
	Iron	22240	mg/kg	22240	mg/kg	M	2.84E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.5E-01
	Manganese	989	mg/kg	989	mg/kg	M	1.26E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.7E-01
	Zinc	967	mg/kg	967	mg/kg	M	1.24E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.1E-02
	(Total)												2.4E+00
Dermal	Arsenic	22.57	mg/kg	22.57	mg/kg	M	1.90E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.3E-02
	Cadmium	7.63	mg/kg	7.63	mg/kg	M	2.15E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	8.6E-03
	(Total)												7.2E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.4E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.1.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	8.81	mg/kg	8.81	mg/kg	M	4.18E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.0E-01
	Arsenic	20.32	mg/kg	20.32	mg/kg	M	5.79E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.9E-01
	Cadmium	6.46	mg/kg	6.46	mg/kg	M	3.07E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.1E-02
	Iron	21149	mg/kg	21149	mg/kg	M	1.00E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.3E-01
	Manganese	895	mg/kg	895	mg/kg	M	4.25E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	9.0E-02
	Zinc	860	mg/kg	860	mg/kg	M	4.08E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.4E-02
	(Total)												7.7E-01
Dermal	Arsenic	20.32	mg/kg	20.32	mg/kg	M	1.27E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.2E-02
	Cadmium	6.46	mg/kg	6.46	mg/kg	M	1.35E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	5.4E-03
	(Total)												4.8E-02
												Total Hazard Index Across All Exposure Routes/Pathways	8.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.2.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	13.7	mg/kg	13.7	mg/kg	M	5.00E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.3E-01
	Arsenic	22.57	mg/kg	22.57	mg/kg	M	4.95E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.6E-01
	Cadmium	7.63	mg/kg	7.63	mg/kg	M	2.79E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.8E-02
	Iron	22240	mg/kg	22240	mg/kg	M	8.12E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.7E-01
	Manganese	989	mg/kg	989	mg/kg	M	3.61E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	7.7E-02
	Zinc	967	mg/kg	967	mg/kg	M	3.53E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E-02
	(Total)												6.8E-01
Dermal	Arsenic	22.57	mg/kg	22.57	mg/kg	M	5.66E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.9E-02
	Cadmium	7.63	mg/kg	7.63	mg/kg	M	6.38E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.6E-03
	(Total)												2.1E-02
												Total Hazard Index Across All Exposure Routes/Pathways	7.0E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.2.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	8.81	mg/kg	8.81	mg/kg	M	1.28E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.2E-02
	Arsenic	20.32	mg/kg	20.32	mg/kg	M	1.77E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.9E-02
	Cadmium	6.46	mg/kg	6.46	mg/kg	M	9.37E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	9.4E-03
	Iron	21149	mg/kg	21149	mg/kg	M	3.07E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.0E-01
	Manganese	895	mg/kg	895	mg/kg	M	1.30E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.8E-02
	Zinc	860	mg/kg	860	mg/kg	M	1.25E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.2E-03
	(Total)												2.3E-01
Dermal	Arsenic	20.32	mg/kg	20.32	mg/kg	M	4.04E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.3E-02
	Cadmium	6.46	mg/kg	6.46	mg/kg	M	4.28E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.7E-03
	(Total)												1.5E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.5E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.3.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.49	ug/L	0.49	ug/L	M	3.16E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.1E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.3.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.42	ug/L	0.42	ug/L	M	1.80E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.0E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.0E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.4.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.49	ug/L	0.49	ug/L	M	1.71E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	5.7E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.4.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.42	ug/L	0.42	ug/L	M	8.18E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.7E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.5.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Wallace - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	18.22	mg/kg	18.22	mg/kg	M	1.82E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	4.5E-02
	Arsenic	27.65	mg/kg	27.65	mg/kg	M	1.66E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.5E-02
	Cadmium	10.72	mg/kg	10.72	mg/kg	M	1.07E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.1E-02
	Iron	28639	mg/kg	28639	mg/kg	M	2.86E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.5E-02
	Manganese	1502	mg/kg	1502	mg/kg	M	1.50E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.2E-02
	Zinc	1921	mg/kg	1921	mg/kg	M	1.92E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	6.4E-03
	(Total)												2.4E-01
Dermal	Arsenic	27.65	mg/kg	27.65	mg/kg	M	5.61E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.9E-02
	Cadmium	10.72	mg/kg	10.72	mg/kg	M	7.25E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.9E-03
	(Total)												2.2E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.7E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.5.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Wallace - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	17.83	mg/kg	17.83	mg/kg	M	3.56E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	8.9E-03
	Arsenic	25.8	mg/kg	25.8	mg/kg	M	3.09E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.0E-02
	Cadmium	9.64	mg/kg	9.64	mg/kg	M	1.92E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.9E-03
	Iron	27036	mg/kg	27036	mg/kg	M	5.40E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.8E-02
	Manganese	1366	mg/kg	1366	mg/kg	M	2.73E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	5.8E-03
	Zinc	1775	mg/kg	1775	mg/kg	M	3.54E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E-03
	(Total)												4.6E-02
Dermal	Arsenic	25.8	mg/kg	25.8	mg/kg	M	2.62E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.7E-03
	Cadmium	9.64	mg/kg	9.64	mg/kg	M	3.26E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.3E-03
	(Total)												1.0E-02
												Total Hazard Index Across All Exposure Routes/Pathways	5.6E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.6.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Wallace - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	18.22	mg/kg	18.22	mg/kg	M	3.39E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	8.5E-02
	Arsenic	27.65	mg/kg	27.65	mg/kg	M	3.09E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.0E-01
	Cadmium	10.72	mg/kg	10.72	mg/kg	M	2.00E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.0E-02
	Iron	28639	mg/kg	28639	mg/kg	M	5.34E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.8E-01
	Manganese	1502	mg/kg	1502	mg/kg	M	2.80E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	6.0E-02
	Zinc	1921	mg/kg	1921	mg/kg	M	3.58E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E-02
	(Total)												4.6E-01
Dermal	Arsenic	27.65	mg/kg	27.65	mg/kg	M	4.53E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.5E-02
	Cadmium	10.72	mg/kg	10.72	mg/kg	M	5.86E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.3E-03
	(Total)												1.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	4.7E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.6.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Wallace - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	17.83	mg/kg	17.83	mg/kg	M	6.64E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.7E-02
	Arsenic	25.8	mg/kg	25.8	mg/kg	M	5.77E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.9E-02
	Cadmium	9.64	mg/kg	9.64	mg/kg	M	3.59E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.6E-03
	Iron	27036	mg/kg	27036	mg/kg	M	1.01E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.4E-02
	Manganese	1366	mg/kg	1366	mg/kg	M	5.09E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.1E-02
	Zinc	1775	mg/kg	1775	mg/kg	M	6.61E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.2E-03
	(Total)												8.6E-02
Dermal	Arsenic	25.8	mg/kg	25.8	mg/kg	M	2.11E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.0E-03
	Cadmium	9.64	mg/kg	9.64	mg/kg	M	2.63E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.1E-03
	(Total)												8.1E-03
												Total Hazard Index Across All Exposure Routes/Pathways	9.4E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.7.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Wallace - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	18.22	mg/kg	18.22	mg/kg	M	8.50E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.1E-02
	Arsenic	27.65	mg/kg	27.65	mg/kg	M	7.74E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.6E-02
	Cadmium	10.72	mg/kg	10.72	mg/kg	M	5.00E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	5.0E-03
	Iron	28639	mg/kg	28639	mg/kg	M	1.34E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.5E-02
	Manganese	1502	mg/kg	1502	mg/kg	M	7.01E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.5E-02
	Zinc	1921	mg/kg	1921	mg/kg	M	8.96E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.0E-03
	(Total)												1.1E-01
Dermal	Arsenic	27.65	mg/kg	27.65	mg/kg	M	1.35E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.5E-03
	Cadmium	10.72	mg/kg	10.72	mg/kg	M	1.74E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	7.0E-04
	(Total)												5.2E-03
												Total Hazard Index Across All Exposure Routes/Pathways	1.2E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.6.7.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Wallace - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	17.83	mg/kg	17.83	mg/kg	M	1.88E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	4.7E-03
	Arsenic	25.8	mg/kg	25.8	mg/kg	M	1.64E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5..5E-03
	Cadmium	9.64	mg/kg	9.64	mg/kg	M	1.02E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.0E-03
	Iron	27036	mg/kg	27036	mg/kg	M	2.86E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.5E-03
	Manganese	1366	mg/kg	1366	mg/kg	M	1.44E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.1E-03
	Zinc	1775	mg/kg	1775	mg/kg	M	1.87E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	6.2E-04
	(Total)												2.4E-02
Dermal	Arsenic	25.8	mg/kg	25.8	mg/kg	M	6.70E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.2E-03
	Cadmium	9.64	mg/kg	9.64	mg/kg	M	8.35E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.3E-04
	(Total)												2.6E-03
												Total Hazard Index Across All Exposure Routes/Pathways	2.7E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.7.1.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
OWS - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	SFCDAR
Receptor Population:	Neighboorhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	38.11	mg/kg	38.11	mg/kg	M	2.35E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	5.9E-02
	Arsenic	52.64	mg/kg	52.64	mg/kg	M	1.95E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.5E-02
	Cadmium	18.89	mg/kg	18.89	mg/kg	M	1.16E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.2E-02
	Iron	41314	mg/kg	41314	mg/kg	M	2.55E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.5E-02
	Manganese	3755	mg/kg	3755	mg/kg	M	2.31E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	4.9E-02
	Zinc	3397	mg/kg	3397	mg/kg	M	2.09E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.0E-03
	(Total)												2.8E-01
Dermal	Arsenic	52.64	mg/kg	52.64	mg/kg	M	1.51E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.0E-02
	Cadmium	18.89	mg/kg	18.89	mg/kg	M	1.80E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	7.2E-03
	(Total)												5.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.3E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.7.1.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
OWS - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	SFCDAR
Receptor Population:	Neighboorhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	25.24	mg/kg	25.24	mg/kg	M	2.96E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	7.4E-03
	Arsenic	36.81	mg/kg	36.81	mg/kg	M	2.59E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.6E-03
	Cadmium	14.16	mg/kg	14.16	mg/kg	M	1.66E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.7E-03
	Iron	38174	mg/kg	38174	mg/kg	M	4.48E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.5E-02
	Manganese	3314	mg/kg	3314	mg/kg	M	3.89E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	8.3E-03
	Zinc	2551	mg/kg	2551	mg/kg	M	3.00E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.0E-03
	(Total)												4.2E-02
Dermal	Arsenic	36.81	mg/kg	36.81	mg/kg	M	5.27E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.8E-02
	Cadmium	14.16	mg/kg	14.16	mg/kg	M	6.76E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.7E-03
	(Total)												2.0E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.2E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.7.2.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
OWS - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	1.096	ug/L	1.096	ug/L	M	3.09E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.0E-03
	Cadmium	4.029	ug/L	4.029	ug/L	M	1.14E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	2.3E-03
	Manganese	32.04	ug/L	32.04	ug/L	M	9.03E-06	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.9E-04
	Mercury	0.122	ug/L	0.122	ug/L	M	3.44E-08	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-04
												Total Hazard Index Across All Exposure Routes/Pathways	3.6E-03

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.7.2.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
OWS - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.78	ug/L	0.78	ug/L	M	1.10E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.7E-04
	Cadmium	10.49	ug/L	10.49	ug/L	M	1.48E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	3.0E-03
	Manganese	39.36	ug/L	39.36	ug/L	M	5.55E-06	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.2E-04
	Mercury	0.1	ug/L	0.1	ug/L	M	1.41E-08	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.7E-05
												Total Hazard Index Across All Exposure Routes/Pathways	3.5E-03

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.7.3.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
OWS - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	9.26	mg/kg	9.26	mg/kg	M	2.12E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	5.3E-02
	Arsenic	27.31	mg/kg	27.31	mg/kg	M	3.75E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.3E-01
	Cadmium	5.21	mg/kg	5.21	mg/kg	M	1.19E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.2E-02
	Iron	21860	mg/kg	21860	mg/kg	M	5.01E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.7E-01
	Manganese	1129	mg/kg	1129	mg/kg	M	2.58E-03	mg/kg-d	1.4E-01	mg/kg-d	N/A	N/A	1.8E-02
	Zinc	752	mg/kg	752	mg/kg	M	1.72E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.7E-03
	(Total)												3.8E-01
Dermal	Arsenic	27.31	mg/kg	27.31	mg/kg	M	1.56E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.2E-03
	Cadmium	5.21	mg/kg	5.21	mg/kg	M	9.94E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	4.0E-04
	(Total)												5.6E-03
												Total Hazard Index Across All Exposure Routes/Pathways	3.9E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.7.3.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
OWS - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	6.69	mg/kg	6.69	mg/kg	M	2.25E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	5.6E-03
	Arsenic	21.87	mg/kg	21.87	mg/kg	M	4.42E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.5E-02
	Cadmium	6.09	mg/kg	6.09	mg/kg	M	2.05E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.0E-03
	Iron	21538	mg/kg	21538	mg/kg	M	7.25E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.4E-02
	Manganese	1077	mg/kg	1077	mg/kg	M	3.63E-04	mg/kg-d	1.4E-01	mg/kg-d	N/A	N/A	2.6E-03
	Zinc	666	mg/kg	666	mg/kg	M	2.24E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.5E-04
	(Total)												5.0E-02
Dermal	Arsenic	21.87	mg/kg	21.87	mg/kg	M	2.76E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	9.2E-04
	Cadmium	6.09	mg/kg	6.09	mg/kg	M	2.56E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.0E-04
	(Total)												1.0E-03
												Total Hazard Index Across All Exposure Routes/Pathways	5.1E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.1.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	11.94	mg/kg	11.94	mg/kg	M	1.53E-04	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.8E-01
	Arsenic	41.62	mg/kg	41.62	mg/kg	M	3.14E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E+00
	Cadmium	6.52	mg/kg	6.52	mg/kg	M	8.34E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	8.3E-02
	Iron	23311	mg/kg	23311	mg/kg	M	2.98E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.9E-01
	Manganese	1250	mg/kg	1250	mg/kg	M	1.60E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.4E-01
	Zinc	1159	mg/kg	1159	mg/kg	M	1.48E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.9E-02
	(Total)												2.9E+00
Dermal	Arsenic	41.62	mg/kg	41.62	mg/kg	M	3.51E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.2E-01
	Cadmium	6.52	mg/kg	6.52	mg/kg	M	1.83E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	7.3E-03
	(Total)												1.2E-01
												Total Hazard Index Across All Exposure Routes/Pathways	3.0E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.1.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	8.21	mg/kg	8.21	mg/kg	M	3.90E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	9.7E-02
	Arsenic	31.58	mg/kg	31.58	mg/kg	M	9.00E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.0E-01
	Cadmium	6	mg/kg	6	mg/kg	M	2.85E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.8E-02
	Iron	21868	mg/kg	21868	mg/kg	M	1.04E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.5E-01
	Manganese	1154	mg/kg	1154	mg/kg	M	5.48E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.2E-01
	Zinc	1112	mg/kg	1112	mg/kg	M	5.28E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.8E-02
	(Total)												9.1E-01
Dermal	Arsenic	31.58	mg/kg	31.58	mg/kg	M	1.98E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.6E-02
	Cadmium	6	mg/kg	6	mg/kg	M	1.25E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	5.0E-03
	(Total)												7.1E-02
												Total Hazard Index Across All Exposure Routes/Pathways	9.8E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.2.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	11.94	mg/kg	11.94	mg/kg	M	4.36E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.1E-01
	Arsenic	41.62	mg/kg	41.62	mg/kg	M	9.12E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.0E-01
	Cadmium	6.52	mg/kg	6.52	mg/kg	M	2.38E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.4E-02
	Iron	23311	mg/kg	23311	mg/kg	M	8.52E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.8E-01
	Manganese	1250	mg/kg	1250	mg/kg	M	4.57E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	9.7E-02
	Zinc	1159	mg/kg	1159	mg/kg	M	4.23E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.4E-02
	(Total)												8.3E-01
Dermal	Arsenic	41.62	mg/kg	41.62	mg/kg	M	1.04E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.5E-02
	Cadmium	6.52	mg/kg	6.52	mg/kg	M	5.45E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.2E-03
	(Total)												3.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	8.7E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.2.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	8.21	mg/kg	8.21	mg/kg	M	1.19E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.0E-02
	Arsenic	31.58	mg/kg	31.58	mg/kg	M	2.75E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	9.2E-02
	Cadmium	6	mg/kg	6	mg/kg	M	8.71E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	8.7E-03
	Iron	21868	mg/kg	21868	mg/kg	M	3.17E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.1E-01
	Manganese	1154	mg/kg	1154	mg/kg	M	1.67E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.6E-02
	Zinc	1112	mg/kg	1112	mg/kg	M	1.61E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.4E-03
	(Total)												2.8E-01
Dermal	Arsenic	31.58	mg/kg	31.58	mg/kg	M	6.27E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.1E-02
	Cadmium	6	mg/kg	6	mg/kg	M	3.97E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.6E-03
	(Total)												2.3E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.0E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.3.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.74	ug/L	0.74	ug/L	M	4.73E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.6E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.6E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.3.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.45	ug/L	0.45	ug/L	M	1.92E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.4E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.4E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.4.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.74	ug/L	0.74	ug/L	M	2.57E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.6E-02
												Total Hazard Index Across All Exposure Routes/Pathways	8.6E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.4.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.45	ug/L	0.45	ug/L	M	8.76E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.9E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.9E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.5.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	2.1	ug/L	2.1	ug/L	M	1.34E-04	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.4E-01
	Arsenic	1.25	ug/L	1.25	ug/L	M	7.99E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.7E-01
	Cadmium	130.85	ug/L	130.85	ug/L	M	8.36E-03	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	1.7E+01
	Zinc	19756	ug/L	19756	ug/L	M	1.26E+00	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.2E+00
												Total Hazard Index Across All Exposure Routes/Pathways	2.2E+01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.5.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	1.67	ug/L	1.67	ug/L	M	7.14E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.8E-01
	Arsenic	0.81	ug/L	0.81	ug/L	M	3.46E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.2E-01
	Cadmium	91.79	ug/L	91.79	ug/L	M	3.92E-03	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	7.8E+00
	Zinc	14239	ug/L	14239	ug/L	M	6.09E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.0E+00
												Total Hazard Index Across All Exposure Routes/Pathways	1.0E+01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.6.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	2.1	ug/L	2.1	ug/L	M	7.29E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.8E-01
	Arsenic	1.25	ug/L	1.25	ug/L	M	4.34E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.4E-01
	Cadmium	130.85	ug/L	130.85	ug/L	M	4.54E-03	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	9.1E+00
	Zinc	19756	ug/L	19756	ug/L	M	6.86E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.3E+00
												Total Hazard Index Across All Exposure Routes/Pathways	1.2E+01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.6.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	1.67	ug/L	1.67	ug/L	M	3.25E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	8.1E-02
	Arsenic	0.81	ug/L	0.81	ug/L	M	1.58E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.3E-02
	Cadmium	91.79	ug/L	91.79	ug/L	M	1.79E-03	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	3.6E+00
	Zinc	14239	ug/L	14239	ug/L	M	2.77E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.2E-01
												Total Hazard Index Across All Exposure Routes/Pathways	4.6E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.7.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Waste piles
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	42.34	mg/kg	42.34	mg/kg	M	2.11E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	5.3E-02
	Arsenic	518.49	mg/kg	518.49	mg/kg	M	1.55E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.2E-01
	Cadmium	31.67	mg/kg	31.67	mg/kg	M	1.58E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.6E-02
	Iron	43032	mg/kg	43032	mg/kg	M	2.15E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.2E-02
	Manganese	1721	mg/kg	1721	mg/kg	M	8.59E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.8E-02
	Zinc	6196	mg/kg	6196	mg/kg	M	3.09E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.0E-02
	(Total)												6.9E-01
Dermal	Arsenic	518.49	mg/kg	518.49	mg/kg	M	5.26E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.8E-01
	Cadmium	31.67	mg/kg	31.67	mg/kg	M	1.07E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	4.3E-03
	(Total)												1.8E-01
												Total Hazard Index Across All Exposure Routes/Pathways	8.7E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.7.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Waste piles
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	19.81	mg/kg	19.81	mg/kg	M	1.98E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	4.9E-03
	Arsenic	95.57	mg/kg	95.57	mg/kg	M	5.72E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.9E-02
	Cadmium	19.39	mg/kg	19.39	mg/kg	M	1.94E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.9E-03
	Iron	36629	mg/kg	36629	mg/kg	M	3.66E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.2E-02
	Manganese	1478	mg/kg	1478	mg/kg	M	1.48E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.1E-03
	Zinc	5147	mg/kg	5147	mg/kg	M	5.14E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.7E-03
	(Total)												4.3E-02
Dermal	Arsenic	95.57	mg/kg	95.57	mg/kg	M	4.85E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.6E-02
	Cadmium	19.39	mg/kg	19.39	mg/kg	M	3.28E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.3E-03
	(Total)												1.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.0E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.8.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Nine Mile/Canyon Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	71.25	mg/kg	71.25	mg/kg	M	4.39E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.1E-01
	Arsenic	50.62	mg/kg	50.62	mg/kg	M	1.87E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.2E-02
	Cadmium	59.2	mg/kg	59.2	mg/kg	M	3.65E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.6E-02
	Iron	36476	mg/kg	36476	mg/kg	M	2.25E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.5E-02
	Manganese	2626	mg/kg	2626	mg/kg	M	1.62E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.4E-02
	Zinc	8883	mg/kg	8883	mg/kg	M	5.48E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.8E-02
	(Total)												3.4E-01
Dermal	Arsenic	50.62	mg/kg	50.62	mg/kg	M	1.45E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.8E-02
	Cadmium	59.2	mg/kg	59.2	mg/kg	M	5.65E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.3E-02
	(Total)												7.1E-02
												Total Hazard Index Across All Exposure Routes/Pathways	4.1E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.8.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Nine Mile/Canyon Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	47.54	mg/kg	47.54	mg/kg	M	5.58E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.4E-02
	Arsenic	23.12	mg/kg	23.12	mg/kg	M	1.63E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.4E-03
	Cadmium	58.8	mg/kg	58.8	mg/kg	M	6.90E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	6.9E-03
	Iron	26575	mg/kg	26575	mg/kg	M	3.12E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.0E-02
	Manganese	1856	mg/kg	1856	mg/kg	M	2.18E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	4.6E-03
	Zinc	8396	mg/kg	8396	mg/kg	M	9.86E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.3E-03
	(Total)												4.5E-02
Dermal	Arsenic	23.12	mg/kg	23.12	mg/kg	M	3.31E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-02
	Cadmium	58.8	mg/kg	58.8	mg/kg	M	2.81E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.1E-02
	(Total)												2.2E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.7E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.9.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Nine Mile/Canyon Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	1.1	ug/L	1.1	ug/L	M	3.10E-07	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.0E-03
	Cadmium	111.02	ug/L	111.02	ug/L	M	3.13E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.3E-02
	Manganese	1699	ug/L	1699	ug/L	M	4.79E-04	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	1.0E-02
	Mercury	0.31	ug/L	0.31	ug/L	M	8.74E-08	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.9E-04
												Total Hazard Index Across All Exposure Routes/Pathways	7.4E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.9.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Nine Mile/Canyon Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.7	ug/L	0.7	ug/L	M	9.86E-08	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.3E-04
	Cadmium	58.92	ug/L	58.92	ug/L	M	8.30E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.7E-02
	Manganese	748.82	ug/L	748.82	ug/L	M	1.06E-04	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	2.2E-03
	Mercury	0.27	ug/L	0.27	ug/L	M	3.80E-08	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.3E-04
												Total Hazard Index Across All Exposure Routes/Pathways	1.9E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.10.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	10.61	mg/kg	10.61	mg/kg	M	2.43E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	6.1E-02
	Arsenic	24.97	mg/kg	24.97	mg/kg	M	3.43E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-01
	Cadmium	6.54	mg/kg	6.54	mg/kg	M	1.50E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.5E-02
	Iron	21976	mg/kg	21976	mg/kg	M	5.03E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.7E-01
	Manganese	1135	mg/kg	1135	mg/kg	M	2.60E-03	mg/kg-d	1.4E-01	mg/kg-d	N/A	N/A	1.9E-02
	Zinc	1070	mg/kg	1070	mg/kg	M	2.45E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.2E-03
	(Total)												3.8E-01
Dermal	Arsenic	24.97	mg/kg	24.97	mg/kg	M	1.43E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.8E-03
	Cadmium	6.54	mg/kg	6.54	mg/kg	M	1.25E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	5.0E-04
	(Total)												5.3E-03
												Total Hazard Index Across All Exposure Routes/Pathways	3.9E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.8.10.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	8.94	mg/kg	8.94	mg/kg	M	3.01E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	7.5E-03
	Arsenic	21.24	mg/kg	21.24	mg/kg	M	4.29E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.4E-02
	Cadmium	6.06	mg/kg	6.06	mg/kg	M	2.04E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.0E-03
	Iron	21336	mg/kg	21336	mg/kg	M	7.18E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.4E-02
	Manganese	1092	mg/kg	1092	mg/kg	M	3.68E-04	mg/kg-d	1.4E-01	mg/kg-d	N/A	N/A	2.6E-03
	Zinc	1000	mg/kg	1000	mg/kg	M	3.37E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.1E-03
	(Total)												5.2E-02
Dermal	Arsenic	21.24	mg/kg	21.24	mg/kg	M	2.68E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.9E-04
	Cadmium	6.06	mg/kg	6.06	mg/kg	M	2.55E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.0E-04
	(Total)												1.0E-03
												Total Hazard Index Across All Exposure Routes/Pathways	5.3E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.1.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	20.05	mg/kg	20.05	mg/kg	M	2.56E-04	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	6.4E-01
	Arsenic	39.04	mg/kg	39.04	mg/kg	M	2.99E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.0E-+00
	Cadmium	6.39	mg/kg	6.39	mg/kg	M	8.17E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	8.2E-02
	Iron	24742	mg/kg	24742	mg/kg	M	3.16E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.1E+00
	Manganese	1628	mg/kg	1628	mg/kg	M	2.08E-02	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	4.4E-01
	Zinc	1375	mg/kg	1375	mg/kg	M	1.76E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.9E-02
	(Total)												3.3E+00
Dermal	Arsenic	39.04	mg/kg	39.04	mg/kg	M	3.29E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-01
	Cadmium	6.39	mg/kg	6.39	mg/kg	M	1.80E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	7.2E-03
	(Total)												1.2E-01
												Total Hazard Index Across All Exposure Routes/Pathways	3.4E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.1.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	11.04	mg/kg	11.04	mg/kg	M	5.24E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.3E-01
	Arsenic	27.3	mg/kg	27.3	mg/kg	M	7.78E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.6E-01
	Cadmium	5.07	mg/kg	5.07	mg/kg	M	2.41E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.4E-02
	Iron	22709	mg/kg	22709	mg/kg	M	1.08E-01	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.6E-01
	Manganese	1412	mg/kg	1412	mg/kg	M	6.71E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.4E-01
	Zinc	1049	mg/kg	1049	mg/kg	M	4.98E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.7E-02
	(Total)												9.3E-01
Dermal	Arsenic	27.3	mg/kg	27.3	mg/kg	M	1.71E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.7E-02
	Cadmium	5.07	mg/kg	5.07	mg/kg	M	1.06E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	4.2E-03
	(Total)												6.1E-02
												Total Hazard Index Across All Exposure Routes/Pathways	9.9E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.2.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	20.05	mg/kg	20.05	mg/kg	M	7.32E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.8E-01
	Arsenic	39.04	mg/kg	39.04	mg/kg	M	8.56E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.9E-01
	Cadmium	6.39	mg/kg	6.39	mg/kg	M	2.33E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.3E-02
	Iron	24742	mg/kg	24742	mg/kg	M	9.04E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.0E-01
	Manganese	1628	mg/kg	1628	mg/kg	M	5.95E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.3E-01
	Zinc	1375	mg/kg	1375	mg/kg	M	5.02E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.7E-02
	(Total)												9.4E-01
Dermal	Arsenic	39.04	mg/kg	39.04	mg/kg	M	9.80E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.3E-02
	Cadmium	6.39	mg/kg	6.39	mg/kg	M	5.35E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	2.1E-03
	(Total)												3.5E-02
												Total Hazard Index Across All Exposure Routes/Pathways	9.7E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.2.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Yard Soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	11.04	mg/kg	11.04	mg/kg	M	1.60E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	4.0E-02
	Arsenic	27.3	mg/kg	27.3	mg/kg	M	2.38E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.9E-02
	Cadmium	5.07	mg/kg	5.07	mg/kg	M	7.36E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	7.4E-03
	Iron	22709	mg/kg	22709	mg/kg	M	3.30E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.1E-01
	Manganese	1412	mg/kg	1412	mg/kg	M	2.05E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	4.4E-02
	Zinc	1049	mg/kg	1049	mg/kg	M	1.52E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.1E-03
	(Total)												2.9E-01
Dermal	Arsenic	27.3	mg/kg	27.3	mg/kg	M	5.42E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.8E-02
	Cadmium	5.07	mg/kg	5.07	mg/kg	M	3.36E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.3E-03
	(Total)												1.9E-02
												Total Hazard Index Across All Exposure Routes/Pathways	3.0E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.3.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.25	ug/L	0.25	ug/L	M	1.60E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.3E-02
												Total Hazard Index Across All Exposure Routes/Pathways	5.3E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.3.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.13	ug/L	0.13	ug/L	M	5.56E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.9E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.9E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.4.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.25	ug/L	0.25	ug/L	M	8.68E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.9E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.9E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.4.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.13	ug/L	0.13	ug/L	M	2.53E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.4E-03
												Total Hazard Index Across All Exposure Routes/Pathways	8.4E-03

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.5.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Waste piles
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	20.3	mg/kg	20.3	mg/kg	M	1.01E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.5E-02
	Arsenic	42.1	mg/kg	42.1	mg/kg	M	1.26E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.2E-02
	Cadmium	13.8	mg/kg	13.8	mg/kg	M	6.89E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	6.9E-03
	Iron	33200	mg/kg	33200	mg/kg	M	1.66E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.5E-02
	Manganese	2750	mg/kg	2750	mg/kg	M	1.37E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.9E-02
	Zinc	2880	mg/kg	2880	mg/kg	M	1.44E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	4.8E-03
	(Total)												1.6E-01
Dermal	Arsenic	42.1	mg/kg	42.1	mg/kg	M	4.27E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.4E-02
	Cadmium	13.8	mg/kg	13.8	mg/kg	M	4.66E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.9E-03
	(Total)												1.6E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.8E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.5.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Waste piles
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	11.84	mg/kg	11.84	mg/kg	M	1.18E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.0E-03
	Arsenic	23.6	mg/kg	23.6	mg/kg	M	1.41E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.7E-03
	Cadmium	5.57	mg/kg	5.57	mg/kg	M	5.56E-07	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	5.6E-04
	Iron	22180	mg/kg	22180	mg/kg	M	2.21E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.4E-03
	Manganese	2103	mg/kg	2103	mg/kg	M	2.10E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	4.5E-03
	Zinc	1042	mg/kg	1042	mg/kg	M	1.04E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	3.5E-04
	(Total)												2.0E-02
Dermal	Arsenic	23.6	mg/kg	23.6	mg/kg	M	1.20E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.0E-03
	Cadmium	5.57	mg/kg	5.57	mg/kg	M	9.41E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.8E-04
	(Total)												4.4E-03
												Total Hazard Index Across All Exposure Routes/Pathways	2.5E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.6.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	4.46	mg/kg	4.46	mg/kg	M	2.75E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	6.9E-03
	Arsenic	15.89	mg/kg	15.89	mg/kg	M	5.88E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.0E-02
	Cadmium	19.83	mg/kg	19.83	mg/kg	M	1.22E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.2E-02
	Iron	47300	mg/kg	47300	mg/kg	M	2.92E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.7E-02
	Manganese	5233	mg/kg	5233	mg/kg	M	3.23E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	6.9E-02
	Zinc	4017	mg/kg	4017	mg/kg	M	2.48E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.3E-03
	(Total)												2.1E-01
Dermal	Arsenic	15.89	mg/kg	15.89	mg/kg	M	4.55E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.5E-02
	Cadmium	19.83	mg/kg	19.83	mg/kg	M	1.89E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	7.6E-03
	(Total)												2.3E-02
												Total Hazard Index Across All Exposure Routes/Pathways	2.4E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.6.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	2.61	mg/kg	2.61	mg/kg	M	3.06E-07	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	7.7E-04
	Arsenic	13.21	mg/kg	13.21	mg/kg	M	9.31E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.1E-03
	Cadmium	9.66	mg/kg	9.66	mg/kg	M	1.13E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.1E-03
	Iron	34079	mg/kg	34079	mg/kg	M	4.00E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.3E-02
	Manganese	3513	mg/kg	3513	mg/kg	M	4.12E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	8.8E-03
	Zinc	2208	mg/kg	2208	mg/kg	M	2.59E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	8.6E-04
	(Total)												2.8E-02
Dermal	Arsenic	13.21	mg/kg	13.21	mg/kg	M	1.89E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.3E-03
	Cadmium	9.66	mg/kg	9.66	mg/kg	M	4.61E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.8E-03
	(Total)												8.1E-03
												Total Hazard Index Across All Exposure Routes/Pathways	3.6E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.7.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.6	ug/L	0.6	ug/L	M	1.69E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.6E-04
	Cadmium	1.24	ug/L	1.24	ug/L	M	3.49E-07	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	7.0E-04
	Manganese	18.96	ug/L	18.96	ug/L	M	5.34E-06	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.1E-04
	Mercury	0.086	ug/L	0.086	ug/L	M	2.42E-08	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.1E-05
												Total Hazard Index Across All Exposure Routes/Pathways	1.5E-03

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.7.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.54	ug/L	0.54	ug/L	M	7.61E-08	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.5E-04
	Cadmium	0.81	ug/L	0.81	ug/L	M	1.14E-07	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	2.3E-04
	Manganese	13.66	ug/L	13.66	ug/L	M	1.92E-06	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	4.1E-05
	Mercury	0.08	ug/L	0.08	ug/L	M	1.13E-08	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.8E-05
												Total Hazard Index Across All Exposure Routes/Pathways	5.6E-04

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.8.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Mullan - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	18.68	mg/kg	18.68	mg/kg	M	4.28E-05	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.1E-01
	Arsenic	31.66	mg/kg	31.66	mg/kg	M	4.35E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.4E-01
	Cadmium	5.04	mg/kg	5.04	mg/kg	M	1.15E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.2E-02
	Iron	26912	mg/kg	26912	mg/kg	M	6.16E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.1E-01
	Manganese	1941	mg/kg	1941	mg/kg	M	4.44E-03	mg/kg-d	1.4E-01	mg/kg-d	N/A	N/A	3.2E-02
	Zinc	953	mg/kg	953	mg/kg	M	2.18E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.3E-03
	(Total)												5.1E-01
Dermal	Arsenic	31.66	mg/kg	31.66	mg/kg	M	1.81E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	6.0E-03
	Cadmium	5.04	mg/kg	5.04	mg/kg	M	9.62E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	3.8E-04
	(Total)												6.4E-03
												Total Hazard Index Across All Exposure Routes/Pathways	5.1E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.9.8.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Mullan - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	14.09	mg/kg	14.09	mg/kg	M	4.74E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.2E-02
	Arsenic	26.48	mg/kg	26.48	mg/kg	M	5.35E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.8E-02
	Cadmium	4.22	mg/kg	4.22	mg/kg	M	1.42E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.4E-03
	Iron	24863	mg/kg	24863	mg/kg	M	8.37E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.8E-02
	Manganese	1688	mg/kg	1688	mg/kg	M	5.68E-04	mg/kg-d	1.4E-01	mg/kg-d	N/A	N/A	4.1E-03
	Zinc	823	mg/kg	823	mg/kg	M	2.77E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	9.2E-04
	(Total)												6.4E-02
Dermal	Arsenic	26.48	mg/kg	26.48	mg/kg	M	3.34E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.1E-03
	Cadmium	4.22	mg/kg	4.22	mg/kg	M	1.78E-09	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	7.1E-05
	(Total)												1.2E-03
												Total Hazard Index Across All Exposure Routes/Pathways	6.5E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.10.1.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Spokane River
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	3.2	mg/kg	3.2	mg/kg	M	5.61E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	1.4E-02
	Arsenic	50	mg/kg	50	mg/kg	M	5.26E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.8E-01
	Cadmium	13.4	mg/kg	13.4	mg/kg	M	2.35E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.3E-02
	Iron	48881	mg/kg	48881	mg/kg	M	8.57E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.9E-01
	Manganese	3666	mg/kg	3666	mg/kg	M	6.43E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	1.4E-01
	Zinc	1800	mg/kg	1800	mg/kg	M	3.16E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.1E-02
	(Total)												6.5E-01
Dermal	Arsenic	50	mg/kg	50	mg/kg	M	1.14E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.8E-02
	Cadmium	13.4	mg/kg	13.4	mg/kg	M	1.02E-07	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	4.1E-03
	(Total)												4.2E-02
												Total Hazard Index Across All Exposure Routes/Pathways	6.9E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.10.1.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Spokane River
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	2.78	mg/kg	2.78	mg/kg	M	9.75E-07	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	2.4E-03
	Arsenic	41.03	mg/kg	41.03	mg/kg	M	8.63E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.9E-02
	Cadmium	11.23	mg/kg	11.23	mg/kg	M	3.94E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.9E-03
	Iron	43485	mg/kg	43485	mg/kg	M	1.52E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.1E-02
	Manganese	2996	mg/kg	2996	mg/kg	M	1.05E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.2E-02
	Zinc	1575	mg/kg	1575	mg/kg	M	5.52E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.8E-03
	(Total)												1.1E-01
Dermal	Arsenic	41.03	mg/kg	41.03	mg/kg	M	4.68E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.6E-02
	Cadmium	11.23	mg/kg	11.23	mg/kg	M	4.27E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.7E-03
	(Total)												1.7E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.3E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.10.2.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Spokane River
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	3.2	mg/kg	3.2	mg/kg	M	1.44E-06	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	3.6E-03
	Arsenic	50	mg/kg	50	mg/kg	M	1.35E-05	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.5E-02
	Cadmium	13.4	mg/kg	13.4	mg/kg	M	6.04E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	6.0E-03
	Iron	48881	mg/kg	48881	mg/kg	M	2.20E-02	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	7.3E-02
	Manganese	3666	mg/kg	3666	mg/kg	M	1.65E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	3.5E-02
	Zinc	1800	mg/kg	1800	mg/kg	M	8.12E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	2.7E-03
	(Total)												1.7E-01
Dermal	Arsenic	50	mg/kg	50	mg/kg	M	4.98E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.7E-02
	Cadmium	13.4	mg/kg	13.4	mg/kg	M	4.45E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	1.8E-03
	(Total)												1.8E-02
												Total Hazard Index Across All Exposure Routes/Pathways	1.8E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.10.2.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Spokane River
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	2.78	mg/kg	2.78	mg/kg	M	2.84E-07	mg/kg-d	4.0E-04	mg/kg-d	N/A	N/A	7.1E-04
	Arsenic	41.03	mg/kg	41.03	mg/kg	M	2.52E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	8.4E-03
	Cadmium	11.23	mg/kg	11.23	mg/kg	M	1.15E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.1E-03
	Iron	43485	mg/kg	43485	mg/kg	M	4.45E-03	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	1.5E-02
	Manganese	2996	mg/kg	2996	mg/kg	M	3.06E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	6.5E-03
	Zinc	1575	mg/kg	1575	mg/kg	M	1.61E-04	mg/kg-d	3.0E-01	mg/kg-d	N/A	N/A	5.4E-04
	(Total)												3.2E-02
Dermal	Arsenic	41.03	mg/kg	41.03	mg/kg	M	2.12E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	7.1E-03
	Cadmium	11.23	mg/kg	11.23	mg/kg	M	1.93E-08	mg/kg-d	2.5E-05	mg/kg-d	N/A	N/A	7.7E-04
	(Total)												7.8E-03
												Total Hazard Index Across All Exposure Routes/Pathways	4.0E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.10.3.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Spokane River
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	20.8	ug/L	20.8	ug/L	M	3.65E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.2E-02
	Cadmium	47.9	ug/L	47.9	ug/L	M	8.40E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	1.7E-02
	Manganese	6980	ug/L	6980	ug/L	M	1.22E-03	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.6E-02
	Mercury	0.1	ug/L	0.1	ug/L	M	1.75E-08	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	5.8E-05
												Total Hazard Index Across All Exposure Routes/Pathways	5.5E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.10.3.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Spokane River
Receptor Population:	Visitor
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	10.56	ug/L	10.56	ug/L	M	9.26E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.1E-03
	Cadmium	20.01	ug/L	20.01	ug/L	M	1.75E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	3.5E-03
	Manganese	2792	ug/L	2792	ug/L	M	2.45E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	5.2E-03
	Mercury	0.09	ug/L	0.09	ug/L	M	7.89E-09	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.6E-05
												Total Hazard Index Across All Exposure Routes/Pathways	1.2E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.10.4.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Spokane River
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	20.8	ug/L	20.8	ug/L	M	1.35E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.5E-03
	Cadmium	47.9	ug/L	47.9	ug/L	M	3.12E-06	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	6.2E-03
	Manganese	6980	ug/L	6980	ug/L	M	4.55E-04	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	9.7E-03
	Mercury	0.1	ug/L	0.1	ug/L	M	6.51E-09	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	2.2E-05
												Total Hazard Index Across All Exposure Routes/Pathways	2.0E-02

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.10.4.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Spokane River
Receptor Population:	Visitor
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	10.56	ug/L	10.56	ug/L	M	3.60E-07	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.2E-03
	Cadmium	20.01	ug/L	20.01	ug/L	M	6.82E-07	mg/kg-d	5.0E-04	mg/kg-d	N/A	N/A	1.4E-03
	Manganese	2792	ug/L	2792	ug/L	M	9.52E-05	mg/kg-d	4.7E-02	mg/kg-d	N/A	N/A	2.0E-03
	Mercury	0.09	ug/L	0.09	ug/L	M	3.07E-09	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	1.0E-05
												Total Hazard Index Across All Exposure Routes/Pathways	4.6E-03

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.1.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
All Areas - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water/sediment
Exposure Medium:	Plant tissue
Exposure Point:	Homegrown vegetables
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.025	mg/kg	0.025	mg/kg	M	1.26E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.2E-01
	Cadmium	0.319	mg/kg	0.319	mg/kg	M	1.61E-03	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.6E+00
												Total Hazard Index Across All Exposure Routes/Pathways	2.0E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.1.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
All Areas - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water/sediment
Exposure Medium:	Plant Tissue
Exposure Point:	Homegrown vegetables
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.02	mg/kg	0.02	mg/kg	M	9.84E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.3E-02
	Cadmium	0.21	mg/kg	0.21	mg/kg	M	1.03E-04		1.0E-03	mg/kg-d	N/A	N/A	1.0E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.4E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.2.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
All Areas - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water/sediment
Exposure Medium:	Plant tissue
Exposure Point:	Homegrown vegetables
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.025	mg/kg	0.025	mg/kg	M	1.26E-04	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	4.2E-01
	Cadmium	0.319	mg/kg	0.319	mg/kg	M	1.61E-03	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.6E+00
												Total Hazard Index Across All Exposure Routes/Pathways	2.0E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.2.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
All Areas - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water/sediment
Exposure Medium:	Plant tissue
Exposure Point:	Homegrown vegetables
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Arsenic	0.02	mg/kg	0.02	mg/kg	M	9.84E-06	mg/kg-d	3.0E-04	mg/kg-d	N/A	N/A	3.3E-02
	Cadmium	0.21	mg/kg	0.21	mg/kg	M	1.03E-04	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.0E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.4E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.3.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
All Areas - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Visitors
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of bullhead	Cadmium	0.008	mg/kg	0.008	mg/kg	M	5.26E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	5.3E-03
	Mercury	0.052	mg/kg	0.052	mg/kg	M	3.42E-05	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	3.4E-01
												Total Hazard Index Across All Exposure Routes/Pathways	3.5E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.3.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
All Areas - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Visitors
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of bullhead	Cadmium	0.01	mg/kg	0.01	mg/kg	M	3.57E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.6E-03
	Mercury	0.05	mg/kg	0.05	mg/kg	M	1.79E-05	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	1.8E-01
												Total Hazard Index Across All Exposure Routes/Pathways	1.8E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.4.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
All Areas - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Visitors
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of pike	Cadmium	0.006	mg/kg	0.006	mg/kg	M	3.94E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.9E-03
	Mercury	0.133	mg/kg	0.133	mg/kg	M	8.74E-05	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	8.7E-01
												Total Hazard Index Across All Exposure Routes/Pathways	8.8E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.4.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
All Areas - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Visitors
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of pike	Cadmium	0.01	mg/kg	0.01	mg/kg	M	3.57E-06	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	3.6E-03
	Mercury	0.12	mg/kg	0.12	mg/kg	M	4.29E-05	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	4.3E-01
												Total Hazard Index Across All Exposure Routes/Pathways	4.3E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.5.RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
All Areas - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Visitors
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of perch	Cadmium	0.037	mg/kg	0.037	mg/kg	M	2.43E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.4E-02
	Mercury	0.089	mg/kg	0.089	mg/kg	M	5.85E-05	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	5.8E-01
												Total Hazard Index Across All Exposure Routes/Pathways	6.1E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.5.CT
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
All Areas - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Visitors
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of perch	Cadmium	0.03	mg/kg	0.03	mg/kg	M	1.07E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.1E-02
	Mercury	0.08	mg/kg	0.08	mg/kg	M	2.86E-05	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	2.9E-01
												Total Hazard Index Across All Exposure Routes/Pathways	3.0E-01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.6 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
All Areas -Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of bullhead	Cadmium	0.008	mg/kg	0.008	mg/kg	M	1.94E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.9E-02
	Mercury	0.052	mg/kg	0.052	mg/kg	M	1.26E-04	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	1.3E+00
												Total Hazard Index Across All Exposure Routes/Pathways	1.3E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.7 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
All Areas - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of pike	Cadmium	0.006	mg/kg	0.006	mg/kg	M	1.46E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	1.5E-02
	Mercury	0.133	mg/kg	0.133	mg/kg	M	3.23E-04	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	3.2E+00
												Total Hazard Index Across All Exposure Routes/Pathways	3.2E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.8 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
All Areas - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of perch	Cadmium	0.037	mg/kg	0.037	mg/kg	M	8.99E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	9.0E-02
	Mercury	0.089	mg/kg	0.089	mg/kg	M	2.16E-04	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	2.2E+00
												Total Hazard Index Across All Exposure Routes/Pathways	2.3E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.9 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
All Areas - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of bullhead	Cadmium	0.008	mg/kg	0.008	mg/kg	M	6.17E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	6.2E-02
	Mercury	0.052	mg/kg	0.052	mg/kg	M	4.01E-04	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	4.0E+00
												Total Hazard Index Across All Exposure Routes/Pathways	4.1E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.10 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
All Areas - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of pike	Cadmium	0.006	mg/kg	0.006	mg/kg	M	4.63E-05	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	4.6E-02
	Mercury	0.133	mg/kg	0.133	mg/kg	M	1.03E-03	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	1.0E+01
												Total Hazard Index Across All Exposure Routes/Pathways	1.0E+01

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 7.11.11 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
All Areas - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water/sediment
Exposure Medium:	Animal tissue
Exposure Point:	Fish in the Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion of perch	Cadmium	0.037	mg/kg	0.037	mg/kg	M	2.85E-04	mg/kg-d	1.0E-03	mg/kg-d	N/A	N/A	2.9E-01
	Mercury	0.089	mg/kg	0.089	mg/kg	M	6.87E-04	mg/kg-d	1.0E-04	mg/kg-d	N/A	N/A	6.9E+00
												Total Hazard Index Across All Exposure Routes/Pathways	7.2E+00

(1) M = Medium-Specific, R = Route-Specific
N/A = not applicable.

TABLE 8.1.1.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	48.53	mg/kg	48.53	mg/kg	M	4.56E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.8E-05
Dermal	Arsenic	48.53	mg/kg	48.53	mg/kg	M	5.22E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.8E-06
										Total Risk Across All Exposure Routes/Pathways	7.6E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.1.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	35.47	mg/kg	35.47	mg/kg	M	3.97E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.0E-06
Dermal	Arsenic	35.47	mg/kg	35.47	mg/kg	M	9.06E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.4E-06
										Total Risk Across All Exposure Routes/Pathways	7.3E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.2.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.97	ug/L	0.97	ug/L	M	1.44E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.2E-05
										Total Risk Across All Exposure Routes/Pathways	2.2E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.2.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.38	ug/L	0.38	ug/L	M	9.51E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.4E-06
										Total Risk Across All Exposure Routes/Pathways	1.4E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.3.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Lower CDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	119.45	mg/kg	119.45	mg/kg	M	4.42E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.6E-06
Dermal	Arsenic	119.45	mg/kg	119.45	mg/kg	M	5.36E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.0E-06
										Total Risk Across All Exposure Routes/Pathways	1.5E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.3.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Lower CDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	114.51	mg/kg	114.51	mg/kg	M	2.30E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.5E-07
Dermal	Arsenic	114.51	mg/kg	114.51	mg/kg	M	7.34E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.1E-06
										Total Risk Across All Exposure Routes/Pathways	1.4E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.4.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Disturbed surface water
Exposure Point:	Lower CDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	79.61	ug/L	79.61	ug/L	M	2.24E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.4E-06
										Total Risk Across All Exposure Routes/Pathways	3.4E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.4.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Disturbed surface water
Exposure Point:	Lower CDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	75.64	ug/L	75.64	ug/L	M	3.05E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.6E-07
										Total Risk Across All Exposure Routes/Pathways	4.6E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.5.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Lower CDAR
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	119.45	mg/kg	119.45	mg/kg	M	1.38E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.1E-05
Dermal	Arsenic	119.45	mg/kg	119.45	mg/kg	M	5.10E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.7E-06
										Total Risk Across All Exposure Routes/Pathways	2.8E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.5.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Lower CDAR
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	114.51	mg/kg	114.51	mg/kg	M	9.04E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.4E-06
Dermal	Arsenic	114.51	mg/kg	114.51	mg/kg	M	7.60E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.1E-06
										Total Risk Across All Exposure Routes/Pathways	2.5E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.6.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Disturbed surface water
Exposure Point:	Lower CDAR
Receptor Population:	Visitors
Receptor Age:	Child/Adult years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	79.61	ug/L	79.61	ug/L	M	2.22E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.3E-06
										Total Risk Across All Exposure Routes/Pathways	3.3E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.6.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Disturbed surface water
Exposure Point:	Lower CDAR
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	75.64	ug/L	75.64	ug/L	M	3.32E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	5.0E-07
										Total Risk Across All Exposure Routes/Pathways	5.0E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.7.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	108.91	mg/kg	108.91	mg/kg	M	5.34E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.0E-05
Dermal	Arsenic	108.91	mg/kg	108.91	mg/kg	M	2.23E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.3E-06
										Total Risk Across All Exposure Routes/Pathways	8.3E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.7.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Lower Basin - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	104.41	mg/kg	104.41	mg/kg	M	1.99E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.0E-06
Dermal	Arsenic	104.41	mg/kg	104.41	mg/kg	M	1.24E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.9E-07
										Total Risk Across All Exposure Routes/Pathways	3.2E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.8.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	124.44	mg/kg	124.44	mg/kg	M	6.91E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.0E-04
Dermal	Arsenic	124.44	mg/kg	124.44	mg/kg	M	4.44E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.7E-05
										Total Risk Across All Exposure Routes/Pathways	1.7E-04

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.9.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	120.96	mg/kg	120.96	mg/kg	M	6.72E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.0E-04
Dermal	Arsenic	120.96	mg/kg	120.96	mg/kg	M	1.30E-04	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.9E-04
										Total Risk Across All Exposure Routes/Pathways	3.0E-04

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.10.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Undisturbed surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	20	ug/L	20	ug/L	M	1.57E-04	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.4E-04
										Total Risk Across All Exposure Routes/Pathways	2.4E-04

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.11.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Disturbed surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	79.61	ug/L	79.61	ug/L	M	7.37E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.1E-05
										Total Risk Across All Exposure Routes/Pathways	1.1E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.12.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Soil
Exposure Medium:	Surface Soil
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	124.44	mg/kg	124.44	mg/kg	M	4.21E-04	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.3E-04
Dermal	Arsenic	124.44	mg/kg	124.44	mg/kg	M	1.35E-04	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.0E-04
										Total Risk Across All Exposure Routes/Pathways	8.3E-04

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.13.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	120.96	mg/kg	120.96	mg/kg	M	2.35E-04	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.5E-04
Dermal	Arsenic	120.96	mg/kg	120.96	mg/kg	M	4.55E-04	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.8E-04
										Total Risk Across All Exposure Routes/Pathways	1.0E-03

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.14.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Undisturbed surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	20	ug/L	20	ug/L	M	9.55E-04	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.4E-03
										Total Risk Across All Exposure Routes/Pathways	1.4E-03

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.1.15.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Medium:	Surface water
Exposure Medium:	Disturbed surface water
Exposure Point:	Lower CDAR
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	79.61	ug/L	79.61	ug/L	M	2.58E-04	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.9E-05
										Total Risk Across All Exposure Routes/Pathways	3.9E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.1.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	25.04	mg/kg	25.04	mg/kg	M	2.35E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.5E-05
Dermal	Arsenic	25.04	mg/kg	25.04	mg/kg	M	2.69E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.0E-06
										Total Risk Across All Exposure Routes/Pathways	3.9E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.1.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	20.35	mg/kg	20.35	mg/kg	M	2.28E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.4E-06
Dermal	Arsenic	20.35	mg/kg	20.35	mg/kg	M	5.20E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.8E-07
										Total Risk Across All Exposure Routes/Pathways	4.2E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.2.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.405	ug/L	0.405	ug/L	M	6.02E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	9.0E-06
										Total Risk Across All Exposure Routes/Pathways	9.0E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.2.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Kingston - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.21	ug/L	0.21	ug/L	M	5.26E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.9E-07
										Total Risk Across All Exposure Routes/Pathways	7.9E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.3.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Pine Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	22.35	mg/kg	22.35	mg/kg	M	8.27E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.2E-06
Dermal	Arsenic	22.35	mg/kg	22.35	mg/kg	M	6.40E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	9.6E-07
										Total Risk Across All Exposure Routes/Pathways	2.2E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.3.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Pine Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	15.84	mg/kg	15.84	mg/kg	M	3.19E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.8E-08
Dermal	Arsenic	15.84	mg/kg	15.84	mg/kg	M	6.48E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	9.7E-08
										Total Risk Across All Exposure Routes/Pathways	1.5E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.4.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	NS Confluence
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	163.2	mg/kg	163.2	mg/kg	M	6.04E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	9.1E-06
Dermal	Arsenic	163.2	mg/kg	163.2	mg/kg	M	7.32E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.1E-05
										Total Risk Across All Exposure Routes/Pathways	2.0E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.4.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	NS Confluence
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	145.44	mg/kg	145.44	mg/kg	M	2.93E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.4E-07
Dermal	Arsenic	145.44	mg/kg	145.44	mg/kg	M	9.32E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.4E-06
										Total Risk Across All Exposure Routes/Pathways	1.8E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.5.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Pine Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.73	ug/L	0.73	ug/L	M	2.06E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.1E-08
										Total Risk Across All Exposure Routes/Pathways	3.1E-08

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.5.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Pine Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.57	ug/L	0.57	ug/L	M	2.29E-09	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.4E-09
										Total Risk Across All Exposure Routes/Pathways	3.4E-09

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.6.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	NS Confluence
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	134	ug/L	134	ug/L	M	3.78E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	5.7E-06
										Total Risk Across All Exposure Routes/Pathways	5.7E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.6.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Kingston - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	NS Confluence
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	36.4	ug/L	36.4	ug/L	M	1.47E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.2E-07
										Total Risk Across All Exposure Routes/Pathways	2.2E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.7.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	NS Confluence
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	163.2	mg/kg	163.2	mg/kg	M	1.89E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.8E-05
Dermal	Arsenic	163.2	mg/kg	163.2	mg/kg	M	6.97E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.0E-05
										Total Risk Across All Exposure Routes/Pathways	3.9E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.7.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	NS Confluence
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	145.44	mg/kg	145.44	mg/kg	M	1.15E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.7E-06
Dermal	Arsenic	145.44	mg/kg	145.44	mg/kg	M	9.65E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.4E-06
										Total Risk Across All Exposure Routes/Pathways	3.2E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.8.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	NS Confluence
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	134	ug/L	134	ug/L	M	3.74E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	5.6E-06
										Total Risk Across All Exposure Routes/Pathways	5.6E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.8.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Kingston - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	NS Confluence
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	36.4	ug/L	36.4	ug/L	M	1.60E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.4E-07
										Total Risk Across All Exposure Routes/Pathways	2.4E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.9.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Kingston - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	34.6	mg/kg	34.6	mg/kg	M	1.70E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.5E-05
Dermal	Arsenic	34.6	mg/kg	34.6	mg/kg	M	7.07E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.1E-06
										Total Risk Across All Exposure Routes/Pathways	2.7E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.2.9.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Kingston - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	29.06	mg/kg	29.06	mg/kg	M	5.53E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.3E-07
Dermal	Arsenic	29.06	mg/kg	29.06	mg/kg	M	3.46E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	5.2E-08
										Total Risk Across All Exposure Routes/Pathways	8.8E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.3.1.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	50.74	mg/kg	50.74	mg/kg	M	4.77E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.1E-05
Dermal	Arsenic	50.74	mg/kg	50.74	mg/kg	M	5.46E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.2E-06
										Total Risk Across All Exposure Routes/Pathways	8.0E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.3.1.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	39.66	mg/kg	39.66	mg/kg	M	4.44E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.7E-06
Dermal	Arsenic	39.66	mg/kg	39.66	mg/kg	M	1.01E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.5E-06
										Total Risk Across All Exposure Routes/Pathways	8.2E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.3.2.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	8.4	ug/L	8.4	ug/L	M	1.25E-04	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.9E-04
										Total Risk Across All Exposure Routes/Pathways	1.9E-04

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.3.2.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	1.92	ug/L	1.92	ug/L	M	4.81E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.2E-06
										Total Risk Across All Exposure Routes/Pathways	7.2E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.3.3.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Elk Creek area
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	98.5	mg/kg	98.5	mg/kg	M	5.90E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.8E-06
Dermal	Arsenic	98.5	mg/kg	98.5	mg/kg	M	2.00E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.0E-06
										Total Risk Across All Exposure Routes/Pathways	1.2E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.3.3.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Elk Creek area
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	79.96	mg/kg	79.96	mg/kg	M	2.74E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.1E-07
Dermal	Arsenic	79.96	mg/kg	79.96	mg/kg	M	2.32E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.5E-07
										Total Risk Across All Exposure Routes/Pathways	7.6E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.3.4.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Elk Creek Pond
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	77.74	mg/kg	77.74	mg/kg	M	2.88E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.3E-06
Dermal	Arsenic	77.74	mg/kg	77.74	mg/kg	M	2.23E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.3E-06
										Total Risk Across All Exposure Routes/Pathways	7.7E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.3.4.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Elk Creek Pond
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	63.76	mg/kg	63.76	mg/kg	M	1.28E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.9E-07
Dermal	Arsenic	63.76	mg/kg	63.76	mg/kg	M	2.61E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.9E-07
										Total Risk Across All Exposure Routes/Pathways	5.8E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.3.5.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Elk Creek Pond
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	32	ug/L	32	ug/L	M	9.02E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.4E-06
										Total Risk Across All Exposure Routes/Pathways	1.4E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.3.5.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Side Gulches - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Elk Creek Pond
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	16.64	ug/L	16.64	ug/L	M	6.70E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.0E-07
										Total Risk Across All Exposure Routes/Pathways	1.0E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.4.1.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	46.74	mg/kg	46.74	mg/kg	M	4.39E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.6E-05
Dermal	Arsenic	46.74	mg/kg	46.74	mg/kg	M	5.03E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.5E-06
										Total Risk Across All Exposure Routes/Pathways	7.3E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.4.1.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	38.95	mg/kg	38.95	mg/kg	M	4.36E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.5E-06
Dermal	Arsenic	38.95	mg/kg	38.95	mg/kg	M	9.95E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.5E-06
										Total Risk Across All Exposure Routes/Pathways	8.0E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.4.2.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.28	ug/L	0.28	ug/L	M	4.16E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.2E-06
										Total Risk Across All Exposure Routes/Pathways	6.2E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.4.2.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Osburn - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.23	ug/L	0.23	ug/L	M	5.76E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.6E-07
										Total Risk Across All Exposure Routes/Pathways	8.6E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.5.1.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	21.46	mg/kg	21.46	mg/kg	M	2.02E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.0E-05
Dermal	Arsenic	21.46	mg/kg	21.46	mg/kg	M	2.31E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.5E-06
										Total Risk Across All Exposure Routes/Pathways	3.4E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.5.1.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	16.52	mg/kg	16.52	mg/kg	M	1.85E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.8E-06
Dermal	Arsenic	16.52	mg/kg	16.52	mg/kg	M	4.22E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.3E-07
										Total Risk Across All Exposure Routes/Pathways	3.4E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.5.2.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Silverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.545	ug/L	0.545	ug/L	M	8.11E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.2E-05
										Total Risk Across All Exposure Routes/Pathways	1.2E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.5.2.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Siverton - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.38	ug/L	0.38	ug/L	M	9.51E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.4E-06
										Total Risk Across All Exposure Routes/Pathways	1.4E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.5.3.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Silverton - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	28.44	mg/kg	28.44	mg/kg	M	1.70E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.6E-06
Dermal	Arsenic	28.44	mg/kg	28.44	mg/kg	M	5.77E-07	mg/kg-d	1.5	(mg/kg-d)-1	8.7E-07
										Total Risk Across All Exposure Routes/Pathways	3.4E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.5.3.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Silverton - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	16.88	mg/kg	16.88	mg/kg	M	5.78E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.7E-08
Dermal	Arsenic	16.88	mg/kg	16.88	mg/kg	M	4.89E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.3E-08
										Total Risk Across All Exposure Routes/Pathways	1.6E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.5.4.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Silverton - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	28.44	mg/kg	28.44	mg/kg	M	3.41E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	5.1E-06
Dermal	Arsenic	28.44	mg/kg	28.44	mg/kg	M	5.94E-07	mg/kg-d	1.5	(mg/kg-d)-1	8.9E-07
										Total Risk Across All Exposure Routes/Pathways	6.0E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.5.4.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Silverton - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	16.88	mg/kg	16.88	mg/kg	M	1.38E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.1E-07
Dermal	Arsenic	16.88	mg/kg	16.88	mg/kg	M	5.64E-08	mg/kg-d	1.5	(mg/kg-d)-1	8.5E-08
										Total Risk Across All Exposure Routes/Pathways	2.9E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.6.1.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	22.57	mg/kg	22.57	mg/kg	M	2.12E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.2E-05
Dermal	Arsenic	22.57	mg/kg	22.57	mg/kg	M	2.43E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.6E-06
										Total Risk Across All Exposure Routes/Pathways	3.5E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.6.1.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	20.32	mg/kg	20.32	mg/kg	M	2.27E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.4E-06
Dermal	Arsenic	20.32	mg/kg	20.32	mg/kg	M	5.19E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.8E-07
										Total Risk Across All Exposure Routes/Pathways	4.2E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.6.2.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.494	ug/L	0.494	ug/L	M	7.35E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.1E-05
										Total Risk Across All Exposure Routes/Pathways	1.1E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.6.2.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Wallace - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.42	ug/L	0.42	ug/L	M	1.05E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.6E-06
										Total Risk Across All Exposure Routes/Pathways	1.6E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.6.3.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Wallace - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	27.65	mg/kg	27.65	mg/kg	M	1.66E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.5E-06
Dermal	Arsenic	27.65	mg/kg	27.65	mg/kg	M	5.61E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.4E-07
										Total Risk Across All Exposure Routes/Pathways	3.3E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.6.3.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Wallace - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	25.8	mg/kg	25.8	mg/kg	M	8.83E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.3E-07
Dermal	Arsenic	25.8	mg/kg	25.8	mg/kg	M	7.47E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.1E-07
										Total Risk Across All Exposure Routes/Pathways	2.4E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.6.4.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Wallace - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	27.65	mg/kg	27.65	mg/kg	M	3.32E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	5.0E-06
Dermal	Arsenic	27.65	mg/kg	27.65	mg/kg	M	5.78E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.7E-07
										Total Risk Across All Exposure Routes/Pathways	5.8E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.6.4.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Wallace - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Upland parks/schools
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	25.8	mg/kg	25.8	mg/kg	M	2.10E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.2E-07
Dermal	Arsenic	25.8	mg/kg	25.8	mg/kg	M	8.62E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.3E-07
										Total Risk Across All Exposure Routes/Pathways	4.4E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.7.1.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Osburn-Wallace-Silverton - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	52.64	mg/kg	52.64	mg/kg	M	1.95E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.9E-06
Dermal	Arsenic	52.64	mg/kg	52.64	mg/kg	M	1.51E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.3E-06
										Total Risk Across All Exposure Routes/Pathways	5.2E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.7.1.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Osburn-Wallace-Silverton - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	36.81	mg/kg	36.81	mg/kg	M	7.41E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.1E-07
Dermal	Arsenic	36.81	mg/kg	36.81	mg/kg	M	1.51E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.3E-07
										Total Risk Across All Exposure Routes/Pathways	3.4E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.7.2.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Osburn-Wallace-Silverton - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	1.096	ug/L	1.096	ug/L	M	3.09E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.6E-08
										Total Risk Across All Exposure Routes/Pathways	4.6E-08

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.7.2.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Osburn-Wallace-Silverton - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.78	ug/L	0.78	ug/L	M	3.14E-09	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.7E-09
										Total Risk Across All Exposure Routes/Pathways	4.7E-09

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.7.3.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Osburn-Wallace-Silverton - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	27.31	mg/kg	27.31	mg/kg	M	1.34E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.0E-05
Dermal	Arsenic	27.31	mg/kg	27.31	mg/kg	M	5.58E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.4E-07
										Total Risk Across All Exposure Routes/Pathways	2.1E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.7.3.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Osburn-Wallace-Silverton - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	21.87	mg/kg	21.87	mg/kg	M	4.16E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.2E-07
Dermal	Arsenic	21.87	mg/kg	21.87	mg/kg	M	2.60E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.9E-08
										Total Risk Across All Exposure Routes/Pathways	6.6E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.1.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	41.62	mg/kg	41.62	mg/kg	M	3.91E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	5.9E-05
Dermal	Arsenic	41.62	mg/kg	41.62	mg/kg	M	4.48E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.7E-06
										Total Risk Across All Exposure Routes/Pathways	6.5E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.1.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	31.58	mg/kg	31.58	mg/kg	M	3.53E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	5.3E-06
Dermal	Arsenic	31.58	mg/kg	31.58	mg/kg	M	8.07E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.2E-06
										Total Risk Across All Exposure Routes/Pathways	6.5E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.2.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.74	ug/L	0.74	ug/L	M	1.10E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.7E-05
										Total Risk Across All Exposure Routes/Pathways	1.7E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.2.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.45	ug/L	0.45	ug/L	M	1.13E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.7E-06
										Total Risk Across All Exposure Routes/Pathways	1.7E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.3.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	1.25	ug/L	1.25	ug/L	M	1.86E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.8E-05
										Total Risk Across All Exposure Routes/Pathways	2.8E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.3.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Residential Exposure Scenario

Scenario Timeframe:	Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.81	ug/L	0.81	ug/L	M	2.03E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.0E-06
										Total Risk Across All Exposure Routes/Pathways	3.0E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.4.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Waste piles
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	518.49	mg/kg	518.49	mg/kg	M	1.55E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.3E-05
Dermal	Arsenic	518.49	mg/kg	518.49	mg/kg	M	5.26E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.9E-06
										Total Risk Across All Exposure Routes/Pathways	3.1E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.4.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Waste piles
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	95.57	mg/kg	95.57	mg/kg	M	1.64E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.5E-07
Dermal	Arsenic	95.57	mg/kg	95.57	mg/kg	M	1.38E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.1E-07
										Total Risk Across All Exposure Routes/Pathways	4.5E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.5.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Nine Mile/Canyon Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	50.62	mg/kg	50.62	mg/kg	M	1.87E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.8E-06
Dermal	Arsenic	50.62	mg/kg	50.62	mg/kg	M	1.45E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.2E-06
										Total Risk Across All Exposure Routes/Pathways	5.0E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.5.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	Nine Mile/Canyon Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	23.12	mg/kg	23.12	mg/kg	M	4.65E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.0E-08
Dermal	Arsenic	23.12	mg/kg	23.12	mg/kg	M	9.46E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.4E-07
										Total Risk Across All Exposure Routes/Pathways	2.1E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.6.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Nine Mile/Canyon Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	1.1	ug/L	1.1	ug/L	M	3.10E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.6E-08
										Total Risk Across All Exposure Routes/Pathways	4.6E-08

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.6.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Nine Mile/Canyon Creek
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.7	ug/L	0.7	ug/L	M	2.82E-09	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.2E-09
										Total Risk Across All Exposure Routes/Pathways	4.2E-09

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.7.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Nine Mile - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	24.97	mg/kg	24.97	mg/kg	M	1.23E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.8E-05
Dermal	Arsenic	24.97	mg/kg	24.97	mg/kg	M	5.10E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.7E-07
										Total Risk Across All Exposure Routes/Pathways	1.9E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.8.7.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Nine Mile - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	21.24	mg/kg	21.24	mg/kg	M	4.04E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.1E-07
Dermal	Arsenic	21.24	mg/kg	21.24	mg/kg	M	2.53E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.8E-08
										Total Risk Across All Exposure Routes/Pathways	6.4E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.1.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	39.04	mg/kg	39.04	mg/kg	M	3.67E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	5.5E-05
Dermal	Arsenic	39.04	mg/kg	39.04	mg/kg	M	4.20E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.3E-06
										Total Risk Across All Exposure Routes/Pathways	6.1E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.1.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Yard soil
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	27.3	mg/kg	27.3	mg/kg	M	3.06E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.6E-06
Dermal	Arsenic	27.3	mg/kg	27.3	mg/kg	M	6.97E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.0E-06
										Total Risk Across All Exposure Routes/Pathways	5.6E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.2.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.25	ug/L	0.25	ug/L	M	3.72E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	5.6E-06
										Total Risk Across All Exposure Routes/Pathways	5.6E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.2.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Mullan - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Groundwater
Exposure Medium:	Groundwater
Exposure Point:	Tap water
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.13	ug/L	0.13	ug/L	M	3.25E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.9E-07
										Total Risk Across All Exposure Routes/Pathways	4.9E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.3.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Waste piles
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	42.1	mg/kg	42.1	mg/kg	M	1.26E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.9E-06
Dermal	Arsenic	42.1	mg/kg	42.1	mg/kg	M	4.27E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.4E-07
										Total Risk Across All Exposure Routes/Pathways	2.5E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.3.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface soil
Exposure Point:	Waste piles
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	23.6	mg/kg	23.6	mg/kg	M	4.04E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.1E-08
Dermal	Arsenic	23.6	mg/kg	23.6	mg/kg	M	3.42E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	5.1E-08
										Total Risk Across All Exposure Routes/Pathways	1.1E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.4.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	15.89	mg/kg	15.89	mg/kg	M	5.88E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.8E-07
Dermal	Arsenic	15.89	mg/kg	15.89	mg/kg	M	4.55E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.8E-07
										Total Risk Across All Exposure Routes/Pathways	1.6E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.4.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Sediment
Exposure Medium:	Sediment
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	13.21	mg/kg	13.21	mg/kg	M	2.66E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.0E-08
Dermal	Arsenic	13.21	mg/kg	13.21	mg/kg	M	5.40E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.1E-08
										Total Risk Across All Exposure Routes/Pathways	1.2E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.5.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.6	ug/L	0.6	ug/L	M	1.69E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.5E-08
										Total Risk Across All Exposure Routes/Pathways	2.5E-08

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.5.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Mullan - Neighborhood Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	SFCDAR
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.54	ug/L	0.54	ug/L	M	2.17E-09	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.3E-09
										Total Risk Across All Exposure Routes/Pathways	3.3E-09

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.6.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Mullan - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	31.66	mg/kg	31.66	mg/kg	M	1.55E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	2.3E-05
Dermal	Arsenic	31.66	mg/kg	31.66	mg/kg	M	6.47E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	9.7E-07
										Total Risk Across All Exposure Routes/Pathways	2.4E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.9.6.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Mullan - Occupational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Surface/subsurface soil
Exposure Point:	Construction site soil
Receptor Population:	Construction worker
Receptor Age:	Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	26.48	mg/kg	26.48	mg/kg	M	5.04E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	7.6E-07
Dermal	Arsenic	26.48	mg/kg	26.48	mg/kg	M	3.15E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.7E-08
										Total Risk Across All Exposure Routes/Pathways	8.0E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.10.1.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Spokane River
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	50	mg/kg	50	mg/kg	M	5.80E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.7E-06
Dermal	Arsenic	50	mg/kg	50	mg/kg	M	2.14E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	3.2E-06
										Total Risk Across All Exposure Routes/Pathways	1.2E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.10.1.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil/sediment
Exposure Medium:	Soil/sediment
Exposure Point:	Spokane River
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	41.03	mg/kg	41.03	mg/kg	M	3.24E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.9E-07
Dermal	Arsenic	41.03	mg/kg	41.03	mg/kg	M	2.72E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	4.1E-07
										Total Risk Across All Exposure Routes/Pathways	8.9E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.10.2.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Spokane River
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	20.8	ug/L	20.8	ug/L	M	5.81E-07	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.7E-07
										Total Risk Across All Exposure Routes/Pathways	8.7E-07

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.10.2.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
Blackwell Island - Public Recreational Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Surface water
Exposure Medium:	Surface water
Exposure Point:	Spokane River
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	10.56	ug/L	10.56	ug/L	M	4.63E-08	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	6.9E-08
										Total Risk Across All Exposure Routes/Pathways	6.9E-08

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.11.1.RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
All Areas Combined - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Plant Tissue
Exposure Point:	Homegrown vegetables
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.025	mg/kg	0.025	mg/kg	M	5.40E-05	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	8.1E-05
										Total Risk Across All Exposure Routes/Pathways	8.1E-05

(1) M = Medium-Specific, R = Route-Specific

TABLE 8.11.1.CT
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
All Areas Combined - Residential Exposure Scenario

Scenario Timeframe:	Current/Future
Medium:	Soil
Exposure Medium:	Plant Tissue
Exposure Point:	Homegrown vegetables
Receptor Population:	Residents
Receptor Age:	Child/Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Arsenic	0.02	mg/kg	0.02	mg/kg	M	1.27E-06	mg/kg-d	1.5	$(mg/kg\text{-}d)^{-1}$	1.9E-06
										Total Risk Across All Exposure Routes/Pathways	1.9E-06

(1) M = Medium-Specific, R = Route-Specific

TABLE 9.1.1.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	2.6E-01	N/A	N/A	2.6E-01
								Arsenic	NOAEL/skin pigmentation	1.2E+00	N/A	1.4E-01	1.3E+00
								Cadmium	NOAEL/proteinuria--kidney	8.8E-02	N/A	7.7E-03	9.6E-02
								Iron	hematology--blood	1.6E+00	N/A	N/A	1.6E+00
								Manganese	NOAEL/Central Nervous System	6.2E-01	N/A	N/A	6.2E-01
								Zinc	enzyme effects--blood	5.1E-02	N/A	N/A	5.1E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		3.9E+00	N/A	1.4E-01	4.0E+00
Groundwater	Groundwater	Tap water	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	2.1E-01	N/A	N/A	2.1E-01

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	4.2E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.1.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	6.7E-02	N/A	N/A	6.7E-02
								Arsenic	NOAEL/skin pigmentation	3.4E-01	N/A	7.4E-02	4.1E-01
								Cadmium	NOAEL/proteinuria--kidney	2.4E-02	N/A	4.2E-03	2.8E-02
								Iron	hematology--blood	4.7E-01	N/A	N/A	4.7E-01
								Manganese	NOAEL/Central Nervous System	1.7E-01	N/A	N/A	1.7E-01
								Zinc	enzyme effects--blood	1.3E-02	N/A	N/A	1.3E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.1E+00	N/A	7.8E-02	1.2E+00
Groundwater	Groundwater	Tap water											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	5.4E-02	N/A	N/A	5.4E-02

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	1.2E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.2.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe: Current/Future
Receptor Population: Residents
Receptor Age: Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	6.8E-05	N/A	7.8E-06	7.6E-05	Antimony	longevity, glucose/ cholesterol--blood	7.5E-02	N/A	N/A	7.5E-02
								Arsenic	NOAEL/skin pigmentation	3.5E-01	N/A	4.1E-02	3.9E-01
								Cadmium	NOAEL/proteinuria--kidney	2.5E-02	N/A	2.3E-03	2.7E-02
								Iron	hematology--blood	4.6E-01	N/A	N/A	4.6E-01
								Manganese	NOAEL/Central Nervous System	1.8E-01	N/A	N/A	1.8E-01
								Zinc	enzyme effects--blood	1.5E-02	N/A	N/A	1.5E-02
			(Total)	6.8E-05	N/A	7.8E-06	7.6E-05	(Total)		1.1E+00	N/A	4.3E-02	1.1E+00
Groundwater	Groundwater	Tap water	Arsenic	2.2E-05	N/A	N/A	2.2E-05	Arsenic	NOAEL/skin pigmentation	1.1E-01	N/A	N/A	1.1E-01
						Total Risk Across Soil	7.6E-05					Total Hazard Index Across All Media and All Exposure Routes	1.3E+00
						Total Risk Across Groundwater	2.2E-05						
						Total Risk Across All Media and All Exposure Routes	9.8E-05						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.2.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	6.0E-06	N/A	1.4E-06	7.3E-06	Antimony	longevity, glucose/ cholesterol--blood	2.0E-02	N/A	N/A	2.0E-02
								Arsenic	NOAEL/skin pigmentation	1.0E-01	N/A	2.3E-02	1.2E-01
								Cadmium	NOAEL/proteinuria--kidney	7.2E-03	N/A	1.3E-03	8.5E-03
								Iron	hematology--blood	1.5E-01	N/A	N/A	1.5E-01
								Manganese	NOAEL/Central Nervous System	5.1E-02	N/A	N/A	5.1E-02
								Zinc	enzyme effects--blood	4.1E-03	N/A	N/A	4.1E-03
			(Total)	6.0E-06	N/A	1.4E-06	7.3E-06	(Total)		3.3E-01	N/A	2.5E-02	3.6E-01
Groundwater	Groundwater	Tap water	Arsenic	1.4E-06	N/A	N/A	1.4E-06	Arsenic	NOAEL/skin pigmentation	2.5E-02	N/A	N/A	2.5E-02

Total Risk Across Soil	7.3E-06
Total Risk Across Groundwater	1.4E-06
Total Risk Across All Media and All Exposure Routes	8.7E-06
Total Hazard Index Across All Media and All Exposure Routes	3.8E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.3.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Lower CDAR	Arsenic	6.6E-06	N/A	8.0E-06	1.5E-05	Antimony	longevity, glucose/ cholesterol--blood	3.6E-02	N/A	N/A	3.6E-02
								Arsenic	NOAEL/skin pigmentation	1.5E-01	N/A	1.8E-01	3.3E-01
								Cadmium	NOAEL/proteinuria--kidney	2.2E-02	N/A	2.1E-02	4.3E-02
								Iron	hematology--blood	2.2E-01	N/A	N/A	2.2E-01
								Manganese	NOAEL/Central Nervous System	1.3E-01	N/A	N/A	1.3E-01
								Zinc	enzyme effects--blood	1.2E-02	N/A	N/A	1.2E-02
			(Total)	6.6E-06	N/A	8.0E-06	1.5E-05	(Total)		5.6E-01	N/A	2.0E-01	7.6E-01
Surface Water	Disturbed surface water	Lower CDAR	Arsenic	3.4E-06	N/A	N/A	3.4E-06	Arsenic	NOAEL/skin pigmentation	7.5E-02	N/A	N/A	7.5E-02
								Cadmium	NOAEL/proteinuria--kidney	4.1E-02	N/A	N/A	4.1E-02
								Manganese	NOAEL/Central Nervous System	1.0E-01	N/A	N/A	1.0E-01
								Mercury	autoimmunity	4.2E-03	N/A	N/A	4.2E-03
			(Total)	3.4E-06	N/A	N/A	3.4E-06	(Total)		2.2E-01	N/A	N/A	2.2E-01

Total Risk Across Soil/sediment	1.5E-05
Total Risk Across Surface water	3.4E-06
Total Risk Across All Media and All Exposure Routes	1.8E-05
Total Hazard Index Across All Media and All Exposure Routes	9.8E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.3.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Lower CDAR	Arsenic	3.5E-07	N/A	1.1E-06	1.4E-06	Antimony	longevity, glucose/ cholesterol--blood	6.5E-03	N/A	N/A	6.5E-03
								Arsenic	NOAEL/skin pigmentation	2.7E-02	N/A	8.6E-02	1.1E-01
								Cadmium	NOAEL/proteinuria--kidney	3.9E-03	N/A	9.9E-03	1.4E-02
								Iron	hematology--blood	4.0E-02	N/A	N/A	4.0E-02
								Manganese	NOAEL/Central Nervous System	2.3E-02	N/A	N/A	2.3E-02
								Zinc	enzyme effects--blood	2.1E-03	N/A	N/A	2.1E-03
			(Total)	3.5E-07	N/A	1.1E-06	1.4E-06	(Total)		1.0E-01	N/A	9.6E-02	2.0E-01
Surface Water	Disturbed surface water	Lower CDAR	Arsenic	4.6E-07	N/A	N/A	4.6E-07	Arsenic	NOAEL/skin pigmentation	3.6E-02	N/A	N/A	3.6E-02
								Cadmium	NOAEL/proteinuria--kidney	1.5E-02	N/A	N/A	1.5E-02
								Manganese	NOAEL/Central Nervous System	6.4E-02	N/A	N/A	6.4E-02
								Mercury	autoimmunity	2.1E-03	N/A	N/A	2.1E-03
			(Total)	4.6E-07	N/A	N/A	4.6E-07	(Total)		1.2E-01	N/A	N/A	1.2E-01

Total Risk Across Soil/sediment	1.4E-06
Total Risk Across Surface water	4.6E-07
Total Risk Across All Media and All Exposure Routes	1.9E-06
Total Hazard Index Across All Media and All Exposure Routes	3.1E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.4.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Lower CDAR	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.0E-01	N/A	N/A	1.0E-01
								Arsenic	NOAEL/skin pigmentation	4.2E-01	N/A	9.1E-02	5.1E-01
								Cadmium	NOAEL/proteinuria--kidney	6.1E-02	N/A	1.1E-02	7.2E-02
								Iron	hematology--blood	6.2E-01	N/A	N/A	6.2E-01
								Manganese	NOAEL/Central Nervous System	3.7E-01	N/A	N/A	3.7E-01
								Zinc	enzyme effects--blood	3.3E-02	N/A	N/A	3.3E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.6E+00	N/A	1.0E-01	1.7E+00
Surface Water	Disturbed surface water	Lower CDAR	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	4.7E-02	N/A	N/A	4.7E-02
								Cadmium	NOAEL/proteinuria--kidney	2.6E-02	N/A	N/A	2.6E-02
								Manganese	NOAEL/Central Nervous System	6.2E-02	N/A	N/A	6.2E-02
								Mercury	autoimmunity	2.6E-03	N/A	N/A	2.6E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.4E-01	N/A	N/A	1.4E-01

Total Risk Across Soil/sediment	N/A
Total Risk Across Surface water	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	1.8E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.4.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Lower CDAR	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.9E-02	N/A	N/A	1.9E-02
								Arsenic	NOAEL/skin pigmentation	8.0E-02	N/A	4.4E-02	1.2E-01
								Cadmium	NOAEL/proteinuria--kidney	1.2E-02	N/A	5.0E-03	1.7E-02
								Iron	hematology--blood	1.2E-01	N/A	N/A	1.2E-01
								Manganese	NOAEL/Central Nervous System	7.0E-02	N/A	N/A	7.0E-02
								Zinc	enzyme effects--blood	6.2E-03	N/A	N/A	6.2E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		3.1E-01	N/A	4.9E-02	3.5E-01
Surface Water	Disturbed surface water	Lower CDAR	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	2.2E-02	N/A	N/A	2.2E-02
								Cadmium	NOAEL/proteinuria--kidney	9.6E-03	N/A	N/A	9.6E-03
								Manganese	NOAEL/Central Nervous System	4.0E-02	N/A	N/A	4.0E-02
								Mercury	autoimmunity	1.3E-03	N/A	N/A	1.3E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		7.3E-02	N/A	N/A	7.3E-02

Total Risk Across Soil/sediment	N/A
Total Risk Across Surface water	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	4.3E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.5.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Lower CDAR	Arsenic	2.1E-05	N/A	7.7E-06	2.8E-05	Antimony	longevity, glucose/ cholesterol--blood	2.6E-02	N/A	N/A	2.6E-02
								Arsenic	NOAEL/skin pigmentation	1.1E-01	N/A	4.0E-02	1.5E-01
								Cadmium	NOAEL/proteinuria--kidney	1.6E-02	N/A	4.7E-03	2.0E-02
								Iron	hematology--blood	1.6E-01	N/A	N/A	1.6E-01
								Manganese	NOAEL/Central Nervous System	9.5E-02	N/A	N/A	9.5E-02
								Zinc	enzyme effects--blood	8.5E-03	N/A	N/A	8.5E-03
			(Total)	2.1E-05	N/A	7.7E-06	2.8E-05	(Total)		4.1E-01	N/A	4.4E-02	4.6E-01
Surface Water	Disturbed surface water	Lower CDAR	Arsenic	3.3E-06	N/A	N/A	3.3E-06	Arsenic	NOAEL/skin pigmentation	1.7E-02	N/A	N/A	1.7E-02
								Cadmium	NOAEL/proteinuria--kidney	9.5E-03	N/A	N/A	9.5E-03
								Manganese	NOAEL/Central Nervous System	2.3E-02	N/A	N/A	2.3E-02
								Mercury	autoimmunity	9.8E-04	N/A	N/A	9.8E-04
			(Total)	3.3E-06	N/A	N/A	3.3E-06	(Total)		5.1E-02	N/A	N/A	5.1E-02

Total Risk Across Soil/sediment	2.8E-05
Total Risk Across Surface water	3.3E-06
Total Risk Across All Media and All Exposure Routes	3.2E-05
Total Hazard Index Across All Media and All Exposure Routes	5.1E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.5.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Lower CDAR	Arsenic	1.4E-06	N/A	1.1E-06	2.5E-06	Antimony	longevity, glucose/ cholesterol--blood	5.6E-03	N/A	N/A	5.6E-03
								Arsenic	NOAEL/skin pigmentation	2.3E-02	N/A	2.0E-02	4.3E-02
								Cadmium	NOAEL/proteinuria--kidney	3.4E-03	N/A	2.3E-03	5.7E-03
								Iron	hematology--blood	3.5E-02	N/A	N/A	3.5E-02
								Manganese	NOAEL/Central Nervous System	2.0E-02	N/A	N/A	2.0E-02
								Zinc	enzyme effects--blood	1.8E-03	N/A	N/A	1.8E-03
			(Total)	1.4E-06	N/A	1.1E-06	2.5E-06	(Total)		8.9E-02	N/A	2.2E-02	1.1E-01
Surface Water	Disturbed surface water	Lower CDAR	Arsenic	5.0E-07	N/A	N/A	5.0E-07	Arsenic	NOAEL/skin pigmentation	8.6E-03	N/A	N/A	8.6E-03
								Cadmium	NOAEL/proteinuria--kidney	3.7E-03	N/A	N/A	3.7E-03
								Manganese	NOAEL/Central Nervous System	1.5E-02	N/A	N/A	1.5E-02
								Mercury	autoimmunity	5.1E-04	N/A	N/A	5.1E-04
			(Total)	5.0E-07	N/A	N/A	5.0E-07	(Total)		2.8E-02	N/A	N/A	2.8E-02

Total Risk Across Soil/sediment	2.5E-06
Total Risk Across Surface water	5.0E-07
Total Risk Across All Media and All Exposure Routes	3.0E-06
Total Hazard Index Across All Media and All Exposure Routes	1.4E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.6.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Construction Workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	8.0E-05	N/A	3.3E-06	8.3E-05	Antimony	longevity, glucose/ cholesterol--blood	1.2E-01	N/A	N/A	1.2E-01
								Arsenic	NOAEL/skin pigmentation	5.0E-01	N/A	2.1E-02	5.2E-01
								Cadmium	NOAEL/proteinuria--kidney	7.0E-02	N/A	2.3E-03	7.3E-02
								Iron	hematology--blood	7.4E-01	N/A	N/A	7.4E-01
								Manganese	NOAEL/Central Nervous System	1.5E-01	N/A	N/A	1.5E-01
								Zinc	enzyme effects--blood	3.8E-02	N/A	N/A	3.8E-02
			(Total)	8.0E-05	N/A	3.3E-06	8.3E-05	(Total)		1.6E+00	N/A	2.3E-02	1.6E+00
						Total Risk Across Soil	8.3E-05					Total Hazard Index Across All Media and All Exposure Routes	1.6E+00
						Total Risk Across All Media and All Exposure Routes	8.3E-05						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.6.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Construction workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	3.0E-06	N/A	1.9E-07	3.2E-06	Antimony	longevity, glucose/ cholesterol--blood	1.7E-02	N/A	N/A	1.7E-02
								Arsenic	NOAEL/skin pigmentation	7.0E-02	N/A	4.4E-03	7.5E-02
								Cadmium	NOAEL/proteinuria--kidney	9.8E-03	N/A	4.9E-04	1.0E-02
								Iron	hematology--blood	1.0E-01	N/A	N/A	1.0E-01
								Manganese	NOAEL/Central Nervous System	2.0E-02	N/A	N/A	2.0E-02
								Zinc	enzyme effects--blood	5.3E-03	N/A	N/A	5.3E-03
			(Total)	3.0E-06	N/A	1.9E-07	3.2E-06	(Total)		2.3E-01	N/A	4.9E-03	2.3E-01
						Total Risk Across Soil	3.2E-06					Total Hazard Index Across All Media and All Exposure Routes	2.3E-01
						Total Risk Across All Media and All Exposure Routes	3.2E-06						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.7.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water/Sediment	Plant Tissue	Water Potato (With Skin)	N/A	N/A	N/A	N/A	N/A	Cadmium	NOAEL/proteinuria--kidney	8.0E-01	N/A	N/A	8.0E-01
			(Total)	N/A	N/A	N/A	N/A	(Total)		8.0E-01	N/A	0.0E+00	8.0E-01
				Total Risk Across All Media and All Exposure Routes			N/A		Total Hazard Index Across All Media and All Exposure Routes				8.0E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.8.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Lower CDAR	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.7E-01	N/A	N/A	1.7E-01
								Arsenic	NOAEL/skin pigmentation	8.2E-01	N/A	3.1E-01	1.1E+00
								Cadmium	NOAEL/proteinuria--kidney	1.0E-01	N/A	3.0E-02	1.3E-01
								Iron	hematology--blood	1.1E+00	N/A	N/A	1.1E+00
								Manganese	NOAEL/Central Nervous System	6.3E-01	N/A	N/A	6.3E-01
								Zinc	enzyme effects--blood	5.3E-02	N/A	N/A	5.3E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		2.8E+00	N/A	3.4E-01	3.2E+00
Sediment	Sediment	Lower CDAR	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	2.1E-01	N/A	N/A	2.1E-01
								Arsenic	NOAEL/skin pigmentation	8.0E-01	N/A	6.9E-01	1.5E+00
								Cadmium	NOAEL/proteinuria--kidney	1.3E-01	N/A	9.0E-02	2.2E-01
								Iron	hematology--blood	1.2E+00	N/A	N/A	1.2E+00
								Manganese	NOAEL/Central Nervous System	7.5E-01	N/A	N/A	7.5E-01
								Zinc	enzyme effects--blood	7.0E-02	N/A	N/A	7.0E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		3.2E+00	N/A	7.8E-01	4.0E+00

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Undisturbed Surface Water	Udisturbed Surface Water	Lower CDAR											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.1E+00	N/A	N/A	1.1E+00
								Cadmium	NOAEL/proteinuria--kidney	1.1E-01	N/A	N/A	1.1E-01
								Manganese	NOAEL/Central Nervous System	1.3E-01	N/A	N/A	1.3E-01
								Mercury		1.4E-01	N/A	N/A	1.4E-01
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.5E+00	N/A	N/A	1.5E+00
Disturbed Surface Water	Disturbed Surface Water	Lower CDAR											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	8.72E-02	N/A	N/A	8.7E-02
								Cadmium	NOAEL/proteinuria--kidney	4.8E-02	N/A	N/A	4.8E-02
								Manganese	NOAEL/Central Nervous System	1.2E-01	N/A	N/A	1.2E-01
								Mercury		4.9E-03	N/A	N/A	4.9E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		2.6E-01	N?A	N/A	2.6E-01
							Total Risk Across Soil						Total Risk Across Soil
							N/A						3.2E+00
							Total Risk Across Sediment						Total Risk Across Sediment
							N/A						4.0E+00
							Total Risk Across Undisturbed Surface Water						Total Risk Across Undisturbed Surface Water
							N/A						1.5E+00
							Total Risk Across Disturbed Surface Water						Total Risk Across Disturbed Surface Water
							N/A						2.6E-01
							Total Risk Across All Media and All Exposure Routes						Total Risk Across All Media and All Exposure Routes
							N/A						8.6E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.9.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin - Modern Subsistence Exposure Scenario

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Lower CDAR											
			Arsenic	1.0E-04	N/A	6.7E-05	1.7E-04	Antimony	longevity, glucose/ cholesterol--blood	4.9E-02	N/A	N/A	4.9E-02
								Arsenic	NOAEL/skin pigmentation	2.3E-01	N/A	1.5E-01	3.8E-01
								Cadmium	NOAEL/proteinuria--kidney	2.8E-02	N/A	1.4E-02	4.3E-02
								Iron	hematology--blood	3.0E-01	N/A	N/A	3.0E-01
								Manganese	NOAEL/Central Nervous System	1.8E-01	N/A	N/A	1.8E-01
								Zinc	enzyme effects--blood	1.5E-02	N/A	N/A	1.5E-02
			(Total)	1.0E-04	N/A	6.7E-05	1.7E-04	(Total)		8.0E-01	N/A	1.6E-01	9.6E-01
Sediment	Sediment	Lower CDAR											
			Arsenic	1.0E-04	N/A	1.9E-04	3.0E-04	Antimony	longevity, glucose/ cholesterol--blood	5.8E-02	N/A	N/A	5.8E-02
								Arsenic	NOAEL/skin pigmentation	2.2E-01	N/A	4.3E-01	6.5E-01
								Cadmium	NOAEL/proteinuria--kidney	3.6E-02	N/A	5.6E-02	9.3E-02
								Iron	hematology--blood	3.5E-01	N/A	N/A	3.5E-01
								Manganese	NOAEL/Central Nervous System	2.1E-01	N/A	N/A	2.1E-01
								Zinc	enzyme effects--blood	2.0E-02	N/A	N/A	2.0E-02
			(Total)	1.0E-04	N/A	1.9E-04	3.0E-04	(Total)		9.0E-01	N/A	4.9E-01	1.4E+00

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Undisturbed Surface Water	Udisturbed Surface Water	Lower CDAR											
			Arsenic	2.4E-04	N/A	N/A	2.4E-04	Arsenic	NOAEL/skin pigmentation	5.2E-01	N/A	N/A	5.2E-01
								Cadmium	NOAEL/proteinuria--kidney	5.1E-02	N/A	N/A	5.1E-02
								Manganese	NOAEL/Central Nervous System	6.3E-02	N/A	N/A	6.3E-02
								Mercury		6.5E-02	N/A	N/A	6.5E-02
			(Total)	2.36E-04	N/A	N/A	2.4E-04	(Total)		7.0E-01	N/A	N/A	7.0E-01
Disturbed Surface Water	Disturbed Surface Water	Lower CDAR											
			Arsenic	1.11E-05	N/A	N/A	1.1E-05	Arsenic	NOAEL/skin pigmentation	2.46E-02	N/A	N/A	2.5E-02
								Cadmium	NOAEL/proteinuria--kidney	1.3E-02	N/A	N/A	1.3E-02
								Manganese	NOAEL/Central Nervous System	3.3E-02	N/A	N/A	3.3E-02
								Mercury		1.4E-03	N/A	N/A	1.4E-03
			(Total)	1.1E-05	N/A	N/A	1.1E-05	(Total)		7.2E-02	N/A	N/A	7.2E-02
						Total Risk Across Soil	1.7E-04					Total Risk Across Soil	9.6E-01
						Total Risk Across Sediment	3.0E-04					Total Risk Across Sediment	1.4E+00
						Total Risk Across Undisturbed Surface Water	2.4E-04					Total Risk Across Undisturbed Surface Water	7.0E-01
						Total Risk Across Disturbed Surface Water	1.1E-05					Total Risk Across Disturbed Surface Water	7.2E-02
						Total Risk Across All Media and All Exposure Routes	7.1E-04					Total Risk Across All Media and All Exposure Routes	3.1E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.10.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water/Sediment	Plant Tissue	Water Potato (With Skin)	N/A	N/A	N/A	N/A	N/A	Cadmium	NOAEL/proteinuria--kidney	4.0E+00	N/A	N/A	4.0E+00
			(Total)	N/A	N/A	N/A	N/A	(Total)		4.0E+00	N/A	0.0E+00	4.0E+00
						Total Risk Across All Media and All Exposure Routes	N/A					Total Hazard Index Across All Media and All Exposure Routes	4.0E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.11.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Lower CDAR	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.1E+00	N/A	N/A	1.1E+00
								Arsenic	NOAEL/skin pigmentation	5.0E+00	N/A	1.5E+00	6.5E+00
								Cadmium	NOAEL/proteinuria--kidney	6.1E-01	N/A	1.4E-01	7.5E-01
								Iron	hematology--blood	6.5E+00	N/A	N/A	6.5E+00
								Manganese	NOAEL/Central Nervous System	3.8E+00	N/A	N/A	3.8E+00
								Zinc	enzyme effects--blood	3.2E-01	N/A	N/A	3.2E-01
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.7E+01	N/A	1.6E+00	1.9E+01
Sediment	Sediment	Lower CDAR	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	7.2E-01	N/A	N/A	7.2E-01
								Arsenic	NOAEL/skin pigmentation	2.8E+00	N/A	2.4E+00	5.2E+00
								Cadmium	NOAEL/proteinuria--kidney	4.5E-01	N/A	3.1E-01	7.7E-01
								Iron	hematology--blood	4.3E+00	N/A	N/A	4.3E+00
								Manganese	NOAEL/Central Nervous System	2.6E+00	N/A	N/A	2.6E+00
								Zinc	enzyme effects--blood	2.5E-01	N/A	N/A	2.5E-01
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.1E+01	N/A	2.7E+00	1.4E+01

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Undisturbed Surface Water	Udisturbed Surface Water	Lower CDAR	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	6.7E+00	N/A	N/A	6.7E+00
								Cadmium	NOAEL/proteinuria--kidney	6.5E-01	N/A	N/A	6.5E-01
								Manganese	NOAEL/Central Nervous System	8.1E-01	N/A	N/A	8.1E-01
								Mercury		8.3E-01	N/A	N/A	8.3E-01
			(Total)	N/A	N/A	N/A	N/A	(Total)		9.0E+00	N/A	N/A	9.0E+00
Disturbed Surface Water	Disturbed Surface Water	Lower CDAR	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	3.05E-01	N/A	N/A	3.1E-01
								Cadmium	NOAEL/proteinuria--kidney	1.7E-01	N/A	N/A	1.7E-01
								Manganese	NOAEL/Central Nervous System	4.1E-01	N/A	N/A	4.1E-01
								Mercury		1.7E-02	N/A	N/A	1.7E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		9.0E-01	N?A	N/A	9.0E-01
						Total Risk Across Soil	N/A					Total Risk Across Soil	1.9E+01
						Total Risk Across Sediment	N/A					Total Risk Across Sediment	1.4E+01
						Total Risk Across Undisturbed Surface Water	N/A					Total Risk Across Undisturbed Surface Water	9.0E+00
						Total Risk Across Disturbed Surface Water	N/A					Total Risk Across Disturbed Surface Water	9.0E-01
						Total Risk Across All Media and All Exposure Routes	N/A					Total Risk Across All Media and All Exposure Routes	4.3E+01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.1.12.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin - Traditional Subsistence Exposure Scenario

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Lower CDAR											
			Arsenic	6.3E-04	N/A	2.0E-04	8.3E-04	Antimony	longevity, glucose/ cholesterol--blood	3.0E-01	N/A	N/A	3.0E-01
								Arsenic	NOAEL/skin pigmentation	1.4E+00	N/A	4.5E-01	1.9E+00
								Cadmium	NOAEL/proteinuria--kidney	1.7E-01	N/A	4.4E-02	2.2E-01
								Iron	hematology--blood	1.8E+00	N/A	N/A	1.8E+00
								Manganese	NOAEL/Central Nervous System	1.1E+00	N/A	N/A	1.1E+00
								Zinc	enzyme effects--blood	9.1E-02	N/A	N/A	9.1E-02
			(Total)	6.3E-04	N/A	2.0E-04	8.3E-04	(Total)		4.9E+00	N/A	4.9E-01	5.4E+00
Sediment	Sediment	Lower CDAR											
			Arsenic	3.5E-04	N/A	6.8E-04	1.0E-03	Antimony	longevity, glucose/ cholesterol--blood	2.0E-01	N/A	N/A	2.0E-01
								Arsenic	NOAEL/skin pigmentation	7.8E-01	N/A	1.5E+00	2.3E+00
								Cadmium	NOAEL/proteinuria--kidney	1.3E-01	N/A	2.0E-01	3.2E-01
								Iron	hematology--blood	1.2E+00	N/A	N/A	1.2E+00
								Manganese	NOAEL/Central Nervous System	7.4E-01	N/A	N/A	7.4E-01
								Zinc	enzyme effects--blood	6.9E-02	N/A	N/A	6.9E-02
			(Total)	3.5E-04	N/A	6.8E-04	1.0E-03	(Total)		3.1E+00	N/A	1.7E+00	4.9E+00

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Undisturbed Surface Water	Udisturbed Surface Water	Lower CDAR	Arsenic	1.4E-03	N/A	N/A	1.4E-03	Arsenic	NOAEL/skin pigmentation	3.2E+00	N/A	N/A	3.2E+00
								Cadmium	NOAEL/proteinuria--kidney	3.1E-01	N/A	N/A	3.1E-01
								Manganese	NOAEL/Central Nervous System	3.9E-01	N/A	N/A	3.9E-01
								Mercury		4.0E-01	N/A	N/A	4.0E-01
			(Total)	1.43E-03	N/A	N/A	1.4E-03	(Total)		4.3E+00	N/A	N/A	4.3E+00
Disturbed Surface Water	Disturbed Surface Water	Lower CDAR	Arsenic	3.87E-05	N/A	N/A	3.9E-05	Arsenic	NOAEL/skin pigmentation	8.60E-02	N/A	N/A	8.6E-02
								Cadmium	NOAEL/proteinuria--kidney	4.7E-02	N/A	N/A	4.7E-02
								Manganese	NOAEL/Central Nervous System	1.1E-01	N/A	N/A	1.1E-01
								Mercury		4.9E-03	N/A	N/A	4.9E-03
			(Total)	3.9E-05	N/A	N/A	3.9E-05	(Total)		2.5E-01	N?A	N/A	2.5E-01
						Total Risk Across Soil	8.3E-04					Total Risk Across Soil	5.4E+00
						Total Risk Across Sediment	1.0E-03					Total Risk Across Sediment	4.9E+00
						Total Risk Across Undisturbed Surface Water	1.4E-03					Total Risk Across Undisturbed Surface Water	4.3E+00
						Total Risk Across Disturbed Surface Water	3.9E-05					Total Risk Across Disturbed Surface Water	2.5E-01
						Total Risk Across All Media and All Exposure Routes	3.3E-03					Total Hazard Index Across All Media and All Exposure Routes	1.5E+01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.2.1.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.3E-01	N/A	N/A	1.3E-01
								Arsenic	NOAEL/skin pigmentation	6.4E-01	N/A	7.0E-02	7.1E-01
								Cadmium	NOAEL/proteinuria--kidney	5.0E-02	N/A	4.4E-03	5.5E-02
								Iron	hematology--blood	9.4E-01	N/A	N/A	9.4E-01
								Manganese	NOAEL/Central Nervous System	3.0E-01	N/A	N/A	3.0E-01
								Zinc	enzyme effects--blood	3.1E-02	N/A	N/A	3.1E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		2.1E+00	N/A	7.5E-02	2.2E+00
Groundwater	Groundwater	Tap water	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	8.6E-02	N/A	N/A	8.6E-02

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	2.2E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.2.1.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	3.6E-02	N/A	N/A	3.6E-02
								Arsenic	NOAEL/skin pigmentation	1.9E-01	N/A	4.3E-02	2.4E-01
								Cadmium	NOAEL/proteinuria--kidney	1.7E-02	N/A	3.0E-03	2.0E-02
								Iron	hematology--blood	3.0E-01	N/A	N/A	3.0E-01
								Manganese	NOAEL/Central Nervous System	7.9E-02	N/A	N/A	7.9E-02
								Zinc	enzyme effects--blood	7.1E-03	N/A	N/A	7.1E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		6.4E-01	N/A	4.6E-02	6.8E-01
Groundwater	Groundwater	Tap water											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	3.0E-02	N/A	N/A	3.0E-02

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	7.1E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.2.2.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	3.5E-05	N/A	4.0E-06	3.9E-05	Antimony	longevity, glucose/ cholesterol--blood	3.7E-02	N/A	N/A	3.7E-02
								Arsenic	NOAEL/skin pigmentation	1.8E-01	N/A	2.1E-02	2.0E-01
								Cadmium	NOAEL/proteinuria--kidney	1.4E-02	N/A	1.3E-03	1.6E-02
								Iron	hematology--blood	2.7E-01	N/A	N/A	2.7E-01
								Manganese	NOAEL/Central Nervous System	8.4E-02	N/A	N/A	8.4E-02
								Zinc	enzyme effects--blood	8.8E-03	N/A	N/A	8.8E-03
			(Total)	3.5E-05	N/A	4.0E-06	3.9E-05	(Total)		5.9E-01	N/A	2.2E-02	6.2E-01
Groundwater	Groundwater	Tap water											
			Arsenic	9.0E-06	N/A	N/A	9.0E-06	Arsenic	NOAEL/skin pigmentation	4.7E-02	N/A	N/A	4.7E-02

Total Risk Across Soil	
Total Risk Across Groundwater	9.0E-06
Total Risk Across All Media and All Exposure Routes	4.8E-05

Total Hazard Index Across All Media and All Exposure Routes	6.6E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.2.2.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	3.4E-06	N/A	7.8E-07	4.2E-06	Antimony	longevity, glucose/ cholesterol--blood	1.1E-02	N/A	N/A	1.1E-02
								Arsenic	NOAEL/skin pigmentation	5.9E-02	N/A	1.3E-02	7.2E-02
								Cadmium	NOAEL/proteinuria--kidney	5.2E-03	N/A	9.5E-04	6.2E-03
								Iron	hematology--blood	9.3E-02	N/A	N/A	9.3E-02
								Manganese	NOAEL/Central Nervous System	2.4E-02	N/A	N/A	2.4E-02
								Zinc	enzyme effects--blood	2.2E-03	N/A	N/A	2.2E-03
			(Total)	3.4E-06	N/A	7.8E-07	4.2E-06	(Total)		1.9E-01	N/A	1.4E-02	2.1E-01
Groundwater	Groundwater	Tap water	Arsenic	7.9E-07	N/A	N/A	7.9E-07	Arsenic	NOAEL/skin pigmentation	1.4E-02	N/A	N/A	1.4E-02
						Total Risk Across Soil	4.2E-06					Total Hazard Index Across All Media and All Exposure Routes	2.2E-01
						Total Risk Across Groundwater	7.9E-07						
						Total Risk Across All Media and All Exposure Routes	5.0E-06						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.2.3.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	NS Confluence (2)											
			Arsenic	9.1E-06	N/A	1.1E-05	2.0E-05	Antimony	longevity, glucose/ cholesterol--blood	6.1E-02	N/A	N/A	6.1E-02
								Arsenic	NOAEL/skin pigmentation	2.0E-01	N/A	2.4E-01	4.4E-01
								Cadmium	NOAEL/proteinuria--kidney	1.6E-02	N/A	1.6E-02	3.2E-02
								Iron	hematology--blood	2.1E-01	N/A	N/A	2.1E-01
								Manganese	NOAEL/Central Nervous System	1.1E-01	N/A	N/A	1.1E-01
								Zinc	enzyme effects--blood	7.7E-03	N/A	N/A	7.7E-03
			(Total)	9.1E-06	N/A	1.1E-05	2.0E-05	(Total)		6.1E-01	N/A	2.6E-01	8.7E-01
Surface Water	Surface water	NS Confluence (2)											
			Arsenic	5.7E-06	N/A	N/A	5.7E-06	Arsenic	NOAEL/skin pigmentation	1.3E-01	N/A	N/A	1.3E-01
								Cadmium	NOAEL/proteinuria--kidney	4.7E-02	N/A	N/A	4.7E-02
								Manganese	NOAEL/Central Nervous System	5.7E-02	N/A	N/A	5.7E-02
								Mercury	autoimmunity	4.3E-03	N/A	N/A	4.3E-03
								(Total)		2.3E-01	N/A	N/A	2.3E-01

Total Risk Across Soil/sediment	2.0E-05
Total Risk Across Surface water	5.7E-06
Total Risk Across All Media and All Exposure Routes	2.6E-05
Total Hazard Index Across All Media and All Exposure Routes	1.1E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

(2) Both Pine Creek and NS Confluence were sampled and risks calculated. When determining total risk across media and across all media and exposure routes, it was assumed that all exposure occurred at the NS Confluence because it posed a greater risk tha Therefore, total risk estimates across media and across all media and exposure routes for this receptor scenario are higher (more conservative) than those associated with exposure to Pine Creek sediment and water.

N/A = not applicable

TABLE 9.2.3.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	NS Confluence (2)											
			Arsenic	4.4E-07	N/A	1.4E-06	1.8E-06	Antimony	longevity, glucose/ cholesterol--blood	1.0E-02	N/A	N/A	1.0E-02
								Arsenic	NOAEL/skin pigmentation	3.4E-02	N/A	1.1E-01	1.4E-01
								Cadmium	NOAEL/proteinuria--kidney	2.6E-03	N/A	6.6E-03	9.2E-03
								Iron	hematology--blood	3.5E-02	N/A	N/A	3.5E-02
								Manganese	NOAEL/Central Nervous System	1.9E-02	N/A	N/A	1.9E-02
								Zinc	enzyme effects--blood	1.3E-03	N/A	N/A	1.3E-03
			(Total)	4.4E-07	N/A	1.4E-06	1.8E-06	(Total)		1.0E-01	N/A	1.2E-01	2.2E-01
Surface Water	Surface water	NS Confluence (2)											
			Arsenic	2.2E-07	N/A	N/A	2.2E-07	Arsenic	NOAEL/skin pigmentation	1.7E-02	N/A	N/A	1.7E-02
								Cadmium	NOAEL/proteinuria--kidney	8.3E-03	N/A	N/A	8.3E-03
								Manganese	NOAEL/Central Nervous System	9.4E-03	N/A	N/A	9.4E-03
								Mercury	autoimmunity	6.1E-04	N/A	N/A	6.1E-04
			(Total)	2.2E-07	N/A	N/A	2.2E-07	(Total)		3.5E-02	N/A	N/A	3.5E-02
						Total Risk Across Soil/sediment	1.8E-06					Total Hazard Index Across All Media and All Exposure Routes	2.5E-01
						Total Risk Across Surface water	2.2E-07						
						Total Risk Across All Media and All Exposure Routes	2.1E-06						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

(2) Both Pine Creek and NS Confluence were sampled and risks calculated. When determining total risk across media and across all media and exposure routes, it was assumed that all exposure occurred at the NS Confluence because it posed a greater risk tha Therefore, total risk estimates across media and across all media and exposure routes for this receptor scenario are higher (more conservative) than those associated with exposure to Pine Creek sediment and water.

N/A = not applicable

TABLE 9.2.4.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	NS Confluence	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.7E-01	N/A	N/A	1.7E-01
								Arsenic	NOAEL/skin pigmentation	5.7E-01	N/A	1.2E-01	6.9E-01
								Cadmium	NOAEL/proteinuria--kidney	4.7E-02	N/A	8.1E-03	5.5E-02
								Iron	hematology--blood	5.9E-01	N/A	N/A	5.9E-01
								Manganese	NOAEL/Central Nervous System	3.2E-01	N/A	N/A	3.2E-01
								Zinc	enzyme effects--blood	2.2E-02	N/A	N/A	2.2E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.7E+00	N/A	1.3E-01	1.9E+00
Surface Water	Surface water	NS Confluence	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	7.8E-02	N/A	N/A	7.8E-02
								Cadmium	NOAEL/proteinuria--kidney	2.9E-02	N/A	N/A	2.9E-02
								Manganese	NOAEL/Central Nervous System	3.5E-02	N/A	N/A	3.5E-02
								Mercury	autoimmunity	2.7E-03	N/A	N/A	2.7E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.5E-01	N/A	N/A	1.5E-01

Total Risk Across Soil/sediment	N/A
Total Risk Across Surface water	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	2.0E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.2.4.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	NS Confluence	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	3.0E-02	N/A	N/A	3.0E-02
								Arsenic	NOAEL/skin pigmentation	1.0E-01	N/A	5.5E-02	1.6E-01
								Cadmium	NOAEL/proteinuria--kidney	7.7E-03	N/A	3.3E-03	1.1E-02
								Iron	hematology--blood	1.0E-01	N/A	N/A	1.0E-01
								Manganese	NOAEL/Central Nervous System	5.6E-02	N/A	N/A	5.6E-02
								Zinc	enzyme effects--blood	3.8E-03	N/A	N/A	3.8E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		3.0E-01	N/A	5.9E-02	3.6E-01
Surface Water	Surface water	NS Confluence	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.1E-02	N/A	N/A	1.1E-02
								Cadmium	NOAEL/proteinuria--kidney	5.2E-03	N/A	N/A	5.2E-03
								Manganese	NOAEL/Central Nervous System	5.9E-03	N/A	N/A	5.9E-03
								Mercury	autoimmunity	3.8E-04	N/A	N/A	3.8E-04
			(Total)	N/A	N/A	N/A	N/A	(Total)		2.2E-02	N/A	N/A	2.2E-02

Total Risk Across Soil/sediment	N/A
Total Risk Across Surface water	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	3.8E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.2.5.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	NS Confluence	Arsenic	2.8E-05	N/A	1.0E-05	3.9E-05	Antimony	longevity, glucose/ cholesterol--blood	4.5E-02	N/A	N/A	4.5E-02
								Arsenic	NOAEL/skin pigmentation	1.5E-01	N/A	5.4E-02	2.0E-01
								Cadmium	NOAEL/proteinuria--kidney	1.2E-02	N/A	3.5E-03	1.6E-02
								Iron	hematology--blood	1.5E-01	N/A	N/A	1.5E-01
								Manganese	NOAEL/Central Nervous System	8.2E-02	N/A	N/A	8.2E-02
								Zinc	enzyme effects--blood	5.6E-03	N/A	N/A	5.6E-03
			(Total)	2.8E-05	N/A	1.0E-05	3.9E-05	(Total)		4.4E-01	N/A	5.8E-02	5.0E-01
Surface Water	Surface water	NS Confluence	Arsenic	5.6E-06	N/A	N/A	5.6E-06	Arsenic	NOAEL/skin pigmentation	2.9E-02	N/A	N/A	2.9E-02
								Cadmium	NOAEL/proteinuria--kidney	1.1E-02	N/A	N/A	1.1E-02
								Manganese	NOAEL/Central Nervous System	1.3E-02	N/A	N/A	1.3E-02
								Mercury	autoimmunity	1.0E-03	N/A	N/A	1.0E-03
			(Total)	5.6E-06	N/A	N/A	5.6E-06	(Total)		5.4E-02	N/A	N/A	5.4E-02
						Total Risk Across Soil/sediment	3.9E-05					Total Hazard Index Across All Media and All Exposure Routes	5.6E-01
						Total Risk Across Surface water	5.6E-06						
						Total Risk Across All Media and All Exposure Routes	4.4E-05						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.2.5.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	NS Confluence	Arsenic	1.7E-06	N/A	1.4E-06	3.2E-06	Antimony	longevity, glucose/ cholesterol--blood	8.8E-03	N/A	N/A	8.8E-03
								Arsenic	NOAEL/skin pigmentation	3.0E-02	N/A	2.5E-02	5.5E-02
								Cadmium	NOAEL/proteinuria--kidney	2.3E-03	N/A	1.5E-03	3.8E-03
								Iron	hematology--blood	3.0E-02	N/A	N/A	3.0E-02
								Manganese	NOAEL/Central Nervous System	1.6E-02	N/A	N/A	1.6E-02
								Zinc	enzyme effects--blood	1.1E-03	N/A	N/A	1.1E-03
			(Total)	1.7E-06	N/A	1.4E-06	3.2E-06	(Total)		8.9E-02	N/A	2.7E-02	1.2E-01
Surface Water	Surface water	NS Confluence	Arsenic	2.4E-07	N/A	N/A	2.4E-07	Arsenic	NOAEL/skin pigmentation	4.1E-03	N/A	N/A	4.1E-03
								Cadmium	NOAEL/proteinuria--kidney	2.0E-03	N/A	N/A	2.0E-03
								Manganese	NOAEL/Central Nervous System	2.3E-03	N/A	N/A	2.3E-03
								Mercury	autoimmunity	1.5E-04	N/A	N/A	1.5E-04
			(Total)	2.4E-07	N/A	N/A	2.4E-07	(Total)		8.6E-03	N/A	N/A	8.6E-03
						Total Risk Across Soil/sediment	3.2E-06					Total Hazard Index Across All Media and All Exposure Routes	1.2E-01
						Total Risk Across Surface water	2.4E-07						
						Total Risk Across All Media and All Exposure Routes	3.4E-06						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.2.6.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Construction Workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	2.5E-05	N/A	1.1E-06	2.7E-05	Antimony	longevity, glucose/ cholesterol--blood	3.9E-02	N/A	N/A	3.9E-02
								Arsenic	NOAEL/skin pigmentation	1.6E-01	N/A	6.6E-03	1.7E-01
								Cadmium	NOAEL/proteinuria--kidney	1.2E-02	N/A	3.9E-04	1.2E-02
								Iron	hematology--blood	2.2E-01	N/A	N/A	2.2E-01
								Manganese	NOAEL/Central Nervous System	2.8E-02	N/A	N/A	2.8E-02
								Zinc	enzyme effects--blood	8.1E-03	N/A	N/A	8.1E-03
			(Total)	2.5E-05	N/A	1.1E-06	2.7E-05	(Total)		4.7E-01	N/A	7.0E-03	4.7E-01
						Total Risk Across Soil	2.7E-05					Total Hazard Index Across All Media and All Exposure Routes	4.7E-01
						Total Risk Across All Media and All Exposure Routes	2.7E-05						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.2.6.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Construction workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	8.3E-07	N/A	5.2E-08	8.8E-07	Antimony	longevity, glucose/ cholesterol--blood	4.6E-03	N/A	N/A	4.6E-03
								Arsenic	NOAEL/skin pigmentation	2.0E-02	N/A	1.2E-03	2.1E-02
								Cadmium	NOAEL/proteinuria--kidney	1.7E-03	N/A	8.7E-05	1.8E-03
								Iron	hematology--blood	2.9E-02	N/A	N/A	2.9E-02
								Manganese	NOAEL/Central Nervous System	3.3E-03	N/A	N/A	3.3E-03
								Zinc	enzyme effects--blood	8.9E-04	N/A	N/A	8.9E-04
			(Total)	8.3E-07	N/A	5.2E-08	8.8E-07	(Total)		5.9E-02	N/A	1.3E-03	6.0E-02
						Total Risk Across Soil	8.8E-07					Total Hazard Index Across All Media and All Exposure Routes	6.0E-02
						Total Risk Across All Media and All Exposure Routes	8.8E-07						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.3.1.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Side Gulches

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	5.1E-01	N/A	N/A	5.1E-01
								Arsenic	NOAEL/skin pigmentation	1.3E+00	N/A	1.4E-01	1.4E+00
								Cadmium	NOAEL/proteinuria--kidney	1.2E-01	N/A	1.1E-02	1.3E-01
								Iron	hematology--blood	1.2E+00	N/A	N/A	1.2E+00
								Manganese	NOAEL/Central Nervous System	3.7E-01	N/A	N/A	3.7E-01
								Zinc	enzyme effects--blood	5.6E-02	N/A	N/A	5.6E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		3.5E+00	N/A	1.5E-01	3.7E+00
Groundwater	Groundwater	Tap water											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.8E+00	N/A	N/A	1.8E+00

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	5.5E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.3.1.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Side Gulches

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.2E-01	N/A	N/A	1.2E-01
								Arsenic	NOAEL/skin pigmentation	3.8E-01	N/A	8.3E-02	4.6E-01
								Cadmium	NOAEL/proteinuria--kidney	3.3E-02	N/A	5.8E-03	3.9E-02
								Iron	hematology--blood	3.9E-01	N/A	N/A	3.9E-01
								Manganese	NOAEL/Central Nervous System	1.2E-01	N/A	N/A	1.2E-01
								Zinc	enzyme effects--blood	1.3E-02	N/A	N/A	1.3E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.1E+00	N/A	8.9E-02	1.1E+00
Groundwater	Groundwater	Tap water											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	2.7E-01	N/A	N/A	2.7E-01

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	1.4E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.3.2.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Side Gulches

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	7.1E-05	N/A	8.2E-06	8.0E-05	Antimony	longevity, glucose/ cholesterol--blood	1.5E-01	N/A	N/A	1.5E-01
								Arsenic	NOAEL/skin pigmentation	3.7E-01	N/A	4.2E-02	4.1E-01
								Cadmium	NOAEL/proteinuria--kidney	3.5E-02	N/A	3.2E-03	3.8E-02
								Iron	hematology--blood	3.3E-01	N/A	N/A	3.3E-01
								Manganese	NOAEL/Central Nervous System	1.1E-01	N/A	N/A	1.1E-01
								Zinc	enzyme effects--blood	1.6E-02	N/A	N/A	1.6E-02
			(Total)	7.1E-05	N/A	8.2E-06	8.0E-05	(Total)		1.0E+00	N/A	4.6E-02	1.1E+00
Groundwater	Groundwater	Tap water											
			Arsenic	1.9E-04	N/A	N/A	1.9E-04	Arsenic	NOAEL/skin pigmentation	9.7E-01	N/A	N/A	9.7E-01

Total Risk Across Soil	8.0E-05
Total Risk Across Groundwater	1.9E-04
Total Risk Across All Media and All Exposure Routes	2.7E-04

Total Hazard Index Across All Media and All Exposure Routes	2.0E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.3.2.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Side Gulches

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	6.7E-06	N/A	1.5E-06	8.2E-06	Antimony	longevity, glucose/ cholesterol--blood	3.7E-02	N/A	N/A	3.7E-02
								Arsenic	NOAEL/skin pigmentation	1.2E-01	N/A	2.6E-02	1.4E-01
								Cadmium	NOAEL/proteinuria--kidney	1.0E-02	N/A	1.8E-03	1.2E-02
								Iron	hematology--blood	1.2E-01	N/A	N/A	1.2E-01
								Manganese	NOAEL/Central Nervous System	3.6E-02	N/A	N/A	3.6E-02
								Zinc	enzyme effects--blood	4.1E-03	N/A	N/A	4.1E-03
			(Total)	6.7E-06	N/A	1.5E-06	8.2E-06	(Total)		3.2E-01	N/A	2.8E-02	3.5E-01
Groundwater	Groundwater	Tap water	Arsenic	7.2E-06	N/A	N/A	7.2E-06	Arsenic	NOAEL/skin pigmentation	1.2E-01	N/A	N/A	1.2E-01

Total Risk Across Soil	8.2E-06
Total Risk Across Groundwater	7.2E-06
Total Risk Across All Media and All Exposure Routes	1.5E-05
Total Hazard Index Across All Media and All Exposure Routes	4.8E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.3.3.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Side Gulches

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Elk Creek Area	Arsenic	8.8E-06	N/A	3.0E-06	1.2E-05	Antimony	longevity, glucose/ cholesterol--blood	1.4E-01	N/A	N/A	1.4E-01
								Arsenic	NOAEL/skin pigmentation	2.0E-01	N/A	6.7E-02	2.6E-01
								Cadmium	NOAEL/proteinuria--kidney	6.4E-02	N/A	1.7E-02	8.2E-02
								Iron	hematology--blood	3.3E-01	N/A	N/A	3.3E-01
								Manganese	NOAEL/Central Nervous System	2.0E-01	N/A	N/A	2.0E-01
								Zinc	enzyme effects--blood	2.0E-02	N/A	N/A	2.0E-02
			(Total)	8.8E-06	N/A	3.0E-06	1.2E-05	(Total)		9.5E-01	N/A	8.4E-02	1.0E+00
Sediment	Sediment	Elk Creek Pond	Arsenic	4.3E-06	N/A	3.3E-06	7.7E-06	Antimony	longevity, glucose/ cholesterol--blood	4.3E-02	N/A	N/A	4.3E-02
								Arsenic	NOAEL/skin pigmentation	9.6E-02	N/A	7.4E-02	1.7E-01
								Cadmium	NOAEL/proteinuria--kidney	2.4E-02	N/A	1.5E-02	3.8E-02
								Iron	hematology--blood	7.4E-02	N/A	N/A	7.4E-02
								Manganese	NOAEL/Central Nervous System	2.9E-02	N/A	N/A	2.9E-02
								Zinc	enzyme effects--blood	1.0E-02	N/A	N/A	1.0E-02
			(Total)	4.3E-06	N/A	3.3E-06	7.7E-06	(Total)		2.8E-01	N/A	8.9E-02	3.6E-01
Surface Water	Surface water	Elk Creek Pond	Arsenic	1.4E-06	N/A	N/A	1.4E-06	Arsenic	NOAEL/skin pigmentation	3.0E-02	N/A	N/A	3.0E-02
								Cadmium	NOAEL/proteinuria--kidney	3.4E-02	N/A	N/A	3.4E-02
								Manganese	NOAEL/Central Nervous System	5.1E-02	N/A	N/A	5.1E-02
								Mercury	autoimmunity	3.3E-03	N/A	N/A	3.3E-03
			(Total)	1.4E-06	N/A	N/A	1.4E-06	(Total)		1.2E-01	N/A	N/A	1.2E-01

Total Risk Across Soil	1.2E-05
Total Risk Across Sediment	7.7E-06
Total Risk Across Surface water	1.4E-06
Total Risk Across All Media and All Exposure Routes	2.1E-05

Total Hazard Index Across All Media and All Exposure Routes	1.5E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.3.3.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Side Gulches

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Elk Creek Area	Arsenic	4.1E-07	N/A	3.5E-07	7.6E-07	Antimony	longevity, glucose/ cholesterol--blood	2.3E-02	N/A	N/A	2.3E-02
								Arsenic	NOAEL/skin pigmentation	3.2E-02	N/A	2.7E-02	5.9E-02
								Cadmium	NOAEL/proteinuria--kidney	4.4E-03	N/A	2.9E-03	7.3E-03
								Iron	hematology--blood	5.7E-02	N/A	N/A	5.7E-02
								Manganese	NOAEL/Central Nervous System	2.9E-02	N/A	N/A	2.9E-02
								Zinc	enzyme effects--blood	2.2E-03	N/A	N/A	2.2E-03
			(Total)	4.1E-07	N/A	3.5E-07	7.6E-07	(Total)		1.5E-01	N/A	3.0E-02	1.8E-01
Sediment	Sediment	Elk Creek Pond	Arsenic	1.9E-07	N/A	3.9E-07	5.8E-07	Antimony	longevity, glucose/ cholesterol--blood	5.2E-03	N/A	N/A	5.2E-03
								Arsenic	NOAEL/skin pigmentation	1.5E-02	N/A	3.0E-02	4.5E-02
								Cadmium	NOAEL/proteinuria--kidney	5.0E-03	N/A	8.2E-03	1.3E-02
								Iron	hematology--blood	1.2E-02	N/A	N/A	1.2E-02
								Manganese	NOAEL/Central Nervous System	3.7E-03	N/A	N/A	3.7E-03
								Zinc	enzyme effects--blood	1.7E-03	N/A	N/A	1.7E-03
			(Total)	1.9E-07	N/A	3.9E-07	5.8E-07	(Total)		4.2E-02	N/A	3.9E-02	8.1E-02
Surface Water	Surface water	Elk Creek Pond	Arsenic	1.0E-07	N/A	N/A	1.0E-07	Arsenic	NOAEL/skin pigmentation	7.8E-03	N/A	N/A	7.8E-03
								Cadmium	NOAEL/proteinuria--kidney	7.5E-03	N/A	N/A	7.5E-03
								Manganese	NOAEL/Central Nervous System	6.9E-03	N/A	N/A	6.9E-03
								Mercury	autoimmunity	4.0E-04	N/A	N/A	4.0E-04
			(Total)	1.0E-07	N/A	N/A	1.0E-07	(Total)		2.3E-02	N/A	N/A	2.3E-02

Total Risk Across Soil	7.6E-07
Total Risk Across Sediment	5.8E-07
Total Risk Across Surface water	1.0E-07
Total Risk Across All Media and All Exposure Routes	1.4E-06

Total Hazard Index Across All Media and All Exposure Routes	2.8E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.4.1.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Osburn

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	4.0E-01	N/A	N/A	4.0E-01
								Arsenic	NOAEL/skin pigmentation	1.2E+00	N/A	1.3E-01	1.3E+00
								Cadmium	NOAEL/proteinuria--kidney	6.7E-02	N/A	5.9E-03	7.3E-02
								Iron	hematology--blood	9.6E-01	N/A	N/A	9.6E-01
								Manganese	NOAEL/Central Nervous System	3.3E-01	N/A	N/A	3.3E-01
								Zinc	enzyme effects--blood	2.5E-02	N/A	N/A	2.5E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		3.0E+00	N/A	1.4E-01	3.1E+00
Groundwater	Groundwater	Tap water	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	6.0E-02	N/A	N/A	6.0E-02

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	3.2E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.4.1.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENDCY EXPOSURE

Osburn

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.2E-01	N/A	N/A	1.2E-01
								Arsenic	NOAEL/skin pigmentation	3.7E-01	N/A	8.1E-02	4.5E-01
								Cadmium	NOAEL/proteinuria--kidney	2.3E-02	N/A	4.0E-03	2.7E-02
								Iron	hematology--blood	3.4E-01	N/A	N/A	3.4E-01
								Manganese	NOAEL/Central Nervous System	1.1E-01	N/A	N/A	1.1E-01
								Zinc	enzyme effects--blood	8.3E-03	N/A	N/A	8.3E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		9.7E-01	N/A	8.5E-02	1.1E+00
Groundwater	Groundwater	Tap water											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	3.3E-02	N/A	N/A	3.3E-02

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	1.1E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.4.2.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Osburn

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	6.6E-05	N/A	7.5E-06	7.3E-05	Antimony	longevity, glucose/ cholesterol--blood	1.1E-01	N/A	N/A	1.1E-01
								Arsenic	NOAEL/skin pigmentation	3.4E-01	N/A	3.9E-02	3.8E-01
								Cadmium	NOAEL/proteinuria--kidney	1.9E-02	N/A	1.8E-03	2.1E-02
								Iron	hematology--blood	2.7E-01	N/A	N/A	2.7E-01
								Manganese	NOAEL/Central Nervous System	9.3E-02	N/A	N/A	9.3E-02
								Zinc	enzyme effects--blood	7.3E-03	N/A	N/A	7.3E-03
			(Total)	6.6E-05	N/A	7.5E-06	7.3E-05	(Total)		8.5E-01	N/A	4.1E-02	8.9E-01
Groundwater	Groundwater	Tap water											
			Arsenic	6.2E-06	N/A	N/A	6.2E-06	Arsenic	NOAEL/skin pigmentation	3.2E-02	N/A	N/A	3.2E-02

Total Risk Across Soil	7.3E-05
Total Risk Across Groundwater	6.2E-06
Total Risk Across All Media and All Exposure Routes	8.0E-05
Total Hazard Index Across All Media and All Exposure Routes	9.2E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.4.2.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENDCY EXPOSURE

Osburn

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	6.5E-06	N/A	1.5E-06	8.0E-06	Antimony	longevity, glucose/ cholesterol--blood	3.6E-02	N/A	N/A	3.6E-02
								Arsenic	NOAEL/skin pigmentation	1.1E-01	N/A	2.6E-02	1.4E-01
								Cadmium	NOAEL/proteinuria--kidney	7.0E-03	N/A	1.3E-03	8.3E-03
								Iron	hematology--blood	1.0E-01	N/A	N/A	1.0E-01
								Manganese	NOAEL/Central Nervous System	3.5E-02	N/A	N/A	3.5E-02
								Zinc	enzyme effects--blood	2.5E-03	N/A	N/A	2.5E-03
			(Total)	6.5E-06	N/A	1.5E-06	8.0E-06	(Total)		3.0E-01	N/A	2.7E-02	3.2E-01
Groundwater	Groundwater	Tap water	Arsenic	8.6E-07	N/A	N/A	8.6E-07	Arsenic	NOAEL/skin pigmentation	1.5E-02	N/A	N/A	1.5E-02

Total Risk Across Soil	8.0E-06
Total Risk Across Groundwater	8.6E-07
Total Risk Across All Media and All Exposure Routes	8.9E-06
Total Hazard Index Across All Media and All Exposure Routes	3.4E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.5.1.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	2.8E-01	N/A	N/A	2.8E-01
								Arsenic	NOAEL/skin pigmentation	5.5E-01	N/A	6.0E-02	6.1E-01
								Cadmium	NOAEL/proteinuria--kidney	7.0E-02	N/A	6.1E-03	7.6E-02
								Iron	hematology--blood	8.6E-01	N/A	N/A	8.6E-01
								Manganese	NOAEL/Central Nervous System	2.9E-01	N/A	N/A	2.9E-01
								Zinc	enzyme effects--blood	3.5E-02	N/A	N/A	3.5E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		2.1E+00	N/A	6.6E-02	2.2E+00
Groundwater	Groundwater	Tap water	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.2E-01	N/A	N/A	1.2E-01

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	2.3E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.5.1.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	5.8E-02	N/A	N/A	5.8E-02
								Arsenic	NOAEL/skin pigmentation	1.6E-01	N/A	3.5E-02	1.9E-01
								Cadmium	NOAEL/proteinuria--kidney	2.1E-02	N/A	3.7E-03	2.5E-02
								Iron	hematology--blood	2.9E-01	N/A	N/A	2.9E-01
								Manganese	NOAEL/Central Nervous System	9.4E-02	N/A	N/A	9.4E-02
								Zinc	enzyme effects--blood	8.9E-03	N/A	N/A	8.9E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		6.3E-01	N/A	3.8E-02	6.7E-01
Groundwater	Groundwater	Tap water											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	5.4E-02	N/A	N/A	5.4E-02

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	7.2E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.5.2.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	3.0E-05	N/A	3.5E-06	3.4E-05	Antimony	longevity, glucose/ cholesterol--blood	8.1E-02	N/A	N/A	8.1E-02
								Arsenic	NOAEL/skin pigmentation	1.6E-01	N/A	1.8E-02	1.7E-01
								Cadmium	NOAEL/proteinuria--kidney	2.0E-02	N/A	1.8E-03	2.2E-02
								Iron	hematology--blood	2.5E-01	N/A	N/A	2.5E-01
								Manganese	NOAEL/Central Nervous System	8.3E-02	N/A	N/A	8.3E-02
								Zinc	enzyme effects--blood	9.9E-03	N/A	N/A	9.9E-03
			(Total)	3.0E-05	N/A	3.5E-06	3.4E-05	(Total)		6.0E-01	N/A	2.0E-02	6.2E-01
Groundwater	Groundwater	Tap water											
			Arsenic	1.2E-05	N/A	N/A	1.2E-05	Arsenic	NOAEL/skin pigmentation	6.3E-02	N/A	N/A	6.3E-02

Total Risk Across Soil	3.4E-05
Total Risk Across Groundwater	1.2E-05
Total Risk Across All Media and All Exposure Routes	4.7E-05
Total Hazard Index Across All Media and All Exposure Routes	6.8E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.5.2.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	2.8E-06	N/A	6.3E-07	3.4E-06	Antimony	longevity, glucose/ cholesterol--blood	1.8E-02	N/A	N/A	1.8E-02
								Arsenic	NOAEL/skin pigmentation	4.8E-02	N/A	1.1E-02	5.9E-02
								Cadmium	NOAEL/proteinuria--kidney	6.4E-03	N/A	1.2E-03	7.6E-03
								Iron	hematology--blood	8.9E-02	N/A	N/A	8.9E-02
								Manganese	NOAEL/Central Nervous System	2.9E-02	N/A	N/A	2.9E-02
								Zinc	enzyme effects--blood	2.7E-03	N/A	N/A	2.7E-03
			(Total)	2.8E-06	N/A	6.3E-07	3.4E-06	(Total)		1.9E-01	N/A	1.2E-02	2.0E-01
Groundwater	Groundwater	Tap water	Arsenic	1.4E-06	N/A	N/A	1.4E-06	Arsenic	NOAEL/skin pigmentation	2.5E-02	N/A	N/A	2.5E-02
						Total Risk Across Soil	3.4E-06					Total Hazard Index Across All Media and All Exposure Routes	2.3E-01
						Total Risk Across Groundwater	1.4E-06						
						Total Risk Across All Media and All Exposure Routes	4.8E-06						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.5.3.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	2.6E-06	N/A	8.7E-07	3.4E-06	Antimony	longevity, glucose/ cholesterol--blood	1.7E-02	N/A	N/A	1.7E-02
								Arsenic	NOAEL/skin pigmentation	5.7E-02	N/A	1.9E-02	7.6E-02
								Cadmium	NOAEL/proteinuria--kidney	4.6E-03	N/A	1.2E-03	5.8E-03
								Iron	hematology--blood	6.7E-02	N/A	N/A	6.7E-02
								Manganese	NOAEL/Central Nervous System	2.3E-02	N/A	N/A	2.3E-02
								Zinc	enzyme effects--blood	2.5E-03	N/A	N/A	2.5E-03
			(Total)	2.6E-06	N/A	8.7E-07	3.4E-06	(Total)		1.7E-01	N/A	2.0E-02	1.9E-01
						Total Risk Across Soil	3.4E-06					Total Hazard Index Across All Media and All Exposure Routes	1.9E-01
						Total Risk Across All Media and All Exposure Routes	3.4E-06						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.5.3.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood Residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools											
			Arsenic	8.7E-08	N/A	7.3E-08	1.6E-07	Antimony	longevity, glucose/ cholesterol--blood	2.1E-03	N/A	N/A	2.1E-03
								Arsenic	NOAEL/skin pigmentation	6.7E-03	N/A	5.7E-03	1.2E-02
								Cadmium	NOAEL/proteinuria--kidney	1.0E-03	N/A	6.9E-04	1.7E-03
								Iron	hematology--blood	1.3E-02	N/A	N/A	1.3E-02
								Manganese	NOAEL/Central Nervous System	4.4E-03	N/A	N/A	4.4E-03
								Zinc	enzyme effects--blood	3.6E-04	N/A	N/A	3.6E-04
			(Total)	8.7E-08	N/A	7.3E-08	1.6E-07	(Total)		2.8E-02	N/A	6.4E-03	3.4E-02
							Total Risk Across Soil						Total Hazard Index Across All Media and All Exposure Routes
							1.6E-07						3.4E-02
							Total Risk Across All Media and All Exposure Routes						
							1.6E-07						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.5.4.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	3.2E-02	N/A	N/A	3.2E-02
								Arsenic	NOAEL/skin pigmentation	1.1E-01	N/A	1.6E-02	1.2E-01
								Cadmium	NOAEL/proteinuria--kidney	8.5E-03	N/A	1.0E-03	9.5E-03
								Iron	hematology--blood	1.2E-01	N/A	N/A	1.2E-01
								Manganese	NOAEL/Central Nervous System	4.3E-02	N/A	N/A	4.3E-02
								Zinc	enzyme effects--blood	4.6E-03	N/A	N/A	4.6E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		3.2E-01	N/A	1.7E-02	3.4E-01
						Total Risk Across Soil	N/A					Total Hazard Index Across All Media and All Exposure Routes	3.4E-01
						Total Risk Across All Media and All Exposure Routes	N/A						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.5.4.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	4.0E-03	N/A	N/A	4.0E-03
								Arsenic	NOAEL/skin pigmentation	1.3E-02	N/A	4.6E-03	1.7E-02
								Cadmium	NOAEL/proteinuria--kidney	1.9E-03	N/A	5.6E-04	2.5E-03
								Iron	hematology--blood	2.4E-02	N/A	N/A	2.4E-02
								Manganese	NOAEL/Central Nervous System	8.2E-03	N/A	N/A	8.2E-03
								Zinc	enzyme effects--blood	6.7E-04	N/A	N/A	6.7E-04
			(Total)	N/A	N/A	N/A	N/A	(Total)		5.2E-02	N/A	5.2E-03	5.7E-02
							Total Risk Across Soil					Total Hazard Index Across All Media and All Exposure Routes	5.7E-02
							Total Risk Across All Media and All Exposure Routes						
							N/A						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.5.5.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	5.1E-06	N/A	8.9E-07	6.0E-06	Antimony	longevity, glucose/ cholesterol--blood	8.1E-03	N/A	N/A	8.1E-03
								Arsenic	NOAEL/skin pigmentation	2.7E-02	N/A	4.6E-03	7.3E-02
								Cadmium	NOAEL/proteinuria--kidney	2.1E-03	N/A	3.0E-04	2.4E-03
								Iron	hematology--blood	3.1E-02	N/A	N/A	3.1E-02
								Manganese	NOAEL/Central Nervous System	1.1E-02	N/A	N/A	1.1E-02
								Zinc	enzyme effects--blood	1.1E-03	N/A	N/A	1.1E-03
			(Total)	5.1E-06	N/A	8.9E-07	6.0E-06	(Total)		8.0E-02	N/A	4.9E-03	8.5E-02
						Total Risk Across Soil	6.0E-06					Total Hazard Index Across All Media and All Exposure Routes	8.5E-02
						Total Risk Across All Media and All Exposure Routes	6.0E-06						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.5.5.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	2.1E-07	N/A	8.5E-08	2.9E-07	Antimony	longevity, glucose/ cholesterol--blood	1.1E-03	N/A	N/A	1.1E-03
								Arsenic	NOAEL/skin pigmentation	3.6E-03	N/A	1.5E-03	5.1E-03
								Cadmium	NOAEL/proteinuria--kidney	5.4E-04	N/A	1.8E-04	7.2E-04
								Iron	hematology--blood	6.9E-03	N/A	N/A	6.9E-03
								Manganese	NOAEL/Central Nervous System	2.3E-03	N/A	N/A	2.3E-03
								Zinc	enzyme effects--blood	1.9E-04	N/A	N/A	1.9E-04
			(Total)	2.1E-07	N/A	8.5E-08	2.9E-07	(Total)		1.5E-02	N/A	1.6E-03	1.6E-02
						Total Risk Across Soil	2.9E-07					Total Hazard Index Across All Media and All Exposure Routes	1.6E-02
						Total Risk Across All Media and All Exposure Routes	2.9E-07						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.6.1.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	4.4E-01	N/A	N/A	4.4E-01
								Arsenic	NOAEL/skin pigmentation	5.8E-01	N/A	6.3E-02	6.4E-01
								Cadmium	NOAEL/proteinuria--kidney	9.8E-02	N/A	8.6E-03	1.1E-01
								Iron	hematology--blood	9.5E-01	N/A	N/A	9.5E-01
								Manganese	NOAEL/Central Nervous System	2.7E-01	N/A	N/A	2.7E-01
								Zinc	enzyme effects--blood	4.1E-02	N/A	N/A	4.1E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		2.4E+00	N/A	7.2E-02	2.4E+00
Groundwater	Groundwater	Tap water	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.1E-01	N/A	N/A	1.1E-01

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	2.5E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.6.1.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.0E-01	N/A	N/A	1.0E-01
								Arsenic	NOAEL/skin pigmentation	1.9E-01	N/A	4.2E-02	2.3E-01
								Cadmium	NOAEL/proteinuria--kidney	3.1E-02	N/A	5.4E-03	3.6E-02
								Iron	hematology--blood	3.3E-01	N/A	N/A	3.3E-01
								Manganese	NOAEL/Central Nervous System	9.0E-02	N/A	N/A	9.0E-02
								Zinc	enzyme effects--blood	1.4E-02	N/A	N/A	1.4E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		7.7E-01	N/A	4.8E-02	8.1E-01
Groundwater	Groundwater	Tap water											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	6.0E-02	N/A	N/A	6.0E-02

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	8.7E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.6.2.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	3.2E-05	N/A	3.6E-06	3.5E-05	Antimony	longevity, glucose/ cholesterol--blood	1.3E-01	N/A	N/A	1.3E-01
								Arsenic	NOAEL/skin pigmentation	1.6E-01	N/A	1.9E-02	1.8E-01
								Cadmium	NOAEL/proteinuria--kidney	2.8E-02	N/A	2.6E-03	3.0E-02
								Iron	hematology--blood	2.7E-01	N/A	N/A	2.7E-01
								Manganese	NOAEL/Central Nervous System	7.7E-02	N/A	N/A	7.7E-02
								Zinc	enzyme effects--blood	1.2E-02	N/A	N/A	1.2E-02
			(Total)	3.2E-05	N/A	3.6E-06	3.5E-05	(Total)		6.8E-01	N/A	2.1E-02	7.0E-01
Groundwater	Groundwater	Tap water											
			Arsenic	1.1E-05	N/A	N/A	1.1E-05	Arsenic	NOAEL/skin pigmentation	5.7E-02	N/A	N/A	5.7E-02

Total Risk Across Soil	3.5E-05
Total Risk Across Groundwater	1.1E-05
Total Risk Across All Media and All Exposure Routes	4.7E-05
Total Hazard Index Across All Media and All Exposure Routes	7.6E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.6.2.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	3.4E-06	N/A	7.8E-07	4.2E-06	Antimony	longevity, glucose/ cholesterol--blood	3.2E-02	N/A	N/A	3.2E-02
								Arsenic	NOAEL/skin pigmentation	5.9E-02	N/A	1.3E-02	7.2E-02
								Cadmium	NOAEL/proteinuria--kidney	9.4E-03	N/A	1.7E-03	1.1E-02
								Iron	hematology--blood	1.0E-01	N/A	N/A	1.0E-01
								Manganese	NOAEL/Central Nervous System	2.8E-02	N/A	N/A	2.8E-02
								Zinc	enzyme effects--blood	4.2E-03	N/A	N/A	4.2E-03
			(Total)	3.4E-06	N/A	7.8E-07	4.2E-06	(Total)		2.3E-01	N/A	1.5E-02	2.5E-01
Groundwater	Groundwater	Tap water	Arsenic	1.6E-06	N/A	N/A	1.6E-06	Arsenic	NOAEL/skin pigmentation	2.7E-02	N/A	N/A	2.7E-02

Total Risk Across Soil	4.2E-06
Total Risk Across Groundwater	1.6E-06
Total Risk Across All Media and All Exposure Routes	5.8E-06
Total Hazard Index Across All Media and All Exposure Routes	2.8E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.6.3.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	2.5E-06	N/A	8.4E-07	3.3E-06	Antimony	longevity, glucose/ cholesterol--blood	4.5E-02	N/A	N/A	4.5E-02
								Arsenic	NOAEL/skin pigmentation	5.5E-02	N/A	1.9E-02	7.4E-02
								Cadmium	NOAEL/proteinuria--kidney	1.1E-02	N/A	2.9E-03	1.4E-02
								Iron	hematology--blood	9.5E-02	N/A	N/A	9.5E-02
								Manganese	NOAEL/Central Nervous System	3.2E-02	N/A	N/A	3.2E-02
								Zinc	enzyme effects--blood	6.4E-03	N/A	N/A	6.4E-03
			(Total)	2.5E-06			3.3E-06	(Total)		2.4E-01	N/A	2.2E-02	2.7E-01
						Total Risk Across Soil	3.3E-06					Total Hazard Index Across All Media and All Exposure Routes	2.7E-01
						Total Risk Across All Media and All Exposure Routes	3.3E-06						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.6.3.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood Residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	1.3E-07	N/A	1.1E-07	2.4E-07	Antimony	longevity, glucose/ cholesterol--blood	8.9E-03	N/A	N/A	8.9E-03
								Arsenic	NOAEL/skin pigmentation	1.0E-02	N/A	8.7E-03	1.9E-02
								Cadmium	NOAEL/proteinuria--kidney	1.9E-03	N/A	1.3E-03	3.2E-03
								Iron	hematology--blood	1.8E-02	N/A	N/A	1.8E-02
								Manganese	NOAEL/Central Nervous System	5.8E-03	N/A	N/A	5.8E-03
								Zinc	enzyme effects--blood	1.2E-03	N/A	N/A	1.2E-03
			(Total)	1.3E-07			2.4E-07	(Total)		4.6E-02	N/A	1.0E-02	5.6E-02
						Total Risk Across Soil	2.4E-07					Total Hazard Index Across All Media and All Exposure Routes	5.6E-02
						Total Risk Across All Media and All Exposure Routes	2.4E-07						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.6.4.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	8.5E-02	N/A	N/A	8.5E-02
								Arsenic	NOAEL/skin pigmentation	1.0E-01	N/A	1.5E-02	1.2E-01
								Cadmium	NOAEL/proteinuria--kidney	2.0E-02	N/A	2.3E-03	2.2E-02
								Iron	hematology--blood	1.8E-01	N/A	N/A	1.8E-01
								Manganese	NOAEL/Central Nervous System	6.0E-02	N/A	N/A	6.0E-02
								Zinc	enzyme effects--blood	1.2E-02	N/A	N/A	1.2E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		4.6E-01	N/A	1.7E-02	4.7E-01
							Total Risk Across Soil					Total Hazard Index Across All Media and All Exposure Routes	4.7E-01
							Total Risk Across All Media and All Exposure Routes						
							N/A						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.6.4.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.7E-02	N/A	N/A	1.7E-02
								Arsenic	NOAEL/skin pigmentation	1.9E-02	N/A	7.0E-03	2.6E-02
								Cadmium	NOAEL/proteinuria--kidney	3.6E-03	N/A	1.1E-03	4.6E-03
								Iron	hematology--blood	3.4E-02	N/A	N/A	3.4E-02
								Manganese	NOAEL/Central Nervous System	1.1E-02	N/A	N/A	1.1E-02
								Zinc	enzyme effects--blood	2.2E-03	N/A	N/A	2.2E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		8.6E-02	N/A	8.1E-03	9.4E-02
							Total Risk Across Soil					Total Hazard Index Across All Media and All Exposure Routes	9.4E-02
							Total Risk Across All Media and All Exposure Routes						
							N/A						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.6.5.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	5.0E-06	N/A	8.7E-07	5.8E-06	Antimony	longevity, glucose/ cholesterol--blood	2.1E-02	N/A	N/A	2.1E-02
								Arsenic	NOAEL/skin pigmentation	2.6E-02	N/A	4.5E-03	3.0E-02
								Cadmium	NOAEL/proteinuria--kidney	5.0E-03	N/A	7.0E-04	5.7E-03
								Iron	hematology--blood	4.5E-02	N/A	N/A	4.5E-02
								Manganese	NOAEL/Central Nervous System	1.5E-02	N/A	N/A	1.5E-02
								Zinc	enzyme effects--blood	3.0E-03	N/A	N/A	3.0E-03
			(Total)	5.0E-06	N/A	8.7E-07	5.8E-06	(Total)		1.1E-01	N/A	5.2E-03	1.2E-01
						Total Risk Across Soil	5.8E-06					Total Hazard Index Across All Media and All Exposure Routes	1.2E-01
						Total Risk Across All Media and All Exposure Routes	5.8E-06						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.6.5.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	3.2E-07	N/A	1.3E-07	4.4E-07	Antimony	longevity, glucose/ cholesterol--blood	4.7E-03	N/A	N/A	4.7E-03
								Arsenic	NOAEL/skin pigmentation	5.5E-03	N/A	2.2E-03	7.7E-03
								Cadmium	NOAEL/proteinuria--kidney	1.0E-03	N/A	3.3E-04	1.4E-03
								Iron	hematology--blood	9.5E-03	N/A	N/A	9.5E-03
								Manganese	NOAEL/Central Nervous System	3.1E-03	N/A	N/A	3.1E-03
								Zinc	enzyme effects--blood	6.2E-04	N/A	N/A	6.2E-04
			(Total)	3.2E-07	N/A	1.3E-07	4.4E-07	(Total)		2.4E-02	N/A	2.6E-03	2.7E-02
						Total Risk Across Soil	4.4E-07					Total Hazard Index Across All Media and All Exposure Routes	2.7E-02
						Total Risk Across All Media and All Exposure Routes	4.4E-07						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.7.1.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Osburn, Wallace, and Silverton Combined

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Sediment	SFCDAR	Arsenic	2.9E-06	N/A	2.3E-06	5.2E-06	Antimony	longevity, glucose/ cholesterol--blood	5.9E-02	N/A	N/A	5.9E-02
								Arsenic	NOAEL/skin pigmentation	6.5E-02	N/A	5.0E-02	1.1E-01
								Cadmium	NOAEL/proteinuria--kidney	1.2E-02	N/A	7.2E-03	1.9E-02
								Iron	hematology--blood	8.5E-02	N/A	N/A	8.5E-02
								Manganese	NOAEL/Central Nervous System	4.9E-02	N/A	N/A	4.9E-02
								Zinc	enzyme effects--blood	7.0E-03	N/A	N/A	7.0E-03
			(Total)	2.9E-06	N/A	2.3E-06	5.2E-06	(Total)		2.8E-01	N/A	5.7E-02	3.3E-01
Surface Water	Surface water	SFCDAR	Arsenic	4.6E-08	N/A	N/A	4.6E-08	Arsenic	NOAEL/skin pigmentation	1.0E-03	N/A	N/A	1.0E-03
								Cadmium	NOAEL/proteinuria--kidney	2.3E-03	N/A	N/A	2.3E-03
								Manganese	NOAEL/Central Nervous System	1.9E-04	N/A	N/A	1.9E-04
								Mercury	autoimmunity	1.1E-04	N/A	N/A	1.1E-04
			(Total)	4.6E-08	N/A	N/A	4.6E-08	(Total)		3.6E-03	N/A	N/A	3.6E-03
						Total Risk Across Soil/sediment	5.2E-06					Total Hazard Index Across All Media and All Exposure Routes	3.4E-01
						Total Risk Across Surface water	4.6E-08						
						Total Risk Across All Media and All Exposure Routes	5.2E-06						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.7.1.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Osburn, Wallace, and Silverton Combined

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Sediment	SFCDAR											
			Arsenic	1.1E-07	N/A	2.3E-07	3.4E-07	Antimony	longevity, glucose/ cholesterol--blood	7.4E-03	N/A	N/A	7.4E-03
								Arsenic	NOAEL/skin pigmentation	8.6E-03	N/A	1.8E-02	2.7E-02
								Cadmium	NOAEL/proteinuria--kidney	1.7E-03	N/A	2.7E-03	4.4E-03
								Iron	hematology--blood	1.5E-02	N/A	N/A	1.5E-02
								Manganese	NOAEL/Central Nervous System	8.3E-03	N/A	N/A	8.3E-03
								Zinc	enzyme effects--blood	1.0E-03	N/A	N/A	1.0E-03
			(Total)	1.1E-07	N/A	2.3E-07	3.4E-07	(Total)		4.2E-02	N/A	2.0E-02	6.2E-02
Surface Water	Surface water	SFCAR											
			Arsenic	4.7E-09	N/A	N/A	4.7E-09	Arsenic	NOAEL/skin pigmentation	3.7E-04	N/A	N/A	3.7E-04
								Cadmium	NOAEL/proteinuria--kidney	3.0E-03	N/A	N/A	3.0E-03
								Manganese	NOAEL/Central Nervous System	1.2E-04	N/A	N/A	1.2E-04
								Mercury	autoimmunity	4.7E-05	N/A	N/A	4.7E-05
			(Total)	4.7E-09	N/A	N/A	4.7E-09	(Total)		3.5E-03	N/A	N/A	3.5E-03
						Total Risk Across Soil/sediment	3.4E-07					Total Hazard Index Across All Media and All Exposure Routes	6.6E-02
						Total Risk Across Surface water	4.7E-09						
						Total Risk Across All Media and All Exposure Routes	3.4E-07						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.7.2.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Osburn, Wallace, and Silverton Combined

Scenario Timeframe:	Current/Future
Receptor Population:	Construction Workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	2.0E-05	N/A	8.4E-07	2.1E-05	Antimony	longevity, glucose/ cholesterol--blood	5.3E-02	N/A	N/A	5.3E-02
								Arsenic	NOAEL/skin pigmentation	1.3E-01	N/A	5.2E-03	1.3E-01
								Cadmium	NOAEL/proteinuria--kidney	1.2E-02	N/A	4.0E-04	1.2E-02
								Iron	hematology--blood	1.7E-01	N/A	N/A	1.7E-01
								Manganese	NOAEL/Central Nervous System	1.8E-02	N/A	N/A	1.8E-02
								Zinc	enzyme effects--blood	5.7E-03	N/A	N/A	5.7E-03
			(Total)	2.0E-05	N/A	8.4E-07	2.1E-05	(Total)		3.8E-01	N/A	5.6E-03	3.9E-01
						Total Risk Across Soil	2.1E-05					Total Hazard Index Across All Media and All Exposure Routes	3.9E-01
						Total Risk Across All Media and All Exposure Routes	2.1E-05						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.7.2.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Osburn, Wallace, and Silverton Combined

Scenario Timeframe:	Current/Future
Receptor Population:	Construction workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	6.2E-07	N/A	3.9E-08	6.6E-07	Antimony	longevity, glucose/ cholesterol--blood	5.6E-03	N/A	N/A	5.6E-03
								Arsenic	NOAEL/skin pigmentation	1.5E-02	N/A	9.2E-04	1.6E-02
								Cadmium	NOAEL/proteinuria--kidney	2.0E-03	N/A	1.0E-04	2.2E-03
								Iron	hematology--blood	2.4E-02	N/A	N/A	2.4E-02
								Manganese	NOAEL/Central Nervous System	2.6E-03	N/A	N/A	2.6E-03
								Zinc	enzyme effects--blood	7.5E-04	N/A	N/A	7.5E-04
			(Total)	6.2E-07	N/A	3.9E-08	6.6E-07	(Total)		5.0E-02	N/A	1.0E-03	5.1E-02
						Total Risk Across Soil	6.6E-07					Total Hazard Index Across All Media and All Exposure Routes	5.1E-02
						Total Risk Across All Media and All Exposure Routes	6.6E-07						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.8.1.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	3.8E-01	N/A	N/A	3.8E-01
								Arsenic	NOAEL/skin pigmentation	1.1E+00	N/A	1.2E-01	1.2E+00
								Cadmium	NOAEL/proteinuria--kidney	8.3E-02	N/A	7.3E-03	9.1E-02
								Iron	hematology--blood	9.9E-01	N/A	N/A	9.9E-01
								Manganese	NOAEL/Central Nervous System	3.4E-01	N/A	N/A	3.4E-01
								Zinc	enzyme effects--blood	4.9E-02	N/A	N/A	4.9E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		2.9E+00	N/A	1.2E-01	3.0E+00
Groundwater	Groundwater	Tap water											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.6E-01	N/A	N/A	1.6E-01

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	3.2E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.8.1.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	9.7E-02	N/A	N/A	9.7E-02
								Arsenic	NOAEL/skin pigmentation	3.0E-01	N/A	6.6E-02	3.7E-01
								Cadmium	NOAEL/proteinuria--kidney	2.8E-02	N/A	5.0E-03	3.4E-02
								Iron	hematology--blood	3.5E-01	N/A	N/A	3.5E-01
								Manganese	NOAEL/Central Nervous System	1.2E-01	N/A	N/A	1.2E-01
								Zinc	enzyme effects--blood	1.8E-02	N/A	N/A	1.8E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		9.1E-01	N/A	7.1E-02	9.8E-01
Groundwater	Groundwater	Tap water											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	6.4E-02	N/A	N/A	6.4E-02

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	1.0E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.8.2.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	5.9E-05	N/A	6.7E-06	6.5E-05	Antimony	longevity, glucose/ cholesterol--blood	1.1E-01	N/A	N/A	1.1E-01
								Arsenic	NOAEL/skin pigmentation	3.0E-01	N/A	3.5E-02	3.4E-01
								Cadmium	NOAEL/proteinuria--kidney	2.4E-02	N/A	2.2E-03	2.6E-02
								Iron	hematology--blood	2.8E-01	N/A	N/A	2.8E-01
								Manganese	NOAEL/Central Nervous System	9.7E-02	N/A	N/A	9.7E-02
								Zinc	enzyme effects--blood	1.4E-02	N/A	N/A	1.4E-02
			(Total)	5.9E-05	N/A	6.7E-06	6.5E-05	(Total)		8.3E-01	N/A	3.7E-02	8.7E-01
Groundwater	Groundwater	Tap water	Arsenic	1.7E-05	N/A	N/A	1.7E-05	Arsenic	NOAEL/skin pigmentation	8.6E-02	N/A	N/A	8.6E-02

Total Risk Across Soil	6.5E-05
Total Risk Across Groundwater	1.7E-05
Total Risk Across All Media and All Exposure Routes	8.2E-05
Total Hazard Index Across All Media and All Exposure Routes	9.5E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.8.2.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	5.3E-06	N/A	1.2E-06	6.5E-06	Antimony	longevity, glucose/ cholesterol--blood	3.0E-02	N/A	N/A	3.0E-02
								Arsenic	NOAEL/skin pigmentation	9.2E-02	N/A	2.1E-02	1.1E-01
								Cadmium	NOAEL/proteinuria--kidney	8.7E-03	N/A	1.6E-03	1.0E-02
								Iron	hematology--blood	1.1E-01	N/A	N/A	1.1E-01
								Manganese	NOAEL/Central Nervous System	3.6E-02	N/A	N/A	3.6E-02
								Zinc	enzyme effects--blood	5.4E-03	N/A	N/A	5.4E-03
			(Total)	5.3E-06	N/A	1.2E-06	6.5E-06	(Total)		2.8E-01	N/A	2.3E-02	3.0E-01
Groundwater	Groundwater	Tap water	Arsenic	1.7E-06	N/A	N/A	1.7E-06	Arsenic	NOAEL/skin pigmentation	2.9E-02	N/A	N/A	2.9E-02

Total Risk Across Soil	6.5E-06
Total Risk Across Groundwater	1.7E-06
Total Risk Across All Media and All Exposure Routes	6.1E-06
Total Hazard Index Across All Media and All Exposure Routes	3.3E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.8.3.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Groundwater	Groundwater	Tap water	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	3.4E-01	N/A	N/A	3.4E-01
								Arsenic	NOAEL/skin pigmentation	2.7E-01	N/A	N/A	2.7E-01
								Cadmium	NOAEL/proteinuria--kidney	1.7E+01	N/A	N/A	1.7E+01
								Zinc	enzyme effects--blood	4.2E+00	N/A	N/A	4.2E+00
			(Total)	N/A	N/A	N/A	N/A	(Total)		2.2E+01	N/A	N/A	2.2E+01
							Total Risk Across Groundwater						Total Hazard Index Across All Media and All Exposure Routes
							N/A						2.2E+01
							Total Risk Across All Media and All Exposure Routes						
							N/A						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol and serum superoxide dismutase activity for antimony and zinc, respectively).

N/A = not applicable

TABLE 9.8.3.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Nine Mile

Scenario Timeframe:	Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Groundwater	Groundwater	Tap water	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.8E-01	N/A	N/A	1.8E-01
								Arsenic	NOAEL/skin pigmentation	1.2E-01	N/A	N/A	1.2E-01
								Cadmium	NOAEL/proteinuria--kidney	7.8E+00	N/A	N/A	7.8E+00
								Zinc	enzyme effects--blood	2.0E+00	N/A	N/A	2.0E+00
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.0E+01	N/A	N/A	1.0E+01
							Total Risk Across Groundwater						Total Hazard Index Across All Media and All Exposure Routes
							N/A						1.0E+01
							Total Risk Across All Media and All Exposure Routes						
							N/A						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol and serum superoxide dismutase activity for antimony and zinc, respectively).

N/A = not applicable

TABLE 9.8.4.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Groundwater	Groundwater	Tap water	Arsenic	2.8E-05	N/A	N/A	2.8E-05	Antimony	longevity, glucose/ cholesterol--blood	1.8E-01	N/A	N/A	1.8E-01
								Arsenic	NOAEL/skin pigmentation	1.4E-01	N/A	N/A	1.4E-01
								Cadmium	NOAEL/proteinuria--kidney	9.1E+00	N/A	N/A	9.1E+00
								Zinc	enzyme effects--blood	2.3E+00	N/A	N/A	2.3E+00
			(Total)	2.8E-05	N/A	N/A	2.8E-05	(Total)		1.2E+01	N/A	N/A	1.2E+01
						Total Risk Across Groundwater	2.8E-05					Total Hazard Index Across All Media and All Exposure Routes	1.2E+01
						Total Risk Across All Media and All Exposure Routes	2.8E-05						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol and serum superoxide dismutase activity for antimony and zinc, respectively).

N/A = not applicable

TABLE 9.8.4.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Nine Mile

Scenario Timeframe:	Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Groundwater	Groundwater	Tap water	Arsenic	3.0E-06	N/A	N/A	3.0E-06	Antimony	longevity, glucose/ cholesterol--blood	8.1E-02	N/A	N/A	8.1E-02
								Arsenic	NOAEL/skin pigmentation	5.3E-02	N/A	N/A	5.3E-02
								Cadmium	NOAEL/proteinuria--kidney	3.6E+00	N/A	N/A	3.6E+00
								Zinc	enzyme effects--blood	9.2E-01	N/A	N/A	9.2E-01
			(Total)	3.0E-06	N/A	N/A	3.0E-06	(Total)		4.6E+00	N/A	N/A	4.6E+00
						Total Risk Across Groundwater	3.0E-06					Total Hazard Index Across All Media and All Exposure Routes	4.6E+00
						Total Risk Across All Media and All Exposure Routes	3.0E-06						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol and serum superoxide dismutase activity for antimony and zinc, respectively).

N/A = not applicable

TABLE 9.8.5.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Waste piles	Arsenic	2.3E-05	N/A	7.9E-06	3.1E-05	Antimony	longevity, glucose/ cholesterol--blood	5.3E-02	N/A	N/A	5.3E-02
								Arsenic	NOAEL/skin pigmentation	5.2E-01	N/A	1.8E-01	7.0E-01
								Cadmium	NOAEL/proteinuria--kidney	1.6E-02	N/A	4.3E-03	2.0E-02
								Iron	hematology--blood	7.2E-02	N/A	N/A	7.2E-02
								Manganese	NOAEL/Central Nervous System	1.8E-02	N/A	N/A	1.8E-02
								Zinc	enzyme effects--blood	1.0E-02	N/A	N/A	1.0E-02
			(Total)	2.3E-05	N/A	7.9E-06	3.1E-05	(Total)		6.9E-01	N/A	1.8E-01	8.7E-01
Sediment	Sediment	Nine Mile/Canyon Creek	Arsenic	2.8E-06	N/A	2.2E-06	5.0E-06	Antimony	longevity, glucose/ cholesterol--blood	1.1E-01	N/A	N/A	1.1E-01
								Arsenic	NOAEL/skin pigmentation	6.2E-02	N/A	4.8E-02	1.1E-01
								Cadmium	NOAEL/proteinuria--kidney	3.6E-02	N/A	2.3E-02	5.9E-02
								Iron	hematology--blood	7.5E-02	N/A	N/A	7.5E-02
								Manganese	NOAEL/Central Nervous System	3.4E-02	N/A	N/A	3.4E-02
								Zinc	enzyme effects--blood	1.8E-02	N/A	N/A	1.8E-02
			(Total)	2.8E-06	N/A	2.2E-06	5.0E-06	(Total)		3.4E-01	N/A	7.1E-02	4.1E-01
Surface Water	Surface water	Nine Mile/Canyon Creek	Arsenic	4.6E-08	N/A	N/A	4.6E-08	Arsenic	NOAEL/skin pigmentation	1.0E-03	N/A	N/A	1.0E-03
								Cadmium	NOAEL/proteinuria--kidney	6.3E-02	N/A	N/A	6.3E-02
								Manganese	NOAEL/Central Nervous System	1.0E-02	N/A	N/A	1.0E-02
								Mercury	autoimmunity	2.9E-04	N/A	N/A	2.9E-04
			(Total)	4.6E-08	N/A	N/A	4.6E-08	(Total)		7.4E-02	N/A	N/A	7.4E-02

Total Risk Across Soil	3.1E-05
Total Risk Across Sediment	5.0E-06
Total Risk Across Surface water	4.6E-08
Total Risk Across All Media and All Exposure Routes	3.6E-05

Total Hazard Index Across All Media and All Exposure Routes	1.3E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.8.5.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Waste piles											
			Arsenic	2.5E-07	N/A	2.1E-07	4.5E-07	Antimony	longevity, glucose/ cholesterol--blood	4.9E-03	N/A	N/A	4.9E-03
								Arsenic	NOAEL/skin pigmentation	1.9E-02	N/A	1.6E-02	3.9E-02
								Cadmium	NOAEL/proteinuria--kidney	1.9E-03	N/A	1.3E-03	3.2E-03
								Iron	hematology--blood	1.2E-02	N/A	N/A	1.2E-02
								Manganese	NOAEL/Central Nervous System	3.1E-03	N/A	N/A	3.1E-03
								Zinc	enzyme effects--blood	1.7E-03	N/A	N/A	1.7E-03
			(Total)	2.5E-07	N/A	2.1E-07	4.5E-07	(Total)		4.3E-02	N/A	1.7E-02	6.0E-02
Sediment	Sediment	Nine Mile/Canyon Creek											
			Arsenic	7.0E-08	N/A	1.4E-07	2.1E-07	Antimony	longevity, glucose/ cholesterol--blood	1.4E-02	N/A	N/A	1.4E-02
								Arsenic	NOAEL/skin pigmentation	5.4E-03	N/A	1.1E-02	1.6E-02
								Cadmium	NOAEL/proteinuria--kidney	6.9E-03	N/A	1.1E-02	1.8E-02
								Iron	hematology--blood	1.0E-02	N/A	N/A	1.0E-02
								Manganese	NOAEL/Central Nervous System	4.6E-03	N/A	N/A	4.6E-03
								Zinc	enzyme effects--blood	3.3E-03	N/A	N/A	3.3E-03
			(Total)	7.0E-08	N/A	1.4E-07	2.1E-07	(Total)		4.5E-02	N/A	2.2E-02	6.7E-02
Surface Water	Surface water	Nine Mile/Canyon Creek											
			Arsenic	4.2E-09	N/A	N/A	4.2E-09	Arsenic	NOAEL/skin pigmentation	3.3E-04	N/A	N/A	3.3E-04
								Cadmium	NOAEL/proteinuria--kidney	1.7E-02	N/A	N/A	1.7E-02
								Manganese	NOAEL/Central Nervous System	2.2E-03	N/A	N/A	2.2E-03
								Mercury	autoimmunity	1.3E-04	N/A	N/A	1.3E-04
			(Total)	4.2E-09	N/A	N/A	4.2E-09	(Total)		1.9E-02	N/A	N/A	1.9E-02

Total Risk Across Soil	4.5E-07
Total Risk Across Sediment	2.1E-07
Total Risk Across Surface water	4.2E-09
Total Risk Across All Media and All Exposure Routes	6.7E-07
Total Hazard Index Across All Media and All Exposure Routes	1.5E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.8.6.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Construction Workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	1.8E-05	N/A	7.7E-07	1.9E-05	Antimony	longevity, glucose/ cholesterol--blood	6.1E-02	N/A	N/A	6.1E-02
								Arsenic	NOAEL/skin pigmentation	1.1E-01	N/A	4.8E-03	1.2E-01
								Cadmium	NOAEL/proteinuria--kidney	1.5E-02	N/A	5.0E-04	1.5E-02
								Iron	hematology--blood	1.7E-01	N/A	N/A	1.7E-01
								Manganese	NOAEL/Central Nervous System	1.9E-02	N/A	N/A	1.9E-02
								Zinc	enzyme effects--blood	8.2E-03	N/A	N/A	8.2E-03
			(Tota)	1.8E-05	N/A	7.7E-07	1.9E-05		(Total)	3.8E-01	N/A	5.3E-03	3.9E-01
						Total Risk Across Soil	1.9E-05					Total Hazard Index Across All Media and All Exposure Routes	3.9E-01
						Total Risk Across All Media and All Exposure Routes	1.9E-05						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.8.6.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Construction workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	6.1E-07	N/A	3.8E-08	6.4E-07	Antimony	longevity, glucose/ cholesterol--blood	7.5E-03	N/A	N/A	7.5E-03
								Arsenic	NOAEL/skin pigmentation	1.4E-02	N/A	8.9E-04	1.5E-02
								Cadmium	NOAEL/proteinuria--kidney	2.0E-03	N/A	1.0E-04	2.1E-03
								Iron	hematology--blood	2.4E-02	N/A	N/A	2.4E-02
								Manganese	NOAEL/Central Nervous System	2.6E-03	N/A	N/A	2.6E-03
								Zinc	enzyme effects--blood	1.1E-03	N/A	N/A	1.1E-03
			(Total)	6.1E-07	N/A	3.8E-08	6.4E-07	(Total)		5.2E-02	N/A	1.0E-03	5.3E-02
						Total Risk Across Soil	6.4E-07					Total Hazard Index Across All Media and All Exposure Routes	5.3E-02
						Total Risk Across All Media and All Exposure Routes	6.4E-07						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.9.1.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	6.4E-01	N/A	N/A	6.4E-01
								Arsenic	NOAEL/skin pigmentation	1.0E+00	N/A	1.1E-01	1.1E+00
								Cadmium	NOAEL/proteinuria--kidney	8.2E-02	N/A	7.2E-03	8.9E-02
								Iron	hematology--blood	1.1E+00	N/A	N/A	1.1E+00
								Manganese	NOAEL/Central Nervous System	4.4E-01	N/A	N/A	4.4E-01
								Zinc	enzyme effects--blood	5.9E-02	N/A	N/A	5.9E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		3.3E+00	N/A	1.2E-01	3.4E+00
Groundwater	Groundwater	Tap water											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	5.3E-02	N/A	N/A	5.3E-02

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	3.4E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.9.1.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.3E-01	N/A	N/A	1.3E-01
								Arsenic	NOAEL/skin pigmentation	2.6E-01	N/A	5.7E-02	3.2E-01
								Cadmium	NOAEL/proteinuria--kidney	2.4E-02	N/A	4.2E-03	2.8E-02
								Iron	hematology--blood	3.6E-01	N/A	N/A	3.6E-01
								Manganese	NOAEL/Central Nervous System	1.4E-01	N/A	N/A	1.4E-01
								Zinc	enzyme effects--blood	1.7E-02	N/A	N/A	1.7E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		9.3E-01	N/A	6.1E-02	9.9E-01
Groundwater	Groundwater	Tap water											
			Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.9E-02	N/A	N/A	1.9E-02

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	1.0E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.9.2.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	5.5E-05	N/A	6.3E-06	6.1E-05	Antimony	longevity, glucose/ cholesterol--blood	1.8E-01	N/A	N/A	1.8E-01
								Arsenic	NOAEL/skin pigmentation	2.9E-01	N/A	3.3E-02	3.2E-01
								Cadmium	NOAEL/proteinuria--kidney	2.3E-02	N/A	2.1E-03	2.5E-02
								Iron	hematology--blood	3.0E-01	N/A	N/A	3.0E-01
								Manganese	NOAEL/Central Nervous System	1.3E-01	N/A	N/A	1.3E-01
								Zinc	enzyme effects--blood	1.7E-02	N/A	N/A	1.7E-02
			(Total)	5.5E-05	N/A	6.3E-06	6.1E-05	(Total)		9.4E-01	N/A	3.5E-02	9.7E-01
Groundwater	Groundwater	Tap water											
			Arsenic	5.6E-06	N/A	N/A	5.6E-06	Arsenic	NOAEL/skin pigmentation	2.9E-02	N/A	N/A	2.9E-02

Total Risk Across Soil	6.1E-05
Total Risk Across Groundwater	5.6E-06
Total Risk Across All Media and All Exposure Routes	6.7E-05
Total Hazard Index Across All Media and All Exposure Routes	1.0E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.9.2.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil											
			Arsenic	4.6E-06	N/A	1.0E-06	5.6E-06	Antimony	longevity, glucose/ cholesterol--blood	4.0E-02	N/A	N/A	4.0E-02
								Arsenic	NOAEL/skin pigmentation	7.9E-02	N/A	1.8E-02	9.7E-02
								Cadmium	NOAEL/proteinuria--kidney	7.4E-03	N/A	1.3E-03	8.7E-03
								Iron	hematology--blood	1.1E-01	N/A	N/A	1.1E-01
								Manganese	NOAEL/Central Nervous System	4.4E-02	N/A	N/A	4.4E-02
								Zinc	enzyme effects--blood	5.1E-03	N/A	N/A	5.1E-03
			(Total)	4.6E-06	N/A	1.0E-06	5.6E-06	(Total)		2.9E-01	N/A	1.9E-02	3.0E-01
Groundwater	Groundwater	Tap water											
			Arsenic	4.9E-07	N/A	N/A	4.9E-07	Arsenic	NOAEL/skin pigmentation	8.4E-03	N/A	N/A	8.4E-03

Total Risk Across Soil	5.6E-06
Total Risk Across Groundwater	4.9E-07
Total Risk Across All Media and All Exposure Routes	6.1E-06
Total Hazard Index Across All Media and All Exposure Routes	3.1E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.9.3.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Waste piles	Arsenic	1.9E-06	N/A	6.4E-07	2.5E-06	Antimony	longevity, glucose/ cholesterol--blood	2.5E-02	N/A	N/A	2.5E-02
								Arsenic	NOAEL/skin pigmentation	4.2E-02	N/A	1.4E-02	5.6E-02
								Cadmium	NOAEL/proteinuria--kidney	6.9E-03	N/A	1.9E-03	8.8E-03
								Iron	hematology--blood	5.5E-02	N/A	N/A	5.5E-02
								Manganese	NOAEL/Central Nervous System	2.9E-02	N/A	N/A	2.9E-02
								Zinc	enzyme effects--blood	4.8E-03	N/A	N/A	4.8E-03
			(Total)	1.9E-06	N/A	6.4E-07	2.5E-06	(Total)		1.6E-01	N/A	1.6E-02	1.8E-01
Sediment	Sediment	SFCDAR	Arsenic	8.8E-07	N/A	6.8E-07	1.6E-06	Antimony	longevity, glucose/ cholesterol--blood	6.9E-03	N/A	N/A	6.9E-03
								Arsenic	NOAEL/skin pigmentation	2.0E-02	N/A	1.5E-02	3.5E-02
								Cadmium	NOAEL/proteinuria--kidney	1.2E-02	N/A	7.6E-03	2.0E-02
								Iron	hematology--blood	9.7E-02	N/A	N/A	9.7E-02
								Manganese	NOAEL/Central Nervous System	6.9E-02	N/A	N/A	6.9E-02
								Zinc	enzyme effects--blood	8.3E-03	N/A	N/A	8.3E-03
			(Total)	8.8E-07	N/A	6.8E-07	1.6E-06	(Total)		2.1E-01	N/A	2.3E-02	2.4E-01
Surface Water	Surface water	SFCDAR	Arsenic	2.5E-08	N/A	N/A	2.5E-08	Arsenic	NOAEL/skin pigmentation	5.6E-04	N/A	N/A	5.6E-04
								Cadmium	NOAEL/proteinuria--kidney	7.0E-04	N/A	N/A	7.0E-04
								Manganese	NOAEL/Central Nervous System	1.1E-04	N/A	N/A	1.1E-04
								Mercury	autoimmunity	8.1E-05	N/A	N/A	8.1E-05
			(Total)	2.5E-08	N/A	N/A	2.5E-08	(Total)		1.5E-03	N/A	N/A	1.5E-03

Total Risk Across Soil	2.5E-06
Total Risk Across Sediment	1.6E-06
Total Risk Across Surface water	2.5E-08
Total Risk Across All Media and All Exposure Routes	4.1E-06

Total Hazard Index Across All Media and All Exposure Routes	4.2E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.9.3.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Waste piles											
			Arsenic	6.1E-08	N/A	5.1E-08	1.1E-07	Antimony	longevity, glucose/ cholesterol--blood	3.0E-03	N/A	N/A	3.0E-03
								Arsenic	NOAEL/skin pigmentation	4.7E-03	N/A	4.0E-03	8.7E-03
								Cadmium	NOAEL/proteinuria--kidney	5.6E-04	N/A	3.8E-04	9.3E-04
								Iron	hematology--blood	7.4E-03	N/A	N/A	7.4E-03
								Manganese	NOAEL/Central Nervous System	4.5E-03	N/A	N/A	4.5E-03
								Zinc	enzyme effects--blood	3.5E-04	N/A	N/A	3.5E-04
			(Total)	6.1E-08	N/A	5.1E-08	1.1E-07	(Total)		2.0E-02	N/A	4.4E-03	2.5E-02
Sediment	Sediment	SFCDAR											
			Arsenic	4.0E-08	N/A	8.1E-08	1.2E-07	Antimony	longevity, glucose/ cholesterol--blood	7.7E-04	N/A	N/A	7.7E-04
								Arsenic	NOAEL/skin pigmentation	3.1E-03	N/A	6.3E-03	9.4E-03
								Cadmium	NOAEL/proteinuria--kidney	1.1E-03	N/A	1.8E-03	3.0E-03
								Iron	hematology--blood	1.3E-02	N/A	N/A	1.3E-02
								Manganese	NOAEL/Central Nervous System	8.8E-03	N/A	N/A	8.8E-03
								Zinc	enzyme effects--blood	8.6E-04	N/A	N/A	8.6E-04
			(Total)	4.0E-08	N/A	8.1E-08	1.2E-07	(Total)		2.8E-02	N/A	8.1E-03	3.6E-02
Surface Water	Surface water	SFCDAR											
			Arsenic	3.3E-09	N/A	N/A	3.3E-09	Arsenic	NOAEL/skin pigmentation	2.5E-04	N/A	N/A	2.5E-04
								Cadmium	NOAEL/proteinuria--kidney	2.3E-04	N/A	N/A	2.3E-04
								Manganese	NOAEL/Central Nervous System	4.1E-05	N/A	N/A	4.1E-05
								Mercury	autoimmunity	3.8E-05	N/A	N/A	3.8E-05
			(Total)	3.3E-09	N/A	N/A	3.3E-09	(Total)		5.6E-04	N/A	N/A	5.6E-04

Total Risk Across Soil	1.1E-07
Total Risk Across Sediment	1.2E-07
Total Risk Across Surface water	3.3E-09
Total Risk Across All Media and All Exposure Routes	2.4E-07
Total Hazard Index Across All Media and All Exposure Routes	6.1E-02

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.9.4.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Construction Workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	2.3E-05	N/A	9.7E-07	2.4E-05	Antimony	longevity, glucose/ cholesterol--blood	1.1E-01	N/A	N/A	1.1E-01
								Arsenic	NOAEL/skin pigmentation	1.4E-01	N/A	6.0E-03	1.5E-01
								Cadmium	NOAEL/proteinuria--kidney	1.2E-02	N/A	3.8E-04	1.2E-02
								Iron	hematology--blood	2.1E-01	N/A	N/A	2.1E-01
								Manganese	NOAEL/Central Nervous System	3.2E-02	N/A	N/A	3.2E-02
								Zinc	enzyme effects--blood	7.3E-03	N/A	N/A	7.3E-03
			(Total)	2.3E-05	N/A	9.7E-07	2.4E-05	(Total)		5.1E-01	N/A	6.4E-03	5.1E-01
						Total Risk Across Soil	2.4E-05					Total Hazard Index Across All Media and All Exposure Routes	5.1E-01
						Total Risk Across All Media and All Exposure Routes	2.4E-05						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.9.4.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Construction workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	7.6E-07	N/A	4.7E-08	8.0E-07	Antimony	longevity, glucose/ cholesterol--blood	1.2E-02	N/A	N/A	1.2E-02
								Arsenic	NOAEL/skin pigmentation	1.8E-02	N/A	1.1E-03	1.9E-02
								Cadmium	NOAEL/proteinuria--kidney	1.4E-03	N/A	7.1E-05	1.5E-03
								Iron	hematology--blood	2.8E-02	N/A	N/A	2.8E-02
								Manganese	NOAEL/Central Nervous System	4.1E-03	N/A	N/A	4.1E-03
								Zinc	enzyme effects--blood	9.2E-04	N/A	N/A	9.2E-04
			(Total)	7.6E-07	N/A	4.7E-08	8.0E-07	(Total)		6.4E-02	N/A	1.2E-03	6.5E-02
						Total Risk Across Soil	8.0E-07					Total Hazard Index Across All Media and All Exposure Routes	6.5E-02
						Total Risk Across All Media and All Exposure Routes	8.0E-07						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.10.1.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Blackwell Island

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Spokane River	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.4E-02	N/A	N/A	1.4E-02
								Arsenic	NOAEL/skin pigmentation	1.8E-01	N/A	3.8E-02	2.1E-01
								Cadmium	NOAEL/proteinuria--kidney	2.3E-02	N/A	4.1E-03	2.8E-02
								Iron	hematology--blood	2.9E-01	N/A	N/A	2.9E-01
								Manganese	NOAEL/Central Nervous System	1.4E-01	N/A	N/A	1.4E-01
								Zinc	enzyme effects--blood	1.1E-02	N/A	N/A	1.1E-02
			(Total)	N/A	N/A	N/A	N/A	(Total)		6.5E-01	N/A	4.2E-02	6.9E-01
Surface Water	Surface water	Spokane River	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.2E-02	N/A	N/A	1.2E-02
								Cadmium	NOAEL/proteinuria--kidney	1.7E-02	N/A	N/A	1.7E-02
								Manganese	NOAEL/Central Nervous System	2.6E-02	N/A	N/A	2.6E-02
								Mercury	autoimmunity	5.8E-05	N/A	N/A	5.8E-05
			(Total)	N/A	N/A	N/A	N/A	(Total)		5.5E-02	N/A	N/A	5.5E-02
						Total Risk Across Soil/sediment	N/A					Total Hazard Index Across All Media and All Exposure Routes	7.4E-01
						Total Risk Across Surface water	N/A						
						Total Risk Across All Media and All Exposure Routes	N/A						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.10.1.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Blackwell Island

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Spokane River	Arsenic	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	2.4E-03	N/A	N/A	2.4E-03
								Arsenic	NOAEL/skin pigmentation	2.9E-02	N/A	1.6E-02	4.5E-02
								Cadmium	NOAEL/proteinuria--kidney	3.9E-03	N/A	1.7E-03	5.6E-03
								Iron	hematology--blood	5.1E-02	N/A	N/A	5.1E-02
								Manganese	NOAEL/Central Nervous System	2.2E-02	N/A	N/A	2.2E-02
								Zinc	enzyme effects--blood	1.8E-03	N/A	N/A	1.8E-03
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.1E-01	N/A	1.7E-02	1.3E-01
Surface Water	Surface water	Spokane River	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	3.1E-03	N/A	N/A	3.1E-03
								Cadmium	NOAEL/proteinuria--kidney	3.5E-03	N/A	N/A	3.5E-03
								Manganese	NOAEL/Central Nervous System	5.2E-03	N/A	N/A	5.2E-03
								Mercury	autoimmunity	2.6E-05	N/A	N/A	2.6E-05
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.2E-02	N/A	N/A	1.2E-02

Total Risk Across Soil/sediment	N/A
Total Risk Across Surface water	N/A
Total Risk Across All Media and All Exposure Routes	N/A
Total Hazard Index Across All Media and All Exposure Routes	1.4E-01

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.10.2.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Blackwell Island

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Spokane River	Arsenic	8.7E-06	N/A	3.2E-06	1.2E-05	Antimony	longevity, glucose/ cholesterol--blood	3.6E-03	N/A	N/A	3.6E-03
								Arsenic	NOAEL/skin pigmentation	4.5E-02	N/A	1.7E-02	6.2E-02
								Cadmium	NOAEL/proteinuria--kidney	6.0E-03	N/A	1.8E-03	7.8E-03
								Iron	hematology--blood	7.3E-02	N/A	N/A	7.3E-02
								Manganese	NOAEL/Central Nervous System	3.5E-02	N/A	N/A	3.5E-02
								Zinc	enzyme effects--blood	2.7E-03	N/A	N/A	2.7E-03
			(Total)	8.7E-06	N/A	3.2E-06	1.2E-05	(Total)		1.7E-01	N/A	1.8E-02	1.8E-01
Surface Water	Surface water	Spokane River	Arsenic	8.7E-07	N/A	N/A	8.7E-07	Arsenic	NOAEL/skin pigmentation	4.5E-03	N/A	N/A	4.5E-03
								Cadmium	NOAEL/proteinuria--kidney	6.2E-03	N/A	N/A	6.2E-03
								Manganese	NOAEL/Central Nervous System	9.7E-03	N/A	N/A	9.7E-03
								Mercury	autoimmunity	2.2E-05	N/A	N/A	2.2E-05
			(Total)	8.7E-07	N/A	N/A	8.7E-07	(Total)		2.0E-02	N/A	N/A	2.0E-02
						Total Risk Across Soil/sediment	1.2E-05					Total Hazard Index Across All Media and All Exposure Routes	2.0E-01
						Total Risk Across Surface water	8.7E-07						
						Total Risk Across All Media and All Exposure Routes	1.3E-05						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.10.2.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Blackwell Island

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Spokane River	Arsenic	4.9E-07	N/A	4.1E-07	8.9E-07	Antimony	longevity, glucose/ cholesterol--blood	7.1E-04	N/A	N/A	7.1E-04
								Arsenic	NOAEL/skin pigmentation	8.4E-03	N/A	7.1E-03	1.5E-02
								Cadmium	NOAEL/proteinuria--kidney	1.1E-03	N/A	7.7E-04	1.9E-03
								Iron	hematology--blood	1.5E-02	N/A	N/A	1.5E-02
								Manganese	NOAEL/Central Nervous System	6.5E-03	N/A	N/A	6.5E-03
								Zinc	enzyme effects--blood	5.4E-04	N/A	N/A	5.4E-04
			(Total)	4.9E-07	N/A	4.1E-07	8.9E-07	(Total)		3.2E-02	N/A	7.8E-03	4.0E-02
Surface Water	Surface water	Spokane River	Arsenic	6.9E-08	N/A	N/A	6.9E-08	Arsenic	NOAEL/skin pigmentation	1.2E-03	N/A	N/A	1.2E-03
								Cadmium	NOAEL/proteinuria--kidney	1.4E-03	N/A	N/A	1.4E-03
								Manganese	NOAEL/Central Nervous System	2.0E-03	N/A	N/A	2.0E-03
								Mercury	autoimmunity	1.0E-05	N/A	N/A	1.0E-05
			(Total)	6.9E-08	N/A	N/A	6.9E-08	(Total)		4.6E-03	N/A	N/A	4.6E-03
						Total Risk Across Soil/sediment	8.9E-07					Total Hazard Index Across All Media and All Exposure Routes	4.5E-02
						Total Risk Across Surface water	6.9E-08						
						Total Risk Across All Media and All Exposure Routes	9.6E-07						

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 9.11.1.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Vegetable Ingestion in Residential Areas

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Plant tissue	Homegrown vegetables	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	4.2E-01	N/A	N/A	4.2E-01
								Cadmium	NOAEL/proteinuria--kidney	1.6E+00	N/A	N/A	1.6E+00
								(Total)		2.0E+00	N/A	N/A	2.0E+00
							Total Risk Across Soil						Total Hazard Index Across All Media and All Exposure Routes
							N/A						2.0E+00
							Total Risk Across All Media and All Exposure Routes						
							N/A						

N/A = not applicable

TABLE 9.11.1.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Vegetable Ingestion in Residential Areas

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Plant tissue	Homegrown vegetables	Arsenic	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	3.3E-02	N/A	N/A	3.3E-02
								Cadmium	NOAEL/proteinuria--kidney	1.0E-01	N/A	N/A	1.0E-01
								(Total)		1.4E-01	N/A	N/A	1.4E-01
							Total Risk Across Soil						Total Hazard Index Across All Media and All Exposure Routes
							N/A						1.4E-01
							Total Risk Across All Media and All Exposure Routes						
							N/A						

N/A = not applicable

TABLE 9.11.2.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Vegetable Ingestion in Residential Areas

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Plant tissue	Homegrown vegetables	Arsenic	8.1E-05	N/A	N/A	8.1E-05	Arsenic	NOAEL/skin pigmentation	4.2E-01	N/A	N/A	4.2E-01
								Cadmium	NOAEL/proteinuria--kidney	1.6E+00	N/A	N/A	1.6E+00
			(Total)	8.1E-05	N/A	N/A	8.1E-05	(Total)		2.0E+00	N/A	N/A	2.0E+00
						Total Risk Across Soil	8.1E-05					Total Hazard Index Across All Media and All Exposure Routes	2.0E+00
						Total Risk Across All Media and All Exposure Routes	8.1E-05						

N/A = not applicable

TABLE 9.11.2.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Vegetable Ingestion in Residential Areas

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Plant tissue	Homegrown vegetables	Arsenic	1.9E-06	N/A	N/A	1.9E-06	Arsenic	NOAEL/skin pigmentation	3.3E-02	N/A	N/A	3.3E-02
								Cadmium	NOAEL/proteinuria--kidney	1.0E-01	N/A	N/A	1.0E-01
			(Total)	1.9E-06	N/A	N/A	1.9E-06	(Total)		1.4E-01	N/A	N/A	1.4E-01
						Total Risk Across Soil	1.9E-06					Total Hazard Index Across All Media and All Exposure Routes	1.4E-01
						Total Risk Across All Media and All Exposure Routes	1.9E-06						

N/A = not applicable

TABLE 9.11.3.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point (1)	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface water/sediment	Animal Tissue	Pike (fish) in Lower CDAR	N/A	N/A	N/A	N/A	N/A	Cadmium	NOAEL/proteinuria--kidney	3.9E-03	N/A	N/A	3.9E-03
								Mercury	autoimmunity	8.7E-01	N/A	N/A	8.7E-01
								(Total)		8.8E-01	N/A	N/A	8.8E-01
						Total Risk Across Surface water/sediment	N/A					Total Hazard Index Across All Media and All Exposure Routes	8.8E-01
						Total Risk Across All Media and All Exposure Routes	N/A						

(1) Ingestion of fish tissue as shown assumes all fish ingested are pike. Pike was selected over perch and bullhead because the greatest risk due to fish tissue ingestion was associated with pike. Risks would be less for ingestion of perch or bullhead.

N/A = not applicable

TABLE 9.11.3.CT

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point (1)	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface water/sediment	Animal Tissue	Pike (fish) in the Lower CDAR	N/A	N/A	N/A	N/A	N/A	Cadmium	NOAEL/proteinuria--kidney	3.6E-03	N/A	N/A	3.6E-03
								Mercury	autoimmunity	4.3E-01	N/A	N/A	4.3E-01
								(Total)		4.3E-01	N/A	N/A	4.3E-01
						Total Risk Across Surface water/sediment	N/A					Total Hazard Index Across All Media and All Exposure Routes	4.3E-01
						Total Risk Across All Media and All Exposure Routes	N/A						

(1) Ingestion of fish tissue as shown assumes all fish ingested are pike. Pike was selected over perch and bullhead because the greatest risk due to fish tissue ingestion was associated with pike. Risks would be less for ingestion of perch or bullhead.

N/A = not applicable

TABLE 9.11.4.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Modern Subsistence Exposure Scenario - Fish Ingestion

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point (1)	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface water/sediment	Animal Tissue	Pike (fish) in Lower CDAR	N/A	N/A	N/A	N/A	N/A	Cadmium	NOAEL/proteinuria--kidney	1.5E-02	N/A	N/A	1.5E-02
								Mercury	autoimmunity	3.2E+00	N/A	N/A	3.2E+00
								(Total)		3.2E+00	N/A	N/A	3.2E+00
						Total Risk Across Surface water/sediment	N/A					Total Hazard Index Across All Media and All Exposure Routes	3.2E+00
						Total Risk Across All Media and All Exposure Routes	N/A						

(1) Ingestion of fish tissue as shown assumes all fish ingested are pike. Pike was selected over perch and bullhead because the greatest risk due to fish tissue ingestion was associated with pike. Risks would be less for ingestion of perch or bullhead.

N/A = Not Applicable

TABLE 9.11.5.RME

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

Traditional Subsistence Exposure Scenario - Fish Ingestion

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point (1)	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface water/sediment	Animal Tissue	Pike (fish) in Lower CDAR	N/A	N/A	N/A	N/A	N/A	Cadmium	NOAEL/proteinuria--kidney	4.6E-02	N/A	N/A	4.6E-02
								Mercury	autoimmunity	1.0E+01	N/A	N/A	1.0E+01
								(Total)		1.0E+01	N/A	N/A	1.0E+01
						Total Risk Across Surface water/sediment	N/A					Total Hazard Index Across All Media and All Exposure Routes	1.0E+01
						Total Risk Across All Media and All Exposure Routes	N/A						

(1) Ingestion of fish tissue as shown assumes all fish ingested are pike. Pike was selected over perch and bullhead because the greatest risk due to fish tissue ingestion was associated with pike. Risks would be less for ingestion of perch or bullhead.

N/A = not applicable

TABLE 10.1.1.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil						Arsenic	NOAEL/skin pigmentation	1.2E+00	N/A	1.4E-01	1.4E+00
								Iron	hematology--blood	1.6E+00	N/A	N/A	1.6E+00
			N/A	N/A	N/A	N/A	N/A	(Total)		2.8E+00	N/A	1.4E-01	3.0E+00
							Total Risk Across Soil					Total Hazard Index Across All Media and All Exposure Routes	3.0E+00
							Total Risk Across All Media and All Exposure Routes						
							N/A						

Total skin HI =	1.4E+00
Total blood HI =	1.6E+00

N/A = not applicable

TABLE 10.1.2.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	6.0E-06	N/A	1.4E-06	7.3E-06	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	1.4E-06	N/A	N/A	1.4E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	7.3E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	1.4E-06						
						Total Risk Across All Media and All Exposure Routes	8.7E-06						

N/A = not applicable

TABLE 10.1.2.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	6.8E-05	N/A	7.8E-06	7.6E-05	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	2.2E-05	N/A	N/A	2.2E-05	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	7.6E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	2.2E-05						
						Total Risk Across All Media and All Exposure Routes	9.8E-05						

N/A = not applicable

TABLE 10.1.3.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Lower CDAR	Arsenic	3.5E-07	N/A	1.1E-06	1.4E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil/sediment	1.4E-06		Total Hazard Index Across All Media and All Exposure Routes				N/A
						Total Risk Across All Media and All Exposure Routes	1.4E-06						

N/A = not applicable

TABLE 10.1.3.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Lower CDAR	Arsenic	6.6E-06	N/A	8.0E-06	1.5E-05	N/A	N/A	N/A	N/A	N/A	N/A
Surface Water	Disturbed surface water	Lower CDAR	Arsenic	3.4E-06	N/A	N/A	3.4E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil/sediment	1.5E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Surface water	3.4E-06						
						Total Risk Across All Media and All Exposure Routes	1.8E-05						

N/A = not applicable

TABLE 10.1.4.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Lower CDAR	Arsenic	1.4E-06	N/A	1.1E-06	2.5E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil/sediment	2.5E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	2.5E-06						

N/A = not applicable

TABLE 10.1.4.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Lower CDAR	Arsenic	2.1E-05	N/A	7.7E-06	2.8E-05	N/A	N/A	N/A	N/A	N/A	N/A
Surface Water	Disturbed surface water	Lower CDAR	Arsenic	3.3E-06	N/A	N/A	3.3E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil/sediment	2.8E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Surface water	3.3E-06						
						Total Risk Across All Media and All Exposure Routes	3.2E-05						

N/A = not applicable

TABLE 10.1.5.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Construction workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	3.0E-06	N/A	1.9E-07	3.2E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	3.2E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	3.2E-06						

N/A = not applicable

TABLE 10.1.5.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Current/Future
Receptor Population:	Construction Workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	8.0E-05	N/A	3.3E-06	8.3E-05	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	8.3E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	8.3E-05						

N/A = not applicable

TABLE 10.1.6

RISK ASSESSMENT SUMMARY

MODERN SUBSISTENCE - REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Lower CDAR	N/A	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	8.2E-01	N/A	3.1E-01	1.1E+00
								Iron	hematology--blood	1.1E+00	N/A	N/A	1.1E+00
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.9E+00	N/A	3.1E-01	2.2E+00
Sediment	Sediment	Lower CDAR	N/A	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	8.0E-01	N/A	6.9E-01	1.5E+00
								Iron	hematology--blood	1.2E+00	N/A	N/A	1.2E+00
			(Total)	N/A	N/A	N/A	N/A	(Total)		2.0E+00	N/A	6.9E-01	2.7E+00
Undisturbed Surface Water	Undisturbed Surface Water	Lower CDAR	N/A	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.1E+00	N/A	N/A	1.1E+00
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.1E+00	N/A	N/A	1.1E+00
Disturbed Surface Water	Disturbed Surface Water	Lower CDAR	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
			(Total)	N/A	N/A	N/A	N/A	(Total)		N/A	N/A	N/A	N/A

Total Risk Across Soil	N/A
Total Risk Across Sediment	N/A
Total Risk Across Undisturbed Surface Water	N/A
Total Risk Across Disturbed Surface Water	N/A
Total Risk Across All Media and All Exposure Routes	N/A

Total Hazard Index Across All Media and All Exposure Routes	6.0E+00
Total skin HI =	3.7E+00
Total blood HI =	2.3E+00

N/A = not applicable

TABLE 10.1.7

Risk Assessment Summary

MODERN SUBSISTENCE - REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Lower CDAR	Arsenic	1.0E-04	N/A	6.7E-05	1.7E-04	N/A	N/A	N/A	N/A	N/A	N/A
			(Total)	1.0E-04	N/A	6.7E-05	1.7E-04	(Total)		N/A	N/A	N/A	N/A
Sediment	Sediment	Lower CDAR	Arsenic	1.0E-04	N/A	1.9E-04	3.0E-04	Arsenic	NOAEL/skin pigmentation	2.2E-01	N/A	4.3E-01	6.6E-01
								Iron	hematology--blood	3.5E-01	N/A	N/A	3.5E-01
			(Total)	1.0E-04	N/A	1.9E-04	3.0E-04	(Total)		5.7E-01	N/A	4.3E-01	1.0E+00
Undisturbed Surface Water	Undisturbed Surface Water	Lower CDAR	Arsenic	2.4E-04	N/A	N/A	2.4E-04	Arsenic	NOAEL/skin pigmentation	5.2E-01	N/A	N/A	5.2E-01
			(Total)	2.4E-04	N/A	N/A	2.4E-04	(Total)		5.2E-01	N/A	N/A	5.2E-01
Disturbed Surface Water	Disturbed Surface Water	Lower CDAR	Arsenic	1.11E-05	N/A	N/A	1.1E-05	N/A	N/A	N/A	N/A	N/A	N/A
			(Total)	1.11E-05	N/A	N/A	1.1E-05	(Total)		N/A	N/A	N/A	N/A

Total Risk Across Soil	1.7E-04
Total Risk Across Sediment	3.0E-04
Total Risk Across Undisturbed Surface Water	2.4E-04
Total Risk Across Disturbed Surface Water	1.1E-05
Total Risk Across All Media and All Exposure Routes	7.1E-04

Total Hazard Index Across All Media and All Exposure Routes	1.5E+00
Total skin HI =	1.2E+00
Total blood HI =	3.5E-01

N/A = not applicable

TABLE 10.1.8

RISK ASSESSMENT SUMMARY

TRADITIONAL SUBSISTENCE - REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water/Sediment	Plant Tissue	Water Potato (With Skin)	N/A	N/A	N/A	N/A	N/A	Cadmium	NOAEL/proteinuria--kidney	4.0E+00	N/A	N/A	4.0E+00
			(Total)	N/A	N/A	N/A	N/A	(Total)		4.0E+00	N/A	0.0E+00	4.0E+00
			Total Risk Across All Media and All Exposure Routes				N/A	Total Hazard Index Across All Media and All Exposure Routes					4.0E+00

(1) Total organ Hazard Indices were not calculated because the effects on each target organ differs, and are therefore not additive. Specifically, although the effects of antimony, iron, and zinc can all be measured in blood, each chemical affects blood chemistry in a different and unrelated way (serum glucose/cholesterol, serum ferritin and percent transferrin saturation, and serum superoxide dismutase activity for antimony, iron, and zinc, respectively).

N/A = not applicable

TABLE 10.1.9

RISK ASSESSMENT SUMMARY

TRADITIONAL SUBSISTENCE - REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Lower CDAR	N/A	N/A	N/A	N/A	N/A	Antimony	longevity, glucose/ cholesterol--blood	1.1E+00	N/A	N/A	1.1E+00
								Arsenic	NOAEL/skin pigmentation	5.0E+00	N/A	1.5E+00	6.4E+00
								Iron	hematology--blood	6.5E+00	N/A	N/A	6.5E+00
								Manganese	NOAEL/Central Nervous System	3.8E+00	N/A	N/A	3.8E+00
			(Total)	N/A	N/A	N/A	N/A	(Total)		1.6E+01	N/A	1.5E+00	1.8E+01
Sediment	Sediment	Lower CDAR	N/A	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	2.8E+00	N/A	2.4E+00	5.2E+00
								Iron	hematology--blood	4.3E+00	N/A	N/A	4.3E+00
			(Total)	N/A	N/A	N/A	N/A	(Total)		7.1E+00	N/A	2.4E+00	9.5E+00
Undisturbed Surface Water	Undisturbed Surface Water	Lower CDAR	N/A	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	6.7E+00	N/A	N/A	6.7E+00
			(Total)	N/A	N/A	N/A	N/A	(Total)		6.7E+00	N/A	N/A	6.7E+00
Disturbed Surface Water	Disturbed Surface Water	Lower CDAR	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
			(Total)	N/A	N/A	N/A	N/A	(Total)	N/A	N/A	N/A	N/A	N/A

Total Risk Across Soil	N/A
Total Risk Across Sediment	N/A
Total Risk Across Undisturbed Surface Water	N/A
Total Risk Across Disturbed Surface Water	N/A
Total Risk Across All Media and All Exposure Routes	N/A

Total Hazard Index Across All Media and All Exposure Routes	3.4E+01
Total skin HI =	1.8E+01
Total blood HI =	1.2E+01
Total CNS =	3.8E+00

N/A = not applicable

TABLE 10.1.10

RISK ASSESSMENT SUMMARY

TRADITIONAL SUBSISTENCE - REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Lower CDAR	Arsenic	6.3E-04	N/A	2.0E-04	8.3E-04	Arsenic	NOAEL/skin pigmentation	1.4E+00	N/A	4.5E-01	1.9E+00
								Iron	hematology--blood	1.8E+00	N/A	N/A	1.8E+00
			(Total)	6.3E-04	N/A	2.0E-04	8.3E-04	(Total)		3.2E+00	N/A	4.5E-01	3.7E+00
Sediment	Sediment	Lower CDAR	Arsenic	3.5E-04	N/A	6.8E-04	1.0E-03	Arsenic	NOAEL/skin pigmentation	7.8E-01	N/A	1.5E+00	2.3E+00
								Iron	hematology--blood	1.2E+00	N/A	N/A	1.2E+00
			(Total)	3.5E-04	N/A	6.8E-04	1.0E-03	(Total)		2.0E+00	N/A	1.5E+00	3.5E+00
Undisturbed Surface Water	Undisturbed Surface Water	Lower CDAR	Arsenic	1.4E-03	N/A	N/A	1.4E-03	Arsenic	NOAEL/skin pigmentation	3.2E+00	N/A	N/A	3.2E+00
			(Total)	1.43E-03	N/A	N/A	1.4E-03	(Total)		3.2E+00	N/A	N/A	3.2E+00
Disturbed Surface Water	Disturbed Surface Water	Lower CDAR	Arsenic	3.87E-05	N/A	N/A	3.9E-05	N/A	N/A	N/A	N/A	N/A	N/A
			(Total)	3.9E-05	N/A	N/A	3.9E-05	(Total)		N/A	N?A	N/A	N/A

Total Risk Across Soil	8.3E-04
Total Risk Across Sediment	1.0E-03
Total Risk Across Undisturbed Surface Water	1.4E-03
Total Risk Across Disturbed Surface Water	3.9E-05
Total Risk Across All Media and All Exposure Routes	3.3E-03

Total Hazard Index Across All Media and All Exposure Routes	1.0E+01
Total skin HI =	7.3E+00
Total blood HI =	3.1E+00

N/A = not applicable

TABLE10.2.1.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	N/A	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	6.4E-01	N/A	7.0E-02	7.1E-01
						Total Risk Across Soil	N/A					Total Hazard Index Across All Media and All Exposure Routes	7.1E-01
						Total Risk Across Groundwater	N/A						
						Total Risk Across All Media and All Exposure Routes	N/A					Total skin HI =	7.1E-01

N/A = not applicable

TABLE 10.2.2.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	3.4E-06	N/A	7.8E-07	4.2E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	4.2E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	4.2E-06						

N/A = not applicable

TABLE 10.2.2.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	3.5E-05	N/A	4.0E-06	3.9E-05	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	9.0E-06	N/A	N/A	9.0E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	3.9E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	9.0E-06						
						Total Risk Across All Media and All Exposure Routes	4.8E-05						

N/A = not applicable

TABLE 10.2.3.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	NS Confluence (1)	Arsenic	4.4E-07	N/A	1.4E-06	1.8E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil/sediment	1.8E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	1.8E-06						

(1) Both Pine Creek and NS Confluence were sampled and risks calculated. When determining total risk across media and across all media and exposure routes, it was assumed that all exposure occurred at the NS Confluence be
Therefore, total risk estimates across media and across all media and exposure routes for this receptor scenario are higher (more conservative) than those associated with exposure to Pine Creek sediment and water.

N/A = not applicable

TABLE 10.2.3.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	NS Confluence (1)	Arsenic	9.1E-06	N/A	1.1E-05	2.0E-05	N/A	N/A	N/A	N/A	N/A	N/A
Surface Water	Surface water	NS Confluence (1)	Arsenic	5.7E-06	N/A	N/A	5.7E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil/sediment	2.0E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Surface water	5.7E-06						
						Total Risk Across All Media and All Exposure Routes	2.6E-05						

N/A = not applicable

TABLE 10.2.4.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	NS Confluence	N/A	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	5.7E-01	N/A	1.2E-01	7.0E-01
						Total Risk Across Soil/sediment	NA					Total Hazard Index Across All Media and All Exposure Routes	7.0E-01
						Total Risk Across Surface water	N/A						
						Total Risk Across All Media and All Exposure Routes	N/A					Total skin HI =	7.0E-01

N/A = not applicable

TABLE 10.2.5.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	NS Confluence	Arsenic	1.7E-06	N/A	1.4E-06	3.2E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil/sediment	3.2E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	3.2E-06						

N/A = not applicable

TABLE 10.2.5.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	NS Confluence	Arsenic	2.8E-05	N/A	1.0E-05	3.9E-05	N/A	N/A	N/A	N/A	N/A	N/A
Surface Water	Surface water	NS Confluence	Arsenic	5.6E-06	N/A	N/A	5.6E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil/sediment	3.9E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Surface water	5.6E-06						
						Total Risk Across All Media and All Exposure Routes	4.4E-05						

N/A = not applicable

TABLE 10.2.6.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Kingston

Scenario Timeframe:	Current/Future
Receptor Population:	Construction Workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	2.5E-05	N/A	1.1E-06	2.7E-05	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	2.7E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	2.7E-05						

N/A = not applicable

TABLE 10.3.1.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Side Gulches

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil						Arsenic	NOAEL/skin pigmentation	1.3E+00	N/A	1.4E-01	1.4E+00
								Iron	hematology--blood	1.2E+00	N/A	N/A	1.2E+00
			N/A	N/A	N/A	N/A	N/A	(Total)		2.5E+00	N/A	1.4E-01	2.6E+00
Groundwater	Groundwater	Tap water											
			N/A	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.8E+00	N/A	N/A	1.8E+00

Total Risk Across Soil	N/A
Total Risk Across Groundwater	N/A
Total Risk Across All Media and All Exposure Routes	N/A

Total Hazard Index Across All Media and All Exposure Routes	4.4E+00
Total skin HI =	3.2E+00
Total blood HI =	1.2E+00

N/A = not applicable

TABLE 10.3.2.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Side Gulches

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	6.7E-06	N/A	1.5E-06	8.2E-06	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	7.2E-06	N/A	N/A	7.2E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	8.2E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	7.2E-06						
						Total Risk Across All Media and All Exposure Routes	1.5E-05						

N/A = not applicable

TABLE 10.3.2.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Side Gulches

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	7.1E-05	N/A	8.2E-06	8.0E-05	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	1.9E-04	N/A	N/A	1.9E-04	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	8.0E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	1.9E-04						
						Total Risk Across All Media and All Exposure Routes	2.7E-04						

N/A = not applicable

TABLE 10.3.3.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Side Gulches

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Elk Creek Area	Arsenic	8.8E-06	N/A	3.0E-06	1.2E-05	N/A	N/A	N/A	N/A	N/A	N/A
Sediment	Sediment	Elk Creek Pond	Arsenic	4.3E-06	N/A	3.3E-06	7.7E-06	N/A	N/A	N/A	N/A	N/A	N/A
Surface Water	Surface water	Elk Creek Pond	Arsenic	1.4E-06	N/A	N/A	1.4E-06	N/A	N/A	N/A	N/A	N/A	N/A

Total Risk Across Soil	1.2E-05
Total Risk Across Sediment	7.7E-06
Total Risk Across Surface water	1.4E-06
Total Risk Across All Media and All Exposure Routes	2.1E-05

Total Hazard Index Across All Media and All Exposure Routes	N/A

N/A = not applicable

TABLE 10.4.1.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Osburn

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	N/A	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.2E+00	N/A	1.3E-01	1.3E+00
						Total Risk Across Soil	N/A					Total Hazard Index Across All Media and All Exposure Routes	1.3E+00
						Total Risk Across All Media and All Exposure Routes	N/A						
												Total skin HI =	1.3E+00

N/A = not applicable

TABLE 10.4.2.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENDCY EXPOSURE

Osburn

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	6.5E-06	N/A	1.5E-06	8.0E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	8.0E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	8.0E-06						

N/A = not applicable

TABLE 10.4.2.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Osburn

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	6.6E-05	N/A	7.5E-06	7.3E-05	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	6.2E-06	N/A	N/A	6.2E-06	N/A	N/A	N/A	N/A	N/A	N/A

Total Risk Across Soil	7.3E-05	Total Hazard Index Across All Media and All Exposure Routes	N/A
Total Risk Across Groundwater	6.2E-06		
Total Risk Across All Media and All Exposure Routes	8.0E-05		

N/A = not applicable

TABLE 10.5.1.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	2.8E-06	N/A	6.3E-07	3.4E-06	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	1.4E-06	N/A	N/A	1.4E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	3.4E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	1.4E-06						
						Total Risk Across All Media and All Exposure Routes	4.8E-06						

N/A = not applicable

TABLE 10.5.1.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	3.0E-05	N/A	3.5E-06	3.4E-05	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	1.2E-05	N/A	N/A	1.2E-05	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	3.4E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	1.2E-05						
						Total Risk Across All Media and All Exposure Routes	4.6E-05						

N/A = not applicable

TABLE 10.5.2.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	2.6E-06	N/A	8.7E-07	3.4E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	3.4E-06		Total Hazard Index Across All Media and All Exposure Routes				N/A
						Total Risk Across All Media and All Exposure Routes	3.4E-06						

N/A = not applicable

TABLE 10.5.3.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Silverton

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	5.1E-06	N/A	8.9E-07	6.0E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	6.0E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	6.0E-06						

N/A = not applicable

TABLE 10.6.1.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	N/A	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	5.8E-01	N/A	6.3E-02	6.4E-01
						Total Risk Across Soil	N/A					Total Hazard Index Across All Media and All Exposure Routes	6.4E-01
						Total Risk Across All Media and All Exposure Routes	N/A						
												Total skin HI =	6.4E-01

N/A = not applicable

TABLE 10.6.2.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	3.4E-06	N/A	7.8E-07	4.2E-06	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	1.6E-06	N/A	N/A	1.6E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	4.2E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	1.6E-06						
						Total Risk Across All Media and All Exposure Routes	5.8E-06						

N/A = not applicable

TABLE 10.6.2.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	3.2E-05	N/A	3.6E-06	3.5E-05	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	1.1E-05	N/A	N/A	1.1E-05	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	3.5E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	1.1E-05						
						Total Risk Across All Media and All Exposure Routes	4.6E-05						

N/A = not applicable

TABLE 10.6.3.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	2.5E-06	N/A	8.4E-07	3.3E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	3.3E-06		Total Hazard Index Across All Media and All Exposure Routes				N/A
						Total Risk Across All Media and All Exposure Routes	3.3E-06						

N/A = not applicable

TABLE 10.6.4.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Wallace

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Upland parks/schools	Arsenic	5.0E-06	N/A	8.7E-07	5.8E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	5.8E-06		Total Hazard Index Across All Media and All Exposure Routes				N/A
						Total Risk Across All Media and All Exposure Routes	5.8E-06						

N/A = not applicable

TABLE 10.7.1.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Osburn, Wallace, and Silverton Combined

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Sediment	SFCDAR	Arsenic	2.9E-06	N/A	2.3E-06	5.2E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil/sediment	5.2E-06					Total Hazard Index Across All Media and All Exposure Routes	N/A
						Total Risk Across All Media and All Exposure Routes	5.2E-06						

N/A = not applicable

TABLE 10.7.2.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Osburn, Wallace, and Silverton Combined

Scenario Timeframe:	Current/Future
Receptor Population:	Construction Workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	2.0E-05	N/A	8.4E-07	2.1E-05	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	2.1E-05		Total Hazard Index Across All Media and All Exposure Routes				N/A
						Total Risk Across All Media and All Exposure Routes	2.1E-05						

N/A = not applicable

TABLE 10.8.1.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	N/A	N/A	N/A	N/A	N/A	Arsenic	NOAEL/skin pigmentation	1.1E+00	N/A	1.2E-01	1.2E+00
						Total Risk Across Soil	N/A					Total Hazard Index Across All Media and All Exposure Routes	1.2E+00
						Total Risk Across All Media and All Exposure Routes	N/A						
												Total skin HI =	1.2E+00

N/A = not applicable

TABLE 10.8.2.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	5.3E-06	N/A	1.2E-06	6.5E-06	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	1.7E-06	N/A	N/A	1.7E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	6.5E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	1.7E-06						
						Total Risk Across All Media and All Exposure Routes	8.2E-06						

N/A = not applicable

TABLE 10.8.2.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	5.9E-05	N/A	6.7E-06	6.5E-05	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	1.7E-05	N/A	N/A	1.7E-05	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	6.5E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	1.7E-05						
						Total Risk Across All Media and All Exposure Routes	8.2E-05						

N/A = not applicable

TABLE 10.8.3.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Nine Mile

Scenario Timeframe:	Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Groundwater	Groundwater	Tap water						Cadmium	NOAEL/proteinuria--kidney	7.8E+00	N/A	N/A	7.8E+00
								Zinc	enzyme effects--blood	2.0E+00	N/A	N/A	2.0E+00
			N/A	N/A	N/A	N/A	N/A	(Total)		9.9E+00	N/A	N/A	9.9E+00
						Total Risk Across Groundwater	N/A					Total Hazard Index Across All Media and All Exposure Routes	9.9E+00
						Total Risk Across All Media and All Exposure Routes	N/A						

Total kidney HI =	7.8E+00
Total blood HI =	2.0E+00

N/A = not applicable

TABLE 10.8.3.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Groundwater	Groundwater	Tap water						Cadmium	NOAEL/proteinuria--kidney	1.7E+01	N/A	N/A	1.7E+01
								Zinc	enzyme effects--blood	4.2E+00	N/A	N/A	4.2E+00
			N/A	N/A	N/A	N/A	N/A	(Total)		2.1E+01	N/A	N/A	2.1E+01
							Total Risk Across Groundwater					Total Hazard Index Across All Media and All Exposure Routes	2.1E+01
							Total Risk Across All Media and All Exposure Routes						

Total kidney HI =	1.7E+01
Total blood HI =	4.2E+00

N/A = not applicable

TABLE 10.8.4.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Nine Mile

Scenario Timeframe:	Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Groundwater	Groundwater	Tap water	Arsenic	3.0E-06	N/A	N/A	3.0E-06	Cadmium	NOAEL/proteinuria--kidney	3.6E+00	N/A	N/A	3.6E+00
						Total Risk Across Groundwater	3.0E-06					Total Hazard Index Across All Media and All Exposure Routes	3.6E+00
						Total Risk Across All Media and All Exposure Routes	3.0E-06						
												Total kidney HI =	3.6E+00

N/A = not applicable

TABLE 10.8.4.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Groundwater	Groundwater	Tap water	Arsenic	2.8E-05	N/A	N/A	2.8E-05	Cadmium	NOAEL/proteinuria--kidney	9.1E+00	N/A	N/A	9.1E+00
								Zinc	enzyme effects--blood	2.3E+00	N/A	N/A	2.3E+00
			(Total)	2.8E-05	N/A	N/A	2.8E-05	(Total)		1.1E+01	N/A	N/A	1.1E+01
						Total Risk Across Groundwater	2.8E-05					Total Hazard Index Across All Media and All Exposure Routes	1.1E+01
						Total Risk Across All Media and All Exposure Routes	2.8E-05						

Total kidney HI =	9.1E+00
Total blood HI =	2.3E+00

N/A = not applicable

TABLE 10.8.5.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ (1)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Waste piles	Arsenic	2.3E-05	N/A	7.9E-06	3.1E-05	Arsenic	NOAEL/skin pigmentation	5.2E-01	N/A	1.8E-01	6.9E-01
Sediment	Sediment	Nine Mile/Canyon Creek	Arsenic	2.8E-06	N/A	2.2E-06	5.0E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	3.1E-05					Total Hazard Index Across All Media and All Exposure Routes	6.9E-01
						Total Risk Across Sediment	5.0E-06						
						Total Risk Across All Media and All Exposure Routes	3.6E-05					Total skin HI =	6.9E-01

N/A = not applicable

TABLE 10.8.6.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Nine Mile

Scenario Timeframe:	Current/Future
Receptor Population:	Construction Workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	1.8E-05	N/A	7.7E-07	1.9E-05	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	1.9E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	1.9E-05						

N/A = not applicable

TABLE 10.9.1.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil						Arsenic	NOAEL/skin pigmentation	1.0E+00	N/A	1.1E-01	1.1E+00
								Iron	hematology--blood	1.1E+00	N/A	N/A	1.1E+00
			N/A	N/A	N/A	N/A	N/A	(Total)		2.1E+00	N/A	1.1E-01	2.2E+00
							Total Risk Across Soil					Total Hazard Index Across All Media and All Exposure Routes	2.2E+00
							Total Risk Across All Media and All Exposure Routes						
							N/A						

Total skin HI =	1.1E+00
Total blood HI =	1.1E+00

N/A = not applicable

TABLE 10.9.2.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	4.6E-06	N/A	1.0E-06	5.6E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	5.6E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	5.6E-06						

N/A = not applicable

TABLE 10.9.2.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Yard soil	Arsenic	5.5E-05	N/A	6.3E-06	6.1E-05	N/A	N/A	N/A	N/A	N/A	N/A
Groundwater	Groundwater	Tap water	Arsenic	5.6E-06	N/A	N/A	5.6E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	6.1E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Groundwater	5.6E-06						
						Total Risk Across All Media and All Exposure Routes	6.7E-05						

N/A = not applicable

TABLE 10.9.3.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Neighborhood residents
Receptor Age:	Child 4-11 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface soil	Waste piles	Arsenic	1.9E-06	N/A	6.4E-07	2.5E-06	N/A	N/A	N/A	N/A	N/A	N/A
Sediment	Sediment	SFCDAR	Arsenic	8.8E-07	N/A	6.8E-07	1.6E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	2.5E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across Sediment	1.6E-06						
						Total Risk Across All Media and All Exposure Routes	4.1E-06						

N/A = not applicable

TABLE 10.9.4.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Mullan

Scenario Timeframe:	Current/Future
Receptor Population:	Construction Workers
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Subsurface/surface soil	Construction site soil	Arsenic	2.3E-05	N/A	9.7E-07	2.4E-05	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	2.4E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	2.4E-05						

N/A = not applicable

TABLE 10.10.1.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Blackwell Island

Scenario Timeframe:	Current/Future
Receptor Population:	Visitors
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil/Sediment	Soil/sediment	Spokane River	Arsenic	8.7E-06	N/A	3.2E-06	1.2E-05	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil/sediment	1.2E-05	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	1.2E-05						

N/A = not applicable

TABLE 10.11.1.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Vegetable Ingestion in Residential Areas

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child 0-6 years old

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Plant tissue	Homegrown vegetables	N/A	N/A	N/A	N/A	N/A	Cadmium	NOAEL/proteinuria--kidney	1.6E+00	N/A	N/A	1.6E+00
						Total Risk Across Soil	N/A					Total Hazard Index Across All Media and All Exposure Routes	1.6E+00
						Total Risk Across All Media and All Exposure Routes	N/A						
												Total kidney HI =	1.6E+00

N/A = not applicable

TABLE 10.11.2.CT

RISK ASSESSMENT SUMMARY

CENTRAL TENDENCY EXPOSURE

Vegetable Ingestion in Residential Areas

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Plant tissue	Homegrown vegetables	Arsenic	1.9E-06	N/A	N/A	1.9E-06	N/A	N/A	N/A	N/A	N/A	N/A
						Total Risk Across Soil	1.9E-06	Total Hazard Index Across All Media and All Exposure Routes					N/A
						Total Risk Across All Media and All Exposure Routes	1.9E-06						

N/A = not applicable

TABLE 10.11.2.RME

RISK ASSESSMENT SUMMARY

REASONABLE MAXIMUM EXPOSURE

Vegetable Ingestion in Residential Areas

Scenario Timeframe:	Current/Future
Receptor Population:	Residents
Receptor Age:	Child/Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Plant tissue	Homegrown vegetables	Arsenic	8.1E-05	N/A	N/A	8.1E-05	Cadmium	NOAEL/proteinuria--kidney	1.6E+00	N/A	N/A	1.6E+00
						Total Risk Across Soil	8.1E-05					Total Hazard Index Across All Media and All Exposure Routes	1.6E+00
						Total Risk Across All Media and All Exposure Routes	8.1E-05						
												Total kidney HI =	1.6E+00

N/A = not applicable

TABLE 10.11.3

RISK ASSESSMENT SUMMARY

MODERN SUBSISTENCE - REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface water/sediment	Animal Tissue	Fish in Lower CDAR Nothern Pike	N/A	N/A	N/A	N/A	N/A	Mercury	autoimmunity	1.1E+00	N/A	N/A	3.2E+00
								(Total)		1.1E+00	N/A	N/A	3.2E+00
						Total Risk Across Surface water/sediment	N/A					Total Hazard Index Across All Media and All Exposure Routes	3.2E+00
						Total Risk Across All Media and All Exposure Routes	N/A						
												Total autoimmunity HI=	3.2E+00

N/A = Not Applicable

TABLE 10.11.4

RISK ASSESSMENT SUMMARY

TRADITIONAL SUBSISTENCE - REASONABLE MAXIMUM EXPOSURE

Lower Basin

Scenario Timeframe:	Future
Receptor Population:	Tribal Member
Receptor Age:	Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface water/sediment	Animal Tissue	Fish in Lower CDAR Nothern Pike	N/A	N/A	N/A	N/A	N/A	Mercury	autoimmunity	1.0E+01	N/A	N/A	1.0E+01
								(Total)		1.0E+01	N/A	N/A	1.0E+01
						Total Risk Across Surface water/sediment	N/A					Total Hazard Index Across All Media and All Exposure Routes	1.0E+01
						Total Risk Across All Media and All Exposure Routes	N/A						
												Total autoimmunity HI =	1.0E+01

N/A = not applicable

APPENDIX B

Expedited Screening Level Risk Assessment for Common Use Areas, Coeur d'Alene River Basin

Appendix B

Expedited Screening Level Risk Assessment for Common Use Areas, Coeur d'Alene River Basin

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This appendix consists of the screening level risk assessment report for Coeur d'Alene Lake completed in October 1999. It is a part of this baseline risk assessment for the entire Coeur d'Alene basin.

DRAFT FINAL
COEUR D'ALENE BASIN RI/FS
EXPEDITED SCREENING LEVEL RISK ASSESSMENT FOR COMMON USE AREAS
COEUR D'ALENE RIVER BASIN, IDAHO

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October 18, 1999

EXECUTIVE SUMMARY

This report evaluates metal concentrations in soil and water located on public and private lands around Coeur d'Alene Lake (Lake) and the Spokane River. Many of the areas surrounding the Lake and the River are used for recreational purposes. Concern over the presence of excessive metal concentrations from past mining activities in these areas prompted investigations at beaches and other common use areas (CUAs) throughout the Coeur d'Alene River Basin. Mining-impacted materials were anticipated to be present near the water bodies capable of transporting metals generated by upstream mining activities.

The EPA, the local health department, and Bureau of Land Management personnel familiar with the area selected beaches and parks used by the public as areas of concern. Sampling activities were conducted at 24 CUAs around the Lake and the Spokane River. Analytical results were compared to risk-based screening concentrations (RBCs) considered protective of human health. The comparison was intended to determine if conditions at the CUAs would pose any potential health risks to recreational users. CUAs identified as posing a potential risk to human health would be further evaluated in a more comprehensive risk assessment. In contrast, sites considered to pose negligible risk were excluded from further consideration. The sites evaluated in this screening assessment consist primarily of beaches and selected upland picnic areas.

Based on an assessment of site characteristics, sampling of soil, sediment, surface water, and drinking water was conducted at several CUAs. Drinking water samples collected from the tap were evaluated at only two locations. Soil was defined as material above the flood plain (high water mark) of the Lake. Samples were collected in picnic- or play-areas at a distance from the Lake sufficient to avoid inclusion of beach material. Soil in areas where only surficial play is expected was collected from 0 to 1 inch deep. In grassy areas, sod was removed and soil collected both from the root zone and down to the 1-inch level. Sediment was defined as material at the shoreline, below the high water mark. Sediment samples collected in July of 1998 included beach sand, gravel, or other material present above and below the waterline. Dry sediment along beaches where digging play is expected was sampled to a depth of 12 inches. Sediment in wading portions of the beach (waterline to a depth of 3 feet) was sampled from 0 to 6 inches. Surface water samples were collected from 0 to 3 feet below water level after sediments were stirred for two minutes by a field sampler; thus, the surface water samples included suspended solids.

Because children are considered the most sensitive population group, RBCs developed to ensure protection of children are assumed to be protective of adults. RBCs protective of children playing with beach sand and water were developed for recreational exposures. The RBC for soil assumes children will be exposed to beach sand through ingestion and dermal contact and will ingest more soil (i.e., eat more dirt) than they would in their home setting on a per day basis. The RBC for

water assumes children will play in the near-shore area and be exposed to site chemicals through incidental ingestion of disturbed (or stirred-up) sediments in water and through skin absorption of chemicals. Children are assumed to play in soil/sediment and water two days per week (all day, 10+ hours) for four months of the year. A RBC was developed for each of the seven chemicals of concern identified in the 21-square-mile area commonly referred to as the Bunker Hill Superfund site. RBCs were compared to an estimate of the average chemical concentrations in soil, sediment, and surface water at each site. Drinking water concentrations were compared to federal Maximum Contaminant Levels (MCLs) established for drinking water.

Lead RBC values were calculated using the Integrated Exposure Uptake Biokinetic (IEUBK) Model for lead according to U.S. EPA guidance. The Model has built-in assumptions regarding lead exposure, uptake, and its behavior in the body to estimate blood lead concentrations in a child. RBCs were calculated using a target risk goal of not more than a 5 percent risk that a child would have a blood lead level exceeding 10 µg/dL. An initial soil RBC of 1,400 mg/kg was estimated as protective at beaches if soil at the homes contained no greater than 200 mg/kg of lead. If lead concentrations in soil or sediment exceeded 1,400 mg/kg, the CUA was retained for further evaluation. After screening soil, a second step involved combining sediment and surface water exposures. If combined exposures resulted in a predicted risk greater than 5 percent of a child exceeding the blood lead goal, the site was retained for further evaluation.

For chemicals other than lead, RBCs were calculated using standard U.S. EPA risk equations and solving for a concentration. Target risk goals were established at 1×10^{-5} for carcinogens (excess cancer risk of 1 in 100,000) and a hazard quotient of 0.1 for noncarcinogens (one-tenth of the U.S. EPA Reference Dose). Arsenic is the only carcinogen evaluated in this assessment. The following table presents the RBC and MCL values for chemicals other than lead:

Chemical	**Soil and Sediment RBC** (mg/kg)	**Surface Water RBC** (µg/L)	**Drinking Water MCLs** (µg/L)
Antimony	23	200	6
Arsenic	23[a]	140	50
Cadmium	40	230	5
Copper	2,110	17,000	1,300
Mercury	17	140	2
Zinc	17,100	140,000	5,000

[a]Arsenic has both carcinogenic and noncarcinogenic potential. The RBC for arsenic was selected based on noncarcinogenic potential in children because this RBC was lower than the RBC based on the cancer endpoint. Furthermore, because arsenic's soil RBC is below an estimate of its natural background concentration of 35 mg/kg for the area, site soil and sediments were screened against the background level rather than the RBC.

Of the seven chemicals of concern, only lead and arsenic exceeded RBC values at Harrison Beach (north) and at Blackwell Island, respectively. Combined sediment and water exposures for lead slightly exceeded the predicted risk goal at Harrison Beach on the north side closest to the mouth of the Coeur d'Alene River (CUA Number 18). Of the 23 remaining sites, 22 had concentrations of lead in soil and sediments at or below the Office of Solid Waste and Emergency Response (OSWER) screening level of 400 ppm (U.S. EPA 1994d). The 400 ppm level of concern is used to screen residential yards, where exposure is different from recreational sites. Corbin Park had a sediment lead concentration of 412 ppm, which slightly exceeded the EPA residential screening level, but was substantially below the RBC for combined sediment and water exposures at CUAs. Arsenic concentrations in sediment exceeded arsenic's background level of 35 mg/kg only at Blackwell Island near the mouth of the Spokane River (CUA Number 21). These two areas are retained for further evaluation in the baseline risk assessment. The other 22 sites evaluated in this report will not be considered further in Coeur d'Alene River Basin risk assessments.

Analytical results for drinking water samples collected from two sites, Harrison Beach campground and Loffs Bay, did not exceed MCLs. The total lead concentration at Harrison Beach was 15.5 μg/L, which is approximately equal to the tap water action level for lead of 15 μg/L. Lead in drinking water at the campground will be evaluated further as Harrison Beach (north) is assessed in the Baseline Human Health Risk Assessment.

CONTENTS

Section — Page

EXECUTIVE SUMMARY iii

ABBREVIATIONS AND ACRONYMS xiii

1.0 INTRODUCTION 1-1
- 1.1 PURPOSE 1-1
- 1.2 BACKGROUND 1-2
- 1.3 SITE DESCRIPTION 1-2
- 1.4 CONCEPTUAL SITE MODEL 1-6
  - 1.4.1 Beach Recreation 1-6
  - 1.4.2 Upland Recreation—General Recreation 1-7
  - 1.4.3 Other Considerations 1-8
- 1.5 METHODOLOGY 1-8

2.0 DATA EVALUATION 2-1
- 2.1 SAMPLING INVESTIGATIONS 2-2
  - 2.1.1 Soil and Sediment Sampling 2-2
  - 2.1.2 Surface Water Sampling 2-3
  - 2.1.3 Sampling Drinking Water From Local Wells 2-4
  - 2.1.4 Statistical Analysis and Results 2-4
- 2.2 ESTIMATED SOIL BACKGROUND CONCENTRATIONS USED FOR SCREENING CUA SITES 2-5
  - 2.2.1 Introduction 2-5
  - 2.2.2 Applicability of Data for Evaluating Background Concentrations 2-7
  - 2.2.3 Estimated Background Concentration for Screening of CUA Sites 2-7

3.0 ESTIMATION OF LEAD RBCs AT CUAs 3-1
- 3.1 OBJECTIVES 3-1
- 3.2 GENERAL APPROACH 3-1
- 3.3 MODELING APPROACH 3-2
- 3.4 IMPORTANT ASSUMPTIONS AND UNCERTAINTIES ASSOCIATED WITH THE MODELING APPROACH 3-3
- 3.5 EXPOSURE ASSUMPTIONS 3-4
  - 3.5.1 Exposure Frequency 3-4
  - 3.5.2 Soil Ingestion Rate (IR_S) 3-5
  - 3.5.3 Soil Lead Ingestion at the CUA 3-6
  - 3.5.4 Surface-Water Ingestion While Swimming or Wading 3-7

3.6 LEAD UPTAKE FROM THE DERMAL ROUTE 3-7
3.6.1 Other Exposure Variables 3-8
3.7 RBCs FOR UPLAND CUAs 3-8
3.8 RBCs FOR CUA SHORELINES AND BEACHES 3-8
3.9 COMPUTATIONS 3-9

4.0 SCREENING OF COMMON USE AREAS FOR LEAD 4-1
4.1 SCREENING METHODOLOGY 4-1
4.2 RESULTS OF RISK-BASED SCREENING 4-3
4.2.1 Upland Soil Ingestion 4-3
4.2.2 Beach Sand/Sediment Ingestion 4-3
4.2.3 Combined Beach Sediment Ingestion and Incidental Ingestion of Water and Suspended Sediment While Swimming 4-4
4.2.4 Conclusions from Risk-based Screening 4-5

5.0 DEVELOPMENT OF RBCs FOR CHEMICALS OTHER THAN LEAD 5-1
5.1 EXPOSURE ASSESSMENT 5-1
5.1.1 Characterization of Exposed Populations 5-2
5.1.2 Exposure Scenarios 5-2
5.1.3 Quantitative Assessment of Exposure 5-5
5.2 TOXICITY CRITERIA 5-12
5.2.1 Oral Toxicity Criteria 5-12
5.2.2 Dermal Toxicity Criteria 5-13
5.3 CALCULATION OF RBCs 5-13
5.3.1 Calculation Methods 5-14

6.0 SCREENING OF COMMON USE AREAS FORCHEMICALS OTHER THAN LEAD 6-1
6.1 SCREENING METHODOLOGY 6-1
6.1.1 Estimate of the Average Concentration 6-2
6.2 RESULTS OF RISK-BASED SCREENING 6-2
6.2.1 Upland Soils 6-2
6.2.2 Sediments 6-3
6.2.3 Lake and River Water 6-3
6.2.4 Drinking Water 6-4

7.0 UNCERTAINTIES IN THE EXPEDITED SCREENING ASSESSMENT 7-1
7.1 UNCERTAINTIES IN THE DEVELOPMENT OF RBCs 7-2
7.1.1 Site-Specific Uncertainties in the Development of RBCs 7-2
7.1.2 General Uncertainties in the Development of RBCs 7-4
7.2 UNCERTAINTIES IN THE DEVELOPMENT OF MEDIA CONCENTRATIONS 7-5
7.3 UNCERTAINTIES SPECIFIC TO LEAD 7-7

7.3.1 Exposure Scenarios 7-7
7.3.2 Concentration Term in the IEUBK Model 7-8
7.4 SUMMARY 7-9

8.0 SUMMARY AND CONCLUSIONS 8-1

9.0 REFERENCES 9-1

CONTENTS (Continued)

APPENDIXES Page

A Representative Photographs of Common Use Areas
B Sample Location Maps
C Data Summaries
D Statistical Data Summary for Seven Metals of Concern in Sediments of the Lower Coeur d'Alene River Basin
E Summary Intake Factors and RBC Calculations: Chemicals Other Than Lead
F Chemical Toxicity Profile
G Distribution Check and UCL_{95} Calculations for Arsenic and Lead
H Arsenic Background Concentrations, Faxed Memorandum from SRC dated November 18, 1998 and Lead Background Concentrations in the CDARB, report prepared by SRC dated February 1999

FIGURES

1-1 Coeur d'Alene Lake Beach Area Sampling Sites 1-11
1-2 Conceptual Site Model—Coeur d'Alene Lake and Spokane River Beaches..... 1-13
3-1 Lead RBCs for Soil Ingestion Scenario (One Year Exposure Duration) 3-10
3-2 Lead RBCs for Soil Ingestion Scenario (Six Year Exposure Duration)..... 3-11
3-3 Relationship Between Water/Sediment RBCs for Swimming Scenario and Soil Lead Concentration at the Beach (PbS_{cua}) for Four Residential Soil Lead Levels (PbS_{res}=50, 100, 200, 300 ppm) 3-12
4-1 Decision Tree for Risk-Based Screening of Lower Basin Shoreline Parks and Beaches . 4-7

TABLES

1-1_ CUAs in the Coeur d'Alene River Basin 1-14
2-1 Summary of the Number of Samples Collected at the CUA Sites 2-13
2-2 Summary of Analytical Results for Soils and Sediments 2-14
2-3 Summary of Analytical Results for Surface Water 2-15
2-4 USGS 90th Percentile Background Concentrations 2-16
2-5 Estimated 95th Percentile Background Concentrations for Use in Screening CUAs 2-17
3-1 PbB Central Tendency Estimate (µg/dL) 3-13
3-2 P_{10} (%) Corresponding to PbB Central Tendency Estimate 3-13
3-3 Age-Specific PbB Central Tendency Estimate (µg/dL) 3-13
3-4 Exposure Frequency to CUAs by Age 3-15
3-5 Soil RBCs Corresponding to Various CUA Exposure Frequencies 3-15
3-6 CDF for Soil Ingestion in Children Ages 1–4 3-16
3-7 Medium Category[a] Age-Specific Values for Soil Ingestion in Children[b] 3-17
3-8 Lead Uptake Resulting From Dermal Contact With Water While Swimming,Incidental Ingestion of Water While Swimming, and UptakeFrom Ingestion of CUA and Residential Soil 3-18
3-9 Comparison of Estimated Lead Uptake Resulting From Dermal Contactand Ingestion of CUA Soil and Residential Soil (PbS_{res} = 200 mg/kg)[a] 3-19
4-1 Risk-Based Concentrations (RBCs) for Upland Soil at Beaches 4-8
4-2 Risk-Based Concentrations (RBCs) for Shoreline Sediment at Beaches 4-9
4-3 Risk-Based Concentrations (RBCs) for Water and Suspended Sediment at Beaches (Mean Lead Concentrations) 4-10
4-4 Risk-Based Concentrations (RBCs, µg/L) for Water and Suspended Sediment at Beaches (UCL_{95} Lead Concentrations) 4-11
5-1 Exposure Factors 5-16
5-2 Absorption of Chemicals From Soil 5-16
5-3 Permeability Coefficients From Water 5-17
5-4 Oral Toxicity Criteria 5-17
5-5 Risk-Based Concentrations 5-18
6-1 Screening Results for Arsenic (All Sites) and Antimony (Site 18 Only) 6-5

ABBREVIATIONS AND ACRONYMS

AF	adherence factor
ATV	all terrain vehicle
BLM	U.S. Bureau of Land Management
CDARB	Coeur d'Alene River Basin
CDF	cumulative distribution function
CFD	cumulative frequency distribution
COC	chemical of concern
CTE	central tendency estimate
CUA	common use area
dL	deciliter
EF	exposure frequency
EPA	U.S. Environmental Protection Agency
FSPA	Field Sampling Plan Addendum
HHRA	human health risk assessment
HQ	hazard quotient
IEUBK	Integrated Exposure Biokinetic Model for Lead in Children
IR	ingestion rate
IRIS	Integrated Risk Information System
kg	kilogram
LOAEL	lowest-observed-adverse-effect level
MCL	maximum contaminant level
mg	milligram
µg	microgram
NOAEL	no-observed-adverse-effect level
OSWER	Office of Solid Waste and Emergency Response
PbB	lead concentration in blood
PbS	lead concentration in soil
PbWS	lead concentration in water/sediment
P_{10}	probability of exceeding a blood lead concentration of 10 µg/dL
ppb	parts per billion
ppm	parts per million
RBC	risk-based concentration
RfD	reference dose
RME	reasonable maximum exposure
SA	surface area
SF	slope factor
SIF	summary intake factor

ABBREVIATIONS AND ACRONYMS (Continued)

SVNRTS	Silver Valley Natural Resource Trustees
TRW	Technical Review Group
URSG	URS Greiner, Inc.
UCL_{95}	95 percent Upper Confidence Limit

2.0 INTRODUCTION

2.1 PURPOSE

This report provides a screening evaluation of metal concentrations in beach sediment, soil and water at selected sites (Common Use Areas, or CUAs) located on public and private lands along the shores of Coeur d'Alene Lake (Lake) and the Spokane River. Past mining activities in the Coeur d'Alene River Basin (CDARB) are known to have released metals into the watershed. Consequently, these metals have been transported to the Lake. Concern developed regarding the potential for exposure to metals at recreational sites, since the general public visit the CUAs for wading, swimming, picnicking, and other recreational activities. The purpose of this screening evaluation is to evaluate the CUAs to determine which CUAs, if any, could be eliminated from further regulatory concern.

Data were gathered at CUAs throughout the CDARB during the summer field season of 1998. However, only CUAs on the shores of the Lake and the Spokane River are evaluated in this report. CUAs not evaluated here will be evaluated in a baseline human health risk assessment (HHRA) in 1999. Sites deferred to the baseline HHRA include all sites along the Coeur d'Alene River and its tributaries upstream from the confluence of the river and the lake at Harrison.

To conduct the screening, concentrations of metals in sediment, soil, and water at the selected CUAs are compared to risk-based screening concentrations (RBCs) protective of human health. Sediment refers to materials at the shoreline, including beach sand, gravel, or whatever materials are present above and below the waterline. Soil refers to materials away from the shoreline, such as soil in picnic- or play-areas. Based on this comparison, sites will either be:

- Excluded from further consideration because they are unlikely to pose a threat to human health; or

- Carried forward for additional, more detailed evaluation in the baseline HHRA

The screening is done by comparing contaminant concentrations in specific media, such as surface soil or beach sand, to RBCs developed for the contaminant in that medium. If media concentrations are below the RBC, the contaminant is unlikely to present a health risk in the given medium at the given site. If measured concentrations exceed RBCs, exposure to contaminants at the site requires additional, more detailed analysis.

2.2 BACKGROUND

Environmental problems in the CDARB have accumulated from a number of different sources, including more than 100 years of mining, milling, and ore processing in the Silver Valley. The residual tailings, which are a waste product of ore processing, are suspected to be major contributors of metals contamination. Waste rock piles produced by mining operations could also contribute metal contaminants. Surface-water runoff from tailings piles into streams and rivers, actual use of tailings in construction activities and other activities have distributed contaminants into areas where people can be exposed to them. In addition, air-dispersed metals generated by the mining and smelter operations contributed to surface soil contamination throughout the CDARB.

To assess potential contamination at CUAs in the CDARB, the Panhandle Health District (PHD) investigated lead at eight public beaches along the Lake and the Spokane River in 1997. In the PHD study, samples were taken from beach sediment and analyzed only for lead. Water was not sampled. Results of the investigation indicated that most of the beaches had relatively low lead levels (ranging from <10 to 90 parts per million [ppm]). However, Harrison Beach, located close to the confluence of the Coeur d'Alene River and the Lake, had reported concentrations of lead ranging from 155 to 344 ppm.

2.3 SITE DESCRIPTION

The CDARB is a major recreational area for people from in and out of the state of Idaho. This evaluation covers 24 developed recreational areas from Corbin Park, west of Post Falls, Idaho, on the Spokane River, to Fuller Landing, south of Harrison on the Lake (Figure 1-1). The sites were selected in a two-part process. First, a preliminary list of CUAs was developed based on input from M. Calabretta, a Silver Valley Natural Resource Trustees representative, and E. Liverman, the U.S. EPA Coeur d'Alene field representative. Subsequently, during the week of June 8, 1998, field teams visited most of the sites on the preliminary list. E. Liverman accompanied the field team to clarify site locations. Discussion of the preliminary list and the findings of the field visits focused the list of potential sites to those provided in Table 1-1. This table lists the CUAs included in this screening evaluation. Additional information regarding site selection is in the *Field Sampling Plan Addendum 05* (FSPA 05) report prepared by URS Greiner, Inc. (URSG 1998a).

Table 1-1 groups the sites into two geographical locations: (1) Spokane River (CUA Number 30) and (2) Coeur d'Alene Lake. In general, the Spokane River and the Lake sites are all beaches where people play and swim at the water's edge. Sediment samples from shoreline areas were taken at all sites except three: Harrison Beach (Site 1, West, CUA Number 17), Fuller Landing, and Rockford Bay. The latter two areas were both restricted to a boat ramp and upland play areas. Shoreline samples (referred to as "sediment" in this report) were taken above and below the waterline, in the "beach zone" and "wading zone," respectively. In some cases, rocky or cobbled

beaches precluded sampling above the water line; therefore, sufficient sample materials could not be obtained.

Some of the CUAs include upland areas away from the shore where people may picnic or use play areas and fields for recreational activities. Upland areas were sampled if it seemed possible that high water events could have inundated these areas, potentially depositing contaminated sediments. Drinking water was not available at most of the sites; however, several of the developed city beaches and parks had drinking water supplied from the city systems. Municipally supplied water routinely is tested by the State of Idaho and meets drinking water standards; therefore, municipal sources were not sampled. Two sites, Harrison Beach and Loff's Bay, had water supplies originating from groundwater sources. If the drinking water source was not public or not known, drinking water was sampled. A brief overview of each site is provided below, and representative photographs are provided in Appendix A.

North Idaho College Beach, Along the Spokane River (CUA Number 1). This site is associated with North Idaho College, and is a developed beach where multiple recreational activities occur, including swimming and wading. The shoreline consists of gravel. There is a park area with picnic tables. Soil samples were taken up from the beach based on an assumption that high water may inundate this area. Drinking water is supplied from the municipal system.

Post Falls City Beach/River Park (CUA Number 3). This is a developed park that includes a volleyball court, picnic areas and playgrounds, as well as the wading and swimming beach. The beach consists of sand. Upland soil samples were taken because of the relatively flat slope from the beach to the picnic and play areas. Inundation of the upland areas during high water events was assumed. Drinking water is supplied from the municipal system.

Green Ferry Bay County Park (CUA Number 5). This is a developed park that includes a horseshoe pit, volleyball court, and picnic area, as well as the wading and swimming beach. The beach is primarily gravel. Soil samples were taken at upland areas based on an assumption that high water may inundate this area. Drinking water is not available.

Black Bay (CUA Number 6). This is a small recreation area that includes a small beach and wading area. The site is relatively undeveloped, limited to the small beach and some trails, and is used as a general gathering place. Soil samples were taken up from the beach based on an assumption that high water may inundate this area. Drinking water is not available.

BLM Pump Station (CUA Number 7). This site is relatively small and less developed than the city beaches. It includes a beach with a picnic table, a park area, and some trails. Soil samples were taken up from the beach based on an assumption that high water may inundate this area. Drinking water is not available.

Corbin Park (CUA Number 8). Corbin Park has multiple uses, including the beach, picnic areas, playfields, and a boat ramp. The playfields include volleyball courts and a baseball diamond. The play areas were assumed to be above the high water levels, so samples were taken only from the beach. The beach itself is fairly rocky, which likely limits the amount of beach play that might occur on a sandier beach. Drinking water is supplied from the municipal system.

Blackwell Island (CUA Number 21). The Blackwell Island site is a very small, undeveloped stretch of gravel and sand where people can access the Spokane River. There are also trails to an undeveloped upland area where people may gather. Drinking water is not available. Samples were taken from the upland area and the beach.

North Idaho College Beach, Along Coeur d'Alene Lake (CUA Number 2). This site is associated with North Idaho College, and is a developed beach where multiple recreational activities occur, including swimming, wading, and sailboat launching from the beach. There is a park area with picnic tables. However, high water is not likely to inundate this area, so upland soil samples were not taken. Drinking water is supplied from the municipal system.

Coeur d'Alene Beach at City Park (CUA Number 9). This is a highly developed beach and park. Facilities include the beach itself, a park, picnic areas, playfields, and playgrounds. Drinking water is supplied from the municipal system. A retaining wall separates the beach from the rest of the park, so high water is not likely to inundate the upland areas. Based on this, samples were taken only from the beach.

Tubbs Hill, Site 1 (CUA Number 10). This site consists of a long, narrow, sandy beach on the west side of Tubbs Point. The beach is approximately 150-feet long and from 6-feet to 30-feet wide. The site is used primarily for swimming, wading, and general gathering. There is no drinking water available. Samples were taken only from the beach.

Tubbs Hill, Site 2 (CUA Number 11). This site consists of a sandy beach on the east side of Tubbs Point. The site is used primarily for swimming, wading, and general gathering. There is no drinking water available. Samples were taken only from the beach.

Tubbs Hill, Site 3 (CUA Number 12). This site consists of a sandy beach east of the other Tubbs Hill beaches. The site is used primarily for swimming, wading, and general gathering. There is no drinking water available. Samples were taken only from the beach.
Higgin's Point, Sites 1 and 2 (CUA Numbers 15 and 16). Samples were taken only from the beach area. Photographs are not available for these two sites.

Harrison Beach, West (CUA Number 17). This site is on the west side of the town of Harrison. The beach site is very rocky, such that it was not possible to obtain sediment samples. The upland areas have been developed for use as an RV campground, boat launch, and fishing pier. Picnic

areas are also available. A drinking water source is available at this site, so a sample was collected. Surface soils were sampled at this site. People using this site are likely to use Harrison Beach Sites for swimming.

Harrison Beach, North (CUA Number 18). This site is north of Harrison Beach West, and consists of a sandy beach developed for wading and swimming. Sediment was sampled at this site.

Cougar Bay (CUA Number 19). Cougar Bay is a small, less developed site used for wading, swimming, and fishing. Drinking water is not available. Samples were taken from the upland areas based on the assumption that high water could inundate the area.

Bell Bay (CUA Number 23). Bell Bay is primarily a set of docks for boating and fishing. It also includes camp sites and picnic areas. Wading and swimming are expected, but there is no beach area available for typical beach play. The shoreline consists primarily of cobbles. Drinking water is not available down at the beach. Upland areas were sampled.

Mica Bay (CUA Number 24). Mica Bay is a developed recreational area that includes a campground, boat docks, playgrounds, picnic areas, and playfields. Drinking water is not available. Upland areas were sampled.

Rockford Bay (CUA Number 25). Rockford Bay is one of the sites evaluated in this assessment that does not include a beach. The site is small, and limited to a boat ramp and picnic area. Only upland areas were sampled.

Loff's Bay (CUA Number 26). Loff's Bay includes both upland and beach uses, so both soil and beach sediment samples were collected. In addition, Loff's Bay has a drinking water source consisting of a simple pipe running from a hillside. People can collect water as it runs from the open end of the pipe. However, the water source is posted with a warning stating that the water likely contains unacceptable levels of fecal coliform bacteria, and that water should be boiled before use in drinking and cooking. One sample was taken from this source.

Windy Bay (CUA Number 27). Windy Bay has a gravelly shore, though it is used as a place to enter the water for swimming. The site also has a boat dock, a campground, and picnic areas. Upland areas were sampled.

Spokane Point (CUA Number 29). Spokane Point was sampled as a potential wading and swimming area, though there is some doubt as to whether there is much wading activity at the site. The pitch of the shoreline below the waterline is moderately steep. A boat dock is present. No upland recreational uses were noted, so upland samples were not gathered. Drinking water is not available.

Fuller Landing (CUA Number 30). Fuller Landing is a boat launch area, but not a significant beach area. Wading is expected in the process of launching boats, but the shoreline is fairly steep. The shoreline is cobbled. Only upland areas were sampled.

2.4 CONCEPTUAL SITE MODEL

Exposure at the CUAs on the Lake and the Spokane River is incurred through recreational activities. The following are the two types of recreational activity that are considered in this assessment (depends on the characteristics of the site):

- Beach recreation
- Upland recreation—general recreation

Each activity type is discussed below. Pathways are presented graphically in the conceptual site model (CSM) for the Lake and the Spokane River (Figure 1-2). RBCs were only developed for beach recreation because that is the most intensive exposure to sediments and water. RBCs protective of beach play will also be protective of activities in the upland areas.

2.4.1 Beach Recreation

Typical recreational uses in the beach areas are:

- Dry beach play—playing in the sand, building sand castles
- Shallow-water play—wading, splashing, playing catch in shallow water
- Swimming

These recreational activities produce intensive contact with sediments, especially when individuals are moving in and out of the water and in contact with wet surfaces. Of particular interest is a child playing in the sand, where wet materials are likely to adhere to the skin surface, and a large proportion of skin surface is exposed. Under such conditions, adhered materials are available for hand-to-mouth transport, and as a source for contaminant transport across the dermal barrier.

Playing in shallow water is also a concern, because wading and splashing are likely to resuspend sediments. The suspended sediments provide a source for incidental ingestion of contaminants as people play in the water. For this reason, surface-water samples were taken in shallow water after the water and sediments were agitated by the sampling crew to simulate beach play.

Swimming beyond the shallow-water zones into clear surface water is a possible exposure route because there may be dissolved metals in the water column. Previously measured dissolved-metals concentrations in offshore waters of the Lake are fairly low, making this pathway less significant for the lake and its major drainage, the Spokane River (Woods and Beckwich 1997). On this basis, only exposures to shallow water areas were evaluated in this assessment.

2.4.2 Upland Recreation—General Recreation

Upland recreation is distinguished from the beach recreation activities specified above, and includes all other activities that do not pertain to shoreline play. At the CUAs on the Lake and the Spokane River, the following uses occur:

- Playgrounds and ball fields (e.g., areas used for ball games and other non-digging types of play)
- Picnicking
- Camping
- Drinking public supplied water from local wells (two sites)
- Trail use

Pathways of concern in the upland areas include exposure to surface soils, followed by incidental ingestion and dermal absorption. Inhalation of particulates entrained in the air is a possibility at sites where the ground surface is bare and dusty, but this represented a minority of sites and was not included in the development of RBCs for screening.

Playgrounds that have sandboxes or other digging types of play areas were found at the Post Falls City Beach and the Coeur d'Alene Beach at City Park. However, these playground areas were determined to be above the high water level, and so these areas were not sampled.

2.4.3 Other Considerations

Incidental exposure to soil or water could occur during fishing or gathering of other food items from the Lake or the Spokane River. However, a recent Agency for Toxic Substances and Disease Registry (ATSDR) evaluation (1998) determined that consumption of fish from the Lake presents insignificant risks to those who do not experience excessive lead exposures elsewhere, and surface water concentrations of metals in the lake are relatively low. Therefore, exposure during fishing and other food gathering activities was not evaluated.

It is also possible that park maintenance workers could be exposed to contaminants in soil, sediment and water during the course of their work activities. However, the screening concentrations developed in this assessment were developed to protect the most sensitive population, children, under conditions of intensive exposure during beach play. These screening concentrations will be protective of adult maintenance workers.

2.5 METHODOLOGY

The focus of this expedited risk assessment is the development of screening RBCs in soil and water that will protect all visitors to CUAs around the Lake and the Spokane River. Recreational exposure was evaluated based on children ingesting soil and getting soil on their skin (dermal contact). In addition, the beach areas were also evaluated for child exposures while swimming. Children were selected as the most sensitive population.

This report was prepared in accordance with EPA's current risk assessment guidelines (U.S. EPA 1989a, 1991a, 1991b, 1993, 1994, and 1997a). Exposure assumptions are based on federal and EPA Region 10 recommended exposure factors (U.S. EPA 1991c); however, in the absence of appropriate regulatory guidance (e.g., site-specific conditions), the evaluation follows the best available science and professional judgment.

The accuracy of this report depends in part on the quality and representativeness of the available sampling, exposure, and toxicological data. Where information is incomplete, conservative assumptions were made so that public health risks were not underestimated. Section 7 presents a discussion of uncertainties in the risk assessment resulting from data limitations.

The risk assessment includes the descriptions and evaluations of the sampling data presented in Section 2. Section 3 describes the development of RBCs for lead (according to EPA policy, lead is evaluated differently from other metals). Section 4 describes the site screening methodology for lead, and the screening results. Section 5 describes the development of screening RBCs for chemicals other than lead. Section 6 compares the sampling data with the site-specific RBCs for chemicals other than lead. Section 8 presents a summary and the conclusions of the report.

Figure 1-1 Coeur d'Alene Lake Beach Area Sampling Sites

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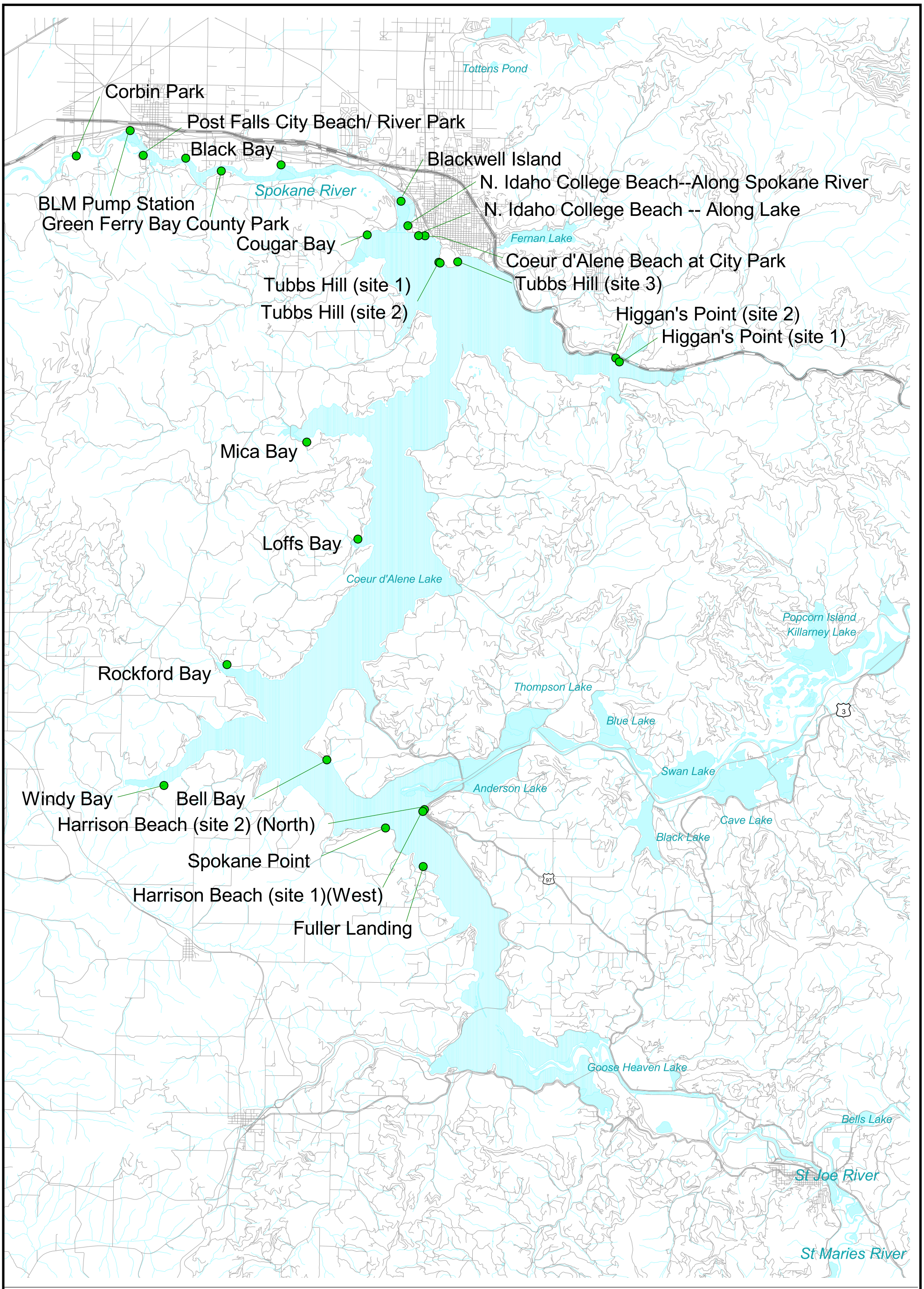

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VIEW:LAKE
LAY:Common Use Ares
Plot Date: 11/11/98

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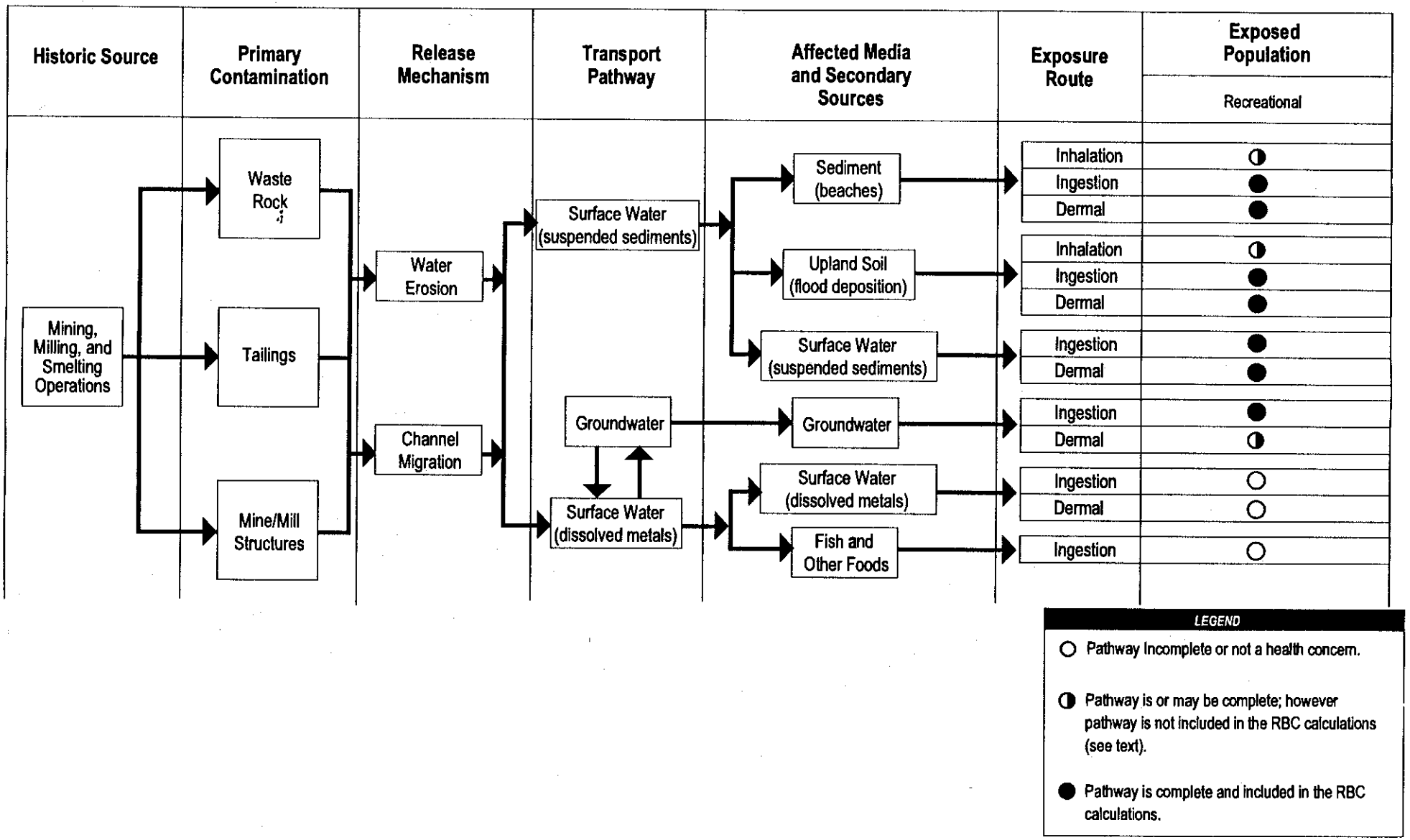
Coeur d'Alene Basin RI/FS

Figure 1-1
Coeur d'Alene Lake
Sediment & Soil Sampling Sites 1998
Common Use Areas
-DRAFT FINAL-



EPA REGION 10

Figure 1-2
Conceptual Site Model
Coeur d'Alene Lake and Spokane River Beaches

54-50-0C2Q
Draft Final Coeur d'Alene RI/FS
CUA RISK ASSESSMENT
CDARB

53F4002700070170 0-1a-101199

Table 1-1
CUAs in the Coeur d'Alene River Basin

Site Location	CUA ID	Site Name	Use Category	Use Frequency Days/Week	Use Duration Months/Year
Spokane River (7 sites)	1	N. Idaho College Beach (Spokane River)	Beach & Upland Area	2	4
	3	Post Falls City Beach/River Park	Beach & Upland Area	2	4
	5	Green Ferry Bay County Park	Beach & Upland Area	2	4
	6	Black Bay	Beach & Upland Area	2	4
	7	BLM Pump Station	Beach & Upland Area	2	4
	8	Corbin Park	Beach Only	2	4
	21	Blackwell Island	Beach & Upland Area	2	4
Coeur d'Alene Lake (17 Sites)	2	N. Idaho College Beach (CdA Lake)	Beach Only	2	4
	9	Coeur d'Alene Beach at City Park	Beach & Upland Area	2	4
	10	Tubbs Hill (Site 1)	Beach Only	2	4
	11	Tubbs Hill (Site 2)	Beach Only	2	4
	12	Tubbs Hill (Site 3)	Beach Only	2	4
	15	Higgan's Point (Site 1)	Beach Only	2	4
	16	Higgan's Point (Site 2)	Beach Only	2	4
	17	Harrison Beach (Site 1, West)	Upland Area Only	2	4
	18	Harrison Beach (Site 2, North)	Beach Only	2	4
	19	Cougar Bay	Beach & Upland Area	2	4
	23	Bell Bay	Beach & Upland Area	2	4
	24	Mica Bay	Beach & Upland Area	2	4
	25	Rockford Bay	Upland Area Only	2	4
	26	Loffs Bay	Beach & Upland Area	2	4
	27	Windy Bay	Beach & Upland Area	2	4
	29	Spokane Point (on reservation)	Beach Only	2	4
	30	Fuller Landing	Upland Area Only	2	4

Six of the original 30 CUAs were eliminated from the sampling effort for various reasons (Sites 4, 13, 14, 20, 22, and 28). Of these six sites, five were simply boat ramps where activities other then launching boats were not expected. These sites did not include beaches or improvements that would encourage recreational activities and produce exposure. The sixth site was a beach fronted by several private residences (Sanders Beach). EPA sought permission from the homeowners to sample their beaches. Several homeowners denied permission to access their property; therefore, EPA decided not to sample. Sanders Beach was indirectly represented by other beaches sampled at the north end of Coeur d'Alene Lake, particularly Tubbs Hill (Site 3), which is very near Sanders Beach.

Notes:
BLM - U.S. Bureau of Land Management
CUA - Common Use Areas
CdA - Coeur d'Alene

3.0 DATA EVALUATION

This section provides a brief summary of the sampling and analysis completed to support this screening assessment. Samples were collected from soil, sediments, surface water, and at two sites, drinking water. Maps showing the sample locations at each common use area (CUA) are presented in Appendix B. Sections below describe the numbers and types of samples collected at each CUA and present analytical results. Also described are the methods used to estimate background concentrations of metals in soil and surface water, and to select chemicals of concern (COCs).

Data were gathered for this screening level analysis as described in the *Field Sampling Plan Addendum 05* (FSPA 05) report prepared by URSG (1998a). The overall objectives of FSPA 05 included the following:

- Provide adequate data to support conclusions that areas presently assumed to be clean are in fact clean and may be eliminated from further investigation.
- Provide adequate data to support an assessment of risks to human health in each investigation location.
- Provide data to support decision-making regarding the need for and nature of potential remedial measures at investigation locations.

To achieve these objectives, the following activities were completed:

- Collect data on the potentially affected media found in CUAs in selected locations throughout the Coeur d'Alene River Basin (CDARB).
- Collect data on the potentially affected media found in recreational beach areas in selected locations along Coeur d'Alene Lake (Lake) and the Spokane River.
- Observe common recreational activities at study locations to provide input to assumptions used in exposure models
- Supplement existing data for the study locations.

The constituents of concern are:

- Antimony

- Arsenic
- Cadmium
- Copper
- Lead
- Mercury
- Zinc

These metals were selected based on those constituents previously identified as contaminants of concern in the 21-square-mile area commonly referred to as the Bunker Hill Superfund site.

3.1 SAMPLING INVESTIGATIONS

Based on known public uses of the CDARB and the possibility of human health risks from exposure to metal contaminants, samples were collected from sediment, soil, and surface water at the CUAs. In addition, drinking water sources were sampled at two locations because the water source was not a public water supply. Public-supplied water at the developed beaches is delivered from municipal water systems that are tested and known to be free from contamination. Table 2-1 is a summary of the media sampled and the number of samples collected at each CUA.

3.1.1 Soil and Sediment Sampling

Table 1-1 lists the CUAs that were sampled in FSPA 05. The objective of the sampling was to produce sufficient data for screening against RBCs, and, if necessary, to estimate an upper confidence limit on the mean concentration (see Section 3.3 of FSPA 05).

Contaminant concentrations in beach sediment along the Lake and the Spokane River were expected to be relatively uniform within the span of any single beach due to the nature of sediment deposition during flooding events. Given a homogenous distribution, the statistical variability in contaminant concentrations in beach sediment along the Lake and the Spokane River was expected to be relatively low. Based on this expectation, the "Max of N" method (Conover, 1980) was used to calculate the number of samples to be collected, as described in Section 3.5 and Attachment E in FSPA 05.

The "Max of N" method is a nonparametric technique used to calculate the number of samples needed to estimate a prespecified tolerance interval of the sampled population with a prespecified level of confidence. In FSPA 05, the technique was used to calculate the number of samples needed to estimate the median concentration of the sampled population with 95 percent confidence. Based on this method, collecting 5 samples would assure that the maximum detected value of the samples would be greater than the median of the population. In other words, the data set would bracket the median (as opposed to being lower than the median). Therefore, comparing the

maximum detected values to RBCs would assure that the median concentration was less than the RBC. In turn, these data could be used to calculate the mean or upper confidence limit on the mean. Although 5 samples were determined to be sufficient for screening purposes, the number of samples was increased to 7 to increase confidence in the results, particularly due to the expectation that metals at the beaches would not exceed screening concentrations.

Both sediment (i.e., river and lake sediment) and soil (i.e., playground sand, play field soil, and other soil as identified) were collected in common and recreational areas of interest. Sampling was based on an assumption of two types of exposure to sediment or soils: (1) children or others digging in beach sand or playground soil; and (2) children or others contacting the surface material during play. Soil in areas where only surficial play is expected was collected from 0 to 1 inch deep. In grassy areas, sod was removed and soil collected both from the root zone and down to the 1-inch level. Dry sediment along beaches where digging play is expected was sampled to a depth of 12 inches. Sediment in the wading portion of the beach (waterline to a depth of 3 feet) was sampled from 0 to 6 inches. The soil and sediment collection methods used were taken from the *Generic Field Sampling Plan and Generic Quality Assurance Project Plan for the Bunker Hill Facility* prepared by URSG (Field Sampling Plan; 1997), Section 4.2.1 and 4.2.3, respectively. Soil and sediment samples were sieved through an 80 screen mesh sieve following ASTM Method D-422. Sieving was done to produce particles for analysis that represent the size of particles expected to adhere to skin. Table 2-2 provides a summary of analytical results for metals in soils and sediments from samples gathered at the CUAs. Only two sediment samples were collected from Bell Bay (CUA Number 23) and two from Windy Bay (CUA Number 27) because an insufficient amount of fine materials was present at these two sites. The wet sediments consisted of very coarse sand to large cobble, with no materials present in the 80 mesh range. Wave action likely moved fine sediments to deeper depths.

3.1.2 Surface Water Sampling

Incidental ingestion is possible during swimming or wading activities. Therefore, surface water was sampled from selected river and lake areas. Samples were taken at shallow-water beach locations with a low or moderate slope underwater based on the assumption that people are unlikely to attempt to wade on steeply sloped beaches. Prior to surface water collection, field samplers disturbed sediments at a randomly located sample point in an effort to mimic surface water conditions during water play activities. Sampling of disturbed water that contained suspended sediments was used as a protective, worst case scenario and should be protective of ingestion of undisturbed surface water. Randomly placed surface water samples were collected from the mid-water column depth (0 to 3 feet below water level) in the disturbed area. The collection methods were taken from the Field Sampling Plan (URSG 1997) (Section 4.2.2). Table 2-3 provides a summary of the analytical results for samples taken from surface water in which sediments were resuspended by the field crew, and, for two sites, drinking water samples.

3.1.3 Sampling Drinking Water From Local Wells

Two CUAs have public drinking water supplies. Harrison Beach (CUA Number 17) supplies water at the campground. At Windy Bay, a simple pipe carries water, presumably from groundwater, from a hillside. One sample was collected at each of these sources. First draw water samples were collected. That is, water that had been standing in the water pipes for several hours was collected and analyzed. Table 2-3 presents the results of the drinking water sampling.

3.1.4 Statistical Analysis and Results

Modeling requirements necessitated different statistical treatment of lead and non-lead metals. For lead, arithmetic average concentrations were used in the screening, consistent with input requirements for the Integrated Uptake Biokinetic (IEUBK) model (see Sections 3 and 4). It is inappropriate to use other than arithmetic average concentrations in the IEUBK model.

For non-lead metals, maximum concentrations were used for all metals except arsenic (see below). These metals were antimony, cadmium, copper, mercury, and zinc. Comparing maximum concentrations to RBCs represents the most conservative screening scenario, since the sampling plan was designed to assure, with 95 percent confidence, that maximum concentrations would exceed the median of the data set. Therefore, if the maximum concentration for a given metal is less than its RBC, then the median value will be less than the RBC, and risks are not expected to be significant.

For arsenic, the 95 percent upper confidence limit (UCL_{95}) on the mean was used for comparison to the screening concentrations. The UCL_{95} was used because data from the CUA sites were compared to a background arsenic concentration specifically developed for sediments (see Section 2.2) instead of the arsenic RBC, which falls below background. Background concentrations were set at the 95th percentile of the background data set, as directed by EPA, to assure that concentrations below this percentile are regarded as background.

To generate the UCL_{95}, data distributions were first checked to determine if the data were normally or lognormally distributed. If the data were lognormally distributed, then the UCL_{95} on the lognormal distribution was used for screening. If the data were normally distributed, or did not fit either a lognormal or normal distribution, then the UCL_{95} on the normal distribution was used. In most cases, the data were lognormally distributed. The values for arsenic presented in Table 2-2 are lognormal UCL_{95}s unless otherwise noted.

Distribution tests and UCL_{95}s were completed using the MTCA*Stat* add-in (Version 2.1) to Microsoft Excel. MTCA*Stat* is available from the Washington State Department of Ecology. A summary of the results from the MTCA*Stat* application is provided in Appendix G. Appendix B shows sample locations for each site and Appendix C contains detailed data summaries for each

location. Table 2-2 presents the maximum concentrations used in the screening evaluation for the chemicals detected in soil and sediments and Table 2-3 presents the maximum concentrations used in the screening evaluation for the chemicals detected in surface water at each CUA.

3.2 ESTIMATED SOIL BACKGROUND CONCENTRATIONS USED FOR SCREENING CUA SITES

A background concentration for each of the seven metals of concern was developed for the specific task of screening the CUAs located around the Lake and the Spokane River. Use of the background concentrations presented below should be limited to the CUA screening process, and not extended basin-wide. Different background concentrations may be required elsewhere in the CDARB due to differences in geology.

Given the size and complexity of the CDARB, a range of background concentrations rather than a single value is a better representation of the variations in metal concentrations throughout the basin. However, an upper estimate of background was calculated here for the purpose of screening. The intent is not to retain sites for further evaluation if the concentrations at the site are in the range of possible background values.

3.2.1 Introduction

The United States Geological Survey (USGS) conducted a study in the Coeur d'Alene Mining District to evaluate the use of geochemical anomalies in predicting the presence of concealed ore deposits (Gott and Cathrall 1980). To develop analytical data for the study, approximately 8,700 soil and 4,000 rock samples were collected throughout the district and analyzed for a selected suite of elements.

Rock samples collected were screened, and only the minus 100-mesh or finer portion was used for analysis. The soil samples collected were screened, and only the minus 80-mesh portion was used for analysis. The analytical data was then evaluated to identify which elements might be useful as indicators of known and potentially undiscovered ore deposits. The study covered approximately 300 square miles of the Coeur d'Alene Mining District. The area included in the USGS study is the probable source area of most sediments deposited in and around the Lake.

Geochemical maps were gridded to a rectangular coordinate system. Sample locations (rock and soil) within an 800-foot radius of a grid mesh point were transposed to the mesh point and weighted based on distance moved. All data at a mesh point were then averaged, and the average value was used to generate geochemical contour maps.

The aspect of the USGS study that is of interest to the CUA screening process is the reported background concentrations for the seven metals of concern. As used in the study, each background metal concentration represents a threshold above which the metal concentration has been enriched in rock or soil by some process, possibly emplacement of an ore deposit.

Past health risk studies in the CDARB have used the background data to help establish thresholds above which metal concentrations in soil or rock may be elevated as a result of mining activity. As part of the current screening evaluation, the USGS study was reviewed to assess its applicability for development of background concentrations for the seven metals of concern. However, the current CUA screening process focuses on sites that lie outside the USGS study area. Consequently, a background analysis of the seven metals of concern was conducted using available sample data developed by URSG (1998a) in the lower basin sediments and CUA areas throughout the basin. The following sections summarize background information presented in the USGS report and development of background concentrations for use in the CUA Screening process based on URSG's (1998a) sediment sampling.

The objective of the sediment sampling effort conducted by URSG (1997 and 1998a) was to collect data to define the vertical extent of mining waste deposits within the Coeur d'Alene River main stem, Lateral Lakes, and Coeur d'Alene River floodplains. The data was used to estimate the volume of sediments within the lower CDARB that is contaminated with mining waste. The second phase of the field investigation involved estimating the vertical distribution of metals in the sediments down to the pre-mining sedimentary material and collecting sediment core samples within the Coeur d'Alene River main stem, the Lateral Lakes, and the Coeur d'Alene River floodplains.

USGS established four core transect sites along the lower Coeur d'Alene River: Harrison, Swan, Medimont, and Cataldo. Five cores were collected per transect with one sample per meter of sediment core. The Harrison Transect consisted of 26 one-meter core segments, the Swan and Medimont Transects each with 28, the Cataldo Transect with 44 (total samples = 126). A total of 16 cores were collected at four sediment coring locations within each of the accessible Lateral Lakes: Cave, Medicine, Killarney, and Rose (four cores each), with three samples per three-meter depths (total samples = 48). In addition, sediment core samples were collected from the floodplains at six river transects: Cataldo, Dudley, Killarney, Medimont, Swan, and Harrison. At each floodplain transect, 10 sediment cores were collected at three-meter depths with three samples per three-meter core (total samples = 180). The average depth of mining-impacted sediments was approximately 12 feet. The sediment core samples were collected at depths between 6 meters (20 feet) and 10 meters (approximately 33 feet).

3.2.2 Applicability of Data for Evaluating Background Concentrations

The 90th percentile distribution for the seven metals of concern in soil for all formations sampled in the USGS study (1980) is presented in Table 2-4. The 95th percentile values from this same study are presented in Table 2-5 for ease of comparison to the background concentrations developed later in this section.

Of the seven metals of concern, antimony, copper, lead and mercury appear to partially define mineral belts in the district. Arsenic, cadmium and zinc do not appear definitive of the known mineral belts. As discussed in the USGS Study, when plotted on a map of the district, some metal concentrations below the background threshold can be associated with known mineralization. Metal concentrations above the background threshold (referred to as anomalous) when plotted on a map of the district do not always correlate with known mineralization trends. This would suggest that some of the anomalies reflect metal enrichment by natural processes not directly related to ore deposits.

Use of the USGS data to indicate metal concentrations that are related to mining impacts should recognize a range of background values for each metal of concern as will be discussed in the following sections below. This could account for metal variability in the district not directly related to emplacement of ore deposits.

3.2.3 Estimated Background Concentration for Screening of CUA Sites

This section presents the estimated background concentrations for seven metals of concern that were used in screening the subject CUA sites. The background estimates were based on statistical analysis of laboratory-reported concentrations of antimony, arsenic, cadmium, copper, lead, mercury, and zinc from available sediment samples taken in the lower CDARB, as reported in URSG (1998b). As will be explained, background estimates for antimony at the subject CUA sites also required analysis of laboratory-reported concentrations of antimony, as contained in the project data base, from soil sampling done in the common use areas throughout the basin. The statistical analysis methodology used to make the background estimates is also summarized.

Background concentrations at a given site exist over a range of values, described by the background population distribution. For the subject CUA sites, EPA has selected a background concentration based on the 95th percentile of the background population distribution. Selection of the 95th percentile, or any percentile, is a risk management decision. Use of the 95th percentile helps assure that concentrations below that value are likely to be within the background range.

3.2.3.1 Statistical Analysis Methodology

The statistical analysis methodology used to estimate background concentrations was based on the California EPA (Cal/EPA) final policy on selecting inorganic constituents as chemicals of potential concern for risk assessment at hazardous waste sites (Cal/EPA 1997). For each

chemical, available sample concentrations were analyzed to determine the cumulative frequency distribution (CFD), or cumulative probability plot, of the data. Where the CFD showed two or more populations, "ambient" or background conditions were defined as the distribution of concentrations associated with the lowest-concentration population. The background population distribution was then used to estimate a background concentration based on the 95th percentile of the distribution.

Because trace chemicals generally follow a lognormal probability distribution, the sample concentration data for each chemical was analyzed as a lognormal CFD. For chemicals following a lognormal distribution, the log-transformed concentrations follow a normal distribution, with the log concentrations versus standardized normal variate plotting as a straight line. The plot of log concentration versus standardized normal variate is called a lognormal CFD.

The lognormal CFD for each of the seven chemicals was estimated by the following procedure (adapted from WDOE 1993 and Cal/EPA 1997). First, all non-detected values were given a sample concentration equal to one half of the laboratory detection limit for that sample. Next, all sample concentrations were log transformed by taking the natural logarithm of each concentration. Then, for each chemical, the sample log-transformed concentrations were ranked from lowest to highest. The ranked log-transformed concentrations were then assigned the following plotting positions: (I-3/8)/(N+0.25); were N is the total number of samples, and I is the rank order, from one (lowest concentration) to N (highest concentration). The plotting positions are unbiased estimates of the cumulative probabilities associated with corresponding log-transformed sample concentrations (Cunnane 1978). For each detected concentration, the standardized normal variate, u, associated with the estimated probability, (I-3/8)/(N+0.25), was determined from the inverse standard normal cumulative distribution function evaluated at the estimated probability. To create the lognormal CFD, the standardized normal variates, u, were plotted against the corresponding log-transformed concentration, log concentration, of all samples having detected concentrations (i.e., non-detects were not plotted).

An alternative statistical method applied to lead is described in a report prepared by SRC and can be found in Appendix H. Their methodology took into consideration the geology of the area and local anthropogenic sources of lead other than mining (e.g., automobile emissions and deteriorating lead-based paint). SRC (1999) estimated the local background concentrations of lead in the CDARB to range from 120 to 2,700 ppm. The lower and upper bounds on this range represent the sums of the lower and upper bound estimates, respectively, of the following three major contributing sources to local background:

- Regional sources: 20 - 250
- Exterior lead-based paint: 100 - 2,000
- Automotive emissions: 10 - 450

These three sources are considered to be the major potential anthropogenic sources of soil lead, other than mining and smelting related contamination of soil (SRC 1999).

SRC used the same data set as URSG (collected from the lateral lakes and lower sediment coring) and an alternative statistical method to estimate a 95th percentile value for arsenic as described in a report found in Appendix H. They evaluated different truncation levels in order to investigate the uncertainty associated with where the "bright line" is drawn to distinguish background samples versus the site-impacted samples. The three truncation points at 12.5, 19.7, and 41.8 ppm yielded 95th percentiles of 12.5, 15.9, and 24.1 ppm, respectively. This range of values is lower than the concentrations presented by USGS and URSG, as summarized in Table 2-5. The 35 ppm value calculated by URSG was selected as the background in this study. The uncertainties surrounding the use of these different statistical methods are further discussed in the uncertainty section.

3.2.3.2 Results And Discussion

The lognormal CFD for each chemical resulting from the analysis summarized in the previous section is presented in Appendix D. Using the sediment data from the lower basin, except for mercury and antimony, the lognormal CFDs showed two reasonably distinct straight-line portions. These results are indicative of two mixed lognormal populations: a lower-concentration population and a higher-concentration population. The lower-concentration population was considered representative of the natural background in sediments. The higher-concentration population was considered representative of mining-impacted sediments.

Since the populations were mixed to variable and uncertain extents, the upper tails of the background (lower-concentration) populations were confounded by the lower tails of the mining-impacted (higher-concentration) populations. The mixing manifested itself as curved transition zones between the lower and higher distributions, as evidenced by the figures in Appendix D. Each transition zone was a mix of the upper tail of the lower-concentration population and the lower tail of the higher-concentration population. Unfortunately, it appeared fundamentally impossible to accurately determine which values in the transition zone belonged to the upper tail of the lower-concentration population and which belonged to lower tail of the higher-concentration population.

Therefore, since the upper tail of the lower distributions was obscured in the transition zone, it was necessary to extrapolate the straight-line portion of the lower distribution to estimate a 95th percentile value. As was confirmed by numerical simulations, extrapolation of the lower straight-line portion of the CFD is consistent with mixed lognormal distributions and is a technically valid way to recover the entire, unmixed lower distribution (the same holds for the upper straight-line portion).

For each chemical, the extrapolation was based on a visual best fit using professional judgment, although more mathematically elaborate methods could be used (e.g., weighted least squares regression). The quality (reliability or statistical certainty) of the fit and resultant extrapolation was dependent on the extent and variability of the straight-line portion of the lower population, which varied with the chemicals. Because a substantial number of samples were clearly below the transition zone and above detection limits, the highest quality fits, which appeared to have the highest reliability or statistical certainty associated with the 95th percentile estimate, occurred for arsenic, copper, lead, and zinc. In contrast, the fit for cadmium was limited by the relatively low number of lower population samples that were clearly below the transition zone and above detection limits. Results are presented in Table 2-5.

The advantage of the CFD method of estimating background populations is that it avoided the need to first determine which individual samples belonged to the background population and which were mining affected. As discussed, this determination cannot be made accurately where the tails of the two distributions overlap (which occurs in the transition zone) nor is it clear, without the CFD, where the overlap occurs. This difficulty is reflected in the data. For example, when sediment concentrations were plotted as a function of depth for all locations combined, it was found that concentrations at a given depth varied from low to high, with no clear way to distinguish background from mining-affected samples. When concentrations were plotted as a function of depth at given locations, there was also no clear way to separate background from mining-affected samples. That is, cores displayed concentrations that could increase, decrease, or remain reasonably constant with depth, and with relatively low or high values at any depth, although if the cores were deep enough, high values did stop occurring. In contrast, the lognormal CFD showed, with reasonable clarity, which samples belonged to the lower, background population; which samples belonged to the upper, mining-affected population; and which samples could belong to *either* the lower *or* the upper populations (i.e., the samples in the transition zone).

However, the CFD method fails when two (straight-line) populations are not evident, as was the case for mercury and antimony. In both cases, it appeared that the lower population samples that were below the transition zone were also below the sample detection limits. The concentrations of samples below the detection limit are censored and cannot elucidate the shape of the lower population. Therefore, a straight-line portion of the background population could not be estimated from the sediment samples for either mercury or antimony.

In an attempt to estimate a background concentration for antimony, all available soil samples from common use areas throughout the basin were analyzed to determine if the straight-line portion of a lower population could be discerned. The lognormal CFD using the soil samples was determined in the same way as for the sediment samples. The lognormal CFD, presented in Appendix D, did show a lower population that, although somewhat ambiguous, could be reasonably extrapolated to estimate a 95th percentile. Therefore, the lower population from the common use soil sampling

was used to estimate the background concentration for the subject CUA sites. The result is presented in Table 2-5.

Unfortunately, the approach used for antimony did not work for mercury. Similar to the sediment samples, the common-use-area soil samples indicated that lower population samples that were below the transition zone were also below the sample detection limits for mercury. Thus, the lower population for mercury could not be determined.

However, to provide some rational basis for an estimate, an attempt was made to separate potential background concentrations for mercury by evaluating sediment concentrations at each sampling location. Samples were judged to be potentially representative of background if concentrations were non-detect or close to non-detect, particularly if a significant decrease of concentration occurred with depth. As discussed, because of mixing and confounding, this judgment-based process was recognized as likely to misjudge some samples as background when they were not, and vice versa.

One hundred-fifty two (152) samples were judged as potentially representative of background for mercury. A lognormal CFD was determined, as presented in Appendix D, using the 152 samples. The fit to a lognormal CFD was rather poor, reflecting the inaccuracy of the judgment-based selection process. Therefore, the estimate of a 95th percentile background was simply taken as the 95th percentile of the 152 samples potentially representative of background for mercury. The result is presented in Table 2-5.

The 95th percentile estimates presented in Table 2-5 are reasonably consistent with the 95th percentile concentrations from the USGS Study (Gott and Cathrall 1980), as summarized in Section 2.2.2 and also presented in Table 2-5. This consistency helps empirically validate that the lower-concentration distributions identified in the lognormal CFDs are representative of background population distributions. Thus, the URSG estimates in Table 2-5 are considered representative of true background concentrations for use in screening the subject CUA sites.

The following is a summary of this section, "Estimated Soil Background Concentrations Used for Screening CUA Sites":

- USGS studied a large 300 square mile area centered on the CDARB, which includes variations in natural geology and levels of mining and ore processing impacts.
- The 'true' natural background levels are a range rather than a single value.
- Arsenic sites will be screened at 35 ppm, which was selected as the 95th percentile of the range of background values calculated using the Cal EPA methodology.

- Selection of 35 ppm as the 95th percentile value for arsenic resulted from a combination of quantitative data analysis, professional judgement, and risk management by EPA Superfund Management.

- The method used by SRC is also valid and not necessarily in conflict with the Cal EPA methods (see discussion in uncertainty section).

- The USGS soil results provide additional confidence in both analyses.

Table 2-1
Summary of the Number of Samples Collected at the CUA Sites

Site	Site Name	Soil[a]	Sediment[b]	Surface Water	Drinking Water
1	N. Idaho College Beach-along Spokane River	9	14	8	ns
2	N. Idaho College Beach-along Lake	ns	17	7	ns
3	Post Falls City Beach/River Park	10	16	8	ns
5	Green Ferry Bay County Park	7	16	7	ns
6	Black Bay	ns	14	7	ns
7	BLM Pump Station	8	15	8	ns
8	Corbin Park	ns	15	8	ns
9	Coeur d'Alene Beach at City Park	ns	29	8	ns
10	Tubbs Hill (site 1)	ns	15	7	ns
11	Tubbs Hill (site 2)	ns	26	8	ns
12	Tubbs Hill (site 3)	ns	15	8	ns
15	Higgan's Point (site 1)	ns	15	7	ns
16	Higgan's Point (site 2)	ns	16	8	ns
17	Harrison Beach (site 1 - West)	7	ns	8	1
18	Harrison Beach (site 2 - North)	ns	31	9	ns
19	Cougar Bay	8	15	8	ns
21	Blackwell Island	9	16	8	ns
23	Bell Bay	7	2	8	ns
24	Mica Bay	10	15	7	ns
25	Rockford Bay	8	ns	ns	ns
26	Loffs Bay	10	10	10	ns
27	Windy Bay	8	2	7	1
29	Spokane Point (on reservation)	ns	7	8	ns
30	Fuller Landing	1	ns	7	ns

Notes:
ns - not sampled
Number of samples includes field duplicates
BLM - Bureau of Land Management
a) Soil samples collected from the top inch of soil
b) Sediment samples include those collected from 0 to 6 inches and from 0 to 12 inches.

Table 2-2 Summary of Analytical Results for Soils and Sediments

Table 2-3 Summary of Analytical Results for Surface Water

Table 2-4
USGS 90th Percentile Background Concentrations

Chemical	USGS 90th Percentile (mg/kg)	Number of Samples
Antimony	5.8	8,153
Arsenic	22	8,265
Cadmium	2.7	7,176
Copper	53	8,695
Lead	171	8,514
Mercury	0.3	8,124
Zinc	280	8,684

Table 2-5
Estimated 95th Percentile Background Concentrations for Use in Screening CUAs

Chemical	SRC 95th Percentile Range[a] (mg/kg)	Estimated USGS 95th Percentile[b] (mg/kg)	URSG Estimated 95th Percentile[c,d] (mg/kg)	Relative Quality of URSG Estimate
Antimony	NA	7.5	5	Moderate
Arsenic	12.5, 15.9, 24.1	28	35	High
Cadmium	NA	4.2	8	Moderate
Copper	NA	60	45	High
Lead	120 to 2,700	280	150	High
Mercury	NA	0.55	1[e]	Low
Zinc	NA	420	220	High

[a]95th percentiles estimated using Regression Statistics
[b]95th percentiles of USGS data visually estimated from the CFD by URSG
[c]Antimony, arsenic, cadmium, copper, lead, and zinc background concentrations based on straight-line lognormal extrapolation of population closest to origin to the 95th percentile of lognormal distribution. Arsenic, cadmium, copper, lead, and zinc estimates based on 283 total available sediment samples from the lower basin. Antimony estimates based on 338 total available soil samples for common use areas throughout the basin.
[d]Relative quality of estimate is a qualitative judgment of the reliability or statistical certainty associated with background estimate, and is based on the number of lower-concentration samples that are clearly below the transition zone and above the detection limit and the apparent "goodness of fit" to a straight line.
[e]Mercury estimate based on 95th percentile of 152 potential background concentrations selected from 283 total available sediment samples from the lower basin.

Notes:
CUAs - common use areas
NA - not available

4.0 ESTIMATION OF LEAD RBCs AT CUAs

The current EPA risk assessment method for evaluating lead uses a mathematical model to estimate blood lead levels in children (age zero to seven years). The EPA model is referred to as the Integrated Exposure Uptake Biokinetic Model (IEUBK Model). EPA version 0.99d of the model was used following recent EPA guidance (EPA 1994a, b, c, and d).

The IEUBK Model combines assumptions about lead exposure (environmental lead concentrations, intake rates), and uptake (absorption factors for air, diet, water, soil), with assumptions on how lead behaves in the body (biokinetic parameters) to predict a central tendency estimate (CTE) blood lead concentration for a child. In addition, an estimation of variation in blood is applied to the CTE to predict the probability of an individual child exceeding a given blood lead level. In accordance with EPA policy (EPA 1994d), soil and water RBCs for recreational exposure at the CUAs were estimated based on a model prediction of no more than 5 percent risk for a child to have a blood lead level exceeding 10 µg/dL.

4.1 OBJECTIVES

The objective of this effort is to evaluate lead exposures at CUAs against risk-based criteria in order to identify those CUAs that: (1) must be more thoroughly evaluated in the baseline HHRA; or (2) may not need further evaluation in the baseline HHRA because projected lead risks are sufficiently low.

4.2 GENERAL APPROACH

1. Risk-based concentrations (RBCs) for lead are based on estimated risks from exposures to children.

2. The underlying assumption is that CUA exposures that pose *sufficiently low risks to children* will also pose sufficiently low risks to fetuses carried by women who are exposed to the CUA.

3. Sufficiently low risk to children is defined for the purpose of deriving RBCs for lead as a probability of exceeding a blood lead concentration (PbB) of 10 µg/dL that is no greater than 5 percent (i.e., $P_{10} \leq 5$ percent).

4. Lead risks (P_{10}) are estimated using the Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children (U.S. EPA 1994a, b, and c).

5. Lead exposures are modeled by *summing* exposures at the CUA with assumed *post-remediation* or *background* exposures expected at the residence.

6. Exposure factors used in modeling lead risk are intended to be as consistent as possible with factors used to assess other chemical risk at the site, to the extent that such consistency does not conflict with the IEUBK model concept and can be accommodated by software to implement the IEUBK model (U.S. EPA 1994a, b, c).

4.3 MODELING APPROACH

The IEUBK model was used to construct matrices of P_{10}s for ranges of residential and CUA lead exposure concentrations (e.g., residential and CUA soil lead concentrations). An example of this approach is presented in Tables 3-1 and 3-2. Table 3-1 shows the central tendency estimates (CTEs) of PbBs for 50 different combinations of residential soil lead concentration (PbS_{res}, range = 50 - 400 ppm) and CUA soil lead concentrations (PbS_{cua}), (range = 400 - 4,000 ppm). For example, exposure to 200 ppm PbS_{res} and 1,200 ppm PbS_{cua} would correspond to an estimated CTE PbB of 4.4 µg/dL. Table 3-2 presents the estimated P_{10} (percent probability of exceeding 10 µg/dL) that corresponds to each of the CTE PbBs in Table 3-1. For example, exposure to a PbS_{res} of 200 ppm and a PbS_{cua} of 1,200 ppm would correspond to a P_{10} of 4.1 percent; exposure to a PbS_{res} of 400 ppm and a PbS_{cua} of 600 ppm would correspond to a P_{10} of 4.6 percent, and so forth. Figure 3-1 plots the P_{10} vs the PbS_{cua} for various assumed values of PbS_{res}. The RBCs for each PbS_{res} scenario can be estimated by dropping a vertical line to the PbS_{cua} axis from the intercept with the horizontal P_{10} = 5 percent line. The RBC_{soil} for this CUA, assuming a residential PbS_{res} of 200 ppm and all other exposure assumptions as described below, would be approximately 1,400 ppm.

Each CTE PbB shown in Table 3-1 is the arithmetic mean of six PbBs; one for each of six age-years in which contact with the CUA was assumed to occur (i.e., age-years two, three, four, five, six, or seven). For example, the CTE PbB value of 4.4 µg/dL for the combined exposure to a PbS_{res} of 200 ppm and a PbS_{cua} of 1,200 ppm is derived from the results of the six age-year IEUBK model simulations, shown in Table 3-3. In the first simulation, no CUA contact was assumed to occur (U.S. EPA 1998a). In the second simulation, contact with the CUA was assumed to occur during age-year two (months 12–23); the resulting CTE PbB corresponding to the year of CUA contact (age-year two) was 5.8 µg/dL (shown in bold in Table 3-3). In the third simulation, CUA contact was assumed to occur during age-year three, and the resulting CTE PbB was 5.3 µg/dL, and so forth. The mean of the six beach-impacted PbBs was 4.4 µg/dL, shown in the lower right corner of Table 3-3; this value was used to represent the CTE PbB for the combined

exposure to PbS_{res} of 200 ppm and PbS_{cua} 1,200 ppm, shown in Table 3-1. The basis for averaging of the age-year PbBs is the assumption that contact with the CUA is seasonal and will occur only for a fraction of the year, and that contact is random with respect to age; that is, there is an equal likelihood for contact with CUA soil at any age. Note, exposures cannot be simulated for durations less than one year, because the IEUBK model uses a fixed time step of one year for all exposure variables.

4.4 IMPORTANT ASSUMPTIONS AND UNCERTAINTIES ASSOCIATED WITH THE MODELING APPROACH

Table 3-2 presents an estimate of risk (P_{10}) associated with one-year exposures, two days per week, to the CUA soil (see below for further discussion of exposure frequency). In deriving this estimate, two important simplifying assumptions were made that depart from the expected exposure: (1) an exposure duration of one year was assumed, whereas the expected exposure is seasonal (≤ 6 months per year); (2) the exposure was assumed to occur within a single age year for a given child, whereas, repeated seasonal exposures are likely.

All examples of risk estimates and RBCs shown in subsequent sections of this report are based on the two assumptions discussed in the preceding paragraph. Assumption 1 will tend to result in predictions of higher age-year PbBs than might be expected after seasonal exposures, because elimination of a part of the CUA-associated lead burden would be expected during the part of the year in which CUA exposure does not occur (post-seasonal). Assumption 2 will tend to result in lower predicted PbBs than might be expected for multiple age-year exposures to a child, because the CUA-associated lead burden that is not eliminated during the post-seasonal period is not accumulated across age-years. This is shown in Figure 3-2, which presents the risk estimates (P_{10}s) obtained when exposures are assumed to occur for all years between ages two and seven years. The mean PbBs for ages 2–7 years and the corresponding P_{10}s are slightly higher than predicted for single age-year exposures (Figure 3-1). For example, the P_{10} corresponding to exposure to a PbS_{res} of 200 ppm and a PbS_{cua} of 1,200 ppm is 6.0 percent when multiple-year exposures are assumed and 4.1 when single age-year exposures are assumed. These two risk estimates can be interpreted as bounding estimates for this residential-CUA exposure scenario; risk can be expected to be within the range of 4.1 percent to 6.0 percent. All examples of risk estimates and RBCs shown in subsequent sections of this briefing report are based on the aggregation of age-year exposures. The rationale is that these estimates are adequately conservative given the exposure assumptions used in the model and given high confidence that CUA exposures are seasonal and limited to annual durations of no more than 6 months.

4.5 EXPOSURE ASSUMPTIONS

Total lead intake ($INTAKE_{total}$) is defined for the purpose of this screening assessment as the sum of lead intakes at the residence ($INTAKE_{res}$) and lead intake at the CUA ($INTAKE_{cua}$):

$$INTAKE_{total} = INTAKE_{res} + INTAKE_{cua}$$

Lead intake at the residence is estimated using the IEUBK model as the sum of intakes resulting from exposure to lead in air, food, drinking water, soil, and house dust at the residence:

$$INTAKE_{res} = INTAKE_{air,res} + INTAKE_{food,res} + INTAKE_{water,res} + INTAKE_{soil,res} + INTAKE_{dust,res}$$

Lead intake at the CUA is defined for the purpose of this screening assessment as the sum of intakes from ingestion of soil (upland recreational areas, $INTAKE_{soil,cua}$) or sediment (beaches, $INTAKE_{sed,cua}$), and ingestion of surface water and suspended sediment while swimming and wading at beaches ($INTAKE_{water/sed,cua}$):

$$INTAKE_{cua} = INTAKE_{soil\ or\ sed,cua} + INTAKE_{water/sed,cua}$$

The exposure variables considered in estimating lead intake at CUAs include: exposure frequency, soil or sediment ingestion rate, soil or sediment lead concentrations at the CUA, surface water ingestion during swimming and wading activities, and surface water/sediment lead concentrations at the CUA. The bases for assumptions regarding these variables are described in the sections that follow (Sections 3.5.1 - 3.5.4).

4.5.1 Exposure Frequency

The IEUBK model does not use an explicit variable for exposure frequency. Media intakes (e.g., soil ingestion rates) used in the model represent average daily intakes over an age-year and lead intakes are calculated assuming the average media intakes for every day in the age-year (U.S. EPA 1994a and b). This is computationally equivalent to assuming an exposure frequency of seven days per week. In order to simulate the soil ingestion (or ingestion of other media) that might occur on days in which children visit a CUA, an incremental increase in soil ingestion associated with the CUA was calculated. This was distributed across each day of the age-year according to an assumed CUA exposure frequency (see Sections 3.5.3 as an example).

An exposure frequency (EF_{cua}) of two days/week was chosen to represent a reasonably typical frequency of seasonal contact with the CUAs. The estimate of twice per week is based on professional judgement and takes into consideration the climate of the CDARB. The estimate is consistent with data on child outdoor activity patterns in the upper CDARB (Jacobs Engineering et al. 1989) and with the U.S. EPA Exposure Factors Handbook (U.S. EPA 1997a), as is discussed in greater detail in Section 5.1.3.4 of this report. However, it is likely that the exposure frequency

for children varies in the CDARB, depending on the accessibility of the CUA. To explore this possibility further, CUAs were classified into four exposure frequency categories:

1. Relatively remote or limited access sites

2. Popular public use areas, such as public beaches and parks, that are easily accessed by automobile and not adjacent to residential areas

3. Sites adjacent to residential areas and/or readily accessible to young children (e.g., on foot with an older sibling)

4. High-use sites where regular extensive contact is expected, such as play areas adjoining schools and daycare centers

These exposure frequency categories are broken down by age group in Table 3-4.

Note, exposure to CUAs was assumed to be minimal during the first year post-natal. The RBCs corresponding to each EF_{cua} category are shown in Table 3-5. The RBCs would be approximately 2.5 times higher at CUAs that are highly accessible to children (EF_{cua} category 4 compared to category 2).

4.5.2 Soil Ingestion Rate (IR_S)

The EPA Technical Review Workgroup (TRW) for Lead has recommended values for nonresidential soil ingestion rates in children to be used in the IEUBK model (U.S. EPA 1998a). This approach identifies four categories of intensity of soil ingestion at nonresidential sites: low, intermediate, medium, and high. In each category, soil ingestion during the first year of life is assumed to be represented by the IEUBK model default values. The high-intensity category, 200 mg/day, corresponds to EPA Office of Solid Waste and Emergency Response (OSWER) guidance for the reasonable maximum exposure (RME); this value was selected as the RME value used in the risk assessment for contaminants other than lead, as discussed elsewhere in this report (Section 5.1.3.1). For the purpose of predicting the PbB CTEs associated with CUA contact, the medium category values recommended by the TRW were used in the IEUBK model. These values are assumed to represent CTEs of soil ingestion at the various CUAs where soil ingestion is expected to be, on average, higher than at the residence (e.g., river shorelines and beaches, soil surface play areas, trails). The medium soil ingestion values identified by the TRW fall between the 90th and 95th percentile range of empirically derived estimates of soil ingestion in children.

Based on an analysis of all data available on soil ingestion rates in children, the cumulative distribution function (CDF [similar to CFD presented in Section 2.2]) for soil ingestion in children ages 1 to 4 years was estimated (Goodrum and Diamond 1998) based on the data reported in

Calabrese et al. (1989). This CDF is given in Table 3-6. The IEUBK model default for age range years 2 to 4, 135 mg/day, corresponds to the 83rd percentile of this CDF. The intermediate, medium, and high categories recommended by the TRW correspond to the 87th , 92nd, and 99th percentiles, respectively, of the empirical CDF. The medium age-specific values for soil ingestion used in the IEUBK model simulations are presented in Table 3-7.

Note, consistent with U.S. EPA (1998a), soil ingestion is not assumed to be higher than the model default value for the first post-natal year because contact with CUA soil is expected to be minimal during the first six to eight months. The soil ingestion rates shown in Table 3-5 were assumed for all CUAs, although it is likely that soil ingestion varies depending on surface characteristics and activity. The assumption is that, on average, we expect soil ingestion rates at CUAs to be reasonably represented by these values.

4.5.3 Soil Lead Ingestion at the CUA

The assumed age-specific ingestion rates at the residence are shown in Table 3-7 and are those recommended for use in the IEUBK model (U.S. EPA 1994a and b).

Soil lead ingestion at the CUA ($INTAKE_{soil,cua}$, μg/day) corresponding to the above $IR_{s,cua}$ and EF_{cua} was calculated as follows:

$$INTAKE_{soil,cua} = PbS_{cua} \times IR_{s,cua} \times EF_{cua}$$

where PBS_{cua} is the soil or sediment concentration, $IR_{s,cua}$ is the age-specific value for the *incremental* soil ingestion shown in Table 3-7, and EF_{cua} is expressed as the fraction of the week spent at the CUA (e.g., 2 days/7 days). The incremental CUA-related soil ingestion rates ($IR_{s,cua}$) represent the incremental (above the amount expected at the residence) soil or sediment ingestion attributed to activities at CUA beaches. This is conceptually equivalent to assuming that, on days at which a child visits a CUA, the soil ingestion is assumed to be one of the age-specific values shown for *Total* intake (fourth column in Table 3.7). The model actually computes the associated CUA-related increment in lead intake as $INTAKE_{soil,cua}$ (from the above equation) occurring, in addition to all other sources of lead intake, on each day of an age-year (i.e., seven days per week). $INTAKE_{cua}$ was represented in the IEUBK model as Other (Alternate Source) lead intake (U.S. EPA 1998a).

4.5.4 Surface-Water Ingestion While Swimming or Wading

Nearly all the CUAs in the CDARB include river and lake shore areas where children play at the land/water boundary, and where the potential for ingestion of surface water and suspended sediments exists. In order to model the cumulative exposures from soil water/sediment ingestion at these CUAs, lead intake from the water/sediment pathway was estimated and included in the

IEUBK model. Intakes from the CUA soil and water/sediment were summed to yield the total average daily lead intake associated with exposures at the CUA, and the sum was included in the Other (Alternate Source) input to the model:

$$INTAKE_{cua} = INTAKE_{soil\ or\ sed,\ cua} + INTAKE_{water/sed,\ cua}$$

Water/sediment intake $INTAKE_{water/sed,cua}$ at the CUA was estimated as follows:

$$INTAKE_{water/sed,cua} = PbW/S_{cua} \times IR_{w/s,cua} \times EF_{cua}$$

where PbW/S_{cua} is the total lead concentration in the surface water, including suspended sediment, and EF_{cua} is the fraction of the week spent at the CUA. An exposure frequency of 2 days per week and an ingestion rate of 30 mL/hour was assumed for the swimming or wading exposure scenario (see Section 5.1.3 for an explanation of the 30 mL/hour value).

4.6 LEAD UPTAKE FROM THE DERMAL ROUTE

Swimming and wading at shorelines and beaches may result in dermal contact with dissolved lead in the water column. The IEUBK model does not have an exposure or biokinetic module for translating such exposures into estimates of PbB. However, the absorption algorithms used in the IEUBK model to calculate lead uptake from the gastrointestinal tract can be reproduced and implemented outside of the IEUBK model. This allows one to estimate annual average rates of lead uptake associated with various exposure scenarios. Using this approach, it can be shown, that for plausible ranges of water lead concentrations, lead uptake from dermal absorption will be insignificant relative to other exposure pathways. Table 3-8 compares the estimated lead uptakes from dermal contact and ingestion of water while swimming to rates of uptake estimated for exposure to residential and CUA soil. The table shows that for dissolved lead concentrations in water as high as 4,000 parts per billion (ppb), lead uptake from the dermal route is less than 0.1 percent of total uptake from ingestion of surface water and soil. Thus, for the purpose of deriving lead RBCs due to surface water contact, the dermal absorption pathway is assumed to be insignificant.

Table 3-9 shows a similar comparison for dermal uptake of lead from soil and uptake associated with ingested lead at the residence and CUA. The dermal pathway appears to be a significant pathway for both the upland and beach scenarios, accounting for 5 to 16 percent of total soil lead uptake for the upland dermal exposure scenario and, 17 to 37 percent of total for the beach dermal scenario. The above estimates assume a dermal absorption fraction of 0.01 for soil lead, for which there is little if any direct empirical support. U.S. EPA (1992a) cites a range of 0.001 to 0.01 for the dermal absorption fraction of cadmium. If the low end of this range is representative of dermal lead absorption, the dermal contribution to total uptake would be a factor of 10 lower

than those shown in Table 3-9. Additional examination of empirical support for values for the dermal absorption fraction of soil lead is warranted to assess whether RBCs for soil may be underestimated by not accounting for the dermal pathway.

4.6.1 Other Exposure Variables

Default values were assumed for all other IEUBK model variables (U.S. EPA 1994a, b, c). The mass fraction of soil in indoor dust (M_{sd}) at the residence was assumed to be 0.7, the IEUBK model default; this corresponds to a concentration of soil-derived lead in indoor dust of 0.7 times the assumed soil lead concentration ($PbDust_{res} = 0.7 \times PbS_{res}$).

4.7 RBCs FOR UPLAND CUAs

Exposure pathways for upland CUAs will include dermal contact and ingestion of soil. Table 3-7 shows the P_{10}s for the ingestion pathway, with various assumptions made about exposure frequency and residential soil lead concentrations. At an exposure frequency of 2 days/wk, if the residential soil lead concentration is assumed to be 200 ppm, the RBC for CUA soil is approximately 1,400 ppm. If the residential soil lead concentration is assumed to be 300 ppm, the RBC for CUA soil is approximately 500 ppm.

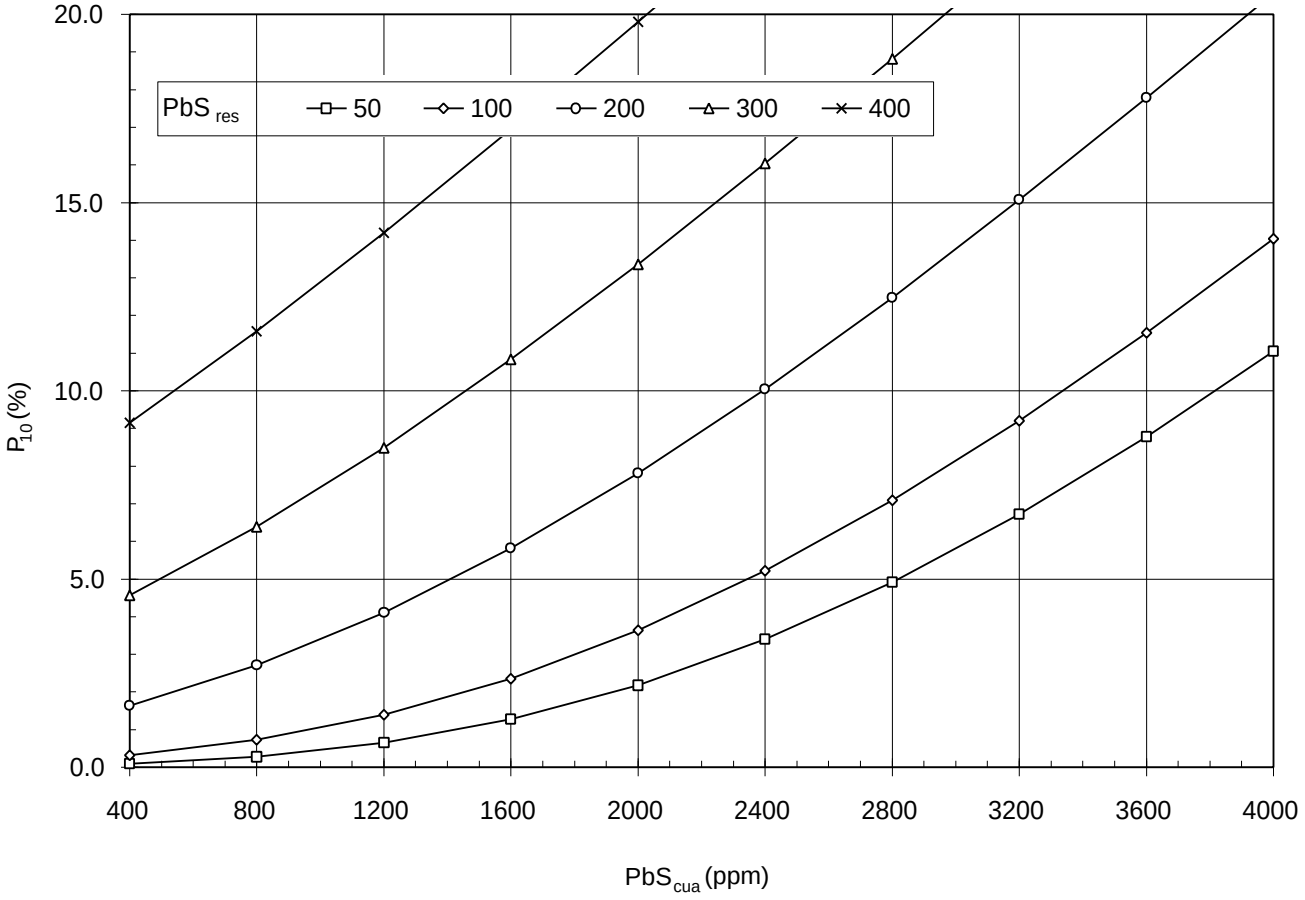
4.8 RBCs FOR CUA SHORELINES AND BEACHES

Exposure pathways considered at shorelines and beaches include dermal contact and soil ingestion and dermal contact and ingestion of surface water and sediment during wading or swimming. The RBCs for the combined ingestion pathways in a swimming scenario are shown in Figure 3-3 (the dermal pathways cannot be estimated with the IEUBK model). Figure 3-3 is a plot of the RBCs for ingested water/sediment lead vs CUA soil lead concentration. Each line in Figure 3-3 corresponds to a different assumption about soil lead concentration at the residence. For example, the water/sediment RBC corresponding to a residential soil concentration of 200 ppm, and a CUA soil concentration of 1,000 ppm is approximately 700 µg/L. Figure 3-6 shows P_{10s} and RBCs that correspond with the combination of ingestion of soil, sediment, and surface water (the dermal pathway cannot be estimated with the IEUBK model), in addition to exposure at the residence, and assuming a CUA soil lead concentration of 100 ppm.

4.9 COMPUTATIONS

The PbBs used in the derivation of P_{10}s (e.g., Table 3-3) were calculated using a batch file processor that was developed for the IEUBK model software, v.0.99. The P_{10}s corresponding to

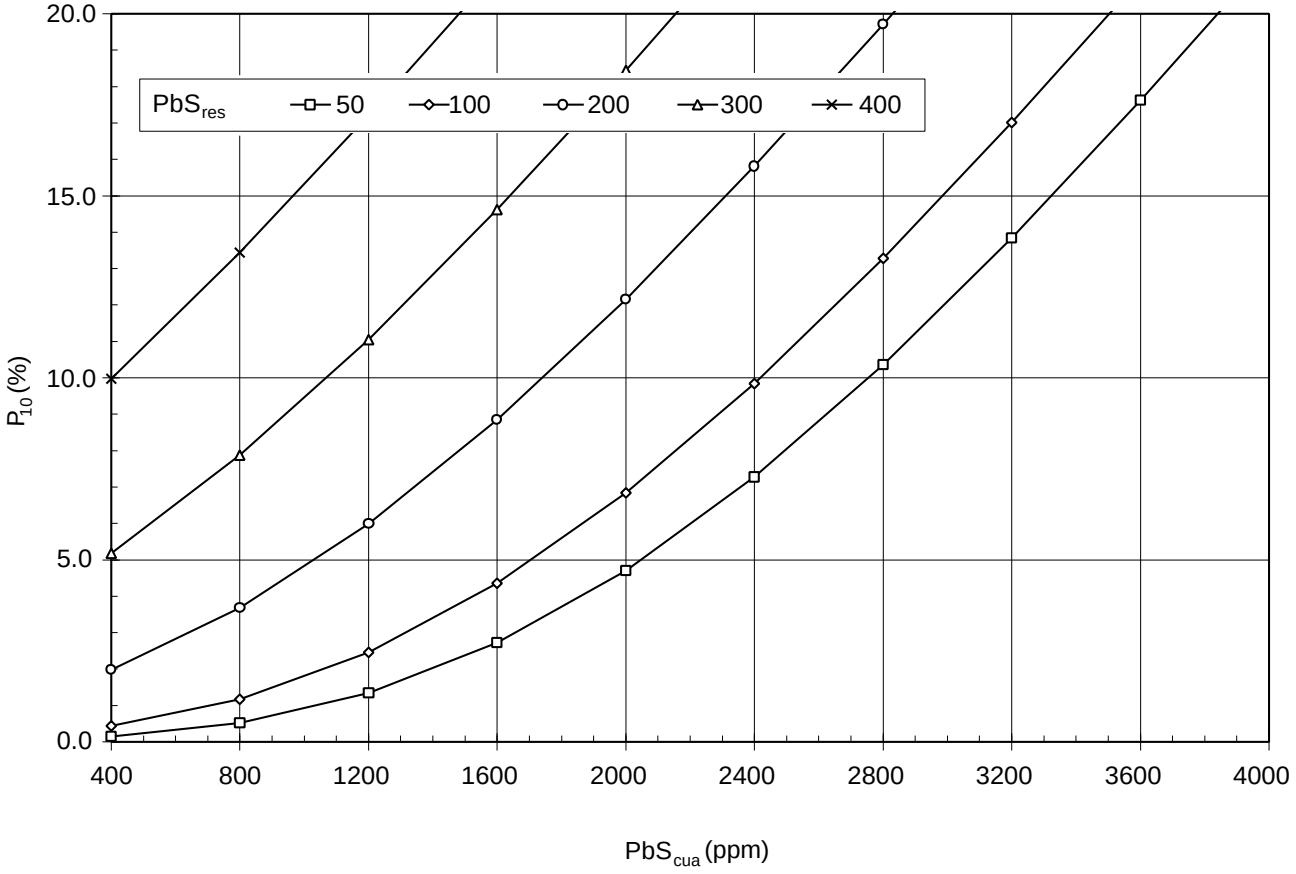
the CTE PbBs were estimated using the 5th-degree polynomial approximation of Hastings (1955). This approximation agrees well with the graphical approximation method used in the IEUBK model and can be implemented in a spreadsheet. All calculations made outside of IEUBK model v. 99 were made using Microsoft Excel, v. 7.0.



(EF_{cua} = 2 dy/wk, ED = 1 yr, $IR_{s,cua}$ = 0.45 x default)

EPA REGION 10	**Figure 3-1** **Lead RBCs for Soil Ingestion Scenario** **(One Year Exposure Duration)**	54-50-0C2Q Draft Final Coeur d'Alene RI/FS CUA RISK ASSESSMENT CDARB

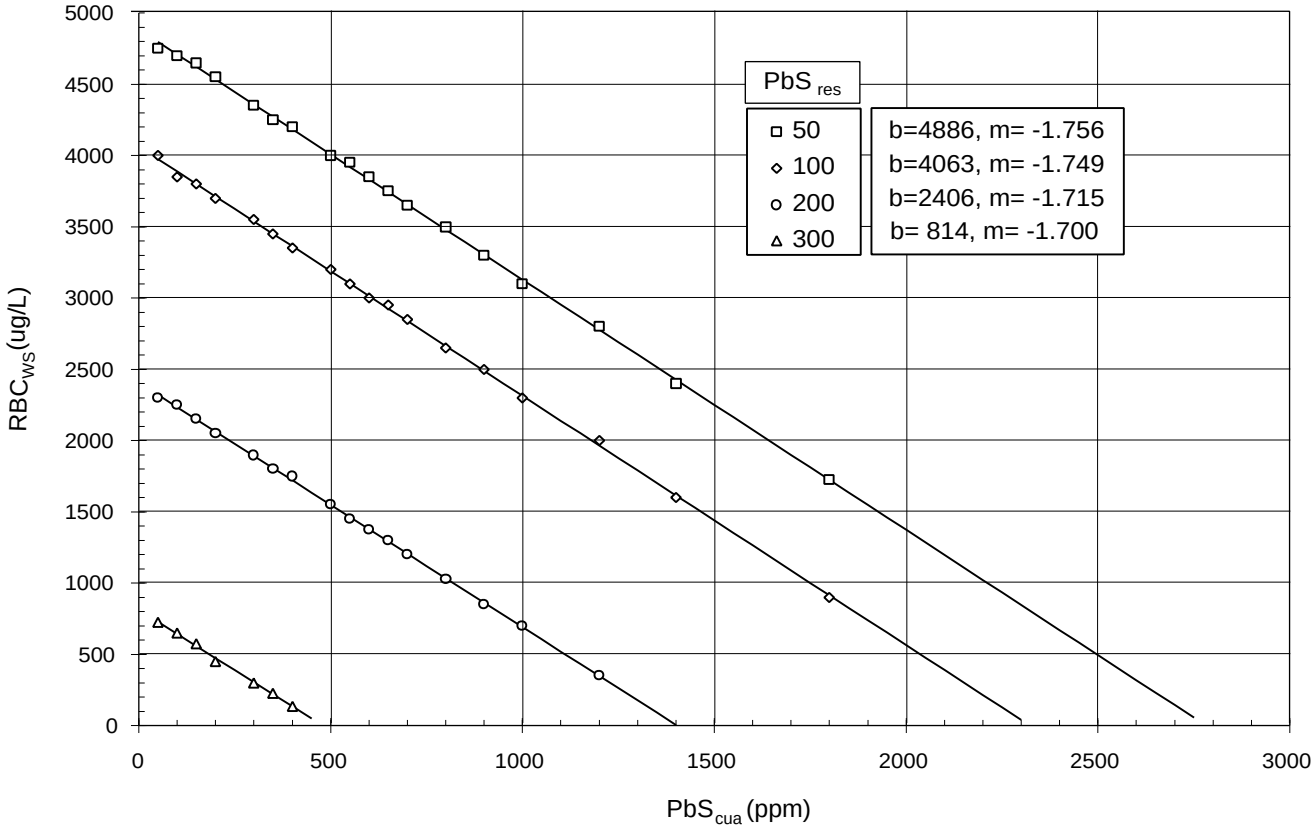
53F40027000701700-2-101299



(EF_{cua} = 2 dy/wk, ED = 6 yr, $IR_{s,cua}$ = 0.45 x default)

EPA REGION 10	**Figure 3-2** **Lead RBCs for Soil Ingestion Scenario** **(Six Year Exposure Duration)**	54-50-0C2Q Draft Final Coeur d'Alene RI/FS CUA RISK ASSESSMENT CDARB

53F40027000701700-3-101299



Note: Linear regression equations for each PbS_{res} are shown.

EPA REGION 10	**Figure 3-3** **Relationship Between Water/Sediment RBCs for Swimming Scenario and Soil Lead Concentration at the Beach (PbS_{cua}) for Four Residential Soil Lead Levels (PbS_{res} = 50, 100, 200, 300 ppm)**	54-50-0C2Q Draft Final Coeur d'Alene RI/FS CUA RISK ASSESSMENT CDARB

Table 3-1
PbB Central Tendency Estimate (µg/dL)

PbS (res)	**PbS_{cua}** (ppm)									
	400	**800**	**1200**	**1600**	**2000**	**2400**	**2800**	**3200**	**3600**	**4000**
50	2.3	2.7	3.1	3.5	3.9	4.2	4.6	4.9	5.3	5.6
100	2.8	3.2	3.6	3.9	4.3	4.7	5.0	5.4	5.7	6.0
200	3.7	4.0	4.4	4.8	5.1	5.5	5.8	6.2	6.5	6.8
300	4.5	4.9	5.2	5.6	5.9	6.3	6.6	6.9	7.2	7.5
400	5.3	5.7	6.0	6.4	6.7	7.0	7.4	7.7	8.0	8.3

Table 3-2
P_{10} (%) Corresponding to PbB Central Tendency Estimate

PbS (res)	**PbS_{cua}** (ppm)									
	400	**800**	**1200**	**1600**	**2000**	**2400**	**2800**	**3200**	**3600**	**4000**
50	0.1	0.3	0.7	1.3	2.2	3.4	4.9	6.7	8.8	11.1
100	0.3	0.7	1.4	2.4	3.6	5.2	7.1	9.2	11.5	14.0
200	1.6	2.7	4.1	5.8	7.8	10.0	12.5	15.1	17.8	20.6
300	4.6	6.4	8.5	10.8	13.4	16.0	18.8	21.7	24.5	27.4
400	9.1	11.6	14.2	17.0	19.8	22.7	25.6	28.5	31.4	34.3

Table 3-3
Age-Specific PbB Central Tendency Estimate (µg/dL)

Age (m)	**Age-Year of CUE Exposure** (m)							
	0-11	**12-23**	**24-35**	**36-47**	**48-59**	**60-71**	**72-84**	**12-84**
0-11	3.7	2.2	2.2	2.2	2.2	2.2	2.2	
12-23	2.3	**5.8**	2.3	2.3	2.3	2.3	2.3	
24-35	2.1	2.9	**5.3**	2.1	2.1	2.1	2.1	
36-47	2.0	2.0	2.8	**4.9**	2.0	2.0	2.0	
48-59	1.8	1.8	1.8	2.7	**4.0**	1.8	1.8	
60-71	1.7	1.7	1.7	1.7	2.4	**3.4**	1.7	
72-84	1.6	1.6	1.6	1.6	1.7	2.2	**3.1**	
12-84								**4.4**

Table 3-3
Age-Specific PbB Central Tendency Estimate (µg/dL)

Notes:

CUA Exposure Factors Input to Model

IR_{cua} - mg/d	1.45 x default
EF - dy/dy	2/7
ED - yr	1 (age year 2, 3, 4, 5, 6, or 7)
PbDust - µg/g	0.7 x PbS_{res}
PbAir - µg/m^3	default
PbWater - µg/L	default
PbDiet - µg/d	default

CUA - common use area
PbB - blood lead concentration
PbS - lead concentrations in soil
ppm - parts per million
µg/dL - micrograms (of lead) per deciliter (of blood)

Table 3-4
Exposure Frequency to CUAs by Age

Age (months)	**Exposure Frequency (EF_{cua})** (days/week) **by Category**				**Outdoor Time**[1] (hr)
	1 remote/low accessibility	**2 moderate accessibility**	**3 high accessibility**	**4 high use**	
0–11	0	0	0	0	1
12–23	1	2	3	5	2
24–35	1	2	3	5	3
36–47	1	2	3	5	4
48–59	1	2	3	5	4
60–71	1	2	3	5	4
72–84	1	2	3	5	4

Table 3-5
Soil RBCs Corresponding to Various CUA Exposure Frequencies

PbS_{res} (ppm)	**Soil RBC** (ppm)			
	Exposure Frequency (days/week)			
	1	**2**	**3**	**4**
50	5625	2800	1875	1125
100	4700	2650	1500	935
200	2800	1400	950	565
300	1000	500	325	200
400	--	--	--	–

Table 3-6
CDF for Soil Ingestion in Children Ages 1–4

CDF Statistic	Soil Ingestion Age 2–4 Years[a] (mg/day)
minimum	0
25th percentile	10
50th percentile	45
75th percentile	88
90th percentile	186
95th percentile	208
99th percentile	225
maximum	7,000

[a]From Goodrum and Diamond 1988

Notes:
CDF - cumulative distribution function, similar to CFD (cumulative frequency distribution) presented in Section 2.2
mg/day - milligrams per day

Table 3-7
Medium Category[a] Age-Specific Values for Soil Ingestion in Children[b]

Age	Default (mg/day)	Increment (mg/day)	Total (mg/day)
0–11	0.085	0.000	0.085
12–23	0.135	0.061	0.196
24–35	0.135	0.061	0.196
36–47	0.135	0.061	0.196
48–59	0.100	0.045	0.145
60–71	0.090	0.041	0.131
72–84	0.085	0.038	0.123
Mean	0.109	0.044	0.153

[a]TRW report (U.S. EPA 1998a)
[b]Values from this table were used in the expedited screening level CUA risk assessment. The *incremental* values were used to represent the incremental (above the amount expected at the residence) soil or sediment ingestion attributed to activities at CUA beaches.

Note:
mg/day - milligrams per day

Table 3-8
Lead Uptake Resulting From Dermal Contact With Water While Swimming, Incidental Ingestion of Water While Swimming, and Uptake From Ingestion of CUA and Residential Soil

PbW_{cua} (µg/L)	**Pb Uptake** (µg/d)			
	Dermal Contact From Swimming[a]	**Incidental Ingestion While Swimming[a]**	**No Swimming, PbS_{res} = 200**	
			PbS_{cua} = 400	**PbS_{cua} = 100**
4	0.00003[b]	0.016[c]	8.5 - 13.2[d]	10.0 - 15.8[d]
40	0.0003	0.16	8.5 - 13.2	10.0 - 15.8
400	0.003	1.6	8.5 - 13.2	10.0 - 15.8
4,000	0.03	16	8.5 - 13.2	10.0 - 15.8

Notes:
[a]Swimming at the CUA is assumed to occur for 1hr/day, 2 days/week.
[b]Assuming a Kp of 4E-6 (an experimental value for lead acetate).
[c]An uptake/intake ratio of 0.46 was used to calculate uptake from intake. This is the midpoint of the range of uptake/intake ratios (0.44 - 0.48) for 1- to 7-year-old children in the scenarios excluding swimming at the CUA with PbS_{res} = 200 mg/kg and PbS_{cua} = 400 or 1,000 mg/kg.
[d]The range reflects age-specific differences in 1- to 7-year-olds.

CUA - common use area
Kp - permeability coefficient
Pb - lead
PbS_{cua} - lead concentrations in common use area soil
PbS_{res} - lead concentration in residential soil
PbW - lead concentration in water

Table 3-9
Comparison of Estimated Lead Uptake Resulting From Dermal Contact and Ingestion of CUA Soil and Residential Soil (PbS_{res} = 200 mg/kg)[a]

PbS_{cua} (mg/kg)	Pb Uptake (µg/d)			
	Dermal Contact		Ingestion	
	Beach CUA[b]	Upland CUA[c]	CUA Soil	Total
400	1.5	0.5	1.2 - 1.9[d]	8.5 - 13.2[d]
1,000	3.7	1.3	2.9 - 4.7	10.0 - 15.8
1,600	5.9	2.0	4.5 - 7.4	11.5 - 18.3
2,000	7.4	2.5	5.5 - 9.2	12.5 - 19.9

Notes:
[a]A dermal absorption fraction of 0.01 was used for lead from soil. Exposure to CUA soil was assumed to occur twice per week.
[b]Children assumed to wear only a bathing suit (SA = 6,500 cm^2).
[c]Children assumed to wear shorts and short-sleeved shirts and to go barefoot (SA = 2,200 cm^2).
[d]The range reflects age-specific differences in 1–7 year olds.

CUA - common use area
PbS - lead concentrations in residential soil
PbS_{cua} - lead concentrations in common use area soil
SA - surface area

5.0 SCREENING OF COMMON USE AREAS FOR LEAD

5.1 SCREENING METHODOLOGY

Exposure pathways for children at beaches and shoreline parks include dermal contact and ingestion of upland soil, beach sediment, surface water, and suspended sediment. The dermal pathway can not be estimated using the IEUBK model and there is no other basis for estimating its contribution to lead uptake and risks in the above exposure scenarios. Therefore, beaches were screened against the following three criteria, assuming that the major sources of lead uptake would result from the ingestion pathway:

1. Does the central tendency estimate (CTE) of the lead concentration in upland soil exceed risk-based concentrations (RBCs) for the soil ingestion scenario?

2. Does the CTE of the lead concentrations in beach sediment exceed RBCs for the soil ingestion scenario?

3. Does the CTE of the lead concentration in agitated surface water (shoreline water and suspended sediment) exceed RBCs for incidental ingestion of surface water and suspended sediment while swimming?

If the answer to <u>any</u> of the above questions was *"yes"*, the site was classified as *"possible risk to children"*, warranting further evaluation in the baseline human health risk assessment (HHRA). If the answer to <u>all</u> questions was *"no"*, the site was classified as *"sufficiently low risk to children"*, such that further evaluation in the baseline HHRA might not be necessary. This approach is depicted in the decision tree shown in Figure 4-1.

The arithmetic mean concentration was used as the CTE of lead concentrations in upland soil, sediment, and water. The basis for using the arithmetic mean is as follows:

1. Validation studies have shown good agreement between blood lead concentration distributions predicted by the IEUBK model and observed blood lead concentrations at Superfund sites, when the inputs to the model are arithmetic means of the exposure concentrations (Hogan et al. 1998). There is no evidence that equally good agreement can be expected if other CTEs are used in the model.

2. The upper 95 percent confidence limit for the mean (UCL_{95}) is the CTE that is recommended for RME estimates for other chemicals (U.S. EPA 1992b). Use of the UCL_{95} in an RME estimate accounts for variability and uncertainty associated

with the estimate of the mean exposure concentration that may derive from spatial or temporal variability and measurement error. In the IEUBK model, these sources of variability are represented in the blood lead concentration term, the integrated exposure metric, as the geometric standard deviation (GSD) of the blood lead concentration. By selecting the 95th percentile blood lead concentration as the basis for the risk estimate (i.e., P_{10}=5%), variability and uncertainty associated with the estimate of the mean exposure concentration is accounted for in the risk estimate. If the UCL_{95} is used in the model to represent the CTE of environmental concentrations, and the 95th percentile blood lead concentration is used as the basis for the risk estimate, then the resulting risk estimate (or RBC) derived from the IEUBK model can be expected to overestimate actual risk. The UCL_{95} for soil and water/sediment concentrations were used in the latter context in this assessment to derive a highly conservative upper bound estimate of risk. Thus, we can be reasonably certain that there is no significant lead health risk to children where the arithmetic mean exposure concentration does not exceed the RBCs, and we have greater confidence in this conclusion where the UCL_{95} for the exposure concentrations does not exceed the RBC.

The above two arguments for using the arithmetic mean for CTE of the concentration term apply to assessments of residential lead exposure. However, they would be expected to also apply to other exposure scenarios in which variability in the exposure concentration term(s) would be similar to, or at least no greater than that typically observed at a residence. This has been assumed to be the case in this screening assessment, *in lieu* of data to the contrary.

In the development of RBC's for CUAs, lead exposures are modeled by *summing* exposures at the CUA with exposures expected at the residence. The screening process has preceded completion of data collection at the site, therefore, assumptions have been made about central tendencies of residential exposure levels. In the absence of data on the residential soil lead concentration, a range of 200 - 300 ppm was assumed. The lower end of the range, 200 ppm, is the IEUBK model default and is considered a plausible residential soil concentration for an urban setting not impacted by point sources (U.S. EPA 1989b, 1994a,b,c). Gott and Cathrall (1980) collected 8,700 soil samples from the upper basin of the Coeur d'Alene District, which included the Coeur d'Alene River Basin and the source area for sediments found in the lower basin. The 50th and 90th percentile lead concentrations were 43 and 171 ppm, respectively. Thus, the low end of the assumed range for residential soil lead, 200 ppm, represents an upper percentile estimate of expected background concentrations in the region. Hogan et al. (1998), as part of a model validation exercise, analyzed soil lead data from various Superfund sites; the geometric mean (and 95 percent confidence intervals) were as follows: Palmerton PA, 201 ppm (142-284); Madison County IL, 333 ppm (310-358); Galena, KS and Jasper MO (combined), 254 ppm (216-297). Thus, the range of 200-300 ppm is reasonably plausible for a residential setting, and may be

more health protective when applied to lower basin areas that are not impacted by upper basin sediments and, therefore, would be expected to have levels closer to background.

The mass fraction of soil in indoor dust at the residence was assumed to be 0.7, the IEUBK model default; this corresponds to a concentration of soil-derived lead in indoor dust of 0.7 times the assumed soil lead concentration ($PbDust_{res} = 0.7 \times PbS_{res}$). IEUBK model default values were assumed for all other residential exposure pathways.

5.2 RESULTS OF RISK-BASED SCREENING

5.2.1 Upland Soil Ingestion

Table 4-1 shows the results of the screening of upland soil at beaches against RBCs for soil. The soil RBCs corresponding to residential soil lead concentrations of 200 and 300 ppm are 1400 and 500 ppm, respectively. The arithmetic mean lead concentrations at all sites are below the RBCs. Therefore, the probability of children having a blood lead concentration greater than 10 µg/dL as a result of ingesting upland soil, in addition to the assumed residential exposures, can be expected to be less than 5 percent at each site.

Table 4-1 also shows a comparison between the UCL_{95} soil lead concentrations and the soil RBCs. No sites exceed the RBC corresponding to a 200 ppm residential soil lead concentration; two sites, the BLM pump station and Blackwell Island, exceed the RBC corresponding to a residential soil lead concentration of 300 ppm. As noted previously, higher estimates of risk are generally derived from the IEUBK model if the UCL_{95} is used to represent the CTE for soil. Therefore, these results indicate a high confidence that all of the sites, with the possible exception of the BLM pump station and Blackwell Island, pose a *sufficiently low risk* from the soil ingestion pathway.

5.2.2 Beach Sand/Sediment Ingestion

Table 4-2 compares the observed concentrations of lead in sediment (i.e., sand or lake bottom deposits at the shoreline) with the soil RBCs corresponding to residential soil lead concentrations of 200 ppm or 300 ppm (RBCs are 1400 ppm or 500ppm, respectively). The arithmetic mean sediment concentrations at all sites are below the RBCs corresponding to lead at 200 ppm in residential soil; one site, Harrison Beach (north), exceeds the RBC for lead at 300 ppm in residential soil. Therefore, the probability of children having a blood lead concentration greater than 10 µg/dL as a result of ingesting beach sediment, in addition to the assumed residential exposures, can be expected to be less than 5 percent for all sites, with the possible exception of Harrison Beach (north).

A high confidence in the above conclusion is indicated from the comparison between UCL_{95} values for sediment lead and the soil RBCs (Table 4-2). None of the sediments, with the exception of Harrison Beach (north), exceed the RBCs corresponding to a residential soil lead concentration of 200 ppm. The following five sites exceed the RBCs corresponding to a 300 ppm residential soil lead concentration: North Idaho College Beach (Spokane River), Corbin Park, Harrison Beach (north), and Blackwell Island.

5.2.3 Combined Beach Sediment Ingestion and Incidental Ingestion of Water and Suspended Sediment While Swimming

Table 4-3 shows the results of the screening for the combined scenarios of ingestion of beach sediment and incidental ingestion of water and suspended sediment while swimming. The RBCs for water and suspended sediment ($RBC_{w,ss}$) shown in Table 4-3 correspond to the arithmetic mean sediment concentrations at each site and residential soil lead concentrations of either 200 or 300 ppm (columns 6 and 7 of Table 4-3). Sites at which sediment lead concentrations exceed soil RBCs, shown in Table 4-2, will also exceed water/sediment RBCs shown in Table 4-3 because the two pathways, beach sediment ingestion and incidental water/sediment ingestion while swimming, are summed in the development of the water/sediment RBCs. Thus, the mean sediment lead concentration at Harrison Beach (north) exceeds the soil RBC if a residential soil lead concentration of 300 ppm is assumed (Table 4-2). However, when the ingestion pathways for both beach sediment and water/suspended sediment are considered, the water/sediment concentration exceeds the water/sediment RBC at a residential soil concentration of 200 ppm. Two additional sites, Blackwell Island and Corbin Park, exceed the water/sediment RBC if the residential soil lead concentration is assumed to be 300 ppm.

Comparisons of the water/sediment RBCs with the UCL_{95} concentrations provide a method to evaluate the degree of confidence in the results. The UCL_{95} values for water/sediment lead concentrations, and all of the sites, with the exception of Harrison Beach (north), are below the RBCs corresponding to a residential lead soil concentration of 200 ppm. The UCL_{95}s at two other sites, Blackwell Island and Corbin Park, exceed the RBCs corresponding to a 300 ppm residential soil lead concentration.

The RBCs for water and suspended sediment ($RBC_{w,ss}$) shown in Table 4-4 correspond to the UCL_{95} sediment concentrations at each site and residential soil lead concentrations of either 200 ppm or 300 ppm (columns 6 and 7 of Table 4-3). As expected, all sites for which UCL_{95} sediment concentrations exceed sediment RBCs (Table 4-2), also exceed water/sediment RBCs based on UCL_{95} sediment concentrations in Table 4-4. These include North Idaho College Beach (Spokane River), Corbin Park, Harrison Beach (north), and Blackwell Island. One other site, Coeur d'Alene Beach (City Park), exceeded water/sediment RBCs based on the UCL_{95} sediment lead concentration.

5.2.4 Conclusions from Risk-based Screening

The risk-based screening of beaches identified four categories of sites:

> *Category 1*: If the residential soil lead concentration (central tendency) is assumed to be 200 ppm or higher, *Harrison Beach* (north) exceeds risk-based criteria for lead.
>
> *Category 2*: If the residential soil lead concentration (central tendency) is assumed to be 300 ppm or higher, *Harrison Beach (north), Blackwell Island* and *Corbin Park* exceed the risk-based criteria for lead.
>
> *Category 3*: If the UCL_{95}, rather than the mean, is used to represent the central tendency of beach exposure concentrations, three additional sites may exceed risk-based criteria: *North Idaho College Beach (Spokane River), and Coeur d'Alene Beach (City Park);* in addition to *Harrison Beach (north) and Blackwell Island*.
>
> *Category 4:* The following sites do not exceed risk-based lead criteria regardless of which estimate of the central tendency is used in the screening procedure: *Post Falls City Beach, Green Ferry Bay park Beach, Black Bay, N. Idaho College Beach (along Lake), Tubbs Hill (sites 1,2,3), Higgans Point (sites 1, 2), Cougar Bay, Bell Bay, Mica Bay, Rockford Bay, Loffs Bay, Windy Bay, Spokane Point (on reservation) and Fuller Landing.*

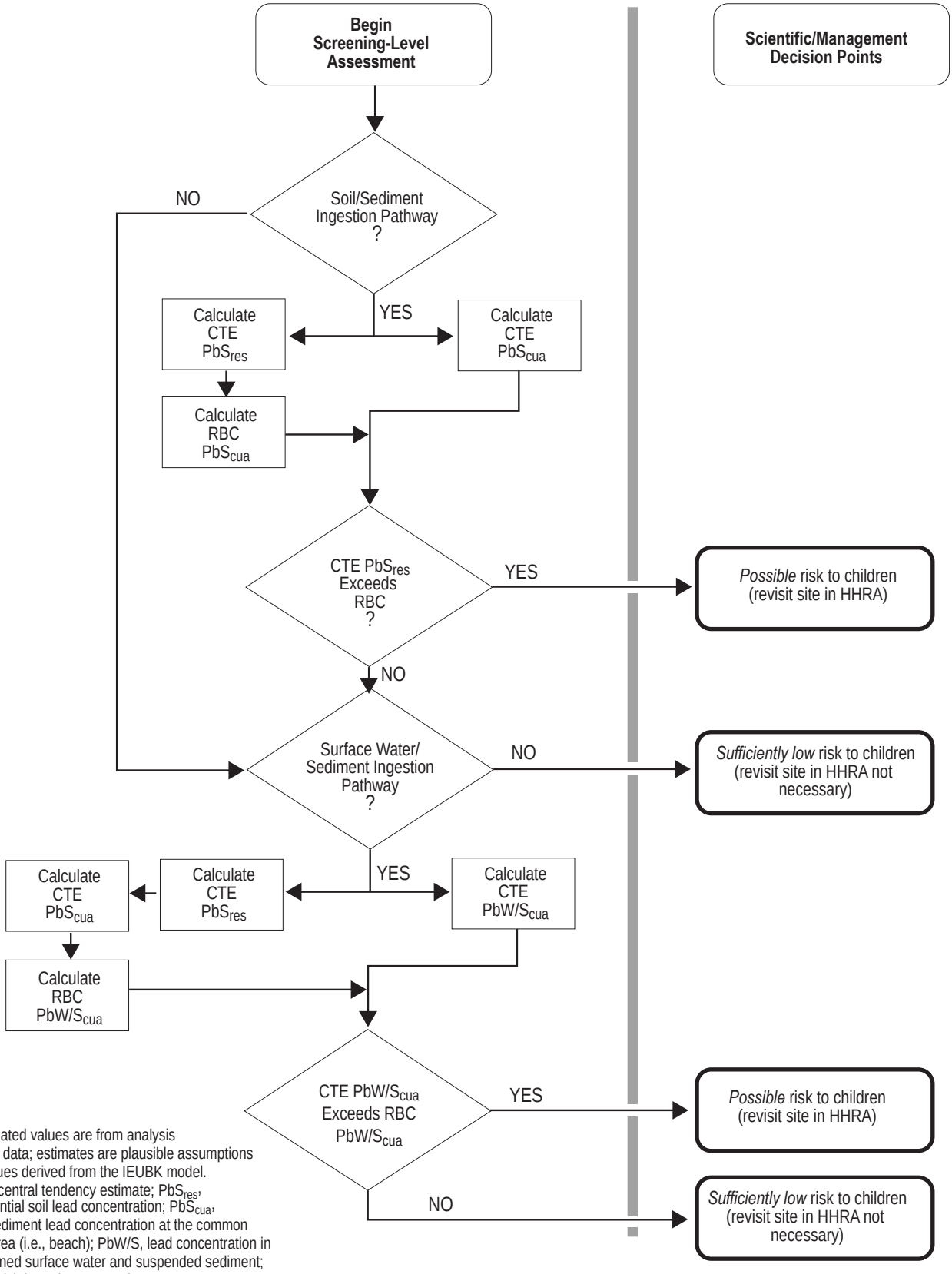
Use of the UCL_{95} as the CTE for lead exposure concentrations at the sites derives a high confidence in category 4. As noted previously, when exposure concentrations are represented in the IEUBK model with theUCL_{95}, overestimates of actual risk can be expected. Thus, we can be highly certain that the sites in category 4 do not pose a significant lead health risk if the residential soil lead concentrations are no more than 300 ppm and all other assumptions about the residential exposures are accurate.

For the same reason, we would not expect the sites in category 3 to pose a significant lead risk. The sediment lead concentrations at the category 3 sites, do not exceed 400 ppm; this concentration has been used as a residential screening level at other sites in the Superfund program (U.S. EPA 1994b). Typical residential exposures to 400 ppm would not be associated with significant health risks.

Sites in categories 1 and 2, Harrison Beach (north), Blackwell Island and Corbin Park, exceed the risk-based criteria when plausible assumptions are made about central tendency residential exposures and the mean is used to represent the central tendency of exposure concentrations at the beaches. Lead exposures at Harrison Beach (north) may pose a significant health risk when combined with residential exposures. Blackwell Island and Corbin Park are not considered sites of concern for the following two reasons:

1. The sites do not exceed risk-based screening concentrations when the residential exposure is assumed to be 200 ppm; and

2. The mean sediment lead concentrations do not significantly exceed 400 ppm, which is the EPA residential screening level (U.S. EPA 1994d) that has been used in the Superfund program (note: the arithmetic mean sediment lead concentration at Corbin Park was 412 ppm, which is only slightly above 400 ppm).



Note:
Calculated values are from analysis of site data; estimates are plausible assumptions or values derived from the IEUBK model.
CTE, central tendency estimate; PbS$_{res}$, residential soil lead concentration; PbS$_{cua}$, soil/sediment lead concentration at the common use area (i.e., beach); PbW/S, lead concentration in combined surface water and suspended sediment; RBC, risk-based concentration.

EPA REGION 10

Figure 4-1
Decision Tree for Risk-Based Screening of Lower Basin Shoreline Parks and Beaches

54-50-0C2Q
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Table 4-1
Risk-based Concentrations (RBCs) for Upland Soil at Beaches

Site Location	Site ID	Site Name	RBC (ppm)		AM_{soil} (ppm)	AM_{soil} Exceeds RBC?		UCL_{95} (ppm)	UCL_{95} Exceeds RBC?	
			PbS_{res} (ppm)			PbS_{res} (ppm)			PbS_{res} (ppm)	
			200	300		200	300		200	300
Spokane River (7 Sites)	1	N. Idaho College Beach (Spokane River)	1400	500	204	NO	NO	294	NO	NO
	3	Post Falls City Beach/River Park	1400	500	27.6	NO	NO	40.1	NO	NO
	5	Green Ferry Bay County Park	1400	500	78.5	NO	NO	261	NO	NO
	6	Black Bay	1400	500	55.5	NO	NO	60.1	NO	NO
	7	BLM Pump Station	1400	500	155	NO	NO	641	NO	YES
	8	Corbin Park	1400	500	NA	nd	nd	NA	nd	nd
	21	Blackwell Island	1400	500	356	NO	NO	835	NO	YES
Coeur d'Alene Lake (17 Sites)	2	N. Idaho College Beach (CdA Lake)	1400	500	NA	nd	nd	NA	nd	nd
	9	Coeur d'Alene Beach at City Park	1400	500	125	NO	NO	188	NO	NO
	10	Tubbs Hill (site 1)	1400	500	NA	nd	nd	NA	nd	nd
	11	Tubbs Hill (site 2)	1400	500	NA	nd	nd	NA	nd	nd
	12	Tubbs Hill (site 3)	1400	500	NA	nd	nd	NA	nd	nd
	15	Higgan's Point (site 1)	1400	500	NA	nd	nd	NA	nd	nd
	16	Higgan's Point (site 2)	1400	500	NA	nd	nd	NA	nd	nd
	17	Harrison Beach (site 1, West)	1400	500	38.4	NO	NO	147	NO	NO
	18	Harrison Beach (site 2, North)	1400	500	NA	nd	nd	NA	nd	nd
	19	Cougar Bay	1400	500	98.5	NO	NO	187	NO	NO
	23	Bell Bay	1400	500	197	NO	NO	443	NO	NO
	24	Mica Bay	1400	500	44.8	NO	NO	77.8	NO	NO
	25	Rockford Bay	1400	500	67.3	NO	NO	103	NO	NO
	26	Loffs Bay	1400	500	102	NO	NO	144	NO	NO
	27	Windy Bay	1400	500	18.1	NO	NO	21.8	NO	NO
	29	Spokane Point (on reservation)	1400	500	NA	nd	nd	NA	nd	nd
	30	Fuller Landing	1400	500	31.7	NO	NO	NA	nd	nd

Notes:
RBCs are for combined soil ingestion at the beach and residential lead exposure. RBCs assume: 1) ingestion of upland soil at the arithmetic mean (AM) or upper 95 percent confidence limit (UCL_{95}) lead concentration (ppm); and 2) residential exposures to soil (PbS_{res}) at 200 or 300 ppm. NA, data not available; nd, not determined.

CdA - Coeur d'Alene

Table 4-2
Risk-Based Concentrations (RBCs) for Shoreline Sediment at Beaches

Site Location	Site ID	Site Name	RBC (ppm)		AM_{sed} (ppm)	AM_{sed} Exceeds RBC?		UCL_{95sed} (ppm)	UCL_{95} Exceeds RBC?	
			PbS_{res} (ppm)			PbS_{res} (ppm)			PbS_{res} (ppm)	
			200	300		200	300		200	300
Spokane River (7 Sites)	1	N. Idaho College Beach (Spokane River)	1400	500	323	NO	NO	687	NO	YES
	3	Post Falls City Beach/River Park	1400	500	85.2	NO	NO	134	NO	NO
	5	Green Ferry Bay County Park	1400	500	101	NO	NO	140.2	NO	NO
	6	Black Bay	1400	500	105	NO	NO	165	NO	NO
	7	BLM Pump Station	1400	500	112	NO	NO	178	NO	NO
	8	Corbin Park	1400	500	412	NO	NO	562	NO	YES
	21	Blackwell Island	1400	500	397	NO	NO	603	NO	YES
Coeur d'Alene Lake (17 Sites)	2	N. Idaho College Beach (CdA Lake)	1400	500	146	NO	NO	183	NO	NO
	9	Coeur d'Alene Beach at City Park	1400	500	128	NO	NO	174	NO	NO
	10	Tubbs Hill (site 1)	1400	500	49.5	NO	NO	69.6	NO	NO
	11	Tubbs Hill (site 2)	1400	500	96.3	NO	NO	118	NO	NO
	12	Tubbs Hill (site 3)	1400	500	44.1	NO	NO	52	NO	NO
	15	Higgan's Point (site 1)	1400	500	63.8	NO	NO	83.7	NO	NO
	16	Higgan's Point (site 2)	1400	500	90.4	NO	NO	139	NO	NO
	17	Harrison Beach (site 1, West)	1400	500	NA	nd	nd	NA	nd	nd
	18	Harrison Beach (site 2, North)	1400	500	1250	NO	YES	3730	YES	YES
	19	Cougar Bay	1400	500	90.7	NO	NO	180	NO	NO
	23	Bell Bay	1400	500	96.3	NO	NO	NA	nd	nd
	24	Mica Bay	1400	500	30.7	NO	NO	34.6	NO	NO
	25	Rockford Bay	1400	500	NA	nd	nd	NA	nd	nd
	26	Loffs Bay	1400	500	52.9	NO	NO	62.9	NO	NO
	27	Windy Bay	1400	500	23	NO	NO	NA	nd	nd
	29	Spokane Point (on reservation)	1400	500	126	NO	NO	293	NO	NO
	30	Fuller Landing	1400	500	NA	nd	nd	NA	nd	nd

Notes:
RBCs are for combined sediment ingestion at the beach and residential lead exposure. RBCs assume: 1) ingestion of beach sediment at the arithmetic mean (AM) or upper 95 percent confidence limit (UCL_{95}) lead concentration (ppm); and 2) residential exposures to soil (PbS_{res}) at 200 or 300 ppm. NA, data not available; nd, not determined.

Table 4-3
Risk-Based Concentrations (RBCs) for Water and Suspended Sediment at Beaches (Mean Lead Concentrations)

Site Location	Site ID	Site Name	AM_{sed} (ppm)	$RBC_{w,ss}$ (µg/L) PbS_{res} (ppm)		$AM_{w,ss}$ (µg/L)	AM Exceeds RBC? PbSres (ppm)		$UCL_{95w,ss}$ (µg/L)	UCL_{95} Exceeds RBC? PbS_{res} (ppm)	
				200	300		200	300		200	300
Spokane	1	N. Idaho College Beach (Spokane River)	323	1838	246	131	NO	NO	192	NO	NO
River	3	Post Falls City Beach	85.2	2256	664	92.7	NO	NO	154	NO	NO
(7 Sites)	5	Green Ferry Bay park	101	2228	636	11.7	NO	NO	18.6	NO	NO
	6	Black Bay	105	2221	629	54.7	NO	NO	80	NO	NO
	7	BLM Pump Station	112	2209	617	59.2	NO	NO	110	NO	NO
	8	Corbin Park	412	1682	90	117	NO	YES	219	NO	YES
	21	Blackwell Island	397	1708	116	417	NO	YES	651	NO	YES
Coeur	2	N. Idaho College Beach (CdA Lake)	146	2149	557	42.2	NO	NO	69.3	NO	NO
d'Alene	9	Coeur d'Alene Beach at City Park	128	2181	589	30.6	NO	NO	42.1	NO	NO
Lake	10	Tubbs Hill (site 1)	49.5	2319	727	9.93	NO	NO	13.7	NO	NO
(17 Sites)	11	Tubbs Hill (site 2)	96.3	2237	645	3.84	NO	NO	7.7	NO	NO
	12	Tubbs Hill (site 3)	44.1	2328	736	8.26	NO	NO	12.9	NO	NO
	15	Higgans Point (site 1)	63.8	2294	702	11.4	NO	NO	21.6	NO	NO
	16	Higgans Point (site 2)	90.4	2247	655	26.6	NO	NO	37.1	NO	NO
	17	Harrison Beach (site 1, West)	NA	nd	nd	56.2	nd	nd	102	nd	nd
	18	Harrison Beach (site 2, North)	1250	210	0	267	YES	YES	355	YES	YES
	19	Cougar Bay	90.7	2246	654	91	NO	NO	171	NO	NO
	23	Bell Bay	96.3	2237	645	29.4	NO	NO	40.6	NO	NO
	24	Mica Bay	30.7	2352	760	21.3	NO	NO	28.4	NO	NO
	25	Rockford Bay	NA	nd	nd	NA	nd	nd	NA	nd	nd
	26	Loffs Bay	52.9	2313	721	44.3	NO	NO	56.5	NO	NO
	27	Windy Bay	23	2365	773	4.18	NO	NO	6.81	NO	NO
	29	Spokane Point (on reservation)	126	2184	592	159	NO	NO	241	NO	NO
	30	Fuller Landing	NA	nd	nd	4	nd	nd	7.5	nd	nd

Notes:
RBCs are for combined beach sediment ingestion and incidental water/sediment ingestion at the beach while swimming and residential lead exposure. RBCs assume: 1) ingestion of beach sediment at the arithmetic mean (AM) concentration (ppm); ingestion of water and suspended sediment (w,ss) at the arithmetic mean ($AM_{w,ss}$) or upper 95 percent confidence limit ($UCL_{95w.ss}$) lead concentration (ppm) and 3) residential exposures to soil (PbS_{res})at 200 or 300 ppm.

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Table 4-4
Risk-based Concentrations (RBCs, µg/L) for Water and Suspended Sediment at Beaches (UCL_{95} Lead Concentrations)

Site Location	Site ID	Site Name	UCL_{sed} (ppm)	$RBC_{w,ss}$ PbS_{res} (ppm)		$AM_{w,ss}$ (µg/L)	AM Exceeds RBC? bS_{res} (ppm)		$UCL_{95w,ss}$ (µg/L)	UCL_{95} Exceeds RBC? PbS_{res} (ppm)	
				200	300		200	300		200	300
Spokane River (7 Sites)	1	N. Idaho College Beach (Spokane River)	687	1199	0	131	NO	YES	192	NO	YES
	3	Post Falls City Beach	134	2170	578	92.7	NO	NO	154	NO	NO
	5	Green Ferry Bay park	140.2	2159	567	11.7	NO	NO	18.6	NO	NO
	6	Black Bay	165	2116	524	54.7	NO	NO	80	NO	NO
	7	BLM Pump Station	178	2093	501	59.2	NO	NO	110	NO	NO
	8	Corbin Park	562	1419	0	117	NO	YES	219	NO	YES
	21	Blackwell Island	603	1347	0	417	NO	YES	651	NO	YES
Coeur d'Alene Lake (17 Sites)	2	N. Idaho College Beach (CdA Lake)	183	2084	492	42.2	NO	NO	69.3	NO	NO
	9	Coeur d Alene Beach at City Park	174	2100	0	30.6	NO	YES	42.1	NO	YES
	10	Tubbs Hill (site 1)	69.6	2283	691	9.93	NO	NO	13.7	NO	NO
	11	Tubbs Hill (site 2)	118	2198	606	3.84	NO	NO	7.7	NO	NO
	12	Tubbs Hill (site 3)	52	2314	722	8.26	NO	NO	12.9	NO	NO
	15	Higgans Point (site1)	83.7	2259	667	11.4	NO	NO	21.6	NO	NO
	16	Higgans Point (site2)	139	2162	569	26.6	NO	NO	37.1	NO	NO
	17	Harrison Beach (site 1, West)	NA	nd	nd	56.2	nd	nd	102	nd	nd
	18	Harrison Beach (site 2, North)	3730	0	0	267	YES	YES	355	YES	YES
	19	Cougar Bay	180	2089	497	91	NO	NO	171	NO	NO
	23	Bell Bay	NA	nd	nd	29.4	nd	nd	40.6	nd	nd
	24	Mica Bay	34.6	2345	753	21.3	NO	NO	28.4	NO	NO
	25	Rockford Bay	NA	nd	nd	NA	nd	nd	NA	nd	nd
	26	Loffs Bay	62.9	2295	703	44.3	NO	NO	56.5	NO	NO
	27	Windy Bay	NA	nd	nd	4.18	nd	nd	6.81	nd	nd
	29	Spokane Point (on reservation)	293	1891	299	159	NO	NO	241	NO	NO
	30	Fuller Landing	NA	nd	nd	4	nd	nd	7.5	nd	nd

Notes:
RBCs are for combined beach sediment ingestion and incidental water/sediment ingestion at the beach while swimming and residential lead exposure. RBCs assume: 1) ingestion of beach sediment at the upper 95 percent confidence limit (UCL_{95}) concentration (ppm); ingestion of water and suspended sediment (w,ss) at the arithmetic mean ($AM_{w,ss}$) or upper 95 percent confidence limit ($UCL_{95w.ss}$) lead concentration (ppm) and 3) residential exposures to soil at 200 or 300 ppm.

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6.0 DEVELOPMENT OF RBCs FOR CHEMICALS OTHER THAN LEAD

The purpose of establishing a risk-based screening concentration (RBC) is to provide a soil or water action level below which there is a high degree of confidence that a health threat does not exist. In order to develop an RBC, the amount of exposure to a given chemical must be assessed, an estimate of the toxicity of each chemical must be available, and target health risk goals must be established. Each of these three categories—exposure, toxicity, and risk—are quantified and used in standard risk equations to calculate a chemical-specific concentration in soil or water. The result of this process is to arrive at a protective soil or water concentration (RBC) based on potential multiple routes of exposure and a target health goal.

6.1 EXPOSURE ASSESSMENT

The exposure assessment evaluates sources, pathways, receptors, duration and frequency, and routes of exposure to assess total human exposure to the substances of concern in the common use areas (CUAs). This process identifies the human populations potentially exposed to chemicals in the CUAs, the means by which exposure occurs, and the amount of chemical taken into the body (intake) from each exposure medium. Exposure is assessed using the following steps:

- Exposed populations are characterized
- Exposure pathways are identified
- Exposure is quantitatively assessed

The result of this process is a calculated daily intake per body weight for each medium of concern. The daily intake rate per body weight (summary intake factor) is combined with chemical-specific toxicity criteria (Table 5-4) and target health risk goals (Section 5.3) to calculate a health-protective RBC.

To develop RBCs, exposure for target populations is calculated under "reasonable maximum" (upper-bound) exposure conditions. Reasonable maximum exposure (RME) incorporates a number of conservative assumptions in estimating chemical intake rates and characteristics of the receptor population. RME is thus an estimate of the highest exposure that is reasonably expected to occur at the site and may overestimate actual exposure for the majority of the population. The intent of the guidance is for the combined exposure and toxicity variables to result in an estimate of RME, even though some intake variables may not be at their individual maximum value (U.S. EPA 1989a, 1991a). As stated by U.S. EPA (1991a), "the goal of RME is to combine upper-bound and mid-range exposure factors . . . so that the result represents an exposure scenario that is both protective and reasonable; not the worst possible case."

RME conditions are selected to evaluate exposures at the CUAs. If a site is "screened out" by the RBCs developed here, then it is unlikely to represent a health risk.

6.1.1 Characterization of Exposed Populations

This screening level risk assessment focuses on the portion of the population that receives the most exposure to site chemicals or is more sensitive to the toxic effects of chemicals. Because the CUAs evaluated in this report are not individual residences or work places, the population of concern is considered to be recreational and composed of both adults and children. As described in Section 1.4, recreational populations were subdivided into users of beaches and upland areas (e.g., parks). The most-exposed or most-sensitive group is considered to be children. Young children tend to have greater exposures to soil because of their hand-to-mouth behavior and greater inherent susceptibility to toxic effects of chemicals. Factors contributing to this susceptibility are:

- More efficient absorption of many substances from the gastrointestinal tract than adults
- Higher intake levels of soil

Consequently, the RBCs developed consider the young children at the parks and beaches to be the exposed population of concern.

6.1.2 Exposure Scenarios

Several possible pathways of exposure exist in the CUAs. An exposure pathway is the mechanism by which a receptor (person) is exposed to chemicals from a source. Four elements comprise a complete exposure pathway:

- A source of chemical release
- A retention or transport medium (for example, soil or water)
- A point of potential human contact with the medium
- A means of entry into the body (e.g., ingestion) at the contact point.

Only complete pathways containing all four elements result in exposures. Potential pathways at the site that were selected for completeness include (see also Section 1.3 and Figure 1-2):

- Incidental ingestion of chemicals in soil
- Contact with soil and absorption of chemicals through the skin

- Inhalation of chemicals adsorbed to dust
- Incidental ingestion of chemicals in water while swimming
- Skin contact with chemicals in water and absorption through the skin while swimming
- Ingestion of drinking water (two locations only)

The potential receptors (children visiting the CUAs) and the routes of exposure from these media are presented in the conceptual site model (Figure 1-2). Such a model describes the sources of chemicals at a site, their release and transfer through environmental media (e.g., soil and air), and the points and means by which receptors might contact the chemicals.

Pathways included in the quantitative development of risk-based screening concentration calculations are discussed below, along with the rationale for eliminating the pathway considered a relatively insignificant source of risk (inhalation of airborne dust), and the pathway (drinking water) evaluated by maximum contaminant levels (MCLs) established by regulation for drinking water. In this expedited screening level assessment, fish ingestion is not a pathway of concern based on an ATSDR report (1998) where it was concluded that adverse health effects were unlikely from eating fish caught in Coeur d'Alene Lake, provided that excessive lead exposures from other sources were not occurring.

6.1.2.1 Ingestion of Soil

Soil ingestion is considered a complete pathway and is evaluated quantitatively in the RBC calculations. Incidental ingestion of soil is considered the primary route of exposure for metals in recreational settings. Young children are more likely to ingest soil during outdoor play than adults because of their more frequent hand-to-mouth actions and tendency to play in the dirt. Adults typically ingest less soil than children but may also ingest small amounts of soil during outdoor activities. Because of their lesser ingestion rate, adults will be protected at an RBC calculated as protective of children.

6.1.2.2 Dermal Contact With Soil

Dermal contact with soil is considered a complete pathway and quantitatively evaluated in the RBC calculations; however, the dermal exposure pathway is not as well characterized as ingestion. Therefore, the relationship between dermal exposure and actual exposure dose is uncertain and EPA toxicity criteria have been derived only for the oral and inhalation routes. Although uncertain, dermal route exposures have been quantitatively evaluated in this study because absorption of contaminants from soil or water are potentially significant routes of

exposure relative to ingestion of soil and dust (Johnson and Kissel 1996). U.S. EPA recommends the use of oral toxicity criteria for the dermal pathway, with a correction factor to correct the oral toxicity criteria to an internal absorbed dose, and an absorption factor for the amount of chemicals which cross the skin and enter the blood stream (U.S. EPA 1992a). The importance of dermal relative to ingestion exposures depends on the chemical-specific absorption fraction, chemical-specific permeability coefficient, and relative bioavailability factors associated with the dermal and ingestion routes. For arsenic, dermal exposure from soil amounts to 18 percent of exposures attributable to the ingestion pathway (cancer endpoint). Dermal route exposures were omitted in previous studies because data needed to measure dermal exposures have only recently been developed. Therefore, this pathway is quantitatively included in order not to underestimate health risks.

6.1.2.3 Incidental Ingestion of Water While Swimming

Swimming in Coeur d'Alene Lake is common during the summer. Some water is typically swallowed while swimming and may be ingested during other water activities such as water skiing and canoeing. Consequently, the incidental ingestion of water while swimming is quantitatively evaluated in the RBC calculations for water exposure.

6.1.2.4 Dermal Contact With Water While Swimming

The previous discussion for dermal contact with soil also applies to the swimming pathway.

6.1.2.5 Inhalation of Airborne Dust

Exposure to chemicals of concern (COCs) may result from inhalation of resuspended dust. Inhalation exposure to nonvolatile compounds is typically minor in resuspended dust when compared to direct ingestion exposure (U.S. EPA 1986, Glass and SAIC 1992) and is unlikely to significantly lower RBC values based on ingestion and dermal exposure. Consequently, recreational users of the areas were not evaluated quantitatively for inhalation, because they receive most site chemicals through ingestion.

In addition, each CUA is relatively small (generally less than one acre); therefore, wind blowing across the sites and inhaled by site visitors is unlikely to entrain much dust. A large amount of air monitoring data has been collected in the vicinity of Kellogg and Smelterville, Idaho, and air concentrations of COCs have not been found to exceed air quality standards. The presence of vegetation at many sites also limits fugitive dust emissions.

6.1.2.6 Ingestion of Drinking Water

Two of the 24 CUA sites evaluated in this screening level risk assessment have a potable water source (Harrison Beach and Windy Bay). Samples were collected from tap water or drinking fountains at each site and analyzed for metals. However, because of the small number of sites compared to those without drinking water sources and the relatively small exposures, site-specific RBCs were not developed for this pathway. Chemicals in water will be compared to their MCL concentrations (presented in Section 6.0) for screening purposes.

6.1.3 Quantitative Assessment of Exposure

This section quantifies the magnitude, frequency, and duration of exposure to chemicals in soil and surface water. Recreational intakes of chemicals are quantified for soil and water ingestion, and dermal absorption of chemicals from soil and water. Calculation of intakes involves estimating the amount of media (soil, water) containing chemicals that an individual might eat or contact dermally.

Intake rates of water and soil are combined with frequencies of exposure, and fraction of absorption to calculate a summary intake factor. Depending on the pathway, intake rates are based on average lifetime parameters, such as a 70-kg body weight, or are broken down separately for younger and older age groups. The breakdown is performed for pathways such as soil ingestion, for which children would have a much higher dose per body weight because of their behavior. For these pathways, intake rates are based on young children from birth to age 7 weighing on average 15 kg, and on ages 7 to 30 weighing on average 70 kg (U.S. EPA 1991a). For all the RBC values, only child exposures are considered because the child-only assumption produces the lowest RBC concentrations (i.e., most health protective). Because intake exposures for carcinogens are doses averaged over a lifetime, relatively short-term child exposure to carcinogens (6 years) take into account the lower dose per body weight for the older age group (see formulas presented at the end of this section).

Calculated intake for each pathway is expressed as the amount of media (e.g., water, soil) taken into the body per unit concentration of chemical in soil. Table 5-1 summarizes exposure factors.

6.1.3.1 Soil Intake Rates

The rate of soil ingestion is based on the amount of soil and dust a child or adult inadvertently swallows in a given day from all sources, both indoors and outdoors. Preschool-age children would have the highest intake rates because of their hand-to-mouth behavior and tendency to play in dirt or on the floor. Accordingly, most studies have concentrated on these younger age groups for measurement of soil ingestion rates.

The most accurate estimates of soil ingestion rates in children are from studies measuring certain tracer elements in soil and in feces. These tracer elements have a low content in the diet and low

gastrointestinal absorption, characteristics that make them good indicators in feces of the amount of soil that was ingested. An important distinction is that tracer studies measure all sources of tracers that were ingested including outdoor soil, house dust indoors, airborne dust that is trapped in the upper respiratory tract and swallowed, food, medicines, vitamins, paint chips, baby powder, and toothpaste. The most reliable studies (e.g., Calabrese et al. 1989, U.S. EPA 1997a) have attempted to correct for the contribution of tracers from the diet and from medicines. Any unaccounted sources of tracers would tend to inflate soil ingestion estimates, although these sources are assumed to be negligible.

For residential exposure, the U.S. EPA (1991a) has recommended RME soil ingestion rates of 200 mg/day for young children (ages 0 to 6, with an average weight of 15 kg) and 100 mg/day for older age groups (with an average weight of 70 kg). These values are stated to represent upper-bound estimates of average values for soil and dust ingestion over a chronic period of exposure (U.S. EPA 1991a) based on EPA's review of recent soil ingestion studies (Calabrese et al. 1989 and 1990, Davis et al. 1990, van Wijnen et al. 1990).

At the beach, children are assumed to potentially ingest greater amounts of soil than they would at home; consequently, the soil ingestion rate selected for the RBC calculations is 300 mg/day, rather than 200 mg/day. The value of 300 mg/day is the upper-bound (90th percentile) intake from a soil and feces tracer study by van Wijnen et al. (1990, as cited in U.S. EPA 1997a) where ingestion rates were measured in 78 children while they were at campgrounds adjacent to a lake.

6.1.3.2 Dermal Contact Rates

The amount of a chemical that is absorbed into the body through the dermal route from soil depends on three factors:

1. The amount of chemical absorption through the skin
2. The amount of soil adhering to the skin
3. The surface area of skin in contact with soil

Factors one and three also apply to absorption into the body from water. The first factor is discussed under "absorption" placed later in this section. The exposure parameters selected for the latter two items are described below.

<u>Soil to Skin Adherence Factors</u>: Quantitative estimates of dermal absorption of chemicals from soil assume that all of the soil adhered to the skin is in contact with the skin. If a thick layer of soil adheres to the skin, then only the layer that is in contact with the skin would transfer chemicals into the skin. Soil particles that are on top of other soil particles have a reduced potential to transfer chemicals through the skin. There is evidence that soil does not adhere to skin in a uniform pattern (Kissel et al. 1998) indicating that assumptions of uniform coverage are not often met and might result in an overestimate of absorption.

The adherence factor (AF) is a measure of the mass of soil in contact with a unit area of skin (mg soil per cm^2 skin). The AF is a quantitative measure of how dirty a person gets. Risks associated with dermal exposure to contaminated soil are not well-characterized, but nevertheless must be estimated to define endpoints for remedial strategies (Holmes et al. 1998). The AF is dependent upon environmental conditions, including soil type, particle size, moisture content, and receptor behavior (Kissel et al. 1996a,b). The AFs are based on studies conducted by Kissel et al. (1996a,b) and Holmes et al. (1998). The child AF is based on experiments in which soil loading was measured following playing in raised beds filled with moist, bare soil. The adult AF is based on measurements following unstaged gardening activities.

Skin Surface Area: Surface area (SA) is a measure of the area of skin potentially exposed to a contaminated medium. The SA used depends on the exposure scenario and activity evaluated. SAs and AFs are summarized in Table 5-1. For the swimming and beach scenarios, the skin surface area is 6,500 cm^2 and assumes the child will be wearing a bathing suit. The skin area values represent the 50th percentiles for ages 2–7 years (U.S. EPA 1997a). Adult values for skin area are needed in the child cancer RBC calculations to account for the lifetime exposure assumed for cancer. For the soil dermal route for adults, the skin surface area used in the equations is 4,800 cm^2; it assumes the face, hands, forearms, and lower legs could come into contact with soil.

6.1.3.3 Water Intake Rates

The incidental ingestion rate for water while swimming is 30 mL/hour derived from estimates about the amount of water in a mouthful and the amount of time people might be in contact with recreational water (U.S. EPA 1998b). The 30 mL/hour value is the basis of the 10 mL/day proposed in the Draft Water Quality Criteria Methodology Revisions published in the August 14, 1998 Federal Register and is also being proposed for use in the Water Quality Guidance for the Great Lakes (58 FR 29869).

6.1.3.4 Exposure Frequency

At the Sites: To adjust for the amount of time that people would be exposed to chemicals in soil or water, exposure is multiplied by a correction factor for different site uses, exposure scenarios, and pathways. Exposure for recreational uses of the site may vary widely depending not only on frequency of visits to the site but also on the type of activity. The frequency of twice per week for four months (32 days) is professional judgment and takes into consideration the climate of the CDARB. The assumption is that an entire day twice per week would be spent at a particular CUA when it was visited during the warmer months (if sites are visited during rain events or while snow is on the ground, no significant soil exposure would occur because of either increased clothing and decreased soil contact during the rainy season; and soil covered by snow). Although the assumption of an entire day (10+ hours) is conservative when compared to the studies

described below, the assumption would account for the wide variation in visitation patterns and cover the campgrounds where a stay of 14 consecutive days is possible.

Two other sources of information on potential length of time at CUAs were consulted, the *Risk Assessment Protocol* Document (Jacobs Engineering et al. 1989) developed for the 21-square-mile area commonly referred to as the Bunker Hill Superfund site, and U.S. EPA's *Exposure Factors Handbook* (1997a). The *Protocol* document divided the year into three periods: winter (18 weeks), spring and fall (17 weeks), and summer (17 weeks). They estimated time spent outdoors and not at home for five different age groups for each period. For children, time periods for age 2–6 years were one hour daily for spring/fall and 2 hours daily during summer (approximately equivalent to 15 days per year). Adults were assumed by the *Protocol* document to have no significant contact with non-yard soil in the winter, spring, and fall.

U.S. EPA (1997a) collected information on the amount of time spent outdoors and not at home for various activities from a comprehensive survey on human activity patterns in the United States. The survey gathered data from over 9,000 people who kept 24-hour diaries (Tsang and Klepsis 1996 as cited in U.S. EPA 1997a). Participants were selected randomly through the telephone book; the study had an overall response rate of 63 percent. The survey indicated that for most outdoor recreation activities, time spent outdoors ranges from 1 to 3 hours per visit for the 50th percentile and 4 to 10.5 hours for the 95th percentile (U.S. EPA 1997a). Recommended Outdoor Activity Factors from U.S. EPA (1997a) are:

- Children (boys and girls aged 3–11 years): 5 hours per day (weekday) and 7 hours/day (weekend)

- Adults ($\geq$ 12 years): 1.5 hours/day.

Assuming twice weekly visits of 7 hours each (the *Handbook's* child weekend time), the total is approximately 13 days per year, similar to the assumptions in the protocol document. Therefore, our assumption of 10+ hours and 32 days per year is health-protective because it is unlikely to underestimate time spent at the beaches and parks. Both Jacobs et al. (1989) and Tsang and Klepsis (1996 as cited in U.S. EPA 1997a) assume less time outdoors.

<u>In the Water</u>: While beach visits are assumed to occur twice a week, the entire day would not be spent in the water. The exposure factor selected for swimming is EPA's (1997a) recommended swimming activity factor for length of time in the water of 1 hr/swim event (assumed to occur only once a month from *Handbook*). One hr/swim event is the 50th percentile value and 3 hours/swim event is the 95th percentile value. The exposure duration used here assumes a twice-weekly visit to the beach during June through September with one hour being spent in the water for each visit. Another source of information of amount of time spent in the water is the Michigan Recreational Surveys (cited in U.S. EPA 1998b), which also estimated four months of the year as reasonable for

swimming. The Michigan surveys indicated total hours of water exposure from swimming during the season at 28 hours. This is similar to the assumption presented in Table 5-1 of 32 total hours per season (one hour per visit, 32 visits per year).

6.1.3.5 Absorption

Gastrointestinal Absorption: The dose calculated by the exposure assessment is considered an "administered" or "applied" dose unless it is corrected for the extent of systemic absorption into the blood stream ("absorbed" dose). In general, the amount of absorption of chemicals should be adjusted in assessing exposure by a given route if absorption for the population at risk differs from the population (human or laboratory animals) used to develop the relevant toxicity criteria (see Section 5.2, Toxicity Criteria). This discrepancy may result from differences in the administered form of the toxicant, or from differences in physiological processes. A correction for gastrointestinal absorption via soil ingestion was considered appropriate only for arsenic, as discussed below.

Gastrointestinal absorption of ingested arsenic varies greatly with the water solubility of the arsenic compound and the physical form administered (U.S. EPA 1984). For example, absorption of arsenic trioxide is reported to be 30 to 40 percent for the compound in suspension, but as high as 95 percent and greater for the compound in solution (Ariyoshi and Ikeda 1974; U.S. EPA 1984). Because the toxicity criterion is based on inorganic arsenic dissolved in drinking water, an absorption correction should be considered for the differences between arsenic absorption from soil versus from drinking water.

Because of the uncertainty surrounding arsenic's bioavailability in soil, the differences in soil types, and the lack of human data, EPA Region 10 recommends using a bioavailability of 60 percent for arsenic in soil (personal communication, Roseanne Lorenzana 1998).

Dermal Absorption: Dermal contact with soil appears to occur during discrete exposure episodes that depend on the activity performed. Little is known about the kinetics of dermal absorption of various compounds from soil. Percutaneous absorption rates vary with the specific compound and soil matrix attributes. Contaminants may be less available for absorption from a soil with a high organic content due to an increase in anticipated partitioning into the organic phase of the soil. The absorption factors selected for soil and the study from which the value was derived are presented in Table 5-2.

To evaluate dermal contact with constituents in water, dermal absorption across the skin is determined using constituent-specific dermal permeability coefficients, expressed in units of centimeters per hour. Equations for calculating dermal permeability coefficients are presented in *Dermal Exposure Assessment: Principles and Applications* (U.S. EPA 1992a). The selected coefficients are presented in Table 5-3.

6.1.3.6 Intake Calculations

For each exposure pathway and age group, the following equation calculates unit exposure, as dose per mg/kg of chemical in soil or water based on the exposure assumptions (see Appendix E for detailed calculations):

Soil Ingestion:

$$\text{Summary Intake Factor (SIF)} = IR_s \times EF \times ED \times ABS/(BW \times AT)$$

Water Ingestion:

$$SIF = IR_w \times EF \times ED \times ABS/(BW \times AT)$$

Dermal Water Contact:

$$SIF = SA \times EF \times ED \times ABS \times Kp/(BW \times AT)$$

Dermal Soil Contact:

$$SIF = SA \times EF \times ED \times ABS \times AF/(BW \times AT)$$

where:

IR_s	=	soil ingestion rate (mg/day) (Table 5-1)
IR_w	=	water ingestion rate (mL/hour) (Table 5-1)
EF	=	exposure frequency (days/year) (Table 5-1)
ED	=	exposure duration (Table 5-1)
ABS	=	percent absorption (assumed to be 100 percent, except for gastrointestinal absorption of arsenic and dermal absorption of all chemicals)
AF	=	Adherence Factor (mg/cm^2) (Table 5-1)
Kp	=	Permeability coefficient (cm/hour) (Table 5-1)
BW	=	body weight (kg) (Table 5-1)
AT	=	averaging time (days) (ED x 365 days).
SA	=	skin surface area (cm^2) (Table 5-1)

Exposure is calculated separately for assessing carcinogenic risk versus noncarcinogenic hazard. The averaging time for noncarcinogenic effects is the same as the exposure period (6 or 24 years), whereas for carcinogenic effects the averaging time is equivalent to a lifetime (70 years, U.S. EPA 1991a).

For evaluation of carcinogenic exposure, pathways with different exposures for two age groups (e.g., child soil ingestion and dermal contact), the total dose is calculated by:

1. Weighting the intake of each age group (e.g., 0- to 6-year-olds) by the length of time spent in that age group (e.g., 6 years)

2. Summing the time-weighted doses from all age groups

3. Dividing by the averaging time, as follows:

$$SIF_{soil} = [ABS \times (IR_{child} \times EF_{child} \times 6\ yrs/BW_{child}) + (IR_{adult} \times EF_{adult} \times 24\ yrs/BW_{adult})]/\text{Averaging Time}$$

$$SIF_{dermal} = [ABS \times (SA_{child} \times EF_{child} \times 6\ years/BW_{child}) + (SA_{adult} \times EF_{adult} \times 24\ yrs/BW_{adult})$$

The dose for each pathway of exposure (ingestion of soil or water, dermal contact) will be combined with the relevant EPA toxicity criteria (Section 4.2) and target health goals (Section 5.3) to estimate RBCs. Appendix E contains the spreadsheets with calculation details and a presentation of each formula used.

6.2 TOXICITY CRITERIA

This section summarizes the relevant toxicity criteria that are used to calculate health protective RBCs associated with the dose of the COCs. A fundamental principle of toxicology is that the dose determines whether a chemical is toxic. Accordingly, the toxicity criteria describe the quantitative relationship between a chemical's dose and magnitude of toxic effect. The criteria are described below; toxicity criteria used in this assessment are summarized in Table 5-4 and a brief discussion of the basis of the criteria is presented for each chemical in Appendix F.

6.2.1 Oral Toxicity Criteria

Key dose-response criteria are EPA slope factors for assessing cancer risks, and EPA-verified reference dose (RfD) values for evaluating noncarcinogenic effects. These criteria are from the EPA's online data base Integrated Risk Information System (IRIS; U.S. EPA, 1998c).

6.2.1.1 Carcinogenic Effects

The carcinogenic SF (expressed as mg/kg-day^{-1}) expresses excess cancer risk as a function of dose. The dose-response model is based on high- to low-dose extrapolation, and assumes that there is no lower threshold for the initiation of toxic effects. Specifically, toxic effects observed at

high doses in laboratory animals or from occupational or epidemiological studies are extrapolated, using mathematical models, to low doses common to environmental exposures. These models are essentially linear at low doses, such that no dose is without some risk of cancer.

6.2.1.2 Noncarcinogenic Effects

The chronic RfD (expressed in units of mg/kg-day) is an estimated daily chemical intake rate for the human population, including sensitive subgroups, that appears to be without appreciable risk of noncarcinogenic effects if ingested over a lifetime. Chronic criteria are based on lifetime average body weight and intake assumptions.

RfD values are derived from experimental data on a no-observed-adverse-effect level (NOAEL) or lowest-observed-adverse-effect level (LOAEL) in animals or humans. A NOAEL is the highest tested chemical dose given to animals or humans that has not been associated with any adverse health effects. A LOAEL is the lowest chemical dose at which health effects have been reported. RfDs are calculated by dividing a NOAEL or LOAEL by a total "uncertainty factor," which represents a combination of individual factors for various sources of uncertainty in the data base for a particular chemical or in extrapolating animal data to humans. RfDs and associated uncertainty factors are summarized in Table 5-4 for each chemical. The EPA also assigns a level of confidence in the RfD, which is listed in the IRIS data base. The level of confidence is rated as either high, medium, or low based on the confidence in the study and confidence in the data base.

6.2.2 Dermal Toxicity Criteria

No RfDs or slope factors (SFs) are specifically available for percutaneous exposures. Risks and hazards associated with dermal exposure are evaluated using an oral toxicity factor corrected for absorption. This route-to-route extrapolation assumes that the toxicity of a hazardous constituent is the same regardless of the actual route of exposure. It is not appropriate to use oral toxicity factors to evaluate the dermal pathway when the compound exerts a specific point-of-contact effect (e.g., benzo(a)pyrene tumors originate on mouse skin following dermal application); however, that is not the case for any of the chemicals evaluated in this report.

Though toxicity criteria for dermal exposure are lacking, oral toxicity values are used instead to assess risks from dermal exposure. To determine dermal exposures, the oral toxicity value must be adjusted from an administered to an absorbed dose. An administered dose is one that is presented to a person's "exchange surfaces" or points of contact with the external world, including the mouth, skin, and nose. An absorbed dose is the fraction of the administered dose that actually enters the body's general circulation. Because the skin forms an effective barrier to many chemicals, only a fraction of the dose administered on the skin's surface will be absorbed through the skin into the bloodstream. Therefore in the RBC calculations, dermal exposure to contaminants in water and soil, was evaluated using the oral toxicity value adjusted to an absorbed dose. If the

oral toxicity factor is used unadjusted, the resulting risk or hazard estimates are less conservative because adjusted values are more protective than unadjusted oral values.

The chronic RfD for arsenic was not adjusted because the RfD is based on the no-observable-adverse-effect level (NOAEL) for skin effects from a study involving arsenic exposures to over 40,000 people in Taiwan. These people were exposed for a significant portion of their lifetime to arsenic-impacted groundwater used as drinking water; therefore the administered RfD is a good approximation of their absorbed dose (U.S. EPA 1998c). For cadmium, the administered oral RfD of 0.001 mg/kg-day (food) was multiplied by a gastrointestinal fraction of one percent to derive the dermal RfD of 0.00001 mg/kg-day (U.S. EPA 1998d).

6.3 CALCULATION OF RBCs

This section calculates potential health-based RBCs in soil and water at the various CUAs. Preceding sections quantified the possible amount of exposure in terms of a unit dose of chemical along with the relative toxicity associated with exposure. This section uses this information to calculate soil and water RBCs that are protective of health for the pathways of concern.

6.3.1 Calculation Methods

RBCs are calculated by defining a target risk goal, then solving the basic risk assessment equations for soil or water concentration rather than for risk (U.S. EPA 1991b). Target risk goals and equations differ for carcinogenic or non-carcinogenic effects.

Target cancer risk goals set by EPA for carcinogenic risk are defined over a range of 10^{-6} to 10^{-4} (U.S. EPA 1990a). The increased likelihood of cancer due to exposure to a particular chemical is defined as the excess cancer risk (i.e., in excess of a background cancer risk of 3 in 10 or 3×10^{-1}). The risk is estimated as the upper-bound probability of an individual developing cancer over a lifetime as a result of the exposure assumed in Section 5.1 (i.e., average lifetime dose). For example, 1×10^{-6} refers to an upper-bound increased chance of one in a million of developing cancer over a lifetime (0.0003 percent increase over background). The target risk goal is divided by the exposure estimate multiplied by the SF for each chemical to arrive at a soil or water concentration protective of human health at the target risk goal. The target risk goal selected for this evaluation is 1×10^{-5} because arsenic's (the only carcinogen) natural background concentration was above a 1×10^{-6} risk.

The following equation was used for calculation of RBCs for oral and dermal exposure to arsenic (the only carcinogen in this assessment):

$$RBC = \text{Target Risk}/SF \times (SIF_{soil\ or\ water} + SIF_{dermal})$$

where

Target Risk	=	Chance of developing cancer (1×10^{-5})
SF	=	Slope Factor (Table 5-4)
SIF	=	Summary Intake Factor

The target risk goal for noncarcinogenic hazards is typically a hazard quotient (HQ) of 1.0. An HQ of 1.0 is the point at which the estimated dose equals the RfD. The target health goal used in this assessment is an HQ of 0.1. A tenth of the RfD is assumed as a protective means of addressing additivity at the screening level. Other HQ assumptions that have been used are 0.25 in a previous risk assessment done on the 21-square-mile area commonly referred to as the Bunker Hill Superfund Site (SAIC 1991) and 0.2 in the Draft Water Quality Criteria Methodology Revisions (U.S. EPA 1998b).

Note that use of 1×10^{-5} risk threshold for cancer effects and a 0.1 target health goal for noncarcinogenic effects produced a lower RBC for noncancer effects of arsenic than cancer effects. The RBC for noncancer effects was used to screen sites.

RBCs for oral and dermal exposures were thus calculated using the following general equation for each pathway (see Appendix E for detailed calculations):

$$\text{Soil RBC} = \text{HQ} \times \text{RfD}/(\text{SIF}_{\text{soil or water}} + \text{SIF}_{\text{dermal}})$$

where

HQ	=	Hazard Quotient of 0.1
RfD	=	Reference Dose (Table 5-4)
SIF	=	Summary Intake Factor

The RBCs calculated for soil and water protective of children playing at the beach are presented on Table 5-5. In addition, the MCLs are included on this table as they are used in Section 6.0 to screen the two drinking water samples collected at CUA Numbers 17 and 27.

Table 5-1
Exposure Factors

Exposure Factors	Soil Ingestion/ Dermal Soil Exposure	Water Exposure While Swimming: Water Ingestion and Dermal Contact
Age Group	Child (1-7 yr)	Child (1-7 yr)
Body Weight (BW)	15 kg	15 kg
Ingestion Rate (IR)	300 mg/day	30 mL/hour
Skin Surface Area (SA)	6,500 cm^2	6,500 cm^2
Event Time- Swimming Only (hours/event)	--	1 hour
Exposure Frequency (EF) (days/year)	Twice a week June to September: 32 days/year	Twice a week June to September: 32 days/year
Exposure Duration (ED)	6 years	6 years
Adherence Factor (Soil) or Permeability Coefficient (water)	0.2 mg/cm^2	0.001 cm/hour
Gastrointestinal Absorption (ABS)	Arsenic, 60% relative to RfD Other metals, 100% relative to RfD	–

Table 5-2
Absorption of Chemicals From Soil

Compound	Dermal Absorption Factor	Reference
Arsenic	0.03	Wester et al. (1993)
Cadmium	0.001	U.S. EPA (1998d); Wester et al. (1992)
Inorganic Compounds (including lead)	0.01	Ryan et al. (1987)

Table 5-3
Permeability Coefficients From Water

Compound	Dermal Absorption Factor	Reference
Lead	0.0001	U.S. EPA (1992a)
Cadmium	0.001	U.S. EPA (1992a)
Default for other Inorganic Compounds (including arsenic)	0.001	Wester et al. (1993)

Table 5-4
Oral Toxicity Criteria

Chemical	Cancer: SF $(mg/kg\text{-}day)^{-1}$	Noncancer: RFD (mg/kg-day)	Toxic Endpoint	Uncertainty/Level of Confidence (only applies to RfD values)	Reference
Antimony	None	0.0004	Reduced lifespan, altered cholesterol levels	1,000 / Low Confidence	U.S. EPA 1998c
Arsenic	1.5	0.0003	Skin cancer (SF), hyper pigmentation and hyperkeratosis of the skin (RfD)	3 / Medium Confidence	U.S. EPA 1998c
Cadmium	None	0.001 (food) 0.0005 (water)[1]	Kidney proteinuria	10 / High confidence	U.S. EPA 1998c
Copper	None	0.037	Gastrointestinal irritation, flu-like symptoms	Not rated	U.S. EPA 1997b
Mercury	None	0.0003	Kidney damage	1,000 / Low confidence	U.S. EPA 1998c
Zinc	None	0.3	Anemia	3 / Medium confidence	U.S. EPA 1998c

Notes:
[1]Cadmium's food RfD was used in the soil RBC calculations and the water RfD was used in the water RBC calculations.

A brief discussion of the basis for the toxicity criteria is provided in Appendix F.

RfD - reference dose
SF - slope factor

Table 5-5
Risk-Based Concentrations

Chemical	Soil (mg/kg)	Water (µg/L)	MCL (µg/L)
Antimony	23	200	6
Arsenic	23[b]	140	50
Cadmium	40	230	5
Copper	2,110	17,000	1,300
Mercury	17	140	2
Zinc	17,100	140,000	5,000[a]

Notes:
See Appendix E for details of calculations.

[a]secondary standard
[b]screening was conducted using an estimated 95th percentile background concentration of 35 ppm rather than the risk-based concentration because the risk-based concentration was below the estimated background concentration.

7.0 SCREENING OF COMMON USE AREAS FOR CHEMICALS OTHER THAN LEAD

7.1 SCREENING METHODOLOGY

Four different media were sampled during the common use area (CUA) investigation (see Section 2.1) and require screening: (1) upland soils, (2) sediments (material below the high water mark), (3) Lake or River water in the "play-zone" (within three feet of shore), and (4) drinking water (only two locations). Sediment samples included material collected above the water line (on the beach) and below the water line in the "play zone" close to shore. Concentrations of chemicals were screened against their applicable risk-based screening concentrations (RBCs), i.e., soil and sediment were screened against soil RBCs, Lake and River water were screened against water RBCs, and drinking water was screened against MCLs. For all media except drinking water, screening was conducted in the step-wise fashion described below:

1. Does the maximum concentration of the chemical in soil, sediment, or water exceed the applicable RBC (soil ingestion scenario for soil and sediments, water ingestion while swimming scenario for water)?

If the answer to question one was *"no"*, the site was classified as *"sufficiently low risk to children"*, such that further evaluation in the baseline human health risk assessment (HHRA) would not be necessary. If the answer to question one was *"yes"*, a second question was asked:

2. Does an estimate of the upper 95 percent confidence limit (UCL_{95}) average concentration in beach soil, sediment, or water exceed the applicable RBCs?

If the answer to question two was *"no"*, the site was classified as *"sufficiently low risk to children"*, such that further evaluation in the HHRA would not be necessary. If the answer to question two was *"yes"*, the site was classified as *"possible risk to children"*, warranting further evaluation in the baseline HHRA. For arsenic, the soil RBC exceeded an estimate of natural background for the area (see Section 2.2); consequently, for arsenic in soil and sediment, if the answer to question two was yes, a final question was asked:

3. Does the estimate of the UCL_{95} average concentration in beach soil or sediment exceed the natural background concentration for the chemical?

If the answer to questions two and three were *"yes"* for arsenic, the site was classified as *"possible risk to children"*, warranting further evaluation in the baseline HHRA. If the answer to

question three was *"no"*, the site was classified as *"sufficiently low risk to children"* even if RBCs were exceeded, such that further evaluation in the HHRA would not be necessary.

Drinking water samples were evaluated as to whether chemical concentrations were above or below MCLs. Chemicals below MCLs were not considered further

7.1.1 Estimate of the Average Concentration

A person is not continuously exposed to the maximum concentration at a particular site but exposed to some average value of the range of concentrations present at a given location (i.e., person does not stand/play only at the maximum concentration location on every visit to the site). According to U.S. EPA (U.S. EPA 1991a, 1992b), when evaluating risks under an RME scenario, the site concentration should be a conservative estimate of the average concentration to which an individual would be exposed over a significant part of a lifetime. The use of the UCL_{95} of the arithmetic mean is generally recommended as the conservative estimate of the arithmetic mean (U.S. EPA 1991a, 1992b). At the UCL_{95} the probability of underestimating the true mean is less than 5 percent.

The formula used to calculate an UCL_{95} depends on the distribution of the data, i.e., the "shape" of the curve (U.S. EPA 1992b). The most common distribution for chemicals at impacted sites is lognormal; however, EPA recommends (1) performing a test on the data set to determine its distribution, and (2) graphing the data. For the chemicals other than lead, no maximum site concentrations exceeded RBCs except arsenic at eight locations and antimony at one location. Therefore, estimates of the average concentration at a CUA were calculated for arsenic and the single location for antimony. Appendix G contains a table of the results of a distribution check for arsenic and the appropriate UCL_{95}. The arsenic UCL_{95} values used in screening are also presented on Table 2-2 and Table 6-1.

7.2 RESULTS OF RISK-BASED SCREENING

Table 6-1 summarizes the screening process for arsenic and antimony (one site). Blackwell Island requires further evaluation in the baseline risk assessment due to the presence of arsenic in sediments. Details of the screening process follow.

7.2.1 Upland Soils

Upland soil data was collected from 14 of the 24 sites (see Section 2.1). The remaining ten locations did not have an upland area that people would use for recreation purposes, or the upland areas were above the high water level for the lake, such that sediment deposition was deemed

unlikely. The maximum detected arsenic concentration exceeded its RBC of 23 mg/kg at three locations (step one of screening methodology):

- Site #3 - Post Falls City Beach,
- Site #7 - BLM Pump Station,
- Site #26 - Loffs Bay.

In step two, the UCL_{95} for arsenic was greater than the RBC at two of three sites. BLM Pump Station and Loffs Bay had a UCL_{95} that exceeded the RBC. However, the UCL_{95} concentration does not exceed the estimated 95th percentile background concentration for arsenic of 35 mg/kg (step three); consequently, upland soils do not require further evaluation and are considered sufficiently low risk to children at all CUAs.

7.2.2 Sediments

Sediments were analyzed at 21 of the 24 sites. Of the remaining three locations, two (Rockford Bay #25 and Fuller Landing #30) only have upland picnic areas with boat ramps so there would be no exposure to sediments. The third location, #17 - Harrison Beach (west) has an extremely rocky shoreline and again, there would be no sediment exposure. Maximum arsenic concentrations exceeded the RBC at five locations and the maximum antimony concentration exceeded the RBC at one location:

- Site #8 - Corbin Park,
- Site #9 - Coeur d'Alene Beach at City Park,
- Site #18 - Harrison Beach (north) exceeded for both arsenic and antimony,
- Site #21 - Blackwell Island, and
- Site #24 - Mica Bay.

The UCL_{95} concentration for antimony at site #18 - Harrison Beach (north) was well below its RBC and thus site #18 was not selected for further evaluation based on antimony. Arsenic UCL_{95} concentrations exceeded the RBC at three of five sites; however, the UCL_{95} exceeded the natural background concentration for arsenic only at Blackwell Island. Therefore, Blackwell Island is retained for further evaluation in the baseline risk assessment because of a possible risk to children playing in the sediments.

7.2.3 Lake and River Water

Water samples of Coeur d'Alene Lake or the Spokane River were collected at each of the 24 CUAs as described in Section 2.1. Maximum concentrations of chemicals in the water did not exceed any RBC values (see Table 2-3 for maximum concentrations and Table 5-5 for RBCs).

Consequently, no further evaluation is necessary and the water is classified as of sufficiently low risk to children and is eliminated as an area of concern in the baseline risk assessment.

7.2.4 Drinking Water

Drinking water samples were collected at two locations: the campground at Harrison Beach (west) and a pipe at Windy Bay. Neither sample had any concentration above an MCL for the non-lead chemicals. At Harrison Beach, the total lead concentration was 15.5 µg/L, approximately equal to lead's action level at the tap of 15 µg/L. Harrison Beach (north), which would also receive drinking water from the campground, is retained for further evaluation based on lead concentrations in sediments (see Section 4.2) and the lead in drinking water will also be evaluated further at this location.

Table 6-1
Screening Results for Arsenic (All Sites) and Antimony (Site 18 Only)

Site Location	CUA Site ID	Site Name	Max. Conc. mg/kg	Max. Conc. Exceeds RBC?	UCL_{95} (normal dist.) mg/kg	Normal UCL Exceeds RBC?	UCL_{95} (lognormal dist.) mg/kg	Lognormal UCL Exceeds RBC?	UCL_{95} Exceeds Background of 35 mg/kg?	Selected for Evaluation in the Baseline RA?
Spokane River (7 Sites)	1	N. Idaho College Beach (Spokane River)	As = 20.6 (sed.) As = 12.5 (soil)	No	--	--	--	--	--	No
	3	Post Falls City Beach/River Park	As = 23 (sed.) As = 28.1 (soil)	Yes for soil	not normal	--	18.2	No	No	No
	5	Green Ferry Bay County Park	As = 15.5 (sed.) As = 9.1 (soil)	No	--	--	--	--	--	No
	6	Black Bay	As = 9.2 (sed.) As = 15.7 (soil)	No	--	--	--	--	--	No
	7	BLM Pump Station	As = 22.4 (sed.) As = 29.5 (soil)	Yes for soil	not normal	--	24.9	Yes	No	No
	8	Corbin Park	As = 38.7 (sed.) no soil data	Yes for sed.	not normal	--	25.9	Yes	No	No
	21	Blackwell Island	As = 83.4 (sed.) As = 20.3 (soil)	Yes for sed.	not normal	--	67.3	Yes	Yes	Yes
Coeur d'Alene Lake (17 Sites)	2	N. Idaho College Beach (CdA Lake)	As = 18.1 (sed.) no soil data	No	--	--	--	--	--	No
	9	Coeur d'Alene Beach at City Park	As = 29.1 (sed.) no soil data	Yes for sed.	not normal	--	14.9	No	No	No
	10	Tubbs Hill (site 1)	As = 6.3 (sed.) no soil data	No	--	--	--	--	--	No
	11	Tubbs Hill (site 2)	As = 14 (sed.) no soil data	No	--	--	--	--	--	No
	12	Tubbs Hill (site 3)	As = 9.1 (sed.) no soil data	No	--	--	--	--	--	No
	15	Higgan's Point (site 1)	As = 11 (sed.) no soil data	No	--	--	--	--	--	No
	16	Higgan's Point (site 2)	As = 8.9 (sed.) no soil data	No	--	--	--	--	--	No
	17	Harrison Beach (site 1, West)	As = 7 (soil) no sed. data	No	--	--	--	--	--	No
	18	Harrison Beach (site 2, North)	As = 158 (sed.) no soil data	Yes for sed.	not normal	--	29.8	Yes	No	No
			Sb = 55.6 (sed.) no soil data	Yes for sed.	11	No	not lognormal	--	--	No
	19	Cougar Bay	As = 10.3 (sed.) As = 11.5 (soil)	No	--	--	--	--	--	No
	23	Bell Bay	As = 4.9 (sed.) As = 23 (soil)	No	--	--	--	--	--	No
	24	Mica Bay	As = 27.1 (sed.) As = 18 (soil)	Yes for sed.	18.7	No	not lognormal	--	--	No
	25	Rockford Bay	As = 8.1 (soil) no sed. data	No	--	--	--	--	--	No
	26	Loffs Bay	As = 6.6 (sed.) As = 61.6 (soil)	Yes for soil	24.3	Yes	not lognormal	--	No	No
	27	Windy Bay	As = 5.8 (sed.) As = 3.8 (soil)	No	--	--	--	--	--	No
	29	Spokane Point (on reservation)	As = 5.6 (sed.) no soil data	No	--	--	--	--	--	No
	30	Fuller Landing	As = 4 (soil) no sed. data	No	--	--	--	--	--	No

Notes
CUA - Common Use Area
UCL_{95} - 95 percent Upper Confidence Limit
RBC - Risk-based Concentration
RA - risk assessment
As - Arsenic
Sb - Antimony
-- - not applicable
sed. - sediments
CdA - Coeur d'Alene
Data was entered in the UCL columns only if a maximum concentration exceeded the RBC fo 23 mg/kg.

8.0 UNCERTAINTIES IN THE EXPEDITED SCREENING ASSESSMENT

The purpose of the screening assessment was to identify CUAs that should be further evaluated in the Coeur d'Alene Basin RI/FS, or that could be eliminated from further concern. Uncertainty in the screening assessment produces the potential for two kinds of errors. The first is the potential to falsely retain a site for additional risk assessment when, in fact, the site need not be considered a concern (false positive conclusion). The second is to falsely eliminate a site from further consideration when, in fact, there should be a concern (false negative conclusion).

In the screening assessment, uncertainties were handled conservatively. This strategy is more likely to produce false positive errors than false negative errors. False positive errors are expected to be identified and corrected during the risk assessment to be completed as part of the Coeur d'Alene Basin RI/FS. Correcting false positive errors will prevent response actions where they are not necessary. On the other hand, if false negative errors are made during the screening assessment, a potentially hazardous site could remain in the public domain, and adverse effects on public health could occur. Therefore, uncertainties were handled conservatively in this screening assessment to reduce the potential for false negative conclusions.

Uncertainties reflect limitations in knowledge. In this assessment, uncertainties relate to: 1) the development of RBCs; and 2) the development of media concentrations that were compared with RBCs. The development of RBCs is uncertain in a number of assumptions regarding both exposure and toxicity, which include both site-specific and general uncertainties. Based on the treatment of uncertainty in RBC development, RBCs are likely to be overprotective, rather than underprotective. The RBCs developed for this screening assessment are more likely to cause sites to be retained although health risks are negligible. They are unlikely to screen out sites that may be problematic.

Uncertainty in the development of media concentrations is due to the inability to sample every square inch of potentially impacted media at a site. Instead, a limited number of samples must be acquired to represent the contaminant characteristics of a larger medium. The sampling strategy for this assessment was designed to prevent underestimates of media concentrations, which would lead to screening out sites that may pose a risk to public health.

The following sections provide additional detail regarding uncertainty in the development of RBCs and media concentrations.

8.1 UNCERTAINTIES IN THE DEVELOPMENT OF RBCs

RBC development requires assumptions about exposure and toxicity. Assumptions about exposure are generally site-specific, although some assumptions may rely on national databases or EPA risk assessment policy. Assumptions about toxicity are generally independent of the site, and depend primarily on EPA risk assessment policy.

8.1.1 Site-Specific Uncertainties in the Development of RBCs

Development of RBCs was based upon reasonable maximum exposure (RME) scenarios for exposures expected to occur in CUAs. Under the RME, exposure assumptions are based on upper 90th percentile values or upper-bound estimates of national averages. The intent of RME, as discussed by the EPA Deputy Administrator and the Risk Assessment Council (Habicht 1992), is "to estimate the risks that are expected to occur in small but definable 'high end' segments of the subject population." RMEs are not worst-case scenarios because "although it is possible that such an exposure, dose, or sensitivity combination might occur in a given population of interest, the probability of an individual receiving this combination of events and conditions is usually small, and often so small that such a combination will not occur in a particular, actual population." Thus, EPA makes a distinction between scenarios that are possible but highly improbable and those that are conservative but more likely to occur within a population.

The RBCs developed in this screening assessment are consistent with the latter. In other words, very few if any people would be likely to incur adverse effects following exposure to media concentrations at or below the RBCs. The following points outline some of the uncertainties in exposure parameters used to develop RBCs, and the expected impact on RBC development of how the uncertainties were treated.

- RBCs for soil and sediment included an assumption that **ingestion of soil and dust** during recreational activities were 300 mg/day for young children, and 100 mg/day for older children and adults. This applied to all chemicals except lead, because different values are used in the IEUBK lead model. The intake rate of 300 mg/kg day is the 90th percentile value from a study done by van Wijnen (1990) on the amount of soil ingested by children while camping. The average value from this study was 120 mg/day.

  If the average value was used to calculate RBCs instead of the 90th percentile value, RBC concentrations would increase by 40 to 50 percent. However, the conclusions of the screening assessment would not change. Blackwell Island would still be selected due to arsenic, even at an RBC based on a lower ingestion rate (46 mg/kg). At Harrison Beach (north), the maximum antimony concentration would equal the RBC based on a lower soil ingestion rate (55 mg/kg), suggesting

that antimony at this site is probably not a problem. However, Harrison Beach (north) would still be selected due to lead in sediment and water.

Individuals within a population may exceed assumed exposure rates. For example, a child on a given day may ingest a handful of dirt rather than 300 mg. A recent evaluation by Calabrese and Stanek (1995) suggests that the 90th percentile level for the average daily soil ingestion rate may be as high as 1,100 mg/day assuming the variability measured in warmer seasons can be extrapolated over a year. The 90th percentile of the average soil ingestion rate during the measurement period was about 180 mg/day (Calabrese and Stanek 1995). The soil ingestion rate is intended to be a daily average over the exposure period, rather than a maximum value, i.e., an actual child may ingest more than 300 mg one day but less than 300 mg on other days.

- Recreational users of the CUAs may have a higher **exposure frequency** than the two days per week assumed for the RBC calculations. If three days at the beach are assumed instead of two, RBC values drop by approximately 30 to 40 percent. However, no additional sites would be selected because UCL_{95} values did not exceed RBCs based on an exposure frequency of three days per week. (Note that arsenic screening was based on the background concentration rather than an RBC.) If the exposure frequency was increased to four days per week, Harrison Beach (north) would be selected based on antimony. However, Harrison Beach (north) has already been selected because of the lead concentrations in sediment and water. Therefore, uncertainty regarding exposure frequency does not appear likely to incorrectly exclude sites that may be a problem.

- Recreational users of the CUAs may have a shorter **exposure duration** than the thirty year total assumed for the RBC calculations. Shorter exposure durations would produce less-stringent RBCs. Use of the RME exposure duration in RBC calculations is likely to cause sites to be carried forward for further evaluation.

- RBC development did not include all possible exposure pathways. For example, the inhalation pathway was only discussed qualitatively because most information indicates that this pathway would be negligible when compared to ingestion. Therefore, the pathways that were not included in the calculation of RBCs were not expected to significantly lower the RBCs. It is unlikely that sites were inaccurately excluded because of omitted pathways.

- It is possible that CUA sites were either omitted or misclassified regarding uses and activities. If misclassification resulted in an underestimate of exposure

potential at a site, then a site could be omitted from further consideration when it should have been included.

8.1.2 General Uncertainties in the Development of RBCs

Development of RBCs requires toxicity criteria in addition to exposure assumptions. This screening assessment used toxicity values developed by the EPA from available toxicological data. EPA's development of toxicity values frequently relies on extrapolations from high-dose toxicity studies to low-doses incurred during environmental exposures. Also, toxicity criteria are often derived from animal rather than human data. Finally, there may be few studies available for a particular chemical. As the applicability, quality, and quantity of toxicity information decreases, the uncertainty of the toxicity value increases. This uncertainty is typically addressed by using uncertainty factors to reduce RfDs, and by deriving slope factors using a conservative model. The treatment of uncertainty applied by EPA is designed to overestimate toxicity. When applied to the development of RBCs, this conservatism will produce stringent RBCs. Sites are unlikely to be screened from further consideration due to underestimates of the toxic potential of chemicals. Several specific sources of uncertainty in the toxicity criteria are discussed below.

- For carcinogenic effects, U.S. EPA develops slope factors for risk assessment such that "… actual human risk probably does not exceed the upper limit and it is likely to be less. The actual cancer risk may even be zero in some situations" (EPA Guidelines for Cancer Risk Assessment, EPA/600/8-87/045, August 1987). Arsenic was the only carcinogen screened in this assessment. However, arsenic was screened based on background concentrations, which are higher than RBCs. Therefore, there is a potential risk from arsenic even at natural background concentrations. This uncertainty does not affect the screening of sites, however, since sites above natural background will be carried forward for additional analysis.

- The target hazard quotient goal selected for noncarcinogenic RBCs was 0.1. That is, RBCs were one-tenth of a concentration that might produce an adverse effect if all other exposure assumptions were realized. This assumption was considered appropriate for a screening level assessment where the intent was to assure an appropriate decision to exclude a site from further regulatory concern. However, in a baseline risk assessment, hazard quotients up to 1.0 may be considered acceptable depending on the chemicals and pathways involved. If a target hazard goal of 1.0 was used to calculate RBCs, no additional sites would be excluded from further consideration.
- The uncertainty in setting the bioavailability of arsenic at 60 percent to calculate the RBC does not affect the screening process. Arsenic site concentrations were compared to the background concentration of 35 mg/kg instead of screened against

the RBC of 23 mg/kg. If the RBC is used for screening, potentially more CUA sites would have been retained because setting the bioavailability of arsenic higher than 60 percent would have produced a lower RBC than 23 mg/kg.

- The exact absorbed dose by dermal contact for the toxicity criteria of the chemicals of concern is unknown. This uncertainty may produce either underprotective or overly protective RBCs.

- Interaction effects from simultaneous exposure to multiple chemicals can be additive, antagonistic (less than expected), or synergistic (more than expected). Whether chemical effects interact depends on the dose and mechanism of chemical action. For example, at high doses lead and cadmium may both affect the kidneys. At the RfDs used to calculate RBCs, none of the metals are expected to interact synergistically. Interactions among metals are often antagonistic (i.e., tending to cancel each other out) by competition for gastrointestinal absorption or by mechanisms related to detoxification processes (summarized by Goyer, 1996). For example, iron, calcium, and zinc decrease absorption and toxicity of cadmium and lead.

8.2 UNCERTAINTIES IN THE DEVELOPMENT OF MEDIA CONCENTRATIONS

The screening evaluation depends heavily on the quality and representativeness of the sampling data. Data were collected from environmental media at the CUAs for comparison with RBCs. The data evaluation process addressed whether: 1) chemicals were potentially present in various environmental media; 2) media concentrations were different from background; and 3) sufficient samples were collected to represent potential contamination at the sites.

During site characterization, over 380 soil and sediment samples were collected from the 24 CUAs. Sampling was intended to characterize sites based on historical and theoretical factors. CUAs along Coeur d'Alene Lake and the Spokane River were identified based on historical understanding of lake levels and flood events. All sites that might have been inundated with water bearing sediments were included.

Sample numbers at each location ranged from seven to twenty-six. Sample locations at each CUA were randomly selected. The number of samples collected was determined using the Max of N method (Conover 1980). The Max of N method was applied to make sure the data would bracket the 50th percentile of the population with a 95 percent confidence level. This assures that the data will not underestimate the mean of the population, which is the statistic used in risk assessment to evaluate long-term exposure. It is unlikely that chemical concentrations in the CUAs would be significantly higher than reported.

Uncertainties contributing to sample variation may involve the heterogeneity of the sample matrix (e.g., particle sizes in soil) and the field or laboratory analytical techniques. These sampling and analytical uncertainties may underestimate or overestimate site concentrations.

The expedited risk assessment only addressed seven metals; the seven metals that had been selected as a concern for the Bunker Hill Superfund Site. Analyses for other chemicals might indicate additional chemicals of concern, and identify sites that might be a concern if the chemicals were included in the screening. This source of uncertainty is expected to be low based on historical information about the site and information from other mining sites.

The development of background media concentrations includes some uncertainty. Data used to develop background concentrations came from Gott and Cathrall's regional study, and from the sediment study undertaken by URSG. The applicability of the data from these studies to background conditions at CUAs is uncertain, meaning that true background at any CUA may be higher or lower than the concentrations used for screening. In addition, the data sets themselves may be statistically evaluated by a number of methods, which may produce slightly different estimates of background. However, the methods are in relatively good agreement regarding background estimates, so the values presented in Section 2 of the report are expected to be reasonably representative of background. However, if the true background concentration of metals at sites is lower than the screening level, then it is possible that sites may have been excluded from further consideration when they should have been carried forward. For example, if arsenic background was 23 mg/kg, instead of the background value of 35 mg/kg used for screening, the following sites would have been retained for additional analysis based on the upper confidence limit (UCL_{95}) of the average arsenic concentrations:

- CUA #7 - BLM Pump Station
- CUA #8 - Corbin Park
- CUA #18 - Harrison Beach, North
- CUA #21 - Blackwell Island
- CUA #26 - Loffs Bay

It is possible to have missed hots spots, or smaller areas with elevated concentrations of metals, during site sampling. However, the theoretical basis for metals deposition on beaches involves transport of sediments in surface water. This mechanism should produce relatively homogeneous distributions of metals on the beaches of Coeur d'Alene Lake and along the Spokane River (this is not the case along the Coeur d'Alene River). Therefore, the chance of screening out sites that contained hot spots is considered small.

Integrating concentrations over depths may underestimate concentrations of metals on beaches, where samples were taken over a 12-inch horizon. This was considered a reasonable horizon

because beach sand may be mixed easily during beach play, especially digging. However, if metals have been deposited and remained primarily in a shallower horizon, concentrations may be underestimated. This could lead to screening out sites that would otherwise be carried forward for additional evaluation.

Finally, with any sampling event, the samples obtained are essentially a snapshot of site concentrations at the time of the sampling event. It can only be assumed, without prolonged monitoring programs, that the samples are representative of long-term exposure conditions. However, it is possible that, over the exposure durations assumed to develop RBCs, concentrations in the CUAs may become higher or lower. This possibility may result in inaccurately including or excluding sites.

8.3 UNCERTAINTIES SPECIFIC TO LEAD

The screening assessment for lead was conducted using the IEUBK model for lead in children (U.S. EPA 1994). The IEUBK model estimates the probability that children ages 6 months to 7 years exposed to lead in environmental media will have elevated blood lead concentrations (PbB). The following are sources of uncertainty in the risk assessment for lead:

1. Uncertainty in exposure scenarios, including exposure pathways and activity patterns;

2. Uncertainty in the appropriate input variables to the IEUBK model, including site characterization of environmental concentrations of lead; and

3. Uncertainty in the use of the 95th percentile blood lead concentration as a measure of health risk from lead exposure.

8.3.1 Exposure Scenarios

Potential lead exposure pathways for children include soil and dust ingestion, water ingestion, food ingestion, inhalation, and dermal contact. The dermal pathway cannot be estimated using the IEUBK model and there is no basis for estimating its contribution to lead uptake and risk; therefore, excluding the dermal pathway may underestimate lead exposures to some unknown degree.

The IEUBK model is generally structured to assess lead risks for children that are exposed at a residential location (i.e., individual risks). The predicted probability distribution for PbB reflects the differences in PbB that would occur among different children exposed at the same residential location (now, or in the future). In addition, multiple simulations for different residences may be

aggregated to assess a community level risk. For the screening assessment, exposure was assumed to occur at both residential and non-residential locations (e.g., parks and beaches). In the development of RBCs for CUAs, lead exposures are modeled by *summing* exposures at non-residential and residential locations, rather than estimating a time-weighted average intake. This approach may overestimate total lead intake if the time spent away from home actually reduces the total intake from residential exposures. Finally, there is uncertainty in the activity patterns of children at CUAs (e.g., it is unclear if certain areas within a beach or park are likely to be preferentially visited); therefore, for the screening assessment, non-residential exposure units are defined by the areas sampled during site characterization.

8.3.2 Concentration Term in the IEUBK Model

For most chemicals, EPA recommends using the UCL_{95} for the mean concentration to estimate CTE and RME risks (U.S. EPA 1992b). The fundamental concept is to use a measure of the (spatial) arithmetic mean concentration to yield a plausible central estimate of the typical (time average) exposure concentration contacted by an individual. The UCL_{95} addresses uncertainties due to limited site sampling and measurement error. For RME calculations, the use of a measure of central tendency is counterbalanced by the selection of upper range estimates for some of the other exposure variables, so that the end results is a "reasonable maximum" estimate of exposure.

For lead risk assessment, the input to the IEUBK model is generally the mean concentration rather than the UCL_{95}. The combination of central tendency estimates for all exposure variables yields a central estimate of PbB that is assumed to be the geometric mean value. A lognormal distribution of PbB is estimated from the geometric mean and an assumed (or empirical) geometric standard deviation (GSD). The GSD parameter represents an empirical estimate of the variability in PbB that is observed in children exposed to similar environmental lead concentrations. By selecting the 95th percentile PbB as the basis for the risk estimate (i.e., P10 = 5%), variability and uncertainty associated with the estimate of the mean exposure concentration is accounted for in the risk estimate.

The arithmetic mean was used as the CTE of lead concentrations in upland soil, sediment, and water. The basis for using the arithmetic mean is as follows:

1. Validation studies have shown good agreement between PbB distributions predicted by the IEUBK model and observed PbB at Superfund sites, when the inputs to the model are arithmetic means of the exposure concentrations (Hogan et al. 1998). There is no evidence that equally good agreement can be expected if other CTEs are used in the model.

2. If the UCL_{95} is used in the model to represent the CTE of environmental concentrations, and the 95th percentile PbB is used as the basis for the risk

estimate, then the resulting risk estimate (or RBC) derived from the IEUBK model can be expected to overestimate actual risk.

The UCL_{95} for soil and water/sediment concentrations were used in the latter context in the screening assessment to derive a highly conservative upper bound estimate of risk. Thus, we can be reasonably certain that there is no significant lead health risk to children where the arithmetic mean exposure concentration does not exceed the RBCs, and we have greater confidence in this conclusion where the UCL_{95} for the exposure concentrations does not exceed the RBC.

The screening process has preceded completion of data collection at the site, therefore, assumptions have been made about central tendencies of residential exposure levels. In the absence of data on the residential soil lead concentration, a range of 200 - 300 ppm was assumed. The lower end of the range, 200 ppm, is the IEUBK model default and is considered a plausible residential soil concentration for an urban setting not impacted by point sources (U.S. EPA 1989b, 1994d). This estimate of a plausible range is supported by preliminary soil samples from the upper basin of the Coeur d'Alene District (Gott and Cathrall 1980) as well as analyses of soils from other Superfund sites (Hogan et al. 1998). The range may be more health protective when applied to lower basin areas that are not impacted by upper basin sediments and, therefore, would be expected to have levels closer to background.

8.4 SUMMARY

Every aspect of the screening assessment contains multiple sources of uncertainty. Simplifying assumptions were made to develop RBCs . RME assumptions were applied to RBC development to produce relatively protective screening levels. Because of this, it is possible that sites have been selected for additional risk assessment in the Coeur d'Alene Basin RI/FS, even though they may not present a potential risk to public health. While it is also possible that, due to uncertainty, sites have been incorrectly excluded from further regulatory concern, the use of conservative assumptions in RBC development was intended to prevent this. The results of the RBC calculations and the screening evaluation therefore are likely to be protective of health despite the uncertainties inherent in the process.

8.0 SUMMARY AND CONCLUSIONS

Twenty-four locations were selected around Coeur d'Alene Lake and the Spokane River as representative sites, which were used for recreation. These Common Use Areas (CUAs) were the focus of this screening health evaluation. Concentrations of chemicals in soils, sediments, and water were compared to risk-based screening concentrations (RBCs). If an environmental concentration exceeded a RBC, the site was retained for further evaluation in the upcoming Baseline Human Health Risk Assessment. If a concentration was below its RBC, the site was considered to have sufficiently low risk to children (the most sensitive population) and was eliminated from further consideration. Only two sites were selected for the Baseline Risk Assessment: Harrison Beach (north) and Blackwell Island.

The 24 locations were sampled for upland soils, sediments (below high water mark), surface water in the "play-zone", and drinking water was sampled at two locations. Soil, sediment, and surface water sample results were compared to RBCs protective of a child playing at the beach in the soil and water two days per week for four months of the year. Drinking water results were compared to MCLs. Soil and water RBCs were developed for seven metals of concern (antimony, arsenic, cadmium, copper, lead, mercury, and zinc). These seven metals were chosen as chemicals of concern based on findings from the Bunker Hill Superfund risk assessment (Jacobs Engineering et al. 1989).

The RBC for lead was developed using the IEUBK Model with recreational assumptions for exposure frequency (two days per week) and increased soil ingestion rates over the Model defaults. Results of the modeling predicted soil and water concentrations such that the chance of a child exceeding the blood lead goal of 10 µg/dL was no more than 5 percent (or a 5 percent chance to exceed a blood lead level of 10 µg/dL). Sites were screened using a decision tree approach:

- Did the average lead concentration in soil or sediment exceed the soil ingestion RBC?
- Did the average lead concentration in surface water exceed the water ingestion RBC?

Sites were selected as needing further evaluation if the answer to any of the above questions was yes. Harrison Beach (north) was the only site retained for further evaluation because surface water ingestion combined with sediment ingestion exceeded the blood lead goal.

RBCs for chemicals other than lead were calculated using EPA's standard risk equations and calculating a soil or water concentration rather than risk or hazard. A target risk cancer goal of 1 x 10^{-5} (one excess cancer in 100,000 allowed) was selected for arsenic (the only carcinogen). A hazard quotient of 0.1 was selected as a goal for the non-cancer health endpoints. RBCs were compared initially to the maximum concentrations at a site. If the maximum concentration exceeded, an estimate of the average concentration (UCL_{95} of the mean) was compared to the RBC. If the UCL_{95} exceeded the RBC, the UCL_{95} was compared to the natural background concentration of the metal. The result of the screening found only Blackwell Island sediments exceeding both the RBC and natural background concentrations of arsenic.

Analytical results for drinking water samples collected from the Harrison Beach campground and Loffs Bay did not exceed MCLs. The total lead concentration at Harrison Beach was 15.5 µg/L, which is approximately equal to the tap water action level for lead of 15 µg/L. Harrison Beach (north) is already retained for further evaluation based on lead in sediments and surface water. Drinking water at the campground will also be further evaluated.

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FAX COVER SHEET

Date: November 18, 1998 Pages with Cover: 5

To: Cd'A Distribution Fax: Distribution

From: Philip E. Goodrum Fax: 315-452-8413
goodrum@syrres.com Phone: 315-452-8440

Subject: Arsenic - Background concentrations

Name	Fax
• Jerry Cobb, PHD	208-783-4242
• Ed Leach, CH2MHill	425-462-5957
• Ian Von Lindern, Terragraphics	208 883-3785
• Roseanne Lorenzana, EPA R10	206-553-0119
• Sean Sheldrake, EPA R10	206-553-0124
• Marc Stifelman, EPA R10	206-553-0119

We evaluated different truncation levels in order to investigate the uncertainty associated with where one draws the "bright line" to distinguish the background samples vs. the site-impacted samples. Graphs are attached - Fig 1 shows all of the data and the 3 truncation points, Figs 1A, 1B, and 1C give the regression statistics for the subset of the sample data associated with each truncation. We also calculated the 95% LCL and 95% UCL for the parameters of the regression line, then used the COMBINED 95%LCLs to estimate a conservatively low confidence limit on the upper percentiles, and the COMBINED 95% UCLs to estimate a conservatively high confidence limit. The true CI's for each percentile would be somewhat narrower, but the analysis is a little more complex. Given the high r^2 values(~0.98), it's probably not necessary to calculate these exactly (i.e., the CI's are very narrow already).

Figure	Truncation	N	90th %ile (LCL, UCL)	95th %ile	99th %ile
1-A	<= 12.5	116	10.7 (10.4, 11.0)	12.5 (12.1, 12.9)	16.7 (16.1, 17.4)
1-B	<= 19.7	131	13.2 (13.0, 13.4)	15.9 (15.6, 16.1)	22.4 (21.9, 22.9)
1-C	<= 41.8	149	19.0 (18.3, 19.7)	24.1 (23.1, 25.1)	37.7 (35.8, 39.6)

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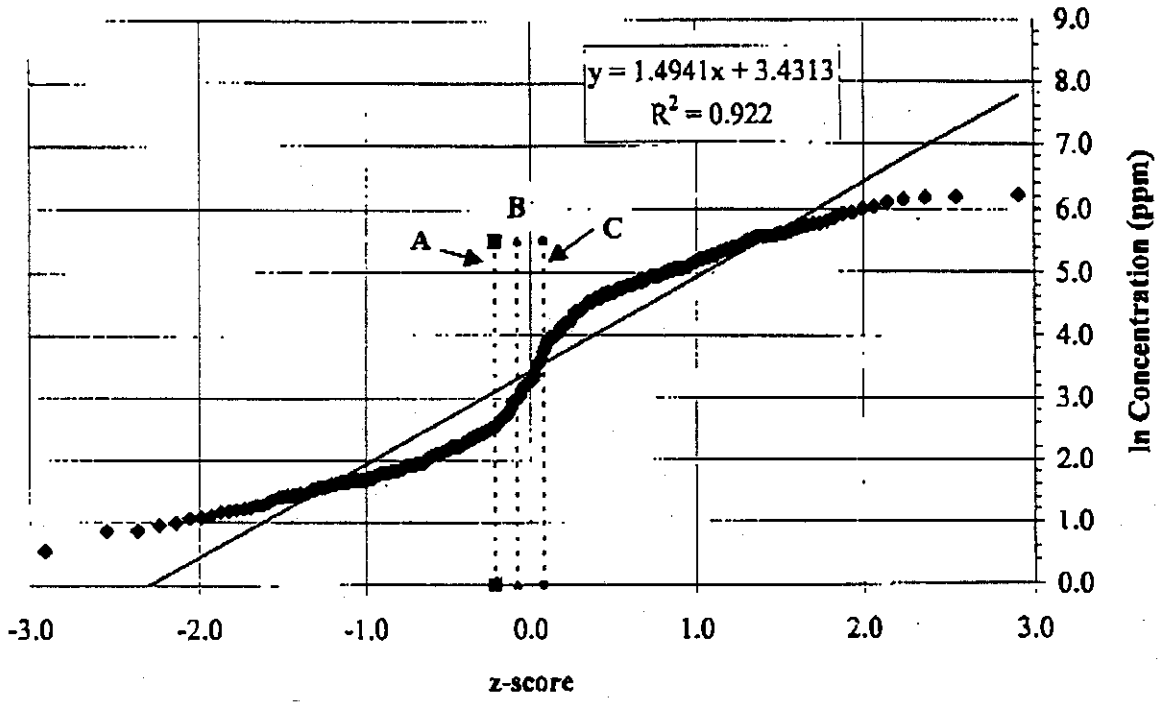
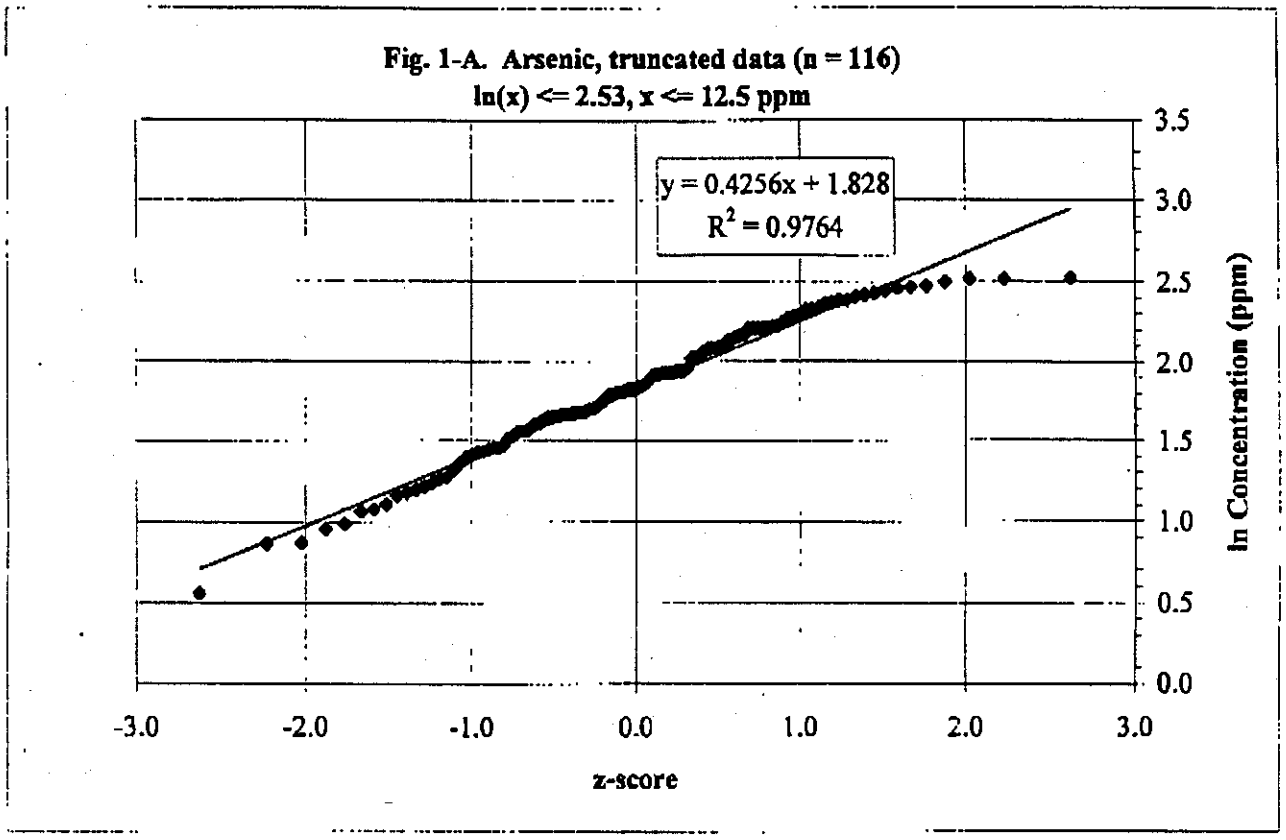
Fig. 1. Arsenic, all data (n = 279), 3 Truncation Levels for Background



Summary Statistics			
slope	1.49		
95% LCL	1.44		
95% UCL	1.55		
intercept	3.43		
95% LCL	3.38		
95% UCL	3.48		
r^2	0.922		
n	279		
AM	83.78		
SD	106.05		
GM	30.92	Combination of	
GSD	4.46	LCL's	UCL's
90th %ile	210	187	236
95th %ile	361	315	414
99th %ile	999	842	1186

Description of Data Set: ARSENIC

- from URSG file: soil_sed_metals_11_12_98.xls
- sediment in LC {lower basin}
- detects only
- All conc. data



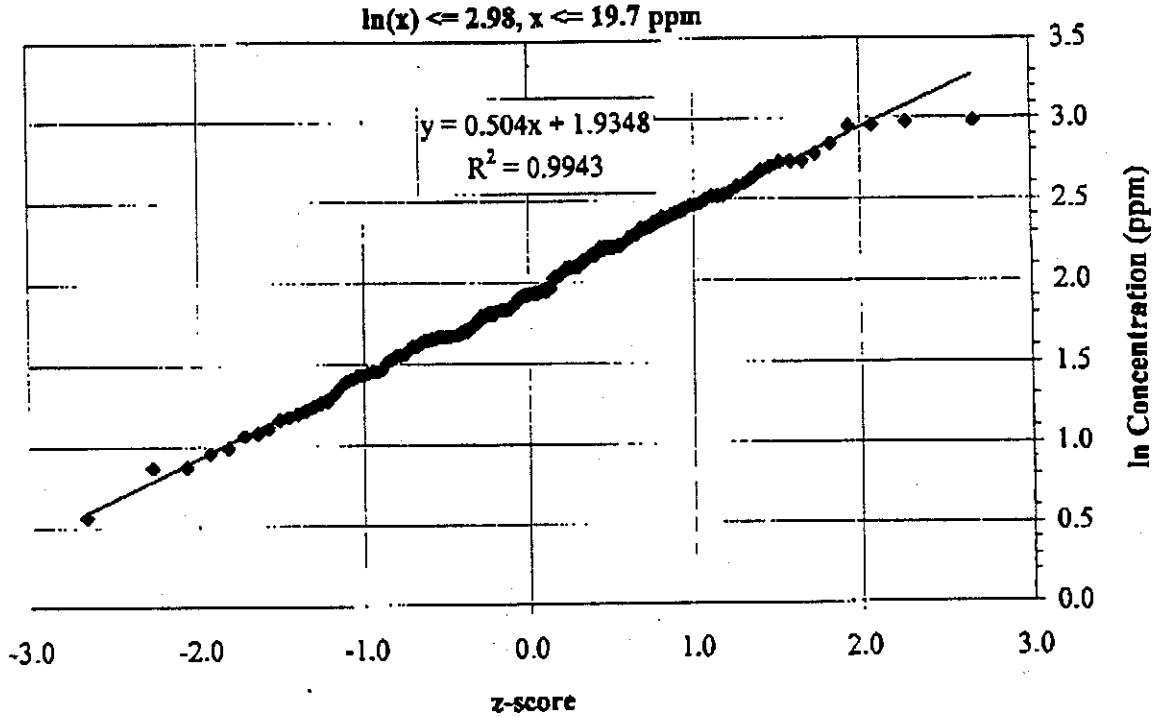
Summary Statistics	
slope	0.43
95% LCL	0.41
95% UCL	0.44
intercept	1.83
95% LCL	1.82
95% UCL	1.84
r^2	0.976
n	116
AM	6.78
SD	2.71
GM	6.22
GSD	1.53
90th %ile	10.7
95th %ile	12.5
99th %ile	16.7

Combination of	
LCL's	UCL's
10.4	11.0
12.1	12.9
16.1	17.4

Description of Data Set: ARSENIC

- from URSG file: soil_sed_metals_11_12_98.xls
- sediment in LC {lower basin}
- detects only
- Background defined as ln(x) <= 2.53

Fig. 1-B. Arsenic, truncated data (n = 131)
ln(x) <= 2.98, x <= 19.7 ppm



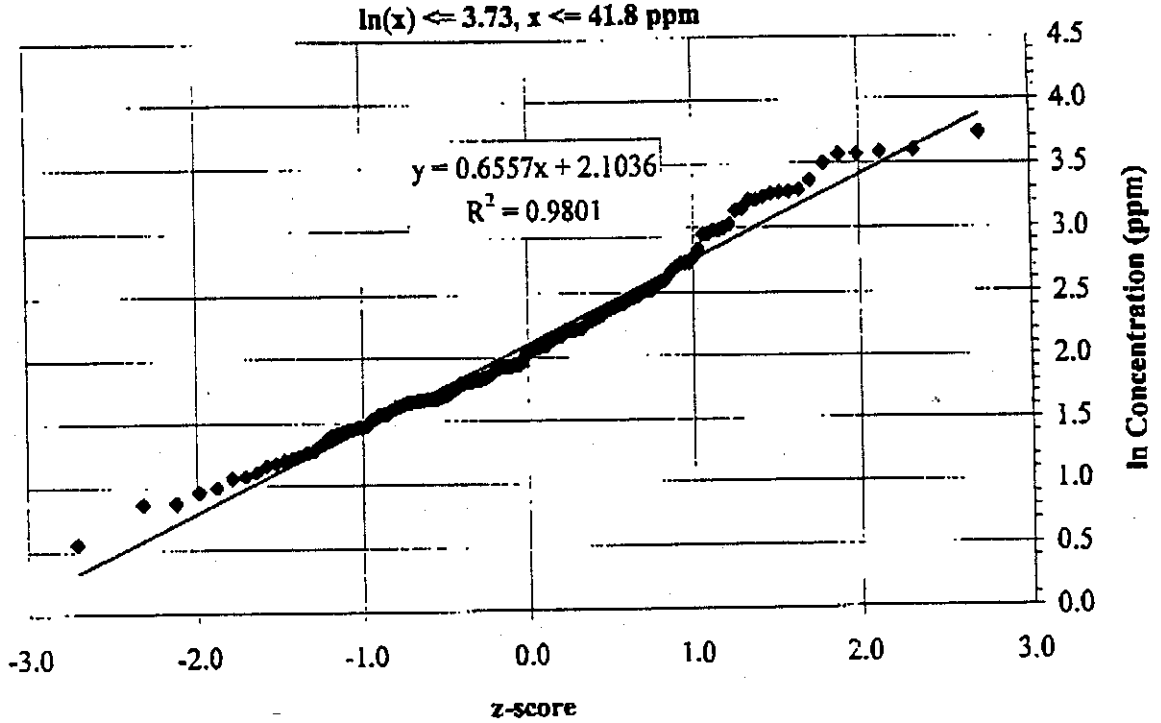
Summary Statistics	
slope	0.50
95% LCL	0.50
95% UCL	0.51
intercept	1.93
95% LCL	1.93
95% UCL	1.94
r^2	0.994
n	131
AM	7.83
SD	3.97
GM	6.92
GSD	1.66
90th %ile	13.2
95th %ile	15.9
99th %ile	22.4

Combination of	
LCL's	UCL's
13.0	13.4
15.6	16.1
21.9	22.9

Description of Data Set: ARSENIC

- from URSG file: soil_sed_metals_11_12_98.xls
- sediment in LC {lower basin}
- detects only
- Background defined as ln(x) <= 2.98

Fig. 1-C. Arsenic, truncated data (n = 149)
ln(x) <= 3.73, x <= 41.8 ppm



Summary Statistics			
slope	0.66		
95% LCL	0.64		
95% UCL	0.67		
intercept	2.10		
95% LCL	2.09		
95% UCL	2.12		
r^2	0.980		
n	149		
AM	10.34		
SD	8.03		
GM	8.20	Combination of	
GSD	1.93	LCL's	UCL's
90th %ile	19.0	18.3	19.7
95th %ile	24.1	23.1	25.1
99th %ile	37.7	35.8	39.6

Description of Data Set: ARSENIC

- from URSG file: soil_sed_metals_11_12_98.xls
- sediment in LC {lower basin}
- detects only
- Background defined as ln(x) <= 3.73

Coeur d'Alene, Idaho River Basin
Common Use Areas
Risk Assessment Report

SRC TR-99-001

Draft Report
February 1999

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No. SE-98-A-713
Task No. 2

EXECUTIVE SUMMARY

The purpose of this report is to provide an estimate of the concentrations (or a range of concentrations) of lead that would be expected in soil, groundwater and surface water in the Coeur d'Alene River Basin had local mining and smelting operations not occurred. The estimates take into consideration the geology of the area and local anthropogenic sources of lead, including automobile emissions and deteriorating exterior lead based paint.

The term *local background concentration* is used in this report to refer to the concentrations of lead that would be expected in the Coeur d'Alene Basin in the absence of historic and on-going emissions from local mining, smelting and other ore processing operations. This definition is consistent with EPA guidance, which defines background for inorganics as *"...the concentration of inorganics found in soils or sediments surrounding a waste site, but which are not influenced by site activities or releases."* (EPA, 1995) *Regional background* refers to the contributions from long-range atmospheric transport of anthropogenic and natural sources, and contributions from geologic formations in the area.

Figure 6, in the main body of the report, shows the estimated range for local background soil lead concentration for the Coeur d'Alene Basin, along with the estimated ranges for the contributions from regional sources, automobile emissions and lead based paint. The latter are considered to be the major potential anthropogenic sources of soil lead, other than mining and smelting related contamination of soil. The range for the local soil lead concentrations is estimated to be 120-2700 ppm; the lower and upper bounds on the range represent the sums of the lower and upper bound estimates, respectively, of the three major contributing sources to local background.

The global/regional contribution is estimated to range from approximately 20 ppm to 250 ppm. Locations in the basin where the regional geology and long-range atmospheric transport of lead are the dominant sources of lead in soil would be expected to have soil lead concentrations that fall within this range.

The potential contribution of historic automobile emissions to local background may range from approximately 10 to 450 ppm. This range would apply to areas in the basin adjacent to high-volume roads (e.g., Interstate 90); a lower upper bound estimate of 120 ppm may apply to low-volume roads. These estimates are highly uncertain, primarily because of the lack of data on automobile impacts in the basin region. What data are available relate to transportation corridors that were heavily impacted by local mining and smelting operations. Thus, the estimate of the automobile contribution to local background is derived from studies conducted outside of the basin, and may not be directly applicable to the basin region.

The potential contribution from lead based paint to local soil lead background may range from approximately 100 ppm to 2000 ppm. Soil adjacent to structures that have or had deteriorating exterior lead based painted surfaces may have soil lead concentrations that fall within this range. Here again, considerable uncertainty is associated with these estimates because of a lack of site-specific data paint lead impacts on soil in the Basin. The estimates are derived from studies outside the basin and may not be directly applicable to any given location within the Basin.

In summary, the major sources of uncertainty in the estimated range of local background soil lead concentrations in the basin include the following:

1) The broad range of arithmetic mean soil lead concentrations attributed to exterior lead-based paint reported in the literature (100-2,000 ppm). (Chaney et al., 1988; Hardy et al., 1971; Mielke et al., 1983; Ter Haar and Aronow, 1974)

2) The broad range in automobile contributions to local background soil lead concentrations (11-442 ppm) reported in the literature (Agrawal et al., 1981; Case et al., 1989; Motto et al., 1970; Ward et al., 1975; Wheeler and Rolfe, 1979; Wong and Tam, 1978)

3) Ignoring the geo-spatial information present in the available data. The range in regional soil lead concentrations shown in Figures 5 and 6 represents information obtained from soil and sediment samples collected throughout the Coeur d'Alene River Basin. (Gott and Cathrall, 1980; Hagler-Bailly, 1995; Reece et al., 1978; URSG, 1998) It is likely that the regional soil lead concentration varies with location.

4) Combining soil and sediment data to develop an estimate of regional soil lead background. In some areas within the Coeur d'Alene River Basin such as the floodplains, combining data from both media may be reasonable. In areas not located within the floodplains, the appropriateness of combining soil and sediment data is questionable.

Collection and analysis of site specific data would be expected to decrease the level of uncertainty in the estimated local background soil lead. Consideration of the spatial information in the data would tend to decrease the level of uncertainty in the estimate attributed to all of the sources listed above. Geo-spatial information could be used to address the spatial distribution of anthropogenic contributions (i.e., lead-based paint and automobile emissions) to the local background soil lead concentration as well as the spatial distribution of regional soil and sediment lead background concentrations.

The estimated range for local background groundwater lead concentration in the Coeur d'Alene River Basin is 5-15 μg/L. Estimates for the local background dissolved and total lead concentrations in surface water are 3 and 7 μg/L, respectively.

Uncertainties associated with the groundwater and surface water background estimates is primarily due to the limited data. Collection of site specific data and consideration of temporal and spatial trends would tend to decrease the uncertainty in the estimates of local background lead concentration in groundwater and surface water.

1.0 INTRODUCTION AND OBJECTIVES

One objective of the Coeur d'Alene Basin-Wide Remedial Investigation/Feasibility Study (RI/FS) is to estimate human health risks associated with lead contamination of the basin from local mining and smelting operations. This report summarizes available information relevant to estimating concentrations (or a range of concentrations) of lead that would be expected in soil, groundwater and surface water had local mining and smelting operations not occurred, taking into consideration the geology of the area and other local anthropogenic sources of lead.

2.0 DEFINITION OF BACKGROUND

The term *local background concentration* is used in this report to refer to the concentrations of lead that would be expected in the Coeur d'Alene basin in the absence of historic and on-going emissions from local mining, smelting and other ore processing operations. This definition is consistent with EPA guidance, which defines background for inorganics as *"...the concentration of inorganics found in soils or sediments surrounding a waste site, but which are not influenced by site activities or releases".* (EPA, 1995).

Contributions to local background, as defined above, include both natural and anthropogenic sources of lead. Natural contributions to local background include the decomposition of lead-bearing rock and volcanic activity. Anthropogenic contributions to local background in the Coeur d'Alene basin may include coal and oil/waste oil combustion, solid waste disposal, and historic deposits from leaded gasoline combustion and dusts from lead-based paint. (EPA, 1986)

Background sources can be classified into geographic source categories (EPA, 1995):

> *Global Contributions* – from long-range atmospheric transport, including anthropogenic and natural sources.
>
> *Regional Contributions* –from geologic formations in the area.
>
> *Local Contributions* - from local anthropogenic activities.

For the purpose of estimating local background concentrations for use in the RI/FS, in which we are interested in background from sources other than mining and smelting, the global and regional categories have been combined, and background is assumed to consists of two major geographic sources categories:

> *Regional Contributions* - from long-range atmospheric transport of anthropogenic and natural sources, and contributions from geologic formations in the area.
>
> *Local Anthropogenic Contributions* - from local anthropogenic activities other than those attributed to mining and smelting operations.

3.0 APPROACH TO ESTIMATING LOCAL BACKGROUND

3.1 Information Sources

The human health risk assessments of the Bunker Hill Superfund Site (Jacobs, 1989; SAIC, 1991; TerraGraphics, 1990) (including the reference sections) and USEPA Air Quality Criteria for Lead, Vol. II (EPA, 1986) were reviewed for potential sources of information relevant to the estimation of background concentration. In addition, scientists at URS Greiner (Sharon Quiring and Steven Hughes) were queried for reports, data, etc., relevant to the estimation of background lead concentration. A list of all references surveyed for information relevant to local background is included in Appendix A.

3.2 Local Soil Background

Ideally, local background concentrations in the Coeur d'Alene basin could be estimated from surface soil samples collected at locations in the basin where contributions from mining and smelting operations are negligable, by virtue of distance and/or direction from the source(s). Although such samples may have been collected and analyzed for lead, interpretation of reported data available for this assessment is problematic because precise georeferences to the samples, point sources and potential transport mechanisms have not been reported. This is a major uncertainty in the estimates of local background provided in this report.

Background concentrations are sometimes estimated from subsurface samples collected at depths considered to not be impacted by transport from the source. However, this requires that a depth or range of depths be defined that comprises the impacted zone. Depth of the impacted zone can be expected to be influenced by several factors. In soil, lead is transported from the surface by infiltrating precipitation. The depth to which lead is transported is a function of physical and chemical characteristics of the soil and chemistry of the precipitation. Stable lead isotope signatures have been used to detect anthropogenic impacts to soil strata (Shotyk et al., 1998, Gulson et al., 1981), however, an analysis of this type on Coeur d'Alene Basin soil strata has not been reported. Some investigators have made the assumption that soil samples collected at a depth greater than six inches are not likely to be have been significantly impacted by anthropogenic sources of lead and, therefore, provide reasonable estimates local background (Gott and Cathrall, 1980; Shacklette and Boerngen, 1984). In general, this is a reasonable assumption; several studies have shown that lead tends to remain in the upper 2-5 cm of undisturbed soil (EPA, 1986; Little and Martin, 1972; Motto et al., 1970; Ward et al., 1975). However, a rigorous evaluation of the applicability of the general case to soils in the Coeur d'Alene Basin has not been reported. Determining the impacted zone in sediments is even more problematic because of the erosion and deposition of the sediments which constantly changes the horizontal and vertical distribution of the impacted sediments. Annual flooding along the Coeur d'Alene River and its tributaries, and the resulting deposition of contaminated and uncontaminated sediments on the soil surface also complicates the determination of contaminated zones in soil. This is an additional uncertainty associated with estimates based on subsurface soils.

Subsurface soil samples collected below the impacted zone will not reflect local anthropogenic contributions, including those that are unrelated to the primary impacts of concern, mining and smelting operations. For example, in the Coeur d'Alene Basin, automotive emissions and lead based paint can be expected to contribute to local background; however, these impacts may not be reflected in subsurface soil samples. Thus, soil lead concentrations in subsurface samples can be expected to underestimate local background contributed by activities unrelated to mining and smelting. Impacts of automobile emissions and lead-based paint on soil lead contributions measured at other locations can be used to estimate the range of potential impacts that these sources may have on the Coeur d'Alene Basin soils.

3.3 Local Background of Surface and Groundwater

This report focuses on estimating local background concentrations in soil primarily because contaminated soil is likely to be the most significant contributor to lead intakes in young children, the primary receptor of concern for lead. Many of the above principles discussed in relation to soil background estimates also apply to estimations of background concentrations in surface and groundwater. Available data on lead concentrations in surface water and groundwater in the basin and estimates of the local background lead concentrations are provided in Section 5 of this report.

4.0 LAND USE HISTORY RELEVANT TO LOCAL LEAD BACKGROUND

A general discussion of the land use in the area during and after the operation of the Bunker Hill Complex is provided below. The purpose of this discussion is to 1) identify significant, non-mining/smelting anthropogenic contributions to the lead concentrations observed in soil and sediment located within the basin; 2) develop a general description of the area that will allow comparison of typical lead concentrations found outside the basin to concentrations detected within the basin.

4.1 Mining and Smelting History

Mining within the Coeur d' Alene Basin started in 1883 soon after the discovery of gold. In 1885 mining of lead ore was initiated. Ore processing has taken place at the Bunker Hill complex site since the first mill was built in 1886. Lead smelting began in 1917 followed by zinc smelting in 1927. The complex ceased all operations in 1981. (Jacobs, 1989) Mining has taken place primarily along the South Fork of the Coeur d' Alene River and its tributaries. Approximately 97 abandoned mines exist within the drainage basin (SAIC, 1993). Human activity in the area prior to mining/ore processing is assumed to be insignificant with respect to potential contributions of lead to the environment.

Potentially significant, local, non-mining/smelting anthropogenic sources of lead in the region include gasoline/diesel combustion, coal combustion, oil/waste oil combustion, solid waste disposal and foundries (EPA, 1986, 1989; SAIC, 1991). Other than mining, the major industry in the area has been logging (SAIC, 1991).

4.2 Industrial Sources of Lead Emissions other than Mining and Smelting.

4.2.1 Review of the TRIS and AIRS/AFS Databases.

A search of the EPA Toxic Release Inventory data base (TRI) for reporting year 1996 failed to find any facilities in Shoshone or Kootenai counties emitting lead to the environment. The TRI database includes only those facilities classified under certain Standard Industrial Codes (SICs) that employ more than 10 people and manufacture or process more than 25,000 lbs. of lead per year, or otherwise use more than 10,000 lbs. of lead per year. (EPA, 1998) The completeness and accuracy of the data in TRIS depends upon the compliance of the regulated community. It is likely however that major sources of lead have been identified. Relative to the other anthropogenic sources, particularly the smelter, mining and automobile sources, other minor sources are probably not significant with respect to determining the range of local background concentrations.

The EPA Aerometric Information Retrieval System (AIRS)/ AIRS Facility Subsystem (AFS) was also searched for potential sources of lead emissions within the basin. The search did not find any sources of lead emissions within Shoshone or Kootenai counties. However, reports for many of the facilities did not include a list of the pollutants emitted. The AIRS/AFS database includes point sources that emit more than threshold amounts of pollutants. For lead the threshold is 5 tons per year. Data in AIRS/AFS are obtained from state databases, inspection reports and facility personnel. The data may not be accurate or complete.

Power plants are a potentially significant source of lead from the combustion of coal and fuel oil; however, data on the local power plants are not readily available. In 1998, power plants will be required to report their emissions under Title III, Section 313 of the Superfund Amendments and Reauthorization Act of 1986 for the first time. However, lead emissions from coal and fuel oil combustion were three orders of magnitude less than gasoline, in 1968, for the country as a whole (Lovering, 1976). More recent data on the relative contributions of lead from power plants and automobiles have not been identified.

4.3 Non-Industrial Sources

In general, soil lead concentrations from 30 to 2000 ppm above background have been attributed to automobile emissions at locations near roadways, depending on daily traffic volume and distance from roadways (EPA, 1986). Interstate 90 runs along the South Fork of the Coeur d'Alene River, passing by the highest populated towns and cities in the river basin. Although leaded gasoline is no longer in use, it is likely that soil lead contamination due to automobile emissions prior to the phase-out of leaded gasoline is still present in the soil due to the persistent nature of lead in soil (Chaney, et al, 1988; EPA 1989). A discussion of the potential impacts of automobile emissions from vehicle traffic on Interstate 90 and local roads is discussed in Section 6.3.

Elevated soil lead concentrations as high as 2000 ppm have been associated with the use of lead-based paint on the exterior of homes (Chaney et al., 1988; Hardy et al., 1971; Ter Haar and

Aronow, 1974). Forty-four percent (329) of the households assessed by the Idaho Department of Health and Welfare for exterior lead based paint hazard contained lead based paint. (IDHW, 1998) Of the 329 homes, the exterior lead paint on approximately 39% (138) was characterized as being in poor condition. (IDHW, 1998) This potential source of lead contamination is discussed further in Section 6.4.

5.0 REVIEW OF DATA PERTINENT TO ESTIMATING REGIONAL BACKGROUND SOIL LEAD CONCENTRATIONS

5.1 Studies Performed Within the Basin

The following section summarizes studies undertaken within the basin that have generated data that may be useful for estimating local background soil lead concentrations.

5.1.1 Gott and Cathrall (1980)

Data from Gott and Cathrall (1980) were used as a basis for estimating background lead concentrations in soil and sediment in previous assessments of the Bunker Hill Superfund Site including the HHRA Protocol for the Populated Areas (Jacobs, 1989), Risk Assessment Data Evaluation Report for the Populated Areas (TerraGraphics, 1990), and HHRA for the Non-Populated Areas (SAIC, 1991).

The purpose of the Gott and Cathrall (1980) study was to determine if geochemical methods could be used to detect ore deposits that had not been discovered by other methods. The study collected 8,700 soil samples from the Coeur d'Alene District, which includes the Coeur d'Alene River Basin and the source area for sediments found in the lower basin. Samples were collected at different geographic intervals depending upon the terrain. Along jeep trails, samples were collected at 0.1-mile intervals. Samples were collected at approximately 300-foot intervals along ridgelines that were traversed on foot. Soil samples were collected at a minimum depth of six inches below the A soil horizon. Alluvial soils were not sampled. Lead concentrations were determined by atomic-absorption spectroscopy of the minus 80-mesh portion of the samples. The detection limit was reported as 25 ppm.

Table 1 shows the percentile soil lead concentrations reported by Gott and Cathrall (1980) and predicted percentile values based on the best-fit lognormal probability density function (PDF). The median and 90th percentile lead concentrations of the 8,514 samples were 43 and 171 ppm, respectively. The 95th percentile predicted from a best-fit lognormal PDF is 253 ppm; this appears to match the 95th percentile estimated from Figure 14 of the Gott and Cathrall (1980) report, which shows a log-probability plot of what appear to be subsample means of the soil data.

Gott and Cathrall (1980) also presented the soil data as a log-probability plot (Figure 14 of their report). The plot was truncated and presented as two lines which intersected at approximately 60 ppm. The 60 ppm intercept was interpreted by Gott and Cathrall (1980) to be an estimate of the *"threshold between background and anomalously high values"*, attributable to the influence of mineral belts containing high lead concentrations".

The Gott and Cathrall (1980) study provides the largest single data set of soil lead concentrations from which background lead concentrations for the Coeur d'Alene Basin can be estimated. A reasonable estimate of soil lead background based on this data is 43-253 ppm (the 50th-95th percentile range). A major uncertainty in background estimates derived from this data set is the degree to which the samples represent soils that have not been impacted by local anthropogenic sources of lead. Gott and Cathrall (1980) refer to *"special test studies"* that were performed that *"indicated that soil samples collected in the district at depths greater than about 6 inches...below the A soil horizon are free from contamination except in the vicinity of the smelter at Kellogg"*; these tests are not described further in the report. Also cited in support of the 6-inch limit of the impacted zone is a study by Canney (1959). Canney (1959) notes that *serious* lead contamination below 6 inches was rare in collected samples, however, samples should be collected at a depth of at least 10 inches to ensure collection of uncontaminated samples. He also states that lead contamination at depths greater than 6 inches observed at some locations may be due to high permeability soils that allow the mechanical transport of lead in suspension (Canney, 1959).

Gott and Cathrall (1980) also reported lead concentrations measured in rock from the same sites where soils samples were collected; these are shown in Table 1. The median and 90th percentile values were 19 and 140 ppm, respectively. Thus, lead enrichment of soils with respect to adjacent or underlying rock was relatively minor; this is consistent with the soil samples having been relatively free of impacts from local anthropogenic sources.

In as much as the purpose of the Gott and Cathrall investigation was to identify unexplored areas that may contain mineral deposits, including lead ores, the use of the percentiles reported by Gott and Cathrall may result in a biased high estimate of the regional background soil lead concentration.

5.1.2 Hagler-Bailly Consulting (1995)

The Natural Resources Injury Assessment conducted by Hagler-Bailly Consulting (HBC, 1995) included the collection of *background* soil samples. Samples were collected along Canyon Creek near Sawmill Gulch, from the lower section of the West Fork of Ninemile Creek and from the North Fork of the Coeur d'Alene River. All samples were collected within the floodplains of the respective water bodies. Sample locations were determined by random (Canyon, Ninemile Creek samples) or stratified-random (North Fork samples) methods. Five samples were collected from each site and combined into 1 liter composite samples. Samples were collected from 17 sites along the North Fork, three sites along Canyon Creek and three sites along Ninemile Creek. The depth from which the samples were collected is not stated in the report.

Samples collected along Ninemile and Canyon Creeks were sieved in the field to remove the greater than 6-mm fraction. All samples were dry sieved in the lab prior to analysis to remove the greater than 2-mm fraction. Lead concentration was determined by inductively coupled plasma following Contract Lab Procedures. The detection limit was not specified. Samples were prepared by nitric acid extraction.

The arithmetic mean lead concentration for the Canyon Creek, Ninemile Creek and North Fork background sites were 802 ppm, 174 ppm and 16.8 ppm, respectively; the maximum values were 1,040, 323, 33 ppm, respectively. Based on a comparison to the Gott and Cathrall (1980) data and field observations made at the Canyon Creek collection sites (soil texture/structure and vegetation), Hagler-Bailly Consulting (1995) concluded that the data from the Canyon Creek sites did not represent background concentrations, while data from the North Fork and Ninemile Creek sites did represent background. The mean lead concentrations for the Ninemile Creek and North Fork sites correspond approximately to the 90th and 20th percentiles from Gott and Cathrall (1980), respectively. The Gott and Cathrall (1980) report did not include raw data that would allow more detailed comparisons.

Hagler-Bailly (1995) also used a multivariate statistical technique, principal components analysis (PCA), to delineate background sites from contaminated sites. The data set consisted of the concentrations of the seven metals arsenic, cadmium, copper, iron, manganese, lead and zinc. A scatter plot of these data would be seven-dimensional, with one axis representing each of the metals. It is difficult, if not impossible, to construct a meaningful representation of the data in seven dimensions so some means of reducing the number of dimensions is required. PCA is a technique in which some cases results in a reduction in dimensionality by exploiting the intercorrelations between the dimensions. PCA involves a rigid rotation of the axes so that they orient with the data. This results in a new set of variables, the principal components, equal in number to the number of variables in the original data set. The principal components are uncorrelated and have the property that the sum of their variances is equal to the sum of the variances of the original variables. Furthermore, the principal components are extracted in order according to their variance, with the most variable being first. Thus if the first few principal components account for the majority of the variability in the original variables, the remaining principal components may be ignored, resulting in a reduction in dimensionality.

Hagler-Bailly selected the first two principal components, dropping the remaining five. Unfortunately, they did not indicate what portion of the total variance these two components represent. Furthermore, they first standardized the original variables so that each had a mean of zero and a variance of one. It is customary to standardize when the original variables are expressed in disparate units, but in this application all seven variables are concentrations with the same units. Transforming the concentrations so that the mean for each of the seven metals is zero and the variance is one results in an unnecessary loss of information. Finally, the approach used by Hagler-Bailly to identify the principal components is not clear. They indicate the identification of the first principal component as "increasing concentration" and the second principal component as "increasing divergence" is based on the fact that all coefficients for the first were positive while for the second some were positive and some were negative. It is not clear whether they expressed those coefficients as the correlations between the principal components and the original variables. For example, a simple scaling converts the coefficient of arsenic in the first principal component to the correlation between arsenic and that principal component. Thus the actual magnitude (as well as the direction positive or negative) of the contribution of each of the metals to the principal components can be evaluated.

An uncertainty with this approach arises from the interpretation of the principal components, specifically the second principal component. It is not clear why the divergence of metal concentrations is associated with impacted soil. The conclusion from this analysis was that the North Fork and Ninemile Creek samples appeared to represent background concentrations while the Canyon Creek samples did not. The study also detected significant differences in soil texture, organic content, phosphorus or pH between most of the background sites and impacted sites.

Hagler-Bailly (1995) proposed the 90th percentile of the Gott and Cathrall (1980) data, 171 ppm, as a conservative, upper bound estimate of background. A histogram included in the Hagler-Bailly (1995) report (Figure 3-16) indicates that 5 of the 23 observations exceed 171 ppm. Four of them were from the Canyon Creek sites and one was from a Ninemile Creek site (323 ppm).

The background sites in the Hagler-Bailly 1995 study were located upstream of known releases of mining waste. However, as shown by the Canyon Creek site results, the sites may be impacted by unknown sources of contamination. Another source of uncertainty is the lack of knowledge of the sampling depth. Although the North Fork and Ninemile Creek sites may be not be impacted by upstream mining sites, they may be impacted by contamination from the smelter or other unidentified sources.

5.1.3 Reece et al. (1978)

Reece et al. (1978) collected sediment samples from the North and South Fork and the main stem of the Coeur d'Alene River to investigate the effects of contaminated sediments on water quality. Samples were collected from the top 3 inches of sediment and analyzed by atomic absorption spectroscopy. Background sediment samples were collected from the North Fork and the St. Joe River; the latter empties into the southern portion of Coeur d'Alene Lake. Lead concentrations in samples collected from the North Fork ranged from 26 to 200 ppm with an arithmetic mean of 79 ppm. Samples collected from the South Fork near Mullan, which, according to the author, is upgradient of the intensively mined areas, had a concentration range of 50-596 ppm with an arithmetic mean of 202 ppm. Lead concentrations observed in the sediment samples collected from the St Joe River ranged from 3 to13 ppm. The relatively low lead concentrations observed in the St. Joe River sediments may indicate a difference in the geology of the St. Joe watershed and the geology of the Coeur d'Alene River Basin (i.e., with respect to mineral deposits). Therefore, the regional background for the St. Joe River sediments may be lower than the regional background for the Coeur d'Alene River Basin sediments.

The lead concentrations in the St. Joe sediments may represent a lower bound on the estimate of the regional background for sediment. The lack of knowledge regarding the geology of the St. Joe Drainage Basin results in some uncertainty in the use of the data to estimate the regional background concentration for the Coeur d'Alene River Basin. If the Mullan samples were collected from areas upgradient of the intensively mined areas, the range of concentrations reported for Mullan probably represent a lower bound of the lead concentration in contaminated sediments.

Hoffman et al. (1992)

Hoffman et al. (1992) collected sediment samples from five of the lateral lakes: Killarney, Medicine, Swan, Thompson and Bull Run. Background samples were also collected from Bells Lake, which is located in the St. Joe River Basin. Previous investigations cited by Hoffman et al. (1992) identified 6,700 year old volcanic ash from Mt. Mazama (Crater Lake) in the region. Using the volcanic ash as an indicator of the age of the sediments, Hoffman et al., also collected samples from sediments in Killarney Lake and Bells Lake to compare pre-mining lead concentrations with post-mining concentrations. Samples underwent nitric acid digestion followed by atomic absorption spectroscopy (Killarney, Swan and Bull Run) or inductively coupled plasma (Medicine and Thompson). Unfortunately, the results are provided as a series of graphs from which an estimate of background concentration can not be made.

5.1.5 URSG/CH2M Hill (1998)

In November and December of 1997, URS Greiner (URSG) and CH2M Hill collected sediment samples from the Lower Coeur d'Alene River Basin. Samples were collected along 10 transects within the main stem and floodplain of the lower Coeur d'Alene River (CDR) and four of the lateral lakes (Cave, Medicine, Killarney and Rose). The samples were collected to determine the vertical and lateral extent of contaminated sediments. The locations of the main stem cores were based on the erosion/deposition characteristics of the river channel. Lateral lake sediment cores were located in areas where previous sampling had shown high concentrations of contamination. Floodplain cores were collected from both sides of the river, at distances of 50, 100, 200, 300, and 400 meters from the river bank. At four of the six floodplain transects, some cores were not collected due to site inaccessibility.

Cores obtained from the main stem were divided into 1-meter segments. A sample from each segment was sent to the laboratory for analysis. Cores obtained from the floodplain and lakes were field-screened to delineate mining-impacted sediment from non-impacted sediment. A field test kit with an approximate detection limit of 20 ppm was used to delineate impacted sediments from non-impacted sediments. All sediments above 20 ppm were considered to be *potentially impacted*. Two samples of the *impacted sediment* (if sufficient material was present) were sent to the laboratory for analysis. Of the two samples collected from the *impacted sediments*, one was obtained from the top half of the impacted sediment interval and one was obtained from the bottom half of the impacted interval. One sample of the unimpacted sediment (based on the results of the field screening) was sent to the laboratory for analysis. Lead concentrations were determined in accordance with CLP methodology (ILMO 4.0).

Figure 1 shows a probability plot of the log-transformed concentrations from 283 core samples. The plot can be represented by two lines representing distinct log-normal distributions, a *high* lead distribution and a *low* lead distribution, joined by a transition zone. To estimate the parameters of the *low* lead distribution, the data set was truncated in the transition zone and a probability plot of the data to the left of the truncation point was constructed. The parameters of the lognormal PDF were estimated from the slope and intercept of the best fit line. Figures 2-4

show the probability plots and estimated distribution parameters resulting from three different truncation points shown in Figure 1; percentiles are shown in Table 2.

The URSG/CH2M Hill (1998) study provides the largest single data set of sediment lead concentrations in the Coeur d'Alene Basin. The data set can be used to estimate background concentrations of lead in soil if several assumptions are accepted: 1) sediment lead concentrations reflect soil lead concentrations; 2) the *low* distribution shown in Figure 1 represents lead background concentrations; 3) the parameters of the *low* distribution can be estimated from the sample. Given the above assumptions, a reasonable estimate of soil lead background based on this data is 42-116 ppm (the 95th percentile range resulting from truncations A-C).

The major uncertainties in background estimates derived from this data set relate to the above three assumptions. With respect to the first assumption, typically sediment and soil are treated as two different media, with a local background concentration estimated for each. It is reasonable to assume that the soil located within the flood plain of the Coeur d'Alene River consists primarily of sediment deposited by the river. Therefore, for areas located within the floodplain, the use of sediment data in formulating an estimate of local background appears to be reasonable. Soils in the upland areas, however, have been formed in environments significantly different than soils in the areas located within the floodplain. Therefore, the use of sediment data to estimate local background concentrations in the upland areas introduces additional uncertainty.

Regarding the second assumption, probability plots were used to delineate impacted sediments from non-impacted sediments. While the plots do indicate the presence of two populations, this is a statistical approach that does not take the location of the samples into consideration. It is possible that the two populations represent two populations of regional background concentrations. The high concentrations may be associated with sediments derived from ore-bearing rock that has entered the river environment due to natural erosion mechanisms such as landslides and runoff. The level of uncertainty associated with this approach may be reduced by locating the samples in each population on a map. Visual inspection and statistical analysis of such a map would either increase or decrease confidence in the assumption that the two populations represent impacted and non-impacted sediments. Another source of uncertainty arises from the selection of the truncation point. As shown in Table 2, the selection of the truncation point has a major effect on the estimated parameters of the distribution.

A third source of uncertainty is the use of the sample data to estimate population parameters. This source of uncertainty is not unique to the method discussed above to estimate local background concentration. Estimates of population parameters using sample data contain some degree of uncertainty depending upon the number of observations included in the data set, the variability of the data and how well the data fits the distribution chosen to model the data. The number of samples used to estimate the population parameters varied from 77 to 115, depending upon the truncation point selected (A-C). In all three cases the number of samples should be adequate to calculate a reasonable estimate of the population parameters. The variability of the sample data represents sampling, handling and analytical errors as well as the true variation of lead in the sediment. The coefficient of variation ranges from 0.31 to 0.95 indicating the data are

moderately variable. The probability plots shown in Figures 2-4 indicate the data fits a lognormal distribution reasonably well. The Shapiro-Wilk goodness-of-fit test, however, indicates the data does not fit a lognormal distribution ($p = 0.0001$ for all three truncation points; H_0: data are log-normally distributed). Visual inspection of the histograms corresponding to the three truncation points also does not indicate the data are log-normally distributed.

An attempt was made to use the approach described above to estimate the background lead concentration in soil samples collected from the common use areas in the Coeur d'Alene Basin. The probability plot did not indicate the presence of two populations.

5.2 Studies Performed Outside of the Coeur d'Alene Basin

Other efforts to determine typical background concentrations for inorganics are summarized below. The summary is intended to provide additional context that will be useful for estimating the natural background soil lead concentration.

5.2.1 Shacklette and Boerngen. (1984)

Shacklette and Boerngen (1984) collected data on the distribution of element concentrations throughout the United States. The purpose of their efforts was to provide estimates of the ranges of naturally occurring element concentrations in surficial materials. A total of 1218 sites were sampled between 1961 and 1975. Samples were collected along roadways, at approximately 8 inches (20 cm) below the ground, if sufficient material was present. An effort was made to collect samples from sites that represented unaltered, natural conditions, although in some cases, samples were collected from cultivated fields. Sample locations were approximately located at 50-mile intervals. Samples were shipped in paper envelopes to the USGS laboratory, which could have resulted in sample contamination during shipment to the laboratory.

All samples were sieved to remove the greater than 2-mm fraction. The less than 2-mm fraction was analyzed by an emission spectrographic method. The approximate detection limit for lead was 10 ppm. The study reported a range of arithmetic mean lead concentrations of 17-20 ppm and a geometric mean of 16 ppm. The higher arithmetic mean represents the western United States; the range of observations in the western United States was less than10-700 ppm.

Although an effort was made to collect samples from 8 inches below the ground surface to avoid anthropogenic contamination, the authors acknowledged that they "had no adequate way of measuring any contamination that may have occurred" (Shacklette and Boerngen, 1984). Therefore, the values reported may be biased high. The Shacklette and Boerngen data suggests an arithmetic mean regional background concentration of 20 ppm. Unfortunately, the report does not provide other statistics of the sample distribution such as the median and percentiles. The mean reported by Shacklette and Boerngen may be considered an estimate of the lower bound of the mean regional background soil lead concentration for the Coeur d'Alene River Basin. The mean regional background soil lead concentration for the Coeur d'Alene River Basin is likely to be higher than the mean for the entire western United States because of the presence of lead ore in the basin.

5.2.2 Washington State (1994)

A report on the natural background concentrations of metals in soils in Washington State was prepared by Charles San Juan of the Washington State Department of Ecology, Toxics Cleanup Program. (WSDE, 1994) The state was divided into 24 regions based on geology, soils and climate. The twelve regions with the greatest industrial development were included in the study. A total of 166 soil samples were collected from the A, B and C soil horizons at locations throughout the 12 regions between 1987 and 1993. Total metals concentration of the less than 2-mm fraction of the samples was determined by inductively coupled plasma (ICP). The concentrations measured at each site (including splits and duplicates) were averaged.

Lead was detected in 151 of the 166 samples analyzed for lead content. The report indicates that the detection limits for the samples with non-detects ranged from 2 to 4 ppm. Based on statistics provided in the report, the statewide mean lead concentration is 10 ppm and the range is 2-208 ppm. The report did not include all of the lead data for the entire state, therefore estimates for the percentiles not provided in the report could not be calculated. The mean lead concentration for the Spokane Basin is 11 ppm and the range is 7-16 ppm. Data for the Spokane basin was included in the report and is summarized in Table 3. Note that the estimated 90th percentile in Table 3 does not match the estimate provided in the WDOE report, although the values are similar. The difference may be due to rounding error or the use of different methods to estimate the percentiles.

The concentrations reported by the Washington State Department of Ecology are considerably lower than those reported by Shacklette and Boerngen (1984). It is likely that the estimates provided in Table 3 are lower than the local background concentration for the Coeur d'Alene River Basin due to the presence of lead ore in the basin. The data included in the Washington report represents the lead concentration in three soil horizons. The variation of regional background soil lead concentration with depth is unknown. This introduces some uncertainty into the use of the Washington State data to develop an estimate of the local background soil lead concentration for the Coeur d'Alene Basin.

5.2.3 Bloom and Crecelius (1987)

Bloom and Crecelius (1987) measured concentrations of heavy metals in the Puget Sound. Samples were collected along four transects which were located to ensure samples of various grain sizes were collected. Samples were analyzed by atomic absorption spectroscopy. Lead concentration in surface sediments (0-5 cm) ranged from 13-53 ppm. Based on complete data provided for four cores, the maximum concentration observed was 65 ppm. The highest lead concentrations were found in fine-grained particles. The Bloom and Crecelius data indicates that lead concentration may be a function of grain size. Thus, the grain size distribution of soil and sediment samples is another source of uncertainty in estimating background concentration. This source of uncertainty can be limited with consistent sample preparation (i.e., sieving) prior to analysis. Not enough information is available about the origin of the sediments sampled by

Bloom and Crecelius for the analytical results to be used towards developing an estimate of regional background lead concentration for the Coeur d'Alene River Basin.

5.2.4 McGovern (ND)

In an unpublished report, McGovern (ND) provides a summary of 12 studies that investigated the natural level of inorganics present in soils in the United States and Canada. The reported range of concentrations was 2-200 ppm. The arithmetic mean concentration of lead ranged from 10 to 24 ppm.

5.3 Conclusions from Soil and Sediment Sampling

Figure 5 shows the range of estimates of regional background soil lead concentrations in the Coeur d'Alene Basin derived from various studies performed in the basin. The estimates shown in the figure were either as reported or were estimated from data provided in the reports. The figure includes the studies considered to be most relevant to the estimation of regional background soil concentration on the site(s).

Central tendency estimates (arithmetic mean or 50th percentiles) from the above studies range from 17 ppm (Hagler-Bailly, 1995) to 43 ppm (Gott and Cathrall, 1980). The upper bound estimates (maximum or 95th percentiles) range from 33ppm (Hagler-Bailly, 1995) to 253 ppm (Gott and Cathrall, 1980). The estimates from Gott and Cathrall (1980) are probably the most robust in that the study represents a large number of soil samples collected from a broad area within the basin.

6.0 NON-MINING/SMELTING ANTHROPOGENIC SOURCES OF LEAD

6.1 Industrial Sources - General

Both natural and anthropogenic sources of lead can be expected to contribute to the background lead concentrations in the Coeur d'Alene Basin. A search of the TRI and AIRS databases did not locate any sources of lead emissions. Power plants are a potentially significant source of lead through the combustion of oil. However, 1998 will be the first year they will be required to report their emissions under Title III, Section 313 of the Superfund Amendments and Reauthorization Act of 1986.

6.2 Automobiles

Studies performed in the late 1970's and early 1980's found that more than 80% of the total lead emissions in the United States were from motor vehicles. Approximately 90% of the lead was emitted as inorganic particles with the remainder emitted as volatile organic compounds (lead alkyls). Approximately 35% of the emitted lead particles are classified as small (less than 0.25 MMAD [Mass Median Aerodynamic Diameter]) and 40% are classified as large (EPA, 1986).

Small particles may stay in the atmosphere for days and travel thousands of miles from the point of release and thus become part of the global contributions to background. Large particles tend to travel shorter distances, contributing to local background lead levels. The concentration of lead particles in air next to roadways has been found to be a function of windspeed and direction, temperature and inversion frequencies and duration. The organic lead vapors are photoreactive, with a half-life of 12 hours under summertime conditions. Studies have shown that they contributed less than 10% to the total lead present in the atmosphere. (EPA, 1986)

Lead particles released from automobiles are transferred to the ground surface by dry and wet deposition. The rate of dry deposition depends upon the size of the particles, wind speed and type and amount of vegetative cover. Wet deposition removes lead particles from the air during formation of the rain droplets (rainout) or when the raindrops are falling to the ground (washout). The relative contributions of wet and dry deposition vary on a seasonal basis. (EPA, 1986)

Near roadsides, the primary mechanism of transfer is dry deposition. One study found that approximately 54% of the particles (by weight) are deposited near the roadside (Wheeler and Rolfe, 1979). Soil lead concentrations decrease rapidly within 10-25 meters of the road edge Agrawal et al., 1981; EPA, 1986; Motto et al., 1970; Ward et al., 1975; Wheeler and Rolfe, 1979; Wong and Tam, 1978). The increment in soil lead concentration resulting from motor vehicles has been estimated to range from 10 to 2000 ppm. (EPA, 1986, 1989)

The decline in use of leaded gasoline started around 1973 due to EPA regulations promulgated that year. By 1984, the use of lead alkyl additives in gasoline in the United States declined by a factor of three over 1973 levels. EPA ambient air monitoring data indicated the 73% reduction in leaded gasoline combustion between 1975 and 1984 coincided with a 71% reduction in ambient air lead concentrations. (EPA, 1986)

Although the combustion of leaded gasoline in automobiles has been phased out, the amount of lead in soil due to past atmospheric contributions has likely remained approximately constant due to its low mobility (EPA, 1986, 1989; Lovering, 1976). Studies conducted along roadways have consistently shown elevated lead concentrations are restricted to shallow soil depths (Agrawal et al., 1981; Chaney et al., 1988; Motto et al., 1970).

Only one study has attempted to compare residential soil lead concentrations and adjacent or nearby roadside concentration in the Coeur d'Alene Basin (Terragraphics, 1990), however, the study is not particularly relevant to estimating local background because the roadside areas sampled are expected to have been impacted by mining and smelting related operations, including vehicular transport of ore. Numerous studies conducted outside of the Coeur d'Alene Basin have explored the relationship between soil lead concentrations and automobile emissions. Most of these studies were conducted before or shortly after the phase out of leaded gasoline began, thus, the soil lead concentrations reported may be greater than current automobile-related concentrations. Nevertheless, these studies were conducted in areas that were not impacted by mining and smelting operations and, therefore, provide some useful information on the potential magnitude of automobile emissions on local background in the Basin, although quantitative estimates for the Basin can not be made from these studies.

6.2.1 Studies Performed Within the Basin

6.2.1.1 Terragraphics (1990)

The Risk Assessment Data Evaluation Report for the Populated Areas of the Bunker Hill Superfund Site (RADER) (Terragraphics, 1990) included data on lead concentrations in residential yards and along roadways located in the same communities. An attempt is made here to compare the two sets of data to determine elevated concentrations of soil lead due to automobile emissions.

The RADER included a summary of the analytical results of 25 samples collected in 1989 from curbless road shoulders. Sampling was performed as part of the Phase II Remedial Investigation (RI). The roads were located within the project area, in the towns of Smelterville, the Sunnyside and Old town areas of Kellogg, Warder, Pinehurst, Page and Elizabeth Park. The method used to determine sample location was not specified. Samples were collected from the top one-inch of the soil profile. It is assumed that the mineral soil was sampled, not the litter material (decaying vegetative matter and sod). Analytical method and detection limits were not specified although Contract Laboratory Protocol procedures reportedly were followed.

Lead concentrations ranged from 249 to 60,100 ppm, with mean and median values of 4743 and 1580, respectively. Observed concentrations varied considerably both within and between towns. The highest arithmetic mean/median concentrations were observed in Smelterville (14,150/5970), followed by the Old Town (4497/4070) and Sunnyside (1935/1935) areas of Kellogg. The results are summarized in Table 4.

Residential yards were sampled during the Phase I and II RI efforts. The Phase I sampling occurred in 1986-1987 and included the communities of Smelterville, Kellogg, Warder and Page. Phase II sampling occurred in 1989 and included Pinehurst and Elizabeth Park. An attempt was made to collect samples from every home during the phase I sampling event. Approximately 66% (1,020) of the homes were sampled. The percentage of homes sampled ranged from 62.9% for Kellogg to 78.5% for Page. Samples were not collected when homeowners either refused permission to collect samples, the homeowner could not be contacted or the home was vacant. Results may be biased due to non-random sampling method. Two eight-point composite samples were collected from each residential yard. One composite sample was collected from the top one inch of litter and the other consisted of the top one inch of mineral soil. Analytical method and detection limits were not specified although Contract Laboratory Protocol procedures reportedly were followed.

A total of 1113 mineral soil samples were collected. Analysis of the mineral soil samples showed the arithmetic mean/median concentrations for the four communities ranged from 3,580/3010 at Kellogg to 1,090/810 at Page. The results are summarized in Table 4.

The Phase II sampling RI effort included collecting soil samples from residential yards in Pinehurst and Elizabeth Park. Details of the sampling and analytical methods were not provided

in the RADER. Samples were collected from the yards of 127 residences. These results are also summarized in Table 4.

Table 4 does not include the highest observation (60,100 ppm) recorded for the Smelterville road shoulder data. The highest observation was 6 times greater than the next highest observation. The arithmetic mean/median concentrations for Smelterville are 14,150/5970 with the highest observation included. The totals for the roadside data with the highest observation for Smelterville included are 4,743/1,580. This results in the difference between the totals for residential and roadside means/medians equal to 2,912/81.

The above analysis suggests there is a difference between the mean for the road shoulder data and the residential data. With the highest road shoulder observation included in the comparison, the difference between the two sets of data is even greater. However, when the highest observation is removed, the median for the road shoulder data is less than the median of the residential data. Also surprising is the data for Warder, Page and Elizabeth Park that indicates the road shoulder soil contains less lead than the residential soil.

The above summary does not provide any clear conclusions regarding the contribution of automobiles to the local background soil lead level. The difference between arithmetic mean roadside and residential lead concentrations are greatest in the communities that are the closest to the former smelter location. Some of the apparently anomalous results may be due to small sample sizes for the road shoulder data, non-random sampling techniques, possibly inconsistent sampling and analysis between the three sets of data and the time interval between the sampling events.6.2.2 Studies Outside of the Coeur d'Alene Basin

The following studies are a sample of the available information on the contribution of automobiles to local background soil lead concentrations. The studies are located in areas of varied urbanization, from remote locations to highly developed areas. The purpose of including the selected studies in this report is to provide the reader with information about the potential range of the automotive contributions to local background lead concentration. The potential contribution of automobile emissions to the local background lead concentrations is likely to vary considerably within the Coeur d'Alene Basin. In areas near Interstate 90, the automotive contributions will tend to be high while in remote areas such as some of the common use areas located in the lower basin, the contribution is likely to be much lower. Uncertainties associated with the use of the data presented in the following reports to estimate the automotive contribution to local background concentration in the Coeur d'Alene Basin are discussed in the Section 6.2.3.

6.2.2.1 Wheeler and Rolfe (1979)

Wheeler and Rolfe (1979) investigated the relationship between average daily traffic (ADT) (vehicles per day) and lead concentrations in roadside soils and vegetation in central Illinois. Four highways located in rural areas were included in the study. The ADTs for the highways ranged from 550 to 8100. Soil samples were collected from 0 to10 cm below the ground surface and from varying distances from the roadway. The samples were dried and ground to pass a 2-mm sieve, then analyzed by atomic absorption spectroscopy.

Results of the study indicated the soil lead concentrations decreased exponentially with increased distance from the roadway and were reduced to *background concentrations* within 25-200 meters from the road surface, depending upon the ADT. *Background concentration* is defined here as the concentration of lead which was approached asymptotically with increased distance from the roadway. A non-linear regression equation used to predict soil lead distribution included two exponent terms. Wheeler and Rolfe (1979) hypothesized that the first term characterized the rapid deposition of large particles within 5 meters of the roadway and the second term represented the slower deposition of smaller particles within 100 meters. Linear regression was used to estimate the relationship between ADT and soil lead concentration. The increment in soil lead concentration above background ranged from 11 to1,211 ppm, depending upon the ADT and distance from the roadway. For ADTs less than 1500, the background concentration was observed at approximately 25 meters from the edge of the road. At 5 meters from the edge of the road, the concentration increment was 31 and 21 for ADTs equal to 1500 and 550, respectively.

The findings of this study show the automotive contribution to local background concentration varies considerably with traffic volume as well as distance from the roadway. Similarly, the potential automotive contribution to local background lead concentration in the Coeur d'Alene Basin may vary with the location of the sites relative to roadways and the volume of traffic on those roadways.

6.2.2.2 Agrawal et al. (1981)

Agrawal et al., (1981) investigated the relationship between soil lead concentration and distance from a roadway. The study area was located next to a national highway (ADT = 17,000) in Baroda, India. Samples were collected from varying depths between 0.5-10 cm below ground surface and at varying distances from the roadway. Samples were dried and sieved to remove the greater than 60-mesh portion. Analysis was performed by atomic absorption spectroscopy. Arithmetic mean increments above background were: 272 ppm at the roadside edge; 237 at 5 meters from the roadway; 117 at 10 meters; 82 at 20 meters and 6 ppm at 80 meters from the roadway. Background was defined by the author as the lead concentration observed at 300 meters from the roadway (3 ppm). Lead concentration was found to decrease rapidly with depth. At a depth of 10 cm, the concentration increment at 20 meters from the roadway was 17 ppm, at 40 meters, the background concentration was observed.

This study provides additional information on the contribution of automobiles to local background in areas adjacent to high volume roadways such as Interstate 90. The study found that even at high traffic volumes, automotive contributions are limited to within 100 meters of the roadway.

6.2.2.3 Wong and Tam (1978)

Wong and Tam (1978) investigated elevated lead concentrations in soil alongside three major roadways in Hong Kong. The study included seven sites located along roadways that connect major cities and a control site located approximately 200 meters from any roadway. Soil samples

were collected from 0to10 cm below the ground surface. Samples were air dried and sieved to remove the greater than 2-mm portion prior to analysis by atomic absorption spectroscopy. Increments in soil lead concentrations above background (defined as the lead concentration observed in the soil at the control site: 26.2 ppm) were: 54 ppm at the roadside edge; approximately 49 ppm at 5 meters (interpolated from graph); 48 ppm at 15 meters; and 3 ppm at 75 meters from the roadway.

This study also provides information regarding the automotive contribution to local background soil lead concentration in areas adjacent to a major roadway such as Interstate 90.

6.2.2.4 Motto et al. (1970)

Motto et al., (1970) investigated the relationship between soil lead concentration and traffic volume and distance from highways in northeastern New Jersey. Soil samples were collected from thirteen sites with land use that ranged from rural to industrial. ADTs ranged from 12,800 to 54,700 vehicles per day. Samples were collected from two depth intervals: 0-6 and 6-12 inches below ground surface. The samples were air dried and ground to pass a 200-mesh screen prior to analysis by atomic absorption spectroscopy. A separate background concentration was defined for each site as the lead concentration observed at 225 feet from the edge of the roadway. The background concentrations varied widely between the sites. Motto et al. (1970) attributed this to varying geologic factors and historical and current land uses. In general, the sites are located in a highly industrialized region of the country.

The soil data indicated that lead concentrations were above background within 100 feet of the roadways. Within 25 feet of the roadway, the concentration increment at 0-6" depth ranged from 25 to 402 ppm, with an arithmetic mean of 160 ppm. Within 75 feet of the roadway, the elevated lead concentrations ranged from 3 to 93 ppm. At some sites, the soil lead concentration increased with distance from the road edge, indicating soil in the area had been moved during site work. A clear relationship between traffic volume and elevated lead concentrations was also not indicated by the data. The authors attribute this to the history of the sites, which includes soil removal, filling and grading. The soil lead concentrations in the 0-6 inch depth interval were significantly greater than those observed in the 6-12 inch interval.

This study provides information on the lead concentrations observed in soils adjacent to major roadways such as Interstate 90. The study also illustrates the confounding effects of historical site uses, including soil removal and emplacement, on the determination of local background soil lead concentration.

6.2.2.5 Ward et al. (1975)

Ward et al. (1975) investigated the accumulation of lead in soils adjacent to a highway in New Zealand in a remote area characterized by low traffic volume (ADT = 1,200). The site was located far from any urban or industrial areas, farmland or other highways. Soil cores were collected from the top 6 cm of soil at approximately 5-10 meter intervals along three transects oriented perpendicular to the highway. Cores located within 3 meters of the roadway were

collected to a depth of 10 cm. Soil cores were divided into 1 cm intervals prior to analysis. All samples were air dried, sieved to remove the less than 60-mesh portion and analyzed by atomic absorption spectroscopy.

A background concentration of 42 ppm was estimated by calculating the mean of 22 observations from soil samples collected at depths ≥ 7 cm within 100 m of the roadway and ≥ 3 cm at distances greater than 100 m. Arithmetic mean increments above background in the top 1 cm of soil were approximately 120 ppm within 10m of the roadway, 50 ppm within 30m and less than 20 ppm within 100 m. At 3 cm depth, arithmetic mean increments were approximately 35 ppm within 10 m, 20 ppm within 30m and at background within 100m (estimated from inspection of Figure 3 of Ward et al., 1975). A plot of arithmetic mean soil lead concentration vs depth (Figure 3 in the Ward et al. report), shows the concentration approaches background concentration rapidly with depth and the depth at which background is reached depends upon the distance from the roadway. At 100 m background was observed at a depth of 3 cm; at 10 m background was observed at 6 cm.

This study provides information on the potential automotive contributions to local background soil lead concentration in areas adjacent to roadways that handle low traffic volumes such as some of those found within the communities located in the basin. The study also provides additional information on the relationship between soil depth and lead concentration in areas adjacent to roadways.

6.2.2.6 Case et al. (1989)

Case et al. (1989) examined the impact of anthropogenic sources of lead, primarily vehicle emissions, on the lead concentrations in lake and pond sediments in Luzerne County, Pennsylvania. Samples collected from 15 lakes and ponds were analyzed by atomic absorption spectroscopy. The samples were collected from two depth intervals within the sediments: 0-3 cm and 12-15 cm. The concentrations in the 0-3 cm interval ranged from 85 to 407 ppm, while those in the 12-15 cm interval ranged from 55 to 291 ppm. Higher lead concentrations were observed in lakes and ponds that reportedly have high traffic volume along their perimeter roads. Estimates of the background concentrations were not provided in the report.

This study indicates that automotive contributions to local background sediment lead concentrations should be considered in the Coeur d'Alene Basin. The automotive contributions will likely be greater in sediments found in water bodies adjacent to Interstate 90 than in remote areas.

6.2.3 Conclusions Regarding Contributions of Automobile Emissions to Local Background Soil Lead Concentrations

Soil lead concentrations 30-2000 ppm above background have been attributed to automobile emissions (EPA, 1986). A review of the literature indicates that the elevated soil lead concentrations due to automobile emissions is highly variable and dependent on many factors including traffic volume and distance from the roadway. However, some general statements can

be made. Data from Wheeler and Rolfe (1979) and Ward et al. (1975) indicate that elevated soil lead concentrations in the vicinity of roads that handle low traffic volumes ranges from approximately 6-120 ppm at 10 meters from the roadway. Wheeler and Rolfe found elevated soil lead concentrations next to high volume roads (8100 ADT) ranged from 80 ppm at 5 meters from the road to 11 ppm at 25 meters. Agrawal et al. (1981) found elevated soil lead concentrations next to a highway with an ADT of 17,000 equal to 237 ppm at 5 meters from the road edge and 67 ppm at 25 meters. Motto et al. (1970) found elevated soil lead concentration adjacent to highways with ADTs of 17,300 and 17,700 equal to 154 and 442 ppm, respectively at 25 feet and 42 and 43 ppm, respectively at 75 feet.

Based on the above data it seems reasonable to suggest that soil at sites located near low volume roads may contain 6-120 ppm of lead over the regional background concentration and soils adjacent to high volume roadways may contain 11-442 ppm of lead above regional background concentration.

There is considerable uncertainty associated with the use of data from the investigations such as those summarized above to estimate the automotive contribution to local background lead concentrations in the Coeur d'Alene Basin. The uncertainty effects the validity of comparing data generated from one investigation to data generated by another investigation. The uncertainty also effects how data from investigations such as those summarized above can be used to develop an estimate of the automotive contribution to local background soil lead concentration. Sources of uncertainty include differences in the sampling and analysis methods employed in the investigations, atmospheric conditions at each site such as arithmetic mean wind speed and direction, driving conditions including acceleration/deceleration zones, type of vehicle traffic (i.e., trucks, buses, passenger cars) and drainage features that may effect the transport of lead emissions from the roadway to the adjacent soil.

With respect to sampling and analytical methods used in the above investigations, sources of uncertainty include the effect of the differences in the size fraction of the sample retained for analysis and the digestion procedures used. Wheeler and Rolfe and Motto et al. analyzed the less than 200-mesh portion of the sample while Ward et al. and Agrawal et al. analyzed the less than 60-mesh portion and Wong and Tam analyzed the less than 2-mm portion. Various acid mixtures were used in the digestive procedure including 1:1 mixture of nitric/perchloric acids (Agrawal et al., 1981) 1:1 nitric/hydrofluoric (Ward et al., 1975), 1:1 HCL/water (Wheeler and Rolfe, 1979) and 72% perchloric (Motto et al., 1970). Wong and Tam used a neutral solution of ammonium acetate. In all cases, soil lead concentrations were determined by atomic absorption spectroscopy. The effect of the digestion procedure and size fraction of the sample on the reported concentrations is not clear from the results however, it does appear that the values reported by Wong and Tam and Wheeler and Rolfe are significantly lower than the values reported by the other investigators.

Atmospheric conditions were not reported by the investigators but may have a significant effect on the distribution of lead in the soils adjacent to the roadways. Driving conditions were also not reported by the investigators. The fuel efficiency of the vehicles that use the roadway will have an impact on the quantity of lead emitted. Higher lead concentrations would likely be found in

soils adjacent to roadways with high truck volumes than those with low truck traffic. The investigators did not characterize the vehicle traffic other than provide the ADT. The investigators also did not note drainage features. The location of samples along drainage pathways that convey water from the road surface would result in higher concentrations than samples located in areas that did not receive runoff from the roadway.

6.3 Lead Based Paint

6.3.1 Relevant Literature

Paint samples collected by the Idaho Department of Health and Welfare revealed the presence of lead based paint on the exterior of 44% of assessed homes in the basin. Of the homes with lead-based paint, the condition of the paint on 39% was characterized as being in poor condition. (IDHW, 1998) Investigations on the contribution of lead-based paint to soil lead concentration in the basin have not been identified. To estimate the potential contribution of exterior lead-based paint on the lead concentration in residential yard soils, investigations performed outside of the basin were reviewed. The findings of the investigators are summarized below.

Hardy et al. (1971) found soil adjacent to two homes contained lead at 2,000-5,500 ppm. The authors also measured the soil lead concentration at varying distances from a barn painted with lead-based paint. Soil lead concentrations ranged from 2,000 ppm at the barn foundation to 570 ppm at 10 feet from the barn, 160 ppm at 20 feet and 60 ppm at 50 feet from the barn.

Chaney et al. (1988) collected samples at varying distances from three sides of a house with exterior lead-based paint. At 0-1 meter from the house, lead concentrations ranged from 1,050 to 44,700 ppm in the 0-5 cm depth interval and from 940 to 7,270 at the 10-15 cm depth interval. At 5 meters from the house the lead concentration ranged from 110to431 at the 0-5 cm depth interval and from 366 to 2020 at 5-10 cm below the ground surface.

As part of a larger study of soil lead concentration in the Minneapolis area, Mielke et al. (1983) looked at the relationship between soil lead concentration and painted homes in an inner city environment. Forty-four residences were split into two groups based on the type of construction. It was assumed that the old wood frame homes were painted with lead-based paint while brick, stucco and newly constructed homes were not. The soil lead concentrations adjacent to the wood frame homes ranged from 475 to 6,150 ppm with a median of 938 ppm. Soils adjacent to the homes constructed of brick or stucco contained lead concentrations from 130 to 1,840 ppm, with a median of 526 ppm. Based on this data, lead concentrations in soil adjacent to painted buildings appears to be greater than non-painted structures.

Mielke et al. (1983) also found the soil lead concentrations observed adjacent to the urban, non-painted buildings was greater than the soil lead observed in a residential area located away from the inner city. Soil lead in the non-urban area ranged from 13 to 848 ppm with a median of 48 ppm. In addition, the lead concentrations in samples collected along sidewalks in the urban neighborhoods ranged from 58 to 1290 ppm with a median of 290, substantially lower than the lead concentrations observed adjacent to homes in the same neighborhood that are not painted

with lead-based paint. The authors suggest that the elevated soil lead concentrations observed adjacent to the homes was partly due to lead based paint and partly due to the collection of lead dust and aerosols on the roofs and sides of the homes that are periodically transported to the soil during rain events (EPA, 1986, 1989; Mielke et al., 1983).

Ter Haar and Aronow (1974) collected samples around urban homes and farmhouses to determine the significant sources of elevated lead concentrations in soil adjacent to urban homes. Samples were collected from nine locations around 18 wood frame houses and 18 brick homes located in urban areas of Detroit. The analytical results showed that the arithmetic mean soil lead concentration was 2,010 ppm within 2 feet of painted homes and 468 ppm within 2 feet of brick homes. The arithmetic mean concentrations at 10 feet from the painted and brick homes were 436 and 178 ppm, respectively. Lead concentrations in soil adjacent to the curb were similar for both types of homes. Lead concentrations within 2, 10 and 20-ft from the farmhouses averaged 2,529, 609 and 209 ppm, respectively. The soil lead concentrations for the farmhouses are similar to the urban wood frame homes, with the exception of the sample collected at 20-ft from the home. This level is significantly lower than the concentration observed next to the curb in the urban environment.

Ter Haar and Aronow concluded that lead-based paint is the main source of soil lead and the contribution of automobile emissions is insignificant. The authors attribute the elevated lead concentrations within two feet of the brick homes to the painted exterior trim. However, as noted by Mielke et al. (1983), the elevated concentrations may also be due to the transport of dust and aerosols from the roof and sides of the home. Comparison of the lead concentration at 20-ft from the rural homes (209 ppm) to the lead concentrations observed in next to the curbs in the urban areas (592 ppm) also indicates a significant contribution from automobiles.

6.3.2 Conclusions Regarding Contributions of Lead Based Paint to Local Background Soil Lead Concentrations

Elevated soil lead concentrations as high as 2000 ppm have been associated with the use of lead-based paint on the exterior of buildings. Elevated soil lead concentrations reported in the literature varied widely. Hardy et al. (1971) reported soil adjacent to two homes and a barn contained lead at 2,000-5,500. Chaney et al. (1988) found lead concentrations ranged from 1,050 to 44,700 ppm within 1 meter of building foundations.

The difference between the median concentrations reported by Mielke et al. (1983) for urban wood frame homes and brick/stucco homes suggests an elevated soil lead concentration of approximately 400 ppm due to lead based paint. The data provided by Ter Haar and Aronow (1974) indicate a lead based paint contribution of approximately 350 ppm. However, as noted above, a portion of this elevated soil lead concentration is probably due to the transport of lead dust and aerosols from the sides and roofs of the homes to the soil during rain events. Data from Hardy et al. (1971) and Ter Haar and Aronow (1974) indicates elevated soil concentrations in rural areas of approximately 600 ppm at 10 feet from the structure and 160-200 ppm at 20 feet from the structure.

Similar to the problem of estimating contributions of automotive emissions to soil lead concentrations, it is difficult to estimate the contribution of lead based paint to soil lead concentrations. For instance, the maintenance of the structure painted with lead based paint would have a direct impact on the amount of lead chips in the soil. In addition, it is reasonable to assume that a significant amount of the lead found in soils adjacent to structures in the basin was transported from the roofs and sides of the structures by rain. Based on the literature, soil located next to structures painted with lead based paint may contain 100-2000 ppm.

The use of data from investigations such as those summarized above to estimate the exterior lead-based paint contribution to local background lead concentrations in the Coeur d'Alene Basin involves a significant amount of uncertainty. Sources of uncertainty include differences in the sampling and analysis methods employed in the investigations, degree of urbanization in the study areas, condition of the lead-based paint and drainage features that may effect the transport of lead away from the residences.

With respect to sampling and analytical methods used in the above investigations, sources of uncertainty include the effect of the differences in the size fraction of the sample retained for analysis and the sample digestion and analytical procedures used. Hardy et al. (1971) did not report the size fraction retained or the analytical methods used. Chaney et al. (1988) and Mielke et al. (1983) analyzed the less than 2-mm fraction. Ter Haar and Aronow (1974) did not report the size fraction analyzed. Sample digestion procedures appeared to be consistent. Mielke et al. and Chaney et al. used a 1M nitric acid solution. Ter Haar and Aronow used a "dilute" nitric acid solution of unspecified concentration. The studies cited above cover a broad range in the degree of urbanization. An effort was made to account for the contributions from non-lead-based paint sources (e.g., automobiles) however, separating the contributions from lead-based paint from other sources of lead could not be accomplished with a high degree of certainty. The condition of the lead-based paint was not taken into consideration in the above investigations. The condition of lead-containing paint would likely have a direct effect on the soil lead concentration near the structures. The investigators did not report on efforts taken in their sampling strategies to account for the potential impact of drainage patterns on soil lead concentrations. For example, soil samples collected from beneath or adjacent to roof gutter downspouts would likely yield biased results due to the transport of lead-containing aerosols and dusts from the roof of the residence.

7.0 GROUNDWATER AND SURFACE WATER

Two sources of information on lead concentration in groundwater in the Coeur d'Alene Basin are discussed in Section 7.1. Data from the USGS National Stream Water-Quality Monitoring Networks is discussed in Section 7.2. The reports provide some information upon which to base preliminary estimates of background lead concentration in surface water and groundwater within the basin.

7.1 Groundwater

7.1.1 Spruill (1992)

Spruill (1992) collected groundwater samples from four monitoring wells and one piezometer installed in sediments near Killarney Lake. Well screens were installed in valley sediments at depths ranging from 6 to 33 feet below the ground surface. One well volume was purged prior to sampling. Samples were collected with a Teflon bailer and filtered through a 0.45-micron filter. The analytical method was not specified.

Reportedly, monitoring wells 2A and 5, both of which had no detectable lead (<10 ug/L), represent groundwater that has not been impacted by mining waste. According to the authors, the lead concentrations observed in samples collected from monitoring wells 2B and 4, 30 and 300 ug/L respectively, indicate groundwater in the vicinity has been impacted by mining waste. Sediment removed from the borehole during the installation of monitoring wells 2B and 4 contained less than100 and 5,000 ppm lead, respectively.

7.1.2 Parliman et al. (1980)

Parliman et al. (1980) conducted a survey of the groundwater quality in northern Idaho. Between July 1977 and May 1978, groundwater samples were collected from 116 existing, privately owned wells. The sampling area covered from the Palouse River to the Canadian border, which includes Kootenai and Shoshone Counties. Seven wells located in Shoshone County, along the main stem of the Coeur d'Alene, North Fork Coeur d'Alene and South Fork Coeur d'Alene Rivers were analyzed for trace metals including lead. The wells obtained water from various depths ranging from12 to 85 feet below the ground surface and from various geologic materials. Dissolved lead ranged from 5 to 15 µg/l. The highest concentration (15µg/l) was observed east of Wallace, upstream of the high intensity mining area. The well located on the North Fork contained 7 µg/l.

7.1.3 Prior Bunker Hill Risk Assessments for Populated Areas.

The Human Health Risk Assessment Protocol for the Populated Areas (Jacobs, 1989) and the RADER for the Populated Areas (TerraGraphics, 1990) used the mean of the seven wells located in Shoshone County (8.5 µg/l) as the background concentration. The Human Health Risk Assessment for the Non-Populated Areas (SAIC, 1991) used a background concentration range of 5-15 ppm. While no reference was provided, it appears they used the data from Parliman et al. as well.

7.2 Surface Water

Dissolved lead is typically found at low concentrations in water due to its tendency to form low solubility compounds with inorganic and organic material present (Lovering, 1976; Spruill, 1992). Lovering suggests a median concentration of lead in surface water in the U.S. of 2 µg/L (Lovering, 1976).

7.2.1 USGS National Stream Water-Quality Monitoring Data

Data obtained from the USGS National Stream Water-Quality Monitoring Networks (WQN) was used to estimate the background concentration of lead in surface water in the basin. Ten monitoring stations were identified in Idaho. The WQN includes data from 1967 to 1995, although not all of the 10 stations include data from each of those years. The data is summarized in Table 5.

7.2.2 Prior Bunker Hill Risk Assessments for Non-Populated Areas

The Human Health Risk Assessment for the Non-Populated Areas (SAIC, 1991) used a value of 19 µg/L as the background concentration but did not provide a reference. The Human Health Risk Assessment Protocol for the Populated Areas (Jacobs, 1989) and the RADER for the Populated Areas (TerraGraphics, 1990) did not provide a background lead concentration for surface water (the surface water pathway was not evaluated in these assessments).

7.3 Conclusions Regarding Background Groundwater and Surface Water Lead Concentrations

Based on the Parliman et al. and Spruill data, a reasonable range in background concentration for lead in groundwater is 5-15 ug/l. The uncertainty associated with this estimate is primarily due to the limited amount of data upon which the estimate is based. The data used to arrive at the above estimate was obtained from residences located along the Coeur d'Alene River and monitoring wells located near Killarney Lake. Uncertainty in the estimates is due to a lack of information on potential temporal and spatial trends in the regional background lead concentrations. Additional data may produce a better estimate of the variability of the regional background concentration of lead. There is also a lack of information on the location of the monitoring/residential wells with respect to anthropogenic and non-anthropogenic sources of lead. Another source of uncertainty is the lack of information on the sampling and analysis methods employed by Parliman et al. and Spruill. For example the size of the filter used to collect the samples for dissolved lead analysis was not specified by Parliman et al., and neither report specified the analytical method used to determine the lead concentration.

Table 5 indicates a reasonable value for the local background dissolved and total lead concentration in surface water is 3 and 7 µg/L, respectively. These estimates may be conservative but appear reasonable given the data summarized in Table 5. The suggested background for dissolved lead equals the lowest mean observed at the 10 monitoring stations located throughout Idaho but is only slightly greater than the mean concentration observed in the Coeur d'Alene River at Cataldo, an area where mine tailings are known to exist in the river sediments. The background for total lead concentration also equals the lowest mean concentration observed at the 10 Idaho monitoring stations and the one observation available for Hayden Creek. It however is one third of the mean total lead concentration recorded at Cataldo.

Sources of uncertainty in the estimates for background surface water quality include the use of different analytical methods to determine the lead concentrations, the lack of information on the location of the monitoring stations with respect to anthropogenic and non-anthropogenic sources of lead, and the lack of information on potential temporal and spatial trends in the regional background lead concentrations. The USGS database does not indicate the analytical methods used for samples analyzed prior to 1985. From 1985to1995, samples were analyzed by atomic absorption spectroscopy although the method detection limits varied depending upon whether the analysis was performed using flame atomic absorption or graphite furnace atomic absorption. The arithmetic means shown in Table 5 were calculated by setting all non-detects equal to one half the detection limit. For the years prior to 1985, non-detects were simply reported as 'U', with no detection limit indicated. This would tend to result in a low bias in the means shown in Table 5. While the impact of the lack of information on the location of monitoring stations with respect to anthropogenic and non-anthropogenic sources of lead can not be quantified, it may have been minimized by selecting concentrations towards the lower end of observed values.

8.0 CONCLUSIONS

Figure 6, in the main body of the report, shows the estimated range for local background soil lead concentration for the Coeur d'Alene Basin, along with the estimated ranges for the contributions from regional sources, automobile emissions and lead based paint. The latter are considered to be the major potential anthropogenic sources of soil lead, other than mining and smelting related contamination of soil. The range for the local soil lead concentrations is estimated to be 120-2700 ppm; the lower and upper bounds on the range represent the sums of the lower and upper bound estimates, respectively, of the three major contributing sources to local background.

The global/regional contribution is estimated to range from approximately 20 ppm to 250 ppm. Locations in the basin where the regional geology and long-range atmospheric transport of lead are the dominant sources of lead in soil would be expected to have soil lead concentrations that fall within this range.

The potential contribution of historic automobile emissions to local background may range from approximately 10 to 450 ppm. This range would apply to areas in the basin adjacent to high-volume roads (e.g., Interstate 90); a lower upper bound estimate of 120 ppm may apply to low-volume roads. These estimates are highly uncertain, primarily because of the lack of data on automobile impacts in the basin region. What data are available relate to transportation corridors that were heavily impacted by local mining and smelting operations. Thus, the estimate of the automobile contribution to local background is derived from studies conducted outside of the basin, and may not be directly applicable to the basin region.

The potential contribution from lead based paint to local soil lead background may range from approximately 100 ppm to 2000 ppm. Soil adjacent to structures that have or had deteriorating exterior lead based painted surfaces may have soil lead concentrations that fall within this range. Here again, considerable uncertainty is associated with these estimates because of a lack of site-specific data paint lead impacts on soil in the Basin. The estimates are derived from studies outside the basin and may not be directly applicable to any given location within the Basin.

In summary, the major sources of uncertainty in the estimated range of local background soil lead concentrations in the basin include the following:

1) The broad range of arithmetic mean soil lead concentrations attributed to exterior lead-based paint reported in the literature (100-2,000 ppm). (Chaney et al., 1988; Hardy et al., 1971; Mielke et al., 1983; Ter Haar and Aronow, 1974)

2) The broad range in automobile contributions to local background soil lead concentrations (11-442 ppm) reported in the literature (Agrawal et al., 1981; Case et al., 1989; Motto et al., 1970; Ward et al., 1975; Wheeler and Rolfe, 1979; Wong and Tam, 1978)

3) Ignoring the geo-spatial information present in the available data. The range in regional soil lead concentrations shown in Figures 5 and 6 represents information obtained from soil and sediment samples collected throughout the Coeur d'Alene River Basin. (Gott and Cathrall, 1980; Hagler-Bailly, 1995; Reece et al., 1978; URSG, 1998) It is likely that the regional soil lead concentration varies with location.

4) Combining soil and sediment data to develop an estimate of regional soil lead background. In some areas within the Coeur d'Alene River Basin such as the floodplains, combining data from both media may be reasonable. In areas not located within the floodplains, the appropriateness of combining soil and sediment data is questionable.

Collection and analysis of site specific data would be expected to decrease the level of uncertainty in the estimated local background soil lead. Consideration of the spatial information in the data would tend to decrease the level of uncertainty in the estimate attributed to all of the sources listed above. Geo-spatial information could be used to address the spatial distribution of anthropogenic contributions (i.e., lead-based paint and automobile emissions) to the local background soil lead concentration as well as the spatial distribution of regional soil and sediment lead background concentrations.

The estimated range for local background groundwater lead concentration in the Coeur d'Alene River Basin is 5-15 μg/L. Estimates for the local background dissolved and total lead concentrations in surface water are 3 and 7 μg/L, respectively.

Uncertainties associated with the groundwater and surface water background estimates is primarily due to the limited data. Collection of site specific data and consideration of temporal and spatial trends would tend to decrease the uncertainty in the estimates of background lead concentrations.

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Table 1. Reported and Predicted Soil and Rock Lead Concentration Percentiles from Gott and Cathrall (1980)			
	Soil (ppm)		Rock (ppm)
Percentile	Reported	Predicted[1]	Reported
25	28	20	8
50	43	42	19
75	75	88	43
90	171	170	140
95	na	253	na

[1]Maximum likelihood best fit lognormal PDF (AM, 76.4; SD, 115; GM, 4.23; GSD, 2.97). na, not reported in Gott and Cathrall (1980)..

Table 2 Sediment Lead Concentration Percentiles for the *Low* Distribution Estimated from URSG/CH2M Hill Data			
Percentile [1]	Sediment Lead Concentration (ppm)		
	Truncation A[2]	Truncation B	Truncation C
25th	18	20	22
50th	23	27	35
75th	29	37	57
90th	36	48	89
95th	42	57	116

[1] Percentiles calculated as: $Xp = \exp(y + Z_p s_y)$; x_p, estimated percentile; exp, inverse natural logarithm; y, mean of the logarithms of the data; Z_p, Z statistic at probability, p; s_y, standard deviation of the logarithms of the data.
[2]Truncation points shown in Figures 2-4.

TABLE 3 SOIL LEAD CONCENTRATION PERCENTILES FOR SPOKANE BASIN			
Percentile	Sediment Lead Concentration (ppm)		
	Statewide	Spokane Basin	
		Reported[1]	Estimated[2]
25th	not provided	not provided	10
50th	7	11	11.0
75th	not provided	not provided	13
90th	17	15	15
95th	not provided	not provided	16

[1] Reported by WSDE 1994.

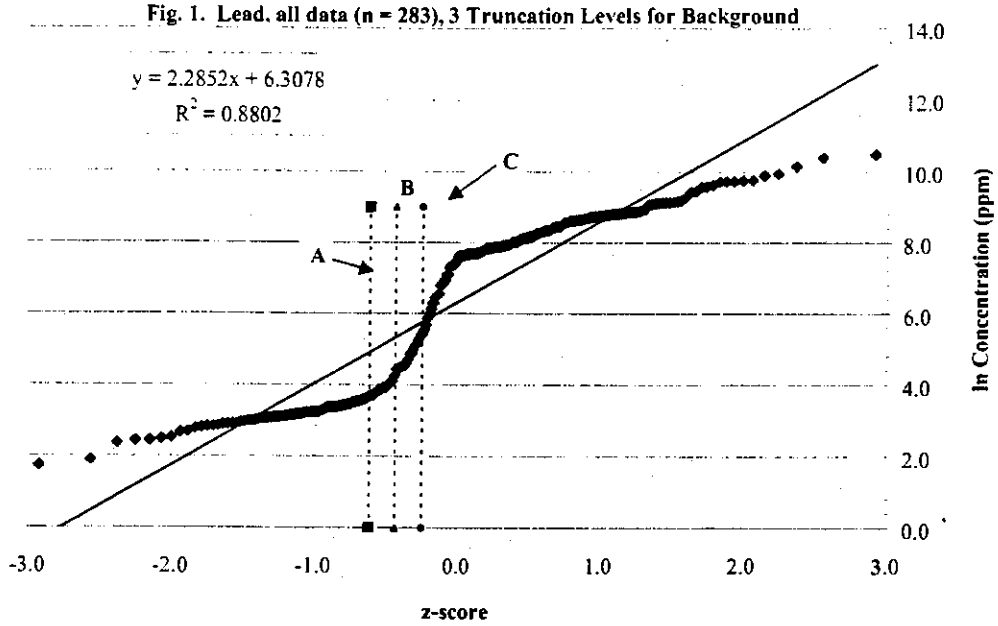
[2] Percentiles calculated as: $Xp = \exp(y + Z_p s_y)$; x_p, estimated percentile; exp, inverse natural logarithm; y, mean of the logarithms of the data; Z_p, Z statistic at probability, p; s_y, standard deviation of the logarithms of the data.

Table 4 Comparison of Residential Yard and Roadside Soil Lead Concentrations from Terragraphics (1990)								
Location	Soil Lead Concentration (ppm)							
	Residential			Road Shoulders			Difference (Road-Res.)	
	n	Mean	Median	N	Mean	Median	Mean	Median
Smelterville	200	3580	3010	6	4960	4970	1380	1960
Kellogg	771	2796	2440	5	3472	2280	676	-160
Wardner	92	2040	1500	1	1300	1300	-740	-200
Page	50	1090	810	3	818	595	-272	-215
Elizabeth Park	27	799	734	2	695	695	-104	-39
Pinehurst	100	683	501	8	1396	1015	713	514
Total	1240	1831	1499	24	2436	1480	605	-19

TABLE 5 SURFACE WATER LEAD CONCENTRATIONS						
	Total Lead (µg/L)			Dissolved Lead (µg/L)		
	Range in Data	Mean	Range ln Means	Range in Data	Mean	Range ln Means
10 monitoring stations in Idaho	<1 - 350	42	7 - 67	<1 - 280	6	3 - 14
Coeur d'Alene at Cataldo	<0.5 - 81	21	-	<0.5 - 24	4	-
Hayden Creek (north of Lake Coeur d'Alene)[1]	-	7	-	<0.5 - 44	4	-

1. There was only one observation for total lead concentration in the database for Hayden Creek, collected in 1971.

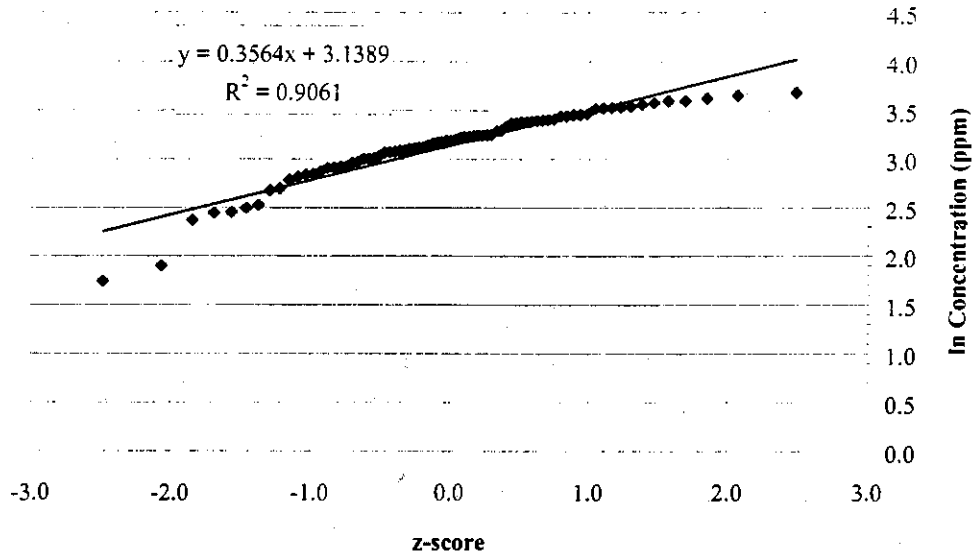
Fig. 1. Lead, all data (n = 283), 3 Truncation Levels for Background



Summary Statistics			
slope	2.29		
95% LCL	2.19		
95% UCL	2.38		
intercept	6.31		
95% LCL	6.21		
95% UCL	6.41		
r^2	0.880		
n	283		
AM	3186.95		
SD	4887.23		
GM	548.83	Combination of	
GSD	9.83	LCL's	UCL's
25th %ile	117.5	113.8	121.3
75th %ile	2563.5	2172.4	3025.0
90th %ile	10264.0	8190.9	12861.9
95th %ile	23543.9	18124.7	30583.4
99th %ile	111744.5	80412.0	155285.6

Description of Data Set: LEAD
from URSG file: soil_sed_metals_11_12_98.xls
sediment in LC {lower basin}
detects only
All Conc. Data (background and site-impacted)

Fig. 2. Lead, Truncation Point "A" (n = 77)
ln(x) <= 3.69, x <= 39.9 ppm



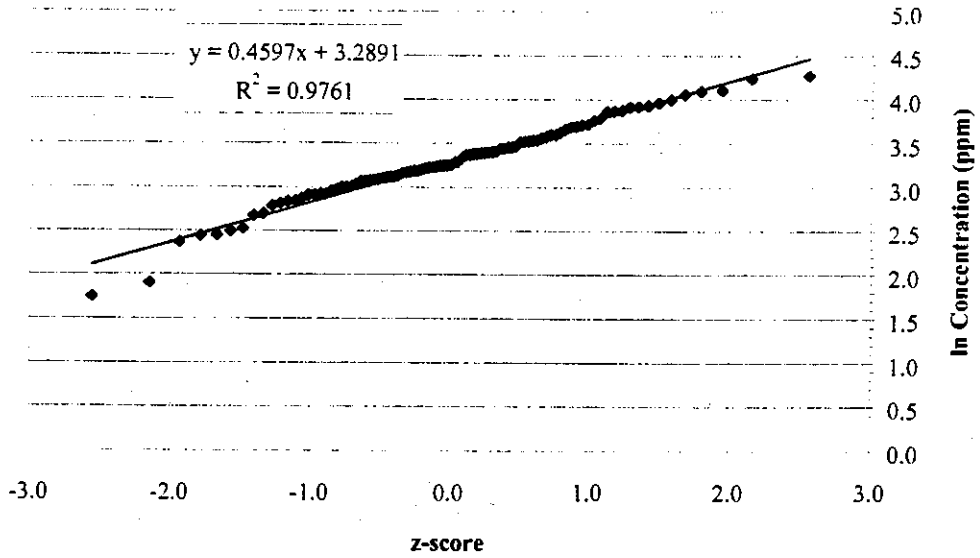
Summary Statistics	
slope	0.36
95% LCL	0.33
95% UCL	0.38
intercept	3.14
95% LCL	3.11
95% UCL	3.17
r^2	0.91
n	77
AM	24.49
SD	7.62
GM	23.08
GSD	1.43

	Combination of	
	LCL's	UCL's
25th %ile	18.1	18.3
75th %ile	29.3	28.1	30.7
90th %ile	36.4	34.3	38.7
95th %ile	41.5	38.7	44.5
99th %ile	52.9	48.4	57.7

Description of Data Set: LEAD

- from URSG file: soil_sed_metals_11_12_98.xls
- sediment in LC {lower basin}
- detects only
- Background defined as ln(x) <= 3.69

Fig. 3. Lead, Truncation Point "B" (n = 95)
ln(x) <= 4.27, x <= 71.7 ppm

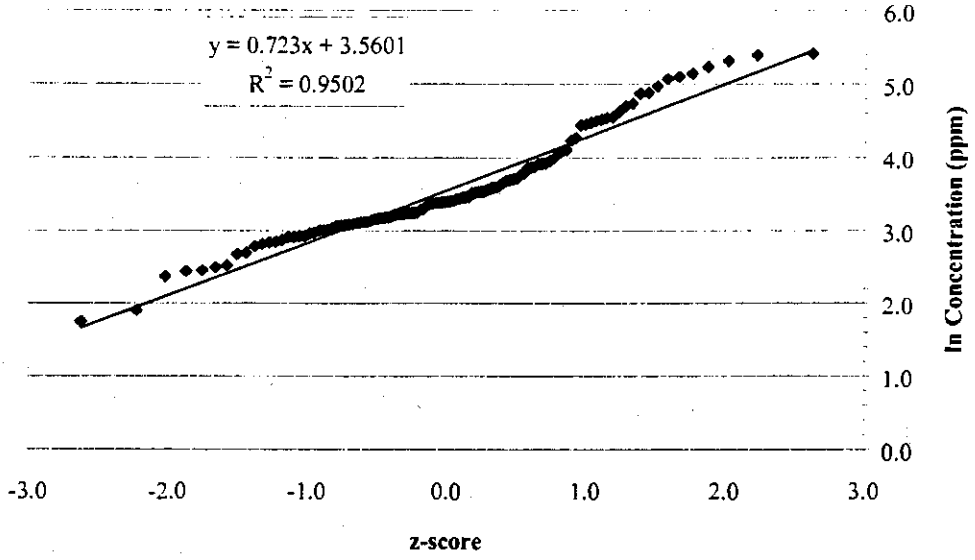


Summary Statistics			
slope	0.46		
95% LCL	0.44		
95% UCL	0.47		
intercept	3.29		
95% LCL	3.27		
95% UCL	3.30		
r^2	0.98		
n	95		
AM	29.65		
SD	13.32		
GM	26.82	**Combination of**	
GSD	1.58	LCL's	UCL's
25th %ile	19.7	19.6	19.8
75th %ile	36.6	35.7	37.5
90th %ile	48.3	46.7	50.0
95th %ile	57.1	54.9	59.4
99th %ile	78.1	74.4	82.1

Description of Data Set: LEAD

- from URSG file: soil_sed_metals_11_12_98.xls
- sediment in LC {lower basin}
- detects only
- Background defined as ln(x) <= 4.27

Fig. 4. Lead, Truncation Point "C" (n = 115)
ln(x) <= 5.42, x <= 225.0 ppm



Summary Statistics	
slope	0.72
95% LCL	0.69
95% UCL	0.75
intercept	3.56
95% LCL	3.53
95% UCL	3.59
r^2	0.95
n	115
AM	47.91
SD	45.96
GM	35.17
GSD	2.06

	Combination of	
	LCL's	UCL's
25th %ile	21.6	21.4	21.8
75th %ile	57.3	54.4	60.3
90th %ile	88.8	82.8	95.3
95th %ile	115.5	106.5	125.3
99th %ile	189.0	170.6	209.4

Description of Data Set: LEAD

- from URSG file: soil_sed_metals_11_12_98.xls
- sediment in LC {lower basin}
- detects only
- Background defined as ln(x) <= 5.42

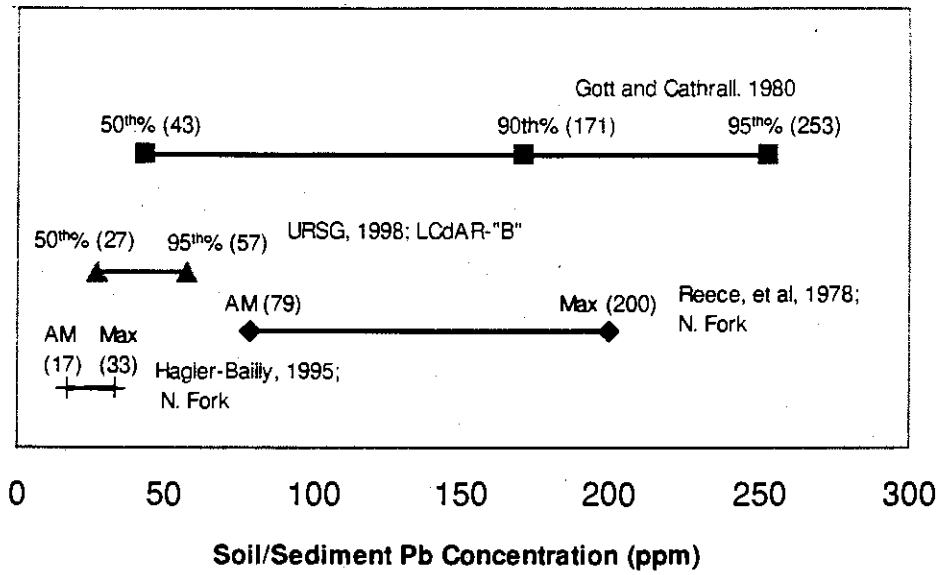


Figure 5. Estimates of Regional Background Concentrations of Lead in Soil and Sediment in the Coeur d'Alene River Basin. Shown are estimated ranges of regional background based on data reported in Reece et al (1978, diamonds), URSG (1998, triangles), Gott and Cathrall (1980, squares) and Hagler-Bailly (1995, crosses). AM and Max refer to arithmetic mean and maximum, respectively; percent values are percentiles. The numbers in parentheses are the bounds on the ranges.

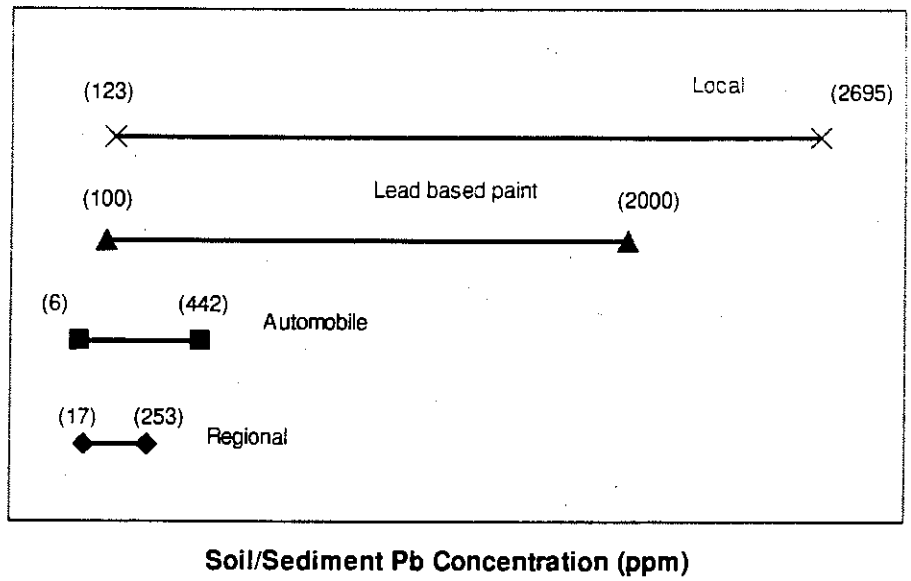


Figure 6. Estimates of Background Concentrations of Lead in Soil in the Coeur d'Alene River Basin. Shown are the estimated contributions of regional sources (diamonds), exterior lead based paint (triangles), and automotive emissions (squares) to local background concentrations of lead in the Coeur d'Alene River Basin (crosses). The horizontal lines represent the estimated range of the contributions from each source category. The numbers in parentheses are the bounds on the ranges.

Appendix A

Potentially Relevant Sources for Determining Background Lead Concentrations in the Coeur d'Alene River Basin not Cited in the Main Body of the Report

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Daines, R.H., H. Motto, D.M. Chilko. 1970. Atmospheric Lead: Its Relationship to Traffic Volume and Proximity to Highways. Environmental Science and Technology. 4(4): 318-322.

Ganje, T.J. and A.L. Page. 1972. Lead Concentrations of Plants, Soil and Air Near Highways. California Agriculture, April, p. 7-9.

Huntzicker, J.J., S.K. Friedlander, C.I. Davidson. 1975. Material Balance for Automobile-Emitted Lead in Los Angeles Basin. Environmental Science and Technology. 9(5): 448-457.

Little, P. and M.H. Martin. 1972. A Survey of Zinc, Lead and Cadmium in Soil and Natural Vegetation Around a Smelting Complex. Environ. Pollut. (3): 241-254.

Maxfield, D., J.M. Rodriguez, M. Buettner, J. Davis, L. Forbes, R. Kovacs, W. Russel, L. Schultz, R. Smith, and C.M. Wai. 1974. Heavy Metal Pollution in the Sediments of the Coeur d'Alene River Delta. Environ. Pollut. (7): 1-6.

Maxfield, D., J.M. Rodriguez, M. Buettner, J. Davis, L. Forbes, R. Kovacs, W. Russel, L. Schultz, R. Smith, J. Stanton, and C.M. Wai. 1974. Heavy Metal Content in the Sediments of the Southern Part of the Coeur d'Alene Lake. Environ. Pollut. (6): 263-266.

Mielke, H.W., S. Burroughs, R. Wade, T. Yarrow, and P.W. Mielke. 1984. Urban Soil Lead in Minnesota: Soil Transect Results of Four Cities. 50 (1): 19-24.

Palmer, K.T. and C.L. Kucera. 1980. Lead Contamination of Sycamore and Soil from Lead Mining and Smelting Operations in Eastern Missouri. J. Environ. Qual. 9 (1): 106-111.

Appendix F
Chemical Toxicity Profiles

1.0 Chemical Profiles

Toxic effects of the chemicals of concern are summarized below along with the toxicity criteria used in the baseline risk assessment for assessing non-carcinogenic and carcinogenic effects.

1.1 Antimony

Literature on the health effects of antimony in humans is mostly from reports on high-dose occupational exposures by inhalation in antimony smelting and processing plants.

The U.S. EPA has derived a RfD of **0.0004 mg/kg-day** for ingestion of antimony (U.S. EPA 1998a). In the study upon which the RfD was based (Schroeder et al. 1970 as cited in U.S. EPA 1998a), the administered form of antimony was potassium antimony tartrate in water to male and female rats. The critical effects included reduced lifespan, altered cholesterol levels in both sexes, decreased non-fasting blood glucose levels and mean heart weight in treated males. No increase in tumors was observed as a result of the treatment.

The oral reference dose for antimony was based on an uncertainty factor of 1000, which includes a factor of 10 each to account for interspecies conversion, protection of sensitive individuals, and extrapolation from a lowest-observable-adverse-effect level (LOAEL) to a no-observable-adverse-effect level (NOAEL). Confidence in the chosen study (Shroeder et al. 1970 as cited in U.S. EPA 1998a) was rated as low, since only one dose level of antimony was administered, only one species was tested, and gross pathology and histopathology were not adequately described. Confidence in the data was also rated low because of a general lack of adequate oral exposure investigations. Consequently, a low confidence was assigned to the RfD for antimony.

1.2 Arsenic

The toxicity of arsenic varies with its chemical form. The primary valence forms are trivalent and pentavalent inorganic arsenic. Trivalent (arsenite) compounds are generally more acutely toxic than pentavalent (arsenate) compounds, but in the environment arsenite is converted to arsenate (Goyer 1996). Chemical mineralogy and physical characteristics also affect acute and chronic toxicity.

In humans, arsenic does not appear to accumulate in physiologically active compartments of the body (U.S. EPA 1984). Arsenite reacts with sulfhydryl groups of proteins, which leads to higher amounts of arsenic in hair, skin, and stomach (U.S. EPA 1988; Goyer 1996). The liver readily

converts absorbed arsenic into compounds that are rapidly excreted in the urine. About 50 to 80 percent of absorbed arsenic is eliminated by urinary excretion (Goyer 1996). Consequently, the amount of arsenic excreted is a reliable indicator of the level of recent arsenic exposure. Other elimination routes include feces, hair, sweat, and desquamation of the skin.

1.2.1 Carcinogenic Effects of Arsenic

Risk assessments for arsenic are generally based on the induction of lung cancer by inhaled arsenic and the risk of skin cancer by ingested arsenic. These are the effects of greatest concern for chronic exposure and for assessing remedial objectives.

The oral slope factor based on skin cancer was derived from a study involving arsenic exposures to over 40,000 people in Taiwan. These people were exposed to arsenic in groundwater used for drinking water for a significant portion of their lifetime. Despite the many uncertainties in the study, this database on arsenic represents one of the best available sources of dose-response information in humans. Unfortunately, the study design limited its usefulness to derive precise risk estimates. Specifically, the subjects were classified into three exposure groups (high, medium, or low) because of the lack of information on the amount of exposure. Skin cancer has been noted in arsenic-exposed populations in Chile, Argentina, and Mexico, although no association has been found in the U.S. between arsenic and cancer incidence. U.S. EPA (1998a) notes that sample sizes of exposed populations in the U.S. may have been too small to statistically detect a relationship.

The U.S. EPA (1988 and 1998a) derived the oral slope factor of **1.5 (mg/kg-day)$^{-1}$** used in this risk assessment. This value is an order of magnitude lower than the previous slope factor of 15 (mg/kg-day)$^{-1}$ issued by U.S. EPA (1984). The difference is due to a revised evaluation of the Taiwan study. The dose-specific and age-specific skin cancer rates associated with exposure to arsenic in drinking water were predicted using the modified cancer risk model and incorporating assumptions that are more realistic for the U.S. population.

1.2.2 Non-carcinogenic Effects of Arsenic

Exposure and health effects associated with chronic exposure to elevated levels of arsenic have been documented in the U.S. and throughout the world, most commonly through natural occurrence of high levels in drinking water and also through agricultural and medicinal uses of arsenic. Gastrointestinal irritation, skin disorders, anemia, injury to the peripheral and central nervous systems are some of the non-carcinogenic effects noted in populations exposed to high levels of arsenic (U.S. EPA 1988; ATSDR 1993a). In addition, a cardiovascular disorder known as blackfoot disease, which is an endemic peripheral artery disease causing discoloration or gangrene of the lower extremities, was also observed in Taiwan (U.S. EPA 1988; ATSDR 1993a). Skin effects are generally observed at the lowest levels of exposure that cause

observable effects such as hyperpigmentation (excess pigment) and hyperkeratosis (excess keratin leading to wart-like skin thickening).

The chronic RfD for arsenic is based on the NOAEL for skin effects from the same study as described above for the slope factor. The RfD listed in the IRIS database is **0.0003 mg/kg-day**, which incorporates an uncertainty factor of 3. The uncertainty factor was based on the lack of data to preclude reproductive toxicity as a critical effect and on the uncertainty as to whether all sensitive individuals will be protected. Because U.S. EPA scientists are not in agreement on the interpretation of the data and the appropriate oral RfD for arsenic, the agency allows flexibility in the RfD from 0.0001 to 0.0008 mg/kg-day (U.S. EPA 1998a).

Confidence in the chosen studies (Tseng 1977; Tseng et al. 1968 as cited in U.S. EPA 1998a) were considered medium, since an extremely large number of individuals (greater than 40,000) were included in the assessment. However, the doses were not well characterized and other contaminants were also present. In addition, problems were noted in several epidemiological studies. The Tseng studies in particular did not look at potential exposures from food or any other sources. Consequently, a medium confidence was assigned to the RfD for arsenic.

1.3 Cadmium

Long-term exposure to cadmium by both inhalation and ingestion is associated with proteinuria (protein in the urine indicating kidney effects) in both worker and general populations (ATSDR 1993b; U.S. EPA 1998a). In both occupational and non-occupational populations, kidney effects were found only after long-term (greater than 30 years) chronic exposure to fairly high levels of cadmium. In addition, a threshold amount of cadmium apparently must be inhaled or ingested before kidney effects are observed. A threshold estimate of 2,000-mg over 50 years was approximated from a study of a population in Japan that ate rice grown in cadmium-polluted waters (Nogawa et al. 1989).

A concentration of 200 micrograms cadmium per gram wet weight kidney cortex is the highest renal level not associated with proteinuria in humans based on chronic exposure (U.S. EPA 1998a). U.S. EPA extrapolated this level to a NOAEL of 0.005 mg/kg-day in water and 0.01 mg/kg-day in food using a toxicokinetic model assuming 5 percent and 2.5 percent absorption, respectively. U.S. EPA's oral RfD was thus calculated as **0.0005 mg/kg-day** cadmium ingested in water and **0.001 mg/kg-day** for cadmium ingested in food, using an uncertainty factor of 10 to account for variation in individual sensitivity. Because the NOAEL was based on data obtained from many studies on the toxicity of cadmium in both humans and animals, confidence in the data and in the RfD values was rated as high. In addition, the data allows calculation of cadmium absorption, distribution, metabolism and elimination.

In the absence of a dermal RfD, U.S. EPA recommends using an oral RfD corrected to an absorbed dose for evaluating the systemic effects via dermal absorption of chemicals from contact with soil. In developing an RfD, U.S. EPA (U.S. EPA 1998a) assumed absorption percentages to develop administered doses for food or water from the absorbed dose predicted by the toxicokinetic model. The dermal absorbed RfD for food is thus **0.00001 mg/kg-day**, which is obtained by multiplying the administered oral RfD of 0.001 mg/kg-day by the gastrointestinal absorption factor of 1 percent (U.S. EPA 1998b).

Cadmium has not been shown to be carcinogenic in laboratory animals via oral exposure at doses of 2.5 mg/kg-day and below (ATSDR 1993b). Studies in humans likewise have not shown evidence of cadmium causing carcinogenic effects following oral exposure, although these studies may have limited sensitivity to detect increases in cancer incidence (ATSDR 1993b). However, U.S. EPA classified cadmium as a B1 or probable human carcinogen by the inhalation route (U.S. EPA 1998a). However, in the risk assessment, cadmium is only evaluated as a non-carcinogen, since exposure to cadmium by inhalation was not considered a concern at this site.

1.4 Copper

Copper is an essential element for humans. Copper is naturally found in food and is necessary in the diet for good health. Approximately 1 milligram of copper is consumed from food and/or ingested from water every day.

The toxic effects of the chemical at the acute LOAEL for sensitive individuals appear to occur at about twice the daily level required for health. The recommended dietary allowance (RDA; NAS 1980 as cited in ATSDR 1990a) estimates 2 to 3 mg/day (0.03 to 0.04 mg/kg-day) are required for health. The onset of gastrointestinal irritation from copper ingestion in humans has been observed at a wide range of doses from 5 to 420 mg/day, (0.07 to 6 mg/kg-day) (ATSDR 1990a). Other effects appear to be a flu-like disease linked with the inhalation of copper fume by workers (ATSDR 1990a). The liver also appears to be the primary target organ for toxicity in subchronic studies in rats and pigs (Hurst 1991).

The toxic effects of exposure to copper and its compounds include widespread capillary damage, kidney and liver injury, central nervous excitation, jaundice, pain over the liver, and depression. Copper intake at high doses may lead to lethargy, coma, and refractory hypotension (EHC 1998). Exposure to copper dusts can cause dermatitis, discoloring of the skin, irritation of the eyes, nose and throat, and cause headaches, dizziness, nausea, and diarrhea. Vomiting, diarrhea, stomach cramps, and nausea may result after drinking water with high levels of copper (ATSDR 1990b).

U.S. EPA's Environmental Criteria and Assessment Office (ECAO; Hurst 1991, ECAO is now called the National Center for Environmental Assessment, NCEA) recommended an oral RfD of

0.04 to 0.07 mg/kg-day for copper. This recommendation was based on chronic gastrointestinal effects in humans from drinking beverages or water contaminated with copper (ATSDR 1990a). The latest publication of the Health Effects Assessment Summary Tables (U.S. EPA 1997) lists a maximum allowable concentration for copper in drinking water of 1.3 mg/L. This concentration translates into **0.037 mg/kg-day** (1.3 mg/L x 2 L/day [water ingestion rate] / 70 kg [adult body weight]), and is the lower end of the range recommended by ECAO (Hurst 1991). Therefore, this value was used as the oral RfD for copper in this risk assessment.

Copper is not known to cause cancer. U.S. EPA has classified copper as Group D based on a lack of human data and inadequate animal data on its carcinogenicity (U.S. EPA 1998a).

1.5 Lead

As summarized by ATSDR (1993c) and U.S. EPA (1986), lead is ubiquitous in the environment due to its widespread historical uses as a fuel additive, in paints, solders, and other consumer products. Lead can cause a wide range of toxic effects, mainly at high doses. The primary effects of concern for chronic exposures to environmental lead levels, however, are subtle neurobehavioral effects in young children. Subclinical effects on the blood-forming system are a secondary issue at low levels of exposure. Controversy continues to surround the question of low-level health effects from lead, which are often indistinguishable from other factors, particularly socioeconomic influences.

Lead can result in a wide range of biological effects depending upon the level and duration of exposure. Children are considered to be the most sensitive population. This higher susceptibility derives from numerous factors including metabolic, neurological, and behavioral reasons. Children absorb a larger fraction of ingested lead than do adults; thus, children will experience a higher internal lead dose per unit of body mass than adults at similar exposure concentrations. Absorption of lead appears to be higher in children who have low dietary iron or calcium intakes; thus, dietary insufficiencies, which are not uncommon in children, may contribute to their susceptibility to lead toxicity. Infants are born with a lead body burden that reflects the burden of the mother during gestation, lead from the maternal skeleton is transferred across the placenta to the fetus and additional lead exposure may occur during breast-feeding. Exposures *in utero* and during early infancy may contribute to susceptibility to lead later in childhood.

Epidemiologic studies have provided evidence for a relationship between prenatal and postnatal lead exposure in infants and young children, and support the use of PbB as an index of toxicological effect. Measurable effects include impaired or delayed mental development, disorders of heme metabolism, and other biochemical effects on blood cells and decreased serum levels of vitamin D. Although a PbB threshold for some of these effects has not been established, the evidence suggests that it may lie within 10-15 micrograms per deciliter of blood

(µg/dL). For neurobehavioral effects in particular, the existing epidemiological studies do not provide definite evidence of a threshold. As blood lead increases above the range of 10-15 µg/dL, the risk for more pronounced effects on all of the above endpoints increases. At levels greater than 30 µg/dL, the risk for nephrotoxicity and overt neurological effects (i.e., encephalopathy) become substantial.

In light of these data, the CDC has issued guidance for appropriate screening and for developing preventive measures aimed at reducing children's PbB below 10 µg/dL, the intervention level (CDC 1991). This level for children has come to be used as an initial level in screening for exposure. We can be fairly certain that the use of PbB, as estimated by the IEUBK model, provides a reasonable measure of the potential health risks associated with lead exposures, and an appropriate basis for developing RBCs.

Remedial actions, such as community wide prevention activities and blood lead screening, are usually recommended by the CDC when blood lead levels exceed 10 µg/dL. EPA has also identified 10 µg/dL as a level of concern for the developing fetus in a pregnant woman, and the goal is a no more than 5% probability that the fetal blood lead will exceed 10 µg/dL if the mother is exposed. For adults, the concerns are peripheral neuropathy (i.e., footdrop and wristdrop characteristic of the painter or other workers with excessive occupational exposure to lead) or chronic nephropathy (morphological and functional changes in the kidney), under excess occupational or even accidental exposures.

Excess lead exposure has multiple hematological effects. Lead-induced anemia, in particular, results from a shortened lifespan of red blood cells and impairment of heme synthesis. Other target organs are the gastrointestinal and reproductive systems. However, the most sensitive adverse health effect for adults in the general population may actually be hypertension (U.S. EPA 1989). A number of epidemiological studies provided evidence for an association between increased blood pressure and elevated body burden of lead in adults (ATSDR 1993c). However, even where an association was found, the increase in blood pressure was very slight (Schwartz 1995).

1.6 Mercury and Compounds

Long-term exposure to inorganic mercury can permanently damage the brain, kidneys, and developing fetuses. The form of mercury and the way an individual is exposed to it determine which of these health effects will be more severe. Organic mercury that is eaten in contaminated fish or grain may cause greater harm to the brain and developing fetuses than to the kidney. Breathed metallic mercury vapor may cause greater harm to the brain. Inorganic mercury salts that are eaten in contaminated food or drunk in water may cause greater harm to the kidneys (ATSDR 1990c). Short-term exposure to high levels of inorganic and organic mercury will have

similar health effects; but full recovery (excretion of the chemical from the body) is more likely after short-term exposures. Mercury has not been shown to cause cancer in humans. U.S. EPA has classified inorganic mercury as a Group D carcinogen; because no human data were available and animal and other supporting data are inadequate (U.S. EPA 1998a). For this risk assessment, only inorganic mercury was considered a chemical of concern.

In October of 1987, a panel of mercury experts met at a Peer Review Workshop on Mercury Issues in Cincinnati, Ohio, and reviewed outstanding issues concerning the health effects and risk assessment of inorganic mercury (U.S. EPA 1987 as cited in U.S. EPA 1998a). The panel decided that the most sensitive adverse effect for mercury risk assessment is formation of mercuric-mercury-induced autoimmune glomerulonephritis. The production and deposition of IgG antibodies to the glomerular basement membrane in the kidney can be considered the first step in the formation of the mercuric-mercury-induced autoimmune effect.

Three studies were chosen from a larger selection of studies as the basis for developing the oral RfD for mercury. In the Druet et al. (1978 as cited in U.S. EPA 1998a) study, an immune response was observed in Brown Norway rats injected with mercuric chloride ($HgCl_2$). This response was accompanied by proteinuria and in some cases by a nephrotic syndrome. Mercurials administered by inhalation or ingestion to Brown Norway rats also developed a systemic autoimmune disease (Bernaudin et al. 1981 as cited in U.S. EPA 1998a). After 60 days of $HgCl_2$ exposure, 100% (5/5) of the rats were observed with a mixed linear and granular pattern of IgG deposition in the glomeruli and granular IgG deposition in the arteries. Weak proteinuria was also observed.

Brown Norway $HgCl_2$-treated rats started to lose weight and hair after being administered with $HgCl_2$ by gavage (Andres 1984 as cited in U.S. EPA 1998a). Two of the $HgCl_2$-treated rats died 30-40 days after beginning the study. Examination of the kidneys by immunofluorescence showed deposits of IgG present in the renal glomeruli of the mercuric-treated rats. In addition, morphological lesions of the ileum and colon with abnormal deposits of IgA in the basement membranes of the intestinal glands were also observed.

The kidney and central nervous system are the major target organs for toxicity induced by inorganic mercury following ingestion or inhalation, respectively, in humans (ATSDR 1990c). The oral RfD value for inorganic mercury is **0.0003 mg/kg-day**. This RfD is based on kidney effects resulting from oral and parenteral (e.g., subcutaneous or intravenous injection) administration of mercury in rats. An uncertainty factor of 1000 was applied for the LOAEL to NOAEL conversion to account for use of subchronic studies, animal to human extrapolation, and sensitive human populations. Based on the weight of evidence from the studies mentioned above using Brown Norway rats and the entire mercuric mercury database, the level of confidence for the oral RfD was rated as high. In addition, the oral RfD was derived after intensive review and

workshop discussions of the entire inorganic mercury database, not just from one study (U.S. EPA 1998a).

1.7 Zinc

Zinc is an essential trace element for which reports of health effects are more common for deficiency than for toxicity (Goyer 1996). Chronic ingestion of zinc in humans, including therapeutic use, can interfere with iron and copper absorption. The effects on copper and iron biochemistry are considered of concern since long-term iron or copper deficiency could result in significant adverse effects such as hypochromic anemia (U.S. EPA 1998a). In addition, several studies have investigated the effects of zinc supplementation on the high-density lipoprotein (HDL) levels of adult males. High ingestion of zinc can decrease HDL levels. The observed change in HDL values in males may be significant since a sustained decrease in HDL concentrations may be associated with increased risk of coronary artery disease when in conjunction with an increase in low-density lipoprotein (LDL) cholesterol (U.S. EPA 1998a). The most common syndrome reported in humans exposed to zinc by inhalation is metal fume fever. This reversible condition is typically caused by occupational exposure to fumes of zinc or zinc oxide dust and is associated with chills, fever, sweating, and weakness.

The oral RfD is **0.3 mg/kg-day** for zinc and zinc compounds (U.S. EPA 1998a). The RfD is based on an oral study in humans in which the critical effect was a decrease in erythrocyte superoxide dismutase in adult women after 10 weeks of exposure (Yadrick et al. 1989 as cited in U.S. EPA 1998a). An uncertainty factor of 3 is attached to the RfD based on a minimal LOAEL from a moderate-duration study of sensitive individuals and consideration of zinc as an essential dietary nutrient. The level of confidence in the studies (as cited in U.S. EPA 1998a) were considered medium, since they were well conducted with many biochemical parameters investigated. However, only a few numbers of individuals were tested and the studies were all of short duration. Consequently, a medium confidence was assigned to the RfD for zinc.

The RfD of 0.3 mg/kg-day should supply an adequate amount of zinc for adolescents and adults without physiological impairment. This amount, however, is inadequate for the recommended dietary allowance (RDA) for infants, preadolescent children, or for lactating women. The RDA values for zinc range from 5 to 15 mg/day for different age and gender categories. This range accounts for the amount needed for growth, development, metabolism and tissue maintenance for the American population (U.S. EPA 1998a).

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APPENDIX C

Summary Table of Potential Receptor Groups

Summary Table of Receptor Groups

CSM Unit	Watershed	Segment Name	Segment Description	Comments Regarding Watershed
Residential Only				
CSM Unit 01	Big Creek	BigCrkSeg04	Big Creek Segment 04: Big Creek Below East Fork Tributary	
Residential and Water Play				
CSM Unit 01	Nine Mile Creek	NMSeg03	Nine Mile Creek Segment 03: West Fork Nine Mile Creek	Are waste piles present? Water play is associated with child residents in undeveloped water access sites near homes, not developed water access
Residential, Water Play, and Waste Piles				
CSM Unit 01	Canyon Creek	CCSeg02	Canyon Creek Segment 02: Hecla Intake to confluence with Gorge Gulch and O'Neil Gulch	Water play is associated with child residents in undeveloped water access sites near homes, not developed water
		CCSeg03	Canyon Creek Segment 03: Gorge Gulch	Water play is associated with child residents in undeveloped water access sites near homes, not developed water
		CCSeg04	Canyon Creek Segment 04: Gorge Gulch to Woodland Park	Water play is associated with child residents in undeveloped water access sites near homes, not developed water
		CCSeg05	Canyon Creek Segment 05: Woodland Park to confluence with South Fork Coeur d'Alene River	Extends below Gem portal to mouth. Water play is associated with child residents in undeveloped water access sites near homes, not developed water
	Nine Mile Creek	NMSeg02	Nine Mile Creek Segment 02: East Fork Nine Mile Creek from Interstate Millsite to confluence with West Fork Nine Mile Creek	Water play is associated with child residents in undeveloped water access sites near homes, not developed water access areas
		NMSeg04	Nine Mile Creek Segment 04: Lower Nine Mile Creek from confluence with West Fork to	
	Pine Creek	PineCrkSeg02	Pine Creek Segment 02: Upper Pine Creek and tributaries.	Water play is associated with child residents in undeveloped water access sites near homes, not developed water
		PineCrkSeg03	Pine Creek Segment 03: Pine Creek from East Fork tributary to mouth.	About half of the area in this segment is within the Bunker Hill Superfund Site. Water play is associated with child residents in undeveloped water access sites near homes, not developed water

Summary Table of Receptor Groups

CSM Unit	Watershed	Segment Name	Segment Description	Comments Regarding Watershed
Residential, Water Play, Waste Piles, and Schools/Day Care/Parks				
CSM Unit 01	Upper South Fork	UpperSFCDRSeg 01	Upper South Fork Coeur d'Alene River: Entire watershed.	
CSM Unit 02	South Fork	MidGradSeg01	South Fork Segment 01: South Fork Coeur d'Alene River from Canyon Creek to Kellogg. - Where Canyon Creek enters South Fork Creek to Elizabeth Park where Elk Creek enters at bridge	
Residential and Waste Piles				
CSM Unit 01	Moon Creek	MoonCrkSeg02	Moon Creek Segment 02: Moon Creek watershed except West Fork of Moon Creek	
Waste Piles Only				
CSM Unit 01	Pine Creek	PineCrkSeg01	Pine Creek Segment 01: East Fork and tributaries.	Waste piles present, but no residential. Area remote for easy recreational access
Residential, Water Play, and Recreational (Upland Parks)				
CSM Unit 02	Coeur d'Alene River Above Cataldo	MidGradSeg04	Mid-Gradient Streams Segment 04: South Fork Coeur d'Alene River from Enaville to Cataldo. - Coeur d'Alene River below confluence of North and South Forks of Coeur d'Alene River to the	
CSM Unit 03	Coeur d'Alene River Below Cataldo	LCDRSeg01	Lower Coeur d'Alene River Segment 01: Old Cataldo Bridge to Cataldo boat landing below	
		LCDRSeg02	Lower Coeur d'Alene River Segment 02: Cataldo Boat landing to Killarney Lake Road	
		LCDRSeg03	Lower Coeur d'Alene River Segment 03: Killarney Lake Road to Killarney Lake Canal	
		LCDRSeg04	Lower Coeur d'Alene River Segment 04: Killarney Lake Canal to upper end of Bear Marsh,	

Summary Table of Receptor Groups

CSM Unit	Watershed	Segment Name	Segment Description	Comments Regarding Watershed
		LCDRSeg05	Lower Coeur d'Alene River Segment 05: Upper end Bear	
		LCDRSeg06	Lower Coeur d'Alene River Segment 06: Harrison Bridge to	
Water Play and Recreational (Upland Parks)				
CSM Unit 01	Beaver Creek	BvrCrkSeg01	Beaver Creek Segment 01: Entire watershed.	The upper tributaries have waste pile areas with meadows that could be attractive for camping and water play. Contamination in the lower portion of the watershed is likely limited to contaminated sediments precipitating out in beaver
CSM Unit 04	Coeur d'Alene Lake	CDALakeSeg01	Coeur d'Alene Lake Segment 01: South of Harrison.	
		CDALakeSeg02	Coeur d'Alene Lake Segment 02: Harrison to Spokane River.	
		CDALakeSeg03	Coeur d'Alene Lake Segment 03: Wolf Lodge Bay.	
CSM Unit 05	Spokane River	SpokaneRSeg01	Spokane River Segment 01: Coeur d'Alene Lake to State Line.	
		SpokaneRSeg02	Spokane River Segment 02: State Line to Long Lake.	
		SpokaneRSeg03	Spokane River Segment 03: Long Lake and Lake Roosevelt.	
Relatively Uncontaminated				
CSM Unit 01	Big Creek	BigCrkSeg01	Big Creek Segment 01: Big Creek Above East Fork Tributary	
		BigCrkSeg02	Big Creek Segment 02: Big Creek East Fork	
		BigCrkSeg03	Big Creek Segment 03: Big Creek West Fork	
	Canyon Creek	CCSeg01	Canyon Creek Segment 01: Upper Canyon Creek above the Hecla	
	Moon Creek	MoonCrkSeg01	Moon Creek Segment 01: West Fork of Moon Creek.	Has this segment been remediated?

Summary Table of Receptor Groups

CSM Unit	Watershed	Segment Name	Segment Description	Comments Regarding Watershed
	Upper North Fork	NrthFrkSeg01	North Fork Segment 01: North Fork Coeur d'Alene River above	
	Nine Mile Creek	NMSeg01	Nine Mile Creek Segment 01: Upper East Fork Nine Mile Creek above Interstate Millsite	

Summary Table of Receptor Groups

CSM Unit	Watershed	Segment Name	Segment Description	Comments Regarding Watershed
	Prichard Creek	PrichCrkSeg01	Prichard Creek Segment 01: Prichard Creek above Paragon	
		PrichCrkSeg02	Prichard Creek Segment 02: Bear Gulch Tributary of Prichard Creek	
		PrichCrkSeg03	Prichard Creek Segment 03: Prichard Creek below Paragon Gulch including Paragon Millsites	
CSM Unit 02	South Fork	MidGradSeg02	South Fork Segment 02: South Fork Coeur d'Alene River from Kellogg to Enaville. - Elizabeth Park through superfund site to Enaville at confluence with North Fork	Mid-gradient segment 02 is primarily the Bunker Hill Superfund Site, which includes nearly all of this segment. Portions of the segment outside of the "box" are high in the watershed, and are likely to be less contaminated, except where the active
	North Fork	MidGradSeg03	Mid-Gradient Streams Segment 03: North Fork Coeur d'Alene River from Prichard to Enaville. - North Fork from mouth of Prichard	

Appendix C

Summary Table of Potential Receptor Groups

This table provides a list of segment names and descriptions for each potential receptor group.

APPENDIX D

Graphs of Concentration Distributions of Mercury, Thallium, and Manganese

Appendix D

Graphs of Concentration Distributions of Mercury, Thallium, and Manganese

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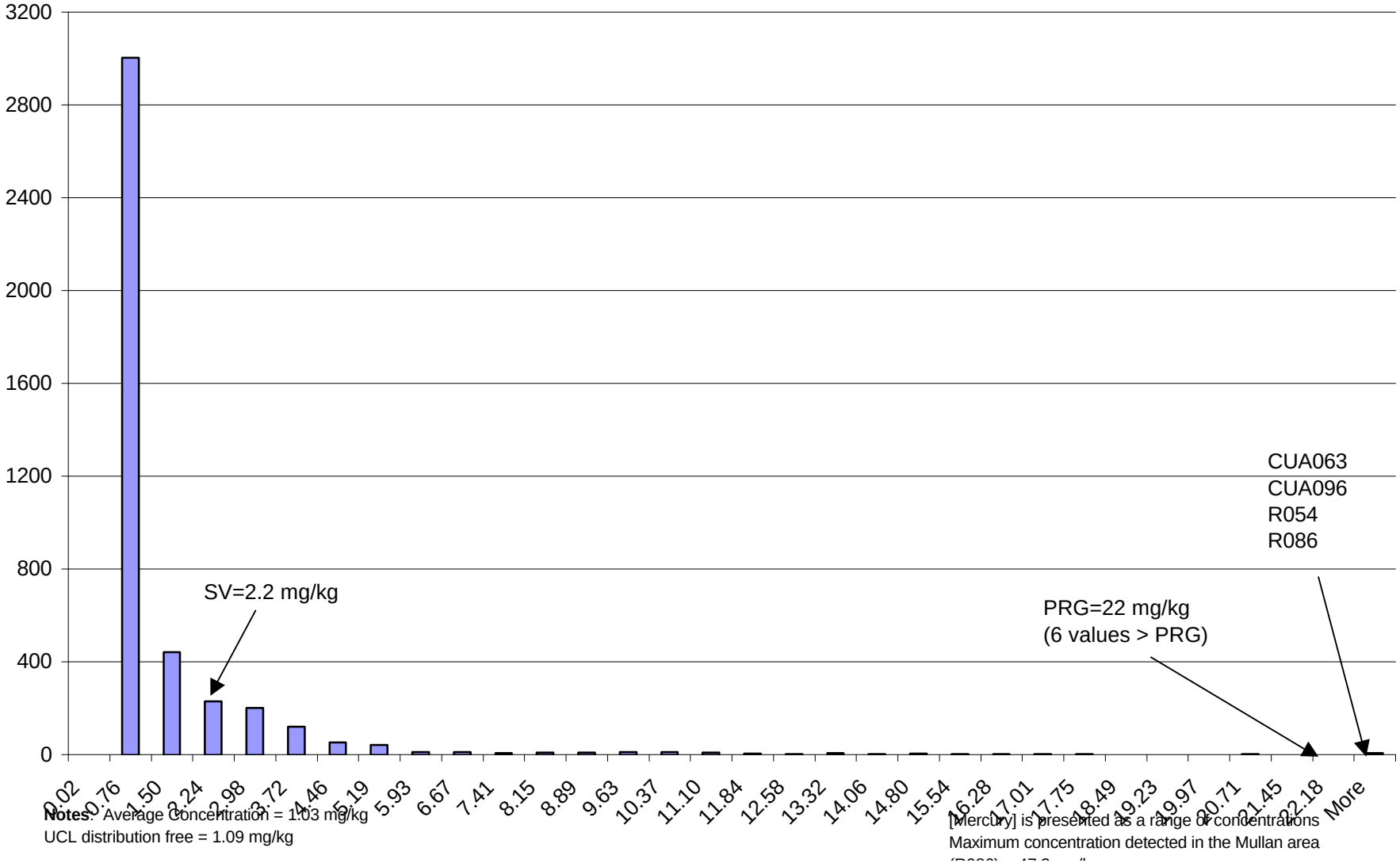
The histograms in this appendix shows the concentration ranges of mercury and thallium in soil/sediment and manganese in groundwater (future use) in the basin and the frequency with which they occur.

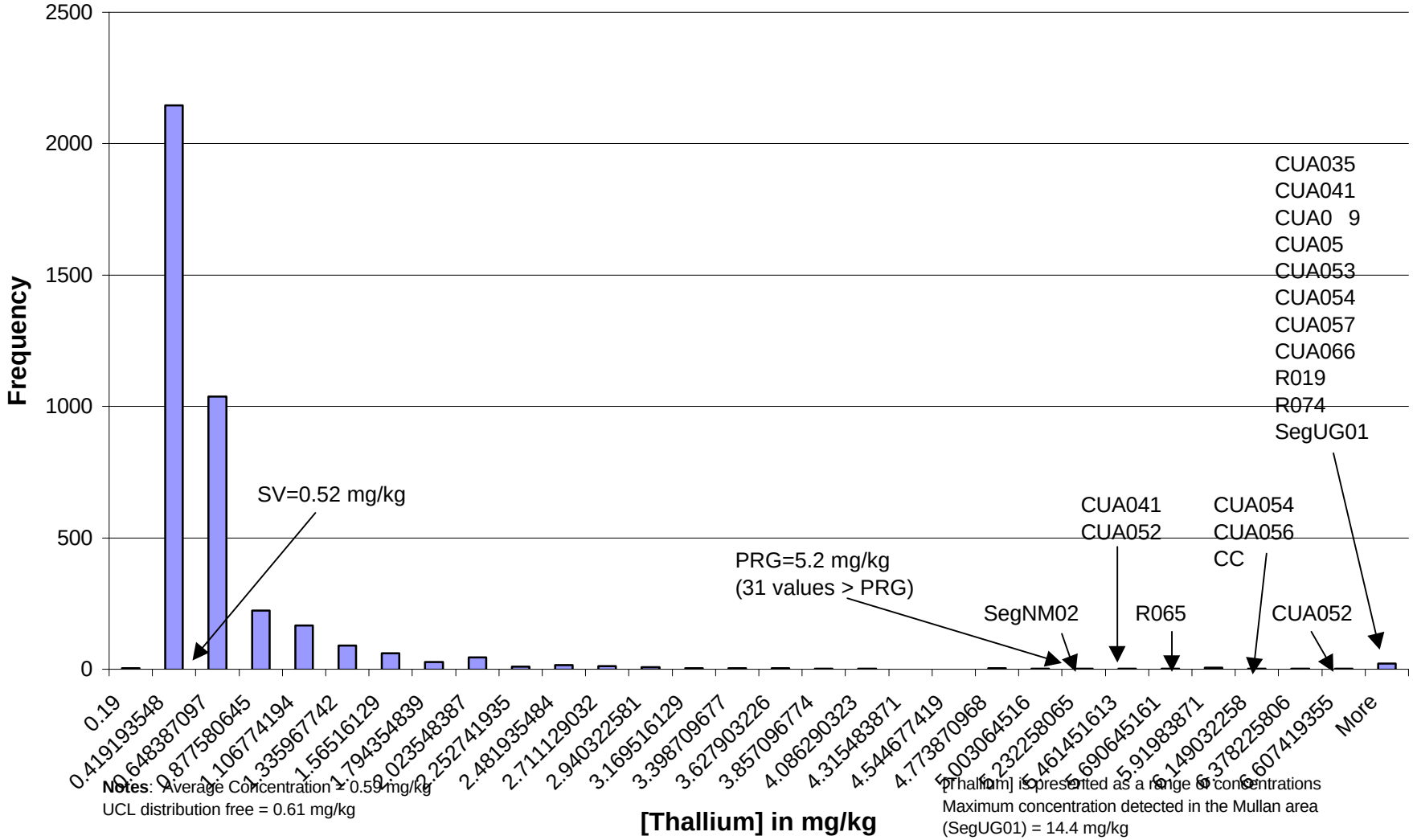
Distribution for Mercury in CSMs 1, 2, & 3



Notes: Average Concentration = 1.03 mg/kg
UCL distribution free = 1.09 mg/kg

[Mercury] is presented as a range of concentrations
Maximum concentration detected in the Mullan area

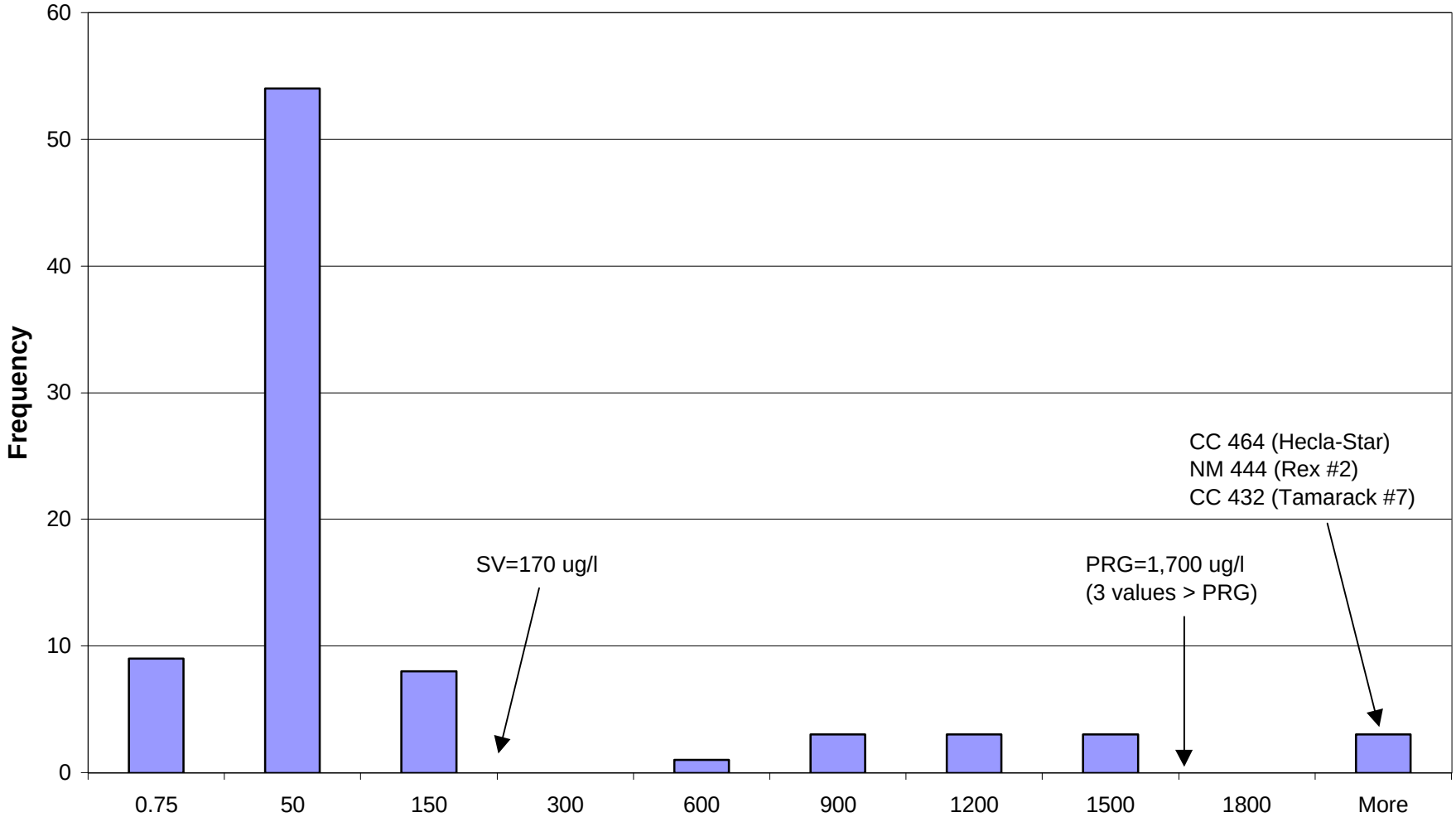
Distribution for Thallium in CSMs 1, 2, & 3



Notes: Average Concentration = 0.59 mg/kg
UCL distribution free = 0.61 mg/kg

[Thallium] is presented as a range of concentrations
Maximum concentration detected in the Mullan area
(SegUG01) = 14.4 mg/kg

Distribution for Manganese, Future Groundwater Use in CSM 1 *



Notes: *Only CSM 1 has groundwater data.
Average Concentration = 305 ug/l
UCL distribution free = 628 ug/l

[Manganese] is presented as a range of concentrations
Maximum concentration detected in Canyon Creek area,
Hecla-Star (CC 464) = 8030 ug/l

APPENDIX E

Data Set Used to Calculate Human Health Exposure Point Concentrations

Appendix E

Data Set Used to Calculate Human Health Exposure Point Concentrations

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Included in this appendix are tables listing all the concentrations for the metals of concern and the site and location ID for each sample. The following table is a reference table of all the site identifications by geographical area and CSM unit for the tables. These values were used to calculate the exposure point concentrations for the basin. Note that residential data are not included in the data set in order to protect the privacy of the homeowner.

Appendix E
Summary of Site Data by Geographical Area for the Baseline Risk Assessment

CSM 4	CSM 3		CSM 1& 2		CSM 1 & 2		CSM 2	CSM 2	CSM 2	CSM 1		CSM 1
Blackwell Island	**Lower Basin**		**Kingston**		**Side Gulches**		**Osburn[a]**	**Silverton**	**Wallace**	**Ninemile**		**Mullan**
Site ID	***Site ID***	***Description***	***Site ID***	***Description***	***Site ID***	***Description***	***Site ID***	***Site ID***	***Site ID***	***Site ID***	***Description***	***Site ID***
CUA021	CUA018	Harrison Bch	CUA077	Confluence of North and South Forks	CUA080	Elk Crk		CUA089	CUA096	CC	Canyon Crk	segUG01
	CUA033	Harrison			CUA081	Elk Crk		CUA090	CUA097	NM	Nine Mile	SF 401 - 405
	CUA035	Black Lake			segBIG04	Big Crk		CUA091	CUA098	segCC02	Canyon Crk	
	CUA036	Black Lake	PC	Pine Creek				CUA092	CUA099	segCC03	Canyon Crk	
	CUA038	Black Lake	segPC02	Pine Creek	segMG01	Twomile, Terror, & Montgomery Gulches, & Elk Creek		CUA094	CUA100	segCC04	Canyon Crk	
	CUA039	Black Lake	segPC03	Pine Creek				CUA095	CUA101	SegCC05	Canyon Crk	
	CUA041	Black Lake	SF 432	South Fork					CUA102	segNM02	Nine Mile	
	CUA043	Medimont			segMN02	Moon Creek				segNM03	Nine Mile	
	CUA044	Medimont								segNM04	Nine Mile	
	CUA045	Medimont										
	CUA046	Medimont										
	CUA047	Medimont										
	CUA048	Medimont										
	CUA049	Lane										
	CUA050	Lane										
	CUA051	Lane										
	CUA052	Lane										
	CUA053	Lane										
	CUA054	Lane										
	CUA055	Lane										
	CUA056	Lane										
	CUA057	Lane										
	CUA058	Lane										
	CUA059	Rose Lake										
	CUA060	Rose Lake										
	CUA063	Rose Lake										
	CUA064	Rose Lake										
	CUA065	Rose Lake										
	CUA066	Cataldo										
	CUA067	Cataldo										
	CUA068	Cataldo										
	CUA069	Cataldo										

Notes:

No residential sampling locations are included on this table in order to protect the privacy of the home owner.

CUA - Common use area samples;

[a]All of the sampling locations from the Osburn geographical area are residential

Data Used to Calculate Human Health Exposure Point Concentrations
Table 1.1 - Lower Basin Surface Soil

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA033	1	SS	0		8.9	J	93.7		12.7		54800		1930		4370		2.4		1800	
CUA033	2	SS	0	0.08	6.9	J	84.8		8.4		51200		1500		3810		1.4		1070	
CUA033	3	SS	0		12.7		128		16.3		74000		2470		5510		2.1		2290	
CUA033	4	SS	0	0.08	12.8		108		19.6		69700		2400		5800		1.1		2940	
CUA033	5	SS	0	0.08	11	J	96.6		14.2		64900		1980		4930		1.5		1960	
CUA035	6	SS	0	0.08	20.4		135		29.7		97700		2710		8470	J	2.1		4780	
CUA035	7	SS	0	0.08	21.6		138		27.5		93500		2580		8100	J	2		4550	
CUA035	8	SS	0	0.08	18.7		110		19.1		78300		2140		6900	J	1.4		3160	
CUA035	9	SS	0	0.08	22.4		148		28.8		104000		2530		8940	J	2.1		4730	
CUA035	10	SS	0	0.08	17.7		102		18		74300		2240		6600	J	2		3050	
CUA036	6	SS	0	0.08	23.3		189		37.7		138000		4890		10300		4		5070	
CUA036	7	SS	0	0.08	34.7		351		10.2		151000		3940		9370		5.8		1580	
CUA036	8	SS	0	0.08	27		150		37.6		128000		3020		10200		2.5		5830	
CUA036	9	SS	0	0.08	27.9		216		61.6		154000		7250		11600		5		6780	
CUA036	10	SS	0	0.08	20.2		274		10.7		96700		2840		5740		2.8		1120	
CUA038	11	SS	0	0.08	0.5	U	5.4		2.2		18100		366		1020		0.05	U	448	
CUA038	12	SS	0	0.08	4.3		13.2		6.5		15900		2420		780		0.45		800	
CUA038	13	SS	0	0.08	2.1		17.5		5.1		18600		725		1200		0.12		772	
CUA038	14	SS	0	0.08	0.48	U	5.8		1.9		16700		250		739		0.05	U	370	
CUA038	15	SS	0	0.08	1.3		36.4		2.3		20900		329		881		0.05	U	411	J
CUA039	6	SS	0	0.08	30.3		159		38.3		121000		2830		10000	J	2		5360	
CUA039	7	SS	0	0.08	35.8		167		41.3		132000		3090		12800	J	2.3		5810	
CUA039	8	SS	0	0.08	30.1		170		32.7		118000		3580		9380	J	2.8		4530	
CUA039	9	SS	0	0.08	37.9		203		43.3		142000		3550		13900	J	2.3		6030	
CUA039	10	SS	0	0.08	32.3		190		42.2		133000		3400		13100	J	2.5		5900	
CUA041	11	SS	0	0.08	0.5	U	15.3		2.3		25300		424		1290		0.12		429	
CUA041	12	SS	0	0.08	2.4		32.2		4.2		29800		829		2110		0.12		731	
CUA041	13	SS	0	0.08	3.3		75.4		4.7		43700		1540		2880		0.77		848	
CUA041	14	SS	0	0.08	0.5	U	17.5		3.6		22400		382		1450		0.12		649	
CUA041	15	SS	0	0.08	14.2		263		8.2		80000		2150		4810		2.5		1370	
CUA043	1	SS	0	0.08	19.3	J	124	J	26.6		95900		4210		10100		3.5		4510	
CUA043	2	SS	0	0.08	22.7	J	121	J	37		123000		4140		13100		3.8		6150	
CUA043	3	SS	0	0.08	20.7	J	116	J	30.5		114000		3990		12100		3.3		5150	
CUA043	4	SS	0	0.08	15.9	J	132	J	22.4		94700		3540		9500		3.1		3730	
CUA043	5	SS	0	0.08	19.1	J	128		42.2	J	115000	J	3790		12100		2.2	J	6540	
CUA044	1	SS	0	0.08	5.7	J	85.4	J	14.6		75400		2160		6870		1.8		2030	
CUA044	2	SS	0	0.08	6	J	79.9	J	17.8		82700		2330		7240		1.8		2700	
CUA044	3	SS	0	0.08	13.9	J	100	J	23.7		79600		3730		8010		2.3		3880	
CUA044	4	SS	0	0.08	9	J	95.2	J	22.8		93900		2360		9860		1.9		3520	
CUA044	5	SS	0	0.08	11.7	J	117	J	27.4		112000		2730		11000		2.8		4660	
CUA045	11	SS	0	0.08	19.4		114		18		110000		2980		7900	J	2.3		3720	J
CUA045	12	SS	0	0.08	18.9		105		25.4		95700		2840		7570	J	2.1		4520	J

Table 1.1 - Lower Basin Surface Soil

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA045	13	SS	0	0.08	21.1		167		25.9		108000		3490		7840	J	2.4		4650	J
CUA045	14	SS	0	0.08	21.2		110		35		116000		3550		9020	J	1.8		6290	J
CUA045	15	SS	0	0.08	11.95	U	130		23.4		105000		4690		7470	J	3.6		4460	
CUA046	1	SS	0	0.08	28.2		132		29.7		112000		4180		8570	J	2.3		4630	
CUA046	2	SS	0	0.08	24.4		138		37		126000		4190		9660	J	2.2		5600	
CUA046	3	SS	0	0.08	21.6		150		38.4		126000		4540		9890	J	2.3		5910	
CUA046	4	SS	0	0.08	20.8		144		31.7		126000		4050		9700	J	2.2		4990	
CUA046	5	SS	0	0.08	21.3		139		28.2		124000		3770		9500	J	2		4330	
CUA047	1	SS	0	0.08	15.6		106		18.2		72600		3290		5480		1.6		2370	
CUA047	2	SS	0	0.08	13.7		87.9		16.3		81000		2170		6050		2		2600	
CUA047	3	SS	0	0.08	10.4	J	62		9.3		60700		1710		4530		1.8		1520	
CUA047	4	SS	0	0.08	6.6	J	40.9		7.5		53300		1060		3320		1		1160	
CUA047	5	SS	0	0.08	7.9	J	64.1		7.4		68200		1420		4330		1.5		1350	
CUA048	6	SS	0	0.08	26.2		121		32.1		119000		3680		9020		2.2		5810	
CUA048	7	SS	0	0.08	25.7		117		32.1		110000		3640		8580		1.9		5910	
CUA048	8	SS	0	0.08	21.1		103		29.2		103000		3570		8100		2.5		5280	
CUA048	9	SS	0	0.08	21.5		87.9		31.1		97200		3000		7920		1.6		5600	
CUA048	10	SS	0	0.08	20.9		102		32.2		103000		3590		8280		2.3		5690	
CUA049	1	SS	0	0.08	16.6	J	106		29.6		79700		4720		8510		2.9		4390	
CUA049	2	SS	0	0.08	54.6	J	114		39.8		79100		4900		8560		2.5		4480	
CUA049	3	SS	0	0.08	16.3	J	87.9		35.9		79300		3960		9240		2.4		5570	
CUA049	4	SS	0	0.08	22.3	J	94.2		46.7		117000		3870		12800		2.3		7350	
CUA049	5	SS	0	0.08	24.7	J	91.4		37.1		82400		3740		9870		2.7		5420	
CUA050	1	SS	0	0.08	1.2	J	9.9		0.6	J	19900		44.6		722		0.05	U	123	
CUA050	2	SS	0	0.08	0.495	U	7.9		0.25	J	17200		16.7		622		0.05	U	94.9	
CUA050	3	SS	0	0.08	0.5	U	8.4		0.21	J	12700		15.3		511		0.05	U	65.7	
CUA050	4	SS	0	0.08	1.8	J	9.7		1	J	22200		271		749		0.05	U	155	
CUA050	5	SS	0	0.08	0.5	U	8.9		0.23	J	15700		17.3		582		0.05	U	74.1	
CUA051	1	SS	0	0.08	19.4	J	106		40.9		94800		3910		10900		2.6		6600	
CUA051	2	SS	0	0.08	15.5	J	139		31.6		81800		4000		9280		2.8		4490	
CUA051	3	SS	0	0.08	14.2	J	106		27.6		74900		4850		7620		2.3		3930	
CUA051	4	SS	0	0.08	16.2	J	103		36.4		80600		4440		9250		3.7		5470	
CUA051	5	SS	0	0.08	18.1	J	111		38.5		98700		4490		11000		2.9		5730	
CUA052	1	SS	0	0.08	25.9	J	112		47		116000		4370		12700		2.2		7910	
CUA052	2	SS	0	0.08	17.7	J	101		44.2		93200		4200		10200		1.9		7240	
CUA052	3	SS	0	0.08	27.9	J	151		52.8		149000		4040		16300		2.7		8710	
CUA052	4	SS	0	0.08	17.8	J	110		37.7		100000		4060		11200		2.4		6240	
CUA052	5	SS	0	0.08	15.1	J	111		35.7		91400		4560		10000		2.2		5650	
CUA053	1	SS	0	0.08	33.3		153		58.8		166000		4580		16300		2.9		9740	J
CUA053	2	SS	0	0.08	22.4		121		38.1		119000		4460		9490		2.6		6930	J
CUA053	3	SS	0	0.08	20.4		109		31.7		112000		4270		9030		2.9		5810	J
CUA053	4	SS	0	0.08	40.7		219		54.2		185000		4890		18400		2.8		9690	J
CUA053	5	SS	0	0.08	42.2		229		57.8		195000		5330		19100		2.5		10200	J

Table 1.1 - Lower Basin Surface Soil

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA054	1	SS	0	0.08	19.3		96.5		27.9		78900		4540		6220	J	2.1	J	4220	J
CUA054	2	SS	0	0.08	14.6		135		13.7		85300		3580		5700	J	4	J	2280	J
CUA054	3	SS	0	0.08	14.4	J	83.9		29.9		76300		4640		8350		3.3		4690	
CUA054	4	SS	0	0.08	16.5	J	100		41.8		96300		5120		11000		3.9		6670	
CUA054	5	SS	0	0.08	17.1	J	97.8		31		83500		4600		9590		2.7		4960	
CUA055	1	SS	0	0.08	17.5		84.8		28.9		87500		4270		7520		2.9	J	4630	J
CUA055	2	SS	0	0.08	18.1		94.3		29.9		95000		4100		7930		2.9	J	4670	J
CUA055	3	SS	0	0.08	23.5		113		33.4		102000		4910		8250		2.6	J	5380	J
CUA055	4	SS	0	0.08	22.4		96.9		29.2		85800		4170		7090		2.8	J	4730	J
CUA055	5	SS	0	0.08	20.5		95		30.6		85400		4390		7180		2.9	J	4770	J
CUA056	11	SS	0	0.08	21	J	111	J	22.5		78400		4780		8290		2.6	J	3340	
CUA056	12	SS	0	0.08	14.7	J	75.1	J	21.1		62800		3690		5960		2.2	J	2990	
CUA056	13	SS	0	0.08	20.4	J	94.5	J	23.9		73500		4850		7710		2.9	J	3360	
CUA056	14	SS	0	0.08	18.4	J	98.7	J	32.5		86600		4160		8780		2.2	J	5210	
CUA056	15	SS	0	0.08	19.4	J	113	J	29.5		89900		4370		8860		2.4	J	4400	
CUA057	1	SS	0	0.08	26.6	J	97.3		60.8		119000		4760		12900		2.5		9630	
CUA057	2	SS	0	0.08	14.5	J	91.8		20.7		68300		4060		6780		3.1		2950	
CUA057	3	SS	0	0.08	11.5	J	86.1		14.1		60500		3190		5490		2.8		1890	
CUA057	4	SS	0	0.08	20.9	J	119		44.5		95300		4610		10700		3.1		6850	
CUA057	5	SS	0	0.08	19.9	J	117		48.2		113000		5140		12000		3.3		7520	
CUA058	1	SS	0	0.08	35.8		217		59.4		177000		3910		17700		2.6		10100	
CUA058	2	SS	0	0.08	49.9		329		84.1		222000		5900		21100		1.9		13600	
CUA058	3	SS	0	0.08	30.5		213		75.8		214000		6080		21200		2.6		12500	
CUA058	4	SS	0	0.08	35.7		163		85.8		190000		5570		19600		2		13600	
CUA058	5	SS	0	0.08	20.4		115		41.2		113000		4420		8900		1.9		6700	
CUA059	6	SS	0	0.08	30.4		114		36.2		98600		4330		8040		3.2		5650	J
CUA059	7	SS	0	0.08	46.9		163		73.8		167000		6010		17100		4.1		10900	J
CUA059	8	SS	0	0.08	36.9		256		41.3		144000		5590		25200		4.1		6710	J
CUA059	9	SS	0	0.08	18.7		104		41.7		106000		4140		8400		4	J	6810	
CUA059	10	SS	0	0.08	12.5		75.1		26.7		74200		2970		6150		3.1	J	4580	
CUA060	6	SS	0	0.08	56.5		154		82.1		188000		5580		19300		2.5		12100	J
CUA060	7	SS	0	0.08	31.8		107		53.3		131000		4960		13300		2.9		8300	J
CUA060	8	SS	0	0.08	37.4		110		57.9		136000		5140		14400		2.8		8910	J
CUA060	9	SS	0	0.08	29.8		103		29.1		104000		4420		8320		3		4720	J
CUA060	10	SS	0	0.08	41.5		123		53.6		135000		4420		14200		4.2		8550	J
CUA063	1	SS	0	0.08	17.8	J	116	J	19.8		81100		4300		8460		2.8		2790	J
CUA063	2	SS	0	0.08	34.9	J	150	J	48.8		131000		4930		13900		1.4		7480	J
CUA063	3	SS	0	0.08	45.9	J	187	J	86.4		184000		6390		20000		3.5		14200	J
CUA063	4	SS	0	0.08	19.7	J	109	J	26.8		82600		4290		9390		1.6		3720	J
CUA063	5	SS	0	0.08	19.3	J	99.9	J	24.9		75200		4040		7990		1.9		3310	J
CUA064	1	SS	0	0.08	17.9	J	86.1	J	40.9		63700		3560		6680		2	J	6320	
CUA064	2	SS	0	0.08	20.4	J	100	J	32.3		85000		3720		9300		2.4	J	5240	
CUA064	3	SS	0	0.08	17.5	J	97.6	J	34.6		70700		3360		7190		2.5	J	5310	

Table 1.1 - Lower Basin Surface Soil

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA064	4	SS	0	0.08	18.8	J	105	J	38.5		79500		3920		8730		2.3	J	6910	
CUA064	5	SS	0	0.08	17.4	J	82.5	J	31.5		67000		3490		6790		2	J	4830	
CUA065	16	SS	0	0.08	30.9		94.8		37		94800		3530		7120	J	1.1		6370	
CUA065	17	SS	0	0.08	10.4	U	70		21.4		62200		3280		4810	J	2.8		3750	
CUA065	18	SS	0	0.08	10.55	U	115		20.3		86100		3060		5940	J	3.4		3590	
CUA065	19	SS	0	0.08	10.75	U	82.5		22		72000		3400		5440	J	2.7		3840	
CUA065	20	SS	0	0.08	15	U	149		15.3		100000		3460		6590	J	11		3030	
CUA066	1	SS	0	0.08	36.1	J	256	J	15.5	J	139000		2670		10900	J	1.1		2520	J
CUA066	2	SS	0	0.08	23.9	J	160	J	8.2		89200		3520		9180		1.5		5060	
CUA066	3	SS	0	0.08	58.6	J	492	J	21.3	J	145000		3470	J	12800		1.1	J	3530	J
CUA066	4	SS	0	0.08	19.9	J	144	J	15		80500		3060		7810		1.6	J	3190	
CUA066	5	SS	0	0.08	22.2	J	151	J	11.4		80900		2940		7770		1.9	J	1850	
CUA067	1	SS	0	0.08	2.5		21		1.5		38400		355		1350		0.055	U	267	
CUA067	2	SS	0	0.08	0.5	U	13.5		0.38		31600		123		979		0.05	U	148	
CUA067	3	SS	0	0.08	3		27.2		2.5		31700		536		1410		0.56		450	
CUA067	4	SS	0	0.08	7		59.7		7.7		29600		1270		1480		1.6		1470	
CUA067	5	SS	0	0.08	1.4		17.9		1.4		31800		321		1150		0.29		315	
CUA068	1	SS	0	0.08	16.8	J	72.9		10.8		46300		3050		3860		1.8		1640	
CUA068	2	SS	0	0.08	21.7	J	88.8		13.5		62700		3740		5920		1.7		1940	
CUA068	3	SS	0	0.08	17.9	J	83.8		12.6		54300		3280		4970		1.7		1780	
CUA068	4	SS	0	0.08	13.1	J	64.3		8.2		38300		2270		3220		1		1320	
CUA068	5	SS	0	0.08	13.1	J	72		10		45800		2190		3730		0.86		1590	
CUA069	1	SS	0	0.08	24	J	125		13.9		76561	J	3460		5630		1.6		2070	
CUA069	2	SS	0	0.08	25.1	J	135		17.6		91800	J	3580		6910		1.5		2530	
CUA069	3	SS	0	0.08	26.8	J	150		13.9		85966	J	3690		5750		1.6		1770	
CUA069	4	SS	0	0.08	24.4	J	127		10.2		87495	J	2430		7290		0.85		1610	
CUA069	5	SS	0	0.08	22	J	135		14		83007	J	2740		7090		1.4		1680	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 1.2 - Lower Basin Sediment

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA018	1	SD	0		1.3	J	3.9		0.6		11500	J	121	J	195	J	0.07	UJ	184	J
CUA018	2	SD	0	0.5	2	J	9.4		1.6		16200	J	282	J	348	J	0.11	UJ	452	J
CUA018	3	SD	0		5.7	J	22.3		2.8		27400	J	429	J	759	J	0.105	UJ	577	J
CUA018	4	SD	0	0.5	2.1	J	10.4		1.9		15400	J	348	J	467	J	0.11	UJ	587	J
CUA018	5	SD	0	0.5	3.7	J	14.2		2.6		23900	J	594	J	592	J	0.54	J	522	J
CUA018	6	SD	0	0.5	3.3	J	20		4.8		18900	J	1030	J	1700	J	0.18	UJ	1010	J
CUA018	7	SD	0	0.5	4.5	J	19		2.2		17200	J	992	J	783	J	0.2	UJ	562	J
CUA018	15	SD	0	0.5	5.9	J	18		3.6		38700		1740		1370		0.73		890	
CUA018	16	SD	0	0.5	0.5	U	3		0.29	J	12900		70.7		233		0.055	U	75.1	
CUA018	17	SD	0	1	4.6	J	8.4		2.4		15500		526		779		0.05	U	560	
CUA018	18	SD	0	1	1.5	J	4.9		1.4		17800		183		377		0.05	U	215	
CUA018	19	SD	0	1	0.5	U	2.6		0.24	J	12500		33		197		0.05	U	45.1	
CUA018	20	SD	0	1	0.5	U	3.9		0.39	J	16700		53.7		259		0.05	U	70	
CUA018	21	SD	0	1	0.5	U	3.8		0.1	U	16100		21.1		256		0.05	U	37.7	
CUA018	22	SD	0	1	1	J	6.8		1.1		16200		211		341		0.05	U	256	
CUA018	23	SD	0	1	4.5	J	11.6		1.5		23100		710		805		0.15		389	
CUA018	24	SD	0	1	1.2	J	4.5		0.55	J	14600		139		373		0.055	U	90.9	
CUA018	25	SD	0	1	8	J	35		4.5		27500		2990		1810		1.4		1380	
CUA018	26	SD	0	1	55.6		158		14.8		54900		12100		4780		3.1		4310	
CUA018	27	SD	0	1	0.49	U	3.3		0.34	J	11300		96.8		243		0.05	U	69.3	
CUA018	28	SD	0	1	3.4	J	23.8		3.2		20400		1340		875		0.38		757	
CUA018	29	SD	0	1	2.4	J	16.3		1.6		18800		1040		586		0.32		462	
CUA018	30	SD	0	1	3.5	J	22.2		2.7		20300		1070		797		0.22		772	
CUA018	31	SD	0	1	1.8	J	10.2		1.2		17700		557		432		0.05	U	306	
CUA018	32	SD	0	1	15.6		58		7.4		35500		4770		2820		1.4		1960	
CUA018	33	SD	0	1	3.4	J	16.3		2		17100		1120		728		0.43		465	
CUA033	6	SD	0	1	19.9		254		18.5		102000		2800		6900		2.8		2990	
CUA033	7	SD	0	1	15.4		154		20.2		90100		2650		6850		2.4		3180	
CUA033	8	SD	0	1	14.1		161		14.2		83000		2780		5590		2.7		2360	
CUA033	9	SD	0	1	13		152		16		74200		2520		5670		2.4		2430	
CUA033	10	SD	0	1	15.2		142		16.9		80100		2290		6060		3.4		2580	J
CUA035	1	SD	0	1	17.4		148		19.1		82200		2310		6900		3.3		2750	
CUA035	2	SD	0	1	18.6		155		24.3		102000		2600		8380		3.2		3680	
CUA035	3	SD	0	1	16.1		123		15.6		96500		2260		7760		3.9		2330	
CUA035	4	SD	0	1	14.3		133		22.1		104000		2550		8510		3.1		3440	
CUA035	5	SD	0	1	8.9	J	67.3		13.3		55400		1460		4650		2.6		2090	
CUA035	11	SD	0	0.5	26.5		124		27.4		98700		2190		8650	J	1.6		4700	
CUA035	12	SD	0	0.5	20.8		105		24.9		88200		2170		7860	J	1.7		4270	
CUA035	13	SD	0	0.5	19.7		113		23.3		92000		1850		8130	J	1.8		4030	
CUA035	14	SD	0	0.5	20.4		98.1		22.8		75600		2360		6810	J	2		3800	
CUA035	15	SD	0	0.5	25.4		146		36.1		146000		2500		13000		2.3		5720	
CUA036	1	SD	0	1	23.7		168		29.6		126000		3160		10000		3.3		4400	

Table 1.2 - Lower Basin Sediment

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA036	2	SD	0	1	21.9		250		28.2		123000		3470		9560		4.4		3650	
CUA036	3	SD	0	1	16.7		132		28.4		106000		2710		8940		2.6		4340	
CUA036	4	SD	0	1	20.7		209		34.3		125000		2940		9880		3.4		4900	
CUA036	5	SD	0	1	23.5		154		33		144000		2800		13000		2.8		5120	
CUA036	11	SD	0	0.5	24		159		33.1		128000		3030		10100		2		5310	
CUA036	12	SD	0	0.5	27.3		167		35.4		168000		2560		14800		2.6		5680	
CUA036	13	SD	0	0.5	23.3		169		40.7		143000		3580		11400		2.3		6390	
CUA036	14	SD	0	0.5	28.6		162		35.8		159000		2690		14100		4.4		5610	
CUA036	15	SD	0	0.5	16.2		119		28.3		102000		3250		8190		2		4330	
CUA038	1	SD	0	0.5	0.5	U	3.6		0.28		7370		117		271		0.05	U	106	
CUA038	2	SD	0	0.5	0.495	U	1.8		0.1	U	6080		92.5		154		0.05	U	64.7	
CUA038	3	SD	0	0.5	0.495	U	1.5		0.1	U	4450		18.3		141		0.05	U	14.3	
CUA038	4	SD	0	0.5	0.495	U	2.4		0.1	U	7870		22.5		97.5		0.05	U	19.5	
CUA038	5	SD	0	0.5	0.495	U	4		0.1	U	10000		27.8		92.3		0.045	U	30.8	
CUA039	1	SD	0	1	23		153		29.7		129000		2980		10300		2.3		4510	
CUA039	2	SD	0	1	28.2		188		37.3		165000		2680		14200		2.3		5880	
CUA039	3	SD	0	1	32.1		148		37.7		144000		2600		14300	J	2.2		5530	
CUA039	4	SD	0	1	25.5		172		33.5		126000		2860		12500	J	2.4		4870	
CUA039	5	SD	0	1	23.8		179		26.7		104000		2820		8200	J	3.4		3150	
CUA039	11	SD	0	0.5	29.2		318		28.9		103000		3890		7740	J	2.8		4180	
CUA039	12	SD	0	0.5	25.8		118		30.5		83300		4250		6490	J	2		4270	
CUA039	13	SD	0	0.5	34.3		143		40.3		141000		2520		14100	J	2.1		5750	
CUA039	14	SD	0	0.5	34.7		153		36.5		125000		3340		10100	J	1.7		5430	
CUA039	15	SD	0	0.5	35.4		122		30.9		116000		7480		9400	J	3.6		5020	
CUA041	1	SD	0	0.5	0.5	U	5.9		1.9		17100		154		386		0.05	U	421	
CUA041	2	SD	0	0.5	0.49	U	4.3		0.38		15900		38.6		138		0.05	U	195	
CUA041	3	SD	0	0.5	5.5		68.9		11.9		55200		3420		4120		1.5		2550	
CUA041	4	SD	0	0.5	0.5	U	17.9		2.6		21700		997		1160		0.39		510	
CUA041	5	SD	0	0.5	0.5	U	7		1.9		16200		288		322		0.12		378	
CUA045	1	SD	0	0.5	26.7		142		39.4		135000		3290		9970	J	2.1		6990	J
CUA045	2	SD	0	0.5	21.1		130		41.9		138000		3790		13900	J	1.5		7410	J
CUA045	3	SD	0	0.5	26.2		118		35.7		127000		3110		9660	J	1.6		6490	J
CUA045	4	SD	0	0.5	21.6		136		32.1		139000		2740		13600	J	1.6		6040	J
CUA045	5	SD	0	0.5	18.8		152		28		155000		2620		14000	J	2		5310	J
CUA047	6	SD	0	0.5	16		122		18.7		70900		2180		5430		1.2		2220	
CUA047	7	SD	0	0.5	27.5		166		22.3		112000		3590		8970		1.2		3260	
CUA048	1	SD	0	1	33.1		147		35.4		127000		4090		12500	J	2.8		4960	
CUA048	2	SD	0	1	35.5		153		40.1		131000		4260		13300	J	2.6		5720	
CUA048	3	SD	0	1	36.1		165		33		144000		4170		14000	J	3		4610	
CUA048	4	SD	0	1	28		160		26.2		111000		4400		8040	J	2.9		3630	
CUA048	5	SD	0	1	41.1		194		45.7		174000		5000		16400	J	2.6		6830	
CUA048	11	SD	0	0.5	30.2		137		42.3		150000		3770		14400		1.9		7400	
CUA048	12	SD	0	0.5	18.5		111		50.6		87700		3160		6650		1.6		5390	

Table 1.2 - Lower Basin Sediment

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA048	13	SD	0	0.5	25.3		128		55		107000		3620		8040		2.5		6650	
CUA048	14	SD	0	0.5	28.5		127		40.1		129000		3010		9470	J	1.8	J	7160	J
CUA048	15	SD	0	0.5	35.3		170		44.2		140000		4370		11900	J	1.6	J	7810	J
CUA049	6	SD	0	1	22.8		112		20.4		109000		3510		8470		2.5	J	4020	
CUA049	7	SD	0	1	36.1		168		43.8		172000		4670		16800		2.3	J	7920	
CUA049	8	SD	0	1	13.35	U	99.1		33.3		115000		3460		9190		1.9	J	6300	
CUA049	9	SD	0	1	12.55	U	101		37.1		110000		3670		8890		2.1	J	6500	
CUA049	10	SD	0	1	13.4		85.8		25.5		114000		3060		8950		2.5		4450	J
CUA049	11	SD	0	0.5	28.3		148		55.4		150000		4110		15700		2	J	9230	
CUA049	12	SD	0	0.5	27.4		155		46.7		163000		3910		16300		2.3	J	8180	
CUA049	13	SD	0	0.5	30.5		129		40.1		146000		3320		14500		2.1	J	7030	
CUA049	14	SD	0	0.5	28.9		123		33.9		143000		3390		14600		2.4	J	6280	
CUA049	15	SD	0	0.5	27		115		33.6		154000		2960		15700		2	J	6380	
CUA051	6	SD	0	1	13.8	J	128		25.7		84500		4260		9040		2.4		3790	
CUA051	7	SD	0	1	13.5	U	128		30.3		121000		3870		9410		2.8	J	5270	
CUA051	8	SD	0	1	8.3	U	124		18.1		102000		3930		7860		4.8	J	3010	
CUA051	9	SD	0	1	11.65	U	132		35.2		125000		3460		9740		2.1	J	6250	
CUA051	10	SD	0	1	22.6		122		36.4		132000		3670		13300		2.5	J	6570	
CUA051	11	SD	0	0.5	32		114		47.3		142000		3410		14600		2.1	J	8290	
CUA051	12	SD	0	0.5	40.7		165		70.5		183000		4450		18700		1.9	J	11500	
CUA051	13	SD	0	0.5	25.3		118		39.2		140000		3430		14200		2.4	J	7070	
CUA051	14	SD	0	0.5	25.6		122		40.9		114000		3790		9000		2.1	J	7100	
CUA051	15	SD	0	0.5	28.3		127		52.5		136000		4000		13700		2.1	J	8760	
CUA052	6	SD	0	1	19.8	J	124		31		128000		3960		13800		3.3		4700	
CUA052	7	SD	0	1	17	J	148		36.1		109000		3900		11100		3.3		5240	
CUA052	8	SD	0	1	22.1	J	112		42.4		124000		4460		13400		3.5		6830	
CUA052	9	SD	0	1	21	J	134		38.5		136000		4150		14800		3		6300	
CUA052	10	SD	0	1	28	J	180		60.7		159000		5960		17100		3.3		10200	
CUA052	11	SD	0	0.5	21.4	J	123		49.1		134000		3920		14600		2.6		8130	
CUA052	12	SD	0	0.5	25.3	J	138		60.6		138000		4140		15200		2.1		10500	
CUA052	13	SD	0	0.5	49.1	J	145		74.7		163000		4350		17800		4.5		11900	
CUA052	14	SD	0	0.5	25.6	J	111		50.6		102000		4030		10800		3		7120	
CUA052	15	SD	0	0.5	46	J	144		77.3		169000		4860		18300		4.3		12700	
CUA053	6	SD	0	1	27.3		164		42.2		153000		4800		14800		3.3		7380	J
CUA053	7	SD	0	1	39.9		178		54.9		190000		5760		18900		2.7		9620	J
CUA053	8	SD	0	1	34.3		172		49.1		176000		4950		17700		2.9		8800	J
CUA053	9	SD	0	1	29.7		162		39		162000		4680		16200		3.4		7060	J
CUA053	10	SD	0	1	19.6	J	120		36.3		131000		3900		12900		2.8		6000	J
CUA053	11	SD	0	0.5	48.5	J	171		100		197000		5530		22000		2.7		17200	
CUA053	12	SD	0	0.5	36	J	137		88.9		182000		4800		20500		3.5		15000	
CUA053	13	SD	0	0.5	19.8	J	108		57.5		86800		4060		9810		2.7		6950	
CUA053	14	SD	0	0.5	36.1	J	106		79.6		188000		4050		21600		3.3		13300	
CUA053	15	SD	0	0.5	35.8	J	56		88.6		94500		15000		11000		7		14000	

Table 1.2 - Lower Basin Sediment

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA054	6	SD	0	1	14.1	J	108		22.9		136000		6550		15000		4		2630	
CUA054	7	SD	0	1	31.1	J	112		75.4		165000		4700		18600		4.7		12800	
CUA054	8	SD	0	1	26.6	J	115		73.6		157000		4640		17600		4		12800	
CUA054	9	SD	0	1	17.3	J	136		59.6		133000		7120		13600		4.9		7260	
CUA054	10	SD	0	1	16.4	J	133		31.3		112000		4780		11700		4.2		4920	
CUA054	11	SD	0	0.5	20.9	J	84.4		56.6		121000		4030		13400		4.1		9490	
CUA054	12	SD	0	0.5	44.2		111		85.9		180000		4020		18900	J	2	J	12200	J
CUA054	13	SD	0	0.5	51.8		113		87.1		189000		4450		20200		2.7		17400	J
CUA054	14	SD	0	0.5	37.9		132		70.1		170000		4490		18300		2.7		11900	J
CUA054	15	SD	0	0.5	24.5		102		48		123000		3580		12700		3		8360	J
CUA055	6	SD	0	1	29.8		141		44.7		149000		4530		15800		3.6	J	7280	J
CUA055	7	SD	0	1	34.7		143		63.1		145000		4870		15300		2.6	J	9950	J
CUA055	8	SD	0	1	40		150		59		149000		4980		15600		2.6	J	9320	J
CUA055	9	SD	0	1	29.2		126		64		143000		4430		13200	J	2	J	9260	J
CUA055	10	SD	0	1	31.4		107		58		154000		4330		16000	J	2.3	J	9370	J
CUA055	11	SD	0	0.5	42.8		154		96.7		194000		5100		20400	J	1.8	J	17800	J
CUA055	12	SD	0	0.5	36.5		164		94.3		186000		5410		19900	J	1.9	J	13900	J
CUA055	13	SD	0	0.5	47		143		83		160000		4970		16500	J	2.5	J	12100	J
CUA055	14	SD	0	0.5	41.9		137		83.1		174000		4760		17700	J	1.8	J	12100	J
CUA055	15	SD	0	0.5	44.5		123		77.4		155000		4720		15800	J	1.9	J	11400	J
CUA056	1	SD	0	0.5	31.7	J	130	J	55.1		125000		4450		13100		1.8	J	9410	
CUA056	2	SD	0	0.5	9	J	44.7	J	19.9		48000		1750		4270		0.55	J	3090	
CUA056	3	SD	0	0.5	22	J	99.8	J	44.1		88100		4740		9310		1.7	J	6920	
CUA056	4	SD	0	0.5	27.1	J	105	J	54.1		115000		4400		11800		1.5	J	8610	
CUA056	5	SD	0	0.5	35.4	J	138	J	54.4		117000		5000		11700		2.3	J	8830	
CUA057	6	SD	0	1	20.8	J	139		26.8		118000		5610		11500		2.9		3850	
CUA057	7	SD	0	1	42.7	J	127		94.4		180000		5360		20000		3.3		15300	
CUA057	8	SD	0	1	46.5	J	153		87.6		178000		6080		19600		4		13700	
CUA057	9	SD	0	1	40.3	J	151		87.2		183000		5280		20300		4.1		14200	
CUA057	10	SD	0	1	52.4		208		80.9		203000		5020		18200		2.5		12800	
CUA057	11	SD	0	0.5	67.4		146		104		256000		4760		25500		2.5		21800	
CUA057	12	SD	0	0.5	51.2		137		105		222000		5200		22200		1.8		20900	
CUA057	13	SD	0	0.5	42.5		126		82.9		190000		4080		19200		1.3		13800	
CUA057	14	SD	0	0.5	48.6		143		95.6		212000		4550		20900		2.1		19000	
CUA057	15	SD	0	0.5	38.8		108		83.3		206000		4010		20400		2.8		16800	
CUA058	6	SD	0	1	21		139		39.6		142000		4500		15200		2.4		6280	
CUA058	7	SD	0	1	26.3		126		42.9		132000		4610		14000		2.8		6720	
CUA058	8	SD	0	1	23.8		135		52.9		148000		5280		26400		2.6		8030	
CUA058	9	SD	0	1	28.7		109		56.5		146000		4060		14000		3		8540	
CUA058	10	SD	0	1	28.4		132		54.2		138000		4680		14400		2.4		8010	
CUA058	11	SD	0	0.5	49.4		185		80.9		190000		5200		20400		2.9	J	13000	J
CUA058	12	SD	0	0.5	48.6		184		84.1		185000		5300		19700		2.7	J	12900	J
CUA058	13	SD	0	0.5	39.3		117		79.6		184000		4010		19800		2.9	J	13200	J

Table 1.2 - Lower Basin Sediment

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA058	14	SD	0	0.5	50		136		88		176000		4560		18600		2.5	J	17900	J
CUA058	15	SD	0	0.5	39.5		129		81.7		179000		4340		18900		2.4	J	13300	J
CUA059	1	SD	0	1	44.2		209		67.4		162000		5170		16900		4.7		9530	J
CUA059	2	SD	0	1	36.9		148		32.8		120000		4740		9470		13.5		5300	J
CUA059	3	SD	0	1	44.5		173		51.6		150000		6070		15200		4.2		8160	J
CUA059	4	SD	0	1	36.2		136		43.8		132000		6120		13300		3.5		5780	J
CUA059	5	SD	0	1	39.3		111		51.4		139000		4220		15000		3		8090	J
CUA059	11	SD	0	0.5	11.8		87.9		27.7		66500		2930		4930		3.9	J	3970	
CUA059	12	SD	0	0.5	0.5	U	9.5		4.3		13500		282		708		0.34	J	589	
CUA059	13	SD	0	0.5	13		72.1		34.4		90100		1860		7260		2.6	J	5960	
CUA059	14	SD	0	0.5	13.7		73.9		32.8		75400		3340		5710		3.3	J	5940	
CUA059	15	SD	0	0.5	24.3		88.3		62.3		105000		3060		8290		2.6	J	8260	
CUA060	1	SD	0	1	50.8		375		30.1		152000		3590		13700		4.2		4650	J
CUA060	2	SD	0	1	46		162		56.1		182000		5310		18000		4.7		8780	J
CUA060	3	SD	0	1	48.5		146		74.6		217000		4470		21000		3		11300	J
CUA060	4	SD	0	1	51.5		304		51.7		189000		5910		18600		15.2		8690	J
CUA060	5	SD	0	1	36.9		146		49.8		132000		4790		13000		2.4		7730	J
CUA060	11	SD	0	0.5	16.1		85.5		36.2		87000		2610		6890		3.1	J	5600	
CUA060	12	SD	0	0.5	13.5		93.2		24.8		72500		3390		5130		3.6	J	4210	
CUA060	13	SD	0	0.5	24.4		111		67		115000		3510		13000		2.2	J	9850	
CUA060	14	SD	0	0.5	0.5	U	6.3		1		11400		34		112	J	0.05	U	235	
CUA060	15	SD	0	0.5	5.6		25.6		3.3		24800		705		1280	J	0.9		826	
CUA063	6	SD	0	1	38.8	J	163	J	35.7		179000		9070		19200		3.6		5580	J
CUA063	7	SD	0	1	33	J	165	J	30.4		137000		6390		12700		1.9		4400	J
CUA063	8	SD	0	1	31	J	151	J	50.6		135000		5940		13500		4.8		7170	J
CUA063	9	SD	0	1	15.3		96.7		23.1		81100		3760		6340	J	4.5		4060	
CUA063	10	SD	0	1	34.7		182		62.7		168000		7500		16500	J	3		10700	
CUA063	11	SD	0	0.5	10.4		45.2		12.3		58100		2830		3780	J	2.3		2550	
CUA063	12	SD	0	0.5	11.9		46.1		12.9		73000		5410		5210	J	3.4		2910	
CUA063	13	SD	0	0.5	73.7		84.3		25.3		124000		29200		4430	J	23		7340	
CUA063	14	SD	0	0.5	53		68.1		4.7		116000		16800		3540	J	20.2		4900	
CUA063	15	SD	0	0.5	24.6		73.4		17.5		98100		8680		6140	J	9.1		4230	
CUA065	1	SD	0	0.5	26.7	J	94.7		41.2		71800		3730		6270		2.3	J	6680	
CUA065	2	SD	0	0.5	31	J	112		38.2		93700		3120		9140		1.9	J	6330	
CUA065	3	SD	0	0.5	25.8	J	94.1		67.5		79600		4590		7340		1.8	J	7690	
CUA065	4	SD	0	0.5	12.2	J	51.8		32.4		50500		2420		4160		1.6	J	2870	
CUA065	5	SD	0	0.5	17.7	J	73.6		29.1		75800		3490		7280		2.9	J	4400	
CUA065	11	SD	0	1	29.6	J	137		52.3		126000		4470		11400		3.4	J	8340	
CUA065	12	SD	0	1	20.9	J	136		25		85600		3400		8080		3.6	J	3750	
CUA065	13	SD	0	1	42.7	J	147		59.2		117000		4810		10900		2.7	J	9560	
CUA065	14	SD	0	1	29.1	J	154		40.3		111000		4500		9840		3.6	J	6140	
CUA065	15	SD	0	1	8.35	U	94.6		16.5		63700		3150		4500	J	3		2840	
CUA068	6	SD	0	1	15.1	J	85.2		12.5		53500		2480		4830		1.4		1690	

Table 1.2 - Lower Basin Sediment

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA068	7	SD	0	1	17	J	95.8		13.3		66400		2640		6320		1.2		1920	
CUA068	8	SD	0	1	14.1	J	81.4		9.3		47000		2340		3940		2.5		1400	
CUA068	9	SD	0	1	10.9		63.5		8.8		39100		1950		2980		2		1440	
CUA068	10	SD	0	1	14.8		84.8		4.8		57000		2340		3720		2.4		1440	
CUA068	11	SD	0	0.5	16.6	J	92.8	J	18.2		64000		2660		6390		1.1	J	1990	
CUA068	12	SD	0	0.5	16.1	J	79.7	J	13.2		62000		2400		5490		1.1	J	1730	
CUA068	13	SD	0	0.5	17.8	J	78.6	J	30.9		58100		2540		5000		1.2	J	2790	
CUA068	14	SD	0	0.5	14.7	J	69		24.3		47700		2450		4100		0.94		2310	
CUA068	15	SD	0	0.5	15.1	J	87.8		15.6		54400		2510		5450		1.6		1720	
CUA069	6	SD	0	1	24.7	J	135		20.7		62159		3600		6680		1.9		2540	
CUA069	7	SD	0	1	17.9	J	141		18.2		70286		2950		7300		1.5		2250	
CUA069	8	SD	0	1	21		117		26.4		53300		2630		4500		2.6		2120	
CUA069	9	SD	0	1	17.5		120		9.7		55200		2100		4150		2		1890	
CUA069	10	SD	0	1	21		110		8.1		69000		2660		5560		2.3		1700	
CUA069	11	SD	0	0.5	10.5		132		20.9		79100		3010		6560		1.8		2710	
CUA069	12	SD	0	0.5	13.9		156		21		71000		2750		6300		1.9		2650	
CUA069	13	SD	0	0.5	15.5		143		19.4		70900		2680		5800		1.9		2590	
CUA069	14	SD	0	0.5	12.8		150		19.7		65500		2710		5660		2		2510	
CUA069	15	SD	0	0.5	7.8		79.3		25.3		48200		2060		5010		1.2		2930	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 1.3 - Lower Basin Surface Water

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA018	1	SW			1.4		8.6		3.6		10800	J	315		517		0.23		519	
CUA018	2	SW			1.3		6.8		1.8		2230	J	182		187		0.1	U	238	
CUA018	3	SW			1.5		8		1.3		3520		171		210		0.1	U	209	
CUA018	4	SW			3.5		15.3		2.4		8450		469		455		0.35		748	
CUA018	5	SW			0.99		6.3		0.5	U	1650	J	117		124		0.1	U	181	
CUA018	6	SW			1.4		8.2		1.8		3490	J	298		240		0.1	U	360	
CUA018	7	SW			2.3		9.7		2		3530	J	319		285		0.1	U	347	
CUA035	11	SW			5.4	U	17.2		3.7	J	21100		661		2460		0.6		604	J
CUA035	12	SW			5.2	U	22.5		4.7	J	42300		1170		4960		0.34		809	
CUA035	13	SW			9.1	U	40.5		7.3	J	88600		2210		11100		0.71		1280	
CUA035	14	SW			10.5	U	33.9		7.1	J	65200		1490		7730		0.24		1270	
CUA035	15	SW			13.7	U	52		13.2	J	95500		2810		12100		1.1		2030	
CUA036	11	SW			21.5		85.5		56.3		111000		10400		13300		11.1		5400	
CUA036	12	SW			9.3		140		27.9		175000		9280		23400		0.38		4660	
CUA036	13	SW			23.3		130		206		285000		56500		35300		4.9		19700	
CUA036	14	SW			6.8		75.5		30.7		94900		8970		13500		2		4420	
CUA036	15	SW			12.7		98.7		20.6		131000		6310		17300		0.46		3480	
CUA038	1	SW			2.3		3.4		0.5	U	3120		199		357		0.1	U	120	
CUA038	2	SW			3.9		6.2		2.9		9330		876		715		0.1	U	382	
CUA038	3	SW			1.6		4		0.5	U	6170		166		636		0.1	U	114	
CUA038	4	SW			2		4.6		1.8		13200		433		961		0.1	U	271	
CUA038	5	SW			1.7		3.8		0.5	U	7920		190		505		0.1	U	145	
CUA039	11	SW			16		74.9		170		329000		29600		40900		14.1		11100	
CUA039	12	SW			12.7		54.1		26.5		82900		7190		9400		1		2670	
CUA039	13	SW			10.8		53.6		26.5		127000		7390		17000		0.91		3770	
CUA039	14	SW			18.3		128		75.8		294000		31700		33700		6.3		10300	
CUA039	15	SW			21.3		49.3		137		364000		38800		47300		18		12100	
CUA041	1	SW			11.5		34.6	J	65.8		93000		8230		10000		4.3	J	5610	
CUA041	2	SW			12.4	J	31.6	J	52.4	J	129000		11400		13600		4.1	J	10500	
CUA041	3	SW			13.1	J	31.8	J	59.8	J	159000		15600		16400		6.8	J	8500	
CUA041	4	SW			8.7		16.5	J	38.1		93200		9010		9430		3.7	J	4080	
CUA041	5	SW			5.8		9.3	J	11.6		42100		2710		2420		1.8	J	1760	
CUA045	1	SW			6.8	U	35.8		8.8		108000		5600		13500		0.84	J	1840	
CUA045	2	SW			3.3	U	8.4		3.5		5750		776		614		0.12	UJ	674	
CUA045	3	SW			3.7	U	8.5		3.7		6750		864		816		0.97	J	589	
CUA045	4	SW			6.2	U	38.9		23.2		87200		6520		10700		1.2	J	3550	
CUA045	5	SW			3.8	U	11.3		7.4		8140		1310		879		0.245	UJ	1260	
CUA047	6	SW			6.7		57		7.6		23700	J	1230		2310		0.55		683	
CUA047	7	SW			21.4		173		14.1		46300	J	3280		4750		1.7		1550	
CUA047	8	SW			4.1		17.3		4.6		12000	J	534		1320		0.26		603	
CUA047	9	SW			5.5		25.2		2.8		10700	J	452		1120		0.23		451	
CUA047	10	SW			12.6		136		15.7		51900	J	2420		5310		0.98		1800	

Table 1.3 - Lower Basin Surface Water

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA048	11	SW			27	J	130	J	56.8	J	120000		16600	J	13100		3.6		7870	
CUA048	12	SW			25	J	263	J	338	J	251000		35600	J	22500		13.5		29500	
CUA048	13	SW			27.1	J	600	J	1200	J	1000000		81500	J	84900		43.9		116000	
CUA048	14	SW			26.9	J	254	J	66.2	J	182000		27500	J	20600		3.6		10900	
CUA048	15	SW			26.1	J	178	J	166	J	302000		45100	J	32100		7.1		26200	
CUA049	11	SW			16.4		69.3		68.9		225000		21300		27100		4		8360	
CUA049	12	SW			12.8	U	41.8		31		148000		13400		18100				5080	
CUA049	13	SW			14		41.8		25.5		155000		11900		19700		1.6		3520	
CUA049	14	SW			7.7	U	25.5		12.6		84100		8090		11100		0.97		2420	
CUA049	15	SW			12.1	U	44		33.9		129000		18900		17600		1.9		6060	
CUA051	11	SW			17.3		55.6		34		219000		21300		27900		1.4		6290	
CUA051	12	SW			15.7		56.7		47.9		244000		29400		32000		1.9		8410	
CUA051	13	SW			20.9		70.6		48.8		260000		27000		34200		3		8770	
CUA051	14	SW			17.9		67.3		45.8		279000		28300		36500		2.7		7730	
CUA051	15	SW			20.8		108		109		273000		52300		30000		11.3		16600	
CUA052	11	SW			22.1	J	139		60.4	J	164000		32400		19500		3.7		9540	
CUA052	12	SW			26.7	J	157		41.2	J	141000		23500		17500		3		6700	
CUA052	13	SW			17.9	J	108		49.4	J	171000		39600		21200		3.4		8650	
CUA052	14	SW			15.6		109		85.2		86900		11300		7040		3.9		8870	
CUA052	15	SW			21.3	J	66.2		46.5	J	121000		19700		14600		3.1		6970	
CUA053	11	SW			21.1	J	106		35.1	J	217000		29600		26700		2.5		6960	
CUA053	12	SW			2	J	117		44.7	J	176000		26100		21500		3.3		7730	
CUA053	13	SW			27.5	J	145		139	J	155000		24600		16700		8.2		13200	
CUA053	14	SW			22.2		116		41.9		87800		27200		11000		1.6		7500	
CUA053	15	SW			39.5	J	43.5		138		230000		46300		24200		17.3		22300	
CUA054	11	SW			20	J	69.9		23.9		92400	J	12300		10900	J	2.2		4750	J
CUA054	12	SW			15.6	J	36.2		16		79400	J	6300		9450	J	0.93		3190	J
CUA054	13	SW			28.2	J	159		68.4	J	256000	J	29300		30600	J	3.9		12700	J
CUA054	14	SW			18.8	J	71.4		41.7	J	167000	J	24500		20500	J	1.2		8400	J
CUA054	15	SW			19.1	J	66.3		34.1	J	107000	J	9330		11700	J	3.2		5140	J
CUA055	11	SW			21.7	J	116		95	J	234000	J	24300		25900	J	5		15700	J
CUA055	12	SW			28.4	J	181		83.5	J	169000	J	31000		19300	J	4.7		13400	J
CUA055	13	SW			19.8	J	61.6		58.5	J	235000		17500		27200		3.3		10300	
CUA055	14	SW			12.1		35.7		15.8		76300		7520		9140		1.3		3210	
CUA055	15	SW			21.3	J	67.3		34.9	J	153000		13900		17800		3.9		5680	
CUA056	1	SW			18.7	J	107	J	20.3	J	127000		10700		12900		9	J	3950	
CUA056	2	SW			10.9		34	J	17		48600		4680		4780		1.7	J	2170	
CUA056	3	SW			23.1	J	137	J	72.6	J	250000		25800		26900		6.9	J	10200	
CUA056	4	SW			11.3		26.9	J	16.6		35600		3240		3600		1.2	J	1960	
CUA056	5	SW			12.3		33	J	13.8		50700		4030		5400		1.2	J	2490	
CUA057	11	SW			13.1	J	36.5	J	20.6	J	211000		13700		25100		3.9	J	5300	
CUA057	12	SW			21.9	J	131	J	52.8	J	412000		38800		50500		5.5	J	11200	
CUA057	13	SW			22.1	J	133	J	47.2	J	451000		34300		55800		4.5	J	10500	

Table 1.3 - Lower Basin Surface Water

				AnalyteName	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA057	14	SW			27.1	J	141	J	40.3	J	334000		29800		40600		5.4	J	8740	
CUA057	15	SW			24.9	J	114	J	54	J	406000		27700		50300		4.9	J	11400	
CUA058	11	SW			14.3	J	59		41.9	J	113000	J	22600		14100	J	1.7		7200	J
CUA058	12	SW			10.3	J	32.8		17.3		88700	J	7640		10800	J	0.4		3400	J
CUA058	13	SW			12.8	J	53.3		44.9	J	133000	J	21500		16600	J	2.3		7460	J
CUA058	14	SW			18.9	J	117		408	J	439000	J	59400		45600	J	16.7		40300	J
CUA058	15	SW			27.7	J	125		84.9	J	262000	J	34600		31400	J	5.7		13400	J
CUA059	11	SW			14.9		34.2	J	25		36800		4760	J	4660		1.8		3260	
CUA059	12	SW			10.6		45.1	J	11.9		44800		2940	J	4330		2.2		1890	
CUA059	13	SW			20.8	J	167	J	214	J	389000		72500	J	46700		32.2		30700	
CUA059	14	SW			20.9	J	27	J	30.1	J	110000		18800	J	13000		4.1		5280	
CUA059	15	SW			18.7	J	107	J	58	J	122000		27400	J	14600		2.5		9290	
CUA060	11	SW			25.1	J	149	J	115	J	156000		40300	J	18100		5.4		12800	
CUA060	12	SW			19.5	J	131	J	81.2	J	167000		32400	J	18900		5		14400	
CUA060	13	SW			17.3		60.4	J	44		64000		9990	J	6310		3.6		5900	
CUA060	14	SW			1.7	J	25.6	J	36.7	J	135000		3790	J	3860		1.3		5960	
CUA060	15	SW			24.3	J	135		25.6	J	167000	J	20800		19600	J	10.9		4990	J
CUA063	11	SW			6.4	J	6.8		9.6		27600	J	2310		2600	J	1.2		1590	J
CUA063	12	SW			12	J	9.1		16.3		37200	J	5270		4850	J	2.6		2270	J
CUA063	13	SW			27.1	J	3.7		8.5		40300	J	11800		2450	J	7.8		2020	J
CUA063	14	SW			15	J	38		14.7		29500	J	3780		2590	J	2.8		2020	J
CUA063	15	SW			11.9	J	8.2		9.5		32000	J	3260		3220	J	2.8		1570	J
CUA065	1	SW			6.1	U	22.3		19.9		35600		3650		4190		0.91	J	2090	
CUA065	2	SW			8	U	24.9		24.7		48900		4900		6360		1.4	J	2650	
CUA065	3	SW			4.6	U	11.8		8.9		9140		1980		1110		0.53	J	1260	
CUA065	4	SW			5.1	U	22.5		26.3		14400		2300		1480		1	J	2660	
CUA065	5	SW			9.7	U	61.6		65.4		43600		8430		4580		3	J	6050	
CUA068	11	SW			4	U	5.2		8.2		3720		251		576		0.13		733	
CUA068	12	SW			7.7	U	18.4		34.2		17500		1080		2270		0.12		1850	
CUA068	13	SW			6.6	U	27.2		50.5		30600		1690		3170		0.29		2590	
CUA068	14	SW			8.8	U	28.4		41.4		23500		1640		1530		0.76		2790	
CUA068	15	SW			9.2	U	26.8		30.8		20200		1240		4720		0.21		2250	
CUA069	11	SW			9.9	U	23.5		55.4		20300		1370		9350		0.53		4780	
CUA069	12	SW			7	U	15		36.5		10300		717		6520		0.22		3170	
CUA069	13	SW			3.3	U	8.6		8.8		5140		344		1280		0.14		899	
CUA069	14	SW			7.6	U	18.2		29.6		1870		716		5130		0.05	U	2670	
CUA069	15	SW			3.2	U	3.5		5.6		1870		141		680		0.12		795	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 2.1 - Kingston Sediment

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA07	1	SD	0	1	64.3		266		63.3		174000		6190		14800		0.3		6260	
CUA07	2	SD	0	1	53.9		209		29.6		120000		6360		9510		0.35		4810	
CUA07	3	SD	0	1	46.6		134		23		91700		5160		7460		2.6		3480	
CUA07	4	SD	0	1	31.9		136		13.7		74900		3780		6120		2.3		2470	
CUA07	5	SD	0	1	44.9		166		18.6		99000		4530		7810		3.6		3250	
CUA07	6	SD	0	1	45.1		174		31.6		118000		4930		14300		3.6		5040	
CUA07	7	SD	0	1	49.5		209		23		120000		4530		9360		2.8		4130	
CUA07	8	SD	0	1	36.7		143		18.2		93500		4540		7540		3.1		3040	
CUA07	9	SD	0	1	36.1		140		18.4		89200		4630		7420		3.1		2960	
CUA07	10	SD	0	1	26.6		107		23.5		75500		3460		6830		2.7		3450	
CUA07	12	SD	0	0.5	23.2		113		13.6		77700		3050		6130		2.2		2240	
CUA07	13	SD	0	0.5	28.7		123		21.4		77000		3670		6290		2.8		3320	
CUA07	14	SD	0	0.5	24.8		104		22.7		76400		3850		6960		2.6		3420	
CUA07	15	SD	0	0.5	17.8		93.9		19		49200		3010		3240		2.5		2280	
PC	313	SD	0	0.25	3.97	U	15.5	J	0.982	J	13100		217		449	J	0.0605	J	341	J
PC	313	SD	0	0.33	1.02	U	14	J	0.378	J	8200		64.9		162	J	0.0474	J	166	J
PC	806	SD	0	0.5	29	J	11.1	J	1.02	J	10100	J	163	J	604	J	0.0533	U	187	J
PC	808	SD	0.5	1	31.8	J	34.4	J	0.959	J	24200	J	249	J	424	J	0.0562	U	452	J
PC	810	SD	0	1	19.6	J	60.6	J	0.417	J	14400	J	83.9	J	319	J	0.051	U	182	J
PC	811	SD	0.5	1	1.01	J	10.8	J	1.25	J	13600	J	228	J	338	J	0.05	U	393	J
PC	812	SD	0	2	0.93	J	2.95	J	0.55	J	12300	J	24.8	J	223	J	0.0478	U	211	J
PC	813	SD	0.5	1	0.963	J	2.95	J	0.581	J	16000	J	183	J	355	J	0.0499	U	237	J
PC	815	SD	0.5	1	0.92	J	2.79	J	0.82	J	9170	J	108	J	739	J	0.0494	U	235	J
PC	820	SD	0	4.5	1.01	J	5.98	J	0.912	J	15600	J	134	J	420	J	0.052	U	320	J
PC	856	SD	0	0.5	0.51	U	16.6	J	1.14		17900		218		438	J	0.0582	J	404	J
PC	857	SD	0	0.33	0.4745	U	13.4	J	0.809	J	17100		143		316	J	0.0478	J	262	J
PC	858	SD	0.17	0.5	0.49	U	8.34	J	0.738	J	17600		111		271	J	0.0507	J	310	J

Data Used to Calculate Human Health Exposure Point Concentrations
Table 2.2 - Kingston Surface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA07	16	SS	0	0.08	33.3		128		18		87200		4020		6690		2.5		2950	
CUA07	17	SS	0	0.08	16.5		73		13.9		39900		2660		3000		2.4		1740	
CUA07	18	SS	0	0.08	26.7		151		15.2		76600		3590		5830		2.5		2470	
CUA07	19	SS	0	0.08	21.8		133		14.6		68400		3610		5450		2.7		2290	
CUA07	20	SS	0	0.08	23.9		155		17.2		83800		3510		6440		2.4		2780	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 2.3 - Kingston Ground Water

				Analyte	Antimony		Arsenic		Cadmium		Calcium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
SF	432	GW	15	15	7.5		1	U	1.9		13900		269		2.3		66.4		0.2	U	563	
SF	432	GW	21	21	8.3		1	U	1	U	4060		75		1.1		5	U	0.2	U	160	
SF	432	GW	58	58	8.1		1	U	1	U	3990		50	U	1	U	5	U	0.2	U	147	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 2.4 - Kingston Surface Water

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	LocationID	Matrix	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA07	11	SW			7.3		3.6	J	11.6		3070		85.3	J	857		0.2	U	2070	
CUA07	12	SW			17.4		26.7		26		15900		2820		3020		1.5	J	3310	
CUA07	13	SW			9.9		7.2		13.4		8120		474		1280		0.2		2210	
CUA07	14	SW			9.2		10.5		12.7		2890		446		1100		0.2	U	2050	
CUA07	15	SW			36.7		134	J	83.8		81100		12500	J	9470		4.6		9900	
PC	311	SW			0.2	U	1	U	0.1	U	20		0.2	U	2.5	U	0.1	U	10	U
PC	311	SW			0.1	U	0.08	U	0.0345	U	9.4	U	0.15	J	2.3	J	0.05	U	13.2	U
PC	313	SW			1.2		1	U	0.2		23		0.4		2.5	U	0.1	U	62	
PC	314	SW			0.4		1	U	0.1	U	58		0.6		8		0.1	U	66	
PC	314	SW			0.67	U	0.08	U	0.0345	U	34.9	U	0.67		4.3	J	0.05	U	70.3	
PC	315	SW			3.6	J	0.39	J	0.29	J	5	U	0.62		3.7	J	0.05	UJ	106	
PC	315	SW			4.4	J	0.15	UJ	0.26	J	10.9	U	0.39	J	3	J	0.05	U	98.6	
PC	315	SW			1.1		1	U	0.5		28		2.3		2.5	U	0.1	U	66	
PC	315	SW			0.5	U	0.5	U	0.32		20	U	1.1		2.5	U	0.1	U	70.4	
PC	329	SW			1.1		0.5	U	1.2		23.5		11.8		18.1		0.1	U	306	
PC	329	SW			2.3	J	1		3.7		128		37.7		144		0.05	U	1470	
PC	339	SW			0.44	U	0.115	U	0.25	J	30.9	J	0.69	J	1.7	J	0.08	U	86.8	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 3.1 - Side Gulches Surface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA080	1	SS	0	0.08	31.2	J	68.9		8.1		67053		7870		3590		7.1		1960	
CUA080	2	SS	0	0.08	53.3	J	87.2		64.5		99928		14100		9280		8.6		6130	
CUA080	3	SS	0	0.08	41.9	J	98.5		11.7		84077		9970		5060		4.8		2210	
CUA080	4	SS	0	0.08	54.9	J	72.1		14.4		88771	J	15400		8470		11.9		3410	
CUA080	5	SS	0	0.08	49.8	J	73.1		10.3		88119	J	13400		7530		11.5		2930	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 3.2 - Side Gulches Sediment

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA081	1	SD	0	1	27		53.9		5.6		43200		4820		2610		5.7	J	1300	
CUA081	2	SD	0	1	0.49	U	41.8		0.1	U	19500		57.6		469		0.05	UJ	66.4	
CUA081	3	SD	0	1	28.7		98.5		3		41700		4440		3040		11.9	J	1450	
CUA081	4	SD	0	1	0.5	U	29.6		0.1	U	20500		45.3		640		0.05	UJ	112	
CUA081	5	SD	0	1	36.1		81.2		15.8		52200		7340		4410		12.5	J	3990	
CUA081	6	SD	0	0.5	27.9		74.7		112		23800		4000		654		2	J	10500	
CUA081	7	SD	0	0.5	2.7		43.7		1.7		21000		283		394		0.14	J	639	
CUA081	8	SD	0	0.5	5.4		64.6		1.6		26100		476		546		0.24	J	615	
CUA081	9	SD	0	0.5	0.93	J	21.8		1.1		25233	J	45.8		665		0.025	U	276	
CUA081	10	SD	0	0.5	47.1	J	113	J	45.6		21000		14800	J	1250		10.3		9340	J
SF	505	SD	0	0.5	623		22	J	9.11		39900		1900		3060		0.54		1470	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 3.3 - Side Gulches Surface Water

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
BC	260	SW			5.8		0.5	U	0.05	U	29.8		1.7		11.5		0.1	UJ	2.5	U
BC	260	SW			7.3		1		0.0345	U	53.3	J	0.165	U	63.6		0.13	J	6.15	U
CUA081	6	SW			3.6	U	12.2		60.3		5230		439		122		0.22		4730	
CUA081	7	SW			0.9	U	5.5		5.8		4570		34.9		152		0.05	U	738	
CUA081	8	SW			1.65	U	32		20.9		48800		413		1520		0.31		2310	
CUA081	9	SW			0.55	U	7.8		12.1		34500		96.1		1120		0.18		1630	
CUA081	10	SW			35		25.7		33.1		59800		7180		8570		3.5		5650	
MC	262	SW			0.14	U	0.42		0.56		2.5	U	0.47	J	1.8	J	0.05	U	135	
SF	248	SW			0.2		1	U	0.1	U	40		0.4		2.5	U	0.1	U	5	U
SF	248	SW			0.145	U	0.21	J	0.0345	U	62.6	J	3		8	J	0.14	UJ	11.4	J
SF	252	SW			0.26	U	0.3		0.0345	U	36.1	J	0.73		7.9	J	0.05	U	35	
SF	252	SW			0.016	U	0.38	J	0.021	U	61.2	J	0.34	J	4.1	J	0.08	U	19	J
SF	266	SW			0.1	U	1	U	0.1	U	50		2.8		2.5	U	0.1	U	5	U
SF	266	SW			0.125	U	0.08	U	0.0345	U	5.3	U	0.4	J	1.1	J	0.05	UJ	11.1	J
SF	267	SW			1		0.5	U	0.05	U	36.8		1.3		5.3		0.1	UJ	2.5	U
SF	267	SW			2	J	0.9	J	0.0345	U	2.5	U	0.34	J	2.5	J	0.05	UJ	14.8	J

Data Used to Calculate Human Health Exposure Point Concentrations
Table 4.1 - Silverton Surface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA096	1	SS	0	0.08	15.6	J	31.3	J	10.8		34700		1670		2150		0.82		3680	
CUA096	2	SS	0	0.08	10	J	18.5	J	12.9		23100		3090		1360		2.2		1680	
CUA096	3	SS	0	0.08	15.3	J	20.7	J	13.1		25800		4030		1780		2.4		1930	
CUA096	4	SS	0	0.08	24	J	38.1	J	12.9		34700		2203	J	1930		1.2		3345	J
CUA096	5	SS	0	0.08	20.7	J	34.1	J	17		33000		7222	J	2730		3.8		2411	J
CUA096	6	SS	0	0.08	17.7		25.6		16.1		24800		2490		1310		1.5	J	1920	
CUA096	7	SS	0	0.08	7.4		16.1		7.5		17700		1450		920		1		899	
CUA097	1	SS	0	0.08	9.9		16.5		7.1		17100		1690		858		1.1		1050	
CUA097	2	SS	0	0.08	21.4		32.7		9.6		23900		4710		1180		1.5		1900	
CUA097	3	SS	0	0.08	12.9	J	30.4		13.8		20900		2880		840		1		1220	
CUA097	4	SS	0	0.08	6.1	J	7		4.5		16700		1140		613		0.56		317	
CUA097	5	SS	0	0.08	8.6	J	16.3		6.6		20000		1650		959		0.71		797	
CUA097	6	SS	0	0.08	7.3	J	18.8		8.1		18400		1250		892		1.3	J	703	
CUA097	7	SS	0	0.08	8.4	J	20		8.6		19600		1730		945		1.6	J	991	
CUA098	1	SS	0	0.08	2.2		13.7		2.5		20900		685		329		0.46	J	358	
CUA098	2	SS	0	0.08	1.1		10.7		1.8		18700		597		398		0.23	J	211	
CUA098	3	SS	0	0.08	2.4		10.9		3.9		18900		543		467		0.28	J	380	
CUA098	4	SS	0	0.08	1.6		15.5		1.3		24500		179		768		0.05	UJ	256	
CUA098	5	SS	0	0.08	2.7		10.9		2.1		20300		350		462		0.3	J	286	
CUA098	6	SS	0	0.08	3.2		11.4		2.7		17600		624		615		0.68	J	369	J
CUA098	7	SS	0	0.08	1.9		7.7		2.4		13900		270		390		0.55	J	180	J
CUA099	1	SS	0	0.08	1.1	J	13.7		0.38		20400		65.4		753		0.075	UJ	138	
CUA099	2	SS	0	0.08	0.67	J	13.1		0.15	U	21400		65.5		711		0.085	UJ	114	
CUA099	3	SS	0	0.08	2	J	14.9		0.38		22400		60		789		0.075	UJ	118	
CUA099	4	SS	0	0.08			14		1.3		23400		167		917		0.15		385	
CUA099	5	SS	0	0.08			14		0.06		21700		51.7		534		0.18		107	
CUA099	6	SS	0	0.08	1.7		13.7		2.5		20200		260		717	J	0.29	J	359	J
CUA099	7	SS	0	0.08	3.7		18.9		2.9		26200		596		1020	J	0.29	J	481	J
CUA100	1	SS	0	0.08	5.9	J	21		7.4		21400		1790		1120		0.56	J	1120	
CUA100	2	SS	0	0.08			12.9		0.06		21500		64.6		837		0.025	UJ	110	
CUA100	3	SS	0	0.08	1.1	J	13.2		1.2		21500		290		953		0.06	J	224	
CUA100	4	SS	0	0.08	3	J	13		1.4		13762	J	239		462		0.11		221	
CUA100	5	SS	0	0.08	29.2		22.7		10.1	J	24800		6900	J	1050	J	7.7		1620	J
CUA100	6	SS	0	0.08	9.8		15.5		9.9	J	18400		2360	J	762	J	1.5		1070	J
CUA100	7	SS	0	0.08	0.5	U	11.2		1.8	J	17400		157	J	602	J	0.05	U	159	J
CUA100	8	SS	0	0.08	4.4		19.2		3.6		16800		790		739		0.52		523	
CUA100	9	SS	0	0.08	5.6		17.4		3.7		15800		1150		633		0.8		839	
CUA100	10	SS	0	0.08	12.9		16.2		7.2	J	22900		2860	J	860	J	2		887	J
CUA100	11	SS	0	0.08	7.7		13.4		7.3		17600		1340		614		0.55		968	
CUA100	12	SS	0	0.08	9.7		15.8		2.7		21900		2000		894		1.5		616	
CUA100	13	SS	0	0.08	1.9		14.9		1.5		19000		315		714		0.98		240	
CUA100	14	SS	0	0.08	1.3		15.8		1.1		21000		347		840		0.34		303	

Table 4.1 - Silverton Surface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA100	15	SS	0	0.08	9.3		33.5		5.6		23700		1920		1170		0.23		1130	
CUA100	16	SS	0	0.08	7.2		18		6.4		19200		1310		601		0.8		823	
CUA100	17	SS	0	0.08	0.495	U	12.8		0.4		19800		114		713		0.05	U	183	
CUA100	18	SS	0	0.08	155	J	41.3	J	2.4		19000		1500		479		0.53		505	J
CUA100	19	SS	0	0.08	6.3	J	21.6	J	6.2		21300		1670		1000		0.73		777	J
CUA101	1	SS	0	0.08	0.5	U	12.3		0.1	U	20800		93.4		665		0.05	U	173	
CUA101	2	SS	0	0.08	1.8		13.8		0.52		21400		210		802		0.05	U	381	J
CUA101	3	SS	0	0.08	0.75	J	11.2		0.49	J	19400		141		694		0.025	U	106	
CUA101	4	SS	0	0.08	4.5	J	15.8		2.9	J	19800		673		1020		0.1	U	688	
CUA101	5	SS	0	0.08	0.67	J	10.8	J	0.3		20000		77.6		717		0.025	U	120	
CUA101	6	SS	0	0.08	0.49	U	11.2		0.1	U	21400		76.8		660		0.05	U	166	
CUA101	7	SS	0	0.08	0.5	U	11.7		0.1	U	19700		117		690		0.05	U	226	
CUA102	1	SS	0	0.08	12.1		23		22.5		24600		2400		1230	J	2		3360	J
CUA102	2	SS	0	0.08	6.7		20		4.2		21200		845		923	J	0.37		1110	J
CUA102	3	SS	0	0.08	6		20.6		4		22000		967		830	J	0.49		917	J
CUA102	4	SS	0	0.08	8.6		21.2		5.1		21300		828		837		0.36		1050	
CUA102	5	SS	0	0.08	17.9		33.5		10.7		30300		2530		1390		1.3		2400	
CUA102	6	SS	0	0.08	19.7		30.9		9.2		28200		2180		1330		0.29		1780	
CUA102	7	SS	0	0.08	17.4		31.2		13		28600		2670		1220		1.2		2180	
CUA102	8	SS	0	0.08	0.335	UJ	11		0.39		21900		31.3		861		0.025	UJ	85	
CUA102	9	SS	0	0.08	0.335	UJ	12.5		0.44		22900		32.6		690		0.025	UJ	88.6	
CUA102	10	SS	0	0.08	0.335	UJ	11.2		0.74		21300		84.5		679		0.025	UJ	117	
CUA102	11	SS	0	0.08	0.335	UJ	10.8		0.52		22100		58.4		684		0.06	J	99.2	
CUA102	12	SS	0	0.08	0.335	UJ	10.7		0.34		21000		29.8		819		0.025	UJ	80.7	
CUA102	13	SS	0	0.08	2	J	20.2		1.6		22000		262		752		0.14	J	194	
CUA102	14	SS	0	0.08	0.335	UJ	12.5		0.285	U	19600		55.2		577		0.16	J	97.7	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 4.2 - Silverton Subsurface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA096	1	SB	0.08	0.5	18.1	J	27	J	13.7		28700		3450		1810		1.9		2430	
CUA096	1	SB	0.5	1	9	J	18.5	J	11.3		23500		2220		1340		1.2		1760	
CUA096	1	SB	1	1.5	9.1	J	17.9	J	10.7		23900		2110		1400		1.4		1960	
CUA096	2	SB	0.08	0.5	14.4	J	22.2	J	15.6		27400		5150		1800		3.8		1960	
CUA096	2	SB	0.5	1	56	J	41.4	J	25.9		46800		14000		3820		11.5		2650	
CUA096	2	SB	1	1.5	73.8	J	53	J	26.3		60000		19600		5150		14.4		2900	
CUA096	3	SB	0.08	0.5	37.5	J	45.4	J	18.6		28700		35123	J	1790		4.8		14655	J
CUA096	3	SB	0.5	1	12.5	J	20.7	J	17.3		25300		16439	J	1140		3.1		11602	J
CUA096	3	SB	1	1.5	27.6	J	27.3	J	14.5		31500		7767	J	1840		7.5		3941	J
CUA096	4	SB	0.08	0.5	13.5	J	26.8	J	14.6		28100		3817	J	1630		2.4		2660	J
CUA096	4	SB	0.5	1	34.4	J	36.4	J	23.4		38300		6188	J	2880		4.8		5677	J
CUA096	4	SB	1	1.5	41.8	J	62.3	J	44.8		51300		10228	J	4880		10		10623	J
CUA096	5	SB	0.08	0.5	93.2	J	61.2	J	18.5		76600		23900		9030		13.3		2910	
CUA096	5	SB	0.5	1	261	J	120	J	61.3		152000		56900		18200		31.1		9120	
CUA096	6	SB	0.08	0.5	33.3		18.6		6		25900		4600		1320		4.1		1200	
CUA096	6	SB	0.5	1	60.7		30		5.7		33100		7360		1710		15.3		1210	
CUA096	6	SB	1	1.5	48.1		21.5		6.6		29000		6330		1540		15.3		1260	
CUA096	7	SB	0.08	0.5	5.1		13.5		4.7		15600		971		806		0.81		599	
CUA096	7	SB	0.5	1	163		49.5		7.3		70300		22300		5340		26.8		1640	
CUA096	7	SB	1	1.5	11.2		11		9.4		21100		2710		1090		3.1		1100	
CUA096	7	SB	1.5	2	26.3		19.9		11.4		25600		4270		1430		9.2		1540	
CUA097	1	SB	0.08	0.5	7.8		13.4		6.7		15800		1390		785		1.1		930	
CUA097	1	SB	0.5	1	3.8		6.2		1.3		10800		414		341		0.62		375	
CUA097	1	SB	1	1.5	9.2		10.4		2.6		16000		1360		787		1.5		546	
CUA097	2	SB	0.08	0.5	6.8		16.5		9.4		18400		1490		760		0.86		1790	
CUA097	3	SB	0.08	0.5	7.2	J	26.5		6.1		17500		1340		491		0.59		727	
CUA097	3	SB	0.5	1	55.7	J	26.9		10.5		29700		10500		1890		3.6		1700	
CUA097	3	SB	1	1.5	77.7	J	47.3		22.4		38100		18000		2870		11.5	J	2720	
CUA097	4	SB	0.08	0.5	0.33	UJ	5.3		2.3		15900		142		551		0.055	U	164	
CUA097	4	SB	0.5	1	17.1	J	13.7		6.4		20600		3410		1100		1.2		710	
CUA097	4	SB	1	1.5	53.4	J	21.7		7.4		38200		8440		3060		4.1		968	
CUA097	5	SB	0.08	0.5	10.4	J	15.5		5.9		20100		1990		1020		0.73		691	
CUA097	5	SB	0.5	1	9.8	J	11.7		3.2		18900		1660		801		1.3	J	471	
CUA097	5	SB	1	1.5	58.2	J	22.5		2.7		43700		11100		3000		14.3	J	742	
CUA097	6	SB	0.08	0.5	6	J	12.5		4.4		19500		1060		936		1	J	682	
CUA097	6	SB	0.5	1	16.3	J	16.4		6.8		20400		2790		1190		3.3	J	861	
CUA097	6	SB	1	1.5	74.1	J	28.7		1.9		45400		13600		3790		7.4	J	868	
CUA097	7	SB	0.08	0.5	4.9	J	13.1		4.5		18300		1150		696		0.95	J	695	
CUA097	7	SB	0.5	1	8.3	J	10.1		4.8		20900		2820		1010		1.1		509	
CUA097	7	SB	1	1.5	16.5	J	12.3		3.7		21800		3860		1170		1.9		546	
CUA097	7	SB	1.5	2	7.2	J	8.9		4.2		19800		2610		901		0.71		483	
CUA098	1	SB	0.08	0.5	1.4		11		3.4		20500		218		510		0.17	J	261	

Table 4.2 - Silverton Subsurface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA098	1	SB	0.5	1	34.4		50.9		19.8		51100		11000		3230		4.5	J	3850	
CUA098	1	SB	1	1.5	26.5		52.6		37.1		52900		10800		1940		2.9	J	7550	
CUA098	2	SB	0.08	0.5	5.2		18.7		4.3		30900		883		789		0.98	J	453	
CUA098	2	SB	0.5	1	48.8		67		12		44400		7070		2810		4.6	J	2370	
CUA098	2	SB	1	1.5	108		58.8		26.1		77300		25500		5990		20.1	J	5370	
CUA098	3	SB	0.08	0.5	0.5	U	9.9		3		18900		248		465		0.28	J	1030	
CUA098	3	SB	0.5	1	17		32.3		13.3		30200		3940		1290		2.5	J	2260	
CUA098	3	SB	1	1.5	27.4		64.9		16.6		37400		6190		1670		3.4	J	3360	
CUA098	4	SB	0.08	0.5	2.3		16.2		1.6		26300		292		975		0.24	J	333	
CUA098	4	SB	0.5	1	5.8		19.3		4.9		24600		1090		811		0.53	J	800	
CUA098	4	SB	1	1.5	65.4		51.2		15.3		43700		14600		2740		4.2	J	3360	
CUA098	5	SB	0.08	0.5	1.5		10.8		3.6		20000		323		553		0.34	J	282	
CUA098	5	SB	0.5	1	18.7		39.7		11.8		31500		5610		1170		7.7	J	2130	J
CUA098	5	SB	1	1.5	22.6		53.5		9.8		24100		3240		1260		2.7	J	1620	
CUA098	6	SB	0.08	0.5	2.7		9.2		3.3		16400		448		454		0.51	J	323	J
CUA098	6	SB	0.5	1	3.1		9.5		2.8		15000		392		331		0.59	J	229	J
CUA098	6	SB	1	1.5	64.1		50.6		23.8		42400		13000		2960		17.5	J	4050	J
CUA098	7	SB	0.08	0.5	2.1		8.9		3.6		16400		216		311		0.44	J	201	J
CUA098	7	SB	0.5	1	60.9		103		20.9		37400		6640		2070		8.9	J	2440	J
CUA098	7	SB	1	1.5	50.7		168		34.8		49200		10400		3140		11		4260	J
CUA099	1	SB	0.08	0.5	1.4	J	14.5		0.87		24100		152		721		0.13	UJ	149	
CUA099	1	SB	0.5	1	1.4	J	13.4		0.15	U	20200		87.3		463		0.075	UJ	96.7	
CUA099	1	SB	1	1.5	2.5	J	20.9		0.96		23800		341		708		0.115	UJ	297	
CUA099	2	SB	0.08	0.5	1.6	J	13.8		1.1		23200		208		647		0.14	UJ	160	
CUA099	2	SB	0.5	1	1.3	J	16.5		0.03	U	20100		65.9		378		0.08	UJ	76.3	
CUA099	2	SB	1	1.5	1.3	J	19.3		0.03	U	20200		100		366		0.055	UJ	105	
CUA099	3	SB	0.08	0.5			14.4		1.1		23900		163		751		0.15		141	
CUA099	3	SB	0.5	1			17.2		0.62		22400		177		662		0.17		391	
CUA099	3	SB	1	1.5	20.2	J	23.9		6.1		50600		1810		2800		0.77		4280	
CUA099	4	SB	0.08	0.5	23.1	J	45.5		9.6		26400		4150		1430		1.5		1040	
CUA099	4	SB	0.5	1	9.1	J	23.4		7.5		20500		1580		871		0.79		1010	
CUA099	4	SB	1	1.5	9.1	J	24		7.1		20700		4260		1110		1.6		1090	
CUA099	4	SB	1.5	2	11	J	24.8		9.7		20600		3760		1060		2		1210	
CUA099	5	SB	0.08	0.5	2.4	J	17.2		2.7		21000		309		742		0.1	U	432	
CUA099	5	SB	0.5	1	1.1	J	10.6		3.1		21600		201		1150		0.09	U	149	
CUA099	5	SB	1	1.5	6.5	J	19		7.5		35900		600		1730		0.47		2340	
CUA099	6	SB	0.08	0.5	6.4		23.1		9.4		21600		1430		1020	J	1	J	1190	J
CUA099	6	SB	0.5	1	9		28.1		17.2		24200		2160		1270	J	2.1	J	2010	J
CUA099	6	SB	1	1.5	7.8		22.4		22.4		21000		1530		1020	J	3.1	J	2470	J
CUA099	7	SB	0.08	0.5	6.3		18.9		4.2		25400		1030		1010	J	0.42	J	720	J
CUA099	7	SB	0.5	1	3.3		19.3		3.5		18000		720		832	J	0.54	J	570	J
CUA099	7	SB	1	1.5	2.5		21.4		3.1		21900		625		975	J	0.37	J	551	J
CUA100	1	SB	0.08	0.5	4.4	J	23.6		5.9		21300		1180		1020		0.47	J	857	

Table 4.2 - Silverton Subsurface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA100	1	SB	0.5	1	5.6	J	28.8		7.1		25100		1990		1390		1.1	J	1470	
CUA100	1	SB	1	1.5	5.6	J	39.2		7.4		28400		1880		1970		0.98	J	1500	
CUA100	2	SB	0.08	0.5	2.1	J	21.4		0.96		22500		245		720		0.09	J	236	
CUA100	2	SB	0.5	1	6.6	J	25.4		7.3		23100		1930		925		0.89	J	1300	
CUA100	2	SB	1	1.5	7.6	J	33.2		9		25200		2450		1290		1.1	J	1630	
CUA100	3	SB	0.08	0.5	7.1	J	18.3		7.5		19600		1580		1050		0.49	J	876	
CUA100	3	SB	0.5	1	10.5	J	168		10.7		22197	J	2410		1220		1.1		1410	
CUA100	3	SB	1	1.5	9.9	J	55.9		8.7		23266	J	2150		1350		0.8		1440	
CUA100	4	SB	0.08	0.5	4.5	J	24.9		5.5	J	22538	J	1110		1230		0.74		805	J
CUA100	4	SB	0.5	1	4.5		30.3		5.7	J	24200		1010	J	1010	J	0.7		949	J
CUA100	4	SB	1	1.5	4.5		28.9		5.7	J	24300		663	J	980	J	0.57		897	J
CUA100	5	SB	0.08	0.5	59.8		29		18.5	J	32400		11000	J	1380	J	11		2380	J
CUA100	5	SB	0.5	1	66.4		30.4		18.7	J	34600		13300	J	1280	J	14.1		2360	J
CUA100	5	SB	1	1.5	21.9		17.1		63.9	J	23200		4930	J	672	J	3.1		3150	J
CUA100	5	SB	1.5	2	10.7		26.9		39.8	J	42100		3310	J	2980	J	2.5		4280	J
CUA100	6	SB	0.08	0.5	15.2		19.9		18.3	J	23800		3980	J	872	J	2.4		2020	J
CUA100	6	SB	0.5	1	39.8		24.6		26.7	J	28400		8570	J	1230	J	11		2840	J
CUA100	6	SB	1	1.5	46.6		23.1		28.9	J	25900		10500	J	1090	J	13.1		2720	J
CUA100	7	SB	0.08	0.5	8.1		22.1		5.2	J	20600		1610	J	867	J	0.87		698	J
CUA100	7	SB	0.5	1	5.2		22.2		3		21400		920		774		0.57		634	
CUA100	7	SB	1	1.5	3.1		22.2		1.8		20300		546		746		0.44		515	
CUA100	8	SB	0.08	0.5	6.2		21.3		4.8		18600		941		829		0.57		707	
CUA100	8	SB	0.5	1	5.4		45.4		4.8		21000		1560		1220		1.4		1070	
CUA100	8	SB	1	1.5	8.4		56.5		8.3		22200		2210		1310		1.7		1700	
CUA100	9	SB	0.08	0.5	9.7		28.8		7.6		24400		1970		1320		1.7		1330	
CUA100	9	SB	0.5	1	9.7		27.7		8.2		23600		2160		1290		1.9		1270	
CUA100	9	SB	1	1.5	9.3		30.3		9.3	J	25700		2090	J	1400	J	2.5		1370	J
CUA100	10	SB	0.08	0.5	41.9		22.4		14.4	J	28100		8850	J	948	J	8.8		1950	J
CUA100	10	SB	0.5	1	77.8		31.8		26.7	J	35000		16700	J	1090	J	16.4		3070	J
CUA100	10	SB	1	1.5	31.3		18.5		26.2	J	23400		6730	J	1000	J	12.6		2600	J
CUA100	11	SB	0.08	0.5	14.3		19.3		11		19300		2960		979		1.5		1640	
CUA100	11	SB	0.5	1	49.1		24		18.9		27300		10600		1300		14.2		2440	
CUA100	11	SB	1	1.5	39.9		23.2		28.2		27000		9110		939		10.9		2810	
CUA100	12	SB	0.08	0.5	45		23.7		7.2		27300		8190		1130		8.8		1430	
CUA100	12	SB	0.5	1	48.2		24.1		30.2		30000		10500		1090		2.8		2340	
CUA100	12	SB	1	1.5	7.5		15.8		42.4		23900		2270		1240		9.4		3160	
CUA100	13	SB	0.08	0.5	6.6		32.2		4.2		23600		1240		1200		1.5		1040	
CUA100	13	SB	0.5	1	6.5		28.5		5		22300		1400		1110		2		1080	
CUA100	13	SB	1	1.5	7.6		34.4		7		23500		1660		1240		1.6		1460	
CUA100	14	SB	0.08	0.5	13.3		24.4		16.3		37900		2900		2570		2		6320	
CUA100	14	SB	0.5	1	15.5		28.3		21.4		48600		4090		3670		3.2		9350	
CUA100	14	SB	1	1.5	17		29.3		19		43000		3720		2960		3.4		8800	
CUA100	15	SB	0.08	0.5	9.1		30.8		5.3		23000		1800		1100		1.2		1080	

Table 4.2 - Silverton Subsurface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA100	15	SB	0.5	1	10.4		34.8		5.6		24300		1910		1160		1.6		1130	
CUA100	15	SB	1	1.5	8.2		43.5		7.2		24100		1840		1320		4.7		1530	
CUA100	16	SB	0.08	0.5	18.2		30.5		7.8		24600		2980		1060		2.1		1260	
CUA100	16	SB	0.5	1	28.8		40		20.2		27400		5100		1230		3.9		2850	
CUA100	16	SB	1	1.5	12.4		34.1		12.1		22700		2110		1040		2.6		2320	
CUA100	17	SB	0.08	0.5	5.9		23.5		3.6		20600		859		817		0.56		614	
CUA100	17	SB	0.5	1	0.5	U	17.4		0.76		17600		130		559		0.84		328	
CUA100	17	SB	1	1.5	2.5	J	24	J	1.8		21900		407		915		0.19		348	J
CUA100	18	SB	0.08	0.5	6	J	27.7	J	5.5		22500		1080		1080		0.52		733	J
CUA100	18	SB	0.5	1	4	J	30.2	J	5.1		23000		919		956		0.57		794	J
CUA100	18	SB	1	1.5	8.1	J	26.5	J	4.6		22000		695		885		0.35		795	J
CUA100	19	SB	0.08	0.5	8.1	J	26.7	J	7.7		23700		2180		1260		1.1		1030	J
CUA100	19	SB	0.5	1	6.4	J	37.4	J	8.5		24300		1760		1420		0.95		1390	J
CUA100	19	SB	1	1.5	5.2	J	39	J	5.7		22700		1400		1200		0.7		1030	
CUA101	1	SB	0.08	0.5	2.7		15.1		3.3		23500		967		805		0.25		469	
CUA101	1	SB	0.5	1	5.2		11.8		12.4		19600		1310		672		0.58		2040	
CUA101	1	SB	1	1.5	2.5		10.8		9.1		20900		925		861		0.39		1950	J
CUA101	2	SB	0.08	0.5	4.9		18.5		5.1		22900		1500		1180		0.73		871	J
CUA101	2	SB	0.5	1	15.4		36		14.6		34500		4210		1930		2.1		2320	J
CUA101	2	SB	1	1.5	28.1	J	37.9		20.7	J	40800		6460		2640		2.7		2730	
CUA101	3	SB	0.08	0.5	5.1	J	14.6		5.4	J	21700		1060		846		0.5		586	
CUA101	3	SB	0.5	1	15.2	J	19.8		15.1	J	24100		2650		1140		1.1		2240	
CUA101	3	SB	1	1.5	13.7	J	17.5		14.8	J	21200		2830		1160		1.1		2310	
CUA101	3	SB	1.5	2	5.2	J	9.9		5.5	J	17600		925		1180		0.3		1150	
CUA101	4	SB	0.08	0.5	20	J	28.4	J	12.8		42300		4730		2640		2.2		2150	
CUA101	4	SB	0.5	1	33.1	J	31.5	J	30.7		45200		6840		2990		3.5		3660	
CUA101	4	SB	1	1.5	18.1	J	24.7	J	22		34400		4750		1990		2.3		3160	
CUA101	5	SB	0.08	0.5	2.8		15.2		2.1		25800		651		911		0.18		471	
CUA101	5	SB	0.5	1	18.6		30.4		17.7		37000		4090		1640		1.1		2630	
CUA101	5	SB	1	1.5	11.4		32.5		28		37400		5640		1640		1.5		3850	
CUA101	6	SB	0.08	0.5	31.3		45.8		30.8		51900		7530		2860		4.5		4050	
CUA101	6	SB	0.5	1	29.7		49.1		64.1		51100		8460		2730		4.7		7110	
CUA101	6	SB	1	1.5	25.7		40		55.3		37100		6690		1990		4.4		6240	J
CUA101	7	SB	0.08	0.5	0.495	U	10.4		0.1	U	20500		38.1		639		0.05	U	112	
CUA101	7	SB	0.5	1	3.95	U	21.9		6.7		26400		1740		1230		0.58		1000	
CUA101	7	SB	1	1.5	9.2		36.6		15.8		33400		3630		2040		1.8		2480	J
CUA102	1	SB	0.08	0.5	14.4		22.7		28.5		25100		3230		1390	J	2.9		4240	J
CUA102	1	SB	0.5	1	13.7		23.9		25.1		29100		3380		1530	J	2.6		4100	J
CUA102	1	SB	1	1.5	12.4		22.2		24.6		27100		2950		1410	J	2.7		3930	J
CUA102	2	SB	0.08	0.5	2.4		18.1		1.7		18700		429		706	J	0.52		585	J
CUA102	2	SB	0.5	1	0.5	U	13.6		1.2		19900		283		655	J	0.25		482	J
CUA102	2	SB	1	1.5	1.3		11.9		1.1		17400		264		555	J	0.23		436	J
CUA102	3	SB	0.08	0.5	1.45	U	16.7		3.3		21000		1060		832		0.59		840	

Table 4.2 - Silverton Subsurface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA102	3	SB	0.5	1	3.3		14.3		2.7		22700		756		795		0.56		805	
CUA102	3	SB	1	1.5	3.2		13.8		2.9		22800		796		716		0.45		796	
CUA102	3	SB	1.5	2	2.7		14.1		2.7		22600		772		709		0.42		780	
CUA102	4	SB	0.08	0.5	2.6		22.4		1.1		18900		191		608		0.19		538	
CUA102	4	SB	0.5	1	2		24		1.3		19900		199		604		0.25		584	
CUA102	4	SB	1	1.5	1.35	U	22.9		1.6		19700		238		592		0.25		606	
CUA102	5	SB	0.08	0.5	10.1		64.8		36.3		143000		1270		7230		1		13800	
CUA102	5	SB	0.5	1	22.1		87.7		22.9		71900		3780		3450		2.1		7160	
CUA102	5	SB	1	1.5	27.3		77.7		27		69400		5440		3160		2.8		7290	
CUA102	6	SB	0.08	0.5	11.7		41		13		50100		2370		2890		1.8		4750	
CUA102	6	SB	0.5	1	13.3		47.4		15.1		53900		2650		3210		2.1		5020	
CUA102	6	SB	1	1.5	8.2		33.8		9.1		38100		1640		1980		1.3		2870	
CUA102	7	SB	0.08	0.5	6.8		26.5		5.2		22400		885		667		1.1		1160	
CUA102	7	SB	0.5	1	1.9	J	22.3		5.3		19800		631		619		0.54	J	992	
CUA102	7	SB	1	1.5	1.8	J	22.3		4.8		20400		565		592		0.51	J	892	
CUA102	8	SB	0.08	0.5	2	J	12.9		2.6		18600		479		693		0.33	J	458	
CUA102	8	SB	0.5	1	1.2	J	12.8		1.7		17600		349		634		0.2	J	350	
CUA102	8	SB	1	1.5	1.3	J	10.8		0.98		18200		188		589		0.12	J	209	
CUA102	9	SB	0.08	0.5	0.335	UJ	11.7		1.2		20300		238		671		0.09	J	206	
CUA102	10	SB	0.08	0.5	0.76	J	11		1.4		18900		256		607		0.1	J	257	
CUA102	10	SB	0.5	1	0.85	J	14.3		0.83		18700		177		579		0.09	J	240	
CUA102	11	SB	0.08	0.5	0.335	UJ	9.9		0.55		21900		49.9		710		0.09	J	98.6	
CUA102	11	SB	0.5	1	14.1	J	30.9		8.2		21900		3480		843		0.85	J	1330	
CUA102	11	SB	1	1.5	5.4	J	25.4		4.1		19100		1500		697		0.45	J	767	
CUA102	12	SB	0.08	0.5	0.335	UJ	6.5		0.76		18800		56		791		0.07	J	102	
CUA102	12	SB	0.5	1	4.6	J	18.1		5.8		19300		1020		782		0.66	J	971	
CUA102	12	SB	1	1.5	1.3	J	11		2.1		18800		287		654		0.17	J	307	
CUA102	13	SB	0.08	0.5	2.8	J	22.8		2.1		22500		340		767		0.16	J	213	
CUA102	14	SB	0.08	0.5	4.1	J	35.6		2.2		23700		506		855		0.25	J	342	
CUA102	14	SB	0.5	1	4.3	J	33.5		2.4		24000		558		861		0.22	J	392	
CUA102	14	SB	1	1.5	3.2	J	29.3		2.5		22200		474		828		0.26	J	391	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 5.1 - Wallace Surface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA096	1	SS	0	0.08	15.6	J	31.3	J	10.8		34700		1670		2150		0.82		3680	
CUA096	2	SS	0	0.08	10	J	18.5	J	12.9		23100		3090		1360		2.2		1680	
CUA096	3	SS	0	0.08	15.3	J	20.7	J	13.1		25800		4030		1780		2.4		1930	
CUA096	4	SS	0	0.08	24	J	38.1	J	12.9		34700		2203	J	1930		1.2		3345	J
CUA096	5	SS	0	0.08	20.7	J	34.1	J	17		33000		7222	J	2730		3.8		2411	J
CUA096	6	SS	0	0.08	17.7		25.6		16.1		24800		2490		1310		1.5	J	1920	
CUA096	7	SS	0	0.08	7.4		16.1		7.5		17700		1450		920		1		899	
CUA097	1	SS	0	0.08	9.9		16.5		7.1		17100		1690		858		1.1		1050	
CUA097	2	SS	0	0.08	21.4		32.7		9.6		23900		4710		1180		1.5		1900	
CUA097	3	SS	0	0.08	12.9	J	30.4		13.8		20900		2880		840		1		1220	
CUA097	4	SS	0	0.08	6.1	J	7		4.5		16700		1140		613		0.56		317	
CUA097	5	SS	0	0.08	8.6	J	16.3		6.6		20000		1650		959		0.71		797	
CUA097	6	SS	0	0.08	7.3	J	18.8		8.1		18400		1250		892		1.3	J	703	
CUA097	7	SS	0	0.08	8.4	J	20		8.6		19600		1730		945		1.6	J	991	
CUA098	1	SS	0	0.08	2.2		13.7		2.5		20900		685		329		0.46	J	358	
CUA098	2	SS	0	0.08	1.1		10.7		1.8		18700		597		398		0.23	J	211	
CUA098	3	SS	0	0.08	2.4		10.9		3.9		18900		543		467		0.28	J	380	
CUA098	4	SS	0	0.08	1.6		15.5		1.3		24500		179		768		0.05	UJ	256	
CUA098	5	SS	0	0.08	2.7		10.9		2.1		20300		350		462		0.3	J	286	
CUA098	6	SS	0	0.08	3.2		11.4		2.7		17600		624		615		0.68	J	369	J
CUA098	7	SS	0	0.08	1.9		7.7		2.4		13900		270		390		0.55	J	180	J
CUA099	1	SS	0	0.08	1.1	J	13.7		0.38		20400		65.4		753		0.075	UJ	138	
CUA099	2	SS	0	0.08	0.67	J	13.1		0.15	U	21400		65.5		711		0.085	UJ	114	
CUA099	3	SS	0	0.08	2	J	14.9		0.38		22400		60		789		0.075	UJ	118	
CUA099	4	SS	0	0.08			14		1.3		23400		167		917		0.15		385	
CUA099	5	SS	0	0.08			14		0.06		21700		51.7		534		0.18		107	
CUA099	6	SS	0	0.08	1.7		13.7		2.5		20200		260		717	J	0.29	J	359	J
CUA099	7	SS	0	0.08	3.7		18.9		2.9		26200		596		1020	J	0.29	J	481	J
CUA100	1	SS	0	0.08	5.9	J	21		7.4		21400		1790		1120		0.56	J	1120	
CUA100	2	SS	0	0.08			12.9		0.06		21500		64.6		837		0.025	UJ	110	
CUA100	3	SS	0	0.08	1.1	J	13.2		1.2		21500		290		953		0.06	J	224	
CUA100	4	SS	0	0.08	3	J	13		1.4		13762	J	239		462		0.11		221	
CUA100	5	SS	0	0.08	29.2		22.7		10.1	J	24800		6900	J	1050	J	7.7		1620	J
CUA100	6	SS	0	0.08	9.8		15.5		9.9	J	18400		2360	J	762	J	1.5		1070	J
CUA100	7	SS	0	0.08	0.5	U	11.2		1.8	J	17400		157	J	602	J	0.05	U	159	J
CUA100	8	SS	0	0.08	4.4		19.2		3.6		16800		790		739		0.52		523	
CUA100	9	SS	0	0.08	5.6		17.4		3.7		15800		1150		633		0.8		839	
CUA100	10	SS	0	0.08	12.9		16.2		7.2	J	22900		2860	J	860	J	2		887	J
CUA100	11	SS	0	0.08	7.7		13.4		7.3		17600		1340		614		0.55		968	
CUA100	12	SS	0	0.08	9.7		15.8		2.7		21900		2000		894		1.5		616	
CUA100	13	SS	0	0.08	1.9		14.9		1.5		19000		315		714		0.98		240	
CUA100	14	SS	0	0.08	1.3		15.8		1.1		21000		347		840		0.34		303	

Table 5.1 - Wallace Surface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA100	15	SS	0	0.08	9.3		33.5		5.6		23700		1920		1170		0.23		1130	
CUA100	16	SS	0	0.08	7.2		18		6.4		19200		1310		601		0.8		823	
CUA100	17	SS	0	0.08	0.495	U	12.8		0.4		19800		114		713		0.05	U	183	
CUA100	18	SS	0	0.08	155	J	41.3	J	2.4		19000		1500		479		0.53		505	J
CUA100	19	SS	0	0.08	6.3	J	21.6	J	6.2		21300		1670		1000		0.73		777	J
CUA101	1	SS	0	0.08	0.5	U	12.3		0.1	U	20800		93.4		665		0.05	U	173	
CUA101	2	SS	0	0.08	1.8		13.8		0.52		21400		210		802		0.05	U	381	J
CUA101	3	SS	0	0.08	0.75	J	11.2		0.49	J	19400		141		694		0.025	U	106	
CUA101	4	SS	0	0.08	4.5	J	15.8		2.9	J	19800		673		1020		0.1	U	688	
CUA101	5	SS	0	0.08	0.67	J	10.8	J	0.3		20000		77.6		717		0.025	U	120	
CUA101	6	SS	0	0.08	0.49	U	11.2		0.1	U	21400		76.8		660		0.05	U	166	
CUA101	7	SS	0	0.08	0.5	U	11.7		0.1	U	19700		117		690		0.05	U	226	
CUA102	1	SS	0	0.08	12.1		23		22.5		24600		2400		1230	J	2		3360	J
CUA102	2	SS	0	0.08	6.7		20		4.2		21200		845		923	J	0.37		1110	J
CUA102	3	SS	0	0.08	6		20.6		4		22000		967		830	J	0.49		917	J
CUA102	4	SS	0	0.08	8.6		21.2		5.1		21300		828		837		0.36		1050	
CUA102	5	SS	0	0.08	17.9		33.5		10.7		30300		2530		1390		1.3		2400	
CUA102	6	SS	0	0.08	19.7		30.9		9.2		28200		2180		1330		0.29		1780	
CUA102	7	SS	0	0.08	17.4		31.2		13		28600		2670		1220		1.2		2180	
CUA102	8	SS	0	0.08	0.335	UJ	11		0.39		21900		31.3		861		0.025	UJ	85	
CUA102	9	SS	0	0.08	0.335	UJ	12.5		0.44		22900		32.6		690		0.025	UJ	88.6	
CUA102	10	SS	0	0.08	0.335	UJ	11.2		0.74		21300		84.5		679		0.025	UJ	117	
CUA102	11	SS	0	0.08	0.335	UJ	10.8		0.52		22100		58.4		684		0.06	J	99.2	
CUA102	12	SS	0	0.08	0.335	UJ	10.7		0.34		21000		29.8		819		0.025	UJ	80.7	
CUA102	13	SS	0	0.08	2	J	20.2		1.6		22000		262		752		0.14	J	194	
CUA102	14	SS	0	0.08	0.335	UJ	12.5		0.285	U	19600		55.2		577		0.16	J	97.7	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 5.2 - Wallace Subsurface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA096	1	SB	0.08	0.5	18.1	J	27	J	13.7		28700		3450		1810		1.9		2430	
CUA096	1	SB	0.5	1	9	J	18.5	J	11.3		23500		2220		1340		1.2		1760	
CUA096	1	SB	1	1.5	9.1	J	17.9	J	10.7		23900		2110		1400		1.4		1960	
CUA096	2	SB	0.08	0.5	14.4	J	22.2	J	15.6		27400		5150		1800		3.8		1960	
CUA096	2	SB	0.5	1	56	J	41.4	J	25.9		46800		14000		3820		11.5		2650	
CUA096	2	SB	1	1.5	73.8	J	53	J	26.3		60000		19600		5150		14.4		2900	
CUA096	3	SB	0.08	0.5	37.5	J	45.4	J	18.6		28700		35123	J	1790		4.8		14655	J
CUA096	3	SB	0.5	1	12.5	J	20.7	J	17.3		25300		16439	J	1140		3.1		11602	J
CUA096	3	SB	1	1.5	27.6	J	27.3	J	14.5		31500		7767	J	1840		7.5		3941	J
CUA096	4	SB	0.08	0.5	13.5	J	26.8	J	14.6		28100		3817	J	1630		2.4		2660	J
CUA096	4	SB	0.5	1	34.4	J	36.4	J	23.4		38300		6188	J	2880		4.8		5677	J
CUA096	4	SB	1	1.5	41.8	J	62.3	J	44.8		51300		10228	J	4880		10		10623	J
CUA096	5	SB	0.08	0.5	93.2	J	61.2	J	18.5		76600		23900		9030		13.3		2910	
CUA096	5	SB	0.5	1	261	J	120	J	61.3		152000		56900		18200		31.1		9120	
CUA096	6	SB	0.08	0.5	33.3		18.6		6		25900		4600		1320		4.1		1200	
CUA096	6	SB	0.5	1	60.7		30		5.7		33100		7360		1710		15.3		1210	
CUA096	6	SB	1	1.5	48.1		21.5		6.6		29000		6330		1540		15.3		1260	
CUA096	7	SB	0.08	0.5	5.1		13.5		4.7		15600		971		806		0.81		599	
CUA096	7	SB	0.5	1	163		49.5		7.3		70300		22300		5340		26.8		1640	
CUA096	7	SB	1	1.5	11.2		11		9.4		21100		2710		1090		3.1		1100	
CUA096	7	SB	1.5	2	26.3		19.9		11.4		25600		4270		1430		9.2		1540	
CUA097	1	SB	0.08	0.5	7.8		13.4		6.7		15800		1390		785		1.1		930	
CUA097	1	SB	0.5	1	3.8		6.2		1.3		10800		414		341		0.62		375	
CUA097	1	SB	1	1.5	9.2		10.4		2.6		16000		1360		787		1.5		546	
CUA097	2	SB	0.08	0.5	6.8		16.5		9.4		18400		1490		760		0.86		1790	
CUA097	3	SB	0.08	0.5	7.2	J	26.5		6.1		17500		1340		491		0.59		727	
CUA097	3	SB	0.5	1	55.7	J	26.9		10.5		29700		10500		1890		3.6		1700	
CUA097	3	SB	1	1.5	77.7	J	47.3		22.4		38100		18000		2870		11.5	J	2720	
CUA097	4	SB	0.08	0.5	0.33	UJ	5.3		2.3		15900		142		551		0.055	U	164	
CUA097	4	SB	0.5	1	17.1	J	13.7		6.4		20600		3410		1100		1.2		710	
CUA097	4	SB	1	1.5	53.4	J	21.7		7.4		38200		8440		3060		4.1		968	
CUA097	5	SB	0.08	0.5	10.4	J	15.5		5.9		20100		1990		1020		0.73		691	
CUA097	5	SB	0.5	1	9.8	J	11.7		3.2		18900		1660		801		1.3	J	471	
CUA097	5	SB	1	1.5	58.2	J	22.5		2.7		43700		11100		3000		14.3	J	742	
CUA097	6	SB	0.08	0.5	6	J	12.5		4.4		19500		1060		936		1	J	682	
CUA097	6	SB	0.5	1	16.3	J	16.4		6.8		20400		2790		1190		3.3	J	861	
CUA097	6	SB	1	1.5	74.1	J	28.7		1.9		45400		13600		3790		7.4	J	868	
CUA097	7	SB	0.08	0.5	4.9	J	13.1		4.5		18300		1150		696		0.95	J	695	
CUA097	7	SB	0.5	1	8.3	J	10.1		4.8		20900		2820		1010		1.1		509	
CUA097	7	SB	1	1.5	16.5	J	12.3		3.7		21800		3860		1170		1.9		546	
CUA097	7	SB	1.5	2	7.2	J	8.9		4.2		19800		2610		901		0.71		483	
CUA098	1	SB	0.08	0.5	1.4		11		3.4		20500		218		510		0.17	J	261	

Table 5.2 - Wallace Subsurface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA098	1	SB	0.5	1	34.4		50.9		19.8		51100		11000		3230		4.5	J	3850	
CUA098	1	SB	1	1.5	26.5		52.6		37.1		52900		10800		1940		2.9	J	7550	
CUA098	2	SB	0.08	0.5	5.2		18.7		4.3		30900		883		789		0.98	J	453	
CUA098	2	SB	0.5	1	48.8		67		12		44400		7070		2810		4.6	J	2370	
CUA098	2	SB	1	1.5	108		58.8		26.1		77300		25500		5990		20.1	J	5370	
CUA098	3	SB	0.08	0.5	0.5	U	9.9		3		18900		248		465		0.28	J	1030	
CUA098	3	SB	0.5	1	17		32.3		13.3		30200		3940		1290		2.5	J	2260	
CUA098	3	SB	1	1.5	27.4		64.9		16.6		37400		6190		1670		3.4	J	3360	
CUA098	4	SB	0.08	0.5	2.3		16.2		1.6		26300		292		975		0.24	J	333	
CUA098	4	SB	0.5	1	5.8		19.3		4.9		24600		1090		811		0.53	J	800	
CUA098	4	SB	1	1.5	65.4		51.2		15.3		43700		14600		2740		4.2	J	3360	
CUA098	5	SB	0.08	0.5	1.5		10.8		3.6		20000		323		553		0.34	J	282	
CUA098	5	SB	0.5	1	18.7		39.7		11.8		31500		5610		1170		7.7	J	2130	J
CUA098	5	SB	1	1.5	22.6		53.5		9.8		24100		3240		1260		2.7	J	1620	
CUA098	6	SB	0.08	0.5	2.7		9.2		3.3		16400		448		454		0.51	J	323	J
CUA098	6	SB	0.5	1	3.1		9.5		2.8		15000		392		331		0.59	J	229	J
CUA098	6	SB	1	1.5	64.1		50.6		23.8		42400		13000		2960		17.5	J	4050	J
CUA098	7	SB	0.08	0.5	2.1		8.9		3.6		16400		216		311		0.44	J	201	J
CUA098	7	SB	0.5	1	60.9		103		20.9		37400		6640		2070		8.9	J	2440	J
CUA098	7	SB	1	1.5	50.7		168		34.8		49200		10400		3140		11		4260	J
CUA099	1	SB	0.08	0.5	1.4	J	14.5		0.87		24100		152		721		0.13	UJ	149	
CUA099	1	SB	0.5	1	1.4	J	13.4		0.15	U	20200		87.3		463		0.075	UJ	96.7	
CUA099	1	SB	1	1.5	2.5	J	20.9		0.96		23800		341		708		0.115	UJ	297	
CUA099	2	SB	0.08	0.5	1.6	J	13.8		1.1		23200		208		647		0.14	UJ	160	
CUA099	2	SB	0.5	1	1.3	J	16.5		0.03	U	20100		65.9		378		0.08	UJ	76.3	
CUA099	2	SB	1	1.5	1.3	J	19.3		0.03	U	20200		100		366		0.055	UJ	105	
CUA099	3	SB	0.08	0.5			14.4		1.1		23900		163		751		0.15		141	
CUA099	3	SB	0.5	1			17.2		0.62		22400		177		662		0.17		391	
CUA099	3	SB	1	1.5	20.2	J	23.9		6.1		50600		1810		2800		0.77		4280	
CUA099	4	SB	0.08	0.5	23.1	J	45.5		9.6		26400		4150		1430		1.5		1040	
CUA099	4	SB	0.5	1	9.1	J	23.4		7.5		20500		1580		871		0.79		1010	
CUA099	4	SB	1	1.5	9.1	J	24		7.1		20700		4260		1110		1.6		1090	
CUA099	4	SB	1.5	2	11	J	24.8		9.7		20600		3760		1060		2		1210	
CUA099	5	SB	0.08	0.5	2.4	J	17.2		2.7		21000		309		742		0.1	U	432	
CUA099	5	SB	0.5	1	1.1	J	10.6		3.1		21600		201		1150		0.09	U	149	
CUA099	5	SB	1	1.5	6.5	J	19		7.5		35900		600		1730		0.47		2340	
CUA099	6	SB	0.08	0.5	6.4		23.1		9.4		21600		1430		1020	J	1	J	1190	J
CUA099	6	SB	0.5	1	9		28.1		17.2		24200		2160		1270	J	2.1	J	2010	J
CUA099	6	SB	1	1.5	7.8		22.4		22.4		21000		1530		1020	J	3.1	J	2470	J
CUA099	7	SB	0.08	0.5	6.3		18.9		4.2		25400		1030		1010	J	0.42	J	720	J
CUA099	7	SB	0.5	1	3.3		19.3		3.5		18000		720		832	J	0.54	J	570	J
CUA099	7	SB	1	1.5	2.5		21.4		3.1		21900		625		975	J	0.37	J	551	J
CUA100	1	SB	0.08	0.5	4.4	J	23.6		5.9		21300		1180		1020		0.47	J	857	

Table 5.2 - Wallace Subsurface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA100	1	SB	0.5	1	5.6	J	28.8		7.1		25100		1990		1390		1.1	J	1470	
CUA100	1	SB	1	1.5	5.6	J	39.2		7.4		28400		1880		1970		0.98	J	1500	
CUA100	2	SB	0.08	0.5	2.1	J	21.4		0.96		22500		245		720		0.09	J	236	
CUA100	2	SB	0.5	1	6.6	J	25.4		7.3		23100		1930		925		0.89	J	1300	
CUA100	2	SB	1	1.5	7.6	J	33.2		9		25200		2450		1290		1.1	J	1630	
CUA100	3	SB	0.08	0.5	7.1	J	18.3		7.5		19600		1580		1050		0.49	J	876	
CUA100	3	SB	0.5	1	10.5	J	168		10.7		22197	J	2410		1220		1.1		1410	
CUA100	3	SB	1	1.5	9.9	J	55.9		8.7		23266	J	2150		1350		0.8		1440	
CUA100	4	SB	0.08	0.5	4.5	J	24.9		5.5	J	22538	J	1110		1230		0.74		805	J
CUA100	4	SB	0.5	1	4.5		30.3		5.7	J	24200		1010	J	1010	J	0.7		949	J
CUA100	4	SB	1	1.5	4.5		28.9		5.7	J	24300		663	J	980	J	0.57		897	J
CUA100	5	SB	0.08	0.5	59.8		29		18.5	J	32400		11000	J	1380	J	11		2380	J
CUA100	5	SB	0.5	1	66.4		30.4		18.7	J	34600		13300	J	1280	J	14.1		2360	J
CUA100	5	SB	1	1.5	21.9		17.1		63.9	J	23200		4930	J	672	J	3.1		3150	J
CUA100	5	SB	1.5	2	10.7		26.9		39.8	J	42100		3310	J	2980	J	2.5		4280	J
CUA100	6	SB	0.08	0.5	15.2		19.9		18.3	J	23800		3980	J	872	J	2.4		2020	J
CUA100	6	SB	0.5	1	39.8		24.6		26.7	J	28400		8570	J	1230	J	11		2840	J
CUA100	6	SB	1	1.5	46.6		23.1		28.9	J	25900		10500	J	1090	J	13.1		2720	J
CUA100	7	SB	0.08	0.5	8.1		22.1		5.2	J	20600		1610	J	867	J	0.87		698	J
CUA100	7	SB	0.5	1	5.2		22.2		3		21400		920		774		0.57		634	
CUA100	7	SB	1	1.5	3.1		22.2		1.8		20300		546		746		0.44		515	
CUA100	8	SB	0.08	0.5	6.2		21.3		4.8		18600		941		829		0.57		707	
CUA100	8	SB	0.5	1	5.4		45.4		4.8		21000		1560		1220		1.4		1070	
CUA100	8	SB	1	1.5	8.4		56.5		8.3		22200		2210		1310		1.7		1700	
CUA100	9	SB	0.08	0.5	9.7		28.8		7.6		24400		1970		1320		1.7		1330	
CUA100	9	SB	0.5	1	9.7		27.7		8.2		23600		2160		1290		1.9		1270	
CUA100	9	SB	1	1.5	9.3		30.3		9.3	J	25700		2090	J	1400	J	2.5		1370	J
CUA100	10	SB	0.08	0.5	41.9		22.4		14.4	J	28100		8850	J	948	J	8.8		1950	J
CUA100	10	SB	0.5	1	77.8		31.8		26.7	J	35000		16700	J	1090	J	16.4		3070	J
CUA100	10	SB	1	1.5	31.3		18.5		26.2	J	23400		6730	J	1000	J	12.6		2600	J
CUA100	11	SB	0.08	0.5	14.3		19.3		11		19300		2960		979		1.5		1640	
CUA100	11	SB	0.5	1	49.1		24		18.9		27300		10600		1300		14.2		2440	
CUA100	11	SB	1	1.5	39.9		23.2		28.2		27000		9110		939		10.9		2810	
CUA100	12	SB	0.08	0.5	45		23.7		7.2		27300		8190		1130		8.8		1430	
CUA100	12	SB	0.5	1	48.2		24.1		30.2		30000		10500		1090		2.8		2340	
CUA100	12	SB	1	1.5	7.5		15.8		42.4		23900		2270		1240		9.4		3160	
CUA100	13	SB	0.08	0.5	6.6		32.2		4.2		23600		1240		1200		1.5		1040	
CUA100	13	SB	0.5	1	6.5		28.5		5		22300		1400		1110		2		1080	
CUA100	13	SB	1	1.5	7.6		34.4		7		23500		1660		1240		1.6		1460	
CUA100	14	SB	0.08	0.5	13.3		24.4		16.3		37900		2900		2570		2		6320	
CUA100	14	SB	0.5	1	15.5		28.3		21.4		48600		4090		3670		3.2		9350	
CUA100	14	SB	1	1.5	17		29.3		19		43000		3720		2960		3.4		8800	
CUA100	15	SB	0.08	0.5	9.1		30.8		5.3		23000		1800		1100		1.2		1080	

Table 5.2 - Wallace Subsurface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA100	15	SB	0.5	1	10.4		34.8		5.6		24300		1910		1160		1.6		1130	
CUA100	15	SB	1	1.5	8.2		43.5		7.2		24100		1840		1320		4.7		1530	
CUA100	16	SB	0.08	0.5	18.2		30.5		7.8		24600		2980		1060		2.1		1260	
CUA100	16	SB	0.5	1	28.8		40		20.2		27400		5100		1230		3.9		2850	
CUA100	16	SB	1	1.5	12.4		34.1		12.1		22700		2110		1040		2.6		2320	
CUA100	17	SB	0.08	0.5	5.9		23.5		3.6		20600		859		817		0.56		614	
CUA100	17	SB	0.5	1	0.5	U	17.4		0.76		17600		130		559		0.84		328	
CUA100	17	SB	1	1.5	2.5	J	24	J	1.8		21900		407		915		0.19		348	J
CUA100	18	SB	0.08	0.5	6	J	27.7	J	5.5		22500		1080		1080		0.52		733	J
CUA100	18	SB	0.5	1	4	J	30.2	J	5.1		23000		919		956		0.57		794	J
CUA100	18	SB	1	1.5	8.1	J	26.5	J	4.6		22000		695		885		0.35		795	J
CUA100	19	SB	0.08	0.5	8.1	J	26.7	J	7.7		23700		2180		1260		1.1		1030	J
CUA100	19	SB	0.5	1	6.4	J	37.4	J	8.5		24300		1760		1420		0.95		1390	J
CUA100	19	SB	1	1.5	5.2	J	39	J	5.7		22700		1400		1200		0.7		1030	
CUA101	1	SB	0.08	0.5	2.7		15.1		3.3		23500		967		805		0.25		469	
CUA101	1	SB	0.5	1	5.2		11.8		12.4		19600		1310		672		0.58		2040	
CUA101	1	SB	1	1.5	2.5		10.8		9.1		20900		925		861		0.39		1950	J
CUA101	2	SB	0.08	0.5	4.9		18.5		5.1		22900		1500		1180		0.73		871	J
CUA101	2	SB	0.5	1	15.4		36		14.6		34500		4210		1930		2.1		2320	J
CUA101	2	SB	1	1.5	28.1	J	37.9		20.7	J	40800		6460		2640		2.7		2730	
CUA101	3	SB	0.08	0.5	5.1	J	14.6		5.4	J	21700		1060		846		0.5		586	
CUA101	3	SB	0.5	1	15.2	J	19.8		15.1	J	24100		2650		1140		1.1		2240	
CUA101	3	SB	1	1.5	13.7	J	17.5		14.8	J	21200		2830		1160		1.1		2310	
CUA101	3	SB	1.5	2	5.2	J	9.9		5.5	J	17600		925		1180		0.3		1150	
CUA101	4	SB	0.08	0.5	20	J	28.4	J	12.8		42300		4730		2640		2.2		2150	
CUA101	4	SB	0.5	1	33.1	J	31.5	J	30.7		45200		6840		2990		3.5		3660	
CUA101	4	SB	1	1.5	18.1	J	24.7	J	22		34400		4750		1990		2.3		3160	
CUA101	5	SB	0.08	0.5	2.8		15.2		2.1		25800		651		911		0.18		471	
CUA101	5	SB	0.5	1	18.6		30.4		17.7		37000		4090		1640		1.1		2630	
CUA101	5	SB	1	1.5	11.4		32.5		28		37400		5640		1640		1.5		3850	
CUA101	6	SB	0.08	0.5	31.3		45.8		30.8		51900		7530		2860		4.5		4050	
CUA101	6	SB	0.5	1	29.7		49.1		64.1		51100		8460		2730		4.7		7110	
CUA101	6	SB	1	1.5	25.7		40		55.3		37100		6690		1990		4.4		6240	J
CUA101	7	SB	0.08	0.5	0.495	U	10.4		0.1	U	20500		38.1		639		0.05	U	112	
CUA101	7	SB	0.5	1	3.95	U	21.9		6.7		26400		1740		1230		0.58		1000	
CUA101	7	SB	1	1.5	9.2		36.6		15.8		33400		3630		2040		1.8		2480	J
CUA102	1	SB	0.08	0.5	14.4		22.7		28.5		25100		3230		1390	J	2.9		4240	J
CUA102	1	SB	0.5	1	13.7		23.9		25.1		29100		3380		1530	J	2.6		4100	J
CUA102	1	SB	1	1.5	12.4		22.2		24.6		27100		2950		1410	J	2.7		3930	J
CUA102	2	SB	0.08	0.5	2.4		18.1		1.7		18700		429		706	J	0.52		585	J
CUA102	2	SB	0.5	1	0.5	U	13.6		1.2		19900		283		655	J	0.25		482	J
CUA102	2	SB	1	1.5	1.3		11.9		1.1		17400		264		555	J	0.23		436	J
CUA102	3	SB	0.08	0.5	1.45	U	16.7		3.3		21000		1060		832		0.59		840	

Table 5.2 - Wallace Subsurface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA102	3	SB	0.5	1	3.3		14.3		2.7		22700		756		795		0.56		805	
CUA102	3	SB	1	1.5	3.2		13.8		2.9		22800		796		716		0.45		796	
CUA102	3	SB	1.5	2	2.7		14.1		2.7		22600		772		709		0.42		780	
CUA102	4	SB	0.08	0.5	2.6		22.4		1.1		18900		191		608		0.19		538	
CUA102	4	SB	0.5	1	2		24		1.3		19900		199		604		0.25		584	
CUA102	4	SB	1	1.5	1.35	U	22.9		1.6		19700		238		592		0.25		606	
CUA102	5	SB	0.08	0.5	10.1		64.8		36.3		143000		1270		7230		1		13800	
CUA102	5	SB	0.5	1	22.1		87.7		22.9		71900		3780		3450		2.1		7160	
CUA102	5	SB	1	1.5	27.3		77.7		27		69400		5440		3160		2.8		7290	
CUA102	6	SB	0.08	0.5	11.7		41		13		50100		2370		2890		1.8		4750	
CUA102	6	SB	0.5	1	13.3		47.4		15.1		53900		2650		3210		2.1		5020	
CUA102	6	SB	1	1.5	8.2		33.8		9.1		38100		1640		1980		1.3		2870	
CUA102	7	SB	0.08	0.5	6.8		26.5		5.2		22400		885		667		1.1		1160	
CUA102	7	SB	0.5	1	1.9	J	22.3		5.3		19800		631		619		0.54	J	992	
CUA102	7	SB	1	1.5	1.8	J	22.3		4.8		20400		565		592		0.51	J	892	
CUA102	8	SB	0.08	0.5	2	J	12.9		2.6		18600		479		693		0.33	J	458	
CUA102	8	SB	0.5	1	1.2	J	12.8		1.7		17600		349		634		0.2	J	350	
CUA102	8	SB	1	1.5	1.3	J	10.8		0.98		18200		188		589		0.12	J	209	
CUA102	9	SB	0.08	0.5	0.335	UJ	11.7		1.2		20300		238		671		0.09	J	206	
CUA102	10	SB	0.08	0.5	0.76	J	11		1.4		18900		256		607		0.1	J	257	
CUA102	10	SB	0.5	1	0.85	J	14.3		0.83		18700		177		579		0.09	J	240	
CUA102	11	SB	0.08	0.5	0.335	UJ	9.9		0.55		21900		49.9		710		0.09	J	98.6	
CUA102	11	SB	0.5	1	14.1	J	30.9		8.2		21900		3480		843		0.85	J	1330	
CUA102	11	SB	1	1.5	5.4	J	25.4		4.1		19100		1500		697		0.45	J	767	
CUA102	12	SB	0.08	0.5	0.335	UJ	6.5		0.76		18800		56		791		0.07	J	102	
CUA102	12	SB	0.5	1	4.6	J	18.1		5.8		19300		1020		782		0.66	J	971	
CUA102	12	SB	1	1.5	1.3	J	11		2.1		18800		287		654		0.17	J	307	
CUA102	13	SB	0.08	0.5	2.8	J	22.8		2.1		22500		340		767		0.16	J	213	
CUA102	14	SB	0.08	0.5	4.1	J	35.6		2.2		23700		506		855		0.25	J	342	
CUA102	14	SB	0.5	1	4.3	J	33.5		2.4		24000		558		861		0.22	J	392	
CUA102	14	SB	1	1.5	3.2	J	29.3		2.5		22200		474		828		0.26	J	391	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 6.1 - Osburn, Wallace, and Silverton Sediment

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Zone ID	**Matrix**	**Location ID**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
SEGMG01	SD	504	0	0.5	25.3		36.4	J	5.67	J	33600		738		3120		0.125		1160	
SEGMG01	SD	506	0.5	0.67	21.5	J	25.4	J	8.76	J	36700		3040		5230		1.5		2370	
SEGMG01	SD	506	0	0.5	14	J	124	J	13.1		54700		2200		2590		1.99		1440	
SEGMG01	SD	508	0	0.5	15.2	J	4.42	J	29.3		40000		2410		3530		1.61		4430	
SEGMG01	SD	509	0	0.5	31.7		52	J	12.6		41500		5080		3740		3.96		2270	
SEGMG01	SD	512	0.25	0.8	3.69	**UJ**	3.81	J	7.09		25600		1890		2920		0.14		1000	
SEGMG01	SD	513	0	0.33	20	J	26.7	J	17		39300		3390		2560		1.96		3150	
SEGMG01	SD	513	0.33	1	24.2	J	50.1	J	19.8		54900		4500		4790		2.45		3270	
SEGMG01	SD	515	0	0.33	4.26	**UJ**	33.6	J	6.07	J	39900		2710		3170		1.11		922	
SEGMG01	SD	516	0	0.04	30.6		51.7	J	20.4		45400		5720		3210		2.92		4170	
SEGMG01	SD	517	0	0.17	34		9.48	J	5.5		27400		1010		2020	J	0.159	J	1130	J
SEGMG01	SD	518	0	0.17	1.695	**UJ**	6.52	J	15.1		54000		1320		5760	J	0.096	J	2820	J
SEGMG01	SD	519	0	0.5	22.1	J	34.8	J	20.3		48200		6160		4100	J	2.38	J	3840	J
SEGMG01	SD	520	0	0.5	0.983	J	7.01	J	64.4		57200		4450		6620	J	0.111	J	11500	J
SEGMG01	SD	541	0	0.5	3.235	**UJ**	18.2	J	12.3	J	32600		3910	J	2900		2.12		1870	J
SEGMG01	SD	543	0	1.5	1.055	**UJ**	9.24	J	5.27	J	27900		1490	J	2210		0.234		1480	J
SEGMG01	SD	544	0	0.5	174		184	J	5.47	J	27600		1600	J	1820		0.43		1410	J
SEGMG01	SD	544	0	0.5	1.755	**UJ**	10.3	J	5.38	J	22700		1370	J	1780		0.361		1420	J
SEGMG01	SD	547	0.5	1	2.24	**UJ**	20.8	J	5.96	J	29000		929	J	2040		0.816		1080	J
SEGMG01	SD	549	0	2	27.5		22.6	J	8.77	J	35500		5340	J	3480		2.36		1450	J
SEGMG01	SD	550	0	0.5	3.55	**UJ**	25.2	J	9.13	J	26400		1610	J	1910		0.896		1390	J

Data Used to Calculate Human Health Exposure Point Concentrations
Table 6.2 - Osburn, Wallace and Siverton Surface Water

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Zone ID	Matrix	Location ID	Beginning Depth	Ending Depth	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
SEGMG01	SW	250			0.4		1	U	2.2		35		1.4		6		0.1	U	271	
SEGMG01	SW	245			0.415	U	0.23	J	0.0345	U	6.5	U	0.88		3	J	0.065	UJ	21.4	
SEGMG01	SW	245			0.7		1	U	0.1	U	44		1.1		2.5	U	0.1	U	12	
SEGMG01	SW	236			1.8	J	7.1		0.0345	U	2.5	UJ	0.18	J	0.6	U	0.05	U	10.55	U
SEGMG01	SW	236			1.1		7.3		0.05	U	10	U	0.25	U	2.5	U	0.1	UJ	2.5	U
SEGMG01	SW	239			2.8	J	0.23	J	7.2		63.1	J	43.3		47.6		0.84	J	1200	
SEGMG01	SW	239			0.315	U	0.23		2.6		81.4		18.2		22.2		0.08	U	415	
SEGMG01	SW	241			2.9	J	0.61	J	7.1		64	J	40.8		49.2		0.14	UJ	1130	
SEGMG01	SW	272			1.1		0.5	U	2.6		65.4		20.6		21.7		0.1	UJ	350	
SEGMG01	SW	249			1.4	U	0.47		7.5		43.4	J	30.8		47.1		0.05	U	1150	
SEGMG01	SW	249			1.2		0.5	U	2.9		72.5		21.4		22.4		0.1	UJ	379	
SEGMG01	SW	253			1.35	U	0.39		7.9		58	J	26.8		48.1		0.05	U	1330	
SEGMG01	SW	253			1.1		0.5	U	3.1		90.6		21.9		25.2		0.1	UJ	436	
SEGMG01	SW	253			1.2		0.5	U	3.6		207		15.9		26.5		0.1	U	517	
SEGMG01	SW	254			1.75	U	0.29		8.4		33.1	J	27.1		46.6		0.05	U	1380	
SEGMG01	SW	254			1.2		0.5	U	3.1		84.6		24.7		25.2		0.1	UJ	416	
SEGMG01	SW	259			1.25	U	0.25		8.7		528		22.3		48.3		0.05	U	1490	
SEGMG01	SW	259			1.1		0.5	U	3.2		60.8		17		23		0.1	U	446	
SEGMG01	SW	263			7.5		0.72		6.7		39.7	J	22		51.2		0.05	U	1080	
SEGMG01	SW	264			7		0.66	J	6.6		36.1	J	19.9		48.3		0.05	UJ	1090	
SEGMG01	SW	264			2.15	U	0.51	J	3	J	108	J	17.4		25.3	J	0.08	U	473	
SEGMG01	SW	232			3.1	J	0.175	U	8.4		37	U	36.8		58		0.05	U	1400	
SEGMG01	SW	233			2.7	J	0.215	U	8.3		38.4	U	30.6		55.1		0.05	U	1420	
SEGMG01	SW	233			0.6		0.3		2.7		79.6		21.7		24.3		0.08	U	447	
SEGMG01	SW	234			0.31	U	0.215	UJ	0.0345	U	7.85	U	0.16	J	0.5	U	0.05	U	4.95	U
SEGMG01	SW	234			0.25	U	0.5	U	0.05	U	78.4		0.25	U	2.5	U	0.1	UJ	2.5	U
SEGMG01	SW	234			0.6	U	0.115	U	3.3	J	104	J	24.5		27	J	0.08	U	532	
SEGMG01	SW	235			3.2	J	0.51	J	6.9		28.3	UJ	49.1		46.2		0.05	U	1020	
SEGMG01	SW	235			0.51		0.115	U	2.4		107		18.2		23.7		0.08	U	377	
SEGMG01	SW	237			2.8	J	0.36	J	7.2		15.1	UJ	32.1		47.4		0.05	U	1130	
SEGMG01	SW	238			7.5		3.9		0.0345	U	38.4	UJ	1.5		174		0.05	U	10.1	U
SEGMG01	SW	238			1	J	1.2	J	0.021	U	146		1.1	J	34.3		0.08	U	6	J
SEGMG01	SW	240			1.8	J	0.63	J	0.0345	U	2.5	UJ	0.54		0.55	U	0.05	U	8.4	U
SEGMG01	SW	240			1.2		0.5	U	0.05	U	10	U	0.61		2.5	U	0.1	UJ	6.8	
SEGMG01	SW	242			7		0.8	J	0.1	J	8.5	UJ	0.94		2.25	U	0.05	U	13.7	U
SEGMG01	SW	242			3.6		1	U	0.1	U	10	U	0.6		2.5	U	0.25	U	17	
SEGMG01	SW	243			3	J	0.48	J	7.6		15.75	UJ	31.6		49.3		0.05	U	1160	
SEGMG01	SW	244			6.3		0.6	J	0.0345	U	2.5	UJ	0.2	J	0.85	U	0.05	U	4.85	U
SEGMG01	SW	244			1.4		1	U	0.1	U	35		0.3		7		0.25	U	5	U
SEGMG01	SW	246			0.7		1	U	0.1	U	28		1.6		2.5	U	0.25	U	5	U
SEGMG01	SW	247			2.8	J	0.42	J	7.8		45.4	J	31.2		49.5		0.05	UJ	1320	
SEGMG01	SW	251			0.23	U	0.49		0.0345	U	44.8	J	0.6		3.3	J	0.05	U	4.4	U

Table 6.2 - Osburn, Wallace and Siverton Surface Water

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Zone ID	**Matrix**	**Location ID**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
SEGMG01	SW	251			0.3		1	U	0.1	U	269		0.7		16		0.1	U	5	U
SEGMG01	SW	255			0.55	U	0.08	U	0.0345	U	5.8	J	0.135	U	1.3	J	0.05	U	7.8	U
SEGMG01	SW	255			0.46	J	0.115	U	0.021	U	96.7	J	0.35	J	2	J	0.08	U	13.1	J
SEGMG01	SW	256			0.485	U	0.31	J	0.0345	U	2.5	U	0.2	J	0.5	U	0.05	UJ	29.9	
SEGMG01	SW	256			0.33	J	0.115	U	0.021	U	67.1	J	0.47	J	0.83	J	0.08	U	1.8	J
SEGMG01	SW	257			0.6	U	0.41	J	0.0345	U	44.4	J	0.84		12		0.05	UJ	15	J
SEGMG01	SW	257			2.1		1	U	0.1	U	29		0.3		2.5	U	0.1	U	5	U
SEGMG01	SW	258			1.3	U	0.2		8.3		30.1	J	25.4		46.2		0.05	U	1460	
SEGMG01	SW	261			0.55	U	0.08	U	11		8.9	J	9.5		34.1		0.05	U	1640	
SEGMG01	SW	261			0.8		1	U	12.5		68		17.3		65		0.1	U	1670	
SEGMG01	SW	265			0.6	U	0.33	J	0.18	J	71	J	0.83		9.1	J	0.05	UJ	32.2	
SEGMG01	SW	265			1.4		0.5	U	0.22		175		1.8		11.7		0.1	UJ	23.3	
SEGMG01	SW	273			0.6		0.115	U	1.9		82.4		20		23.1		0.08	U	295	
SEGMG01	SW	274			0.51		0.115	U	2.4		143		19.3		24.9		0.08	U	386	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 7.1 - Ninemile Surface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				Units	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
SiteID	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CC	404	SS	0	0.5			44.6		17.3		23400		2850		2580		0.34	J	1810	
CC	405	SS	0	0.5	79.3	J	103		53		87900		4970		3360		4.3	J	7180	
CC	406	SS	0	0.5	12.7	J	141		15.7		25200		3590		1020		0.6	J	2680	
CC	407	SS	0	0.5	25.3	J	3610		1.9		46300		1510		1800		0.79	J	591	
CC	408	SS	0	0.5	242	J	84.7		21.8		77400		4750		2930		6	J	4340	
CC	426	SS	0	0.08	1.7	J	5.8		1.8		18800		306		882		0.19	J	251	
CC	427	SS	0	0.08	2.2	J	6.8		0.175	UJ	19300		104		503		0.12	J	145	
CC	428	SS	0	0.08	1.7	J	6.9		3.4		16000		311		799		0.11	J	245	
CC	429	SS	0	0.08	51.6	J	97		146		46000		63700		3020		2.2		25800	
CC	430	SS	0	0.08	10.6	J	25.8		9.4		20100		4760		1270		0.81		2870	
NM	413	SS	0	0.08	3.2	J	14.1		6.4		32700		4200		1270		0.2	J	2440	
NM	414	SS	0	0.08	2.3	J	12.8		10.9		31800		2350		1250		0.11	J	2560	
NM	415	SS	0	0.08	1.8	J	75.3		19.3		36000		2050		810		0.15	J	5840	
NM	416	SS	0	0.08	2.7	J	20.1		13.5		34900		4500		1360		0.82		5050	
NM	417	SS	0	0.08	3.7	J	49.8		3.3		35300		3870		910		0.09		1110	
NM	429	SS	0	0.5	0.21	UJ	1.6	J	0.12	U	18500		83.4		2570		9		109	
NM	430	SS	0	0.5	2.8	J	10		16.4		27900		3230		798		2.2		3160	
NM	431	SS	0	0.5	9.3	J	23.8		19.9		49500		4540		326		9.5		3980	
NM	432	SS	0	0.5	6.6	J	7.5		3.1		37700		6210		351		6.1		1090	
NM	433	SS	0	0.5	4.2	J	5.4		40.1		28400		4070		1560		2.5		9300	
NM	461	SS	0	0.5	10.5	J	43.6		14.5		64400		16100		1340		1.3		4110	
NM	462	SS	0	0.5	3.1	J	14.5		8.5		29300		4610		1080		0.62		2970	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 7.2 - Ninemile Sediment

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				Units	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
SiteID	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CC	695	SD	0	2	288	J	8.4		132		28100		67100		1820		1.1		22400	
CC	699	SD	0	2	4.25	UJ	15.9		5.3		20200		3870		1130		0.27		972	
CC	702	SD	0	2	2.45	UJ	4.5		8.2		8090		858		330		0.68		1480	
CC	705	SD	0	2	18.6	J	14.3		6.3		13000		4030		1130		0.07	J	844	
CC	707	SD	0.5	1	1.1	J	3.6		0.52	J	11600		88		428		0.13		220	
CC	708	SD	0	0.5	1.7	J	4.5		0.65	J	11700		160		665		0.09	U	162	
NM	751	SD	0	0.5	37.1	J	30.3	J	40.9	J	37100	J	14100	J	6830		0.0545	U	6300	J
NM	753	SD	0	1.5	2.33	UJ	3.11	J	27.3	J	14200	J	1730	J	759		0.0485	U	5130	J
NM	754	SD	0.5	1	53	J	9.12	J	1.26	J	15500	J	6930	J	1250		0.0543	U	405	J
NM	756	SD	0.5	1	72.3	J	112	J	85.6	J	35200	J	12100	J	1600		0.313		17600	J
NM	757	SD	0	1.5	169	J	216	J	194	J	47800	J	20100	J	2690		0.34		15900	J
NM	758	SD	0.5	1	9.74	J	11.5	J	22.1	J	28800	J	5070	J	1920		0.201		2470	J
NM	760	SD	0	0.5	1.14	J	11.4	J	4.89	J	21900	J	154	J	693		0.0877	J	1120	J
NM	762	SD	0	2	0.605	UJ	2.34	J	3.01	J	21600	J	527	J	529		0.0714	J	1170	J
NM	766	SD	0	2	1.53	UJ	1.91	J	34.7	J	14300	J	723	J	575		0.0499	U	6940	J
NM	770	SD	0	0.5	1.1	J	8.9		13.3		98700		1760		6810		0.41		12200	
NM	771	SD	0	0.5	5.7	J	13.2		55		29200		5540		2700		1.3		7310	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 7.3 - Ninemile Surface Water

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				Units	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	**Location**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CC	273	SW			0.08	U	0.08	U	0.0345	U	6.7	J	0.45	J	2	J	0.5	U	5.7	U
CC	273	SW			0.25	U	0.5	U	0.05	U	10	U	1		2.5	U	0.1	U	2.5	U
CC	274	SW			0.09	U	0.08	U	0.0345	U	58.1	J	0.44	J	0.5	U	0.5	U	11.75	U
CC	276	SW			0.71		1		0.26		48.6	U	6.4		12.7		0.1	U	41.5	
CC	276	SW			2.6		0.42		0.35	J	6.8	J	2.8		10		0.5	U	48.1	J
CC	277	SW			0.66		1.8		0.35		39.85	U	3.4		14.2		0.1	U	47.2	
CC	277	SW			2.8		0.62		0.79		228		4.8		33.3		0.5	U	117	J
CC	278	SW			3.4		0.32		2.2		158		7.4		27.6		0.5	U	352	J
CC	278	SW			0.6		1	U	0.3		80		6.6		13		0.1	U	78	
CC	279	SW			5.3		2.7		0.0345	U	122		2		23.6		0.5	U	487	
CC	279	SW			0.8		1	U	0.9		90		12		14		0.1	U	141	
CC	280	SW			3.5	J	0.385	U	3.2		140		12.3		35		0.5	U	543	
CC	280	SW			4.6	J	0.55	J	3.9		183		16.4		37.6		0.2		656	
CC	281	SW			0.9		1	U	1.4		73		16.9		15		0.1	U	208	
CC	281	SW			4.1	J	0.21	U	4.3		111		30.3		29.4		0.5	U	673	
CC	281	SW			5.2		0.45	J	5.4		129		31.9		32.2		0.5	U	860	
CC	282	SW			1.2		1	U	2.1		98		32.5		30		0.1	U	312	
CC	282	SW			4.5	J	0.235	U	7.1		144		58.3		69.3		0.5	U	1110	
CC	282	SW			10.4		1.4	J	10.4		625		409		139		0.15	J	1530	
CC	283	SW			1.5		0.5	U	2.5		46.4	U	30.3		34.6		0.1	U	368	
CC	283	SW			4.7	J	0.255	U	7.8		132		66.3		67.3		0.5	U	1190	
CC	283	SW			5.4		0.34	J	12.7		144		92		81.4		0.5	UJ	1980	
CC	284	SW			1.6		0.5	U	2.8		48.3	U	36.2		34.3		0.1	U	402	
CC	284	SW			4.8	J	0.28	U	7.7		140		70.7		67.5		0.5	U	1290	
CC	284	SW			5.7		0.37	J	13.9		164		118		84.8		0.5	UJ	2010	
CC	285	SW			1.4		1	U	3.3		119		43.1		31		0.1	U	424	
CC	285	SW			5.3		0.23	J	17.7		171		105		80.5		0.5	UJ	2570	
CC	285	SW			5		0.175	U	9.6		122		77.6		59.9		0.5	U	1550	
CC	286	SW			1.8		0.5	U	4.9		53	U	44.6		39.1		0.1	U	660	
CC	286	SW			7.3		0.84	J	20.7		400		296		135		0.17	J	2970	
CC	286	SW			5		0.31	U	13.5		99.6	J	74.1		60		0.5	U	2140	
CC	287	SW			5.4		0.55	J	31		302		179		118		0.5	UJ	4270	
CC	287	SW			1.5		1	U	5.1		107		48.8		35		0.1	U	641	
CC	287	SW			4.7	J	0.165	U	17.8		104		74.7		70.8		0.5	U	2680	
CC	288	SW			1	U	0.115	U	6.7	J	106	J	64.3	J	42.9	J	0.08	U	5410	
CC	288	SW			1.4		1	U	5.2		108		51.1		37		0.1	U	675	
CC	288	SW			5		0.195	U	18.2		124		77.5		69.6		0.5	U	2750	
CC	288	SW			5		0.33	J	31.5		187		115		108		0.5	UJ	4410	
CC	290	SW			0.25	U	0.5	U	0.05	U	10	U	0.5	U	2.5	U	0.1	U	6.4	

Table 7.3 - Ninemile Surface Water

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				Units	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	**Location**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CC	291	SW			0.8		1	U	1.5		98		10.6		23		0.1	U	254	
CC	357	SW			0.5	U	0.3	U	52.9	J	1130	J	198		3170		0.1	U	9720	
CC	392	SW			5.2		2		2		42.75	U	27.4		81.1		0.1	U	180	
NM	291	SW			0.06	U	0.19		1.1		63.1	J	1.5		2.8	J	0.5	U	328	
NM	291	SW			0.1	U	1	U	0.1	U	22		0.3		2.5	U	0.1	U	5	U
NM	292	SW			0.1	U	1	U	0.2		42		1.9		2.5	U	0.1	U	34	
NM	292	SW			0.025	U	0.08	U	0.0345	U	145		1.7		7.4	J	0.5	U	6.8	U
NM	293	SW			0.1	U	1	U	5.4		78		19.7		61		0.1	U	1340	
NM	293	SW			0.14	U	0.08	U	15.8		370		15.6		94.1		0.5	U	4450	
NM	294	SW			0.1	U	1	U	13.2		166		19.1		141		0.1	U	4810	
NM	294	SW			0.18	U	0.25		16		324		75		126		0.5	U	6360	
NM	295	SW			0.25	U	0.5	U	6.5		14.8	U	13.9		51.5		0.1	UJ	1570	
NM	295	SW			0.135	U	0.08	U	16.4		268		15.8		37.4		0.5	U	4280	
NM	296	SW			0.35	U	0.1	U	10	J	45.7		26.8		66.6	J	0.1	U	2970	
NM	296	SW			0.205	U	0.1		38		7.55	U	80.5		51.3		0.5	U	8080	
NM	297	SW			0.25	U	0.5	U	11.4		24.1	U	46.4		47.3		0.1	U	2290	
NM	297	SW			0.26	U	0.12		40.4		14.7	U	129		42.2		0.5	U	8240	
NM	298	SW			0.1	U	1	U	10.5		37		48.2		42		0.1	U	2050	
NM	298	SW			0.26	U	0.18		42.7		9.55	U	138		45.1		0.15	J	8450	
NM	299	SW			0.13	U	0.31		0.0345	U	157		0.38	J	35		0.5	U	8.1	U
NM	299	SW			0.2		1	U	0.4		81		0.9		11		0.1	U	13	
NM	300	SW			0.215	U	0.37		0.5		144		5		72.8		0.055	U	82.5	J
NM	300	SW			0.3		1	U	0.3		531		36		52		0.1	U	65	
NM	301	SW			0.25	U	0.5	U	11.1		19.6	U	44.7		43.9		0.1	UJ	2120	
NM	301	SW			0.5	U	0.24		33		60.2	J	93.6		72		0.5	U	6520	J
NM	302	SW			0.25	U	0.5	U	0.05	U	10	U	0.61		2.5	U	0.1	U	5.4	
NM	302	SW			0.285	U	0.08	U	0.0345	U	7.6	J	1.6		1.1	J	0.5	U	13.3	U
NM	303	SW			0.45	U	0.08	U	29.5		16.7	U	60.2		59.3		0.055	UJ	5510	
NM	303	SW			0.3		1	U	10.2		43		46.1		37		0.1	U	1850	
NM	304	SW			0.495	U	0.115	UJ	28.2		17.75	U	53.2		52.8		0.5	U	5290	
NM	304	SW			0.25	U	0.5	U	11.3		22.55	U	42		39		0.1	U	2160	
NM	305	SW			0.3	U	0.115	U	12.5	J	80.8	J	61.6	J	35.6	J	0.08	U	6750	
NM	305	SW			0.6	U	0.08	U	27.4		22.9	U	47.6		44.6		0.06	UJ	5140	
NM	305	SW			0.25	U	0.5	U	11.2		22.35	UJ	39.7		36.8		0.1	U	2130	
NM	362	SW			0.016	U	0.115	U	0.41		14.4		1.1		0.4		0.08	U	115	
NM	362	SW			0.075	U	0.26	J	0.87		5.95	U	1.9		0.5	U	0.5	U	262	J
NM	363	SW			9.4		10.9		1520		16600		1650		26800		0.175	U	430000	J
NM	363	SW			0.1	U	8.1		1810	J	2220	J	270		25300		0.1	U	540000	
NM	368	SW			0.19	U	0.28	J	13.1		2320		21.2		1090		0.5	U	8530	J
NM	368	SW			0.016	U	0.42		15.9		94.4		113		8.6		0.08	U	2900	

Table 7.3 - Ninemile Surface Water

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				Units	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	**Location**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
NM	374	SW			0.225	U	0.96	J	156	J	314	J	1300	J	216	J	0.08	U	34100	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 8.1 - Mullan Surface Soil

				Analyte	Antimony		Arsenic		Cadmium		Iron		Manganese		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
SF	401	SS	0	0.5	19.7	J	42.1		11.9		29700		2130		1820	
SF	402	SS	0	0.5	20.3	J	35.4		13.8		33200		2100		2880	
SF	403	SS	0	0.5	16.5	J	28.2		1.1		22600		864		324	
SF	404	SS	0	0.5	1.5	J	5.6		0.55	J	12900		2750		97.2	
SF	405	SS	0	0.5	1.2	J	6.7		0.51	J	12500		2670		89.7	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 8.2 - Mullan Sediment

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
SF	208	SD	0	0.33	1.595	U	10.3	J	1.17	J	12800		114	J	1960		0.0728	J	428	J
SF	208	SD	0	0.5	3.755	U	24.4	J	2.66	J	15000		361	J	3060		0.268		560	J
SF	522	SD	0	0.04	4.54	UJ	15.1	J	21.8		53100		4610		4900	J	0.234	J	5570	J
SF	523	SD	0	0.5	2.035	UJ	9.62	J	7.1		55300		5270		6030	J	0.72	J	2530	J
SF	524	SD	0	1	1.555	U	11	J	2.58		18200		726		1310	J	0.31	J	439	J
SF	525	SD	0	0.25	1.165	UJ	10.1	J	3.83		21000		611		1600	J	0.225	J	1180	J
SF	526	SD	0	0.17	2.62	UJ	11	J	2.49		42500		1850		3880	J	0.199	J	1890	J
SF	526	SD	0.17	0.5	0.948	J	9.93	J	4.56		33700		1230		3070	J	0.537	J	1630	J
SF	528	SD	0	0.5	1.35	UJ	8.95	J	2.89		33400		861		2700	J	0.146	J	1510	J
SF	529	SD	0	0.17	2.27	UJ	18	J	5.92		28300		666		1430	J	0.154	J	1820	J
SF	530	SD	0	0.42	0.56	U	3.19	J	1.61		6160		527		391	J	0.0533	J	305	J
SF	531	SD	0	0.17	0.505	U	15.7	J	0.873	J	18800		200	J	1420	J	0.0794	J	636	J
SF	536	SD	0	1.5	14.7	J	28.4	J	81	J	121000		13800	J	15700		3.55		13700	J
SF	539	SD	0	1	0.498	U	4.43	J	0.143	J	10700		31.1	J	628		0.0515	U	471	J

Data Used to Calculate Human Health Exposure Point Concentrations
Table 8.3 - Mullan Surface Water

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	alifier	Analysis Value	lifier	Analysis Value	alifier	Analysis Value	lifier	Analysis Value	lifier	Analysis Value	alifier	Analysis Value	lifier	Analysis Value	alifier
SF	201	SW			0.72		0.5	U	0.62		80		3.7		6.3		0.1	UJ	120	
SF	201	SW			0.75	U	1.4	J	0.0345	U	16.6	U	0.41	J	2	J	0.05	U	26.1	U
SF	202	SW			0.5	U	0.5	U	0.05	U	31.2		0.5	U	2.5	U	0.1	UJ	5	U
SF	202	SW			0.25	U	0.135	UJ	0.0345	U	5	U	0.19	J	1	J	0.05	U	10.5	U
SF	204	SW			0.05	U	0.08	U	0.0345	U	5	U	0.026	U	1.1	J	0.05	U	12.2	U
SF	204	SW			0.5	U	0.5	U	0.05	U	141		4.4		42.2		0.1	UJ	9.5	
SF	205	SW			0.2	U	1	U	0.1	U	132		3.6		13		0.1	U	10	U
SF	205	SW			0.2	U	0.22		0.0345	U	52.9	J	0.27	J	12.1		0.05	U	18.4	U
SF	206	SW			2.7		0.6		0.0345	U	5	U	0.13	J	0.5	U	0.145	U	9.5	U
SF	206	SW			4		1	U	0.1	U	28		1.3		2.5	U	0.1	U	10	U
SF	207	SW			1.3	U	0.31		0.0345	U	5	U	0.097	J	1.2	J	0.05	U	9.8	U
SF	207	SW			1		1	U	0.1	U	20	U	0.2		2.5	U	0.1	U	10	U
SF	208	SW			0.5	U	0.5	U	0.05	U	75		2.7		27.5		0.1	UJ	7	
SF	208	SW			1.1	U	0.47		0.0345	U	103		2.3		127		0.05	U	24.2	U
SF	209	SW			3		0.91		0.0345	U	5	U	1.2		1.3	J	0.05	U	13.3	U
SF	209	SW			1		1	U	0.1	U	32		3.1		2.5	U	0.1	U	10	U
SF	209	SW			0.43	U	0.27		0.0205	U	85.3	J	0.66		38.5	J	0.08	U	4.7	
SF	210	SW			0.2	U	1	U	0.1	U	44		0.5		2.5	U	0.1	U	10	U
SF	210	SW			0.27	U	0.22	J	0.0345	U	5	U	0.24	J	0.5	U	0.05	U	11.1	U
SF	211	SW			0.46	U	0.315	U	0.145	U	5	U	0.38	J	1.4	J	0.05	U	31.8	U
SF	211	SW			0.2	U	1	U	0.1	U	30		1.5		2.5	U	0.1	U	10	U
SF	212	SW			2.5		1	U	0.1	U	20	U	2.7		2.5	U	0.1	U	10	U
SF	212	SW			3.5	J	0.215	U	2.9		6.1	U	12.6		1.8	J	0.05	U	13.9	U
SF	213	SW			0.2	U	1	U	0.1	U	38		0.9		7		0.1	U	10	U
SF	213	SW			0.29	U	0.08	U	0.0345	U	10	J	0.19	J	3.4	J	0.05	U	13.4	U
SF	214	SW			0.17	U	0.08	U	0.0345	U	9.9	U	0.33	J	2.6	J	0.05	U	29.5	U
SF	214	SW			0.2	U	1	U	0.1	U	28		0.7		2.5	U	0.1	U	10	U
SF	215	SW			0.032	U	0.31	J	0.021	U	94.5	J	3.4		18.9		0.08	U	9.1	J
SF	215	SW			4	J	0.16	U	4.4		48.7	U	32.8		60.2		0.05	U	28.2	U
SF	216	SW			0.67	U	0.1	U	0.08	U	23.1	U	1.3		5.8	J	0.05	U	47.5	
SF	218	SW			0.9		1	U	0.9		23		5		6		0.1	U	156	
SF	218	SW			19		1.8	J	0.92		5	UJ	7		17.8		0.05	U	202	
SF	219	SW			0.5	U	0.5	U	0.05	U	81.8	U	0.85		11.5		0.32		5	U
SF	219	SW			0.05	U	0.34	J	0.0345	U	5	UJ	0.17	J	0.5	U	0.05	U	14.4	U
SF	220	SW			0.43	J	0.41	J	0.17	J	92.3	J	5.8	J	11.75	U	0.08	U	38.8	
SF	220	SW			2.1	J	0.5	J	0.56		39.4	UJ	8.4		81.6		0.05	U	146	
SF	221	SW			0.05	U	0.2	J	0.0345	U	5	UJ	0.12	J	0.5	U	0.05	U	8.1	U
SF	221	SW			0.5	U	0.5	U	0.05	U	30.5	U	0.5	U	2.5	U	0.1	U	5	U
SF	222	SW			0.05	U	0.08	U	0.0345	U	5	UJ	0.15	J	0.5	U	0.05	U	7.4	U
SF	222	SW			0.5	U	0.5	U	0.05	U	20	U	0.5	U	2.5	U	0.1	U	5	U
SF	223	SW			0.5	U	0.5	U	8.8		20	U	12.2		7.5		0.1	U	1370	
SF	223	SW			3.1	J	0.41	J	7.3		5	UJ	9.5		1.55	U	0.05	U	1440	

Table 8.3 - Mullan Surface Water

				Analyte	Antimony		Arsenic		Cadmium		Iron		Lead		Manganese		Mercury		Zinc	
				UOM	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	**LocationID**	**Matrix**	**Beginning Depth**	**Ending Depth**	Analysis Value	alifier	Analysis Value	lifier	Analysis Value	alifier	Analysis Value	lifier	Analysis Value	lifier	Analysis Value	alifier	Analysis Value	lifier	Analysis Value	alifier
SF	224	SW			0.5	U	0.5	U	0.05	U	38	U	24.7		6		0.1	U	5	U
SF	224	SW			0.92	U	0.32	J	0.0345	U	5	UJ	10.5		0.6	U	0.05	U	10.3	U
SF	225	SW			0.42	U	0.08	U	0.0345	U	76.4	UJ	0.1	J	0.5	U	0.05	U	9.6	U
SF	225	SW			0.2	U	1	U	0.1	U	51		0.2	U	2.5	U	0.1	U	10	U
SF	226	SW			0.18	U	0.08	U	0.0345	U	10	U	1.7		4.6	J	0.05	UJ	14.7	J
SF	226	SW			0.2	U	1	U	0.1	U	65		1.8		2.5	U	0.1	U	10	U
SF	227	SW			0.24	J	0.57	J	0.36	J	93.5	J	6		21.7		0.08	U	66.9	
SF	227	SW			1.8	U	0.28		0.79		51	J	11		50.6		0.05	U	168	
SF	228	SW			1.9	U	0.37		0.88		193		7.3		60.9		0.05	U	225	
SF	228	SW			0.3	J	0.34	J	0.32	J	84.3	J	5.1		19.1		0.08	U	67.5	
SF	229	SW			0.3		1	U	0.1	U	49		1.2		2.5	U	0.1	U	24	
SF	229	SW			0.3	U	0.08	U	0.0345	U	5	U	0.58		0.5	U	0.05	U	29.6	
SF	230	SW			0.2	U	1	U	0.1	U	51		0.3		2.5	U	0.1	U	10	U
SF	230	SW			0.23	U	0.23		0.0345	U	5	U	0.13	U	0.5	U	0.05	U	10.9	U
SF	231	SW			0.32	U	0.23		0.0345	U	5	U	0.026	U	0.5	U	0.05	U	10.9	U
SF	275	SW			0.032	U	0.115	U	0.16	J	40.5	J	2.8	J	4	J	0.08	U	43.7	
SF	316	SW			0.4		1	U	0.1	U	255		3.2		28		0.1	U	10	U
SF	317	SW			0.032	U	0.115	U	0.021	U	77.9	J	0.11	U	3.2	J	0.08	U	0.94	J
SF	318	SW			0.032	U	0.115	U	15.2		25.8	J	43.9		107		0.08	U	2120	
SF	319	SW			0.032	U	0.115	U	2.5	J	15.1	J	18.7		31.6		0.08	U	334	
SF	320	SW			0.032	U	0.115	U	0.021	U	5.6	U	0.47	J	1	J	0.08	U	2.6	J
SF	321	SW			0.032	U	0.115	U	0.021	U	5.6	U	3		0.65	J	0.08	U	9.8	J
SF	328	SW			1.8		1	U	3.6		62		11.9		6		0.1	U	121	
SF	328	SW			1.9	J	0.45	J	0.64		5	UJ	7.1		1.9	U	0.05	U	110	
SF	398	SW			0.28		0.33		0.4		84.9		5.2		19		0.08	U	84.3	

Data Used to Calculate Human Health Exposure Point Concentrations

Table 9.1 - Blackwell Island Surface Soil

					Analyte Name	Antimony		Arsenic		Cadmium		Iron		Manganese		Zinc	
					UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**Location ID**	**Matrix**	**Location Cross Reference**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA021	8	SS	21-8U.08	0	0.08	1.2		9.7		2.2		19500		859		265	
CUA021	9	SS	21-9U.08	0	0.08	1.8		12.4		9.5		19900		1210		1040	
CUA021	10	SS	21-10U.08	0	0.08	1.5		16.7	J	3.2		29600		1300		577	
CUA021	11	SS	21-11U.08	0	0.08	1.2	J	12.5	J	6.1		24800		1260		777	
CUA021	12	SS	21-12U.08	0	0.08	3.1	J	20.3	J	13.7		29100		1780		1640	
CUA021	13	SS	21-13U.08	0	0.08	1.1	J	15.7	J	2.8		28600		1160		448	
CUA021	14	SS	21-14U.08	0	0.08	1.9	J	14.2	J	12.5		26300		1380		1530	

Data Used to Calculate Human Health Exposure Point Concentrations
Table 9.2 - Blackwell Island Sediment

					Analyte Name	Antimony		Arsenic		Cadmium		Iron		Manganese		Zinc	
					UOM	mg/kg		mg/kg		mg/kg		mg/kg		mg/kg		mg/kg	
Site	**Location ID**	**Matrix**	**Location Cross Reference**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA021	1	SD	21-1S/W.5	0	0.5	5.5		59.3		17		61100		2920		2460	
CUA021	2	SD	21-2S/W.5	0	0.5	5		74		7.7		66800		3040		2030	
CUA021	3	SD	21-3S/W.5	0	0.5	3.4		77		11.1		64100		5470		2080	
CUA021	4	SD	21-4S/W.5	0	0.5	1.4		53.4		2.1		58300		3330		441	
CUA021	5	SD	21-5S/W.5	0	0.5	3.1		59.2		12.6		53300		3920		1910	
CUA021	6	SD	21-6S/W.5	0	0.5	2.3		37.2		9.6		41700		2180		1590	
CUA021	7	SD	21-7S/W.5	0	0.5	4.2		56.7		8.7		61300		2320		1880	
CUA021	22	SD	21-22B01	0	1	4.1	J	39.8		21.6		41000		4380		2020	
CUA021	23	SD	21-23B01	0	1	2.3		63.5		17.6		51500		6300		2020	
CUA021	24	SD	21-24B01	0	1	3.1		83.4		17.2		65600		7480		2170	
CUA021	25	SD	21-25B01	0	1	3.5		52.2		11.3		48000		3720		1900	
CUA021	26	SD	21-26B01	0	1	2.4		19.8		16.6		31600		1380		2080	
CUA021	27	SD	21-27B01	0	1	3.3		45.8		17.3		41500		3850		2050	
CUA021	28	SD	21-28B01	0	1	2.4		38.8		15.5		42200		2840		2170	

Data Used to Calculate Human Health Exposure Point Concentrations

Table 9.3 - Blackwell Island Surface Water

					Analyte Name	Antimony		Arsenic		Cadmium		Iron		Manganese		Zinc	
					UOM	ug/l		ug/l		ug/l		ug/l		ug/l		ug/l	
Site	**Location ID**	**Matrix**	**Location Cross Reference**	**Beginning Depth**	**Ending Depth**	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ	Analysis Value	LQ
CUA021	1	SW	21-1S/W.5			1.3	U	5.6		7		17400		890		934	
CUA021	2	SW	21-2S/W.5			1.4		2.9		4.2		9470		561		564	
CUA021	3	SW	21-3S/W.5			2.8		20.8		47.9		60900		6980		5010	
CUA021	4	SW	21-4S/W.5			1.5		7.6		4.9		15200		942		876	
CUA021	5	SW	21-5S/W.5			2.3		14.8		42.5		57800		6300		5720	
CUA021	6	SW	21-6S/W.5			1.6		9.8		13.3		29800		1340		1890	
CUA021	7	SW	21-7S/W.5			2		12.4		20.3		45500		2530		2690	

APPENDIX F

Distribution Checks and Statistical Summaries of Data by Geographical Area and Medium

Appendix F

Distribution Checks and Statistical Summaries of Data by Geographical Area and Medium

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This appendix contains the MTCA*Stat v*.2.1 tables. MTCA*Stat v*.2.1 was used in this risk assessment to determine the distribution of the data and to calculate a UCL_{95} for normally distributed data. If the data were lognormally distributed or neither lognormally nor normally distributed, Systat *v*.9 was used to calculate the UCL_{95} (see Appendix G for Systat *v*.9 results). The data in the Table 3 series in Appendix A are taken from the information in this appendix with the exception of the 95 percent UCL (UCL_{95}) for data with a lognormal distribution and data with neither a normal nor a lognormal distribution. UCL_{95} values for these two data distributions are provided in Appendix G.

Lower Basin Residential Surface S

Antimony

Analytical Results (mg/kg)
5.7
0.1
1
12
0.495
16.7
10
22.2
25.6
10.5
18.1
0.47
0.54
0.21
0.65
0.21
0.235
0.345
0.5
0.5
2.2
1.6
0.5
1
3.8
0.6
15.9

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	27	Mean	5.62
Censored		Lognormal mean	7.18
Detection limit or PQL		Std. devn.	7.767855
Method detection limit		Median	1
TOTAL	27	Min.	0.1
		Max.	25.6

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.9015. This is less than the tabled value of 0.923
Reject normal distribution.
W value is 0.728. This is less than the tabled value of 0.923

UCL (based on t-statistic) is 8.17

Lower Basin Residential Surface Soil

Arsenic

Analytical Results (mg/kg)
108
14.5
90.9
78.5
4.8
86.6
65.2
115
95.6
57.3
93.3
10.9
13.6
9
4.8
7.5
6.1
8.7
10.3
4.6
5.6
5.6
9.5
5.6
4.3
50.7
8.3
18.4

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	28	Mean	35.47
Censored		Lognormal mean	37.27
Detection limit or PQL		Std. devn.	39.00628
Method detection limit		Median	10.6
TOTAL	28	Min.	4.3
		Max.	115

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8378. This is less than the tabled value of 0.924
Reject normal distribution.
W value is 0.7487. This is less than the tabled value of 0.924

UCL (based on t-statistic) is 48.03

Lower Basin Residential Surface Soil

Cadmium

Analytical Results
(mg/kg)

10.8
0.35
12.5
15.2
0.1
15.5
5.7
14.6
12
9.4
12.2
0.04
0.04
0.04
0.04
0.04
0.04
0.09
0.09
1.5
0.5
2
2.4
0.5
2.3
6.4
0.1
14.7

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	28	Mean	4.97
Censored		Lognormal mean	16.16
Detection limit or PQL		Std. devn.	5.945193
Method detection limit		Median	1.75
TOTAL	28	Min.	0.04
		Max.	15.5

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8388. This is less than the tabled value of 0.924
Reject normal distribution.
W value is 0.7648. This is less than the tabled value of 0.924

UCL (based on t-statistic) is 6.88

Lower Basin Residential Surface Soil

Iron

Analytical Results (mg/kg)
60800
9710
70100
34200
93000
83100
45700
57500
21900
20000
20000
14900
20000
17000
20300
21500
12690
11740
14190
16170
13300
12100
27200
15500

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	25	Mean	29992.00
Censored		Lognormal mean	29426.68
Detection limit or PQL		Std. devn.	23945.95
Method detection limit		Median	20000
TOTAL	25	Min.	9710
		Max.	93000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8856. This is less than the tabled value of 0.918
Reject normal distribution.
W value is 0.7489. This is less than the tabled value of 0.918

UCL (based on t-statistic) is 38186.3

Lower Basin Residential Surface Soil

Manganese

Analytical Results (mg/kg)
4560
260
5300
2360
6620
6190
3250
3830
360
400
356
283
365
259
371
362
533
354
436
681
391
535
1890
420
597

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	25	Mean	1638.52
Censored		Lognormal mean	1576.55
Detection limit or PQL		Std. devn.	2054.94
Method detection limit		Median	436
TOTAL	25	Min.	259
		Max.	6620

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.797. This is less than the tabled value of 0.918
Reject normal distribution.
W value is 0.6911. This is less than the tabled value of 0.918

UCL (based on t-statistic) is 2341.72

Lower Basin Residential Surface Soil

Zinc

Analytical Results (mg/kg)
2670
311
2430
2420
37.1
2500
1070
2840
2900
1420
1540
54.2
54.4
54.5
34.7
54.8
39.4
58
52.4
158
80.3
217
296
56.9
232
937
82.3
1160

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	28	Mean	848.57
Censored		Lognormal mean	1123.78
Detection limit or PQL		Std. devn.	1046.316
Method detection limit		Median	224.5
TOTAL	28	Min.	34.7
		Max.	2900

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8493. This is less than the tabled value of 0.924
Reject normal distribution.
W value is 0.7466. This is less than the tabled value of 0.924

UCL (based on t-statistic) is 1185.31

Lower Basin Residential Tap Water
Arsenic

Site ID	**Static Result** (ug/L)	**Purged Result** (ug/L)	**Average Result** (ug/L)	
R007	0.26	0.1 U	0.18	
R025	0.17 U	0.1 U	0.135	
R115	0.25 U	0.25 U	0.25	
R144	0.83	1.1	0.965	
			0.965	**MAX**
			0.3825	**Average**
			0.3912	**Std Dev**
			4	**Count**
			2	**Count^0.5**
			2.353	**t-statistic**
			0.8428	**UCL_{95}normal**

Lower Basin Neighboorhood and Public Recreational Soil/Sediment

Antimony

Analytical Results

(mg/kg)

8.9	15.9	0.5	19	10.6	50.8	28	27.5	15	21	43	40
6.9	19.1	0.5	27	10.8	26.7	27.1	36.1	11	27	18	24
12.7	5.7	1.8	15	15	2	36.2	13.5	18	26	16	5.6
12.8	6	0.5	12	36.1	18.6	51.5	17	15	19	20	25
11	13.9	19.4	21	23.9	21.9	12.2	39.9	13	27	29	8.4
20.4	9	15.5	20	58.6	0.5	3.7	31.1	23	41	35	15
21.6	11.7	14.2	36	19.9	28.2	8.9	34.7	28	25	29	7.8
18.7	19.4	16.2	50	22.2	0.49	23.5	42.7	20	36	29	0.5
22.4	18.9	18.1	31	2.5	21.1	0.5	26.3	16	44	26	4.6
17.7	21.1	25.9	36	0.5	35.5	23.8	33	31	37	26	1.5
23.3	21.2	17.7	20	3	9	0.5	17	52	51	36	0.5
34.7	12	27.9	30	7	36.9	18.8	17.9	28	49	38	0.5
27	28.2	17.8	47	1.4	46	41.1	14.1	35	0.5	42	0.5
27.9	24.4	15.1	37	16.8	31	35.4	13.4	15	14	49	1
20.2	21.6	33.3	19	21.7	5.7	39.3	8.3	21	12	50	4.5
0.5	20.8	22.4	13	17.9	16.1	36.9	22.1	27	21	14	1.2
4.3	21.3	20.4	57	13.1	16.7	17.7	34.3	24	16	0.5	8
2.1	15.6	40.7	32	13.1	0.5	3.3	26.6	29	14	53	56
0.48	13.7	42.2	37	24	32.1	19.9	40	30	20	29	0.5
1.3	10.4	19.3	30	25.1	5.5	16	46.5	28	23	15	3.4
30.3	6.6	14.6	42	26.8	26.2	22.8	23.8	32	34	13	2.4
35.8	7.9	14.4	18	24.4	36.1	13.8	31	21	25	5.9	3.5
30.1	26.2	16.5	35	22	22	19.8	14.1	49	31	25	1.8
37.9	25.7	17.1	46	1.3	44.5	27.3	21	21	25	16	16
32.3	21.1	17.5	20	17.4	48.5	14.1	13	43	49	35	3.4
0.5	21.5	18.1	19	23.7	25.8	29.8	12.6	67	20	35	
2.4	20.9	23.5	18	0.5	2.1	20.8	11.7	49	52	27	
3.3	16.6	22.4	20	23	14.3	21	21	12	47	28	
0.5	54.6	20.5	18	0.5	20.7	38.8	29.7	16	43	46	
14.2	16.3	21	19	26.7	0.5	15.1	17.3	10	39	36	
19.3	22.3	14.7	17	33.1	25.5	24.7	29.2	30	13	25	
22.7	24.7	20.4	31	31.7	0.5	4.5	40.3	17	24	45	
20.7	1.2	18.4	10	44.2	21.6	15.4	28.7	11	74	39	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	388	Mean	22.08
Censored		Lognormal mean	29.41
Detection limit or PQL		Std. devn.	13.59137
Method detection limit		Median	20.85
TOTAL	388	Min.	0.48
		Max.	73.7

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -26.7947. This lies outside the tabled values of 1.6275 and -2.275
Assume normal distribution.
Y value is -1.928. This lies within the tabled values of 1.6275 and -2.275

UCL (based on t-statistic) is 23.22

Lower Basin Neighboorhood and Public Recreational Soil/Sediment

Arsenic

Analytical Results
(mg/kg)

93.7	116	91	75	105	5.9	133	139	101	154	118	146	22
84.8	132	9.9	95	83	142	209	139	132	146	145	119	10
128	128	7.9	99	95	147	2.4	163	134	185	108	122	58
108	85	8.4	113	70	130	172	85	162	88	113	170	16
96.6	80	9.7	97	115	209	18	135	136	86	143	115	
135	100	8.9	92	83	375	136	19	126	45	126	127	
138	95	106	86	149	95	160	154	151	137	117	144	
110	117	139	119	256	9.4	105	166	109	93	72	56	
148	114	106	117	160	155	136	168	97	132	111	102	
102	105	103	217	492	250	304	128	64	105	84	123	
189	167	111	329	144	1.8	52	148	120	167	147	108	
351	110	112	213	151	188	14	178	142	118	79	129	
150	130	101	163	21	4.3	67	112	86	111	143	88	
216	132	151	115	14	130	154	143	122	155	98	26	
274	138	110	114	27	153	4	127	180	165	162	73	
5.4	150	111	163	60	45	179	126	120	138	153	95	
13.2	144	153	256	18	148	7	165	133	137	127	88	
17.5	139	121	104	73	162	152	96	107	111	123	79	
5.8	106	109	75	89	112	194	141	208	164	122	3	
36.4	88	219	154	84	22	138	161	132	137	111	8.4	
159	62	229	107	64	123	111	99	182	184	106	4.9	
167	41	97	110	72	132	146	124	85	9.5	132	2.6	
170	64	135	103	125	1.5	74	112	110	93	137	3.9	
203	121	84	123	135	148	20	172	124	46	143	3.8	
190	117	100	116	150	69	254	115	159	136	136	6.8	
15.3	103	98	150	127	118	122	150	318	80	74	12	
32.2	88	85	187	135	165	112	153	137	156	6.3	4.5	
75.4	102	94	109	3.9	100	128	135	148	113	68	35	
17.5	106	113	100	148	173	124	151	114	169	154	158	
263	114	97	86	168	146	164	81	123	143	69	3.3	
124	88	95	100	3.6	94	108	117	171	128	150	24	
121	94	111	98	153	10	141	152	84	129	18	16	

MTCA*Stat* 2.1

Number of samples	Uncensored values		
Uncensored	388	Mean	114.51
Censored		Lognormal mean	140.92
Detection limit or PQL		Std. devn.	61.85832
Method detection limit		Median	116
TOTAL	388	Min.	1.5
		Max.	492

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -35.3013. This lies outside the tabled values of 1.6275 and -2.275
Reject normal distribution.
Y value is -14.0119. This lies outside the tabled values of 1.6275 and -2.275

UCL (based on t-statistic) is 119.72

Lower Basin Neighboorhood and Public Recreational Soil/Sediment

Cadmium

Analytical Results

(mg/kg)

12.7	23	42	20	33	31	23	84	36
8.4	30	31	22	56	42	8.8	4.3	28
16.3	37	29	15	38	23	9.7	25	31
19.6	38	30	16	2.8	45	17	13	44
14.2	32	33	8.2	16	27	26	25	34
29.7	28	29	21	28	40	36	13	53
27.5	18	31	15	0.1	36	61	21	77
19.1	16	23	11	38	13	36	23	89
28.8	9.3	21	1.5	12	21	31	41	48
18	7.5	24	0.4	36	2.2	58	40	77
37.7	7.4	33	2.5	33	20	81	55	83
10.2	32	30	7.7	44	22	54	40	82
37.6	32	61	1.4	52	44	63	39	62
61.6	29	21	11	75	30	4.8	75	3.3
10.7	31	14	14	68	36	8.1	58	18
2.2	32	45	13	1.9	55	27	87	17
6.5	30	48	8.2	22	75	33	83	16
5.1	40	59	10	34	63	29	83	25
1.9	36	84	14	0.1	94	42	80	0.3
2.3	47	76	18	34	43	55	34	2.4
38.3	37	86	14	2.6	30	47	67	1.4
41.3	0.6	41	10	32	13	49	25	0.2
32.7	0.3	36	14	26	18	100	59	0.4
43.3	0.2	74	0.6	54	14	57	31	0.1
42.2	1	41	19	44	33	97	19	1.1
2.3	0.2	42	30	52	18	104	23	1.5
4.2	41	27	0.3	32	42	81	36	0.6
4.7	32	82	30	2.6	49	28	37	4.5
3.6	28	53	1.9	13	74	36	40	15
8.2	36	58	39	33	59	12	34	0.3
26.6	39	29	35	0.1	88	52	41	3.2
37	47	54	55	27	53	18	51	1.6
30.5	44	20	67	1.9	51	21	80	2.7
22.4	53	49	30	28	9.3	25	70	1.2
42.2	38	86	41	46	26	35	83	7.4
14.6	36	27	1.6	54	16	31	96	2
17.8	59	25	24	51	37	51	88	
23.7	38	41	28	50	35	47	33	
22.8	32	32	0.1	29	39	71	1	
27.4	54	35	37	4.8	39	61	4.7	
18	58	39	0.4	19	60	89	40	
25.4	28	32	42	19	64	86	24	
25.9	14	37	40	20	87	94	20	
35	30	21	20	26	57	105	3.6	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	388	Mean	33.14
Censored		Lognormal mean	51.80
Detection limit or PQL		Std. devn.	23.72412
Method detection limit		Median	30.5
TOTAL	388	Min.	0.1
		Max.	105

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -25.0406. This lies outside the tabled values of 1.6275 and -2.
Reject normal distribution.
Y value is -3.5563. This lies outside the tabled values of 1.6275 and -2.2

UCL (based on t-statistic) is 35.13

Lower Basin Neighboorhood and Public Recreational Soil/Sediment

Iron

Analytical Results

(mg/kg)

54800	1E+05	85300	67000	15900	18900	1E+05	2E+05	2E+05	17100
51200	1E+05	76300	94800	1E+05	1E+05	2E+05	2E+05	75400	
74000	1E+05	96300	62200	1E+05	70900	1E+05	1E+05	11400	
69700	1E+05	83500	86100	48000	1E+05	1E+05	2E+05	1E+05	
64900	1E+05	87500	72000	1E+05	84500	2E+05	2E+05	1E+05	
97700	1E+05	95000	1E+05	2E+05	1E+05	1E+05	2E+05	47700	
93500	1E+05	1E+05	1E+05	93700	2E+05	81100	2E+05	65500	
78300	72600	85800	89200	27400	1E+05	39100	2E+05	38700	
#####	81000	85400	1E+05	96500	1E+05	55200	13500	1E+05	
74300	60700	78400	80500	1E+05	1E+05	80100	72500	1E+05	
#####	53300	62800	80900	4450	1E+05	1E+05	73000	1E+05	
#####	68200	73500	38400	1E+05	2E+05	1E+05	85600	1E+05	
#####	1E+05	86600	31600	55200	53500	2E+05	62000	2E+05	
#####	1E+05	89900	31700	1E+05	62159	1E+05	71000	1E+05	
96700	1E+05	1E+05	29600	1E+05	17200	1E+05	92000	2E+05	
18100	97200	68300	31800	88100	90100	2E+05	1E+05	94500	
15900	1E+05	60500	46300	2E+05	1E+05	2E+05	1E+05	1E+05	
18600	79700	95300	62700	2E+05	2E+05	1E+05	1E+05	2E+05	
16700	79100	1E+05	54300	79600	1E+05	2E+05	1E+05	2E+05	
20900	79300	2E+05	38300	15400	1E+05	57000	1E+05	2E+05	
#####	1E+05	2E+05	45800	1E+05	2E+05	69000	2E+05	1E+05	
#####	82400	2E+05	76561	1E+05	2E+05	98700	86800	24800	
#####	19900	2E+05	91800	7870	1E+05	1E+05	2E+05	98100	
#####	17200	1E+05	85966	1E+05	2E+05	1E+05	2E+05	63700	
#####	12700	98600	87495	21700	1E+05	2E+05	2E+05	54400	
25300	22200	2E+05	83007	1E+05	1E+05	2E+05	2E+05	48200	
29800	15700	1E+05	11500	1E+05	66400	1E+05	90100	12900	
43700	94800	1E+05	82200	1E+05	70286	1E+05	1E+05	15500	
22400	81800	74200	1E+05	1E+05	83000	2E+05	1E+05	17800	
80000	74900	2E+05	7370	2E+05	1E+05	1E+05	1E+05	12500	
95900	80600	1E+05	1E+05	50500	1E+05	2E+05	58100	16700	
#####	98700	1E+05	17100	23900	1E+05	3E+05	70900	16100	
#####	1E+05	1E+05	1E+05	55400	2E+05	2E+05	75600	16200	
94700	93200	1E+05	1E+05	1E+05	2E+05	66500	2E+05	23100	
#####	1E+05	81100	1E+05	10000	1E+05	87000	1E+05	14600	
75400	1E+05	1E+05	2E+05	1E+05	2E+05	58100	1E+05	27500	
82700	91400	2E+05	2E+05	16200	1E+05	1E+05	1E+05	54900	
79600	2E+05	82600	71800	2E+05	1E+05	64000	1E+05	11300	
93900	1E+05	75200	16200	2E+05	47000	79100	1E+05	20400	
#####	1E+05	63700	1E+05	1E+05	53300	88200	2E+05	18800	
#####	2E+05	85000	1E+05	1E+05	74200	2E+05	2E+05	20300	
95700	2E+05	70700	6080	1E+05	1E+05	83300	2E+05	17700	
#####	78900	79500	2E+05	75800	1E+05	87700	2E+05	35500	

MTCA*Stat* 2.1

Number of samples	Uncensored values		
Uncensored	388	Mean	########
Censored		Lognormal mean	########
Detection limit or PQL		Std. devn.	50951.62
Method detection limit		Median	101000
TOTAL	388	Min.	4450
		Max.	256000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -15.2283. This lies outside the tabled values of 1.6275 and -2.275
Reject normal distribution.
Y value is 1.6645. This lies outside the tabled values of 1.6275 and -2.275

UCL (based on t-statistic) is 105568.97

Lower Basin Neighboorhood and Public Recreational Soil/Sediment

Manganese

Analytical Results

(mg/kg)

4370	7470	####	5940	9470	####	6340	####	####
3810	8570	9590	5440	####	####	2980	708	8190
5510	9660	7520	6590	9140	####	4150	5130	9400
5800	9890	7930	####	759	####	6060	5210	####
4930	9700	8250	9180	7760	####	8950	8080	####
8470	9500	7090	####	8940	####	####	5490	####
8100	5480	7180	7810	141	####	####	6300	####
6900	6050	8290	7770	####	4830	####	8130	####
8940	4530	5960	1350	4120	6680	####	####	####
6600	3320	7710	979	9660	783	####	####	####
10300	4330	8780	1410	####	6850	####	8040	####
9370	9020	8860	1480	9310	8970	####	####	####
10200	8580	####	1150	####	####	####	####	8290
11600	8100	6780	3860	####	9410	3720	####	1280
5740	7920	5490	5920	7340	####	5560	9810	6140
1020	8280	####	4970	467	####	8650	####	4500
780	8510	####	3220	8510	####	####	####	5450
1200	8560	####	3730	9880	####	7740	####	5010
739	9240	####	5630	97.5	####	####	####	233
881	####	####	6910	####	####	####	7260	779
10000	9870	####	5750	1160	####	####	####	377
12800	722	8900	7290	####	6320	####	4430	197
9380	622	8040	7090	8040	7300	####	####	259
13900	511	####	195	####	5590	####	5000	256
13100	749	####	6900	####	9190	####	5800	341
1290	582	8400	####	####	7860	####	6810	805
2110	####	6150	271	4160	####	####	####	373
2880	9280	####	####	592	####	4930	####	1810
1450	7620	####	386	4650	####	6890	9470	4780
4810	9250	####	9970	####	####	3780	####	243
10100	####	8320	####	92.3	####	####	9000	875
13100	####	####	####	8200	####	6390	####	586
12100	####	8460	####	322	####	6560	####	797
9500	####	####	####	####	3940	7860	####	432
12100	####	####	6270	####	4500	####	####	2820
6870	####	9390	348	####	5670	6490	####	728
7240	####	7990	8380	####	8890	6650	####	
8010	9490	6680	9560	####	9740	####	5710	
9860	9030	9300	154	7280	####	####	112	
11000	####	7190	####	1700	####	####	3540	
7900	####	8730	138	6900	####	####	9840	
7570	6220	6790	####	5430	####	####	4100	
7840	5700	7120	####	8470	####	####	5660	
9020	8350	4810	4270	9040	####	####	1370	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	388	Mean	9402.87
Censored		Lognormal mean	12302.22
Detection limit or PQL		Std. devn.	5745.032
Method detection limit		Median	8875
TOTAL	388	Min.	92.3
		Max.	26400

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -24.3552. This lies outside the tabled values of 1.6275 ar
Assume normal distribution.
Y value is 0.6914. This lies within the tabled values of 1.6275 and -

UCL (based on t-statistic) is 9886.44

Lower Basin Neighboorhood and Public Recreational Soil/Sediment

Zinc

Analytical Results

(mg/kg)

1800	4460	6670	###	5300	4700	4060	####	5720
1070	4630	4960	###	8780	7380	1440	589	4330
2290	5600	4630	###	6330	2630	1890	4210	5020
2940	5910	4670	###	577	7280	2580	2910	7810
1960	4990	5380	###	2330	3850	4450	3750	6380
4780	4330	4730	###	4340	6280	6570	1730	8760
4550	2370	4770	###	14.3	5580	####	2650	####
3160	2600	3340	###	5530	1690	6000	4030	####
4730	1520	2990	267	2550	2540	4920	6390	8360
3050	1160	3360	148	6490	562	9370	5750	####
5070	1350	5210	450	4610	3180	####	6650	####
1580	5810	4400	###	6920	3260	8010	7030	####
5830	5910	9630	315	8160	7920	####	7070	8260
6780	5280	2950	###	####	5270	1440	####	826
1120	5600	1890	###	7690	5240	1700	6950	4230
448	5690	6850	###	587	9620	4700	####	2840
800	4390	7520	###	3440	####	5310	####	1720
772	4480	####	###	4900	9950	4180	####	2930
370	5570	####	###	19.5	####	7400	####	75.1
411	7350	####	###	4870	6720	9230	5960	560
5360	5420	####	###	510	4400	8290	9850	215
5810	123	6700	###	6040	1920	8130	7340	45.1
4530	94.9	5650	###	3630	2250	####	9560	70
6030	65.7	####	184	8610	2360	9490	2790	37.7
5900	155	6710	###	5780	6300	####	2590	256
429	74.1	6810	###	8690	3010	####	3800	389
731	6600	4580	106	2870	6830	####	5610	90.9
848	4490	####	###	522	8800	3970	5430	1380
649	3930	8300	421	2090	####	5600	7160	4310
1370	5470	8910	###	5120	9320	2550	6280	69.3
4510	5730	4720	###	30.8	####	8340	7100	757
6150	7910	8550	###	3150	8030	1990	7120	462
5150	7240	2790	###	378	7170	2710	####	772
3730	8710	7480	###	5310	1400	4270	####	306
6540	6240	####	###	6830	2120	5680	####	1960
2030	5650	3720	452	8830	2430	4270	####	465
2700	9740	3310	###	8090	6500	5390	####	
3880	6930	6320	###	7730	6250	8180	5940	
3520	5810	5240	65	4400	6300	####	235	
4660	9690	5310	###	1010	7060	####	4900	
3720	####	6910	195	2990	7260	####	6140	
4520	4220	4830	###	2220	9260	####	2310	
4650	2280	6370	###	4020	####	####	2510	
6290	4690	3750	###	3790	8540	####	890	

MTCA*Stat* 2.1

Number of samples	Uncensored values		
Uncensored	388	Mean	5337.10
Censored		Lognormal mean	7421.19
Detection limit or PQL		Std. devn.	3974.565
Method detection limit		Median	4750
TOTAL	388	Min.	14.3
		Max.	21800

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -20.7149. This lies outside the tabled values of 1.6275 a
Reject normal distribution.
Y value is -7.7719. This lies outside the tabled values of 1.6275 ar

UCL (based on t-statistic) is 5671.65

Lower Basin Neighborhood and Public Recreational Exposures to Surface Water

Arsenic

Analytical Results (ug/L)

8.6	8.5	36.2	6.8
6.8	38.9	159	9.1
8	11.3	71.4	3.7
15.3	57	66.3	38
6.3	173	116	8.2
8.2	17.3	181	22.3
9.7	25.2	61.6	24.9
17.2	136	35.7	11.8
22.5	130	67.3	22.5
40.5	263	107	61.6
33.9	600	34	5.2
52	254	137	18.4
85.5	178	26.9	27.2
140	69.3	33	28.4
130	41.8	36.5	26.8
75.5	41.8	131	23.5
98.7	25.5	133	15
3.4	44	141	8.6
6.2	55.6	114	18.2
4	56.7	59	3.5
4.6	70.6	32.8	
3.8	67.3	53.3	
74.9	108	117	
54.1	139	125	
53.6	157	34.2	
128	108	45.1	
49.3	109	167	
34.6	66.2	27	
31.6	106	107	
31.8	117	149	
16.5	145	131	
9.3	116	60.4	
35.8	43.5	25.6	
8.4	69.9	135	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	122	Mean	68.02
Censored		Lognormal mean	75.64
Detection limit or PQL		Std. devn.	73.69277
Method detection limit		Median	43.75
TOTAL	122	Min.	3.4
		Max.	600

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is 0.1902. This lies within the tabled values of 1.3558 and -2.508

UCL (based on t-statistic) is 79.08

Lower Basin Neighborhood and Public Recreational Exposures to Surface Water

Cadmium

Analytical Results (ug/L)

3.6	3.7	16	9.6
1.8	23.2	68.4	16.3
1.3	7.4	41.7	8.5
2.4	7.6	34.1	14.7
0.5	14.1	95	9.5
1.8	4.6	83.5	19.9
2	2.8	58.5	24.7
3.7	15.7	15.8	8.9
4.7	56.8	34.9	26.3
7.3	338	20.3	65.4
7.1	1200	17	8.2
13.2	66.2	72.6	34.2
56.3	166	16.6	50.5
27.9	68.9	13.8	41.4
206	31	20.6	30.8
30.7	25.5	52.8	55.4
20.6	12.6	47.2	36.5
0.5	33.9	40.3	8.8
2.9	34	54	29.6
0.5	47.9	41.9	5.6
1.8	48.8	17.3	
0.5	45.8	44.9	
170	109	408	
26.5	60.4	84.9	
26.5	41.2	25	
75.8	49.4	11.9	
137	85.2	214	
65.8	46.5	30.1	
52.4	35.1	58	
59.8	44.7	115	
38.1	139	81.2	
11.6	41.9	44	
8.8	138	36.7	
3.5	23.9	25.6	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	122	Mean	54.82
Censored		Lognormal mean	62.86
Detection limit or PQL		Std. devn.	120.0504
Method detection limit		Median	30.75
TOTAL	122	Min.	0.5
		Max.	1200

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -3.6937. This lies outside the tabled values of 1.3558 and -2.508
Reject normal distribution.
Y value is -51.7565. This lies outside the tabled values of 1.3558 and -2.508

UCL (based on t-statistic) is 72.84

Lower Basin Neighborhood and Public Recreational Exposures to Surface Water

Manganese

Analytical Results (ug/L)

517	816	9450	2600
187	10700	30600	4850
210	879	20500	2450
455	2310	11700	2590
124	4750	25900	3220
240	1320	19300	4190
285	1120	27200	6360
2460	5310	9140	1110
4960	13100	17800	1480
11100	22500	12900	4580
7730	84900	4780	576
12100	20600	26900	2270
13300	32100	3600	3170
23400	27100	5400	1530
35300	18100	25100	4720
13500	19700	50500	9350
17300	11100	55800	6520
357	17600	40600	1280
715	27900	50300	5130
636	32000	14100	680
961	34200	10800	
505	36500	16600	
40900	30000	45600	
9400	19500	31400	
17000	17500	4660	
33700	21200	4330	
47300	7040	46700	
10000	14600	13000	
13600	26700	14600	
16400	21500	18100	
9430	16700	18900	
2420	11000	6310	
13500	24200	3860	
614	10900	19600	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	122	Mean	14678.17
Censored		Lognormal mean	21268.93
Detection limit or PQL		Std. devn.	14652.28
Method detection limit		Median	11050
TOTAL	122	Min.	124
		Max.	84900

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -2.3347. This lies within the tabled values of 1.3558 and -2.508

UCL (based on t-statistic) is 16877.6

Lower Basin Neighborhood and Public Recreational Exposures to Surface Water

Mercury

Analytical Results (ug/L)

0.23	0.12	2.2	1.3
0.1	0.97	0.93	10.9
0.1	1.2	3.9	1.2
0.35	0.245	1.2	2.6
0.1	0.55	3.2	7.8
0.1	1.7	5	2.8
0.1	0.26	4.7	2.8
0.6	0.23	3.3	0.91
0.34	0.98	1.3	1.4
0.71	3.6	3.9	0.53
0.24	13.5	9	1
1.1	43.9	1.7	3
11.1	3.6	6.9	0.13
0.38	7.1	1.2	0.12
4.9	4	1.2	0.29
2	1.6	3.9	0.76
0.46	0.97	5.5	0.21
0.1	1.9	4.5	0.53
0.1	1.4	5.4	0.22
0.1	1.9	4.9	0.14
0.1	3	1.7	0.05
0.1	2.7	0.4	0.12
14.1	11.3	2.3	
1	3.7	16.7	
0.91	3	5.7	
6.3	3.4	1.8	
18	3.9	2.2	
4.3	3.1	32.2	
4.1	2.5	4.1	
6.8	3.3	2.5	
3.7	8.2	5.4	
1.8	1.6	5	
0.84	17.3	3.6	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	121	Mean	3.62
Censored		Lognormal mean	4.51
Detection limit or PQL		Std. devn.	5.82803
Method detection limit		Median	1.8
TOTAL	121	Min.	0.05
		Max.	43.9

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -0.0322. This lies within the tabled values of 1.3534 and -2.51

UCL (based on t-statistic) is 4.5

Lower Basin Subsistence Sediment

Antimony

Analytical Results (mg/kg)

1.3	28	17	32	13	0.5
17.4	27.1	17.9	21.4	24.4	4.6
23.7	36.2	14.1	48.5	73.7	1.5
0.5	51.5	13.35	20.9	42.7	0.5
23	12.2	8.3	42.8	17.8	0.5
0.5	3.7	22.1	67.4	15.5	0.5
26.7	8.9	34.3	49.4	20.4	1
33.1	23.5	26.6	11.8	28.6	4.5
31.7	0.495	40	16.1	34.7	1.2
44.2	23.8	46.5	10.4	28.5	8
50.8	0.5	23.8	29.6	28.9	55.6
26.7	18.8	31	16.6	25.6	0.49
2	41.1	14.1	10.5	25.6	3.4
18.6	35.4	21	20.8	36.1	2.4
21.9	39.3	13	27.3	37.9	3.5
0.495	36.9	12.55	25.8	41.9	1.8
28.2	17.7	11.65	18.5	48.6	15.6
0.49	3.3	21	27.4	50	3.4
21.1	19.9	29.7	40.7	13.7	
35.5	16	17.3	25.3	0.5	
9	22.8	29.2	36	53	
36.9	13.8	40.3	44.2	29.1	
46	19.8	28.7	36.5	14.7	
31	27.3	15.3	51.2	12.8	
5.7	14.1	10.9	48.6	5.9	
16.1	29.8	17.5	0.5	25.4	
16.7	20.8	15.2	13.5	16.2	
0.495	21	13.4	11.9	35.4	
32.1	38.8	22.6	20.9	35.3	
5.5	15.1	28	16.1	27	
26.2	24.7	19.6	13.9	28.3	
36.1	4.5	16.4	19.7	46	
22	15.4	31.4	23.3	35.8	
44.5	27.5	52.4	34.3	24.5	
48.5	36.1	28.4	25.3	44.5	
25.8	13.5	34.7	30.5	38.8	
2.1	17	14.8	25.3	39.5	
14.3	39.9	21	49.1	24.3	
20.7	31.1	26.5	19.8	5.6	
0.495	34.7	24	51.8	24.6	
25.5	42.7	29.2	47	8.35	
0.5	26.3	30.2	42.5	15.1	
21.6	33	28.3	39.3	7.8	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	233	Mean	23.62
Censored		Lognormal mean	32.73
Detection limit or PQL		Std. devn.	14.59707309
Method detection limit		Median	23.3
TOTAL	233	Min.	0.49
		Max.	73.7

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -20.1835. This lies outside the tabled values of 1.5283 and -2.3626
Assume normal distribution.
Y value is 0.2467. This lies within the tabled values of 1.5283 and -2.3626

UCL (based on t-statistic) is 25.2

Lower Basin Subsistence Sediment

Arsenic

Analytical Results (mg/kg)

3.9	160	95.8	114	72.1	3
148	105	141	123	111	8.4
168	136	161	171	84.3	4.9
3.6	304	99.1	84.4	147	2.6
153	51.8	124	154	78.6	3.9
5.9	14.2	112	146	143	3.8
26.7	67.3	172	185	98.1	6.8
147	154	115	87.9	162	11.6
130	4	150	85.5	153	4.5
209	179	153	45.2	127	35
375	7	135	137	123	158
94.7	152	151	92.8	122	3.3
9.4	194	81.4	132	111	23.8
155	138	117	105	106	16.3
250	111	152	167	132	22.2
1.8	146	101	118	137	10.2
188	73.6	132	111	143	58
4.3	20	134	155	136	16.3
130	254	162	165	73.9	
153	122	136	138	6.3	
44.7	112	126	137	68.1	
148	128	151	111	154	
162	124	109	164	69	
112	164	96.7	137	150	
22.3	108	63.5	184	18	
123	141	120	9.5	146	
132	139	142	93.2	119	
1.5	139	85.8	46.1	122	
148	163	122	136	170	
68.9	85.2	180	79.7	115	
118	135	120	156	127	
165	19	133	113	144	
99.8	154	107	169	56	
173	166	208	143	102	
146	168	132	128	123	
94.1	128	182	129	108	
10.4	148	84.8	118	129	
133	178	110	145	88.3	
209	112	124	108	25.6	
2.4	143	159	113	73.4	
172	127	318	143	94.6	
17.9	126	137	126	87.8	
136	165	148	117	79.3	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	233	Mean	113.77
Censored		Lognormal mean	150.52
Detection limit or PQL		Std. devn.	59.3703804
Method detection limit		Median	124
TOTAL	233	Min.	1.5
		Max.	375

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -28.3113. This lies outside the tabled values of 1.5283 and -2.3626
Reject normal distribution.
Y value is -7.6397. This lies outside the tabled values of 1.5283 and -2.3626

UCL (based on t-statistic) is 120.22

Lower Basin Subsistence Sediment

Cadmium

Analytical Results (mg/kg)

0.6	26.2	13.3	47.3	34.4	0.29
19.1	54.1	18.2	49.1	67	2.4
29.6	43.8	14.2	100	25.3	1.4
0.28	51.7	33.3	56.6	59.2	0.24
29.7	32.4	18.1	96.7	30.9	0.39
1.9	2.6	42.4	104	19.4	0.1
26.7	13.3	49.1	80.9	22.8	1.1
35.4	33	73.6	27.7	35.8	1.5
55.1	0.1	59	36.2	36.5	0.55
67.4	26.7	87.6	12.3	40.1	4.5
30.1	1.9	52.9	52.3	33.9	14.8
41.2	28	50.6	18.2	40.9	0.34
1.6	45.7	9.3	20.9	50.6	3.2
24.3	54.4	26.4	24.9	79.6	1.6
28.2	51.4	16	35.4	70.1	2.7
0.1	49.8	37.1	30.5	83.1	1.2
37.3	29.1	35.2	50.6	95.6	7.4
0.38	4.8	38.5	46.7	88	2
41.9	18.5	39	70.5	32.8	
40.1	18.7	59.6	60.6	1	
19.9	20.4	64	88.9	4.7	
32.8	25.7	87.2	85.9	40.3	
56.1	31	56.5	94.3	24.3	
38.2	42.2	23.1	105	19.7	
2.8	22.9	8.8	84.1	3.6	
15.6	44.7	9.7	4.3	36.1	
28.4	26.8	16.9	24.8	28.3	
0.1	39.6	25.5	12.9	30.9	
37.7	35.7	36.4	25	44.2	
11.9	12.5	60.7	13.2	33.6	
35.7	20.7	36.3	21	52.5	
33	2.2	31.3	23.3	77.3	
44.1	20.2	58	40.7	88.6	
51.6	22.3	80.9	40.3	48	
74.6	43.8	54.2	55	77.4	
67.5	30.3	62.7	40.1	83.3	
1.9	36.1	4.8	39.2	81.7	
22.1	54.9	8.1	74.7	62.3	
34.3	75.4	27.4	57.5	3.3	
0.1	63.1	33.1	87.1	17.5	
33.5	94.4	28.9	83	16.5	
2.6	42.9	42.3	82.9	15.6	
32.1	30.4	55.4	79.6	25.3	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	233	Mean	36.49
Censored		Lognormal mean	64.72
Detection limit or PQL		Std. devn.	26.15683314
Method detection limit		Median	33.3
TOTAL	233	Min.	0.1
		Max.	105

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -19.7872. This lies outside the tabled values of 1.5283 and -2.3626
Assume normal distribution.
Y value is -0.5865. This lies within the tabled values of 1.5283 and -2.3626

UCL (based on t-statistic) is 39.33

Lower Basin Subsistence Sediment

Iron

Analytical Results (mg/kg)

11500	111000	66400	142000	90100	12900
82200	115000	70286	134000	115000	15500
126000	132000	83000	197000	124000	17800
7370	189000	115000	121000	117000	12500
129000	50500	102000	194000	58100	16700
17100	23900	124000	256000	70900	16100
26.7	55400	176000	190000	75600	16200
127000	144000	157000	66500	159000	23100
125000	10000	149000	87000	125000	14600
162000	104000	178000	58100	129000	27500
152000	16200	148000	126000	143000	54900
71800	155000	135000	64000	114000	11300
16200	174000	47000	79100	102000	20400
102000	117000	53300	88200	188000	18800
123000	139000	74200	168000	170000	20300
6080	132000	110000	83300	174000	17700
165000	75800	125000	87700	212000	35500
15900	18900	136000	163000	176000	17100
138000	102000	162000	183000	75400	
131000	70900	133000	138000	11400	
48000	109000	143000	182000	116000	
120000	84500	183000	180000	111000	
182000	128000	146000	186000	47700	
93700	153000	81100	222000	65500	
27400	136000	39100	185000	38700	
96500	149000	55200	13500	146000	
106000	118000	80100	72500	102000	
4450	142000	114000	73000	116000	
144000	179000	132000	85600	140000	
55200	53500	159000	62000	154000	
127000	62159	131000	71000	136000	
144000	17200	112000	92000	169000	
88100	90100	154000	143000	94500	
150000	112000	203000	141000	123000	
217000	172000	138000	107000	155000	
79600	121000	168000	146000	206000	
15400	109000	57000	140000	179000	
104000	190000	69000	163000	105000	
125000	165000	98700	86800	24800	
7870	145000	128000	189000	98100	
126000	180000	103000	160000	63700	
21700	132000	150000	190000	54400	
139000	137000	150000	184000	48200	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	233	Mean	107364.01
Censored		Lognormal mean	119522.30
Detection limit or PQL		Std. devn.	55679.2942
Method detection limit		Median	115000
TOTAL	233	Min.	4450
		Max.	256000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -13.1521. This lies outside the tabled values of 1.5283 and -2.3626
Reject normal distribution.
Y value is 1.6478. This lies outside the tabled values of 1.5283 and -2.3626

UCL (based on t-statistic) is 113411.85

Lower Basin Subsistence Sediment

Manganese

Analytical Results (mg/kg)

195	8040	6320	14600	7260	233
6900	11800	7300	14600	13000	779
10000	13300	5590	22000	4430	377
271	18600	9190	13400	10900	197
10300	4160	7860	20400	5000	259
386	592	13400	25500	5800	256
26.7	4650	17700	20400	6810	341
12500	13000	17600	4930	14100	805
13100	92.3	15600	6890	10100	373
16900	8200	19600	3780	9470	1810
13700	322	26400	11400	14600	4780
6270	14000	13500	6390	9000	243
348	16400	3940	6560	10800	875
8380	11700	4500	7860	21600	586
9560	15000	5670	14800	18300	797
154	13000	8890	6490	17700	432
14200	7280	9740	6650	20900	2820
138	1700	14800	16300	18600	728
13900	6900	16200	18700	5710	
13300	5430	13600	15200	112	
4270	8470	13200	20500	3540	
9470	9040	20300	18900	9840	
18000	13800	14000	19900	4100	
9140	14800	6340	22200	5660	
759	15000	2980	19700	1370	
7760	15800	4150	708	13000	
8940	11500	6060	5130	8190	
141	15200	8950	5210	9400	
14300	19200	13300	8080	11900	
4120	4830	17100	5490	15700	
9660	6680	12900	6300	13700	
14000	783	11700	8130	18300	
9310	6850	16000	11400	11000	
15200	8970	18200	14100	12700	
21000	16800	14400	8040	15800	
7340	9410	16500	14500	20400	
467	11100	3720	14200	18900	
8510	18900	5560	17800	8290	
9880	18600	8650	9810	1280	
97.5	15300	10100	20200	6140	
12500	20000	7740	16500	4500	
1160	14000	14400	19200	5450	
13600	12700	15700	19800	5010	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	233	Mean	10081.75
Censored		Lognormal mean	14904.65
Detection limit or PQL		Std. devn.	6305.62504
Method detection limit		Median	9660
TOTAL	233	Min.	92.3
		Max.	26400

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -18.8747. This lies outside the tabled values of 1.5283 and -2.3626
Reject normal distribution.
Y value is 2.3779. This lies outside the tabled values of 1.5283 and -2.3626

UCL (based on t-statistic) is 10766.66

Lower Basin Subsistence Sediment

Zinc

Analytical Results (mg/kg)

184	3630	1920	8290	5960	75.1
2750	8610	2250	8130	9850	560
4400	5780	2360	17200	7340	215
106	8690	6300	9490	9560	45.1
4510	2870	3010	17800	2790	70
421	522	6830	21800	2590	37.7
26.7	2090	8800	13000	3800	256
4960	5120	12800	3970	5610	389
9410	30.8	9320	5600	5430	90.9
9530	3150	13700	2550	7160	1380
4650	378	8030	8340	6280	4310
6680	5310	7170	1990	7100	69.3
452	6830	1400	2710	7120	757
3680	8830	2120	4270	13300	462
3650	8090	2430	5680	11900	772
64.7	7730	6500	4270	12100	306
5880	4400	6250	5390	19000	1960
195	1010	6300	8180	17900	465
7410	2990	7060	11500	5940	
5720	2220	7260	10500	235	
3090	4020	9260	15000	4900	
5300	3790	14200	12200	6140	
8780	4700	8540	13900	2310	
6330	7380	4060	20900	2510	
577	2630	1440	12900	890	
2330	7280	1890	589	5720	
4340	3850	2580	4210	4330	
14.3	6280	4450	2910	5020	
5530	5580	6570	3750	7810	
2550	1690	10200	1730	6380	
6490	2540	6000	2650	8760	
4610	562	4920	4030	12700	
6920	3180	9370	6390	14000	
8160	3260	12800	5750	8360	
11300	7920	8010	6650	11400	
7690	5270	10700	7030	16800	
587	5240	1440	7070	13300	
3440	9620	1700	11900	8260	
4900	12800	4700	6950	826	
19.5	9950	5310	17400	4230	
4870	15300	4180	12100	2840	
510	6720	7400	13800	1720	
6040	4400	9230	13200	2930	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	233	Mean	5914.22
Censored		Lognormal mean	9066.45
Detection limit or PQL		Std. devn.	4450.102
Method detection limit		Median	5310
TOTAL	233	Min.	14.3
		Max.	21800

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -16.8935. This lies outside the tabled values of 1.5283 and -2.3626
Reject normal distribution.
Y value is -4.0289. This lies outside the tabled values of 1.5283 and -2.3626

UCL (based on t-statistic) is 6397.59

Lower Basin Water Potatoes

Without Skin

Analytical Results
(mg/kg)

0.0885	0.0722	0.2562
0.1105	0.0685	0.123
0.0852	0.0955	0.3734
0.0318	0.0906	0.2592
0.1223	0.4544	0.536
0.2468	0.2375	1.1874
0.1565	0.4041	1.1145
0.2471	0.03505	1.1186
0.0377	0.2995	2.7978
0.1269	0.03675	0.0706
0.6009	0.2587	0.2855
0.4595	0.03	0.0303
0.3846	0.203	0.1437
0.5244	0.0906	0.0913
0.124	0.0971	0.03685
0.0934	0.0286	0.2089
0.0882	0.03415	0.3302
0.03195	0.2045	0.2204
0.029	0.03145	0.03035
0.03205	0.0371	0.0249
0.3013	0.115	0.071
0.3526	0.0789	0.2437
0.1885	0.1564	0.03735
0.1055	0.0812	0.1109
0.0325	0.2907	0.0262
0.1422	0.0844	0.1343
0.212	0.0802	0.027
0.146	0.03055	0.0868
0.2562	0.03245	0.0659
0.3102	0.0809	0.1228
0.0324	0.1082	1.1944

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	93	Mean	0.23
Censored		Lognormal mean	0.21
Detection limit or PQL		Std. devn.	0.362184
Method detection limit		Median	0.1109
TOTAL	93	Min.	0.0249
		Max.	2.7978

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -0.3477. This lies within the tabled values of 1.2785 and -2.5716

UCL (based on t-statistic) is 0.29

Lower Basin Water Potatoes

With Skin

Analytical Results
(mg/kg)

0.1221	0.03775	0.1287
0.2886	0.1209	0.1043
0.115	0.14	0.1626
0.0402	0.1254	0.2733
0.0998	0.173	0.5275
0.2676	0.2514	0.4397
0.2634	0.3386	2.0417
0.2219	0.1633	1.8434
0.5581	0.0311	1.6312
0.0909	0.1136	1.8525
0.7281	0.5083	3.7092
1.5347	0.4767	0.4399
0.6027	0.0366	1.2285
0.4686	0.1595	0.2375
0.4862	0.1685	0.3375
0.3893	0.6096	0.733
0.1487	0.2855	0.7985
0.1483	0.2327	0.2424
0.1255	0.1562	0.3476
0.2728	0.036	0.1985
0.1256	0.0391	0.102
0.5317	0.1019	0.1764
0.6455	0.1169	0.3421
0.2172	0.1762	0.6386
0.1138	0.1473	0.1864
0.0976	0.1897	0.2404
0.1749	0.3034	0.6601
0.1645	0.0977	0.4189
0.3375	0.0823	0.0675
0.294	0.0351	0.3152
0.5973	0.1307	0.0721
		0.1128
		0.9914

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	95	Mean	0.39
Censored		Lognormal mean	0.38
Detection limit or PQL		Std. devn.	0.531226
Method detection limit		Median	0.2219
TOTAL	95	Min.	0.0311
		Max.	3.7092

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -0.9548. This lies within the tabled values of 1.2855 and -2.566

UCL (based on t-statistic) is 0.48

Lower Basin Occupational Exposure to Soil/Sediment

Antimony

Analytical Results

(mg/kg)

2.9	0.49	16.1	29.8	17.5	0.5	25.4	18.7	15.6	22.4	23.9
3.7	0.52	16.7	20.8	15.2	13.5	16.2	22.4	13.7	20.5	58.6
5	0.21	0.5	21	13.4	11.9	35.4	17.7	10.4	21	19.9
5.7	0.65	32.1	38.8	22.6	20.9	35.3	23.3	6.6	14.7	22.2
1	0.51	5.5	15.1	28	16.1	27	34.7	7.9	20.4	2.5
2.4	0.43	26.2	24.7	19.6	13.9	28.3	27	26.2	18.4	0.5
4	0.21	36.1	4.5	16.4	19.7	46	27.9	25.7	19.4	3
0.1	0.24	22	15.4	31.4	23.3	35.8	20.2	21.1	26.6	7
2.4	0.32	44.5	27.5	52.4	34.3	24.5	0.5	21.5	14.5	1.4
2.9	0.35	48.5	36.1	28.4	25.3	44.5	4.3	20.9	11.5	16.8
4.4	0.5	25.8	13.5	34.7	30.5	38.8	2.1	16.6	20.9	21.7
1	0.5	2.1	17	14.8	25.3	39.5	0.48	54.6	19.9	17.9
27.4	2.2	14.3	39.9	21	49.1	24.3	1.3	16.3	35.8	13.1
29.5	1.6	20.7	31.1	26.5	19.8	5.6	30.3	22.3	49.9	13.1
35.1	0.5	0.5	34.7	24	51.8	24.6	35.8	24.7	30.5	24
12	1	25.5	42.7	29.2	47	8.35	30.1	1.2	35.7	25.1
0.5	3.8	0.5	26.3	30.2	42.5	15.1	37.9	0.5	20.4	26.8
5.7	0.6	21.6	33	28.3	39.3	7.8	32.3	0.5	30.4	24.4
9.8	1.3	28	17	32	13	0.5	0.5	1.8	46.9	22
16.2	17.4	27.1	17.9	21.4	24.4	4.6	2.4	0.5	36.9	15.9
19	23.7	36.2	14.1	48.5	73.7	1.5	3.3	19.4	18.7	
16.7	0.5	51.5	13.4	20.9	42.7	0.5	0.5	15.5	12.5	
10	23	12.2	8.3	42.8	17.8	0.5	14.2	14.2	56.5	
5.4	0.5	3.7	22.1	67.4	15.5	0.5	19.3	16.2	31.8	
21.1	26.7	8.9	34.3	49.4	20.4	1	22.7	18.1	37.4	
31.2	33.1	23.5	26.6	11.8	28.6	4.5	20.7	25.9	29.8	
22.2	31.7	0.5	40	16.1	34.7	1.2	15.9	17.7	41.5	
25.6	44.2	23.8	46.5	10.4	28.5	8	19.1	27.9	17.8	
1.7	50.8	0.5	23.8	29.6	28.9	55.6	5.7	17.8	34.9	
12.8	26.7	18.8	31	16.6	25.6	0.49	6	15.1	45.9	
13.8	2	41.1	14.1	10.5	25.6	3.4	13.9	33.3	19.7	
10.5	18.6	35.4	21	20.8	36.1	2.4	9	22.4	19.3	
1.7	21.9	39.3	13	27.3	37.9	3.5	11.7	20.4	17.9	
5.7	0.5	36.9	12.6	25.8	41.9	1.8	19.4	40.7	20.4	
6.8	28.2	17.7	11.7	18.5	48.6	15.6	18.9	42.2	17.5	
18.1	0.49	3.3	21	27.4	50	3.4	21.1	19.3	18.8	
0.21	21.1	19.9	29.7	40.7	13.7	8.9	21.2	14.6	17.4	
0.21	35.5	16	17.3	25.3	0.5	6.9	12	14.4	30.9	
0.21	9	22.8	29.2	36	53	12.7	28.2	16.5	10.4	
0.47	36.9	13.8	40.3	44.2	29.1	12.8	24.4	17.1	10.6	
0.54	46	19.8	28.7	36.5	14.7	11	21.6	17.5	10.8	
0.21	31	27.3	15.3	51.2	12.8	20.4	20.8	18.1	15	
0.21	5.7	14.1	10.9	48.6	5.9	21.6	21.3	23.5	36.1	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	450	Mean	19.98
Censored		Lognormal mean	31.28
Detection limit or PQL		Std. devn.	14.0724
Method detection limit		Median	19.35
TOTAL	450	Min.	0.1
		Max.	73.7

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -22.0133. This lies outside the tabled values of 1.652 and -2.2
Assume normal distribution.
Y value is -1.3214. This lies within the tabled values of 1.652 and -2.253

UCL (based on t-statistic) is 21.08

Lower Basin Occupational Exposure to Soil/Sediment

Arsenic

Analytical Results (mg/kg)

87.3	7.2	130	254	162	165	73.9	93.7	110	135	82.5
101	5.9	153	122	136	138	6.3	84.8	130	83.9	94.8
107	9	44.7	112	126	137	68.1	128	132	100	70
108	4.8	148	128	151	111	154	108	138	97.8	115
119	8.2	162	124	109	164	69	96.6	150	84.8	82.5
123	7.3	112	164	96.7	137	150	135	144	94.3	149
125	7.4	22.3	108	63.5	184	18	138	139	113	256
14.5	7.5	123	141	120	9.5	146	110	106	96.9	160
92.2	6.1	132	139	142	93.2	119	148	87.9	95	492
95.3	9.6	1.5	139	85.8	46.1	122	102	62	111	144
103	8	148	163	122	136	170	189	40.9	75.1	151
90.9	8.5	68.9	85.2	180	79.7	115	351	64.1	94.5	21
112	8.7	118	135	120	156	127	150	121	98.7	13.5
115	8.9	165	19	133	113	144	216	117	113	27.2
142	8.5	99.8	154	107	169	56	274	103	97.3	59.7
78.5	11.7	173	166	208	143	102	5.4	87.9	91.8	17.9
4.8	10.3	146	168	132	128	123	13.2	102	86.1	72.9
28.4	4.6	94.1	128	182	129	108	17.5	106	119	88.8
65.7	5.6	10.4	148	84.8	118	129	5.8	114	117	83.8
88.4	5.6	133	178	110	145	88.3	36.4	87.9	217	64.3
113	9.5	209	112	124	108	25.6	159	94.2	329	72
86.6	5.6	2.4	143	159	113	73.4	167	91.4	213	125
65.2	4.3	172	127	318	143	94.6	170	9.9	163	135
40.6	50.7	17.9	126	137	126	87.8	203	7.9	115	150
132	8.3	136	165	148	117	79.3	190	8.4	114	127
135	3.9	160	95.8	114	72.1	3	15.3	9.7	163	135
115	148	105	141	123	111	8.4	32.2	8.9	256	18.4
95.6	168	136	161	171	84.3	4.9	75.4	106	104	
11.5	3.6	304	99.1	84.4	147	2.6	17.5	139	75.1	
50.3	153	51.8	124	154	78.6	3.9	263	106	154	
59.2	5.9	14.2	112	146	143	3.8	124	103	107	
57.3	142	67.3	172	185	98.1	6.8	121	111	110	
16.9	147	154	115	87.9	162	11.6	116	112	103	
22.6	130	4	150	85.5	153	4.5	132	101	123	
68.2	209	179	153	45.2	127	35	128	151	116	
93.3	375	7	135	137	123	158	85.4	110	150	
9.9	94.7	152	151	92.8	122	3.3	79.9	111	187	
10.5	9.4	194	81.4	132	111	23.8	100	153	109	
9.6	155	138	117	105	106	16.3	95.2	121	99.9	
10.9	250	111	152	167	132	22.2	117	109	86.1	
13.6	1.8	146	101	118	137	10.2	114	219	100	
7.7	188	73.6	132	111	143	58	105	229	97.6	
7.8	4.3	20	134	155	136	16.3	167	96.5	105	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	457	Mean	104.41
Censored		Lognormal mean	133.99
Detection limit or PQL		Std. devn.	64.3352305
Method detection limit		Median	111
TOTAL	457	Min.	1.5
		Max.	492

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -26.2398. This lies outside the tabled values of 1.6542 and -2.251
Reject normal distribution.
Y value is -10.9881. This lies outside the tabled values of 1.6542 and -2.251

UCL (based on t-statistic) is 109.4

Lower Basin Occupational Exposure to Soil/Sediment

Cadmium

Analytical Results (mg/kg)

12.3	0.04	41.9	18.5	39	70.5	32.8	12.7	35	13.7	31.5
12.6	0.04	40.1	18.7	59.6	60.6	1	8.4	23.4	29.9	37
14.7	0.04	19.9	20.4	64	88.9	4.7	16.3	29.7	41.8	21.4
10.8	0.04	32.8	25.7	87.2	85.9	40.3	19.6	37	31	20.3
7.3	0.04	56.1	31	56.5	94.3	24.3	14.2	38.4	28.9	22
9.5	0.04	38.2	42.2	23.1	105	19.7	29.7	31.7	29.9	15.3
15.6	0.16	2.8	22.9	8.8	84.1	3.6	27.5	28.2	33.4	15.5
0.35	0.04	15.6	44.7	9.7	4.3	36.1	19.1	18.2	29.2	8.2
13	0.04	28.4	26.8	16.9	24.8	28.3	28.8	16.3	30.6	21.3
13.3	0.04	0.1	39.6	25.5	12.9	30.9	18	9.3	22.5	15
14.6	0.04	37.7	35.7	36.4	25	44.2	37.7	7.5	21.1	11.4
12.5	0.04	11.9	12.5	60.7	13.2	33.6	10.2	7.4	23.9	1.5
11.8	0.09	35.7	20.7	36.3	21	52.5	37.6	32.1	32.5	0.38
13.7	0.04	33	2.2	31.3	23.3	77.3	61.6	32.1	29.5	2.5
14.5	0.13	44.1	20.2	58	40.7	88.6	10.7	29.2	60.8	7.7
15.2	0.08	51.6	22.3	80.9	40.3	48	2.2	31.1	20.7	1.4
0.1	0.09	74.6	43.8	54.2	55	77.4	6.5	32.2	14.1	10.8
6.7	1.5	67.5	30.3	62.7	40.1	83.3	5.1	29.6	44.5	13.5
7.7	0.5	1.9	36.1	4.8	39.2	81.7	1.9	39.8	48.2	12.6
10.1	2	22.1	54.9	8.1	74.7	62.3	2.3	35.9	59.4	8.2
15.9	2.4	34.3	75.4	27.4	57.5	3.3	38.3	46.7	84.1	10
15.5	0.5	0.1	63.1	33.1	87.1	17.5	41.3	37.1	75.8	13.9
5.7	2.3	33.5	94.4	28.9	83	16.5	32.7	0.6	85.8	17.6
8.9	6.4	2.6	42.9	42.3	82.9	15.6	43.3	0.25	41.2	13.9
10	0.1	32.1	30.4	55.4	79.6	25.3	42.2	0.21	36.2	10.2
13.5	0.6	26.2	13.3	47.3	34.4	0.29	2.3	1	73.8	14
14.6	19.1	54.1	18.2	49.1	67	2.4	4.2	0.23	41.3	14.7
12	29.6	43.8	14.2	100	25.3	1.4	4.7	40.9	41.7	
6.1	0.28	51.7	33.3	56.6	59.2	0.24	3.6	31.6	26.7	
8.3	29.7	32.4	18.1	96.7	30.9	0.39	8.2	27.6	82.1	
9.4	1.9	2.6	42.4	104	19.4	0.1	26.6	36.4	53.3	
9.4	39.4	13.3	49.1	80.9	22.8	1.1	37	38.5	57.9	
1.3	35.4	33	73.6	27.7	35.8	1.5	30.5	47	29.1	
2.5	55.1	0.1	59	36.2	36.5	0.55	22.4	44.2	53.6	
3.3	67.4	26.7	87.6	12.3	40.1	4.5	42.2	52.8	19.8	
12.2	30.1	1.9	52.9	52.3	33.9	14.8	14.6	37.7	48.8	
0.04	41.2	28	50.6	18.2	40.9	0.34	17.8	35.7	86.4	
0.04	1.6	45.7	9.3	20.9	50.6	3.2	23.7	58.8	26.8	
0.04	24.3	54.4	26.4	24.9	79.6	1.6	22.8	38.1	24.9	
0.04	28.2	51.4	16	35.4	70.1	2.7	27.4	31.7	40.9	
0.04	0.1	49.8	37.1	30.5	83.1	1.2	18	54.2	32.3	
0.04	37.3	29.1	35.2	50.6	95.6	7.4	25.4	57.8	34.6	
0.04	0.38	4.8	38.5	46.7	88	2	25.9	27.9	38.5	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	457	Mean	29.00
Censored		Lognormal mean	80.50
Detection limit or PQL		Std. devn.	24.068877
Method detection limit		Median	26.6
TOTAL	457	Min.	0.04
		Max.	105

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -26.6363. This lies outside the tabled values of 1.6542 and -2.251
Reject normal distribution.
Y value is -4.997. This lies outside the tabled values of 1.6542 and -2.251

UCL (based on t-statistic) is 30.87

Lower Basin Occupational Exposure to Soil/Sediment

Iron

Analytical Results (mg/kg)

94300	20500	88100	112000	138000	146000	105000	132000	17200	167000
105000	19300	150000	172000	168000	140000	24800	118000	12700	144000
106000	19400	217000	121000	57000	163000	98100	142000	22200	106000
60800	21500	79600	109000	69000	86800	63700	133000	15700	74200
9710	12690	15400	190000	98700	189000	54400	25300	94800	188000
27500	11740	104000	165000	128000	160000	48200	29800	81800	131000
50800	14190	125000	145000	103000	190000	12900	43700	74900	136000
65000	16170	7870	180000	150000	184000	15500	22400	80600	104000
75800	13300	126000	132000	150000	90100	17800	80000	98700	135000
70100	12100	21700	137000	142000	115000	12500	95900	116000	81100
34200	27200	139000	66400	134000	124000	16700	123000	93200	131000
38600	15500	111000	70286	197000	117000	16100	114000	149000	184000
89600	11500	115000	83000	121000	58100	16200	94700	100000	82600
125000	82200	132000	115000	194000	70900	23100	115000	91400	75200
93000	126000	189000	102000	256000	75600	14600	75400	166000	63700
83100	7370	50500	124000	190000	159000	27500	82700	119000	85000
17600	129000	23900	176000	66500	125000	54900	79600	112000	70700
42000	17100	55400	157000	87000	129000	11300	93900	185000	79500
50100	135000	144000	149000	58100	143000	20400	112000	195000	67000
45700	127000	10000	178000	126000	114000	18800	110000	78900	94800
19700	125000	104000	148000	64000	102000	20300	95700	85300	62200
25900	162000	16200	135000	79100	188000	17700	108000	76300	86100
33600	152000	155000	47000	88200	170000	35500	116000	96300	72000
57500	71800	174000	53300	168000	174000	17100	105000	83500	100000
23100	16200	117000	74200	83300	212000	54800	112000	87500	139000
28600	102000	139000	110000	87700	176000	51200	126000	95000	89200
26000	123000	132000	125000	163000	75400	74000	126000	102000	145000
21900	6080	75800	136000	183000	11400	69700	126000	85800	80500
20000	165000	18900	162000	138000	116000	64900	124000	85400	80900
20100	15900	102000	133000	182000	111000	97700	72600	78400	38400
19300	138000	70900	143000	180000	47700	93500	81000	62800	31600
19100	131000	109000	183000	186000	65500	78300	60700	73500	31700
18000	48000	84500	146000	222000	38700	104000	53300	86600	29600
20000	120000	128000	81100	185000	146000	74300	68200	89900	31800
14900	182000	153000	39100	13500	102000	138000	119000	119000	46300
20600	93700	136000	55200	72500	116000	151000	110000	68300	62700
20100	27400	149000	80100	73000	140000	128000	103000	60500	54300
19900	96500	118000	114000	85600	154000	154000	97200	95300	38300
20000	106000	142000	132000	62000	136000	96700	103000	113000	45800
17000	4450	179000	159000	71000	169000	18100	79700	177000	76561
21300	144000	53500	131000	92000	94500	15900	79100	222000	91800
19200	55200	62159	112000	143000	123000	18600	79300	214000	85966
20400	127000	17200	154000	141000	155000	16700	117000	190000	87495
20300	144000	90100	203000	107000	206000	20900	82400	113000	83007
					179000	121000	19900	98600	17200

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	445	Mean	92916.73
Censored		Lognormal mean	100680.20
Detection limit or PQL		Std. devn.	53324.13
Method detection limit		Median	92000
TOTAL	445	Min.	4450
		Max.	256000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -8.5618. This lies outside the tabled values of 1.6501 and -2.2
Reject normal distribution.
Y value is 2.402. This lies outside the tabled values of 1.6501 and -2.254

UCL (based on t-statistic) is 97107.82

Lower Basin Occupational Exposure to Soil/Sediment

Manganese

Analytical Results (mg/kg)

6410	350	9310	8970	14400	14500	8290	12800	622	17100
7560	344	15200	16800	16500	14200	1280	9380	511	25200
7830	379	21000	9410	3720	17800	6140	13900	749	8400
4560	362	7340	11100	5560	9810	4500	13100	582	6150
260	533	467	18900	8650	20200	5450	1290	10900	19300
1410	354	8510	18600	10100	16500	5010	2110	9280	13300
3520	436	9880	15300	7740	19200	233	2880	7620	14400
3720	681	97.5	20000	14400	19800	779	1450	9250	8320
5660	391	12500	14000	15700	7260	377	4810	11000	14200
5300	535	1160	12700	14600	13000	197	10100	12700	8460
2360	1890	13600	6320	14600	4430	259	13100	10200	13900
2730	420	8040	7300	22000	10900	256	12100	16300	20000
5820	195	11800	5590	13400	5000	341	9500	11200	9390
8680	6900	13300	9190	20400	5800	805	12100	10000	7990
6620	10000	18600	7860	25500	6810	373	6870	16300	6680
6190	271	4160	13400	20400	14100	1810	7240	9490	9300
807	10300	592	17700	4930	10100	4780	8010	9030	7190
2900	386	4650	17600	6890	9470	243	9860	18400	8730
3500	9970	13000	15600	3780	14600	875	11000	19100	6790
3250	12500	92.3	19600	11400	9000	586	7900	6220	7120
725	13100	8200	26400	6390	10800	797	7570	5700	4810
1160	16900	322	13500	6560	21600	432	7840	8350	5940
1780	13700	14000	3940	7860	18300	2820	9020	11000	5440
3830	6270	16400	4500	14800	17700	728	7470	9590	6590
404	348	11700	5670	6490	20900	4370	8570	7520	10900
526	8380	15000	8890	6650	18600	3810	9660	7930	9180
451	9560	13000	9740	16300	5710	5510	9890	8250	12800
360	154	7280	14800	18700	112	5800	9700	7090	7810
400	14200	1700	16200	15200	3540	4930	9500	7180	7770
387	138	6900	13600	20500	9840	8470	5480	8290	1350
375	13900	5430	13200	18900	4100	8100	6050	5960	979
549	13300	8470	20300	19900	5660	6900	4530	7710	1410
531	4270	9040	14000	22200	1370	8940	3320	8780	1480
356	9470	13800	6340	19700	13000	6600	4330	8860	1150
283	18000	14800	2980	708	8190	10300	9020	12900	3860
367	9140	15000	4150	5130	9400	9370	8580	6780	5920
344	759	15800	6060	5210	11900	10200	8100	5490	4970
412	7760	11500	8950	8080	15700	11600	7920	10700	3220
365	8940	15200	13300	5490	13700	5740	8280	12000	3730
259	141	19200	17100	6300	18300	1020	8510	17700	5630
354	14300	4830	12900	8130	11000	780	8560	21100	6910
278	4120	6680	11700	11400	12700	1200	9240	21200	5750
306	9660	783	16000	14100	15800	739	12800	19600	7290
371	14000	6850	18200	8040	20400	881	9870	8900	7090
					18900	10000	722	8040	597

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	445	Mean	8449.09
Censored		Lognormal mean	11873.11
Detection limit or PQL		Std. devn.	5973.72812
Method detection limit		Median	8040
TOTAL	445	Min.	92.3
		Max.	26400

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -16.1585. This lies outside the tabled values of 1.6501 and -2.2547
Assume normal distribution.
Y value is 0.6709. This lies within the tabled values of 1.6501 and -2.2547

UCL (based on t-statistic) is 8918.6

Lower Basin Occupational Exposure to Soil/Sediment

Zinc

Analytical Results (mg/kg)

2330	58.7	7410	2990	7060	11500	5940	1800	6290	2280	4830
2590	54.3	5720	2220	7260	10500	235	1070	4460	4690	6370
2940	54.5	3090	4020	9260	15000	4900	2290	4630	6670	3750
2670	34.7	5300	3790	14200	12200	6140	2940	5600	4960	3590
2450	53.3	8780	4700	8540	13900	2310	1960	5910	4630	3840
2700	50.3	6330	7380	4060	20900	2510	4780	4990	4670	3030
3280	51.7	577	2630	1440	12900	890	4550	4330	5380	2520
311	54.8	2330	7280	1890	589	5720	3160	2370	4730	5060
2880	39.4	4340	3850	2580	4210	4330	4730	2600	4770	3530
2900	47.4	14.3	6280	4450	2910	5020	3050	1520	3340	3190
2940	45.2	5530	5580	6570	3750	7810	5070	1160	2990	1850
2430	48	2550	1690	10200	1730	6380	1580	1350	3360	267
2720	58	6490	2540	6000	2650	8760	5830	5810	5210	148
3440	51.9	4610	562	4920	4030	12700	6780	5910	4400	450
3530	49.7	6920	3180	9370	6390	14000	1120	5280	9630	1470
2420	138	8160	3260	12800	5750	8360	448	5600	2950	315
37.1	52.4	11300	7920	8010	6650	11400	800	5690	1890	1640
903	158	7690	5270	10700	7030	16800	772	4390	6850	1940
1250	80.3	587	5240	1440	7070	13300	370	4480	7520	1780
1570	217	3440	9620	1700	11900	8260	411	5570	10100	1320
2660	296	4900	12800	4700	6950	826	5360	7350	13600	1590
2500	56.9	19.5	9950	5310	17400	4230	5810	5420	12500	2070
1070	232	4870	15300	4180	12100	2840	4530	123	13600	2530
1270	937	510	6720	7400	13800	1720	6030	94.9	6700	1770
2750	82.3	6040	4400	9230	13200	2930	5900	65.7	5650	1610
4180	184	3630	1920	8290	5960	75.1	429	155	10900	1680
2840	2750	8610	2250	8130	9850	560	731	74.1	6710	1160
2900	4400	5780	2360	17200	7340	215	848	6600	6810	
462	106	8690	6300	9490	9560	45.1	649	4490	4580	
1210	4510	2870	3010	17800	2790	70	1370	3930	12100	
1500	421	522	6830	21800	2590	37.7	4510	5470	8300	
1420	6990	2090	8800	13000	3800	256	6150	5730	8910	
353	4960	5120	12800	3970	5610	389	5150	7910	4720	
549	9410	30.8	9320	5600	5430	90.9	3730	7240	8550	
641	9530	3150	13700	2550	7160	1380	6540	8710	2790	
1540	4650	378	8030	8340	6280	4310	2030	6240	7480	
55.2	6680	5310	7170	1990	7100	69.3	2700	5650	14200	
67.8	452	6830	1400	2710	7120	757	3880	9740	3720	
60	3680	8830	2120	4270	13300	462	3520	6930	3310	
54.2	3650	8090	2430	5680	11900	772	4660	5810	6320	
54.4	64.7	7730	6500	4270	12100	306	3720	9690	5240	
52.1	5880	4400	6250	5390	19000	1960	4520	10200	5310	
50.3	195	1010	6300	8180	17900	465	4650	4220	6910	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	457	Mean	4703.47
Censored		Lognormal mean	8110.80
Detection limit or PQL		Std. devn.	3987.5019
Method detection limit		Median	4230
TOTAL	457	Min.	14.3
		Max.	21800

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -17.4671. This lies outside the tabled values of 1.6542 and -2.25
Reject normal distribution.
Y value is -9.3486. This lies outside the tabled values of 1.6542 and -2.251

UCL (based on t-statistic) is 5012.73

Lower Basin Subsistence Surface Soil

Antimony

Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID
8.9	CUA033	28.2	CUA046	17.5	CUA055	36.1	CUA066
6.9	CUA033	24.4	CUA046	18.1	CUA055	23.9	CUA066
12.7	CUA033	21.6	CUA046	23.5	CUA055	58.6	CUA066
12.8	CUA033	20.8	CUA046	22.4	CUA055	19.9	CUA066
11	CUA033	21.3	CUA046	20.5	CUA055	22.2	CUA066
20.4	CUA035	15.6	CUA047	21	CUA056	2.5	CUA067
21.6	CUA035	13.7	CUA047	14.7	CUA056	0.5	CUA067
18.7	CUA035	10.4	CUA047	20.4	CUA056	3	CUA067
22.4	CUA035	6.6	CUA047	18.4	CUA056	7	CUA067
17.7	CUA035	7.9	CUA047	19.4	CUA056	1.4	CUA067
23.3	CUA036	26.2	CUA048	26.6	CUA057	16.8	CUA068
34.7	CUA036	25.7	CUA048	14.5	CUA057	21.7	CUA068
27	CUA036	21.1	CUA048	11.5	CUA057	17.9	CUA068
27.9	CUA036	21.5	CUA048	20.9	CUA057	13.1	CUA068
20.2	CUA036	20.9	CUA048	19.9	CUA057	13.1	CUA068
0.5	CUA038	16.6	CUA049	35.8	CUA058	24	CUA069
4.3	CUA038	54.6	CUA049	49.9	CUA058	25.1	CUA069
2.1	CUA038	16.3	CUA049	30.5	CUA058	26.8	CUA069
0.48	CUA038	22.3	CUA049	35.7	CUA058	24.4	CUA069
1.3	CUA038	24.7	CUA049	20.4	CUA058	22	CUA069
30.3	CUA039	1.2	CUA050	30.4	CUA059		
35.8	CUA039	0.495	CUA050	46.9	CUA059		
30.1	CUA039	0.5	CUA050	36.9	CUA059		
37.9	CUA039	1.8	CUA050	18.7	CUA059		
32.3	CUA039	0.5	CUA050	12.5	CUA059		
0.5	CUA041	19.4	CUA051	56.5	CUA060		
2.4	CUA041	15.5	CUA051	31.8	CUA060		
3.3	CUA041	14.2	CUA051	37.4	CUA060		
0.5	CUA041	16.2	CUA051	29.8	CUA060		
14.2	CUA041	18.1	CUA051	41.5	CUA060		
19.3	CUA043	25.9	CUA052	17.8	CUA063		
22.7	CUA043	17.7	CUA052	34.9	CUA063		
20.7	CUA043	27.9	CUA052	45.9	CUA063		
15.9	CUA043	17.8	CUA052	19.7	CUA063		
19.1	CUA043	15.1	CUA052	19.3	CUA063		
5.7	CUA044	33.3	CUA053	17.9	CUA064		
6	CUA044	22.4	CUA053	20.4	CUA064		
13.9	CUA044	20.4	CUA053	17.5	CUA064		
9	CUA044	40.7	CUA053	18.8	CUA064		
11.7	CUA044	42.2	CUA053	17.4	CUA064		
19.4	CUA045	19.3	CUA054	30.9	CUA065		
18.9	CUA045	14.6	CUA054	10.4	CUA065		
21.1	CUA045	14.4	CUA054	10.55	CUA065		
21.2	CUA045	16.5	CUA054	10.75	CUA065		
11.95	CUA045	17.1	CUA054	15	CUA065		

MTCA*Stat* 2.1

Number of samples	Uncensored values		
Uncensored	155	Mean	19.76
Censored		Lognormal mean	25.07
Detection limit or PQL		Std. devn.	11.58687
Method detection limit		Median	19.3
TOTAL	155	Min.	0.48
		Max.	58.6

Lognormal distribution? Normal distribution?

r-squared is: r-squared is:

Recommendations:

Reject lognormal distribution.

Y value is -19.4769. This lies outside the tabled values of 1.4303 and -2.4459

Reject normal distribution.

Y value is -4.5552. This lies outside the tabled values of 1.4303 and -2.4459

UCL (based on t-statistic) is 21.31

Lower Basin Subsistence Surface Soil

Arsenic

Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID
93.7	CUA033	132	CUA046	84.8	CUA055	256	CUA066
84.8	CUA033	138	CUA046	94.3	CUA055	160	CUA066
128	CUA033	150	CUA046	113	CUA055	492	CUA066
108	CUA033	144	CUA046	96.9	CUA055	144	CUA066
96.6	CUA033	139	CUA046	95	CUA055	151	CUA066
135	CUA035	106	CUA047	111	CUA056	21	CUA067
138	CUA035	87.9	CUA047	75.1	CUA056	13.5	CUA067
110	CUA035	62	CUA047	94.5	CUA056	27.2	CUA067
148	CUA035	40.9	CUA047	98.7	CUA056	59.7	CUA067
102	CUA035	64.1	CUA047	113	CUA056	17.9	CUA067
189	CUA036	121	CUA048	97.3	CUA057	72.9	CUA068
351	CUA036	117	CUA048	91.8	CUA057	88.8	CUA068
150	CUA036	103	CUA048	86.1	CUA057	83.8	CUA068
216	CUA036	87.9	CUA048	119	CUA057	64.3	CUA068
274	CUA036	102	CUA048	117	CUA057	72	CUA068
5.4	CUA038	106	CUA049	217	CUA058	125	CUA069
13.2	CUA038	114	CUA049	329	CUA058	135	CUA069
17.5	CUA038	87.9	CUA049	213	CUA058	150	CUA069
5.8	CUA038	94.2	CUA049	163	CUA058	127	CUA069
36.4	CUA038	91.4	CUA049	115	CUA058	135	CUA069
159	CUA039	9.9	CUA050	114	CUA059		
167	CUA039	7.9	CUA050	163	CUA059		
170	CUA039	8.4	CUA050	256	CUA059		
203	CUA039	9.7	CUA050	104	CUA059		
190	CUA039	8.9	CUA050	75.1	CUA059		
15.3	CUA041	106	CUA051	154	CUA060		
32.2	CUA041	139	CUA051	107	CUA060		
75.4	CUA041	106	CUA051	110	CUA060		
17.5	CUA041	103	CUA051	103	CUA060		
263	CUA041	111	CUA051	123	CUA060		
124	CUA043	112	CUA052	116	CUA063		
121	CUA043	101	CUA052	150	CUA063		
116	CUA043	151	CUA052	187	CUA063		
132	CUA043	110	CUA052	109	CUA063		
128	CUA043	111	CUA052	99.9	CUA063		
85.4	CUA044	153	CUA053	86.1	CUA064		
79.9	CUA044	121	CUA053	100	CUA064		
100	CUA044	109	CUA053	97.6	CUA064		
95.2	CUA044	219	CUA053	105	CUA064		
117	CUA044	229	CUA053	82.5	CUA064		
114	CUA045	96.5	CUA054	94.8	CUA065		
105	CUA045	135	CUA054	70	CUA065		
167	CUA045	83.9	CUA054	115	CUA065		
110	CUA045	100	CUA054	82.5	CUA065		
130	CUA045	97.8	CUA054	149	CUA065		

MTCA*Stat* 2.1

Number of samples	Uncensored values		
Uncensored	155	Mean	115.62
Censored		Lognormal mean	127.31
Detection limit or PQL		Std. devn.	65.60195
Method detection limit		Median	109
TOTAL	155	Min.	5.4
		Max.	492

Lognormal distribution? Normal distribution?

r-squared is: r-squared is:

Recommendations:

Reject lognormal distribution.

Y value is -19.0494. This lies outside the tabled values of 1.4303 and -2.4459

Reject normal distribution.

Y value is -14.74. This lies outside the tabled values of 1.4303 and -2.4459

UCL (based on t-statistic) is 124.35

Lower Basin Subsistence Surface Soil

Cadmium

Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID
12.7	CUA033	29.7	CUA046	28.9	CUA055	15.5	CUA066
8.4	CUA033	37	CUA046	29.9	CUA055	8.2	CUA066
16.3	CUA033	38.4	CUA046	33.4	CUA055	21.3	CUA066
19.6	CUA033	31.7	CUA046	29.2	CUA055	15	CUA066
14.2	CUA033	28.2	CUA046	30.6	CUA055	11.4	CUA066
29.7	CUA035	18.2	CUA047	22.5	CUA056	1.5	CUA067
27.5	CUA035	16.3	CUA047	21.1	CUA056	0.38	CUA067
19.1	CUA035	9.3	CUA047	23.9	CUA056	2.5	CUA067
28.8	CUA035	7.5	CUA047	32.5	CUA056	7.7	CUA067
18	CUA035	7.4	CUA047	29.5	CUA056	1.4	CUA067
37.7	CUA036	32.1	CUA048	60.8	CUA057	10.8	CUA068
10.2	CUA036	32.1	CUA048	20.7	CUA057	13.5	CUA068
37.6	CUA036	29.2	CUA048	14.1	CUA057	12.6	CUA068
61.6	CUA036	31.1	CUA048	44.5	CUA057	8.2	CUA068
10.7	CUA036	32.2	CUA048	48.2	CUA057	10	CUA068
2.2	CUA038	29.6	CUA049	59.4	CUA058	13.9	CUA069
6.5	CUA038	39.8	CUA049	84.1	CUA058	17.6	CUA069
5.1	CUA038	35.9	CUA049	75.8	CUA058	13.9	CUA069
1.9	CUA038	46.7	CUA049	85.8	CUA058	10.2	CUA069
2.3	CUA038	37.1	CUA049	41.2	CUA058	14	CUA069
38.3	CUA039	0.6	CUA050	36.2	CUA059		
41.3	CUA039	0.25	CUA050	73.8	CUA059		
32.7	CUA039	0.21	CUA050	41.3	CUA059		
43.3	CUA039	1	CUA050	41.7	CUA059		
42.2	CUA039	0.23	CUA050	26.7	CUA059		
2.3	CUA041	40.9	CUA051	82.1	CUA060		
4.2	CUA041	31.6	CUA051	53.3	CUA060		
4.7	CUA041	27.6	CUA051	57.9	CUA060		
3.6	CUA041	36.4	CUA051	29.1	CUA060		
8.2	CUA041	38.5	CUA051	53.6	CUA060		
26.6	CUA043	47	CUA052	19.8	CUA063		
37	CUA043	44.2	CUA052	48.8	CUA063		
30.5	CUA043	52.8	CUA052	86.4	CUA063		
22.4	CUA043	37.7	CUA052	26.8	CUA063		
42.2	CUA043	35.7	CUA052	24.9	CUA063		
14.6	CUA044	58.8	CUA053	40.9	CUA064		
17.8	CUA044	38.1	CUA053	32.3	CUA064		
23.7	CUA044	31.7	CUA053	34.6	CUA064		
22.8	CUA044	54.2	CUA053	38.5	CUA064		
27.4	CUA044	57.8	CUA053	31.5	CUA064		
18	CUA045	27.9	CUA054	37	CUA065		
25.4	CUA045	13.7	CUA054	21.4	CUA065		
25.9	CUA045	29.9	CUA054	20.3	CUA065		
35	CUA045	41.8	CUA054	22	CUA065		
23.4	CUA045	31	CUA054	15.3	CUA065		

MTCA*Stat* 2.1

Number of samples	Uncensored values		
Uncensored	155	Mean	28.09
Censored		Lognormal mean	37.20
Detection limit or PQL		Std. devn.	18.46822
Method detection limit		Median	28.2
TOTAL	155	Min.	0.21
		Max.	86.4

Lognormal distribution? Normal distribution?

r-squared is: r-squared is:

Recommendations:

Reject lognormal distribution.

Y value is -15.8548. This lies outside the tabled values of 1.4303 and -2.4459

Reject normal distribution.

Y value is -3.1882. This lies outside the tabled values of 1.4303 and -2.4459

UCL (based on t-statistic) is 30.55

Lower Basin Subsistence Surface Soil

Iron

Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID
54800	CUA033	112000	CUA046	87500	CUA055	139000	CUA066
51200	CUA033	126000	CUA046	95000	CUA055	89200	CUA066
74000	CUA033	126000	CUA046	102000	CUA055	145000	CUA066
69700	CUA033	126000	CUA046	85800	CUA055	80500	CUA066
64900	CUA033	124000	CUA046	85400	CUA055	80900	CUA066
97700	CUA035	72600	CUA047	78400	CUA056	38400	CUA067
93500	CUA035	81000	CUA047	62800	CUA056	31600	CUA067
78300	CUA035	60700	CUA047	73500	CUA056	31700	CUA067
104000	CUA035	53300	CUA047	86600	CUA056	29600	CUA067
74300	CUA035	68200	CUA047	89900	CUA056	31800	CUA067
138000	CUA036	119000	CUA048	119000	CUA057	46300	CUA068
151000	CUA036	110000	CUA048	68300	CUA057	62700	CUA068
128000	CUA036	103000	CUA048	60500	CUA057	54300	CUA068
154000	CUA036	97200	CUA048	95300	CUA057	38300	CUA068
96700	CUA036	103000	CUA048	113000	CUA057	45800	CUA068
18100	CUA038	79700	CUA049	177000	CUA058	76561	CUA069
15900	CUA038	79100	CUA049	222000	CUA058	91800	CUA069
18600	CUA038	79300	CUA049	214000	CUA058	85966	CUA069
16700	CUA038	117000	CUA049	190000	CUA058	87495	CUA069
20900	CUA038	82400	CUA049	113000	CUA058	83007	CUA069
121000	CUA039	19900	CUA050	98600	CUA059		
132000	CUA039	17200	CUA050	167000	CUA059		
118000	CUA039	12700	CUA050	144000	CUA059		
142000	CUA039	22200	CUA050	106000	CUA059		
133000	CUA039	15700	CUA050	74200	CUA059		
25300	CUA041	94800	CUA051	188000	CUA060		
29800	CUA041	81800	CUA051	131000	CUA060		
43700	CUA041	74900	CUA051	136000	CUA060		
22400	CUA041	80600	CUA051	104000	CUA060		
80000	CUA041	98700	CUA051	135000	CUA060		
95900	CUA043	116000	CUA052	81100	CUA063		
123000	CUA043	93200	CUA052	131000	CUA063		
114000	CUA043	149000	CUA052	184000	CUA063		
94700	CUA043	100000	CUA052	82600	CUA063		
115000	CUA043	91400	CUA052	75200	CUA063		
75400	CUA044	166000	CUA053	63700	CUA064		
82700	CUA044	119000	CUA053	85000	CUA064		
79600	CUA044	112000	CUA053	70700	CUA064		
93900	CUA044	185000	CUA053	79500	CUA064		
112000	CUA044	195000	CUA053	67000	CUA064		
110000	CUA045	78900	CUA054	94800	CUA065		
95700	CUA045	85300	CUA054	62200	CUA065		
108000	CUA045	76300	CUA054	86100	CUA065		
116000	CUA045	96300	CUA054	72000	CUA065		
105000	CUA045	83500	CUA054	100000	CUA065		

MTCA*Stat* 2.1

Number of samples	Uncensored values		
Uncensored	155	Mean	92135.03
Censored		Lognormal mean	95235.05
Detection limit or PQL		Std. devn.	41392.02
Method detection limit		Median	87500
TOTAL	155	Min.	12700
		Max.	222000

Lognormal distribution? Normal distribution?

r-squared is: r-squared is:

Recommendations:

Reject lognormal distribution.

Y value is -9.4591. This lies outside the tabled values of 1.4303 and -2.4459

Reject normal distribution.

Y value is -3.118. This lies outside the tabled values of 1.4303 and -2.4459

UCL (based on t-statistic) is 97647.36

Lower Basin Subsistence Surface Soil

Manganese

Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID
4370	CUA033	8570	CUA046	7520	CUA055	10900	CUA066
3810	CUA033	9660	CUA046	7930	CUA055	9180	CUA066
5510	CUA033	9890	CUA046	8250	CUA055	12800	CUA066
5800	CUA033	9700	CUA046	7090	CUA055	7810	CUA066
4930	CUA033	9500	CUA046	7180	CUA055	7770	CUA066
8470	CUA035	5480	CUA047	8290	CUA056	1350	CUA067
8100	CUA035	6050	CUA047	5960	CUA056	979	CUA067
6900	CUA035	4530	CUA047	7710	CUA056	1410	CUA067
8940	CUA035	3320	CUA047	8780	CUA056	1480	CUA067
6600	CUA035	4330	CUA047	8860	CUA056	1150	CUA067
10300	CUA036	9020	CUA048	12900	CUA057	3860	CUA068
9370	CUA036	8580	CUA048	6780	CUA057	5920	CUA068
10200	CUA036	8100	CUA048	5490	CUA057	4970	CUA068
11600	CUA036	7920	CUA048	10700	CUA057	3220	CUA068
5740	CUA036	8280	CUA048	12000	CUA057	3730	CUA068
1020	CUA038	8510	CUA049	17700	CUA058	5630	CUA069
780	CUA038	8560	CUA049	21100	CUA058	6910	CUA069
1200	CUA038	9240	CUA049	21200	CUA058	5750	CUA069
739	CUA038	12800	CUA049	19600	CUA058	7290	CUA069
881	CUA038	9870	CUA049	8900	CUA058	7090	CUA069
10000	CUA039	722	CUA050	8040	CUA059		
12800	CUA039	622	CUA050	17100	CUA059		
9380	CUA039	511	CUA050	25200	CUA059		
13900	CUA039	749	CUA050	8400	CUA059		
13100	CUA039	582	CUA050	6150	CUA059		
1290	CUA041	10900	CUA051	19300	CUA060		
2110	CUA041	9280	CUA051	13300	CUA060		
2880	CUA041	7620	CUA051	14400	CUA060		
1450	CUA041	9250	CUA051	8320	CUA060		
4810	CUA041	11000	CUA051	14200	CUA060		
10100	CUA043	12700	CUA052	8460	CUA063		
13100	CUA043	10200	CUA052	13900	CUA063		
12100	CUA043	16300	CUA052	20000	CUA063		
9500	CUA043	11200	CUA052	9390	CUA063		
12100	CUA043	10000	CUA052	7990	CUA063		
6870	CUA044	16300	CUA053	6680	CUA064		
7240	CUA044	9490	CUA053	9300	CUA064		
8010	CUA044	9030	CUA053	7190	CUA064		
9860	CUA044	18400	CUA053	8730	CUA064		
11000	CUA044	19100	CUA053	6790	CUA064		
7900	CUA045	6220	CUA054	7120	CUA065		
7570	CUA045	5700	CUA054	4810	CUA065		
7840	CUA045	8350	CUA054	5940	CUA065		
9020	CUA045	11000	CUA054	5440	CUA065		
7470	CUA045	9590	CUA054	6590	CUA065		

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	155	Mean	8382.35
Censored		Lognormal mean	9245.81
Detection limit or PQL		Std. devn.	4614.873
Method detection limit		Median	8250
TOTAL	155	Min.	511
		Max.	25200

Lognormal distribution? Normal distribution?

r-squared is: r-squared is:

Recommendations:

Reject lognormal distribution.

Y value is -13.5781. This lies outside the tabled values of 1.4303 and -2.4459

Reject normal distribution.

Y value is -5.2218. This lies outside the tabled values of 1.4303 and -2.4459

UCL (based on t-statistic) is 8996.93

Lower Basin Subsistence Surface Soil

Zinc

Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID	Analysis Value mg/kg	Site ID
1800	CUA033	4630	CUA046	4630	CUA055	2520	CUA066
1070	CUA033	5600	CUA046	4670	CUA055	5060	CUA066
2290	CUA033	5910	CUA046	5380	CUA055	3530	CUA066
2940	CUA033	4990	CUA046	4730	CUA055	3190	CUA066
1960	CUA033	4330	CUA046	4770	CUA055	1850	CUA066
4780	CUA035	2370	CUA047	3340	CUA056	267	CUA067
4550	CUA035	2600	CUA047	2990	CUA056	148	CUA067
3160	CUA035	1520	CUA047	3360	CUA056	450	CUA067
4730	CUA035	1160	CUA047	5210	CUA056	1470	CUA067
3050	CUA035	1350	CUA047	4400	CUA056	315	CUA067
5070	CUA036	5810	CUA048	9630	CUA057	1640	CUA068
1580	CUA036	5910	CUA048	2950	CUA057	1940	CUA068
5830	CUA036	5280	CUA048	1890	CUA057	1780	CUA068
6780	CUA036	5600	CUA048	6850	CUA057	1320	CUA068
1120	CUA036	5690	CUA048	7520	CUA057	1590	CUA068
448	CUA038	4390	CUA049	10100	CUA058	2070	CUA069
800	CUA038	4480	CUA049	13600	CUA058	2530	CUA069
772	CUA038	5570	CUA049	12500	CUA058	1770	CUA069
370	CUA038	7350	CUA049	13600	CUA058	1610	CUA069
411	CUA038	5420	CUA049	6700	CUA058	1680	CUA069
5360	CUA039	123	CUA050	5650	CUA059		
5810	CUA039	94.9	CUA050	10900	CUA059		
4530	CUA039	65.7	CUA050	6710	CUA059		
6030	CUA039	155	CUA050	6810	CUA059		
5900	CUA039	74.1	CUA050	4580	CUA059		
429	CUA041	6600	CUA051	12100	CUA060		
731	CUA041	4490	CUA051	8300	CUA060		
848	CUA041	3930	CUA051	8910	CUA060		
649	CUA041	5470	CUA051	4720	CUA060		
1370	CUA041	5730	CUA051	8550	CUA060		
4510	CUA043	7910	CUA052	2790	CUA063		
6150	CUA043	7240	CUA052	7480	CUA063		
5150	CUA043	8710	CUA052	14200	CUA063		
3730	CUA043	6240	CUA052	3720	CUA063		
6540	CUA043	5650	CUA052	3310	CUA063		
2030	CUA044	9740	CUA053	6320	CUA064		
2700	CUA044	6930	CUA053	5240	CUA064		
3880	CUA044	5810	CUA053	5310	CUA064		
3520	CUA044	9690	CUA053	6910	CUA064		
4660	CUA044	10200	CUA053	4830	CUA064		
3720	CUA045	4220	CUA054	6370	CUA065		
4520	CUA045	2280	CUA054	3750	CUA065		
4650	CUA045	4690	CUA054	3590	CUA065		
6290	CUA045	6670	CUA054	3840	CUA065		
4460	CUA045	4960	CUA054	3030	CUA065		

MTCA*Stat* 2.1

Number of samples	Uncensored values		
Uncensored	155	Mean	4469.55
Censored		Lognormal mean	5495.17
Detection limit or PQL		Std. devn.	2933.074
Method detection limit		Median	4530
TOTAL	155	Min.	65.7
		Max.	14200

Lognormal distribution?	Normal distribution?
r-squared is:	r-squared is:

Recommendations:

Reject lognormal distribution.

Y value is -12.3251. This lies outside the tabled values of 1.4303 and -2.445

Reject normal distribution.

Y value is -3.4414. This lies outside the tabled values of 1.4303 and -2.4459

UCL (based on t-statistic) is 4860.16

Lower Basin Subsistence Undisturbed Surface Water

Zinc

Analysis Value ug/L	Site ID	Analysis Value ug/L	Site ID	Analysis Value ug/L	Site ID
156	LC3	396	LC55	202	LC55
392	LC3	290	LC60	320	LC55
454	LC55	262	LC55	334	LC60
444	LC60	212	LC60	309	LC60
592	LC60	209	LC55	369	LC55
613	LC55	222	LC60	281	LC60
494	LC60	179	LC55	440	LC55
489	LC55	500	LC55	521	LC55
382	LC60	420	LC55	345	LC60
434	LC55	420	LC55	627	LC55
436	LC60	690	LC55	500	LC60
328	LC55	180	LC55	363	LC55
238	LC60	140	LC55	410	LC60
247	LC55	120	LC55	207	LC55
244	LC60	570	LC60	206	LC60
196	LC55	600	LC60	237	LC55
300	LC55	380	LC60	258	LC60
281	LC60	210	LC60	368	LC55
278	LC55	300	LC60	369	LC60
401	LC60	120	LC60	265	LC55
368	LC55	240	LC60	280	LC60
428	LC60	140	LC60	431	LC2918
513	LC55	237	LC60	512	LC2919
454	LC60	312	LC60	410	LC2920
518	LC55	340	LC60	589	LC2921
373	LC60	126	LC55	6	LC8257
516	LC55	239	LC55	1.5	LC8261
441	LC60	313	LC55	1.5	LC8262
336	LC55	395	LC55		
391	LC60	222	LC60		
476	LC55	179	LC55		
466	LC60	234	LC60		

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	92	Mean	339.54
Censored		Lognormal mean	444.28
Detection limit or PQL		Std. devn.	144.488576
Method detection limit		Median	338
TOTAL	92	Min.	1.5
		Max.	690

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -28.8082. This lies outside the tabled values of 1.275 and -2.5744
Assume normal distribution.
Y value is 0.3149. This lies within the tabled values of 1.275 and -2.5744

UCL (based on t-statistic) is 364.61

Lower Basin Subsistence Undisturbed Surface Water

Manganese

Analysis Value ug/L	Site ID
46.3	LC3
122	LC3
210	LC55
380	LC60
6	LC8257
1	LC8261
1	LC8262

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	7	Mean	109.47
Censored		Lognormal mean	506.19
Detection limit or PQL		Std. devn.	142.406539
Method detection limit		Median	46.3
TOTAL	7	Min.	1
		Max.	380

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8804. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 214.05

Lower Basis Subsistence Undisturbed Surface Water

Iron

Analysis Value ug/L	Site ID
82.5	LC3
174	LC3
2200	LC55
3700	LC60
18	LC8257
6	LC8261
6	LC8262

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	7	Mean	883.79
Censored		Lognormal mean	3103.22
Detection limit or PQL		Std. devn.	1477.60718
Method detection limit		Median	82.5
TOTAL	7	Min.	6
		Max.	3700

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8958. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 1968.92

Lower Basin Subsistence Undisturbed Surface Water

Arsenic

Analysis Value ug/L	Site ID
0.5	LC3
0.335	LC3
7	LC2918
20	LC2919
17	LC2920
16	LC2921
14.5	LC8257
14.5	LC8261
14.5	LC8262

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	9	Mean	11.59
Censored		Lognormal mean	22.97
Detection limit or PQL		Std. devn.	7.20745929
Method detection limit		Median	14.5
TOTAL	9	Min.	0.335
		Max.	20

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.6796. This is less than the tabled value of 0.829
Assume normal distribution.
W value is 0.8423. This exceeds the tabled value of 0.829

UCL (based on t-statistic) is 16.06

Lower Basin Subsistence Undisturbed Surface Water

Cadmium

Analysis Value ug/L	Site ID	Analysis Value ug/L	Site ID	Analysis Value ug/L	Site ID
1.1	LC3	2.4	LC55	3	LC60
1.9	LC3	1.2	LC55	2	LC60
2.3	LC55	1.6	LC55	2	LC60
3.3	LC55	2.1	LC55	2	LC60
4.5	LC55	2.2	LC55	1	LC60
2.4	LC55	2.2	LC55	2	LC60
2.9	LC55	3	LC55	1	LC60
1.7	LC55	2.9	LC55	1.6	LC60
1	LC55	2.7	LC55	2	LC60
1.6	LC55	1.5	LC55	2.1	LC60
1.4	LC55	1.4	LC55	1.3	LC60
1.8	LC55	2.4	LC55	1.5	LC60
2.3	LC55	2	LC55	2.4	LC60
2.6	LC55	1.9	LC60	1.8	LC60
2.6	LC55	3.6	LC60	1.4	LC60
1.8	LC55	4.8	LC60	1.6	LC60
2.3	LC55	2.1	LC60	2.4	LC60
2.7	LC55	1.9	LC60	2.4	LC60
1.6	LC55	1.5	LC60	2.1	LC60
1.3	LC55	1.4	LC60	2.2	LC60
1.2	LC55	1.3	LC60	2.3	LC60
3	LC55	2	LC60	2.2	LC60
3	LC55	2.1	LC60	6	LC2918
3	LC55	2.2	LC60	21	LC2919
3	LC55	2.1	LC60	11	LC2920
1	LC55	2.2	LC60	17	LC2921
1	LC55	2	LC60	10	LC8257
1	LC55	2.6	LC60	4	LC8261
2	LC55	1.6	LC60	1.5	LC8262
0.89	LC55	1.3	LC60		
1.5	LC55	1.3	LC60		
2.1	LC55	3	LC60		

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	93	Mean	2.66
Censored		Lognormal mean	2.49
Detection limit or PQL		Std. devn.	2.8688416
Method detection limit		Median	2.1
TOTAL	93	Min.	0.89
		Max.	21

Lognormal distribution? Normal distribution?

r-squared is: r-squared is:

Recommendations:

Reject lognormal distribution.

Y value is -10.4105. This lies outside the tabled values of 1.2785 and -2.5716

Reject normal distribution.

Y value is -38.3913. This lies outside the tabled values of 1.2785 and -2.5716

UCL (based on t-statistic) is 3.15

Lower Basin Subsistence Undisturbed Surface Water

Lead

Analysis Value ug/L	Site ID	Analysis Value ug/L	Site ID	Analysis Value ug/L	Site ID
27.1	LC3	9	LC55	8	LC55
17.8	LC3	26	LC55	41	LC60
10	LC55	25	LC55	9	LC55
28	LC60	14	LC55	24	LC60
30	LC60	49	LC55	9	LC55
10	LC55	67	LC55	17	LC60
26	LC60	11	LC55	10	LC55
21	LC55	230	LC55	42	LC60
40	LC60	27	LC60	7	LC2918
11	LC55	27	LC60	12	LC2919
36	LC60	22	LC60	12	LC2920
46	LC55	110	LC60	13	LC2921
40	LC60	430	LC60	7.5	LC8257
24	LC55	22	LC60	7.5	LC8261
25	LC60	300	LC60	2	LC8262
8	LC55	32	LC60		
303	LC55	27.2	LC60		
31	LC60	30.4	LC60		
17	LC55	17.9	LC60		
83	LC60	36.1	LC55		
20	LC55	8.5	LC55		
32	LC60	24.4	LC55		
23	LC55	18	LC55		
25	LC60	30	LC60		
23	LC55	42	LC55		
30	LC60	21	LC60		
10	LC55	64	LC55		
18	LC60	11	LC55		
9	LC55	20	LC60		
56	LC60	47	LC60		
40	LC55	26	LC55		
27	LC60	19	LC60		
10	LC55	21	LC55		
16	LC60	8	LC55		
9	LC55	15	LC60		
44	LC60	12	LC55		
2.5	LC55	21	LC60		
30	LC60	28	LC55		
42	LC55	60	LC60		

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	93	Mean	37.96
Censored		Lognormal mean	33.87
Detection limit or PQL		Std. devn.	63.5183686
Method detection limit		Median	24
TOTAL	93	Min.	2
		Max.	430

Lognormal distribution? Normal distribution?

r-squared is: r-squared is:

Recommendations:

Reject lognormal distribution.

Y value is -5.5719. This lies outside the tabled values of 1.2785 and -2.5716

Reject normal distribution.

Y value is -39.2845. This lies outside the tabled values of 1.2785 and -2.5716

Lower Basin Subsistence Undisturbed Surface Water

Mercury

Analysis Value ug/L	Site ID
0.1	LC3
0.05	LC3
2.5	LC8257
2.5	LC8261
2.5	LC8262

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	1.53
Censored		Lognormal mean	4.17
Detection limit or PQL		Std. devn.	1.32834483
Method detection limit		Median	2.5
TOTAL	5	Min.	0.05
		Max.	2.5

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.7358. This is less than the tabled value of 0.762
Reject normal distribution.
W value is 0.6904. This is less than the tabled value of 0.762

UCL (based on t-statistic) is 2.8

Kingston Residential Surface Soil
Antimony

Analytical Results (mg/kg)

0.35	1.7	1.6
0.74	1.05	0.77
0.84	0.87	1.4
0.54	1.2	0.44
0.56	0.8	
0.85	0.65	
0.495	2.8	
0.5	0.495	
1.05	1.05	
1.55	17.1	
3.2	1.3	
1.4	2.9	
10.9	2.6	
25.8	1.15	
1.2	1	
2.65	21.4	
0.85	0.73	
0.85	1.7	
0.65	3.1	
0.56	1	
6.1	1.1	
1.25	2.3	
1.9	1	
1.25	4.4	
1.75	1.9	
2.8	2.9	
11.6	2.5	
8.2	7.8	
1.4	1.1	
2	3.4	
9.4	0.6	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	66	Mean	3.05
Censored		Lognormal mean	2.68
Detection limit or PQL		Std. devn.	4.76739
Method detection limit		Median	1.275
TOTAL	66	Min.	0.35
		Max.	25.8

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -2.8994. This lies outside the tabled values of 1.1516 and -2.6708
Reject normal distribution.
Y value is -24.8558. This lies outside the tabled values of 1.1516 and -2.6708

UCL (based on t-statistic) is 4.03

Kingston Residential Surface Soil
Arsenic

Analytical Results (mg/kg)

25.6	10.3	28.4
25.5	15.5	16.6
35.4	14.7	15.8
11.7	10.5	66.7
12.1	19.1	8.1
11.9	103	12.3
12	27.2	8.6
10.3	8.1	7.2
15.6	10.2	10.9
6.6	11.4	
9.5	10.2	
10.7	8.2	
12.4	12.6	
105	84	
78.4	10.7	
13.2	10.2	
13.9	22.3	
9.3	19.7	
9.2	9.5	
7.1	95.2	
15.8	12.2	
34.6	9.9	
15.4	12.9	
7.8	14.7	
11.6	14.9	
20.6	12	
21.8	8.1	
14.6	5.1	
32.7	12.5	
14.2	7.2	
8.2	15.3	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	71	Mean	20.35
Censored		Lognormal mean	18.88
Detection limit or PQL		Std. devn.	22.25628
Method detection limit		Median	12.4
TOTAL	71	Min.	5.1
		Max.	105

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -7.2413. This lies outside the tabled values of 1.181 and -2.6481
Reject normal distribution.
Y value is -24.0776. This lies outside the tabled values of 1.181 and -2.6481

UCL (based on t-statistic) is 24.76

Kingston Residential Surface Soil
Cadmium

Analytical Results (mg/kg)

4.8	3.9	4.1
4.6	0.58	2.5
5.6	13.9	2.5
2.2	0.55	3
3.2	3.3	0.47
1.5	0.77	1.3
0.1	0.13	0.75
0.34	1.9	1.7
0.4	1.3	0.08
1.2	0.31	
2.5	9.2	
3.7	0.72	
3.4	0.52	
2.8	3	
4.8	0.41	
2.1	0.44	
2.7	2.4	
2.5	1.4	
3.2	3.9	
1.8	26	
2.6	4.3	
3.1	0.96	
2.8	5.8	
1.8	7.6	
2	8.6	
6.4	8.8	
5	1	
0.86	0.5	
5.2	2.9	
6.8	1.9	
1.9	2.2	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	71	Mean	3.20
Censored		Lognormal mean	3.59
Detection limit or PQL		Std. devn.	3.738305
Method detection limit		Median	2.5
TOTAL	71	Min.	0.08
		Max.	26

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -2.1395. This lies within the tabled values of 1.181 and -2.6481

UCL (based on t-statistic) is 3.94

Kingston Residential Surface Soil
Iron

Analytical Results (mg/kg)	
21500	14800
20500	17500
20600	16600
13800	32800
17100	17100
18700	15400
18400	16900
16900	15500
17700	96300
17400	16900
17100	17000
17900	15500
14100	17000
16800	14900
21300	15700
20300	14600
19200	17200
21500	11600
16200	12410
16000	12930
15800	14350
35100	18500
17800	14410
17100	17000
19700	22800
23100	17500
18800	15800
13900	15500
18400	13150
34200	17300

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	60	Mean	19197.50
Censored		Lognormal mean	18815.68
Detection limit or PQL		Std. devn.	11061.73
Method detection limit		Median	17100
TOTAL	60	Min.	11600
		Max.	96300

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -16.4287. This lies outside the tabled values of 1.115 and -2.699
Reject normal distribution.
Y value is -34.1676. This lies outside the tabled values of 1.115 and -2.699

UCL (based on t-statistic) is 21584.72

Kingston Residential Surface Soil
Manganese

Analytical Results (mg/kg)

638	530
774	492
739	1440
505	626
629	473
891	522
611	431
527	742
445	9790
477	850
406	616
624	595
594	807
533	423
758	892
680	441
635	343
1010	446
1040	350
485	759
634	751
625	643
1130	716
575	681
710	596
718	816
471	583
468	523
564	232
505	
478	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	60	Mean	783.13
Censored		Lognormal mean	704.49
Detection limit or PQL		Std. devn.	1199.609
Method detection limit		Median	613.5
TOTAL	60	Min.	232
		Max.	9790

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -15.6886. This lies outside the tabled values of 1.115 and -2.699
Reject normal distribution.
Y value is -45.4684. This lies outside the tabled values of 1.115 and -2.699

UCL (based on t-statistic) is 1042.02

Kingston Residential Surface Soil
Zinc

Analytical Results (mg/kg)

526	170	423
842	226	252
1060	1850	320
406	93.1	611
1190	598	126
166	745	167
74.3	95.8	131
258	145	217
81.8	418	49.6
259	1340	
333	452	
633	506	
273	128	
916	937	
455	129	
264	107	
282	407	
240	275	
250	415	
174	8150	
320	241	
543	257	
460	188	
246	536	
287	246	
698	270	
509	174	
122	78.7	
946	202	
900	175	
133	239	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	71	Mean	506.16
Censored		Lognormal mean	448.95
Detection limit or PQL		Std. devn.	978.7587
Method detection limit		Median	270
TOTAL	71	Min.	49.6
		Max.	8150

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -2.331. This lies within the tabled values of 1.181 and -2.6481

UCL (based on t-statistic) is 700.01

Kingston Residential Tap Water

Arsenic

Analytical Results

(ug/L)
0.23
0.31
0.1
0.125
0.19
0.1
0.405

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	7	Mean	0.21
Censored		Lognormal mean	0.21
Detection limit or PQL		Std. devn.	0.11564
Method detection limit		Median	0.19
TOTAL	7	Min.	0.1
		Max.	0.405

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9196. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 0.29

Site ID	Static Result	Purged Result	Average Result
R015	0.36	0.1 U	0.23
R016	0.42	0.2	0.31
R018	0.1 UJ	0.1 UJ	0.1
R019	0.1 UJ	0.15 U	0.125
R063	0.1 U	0.28	0.19
R070	0.1 UJ	0.1 UJ	0.1
R089	0.1 U	0.71	0.405

Kingston Neighborhood Exposure to Sediments

Antimony

Analytical Results (mg/kg)
1.01
0.92
0.93
1.01
0.963
0.4745
0.49
0.51
19.6
29
31.8
1.02
3.97

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	13	Mean	7.05
Censored		Lognormal mean	6.78
Detection limit or PQL		Std. devn.	11.58952
Method detection limit		Median	1.01
TOTAL	13	Min.	0.4745
		Max.	31.8

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.7655. This is less than the tabled value of 0.866
Reject normal distribution.
W value is 0.6119. This is less than the tabled value of 0.866

UCL (based on t-statistic) is 12.78

Kingston Neighborhood Exposure to Sediments

Arsenic

Analytical Results (mg/kg)
5.98
2.79
2.95
10.8
2.95
13.4
8.34
16.6
60.6
11.1
34.4
14
15.5

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	13	Mean	15.34
Censored		Lognormal mean	15.84
Detection limit or PQL		Std. devn.	15.96519
Method detection limit		Median	11.1
TOTAL	13	Min.	2.79
		Max.	60.6

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9385. This exceeds the tabled value of 0.866

UCL (based on t-statistic) is 23.23

Kingston Neighborhood Exposure to Sediments

Cadmium

Analytical Results (mg/kg)
0.912
0.82
0.55
1.25
0.581
0.809
0.738
1.14
0.417
1.02
0.959
0.378
0.982

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	13	Mean	0.81
Censored		Lognormal mean	0.82
Detection limit or PQL		Std. devn.	0.270052
Method detection limit		Median	0.82
TOTAL	13	Min.	0.378
		Max.	1.25

Lognormal distribution?	Normal distribution?
r-squared is:	r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9253. This exceeds the tabled value of 0.866

UCL (based on t-statistic) is 0.95

Kingston Neighborhood Exposure to Sediments

Iron

Analytical Results (mg/kg)
15600
9170
12300
13600
16000
17100
17600
17900
14400
10100
24200
8200
13100

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	13	Mean	14559.23
Censored		Lognormal mean	14622.35
Detection limit or PQL		Std. devn.	4286.736
Method detection limit		Median	14400
TOTAL	13	Min.	8200
		Max.	24200

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9702. This exceeds the tabled value of 0.866

UCL (based on t-statistic) is 16677.9

Kingston Neighborhood Exposure to Sediments

Manganese

Analytical Results (mg/kg)
420
739
223
338
355
316
271
438
319
604
424
162
449

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	13	Mean	389.08
Censored		Lognormal mean	392.21
Detection limit or PQL		Std. devn.	153.8145
Method detection limit		Median	355
TOTAL	13	Min.	162
		Max.	739

Lognormal distribution?	Normal distribution?
r-squared is:	r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9779. This exceeds the tabled value of 0.866

UCL (based on t-statistic) is 465.1

Kingston Neighborhood Exposure to Sediments

Zinc

Analytical Results (mg/kg)
320
235
211
393
237
262
310
404
182
187
452
166
341

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	13	Mean	284.62
Censored		Lognormal mean	285.89
Detection limit or PQL		Std. devn.	93.04169
Method detection limit		Median	262
TOTAL	13	Min.	166
		Max.	452

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.953. This exceeds the tabled value of 0.866

UCL (based on t-statistic) is 330.6

Kingston Neighborhood Exposure to Surface Water

Arsenic

Analytical Results
(ug/L)

1
0.5
0.08
1
1
0.08
1
0.39
0.15
1
0.5
0.115

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	12	Mean	0.57
Censored		Lognormal mean	0.66
Detection limit or PQL		Std. devn.	0.407907
Method detection limit		Median	0.5
TOTAL	12	Min.	0.08
		Max.	1

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8116. This is less than the tabled value of 0.859
Reject normal distribution.
W value is 0.7977. This is less than the tabled value of 0.859

UCL (based on t-statistic) is 0.78

Kingston Neighborhood Exposure to Surface Water

Cadmium

Analytical Results
(ug/L)

3.7
1.2
0.0345
0.1
0.2
0.0345
0.1
0.29
0.26
0.5
0.32
0.25

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	12	Mean	0.58
Censored		Lognormal mean	0.58
Detection limit or PQL		Std. devn.	1.030819
Method detection limit		Median	0.255
TOTAL	12	Min.	0.0345
		Max.	3.7

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9488. This exceeds the tabled value of 0.859

UCL (based on t-statistic) is 1.12

Kingston Neighborhood Exposure to Surface Water

Manganese

Analytical Results
(ug/L)

144
18.1
2.3
2.5
2.5
4.3
8
3.7
3
2.5
2.5
1.7

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	12	Mean	16.26
Censored		Lognormal mean	10.51
Detection limit or PQL		Std. devn.	40.48565
Method detection limit		Median	2.75
TOTAL	12	Min.	1.7
		Max.	144

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.7187. This is less than the tabled value of 0.859
Reject normal distribution.
W value is 0.3994. This is less than the tabled value of 0.859

UCL (based on t-statistic) is 37.25

Kingston Neighborhood Exposure to Surface Water

Mercury

Analytical Results
(ug/L)

0.05
0.1
0.05
0.1
0.1
0.05
0.1
0.05
0.05
0.1
0.1
0.08
all nondetects

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	12	Mean	0.08
Censored		Lognormal mean	0.08
Detection limit or PQL		Std. devn.	0.024909
Method detection limit		Median	0.09
TOTAL	12	Min.	0.05
		Max.	0.1

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.6894. This is less than the tabled value of 0.859
Reject normal distribution.
W value is 0.6886. This is less than the tabled value of 0.859

UCL (based on t-statistic) is 0.09

Kingston Neighborhood and Public Recreational Exposures to Soil/Sediment

Antimony

Analytical Results
(mg/kg)

33.3
16.5
26.7
21.8
23.9
64.3
53.9
46.6
31.9
44.9
45.1
49.5
36.7
36.1
26.6
23.2
28.7
24.8
17.8

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	19	Mean	34.33
Censored		Lognormal mean	34.47
Detection limit or PQL		Std. devn.	13.17953
Method detection limit		Median	31.9
TOTAL	19	Min.	16.5
		Max.	64.3

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.975. This exceeds the tabled value of 0.901

UCL (based on t-statistic) is 39.57

Kingston Neighborhood and Public Recreational Exposures to Soil/Sediment

Arsenic

Analytical Results
(mg/kg)

128
73
151
133
155
266
209
134
136
166
174
209
143
140
107
113
123
104
93.9

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	19	Mean	145.15
Censored		Lognormal mean	145.44
Detection limit or PQL		Std. devn.	45.54776
Method detection limit		Median	136
TOTAL	19	Min.	73
		Max.	266

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9848. This exceeds the tabled value of 0.901

UCL (based on t-statistic) is 163.27

Kingston Neighborhood and Public Recreational Exposures to Soil/Sediment

Cadmium

Analytical Results
(mg/kg)

18
13.9
15.2
14.6
17.2
63.3
29.6
23
13.7
18.6
31.6
23
18.2
18.4
23.5
13.6
21.4
22.7
19

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	19	Mean	22.03
Censored		Lognormal mean	21.81
Detection limit or PQL		Std. devn.	11.18967
Method detection limit		Median	18.6
TOTAL	19	Min.	13.6
		Max.	63.3

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8532. This is less than the tabled value of 0.901
Reject normal distribution.
W value is 0.6476. This is less than the tabled value of 0.901

UCL (based on t-statistic) is 26.48

Kingston Neighborhood and Public Recreational Exposures to Soil/Sediment

Iron

Analytical Results
(mg/kg)

87200
39900
76600
68400
83800
174000
120000
91700
74900
99000
118000
120000
93500
89200
75500
77700
77000
76400
49200

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	19	Mean	89052.63
Censored		Lognormal mean	89353.78
Detection limit or PQL		Std. devn.	29373.35
Method detection limit		Median	83800
TOTAL	19	Min.	39900
		Max.	174000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9414. This exceeds the tabled value of 0.901

UCL (based on t-statistic) is 100737.55

Kingston Neighborhood and Public Recreational Exposures to Soil/Sediment

Manganese

Analytical Results
(mg/kg)

6690
3000
5830
5450
6440
14800
9510
7460
6120
7810
14300
9360
7540
7420
6830
6130
6290
6960
3240

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	19	Mean	7430.53
Censored		Lognormal mean	7466.51
Detection limit or PQL		Std. devn.	2983.271
Method detection limit		Median	6830
TOTAL	19	Min.	3000
		Max.	14800

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9118. This exceeds the tabled value of 0.901

UCL (based on t-statistic) is 8617.29

Kingston Neighborhood and Public Recreational Exposures to Soil/Sediment

Zinc

Analytical Results
(mg/kg)

2950
1740
2470
2290
2780
6260
4810
3480
2470
3250
5040
4130
3040
2960
3450
2240
3320
3420
2280

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	19	Mean	3283.16
Censored		Lognormal mean	3285.72
Detection limit or PQL		Std. devn.	1115.462
Method detection limit		Median	3040
TOTAL	19	Min.	1740
		Max.	6260

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9706. This exceeds the tabled value of 0.901

UCL (based on t-statistic) is 3726.9

Kingston Public Recreational Exposure to Surface Water

Arsenic

Analytical Results
(ug/L)

3.6
26.7
7.2
10.5
134

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	36.40
Censored		Lognormal mean	41.89
Detection limit or PQL		Std. devn.	55.27011
Method detection limit		Median	10.5
TOTAL	5	Min.	3.6
		Max.	134

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9464. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 89.1

Kingston Public Recreational Exposure to Surface Water

Cadmium

Analytical Results
(ug/L)

11.6
26
13.4
12.7
83.8

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	29.50
Censored		Lognormal mean	29.99
Detection limit or PQL		Std. devn.	30.91359
Method detection limit		Median	13.4
TOTAL	5	Min.	11.6
		Max.	83.8

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.7991. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 58.97

Kingston Public Recreational Exposure to Surface Water

Manganese

Analytical Results
(ug/L)

857
3020
1280
1100
9470

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	3145.40
Censored		Lognormal mean	3290.13
Detection limit or PQL		Std. devn.	3637.184
Method detection limit		Median	1280
TOTAL	5	Min.	857
		Max.	9470

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8771. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 6613.31

Kingston Public Recreational Exposure to Surface Water

Mercury

Analytical Results
(ug/L)

0.1
1.5
0.2
0.1
4.6

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	1.30
Censored		Lognormal mean	1.91
Detection limit or PQL		Std. devn.	1.937782
Method detection limit		Median	0.2
TOTAL	5	Min.	0.1
		Max.	4.6

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8509. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 3.15

Kingston Occupational Exposure to Soil

Antimony

Analytical Results (mg/kg)

0.1	0.6	0.495	2.2	0.72	31.9
0.21	2.1	0.6	1.35	1.2	44.9
0.27	3.2	1	1.7	1	45.1
0.39	0.56	1.05	1.05	1	49.5
0.35	0.59	1.25	0.87	21.4	36.7
0.21	1.4	0.5	0.49	1.1	36.1
0.64	3.9	0.5	0.5	1.1	26.6
0.74	25.9	0.7	0.5	0.65	23.2
0.1	10.9	1.9	1.2	0.73	28.7
0.25	7.8	0.7	0.85	1.4	24.8
0.84	26	1.1	0.85	1.7	17.8
0.37	25.8	1.1	0.9	3.1	33.3
0.64	0.5	1.25	1.05	0.7	16.5
0.66	0.65	1	0.8	0.85	26.7
0.69	1.25	6.9	0.65	0.275	21.8
0.54	1.7	30.4	1	1	23.9
0.44	1.2	1.75	1.1	3.7	
1.2	0.75	1.2	2	1.35	
0.56	2.4	1.25	2.8	1.35	
0.64	18	1.9	0.495	1.1	
0.86	2.65	2.8	1.35	2.3	
0.85	0.8	1.5	1.4	1	
0.55	0.95	1.7	1.05	4.4	
0.495	1.05	2.4	17.1	1.9	
0.65	0.85	11.6	0.5	2.9	
1.15	0.8	7.5	1	2.5	
1.35	0.9	9.3	1.3	7.8	
0.5	1	12.2	1.8	1.1	
1.2	0.85	8.2	2.5	3.4	
1.5	0.485	1	2.9	0.6	
1.7	0.5	1.2	0.485	1.6	
1.05	0.55	1.4	0.9	0.77	
1.8	0.65	2	2	1.4	
1.85	0.3	5.7	2.6	0.44	
1.9	0.56	6.2	0.65	64.3	
1.55	1.1	7.7	0.9	53.9	
0.485	1.7	9.4	1.65	46.6	
0.5	6.1	0.8	1.15		

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	205	Mean	5.51
Censored		Lognormal mean	4.30
Detection limit or PQL		Std. devn.	10.99858
Method detection limit		Median	1.2
TOTAL	205	Min.	0.1
		Max.	64.3

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -7.987. This lies outside the tabled values of 1.5009 and -2.3867
Reject normal distribution.
Y value is -46.5017. This lies outside the tabled values of 1.5009 and -2.3867

UCL (based on t-statistic) is 6.78

Kingston Occupational Exposure to Soil

Arsenic

Analytical Results (mg/kg)

266	16.1	13.2	9.4	6.4	12.1
209	19.8	6.4	17.5	10.8	9.5
134	11.9	10.5	184	27.2	95.2
136	9.8	43.6	20.6	6.4	13.4
166	12	13.9	19.4	6.9	11.8
174	13.5	8.8	21.6	7.5	8.3
209	13.9	8.9	27.2	8.1	8.5
143	15.7	9.4	21.8	8.3	12.2
140	10.3	9.3	18.1	8.3	9.9
107	15.6	7.4	19.8	10.9	13.2
113	8.5	7.9	28.2	11.7	13.3
123	21.8	8	14.6	10.2	13.2
104	25.4	9.2	32.7	11.4	12.9
93.9	6.6	5.8	12.8	6.4	14.7
128	10.1	6.4	14.3	10.3	19.2
73	19.3	6.8	23.4	11	19.9
151	34.2	7.1	14.2	10.2	22.2
133	9.5	4.8	7.6	8.2	14.9
155	7.8	10.7	7.8	14.8	17.7
10	8.9	11.2	7.8	40.3	15.3
20.4	10.6	14.7	8.2	12.6	15.5
27.3	13.1	15.8	6.9	84	12
32.1	10.7	24.8	7.8	7.8	8.1
25.6	6.1	28.3	9.3	9.8	5.1
20.6	8.3	34.6	10.3	10.7	12.5
38.7	8.8	7	10.3	9.4	7.2
25.5	10	9.5	15.5	13.8	15.3
11.9	12.4	11.8	13.4	10.2	28.4
18.2	32.3	14	13.4	14	16.6
35.4	133	15.4	13.4	22.5	15.8
14.9	105	4.1	14.7	24.8	66.7
15.6	30.6	6.2	10.5	22.3	8.1
21.4	93.1	6.6	9.6	20.4	12.3
28.4	78.4	7.8	17.7	49.6	8.6
11.7	5.1	8.6	19.2	51.5	7.2
12.7	7.1	8.6	19.1	19.7	10.9
17.5	10.7	11.7	103	10.3	
12.1	14.8	11.6	5.8	11	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	226	Mean	29.06
Censored		Lognormal mean	25.13
Detection limit or PQL		Std. devn.	42.70652
Method detection limit		Median	13.2
TOTAL	226	Min.	4.1
		Max.	266

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -11.1267. This lies outside the tabled values of 1.5215 and -2.3686
Reject normal distribution.
Y value is -45.3618. This lies outside the tabled values of 1.5215 and -2.3686

UCL (based on t-statistic) is 33.77

Kingston Occupational Exposure to Soil

Cadmium

Analytical Results (mg/kg)

63.3	1.2	2.1	3.4	0.04	3
29.6	1.6	1.8	4.7	0.04	3.9
23	1.5	3.3	12.2	0.13	26
13.7	10.9	5.7	6.4	0.44	4.3
18.6	0.1	2.7	2.3	0.91	2.7
31.6	1.7	1.4	2.4	1.5	0.9
23	1.9	1.5	3.2	1.9	0.26
18.2	2.8	1.6	5	0.095	4.3
18.4	0.34	2.5	1.9	0.1	0.96
23.5	0.4	2.1	2	0.61	2.4
13.6	0.58	2.2	2.3	1.7	0.75
21.4	0.88	2.6	0.86	1.3	0.25
22.7	1.4	3.2	5.2	0.31	5.8
19	1.2	0.095	2.4	0.65	7.6
18	0.69	0.1	6.9	2.1	3.5
13.9	0.88	0.24	7.8	5.9	1.9
15.2	1.8	1.8	6.8	9.2	0.59
14.6	2.5	0.35	0.53	0.72	8.6
17.2	0.1	1.5	0.78	0.1	9.2
0.75	0.1	1.8	1.6	0.37	5
3.6	0.83	2.1	1.9	0.52	6.2
4	2.3	2.6	0.23	3	8.8
4.6	3.7	1.9	0.23	0.1	1
4.8	0.235	2.6	1.4	0.33	0.5
1.9	1.3	3.1	2.6	0.41	2.9
4.1	1.4	0.1	3.9	0.1	1.9
4.6	2.1	0.1	0.58	0.41	2.2
1.9	3.4	1.5	6.1	0.44	4.1
2.9	4.3	1.9	11.1	0.33	2.5
5.6	5.2	2.8	14	0.74	2.5
3.4	2.8	0.095	13.9	1.1	3
3.8	2	0.1	0.55	2.4	0.47
4	6.4	0.105	0.1	0.095	1.3
5.2	4.8	1.8	0.9	0.1	0.75
2.2	0.1	0.69	2	0.95	1.7
2.8	0.47	0.98	3.3	1.4	0.08
12.4	1.3	2	0.77	4	
3.2	2.1	2	0.04	3.6	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	226	Mean	4.24
Censored		Lognormal mean	5.14
Detection limit or PQL		Std. devn.	6.918806
Method detection limit		Median	2
TOTAL	226	Min.	0.04
		Max.	63.3

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -1.7908. This lies within the tabled values of 1.5215 and -2.3686

UCL (based on t-statistic) is 5.01

Kingston Occupational Exposure to Soil

Iron

Analytical Results (mg/kg)

2E+05	15200	20100	15400	18300
1E+05	15900	20500	16300	18300
91700	16500	22700	13900	19900
74900	18300	20300	15800	16900
99000	18400	19800	17400	17000
1E+05	15400	20000	19800	18600
1E+05	16000	21400	20900	21200
93500	21300	19200	18400	21600
89200	16900	21500	34200	15500
75500	19700	14400	15000	17000
77700	19800	18900	15200	18500
77000	20000	19100	16500	19800
76400	17700	16200	14800	21300
49200	16500	16200	17500	14900
87200	17000	16300	16800	20800
39900	17000	16600	19400	22600
76600	17400	16000	16600	22500
68400	17700	15400	32800	15700
83800	17800	16300	17600	14600
21500	18500	16500	18500	11600
16200	17100	16600	17100	12410
17300	16700	15800	15600	12930
17900	17600	35100	19800	14350
20500	19000	19200	15400	18500
20600	19800	19900	16300	14410
17100	17900	21500	17500	17000
17500	12800	17800	18200	22800
18300	15400	17100	16900	17500
13800	16900	16500	19700	15800
18000	14100	17700	22400	15500
21500	14400	18100	25000	13150
21700	14800	19700	17200	17300
17100	17900	23100	17000	
15300	16800	14200	17000	
15800	17300	14700	18900	
18300	17400	17800	15500	
28500	20300	18800	96300	
18700	21300	15300	18000	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	184	Mean	25811.14
Censored		Lognormal mean	24201.13
Detection limit or PQL		Std. devn.	24312.66
Method detection limit		Median	17800
TOTAL	184	Min.	11600
		Max.	174000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -30.9251. This lies outside the tabled values of 1.4726 and -2.4105
Reject normal distribution.
Y value is -49.4006. This lies outside the tabled values of 1.4726 and -2.4105

UCL (based on t-statistic) is 28782.86

Kingston Occupational Exposure to Soil

Manganese

Analytical Results (mg/kg)

14800	202	776	407	891
9510	310	789	464	673
7460	491	792	468	373
6120	631	680	324	850
7810	611	507	352	616
14300	284	603	654	917
9360	498	664	847	874
7540	796	635	564	609
7420	527	1010	505	595
6830	491	548	278	807
6130	495	1330	455	820
6290	499	1340	469	823
6960	445	1040	478	630
3240	444	400	530	423
6690	474	445	404	1360
3000	492	451	538	1060
5830	477	485	492	1090
5450	236	265	1440	892
6440	272	454	316	441
638	360	635	609	343
566	406	662	626	446
683	534	634	534	350
715	538	625	604	759
774	600	803	473	751
739	692	1230	514	643
539	624	1300	569	716
617	573	1130	773	681
691	674	575	522	596
505	681	420	466	816
638	594	607	474	583
652	535	647	545	523
700	546	710	431	232
629	585	718	785	
553	533	183	796	
562	661	200	830	
792	701	278	742	
1400	898	471	9790	
891	758	397	966	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	184	Mean	1376.57
Censored		Lognormal mean	1110.24
Detection limit or PQL		Std. devn.	2371.79
Method detection limit		Median	624.5
TOTAL	184	Min.	183
		Max.	14800

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -20.543. This lies outside the tabled values of 1.4726 and -2.4105
Reject normal distribution.
Y value is -52.2377. This lies outside the tabled values of 1.4726 and -2.4105

UCL (based on t-statistic) is 1666.47

Kingston Occupational Exposure to Soil

Zinc

Analytical Results (mg/kg)

6260	140	264	415	43.8	158
4810	159	101	473	44.5	415
3480	166	187	676	95.8	8150
2470	24300	308	698	82.1	238
3250	74.3	282	463	108	172
5040	354	189	483	132	97.5
4130	413	194	528	145	77.1
3040	427	205	509	77.4	241
2960	258	240	230	82.4	257
3450	81.8	168	264	207	141
2240	131	174	433	238	95.3
3320	140	181	122	418	78
3420	154	250	946	1340	188
2280	259	52.7	329	74.3	536
2950	131	60.4	990	165	203
1740	189	103	1090	322	126
2470	290	174	900	452	84.1
2290	333	74.7	56	506	246
2780	196	273	74.8	97.9	291
211	220	292	116	108	207
598	484	293	133	128	224
650	1310	320	51.9	937	270
694	633	397	66.6	62	174
526	71.3	436	103	99.3	78.7
429	136	543	143	129	202
789	143	139	170	82.5	175
842	191	252	226	82.6	239
900	273	327	1010	107	423
1370	842	349	1760	98.9	252
1060	1240	460	2130	106	320
649	916	60.2	1850	245	611
951	307	79.8	93.1	407	126
1510	929	80.2	144	110	167
1820	455	246	346	131	131
406	59.2	210	392	256	217
967	113	230	598	275	49.6
11600	189	284	745	383	
1190	275	287	37	210	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	226	Mean	793.51
Censored		Lognormal mean	632.85
Detection limit or PQL		Std. devn.	2032.807
Method detection limit		Median	254
TOTAL	226	Min.	37
		Max.	24300

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -3.3951. This lies outside the tabled values of 1.5215 and -2.3686
Reject normal distribution.
Y value is -73.3778. This lies outside the tabled values of 1.5215 and -2.3686

UCL (based on t-statistic) is 1017.71

Side Gulches Residential Tap Water

Arsenic

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	8	Mean	1.92
Censored		Lognormal mean	2.18
Detection limit or PQL		Std. devn.	2.784822
Method detection limit		Median	0.72625
TOTAL	8	Min.	0.19
		Max.	8.4

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8903. This exceeds the tabled value of 0.818

UCL (based on t-statistic) is 3.78

Site ID	Static Result (ug/L)	Purged Result (u	Average Result (ug/L)
R048	0.85 **U**	1.4 J	1.125
R057	0.2 J	0.29 J	0.245
R064	0.37	0.285 **U**	0.3275
R066	2	3.2	2.6
R072	2.3	2.1	2.2
R085	7.6	9.2	8.4
R088	0.28	0.1 **U**	0.19
R120	0.25 **U**	0.25 **U**	0.25

Side Gulches Neighborhood Exposure to Surface Soil

Antimony

Analytical Result (mg/kg)
31.2
53.3
41.9
54.9
49.8

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	46.22
Censored		Lognormal mean	46.54
Detection limit or PQL		Std. devn.	9.779417
Method detection limit		Median	49.8
TOTAL	5	Min.	31.2
		Max.	54.9

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8644. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 55.54

Side Gulches Neighborhood Exposure to Surface Soil

Arsenic

Analytical Result (mg/kg)
68.9
87.2
98.5
72.1
73.1

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	79.96
Censored		Lognormal mean	80.13
Detection limit or PQL		Std. devn.	12.5231
Method detection limit		Median	73.1
TOTAL	5	Min.	68.9
		Max.	98.5

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8778. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 91.9

Side Gulches Neighborhood Exposure to Surface Soil

Cadmium

Analytical Result (mg/kg)
8.1
64.5
11.7
14.4
10.3

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	21.80
Censored		Lognormal mean	21.80
Detection limit or PQL		Std. devn.	23.97916
Method detection limit		Median	11.7
TOTAL	5	Min.	8.1
		Max.	64.5

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.7876. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 44.66

Side Gulches Neighborhood Exposure to Surface Soil

Iron

Analytical Result (mg/kg)
67053
99928
84077
88771
88119

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	85589.60
Censored		Lognormal mean	85798.19
Detection limit or PQL		Std. devn.	11916.4
Method detection limit		Median	88119
TOTAL	5	Min.	67053
		Max.	99928

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9029. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 96951.4

Side Gulches Neighborhood Exposure to Surface Soil

Manganese

Analytical Result (mg/kg)
3590
9280
5060
8470
7530

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	6786.00
Censored		Lognormal mean	6928.34
Detection limit or PQL		Std. devn.	2387.62
Method detection limit		Median	7530
TOTAL	5	Min.	3590
		Max.	9280

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9025. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 9062.5

Side Gulches Neighborhood Exposure to Surface Soil

Zinc

Analytical Result (mg/kg)
1960
6130
2210
3410
2930

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	3328.00
Censored		Lognormal mean	3374.51
Detection limit or PQL		Std. devn.	1668.658
Method detection limit		Median	2930
TOTAL	5	Min.	1960
		Max.	6130

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.929. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 4919

Side Gulches Neighborhood Exposure to Sediments

Antimony

Analytical Result (mg/kg)
27
0.49
28.7
0.5
36.1
27.9
2.7
5.4
0.93
47.1

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	10	Mean	17.68
Censored		Lognormal mean	36.16
Detection limit or PQL		Std. devn.	17.52457
Method detection limit		Median	16.2
TOTAL	10	Min.	0.49
		Max.	47.1

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8415. This is less than the tabled value of 0.842
Assume normal distribution.
W value is 0.846. This exceeds the tabled value of 0.842

UCL (based on t-statistic) is 27.84

Side Gulches Neighborhood Exposure to Sediments

Arsenic

Analytical Result (mg/kg)
53.9
41.8
98.5
29.6
81.2
74.7
43.7
64.6
21.8
113

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	10	Mean	62.28
Censored		Lognormal mean	63.76
Detection limit or PQL		Std. devn.	29.70876
Method detection limit		Median	59.25
TOTAL	10	Min.	21.8
		Max.	113

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9682. This exceeds the tabled value of 0.842

UCL (based on t-statistic) is 79.5

Side Gulches Neighborhood Exposure to Sediments

Cadmium

Analytical Result (mg/kg)
5.6
0.1
3
0.1
15.8
112
1.7
1.6
1.1
45.6

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	10	Mean	18.66
Censored		Lognormal mean	42.77
Detection limit or PQL		Std. devn.	35.64966
Method detection limit		Median	2.35
TOTAL	10	Min.	0.1
		Max.	112

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9507. This exceeds the tabled value of 0.842

UCL (based on t-statistic) is 39.32

Side Gulches Neighborhood Exposure to Sediments

Iron

Analytical Result (mg/kg)
43200
19500
41700
20500
52200
23800
21000
26100
25233
21000

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	10	Mean	29423.30
Censored		Lognormal mean	29489.62
Detection limit or PQL		Std. devn.	11735.6
Method detection limit		Median	24516.5
TOTAL	10	Min.	19500
		Max.	52200

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8322. This is less than the tabled value of 0.842
Reject normal distribution.
W value is 0.7929. This is less than the tabled value of 0.842

UCL (based on t-statistic) is 36225.79

Side Gulches Neighborhood Exposure to Sediments

Manganese

Analytical Result (mg/kg)
2610
469
3040
640
4410
654
394
546
665
1250

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	10	Mean	1467.80
Censored		Lognormal mean	1485.79
Detection limit or PQL		Std. devn.	1393.349
Method detection limit		Median	659.5
TOTAL	10	Min.	394
		Max.	4410

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8658. This exceeds the tabled value of 0.842

UCL (based on t-statistic) is 2275.45

Side Gulches Neighborhood Exposure to Sediments

Zinc

Analytical Result (mg/kg)
1300
66.4
1450
112
3990
10500
639
615
276
9340

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	10	Mean	2828.84
Censored		Lognormal mean	4221.52
Detection limit or PQL		Std. devn.	3916.553
Method detection limit		Median	969.5
TOTAL	10	Min.	66.4
		Max.	10500

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9559. This exceeds the tabled value of 0.842

UCL (based on t-statistic) is 5099.05

Side Gulches Neighborhood Exposure to Surface Water

Arsenic

Analytical Results (ug/L)
12.2
5.5
32
7.8
25.7

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	16.64
Censored		Lognormal mean	17.80
Detection limit or PQL		Std. devn.	11.61865
Method detection limit		Median	12.2
TOTAL	5	Min.	5.5
		Max.	32

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9339. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 27.72

Side Gulches Neighborhood Exposure to Surface Water

Cadmium

Analytical Results (ug/L)
60.3
5.8
20.9
12.1
33.1

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	26.44
Censored		Lognormal mean	29.52
Detection limit or PQL		Std. devn.	21.52436
Method detection limit		Median	20.9
TOTAL	5	Min.	5.8
		Max.	60.3

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9937. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 46.96

Side Gulches Neighborhood Exposure to Surface Water

Manganese

Analytical Results (ug/L)
122
152
1520
1120
8570

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	2296.80
Censored		Lognormal mean	3643.89
Detection limit or PQL		Std. devn.	3559.187
Method detection limit		Median	1120
TOTAL	5	Min.	122
		Max.	8570

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9195. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 5690.34

Side Gulches Neighborhood Exposure to Surface Water

Mercury

Analytical Results (ug/L)
0.22
0.05
0.31
0.18
3.5

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	0.85
Censored		Lognormal mean	0.97
Detection limit or PQL		Std. devn.	1.48323
Method detection limit		Median	0.22
TOTAL	5	Min.	0.05
		Max.	3.5

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9093. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 2.27

Osburn Residential Exposure to Surface Soil

Antimony

Analytical Results

(mg/kg)

0.32	0.1	35.7	5.8	2.8	9.1	170	0.485	1.2
0.22	0.24	1.6	2.7	2.35	3.5	0.48	0.75	1.05
2.9	0.31	2.55	103	2.1	14.3	0.485	0.49	0.5
8.7	0.1	1	3.6	2.2	1.65	0.65	3.1	4.3
2.6	2.9	1.4	2.6	2.2	7.8	0.485	7.4	1.1
2.1	11.5	43.9	5.4	190	48	0.48	1	14.7
6.9	1.6	5.8	5.4	4.9	1.75	0.95	8.2	1.45
3.6	2.4	2.1	2.1	2.9	8.8	0.5	1.05	13.2
2	2.2	10.6	8.8	1.6	29.8	19.3	1.4	6.9
1.2	2.2	2.4	3	0.9	4.1	0.75	0.9	0.7
1.7	2	5	3	2.4	4.9	2.9	1.9	0.75
1.9	2.6	6.6	2.2	2	248	3.5	2	7.3
10	7	3.1	2.7	42.8	1.1	3.2	1.6	0.75
9.8	2.3	3	3.1	10	1.55	1.15	0.65	0.6
13.2	2.3	3.8	3	25.5	5.5	0.8	1.45	0.65
2.85	3.1	4.6	2.2	57.1	1.55	5.4	5.2	6.2
4.8	0.78	4.1	2.3	6.8	10.7	0.56	3.3	0.41
4.5	1.7	5.9	5.4	38.8	1.05	10.4	1.05	0.7
0.34	1.9	3.4	27.8	4.5	4.7	5.8	1.45	1.6
0.36	0.63	5.5	6.9	72.4	19.6	5.1	0.75	8.3
1.9	47.2	0.5	16	5.8	2.4	2	1.2	1.35
3	3.7	96.6	6.1	8.3	2.2	4.4	0.8	11.3
13.8	3.9	2.2	4.4	4.6	18.9	1.45	21.2	1.9
1	3.5	0.495	3.3	3.7	2.4	5.6	1	4.7
0.55	1.7	28.4	6	2.3	4.1	1.1	114	12.8
1.8	3	4.1	1.45	174	2	7.1	10.4	1.2
1.2	11	46.3	0.5	2	1.25	5.7	1.05	22.1
2.2	2.8	5.8	1.15	4.4	24.6	8.7	8.7	
2.2	102	6.5	2.7	3	1.25	10.1	9.9	
3.9	17.3	4.3	1.25	4.8	14.4	8.3	1.05	
3.2	25.3	6.7	2.35	2.4	1.2	1.15	29.4	
0.85	19	3.5	4.3	7.2	10.6	0.75	7.6	
4.2	2.2	12.2	2.1	1.6	1.4	1.1	3.7	
3.4	0.9	12.9	1.7	2.5	1.15	1.05	3.4	
3.8	138	4.6	1.6	5.5	0.7	0.55	0.8	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	307	Mean	10.10
Censored		Lognormal mean	7.81
Detection limit or PQL		Std. devn.	27.06608372
Method detection limit		Median	2.9
TOTAL	307	Min.	0.1
		Max.	248

Lognormal distribution? Normal distribution?

r-squared is: r-squared is:

Recommendations:

Reject lognormal distribution.

Y value is -4.427. This lies outside the tabled values of 1.5816 and -2.3125

Reject normal distribution.

Y value is -84.2673. This lies outside the tabled values of 1.5816 and -2.3125

UCL (based on t-statistic) is 12.66

Osburn Residential Tap Water

Arsenic

Site ID	Static Result (ug/L)	Purged Result (ug/L)	Average Result (ug/L)
R001	0.22 J	0.28 J	0.25
R003	0.1 U	0.1 U	0.1
R004	0.1 U	0.1 U	0.1
R005	0.27 U	0.22 U	0.245
R008	0.25	0.31	0.28
R012	0.3	0.47	0.385
R013	0.22	0.1 U	0.16
R014	0.23	0.1 U	0.165
R017	0.1 U	0.1 U	0.1
R020	0.3 J	0.34 J	0.32
R021	0.34	0.31	0.325
R024	0.31	0.21	0.26
R028	0.35	0.44	0.395
R029	0.1 U	0.1 U	0.1
R033	0.1 U	0.1 U	0.1
R036	0.1 U	0.1 U	0.1
R043	0.39	1.4	0.895
R047	0.31 J	0.29 J	0.3
R051	0.195 U	0.35 U	0.2725
R058	0.1 U	0.24	0.17
R059	0.235 U	0.34 J	0.2875
R060	0.1 U	0.1 U	0.1
R061	0.1 U	0.1 U	0.1
R062	0.1 U	0.1 U	0.1
R073	0.21 U	0.32 U	0.265
R084	0.1 U	0.24	0.17
R090	0.25	0.1 U	0.175
R109	0.25 U	0.25 U	0.25
R121	0.25 U	0.25 U	0.25
R128	0.25 U	0.25 U	0.25
R129	0.25 U	0.25 U	0.25
R130	0.25 U	0.25 U	0.25

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	32	Mean	0.23
Censored		Lognormal mean	0.23
Detection limit or PQL		Std. devn.	0.150154
Method detection limit		Median	0.25
TOTAL	32	Min.	0.1
		Max.	0.895

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8837. This is less than the tabled value of 0.93
Reject normal distribution.
W value is 0.7185. This is less than the tabled value of 0.93

UCL (based on t-statistic) is 0.28

Silverton Residential Surface Soil

Antimony

Analytical Results
(mg/kg)

0.35	30.7
2.5	0.48
22.4	1.45
6.1	0.85
2.9	0.84
4	0.67
3.3	0.84
2.2	1
82.5	4.2
2	4.8
2.2	2.7
2.1	11
4	5.8
1.4	1.9
0.5	3.7
0.55	1.2
23	0.5
2	3.9
3.9	4.3
1.8	0.5
4.3	0.5
1.4	1.8
4.3	
28	
0.9	
1.7	
6.1	
1.8	
2.5	
3.6	
0.31	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	53	Mean	5.82
Censored		Lognormal mean	4.89
Detection limit or PQL		Std. devn.	12.6157
Method detection limit		Median	2.2
TOTAL	53	Min.	0.31
		Max.	82.5

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -1.9509. This lies within the tabled values of 1.0611 and -2.7396

UCL (based on t-statistic) is 8.72

Silverton Residential Surface Soil

Arsenic

Analytical Results
(mg/kg)

12.4	4.6
10.1	133
30.5	4.8
16.3	7.1
10.8	19.1
33.5	4.1
11	4.6
17	5.3
25.6	8.5
19.8	8.7
15.7	17.3
7.2	13.4
15.7	12
6.25	15.7
13.6	14.3
10.8	4.61
11.7	15.3
38.9	7.8
12.8	4.6
17.5	10.7
10.4	12.2
16.5	14.9
11.3	15.6
18.5	17.4
51.5	
13.9	
17.9	
20.1	
8.8	
11.9	
15.1	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	55	Mean	16.52
Censored		Lognormal mean	15.79
Detection limit or PQL		Std. devn.	18.16282
Method detection limit		Median	13.4
TOTAL	55	Min.	4.1
		Max.	133

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -4.2128. This lies outside the tabled values of 1.0765 and -2.728
Reject normal distribution.
Y value is -27.3967. This lies outside the tabled values of 1.0765 and -2.728

UCL (based on t-statistic) is 20.62

Silverton Residential Surface Soil

Cadmium

Analytical Results
(mg/kg)

6	0.5
3	7.2
1.2	0.55
5.3	0.66
3.1	1.7
3.9	1.6
4	2.9
4.9	2.7
7.9	2.1
1.9	1.5
2.2	1.7
2	4.1
4.1	3.9
23.8	11.9
1.4	7.8
1.6	1.8
2.9	3.9
1.7	3.7
2.8	1.4
6.5	2.8
3.6	3.8
7.6	3.3
2.5	3.9
9	3.5
14.6	
5.3	
3.6	
12.7	
2.2	
3.7	
8.1	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	55	Mean	4.40
Censored		Lognormal mean	4.41
Detection limit or PQL		Std. devn.	4.011165
Method detection limit		Median	3.5
TOTAL	55	Min.	0.5
		Max.	23.8

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -1.3422. This lies within the tabled values of 1.0765 and -2.728

UCL (based on t-statistic) is 5.31

Silverton Residential Surface Soil

Iron

Analytical Results
(mg/kg)

15300	56700
25200	13900
17000	16000
14400	16800
9690	15300
16200	13500
16400	12900
47000	16100
25500	18300
22400	16900
18500	16940
20400	15140
15100	18600
19900	15540
18800	12540
15200	13430
14800	14220
17000	11890
14300	12490
13900	16200
19300	14460
16000	15970
17500	16610
38500	
21500	
20000	
21100	
14800	
19200	
21100	
15100	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	54	Mean	18361.48
Censored		Lognormal mean	18182.72
Detection limit or PQL		Std. devn.	7957.572
Method detection limit		Median	16200
TOTAL	54	Min.	9690
		Max.	56700

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -9.5761. This lies outside the tabled values of 1.0688 and -2.7338
Reject normal distribution.
Y value is -19.8476. This lies outside the tabled values of 1.0688 and -2.7338

UCL (based on t-statistic) is 20175.92

Silverton Residential Surface Soil

Manganese

Analytical Results
(mg/kg)

601	3440
1460	436
1010	574
623	798
793	797
690	472
885	381
3390	486
1080	598
973	699
859	793
1040	923
594	1230
1060	785
923	501
785	727
662	722
596	472
679	284
569	918
1160	640
610	588
1030	706
2270	
1080	
1570	
1510	
678	
1050	
1480	
427	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	54	Mean	927.91
Censored		Lognormal mean	912.43
Detection limit or PQL		Std. devn.	605.1059
Method detection limit		Median	785
TOTAL	54	Min.	284
		Max.	3440

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -3.4124. This lies outside the tabled values of 1.0688 and -2.7338
Reject normal distribution.
Y value is -15.479. This lies outside the tabled values of 1.0688 and -2.7338

UCL (based on t-statistic) is 1065.88

Silverton Residential Surface Soil

Zinc

Analytical Results
(mg/kg)

496	62.3
274	5000
1530	71.8
672	154
328	208
548	101
379	200
558	162
1460	214
427	154
409	148
471	635
489	274
596	1010
269	748
253	116
373	287
309	288
420	119
1290	173
304	396
1190	316
313	298
763	403
3500	
545	
405	
1020	
323	
391	
903	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	55	Mean	595.38
Censored		Lognormal mean	561.02
Detection limit or PQL		Std. devn.	805.4685
Method detection limit		Median	379
TOTAL	55	Min.	62.3
		Max.	5000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -2.1282. This lies within the tabled values of 1.0765 and -2.728

UCL (based on t-statistic) is 777.29

Silverton Residential Tap Water
Arsenic

Site ID	**Static Result** (ug/L)	**Purged Result** (ug/L)	**Average Result** (ug/L)	
R022	0.56	0.53	0.545	
R027	0.33	0.23	0.280	
R045	0.31	0.39	0.350	
R075	0.38	0.3	0.340	
			0.545	**MAX**
			0.37875	**Average**
			0.1151	**Std Dev**
			4	**Count**
			2	**Count^0.5**
			2.353	**t-statistic**
			0.5141	**UCL_{95}normal**

Silverton Public Recreational Exposure to Soil

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
0.34	-1.0788	6	1.7918	0.39	-0.9416	14600	9.5888	613	6.4184	42.3	3.7448
0.95	-0.0513	6.2	1.8245	0.53	-0.6349	16400	9.7050	633	6.4505	57.4	4.0500
1.3	0.2624	7.7	2.0412	0.83	-0.1863	17800	9.7870	735	6.5999	113	4.7274
1.4	0.3365	8.7	2.1633	2.4	0.8755	17900	9.7926	1110	7.0121	273	5.6095
1.6	0.4700	9.6	2.2618	3.6	1.2809	16400	9.7050	931	6.8363	236	5.4638
1.6	0.4700	8.7	2.1633	2.1	0.7419	18800	9.8416	1030	6.9373	275	5.6168
2.2	0.7885	10.2	2.3224	3.3	1.1939	19000	9.8522	1180	7.0733	358	5.8805
2.5	0.9163	14.9	2.7014	3.4	1.2238	19500	9.8782	1260	7.1389	400	5.9915
1.9	0.6419	10.7	2.3702	3.6	1.2809	16700	9.7232	1120	7.0211	327	5.7900
1	0.0000	6	1.7918	0.03	-3.5066	14700	9.5956	444	6.0958	43.6	3.7751
1.9	0.6419	7.2	1.9741	0.03	-3.5066	15500	9.6486	645	6.4693	59.5	4.0860
1.5	0.4055	7.9	2.0669	0.93	-0.0726	15900	9.6741	705	6.5582	152	5.0239
1.3	0.2624	9.7	2.2721	2.8	1.0296	14800	9.6024	771	6.6477	226	5.4205
0.82	-0.1985	6.1	1.8083	0.03	-3.5066	14000	9.5468	552	6.3135	37.4	3.6217
1.3	0.2624	7.6	2.0281	0.03	-3.5066	14600	9.5888	589	6.3784	43.9	3.7819
1.3	0.2624	7.9	2.0669	1.3	0.2624	16600	9.7172	813	6.7007	212	5.3566
1.6	0.4700	8.6	2.1518	2.3	0.8329	15800	9.6678	833	6.7250	272	5.6058
0.97	-0.0305	8	2.0794	0.74	-0.3011	18100	9.8037	846	6.7405	140	4.9416
2.2	0.7885	11.2	2.4159	1.6	0.4700	19400	9.8730	1120	7.0211	332	5.8051
2.9	1.0647	12.1	2.4932	2.3	0.8329	20100	9.9085	1140	7.0388	354	5.8693
1.1	0.0953	10	2.3026	2.9	1.0647	16400	9.7050	1020	6.9276	316	5.7557
1.4	0.3365	7.7	2.0412	0.03	-3.5066	18200	9.8092	556	6.3208	79.6	4.3770
0.75	-0.2877	8.8	2.1748	0.95	-0.0513	19400	9.8730	861	6.7581	284	5.6490
1.8	0.5878	10.7	2.3702	4.1	1.4110	19900	9.8985	1130	7.0300	679	6.5206
1.8	0.5878	12.7	2.5416	3.9	1.3610	16800	9.7291	724	6.5848	431	6.0661
2.2	0.7885	3.6	1.2809	0.03	-3.5066	7420	8.9119	202	5.3083	19.8	2.9857
0.69	-0.3711	3.8	1.3350	0.03	-3.5066	8310	9.0252	212	5.3566	39.5	3.6763
0.77	-0.2614	8.3	2.1163	1.1	0.0953	16600	9.7172	782	6.6619	229	5.4337
1.5	0.4055	9.4	2.2407	2.8	1.0296	16000	9.6803	761	6.6346	276	5.6204
1.8	0.5878	5.8	1.7579	0.03	-3.5066	13100	9.4804	402	5.9965	94.8	4.5518
1.6	0.4700	10.6	2.3609	2.2	0.7885	17500	9.7700	934	6.8395	345	5.8435
1.7	0.5306	11	2.3979	2.6	0.9555	18000	9.7981	938	6.8437	350	5.8579
2.1	0.7419	11.5	2.4423	3.8	1.3350	16000	9.6803	826	6.7166	320	5.7683
1.7	0.5306	4.7	1.5476	0.03	-3.5066	11500	9.3501	350	5.8579	46.5	3.8395
0.485	-0.7236	6.4	1.8563	0.18	-1.7148	15900	9.6741	557	6.3226	130	4.8675
0.5	-0.6931	8.2	2.1041	2.6	0.9555	17400	9.7642	772	6.6490	460	6.1312
1.3	0.2624	8.6	2.1518	3.8	1.3350	15700	9.6614	786	6.6670	375	5.9269
1.1	0.0953	10.8	2.3795	1.9	0.6419	18800	9.8416	1060	6.9660	358	5.8805
0.485	-0.7236	11.3	2.4248	2	0.6931	19600	9.8833	1120	7.0211	370	5.9135
0.5	-0.6931	11.7	2.4596	2.1	0.7419	20200	9.9134	1160	7.0562	403	5.9989
1.3	0.2624	9.8	2.2824	2.5	0.9163	16500	9.7111	1010	6.9177	287	5.6595
1.4	0.3365	6.9	1.9315	0.095	-2.3539	13300	9.4955	419	6.0379	43.8	3.7796
1.3	0.2624	6.9	1.9315	0.1	-2.3026	14900	9.6091	451	6.1115	63	4.1431
1.4	0.3365	8.1	2.0919	0.56	-0.5798	19600	9.8833	735	6.5999	224	5.4116
1.5	0.4055	8.7	2.1633	2	0.6931	16200	9.6928	685	6.5294	266	5.5835
1.7	0.5306	6.2	1.8245	0.095	-2.3539	13500	9.5104	473	6.1591	46.1	3.8308
1.7	0.5306	6.9	1.9315	0.1	-2.3026	15800	9.6678	499	6.2126	62	4.1271
2	0.6931	8.1	2.0919	1.1	0.0953	18000	9.7981	774	6.6516	279	5.6312
2.4	0.8755	9.5	2.2513	3.3	1.1939	18100	9.8037	921	6.8255	428	6.0591
3	1.0986	10.7	2.3702	1.3	0.2624	18000	9.7981	1060	6.9660	327	5.7900
0.495	-0.7032	10.9	2.3888	1.9	0.6419	19100	9.8574	1150	7.0475	352	5.8636
0.5	-0.6931	12.2	2.5014	1.9	0.6419	20100	9.9085	1160	7.0562	364	5.8972
1.7	0.5306	10.1	2.3125	2.5	0.9163	17000	9.7410	1110	7.0121	332	5.8051
1.8	0.5878	10.3	2.3321	1.9	0.6419	16900	9.7351	1050	6.9565	335	5.8141
0.495	-0.7032	11.8	2.4681	2.4	0.8755	18700	9.8363	1110	7.0121	381	5.9428
0.5	-0.6931	12.9	2.5572	3.6	1.2809	22300	10.0123	1160	7.0562	420	6.0403
0.5	-0.6931	13.2	2.5802	2.7	0.9933	17400	9.7642	1020	6.9276	358	5.8805
2.1	0.7419	7.2	1.9741	0.68	-0.3857	15800	9.6678	589	6.3784	39.2	3.6687
1.6	0.4700	7.4	2.0015	1.4	0.3365	17100	9.7468	601	6.3986	125	4.8283
2	0.6931	9.6	2.2618	3.4	1.2238	19000	9.8522	1020	6.9276	347	5.8493
4.8	1.5686	9.9	2.2925	4.6	1.5261	19300	9.8679	1070	6.9754	363	5.8944
4.9	1.5892	7	1.9459	0.78	-0.2485	16000	9.6803	765	6.6399	70.5	4.2556
0.495	-0.7032	7.6	2.0281	1.1	0.0953	18000	9.7981	874	6.7731	108	4.6821
1.6	0.4700	7.9	2.0669	2	0.6931	20900	9.9475	1210	7.0984	214	5.3660
2	0.6931	9.2	2.2192	4.5	1.5041	17000	9.7410	807	6.6933	458	6.1269
5.4	1.6864	9.5	2.2513	3.5	1.2528	18100	9.8037	820	6.7093	476	6.1654
1.6	0.4700	10.4	2.3418	4	1.3863	18100	9.8037	868	6.7662	497	6.2086
6.1	1.8083	23	3.1355	4.9	1.5892	18300	9.8147	982	6.8896	516	6.2461
23.7	3.1655	38.9	3.6610	5	1.6094	24700	10.1146	1170	7.0648	602	6.4003
18.1	2.8959	7	1.9459	1.9	0.6419	14600	9.5888	471	6.1549	245	5.5013
0.5	-0.6931	9.8	2.2824	2.8	1.0296	18900	9.8469	891	6.7923	333	5.8081
0.5	-0.6931	19.5	2.9704	4	1.3863	21500	9.9758	949	6.8554	350	5.8579
1.2	0.1823	34.3	3.5351	4	1.3863	24300	10.0982	990	6.8977	466	6.1442
1.9	0.6419	11.1	2.4069	2.7	0.9933	18100	9.8037	1030	6.9373	379	5.9375
1.8	0.5878	32.7	3.4874	4.8	1.5686	23500	10.0648	1840	7.5175	1100	7.0031
0.5	-0.6931	145	4.9767	6.4	1.8563	40900	10.6189	2600	7.8633	2900	7.9725
1.2	0.1823	68.6	4.2283	7.9	2.0669	44200	10.6965	2530	7.8360	4070	8.3114
1.6	0.4700	8.1	2.0919	0.1	-2.3026	17800	9.7870	587	6.3750	88.7	4.4853
1.5	0.4055	10.1	2.3125	1	0.0000	19800	9.8934	960	6.8669	296	5.6904

Silverton Public Recreational Exposure to Soil

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
1	0.0000	10.7	2.3702	1.8	0.5878	19800	9.8934	1050	6.9565	386	5.9558
0.5	-0.6931	11.4	2.4336	2.5	0.9163	20100	9.9085	1140	7.0388	386	5.9558
1.3	0.2624	11.2	2.4159	2.1	0.7419	19600	9.8833	1090	6.9939	407	6.0088
1.9	0.6419	10.7	2.3702	2	0.6931	19300	9.8679	1040	6.9470	311	5.7398
2.3	0.8329	11.2	2.4159	2	0.6931	20200	9.9134	1110	7.0121	329	5.7961
2.4	0.8755	12.1	2.4932	2.6	0.9555	21700	9.9851	1160	7.0562	342	5.8348
2.6	0.9555	9.2	2.2192	3.6	1.2809	16600	9.7172	954	6.8607	338	5.8230
1.7	0.5306	7.7	2.0412	0.1	-2.3026	16300	9.6989	596	6.3902	68.3	4.2239
0.72	-0.3285	9	2.1972	0.28	-1.2730	17900	9.7926	699	6.5497	84.2	4.4332
0.9	-0.1054	9.2	2.2192	1.2	0.1823	18400	9.8201	836	6.7286	181	5.1985
1.2	0.1823	10.2	2.3224	2.8	1.0296	18400	9.8201	1000	6.9078	280	5.6348
0.5	-0.6931	11	2.3979	3.1	1.1314	18800	9.8416	1050	6.9565	406	6.0064
1.3	0.2624	12	2.4849	4.1	1.4110	20200	9.9134	1130	7.0300	435	6.0753
1.4	0.3365	12.3	2.5096	4.2	1.4351	21600	9.9804	1240	7.1229	441	6.0890
2.3	0.8329	10.7	2.3702	3.8	1.3350	18600	9.8309	996	6.9037	372	5.9189
1.9	0.6419	7	1.9459	0.03	-3.5066	15100	9.6225	479	6.1717	58.6	4.0707
0.88	-0.1278	7.3	1.9879	0.03	-3.5066	15600	9.6550	481	6.1759	62.3	4.1320
1.1	0.0953	9.6	2.2618	1.2	0.1823	20500	9.9282	810	6.6970	276	5.6204
1.8	0.5878	9.3	2.2300	1.5	0.4055	17200	9.7527	803	6.6884	246	5.5053
3.1	1.1314	10.6	2.3609	0.36	-1.0217	20100	9.9085	771	6.6477	183	5.2095
1	0.0000	11	2.3979	1.9	0.6419	20800	9.9427	1120	7.0211	339	5.8260
2.1	0.7419	11.9	2.4765	3	1.0986	20900	9.9475	1150	7.0475	343	5.8377
1.8	0.5878	10.5	2.3514	3.6	1.2809	18800	9.8416	1030	6.9373	342	5.8348
0.89	-0.1165	7.6	2.0281	0.03	-3.5066	15100	9.6225	658	6.4892	44.1	3.7865
1.3	0.2624	7.9	2.0669	0.03	-3.5066	15900	9.6741	733	6.5971	86.2	4.4567
1.4	0.3365	10.1	2.3125	2	0.6931	18100	9.8037	950	6.8565	353	5.8665
1.7	0.5306	14.8	2.6946	3.6	1.2809	18600	9.8309	1090	6.9939	523	6.2596
0.49	-0.7133	7.6	2.0281	0.03	-3.5066	17800	9.7870	584	6.3699	81.4	4.3994
0.5	-0.6931	9.2	2.2192	0.03	-3.5066	21100	9.9570	910	6.8134	168	5.1240
1.4	0.3365	11.2	2.4159	2	0.6931	21900	9.9942	1150	7.0475	388	5.9610
2.2	0.7885	9.9	2.2925	2.7	0.9933	17300	9.7585	1010	6.9177	304	5.7170
0.495	-0.7032	8.2	2.1041	0.22	-1.5141	18700	9.8363	894	6.7957	100	4.6052
1	0.0000	9.5	2.2513	0.33	-1.1087	20400	9.9233	934	6.8395	127	4.8442
0.5	-0.6931	10.2	2.3224	1.6	0.4700	20600	9.9330	945	6.8512	230	5.4381
1.6	0.4700	9.6	2.2618	3.1	1.1314	17400	9.7642	969	6.8763	257	5.5491
0.5	-0.6931	9.3	2.2300	0.1	-2.3026	19400	9.8730	617	6.4249	54.8	4.0037
1.7	0.5306	9.4	2.2407	0.1	-2.3026	19500	9.8782	640	6.4615	62.3	4.1320
2.2	0.7885	10.2	2.3224	1.8	0.5878	21600	9.9804	810	6.6970	319	5.7652
2.1	0.7419	11.1	2.4069	3.5	1.2528	18800	9.8416	766	6.6412	385	5.9532
1.1	0.0953	9.3	2.2300	0.37	-0.9943	22800	10.0345	766	6.6412	135	4.9053
2.1	0.7419	12.9	2.5572	2.2	0.7885	23700	10.0732	1140	7.0388	399	5.9890
3.5	1.2528	13	2.5649	2.7	0.9933	25600	10.1503	1150	7.0475	420	6.0403
7.2	1.9741	12.9	2.5572	4.7	1.5476	22600	10.0257	1070	6.9754	429	6.0615
1.4	0.3365	9.6	2.2618	0.33	-1.1087	23800	10.0774	631	6.4473	136	4.9127
1.8	0.5878	12.4	2.5177	2.4	0.8755	24400	10.1023	1060	6.9660	479	6.1717
2.2	0.7885	14.2	2.6532	4.8	1.5686	24600	10.1105	1360	7.2152	762	6.6359
0.99	-0.0101	12.9	2.5572	5.3	1.6677	23000	10.0432	1190	7.0817	703	6.5554
0.92	-0.0834	11.4	2.4336	0.96	-0.0408	23300	10.0562	1370	7.2226	207	5.3327
1.8	0.5878	16	2.7726	2.9	1.0647	24000	10.0858	1390	7.2371	448	6.1048
3.8	1.3350	21	3.0445	3.3	1.1939	24200	10.0941	2040	7.6207	450	6.1092
4.2	1.4351	38.4	3.6481	4.2	1.4351	25900	10.1620	1420	7.2584	452	6.1137
1.4	0.3365	10.8	2.3795	1.7	0.5306	19200	9.8627	1040	6.9470	331	5.8021
1.7	0.5306	10.8	2.3795	2.2	0.7885	19300	9.8679	1090	6.9939	370	5.9135
1.8	0.5878	12.3	2.5096	4.2	1.4351	22300	10.0123	1190	7.0817	441	6.0890
1.8	0.5878	12.5	2.5257	4.4	1.4816	21700	9.9851	1170	7.0648	435	6.0753
10	2.3026	6.2	1.8245	0.03	-3.5066	16400	9.7050	671	6.5088	77.8	4.3541
19	2.9444	8.6	2.1518	0.87	-0.1393	18200	9.8092	1090	6.9939	273	5.6095
32.8	3.4904	15.1	2.7147	4.5	1.5041	19400	9.8730	1210	7.0984	695	6.5439
6.9	1.9315	17.7	2.8736	4.6	1.5261	18000	9.7981	1120	7.0211	632	6.4489
0.335	-1.0936	10.3	2.3321	2.2	0.7885	18500	9.8255	1060	6.9660	361	5.8889
0.7	-0.3567	10.8	2.3795	2.4	0.8755	19200	9.8627	1140	7.0388	377	5.9322
1	0.0000	11.1	2.4069	2.7	0.9933	19700	9.8884	1180	7.0733	390	5.9661
1.2	0.1823	9.8	2.2824	3.1	1.1314	16900	9.7351	1010	6.9177	335	5.8141
0.76	-0.2744	80.3	4.3858	3.5	1.2528	24700	10.1146	1480	7.2998	362	5.8916
1.1	0.0953	241	5.4848	3.6	1.2809	30000	10.3090	1900	7.5496	553	6.3154
1.3	0.2624	415	6.0283	3.9	1.3610	35900	10.4885	2590	7.8594	560	6.3279
2	0.6931	53.9	3.9871	1.9	0.6419	23700	10.0732	1190	7.0817	350	5.8579
5.8	1.7579	5.2	1.6487	0.03	-3.5066	12700	9.4494	253	5.5334	31.9	3.4626
7.2	1.9741	6.3	1.8405	0.1	-2.3026	16900	9.7351	582	6.3665	55.9	4.0236
24.7	3.2068	8.6	2.1518	0.85	-0.1625	18800	9.8416	938	6.8437	176	5.1705
53.5	3.9797	8.8	2.1748	2.4	0.8755	16900	9.7351	1060	6.9660	268	5.5910
27	3.2958	6.8	1.9169	0.33	-1.1087	15600	9.6550	638	6.4583	49.3	3.8979
51	3.9318	7.4	2.0015	0.57	-0.5621	16100	9.6866	695	6.5439	75.3	4.3215
137	4.9200	7.5	2.0149	1.2	0.1823	16700	9.7232	737	6.6026	121	4.7958
72.8	4.2877	8.4	2.1282	2.7	0.9933	15500	9.6486	855	6.7511	177	5.1761
13.8	2.6247	26.5	3.2771	7.1	1.9601	22500	10.0213	1380	7.2298	1510	7.3199
17.8	2.8792	37.7	3.6297	11.7	2.4596	24100	10.0900	1470	7.2930	1760	7.4731
22.3	3.1046	129	4.8598	14.6	2.6810	32100	10.3766	2030	7.6158	2040	7.6207
19.9	2.9907	175	5.1648	8.4	2.1282	44800	10.7100	3140	8.0520	2390	7.7790

Silverton Public Recreational Exposure to Soil

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
3.7	1.3083	202	5.3083	4.2	1.4351	33300	10.4133	2480	7.8160	445	6.0981
4.3	1.4586	464	6.1399	4.4	1.4816	39300	10.5790	3680	8.2107	598	6.3936
5.8	1.7579	796	6.6796	4.5	1.5041	79800	11.2873	5940	8.6895	949	6.8554
12	2.4849	422	6.0450	4.3	1.4586	55400	10.9223	3980	8.2890	1420	7.2584
1.3	0.2624	43.1	3.7635	5.4	1.6864	26200	10.1735	2110	7.6544	1370	7.2226
1.4	0.3365	69.6	4.2428	7	1.9459	30800	10.3353	2230	7.7098	2160	7.6779
1.6	0.4700	73.1	4.2918	7.5	2.0149	34300	10.4429	2280	7.7319	2780	7.9302
2.4	0.8755	43.9	3.7819	6.5	1.8718	36300	10.4996	2410	7.7874	3080	8.0327
4	1.3863	13.9	2.6319	1.5	0.4055	12500	9.4335	666	6.5013	501	6.2166
2.25	0.8109	17.8	2.8792	1.7	0.5306	17300	9.7585	883	6.7833	635	6.4536
2.4	0.8755	21.9	3.0865	2.6	0.9555	21900	9.9942	1250	7.1309	884	6.7845
11.5	2.4423	51.5	3.9416	6.6	1.8871	23100	10.0476	1330	7.1929	1470	7.2930
32.8	3.4904	8.8	2.1748	0.41	-0.8916	14300	9.5680	524	6.2615	192	5.2575
2.1	0.7419	9.8	2.2824	0.56	-0.5798	15700	9.6614	550	6.3099	224	5.4116
3.4	1.2238	10.1	2.3125	0.8	-0.2231	16100	9.6866	717	6.5751	287	5.6595
3.8	1.3350	10.9	2.3888	2	0.6931	17000	9.7410	720	6.5793	511	6.2364
10.4	2.3418	14.3	2.6603	3.9	1.3610	16300	9.6989	670	6.5073	923	6.8276
7.9	2.0669	13.5	2.6027	2.5	0.9163	20100	9.9085	925	6.8298	659	6.4907
13	2.5649	17.4	2.8565	4	1.3863	23600	10.0690	1170	7.0648	1040	6.9470
23.4	3.1527	26.9	3.2921	7.5	2.0149	25400	10.1425	1460	7.2862	1730	7.4559
109	4.6913	59.5	4.0860	8.6	2.1518	41900	10.6430	2590	7.8594	4180	8.3381
1.3	0.2624	12.2	2.5014	0.55	-0.5978	19300	9.8679	781	6.6606	337	5.8201
1.6	0.4700	14.6	2.6810	2.5	0.9163	19500	9.8782	1050	6.9565	567	6.3404
2.5	0.9163	27	3.2958	7.8	2.0541	21400	9.9711	1580	7.3652	1190	7.0817
7.6	2.0281	34.4	3.5381	1.9	0.6419	20400	9.9233	1460	7.2862	854	6.7499
3.1	1.1314	22.9	3.1311	15.6	2.7473	22800	10.0345	1350	7.2079	2590	7.8594
3.3	1.1939	26.5	3.2771	16.4	2.7973	23800	10.0774	1420	7.2584	2710	7.9047
134	4.8978	38.1	3.6402	16.6	2.8094	35100	10.4660	2580	7.8555	2750	7.9194
17.7	2.8736	240	5.4806	2.4	0.8755	42200	10.6502	2730	7.9121	1220	7.1066
1.7	0.5306	8.8	2.1748	0.1	-2.3026	17000	9.7410	621	6.4313	145	4.9767
1.8	0.5878	9.1	2.2083	0.61	-0.4943	17000	9.7410	835	6.7274	235	5.4596
7.1	1.9601	9.2	2.2192	1.8	0.5878	17900	9.7926	857	6.7534	386	5.9558
18	2.8904	22	3.0910	1.4	0.3365	18200	9.8092	1230	7.1148	775	6.6529
18.6	2.9232	10	2.3026	1.6	0.4700	17100	9.7468	650	6.4770	352	5.8636
45.2	3.8111	11.4	2.4336	1.9	0.6419	19700	9.8884	861	6.7581	460	6.1312
166	5.1120	282	5.6419	4	1.3863	41900	10.6430	2780	7.9302	1150	7.0475
233	5.4510	31.5	3.4500	11	2.3979	30300	10.3189	2180	7.6871	2220	7.7053
0.55	-0.5978	12	2.4849	0.1	-2.3026	23400	10.0605	1400	7.2442	253	5.5334
1.7	0.5306	16.3	2.7912	0.1	-2.3026	24700	10.1146	1620	7.3902	262	5.5683
7.8	2.0541	20.2	3.0057	0.86	-0.1508	25600	10.1503	1670	7.4206	541	6.2934
8.8	2.1748	49.8	3.9080	3.3	1.1939	30000	10.3090	1640	7.4025	1810	7.5011
0.7	-0.3567	132	4.8828	2.1	0.7419	28600	10.2612	1760	7.4731	645	6.4693
1.2	0.1823	308	5.7301	2.1	0.7419	34600	10.4516	2230	7.7098	693	6.5410
2.25	0.8109	681	6.5236	4.2	1.4351	51900	10.8571	3350	8.1167	1230	7.1148
7.5	2.0149	1060	6.9660	6.3	1.8405	69700	11.1520	4580	8.4295	1760	7.4731
7.6	2.0281	11.4	2.4336	2.3	0.8329	14400	9.5750	227	5.4250	438	6.0822
26.4	3.2734	14.1	2.6462	3.7	1.3083	21900	9.9942	276	5.6204	566	6.3386
26.8	3.2884	93.1	4.5337	9.6	2.2618	27500	10.2219	1210	7.0984	2140	7.6686
22.8	3.1268	66.3	4.1942	12.1	2.4932	28200	10.2471	1320	7.1854	2840	7.9516
8.6	2.1518	13.7	2.6174	0.1	-2.3026	18500	9.8255	791	6.6733	183	5.2095
46.1	3.8308	14.8	2.6946	0.1	-2.3026	19700	9.8884	1040	6.9470	250	5.5215
53.8	3.9853	15	2.7081	0.66	-0.4155	19800	9.8934	1240	7.1229	374	5.9243
27.3	3.3069	21.7	3.0773	1.5	0.4055	18600	9.8309	1220	7.1066	675	6.5147
2.9	1.0647	14.8	2.6946	0.77	-0.2614	8860	9.0893	186	5.2257	200	5.2983
3.6	1.2809	51.5	3.9416	4.3	1.4586	19200	9.8627	686	6.5309	840	6.7334
4.3	1.4586	54.1	3.9908	5.4	1.6864	20500	9.9282	761	6.6346	1070	6.9754
6.9	1.9315	48	3.8712	5.6	1.7228	17900	9.7926	795	6.6783	1150	7.0475
1.8	0.5878	21.5	3.0681	5.2	1.6487	18300	9.8147	775	6.6529	1190	7.0817
2.3	0.8329	121	4.7958	6.1	1.8083	22200	10.0078	1620	7.3902	1250	7.1309
3	1.0986	133	4.8903	8.2	2.1041	27700	10.2292	1860	7.5283	1340	7.2004
4.6	1.5261	58.7	4.0724	3	1.0986	23100	10.0476	1450	7.2793	1330	7.1929
0.86	-0.1508	16.5	2.8034	3.3	1.1939	21100	9.9570	1080	6.9847	565	6.3368
0.99	-0.0101	20.2	3.0057	5.5	1.7047	21200	9.9618	1230	7.1148	964	6.8711
7.9	2.0669	20.4	3.0155	9.2	2.2192	21500	9.9758	1260	7.1389	1540	7.3395
0.5	-0.6931	26.8	3.2884	12.5	2.5257	23100	10.0476	1380	7.2298	2000	7.6009
0.5	-0.6931	6.8	1.9169	0.81	-0.2107	19400	9.8730	934	6.8395	160	5.0752
1.15	0.1398	11.1	2.4069	3.9	1.3610	21300	9.9665	1270	7.1468	530	6.2729
1.3	0.2624	11.5	2.4423	8	2.0794	21500	9.9758	1530	7.3330	1150	7.0475
0.495	-0.7032	18.9	2.9392	2	0.6931	19300	9.8679	1400	7.2442	668	6.5043
0.495	-0.7032	8.3	2.1163	1.4	0.3365	19500	9.8782	1200	7.0901	203	5.3132
0.5	-0.6931	12.8	2.5494	1.6	0.4700	20300	9.9184	1520	7.3265	257	5.5491
1.4	0.3365	20.7	3.0301	2.4	0.8755	18700	9.8363	1440	7.2724	815	6.7032
0.5	-0.6931	3.9	1.3610	0.105	-2.2538	11400	9.3414	354	5.8693	49.5	3.9020
0.55	-0.5978	4.4	1.4816	0.36	-1.0217	12300	9.4174	427	6.0568	67.5	4.2121
0.55	-0.5978	6.6	1.8871	5.4	1.6864	13500	9.5104	567	6.3404	591	6.3818
4.1	1.4110	5.5	1.7047	2.4	0.8755	14100	9.5539	603	6.4019	156	5.0499
0.49	-0.7133	3.1	1.1314	0.1	-2.3026	11400	9.3414	279	5.6312	37.6	3.6270
0.495	-0.7032	3.7	1.3083	0.1	-2.3026	11800	9.3759	285	5.6525	40.2	3.6939
0.5	-0.6931	3.8	1.3350	0.66	-0.4155	11900	9.3843	408	6.0113	76.2	4.3334

Silverton Public Recreational Exposure to Soil

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
1.7	0.5306	6.2	1.8245	3.7	1.3083	14500	9.5819	532	6.2766	185	5.2204
0.49	-0.7133	5.1	1.6292	0.1	-2.3026	13500	9.5104	645	6.4693	72.8	4.2877
0.495	-0.7032	7.3	1.9879	0.89	-0.1165	14600	9.5888	1010	6.9177	120	4.7875
1.2	0.1823	9.8	2.2824	2.6	0.9555	14700	9.5956	1160	7.0562	226	5.4205
8	2.0794	8	2.0794	3.2	1.1632	11500	9.3501	798	6.6821	285	5.6525
0.49	-0.7133	4.6	1.5261	0.1	-2.3026	10500	9.2591	527	6.2672	53.6	3.9815
0.5	-0.6931	5.3	1.6677	0.71	-0.3425	11100	9.3147	758	6.6307	91.7	4.5185
0.5	-0.6931	5.4	1.6864	1.7	0.5306	11900	9.3843	762	6.6359	136	4.9127
1.5	0.4055	6.5	1.8718	3.2	1.1632	10900	9.2965	663	6.4968	182	5.2040
0.5	-0.6931	7.1	1.9601	0.1	-2.3026	16900	9.7351	1470	7.2930	83.1	4.4200
0.8	-0.2231	7.6	2.0281	0.21	-1.5606	17200	9.7527	2080	7.6401	108	4.6821
1.7	0.5306	12.3	2.5096	1.4	0.3365	23300	10.0562	2360	7.7664	280	5.6348
5.6	1.7228	25.5	3.2387	4.8	1.5686	27800	10.2328	2640	7.8785	829	6.7202
0.485	-0.7236	4.8	1.5686	0.1	-2.3026	12000	9.3927	528	6.2691	46.2	3.8330
0.495	-0.7032	5.6	1.7228	0.1	-2.3026	13400	9.5030	652	6.4800	54.6	4.0000
0.65	-0.4308	6.8	1.9169	0.86	-0.1508	13500	9.5104	1010	6.9177	116	4.7536
1.5	0.4055	8.4	2.1282	2.9	1.0647	12100	9.4010	952	6.8586	206	5.3279
0.495	-0.7032	7.1	1.9601	1.4	0.3365	14200	9.5610	897	6.7991	218	5.3845
0.5	-0.6931	9.7	2.2721	1.7	0.5306	15600	9.6550	1180	7.0733	231	5.4424
1	0.0000	18.7	2.9285	3.7	1.3083	16800	9.7291	1190	7.0817	527	6.2672
1.2	0.1823	4.4	1.4816	3.9	1.3610	7420	8.9119	359	5.8833	860	6.7569
0.76	-0.2744	4.2	1.4351	0.095	-2.3539	12200	9.4092	261	5.5645	45.7	3.8221
1	0.0000	6.1	1.8083	0.45	-0.7985	12600	9.4415	307	5.7268	77.9	4.3554
3	1.0986	9.6	2.2618	1.2	0.1823	12600	9.4415	395	5.9789	145	4.9767
0.5	-0.6931	12.7	2.5416	1.6	0.4700	14800	9.6024	495	6.2046	214	5.3660
0.66	-0.4155	3.2	1.1632	0.54	-0.6162	5980	8.6962	372	5.9189	68.7	4.2297
0.88	-0.1278	4.4	1.4816	0.69	-0.3711	12100	9.4010	500	6.2146	97.3	4.5778
2.7	0.9933	6.3	1.8405	2	0.6931	16300	9.6989	890	6.7912	157	5.0562
1.5	0.4055	5.8	1.7579	0.96	-0.0408	14000	9.5468	582	6.3665	177	5.1761
1.6	0.4700	6.6	1.8871	1.1	0.0953	15300	9.6356	820	6.7093	76.1	4.3320
1.8	0.5878	7	1.9459	1.1	0.0953	16700	9.7232	849	6.7441	77.7	4.3529
2.9	1.0647	7.9	2.0669	1.1	0.0953	17500	9.7700	851	6.7464	96.5	4.5695
0.73	-0.3147	8.3	2.1163	1.9	0.6419	17900	9.7926	984	6.8916	141	4.9488
0.495	-0.7032	11.8	2.4681	5.3	1.6677	13300	9.4955	1110	7.0121	384	5.9506
1.5	0.4055	4	1.3863	1	0.0000	14600	9.5888	409	6.0137	58.8	4.0741
2	0.6931	4.4	1.4816	1.4	0.3365	15000	9.6158	473	6.1591	75.3	4.3215
0.5	-0.6931	4.6	1.5261	1.7	0.5306	16300	9.6989	618	6.4265	88.2	4.4796
1	0.0000	7.3	1.9879	2.1	0.7419	14400	9.5750	565	6.3368	136	4.9127
1.3	0.2624	7.5	2.0149	1.2	0.1823	17200	9.7527	1240	7.1229	107	4.6728
2	0.6931	7.8	2.0541	2.1	0.7419	17300	9.7585	1260	7.1389	115	4.7449
4	1.3863	8.4	2.1282	2.6	0.9555	19000	9.8522	1440	7.2724	138	4.9273
3	1.0986	10	2.3026	4.1	1.4110	14200	9.5610	988	6.8957	203	5.3132
4.5	1.5041	6.8	1.9169	1.8	0.5878	17000	9.7410	927	6.8320	151	5.0173
12.7	2.5416	7.5	2.0149	1.9	0.6419	17100	9.7468	1010	6.9177	159	5.0689
16.6	2.8094	7.8	2.0541	2	0.6931	17700	9.7813	1020	6.9276	168	5.1240
0.49	-0.7133	10.3	2.3321	3.9	1.3610	13300	9.4955	584	6.3699	232	5.4467
0.49	-0.7133	4.2	1.4351	0.03	-3.5066	15700	9.6614	126	4.8363	23.3	3.1485
0.49	-0.7133	4.2	1.4351	0.04	-3.2189	16000	9.6803	158	5.0626	27.9	3.3286
1.4	0.3365	5.3	1.6677	0.34	-1.0788	16400	9.7050	427	6.0568	63.9	4.1573
0.49	-0.7133	6.3	1.8405	0.98	-0.0202	14100	9.5539	616	6.4232	138	4.9273
0.5	-0.6931	6.3	1.8405	0.49	-0.7133	16700	9.7232	888	6.7890	84.3	4.4344
1.3	0.2624	7.3	1.9879	1.4	0.3365	17400	9.7642	996	6.9037	123	4.8122
0.5	-0.6931	7.9	2.0669	1.7	0.5306	19800	9.8934	1390	7.2371	151	5.0173
0.5	-0.6931	9.4	2.2407	2.7	0.9933	13600	9.5178	698	6.5482	178	5.1818
0.5	-0.6931	5.8	1.7579	0.79	-0.2357	11600	9.3588	772	6.6490	149	5.0039
1.9	0.6419	7.4	2.0015	2.3	0.8329	17300	9.7585	1260	7.1389	255	5.5413
0.485	-0.7236	8.7	2.1633	3.2	1.1632	18100	9.8037	1370	7.2226	261	5.5645
0.5	-0.6931	9.9	2.2925	5.8	1.7579	13700	9.5252	959	6.8659	304	5.7170
0.5	-0.6931	8.2	2.1041	1.3	0.2624	16000	9.6803	1490	7.3065	150	5.0106
6.4	1.8563	8.9	2.1861	1.4	0.3365	19100	9.8574	2110	7.6544	185	5.2204
0.49	-0.7133	15.6	2.7473	4.3	1.4586	20200	9.9134	3080	8.0327	432	6.0684
1.1	0.0953	15.4	2.7344	3.3	1.1939	24200	10.0941	1880	7.5390	412	6.0210
1.2	0.1823	21.6	3.0727	5.7	1.7405	27000	10.2036	2330	7.7536	791	6.6733
3.1	1.1314	49.8	3.9080	10.7	2.3702	42600	10.6596	5050	8.5271	1760	7.4731
0.5	-0.6931	16	2.7726	3.2	1.1632	12800	9.4572	1140	7.0388	448	6.1048
2.6	0.9555	3.9	1.3610	0.48	-0.7340	15700	9.6614	529	6.2710	72.3	4.2808
5.9	1.7750	5.1	1.6292	0.53	-0.6349	16200	9.6928	691	6.5381	74.5	4.3108
0.94	-0.0619	5.3	1.6677	0.97	-0.0305	16500	9.7111	755	6.6267	88.1	4.4785
2	0.6931	5.7	1.7405	1.8	0.5878	16700	9.7232	781	6.6606	138	4.9273
7	1.9459	6.9	1.9315	2.1	0.7419	16100	9.6866	755	6.6267	145	4.9767
0.72	-0.3285	7.1	1.9601	0.35	-1.0498	16000	9.6803	1190	7.0817	82.9	4.4176
1.9	0.6419	10.8	2.3795	0.77	-0.2614	19000	9.8522	1620	7.3902	106	4.6634
5.8	1.7579	12.8	2.5494	2.6	0.9555	22100	10.0033	3590	8.1859	251	5.5255
3.5	1.2528	5.8	1.7579	0.56	-0.5798	13300	9.4955	1260	7.1389	55.7	4.0200
0.82	-0.1985	9.3	2.2300	1.1	0.0953	19900	9.8985	1420	7.2584	115	4.7449
0.49	-0.7133	10.7	2.3702	1.7	0.5306	21100	9.9570	1720	7.4501	149	5.0039
3.8	1.3350	11.7	2.4596	4.3	1.4586	16900	9.7351	1260	7.1389	246	5.5053
0.495	-0.7032	11.8	2.4681	0.9	-0.1054	24900	10.1226	1870	7.5337	136	4.9127
6.2	1.8245	13.4	2.5953	1.3	0.2624	27000	10.2036	1970	7.5858	188	5.2364

Silverton Public Recreational Exposure to Soil

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
19.8	2.9857	14.3	2.6603	2.2	0.7885	27600	10.2256	2010	7.6059	242	5.4889
9.1	2.2083	20.7	3.0301	8.7	2.1633	25700	10.1542	1820	7.5066	968	6.8752
0.495	-0.7032	6.9	1.9315	0.93	-0.0726	16000	9.6803	612	6.4167	121	4.7958
0.5	-0.6931	9.5	2.2513	1.8	0.5878	20100	9.9085	1090	6.9939	238	5.4723
0.5	-0.6931	10.8	2.3795	3.8	1.3350	20200	9.9134	1360	7.2152	391	5.9687
0.325	-1.1239	11.7	2.4596	7.6	2.0281	16400	9.7050	1310	7.1778	487	6.1883
0.485	-0.7236	5.3	1.6677	1.1	0.0953	13500	9.5104	918	6.8222	112	4.7185
0.49	-0.7133	10.7	2.3702	4	1.3863	20000	9.9035	1460	7.2862	407	6.0088
0.5	-0.6931	15.5	2.7408	17.4	2.8565	21000	9.9523	1730	7.4559	790	6.6720
0.97	-0.0305	10.5	2.3514	13.5	2.6027	12000	9.3927	1320	7.1854	592	6.3835
1.4	0.3365	5.3	1.6677	0.79	-0.2357	14900	9.6091	653	6.4816	100	4.6052
1.5	0.4055	9.3	2.2300	3.3	1.1939	17200	9.7527	1270	7.1468	256	5.5452
1.7	0.5306	12.4	2.5177	6	1.7918	17800	9.7870	1340	7.2004	373	5.9216
1.3	0.2624	14.7	2.6878	5.7	1.7405	14800	9.6024	1090	6.9939	374	5.9243
1.4	0.3365	9.6	2.2618	1.3	0.2624	18600	9.8309	1590	7.3715	148	4.9972
1.4	0.3365	10.6	2.3609	2	0.6931	19600	9.8833	1980	7.5909	216	5.3753
8.9	2.1861	11.6	2.4510	3.3	1.1939	22400	10.0168	2380	7.7749	346	5.8464
3.8	1.3350	10.2	2.3224	3.4	1.2238	13800	9.5324	1160	7.0562	351	5.8608
0.49	-0.7133	4.3	1.4586	0.22	-1.5141	15100	9.6225	482	6.1779	105	4.6540
2.4	0.8755	4.4	1.4816	0.73	-0.3147	16200	9.6928	757	6.6294	113	4.7274
2.8	1.0296	6	1.7918	1.7	0.5306	16300	9.6989	854	6.7499	269	5.5947
3.6	1.2809	16.3	2.7912	3.9	1.3610	21100	9.9570	1410	7.2513	622	6.4329
0.49	-0.7133	4.8	1.5686	0.1	-2.3026	11200	9.3237	472	6.1570	52.8	3.9665
3.5	1.2528	20.9	3.0397	2.4	0.8755	26000	10.1659	2050	7.6256	637	6.4568
9.6	2.2618	51.7	3.9455	7.5	2.0149	43300	10.6759	4130	8.3260	1850	7.5229
14.2	2.6532	12.7	2.5416	3.4	1.2238	8680	9.0688	1140	7.0388	418	6.0355
6.8	1.9169	4	1.3863	0.29	-1.2379	13400	9.5030	510	6.2344	92.2	4.5240
8.7	2.1633	6	1.7918	0.3	-1.2040	14300	9.5680	545	6.3008	93.5	4.5380
18.3	2.9069	9.5	2.2513	0.49	-0.7133	16100	9.6866	604	6.4036	109	4.6913
4.1	1.4110	5.9	1.7750	1.7	0.5306	15300	9.6356	675	6.5147	128	4.8520
0.5	-0.6931	4.1	1.4110	0.1	-2.3026	13100	9.4804	412	6.0210	64.2	4.1620
0.5	-0.6931	4.2	1.4351	0.32	-1.1394	14400	9.5750	500	6.2146	80.9	4.3932
3.5	1.2528	4.2	1.4351	0.42	-0.8675	14400	9.5750	508	6.2305	96.3	4.5675
5.8	1.7579	4.3	1.4586	1.4	0.3365	15200	9.6291	634	6.4520	107	4.6728
0.49	-0.7133	9.1	2.2083	2.1	0.7419	14800	9.6024	713	6.5695	190	5.2470
0.5	-0.6931	13.9	2.6319	2.3	0.8329	14900	9.6091	955	6.8617	204	5.3181
6	1.7918	25.4	3.2347	3.6	1.2809	16900	9.7351	1070	6.9754	270	5.5984
2.1	0.7419	49	3.8918	0.77	-0.2614	28000	10.2400	785	6.6657	120	4.7875
0.55	-0.5978	4	1.3863	1.2	0.1823	11700	9.3673	213	5.3613	213	5.3613
1.6	0.4700	6.7	1.9021	2.5	0.9163	13300	9.4955	404	6.0014	364	5.8972
3.7	1.3083	12.6	2.5337	8.9	2.1861	17200	9.7527	1870	7.5337	1240	7.1229
0.5	-0.6931	13	2.5649	8.2	2.1041	18600	9.8309	816	6.7044	648	6.4739
0.485	-0.7236	4.8	1.5686	0.33	-1.1087	13900	9.5396	220	5.3936	59.8	4.0910
0.49	-0.7133	5.8	1.7579	2	0.6931	13900	9.5396	532	6.2766	340	5.8289
1.1	0.0953	6.4	1.8563	2.4	0.8755	15500	9.6486	721	6.5806	471	6.1549
1.4	0.3365	8.8	2.1748	6.5	1.8718	18400	9.8201	854	6.7499	655	6.4846
0.485	-0.7236	3.7	1.3083	0.39	-0.9416	12500	9.4335	219	5.3891	69.5	4.2413
0.5	-0.6931	10.1	2.3125	6	1.7918	15900	9.6741	766	6.6412	967	6.8742
0.5	-0.6931	19.7	2.9806	11.9	2.4765	21100	9.9570	1920	7.5601	1870	7.5337
1	0.0000	15.4	2.7344	10.7	2.3702	22200	10.0078	1870	7.5337	1420	7.2584
1.7	0.5306	12.4	2.5177	2.8	1.0296	18600	9.8309	1330	7.1929	538	6.2879
0.485	-0.7236	14.6	2.6810	6.6	1.8871	19000	9.8522	2200	7.6962	994	6.9017
0.49	-0.7133	24.4	3.1946	7.1	1.9601	22800	10.0345	2690	7.8973	1160	7.0562
0.5	-0.6931	8.4	2.1282	7.2	1.9741	19800	9.8934	887	6.7878	950	6.8565
0.495	-0.7032	1.8	0.5878	0.1	-2.3026	5360	8.5867	122	4.8040	80.9	4.3932
0.485	-0.7236	5.3	1.6677	0.48	-0.7340	9130	9.1193	287	5.6595	91	4.5109
0.495	-0.7032	5.4	1.6864	3.9	1.3610	16800	9.7291	684	6.5280	536	6.2841
0.5	-0.6931	8.7	2.1633	7.5	2.0149	18900	9.8469	806	6.6921	907	6.8101
1.4	0.3365	4.1	1.4110	0.1	-2.3026	16200	9.6928	266	5.5835	89.4	4.4931
0.335	-1.0936	5.1	1.6292	0.23	-1.4697	17500	9.7700	282	5.6419	159	5.0689
0.485	-0.7236	7.9	2.0669	5.4	1.6864	18400	9.8201	809	6.6958	832	6.7238
1.3	0.2624	7.1	1.9601	4	1.3863	10900	9.2965	358	5.8805	463	6.1377
0.495	-0.7032	3	1.0986	0.11	-2.2073	11300	9.3326	286	5.6560	65.6	4.1836
0.67	-0.4005	5.6	1.7228	2.3	0.8329	15500	9.6486	481	6.1759	405	6.0039
0.92	-0.0834	8.2	2.1041	6.5	1.8718	17200	9.7527	824	6.7142	893	6.7946
0.99	-0.0101	4.2	1.4351	0.78	-0.2485	11500	9.3501	563	6.3333	83.1	4.4200
0.93	-0.0726	8.1	2.0919	2.2	0.7885	14800	9.6024	1140	7.0388	159	5.0689
0.8	-0.2231	9.1	2.2083	2.5	0.9163	15900	9.6741	1500	7.3132	179	5.1874
1.7	0.5306	9.9	2.2925	2.7	0.9933	16800	9.7291	1570	7.3588	188	5.2364
0.83	-0.1863	9.4	2.2407	21.6	3.0727	13100	9.4804	772	6.6490	259	5.5568
0.87	-0.1393	7.8	2.0541	0.29	-1.2379	14700	9.5956	1060	6.9660	106	4.6634
1.1	0.0953	8.1	2.0919	1.3	0.2624	16100	9.6866	1080	6.9847	160	5.0752
2.1	0.7419	8.2	2.1041	2.3	0.8329	16700	9.7232	1520	7.3265	185	5.2204
1.5	0.4055	10.7	2.3702	2.9	1.0647	19000	9.8522	1760	7.4731	248	5.5134
0.82	-0.1985	7.6	2.0281	9.4	2.2407	13400	9.5030	735	6.5999	200	5.2983
0.88	-0.1278	5.7	1.7405	0.49	-0.7133	12600	9.4415	890	6.7912	106	4.6634
0.94	-0.0619	6.6	1.8871	0.57	-0.5621	15400	9.6421	928	6.8330	108	4.6821
1.9	0.6419	10.3	2.3321	0.73	-0.3147	16100	9.6866	950	6.8565	124	4.8203
1	0.0000	7.6	2.0281	3.6	1.2809	11500	9.3501	588	6.3767	218	5.3845

Silverton Public Recreational Exposure to Soil

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
1.7	0.5306	5	1.6094	0.1	-2.3026	10400	9.2496	299	5.7004	36.9	3.6082
1.7	0.5306	5.3	1.6677	0.1	-2.3026	13800	9.5324	458	6.1269	55.3	4.0128
0.92	-0.0834	7.3	1.9879	0.49	-0.7133	14300	9.5680	602	6.4003	80.9	4.3932
1.3	0.2624	8.5	2.1401	2.9	1.0647	13800	9.5324	587	6.3750	166	5.1120
2	0.6931	5.6	1.7228	0.03	-3.5066	14600	9.5888	720	6.5793	94.2	4.5454
3.8	1.3350	6.2	1.8245	0.73	-0.3147	18500	9.8255	935	6.8405	121	4.7958
43.2	3.7658	7.9	2.0669	1.1	0.0953	18600	9.8309	1190	7.0817	135	4.9053
0.5	-0.6931	4.6	1.5261	1.5	0.4055	10400	9.2496	460	6.1312	107	4.6728
0.5	-0.6931	7	1.9459	0.03	-3.5066	15400	9.6421	943	6.8491	90.8	4.5087
1	0.0000	7.5	2.0149	0.21	-1.5606	16500	9.7111	1040	6.9470	102	4.6250
1.6	0.4700	8	2.0794	0.94	-0.0619	16900	9.7351	1100	7.0031	152	5.0239
0.5	-0.6931	8.3	2.1163	2	0.6931	20100	9.9085	1360	7.2152	156	5.0499
0.5	-0.6931	7.4	2.0015	1	0.0000	16400	9.7050	619	6.4281	110	4.7005
1.9	0.6419	8.1	2.0919	0.03	-3.5066	17500	9.7700	595	6.3886	62.7	4.1384
2.4	0.8755	8.2	2.1041	0.03	-3.5066	18800	9.8416	722	6.5820	68.4	4.2254
0.49	-0.7133	13	2.5649	0.5	-0.6931	21000	9.9523	1170	7.0648	143	4.9628
1	0.0000	7.4	2.0015	0.17	-1.7720	17300	9.7585	582	6.3665	87.1	4.4671
1.5	0.4055	8	2.0794	0.03	-3.5066	16900	9.7351	365	5.8999	78.4	4.3618
0.5	-0.6931	8.1	2.0919	0.34	-1.0788	17500	9.7700	683	6.5265	97	4.5747
0.98	-0.0202	9.5	2.2513	1.6	0.4700	19500	9.8782	1140	7.0388	185	5.2204
1.2	0.1823	10.1	2.3125	4.5	1.5041	16800	9.7291	1590	7.3715	356	5.8749
1.8	0.5878	4.8	1.5686	0.03	-3.5066	14500	9.5819	211	5.3519	38.3	3.6454
2.9	1.0647	5	1.6094	0.14	-1.9661	16500	9.7111	270	5.5984	46.3	3.8351
1.4	0.3365	5.4	1.6864	0.14	-1.9661	17000	9.7410	430	6.0638	80.8	4.3920
2.2	0.7885	6.5	1.8718	2.4	0.8755	14400	9.5750	498	6.2106	210	5.3471
3.7	1.3083	6.6	1.8871	0.24	-1.4271	15700	9.6614	738	6.6039	78.1	4.3580
20.9	3.0397	7.5	2.0149	1	0.0000	16200	9.6928	1250	7.1309	143	4.9628
2.9	1.0647	8.3	2.1163	2.1	0.7419	16500	9.7111	1310	7.1778	166	5.1120
3.4	1.2238	7.3	1.9879	7.7	2.0412	12200	9.4092	698	6.5482	208	5.3375
4.1	1.4110	6.3	1.8405	1.1	0.0953	16400	9.7050	560	6.3279	86.4	4.4590
8.5	2.1401	7.8	2.0541	1.5	0.4055	17200	9.7527	757	6.6294	105	4.6540
4.1	1.4110	8.4	2.1282	1.9	0.6419	17600	9.7757	770	6.6464	149	5.0039
21.6	3.0727	9.1	2.2083	5.5	1.7047	14900	9.6091	975	6.8824	270	5.5984
25.6	3.2426	8.4	2.1282	0.37	-0.9943	16500	9.7111	1270	7.1468	179	5.1874
3.6	1.2809	9.7	2.2721	2	0.6931	17200	9.7527	1670	7.4206	187	5.2311
2.6	0.9555	10	2.3026	2.5	0.9163	24200	10.0941	2000	7.6009	218	5.3845
3.1	1.1314	8	2.0794	6.2	1.8245	13700	9.5252	975	6.8824	247	5.5094
3.7	1.3083	6.4	1.8563	0.03	-3.5066	16400	9.7050	736	6.6012	68.8	4.2312
3.5	1.2528	6.6	1.8871	0.09	-2.4079	17100	9.7468	1080	6.9847	79.6	4.3770
0.87	-0.1393	8.3	2.1163	1.2	0.1823	19600	9.8833	1590	7.3715	109	4.6913
1.6	0.4700	6.6	1.8871	0.72	-0.3285	16600	9.7172	577	6.3578	94.5	4.5486
3.8	1.3350	9.9	2.2925	1.4	0.3365	16600	9.7172	1400	7.2442	218	5.3845
3.1	1.1314	12.5	2.5257	2.2	0.7885	17600	9.7757	1810	7.5011	230	5.4381
1.1	0.0953	13.8	2.6247	3.2	1.1632	18600	9.8309	1820	7.5066	316	5.7557
1.3	0.2624	42.2	3.7424	17.2	2.8449	18800	9.8416	1800	7.4955	2030	7.6158
2.7	0.9933	6.4	1.8563	0.55	-0.5978	14700	9.5956	830	6.7214	78.9	4.3682
3	1.0986	6.7	1.9021	0.61	-0.4943	15100	9.6225	904	6.8068	79	4.3694
0.5	-0.6931	6.7	1.9021	1.4	0.3365	16100	9.6866	1060	6.9660	120	4.7875
0.5	-0.6931	6.7	1.9021	2.3	0.8329	14900	9.6091	535	6.2823	125	4.8283
1.4	0.3365	5.2	1.6487	0.1	-2.3026	12100	9.4010	473	6.1591	45.1	3.8089
2.2	0.7885	6.9	1.9315	0.71	-0.3425	16100	9.6866	896	6.7979	109	4.6913
2.2	0.7885	8.2	2.1041	0.92	-0.0834	16200	9.6928	1210	7.0984	143	4.9628
1.8	0.5878	9.2	2.2192	3.6	1.2809	15400	9.6421	819	6.7081	272	5.6058
2.8	1.0296	7.2	1.9741	0.81	-0.2107	14800	9.6024	689	6.5352	120	4.7875
32.8	3.4904	7.3	1.9879	1.2	0.1823	14900	9.6091	712	6.5681	150	5.0106
0.49	-0.7133	8.5	2.1401	2.6	0.9555	17000	9.7410	1080	6.9847	158	5.0626
0.5	-0.6931	9.1	2.2083	5.1	1.6292	15100	9.6225	522	6.2577	131	4.8752
0.5	-0.6931	7.9	2.0669	0.65	-0.4308	15900	9.6741	1070	6.9754	128	4.8520
0.5	-0.6931	8	2.0794	1.8	0.5878	17800	9.7870	1200	7.0901	180	5.1930
1.3	0.2624	9.4	2.2407	2.4	0.8755	18600	9.8309	1450	7.2793	212	5.3566
1	0.0000	12	2.4849	54.4	3.9964	12700	9.4494	605	6.4052	268	5.5910
1.5	0.4055	10.9	2.3888	0.54	-0.6162	18400	9.8201	912	6.8156	168	5.1240
3.3	1.1939	12.7	2.5416	1.4	0.3365	21500	9.9758	1370	7.2226	274	5.6131
0.5	-0.6931	14.9	2.7014	2.9	1.0647	23400	10.0605	1650	7.4085	387	5.9584
1.2	0.1823	31.2	3.4404	7.3	1.9879	23700	10.0732	1670	7.4206	1100	7.0031
1.4	0.3365	17.1	2.8391	3.5	1.2528	22400	10.0168	545	6.3008	433	6.0707
1.8	0.5878	19.1	2.9497	3.6	1.2809	26800	10.1962	876	6.7754	436	6.0776
3.1	1.1314	19.2	2.9549	7.9	2.0669	29700	10.2989	927	6.8320	728	6.5903
1.6	0.4700	19.9	2.9907	7	1.9459	15900	9.6741	906	6.8090	1030	6.9373
1.9	0.6419	16.3	2.7912	5.2	1.6487	28200	10.2471	1240	7.1229	519	6.2519
3.3	1.1939	30.4	3.4144	19.5	2.9704	46400	10.7451	3410	8.1345	3370	8.1227
2.8	1.0296	32.8	3.4904	31.9	3.4626	86000	11.3621	10000	9.2103	4450	8.4007
3.6	1.2809	12.9	2.5572	6.6	1.8871	26000	10.1659	1110	7.0121	346	5.8464
1.1	0.0953	17.2	2.8449	4.7	1.5476	19000	9.8522	978	6.8855	509	6.2324
1.5	0.4055	19.2	2.9549	5.1	1.6292	20300	9.9184	1100	7.0031	690	6.5367
1.7	0.5306	21.6	3.0727	6.6	1.8871	24400	10.1023	1250	7.1309	841	6.7346
3.6	1.2809	18.6	2.9232	2.3	0.8329	21500	9.9758	840	6.7334	313	5.7462
1.1	0.0953	12.5	2.5257	0.72	-0.3285	16000	9.6803	692	6.5396	182	5.2040
1.7	0.5306	17.8	2.8792	2.7	0.9933	22000	9.9988	703	6.5554	205	5.3230

Silverton Public Recreational Exposure to Soil

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
2.4	0.8755	18.5	2.9178	6.5	1.8718	24600	10.1105	964	6.8711	1080	6.9847
1.4	0.3365	12.5	2.5257	7.8	2.0541	14100	9.5539	568	6.3421	585	6.3716
1.5	0.4055	6.8	1.9169	0.33	-1.1087	14800	9.6024	557	6.3226	132	4.8828
1.7	0.5306	10.3	2.3321	0.84	-0.1744	21600	9.9804	900	6.8024	240	5.4806
6.7	1.9021	17.4	2.8565	3.1	1.1314	22400	10.0168	923	6.8276	504	6.2226
11.5	2.4423	13.3	2.5878	4.3	1.4586	17000	9.7410	712	6.5681	567	6.3404
0.93	-0.0726	6.7	1.9021	0.1	-2.3026	14600	9.5888	392	5.9713	78.7	4.3656
1.6	0.4700	7.3	1.9879	0.1	-2.3026	15800	9.6678	399	5.9890	112	4.7185
2.1	0.7419	7.7	2.0412	1.5	0.4055	18000	9.7981	416	6.0307	319	5.7652
3.4	1.2238	8.8	2.1748	1.7	0.5306	18700	9.8363	463	6.1377	361	5.8889
0.76	-0.2744	10.5	2.3514	3.8	1.3350	18100	9.8037	560	6.3279	359	5.8833
1.1	0.0953	14.8	2.6946	0.62	-0.4780	21400	9.9711	357	5.8777	173	5.1533
2.3	0.8329	20.4	3.0155	0.68	-0.3857	22900	10.0389	604	6.4036	189	5.2417
5.3	1.6677	32.4	3.4782	2.3	0.8329	25900	10.1620	730	6.5930	204	5.3181
1.3	0.2624	3.5	1.2528	1.6	0.4700	7890	8.9734	150	5.0106	151	5.0173
3.4	1.2238	6.4	1.8563	0.1	-2.3026	15000	9.6158	418	6.0355	55.2	4.0110
3.5	1.2528	7.7	2.0412	0.105	-2.2538	16000	9.6803	526	6.2653	64.7	4.1698
5.4	1.6864	8.9	2.1861	1.1	0.0953	16200	9.6928	602	6.4003	149	5.0039
2	0.6931	9	2.1972	2.5	0.9163	14300	9.5680	655	6.4846	209	5.3423
2.2	0.7885	6.4	1.8563	1	0.0000	14500	9.5819	374	5.9243	123	4.8122
4.5	1.5041	13.6	2.6101	2.9	1.0647	14900	9.6091	387	5.9584	321	5.7714
1.6	0.4700	19.4	2.9653	4.3	1.4586	26700	10.1924	763	6.6373	504	6.2226
3.5	1.2528	22	3.0910	0.73	-0.3147	21800	9.9897	728	6.5903	407	6.0088
3.7	1.3083	6	1.7918	0.29	-1.2379	14000	9.5468	570	6.3456	35.8	3.5779
12.9	2.5572	6.6	1.8871	0.99	-0.0101	16100	9.6866	676	6.5162	74.3	4.3081
5.7	1.7405	11.8	2.4681	2.2	0.7885	21400	9.9711	707	6.5610	169	5.1299
0.495	-0.7032	13	2.5649	4.7	1.5476	17800	9.7870	506	6.2265	257	5.5491
2.6	0.9555	11.2	2.4159	2.6	0.9555	19200	9.8627	674	6.5132	367	5.9054
6.8	1.9169	15.1	2.7147	3	1.0986	20700	9.9379	953	6.8596	383	5.9480
1.3	0.2624	16.4	2.7973	3.3	1.1939	24000	10.0858	1160	7.0562	399	5.9890
2	0.6931	9.2	2.2192	5.8	1.7579	14800	9.6024	491	6.1964	408	6.0113
4.2	1.4351	11.8	2.4681	5.9	1.7750	15400	9.6421	786	6.6670	459	6.1291
12.8	2.5494	8.6	2.1518	1.1	0.0953	17500	9.7700	515	6.2442	114	4.7362
15.3	2.7279	9.3	2.2300	1.2	0.1823	18100	9.8037	691	6.5381	129	4.8598
6.4	1.8563	10.2	2.3224	3	1.0986	18900	9.8469	746	6.6147	271	5.6021
14.5	2.6741	12.3	2.5096	5.4	1.6864	17900	9.7926	749	6.6187	297	5.6937
20.8	3.0350	7.4	2.0015	1.2	0.1823	13200	9.4880	224	5.4116	126	4.8363
5.1	1.6292	7.7	2.0412	2	0.6931	16900	9.7351	306	5.7236	248	5.5134
2.8	1.0296	17.2	2.8449	7	1.9459	25200	10.1346	605	6.4052	982	6.8896
7.2	1.9741	2.8	1.0296	3.3	1.1939	6370	8.7594	125	4.8283	445	6.0981
7.7	2.0412	11	2.3979	0.56	-0.5798	22600	10.0257	736	6.6012	225	5.4161
3.9	1.3610	12	2.4849	0.96	-0.0408	23000	10.0432	891	6.7923	324	5.7807
0.5	-0.6931	23.5	3.1570	6.8	1.9169	26400	10.1811	1800	7.4955	991	6.8987
1.5	0.4055	35.3	3.5639	10	2.3026	29900	10.3056	2910	7.9759	1290	7.1624
2.1	0.7419	18.6	2.9232	0.65	-0.4308	23200	10.0519	497	6.2086	240	5.4806
4.9	1.5892	21.6	3.0727	1.1	0.0953	27300	10.2146	586	6.3733	447	6.1026
2.3	0.8329	25.1	3.2229	1.8	0.5878	41800	10.6407	688	6.5338	540	6.2916
2.35	0.8544	16.7	2.8154	6.4	1.8563	16500	9.7111	550	6.3099	364	5.8972
11.8	2.4681	6.8	1.9169	0.83	-0.1863	14400	9.5750	437	6.0799	101	4.6151
4.5	1.5041	7.1	1.9601	1.3	0.2624	15600	9.6550	440	6.0868	138	4.9273
0.48	-0.7340	9.1	2.2083	1.4	0.3365	19500	9.8782	538	6.2879	280	5.6348
1.4	0.3365	12.1	2.4932	5.5	1.7047	18300	9.8147	665	6.4998	529	6.2710
0.8	-0.2231	9.1	2.2083	1.7	0.5306	18400	9.8201	591	6.3818	239	5.4765
1.5	0.4055	13.3	2.5878	4	1.3863	19800	9.8934	647	6.4723	719	6.5779
0.94	-0.0619	14.2	2.6532	4.5	1.5041	20400	9.9233	948	6.8544	818	6.7069
1.2	0.1823	13.6	2.6101	4.7	1.5476	17100	9.7468	558	6.3244	676	6.5162
1.3	0.2624	12.1	2.4932	1.4	0.3365	18800	9.8416	733	6.5971	380	5.9402
1.3	0.2624	12.6	2.5337	3.2	1.1632	19900	9.8985	986	6.8937	598	6.3936
2.7	0.9933	19.8	2.9857	5.2	1.6487	22000	9.9988	1270	7.1468	798	6.6821
1.7	0.5306	11.2	2.4159	2.6	0.9555	18000	9.7981	728	6.5903	445	6.0981
3.3	1.1939	12.5	2.5257	2.4	0.8755	22600	10.0257	1400	7.2442	429	6.0615
3.8	1.3350	13.6	2.6101	4.4	1.4816	25300	10.1386	1600	7.3778	642	6.4646
1.6	0.4700	40.8	3.7087	6.5	1.8718	29100	10.2785	2000	7.6009	1190	7.0817
1.9	0.6419	18.9	2.9392	5	1.6094	17800	9.7870	1110	7.0121	756	6.6280
1.9	0.6419	20.2	3.0057	0.1	-2.3026	23400	10.0605	668	6.5043	96.9	4.5737
2.1	0.7419	20.3	3.0106	1.4	0.3365	25200	10.1346	694	6.5425	245	5.5013
4.4	1.4816	20.8	3.0350	2.9	1.0647	27300	10.2146	785	6.6657	539	6.2897
4.6	1.5261	15.3	2.7279	2.6	0.9555	16600	9.7172	485	6.1841	328	5.7930
6.1	1.8083	9.1	2.2083	0.64	-0.4463	18300	9.8147	635	6.4536	238	5.4723
6.4	1.8563	20.3	3.0106	5.4	1.6864	20700	9.9379	639	6.4599	710	6.5653
4.3	1.4586	29.2	3.3742	7.5	2.0149	29000	10.2751	1550	7.3460	1500	7.3132
2.8	1.0296	21.8	3.0819	7.5	2.0149	22400	10.0168	986	6.8937	1290	7.1624
5.2	1.6487	22.8	3.1268	6.2	1.8245	24200	10.0941	817	6.7056	1620	7.3902
8.3	2.1163	36.9	3.6082	12.7	2.5416	26700	10.1924	1320	7.1854	2810	7.9409
10.5	2.3514	50.4	3.9200	14.2	2.6532	28300	10.2506	2010	7.6059	3010	8.0097
0.5	-0.6931	29	3.3673	5.3	1.6677	22000	9.9988	620	6.4297	737	6.6026
0.55	-0.5978	9.7	2.2721	0.44	-0.8210	16900	9.7351	175	5.1648	138	4.9273
0.55	-0.5978	12	2.4849	0.55	-0.5978	19500	9.8782	184	5.2149	198	5.2883
1.9	0.6419	12.1	2.4932	0.87	-0.1393	20200	9.9134	201	5.3033	232	5.4467

Silverton Public Recreational Exposure to Soil

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
0.495	-0.7032	9.9	2.2925	2.6	0.9555	15000	9.6158	312	5.7430	328	5.7930
2.1	0.7419	9.2	2.2192	0.1	-2.3026	18700	9.8363	421	6.0426	144	4.9698
2.2	0.7885	9.4	2.2407	0.1	-2.3026	19200	9.8627	453	6.1159	156	5.0499
1.6	0.4700	10	2.3026	0.6	-0.5108	20400	9.9233	611	6.4151	232	5.4467
18.3	2.9069	12.3	2.5096	2.4	0.8755	17300	9.7585	392	5.9713	378	5.9349
22.3	3.1046	15.3	2.7279	3.3	1.1939	21200	9.9618	536	6.2841	706	6.5596
28.4	3.3464	17.7	2.8736	4.8	1.5686	23800	10.0774	670	6.5073	939	6.8448
20.7	3.0301	24.7	3.2068	6	1.7918	25500	10.1464	761	6.6346	1490	7.3065
11.1	2.4069	15.5	2.7408	12.3	2.5096	17400	9.7642	823	6.7130	3280	8.0956
22	3.0910	7.7	2.0412	0.095	-2.3539	14800	9.6024	528	6.2691	71.8	4.2739
25.1	3.2229	9	2.1972	0.48	-0.7340	16600	9.7172	534	6.2804	182	5.2040
19.1	2.9497	9.9	2.2925	1	0.0000	19200	9.8627	640	6.4615	316	5.7557
0.94	-0.0619	13.7	2.6174	2.5	0.9163	19000	9.8522	346	5.8464	387	5.9584
1.7	0.5306	7.4	2.0015	0.42	-0.8675	15700	9.6614	498	6.2106	68.9	4.2327
6.6	1.8871	7.7	2.0412	0.49	-0.7133	15800	9.6678	521	6.2558	115	4.7449
10.1	2.3125	8.7	2.1633	0.77	-0.2614	16600	9.7172	537	6.2860	133	4.8903
13.5	2.6027	9.1	2.2083	1.7	0.5306	16800	9.7291	553	6.3154	258	5.5530
14.6	2.6810	11.9	2.4765	4	1.3863	18300	9.8147	754	6.6254	350	5.8579
15.4	2.7344	14.6	2.6810	1.7	0.5306	24000	10.0858	751	6.6214	221	5.3982
18.7	2.9285	17.9	2.8848	2.3	0.8329	26900	10.1999	789	6.6708	276	5.6204
4.7	1.5476	18.1	2.8959	3.2	1.1632	28100	10.2435	1280	7.1546	566	6.3386
4.8	1.5686	16.8	2.8214	2.3	0.8329	22500	10.0213	643	6.4661	196	5.2781
38.1	3.6402	11.3	2.4248	1.8	0.5878	22200	10.0078	494	6.2025	220	5.3936
26.2	3.2658	17.7	2.8736	2.6	0.9555	22300	10.0123	667	6.5028	283	5.6454
		19.6	2.9755	2.8	1.0296	24500	10.1064	994	6.9017	358	5.8805
		17	2.8332	5.2	1.6487	17000	9.7410	608	6.4102	368	5.9081
		36.2	3.5891	3.4	1.2238	23500	10.0648	744	6.6120	488	6.1903
		43.2	3.7658	7.6	2.0281	29300	10.2853	772	6.6490	1140	7.0388
		43.5	3.7728	8.1	2.0919	32400	10.3859	888	6.7890	1220	7.1066
		35.5	3.5695	4.2	1.4351	19700	9.8884	647	6.4723	414	6.0259
		11.7	2.4596	2.8	1.0296	17800	9.7870	329	5.7961	323	5.7777
		14.7	2.6878	3.2	1.1632	24100	10.0900	404	6.0014	455	6.1203
		25.9	3.2542	6.9	1.9315	24200	10.0941	847	6.7417	1050	6.9565
		23.6	3.1612	17.3	2.8507	16700	9.7232	1050	6.9565	2100	7.6497
		6.5	1.8718	0.1	-2.3026	15300	9.6356	441	6.0890	45.8	3.8243
		6.5	1.8718	0.105	-2.2538	15400	9.6421	642	6.4646	50.5	3.9220
		6.6	1.8871	0.29	-1.2379	16700	9.7232	693	6.5410	80.4	4.3870
		6.3	1.8405	2	0.6931	10900	9.2965	222	5.4027	170	5.1358
		6	1.7918	0.49	-0.7133	10800	9.2873	407	6.0088	202	5.3083
		16	2.7726	0.84	-0.1744	23200	10.0519	609	6.4118	245	5.5013
		17.3	2.8507	1.2	0.1823	25100	10.1306	656	6.4862	263	5.5722
		11.3	2.4248	1.8	0.5878	18200	9.8092	466	6.1442	199	5.2933
		17.2	2.8449	7.7	2.0412	20600	9.9330	735	6.5999	1990	7.5959
		25	3.2189	17.8	2.8792	30800	10.3353	1070	6.9754	3720	8.2215
		34.5	3.5410	32.9	3.4935	31200	10.3482	1720	7.4501	7100	8.8679
		38.3	3.6454	47.4	3.8586	27400	10.2183	2630	7.8747	10600	9.2686
		32.8	3.4904	48.3	3.8774	27100	10.2073	1620	7.3902	9920	9.2023
		41.2	3.7184	68.4	4.2254	28400	10.2541	2460	7.8079	11000	9.3057
		52.2	3.9551	81	4.3944	33600	10.4223	2730	7.9121	12100	9.4010
		41.7	3.7305	90.8	4.5087	33600	10.4223	2470	7.8120	18000	9.7981
		9.9	2.2925	1.4	0.3365	22500	10.0213	394	5.9764	276	5.6204
		10.2	2.3224	4	1.3863	25300	10.1386	500	6.2146	488	6.1903
		17.2	2.8449	16.7	2.8154	27500	10.2219	1040	6.9470	2440	7.7998
		20.9	3.0397	23	3.1355	19900	9.8985	1180	7.0733	3210	8.0740
		34	3.5264	18.9	2.9392	21900	9.9942	1380	7.2298	2570	7.8517
		49.2	3.8959	24.5	3.1987	23300	10.0562	1480	7.2998	3540	8.1719
		71.3	4.2669	35.5	3.5695	26200	10.1735	1560	7.3524	5110	8.5390
		73.6	4.2986	17.7	2.8736	21000	9.9523	1690	7.4325	2350	7.7622
		23.6	3.1612	9.9	2.2925	26800	10.1962	836	6.7286	1580	7.3652
		27.6	3.3178	10.1	2.3125	28400	10.2541	906	6.8090	1590	7.3715
		30.6	3.4210	53.1	3.9722	31500	10.3577	1340	7.2004	7960	8.9822
		49	3.8918	64.9	4.1728	29800	10.3023	3610	8.1915	15900	9.6741
Antimony		**Arsenic**		**Cadmium**		**Iron**		**Manganese**		**Zinc**	
Average	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**
5.7239	0.7339	23.1793	2.4925	3.9923	0.4584	19535.4797	9.8288	1038.7935	6.7668	633.4094	5.6621
Std Dev	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**
16.603	1.199	71.144	0.817	8.148	1.530	7374.056	0.307	757.704	0.592	1477.588	1.117
Count	**Std Dev^2**	**Count**	**Std Dev^2**	**Count**	**Std Dev^2**	**Count**	**Std Dev^2**	**Count**	**Std Dev^2**	**Count**	**Std Dev^2**
577	1.44	615	0.67	615	2.34	615	0.09	615	0.35	615	1.25
Max	**(Std Dev^2)/2**	**Max**	**(Std Dev^2)/2**	**Max**	**(Std Dev^2)/2**	**Max**	**(Std Dev^2)/2**	**Max**	**(Std Dev^2)/2**	**Max**	**(Std Dev^2)/2**
233	0.7193	1060	0.3337	90.8	1.171	86000	0.0471	10000	0.1754	18000	0.6243
Min	1.45	**Min**	2.83	**Min**	1.63	**Min**	9.88	**Min**	6.94	**Min**	6.29
0.325	**$Mean_{lognormal}$**	1.8	**$Mean_{lognormal}$**	0.03	**$Mean_{lognormal}$**	5360	**$Mean_{lognormal}$**	122	**$Mean_{lognormal}$**	19.8	**$Mean_{lognormal}$**
Count^0.5	4.2767	**Count^0.5**	16.8823	**Count^0.5**	5.1011	**Count^0.5**	19454.91	**Count^0.5**	1035.02	**Count^0.5**	537.23
24.0208		24.7992		24.7992		24.7992		24.7992		24.7992	
t-statistic		**t-statistic**		**t-statistic**		**t-statistic**		**t-statistic**		**t-statistic**	
1.645		1.645		1.645		1.645		1.645		1.645	
UCL_{95}normal		**UCL_{95}normal**		**UCL_{95}normal**		**UCL_{95}normal**		**UCL_{95}normal**		**UCL_{95}normal**	
6.8609		27.8985		4.5327		20024.6215		1089.0541		731.4220	

Wallace Residential Surface Soil

Zinc

Analytical Results
(mg/kg)

723	2200	2080
721	674	733
2040	1810	636
201	419	530
387	1840	156
475	468	545
218	302	531
464	603	473
341	634	
249	1290	
766	579	
531	1610	
1310	643	
655	935	
1190	985	
548	593	
434	1260	
1180	352	
1360	896	
544	383	
396	238	
633	102	
2120	376	
531	490	
1680	548	
1750	1250	
2600	2190	
3150	604	
2210	574	
680	392	
1600	509	
920	337	
193	419	
749	518	
780	453	
539	826	
1050	437	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	82	Mean	857.82
Censored		Lognormal mean	860.22
Detection limit or PQL		Std. devn.	635.6446
Method detection limit		Median	603.5
TOTAL	82	Min.	102
		Max.	3150

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -0.9031. This lies within the tabled values of 1.2344 and -2.6064

UCL (based on t-statistic) is 974.79

Wallace Residential Surface Soil

Antimony

Analytical Results
(mg/kg)

3.3	2.4	3.2
2.4	13.6	1.1
4.9	2.1	1.4
2.2	2	9.3
2.4	1.9	8.2
2.4	6.2	13.5
1.1	2.5	8
1.9	2.1	13.3
3.3	4.5	1.65
1.4	3.9	7.9
0.55	1.5	
4.6	2.6	
4.1	6.4	
4	2.3	
1.55	3.9	
2.7	4.2	
1.7	2.3	
4	9.9	
1.9	2.6	
0.8	4.4	
1.1	1.6	
6	1.2	
8	0.88	
1.3	1.3	
42.6	2.1	
43.8	1.55	
137	4.8	
161	1.4	
75.1	1	
2	0.8	
6.2	0.9	
4.2	1	
0.49	1.5	
6.1	1.15	
1.6	1.45	
2.4	0.8	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	82	Mean	8.81
Censored		Lognormal mean	5.85
Detection limit or PQL		Std. devn.	24.66527
Method detection limit		Median	2.4
TOTAL	82	Min.	0.49
		Max.	161

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -6.5821. This lies outside the tabled values of 1.2344 and -2.6064
Reject normal distribution.
Y value is -45.4442. This lies outside the tabled values of 1.2344 and -2.6064

UCL (based on t-statistic) is 13.35

Wallace Residential Surface Soil

Manganese

Analytical Results
(mg/kg)

432	587	1060
525	476	770
608	438	982
600	875	889
554	1720	800
544	806	772
447	1230	739
1100	650	186
1280	1380	794
878	598	650
1010	556	750
1030	845	724
769	899	834
772	849	818
609	934	782
994	955	873
800	1790	689
1070	807	1100
627	533	541
2290	538	
775	1340	
2130	437	
1760	560	
3470	746	
2260	533	
1770	362	
535	590	
900	614	
720	837	
304	871	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	79	Mean	894.58
Censored		Lognormal mean	888.07
Detection limit or PQL		Std. devn.	510.953
Method detection limit		Median	782
TOTAL	79	Min.	186
		Max.	3470

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -4.1594. This lies outside the tabled values of 1.221 and -2.6169
Reject normal distribution.
Y value is -14.67. This lies outside the tabled values of 1.221 and -2.6169

UCL (based on t-statistic) is 990.42

Wallace Residential Surface Soil

Iron

Analytical Results
(mg/kg)

13900	27100	16700
17800	14900	19000
17900	11200	18700
16500	19600	
18000	19100	
17000	24400	
16200	25300	
24700	27300	
25800	17800	
24000	19600	
19600	19400	
24800	21300	
20300	26700	
19800	15000	
22100	20200	
22600	17400	
23000	17800	
27000	24600	
19400	19000	
24200	19400	
20600	18100	
32900	19400	
28800	14000	
46700	17000	
45800	18000	
34100	15000	
21000	19200	
23400	17100	
25600	20600	
23900	17000	
21200	20000	
21900	17200	
14900	20000	
17600	18800	
34100	18500	
19200	18600	
25300	15700	
15700	17800	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	79	Mean	21149.37
Censored		Lognormal mean	21103.66
Detection limit or PQL		Std. devn.	6035.151
Method detection limit		Median	19400
TOTAL	79	Min.	11200
		Max.	46700

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -4.4477. This lies outside the tabled values of 1.221 and -2.6169
Reject normal distribution.
Y value is -11.1207. This lies outside the tabled values of 1.221 and -2.6169

UCL (based on t-statistic) is 22281.34

Wallace Residential Surface Soil

Cadmium

Analytical Results
(mg/kg)

6.9	2.9	4.5
5.9	3.8	6.1
14.8	3.1	1.2
1.8	8.8	5.8
2.8	3.3	1.4
3.2	3.2	3.7
2.3	5.9	
2.1	5.3	
3.3	7.5	
1.7	4.3	
6.2	5.9	
3.9	4.8	
7.9	5.1	
5.7	13.3	
5.8	4.9	
6.9	46.8	
4.3	2.9	
21.9	6.4	
4.1	4.5	
3	2.5	
3.2	0.28	
2.7	1.6	
13.3	2	
2.2	6	
13.4	16.4	
14.8	5.5	
17	7	
22.9	3	
25.8	3.9	
4.8	4.3	
6.6	3.9	
4.2	4.5	
1.1	5.1	
3.3	5.3	
3.3	6.5	
2.2	4.5	
6.4	9.9	
3	7.1	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	82	Mean	6.46
Censored		Lognormal mean	6.37
Detection limit or PQL		Std. devn.	6.650971
Method detection limit		Median	4.5
TOTAL	82	Min.	0.28
		Max.	46.8

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -4.4969. This lies outside the tabled values of 1.2344 and -2.6064
Reject normal distribution.
Y value is -23.0252. This lies outside the tabled values of 1.2344 and -2.6064

UCL (based on t-statistic) is 7.68

Wallace Residential Surface Soil

Arsenic

Analytical Results
(mg/kg)

8.8	9.4	19
6.8	26	24.4
8.6	13.1	28.3
12.1	14.9	5.7
6.2	14.3	9.2
25.3	25.2	22.3
26.6	16.7	19.1
19.7	14.8	12.1
43.3	56.5	23.4
26.3	28.8	15.5
32.9	66.1	19.6
20.6	11.4	38.4
14.3	37.5	21.8
18.3	20.6	18.1
14.9	8.3	14.2
14.5	8.4	19.1
8.8	9.4	53.9
28.9	10.6	27.9
13.8	19.1	7.8
37.7	19.1	15.8
32	11.7	15.7
50.7	17.2	14.2
46.9	16.6	
36.4	13	
8.8	22	
13.7	20.7	
16.7	18	
8.2	18.7	
11.9	15.9	
9.9	18.4	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	82	Mean	20.38
Censored		Lognormal mean	20.32
Detection limit or PQL		Std. devn.	12.11973668
Method detection limit		Median	17.6
TOTAL	82	Min.	5.7
		Max.	66.1

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -0.0191. This lies within the tabled values of 1.2344 and -2.6064

UCL (based on t-statistic) is 22.61

Wallace Residential Tap Water

Arsenic

Site ID	Static Result (ug/L)	Purged Result (ug/L)	Average Result (ug/L)
R006	0.29 J	0.44	0.365
R030	0.48	0.34	0.41
R031	0.28	0.23	0.255
R032	0.46	0.36	0.41
R049	0.1 U	0.38	0.24
R056	0.53	0.48	0.505
R069	0.4 U	0.31 U	0.355
R076	1.1	0.51	0.805
R079	0.27	0.59	0.43
R081	0.6	0.48	0.54
R145	0.25 U	0.25 U	0.25

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	11	Mean	0.42
Censored		Lognormal mean	0.42
Detection limit or PQL		Std. devn.	0.162988
Method detection limit		Median	0.41
TOTAL	11	Min.	0.24
		Max.	0.805

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9415. This exceeds the tabled value of 0.85

UCL (based on t-statistic) is 0.5

Wallace Public Recreational Exposure to Soil

Antimony

Analytical Results (mg/kg)

9	0.33	3.1	7.6	7.5	28.1	1.35
9.1	17.1	64.1	7.1	45	1.8	8.6
18.1	53.4	3.2	9.9	48.2	5.1	10.1
15.6	6.1	2.1	10.5	9.7	5.2	22.1
14.4	9.8	50.7	1.1	6.5	13.7	27.3
56	10.4	60.9	4.5	6.6	15.2	17.9
73.8	58.2	1.9	4.5	7.6	0.75	8.2
10	8.6	1.4	4.5	1.9	18.1	11.7
12.5	6	1.4	3	13.3	20	13.3
27.6	16.3	2.5	10.7	15.5	33.1	19.7
37.5	74.1	1.1	21.9	17	4.5	1.8
15.3	7.3	1.3	59.8	1.3	2.8	1.9
13.5	4.9	1.3	66.4	8.2	11.4	6.8
34.4	7.2	1.6	29.2	9.1	18.6	17.4
41.8	8.3	0.67	15.2	10.4	0.67	1.2
24	16.5	20.2	39.8	9.3	25.7	1.3
93.2	8.4	2	46.6	12.4	29.7	2
261	1.4	9.1	9.8	18.2	31.3	0.335
20.7	26.5	9.1	3.1	28.8	0.49	0.335
33.3	34.4	11	5.2	7.2	0.495	0.335
48.1	2.2	23.1	8.1	0.5	3.95	0.76
60.7	5.2	1.1	0.5	2.5	9.2	0.85
17.7	48.8	2.4	5.4	5.9	0.5	0.335
5.1	108	6.5	6.2	0.495	12.4	0.335
11.2	1.1	6.4	8.4	4	13.7	5.4
26.3	0.5	7.8	4.4	6	14.4	14.1
163	17	9	9.3	8.1	12.1	0.335
7.4	27.4	1.7	9.7	155	0.5	0.335
3.8	2.4	2.5	9.7	5.2	1.3	1.3
7.8	2.3	3.3	5.6	6.4	2.4	4.6
9.2	5.8	6.3	31.3	8.1	6.7	0.335
9.9	65.4	3.7	41.9	6.3	2.7	2.8
6.8	1.6	4.4	77.8	2.5	1.45	2
21.4	1.5	5.6	12.9	2.7	3.2	3.2
7.2	18.7	5.6	14.3	5.2	3.3	4.1
55.7	22.6	5.9	39.9	0.5	6	4.3
77.7	2.7	2.1	49.1	4.9	2	0.335
12.9	2.7	6.6	7.7	15.4	2.6	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	265	Mean	16.03
Censored		Lognormal mean	17.83
Detection limit or PQL		Std. devn.	26.62976
Method detection limit		Median	7.6
TOTAL	265	Min.	0.33
		Max.	261

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is 0.0104. This lies within the tabled values of 1.5546 and -2.3384

UCL (based on t-statistic) is 18.74

Wallace Public Recreational Exposure to Soil

Arsenic

Analytical Results (mg/kg)

17.9	5.3	9.5	39.2	16.2	11.8	14.3	29.3
18.5	13.7	50.6	21	19.3	15.1	16.7	33.5
27	21.7	11.4	21.4	23.2	12.3	20.6	35.6
31.3	7	8.9	25.4	24	18.5	22.4	12.5
22.2	11.7	103	33.2	13.4	36	22.9	
41.4	15.5	168	12.9	15.8	37.9	24	
53	22.5	7.7	18.3	23.7	13.8	21.2	
18.5	16.3	13.4	55.9	24.1	9.9	64.8	
20.7	12.5	14.5	168	15.8	14.6	77.7	
27.3	16.4	20.9	13.2	28.5	17.5	87.7	
45.4	28.7	13.7	24.9	32.2	19.8	33.5	
20.7	18.8	13.8	28.9	34.4	11.2	33.8	
26.8	8.9	16.5	30.3	14.9	24.7	41	
36.4	10.1	19.3	13	24.4	28.4	47.4	
62.3	12.3	13.1	17.1	28.3	31.5	30.9	
38.1	13.1	14.4	26.9	29.3	15.8	22.3	
61.2	20	17.2	29	15.8	15.2	22.3	
120	11	23.9	30.4	30.8	30.4	26.5	
34.1	50.9	14.9	22.7	34.8	32.5	31.2	
18.6	52.6	23.4	19.9	43.5	10.8	10.8	
21.5	13.7	24	23.1	33.5	40	12.8	
30	18.7	24.8	24.6	30.5	45.8	12.9	
25.6	58.8	45.5	15.5	34.1	49.1	11	
11	67	14	22.1	40	11.2	11.7	
13.5	10.7	10.6	22.2	18	10.4	12.5	
19.9	9.9	17.2	22.2	17.4	21.9	11	
49.5	32.3	19	11.2	23.5	36.6	14.3	
16.1	64.9	14	21.3	24	11.7	11.2	
6.2	10.9	22.4	45.4	12.8	22.2	9.9	
10.4	16.2	23.1	56.5	26.5	22.7	25.4	
13.4	19.3	28.1	19.2	27.7	23.9	30.9	
16.5	51.2	13.7	27.7	30.2	23	10.8	
16.5	15.5	18.9	28.8	41.3	11.9	6.5	
32.7	10.8	19.3	30.3	26.7	13.6	11	
26.5	39.7	21.4	17.4	37.4	18.1	18.1	
26.9	53.5	18.9	18.5	39	20	10.7	
47.3	10.9	23.6	22.4	21.6	13.8	22.8	
30.4	9.2	28.8	31.8	10.8	14.1	20.2	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	270	Mean	25.80
Censored		Lognormal mean	25.30
Detection limit or PQL		Std. devn.	19.56057
Method detection limit		Median	21.55
TOTAL	270	Min.	5.3
		Max.	168

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -2.6915. This lies outside the tabled values of 1.5578 and -2.3352
Reject normal distribution.
Y value is -36.9325. This lies outside the tabled values of 1.5578 and -2.3352

UCL (based on t-statistic) is 27.78

Wallace Public Recreational Exposure to Soil

Cadmium

Analytical Results (mg/kg)

10.7	2.3	3.3	7.4	7.2	9.1	2.9	2.2
11.3	6.4	23.8	7.4	11	12.4	3.3	2.4
13.7	7.4	2.7	0.96	18.9	0.1	4	2.5
10.8	4.5	3.6	7.3	28.2	5.1	1.1	0.285
15.6	2.7	20.9	9	7.3	14.6	1.3	
25.9	3.2	34.8	0.06	7.2	20.7	1.6	
26.3	5.9	2.4	7.5	30.2	0.52	5.1	
12.9	6.6	0.15	8.7	42.4	5.4	22.9	
14.5	1.9	0.87	10.7	2.7	5.5	27	
17.3	4.4	0.96	1.2	4.2	14.8	36.3	
18.6	6.8	0.38	5.5	5	15.1	10.7	
13.1	8.1	0.03	5.7	7	0.49	9.1	
14.6	3.7	0.03	5.7	1.5	12.8	13	
23.4	4.2	1.1	1.4	16.3	22	15.1	
44.8	4.5	0.15	18.5	19	30.7	9.2	
12.9	4.8	0.62	18.7	21.4	2.9	4.8	
18.5	8.6	1.1	39.8	1.1	2.1	5.2	
61.3	3.4	6.1	63.9	5.3	17.7	5.3	
17	19.8	0.38	10.1	5.6	28	13	
5.7	37.1	7.1	18.3	7.2	0.3	0.98	
6	2.5	7.5	26.7	5.6	30.8	1.7	
6.6	4.3	9.6	28.9	7.8	55.3	2.6	
16.1	12	9.7	9.9	12.1	64.1	0.39	
4.7	26.1	1.3	1.8	20.2	0.1	1.2	
7.3	1.8	2.7	3	6.4	0.1	0.44	
9.4	3	3.1	5.2	0.76	6.7	0.83	
11.4	13.3	7.5	1.8	1.8	15.8	1.4	
7.5	16.6	0.06	4.8	3.6	0.1	0.74	
1.3	3.9	9.4	4.8	0.4	24.6	0.55	
2.6	1.6	17.2	8.3	4.6	25.1	4.1	
6.7	4.9	22.4	3.6	5.1	28.5	8.2	
7.1	15.3	2.5	7.6	5.5	22.5	0.52	
9.4	1.3	3.1	8.2	2.4	1.1	0.76	
9.6	3.6	3.5	9.3	5.7	1.2	2.1	
6.1	9.8	4.2	3.7	7.7	1.7	5.8	
10.5	11.8	2.9	14.4	8.5	4.2	0.34	
22.4	2.1	5.9	26.2	6.2	2.7	2.1	
13.8	2.8	7.1	26.7	3.3	2.7	1.6	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	270	Mean	9.64
Censored		Lognormal mean	12.96
Detection limit or PQL		Std. devn.	10.83417
Method detection limit		Median	5.95
TOTAL	270	Min.	0.03
		Max.	64.1

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -6.3096. This lies outside the tabled values of 1.5578 and -2.3352
Reject normal distribution.
Y value is -23.7644. This lies outside the tabled values of 1.5578 and -2.3352

UCL (based on t-statistic) is 10.73

Wallace Public Recreational Exposure to Soil

Iron

Analytical Results (mg/kg)

23500	15900	16400	28400	22900	20900	22700	22200
23900	20600	42400	21400	19300	23500	22800	23700
28700	38200	17600	22500	27000	20800	22000	24000
34700	16700	16400	23100	27300	22900	18900	19600
27400	18900	37400	25200	17600	34500	19700	
46800	20100	49200	21500	23900	40800	19900	
60000	43700	13900	19600	27300	21400	21300	
23100	20000	20200	22197	30000	17600	69400	
25300	19500	23800	23266	21900	21200	71900	
28700	20400	24100	21500	22300	21700	143000	
31500	45400	20400	22538	23500	24100	30300	
25800	18400	20100	24200	23600	19400	38100	
28100	18300	20200	24300	19000	34400	50100	
38300	19800	23200	13762	37900	42300	53900	
51300	20900	21400	23200	43000	45200	28200	
34700	21800	22400	32400	48600	19800	19800	
76600	19600	23900	34600	21000	25800	20400	
152000	20500	50600	42100	23000	37000	22400	
33000	51100	22400	24800	24100	37400	28600	
25900	52900	20500	23800	24300	20000	17600	
29000	20900	20600	25900	23700	37100	18200	
33100	30900	20700	28400	22700	51100	18600	
24800	44400	26400	18400	24600	51900	21900	
15600	77300	23400	20300	27400	21400	20300	
21100	18700	21000	20600	19200	20500	22900	
25600	18900	21600	21400	17600	26400	18700	
70300	30200	35900	17400	20600	33400	18900	
17700	37400	21700	18600	21900	19700	21300	
10800	18900	21000	21000	19800	25100	19100	
15800	24600	21600	22200	22000	27100	21900	
16000	26300	24200	16800	22500	29100	21900	
17100	43700	20200	23600	23000	24600	22100	
18400	24500	18000	24400	19000	17400	18800	
23900	20000	21900	25700	22700	18700	18800	
17500	24100	25400	15800	23700	19900	19300	
29700	31500	26200	23400	24300	21200	21000	
38100	20300	21300	28100	21300	21000	22500	
20900	15000	25100	35000	19600	22600	22000	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	270	Mean	27036.16
Censored		Lognormal mean	26589.18
Detection limit or PQL		Std. devn.	14897.63
Method detection limit		Median	22650
TOTAL	270	Min.	10800
		Max.	152000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -17.1969. This lies outside the tabled values of 1.5578 and -2.3352
Reject normal distribution.
Y value is -48.8187. This lies outside the tabled values of 1.5578 and -2.3352

UCL (based on t-statistic) is 28539.37

Wallace Public Recreational Exposure to Soil

Manganese

Analytical Results (mg/kg)

1340	551	454	1970	860	805	795	828
1400	1100	2960	1120	939	861	832	855
1810	3060	615	720	979	665	830	861
2150	613	311	925	1300	1180	592	577
1800	801	2070	1290	614	1930	604	
3820	1020	3140	837	1090	2640	608	
5150	3000	390	1050	1130	802	837	
1360	959	463	1220	1240	846	3160	
1140	936	708	1350	894	1140	3450	
1790	1190	721	953	1110	1160	7230	
1840	3790	753	980	1200	1180	1390	
1780	892	366	1010	1240	694	1980	
1630	696	378	1230	714	1990	2890	
2880	901	647	462	2570	2640	3210	
4880	1010	711	672	2960	2990	1330	
1930	1170	662	1280	3670	1020	592	
9030	945	751	1380	840	911	619	
18200	510	2800	2980	1100	1640	667	
2730	1940	789	1050	1160	1640	1220	
1320	3230	871	872	1320	717	589	
1540	329	1060	1090	1170	1990	634	
1710	789	1110	1230	1040	2730	693	
1310	2810	1430	762	1060	2860	861	
806	5990	917	746	1230	660	671	
1090	398	742	774	601	639	690	
1430	465	1150	867	559	1230	579	
5340	1290	1730	602	817	2040	607	
920	1670	534	829	915	690	679	
341	467	1020	1220	713	1390	697	
785	811	1020	1310	885	1410	710	
787	975	1270	739	956	1530	843	
858	2740	717	1290	1080	1230	684	
760	768	832	1320	479	555	654	
1180	553	975	1400	1200	655	782	
491	1170	1010	633	1260	706	791	
1890	1260	1020	948	1420	923	819	
2870	462	1020	1000	1000	709	767	
840	331	1390	1090	672	716	752	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	270	Mean	1365.88
Censored		Lognormal mean	1297.84
Detection limit or PQL		Std. devn.	1473.46
Method detection limit		Median	990
TOTAL	270	Min.	311
		Max.	18200

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -7.8751. This lies outside the tabled values of 1.5578 and -2.3352
Reject normal distribution.
Y value is -59.4389. This lies outside the tabled values of 1.5578 and -2.3352

UCL (based on t-statistic) is 1514.56

Wallace Public Recreational Exposure to Soil

Zinc

Analytical Results (mg/kg)

1760	164	323	1500	887	1950	805	342
1960	710	4050	1120	1640	2040	840	391
2430	968	369	236	2440	173	917	392
3680	317	201	1300	2810	871	538	97.7
1960	471	2440	1630	968	2320	584	
2650	691	4260	110	1430	2730	606	
2900	742	180	876	2340	381	1050	
1680	797	96.7	1410	3160	586	7160	
3941	682	149	1440	616	1150	7290	
11602	861	297	224	1040	2240	13800	
14655	868	138	805	1080	2310	2400	
1930	703	76.3	897	1460	106	2870	
2660	483	105	949	240	2150	4750	
5677	509	160	221	6320	3160	5020	
10623	546	114	2360	8800	3660	1780	
3345	695	141	2380	9350	688	892	
2910	991	391	3150	303	471	992	
9120	261	4280	4280	1080	2630	1160	
2411	3850	118	1620	1130	3850	2180	
1200	7550	1010	2020	1530	120	209	
1210	358	1040	2720	1130	4050	350	
1260	453	1090	2840	1260	6240	458	
1920	2370	1210	1070	2320	7110	85	
599	5370	385	515	2850	166	206	
1100	211	149	634	823	112	88.6	
1540	1030	432	698	328	1000	240	
1640	2260	2340	159	348	2480	257	
899	3360	107	707	614	226	117	
375	380	1190	1070	183	3930	98.6	
546	333	2010	1700	733	4100	767	
930	800	2470	523	794	4240	1330	
1050	3360	359	1270	795	3360	99.2	
1790	256	551	1330	505	436	102	
1900	282	570	1370	1030	482	307	
727	1620	720	839	1030	585	971	
1700	2130	481	1950	1390	1110	80.7	
2720	286	857	2600	777	780	213	
1220	229	1470	3070	469	796	194	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	270	Mean	1681.15
Censored		Lognormal mean	1774.94
Detection limit or PQL		Std. devn.	2123.855
Method detection limit		Median	991.5
TOTAL	270	Min.	76.3
		Max.	14655

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is 1.0928. This lies within the tabled values of 1.5578 and -2.3352

UCL (based on t-statistic) is 1895.46

Nine Mile Residential Surface Soil

Arsenic

Analytical Results
(mg/kg)

23.4	18.5	11	19.3	8.4	9.5	49.7
99.6	53.8	47.8	14.1	8.3	14.2	15.6
26.5	12.9	35.5	16.8	8.3	31.7	61.3
30.8	13.9	18.2	15.4	8.3	14.3	11.5
20.5	9.1	26.9	17.1	9.7	27.3	19
10.9	30.9	19.2	32	151	14.9	15.2
6.8	24	18	49	8.9	31.5	11.3
10.5	10.8	22.5	59.6	10.7	13.1	12.8
7.5	19.7	28.9	20.6	15.1	17	12.9
9.8	16.4	24.9	19.2	12.5	92.6	16.5
535	32.2	38.5	15.3	14.6	6.9	11.8
13	10.8	46.6	36.2	10.3	5.8	11.2
484	26.3	12.9	21.7	16.7	10.2	15.3
19.7	8.1	43.3	19.5	289	14.8	14.8
19.9	7.3	29.4	8	11.5	11.9	12.4
153	35	35.9	16.9	13.2	18.3	14.1
20.4	10.2	33.6	5.8	23.7	13.2	
11.6	10.8	13.9	16.5	19.7	7.7	
10.9	13.7	100	4.7	30.9	15.5	
28.2	16.5	16.5	9.7	16.6	11.6	
13.2	35	11	7.9	9.9	8.8	
11.7	34.1	10	18.5	53	14.7	
8.8	12.6	12.3	25	13	17.5	
23	6.95	22.5	17.1	15.6	2.9	
3.95	11	1150	36.2	26.1	27.1	
9.2	16.1	37.7	10.2	9	14.3	
8.5	12	13.2	14.1	15.6	8.9	
8.5	9.6	8.8	45.5	24.4	19.1	
7.8	38.6	13.3	15.8	13.7	10.7	
9.1	14.4	8.6	18.4	15.2	6.1	
14.4	10.8	8.7	33	11.2	10.7	
4	47.9	12.9	19.4	14.7	9	
18.2	11.1	37.6	9.6	10.4	11.6	
131	17.8	10.9	19.8	8.7	9.4	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	220	Mean	31.58
Censored		Lognormal mean	23.62
Detection limit or PQL		Std. devn.	92.94891635
Method detection limit		Median	14.8
TOTAL	220	Min.	2.9
		Max.	1150

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -15.9308. This lies outside the tabled values of 1.5156 and -2.3738
Reject normal distribution.
Y value is -90.3503. This lies outside the tabled values of 1.5156 and -2.3738

UCL (based on t-statistic) is 41.97

Nine Mile Residential Surface Soil

Cadmium

Analytical Results
(mg/kg)

7.3	5.9	0.22	5.6	15.3	4	12.8
13.8	20.9	6.5	5.6	4.3	2.9	5.8
3.9	3.6	6.1	21.3	7.9	4.7	4.3
9.4	3.8	12.1	5	12.3	0.37	1.7
3.6	5.3	6.4	8.4	1.8	6.8	1.5
0.85	13.5	4.2	9.6	18.2	0.88	2.1
3.8	4	5.3	7.8	2.3	5.8	1.9
0.1	7	7.5	7.4	3.6	5.7	1.2
2.4	2.5	9.1	5.3	1.3	9.8	1.4
1.4	4.3	5.6	5.2	2.6	12.8	7.9
0.86	4.6	7.9	6.9	1.4	9.4	4.5
4.1	7.1	8.8	11.4	2.6	2.2	0.88
4.4	14.7	4.7	7	7.2	19.8	7.1
3	5.6	6.8	6.6	8.1	19.8	6.3
0.51	5.6	7.8	15.1	5.8	12.2	3.7
12	4	12.5	0.1	4.8	13.3	4.2
1.2	8.6	11.3	13.4	5.4	2.3	
1	4.9	4.2	0.1	6.5	2.7	
20.7	5.5	12.5	16.9	10.9	2.8	
1.8	3	1.1	8.2	8.1	2.5	
7	5.9	2	15.3	0.35	2.5	
16.2	27.1	1.5	0.1	11.8	2.6	
8.6	0.2	1.9	4.4	4.2	3.6	
17.5	7.9	6.3	5	3.7	0.87	
5.7	0.59	0.04	24.1	12	6.2	
5.9	4.8	5.8	3.8	0.56	2.2	
3.8	2.3	2.7	6.2	6.1	1.1	
6.3	0.04	1.5	5.8	1.6	7.9	
4.2	10.5	1.6	11.5	4.1	2	
14.3	0.33	1.4	12.2	5.6	1.4	
1.9	0.235	1.6	6.6	4	3.3	
0.04	4.6	4.3	2.4	4.1	1.9	
7.9	0.5	20.3	4.7	5.7	1.5	
4.8	3.2	8.4	1.3	5.3	1.6	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	220	Mean	6.00
Censored		Lognormal mean	7.66
Detection limit or PQL		Std. devn.	5.034372381
Method detection limit		Median	4.8
TOTAL	220	Min.	0.04
		Max.	27.1

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -11.2272. This lies outside the tabled values of 1.5156 and -2.3738
Reject normal distribution.
Y value is -10.5229. This lies outside the tabled values of 1.5156 and -2.3738

UCL (based on t-statistic) is 6.56

Nine Mile Residential Surface Soil

Iron

Analytical Results
(mg/kg)

15200	39700	25400	16300	14800	20000	31600
29800	23500	37400	20900	16200	21400	17500
15800	22600	22800	28100	18300	20400	26200
13700	21700	35600	38600	25800	18100	12100
64300	18600	28600	34800	18400	20300	17500
19400	17700	32900	28100	20000	22200	13300
55400	16600	30300	26200	19600	28200	12800
16900	35100	41700	23900	19100	14000	12800
29800	15200	26700	39000	19800	22400	12600
33200	17200	31200	24600	17900	10100	18700
27000	23100	28400	24200	20400	5910	15800
21100	17600	41100	11000	42900	9770	18200
22600	16300	37300	21500	18400	11300	20300
19700	16900	16000	8120	24300	9880	17900
25800	23600	34100	20000	22800	6600	18600
27700	37300	15500	7160	28000	12400	17300
12600	21800	15300	11100	25900	12400	
15800	19500	14800	11400	21800	16300	
11500	18900	15100	22700	17000	17000	
13700	19600	32900	23600	30200	14200	
10100	20000	123000	22100	18200	13600	
12700	20900	31600	88400	15500	16400	
10400	19800	20300	16700	17600	8780	
12400	26800	20600	19800	16100	22400	
20000	18600	18400	40900	18300	22000	
9270	19200	22000	18600	22400	14900	
23800	30800	22400	27600	18300	20200	
15500	19500	17800	20600	17100	18200	
25500	23800	38200	17800	16600	19100	
19900	18600	17300	19900	19300	18600	
20300	25100	19400	19900	20300	19000	
17100	24300	13800	17100	16900	20700	
18400	24400	18100	17800	17000	19400	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	214	Mean	21867.71
Censored		Lognormal mean	21677.62
Detection limit or PQL		Std. devn.	11606.26178
Method detection limit		Median	19550
TOTAL	214	Min.	5910
		Max.	123000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -8.4723. This lies outside the tabled values of 1.5097 and -2.379
Reject normal distribution.
Y value is -35.0103. This lies outside the tabled values of 1.5097 and -2.379

UCL (based on t-statistic) is 23183.15

Nine Mile Residential Surface Soil

Manganese

Analytical Results
(mg/kg)

752	1620	1150	613	800	1860	1770
566	972	1130	607	697	1650	1070
750	635	1030	1970	692	1110	1500
401	707	1310	1820	2220	1820	789
4010	1350	1060	1740	803	1160	747
664	552	1530	920	948	2720	937
4000	1190	1980	923	617	1680	705
543	2030	2100	1130	807	710	771
1010	1600	930	1800	626	987	749
1980	1420	1640	922	793	542	1140
1100	1070	1610	763	1250	665	1000
838	1570	2320	353	2760	413	644
917	1540	1600	655	1020	480	1140
1490	1150	1130	227	1240	472	1060
869	1110	2300	469	787	342	1000
916	2560	961	242	1000	747	819
947	1180	984	641	1020	897	
1530	385	1070	432	986	924	
550	1030	935	466	558	1140	
1240	644	871	889	1730	1060	
587	1100	6580	1160	802	745	
1220	848	1070	7540	754	869	
608	606	872	647	1250	867	
889	1730	541	1480	436	1960	
758	873	828	3390	762	1400	
980	817	591	1130	753	1070	
1200	1880	661	1760	1030	2500	
1510	812	744	737	790	1650	
1210	1120	2530	800	802	1730	
2000	757	758	973	1190	732	
818	1530	807	945	829	734	
768	1530	401	768	597	863	
753	1110	580	755	760	865	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	214	Mean	1154.14
Censored		Lognormal mean	1134.18
Detection limit or PQL		Std. devn.	821.7610041
Method detection limit		Median	941
TOTAL	214	Min.	227
		Max.	7540

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -5.4607. This lies outside the tabled values of 1.5097 and -2.379
Reject normal distribution.
Y value is -35.7354. This lies outside the tabled values of 1.5097 and -2.379

UCL (based on t-statistic) is 1247.28

Nine Mile Residential Surface Soil

Antimony

Analytical Results

(mg/kg)

4.4	2.5	14.7	3.4	1.35	5.3	15.8
20.4	4.2	17.3	3.1	4.3	8.7	5.8
3.9	1.4	12	3.3	1.1	5.3	76.8
5.3	34.4	19.1	4.6	2.1	20.2	1.8
2.1	2.6	6.4	12.2	3.8	7.4	7.2
1.2	2.05	20.2	20.7	54.5	20.2	2.3
1.3	10.1	32.4	36.4	2.9	5.4	2.6
145	5.2	11.6	7.2	4.3	4.5	1.8
1.8	4.9	16.7	9	2.4	24.6	1.9
105	5.8	16	3.4	3.3	1.3	5.5
4.7	30.4	9.2	17.5	4.2	2.4	1.45
0.5	2.8	14.6	24.9	44.2	4.3	0.55
17.5	2.3	17.9	4.5	11.7	7.3	3.9
1.5	8.5	19.9	3.2	4.4	8.4	4.7
0.5	3.9	29	1	41.8	2.6	0.95
1.6	1.35	3.3	1.3	22	0.7	0.9
1.5	3.6	24.5	0.5	25.1	0.95	
1	2.3	1.5	2.4	8.2	1.15	
1	20.9	1.2	2.4	0.55	1.1	
2.6	7.1	1.5	4.2	18.5	0.65	
3.7	0.5	1.5	0.5	1.6	0.6	
2	1.1	4.4	3.7	3.4	1.2	
2.3	1.3	222	5.5	8.7	0.8	
1.7	1.8	5.9	30.8	0.265	8.7	
2.4	1.8	1.5	1.3	4.4	1.4	
1.5	0.84	1.6	4.3	0.8	3.7	
2.8	17.1	1.2	16.9	1.15	5.4	
8	1.5	0.5	7.5	1.05	3.8	
1.6	1.1	0.495	5	1.1	1.3	
5.3	13.7	4.7	1.4	1	2	
22.2	1.3	12.1	3.6	1.5	1.7	
6.2	2.3	1.9	1.2	1.15	0.67	
17.8	9.7	2.9	5.5	1.3	0.335	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	214	Mean	9.34
Censored		Lognormal mean	8.21
Detection limit or PQL		Std. devn.	21.15274779
Method detection limit		Median	3.5
TOTAL	214	Min.	0.265
		Max.	222

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -0.5207. This lies within the tabled values of 1.5097 and -2.379

UCL (based on t-statistic) is 11.74

Nine Mile Residential Surface Soil

Zinc

Analytical Results

(mg/kg)

972	1370	173	502	1240	511	2150
3580	3360	1860	716	781	615	875
677	926	1940	894	947	1090	1560
1460	877	1930	646	1060	324	212
495	651	1580	899	295	756	371
221	1900	734	2060	2580	401	281
469	1030	1140	3100	246	1420	345
159	1020	1240	2910	380	1210	232
317	604	1540	1120	107	1520	203
360	806	2050	1340	461	2300	1040
373	762	2720	972	107	1590	726
544	1050	2880	3110	297	459	537
751	4130	927	1380	1120	2510	970
425	740	1780	1190	2230	803	839
245	866	2050	2440	914	1800	498
1590	944	2300	96	1020	1820	649
351	1180	1870	2010	759	424	
242	1620	622	115	1030	382	
3020	693	1650	2680	1790	513	
401	1300	198	1330	1170	440	
953	3530	307	2230	89	442	
2000	1940	226	80.6	2380	346	
1310	162	235	474	586	545	
3180	782	1300	1620	471	299	
997	529	421	4370	1590	1320	
989	1010	926	166	109	2270	
730	576	390	830	648	408	
1110	102	287	1860	387	4420	
792	2280	848	1550	428	468	
1060	279	242	2210	667	344	
899	350	328	979	400	618	
86.6	2070	435	594	394	356	
1280	196	3030	765	524	238	
1020	545	792	732	492	350	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	220	Mean	1066.63
Censored		Lognormal mean	1112.24
Detection limit or PQL		Std. devn.	872.5293297
Method detection limit		Median	804.5
TOTAL	220	Min.	80.6
		Max.	4420

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is 0.6886. This lies within the tabled values of 1.5156 and -2.3738

UCL (based on t-statistic) is 1164.17

Nine Mile Residential Tap Water

Arsenic

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	26	Mean	0.45
Censored		Lognormal mean	0.37
Detection limit or PQL		Std. devn.	0.825072794
Method detection limit		Median	0.22
TOTAL	26	Min.	0.1
		Max.	3.8

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8047. This is less than the tabled value of 0.92
Reject normal distribution.
W value is 0.4459. This is less than the tabled value of 0.92

UCL (based on t-statistic) is 0.73

Site ID	**Static Result** (ug/L)	**Purged Result** (ug/L)	**Average Result** (ug/L)
R010	0.1 U	0.1 U	0.1
R011	0.1 U	0.26	0.18
R026	0.4	0.1 U	0.25
R034	0.26	0.1 U	0.18
R035	0.1 U	0.41	0.255
R037	0.1 U	0.1 U	0.1
R039	3.4	4.2	3.8
R040	0.1 U	0.1 U	0.1
R042	0.1 U	0.24	0.17
R044	0.1 U	0.1 U	0.1
R050	0.395 U	0.3 U	0.3475
R052	0.24	0.21	0.225
R053	0.1 U	0.1 U	0.1
R054	0.1 U	0.1 U	0.1
R055	2.3	2.6	2.45
R065	0.29	0.26	0.275
R067	0.25 J	0.43	0.34
R068	0.1 U	0.1 U	0.1
R071	0.33	0.1 U	0.215
R077	0.1 U	0.1 U	0.1
R078	0.1 U	0.22	0.16
R080	0.26 U	0.345 U	0.3025
R087	0.92	0.8	0.86
R126	0.25 U	0.25 U	0.25
R133	0.25 U	0.25 U	0.25
R143	0.59	0.25 U	0.42

Nine Mile Future Residential Tap Water (GW data)

Antimony

Analytical Results

(ug/L)	
1.8	0.195
2	0.07
0.5	0.075
0.5	0.08
0.5	0.08
0.5	0.095
0.5	0.115
1.6	0.75
1.8	1.6
5.5	1.7
0.0245	0.225
0.0495	0.265
0.15	0.27
0.06	0.26
0.06	0.05
0.07	0.355
0.26	0.37
0.27	1.2
0.81	1.2
18	0.075
2	0.165
4.8	0.18
6.8	2.1
7	2.7
8.6	5
10.5	0.325
0.055	0.345
0.095	0.335
7	0.36
7.1	0.5
2.1	2.4
0.5	2.4
0.5	0.5
0.5	0.5
0.5	1
0.5	1.6
1.4	1.6
0.5	0.5
0.18	0.5
0.185	0.5

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	80	Mean	1.60
Censored		Lognormal mean	1.67
Detection limit or PQL		Std. devn.	2.853618
Method detection limit		Median	0.5
TOTAL	80	Min.	0.0245
		Max.	18

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is 0.311. This lies within the tabled values of 1.226 and -2.613

UCL (based on t-statistic) is 2.14

Nine Mile Future Residential Tap Water (GW data)

Arsenic

Analytical Results

	(ug/L)
0.5	0.205
0.5	0.16
0.5	0.165
0.5	0.195
0.5	0.26
0.5	0.29
0.5	0.305
2.2	0.1
3.8	0.15
2.2	0.235
0.1	0.13
0.1	0.185
0.26	0.195
0.1	1.3
0.1	0.37
0.25	0.57
0.97	0.62
1	0.38
1.1	0.39
0.28	0.1
0.1	0.28
0.1	0.37
0.1	0.1
0.1	0.21
0.1	0.27
0.1	0.1
0.1	0.1
0.1	12.8
0.5	16.1
0.5	0.5
0.5	0.5
0.5	1.1
0.5	0.5
0.5	0.5
0.5	0.5
0.5	0.5
0.5	0.5
0.5	0.5
0.1	0.5
0.105	0.5

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	80	Mean	0.81
Censored		Lognormal mean	0.58
Detection limit or PQL		Std. devn.	2.278307
Method detection limit		Median	0.385
TOTAL	80	Min.	0.1
		Max.	16.1

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -6.6759. This lies outside the tabled values of 1.226 and -2.613
Reject normal distribution.
Y value is -48.4185. This lies outside the tabled values of 1.226 and -2.613

UCL (based on t-statistic) is 1.23

Nine Mile Future Residential Tap Water (GW data)

Cadmium

Analytical Results

(ug/L)

5.1	1.3
6.7	4.4
5.9	4.5
7	4.8
0.5	157
0.5	222
0.5	226
3	42.3
0.69	50.2
3.2	56.5
0.0395	138
0.0395	147
0.1	149
13.7	0.35
13.7	240
19.2	246
770	258
944	185
996	186
0.34	131
19.3	211
48.1	262
29.6	20.5
29.7	25.4
28.7	48.1
28.9	11.2
0.14	11.6
0.48	0.0395
207	0.2
209	4.7
8.2	4.8
0.5	5
0.5	50.6
0.5	51
0.5	52.6
7.3	43.4
9.4	44.5
43.1	7.3
1.1	572
1.1	4.8

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	80	Mean	91.79
Censored		Lognormal mean	310.09
Detection limit or PQL		Std. devn.	188.9217
Method detection limit		Median	13.7
TOTAL	80	Min.	0.0395
		Max.	996

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is 0.5129. This lies within the tabled values of 1.226 and -2.613

UCL (based on t-statistic) is 127

Nine Mile Future Residential Tap Water (GW data)

Zinc

Analytical Results

(ug/L)	
817	227
4600	495
1480	495
1730	498
45.8	35100
59.8	35400
61.9	36300
610	5640
76.1	6340
442	6970
2.8	16400
5	18400
3.5	19200
3170	68.6
3370	41900
4730	43100
101000	45400
135000	37700
145000	38500
35.7	14400
2870	39200
7950	42500
4350	2990
4400	3690
4280	6870
4380	3020
36.6	3020
76.2	17.4
33600	36.5
33800	966
1110	972
5.3	1190
11	9170
16.1	9220
2.5	9270
1080	6040
1240	6190
5360	1690
194	82100
212	1190

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	80	Mean	14238.61
Censored		Lognormal mean	86970.91
Detection limit or PQL		Std. devn.	27547.22
Method detection limit		Median	3095
TOTAL	80	Min.	2.5
		Max.	145000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -0.6579. This lies within the tabled values of 1.226 and -2.613

UCL (based on t-statistic) is 19372.4

Nine Mile Neighborhood Exposure to Waste Piles

Antimony

Analytical Results
(mg/kg)

79.3
12.7
25.3
242
3.2
2.3
1.8
2.7
3.7
1.7
2.2
1.7
51.6
0.21
2.8
10.6
9.3
6.6
4.2
10.5
3.1

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	21	Mean	22.74
Censored		Lognormal mean	19.81
Detection limit or PQL		Std. devn.	53.80367
Method detection limit		Median	3.7
TOTAL	21	Min.	0.21
		Max.	242

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9361. This exceeds the tabled value of 0.908

UCL (based on t-statistic) is 42.99

Nine Mile Neighborhood Exposure to Waste Piles

Arsenic

Analytical Results
(mg/kg)

44.6
103
141
3610
84.7
14.1
12.8
75.3
20.1
49.8
5.8
6.8
6.9
1.6
97
10
25.8
23.8
7.5
5.4
43.6
14.5

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	22	Mean	200.19
Censored		Lognormal mean	95.57
Detection limit or PQL		Std. devn.	762.5801137
Method detection limit		Median	21.95
TOTAL	22	Min.	1.6
		Max.	3610

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9162. This exceeds the tabled value of 0.911

UCL (based on t-statistic) is 479.99

Nine Mile Neighborhood Exposure to Waste Piles

Cadmium

Analytical Results
(mg/kg)

17.3
53
15.7
1.9
21.8
6.4
10.9
19.3
13.5
3.3
1.8
0.175
3.4
146
0.12
9.4
16.4
19.9
3.1
40.1
14.5
8.5

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	22	Mean	19.39
Censored		Lognormal mean	31.00
Detection limit or PQL		Std. devn.	31.07229266
Method detection limit		Median	12.2
TOTAL	22	Min.	0.12
		Max.	146

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.9085. This is less than the tabled value of 0.911
Reject normal distribution.
W value is 0.5555. This is less than the tabled value of 0.911

UCL (based on t-statistic) is 30.79

Nine Mile Neighborhood Exposure to Waste Piles

Iron

Analytical Results
(mg/kg)

23400
87900
25200
46300
77400
32700
31800
36000
34900
35300
18800
19300
16000
18500
46000
27900
20100
49500
37700
28400
64400
29300

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	22	Mean	36672.73
Censored		Lognormal mean	36628.74
Detection limit or PQL		Std. devn.	19027.65361
Method detection limit		Median	32250
TOTAL	22	Min.	16000
		Max.	87900

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9609. This exceeds the tabled value of 0.911

UCL (based on t-statistic) is 43654.32

Nine Mile Neighborhood Exposure to Waste Piles

Manganese

Analytical Results
(mg/kg)

2580
3360
1020
1800
2930
1270
1250
810
1360
910
882
503
799
2570
3020
798
1270
326
351
1560
1340
1080

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	22	Mean	1444.95
Censored		Lognormal mean	1477.70
Detection limit or PQL		Std. devn.	890.5924982
Method detection limit		Median	1260
TOTAL	22	Min.	326
		Max.	3360

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9541. This exceeds the tabled value of 0.911

UCL (based on t-statistic) is 1771.73

Nine Mile Neighborhood Exposure to Waste Piles

Zinc

Analytical Results
(mg/kg)

1810
7180
2680
591
4340
2440
2560
5840
5050
1110
251
145
245
109
25800
2870
3160
3980
1090
9300
4110
2970

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	22	Mean	3983.23
Censored		Lognormal mean	5146.98
Detection limit or PQL		Std. devn.	5428.052899
Method detection limit		Median	2775
TOTAL	22	Min.	109
		Max.	25800

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9303. This exceeds the tabled value of 0.911

UCL (based on t-statistic) is 5974.88

Nine Mile Neighborhood Exposure to Sediments

Antimony

Analytical Results

(mg/kg)

1.1
1.7
2.45
288
4.25
18.6
1.53
1.1
5.7
37.1
2.33
53
72.3
169
9.74
1.14
0.605

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	17	Mean	39.39
Censored		Lognormal mean	47.54
Detection limit or PQL		Std. devn.	77.07623
Method detection limit		Median	4.25
TOTAL	17	Min.	0.605
		Max.	288

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9122. This exceeds the tabled value of 0.892

UCL (based on t-statistic) is 72.03

Nine Mile Neighborhood Exposure to Sediments

Arsenic

Analytical Results

(mg/kg)

3.6
4.5
4.5
8.4
15.9
14.3
1.91
8.9
13.2
30.3
3.11
9.12
112
216
11.5
11.4
2.34

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	17	Mean	27.70
Censored		Lognormal mean	23.12
Detection limit or PQL		Std. devn.	54.92533445
Method detection limit		Median	9.12
TOTAL	17	Min.	1.91
		Max.	216

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9087. This exceeds the tabled value of 0.892

UCL (based on t-statistic) is 50.96

Nine Mile Neighborhood Exposure to Sediments

Cadmium

Analytical Results

(mg/kg)

0.52
0.65
8.2
132
5.3
6.3
34.7
13.3
55
40.9
27.3
1.26
85.6
194
22.1
4.89
3.01

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	17	Mean	37.35
Censored		Lognormal mean	58.80
Detection limit or PQL		Std. devn.	53.70565835
Method detection limit		Median	13.3
TOTAL	17	Min.	0.52
		Max.	194

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9668. This exceeds the tabled value of 0.892

UCL (based on t-statistic) is 60.1

Nine Mile Neighborhood Exposure to Sediments

Iron

Analytical Results

(mg/kg)

11600
11700
8090
28100
20200
13000
14300
98700
29200
37100
14200
15500
35200
47800
28800
21900
21600

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	17	Mean	26881.76
Censored		Lognormal mean	26574.83
Detection limit or PQL		Std. devn.	21411.82566
Method detection limit		Median	21600
TOTAL	17	Min.	8090
		Max.	98700

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.96. This exceeds the tabled value of 0.892

UCL (based on t-statistic) is 35948.97

Nine Mile Neighborhood Exposure to Sediments

Manganese

Analytical Results

(mg/kg)

428
665
330
1820
1130
1130
575
6810
2700
6830
759
1250
1600
2690
1920
693
529

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	17	Mean	1874.06
Censored		Lognormal mean	1855.61
Detection limit or PQL		Std. devn.	1998.776
Method detection limit		Median	1130
TOTAL	17	Min.	330
		Max.	6830

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9487. This exceeds the tabled value of 0.892

UCL (based on t-statistic) is 2720.47

Nine Mile Neighborhood Exposure to Sediments

Zinc

Analytical Results

(mg/kg)

220
162
1480
22400
972
844
6940
12200
7310
6300
5130
405
17600
15900
2470
1120
1170

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	17	Mean	6036.65
Censored		Lognormal mean	8396.23
Detection limit or PQL		Std. devn.	6955.61
Method detection limit		Median	2470
TOTAL	17	Min.	162
		Max.	22400

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9449. This exceeds the tabled value of 0.892

UCL (based on t-statistic) is 8982.12

Nine Mile Neighborhood Exposure to Surface Water

Arsenic

Analytical Results

(ug/L)	
0.3	0.62
10.9	1.8
8.1	0.42
0.28	1
0.42	0.12
0.96	0.5
0.31	0.1
0.84	0.1
0.5	0.08
0.08	0.5
0.5	0.08
0.08	1
0.235	0.19
1.4	1
1	2
0.255	0.26
0.34	0.115
0.5	0.08
0.28	0.5
0.37	0.115
0.5	0.08
0.175	1
0.23	0.08
1	1
0.165	0.25
0.55	1
1	0.18
0.195	1
0.33	0.24
1	0.5
0.115	0.37
0.21	1
0.45	0.08
1	0.5
0.385	0.115
0.55	0.5
2.7	0.31
1	1
0.32	0.5
1	1

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	80	Mean	0.77
Censored		Lognormal mean	0.70
Detection limit or PQL		Std. devn.	1.500935146
Method detection limit		Median	0.435
TOTAL	80	Min.	0.08
		Max.	10.9

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -1.685. This lies within the tabled values of 1.226 and -2.613

UCL (based on t-statistic) is 1.05

Nine Mile Neighborhood Exposure to Surface Water

Cadmium

Analytical Results

(ug/L)	
52.9	0.79
1520	0.35
1810	0.35
13.1	0.26
15.9	40.4
156	11.4
13.5	38
20.7	10
4.9	16.4
0.0345	6.5
0.05	15.8
0.0345	5.4
7.1	1.1
10.4	0.1
2.1	2
7.8	0.87
12.7	0.41
2.5	27.4
7.7	11.2
13.9	12.5
2.8	29.5
9.6	10.2
17.7	0.0345
3.3	0.2
17.8	16
31	13.2
5.1	42.7
18.2	10.5
31.5	33
5.2	11.1
6.7	0.5
4.3	0.3
5.4	0.0345
1.4	0.05
3.2	28.2
3.9	11.3
0.0345	0.0345
0.9	0.4
2.2	0.05
0.3	1.5

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	80	Mean	53.15
Censored		Lognormal mean	58.92
Detection limit or PQL		Std. devn.	261.5187284
Method detection limit		Median	6.6
TOTAL	80	Min.	0.0345
		Max.	1810

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -2.5864. This lies within the tabled values of 1.226 and -2.613

UCL (based on t-statistic) is 101.89

Nine Mile Neighborhood Exposure to Surface Water

Manganese

Analytical Results

(ug/L)	
3170	33.3
26800	14.2
25300	10
1090	12.7
8.6	42.2
216	47.3
60	51.3
135	66.6
39.1	37.4
2	51.5
2.5	94.1
0.5	61
69.3	2.8
139	2.5
30	81.1
67.3	0.5
81.4	0.4
34.6	44.6
67.5	36.8
84.8	35.6
34.3	59.3
59.9	37
80.5	7.4
31	2.5
70.8	126
118	141
35	45.1
69.6	42
108	72
37	43.9
42.9	72.8
29.4	52
32.2	1.1
15	2.5
35	52.8
37.6	39
23.6	35
14	11
27.6	2.5
13	23

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	80	Mean	748.82
Censored		Lognormal mean	195.63
Detection limit or PQL		Std. devn.	4095.369786
Method detection limit		Median	39.05
TOTAL	80	Min.	0.4
		Max.	26800

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -10.5431. This lies outside the tabled values of 1.226 and -2.613
Reject normal distribution.
Y value is -58.4507. This lies outside the tabled values of 1.226 and -2.613

UCL (based on t-statistic) is 1512.05

Nine Mile Neighborhood Exposure to Surface Water

Mercury

Analytical Results

(ug/L)		
0.1	0.5	
0.175	0.1	
0.1	0.5	
0.5	0.1	
0.08	0.5	
0.08	0.1	
0.5	0.5	
0.17	0.1	
0.1	0.5	
0.5	0.1	
0.1	0.5	
0.5	0.1	
0.5	0.5	
0.15	0.1	
0.1	0.1	
0.5	0.5	
0.5	0.08	
0.1	0.06	
0.5	0.1	
0.5	0.08	
0.1	0.055	
0.5	0.1	
0.5	0.5	
0.1	0.1	
0.5	0.5	
0.5	0.1	
0.1	0.15	
0.5	0.1	
0.5	0.5	
0.1	0.1	
0.08	0.055	
0.5	0.1	
0.5	0.5	
0.1	0.1	
0.5	0.5	
0.2	0.1	
0.5	0.5	
0.1	0.1	
0.5	0.1	
0.1	0.1	**only 4 detects**

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	80	Mean	0.27
Censored		Lognormal mean	0.28
Detection limit or PQL		Std. devn.	0.198790956
Method detection limit		Median	0.1
TOTAL	80	Min.	0.055
		Max.	0.5

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -5.9708. This lies outside the tabled values of 1.226 and -2.613
Reject normal distribution.
Y value is -8.0348. This lies outside the tabled values of 1.226 and -2.613

UCL (based on t-statistic) is 0.31

Nine Mile Occupational Soil (mg/kg)

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
4.4	1.4816	23.4	3.1527	7.3	1.9879	15200	9.6291	752	6.6227	972	6.8794
20.4	3.0155	99.6	4.6012	13.8	2.6247	29800	10.3023	566	6.3386	3580	8.1831
3.9	1.3610	26.5	3.2771	3.9	1.3610	15800	9.6678	750	6.6201	677	6.5177
5.3	1.6677	30.8	3.4275	9.4	2.2407	13700	9.5252	401	5.9940	1460	7.2862
2.1	0.7419	20.5	3.0204	3.6	1.2809	64300	11.0713	4010	8.2965	495	6.2046
1.2	0.1823	10.9	2.3888	0.85	-0.1625	19400	9.8730	664	6.4983	221	5.3982
1.3	0.2624	6.8	1.9169	3.8	1.3350	55400	10.9223	4000	8.2940	469	6.1506
145	4.9767	10.5	2.3514	0.1	-2.3026	16900	9.7351	543	6.2971	159	5.0689
1.8	0.5878	7.5	2.0149	2.4	0.8755	29800	10.3023	1010	6.9177	317	5.7589
105	4.6540	9.8	2.2824	1.4	0.3365	33200	10.4103	1980	7.5909	360	5.8861
4.7	1.5476	535	6.2823	0.86	-0.1508	27000	10.2036	1100	7.0031	373	5.9216
0.5	-0.6931	13	2.5649	4.1	1.4110	21100	9.9570	838	6.7310	544	6.2989
17.5	2.8622	484	6.1821	4.4	1.4816	22600	10.0257	917	6.8211	751	6.6214
1.5	0.4055	19.7	2.9806	3	1.0986	19700	9.8884	1490	7.3065	425	6.0521
0.5	-0.6931	19.9	2.9907	0.51	-0.6733	25800	10.1581	869	6.7673	245	5.5013
1.6	0.4700	153	5.0304	12	2.4849	27700	10.2292	916	6.8200	1590	7.3715
1.5	0.4055	20.4	3.0155	1.2	0.1823	12600	9.4415	947	6.8533	351	5.8608
1	0.0000	11.6	2.4510	1	0.0000	15800	9.6678	1530	7.3330	242	5.4889
1	0.0000	10.9	2.3888	20.7	3.0301	11500	9.3501	550	6.3099	3020	8.0130
2.6	0.9555	28.2	3.3393	1.8	0.5878	13700	9.5252	1240	7.1229	401	5.9940
3.7	1.3083	13.2	2.5802	7	1.9459	10100	9.2203	587	6.3750	953	6.8596
2	0.6931	11.7	2.4596	16.2	2.7850	12700	9.4494	1220	7.1066	2000	7.6009
2.3	0.8329	8.8	2.1748	8.6	2.1518	10400	9.2496	608	6.4102	1310	7.1778
1.7	0.5306	23	3.1355	17.5	2.8622	12400	9.4255	889	6.7901	3180	8.0646
2.4	0.8755	3.95	1.3737	5.7	1.7405	20000	9.9035	758	6.6307	997	6.9048
1.5	0.4055	9.2	2.2192	5.9	1.7750	9270	9.1345	980	6.8876	989	6.8967
2.8	1.0296	8.5	2.1401	3.8	1.3350	23800	10.0774	1200	7.0901	730	6.5930
8	2.0794	8.5	2.1401	6.3	1.8405	15500	9.6486	1510	7.3199	1110	7.0121
1.6	0.4700	7.8	2.0541	4.2	1.4351	25500	10.1464	1210	7.0984	792	6.6746
5.3	1.6677	9.1	2.2083	14.3	2.6603	19900	9.8985	2000	7.6009	1060	6.9660
22.2	3.1001	14.4	2.6672	1.9	0.6419	20300	9.9184	818	6.7069	899	6.8013
6.2	1.8245	4	1.3863	0.04	-3.2189	17100	9.7468	768	6.6438	86.6	4.4613
17.8	2.8792	18.2	2.9014	7.9	2.0669	18400	9.8201	753	6.6241	1280	7.1546
2.5	0.9163	131	4.8752	4.8	1.5686	39700	10.5891	1620	7.3902	1020	6.9276
4.2	1.4351	18.5	2.9178	5.9	1.7750	23500	10.0648	972	6.8794	1370	7.2226
1.4	0.3365	53.8	3.9853	20.9	3.0397	22600	10.0257	635	6.4536	3360	8.1197
34.4	3.5381	12.9	2.5572	3.6	1.2809	21700	9.9851	707	6.5610	926	6.8309
2.6	0.9555	13.9	2.6319	3.8	1.3350	18600	9.8309	1350	7.2079	877	6.7765
2.05	0.7178	9.1	2.2083	5.3	1.6677	17700	9.7813	552	6.3135	651	6.4785
10.1	2.3125	30.9	3.4308	13.5	2.6027	16600	9.7172	1190	7.0817	1900	7.5496
5.2	1.6487	24	3.1781	4	1.3863	35100	10.4660	2030	7.6158	1030	6.9373
4.9	1.5892	10.8	2.3795	7	1.9459	15200	9.6291	1600	7.3778	1020	6.9276
5.8	1.7579	19.7	2.9806	2.5	0.9163	17200	9.7527	1420	7.2584	604	6.4036
30.4	3.4144	16.4	2.7973	4.3	1.4586	23100	10.0476	1070	6.9754	806	6.6921
2.8	1.0296	32.2	3.4720	4.6	1.5261	17600	9.7757	1570	7.3588	762	6.6359
2.3	0.8329	10.8	2.3795	7.1	1.9601	16300	9.6989	1540	7.3395	1050	6.9565
8.5	2.1401	26.3	3.2696	14.7	2.6878	16900	9.7351	1150	7.0475	4130	8.3260
3.9	1.3610	8.1	2.0919	5.6	1.7228	23600	10.0690	1110	7.0121	740	6.6067
1.35	0.3001	7.3	1.9879	5.6	1.7228	37300	10.5267	2560	7.8478	866	6.7639
3.6	1.2809	35	3.5553	4	1.3863	21800	9.9897	1180	7.0733	944	6.8501
2.3	0.8329	10.2	2.3224	8.6	2.1518	19500	9.8782	385	5.9532	1180	7.0733
20.9	3.0397	10.8	2.3795	4.9	1.5892	18900	9.8469	1030	6.9373	1620	7.3902
7.1	1.9601	13.7	2.6174	5.5	1.7047	19600	9.8833	644	6.4677	693	6.5410
0.5	-0.6931	16.5	2.8034	3	1.0986	20000	9.9035	1100	7.0031	1300	7.1701
1.1	0.0953	35	3.5553	5.9	1.7750	20900	9.9475	848	6.7429	3530	8.1691
1.3	0.2624	34.1	3.5293	27.1	3.2995	19800	9.8934	606	6.4069	1940	7.5704
1.8	0.5878	12.6	2.5337	0.2	-1.6094	26800	10.1962	1730	7.4559	162	5.0876
1.8	0.5878	6.95	1.9387	7.9	2.0669	18600	9.8309	873	6.7719	782	6.6619
0.84	-0.1744	11	2.3979	0.59	-0.5276	19200	9.8627	817	6.7056	529	6.2710
17.1	2.8391	16.1	2.7788	4.8	1.5686	30800	10.3353	1880	7.5390	1010	6.9177
1.5	0.4055	12	2.4849	2.3	0.8329	19500	9.8782	812	6.6995	576	6.3561
1.1	0.0953	9.6	2.2618	0.04	-3.2189	23800	10.0774	1120	7.0211	102	4.6250
13.7	2.6174	38.6	3.6533	10.5	2.3514	18600	9.8309	757	6.6294	2280	7.7319
1.3	0.2624	14.4	2.6672	0.33	-1.1087	25100	10.1306	1530	7.3330	279	5.6312
2.3	0.8329	10.8	2.3795	0.235	-1.4482	24300	10.0982	1530	7.3330	350	5.8579
9.7	2.2721	47.9	3.8691	4.6	1.5261	24400	10.1023	1110	7.0121	2070	7.6353
14.7	2.6878	11.1	2.4069	0.5	-0.6931	25400	10.1425	1150	7.0475	196	5.2781
17.3	2.8507	17.8	2.8792	3.2	1.1632	37400	10.5294	1130	7.0300	545	6.3008
12	2.4849	11	2.3979	0.22	-1.5141	22800	10.0345	1030	6.9373	173	5.1533
19.1	2.9497	47.8	3.8670	6.5	1.8718	35600	10.4801	1310	7.1778	1860	7.5283
6.4	1.8563	35.5	3.5695	6.1	1.8083	28600	10.2612	1060	6.9660	1940	7.5704
20.2	3.0057	18.2	2.9014	12.1	2.4932	32900	10.4012	1530	7.3330	1930	7.5653
32.4	3.4782	26.9	3.2921	6.4	1.8563	30300	10.3189	1980	7.5909	1580	7.3652
11.6	2.4510	19.2	2.9549	4.2	1.4351	41700	10.6383	2100	7.6497	734	6.5985
16.7	2.8154	18	2.8904	5.3	1.6677	26700	10.1924	930	6.8352	1140	7.0388
16	2.7726	22.5	3.1135	7.5	2.0149	31200	10.3482	1640	7.4025	1240	7.1229
9.2	2.2192	28.9	3.3638	9.1	2.2083	28400	10.2541	1610	7.3840	1540	7.3395
14.6	2.6810	24.9	3.2149	5.6	1.7228	41100	10.6238	2320	7.7493	2050	7.6256

Nine Mile Occupational Soil (mg/kg)

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
17.9	2.8848	38.5	3.6507	7.9	2.0669	37300	10.5267	1600	7.3778	2720	7.9084
19.9	2.9907	46.6	3.8416	8.8	2.1748	16000	9.6803	1130	7.0300	2880	7.9655
29	3.3673	12.9	2.5572	4.7	1.5476	34100	10.4371	2300	7.7407	927	6.8320
3.3	1.1939	43.3	3.7682	6.8	1.9169	15500	9.6486	961	6.8680	1780	7.4844
24.5	3.1987	29.4	3.3810	7.8	2.0541	15300	9.6356	984	6.8916	2050	7.6256
1.5	0.4055	35.9	3.5807	12.5	2.5257	14800	9.6024	1070	6.9754	2300	7.7407
1.2	0.1823	33.6	3.5145	11.3	2.4248	15100	9.6225	935	6.8405	1870	7.5337
1.5	0.4055	13.9	2.6319	4.2	1.4351	32900	10.4012	871	6.7696	622	6.4329
1.5	0.4055	100	4.6052	12.5	2.5257	123000	11.7199	6580	8.7918	1650	7.4085
4.4	1.4816	16.5	2.8034	1.1	0.0953	31600	10.3609	1070	6.9754	198	5.2883
222	5.4027	11	2.3979	2	0.6931	20300	9.9184	872	6.7708	307	5.7268
5.9	1.7750	10	2.3026	1.5	0.4055	20600	9.9330	541	6.2934	226	5.4205
1.5	0.4055	12.3	2.5096	1.9	0.6419	18400	9.8201	828	6.7190	235	5.4596
1.6	0.4700	22.5	3.1135	6.3	1.8405	22000	9.9988	591	6.3818	1300	7.1701
1.2	0.1823	1150	7.0475	0.04	-3.2189	22400	10.0168	661	6.4938	421	6.0426
0.5	-0.6931	37.7	3.6297	5.8	1.7579	17800	9.7870	744	6.6120	926	6.8309
0.495	-0.7032	13.2	2.5802	2.7	0.9933	38200	10.5506	2530	7.8360	390	5.9661
4.7	1.5476	8.8	2.1748	1.5	0.4055	17300	9.7585	758	6.6307	287	5.6595
12.1	2.4932	13.3	2.5878	1.6	0.4700	19400	9.8730	807	6.6933	848	6.7429
1.9	0.6419	8.6	2.1518	1.4	0.3365	13800	9.5324	401	5.9940	242	5.4889
2.9	1.0647	8.7	2.1633	1.6	0.4700	18100	9.8037	580	6.3630	328	5.7930
3.4	1.2238	12.9	2.5572	4.3	1.4586	16300	9.6989	613	6.4184	435	6.0753
3.1	1.1314	37.6	3.6270	20.3	3.0106	20900	9.9475	607	6.4085	3030	8.0163
3.3	1.1939	10.9	2.3888	8.4	2.1282	28100	10.2435	1970	7.5858	792	6.6746
4.6	1.5261	19.3	2.9601	5.6	1.7228	38600	10.5610	1820	7.5066	502	6.2186
12.2	2.5014	14.1	2.6462	5.6	1.7228	34800	10.4574	1740	7.4616	716	6.5737
20.7	3.0301	16.8	2.8214	21.3	3.0587	28100	10.2435	920	6.8244	894	6.7957
36.4	3.5946	15.4	2.7344	5	1.6094	26200	10.1735	923	6.8276	646	6.4708
7.2	1.9741	17.1	2.8391	8.4	2.1282	23900	10.0816	1130	7.0300	899	6.8013
9	2.1972	32	3.4657	9.6	2.2618	39000	10.5713	1800	7.4955	2060	7.6305
3.4	1.2238	49	3.8918	7.8	2.0541	24600	10.1105	922	6.8265	3100	8.0392
17.5	2.8622	59.6	4.0877	7.4	2.0015	24200	10.0941	763	6.6373	2910	7.9759
24.9	3.2149	20.6	3.0253	5.3	1.6677	11000	9.3057	353	5.8665	1120	7.0211
4.5	1.5041	19.2	2.9549	5.2	1.6487	21500	9.9758	655	6.4846	1340	7.2004
3.2	1.1632	15.3	2.7279	6.9	1.9315	8120	9.0021	227	5.4250	972	6.8794
1	0.0000	36.2	3.5891	11.4	2.4336	20000	9.9035	469	6.1506	3110	8.0424
1.3	0.2624	21.7	3.0773	7	1.9459	7160	8.8763	242	5.4889	1380	7.2298
0.5	-0.6931	19.5	2.9704	6.6	1.8871	11100	9.3147	641	6.4630	1190	7.0817
2.4	0.8755	8	2.0794	15.1	2.7147	11400	9.3414	432	6.0684	2440	7.7998
2.4	0.8755	16.9	2.8273	0.1	-2.3026	22700	10.0301	466	6.1442	96	4.5643
4.2	1.4351	5.8	1.7579	13.4	2.5953	23600	10.0690	889	6.7901	2010	7.6059
0.5	-0.6931	16.5	2.8034	0.1	-2.3026	22100	10.0033	1160	7.0562	115	4.7449
3.7	1.3083	4.7	1.5476	16.9	2.8273	88400	11.3896	7540	8.9280	2680	7.8936
5.5	1.7047	9.7	2.2721	8.2	2.1041	16700	9.7232	647	6.4723	1330	7.1929
30.8	3.4275	7.9	2.0669	15.3	2.7279	19800	9.8934	1480	7.2998	2230	7.7098
1.3	0.2624	18.5	2.9178	0.1	-2.3026	40900	10.6189	3390	8.1286	80.6	4.3895
4.3	1.4586	25	3.2189	4.4	1.4816	18600	9.8309	1130	7.0300	474	6.1612
16.9	2.8273	17.1	2.8391	5	1.6094	27600	10.2256	1760	7.4731	1620	7.3902
7.5	2.0149	36.2	3.5891	24.1	3.1822	20600	9.9330	737	6.6026	4370	8.3825
5	1.6094	10.2	2.3224	3.8	1.3350	17800	9.7870	800	6.6846	166	5.1120
1.4	0.3365	14.1	2.6462	6.2	1.8245	19900	9.8985	973	6.8804	830	6.7214
3.6	1.2809	45.5	3.8177	5.8	1.7579	19900	9.8985	945	6.8512	1860	7.5283
1.2	0.1823	15.8	2.7600	11.5	2.4423	17100	9.7468	768	6.6438	1550	7.3460
5.5	1.7047	18.4	2.9124	12.2	2.5014	17800	9.7870	755	6.6267	2210	7.7007
1.35	0.3001	33	3.4965	6.6	1.8871	14800	9.6024	800	6.6846	979	6.8865
4.3	1.4586	19.4	2.9653	2.4	0.8755	16200	9.6928	697	6.5468	594	6.3869
1.1	0.0953	9.6	2.2618	4.7	1.5476	18300	9.8147	692	6.5396	765	6.6399
2.1	0.7419	19.8	2.9857	1.3	0.2624	25800	10.1581	2220	7.7053	732	6.5958
3.8	1.3350	8.4	2.1282	15.3	2.7279	18400	9.8201	803	6.6884	1240	7.1229
54.5	3.9982	8.3	2.1163	4.3	1.4586	20000	9.9035	948	6.8544	781	6.6606
2.9	1.0647	8.3	2.1163	7.9	2.0669	19600	9.8833	617	6.4249	947	6.8533
4.3	1.4586	8.3	2.1163	12.3	2.5096	19100	9.8574	807	6.6933	1060	6.9660
2.4	0.8755	9.7	2.2721	1.8	0.5878	19800	9.8934	626	6.4394	295	5.6870
3.3	1.1939	151	5.0173	18.2	2.9014	17900	9.7926	793	6.6758	2580	7.8555
4.2	1.4351	8.9	2.1861	2.3	0.8329	20400	9.9233	1250	7.1309	246	5.5053
44.2	3.7887	10.7	2.3702	3.6	1.2809	42900	10.6666	2760	7.9230	380	5.9402
11.7	2.4596	15.1	2.7147	1.3	0.2624	18400	9.8201	1020	6.9276	107	4.6728
4.4	1.4816	12.5	2.5257	2.6	0.9555	24300	10.0982	1240	7.1229	461	6.1334
41.8	3.7329	14.6	2.6810	1.4	0.3365	22800	10.0345	787	6.6682	107	4.6728
22	3.0910	10.3	2.3321	2.6	0.9555	28000	10.2400	1000	6.9078	297	5.6937
25.1	3.2229	16.7	2.8154	7.2	1.9741	25900	10.1620	1020	6.9276	1120	7.0211
8.2	2.1041	289	5.6664	8.1	2.0919	21800	9.9897	986	6.8937	2230	7.7098
0.55	-0.5978	11.5	2.4423	5.8	1.7579	17000	9.7410	558	6.3244	914	6.8178
18.5	2.9178	13.2	2.5802	4.8	1.5686	30200	10.3156	1730	7.4559	1020	6.9276
1.6	0.4700	23.7	3.1655	5.4	1.6864	18200	9.8092	802	6.6871	759	6.6320
3.4	1.2238	19.7	2.9806	6.5	1.8718	15500	9.6486	754	6.6254	1030	6.9373
8.7	2.1633	30.9	3.4308	10.9	2.3888	17600	9.7757	1250	7.1309	1790	7.4900
0.265	-1.3280	16.6	2.8094	8.1	2.0919	16100	9.6866	436	6.0776	1170	7.0648

Nine Mile Occupational Soil (mg/kg)

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
4.4	1.4816	9.9	2.2925	0.35	-1.0498	18300	9.8147	762	6.6359	89	4.4886
0.8	-0.2231	53	3.9703	11.8	2.4681	22400	10.0168	753	6.6241	2380	7.7749
1.15	0.1398	13	2.5649	4.2	1.4351	18300	9.8147	1030	6.9373	586	6.3733
1.05	0.0488	15.6	2.7473	3.7	1.3083	17100	9.7468	790	6.6720	471	6.1549
1.1	0.0953	26.1	3.2619	12	2.4849	16600	9.7172	802	6.6871	1590	7.3715
1	0.0000	9	2.1972	0.56	-0.5798	19300	9.8679	1190	7.0817	109	4.6913
1.5	0.4055	15.6	2.7473	6.1	1.8083	20300	9.9184	829	6.7202	648	6.4739
1.15	0.1398	24.4	3.1946	1.6	0.4700	16900	9.7351	597	6.3919	387	5.9584
1.3	0.2624	13.7	2.6174	4.1	1.4110	17000	9.7410	760	6.6333	428	6.0591
5.3	1.6677	15.2	2.7213	5.6	1.7228	20000	9.9035	1860	7.5283	667	6.5028
8.7	2.1633	11.2	2.4159	4	1.3863	21400	9.9711	1650	7.4085	400	5.9915
5.3	1.6677	14.7	2.6878	4.1	1.4110	20400	9.9233	1110	7.0121	394	5.9764
20.2	3.0057	10.4	2.3418	5.7	1.7405	18100	9.8037	1820	7.5066	524	6.2615
7.4	2.0015	8.7	2.1633	5.3	1.6677	20300	9.9184	1160	7.0562	492	6.1985
20.2	3.0057	9.5	2.2513	4	1.3863	22200	10.0078	2720	7.9084	511	6.2364
5.4	1.6864	14.2	2.6532	2.9	1.0647	28200	10.2471	1680	7.4265	615	6.4216
4.5	1.5041	31.7	3.4563	4.7	1.5476	14000	9.5468	710	6.5653	1090	6.9939
24.6	3.2027	14.3	2.6603	0.37	-0.9943	22400	10.0168	987	6.8947	324	5.7807
1.3	0.2624	27.3	3.3069	6.8	1.9169	10100	9.2203	542	6.2953	756	6.6280
2.4	0.8755	14.9	2.7014	0.88	-0.1278	5910	8.6844	665	6.4998	401	5.9940
4.3	1.4586	31.5	3.4500	5.8	1.7579	9770	9.1871	413	6.0234	1420	7.2584
7.3	1.9879	13.1	2.5726	5.7	1.7405	11300	9.3326	480	6.1738	1210	7.0984
8.4	2.1282	17	2.8332	9.8	2.2824	9880	9.1983	472	6.1570	1520	7.3265
2.6	0.9555	92.6	4.5283	12.8	2.5494	6600	8.7948	342	5.8348	2300	7.7407
0.7	-0.3567	6.9	1.9315	9.4	2.2407	12400	9.4255	747	6.6161	1590	7.3715
0.95	-0.0513	5.8	1.7579	2.2	0.7885	12400	9.4255	897	6.7991	459	6.1291
1.15	0.1398	10.2	2.3224	19.8	2.9857	16300	9.6989	924	6.8287	2510	7.8280
1.1	0.0953	14.8	2.6946	19.8	2.9857	17000	9.7410	1140	7.0388	803	6.6884
0.65	-0.4308	11.9	2.4765	12.2	2.5014	14200	9.5610	1060	6.9660	1800	7.4955
0.6	-0.5108	18.3	2.9069	13.3	2.5878	13600	9.5178	745	6.6134	1820	7.5066
1.2	0.1823	13.2	2.5802	2.3	0.8329	16400	9.7050	869	6.7673	424	6.0497
0.8	-0.2231	7.7	2.0412	2.7	0.9933	8780	9.0802	867	6.7650	382	5.9454
8.7	2.1633	15.5	2.7408	2.8	1.0296	22400	10.0168	1960	7.5807	513	6.2403
1.4	0.3365	11.6	2.4510	2.5	0.9163	22000	9.9988	1400	7.2442	440	6.0868
3.7	1.3083	8.8	2.1748	2.5	0.9163	14900	9.6091	1070	6.9754	442	6.0913
5.4	1.6864	14.7	2.6878	2.6	0.9555	20200	9.9134	2500	7.8240	346	5.8464
3.8	1.3350	17.5	2.8622	3.6	1.2809	18200	9.8092	1650	7.4085	545	6.3008
1.3	0.2624	2.9	1.0647	0.87	-0.1393	19100	9.8574	1730	7.4559	299	5.7004
2.2	0.7885	27.1	3.2995	6.2	1.8245	15400	9.6421	455	6.1203	1320	7.1854
3.1	1.1314	14.3	2.6603	2.2	0.7885	15700	9.6614	518	6.2500	2270	7.7275
3.4	1.2238	8.9	2.1861	1.1	0.0953	16200	9.6928	745	6.6134	408	6.0113
6.3	1.8405	19.1	2.9497	7.9	2.0669	15100	9.6225	508	6.2305	4420	8.3939
19.4	2.9653	10.7	2.3702	2	0.6931	16000	9.6803	521	6.2558	468	6.1485
22.9	3.1311	6.1	1.8083	1.4	0.3365	16100	9.6866	728	6.5903	344	5.8406
1.8	0.5878	14.5	2.6741	3.4	1.2238	15900	9.6741	539	6.2897	550	6.3099
2.1	0.7419	17.3	2.8507	3.7	1.3083	19300	9.8679	578	6.3596	647	6.4723
2.5	0.9163	17.7	2.8736	3.9	1.3610	20000	9.9035	754	6.6254	662	6.4953
4.2	1.4351	31.6	3.4532	5.6	1.7228	21600	9.9804	849	6.7441	773	6.6503
4.5	1.5041	62.6	4.1368	28.1	3.3358	17900	9.7926	639	6.4599	4550	8.4229
4.6	1.5261	73.1	4.2918	29.8	3.3945	18300	9.8147	652	6.4800	5160	8.5487
1.4	0.3365	14.1	2.6462	1.3	0.2624	24900	10.1226	1140	7.0388	309	5.7333
1.7	0.5306	22.9	3.1311	2.6	0.9555	14800	9.6024	551	6.3117	523	6.2596
2	0.6931	26.3	3.2696	3.3	1.1939	16200	9.6928	552	6.3135	601	6.3986
0.77	-0.2614	29	3.3673	4.8	1.5686	17100	9.7468	556	6.3208	825	6.7154
0.92	-0.0834	36.2	3.5891	12.5	2.5257	24400	10.1023	973	6.8804	2080	7.6401
1.2	0.1823	40.1	3.6914	13.5	2.6027	25100	10.1306	1310	7.1778	2280	7.7319
0.88	-0.1278	18.9	2.9392	2.1	0.7419	28200	10.2471	1580	7.3652	310	5.7366
0.95	-0.0513	19.5	2.9704	3.1	1.1314	22300	10.0123	1110	7.0121	423	6.0474
2.3	0.8329	19.9	2.9907	3.4	1.2238	22800	10.0345	1360	7.2152	472	6.1570
2.9	1.0647	10.9	2.3888	0.25	-1.3863	24600	10.1105	1390	7.2371	182	5.2040
13.7	2.6174	11	2.3979	0.54	-0.6162	26400	10.1811	1610	7.3840	188	5.2364
2.25	0.8109	21	3.0445	0.58	-0.5447	14100	9.5539	366	5.9026	224	5.4116
6.1	1.8083	7.1	1.9601	0.18	-1.7148	14600	9.5888	442	6.0913	130	4.8675
6.6	1.8871	7.3	1.9879	0.6	-0.5108	16300	9.6989	590	6.3801	183	5.2095
1	0.0000	7.6	2.0281	2.4	0.8755	20400	9.9233	961	6.8680	385	5.9532
1.1	0.0953	7.1	1.9601	0.56	-0.5798	24500	10.1064	963	6.8701	130	4.8675
1.5	0.4055	7.8	2.0541	0.69	-0.3711	26800	10.1962	1060	6.9660	154	5.0370
1	0.0000	10.4	2.3418	2.4	0.8755	12000	9.3927	831	6.7226	306	5.7236
1.5	0.4055	12.4	2.5177	0.9	-0.1054	16700	9.7232	950	6.8565	420	6.0403
1.7	0.5306	18.1	2.8959	1.3	0.2624	20500	9.9282	1060	6.9660	496	6.2066
2.1	0.7419	20.1	3.0007	3.8	1.3350	10400	9.2496	958	6.8648	799	6.6834
0.49	-0.7133	24.8	3.2108	4	1.3863	10500	9.2591	973	6.8804	1090	6.9939
0.49	-0.7133	15.2	2.7213	8.7	2.1633	10900	9.2965	1070	6.9754	1290	7.1624
1.7	0.5306	15.9	2.7663	12.2	2.5014	10300	9.2399	307	5.7268	1910	7.5549
0.97	-0.0305	29.4	3.3810	34	3.5264	11400	9.3414	378	5.9349	2460	7.8079
1	0.0000	19.7	2.9806	2.1	0.7419	11600	9.3588	520	6.2538	378	5.9349
1	0.0000	23.8	3.1697	2.9	1.0647	12200	9.4092	524	6.2615	440	6.0868
0.9	-0.1054	24	3.1781	3	1.0986	9790	9.1891	539	6.2897	443	6.0936

Nine Mile Occupational Soil (mg/kg)

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
1	0.0000	13.9	2.6319	0.1	-2.3026	10600	9.2686	545	6.3008	173	5.1533
1.75	0.5596	15.8	2.7600	1.8	0.5878	10900	9.2965	598	6.3936	468	6.1485
3.1	1.1314	20.3	3.0106	5.1	1.6292	11000	9.3057	606	6.4069	1030	6.9373
3.3	1.1939	10.4	2.3418	1.3	0.2624	11200	9.3237	658	6.4892	361	5.8889
3.6	1.2809	11.1	2.4069	2	0.6931	13100	9.4804	743	6.6107	411	6.0186
0.9	-0.1054	16.3	2.7912	3.3	1.1939	6340	8.7546	400	5.9915	502	6.2186
1.3	0.2624	19.8	2.9857	4.2	1.4351	7910	8.9759	518	6.2500	729	6.5917
1.4	0.3365	7.3	1.9879	5.3	1.6677	11800	9.3759	876	6.7754	1020	6.9276
1.5	0.4055	7.4	2.0015	5.6	1.7228	39800	10.5916	2140	7.6686	1050	6.9565
1.5	0.4055	7.7	2.0412	10.3	2.3321	41400	10.6310	2180	7.6871	1710	7.4442
1.7	0.5306	7.5	2.0149	3.1	1.1314	56800	10.9473	3050	8.0229	519	6.2519
1.9	0.6419	11.2	2.4159	5.9	1.7750	15700	9.6614	886	6.7867	739	6.6053
1.1	0.0953	12.1	2.4932	7.6	2.0281	19000	9.8522	958	6.8648	1110	7.0121
1.6	0.4700	6.2	1.8245	12.6	2.5337	19100	9.8574	1040	6.9470	1620	7.3902
2.3	0.8329	8	2.0794	15.3	2.7279	21500	9.9758	1140	7.0388	1760	7.4731
0.85	-0.1625	9.9	2.2925	19.5	2.9704	19100	9.8574	960	6.8669	2180	7.6871
0.95	-0.0513	7.7	2.0412	9.4	2.2407	19500	9.8782	969	6.8763	1540	7.3395
1.35	0.3001	11.7	2.4596	9.6	2.2618	20700	9.9379	1030	6.9373	1560	7.3524
6.6	1.8871	6.2	1.8245	11.2	2.4159	15000	9.6158	842	6.7358	1780	7.4844
57.4	4.0500	4.3	1.4586	4.2	1.4351	16600	9.7172	1020	6.9276	740	6.6067
72	4.2767	2.6	0.9555	4.6	1.5261	20000	9.9035	1030	6.9373	813	6.7007
1.8	0.5878	3.85	1.3481	4.8	1.5686	17700	9.7813	864	6.7616	900	6.8024
2.4	0.8755	3.95	1.3737	5.1	1.6292	18100	9.8037	1040	6.9470	958	6.8648
3	1.0986	6.9	1.9315	3.8	1.3350	18300	9.8147	1050	6.9565	683	6.5265
3.9	1.3610	8.3	2.1163	3.9	1.3610	20400	9.9233	663	6.4968	683	6.5265
6.8	1.9169	8.6	2.1518	3.9	1.3610	20900	9.9475	720	6.5793	747	6.6161
9.4	2.2407	6.1	1.8083	5.2	1.6487	22100	10.0033	784	6.6644	902	6.8046
9.9	2.2925	7.8	2.0541	6.3	1.8405	22200	10.0078	812	6.6995	982	6.8896
3	1.0986	10.1	2.3125	13	2.5649	51200	10.8435	2990	8.0030	1800	7.4955
4.1	1.4110	4.4	1.4816	1.9	0.6419	52000	10.8590	3020	8.0130	323	5.7777
5.7	1.7405	5.6	1.7228	3.3	1.1939	55400	10.9223	3210	8.0740	561	6.3297
3.2	1.1632	8.9	2.1861	7.7	2.0412	16500	9.7111	798	6.6821	1120	7.0211
4.3	1.4586	16.4	2.7973	3.8	1.3350	17100	9.7468	803	6.6884	2770	7.9266
4.8	1.5686	135	4.9053	23.5	3.1570	18000	9.7981	984	6.8916	7490	8.9213
1	0.0000	172	5.1475	38.1	3.6402	22100	10.0033	338	5.8230	8090	8.9984
1.3	0.2624	6.6	1.8871	1.5	0.4055	22200	10.0078	410	6.0162	362	5.8916
1.55	0.4383	9.6	2.2618	1.6	0.4700	25200	10.1346	540	6.2916	407	6.0088
3.7	1.3083	9.7	2.2721	1.8	0.5878	21700	9.9851	1540	7.3395	441	6.0890
94.4	4.5475	15	2.7081	3.7	1.3083	22000	9.9988	1720	7.4501	776	6.6542
117	4.7622	17.6	2.8679	2.3	0.8329	22800	10.0345	1820	7.5066	462	6.1356
118	4.7707	25.8	3.2504	3.2	1.1632	17400	9.7642	899	6.8013	480	6.1738
1.4	0.3365	33.3	3.5056	3.2	1.1632	18700	9.8363	1210	7.0984	656	6.4862
1.8	0.5878	9	2.1972	2.4	0.8755	19200	9.8627	1410	7.2513	725	6.5862
2.5	0.9163	10.4	2.3418	3.5	1.2528	16900	9.7351	1690	7.4325	762	6.6359
1.2	0.1823	15.3	2.7279	6.4	1.8563	18100	9.8037	1710	7.4442	1490	7.3065
0.8	-0.2231	13	2.5649	2.3	0.8329	19000	9.8522	1740	7.4616	508	6.2305
1.6	0.4700	13.6	2.6101	2.7	0.9933	16800	9.7291	1030	6.9373	616	6.4232
2.4	0.8755	15.1	2.7147	2.8	1.0296	17800	9.7870	1270	7.1468	696	6.5453
4.2	1.4351	9.2	2.2192	1.2	0.1823	17900	9.7926	1390	7.2371	305	5.7203
6.3	1.8405	10	2.3026	1.4	0.3365	18600	9.8309	1440	7.2724	420	6.0403
2.1	0.7419	10.1	2.3125	2.2	0.7885	18100	9.8037	1340	7.2004	434	6.0730
2.5	0.9163	11.1	2.4069	4.8	1.5686	18300	9.8147	1530	7.3330	633	6.4505
3.4	1.2238	50.6	3.9240	19.6	2.9755	18300	9.8147	1620	7.3902	3790	8.2401
1.8	0.5878	55.8	4.0218	24.6	3.2027	16000	9.6803	1210	7.0984	4150	8.3309
2.4	0.8755	57.8	4.0570	25.3	3.2308	19600	9.8833	1300	7.1701	4720	8.4596
2.8	1.0296	7.4	2.0015	1.2	0.1823	20000	9.9035	1590	7.3715	363	5.8944
0.405	-0.9039	8.2	2.1041	3	1.0986	30000	10.3090	1720	7.4501	675	6.5147
1	0.0000	12.8	2.5494	3.8	1.3350	30200	10.3156	1840	7.5175	871	6.7696
1.9	0.6419	6.2	1.8245	0.61	-0.4943	32400	10.3859	1890	7.5443	223	5.4072
2	0.6931	6.4	1.8563	0.91	-0.0943	23200	10.0519	1030	6.9373	229	5.4337
1.4	0.3365	8.7	2.1633	4.5	1.5041	27200	10.2110	1320	7.1854	683	6.5265
2.1	0.7419	11.8	2.4681	1.8	0.5878	37800	10.5401	1920	7.5601	621	6.4313
3.4	1.2238	13.6	2.6101	3.2	1.1632	16200	9.6928	1120	7.0211	770	6.6464
2.2	0.7885	14.4	2.6672	5.6	1.7228	16400	9.7050	1140	7.0388	1160	7.0562
3	1.0986	10.3	2.3321	2.5	0.9163	17500	9.7700	1150	7.0475	420	6.0403
1.85	0.6152	10.5	2.3514	4.8	1.5686	19400	9.8730	1020	6.9276	851	6.7464
2.8	1.0296	13.2	2.5802	5	1.6094	21200	9.9618	1030	6.9373	937	6.8427
2.9	1.0647	8.8	2.1748	4.5	1.5041	21200	9.9618	1040	6.9470	596	6.3902
3.7	1.3083	8.8	2.1748	5.1	1.6292	21300	9.9665	1090	6.9939	667	6.5028
1.5	0.4055	12.6	2.5337	6	1.7918	18600	9.8309	1020	6.9276	789	6.6708
4.8	1.5686	5.7	1.7405	1.4	0.3365	19800	9.8934	1360	7.2152	215	5.3706
9.3	2.2300	6	1.7918	1.9	0.6419	20000	9.9035	1380	7.2298	325	5.7838
0.68	-0.3857	6.4	1.8563	4.1	1.4110	16500	9.7111	673	6.5117	680	6.5221
1.5	0.4055	7.1	1.9601	5.6	1.7228	17500	9.7700	856	6.7523	827	6.7178
1.6	0.4700	7	1.9459	2.6	0.9555	18300	9.8147	1010	6.9177	370	5.9135
2.1	0.7419	7.2	1.9741	3.5	1.2528	24000	10.0858	1270	7.1468	499	6.2126
2.6	0.9555	8.2	2.1041	6.2	1.8245	25800	10.1581	1540	7.3395	933	6.8384
1.5	0.4055	9.4	2.2407	2.4	0.8755	28100	10.2435	1750	7.4674	412	6.0210

Nine Mile Occupational Soil (mg/kg)

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
1.9	0.6419	11	2.3979	2.4	0.8755	15100	9.6225	708	6.5624	428	6.0591
2.5	0.9163	18.9	2.9392	2.6	0.9555	15300	9.6356	812	6.6995	569	6.3439
0.95	-0.0513	13.9	2.6319	5.8	1.7579	16600	9.7172	1100	7.0031	2570	7.8517
1.1	0.0953	20.6	3.0253	8.5	2.1401	18800	9.8416	1160	7.0562	2830	7.9480
2	0.6931	22.8	3.1268	9.6	2.2618	16400	9.7050	712	6.5681	3270	8.0925
6.4	1.8563	18	2.8904	8.1	2.0919	17900	9.7926	819	6.7081	1360	7.2152
11.2	2.4159	19.3	2.9601	13.4	2.5953	18100	9.8037	1040	6.9470	3080	8.0327
13.2	2.5802	24.3	3.1905	15.8	2.7600	16400	9.7050	734	6.5985	3390	8.1286
1.2	0.1823	6.1	1.8083	0.03	-3.5066	16900	9.7351	947	6.8533	97.4	4.5788
1.2	0.1823	3.75	1.3218	0.66	-0.4155	19600	9.8833	1070	6.9754	202	5.3083
1.1	0.0953	4.2	1.4351	1.5	0.4055	27800	10.2328	1080	6.9847	278	5.6276
0.92	-0.0834	4.7	1.5476	4.8	1.5686	30000	10.3090	1240	7.1229	859	6.7558
9.3	2.2300	15.7	2.7537	6.3	1.8405	42200	10.6502	2170	7.6825	1290	7.1624
13.7	2.6174	16.5	2.8034	6.9	1.9315	23500	10.0648	1050	6.9565	1340	7.2004
17.8	2.8792	17.3	2.8507	7.6	2.0281	23800	10.0774	1080	6.9847	1390	7.2371
7.6	2.0281	8.7	2.1633	1.9	0.6419	24100	10.0900	1210	7.0984	495	6.2046
7.6	2.0281	9.4	2.2407	2.3	0.8329	20100	9.9085	656	6.4862	536	6.2841
14.4	2.6672	9.6	2.2618	3.3	1.1939	23700	10.0732	825	6.7154	681	6.5236
4.6	1.5261	7.1	1.9601	0.13	-2.0402	27700	10.2292	826	6.7166	116	4.7536
10.4	2.3418	10.4	2.3418	0.21	-1.5606	28200	10.2471	1300	7.1701	118	4.7707
11.5	2.4423	13.5	2.6027	0.29	-1.2379	23500	10.0648	679	6.5206	171	5.1417
19.7	2.9806	24.5	3.1987	4.1	1.4110	25300	10.1386	737	6.6026	1360	7.2152
2.5	0.9163	40.4	3.6988	5.1	1.6292	27200	10.2110	1110	7.0121	1440	7.2724
2.9	1.0647	40.5	3.7013	6.9	1.9315	23400	10.0605	1150	7.0475	1790	7.4900
8.9	2.1861	5.2	1.6487	0.08	-2.5257	23900	10.0816	1160	7.0562	174	5.1591
20.8	3.0350	6.2	1.8245	0.215	-1.5371	27000	10.2036	1240	7.1229	228	5.4293
28.4	3.3464	7.9	2.0669	0.78	-0.2485	31300	10.3514	792	6.6746	238	5.4723
29.2	3.3742	10.9	2.3888	1.9	0.6419	33500	10.4193	924	6.8287	388	5.9610
18.5	2.9178	10.7	2.3702	0.39	-0.9416	34900	10.4602	984	6.8916	162	5.0876
29.2	3.3742	11.3	2.4248	0.76	-0.2744	26200	10.1735	635	6.4536	202	5.3083
31.5	3.4500	12.8	2.5494	0.9	-0.1054	28600	10.2612	750	6.6201	220	5.3936
4	1.3863	6.1	1.8083	0.29	-1.2379	30400	10.3222	1340	7.2004	181	5.1985
5.7	1.7405	6.7	1.9021	0.39	-0.9416	36000	10.4913	1770	7.4787	217	5.3799
14.6	2.6810	11.4	2.4336	0.82	-0.1985	26600	10.1887	1410	7.2513	293	5.6802
19.9	2.9907	16.5	2.8034	7.5	2.0149	26900	10.1999	1520	7.3265	1120	7.0211
10.1	2.3125	17.5	2.8622	8.2	2.1041	41000	10.6213	2200	7.6962	1320	7.1854
20.6	3.0253	23	3.1355	9.2	2.2192	27700	10.2292	1300	7.1701	3530	8.1691
68.1	4.2210	18.6	2.9232	5.6	1.7228	37400	10.5294	2090	7.6449	993	6.9007
14.2	2.6532	19.8	2.9857	5.9	1.7750	42000	10.6454	2390	7.7790	1100	7.0031
18.2	2.9014	20.1	3.0007	10.9	2.3888	23900	10.0816	1110	7.0121	1570	7.3588
18.9	2.9392	5.3	1.6677	3.1	1.1314	34000	10.4341	1640	7.4025	613	6.4184
33.2	3.5025	7.4	2.0015	5.9	1.7750	47200	10.7621	1690	7.4325	953	6.8596
48.8	3.8877	9.5	2.2513	7.7	2.0412	19600	9.8833	1360	7.2152	1050	6.9565
124	4.8203	33.6	3.5145	11.8	2.4681	20500	9.9282	1440	7.2724	2140	7.6686
2.1	0.7419	7.1	1.9601	2.8	1.0296	21000	9.9523	1440	7.2724	501	6.2166
3.1	1.1314	7.7	2.0412	3.6	1.2809	18500	9.8255	1240	7.1229	568	6.3421
4.7	1.5476	13.2	2.5802	6.2	1.8245	19500	9.8782	1420	7.2584	966	6.8732
2.5	0.9163	14.8	2.6946	5.9	1.7750	21300	9.9665	1490	7.3065	1200	7.0901
3.4	1.2238	21.7	3.0773	7.2	1.9741	21600	9.9804	1560	7.3524	1570	7.3588
4.2	1.4351	25	3.2189	12.2	2.5014	15700	9.6614	731	6.5944	1660	7.4146
4.4	1.4816	9.7	2.2721	2.3	0.8329	17100	9.7468	889	6.7901	678	6.5191
0.5	-0.6931	11.9	2.4765	3	1.0986	17200	9.7527	1130	7.0300	710	6.5653
1.3	0.2624	12.4	2.5177	3.3	1.1939	14900	9.6091	780	6.6593	714	6.5709
1.7	0.5306	10.4	2.3418	3.6	1.2809	15800	9.6678	1080	6.9847	792	6.6746
1.2	0.1823	11.6	2.4510	7	1.9459	16100	9.6866	1110	7.0121	1060	6.9660
1.3	0.2624	26.7	3.2847	8.9	2.1861	16300	9.6989	592	6.3835	1340	7.2004
1.5	0.4055	47.6	3.8628	10.4	2.3418	16700	9.7232	703	6.5554	1750	7.4674
0.91	-0.0943	20.6	3.0253	4.4	1.4816	17000	9.7410	1040	6.9470	835	6.7274
1.1	0.0953	26.2	3.2658	4.8	1.5686	25300	10.1386	676	6.5162	949	6.8554
1.8	0.5878	40.1	3.6914	5.9	1.7750	29000	10.2751	1040	6.9470	1380	7.2298
1.4	0.3365	18.5	2.9178	9.9	2.2925	33600	10.4223	1170	7.0648	1350	7.2079
3.1	1.1314	23.6	3.1612	11.9	2.4765	29600	10.2955	963	6.8701	1680	7.4265
6.4	1.8563	25.6	3.2426	12.9	2.5572	33000	10.4043	984	6.8916	1890	7.5443
1.7	0.5306	21.4	3.0634	4.8	1.5686	38000	10.5453	1060	6.9660	924	6.8287
3.2	1.1632	35	3.5553	8.2	2.1041	39900	10.5941	1230	7.1148	1490	7.3065
4.1	1.4110	51.1	3.9338	8.5	2.1401	30800	10.3353	852	6.7476	1820	7.5066
4.6	1.5261	13.9	2.6319	1.5	0.4055	64400	11.0729	2040	7.6207	377	5.9322
2.3	0.8329	14	2.6391	3.4	1.2238	83300	11.3302	3360	8.1197	656	6.4862
5.4	1.6864	17.5	2.8622	6.2	1.8245	26400	10.1811	730	6.5930	881	6.7811
8.1	2.0919	16.9	2.8273	4.5	1.5041	54800	10.9114	1480	7.2998	621	6.4313
1.1	0.0953	18.4	2.9124	5.3	1.6677	89500	11.4020	2520	7.8320	640	6.4615
1.1	0.0953	19.6	2.9755	5.4	1.6864	17900	9.7926	575	6.3544	675	6.5147
2	0.6931	20.3	3.0106	7.2	1.9741	20400	9.9233	775	6.6529	755	6.6267
3.5	1.2528	10.5	2.3514	0.1	-2.3026	25900	10.1620	1110	7.0121	126	4.8363
7.9	2.0669	12.7	2.5416	0.95	-0.0513	31100	10.3450	1610	7.3840	268	5.5910
19	2.9444	12.9	2.5572	2.2	0.7885	31700	10.3641	2310	7.7450	339	5.8260
21.4	3.0634	10.8	2.3795	0.1	-2.3026	32200	10.3797	2400	7.7832	88.5	4.4830
48	3.8712	10.9	2.3888	0.59	-0.5276	11800	9.3759	114	4.7362	162	5.0876

Nine Mile Occupational Soil (mg/kg)

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
1.2	0.1823	11.5	2.4423	1.4	0.3365	11900	9.3843	220	5.3936	220	5.3936
1.7	0.5306	13.8	2.6247	0.04	-3.2189	14600	9.5888	546	6.3026	84.2	4.4332
2.1	0.7419	15	2.7081	0.04	-3.2189	16100	9.6866	712	6.5681	107	4.6728
3.2	1.1632	15	2.7081	1.7	0.5306	8910	9.0949	111	4.7095	237	5.4681
0.5	-0.6931	11.4	2.4336	2.8	1.0296	12800	9.4572	142	4.9558	915	6.8189
0.5	-0.6931	20.3	3.0106	5.1	1.6292	14100	9.5539	399	5.9890	1240	7.1229
3.1	1.1314	37.4	3.6217	8	2.0794	16400	9.7050	355	5.8721	1940	7.5704
2.1	0.7419	17.4	2.8565	1.8	0.5878	17100	9.7468	637	6.4568	616	6.4232
3	1.0986	18.4	2.9124	5.2	1.6487	21700	9.9851	660	6.4922	916	6.8200
8.1	2.0919	26.2	3.2658	5.8	1.7579	19700	9.8884	705	6.5582	964	6.8711
2.7	0.9933	26.8	3.2884	6.2	1.8245	27700	10.2292	851	6.7464	1150	7.0475
4.4	1.4816	14.7	2.6878	2	0.6931	31200	10.3482	885	6.7856	483	6.1800
9.9	2.2925	26.4	3.2734	2.1	0.7419	27200	10.2110	1440	7.2724	809	6.6958
9.2	2.2192	28.2	3.3393	5.6	1.7228	33000	10.4043	1950	7.5756	1020	6.9276
12.9	2.5572	10.8	2.3795	0.94	-0.0619	33400	10.4163	1980	7.5909	285	5.6525
14.8	2.6946	18.6	2.9232	4.4	1.4816	21400	9.9711	734	6.5985	802	6.6871
1.9	0.6419	22	3.0910	11.8	2.4681	24100	10.0900	851	6.7464	1530	7.3330
2.9	1.0647	6.4	1.8563	0.61	-0.4943	25000	10.1266	1060	6.9660	212	5.3566
3.7	1.3083	12.4	2.5177	5	1.6094	25000	10.1266	1410	7.2513	991	6.8987
5.9	1.7750	18.9	2.9392	15.2	2.7213	24400	10.1023	752	6.6227	2200	7.6962
1.8	0.5878	20.5	3.0204	10.4	2.3418	25500	10.1464	931	6.8363	1360	7.2152
3.1	1.1314	21.3	3.0587	14.7	2.6878	29200	10.2819	1480	7.2998	1520	7.3265
7.9	2.0669	26.5	3.2771	14.8	2.6946	23600	10.0690	811	6.6983	1570	7.3588
2.8	1.0296	8.8	2.1748	2.5	0.9163	23600	10.0690	1250	7.1309	530	6.2729
1.7	0.5306	8.9	2.1861	2.9	1.0647	39300	10.5790	1540	7.3395	692	6.5396
90.2	4.5020	9	2.1972	7.2	1.9741	21100	9.9570	592	6.3835	787	6.6682
2.15	0.7655	10	2.3026	9.8	2.2824	42000	10.6454	828	6.7190	823	6.7130
65.4	4.1805	9.8	2.2824	1.4	0.3365	49500	10.8097	1640	7.4025	307	5.7268
237	5.4681	12.5	2.5257	1.9	0.6419	7440	8.9146	141	4.9488	434	6.0730
1.6	0.4700	14.1	2.6462	5.7	1.7405	9050	9.1105	193	5.2627	775	6.6529
2.3	0.8329	12.5	2.5257	3.8	1.3350	9560	9.1653	273	5.6095	599	6.3953
3.3	1.1939	15.4	2.7344	6.8	1.9169	6930	8.8436	102	4.6250	1150	7.0475
0.5	-0.6931	23.8	3.1697	10.4	2.3418	7180	8.8791	103	4.6347	1350	7.2079
0.5	-0.6931	18	2.8904	11.7	2.4596	7600	8.9359	141	4.9488	1090	6.9939
0.5	-0.6931	19.8	2.9857	30.1	3.4045	4160	8.3333	70.8	4.2599	2050	7.6256
0.5	-0.6931	20.9	3.0397	31.9	3.4626	4420	8.3939	86.1	4.4555	3090	8.0359
1.4	0.3365	23.3	3.1485	7.4	2.0015	5120	8.5409	152	5.0239	1380	7.2298
2	0.6931	29.6	3.3878	12.9	2.5572	5950	8.6911	169	5.1299	2010	7.6059
2.5	0.9163	35.8	3.5779	21.2	3.0540	5310	8.5773	85.5	4.4485	2830	7.9480
0.485	-0.7236	16.4	2.7973	2.4	0.8755	6150	8.7242	110	4.7005	435	6.0753
1.3	0.2624	16.5	2.8034	3.1	1.1314	9430	9.1517	182	5.2040	607	6.4085
2.4	0.8755	17.3	2.8507	3.2	1.1632	18800	9.8416	492	6.1985	638	6.4583
0.485	-0.7236	21.9	3.0865	4.2	1.4351	22000	9.9988	547	6.3044	751	6.6214
0.5	-0.6931	15.9	2.7663	2.5	0.9163	24800	10.1186	559	6.3261	477	6.1675
0.5	-0.6931	16.8	2.8214	3.2	1.1632	20200	9.9134	580	6.3630	549	6.3081
2	0.6931	19.4	2.9653	9.5	2.2513	21000	9.9523	675	6.5147	1210	7.0984
2.4	0.8755	16.7	2.8154	3.5	1.2528	21200	9.9618	904	6.8068	664	6.4983
2.5	0.9163	17.8	2.8792	5	1.6094	21100	9.9570	1710	7.4442	1020	6.9276
6.2	1.8245	51.3	3.9377	9.1	2.2083	26900	10.1999	2260	7.7231	1660	7.4146
8.3	2.1163	14.3	2.6603	2.8	1.0296	75200	11.2279	6500	8.7796	443	6.0936
26.8	3.2884	32.5	3.4812	2.9	1.0647	16800	9.7291	651	6.4785	1250	7.1309
1.3	0.2624	56.7	4.0378	7.6	2.0281	17400	9.7642	668	6.5043	1570	7.3588
1.5	0.4055	6.2	1.8245	12.7	2.5416	20200	9.9134	1110	7.0121	2360	7.7664
6	1.7918	6.3	1.8405	15.5	2.7408	36000	10.4913	3010	8.0097	2450	7.8038
13.6	2.6101	7.9	2.0669	17.1	2.8391	19600	9.8833	841	6.7346	2750	7.9194
1.2	0.1823	2.9	1.0647	4.3	1.4586	21500	9.9758	995	6.9027	1100	7.0031
1.3	0.2624	3.1	1.1314	5.8	1.7579	21900	9.9942	1100	7.0031	1250	7.1309
3.3	1.1939	4.1	1.4110	6	1.7918	9350	9.1431	414	6.0259	1570	7.3588
1	0.0000	1.9	0.6419	11.7	2.4596	23700	10.0732	1320	7.1854	1550	7.3460
1.2	0.1823	3.3	1.1939	14.8	2.6946	24200	10.0941	1390	7.2371	1980	7.5909
0.8	-0.2231	4.1	1.4110	17.5	2.8622	18400	9.8201	495	6.2046	2430	7.7956
0.85	-0.1625	4.2	1.4351	27	3.2958	18500	9.8255	727	6.5889	3370	8.1227
1.1	0.0953	1.8	0.5878	6.7	1.9021	20600	9.9330	866	6.7639	1220	7.1066
0.55	-0.5978	2.1	0.7419	13.5	2.6027	14600	9.5888	441	6.0890	2270	7.7275
0.6	-0.5108	5.8	1.7579	15.1	2.7147	15600	9.6550	501	6.2166	2480	7.8160
0.6	-0.5108	13.2	2.5802	0.1	-2.3026	15700	9.6614	557	6.3226	79.6	4.3770
0.7	-0.3567	16.6	2.8094	1.1	0.0953	16000	9.6803	876	6.7754	195	5.2730
0.95	-0.0513	17.9	2.8848	6.2	1.8245	16800	9.7291	752	6.6227	1150	7.0475
1.5	0.4055	16.1	2.7788	2.7	0.9933	17400	9.7642	789	6.6708	317	5.7589
1.6	0.4700	17.4	2.8565	3.8	1.3350	18200	9.8092	844	6.7382	399	5.9890
1.05	0.0488	18.2	2.9014	3.9	1.3610	15100	9.6225	709	6.5639	451	6.1115
1.15	0.1398	12.4	2.5177	5.5	1.7047	17500	9.7700	987	6.8947	871	6.7696
2	0.6931	13.6	2.6101	7.1	1.9601	17500	9.7700	1220	7.1066	1190	7.0817
1.2	0.1823	31	3.4340	21.8	3.0819	18000	9.7981	600	6.3969	3830	8.2506
1.3	0.2624	10.4	2.3418	2.5	0.9163	18900	9.8469	849	6.7441	149	5.0039
1.7	0.5306	11.2	2.4159	2.7	0.9933	20300	9.9184	899	6.8013	165	5.1059
1.8	0.5878	14.5	2.6741	3.4	1.2238	17800	9.7870	676	6.5162	897	6.7991
2.3	0.8329	39.5	3.6763	5.4	1.6864	19000	9.8522	740	6.6067	1640	7.4025

Nine Mile Occupational Soil (mg/kg)

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
2.4	0.8755	11.8	2.4681	1.4	0.3365	20100	9.9085	845	6.7393	170	5.1358
2.4	0.8755	14.9	2.7014	1.9	0.6419	17300	9.7585	1060	6.9660	217	5.3799
3.3	1.1939	15.1	2.7147	4.9	1.5892	17900	9.7926	1230	7.1148	733	6.5971
4.6	1.5261	8.4	2.1282	0.045	-3.1011	18700	9.8363	1270	7.1468	32.8	3.4904
3.5	1.2528	18.4	2.9124	3	1.0986	19200	9.8627	1340	7.2004	194	5.2679
4.6	1.5261	18.4	2.9124	3.4	1.2238	19500	9.8782	628	6.4425	204	5.3181
7.8	2.0541	8.1	2.0919	2.4	0.8755	20900	9.9475	737	6.6026	653	6.4816
3.1	1.1314	16.7	2.8154	3.6	1.2809	21700	9.9851	901	6.8035	718	6.5765
3.2	1.1632	49.2	3.8959	4.7	1.5476	19100	9.8574	907	6.8101	780	6.6593
3.4	1.2238	5.5	1.7047	0.49	-0.7133	20400	9.9233	924	6.8287	181	5.1985
2.7	0.9933	5.9	1.7750	0.54	-0.6162	20400	9.9233	1010	6.9177	188	5.2364
14.6	2.6810	6.2	1.8245	0.54	-0.6162	18800	9.8416	900	6.8024	191	5.2523
28.2	3.3393	6.6	1.8871	2.6	0.9555	19600	9.8833	1010	6.9177	432	6.0684
9.8	2.2824	8	2.0794	15.4	2.7344	20100	9.9085	1050	6.9565	1210	7.0984
25.3	3.2308	8.5	2.1401	24.8	3.2108	21100	9.9570	1350	7.2079	1710	7.4442
29.1	3.3707	8.7	2.1633	27.5	3.3142	24600	10.1105	1600	7.3778	2040	7.6207
1.7	0.5306	5.9	1.7750	7.5	2.0149	25500	10.1464	2010	7.6059	1260	7.1389
4	1.3863	6.5	1.8718	8.4	2.1282	13200	9.4880	557	6.3226	1440	7.2724
29.3	3.3776	11.2	2.4159	31.8	3.4595	16400	9.7050	641	6.4630	2510	7.8280
13.3	2.5878	5.3	1.6677	0.94	-0.0619	23300	10.0562	917	6.8211	393	5.9738
59.2	4.0809	5.6	1.7228	2.4	0.8755	18900	9.8469	608	6.4102	685	6.5294
69.7	4.2442	6.9	1.9315	5.3	1.6677	23300	10.0562	1180	7.0733	972	6.8794
96.6	4.5706	9.4	2.2407	0.58	-0.5447	23600	10.0690	1320	7.1854	81.1	4.3957
24.6	3.2027	11.5	2.4423	1.1	0.0953	21900	9.9942	914	6.8178	166	5.1120
242	5.4889	12.4	2.5177	2	0.6931	26400	10.1811	1180	7.0733	323	5.7777
256	5.5452	9.5	2.2513	2.5	0.9163	27600	10.2256	1250	7.1309	353	5.8665
1.15	0.1398	10.1	2.3125	3.9	1.3610	29800	10.3023	1380	7.2298	418	6.0355
2	0.6931	10.2	2.3224	4.2	1.4351	41200	10.6262	1370	7.2226	443	6.0936
2.7	0.9933	10.5	2.3514	4.5	1.5041	48000	10.7790	1990	7.5959	477	6.1675
0.9	-0.1054	16.5	2.8034	0.77	-0.2614	49900	10.8178	2200	7.6962	70.9	4.2613
1.1	0.0953	20.2	3.0057	1.2	0.1823	18900	9.8469	712	6.5681	102	4.6250
2.05	0.7178	21.9	3.0865	2.5	0.9163	29500	10.2921	953	6.8596	191	5.2523
1.4	0.3365	12.3	2.5096	1.9	0.6419	27200	10.2110	993	6.9007	280	5.6348
6.4	1.8563	13	2.5649	18.4	2.9124	19000	9.8522	679	6.5206	793	6.6758
3.2	1.1632	15.1	2.7147	97	4.5747	19500	9.8782	666	6.5013	2700	7.9010
5.7	1.7405	10	2.3026	3.9	1.3610	23000	10.0432	1050	6.9565	389	5.9636
1.1	0.0953	10.2	2.3224	6.1	1.8083	25100	10.1306	1000	6.9078	444	6.0958
0.9	-0.1054	10.4	2.3418	6.4	1.8563	23500	10.0648	1060	6.9660	460	6.1312
0.75	-0.2877	13.3	2.5878	5.9	1.7750	21200	9.9618	843	6.7370	1020	6.9276
0.55	-0.5978	17.8	2.8792	23.3	3.1485	28000	10.2400	941	6.8469	2810	7.9409
2	0.6931	26	3.2581	27.9	3.3286	17800	9.7870	563	6.3333	2910	7.9759
2.9	1.0647	8.2	2.1041	11.2	2.4159	36000	10.4913	919	6.8233	1600	7.3778
0.95	-0.0513	16.5	2.8034	22.4	3.1091	30700	10.3320	768	6.6438	2300	7.7407
0.75	-0.2877	18.9	2.9392	30.4	3.4144	21800	9.9897	723	6.5834	3720	8.2215
0.85	-0.1625	11.2	2.4159	2.8	1.0296	33400	10.4163	1080	6.9847	434	6.0730
1.3	0.2624	18.5	2.9178	4.2	1.4351	24100	10.0900	816	6.7044	845	6.7393
1.3	0.2624	35.2	3.5610	16.7	2.8154	18700	9.8363	948	6.8544	1970	7.5858
1.15	0.1398	15.5	2.7408	3.7	1.3083	16600	9.7172	619	6.4281	523	6.2596
0.55	-0.5978	28.4	3.3464	5.4	1.6864	19900	9.8985	989	6.8967	1170	7.0648
1.1	0.0953	30.7	3.4243	15.7	2.7537	16900	9.7351	784	6.6644	1470	7.2930
0.65	-0.4308	45.3	3.8133	18.4	2.9124	15600	9.6550	767	6.6425	1600	7.3778
0.8	-0.2231	31.4	3.4468	7.1	1.9601	20100	9.9085	862	6.7593	2920	7.9793
1.05	0.0488	94.2	4.5454	20.2	3.0057	19500	9.8782	724	6.5848	3000	8.0064
0.6	-0.5108	121	4.7958	24.6	3.2027	21400	9.9711	1260	7.1389	4340	8.3756
0.65	-0.4308	12.9	2.5572	2.2	0.7885	18200	9.8092	805	6.6908	386	5.9558
1.25	0.2231	13.8	2.6247	6.1	1.8083	16200	9.6928	723	6.5834	1040	6.9470
0.8	-0.2231	15.2	2.7213	7.9	2.0669	20000	9.9035	718	6.5765	1220	7.1066
0.9	-0.1054	11.8	2.4681	2.7	0.9933	20400	9.9233	730	6.5930	347	5.8493
4.5	1.5041	13	2.5649	2.9	1.0647	21700	9.9851	759	6.6320	389	5.9636
3.3	1.1939	14	2.6391	12.1	2.4932	18400	9.8201	821	6.7105	1480	7.2998
3.6	1.2809	11.1	2.4069	8.1	2.0919	21200	9.9618	905	6.8079	1130	7.0300
14.5	2.6741	25.2	3.2268	9	2.1972	19200	9.8627	715	6.5723	1190	7.0817
7.1	1.9601	15.9	2.7663	2.9	1.0647	19900	9.8985	2110	7.6544	357	5.8777
3.3	1.1939	14.3	2.6603	22.1	3.0956	21400	9.9711	1350	7.2079	2520	7.8320
3.2	1.1632	9.9	2.2925	2.8	1.0296	20500	9.9282	836	6.7286	407	6.0088
84.1	4.4320	9.4	2.2407	2.6	0.9555	22400	10.0168	2490	7.8200	423	6.0474
25	3.2189	9.4	2.2407	1.9	0.6419	22200	10.0078	2050	7.6256	333	5.8081
35.4	3.5667	16.7	2.8154	1.4	0.3365	20400	9.9233	1430	7.2654	364	5.8972
8.8	2.1748	49	3.8918	7	1.9459	18900	9.8469	1080	6.9847	1630	7.3963
15.6	2.7473	26.2	3.2658	8.2	2.1041	19100	9.8574	2630	7.8747	1620	7.3902
10.8	2.3795	11.8	2.4681	3	1.0986	21600	9.9804	2630	7.8747	390	5.9661
6.4	1.8563	8	2.0794	1.2	0.1823	21600	9.9804	2080	7.6401	285	5.6525
4.3	1.4586	12.5	2.5257	3.8	1.3350	19800	9.8934	2220	7.7053	470	6.1527
1.8	0.5878	14.4	2.6672	4.9	1.5892	21000	9.9523	3070	8.0294	593	6.3852
1.25	0.2231	13	2.5649	2.9	1.0647	21400	9.9711	3070	8.0294	400	5.9915
1	0.0000	11.3	2.4248	3.4	1.2238	34700	10.4545	1860	7.5283	599	6.3953
0.255	-1.3665	12.4	2.5177	3.2	1.1632	31600	10.3609	2270	7.7275	489	6.1924
1.3	0.2624	14.7	2.6878	3.4	1.2238	31100	10.3450	2010	7.6059	414	6.0259

Nine Mile Occupational Soil (mg/kg)

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
0.475	-0.7444	9.6	2.2618	1.6	0.4700	13100	9.4804	543	6.2971	302	5.7104
0.445	-0.8097	7.1	1.9601	1.7	0.5306	11200	9.3237	440	6.0868	317	5.7589
15.9	2.7663	8.1	2.0919	2.6	0.9555	7410	8.9106	205	5.3230	419	6.0379
26.7	3.2847	8.5	2.1401	1.6	0.4700	10800	9.2873	339	5.8260	383	5.9480
3.7	1.3083	8.4	2.1282	1.4	0.3365	6360	8.7578	166	5.1120	386	5.9558
8.4	2.1282	9.2	2.2192	3.5	1.2528	6250	8.7403	133	4.8903	484	6.1821
7.4	2.0015	9.3	2.2300	2.8	1.0296	5410	8.5960	45.6	3.8199	501	6.2166
2.7	0.9933	7.7	2.0412	1.7	0.5306	12600	9.4415	462	6.1356	377	5.9322
4	1.3863	12.2	2.5014	2.5	0.9163	19500	9.8782	790	6.6720	618	6.4265
1.2	0.1823	12.5	2.5257	1.4	0.3365	6300	8.7483	164	5.0999	423	6.0474
0.55	-0.5978	14.4	2.6672	0.99	-0.0101	10100	9.2203	552	6.3135	260	5.5607
0.7	-0.3567	45	3.8067	10.2	2.3224	9760	9.1860	350	5.8579	1530	7.3330
0.55	-0.5978	22.1	3.0956	5.5	1.7047	8550	9.0537	1470	7.2930	986	6.8937
0.49	-0.7133	13.1	2.5726	2.3	0.8329	7830	8.9657	1850	7.5229	483	6.1800
0.42	-0.8675	11.6	2.4510	1.2	0.1823	6650	8.8024	281	5.6384	264	5.5759
0.485	-0.7236	46.1	3.8308	11.1	2.4069	4780	8.4722	84.4	4.4356	952	6.8586
0.435	-0.8324	29.9	3.3979	4.7	1.5476	14500	9.5819	868	6.7662	714	6.5709
0.33	-1.1087	29.5	3.3844	6.2	1.8245	16000	9.6803	664	6.4983	661	6.4938
0.55	-0.5978	20.9	3.0397	8.2	2.1041	15500	9.6486	360	5.8861	1460	7.2862
0.55	-0.5978	23.7	3.1655	4.4	1.4816	15500	9.6486	1000	6.9078	1140	7.0388
6.4	1.8563	15.2	2.7213	1.9	0.6419	15900	9.6741	1100	7.0031	699	6.5497
0.6	-0.5108	12.5	2.5257	6	1.7918	16200	9.6928	786	6.6670	1410	7.2513
0.395	-0.9289	10.7	2.3702	3.8	1.3350	15900	9.6741	797	6.6809	1120	7.0211
0.65	-0.4308	10.1	2.3125	3.8	1.3350	16200	9.6928	1160	7.0562	1090	6.9939
0.6	-0.5108	11.8	2.4681	6.3	1.8405	16600	9.7172	855	6.7511	1130	7.0300
0.315	-1.1552	9.8	2.2824	5	1.6094	18800	9.8416	789	6.6708	850	6.7452
1.1	0.0953	5.3	1.6677	7.4	2.0015	15800	9.6678	891	6.7923	992	6.8997
1.35	0.3001	6.7	1.9021	8.2	2.1041	15600	9.6550	655	6.4846	1230	7.1148
1.05	0.0488	5.7	1.7405	6.2	1.8245	14600	9.5888	348	5.8522	866	6.7639
1.8	0.5878	4.4	1.4816	4.8	1.5686	16800	9.7291	814	6.7020	809	6.6958
1.5	0.4055	5	1.6094	4.1	1.4110	16500	9.7111	707	6.5610	708	6.5624
4.6	1.5261	16	2.7726	16.7	2.8154	16900	9.7351	1450	7.2793	2480	7.8160
5	1.6094	28.7	3.3569	21.4	3.0634	13800	9.5324	1130	7.0300	3030	8.0163
5.9	1.7750	5.9	1.7750	19.5	2.9704	16000	9.6803	835	6.7274	2220	7.7053
4.6	1.5261	15.5	2.7408	15.1	2.7147	15200	9.6291	778	6.6567	2330	7.7536
3.4	1.2238	11.1	2.4069	13.6	2.6101	20300	9.9184	1250	7.1309	2040	7.6207
4	1.3863	8.1	2.0919	14.2	2.6532	15400	9.6421	924	6.8287	2290	7.7363
3.7	1.3083	18.1	2.8959	20.6	3.0253	19700	9.8884	1000	6.9078	3080	8.0327
2.4	0.8755	11.9	2.4765	17.7	2.8736	20100	9.9085	1460	7.2862	2160	7.6779
9.5	2.2513	5.7	1.7405	11.1	2.4069	19900	9.8985	1400	7.2442	1430	7.2654
1.35	0.3001	16.5	2.8034	2.2	0.7885	19100	9.8574	1370	7.2226	417	6.0331
0.95	-0.0513	10.9	2.3888	0.68	-0.3857	18700	9.8363	1300	7.1701	217	5.3799
1	0.0000	8	2.0794	0.27	-1.3093	18700	9.8363	1930	7.5653	254	5.5373
2.5	0.9163	8.5	2.1401	1.9	0.6419	21800	9.9897	2520	7.8320	339	5.8260
2	0.6931	7.9	2.0669	1.6	0.4700	24100	10.0900	1820	7.5066	456	6.1225
1.7	0.5306	7.4	2.0015	0.83	-0.1863	23700	10.0732	1840	7.5175	310	5.7366
0.67	-0.4005	6.7	1.9021	0.66	-0.4155	25600	10.1503	2470	7.8120	329	5.7961
1.6	0.4700	9.8	2.2824	1.6	0.4700	25000	10.1266	2230	7.7098	345	5.8435
1.7	0.5306	8.6	2.1518	0.58	-0.5447	17900	9.7926	379	5.9375	211	5.3519
0.67	-0.4005	97.7	4.5819	0.42	-0.8675	18800	9.8416	788	6.6695	200	5.2983
0.335	-1.0936	20.6	3.0253	1.6	0.4700	18600	9.8309	732	6.5958	327	5.7900
15.8	2.7600	9	2.1972	0.14	-1.9661	19000	9.8522	734	6.5985	183	5.2095
3.4	1.2238	6.7	1.9021	0.04	-3.2189	18300	9.8147	702	6.5539	169	5.1299
3.8	1.3350	16.8	2.8214	3.7	1.3083	18400	9.8201	750	6.6201	600	6.3969
3.9	1.3610	11.9	2.4765	0.7	-0.3567	21400	9.9711	833	6.7250	277	5.6240
5.8	1.7579	8.1	2.0919	0.18	-1.7148	20700	9.9379	863	6.7604	177	5.1761
76.8	4.3412	6.7	1.9021	0.35	-1.0498	19400	9.8730	865	6.7627	244	5.4972
0.95	-0.0513	9.6	2.2618	0.08	-2.5257	31600	10.3609	1770	7.4787	192	5.2575
0.95	-0.0513	8.4	2.1282	0.04	-3.2189	19300	9.8679	1230	7.1148	162	5.0876
1.3	0.2624	14.4	2.6672	1	0.0000	20200	9.9134	1370	7.2226	824	6.7142
1.8	0.5878	10.7	2.3702	0.52	-0.6539	20600	9.9330	1490	7.3065	395	5.9789
1.8	0.5878	13.7	2.6174	0.56	-0.5798	17500	9.7700	1070	6.9754	533	6.2785
7.2	1.9741	12.6	2.5337	0.66	-0.4155	26200	10.1735	1500	7.3132	370	5.9135
0.405	-0.9039	12.4	2.5177	0.53	-0.6349	11100	9.3147	468	6.1485	329	5.7961
1.3	0.2624	12.5	2.5257	0.66	-0.4155	12200	9.4092	613	6.4184	334	5.8111
2.3	0.8329	11.4	2.4336	0.83	-0.1863	12200	9.4092	813	6.7007	401	5.9940
2.3	0.8329	10.4	2.3418	3.1	1.1314	12300	9.4174	846	6.7405	586	6.3733
2.6	0.9555	9.5	2.2513	3.8	1.3350	12100	9.4010	789	6.6708	807	6.6933
1.3	0.2624	10.3	2.3321	1.9	0.6419	17500	9.7700	747	6.6161	681	6.5236
1.6	0.4700	12	2.4849	1.7	0.5306	3190	8.0678	212	5.3566	461	6.1334
1.8	0.5878	10.1	2.3125	1.6	0.4700	13500	9.5104	749	6.6187	516	6.2461
1.8	0.5878	10	2.3026	1.2	0.1823	13600	9.5178	937	6.8427	390	5.9661
1.3	0.2624	9.1	2.2083	2	0.6931	13300	9.4955	937	6.8427	473	6.1591
1.6	0.4700	10	2.3026	2.2	0.7885	12800	9.4572	705	6.5582	524	6.2615
1.8	0.5878	10.7	2.3702	3.3	1.1939	13500	9.5104	842	6.7358	618	6.4265
1.9	0.6419	9	2.1972	1.9	0.6419	14300	9.5680	907	6.8101	356	5.8749
4.5	1.5041	9.8	2.2824	1.3	0.2624	14300	9.5680	942	6.8480	242	5.4889
8.1	2.0919	11.6	2.4510	2.5	0.9163	12800	9.4572	771	6.6477	562	6.3315

Nine Mile Occupational Soil (mg/kg)

Antimony	LN[Sb]	Arsenic	LN[As]	Cadmium	LN[Cd]	Iron	LN[Fe]	Manganese	LN[Mn]	Zinc	LN[Zn]
6.2	1.8245	14.9	2.7014	3.4	1.2238	13000	9.4727	780	6.6593	720	6.5793
5.5	1.7047	11.6	2.4510	1.5	0.4055	13400	9.5030	780	6.6593	238	5.4723
1	0.0000	9.4	2.2407	1.6	0.4700	13600	9.5178	799	6.6834	350	5.8579
1.05	0.0488	49.7	3.9060	12.8	2.5494	12600	9.4415	749	6.6187	2150	7.6732
0.255	-1.3665	14.2	2.6532	7.9	2.0669	20800	9.9427	1300	7.1701	1090	6.9939
0.475	-0.7444	15.3	2.7279	8.1	2.0919	24200	10.0941	1430	7.2654	1130	7.0300
1.45	0.3716	15.5	2.7408	9.4	2.2407	24400	10.1023	1410	7.2513	1340	7.2004
0.55	-0.5978	15.6	2.7473	5.8	1.7579	18700	9.8363	1140	7.0388	875	6.7742
1.65	0.5008	61.3	4.1158	4.3	1.4586	17100	9.7468	1050	6.9565	1560	7.3524
4.3	1.4586	10	2.3026	0.09	-2.4079	16300	9.6989	1040	6.9470	91.8	4.5196
1.55	0.4383	12.1	2.4932	0.1	-2.3026	17000	9.7410	766	6.6412	102	4.6250
3.9	1.3610	12.4	2.5177	0.68	-0.3857	15100	9.6225	1150	7.0475	131	4.8752
4.7	1.5476	12.6	2.5337	1.5	0.4055	15800	9.6678	1000	6.9078	192	5.2575
0.95	-0.0513	11.5	2.4423	1.7	0.5306	18200	9.8092	644	6.4677	212	5.3566
0.8	-0.2231	19	2.9444	1.5	0.4055	20600	9.9330	1230	7.1148	371	5.9162
1.15	0.1398	3.2	1.1632	0.04	-3.2189	20400	9.9233	1230	7.1148	44.6	3.7977
0.95	-0.0513	15.5	2.7408	0.13	-2.0402	18500	9.8255	1150	7.0475	116	4.7536
1.15	0.1398	16.5	2.8034	1.7	0.5306	20300	9.9184	1140	7.0388	237	5.4681
1.4	0.3365	15.2	2.7213	2.1	0.7419	17900	9.7926	1060	6.9660	281	5.6384
1.15	0.1398	11.3	2.4248	1.9	0.6419	18300	9.8147	993	6.9007	345	5.8435
0.9	-0.1054	13.7	2.6174	0.18	-1.7148	19000	9.8522	1040	6.9470	134	4.8978
7.4	2.0015	14.4	2.6672	0.73	-0.3147	21900	9.9942	1230	7.1148	166	5.1120
7.1	1.9601	14.5	2.6741	1.2	0.1823	18600	9.8309	1000	6.9078	187	5.2311
		12.8	2.5494	1.2	0.1823	19500	9.8782	1090	6.9939	232	5.4467
		14.2	2.6532	0.31	-1.1712	18900	9.8469	1150	7.0475	119	4.7791
		14.4	2.6672	0.69	-0.3711	16500	9.7111	910	6.8134	139	4.9345
		15.8	2.7600	1.1	0.0953	17300	9.7585	819	6.7081	169	5.1299
		12.9	2.5572	1.4	0.3365	13650	9.5215	661	6.4938	203	5.3132
		17.3	2.8507	10.7	2.3702	19520	9.8792	776	6.6542	1400	7.2442
		18.8	2.9339	16.3	2.7912					2020	7.6109
		19.5	2.9704	15.4	2.7344					1830	7.5121
		16.5	2.8034	7.9	2.0669					1040	6.9470
		12	2.4849	4.9	1.5892					814	6.7020
		11	2.3979	3.9	1.3610					707	6.5610
		5.8	1.7579	0.1	-2.3026					177	5.1761
		10.1	2.3125	1.2	0.1823					284	5.6490
		11.8	2.4681	4.5	1.5041					726	6.5876
		11.2	2.4159	0.88	-0.1278					537	6.2860
		15.4	2.7344	7	1.9459					1080	6.9847
		13.6	2.6101	6.2	1.8245					939	6.8448
		12.8	2.5494	4.8	1.5686					793	6.6758
		15.3	2.7279	7.1	1.9601					970	6.8773
		14.8	2.6946	6.3	1.8405					839	6.7322
		10.7	2.3702	3.2	1.1632					485	6.1841
		12.8	2.5494	3.2	1.1632					515	6.2442
		15.4	2.7344	3.6	1.2809					677	6.5177
		12.4	2.5177	3.7	1.3083					498	6.2106
		13.5	2.6027	5.4	1.6864					873	6.7719
		14.6	2.6810	4.9	1.5892					819	6.7081
		11.9	2.4765	5	1.6094					1090	6.9939
		14.1	2.6462	4.2	1.4351					649	6.4754
		11.2	2.4159	3.4	1.2238					464	6.1399
		11.8	2.4681	4.1	1.4110					364	5.8972
Antimony		**Arsenic**		**Cadmium**		**Iron**		**Manganese**		**Zinc**	
Average	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**	**Average**
8.94	1.13	21.24	2.66	6.06	1.22	21336.52	9.88	1092.21	6.83	1000.06	6.53
Std Dev	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**	**Std Dev**
24.04	1.26	54.85	0.69	6.95	1.28	10498.23	0.42	697.63	0.61	929.01	0.90
Count	**Std Dev^2**	**Count**	**Std Dev^2**	**Count**	**Std Dev^2**	**Count**	**Std Dev^2**	**Count**	**Std Dev^2**	**Count**	**Std Dev^2**
647	1.60	677	0.48	677	1.63	653	0.18	653	0.37	677	0.81
SQRT	**(Std Dev^2)/2**	**SQRT**	**(Std Dev^2)/2**	**SQRT**	**(Std Dev^2)/2**	**SQRT**	**(Std Dev^2)/2**	**SQRT**	**(Std Dev^2)/2**	**SQRT**	**(Std Dev^2)/2**
25.3968502	0.79959844	25.9807621	0.23953067	25.9807621	0.8149894	25.5147016	0.08939246	25.5147016	0.18663857	25.9807621	0.40270831
UCL_{95}normal	1.93	**UCL_{95}**normal	2.90	**UCL_{95}**normal	2.03	**UCL_{95}**normal	9.97	**UCL_{95}**normal	7.02	**UCL_{95}**normal	6.94
10.5011607	**$Mean_{lognormal}$**	24.7139873	**$Mean_{lognormal}$**	6.49594209	**$Mean_{lognormal}$**	22013.3722	**$Mean_{lognormal}$**	1137.18385	**$Mean_{lognormal}$**	1058.88363	**$Mean_{lognormal}$**
	6.8894		18.1617		7.6441		21307.72		1114.52		1030.30

Sb	As	Cd	Fe	Mn	Zn
1	9.1	2	17900	379	473
2.5	10	2.2	18800	788	524
2	10.7	3.3	18600	732	618
1.7	9	1.9	19000	734	356
0.67	9.8	1.3	18300	702	242
1.6	11.6	2.5	18400	750	562
1.7	14.9	3.4	21400	833	720
0.67	11.6	1.5	20700	863	238
0.335	9.4	1.6	19400	865	350
15.8	49.7	12.8	31600	1770	2150
3.4	14.2	7.9	19300	1230	1090
3.8	15.3	8.1	20200	1370	1130
3.9	15.5	9.4	20600	1490	1340
5.8	15.6	5.8	17500	1070	875
76.8	61.3	4.3	26200	1500	1560
0.95	10	0.09	11100	468	91.8
0.95	12.1	0.1	12200	613	102
1.3	12.4	0.68	12200	813	131
1.8	12.6	1.5	12300	846	192
1.8	11.5	1.7	12100	789	212
7.2	19	1.5	17500	747	371
0.405	3.2	0.04	3190	212	44.6
1.3	15.5	0.13	13500	749	116
2.3	16.5	1.7	13600	937	237
2.3	15.2	2.1	13300	937	281
2.6	11.3	1.9	12800	705	345
1.3	13.7	0.18	13500	842	134
1.6	14.4	0.73	14300	907	166
1.8	14.5	1.2	14300	942	187
1.8	12.8	1.2	12800	771	232
1.3	14.2	0.31	13000	780	119
1.6	14.4	0.69	13400	780	139
1.8	15.8	1.1	13600	799	169
1.9	12.9	1.4	12600	749	203
4.5	17.3	10.7	20800	1300	1400
8.1	18.8	16.3	24200	1430	2020
6.2	19.5	15.4	24400	1410	1830
5.5	16.5	7.9	18700	1140	1040
1	12	4.9	17100	1050	814
1.05	11	3.9	16300	1040	707
0.255	5.8	0.1	17000	766	177
0.475	10.1	1.2	15100	1150	284
1.45	11.8	4.5	15800	1000	726
0.55	11.2	0.88	18200	644	537
1.65	15.4	7	20600	1230	1080
4.3	13.6	6.2	20400	1230	939
1.55	12.8	4.8	18500	1150	793
3.9	15.3	7.1	20300	1140	970
4.7	14.8	6.3	17900	1060	839
0.95	10.7	3.2	18300	993	485

0.8	12.8	3.2	19000	1040	515
1.15	15.4	3.6	21900	1230	677
0.95	12.4	3.7	18600	1000	498
1.15	13.5	5.4	19500	1090	873
1.4	14.6	4.9	18900	1150	819
1.15	11.9	5	16500	910	1090
0.9	14.1	4.2	17300	819	649

Mullan Residential Soil

Antimony

Analytical Results
(mg/kg)

2.6	2.3	4.4
2.5	1	0.6
2.3	3.4	1.15
133	1.9	1.2
5	9.1	1.4
11.7	28.4	4.5
7	5.9	1.4
1.5	13.4	4
7.1	2.6	3.7
12.9	23.5	6.2
2.5	79.3	5.7
28.8	6.2	9
3.4	19	1.3
2.1	57.3	10.4
2.5	68.4	1.5
2.3	18.1	12.4
2.3	2.1	4
2.2	31	14.5
3.4	3.9	2.1
63.3	201	2
3.2	1.25	16.4
4.2	3	
12	1.55	
6.3	15	
12.6	4.9	
1.5	2.05	
7.8	0.5	
2.1	0.55	
5.7	0.39	
1	0.9	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	81	Mean	13.22
Censored		Lognormal mean	11.04
Detection limit or PQL		Std. devn.	29.29594
Method detection limit		Median	3.9
TOTAL	81	Min.	0.39
		Max.	201

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -1.4982. This lies within the tabled values of 1.2302 and -2.6097

UCL (based on t-statistic) is 18.64

Mullan Residential Soil

Arsenic

Analytical Results
(mg/kg)

21.4	9.5	14.2
45.8	10.8	7.9
15.8	13.7	7.8
92.1	12.5	8.5
10.9	18	7.3
30.6	87.2	15.2
12.3	14.2	8.1
10.4	37.7	19.7
11.3	11.7	8.4
37.9	56.8	24.2
9.9	138	6.2
24.4	30.6	32.5
9.4	41.2	6.4
10.3	433	18.8
10.7	55.1	8
11.1	26.9	24
8.2	26.8	9
10.8	37.5	13.6
5.1	12	6.6
50.9	165	19.3
7.7	11	17.2
12.3	11.3	
17.5	11.6	
22.1	23.2	
20.4	16.5	
3.4	13.9	
16.5	6.8	
14.3	6.3	
9.8	6.9	
12.9	6.5	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	81	Mean	27.30
Censored		Lognormal mean	23.05
Detection limit or PQL		Std. devn.	52.70697
Method detection limit		Median	13.6
TOTAL	81	Min.	3.4
		Max.	433

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -5.6296. This lies outside the tabled values of 1.2302 and -2.6097
Reject normal distribution.
Y value is -41.4803. This lies outside the tabled values of 1.2302 and -2.6097

UCL (based on t-statistic) is 37.06

Mullan Residential Soil

Cadmium

Analytical Results
(mg/kg)

2.7	1.9	3.7
2.7	4.8	9
1.6	8.1	2
6.6	6.1	12.2
2.7	9.1	0.5
1.8	1.8	2.7
2.1	4.9	1.7
1.8	4.6	13.4
1.8	0.21	5.2
3	3.1	6.1
0.61	4.3	1.5
7.2	42.5	3.1
1.9	1.5	10.5
0.58	1.6	5.2
2.2	3.4	39.2
1.1	4.1	7.9
4.2	2.8	29.3
1.7	2.2	6.2
8	1.2	0.97
3.1	1.2	7.1
5	0.86	16.2
2.9	1.7	
3	1.4	
2.9	1.1	
4.5	2.1	
2.1	2.2	
5.6	3.5	
5.9	2.8	
3.1	2.7	
2.9	3.9	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	81	Mean	5.07
Censored		Lognormal mean	4.82
Detection limit or PQL		Std. devn.	7.060986
Method detection limit		Median	2.9
TOTAL	81	Min.	0.21
		Max.	42.5

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -2.9171. This lies outside the tabled values of 1.2302 and -2.6097
Reject normal distribution.
Y value is -28.9652. This lies outside the tabled values of 1.2302 and -2.6097

UCL (based on t-statistic) is 6.38

Mullan Residential Soil

Iron

Analytical Results
(mg/kg)

26500	23400	15700
32300	19600	16200
15000	21000	14600
37600	41900	22300
19200	19200	11100
17800	25900	22100
18600	15400	12500
35900	31000	25400
22500	53600	8730
24300	18700	23220
17900	36100	13390
18600	70400	19790
18100	60300	13410
20700	31300	22000
13500	25100	17320
16500	51100	18870
15800	16700	14660
21600	48400	14940
17100	18400	21770
13700	17900	
14300	17600	
15300	16800	
17100	18100	
17500	17900	
43300	16800	
35700	15800	
27400	16700	
17800	17100	
22700	15500	
18300	15700	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	79	Mean	22708.86
Censored		Lognormal mean	22473.77
Detection limit or PQL		Std. devn.	11382.2
Method detection limit		Median	18400
TOTAL	79	Min.	8730
		Max.	70400

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -5.176. This lies outside the tabled values of 1.221 and -2.6169
Reject normal distribution.
Y value is -14.2084. This lies outside the tabled values of 1.221 and -2.6169

UCL (based on t-statistic) is 24843.75

Mullan Residential Soil

Manganese

Analytical Results
(mg/kg)

1440	1400	799
2570	773	879
934	1180	1460
3530	3210	1330
1330	1380	914
454	1680	1200
1280	984	1310
3310	1980	2430
1040	3640	505
882	1550	1920
1400	2490	414
1120	3900	1300
986	4830	535
877	2290	1770
718	912	1930
1070	4010	1360
594	787	657
1270	4170	796
818	778	2400
622	679	
834	781	
749	913	
1300	735	
791	679	
2470	589	
2160	658	
1560	754	
806	552	
1540	650	
1090	590	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	79	Mean	1430.10
Censored		Lognormal mean	1412.30
Detection limit or PQL		Std. devn.	978.4843
Method detection limit		Median	1090
TOTAL	79	Min.	414
		Max.	4830

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -0.3121. This lies within the tabled values of 1.221 and -2.6169

UCL (based on t-statistic) is 1613.63

Mullan Residential Soil

Zinc

Analytical Results
(mg/kg)

580	611	369
620	4450	301
944	1130	510
6280	446	511
987	1200	486
2530	2680	787
932	665	481
265	1060	1220
953	439	559
2610	1170	1390
497	1040	302
845	1150	1890
600	1650	88.5
708	266	488
407	762	167
379	706	1650
467	127	619
621	697	874
236	524	171
1870	6450	449
371	289	1510
1290	277	
444	557	
2840	815	
922	586	
426	379	
3780	258	
2840	384	
1470	299	
3690	344	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	81	Mean	1081.94
Censored		Lognormal mean	1048.74
Detection limit or PQL		Std. devn.	1213.294
Method detection limit		Median	620
TOTAL	81	Min.	88.5
		Max.	6450

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -1.1209. This lies within the tabled values of 1.2302 and -2.6097

UCL (based on t-statistic) is 1306.63

Mullan Residential Tap Water

Arsenic

Site ID	Static Result (ug/L)	Purged Result (ug/L)	Average Result (ug/L)
R009	0.1 U	0.1 U	0.1
R023	0.1 U	0.1 U	0.1
R038	0.1 U	0.1 U	0.1
R041	0.1 U	0.1 U	0.1
R046	0.1 U	0.1 U	0.1
R074	0.1 U	0.1 U	0.1
R086	0.1 U	0.35	0.225
R105	0.25 U	0.25 U	0.25

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	8	Mean	0.13
Censored		Lognormal mean	0.13
Detection limit or PQL		Std. devn.	0.064
Method detection limit		Median	0.1
TOTAL	8	Min.	0.1
		Max.	0.25

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.5852. This is less than the tabled value of 0.818
Reject normal distribution.
W value is 0.5929. This is less than the tabled value of 0.818

UCL (based on t-statistic) is 0.18

Mullan Neighborhood Exposure to Waste Piles

Antimony

Analytical Results (mg/kg)
19.7
20.3
16.5
1.5
1.2

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	11.84
Censored		Lognormal mean	18.66
Detection limit or PQL		Std. devn.	9.684937
Method detection limit		Median	16.5
TOTAL	5	Min.	1.2
		Max.	20.3

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.7448. This is less than the tabled value of 0.762
Assume normal distribution.
W value is 0.778. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 21.07

Mullan Neighborhood Exposure to Waste Piles

Arsenic

Analytical Results
(mg/kg)

42.1
35.4
28.2
5.6
6.7

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	23.60
Censored		Lognormal mean	27.62
Detection limit or PQL		Std. devn.	16.67528
Method detection limit		Median	28.2
TOTAL	5	Min.	5.6
		Max.	42.1

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8173. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 39.5

Mullan Neighborhood Exposure to Waste Piles

Cadmium

Analytical Results
(mg/kg)

11.9
13.8
1.1
0.55
0.51

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	5.57
Censored		Lognormal mean	8.42
Detection limit or PQL		Std. devn.	6.681816
Method detection limit		Median	1.1
TOTAL	5	Min.	0.51
		Max.	13.8

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.7998. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 11.94

Mullan Neighborhood Exposure to Waste Piles

Iron

Analytical Results
(mg/kg)

29700
33200
22600
12900
12500

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	22180.00
Censored		Lognormal mean	22731.52
Detection limit or PQL		Std. devn.	9460.285
Method detection limit		Median	22600
TOTAL	5	Min.	12500
		Max.	33200

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8623. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 31200

Mullan Neighborhood Exposure to Waste Piles

Manganese

Analytical Results
(mg/kg)

2130
2100
864
2750
2670

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	2102.80
Censored		Lognormal mean	2183.03
Detection limit or PQL		Std. devn.	754.3137
Method detection limit		Median	2130
TOTAL	5	Min.	864
		Max.	2750

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.7668. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 2822.01

Mullan Neighborhood Exposure to Waste Piles

Zinc

Analytical Results
(mg/kg)

1820
2880
324
97.2
89.7

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	5	Mean	1042.18
Censored		Lognormal mean	1589.22
Detection limit or PQL		Std. devn.	1254.848
Method detection limit		Median	324
TOTAL	5	Min.	89.7
		Max.	2880

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8688. This exceeds the tabled value of 0.762

UCL (based on t-statistic) is 2238.63

Mullan Neighborhood Exposure to Sediments

Antimony

Analytical Results
(mg/kg)

4.54
2.035
1.555
1.165
2.62
0.948
1.35
2.27
0.56
0.505
1.595
3.755
14.7
0.498

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	14	Mean	2.72
Censored		Lognormal mean	2.61
Detection limit or PQL		Std. devn.	3.64958
Method detection limit		Median	1.575
TOTAL	14	Min.	0.498
		Max.	14.7

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9456. This exceeds the tabled value of 0.874

UCL (based on t-statistic) is 4.45

Mullan Neighborhood Exposure to Sediments

Arsenic

Analytical Results
(mg/kg)

15.1
9.62
11
10.1
11
9.93
8.95
18
3.19
15.7
24.4
10.3
28.4
4.43

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	14	Mean	12.87
Censored		Lognormal mean	13.21
Detection limit or PQL		Std. devn.	6.9846
Method detection limit		Median	10.65
TOTAL	14	Min.	3.19
		Max.	28.4

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9311. This exceeds the tabled value of 0.874

UCL (based on t-statistic) is 16.17

Mullan Neighborhood Exposure to Sediments

Cadmium

Analytical Results

(mg/kg)

21.8
7.1
2.58
3.83
2.49
4.56
2.89
5.92
1.61
0.873
1.17
2.66
81
0.143

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	14	Mean	9.90
Censored		Lognormal mean	9.66
Detection limit or PQL		Std. devn.	21.15425
Method detection limit		Median	2.775
TOTAL	14	Min.	0.143
		Max.	81

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9491. This exceeds the tabled value of 0.874

UCL (based on t-statistic) is 19.91

Mullan Neighborhood Exposure to Sediments

Iron

Analytical Results

(mg/kg)

53100
55300
18200
21000
42500
33700
33400
28300
6160
18800
12800
15000
121000
10700

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	14	Mean	33568.57
Censored		Lognormal mean	34078.54
Detection limit or PQL		Std. devn.	29441.15
Method detection limit		Median	24650
TOTAL	14	Min.	6160
		Max.	121000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9904. This exceeds the tabled value of 0.874

UCL (based on t-statistic) is 47503.65

Mullan Neighborhood Exposure to Sediments

Manganese

Analytical Results
(mg/kg)

4900
6030
1310
1600
3880
3070
2700
1430
391
1420
1960
3060
15700
628

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	14	Mean	3434.21
Censored		Lognormal mean	3512.59
Detection limit or PQL		Std. devn.	3879.001
Method detection limit		Median	2330
TOTAL	14	Min.	391
		Max.	15700

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9806. This exceeds the tabled value of 0.874

UCL (based on t-statistic) is 5270.22

Mullan Neighborhood Exposure to Sediments

Zinc

Analytical Results

(mg/kg)

5570
2530
439
1180
1890
1630
1510
1820
305
636
428
560
13700
471

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	14	Mean	2333.50
Censored		Lognormal mean	2207.74
Detection limit or PQL		Std. devn.	3546.455
Method detection limit		Median	1345
TOTAL	14	Min.	305
		Max.	13700

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9276. This exceeds the tabled value of 0.874

UCL (based on t-statistic) is 4012.11

Mullan Neighborhood Exposure to Surface Water

Arsenic

Analytical Results

(ug/L)

0.45	1
1	0.08
0.22	1
1	0.1
0.16	1.8
0.31	1
0.5	0.34
0.41	0.5
0.28	0.2
0.57	0.5
0.37	0.08
0.34	0.5
1.4	0.41
0.5	0.5
0.135	0.32
0.5	0.5
0.08	0.08
0.5	1
0.6	0.08
1	1
0.31	0.08
1	1
0.47	0.23
0.5	1
0.91	0.33
1	1
0.27	0.115
0.22	0.115
1	0.115
0.315	0.115
1	0.115
0.215	0.23
1	0.115
0.08	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	67	Mean	0.51
Censored		Lognormal mean	0.54
Detection limit or PQL		Std. devn.	0.390026
Method detection limit		Median	0.41
TOTAL	67	Min.	0.08
		Max.	1.8

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is 0.3145. This lies within the tabled values of 1.1577 and -2.6661

UCL (based on t-statistic) is 0.59

Mullan Neighborhood Exposure to Surface Water

Cadmium

Analytical Results

(ug/L)

0.64	0.1
3.6	0.0345
0.0345	0.1
0.1	0.08
4.4	0.92
0.021	0.9
0.56	0.0345
0.17	0.05
0.79	0.0345
0.36	0.05
0.88	0.0345
0.32	0.05
0.0345	7.3
0.62	8.8
0.0345	0.0345
0.05	0.05
0.0345	0.0345
0.05	0.1
0.0345	0.0345
0.1	0.1
0.0345	0.0345
0.1	0.1
0.0345	0.0345
0.05	0.1
0.0345	0.4
0.1	0.1
0.0205	0.021
0.0345	15.2
0.1	2.5
0.145	0.021
0.1	0.021
2.9	0.0345
0.1	0.16
0.0345	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	67	Mean	0.81
Censored		Lognormal mean	0.48
Detection limit or PQL		Std. devn.	2.366885
Method detection limit		Median	0.1
TOTAL	67	Min.	0.0205
		Max.	15.2

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -6.3479. This lies outside the tabled values of 1.1577 and -2.6661
Reject normal distribution.
Y value is -37.2298. This lies outside the tabled values of 1.1577 and -2.6661

UCL (based on t-statistic) is 1.29

Mullan Neighborhood Exposure to Surface Water

Manganese

Analytical Results

(ug/L)

1.9	7
6	2.6
12.1	2.5
13	5.8
60.2	17.8
18.9	6
81.6	0.5
11.75	11.5
50.6	0.5
21.7	2.5
60.9	0.5
19.1	2.5
2	1.55
6.3	7.5
1	0.6
2.5	6
1.1	0.5
42.2	2.5
0.5	4.6
2.5	2.5
1.2	0.5
2.5	2.5
127	0.5
27.5	2.5
1.3	19
2.5	28
38.5	3.2
0.5	107
2.5	31.6
1.4	1
2.5	0.65
1.8	0.5
2.5	4
3.4	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	67	Mean	13.69
Censored		Lognormal mean	13.66
Detection limit or PQL		Std. devn.	24.7081
Method detection limit		Median	2.5
TOTAL	67	Min.	0.5
		Max.	127

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -0.0227. This lies within the tabled values of 1.1577 and -2.6661

UCL (based on t-statistic) is 18.73

Mullan Neighborhood Exposure to Surface Water

Mercury

Analytical Results

(ug/L)

0.05	0.1
0.1	0.05
0.05	0.1
0.1	0.05
0.05	0.05
0.08	0.1
0.05	0.05
0.08	**0.32**
0.05	0.05
0.08	0.1
0.05	0.05
0.08	0.1
0.05	0.05
0.1	0.1
0.05	0.05
0.1	0.1
0.05	0.05
0.1	0.1
0.145	0.05
0.1	0.1
0.05	0.05
0.1	0.1
0.05	0.05
0.1	0.1
0.05	0.08
0.1	0.1
0.08	0.08
0.05	0.08
0.1	0.08
0.05	0.08
0.1	0.08
0.05	0.05
0.1	0.08
0.05	all nondetects except for one (detect is bolded)

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	67	Mean	0.08
Censored		Lognormal mean	0.08
Detection limit or PQL		Std. devn.	0.038418975
Method detection limit		Median	0.08
TOTAL	67	Min.	0.05
		Max.	0.32

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -7.0632. This lies outside the tabled values of 1.1577 and -2.6661
Reject normal distribution.
Y value is -19.769. This lies outside the tabled values of 1.1577 and -2.6661

UCL (based on t-statistic) is 0.09

Mullan Occupational Exposure to Soil

Antimony

Analytical Results
(mg/kg)

0.28	3.1	4.8	174	2.05	6.2
0.72	3.6	5.7	19	0.42	5.7
9.3	4.5	6.5	57.3	0.27	9
17.4	2.5	5.7	35	0.5	1.3
2.6	2.3	1	88.5	0.365	10.4
2.9	4.2	2.4	99.4	0.435	1.5
7.3	6.2	3.1	167	0.35	12.4
2.5	8.5	3.4	68.4	0.55	4
2.25	2.3	2.3	16.9	0.39	14.5
10.2	0.49	1	20.7	0.75	2
10.7	0.5	1.8	18.1	0.395	16.4
2.3	1.5	2.2	2.1	0.48	
133	2.2	3.4	2.7	0.9	
4.4	1.1	3.4	23.8	4.4	
4.5	1.4	1.1	24.4	0.95	
5.2	2.4	1.4	2.1	7.1	
5	3.4	1.9	26.7	1.55	
1.8	63.3	3.3	71.8	0.6	
1.5	0.33	4.2	76.8	1.1	
1.6	0.335	5.4	31	0.27	
1.75	0.335	9.1	3	0.75	
11.7	1.3	28.4	1.15	1.15	
7	3.2	2.5	1.15	0.9	
6.3	4.2	3.9	3.9	0.65	
12	0.34	4.2	201	0.9	
14.1	0.345	4.7	1.35	1.2	
1.5	2.1	5.9	2.5	0.75	
7.1	12	13.4	0.8	0.385	
1.3	6.3	0.5	1.25	0.7	
5.2	1.3	1.5	1.25	1.4	
15	1.4	4.9	1.5	4.5	
12.9	7.7	2.6	1.15	1.3	
0.5	12.6	23.5	0.75	0.95	
0.5	0.49	121	3	1	
3	1.3	278	1.55	1.4	
2.5	1.6	295	1.95	4	
28.8	1.5	79.3	1.65	4.2	
0.5	3.4	20.7	1.8	3.9	
0.5	3.7	25.2	15	1.4	
0.55	4.7	35.4	4.9	3.7	
2	7.8	6.2	1.55	3.2	
3.4	2.1	23.2	0.9	7.9	
2.1	4.3	131	0.7	3.9	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	226	Mean	14.09
Censored		Lognormal mean	10.49
Detection limit or PQL		Std. devn.	38.29095
Method detection limit		Median	2.8
TOTAL	226	Min.	0.27
		Max.	295

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -2.734. This lies outside the tabled values of 1.5215 and -2.3686
Reject normal distribution.
Y value is -68.3815. This lies outside the tabled values of 1.5215 and -2.3686

UCL (based on t-statistic) is 18.31

Mullan Occupational Exposure to Soil

Arsenic

Analytical Results

(mg/kg)

2.5	11.7	12.7	104	11.5	8.4
10.1	11.9	14	125	13.9	57.1
40.1	13.2	14.2	41.2	6.7	19.3
62.6	10.7	9.8	433	4.2	9.4
21.4	11.1	12.9	36.9	5	24.2
75.7	12.9	10.4	85.9	6.8	6.2
76	17.5	11.5	88.3	6	32.5
45.8	25.7	11.8	101	4.3	6.4
18.7	8.2	9.5	55.1	3.9	18.8
40.3	8.1	10.8	18.7	3.8	8
92.9	8.5	10	25.7	6.3	24
15.8	9	13.2	26.9	6.9	9
92.1	10.8	16.2	30.1	7.9	13.6
9.7	7.6	13.7	38.7	5.8	6.6
10.8	4.25	9.8	38.9	4.9	19.3
14.3	5.4	12.6	26.8	6.5	17.2
10.9	5.1	13	55	14.2	
10.5	50.9	12.5	123	8	
11.1	4.8	9	193	11.3	
11.3	4.9	10.7	37.5	8.9	
12.7	5.3	11.4	10.7	7.9	
30.6	6.6	18	9.3	7.5	
12.3	7.7	87.2	10.3	6.8	
23.4	12.3	10.4	12	10.1	
33.8	4.6	12.2	165	7.8	
115	5.3	13.7	11.1	8.1	
10.4	15.7	13.9	10.1	8	
11.3	17.5	14.2	9.8	6.6	
8.1	22.1	37.7	11	8.5	
15.7	10.1	11.4	11.6	5.1	
31.4	10.5	13.4	11	7.4	
37.9	18.7	18.5	10.7	8	
5.4	20.4	11.7	12.8	6.8	
6.2	6	56.8	11.3	7.3	
9.3	6.9	163	11.6	15.2	
9.9	8.8	260	11.2	9.1	
24.4	3.4	379	9.7	6.7	
5.1	11.1	138	10.5	5	
5.4	11.4	57.2	23.2	8.1	
6	11.8	64.8	16.5	19.7	
6.8	16.5	71.9	11.6	9.2	
9.4	14.3	30.6	9.7	8.8	
10.3	10.9	27		5.9	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	230	Mean	26.48
Censored		Lognormal mean	22.17
Detection limit or PQL		Std. devn.	48.80138913
Method detection limit		Median	11.35
TOTAL	230	Min.	2.5
		Max.	433

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -9.9427. This lies outside the tabled values of 1.5254 and -2.3652
Reject normal distribution.
Y value is -61.9861. This lies outside the tabled values of 1.5254 and -2.3652

UCL (based on t-statistic) is 31.81

Mullan Occupational Exposure to Soil

Cadmium

Analytical Results

(mg/kg)

5.4	0.03	3.3	4.3	2.1	2.3
7.1	0.35	3.4	42.5	1.4	8.6
7.1	4.5	4.2	1.6	0.91	14.9
8	2.2	3.1	1	1.1	4.7
2.7	1.1	2.9	0.55	2.2	8.2
4.5	3.1	0.105	1.5	3.1	8.9
8.7	3.2	0.73	1.5	0.44	2.9
2.7	5.2	2.2	1.4	1.1	3
0.66	4.2	1.9	0.78	1.4	3.3
1.9	0.76	4.8	0.57	3.5	3.4
2	1.2	6.5	1.6	2.8	2.9
1.6	1.3	9.7	3.4	2.8	4.1
6.6	1.7	10.8	1.2	1.9	4.6
0.48	3.3	8.1	1.1	1.1	0.52
0.58	3.3	2.6	1.4	2.7	2.6
0.82	5.8	6.3	4.1	3.9	10.6
1.6	8	8.1	2.8	3.1	
2.7	3.1	6.1	1.6	2.3	
1.8	4	9	0.6	2	
3.3	4.1	34.3	0.53	3.7	
3.7	4.6	73.4	2.2	9.7	
3.9	4.9	9.1	0.74	4.5	
2.1	5	1.8	0.04	2.9	
1.8	2.9	0.67	1.2	9	
2.8	2.5	4.1	0.96	2	
5.2	4	4.4	0.22	12.2	
6.7	4	12.2	0.04	0.5	
1.8	3	4.9	0.04	2.7	
1.3	2.9	3.9	1.2	1.7	
1.8	1.1	7.5	0.86	13.4	
1.9	2.8	4.6	1.7	5.2	
3	3.4	0.1	0.64	6.1	
0.13	4.5	0.1	0.46	1.5	
0.15	0.39	2.7	1.7	3.1	
1.5	1.1	3	1.4	10.5	
0.61	1.4	0.21	2.3	5.2	
7.2	2.1	4.4	4.2	39.2	
0.17	1.5	5.8	6.7	7.9	
0.47	2.3	7.4	1.1	29.3	
0.5	3.1	3.1	1.4	6.2	
1.8	5.6	3	0.045	0.97	
1.9	5.9	1.4	2.2	7.1	
0.58	2.1	1.5		16.2	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	230	Mean	4.22
Censored		Lognormal mean	4.73
Detection limit or PQL		Std. devn.	6.95649
Method detection limit		Median	2.75
TOTAL	230	Min.	0.03
		Max.	73.4

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -8.5282. This lies outside the tabled values of 1.5254 and -2.3652
Reject normal distribution.
Y value is -58.8842. This lies outside the tabled values of 1.5254 and -2.3652

UCL (based on t-statistic) is 4.99

Mullan Occupational Exposure to Soil

Iron

Analytical Results
(mg/kg)

30200	13500	22700	60300	13300	19790
38500	12900	18300	22900	13200	13410
49300	15200	21200	30200	15800	22000
26500	15500	21400	31300	16700	17320
32300	16500	23500	26100	17900	18870
13600	14800	23400	32400	17200	14660
15400	15900	19200	39200	17700	14940
16500	18700	21000	25100	17100	21770
15000	15800	23600	56500	15500	
17500	21600	19600	113000	17100	
18400	17900	17900	124000	21800	
19600	18000	18400	51100	20000	
21500	18800	18800	17500	15700	
37600	19400	21000	17100	15100	
19200	17100	41900	16400	18100	
19900	13700	14800	16700	15900	
24000	16500	16800	48400	15700	
42100	18700	17300	17000	18200	
17800	21100	19200	18000	18200	
18600	14300	19200	18700	14600	
16300	15300	25900	18400	16200	
16500	18600	11800	17900	16000	
26800	19300	14200	18600	20100	
35900	19900	18500	18700	19200	
11800	17100	15400	21100	16800	
13100	16300	31000	17900	14600	
14600	17600	62000	17600	22300	
22500	18100	113000	13000	12300	
24300	17500	147000	12000	11000	
11200	25800	53600	15700	8080	
11900	26500	36400	16800	11100	
12400	35700	50400	18100	22100	
14300	43300	60300	17500	13700	
17900	35700	18700	18500	13400	
18600	26700	31800	20100	10900	
16600	28900	88400	17900	12500	
18000	28900	117000	20400	20300	
18600	39600	36100	14000	17700	
18100	27400	70400	14900	14000	
20700	17800	35100	16800	25400	
16900	22200	73700	16800	8730	
19700	22800	77200	14900	23220	
22800	22900	99700		13390	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	222	Mean	24863.42
Censored		Lognormal mean	23899.14
Detection limit or PQL		Std. devn.	19945.56533
Method detection limit		Median	18450
TOTAL	222	Min.	8080
		Max.	147000

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -16.4666. This lies outside the tabled values of 1.5176 and -2.3721
Reject normal distribution.
Y value is -45.2725. This lies outside the tabled values of 1.5176 and -2.3721

UCL (based on t-statistic) is 27082.92

Mullan Occupational Exposure to Soil

Manganese

Analytical Results

(mg/kg)

1920	718	1540	4830	209	1300
2010	429	1090	1730	137	535
4720	673	1510	2330	658	1770
1440	890	1650	2290	754	1930
2570	1070	1660	867	597	1360
888	804	1400	2300	502	657
1110	892	678	2810	618	796
1150	1020	722	912	552	2400
934	594	742	5090	650	
903	1270	773	9270	680	
1100	367	859	9960	1150	
1140	503	938	4010	941	
1300	657	1020	696	590	
3530	1020	1180	619	735	
1330	818	3210	655	769	
1240	622	419	787	993	
1680	305	790	4170	799	
3680	657	872	780	775	
454	1580	1370	700	879	
1280	834	1380	618	613	
602	749	1680	778	879	
914	1010	188	681	1470	
2290	1220	339	639	430	
3310	1640	917	572	509	
599	1300	984	597	730	
691	518	1980	679	1460	
982	732	4770	781	1330	
1040	735	8410	468	817	
882	791	17300	445	474	
525	1400	3640	626	319	
549	1520	3560	913	914	
559	1900	6500	735	1200	
909	2470	7360	641	1170	
1400	2160	1550	569	810	
1120	1350	2450	685	666	
891	1590	15600	679	1310	
896	2560	20200	585	2400	
1170	2620	2490	243	1240	
986	1560	3900	243	1120	
877	806	3680	589	2430	
818	1550	5080	702	505	
972	1670	6000	427	1920	
1270	1970	6720		414	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	222	Mean	1688.48
Censored		Lognormal mean	1536.48
Detection limit or PQL		Std. devn.	2411.869
Method detection limit		Median	925.5
TOTAL	222	Min.	137
		Max.	20200

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -7.2461. This lies outside the tabled values of 1.5176 and -2.3721
Reject normal distribution.
Y value is -54.0855. This lies outside the tabled values of 1.5176 and -2.3721

UCL (based on t-statistic) is 1956.87

Mullan Occupational Exposure to Soil

Zinc

Analytical Results

(mg/kg)

1130	703	1090	4290	127	559
1250	721	1190	9250	379	1110
1280	799	1640	1650	187	681
1550	407	1470	266	54.7	484
580	379	3690	501	51.5	1390
820	644	706	1010	258	302
1620	1110	793	1060	265	1890
620	1310	1040	1130	92.8	88.5
555	467	611	762	49.5	488
1560	375	4450	525	32.6	167
2430	423	305	836	384	1650
944	482	623	706	299	619
6280	621	836	142	361	874
701	127	1130	498	257	171
1070	136	198	545	231	449
1140	353	249	127	344	1510
987	236	282	881	369	
474	1870	446	965	672	
480	106	531	1150	1010	
603	145	604	697	995	
643	145	769	375	301	
2530	346	1200	213	318	
932	371	2680	256	180	
502	1290	515	524	447	
624	106	627	6450	510	
770	203	648	277	378	
265	1080	785	189	326	
953	444	665	144	232	
174	2840	1060	289	511	
512	743	88.7	261	459	
1750	848	189	244	164	
2610	1090	419	149	180	
237	922	439	147	252	
462	185	1170	277	486	
573	255	1050	557	787	
497	290	1850	256	484	
845	426	2340	190	337	
127	931	1040	237	236	
188	1090	805	815	481	
235	2620	944	586	1220	
351	3780	1490	266	501	
600	2840	1150	139	387	
708	895	1300		318	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	230	Mean	833.05
Censored		Lognormal mean	823.04
Detection limit or PQL		Std. devn.	1040.644
Method detection limit		Median	538
TOTAL	230	Min.	32.6
		Max.	9250

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -1.5253. This lies within the tabled values of 1.5254 and -2.3652

UCL (based on t-statistic) is 946.82

Blackwell Island Public Recreational Exposure to Soil/Sediments

Antimony

Analytical Results (mg/kg)
5.5
5
3.4
1.4
3.1
2.3
4.2
1.2
1.8
1.5
1.2
3.1
1.1
1.9
4.1
2.3
3.1
3.5
2.4
3.3
2.4

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	21	Mean	2.75
Censored		Lognormal mean	2.78
Detection limit or PQL		Std. devn.	1.252844
Method detection limit		Median	2.4
TOTAL	21	Min.	1.1
		Max.	5.5

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9571. This exceeds the tabled value of 0.908

UCL (based on t-statistic) is 3.22

Blackwell Island Public Recreational Exposure to Soil/Sediments

Arsenic

Analytical Results (mg/kg)
59.3
74
77
53.4
59.2
37.2
56.7
9.7
12.4
16.7
12.5
20.3
15.7
14.2
39.8
63.5
83.4
52.2
19.8
45.8
38.8

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	21	Mean	41.03
Censored		Lognormal mean	42.75
Detection limit or PQL		Std. devn.	23.83403
Method detection limit		Median	39.8
TOTAL	21	Min.	9.7
		Max.	83.4

Lognormal distribution?	Normal distribution?
r-squared is:	r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8981. This is less than the tabled value of 0.908
Assume normal distribution.
W value is 0.9208. This exceeds the tabled value of 0.908

UCL (based on t-statistic) is 50

Blackwell Island Public Recreational Exposure to Soil/Sediments

Cadmium

Analytical Results (mg/kg)
17
7.7
11.1
2.1
12.6
9.6
8.7
2.2
9.5
3.2
6.1
13.7
2.8
12.5
21.6
17.6
17.2
11.3
16.6
17.3
15.5

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	21	Mean	11.23
Censored		Lognormal mean	12.01
Detection limit or PQL		Std. devn.	5.752594
Method detection limit		Median	11.3
TOTAL	21	Min.	2.1
		Max.	21.6

Lognormal distribution?	Normal distribution?
r-squared is:	r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8514. This is less than the tabled value of 0.908
Assume normal distribution.
W value is 0.9501. This exceeds the tabled value of 0.908

UCL (based on t-statistic) is 13.4

Blackwell Island Public Recreational Exposure to Soil/Sediments

Iron

Analytical Results (mg/kg)
61100
66800
64100
58300
53300
41700
61300
19500
19900
29600
24800
29100
28600
26300
41000
51500
65600
48000
31600
41500
42200

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	21	Mean	43133.33
Censored		Lognormal mean	43484.59
Detection limit or PQL		Std. devn.	15869.19
Method detection limit		Median	41700
TOTAL	21	Min.	19500
		Max.	66800

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9249. This exceeds the tabled value of 0.908

UCL (based on t-statistic) is 49106.91

Blackwell Island Public Recreational Exposure to Soil/Sediments

Manganese

Analytical Results (mg/kg)
2920
3040
5470
3330
3920
2180
2320
859
1210
1300
1260
1780
1160
1380
4380
6300
7480
3720
1380
3850
2840

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	21	Mean	2956.14
Censored		Lognormal mean	2996.41
Detection limit or PQL		Std. devn.	1818.217
Method detection limit		Median	2840
TOTAL	21	Min.	859
		Max.	7480

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9582. This exceeds the tabled value of 0.908

UCL (based on t-statistic) is 3640.57

Blackwell Island Public Recreational Exposure to Soil/Sediments

Zinc

Analytical Results (mg/kg)
2460
2030
2080
441
1910
1590
1880
265
1040
577
777
1640
448
1530
2020
2020
2170
1900
2080
2050
2170

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	21	Mean	1575.14
Censored		Lognormal mean	1672.13
Detection limit or PQL		Std. devn.	683.3225
Method detection limit		Median	1900
TOTAL	21	Min.	265
		Max.	2460

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.7645. This is less than the tabled value of 0.908
Reject normal distribution.
W value is 0.8454. This is less than the tabled value of 0.908

UCL (based on t-statistic) is 1832.36

Blackwell Island Public Recreational Exposure to Surface Water

Arsenic

Analytical Results
(ug/L)

5.6
2.9
20.8
7.6
14.8
9.8
12.4

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	7	Mean	10.56
Censored		Lognormal mean	11.12
Detection limit or PQL		Std. devn.	6.039276
Method detection limit		Median	9.8
TOTAL	7	Min.	2.9
		Max.	20.8

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9756. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 14.99

Blackwell Island Public Recreational Exposure to Surface Water

Cadmium

Analytical Results
(ug/L)

7
4.2
47.9
4.9
42.5
13.3
20.3

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	7	Mean	20.01
Censored		Lognormal mean	21.99
Detection limit or PQL		Std. devn.	18.14887
Method detection limit		Median	13.3
TOTAL	7	Min.	4.2
		Max.	47.9

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.912. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 33.34

Blackwell Island Public Recreational Exposure to Surface Water

Manganese

Analytical Results
(ug/L)

890
561
6980
942
6300
1340
2530

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	7	Mean	2791.86
Censored		Lognormal mean	2995.92
Detection limit or PQL		Std. devn.	2709.238
Method detection limit		Median	1340
TOTAL	7	Min.	561
		Max.	6980

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9019. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 4781.48

Blackwell Island Public Recreational Exposure to Surface Water

Mercury

Analytical Results (ug/L)
0.05
0.05
0.1
0.1
0.1
0.1
0.1
all nondetects

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	7	Mean	0.09
Censored		Lognormal mean	0.09
Detection limit or PQL		Std. devn.	0.024398
Method detection limit		Median	0.1
TOTAL	7	Min.	0.05
		Max.	0.1

Lognormal distribution?
r-squared is:

Normal distribution?
r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.6007. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.588. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.1

All Geographical Areas Vegetable Data

Arsenic

Analytical Results
(mg/kg)

0.048	0.003
0.012	0.002
0.026	0.016
0.003	0.017
0.005	0.011
0.009	0.016
0.026	0.008
0.008	0.053
0.020	0.003
0.018	0.006
0.005	0.011
0.007	0.074
0.003	0.113
0.015	0.004
0.004	0.010
0.029	0.019
0.021	0.034
0.012	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	35	Mean	0.02
Censored		Lognormal mean	0.02
Detection limit or PQL		Std. devn.	0.022531
Method detection limit		Median	0.01175
TOTAL	35	Min.	0.00246
		Max.	0.1128

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9728. This exceeds the tabled value of 0.934

UCL (based on t-statistic) is 0.03

All Geographical Areas Vegetable Data

Cadmium

Analytical Results
(mg/kg)

0.603	0.023
0.089	0.032
0.187	0.225
0.021	0.325
0.095	0.208
0.043	0.379
0.103	0.166
0.053	1.849
0.465	0.086
0.209	0.222
0.232	0.113
0.086	0.039
0.118	0.102
0.762	0.048
0.153	0.192
0.068	0.072
0.128	0.165
0.123	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	35	Mean	0.22
Censored		Lognormal mean	0.21
Detection limit or PQL		Std. devn.	0.326296
Method detection limit		Median	0.12267
TOTAL	35	Min.	0.02128
		Max.	1.84898

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9799. This exceeds the tabled value of 0.934

UCL (based on t-statistic) is 0.32

All Geographical Areas Bullhead Fish Data

Cadmium

Analytical Results
(mg/kg)

0.05	0.005	0.005	0.03	0.014
0.005	0.015	0.005	0.005	0.005
0.005	0.005	0.005	0.005	0.005
0.005	0.015	0.005	0.02	0.005
0.005	0.013	0.005	0.005	0.005
0.005	0.005	0.005	0.005	0.005
0.005	0.014	0.005	0.005	
0.005	0.012	0.005	0.005	
0.005	0.069	0.005	0.005	
0.005	0.011	0.005	0.005	
0.005	0.017	0.005	0.005	
0.005	0.01	0.005	0.025	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.005	
0.015	0.005	0.005	0.005	
0.012	0.005	0.005	0.005	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.01	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.012	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.005	
0.005	0.005	0.005	0.005	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	126	Mean	0.01
Censored		Lognormal mean	0.01
Detection limit or PQL		Std. devn.	0.007837
Method detection limit		Median	0.005
TOTAL	126	Min.	0.005
		Max.	0.069

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -43.9846. This lies outside the tabled values of 1.3654 and -2.5
Reject normal distribution.
Y value is -57.7151. This lies outside the tabled values of 1.3654 and -2.5

UCL (based on t-statistic) is 0.01

All Geographical Areas Bullhead Fish Data

Mercury

Analytical Results
(mg/kg)

0.025	0.05	0.025	0.025	0.09
0.05	0.05	0.03	0.025	0.07
0.025	0.05	0.05	0.025	0.08
0.025	0.08	0.025	0.025	0.08
0.025	0.06	0.05	0.025	0.05
0.025	0.025	0.05	0.025	
0.025	0.09	0.06	0.025	
0.07	0.025	0.025	0.025	
0.05	0.11	0.025	0.025	
0.025	0.09	0.025	0.025	
0.05	0.21	0.03	0.08	
0.025	0.025	0.025	0.025	
0.025	0.05	0.05	0.025	
0.025	0.03	0.025	0.025	
0.09	0.03	0.025	0.025	
0.06	0.08	0.025	0.025	
0.06	0.05	0.025	0.07	
0.025	0.025	0.05	0.12	
0.025	0.05	0.06	0.05	
0.025	0.05	0.03	0.025	
0.025	0.03	0.05	0.025	
0.025	0.025	0.07	0.06	
0.06	0.07	0.1	0.11	
0.05	0.025	0.025	0.025	
0.025	0.06	0.07	0.06	
0.06	0.05	0.025	0.025	
0.05	0.05	0.07	0.09	
0.06	0.05	0.025	0.025	
0.05	0.06	0.025	0.09	
0.06	0.05	0.025	0.18	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	125	Mean	0.05
Censored		Lognormal mean	0.05
Detection limit or PQL		Std. devn.	0.030096
Method detection limit		Median	0.03
TOTAL	125	Min.	0.025
		Max.	0.21

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -5.6295. This lies outside the tabled values of 1.363 and -2.502
Reject normal distribution.
Y value is -17.0153. This lies outside the tabled values of 1.363 and -2.502

UCL (based on t-statistic) is 0.05

All Geographical Areas Northern Pike Fish Data

Cadmium

Analytical Results
(mg/kg)

0.005	0.005	0.015
0.005	0.005	0.014
0.005	0.005	0.005
0.005	0.005	
0.011	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.02	
0.005	0.005	
0.005	0.014	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.014	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	
0.005	0.005	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	63	Mean	0.01
Censored		Lognormal mean	0.01
Detection limit or PQL		Std. devn.	0.00298
Method detection limit		Median	0.005
TOTAL	63	Min.	0.005
		Max.	0.02

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
Y value is -36.0566. This lies outside the tabled values of 1.1333 and -2.6849
Reject normal distribution.
Y value is -36.8385. This lies outside the tabled values of 1.1333 and -2.6849

UCL (based on t-statistic) is 0.01

All Geographical Areas Northern Pike Fish Data

Mercury

Analytical Results
(mg/kg)

0.11	0.07	0.025
0.03	0.03	0.48
0.08	0.16	0.4
0.1	0.11	
0.11	0.025	
0.12	0.09	
0.1	0.025	
0.09	0.12	
0.12	0.025	
0.12	0.19	
0.19	0.08	
0.025	0.3	
0.09	0.025	
0.1	0.05	
0.12	0.025	
0.15	0.025	
0.15	0.1	
0.09	0.08	
0.13	0.11	
0.2	0.06	
0.22	0.17	
0.025	0.05	
0.025	0.1	
0.22	0.11	
0.11	0.07	
0.24	0.025	
0.15	0.1	
0.1	0.06	
0.09	0.18	
0.13	0.19	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	63	Mean	0.11
Censored		Lognormal mean	0.12
Detection limit or PQL		Std. devn.	0.085625
Method detection limit		Median	0.1
TOTAL	63	Min.	0.025
		Max.	0.48

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is -1.6301. This lies within the tabled values of 1.1333 and -2.6849

UCL (based on t-statistic) is 0.13

All Geographical Areas Perch Fish Data

Cadmium

Analytical Results
(mg/kg)

0.06	0.058	0.011	0.005	0.005
0.05	0.1	0.022	0.064	0.019
0.034	0.061	0.014	0.019	0.005
0.016	0.019	0.005	0.005	
0.133	0.005	0.013	0.005	
0.033	0.037	0.016	0.021	
0.012	0.097	0.09	0.005	
0.028	0.014	0.115	0.005	
0.011	0.005	0.099	0.005	
0.041	0.005	0.038	0.013	
0.02	0.005	0.035	0.015	
0.016	0.049	0.039	0.01	
0.015	0.015	0.027	0.04	
0.049	0.005	0.01	0.005	
0.014	0.078	0.015	0.012	
0.035	0.081	0.011	0.005	
0.021	0.056	0.012	0.005	
0.055	0.045	0.054	0.005	
0.038	0.061	0.052	0.005	
0.064	0.034	0.052	0.005	
0.047	0.071	0.038	0.036	
0.075	0.082	0.01	0.005	
0.169	0.061	0.005	0.005	
0.011	0.05	0.005	0.02	
0.065	0.109	0.119	0.022	
0.015	0.045	0.005	0.005	
0.005	0.092	0.042	0.005	
0.01	0.039	0.018	0.005	
0.005	0.016	0.012	0.005	
0.039	0.011	0.019	0.005	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	123	Mean	0.03
Censored		Lognormal mean	0.03
Detection limit or PQL		Std. devn.	0.032234
Method detection limit		Median	0.019
TOTAL	123	Min.	0.005
		Max.	0.169

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is 1.1584. This lies within the tabled values of 1.3582 and -2.506

UCL (based on t-statistic) is 0.04

All Geographical Areas Perch Fish Data

Mercury

Analytical Results
(mg/kg)

0.05	0.11	0.025	0.025
0.04	0.2	0.025	0.025
0.05	0.09	0.08	0.025
0.14	0.06	0.06	0.025
0.06	0.2	0.09	0.025
0.025	0.21	0.08	0.05
0.15	0.07	0.025	0.07
0.11	0.07	0.05	0.06
0.07	0.025	0.025	0.025
0.13	0.2	0.2	0.05
0.08	0.21	0.025	0.05
0.08	0.05	0.1	0.14
0.12	0.045	0.17	0.08
0.05	0.025	0.025	0.05
0.15	0.025	0.09	0.12
0.17	0.03	0.05	0.16
0.06	0.025	0.025	0.05
0.12	0.045	0.17	0.025
0.1	0.17	0.11	0.06
0.13	0.03	0.23	0.15
0.08	0.025	0.2	0.025
0.15	0.12	0.025	0.05
0.11	0.08	0.025	0.16
0.1	0.08	0.025	0.17
0.05	0.025	0.025	0.025
0.07	0.025	0.025	0.07
0.055	0.025	0.15	0.08
0.07	0.16	0.07	0.09
0.12	0.05	0.025	
0.025	0.09	0.035	

MTCA*Stat* 2.1

Number of samples		Uncensored values	
Uncensored	118	Mean	0.08
Censored		Lognormal mean	0.08
Detection limit or PQL		Std. devn.	0.055646
Method detection limit		Median	0.065
TOTAL	118	Min.	0.025
		Max.	0.23

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
Y value is 0.9808. This lies within the tabled values of 1.3462 and -2.516

UCL (based on t-statistic) is 0.09

APPENDIX G

95 Percent Upper Confidence Limit Statistical Summary Results by Geographical Area and Medium

Appendix G

95 Percent Upper Confidence Limit Statistical Summary Results by Geographical Area and Medium

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The tables in this appendix are the Systat *v*.9 results for each metal of concern. Systat *v*.9 was used to calculate a UCL_{95} for lognormally and neither lognormally nor normally distributed data. The UCL_{95} values for data that fall into these two distribution categories can also be found in the Table 3 series in Appendix A. Systat *v*.9 uses the bootstrap method, a nonparametric statistical technique, to calculate the UCL_{95}.

Appendix G

95 Percent Upper Confidence Limit Statistical Summary Results by Geographical Area and Medium

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The tables in this appendix are the Systat *v*.9 results for each metal of concern. Systat *v*.9 was used to calculate a UCL_{95} for lognormally and neither lognormally nor normally distributed data. The UCL_{95} values for data that fall into these two distribution categories can also be found in the Table 3 series in Appendix A. Systat *v*.9 uses the bootstrap method, a nonparametric statistical technique, to calculate the UCL_{95}.

Lower Basin

3.1.1 Lower Basin Residential Surface Soil - Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	8.245	476.000	95.100
2	Mean	8.246	477.000	95.300
3	Mean	8.294	478.000	95.500
4	Mean	8.321	479.000	95.700
5	Mean	8.325	480.000	95.900

3.1.1 Lower Basin Residential Surface Soil - Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	48.529	476.000	95.100
2	Mean	48.729	477.000	95.300
3	Mean	48.911	478.000	95.500
4	Mean	48.950	479.000	95.700
5	Mean	48.993	480.000	95.900

3.1.1 Lower Basin Residential Surface Soil - Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	6.865	476.000	95.100
2	Mean	6.887	477.000	95.300
3	Mean	6.891	478.000	95.500
4	Mean	6.941	479.000	95.700
5	Mean	6.951	480.000	95.900

3.1.1 Lower Basin Residential Surface Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	37702.609	476.000	95.100
2	Mean	37735.600	477.000	95.300
3	Mean	38077.407	478.000	95.500
4	Mean	38171.481	479.000	95.700
5	Mean	38189.130	480.000	95.900

3.1.1 Lower Basin Residential Surface Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	2292.080	476.000	95.100
2	Mean	2293.500	477.000	95.300
3	Mean	2295.815	478.000	95.500
4	Mean	2303.040	479.000	95.700
5	Mean	2314.160	480.000	95.900

3.1.1 Lower Basin Residential Surface Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	1199.321	476.000	95.100
2	Mean	1200.014	477.000	95.300
3	Mean	1202.407	478.000	95.500
4	Mean	1205.743	479.000	95.700
5	Mean	1209.732	480.000	95.900

3.1.3 Lower Basin Neighborhood and Public Recreational Soil/Sediment – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	23.150	476.000	95.100
2	Mean	23.198	477.000	95.300
3	Mean	23.203	478.000	95.500
4	Mean	23.206	479.000	95.700
5	Mean	23.222	480.000	95.900

3.1.3 Lower Basin Neighborhood and Public Recreational Soil/Sediment – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	119.449	476.000	95.100
2	Mean	119.579	477.000	95.300
3	Mean	119.648	478.000	95.500
4	Mean	119.666	479.000	95.700
5	Mean	119.752	480.000	95.900

3.1.3 Lower Basin Neighborhood and Public Recreational Soil/Sediment – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	35.047	476.000	95.100
2	Mean	35.054	477.000	95.300
3	Mean	35.081	478.000	95.500
4	Mean	35.105	479.000	95.700
5	Mean	35.108	480.000	95.900

3.1.3 Lower Basin Neighborhood and Public Recreational Soil/Sediment – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	105451.381	476.000	95.100
2	Mean	105478.186	477.000	95.300
3	Mean	105546.570	478.000	95.500
4	Mean	105550.121	479.000	95.700
5	Mean	105577.706	480.000	95.900

3.1.3 Lower Basin Neighborhood and Public Recreational Soil/Sediment – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	9875.174	476.000	95.100
2	Mean	9879.651	477.000	95.300
3	Mean	9882.061	478.000	95.500
4	Mean	9895.101	479.000	95.700
5	Mean	9912.881	480.000	95.900

3.1.3 Lower Basin Neighborhood and Public Recreational Soil/Sediment – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	5666.072	476.000	95.100
2	Mean	5666.730	477.000	95.300
3	Mean	5669.737	478.000	95.500
4	Mean	5672.788	479.000	95.700
5	Mean	5677.255	480.000	95.900

3.1.4 Lower Basin Neighborhood and Public Recreational Surface Water – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	79.607	238.000	95.000
2	Mean	79.614	239.000	95.400
3	Mean	79.866	240.000	95.800

3.1.4 Lower Basin Neighborhood and Public Recreational Surface Water – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	72.901	238.000	95.000
2	Mean	73.142	239.000	95.400
3	Mean	73.384	240.000	95.800

3.1.4 Lower Basin Neighborhood and Public Recreational Surface Water – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	16650.590	238.000	95.000
2	Mean	16673.107	239.000	95.400
3	Mean	16734.713	240.000	95.800

3.1.4 Lower Basin Neighborhood and Public Recreational Surface Water – Mercury

	STATISTC$	HG	RHG	PCTILE
1	Mean	4.497	238.000	95.000
2	Mean	4.525	239.000	95.400
3	Mean	4.619	240.000	95.800

3.1.5 Lower Basin Subsistence Sediment – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	25.201	238.000	95.000
2	Mean	25.239	239.000	95.400
3	Mean	25.286	240.000	95.800

3.1.5 Lower Basin Subsistence Sediment – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	120.955	238.000	95.000
2	Mean	121.138	239.000	95.400
3	Mean	121.241	240.000	95.800

3.1.5 Lower Basin Subsistence Sediment – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	39.118	238.000	95.000
2	Mean	39.180	239.000	95.400
3	Mean	39.366	240.000	95.800

3.1.5 Lower Basin Subsistence Sediment – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	113072.940	238.000	95.000
2	Mean	113259.511	239.000	95.400
3	Mean	113368.052	240.000	95.800

3.1.5 Lower Basin Subsistence Sediment –Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	10700.152	238.000	95.000
2	Mean	10710.812	239.000	95.400
3	Mean	10770.307	240.000	95.800

3.1.5 Lower Basin Subsistence Sediment – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	6400.174	238.000	95.000
2	Mean	6402.391	239.000	95.400
3	Mean	6402.969	240.000	95.800

3.1.6 Lower Basin Subsistence Plant Tissue (Water Potato) – Cadmium (without skin)

	STATISTC$	CD	RCD	PCTILE
1	Mean	0.291	238.000	95.000
2	Mean	0.293	239.000	95.400
3	Mean	0.293	240.000	95.800

3.1.6 Lower Basin Subsistence Plant Tissue (Water Potato) – Cadmium (with skin)

	STATISTC$	CD	RCD	PCTILE
1	Mean	0.489	238.000	95.000
2	Mean	0.496	239.000	95.400
3	Mean	0.503	240.000	95.800

3.1.7 Lower Basin Occupational Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	20.999	476.000	95.100
2	Mean	21.008	477.000	95.300
3	Mean	21.034	478.000	95.500
4	Mean	21.054	479.000	95.700
5	Mean	21.056	480.000	95.900

3.1.7 Lower Basin Occupational Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	108.907	476.000	95.100
2	Mean	109.013	477.000	95.300
3	Mean	109.092	478.000	95.500
4	Mean	109.107	479.000	95.700
5	Mean	109.132	480.000	95.900

3.1.7 Lower Basin Occupational Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	30.705	476.000	95.100
2	Mean	30.743	477.000	95.300
3	Mean	30.783	478.000	95.500
4	Mean	30.862	479.000	95.700
5	Mean	30.889	480.000	95.900

3.1.7 Lower Basin Occupational Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	97012.260	476.000	95.100
2	Mean	97072.912	477.000	95.300
3	Mean	97091.677	478.000	95.500
4	Mean	97103.526	479.000	95.700
5	Mean	97132.094	480.000	95.900

3.1.7 Lower Basin Occupational Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	8897.224	476.000	95.100
2	Mean	8897.973	477.000	95.300
3	Mean	8903.207	478.000	95.500
4	Mean	8906.788	479.000	95.700
5	Mean	8911.738	480.000	95.900

3.1.7 Lower Basin Occupational Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	4997.719	476.000	95.100
2	Mean	4998.346	477.000	95.300
3	Mean	4998.969	478.000	95.500
4	Mean	5004.556	479.000	95.700
5	Mean	5007.346	480.000	95.900

3.1.8 Lower Basin Subsistence Surface Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	124.444	476.000	95.100
2	Mean	124.519	477.000	95.300
3	Mean	124.585	478.000	95.500
4	Mean	124.619	479.000	95.700
5	Mean	124.774	480.000	95.900

3.1.8 Lower Basin Subsistence Surface Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	21.163	476.000	95.100
2	Mean	21.241	477.000	95.300
3	Mean	21.247	478.000	95.500
4	Mean	21.284	479.000	95.700
5	Mean	21.287	480.000	95.900

3.1.8 Lower Basin Subsistence Surface Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	30.451	476.000	95.100
2	Mean	30.477	477.000	95.300
3	Mean	30.480	478.000	95.500
4	Mean	30.485	479.000	95.700
5	Mean	30.539	480.000	95.900

3.1.8 Lower Basin Subsistence Surface Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	97440.097	476.000	95.100
2	Mean	97489.284	477.000	95.300
3	Mean	97496.742	478.000	95.500
4	Mean	97560.361	479.000	95.700
5	Mean	97590.277	480.000	95.900

3.1.8 Lower Basin Subsistence Surface Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	8960.394	476.000	95.100
2	Mean	8960.697	477.000	95.300
3	Mean	8980.110	478.000	95.500
4	Mean	8984.703	479.000	95.700
5	Mean	8985.258	480.000	95.900

3.1.8 Lower Basin Subsistence Surface Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	4856.142	476.000	95.100
2	Mean	4859.357	477.000	95.300
3	Mean	4868.925	478.000	95.500
4	Mean	4868.946	479.000	95.700
5	Mean	4870.239	480.000	95.900

3.1.9 Lower Basin Undisturbed Surface Water – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	15.214	476.000	95.100
2	Mean	15.250	477.500	95.400
3	Mean	15.250	477.500	95.400
4	Mean	15.292	479.000	95.700
5	Mean	15.313	480.000	95.900

3.1.9 Lower Basin Undisturbed Surface Water – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	3.227	476.000	95.100
2	Mean	3.237	477.000	95.300
3	Mean	3.240	478.000	95.500
4	Mean	3.249	479.000	95.700
5	Mean	3.251	480.000	95.900

3.1.9 Lower Basin Undisturbed Surface Water – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	.	.	.
2	Mean	197.000	476.000	95.100
3	Mean	197.330	477.000	95.300
4	Mean	197.700	478.000	95.500
5	Mean	198.482	479.000	95.700
6	Mean	198.667	480.000	95.900

Kingston

3.2.1 Kingston Residential Surface Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	4.062	476.000	95.100
2	Mean	4.102	477.000	95.300
3	Mean	4.127	478.000	95.500
4	Mean	4.131	479.000	95.700
5	Mean	4.167	480.000	95.900

3.2.1 Kingston Residential Surface Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	25.037	476.000	95.100
2	Mean	25.097	477.000	95.300
3	Mean	25.142	478.000	95.500
4	Mean	25.172	479.000	95.700
5	Mean	25.183	480.000	95.900

3.2.1 Kingston Residential Surface Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	3.919	476.000	95.100
2	Mean	3.967	477.000	95.300
3	Mean	3.970	478.000	95.500
4	Mean	3.976	479.000	95.700
5	Mean	4.001	480.000	95.900

3.2.1 Kingston Residential Surface Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	21970.806	476.000	95.100
2	Mean	22076.545	477.000	95.300
3	Mean	22135.263	478.000	95.500
4	Mean	22140.893	479.000	95.700
5	Mean	22158.689	480.000	95.900

3.2.1 Kingston Residential Surface Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1084.683	476.000	95.100
2	Mean	1084.845	477.000	95.300
3	Mean	1086.281	478.000	95.500
4	Mean	1087.617	479.000	95.700
5	Mean	1087.694	480.000	95.900

3.2.1 Kingston Residential Surface Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	719.155	476.000	95.100
2	Mean	721.215	477.000	95.300
3	Mean	721.731	478.000	95.500
4	Mean	723.231	479.000	95.700
5	Mean	724.132	480.000	95.900

3.2.3 Kingston Neighborhood and Public Recreational Sediment (Pine Creek) – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	12.832	476.000	95.100
2	Mean	13.040	477.000	95.300
3	Mean	13.044	478.000	95.500
4	Mean	13.088	479.000	95.700
5	Mean	13.118	480.000	95.900

3.2.3 Kingston Neighborhood and Public Recreational Sediment (Pine Creek) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	22.345	476.000	95.100
2	Mean	22.445	477.000	95.300
3	Mean	22.790	478.000	95.500
4	Mean	22.854	479.000	95.700
5	Mean	22.952	480.000	95.900

3.2.3 Kingston Neighborhood and Public Recreational Sediment (Pine Creek) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	0.917	476.000	95.100
2	Mean	0.920	477.000	95.300
3	Mean	0.920	478.000	95.500
4	Mean	0.921	479.000	95.700
5	Mean	0.927	480.000	95.900

3.2.3 Kingston Neighborhood and Public Recreational Sediment (Pine Creek) – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	16300.000	476.000	95.100
2	Mean	16300.000	477.000	95.300
3	Mean	16313.077	478.000	95.500
4	Mean	16338.462	479.000	95.700
5	Mean	16338.462	480.000	95.900

3.2.3 Kingston Neighborhood and Public Recreational Sediment (Pine Creek) – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	451.615	476.000	95.100
2	Mean	453.231	477.000	95.300
3	Mean	454.077	478.000	95.500
4	Mean	454.846	479.000	95.700
5	Mean	455.846	480.000	95.900

3.2.3 Kingston Neighborhood and Public Recreational Sediment (Pine Creek) – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	320.308	476.000	95.100
2	Mean	320.308	477.000	95.300
3	Mean	320.615	478.000	95.500
4	Mean	320.692	479.000	95.700
5	Mean	320.692	480.000	95.900

3.2.4 Kingston Neighborhood and Public Recreational Surface Water (Pine Creek) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	0.725	476.000	95.100
2	Mean	0.725	477.000	95.300
3	Mean	0.727	478.500	95.600
4	Mean	0.727	478.500	95.600
5	Mean	0.728	480.000	95.900

3.2.4 Kingston Neighborhood and Public Recreational Surface Water (Pine Creek) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	1.080	475.500	95.000
2	Mean	1.080	475.500	95.000
3	Mean	1.082	477.000	95.300
4	Mean	1.090	478.000	95.500
5	Mean	1.093	479.000	95.700
6	Mean	1.095	480.000	95.900

3.2.4 Kingston Neighborhood and Public Recreational Surface Water (Pine Creek) – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	38.458	476.000	95.100
2	Mean	38.508	477.000	95.300
3	Mean	38.517	478.000	95.500
4	Mean	38.600	479.000	95.700
5	Mean	38.642	480.000	95.900

3.2.4 Kingston Neighborhood and Public Recreational Surface Water (Pine Creek) – Mercury

	STATISTC$	HG	RHG	PCTILE
1	Mean	0.088	480.000	95.900
2	Mean	0.088	480.000	95.900
3	Mean	0.088	480.000	95.900
4	Mean	0.088	480.000	95.900
5	Mean	0.088	480.000	95.900

3.2.5 Kingston Neighborhood and Public Recreational Soil/Sediment (NS Confluence) – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	39.779	476.000	95.100
2	Mean	39.868	477.000	95.300
3	Mean	39.979	478.000	95.500
4	Mean	40.332	479.000	95.700
5	Mean	40.358	480.000	95.900

3.2.5 Kingston Neighborhood and Public Recreational Soil/Sediment (NS Confluence) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	163.200	476.000	95.100
2	Mean	163.263	477.000	95.300
3	Mean	163.684	478.000	95.500
4	Mean	164.105	479.000	95.700
5	Mean	164.263	480.000	95.900

3.2.5 Kingston Neighborhood and Public Recreational Soil/Sediment (NS Confluence) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	26.584	476.000	95.100
2	Mean	26.611	477.000	95.300
3	Mean	26.616	478.000	95.500
4	Mean	26.821	479.000	95.700
5	Mean	26.868	480.000	95.900

3.2.5 Kingston Neighborhood and Public Recreational Soil/Sediment (NS Confluence) – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	100621.053	476.000	95.100
2	Mean	100705.263	477.000	95.300
3	Mean	100726.316	478.000	95.500
4	Mean	100836.842	479.000	95.700
5	Mean	100857.895	480.000	95.900

3.2.5 Kingston Neighborhood and Public Recreational Soil/Sediment (NS Confluence) – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	8585.263	476.000	95.100
2	Mean	8586.842	477.000	95.300
3	Mean	8594.211	478.000	95.500
4	Mean	8651.053	479.000	95.700
5	Mean	8671.053	480.000	95.900

3.2.5 Kingston Neighborhood and Public Recreational Soil/Sediment (NS Confluence) – Zinc

	STATISTCS	ZN	RZN	PCTILE
1	Mean	3743.684	476.000	95.100
2	Mean	3745.263	477.000	95.300
3	Mean	3745.789	478.000	95.500
4	Mean	3748.421	479.000	95.700
5	Mean	3756.316	480.000	95.900

3.2.7 Kingston Occupational Soil – Antimony

	STATISTCS	SB	RSB	PCTILE
1	Mean	6.858	238.000	95.000
2	Mean	6.958	239.000	95.400
3	Mean	7.107	240.000	95.800

3.2.7 Kingston Occupational Soil – Arsenic

	STATISTCS	AS	RAS	PCTILE
1	Mean	34.603	476.000	95.100
2	Mean	34.726	477.000	95.300
3	Mean	34.728	478.000	95.500
4	Mean	34.785	479.000	95.700
5	Mean	34.886	480.000	95.900

3.2.7 Kingston Occupational Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	5.095	476.000	95.100
2	Mean	5.106	477.000	95.300
3	Mean	5.108	478.000	95.500
4	Mean	5.118	479.000	95.700
5	Mean	5.125	480.000	95.900

3.2.7 Kingston Occupational Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	29003.098	476.000	95.100
2	Mean	29038.297	477.000	95.300
3	Mean	29042.618	478.000	95.500
4	Mean	29148.871	479.000	95.700
5	Mean	29172.760	480.000	95.900

3.2.7 Kingston Occupational Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1692.848	476.000	95.100
2	Mean	1697.649	477.000	95.300
3	Mean	1697.686	478.000	95.500
4	Mean	1707.642	479.000	95.700
5	Mean	1709.878	480.000	95.900

3.2.7 Kingston Occupational Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	1057.311	476.000	95.100
2	Mean	1061.259	477.000	95.300
3	Mean	1062.900	478.000	95.500
4	Mean	1063.816	479.000	95.700
5	Mean	1066.092	480.000	95.900

Side Gulches

3.3.1 Side Gulches Residential Surface Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	15.943	476.000	95.100
2	Mean	15.971	477.000	95.300
3	Mean	16.146	478.000	95.500
4	Mean	16.358	479.000	95.700
5	Mean	16.460	480.000	95.900

3.3.1 Side Gulches Residential Surface Soil –Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	50.743	476.000	95.100
2	Mean	50.781	477.000	95.300
3	Mean	50.805	478.000	95.500
4	Mean	51.011	479.000	95.700
5	Mean	51.198	480.000	95.900

3.3.1 Side Gulches Residential Surface Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	9.621	476.000	95.100
2	Mean	9.663	477.000	95.300
3	Mean	9.676	478.000	95.500
4	Mean	9.742	479.000	95.700
5	Mean	9.756	480.000	95.900

3.3.1 Side Gulches Residential Surface Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	27189.630	476.000	95.100
2	Mean	27246.543	477.000	95.300
3	Mean	27246.667	478.000	95.500
4	Mean	27399.877	479.000	95.700
5	Mean	27435.802	480.000	95.900

3.3.1 Side Gulches Residential Surface Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1366.914	476.000	95.100
2	Mean	1369.358	477.000	95.300
3	Mean	1371.049	478.000	95.500
4	Mean	1382.099	479.000	95.700
5	Mean	1382.136	480.000	95.900

3.3.1 Side Gulches Residential Surface Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	1319.896	476.000	95.100
2	Mean	1321.953	477.000	95.300
3	Mean	1326.433	478.000	95.500
4	Mean	1332.690	479.000	95.700
5	Mean	1338.917	480.000	95.900

3.3.4 Side Gulches Neighborhood Recreational Sediment – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	27.522	476.000	95.100
2	Mean	27.590	477.000	95.300
3	Mean	27.719	478.000	95.500
4	Mean	27.742	479.000	95.700
5	Mean	27.759	480.000	95.900

3.3.4 Side Gulches Neighborhood Recreational Sediment –Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	77.740	476.000	95.100
2	Mean	78.130	477.000	95.300
3	Mean	78.560	478.000	95.500
4	Mean	78.720	479.000	95.700
5	Mean	79.280	480.000	95.900

3.3.4 Side Gulches Neighborhood Recreational Sediment – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	38.380	476.000	95.100
2	Mean	38.570	477.000	95.300
3	Mean	38.640	478.000	95.500
4	Mean	38.730	479.000	95.700
5	Mean	39.180	480.000	95.900

3.3.4 Side Gulches Neighborhood Recreational Sediment – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	35970.000	476.000	95.100
2	Mean	36010.000	477.000	95.300
3	Mean	36046.600	478.000	95.500
4	Mean	36090.000	479.000	95.700
5	Mean	36136.600	480.000	95.900

3.3.4 Side Gulches Neighborhood Recreational Sediment – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	2223.400	476.000	95.100
2	Mean	2235.600	477.000	95.300
3	Mean	2262.300	478.000	95.500
4	Mean	2288.000	479.000	95.700
5	Mean	2288.400	480.000	95.900

3.3.4 Side Gulches Neighborhood Recreational Sediment – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	5030.500	476.000	95.100
2	Mean	5043.840	477.000	95.300
3	Mean	5064.800	478.000	95.500
4	Mean	5128.700	479.000	95.700
5	Mean	5188.840	480.000	95.900

Osburn

3.4.1 Osburn Residential Surface Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	12.539	476.000	95.100
2	Mean	12.567	477.000	95.300
3	Mean	12.630	478.000	95.500
4	Mean	12.663	479.000	95.700
5	Mean	12.684	480.000	95.900

3.4.1 Osburn Residential Surface Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	46.740	476.000	95.100
2	Mean	46.798	477.000	95.300
3	Mean	46.849	478.000	95.500
4	Mean	46.866	479.000	95.700
5	Mean	47.200	480.000	95.900

3.4.1 Osburn Residential Surface Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	5.247	476.000	95.100
2	Mean	5.247	477.000	95.300
3	Mean	5.256	478.000	95.500
4	Mean	5.261	479.000	95.700
5	Mean	5.268	480.000	95.900

3.4.1 Osburn Residential Surface Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	22487.790	476.000	95.100
2	Mean	22493.768	477.000	95.300
3	Mean	22514.357	478.000	95.500
4	Mean	22515.160	479.000	95.700
5	Mean	22515.735	480.000	95.900

3.4.1 Osburn Residential Surface Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1199.339	476.000	95.100
2	Mean	1201.597	477.000	95.300
3	Mean	1202.372	478.000	95.500
4	Mean	1204.423	479.000	95.700
5	Mean	1208.198	480.000	95.900

3.4.1 Osburn Residential Surface Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	597.024	476.000	95.100
2	Mean	597.544	477.000	95.300
3	Mean	598.065	478.000	95.500
4	Mean	598.495	479.000	95.700
5	Mean	599.119	480.000	95.900

3.4.2 Osburn Residential Groundwater (Tap Water) - Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	0.280	476.000	95.100
2	Mean	0.281	477.000	95.300
3	Mean	0.282	478.000	95.500
4	Mean	0.282	479.000	95.700
5	Mean	0.283	480.000	95.900

Silverton

3.5.1 Silverton Residential Surface Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	8.898	476.000	95.100
2	Mean	8.964	477.000	95.300
3	Mean	9.032	478.000	95.500
4	Mean	9.040	479.000	95.700
5	Mean	9.077	480.000	95.900

3.5.1 Silverton Residential Surface Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	21.458	476.000	95.100
2	Mean	21.621	477.000	95.300
3	Mean	21.710	478.000	95.500
4	Mean	21.927	479.000	95.700
5	Mean	21.969	480.000	95.900

3.5.1 Silverton Residential Surface Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	5.439	476.000	95.100
2	Mean	5.451	477.000	95.300
3	Mean	5.474	478.000	95.500
4	Mean	5.477	479.000	95.700
5	Mean	5.480	480.000	95.900

3.5.1 Silverton Residential Surface Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	20197.818	476.000	95.100
2	Mean	20231.321	477.000	95.300
3	Mean	20245.091	478.000	95.500
4	Mean	20281.091	479.000	95.700
5	Mean	20520.000	480.000	95.900

3.5.1 Silverton Residential Surface Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1067.547	476.000	95.100
2	Mean	1069.135	477.000	95.300
3	Mean	1076.218	478.000	95.500
4	Mean	1076.255	479.000	95.700
5	Mean	1076.472	480.000	95.900

3.5.1 Silverton Residential Surface Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	815.644	476.000	95.100
2	Mean	817.878	477.000	95.300
3	Mean	818.138	478.000	95.500
4	Mean	820.787	479.000	95.700
5	Mean	824.402	480.000	95.900

3.5.3 Silverton Neighborhood and Public Recreational Surface Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	6.938	238.000	95.000
2	Mean	6.950	239.000	95.400
3	Mean	7.125	240.000	95.800

3.5.3 Silverton Neighborhood and Public Recreational Surface Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	28.441	238.000	95.000
2	Mean	28.488	239.000	95.400
3	Mean	28.628	240.000	95.800

3.5.3 Silverton Neighborhood and Public Recreational Surface Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	4.572	238.000	95.000
2	Mean	4.575	239.000	95.400
3	Mean	4.582	240.000	95.800

3.5.3 Silverton Neighborhood and Public Recreational Surface Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	20083.610	238.000	95.000
2	Mean	20136.472	239.000	95.400
3	Mean	20147.724	240.000	95.800

3.5.3 Silverton Neighborhood and Public Recreational Surface Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1089.631	238.000	95.000
2	Mean	1097.193	239.000	95.400
3	Mean	1099.029	240.000	95.800

3.5.3 Silverton Neighborhood and Public Recreational Surface Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	739.423	238.000	95.000
2	Mean	740.270	239.000	95.400
3	Mean	741.299	240.000	95.800

Wallace

3.6.1 Wallace Residential Surface Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	13.697	476.000	95.100
2	Mean	13.735	477.000	95.300
3	Mean	13.746	478.000	95.500
4	Mean	13.846	479.000	95.700
5	Mean	13.860	480.000	95.900

3.6.1 Wallace Residential Surface Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	22.574	476.000	95.100
2	Mean	22.589	477.000	95.300
3	Mean	22.640	478.000	95.500
4	Mean	22.685	479.000	95.700
5	Mean	22.733	480.000	95.900

3.6.1 Wallace Residential Surface Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	7.625	476.000	95.100
2	Mean	7.632	477.000	95.300
3	Mean	7.661	478.000	95.500
4	Mean	7.662	479.000	95.700
5	Mean	7.694	480.000	95.900

3.6.1 Wallace Residential Surface Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	22239.506	476.000	95.100
2	Mean	22255.696	477.000	95.300
3	Mean	22352.564	478.000	95.500
4	Mean	22358.974	479.000	95.700
5	Mean	22362.821	480.000	95.900

3.6.1 Wallace Residential Surface Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	989.333	476.000	95.100
2	Mean	989.835	477.000	95.300
3	Mean	992.608	478.000	95.500
4	Mean	996.272	479.000	95.700
5	Mean	996.899	480.000	95.900

3.6.1 Wallace Residential Surface Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	967.061	476.000	95.100
2	Mean	973.988	477.000	95.300
3	Mean	976.512	478.000	95.500
4	Mean	977.244	479.000	95.700
5	Mean	977.951	480.000	95.900

3.6.2 Wallace Residential Groundwater (Tap Water) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	0.494	476.000	95.100
2	Mean	0.495	477.500	95.400
3	Mean	0.495	477.500	95.400
4	Mean	0.496	480.000	95.900
5	Mean	0.496	480.000	95.900
6	Mean	0.496	480.000	95.900

3.6.3 Wallace Neighborhood and Public Recreational Surface Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	18.217	238.000	95.000
2	Mean	18.710	239.000	95.400
3	Mean	18.909	240.000	95.800

3.6.3 Wallace Neighborhood and Public Recreational Surface Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	27.647	238.000	95.000
2	Mean	27.720	239.000	95.400
3	Mean	27.804	240.000	95.800

3.6.3 Wallace Neighborhood and Public Recreational Surface Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	10.723	238.000	95.000
2	Mean	10.753	239.000	95.400
3	Mean	10.754	240.000	95.800

3.6.3 Wallace Neighborhood and Public Recreational Surface Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	28638.800	238.000	95.000
2	Mean	28667.422	239.000	95.400
3	Mean	28682.326	240.000	95.800

3.6.3 Wallace Neighborhood and Public Recreational Surface Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1502.293	238.000	95.000
2	Mean	1508.641	239.000	95.400
3	Mean	1511.233	240.000	95.800

3.6.3 Wallace Neighborhood and Public Recreational Surface Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	1921.270	238.000	95.000
2	Mean	1942.159	239.000	95.400
3	Mean	1943.686	240.000	95.800

MidGradSeg01

3.7.1 MidGradSeg01 Neighborhood Recreational Sediment (South Fork CDAR) – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	38.111	476.000	95.100
2	Mean	38.170	477.000	95.300
3	Mean	38.665	478.000	95.500
4	Mean	38.831	479.000	95.700
5	Mean	38.893	480.000	95.900

3.7.1 MidGradSeg01 Neighborhood Recreational Sediment (South Fork CDAR) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	52.640	476.000	95.100
2	Mean	52.803	477.000	95.300
3	Mean	52.981	478.000	95.500
4	Mean	53.345	479.000	95.700
5	Mean	53.640	480.000	95.900

3.7.1 MidGradSeg01 Neighborhood Recreational Sediment (South Fork CDAR) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	18.890	476.000	95.100
2	Mean	18.900	477.000	95.300
3	Mean	19.014	478.000	95.500
4	Mean	19.044	479.000	95.700
5	Mean	19.076	480.000	95.900

3.7.1 MidGradSeg01 Neighborhood Recreational Sediment (South Fork CDAR) – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	41314.286	476.000	95.100
2	Mean	41338.095	477.000	95.300
3	Mean	41347.619	478.000	95.500
4	Mean	41461.905	479.000	95.700
5	Mean	41466.667	480.000	95.900

3.7.1 MidGradSeg01 Neighborhood Recreational Sediment (South Fork CDAR) – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	3754.762	476.000	95.100
2	Mean	3758.571	477.000	95.300
3	Mean	3766.190	478.000	95.500
4	Mean	3771.905	479.000	95.700
5	Mean	3775.714	480.000	95.900

3.7.1 MidGradSeg01 Neighborhood Recreational Sediment (South Fork CDAR) – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	3396.667	476.000	95.100
2	Mean	3402.000	477.000	95.300
3	Mean	3407.714	478.000	95.500
4	Mean	3419.619	479.000	95.700
5	Mean	3427.714	480.000	95.900

3.7.2 MidGradSeg01 Neighborhood Recreational Surface Water (South Fork CDAR) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	1.096	238.000	95.000
2	Mean	1.115	239.000	95.400
3	Mean	1.135	240.000	95.800

3.7.2 MidGradSeg01 Neighborhood Recreational Surface Water (South Fork CDAR) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	4.029	238.000	95.000
2	Mean	4.032	239.000	95.400
3	Mean	4.053	240.000	95.800

3.7.2 MidGradSeg01 Neighborhood Recreational Surface Water (South Fork CDAR) – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	32.040	238.000	95.000
2	Mean	32.418	239.000	95.400
3	Mean	32.482	240.000	95.800

3.7.2 MidGradSeg01 Neighborhood Recreational Surface Water (South Fork CDAR) – Mercury

	STATISTC$	HG	RHG	PCTILE
1	Mean	0.122	238.000	95.000
2	Mean	0.122	239.000	95.400
3	Mean	0.126	240.000	95.800

3.7.3 MidGradSeg01 Occupational Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	9.262	238.000	95.000
2	Mean	9.280	239.000	95.400
3	Mean	9.344	240.000	95.800

3.7.3 MidGradSeg01 Occupational Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	27.311	238.000	95.000
2	Mean	27.388	239.000	95.400
3	Mean	27.464	240.000	95.800

3.7.3 MidGradSeg01 Occupational Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	5.206	238.000	95.000
2	Mean	5.213	239.000	95.400
3	Mean	5.217	240.000	95.800

3.7.3 MidGradSeg01 Occupational Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	21872.006	476.000	95.100
2	Mean	21880.237	477.000	95.300
3	Mean	21881.846	478.000	95.500
4	Mean	21883.686	479.000	95.700
5	Mean	21886.368	480.000	95.900

3.7.3 MidGradSeg01 Occupational Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1129.167	238.000	95.000
2	Mean	1129.221	239.000	95.400
3	Mean	1129.796	240.000	95.800

3.7.3 MidGradSeg01 Occupational Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	751.761	238.000	95.000
2	Mean	753.086	239.000	95.400
3	Mean	756.657	240.000	95.800

Ninemile

3.8.1 Nine Mile Residential Surface Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	11.939	476.000	95.100
2	Mean	11.947	477.000	95.300
3	Mean	12.041	478.000	95.500
4	Mean	12.107	479.000	95.700
5	Mean	12.169	480.000	95.900

3.8.1 Nine Mile Residential Surface Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	41.619	476.000	95.100
2	Mean	42.005	477.000	95.300
3	Mean	42.470	478.000	95.500
4	Mean	42.489	479.000	95.700
5	Mean	42.492	480.000	95.900

3.8.1 Nine Mile Residential Surface Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	6.517	476.000	95.100
2	Mean	6.525	477.000	95.300
3	Mean	6.525	478.000	95.500
4	Mean	6.526	479.000	95.700
5	Mean	6.527	480.000	95.900

3.8.1 Nine Mile Residential Surface Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	23310.991	476.000	95.100
2	Mean	23319.447	477.000	95.300
3	Mean	23332.037	478.000	95.500
4	Mean	23389.954	479.000	95.700
5	Mean	23455.070	480.000	95.900

3.8.1 Nine Mile Residential Surface Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1249.986	476.000	95.100
2	Mean	1253.760	477.000	95.300
3	Mean	1254.434	478.000	95.500
4	Mean	1257.829	479.000	95.700
5	Mean	1258.118	480.000	95.900

3.8.1 Nine Mile Residential Surface Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	1159.114	476.000	95.100
2	Mean	1160.014	477.000	95.300
3	Mean	1161.309	478.000	95.500
4	Mean	1162.505	479.000	95.700
5	Mean	1162.618	480.000	95.900

3.8.2 Nine Mile Residential Ground Water (Tap Water-Current/Future) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	0.735	476.000	95.100
2	Mean	0.744	477.000	95.300
3	Mean	0.749	478.000	95.500
4	Mean	0.760	479.000	95.700
5	Mean	0.764	480.000	95.900

3.8.3 Nine Mile Residential Ground Water (Tap Water-Future) – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	2.106	238.000	95.000
2	Mean	2.156	239.000	95.400
3	Mean	2.195	240.000	95.800

3.8.3 Nine Mile Residential Ground Water (Tap Water-Future) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	1.251	238.000	95.000
2	Mean	1.265	239.000	95.400
3	Mean	1.267	240.000	95.800

3.8.3 Nine Mile Residential Ground Water (Tap Water-Future) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	130.854	238.000	95.000
2	Mean	131.695	239.000	95.400
3	Mean	131.809	240.000	95.800

3.8.3 Nine Mile Residential Ground Water (Tap Water-Future) – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	19755.511	238.000	95.000
2	Mean	20191.374	239.000	95.400
3	Mean	20191.901	240.000	95.800

3.8.4 Nine Mile Neighborhood Recreational Surface Soil (Waste Piles) – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	42.344	476.000	95.100
2	Mean	43.015	477.000	95.300
3	Mean	43.448	478.000	95.500
4	Mean	43.595	479.000	95.700
5	Mean	44.139	480.000	95.900

3.8.4 Nine Mile Neighborhood Recreational Surface Soil (Waste Piles) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	518.491	476.000	95.100
2	Mean	519.218	477.000	95.300
3	Mean	519.545	478.000	95.500
4	Mean	520.032	479.000	95.700
5	Mean	520.959	480.000	95.900

3.8.4 Nine Mile Neighborhood Recreational Surface Soil (Waste Piles) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	31.672	476.000	95.100
2	Mean	32.333	477.000	95.300
3	Mean	32.455	478.000	95.500
4	Mean	32.456	479.000	95.700
5	Mean	32.897	480.000	95.900

3.8.4 Nine Mile Neighborhood Recreational Surface Soil (Waste Piles) – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	43031.818	476.000	95.100
2	Mean	43186.364	477.000	95.300
3	Mean	43372.727	478.000	95.500
4	Mean	43559.091	479.000	95.700
5	Mean	43768.182	480.000	95.900

3.8.4 Nine Mile Neighborhood Recreational Surface Soil (Waste Piles) – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1720.682	476.000	95.100
2	Mean	1721.955	477.000	95.300
3	Mean	1725.500	478.000	95.500
4	Mean	1725.818	479.000	95.700
5	Mean	1727.409	480.000	95.900

3.8.4 Nine Mile Neighborhood Recreational Surface Soil (Waste Piles) – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	6195.500	476.000	95.100
2	Mean	6236.136	477.000	95.300
3	Mean	6335.045	478.000	95.500
4	Mean	6345.955	479.000	95.700
5	Mean	6347.500	480.000	95.900

3.8.5 Nine Mile Neighborhood Recreational Sediment (Nine Mile/Canyon Creek) – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	71.249	476.000	95.100
2	Mean	71.761	477.000	95.300
3	Mean	73.072	478.000	95.500
4	Mean	73.326	479.000	95.700
5	Mean	73.553	480.000	95.900

3.8.5 Nine Mile Neighborhood Recreational Sediment (Nine Mile/Canyon Creek) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	50.618	476.000	95.100
2	Mean	51.526	477.000	95.300
3	Mean	51.560	478.000	95.500
4	Mean	52.074	479.000	95.700
5	Mean	52.111	480.000	95.900

3.8.5 Nine Mile Neighborhood Recreational Sediment (Nine Mile/Canyon Creek) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	59.196	476.000	95.100
2	Mean	59.398	477.000	95.300
3	Mean	60.172	478.000	95.500
4	Mean	60.195	479.000	95.700
5	Mean	60.492	480.000	95.900

3.8.5 Nine Mile Neighborhood Recreational Sediment (Nine Mile/Canyon Creek) – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	36475.882	476.000	95.100
2	Mean	36522.353	477.000	95.300
3	Mean	36605.294	478.000	95.500
4	Mean	36758.235	479.000	95.700
5	Mean	36834.706	480.000	95.900

3.8.5 Nine Mile Neighborhood Recreational Sediment (Nine Mile/Canyon Creek) – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	2626.353	476.000	95.100
2	Mean	2631.588	477.000	95.300
3	Mean	2651.412	478.000	95.500
4	Mean	2657.176	479.000	95.700
5	Mean	2678.941	480.000	95.900

3.8.5 Nine Mile Neighborhood Recreational Sediment (Nine Mile/Canyon Creek) – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	8883.412	476.000	95.100
2	Mean	8883.529	477.000	95.300
3	Mean	8944.588	478.000	95.500
4	Mean	8977.471	479.000	95.700
5	Mean	9000.294	480.000	95.900

3.8.6 Nine Mile Neighborhood Recreational Surface Water (Nine Mile/Canyon Creek) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	1.102	238.000	95.000
2	Mean	1.128	239.000	95.400
3	Mean	1.128	240.000	95.800

3.8.6 Nine Mile Neighborhood Recreational Surface Water (Nine Mile/Canyon Creek) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	111.023	238.000	95.000
2	Mean	112.776	239.000	95.400
3	Mean	113.778	240.000	95.800

3.8.6 Nine Mile Neighborhood Recreational Surface Water (Nine Mile/Canyon Creek) – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1699.390	238.000	95.000
2	Mean	1719.511	239.000	95.400
3	Mean	1721.198	240.000	95.800

3.8.6 Nine Mile Neighborhood Recreational Surface Water (Nine Mile/Canyon Creek) – Mercury

	STATISTC$	HG	RHG	PCTILE
1	Mean	0.305	238.000	95.000
2	Mean	0.307	239.000	95.400
3	Mean	0.309	240.000	95.800

3.8.7 Nine Mile Occupational Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	10.608	238.000	95.000
2	Mean	10.618	239.000	95.400
3	Mean	10.667	240.000	95.800

3.8.7 Nine Mile Occupational Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	24.967	238.000	95.000
2	Mean	25.105	239.000	95.400
3	Mean	25.124	240.000	95.800

3.8.7 Nine Mile Occupational Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	6.539	238.000	95.000
2	Mean	6.547	239.000	95.400
3	Mean	6.607	240.000	95.800

3.8.7 Nine Mile Occupational Soil –Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	21975.915	238.000	95.000
2	Mean	21986.652	239.000	95.400
3	Mean	22000.874	240.000	95.800

3.8.7 Nine Mile Occupational Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1134.735	238.000	95.000
2	Mean	1135.083	239.000	95.400
3	Mean	1135.326	240.000	95.800

3.8.7 Nine Mile Occupational Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	1069.670	238.000	95.000
2	Mean	1071.289	239.000	95.400
3	Mean	1073.621	240.000	95.800

Mullan

3.9.1 Mullan Residential Surface Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	18.628	476.000	95.100
2	Mean	18.631	477.000	95.300
3	Mean	18.647	478.000	95.500
4	Mean	18.707	479.000	95.700
5	Mean	18.875	480.000	95.900

3.9.1 Mullan Residential Surface Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	31.669	476.000	95.100
2	Mean	31.751	477.000	95.300
3	Mean	31.756	478.000	95.500
4	Mean	31.795	479.000	95.700
5	Mean	31.836	480.000	95.900

3.9.1 Mullan Residential Surface Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	5.035	476.000	95.100
2	Mean	5.037	477.000	95.300
3	Mean	5.040	478.000	95.500
4	Mean	5.055	479.000	95.700
5	Mean	5.069	480.000	95.900

3.9.1 Mullan Residential Surface Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	27360.938	476.000	95.100
2	Mean	27364.777	477.000	95.300
3	Mean	27403.846	478.000	95.500
4	Mean	27410.178	479.000	95.700
5	Mean	27518.468	480.000	95.900

3.9.1 Mullan Residential Surface Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1993.529	476.000	95.100
2	Mean	1998.151	477.000	95.300
3	Mean	1999.564	478.000	95.500
4	Mean	2001.532	479.000	95.700
5	Mean	2003.078	480.000	95.900

3.9.1 Mullan Residential Surface Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	948.373	476.000	95.100
2	Mean	950.786	477.000	95.300
3	Mean	951.881	478.000	95.500
4	Mean	951.959	479.000	95.700
5	Mean	953.013	480.000	95.900

3.9.4 Mullan Neighborhood Recreational Sediment (South Fork CDAR) – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	4.458	476.000	95.100
2	Mean	4.467	477.000	95.300
3	Mean	4.518	478.000	95.500
4	Mean	4.533	479.000	95.700
5	Mean	4.554	480.000	95.900

3.9.4 Mullan Neighborhood Recreational Sediment (South Fork CDAR) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	15.889	476.000	95.100
2	Mean	15.906	477.000	95.300
3	Mean	15.914	478.000	95.500
4	Mean	15.986	479.000	95.700
5	Mean	16.051	480.000	95.900

3.9.4 Mullan Neighborhood Recreational Sediment (South Fork CDAR) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	19.831	476.000	95.100
2	Mean	19.835	477.000	95.300
3	Mean	20.093	478.000	95.500
4	Mean	20.409	479.000	95.700
5	Mean	20.440	480.000	95.900

3.9.4 Mullan Neighborhood Recreational Sediment (South Fork CDAR) – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	47300.000	476.000	95.100
2	Mean	47471.429	477.000	95.300
3	Mean	47754.286	478.000	95.500
4	Mean	47785.714	479.000	95.700
5	Mean	48185.714	480.000	95.900

3.9.4 Mullan Neighborhood Recreational Sediment (South Fork CDAR) – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	5232.929	476.000	95.100
2	Mean	5241.429	477.000	95.300
3	Mean	5254.286	478.000	95.500
4	Mean	5277.786	479.000	95.700
5	Mean	5281.929	480.000	95.900

3.9.4 Mullan Neighborhood Recreational Sediment (South Fork CDAR) – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	4017.286	476.000	95.100
2	Mean	4040.000	477.000	95.300
3	Mean	4057.286	478.000	95.500
4	Mean	4075.714	479.000	95.700
5	Mean	4096.071	480.000	95.900

3.9.5 Mullan Neighborhood Recreational Surface Water (South Fork CDAR) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	0.597	238.000	95.000
2	Mean	0.599	239.000	95.400
3	Mean	0.601	240.000	95.800

3.9.5 Mullan Neighborhood Recreational Surface Water (South Fork CDAR) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	1.244	238.000	95.000
2	Mean	1.249	239.000	95.400
3	Mean	1.258	240.000	95.800

3.9.5 Mullan Neighborhood Recreational Surface Water (South Fork CDAR) – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	18.956	238.000	95.000
2	Mean	18.974	239.000	95.400
3	Mean	19.021	240.000	95.800

3.9.5 Mullan Neighborhood Recreational Surface Water (South Fork CDAR) – Mercury

	STATISTC$	HG	RHG	PCTILE
1	Mean	0.086	238.000	95.000
2	Mean	0.087	239.000	95.400
3	Mean	0.087	240.000	95.800

3.9.6 Mullan Occupational Soil – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	18.677	238.000	95.000
2	Mean	18.741	239.000	95.400
3	Mean	18.885	240.000	95.800

3.9.6 Mullan Occupational Soil – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	31.663	238.000	95.000
2	Mean	31.751	239.000	95.400
3	Mean	31.756	240.000	95.800

3.9.6 Mullan Occupational Soil – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	5.035	238.000	95.000
2	Mean	5.037	239.000	95.400
3	Mean	5.069	240.000	95.800

3.9.6 Mullan Occupational Soil – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	26912.207	238.000	95.000
2	Mean	27086.396	239.000	95.400
3	Mean	27093.423	240.000	95.800

3.9.6 Mullan Occupational Soil – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	1941.464	238.000	95.000
2	Mean	1941.473	239.000	95.400
3	Mean	1944.622	240.000	95.800

3.9.6 Mullan Occupational Soil – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	953.013	238.000	95.000
2	Mean	963.564	239.000	95.400
3	Mean	969.786	240.000	95.800

Blackwell Island

3.10.1 Blackwell Island Public Recreational Soil/Sediment (Spokane River) – Antimony

	STATISTC$	SB	RSB	PCTILE
1	Mean	3.195	476.000	95.100
2	Mean	3.195	477.000	95.300
3	Mean	3.205	478.500	95.600
4	Mean	3.205	478.500	95.600
5	Mean	3.219	480.000	95.900

3.10.1 Blackwell Island Public Recreational Soil/Sediment (Spokane River) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	50.052	476.000	95.100
2	Mean	50.143	477.000	95.300
3	Mean	50.224	478.000	95.500
4	Mean	50.367	479.000	95.700
5	Mean	50.576	480.000	95.900

3.10.1 Blackwell Island Public Recreational Soil/Sediment (Spokane River) –Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	13.314	476.000	95.100
2	Mean	13.386	477.000	95.300
3	Mean	13.395	478.000	95.500
4	Mean	13.395	479.000	95.700
5	Mean	13.405	480.000	95.900

3.10.1 Blackwell Island Public Recreational Soil/Sediment (Spokane River) – Iron

	STATISTC$	FE	RFE	PCTILE
1	Mean	48880.952	476.000	95.100
2	Mean	48966.667	477.000	95.300
3	Mean	49119.048	478.000	95.500
4	Mean	49242.857	479.000	95.700
5	Mean	49495.238	480.000	95.900

3.10.1 Blackwell Island Public Recreational Soil/Sediment (Spokane River) – Manganese

	STATISTC$	MN	RMN	PCTILE
1	Mean	3666.143	476.000	95.100
2	Mean	3666.143	477.000	95.300
3	Mean	3667.143	478.000	95.500
4	Mean	3670.000	479.000	95.700
5	Mean	3687.143	480.000	95.900

3.10.1 Blackwell Island Public Recreational Soil/Sediment (Spokane River) – Zinc

	STATISTC$	ZN	RZN	PCTILE
1	Mean	1800.381	476.000	95.100
2	Mean	1807.238	477.000	95.300
3	Mean	1808.286	478.000	95.500
4	Mean	1815.000	479.000	95.700
5	Mean	1818.667	480.000	95.900

All Geographical Areas

3.11.1 All Areas Residential Plant Tissue (Homegrown Vegetables) – Arsenic

	STATISTC$	AS	RAS	PCTILE
1	Mean	0.025	476.000	95.100
2	Mean	0.025	477.000	95.300
3	Mean	0.025	478.000	95.500
4	Mean	0.025	479.000	95.700
5	Mean	0.025	480.000	95.900

3.11.1 All Areas Residential Plant Tissue (Homegrown Vegetables) – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	0.319	476.000	95.100
2	Mean	0.320	477.000	95.300
3	Mean	0.323	478.000	95.500
4	Mean	0.325	479.000	95.700
5	Mean	0.326	480.000	95.900

3.11.2 All Areas Recreational and Subsistence Animal Tissue (Fish from Lower CDAR) – Bullhead – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	0.008	238.500	95.200
2	Mean	0.008	238.500	95.200
3	Mean	0.008	240.000	95.800

3.11.2 All Areas Recreational and Subsistence Animal Tissue (Fish from Lower CDAR) – Bull – Mercury

	STATISTC$	HG	RHG	PCTILE
1	Mean	0.052	238.000	95.000
2	Mean	0.052	239.000	95.400
3	Mean	0.052	240.000	95.800

3.11.2 All Areas Recreational and Subsistence Animal Tissue (Fish from Lower CDAR) – NP – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	0.006	238.000	95.000
2	Mean	0.006	239.000	95.400
3	Mean	0.007	240.000	95.800

3.11.2 All Areas Recreational and Subsistence Animal Tissue (Fish from Lower CDAR) – NP – Mercury

	STATISTC$	HG	RHG	PCTILE
1	Mean	0.133	238.000	95.000
2	Mean	0.133	239.000	95.400
3	Mean	0.134	240.000	95.800

3.11.2 All Areas Recreational and Subsistence Animal Tissue (Fish from Lower CDAR) – Perch – Cadmium

	STATISTC$	CD	RCD	PCTILE
1	Mean	0.037	238.000	95.000
2	Mean	0.037	239.000	95.400
3	Mean	0.037	240.000	95.800

3.11.2 All Areas Recreational and Subsistence Animal Tissue (Fish from Lower CDAR) – Perch – Mercury

	STATISTC$	HG	RHG	PCTILE
1	Mean	0.089	238.000	95.000
2	Mean	0.089	239.000	95.400
3	Mean	0.089	240.000	95.800

APPENDIX H

Toxicity Profiles for Chemicals of Potential Concern

Appendix H

Toxicity Profiles for Chemicals of Potential Concern

This appendix includes the toxicity profiles for the metals of concern. The profiles summarize the toxic effects of the chemicals of concern along with the toxicity criteria used in the risk assessment for assessing noncarcinogenic and carcinogenic effects.

Antimony

Adverse Health Effects of Antimony (Sb; CAS# 7440-36-0)

The comprehensive review of antimony toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1992 forms the primary basis for this profile. Specific discussion about toxicity values used to characterize health risks potentially associated with exposure to antimony is based on information provided in the U.S. Environmental Protection Agency [EPA] Integrated Risk Information System [IRIS].

Antimony compounds have been shown to be toxic to human populations from occupational inhalation exposure and accidental ingestion, producing effects both in the respiratory and gastrointestinal tracts. Certain antimony compounds may be toxic to the heart. Oral exposure to antimony has been associated with changes in blood chemistry in laboratory animals. There is inconclusive evidence of a relationship between inhalation of antimony trioxide and excess risk of lung cancer (Hathaway et al., 1991).

Elemental antimony is a silvery-white soft metal. It is found at low concentrations in soil, generally 1 part per million (ppm) or lower. The geochemical properties of antimony are similar to those of arsenic (antimony has +3 and +5 valence states). As with arsenic, antimony may be associated with nonferrous ore deposits, and therefore can be a pollutant in industrial environments (Kabata-Pendias and Pendias, 1992). Antimony is a constituent in alloys with nonferrous metals such as tin, lead, and copper. Antimony sulfides are used in the production of rubber and pyrotechnics. Antimony chlorides are used as coloring agents and catalysts. Antimony fluorides are used in organic synthesis and pottery manufacture (Hathaway et. al., 1991).

Pharmacokinetics

Absorption

Antimony is poorly absorbed from the gastrointestinal tract. Gastrointestinal absorption of more soluble forms (antimony tartrate and antimony chloride) in laboratory animals ranges from 2 to 7 percent. For the inhalation route, quantitative information about absorption rate is not available. Although elevated blood and urinary concentrations have been reported in workers exposed to antimony it is uncertain whether absorption was by the inhalation route or by ingestion of inhaled antimony that was cleared from the respiratory tract. Respiratory absorption has, however, been shown to be a function of particle size (ATSDR, 1992). Certain inhaled antimony compounds appear to be retained in the lung for long periods (NLM/HSDB, 2000). No studies were located regarding dermal absorption of antimony in humans, although studies with rabbits suggest that at least some forms of antimony can be absorbed through the skin (ATSDR, 1992).

The issue of bioavailability of antimony is especially important at mining, milling, and smelting sites. This is because the antimony at these sites often exists, at least in part, as a poorly soluble sulfide, and may also occur in particles of inert or insoluble material. These factors all may tend to reduce the bioavailability of antimony.

Distribution and Excretion

The major sites of accumulation for antimony following ingestion are the liver, kidney, bone, skin, and hair. The distribution of antimony may depend upon its valence state in the body. Inhaled trivalent antimony is mainly bound to erythrocytes, while inhaled pentavalent antimony is found in the plasma. Uptake in bone is greater for pentavalent antimony than for trivalent antimony. Absorbed antimony is excreted both through the feces and the urine, however measurements of fecal excretion of absorbed antimony may be complicated by poor gastrointestinal absorption of ingested antimony. Studies in laboratory animals involving the intraperitoneal route of exposure indicate that the valence state of antimony may influence the route of excretion, with pentavalent antimony excreted principally in the urine, and trivalent antimony excreted through the feces. In laboratory animals, excretion of antimony is a two-phase process, consisting of a fast phase where 90 percent of the initial body burden is excreted within 24 hours (the fast phase), and a slow phase with a half-life of 16 days (ATSDR, 1992).

Qualitative Description of Health Effects

Acute Toxicity

Acute ingestion exposure to antimony has produced gastrointestinal effects both in humans and in laboratory animals, with signs and symptoms including pains in the stomach, vomiting and diarrhea. Other than one study noting inflammation in the lungs of rabbits exposed by inhalation to antimony trisulfide, there is no information available regarding toxic effects from acute inhalation exposure to antimony, as a dust or particulate (ATSDR, 1992).

Stibine (antimony hydride) is a colorless gas produced when acid solutions of antimony compounds come into contact with reducing agents. It is a pulmonary irritant and hemolytic agent following short-term exposure in laboratory animals, and it is likely that the same effects would be observed in humans (Hathaway et al., 1991).

Chronic and Subchronic Toxicity

Mild hematological alterations (not specified) and cloudy swelling in the liver were observed in rats administered antimony trioxide orally at 418 mg/kg-day for 24 weeks. Increased serum cholesterol and decreased nonfasting serum glucose levels were observed in rats exposed for a lifetime to 5 ppm potassium antimony tartrate in drinking water. Occupational exposure to high concentrations of antimony trioxide or pentoxide dust (8.87 mg/m^3 as antimony) has produced respiratory irritation, including pneumoconiosis, bronchitis, and alteration in pulmonary function (ATSDR, 1992). Symptoms observed in smelter workers exposed to an average concentrations of antimony of 10 mg/m^3 (highest exposures were 70 mg/m^3) included dermatitis and rhinitis. Less frequent effects included irritation, sore throat, headache, pain or tightness in the chest, metallic taste, nausea, vomiting, diarrhea, and weight loss (Hathaway et al., 1991). Respiratory effects also have been observed in laboratory animals following long-term inhalation of high levels of antimony, including progression from pneumoconiosis to proliferation of alveolar macrophages, interstitial inflammation, and fibrosis (ATSDR, 1992).

Six sudden deaths, two deaths due to chronic heart disease, elevated blood pressure, and abnormal EKG readings were reported in 125 abrasive wheel workers exposed to 2 to

3 mg/m^3 antimony in air as antimony trisulfide for up to 2 years (ATSDR, 1992; Hathaway et al., 1991). Inhalation of antimony trisulfide has produced myocardial effects (degenerative changes in the myocardium and altered EKG readings) in some animal studies but not others (ATSDR, 1992).

Reproductive or Developmental Toxicity

An increased incidence of spontaneous abortions and altered menstrual cycles, compared to a control group, has been reported in a group of women working in an antimony metallurgical plant (Belyaeva, 1967, as cited in ATSDR, 1992). However, the levels of antimony exposure and presence of other compounds was not reported. Additionally, information was not presented demonstrating that the control group population was comparable to the study group in terms of factors other than antimony exposure (ATSDR, 1992). There are no studies evaluating developmental or reproductive toxicity of antimony in humans from ingestion exposure (ATSDR, 1992).

Reproductive effects including failure to conceive and decreased number of offspring were reported in rats exposed to 209 mg/m^3 antimony trioxide in air prior to conception and throughout gestation (Belyaeva, 1967, as cited in ASTDR, 1992). No gross abnormalities were observed in the offspring of rats exposed to antimony trichloride in drinking water.

Mutagenicity and Genotoxicity

There is limited evidence of genotoxicity of antimony in *in vitro* systems, but none in *in vivo* systems. Types of effects reported include gene mutations, chromosomal aberrations, and chromosomal breakage in mammalian cell systems (ATSDR, 1992).

Carcinogenicity

There is no conclusive evidence regarding carcinogenicity of antimony compounds in humans. Antimony trioxide has been identified by the American Conference of Governmental Industrial Hygienists (ACGIH) as a category A2, suspected human carcinogen, based on limited evidence in human populations and sufficient evidence in laboratory animals (Hathaway et al., 1991; ACGIH, 1999). However, inhalation exposure to 8.87 mg/m^3 antimony (as either trioxide or pentoxide dusts) did not affect the incidence of cancer in workers exposed from 9 to 31 years (ATSDR, 1992). The International Agency for Research on Cancer (IARC) has classified antimony trioxide as "possibly carcinogenic" in humans.

The U.S. Environmental Protection Agency (USEPA) has not evaluated the carcinogenicity of antimony (USEPA [IRIS], 2000). There is conflicting evidence regarding the carcinogenicity of antimony in laboratory animals. While increased incidence of lung tumors has been observed in some studies where rats were exposed to antimony trioxide or antimony trisulfide, other studies showed no evidence of excess tumors. The levels of antimony trioxide exposure in which lung tumors were observed in rats were 4 and 36 mg/m^3 (Groth et al., 1986; Watt, 1980; Wong et al., 1979, cited in ATSDR, 1992). Antimony trisulfide produced lung tumors in rats when evaluated at a concentration of 17.5 mg/m^3 (Groth et al., 1986; Wong et al., 1979, cited in ATSDR, 1992). However, an increased incidence of lung tumors was not observed in rats exposed to 4 mg/m^3 antimony trioxide (Biodynamics, 1990, as cited in ATSDR, 1992) or in pigs exposed to 4.2 mg/m^3 as antimony trioxide (Watt, 1983, as cited in ATSDR, 1992). It is not known why there are

inconsistencies between these animal studies, though differences in pulmonary retention and clearance related to particle size of the administered antimony compounds may explain the different results. Also, carcinogenicity in the lung may be related to other pulmonary injuries, such as proliferation of alveolar macrophages, inflammation and fibrosis. Differences in the mechanisms of deposition and clearance of particulates between rats and humans may also explain the apparent evidence of carcinogenicity in laboratory animals with the lack of evidence in humans (ATSDR, 1992).

Exposure Route Considerations

Oral

Antimony is poorly absorbed from the gastrointestinal tract. Acute ingestion exposure is irritating to the gastrointestinal tract. Long-term ingestion exposure in laboratory animals may produce abnormal changes in the blood (increased serum cholesterol and decreased nonfasting serum glucose levels).

Inhalation

Inhalation exposure in workers may be associated with effects to the cardiovascular system (elevated blood pressure), and pneumoconiosis and altered pulmonary function. There is no conclusive evidence that antimony is carcinogenic in humans by inhalation, because evidence from studies in human populations is very limited, and carcinogenicity studies in laboratory animals provide conflicting results.

Dermal

No studies were located characterizing toxicity from dermal exposure to antimony compounds.

Sensitive Populations

No studies were located describing particular sensitivities to antimony exposure. Based on the available information, it is possible to infer that individuals with pre-existing pulmonary disease would be more sensitive to inhalation of antimony under workplace conditions.

Toxicity Factors Derived for Risk Assessment

The oral reference dose (RfD) for antimony is based on decreases in nonfasting blood glucose levels, altered cholesterol levels, and decreased longevity in rats administered 5 ppm antimony in drinking water for life. Since there was only one level of antimony administered, a no observed adverse effect level (NOAEL) was not established in the study. The calculated lowest observed adverse effect level (LOAEL) was 0.35 mg/kg-day. An uncertainty factor of 1000 (10 for interspecies conversion, 10 to protect sensitive individuals, and 10 to convert the LOAEL to a NOAEL) was applied to the LOAEL to obtain an oral RfD of 0.0004 mg/kg-day (USEPA [IRIS], 2000). USEPA's confidence in the oral RfD is reported to be low.

An inhalation reference concentration (RfC) has been developed specifically for antimony trioxide. The RfC is based on the occurrence of chronic interstitial inflammation in the lungs and reduced clearance of inhaled particulates, in rats exposed by inhalation for one year. These data were used in a Benchmark Concentration (BMC) analysis (i.e. pulmonary effects

were grouped as quantal responses, and dose-response was modeled using statistical models). The lower 95 percent confidence limit for the 10 percent relative increase in the probability of pulmonary response was determined to be at 0.87 mg/m^3, which was transformed to a human equivalent concentration of 0.074 mg/m^3. An uncertainty factor of 300 was applied to this NOAEL as follows: an uncertainty factor of 10 is used for the protection of sensitive human subpopulations; an uncertainty factor of 3 is used for interspecies extrapolation; an uncertainty factor of 3 is applied for data base inadequacies (principally, the lack of reproductive and developmental bioassays); and an additional threefold uncertainty factor was applied to account for a less-than-lifetime exposure duration. These factors were rounded to obtain an uncertainty factor of 300. The resulting RfC (0.074 ÷ 300) is 0.0002 mg/m^3 (USEPA [IRIS], 2000).

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Arsenic

Adverse Health Effects of Arsenic (As; CAS# 7440-38-2)

The comprehensive review of arsenic toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1998 forms the primary basis for this profile. Specific discussion about toxicity values used to characterize health risks potentially associated with exposure to arsenic is based on information provided in the U.S. Environmental Protection Agency [USEPA] Integrated Risk Information System [IRIS]. Additional discussions of the current basis (i.e. skin cancer) for characterizing cancer risks were drawn from the reports prepared by the National Research Council (NRC, 1999) and USEPA Risk Assessment Forum (USEPA, 1988). Reanalysis of epidemiological data on arsenic exposures has indicated an increased incidence of internal cancers (liver, kidney and bladder) in addition to skin cancer. The papers discussing these more recent findings have been incorporated into this profile.

Key issues associated with assessment of risks from exposure to arsenic at Superfund sites have been addressed in this profile. These issues include bioavailability in certain media (i.e., soil), chemical forms in which arsenic occurs in the environment (inorganic versus organic arsenic), toxicity of different valences and forms of arsenic, and the basis for toxicity factors (the cancer slope factor and the reference dose).

Arsenic has been shown to be toxic to human populations in areas of the world where it is present in naturally elevated levels in groundwater, and in certain occupations such as copper smelting and chemical plant workers. There is good evidence that arsenic is carcinogenic in humans by both oral and inhalation routes, while studies have shown that most laboratory animals are substantially less susceptible to arsenic toxicity than humans (ATSDR, 1998). Therefore, this profile focuses on toxicity information obtained from observations of human populations, and considers animal toxicity data to the extent that data in human populations are unavailable.

Elemental arsenic is a silver-gray metal, however it occurs in rock or soil most often as the sulfide in a variety of complex minerals containing copper, lead, iron, nickel, cobalt, and other metals. Arsenic occurs in the environment principally in the +3 oxidation state (arsenite) or the +5 oxidation state (arsenate) (ATSDR, 1998). When ores containing copper or lead are smelted, arsenic can be released into the air as a fine dust. Arsenic trioxide is the most commercially important form of arsenic, and is produced primarily from flue dust that is generated at copper and lead smelters. Arsenic trioxide is no longer produced in the United States. The principal use of arsenic (as arsenic trioxide) is in wood preservatives with a smaller proportion used in the production of agricultural chemicals such as insecticides, herbicides, algicides, and growth stimulants for plants and animals. The agricultural use of many arsenical pesticides has been phased out in the United States due to concerns about human health risks during production or use. Smaller amounts of arsenic are also used in the production of glass and nonferrous alloys, and in the semiconductor industry.

Pharmacokinetics

Absorption

Both arsenate and arsenite are well absorbed by both the oral and inhalation routes. Absorption by the dermal route is generally quite low. Overall absorption by the inhalation route following particle deposition in the respiratory tract is estimated to be 30 to 60 percent of inhaled arsenic. Studies with laboratory animals and human volunteers indicate that oral absorption of arsenite or arsenate is relatively high (50 to 95percent of ingested arsenic, depending upon chemical form and species). Oral absorption of less soluble arsenic species, such as sulfides or lead arsenate is lower, around 20 to 30 percent of ingested arsenic (ATSDR, 1998). Studies in rabbits suggest that oral absorption of arsenic from ingestion of contaminated soils are reduced compared to arsenic in solution, although the form of arsenic in the soil, as well as the type of soil, can be assumed to influence the degree of absorption (NRC, 1999). Approximately 80 percent of arsenic in soil (primarily as the less soluble sulfide form) was excreted in the feces, compared with 50 percent excreted when administered as a gavage dose of sodium arsenate (Freeman et al., 1993, as cited in ATSDR, 1998). Arsenic in dust or soil was less 3- to 9-fold less bioavailable than arsenic in solution, depending upon whether bioavailability was based on blood arsenic or urinary excretion of arsenic (ATSDR, 1998).

Good correlations between arsenic in soil and urinary arsenic levels in human receptors were reported at a site where site-specific bioavailability adjustment factors (0.18 to 0.25) were used to account for lower bioavailability of arsenic in soil (Walker and Griffin et al., 1998). In the absence of site-specific data, USEPA Region10 recommends using a default relative bioavailability factor of 0.6 to account for the decreased absorption of ingested arsenic in soil relative to the absorption of soluble arsenic ingested in water (USEPA, 1997; personal communication, Roseanne Lorenzana, 1998, U.S. EPA Region 10).

Percutaneous absorption of arsenic acid mixed with soil was estimated to be 4.5percent after 24hours, as measured in rhesus monkeys and 3 percent absorption based on human cadaver data (Wester et al., 1993, as cited in ATSDR, 1998). Based on this study, USEPA has selected 3 percent as the default dermal absorption factor for arsenic.

Distribution

Limited information is available on distribution of arsenic in the body. However the available studies indicate that arsenic is distributed to all tissues of the body following absorption, indicating there is little tendency to accumulate preferentially in any of the internal organs (ATSDR, 1998).

Metabolism and Excretion

The metabolism of arsenic involves oxidation/reduction reactions interconverting arsenate and arsenite and methylation which converts arsenite to monomethyl arsonic acid (MMA) and dimethyl arsinic acid (DMA, or cacodylic acid). Methylation followed by urinary excretion represents a detoxification pathway for arsenic. Combined excretion of methylated and inorganic arsenic accounts for 75 percent of the absorbed dose (ATSDR, 1998). There is some evidence of a maximum level of arsenic that can be detoxified through this mechanism (EPA, 1988). The main site of methylation is the liver where the process is mediated by enzymes using S-adenosylmethionine as a methyl donor. Severe dietary restrictions (dietary

protein) reportedly can reduce methylating capacity (ATSDR, 1998; EPA, 1988). Very little of absorbed arsenic is excreted in the feces (ATSDR, 1998).

Qualitative Description of Health Effects

Acute Toxicity

Arsenic is a potent toxicant, with the minimum oral lethal dose in humans ranging from 1 to 3 mg/kg. At high levels of exposure, lethality from arsenic ingestion is usually attributed to cardiopulmonary collapse. Lethal doses in animals are higher than in humans, consistent with the trend that animals are less sensitive to arsenic than humans. Nausea, vomiting and diarrhea are common symptoms in humans following acute high-dose ingestion of inorganic arsenic compounds. The effects are likely due to direct irritation of gastric mucosa. Signs of peripheral neuropathy have been experienced in individuals who have ingested inorganic arsenic. The neuropathy is detected as numbness in the hands and feet, progressing to a painful "pins and needles" sensation. Inhalation of dusts containing inorganic arsenic (principally arsenic trioxide dusts) are irritating to the upper respiratory tract (ATSDR, 1998).

Subchronic and Chronic Toxicity

The most distinguishing adverse effects associated with chronic ingestion of arsenic include skin changes and damage to the vascular system. Severe cases of chronic exposure result in a disorder known as "blackfoot disease", which is a progressive loss of circulation in the extremities ultimately leading to gangrene. Blackfoot disease has been reported in one area of Taiwan with elevated levels of arsenic in drinking water supplies (ATSDR, 1998). The "blackfoot disease endemic area" in Taiwan had arsenic concentrations in well water ranging from 0.01 to 1.82 mg/L (Bates et al., 1992). The localized nature of blackfoot disease may be due to the presence of other substances consumed in drinking water (fluorescent substances) that are possible confounders or have caused synergistic effects (ATSDR, 1998; USEPA, 2000). While blackfoot disease has not been reported elsewhere in the world, other less severe signs of vascular injury (such Reynaud's disease) have been reported in other areas). Hyperkeratosis, hyperpigmentation and skin cancer are also distinguishing adverse effects of arsenic exposure, and have been observed in populations in Taiwan, Mexico, India and Chile who consumed drinking water with high levels of arsenic (greater than 0.2 mg/L) (Smith et al., 1992). Hyperkeratosis and hyperpigmentation appear to be the earliest observable signs of chronic exposure. Epidemiological studies identify a lowest observed adverse effects level (LOAEL) of 0.01 to 0.02 mg/kg-day for skin lesions and a no observed adverse effect level (NOAEL) of 0.0004 to 0.0009 mg/kg-day. Inhalation exposure to arsenic concentrations from 0.1 to 1 mg/m^3 also reportedly may lead to hyperkeratosis and hyperpigmentation (ATSDR, 1998).

Reproductive or Developmental Toxicity

Evidence of reproductive or developmental toxicity in humans is limited and inconclusive. The available studies in humans do not provide conclusive evidence that ingestion of arsenic, at the level usually encountered in drinking water, causes developmental toxicity. Studies in laboratory animals suggest that arsenic exhibits developmental toxicity (reduced birth weight, fetal malformations, and increased fetal mortality) at high levels of exposure (20 to

70 mg/kg-day, orally). The data suggest that inorganic arsenic does not pose a significant risk of developmental toxicity except at maternally toxic levels (ATSDR, 1998).

Genotoxicity

Inorganic arsenicals appear to be inactive or weak mutagens, but are capable of producing chromosomal effects (chromosomal aberrations and increased sister chromatid exchange frequency) in test systems. Studies with small human populations have detected increased incidence of chromosomal aberrations in peripheral lymphocytes after inhalation and oral exposure. Arsenic and its metabolites do not appear to directly interact with DNA (ATSDR, 1998).

Carcinogenicity

USEPA has given arsenic a carcinogenicity weight-of-evidence classification of A; human carcinogen. This is based on sufficient evidence in humans, including increased lung cancer mortality observed in human populations exposed through inhalation, increased mortality from internal organ cancers (liver, kidney, lung, and bladder), and an increased incidence of skin cancer observed in populations consuming drinking water high in inorganic arsenic (USEPA [IRIS], 2000). The International Agency for Research on Cancer (IARC) has classified arsenic compounds in Group 1, carcinogenic to humans.

There is clear evidence that oral and inhalation exposure to inorganic arsenic may increase the risk of cancer in humans. Studies of smelter workers and pesticide manufacturing workers populations have all found an association between occupational arsenic exposure and lung cancer mortality. One study of a population residing near a pesticide manufacturing plant revealed that these residents were also at an excess risk of lung cancer. Observations of arsenical pesticide applicators also suggest an association between arsenic exposure and lung cancer (ATSDR, 1998; NRC, 1999; USEPA [IRIS], 2000).

Several epidemiological studies have demonstrated an association between cancer and ingestion of elevated concentrations of arsenic in drinking water. Studies in Taiwan (in the blackfoot disease endemic area) stratified the exposed population into groups based on drinking water exposure to <0.3 mg/L, 0.3 to 0.6 mg/L, and >0.6 mg/L. EPA estimated average drinking water exposure concentrations of 0.17, 0.47, and 0.8 mg/L for purposes of characterizing exposure/incidence relationships. These concentrations corresponded to approximately 0.6 to 2.8 mg/day arsenic intake, using assumptions for a Taiwanese population (3.5-L/day drinking water intake and a 55-kg body weight). Significantly elevated incidences of skin cancer was observed in these exposed populations. Factors influencing the applicability of the Taiwanese studies for assessing arsenic cancer risks in other exposure settings include uncertainties in estimates of exposure to arsenic and presence of specific environmental factors, such as a low protein diet (USEPA, 1988; ATSDR, 1998). In addition to skin cancer, there are several reports indicating that ingestion of arsenic in drinking water increases the risk of cancer in the liver, bladder and kidney in populations from Taiwan, Argentina, and Chile (Bates et al., 1992; ATSDR, 1998, NRC, 1999). Epidemiological studies of drinking water exposure to arsenic in the U.S. have not shown an increased incidence of cancer, with concentrations ranging from 0.05 to 0.2 mg/L. However, the significance of these findings has been considered limited because of the study designs, and small exposed populations that were relatively mobile and that had access to alternate water supplies (USEPA, 1988; Bates et al., 1992).

Exposure Route Considerations

Ingestion

Food and drinking water are the largest sources of arsenic exposure. Total dietary intake of arsenic in the U.S. averages 0.05 to 0.06 mg/day, using residue data combined with food consumption survey data. Average intake of inorganic arsenic in the U.S. was about 0.01 mg/day. A portion of the arsenic ingested in the diet is in the form of low-toxicity organic arsenic. Meat and grains a have relatively lower fraction of organic arsenic, whereas fish, vegetables and fruits have relatively higher fractions of organic arsenic. Organic arsenical compounds are found to accumulate in fish and shellfish. These derivatives (mainly arsenobetaine and arsenocholine, also referred to as "fish arsenic") have been studied by several researchers and have been found to be essentially nontoxic. Estimates of arsenic intake in drinking water in the U.S. are around 0.005 mg/day, but could be greater (0.01 to 0.1 mg/day) in areas with elevated arsenic concentrations in groundwater. Arsenic intake from drinking water is assumed to be entirely inorganic. Naturally occurring arsenic levels in groundwater in the U.S. average around 1 to 2 parts per-billion (ppb), except for some western states with geological features that are naturally elevated in arsenic. Concentrations of naturally occurring arsenic in groundwater in these areas range from 5 to over 500 ppb. In the U.S., over 350,000 people may drink water containing arsenic concentrations higher than the Maximum Contaminant Level (MCL) of 50 ppb (ATSDR, 1998; USEPA, 1988; Smith et al., 1992). USEPA has proposed a new MCL for arsenic of 5 µg/L and estimated over 20 million people may be drinking water containing arsenic at concentrations above the proposed level (Federal Register, 2000). The USGS estimates that just over 13 percent of small systems use water with arsenic concentrations greater than 5 µg/L (USGS, 2000).

Studies in laboratory animals suggest that low levels of dietary arsenic may be beneficial or essential. Laboratory animals on arsenic-free diets show decreased growth, decreased weight gain and decreased reproductive success (NRC, 1999). However, no specific biochemical mechanism is known through which arsenic could exert a beneficial effect. If arsenic is beneficial to humans, then the daily requirement probably lies between 0.01 and 0.05 mg/day, which is within the level of total arsenic provided by a normal diet (food and water) (ATSDR, 1998; USEPA, 1988).

Arsenic has typically been associated with adverse effects in human populations when exposed to levels in drinking water exceeding 300 ppb over a long period of time (ATSDR, 1998). Skin lesions (hyperkeratosis and hyperpigmentation, skin cancer, cancer of internal organs, and [in Taiwan] blackfoot disease) are characteristics signs of chronic ingestion exposure to elevated levels of arsenic.

Inhalation

Arsenic concentrations in ambient air in remote areas are within <1 to 3 ng/m^3, and 20 to 30 ng/m^3 in urban areas. Large cities have higher concentrations in air due to emissions from coal-fired power plants. Occupational exposure (principally smelter workers) has been associated with an increased incidence of lung cancer. Occupational exposures associated with cancer effects range from 50 to 300 µg/m^3 in air (ATSDR, 1998).

Dermal

Occupational exposure to arsenic dusts, or arsenical pesticide solutions have been reported to produce dermatitis (ATSDR, 1998)

Sensitive Populations and Indicators of Exposure

Genetic factors and age may distinguish human subpopulations that are sensitive to arsenic exposure, especially in their ability for metabolism. Individuals with impaired capacity to methylate and detoxify arsenic may be at greater risk of adverse effects from arsenic exposure. Therefore, individuals with dietary deficiencies or impaired liver function may be more sensitive to adverse effects from arsenic exposure (ATSDR, 1998). One study in Finland suggests that children have lower arsenic-methylating ability than adults (NRC, 1999).

Effective biomarkers of arsenic exposure include levels measured in the urine, hair or fingernails. Arsenic affects the functioning of several enzymes that may prove useful as biomarkers of potential exposure. Characteristic skin changes (hyperkeratosis and hyperpigmentation) observed through dermatological examination might also provide indicators of exposure (albeit not early).

Toxicity Factors Derived for Risk Assessment

The oral reference dose (RfD) is based on the occurrence of hyperpigmentation and hyperkeratosis, and vascular complications observed in the Taiwanese population ingesting elevated levels of arsenic in drinking water. The NOAEL was calculated to be 0.0008 mg/kg-day. An uncertainty factor of 3 is applied to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals. The oral RfD for arsenic is 0.0003 mg/kg-day. According to USEPA, strong scientific arguments can be made for various values within a factor of 2 or 3 of the currently recommended RfD value, i.e., 0.0001 to 0.0008 mg/kg/day. An inhalation RfD or reference concentration (RfC) has not been estimated for arsenic (USEPA [IRIS], 2000).

The oral unit risk factor for estimating excess lifetime cancer risks is based on the incidence of skin cancer observed in Taiwanese population ingesting elevated levels of arsenic in drinking water. Doses were converted to equivalent doses for U.S. males and females based on differences in body weights and differences in water consumption. It was assumed that skin cancer risk in the U.S. population would be similar to the Taiwanese population. The maximum likelihood estimate (MLE) of skin cancer risk for a 70 kg person drinking 2 L of water per day ranged from 1×10^{-3} to 2×10^{-3} for an arsenic intake of 1 μg/kg-day. Expressed as a single value, the cancer unit risk for drinking water is 5×10^{-5} per (μg/L). Details of the assessment are in U.S. EPA (1988) (USEPA [IRIS], 2000). Using the assumptions of 2-L/day drinking water consumption and 70-kg body weight, this unit risk factor converts to an oral slope factor of 1.5 $(mg/kg\text{-}day)^{-1}$. It should be noted that USEPA's assessment is based on prevalence of skin cancer rather than mortality because the types of skin cancer produced by arsenic are not normally fatal.

The inhalation unit risk factor for estimating excess lifetime cancer risks is the based on the incidence of lung cancer observed in two different populations of smelter workers. The resulting unit risk factor is 4.3×10^{-3} per $\mu g/m^3$ (USEPA, 1998). Using the assumptions of 20

m^3/day inhalation rate and 70 kg body weight, this unit risk factor converts to an inhalation slope factor of 15 $(mg/kg\text{-}day)^{-1}$.

USEPA has currently proposed a revision of the MCL for arsenic based primarily on the recent National Academy of Science's National Research Council review of arsenic information (NRC, 1999). The NRC reviewed the current state of science for estimating risks associated with arsenic in drinking water and recommended lowering the MCL from the current interim drinking water standard of 50 µg/L. This recommendation is based on NRC's assessments of the risks of skin, lung, and bladder cancer from drinking water containing inorganic arsenic. The report quantified the risks from bladder cancer and describes potential risks of cardiovascular effects. Based primarily on information in the NRC report, the USEPA has published their proposed new MCL of 5 ug/L (Federal Register, 2000).

In addition to information from the NRC report, the USEPA also considered a recent epidemiological study in Utah (Lewis, et al., 1999) when proposing the new MCL. The Utah study found a significant increase in prostate cancer for males with arsenic in their drinking water ranging from 4 to 620 ug/L with all seven communities studied having median exposure concentrations of less than 200 ug/L. While the Utah population is likely not representative of the United States population in general, the EPA considers this study to provide further weight to concerns about arsenic health effects in drinking water at concentrations below the current MCL. The Utah study is of particular interest because of the relatively low range of arsenic water concentrations in contrast to other epidemiologic studies which generally had average arsenic exposure in the several hundreds ug/L range. In addition, the Utah study suggested that cardiovascular effects can occur at lower levels than those seen in the studies reviewed by the NRC.

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Cadmium

Adverse Health Effects of Cadmium (Cd; CAS# 7440-43-9)

The comprehensive review of cadmium toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1999 forms the primary basis for this profile. Specific discussion about toxicity values used to characterize health risks potentially associated with exposure to cadmium is based on information provided in the U.S. Environmental Protection Agency [EPA] Integrated Risk Information System [IRIS].

Cadmium has been shown to be toxic to human populations from occupational inhalation exposure and accidental ingestion of cadmium-contaminated food. Inhalation of cadmium dust in certain occupational settings may be associated with an increased incidence of lung cancer. Ingestion of elevated levels of cadmium has resulted in toxicity to the kidney and skeletal system, and may be associated with an elevated incidence of hypertension and cardiovascular disease.

Elemental cadmium is a soft, silver-white metal; however, cadmium is not usually found in the environment as a metal. Cadmium is found in the earth's crust at concentrations of about 1 to 2 parts per million (ppm), primarily in association with zinc ores. Cadmium (as cadmium oxide) is obtained mainly as a by-product during the processing of zinc-bearing ores and also from the refining of lead and copper from sulfide ores. Cadmium is used primarily for the production of nickel-cadmium batteries, in metal plating, and for the production of pigments, plastics, synthetics and metallic alloys (ATSDR, 1999).

Pharmacokinetics

Absorption

Cadmium is poorly absorbed from the gastrointestinal tract. Long-term absorption and retention of cadmium is approximately 5 to 6 percent the amount ingested. Absorption of cadmium from food may be lower than absorption from water or solution (i.e., approximately 2.5 percent). The body stores of iron influence cadmium absorption. Individuals with low iron stores exhibit higher absorption of cadmium. Dietary deficiencies in calcium and protein also enhance cadmium absorption (ASTDR, 1999; USEPA [IRIS], 2000, Goyer, 1991). Absorption of inhaled cadmium is approximately 5 to 20 percent. Absorption of cadmium inhaled in cigarette smoke is higher than absorption of cadmium inhaled in aerosols, as measured in laboratory animals. Dermal absorption of cadmium from solution or soil is very limited (ATSDR, 1999).

The issue of bioavailability of cadmium is especially important at mining, milling, and smelting sites. The cadmium at these sites can often exist, at least in part, as a poorly soluble sulfide, and may also occur in particles of inert or insoluble material. These factors can collectively reduce the bioavailability of cadmium.

Distribution, Metabolism and Excretion

Cadmium is widely distributed in the body following either ingestion or inhalation exposure, with much of the body burden found in the liver and kidney. Cadmium ions circulate in plasma bound to sulfhydryl groups in proteins such as albumin and metallothionein. Binding to metallothionein is thought to reduce the toxicity of cadmium. Following ingestion, fecal excretion is high due to poor gastrointestinal absorption. Most cadmium that has been absorbed, however, is excreted very slowly, with fecal and urinary excretion being about equal (ASTDR, 1999).

Qualitative Description of Health Effects

Acute Toxicity

Oral exposure to cadmium in high concentrations causes severe irritation to the gastrointestinal tract. Common symptoms in humans following ingestion of food or beverages containing high concentrations of cadmium include nausea, vomiting, salivation, abdominal pain, cramps, and diarrhea. The emetic dose has been estimated to be approximately 0.07 mg/kg. Acute inhalation exposure to high concentrations of cadmium oxide fume is intensely irritating to the respiratory tract. Signs and symptoms include irritation, coughing dyspnea, tightness in the chest and flu-like symptoms (ASTDR, 1999).

Chronic/Subchronic Toxicity

Longer-term ingestion exposure to cadmium has resulted in adverse effects in the kidney, the skeletal system, cardiovascular system, and the blood. The kidney is considered to be the main target organ of cadmium toxicity following extended oral or inhalation exposure. Elevated incidences of tubular proteinuria have been found in epidemiologic studies of residents of cadmium-polluted areas in Japan and China, and in studies of workers occupationally exposed to cadmium by inhalation. The effects include increased excretion of proteins, amino acids and sugars in the urine (proteinuria, aminoaciduria and glucosuria), and tubular cell degeneration followed by inflammation and fibrosis. Comparison of measured cadmium levels in the kidney of humans (using *in vivo* neutron activation analysis) with incidence of tubular proteinuria has shown that a critical concentration of 200 μg cadmium/g wet weight in the kidney produces tubular dysfunction in 10 percent of the population (ATSDR, 1999; Goyer, 1991).

Cadmium toxicity affects calcium metabolism. Associated skeletal changes possibly related to calcium loss include bone pain, osteomalacia and osteoporosis. This disorder is known as "itai-itai" (or "ouch-ouch") disease, and has been observed in humans chronically exposed to cadmium in food in Japan (ATSDR, 1999; Goyer, 1991).

Studies in human populations provide conflicting evidence of a relationship between cadmium ingestion or inhalation and high blood pressure. Smoking is a confounding factor in studies of cadmium inhalation exposure, because of the known cardiovascular toxicity of cigarette smoke (although cigarette smoke is itself a significant source of cadmium). Studies with laboratory animals involving oral exposure to cadmium have shown some effects on the cardiovascular system (ASTDR, 1999).

Oral cadmium exposure reduces gastrointestinal intake of iron, which may result in anemia if dietary intake of iron is low. Studies in human populations provide conflicting evidence of a relationship between cadmium ingestion and the occurrence of anemia (ATSDR, 1999).

Kidney toxicity is considered to be the most sensitive effect of cadmium exposure. The no observed adverse effect level (NOAEL) for kidney toxicity from oral exposure to cadmium ranges from 0.002 to 0.01 mg/kg-day. An inhalation NOAEL (corresponding to a 4 percent incidence of proteinuria) is estimated to be approximately 0.017 mg/m^3 (ATSDR, 1999; USEPA [IRIS], 2000).

Reproductive or Developmental Toxicity

Available studies of human populations have shown no evidence of a relationship between oral or inhalation exposure to cadmium and reproductive or developmental toxicity. Studies with laboratory animals showed no evidence of adverse reproductive effects associated with inhalation exposure. High oral dose exposures to cadmium have shown evidence of reproductive toxicity in male laboratory animals (testicular damage and prostatic lesions). High oral doses in laboratory animals, ranging from 1 to 20 mg/kg, are fetotoxic, resulting in reduced fetal or pup weights and skeletal malformations (ATSDR, 1999).

Mutagenicity and Genotoxicity

There is conflicting evidence as to whether or not cadmium can cause chromosomal aberrations, either in humans or laboratory animals. Cadmium compounds have been shown to be mutagenic in some bacterial or *in vitro* test systems (ATSDR, 1999).

Carcinogenicity

Neither human nor animal studies provide sufficient evidence to determine whether or not cadmium is carcinogenic to humans from ingestion exposure. Ingestion of cadmium did not appear to be carcinogenic in humans in studies of populations in cadmium-impacted areas in England or Belgium. The geographical distribution of elevated prostate cancer rates was shown to parallel distribution of elevated cadmium concentrations in water, soil or crops in Alberta, Canada. Estimates of cadmium exposures were not performed in any of these studies. No evidence of excess cancer mortality was found in populations in Japan consuming cadmium-contaminated rice. ATSDR states that while there is little evidence of an association between ingestion exposure and increased cancer rates, the statistical power of the available studies to detect an effect is not high. Seven studies in rats and mice in which cadmium salts (acetate, sulfate, and chloride) were administered orally have shown no conclusive evidence of a carcinogenic response (USEPA 2000). While one feeding study exhibited increased incidences of tumors of the prostate, testes and hematopoietic system in rats (Waalkes and Rehm, 1992, as cited in ATSDR, 1999), these results are equivocal since the effects were not found to be dose-related, and some of the tumors were benign.

There is conflicting evidence as to whether cadmium is carcinogenic in humans by inhalation exposure. Prolonged inhalation of cadmium by battery and smelter workers, and workers in a cadmium recovery facility may be associated with increased incidences of lung or prostate cancer in some studies. However, in many cases there are confounding factors such as tobacco smoking and exposure to other carcinogenic metals that prevent making definitive conclusions from these epidemiological studies (ATSDR, 1999; Goyer, 1991). The cohort of workers at the cadmium recovery facility (from Colorado) was reevaluated, with

the analysis controlled for ethnicity and smoking history. This study concluded that there was a significant dose-response relationship (both in terms of duration of exposure and concentration in air) between cadmium exposure and lung cancer mortality. Based on this analysis, the excess lifetime cancer risk associated with the previous OSHA standard (100 $\mu g / m^3$) would be approximately 50 to 111 lung cancer deaths per 1,000 workers. At the current OSHA standard of 5 $\mu g / m^3$, the lifetime risk was estimated to be 2.6 to 6 lung cancer deaths per 1,000 workers exposed to cadmium for 45 years (Stayner et al., 1992, as cited in ATSDR, 1999). A parallel analysis of this same cohort of workers, which controlled for arsenic exposure concluded there was no association between cadmium exposure and lung cancer, and that arsenic exposure and cigarette smoking were the major determinants of lung cancer risk (Lamm et al., 1992, as cited in ATSDR, 1999). Further review found limitations with both of these studies (Doll, 1992, as cited in ATSDR, 1999), and more detailed assessment of potential exposures to cadmium and arsenic concluded that it was not possible to state whether or not cadmium was a human carcinogen based on this cohort of workers (Sorahan and Lancashire, 1997).

Studies in laboratory animals provide strong evidence of lung cancer resulting from inhaled cadmium (ATSDR, 1999). The U.S. Environmental Protection Agency has categorized cadmium as a probable human carcinogen by inhalation (Group B1) based on limited evidence in humans and sufficient evidence in laboratory animals (ATSDR, 1999; USEPA [IRIS], 2000). Similarly, the U.S. National Toxicology Program (NTP) has classified certain cadmium compounds as substances reasonably anticipated to be human carcinogens. In addition, the International Agency for Research on Cancer (IARC) has classified cadmium as carcinogenic in humans (ATSDR, 1999).

Pending external review, USEPA (1999) has recommended that cadmium be considered a probable human carcinogen by inhalation exposure. The International Agency for Research on Cancer (IARC) has classified cadmium in Group 2A, probably carcinogenic in humans.

Exposure Route Considerations

Ingestion

Cadmium is poorly absorbed from the gastrointestinal tract, however chronic ingestion exposures in humans have produced adverse effects principally in the kidneys and skeletal system. Ingestion of cadmium may interfere with absorption of dietary iron, and may be related to anemia in some cases. Nutritional deficiencies, particularly those associated with iron, calcium, vitamin D and protein may increase susceptibility to cadmium-related adverse effects. Ingestion of cadmium is not considered to be associated with reproductive toxicity or cancer in humans.

Inhalation

Inhalation exposure to cadmium in humans has been associated with adverse effects to the kidney and possibly with an increased incidence of lung cancer. Cadmium produces lung cancer in laboratory animals following inhalation exposure.

Dermal

There is no specific information about dermal toxicity of cadmium. Cadmium in soil or water appears to be poorly absorbed through the skin (ATSDR, 1999).

Sensitive Populations

Populations potentially sensitive to cadmium have not been studied systematically, however it is possible to infer about potential sensitivities based on the available data. Individuals with poor nutritional status, particularly those with deficiencies in iron and calcium, may experience increased absorption of cadmium from the gastrointestinal tract. Individuals with preexisting kidney damage may experience kidney toxicity at cadmium doses lower than for normal individuals (ATSDR, 1999). Smokers are generally more exposed than nonsmokers.

Indicators of Exposure

Blood cadmium levels are relevant indicators of recent exposure. Urinary cadmium levels are not particularly sensitive to recent exposures, but are relevant indicators of total body burden. When the critical concentration in the kidney is reached, urinary cadmium levels increase sharply due to the release of cadmium from metallothionein in the kidney coupled with decreased renal reabsorption of cadmium. Kidney dysfunction, the most sensitive effect, has been measured by increased levels of solutes (proteins and amino acids) in the urine. Increased urinary excretion of creatinine and metallothionein are additional indicators of kidney dysfunction due to cadmium exposure. Urinary excretion of other proteins and enzymes, while not specific indicators of cadmium-related kidney toxicity, also have been proposed as biomarkers of cadmium-related effects (ATSDR, 1999).

Toxicity Factors Derived for Risk Assessment

The USEPA has recently conducted a toxicological review of cadmium and compounds (USEPA, 1999) in support of a proposed revision of the toxicity factors currently listed in IRIS. However the review is currently under external review and the proposed toxicity factors are not finalized. Both the values currently listed in IRIS and the proposed changes are discussed below.

Toxicity Factors Currently Listed in IRIS

The USEPA recommended two oral reference doses (RfDs) for cadmium, one for cadmium exposure from food and one for cadmium exposure from water. Both RfDs recognize that a concentration of 200 μg/g (wet weight) in the human kidney cortex is the highest renal level not associated with significant proteinuria. A toxicokinetic model was used by USEPA to determine the level of chronic human oral exposure (NOAEL) which results in the critical concentration of cadmium in the kidney of 200 μg/g; the model assumes that 0.01 percent day of the cadmium body burden is eliminated per day (USEPA, 1985, as cited in IRIS). Assuming 2.5 percent absorption of cadmium from food or 5 percent from water, the toxicokinetic model predicts that the NOAEL for chronic cadmium exposure is 0.005 and 0.01 mg/kg-day from water and food, respectively (i.e., the doses corresponding to the 200 μg/g critical kidney concentration). An uncertainty factor of 10 to account for intrahuman variability was applied to these NOAELs to obtain an RfD of 0.0005 mg/kg-day (water) and an RfD of 0.001 mg/kg-day (food) (USEPA [IRIS], 2000). No inhalation RfD or reference concentration (RfC) is currently listed for cadmium.

An inhalation unit risk factor of 1.8×10^{-3} $(\mu g/m^3)^{-1}$ has been estimated from lung cancer incidence in the United States cohort of workers (i.e. from the cadmium recovery facility in Colorado). This corresponds to an inhalation cancer slope factor of 6.3 $(mg/kg\text{-}day)^{-1}$.

Quantitative estimates of oral carcinogenicity have not been developed, based on inadequate evidence that cadmium is carcinogenic in humans by the oral route of exposure (USEPA [IRIS], 2000).

Proposed Changes for IRIS

The critical toxic effect proposed for both the oral RfD and inhalation RfC is renal dysfunction as indicated by minimal proteinuria/enzymuria. This critical effect is supported from several cross-sectional population studies, especially by the CadmiBel population study of Buchet et al. (1990, as cited in USEPA, 1999). A toxicokinetic model was used with the data in this study to calculate both a daily oral intake and a continuous air concentration of cadmium that would result in a 10 percent occurrence of minimal enzymuria (the critical effect) in the population at the age of 70. A representative level of dietary cadmium intake was integrated into the toxicokinetic model. The net oral intake (model result minus diet) of 0.0007 mg/kg-day was designated the oral RfD. USEPA (1999) has proposed that one RfD be used for oral exposures to all media (i.e., separate RfDs were not proposed for ingestion of cadmium in food or water). The modeled concentration of cadmium inhaled concomitant with this same representative dietary intake was designated as the inhalation RfC of 0.0007 mg/m^3. For both the RfD and the RfC, alternate contributions of intake from background (and therefore different RfDs and RfCs) are described in USEPA's toxicological review (USEPA, 1999).

Considering the results of occupational inhalation exposures, and using a Poisson regression model on the epidemiology data, USEPA is proposing an inhalation unit risk of 4.4×10^{-3} (µg/m^3)$^{-1}$ (USEPA, 1999).

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Iron

Adverse Health Effects of Iron (Fe; CAS# 7439-89-6)

The primary sources for preparation of this profile were information obtained online from the National Library of Medicine [NLM] Hazardous Substances Data Bank [HSDB], as well as information from the U.S. Environmental Protection Agency [USEPA] National Center for Environmental Assessment (USEPA, 1996), and secondary reviews in the literature. A toxicological profile for iron has not been prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], nor has iron been incorporated into the U.S. Environmental Protection Agency [USEPA] Integrated Risk Information System [IRIS]. The focus of this profile is on key issues associated with risk assessment and toxicity for iron at Superfund sites (i.e. the critical effects considered in developing toxicity values, essential nutritional levels versus toxic levels, interactions with other metals).

Iron is one of the major constituents in the lithosphere (i.e. soil and rock), constituting approximately 5 percent in soil. Oxides and hydroxides of iron that are strongly pigmented determine the color of many soils. The concentration and form of iron is one factor that influences the bioavailability of other trace metals in soil (Kabata-Pendias and Pendias, 1992). Iron is an essential element in human nutrition. In general, the principal toxicological consequences for iron are associated with accidental acute ingestion exposures, chronic overload resulting from hemochromatosis, excess dietary iron or frequent blood transfusions (Goyer, 1991).

The primary use of iron is in alloys with carbon, manganese, chromium, nickel and other elements to form steel (NLM / HSDB, 1999).

Pharmacokinetics

Approximately 2 to 15 percent of ingested iron is absorbed from the gastrointestinal tract. During periods of increased iron need (childhood, pregnancy, blood loss) absorption of iron is increased (Goyer, 1991). No information was identified quantifying absorption of iron from inhalation. However, inhalation of high concentrations of iron results in pulmonary deposition. Long-term inhalation exposure to iron has resulted in mottling of lungs observable in chest X-rays, a condition referred to as siderosis (Hathaway et al., 1991).

The overall disposition of iron (i.e. absorption and distribution) is closely regulated to maintain homeostasis. While oral absorption of iron can vary over short periods, excretion of absorbed iron does not typically vary and is usually only about 0.01 percent of body burden. Total iron in the body normally ranges from 3 to 5 g, while total elimination is about 0.5 mg / day. Approximately 70 percent of the iron in the body is bound to hemoglobin or myoglobin in the blood. Excess iron is bound to the proteins ferritin and hemosiderin, which are synthesized in the liver. Along with the liver, the reticuloendothelial system (the spleen) is a storage site for excess iron (Goyer, 1991). Excretion of iron occurs primarily from the gastrointestinal tract (i.e., in the feces), with smaller amounts in urine and sweat. The normal breakdown of red blood cells and hemoglobin leads to the release of

iron and production of bile pigments. Measurements of fecal excretion of iron, however, may also be complicated by poor gastrointestinal absorption of ingested iron.

The issue of bioavailability of iron is especially important when considering soil exposure pathways. This is because the iron in soil can exist, at least in part, as a poorly soluble salt, and may also occur in particles of inert or insoluble material. These factors all may tend to reduce the bioavailability of iron.

Qualitative Description of Health Effects

While iron is an essential element in human nutrition, there is the potential for adverse health effects principally from ingestion exposure.

Acute Toxicity

Acute toxicity has resulted from the accidental ingestion of iron-containing medications, principally by children eating ferrous sulfate tablets with candy-like coatings (although these occurrences were more prevalent before the widespread use of "child-proof" caps on prescription medicine containers). Severe toxicity can occur following ingestion of more than 0.5 g of iron. Predominant signs of overexposure include ulceration of the gastrointestinal tract with vomiting (including blood), black stools, damage to the liver and kidneys, and metabolic acidosis. Death is thought to occur from renal failure and cirrhosis of the liver (Goyer, 1991).

Chronic/Subchronic Toxicity

The normal dietary intake of iron is estimated to range from 11 to 19 mg/day (USEPA, 1996). Chronic iron toxicity or iron overload (from ingestion exposure) can occur from excessive accumulation of iron in the body. There are three ways in which this could occur. The first is due to abnormal iron absorption from the gastrointestinal tract (i.e., idiopathic hemachromotosis). The second is through excessive dietary intake. The third may occur from regular blood transfusions required for some forms of anemia. In all cases, the body burden can increase to 20 to 40 g, and the excess iron accumulates in the liver, spleen, pancreas, endocrine organs and the heart. Adverse effects may include disturbance of liver function, diabetes mellitus, disturbance of endocrine function and cardiovascular effects. On a cellular level, increased lipid peroxidation occurs, resulting in membrane damage to cellular organelles (Goyer, 1991).

Inhalation of iron oxide fumes or dusts by workers in metals industries results in deposition of iron particulate in the lung. Inhalation of iron oxide fume or dust over long periods can cause a benign pneumoconiosis referred to as siderosis. Inhaled iron oxide does not cause fibrotic changes in the lungs of laboratory animals, and it is thought that the same applies to humans. Occupational inhalation exposures of 6 to 10 years can produce changes in the lung detectable by X-rays. The retained particulates produce X-ray shadows that may be indistinguishable from fibrotic pneumoconiosis. However, the observation of siderosis typically has not been associated with reductions in pulmonary function, even with exposures to concentrations higher than 10 mg/m^3. Some loss of pulmonary function has been observed in welders exposed to iron oxide fumes. However welders are exposed to a complex mixture of metallic oxide fumes and irritant gases, and the loss of pulmonary function from these causes should not be confused with benign pneumoconiosis caused by iron oxide (Hathaway et al., 1991).

Teratogenicity, Reproductive Toxicity, and Fetotoxicity

A survey in the United Kingdom of iron overdoses by pregnant women using iron supplementation concluded that there was no correlation between serum iron levels, outcome of pregnancy and birth weights, even with serum iron levels sufficient to cause moderate to severe toxicity (McElhatton et al., 1991; 1993, as cited in DART/ETIC). No reports were found of iron toxicity-induced developmental defects in experimental animals.

Mutagenicity and Genotoxicity

Iron is known to catalyze the production of highly reactive oxygen species, which in turn produce DNA damage in cell free systems. Genotoxic effects of iron nitriloacetic acid (iron-NTA) observed in *in vitro* systems include breakage of DNA strands and increased sister chromatid exchange frequency. The implications for DNA *in vivo*, or for human carcinogenicity are not known, though occupational exposure to iron-NTA is suspected of being carcinogenic (Hartwig et al., 1992 as retrieved from EMIC). Hydrogen peroxide-mediated oxidative DNA damage in iron-loaded liver cells may be potentiated by certain iron chelators, while other chelators exert a protective effect (Cragg et al., 1998 as cited in EMIC).

Carcinogenicity

Although a carcinogenic response from chronic ingestion of inorganic iron has not been reported, iron overload may potentiate other carcinogens. In one study, preneoplastic foci were produced in rat liver with diethylnitrosamine as initiator and partial hepatectomy with 2-acetylaminofluorene as promoter. Two weeks after promotion, the rats were fed 1.25 to 2.5 percent (12,500 to 25,000 mg/kg) dietary carbonyl iron for up to 45 weeks. The conclusion from this study was that dietary iron overload resulted in an increased number of preneoplastic foci but did not enhance the progression of these into hepatocellular carcinomas (Stal et al., 1999 as cited in MEDLINE). Iron dextran, which has limited use as a supplement, has been identified as reasonably anticipated to be a human carcinogen by the U.S. National Toxicology Program (NTP, 1998).

An increased incidence of lung cancer has been observed among hematite miners or iron workers exposed to iron oxide. However there may be concomitant factors explaining the observed cancer incidence, including cigarette smoke, silica dust, radon and polycyclic aromatic hydrocarbons (Hathaway et al., 1991, Goyer, 1991). The U.S. Environmental Protection Agency (USEPA) does not provide a weight of evidence classification for iron.

Exposure Route Considerations

Ingestion

The recommended daily allowance (RDA) for iron is 10 mg for children and males, 15 mg for females and 30 mg for pregnant and lactating women (NRC, 1989). Acute oral doses higher than 0.5 g has produced severe gastrointestinal toxicity. Chronic overexposure to iron can result in iron overload, which has produced adverse effects to the liver and other organs. Iron is not considered to be a human carcinogen (except for iron dextran), however iron may potentiate the effects of other carcinogenic substances. Ingestion of iron supplements is not considered to be associated with developmental toxicity.

Inhalation

Inhalation of high concentrations of iron oxide fume produces a benign pneumoconiosis called siderosis. An increased incidence of lung cancer among some populations of workers exposed to iron oxide fume or dust is more likely associated with other carcinogenic agents, including polycyclic aromatic hydrocarbons and radon.

Dermal

There is no information characterizing adverse effects associated with dermal contact with iron compounds.

Sensitive Populations

There are no known sensitive populations for exposure to iron. However, idiopathic hemochromatosis is thought to have a genetic component (Goyer, 1991).

Toxicity Factors Derived for Risk Assessment

USEPA's IRIS database does not currently provide a reference dose, cancer slope factor, or other toxicological information for iron (USEPA 2000). The USEPA Superfund Technical Support Center has developed a provisional oral reference dose (RfD) for iron. USEPA notes that iron is an essential element and that deriving a risk assessment value for it poses special problems in that the dose-response curve is "U-shaped" (i.e. there is a range of doses necessary to maintain health; doses both above and below that range can result in adverse effects). Thus, the provisional RfD must be protective against deficiency as well as toxicity. A NOEL for chronic iron overload has been estimated using the values for dietary intake and iron status indices taken from the second National Health and Nutrition Examination Survey (NHANES II) data base (STSC, 1999; USEPA, 1996). Looker et al. (1988, as cited in USEPA, 1996) made comparisons of dietary iron intake and biochemical indices of iron status using data from NHANES II. The average intakes of iron ranged from 0.15 to 0.27 mg/kg-day. The serum ferritin levels and percent serum transferrin saturation (both indicators of iron overload) were within the normal range. Thus, intake levels of 0.15 to 0.27 mg/kg-day consumed are both considered sufficient to protect against iron deficiency and insufficient to cause the toxic effects of iron overload.

Using the NOAEL of 0.27 (representing the upperbound value in the range of mean dietary iron intakes, dietary plus supplemental, taken from the NHANES II data base) and dividing by an uncertainty factor of 1 yields the provisional chronic oral RfD of 0.3 mg/kg-day. An uncertainty factor of 1 is supported by the fact that iron is an essential element. In addition, the information used to derive the oral RfD for iron was derived from intake data from over 20,000 individuals aged 6 months to 74 years and humans exert an efficient homeostatic control over iron such that body burdens are kept constant with normal variations in diet. This RfD supplies adequate levels of iron to meet the nutritional requirements of adults and adolescents. It does not supply the RDA to members of the population with greater requirements for shorter-than-lifetime durations, including children and pregnant women. Further, this RfD may not be protective of individuals with inherited disorders of iron metabolism, and could be conservative if applied to exposure scenarios involving forms of iron with low bioavailability (USEPA, 1996).

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Lead

Adverse Health Effects of Lead (Pb; CAS# 7439-92-1)

Recent comprehensive reviews of lead toxicity (National Research Council (NRC), 1993; U.S. Environmental Protection Agency [USEPA], 1998a; Agency for Toxic Substances and Disease Registry [ATSDR], 1999; U.S. Department of Health and Human Resources [DHHS], 1997) were the primary sources of information presented in this profile. To a lesser degree, information from USEPA (1998b) on toxicity of lead at low blood lead (PbB) levels was also used. In addition, recent articles (1996-1999) in the peer-reviewed literature on lead toxicity that were not cited in the above documents were also reviewed. In some cases, the primary sources are internally inconsistent. In these cases, this profile relies on the weight of evidence to draw conclusions. In most cases, the weight of evidence is explicitly discussed in the source documents.

Key issues associated with risk assessment and toxicity for lead at Superfund sites (e.g., oral bioavailability, use of pharmacokinetic models, adverse effects from exposure to low levels of lead for subchronic-chronic durations) have received the greatest emphasis in this profile.

Substantial quantities of both human and animal data are available regarding the toxicity of lead. ATSDR (1999) states that human data are preferred to animal data for assessing the potential health effects from lead exposure to persons living or working near hazardous waste sites or to other populations at risk. Therefore, this profile relies primarily on the human data.

Adverse effects of lead in humans are most typically evaluated in terms of PbB level, as an indicator of internal lead dose. External exposure (e.g., mg lead / kg bw-day or mg lead / m^3), as is commonly considered for other chemicals, is a far less accurate indicator of exposure to lead than are PbB levels. Therefore, whenever possible, this profile relates adverse effects to PbB levels rather than to external exposure.

Lead is a soft, bluish-gray metal that melts at 327.4°C (ATSDR, 1999). Lead sulfide, phosphate, and oxides are insoluble or practically insoluble in water; lead chloride is slightly soluble; and lead acetate and nitrate are soluble in water (ATSDR, 1999). Some primary uses of lead in the U.S. are in lead-acid storage batteries, ammunition, bearing metals, brass, bronze, cable covering, extruded products, sheet lead, solder, ceramics, type metal, ballast or weights, pigments, glass, radiation shielding, electronics, tubes or containers, oxides, and gasoline additives (ATSDR, 1999). In 1997, 87 percent of lead use in the U.S. was in the production of lead-acid storage batteries; 7.8 percent was used in metal products; and 5.3 percent was used for miscellaneous applications (Smith, 1998)[1].

Pharmacokinetics

Absorption

Lead absorption is influenced by the route of exposure, the exposure medium, speciation and physiochemical characteristics of lead, and the age and physiological states of the exposed individual.

Inhalation Exposure

Approximately 30-50 percent of particulate airborne lead is deposited in the lower respiratory tract of adult humans (ATSDR, 1999). USEPA (1989)[3] estimated that respiratory deposition of lead in children is 25-45 percent. The extent of deposition of inhaled lead can vary depending on such factors as lead speciation, particle size, and characteristics of the respiratory tract (Fleming, 1998; Spear et al., 1998; USEPA, 1986[1]). Almost all lead deposited in the lower respiratory tract is absorbed (USEPA, 1986; Morrow et al., 1980)[1], while lead deposited in the upper respiratory tract is generally transported to the esophagus, then swallowed.

Oral Exposure

Ingested lead is absorbed primarily in the duodenum (Mushak, 1991)[1]. The extent and rate of oral absorption of lead in humans is influenced by physiological states of the exposed individuals (e.g., age, fasting, nutritional status), physiochemical characteristics of the medium and lead ingested (e.g., type of medium, particle size, mineralogy, and lead solubility and species), and dose (ATSDR, 1999).

There is evidence that the percent absorption of lead in humans and animals decreases as intake of lead increases (Diamond et al., 1998; EPA, 1999b; ATSDR, 1999). Saturable mechanisms for lead absorption has been inferred from measurements of net flux kinetics of lead in perfused intestines of animals (Diamond et al., 1998). In addition, numerous observations of non-linear relationships between PbB concentrations and lead intake in humans provide support for the existence of a saturable absorption mechanism or some other capacity limited process in the distribution of lead in humans. (Diamond et al., 1998). However, pharmacokinetic studies on swine suggest that the non-linearity in the lead dose-blood lead relationship could derive from an effect of lead dose on some aspect of biokinetics of lead other than absorption (Diamond et al., 1998).

Nutritional Status

Gastrointestinal absorption of lead may be influenced by nutritional status. Children who are calcium or iron deficient may absorb more lead and have higher PbB levels (Mahaffey et al., 1986; Mahaffey and Annest, 1986; Marcus and Schwartz, 1987; Ziegler et al., 1978)[1]. Calcium in the diet has been shown to reduce absorption of ingested lead in adults (Blake and Mann, 1983; Heard and Chamberlain, 1982)[1].

Age

Gastrointestinal absorption of lead in young children is much higher than in adults. Children 2 weeks to 8 years of age absorb about 40-50 percent of ingested lead (Alexander et al., 1974; Ziegler et al., 1978)[1]. Non-fasted adults may absorb less than 10 percent of water-soluble lead (USEPA, 1996; O'Flaherty, 1998). No experimental data were available regarding absorption of ingested lead in older children. However, one study provides suggestive evidence that children ages 6-11 years absorb similar amounts of lead as do their mothers (Gulson et al., 1997)[1]. Age-dependent differences in absorption of ingested lead

have also been observed in animals (Forbes and Reina, 1972; Kostial et al., 1978; Pounds et al., 1978)[1], and may reflect physiological differences between immature and mature intestines (USEPA, 1986)[1].

USEPA (1998a), USEPA (1996, Adult Lead Exposure Model [ALEM]), and ATSDR (1999) each reported that absorption of ingested lead may increase during pregnancy. However, direct experimental evidence of increased absorption of lead in humans during pregnancy is not available. Instead, higher lead absorption during pregnancy is postulated based on increased calcium absorption and higher maternal PbB levels during pregnancy.

Fasting vs. Non-Fasting

Gastrointestinal tract status (fasting vs. non-fasting) affects lead absorption. The bioavailability of soluble lead in adults may be less than 10 percent when ingested with a meal, but as high as 60-80 percent when ingested after a fast (Blake, 1976; Blake et al., 1983; Blake and Mann, 1983; Graziano et al., 1995; Heard and Chamberlain, 1982; James et al., 1985; Rabinowitz et al., 1976, 1980)[2]. Fasted adults absorbed an average of 26 percent of lead in soil provided from the Bunker Hill Superfund Site compared to only 2.5 percent by non-fasted adults (Maddaloni et al., 1998). A proposed mechanism for these differences is the presence of certain components of ingesta (e.g., fiber, protein, other inorganics) in the small intestine of non-fasted humans that are known to inhibit absorption of inorganics (Ruoff et al., 1994, 1995; Ruoff, 1995).

Because lead is absorbed in the small intestine, the length of fasting required to affect lead absorption could be approximately equal to the amount of time between ingestion and clearing of the contents from the small intestine. In humans, this can take 12-14 hours. Per this definition, persons in the U.S. are not typically in a fasted state. Experimentally derived absorption rates for lead in non-fasting humans may best reflect bioavailability of lead under the typical (non-fasting) human exposure scenarios evaluated in risk assessment. For example, the absolute bioavailability of soluble lead in pregnant women of 20 percent used in USEPA's Adult Lead Exposure Model was calculated based on an estimate of meal-weighted bioavailability, assuming three meals each day and absolute bioavailability of 10 percent for lead ingested just before or soon after a meal (non-fasted state) and 60 percent for lead ingested at other times of the waking day (fasted state) (USEPA, 1996). In calculating bioavailability, it was assumed that adults are in the non-fasted state for 12 of 16 waking hours.

Exposure Medium

Absorption of lead in soil is generally less than that of soluble lead in water or the diet. USEPA pharmacokinetic models for lead assume that the relative bioavailability of lead in soil is only 60 percent of that for soluble lead in water (USEPA, 1994, USEPA, 1996). Absolute bioavailability of lead from soil is assumed to be 30 percent in children (USEPA, 1994) and 12 percent in pregnant women (USEPA, 1996). Maddaloni et al (1998) reported that non-fasted, non-pregnant adults absorbed an average of 2.5 percent of lead ingested in soil.

Speciation and Physiochemical Characteristics

A number of factors may reduce oral bioavailability of lead in soil relative to that for soluble forms of metals used in toxicity studies. These include adsorption of lead to soil, presence of lead in discrete mineral phases in soil, encapsulation of lead inside of insoluble particles in soil, and larger particle sizes of soil (Chaney, 1989). Site-specific bioavailability values lower

or higher than those assumed in USEPA lead models have been reported for lead in mining waste and weathered siliceous industrial slag (ATSDR, 1999; Freeman et al., 1992, 1994[1], 1996[1]; Dieter et al., 1993; Davis et al., 1997; Polak et al., 1996; USEPA, 1999b). Analysis of lead mineralogy at some sites showed that lead was present in relatively insoluble, discrete mineral phases (e.g., lead phosphate) and was encapsulated inside of particles (e.g., in silicates) (Davis et al., 1993; Davis et al., 1997). These properties of lead-bearing mineral phases and particles inhibited the release of soluble lead in the gastrointestinal tract and decreased its absorption.

USEPA has used an immature swine model to assess relative bioavailability of lead in soil at Superfund sites (LaVelle et al., 1991; Casteel et al., 1997). EPA's Technical Review Workgroup (TRW) for lead states that: "Currently, the juvenile swine model design offers the strongest method to measured site-specific bioavailability [of lead]" (USEPA, 1999b). A summary of results from immature swine model studies at sites impacted by mines and smelters is presented in Table 2-5 in ATSDR (1999). Relative bioavailability of lead in soil ingested by immature swine ranged from 50-82 percent of that of a similar dose of highly water-soluble lead acetate (ATSDR, 1999). USEPA's Integrated Exposure Uptake Biokinetic (IEUBK) model assumes a relative bioavailability of lead in soil of 60 percent (USEPA, 1994).

Dermal exposure

Absorption of lead in soil from the skin in humans is not well understood. Approximate 30 percent of lead nitrate was absorbed when applied to forearms of adult volunteers (Stauber et al., 1994). However, lead measured in blood and urine increased only negligibly, suggesting that the lead absorbed through the skin did not enter the systemic circulation or was present in the circulation in a form not bound to erythrocytes. Moore et al. (1980) reported that percutaneous absorption of lead-203 in humans from cosmetic preparations containing lead acetate was negligible and that lead by this route was unlikely to pose a threat to human health.

Most pharmacokinetic models for lead do not evaluate the dermal route of exposure (USEPA, 1994, 1996; O'Flaherty, 1998; Leggett, 1993; Bowers et al., 1994). An exception is California's Leadspread model which assumes an increase in PbB level of 0.00011 µg/dL per µg lead/day based on dermal absorption of only 0.06 percent of lead in soil (California Environmental Protection Agency, Department of Toxic Substances Control [DTSC], 1992, 1999).

Distribution

Absorbed lead enters blood; 99 percent of lead in blood is located in red blood cells (DeSilva, 1981; USEPA, 1986; Everson and Patterson, 1980; Hursh and Suomela, 1968)[1]. Blood lead rapidly exchanges with lead in other soft tissues (e.g., kidney, liver, lungs, brain) (ATSDR, 1999). The average half-life for lead in blood in adults ranges from 28-36 days (Rabinowitz et al., 1976; Griffin et al., 1975)[1] and for lead in soft tissues is about 40 days (Rabinowitz et al., 1976)[3].

In adults, approximately 94 percent of total body burden of lead is found in bones (Barry, 1975)[1]. In children, only about 73 percent of total lead body burden is in bone (Barry, 1975)[1]. There are two physiological compartments for lead in bone: an inert compartment with a half-life of approximately 27 years (Rabinowitz et al., 1976)[1] and a labile compartment in rapid equilibrium with lead in soft tissues and blood (Alessio, 1988; Chettle et al., 1991;

Hryhirczuk et al., 1985; Nilsson et al., 1991; Rabinowitz et al., 1976)[1]. Bone lead stores in adults can contribute approximately 40-70 percent of the lead in blood (Smith et al., 1996)[1].

Bone lead may be mobilized into maternal blood during pregnancy and lactation in humans (Gulson et al., 1998, 1999) and animals (Franklin et al., 1997. Lead in maternal blood is efficiently transported to the fetus. ATSDR (1999) reports that the fetal/maternal PbB ratio is about 0.9, based on maternal and umbilical cord PbB levels at time of delivery (Abdulla et al., 1997, Goyer, 1990; Graziano et al., 1990; Schuhmacher et al., 1996)[1]. USEPA (1996) recommends use of a $PbB_{fetal}/PbB_{maternal}$ ratio of 0.9 for the Adult Lead Exposure Model. Breast milk can also be a significant source of lead to nursing infants (Gulson et al., 1998; Mushak, 1998, 1999).

Excretion

Lead in the gastrointestinal tract that is not absorbed is eliminated in the feces. Absorbed lead that is not retained is eliminated in the urine or excreted in the feces following biliary secretion into the gastrointestinal tract (ATSDR, 1999).

Lead Pharmacokinetic Models

A number of lead pharmacokinetics models are available to predict PbB levels based on lead intake in various exposure media. These include models by USEPA (1994), USEPA (1996), DTSC (1992; 1999), O'Flaherty (1998), Leggett (1993), and Bowers et al. (1994). ATSDR (1999) presents a detailed review of the IEUBK, O'Flaherty, and Leggett models (ATSDR, 1999). In addition, numerous recent publications evaluate various aspects of the models used in predicting PbB levels (Bowers and Cohen, 1998; Carroll and Galindo, 1998; Griffin et al., 1999; Lakind, 1998; Oreskes, 1998; Rabinowitz, 1998; Tsuji and Serl, 1996) and the EPA TRW for lead presented a review of lead pharmacokinetic models during the March, 2000 meeting of the Society of Toxicologists. The EPA (1994, 1996) models are typically used at Superfund sites to evaluate risk posed from exposure of adults or children to environmental lead.

The Integrated Exposure Uptake Biokinetic (IEUBK) multicompartmental model (USEPA, 1994) predicts PbB levels in young children, age 0 through 6 years, based on lead intake from air, diet, dust, lead-based paint, soil, and water (available from: http://www.epa.gov/superfund/programs/lead/prods.htm#software). The IEUBK model has been shown to predict PbB level distributions reasonably well for children exposed primarily in the home (ATSDR, 1999).

The ALEM (USEPA, 1996) predicts PbB levels of pregnant women exposed to lead in soil/dust at work, and their fetuses. The Bowers et al. (1994) model, which served as a basis for the ALEM, also predicts PbB levels in pregnant women and their fetuses exposed to lead in soil/dust at work. However, the default parameter values used in the two models are different. Bowers and Cohen (1998) reported that parameter values recommended in the Bowers et al. (1994) model were better predictors of measured PbB levels of adults at several Superfund sites, than were parameter values recommended in the ALEM.

California's Leadspread model predicts PbB levels in young children (including pica children) and in adult residents and workers, based on lead in air, soil, water, the diet and homegrown produce (DTSC, 1992, 1999).

The O'Flaherty PBPK model (1998) can be used to estimate PbB levels for fetuses, infants, children, adolescents, and adults (including pregnant women and older adults), based on lead intake in air, diet, dust, lead-based paint, soil, and water. The O'Flaherty model has been shown to accurately predict PbB levels in children and adults, except when lead concentrations are very high (O'Flaherty 1993, 1995)[1]. In addition, the O'Flaherty model can predict short-term peaks in PbB levels in children resulting from subchronic exposure to lead (Lakind, 1998).

The Leggett (1993) multicompartmental model can predict PbB levels for children and adults, based on age-specific estimates of average daily inhalation and ingestion of lead (ATSDR, 1999). The Leggett model has been shown to accurately predict PbB levels in adults exposed to low levels of lead (ATSDR, 1999).

ATSDR (1999) has developed guidance for using environmental lead data and media-specific slope factors to estimate PbB levels. Estimated contributions to PbB from all exposure pathways are summed to yield a total predicted PbB level. ATSDR (1999) states: "[Unpublished] preliminary efforts to test [this model's] predictive power have shown promise" (p D-10).

Qualitative Description of Health Effects

The toxic effects of lead are generally the same regardless of the route of entry. Low level exposure to lead primarily affects the central nervous system, growth and development, vitamin D metabolism, and blood; however, most parts of the body can be damaged by high exposure to lead. The most severe neurological effect of lead is encephalopathy, which can lead to permanent neurological effects and death. At lower levels, lead produces more subtle neurological effects that can also be permanent. High levels of lead can produce anemia in adults and children. Lead has been shown to affect some parameters of heme synthesis at low PbB levels with no apparent threshold.

Other targets of lead include the cardiovascular, gastrointestinal, renal, and reproductive systems. There is uncertainty as to whether there is an association between exposure to lead and chromosomal aberrations and increased risk of cancer in workers. Lead is generally considered to be carcinogenic in animals.

Death

Children

Death can result from acute encephalopathy, which occurs in children at PbB levels as low as 80-100 μg/dL (NRC, 1993; ATSDR, 1999).

Adults

Death can result from acute encephalopathy in adults, which occurs at 100-120 μg/dL (NRC, 1993). ATSDR (1999) also reported that severe encephalopathy and death could occur in adults at PbB levels of 100-120 μg/dL.

Increased mortality rates have been reported in workers chronically exposed to lead, from malignant neoplasm, chronic renal disease, cardiovascular disease, cerebrovascular disease, lung cancer, or renal cancer (Cooper, 1988; Cooper et al., 1985; Fanning, 1988; Michaels et al., 1991; Lundstrom et al., 1997; Cocco et al., 1997)[1]. However, others have found no statistically significant increase in mortality rates from occupational exposure to lead (Gerhardsson

et al., 1986; 1995)[1]. With regard to occupational mortality studies, ATSDR (1999) reported that "the results …… are discrepant, and all the studies have design flaws that limit the validity of conclusions that can be drawn from their results" (p. 264).

Neurological Toxicity

Neurological effects occur in developing fetuses and young children at low PbB levels, without an apparent threshold. The brain has little or no capacity to repair injury cause by lead (Landrigan, 1999). Therefore, some adverse neurological effects of lead may be irreversible.

Children

Encephalopathy can occur in children starting with PbB levels of approximately 80-100 µg/dL (Bradley and Baumgartner, 1958[3]; Gant, 1938[3]; Bradley et al., 1956[3]; National Academy of Science [NAS], 1972[3]; NRC, 1993; Rummo et al., 1979[3]; Smith et al., 1983[3]; USEPA, 1986[3]). The early symptoms of encephalopathy can include irritability, poor attention span, headache, muscular tremor, loss of memory, and hallucinations. As encephalopathy progresses, more severe symptoms can appear, including delirium, convulsions, paralysis, coma, and death (Kumar et al., 1987)[3]. Encephalopathy can produce permanent cognitive impairment in survivors, including retardation and severe behavioral disorders (ATSDR, 1999; NRC, 1993).

NRC (1993) identified PbB level LOELs in children of 70 µg/dL for peripheral neuropathy, 30 µg/dL for slower nerve conduction, <25 µg/dL for decreased reaction time, and <10-15 µg/dL for deficits in neurobehavioral development, electrophysiologic changes, and lower IQ. ATSDR (1999) reported that peripheral neuropathy and reduced motor nerve conduction have been observed in children at PbB levels as low as 20-30 µg/dL (Erenberg et al., 1974; Landrigan et al., 1976; Schwartz et al., 1988; Seto and Freeman, 1964)[1].

ATSDR (1999) reported that neuro-developmental deficits are generally better correlated with PbB levels after birth, than with prenatal maternal or neonatal cord PbB levels. Similarly, NRC (1993) reported that "the findings pertaining to the association between indices of prenatal lead exposure and early development are mixed" and that "there is relative little consistency across the set of prospective studies in terms of the association between indices of prenatal lead exposure and later cognitive functions." For example, studies in Boston, Cincinnati, and Cleveland each reported early developmental delays associated with maternal or cord PbB concentrations, whereas studies in Australia did not find associations between prenatal PbB levels and postnatal indices of development (NRC, 1993). In some studies, associations between prenatal exposure to lead and developmental scores attenuated with increased age of the child (NRC, 1993).

It's clear that postnatal lead exposure can lead to persisting deficits in cognitive function in children. NRC (1993) reports that "there are striking consistencies in inverse associations between PbB levels measured in the first few years post-natally and intellectual performance at ages 6-10 years." NRC (1993) reviewed numerous cross-sectional and prospective studies and reported that most studies suggest a 2- to 4-point IQ deficit for each increase of 10-15 µg/dL in blood lead within the range of 5-35 µg/dL. Schwartz (1992) calculated the IQ decline over the blood lead range of 10-20 µg/dL to be 2.32 points for longitudinal studies and 2.69 points for cross-sectional studies[6].

USEPA (1998a) identified IQ deficit as the neuro-toxicological endpoint to be used to estimate the baseline health risks to young children (ages 1-2 years) from exposures to lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil in U.S. housing. Based on an evaluation of three meta-analyses of the relationship between PbB levels and IQ deficit decrements (Schwartz, 1993; Pocock et al., 1994; Schwartz, 1994), USEPA (1998a) concluded that a 1 µg/dL increase in PbB level would result on the average in a loss of 0.257 IQ points. Therefore, a doubling of PbB levels from 10-20 µg/dL would result in a loss of approximately 2.57 IQ points (USEPA, 1998a). USEPA (1998a) used this mathematical relationship between PbB level and IQ deficit to evaluate the baseline health risks to young children (ages 1-2 years) in the nation's housing.

Based on evaluation of a number of meta-analyses and cross-sectional and/or prospective studies (Needleman and Gatsonis, 1990; Pocock et al., 1994; Schwartz, 1994; Winneke et al., 1990; International Programme on Chemical Safety [IPCS], 1995), ATSDR (1999) reached similar conclusions to those of USEPA (1998a) and NRC (1993) regarding PbB levels and IQ deficits in children. ATSDR (1999) concluded that "there appears to be a modest association between indices of lead burden, usually PbB, and global indices of development or neuropsychological functioning, usually IQ." (p. 278). ATSDR (1999) also concluded "a doubling of PbB from 10 to 20 µg/dL is associated with an average IQ loss of 1-3 points" (p. 278) A threshold below which lead does not affect IQ in children has not been identified (ATSDR, 1999).

Various behavioral disorders may also occur in children at approximately 10 µg/dL (NRC, 1993). NRC (1993) states that "the most consistent finding across all studies of the CNS effects of lead in children is the association of increasing exposure with increasing reaction time, which apparently indicates an attention deficit." Children with higher lead burdens are more frequently classified as learning-disabled (NRC, 1993). In addition, PbB levels of 10 µg/dL and above have been associated with increased frequency of reading disability, disordered conduct, and possibly increased risk of criminal and delinquent behavior in adolescent and adult life (Landrigan, 1999)

Robinson et al. (1985)[1] reported a lead-related decrease in hearing acuity for young children. Hearing thresholds increased linearly throughout the range of PbB levels of 6-56 µg/dL. USEPA (1998b) reported that altered nerve conduction in auditory pathways and decreased hearing acuity have been observed in children with low PbB levels (Otto et al., 1985; Schwartz and Otto, 1987). The probability of increased hearing thresholds was associated with increased PbB levels from below 4 µg/dL to over 50 µg/dL (Schwartz and Otto, 1987)[4]. Osman et al. (1999) reported that hearing thresholds in children in Poland increased significantly with increasing PbB levels at all investigated sound frequencies, and that the relationship remained significant at PbB levels less than 10 µg/dL.

Alterations in brain electrical activity have also been observed in children at PbB levels of 10-15 µg/dL or lower (Benignus et al., 1981; Otto et al., 1981; Otto et al., 1982; Otto et al., 1985; Robinson et al., 1985; Winneke and Kraemer, 1984; Baumann et al., 1987)[4]. However, it is not known whether the measured alterations in brain activity represent adverse effects (USEPA, 1998b).

Adults

Encephalopathy can occur in adults at PbB levels as low as 100-120 µg/dL (Kehoe, 1961a[1], 1961b[1], 1961c[1], NRC, 1993; Smith et al., 1938[1]). DHHS (1997) indicated PbB levels in adults greater than 80 µg/dL may cause coma, encephalopathy, or death. Encephalopathy in adults can lead to peripheral polyneuritis involving sensory or motor nerves (NRC, 1993).

Overt neurological signs and decreased scores on neuro-behavioral tests have been observed in adults at PbB levels as low as 40-60 µg/dL (Baker et al., 1979, 1983; Campara et al., 1984; Haenninen et al., 1979; Maizlish et al., 1995; Williamson and Teo, 1986; Zimmerman-Tanaelia et al., 1983)[1]. DHHS (1997) reported that workers with PbB levels as low as 40-50 µg/dL may experience fatigue, irritability, insomnia, headaches, and subtle evidence of mental and intellectual decline (Mantere et al., 1984; Hogstedt et al., 1983). However, other studies have reported no effects on neuro-behavioral function in occupationally-exposed adults with PbB levels of 40-60 µg/dL (Milburn et al., 1976; Ryan et al., 1987)[1].

NRC (1993) identified a LOEL for peripheral nerve dysfunction (slower nerve conduction) in adults of 40 µg/dL. DHHS (1997) reported that some subclinical symptoms observed in adult workers exposed to lead, such as peripheral nerve dysfunction, can represent the early stages of permanent neurologic damage to the central and peripheral nervous system.

Two recent studies have reported an association between PbB levels and decreased neuro-behavioral performance in aging subjects with low PbB levels (mean of about 5 µg/dL) (Muldoon et al., 1996; Payton et al., 1998)[1].

Hematological Toxicity

Lead interferes with heme synthesis and erythrocyte function. Reduction of the heme body pool can lead to adverse effects in several physiological systems. For example, decreased heme synthesis can result in decreased hemoglobin levels in blood; decreased levels of 1,25-dihydroxyvitamin D, a hormone that regulates calcium metabolism; and increased blood levels of δ-aminolevulenic acid (ALA), a potential neurotoxicant (USEPA, 1998a).

Anemia can occur at PbB levels of 20-25 µg/dL and higher in children and 50 µg/dL and higher in adults, both from decreased hemoglobin production and increased red blood cell destruction (NRC, 1993). Increases in urinary coproporphyrin (CP-U) and δ-aminolevulenic acid (ALA-U) can occur in children and adults at PbB levels of around 40 µg/dL (NRC, 1993). Other symptoms of decreased heme synthesis and erythrocyte function may be observed at lower PbB levels. These symptoms include increased blood and plasma ALA; increased erythrocyte protoporphyrin (EP), and decreased erythrocyte δ-aminolevulinic acid dehydrase (ALAD) and pyrimidine-5'-nucleotidase activity. Some of these indicators (e.g., decreased erythrocyte ALAD and pyrimidine-5'-nucleotidase activity) may occur at PbB levels around 10 µg/dL or lower with no apparent threshold.

Children

NRC (1993) identified LOELs in children of 70 µg/dL for frank anemia, 40 µg/dL for increasing CP-U and ALA-U, and 20-25 µg/dL for anemia as indicated by reduced hematocrit. Subtle indicators of interference with heme synthesis and erythrocyte function have been observed in children at lower PbB levels. NRC (1993) identified LOELs in children of 15-20 µg/dL for increases in erythrocyte protoporphyrin and for pyrimide-5'-nucloetidase inhibition, and <10-15 µg/dL for ALA-D inhibition. ALAD activity has been

inversely correlated with PbB levels in the general population over the range of PbB levels of 3-34 µg/dL (Hernberg and Nikkanen, 1970)[1]. Erythrocyte pyrimidine-5'-nucleotidase activity has been inversely correlated with PbB levels in children over the ranges of PbB levels of 7-80 µg/dL (Angle and McIntire, 1978)[1] and <10-72 µg/dL (Angle et al., 1982)[1]. ATSDR (1999) reported PbB level thresholds for decreased ALAD or pyrimidine-5'-nucleotidase activity in children has not been identified.

Adults

NRC (1993) identified a LOEL for adults of 80 µg/dL for frank anemia, but further stated that lead workers' hemoglobin concentration is inversely and strongly correlated with PbB concentrations at a threshold of approximately 50 µg/L. NRC (1993) identified LOELs of 40 µg/dL for increasing CP-U and ALA-U, 25-30 µg/dL for erythrocyte protoporphyrin increase in males, 15-20 µg/dL for erythrocyte protoporphyrin increase in females, and <10 µg/dL for ALA-D inhibition. Decreased ALAD activity has been shown to be correlated with PbB levels in the general population over the entire range of PbB levels of 3-34 µg/dL, with no apparent threshold.

Renal Toxicity

Acute nephropathy can occur during the early stages of high exposure to lead, especially in children. Characteristic effects of acute nephropathy are morphological and functional changes in the proximal tubular epithelial cells (Loghman-Adham, 1997). Morphological changes consist of nuclear inclusion bodies, swelling of mitochondria, and cytomegaly of the proximal tubular epithelial cells (Loghman-Adham, 1997). Functional changes consist of aminoaciduria, glucosuria, phosphaturia, and hypophosphatemia; increased sodium and decreased uric acid excretion; and increased excretion of low molecular weight proteins and enzymes (ATSDR, 1999; Loghman-Adham, 1997). In acute nephropathy, glomerular effects are either minimal or absent (ATSDR, 1990). The symptoms of acute nephropathy are generally reversible (ATSDR, 1999).

NRC (1993) identified a LOEL in adults of 100-120 µg/dL for chronic neuropathy in adults. Characteristics of chronic lead nephropathy include progressive interstitial fibrosis, dilation of tubules and atrophy or hyperplasia of the tubular epithelial cells, few or no inclusion bodies, reduction in glomerular filtration rates, and azotemia. Chronic nephropathy can proceed to renal failure, with associated hypertension, hyperuricemia, and gout (Loghman-Adham, 1997). Renal changes produced by chronic nephropathy are generally irreversible (ATSDR, 1999).

ATSDR (1999) reports that efforts to evaluate the effects of lead on renal function have not been consistent when renal biopsies were not performed to prove conclusively the occurrence of nephropathy., and that "this could partially be explained by the choice of the renal function parameter studied" (p. 270).

Children

NRC (1993) identified a LOEL in children of 80-100 µg/dL for renal effects.

In a group of children with PbB levels of 40-120 µg/dL, Pueschel et al. (1972) found aminoaciduria in those with PbB levels of 50 µg/dL or more. Significant increases in urinary N-acetyl-B-D-glucosaminidase (NAG) were reported in children with a mean PbB level of 34.2 µg/dL; NAG activity in the children increased an average of 14 percent for each

10 µg/dL increase in PbB levels (Verberk et al., 1996)[1]. Because NAG might be a sensitive indicator of early subclinical renal disease, ATSDR (1999) reported that some level of tubular damage could occur in children at PbB levels less than 40 µg/dL.

Adults

NRC (1993) identified a LOEL of 100-120 µg/dL for chronic neuropathy in adults. Based on an evaluation of the human database, ATSDR (1999) concluded that chronic nephropathy in occupationally exposed workers is usually associated with PbB levels ranging from 60 to greater than 100 µg/dL. Loghman-Adham (1997) reported that chronic lead-induced nephropathy may develop in adults when PbB levels exceed a threshold of 60 µg/dL.

It is less clear whether adverse renal effects can occur in adults at lower PbB levels. Loghman-Adham (1997) reported that there is a correlation between low PbB levels (e.g., less than 40 µg/dL) and indicators of early renal dysfunction such as serum creatinine and creatinine clearance and urinary excretion of low molecular weight proteins and lysosomal enzymes (e.g., NAG). As discussed above, similar subtle effects on renal function have been observed in children at PbB levels less than 40 µg/dL (Verberk et al., 1996)[1].

Cardiovascular

Acute exposures to high levels of lead can produce cardiac lesions, electrocardiographic abnormalities, and hemolytic anemia in children and adults (ATSDR, 1999).

NRC (1993) identified a LOEL in adults of 10-15 µg/dL for increases in systolic and diastolic blood pressure in adults, including pregnant women. DHHS (1997) stated that studies conducted in the general population suggest that increased PbB levels are associated with small increases in blood pressure, and that the correlation may extend to PbB levels below 10 µg/dL (Pocock et al., 1988; Pirkle et al., 1985; Hertz-Picciotto and Croft, 1993; Schwartz, 1995).

Essentially all studies in humans have reported positive associations between PbB levels and blood pressure, and most of them have reported significant results (NRC, 1993). In 11 epidemiological studies, moderate changes in blood pressure were observed ranging from approximately –0.25 to –3.5 per PbB level change of 10 to 5 g/dL (NRC, 1993). In addition, numerous studies on rats have reported increased blood pressure associated with PbB level. For example, NRC (1993) reported that blood pressure in rats increased from about 114 to 136 as PbB levels increased from about 4 to 17 g/dL (based on Boscolo and Carmingnani, 1988).

Gastrointestinal Toxicity

Colic is an early symptom of lead poisoning in children and adults, characterized by abdominal pain, constipation, cramps, nausea, vomiting, anorexia, and weight loss (ATSDR, 1999). NRC (1993) identified a LOEL of 80-100 µg/dL for colic and other gastrointestinal effects in children. ATSDR (1999) reports that colic has been observed in children at PbB levels of 60-100 µg/dL and higher (USEPA, 1986; NAS, 1972). Symptoms of colic generally occur in adults at PbB levels of 100-200 µg/dL, although they have occurred in some workers at PbB levels as low as 40-60 µg/dL (Awad et al., 1986; Baker et al., 1979; Haenninen et al., 1979; Holness and Nethercott, 1988; Kumar et al., 1987; Marino et al., 1989; Matte et al., 1989; Muijser et al., 1987; Pagliuca et al., 1990; Pollock and Ibels, 1986; Schneitzer, 1990)[1].

Vitamin D Metabolism

Lead can interfere with the conversion of vitamin D to its hormonal form, 1,25-dihydroxyvitamin D (ATSDR, 1999). These effects of lead on vitamin D metabolism may be mediated via lead-induced inhibition of heme synthesis. Altered vitamin D metabolism can adversely affect maintenance of extra- and intra-cellular calcium homeostasis associated with cell maturation and tooth and bone development.

NRC (1993) identified a LOEL in children of 15-20 µg/dL for impaired vitamin D metabolism. USEPA (1998b) reported that reduction in vitamin D hormone synthesis has been observed in children with PbB levels of at least 10-15 µg/dL (based on Rosen, 1995). Large reductions in 1,25-dihydroxyvitamin D have been reported in children with PbB levels of 33-55 µg/dL (Rosen et al., 1980)[1] and 33-120 µg/dL (Rosen and Chesney, 1983; Rosen et al., 1980)[1]. However, Koo et al. (1991)[1] reported that no effects were observed on vitamin D metabolism in children with PbB levels of 4.8-23.6 µg/dL and adequate amounts of calcium, phosphorus, and vitamin D in their diet. IPCS (1995)[1] reviewed the human database with regard to vitamin D metabolism and concluded that PbB levels below 20 µg/dL do not affect circulating concentrations of 1,25-dihydroxyvitamin D in humans with adequate nutritional status. Children with nutritional deficiencies may represent sensitive members of the population with regard to the effects of lead on vitamin D metabolism.

Teratogenicity, Reproductive Toxicity, and Fetotoxicity

Fetuses

There is no question that exposure to prenatal exposure to lead can adversely effect fetuses. OSHA (1998) has stated: "Children born of parents either one of whom were exposed to excess lead levels are more likely to have birth defects, mental retardation, behavioral disorders, or die during the first year of childhood."

NRC (1993) identified a LOEL for reduced gestational age and birthweight of <10-15 µg/dL. NRC (1993) also states that: "some striking inconsistencies, yet to be explained, characterize the data on the relationship between prenatal lead exposure and fetal growth and maturation. For instance, in the large cohort (N=907) of women residing in Kosovo (Factor-Litvak et al., 1991), no associations were seen between midpregnancy PbB concentrations (ranging up to approximately 55 µg/dL) and either infant birthweight or length of gestation."

ATSDR (1999) reported that some studies reported that birth weight may be reduced as maternal or cord PbB levels increase (Bornschein et al., 1989; Dietrich et al., 1986; 1987; Bellinger et al., 1984; McMichael et al., 1986)[1], while other studies did not find an association between maternal or cord PbB levels and birth weight (Ernhart et al., 1985, 1986; Factor-Litvak et al., 1991; Greene and Ernhart, 1991; Moore et al., 1982; Needleman et al., 1984)[1]. Similarly, while some studies reported that gestational age may be reduced at PbB levels as low as 15 µg/dL (Dietrich et al., 1986; 1987; McMichael et al., 1986; Moore et al., 1982)[1], other studies did not find a significant relationship between PbB levels and gestational age (Bellinger et al., 1984; Factor-Litvak et al., 1991; Needleman et al., 1984)[1].

Regarding teratogenic effects of lead in humans, NRC (1993) states that impairments of CNS and other organ developments in fetuses occur at PbB levels of approximately 10 µg/dL. In

a retrospective study of 4,354 infants, Needleman et al. (1984) found a significant increase in the number of minor anomalies observed per child but no malformation was found to be associated with lead.[6]

Adults

High PbB levels can affect reproduction in human males and females (ATSDR, 1999; USEPA, 1998a; DHHS, 1997). Women occupationally exposed to high levels of lead during pregnancy have an increased rate of miscarriages and stillbirths (Nordstrom et al., 1979[3]; McMichael et al., 1986[3]; Baghurst et al., 1987[3]; Rom, 1976[5]). NRC (1993) identified a LOEL of 60 µg/dL for reproductive effects in adult females.

Potential reproductive effects in women from chronic low-level exposure to lead are less understood (NRC, 1993; ATSDR, 1999; USEPA, 1998a). Several large cohort studies with low PbB levels (average level during pregnancy of 5-20 µg/dL) did not report an association between lead and abortions or stillbirths (NRC, 1993). For men, NRC (1993) identified a LOEL of 50 µg/dL for altered testicular function. ATSDR (1999) and DHHS (1997) each reported that some reproductive effects (e.g., decreased sperm count, abnormal sperm morphology, decreased sperm mobility, hormonal changes) can occur among male workers with PbB levels as low as 30-40 µg/dL (Lancranjan et al., 1975[5]; Alexander et al., 1996[1,5],1998; Braunstein et al., 1978[5]; Ng et al., 1991[5]; Gennart et al., 1992[1]; Lerda, 1992[1]; Lin et al., 1996[1]).

The Occupational Safety and Health Administration (OSHA) has stated that its current standard for PbB level of 50 µg/dL in workers may not be protective for adverse effects in fetuses or reproductive effects in men and women (OSHA, 1991) Instead, OSHA recommends limiting PbB levels to less than 30 µg/dL for men or women who "intend to parent in the near future ..... to minimize adverse reproductive health effects to the parents and developing fetus" (OSHA, 1991). The American Council of Governmental Industrial Hygienists (ACGIH) recommends that PbB levels for a woman in the workplace remain below 30 µg/dL, "to protect her ability to have children that can develop normally" (ACGIH, 1994).

Developmental Effects in Children

There is uncertainty regarding the potential effects of prenatal lead on growth in children postnatally. In the Cincinnati prospective lead study, infants born to women with lead concentrations greater than 8 µg/dL during pregnancy grew at a lower rate than expected if increased lead exposure continued during the first 15 months of life (NRC, 1993). If postnatal exposure was low, the infants grew at a higher than expected rate (NRC, 1993). At 33 months, sustained exposure to PbB levels greater than 20 µg/dL were associated with reduced stature; however, prenatal exposure was no longer associated with reduced stature (NRC, 1993)

Postnatal exposure to lead affects growth in children. NRC (1993) reviewed the available data and concluded that postnatal PbB levels of 10-15 µg/dL in children had impacts on growth rates and attained stature. Schwartz et al. (1986)[6] evaluated the large NHANES II data set with respect to height, weight, and chest circumference as a function of PbB concentration. The three growth milestones in children under 7 years old were significantly and inversely associated with PbB concentrations, and the association was present over the PbB concentration range of 5-35 µg/dL. Frisancho and Ryan (1991)[6] found an inverse

association between PbB level and stature in a cohort of 1,454 5- to 12-year old children in the Hispanic HANES data set. Lauwers et al. (1986)[6] in Belgium reported statistically significant and inverse association among growth indexes and PbB concentration in children up to the age of 8 years. Prospective studies have also confirmed an association of postnatal lead exposure with retarded growth in infants and children (NRC, 1993).

Genotoxicity

There is uncertainty regarding the potential effects of lead on human chromosomes.

USEPA (1998a) reported that increased frequencies of chromosomal aberrations have been observed in some studies of occupationally-exposed workers, (Nordenson et al., 1978; Huang et al., 1988), but that most studies report no such increase in workers (Schmid et al., 1972; O'Riordan and Evans, 1974; Bauchinger et al., 1977; Maki-Paakkanen et al., 1981), or in children (Bauchinger et al., 1977). USEPA (1998a) reported that sister chromatid exchanges may (Grandjean et al., 1983; Leal-Garza et al., 1986; Huang et al., 1988), or may not (Maki-Paakkanen et al., 1981; Dalpra et al., 1983) be increased as a result of lead exposure. ATSDR (1999) reported that result of studies with human lymphocyte cultures exposed *in vitro* to lead acetate were nearly equally divided between positive and negative. Evidence in animal systems is also inconclusive (ATSDR, 1999).

Carcinogenicity

Lead is generally considered to be carcinogenic in animals. Evidence regarding carcinogenicity of lead in humans is generally considered to be inadequate.

As reported in USEPA (1998a), increased risks of kidney cancer (Selevan et al., 1985; Steenland et al., 1992; Cocco et al., 1997), lung cancer (Cooper et al., 1985; Gerhardsson et al., 1986; Anttila et al., 1995; Lundstrom et al., 1997), glioma (Anttila et al., 1996), rectal cancer (Fayerweather et al., 1997), and total malignant neoplasms (Cooper and Gaffey, 1975; Cooper, 1976, 1981; Kang et al., 1980; Cooper et al., 1985; Anttila et al., 1995; Gerhardsson et al., 1995; Lundstrom et al., 1997) have been observed in occupationally exposed workers. However, these studies lack necessary details to adequately assess carcinogenicity (USEPA, 1998a). ATSDR (1999) states: "the data currently available do not support an assessment of the potential carcinogenic risk of lead in humans" (p. 289) Similarly, USEPA (IRIS) (1999a) concludes: "the available human evidence is considered to be inadequate to refute or demonstrate any potential carcinogenicity for humans from lead exposure."

The carcinogenicity of lead in animals has been conclusively demonstrated (Azar et al., 1973; Koller et al., 1985; Van Esch and Kroes, 1969)[1]. USEPA (1999a) has recommended against using current cancer data in animals to derive a slope factor for use in human risk assessment, stating that "current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk." Based on the animal data, lead has been classified as a probable human carcinogen (B2) by USEPA (1999a), a possible human carcinogen (Group 2B) by IARC, reasonably be anticipated to be a carcinogen by NTP (1998), and an animal carcinogen by ACGIH (1999).

Exposure Route Considerations

The toxic effects of lead are generally considered to be similar regardless of the route of entry (ATSDR, 1999). There is an extensive database relating health effects in humans to

internal dose (e.g., PbB levels), and relatively few data relating human health effects to exposure-route specific external exposure (e.g., mg/kg-day or m^3/day). Therefore, the emphasis in this profile has been to correlate health effects in humans with exposure to lead, using PbB levels as an index of exposure. In some cases (e.g., occupational), PbB levels may reflect lead intake via several routes of exposure.

Numerous data are available in animals relating health effects to external dose (mg/kg-day). ATSDR (1999) summarizes many of these studies. However, ATSDR (1999) recommends against using animal data to quantitate human health hazards from exposure to lead, because animal data on lead toxicity are generally considered less suitable for assessing health effects than are human data. Instead, ATSDR (1999) states that human data are the best basis for assessing the potential health effects from lead exposure to persons living or working near hazardous waste sites or to other populations at risk.

Ingestion

Ingestion is the primary route of exposure for children and other non-occupationally exposed receptors. However, dose-response data based on external ingestion dose (mg/kg-day) in children were not located .

ATSDR (1999) reported that ingestion of 0.02-0.03 mg lead acetate/kg-day by adults for 14 days or less resulted in decreased ALAD (Cools et al., 1976; Stuik, 1974). Ingestion of 0.01-0.02 mg lead acetate/kg-day by adults for subchronic durations (3-7 weeks) resulted in decreased ALAD activity, increased red blood cell (RBC) porphyrin, and increased porphyrin IX in RBCs of adults (Cools et al., 1976; Stuik, 1974)[1].

Inhalation

Adults at work may be exposed to lead via inhalation and ingestion of dust. However, very little dose-response data for workers based on external dose (mg/kg-day or mg/m^3) in workers. ATSDR (1999) reported that humans inhaling lead at a concentration of 0.011 mg/m^3 had a 47 percent decrease in ALAD activity (Griffin et al., 1975). DHHS (1997) reported that severe damage to the peripheral nervous system has occurred historically from chronic, workplace exposures to lead of two or more times higher than the current U.S. Occupation Safety and Health Administration (OSHA) Permissible Exposure Limits [PEL] (Feldman et al., 1977) and that chronic exposure to lead above the OSHA PEL of 0.050 mg/m^3 may result in chronic nephropathy and potentially kidney failure.

Under the OSHA general industry lead standard (29 CFR 1910.1025), the PEL for personal exposure to airborne inorganic lead is 50 micrograms per cubic meter (µg/m^3) as an 8-hour time-weighted average (TWA) (OSHA, 1978). OSHA states that maintaining the concentration of airborne particles of lead in the work environment below the PEL represents a preventive measure intended to protect workers from excessive exposure, which OSHA defines as a PbB level of 40 µg/dL. ACGIH (1999) has recommended that worker lead exposures be kept below 50 µg/m (as an 8-hour TWA).

Dermal Contact

ATSDR (1999) reported that no studies were located regarding toxicity of lead in humans or animals specifically from dermal exposure. Dermally applied lead nitrate is rapidly absorbed into the skin, but the toxicology significance is not known.

Sensitive Populations

There is evidence that low PbB levels (e.g., 10-15 µg/dL or lower) can adversely affect development in humans exposed prenatally, postnatally, or both.

The embryo/fetus/neonate may be at increased risk because of transfer of maternal lead that may become mobilized from bone during pregnancy and lactation (Gulson et al., 1998, 1999; Mushak, 1998, 1999). Increased rates of miscarriages and stillbirths have been reported in women exposed to high levels of lead during pregnancy (Nordstrom et al., 1979[3]; McMichael et al., 1986[3]; Baghurst et al., 1987[3]; Rom, 1976[5]). In addition, low levels of lead have been associated in some studies with reduced birth weight and gestational age (NRC, 1993). The developing nervous systems of embryo/fetus/neonate may be particularly sensitive to lead toxicity (Rodier, 1995; DHHS, 1997). However, ATSDR (1999) reported that neurodevelopmental deficits in children are generally better correlated with PbB levels after birth, than with prenatal maternal or neonatal cord PbB levels.

Young children are generally at greater risk than adults for experiencing lead-induced health effects due to their physiological, developmental, and behavioral differences (ATSDR, 1999). In comparison with adults, young children absorb more lead from the gastrointestinal tract, retain more absorbed lead, and have a greater prevalence of nutritional deficiency (e.g., calcium, iron, zinc) which can increase both absorption and toxic affects of lead. In addition, the blood-brain barrier is incompletely developed in young children, which may allow greater transfer of lead to the brain, and the developing nervous system of children is more sensitive to the effects of PbB than that of adults. Young children also ingest much more soil/dust and more water and food per kg body weight and inhale more air per kg body weight than adults.

Some women may be at greater risk from exposure to lead because the conditions of pregnancy, lactation, and osteoporosis may intensify lead mobilization from bone demineralization, which can result in higher PbB levels (Bonithon-Kopp et al., 1986c; Markowitz and Weinburger, 1990; Silbergeld, 1991; Silbergeld et al., 1988; Thompson et al., 1985)[1]. Persons with pre-existing neurological dysfunction or kidney disease can be more sensitive to the effects of lead (ATSDR, 1999).

Indicators of Exposure

Several indices in blood and body tissues are available to serve as sensitive biomarkers for lead exposure and toxicity, including lead in blood, bone, and teeth, and physiological changes associated with the effects of lead on heme synthesis (ATSDR, 1999).

Lead in Blood

PbB levels are the easiest and most commonly used index of lead exposure and toxicity (ATSDR, 1999). The average half-life of lead in blood ranges from 28-36 days (Rabinowitz et al., 1976; Griffin et al., 1975)[1]; thus levels in blood reflect to a certain extent recent exposure (ATSDR, 1999). However, lead in blood exchanges with lead in other tissues including bone. Therefore, to a lesser degree, PbB can also reflect body burden which is more related to long-term exposure to lead (ATSDR, 1999). Although measured less often due to methodological problems, lead concentrations in plasma may be a more relevant index of lead distribution to target tissues such as bone marrow, kidney, and the nervous system than PbB levels (Bergdahl et al., 1997, 1999).

Fetuses and Children

A fetal PbB level of 10 µg/dL was recommended by USEPA (1996) for use in the ALEM, based on the assumption that the PbB level of concern for fetuses is the same as that for children. The National Research Council (NRC) (1993) has supported this PbB level of concern for fetuses. For children, 10 µg/dL is generally accepted as a PbB level of concern (USEPA, 1986[4]; 1990[4]; 1996; 1998a; 1998b; CDC, 1991[4], 1997; NRC, 1993). The rational for the selection by several government agencies of 10 µg/dL as the PbB level of concern for children is based on weight-of-evidence which indicates that numerous adverse effects may begin to be seen at PbB levels of around 10 µg/dL (as discussed next).

NRC (1993) reported that the adverse effects that occur at around 10 µg/dL in include (1) impairments in cognitive function and initiation of various behavior disorders in young children, and (2) impairments in calcium function and homeostasis in sensitive populations found in relevant organ systems. NRC (1993) also indicated that some of the neurological effects of lead are likely to be permanent.

USEPA (1998b) reported that USEPA's Air Quality Criteria Document for Lead (USEPA, 1986) concluded that for children: "(1) The collective impact of the effects at blood-lead concentrations above 15 µg/dL represents a clear pattern of adverse effects worthy of avoidance; (2) at levels of 10-15 µg/dL there appears to be a convergence of evidence of lead-induced interference with a diverse set of physiological functions and processes, particularly evident in several independent studies showing impaired neurobehavioral function and development; and (3) the available data do not indicate a clear threshold at 10-15 µg/dL, but rather suggest a continuum of health risks approaching the lowest levels measured" (p. 30316 in USEPA [1998b]).

USEPA (1998b) reported that USEPA's Clean Air Scientific Advisory Committee (CASAC) (USEPA, 1990) concluded that (1) various effects starting at blood-lead concentrations around 10-15 µg/dL or even lower in young children "may be argued as becoming biomedically adverse", (2) blood-lead concentrations at or above 10 µg/dL clearly warrant avoidance, especially for the development of adverse human health effects in sensitive populations and (3) "there is no discernible threshold for several lead effects and that biological changes can occur at lower [PbB] levels [than 10 µg/dL]" (p. 30316 in USEPA [1998b]). The SAB proposed setting 10 µg/dL as the maximal safe PbB level in children.

USEPA (1998b) reported that CDC (1991, 1997) stated that ''the scientific evidence showing that some adverse effects occur at blood-lead concentrations at least as low as 10 µg/dL in children has become so overwhelming and compelling that it must be a major force in determining how we approach childhood lead exposure'' (p. 30316 in USEPA, 1998b]).

USEPA (1998b) lists various effects that have been observed at PbB levels of at least 10-15 µg/dL, then states: "While it is possible that some of these effects are reversible (e.g., altered heme synthesis), or have unclear medical or functional implications (e.g., altered brain electrical activity), the Agency believes that the collective impact of these effects on diverse physiological functions and organ systems of young children with blood-lead concentrations as low as 10 µg/dL are clearly adverse" (p. 30316). USEPA (1998b) goes on to state that: "USEPA decided not to establish a level lower than 10 µg/dL because the evidence indicates that health effects at lower levels of exposure are less well substantiated, based on a limited number of studies, a limited number of children, and observation of

subtle molecular changes that are not currently thought to be sufficiently significant to warrant national concern." (p. 30317).

With respect to PbB levels in children higher than 10 µg/dL, CDC (1997) has stated that case and clinical management, environmental investigation, and lead-hazard control should be done for all children with PbB levels of 20 to 44 µg/dL, and that these services are required within 48 hours for children that have blood lead concentrations of 45 µg/dL or higher. PbB levels in a child of 70 µg/dL or higher is considered a medical emergency and that child must be hospitalized immediately.

Adults

In adults, there is less agreement regarding a single PbB level of concern. For pregnant women, a maternal PbB level of concern may be approximately 10 µg/dL for protection of the fetus (USEPA, 1996, NRC, 1993). In contrast, ACGIH has recommended that worker PbB levels be kept below 30 µg/dL, "to protect [a women's] ability to have children that can develop normally" (ACGIH, 1994; 1999), OSHA has said that women planning to have children should be advised to limit their PbB levels to less than 30 µg/dL (OSHA, 1991), and the World Health Organization (WHO) recommends that PbB levels in women of reproductive age not exceed 30 µg/dL (WHO, 1980)[5]. NRC (1993) has identified a LOEL of approximately 10 µg/dL for increases in systolic and diastolic blood pressure in adults including pregnant women. DHHS and NIOSH define elevated PbB levels among U.S. adults as those higher than 25 µg/dL (DHHS, 1997). DHHS has established a national goal to eliminate, by the year 2000, all occupational lead exposures that result in PbB levels great than 25 µg/dL (DHHS, 1997). Protective PbB levels for workers in states that require monitoring of PbB levels range from 10-40 µg/dL (DHHS, 1997). OSHA defines excessive exposure to lead as PbB levels greater than 40 µg/dL and requires medical removal of workers with PbB levels greater than 50 µg/dL (OSHA, 1978).

The World Health Organization (WHO) recommends that exposed workers be limited to PbB levels of less than 40 µg/dL (WHO, 1980)[5]. ATSDR (1999) states that a PbB level of 50 µg/dL has been determined to be an approximate threshold for the expression of lead toxicity in exposed workers.

Lead in Other Tissues

Lead accumulates in bone throughout a person's life. Therefore, lead in bone is considered a biomarker of cumulative exposure to lead in children and adults (ATSDR, 1999). *In vivo* tibial X-ray fluorescence (XRF) provides a non-invasive means of estimating cumulative lead absorption (ATSDR, 1999). Recent studies suggest that bone lead levels may be better predictors of some adverse effects than PbB levels (Gonzalez-Cossio et al., 1997[1]; Hu et al., 1994[1]; 1996b[1]; 1998); however, additional research is needed to better understand the relationship between bone lead, exposure, and adverse effects.

Lead in deciduous (i.e., "baby teeth") has been used as a biomarker of lead exposure (Needleman et al., 1993, 1996; ATSDR, 1999). Lead in enamel primarily reflects lead exposure that occurs *in utero* and early infancy, prior to tooth eruption, and lead in dentin is thought to reflect exposure that occurs up to the time the teeth are shed or extracted (Gulson, 1996; Gulson and Wilson, 1994; Rabinowitz, 1995; Rabinowitz et al., 1993).

ATSDR (1999) reports that urinary lead is not a useful biomarker for estimating low-level exposure to lead and that it is difficult to accurately measure endogenous lead in hair due to the potential for external surface contamination.

Physiological Changes

Other sensitive indices of lead exposure and toxicity are related to the effects of lead on heme synthesis. Lead can inhibit the enzyme ALAD, which may lead to decreased ALAD activity in erythrocytes and increased ALA activity in plasma and urine (ATSDR, 1999). Decreased ALAD in blood is a sensitive indicator of recent exposure to lead and may occur at PbB levels in the general population below 10 µg/dL with no apparent threshold (ATSDR, 1999). NRC (1993) has identified LOELs of <10 to 15 µg/dL for children and <10 µg/dL for adults for ALAD inhibition. Urinary ALA, which becomes elevated at PbB levels of around 40 µg/dL in adults and children, is not as sensitive an indicator as ALAD (NRC, 1993).

Lead can inhibit the enzyme pyrimidine-5'-nucleotidase, resulting in an increase in pyrimidine nucleotides in red blood cells. Inhibition of erythrocyte pyrimidine-5'-nucleotidase activity may occur at PbB levels in children below 10 µg/dL with no apparent threshold (ATSDR, 1999).

Lead can inhibit the enzyme ferrochelatase that transfers iron from ferritin to protoporphyrin to form heme. Inhibition of ferrochelatase can result in accumulation of erythrocyte protoporphyrin (EP) [also measured as free erythrocytes protoporphyrin (FEP) and erythrocyte ZPP] in erythrocytes. EP becomes elevated at PbB levels of 15-20 µg/dL in children and 15-30 µg/dL in adults (NRC, 1993) and reflects average lead levels during erythropoiesis over the previous 4 months (ATSDR, 1999). CDC (1997)[1] has defined lead toxicity in children as PbB levels greater than 10 µg/dL. In medical examinations of lead-exposed workers, OSHA requires measurement of PbB and ZPP levels, hemoglobin and hematocrit determinations, red cell indices, and examination of peripheral blood lead smears to evaluate red blood cell morphology (DHHS, 1997).

Toxicity Factors Derived for Risk Assessment

There is currently no oral reference dose (RfD) for ingestion of lead in IRIS (USEPA, 1999a). USEPA's RfD Workgroup has stated that it would be inappropriate to develop an RfD for lead, because some effects of lead (such as changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development) may occur at blood levels so low as to be essentially without a threshold (USEPA, 1999a).

There is currently no oral slope factor for lead. As stated in USEPA (IRIS) (1999a): "Quantifying lead's cancer risk involves many uncertainties, some of which may be unique to lead. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk. Thus, the Carcinogen Assessment Group recommends that a numerical estimate not be used."

No inhalation reference concentration (RfC) or inhalation slope factor is available for lead (USEPA, 1999).

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Manganese

Adverse Health Effects of Manganese (Mn; CAS# 7439-96-5)

The comprehensive review of manganese toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1997 forms the primary basis for this profile. Specific discussion about toxicity values used to characterize health risks potentially associated with exposure to manganese is based on information provided in the U.S. Environmental Protection Agency [EPA] Integrated Risk Information System [IRIS].

The focus of this profile is on key issues associated with risk assessment and toxicity for manganese at Superfund site (i.e. the critical effects considered in developing toxicity values, essential nutritional levels versus toxic levels, interactions with other metals). The issue of bioavailability of manganese is especially important when considering soil exposure pathways.

Manganese is one of the more abundant trace elements in soil and rock, with concentrations typically ranging from 200 to 3,500 mg/kg. Manganese occurs most commonly as a divalent cation. It is oxidized under atmospheric conditions and is found in the soil principally as oxides and hydroxides in the form of coatings on soil particles. (Kabatas-Pendias and Pendias, 1992). Manganese and its compounds are used in making steel alloys, dry-cell batteries, ceramics, dyes, welding rods, oxidizing agents, fertilizer and animal food additives (Goyer, 1991).

Pharmacokinetics

Absorption of manganese following ingestion ranges from 3 to 5 percent. Dietary iron deficiency appears to lead to increased manganese absorption. Information is not available regarding absorption following inhalation. Manganese is distributed throughout the body, with the highest levels found in the liver, pancreas and kidney. Studies with laboratory animals indicate that inhaled manganese may be preferentially accumulated in the lung and the brain. Ingested manganese that is absorbed is excreted primarily from the intestines via the bile, in the feces. Smaller amounts of absorbed manganese are excreted in the urine. Approximately 60 percent of inhaled manganese is excreted in the feces, and chronic inhalation exposure results in elevated urinary levels of manganese (ATSDR, 1997).

The issue of bioavailability of manganese is especially important when considering soil exposure pathways. This is because the manganese in soil may exist, at least in part, as poorly soluble salts. These factors all may tend to reduce the bioavailability of manganese.

Qualitative Description of Health Effects

Manganese is an essential element in human nutrition, as a cofactor in several enzymatic reactions. When ingested, manganese is considered to be among the least toxic of the trace elements. The adverse health effects from manganese exposure are principally associated with inhalation exposure in workplace settings. Acute inhalation can produce irritation of

the respiratory tract. Chronic inhalation exposure can produce a central nervous system disorder known as manganism.

Acute Toxicity

Reports of adverse effects in humans from excess acute ingestion exposure to manganese are rare. Manganese reportedly has low oral toxicity in laboratory animals. Inhalation of elevated concentrations of manganese compounds in occupational settings can lead to an inflammatory response in the lungs in humans, producing localized edema. Signs and symptoms of lung irritation may include cough, bronchitis, pneumonitis and small losses in pulmonary function (ATSDR, 1997).

Chronic/Subchronic Toxicity

Manganese reduced survival in chronic feeding studies with rats at doses higher than 200 mg/kg-day, with the cause of death attributed to nephrotoxicity and renal failure. Mice appear to be less sensitive to adverse effects from chronic manganese ingestion (ATSDR. 1997).

Reports of human intoxication following ingestion exposures to manganese are not common. However, information suggests that oral exposure to manganese can produce neurological symptoms in some humans. Individuals in aboriginal islander populations near Australia, who were exposed to elevated concentrations of manganese in drinking water have exhibited symptoms including weakness, ataxia, loss of muscle tone and a fixed emotionless face (Kilburn, 1987, as cited in ATSDR, 1997). Data on concentration-response relationships and lack of a suitable control group limit the conclusions from this study. Other factors besides manganese exposure, including genetic factors, dietary deficiencies and alcohol consumption may have been responsible for the observed symptoms (Cawte et al., 1987; 1989, as cited in ATSDR, 1997). Elevated concentrations of manganese in drinking water (1.8 to 2.3 mg/L) reportedly were associated with increased prevalence of neurological signs in the elderly residents in Greek communities. The occurrence of these effects was compared with a control community with low concentrations of manganese in drinking water (Kondakis et al., 1989, as cited in ATSDR, 1997). While limitations with this study prevent drawing conclusions about the relationship between chronic manganism and manganese in drinking water, the results suggest that chronic oral exposure to manganese can lead to neurological changes in humans.

Numerous studies have concluded that chronic inhalation exposure to high concentrations of manganese compounds can lead to a disabling neurological condition resembling Parkinsonism, which is referred to as manganism. Principal signs include tremors, weakness in the legs, staggering gait, behavioral disorders, slurred speech and a fixed facial expression. Levels of exposure associated with manganism are poorly characterized, but may range from 0.14 to 22 mg/m^3. The 0.14 mg/m^3 value has been identified by ATSDR as an indicator of subtle neurological effects, and is considered a lowest observed adverse effect level (LOAEL) (ATSDR, 1997).

Teratogenicity, Reproductive Toxicity, and Fetotoxicity

No studies were located regarding developmental effects in humans following oral exposure. Elevated levels of manganese ingestion in rats may lead to a slight delay in maturation of the male reproductive system, without effects to sperm morphology or

reproductive function. Impotence and loss of libido are common symptoms of manganism following high-dose inhalation exposure in human males. Impaired male fertility at levels not producing frank manganism has been reported in one study (ATSDR, 1997).

Mutagenicity and Genotoxicity

Manganese may be clastogenic in mice following oral gavage exposure. Manganese is mutagenic in some bacterial test systems, but is not mutagenic in others. Genotoxicity has been observed in *in vitro* test systems with mammalian cells. These studies suggest that manganese has some genotoxic potential, however the data are not adequate to assess genotoxic risk to humans (ATSDR, 1997).

Carcinogenicity

Inhalation exposure in humans has not been associated with an increased incidence of cancer. Intraperitoneal injection of mice has resulted in lung tumors, in one study. Chronic oral exposures to mice and rats in other studies have indicated small increases in pancreatic tumors (in rats) and pituitary tumors (in mice), though these effects were not dose-related (ATSDR, 1997). A bioassay performed by the U.S. National Toxicology Program (NTP) concluded there was equivocal evidence of carcinogenicity based on a small increased incidence of thyroid gland follicular adenoma and a significantly increased incidence of follicular cell hyperplasia (NTP, 1992, as cited in ATSDR, 1997). The U.S. Environmental Protection Agency (USEPA) has categorized manganese as Group D, not classifiable with regard to human carcinogenicity. The International Agency for Research on Cancer (IARC) has not classified manganese.

Exposure Route Considerations

Oral

Manganese is an essential element in human nutrition. Therefore, any quantitative risk assessment for manganese must take into account aspects of both the essentiality and the toxicity of manganese. Daily intake ranges from 2 to 9 mg/day (Goyer, 1991). The Food and Nutrition Board of the National Research Council (NRC, 1989, as cited in USEPA 2000) determined an "estimated safe and adequate daily dietary intake" (ESADDI) of manganese to be 2 to 5 mg/day for adults. Manganese is poorly absorbed following oral exposure. Reports of human intoxication following ingestion exposures are not common. However, some studies suggest that neurological effects may be associated with consumption of drinking water with elevated levels of manganese. Although ingestion exposure studies suggest that manganese may be weakly carcinogenic in laboratory animals, these data are inadequate to support a classification as carcinogenic by USEPA.

Inhalation

Several studies have shown that inhalation of manganese in occupational settings is associated with a neurological disorder known as manganism. The principal signs of manganism include tremors, weakness in the legs, staggering gait, behavioral disorders, slurred speech, and a fixed facial expression.

Dermal

Other than burns resulting from contact with manganese-containing oxidizing agents, no reports were located describing toxic effects following dermal exposure.

Sensitive Populations

Several researchers have observed considerable variability in neurological effects resulting from exposure to manganese. While the reasons for this are not clear, it is thought that there may be wide variability in manganese absorption and excretion among individuals following either inhalation or ingestion. This variability may be due to differences in transferrin saturation from dietary iron or other metals, calcium or protein intake, or levels of alcohol consumption. The very young have received attention as a potentially sensitive group, based on studies in laboratory animals indicating that neonates absorb and retain higher levels of manganese compared with adults. There are indications but no direct evidence that neonates are more sensitive to manganese-induced neurotoxicity than adult animals. The elderly may also be more sensitive to manganese-induced neurological effects. Individuals with poor iron nutritional status may absorb manganese more readily, and individuals with liver dysfunction may have impaired excretion of manganese, compared with normal individuals (ATSDR, 1997).

Indicators of Exposure

Manganese levels in blood and urine can be indicators of exposure. Blood levels are considered a better indicator of body burden, while urinary levels are a better indicator of recent exposure (ATSDR, 1997).

Toxicity Factors Derived for Risk Assessment

Development of the oral reference dose (RfD) for manganese recognizes that disease states in humans have been associated with both deficiencies and excess intakes of manganese. The oral RfD for manganese is set at 10 mg/day (0.14 mg/kg-day) and is based on the upper end of the normal dietary intake rate. This value is considered a no observed adverse effect level (NOAEL) for dietary intake and is not adjusted with an uncertainty factor. USEPA emphasizes that individual requirements for, as well as adverse reactions to manganese may be highly variable. The reference dose is estimated to be an intake for the general population that is not associated with adverse health effects; this is not meant to imply that intakes above the reference dose are necessarily associated with toxicity (USEPA [IRIS], 2000).

The oral RfD was evaluated further for manganese in other media (drinking water or soil) based on the epidemiologic study of manganese in drinking water, performed by Kondakis et al., 1989, (as cited in USEPA [IRIS], 2000). While the results from this study do not allow a quantitative evaluation of dose-response, they raise concerns about possible adverse neurological effects at doses not far from the range of essentially. For assessing exposure to manganese from drinking water or soil, USEPA (2000) recommends a modifying factor of 3, yielding an oral RfD of 0.047 mg manganese/kg-day (0.14 ÷ 3). They list four reasons for using the modifying factor to adjust the oral RfD for soil and water exposure: (1) in fasted individuals there may be increased uptake of manganese from water; (2) the study by Kondakis et al. (1989) raises some concern for possible adverse health effects associated with a lifetime consumption of drinking water containing about 2 mg/L of manganese;

(3) because infants can be fed formula that typically has a much higher concentration of manganese than does human milk, manganese in the water could represent an additional source of intake for infants; and (4) neonates may absorb more manganese from the gastrointestinal tract, may be less able to excrete absorbed manganese, and absorbed manganese may more easily cross their the blood-brain barrier.

For the CDL human health risk assessment, an oral RfD of 0.14 mg/kg-day was used to evaluate ingestion of manganese in soil, as recommended by USEPA Region 10.

The inhalation reference concentration (RfC) is based a LOAEL for neurological effects of 0.15 mg/m^3 based on an 8-hour time-weighted average, for 5 days/week, observed in studies of occupational exposure to manganese dust. This value is converted to a human equivalent concentration (HEC) of 0.05 mg/m^3 using USEPA methods for calculating RfCs. An uncertainty factor of 1000 was applied to this value, which reflects a factor of 10 to protect sensitive individuals, 10 for use of a LOAEL, and 10 for database limitations reflecting both the less-than-chronic periods of exposure and the lack of developmental data, providing a RfC of 0.00005 mg/m^3 (USEPA [IRIS], 2000). This corresponds to an inhalation RfD of 0.000014 mg/kg-day

The oral RfDs of 0.047 to 0.14 mg/kg-day and inhalation RfD of 0.000014 mg/kg-day for manganese (USEPA 2000) suggest that inhaled manganese may be much more toxic than ingested manganese. Differences in absorption between the two routes cannot alone account for this very large difference. USEPA reports that after absorption via the respiratory tract into blood, manganese is transported through the blood stream directly to the brain, bypassing the liver and first-pass hepatic clearance. They state that this pathway from the respiratory tract to the brain is the primary reason for the differential toxicity between inhaled and ingested manganese. In addition, recent studies in animals have shown that manganese has a unique ability among metals to be taken up in the brain via olfactory pathways (Tjalve and Henriksson 1997). This process involves direct diffusion of manganese from the nasal cavity into the central nervous system without entering blood, therefore bypassing both the first-pass effects of the liver and the blood-brain barrier (Tjalve and Henriksson 1997). This direct pathway to the central nervous system might account in part for the higher toxicity of inhaled manganese.

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Mercury

Adverse Health Effects of Mercury (Inorganic Hg; CAS# 7487-94-7; Methyl Mercury; CAS#22967-92-6)

The comprehensive review of mercury toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1999 forms the primary basis for this profile. Specific discussion about toxicity values used to characterize health risks potentially associated with exposure to mercury is based on information provided in the U.S. Environmental Protection Agency [USEPA] Integrated Risk Information System [IRIS].

Key issues associated with assessment of risks associated with mercury at Superfund sites, including bioavailability in certain media (i.e. soil), chemical forms in which mercury occurs in the environment (inorganic versus organic), toxicity of different valences and forms of mercury, and the basis for health guidance values (the reference dose and minimal risk level), have been addressed in this profile.

Mercury has been shown to be toxic to human populations as a result of occupational exposure and accidental ingestion of mercury-contaminated food. The nature of mercury toxicity differs with the chemical form. Elemental mercury vapor and organic mercury vapor have produced toxicity to the central nervous system and kidneys following inhalation exposure in workers. Ingestion of inorganic mercury salts in laboratory animals also has produced toxicity in the kidney. Accidental ingestion exposure to high levels of organic mercury compounds has produced developmental toxicity in humans.

Elemental mercury is a silvery metallic liquid that is volatile at room temperature. Mercury, found in soil and rocks, typically occurs as an ore known as cinnabar, consisting or insoluble mercuric sulfide. Concentrations in soil and rock average 0.5 parts per million (ppm), though actual concentrations vary considerably depending upon location. Mercury is recovered by heating cinnabar and condensing the vapor to form elemental mercury. Much of the mercury produced in the United States comes from secondary sources, such as recycling. The largest use of mercury is for electrolytic production of chlorine and caustic soda. Other uses include electrical devices, switches and batteries, measuring and control instruments, medical and dental applications, and electric lighting.

Pharmacokinetics

Absorption

Absorption following inhalation of elemental mercury vapor is relatively high (74 to 80 percent), however gastrointestinal absorption of elemental mercury is low. Following ingestion, organic mercury compounds are absorbed more readily than inorganic mercury. Animal studies indicate that gastrointestinal absorption of inorganic mercury (as mercuric chloride) ranges from 10 to 30 percent. Absorption of organic mercury compounds following ingestion is very high, with absorption from aqueous solutions being nearly 100 percent. However, bioavailability of methylmercury compounds in some foods (particularly

grains) has been shown to be lower compared with aqueous solutions. Although organic mercury compounds (particularly dialkyl mercury) in solution may be readily absorbed through the skin, elemental and inorganic mercury compounds are not absorbed well dermally (ATSDR, 1999).

The issue of bioavailability of mercury is especially important at mining, milling, and smelting sites. This is because the mercury at these sites often exists, at least in part, as a poorly soluble sulfide, and may also occur in particles of inert or insoluble material. These factors all may tend to reduce the bioavailability of mercury from soil.

Distribution, Metabolism, and Excretion

Once absorbed, both elemental mercury and organic mercury compounds distribute throughout the body. Due to their high lipophilicity, they can readily cross blood-brain and placental barriers. The kidney is a major organ for deposition of both elemental and methyl mercury. Inorganic salts of mercury also distribute throughout the body following ingestion, with highest levels found in the liver and kidney and lowest levels in the brain. Mercuric ion does not readily pass the blood-brain or placental barriers. Elemental mercury is oxidized to the divalent inorganic cation (mercuric ion) principally in the liver, although there is limited evidence that mercuric ion can be reduced to elemental mercury, and excreted by inhalation. Organic mercury is demethylated in the liver to form inorganic mercuric ion. Inorganic mercury is excreted through both the urinary and fecal (biliary) routes, whereas organic mercury compounds are principally excreted through the fecal (biliary) route. Elemental mercury is also excreted by exhalation from the lungs (ATSDR, 1999).

Qualitative Description of Health Effects

Acute Toxicity

Acute inhalation exposure to high concentrations of elemental or organic mercury compounds has occurred under occupational or accidental conditions, producing effects to the respiratory tract (dyspnea, tightness and pains in the chest), cardiovascular system (elevation in heart rate and blood pressure) and gastrointestinal tract (stomatitis, anorexia, bleeding from the gums). Acute oral exposure to inorganic or organic mercury compounds are also associated with cardiovascular and gastrointestinal effects.

Subchronic and Chronic Toxicity

The nervous system is the most sensitive target organ for mercury toxicity following chronic exposures, but kidney toxicity can be manifested following high doses. Effects to the kidney and nervous system can occur from both long-term inhalation and oral exposures. Workers chronically exposed to mercury vapor have shown evidence of kidney toxicity, including proteinuria, albuminuria, and tubular damage, as evidenced from biopsies of kidney tissue. Kidney toxicity has been observed in humans accidentally ingesting inorganic mercury salts. In several studies with laboratory animals, kidney toxicity also has been seen following subchronic and chronic oral exposures to inorganic mercury salts. Evidence of mercury-induced neurological effects comes principally from reported human poisonings from ingestion of methylmercury-contaminated fish in the Minamata area of Japan, and ingestion of seed grain treated with methylmercury fungicides in Iraq. Symptoms reported

included ataxia, impaired ability to speak, muscular weakness, abnormal reflexes, mood disorders, distal paresthesias, and impaired hearing and vision (ATSDR, 1999).

Reproductive and Developmental Toxicity

Abortions and decreased litter size are the principal reproductive effects observed in laboratory animals exposed to mercury. Rats administered methylmercuric chloride orally (from 10 to 30 mg/kg) showed increased pre- and postimplantation losses. Maternal body weights were depressed, suggesting that the doses producing reproductive toxicity were maternally toxic. Accidental ingestion of mercury in food has been associated with incidences of developmental toxicity in humans. The large-scale poisonings that occurred in Iraq in 1956, 1960 and 1971-1972, involved ingestion of wheat flour ground from seeds treated with ethylmercury-p-toluene sulfonanilide (a fungicide). Developmental effects included delays in walking and talking, mental retardation and seizures. A dose-response relationship was seen between organic mercury intake and severity of neurological symptoms.

Several ongoing studies of human populations are providing useful information regarding the toxicity of methylmercury. These include studies of populations in the Island of Madeira, Brazil, the Faroe Islands, and the Republic of Seycelles (Risher et al. 1999a). In a cross-section study of the Island of Madeira, 150 first-graders exposed to methylmercury *in utero* and in fish at levels up to 0.8 ppm were evaluated for neurological effects. Mercury levels averaged 14 ppm in the hair of children, and up to 54 ppm in maternal hair (Risher et al. 1999a). Increased latency of the auditory brainstem-evoked potentials in children was found to be related to mercury concentrations in maternal hair. In a cross-sectional study in Brazil, approximately 400 children in 4 villages exposed to mercury *in utero* and in fish were evaluated for neurological effects. Significant correlations were observed between increased mercury concentrations in hair and decreased performance on neurological tests (Risher et al. 1999a). In a study in the Faroe Islands, 917 children, 7 years of age underwent detailed neurobehavioral examination (Grandjean et al. 1997, 1998). Prenatal methylmercury exposure was assessed by measuring maternal hair mercury concentrations. Mild decrements in the domains of motor function, language, and memory were observed in children whose mothers had hair mercury concentrations of 10-20 ppm. The authors concluded that subtle effects on brain function could be detected at "prenatal methyl mercury concentrations currently considered to be safe."(Grandjean et al. 1998). In a prospective longitudinal study in the Republic of Seychelles, children exposed to methylmercury *in utero* and in fish were evaluated. No neurological effects of significance have been detected in this population thus far, in spite of average concentrations of mercury in hair of children of 6.5 ppm (maximum 25.8 ppm) and of mothers of 6.8 ppm (maximum 26.7 ppm) from consumption of an average of 12 fish meals per week (Davidson et al. 1998, as cited in ATSDR, 1999).

Genotoxicity

Mercury may produce chromosomal aberrations in humans and laboratory animals. Studies in human populations consuming methylmercury-contaminated seafood indicates a relationship between exposure and chromosomal breaks in lymphocytes, however the data are limited and considered inconclusive. Studies in rats given high dosages of mercuric chloride by gavage also indicate an dose-related frequency in chromosomal aberrations, including chromatid breaks and unscheduled DNA synthesis (UDS) (ATSDR, 1999).

Carcinogenicity

Results from a 2 year National Toxicology Program bioassay (NTP, 1993, as cited in ATSDR, 1999) indicate that mercuric chloride may cause an increased incidence of thyroid follicular cell tumors and forestomach squamous cell papillomas in rats, and renal carcinomas in mice. Limited animal studies have also shown renal tumors in male rats and male mice following oral exposure to organic mercury. There are no reports describing cancer incidences in human populations exposed to inorganic or organic mercury (dietary exposure or occupational exposure). The U.S. Environmental Protection Agency (USEPA) has classified mercuric chloride and methylmercury into Group C, possible human carcinogens, based on the absence of data in humans and limited evidence of carcinogenicity in animals (USEPA 2000).

Exposure Route Considerations

Ingestion

Adverse effects from ingestion exposure principally have been associated with consumption of grain products or seafood contaminated with organic mercury. The principal adverse effects have been neurological and developmental toxicity. Ingestion of inorganic mercury, the form most likely to be found in soil, has been associated with kidney toxicity in laboratory animals. The adverse effect of concern with soil exposure scenarios therefore is likely to be kidney toxicity. Ingestion studies in laboratory animals exposed to mercury suggest tumor-producing effects.

Inhalation

Adverse effects from inhalation have been associated with occupational exposure to elemental mercury vapor or organic mercury compounds. Accidental poisonings have occurred to children inhaling spilled elemental mercury. The principal adverse effects have been neurological and kidney toxicity. Inhalation toxicity associated with inorganic mercury salts, the form most likely to be found in soil, has not been studied.

Dermal

While organic mercury compounds can be absorbed through the skin, inorganic forms are not expected to be significantly absorbed by this route. Inorganic mercurial compounds used for topical application have produced dermatitis and neurological effects.

Sensitive Populations

Children are considered a sensitive population for exposure to mercury. Differences in sensitivity between children and adults results largely from greater permeability of the underdeveloped blood-brain barrier *in utero* and in infants. Also contributing are differences in routes of exposure and intake rates (for example exposure via ingestion of mothers milk), and importance of developmental milestones during childhood exposure periods (such as language or cognitive development).

In general, young children are exposed to higher doses of methylmercury than are adults (e.g., approximately 1.5- to 2-fold or higher on a body-weight basis). It was recognized that the postnatal nervous system remains vulnerable to methylmercury; however, it is uncertain whether the young child's sensitivity to neurological effects of methylmercury is more like

that of the fetus or that of the adult. Children also appear to have different patterns of tissue distribution of mercury and methylmercury (i.e., biokinetic patterns) than do adults (USEPA, 1999).

Indicators of Exposure

Blood and urinary mercury are typically used as indices of exposure in the workplace. Hair analyses also have been used as indicators of exposure. The most appropriate indicator depends on the form of mercury, the duration of exposure, and time since exposure (ATSDR, 1999).

Toxicity Factors Derived for Risk Assessment

USEPA has published chronic oral reference doses (RfDs) for mercuric chloride and methyl mercury on their Integrated Risk Information System (IRIS) database (USEPA, 2000). The most sensitive adverse effect for mercuric chloride is reported to be formation of mercuric-mercury-induced autoimmune glomerulonephritis. Based on weight of evidence from three subchronic feeding and/or subcutaneous studies in rats, the oral RfD for mercuric chloride is 0.0003 mg/kg-day. All treatment groups exhibited a toxic effect, therefore a no observed adverse effect level (NOAEL) was not reported. An uncertainty factor of 1,000 was applied for extrapolations from LOAEL to NOAEL endpoints, subchronic to chronic exposures, and animal to human populations. USEPA report their confidence in the oral RfD for mercuric chloride is high. A subchronic oral RfD of 0.003 mg/kg-day is provided in the Health Effects Assessment Summary Tables (HEAST) for mercuric chloride, based on autoimmune effects observed in rats from subcutaneous injection (USEPA, 1997).

For methylmercury, the chronic oral RfD in IRIS is 0.0001 mg/kg-day, based on developmental neurologic abnormalities seen in human infants exposed *in utero* due to maternal ingestion of seed grain treated with methylmercury fungicides in Iraq.. Maternal intake levels, estimated based on a concentration of mercury in maternal hair of 11 ppm, were used as the dose surrogate for the observed developmental effects in the infants. An uncertainty factor of 10 was used, and USEPA reported medium confidence in the RfD. A subchronic oral RfD of 0.0001 mg/kg-day is provided in HEAST for methylmercury, based on developmental neurological effects in human infants (USEPA, 1997).

The basis of USEPA's chronic oral RfD of 0.0001 mg/kg-day for methylmercury was described in USEPA's Mercury Study Report to Congress (USEPA 1997). ATSDR (1999) has derived a chronic oral Minimum Risk Level (MRL) of 0.0003 mg/kg-day, based on information from several recent studies of human populations. The MRL was specifically based on the arithmetic mean value of 15.3 ppm mercury in maternal hair during pregnancy for the highest exposed quantile in the 66-month (postnatal) cohort in the Seychelles Child Development Study. Children in this exposure group showed no decrement in performance on neurological tests. An overall uncertainty factor of 4.5 was applied to the NOAEL for mercury exposure to account for potential variability in the U.S. population and possible subtle neurological effects not tested for in the Seychelles Study. Although not identical to the RfD, the ATSDR "safe level" has been reviewed in a number of recent workgroup sessions (Risher et al. 1999a,b), and represents the Department of Health and Human Services official position.

The RfC of 0.0003 mg/m^3 is provided in IRIS for elemental mercury vapor, based on neurotoxicity observed in humans and incorporating an uncertainty factor of 30. Occupational studies supporting the RfC reported incidences of hand tremor, increased memory disturbances, and slight autonomic dysfunction. USEPA has reported medium confidence in the RfC for elemental mercury. The RfC is supported by ATSDR's inhalation MRL of 0.002 mg/m^3.

No cancer slope factors have been developed for mercury compounds. However, USEPA has classified both mercuric chloride and methylmercury in Group C (possible human carcinogen), based on the absence of data in humans and limited evidence of carcinogenicity in animals whereas elemental mercury is in Group D (not classifiable due to inadequate data) (USEPA 2000).

Recently, USEPA has developed the Mercury Research Strategy to address key scientific questions in order to reduce uncertainties currently limiting the Agency's ability to assess and manage mercury and methylmercury risks (USEPA 1999). This will include evaluations to link toxicity to exposure using a biokinetic model, assessment of sensitive subpopulations, evaluation of recent epidemiological studies, and evaluation of immunological effects.

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Zinc

Adverse Health Effects of Zinc (Zn; CAS# 7440-66-6)

A comprehensive review of zinc toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1994 forms the primary source of information for preparation of this profile. Information regarding the development of toxicity values for zinc has been incorporated from the U.S. Environmental Protection Agency [USEPA] Integrated Risk Information System [IRIS], available online. Additional information regarding uses of zinc and occurrence in the environment has been obtained online from the National Library of Medicine [NLM] Hazardous Substances Data Bank [HSDB].

The focus of this profile is on key issues associated with risk assessment and toxicity for zinc at Superfund sites (i.e. the critical effects considered in developing toxicity values, essential nutritional levels versus toxic levels, interactions with other metals).

Zinc is used primarily in galvanized metals and metal alloys. In addition, various inorganic zinc salts have numerous commercial uses. Zinc oxide is used in the rubber industry as a vulcanization activator and accelerator and to slow down oxidation, and also as a reinforcing agent, heat conductor, pigment, UV stabilizer, supplement in animal feeds and fertilizers, catalyst, chemical intermediate, and mildew inhibitor. Zinc sulfate is used in rayon manufacture, agriculture, zinc plating, and as a chemical intermediate and mordant. Zinc chloride is used in smoke bombs, in cements for metals, in wood preservatives, in flux for soldering; in the manufacture of parchment paper, artificial silk, and glues; as a mordant in printing and dye textiles, and as a deodorant, antiseptic, and astringent. Zinc chromate is used as a pigment in paints, varnishes, and oil colors. Zinc compounds are also used as ingredients in products, such as sun blocks, diaper rash ointments, deodorants, athlete's foot preparations, acne and poison ivy preparations, and antidandruff shampoos (ATSDR, 1994).

Pharmacokinetics

The body's natural homeostatic mechanisms control zinc absorption from the gastrointestinal tract. Persons with adequate nutritional levels of zinc absorb approximately 20 to 30 percent of all ingested zinc. However, zinc-deficient individuals absorb greater proportions of administered zinc. Other differences in zinc absorption are probably due to the type of diet (amount of zinc ingested, amount and kind of food eaten). For example, dietary protein facilitates zinc absorption. High phosphorus intakes in animals decrease zinc absorption, and dairy products that contain both calcium and phosphorus reportedly decrease zinc absorption in humans. Complexing of zinc with amino acids generally enhances zinc absorption (ATSDR, 1994).

Absorption of zinc in the lungs has not been quantitatively studied. Zinc absorption in the lungs is dependent on the compound, particle size, solubility, and the condition of the lungs. Inhaled zinc is also subject to gastrointestinal absorption due to ciliary clearance and

swallowing. Elevated levels of zinc have been found in the blood and urine of workers exposed to zinc oxide fumes (ASTDR, 1994).

Zinc is one of the most abundant trace metals in humans. It is found normally in all tissues and tissue fluids and is a cofactor in over 200 enzyme systems. Together, muscle and bone contain approximately 90 percent of the total amount of zinc in the body. Zinc is present in blood plasma, erythrocytes, leukocytes, and platelets, but is chiefly localized within erythrocytes. Zinc deficiency has been demonstrated to decrease the ability of erythrocytes to resist hemolysis *in vitro*, suggesting that zinc stabilizes erythrocyte membranes. Much of the zinc in plasma is bound to albumin. The limited number of binding sites for zinc in plasma albumin may regulate the amount of zinc retained by the body; albumin-bound zinc has been correlated with plasma zinc levels (ATSDR, 1994).

Zinc is found in blood serum at a concentration of approximately 1 mg/L in both men and women. Several studies have reported increased levels of zinc in the serum and urine of humans and animals after inhalation, oral, or dermal exposure to zinc. However, relationships between serum and/or urine levels and zinc exposure levels have not been established. Excretion of zinc from the body occurs mostly from the intestine, in the feces, although some zinc is also excreted through the kidneys, in the urine. Fecal and urinary excretion of zinc increases as intake increases. Following ingestion, fecal excretion is high due to both poor gastrointestinal absorption and biliary secretion of zinc. Studies with rats confirm that zinc is excreted in the bile (ATSDR, 1994).

Qualitative Description of Health Effects

Zinc is an essential element in human nutrition, required for the proper functioning of numerous metalloenzymes and proper cell growth and division. Zinc deficiency has been associated with dermatitis, anorexia, growth retardation, poor wound healing, impaired reproductive capacity, impaired immune function, and depressed mental function; an increased incidence of congenital malformations in infants has also been associated with zinc deficiency in the mothers (ATSDR, 1994). The recommended daily allowance (RDA) is 15 mg for adult males, 12 mg for adult females, 15 mg for pregnant women, 19 mg for nursing mothers during the first 6 months and 16 mg during the second six months, 10 mg for children older than 1 year, and 5 mg for infants 0 to 12 months old (NRC, 1989, as cited in ASTDR, 1994). Excessive exposure to zinc is reported to be relatively uncommon, and requires high levels of exposure. Zinc does not accumulate in the body with continued exposure, and levels in the body are modulated by homeostatic mechanisms (Goyer, 1991).

Acute Toxicity

Gastrointestinal distress is a common symptom following acute oral exposure to zinc compounds. Accidental poisonings have occurred as a result of the use of zinc supplements and from food contamination caused by the use of zinc galvanized containers. Symptoms develop within 24 hours and include nausea, vomiting, diarrhea, and abdominal cramps (Goyer, 1991) A single dose estimated to be 6.7 mg/kg ingested in water (limeade prepared in a galvanized container) produced gastrointestinal distress and diarrhea. Vomiting, abdominal cramps, and diarrhea with blood was observed in one individual after ingestion of 440 mg zinc sulfate/day (2.6 mg zinc/kg-day) in capsules as a medically prescribed treatment. Gastrointestinal upset (abdominal cramps, vomiting, nausea) occurred in 26 of 47 healthy volunteers following ingestion of zinc sulfate tablets (150 mg as zinc ion in three divided doses per day, 2 mg zinc/kg-day) for 6 weeks. Gastrointestinal effects have also

been observed in laboratory animals, including reduced food consumption and ulceration of the stomach lining.

Acute oral exposure to 2.6 mg zinc/kg-day as zinc sulfate for 1 week resulted in anemia in one person, however it was noted that the anemia may have been secondary to the gastrointestinal hemorrhages. Treatment-related changes in hematological parameters have been observed in humans and animals after intermediate or chronic exposure to zinc or zinc-containing compounds.

Inhalation exposure to high concentrations of some zinc compounds (zinc oxide fume) has been associated with "metal fume fever". Attacks of metal fume fever are characterized by chills and fever, weakness, and sweating. Recovery usually occurs within 24 to 48 hours. Exposure of guinea pigs to zinc oxide fumes at a concentration of 5 mg/m^3 (the Threshold Limit Value) 3 hours/day for 6 days produced temporary decrements in lung volume and carbon monoxide diffusing capacity. These functional changes were correlated with increased lung weight, inflammation involving the proximal portion of alveolar ducts and adjacent alveoli, interstitial thickening, and increased pulmonary macrophages and neutrophils in adjacent air spaces (Goyer, 1991; ATSDR, 1994). Zinc chloride, a corrosive inorganic salt, is more damaging than zinc oxide to the mucous membranes of the nasopharynx and respiratory tract upon contact. Zinc chloride is a primary ingredient in smoke bombs used by the military for screening purposes, crowd dispersal, and fire-fighting exercises. Serious respiratory injury has resulted from accidental inhalation of smoke from these bombs (ATSDR, 1994).

Chronic/Subchronic Toxicity

Longer-term administration (1 to 8 years) of zinc supplements (in one case, 2 mg/kg-day as zinc sulfate) has caused anemia in humans. Oral administration of zinc compounds produced decreased hemoglobin, hematocrit, erythrocyte, and/or leukocyte levels in rats. A lowest observed adverse effects level (LOAEL) with subchronic exposure in rats (1 month) was 12 mg/kg-day as zinc chloride. However, a no observed adverse effect level (NOAEL) for hematological effects of 191 mg/kg-day as zinc acetate was reported, following 3 months exposure in rats. The considerable range in effects levels is not clear, but may be due to different zinc compounds or differences in strains or ages of the test animals. Mice appear to be less sensitive to hematological effects from zinc exposure compared to rats.

Exposure to 191 mg/kg-day of zinc acetate administered orally in rats over 3 months produced no liver toxicity, but produced kidney toxicity, with epithelial cell damage in the glomerulus and proximal convoluted tubules and increased plasma creatinine and urea levels. Again, mice appeared to be relatively less sensitive to renal effects from zinc exposure compared to rats (ATSDR, 1994).

Teratogenicity, Reproductive Toxicity, and Fetotoxicity

Little information is available on the developmental and reproductive toxicity of inorganic zinc to humans or animals. Reproductive toxicity observed in laboratory animals (principally rats) includes fetal resorption, increased stillbirths, preimplantation losses and reproductive failure. The lowest observed adverse effects levels for these effects range from 200 to 250 mg/kg-day (ATSDR, 1994). Only one report in the literature suggested adverse developmental effects in humans due to exposure to zinc. Four women were given zinc supplements of 0.6 mg zinc/kg-day as zinc sulfate during the third trimester of pregnancy. Three of the women had premature deliveries, and one delivered a stillborn infant (Kumar

1976, as cited in ATSDR, 1994). However, the significance of these results cannot be determined because very few details were given regarding the study protocol, reproductive histories, and the nutritional status of the women. Other human studies have found no developmental effects in the newborns of mothers consuming up to 0.3 mg zinc/kg-day (as zinc sulfate) during the last two trimesters of pregnancy (ATSDR, 1994).

Information on the developmental and reproductive toxicity of inorganic zinc compounds to humans by other routes of exposure was not available.

Mutagenicity

Genotoxicity studies have provided very limited evidence of mutagenicity and of weak clastogenic effects. Zinc chloride is reported to be positive in the Salmonella assay, negative in the mouse lymphoma assay, and a weak clastogen in cultured human lymphocytes. Zinc sulfate is reported to be not mutagenic in the Salmonella assay, and zinc acetate is reported to not induce chromosomal aberrations in cultured human lymphocytes. Zinc oxide was not mutagenic in Salmonella (USEPA, 2000). Chromosomal aberrations have been observed in bone marrow cells in rats following exposure to 14.8 mg zinc/kg-day as zinc chlorate in drinking water. An increased incidence of sister chromatid exchange was observed in bone marrow cells with a drinking water dose of 17.5 mg zinc/kg-day. Chromosomal aberrations caused by zinc were observed in the bone marrow cells of mice maintained on a low calcium diet. It was thought that calcium may be displaced by zinc in calcium-depleted conditions, leading to chromosome breaks and/or interfering in the repair process (ATSDR, 1994).

Carcinogenicity

Studies of zinc exposure in humans have not specifically evaluated carcinogenicity. Studies of occupational exposure to zinc compounds have also been conducted, but have limited value because they do not correlate exposure with cancer risk. The potential carcinogenicity of zinc has been evaluated in only a few animal studies. A summary of the currently available information is presented in IRIS (USEPA, 2000).

Occupational exposure studies to zinc dust or fumes have not reported an increase in the incidence of cancer, however, the studies were designed to evaluate other endpoints and did not specifically address cancer (USEPA, 2000). Epidemiological studies have examined cancer mortality rates in occupationally exposed workers and in residents in areas with potentially high zinc contamination. No association between cancer mortality and zinc exposure could be established for workers employed in electrolytic zinc and copper refining plants; however, analysis of the data was limited by the small number of deaths in workers exposed to zinc (Logue et al., 1982, cited in ATSDR, 1994). Lung cancer mortality was reported to be elevated in residents living in an old lead/zinc mining and smelting area, but there was no association with environmental levels of zinc (Neuberger and Hollowell, 1982, cited in ATSDR, 1994). Because many confounding factors (i.e., smoking, occupation, and duration of residence) were not considered, it is unlikely that the study could have detected zinc-related effects (ATSDR, 1994).

Newborn Chester Beatty stock mice were maintained for one year on drinking water containing 0, 1,000, or 5,000 ppm Zn (0, 170, 850 mg zinc/kg-day, as zinc sulfate), or on a diet containing zinc oleate (5,000 ppm Zn for 3 months followed by 2,500 ppm for 3 months, and then 1,250 ppm for the rest of the study period). The incidence of hepatomas, malignant lymphomas, and lung adenomas was not statistically different from control values, although the incidence of hepatomas in mice on the zinc-augmented diet was increased over

that in the controls (30.4 percent vs. 12.5 percent). In a 3-year, 5-generation study on tumor resistant and tumor-susceptible strains of mice, zinc concentrations of 10 to 20 mg/L in drinking water resulted in increased frequencies of tumors from the F_0 to the F_4 generation in the resistant strain (from 0.8 to 25.7 percent vs. 0.0004 percent in the controls), and higher tumor frequencies in two susceptible strains (43.4 percent and 32.4 percent vs. 15 percent in the controls). Statistical analysis of the data was not reported. Hypertrophy of the adrenal cortex and pancreatic islets, but no corresponding tumors were reported in C3H mice given drinking water containing 500 mg/L zinc sulfate for 14 months (studies cited in USEPA, 2000).

USEPA has given zinc a carcinogenicity weight-of-evidence classification of D, not classifiable as to human carcinogenicity, based on inadequate evidence in humans and laboratory animals.

Exposure Route Considerations

Ingestion

Zinc is essential for human beings, with the daily requirement recommended at 15 mg for adults and up to 19 mg for nursing mothers (NRC, 1979 as cited in ATSDR, 1994). Reports of toxic effects following ingestion of moderate amounts of zinc are uncommon due to an efficient homeostatic mechanism that regulates zinc levels in the body.

Zinc is usually present in tap water at concentrations below 0.2 mg/l although drinking water in galvanized pipes can contain up to 2 to 5 mg/l (NLM/HSDB, 1999). Typically, concentrations are much less than the secondary Maximum Contaminant Level (MCL) of 5 mg/L. This value is based on the threshold for metallic taste in water. Zinc levels in foods such as meat, fish, and poultry average 24.5 mg/kg, and grains and potatoes contain 8 and 6 mg zinc/kg, respectively. An estimate of daily intake of zinc for the adult U.S. population in food is 10 to 20 mg/day (ATSDR 1994).

Zinc interacts with other trace metals, and has a protective effect against toxicity from exposure to lead and cadmium (NAS, 1977). Excessive dietary zinc produces a copper deficiency in laboratory animals. Similar findings have been observed in humans receiving long-term treatment with zinc (ATSDR, 1994).

Inhalation

The Threshold Limit Values (TLV), 8-hour time weighted averages for zinc compounds in workroom air, are 1 mg/m^3 for zinc chloride fumes and 5 mg/m^{3}for zinc oxide fumes (ACGIH 1999). Inhalation of elevated concentrations of zinc oxide fumes can produce metal fume fever. Zinc chloride particulate in air is more damaging than zinc oxide to the mucous membranes of the nasopharynx and respiratory tract upon contact, because zinc chloride is a corrosive (i.e. acid) salt.

Dermal

Occupational exposure to zinc oxide dust, combined with clogging of glands by dust, perspiration, and bacteria, has produced dermatitis (ATSDR, 1994).

Sensitive Populations and Indicators of Exposure

No specific data regarding human subpopulations that are unusually susceptible to the toxic effects of zinc were located (ATSDR, 1994). People who are malnourished or have a marginal copper status may be more susceptible to the effects of excessive zinc than people who are adequately nourished (Underwood 1977, cited in ATSDR, 1994).

Zinc is found in all human tissues and all body fluids and is essential for growth, development and reproduction. The total zinc content of the human body (70 kg) is about 2,300 mg (NLM/HSDB, 1999). Approximately 20 to 30 percent of ingested zinc is absorbed. It is principally excreted in the feces, though 300 to 600 µg/day is excreted in the urine (Goyer, 1991; ATSDR, 1994).

Toxicity Factors Derived for Risk Assessment

The oral reference dose (RfD) is based on a clinical study that investigated the effects of oral zinc supplements on copper and iron balance. A 10-week study of zinc supplementation in 18 healthy women given zinc gluconate supplements twice daily (50 mg zinc/day, or 1.0 mg/kg-day) resulted in a decrease of erythrocyte superoxide dismutase activity. There was a general decline in the mean serum high-density lipoprotein (HDL)-cholesterol in a higher-dose group (receiving 75 mg/day). USEPA (2000) reported that while it is not absolutely certain that the 50-mg zinc/day supplement (1.0 mg/kg-day) represents a clearly biologically significant endpoint, this level, when viewed collectively with other studies investigating effects on HDL-cholesterol, may signify the beginning of the dose-response trend. The significance of this change is unknown in light of an absence of increase in low-density lipoproteins (LDLs). The 1.0 mg/kg-day level was identified as LOAEL for zinc effects. An uncertainty factor of 3 was used, based on a minimal LOAEL from a moderate-duration study of the most sensitive humans and consideration of a substance that is an essential dietary nutrient. The oral RfD for zinc is 0.3 mg/kg-day (USEPA, 2000).

A RfC or inhalation RfD has not been developed for zinc (USEPA, 2000).

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APPENDIX I

Graphs and Statistical Analysis of House Dust Concentrations

Appendix I

Graphs and Statistical Analysis of House Dust Concentrations

The graphs and statistical summaries in this appendix provide a comparison of indoor concentrations of metals in house dust collected from vacuum bags and floor mats with respect to outdoor metal concentrations in yard soil. The scatter plots of house dust versus soil indicate no clear relationship between concentrations in house dust and yard soil, i.e., as concentrations in soil go up, concentrations in dust do not necessarily increase and vice versa. The results of the paired t-test of the log transformed data reveal that concentrations of antimony, arsenic, cadmium, and zinc all showed a statistically significant enrichment in floor mat dust when compared to outdoor soil. All but arsenic also showed an enrichment, though not as great, in vacuum bag dust.

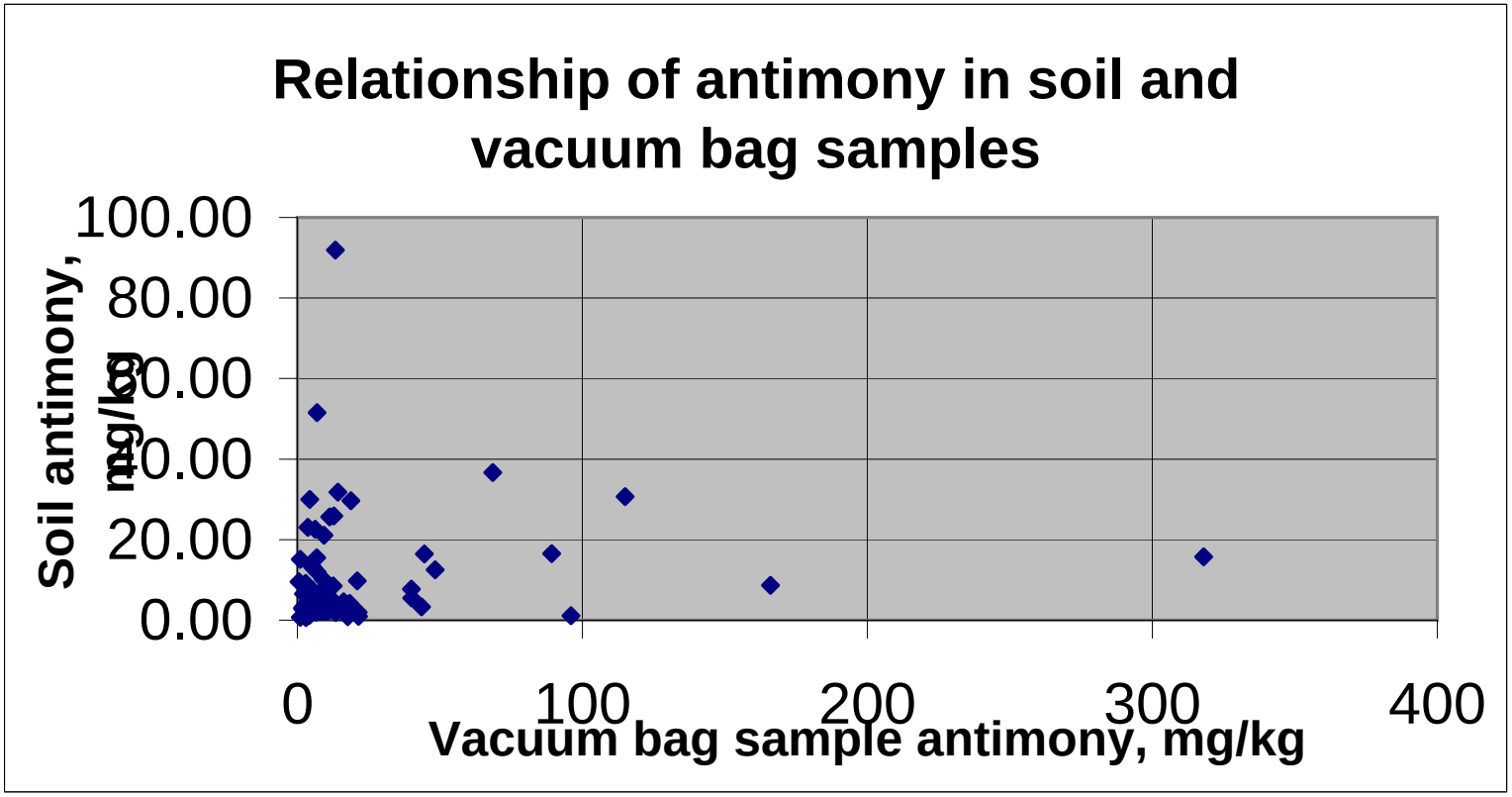
t-Test: Paired Two Sample for Means

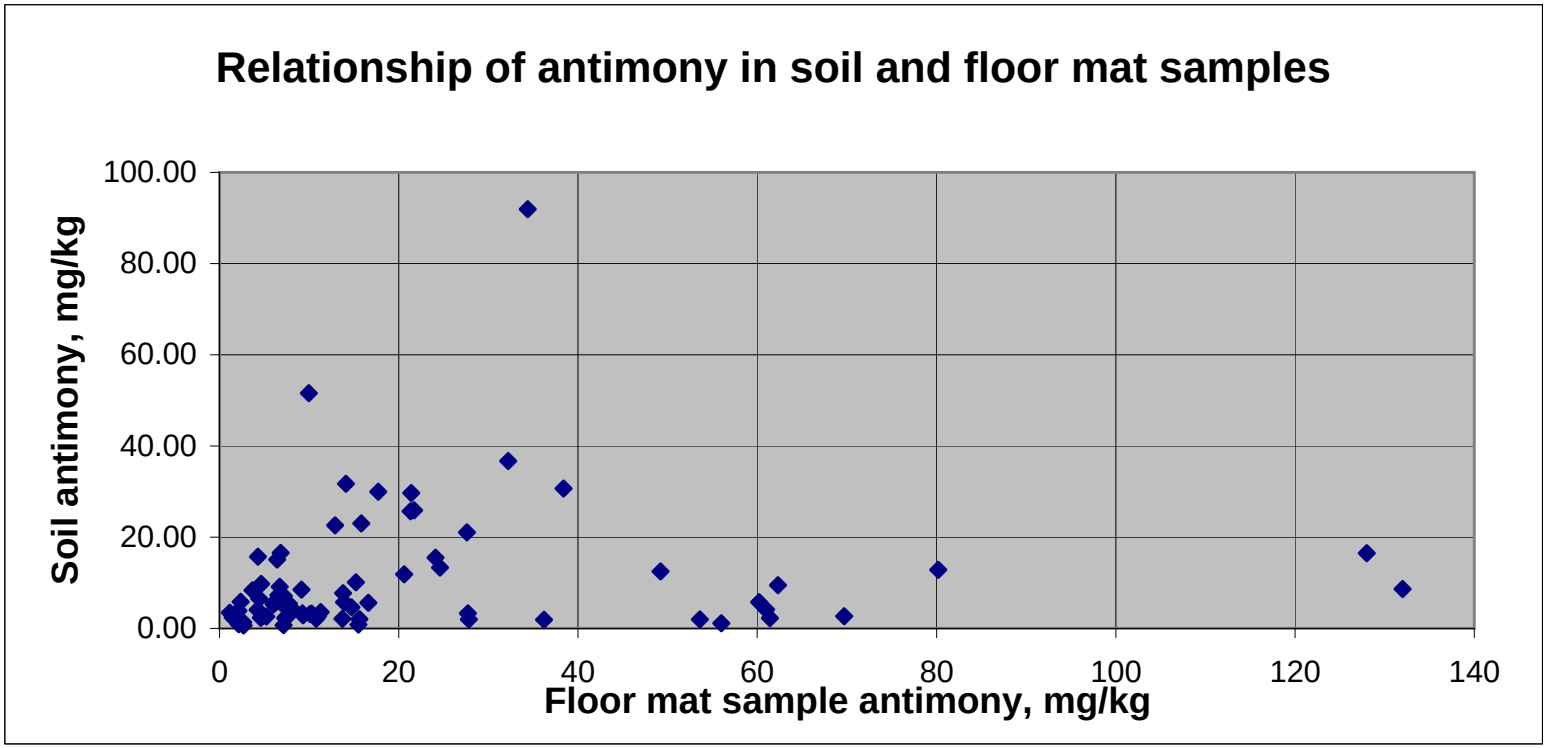
Antimony

	ln Mat	*ln Surface Soil*
Mean	2.518513	1.806210457
Variance	1.228555	1.150192781
Observations	84	84
Pearson Correlation	0.336065	
Hypothesized Mean Difference	0	
df	83	
t Stat	5.194065	
P(T<=t) one-tail	7.21E-07	
t Critical one-tail	1.66342	
P(T<=t) two-tail	1.44E-06	
t Critical two-tail	1.98896	

	ln Vacuum	*ln Surface Soil*
Mean	2.221587	1.745511778
Variance	1.365775	1.169785913
Observations	75	75
Pearson Correlation	0.212224	
Hypothesized Mean Difference	0	
df	74	
t Stat	2.916039	
P(T<=t) one-tail	0.002345	
t Critical one-tail	1.665708	
P(T<=t) two-tail	0.004689	
t Critical two-tail	1.992544	

	ln Mat	*ln Vacuum*
Mean	2.500864	2.221587468
Variance	1.128531	1.365774845
Observations	75	75
Pearson Correlation	0.339615	
Hypothesized Mean Difference	0	
df	74	
t Stat	1.882288	
P(T<=t) one-tail	0.031864	
t Critical one-tail	1.665708	
P(T<=t) two-tail	0.063728	
t Critical two-tail	1.992544	


Relationship of antimony in soil and vacuum bag samples
Soil antimony, mg/kg
100.00
80.00
60.00
40.00
20.00
0.00
0
100
200
300
400
Vacuum bag sample antimony, mg/kg


Relationship of antimony in soil and floor mat samples
Soil antimony, mg/kg
100.00
80.00
60.00
40.00
20.00
0.00
0
20
40
60
80
100
120
140
Floor mat sample antimony, mg/kg

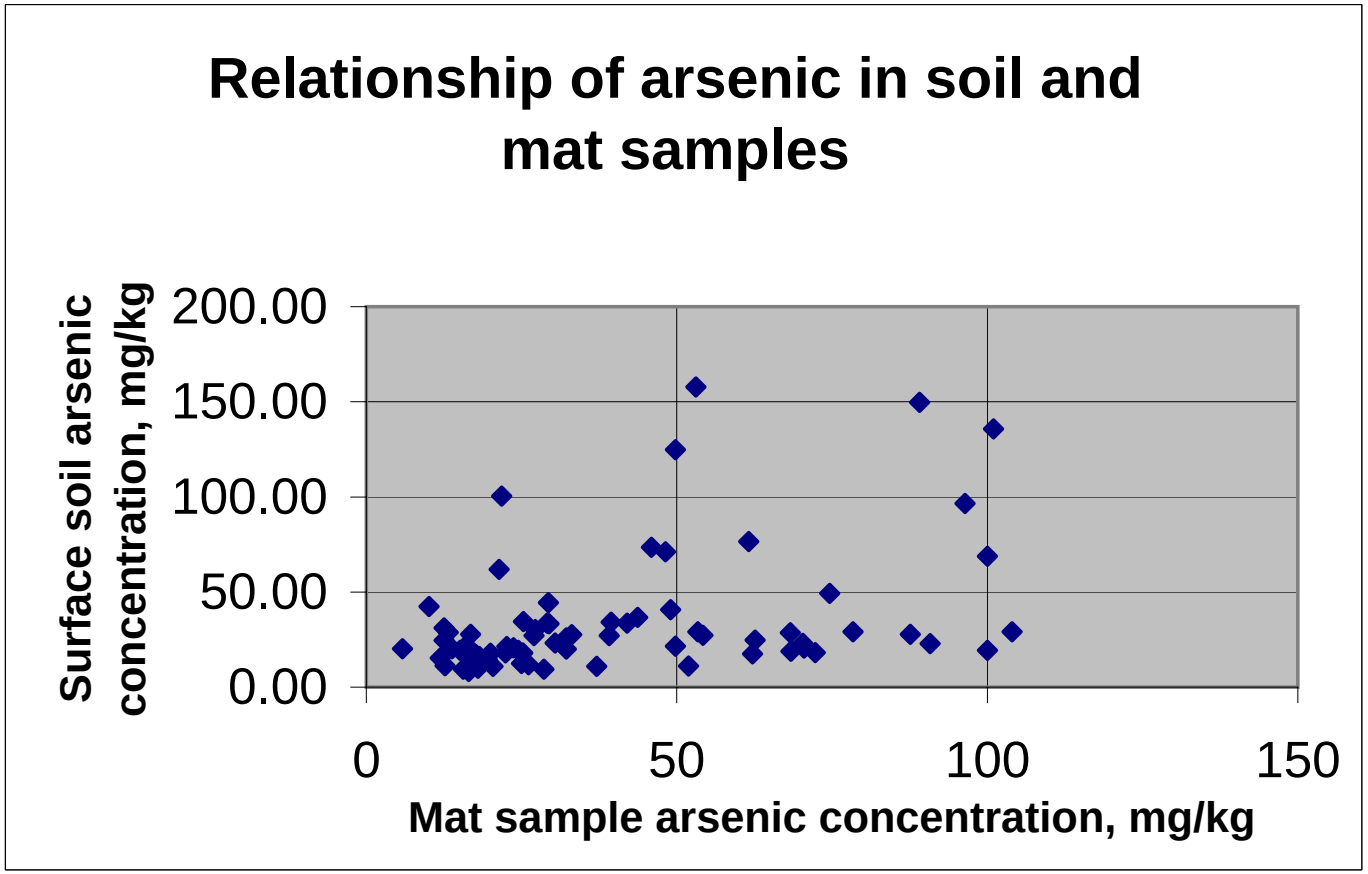
t-Test: Paired Two Sample for Means

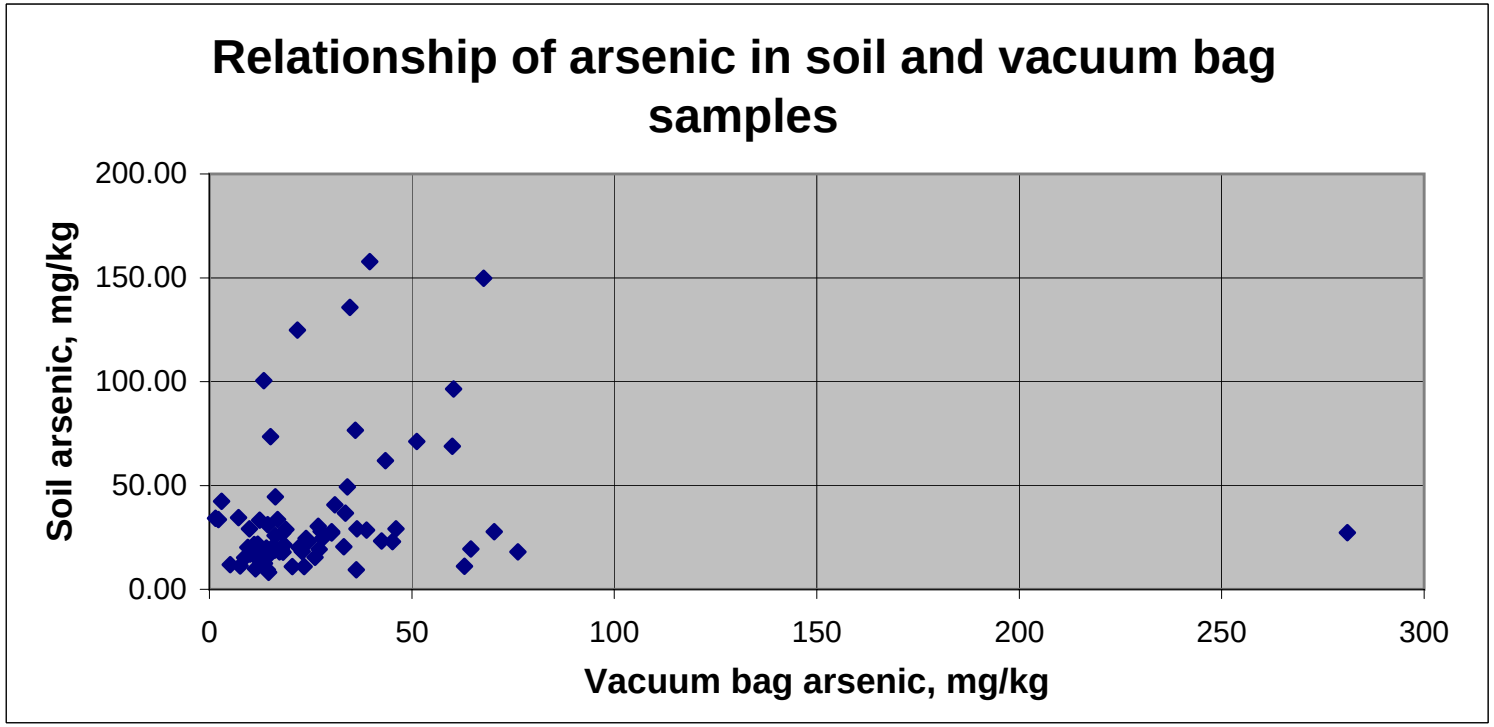
Arsenic

	Mat	*Surface Soil*
Mean	3.493219	3.305739964
Variance	0.454322	0.439358813
Observations	83	83
Pearson Correlation	0.425679	
Hypothesized Mean Difference	0	
df	82	
t Stat	2.38397	
P(T<=t) one-tail	0.009718	
t Critical one-tail	1.663648	
P(T<=t) two-tail	0.019437	
t Critical two-tail	1.98932	

	Vacuum	*Surface Soil*
Mean	3.030311	3.278075442
Variance	0.671703	0.457555357
Observations	74	74
Pearson Correlation	0.231728	
Hypothesized Mean Difference	0	
df	73	
t Stat	-2.282	
P(T<=t) one-tail	0.012703	
t Critical one-tail	1.665996	
P(T<=t) two-tail	0.025406	
t Critical two-tail	1.992998	

	Mat	*Vacuum*
Mean	3.482601	3.030310991
Variance	0.454625	0.671702585
Observations	74	74
Pearson Correlation	0.447162	
Hypothesized Mean Difference	0	
df	73	
t Stat	4.893658	
P(T<=t) one-tail	2.87E-06	
t Critical one-tail	1.665996	
P(T<=t) two-tail	5.74E-06	
t Critical two-tail	1.992998	


Relationship of arsenic in soil and mat samples
Surface soil arsenic concentration, mg/kg
200.00
150.00
100.00
50.00
0.00
0
50
100
150
Mat sample arsenic concentration, mg/kg


Relationship of arsenic in soil and vacuum bag samples
Soil arsenic, mg/kg
200.00
150.00
100.00
50.00
0.00
0
50
100
150
200
250
300
Vacuum bag arsenic, mg/kg

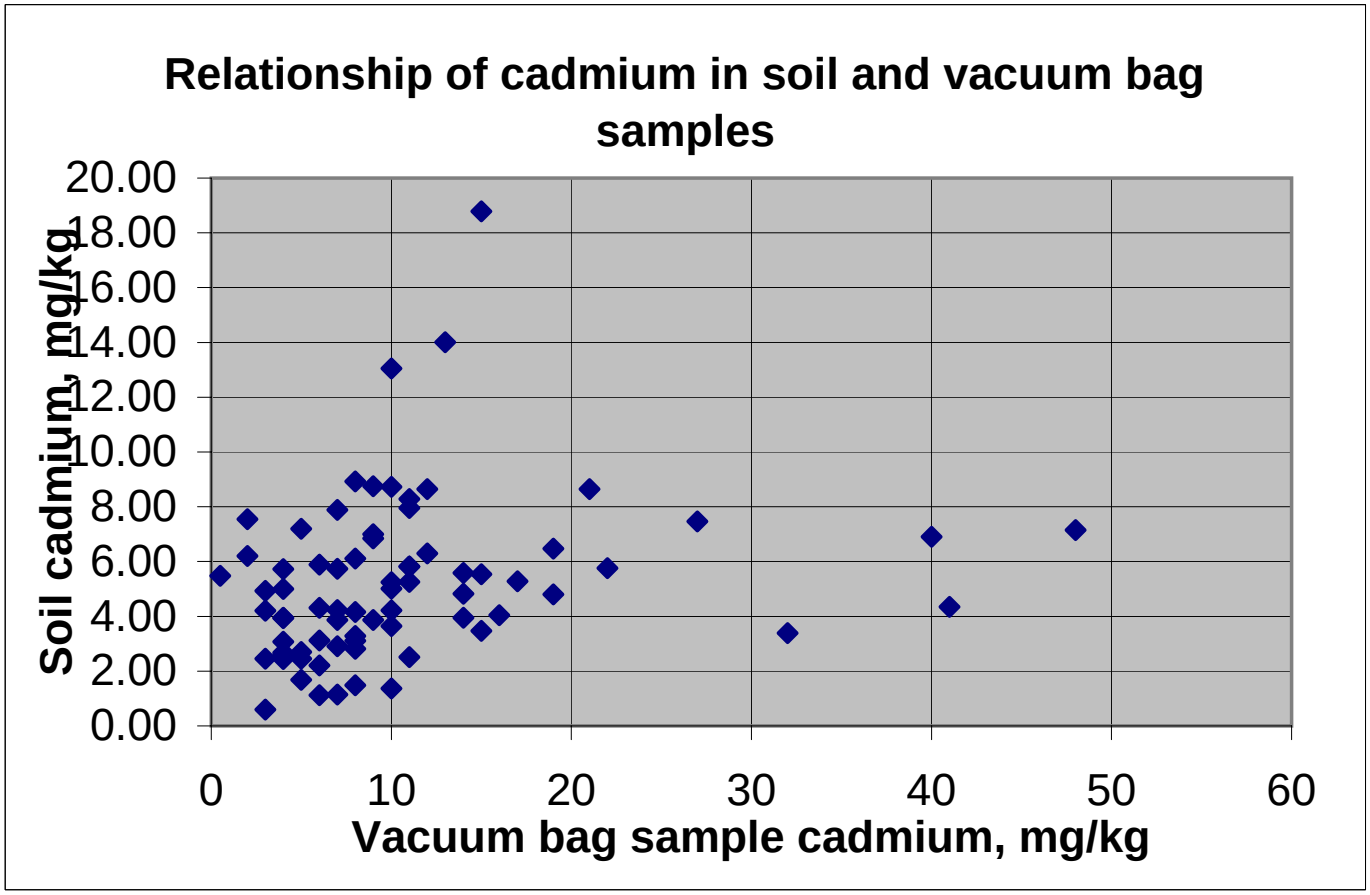
t-Test: Paired Two Sample for Means

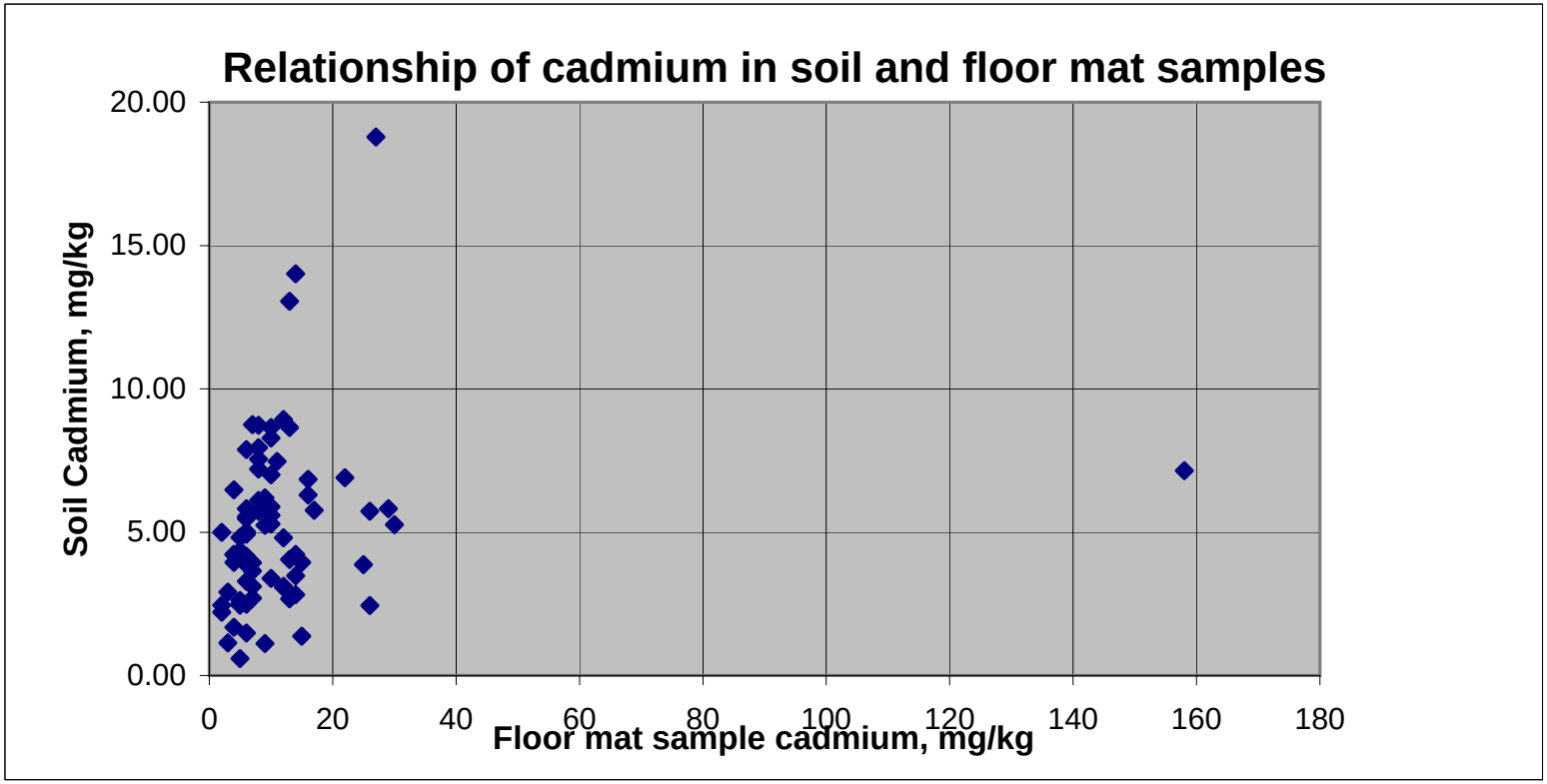
Cadmium

	ln Mat	*ln Surface Soil*
Mean	2.159283	1.535999983
Variance	0.475045	0.368972724
Observations	83	83
Pearson Correlation	0.349041	
Hypothesized Mean Difference	0	
df	82	
t Stat	7.644527	
P(T<=t) one-tail	1.76E-11	
t Critical one-tail	1.663648	
P(T<=t) two-tail	3.52E-11	
t Critical two-tail	1.98932	

	ln Vacuum	*ln Surface Soil*
Mean	2.103044	1.486563745
Variance	0.555945	0.353927022
Observations	75	75
Pearson Correlation	0.29902	
Hypothesized Mean Difference	0	
df	74	
t Stat	6.649771	
P(T<=t) one-tail	2.19E-09	
t Critical one-tail	1.665708	
P(T<=t) two-tail	4.39E-09	
t Critical two-tail	1.992544	

	ln Mat	*ln Vacuum*
Mean	2.160008	2.103043564
Variance	0.495938	0.555945433
Observations	75	75
Pearson Correlation	0.412523	
Hypothesized Mean Difference	0	
df	74	
t Stat	0.627202	
P(T<=t) one-tail	0.266229	
t Critical one-tail	1.665708	
P(T<=t) two-tail	0.532458	
t Critical two-tail	1.992544	


Relationship of cadmium in soil and vacuum bag samples
Soil cadmium, mg/kg
20.00
18.00
16.00
14.00
12.00
10.00
8.00
6.00
4.00
2.00
0.00
0
10
20
30
40
50
60
Vacuum bag sample cadmium, mg/kg


Relationship of cadmium in soil and floor mat samples
Soil Cadmium, mg/kg
20.00
15.00
10.00
5.00
0.00
0
20
40
60
80
100
120
140
160
180
Floor mat sample cadmium, mg/kg

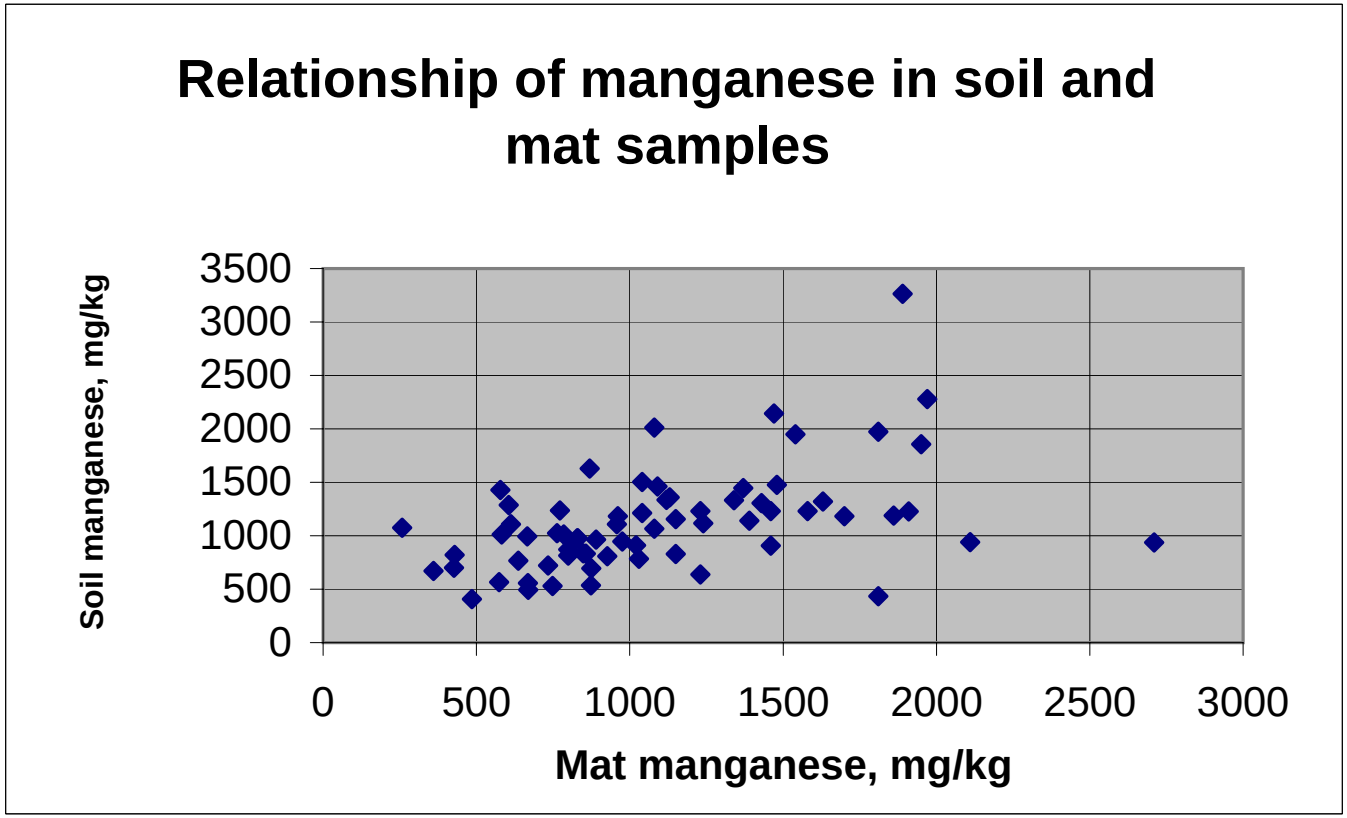
t-Test: Paired Two Sample for Means

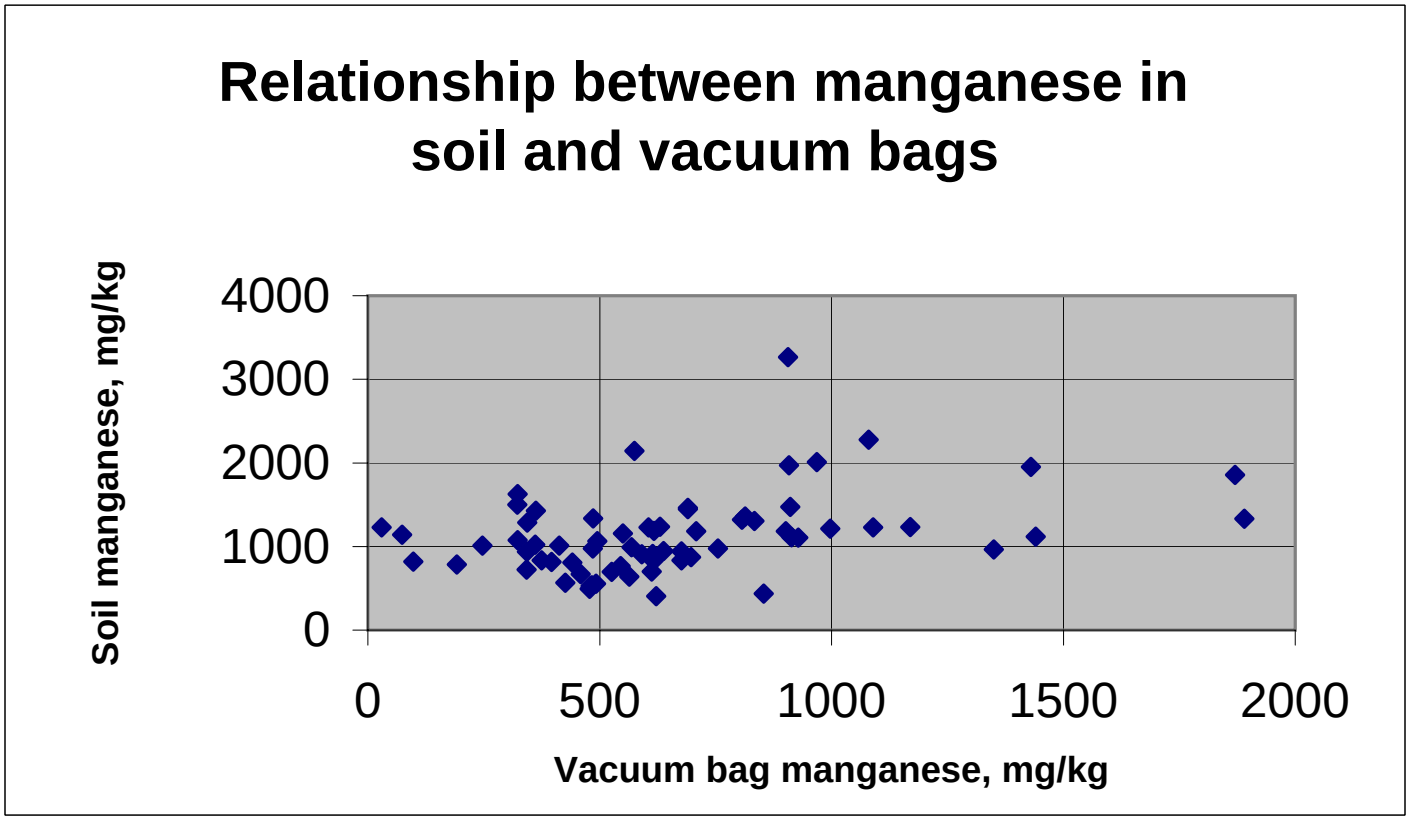
Manganese

	Mat	*Surface Soil*
Mean	6.947894	6.979135489
Variance	0.241356	0.173828249
Observations	75	75
Pearson Correlation	0.553579	
Hypothesized Mean Difference	0	
df	74	
t Stat	-0.62332	
P(T<=t) one-tail	0.267494	
t Critical one-tail	1.665708	
P(T<=t) two-tail	0.534988	
t Critical two-tail	1.992544	

	Vacuum	*Surface Soil*
Mean	6.325727	6.951868085
Variance	0.451097	0.160386613
Observations	68	68
Pearson Correlation	0.237306	
Hypothesized Mean Difference	0	
df	67	
t Stat	-7.42307	
P(T<=t) one-tail	1.32E-10	
t Critical one-tail	1.667916	
P(T<=t) two-tail	2.64E-10	
t Critical two-tail	1.996009	

	Mat	*Vacuum*
Mean	6.909371	6.325727424
Variance	0.210866	0.451096564
Observations	68	68
Pearson Correlation	0.251402	
Hypothesized Mean Difference	0	
df	67	
t Stat	6.759979	
P(T<=t) one-tail	2.03E-09	
t Critical one-tail	1.667916	
P(T<=t) two-tail	4.06E-09	
t Critical two-tail	1.996009	


Relationship of manganese in soil and mat samples
Soil manganese, mg/kg
3500
3000
2500
2000
1500
1000
500
0
0
500
1000
1500
2000
2500
3000
Mat manganese, mg/kg


Relationship between manganese in soil and vacuum bags
Soil manganese, mg/kg
4000
3000
2000
1000
0
0
500
1000
1500
2000
Vacuum bag manganese, mg/kg

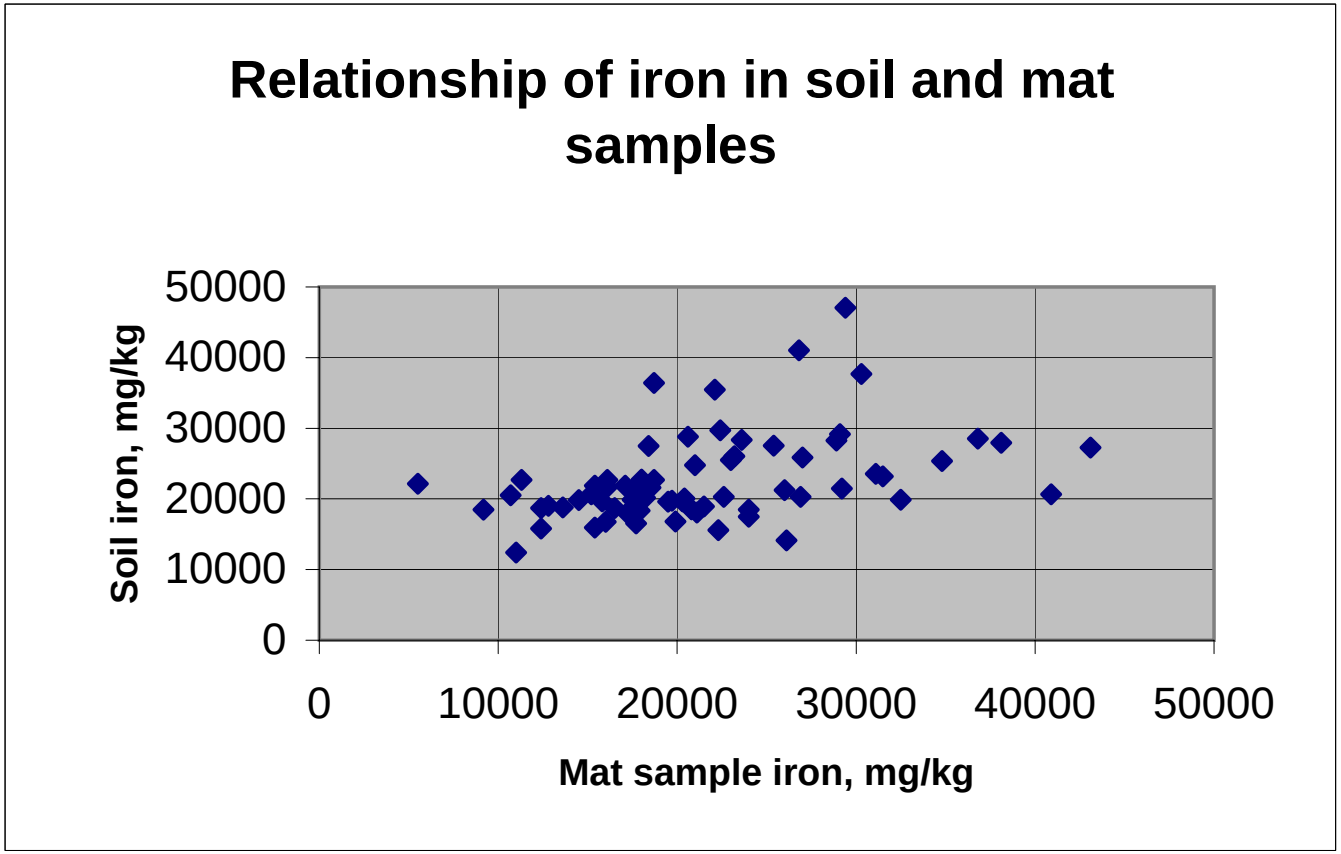
t-Test: Paired Two Sample for Means

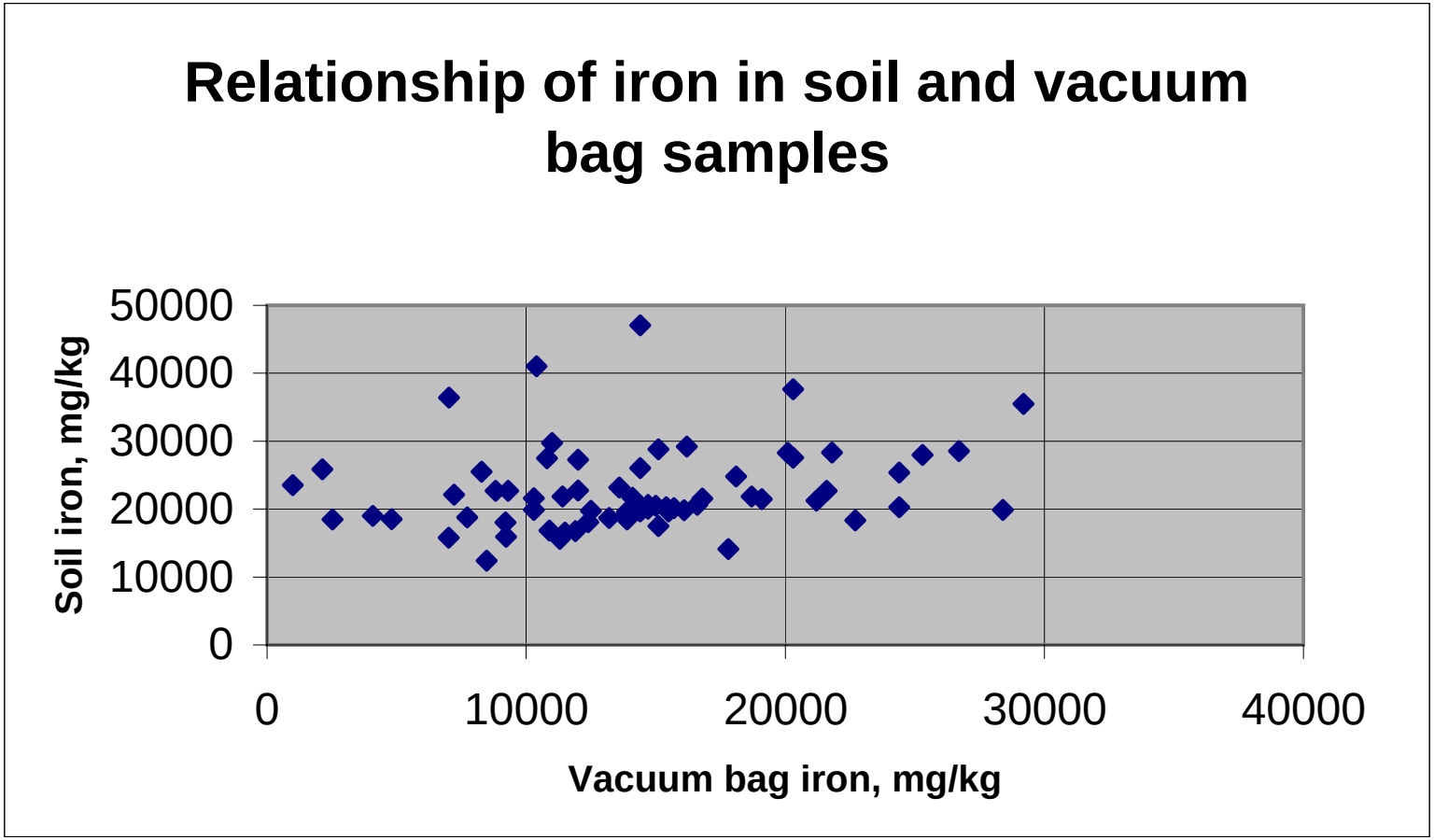
Iron

	Mat	*Surface Soil*
Mean	9.958137	10.0186782
Variance	0.161813	0.070137615
Observations	75	75
Pearson Correlation	0.488109	
Hypothesized Mean Difference	0	
df	74	
t Stat	-1.46574	
P(T<=t) one-tail	0.073478	
t Critical one-tail	1.665708	
P(T<=t) two-tail	0.146955	
t Critical two-tail	1.992544	

	Vacuum	*Surface Soil*
Mean	9.430674	9.999384489
Variance	0.343363	0.061023392
Observations	68	68
Pearson Correlation	0.150917	
Hypothesized Mean Difference	0	
df	67	
t Stat	-7.80864	
P(T<=t) one-tail	2.66E-11	
t Critical one-tail	1.667916	
P(T<=t) two-tail	5.33E-11	
t Critical two-tail	1.996009	

	Mat	*Vacuum*
Mean	9.912493	9.43067417
Variance	0.135422	0.343362888
Observations	68	68
Pearson Correlation	0.277748	
Hypothesized Mean Difference	0	
df	67	
t Stat	6.631181	
P(T<=t) one-tail	3.44E-09	
t Critical one-tail	1.667916	
P(T<=t) two-tail	6.89E-09	
t Critical two-tail	1.996009	


Relationship of iron in soil and mat samples
Soil iron, mg/kg
50000
40000
30000
20000
10000
0
0
10000
20000
30000
40000
50000
Mat sample iron, mg/kg


Relationship of iron in soil and vacuum bag samples
Soil iron, mg/kg
50000
40000
30000
20000
10000
0
0
10000
20000
30000
40000
Vacuum bag iron, mg/kg

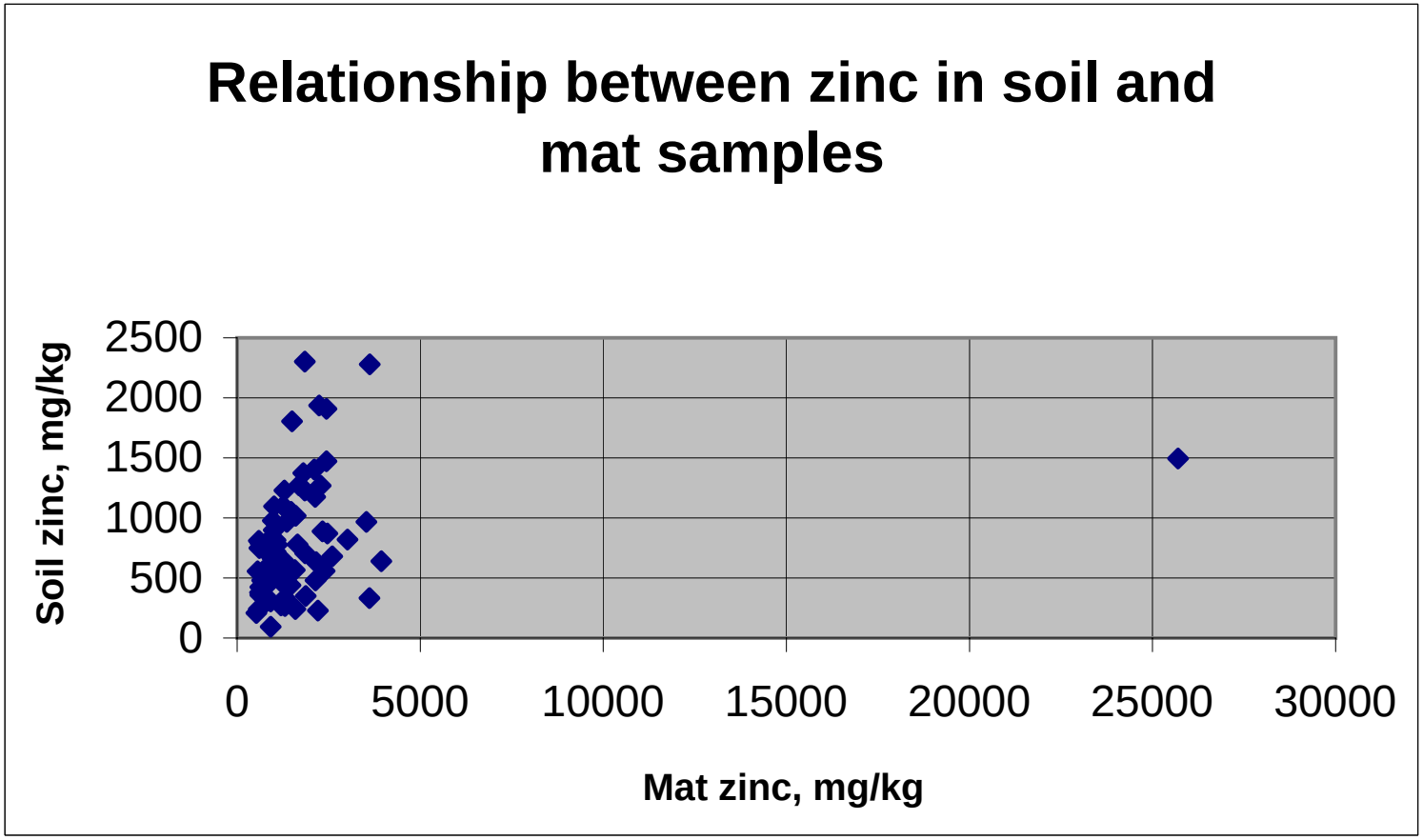
t-Test: Paired Two Sample for Means

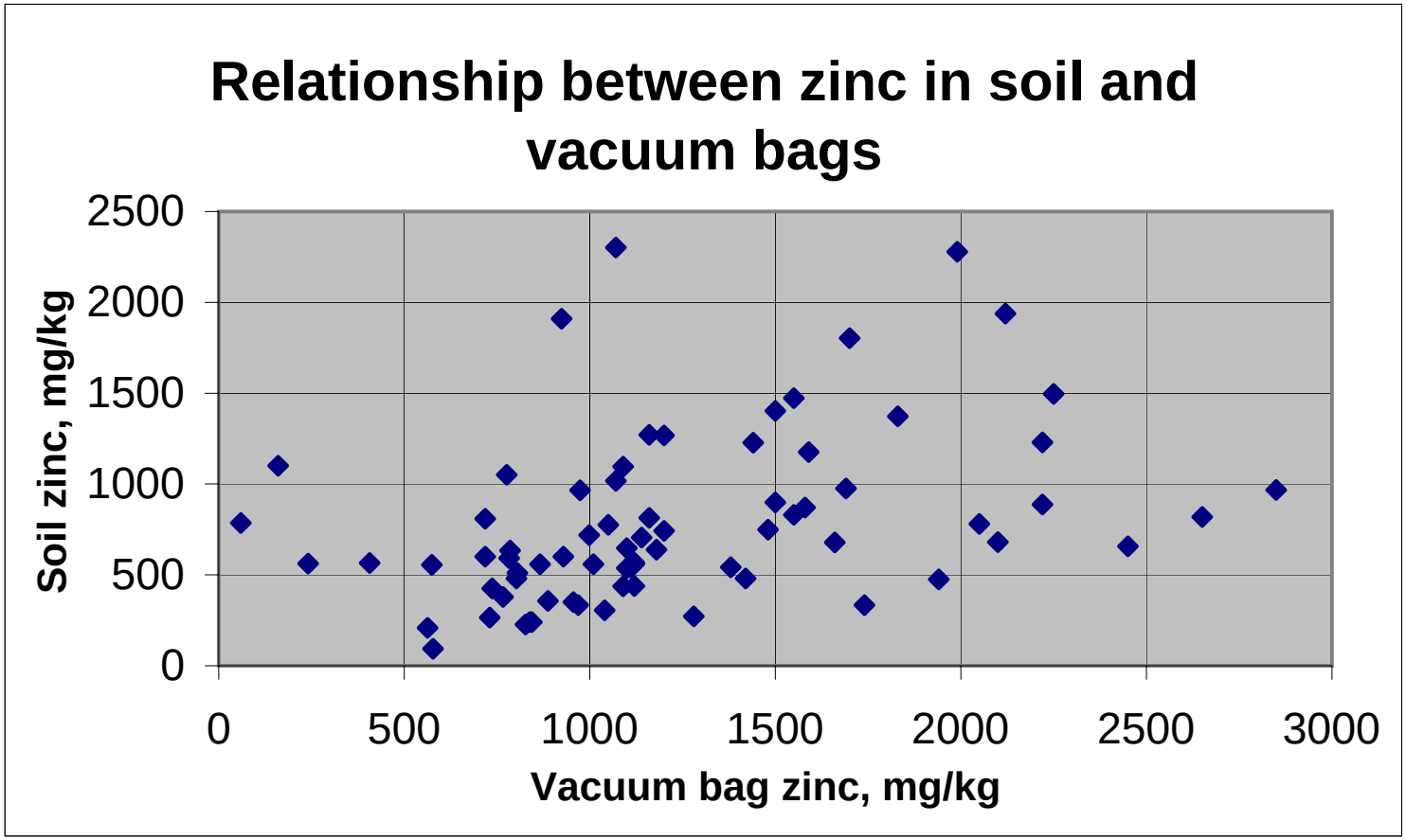
Zinc

	Mat	*Surface Soil*
Mean	7.23734	6.547255113
Variance	0.365593	0.37256025
Observations	82	82
Pearson Correlation	0.376804	
Hypothesized Mean Difference	0	
df	81	
t Stat	9.213368	
P(T<=t) one-tail	1.5E-14	
t Critical one-tail	1.663884	
P(T<=t) two-tail	3.01E-14	
t Critical two-tail	1.989688	

	Vacuum	*Surface Soil*
Mean	6.981535	6.506867644
Variance	0.360555	0.362876917
Observations	74	74
Pearson Correlation	0.291337	
Hypothesized Mean Difference	0	
df	73	
t Stat	5.702765	
P(T<=t) one-tail	1.18E-07	
t Critical one-tail	1.665996	
P(T<=t) two-tail	2.35E-07	
t Critical two-tail	1.992998	

	Mat	*Vacuum*
Mean	7.230723	6.981534516
Variance	0.373734	0.360555141
Observations	74	74
Pearson Correlation	0.409097	
Hypothesized Mean Difference	0	
df	73	
t Stat	3.25408	
P(T<=t) one-tail	0.000862	
t Critical one-tail	1.665996	
P(T<=t) two-tail	0.001725	
t Critical two-tail	1.992998	


Relationship between zinc in soil and mat samples
Soil zinc, mg/kg
2500
2000
1500
1000
500
0
0
5000
10000
15000
20000
25000
30000
Mat zinc, mg/kg


Relationship between zinc in soil and vacuum bags
Soil zinc, mg/kg
2500
2000
1500
1000
500
0
0
500
1000
1500
2000
2500
3000
Vacuum bag zinc, mg/kg

APPENDIX J

Potential Dose From Consumption of Beef

Appendix J

Potential Dose From Consumption of Beef

This appendix includes the equations used to estimate the concentrations of metals in edible beef tissue and the potential dose from ingestion of beef.

Conservative Estimation of Metal Concentrations in Edible Beef Tissue

All equations and values were collected from CEPA 1996, U.S. EPA 1990c, and U.S. EPA 1998c, or assumed as noted.

$$\mathbf{C_{beef} = [(F_f * Q_{pi} * P_i) + (Q_s * C_s * B_s)] * Ba_{beef} * MF}$$

Where:

C_{beef}	=	concentration in edible beef tissue
F_f	=	fraction of plants grown on contaminated soil and ingested, assume 100% or 1
Qp_i	=	quantity of forage eaten by cattle per day, 8.8 kg DW/d
P_i	=	COPC concentration in forage eaten by cattle, see below for calculation
Q_s	=	quantity of soil eaten by animal per day, 0.5 kg/day
C_s	=	average soil concentration over the exposure duration, assume 2 mg arsenic/kg soil or 1 mg antimony or cadmium/kg soil
B_s	=	soil bioavailability (unitless), assume 100% or 1
Ba_{beef}	=	COPC biotransfer factor for beef, 0.001, 0.002, and 0.00055 day/kg FW tissue for Sb, As, and Cd, respectively.
MF	=	metabolism factor, 1 is recommended for all metals

$$\mathbf{P_i = C_s * Br_{forage}}$$

Where:

Br_{forage}	=	ratio of the concentration in forage tissue to the concentration in soil, 0.2, 0.036, and 0.364 (mg/kg DW plant)/(mg/kg soil) for antimony, arsenic, and cadmium, respectivley.

$$\mathbf{Dose\ from\ beef\ ingestion = (IR_{beef} * C_{beef})/BW}$$

Where:

IR_{beef}	=	adult ingestion rate of beef, for purposes of this conservative estimate, the consumption rate of all meat was used and we assumed that all meat ingested was contaminated beef, 0.250 kg meat per day (maximum meat ingestion rate, adult males over 20).
BW	=	adult body weight, assumed 70 kg

APPENDIX K

Summary Statistics for Antimony, Arsenic, Cadmium, Iron, Manganese, and Zinc for the Fall 1999 Coeur d'Alene Basin Sampling Event

Appendix K

Summary Statistics for Antimony, Arsenic, Cadmium, Iron, Manganese, and Zinc for the Fall 1999 Coeur d'Alene Basin Sampling Event

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This appendix contains summary tables for the Fall 1999 sampling event for the Coeur d'Alene Basin residential areas. Metals included in these tables are antimony, arsenic, cadmium, iron, manganese, and zinc. Sample media from this event includes yard soil, vacuum dust, right-of-ways, play areas, garden soil, driveway, and initial and purged drinking water.

Table K-2.1 Yard Soil Arsenic Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	15.00	5.00	9.00	5.00	23.00	4.00	13.00	12.00	86.00
Minimum	5.20	8.20	8.30	11.20	8.90	31.50	5.80	9.90	5.20
Maximum	71.80	138.00	64.20	46.60	47.80	66.70	55.30	25.60	138.00
Arithmetic Mean	15.29	35.98	19.11	25.80	20.63	43.08	20.63	16.82	21.25
Standard	15.96	57.07	17.76	17.89	9.15	16.02	13.65	4.01	18.58
Geometric Mean	12.18	17.29	15.03	21.14	18.91	41.19	17.22	16.39	17.16
Geometric Standard	1.80	3.24	1.93	2.02	1.53	1.39	1.87	1.27	1.83
B Horizon									
Number	15.00	5.00	8.00	5.00	24.00	4.00	12.00	12.00	85.00
Minimum	4.50	8.70	8.30	11.40	5.00	21.00	4.20	11.20	4.20
Maximum	32.40	96.70	76.50	41.20	30.50	89.70	24.50	53.70	96.70
Arithmetic Mean	14.32	27.02	21.58	23.28	16.63	46.00	16.53	20.72	19.63
Standard	7.45	38.96	23.27	13.61	7.33	30.15	7.04	11.38	15.90
Geometric Mean	12.67	15.21	15.58	20.32	14.76	39.82	14.59	18.84	15.96
Geometric Standard	1.68	2.82	2.16	1.78	1.71	1.83	1.79	1.52	1.85
C Horizon									
Number	14.00	5.00	8.00	3.00	24.00	4.00	13.00	10.00	81.00
Minimum	5.40	4.80	4.90	11.90	3.80	14.10	4.20	1.10	1.10
Maximum	16.30	59.40	67.00	51.30	26.30	75.90	34.00	39.80	75.90
Arithmetic Mean	9.46	17.86	16.98	26.73	13.73	36.15	17.32	17.31	16.18
Standard	3.82	23.32	20.74	21.43	6.15	27.22	9.65	10.74	13.37
Geometric Mean	8.80	10.96	11.37	21.81	12.25	29.88	14.80	13.00	12.64
Geometric Standard	1.48	2.68	2.35	2.14	1.68	2.00	1.84	2.69	2.02
D Horizon									
Number	12.00	4.00	8.00	3.00	22.00	4.00	12.00	8.00	73.00
Minimum	4.20	6.90	3.00	13.20	5.30	11.80	2.40	6.70	2.40
Maximum	12.90	18.10	21.00	45.80	25.90	67.10	28.00	20.90	67.10
Arithmetic Mean	8.17	10.85	9.61	28.63	13.05	31.40	14.04	15.45	13.82
Standard	2.83	5.08	6.06	16.37	5.51	24.41	7.33	5.44	9.63
Geometric Mean	7.71	10.08	8.06	25.34	11.97	25.63	12.07	14.44	11.61
Geometric Standard	1.43	1.54	1.90	1.87	1.54	2.05	1.89	1.52	1.79

Table K-2.2 Vacuum Dust Arsenic Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Number	11.00	5.00	8.00	4.00	17.00	1.00	9.00	11.00	66.00
Minimum	7.10	9.10	14.50	8.10	6.20	31.30	10.70	2.50	2.50
Maximum	52.00	24.90	203.00	22.50	138.00	31.30	117.00	120.00	203.00
Arithmetic Mean	16.96	16.72	52.03	15.40	25.25	31.30	30.96	25.05	26.71
Standard	12.60	6.15	64.55	6.89	30.54		35.09	32.36	33.68
Geometric Mean	14.40	15.76	32.41	14.16	18.15	31.30	21.10	16.23	18.45
Geometric Standard	1.73	1.48	2.59	1.62	2.08		2.31	2.52	2.14

Table K-2.3 Right-of-ways Arsenic Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	5.00	3.00	2.00	1.00	3.00	1.00	1.00	1.00	17.00
Minimum	7.30	4.90	12.30	27.60	26.70	153.00	51.10	26.40	4.90
Maximum	54.40	11.60	33.70	27.60	201.00	153.00	51.10	26.40	201.00
Arithmetic Mean	22.18	8.27	23.00	27.60	86.23	153.00	51.10	26.40	41.09
Standard Deviation	19.86	3.35	15.13		99.41				53.85
Geometric Mean	16.47	7.78	20.36	27.60	55.00	153.00	51.10	26.40	23.64
Geometric Standard Deviation	2.32	1.54	2.04		3.08				2.80
B Horizon									
Number	5.00	3.00	2.00	1.00	3.00	1.00		1.00	16.00
Minimum	7.70	3.80	11.10	42.40	5.80	204.00		73.80	3.80
Maximum	904.00	11.80	35.30	42.40	56.90	204.00		73.80	904.00
Arithmetic Mean	213.42	8.37	23.20	42.40	31.47	204.00		73.80	97.08
Standard Deviation	386.83	4.12	17.11		25.55				220.65
Geometric Mean	60.66	7.52	19.79	42.40	21.87	204.00		73.80	31.45
Geometric Standard Deviation	5.65	1.82	2.27		3.27				4.08
C Horizon									
Number	5.00	3.00	2.00	1.00	2.00	1.00		1.00	15.00
Minimum	6.20	3.90	7.10	47.90	4.40	100.00		90.80	3.90
Maximum	74.00	13.20	25.50	47.90	44.10	100.00		90.80	100.00
Arithmetic Mean	38.60	9.97	16.30	47.90	24.25	100.00		90.80	36.18
Standard Deviation	28.75	5.26	13.01		28.07				32.38
Geometric Mean	28.10	8.70	13.46	47.90	13.93	100.00		90.80	22.37
Geometric Standard Deviation	2.70	2.00	2.47		5.10				3.02
D Horizon									
Number	4.00	3.00	1.00	1.00	1.00	1.00		1.00	12.00
Minimum	5.30	4.20	11.40	54.70	8.40	42.20		44.70	4.20
Maximum	38.90	14.10	11.40	54.70	8.40	42.20		44.70	54.70
Arithmetic Mean	25.48	9.63	11.40	54.70	8.40	42.20		44.70	24.35
Standard Deviation	16.17	5.02							18.01
Geometric Mean	19.80	8.56	11.40	54.70	8.40	42.20		44.70	17.71
Geometric Standard Deviation	2.55	1.88							2.43

Table K-2.7 Initial and Purged Drinking Water Arsenic Concentrations (ug/L) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Initial									
Number	15.00	5.00	9.00	4.00	22.00	2.00	13.00	12.00	82.00
Minimum	2.20	2.20	2.20	2.20	2.20	6.00	2.20	2.20	2.20
Maximum	6.20	6.00	6.00	6.00	6.00	12.90	6.50	7.30	12.90
Arithmetic Mean	3.73	3.72	3.90	4.10	4.30	9.45	4.58	2.98	4.08
Standard Deviation	1.94	2.08	1.99	2.19	1.91	4.88	1.96	1.74	2.15
Geometric Mean	3.29	3.29	3.45	3.63	3.84	8.80	4.10	2.68	3.57
Geometric Standard Deviation	1.67	1.73	1.69	1.78	1.65	1.72	1.67	1.53	1.68
Purged									
Number	16.00	5.00	8.00	4.00	24.00	2.00	13.00	12.00	84.00
Minimum	2.20	2.20	2.20	2.20	2.20	6.00	2.20	2.20	2.20
Maximum	6.00	6.00	6.00	6.00	6.00	23.20	6.00	6.00	23.20
Arithmetic Mean	3.39	4.48	3.95	4.35	3.63	14.60	4.25	3.47	4.03
Standard Deviation	1.82	2.08	1.91	1.95	1.88	12.16	1.97	1.87	2.82
Geometric Mean	3.01	4.02	3.53	3.99	3.20	11.80	3.78	3.07	3.44
Geometric Standard Deviation	1.62	1.73	1.67	1.64	1.64	2.60	1.68	1.64	1.71

Table K-2.4 Play Areas Arsenic Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	3.00	1.00	3.00	1.00	3.00		1.00	2.00	14.00
Minimum	10.40	183.00	8.10	49.50	4.60		59.10	7.30	4.60
Maximum	30.50	183.00	60.80	49.50	162.00		59.10	36.40	183.00
Arithmetic Mean	17.87	183.00	28.10	49.50	57.47		59.10	21.85	46.11
Standard Deviation	11.00		28.55		90.53			20.58	57.20
Geometric Mean	15.91	183.00	19.65	49.50	16.29		59.10	16.30	23.81
Geometric Standard Deviation	1.77		2.80		7.34			3.11	3.33
B Horizon									
Number	3.00	1.00	3.00	1.00	4.00		1.00	1.00	14.00
Minimum	9.60	206.00	5.30	51.10	6.40		22.60	50.30	5.30
Maximum	38.30	206.00	69.70	51.10	67.40		22.60	50.30	206.00
Arithmetic Mean	21.63	206.00	30.40	51.10	29.45		22.60	50.30	43.14
Standard Deviation	14.90		34.47		28.79				51.88
Geometric Mean	18.42	206.00	18.16	51.10	18.68		22.60	50.30	25.69
Geometric Standard Deviation	2.00		3.64		3.17				2.89
C Horizon									
Number	3.00	1.00	3.00		3.00		1.00		11.00
Minimum	6.90	165.00	4.50		5.70		14.80		4.50
Maximum	15.80	165.00	38.00		21.90		14.80		165.00
Arithmetic Mean	11.53	165.00	17.00		12.70		14.80		27.59
Standard Deviation	4.46		18.30		8.32				46.55
Geometric Mean	10.91	165.00	11.33		10.94		14.80		14.52
Geometric Standard Deviation	1.52		2.99		1.96				2.75
D Horizon									
Number	2.00	1.00	3.00		2.00				8.00
Minimum	5.60	160.00	3.40		4.10				3.40
Maximum	13.70	160.00	25.30		18.80				160.00
Arithmetic Mean	9.65	160.00	12.00		11.45				29.78
Standard Deviation	5.73		11.68		10.39				53.19
Geometric Mean	8.76	160.00	8.56		8.78				12.50
Geometric Standard Deviation	1.88		2.75		2.94				3.51

Table K-2.5 Garden Arsenic Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Total
F Horizon								
Number	7.00	2.00	1.00	2.00	9.00	1.00	3.00	25.00
Minimum	4.70	6.30	28.50	13.40	5.30	33.70	3.90	3.90
Maximum	20.30	9.30	28.50	14.90	25.50	33.70	6.90	33.70
Arithmetic Mean	11.49	7.80	28.50	14.15	15.69	33.70	5.80	13.80
Standard Deviation	5.00	2.12		1.06	6.12		1.65	7.66
Geometric Mean	10.53	7.65	28.50	14.13	14.46	33.70	5.62	11.91
Geometric Standard Deviation	1.59	1.32		1.08	1.58		1.37	1.76
G Horizon								
Number	7.00	2.00		1.00	9.00	1.00	3.00	23.00
Minimum	4.50	6.80		75.00	5.40	14.10	4.10	4.10
Maximum	13.00	9.10		75.00	25.80	14.10	7.80	75.00
Arithmetic Mean	8.59	7.95		75.00	12.78	14.10	6.37	13.01
Standard Deviation	2.83	1.63			5.85		1.99	14.30
Geometric Mean	8.16	7.87		75.00	11.75	14.10	6.13	10.19
Geometric Standard Deviation	1.42	1.23			1.54		1.42	1.83

Table K-2.6 Driveway Arsenic Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	13.00	5.00	4.00	2.00	14.00	2.00	6.00	3.00	49.00
Minimum	2.50	4.10	15.90	52.20	4.70	33.70	18.00	11.20	2.50
Maximum	115.00	55.30	56.80	57.90	951.00	52.70	747.00	69.20	951.00
Arithmetic Mean	32.58	18.44	34.40	55.05	114.26	43.20	148.40	47.33	71.06
Standard Deviation	32.04	21.05	17.83	4.03	243.49	13.44	293.44	31.52	166.00
Geometric Mean	19.04	12.05	30.84	54.98	43.93	42.14	46.90	36.28	30.07
Geometric Standard Deviation	3.39	2.67	1.74	1.08	3.73	1.37	4.05	2.77	3.30
B Horizon									
Number	12.00	5.00	4.00	2.00	13.00	2.00	6.00	3.00	47.00
Minimum	5.60	3.90	16.20	28.30	4.60	33.10	19.20	10.30	3.90
Maximum	202.00	94.50	40.50	93.30	828.00	48.10	1330.00	371.00	1330.00
Arithmetic Mean	46.68	29.26	26.63	60.80	90.01	40.60	253.72	133.30	87.40
Standard Deviation	54.33	38.95	10.63	45.96	222.58	10.61	527.47	205.90	225.61
Geometric Mean	29.79	13.34	25.09	51.38	28.00	39.90	65.06	41.42	30.98
Geometric Standard Deviation	2.63	4.08	1.49	2.32	3.79	1.30	4.63	6.83	3.37
C Horizon									
Number	12.00	5.00	4.00	2.00	11.00	2.00	6.00	3.00	45.00
Minimum	5.40	5.20	9.40	17.00	4.00	31.00	13.20	16.10	4.00
Maximum	110.00	68.60	36.20	23.90	133.00	148.00	101.00	51.10	148.00
Arithmetic Mean	24.27	26.04	18.85	20.45	34.68	89.50	43.88	27.90	32.12
Standard Deviation	28.38	26.63	11.93	4.88	38.06	82.73	38.25	20.09	34.06
Geometric Mean	16.81	16.26	16.53	20.16	20.89	67.73	32.25	23.85	21.12
Geometric Standard Deviation	2.27	3.06	1.77	1.27	2.96	3.02	2.32	1.93	2.47
D Horizon									
Number	11.00	5.00	4.00	2.00	10.00	2.00	6.00	3.00	43.00
Minimum	4.60	4.30	4.40	9.00	4.20	25.80	10.00	9.20	4.20
Maximum	30.80	519.00	42.00	31.90	85.50	105.00	140.00	16.60	519.00
Arithmetic Mean	16.60	108.78	16.55	20.45	25.49	65.40	41.92	12.90	35.10
Standard Deviation	9.17	229.32	17.27	16.19	23.60	56.00	49.41	3.70	80.17
Geometric Mean	14.17	14.78	11.44	16.94	18.36	52.05	27.00	12.54	17.23
Geometric Standard Deviation	1.85	7.40	2.62	2.45	2.36	2.70	2.63	1.34	2.69

Table K-3.1 Yard Soil Cadmium Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	15.00	5.00	9.00	5.00	23.00	4.00	13.00	12.00	86.00
Minimum	0.06	0.06	0.50	1.10	0.20	4.80	0.52	0.19	0.06
Maximum	7.00	9.70	8.60	24.50	13.50	13.20	11.90	16.50	24.50
Arithmetic Mean	1.56	2.04	2.39	9.08	5.43	8.10	4.13	6.11	4.47
Standard	1.83	4.28	2.54	9.30	3.65	4.05	3.09	4.69	4.37
Geometric Mean	0.87	0.27	1.60	5.57	3.65	7.37	3.12	3.57	2.31
Geometric Standard	3.40	8.13	2.54	3.28	3.16	1.65	2.32	4.23	4.09
B Horizon									
Number	15.00	5.00	8.00	5.00	24.00	4.00	12.00	12.00	85.00
Minimum	0.06	0.06	0.47	1.20	0.06	2.50	0.20	0.19	0.06
Maximum	3.30	8.20	14.20	25.70	15.00	6.20	10.20	42.30	42.30
Arithmetic Mean	1.06	1.74	3.24	9.92	3.69	4.33	3.14	8.30	4.04
Standard	1.10	3.61	4.56	10.33	3.17	1.89	3.13	11.37	5.96
Geometric Mean	0.59	0.26	1.78	5.27	2.31	4.00	1.86	3.45	1.72
Geometric Standard	3.33	7.56	2.99	4.02	3.43	1.59	3.30	5.26	4.45
C Horizon									
Number	14.00	5.00	8.00	3.00	24.00	4.00	13.00	10.00	81.00
Minimum	0.06	0.06	0.06	1.70	0.06	0.29	0.06	0.48	0.06
Maximum	0.93	8.10	28.70	33.30	8.40	3.10	45.30	63.90	63.90
Arithmetic Mean	0.27	1.72	4.33	14.10	1.84	2.07	8.18	15.19	4.94
Standard	0.24	3.57	9.90	16.86	2.44	1.34	15.89	18.68	11.00
Geometric Mean	0.20	0.25	0.57	7.45	0.76	1.50	1.63	7.21	0.93
Geometric Standard	2.15	7.54	8.54	4.43	4.18	3.08	6.90	4.43	6.48
D Horizon									
Number	12.00	4.00	8.00	3.00	22.00	4.00	12.00	8.00	73.00
Minimum	0.06	0.06	0.06	7.60	0.06	0.19	0.06	0.42	0.06
Maximum	0.42	5.40	1.60	32.90	12.60	0.94	10.80	14.50	32.90
Arithmetic Mean	0.18	1.43	0.49	16.37	1.42	0.55	2.56	7.89	2.58
Standard	0.09	2.65	0.57	14.33	2.81	0.41	3.93	4.98	5.13
Geometric Mean	0.16	0.25	0.24	12.91	0.47	0.42	0.72	5.41	0.56
Geometric Standard	1.72	8.38	3.77	2.25	4.26	2.43	6.04	3.29	5.78

Table K-3.2 Vacuum Dust Cadmium Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Number	11.00	5.00	8.00	4.00	17.00	1.00	9.00	11.00	66.00
Minimum	2.60	0.20	2.50	4.20	1.40	6.40	2.70	1.30	0.20
Maximum	10.30	4.30	61.00	16.80	16.00	6.40	9.90	103.00	103.00
Arithmetic Mean	4.87	2.66	13.36	10.15	6.45	6.40	5.39	16.01	8.41
Standard	2.80	1.77	19.70	5.17	4.66		2.50	29.05	14.15
Geometric Mean	4.30	1.75	7.60	9.07	5.05	6.40	4.93	8.08	5.34
Geometric Standard	1.65	3.64	2.69	1.78	2.08		1.55	2.87	2.39

Table K-3.3 Right-of-ways Cadmium Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	5.00	3.00	2.00	1.00	3.00	1.00	1.00	1.00	17.00
Minimum	0.06	0.06	1.60	6.80	0.16	15.10	0.06	2.40	0.06
Maximum	1.10	0.20	5.10	6.80	3.80	15.10	0.06	2.40	15.10
Arithmetic Mean	0.38	0.15	3.35	6.80	1.39	15.10	0.06	2.40	2.21
Standard Deviation	0.41	0.08	2.47		2.09				3.88
Geometric Mean	0.25	0.13	2.86	6.80	0.50	15.10	0.06	2.40	0.55
Geometric Standard Deviation	2.88	2.00	2.27		5.86				6.00
B Horizon									
Number	5.00	3.00	2.00	1.00	3.00	1.00		1.00	16.00
Minimum	0.06	0.06	1.40	49.30	0.20	15.40		15.60	0.06
Maximum	7.50	0.20	8.30	49.30	18.60	15.40		15.60	49.30
Arithmetic Mean	2.30	0.15	4.85	49.30	6.47	15.40		15.60	7.58
Standard Deviation	3.17	0.08	4.88		10.51				12.84
Geometric Mean	0.58	0.13	3.41	49.30	1.31	15.40		15.60	1.27
Geometric Standard Deviation	9.21	2.00	3.52		10.61				10.01
C Horizon									
Number	5.00	3.00	2.00	1.00	2.00	1.00		1.00	15.00
Minimum	0.06	0.06	0.19	114.00	0.19	46.30		14.30	0.06
Maximum	6.50	0.20	15.70	114.00	0.71	46.30		14.30	114.00
Arithmetic Mean	2.43	0.15	7.95	114.00	0.45	46.30		14.30	13.60
Standard Deviation	2.94	0.08	10.97		0.37				30.33
Geometric Mean	0.78	0.13	1.73	114.00	0.37	46.30		14.30	1.22
Geometric Standard Deviation	7.42	1.98	22.68		2.54				12.01
D Horizon									
Number	4.00	3.00	1.00	1.00	1.00	1.00		1.00	12.00
Minimum	0.06	0.06	5.80	50.80	0.06	13.60		12.40	0.06
Maximum	6.60	0.20	5.80	50.80	0.06	13.60		12.40	50.80
Arithmetic Mean	3.34	0.15	5.80	50.80	0.06	13.60		12.40	8.04
Standard Deviation	3.71	0.08							14.34
Geometric Mean	0.85	0.13	5.80	50.80	0.06	13.60		12.40	1.11
Geometric Standard Deviation	11.16	2.00							12.49

Table K-3.7 Initial and Purged Drinking Water Cadmium Concentrations (ug/L) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Initial									
Number	15.00	5.00	9.00	4.00	22.00	2.00	13.00	12.00	82.00
Minimum	0.30	0.30	0.30	0.30	0.30	1.00	0.30	0.30	0.30
Maximum	5.20	1.00	2.00	1.60	2.10	6.60	7.00	2.00	7.00
Arithmetic Mean	0.87	0.62	0.83	0.88	0.87	3.80	1.27	0.65	0.95
Standard Deviation	1.24	0.35	0.54	0.56	0.48	3.96	1.78	0.51	1.15
Geometric Mean	0.56	0.54	0.69	0.73	0.75	2.57	0.78	0.52	0.68
Geometric Standard Deviation	2.29	1.76	1.97	2.05	1.83	3.80	2.56	1.92	2.13
Purged									
Number	16.00	5.00	8.00	4.00	24.00	2.00	13.00	12.00	84.00
Minimum	0.30	0.30	0.30	0.30	0.30	1.00	0.30	0.30	0.30
Maximum	1.00	3.40	1.00	1.00	8.40	1.00	1.80	1.00	8.40
Arithmetic Mean	0.53	1.20	0.56	0.65	0.93	1.00	0.74	0.54	0.74
Standard Deviation	0.33	1.28	0.36	0.40	1.63	0.00	0.47	0.34	0.97
Geometric Mean	0.44	0.79	0.47	0.55	0.58	1.00	0.60	0.46	0.54
Geometric Standard Deviation	1.77	2.76	1.86	2.00	2.25	1.00	1.99	1.79	2.01

Table K-3.4 Play Areas Cadmium Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	3.00	1.00	3.00	1.00	3.00		1.00	2.00	14.00
Minimum	0.06	12.40	0.58	0.06	0.22		3.40	0.70	0.06
Maximum	7.00	12.40	16.90	0.06	53.00		3.40	0.80	53.00
Arithmetic Mean	2.46	12.40	6.16	0.06	18.24		3.40	0.75	7.00
Standard Deviation	3.93		9.30		30.11			0.07	14.22
Geometric Mean	0.52	12.40	2.14	0.06	2.60		3.40	0.75	1.29
Geometric Standard Deviation	11.15		6.11		16.17			1.10	7.75
B Horizon									
Number	3.00	1.00	3.00	1.00	4.00		1.00	1.00	14.00
Minimum	0.06	13.00	0.06	0.06	0.06		2.70	2.10	0.06
Maximum	2.40	13.00	16.80	0.06	21.40		2.70	2.10	21.40
Arithmetic Mean	0.89	13.00	5.86	0.06	5.86		2.70	2.10	4.40
Standard Deviation	1.31		9.48		10.37				7.12
Geometric Mean	0.31	13.00	0.90	0.06	1.04		2.70	2.10	0.85
Geometric Standard Deviation	6.56		16.83		11.12				8.62
C Horizon									
Number	3.00	1.00	3.00		3.00		1.00		11.00
Minimum	0.06	19.20	0.06		0.10		0.06		0.06
Maximum	0.27	19.20	8.90		7.60		0.06		19.20
Arithmetic Mean	0.18	19.20	3.05		2.90		0.06		3.42
Standard Deviation	0.11		5.06		4.10				6.15
Geometric Mean	0.15	19.20	0.47		0.91		0.06		0.48
Geometric Standard Deviation	2.22		13.59		8.73				8.86
D Horizon									
Number	2.00	1.00	3.00		2.00				8.00
Minimum	0.06	15.70	0.06		0.06				0.06
Maximum	0.61	15.70	1.20		0.06				15.70
Arithmetic Mean	0.34	15.70	0.49		0.06				2.24
Standard Deviation	0.39		0.62		0.00				5.45
Geometric Mean	0.19	15.70	0.24		0.06				0.27
Geometric Standard Deviation	5.15		4.52		1.00				7.53

Table K-3.5 Garden Cadmium Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Total
F Horizon								
Number	7.00	2.00	1.00	2.00	9.00	1.00	3.00	25.00
Minimum	0.06	0.06	8.90	1.40	0.29	6.90	0.20	0.06
Maximum	2.20	0.20	8.90	1.70	5.70	6.90	1.20	8.90
Arithmetic Mean	0.65	0.13	8.90	1.55	2.35	6.90	0.71	1.88
Standard Deviation	0.76	0.10		0.21	2.10		0.50	2.39
Geometric Mean	0.36	0.11	8.90	1.54	1.53	6.90	0.56	0.83
Geometric Standard Deviation	3.40	2.34		1.15	2.83		2.52	4.11
G Horizon								
Number	7.00	2.00		1.00	9.00	1.00	3.00	23.00
Minimum	0.06	0.06		8.20	0.20	2.80	0.06	0.06
Maximum	2.20	0.19		8.20	2.80	2.80	2.00	8.20
Arithmetic Mean	0.48	0.13		8.20	0.81	2.80	0.88	1.07
Standard Deviation	0.76	0.09			0.99		1.00	1.81
Geometric Mean	0.25	0.11		8.20	0.46	2.80	0.41	0.41
Geometric Standard Deviation	2.95	2.26			2.86		5.92	3.91

Table K-3.6 Driveway Cadmium Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	13.00	5.00	4.00	2.00	14.00	2.00	6.00	3.00	49.00
Minimum	0.06	0.06	0.16	10.70	0.06	1.90	0.19	2.00	0.06
Maximum	6.60	5.60	12.20	18.70	73.90	6.40	8.40	18.60	73.90
Arithmetic Mean	1.37	1.25	4.47	14.70	11.26	4.15	3.48	7.63	5.73
Standard Deviation	2.14	2.43	5.31	5.66	18.82	3.18	3.11	9.50	11.21
Geometric Mean	0.54	0.31	1.95	14.15	2.59	3.49	2.09	4.41	1.47
Geometric Standard Deviation	4.00	5.51	6.14	1.48	10.98	2.36	3.80	3.49	6.87
B Horizon									
Number	12.00	5.00	4.00	2.00	13.00	2.00	6.00	3.00	47.00
Minimum	0.06	0.06	0.30	23.60	0.06	2.20	0.16	0.97	0.06
Maximum	27.10	11.60	9.40	42.30	24.50	9.00	18.10	5.70	42.30
Arithmetic Mean	4.77	2.42	5.53	32.95	8.00	5.60	5.33	2.92	6.67
Standard Deviation	7.93	5.13	4.20	13.22	8.60	4.81	7.63	2.47	9.13
Geometric Mean	1.44	0.28	3.12	31.60	2.11	4.45	1.18	2.26	1.72
Geometric Standard Deviation	5.99	8.78	4.99	1.51	9.69	2.71	8.52	2.43	7.57
C Horizon									
Number	12.00	5.00	4.00	2.00	11.00	2.00	6.00	3.00	45.00
Minimum	0.06	0.06	0.87	6.60	0.06	0.06	0.06	1.10	0.06
Maximum	4.20	5.40	9.20	7.10	8.30	0.86	10.30	4.50	10.30
Arithmetic Mean	0.98	1.15	4.19	6.85	3.89	0.46	3.01	3.13	2.65
Standard Deviation	1.25	2.37	3.54	0.35	3.19	0.57	4.46	1.80	3.03
Geometric Mean	0.49	0.19	3.08	6.85	1.71	0.23	0.55	2.66	0.87
Geometric Standard Deviation	3.48	7.02	2.63	1.05	6.31	6.57	9.70	2.16	6.11
D Horizon									
Number	11.00	5.00	4.00	2.00	10.00	2.00	6.00	3.00	43.00
Minimum	0.06	0.06	0.06	1.90	0.06	2.00	0.06	0.06	0.06
Maximum	3.40	0.56	8.40	8.50	8.70	2.50	8.10	2.80	8.70
Arithmetic Mean	0.84	0.26	4.29	5.20	2.42	2.25	3.19	1.82	2.13
Standard Deviation	1.03	0.19	3.95	4.67	2.88	0.35	3.75	1.53	2.72
Geometric Mean	0.45	0.20	1.60	4.02	0.92	2.24	0.72	0.76	0.72
Geometric Standard Deviation	3.27	2.25	9.82	2.88	5.55	1.17	10.48	9.00	5.38

Table K-4.1 Yard Soil Iron Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	15.00	5.00	9.00	5.00	23.00	4.00	13.00	12.00	86.00
Minimum	11500.00	14700.00	15900.00	16800.00	13600.00	17900.00	14700.00	17500.00	11500.00
Maximum	22600.00	64400.00	45800.00	42800.00	25100.00	29500.00	25300.00	23900.00	64400.00
Arithmetic Mean	17173.33	26860.00	22433.33	24620.00	20078.26	24650.00	19484.62	20583.33	20669.77
Standard	2944.11	21156.87	8964.23	10391.92	2619.50	4916.30	3277.92	2061.26	6822.56
Geometric Mean	16931.27	22474.81	21359.92	23244.17	19907.34	24246.80	19237.93	20489.54	19971.41
Geometric Standard	1.19	1.84	1.35	1.43	1.15	1.24	1.18	1.10	1.27
B Horizon									
Number	15.00	5.00	8.00	5.00	24.00	4.00	12.00	12.00	85.00
Minimum	12000.00	16200.00	16000.00	18800.00	13700.00	18800.00	13200.00	17900.00	12000.00
Maximum	23500.00	74500.00	53300.00	50200.00	31300.00	31500.00	30200.00	72900.00	74500.00
Arithmetic Mean	18446.67	29480.00	25275.00	26600.00	20662.50	24175.00	21283.33	25966.67	22575.29
Standard	3084.03	25268.99	11598.00	13239.90	3626.21	5368.04	5081.85	15023.94	9985.31
Geometric Mean	18180.20	24010.99	23699.51	24682.64	20362.84	23749.76	20720.10	23838.83	21347.66
Geometric Standard	1.20	1.90	1.42	1.49	1.19	1.24	1.28	1.45	1.35
C Horizon									
Number	14.00	5.00	8.00	3.00	24.00	4.00	13.00	10.00	81.00
Minimum	11500.00	14700.00	16600.00	22600.00	13100.00	17600.00	15200.00	1030.00	1030.00
Maximum	25800.00	54700.00	52500.00	56400.00	25900.00	27300.00	32900.00	31900.00	56400.00
Arithmetic Mean	18185.71	25080.00	23750.00	35033.33	20070.83	23150.00	23284.62	20603.00	21705.31
Standard	4264.68	16866.59	11823.34	18586.64	3286.53	4149.30	6290.45	7852.28	8129.76
Geometric Mean	17713.71	21887.47	22085.72	32161.97	19794.77	22851.27	22529.94	16526.88	20222.64
Geometric Standard	1.27	1.72	1.45	1.64	1.19	1.21	1.30	2.68	1.55
D Horizon									
Number	12.00	4.00	8.00	3.00	22.00	4.00	12.00	8.00	73.00
Minimum	10100.00	13600.00	12500.00	20200.00	13900.00	18600.00	11200.00	19000.00	10100.00
Maximum	25800.00	24100.00	23500.00	51800.00	27400.00	27200.00	25700.00	22700.00	51800.00
Arithmetic Mean	17391.67	18500.00	18550.00	32366.67	20686.36	23175.00	19558.33	20450.00	20195.89
Standard	4865.74	5100.98	3722.52	17007.16	3351.94	4053.29	3925.08	1344.83	5399.91
Geometric Mean	16762.52	17970.66	18200.06	29724.75	20423.16	22903.41	19153.78	20411.96	19602.48
Geometric Standard	1.33	1.32	1.24	1.64	1.18	1.20	1.25	1.07	1.27

Table K-4.2 Vacuum Dust Iron Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Number	11.00	5.00	8.00	4.00	17.00	1.00	9.00	11.00	66.00
Minimum	9670.00	13600.00	8640.00	5630.00	2320.00	19300.00	5780.00	2540.00	2320.00
Maximum	14500.00	18400.00	17500.00	15100.00	19000.00	19300.00	14300.00	27500.00	27500.00
Arithmetic Mean	11347.27	15060.00	13312.50	12307.50	10178.24	19300.00	10292.22	13019.09	11879.09
Standard	1594.98	1966.72	3273.35	4511.42	4749.79		2878.24	6114.89	4279.14
Geometric Mean	11249.16	14965.22	12940.16	11454.54	8866.12	19300.00	9902.03	11492.32	10946.51
Geometric Standard	1.15	1.13	1.30	1.61	1.81		1.35	1.80	1.57

Table K-4.3 Right-of-ways Iron Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	5.00	3.00	2.00	1.00	3.00	1.00	1.00	1.00	17.00
Minimum	13000.00	19900.00	15000.00	36300.00	28300.00	85100.00	27900.00	20600.00	13000.00
Maximum	29800.00	32900.00	26800.00	36300.00	35500.00	85100.00	27900.00	20600.00	85100.00
Arithmetic Mean	18880.00	24800.00	20900.00	36300.00	32100.00	85100.00	27900.00	20600.00	28047.06
Standard Deviation	6398.20	7066.12	8343.86		3616.63				16472.06
Geometric Mean	18144.11	24182.48	20049.94	36300.00	31961.91	85100.00	27900.00	20600.00	25158.40
Geometric Standard Deviation	1.36	1.31	1.51		1.12				1.56
B Horizon									
Number	5.00	3.00	2.00	1.00	3.00	1.00		1.00	16.00
Minimum	20600.00	11900.00	14900.00	36500.00	16600.00	66800.00		37600.00	11900.00
Maximum	88000.00	24400.00	29900.00	36500.00	40300.00	66800.00		37600.00	88000.00
Arithmetic Mean	37140.00	19000.00	22400.00	36500.00	29066.67	66800.00		37600.00	32225.00
Standard Deviation	28707.63	6421.06	10606.60		11898.04				19808.53
Geometric Mean	31253.06	18181.75	21107.11	36500.00	27265.82	66800.00		37600.00	28067.82
Geometric Standard Deviation	1.82	1.46	1.64		1.57				1.68
C Horizon									
Number	5.00	3.00	2.00	1.00	2.00	1.00		1.00	15.00
Minimum	21400.00	12900.00	23900.00	28100.00	15600.00	87900.00		33600.00	12900.00
Maximum	39600.00	22900.00	24100.00	28100.00	28000.00	87900.00		33600.00	87900.00
Arithmetic Mean	27360.00	19066.67	24000.00	28100.00	21800.00	87900.00		33600.00	29013.33
Standard Deviation	7357.51	5392.90	141.42		8768.12				17537.55
Geometric Mean	26659.83	18490.40	23999.79	28100.00	20899.76	87900.00		33600.00	26101.00
Geometric Standard Deviation	1.28	1.37	1.01		1.51				1.54
D Horizon									
Number	4.00	3.00	1.00	1.00	1.00	1.00		1.00	12.00
Minimum	13000.00	11900.00	26000.00	28500.00	18400.00	34400.00		36100.00	11900.00
Maximum	49500.00	23000.00	26000.00	28500.00	18400.00	34400.00		36100.00	49500.00
Arithmetic Mean	27200.00	17466.67	26000.00	28500.00	18400.00	34400.00		36100.00	25383.33
Standard Deviation	15814.97	5550.08							10737.26
Geometric Mean	24127.45	16856.64	26000.00	28500.00	18400.00	34400.00		36100.00	23438.06
Geometric Standard Deviation	1.75	1.39							1.52

Table K-4.7 Initial and Purged Drinking Water Iron Concentrations (ug/L) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Initial									
Number	15.00	5.00	9.00	4.00	22.00	2.00	13.00	12.00	82.00
Minimum	16.00	16.00	18.60	18.60	16.00	93.80	23.30	17.20	16.00
Maximum	520.00	1270.00	1240.00	51.50	447.00	831.00	516.00	205.00	1270.00
Arithmetic Mean	159.30	411.60	297.69	34.10	81.25	462.40	109.35	40.03	144.85
Standard Deviation	191.85	497.13	395.22	17.96	104.23	521.28	134.11	53.68	237.24
Geometric Mean	65.66	196.34	120.60	30.36	49.05	279.19	71.06	27.17	61.61
Geometric Standard Deviation	4.18	4.95	4.71	1.76	2.59	4.68	2.43	2.13	3.43
Purged									
Number	16.00	5.00	8.00	4.00	24.00	2.00	13.00	12.00	84.00
Minimum	16.00	16.00	18.60	16.00	16.00	32.20	16.00	16.00	16.00
Maximum	178.00	871.00	499.00	18.60	160.00	516.00	577.00	117.00	871.00
Arithmetic Mean	46.06	292.80	81.45	17.30	43.30	274.10	84.98	28.68	70.93
Standard Deviation	41.58	350.35	168.76	1.50	35.23	342.10	162.03	28.32	137.93
Geometric Mean	34.84	111.45	31.80	17.25	33.37	128.90	34.50	23.27	34.38
Geometric Standard Deviation	2.08	6.27	3.08	1.09	2.04	7.11	3.16	1.74	2.66

Table K-4.4 Play Areas Iron Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	3.00	1.00	3.00	1.00	3.00		1.00	2.00	14.00
Minimum	18900.00	144000.00	22300.00	27800.00	13400.00		21700.00	5670.00	5670.00
Maximum	20900.00	144000.00	41200.00	27800.00	185000.00		21700.00	20300.00	185000.00
Arithmetic Mean	19600.00	144000.00	29533.33	27800.00	71866.67		21700.00	12985.00	41605.00
Standard Deviation	1126.94		10200.16		97994.76			10344.97	53255.89
Geometric Mean	19578.85	144000.00	28463.74	27800.00	34935.61		21700.00	10728.51	26250.77
Geometric Standard Deviation	1.06		1.38		4.26			2.46	2.43
B Horizon									
Number	3.00	1.00	3.00	1.00	4.00		1.00	1.00	14.00
Minimum	21100.00	163000.00	17200.00	27500.00	17800.00		19900.00	23700.00	17200.00
Maximum	22600.00	163000.00	52600.00	27500.00	84400.00		19900.00	23700.00	163000.00
Arithmetic Mean	21833.33	163000.00	31200.00	27500.00	35950.00		19900.00	23700.00	38357.14
Standard Deviation	750.56		18824.45		32423.60				40257.72
Geometric Mean	21824.74	163000.00	27820.42	27500.00	28244.89		19900.00	23700.00	29025.04
Geometric Standard Deviation	1.03		1.78		2.10				1.94
C Horizon									
Number	3.00	1.00	3.00		3.00		1.00		11.00
Minimum	19300.00	170000.00	17000.00		17600.00		19400.00		17000.00
Maximum	22800.00	170000.00	38100.00		21900.00		19400.00		170000.00
Arithmetic Mean	20966.67	170000.00	25766.67		20066.67		19400.00		35436.36
Standard Deviation	1755.94		10992.88		2218.86				44988.38
Geometric Mean	20917.92	170000.00	24316.94		19982.16		19400.00		25862.96
Geometric Standard Deviation	1.09		1.51		1.12				1.93
D Horizon									
Number	2.00	1.00	3.00		2.00				8.00
Minimum	17700.00	161000.00	14800.00		17200.00				14800.00
Maximum	20200.00	161000.00	73500.00		24100.00				161000.00
Arithmetic Mean	18950.00	161000.00	36233.33		20650.00				43612.50
Standard Deviation	1767.77		32395.11		4879.04				51185.98
Geometric Mean	18908.73	161000.00	28101.30		20359.76				29206.83
Geometric Standard Deviation	1.10		2.33		1.27				2.34

Table K-4.5 Garden Iron Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Total
F Horizon								
Number	7.00	2.00	1.00	2.00	9.00	1.00	3.00	25.00
Minimum	11300.00	16500.00	17900.00	20900.00	15300.00	22100.00	14100.00	11300.00
Maximum	30900.00	22200.00	17900.00	21700.00	25600.00	22100.00	16600.00	30900.00
Arithmetic Mean	19585.71	19350.00	17900.00	21300.00	19366.67	22100.00	15300.00	19144.00
Standard Deviation	6774.57	4030.51		565.69	2953.81		1253.00	4236.16
Geometric Mean	18620.38	19138.97	17900.00	21296.24	19177.36	22100.00	15265.94	18713.50
Geometric Standard Deviation	1.41	1.23		1.03	1.16		1.09	1.24
G Horizon								
Number	7.00	2.00		1.00	9.00	1.00	3.00	23.00
Minimum	11600.00	16700.00		36300.00	16500.00	15400.00	15300.00	11600.00
Maximum	30800.00	20200.00		36300.00	26400.00	15400.00	16600.00	36300.00
Arithmetic Mean	17757.14	18450.00		36300.00	20577.78	15400.00	15933.33	19386.96
Standard Deviation	6381.19	2474.87			2885.64		650.64	5595.23
Geometric Mean	16948.01	18366.82		36300.00	20402.06	15400.00	15924.49	18737.91
Geometric Standard Deviation	1.37	1.14			1.15		1.04	1.29

Table K-4.6 Driveway Iron Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	13.00	5.00	4.00	2.00	14.00	2.00	6.00	3.00	49.00
Minimum	3910.00	21700.00	15100.00	35700.00	8760.00	26700.00	16400.00	28800.00	3910.00
Maximum	46300.00	33500.00	27500.00	59600.00	50900.00	32400.00	77500.00	47300.00	77500.00
Arithmetic Mean	22556.15	26260.00	20400.00	47650.00	29897.14	29550.00	31100.00	36133.33	28042.65
Standard Deviation	11719.75	5406.76	5208.97	16899.85	11574.41	4030.51	23332.64	9827.68	13323.00
Geometric Mean	18845.67	25835.52	19925.62	46127.22	27343.42	29412.24	26323.96	35303.58	24863.98
Geometric Standard Deviation	2.03	1.22	1.28	1.44	1.61	1.15	1.78	1.30	1.71
B Horizon									
Number	12.00	5.00	4.00	2.00	13.00	2.00	6.00	3.00	47.00
Minimum	15400.00	6190.00	17700.00	39900.00	8770.00	27400.00	22000.00	24600.00	6190.00
Maximum	58100.00	59800.00	26800.00	54200.00	40700.00	29900.00	112000.00	38800.00	112000.00
Arithmetic Mean	30866.67	23606.00	22200.00	47050.00	26751.54	28650.00	40800.00	31066.67	30093.62
Standard Deviation	14200.79	21414.26	3715.73	10111.63	8842.37	1767.77	35282.35	7184.24	17033.80
Geometric Mean	28179.82	17352.79	21964.10	46503.55	25059.38	28622.72	33244.90	30525.38	26616.77
Geometric Standard Deviation	1.55	2.40	1.18	1.24	1.50	1.06	1.87	1.26	1.65
C Horizon									
Number	12.00	5.00	4.00	2.00	11.00	2.00	6.00	3.00	45.00
Minimum	11500.00	9880.00	16500.00	18400.00	9040.00	23000.00	19100.00	26500.00	9040.00
Maximum	31600.00	35900.00	34900.00	22800.00	41800.00	29800.00	35600.00	30500.00	41800.00
Arithmetic Mean	21950.00	22216.00	23875.00	20600.00	22894.55	26400.00	26633.33	28233.33	23562.67
Standard Deviation	5892.75	9568.19	8141.41	3111.27	8360.74	4808.33	5455.52	2052.64	6804.63
Geometric Mean	21162.76	20420.14	22906.17	20482.19	21445.94	26180.15	26173.10	28184.38	22510.83
Geometric Standard Deviation	1.34	1.61	1.39	1.16	1.48	1.20	1.23	1.07	1.38
D Horizon									
Number	11.00	5.00	4.00	2.00	10.00	2.00	6.00	3.00	43.00
Minimum	11900.00	10900.00	17500.00	14300.00	15000.00	22400.00	15800.00	16600.00	10900.00
Maximum	32400.00	33800.00	22700.00	28200.00	32500.00	34900.00	33700.00	27900.00	34900.00
Arithmetic Mean	21636.36	18480.00	19050.00	21250.00	21780.00	28650.00	25083.33	21733.33	21858.14
Standard Deviation	6716.89	9289.08	2466.44	9828.78	5256.91	8838.83	6059.51	5720.00	6421.14
Geometric Mean	20639.14	16898.61	18938.63	20081.33	21228.01	27959.97	24426.88	43.00	20923.08
Geometric Standard Deviation	1.39	1.58	1.13	1.62	1.27	1.37	1.30	1.30	1.35

Table K-5.1 Yard Soil Manganese Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	15	5	9	5	23	4	13	21	95
Minimum	362	310	572	676	489	667	564	481	310
Maximum	1270	4120	3100	1200	1610	1430	1400	1720	4120
Arithmetic Mean	648	1174	1272	941	979	962	917	809	916
Standard Deviation	254.22	1651.90	800.39	222.92	308.88	345.06	275.67	264.43	513.64
Geometric Mean	609	661	1107	920	934	919	879	778	828
Geometric Standard Deviation	1.43	2.90	1.70	1.27	1.37	1.41	1.36	1.31	1.53
B Horizon									
Number	15	5	8	5	24	4	12	21	94
Minimum	382	279	779	700	307	586	502	552	279
Maximum	1170	4610	4080	1290	2290	1690	2000	7580	7580
Arithmetic Mean	672	1260	1613	942	1022	972	1068	1161	1060
Standard Deviation	227.33	1878.06	1081.85	225.35	429.34	496.10	492.76	1490.48	930.71
Geometric Mean	639	650	1395	922	939	892	975	903	889
Geometric Standard Deviation	1.39	3.15	1.71	1.26	1.54	1.59	1.56	1.73	1.70
C Horizon									
Number	14	5	8	3	24	4	13	10	81
Minimum	290	132	483	870	184	187	364	490	132
Maximum	1290	3300	5490	2130	1840	1190	3520	2230	5490
Arithmetic Mean	663	891	1504	1423	973	705	1197	1176	1031
Standard Deviation	308.02	1352.20	1653.48	643.84	401.53	416.07	824.25	486.00	777.35
Geometric Mean	603	433	1084	1330	880	581	1013	1090	842
Geometric Standard Deviation	1.57	3.41	2.17	1.57	1.65	2.22	1.78	1.52	1.90
D Horizon									
Number	12	4	8	3	22	4	12	8	73
Minimum	179	184	326	843	305	157	262	575	157
Maximum	1650	923	1900	1840	1780	964	1120	1180	1900
Arithmetic Mean	641	441	1026	1248	903	511	733	885	811
Standard Deviation	396.15	329.90	557.23	524.32	371.90	374.20	265.45	213.45	403.02
Geometric Mean	548	367	892	1180	833	400	681	862	709
Geometric Standard Deviation	1.80	1.97	1.79	1.49	1.52	2.31	1.53	1.28	1.74

Table K-5.2 Vacuum Dust Manganese Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Number	11	5	8	4	17	1	9	11	66
Minimum	373	354	491	259	127	640	264	98	98
Maximum	669	1080	1550	923	1330	640	754	1360	1550
Arithmetic Mean	482	521	838	563	534	640	449	599	564
Standard Deviation	106.00	314.00	341.47	273.38	371.56		143.41	392.93	311.34
Geometric Mean	472	468	785	511	419	640	431	492	488
Geometric Standard Deviation	1.24	1.61	1.46	1.69	2.11		1.35	2.01	1.74

Table K-5.3 Right-of-way Manganese Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	5	3	2	1	3	1	1	1	17
Minimum	689	535	1170	2210	588	4520	661	1140	535
Maximum	1480	623	1730	2210	2030	4520	661	1140	4520
Arithmetic Mean	951	582	1450	2210	1138	4520	661	1140	1255
Standard Deviation	320.46	44.24	395.98		779.68				989.50
Geometric Mean	914	581	1423	2210	983	4520	661	1140	1035
Geometric Standard Deviation	1.36	1.08	1.32		1.91				1.80
B Horizon									
Number	5	3	2	1	3	1		1	16
Minimum	695	334	1200	3900	506	3600		2440	334
Maximum	6050	643	2250	3900	2650	3600		2440	6050
Arithmetic Mean	2008	524	1725	3900	1279	3600		2440	1802
Standard Deviation	2273.42	166.50	742.46		1190.79				1593.75
Geometric Mean	1393	504	1643	3900	970	3600		2440	1287
Geometric Standard Deviation	2.37	1.43	1.56		2.42				2.33
C Horizon									
Number	5	3	2	1	2	1		1	15
Minimum	825	356	614	6380	486	6620		2340	356
Maximum	1770	658	1930	6380	499	6620		2340	6620
Arithmetic Mean	1183	554	1272	6380	493	6620		2340	1763
Standard Deviation	387.39	171.82	930.55		9.19				2009.63
Geometric Mean	1136	534	1089	6380	492	6620		2340	1150
Geometric Standard Deviation	1.37	1.42	2.25		1.02				2.43
D Horizon									
Number	4	3	1	1	1	1		1	12
Minimum	421	335	691	5460	306	1870		2590	306
Maximum	2190	527	691	5460	306	1870		2590	5460
Arithmetic Mean	1166	418	691	5460	306	1870		2590	1403
Standard Deviation	743.87	98.61							1495.80
Geometric Mean	991	411	691	5460	306	1870		2590	921
Geometric Standard Deviation	1.97	1.26							2.53

Table K-5.7 Initial and Purged Drinking Water Manganese Concentrations (ug/L) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Initial									
Number	15	5	9	4	22	2	13	12	82
Minimum	1	1	2	1	1	9	1	1	1
Maximum	38	1920	8	2	5	69	6	4	1920
Arithmetic Mean	18	403	5	2	2	39	2	2	31
Standard Deviation	13.80	848.26	2.18	0.81	1.13	42.85	1.39	0.76	211.63
Geometric Mean	11	26	4	2	2	25	2	2	4
Geometric Standard Deviation	3.16	18.15	1.62	1.66	1.65	4.31	1.73	1.48	3.62
Purged									
Number	16	5	8	4	24	2	13	12	84
Minimum	2	1	1	1	1	1	1	1	1
Maximum	44	1920	8	2	4	82	5	2	1920
Arithmetic Mean	15	401	3	2	2	41	2	2	29
Standard Deviation	15.87	849.94	2.16	0.81	0.88	57.20	1.14	0.69	209.27
Geometric Mean	8	16	2	2	2	9	2	2	3
Geometric Standard Deviation	3.48	23.36	2.02	1.66	1.61	22.54	1.71	1.54	3.56

Table K-5.4 Play Area Manganese Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	3	1	3	1	3		1	10	22
Minimum	565	12100	1210	760	450		1110	412	412
Maximum	740	12100	3250	760	18300		1110	616	18300
Arithmetic Mean	662	12100	1960	760	6466		1110	596	2145
Standard Deviation	88.93		1122.10		10249.31			64.51	4372.39
Geometric Mean	658	12100	1774	760	1747		1110	592	965
Geometric Standard Deviation	1.15		1.70		7.71			1.14	2.69
B Horizon									
Number	3	1	3	1	4		1	9	22
Minimum	756	13400	644	729	399		1030	758	399
Maximum	894	13400	3980	729	6400		1030	758	13400
Arithmetic Mean	820	13400	2031	729	2143		1030	758	1778
Standard Deviation	69.47		1737.40		2851.67				2927.81
Geometric Mean	818	13400	1556	729	1176		1030	758	1055
Geometric Standard Deviation	1.09		2.49		3.32				2.28
C Horizon									
Number	3	1	3		3		1		11
Minimum	703	14400	212		561		721		212
Maximum	1210	14400	2610		2090		721		14400
Arithmetic Mean	890	14400	1387		1360		721		2367
Standard Deviation	278.68		1199.70		766.88				4051.91
Geometric Mean	863	14400	905		1188		721		1212
Geometric Standard Deviation	1.34		3.67		1.97				2.92
D Horizon									
Number	2	1	3		2				8
Minimum	544	10500	209		296				209
Maximum	734	10500	5700		1360				10500
Arithmetic Mean	639	10500	2313		828				2547
Standard Deviation	134.35		2961.81		752.36				3678.78
Geometric Mean	632	10500	1071		634				1095
Geometric Standard Deviation	1.24		5.22		2.94				3.90

Table K-5.5 Garden Manganese Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Total
F Horizon								
Number	7	2	1	2	9	1	3	25
Minimum	345	326	1050	744	457	809	275	275
Maximum	1550	454	1050	917	1680	809	676	1680
Arithmetic Mean	776	390	1050	831	946	809	496	789
Standard Deviation	388.60	90.51		122.33	406.46		203.52	365.45
Geometric Mean	705	385	1050	826	877	809	464	715
Geometric Standard Deviation	1.60	1.26		1.16	1.50		1.60	1.58
G Horizon								
Number	7	2		1	9	1	3	23
Minimum	179	309		1890	416	607	289	179
Maximum	1660	380		1890	1560	607	725	1890
Arithmetic Mean	755	345		1890	960	607	569	818
Standard Deviation	497.87	50.20			382.93		243.01	467.94
Geometric Mean	610	343		1890	887	607	526	692
Geometric Standard Deviation	2.11	1.16			1.55		1.68	1.85

Table K-5.6 Driveway Manganese Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	13	5	4	2	14	2	6	3	49
Minimum	201	223	712	2070	215	903	738	971	201
Maximum	2180	1710	2130	2820	3970	1170	4390	2380	4390
Arithmetic Mean	927	714	1283	2445	1608	1037	1572	1590	1315
Standard Deviation	576.80	575.51	600.55	530.33	1029.21	188.80	1410.25	719.78	910.25
Geometric Mean	751	572	1187	2416	1255	1028	1251	1486	1036
Geometric Standard Deviation	2.06	2.07	1.57	1.24	2.25	1.20	1.95	1.57	2.08
B Horizon									
Number	12	5	4	2	13	2	6	3	47
Minimum	529	77	935	2260	240	879	675	584	77
Maximum	5540	3260	2180	4980	3900	1200	6780	2870	6780
Arithmetic Mean	1982	894	1591	3620	1517	1040	2256	1515	1739
Standard Deviation	1646.11	1346.11	553.78	1923.33	1091.75	226.98	2319.19	1200.71	1478.96
Geometric Mean	1469	354	1513	3355	1163	1027	1584	1222	1208
Geometric Standard Deviation	2.24	4.68	1.46	1.75	2.23	1.25	2.41	2.23	2.57
C Horizon									
Number	12	5	4	2	11	2	6	3	45
Minimum	447	89	1290	913	159	923	547	1120	89
Maximum	2350	2020	1720	1200	3380	1190	2720	2040	3380
Arithmetic Mean	1007	742	1510	1057	1314	1057	1211	1547	1165
Standard Deviation	555.79	773.90	237.07	202.94	952.50	188.80	789.73	463.61	692.02
Geometric Mean	895	456	1496	1047	963	1048	1047	1501	948
Geometric Standard Deviation	1.64	3.23	1.17	1.21	2.52	1.20	1.76	1.35	2.06
D Horizon									
Number	11	5	4	2	10	2	6	3	43
Minimum	446	49	361	465	167	1010	484	835	49
Maximum	1790	1420	1710	1410	2300	1600	2030	1300	2300
Arithmetic Mean	860	511	1135	938	1057	1305	1098	990	957
Standard Deviation	457.67	532.81	618.64	668.22	668.95	417.19	539.44	268.18	541.51
Geometric Mean	767	311	969	810	852	1271	993	968	782
Geometric Standard Deviation	1.63	3.42	2.04	2.19	2.13	1.38	1.64	1.29	2.08

Table K-1.1 Yard Soil Antimony Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	15.00	5.00	9.00	5.00	23.00	4.00	13.00	12.00	86.00
Minimum	0.76	0.42	0.96	0.91	0.79	3.60	0.62	0.77	0.42
Maximum	4.70	22.00	43.20	18.50	125.00	9.40	17.70	14.10	125.00
Arithmetic Mean	1.87	5.12	7.57	6.52	11.59	6.65	5.35	4.86	6.69
Standard	1.08	9.45	13.53	6.94	25.92	2.93	5.44	3.97	14.69
Geometric Mean	1.63	1.46	3.49	4.19	5.15	6.13	3.25	3.42	3.30
Geometric Standard	1.70	5.13	3.09	2.97	2.89	1.61	2.94	2.56	2.89
B Horizon									
Number	15.00	5.00	8.00	5.00	24.00	4.00	12.00	12.00	85.00
Minimum	0.42	0.42	0.96	1.40	0.42	1.40	0.42	0.79	0.42
Maximum	3.50	25.20	48.00	20.50	106.00	15.70	15.00	59.00	106.00
Arithmetic Mean	1.71	5.61	8.26	7.08	8.66	6.53	4.20	8.86	6.42
Standard	1.00	10.96	16.12	7.75	21.09	6.31	4.59	16.14	14.06
Geometric Mean	1.45	1.35	3.31	4.65	3.56	4.57	2.52	4.12	2.85
Geometric Standard	1.84	5.42	3.31	2.73	3.18	2.70	2.89	3.14	3.08
C Horizon									
Number	14.00	5.00	8.00	3.00	24.00	4.00	13.00	10.00	81.00
Minimum	0.41	0.41	0.42	0.97	0.42	1.10	0.42	0.42	0.41
Maximum	2.60	18.20	39.40	54.90	17.90	8.40	13.00	14.40	54.90
Arithmetic Mean	1.23	4.40	5.96	21.22	4.03	3.65	3.56	5.78	4.52
Standard	0.61	7.74	13.53	29.36	4.11	3.27	3.99	4.79	7.99
Geometric Mean	1.09	1.43	1.55	7.46	2.62	2.75	2.22	3.29	2.15
Geometric Standard	1.69	4.78	4.33	7.53	2.60	2.36	2.77	3.81	3.13
D Horizon									
Number	12.00	4.00	8.00	3.00	22.00	4.00	12.00	8.00	73.00
Minimum	0.42	0.42	0.42	5.20	0.43	0.77	0.42	0.88	0.42
Maximum	2.30	6.60	6.70	81.40	5.80	4.70	5.70	6.00	81.40
Arithmetic Mean	1.19	2.18	1.82	31.63	2.31	2.24	2.17	3.22	3.34
Standard	0.55	2.96	2.08	43.13	1.49	1.73	1.97	2.05	9.45
Geometric Mean	1.07	1.18	1.17	15.20	1.84	1.81	1.43	2.61	1.69
Geometric Standard	1.65	3.33	2.68	4.36	2.07	2.14	2.64	2.08	2.60

Table K-1.2 Vacuum Dust Antimony Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Number	11.00	5.00	8.00	4.00	17.00	1.00	9.00	11.00	66.00
Minimum	3.10	2.20	7.90	10.40	3.70	14.00	6.20	1.70	1.70
Maximum	33.10	8.40	102.00	20.60	52.20	14.00	106.00	103.00	106.00
Arithmetic Mean	11.64	5.28	33.88	15.35	19.71	14.00	33.92	31.65	22.57
Standard	9.37	2.81	32.61	4.17	15.88		38.67	34.62	25.63
Geometric Mean	8.78	4.66	24.46	14.91	14.76	14.00	20.24	17.04	14.10
Geometric Standard	2.22	1.77	2.27	1.32	2.19		2.83	3.46	2.60

Table K-1.3 Right-of-ways Antimony Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	5.00	3.00	2.00	1.00	3.00	1.00	1.00	1.00	17.00
Minimum	0.80	0.93	3.90	8.90	4.70	57.90	2.90	6.90	0.80
Maximum	7.30	1.40	24.30	8.90	28.20	57.90	2.90	6.90	57.90
Arithmetic Mean	2.92	1.14	14.10	8.90	13.20	57.90	2.90	6.90	9.55
Standard Deviation	2.53	0.24	14.42		13.03				14.74
Geometric Mean	2.26	1.13	9.73	8.90	9.61	57.90	2.90	6.90	4.35
Geometric Standard Deviation	2.20	1.23	3.65		2.58				3.44
B Horizon									
Number	5.00	3.00	2.00	1.00	3.00	1.00		1.00	16.00
Minimum	1.90	0.72	3.60	28.40	0.42	66.20		16.50	0.42
Maximum	158.00	1.00	23.30	28.40	20.80	66.20		16.50	158.00
Arithmetic Mean	41.04	0.90	13.45	28.40	9.14	66.20		16.50	23.33
Standard Deviation	66.34	0.16	13.93		10.50				39.84
Geometric Mean	12.39	0.89	9.16	28.40	3.78	66.20		16.50	6.95
Geometric Standard Deviation	6.20	1.21	3.75		7.37				5.82
C Horizon									
Number	5.00	3.00	2.00	1.00	2.00	1.00		1.00	15.00
Minimum	0.89	0.62	0.96	27.30	0.51	85.40		24.70	0.51
Maximum	19.60	0.99	23.60	27.30	139.00	85.40		24.70	139.00
Arithmetic Mean	8.86	0.86	12.28	27.30	69.76	85.40		24.70	23.22
Standard Deviation	8.27	0.21	16.01		97.93				38.83
Geometric Mean	4.96	0.84	4.76	27.30	8.42	85.40		24.70	5.59
Geometric Standard Deviation	3.93	1.30	9.62		52.74				6.85
D Horizon									
Number	4.00	3.00	1.00	1.00	1.00	1.00		1.00	12.00
Minimum	1.60	0.60	8.00	20.80	0.42	15.90		27.30	0.42
Maximum	9.70	1.00	8.00	20.80	0.42	15.90		27.30	27.30
Arithmetic Mean	4.98	0.87	8.00	20.80	0.42	15.90		27.30	7.91
Standard Deviation	4.01	0.23							9.01
Geometric Mean	3.67	0.84	8.00	20.80	0.42	15.90		27.30	3.49
Geometric Standard Deviation	2.55	1.34							4.35

Table K-1.7 Initial and Purged Drinking Water Antimony Concentrations (ug/L) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Initial									
Number	15.00	5.00	9.00	4.00	22.00	2.00	13.00	12.00	82.00
Minimum	2.10	2.10	2.10	2.10	2.10	5.00	2.10	2.10	2.10
Maximum	5.00	5.00	5.00	5.00	5.00	5.00	5.00	5.00	5.00
Arithmetic Mean	3.26	3.26	3.39	3.55	3.68	5.00	3.88	2.58	3.44
Standard Deviation	1.47	1.59	1.53	1.67	1.48	0.00	1.47	1.13	1.46
Geometric Mean	2.97	2.97	3.09	3.24	3.37	5.00	3.58	2.43	3.14
Geometric Standard Deviation	1.55	1.61	1.58	1.65	1.56	1.00	1.55	1.40	1.55
Purged									
Number	16.00	5.00	8.00	4.00	24.00	2.00	13.00	12.00	84.00
Minimum	2.10	2.10	2.10	2.10	2.10	5.00	2.10	2.10	2.10
Maximum	5.00	5.00	5.00	5.00	5.00	5.00	5.00	5.00	5.00
Arithmetic Mean	3.01	3.84	3.28	3.55	3.19	5.00	3.66	3.07	3.32
Standard Deviation	1.39	1.59	1.45	1.67	1.43	0.00	1.50	1.43	1.43
Geometric Mean	2.75	3.53	3.01	3.24	2.91	5.00	3.35	2.80	3.02
Geometric Standard Deviation	1.51	1.61	1.54	1.65	1.54	1.00	1.57	1.53	1.53

Table K-1.4 Play Areas Antimony Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	3.00	1.00	3.00	1.00	3.00		1.00	2.00	14.00
Minimum	0.42	37.10	2.00	1.50	0.52		5.70	1.30	0.42
Maximum	4.00	37.10	28.50	1.50	31.10		5.70	3.20	37.10
Arithmetic Mean	1.80	37.10	11.10	1.50	10.83		5.70	2.25	8.57
Standard Deviation	1.93		15.07		17.56			1.34	13.02
Geometric Mean	1.18	37.10	5.42	1.50	2.41		5.70	2.04	3.00
Geometric Standard Deviation	3.12		4.25		9.28			1.89	4.39
B Horizon									
Number	3.00	1.00	3.00	1.00	4.00		1.00	1.00	14.00
Minimum	0.64	43.60	1.20	0.75	0.54		2.60	7.20	0.54
Maximum	3.40	43.60	39.80	0.75	23.40		2.60	7.20	43.60
Arithmetic Mean	1.85	43.60	14.23	0.75	7.42		2.60	7.20	9.43
Standard Deviation	1.41		22.14		10.85				14.91
Geometric Mean	1.48	43.60	4.33	0.75	2.61		2.60	7.20	3.10
Geometric Standard Deviation	2.31		6.88		5.84				4.57
C Horizon									
Number	3.00	1.00	3.00		3.00		1.00		11.00
Minimum	0.70	46.60	0.47		0.53		0.68		0.47
Maximum	2.40	46.60	23.10		10.80		0.68		46.60
Arithmetic Mean	1.36	46.60	8.29		4.14		0.68		8.06
Standard Deviation	0.91		12.83		5.77				14.55
Geometric Mean	1.18	46.60	2.42		1.85		0.68		2.15
Geometric Standard Deviation	1.89		7.54		4.82				5.00
D Horizon									
Number	2.00	1.00	3.00		2.00				8.00
Minimum	0.42	41.50	0.42		0.42				0.42
Maximum	2.40	41.50	38.60		2.80				41.50
Arithmetic Mean	1.41	41.50	13.34		1.61				10.95
Standard Deviation	1.40		21.88		1.68				18.00
Geometric Mean	1.00	41.50	2.53		1.08				2.31
Geometric Standard Deviation	3.43		11.01		3.82				6.80

Table K-1.5 Garden Antimony Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Total
F Horizon								
Number	7.00	2.00	1.00	2.00	9.00	1.00	3.00	25.00
Minimum	0.42	0.48	17.10	1.60	0.85	7.30	0.42	0.42
Maximum	4.20	1.00	17.10	2.80	4.90	7.30	1.10	17.10
Arithmetic Mean	1.59	0.74	17.10	2.20	1.87	7.30	0.77	2.42
Standard Deviation	1.23	0.37		0.85	1.33		0.34	3.45
Geometric Mean	1.28	0.69	17.10	2.12	1.57	7.30	0.72	1.52
Geometric Standard Deviation	2.02	1.68		1.49	1.82		1.63	2.40
G Horizon								
Number	7.00	2.00		1.00	9.00	1.00	3.00	23.00
Minimum	0.42	0.42		35.40	0.42	2.50	0.41	0.41
Maximum	2.20	0.97		35.40	5.20	2.50	0.72	35.40
Arithmetic Mean	1.14	0.70		35.40	1.78	2.50	0.54	2.82
Standard Deviation	0.56	0.39			1.66		0.16	7.20
Geometric Mean	1.03	0.64		35.40	1.25	2.50	0.52	1.18
Geometric Standard Deviation	1.67	1.81			2.41		1.34	2.80

Table K-1.6 Driveway Antimony Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	13.00	5.00	4.00	2.00	14.00	2.00	6.00	3.00	49.00
Minimum	0.89	0.41	2.50	28.00	0.68	9.40	4.10	0.89	0.41
Maximum	46.50	11.40	61.60	147.00	32000.00	10.00	113.00	19.40	32000.00
Arithmetic Mean	10.81	3.46	25.10	87.50	2316.76	9.70	25.13	12.56	675.02
Standard Deviation	14.31	4.54	26.74	84.15	8543.62	0.42	43.13	10.16	4568.41
Geometric Mean	4.60	1.84	13.66	64.16	21.73	9.70	11.15	6.70	9.34
Geometric Standard Deviation	4.04	3.49	4.10	3.23	13.11	1.04	3.33	5.75	6.74
B Horizon									
Number	12.00	5.00	4.00	2.00	13.00	2.00	6.00	3.00	47.00
Minimum	0.99	0.41	3.30	35.90	0.41	2.50	1.90	1.10	0.41
Maximum	464.00	15.90	37.30	61.90	5000.00	13.60	235.00	50.50	5000.00
Arithmetic Mean	46.53	4.96	20.15	48.90	394.39	8.05	46.03	18.67	132.70
Standard Deviation	131.68	6.60	15.59	18.38	1383.84	7.85	92.82	27.62	729.17
Geometric Mean	7.82	2.10	14.13	47.14	9.41	5.83	10.95	6.25	8.26
Geometric Standard Deviation	5.22	4.55	2.99	1.47	10.62	3.31	5.53	6.94	6.13
C Horizon									
Number	12.00	5.00	4.00	2.00	11.00	2.00	6.00	3.00	45.00
Minimum	0.68	0.41	4.20	6.20	0.42	2.80	0.58	3.70	0.41
Maximum	220.00	13.20	46.10	40.20	1690.00	25.60	14.80	34.10	1690.00
Arithmetic Mean	21.35	5.34	15.83	23.20	162.31	14.20	7.65	19.17	51.33
Standard Deviation	62.61	6.52	20.23	24.04	506.78	16.12	5.85	15.21	252.04
Geometric Mean	3.58	1.92	9.45	15.79	8.43	8.47	4.74	13.55	5.65
Geometric Standard Deviation	4.50	5.71	2.95	3.75	9.11	4.78	3.64	3.18	5.24
D Horizon									
Number	11.00	5.00	4.00	2.00	10.00	2.00	6.00	3.00	43.00
Minimum	0.42	0.41	0.42	2.20	0.42	4.10	0.79	2.00	0.41
Maximum	17.40	41.50	50.00	95.50	462.00	22.30	17.60	3.40	462.00
Arithmetic Mean	4.73	8.76	13.76	48.85	57.34	13.20	6.63	2.50	20.83
Standard Deviation	5.62	18.30	24.19	65.97	143.01	12.87	6.61	0.78	71.25
Geometric Mean	2.46	1.29	3.08	14.49	8.57	9.56	3.66	2.43	3.81
Geometric Standard Deviation	3.45	7.17	7.69	14.39	7.50	3.31	3.77	1.34	5.35

Table K-6.1 Yard Soil Zinc Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	15.00	5.00	9.00	5.00	23.00	4.00	13.00	12.00	86.00
Minimum	97.60	51.30	237.00	185.00	58.10	263.00	148.00	94.00	51.30
Maximum	638.00	1820.00	1670.00	3000.00	1400.00	899.00	1440.00	1510.00	3000.00
Arithmetic Mean	281.37	422.84	566.56	1138.20	443.86	577.25	552.46	827.75	543.69
Standard	165.33	781.14	445.45	1099.36	309.57	259.95	418.37	436.21	483.00
Geometric Mean	239.19	137.69	470.83	772.60	349.82	527.99	427.67	646.79	383.50
Geometric Standard	1.82	4.29	1.80	2.80	2.12	1.67	2.11	2.44	2.39
B Horizon									
Number	15.00	5.00	8.00	5.00	24.00	4.00	12.00	12.00	85.00
Minimum	89.40	50.00	257.00	204.00	40.30	195.00	83.90	90.20	40.30
Maximum	549.00	2210.00	2170.00	3310.00	1840.00	586.00	1420.00	12000.00	12000.00
Arithmetic Mean	267.27	496.26	662.50	1177.20	412.30	379.50	495.83	1724.02	655.62
Standard	150.88	958.16	624.69	1265.03	365.79	161.37	418.58	3273.98	1361.00
Geometric Mean	225.41	132.67	520.95	714.19	295.18	352.55	352.47	749.32	351.89
Geometric Standard	1.87	4.92	1.94	3.20	2.45	1.58	2.45	3.48	2.78
C Horizon									
Number	14.00	5.00	8.00	3.00	24.00	4.00	13.00	10.00	81.00
Minimum	56.40	42.60	86.60	275.00	26.60	122.00	41.40	227.00	26.60
Maximum	375.00	1520.00	3000.00	3890.00	882.00	527.00	4140.00	4880.00	4880.00
Arithmetic Mean	164.09	346.62	605.21	1580.67	300.91	322.50	934.72	1535.90	612.79
Standard	108.62	656.10	982.86	2005.63	256.97	174.82	1359.45	1367.72	973.59
Geometric Mean	138.01	101.51	293.51	851.44	210.95	281.66	406.42	1072.97	280.84
Geometric Standard	1.80	4.63	3.18	3.92	2.48	1.89	3.80	2.57	3.30
D Horizon									
Number	12.00	4.00	8.00	3.00	22.00	4.00	12.00	8.00	73.00
Minimum	36.70	41.60	80.50	1110.00	46.30	132.00	36.30	238.00	36.30
Maximum	416.00	590.00	464.00	4490.00	1090.00	219.00	3340.00	3790.00	4490.00
Arithmetic Mean	129.08	188.83	215.58	2256.67	227.60	173.25	606.11	1232.38	460.70
Standard	101.53	267.94	145.82	1934.36	220.63	40.26	919.48	1129.22	795.21
Geometric Mean	105.05	96.71	174.62	1799.91	169.82	169.73	308.27	881.29	222.17
Geometric Standard	1.90	3.45	2.02	2.21	2.12	1.26	3.21	2.46	3.02

Table K-6.2 Vacuum Dust Zinc Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Number	11.00	5.00	8.00	4.00	17.00	1.00	9.00	11.00	66.00
Minimum	327.00	215.00	607.00	675.00	283.00	689.00	527.00	210.00	210.00
Maximum	1070.00	878.00	3490.00	2300.00	1690.00	689.00	1530.00	16000.00	16000.00
Arithmetic Mean	697.18	506.40	1357.38	1518.75	764.29	689.00	907.67	2417.00	1145.05
Standard	235.58	264.60	908.62	672.81	372.23		336.89	4521.36	1928.19
Geometric Mean	658.89	448.80	1182.25	1387.26	681.96	689.00	855.84	1222.30	833.51
Geometric Standard	1.44	1.76	1.68	1.68	1.65		1.44	2.77	1.92

Table K-6.3 Right-of-ways Zinc Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	5.00	3.00	2.00	1.00	3.00	1.00	1.00	1.00	17.00
Minimum	51.00	70.40	387.00	3320.00	128.00	6450.00	255.00	1250.00	51.00
Maximum	615.00	124.00	1830.00	3320.00	1440.00	6450.00	255.00	1250.00	6450.00
Arithmetic Mean	324.64	97.23	1108.50	3320.00	651.33	6450.00	255.00	1250.00	1021.23
Standard Deviation	242.43	26.80	1020.36		695.08				1640.36
Geometric Mean	223.46	94.70	841.55	3320.00	414.37	6450.00	255.00	1250.00	398.73
Geometric Standard Deviation	2.98	1.33	3.00		3.36				4.10
B Horizon									
Number	5.00	3.00	2.00	1.00	3.00	1.00		1.00	16.00
Minimum	89.50	34.00	302.00	7950.00	86.00	4310.00		4420.00	34.00
Maximum	975.00	113.00	2460.00	7950.00	3760.00	4310.00		4420.00	7950.00
Arithmetic Mean	489.10	81.87	1381.00	7950.00	1336.00	4310.00		4420.00	1633.82
Standard Deviation	374.28	42.07	1525.94		2099.59				2319.47
Geometric Mean	355.25	72.36	861.93	7950.00	374.17	4310.00		4420.00	494.13
Geometric Standard Deviation	2.65	1.93	4.41		7.56				5.62
C Horizon									
Number	5.00	3.00	2.00	1.00	2.00	1.00		1.00	15.00
Minimum	165.00	34.50	71.50	14600.00	82.80	7510.00		3390.00	34.50
Maximum	1980.00	91.00	3730.00	14600.00	175.00	7510.00		3390.00	14600.00
Arithmetic Mean	762.80	70.47	1900.75	14600.00	128.90	7510.00		3390.00	2238.98
Standard Deviation	726.94	31.25	2586.95		65.20				4007.60
Geometric Mean	529.99	64.61	516.43	14600.00	120.37	7510.00		3390.00	479.33
Geometric Standard Deviation	2.64	1.72	16.38		1.70				6.80
D Horizon									
Number	4.00	3.00	1.00	1.00	1.00	1.00		1.00	12.00
Minimum	191.00	33.10	1270.00	5560.00	54.30	2480.00		3400.00	33.10
Maximum	2720.00	79.60	1270.00	5560.00	54.30	2480.00		3400.00	5560.00
Arithmetic Mean	1358.50	57.60	1270.00	5560.00	54.30	2480.00		3400.00	1530.93
Standard Deviation	1310.28	23.35							1775.05
Geometric Mean	757.55	54.10	1270.00	5560.00	54.30	2480.00		3400.00	484.81
Geometric Standard Deviation	3.96	1.57							6.64

Table K-6.7 Initial and Purged Drinking Water Zinc Concentrations (ug/L) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
Initial									
Number	15.00	5.00	9.00	4.00	22.00	2.00	13.00	12.00	82.00
Minimum	51.40	30.40	70.40	18.60	26.20	186.00	30.60	12.90	12.90
Maximum	1070.00	997.00	952.00	608.00	1270.00	947.00	8280.00	576.00	8280.00
Arithmetic Mean	397.23	306.68	510.04	171.03	308.81	566.50	914.02	185.43	424.40
Standard Deviation	290.53	395.42	327.25	291.36	336.59	538.11	2223.33	169.81	928.04
Geometric Mean	302.45	159.43	386.93	55.29	183.34	419.69	274.20	112.83	206.62
Geometric Standard Deviation	2.24	3.72	2.43	5.01	2.95	3.16	4.16	3.20	3.29
Purged									
Number	16.00	5.00	8.00	4.00	24.00	2.00	13.00	12.00	84.00
Minimum	12.90	23.30	21.40	12.90	12.90	68.40	19.80	12.90	12.90
Maximum	404.00	846.00	435.00	76.60	1300.00	197.00	438.00	62.90	1300.00
Arithmetic Mean	162.06	238.26	151.95	32.63	130.69	132.70	98.88	30.38	121.22
Standard Deviation	77.88	347.82	134.45	29.54	267.63	90.93	132.94	16.24	184.22
Geometric Mean	139.39	102.14	107.45	25.38	57.25	116.08	59.00	26.65	65.68
Geometric Standard Deviation	2.01	4.26	2.54	2.16	3.17	2.11	2.54	1.71	2.92

Table K-6.4 Play Areas Zinc Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	3.00	1.00	3.00	1.00	3.00		1.00	2.00	14.00
Minimum	97.30	4720.00	266.00	137.00	49.80		652.00	177.00	49.80
Maximum	221.00	4720.00	2410.00	137.00	9640.00		652.00	343.00	9640.00
Arithmetic Mean	176.43	4720.00	1222.33	137.00	3262.90		652.00	260.00	1429.57
Standard Deviation	68.71		1090.56		5522.79			117.38	2691.37
Geometric Mean	165.55	4720.00	859.65	137.00	362.10		652.00	246.40	407.68
Geometric Standard Deviation	1.59		3.03		17.51			1.60	4.75
B Horizon									
Number	3.00	1.00	3.00	1.00	4.00		1.00	1.00	14.00
Minimum	102.00	5190.00	155.00	97.10	48.00		576.00	535.00	48.00
Maximum	145.00	5190.00	2770.00	97.10	3840.00		576.00	535.00	5190.00
Arithmetic Mean	121.33	5190.00	1066.67	97.10	1031.10		576.00	535.00	1006.18
Standard Deviation	21.83		1476.35		1873.01				1663.75
Geometric Mean	120.06	5190.00	490.59	97.10	222.54		576.00	535.00	310.61
Geometric Standard Deviation	1.19		4.60		7.04				4.56
C Horizon									
Number	3.00	1.00	3.00		3.00		1.00		11.00
Minimum	68.30	4630.00	54.20		63.80		267.00		54.20
Maximum	99.30	4630.00	1420.00		561.00		267.00		4630.00
Arithmetic Mean	81.87	4630.00	550.40		260.93		267.00		688.78
Standard Deviation	15.86		755.59		264.10				1367.65
Geometric Mean	80.88	4630.00	238.83		178.16		267.00		217.05
Geometric Standard Deviation	1.21		5.22		2.98				4.17
D Horizon									
Number	2.00	1.00	3.00		2.00				8.00
Minimum	57.40	3660.00	50.70		39.20				39.20
Maximum	70.80	3660.00	1060.00		97.70				3660.00
Arithmetic Mean	64.10	3660.00	410.23		68.45				644.48
Standard Deviation	9.48		563.78		41.37				1266.76
Geometric Mean	63.75	3660.00	186.14		61.89				156.91
Geometric Standard Deviation	1.16		4.79		1.91				5.13

Table K-6.5 Garden Zinc Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Total
F Horizon								
Number	7.00	2.00	1.00	2.00	9.00	1.00	3.00	25.00
Minimum	91.90	62.60	364.00	288.00	62.70	552.00	72.10	62.60
Maximum	2270.00	90.30	364.00	474.00	866.00	552.00	177.00	2270.00
Arithmetic Mean	444.99	76.45	364.00	381.00	339.41	552.00	132.03	335.86
Standard Deviation	805.33	19.59		131.52	250.24		54.03	447.68
Geometric Mean	204.93	75.18	364.00	369.48	264.00	552.00	123.33	217.56
Geometric Standard Deviation	2.96	1.30		1.42	2.19		1.61	2.37
G Horizon								
Number	7.00	2.00		1.00	9.00	1.00	3.00	23.00
Minimum	45.00	60.10		2090.00	51.00	232.00	30.80	30.80
Maximum	1860.00	96.70		2090.00	609.00	232.00	228.00	2090.00
Arithmetic Mean	343.04	78.40		2090.00	206.66	232.00	125.27	309.38
Standard Deviation	669.85	25.88			170.45		98.86	540.74
Geometric Mean	130.42	76.23		2090.00	161.82	232.00	93.66	150.05
Geometric Standard Deviation	3.43	1.40			2.06		2.77	2.87

Table K-6.6 Driveway Zinc Concentrations (mg/kg) in Geographic Sub-areas, Fall 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Total
A Horizon									
Number	13.00	5.00	4.00	2.00	14.00	2.00	6.00	3.00	49.00
Minimum	26.60	62.20	429.00	3340.00	64.30	788.00	220.00	477.00	26.60
Maximum	2420.00	1110.00	1880.00	5970.00	9090.00	927.00	1720.00	4230.00	9090.00
Arithmetic Mean	705.24	310.84	1124.75	4655.00	2010.32	857.50	859.33	1955.67	1334.98
Standard Deviation	698.76	450.01	593.49	1859.69	2291.86	98.29	615.33	1999.02	1654.16
Geometric Mean	384.27	162.04	991.61	4465.40	966.52	854.68	681.40	1327.72	653.89
Geometric Standard Deviation	3.74	3.19	1.85	1.51	4.56	1.12	2.16	3.00	3.88
B Horizon									
Number	12.00	5.00	4.00	2.00	13.00	2.00	6.00	3.00	47.00
Minimum	113.00	19.90	490.00	4310.00	39.10	569.00	244.00	344.00	19.90
Maximum	3990.00	2980.00	1930.00	6250.00	3070.00	1440.00	3700.00	1210.00	6250.00
Arithmetic Mean	1284.50	640.10	1235.00	5280.00	1258.12	1004.50	1206.17	816.67	1322.68
Standard Deviation	1362.28	1308.74	602.91	1371.79	1149.11	615.89	1416.16	438.42	1411.74
Geometric Mean	687.33	99.05	1102.45	5190.13	580.25	905.19	687.31	719.81	614.48
Geometric Standard Deviation	3.51	7.60	1.80	1.30	4.82	1.93	3.09	1.93	4.37
C Horizon									
Number	12.00	5.00	4.00	2.00	11.00	2.00	6.00	3.00	45.00
Minimum	100.00	29.40	238.00	1480.00	32.50	242.00	130.00	507.00	29.40
Maximum	1320.00	1900.00	2450.00	2470.00	1770.00	246.00	2320.00	974.00	2470.00
Arithmetic Mean	430.50	434.98	1141.00	1975.00	721.83	244.00	802.67	756.67	697.09
Standard Deviation	403.73	819.91	976.24	700.04	601.53	2.83	912.98	235.17	698.59
Geometric Mean	296.61	118.35	814.81	1911.96	393.72	243.99	454.85	730.38	380.17
Geometric Standard Deviation	2.41	5.17	2.74	1.44	4.05	1.01	3.18	1.40	3.40
D Horizon									
Number	11.00	5.00	4.00	2.00	10.00	2.00	6.00	3.00	43.00
Minimum	69.00	33.80	98.40	370.00	32.20	459.00	140.00	201.00	32.20
Maximum	1110.00	1170.00	1240.00	2560.00	1730.00	774.00	1800.00	765.00	2560.00
Arithmetic Mean	402.09	294.56	649.35	1465.00	533.38	616.50	780.83	566.67	566.86
Standard Deviation	372.19	491.91	544.00	1548.56	534.10	222.74	725.37	317.06	570.89
Geometric Mean	272.83	112.34	429.53	973.24	309.32	596.04	494.57	483.26	328.81
Geometric Standard Deviation	2.51	4.22	3.23	3.93	3.40	1.45	3.04	2.14	3.13

APPENDIX L

Summary Statistics for Antimony, Arsenic, Cadmium, Iron, Manganese, and Zinc for the Summer 1999 Coeur d'Alene Basin Sampling Event

Appendix L

Summary Statistics for Antimony, Arsenic, Cadmium, Iron, Manganese, and Zinc for the Summer 1999 Coeur d'Alene Basin Sampling Event

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This appendix contains summary tables for the Summer 1999 sampling event for the Coeur d'Alene Basin. Metals included in these tables are antimony, arsenic, cadmium, iron, manganese, and zinc. Sample media from this event includes yard soil, vacuum dust, play areas, garden soil, driveway, and initial and purged drinking water from homes in Harrison and Rocky Point. Right-of-ways data is shown for all Coeur d'Alene Basin geographic subdivisions.

Table L-1.1 Yard Soil Antimony Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison / Rocky Piont	Total
A Horizon		
Number	25.00	25.00
Minimum	0.42	0.42
Maximum	1.80	1.80
Arithmetic Mean	1.02	1.02
Standard Deviation	0.44	0.44
Geometric Mean	0.92	0.92
Geometric Standard Deviation	1.63	1.63
B Horizon		
Number	25.00	25.00
Minimum	0.42	0.42
Maximum	2.30	2.30
Arithmetic Mean	1.03	1.03
Standard Deviation	0.56	0.56
Geometric Mean	0.87	0.87
Geometric Standard Deviation	1.83	1.83
C Horizon		
Number	23.00	23.00
Minimum	0.42	0.42
Maximum	1.90	1.90
Arithmetic Mean	1.00	1.00
Standard Deviation	0.53	0.53
Geometric Mean	0.86	0.86
Geometric Standard Deviation	1.79	1.79

Table L-1.2 Vacuum Dust Antimony Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison / Rocky Piont	Total
Number	20.00	20.00
Minimum	0.43	0.43
Maximum	18.00	18.00
Arithmetic Mean	4.11	4.11
Standard Deviation	4.11	4.11
Geometric Mean	2.88	2.88
Geometric Standard Deviation	2.37	2.37

Table L-1.3 Right-of-way Antimony Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Harrison / Rocky Piont	Total
A Horizon										
Number	5.00	7.00	8.00	6.00	5.00		2.00	7.00	20.00	60.00
Minimum	7.10	0.52	3.90	13.10	15.90		9.60	6.20	0.41	0.41
Maximum	41.90	12.10	26.10	37.20	137.00		34.90	42.90	2.30	137.00
Arithmetic Mean	18.90	4.42	15.10	21.77	66.74		22.25	16.31	0.99	14.82
Standard Deviation	14.05	4.61	7.59	9.23	57.98		17.89	12.25	0.59	24.35
Geometric Mean	15.38	2.67	12.99	20.27	46.55		18.30	13.67	0.83	5.19
Geometric Standard Deviation	2.03	3.06	1.90	1.51	2.66		2.49	1.83	1.86	5.05
B Horizon										
Number	4.00	5.00	8.00	6.00	6.00		1.00	7.00	22.00	59.00
Minimum	2.50	0.52	2.20	7.40	28.70		5.50	14.20	0.41	0.41
Maximum	91.60	8.50	19.10	34.00	59.70		5.50	42.20	2.10	91.60
Arithmetic Mean	48.45	3.08	12.50	24.80	44.37		5.50	24.23	1.02	15.62
Standard Deviation	37.51	3.13	5.57	9.63	11.87			12.22	0.59	19.69
Geometric Mean	27.12	2.09	10.68	22.38	42.99		5.50	21.95	0.85	5.16
Geometric Standard Deviation	5.09	2.74	2.03	1.76	1.32			1.59	1.90	5.62
C Horizon										
Number	5.00	6.00	6.00	7.00	7.00		2.00	6.00	18.00	57.00
Minimum	1.00	0.42	0.73	1.20	3.00		8.10	18.40	0.42	0.42
Maximum	39.90	8.10	24.90	173.00	110.00		11.10	48.80	3.10	173.00
Arithmetic Mean	13.10	3.64	15.22	47.39	28.54		9.60	29.23	1.03	16.20
Standard Deviation	15.45	3.03	9.23	57.35	38.48		2.12	10.93	0.71	28.28
Geometric Mean	7.21	2.44	10.09	24.30	14.61		9.48	27.74	0.84	4.98
Geometric Standard Deviation	3.79	2.97	3.80	4.51	3.39		1.25	1.41	1.89	5.42

Table L-1.4 Driveway Antimony Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison / Rocky Piont	Total
A Horizon		
Number	16.00	16.00
Minimum	0.42	0.42
Maximum	2.90	2.90
Arithmetic Mean	1.38	65.56
Standard Deviation	0.87	0.87
Geometric Mean	1.10	1.10
Geometric Standard Deviation	2.08	2.08
B Horizon		
Number	17.00	17.00
Minimum	0.41	0.41
Maximum	2.30	2.30
Arithmetic Mean	1.29	1.29
Standard Deviation	0.73	0.73
Geometric Mean	1.06	1.06
Geometric Standard Deviation	2.00	2.00
C Horizon		
Number	16.00	16.00
Minimum	0.41	0.41
Maximum	2.10	2.10
Arithmetic Mean	1.15	1.15
Standard Deviation	0.48	0.48
Geometric Mean	1.03	1.03
Geometric Standard Deviation	1.66	1.66

Table L-1.5 Garden Antimony Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison / Rocky Piont	Total
F Horizon		
Number	9.00	9.00
Minimum	0.42	0.42
Maximum	3.50	3.50
Arithmetic Mean	1.42	1.42
Standard Deviation	0.99	0.99
Geometric Mean	1.13	1.13
Geometric Standard Deviation	2.09	2.09
G Horizon		
Number	7.00	7.00
Minimum	0.57	0.57
Maximum	2.20	2.20
Arithmetic Mean	1.30	1.30
Standard Deviation	0.50	0.50
Geometric Mean	1.21	1.21
Geometric Standard Deviation	1.52	1.52

Table L-1.6 Initial and Purged Drinking Water Antimony Concentrations (ug/L) in Geographic Sub-areas, Summer 1999

	Harrison / Rocky Piont	Total
Initial		
Number	22.00	22.00
Minimum	2.10	2.10
Maximum	2.10	2.10
Arithmetic Mean	2.10	2.10
Standard Deviation	0.00	0.00
Geometric Mean	2.10	2.10
Geometric Standard Deviation	1.00	1.00
Purged		
Number	25.00	25.00
Minimum	2.10	2.10
Maximum	5.00	5.00
Arithmetic Mean	2.22	2.22
Standard Deviation	0.58	0.58
Geometric Mean	2.17	2.17
Geometric Standard Deviation	1.19	1.19

Table L-2.1 Yard Soil Arsenic Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
A Horizon		
Number	25	25
Minimum	3.500	3.500
Maximum	8.800	8.800
Arithmetic Mean	5.520	5.520
Standard Deviation	1.239	1.239
Geometric Mean	5.392	5.392
Geometric Standard Deviation	1.25	1.25
B Horizon		
Number	25	25
Minimum	3.8	3.8
Maximum	10.4	10.4
Arithmetic Mean	5.9	5.9
Standard Deviation	1.4	1.4
Geometric Mean	5.8	5.8
Geometric Standard Deviation	1.24	1.24
C Horizon		
Number	23	23
Minimum	4.4	4.4
Maximum	12.2	12.2
Arithmetic Mean	6.4	6.4
Standard Deviation	2.1	2.1
Geometric Mean	6.1	6.1
Geometric Standard Deviation	1.33	1.33

Table L-2.2 Vacuum Dust Arsenic Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
Number	20	20
Minimum	1.300	1.300
Maximum	41.600	41.600
Arithmetic Mean	9.015	9.015
Standard Deviation	9.162	9.162
Geometric Mean	6.273	6.273
Geometric Standard Deviation	2.39	2.39

Table L-2.3 Right-of-way Arsenic Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Harrison/Rocky Point	Total
A Horizon										
Number	5	7	8	6	5		2	7	20	60
Minimum	23.8	4.6	8.3	16.5	47.6		28.2	23.1	1.6	1.6
Maximum	47.6	40.6	49.3	63.9	719.0		78.5	62.2	5.6	719.0
Arithmetic Mean	37.2	13.6	30.8	43.4	312.7		53.4	38.6	3.4	46.6
Standard Deviation	9.04	14.20	14.62	17.42	349.44		35.57	13.81	1.04	123.34
Geometric Mean	36.2	9.2	26.8	39.8	155.6		47.0	36.6	3.2	15.2
Geometric Standard Deviation	1.30	2.43	1.87	1.64	3.94		2.06	1.41	1.40	4.09
B Horizon										
Number	4	5	8	6	6		1	7	22	59
Minimum	17.2	4.5	9.6	19.8	46.8		27.5	31.9	1.8	1.8
Maximum	126.0	32.4	111.0	133.0	299.0		27.5	91.0	9.9	299.0
Arithmetic Mean	71.6	11.9	36.3	50.1	151.2		27.5	54.8	4.0	39.7
Standard Deviation	45.57	11.92	31.77	42.90	97.09			22.02	2.06	56.66
Geometric Mean	57.3	8.6	28.4	39.7	124.9		27.5	51.1	3.6	16.0
Geometric Standard Deviation	2.36	2.34	2.03	2.01	2.01			1.50	1.59	4.20
C Horizon										
Number	5	6	6	7	7		2	6	18	57
Minimum	9.3	9.1	6.4	10.4	19.1		20.4	27.5	2.4	2.4
Maximum	75.9	52.9	48.9	370.0	228.0		33.1	102.0	10.0	370.0
Arithmetic Mean	36.1	19.7	28.1	76.3	76.2		26.8	64.0	5.5	36.3
Standard Deviation	25.41	17.38	14.45	130.00	80.12		8.98	26.74	2.65	59.03
Geometric Mean	28.8	15.4	23.9	36.3	0.0	0.0	26.0	58.8	4.9	17.9
Geometric Standard Deviation	2.20	2.04	2.03	3.10	2.63		1.41	1.61	1.60	3.23

Table L-2.4 Driveway Arsenic Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison / Rocky Point	Total
A Horizon		
Number	16	16
Minimum	1.5	1.5
Maximum	8.5	8.5
Arithmetic Mean	4.7	4.7
Standard Deviation	2.0	2.0
Geometric Mean	4.2	4.2
Geometric Standard Deviation	1.63	1.63
B Horizon		
Number	17	17
Minimum	1.9	1.9
Maximum	6.6	6.6
Arithmetic Mean	4.1	4.1
Standard Deviation	1.5	1.5
Geometric Mean	3.9	3.9
Geometric Standard Deviation	1.44	1.44
C Horizon		
Number	16	16
Minimum	3.3	3.3
Maximum	8.5	8.5
Arithmetic Mean	5.4	5.4
Standard Deviation	1.6	1.6
Geometric Mean	5.2	5.2
Geometric Standard Deviation	1.33	1.33

Table L-2.5 Garden Arsenic Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
F Horizon		
Number	9	9
Minimum	4.0	4.0
Maximum	8.5	8.5
Arithmetic Mean	6.2	6.2
Standard Deviation	1.4	1.4
Geometric Mean	6.0	6.0
Geometric Standard Deviation	1.26	1.26
G Horizon		
Number	7	7
Minimum	5.0	5.0
Maximum	16.2	16.2
Arithmetic Mean	7.7	7.7
Standard Deviation	3.9	3.9
Geometric Mean	7.1	7.1
Geometric Standard Deviation	1.48	1.48

Table L-2.6 Initial and Purged Drinking Water Arsenic Concentrations (ug/L) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
Initial		
Number	22	22
Minimum	2.2	2.2
Maximum	5.6	5.6
Arithmetic Mean	3.0	3.0
Standard Deviation	0.9	0.9
Geometric Mean	2.9	2.9
Geometric Standard Deviation	1.31	1.31
Purged		
Number	25	25
Minimum	2.2	2.2
Maximum	8.5	8.5
Arithmetic Mean	3.2	3.2
Standard Deviation	1.4	1.4
Geometric Mean	2.9	2.9
Geometric Standard Deviation	1.42	1.42

Table L-3.1 Yard Soil Cadmium Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison / Rocky Point	Total
A Horizon		
Number	25	25
Minimum	0.1	0.1
Maximum	1.0	1.0
Arithmetic Mean	0.2	0.2
Standard Deviation	0.2	0.2
Geometric Mean	0.2	0.2
Geometric Standard Deviation	1.95	1.95
B Horizon		
Number	25	25
Minimum	0.1	0.1
Maximum	1.2	1.2
Arithmetic Mean	0.3	0.3
Standard Deviation	0.3	0.3
Geometric Mean	0.2	0.2
Geometric Standard Deviation	2.15	2.15
C Horizon		
Number	23	23
Minimum	0.1	0.1
Maximum	1.7	1.7
Arithmetic Mean	0.2	0.2
Standard Deviation	0.3	0.3
Geometric Mean	0.2	0.2
Geometric Standard Deviation	2.14	2.14

Table L-3.2 Vacuum Dust Cadmium Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
Number	20	20
Minimum	0.7	0.7
Maximum	66.3	66.3
Arithmetic Mean	7.4	7.4
Standard Deviation	15.3	15.3
Geometric Mean	2.9	2.9
Geometric Standard Deviation	3.21	3.21

Table L-3.3 Right-of-way Cadmium Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Harrison/Rocky Point	Total
A Horizon										
Number	5	7	8	6	5		2	7	20	60
Minimum	1.5	0.1	1.0	3.6	0.1		1.6	1.1	0.1	0.1
Maximum	3.6	8.2	12.4	32.1	35.2		5.7	17.3	0.7	35.2
Arithmetic Mean	2.5	1.4	6.8	13.5	12.3		3.7	7.8	0.2	4.7
Standard Deviation	0.80	3.01	3.94	11.68	14.68		2.90	5.13	0.14	7.42
Geometric Mean	2.4	0.3	5.3	9.9	2.3		3.0	6.0	0.1	1.0
Geometric Standard Deviation	1.40	5.60	2.40	2.38	17.28		2.46	2.39	2.02	8.01
B Horizon										
Number	4	5	8	6	6		1	7	22	59
Minimum	3.8	0.1	0.2	4.1	9.7		8.4	2.4	0.1	0.1
Maximum	12.3	1.3	18.7	15.7	45.8		8.4	18.5	0.4	45.8
Arithmetic Mean	6.7	0.4	8.5	9.3	20.5		8.4	9.2	0.2	6.0
Standard Deviation	3.83	0.51	5.86	5.13	13.90			5.50	0.10	8.23
Geometric Mean	6.0	0.2	5.3	8.1	17.4		8.4	7.6	0.1	1.3
Geometric Standard Deviation	1.66	3.01	4.19	1.82	1.82			2.05	1.93	8.85
C Horizon										
Number	5	6	6	7	7		2	6	18	57
Minimum	0.6	0.1	0.1	0.2	2.9		9.0	6.1	0.1	0.1
Maximum	8.7	5.3	40.3	28.5	33.9		10.7	20.0	1.3	40.3
Arithmetic Mean	4.1	1.9	13.4	16.8	17.1		9.9	13.4	0.2	8.0
Standard Deviation	3.01	2.27	14.21	11.34	12.31		1.20	5.84	0.29	10.18
Geometric Mean	3.0	0.7	5.1	8.5	12.3		9.8	12.3	0.2	1.8
Geometric Standard Deviation	2.67	6.34	9.80	6.30	2.67		1.13	1.63	2.39	9.19

Table L-3.4 Driveway Cadmium Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
A Horizon		
Number	16	16
Minimum	0.1	0.1
Maximum	0.7	0.7
Arithmetic Mean	0.2	0.2
Standard Deviation	0.2	0.2
Geometric Mean	0.2	0.2
Geometric Standard Deviation	2.26	2.26
B Horizon		
Number	17	17
Minimum	0.1	0.1
Maximum	0.4	0.4
Arithmetic Mean	0.2	0.2
Standard Deviation	0.1	0.1
Geometric Mean	0.1	0.1
Geometric Standard Deviation	1.87	1.87
C Horizon		
Number	16	16
Minimum	0.1	0.1
Maximum	0.5	0.5
Arithmetic Mean	0.2	0.2
Standard Deviation	0.1	0.1
Geometric Mean	0.2	0.2
Geometric Standard Deviation	1.77	1.77

Table L-3.5 Garden Cadmium Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
F Horizon		
Number	9	9
Minimum	0.1	0.1
Maximum	0.9	0.9
Arithmetic Mean	0.3	0.3
Standard Deviation	0.3	0.3
Geometric Mean	0.2	0.2
Geometric Standard Deviation	2.27	2.27
G Horizon		
Number	7	7
Minimum	0.2	0.2
Maximum	0.9	0.9
Arithmetic Mean	0.3	0.3
Standard Deviation	0.3	0.3
Geometric Mean	0.3	0.3
Geometric Standard Deviation	1.82	1.82

Table L-3.6 Initial and Purged Drinking Water Cadmium Concentrations (ug/L) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
Initial		
Number	22	22
Minimum	0.3	0.3
Maximum	10.9	10.9
Arithmetic Mean	0.9	0.9
Standard Deviation	2.3	2.3
Geometric Mean	0.5	0.5
Geometric Standard Deviation	2.42	2.42
Purged		
Number	25	25
Minimum	0.3	0.3
Maximum	1.0	1.0
Arithmetic Mean	0.4	0.4
Standard Deviation	0.2	0.2
Geometric Mean	0.4	0.4
Geometric Standard Deviation	1.43	1.43

Table L-4.1 Yard Soil Iron Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
A Horizon		
Number	25	25
Minimum	12400	12400
Maximum	22700	22700
Arithmetic Mean	17520	17520
Standard Deviation	2674.10	2674.10
Geometric Mean	17326	17326
Geometric Standard Deviation	1.17	1.17
B Horizon		
Number	25	25
Minimum	11500	11500
Maximum	25700	25700
Arithmetic Mean	18624	18624
Standard Deviation	3398.93	3398.93
Geometric Mean	18314	18314
Geometric Standard Deviation	1.21	1.21
C Horizon		
Number	23	23
Minimum	13100	13100
Maximum	27600	27600
Arithmetic Mean	20139	20139
Standard Deviation	4125.94	4125.94
Geometric Mean	19737	19737
Geometric Standard Deviation	1.23	1.23

Table L-4.2 Vacuum Dust Iron Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
Number	20	20
Minimum	3590	3590
Maximum	19500	19500
Arithmetic Mean	12271	12271
Standard Deviation	4127.71	4127.71
Geometric Mean	11483	11483
Geometric Standard Deviation	1.49	1.49

Table L-4.3 Right-of-way Iron Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Harrison/Rocky Point	Total
A Horizon										
Number	5	7	8	6	5		2	7	20	60
Minimum	20400	7810	21900	20100	31600		27500	17500	12800	7810
Maximum	28100	39300	52200	40100	89700		63200	36000	35800	89700
Arithmetic Mean	24360	27459	30200	30183	54960		45350	28557	21865	28990
Standard Deviation	2770.02	12640.83	10596.90	8002.10	24072.25		25243.71	6235.08	6692.36	13779.75
Geometric Mean	24232	23817	28845	29261	50991		41689	27897	20917	26449
Geometric Standard Deviation	1.12	1.90	1.37	1.32	1.54		1.80	1.27	1.36	1.53
B Horizon										
Number	4	5	8	6	6		1	7	22	59
Minimum	21900	18300	20400	19600	33900		31500	22400	3940	3940
Maximum	58600	44000	50000	67800	70200		31500	45600	34500	70200
Arithmetic Mean	32500	28400	33475	43017	47833		31500	31886	20956	30519
Standard Deviation	17477.03	10695.09	11383.79	19330.02	12392.20			8247.71	7363.98	13978.90
Geometric Mean	29760	26912	31751	39370	46619		31500	30999	19316	27444
Geometric Standard Deviation	1.58	1.44	1.42	1.60	1.28		1.00	1.29	1.59	1.63
C Horizon										
Number	5	6	6	7	7		2	6	18	57
Minimum	20900	17900	16900	21500	22300		23600	25300	2750	2750
Maximum	32300	36400	64500	108000	117000		38800	47800	34300	117000
Arithmetic Mean	25740	27583	35083	41971	42429		31200	33917	22197	30894
Standard Deviation	4992.29	7182.32	16286.00	29483.37	33485.01		10748.02	9523.74	7804.83	18294.25
Geometric Mean	25367	26781	32265	36605	35753		30260	32889	20081	27423
Geometric Standard Deviation	1.21	1.31	1.56	1.66	1.76		1.42	1.31	1.75	1.65

Table L-4.4 Driveway Iron Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
A Horizon		
Number	16	16
Minimum	18500	18500
Maximum	62900	62900
Arithmetic Mean	32169	32169
Standard Deviation	11960.59	11960.59
Geometric Mean	30389	30389
Geometric Standard Deviation	1.40	1.40
B Horizon		
Number	17	17
Minimum	11200	11200
Maximum	59300	59300
Arithmetic Mean	30635	30635
Standard Deviation	12129.71	12129.71
Geometric Mean	28430	28430
Geometric Standard Deviation	1.50	1.50
C Horizon		
Number	16	16
Minimum	13400	13400
Maximum	31600	31600
Arithmetic Mean	23013	23013
Standard Deviation	5535.93	5535.93
Geometric Mean	22373	22373
Geometric Standard Deviation	1.28	1.28

Table L-4.5 Garden Iron Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
F Horizon		
Number	9	9
Minimum	15000	15000
Maximum	22800	22800
Arithmetic Mean	19178	19178
Standard Deviation	2745.35	2745.35
Geometric Mean	18995	18995
Geometric Standard Deviation	1.16	1.16
G Horizon		
Number	7	7
Minimum	14800	14800
Maximum	29000	29000
Arithmetic Mean	19814	19814
Standard Deviation	4718.56	4718.56
Geometric Mean	19382	19382
Geometric Standard Deviation	1.25	1.25

Table L-4.6 Initial and Purged Drinking Water Iron Concentrations (ug/L) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
Initial		
Number	22	22
Minimum	19	19
Maximum	5250	5250
Arithmetic Mean	395	395
Standard Deviation	1104.28	1104.28
Geometric Mean	96	96
Geometric Standard Deviation	4.58	4.58
Purged		
Number	25	25
Minimum	19	19
Maximum	339	339
Arithmetic Mean	108	108
Standard Deviation	100.11	100.11
Geometric Mean	69	69
Geometric Standard Deviation	2.71	2.71

Table L-5.1 Yard Soil Manganese Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
A Horizon		
Number	25	25
Minimum	263	263
Maximum	794	794
Arithmetic Mean	488	488
Standard Deviation	116.62	116.62
Geometric Mean	474	474
Geometric Standard Deviation	1.28	1.28
B Horizon		
Number	25	25
Minimum	268	268
Maximum	677	677
Arithmetic Mean	499	499
Standard Deviation	103.68	103.68
Geometric Mean	487	487
Geometric Standard Deviation	1.27	1.27
C Horizon		
Number	23	23
Minimum	284	284
Maximum	801	801
Arithmetic Mean	500	500
Standard Deviation	136.85	136.85
Geometric Mean	483	483
Geometric Standard Deviation	1.31	1.31

Table L-5.2 Vacuum Dust Manganese Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
Number	20	20
Minimum	114	114
Maximum	726	726
Arithmetic Mean	323	323
Standard Deviation	142.88	142.88
Geometric Mean	296	296
Geometric Standard Deviation	1.54	1.54

Table L-5.3 Right-of-way Manganese Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/Burke	Osburn	Side Gulches	Silverton	Wallace	Harrison/Rocky Point	Total
A Horizon										
Number	5	7	8	6	5		2	7	20	60
Minimum	1040	275	1250	1350	2540		747	716	293	275
Maximum	1330	1470	4030	2600	4960		3490	2180	564	4960
Arithmetic Mean	1142	764	2019	2058	3574		2119	1542	451	1358
Standard Deviation	116.92	441.83	981.84	550.36	1196.53		1939.59	518.00	72.10	1106.51
Geometric Mean	1137	652	1856	1992	3421		1615	1454	445	1004
Geometric Standard Deviation	1.10	1.87	1.52	1.33	1.39		2.97	1.48	1.18	2.20
B Horizon										
Number	4	5	8	6	6		1	7	22	59
Minimum	1320	460	467	1230	2180		1990	866	101	101
Maximum	3920	1590	3130	3970	7990		1990	2370	1100	7990
Arithmetic Mean	2045	929	2102	2447	3895		1990	1797	487	1576
Standard Deviation	1252.03	412.23	916.67	1162.66	2172.75			563.41	179.71	1394.65
Geometric Mean	1829	860	1831	2215	3505		1990	1706	454	1104
Geometric Standard Deviation	1.67	1.56	1.91	1.64	1.61			1.44	1.53	2.41
C Horizon										
Number	5	6	6	7	7		2	6	18	57
Minimum	857	416	290	567	781		1090	866	31	31
Maximum	2070	2340	3750	5680	9820		2210	3010	1000	9820
Arithmetic Mean	1413	1086	2205	2648	3175		1650	1849	479	1589
Standard Deviation	514.55	710.01	1320.42	2084.25	3080.87		791.96	742.49	206.93	1644.48
Geometric Mean	1340	922	1690	2009	2277		1552	1718	409	1050
Geometric Standard Deviation	1.44	1.86	2.59	2.28	2.39		1.65	1.54	2.08	2.63

Table L-5.4 Driveway Manganese Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
A Horizon		
Number	16	16
Minimum	440	440
Maximum	1110	1110
Arithmetic Mean	715	715
Standard Deviation	210.53	210.53
Geometric Mean	688	688
Geometric Standard Deviation	1.33	1.33
B Horizon		
Number	17	17
Minimum	254	254
Maximum	1330	1330
Arithmetic Mean	683	683
Standard Deviation	252.96	252.96
Geometric Mean	641	641
Geometric Standard Deviation	1.45	1.45
C Horizon		
Number	16	16
Minimum	323	323
Maximum	1060	1060
Arithmetic Mean	551	551
Standard Deviation	177.11	177.11
Geometric Mean	527	527
Geometric Standard Deviation	1.35	1.35

Table L-5.5 Garden Manganese Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
F Horizon		
Number	9	9
Minimum	422	422
Maximum	758	758
Arithmetic Mean	590	590
Standard Deviation	126.31	126.31
Geometric Mean	578	578
Geometric Standard Deviation	1.25	1.25
G Horizon		
Number	7	7
Minimum	320	320
Maximum	679	679
Arithmetic Mean	514	514
Standard Deviation	126.73	126.73
Geometric Mean	500	500
Geometric Standard Deviation	1.30	1.30

Table L-5.6 Initial and Purged Drinking Water Manganese Concentrations (ug/L) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
Initial		
Number	22	22
Minimum	2	2
Maximum	225	225
Arithmetic Mean	54	54
Standard Deviation	60.81	60.81
Geometric Mean	22	22
Geometric Standard Deviation	5.00	5.00
Purged		
Number	25	25
Minimum	2	2
Maximum	160	160
Arithmetic Mean	40	40
Standard Deviation	53.91	53.91
Geometric Mean	13	13
Geometric Standard Deviation	4.90	4.90

Table L-6.1 Yard Soil Zinc Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
A Horizon		
Number	16	16
Minimum	1	1
Maximum	512	512
Arithmetic Mean	55	55
Standard Deviation	126.37	126.37
Geometric Mean	9	9
Geometric Standard Deviation	7.41	7.41
B Horizon		
Number	10	10
Minimum	1	1
Maximum	57	57
Arithmetic Mean	16	16
Standard Deviation	21.71	21.71
Geometric Mean	5	5
Geometric Standard Deviation	5.38	5.38
C Horizon		
Number	12	12
Minimum	1	1
Maximum	97	97
Arithmetic Mean	28	28
Standard Deviation	34.16	34.16
Geometric Mean	11	11
Geometric Standard Deviation	4.80	4.80

Table L-6.2 Vacuum Dust Zinc Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
Number	3	3
Minimum	3	3
Maximum	386	386
Arithmetic Mean	214	214
Standard Deviation	194.46	194.46
Geometric Mean	66	66
Geometric Standard Deviation	14.74	14.74

Table L-6.3 Right-of-way Zinc Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Kingston	Lower Basin/Cataldo	Mullan	Nine Mile/burke	Osburn	Side Gulches	Silverton	Wallace	Harrison/Rocky Point	Total
A Horizon										
Number	0	6	2	0	1		1	2	19	31
Minimum	0	83	1670	0	1		561	16	1	1
Maximum	0	713	1780	0	1		561	372	148	1780
Arithmetic Mean	0	253	1725	0	1		561	194	68	232
Standard Deviation	0.00	240.62	77.78	0.00	0.00		0.00	251.73	36.20	428.94
Geometric Mean	1	185	1724	1	1		561	77	50	80
Geometric Standard Deviation	1.00	2.29	1.05	1.00	1.00		1.00	9.25	3.05	5.17
B Horizon										
Number	0	5	1	0	0		0	1	20	27
Minimum	0	7	273	0	0		0	59	1	1
Maximum	0	740	273	0	0		0	59	181	740
Arithmetic Mean	0	260	273	0	0		0	59	60	105
Standard Deviation	0.00	283.84	0.00	0.00	0.00		0.00	0.00	51.93	147.46
Geometric Mean	1	122	273	1	1		1	59	30	44
Geometric Standard Deviation	1.00	5.73	1.00	1.00	1.00		1.00	1.00	4.76	5.15
C Horizon										
Number	1	5	1	1	2		0	0	10	20
Minimum	10	9	136	9	11		0	0	11	9
Maximum	10	614	136	9	698		0	0	149	698
Arithmetic Mean	10	206	136	9	355		0	0	51	120
Standard Deviation	0.00	236.70	0.00	0.00	485.78		0.00	0.00	43.32	191.50
Geometric Mean	10	104	136	9	88		1	1	35	48
Geometric Standard Deviation	1.00	4.69	1.00	1.00	18.82		1.00	1.00	2.56	4.05

Table L-6.4 Driveway Zinc Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
A Horizon		
Number	13	13
Minimum	1	1
Maximum	106	106
Arithmetic Mean	56	56
Standard Deviation	44.08	44.08
Geometric Mean	20	20
Geometric Standard Deviation	7.73	7.73
B Horizon		
Number	12	12
Minimum	1	1
Maximum	91	91
Arithmetic Mean	38	38
Standard Deviation	33.13	33.13
Geometric Mean	16	16
Geometric Standard Deviation	6.20	6.20
C Horizon		
Number	10	10
Minimum	1	1
Maximum	49	49
Arithmetic Mean	17	17
Standard Deviation	19.55	19.55
Geometric Mean	8	8
Geometric Standard Deviation	3.99	3.99

Table L-6.5 Garden Zinc Concentrations (mg/kg) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
F Horizon		
Number	4	4
Minimum	1	1
Maximum	77	77
Arithmetic Mean	23	23
Standard Deviation	36.54	36.54
Geometric Mean	6	6
Geometric Standard Deviation	8.27	8.27
G Horizon		
Number	2	2
Minimum	1	1
Maximum	93	93
Arithmetic Mean	47	47
Standard Deviation	65.05	65.05
Geometric Mean	10	10
Geometric Standard Deviation	24.66	24.66

Table L-6.6 Initial and Purged Drinking Water Zinc Concentrations (ug/L) in Geographic Sub-areas, Summer 1999

	Harrison/Rocky Point	Total
Initial		
Number	22	22
Minimum	1	1
Maximum	1660	1660
Arithmetic Mean	219	219
Standard Deviation	468.40	468.40
Geometric Mean	52	52
Geometric Standard Deviation	5.94	5.94
Purged		
Number	25	25
Minimum	4	4
Maximum	1130	1130
Arithmetic Mean	144	144
Standard Deviation	283.64	283.64
Geometric Mean	44	44
Geometric Standard Deviation	4.22	4.22

APPENDIX M

QA/QC Memoranda for the Summer and Fall 1999 Coeur d'Alene Basin Sampling Events

Appendix M

QA/QC Memoranda for the Summer and Fall 1999 Coeur d'Alene Basin Sampling Events

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This appendix contains three QA/QC memoranda for the Summer and Fall 1999 sampling events for the Coeur d'Alene Basin. The memos outline QA/QC procedures and results for reanalysis of the 1996 IDHW samples, 1999 samples analyzed through EPA Contract Lab Program (CLP) laboratories, and 1999 low weight samples that were analyzed at a local Idaho laboratory because they did not have sufficient amount to be processed through CLP.

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M E M O R A N D U M

To: Gerald B. Lee, TerraGraphics, Moscow

From: Shanda LeVan, TerraGraphics, Moscow

Date: February 17, 2000

Subject: QA/QC Review for the 1996 Basin Archive samples analyzed by Anatek Labs during Fall 1999.

Introduction

The following memorandum summarizes the quality assurance/quality control (QA/QC) review for the 1996 Basin Archive samples analyzed by Anatek Labs during the Fall of 1999. These samples were re-analyzed for 24 metals. No field QA/QC samples were included in the samples sent to Anatek for analysis. Only internal QA/QC for laboratory analysis is available.

General

A QA/QC review was done to evaluate the precision, accuracy, completeness, and representativeness of the data obtained from the laboratory. Procedures for sample labeling, handling, and analysis were as described in the Final Field Work Plan for the Coeur d'Alene River Basin Environmental Health Exposure Assessment (TerraGraphics, 1996). This data validation review indicated all samples were properly labeled and tracked during the project and throughout the archive process. In the fall of 1999, 65 prioritized samples were selected from this archive collection and sent to Anatek Labs, Inc. in Moscow, Idaho for total metal analysis.

Two of the lab's QC batches were below percent recovery for matrix spike/matrix spike duplicate (MS/MSD) analysis for lead in one batch and zinc in the other. Nine samples were qualified as estimates for lead (27136-21 through 27136-29) and nine samples (27136-30 through 27136-38) for zinc. As a result of the required 28 day holding time for mercury, concentrations from all samples for this metal have been rejected. It must also be noted that these archived samples are

more than three years old and holding times for other metals are six months. For the purposes of this memo we will not consider this in determining QA/QC completeness. Data appear on Table 1.

Laboratory Analysis

A total of 65 samples were submitted to Anatek and analyzed for 24 metals. Laboratory QA/QC was not checked externally because duplicate samples and NIST standards were not included due to budget limitations. Anatek provided a copy of their internal QA/QC results for method blanks, laboratory control samples (LCS), and MS/MSD analysis.

Internal QA/QC

Anatek analyzed a total of seven method blanks to ensure no bias was introduced during sample preparation. All prep blanks were below the instrument detection limits for all metals analyzed. No qualifiers were placed on the data based on Anatek's prep blank results.

Internal checks of Anatek's accuracy were assessed by analyzing seven soil laboratory control samples (LCS). All LCS displayed acceptable percent recoveries and/or were within the acceptable range specified by Anatek.

Internal checks of laboratory precision at Anatek were assessed using matrix spike/matrix spike duplicates. MS/MSD were analyzed on six of the 65 samples (approximately 1 in 11). Calculated relative percent difference (RPD) values for the MS/MSD samples were within acceptable limits and ranged from 0.0% to 10.1%, with an average of 2.3%. Spike percent recoveries were within acceptable limits except in two QC batches for lead and zinc. In one batch the percent recovery of lead for the spike and spike duplicate was 63.41% and 68.29%, respectively. In the other batch zinc's percent recovery was 88.59% for the spike and 72.3% for the spike duplicate. The acceptable range for percent recovery is from 75% to 125%. All sample results for lead and zinc contained in the corresponding batches were qualified as estimates.

Conclusions

No field QA/QC results are available due to the lack of field QA/QC samples included in the total samples analyzed.

An internal check of Anatek's laboratory accuracy was assessed using soil LCS. All LCS results were within acceptable limits. Laboratory precision was assessed using duplicate analyses. All duplicates displayed acceptable RPD values. However, nine sample results for lead (27136-21 through 27136-29) and nine samples for zinc (27136-30 through 27136-38) were qualified as estimates based on low percent recoveries during the spike duplicate analysis. All metal concentrations in all laboratory prep blanks were below detection limit. Mercury in all 65 samples was rejected because the required 28 day holding time was grossly exceeded for this metal.

Based on a complete review of the method blanks, LCS, and duplicate analyses, the final completeness for the study was assessed at 99.99%.

Table 1 Coeur d' Alene Basin Archive Sample Results - 11/16/99 and 12/03/99 (EPA 6020 Analysis)

Sample	Lab #	Units	Arsenic	Barium	Cadmium	Chromium	Mercury	Lead	Selenium	Silver	Copper	Calcium	Aluminum	Antimony	Beryllium	Iron	Magnesium	Manganese	Nickel	Potassium	Silica	Sodium	Thallium	Zinc	Vanadium	Cobalt
S-0001	27259-02	mg/Kg	10.8	352	6.9	15.3	0.9R	1000	ND	2.3	53.8	5830	8190	5.1	ND	17560	3180	1090	15.3	1360	NA	114	ND	656	13.9	7.7
S-0010	27178-02	mg/Kg	4.6	202	1.5	8.9	ND	117	ND	ND	34.4	2850	10210	ND	ND	12690	2060	533	7.6	1510	ND	66	ND	158	18.2	6.6
S-0011	27259-01	mg/Kg	22.4	196	6.0	22.6	1.2R	1180	ND	7.0	41	4760	4680	12.9	ND	16890	2430	1150	9.9	1030	NA	157	ND	515	12.9	4.8
S-0034	27259-04	mg/Kg	47.3	307	9.6	17.7	5.1R	5290	ND	17.0	116	1180	4790	40.3	ND	46650	2530	3780	9.6	625	NA	76	ND	1080	11.3	6.0
S-0054	27178-03	mg/Kg	5.6	179	ND	9.11	ND	37.6	ND	ND	17.8	1830	7810	ND	ND	11740	2870	354	24.8	1390	ND	117	ND	80.3	16.8	5.9
S-0057	27259-03	mg/Kg	9.1	168	ND	16.3	ND	33.5	ND	ND	40.1	4000	10930	ND	ND	15490	4680	522	34.5	1440	NA	101	ND	65.9	20.8	6.8
S-0067	27136-28	mg/Kg	5.1	160	ND	9.24	ND	66.1J	ND	ND	13.2	1320	6430	ND	ND	11600	2270	343	7.3	1050	1820	ND	ND	78.7	14.7	5.5
S-0086	27259-05	mg/Kg	3.7	154	0.8	23.8	ND	44.9	ND	1.4	22.7	2200	7470	1.6	ND	13290	2440	413	8.4	1760	NA	174	ND	59.7	23.6	6.5
S-0118	27136-14	mg/Kg	5.6	201	2.0	34.4	ND	439	ND	1.9	30.7	2860	11520	2.2	ND	14190	2730	436	16.7	998	3460	ND	ND	217	19.5	6.9
S-0119	27259-06	mg/Kg	5.7	133	1.4	16.9	ND	240	ND	ND	22	2740	6330	ND	ND	13500	2540	558	13.7	1170	NA	62.6	ND	241	17.8	6.0
S-0127	27259-07	mg/Kg	15.9	295	2.2	22.3	0.5R	463	ND	ND	72.3	2400	11390	7.5	ND	24230	3650	1060	14.8	1650	NA	101	ND	212	31.9	11.3
S-0128	27136-17	mg/Kg	12.5	193	2.9	10.9	ND	334	ND	1.4	26.8	1830	11470	4.4	ND	12410	2750	446	10.6	807	1840	117	ND	202	19.2	6.4
S-0135	27259-08	mg/Kg	6.0	68.1	ND	31.8	ND	14.1	ND	ND	23.6	1250	6260	1.0	ND	14780	3100	251	11.5	1790	NA	81.8	ND	35	24.4	7.6
S-0136	27178-04	mg/Kg	9.5	154	2.4	11.6	ND	326	ND	ND	32.3	2270	8280	1.6	ND	16170	3040	681	24.5	1540	ND	70	ND	296	18.9	7.6
S-0139	27259-09	mg/Kg	2.3	148	2.2	52.0	ND	151	ND	3.0	23.9	3990	8660	2.5	ND	11830	2200	554	7.9	1100	NA	291	ND	107	27.7	5.2
S-0159	27136-16	mg/Kg	7.2	105	1.9	12.7	ND	216	ND	1.2	23.5	1290	6680	1.9	ND	12930	2850	350	8.5	907	1840	ND	ND	175	13.8	6.3
S-0191	27136-27	mg/Kg	5.6	168	ND	9.81	ND	28.3J	ND	ND	15.8	814	17090	ND	ND	13300	2610	391	11.2	885	3600	ND	ND	56.9	20.6	5.7
S-0194	27136-18	mg/Kg	15.3	154	2.2	13.1	ND	243	ND	1.3	24	5010	8710	2.9	ND	14350	1820	759	9.9	658	1830	ND	ND	239	17.7	7.5
S-0201	27178-05	mg/Kg	4.3	197	2.3	7.8	ND	145	ND	ND	23.9	3660	8840	1.0	ND	12100	1910	535	6.2	1030	ND	117	ND	232	25.5	6.0
S-0208	27136-25	mg/Kg	16.6	167	2.5	12.6	ND	161J	ND	1.0	33.8	2670	10310	7.8	ND	14410	2030	643	12.7	676	2260	120	ND	252	18.2	8.3
S-0237	27136-24	mg/Kg	7.2	180	1.7	11.0	ND	156J	ND	ND	32.3	3150	15430	1.4	ND	13150	2970	523	22.0	874	3420	187	ND	217	21.3	6.1
S-0289	27136-21	mg/Kg	7.5	171	2.4	30.4	ND	307J	ND	1.7	22.7	1230	10130	2.2	ND	13870	1310	418	9.5	501	2410	ND	ND	136	16.1	6.5
S-0295	27136-20	mg/Kg	23.2	149	8.1	15.8	ND	498	ND	2.1	38.5	2710	9590	3.3	ND	18360	2390	659	15.6	881	2060	134	ND	390	16.3	10.5
S-0298	27136-29	mg/Kg	34.1	170	4.3	22.6	ND	226J	1.3	5.5	43.4	2660	12020	5.2	ND	24080	2040	788	28.7	926	8790	ND	ND	251	21.3	12.3
S-0299	27136-19	mg/Kg	12.1	238	ND	15.4	ND	27.7	ND	6.1	38.8	3960	19580	ND	ND	14170	5150	471	15.0	2110	3020	150	ND	86.1	23.8	6.3
S-0309	27136-23	mg/Kg	39.7	203	14.6	19.8	1.6R	936J	ND	5.2	43.8	2510	13290	8.4	ND	18880	2550	694	20.7	909	2360	102	ND	440	18.2	9.4
S-0323	27136-22	mg/Kg	22.3	172	15	10.2	4.9R	3150J	ND	10.0	77.1	2890	8130	7.9	ND	22710	1520	1690	31.9	834	2190	126	ND	1320	14.2	9.9
S-0389	27136-30	mg/Kg	6.2	176	2.0	8.08	ND	184	ND	1.6	28.9	3060	9970	5.7	ND	8730	2040	505	6.2	375	ND	192	ND	302J	12.0	5.0
S-0419	27178-01	mg/Kg	11.2	223	3.4	18.5	ND	783	ND	2.0	51.5	4540	9690	7.4	ND	13650	2440	661	10.4	879	ND	109	ND	464	15.7	6.4
S-0423	27178-06	mg/Kg	8.7	179	4.4	6.5	ND	591	ND	1.4	32.4	2940	13760	3.9	ND	11030	1270	864	13.4	700	ND	87	ND	695	15.6	4.9
S-0426	27136-31	mg/Kg	32.5	272	12.2	10.4	2.5R	1820	ND	5.4	88.4	4120	12530	9.0	ND	23220	2890	1920	12.0	551	ND	109	ND	1890J	14.6	12.8
S-0448	27136-32	mg/Kg	6.4	190	ND	11.7	ND	46.5	ND	ND	17.3	647	16650	1.3	ND	13390	2120	414	7.6	457	ND	67	ND	88.5J	23.4	5.4
S-0449	27136-33	mg/Kg	18.8	279	2.7	11.9	ND	602	ND	2.5	95.7	3520	15900	10.4	ND	19790	3350	1300	11.4	615	ND	91	ND	488J	21.0	9.6
S-0453	27136-34	mg/Kg	8.0	234	1.7	58.2	ND	109	ND	ND	38.8	3430	15960	1.5	ND	13410	4220	535	10.1	1080	ND	116	ND	167J	18.4	5.6
S-0458	27136-35	mg/Kg	24.0	254	13.4	14.6	3.7R	3370	ND	9.7	65.8	3960	14770	12.4	ND	22000	3450	1770	10.2	584	ND	120	ND	1650J	18.9	8.6
S-0468	27178-07	mg/Kg	11.0	175	ND	12.0	ND	29	ND	ND	19.1	2900	14750	ND	ND	15750	5700	831	9.4	2130	ND	77	ND	80.6	26.9	7.9
S-0499	27136-15	mg/Kg	9.0	290	5.2	44.9	ND	960	ND	3.9	39.2	3220	16610	4.0	ND	17320	1870	1930	10.5	499	3440	ND	ND	619	20.5	6.8
S-0514	27136-36	mg/Kg	13.6	557	6.1	25.9	ND	1180	ND	5.8	46.4	5400	13000	14.5	ND	18870	1800	1360	7.4	839	ND	146	ND	874J	20.3	7.6

Table 1 Coeur d' Alene Basin Archive Sample Results - 11/16/99 and 12/03/99 (EPA 6020 Analysis)

Sample	Lab #	Units	Arsenic	Barium	Cadmium	Chromium	Mercury	Lead	Selenium	Silver	Copper	Calcium	Aluminum	Antimony	Beryllium	Iron	Magnesium	Manganese	Nickel	Potassium	Silica	Sodium	Thallium	Zinc	Vanadium	Cobalt
S-0516	27136-37	mg/Kg	6.6	300	1.5	11.0	ND	298	ND	ND	31.6	3000	19180	2.1	ND	14660	2740	657	10.5	418	ND	100	ND	171J	27.0	6.5
S-0517	27136-38	mg/Kg	19.3	364	3.1	36.1	ND	548	ND	1.7	43.9	3540	17700	2.0	ND	14940	2160	796	14.0	838	ND	82	ND	449J	26.3	6.6
S-0543	27136-13	mg/Kg	17.2	376	10.5	13.9	7.4R	5140	ND	16.1	377	4630	11950	16.4	ND	21770	1760	2400	13.2	586	2630	ND	ND	1510	13.9	11.4
S-0571	27178-08	mg/Kg	19.9	286	4.0	15.9	1.1R	1180	ND	6.4	85.8	2680	8600	13.8	ND	12930	1260	1190	6.9	368	ND	84	ND	572	10.6	4.6
S-0627	27136-12	mg/Kg	13.4	194	4.1	10.7	ND	591	ND	3.0	39.6	4490	10810	4.8	ND	16940	2580	793	14.2	869	2070	101	ND	635	16.8	8.5
S-0643	27136-11	mg/Kg	12.0	189	3.9	20.3	ND	325	ND	1.8	29.4	2210	8320	2.7	ND	15140	1390	923	15.8	452	1780	ND	ND	274	15.7	7.5
S-0644	27136-26	mg/Kg	15.7	226	11.9	52.9	1.7R	1560J	ND	6.7	53.1	3960	10190	11	ND	18600	1830	1230	11.1	763	1830	113	ND	1010	16.2	7.6
S-0649	27136-09	mg/Kg	14.3	270	7.8	15.0	ND	679	ND	3.5	41.1	2980	12250	5.8	ND	15540	1880	785	10.2	775	2270	104	ND	748	15.6	7.1
S-0651	27178-09	mg/Kg	11.2	217	3.1	15.3	ND	217	ND	ND	25.2	2770	13420	1.8	ND	13370	1420	831	11.3	839	ND	74	ND	257	15.2	6.0
S-0657	27136-10	mg/Kg	4.61	143	1.8	23.7	ND	117	ND	1.4	17.3	1450	7760	1.9	ND	12540	1820	501	11.3	1100	1710	296	ND	116	20.5	7.4
S-0683	27136-03	mg/Kg	15.3	112	3.9	39.9	ND	528	ND	2.1	26.9	2020	5800	3.7	ND	13430	1110	727	5.8	353	1910	124	ND	287	11.7	6.9
S-0696	27136-06	mg/Kg	7.8	153	3.7	13.9	ND	750	ND	1.6	31.9	3020	8870	1.2	ND	14220	1590	722	9.8	573	2070	ND	ND	288	20.0	7.0
S-0698	27136-01	mg/Kg	4.6	147	1.4	10.2	ND	154	ND	ND	16	1160	8290	ND	ND	11890	1740	472	12	663	1870	ND	ND	119	20.1	6.5
S-0708	27136-07	mg/Kg	10.7	86.8	2.8	12.5	ND	219	ND	1.6	21.5	1950	5340	3.9	ND	12490	1750	284	17.8	504	1600	ND	ND	173	14.3	6.2
S-0709	27136-08	mg/Kg	12.2	256	3.8	11.4	ND	383	ND	2.4	31.6	2440	12390	4.3	ND	16200	1470	918	10.9	712	1950	ND	ND	396	14.3	8.1
S-0728	27136-02	mg/Kg	14.9	146	3.3	24.2	ND	221	ND	ND	18.4	2950	7520	ND	ND	14460	1410	640	9.0	719	2100	ND	ND	316	11.2	6.9
S-0734	27136-05	mg/Kg	15.6	131	3.9	77.7	ND	247	ND	ND	29.6	1720	7750	ND	ND	15970	1300	588	11.1	639	2210	ND	ND	298	13.9	7.6
S-0735	27136-04	mg/Kg	17.4	165	3.5	48.4	ND	455	ND	1.4	35.1	3000	8650	1.8	ND	16610	1570	706	17.7	743	2220	ND	ND	403	13.9	8.0
S-0772	27178-10	mg/Kg	11.4	164	6.6	14.2	ND	546	ND	1.5	23.7	2170	9800	4.7	ND	11700	1730	732	6.9	433	ND	84	ND	431	13.1	4.9
S-0793	27178-11	mg/Kg	8.5	195	3.7	11.0	ND	256	ND	ND	18.9	1720	8930	3.3	ND	10160	1200	507	5.3	652	840	39	ND	199	13.5	3.9
S-0824	27178-12	mg/Kg	28.6	206	10.1	8.6	5.2R	10900	ND	44.6	134	1940	8790	120	ND	23320	1790	1530	7.0	499	1070	39	ND	1290	12.8	5.7
S-0825	27178-13	mg/Kg	12.8	203	6.0	9.0	ND	355	ND	1.6	25.7	2970	15370	4.2	ND	13320	2580	637	6.6	1020	1880	70	ND	326	18.3	5.2
S-0847	27178-14	mg/Kg	11.8	211	4.1	32.1	ND	623	ND	3.3	80.7	6880	12480	7.1	ND	19520	4920	776	13.9	862	2390	75	ND	364	33.9	11.7
S-0894	27178-15	mg/Kg	15.8	212	4.7	9.9	ND	404	ND	2.0	37.1	2120	12940	6.6	ND	14830	1420	934	7.5	414	1350	55	ND	332	14.3	5.8
S-0919	27178-16	mg/Kg	24.5	101	3.3	114	ND	372	ND	1.3	27.3	1130	14750	3.1	ND	13130	1230	448	6.0	322	2210	70	ND	164	21.9	3.7
S-0922	27178-17	mg/Kg	13.4	188	7.4	36.8	ND	408	ND	2.5	38.3	2240	15720	9.1	ND	12880	1220	804	6.6	518	2380	96	ND	322	15.8	5.1
S-0950	27178-18	mg/Kg	12.1	227	3.7	13.3	ND	665	ND	1.5	73.3	3860	14300	5.8	ND	13690	1360	880	7.1	681	2460	102	ND	761	16.1	6.6

J: Sample qualified as an estimate

R: Sample result rejected due to grossly exceeded holding times.

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M E M O R A N D U M

To: Jerry Lee, TerraGraphics, Moscow

From: Shanda LeVan, TerraGraphics, Moscow

Date: June 6, 2000

Subject: QA/QC review for soil, dust, and water samples collected during the Summer and Fall 1999 Basin Risk Assessment sampling events, and analyzed through the Contract Laboratory Program (CLP).

Introduction

The following memorandum summarizes the quality assurance/quality control (QA/QC) review for soil, dust, and water samples collected during the Summer and Fall 1999 Basin Risk Assessment sampling events. These samples were analyzed through the Contract Laboratory Program (CLP) for 23 metals by Chemtech Consulting Group, Englewood, NJ., and by Sentinel, Inc., Huntsville, AL.

General

A QA/QC review was done to evaluate the precision, accuracy, completeness, and representativeness of the data obtained from the field. An initial Quality Assurance review of the laboratory data was conducted through EPA Region 10 and is not included in this memo. EPA's overall assessment of the data indicated that all samples were analyzed in accordance with the Quality Control specifications outlined in the CLP Statement of Work (SOW) for inorganic analysis and the data, as qualified, is acceptable for all purposes. All 23 analytes were 100% complete for all samples except for antimony and mercury. For antimony, 82 of the 1445 samples were rejected based on EPA's review of internal laboratory QA/QC methods. The completeness for Antimony is 94.3%. The holding time for mercury was exceeded for all samples, thus all detected mercury results were qualified as estimates and all undetected mercury results were qualified as rejected and unusable. Mercury was rejected for 672 of the 1445 samples resulting in 53.5% completeness.

The purpose of this memo is to evaluate QA/QC for field sampling and integrate this evaluation with EPA's evaluation of laboratory QA/QC. Procedures for sample labeling, handling, and analysis were as described in the Procedures for the Coeur d'Alene River Basin Risk Assessment Sampling Event (TerraGraphics 1999) and in the Field Sampling Plan and Quality Assurance Project Plan for the Bunker Hill Basin-Wide RI/FS (URS Greiner & CH2M Hill 1998). This data validation review indicated all samples were properly labeled and tracked during the project. In December 2000, 1445 samples (including QA/QC) were shipped and analyzed through the Environmental Protection Agency's (EPA) Contract Laboratory Program (CLP). These 1445 samples were analyzed for 23 metals (aluminum, antimony, arsenic, barium, beryllium, cadmium, calcium, chromium, cobalt, copper, iron, lead, manganese, mercury, nickel, potassium, selenium, silver, sodium, thallium, vanadium, and zinc).

Field Sampling QA/QC Results

A total of 1445 samples (including QA/QC) were analyzed. A summary of the Fall and Summer 1999 CLP samples, including the QA/QC samples, is presented in Table 1. Of the 1445 samples, 27 rinsate blanks, 431 yard soil, 265 driveway soil, 76 garden soil, 50 play area soil, 260 Right-of-way (ROW) soil, 118 initial drinking water, 120 purged drinking water, and 98 vacuum dust samples were analyzed; and of these, 132 were duplicates and six were splits. The six splits were collected, although the work plan did not call for the collection of splits. All samples were banked and recorded on a master log, and chain of custody forms were completed and checked before samples were shipped to the lab.

Table 1 Summary of 1999 Fall and Summer CLP Samples

Location/Media	Samples	Duplicates	Splits	Total
Rinsate Blanks	27	--	--	27
Yard Soil	401	30	--	431
Driveway Soil	233	32	--	265
Garden Soil	64	12	--	76
Play Area Soil	47	3	--	50
ROW Soil	241	13	6	260
Initial Drinking Water	107	11	--	118
Purged Drinking Water	101	19	--	120
Vacuum Dust	86	12	--	98
Total	1307	132	6	1445

Rinsate Blanks

Rinsate blanks were collected at the end of each sampling day to ensure decontamination procedures were effective, and that cross-contamination was not significant during field sampling. Rinsate blanks consisted of commercially available distilled water poured over a representative batch of decontaminated sampling equipment. Rinsate blanks were collected into 500 ml plastic bottles and preserved with nitric acid. The bottles were supplied by SVL Analytical Laboratories of Smelterville, Idaho.

Twenty seven rinsate blanks were collected during sampling. Rinsate blank results are presented in Table 2. Metal concentrations were below the CRDL in most rinsate samples, and detected concentrations were well below the concentrations found in the field samples. Based on this information, it was determined that decontamination procedures were adequate for the project and no qualifiers were placed on the data.

Field Duplicates

Field duplicates consisted of two samples taken consecutively at each site. Duplicate samples were used to measure the variability of sampling technique and the natural variation in soil, dust, and water samples. It is expected that the variability in soil and dust is greater than the variability from the drinking water samples. A total of 132 duplicate samples were collected in the field and submitted to the laboratory for analysis.

Results for the field duplicate analysis for yard soil, discrete garden, discrete play area, discrete driveway, ROW, vacuum dust, initial drinking water, and purged drinking water are presented in Tables 3a, 3b, 3c, 3d, 3e, 3f, and 3g, respectively. Data quality objectives for the project specified that the duplicates would have relative percent differences (RPDs) within ±35%. RPDs were not calculated for duplicate pairs that were below the contract required detection limit (CRDL).

Soils from yard, discrete garden, and discrete play areas were sampled using a core barrel and slam bar. Forty five soil core duplicates were sampled. Duplicates were collected by splitting the core sample in half lengthwise, thus duplicate results were expected to be fairly precise. However, 11.5% of the soil core duplicate results were outside of the 35 percent precision limit. Soil from driveway and ROW samples were collected from sample pits about one foot wide. The duplicate samples were collected from the same location in the pit as the original. Fifty one soil pit duplicates were collected in the field. Soil pit duplicates results were similar to soil core results with 9.5% of the results being outside the 35 percent precision limit. RPDs outside of the 35 percent limit for soil samples are likely due to low homogeneity in the soil and/or the occurrence of hot spots. No samples were qualified as estimates based on field duplicate results.

There were 12 vacuum dust duplicate samples collected and analyzed. RPDs were within the 35 percent precision limit for all but 11.1% of the vacuum dust duplicate samples. Twenty four drinking water duplicates were sampled and analyzed. It was expected that the drinking water duplicates would have higher precision than the soil and dust samples. Actually, only 78.1% of drinking water duplicates were within the 35 percent precision limit. It must be noted that this only applies to detected concentrations and that 419 of the 552 or 76% of the water duplicate analyses for all metals were below the CRDL. If below detection values were included in the precision calculation, 94.7% of the drinking water duplicates would be within the 35 percent precision limit.

Field Splits

Split samples were composed of one-half of a soil sample that has been homogenized in the field. Split samples are collected to examine variability in laboratory procedures. Six split samples were collected from ROWs. Only five of the six split pairs were analyzed. Results for the field split analyses are presented in Table 4. One split result had RPDs above 35 percent for 15 of the 23 metals analyzed. For the other three splits, RPDs were within the 35 percent precision limit for 86.7% of the split results for all metals. For two of the splits, the original and split sample were

analyzed by two different labs, one of the samples from each pair had insufficient volume to be sampled CLP, and was analyzed by Anatek Labs, Inc. of Moscow, Idaho. Thus, the data must be interpreted carefully, because analysis occurred at two different labs. With this, and the fact that there were only five split pairs analyzed, it is difficult to determine any recommendations for effect of split results on the field data. Thus, no qualifiers were placed on the data based on field split results.

Conclusions

A check of field decontamination procedures was assessed using rinsate blanks. Metal concentrations were below the CRDL in most rinsate samples, and all detected concentrations were well below the concentrations found in the field samples. No qualifiers were placed on the data based on rinsate blank results.

Field duplicates were sampled to measure the variability of sampling technique and the natural variation in soil, dust, and water samples. Core soil and pit soil duplicates were within the 35 percent precision limit for more than 88% of the soil duplicates. These precision results for soil samples is likely due to low homogeneity in the soil and/or the occurrence of hot spots. Vacuum dust sample duplicate results were also within the 35 percent precision limit for more than 88% of the duplicate analyses. Seventy eight percent (78.1%) of the water duplicates were within the precision limit, with 76% of the duplicates being below the CRDL. No qualifiers were placed on the data based on soil, dust, and water duplicate results.

Although the work plan did not call for them, five split samples were analyzed and compared with the originals. The results were inconclusive based on the low number of splits and due to two pairs being analyzed by two different laboratories. No qualifiers were placed on the data based on field splits.

Based on the review of rinsate blanks, field duplicates, and field splits, the final completeness, based on field QA/QC results, for the study was assessed at 100%. In addition, laboratory QA/QC reviews completed by EPA indicated that all samples were analyzed in accordance with the Quality Control specifications outlined in the CLP Statement of Work (SOW) for inorganic analysis and the data, as qualified, is acceptable for all purposes. Based on EPA's laboratory QA/QC review, completeness was assessed at 100% for all metals except for antimony and mercury, which were 94.3% and 53.5% complete, respectively.

Table 2 - 1999 Coeur d' Alene Basin Summer and Fall CLP Rinsate Blanks

EPA Sample Number	Aluminum ug/L	Antimony ug/L	Arsenic ug/L	Barium ug/L	Beryllium ug/L	Cadmium ug/L	Calcium ug/L	Chromium ug/L	Cobalt ug/L	Copper ug/L	Iron ug/L	Lead ug/L
MJCP05	100.0 U	2.1 UJ	2.2 U	0.8 U	0.1 U	0.3 U	2590.0 U	0.3 U	0.6 U	10.3 U	18.6 U	3.1
MJCP18	34.9 U	2.1 UJ	2.2 U	0.8 U	0.1 U	0.3 U	2590.0 U	0.3 U	0.6 U	1.6 J	45.6 U	1.1 U
MJCP20	34.9 U	2.1 UJ	2.2 U	0.8 U	0.1 U	0.4 U	2590.0 U	3.0 U	0.6 U	2.2 UJ	89.4 U	1.1 U
MJCS41	34.9 J	2.1 J	2.2 U	47.8 U	0.1 U	1.6 J	8590.0	0.3 U	0.6 U	48.8	18.6 J	3.5
MJCS49	34.9 J	2.1 J	2.2 U	0.8 U	0.1 U	0.3 J	2590.0 U	0.3 U	0.6 U	3.3 J	67.8 U	2.0 U
MJCS52	34.9 J	4.2 UJ	12.0 U	0.8 U	0.1 U	0.3 J	2590.0 U	0.3 U	0.6 U	6.4 U	92.4 U	3.2
MJCS76	24.6 J	5.0 UJ	6.0 U	1.1 U	1.4 U	1.0 U	111.0 U	1.0 U	1.0 U	24.0 U	92.1 U	8.1
MJCS98	28.3 U	5.0 UJ	6.0 U	1.1 U	1.0 U	1.0 U	334.0 U	1.0 U	1.0 U	28.1	46.0 U	2.0 U
MJCZ07	34.9 J	2.1 J	2.2 U	0.8 U	0.1 U	0.3 J	2590.0 U	0.4 J	0.6 U	1.8 J	18.6 U	1.7 U
MJCZ11	34.9 J	2.1 J	2.2 U	0.8 U	0.1 U	0.3 J	2590.0 U	0.3 J	0.6 U	1.2 J	18.6 U	1.1 U
MJCZ33	49.2 U	2.1 J	2.2 J	1.0 U	0.1 U	0.3 U	2590.0 U	0.3 J	0.6 U	1.8 U	44.2 U	1.1 U
MJCZ36	34.9 U	2.1 J	2.2 J	0.8 U	0.1 U	0.3 U	2590.0 U	0.3 J	0.6 U	1.3 U	18.6 U	1.1 U
MJCZ38	34.9 U	2.1 J	2.2 J	0.8 U	0.1 U	0.3 U	2590.0 U	0.3 J	0.6 U	0.6 U	18.6 U	1.1 U
MJCZ56	34.9 U	2.1 J	2.2 U	0.8 U	0.1 U	0.3 U	2590.0 U	0.3 J	0.6 U	3.0 UJ	18.6 U	1.1 U
MJCZ60	34.9 U	2.1 J	2.2 U	0.8 U	0.1 U	0.3 U	2590.0 U	0.3 J	0.6 U	1.4 UJ	24.3 U	1.1 U
MJCZ67	30.3 U	5.0 U	6.0 U	1.0 U	1.0 U	1.0 U	16.0 U	1.0 U	1.0 U	1.0 U	16.0 U	2.0 UJ
MJCZ70	51.0 U	5.0 U	6.0 U	1.0 U	1.0 U	1.0 U	68.1 U	1.0 U	1.0 U	9.7 U	19.5 U	2.0 UJ
MJCZ95	14.3 U	5.0 U	6.0 U	1.0 U	1.0 U	1.0 U	61.7 U	1.0 U	1.0 U	5.6 U	55.8 U	2.0 U
MJCZ96	14.0 U	5.0 U	6.0 U	1.0 U	1.0 U	1.0 U	16.0 U	1.0 U	1.0 U	1.0 U	16.0 U	2.0 U
MJDC04	34.9 J	2.1 J	2.2 U	0.8 U	0.1 U	0.3 J	2590.0 U	0.3 U	0.6 U	7.9 U	36.8 U	1.2 U
MJDC15	34.9 J	2.1 J	2.2 U	0.8 U	0.1 J	1.0 UJ	2590.0 U	0.3 J	0.6 J	1.8 U	18.6 U	1.1 U
MJCN95	119.0 U	2.1 UJ	2.2 U	1.0 U	0.1 U	0.3 U	2590.0 U	0.3 U	0.6 U	5.0 U	18.6 U	2.9 U
MJCP12	34.9 U	2.1 UJ	2.2 U	0.8 U	0.1 U	0.3 U	2590.0 U	0.4 U	0.6 U	0.5 UJ	275.0	1.1 U
MJCP24	34.9 U	2.1 UJ	2.2 U	0.8 U	0.1 U	0.8 U	2590.0 U	0.8 U	0.6 U	11.4 J	53.6 U	1.1 U
MJCP32	34.9 U	2.1 UJ	2.2 U	0.8 U	0.1 U	0.3 U	2590.0 U	0.6 J	0.6 U	9.2 U	21.0 U	1.1 U
MJCP44	34.9 U	2.1 UJ	2.2 U	0.8 U	0.1 U	0.3 U	2590.0 U	0.3 UJ	0.6 U	6.2 U	18.6 U	1.1 U
MJCP46	34.9 U	2.1 UJ	2.2 U	0.8 U	0.1 U	0.3 U	2590.0 U	0.3 UJ	0.6 U	18.1 U	18.6 U	1.4 U

R: Result rejected based on the data validation process.
J: The associated value is an estimate.
U: The analyte was not detected at or above the reported result.
UJ: The analyte was not detected at or above the reported result and the value is an estimate.

Table 2 (Cont'd) - 1999 Coeur d' Alene Basin Summer and Fall CLP Rinsate Blanks

EPA Sample Number	Magnesium ug/L	Manganese ug/L	Mercury ug/L	Nickel ug/L	Potassium ug/L	Selenium ug/L	Silver ug/L	Sodium ug/L	Thallium ug/L	Vanadium ug/L	Zinc ug/L
MJCP05	38.8 U	2.4 U	R	1.3 U	60.0 U	1.8 U	0.4 U	173.0 UJ	2.1 U	0.5 U	46
MJCP18	38.8 U	2.4 U	R	1.3 U	38.0 U	1.8 U	0.4 U	155.0 UJ	2.1 UJ	0.5 U	320
MJCP20	38.8 U	2.4 U	R	3.1 U	68.9 U	1.8 U	0.4 U	535.0 UJ	2.1 UJ	0.5 U	327
MJCS41	3400.0 U	2.4 U	R	1.6 U	934.0 J	1.8 U	0.4 U	323.0 U	2.1 U	0.5 U	848.0
MJCS49	38.8 U	2.4 U	R	1.3 U	52.7 J	1.8 U	0.4 U	189.0 UJ	2.1 U	0.5 U	14.2 U
MJCS52	38.8 U	2.4 U	R	1.3 U	47.6 J	1.8 U	0.4 U	155.0 J	2.1 U	0.5 U	54.8
MJCS76	26.1 U	6.5 U	R	2.0 U	39.0 U	5.0 U	1.0 U	175.0 U	7.0 U	2.0 U	75.0
MJCS98	37.0 U	1.0 U	R	2.0 U	39.0 U	5.0 U	1.0 U	251.0 U	7.0 U	2.0 U	159.0
MJCZ07	38.8 U	2.4 U	R	1.7 U	8.3 J	1.8 U	0.4 J	210.0 U	2.1 U	0.5 J	18.4 U
MJCZ11	38.8 U	2.4 U	R	1.3 U	3.2 J	1.8 U	0.4 J	155.0 U	2.1 U	0.5 J	12.9 U
MJCZ33	38.8 U	2.4 U	R	1.3 U	30.6 UJ	1.8 U	0.4 U	155.0 U	2.1 U	0.5 U	12.9 U
MJCZ36	38.8 U	2.4 U	R	1.3 U	35.8 UJ	1.8 U	0.4 U	155.0 U	2.1 U	0.5 U	17.0 U
MJCZ38	38.8 U	2.4 U	R	1.3 U	30.0 UJ	1.8 U	0.4 U	155.0 U	2.1 U	0.5 U	12.9 U
MJCZ56	38.8 U	2.4 U	R	1.3 U	70.4 J	1.8 U	0.4 U	173.0 U	2.4 U	0.5 U	12.9 U
MJCZ60	38.8 U	2.4 U	0.1 J	1.3 U	54.5 J	1.8 U	0.4 U	174.0 U	2.4 U	0.5 U	15.4 U
MJCZ67	23.0 U	1.0 U	R	2.0 U	39.0 U	5.0 U	1.0 U	23.0 U	7.0 U	2.0 U	2.0 U
MJCZ70	23.0 U	1.0 U	R	2.0 U	39.0 U	5.0 U	1.0 U	133.0 U	7.0 U	2.0 U	34.3
MJCZ95	25.8 U	1.0 U	R	2.0 U	39.0 U	5.0 U	1.0 U	129.0 U	7.0 U	2.0 U	13.8 U
MJCZ96	23.0 U	1.0 U	R	2.0 U	39.0 U	5.0 U	1.0 U	23.0 U	7.0 U	2.0 U	2.0 U
MJDC04	38.8 J	2.4 U	R	1.3 U	62.2 U	1.8 U	0.7 U	299.0 UJ	4.1 U	0.5 U	22.4
MJDC15	38.8 U	2.4 U	R	1.3 U	38.7 U	1.8 U	0.4 U	1340.0 UJ	2.4 U	0.5 U	12.9 U
MJCN95	39.0 U	2.4 U	R	1.3 U	68.6 U	1.8 U	0.4 U	155.0 UJ	2.1 U	0.5 U	179
MJCP12	38.8 U	2.4 U	R	1.3 U	54.4 U	1.8 U	0.4 U	325.0 UJ	2.1 UJ	0.5 U	262
MJCP24	38.8 U	2.4 U	R	1.3 U	67.4 U	1.8 U	0.6 U	961.0 UJ	2.1 UJ	0.5 U	320
MJCP32	38.8 U	2.4 U	R	2.4 U	25.9 U	1.8 U	0.4 UJ	199.0 U	2.1 U	0.5 UJ	140
MJCP44	38.8 U	2.4 U	R	1.3 U	32.5 U	1.8 U	0.4 UJ	296.0 U	2.1 U	0.5 UJ	73.2
MJCP46	38.8 U	2.4 U	R	1.3 U	28.3 U	1.8 U	0.4 UJ	155.0 U	2.1 U	0.5 UJ	307

R: Result rejected based on the data validation process.
J: The associated value is an estimate.
U: The analyte was not detected at or above the reported result.
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M E M O R A N D U M

To: Jerry Lee, TerraGraphics, Moscow

From: Shanda LeVan, TerraGraphics, Moscow

Date: April 5, 2000

Subject: QA/QC Review for the low weight soil, vacuum dust, and dust mat samples collected during the Summer and Fall 1999 Basin Risk Assessment sampling events.

Introduction

The following memorandum summarizes the quality assurance/quality control (QA/QC) review for the low weight soil, vacuum dust, and dust mat samples collected during the Summer and Fall 1999 Basin Risk Assessment sampling events. These samples were analyzed for 7 metals by Anatek Labs, Inc. of Moscow.

General

A QA/QC review was done to evaluate the precision, accuracy, completeness, and representativeness of the data obtained from both the field and the laboratory. Procedures for sample labeling, handling, and analysis were as described in the Procedures for the Coeur d' Alene River Basin Risk Assessment Sampling Event (TerraGraphics 1999) and in the Field Sampling Plan and Quality Assurance Project Plan for the Bunker Hill Basin-Wide RI/FS (URS Greiner & CH2M Hill 1998). This data validation review indicated all samples were properly labeled and tracked during the project. In February 2000, 67 prioritized soil, vacuum, and mat dust samples were selected from the low weight samples that did not get analyzed through the Environmental Protection Agency's (EPA) Contract Laboratory Program (CLP). These 67 samples were sent to Anatek Labs, Inc. in Moscow, Idaho for analysis of seven metals (antimony, arsenic, cadmium, iron, lead, manganese, and zinc). All data appear in Table 1.

The residential yard soil field duplicate and corresponding original sample (ALI007; ALI006) had a Relative Percent Difference (RPD) of 29.6 for lead. This RPD is above the accepted range, therefore all residential yard soil samples analyzed by Anatek in this batch were qualified as estimates for lead.

The lead concentration percent recovery was 119% for dust mat standard 99M10566. The average percent recovery for lead concentration was 65% for the 1999 Interior House Dust sampling event within the Bunker Hill Superfund Site. Sample 99M10565 had a lead concentration percent recovery close to this (69%), but sample 99M10566 was an anomoly, especially when compared with past studies. All dust mat samples batched with sample 99M10566 have been qualified as estimates for all metals except iron based on percent recovery of 99M10566.

Two dust mat samples were qualified as estimates for the loading portion of the study based on answers to the questionnaire given upon retrieval of the dust mats. The loading was estimated for sample 99M10544 because it had been shaken once. The loading for sample 99M10551 was determined to be an estimate because the resident indicated that they were gone from the home 10 or more days during the study.

In reviewing the internal QA/QC, one method blank was found to contain a zinc concentration of 3.76 mg/kg. This concentration was significantly lower than zinc concentrations found in the 67 samples, thus no samples were qualified based on the method blank results. Laboratory precision was also assessed using duplicate analyses. One duplicate for arsenic displayed a 27.1 RPD value, which is above the accepted range, and all samples batched with this duplicate were qualified as estimates. No other qualifiers were placed on the data based on internal laboratory QA/QC samples.

Field Sampling QA/QC Results

A total of 67 samples (including QA/QC) were analyzed. Of the 67 samples, 11 vacuum dust samples were analyzed, 28 mat dust samples were analyzed; and of these, two were mat duplicates and two were mat standards. The rest were soil samples with 10 collected from residential yards and 18 from right-of-ways (ROWs). Of the 10 residential yard soil samples, one was a duplicate. Of the 18 ROW samples, one was a split, and one a duplicate; however the original samples were not analyzed by Anatek, they were analyzed through EPAs Contract Laboratory Program (CLP). These two ROW field QC samples will be compared to the results from the original CLP samples and discussed in the 1999 Basin CLP QA/QC Memo.

Duplicates

For dust mats, a field duplicate consisted of a second dust mat being placed directly next to the original. Duplicate dust mat samples were used to examine variability in the field procedures. Two duplicates were collected in the field and submitted to Anatek for analysis. The RPDs were calculated for all 7 metals for samples 99M10552 and 99M10560. These RPDs are displayed in Table 2. RPDs for sample 99M10552 were low for all 7 metals; the RPDs for sample 99M10560 were all significantly higher for the 7 metals. However, the degree of variability is consistent with earlier dust mat sampling programs. Because it was necessary to place the duplicate mats behind the original mats at most homes, many times the duplicates receive considerably less soil than the original mats. No samples were qualified as estimates based on the duplicate dust mat results.

One residential yard soil duplicate was collected in the field. The residential yard soil duplicate was obtained by splitting the soil core sample in half lengthwise and placing half in the original sample bag and the other half in the duplicate sample bag. The residential soil duplicate and the

original sample were both analyzed by Anetek and all RPDs were within the accepted range for all metals except for lead which had an RPD of 29.6. Based on the high RPD for lead in the residential yard soil duplicate, all residential yard soil samples analyzed by Anatek in this batch were qualified as estimates for lead.

The ROW duplicate and split samples will be matched with the original samples (analyzed through CLP). The data will be interpreted carefully, because analysis occurred at two different labs. The review of these two QC samples and their originals will be discussed in the 1999 Basin CLP QA/QC Memo. The residential soil duplicate, the duplicate dust mat results, and the results for the two ROW QC samples are presented in Table 2.

Laboratory Analysis

A total of 60 samples (excluding field QA/QC samples) were submitted to Anatek and analyzed for 7 metals. Laboratory QA/QC was checked externally by duplicate samples collected in the field and by including two dust mat standards submitted blind to the laboratory for analysis of 7 metals. Anatek provided a copy of their internal QA/QC results for method blanks, laboratory control samples (LCS), duplicate analysis, and matrix spike/matrix spike duplicate (MS/MSD) analysis.

External QA/QC

A pre-loaded mat standard is inserted at the University of Idaho vacuum lab for every 20th dust mat sample collected. There were 26 dust mats collected during the summer 1999 Basin Risk Assessment, thus two pre-loaded mat standards were included for vacuuming and the recovered samples were submitted blind to Anatek. Pre-loaded mats contained 10 grams of NIST standard 2711 Montana Soil containing 1162 mg/kg lead, 19.4 mg/kg antimony, 105 mg/kg arsenic, 41.7 mg/kg cadmium, 638 mg/kg Manganese, and 350.4 mg/kg zinc. The iron concentration in the standard was undetermined.

The standards were used to evaluate both the dust recovery of the vacuum method, as well as accuracy of the lab analysis. The percent recoveries on dust mass for the standards were 88% and 82% for 99M10565 and 99M10566, respectively. The percent recoveries for lead concentration were 69% (99M10565) and 119% (99M10566). The average percent recovery for lead concentration was 65% for the 1999 Interior House Dust sampling event within the Bunker Hill Superfund Site. Sample 99M10565 had a lead concentration percent recovery close to this, but sample 99M10566 appeared to be an anomaly when compared with past studies. Therefore, mats batched with 99M10566 during analysis by Anatek will be qualified as estimates. The percent recoveries for lead mass were 61% (99M10565) and 98% (99M10566). Again sample 99M10565 is more in line with average percent recoveries for lead mass in past studies. Percent recovery results are presented in Tables 3a, 3b, and 3c.

In 1997, 1998, and 1999 house dust studies within the Bunker Hill Superfund Site showed standard percent recoveries for lead concentration from mats to be low (around 65%-80%). It was determined from mass balance calculations on the mats using NIST standard soils that fiber dilution of vacuum samples is a possible cause of reduced percent recovery on concentration for low mass recovery samples. Another possible explanation for the decreased percent recovery on concentration is preferential retention of the clays (which are predominately lead bearing) on the

somewhat sticky vinyl surface, thereby reducing the total amount of lead available for vacuum removal. Additionally, the sieved portion of many dust mat samples in past projects have contained significant amounts of fibers. Numerous mat fibers were clearly visible in 1997 and 1998 laboratory photographs of the sieved portion of the samples. For these reasons, no qualifiers were placed on the data based on the standard results.

Internal QA/QC

Internal checks of Anatek's accuracy were assessed by analyzing five soil laboratory control samples (LCS). All LCS displayed acceptable percent recoveries and/or were within the acceptable range specified by Anatek. LCS results are presented in Table 4.

Anatek analyzed a total of five method blanks to ensure no bias was introduced during sample preparation. All method blanks were below the instrument detection limits for all metals analyzed except zinc, which was detected at 3.76 mg/kg in one of the five method blanks. All samples analyzed for zinc contained values significantly higher than that detected in the blank, with the lowest zinc concentration being 51.4 mg/kg, thus no qualifiers were placed on the data based on Anatek's method blank results. Table 5 displays the method blank results.

Internal checks of laboratory precision at Anatek were assessed using matrix spike/matrix spike duplicates (MS/MSD). MS/MSD were analyzed on five of the 67 samples (approximately 1 in 13.4). Calculated RPD values for the MS/MSD samples were within acceptable limits and ranged from 0.0% to 3.9%, with an average of 1.8% for all 7 metals. Spike percent recoveries were within acceptable limits and no qualifiers were placed on the data based on MS/MSD analysis. MS/MSD results are found in Table 6.

Four laboratory duplicates were analyzed by Anatek to further evaluate internal laboratory precision. One duplicate for arsenic displayed a 27.1 RPD value which is above the accepted range and all samples batched with this duplicate were qualified as estimates. No other qualifiers were placed on the data based on laboratory duplicate analysis. Laboratory duplicate results are presented in Table 7.

Conclusions

Field and lab variability were assessed using field duplicates. Analysis of two dust mat duplicates indicated relatively high variability which is attributable to the procedure. The residential yard soil field duplicate and corresponding original sample (ALI007; ALI006) had a Relative Percent Difference (RPD) of 29.6 for lead. This RPD is above the accepted range, therefore all residential yard soil samples analyzed by Anatek in this batch were qualified as estimates for lead.

An external check of Anatek lab accuracy was assessed using soil standards of known concentrations inserted blind with the dust mat field samples. Percent recoveries were low for one mat standard and was likely a result of the sample procedures. No qualifiers were placed on the data based on the standards.

An internal check of Anatek's laboratory accuracy was assessed using soil LCS. All LCS percent recovery results were within acceptable limits. One method blank contained a zinc concentration of 3.76 mg/kg, but was significantly lower than the zinc concentrations found in the 67 samples.

Laboratory precision was also assessed using duplicate analyses. One duplicate (99M10052) for arsenic displayed a 27.1 RPD value which is above the accepted range and all samples batched with this duplicate were qualified as estimates. No other qualifiers were placed on the data based on internal laboratory QA/QC samples. A summary of the sample results with qualifiers is presented in Table 1.

Based on a complete review of the field duplicates and standards, method blanks, LCS, duplicate analyses, and MS/MSD analysis, the final completeness for the study was assessed at 100%.

Table 1 - Coeur d' Alene Basin 1999 Low Weight Sample Results

Sample #	Lab #	Matrix	Units	Antimony	Arsenic	Cadmium	Lead	Iron	Manganese	Zinc
99M10538	27635-01	Mat Dust	mg/Kg	ND	4.05	ND	53.4	14850	402	235
99M10539	27635-02	Mat Dust	mg/Kg	5.75	3.28	ND	111	16390	339	335
99M10540	27635-03	Mat Dust	mg/Kg	2.63	8.66	ND	121	13480	329	339
99M10541	27635-04	Mat Dust	mg/Kg	103	ND	ND	68.7	16630	324	211
99M10542	27635-05	Mat Dust	mg/Kg	4.61	ND	ND	41.3	8050	174	303
99M10543	27635-06	Mat Dust	mg/Kg	3.18	4.49	ND	105	27530	502	200
99M10544	27635-07	Mat Dust	mg/Kg	2.49	4.66	3.11	469	11590	310	603
99M10545	27635-08	Mat Dust	mg/Kg	ND	2.99	ND	93.6	11650	353	946
99M10546	27635-09	Mat Dust	mg/Kg	ND	4.48	5.63	95.4	14840	260	301
99M10547	27635-10	Mat Dust	mg/Kg	ND	5.05	3.84	118	18430	354	170
99M10548	27635-12	Mat Dust	mg/Kg	ND	ND	3.15	289	16240	292	446.00
99M10549	27635-13	Mat Dust	mg/Kg	8.08	ND	ND	57.7	24800	404	170.00
99M10550	27635-14	Mat Dust	mg/Kg	64.1	4.6J	2.03	111	15240	388	370.00
99M10551	27635-15	Mat Dust	mg/Kg	6.85	8.5J	18.6	2390	11440	348	6070.00
99M10552	27635-16	Mat Dust	mg/Kg	ND	3.1J	ND	117	11900	299	274.00
99M10553	27635-17	Mat Dust	mg/Kg	ND	3.5J	ND	132	12090	294	304.00
99M10554	27635-18	Mat Dust	mg/Kg	ND	15.7J	3.21	318	17350	582	878.00
99M10555	27635-19	Mat Dust	mg/Kg	ND	4.2	ND	82.8	15640	352	175.00
99M10556	27635-20	Mat Dust	mg/Kg	ND	3.7J	ND	105	22160	428	356.00
99M10557	27635-21	Mat Dust	mg/Kg	ND	24.6J	ND	33.9	10420	321	1520.00
99M10558	27635-22	Mat Dust	mg/Kg	ND	11.2J	ND	94.3	17040	424	237.00
99M10559	27635-23	Mat Dust	mg/Kg	ND	8.5J	ND	53.6	12760	279	654.00
99M10560	27635-24	Mat Dust	mg/Kg	4.04	86.2J	ND	35.7	9800	263	307.00
99M10562	27635-26	Mat Dust	mg/Kg	ND	209J	ND	77.7	22510	624	542.00
99M10563	27635-27	Mat Dust	mg/Kg	ND	185J	5.77	105	41570	965	334.00
99M10564	27635-28	Mat Dust	mg/Kg	1.44	7.4J	2.21	123	12090	348	348.00
99M10565	27635-11	Mat Dust	mg/Kg	ND	60.7J	25.8	798	11840	321	216.00
99M10566	27635-25	Mat Dust	mg/Kg	5.31	117J	46.4	1380	21190	616	387.00
99M10030	27635-29	ROW Soil	mg/Kg	8.99	16.2J	4.15	1340	32150	1900	2940
99M10040	27635-30	ROW Soil	mg/Kg	22.5	33.2J	29.1	8110	47300	4890	6730
99M10043	27635-31	ROW Soil	mg/Kg	2.85	13.9J	1.94	683	19570	806	923
99M10052	27635-32	ROW Soil	mg/Kg	10.5	33.9J	4.14	972	28540	1540	2020
99M10066	27635-33	ROW Soil	mg/Kg	18.8	25.7J	18.9	2450	60980	4330	7720
99M10069	27635-34	ROW Soil	mg/Kg	42.9	20.6J	15.5	4750	36800	1690	3910

Table 1 (Cont'd) - Coeur d' Alene Basin 1999 Low Weight Sample Results

Sample #	Lab #	Matrix	Units	Antimony	Arsenic	Cadmium	Lead	Iron	Manganese	Zinc
99M10076	27635-35	ROW Soil	mg/Kg	13.1	19.2J	4.44	1150	36110	2250	3020
99M10080	27635-36	ROW Soil	mg/Kg	14.4	23.6J	10.1	1650	35390	1820	3430
99M10084	27635-37	ROW Soil	mg/Kg	7.45	21.3J	2.62	809	17010	886	553
99M10090	27635-38	ROW Soil	mg/Kg	7.88	25.2J	ND	427	27070	617	511
99M10100	27635-39	ROW Soil	mg/Kg	46.4	48.6J	9.17	7350	28000	1690	1310
99M10132	27635-40	ROW Soil	mg/Kg	38	84.6	14	2650	24880	1310	2140
99M10138	27635-41	ROW Soil	mg/Kg	19.4	65	12.5	1890	32440	1460	2290
99M10149	27635-42	ROW Soil	mg/Kg	8.69	22.5	ND	295	15320	750	243
99M10153	27635-43	ROW Soil	mg/Kg	17	41	4.1	1540	18410	1080	896
99M10177	27635-44	ROW Soil	mg/Kg	21.9	34	10.6	4110	39540	2490	3280
99M10424	27635-49	ROW Soil	mg/Kg	ND	5.38	ND	689	20140	736	350
99M10454	27635-50	ROW Soil	mg/Kg	ND	4.5	ND	21.5	16720	753	95.1
99M10286	27635-47	Residential Soil	mg/Kg	ND	3.43	ND	97.9J	14170	516	133
99M10416	27635-48	Residential Soil	mg/Kg	ND	6.73	ND	59.1J	14570	377	90.8
ALI003	27635-55	Residential Soil	mg/Kg	ND	46.2	ND	84.4J	18610	685	98.2
ALI005	27635-57	Residential Soil	mg/Kg	3.07	11.3	2.85	908J	13120	865	490
ALI006	27635-58	Residential Soil	mg/Kg	ND	47.2	ND	77J	19100	526	90.6
ALI007	27635-59	Residential Soil	mg/Kg	ND	45.4	ND	57.1J	19430	524	78
ALI008	27635-60	Residential Soil	mg/Kg	ND	12.7	ND	25.6J	11610	270	51.4
ALI009	27635-61	Residential Soil	mg/Kg	ND	38	ND	80.5J	18190	551	121
ALI010	27635-62	Residential Soil	mg/Kg	ND	15.9	3.6	124J	16430	553	257
ALI012	27635-64	Residential Soil	mg/Kg	31	47.8	16.2	6260J	27340	2350	1870
99M10270	27635-45	Vacuum Dust	mg/Kg	ND	3.47	6.33	896	6060	151	1270
99M10277	27635-46	Vacuum Dust	mg/Kg	3.22	7.66	5.24	893	28800	386	762
99M10493	27635-51	Vacuum Dust	mg/Kg	ND	115	6.19	59.8	11760	315	334
99M10515	27635-52	Vacuum Dust	mg/Kg	ND	8.75	4.46	106	10780	304	723
ALI001	27635-53	Vacuum Dust	mg/Kg	ND	13.6	ND	81.6	15290	710	114
ALI002	27635-54	Vacuum Dust	mg/Kg	ND	5.27	ND	85.8	8610	349	616
ALI004	27635-56	Vacuum Dust	mg/Kg	69.8	44.8	7.24	862	11740	522	1070
ALI011	27635-63	Vacuum Dust	mg/Kg	12.2	21.7	6.52	686	11000	469	916
ALI013	27635-65	Vacuum Dust	mg/Kg	9.8	17.3	7.8	302	10010	289	852
ALI014	27635-66	Vacuum Dust	mg/Kg	18.8	3.24	ND	75.3	2660	133	190
ALI015	27635-67	Vacuum Dust	mg/Kg	53.2	15.6	2.43	507	11200	347	413

ND: Concentration below instrument detection limit

J: Concentration qualified as an estimate.

APPENDIX N

Proposed Geographic Subdivisions and Archived Reanalysis Results for the Coeur d'Alene Basin Human Health Risk Assessment

Appendix N

Proposed Geographic Subdivisions and Archived Reanalysis Results for the Coeur d'Alene Basin Human Health Risk Assessment

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This appendix contains the technical memorandum of the proposed geographic residential subdivisions for the Coeur d'Alene Basin (CDAB) Human Health Risk Assessment that was completed in February 2000. This memo identifies the data sets that support the baseline HHRA, evaluates whether the results of the various surveys can be combined for aggregate analysis, and proposes specific geographic subareas for exposure point concentration and risk estimates. Geographic subareas were selected to appropriately characterize the variation in exposures and risk within the CDAB, while avoiding unnecessary duplication, and protecting confidential health and private property data.

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Date: February 3, 2000

Subject: Proposed Geographic Subdivisions and Archived Reanalysis Results for the Coeur d'Alene Basin Human Health Risk Assessment

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SECTION 1.0 BACKGROUND AND OBJECTIVES

The Coeur d'Alene River Basin (CDARB) in northern Idaho has long been known to be contaminated by historical mining and smelting activity. Public health investigations in the 1970s to 1980s resulted in the designation of a 21 square mile area called the Bunker Hill Superfund Site (BHSS), or "the box", surrounding the former ore refining complex near Kellogg. Recently, the United States Environmental Protection Agency (USEPA) proposed that the original "box" be extended to include the larger area of contaminant release. This expansion resulted from the review of previous studies indicating areas outside of the original site boundaries present a potential threat to human health and the environment. A Remedial Investigation and Feasibility Study (RI/FS) is being undertaken to characterize the degree and extent of the contaminant release. Concurrently, lead health surveys and a Human Health Risk Assessment (HHRA) are being

conducted to determine potential health risks associated with residual contamination in the CDARB.

As part of the HHRA effort, existing health and environmental data have been evaluated for use in characterizing exposures to residents in the CDARB. Data from several basic surveys are, or will be, available to characterize exposures. These studies include a large residential sampling effort undertaken in the summer of 1996 by the Idaho Department of Health and Welfare (IDHW) and the Agency for Toxic Substances and Disease Registry (ATSDR) (IDHW1999), three lead health surveys conducted by the local Panhandle Health District (PHD), three residential USEPA surveys accomplished in the RI/FS (USEPA 1998a, 1998b, and 1998c), the RI/FS, and special studies conducted in the Natural Resources Damage Assessment (NRDA).

The IDHW/ATSDR study (IDHW1999) characterized both environmental contamination and biological indices of human exposure within the basin. During this study, data from 843 residential homes were systematically obtained within the CDARB. Within the scope of the current RI/FS, three additional residential studies have been conducted by the USEPA. These sampling efforts are referred to by Field Sampling Plan Addendum number and are known as FSPA06, FSPA07, and FSPA12 (USEPA 1998a, 1998b, and 1998c). A total of 123 homes within the CDARB were sampled as a result of these efforts. Various environmental data have been obtained from these homes on a voluntary self-identified basis within the past two years.

Additionally, the IDHW and the PHD have conducted fixed site blood lead screening in Upper and Lower Basin CDARB communities over the last three years. A total of 524 children aged 9 months to 9 years have provided venous blood lead samples. These children reside in a total of 260 homes within the CDARB. These homes have been identified and follow-up environmental sampling services have been offered to the parent or home-owner. A total of 128 of these homes were sampled as part of the previous studies discussed above. Of the 132 homes that were not included in previous efforts, 91 of those have been sampled in a special Fall 1999 survey. These data will be available for the HHRA, as well.

In addition, the RI/FS and the associated NRDA activities have conducted various surveys and investigations that can provide information regarding media and tissue contaminant concentrations and active and potential pathways for the HHRA.

The CDARB HHRA will characterize human health risk based on residential exposure, augmented by recreational and occupational exposures outside the home. Due to the large area of concern within the CDARB, it is appropriate to evaluate risk within geographic sub-areas (USEPA 1989). This would serve to avoid both the underestimation of human health risk in areas of the CDARB with significant contamination, as well as the overestimation of risk in areas with little contamination.

The purpose of this memorandum is to identify those data sets that will support the baseline HHRA, to evaluate whether the results of the various surveys can be combined for aggregate analysis, and to propose specific geographic sub-areas for exposure point concentration and risk estimates. Key aspects in the process of evaluating specific geographic sub-areas include identifying sufficient sub-areas to appropriately characterize the variation in exposures and risk within the CDARB, while avoiding unnecessary duplication, and protecting confidential health and private property data.

SECTION 2.0 IDHW/ATSDR AND RI/FS COMMON HOMES DATA EVALUATION

An initial step in the overall evaluation is to determine whether existing survey datasets (IDHW 1999; USEPA 1998a, 1998b, and 1998c) can be combined for further analysis. Due to the similarities of the FSPA06 and FSPA07 surveys, these data sets were previously combined by the USEPA. The same rationale can be applied to the FSPA12 survey, as it was conducted under nearly identical field sampling and analysis protocols.

However, the 1996 IDHW/ATSDR study was conducted under a different protocol than that used in the three USEPA surveys. These protocols differ in two major aspects, including the sampling methodologies employed and how homes were selected.

To evaluate whether the field sampling and analytical techniques used in the surveys produce similar results, surface soil lead and cadmium concentrations from homes common to both the IDHW/ATSDR and the USEPA surveys were compared. Table 1 shows the results for surface soil samples collected at the 23 homes sampled by both protocols.

Surface soil lead and cadmium concentrations (0-1 inch depth horizon) were selected for comparison. These soils are of significant public health concern because these are most likely to be directly contacted by individuals and may migrate into homes or children's play areas. As a result, surface soil concentrations are most often used to characterize media contaminant levels. A second important factor is that previous studies suggest that surface soil contaminant concentrations vary significantly throughout the CDARB. As a result, it is important to characterize this variable by geographic sub-area.

Both protocols recognized the importance of top horizon surface soils and employed similar sampling and analytical techniques. The USEPA protocols required that four discrete surface soil sub-samples be field composited to provide a single sample representative of a particular area of a home yard. These samples were composited by depth 0-1 inch, 1-6 inches, 6-12 inches and 12-18 inches. From five to seven such composite results were obtained at each home depending on yard size or complexity. The 1996 IDHW/ATSDR study required that a minimum of two to a maximum of ten discrete 0-1 inch depth horizon surface soil samples (based on yard area) be field composited to provide one representative analytical result per home. For comparative purposes, it was necessary to average the results obtained under the USEPA protocols for comparison to the 1996 IDHW/ATSDR data.

Tables 2a and 2b show the results of these comparisons utilizing three basic statistical techniques. Relative percent differences ranged from 1% to 126% and averaged 44% for lead, and ranged from 3% to 66% and averaged 28% for cadmium (Table 1). Pearson's correlation coefficients are 0.51 and 0.61 for lead and cadmium untransformed, respectively, and 0.81 and 0.60 for the respective log-transformed variables (Table 2a).

Definitive single factor parametric analysis of variance (ANOVA) tests between individual means ($\alpha = 0.05$) for both raw and natural log transformed data (arithmetic and geometric means) were also evaluated. The results in Table 2a show no significant differences between mean lead ($p=0.76$ arithmetic and $p=0.78$ geometric) and cadmium ($p=0.82$ arithmetic and $p=0.75$ geometric) surface soil concentrations for common homes sampled by both protocols.

Results for linear regression analysis relating cadmium concentrations in a no intercept format in Table 2b show r^2=0.91 and a coefficient of 1.02 that does not vary significantly from 1.0. The regression relating log concentrations shows an r^2=0.96 and a coefficient of 1.02 for cadmium, and an r^2=0.99 and a coefficient of 1.01 for lead. However, the regression for untransformed lead shows an r^2=0.39 with a coefficient of 0.32, suggesting a much weaker relationship and that the 1996 IDHW/ATSDR lead concentrations are considerably greater than the USEPA survey results. However, much of this effect is related to a single outlier value of 12,884 mg/kg found at one home in IDHW/ATSDR dataset. The USEPA survey found 2938 mg/kg at the same home. Tables 2a and 2b show the various statistics with the home containing this outlier removed. The resulting r^2=0.79 and the coefficient of 1.55 for the untransformed lead concentrations suggest that the USEPA lead results may be somewhat higher than the IDHW/ATSDR survey results. Other statistical results are not significantly affected by the removal of the outlier value.

These results suggest that a strong correlation between the two survey results, but lead concentrations determined by the USEPA protocols may be higher than that observed in IDHW/ATSDR survey. This difference, however, was not apparent for cadmium and the magnitude of the increase is likely not indicative of significant methodological differences between the two protocols with respect to exposure point concentrations and risk calculations for other metals. As a result, it is reasonable to combine surface soil results from the two surveys for additional analysis for metals other than lead.

SECTION 3.0 SELECTION OF GEOGRAPHIC SUB-AREAS

Soil contamination data between the IDHW/ATSDR and USEPA surveys may also differ in another important aspect. There may be selection bias associated with the homes sampled in the USEPA surveys. These homes were self-identified based upon a voluntary call-in basis, whereas the IDHW/ATSDR study homes were selected randomly.

To evaluate whether a selection bias exists, data from all surveys were initially combined and parametric single factor ANOVAs applied to the mean soil concentrations by survey within each of the originally identified 1996 IDHW/ATSDR sub-areas (16 total). These analyses showed an insufficient number of common observations to allow a meaningful comparison. This was because the FSPA06/07 and FSPA12 (USEPA) surveys contain relatively few observations (n = 90 and 33, respectively) compared to the 1996 IDHW/ATSDR survey (n = 843). As a result, it was necessary to reduce the number of geographic sub-areas in the analysis.

Further reductions in the number of sub-areas required evaluating confidential IDHW/ATSDR and PHD data sets (1996 to present) to determine the geographic distribution of available blood lead information. These data are key to characterizing blood lead absorption response in the CDARB and to supporting any site-specific dose/response investigation that might be accomplished within the HHRA. Key criteria used in sub-area selection using blood lead data were the inclusion within each sub-area of enough data points to properly estimate blood lead concentrations using established protocols (USEPA 1994), as well as protection of data confidentiality. Due to the relatively large number of observations contained within the IDHW/ATSDR survey dataset, these surface soil lead and cadmium concentrations were also evaluated in this initial determination of proposed geographic subdivisions.

From this evaluation, a total of eight specific sub-areas were selected. Table 3 and Figure 1 show the selected sub-areas. Tables 4 and 5 show total blood lead observation statistics for the Basin by geographic sub-area, respectively. Tables 6 and 7 show summary statistics for IDHW/ATSDR surface soil lead and cadmium concentrations. Total blood lead observations by sub-area range from a minimum of 38 (Mullan) to a maximum of 100 (Osburn). Surface soil lead arithmetic mean concentrations range from 412 (Silverton) to 1212 mg/kg (Mullan). Surface soil cadmium arithmetic mean concentrations range from 1.8 (Lower Basin/Cataldo) to 6.2 mg/kg (Osburn). Total IDHW/ATSDR lead and cadmium observations range from 53 (Silverton), to 189 (Osburn).

These comparisons demonstrate that the proposed sub-areas contain sufficient data to properly characterize blood lead concentrations, and protect data confidentiality. Additionally, both the quantity and range of IDHW/ATSDR surface soil results present within each sub-area are sufficient to properly characterize the variation in exposure and risk from these metals within the CDARB. As a result, the existing surveys can be combined in characterizing these sub-areas with respect to exposure point concentrations and risk calculations.

However, several sub-areas are under-represented in the USEPA surveys. Additionally, the potential for selection bias associated with the USEPA surveys should be evaluated. The USEPA surveys provide the only historical soil concentration data for metals other than lead and cadmium. As a result, several sub-areas lack sufficient data to adequately characterize other metals for the HHRA.

SECTION 4.0 EVALUATION OF ARCHIVAL DATA

To remedy this situation, a number of IDHW/ATSDR samples were retrieved from archives and submitted for re-analysis. Two groups were re-analyzed; 24 samples were analyzed at a USEPA Contract Laboratory Program (CLP) laboratory and 65 samples were re-analyzed at a private laboratory under contract with the State of Idaho (State). Of the 89 total samples re-analyzed, 13 were common homes between the IDHW/ATSDR and USEPA surveys. These re-analyzed samples are excluded in the following analyses. Samples were selected to provide a cross-section of contaminant concentrations within each of the select geographic sub-areas. Table 8 shows comparative results for the re-analyses for lead and cadmium. In this case, IDHW/ATSDR refers to the original 1996 IDHW/ATSDR results from the State laboratory. Archive refers to the re-analyzed samples. Comparison of the results show average relative percent differences (RPD) between IDHW/ATSDR and Archive results of 14% for lead and 16% for cadmium for the combined laboratories, 15% for lead and 28% for cadmium for the CLP laboratory, and 13% for lead and 11% cadmium for the State laboratory.

Single factor ANOVA comparing the arithmetic means (n = 89) show no significant differences overall for the IDHW/ATSDR versus combined laboratory results (p=0.7034 and 0.7533 for lead arithmetic and geometric means, and p=0.7972 and 0.9990 for cadmium arithmetic and geometric means, respectively). Regression analyses relating arithmetic concentrations in a no-intercept format show a coefficient value of 0.87 that is not significantly different from 1.0 and an r^2 =0.997 for lead and a coefficient of 1.0 and an r^2=0.92 for cadmium. These evaluations suggest that analytical results are reproducible for lead and cadmium, and that it is appropriate to use the new archive results to characterize other metals concentrations in soils for the HHRA. As a result, additional historical soil concentration data for these other metals are available and will be

forwarded to EPA contractors for use in determining exposure point concentrations and risk estimates.

However, it is important to determine whether the IDHW/ATSDR/Archive results can appropriately be combined with the USEPA surveys by sub-area as the USEPA homes may exhibit selection bias. Table 9 summarizes results of parametric single factor ANOVAs applied to differences between Archival and USEPA means both within the overall basin and within sub-areas, for each of the seven surface soil contaminants of potential concern (COPCs) currently under consideration in the HHRA. With the exception of antimony, significantly greater overall mean COPC concentrations are associated with the USEPA survey results, suggesting that an *overall site-wide* selection bias may exist between IDHW/ATSDR/Archive and USEPA results. With the exception of both iron in several sub-areas and arsenic, lead, and zinc in the Lower Basin/Cataldo area, this observation is generally not supported by ANOVA results within sub-areas. However, the reduced statistical power of the ANOVA procedure may account for the lack of significant differences within sub-areas (particularly Lower Basin/Cataldo). Due to the significantly greater USEPA iron concentrations within five of eight sub-areas, it is reasonable to conclude that surface soils from homes sampled under the USEPA protocols contain significantly greater amounts of iron.

These results suggest that *overall Basin-wide* COPC concentrations obtained under the USEPA protocols on a voluntary self-identified basis are higher than those observed within the 1996 IDHW/ATSDR archive samples, and that a selection bias may exist with the USEPA results. However, the magnitude of the increase is likely not indicative of significant methodological differences between the two protocols with respect to estimating exposure point concentrations and risk calculations within sub-areas of the HHRA for other metals. As a result, it is reasonable to combine surface soil results from the IDHW/ATSDR/Archive and USEPA surveys for additional analysis. Table 10 shows final combined concentration results for the seven COPC metals by proposed geographic sub-area.

SECTION 5.0 SUMMARY OF PROPOSED GEOGRAPHIC SUBDIVISIONS

The eight residential sub-areas shown in Table 1 and Figure 1 are proposed for use in characterizing both lead and non-lead residential exposure within the CDARB HHRA. Table 10 summarizes the results for the seven COPCs by sub-area. Human health risk within the CDARB will be based primarily upon residential exposure point concentrations developed from this combined data set. Residential exposures will be supplemented by potential recreational and occupational exposure present within each of the five major geographic subdivisions shown in Figure 1. For each city, recreational and occupational exposure will be estimated within the respective major geographic subdivision defined by the Conceptual Site Model (CSM) units where the particular city is located. The residential sub-areas of Wallace, Silverton and Osburn are encompassed in the Side Gulches major geographic sub-division and will have similar recreational and occupational exposure point estimates. This will serve to provide for more appropriate site-specific estimates of total exposure within each of these major geographic subdivisions.

SECTION 6.0 REFERENCES

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Table 1
Common Homes Comparison Surface Soil Lead and Cadmium Concentrations (0-1" depth horizon, mg/kg)

Home	Lead			Cadmium		
	USEPA Result	IDHW/ATSDR Result	Relative Percent Difference	USEPA Result	IDHW/ATSDR Result	Relative Percent Difference
1	475	533	11.4	6.4	7.9	21.2
2	1300	527	84.6	7.3	3.9	61.4
3	265	262	1.1	2.2	3.4	40.0
4	306	450	38.1	4.0	6.4	46.6
5	992	808	20.4	4.6	3.7	23.2
6	297	488	48.6	3.1	3.6	13.8
7	872	815	6.8	6.7	5.3	24.3
8	558	510	9.0	5.9	4.7	22.6
9	439	509	14.8	4.0	6.1	40.8
10	64	34	63.2	0.3	N/A	N/A
11	3230	993	105.9	7.1	3.6	66.2
12	2973	1632	58.3	5.0	4.0	22.2
13	1263	656	63.2	9.5	6.6	35.9
14	750	765	1.94	5.1	4.0	23.3
15*	2938	12884	126.0	10.0	10.8	8.0
16	3377	1525	75.6	7.8	5.7	32.0
17	490	315	43.4	4.4	3.9	13.1
18	421	400	5.0	4.6	5.0	7.5
19	439	1253	96.3	5.6	5.8	3.0
20	482	692	35.8	4.9	7.1	37.4
21	186	265	34.9	3.8	4.7	21.8
22	405	424	4.7	5.2	4.2	22.5
23	325	155	70.7	2.7	1.9	37.6
Minimum	64	34	1.1	0.3	1.9	3.0
Maximum	3377	12884	126.0	10.0	10.8	66.2
Arithmetic Mean	993	1169	44.3	5.2	5.1	28.4

* The outlier is not included in the analysis of variance.
N/A: result not available

Table 2a
Common Homes Comparison Surface Soil Lead and Cadmium Concentrations (0"-1" depth horizon) Correlation and ANOVA Results

Pearson's Correlation

		Raw Data		Natural Log Transformed Data	
	N	r	p	r	p
Lead	23	0.51	0.0128	0.81	0.0001
Cadmium	22	0.61	0.0027	0.6	0.0031
Data with Outlier Removed					
Lead	22	0.79	0.0001	0.82	0.0001

ANOVA

		Raw Data		Natural Log Transformed Data	
	N	F_{calc}	p	F_{calc}	p
Lead	45	0.09	0.7637	0.08	0.7818
Cadmium	44	0.05	0.8225	0.11	0.7471
Data with Outlier Removed					
Lead	44	2.20	0.1451	0.68	0.4131

Table 2b
Common Homes Comparison Surface Soil Lead and Cadmium Concentrations (0"-1" depth horizon) Linear Regression Results

		Raw Data		Natural Log	
	N	r^2	Coefficient[1]	r^2	Coefficient[1]
Lead	23	0.39	0.32	0.99	1.01
Cadmium	22	0.91	1.02	0.96	1.02
Data with Outlier Removed					
Lead	22	0.79	1.55	0.99	1.02

[1]USEPA data were dependent variables in these analyses.

Table 3
Survey Sub-Areas Included within Proposed HHRA Geographic Sub-Divisions

Proposed Geographic Subdivisions	Proposed Area Name	Areas included in IDHW/ATSDR Database	Areas included in USEPA FSPA06/07 Database	Areas included in USEPA FSPA12 Database
Mullan	Mullan	Mullan	Mullan	Mullan
Burke/Nine Mile	Nine Mile	Burke, Nine Mile	Burke, Gem, Black Cloud, Woodland Park	Burke, Black Cloud, Woodland Park
Wallace	Wallace	Wallace	Wallace	Wallace
Silverton	Silverton	Silverton	Silverton	Silverton
Osburn	Osburn	Osburn	Osburn	Osburn
Side Gulches	Side Gulches	Big Creek, Elk Creek, Montgomery Gulch, Moon Gulch, Nuckols Gulch, Sunny Slopes, Terror Gulch, Two Mile	Elk Creek, Moon Gulch, Nuckols Gulch, Terror Gulch	Nuckols Gulch
Kingston	Kingston	Kingston, Pine Creek	Kingston, Pinehurst	Kingston
Lower Basin/Cataldo	Lower Basin	Lower Basin	Cataldo	Cataldo

Table 4
Proposed Geographic Total Sub-Area Blood Lead Summary Data for Children (1-9 years old)

Area	Number of Observations				Mean	Variance	Standard Deviation	Minimum	Maximum
	Total	≥10µg/dl	≥15µg/dl	≥20µg/dl					
Mullan	38	4	-	-	5.2	7.3	2.7	2	12
Burke/ Nine Mile	76	16	10	3	7.4	26.9	5.2	1	21
Wallace	77	10	4	2	6	21.4	4.6	1	29
Silverton	73	6	3	1	4.9	13.4	3.7	1	23
Osburn	100	4	-	-	4.1	5.7	2.4	1	13
Side Gulches	51	2	1	-	4.3	7.9	2.8	1	16
Kingston	54	6	4	-	4.8	16.3	4.0	1	16
Lower Basin/ Cataldo	55	10	3	-	5.5	19.6	4.4	1	18
Total	**524**	**58**	**25**	**6**	**-**	**-**	**-**	**-**	**-**

Table 5
Annual Blood Lead Summary Data by Proposed Geographic Sub-Area for Children
(1-9 years old)

Year	Number of Observations				Mean	Variance	Standard Deviation	Minimum	Maximum
	Total	≥10µg/dl	≥15µg/dl	≥20µg/dl					
Mullan Area									
1996	11	-	-	-	3.7	2.6	1.6	2	7
1997	-	-	-	-	-	-	-	-	-
1998	5	1	-	-	7.6	4.3	2.1	6	11
1999	22	3	-	-	5.3	8.2	2.9	2	12
Total	38	4	-	-	5.2	7.3	2.7	2	12
Burke/Nine Mile Area									
1996	17	6	3	-	8.3	23.9	4.9	1	17
1997	8	3	2	-	8.2	54.8	7.4	2	19
1998	18	4	3	2	7.5	41.0	6.4	2	21
1999	33	3	2	1	6.6	15.9	4.0	1	20
Total	76	16	10	3	7.4	26.9	5.2	1	21
Wallace Area									
1996	14	1	-	-	4.2	7.9	2.8	2	11
1997	1	-	-	-	2	-	-	2	2
1998	28	4	1	-	5.9	12.9	3.6	1	16
1999	34	5	3	2	6.8	33.0	5.8	2	29
Total	77	10	4	2	6	21.4	4.6	1	29
Silverton Area									
1996	14	2	1	-	5.5	14.4	3.8	2	16
1997	5	-	-	-	4.4	6.8	2.6	2	8
1998	26	-	-	-	4.1	3.2	1.8	1	8
1999	28	4	2	1	5.4	23.9	4.9	1	23
Total	73	6	3	1	4.9	13.4	3.7	1	23
Osburn Area									
1996	15	1	-	-	4	8.1	2.9	1	13
1997	7	-	-	-	3.8	3.5	1.9	1	7
1998	22	-	-	-	4	3.7	1.9	1	8
1999	56	3	-	-	4	6.4	2.5	1	11
Total	100	4	-	-	4.1	5.7	2.4	1	13
Side Gulches Area									
1996	8	-	-	-	2.7	1.1	1.0	1	4
1997	-	-	-	-	-	-	-	-	-
1998	12	1	-	-	5	9.7	3.1	3	14
1999	31	1	1	-	4.3	8.4	2.9	1	16
Total	51	2	1	-	4.3	7.9	2.8	1	16

Table 2.1.2
Annual Blood Lead Summary Data by Proposed Geographic Sub-Area for Children (1-9 years old) (continued)

Year	Number of Observations				Mean	Variance	Standard Deviation	Minimum	Maximum
	Total	≥10µg/dl	≥15µg/dl	≥20µg/dl					
Kingston Area									
1996	7	1	1	-	6.4	22.3	4.7	2	16
1997	-	-	-	-	-	-	-	-	-
1998	8	-	-	-	2.8	3.6	1.9	1	7
1999	39	5	3	-	4.8	17.2	4.2	1	16
Total	54	6	4	-	4.8	16.3	4.0	1	16
Lower Basin/Cataldo Area									
1996	12	4	1	-	5.2	31.1	5.6	1	18
1997	5	1	-	-	5.4	15.8	4.0	2	12
1998	9	1	-	-	3.5	14.0	3.8	1	13
1999	29	4	2	-	6.2	17.6	4.2	1	18
Total	55	10	3	-	5.5	19.6	4.4	1	18

Table 6
Proposed Geographic Sub-Area Soil Lead Summary Statistics
from the 1996 IDHW/ATSDR Exposure Survey

Area	Arithmetic Mean	Geometric Mean	N	Standard Deviation	Minimum	Maximum
Mullan	1212	602	88	2403	41	20218
Burke/Nine Mile	1029	623	70	826	32	3250
Wallace	1024	764	78	799	54	4285
Silverton	412	331	53	300	98	1724
Osburn	727	476	189	1227	43	12884
Side Gulches	486	321	108	542	25	3920
Kingston	823	255	77	1824	22	9228
Lower Basin/Cataldo	455	108	152	1196	15	7350

Table 7
Proposed Geographic Area Soil Cadmium Summary Statistics
from the 1996 IDHW/ATSDR Exposure Survey

Area	Arithmetic Mean	Geometric Mean	N	Standard Deviation	Minimum	Maximum
Mullan	3.9	2.7	88	3.6	0.3	16.3
Burke/Nine Mile	4.8	3.3	70	3.6	0.1	21.4
Wallace	5.3	4.6	78	2.7	1.3	14.7
Silverton	4.2	3.4	53	3.5	0.7	24.9
Osburn	6.2	5.4	189	3.6	0.9	21.4
Side Gulches	4.8	3.8	108	2.9	0.3	14.6
Kingston	2.8	1.9	77	3.1	0.1	14.7
Lower Basin/Cataldo	1.8	1	152	2.4	0.1	12.5

Table 8 Comparisons between Original IDHW/ATSDR and Reanalyzed Archived Sample Results

				Lead			Cadmium		
Result	Sub-Area	Laboratory	Common Homes	IDHW/ATSDR 1996 Results	Archive Sample Reanalysis	Relative Percent Difference	IDHW/ATSDR 1996 Results	Archive Sample Reanalysis	Relative Percent Difference
1	KINGSTON	State		157	161	2.3%	2.0	2.5	20.3%
2	LOWER BASIN/CATALDO	State		66	66	0.5%	0.9	0.5	56.1%
3	LOWER BASIN/CATALDO	State		322	334	3.8%	2.5	2.9	14.4%
4	LOWER BASIN/CATALDO	State		199	216	8.3%	1.9	1.9	2.7%
5	KINGSTON	State		226	243	7.2%	2.3	2.2	2.2%
6	KINGSTON	State		145	156	7.5%	1.5	1.7	12.5%
7	LOWER BASIN/CATALDO	State		27	28	3.2%	0.4	0.5	22.2%
8	LOWER BASIN/CATALDO	State		432	439	1.7%	1.7	2.0	14.5%
9	LOWER BASIN/CATALDO	State		134	117	13.7%	1.4	1.5	7.6%
10	LOWER BASIN/CATALDO	State		43	38	13.9%	0.6	0.5	24.6%
11	LOWER BASIN/CATALDO	State		401	326	20.7%	2.5	2.4	2.5%
12	LOWER BASIN/CATALDO	State		175	145	18.6%	2.4	2.3	5.5%
13	MULLAN	State		198	184	7.4%	1.8	2.0	10.5%
14	MULLAN	State		49	47	5.6%	0.5	0.5	0.0%
15	MULLAN	State		696	602	14.4%	2.4	2.7	11.8%
16	MULLAN	State		115	109	5.0%	1.6	1.7	9.2%
17	MULLAN	State		1559	1180	27.7%	6.4	6.1	5.4%
18	MULLAN	State		314	298	5.3%	1.2	1.5	22.2%
19	MULLAN	State		626	548	13.2%	2.8	3.1	10.2%
20	MULLAN	State		2015	1820	10.2%	12.2	12.2	0.0%
21	MULLAN	State		3750	3370	10.7%	12.9	13.4	3.8%
22	MULLAN	State		1095	960	13.1%	5.0	5.2	4.9%
23	MULLAN	State		5437	5140	5.6%	10.7	10.5	1.9%
24	MULLAN	State	x	1632	1180	32.1%	4.0	4.0	0.8%
25	BURKE/NINE MILE	State	x	993	783	23.7%	3.6	3.4	4.6%
26	BURKE/NINE MILE	State	x	815	591	31.9%	5.3	4.4	17.6%
27	BURKE/NINE MILE	State	x	808	665	19.4%	3.7	3.7	1.1%
28	BURKE/NINE MILE	State	x	34	29	14.4%		0.5	
29	OSBURN	State	x	488	372	27.0%	3.6	3.3	7.6%
30	OSBURN	State	x	533	408	26.5%	7.9	7.4	7.0%
31	OSBURN	State	x	510	404	23.3%	4.7	4.7	0.0%
32	OSBURN	State	x	12884	10900	16.7%	10.8	10.1	6.7%
33	OSBURN	State	x	450	355	23.7%	6.4	6.0	5.7%
34	OSBURN	State	x	315	256	20.7%	3.9	3.7	4.2%
35	SIDE GULCHES	State		492	498	1.2%	7.2	8.1	11.4%
36	SIDE GULCHES	State		215	226	5.2%	4.0	4.3	7.7%
37	SIDE GULCHES	State		25	28	9.8%	0.4	0.6	52.6%
38	SIDE GULCHES	State		284	307	7.7%	2.1	2.4	12.9%
39	SIDE GULCHES	State		3356	3150	6.3%	14.6	15.0	2.7%
40	SIDE GULCHES	State		1058	936	12.2%	13.7	14.6	6.4%
41	SILVERTON	State		528	528	0.1%	3.6	3.9	8.8%
42	SILVERTON	State		316	325	2.8%	3.7	3.9	4.5%
43	SILVERTON	State		616	591	4.2%	3.6	4.1	14.1%
44	SILVERTON	State		115	117	1.6%	1.7	1.8	8.1%
45	SILVERTON	State		755	750	0.7%	3.7	3.7	1.4%
46	SILVERTON	State		142	154	8.0%	1.2	1.4	14.6%
47	SILVERTON	State	x	656	546	18.4%	6.6	6.6	0.2%
48	SILVERTON	State		1724	1560	10.0%	11.6	11.9	2.6%
49	SILVERTON	State		747	679	9.5%	7.8	7.8	0.4%
50	SILVERTON	State	x	262	217	18.8%	3.4	3.1	8.0%
51	SILVERTON	State		217	219	1.0%	2.7	2.8	5.5%
52	SILVERTON	State		432	383	12.0%	3.6	3.8	6.2%
53	SILVERTON	State		196	221	12.2%	2.8	3.3	16.0%
54	SILVERTON	State		231	247	6.5%	3.6	3.9	7.7%
55	SILVERTON	State		424	455	7.0%	3.1	3.5	13.4%
56	BURKE/NINE MILE	State	x	765	623	20.4%	4.0	4.1	2.0%
57	LOWER BASIN/CATALDO	State	9&11	1337	1180	12.5%	5.5	6.0	8.2%
58	LOWER BASIN/CATALDO	State	2&1	1231	1000	20.7%	7.1	6.9	3.4%
59	LOWER BASIN/CATALDO	State	21&57	34	34	0.6%	0.4	0.7	66.7%
60	LOWER BASIN/CATALDO	State	10&34	6084	5290	14.0%	10.3	9.6	7.0%
61	LOWER BASIN/CATALDO	State	16&86	51	45	12.1%	0.7	0.8	7.8%
62	LOWER BASIN/CATALDO	State	13&119	285	240	17.0%	1.3	1.4	7.4%
63	LOWER BASIN/CATALDO	State	4&127	269	463	53.0%	1.0	2.2	75.0%
64	LOWER BASIN/CATALDO	State	17&135	26	14	59.0%		0.5	
65	LOWER BASIN/CATALDO	State	19&139	184	151	19.5%	2.6	2.2	15.1%
66	KINGSTON	CLP		572	493	14.8%	4.6	4.1	10.4%
67	KINGSTON	CLP		24	21	14.7%	0.3	0.1	115.8%
68	LOWER BASIN/CATALDO	CLP		178	189	6.1%	1.3	1.0	29.1%
69	KINGSTON	CLP		379	343	9.9%	2.3	2.5	6.6%
70	KINGSTON	CLP		1356	1260	7.3%	3.2	3.0	5.2%
71	KINGSTON	CLP		71	71	0.4%	0.5	0.5	6.2%
72	KINGSTON	CLP		226	208	8.2%	1.6	1.3	19.4%
73	KINGSTON	CLP		111	108	2.4%	0.9	0.8	17.1%
74	LOWER BASIN/CATALDO	CLP		58	333	141.0%	1.0	14.7	175.0%
75	LOWER BASIN/CATALDO	CLP		1407	1280	9.5%	6.9	6.4	7.7%
76	LOWER BASIN/CATALDO	CLP		30	31	3.7%	0.3	0.1	85.7%
77	SIDE GULCHES	CLP		84	73	14.2%	2.3	2.0	14.4%
78	SIDE GULCHES	CLP		430	353	19.6%	5.6	4.7	17.1%
79	SIDE GULCHES	CLP		545	477	13.3%	6.3	6.4	1.6%
80	SIDE GULCHES	CLP		100	93	8.2%	2.5	2.3	8.7%
81	SIDE GULCHES	CLP		811	1030	23.7%	2.2	6.2	97.0%
82	SIDE GULCHES	CLP		417	372	11.5%	7.7	7.4	3.8%
83	SIDE GULCHES	CLP		3920	3300	17.2%	11.7	11.0	6.2%
84	SIDE GULCHES	CLP		96	89	7.7%	2.3	2.1	9.5%

Table 8 Comparisons between Original IDHW/ATSDR and Reanalyzed Archived Sample Results (continued)

Result	Sub-Area	Laboratory	Common Homes	Lead			Cadmium		
				IDHW/ATSDR 1996 Results	Archive Sample Reanalysis	Relative Percent Difference	IDHW/ATSDR 1996 Results	Archive Sample Reanalysis	Relative Percent Difference
85	SIDE GULCHES	CLP		179	160	11.0%	2.1	1.7	19.1%
86	SIDE GULCHES	CLP		801	744	7.4%	8.6	8.6	0.0%
87	SIDE GULCHES	CLP		414	402	2.8%	5.1	5.7	11.1%
88	SIDE GULCHES	CLP		930	863	7.4%	8.2	8.1	1.1%
89	SIDE GULCHES	CLP		509	466	8.9%	6.1	5.9	3.3%
Combined	Arithmetic Mean			835	746	13.9%	4.2	4.3	16.0%
	Geometric Mean			341	320		2.9	2.9	
	Minimum			24	14		0.3	0.1	
	Maximum			12884	10900		14.6	15.0	
	p Value (Geometric Mean)			0.7034			0.7972		
	p Value (Arithmetic Mean)			0.7533			0.9990		

Table 9 Comparison of IDHW/ATSDR and USEPA by Geographic Sub-Area

		N		Arithmetic Means			Geometric Mean		
Area/Subarea	**Chemical of Concern**	**USEPA**	**ARCHIVE**	**USEPA**	**ARCHIVE**	**P**	**USEPA**	**ARCHIVE**	**P**
Overall Basin	Antimony	120	76	5.7	4.9	0.5714	3.5	2.6	0.0394
	Arsenic	120	76	20.8	18.9	0.4944	17.8	12.8	0.0006
	Cadmium	120	76	5.2	4.2	0.0498	4.1	2.7	0.0014
	Iron	111	76	20428.3	17808.3	0.0117	19705.2	16855.3	0.0002
	Lead	120	76	1085.0	658.1	0.0760	559.8	300.5	0.0004
	Manganese	111	76	996.2	870.8	0.1702	902.2	717.2	0.0019
	Zinc	120	76	646.2	427.0	0.0014	503.3	289.7	0.0001
Mullan	Antimony	9	11	9.6	7.2	0.5214	6.2	5.0	0.6423
	Arsenic	9	11	19.3	14.7	0.3572	16.0	12.6	0.3835
	Cadmium	9	11	4.4	5.4	0.6262	3.6	3.6	0.9510
	Iron	9	11	23226.9	17100.0	0.0662	21886.0	16501.0	0.0651
	Lead	9	11	1567.2	1296.1	0.6921	1116.4	594.4	0.2662
	Manganese	9	11	1483.2	1235.2	0.4692	1311.8	1045.3	0.3982
	Zinc	9	11	810.4	746.2	0.8039	717.6	493.8	0.3314
Burke/Nine Mile	Antimony	26	2	4.4	7.3	0.3488	3.4	7.2	0.1245
	Arsenic	26	2	15.8	11.5	0.4083	14.6	11.5	0.4121
	Cadmium	26	2	5.5	3.8	0.4623	4.4	3.7	0.7767
	Iron	24	2	18875.1	16585.0	0.5905	18117.4	16323.2	0.6274
	Lead	26	2	915.2	703.0	0.7323	639.0	698.4	0.8921
	Manganese	24	2	945.8	718.5	0.4092	881.9	716.2	0.4585
	Zinc	26	2	868.6	414.0	0.2410	724.6	411.0	0.2259
Wallace	Antimony	17	0	10.4	-	-	4.6	-	-
	Arsenic	17	0	19.7	-	-	18.2	-	-
	Cadmium	17	0	6.7	-	-	5.7	-	-
	Iron	16	0	21356	-	-	20846.4	-	-
	Lead	17	0	2109.7	-	-	1144.8	-	-
	Manganese	16	0	967.6	-	-	890.6	-	-
	Zinc	17	0	853.5	-	-	758.1	-	-
Silverton	Antimony	7	13	4.9	3.3	0.5165	2.2	2.2	0.9988
	Arsenic	7	13	12.6	12.2	0.8484	12.1	11.3	0.7066
	Cadmium	7	13	4.1	4.3	0.8571	3.2	3.7	0.6350
	Iron	6	13	18464.2	14925.4	0.0107	18203.0	14802.6	0.0128
	Lead	7	13	1271.8	479.2	0.2068	475.9	376.2	0.6274
	Manganese	6	13	896.6	714.5	0.1987	838.8	675.3	0.2594
	Zinc	7	13	417	389.5	0.8193	351.1	321.4	0.7773
Osburn	Antimony	42	0	4.9	-	-	3.7	-	-
	Arsenic	42	0	23.7	-	-	21.0	-	-
	Cadmium	42	0	5.2	-	-	4.3	-	-
	Iron	39	0	19801.9	-	-	19512.4	-	-
	Lead	42	0	723.7	-	-	421.2	-	-
	Manganese	39	0	955.2	-	-	914.2	-	-
	Zinc	42	0	466	-	-	385.1	-	-
Side Gulches	Antimony	8	18	2.6	6.0	0.1861	2.4	3.3	0.4646
	Arsenic	8	18	21.1	35.1	0.3600	19.5	21.3	0.8015
	Cadmium	8	18	3.7	6.2	0.1273	3.1	4.7	0.2488
	Iron	8	18	21737.3	22553.9	0.8318	21565.2	20979.3	0.8373
	Lead	8	18	324.6	727.8	0.2545	267.5	367.3	0.5169
	Manganese	8	18	865.2	1042.7	0.6022	810.8	824.4	0.9459
	Zinc	8	18	317.6	499.8	0.2241	288.2	377.3	0.3806
Kingston	Antimony	8	16	2.5	2.8	0.7494	2.0	1.9	0.9118
	Arsenic	8	16	14.4	15.6	0.8321	13.9	12.4	0.6448
	Cadmium	8	16	3.9	2.1	0.0318	3.4	1.5	0.0553
	Iron	7	16	17239.8	16227.5	0.4635	17223.2	15901.8	0.3254
	Lead	8	16	385.9	333.2	0.6903	350.1	213.0	0.2115
	Manganese	7	16	631.9	626.9	0.9585	623.5	580.8	0.6734
	Zinc	8	16	406.9	252.1	0.0495	377.2	205.3	0.0296
Lower Basin/Cataldo	Antimony	3	16	7.4	5.4	0.7591	3.5	1.8	0.4605
	Arsenic	3	16	67.8	13.1	0.0013	44.2	8.5	0.0135
	Cadmium	3	16	8.1	3.2	0.1041	2.3	1.6	0.7329
	Iron	2	16	43080	17032.5	0.0084	36834.9	15812.3	0.0120
	Lead	3	16	3817.5	605.6	0.0091	766.8	158.7	0.2152
	Manganese	2	16	2537.4	816.6	0.0632	1307.5	613.6	0.2218
	Zinc	3	16	1622.9	332.7	0.0031	679.4	185.1	0.1273

Table 10
Final Database for Characterizing Metals Concentrations other than Lead
by Geographic Sub-area

	Mullan	Burke/ Nine Mile	Wallace	Silverton	Osburn	Side Gulches	Kingston	Lower Basin/ Cataldo
N	20	28	17	20	42	26	24	19
ANTIMONY								
Arithmetic Mean	8.3	4.6	10.4	3.8	4.9	5.0	2.7	5.7
Minimum	1.3	1.1	1.4	0.4	1.3	0.5	0.4	0.5
Maximum	33.2	18.2	91.9	22.8	36.4	28.1	7.8	40.3
Standard Deviation	8.0	4.1	21.8	5.1	5.6	6.1	2.1	9.9
Geometric Mean	5.5	3.6	4.6	2.2	3.7	3.0	2.0	2.0
Geometric Standard Deviation	1.55	1.53	1.70	2.38	1.49	1.93	2.29	3.04
ARSENIC								
Arithmetic Mean	16.8	15.5	19.7	12.3	23.7	30.8	15.2	21.7
Minimum	6.2	7.2	7.3	4.6	9.7	6.2	5.1	2.3
Maximum	42.5	36.9	40.7	17.6	83.2	140.0	66.7	108.0
Standard Deviation	10.9	6.9	8.1	3.9	13.7	35.3	12.2	30.1
Geometric Mean	14.0	14.3	18.2	11.6	21.0	20.7	12.9	11.0
Geometric Standard Deviation	1.23	1.14	1.15	1.16	1.16	1.27	1.21	1.46
CADMIUM								
Arithmetic Mean	4.9	5.3	6.7	4.2	5.2	5.4	2.7	4.0
Minimum	0.5	0.3	1.4	1.0	1.0	0.6	0.1	0.1
Maximum	13.4	12.9	18.8	11.9	19.7	15.0	6.9	14.7
Standard Deviation	4.0	3.1	4.1	2.7	3.5	3.8	1.9	4.7
Geometric Mean	3.6	4.3	5.7	3.5	4.3	4.1	1.9	1.7
Geometric Standard Deviation	1.66	1.51	1.35	1.48	1.45	1.59	2.53	3.91

Table 10
Final Database for Characterizing Metals Concentrations other than Lead by Geographic Sub-area (continued)

	Mullan	Burke/ Nine Mile	Wallace	Silverton	Osburn	Side Gulches	Kingston	Lower Basin/ Cataldo
N	20	28	17	20	42	26	24	19
IRON								
Arithmetic Mean	19857	18699	21356	16043	19802	22303	16536	19927
Minimum	8730	10070	15720	11890	14000	13870	11600	11740
Maximum	43167	36840	37660	23225	36800	52600	24230	65420
Standard Deviation	7470	5625	5326	2962	3764	8775	2961	14022
Geometric Mean	18737	17973	20846	15801	19512	21158	16293	17370
Geometric Standard Deviation	1.03	1.03	1.02	1.02	1.02	1.03	1.02	1.05
LEAD								
Arithmetic Mean	1418	900	2110	757	724	604	351	1113
Minimum	47	64	346	94	110	28	21	14
Maximum	5140	3948	16026	6098	8739	3300	1260	7100
Standard Deviation	1465	823	3703	1315	1360	819	296	2072
Geometric Mean	789	643	1145	408	421	333	251	203
Geometric Standard Deviation	1.18	1.13	1.14	1.17	1.15	1.19	1.16	1.37
MANGANESE								
Arithmetic Mean	1347	928	968	772	955	988	628	1008
Minimum	414	344	510	284	448	408	232	251
Maximum	3159	1946	2278	1326	1819	3600	1090	4712
Standard Deviation	737	365	448	282	288	779	208	1246
Geometric Mean	1158	868	891	723	914	820	593	667
Geometric Standard Deviation	1.08	1.06	1.06	1.06	1.04	1.08	1.06	1.12

Table 10
Final Database for Characterizing Metals Concentrations other than Lead by Geographic Sub-area (continued)

	Mullan	Burke/ Nine Mile	Wallace	Silverton	Osburn	Side Gulches	Kingston	Lower Basin/ Cataldo
N	20	28	17	20	42	26	24	19
ZINC								
Arithmetic Mean	775	836	854	399	466	444	304	536
Minimum	89	205	334	116	131	86	50	35
Maximum	1890	2176	2278	1010	1467	1360	714	2670
Standard Deviation	552	521	472	247	315	347	184	754
Geometric Mean	584	696	758	331	385	347	251	227
Geometric Standard Deviation	1.13	1.10	1.07	1.11	1.10	1.12	1.12	1.25

APPENDIX O

Selected EPA Guidance Documents for Lead

Appendix O

Selected EPA Guidance Documents for Lead

This appendix contains three EPA guidance documents for conducting lead human health risk assessments. These documents are:

1. Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities, written by the Office of Solid Waste and Emergency Response in August 1994 (OSWERdir9355.4-12.pdf).
2. Clarification to the 1994 Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities, written by the Office of Solid Waste and Emergency Response in August 1998 (OSWER98-9200.4-27P.pdf).
3. IEUBK Model Bioavailability Variable, written by the Technical Review Workgroup for Lead in October 1999 (IEUBK Model Bioavailability.pdf).

IEUBK Model Bioavailability Variable

INTRODUCTION

Performance of the Integrated Exposure Uptake Biokinetic Model for Lead (Pb) in Children (IEUBK) is a function of site-specific parameter input values. A site-specific determination of soil-borne lead bioavailability is, therefore, advantageous for improving predictiveness of the model. This short sheet discusses issues to consider and applicable methods for determining a site-specific bioavailability value for soil-borne lead.

The current default estimate for the bioavailability of soil/dust in the IEUBK model is 30 percent as an absolute value. This absorption fraction is partitioned into a non-saturable component (6 percent) and a saturable component (24 percent). Investigators (Casteel *et al.*, 1997; Henningsen *et al.*, 1998) have observed variable bioavailability across different soil/lead matrices, although the majority of samples are generally consistent with the IEUBK default value. Soil particle size (for soils sieved to <250 µm or 60-mesh), mineralogy, and lead speciation are among the factors that influence bioavailability (Steele *et al.*, 1990).

In Vitro techniques, such as the physiologically-based extraction test (PBET - Ruby *et al.*, 1996), have been developed as a means of capturing the impact of the soil/lead matrix on bioavailability. However, physico-chemical characteristics of the soil/lead matrix are not the sole determinants of the highly complex biological process of gastrointestinal absorption. In effect, solubility and bioavailability are not interchangeable terms. Until such time that fully validated *in vitro* techniques become generally accepted, the recommended approach to demonstrating site-specific bioavailability will need to be supported by an appropriate animal bioassay.

This short sheet reaffirms the provisions of the 1995 *Administrative Reform for Lead* that requires review of data that may set a precedent. Bioavailability data (other than from published studies using the juvenile swine model) that are intended for use in an EPA risk assessment using the IEUBK should be sent for review by the Office of Emergency and Remedial Response. This review not only promotes better science but also promotes sharing of information so that all EPA Regions can benefit from new information/analyses.

DEFINITIONS

As indicated in the Guidance Manual for the IEUBK Model, **bioavailability** refers to "the fraction of the total amount of material in contact with a body portal-of-entry (lung, gut, skin) that enters the blood." Bioavailability is also described as absolute or relative (USEPA, 1994). **Absolute bioavailability** is the amount of a substance entering the blood via a particular route of exposure (*e.g.*, gastrointestinal) divided by the total amount administered (*e.g.*, soil lead ingested). **Relative bioavailability** is indexed by measuring the bioavailability of a particular substance relative to the bioavailability of a standardized reference material, such as soluble lead acetate.

It should be noted that the bioavailability input parameter in the IEUBK model is an absolute value, but it may be experimentally determined by relative means, provided that the absolute bioavailability of the "standardized reference material" is known. For the IEUBK model, soluble lead in water and food is estimated to have 50 percent absolute bioavailability. The model presumes that the relative bioavailability of lead in soil is 60 percent, thus producing an absolute bioavailability for soil lead of 30 percent (*i.e.*, 60% x 50% = 30%). It is acknowledged that this value has significant variability and uncertainty, but it is the estimate under which the IEUBK model was validated with comprehensive blood lead study results.

"**Bioaccessability**" is a term used in describing an event that relates to the absorption process. It generally refers to the fraction of administered substance that becomes solubilized in the gastrointestinal fluid. For the most part, solubility is a prerequisite of absorption, although small amounts of lead in particulate or suspended/emulsified form may be absorbed by pinocytosis. Moreover, it is not simply the **fraction** dissolved that determines bioavailability, but also the **rate** of dissolution, which has physiological and geochemical influences. In and of itself, bioaccessability is not a direct measure of the movement of a substance across a biological membrane (*i.e.*, absorption or bioavailability). The relationship of bioaccessability to bioavailability is ancillary and the former need not be known in order to measure the latter.

However, bioaccessability (*i.e.*, solubility) may serve as a surrogate for bioavailability if certain conditions are met (see *Methods and Issues to Consider when Determining Site-Specific Bioavailability of Soil-Borne Lead*).

As previously mentioned, lead absorption is believed to occur by both **active** and **passive** mechanisms. Although the precise subcellular processes involved in lead absorption are not entirely known, active/passive absorption processes (depending on dose) can impart a curvilinear shape to a graph of dose *vs* blood lead concentration. The potential impact of active and passive absorption processes on the determination of relative bioavailability is discussed in a latter section (*Methods and Issues to Consider when Determining Site-Specific Bioavailability of Soil-Borne Lead*).

WHEN TO CONSIDER ADJUSTMENTS IN BIOAVAILABILITY

As stated in the *Introduction*, the bioavailability of soil-borne lead is influenced by numerous characteristics of the soil-lead matrix. Particle size has been demonstrated to effect soil-lead bioavailability (Steele *et al.*, 1990). Although a strong quantitative relationship between particle size and bioavailability has not been established, an understanding of particle size distribution in a soil-lead source may provide qualitative information on the potential bioavailability of the source material. Perhaps more importantly, available data (Henningsen *et al.*, 1998) indicate that lead speciation can have a significant effect on bioavailability.

Currently, *in vivo* bioassays are the only way to quantitatively measure and adjust default bioavailability to fit site soils. However, validation studies are in progress which show promise for *in vitro* tests which may be correlated to the *in vivo* results. Such a test would have obvious and much needed advantages of speed, affordability, simplicity, and higher throughput. Until such tests are sufficiently validated with *in vivo* data, the use of *in vitro* bioaccessibility results are deemed by EPA to represent insufficient evidence for quantitative adjustment of bioavailability. The reason for this position is that small changes in *in vitro* assays, such as pH, time, temperature, volume, other solutes, and agitation regimes, can have relatively large impacts on results of lead solubility. Until validation is confirmed, the use of a simpler, faster, and cheaper lab benchtop test will not, in and of itself, be judged an adequate surrogate for measuring bioavailability.

Results of tests by EPA using animal models have shown a general pattern of relative bioavailability for certain lead salts. While lead speciation is not the sole factor influencing bioavailability, these patterns can, nonetheless, be used to compare a site's form of soil lead to explore differences in bioavailability relative to the defaults. If the lead speciation profile suggests a bioavailability estimate substantially different from the IEUBK model default, then the costs and benefits of performing supporting animal tests for now, and possibly of *in vitro* tests after validation, can be considered for quantitative measures of bioavailability, and adjustments for a specific site. Furthermore, qualitative estimates of relative bioavailability can be made in the uncertainty section of a risk assessment. General patterns of relative bioavailability determined by EPA Region 8 studies of 20 soil lead samples (Henningsen *et al.*, 1998), compared to the default soil relative bioavailability of 60 percent, are shown as groups below:

Potentially Lower Bioavailability (RBA < 25%)	**Intermediate Bioavailability (RBA = 25% to 75%)**	**Potentially Higher Bioavailability (RBA > 75%)**
Galena (PbS) Anglesite ($PbSO_4$) Pb (M) Oxides Pb Fe (M) Sulfates Native Pb	Pb Oxide Pb Fe (M) Oxides Pb Phosphate Slags	Cerrusite ($PbCO_3$) Pb Mn (M) Oxides

Pb = lead, S = sulfur, M = metals, Fe = iron, Mn = manganese

Results of well-conducted blood lead studies can infer relatively low bioavailability of lead in soil. Such findings would not support a quantitative adjustment of bioavailability, but could assist in identifying soils for further study and/or support a qualitative adjustment in the risk characterization section of a risk assessment.

METHODS AND ISSUES TO CONSIDER WHEN DETERMINING SITE-SPECIFIC BIOAVAILABILITY OF SOIL-BORNE LEAD

Ethics aside, in a hypothetical setting the ideal method for making a bioavailability adjustment for soil-borne lead in the IEUBK model would be to dose a large group of young children with soil-borne lead and compare the area-under-the-concentration/time curve (AUC) with the AUC of the same or similar group which received an equal lead dose by intravenous administration. This is the conventional pharmacological and toxicological method for measuring *absolute* bioavailability. Realistically, issues of ethics, cost, and implementation are important determinants of study design. Consequently, an alternate approach is to measure soil-lead bioavailability *relative* to a "standardized reference material" (see *Definitions* section).

Determination of relative bioavailability needs to consider the experimental evidence suggesting that

gastrointestinal lead absorption follows first-order saturation kinetics. An example is presented to illustrate that relative bioavailability, as estimated from experimental studies, can depend strongly on the response levels at which comparisons are made. The approach used to estimate relative bioavailability is to compare doses of lead (in different forms) that, upon ingestion by an experimental animal, produce equal levels of biological response (in this example, blood lead concentrations). The curves in the Figure illustrate relationships that may be fit to experimental data on the relationship between the ingested dose of lead and resulting blood lead measures. The two curves are of the Michaelis-Menten form (Equation 1) with vmax = 30, km = 1 in the soluble lead relationship and vmax = 10, km = 0.4 for the soil lead relationship.

$$\text{Absorption rate} = \frac{vmax \times dose}{km + dose} \qquad \textbf{Equation 1}$$

where

vmax = maximum rate at which an enzyme can function.

km = concentration of substrate that produces 50% maximum velocity of the enzyme.

This example is hypothetical, in that the curves shown are for purposes of illustration and are not intended to represent a specific data set. However, similar models, using Michaelis-Menten form equations, have been presented to EPA as models of bioavailability data from rodent studies conducted with soils from Superfund sites.

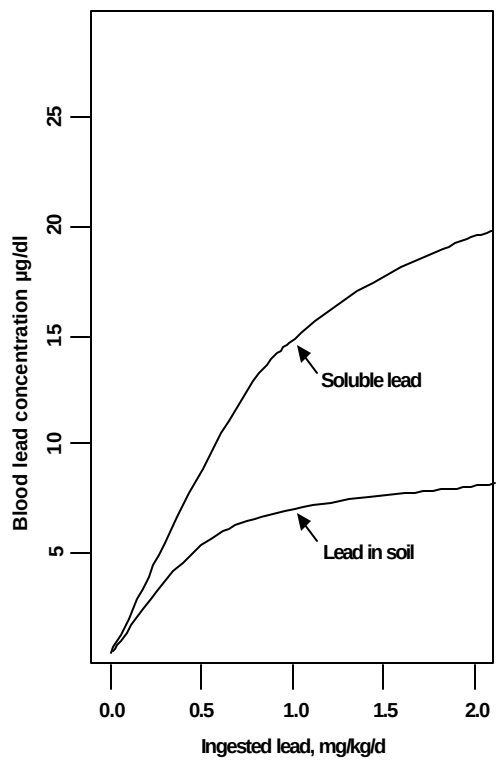
To estimate relative bioavailability in this example, a reference blood lead concentration is selected (5 µg/dL). The dose levels of soluble lead and lead in soil, respectively, at which this blood concentration is produced are then estimated. As illustrated in the Figure, a dose of 0.2 mg/kg/d of soluble lead is associated with a blood lead level of 5 µg/dL, while a dose of lead in soil of 0.4 mg/kg/d is required to achieve this same level. The relative bioavailability is estimated to be 0.5 or 50 percent based on the ratio of these doses (0.2/0.4).

However, in this example, where the soluble lead graph and the soil lead graph show different curvatures (specifically resulting from the different km values in the example), the estimated relative bioavailability depends on blood lead level at which the comparisons are made (see figure below).

Note that at low doses the relative bioavailability of the two materials is similar, while at high doses the relative bioavailability of lead in soil is estimated to be low

Hypothetical response curve for lead uptake study

Example showing different stages of response for soil lead and soluble lead



compared with soluble lead. A variety of different mechanistic factors may affect the bioavailability of lead administered at high doses. In experimental studies of bioavailability, substantial amounts of soil may be administered to the experimental animals, and the presence of these high quantities of soil in the diet may affect the bioavailability of lead. Such effects may be due to alterations to the chemical environment of the GI tract. For example, the presence of substantial quantities of soil may provide additional binding sites for lead, reducing the likelihood that any lead which becomes solubilized will remain in solution and be absorbed.

Blood lead concentration for calculating relative bioavailability (µg/dL)	Dose of soluble lead to achieve this concentration (mg/kg/day)	Dose of soil lead to achieve this concentration (mg/kg/day)	Relative bioavailability
1.00	0.03	0.04	0.78
2.00	0.07	0.10	0.71
3.00	0.11	0.17	0.65
4.00	0.15	0.27	0.58
5.00	0.20	0.40	0.50
6.00	0.25	0.60	0.42
7.00	0.30	0.93	0.33
8.00	0.36	1.60	0.23

Due to the potential for high doses of either soil or lead itself to affect (reduce) absorption, experimental bioavailability studies need to be performed at low enough doses to provide a reasonable comparison with the quantities of soil and lead that humans are likely to ingest. Where experimental limitations necessitate that the quantities of soil or lead administered substantially exceed the expected human doses (on a body weight basis), it should be recognized that an extrapolation to lower doses may be appropriate. This extrapolation step may take the form of an explicit mathematical treatment of the data (and as such would need to address the uncertainty in the predictions at low dose) or it may involve a more qualitative demonstration that under the particular experimental conditions utilized, the estimated bioavailability is not highly sensitive to the lead dosage used for comparison.

SITE SOIL HOMOGENENEITY FOR SAMPLE COLLECTION AND PREPARATION

Soil samples that are tested for *in vivo* bioavailability or *in vitro* bioaccessibility should be obtained from areas that are reasonably similar (*i.e.*, similar geophysical and chemical properties of lead in soil). The top 2 inches of surface soil from residential yards should be representatively sampled and composited for testing. It is critical to sieve soil samples to <250 µm (60 mesh) to more closely represent the size of soil particles that would be expected to adhere to children's hands. An extremely useful tool for geophysical-chemical characterization of lead in soil is the electron microprobe (Medlin, 1997). Soil samples which are characterized or tested for bioavailability must retain their integrity, including chain of custody documentation, and proper mixing that provides a uniform subsample without physically degrading the soil particles.

APPROPRIATE ANIMAL MODEL

Because of the difficulties in gathering data on oral absorption of lead in children, there is no validated ***absolute model*** for experimental uses in measuring bioavailability. Each candidate animal model is expected to respond uniquely to absolute lead absorption (*i.e.*, oral uptake *vs*. intravenous dosing), compared to children, because of differences in physiology, diet, behavior, and development. However, it is possible to use a similar mammalian gastrointestinal system to measure ***relative*** absorption in comparison to the uptake of a soluble lead reference material (*e.g.*, lead acetate). This is the concept underlying the juvenile swine model (Weis *et al.*,1994) which has further advantages of permitting sequential blood sampling and responding to doses similar to those experienced by children. Further details on the appropriate design aspects of such studies can be obtained from Weis *et al.*, 1994; Casteel *et al.*, 1997; and Henningsen *et al.*, 1998.

Previous rodent studies have had limitations due to:

(1) rapid development which often resulted in testing of sexually mature animals which may have lost some of their active transport uptake of lead;

(2) inability to produce AUC blood lead results *vs*. time, due to rodents' small size which precludes repeat blood sampling;

(3) necessity to dose rodents with exceptionally high doses of soil lead to generate elevations in blood lead. Such high doses would fall into the saturation portion of the dose-response curve for other animals and probably for children, making accurate extrapolations of bioavailability difficult, if not impossible;

(4) delivery of soil lead to rodents in food *vs*. in a small amount of vehicle, due to practical matters of dosing by oral gavage. This prevents assessment of bioavailability in a partially fasted state and results in a highly variable dose (mg/kg-d body

weight) over the term of exposure due to high growth rates in rodents; and

(5) other confounders unique to rodents. Other animal models also have had their limitations in estimating quantitative bioavailability of lead in soil, and so the juvenile swine model used by EPA appears to be the most useful model.

Currently, the juvenile swine model (Weis *et al.,* 1994) design offers the strongest method to measure site-specific bioavailability, since it attempts to mimic childhood absorption and doses of lead in soil relative to soluble lead acetate. Critical to this or any future or alternative study is the need to test a representative soil lead sample which best reflects the geophysical and chemical nature of the lead in residential yards. Composite sampling of relatively homogeneous types of lead in surficial soil can produce an acceptable test sample. In the near future, promising *in vitro* models may be validated that correlates well with the *in vivo* swine model results. When approved by EPA, these validated models will have utility for screening soil and dust samples for relative bioavailability and can provide quantitative measures of bioaccessibility that can reasonably predict bioavailability of lead in soils with an acceptable amount of uncertainty.

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9200.4-27
EPA/540/F-98/030
PB98-963244

OSWER Directive # 9200.4-27P

MEMORANDUM

SUBJECT: Clarification to the 1994 Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities

FROM: Timothy Fields, Jr.
Acting Assistant Administrator

TO: Regional Administrators I-X

PURPOSE

This directive clarifies the existing 1994 Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities, OSWER Directive 9355.4-12. Specifically, this directive clarifies OSWER's policy on (1) using EPA's Science Advisory Board (SAB) reviewed Integrated Exposure Uptake Biokinetic Model (IEUBK) and blood lead studies, (2) determining the geographic area to use in evaluating human exposure to lead contamination ("exposure units"), (3) addressing multimedia lead contamination and (4) determining appropriate response actions at lead sites. The purpose for clarifying the existing 1994 directive is to promote national consistency in decision-making at CERCLA and RCRA lead sites across the country.

BACKGROUND

OSWER Directive 9355.4-12, issued on July 14, 1994 established OSWER's current approach to addressing lead in soil at CERCLA and RCRA sites. The existing directive established a streamlined approach for determining protective levels for lead in soil at CERCLA sites and RCRA facilities as follows:

- It recommends a 400 ppm screening level for lead in soil at residential properties;
- It describes how to develop site-specific preliminary remediation goals (PRGs) at CERCLA sites and media cleanup standards at RCRA Corrective Action facilities for residential land use; and,
- It describes a strategy for management of lead contamination at CERCLA sites and RCRA Corrective Action facilities that have multiple sources of lead.

The existing interim directive provides direction regarding risk assessment and risk management approaches for addressing soil lead contaminated sites. The OSWER directive states that, " ... implementation of this guidance is expected to provide more consistent decisions across the country ..." However, since that directive was released, OSWER determined that clarification of the guidance is needed. Key areas being clarified by issuance of this directive include: (1) using the IEUBK model and blood lead studies, (2) determining exposure units to be considered in evaluating risk and developing risk management strategies, (3) addressing multimedia lead contamination and (4) determining appropriate response actions at residential lead sites. The existing directive provides the following guidance on these areas:

1. The OSWER directive recommends using the Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children (Pub. # 9285.7-15-1, PB93-963510) for setting site-specific residential preliminary risk-based remediation goals (PRGs) at CERCLA sites and media cleanup standards (MCSs) at RCRA corrective actions Facilities. The directive states that the IEUBK model is the best tool currently available for predicting the potential blood lead levels of children exposed to lead in the environment. OSWER's directive also recommends the evaluation of blood lead data, where available, and states that well-conducted blood lead studies provide useful information to site managers. The directive however recommends that "... blood lead data not be used <u>alone</u> to assess risk from lead exposure or to develop soil lead cleanup levels."

2. The directive describes OSWER's risk reduction goal as "...generally, OSWER will attempt to limit exposure to soil lead levels such that a typical (or hypothetical) child or group of similarly exposed children would have an estimated risk of no more than 5% of exceeding a 10 ug/dl blood lead level." The directive also states that "... EPA recommends that a soil lead concentration be determined so that a typical child or group of children exposed to lead at this level would have an estimated risk of no more than 5% of exceeding a blood lead of 10 ug/dl." OSWER generally defines an exposure unit as a geographic area where exposures occur to the receptor of concern during the time of interest and believes that for a child or group of similarly exposed children, this is typically the individual residence and other areas where routine exposures are occurring.

3. The directive recommends that risk managers assess the contribution of multiple environmental sources of lead to overall lead exposure (e.g., consideration of the importance of soil lead levels relative to lead from drinking water, paint, and household dust) which promotes development of risk reduction strategies that address all sources that contribute significantly to exposure.

4. The OSWER directive states that the IEUBK model is not the only factor to be considered in establishing lead cleanup goals. Rather, the IEUBK model is the primary risk assessment tool available for evaluating lead risk and the results of the model are used to guide selection of appropriate risk management strategies for each site.

Since the OSWER directive was issued in 1994, there has been a trend toward a more consistent approach to managing risk at residential lead sites, however, OSWER was interested in identifying areas requiring additional clarification to facilitate more effective implementation of the directive. As a first step in the process, meetings were held with various EPA Regions, States and local governments to discuss how the directive has been implemented nationally at lead sites since 1994. By participating in these meetings and by reviewing the decisions that are being made across the country, OSWER believed that clarification of certain aspects of the 1994 directive would be useful.

All of the documents and guidance referenced in this directive are available through the National Technical Information Service (NTIS) at 703-605-6000 or could be downloaded electronically from: http//epa.gov/superfund/oerr/ini_prod/lead/prods.htm.

OBJECTIVE

At lead contaminated residential sites, OSWER seeks assurance that the health of the most susceptible population (children and women of child bearing age) is protected and promotes a program that proactively assesses and addresses risk. OSWER believes that predictive tools should be used to evaluate the risk of lead exposure, and that cleanup actions should be designed to address both current and potential future risk.

While health studies, surveys, and monitoring can be valuable in identifying current exposures and promoting improved public health, they are not definitive tools in evaluating potential risk from exposure to environmental contaminants. In the case of lead exposure, blood lead monitoring programs can be of critical importance in identifying individuals experiencing potential negative health outcomes and directing education and intervention resources to address those risks. However, CERCLA §121(b) requires EPA to select cleanup approaches that are protective of human health and the environment and that utilize permanent solutions to the maximum extent practicable. To comply with the requirements set forth in CERCLA § 121(b), OSWER will generally require selection of cleanup programs that are proactive in mitigating risk and that do not simply rely on biological monitoring programs to determine if an exposure has already occurred.

To meet these objectives, OSWER will seek actions that limit exposure to soil lead levels such that a typical child or group of similarly exposed children would have an estimated risk of no more than 5% of exceeding a 10 µg/dl blood lead level. If lead is predicted to pose a risk to the susceptible population, OSWER recommends that actions be taken to significantly minimize or eliminate this exposure to lead.

The principles laid out in the **four attached fact sheets** (Appendix) support OSWER's goals by encouraging appropriate assessment and response actions at CERCLA and RCRA lead sites across the country.

This clarification directive emphasizes the following key messages regarding the four areas and encourages the users of this directive, be they EPA Regions, States, or other stakeholders, to adopt these principles in assessing and managing CERCLA and RCRA lead sites across the country. The critical elements of the attached papers are as follows:

I. Using Blood Lead Studies and IEUBK Model at Lead Sites:

OSWER emphasizes the use of the IEUBK Model for estimating risks for childhood lead exposure from a number of sources, such as soils, dust, air, water, and other sources to predict blood lead levels in children 6 months to 84 (7 years) months old. The 1994 directive also recommended evaluation of available blood lead data and stated that data from a well-conducted blood lead study of children could provide useful information to site managers. In summary, OSWER's clarification policy on the appropriate use of the IEUBK and blood lead studies is that:

- OSWER recommends that the IEUBK model be used as the primary tool to generate risk-based soil cleanup levels at lead sites for current or future residential land use. If Regions propose an alternative method for generating cleanup levels, they are required to submit their approach to the national Lead Sites Consultation Group (LSCG)[1] for review and comment ;

- Response actions can be taken using IEUBK predictions alone; blood lead studies are not required; and

- Blood lead studies and surveys are useful tools at lead sites and can be used to identify key site-specific exposure pathways and to direct health professionals to individuals needing immediate assistance in minimizing lead exposure; however, OSWER recommends that blood lead studies not be used for establishing long-term remedial or non-time-critical removal cleanup levels at lead sites.

II. Determining Exposure and Remediation Units at Lead Sites

OSWER recommends that cleanup levels at lead sites be designed to reduce risk to a typical or individual child receiving exposures at the residence to meet Agency guidelines (*i.e.*, no greaterthan a 5% chance of exceeding a 10 ug/dl blood-lead level for a full-time child resident). Therefore, it is recommended that risk assessments conducted at lead-contaminated residential sites use the individual residence as the primary exposure unit of concern. This does not mean that a risk assessment should be conducted for every yard, rather that the soil lead contamination

[1]The Lead Sites Consultation Group (LSCG) is comprised of senior management representatives from the Waste Management Divisions in all 10 EPA regions along with senior representatives from the Office of Emergency and Remedial Response in EPA headquarters. The LSCG is supported by EPA's Technical Review Workgroup (TRW) for lead and the national Lead Sites Workgroup (LSW). The TRW consists of key scientific experts in lead risk assessment from various EPA Regions, labs and headquarters. The LSW is comprised of senior Regional Project Managers from various Regions and key representatives from headquarters who are experienced in addressing lead threats at Superfund sites.

data from yards and other residential media (for example, interior dust and drinking water) should be input into the IEUBK model to provide a preliminary remediation goal (PRG) for the residential setting. When applicable, potential exposure to accessible site-related lead sources outside the residential setting should also be evaluated to understand how these other potential exposures contribute to the overall risk to children, and to suggest appropriate cleanup measures for those areas.

III. Addressing Multimedia Contamination at Lead Sites

EPA generally has limited legal authority to use Superfund to address exposure from **interior lead-based paint**. As a policy matter, OSWER recommends that such exposures not be addressed through actual abatement activities. However, EPA Regions should promote addressing interior paint risks through actions by others (e.g., potentially responsible parties (PRPs), other government programs, etc.) as a component of an overall site management strategy. Because of other competing demands on the Superfund Trust Fund**,** OSWER recommends that EPA Regions avoid using the Superfund Trust Fund for removing **exterior lead-based paint** and soil contaminated from lead-based paint. Superfund dollars *may* however be used in limited circumstances to remediate exterior lead-based paint in order to protect the overall site remedy (i.e., to avoid re-contamination of soils that have been remediated) but generally only after determining that other funding sources are unavailable. As with interior lead-based paint abatement, EPA Regions should promote remediation of exterior lead-based paint by others, such as PRPs, local governments or individual homeowners.

IV. Determining Appropriate Response Actions at Lead Sites

In selecting site management strategies, it is OSWER's preference to seek early risk reduction with a combination of engineering controls (actions which permanently remove or treat contaminants, or create reliable barriers to mitigate the risk of exposure) and non-engineering response actions. All potential lead sources should be identified in site assessment activities. Non-engineering response actions, such as education and health intervention programs, should be considered an integral part of early risk reduction efforts because of their potential to provide immediate health benefits. In addition, engineering controls should be implemented early at sites presenting the greatest risk to children and other susceptible subpopulations.

As a given project progresses, OSWER's goal should be to reduce the reliance on education and intervention programs to mitigate risk. The goal should be cleanup strategies that move away from reliance on long-term changes in community behavior to be protective since behavioral changes may be difficult to maintain over time. The actual remedy selected at each CERCLA site must be determined by application of the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) (55 FR 8666- 8865, March 8, 1990) remedy selection criteria to site-specific circumstances. This approach also recognizes the NCP preference for permanent remedies and emphasizes selection of engineering over non-engineering remedies for long-term response actions.

This directive clarifies OSWER's policy on four key issue areas addressed in the 1994 OSWER soil lead directive in order to promote a nationally consistent decision-making process for assessing and managing risks associated with lead contaminated sites across the country. The policy presented in these specific issue areas supersedes all existing OSWER policy and directives on these subjects. No other aspects of the existing 1994 directive are affected.

IMPLEMENTATION

The principles laid out in this directive (which includes the four attached factsheets) are meant to apply to all residential lead sites currently being evaluated through the CERCLA Remedial Investigation/Feasibility Study process and all future CERCLA Sites and RCRA Corrective Action Facilities contaminated with lead. The Regions will be required to submit their rationale for deviating from the policies laid out in this directive to the Lead Sites Consultation Group. This directive does not apply to previous remedy selection decisions.

Attachments

cc: Waste Management Policy Managers (Regions I-X)
Stephen Luftig, OERR
Elizabeth Cotsworth, OSW
James Woolford, FFRRO
Barry Breen, OSRE
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NOTICE: This document provides guidance to EPA staff. The document does not, however, substitute for EPA's statutes or regulations, nor is it a regulation itself. Thus it cannot impose legally-binding requirements on EPA, states, or the regulated community, and may not apply to a particular situation based upon the circumstances. EPA may change this guidance in the future, as appropriate.

Appendix
OSWER Directive # 9200.4-27P
August 1998

Factsheet: Using the IEUBK Model and Blood Lead Studies at Residential Lead Sites

Question: What is OSWER's policy on using the IEUBK model and blood-lead studies in conducting risk assessments and setting cleanup standards at residential lead contamination sites?

Answer: OSWER's policy on using the IEUBK model and blood-lead studies in conducting risk assessment and setting cleanup standards is as follows:

A. Use of the IEUBK Model:

1. The IEUBK model is a good predictor of potential long-term blood-lead levels for children in residential settings. OSWER recommends that the IEUBK model be used as the primary tool to generate risk-based soil cleanup levels at lead sites for current or future residential land use. If Regions propose an alternative method for generating cleanup levels, they are required to submit their approach to the National Lead Sites Consultation Group (LSCG) for review and comment.

2. Blood-lead distributions predicted by the IEUBK model illustrate a plausible range of variability in children's physiology, behavior, and household conditions.

3. Response actions can be taken, and remedial goals developed, using IEUBK predictions alone.

B. Use of Blood-Lead Studies/Data:

1. Blood-lead studies, surveys, and monitoring are useful tools at lead sites and can be used to help identify key site-specific exposure pathways and direct health professionals to individuals needing immediate assistance in minimizing lead exposure.

2. The utility of blood lead testing results and studies depends on how representative the information is of the population being evaluated, the design of the data collection, and the quality of the laboratory analysis. To this end, OSWER recommends that EPA Regions consult with ATSDR or CDC to assess or design studies according to their intended use.

3. Many blood-lead screening, monitoring, or testing programs differ from blood lead studies in that they do not attempt to identify risk factors for childhood exposure to lead sources. Although these programs may be extremely beneficial in identifying children with elevated blood lead levels and identifying candidates for referral to medical professionals for evaluation, they may not provide an accurate representation of community-wide exposure.

4. Well-designed blood lead studies may be used to identify site specific factors and pathways to be considered in applying the IEUBK model at residential lead sites. However, OSWER recommends that blood-lead studies not be used to determine future long-term risk where exposure conditions are expected to change over time; rather, they should be considered a snapshot of ongoing exposure under a specific set of circumstances (including community awareness and education) at a specific time. Long-term studies may be helpful in understanding exposure trends within a community and evaluating the effectiveness of cleanup strategies over time.

C. <u>IEUBK and Blood-Lead Studies/Data:</u>

1. Blood-lead data and IEUBK model predictions are expected to show a general concordance for most sites. However, some deviations between measured and predicted levels are expected. On some occasions, declines in blood-lead levels have been observed in association with lead exposure-reduction and health education. However, long-term cleanup goals should be protective in the absence of changes in community behavior as there is little evidence of the sustained effectiveness of these education/intervention programs over long periods of time.

2. Where actual blood-lead data varies significantly from IEUBK Model predictions, the model parameters should not automatically be changed. In such a case, the issue should be raised to the Lead Technical Review Workgroup (TRW) to further identify the source of those differences. Site work need not be put on hold while the issue is being reviewed by the TRW; the site manager should review other elements of the lead directive and the "Removal Actions at Lead Sites" guidance to determine appropriate interim actions to be taken at the site.

The Regions will be required to submit their rationale for deviating from the policies laid out in this factsheet to the Lead Sites Consultation Group.

Factsheet: Determining Exposure and Remediation Units at Residential Lead Sites

Question: How does OSWER define an exposure unit, and subsequently apply this definition in conducting risk assessment and risk management activities at residential lead sites?

Answer: OSWER recognizes that defining and characterizing exposure unit(s) for a site is critically important in undertaking risk assessment activities and in designing protective cleanup strategies. An **exposure unit** is defined as a geographic area where exposures occur to the receptor of concern during the time of interest and that for a child, or group of similarly exposed children, this is typically the individual residence and other areas where chronic or ongoing exposures are occurring.

Various approaches to characterizing and managing risks by exposure units have been examined by OSWER. OSWER recognizes that lead ingestion can also cause adverse health effects in adults and fetuses but believes that by adequately limiting lead exposures to young children at residential sites, these other receptors will generally be likewise protected from adverse health impacts.

EPA's goal is to protect human health and the environment under current and future exposure scenarios. At lead sites, OSWER wants to assure that children's health is protected and promotes a program that proactively assesses risks rather than relying on biological monitoring to determine if an exposure has already occurred. OSWER emphasizes actions be taken at lead sites that will minimize or eliminate exposure of children to environmental lead contamination.

To achieve the above stated goal, OSWER recommends characterizing **exposure units as exposure potential at the individual residence as the primary unit of concern for evaluating potential risk at lead contaminated residential sites**. This recognizes that there are children whose domain and activities occur principally within the confines of a particular residential property. For determining exposure potential (and ultimately developing protective cleanup levels) at the individual home, OSWER recommends the scenario to be evaluated (through use of the IEUBK Model) would be a young child in full-time residence. This approach helps achieve OSWER's recommended health protection goal that an individual child or group of similarly exposed children would have <5% chance of exceeding a blood-lead concentration of 10 ug/dl. In designing community wide cleanup strategies, it is essential that non-residential areas (e.g., parks, day care facilities, playgrounds, etc.), where lead exposure may occur, also be characterized with respect to their contribution to soil-lead exposure, and appropriate cleanup actions implemented.

OSWER recommends that risk management decisions for response to residential lead contamination sites focus on reducing risk at residences, but also recommends that response strategies be developed for other site locations (exposure units) where children receive exposure. Flexibility in determining appropriate response actions that provide protection at the individual residence should be considered in context of the NCP remedy selection criteria. The lead exposure issues are complex and OSWER recommends that EPA Regions try to communicate clearly the risk characterization and risk management decisions to the site residents. Affected communities must clearly understand the context of risk management decisions, how these decisions affect the health of their children, and how cleanup actions will influence the future growth and development of the community.

The Regions will be required to submit their rationale for deviating from the policies laid out in this factsheet to the Lead Sites Consultation Group.

Factsheet: Addressing Multimedia Contamination at Residential Lead Sites

Question: What is OSWER's policy on addressing multimedia contamination at residential lead sites?

Answer: OSWER recognizes that several sources of lead-contamination, including soil, ground water, airborne particulates, lead plumbing, interior dust, and interior and exterior lead-based paint may be present at Superfund sites where children are at risk or have documented lead exposure. These lead sources may contribute to elevated blood-lead levels and may need to be evaluated in determining risks and cleanup actions at residential lead sites. However, there are limitations on the Agency's statutory authority under CERCLA to abate some of these sources, such as indoor lead-based paint and lead plumbing because CERCLA responses may be taken only to releases or threatened releases into the environment (CERCLA §104 (a)(3) and (4)).

When EPA's resources, or authority to respond or to expend monies under Superfund is limited, OSWER recommends that EPA Regions identify and coordinate to the greatest extent possible with other authorities and funding sources (e.g., other federal agencies and state or local programs). EPA Regions should coordinate with these other authorities to design a comprehensive, cost-effective response strategy that addresses as many sources of lead as practicable. These strategies should include actions to respond to lead-based paint, interior dust, and lead plumbing, as well as ground water sources and lead-contaminated soil.

Although OSWER will encourage that EPA Regions fully cooperate in the development of a comprehensive site management strategy, OSWER realizes that complete active cleanup of these other sources may be difficult to complete due to limited funding available to other authorities. Since complete cleanups of these sources is not guaranteed, and at most sites may be unlikely, OSWER recommends that the soil cleanup levels not be compromised. In other words, the soil cleanup levels should be calculated with the IEUBK model using existing pre-response action site specific data. This is due to the fact that soil cleanup levels at residential lead sites are generally established to protect individuals, from excess exposures to soils, and house dust attributable to those soils, and are not attributable to exposure to other sources such as interior lead paint which should be managed on a residence specific basis. Remediation of non-soil lead sources to mitigate overall lead exposure at individual residences should therefore not be used to modify sitewide soil lead cleanup levels.

The recommendations provided below represent OSWER's policy on addressing lead-contaminated media and/or sources for which EPA has limited or no authority to remediate.

Interior Paint: EPA has limited legal authority to use Superfund to address exposure from interior lead-based paint. As a policy matter, OSWER recommends that such exposures not be addressed through actual abatement activities. However, EPA Regions should promote addressing interior paint risks through actions by others, such as HUD, local governments, or individual home owners as a component of an overall site management strategy. Any activities to clean up interior lead-based paint by PRPs or other parties should not result in an increase of the risk-based soil cleanup levels.

Exterior Paint: Because of other competing demands on the Superfund Trust Fund, OSWER recommends that EPA Regions avoid using the Superfund Trust Fund for removing exterior lead-based paint and soil contaminated from lead-based paint. Superfund dollars *may* be used to respond to exterior lead-based paint for protecting the overall site remedy (i.e., to prevent re-contamination of soils that have been remediated) but only after determining that other funding sources are unavailable. Where other sources of funding are not available, EPA may utilize the CERCLA monies to remediate exterior lead-based paint on homes/buildings, around which soil contaminated by other sources has been cleaned up to prevent recontamination of the soil. The Superfund should not be used to remediate exterior lead-based paint where no soil cleanup has occurred. As with interior lead-based paint abatement, EPA Regions should promote remediation of exterior lead-based paint by others, such as PRPs, local governments or individual homeowners. Cleanup activities of exterior paint conducted by PRPs or other parties should not result in an increase of the risk-based soil cleanup levels.

Interior Dust: Lead contaminated interior dust can be derived from several sources, including interior paint, home owner hobbies, exterior soil, and other exterior sources. In many cases, it may be difficult to differentiate the source(s) for the lead contamination in the dust. In general, EPA Regions should refrain from using the Superfund Trust Fund to remediate interior dust. Because of the multi-source aspects of interior dust contamination, potential for recontamination, and the need for a continuing effort to manage interior dust exposure, OSWER recommends the use of an aggressive health education program to address interior dust exposure. Such programs, administered through the local health department (or other local agency), should be implemented in conjunction with actions to control the dust source. At a minimum, the program should include blood-lead monitoring, and personal hygiene and good housekeeping education for the residents. OSWER believes that EPA Regions can also support the program by providing HEPA vacuums to the health agency for use in thoroughly cleaning home interiors.

Lead Plumbing: Generally CERCLA does not provide for legal authority to respond to risks posed by lead plumbing within residential dwellings. It should be noted that the water purveyor is responsible for providing clean water to the residences. As with interior dust, OSWER recommends that EPA Regions coordinate with local agencies to establish a health education program to inform residents of the hazards associated with lead plumbing and how to protect themselves by regularly flushing, or preferably, replacing lead pipes. Soil cleanup levels should not be adjusted to account for possible remediation of lead plumbing.

Factsheet: Determining Appropriate Response Actions at Residential Lead Sites

Question: What is OSWER's position on the appropriate use of engineering and non-engineering response actions in developing risk management strategies for lead sites?

Answer: One goal emphasized in the recent third round of Superfund Reforms is for EPA to take a consistent approach in selecting and implementing both long- and short-term response actions at lead sites in all regions. One obstacle to achieving this consistency has been differing degrees of reliance on non-engineering response actions in reducing risk.

Site management strategies at lead sites typically include a range of response actions. Alternatives range from engineering controls that permanently remove or treat the contaminant source to non-engineering response actions, such as educational programs and land use restrictions. This continuum represents the range of response options available to risk managers. This position paper clarifies the relationship between engineering and non-engineering response actions in developing site management strategies.

In selecting site management strategies, OSWER's policy will be to seek early risk reduction with a combination of engineering controls (actions which permanently remove or treat contaminants, or which create reliable barriers to mitigate the risk of exposure) and non-engineering response actions. All potential lead sources should be identified in site assessment activities. Non-engineering response actions, such as education and health intervention programs, should be considered an integral part of early risk reduction efforts due to their potential to provide immediate health benefits.[2] In addition, engineering controls should be implemented early at sites presenting the greatest risk to children and other susceptible subpopulations. Community concerns should receive a high priority in site decision-making; local support is vital to the success of health intervention and education programs.

As the project progresses, OSWER's goal should be to reduce reliance on education and intervention programs to mitigate risk. The goal should be cleanup strategies that move away from reliance on long-term changes in community behavior to be protective; behavioral changes

[2]The actual effectiveness of health intervention and educational programs in reducing risk continues to be a subject of discussion. Anecdotal information suggests that such programs can provide short-term benefits in some populations. Rigorous statistical studies demonstrating the benefits of educational programs in preventing lead exposure are lacking. It is generally recognized that not all segments of the population will be influenced by such programs, and that long-term benefits are less certain. Local support for such programs is critical. The active (and long-term) participation of local and state public health agencies is needed in implementing institutional controls, including health intervention and education programs; without local implementation of such programs their success is uncertain. Additional research on the effectiveness of these programs is critical to consideration of their use in future cleanups.

may be difficult to maintain over time. The actual remedy selected at each site must be determined by application of the NCP remedy selection criteria to site-specific circumstances. However, this approach recognizes the NCP preference for permanent remedies and emphasizes the use of engineering controls for long-term response actions. This approach also recognizes that well-designed health intervention and education programs, when combined with deed restrictions and/or other institutional controls, may be appropriate for reducing future exposure potential and may supplement engineering controls.

In instances where Regions believe that the use of engineering controls is impracticable, and education, health intervention, or institutional controls are proposed as the sole remedy, Regions will be required to consult with the LSCG.

UNITED STATES
ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

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OFFICE OF
SOLID WASTE AND EMERGENCY RESPONSE

OSWER Directive # 9355.4-12

MEMORANDUM

SUBJECT: Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities

FROM: Elliott P. Laws
Assistant Administrator

TO: Regional Administrators I-X

PURPOSE

As part of the Superfund Administrative Improvements Initiative, this interim directive establishes a streamlined approach for determining protective levels for lead in soil at CERCLA sites and RCRA facilities that are subject to corrective action under RCRA section 3004 (u) or 3008 (h) as follows:

- It recommends screening levels for lead in soil for residential land use 400 (ppm);[1]
- It describes how to develop site-specific preliminary remediation goals (PRGs) at CERCLA sites and media cleanup standards (MCSs) at RCRA Corrective Action facilities for residential land use; and,
- It describes a plan for soil lead cleanup at CERCLA sites and RCRA Corrective Action Facilities that have multiple sources of lead.

This interim directive replaces all previous directives on soil lead cleanup for CERCLA and RCRA programs (see the Background section, 1989-1991).

KEY MESSAGES

Screening levels are not cleanup goals. Rather, these screening levels may be used as a tool to determine which sites or portions of sites do not require further study and to encourage voluntary cleanup. Screening levels are defined as a level of contamination above which there may be enough concern to warrant site-specific study of risks. Levels of contamination above the screening level would NOT automatically require a removal action, nor designate a site as "contaminated."

The residential screening level for lead described in this directive has been calculated with the Agency's new Integrated Exposure Uptake Biokinetic Model (IEUBK) model (Pub. # 9285.7-15-2, PB93-963511), using default parameters. As outlined in the Guidance Manual for the IEUBK Model for Lead in Children (Pub. #

[1]The residential screening level is the same concept as the action level proposed in the RCRA Corrective Action Subpart S rule (July 27, 1990, 55 *Federal Register* 30798).

9285.7-15-1, PB93-963510, February 1994), this model was developed to: recognize the multimedia nature of lead exposure; incorporate important absorption and pharmacokinetic information; and allow the risk manager to consider the potential distributions of exposure and risk likely to occur at a site (the model goes beyond providing a single point estimate output). For these reasons, this approach is judged to be superior to the more common method for assessing risks of non-cancer health effects which utilizes the reference dose (RfD) methodology. Both the Guidance Manual and the model are available to Superfund staff through the Superfund Document Center (703-603-8917) and to the public through the National Technical Information Service (703-487-4650).

Residential preliminary remediation goals (PRGs) for CERCLA remediations and media cleanup standards (MCSs) for RCRA corrective actions can be developed using the IEUBK model on a site-specific basis, where site data support modification of model default parameters. At some Superfund sites, using the IEUBK model with site-specific soil and dust characteristics, PRGs of more than twice the screening level have been identified. However, it is important to note that the model alone does not determine the cleanup levels required at a site. After considering other factors such as costs of remedial options, reliability of institutional controls, technical feasibility, and/or community acceptance, still higher cleanup levels may be selected.

The implementation of this guidance is expected to provide for more consistent decisions across the country and improve the use of site-specific information for RCRA and CERCLA sites contaminated with lead. The implementation of this guidance will aid in determining when evaluation with the IEUBK model is appropriate in assessing the likelihood that environmental lead poses a threat to the public. Use of the IEUBK model in the context of this guidance will allow risk managers to assess the contribution of different environmental sources of lead to overall blood lead levels (e.g., consideration of the importance of soil lead levels relative to lead from drinking water, paint and household dust). It offers a flexible approach to considering risk reduction options (referred to as the "bubble" concept) that allows for remediation of lead sources that contribute significantly to elevated blood lead. This guidance encourages the risk manager to select, on a site-specific basis, the most appropriate combination of remedial measures needed to address site-specific lead exposure threats. These remedial measures may range widely from intervention to abatement. However, RCRA and CERCLA have very limited authority to address interior exposures from interior paint. For detailed discussion of the decision logic for addressing lead-contaminated sites, see the Implementation section and Appendix A.

Relationship to lead paint guidance. In addition, this interim directive clarifies the relationship between guidance on Superfund and RCRA Corrective Action cleanups, and EPA's guidance on lead-based paint hazards (discussed further in Appendix C). The paint hazard guidance will be issued to provide information until the Agency issues regulations identifying lead-based paint hazards as directed by Section 403 of the Toxic Substances Control Act (TSCA)[2]. Lead-based paint hazards are those lead levels and conditions of paint, and residential soil and dust that would result in adverse health effects.

The two guidance documents have different purposes and are intended to serve very different audiences. As a result the approaches taken differ to some degree. The lead-based paint hazard guidance is intended for use by any person who may be involved in addressing residential lead exposures (from paint, dust or soil). It thus relates to a potentially huge number of sites, and serves a very broad potential audience, including private property owners or residents in addition to federal or state regulators. Much residential lead abatement may take place outside any governmental program, and may not involve extensive site-specific study.

This OSWER guidance, on the other hand, deals with a much smaller number of sites, being addressed under close federal regulatory scrutiny, at which extensive site characterization will have been performed before cleanup decisions are made. Thus, the RCRA and CERCLA programs will often have the benefit of much site-specific exposure information. This guidance is intended for use by the relatively small number of agency officials who oversee and direct these cleanups.

Both the TSCA Section 403 and OSWER programs use a flexible, tiered approach. The OSWER guidance sets a residential screening level at 400 ppm. As noted above, this is not intended to be a "cleanup level" for

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[2]Title IV of TSCA (including section 403) was added by the Residential Lead-Based Paint Hazard Reduction Act of 1992 (Title X of the Housing and Community Development Act of 1992).

CERCLA and RCRA facilities, but only to serve as an indicator that further study is appropriate. The Section 403 guidance indicates that physical exposure-reduction activities may be appropriate at 400 ppm, depending upon site-specific conditions such as use patterns, populations at risk and other factors. Although worded somewhat differently, the guidances are intended to be similar in effect. For neither guidance is 400 ppm to automatically be considered a "cleanup level"; instead, it indicates a need for considering further action, but not necessarily for taking action. Neither is meant to indicate that cleanup is necessarily appropriate at 400 ppm. The greater emphasis in this OSWER guidance on determining the scope of further study reflects the fact that both CERCLA and RCRA cleanups proceed in stages with detailed site characterization preceding response actions in every case.

Above the 400 ppm level, the Section 403 guidance identifies ranges over which various types of responses are appropriate, commensurate with the level of potential risk reduction, and cost incurred to achieve such risk reduction. For example, in the range of 400 to 5000 ppm, limited interim controls are recommended depending, as noted above, on conditions at the site, while above 5000 ppm, soil abatement is recommended. This OSWER guidance does not include comparable numbers above 400 ppm; instead, as discussed above, it recommends the site-specific use of the IEUBK model to set PRGs and MCSs, when necessary. The remedy selection process specified in the National Contingency Plan (NCP) should then be used to decide what type of action is appropriate to achieve those goals.

In general, because the Section 403 guidance was developed for a different purpose and audience, OSWER does not recommend that it be used as a reference in setting PRGs and MCSs or in determining whether action at a particular site is warranted. (To put it another way, it generally should not be treated as a "to be considered" document or "TBC" under CERCLA.) The section 403 guidance is meant to provide generic levels that can be used at thousands of widely varying sites across the nation. The detailed study that goes on at CERCLA or RCRA sites will allow levels to be developed that are more narrowly tailored to the individual site. Nothing in the section 403 guidance discourages setting more site-specific levels for certain situations; in fact, it specifically identifies factors such as bioavailability that may significantly affect the evaluation of risk at some sites.

The IEUBK model. The Agency is further studying both the IEUBK model and analyses of epidemiologic studies in order to better develop the technical basis for rulemaking under TSCA Section 403. The Agency intends to promulgate regulations under Section 403 setting health-based standards for lead in soil and dust. OSWER intends to issue a final soil lead directive once the TSCA Section 403 regulations are finalized. For additional information on TSCA Section 403 developments, call (202) 260-1866.

However, the Agency believes that risk managers (risk assessors, on-scene coordinators, remedial project managers, and other decision-makers at Superfund and RCRA sites) are currently in need of the best guidance available today. The Agency believes that the IEUBK model is the best available tool currently available for assessing blood lead levels in children. Furthermore, use of the IEUBK provides allows the risk manager to consider site-specific information that can be very important in evaluating remediation options. Therefore, using the latest developments in the IEUBK model and the collective experience of the Superfund, RCRA Corrective Action, and TSCA Section 403 programs, the Agency is offering this guidance and is recommending a residential screening level for Superfund and RCRA sites of 400 ppm.

BACKGROUND

Early OSWER guidance (1989-1991). Four guidance documents on the soil lead cleanup were issued by OSWER during the period of 1989 to 1991:

1. September 1989, OSWER Directive #9355.4-02. This guidance recommended a soil lead cleanup level of 500 - 1000 ppm for protection of human health at residential CERCLA sites.

2. May 9, 1990. RCRA Corrective Action program guidance on soil lead cleanup. This guidance described three alternative methods for setting "cleanup levels" (not action levels) for lead in soil at RCRA facilities. One approach was to use levels derived from preliminary results of IEUBK model runs. The other two approaches were to use the range of 500 to 100 provided in the 1989 directive on CERCLA sites, or to use "background" levels at the facility in question.

3. June 1990, OSWER Directive #9355.4-02A. Supplement to Interim Guidance on Establishing Soil Land Cleanup Levels at Superfund Sites. This memorandum reiterated that the September 1989 directive was guidance and should not be interpreted as regulation.

4. August 29, 1991. This supplemental guidance discussed EPA's efforts to develop a new directive that would accomplish two objectives: (1) account for the contribution from multiple media to total lead exposure; and, (2) provide a stronger scientific basis for determining a soil lead cleanup level at a specific site.

Development of the IEUBK Model for OSWER use. During the 1989-91 time period, use of EPA IEUBK model was identified as the best available approach for accomplishing the objectives outlined in the August 1991 guidance. The model integrates exposure from lead in air, water, soil, dust, diet, and paint with pharmacokinetic modeling to predict blood lead levels in children (i.e., Children 6 to 84 months old), a particularly sensitive population.

In the spring of 1991, OSWER organized the Lead Technical Review Workgroup to assist Regional risk assessors and site managers in both using the model and making data collection decisions at CERCLA and RCRA sites. The workgroup was composed of scientists and risk assessors from the Regions and Headquarters, including the Office of Research and Development (ORD), and the Office of Pollution Prevention and Toxic Substances (OPPTS).

In November 1991, the EPA Science Advisory Board (SAB) reviewed the scientific merits of using the IEUBK model for assessing total lead exposure and developing soil lead cleanup levels at CERCLA and RCRA sites. In general, the SAB found the model to be an important advance in assessing potential health risks from environmental contaminants. However, the SAB also recommended additional guidance on the proper use of the model.

In response to SAB concern over the potential for incorrect use of the model and selection of inappropriate input values both for default and site-specific applications, OSWER developed a comprehensive "Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children" (referred to in this interim directive as the "Guidance Manual"). This Guidance Manual assists the user in providing inputs to the model to estimate risks from exposures to lead. It discusses the use of model default values or alternative values, and the application of the model to characterize site risks. Use of the Guidance Manual should facilitate consistent use of the IEUBK model and allow the risk assessor to obtain valid and reliable predictions of lead exposure. The Lead Technical Review Workgroup has been collecting data to further validate the model and to update the Guidance Manual as needed.

Relationship to RCRA Corrective Action "Action" Levels. The approach for calculating a screening level for lead (including exposure assumptions), set forth in this Revised Interim Soil Lead Directive, supersedes the guidance provided for calculating "action" levels set forth in Appendix D of the proposed Subpart S Corrective Action rule. In the July 27, 1990 RCRA proposal (55 *Federal Register* 30798), EPA introduced the concept of "action levels" as trigger levels for further study and subsequent remediation at RCRA facilities. In this respect, the current directive's "screening levels" are analogous to the proposed rule's "action levels." In the proposal, where data were available, action levels were developed for three pathways of human exposure to contaminants: soil ingestion, water ingestion and inhalation of contaminated air. Exposure assumptions used in the calculations were set out in Appendix D of the proposal. For the soil pathway, action levels were calculated two different ways depending on whether the contaminant in the soil was a carcinogen or systemic toxicant. Although lead was listed in Appendix A of the preamble to the rule as a class B2 carcinogen, no action level had been calculated because neither a carcinogenic slope factor (SF) nor a reference dose (RfD) had been developed by the Agency. Although the guidance in Appendix D of the proposed Corrective Action rule remains in effect with respect to other hazardous constituents, this directive now allows for the development of the lead screening ("action") level using the IEUBK model.

Recent developments (1992-Present). Following discussions among senior Regional and OSWER management, the OSWER Soil Lead Directive Workgroup (composed of Headquarters, Regional and other Federal agency representatives) recommended in the spring of 1992 that a "two step" decision framework be developed for establishing cleanup levels at sites with lead-contaminated soils. This framework would identify a single level of

lead in soils that could be used as either the PRG for CERCLA site cleanups or the action level for RCRA Corrective Action sites, but would allow site managers to establish site-specific cleanup levels (where appropriate) based on site-specific circumstances. The IEUBK model would be an integral part of this framework. OSWER then developed a draft of this directive which it circulated for review on June 4, 1992. The draft set 500 ppm as a PRG and an action level for RCRA facilities in residential settings.

Following development of this draft, OSWER held a meeting on July 31, 1992 to solicit a broad range of views and expertise. A wide range of interests, including environmental groups, citizens and representatives from the lead industry attended. This meeting encouraged OSWER to think more broadly about how the directive would affect urban areas, how lead paint and dust contribute to overall risk, and blood lead data could be used to assess risk. In subsequent meetings with the Agency for Toxic Substances and Disease Registry (ATSDR) and Centers for Disease Control (CDC), options were discussed on how to use blood lead data and the need to evaluate the contribution of paint. In addition, during these meetings, a "decision tree" approach was suggested that proposed different threshold levels (primary and secondary) for screening decisions, action decisions and land use patterns.

Findings from the three cities (Baltimore, Boston, and Cincinnati) of the Urban Soil Lead Abatement Demonstration Project (peer review scheduled for completion in late 1994) indicate that dust and paint are major contributors to elevated blood lead levels in children. Furthermore, preliminary findings suggest that any strategy to reduce overall lead risk at a site needs to consider not only soil, but these other sources and their potential exposure pathways. (For further information on this demonstration project, contact Dr. Rob Elias, USEPA/ORD, Environmental Criteria And Assessment Office (ECAO), RTP, (919) 541-4167.)

Finally, in its efforts to develop this interim directive, the OSWER Soil Lead Workgroup has met with other EPA workgroups including the TSCA Section 403, Large Area Land Sites, and Urban Lead workgroups, as well as other Federal agencies including the Agency for Toxic Substances and Disease Registry, the Centers for Disease Control, and Department of Housing and Urban Development.

Derivation of Lead Screening Levels. Development of the residential screening level in this interim directive required two important OSWER decisions. 1) OSWER determined that it would seek to achieve a specific level of protectiveness in site cleanups; generally, OSWER will attempt to limit exposure to soil lead levels such that a typical (or hypothetical) child or group of similarly exposed children would have an estimated risk of no more than 5% exceeding the 10 μg lead/dl blood lead level. This 10 μg/dl blood lead level is based upon analyses conducted by the Centers for Disease Control and EPA that associate blood lead levels of 10 μg/dl and higher with health effects in children; however, this blood lead level is below a level that would trigger medical intervention. 2) In developing the residential screening level, OSWER has decided to apply the EPA's IEUBK model on a site-specific basis. This model has been designed specifically to evaluate exposures for children in a residential setting. Current research indicates that young children are particularly sensitive to the effects of lead and require specific attention in the development of a soil screening level for lead. A screening level that is protective for young children is expected to be protective for older population subgroups.

In general, the model generates a probability distribution of blood lead levels for a typical child, or group of children, exposed to a particular soil lead concentration and concurrent lead exposures from other sources. The spread of the distribution reflects the observed variability of blood lead levels in several communities. This variability arises from several sources including behavioral and cultural factors.

The identification of lead exposures from other sources (due to air, water, diet, paint, etc.) is an essential part of characterizing the appropriate blood lead distribution for a specific neighborhood or site. For the purpose of deriving a residential screening level, the background lead exposure inputs to the IEUBK model were determined using national averages, where suitable, or typical values. Thus, the estimated screening level of 400 ppm is associated with an expected "typical" response to these exposures, and should not be taken to indicate that a certain level of risk (e.g., exactly 5% of children exceeding 10 μg/dl blood) will be observed in specific community, e.g., in a blood lead survey.

Because a child's exposure to lead involves a complex array of variables, because there is population sampling variability, and because there is variability in environmental lead measurements and background levels of

lead in food and drinking water, results from the model may differ from results of blood lead screening of children in a community. Extensive field validation is in progress. The model will be evaluated further once these efforts are completed.

OBJECTIVE

With this interim directive, OSWER recommends using 400 ppm soil lead (based on application of the IEUBK model) as a screening level for lead in soil for residential scenarios at CERCLA sites and at RCRA Corrective Action sites. Residential areas with soil lead below 400 ppm generally require no further action. However, in some special situations, further study is warranted below the screening level. For example, agricultural areas, wetlands, areas with ecological risk, and areas of higher than expected human exposure are all situations that could require further study. For further guidance on ecological risks, Superfund risk managers are encouraged to consult their Regional Biological Technical Assistance Groups (BTAGs; see Appendix D).

Generally, the ground water pathway will not pose a significant risk since many lead compounds are generally not highly mobile. However, there are situations where, because of the form of lead, hydrogeology, or the presence of other contaminants at the site, lead may pose a threat to the ground water. In these situations, additional analysis is warranted, Superfund Regional Toxics Integration Coordinators (RTICs; seeAppendix B) or RCRA hydrogeologists should be consulted.

While recognizing that urban lead is a significant problem, this interim directive is not designed to be applied in addressing the potential threat of lead in urban areas other than at CERCLA or RCRA Corrective Action sites. Guidance and regulations to be developed under TSCA Section 403 will provide an appropriate tool for addressing urban sites of potential concern.

Generally, where the screening level is exceeded, OSWER recommends using the IEUBK model during the Remedial Investigation or the RCRA Facility Investigation for evaluating potential risks to humans from environmental exposures to lead under residential scenarios. Site-specific data need to be collected to determine PRGs or MCSs. At a minimum, this may involve collecting soil and dust samples in appropriate areas of the site. Further guidance on data collection or modification of the non-residential equation can be obtained by contacting the RTICs or RCRA Regional risk assesors, who in turn may consult the Lead Technical Review Workgroup.

The type of site-specific data that should be collected will obviously depend on a number of factors, including the proximity of residences to the contaminated soil, the presence of site access controls, and other factors that would influence the probability of actual human exposure to the soils. At a minimum, when residences are at or near the site, it is expected that using the model will generally involve taking soil and dust samples from appropriate areas of the site. In many cases, it may not be necessary to gather certain types of data for input into the model. For example, when there are no residences nearby, or where there is otherwise no exposure or very limited exposure to lead contamination, it may not be necessary to collect site-specific data (e.g., dust, water, paint, blood-lead, etc.)

In developing a PRG for CERCLA sites or a MCS for RCRA facilities, EPA recommends that a soil lead concentration be determined so that a typical child or group of children exposed to lead at this level would have an estimated risk of no more than 5% of exceeding a blood lead of 10 μg/dl. In applying the IEUBK model for this purpose, appropriate site-specific data on model input parameters, including background exposures to lead, would be identified.

When the PRG or MCS is exceeded, remedial action is generally recommended. Such action does not, however, necessarily involve excavating soil. A range of possible actions may be considered, as discussed in greater detail under the Implementation section of this directive: Issues for Both Programs.

IMPLEMENTATION
Superfund

This interim directive applies to all future CERCLA Remedial Investigation/Feasibility Study (RI/FS) work; this interim directive should generally not be applied at sites for which risk assessments have been completed. For

removal sites, this interim directive recommends that decisions regarding removal actions be considered first by the Regional Decision Team (RDT). The RDT will then refer sites to the removal program for early action, as appropriate.

The approach in this interim directive helps meet the goals set by the Superfund Accelerated Cleanup Model (SACM) for streamlining remedial decision-making. (This streamlined approach is described in Appendix A, Suggested Decision Logic for CERCLA and RCRA Corrective Action.) This interim directive also recognizes that other methods (e.g., slope studies and others) for evaluating risks at lead sites may also be appropriate and may be used in lieu of, or in conjunction with, the IEUBK model. If an alternate approach to lead risk assessment is to be applied, an EPA scientific review should be obtained. For example, expert statisticians would need to review slope factor calculations for statistical biases before their use could be supported. Recognizing that all assessment methods involve some uncertainties, the Agency, at this time, believes the IEUBK model is the most appropriate and widely applicable tool for Superfund and RCRA sites. Alternatively, EPA may require setting cleanup levels below the screening level if site-specific circumstances warrant (e.g., ecological risk). For further information on the use of the IEUBK model at CERCLA sites, contact the Regional Toxics Integration Coordinators identified in Appendix B.

RCRA Corrective Action

It is expected that the RCRA corrective action program will generally follow an approach similar to CERCLA's (as described above) in using the IEUBK model. In the case of RCRA facilities at which lead contaminated soils are of concern, collection and evaluation of data for the purpose of using the model will be primarily the responsibility of the owner/operator.

Issues for Both Programs

Cleanup of soils vs. other lead sources: OSWER's approach to assessing and managing risks from lead is intended to address the multi-media/multi-source nature of environmental lead exposures because it is expected that people at or near CERCLA and RCRA Corrective Action sites will experience lead exposures from sources in addition to contaminated soil. In some instances, these other exposures may be large (e.g., where there are children living in houses with high levels of lead dust form deteriorated paint). The presence of various sources of lead exposure may be very important in both the development of site-specific risk assessments and in the consideration of alternative risk management options.

From an assessment perspective, estimating blood lead levels, that might result from exposures at a site, depends on appropriately integrating exposures from all relevant media. Specifically, it is important to consider direct soil exposures and indoor dust exposures (which can include contributions from both soil and lead-based paint) on a site-specific basis, as well as any contributions from drinking water or other local sources of lead exposure. In using the IEUBK model to estimate blood lead levels, it is important to note that the risk attributable to soil lead exposures is dependent upon the existing level of exposures from other sources. That is, the amount by which the total risk would be lowered if all exposures to lead in soil were removed is not a constant, but varies with the level of existing non-soil exposures. This is because the model derives "distribution" (rather than a simple point estimate) as an output whose shape and size is quite dependent on the predicted variability of exposures from each lead source. As a result, other factors being equal, the risks attributable to soil will generally be higher in the presence of elevated lead exposures from other sources. Therefore, in applying the IEUBK model, the risk attributable to soil lead can be predicted as the difference between the risk estimated when all sources of lead exposure are assessed, and the risk estimated considering only non-soil related exposures. This concept is especially important when evaluating different options for risk reduction at a given site.

From a risk management perspective, achieving a safe environment for populations at CERCLA and RCRA Corrective Action sites may require attention to multiple sources of lead, not all of which may be related to contamination from the source that was the initial concern at the site. Generally, the goal of the Agency, while acting within the constraints of CERCLA and RCRA legal authorities, is to reduce, to the maximum extent feasible, the risk of having significantly elevated blood lead levels. On a site-specific basis this can include remediation approaches that would lead to reduction of exposure from other sources, such as lead-based paint, in conjunction with appropriate soil remediation. Following from the risk assessment discussion in the previous

paragraphs, exposures from lead in soils may have a lesser impact in producing high blood lead level if existing exposures from lead in soils may have a lesser impact in producing high blood lead levels if existing exposures from lead-based paint are reduced.

Abatement vs. Intervention: Remedial measures can be divided into those that remove the source of contamination (abatement) and those that leave the contamination in place but block the exposure pathway (intervention). These combinations of measures might include but not be limited to:

Abatement - Soil removal or interior and exterior lead paint abatement.

Intervention - Institutional controls, education/public outreach, gardening restrictions, indoor cleaning and dust removal, or additional cover.

Generally, the most appropriate CERCLA or RCRA response action or combination of actions will be based, in part, on the estimated level of threat posed at a given site. However, as mentioned earlier, key decision criteria also include the overall protectiveness of response options, attainment of Applicable or Relevant and Appropriate Requirements (for CERCLA), a preference for permanent remedies, implementability, cost-effectiveness, and public acceptance. Intervention measures may be more appropriate than abatement (e.g., soil excavation) at many sites, especially in areas where soil lead levels fall at or near the site-specific PRG or MCS.

Addressing exposure from other sources of lead may reduce risk to a greater extent and yet be less expensive than directly remediating soil. In some cases, cleaning up the soil to low levels may, by itself, provide limited risk reduction because other significant lead sources are present (e.g., contaminated drinking water or lead-based paint in residential housing). If it is possible to address the other sources, the most cost-effective approach may be to remediate the other sources as well as, or (if exposures to lead in soil are relatively low) instead of full soil lead abatement.

Lead-based paint can be a significant source of lead exposure and needs to be considered when determining the most appropriate response action. Interior paint can contribute to elevated indoor dust lead levels. In addition, exterior paint can be a significant source of recontamination of soil. Appendix A-3 of this document contains more information on how to evaluate and address the contribution of paint.

Certain legal considerations arise in considering remediation of sources other than soil. In particular, interior exposures from interior paint generally are not within the jurisdiction of RCRA or CERCLA. In addition, where other sources are addressed, issues may arise regarding the recoverability of costs expended by the Agency, or the possibility of claims being asserted against the Fund where other parties are ordered to do the work.

As discussed above, in considering whether to address sources other than soil, it is necessary to consider the risk that would remain from the lead in the soil. In some cases, after risks from other sources have been addressed, unrestricted exposure to soil could be allowed while still being protective (e.g., where the IEUBK model result was heavily affected by the other sources). In other cases, soil risks may still be high enough to require abatement, containment or institutional controls to prevent high levels of exposure. In such cases, before a conclusion is made that the overall remedy will be protective, institutional controls should be carefully studied to make sure that they will be implementable, effective in both the long-term and short-term, and likely to achieve community acceptance.

A potentially useful approach that can be considered in conjunction with other, more active measures in reducing blood lead levels is to develop and promote public education and awareness programs that focus on the causes and prevention of lead poisoning in children. EPA's Office of Pollution Prevention and Toxics (OPPT) provides information on abatement of lead-based paint by the homeowner as well as inexpensive preventive measures the public can take to reduce their exposure to lead. Additional research to evaluate the effectiveness of educational efforts in reducing lead exposures are needed to allow better evaluation of the usefulness of this option. Further, OPPT is assessing the effectiveness of various lead paint abatement options emphasizing low-cost methods. For additional information, contact the National Lead Information Center at 1-800-424-LEAD.

Mining-related sites: Both risk assessors and site managers should be aware that there are a number of factors that affect the relationship between soil lead concentrations and blood lead levels. These factors include the

variability in soil lead contribution to house dust levels, or differences in the bioavailability of lead. See discussion in next section, Use of blood lead data, for assessing differences between measured and predicted blood lead levels.

Thus, for mining-related sites without significant past smelting/mill activity, this interim directive encourages further research for characterizing the potential impact of particle size and speciation on soil bioavailability.

Site managers and risk assessors are cautioned that most areas impacted by mining activities are also associated with present or historical smelting or milling operations. Generalizations regarding distinct differences between mining and smelting or milling sites should be avoided until adequate site history and characterization are complete.

Use of blood lead data: In conducting Remedial Investigations (RIs) for CERCLA or RCRA Facility Investigations (RFIs) for RCRA Corrective Action, the interim directive recommends evaluating available blood lead data. In some cases, it may be appropriate to collect new or additional blood lead samples. In general, data from well-conducted blood lead studies of children on or near a site can provide useful information to both the risk assessor and site manager. However, the design and conduct of such studies, as well as the interpretation of results, are often difficult because of confounding factors such as a small population sample size. Therefore, any available blood lead data should be carefully evaluated by EPA Regional risk assessors to determine their usefulness. The Guidance Manual discusses how to evaluate observed blood lead survey data and blood lead data predicted by the IEUBK model.

The Guidance Manual recommends that blood lead data not be used alone either to assess risk from lead exposure or to develop soil lead cleanup levels. During its review of the IEUBK model, the SAB supported this position by asserting that site residents may temporarily modify their behavior (e.g., wash their children's hands more frequently) whenever public attention is drawn to a site. In such cases, this behavior could mask the true magnitude of potential risk at a site and lead to only temporary reductions in the blood lead levels of children. Thus, blood lead levels below 10 μg/dl are not necessarily evidence that a potential for significant lead exposure does not exist, or that such potential could not occur in the future.

Non-residential (adult) screening level. EPA also believes there is a strong need to develop a non-residential (adult) screening level. The IEUBK model is, however, not appropriate for calculating this screening level since it is designed specifically for evaluating lead exposures in children. At this time, EPA is considering a few options for developing this screening level. Several adult models have recently become available. Developing a screening level by using any of them is likely to require significant additional work by the Agency. This work might include testing, validation, and selection of one of the existing models or development of its own model, both of which would require a considerable amount of time. Consequently this would probably be a long-term option. A short-term option would be to develop a screening level based on a simple approach that approximates the more complicated biokinetics in humans. This can serve in the interim while more sophisticated adult lead exposure assessment tools can be identified or developed.

NOTICE: Users of this directive should bear in mind that the recommendations in this document are intended solely as guidance, and that EPA risk managers may act at variance with any of these recommendations where site-specific conditions warrant, as has been noted above. These recommendations are not intended, and cannot be relied upon, to create any rights, substantive or procedural, enforceable by any party in litigation with the United States, and may change at any time without public notice.

Because this document and the related Guidance Manual are not legally binding either upon EPA or other parties, Agency personnel should keep in mind if they are questioned or challenged in comments on a proposed remedial plan, such comments must be considered and a substantive explanation must be provided for whatever approach is ultimately selected. For example, while the IEUBK model is recommended here, its use is not a regulatory requirement and comments on the model or its use should be fully considered.

APPENDICES

A Suggested Decision Logic for CERCLA and RCRA Corrective Action

A-1 Suggested Decision Logic for Residential Scenarios for CERCLA and RCRA Corrective Action

A-2 Suggested Decision Logic for Lead-based Paint for CERCLA and RCRA Corrective Action

B Regional Toxics Integration Coordinators (RTICS)

C Relationship between the OSWER Soil Lead Directive and TSCA Section 403 Guidance

D Biological Technical Assistance Group Coordinators (BTAGS)

Appendix A-1

Suggested Decision Logic for Residential Scenarios for CERCLA and RCRA Corrective Action

Step 1: Determine soil lead concentration at the site.

If soil lead is less than 400 ppm:
STOP, no further action is required, UNLESS special circumstances (such as the presence of wetlands, other areas of ecological risk, agricultural areas, shallow aquifers, or other areas of potentially high exposure) warrant further study.

If soil lead is greater than 400 ppm:
PROCEED to Step 2, UNLESS 400 ppm is selected as a cleanup goal based on consideration of all relevant risk management factors.

Step 2: Evaluate probable land use and develop exposure scenarios.

Step 3: Collect appropriate site-specific data based on selected scenarios.

For example, sampling data may include:

- Soil and dust (at a minimum), paint, water, and air,
- For unique site situations, data on speciation and particle size, and behavioral activities may be required.

Available blood lead data:

- If blood lead data are available, consult the Guidance Manual and Regional Risk Assessor.
- If blood lead data are not available, Regional Risk Assessors and site managers should consider the appropriateness of consulting a blood lead study to supplement available data.

Step 4: Run the IEUBK model with site-specific data to estimate risk and evaluate key exposure pathways at the site.

- If blood lead data are available, compare the data to the model results.

Step 5: Where risks are significant, evaluate remedial options.

If lead-based exterior or interior paint is the only major contributor to exposure, no Superfund action or RCRA corrective action is warranted.

If soil is the only major contributor to elevated blood lead, a response to soil contamination is warranted, but paint abatement is not.

If both exterior lead-based paint and soil are major contributors to exposure, consider remediating both sources, using alternative options as described in Appendix A-2.

If indoor dust levels are greater than soil levels, consider evaluating the contribution of interior lead-based paint to the dust levels. If interior lead-based paint is a major contributor, consider remediating indoor paint to achieve a greater overall risk reduction at a lower cost. (See Appendix A-2.)

NOTE: Available authority to remediate lead-based paint under CERCLA and RCRA is extremely limited.)

Step 6: If the IEUBK model predicts elevated blood leads, rerun the model using the site-specific parameters selected to reflect remedial options in Step 5 to determine site-specific PRGs or MCSs for soil.

Appendix A-2

Suggested Decision Logic for Lead-based Paint for CERCLA and RCRA Corrective Action

(If soil lead levels are below screening levels, lead-based paint could be addressed by authorities other than RCRA or CERCLA.)

If soil lead levels are above screening levels:

Step 1: Examine condition of exterior paint and determine its lead content, if any.

- If the paint is deteriorated, assess contribution or potential contribution of paint to elevated soil lead levels through speciation studies, structural equation modeling, or other statistical methods.

Step 2: Evaluate potential for recontamination of soil by exterior paint.

Step 3: Remediate exterior paint only in conjunction with soil.

- Determine appropriate remediation based on risk management factors (e.g., applying the nine criteria), remediating the major contributor first.

Step 4: Examine condition of indoor paint and determine its lead content, if any.

- If indoor dust lead concentration is greater than outdoor soil lead concentration (because of contamination from both interior paint and outdoor soil), remediate indoor dust (e.g., through a removal action, or making HEPA-VACS available to community).

Step 5: Once the risk from indoor paint has been assessed, examine options to abate indoor paint (e.g., PRP, State, local, HUD) and consult TSCA Section 403 program for additional information and/or guidance.

Step 6: While RCRA and CERCLA have very limited authority regarding the cleanup of interior paint, the remedy may take into account the reduction of total risk that may occur if interior paint is addressed by other means. Thus, for example, a Record of Decision (ROD) or Statement of Basis (SB) may recognize that interior lead-based paint is being addressed by other means, and narrow the response accordingly (possibly making this contingent on completion of the interior lead-based paint abatement effort).

Appendix B

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Appendix C

Relationship between the OSWER Soil Lead Directive and TSCA Section 403 Guidance

Since lead exposures occur through all media, a variety of Agency programs address lead under a number of statutes. Lead in soil is addressed under TSCA Section 403, the RCRA Corrective Action program, and CERCLA, each of which differs somewhat in the types of sites that apply and the types of standards that are used. These differences are primarily due to differences in the purposes of the programs and the authority granted by the statutes under which they are developed. Section 403 soil standards will apply only to residential soil and the current TSCA guidance is generic in nature, with the same standards applying on a nationwide basis. Given the wide applicability of Section 403, generic standards are used in the current guidance in order to reduce resource requirements, as compared to site-specific decisions which can involve expensive and time-consuming analyses. Required RCRA and CERCLA activities are determined on a site-specific basis. The agency's recommendations for evaluating RCRA Corrective Action and CERCLA sites are contained in the OSWER Interim Soil Lead Directive.

In all three of these programs, the Agency's approach is to consider soil lead in the context of other lead sources that may be present and contribute to the total risk. For example, TSCA Section 403 specifically requires the Agency to consider the hazards posed by lead-based paint and lead-contaminated interior dust, as well as lead-contaminated soil. Likewise, the OSWER Soil Directive includes evaluation of other lead sources at a site as part of site assessment / investigation procedures. In addition, the primary focus of the three programs is primary prevention -- the prevention of future exposures from the source(s) being remediated.

The fundamental difference between the relatively new TSCA Section 403 program and the RCRA Corrective Action and CERCLA cleanup programs is that, under current guidance the Section 403 program seeks to establish national standards to prioritize responses to lead hazards whereas the other two programs usually develop site-specific cleanup requirements. This is because TSCA Section 403 deals with a potentially huge number of sites, and resources for the investigation needed to accurately identify their risks are typically very limited. Therefore most decisions under Section 403 will be made with little or no regulatory oversight and clear generic guidelines will be more effective. The more established RCRA and CERCLA programs, on the other hand, deal with a much smaller number of sites, at which extensive site characterization will have been performed before cleanup decisions are made. In addition, these programs have well-established funding mechanisms.

Appendix D

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APPENDIX P

Baseline Intakes for the EPA Default and Box Model Scenarios by Area and Age Level

Appendix P

Baseline Intakes for the EPA Default and Box Model Scenarios by Area and Age Level

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The tables and figures in this appendix show lead intake rates for children aged 1 to 4 years, 5 to 6 years, and 0 to 1 years (also 6 to 7 years). These are based on the default values provided in the IEUBK Model for total soil and dust intake. The rates provided for air, water, and market basket intakes are EPA default values that are used for all age groups.

Table P-1a Baseline Intakes for Child Ingestion of Soils (Ages 1-4) - Default Scenario

One to Four Year Olds

Area	Community Average Soil Conc. (mg/kg)	Soil Intake (µg/day)	Community Average Dust Conc. (mg/kg)	Dust Intake (µg/day)	Air Intake (µg/day)	Water Intake (µg/day)	Market Basket Intake (µg/day)	Total Intake (µg/day)
Mullan	628	38	985	73	0.5	2	6	120
Burke/Nine Mile	548	33	879	65	0.5	2	6	107
Wallace	771	47	1004	75	0.5	2	6	130
Silverton	352	21	557	41	0.5	2	6	71
Osburn	418	25	493	37	0.5	2	6	71
Side Gulches	373	23	695	52	0.5	2	6	83
Kingston	257	16	466	35	0.5	2	6	59
Lower Basin	110	7	301	22	0.5	2	6	38

Table P-1b Baseline Intakes for Child Ingestion of Soils (Ages 1-4) - 40:30:30 Scenario

One to Four Year Olds

Area	Community Average Soil Conc. (mg/kg)	Soil Intake (µg/day)	Community Average Dust Conc. (mg/kg)	Dust Intake (µg/day)	Air Intake (µg/day)	Water Intake (µg/day)	Market Basket Intake (µg/day)	Total Intake (µg/day)
Mullan	628	51	985	53	0.5	2	6	113
Burke/Nine Mile	548	44	879	47	0.5	2	6	101
Wallace	771	62	1004	54	0.5	2	6	125
Silverton	352	29	557	30	0.5	2	6	67
Osburn	418	34	493	27	0.5	2	6	69
Side Gulches	373	30	695	38	0.5	2	6	76
Kingston	257	21	466	25	0.5	2	6	55
Lower Basin	110	9	301	16	0.5	2	6	34

Figure P-1a Summary of Children's Recreational Potential Incremental Lead Intakes
Ages 1-4 Years (CT)

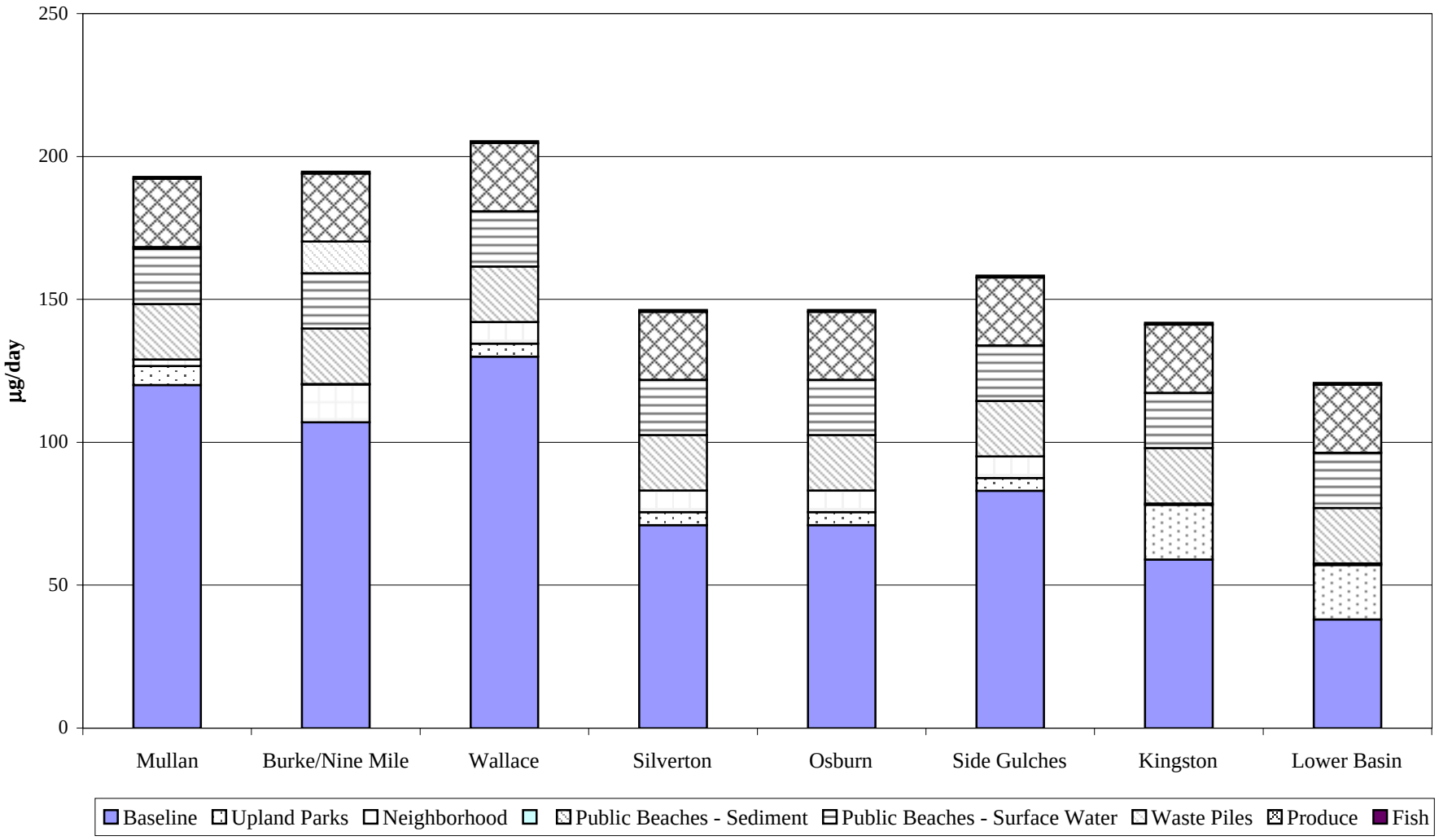


Figure P-1b Summary of Children's Recreational Potential Incremental Lead Intakes
Ages 1-4 yrs. (RME)

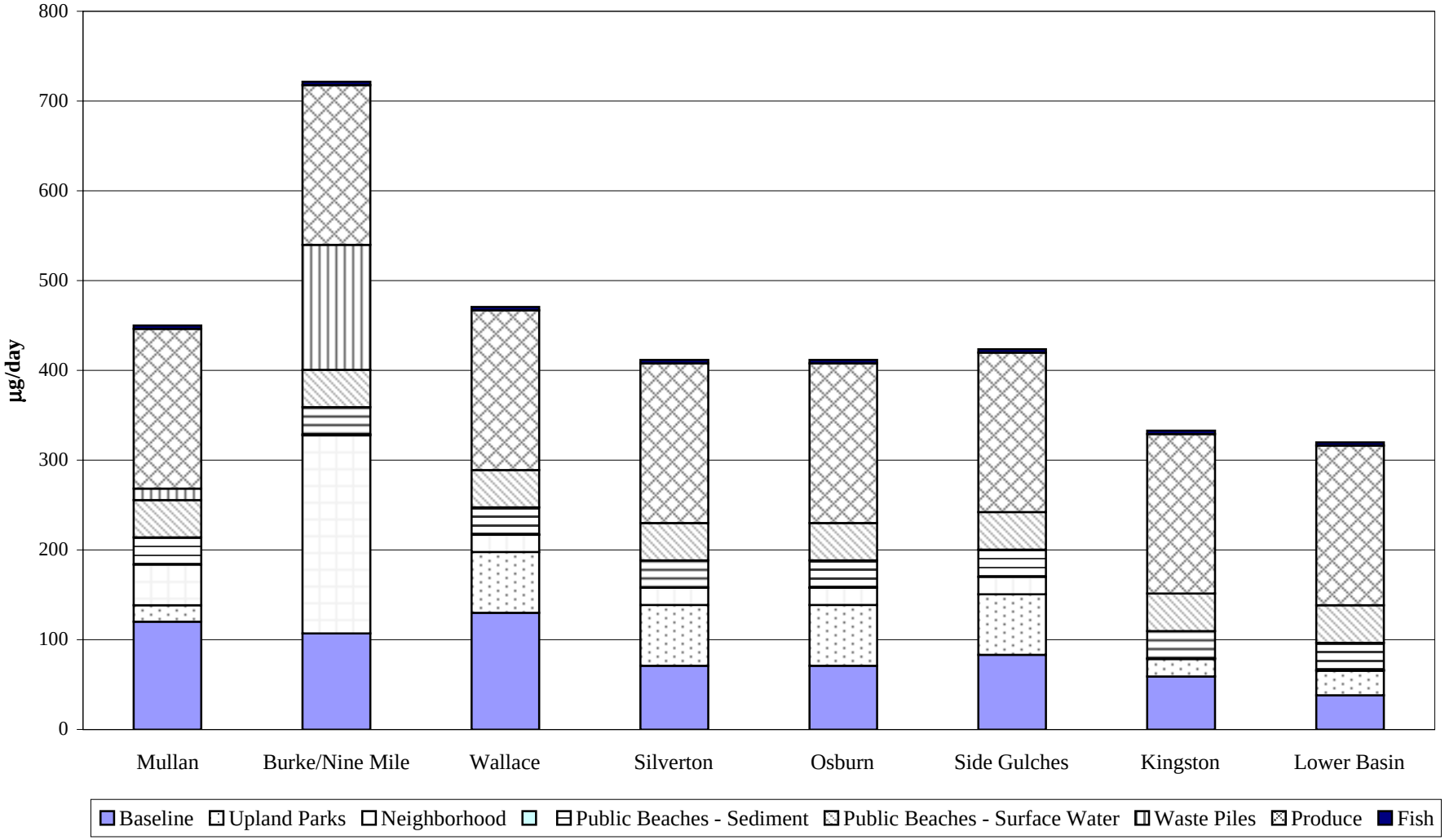


Table P-2a Baseline Intakes for Child Ingestion of Soils (Ages 5-6) - Default Scenario

Five to Six Year Olds

Area	Community Average Soil Conc. (mg/kg)	Soil Intake (µg/day)	Community Average Dust Conc. (mg/kg)	Dust Intake (µg/day)	Air Intake (µg/day)	Water Intake (µg/day)	Market Basket Intake (µg/day)	Total Intake (µg/day)
Mullan	628	25	985	49	0.5	2	6	83
Burke/Nine Mile	548	22	879	44	0.5	2	6	74
Wallace	771	31	1004	50	0.5	2	6	90
Silverton	352	14	557	28	0.5	2	6	51
Osburn	418	17	493	24	0.5	2	6	50
Side Gulches	373	15	695	34	0.5	2	6	58
Kingston	257	10	466	23	0.5	2	6	42
Lower Basin	110	4	301	15	0.5	2	6	28

Table P-2b Baseline Intakes for Child Ingestion of Soils (Ages 5-6) - 40:30:30 Scenario

Five to Six Year Olds

Area	Community Average Soil Conc. (mg/kg)	Soil Intake (µg/day)	Community Average Dust Conc. (mg/kg)	Dust Intake (µg/day)	Air Intake (µg/day)	Water Intake (µg/day)	Market Basket Intake (µg/day)	Total Intake (µg/day)
Mullan	628	34	985	35	0.5	2	6	78
Burke/Nine Mile	548	30	879	32	0.5	2	6	70
Wallace	771	42	1004	36	0.5	2	6	87
Silverton	352	19	557	20	0.5	2	6	48
Osburn	418	23	493	18	0.5	2	6	49
Side Gulches	373	20	695	25	0.5	2	6	54
Kingston	257	14	466	17	0.5	2	6	39
Lower Basin	110	6	301	11	0.5	2	6	25

Figure P-2a Summary of Children's Recreational Potential Incremental Lead Intakes for Ages 5-6 (CT)

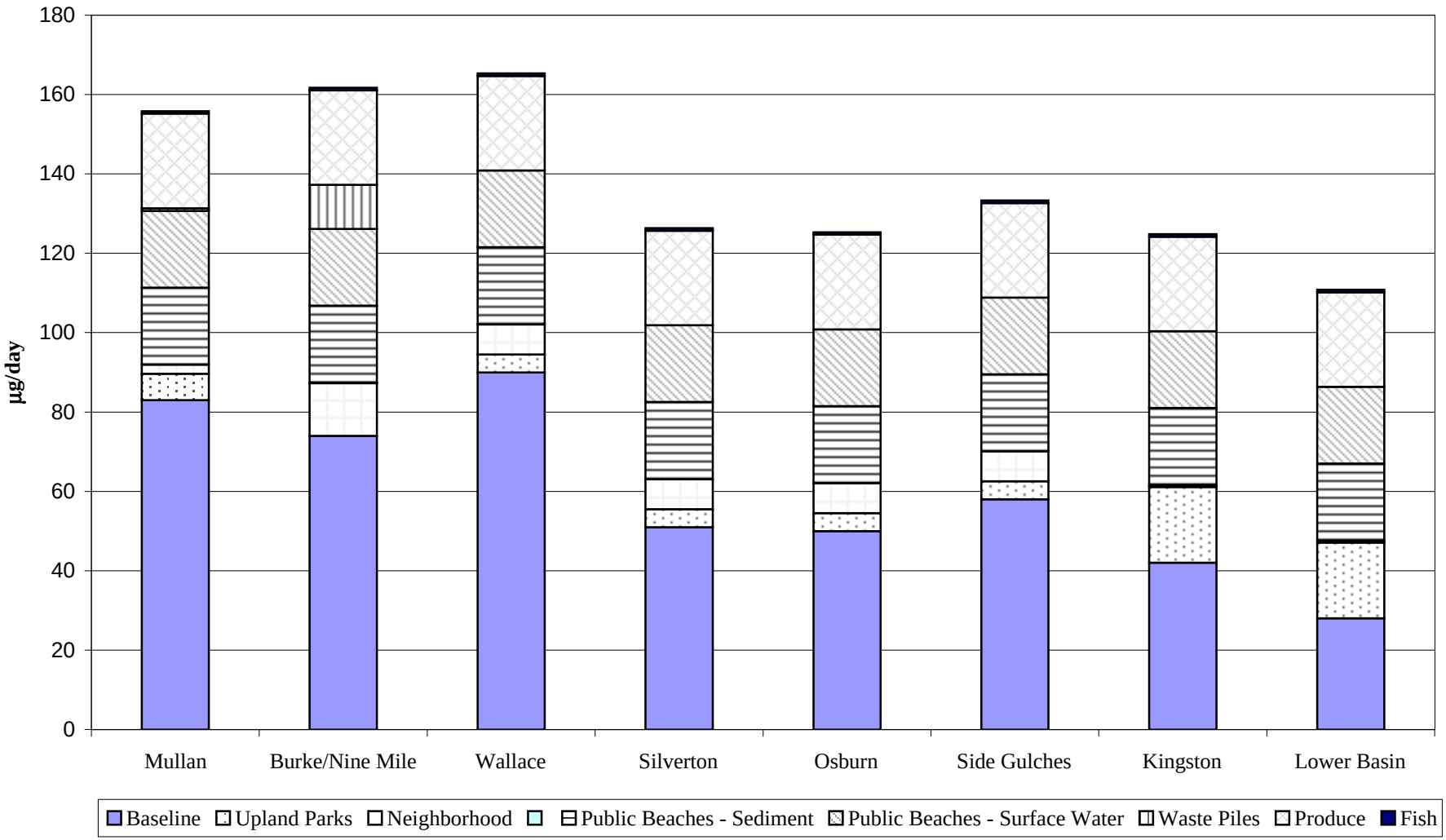


Figure P-2b Summary of Children's Recreational Potential Incremental Lead Intakes for Ages 5-6 (RME)

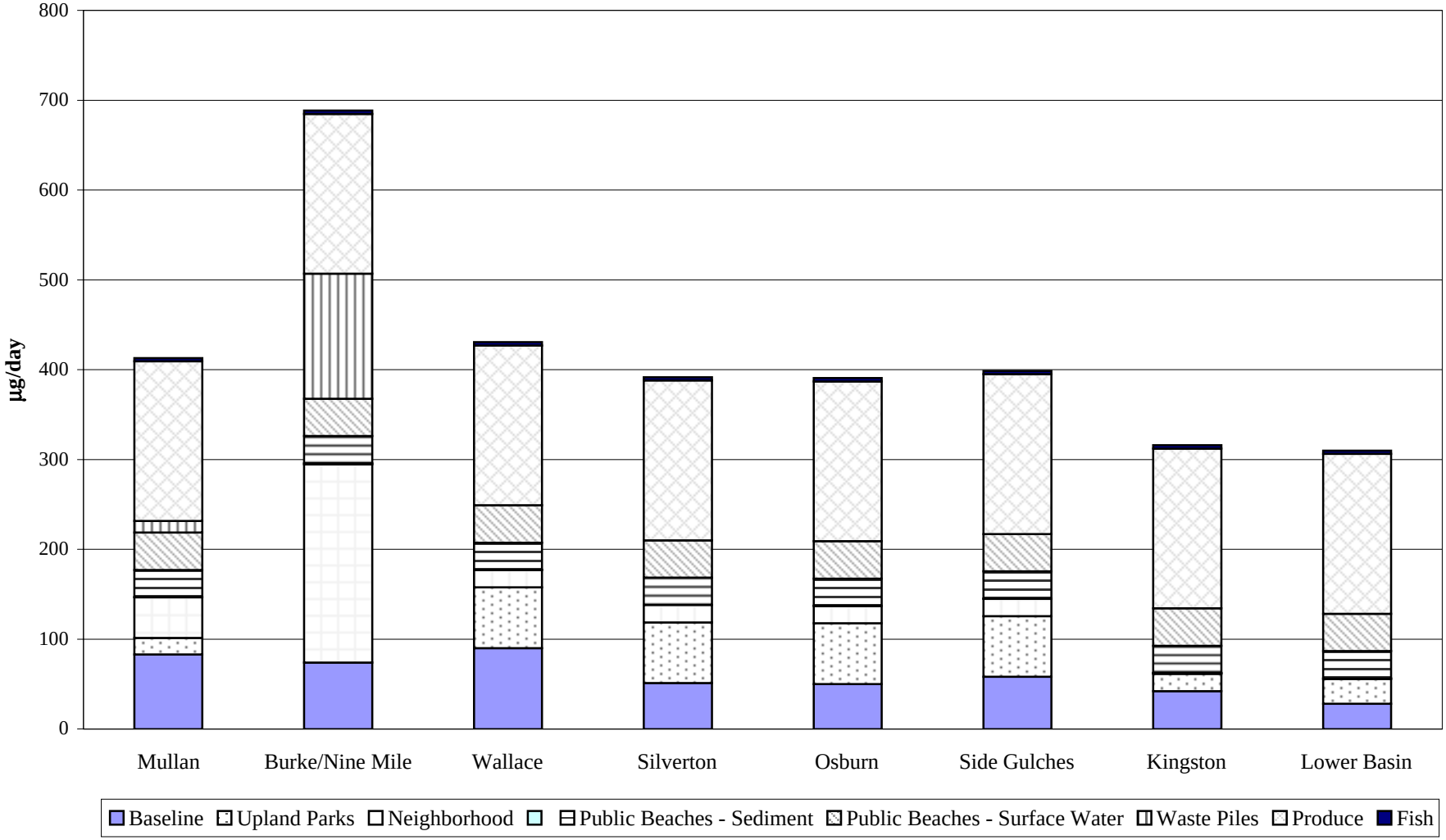


Table P-3a Baseline Intakes for Child Ingestion of Soils (Ages 0-1 and Ages 6-7) - Default Scenario

Zero to One Year Olds and Six to Seven Year Olds

Area	Community Average Soil Conc. (mg/kg)	Soil Intake (µg/day)	Community Average Dust Conc. (mg/kg)	Dust Intake µg/day)	Air Intake (µg/d)	Water Intake (µg/day)	Market Basket Intake (µg/day)	Total Intake (µg/day)
Mullan	628	24	985	46	0.5	2	6	79
Burke/Nine Mile	548	21	879	41	0.5	2	6	71
Wallace	771	30	1004	47	0.5	2	6	85
Silverton	352	13	557	26	0.5	2	6	48
Osburn	418	16	493	23	0.5	2	6	48
Side Gulches	373	14	695	32	0.5	2	6	55
Kingston	257	10	466	22	0.5	2	6	40
Lower Basin	110	4	301	14	0.5	2	6	27

* Note: The ingestion rate for 0-1 year olds and 6-7 year olds is the same, 0.085 gms/day.

Table P-3b Baseline Intakes for Child Ingestion of Soils (Ages 0-1 and Ages 6-7) - 40:30:30 Scenario

Zero to One Year Olds and Six to Seven Year Olds

Area	Community Average Soil Conc. (mg/kg)	Soil Intake (µg/day)	Community Average Dust Conc. (mg/kg)	Dust Intake µg/day)	Air Intake (µg/d)	Water Intake (µg/day)	Market Basket Intake (µg/day)	Total Intake (µg/day)
Mullan	628	32	985	33	0.5	2	6	74
Burke/Nine Mile	548	28	879	30	0.5	2	6	67
Wallace	771	39	1004	34	0.5	2	6	82
Silverton	352	18	557	19	0.5	2	6	46
Osburn	418	21	493	17	0.5	2	6	47
Side Gulches	373	19	695	24	0.5	2	6	51
Kingston	257	13	466	16	0.5	2	6	38
Lower Basin	110	6	301	10	0.5	2	6	25

* Note: The ingestion rate for 0-1 year olds and 6-7 year olds is the same, 0.085 gms/day.

Figure P-3a Summary of Children's Recreational Potential Incremental Lead Intakes
Ages 0-1 and 6-7 (CT)

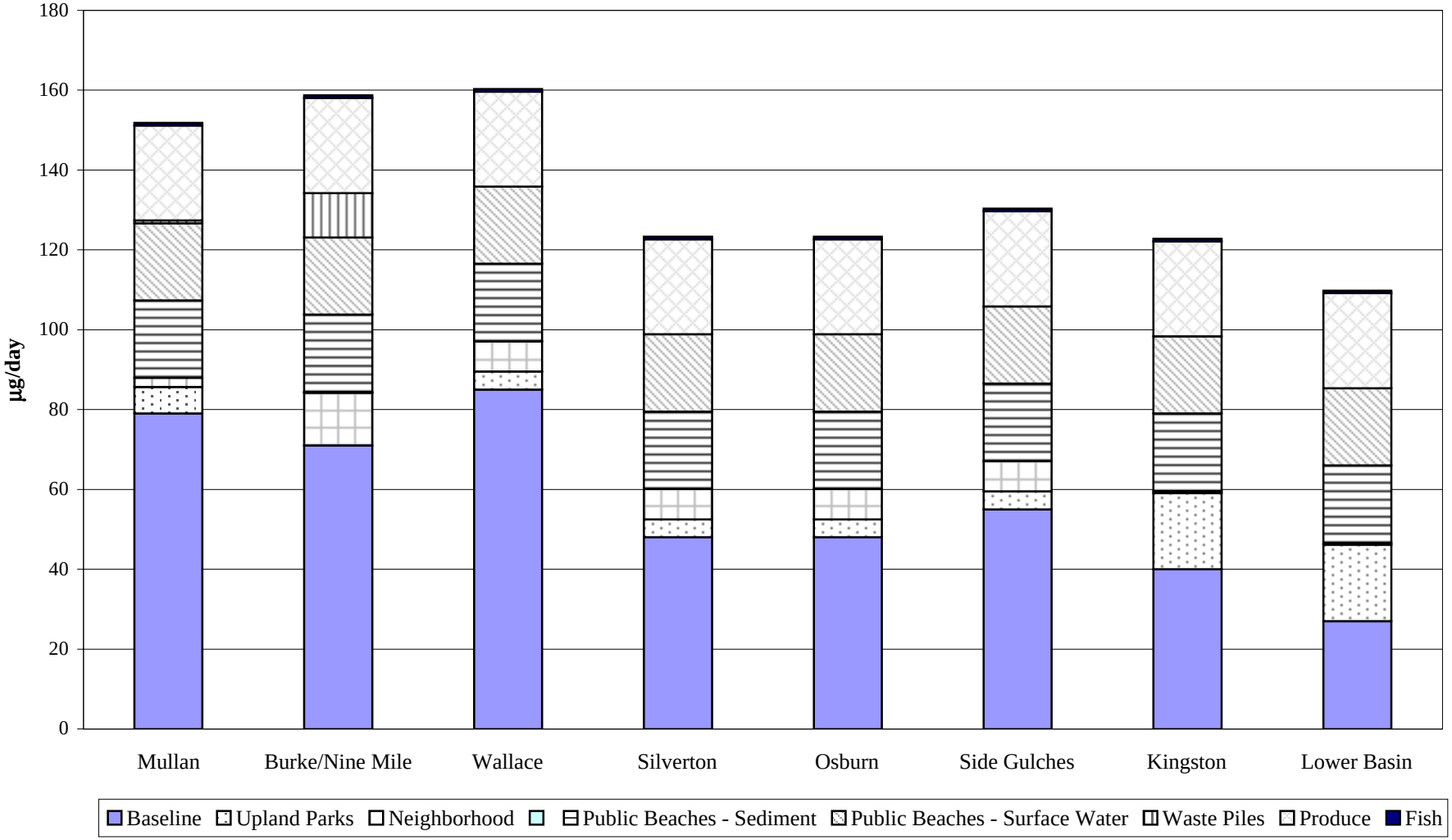
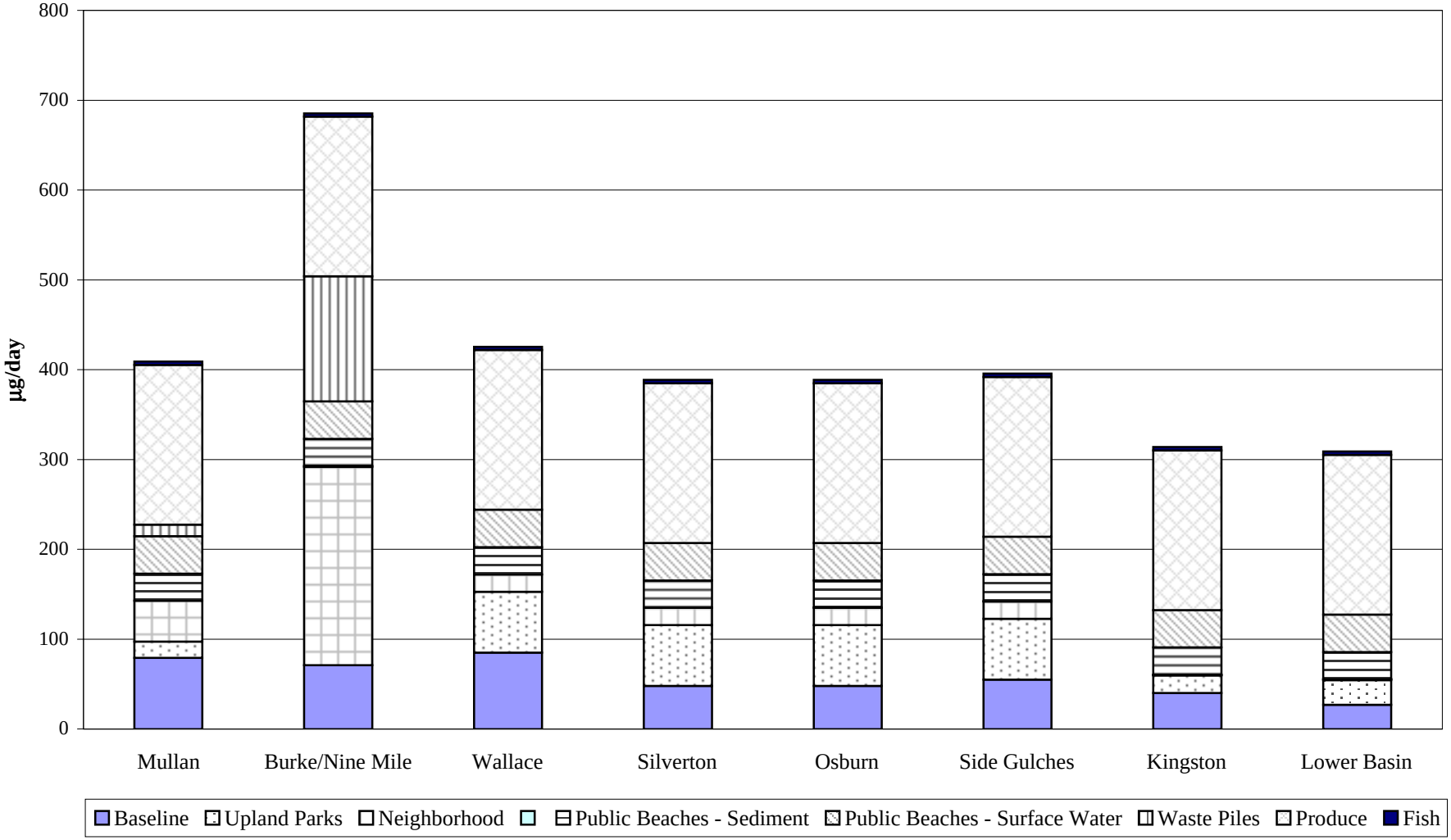


Figure P-3b Summary of Children's Recreational Potential Incremental Lead Intakes
Ages 0-1 and 6-7 (RME)



APPENDIX Q

Development of the IEUBK Bunker Hill Superfund Site Models and Bioavailability Estimates

Appendix Q

Development of the IEUBK Bunker Hill Superfund Site Models and Bioavailability Estimates

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This appendix contains tables and figures illustrating the IEUBK model and the various inputs used in the model. Data presented includes comparisons of geometric mean blood lead levels, geometric standard deviations and predicted percent of children to exceed 10 μg/dl. Other figures in the appendix represent how different bioavailability estimates influence the prediction of blood lead by the IEUBK model.

DEVELOPMENT OF THE IEUBK BUNKER HILL SUPERFUND SITE MODELS AND BIOAVAILABILITY ESTIMATES

- excerpts and additions to the *Final 1999 Five Year Review Report: Bunker Hill Superfund Site* (TerraGraphics 2000a).

Summary of Blood Lead Analyses Performed for the Five Year Review

Both the "Box Model" and Default versions of the Integrated Exposure Uptake Biokinetic (IEUBK) model for lead were analyzed extensively in Chapter 4 of the *Five Year Review for the Populated Areas of the Bunker Hill Superfund Site* (TerraGraphics 2000). The relationship between blood lead, soil, and dust lead levels and the effectiveness of the remedial actions at the BHSS were examined by a variety of techniques in the five-year review. A brief summary of the methods and findings follows.

Two major health response actions, the Lead Health Intervention Program (LHIP) and the Populated Areas soil cleanup, were undertaken to reduce blood lead levels since 1988. Analyses suggest that these programs are largely responsible for the decreases in blood lead levels observed over the past eleven years. The LHIP has monitored children's blood lead levels since 1988, prior to the cleanup of residential properties beginning in 1989 and the two Records of Decision (ROD) cleanups commencing in 1991 and 1994. The LHIP seeks to reduce intake of lead by modifying people's behavior through educating parents and children in the community. The LHIP essentially advises parents how to help their children ingest less dirt through improved hygiene. This concept has been used nation-wide, and has been known since the 1970s to be effective.

Remedial actions seek to reduce intake of lead through cleanup, control, and elimination of sources of lead. The remedial program effectively replaces contaminated soils and dusts throughout the community with clean dirt. The Populated Areas remedial activities are partially completed. Smelterville was finished in 1997 and no home yards now exceed the 1000 mg/kg yard soil lead Remedial Action Objective (RAO). In 1999, about 20% of Kellogg, 44% of Wardner, 17% of Page and 5% of Pinehurst homes have soil levels remaining above 1000 mg/kg lead.

Since the inception of remedial activities in 1989, mean blood lead levels have decreased by 70% (14.2 to 4.3 μg/dl) in Smelterville, 58% in Kellogg (10.8 to 4.5 μg/dl), 55% in Wardner (11.8 to 5.4 μg/dl), 67% in Page (12.5 to 4.1 μg/dl); and 33% in Pinehurst (7.4 to 5.0 μg/dl). The RAO for blood lead levels requires that no more than 5% of children exceed 10 μg/dl and that no more than 1% exceed 15 μg/dl. The percentage of children exceeding 10 μg/dl has decreased from 1989 to 1999 by 78% to 4% in Smelterville, 52% to 6% in Kellogg, 57% to 0% in Page, 54% to 11% in Wardner, and 29% to 9% in Pinehurst, although Pinehurst showed 3% above 10 μg/dl in both 1997 and 1998. The incidence of blood lead levels exceeding 15 μg/dl has decreased from 1989 to 1999 by 42% to 2% in Smelterville, 22% to 0% in Kellogg, 36% to 0% in Page, 31% to

0% in Wardner, and 5% to 2% in Pinehurst.

The LHIP screening of the at-risk population for high blood lead levels and providing follow up services that have substantially reduced blood lead levels among those children has been successfully implemented. Detailed analysis of school records for the 1998-99 school year indicate that the door-to-door survey identified 88% of all-9 month through 9-year old children in the BHSS. The parents of two-thirds of these children elected to participate in the LHIP. Blood lead samples were successfully obtained from 88% of the participants or 375 children. This resulted in the LHIP obtaining samples from 51% of all the children identified in school records, or 68% of those identified in the door-to-door effort. Of those families that declined to participate in 1998, 13% indicated their children were previously tested and did not have a problem, 11% were concerned with the trauma of drawing a venous blood sample from young children, 52% indicated the LHIP was unnecessary at this time, 14% would not specify a reason, and 10% indicated they had recently moved into the area or were planning to leave.

These results are typical of the last eleven years, although refusals have increased in recent years as environmental exposure, blood lead levels, and the number of children at-risk have declined. More than 4000 blood lead samples have been drawn from children. In total, 372 children were targeted for follow up services and 317 or 85% were completed. Each year about half of the participating children have blood lead measurements from the previous year. As a result, this database is large enough and sufficiently representative of the population to support analysis of the blood lead to soil/dust lead relationship and the effectiveness of the remedial actions.

The various sources of lead and the routes by which children are exposed are also discussed. Lead from contaminated soils and dusts are the primary source of lead poisoning at the BHSS. Young children are at greatest risk because of hand-to-mouth activities. These children crawl and play on floors and soils, contaminated dusts adhere to their hands and toys. Children then often place their hands and toys in their mouths, and swallow the dirt. Older children access other contaminated soils outside their home, as they begin to move around the neighborhood and community.

Regression analysis were accomplished to determine the basic relationship between blood lead and soil and dust lead levels. These results suggest that blood lead levels are related to both soil and house dust and that contaminated soil from the overall community, the immediate neighborhood and the home yard all independently contribute to blood lead. The slope or ratio of blood lead decrease per unit of soil/dust lead concentration reduction is about 0.9 μg lead/dl blood per 1000 mg lead/kg dust, and from 0.3 to 2.5 μg lead/dl blood per 1000 mg lead/kg soils. These slopes are consistent with the lower range of similar values from the literature observed at other sites.

More sophisticated statistical analyses (mixed model) were applied to repeated blood lead measurements, or those children sampled in consecutive years. Many of the differences among children are controlled if the same child is examined from year to year, and any blood lead

changes related to modifications in that particular child's environment can be examined. These results show that all children benefitted from the community-wide cleanup efforts through lower blood lead levels. Typical two-year old blood lead levels were reduced more than 5.0 μg/dl through remediation of community and neighborhood soils and consequent dust lead level decreases. Children who had their yard remediated showed additional blood lead reductions of about 1.7 μg/dl in the following year. Children who received a nurse's visit through the LHIP experienced an even greater reduction, averaging about 4.0 μg/dl for the typical recipient over the eleven year period. These effects continue into later years, further reducing blood lead levels in subsequent surveys.

Structural equation analysis is used to examine the pathways or routes by which lead in soils reach children and is absorbed in the blood. These results suggest that typically about 40% of the blood lead due to soil and dust comes through house dust, about 30% from the greater community soils and 30% from the home and immediate neighborhood. Additionally, about 50% to 60% of the lead in house dust due to soils also comes from the greater community with the remainder from the yard and neighborhood. Together with results from analysis of soil/dust lead relationships, these findings suggest that 60% to 80% of lead in both children's blood and house dust derives from soils. Typical house dust concentrations in 1989 and 1999 were about 1400 mg/kg and 600 mg/kg, respectively. Analysis of dusts from similarly situated homes in northern Idaho outside the mining district suggest that about 200 mg/kg to 400 mg/kg of house dust lead is associated with sources other than soil, such as lead paint or residual contamination in structures and carpets. At the conclusion of the soil remediation program, house dust levels are estimated to average about 400 to 500 mg/kg and typical blood lead levels are predicted to be between 3 and 4 μg/dl.

Historic lead intake rates were calculated based on the percentage contributions of the soil and dust sources suggested in the structural equations analysis. These intakes estimate how much lead a typical child ingested each day from house dust and community-wide, neighborhood and home yard soils for the last eleven years. These intake estimates correspond well with observed blood lead levels and help to explain how blood lead levels have changed in response to soil cleanup activities. Lead intake, and consequently, blood lead levels decreased markedly in the first three years (1989-1991) of cleanup as most children's home yards were remediated in the high risk cleanup program. Intake rates and blood lead levels then stabilized from 1992-1996 as more children moved to contaminated homes on the site, off-setting gains in remediation. This stalemate was broken in 1997 as area wide yard removals greatly diminished the number of contaminated home yards and neighborhoods that families could move into. Intakes and blood lead levels have both gradually decreased to historically low levels since 1997.

The intakes calculated above were used in the IEUBK model for lead. IEUBK results suggest that the dose/response rate for blood lead due to soil and dust lead is less than that expected. The response rate or decrease observed in blood lead per 1000 mg/kg soil and dust is about 60% of default values expected at a typical site. This result is similar to the assumptions used in developing the cleanup strategy and establishing the RAOs for the site. This suggests that when

complete, the cleanup strategy, devised in 1990, will be successful in reducing blood lead levels to acceptable criteria. However, based on default response rate parameters suggested by the U.S. EPA, the soil RAO may not be adequate to achieve the blood RAO.

Site-wide Trends in Lead Intake 1988-1998

Typical lead intake from soil and dust sources can be estimated by multiplying soil and dust consumption rates by the respective lead concentrations in each media. This was accomplished for each child for each year assuming a typical combined soil/dust consumption rate of 100 mg/day. Four soil and dust partition scenarios were assumed. Three dust:soil partition scenarios have been applied in the Integrated Exposure Uptake Biokinetic Model (IEUBK) analyses in previous reports and analyses. Those were i) the IEUBK default 55:45 house dust:yard soil partition that assumes that 55% of a child's intake derives from household dust and 45% from residential yard soils, ii) a 40:30:30 house dust:yard soil:community soil partition that assumes that 40% of children's soil/dust ingestion derives from house dust and 30% equally from both home yard and community-wide soils, and iii) a 75:18:7 partition that assumes the child's soil and dust ingestion derives predominantly from house dust, with the remainder proportionately from yard soils and community soils, respectively.

Each of these partitions has been evaluated in previous dose-response analyses in this project as method of encompassing plausible intake rates from soils and house dust. The community-wide geometric soil lead concentrations are used to represent the community soil component in these analyses. The fourth partition added for this report is the 42% house dust / 27% community-wide / 19% neighborhood / 12% yard soil partition suggested by the structural equations analysis. These results are most similar to the 40:30:30 pre-remediation partition noted in historic analyses.

Tables Q4.22a-d through Q4.25a-d summarize the results and trends in estimated lead intake for resident children from soil and dust sources for each year by community and site-wide. All tables and Figures in this appendix are numbered to correspond with similar presentations in Section 4 of the *Five Year Review for the Populated Areas of the Bunker Hill Superfund Site* (TerraGraphics 2000). These results show the combined effects of the high-risk targeting program for yard soils and the portion of the population continuing to live on contaminated yards. These tables show that all the partitions result in similar patterns of decline in overall soil and dust intake estimates over the past decade.

The largest decreases in estimated site-wide intake rates were achieved from 1989-1990 to 1993-1994. Pre-remediation arithmetic mean intake rates were estimated at 250 to 270 μg/day site-wide under the various scenarios. During the first year of remediation 1989-90, intakes decreased by 40-50% to 140 to 155 μg/day lead. Another 20-30% reduction was achieved in 1990-1991 to typical levels of 105-120 μg/day depending on the scenario. From 1991 to 1992 those partitions dominated by house dust showed a 5-15% decrease that was marginally significant. Under the 40/30/30 and default scenarios the 1991-1992 change was not significant

in Kellogg. Intake estimates for 1991-1992 were in the 100-125 μg/day range for Kellogg and Smelterville. From 1992 to 1993 another 15-20% reduction was achieved in all partition scenarios that was highly significant. By 1993, site-wide estimated intake rates were around 80 μg/day or decreased by more than 70% since 1988-89. In 1993-94, a 5 to 10% reduction was noted that was marginally significant (p=0.003-0.02) with site-wide intake rates decreasing to 70-80 μg/day. Geometric mean intake rate estimates show a similar pattern with means reaching 60 μg/day by 1994.

No significant change in typical site-wide intake rates has been noted since 1994, excepting a slight increase in Smelterville in 1998 associated with low dust lead concentrations in a small number of dust samples in 1997. Gradual decreases, however, have occurred as the area-wide cleanups have progressed and site-wide lead intake estimates were in the 55 to 70 μg/day in 1998.

These results are similar to those for blood lead levels in Table Q4.6a-d. Figure Q4-11 contrasts site-wide intake rates and blood lead levels for 1989-1998. All of the dust/soil partitions examined show significant site-wide reductions in estimated lead intake from soil and dust were achieved in 1989-1990, 1990-1991 and 1992-1993. These are the same years in which significant decreases in blood lead levels were also noted site-wide. No significant change in estimated intake rates was evident under any partition scenario for 1994-97 (except Pinehurst in 1994). Similarly, there was little change in mean blood lead levels during those same years. Increases in site-wide blood lead levels were noted in 1991-1992 and 1993-1994. Changes in intake rates were not-significant in those years for those partitions with higher portions of soil and were marginally significant (p=0.02-0.03) for those dominated by house dust. Gradual decreases in both blood lead levels and estimated intake rates have been evident since 1996 as area-wide remediation continues.

Integrated Exposure Biokinetic Model (IEUBK) Analysis

Overview

Determining those soil/dust partitions, intake rates, and structural equation model forms that best describe this population were further assessed through biokinetic analysis. The IEUBK Model for lead that has been applied to the BHSS repeatedly in past analyses. Cleanup criteria for the site were developed using the original version of the IEUBK for lead developed by the USEPA Office of Air Quality Planning and Standards (OAQPS) in 1986. The basic strategy of the analysis conducted during the Populated Areas RI/FS simulated different cleanup scenarios for yard soils. The IEUBK Model was used to estimate resultant blood lead levels, and those were compared to the site RAOs to select a cleanup action level.

The analysis concluded that, in order to meet the RAOs of 95% of the childhood population below 10 μg/dl blood lead, with no (nominally <1%) children exceeding 15 μg/dl:

- All yards with soil concentrations greater than 1000 mg/kg lead must be replaced with soils containing less than 100 mg/kg lead;
- The geometric mean lead concentration of all yards in any community must be less than 350 mg/kg; and
- House dust lead levels must decrease to concentrations similar to those in post remediation yard soils.

In the original RI/FS analysis conducted in 1990, the default soil and dust ingestion/absorption parameters of the IEUBK Model were substantially decreased for the projections to conform with observed site blood lead levels. The 1988-89 data available at that time suggested that the IEUBK default parameters overestimated absorption of soil/dust lead by a factor of about two.

The original OAQPS IEUBK Model's default parameters were adjusted from a general assumption of about 100 mg/day soil ingestion with 30% absorption to 70 mg/day and 20% absorption for the 1989 analyses. This adjustment had the effect of predicting an acceptable action level of 1000 mg/kg rather than a value less than 500 mg/kg, that would have been predicted under the default parameters. Since the RI/FS analyses were conducted, several revisions to the IEUBK Model have been released by the USEPA, and ten years of additional site data have accumulated. These data have been analyzed using both default and adjusted intake and absorption parameters and alternate dust:soil partitions to assess any changes in model performance or site dose-response relationship (TerraGraphics 1993, 1996, 1997, 1998).

Applying the IEUBK Default Parameters

Several community and batch-mode applications were run using the existing blood lead environmental exposure database matching children's observed blood, soil, and house dust lead concentrations. IEUBK model default parameters were used in each case, with the exception of soil and dust lead concentrations. Observed house dust and yard soil lead values were used when available. If the house dust value was missing, the geometric mean house dust lead concentration for that community and year was substituted. Missing yard soil lead values were replaced with the geometric mean pre-remediation community-wide yard soil lead concentration. If both house dust and yard soil lead values were missing, the observation was not included in the analyses. Data available for 8- and 9-year-old children were included by assigning an age of 84 months to these observations.

Three dust:soil partition scenarios were applied in the initial IEUBK analyses. Those were i) the UBK default 55:45 house dust:yard soil partition that assumes that 55% of a child's intake derives from household dust and 45% from residential yard soils, ii) a 40:30:30 house dust:yard

soil:community soil partition that assumes that 40% of children's soil/dust ingestion derives from house dust and 30% equally from both home yard and community-wide soils, and iii) a 75:18:7 partition that assumes the child's soil and dust ingestion derives predominantly from house dust, with the remainder proportionately from yard soils and community soils, respectively. Each of these partitions has been evaluated in previous dose-response analyses at the BHSS. The community-wide geometric soil lead concentrations are used to represent the community soil component in these analyses.

Tables Q4.26 through Q4.28 and Figures Q4-12 through Q4-14 summarize predicted and observed blood lead levels, geometric standard deviations, and percent to exceed RAOs for the period 1988 to 1998, respectively, for Kellogg, Smelterville, and site-wide for each partition scenario. These results are for all ages in the batch mode applications.

Predicted blood lead levels: Table Q4.26 and Figures Q4-12a-c show predicted blood lead levels for 0-84 month-old children. These model runs vary only in soil and house dust lead concentrations as observed in the annual blood lead surveys and the dust:soil partition. Model default values for air, diet, drinking water, and maternal contribution were used. Several of the communities have small numbers of observations and assessment of the effectiveness of model predictions is limited. Additionally, the summary statistics reported in these tables may not agree with those presented in earlier sections of the report due to the deletion of incomplete observations from the IEUBK analyses.

The model results show little difference in predicted blood lead levels among the three dust:soil partition scenarios. This similarity is likely due to the predominance of house dust in each scenario and the apparent relationship between community-wide soils and house dust lead concentrations. All three scenarios seem to over-predict observed blood lead levels by about 12% to 88% over the ten year period.

Figures Q4-12 show that, prior to effects of the yard remediation program, the IEUBK model significantly over-predicts observed blood lead levels. This was noted in 1990 and was the basis for adopting the 70 mg/day ingestion and 20% absorption parameters used in the supporting analyses conducted in the Feasibility Study. However, as the Yard Remediation Program progressed, the degree of over-prediction by IEUBK default parameters decreased. In 1988-89 the effect was nearly 2-fold (i.e., 60-90% over-prediction). Estimated intake rates for lead from soil and dust were near 300 μg/day. From 1990 to 1993, exposure profiles and estimated intake rates were in transition to 70-80 μg/day in 1994. By 1993 the effect was about 25-55% over-prediction. From 1994 through 1998, over-prediction ranged from 7% in 1997 to 57% in 1995. In other years, over-predictions were near 20%.

These observations suggest that the USEPA Default IEUBK Model may be over-predicting absorption from soil and dust sources on the site by about 20 to 25%. Over these years the over-prediction ranged from 7% to 88%, averaging about 25% for the period (i.e., the observed value was about 75% of the predicted value). However, it should be noted that observed blood lead

levels may be suppressed due to lowered soil and dust ingestion rates associated with LHIP efforts. As a result, it is possible that the degree of over-prediction could be significantly less if the population was not pre-conditioned by intervention and education efforts.

Variance in blood lead level estimates: There are differences in predicted geometric standard deviations (gsd) among the three dust:soil partitions that also vary for different cities. Table Q4.27 and Figure Q4-13 show predicted and observed geometric standard deviation for Kellogg, Smelterville, and site-wide scenarios, respectively.

Two different predicted gsds are shown in these tables. The first geometric standard deviation refers to the variation in *community population* estimated blood lead levels for individual homes by batch mode applications. This is the geometric standard deviation associated with the geometric mean for the individual blood levels predicted for each home in the community data base. This gsd reflects the variance associated with soil and dust lead concentrations in the community and age-specific response, as the model is run for all ages and the results are aggregated. This value should not be confused with the *individual* geometric standard deviation used in the community mode IEUBK model. The default gsd value of 1.6 was applied to the individual blood lead estimates to develop a distribution of probable blood lead levels for each individual situation estimated in the batch procedure. These individual distributions were then aggregated and an overall gsd was determined for the entire distribution. The *overall* geometric standard deviation refers to the distribution of blood lead levels (predicted or observed) across each community and reflects variation in exposure among individual situations and age within each community, as well as the potential variation in individual response.

The community predicted gsd is shown in Figures Q4-13a-c. The latter or overall gsd can be compared to the observed geometric standard deviations for the population in Figures Q4-13d-f. Observed gsds have increased gradually from values near 1.6 in 1990 to more than 2.0 (Site-wide, and 1.9 in Kellogg) in 1993 as the cleanup has progressed and have stabilized near 1.8 in recent years. Predicted gsds estimated by the various soil and dust partitions followed a similar pattern, but tended to over-estimate observed values in Kellogg and site-wide prior to 1993, and have generally over-estimated gsds in Smelterville through the entire cleanup.

As there is little reason for the individual gsd to change, most of the temporal differences can likely be attributed to change in exposure variable distributions. Two factors that influence the distribution of individual soil exposures are important to note. Those factors are mobility in the population and the manner in which the cleanup was accomplished. Mobility in the childhood population has had an important effect on exposure profiles for the community. Between 1989 and 1991, overall blood lead levels in young children were reduced by nearly 40% as the percentage of children on contaminated yards was reduced from more than 80% to 25%. However, from 1991 to 1996 the number of children on contaminated yards was unchanged, even increasing in some years. Despite the efforts of the high-risk yard program, there were no significant gains in reducing the number of children at risk since 1991, until area-wide remedial activities were completed in Smelterville in 1995-96. As a result, the only significant exposure

reductions achieved population-wide in 1991 to 1996 were overall decreases in community-wide soils and house dust lead concentrations being achieved through the general cleanup.

The increase in population geometric standard deviation observed in this period is likely in response to the bimodal distribution of yard soil exposures that evolved during the cleanup. The majority of children across all age groups have been in homes with less than 100 mg/kg soil exposures since 1991. The remainder of children, usually those new to the home in the previous year, were on highly contaminated yards. Site-wide, the three scenarios have been fairly consistent in predicted geometric standard deviations, with the default scenario predicting the largest variation in blood levels.

Predicted Toxicity Levels: Table Q4.28 and Figure Q4-14a-c show predicted and observed lead toxicity or percent to exceed 10 μg/dl estimates for the various model runs. Percent toxicity predictions consistently exceed observed values, as expected, because mean blood lead levels are overestimated for most years. However, in those years when the model best predicted the observed values (1994, 1996, 1997, and 1998), percent to exceed were well represented site-wide, slightly over-predicted in Kellogg and, conversely, under-predicted in Smelterville since the completion of remedial actions.

Default Model Discussion: The default application of the IEUBK model has over-predicted blood lead levels and percent of children exceeding 10 μg/dl, since the beginning of cleanup activities. However, the degree of over-prediction has decreased as the cleanup has progressed and intake rates and blood lead levels have been reduced. Predicted exceedances in Smelterville since the completion of cleanup have been similar to observed levels. These results suggest that the slope of the dose-response relationship between blood lead and soil and dust lead may have increased.

There are several factors that could account for this apparent change. Three primary hypotheses for why the dose-response rate has increased as the cleanup proceeded are: i.) demographic changes in the population (i.e., increased soil and dust ingestion rates reflective of demographic and behavioral changes), ii.) changing bio-kinetic relationships at lower blood lead levels (i.e., lead bio-kinetics are not linear and ingested lead is more efficiently absorbed at lower concentrations), and iii.) changes in source characteristics (i.e., as the cleanup proceeds the dominant sources change and differing chemical or physical characteristics affect ingestion or uptake rates).

IEUBK Application of Structural Equation Model Results

Estimating Effective Bioavailability: The three hypotheses discussed above, respectively, suggest that the apparent change in dose-response rate is due to i) higher intake rates, ii) non-linear biokinetics, or iii) increasing bioavailability. Although it is not possible with this database to discern which of these effects is dominant, the latter possibilities can be examined using the results of the structural equations analysis in the IEUBK model. The relationship illustrated in

Figure Q4-11 can be used to estimate the aggregate bioavailability of soils and house dust. This is accomplished by estimating blood lead uptake by dividing observed blood lead levels by the Harley-Kneip age-specific coefficient for lead absorption. Age-specific soil and dust intake rates can be estimated using default ingestion rates from the IEUBK and the soil/dust partition suggested by the structural equations analysis. Aggregate bioavailability of soils and dust can then be estimated by dividing the uptake by the intake.

This analysis is similar to the methodology used to estimate the reciprocal clearance coefficient for lead in earlier IEUBK applications at this site (JEG, et al. 1989; USEPA 1990). However, prior to estimating the effective bioavailability, that portion of lead absorption due to sources other than soils and dusts (i.e., diet, drinking water, and air) must be considered. The calculation can be adjusted to partially account for these factors using the relationship identified in the structural equations analysis for lead. The uptake estimate can be adjusted to reflect only that portion of blood lead related to the soil and dust intake by subtracting the intercept term in the blood lead equation from the observed blood lead and using the resulting difference in the uptake calculation. This corresponds to deducting the other dietary, air and drinking water component uptakes from the absorbed lead. This was accomplished for the site-wide database and Table Q4.29 and Figure Q4-20 summarizes estimated arithmetic and geometric mean bioavailability for the years 1988 to 1998.

The results show that arithmetic mean bioavailability was 17% (or about half the 30% default rate, as used in setting the RAO) in 1988. This value corresponds to the reduced dose response rates used in developing the RAOs. This value then ranged near 22% from the beginning of the cleanup in 1989 through 1995 as was noted in earlier analysis (TerraGraphics 1997). Since 1996, arithmetic mean bioavailability has been near the default level of 30%. The geometric mean bioavailability estimate is lower. The geometric mean was near 14% in 1988 (also near half the default), ranged near 18% in the 1989-95 time period and about 21% since 1996.

This analysis assumes the ingestion rates and underlying bio-kinetics of the IEUBK model. The effective bioavailability is then estimated based on relating observed blood lead absorption and intake rates suggested by the structural equations model. Assuming these parameters apply, assessing the change in bioavailability differs depending on whether the arithmetic or geometric mean is evaluated. Arithmetic mean estimates suggest a marked change in bioavailability to near the 30% default concentrations in 1996. This corresponds to the area-wide cleanups and the resulting effect of re-initiating the downward trend in the percent of children on contaminated home yards. This also marks the period that house dusts become more dominant in the exposure profile.

The structural equations model adds support to the latter hypothesis. These model results suggest that as the area-wide cleanup progresses, community and neighborhood soil concentrations are decreased and the predominant lead source changes from community-wide soils toward house dust. The main pathway to children becomes increasingly dominated by the house dust route and sources other than soil become greater relative contributors. These other sources may be paint or

residual smelter contaminants that are more available, increasing aggregate bioavailability There is little reason to suspect that there are significant differences in chemical or matrix effects between the yard soil and community-wide soil lead. However, there are reasons as to why house dusts would be more bioavailable to children than yard soils. House dust particles are smaller and more accessible to young children than soils. As a result, increased bioavailability, increased dermal adherence, and increased ingestion rates are possible explanations for the enhanced response rate. These dusts are available to children year around, more readily adhere to hands, toys and personal items, and are more prone to absorption by the gut, all other factors being equal. It is also possible that dust ingestion rates have increased as the effectiveness of the health intervention effort has decreased over the years.

Assessing the trend in arithmetic mean bioavailability suggests that blood lead has become more responsive to environmental media concentrations as remediations are completed. The absorption rate has been near the 30% default level since 1996. This suggests that default parameters should be used to predict future compliance with RAOs.

Evaluating the geometric mean bioavailability estimates can lead to a different interpretation. The geometric mean was also near half the 30% default in 1988, as noted. However, the increase noted in 1996 was modest in comparison to the change in the arithmetic mean. Estimated geometric mean bioavailability changed from about 18% to 21%. This change could be due the same factors discussed above, but is not so significant for the bulk of the population. This marked difference in arithmetic and geometric mean estimates suggests that some smaller number of children are responding at much higher rates. These higher response rates could be due to any number of physiological, nutritional, socio-economic, or behavioral causes; or possibly, artifactual due to unaccounted sources.

In any case, it is likely more appropriate to use the geometric mean estimate in evaluating the historic effects of soil and dust exposure in site-specific application of the IEUBK. Using the arithmetic mean results in over-prediction of mean blood lead levels and the percent of children to exceed critical toxicity levels due to the sources in the model. Conversely, use of the geometric mean bioavailability could result in underestimation of mean blood lead levels in the future.

A site-specific application of the IEUBK model was developed for the eleven year period using these results. The 42% house dust:27% community soil:19% neighborhood soil:12% yard soil partition and 18% bioavailability parameters suggested by the structural equations analysis was used. Missing dust lead observations were estimated using the dust lead equation developed in the structural equations model.

Table Q4.30 and Figures Q4-15 through Q4-17 summarize the model results. It should be noted in reviewing the following results that similar results could be obtained by reducing the soil and dust ingestion rate by 40% or several combinations of reduced bioavailability and ingestion rate.

Predicted blood lead levels: Table Q4.30 shows predicted and observed results for two year old and 0-84 month-old children for Kellogg, Smelterville and site-wide, respectively, for all years. This model form reasonably predicts geometric mean blood levels across the site. Both predicted and observed levels have decreased consistently with estimated soil and dust intake rates. Figure Q4-11b shows mean blood lead levels and estimated intake rates calculated by the structural equations 42:27:19:12 partition, similar to Figure Q4-11a for the default model analysis above. This relationship is effectively described by the IEUBK model at the 18% bioavailability level assuming default ingestion rates. Figures Q4-15a-c show predicted and observed values for 0-84 month-old children. Figures Q4-15d-f show similar results for two year-old children. Smelterville results are confounded by few numbers of observations in several years. These figures indicate good correspondence between modeled and observed blood lead levels for this critical segment of the population. Other age groups show similar results, indicating that the 84 month assumption for eight and nine year old children has not significantly biased the outcome predictions.

Variance in blood lead level estimates: Figures Q4-16a-c and Table Q4.30 show observed and the predicted community and overall gsds for all ages for all years and communities. The results are similar to those noted in the EPA Default model applications. Predicted overall gsds exceeded observed levels prior to 1993 and have been near or slightly less than observed levels since. The individual model geometric standard deviation used to estimate individual variation in these analyses was the community mode default 1.6 value. In community mode IEUBK applications the observed gsd is for the community population is consistently under-predicted. Although, community mode results are not shown in these tables, it is important to discuss the estimated gsds.

The under-prediction in the community mode can be resolved by adjusting the individual gsd model input to about 1.8. However, it is not clear whether this adjustment would correctly reflect the exposure-uptake relationship. The under-prediction in population gsd is related to the character and underlying causes for the observed distribution in dose-response. In evaluating the effective bioavailability, a continuing divergence in the estimated arithmetic and geometric means was noted over time. This divergence increased as blood lead and the percent of the population to exceed critical toxicity levels decreased. This suggests that within the population some smaller number of children are responding at higher rates of absorption. This group becomes "more different" as the cleanup proceeds and soil and dust exposures subside. Two possibilities that might explain this divergence are immediately evident. Either these children are more sensitive to the sources included in the model, or they are exposed to sources unaccounted for in the model. If the former is the case, it would be proper to expand the individual gsd to accommodate these response rates. If the latter is true, then the higher blood lead levels are not related to the sources in the model and using an enhanced gsd to predict the response would be incorrect.

In the batch mode application of the IEUBK model, typical (or 50th %-tile) blood lead levels are

estimated for each exposure situation (or home) in the community. These results are accomplished for each age group and then aggregated. Resulting means and variances can be calculated from these results by age or for all ages 0-84 months. Although the means calculated by this method are representative of the population, the associated gsd reflects only the variance in exposures and age-specific response. The variance in individual response is not included. As a result, this gsd is not appropriate to use in estimating probabilities of exceeding critical toxicity criteria. This gsd is shown in Figures Q14-16a-c as the *community* gsd. The potential individual variance should also be applied to the typical blood lead estimates. The individual gsd of 1.6 was applied to the batch mode blood lead estimates and the resulting distributions were aggregated for the community/population. These results are shown in Table Q4.30 and Figures Q16a-c as the predicted *overall* gsd. Using this value, observed overall geometric standard deviations for the population were over-predicted in the early years of the cleanup and have been similar to observed community gsds in recent years.

Predicted Toxicity Levels: In the community mode application, the population variance is underestimated by the pathways model as discussed above. As a result, the percent of children to exceed of critical toxicity levels are also under-predicted. This prediction could also be improved by adjusting the individual gsd from the default value of 1.6 to near 1.8. However, the same considerations as to the appropriateness of this adjustment discussed above applies.

In the batch mode, application of the 1.6 individual gsd to the typical blood lead estimates results in an overall gsd near 1.8 and a reasonable approximation of the percent of children to exceed 10 ug/dl throughout the cleanup years. Table Q4.30 and Figures Q4-17a-c show these results for the batch mode reduced bioavailability model. These results show good correspondence between predicted and observed percentage of children to exceed 10 μg/dl. Percent to exceed values are well predicted for all three areas and all years prior to 1994. Since 1994, percent to exceed values have been slightly under-predicted. This result is not unexpected. It is anticipated that the IEUBK Model would somewhat under-predict exceedance of the 10 μg/dl criteria in recent years. This is because, follow-up investigations have shown that some high blood levels are due to exposures unaccounted for in the model. The model results would not be expected to predict these excursions.

Those children whose apparent greater response rates confound this analysis are, not coincidentally, those intensively evaluated by the PHD follow-up program. Review of these children's histories since 1996, when suggested increases in effective bioavailability were noted, provides some insight regarding the potential sources of this variation.

These histories show that PHD officials found the majority of these children are socio-economically disadvantaged, highly mobile, with care often provided in multiple locations among extended family or cooperative situations. These children tend to exhibit frequent hand to mouth activity and poor to fair personal and home hygiene. Most were also exposed to high concentration soil and dust sources in play areas or away from their home. Generally, those soil sources identified within the BHSS were areas away from the child's home at locations that had

not yet been remediated. These included relatives' and day care yards in un-remediated portions of Kellogg, hillsides, common areas surrounding particular housing complexes and Milo Creek flood debris. Extended recreational activities at contaminated locations and moving from contaminated homes outside of the BHSS has also been noted more frequently in recent years. Several children were noted to be in homes with relatively high dust concentrations. Some were indicated to be in homes with poor interior paint condition.

These observations suggest that the high response rates are due to a number of factors. Frequent hand to mouth activities, poor personal and home hygiene, high house dust concentrations and any paint lead reflected in those dusts are all factors accounted for in the model analysis. It would be appropriate to increase the individual gsd to accommodate these factors in exposure variance. Those factors related to sources outside the home environment, i.e., flood debris, hillsides campgrounds, un-remediated areas in Kellogg, and contaminated soils in the greater Coeur d'Alene Basin are conditions unrelated to the model predictions.

The batch mode results shown in Table Q4.30 and Figures Q4-17a-c indicate that the IEUBK, utilizing the reduced absorption rate and soil partition factors suggested by the structural equations analysis, is an effective predictor of mean blood lead levels throughout the cleanup process. This methodology is useful in reassessing the cleanup criteria and likelihood that blood lead RAOs will be achieved. However, there are exposure factors unaccounted for in the model constructs. These include other sources of exposure and peculiar conditions that affect individual children leaving them at greater risk. With respect to residential soils and dust, these analyses suggest that the cleanup has been effective in reducing risk to acceptable levels (i.e., those reflected in the RAO) for the vast majority of the population. There are, however, individual situations and additional sources of lead remaining in and around the BHSS putting some children at-risk. Some of these sources will be addressed in upcoming remedial activities. Those problems identified outside the scope of the remediation strategy will need to be resolved on a case by case basis.

Figures Q4-18a-b and Q4-19a-b show plots of observed versus predicted arithmetic and geometric mean blood lead levels, respectively, site-wide for all age groups aggregated for all years, for both the EPA Default and Box Model. In these figures the more recent observations are at the lower left and progress back in time toward pre-remedial conditions at the right. Figures Q4-18b and Q4-19b demonstrate that observed mean blood lead levels are accurately reproduced using the dust: soil partitions factors and effective bioavailability suggested by the pathways analysis. Use of default bioavailability results in over-prediction of observed blood lead levels in earlier years, as seen in Figures Q4-18a and Q4-19a. In recent years at low intake rates, the model results are converging, suggesting that default bioavailability may be becoming more appropriate as exposures and blood lead levels approach acceptable criteria.

It must be remembered that use of the reduced or site-specific absorption rate may or may not be appropriate for future site conditions. Caution should be emphasized because the reduced absorption rate could be either due to reduced bioavailability or ingestion rates or some

combination of both. This reduction may reflect temporary modifications in the environment due to ongoing cleanup efforts or behavioral changes due to public awareness associated with the health intervention programs that would not be permanent. In that case, it would be inappropriate to apply reduced factors to evaluations of the long-term risk reduction strategy.

Summary and Conclusions

Quantifying Exposure Pathways: Blood lead and soil and dust pathways were explored through structural equations analysis. These models indicate that from 40% to 50% of the blood lead absorbed from soils and dusts is through house dust with about 30% from community-wide soils and 30% from the home yard and immediate neighborhood (200 foot radius). These relative contributions agree with findings of earlier studies conducted in the early 1980s and analyses used to develop the cleanup criteria for the site.

The same structural equation models suggest that community-wide soil contribute between 50% and 60% of the soil lead component in house dust with the neighborhood and home yard contributing about 20% each. This results in soils overall contributing about 80% of lead to house dust in the pre-remedial environment and an estimated 50% to 60% post-remedial.

Intake rate estimates generated from the results of the pathways model indicate that typical pre-remediation lead intake rates due to soil and dust were 250 to 270 μg/day site-wide. During the first year of remediation 1989-1990, intakes decreased by 40-50% to 140 to 155 μg/day lead. Another 20-30% reduction was achieved in 1990-91 to typical levels of 105-120 μg/day. In 1991-92 only marginal decreases were achieved to the 100-125 μg/day range. From 1992 to 1993 another 15-20% reduction was achieved and estimated site-wide intake rates were about 80 μg/day or decreased by more than 70% since 1988-89. In 1993-94, a 5 to 10% reduction was noted that was marginally significant, with average site-wide intake rates decreasing to 70-80 μg/day. Only gradual decreases, however, have occurred since 1994 as the area-wide cleanups have progressed and site-wide lead intake estimates were in the 55 to 70 μg/day by 1998.

Biokinetic Models: Both mean blood lead levels and percent of children to exceed toxicity levels have paralleled the intake estimates. Analyses of these data indicate that the decreasing blood lead levels are consistent with the estimated intake of lead from soils and dusts. The dose-response ratio of blood lead per unit of soil and dust intake seems to be increasing. This suggests that either ingestion rates are increasing, the lead is becoming more bioavailable, the dose-response curve is changing, and/or the baseline sources of lead (other than soils and dusts) are progressively larger contributors.

Application of the IEUBK model for lead continues to indicate that default parameters over estimate resultant blood lead levels, although the magnitude of over-prediction is diminished at lower intake rates in recent years. Application of the model using the soil and dust partitions suggested in the pathways model, IEUBK ingestion parameters, and an effective bioavailability of 18%; accurately predicts geometric mean blood lead levels throughout the eleven year cleanup

period.

This model form, however, under-predicts the observed variance in the population blood lead levels and consequently the percent of children to exceed critical toxicity criteria. There are significant questions as to whether the highest blood lead concentrations observed in recent years are due to sources accounted for in the remedial strategy and subsequent IEUBK analyses, or represent peculiar exposure situations.

This application is consistent with the analysis used to develop the cleanup criteria for the existing Populated Areas ROD. Substitution of post-remedial concentrations in this model format indicates blood lead RAOs would be achieved. Application of default mode IEUBK parameters would continue to predict excessive absorption rates and failure to meet the RAOs.
In either case, some number of exceedances of toxicity criteria can be expected due to sources of lead in the environment that remain unaddressed by remedial activities. Also, exceedance levels for 1-2 year-old children are expected to be greater than 5%, in any case. It is unclear if a model based on conditions observed during the course of the cleanup and associated intervention activities can accurately predict future blood lead levels.

Although observed blood lead trends at the BHSS have improved significantly during site response efforts, the assumptions and Lead Model inputs used to develop the soil and dust RAOs may not be sufficient to achieve the blood RAOs. The remedy may not be protective because of the observed increase in rates of blood lead dose-response or additional unremediated sources of exposure.

Figure Q4-11a
Site Wide Intake Rates and Geometric Mean Blood Lead Levels

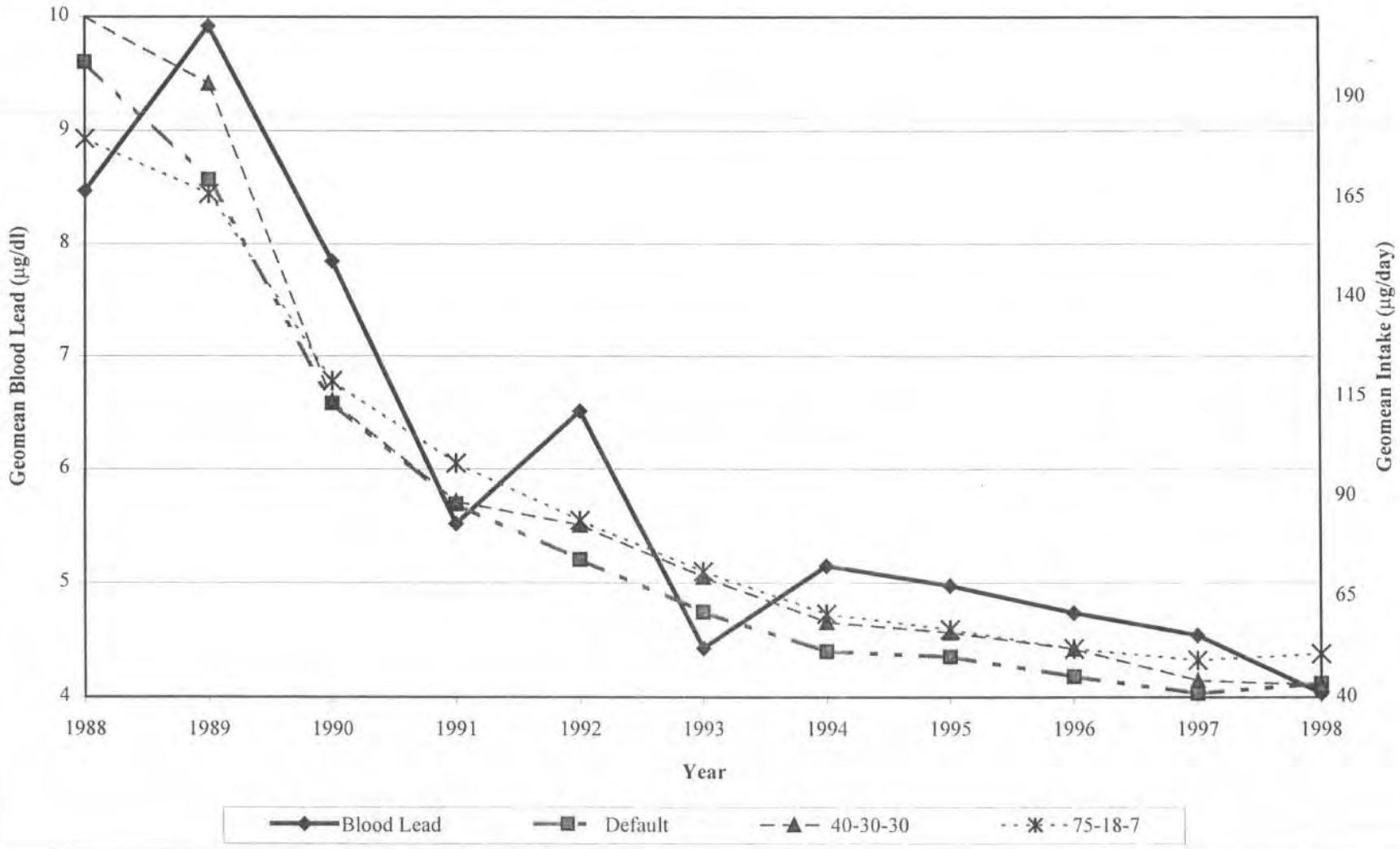
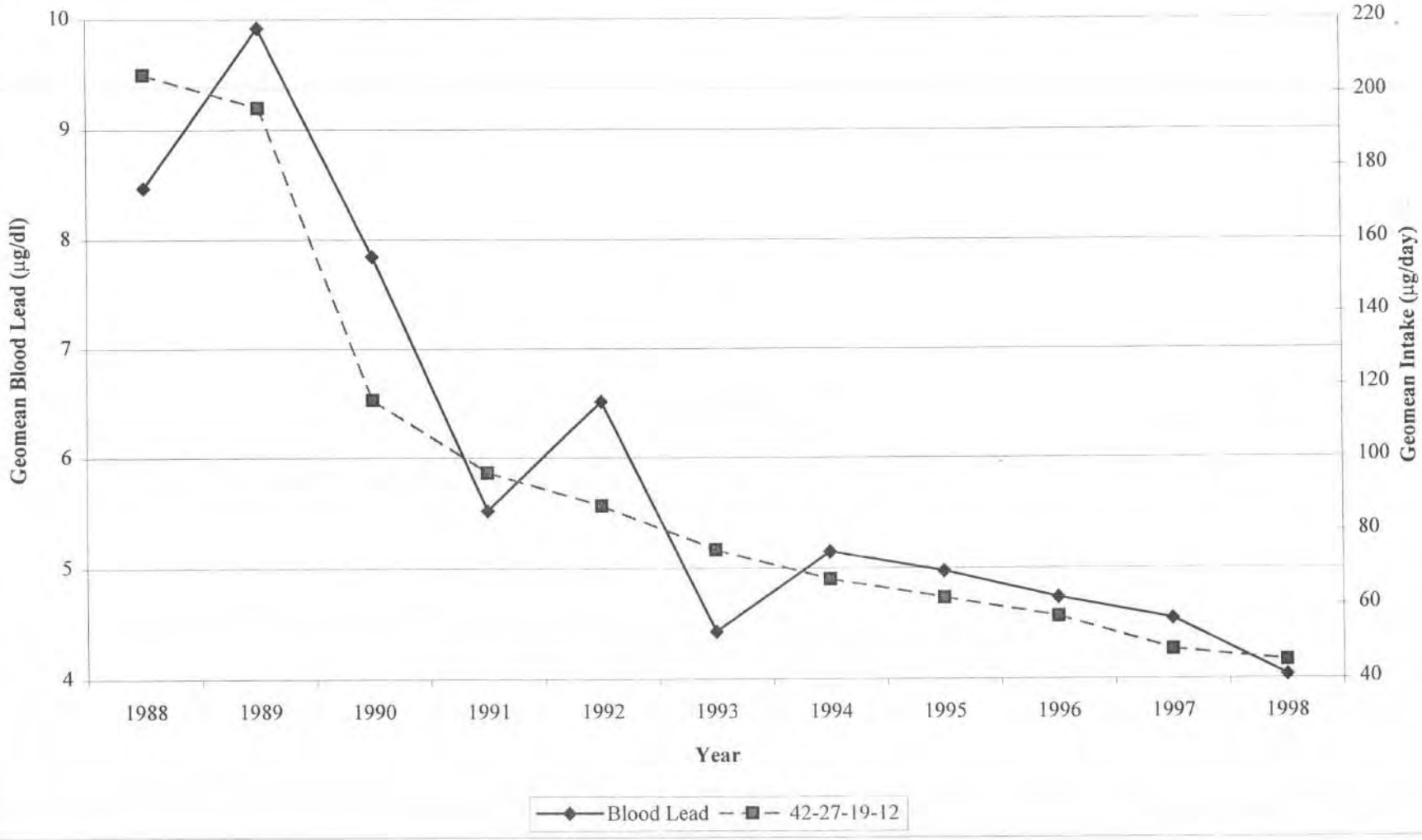


Figure Q4-11b
Site Wide Intake Rates and Geometric Mean Blood Lead Levels



Tables 4.6 a-d
Multiple Comparisons of Analysis of Variance for Mean Blood Lead Levels

Table 4.6 a Blood Lead by Year Site-Wide

Year	N	Mean	GeoMean	GeoP-value
1988	229	9.9	8.5	-
1989	275	11.4	9.9	0.0011
1990	362	8.9	7.8	0.0001
1991	365	6.3	5.5	0.0001
1992	414	7.4	6.5	0.0001
1993	445	5.6	4.4	0.0001
1994	416	6.2	5.1	0.0012
1995	404	6.0	5.0	0.4264
1996	397	5.8	4.7	0.2646
1997	337	5.4	4.5	0.3631
1998	375	4.8	4.0	0.0105
Overall				0.0001

Table 4.6 b Blood Lead by Year Kellogg

Year	N	Mean	GeoMean	GeoP-value
1988	169	9.2	8.1	-
1989	212	10.8	9.3	0.0089
1990	193	9.3	8.3	0.0212
1991	177	6.9	6.0	0.0001
1992	211	8.1	6.9	0.0062
1993	228	6.3	5.2	0.0001
1994	232	6.7	5.5	0.3794
1995	251	6.4	5.2	0.3541
1996	225	6.4	5.1	0.6954
1997	199	5.9	5.0	0.7499
1998	212	4.9	4.0	0.0006
Overall				0.0001

Table 4.6 c Blood Lead by Year Smelterville

Year	N	Mean	GeoMean	GeoP-value
1988	32	14.2	11.6	-
1989	36	14.6	13.2	0.3374
1990	29	9.9	8.7	0.0009
1991	48	6.6	5.9	0.0005
1992	55	8.3	7.4	0.0178
1993	66	6.7	5.8	0.0130
1994	48	6.0	5.3	0.3967
1995	40	7.2	6.2	0.1444
1996	51	6.4	5.8	0.4748
1997	33	5.6	5.2	0.2708
1998	43	6.4	5.8	0.2268
Overall				0.0001

Table 4.6 d Blood Lead by Year Pinehurst

Year	N	Mean	GeoMean	GeoP-value
1988	-	-	-	-
1989	-	-	-	-
1990	107	7.4	6.7	-
1991	116	5.1	4.7	0.0001
1992	119	6.1	5.5	0.0017
1993	119	3.5	2.6	0.0001
1994	109	5.4	4.6	0.0001
1995	97	4.6	4.0	0.1015
1996	103	4.1	3.7	0.2169
1997	86	4.2	3.5	0.5884
1998	100	4.1	3.5	0.9791
Overall				0.0001

Table Q4.22 Estimated Soil and Dust Lead Intake (Default 55:45 Dust/Soil)[1]

Table Q4.22a Site Wide

Year	N	Mean	GeoMean	GeoP-value
1988	70	272	199	-
1989	49	263	169	0.2428
1990	139	154	113	0.0001
1991	162	109	88	0.0028
1992	230	101	74	0.0233
1993	199	81	61	0.0096
1994	203	71	51	0.0248
1995	153	78	50	0.7802
1996	162	68	45	0.2857
1997	100	65	41	0.3536
1998	157	57	43	0.5272
Overall				0.0001

Table Q4.22b Kellogg

Year	N	Mean	GeoMean	GeoP-value
1988	44	314	223.5	-
1989	31	330	204.5	0.6045
1990	68	176	133.3	0.0011
1991	75	126	108.2	0.0757
1992	124	125	91.4	0.1216
1993	111	84	63.7	0.0006
1994	105	75	53.9	0.1210
1995	97	84	57.0	0.6229
1996	107	69	49.5	0.2119
1997	59	73	48.1	0.8206
1998	82	63	47.0	0.8661
Overall				0.0001

Table Q4.22c Smelterville

Year	N	Mean	GeoMean	GeoP-value
1988	21	226	183.7	-
1989	14	162	137.7	0.2170
1990	15	218	186.3	0.3578
1991	27	125	109.5	0.0045
1992	26	76	58.1	0.0006
1993	19	118	84.8	0.1212
1994	35	97	64.5	0.2899
1995	20	118	64.3	0.9896
1996	12	132	46.4	0.4596
1997	15	31	26.9	0.1671
1998	26	41	39.7	0.0026
Overall				0.0001

Table Q4.22d Pinehurst

Year	N	Mean	GeoMean	GeoP-value
1990	46	97	74.8	-
1991	51	82	61.6	0.1612
1992	66	70	58.4	0.6608
1993	55	67	54.1	0.5079
1994	43	41	36.9	0.0022
1995	30	40	30.6	0.1914
1996	37	44	35.9	0.3450
1997	18	80	35.9	0.9986
1998	35	40	34.3	0.8364
Overall				0.0001

[1] assumes dust/soil intake is 55% housedust, 45% yard soil

Table Q4.23 Estimated Soil and Dust Lead Intake (40:30:30)[1]

Table Q4.23a Site Wide

Year	N	Mean	GeoMean	GeoP-value
1988	70	259	210	-
1989	49	256	193	0.4517
1990	139	145	114	0.0001
1991	162	106	89	0.0014
1992	226	100	83	0.2872
1993	197	82	70	0.0037
1994	201	71	59	0.0030
1995	151	76	56	0.5353
1996	160	68	52	0.3642
1997	100	63	44	0.0769
1998	153	54	43	0.7760
Overall				0.0001

Table Q4.23b Kellogg

Year	N	Mean	GeoMean	GeoP-value
1988	44	282	221	-
1989	31	294	210	0.7191
1990	68	176	153	0.0023
1991	75	127	116	0.0009
1992	124	124	106	0.2042
1993	111	91	80	0.0001
1994	105	78	66	0.0055
1995	97	87	70	0.4602
1996	107	75	63	0.1854
1997	59	73	54	0.1556
1998	82	62	49	0.4252
Overall				0.0001

Table Q4.23c Smelterville

Year	N	Mean	GeoMean	GeoP-value
1988	21	245	222	-
1989	14	214	197	0.4460
1990	15	218	204	0.6990
1991	27	137	127	0.0005
1992	26	98	92	0.0029
1993	19	115	100	0.5401
1994	33	89	76	0.0990
1995	20	92	52	0.0999
1996	12	100	40	0.5051
1997	15	26	25	0.1778
1998	26	34	33	0.0074
Overall				0.0001

Table Q4.23d Pinehurst

Year	N	Mean	GeoMean	GeoP-value
1990	46	76	60	-
1991	51	66	52	0.2228
1992	62	57	50	0.7066
1993	53	55	47	0.5968
1994	43	36	33	0.0012
1995	28	34	29	0.2679
1996	35	38	33	0.3963
1997	18	65	34	0.8128
1998	31	36	32	0.7103
Overall				0.0001

[1] assumes dust/soil intake is 40% housedust, 30% yard soil, 30% community soil

Table Q4.24 Estimated Soil and Dust Lead Intake (75:18:7)[1]

Table Q4.24a Site Wide

Year	N	Mean	GeoMean	GeoP-value
1988	70	271	180	-
1989	49	285	166	0.5716
1990	139	153	119	0.0002
1991	162	118	98	0.0123
1992	226	104	84	0.0182
1993	197	87	71	0.0097
1994	201	76	61	0.0169
1995	151	79	57	0.4020
1996	160	72	52	0.3095
1997	100	80	49	0.5740
1998	153	67	51	0.7430
Overall				0.0001

Table Q4.24b Kellogg

Year	N	Mean	GeoMean	GeoP-value
1988	44	324	200	-
1989	31	359	189	0.7662
1990	68	171	141	0.0263
1991	75	137	124	0.1640
1992	124	121	101	0.0140
1993	111	92	79	0.0017
1994	105	80	66	0.0338
1995	97	88	68	0.7320
1996	107	70	59	0.1206
1997	59	89	60	0.9178
1998	82	75	58	0.8169
Overall				0.0001

Table Q4.24c Smelterville

Year	N	Mean	GeoMean	GeoP-value
1988	21	203	168	-
1989	14	175	147	0.5464
1990	15	215	194	0.4579
1991	27	139	133	0.0031
1992	26	103	84	0.0013
1993	19	124	100	0.3688
1994	33	101	79	0.2628
1995	20	103	70	0.5791
1996	12	176	56	0.5829
1997	15	37	31	0.1753
1998	26	50	49	0.0023
Overall				0.0001

Table Q4.24d Pinehurst

Year	N	Mean	GeoMean	GeoP-value
1990	46	108	79	-
1991	51	85	61	0.0731
1992	62	72	59	0.7033
1993	53	67	54	0.4466
1994	43	44	39	0.0112
1995	28	35	28	0.0210
1996	35	44	36	0.1265
1997	18	97	38	0.8356
1998	31	39	32	0.4653
Overall				0.0001

[1] assumes dust/soil intake is 75% housedust, 18% yard soil, 7% community soil

Table Q4-25 Estimated Soil and Dust Lead Intake (42:27:19:12)[1]

Table Q4.25a Site Wide

Year	N	Mean	GeoMean	GeoP-value
1988	70	248	205	-
1989	49	255	196	0.6421
1990	139	139	116	0.0001
1991	162	110	96	0.0046
1992	226	99	87	0.0979
1993	197	83	75	0.0013
1994	201	76	67	0.0290
1995	151	74	62	0.1624
1996	160	68	57	0.1365
1997	100	65	48	0.0437
1998	153	53	45	0.3334
Overall				0.0001

Table Q4.25b Kellogg

Year	N	Mean	GeoMean	GeoP-value
1988	44	274.77	216	-
1989	31	293.6	212	0.8828
1990	68	167.06	156	0.0029
1991	75	130.18	125	0.0001
1992	124	116.57	109	0.0057
1993	111	92.82	88	0.0001
1994	105	81.56	76	0.0021
1995	97	86.08	77	0.8641
1996	107	73.6	68	0.0373
1997	59	74.39	60	0.0896
1998	82	59.75	51	0.0913
Overall				0.0001

Table Q4.25c Smelterville

Year	N	Mean	GeoMean	GeoP-value
1988	21	226	214	-
1989	14	213	203	0.6609
1990	15	212	204	0.9766
1991	27	149	147	0.0001
1992	26	115	110	0.0001
1993	19	119	111	0.9000
1994	33	103	96	0.1913
1995	20	79	60	0.0038
1996	12	105	42	0.2992
1997	15	27	25	0.1391
1998	26	35	34	0.0041
Overall				0.0001

Table Q4.25d Pinehurst

Year	N	Mean	GeoMean	GeoP-value
1990	46	77	63	-
1991	51	64	53	0.1229
1992	62	57	52	0.7879
1993	53	54	48	0.3338
1994	43	39	37	0.0029
1995	28	35	32	0.0875
1996	35	39	36	0.2661
1997	18	69	38	0.7214
1998	31	36	33	0.4254
Overall				0.0001

[1] assumes dust/soil intake is 42% housedust, 27% community soil, 19% neighborhood soil, 12% yard soil

Figure Q4-12a
Predicted and Observed Geometric Mean Blood Lead Levels in 0-84 Month Old Children
Default Parameters - Three Dust:Soil Partition Scenarios
Kellogg 1988-1998

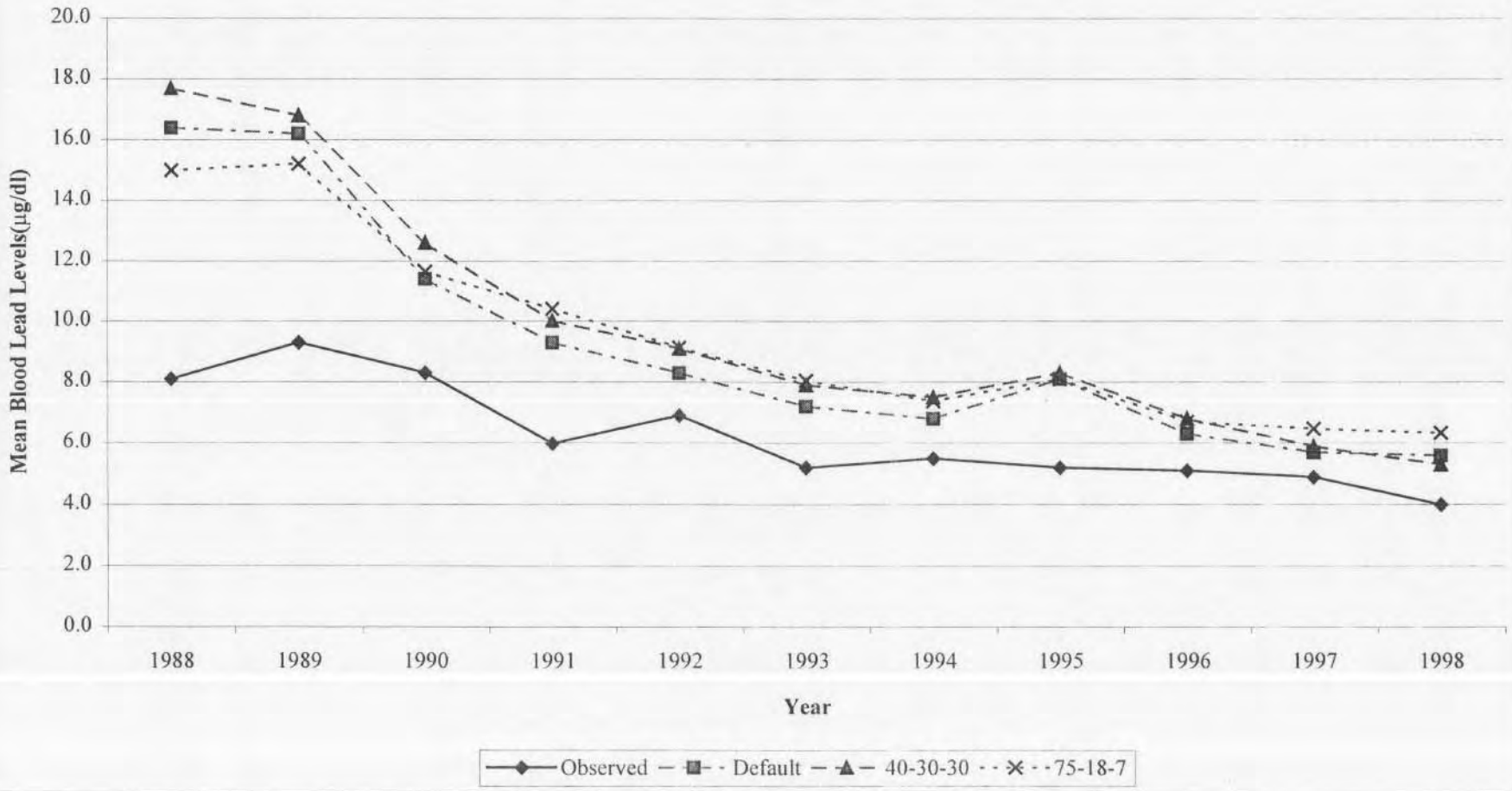


Figure Q4-12b
Predicted and Observed Arithmetic Mean Blood Lead Levels in 0-84 Month Old Children
Default Parameters - Three Dust:Soil Partition Scenarios
Smelterville 1988-1998

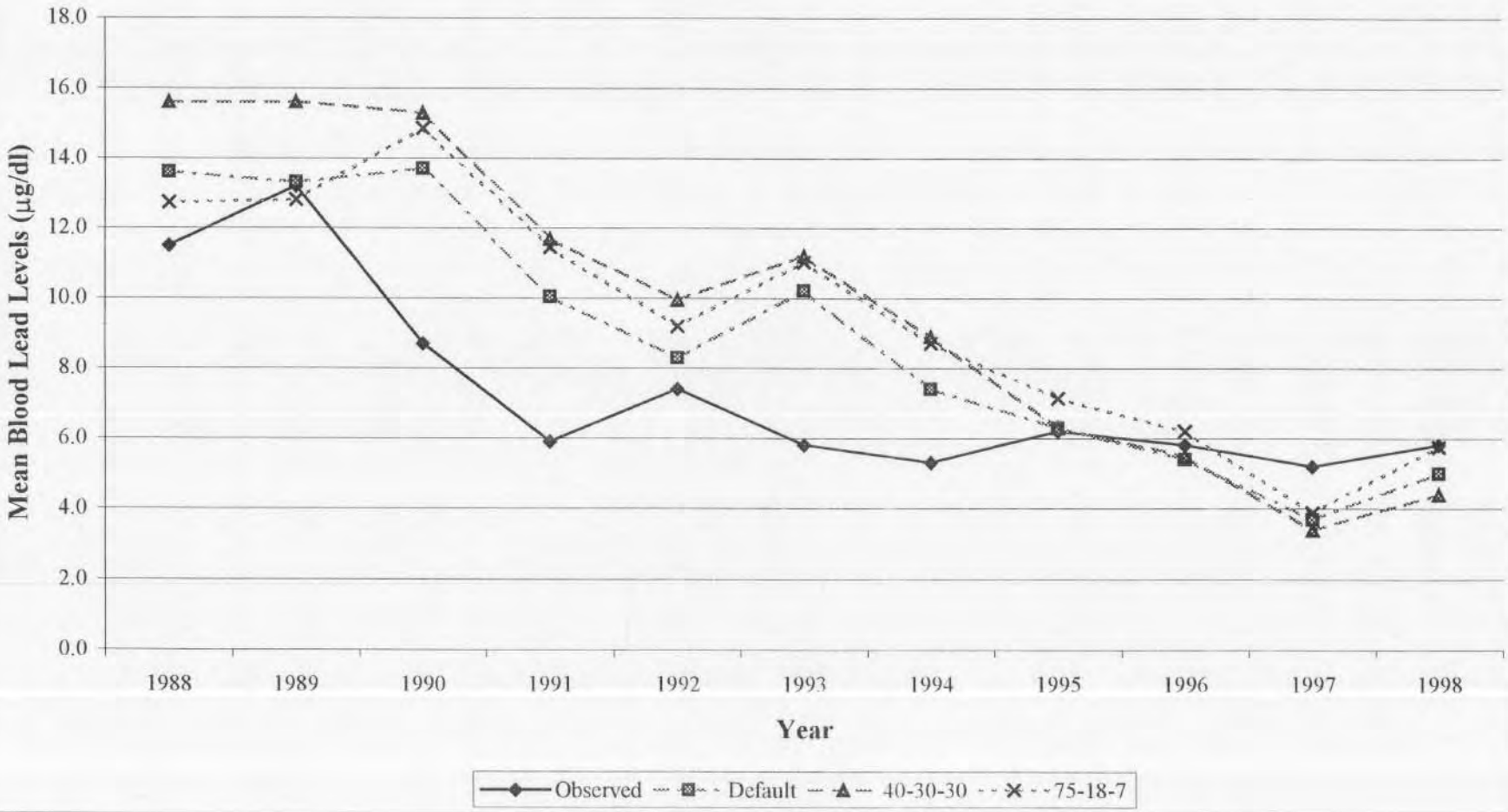


Figure Q4-12c
Predicted and Observed Geometric Mean Blood Lead Levels in 0-84 Month Old Children
Default Parameters - Three Dust:Soil Partition Scenarios
Site Wide 1988-1998

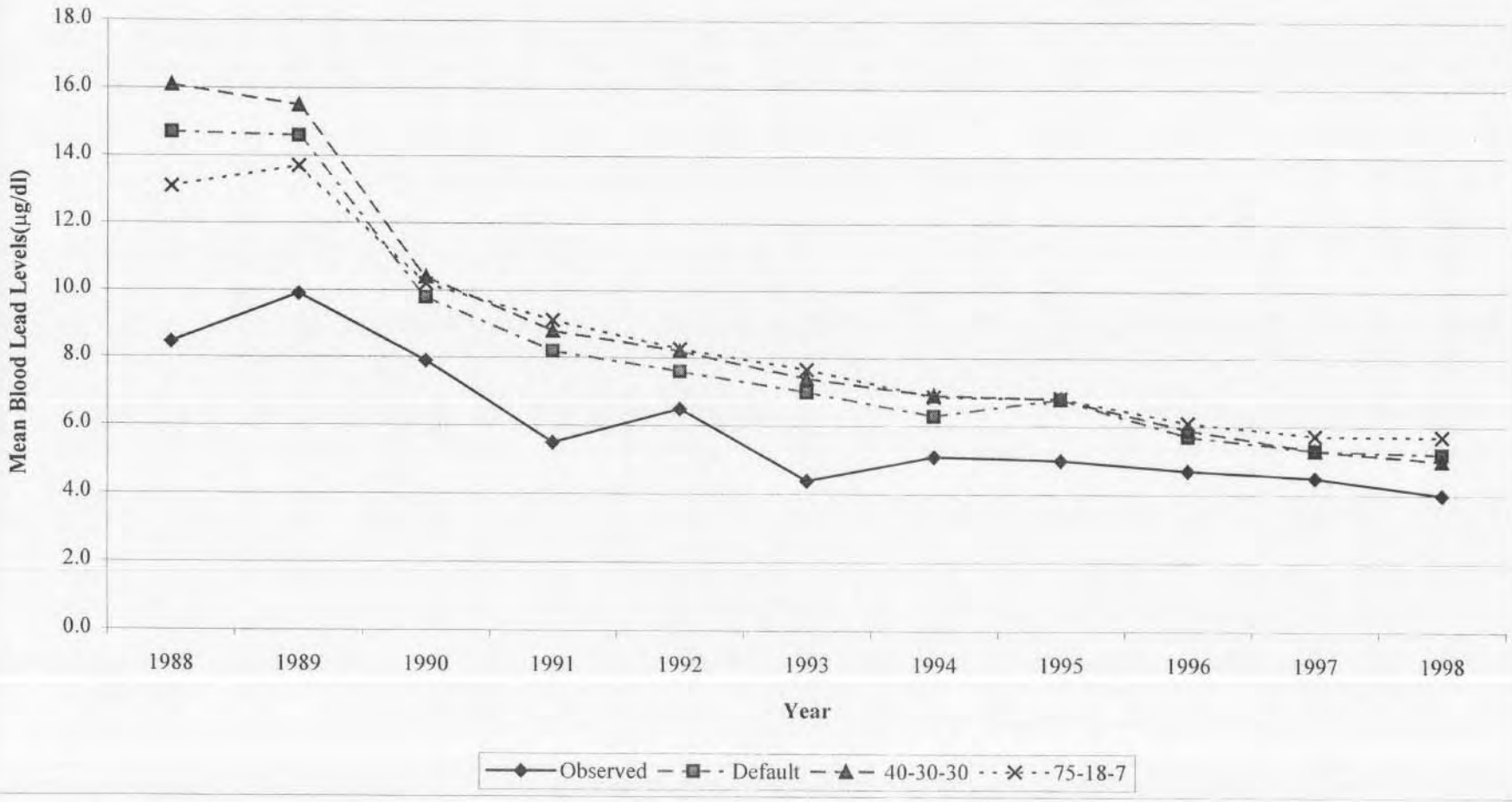


Figure Q4-13a
Predicted Community and Observed Geometric Standard Deviations Default Parameters
Three Dust:Soil Partition Scenarios
Kellogg 1988-1998

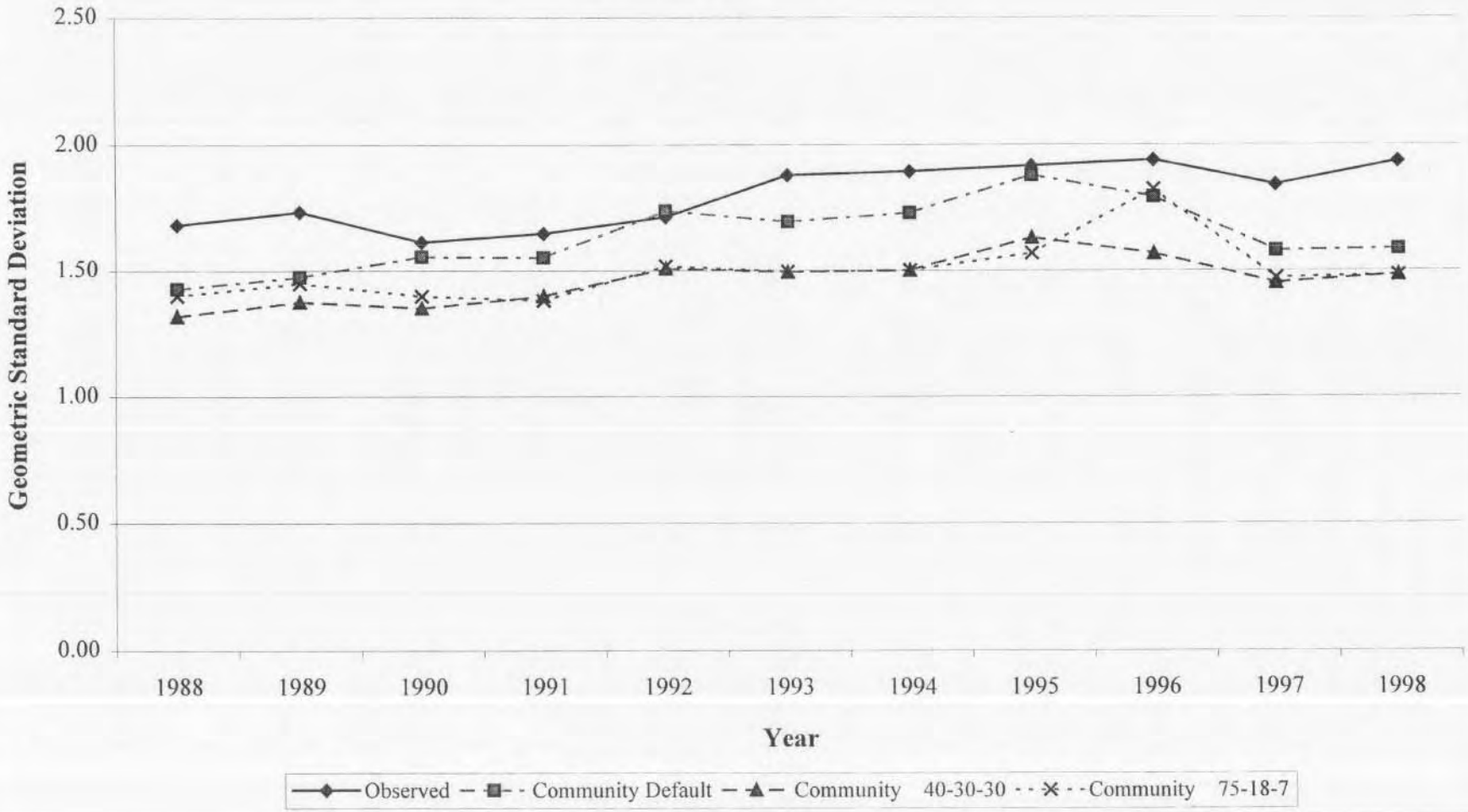


Figure Q4-13b
Predicted Community and Observed Geometric Standard Deviations Default Parameters
Three Dust:Soil Partition Scenarios
Smelterville 1988-1998

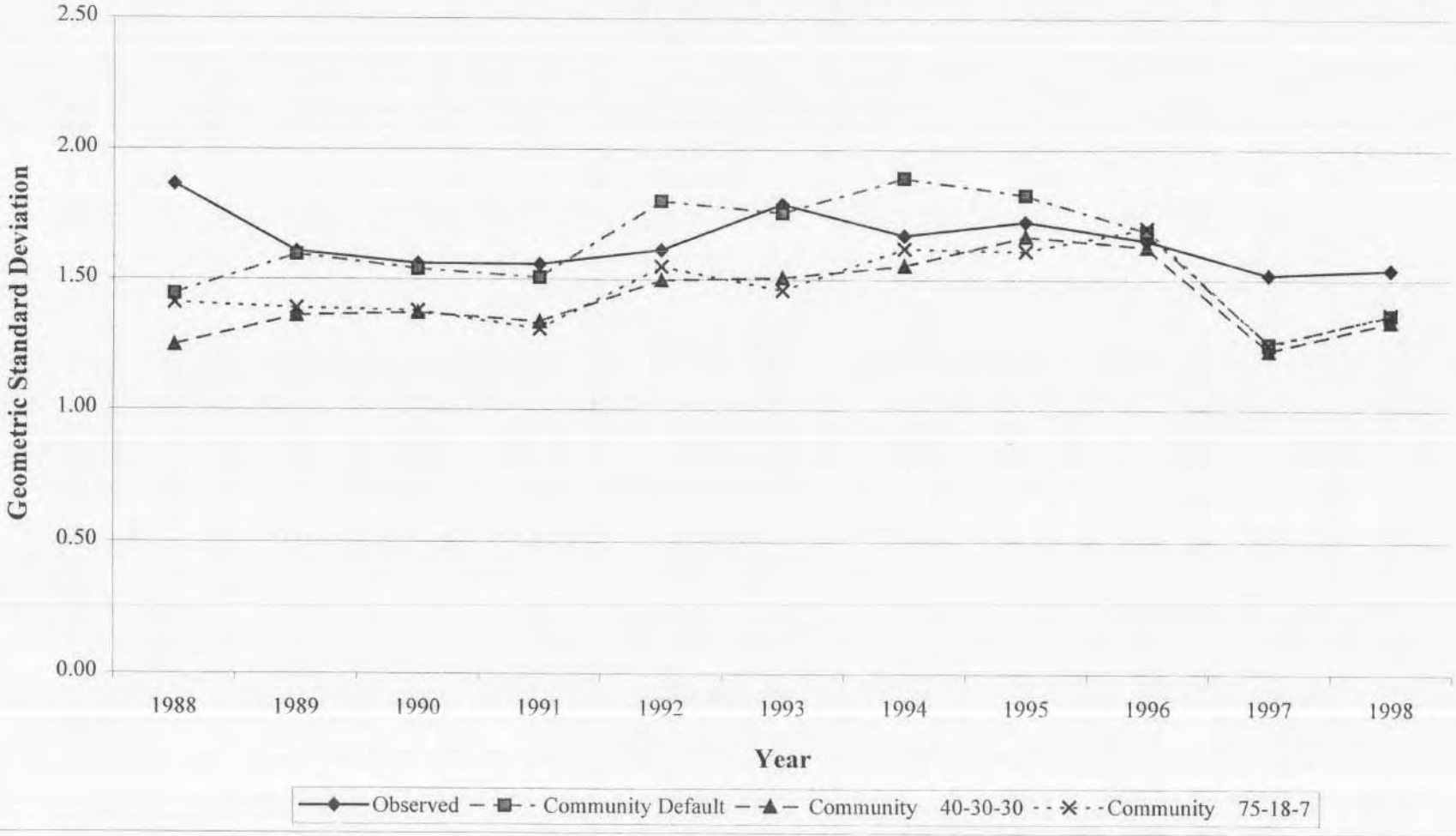


Figure Q4-13c
Predicted Community and Observed Geometric Standard Deviations Default ParametersThree Dust:Soil Partition Scenarios
Site Wide 1988-1998

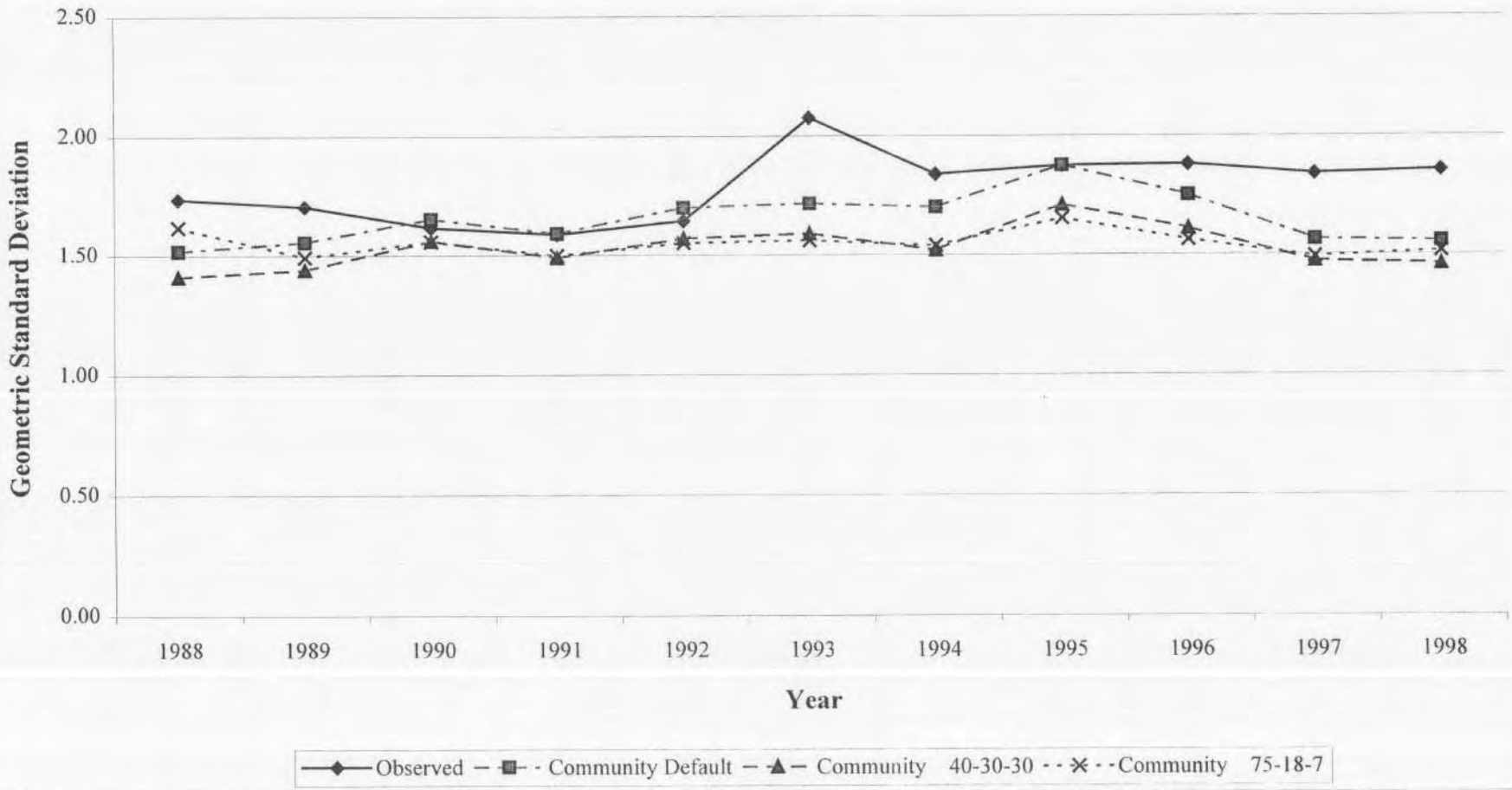


Figure Q4-13d
Predicted Overall and Observed Geometric Standard Deviations Default Parameters
Three Dust:Soil Partition Scenarios
Kellogg 1988-1998

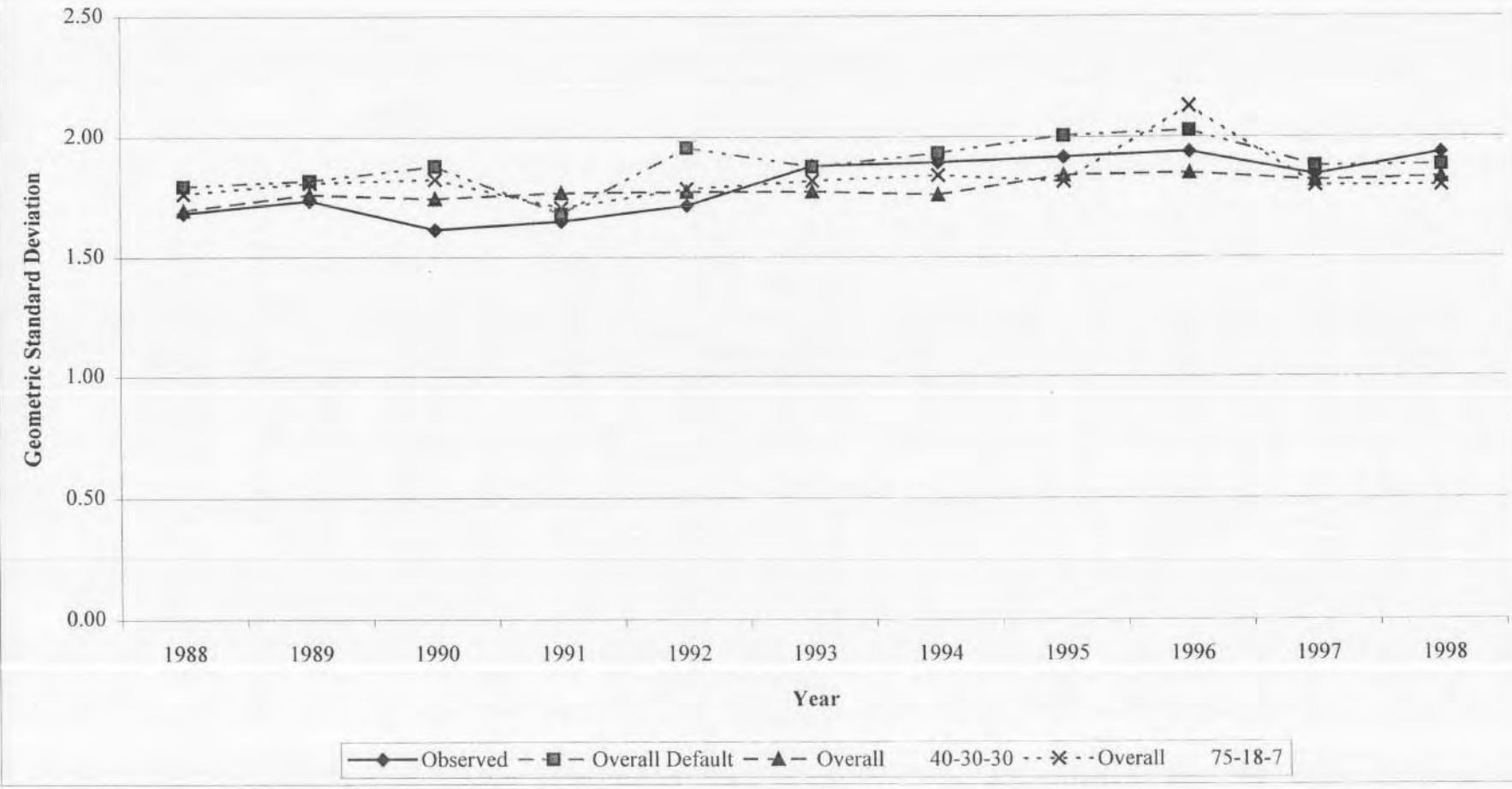


Figure Q4-13e
Predicted Overall and Observed Geometric Standard Deviations Default Parameters
Three Dust:Soil Partition Scenarios
Smelterville 1988-1998

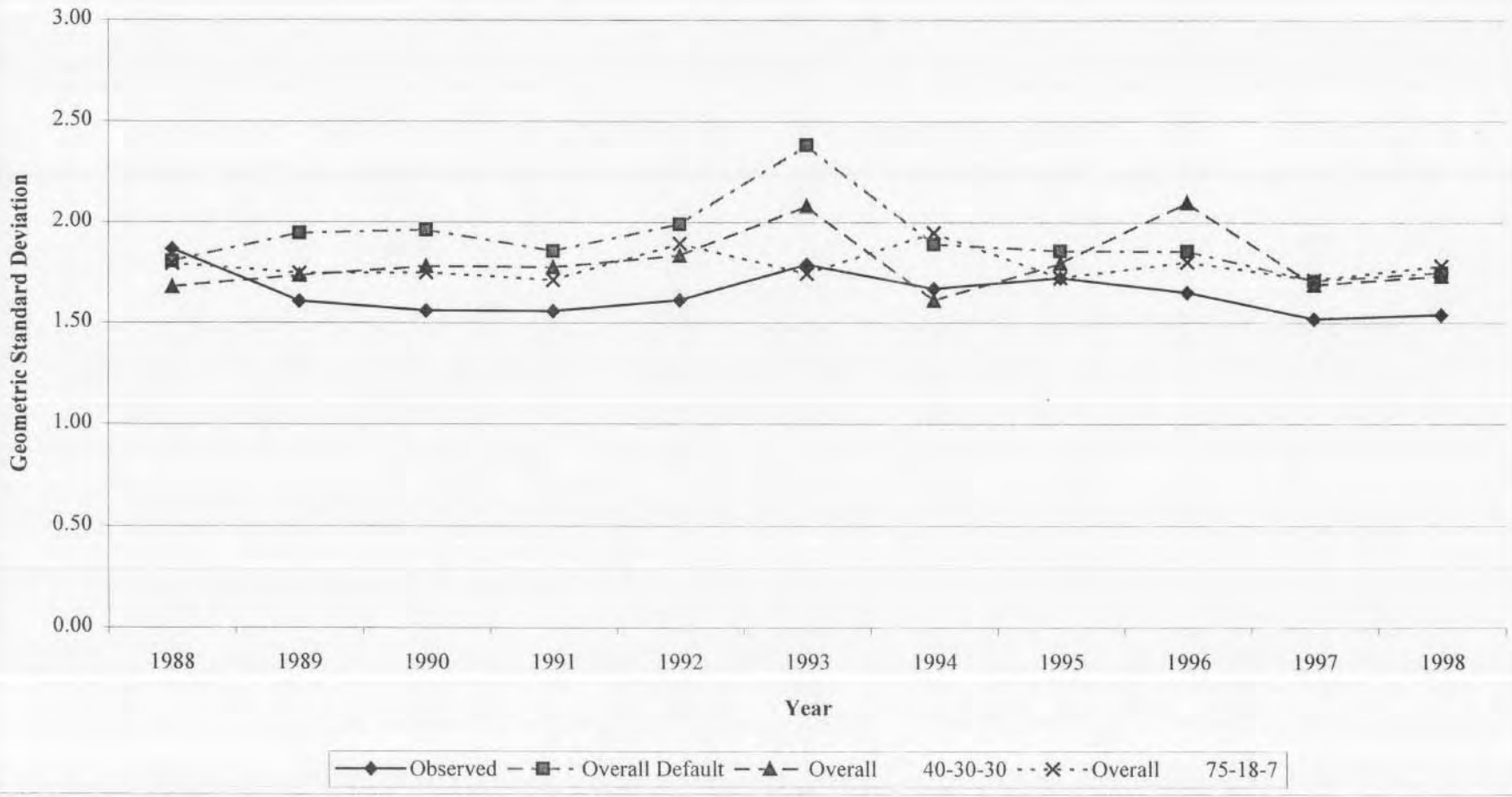


Figure Q4-13f
Predicted Overall and Observed Geometric Standard Deviations Default Parameters
Three Dust:Soil Partition Scenarios
Site Wide 1988-1998

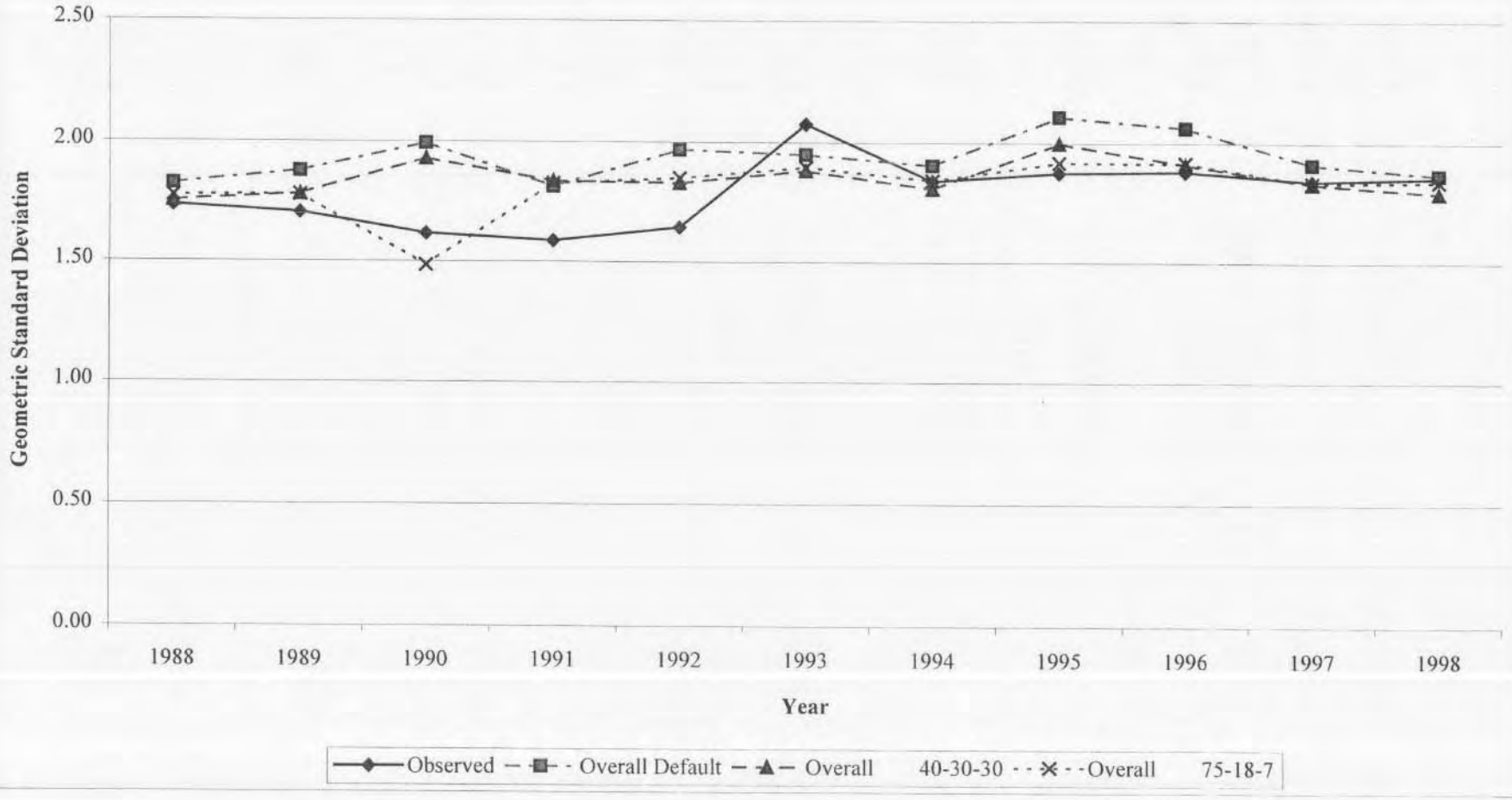


Table Q4.26 Predicted and Observed Geometric Mean Blood Lead Levels for 0-84 Month Old Children, Default Parameters - Three Dust:Soil Partition Scenarios

Observed and Predicted Blood Lead Levels (μg/dl)				
Year	Observed	Default	40-30-30	75-18-7
Kellogg				
1988	8.1	16.4	17.7	15.0
1989	9.3	16.2	16.8	15.2
1990	8.3	11.4	12.6	11.7
1991	6.0	9.3	10.0	10.4
1992	6.9	8.3	9.1	9.1
1993	5.2	7.2	7.9	8.1
1994	5.5	6.8	7.5	7.4
1995	5.2	8.1	8.3	8.1
1996	5.1	6.3	6.8	6.7
1997	4.9	5.7	5.9	6.5
1998	4.0	5.6	5.3	6.3
Smelterville				
1988	11.5	13.6	15.6	12.7
1989	13.2	13.3	15.6	12.8
1990	8.7	13.7	15.3	14.9
1991	5.9	10.1	11.7	11.5
1992	7.4	8.3	10.0	9.2
1993	5.8	10.2	11.2	11.0
1994	5.3	7.4	8.9	8.7
1995	6.2	6.3	6.3	7.1
1996	5.8	5.4	5.5	6.2
1997	5.2	3.7	3.4	3.9
1998	5.8	5.0	4.4	5.7
Site Wide				
1988	8.5	14.7	16.1	13.1
1989	9.9	14.6	15.5	13.7
1990	7.9	9.8	10.4	10.2
1991	5.5	8.2	8.8	9.1
1992	6.5	7.6	8.2	8.3
1993	4.4	7.0	7.4	7.7
1994	5.1	6.3	6.9	6.9
1995	5.0	6.8	6.8	6.8
1996	4.7	5.7	5.9	6.1
1997	4.5	5.3	5.3	5.7
1998	4.0	5.2	5.0	5.7

Table Q4.27 Predicted and Observed Geometric Standard Deviation, 1988-1998, Default Parameters - Three Dust:Soil Partition Scenarios

Observed and Predicted Geometric Standard Deviations							
Year	Observed	Community Default	Overall Default	Community 40-30-30	Overall 40-30-30	Community 75-18-7	Overall 75-18-7
Kellogg							
1988	1.68	1.43	1.80	1.32	1.70	1.40	1.76
1989	1.74	1.48	1.82	1.38	1.76	1.45	1.81
1990	1.61	1.56	1.88	1.35	1.74	1.40	1.83
1991	1.65	1.55	1.68	1.40	1.77	1.38	1.71
1992	1.72	1.74	1.96	1.51	1.77	1.52	1.78
1993	1.88	1.70	1.88	1.50	1.77	1.50	1.82
1994	1.89	1.73	1.93	1.50	1.76	1.50	1.84
1995	1.92	1.88	2.01	1.63	1.84	1.57	1.82
1996	1.94	1.79	2.03	1.57	1.85	1.82	2.13
1997	1.84	1.58	1.88	1.45	1.82	1.47	1.80
1998	1.93	1.59	1.88	1.48	1.83	1.48	1.80
Smelterville							
1988	1.87	1.44	1.80	1.25	1.68	1.41	1.79
1989	1.61	1.60	1.95	1.36	1.74	1.39	1.75
1990	1.56	1.54	1.96	1.37	1.78	1.38	1.75
1991	1.56	1.51	1.86	1.34	1.78	1.31	1.71
1992	1.61	1.80	1.99	1.50	1.83	1.55	1.89
1993	1.79	1.76	2.38	1.51	2.08	1.46	1.74
1994	1.67	1.89	1.89	1.55	1.61	1.62	1.95
1995	1.72	1.83	1.86	1.67	1.80	1.61	1.72
1996	1.65	1.69	1.86	1.63	2.10	1.70	1.80
1997	1.52	1.25	1.71	1.23	1.69	1.25	1.72
1998	1.54	1.37	1.75	1.34	1.73	1.37	1.78
Site Wide							
1988	1.74	1.52	1.83	1.41	1.75	1.62	1.78
1989	1.71	1.56	1.88	1.44	1.78	1.49	1.78
1990	1.62	1.65	1.99	1.56	1.93	1.56	1.48
1991	1.59	1.59	1.81	1.49	1.84	1.50	1.83
1992	1.65	1.70	1.97	1.57	1.83	1.55	1.85
1993	2.08	1.72	1.95	1.59	1.88	1.56	1.90
1994	1.84	1.70	1.91	1.52	1.81	1.54	1.84
1995	1.88	1.88	2.11	1.71	2.00	1.65	1.92
1996	1.88	1.75	2.06	1.61	1.91	1.56	1.92
1997	1.84	1.56	1.91	1.47	1.83	1.49	1.83
1998	1.86	1.56	1.87	1.46	1.79	1.51	1.84

Figure Q4-14a
Predicted and Observed Percent of 0-84 Month Old Children with Blood Lead Level Exceeding 10 μg/dl Default Parameters - Three Dust : Soil Partition Scenarios
Kellogg 1988-1998

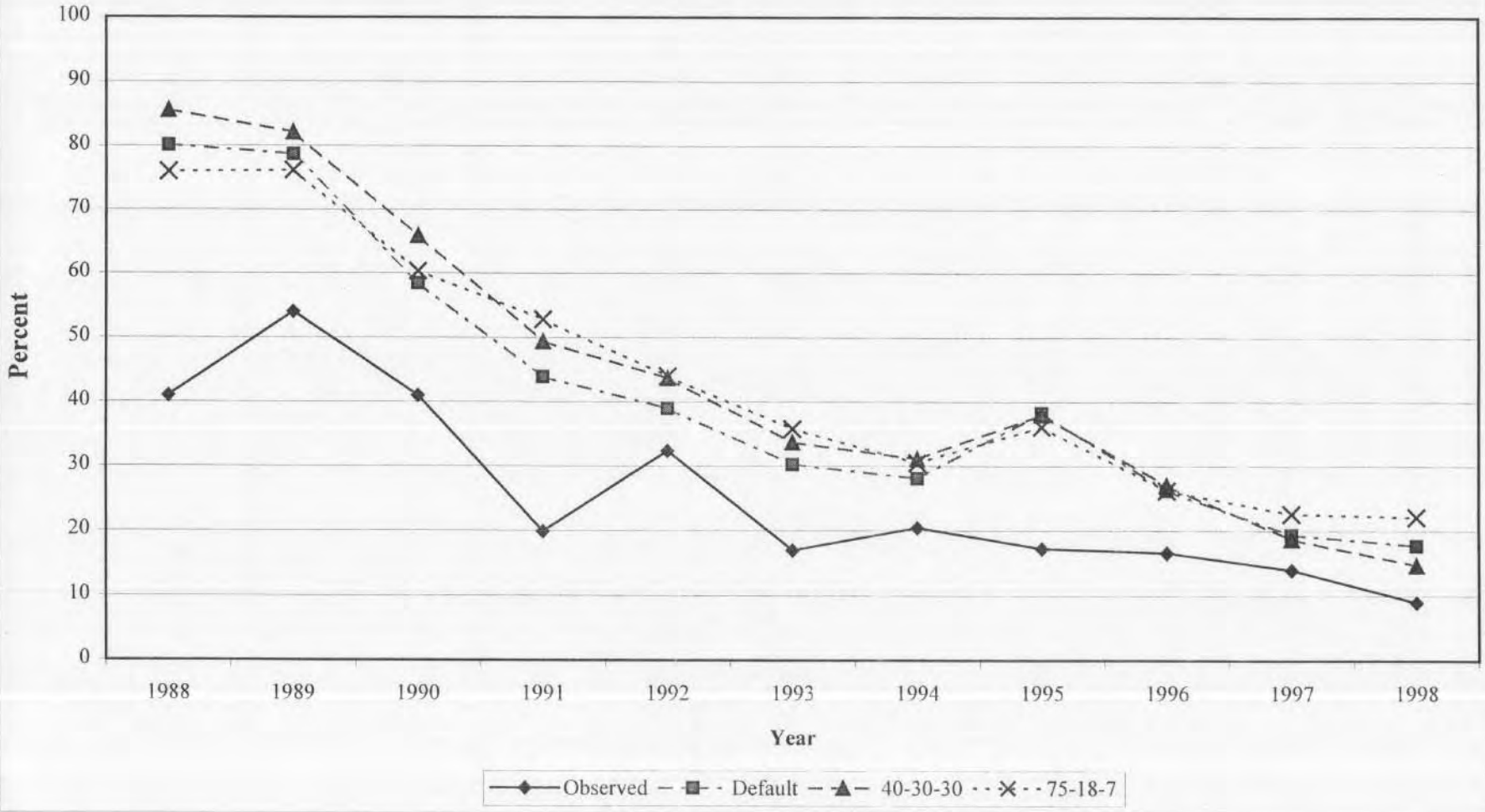


Figure Q4-14b
Predicted and Observed Percent of 0-84 Month Old Children with Blood Lead Level Exceeding 10μg/dl Default Parameters - Three Dust : Soil Partition Scenarios
Smelterville 1988-1998

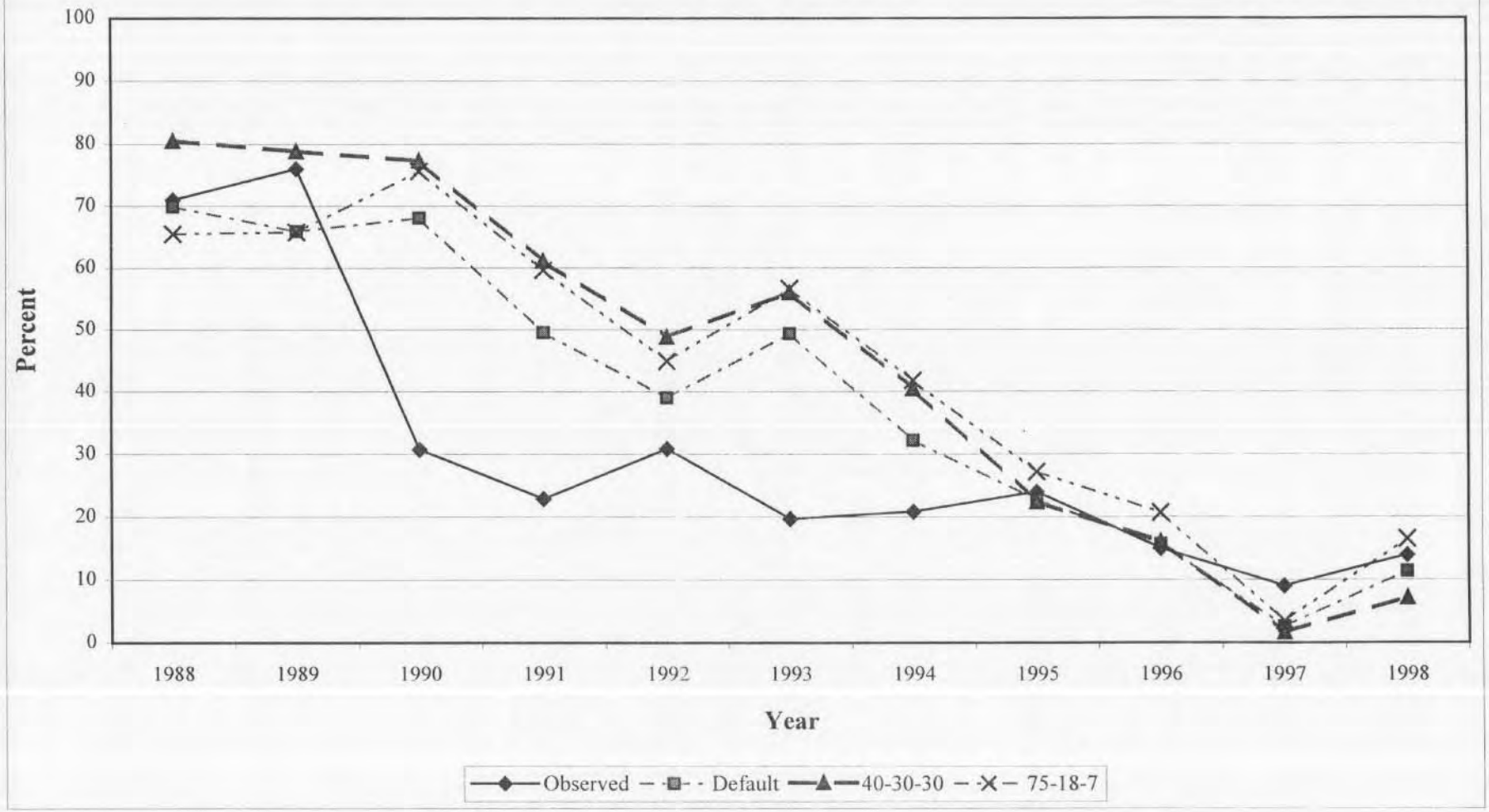


Figure Q4-14c
Predicted and Observed Percent of 0-84 Month Old Children with Blood Lead Level Exceeding 10 μg/dl Default Parameters - Three Dust : Soil Partition Scenarios
Site Wide 1988-1998

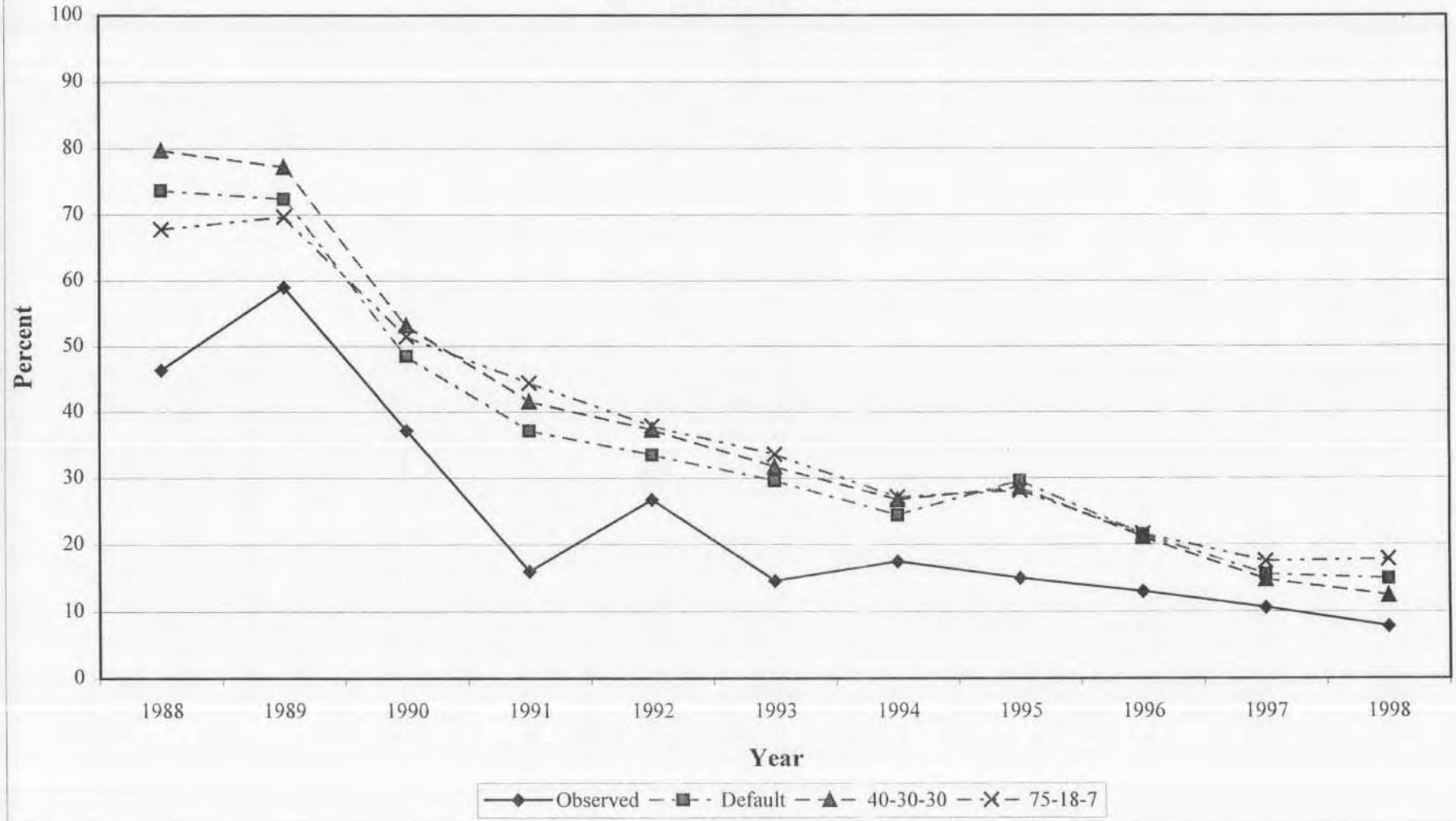


Table Q4.28 Predicted and Observed Lead Toxicity
Percent of 0-84 Month Old Children With Blood Lead Greater Than 10 μg/dl
Default Parameters - Three Dust:Soil Partition Scenarios

Observed and Predicted Lead Toxicity				
Year	Observed	Default	40-30-30	75-18-7
Kellogg				
1988	41	80	86	76
1989	54 (52)[1]	79	82	76
1990	41 (40)	58	66	60
1991	20	44	49	53
1992	32	39	44	44
1993	17 (18)	30	34	36
1994	20	28	31	31
1995	17	38	38	36
1996	17	26	27	26
1997	14 (15)	19	19	23
1998	9 (10)	18	15	22
Smelterville				
1988	71 (72)	70	81	66
1989	76 (78)	66	79	66
1990	31	68	77	76
1991	23	50	61	60
1992	31	39	49	45
1993	20	49	56	57
1994	21	32	41	42
1995	24 (28)	23	22	27
1996	15 (12)	16	16	21
1997	9	3	2	4
1998	14	11	7	17
Site Wide				
1988	46	74	80	68
1989	59 (56)	72	77	70
1990	37	49	53	52
1991	16 (15)	37	42	44
1992	27	34	37	38
1993	15	30	32	34
1994	17	24	27	27
1995	15	30	29	28
1996	13 (12)	22	21	22
1997	11	16	15	18
1998	8	15	12	18

Notes:

1 - Values in parentheses indicate total population values, and the full number of observations were not included in the model runs due to missing environmental media concentrations.

Figure Q4-15a
Predicted and Observed Blood Lead in 0-84 Month Old Children
42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability
Kellogg 1988-1998

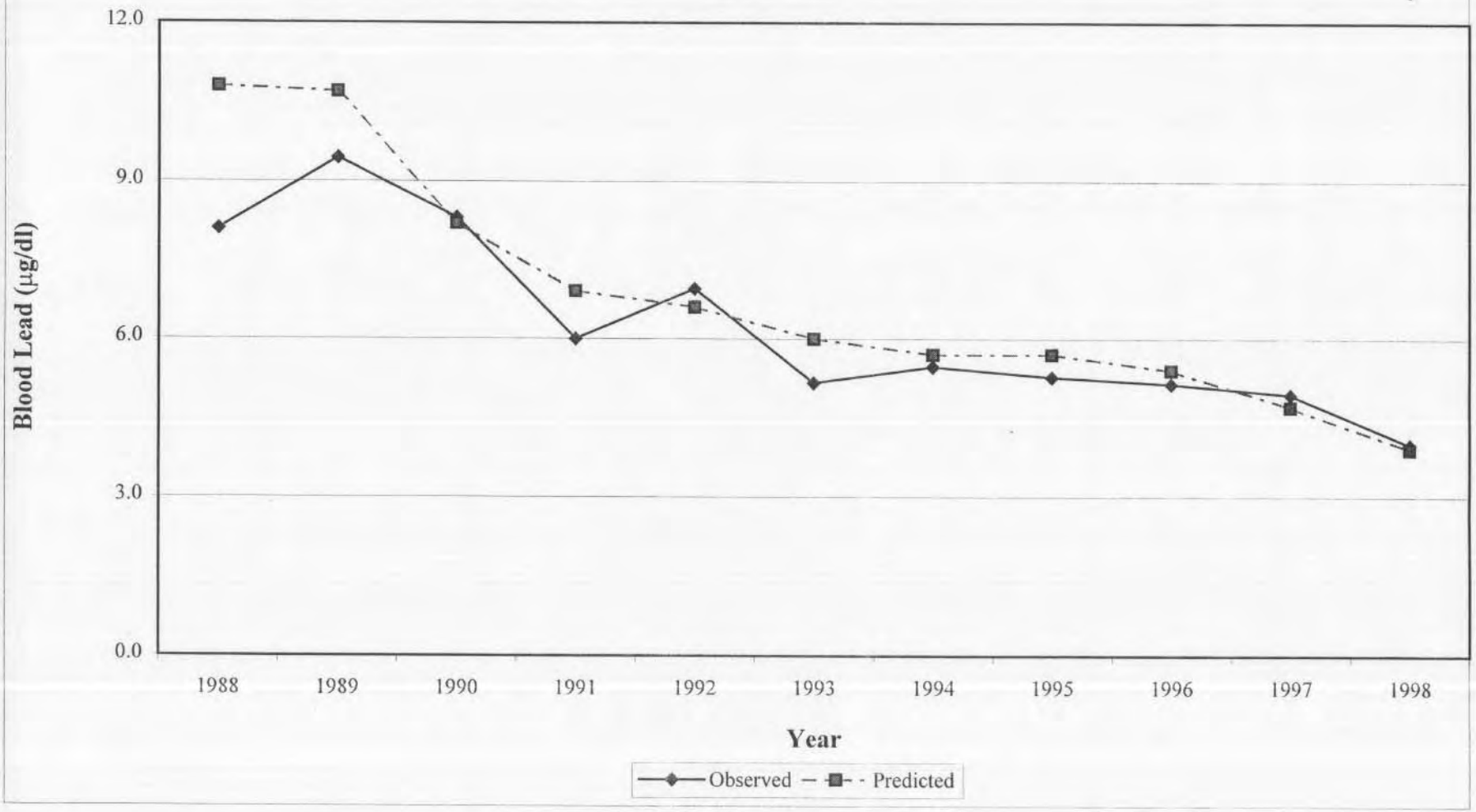


Figure Q4-15b
Predicted and Observed Blood Lead in 0-84 Month Old Children
42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability
Smelterville 1988-1998

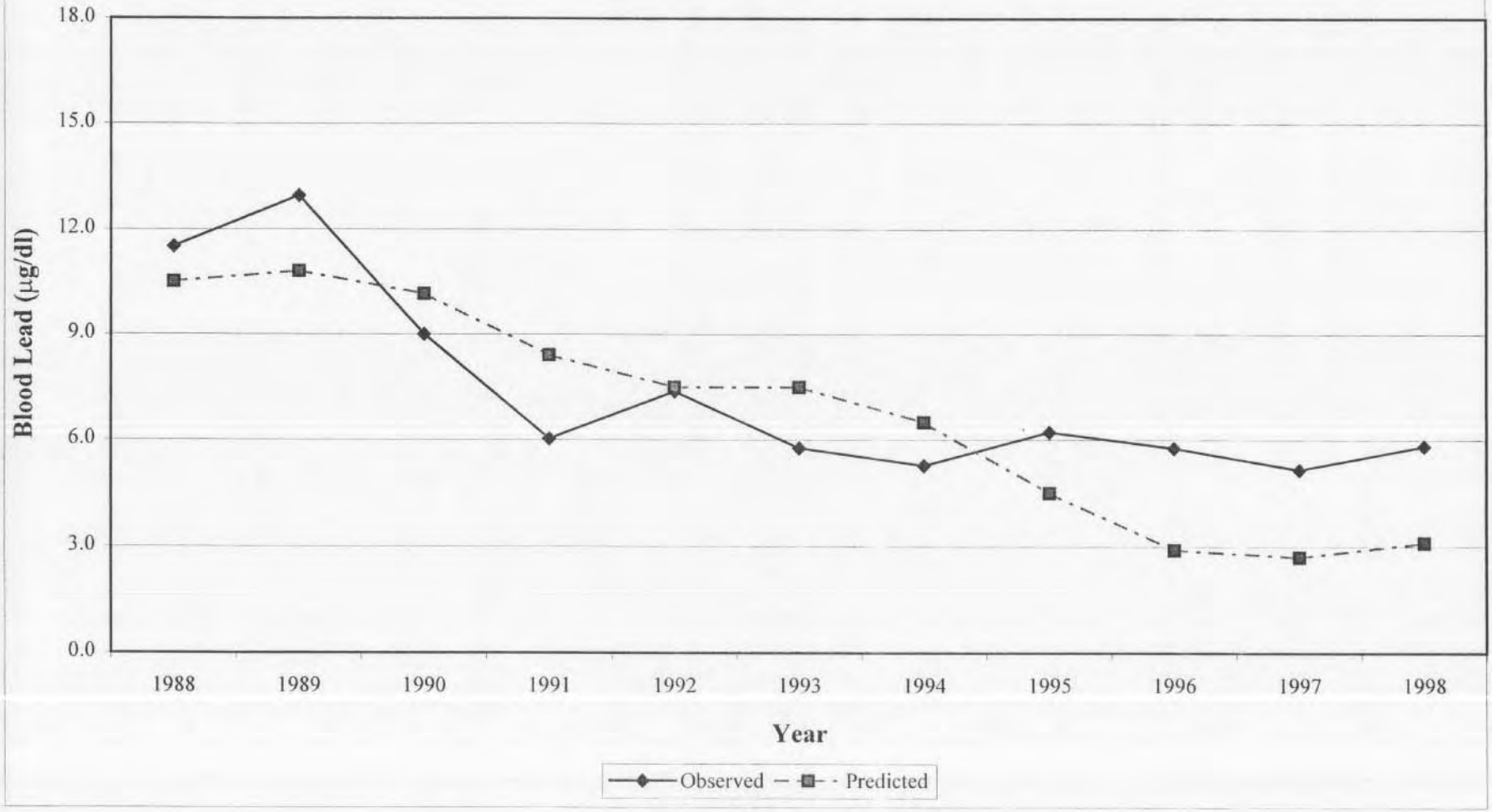


Figure Q4-15c
Predicted and Observed Blood Lead in 0-84 Month Old Children
42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability
Site Wide 1988-1998

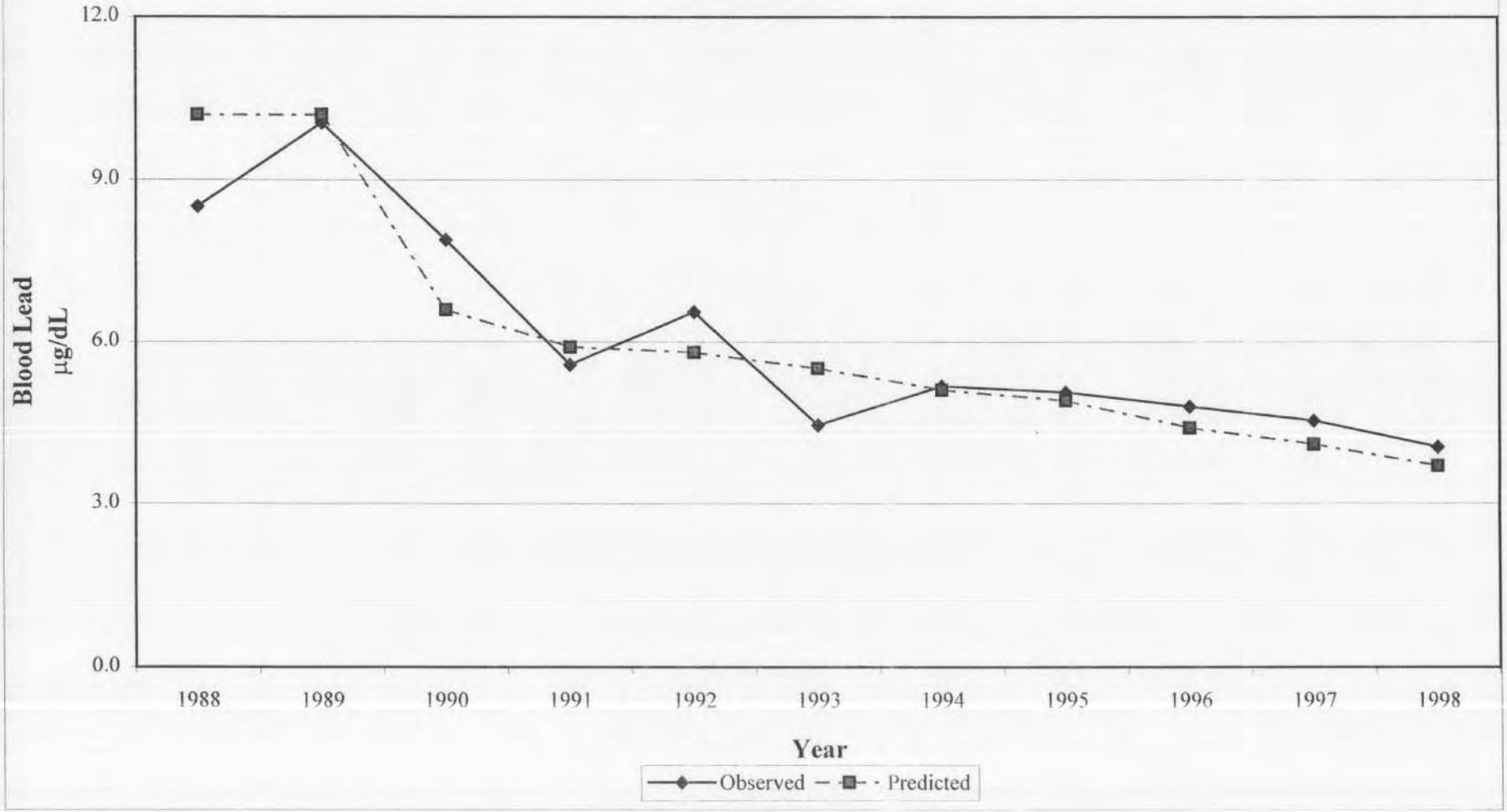


Figure Q4-15d
Predicted and Observed Blood Lead in Two Year Old Children
42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability
Kellogg 1988-1998

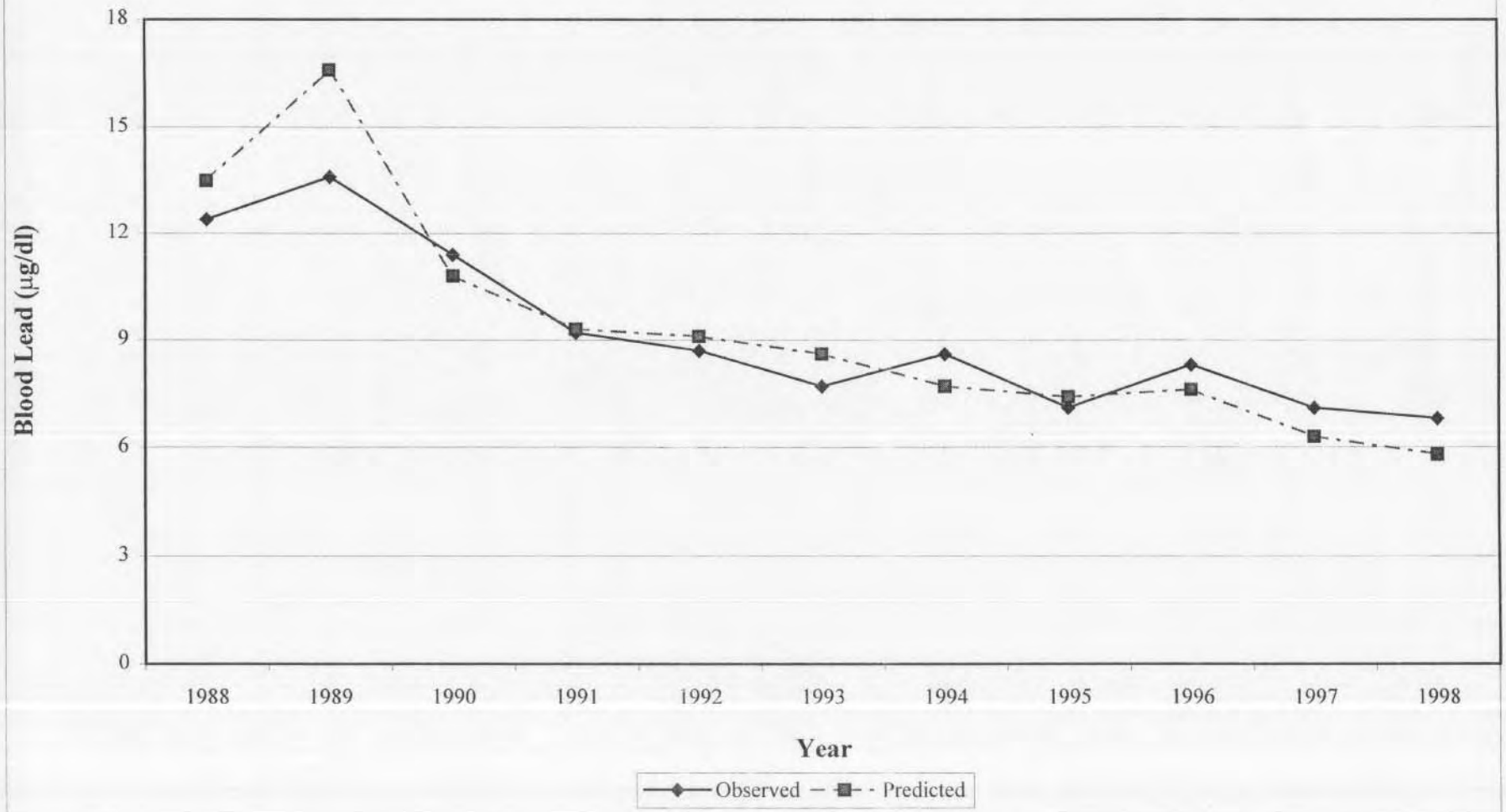


Figure Q4-15e
Predicted and Observed Blood Lead in Two Year Old Children
42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability
Smelterville 1988-1998

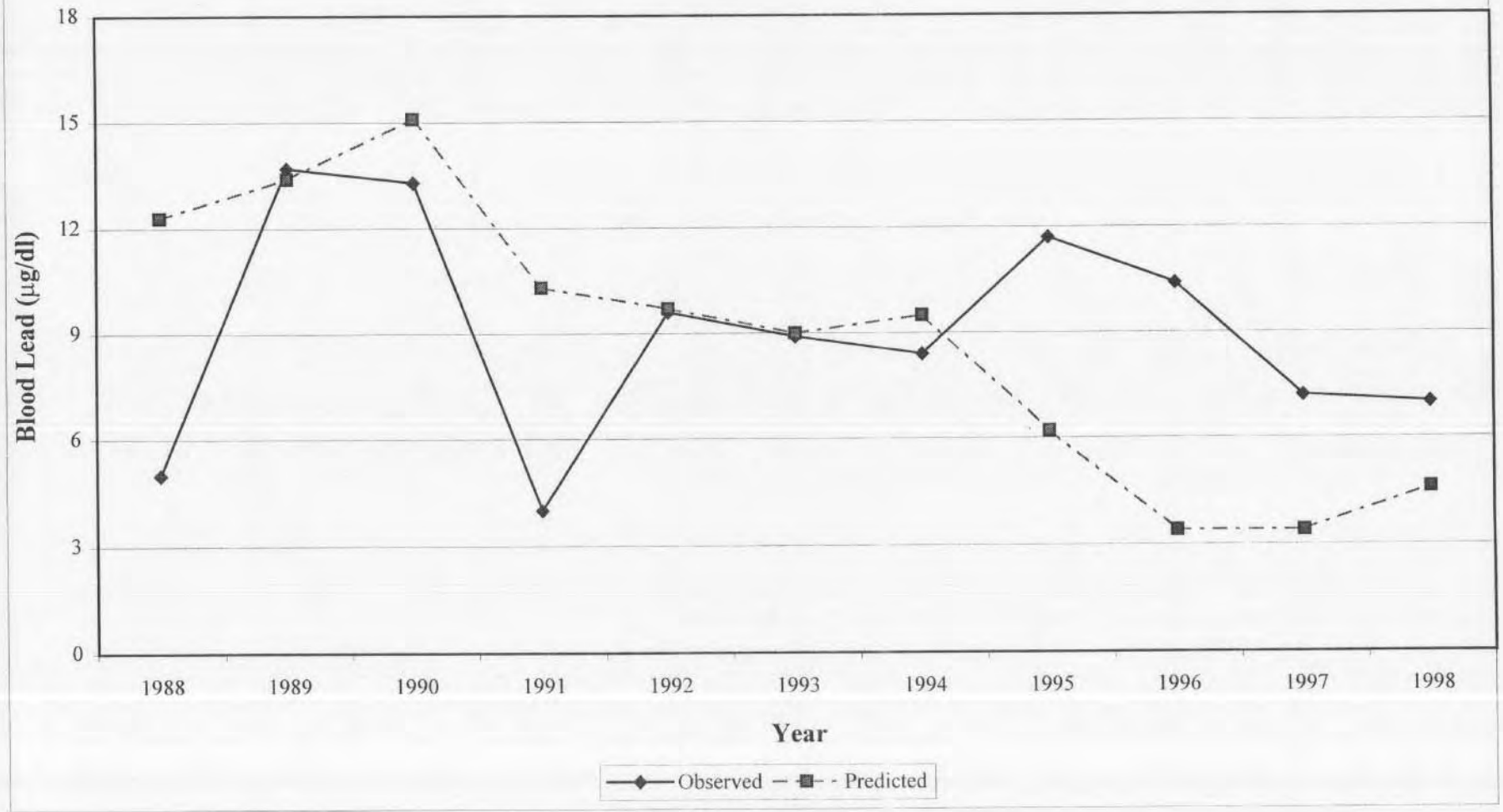


Figure Q4-15f
Predicted and Observed Blood Lead in Two Year Old Children
42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability
Site Wide 1988-1998

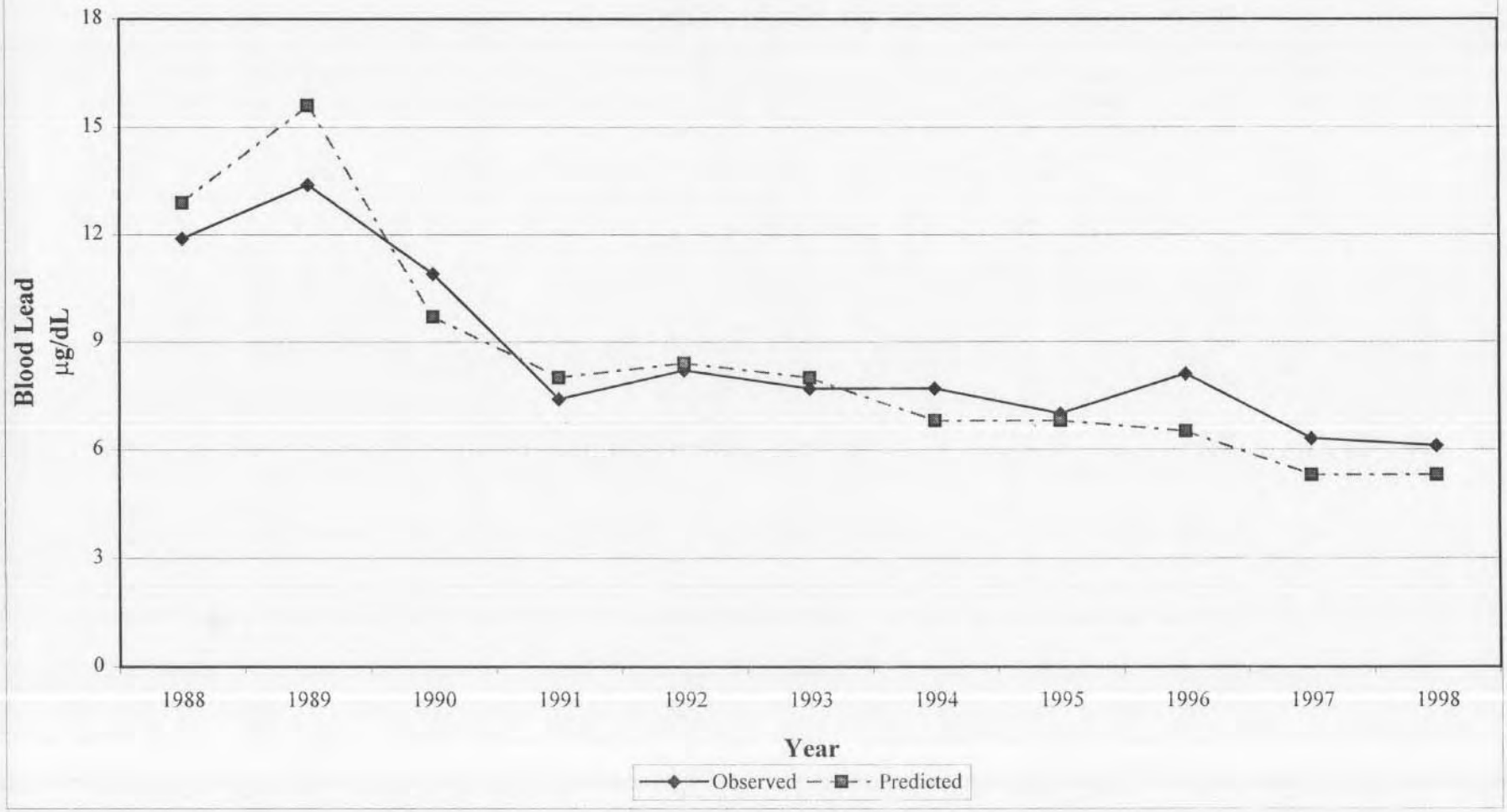


Figure Q4-16a
Predicted and Observed Geometric Standard Deviation
42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability
Kellogg 1988-1998

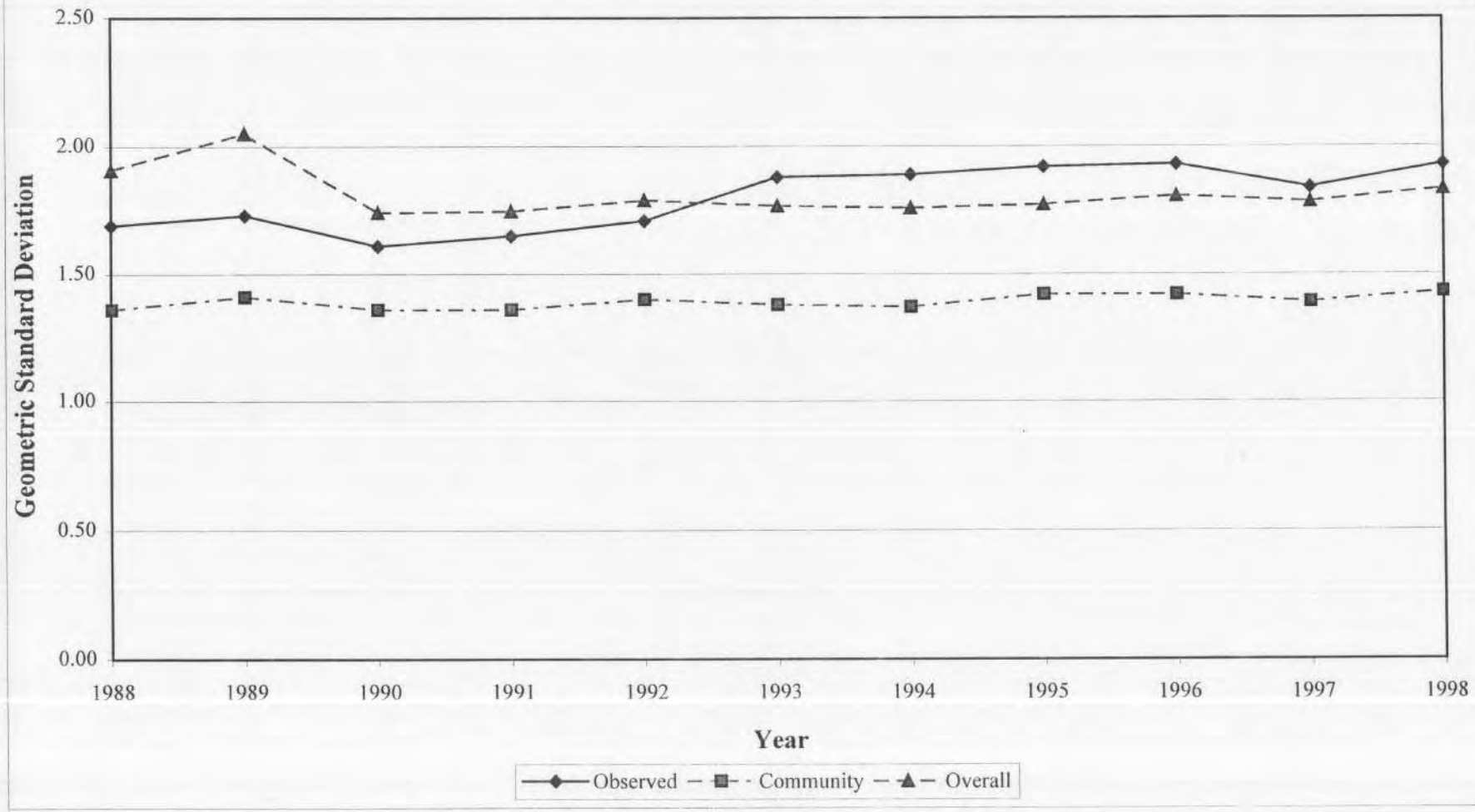


Figure Q4-16b
Predicted and Observed Geometric Standard Deviation
42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability
Smelterville 1988-1998

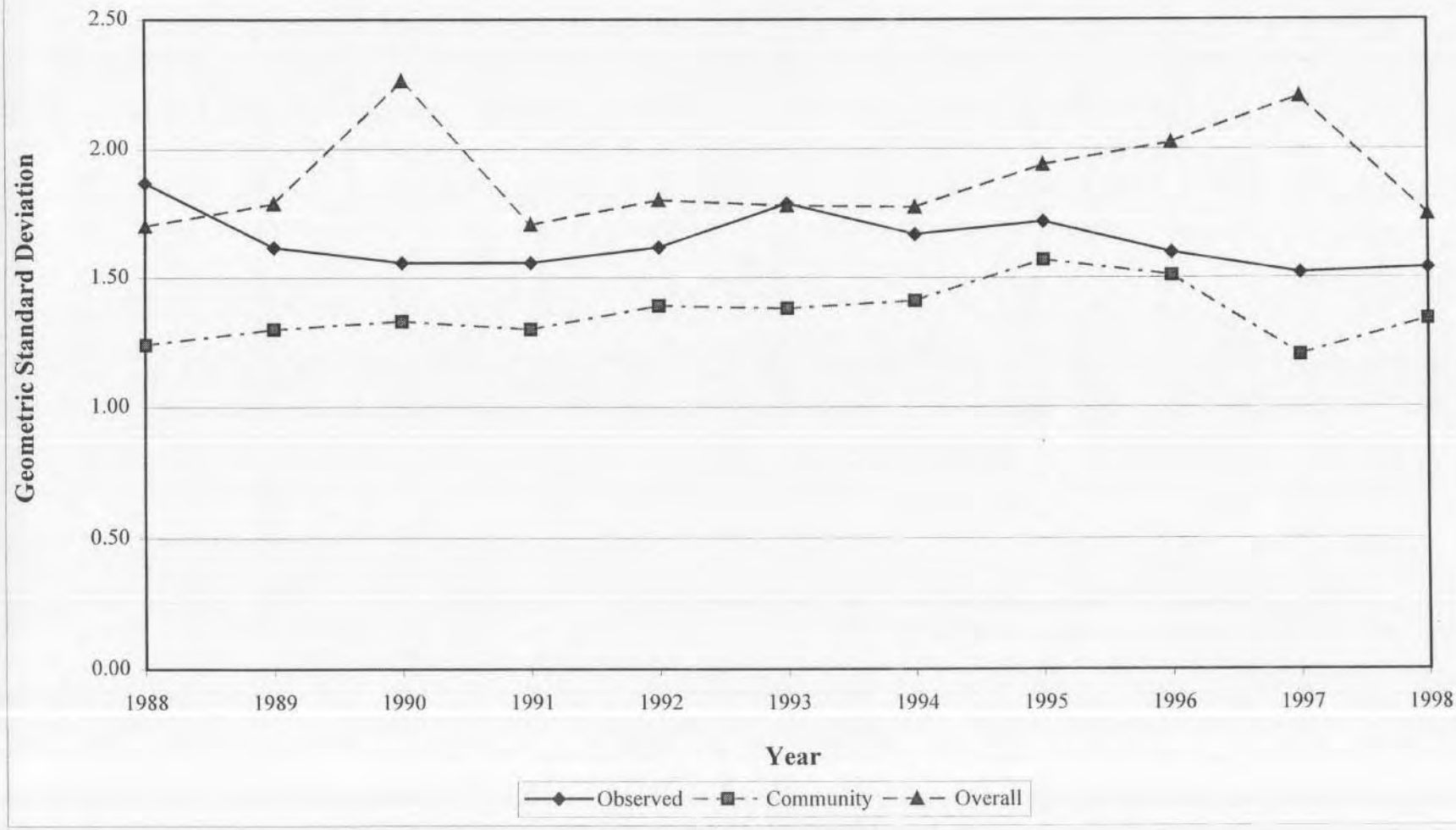


Figure Q4-16c
Predicted and Observed Geometric Standard Deviation
42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability
Site Wide 1988-1998

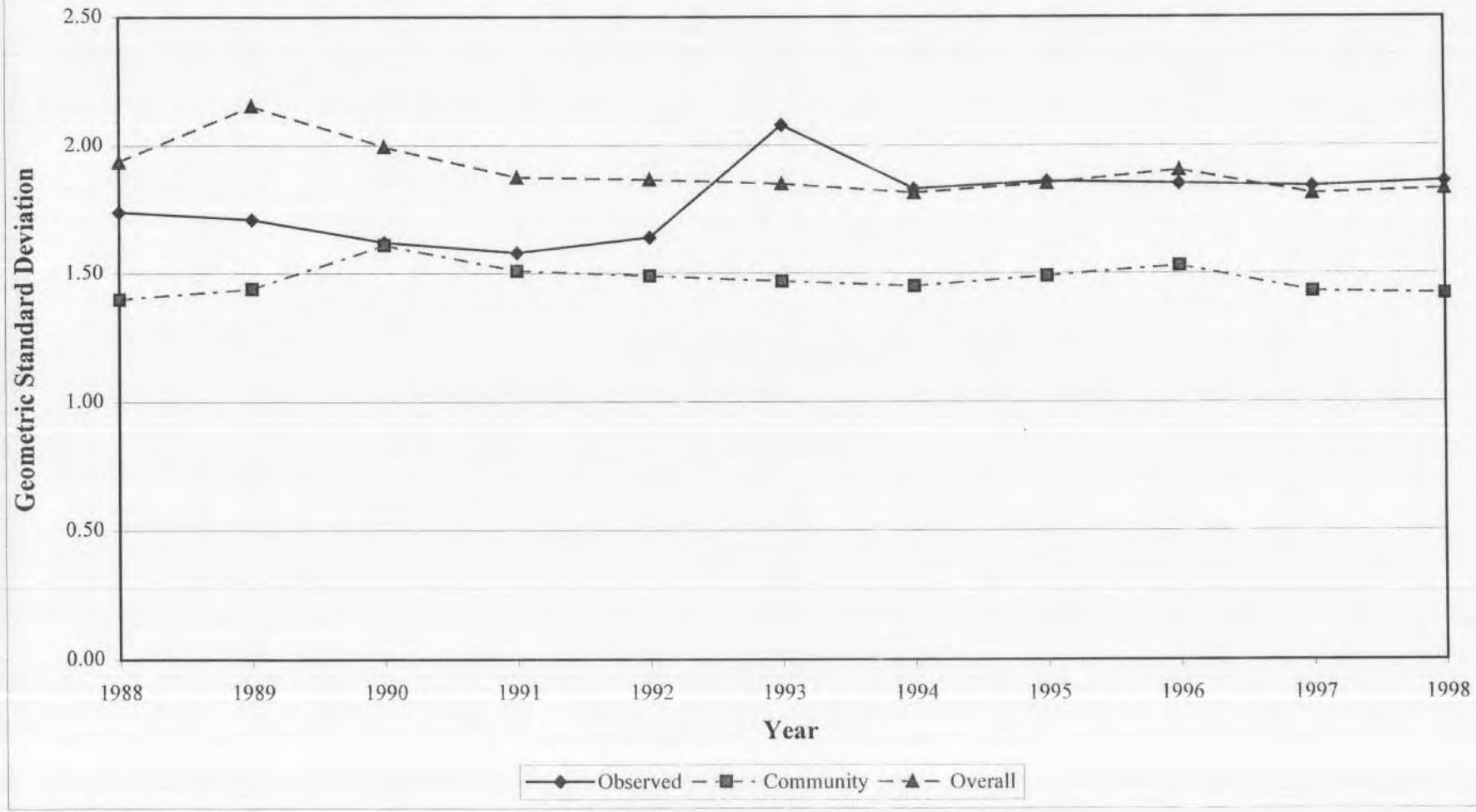


Figure Q4-17a
Predicted and Observed Percent of 0-84 Month Old Children with Blood Lead Level Exceeding 10 μg/dl 42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability
Kellogg 1988-1998

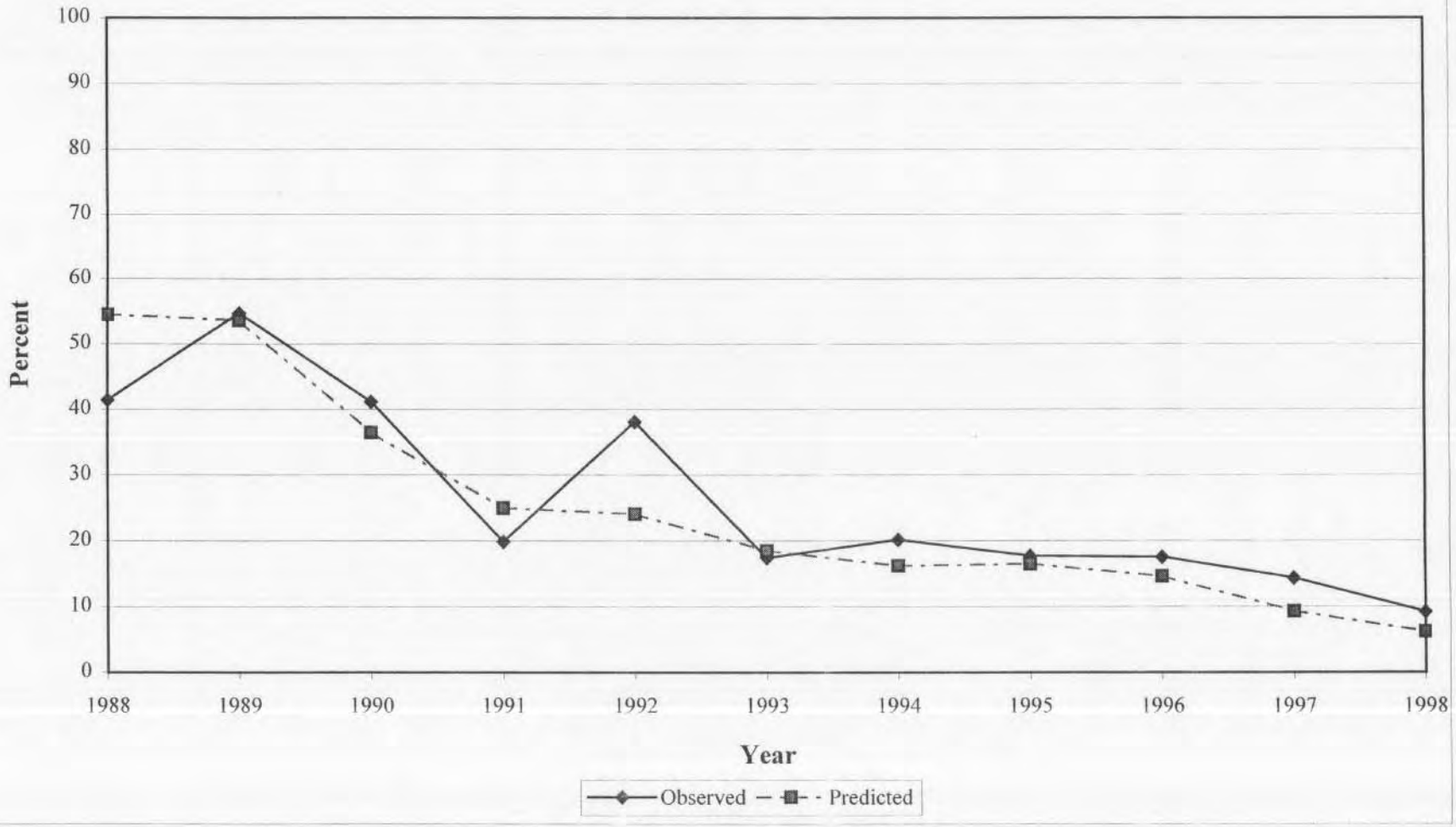


Figure Q4-17b
Predicted and Observed Percent of 0-84 Month Old Children with Blood Lead Level Exceeding 10 μg/dl, 42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability
Smelterville 1988-1998

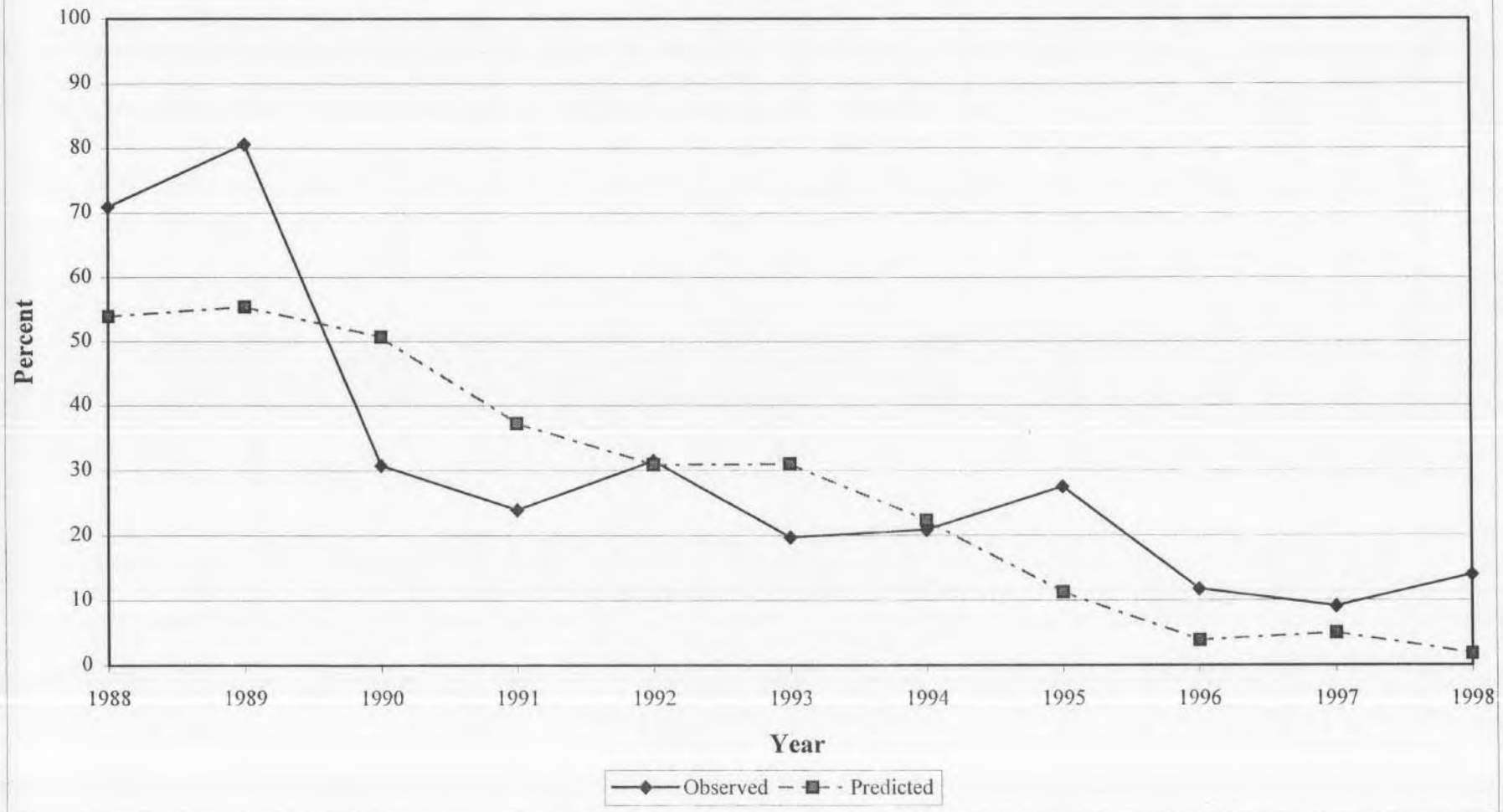


Figure Q4-17c
Predicted and Observed Percent of 0-84 Month Old Children with Blood Lead Level Exceeding 10 μg/dl, 42:27:19:12 Dust:Soil Partition Scenarios at 18% Bioavailability Site Wide 1988-1998

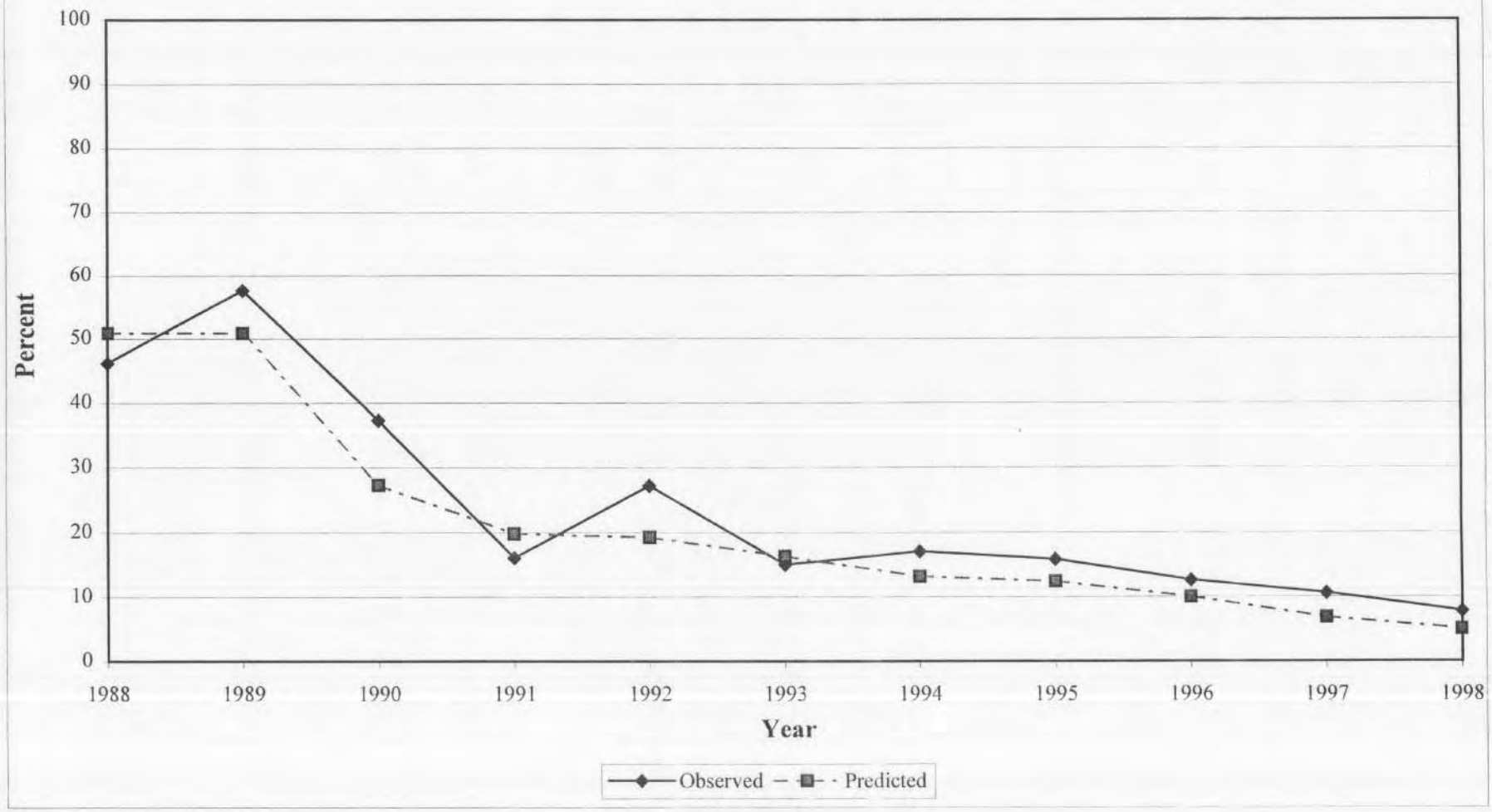


Figure Q4-18a
Observed vs. Predicted Arithmetic Mean Blood Lead Levels
Default Scenario at 30% Bioavailability
Site Wide 1988-1998

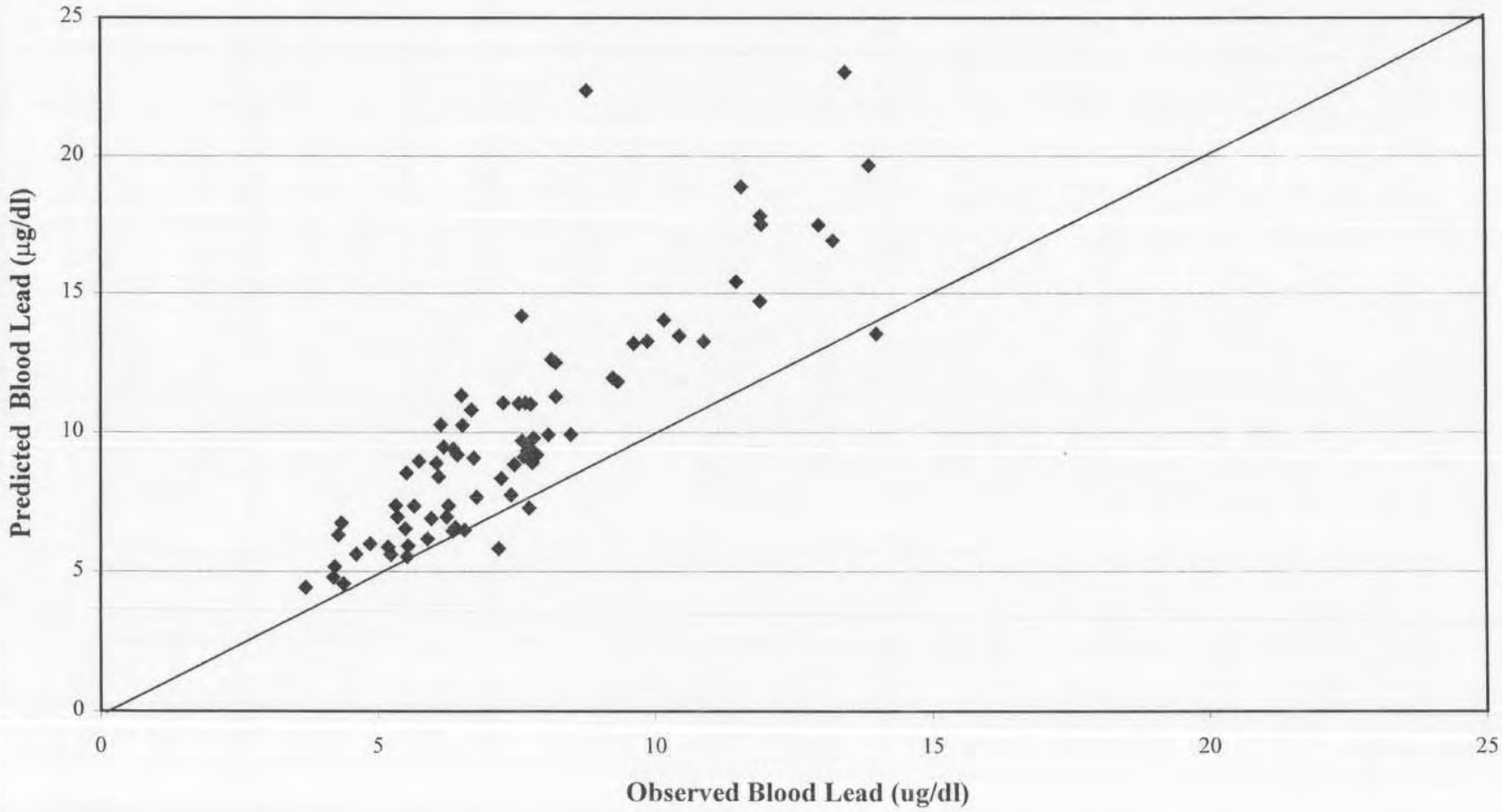


Figure Q4-18b
Observed vs Predicted Arithmetic Mean Blood Lead Levels
42:27:19:12 Dust:Soil Partition Scenario at 18% Bioavailability
Site Wide 1988-1998

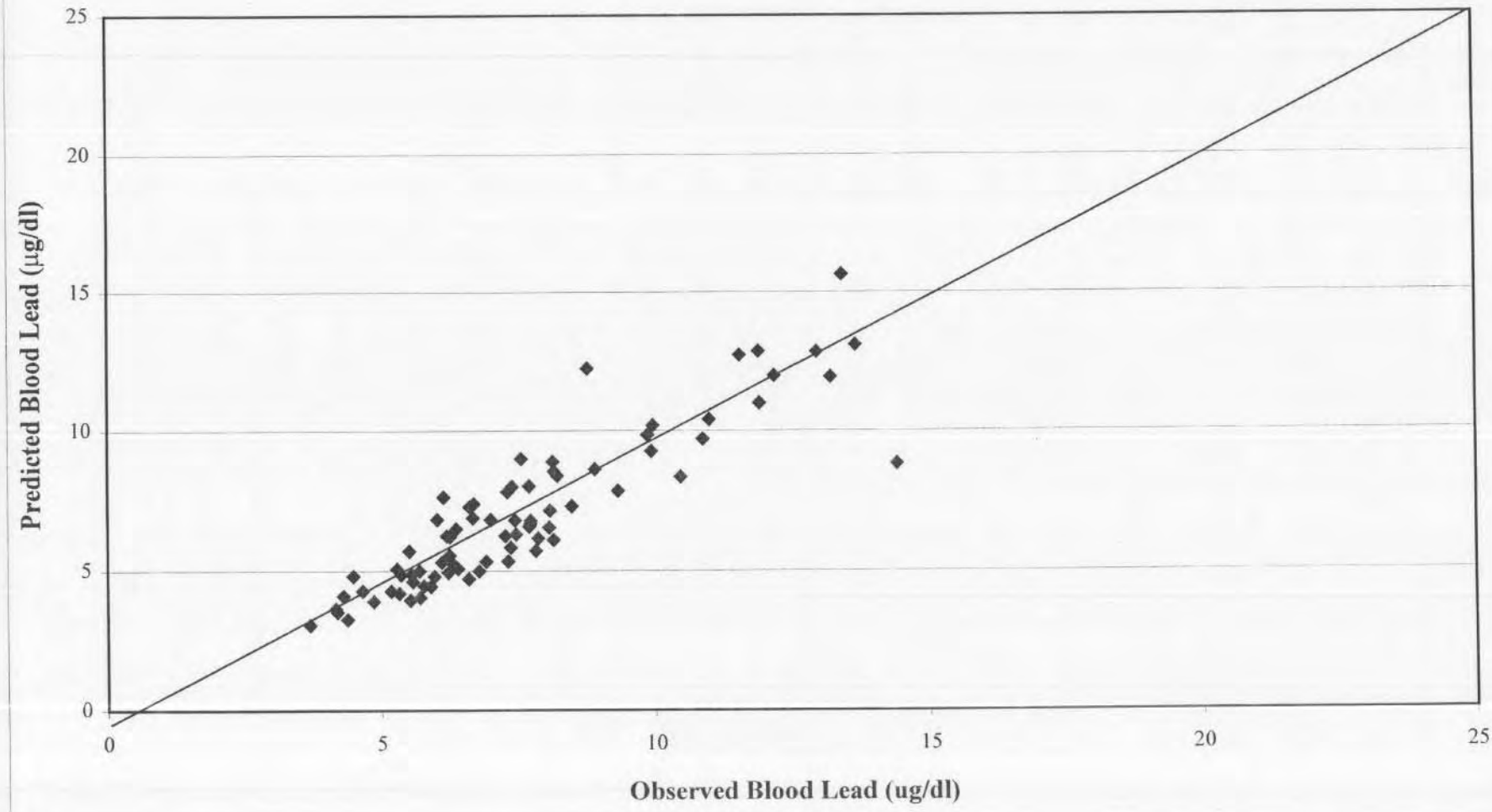


Figure Q4-19a
Observed vs. Predicted Geometric Mean Blood Lead Levels
Default Scenario at 30% Bioavailability
Site Wide 1988-1998

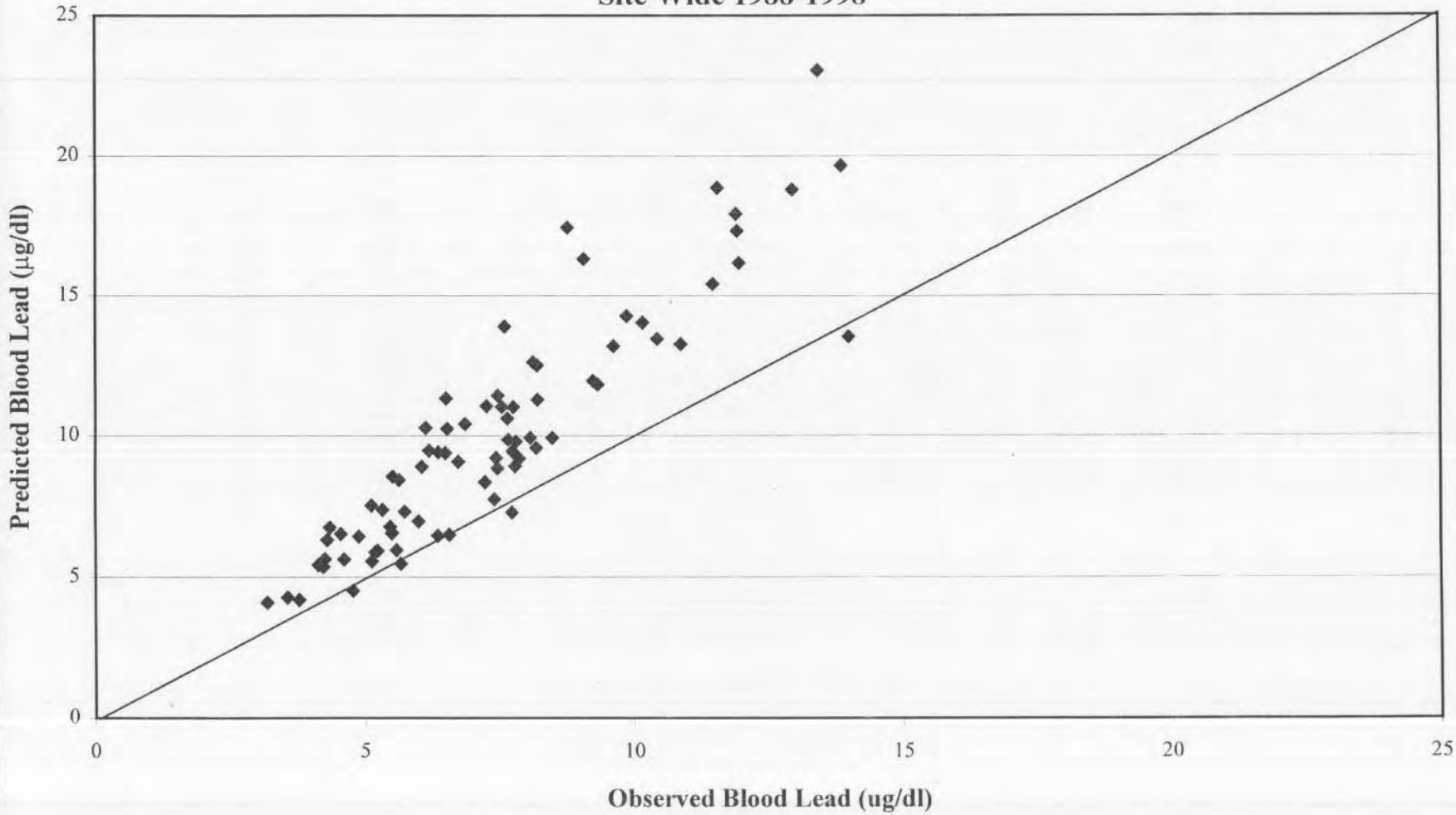


Figure Q4-19b
Observed vs. Predicted Geometric Mean Blood Lead Levels
42:27:19:12 Dust:Soil Partition Scenario at 18% Bioavailability
Site Wide 1988-1998

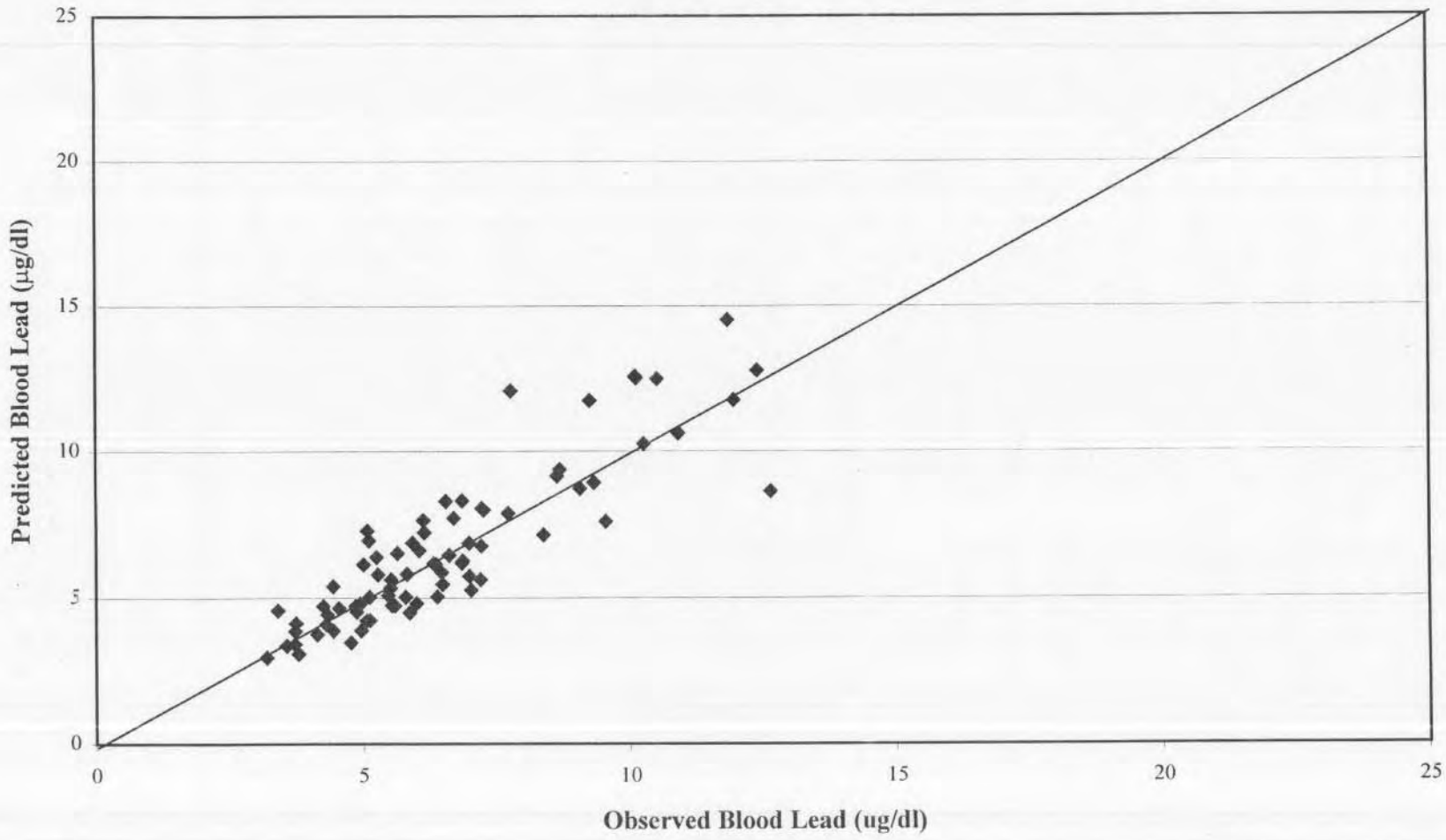


Figure Q4-20
Estimated Bioavailability Trends, 1988-1998

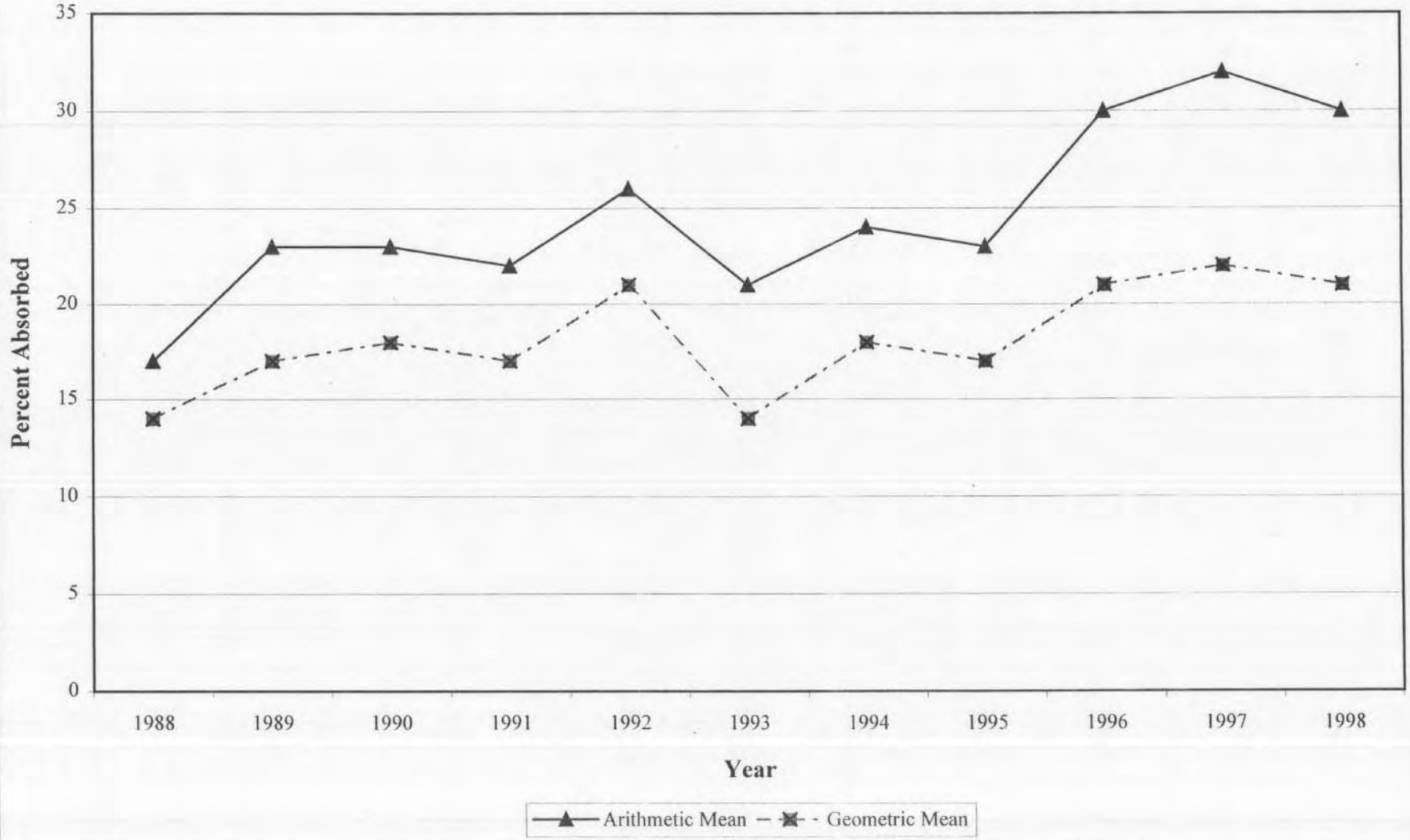


Table Q4.29
Estimated Aggregate Soil and Dust Effective Bioavailability 1988-1998

Year	Bioavailability	
	Arithmetic Mean	Geometric Mean
1988	17	14
1989	23	17
1990	23	18
1991	22	17
1992	26	21
1993	21	14
1994	24	18
1995	23	17
1996	30	21
1997	32	22
1998	30	21

Table Q4.30 Predicted and Observed Geometric Mean Blood Lead Levels, Geometric Standard Deviations, and Toxicity for 0-84 Month Old Children 42:27:19:12 Dust:Soil Partition at 18% Bioavailability

	Mean Blood Lead Levels for Two Year Old Children (μg/dl)		Mean Blood Lead Levels 0-9 Year Old Children (μg/dl)		Geometric Standard Deviations			ercent of Children wit Blood Lead ≥ 10 μg/dl	
Year	Observed	Predicted	Observed	Predicted	Observed	Community	Overall	Observed	Predicted
Kellogg									
1988	12.4	13.5	8.1	10.8	1.69	1.36	1.91	41.4	54.5
1989	13.6	16.6	9.5	10.7	1.73	1.41	2.05	54.7	53.6
1990	11.4	10.8	8.3	8.2	1.61	1.36	1.74	41.1	36.4
1991	9.2	9.3	6.0	6.9	1.65	1.36	1.75	19.9	25.0
1992	8.7	9.1	7.0	6.6	1.71	1.40	1.79	38.1	24.1
1993	7.7	8.6	5.2	6.0	1.88	1.38	1.77	17.4	18.5
1994	8.6	7.7	5.5	5.7	1.89	1.37	1.76	20.2	16.2
1995	7.1	7.4	5.3	5.7	1.92	1.42	1.77	17.7	16.5
1996	8.3	7.6	5.1	5.4	1.93	1.42	1.81	17.6	14.6
1997	7.1	6.3	4.9	4.7	1.84	1.39	1.78	14.4	9.3
1998	6.8	5.8	4.0	3.9	1.93	1.43	1.83	9.2	6.2
Smelterville									
1988	5	12.3	11.5	10.5	1.87	1.24	1.70	71.0	53.9
1989	13.7	13.4	13.0	10.8	1.62	1.30	1.79	80.6	55.4
1990	13.3	15.1	9.0	10.2	1.56	1.33	2.27	30.8	50.7
1991	4	10.3	6.0	8.4	1.56	1.30	1.71	23.9	37.2
1992	9.6	9.7	7.4	7.5	1.62	1.39	1.80	31.5	30.9
1993	8.9	9	5.8	7.5	1.79	1.38	1.78	19.7	31.0
1994	8.4	9.5	5.3	6.5	1.67	1.41	1.78	20.8	22.3
1995	11.7	6.2	6.2	4.5	1.72	1.57	1.94	27.5	11.3
1996	10.4	3.4	5.8	2.9	1.60	1.51	2.03	11.8	3.9
1997	7.2	3.4	5.2	2.7	1.52	1.20	2.20	9.1	5.0
1998	7	4.6	5.8	3.1	1.54	1.34	1.75	14.0	1.8
Site Wide									
1988	11.9	12.9	8.5	10.2	1.74	1.40	1.94	46.2	50.9
1989	13.4	15.6	10.1	10.2	1.71	1.44	2.16	57.7	50.9
1990	10.9	9.7	7.9	6.6	1.62	1.61	1.99	37.3	27.3
1991	7.4	8	5.6	5.9	1.58	1.51	1.87	16.1	19.8
1992	8.2	8.4	6.6	5.8	1.64	1.49	1.87	27.2	19.3
1993	7.7	8	4.5	5.5	2.08	1.47	1.85	15.0	16.3
1994	7.7	6.8	5.2	5.1	1.83	1.45	1.81	17.1	13.2
1995	7	6.8	5.1	4.9	1.86	1.49	1.85	15.9	12.4
1996	8.1	6.5	4.8	4.4	1.85	1.53	1.90	12.6	10.0
1997	6.3	5.3	4.5	4.1	1.84	1.43	1.81	10.6	6.8
1998	6.1	5.3	4.1	3.7	1.86	1.42	1.83	7.8	5.0

APPENDIX R

Observed and Predicted Blood Lead Levels by Age, Area, Dust Concentration and Action Criteria

Appendix R

Observed and Predicted Blood Lead Levels by Age, Area, Dust Concentration and Action Criteria

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This appendix contains figures and tables related to Chapter 6. Included in this appendix are the following:

Figure R-1a - Observed and Predicted Blood Lead Levels for 0-84 Month Old Children – IEUBK Community Mode

Figure R-1b - Observed and Predicted Blood Lead Levels for 0-60 Month Old Children – IEUBK Community Mode

Figure R-1c - Observed and Predicted Blood Lead Levels for 9-24 Month Old Children – IEUBK Community Mode

Figure R-2a - Observed and Predicted Percent to Exceed Levels for 0-84 Month Old Children – IEUBK Community Mode

Figure R-2b - Observed and Predicted Percent to Exceed Levels for 0-60 Month Old Children – IEUBK Community Mode

Figure R-2c - Observed and Predicted Percent to Exceed Levels for 9-24 Month Old Children – IEUBK Community Mode

Figures R-3a-f - Estimated Percent of Children to Exceed 10 μg/dl Blood Lead Associated with Various Yard Soil Cleanup Action Criteria Using Different Dust Concentrations by geographic area (similar to Figures 6-20a-b)

Table R-1 - Estimated EPA Default Model Blood Lead Levels Associated With Various Yard Soil Cleanup Action Criteria

Table R-2 - Estimated Box Model Blood Lead Levels Associated With Various Yard Soil Cleanup Action Criteria

Table R-3 - Estimated Community Blood Lead Levels and Health Risk Associated With Various Yard Soil Cleanup Action Criteria

Table R-4 - Individual Blood Lead Levels and Health Risk at Potential Action Levels

Figure R-1a Observed and Predicted Blood Lead Levels for 0-84 Month Old Children-IEUBK Community Mode

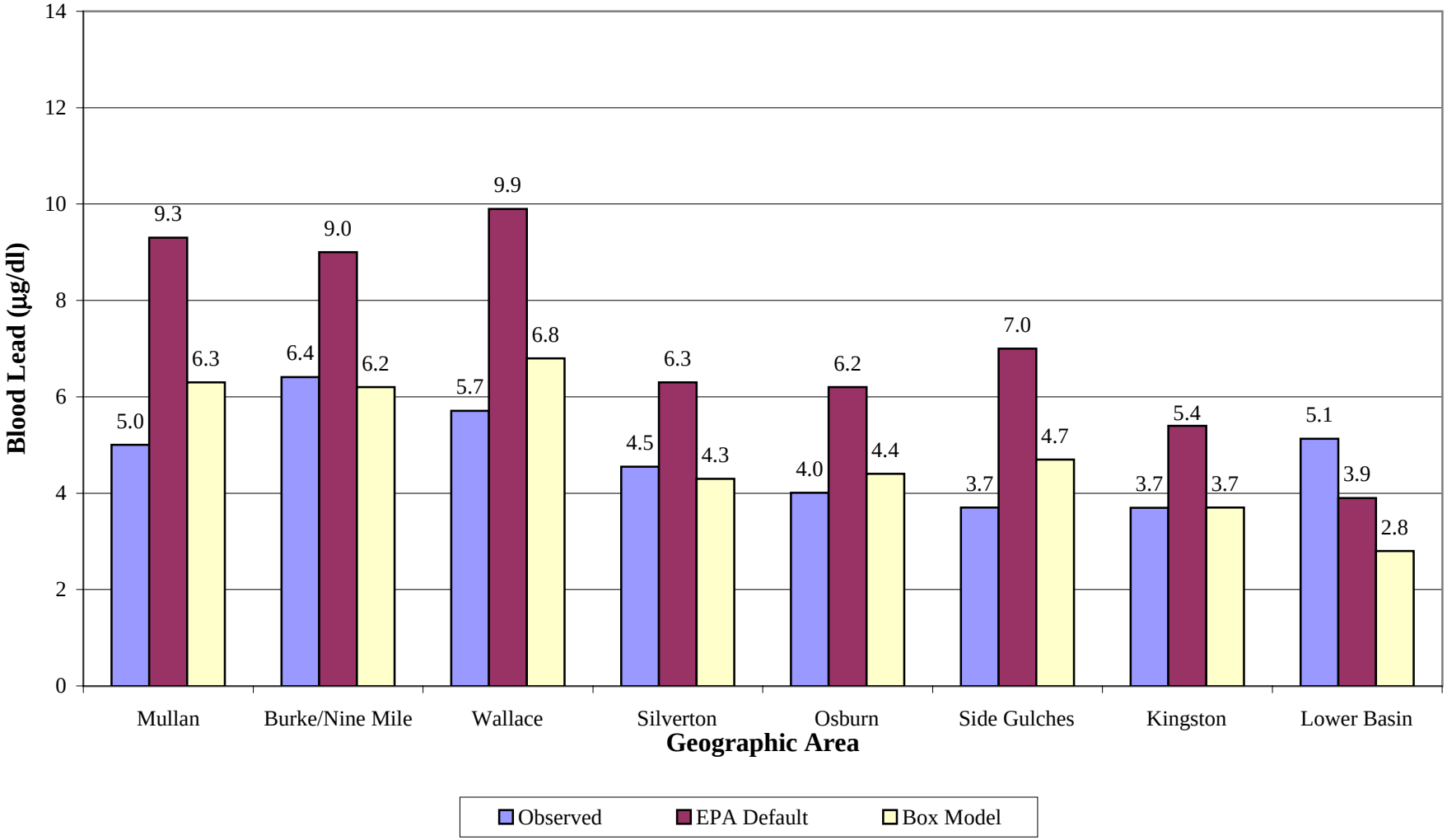


Figure R-1b Observed and Predicted Blood Lead Levels for 0-60 Month Old Children-IEUBK Community Mode

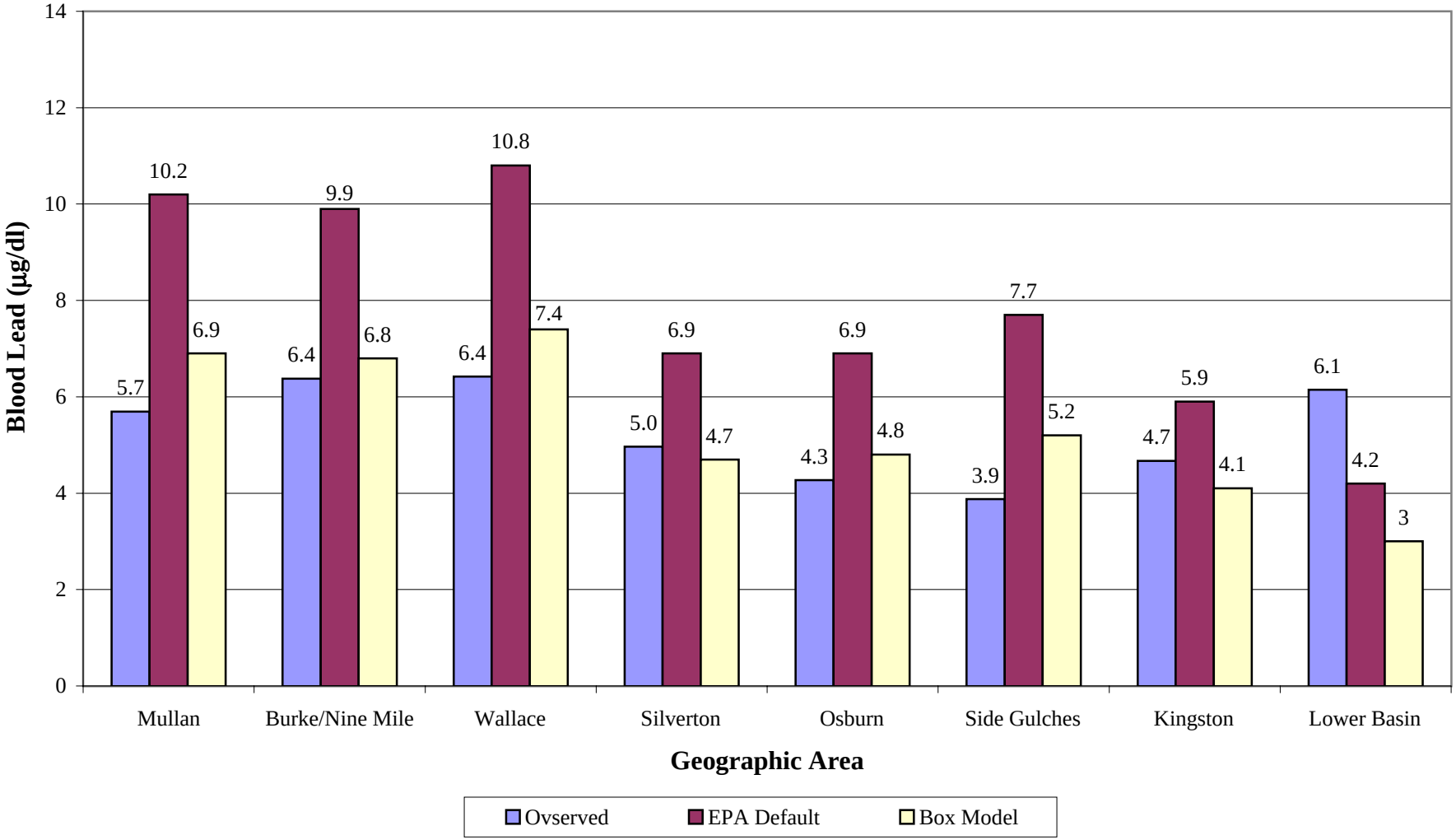


Figure R-1c Observed and Predicted Blood Lead Levels for 9-24 Month Old Children-IEUBK Community Mode

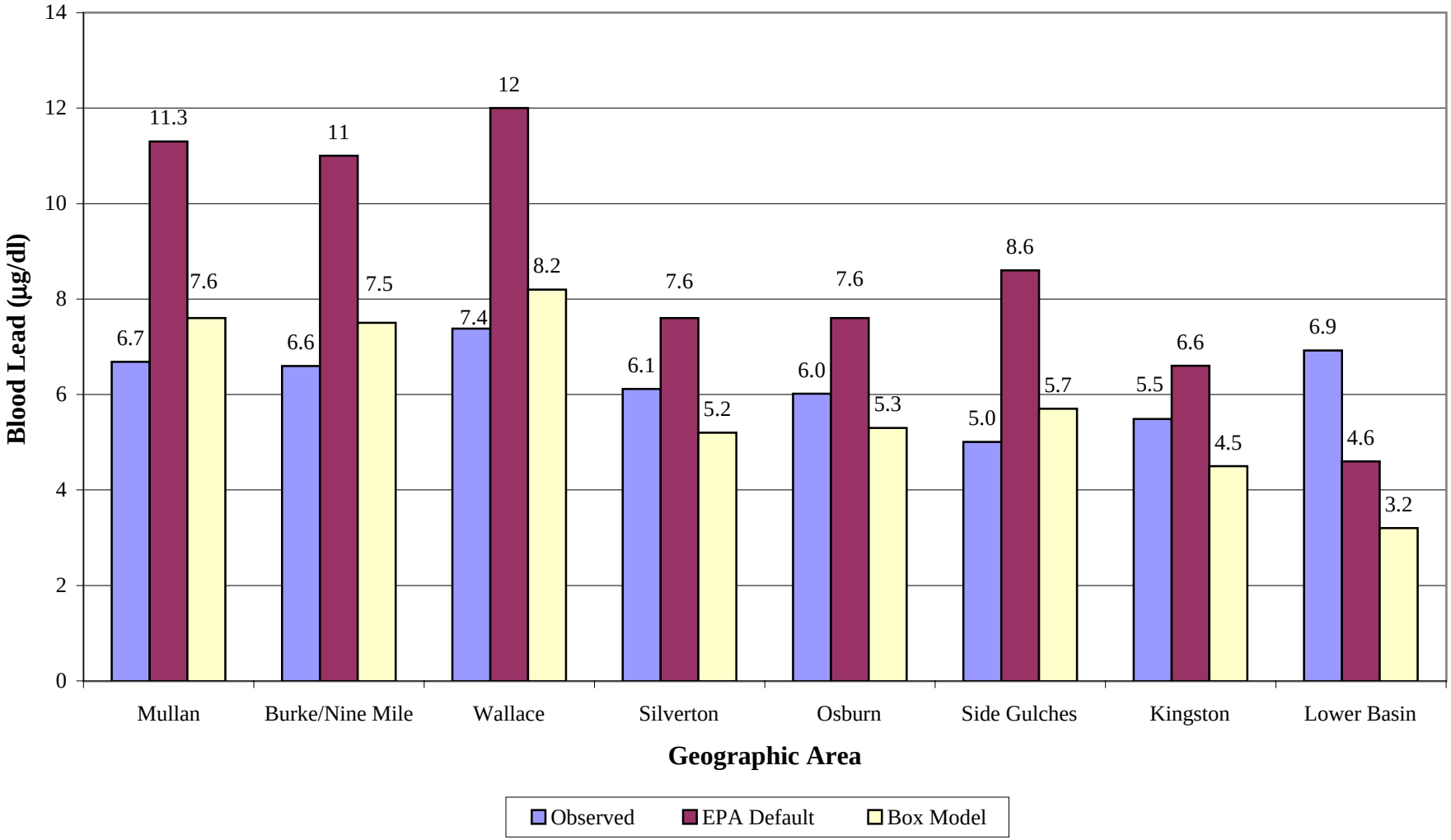


Figure R-2a Observed and Predicted Percent to Exceed Levels for 0-84 Month Old Children-IEUBK Community Mode

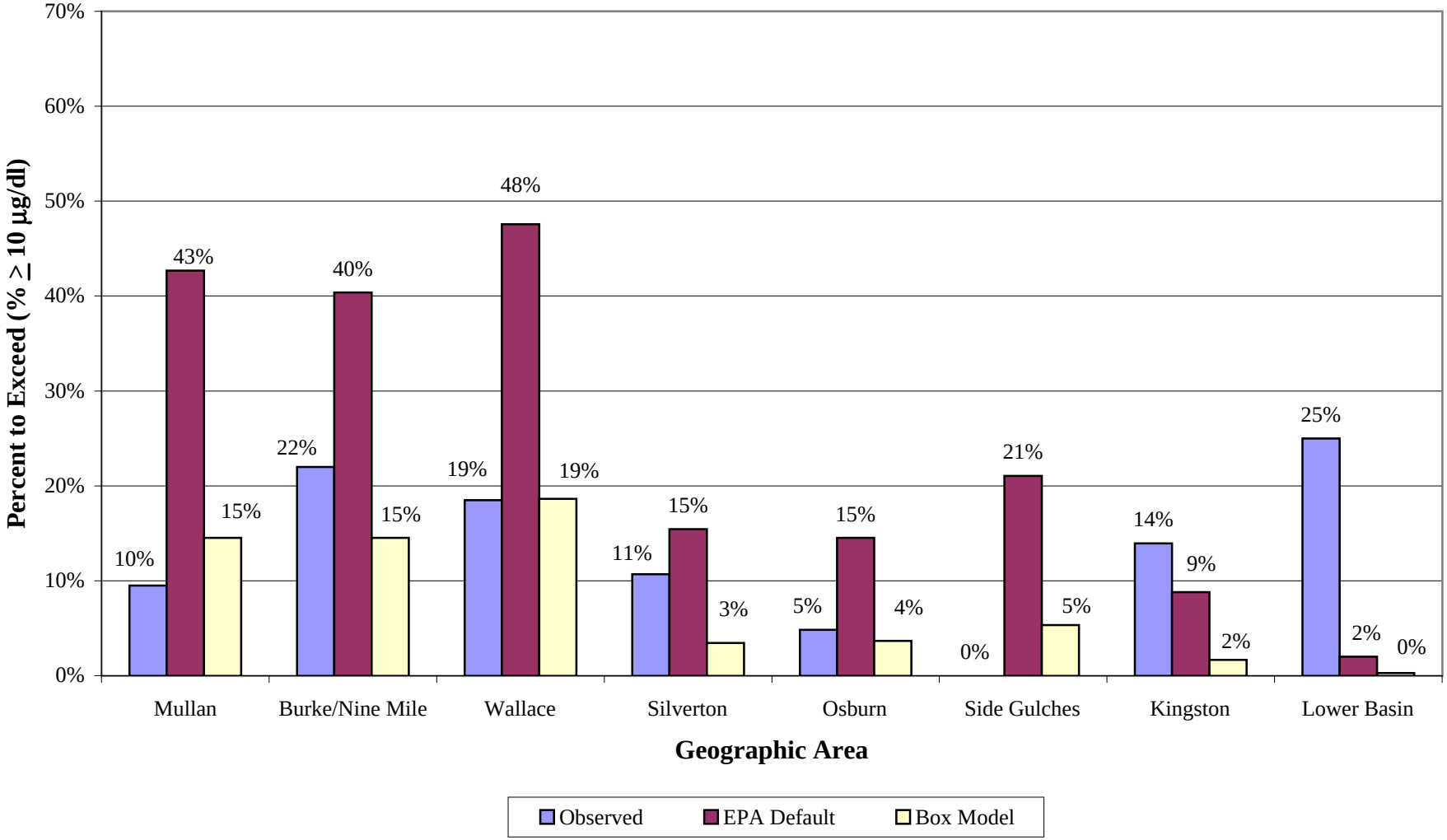


Figure R-2b Observed and Predicted Percent to Exceed Levels for 0-60 Month Old Children-IEUBK Community Mode

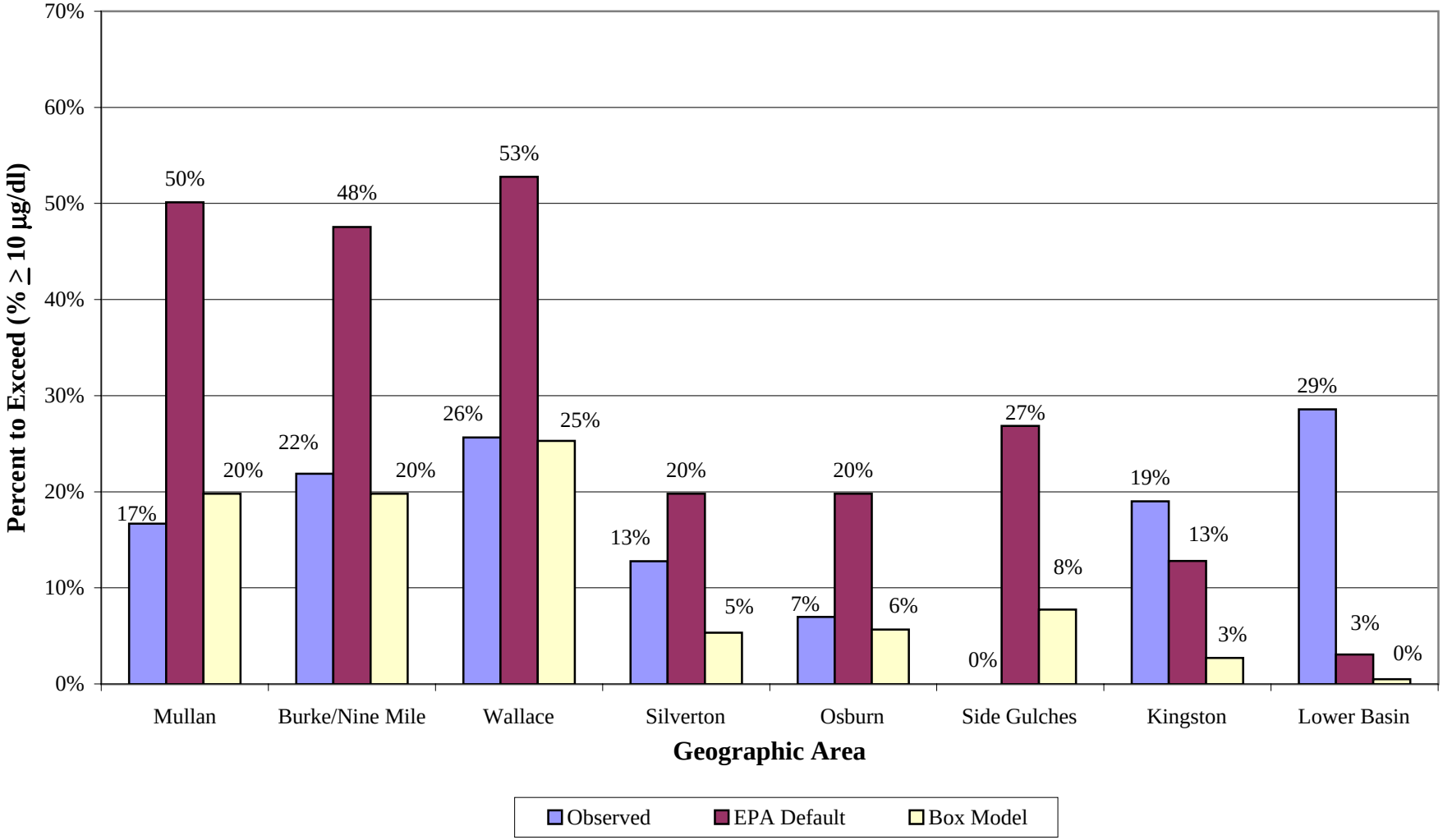


Figure R-2c Observed and Predicted Percent to Exceed Levels for 9-24 Month Old Children-IEUBK Community Mode

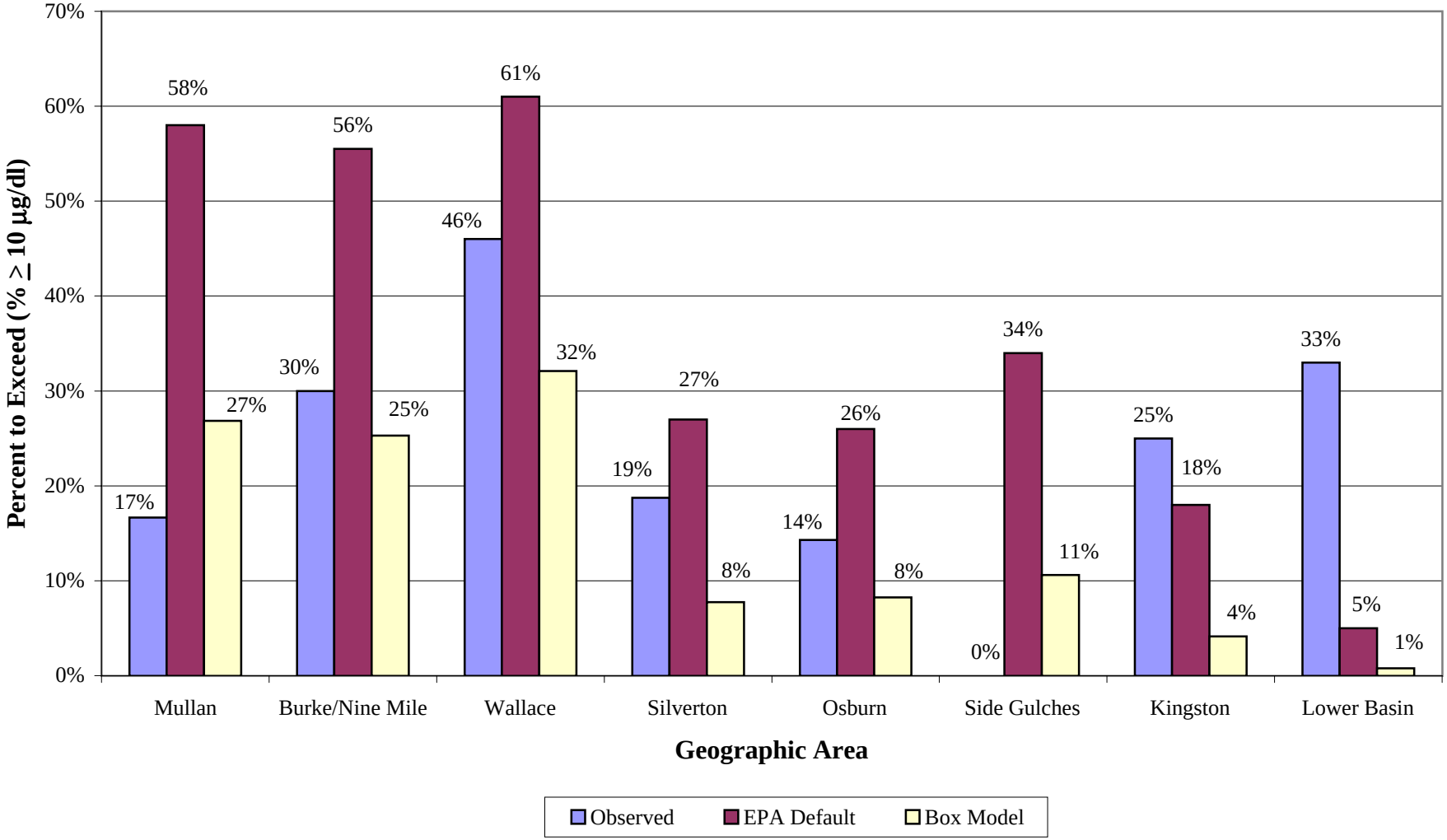


Figure R-3a Estimated Percent of Children to Exceed 10 μg/dl Blood Lead Associated with Various Yard Soil Cleanup Action Criteria Using Different Dust Concentrations - Mullan

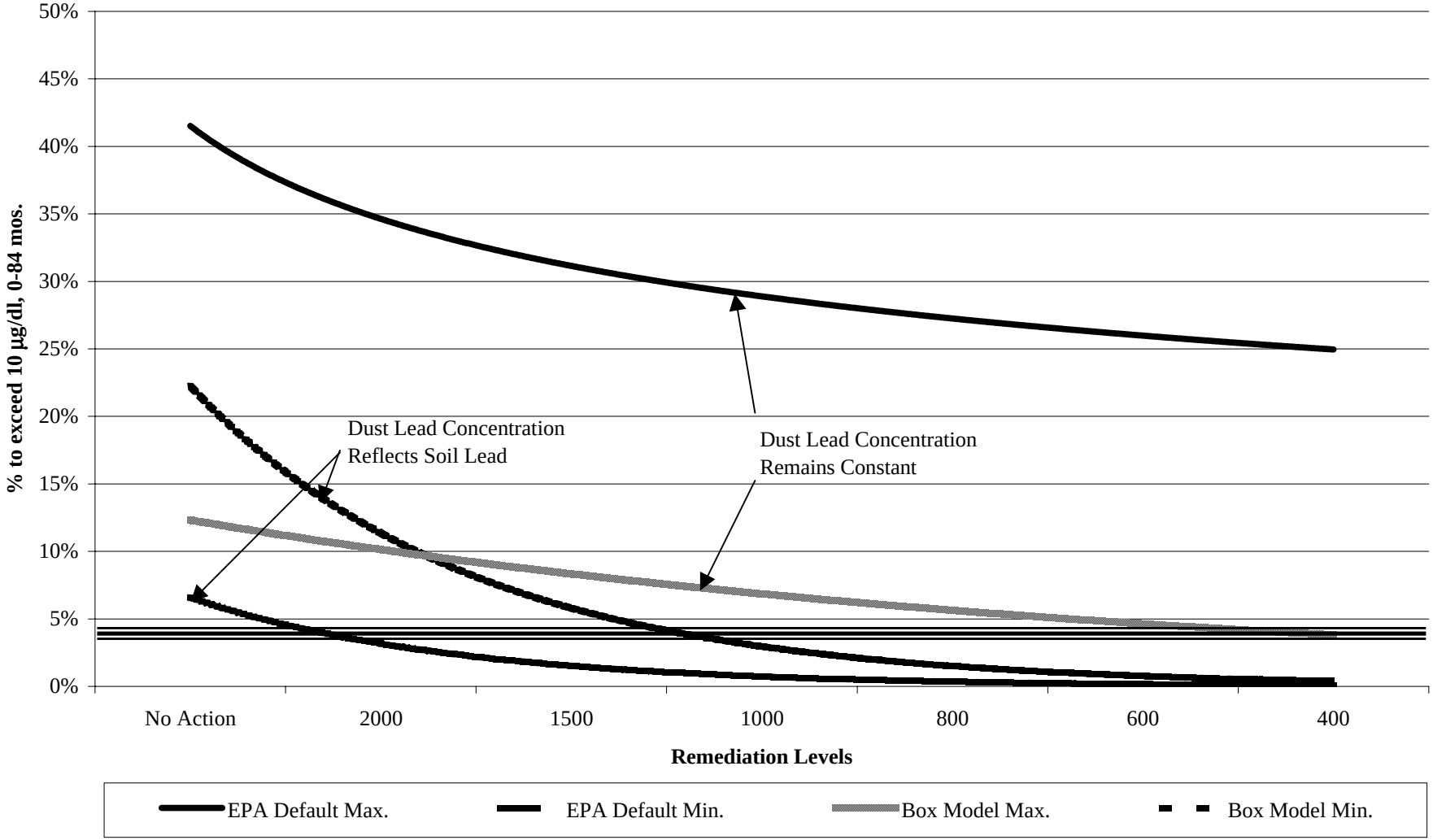


Figure R-3b Estimated Percent of Children to Exceed 10 µg/dl Blood Lead Associated with Various Yard Soil Cleanup Action Criteria Using Different Dust Concentrations - Burke/Nine Mile

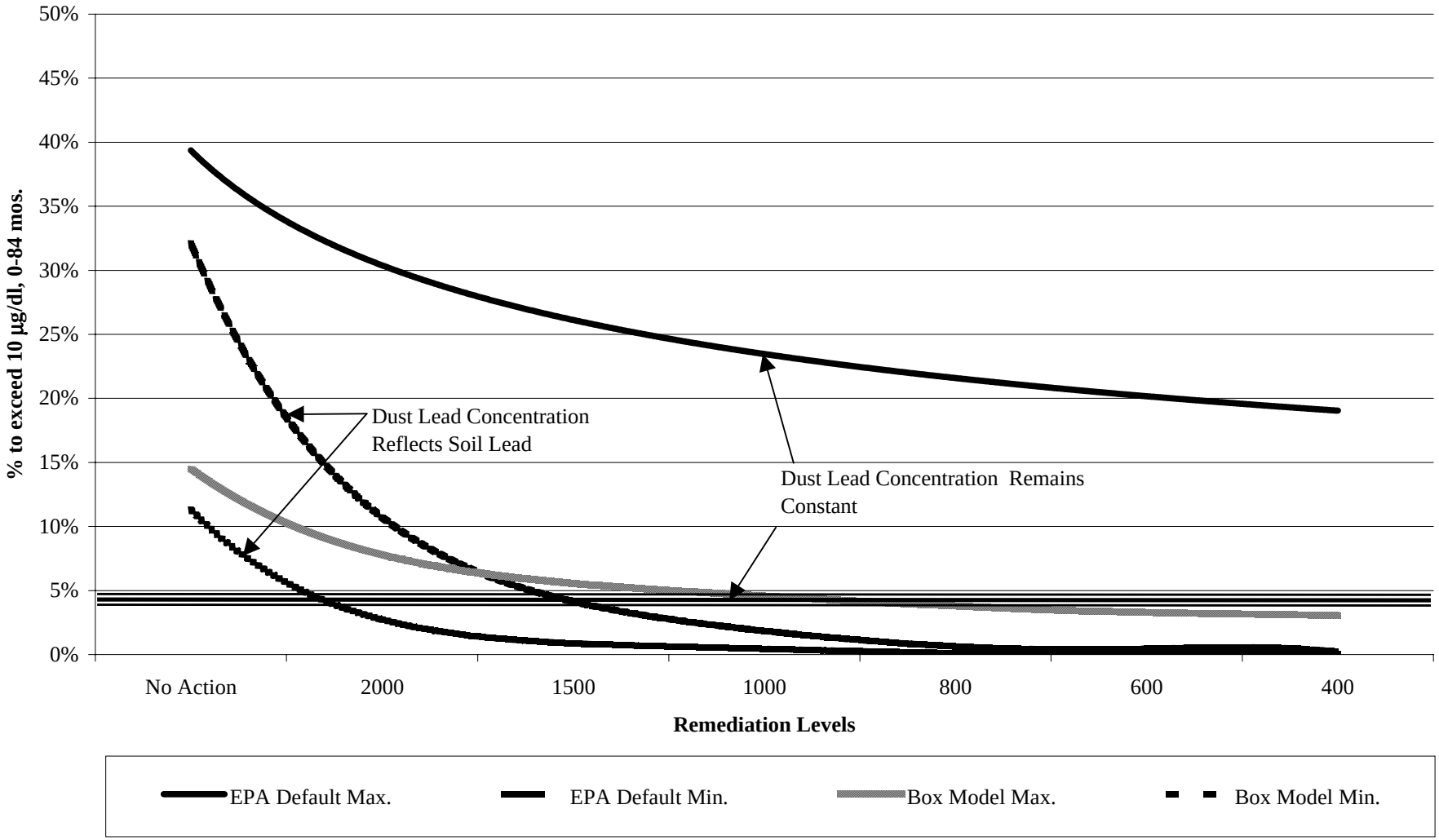
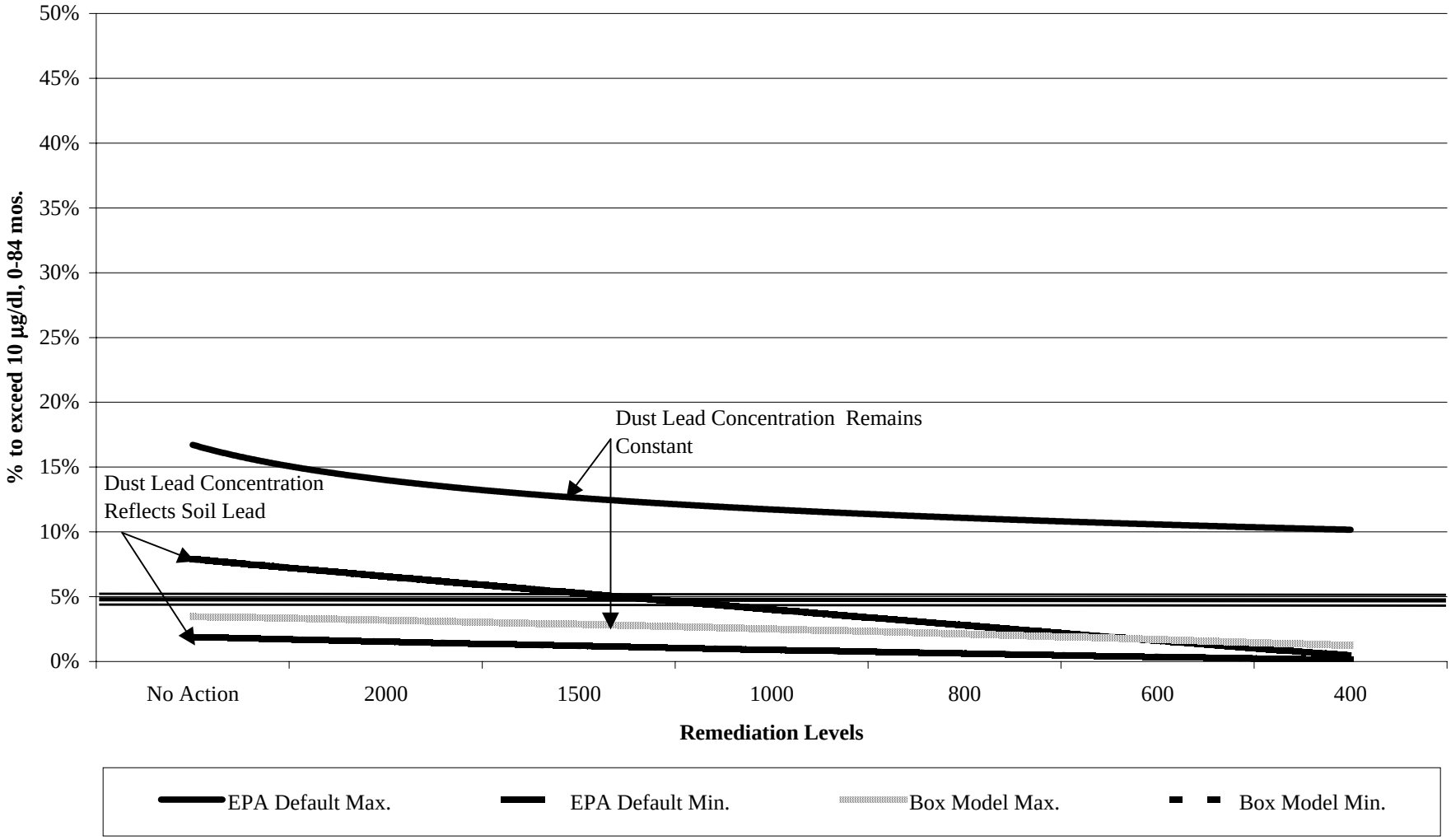


Figure R-3c Estimated Percent of Children to Exceed 10 µg/dl Blood Lead Associated with Various Yard Soil Cleanup Action Criteria Using Different Dust Concentrations - Silverton



FigureR-3d Estimated Percent of Children to Exceed 10 µg/dl Blood Lead Associated with Various Yard Soil Cleanup Action Criteria Using Different Dust Concentrations - Osburn

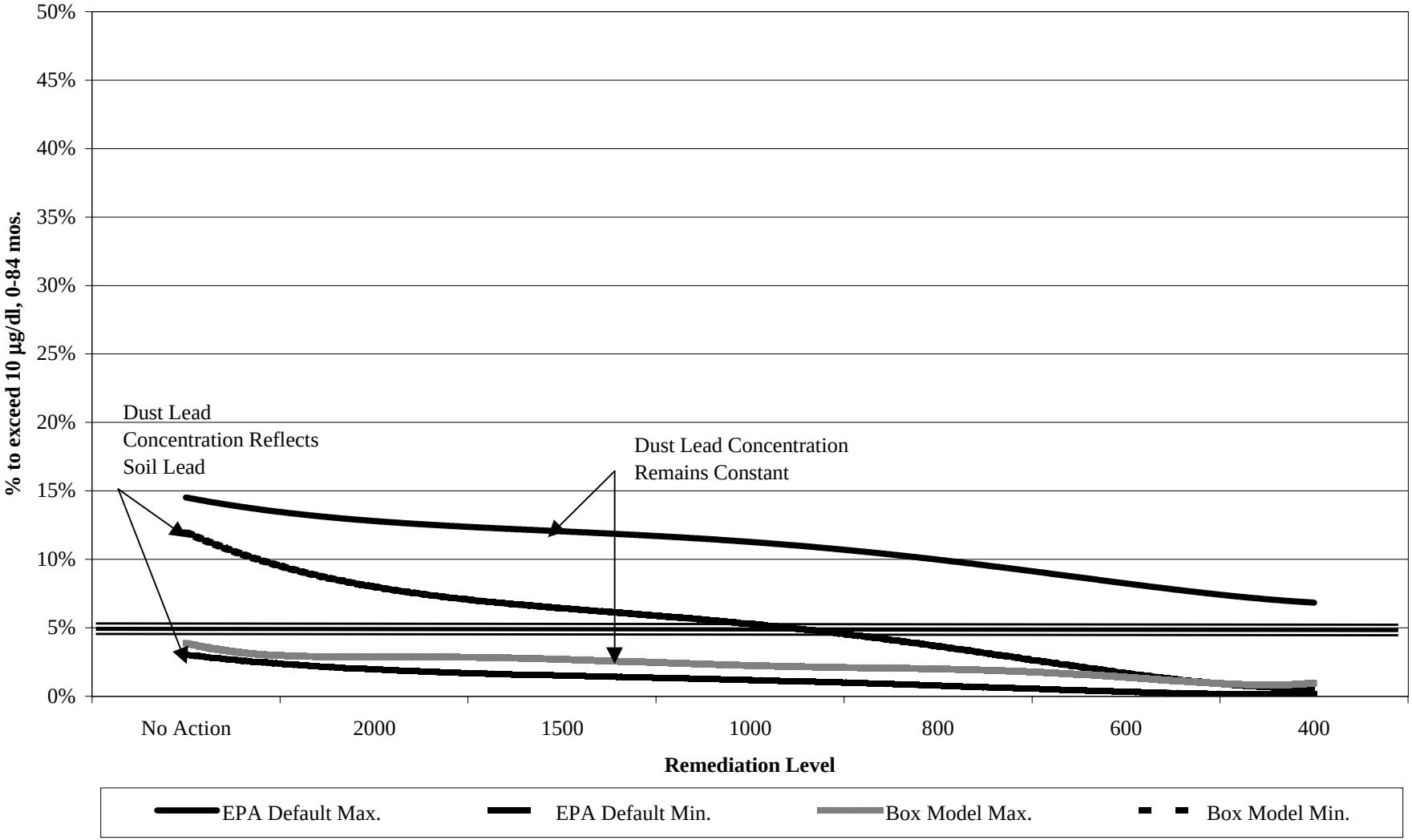


Figure R-3e Estimated Percent of Children to Exceed 10 mg/dl Blood Lead Associated with Various Yard Soil Cleanup Action Criteria Using Different Dust Concentrations - Side Gulches

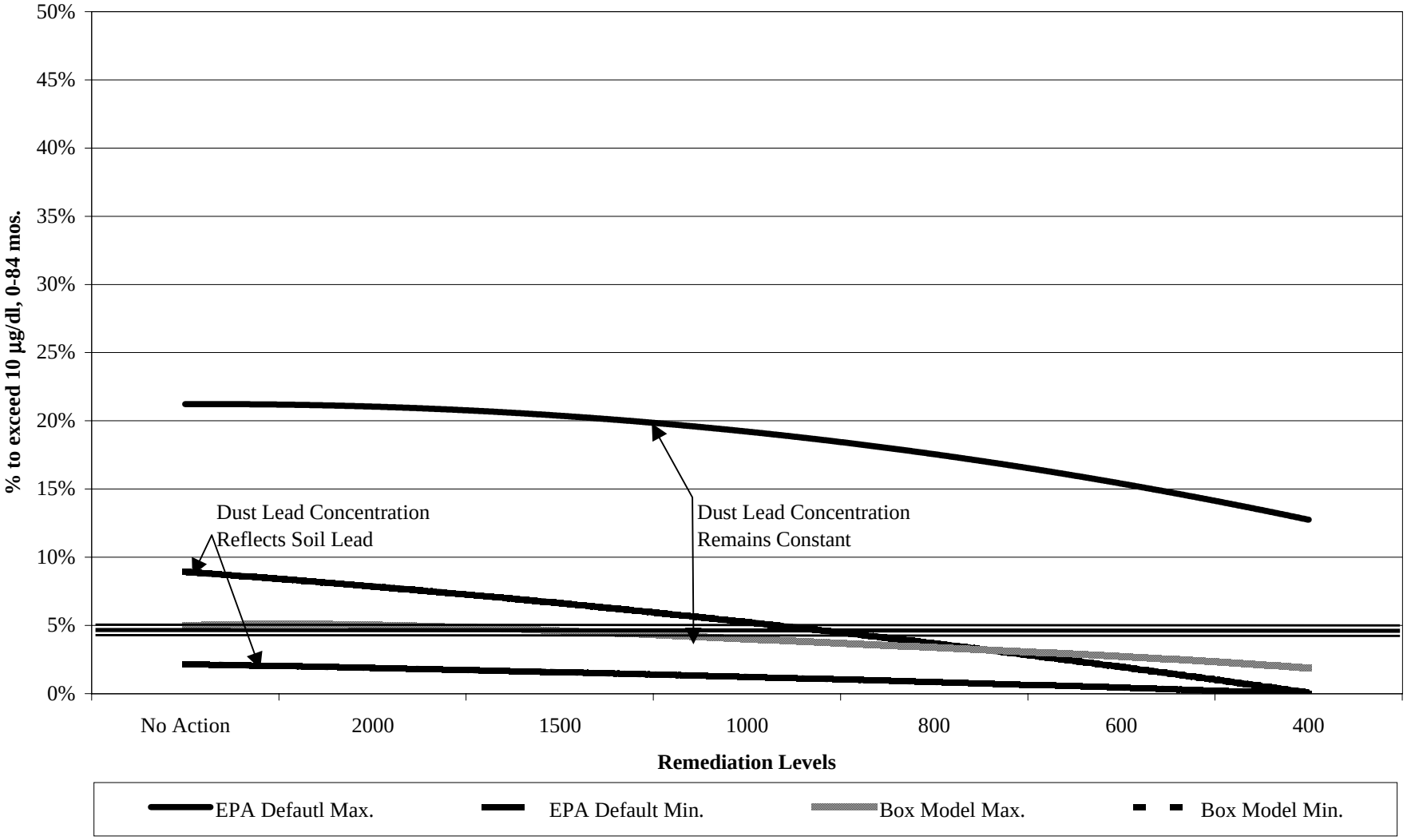


Figure R-3f Estimated Percent of Children to Exceed 10 µg/dl Blood Lead Associated with Various Yard Soil Cleanup Action Criteria Using Different Dust Concentrations - Kingston

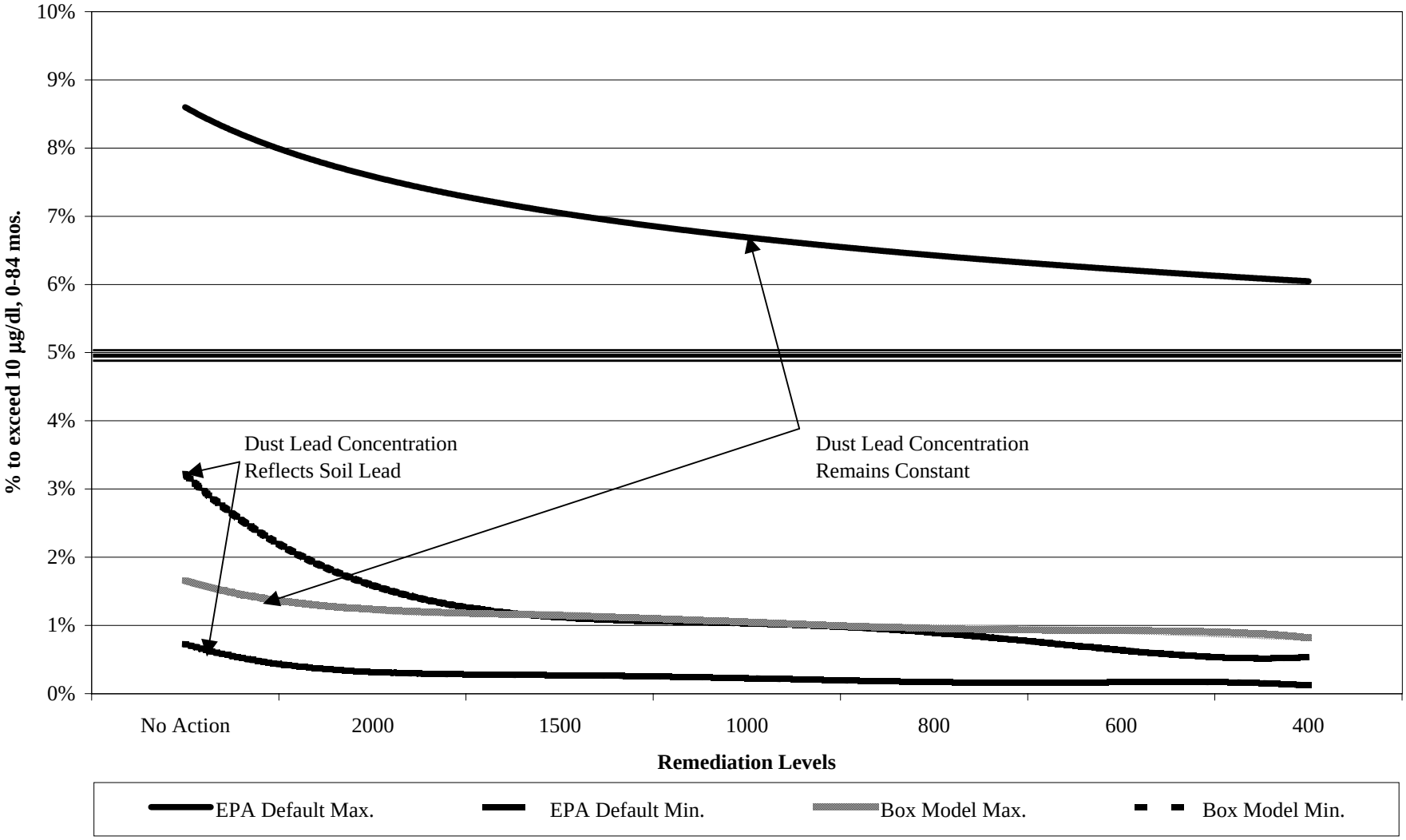


Table R-1 Estimated EPA Default Model Blood Lead Levels Associated With Various Yard Soil Cleanup Action Criteria

Mullan

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	627.8	985.0	11.3	58.29	10.2	50.13	9.3	42.68
400	127.9	985.0	9.1	40.37	8.2	32.10	7.5	25.29
600	158.8	985.0	9.2	40.37	8.3	32.10	7.6	26.86
800	199.4	985.0	9.4	42.68	8.5	34.02	7.7	26.86
1000	230.4	985.0	9.6	42.68	8.6	36.05	7.9	28.52
1500	299.9	985.0	9.9	47.56	8.9	38.16	8.1	30.26
2000	413.1	985.0	10.4	50.13	9.4	42.68	8.5	34.02
Actual	627.8	627.8	9.4	42.68	8.5	34.02	7.7	26.86
400	127.9	127.9	3.5	1.10	3.2	0.70	2.9	0.43
600	158.8	158.8	3.9	1.99	3.6	1.31	3.3	0.82
800	199.4	199.4	4.4	3.90	4.0	2.54	3.7	1.56
1000	230.4	230.4	4.8	5.67	4.4	3.67	4.0	2.39
1500	299.9	299.9	5.7	10.61	5.2	7.28	4.7	5.00
2000	413.1	413.1	7.0	21.06	6.4	15.45	5.8	11.30

Burke/Nine Mile

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	679.1	879.0	11.0	55.50	9.9	47.56	9.0	40.37
400	102.8	879.0	8.3	32.10	7.5	25.29	6.9	19.81
600	118.8	879.0	8.4	34.02	7.6	26.86	6.9	19.81
800	164.8	879.0	8.6	36.05	7.8	28.52	7.1	22.39
1000	195.4	879.0	8.8	36.05	7.9	28.52	7.2	22.39
1500	282.1	879.0	9.2	40.37	8.3	32.10	7.6	25.29
2000	401.4	879.0	9.7	45.07	8.8	36.05	8.0	30.26
Actual	679.1	679.1	9.9	47.56	8.9	38.16	8.1	32.10
400	102.8	102.8	3.1	0.61	2.9	0.39	2.7	0.22
600	118.8	118.8	3.3	0.92	3.1	0.58	2.8	0.35
800	164.8	164.8	4.0	2.25	3.6	1.47	3.3	0.92
1000	195.4	195.4	4.4	3.67	4.0	2.39	3.6	1.47
1500	282.1	282.1	5.5	9.36	5.0	6.42	4.5	4.42
2000	401.4	401.4	6.9	19.81	6.2	14.52	5.7	10.61

Table R-1 Estimated EPA Default Model Blood Lead Levels Associated With Various Yard Soil Cleanup Action Criteria (continued)

Wallace

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	771.5	1004.0	12.0	61.13	10.8	52.78	9.9	47.56
400	111.5	1004.0	9.1	40.37	8.2	32.10	7.5	25.29
600	142.0	1004.0	9.2	40.37	8.4	34.02	7.6	26.86
800	180.8	1004.0	9.4	42.68	8.5	34.02	7.8	28.52
1000	258.6	1004.0	9.8	45.07	8.8	38.16	8.1	30.26
1500	388.8	1004.0	10.4	50.13	9.4	42.68	8.5	34.02
2000	514.3	1004.0	10.9	55.50	9.9	45.07	9.0	38.16
Actual	771.5	771.5	10.8	52.78	9.8	45.07	8.9	38.16
400	111.5	111.5	3.2	0.77	3.0	0.49	2.8	0.28
600	142.0	142.0	3.6	1.47	3.4	0.92	3.1	0.58
800	180.8	180.8	4.2	2.87	3.8	1.87	3.5	1.16
1000	258.6	258.6	5.2	7.75	4.7	5.00	4.3	3.45
1500	388.8	388.8	6.7	18.62	6.1	13.64	5.6	9.96
2000	514.3	514.3	8.2	32.10	7.4	23.80	6.7	18.62

Silverton

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	351.9	557.0	7.6	26.86	6.9	19.81	6.3	15.45
400	137.4	557.0	6.5	16.45	5.9	12.03	5.4	8.79
600	194.6	557.0	6.8	19.81	6.2	14.25	5.6	10.61
800	249.9	557.0	7.1	22.39	6.4	16.45	5.8	12.03
1000	266.2	557.0	7.2	22.39	6.5	16.45	5.9	12.03
1500	306.0	557.0	7.4	23.80	6.7	18.62	6.1	13.64
2000	331.8	557.0	7.5	25.29	6.8	19.81	6.2	14.52
Actual	351.9	351.9	6.3	15.45	5.7	11.30	5.2	7.75
400	137.4	137.4	3.6	1.39	3.3	0.87	3	0.52
600	194.6	194.6	4.3	3.67	4.0	2.39	3.6	1.47
800	249.9	249.9	5.1	6.84	4.6	4.70	4.2	3.05
1000	266.2	266.2	5.3	8.25	4.8	5.67	4.4	3.67
1500	306.0	306.0	5.8	11.30	5.2	7.75	4.8	5.32
2000	331.8	331.8	6.1	13.64	5.5	9.36	5.0	6.84

Table R-1 Estimated EPA Default Model Blood Lead Levels Associated With Various Yard Soil Cleanup Action Criteria (continued)

Osburn

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	418.6	493.0	7.6	25.29	6.8	19.81	6.2	14.52
400	142.5	493.0	6.1	13.64	5.5	9.96	5.1	6.84
600	204.7	493.0	6.4	16.45	5.8	12.03	5.3	8.25
800	263.2	493.0	6.8	18.62	6.1	13.64	5.6	9.96
1000	295.4	493.0	6.9	21.06	6.3	15.45	5.7	11.30
1500	334.1	493.0	7.1	22.39	6.4	16.45	5.9	12.03
2000	348.9	493.0	7.2	22.39	6.5	17.50	5.9	12.81
Actual	418.6	418.6	7.1	22.39	6.4	16.45	5.8	12.03
400	142.5	142.5	3.7	1.47	3.4	0.97	3.1	0.58
600	204.7	204.7	4.5	4.15	4.1	2.70	3.7	1.76
800	263.2	263.2	5.2	7.75	4.8	5.32	4.3	3.67
1000	295.4	295.4	5.6	10.61	5.1	7.28	4.7	5.00
1500	334.1	334.1	6.1	13.64	5.5	9.96	5.0	6.85
2000	348.9	348.9	6.3	15.45	5.7	10.61	5.2	7.75

Side Gulches

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	367.5	695.0	8.5	34.02	7.7	26.86	7.0	21.06
400	123.6	695.0	7.3	23.80	6.6	17.50	6.0	12.81
600	195.6	695.0	7.7	26.86	6.9	21.06	6.3	15.45
800	266.2	695.0	8.0	30.26	7.3	23.80	6.6	17.50
1000	296.8	695.0	8.2	32.10	7.4	23.80	6.7	18.62
1500	344.7	695.0	8.4	34.02	7.6	26.86	6.9	21.06
2000	354.8	695.0	8.5	34.02	7.6	26.86	7.0	21.06
Actual	367.5	367.5	6.5	16.45	5.9	12.03	5.4	8.79
400	123.6	123.6	3.4	1.03	3.1	0.65	2.9	0.39
600	195.6	195.6	4.4	3.67	4.0	2.39	3.7	1.47
800	266.2	266.2	5.3	8.25	4.8	5.67	4.4	3.67
1000	296.8	296.8	5.6	10.61	5.1	7.28	4.7	5.00
1500	344.7	344.7	6.2	14.52	5.6	10.61	5.1	7.28
2000	354.8	354.8	6.3	15.45	5.8	11.30	5.2	7.75

Table R-1 Estimated EPA Default Model Blood Lead Levels Associated With Various Yard Soil Cleanup Action Criteria (continued)

Kingston

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	256.8	466.0	6.5	17.50	5.9	12.81	5.4	8.79
400	135.9	466.0	5.9	12.03	5.4	8.79	4.9	6.03
600	151.8	466.0	6.0	12.81	5.4	9.36	5.0	6.42
800	161.2	466.0	6.0	13.64	5.5	9.36	5.0	6.42
1000	164.7	466.0	6.1	13.64	5.5	9.36	5.0	6.42
1500	187.7	466.0	6.2	14.52	5.6	9.96	5.1	7.28
2000	193.4	466.0	6.2	14.52	5.6	10.61	5.1	7.28
Actual	256.8	256.8	5.1	7.28	4.7	5.00	4.3	3.24
400	135.9	135.9	3.6	1.31	3.3	0.82	3.0	0.52
600	151.8	151.8	3.8	1.76	3.5	1.16	3.2	0.69
800	161.2	161.2	3.9	2.11	3.6	1.39	3.3	0.87
1000	164.7	164.7	3.9	2.25	3.6	1.47	3.3	0.92
1500	187.7	187.7	4.3	3.24	3.9	2.11	3.6	1.31
2000	193.4	193.4	4.3	3.45	4.0	2.25	3.6	1.47

Lower Basin

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	109.5	301.0	4.6	4.70	4.2	3.05	3.8	1.99
400	68.2	301.0	4.4	3.67	4.0	2.39	3.7	1.47
600	72.2	301.0	4.4	3.67	4.0	2.39	3.7	1.56
800	73.1	301.0	4.4	3.67	4.0	2.39	3.7	1.56
1000	74.1	301.0	4.4	3.90	4.0	2.54	3.7	1.56
1500	80.2	301.0	4.4	3.90	4.1	2.54	3.7	1.66
2000	84.5	301.0	4.5	4.15	4.1	2.70	3.7	1.66
Actual	109.5	109.5	3.2	0.73	3.0	0.46	2.7	0.26
400	68.3	68.3	2.6	0.20	2.5	0.13	2.3	0.07
600	72.2	72.2	2.7	0.23	2.5	0.14	2.3	0.08
800	73.1	73.1	2.7	0.23	2.5	0.15	2.3	0.09
1000	74.1	74.1	2.7	0.25	2.5	0.16	2.3	0.09
1500	80.2	80.2	2.8	0.31	2.6	0.19	2.4	0.12
2000	84.5	84.5	2.8	0.35	2.7	0.22	2.5	0.13

Table R-2 Estimated Box Model Blood Lead Levels Associated With Yard Soil Cleanup Action Criteria

Mullan

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	627.8	985.0	7.6	26.86	6.9	19.81	6.3	14.52
400	127.9	985.0	5.5	9.36	5.0	6.42	4.5	4.42
600	158.8	985.0	5.6	9.96	5.1	7.28	4.6	4.70
800	199.4	985.0	5.8	11.30	5.2	7.75	4.8	5.67
1000	230.4	985.0	5.9	12.81	5.4	8.79	4.9	6.03
1500	299.9	985.0	6.2	14.52	5.6	10.61	5.1	7.28
2000	413.1	985.0	6.7	18.62	6.1	13.64	5.5	9.96
Actual	627.8	627.8	6.6	17.50	6.0	12.81	5.5	9.36
400	127.9	127.9	2.7	0.28	2.6	0.17	2.4	0.10
600	158.8	158.8	3.0	0.49	2.8	0.31	2.6	0.18
800	199.4	199.4	3.3	0.92	3.1	0.58	2.8	0.35
1000	230.4	230.4	3.6	1.39	3.3	0.87	3.0	0.55
1500	299.9	299.9	4.2	2.87	3.8	1.87	3.5	1.16
2000	413.1	413.1	5.0	6.84	4.6	4.42	4.2	3.05

Burke/Nine Mile

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	679.1	879.0	7.5	25.29	6.8	19.81	6.2	14.52
400	102.8	879.0	5.0	6.84	4.6	4.42	4.2	3.05
600	118.8	879.0	5.1	7.28	4.6	4.70	4.2	3.24
800	164.8	879.0	5.3	8.25	4.8	5.67	4.4	3.90
1000	195.4	879.0	5.5	9.36	5.0	6.42	4.5	4.42
1500	282.1	879.0	5.8	12.03	5.3	8.25	4.8	5.67
2000	401.4	879.0	6.4	15.45	5.8	11.30	5.2	7.75
Actual	679.1	679.1	7.0	21.06	6.3	15.45	5.7	11.30
400	102.8	102.8	2.5	0.16	2.4	0.10	2.2	0.06
600	118.8	118.8	2.7	0.22	2.5	0.14	2.3	0.08
800	164.8	164.8	3.1	0.55	2.8	0.35	2.6	0.20
1000	195.4	195.4	3.3	0.87	3.1	0.55	2.8	0.33
1500	282.1	282.1	4.0	2.39	3.7	1.56	3.4	0.97
2000	401.4	401.4	4.9	6.42	4.5	4.15	4.1	2.70

Table R-2 Estimated Box Model Blood Lead Levels Associated With Yard Soil Cleanup Action Criteria (continued)

Wallace

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	771.5	1004.0	8.2	32.10	7.4	25.29	6.8	18.62
400	111.5	1004.0	5.4	9.36	5.0	6.42	4.5	4.15
600	142.0	1004.0	5.6	9.96	5..1	6.84	4.6	4.70
800	180.8	1004.0	5.8	11.30	5.2	7.75	4.8	5.32
1000	258.6	1004.0	6.1	13.64	5.5	9.96	5.0	6.84
1500	388.8	1004.0	6.7	18.62	6.0	12.81	5.5	9.36
2000	514.3	1004.0	7.2	22.39	6.5	16.45	5.9	12.03
Actual	771.5	771.5	7.6	26.86	6.9	19.81	6.3	14.52
400	111.5	111.5	2.6	0.19	2.4	0.12	2.3	0.07
600	142.0	142.0	2.9	0.37	2.7	0.22	2.5	0.14
800	180.8	180.8	3.2	0.69	3.0	0.43	2.7	0.26
1000	258.6	258.6	3.8	1.87	3.5	1.23	3.2	0.77
1500	388.8	388.8	4.8	5.67	4.4	3.90	4.0	2.54
2000	514.3	514.3	5.8	11.30	5.3	8.25	4.8	5.67

Silverton

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	351.9	557.0	5.2	7.75	4.7	5.32	4.3	3.45
400	137.4	557.0	4.2	3.05	3.8	1.99	3.5	1.23
600	194.6	557.0	4.5	4.15	4.1	2.70	3.7	1.66
800	249.9	557.0	4.7	5.32	4.3	3.45	3.9	2.25
1000	266.2	557.0	4.8	5.67	4.4	3.67	4.0	2.39
1500	306.0	557.0	5.0	6.42	4.5	4.42	4.1	2.87
2000	331.8	557.0	5.1	7.28	4.6	4.70	4.2	3.24
Actual	351.9	351.9	4.6	4.42	4.2	2.87	3.8	1.87
400	137.4	137.4	2.8	0.33	2.6	0.21	2.4	0.12
600	194.6	194.6	3.3	0.87	3.1	0.55	2.8	0.33
800	249.9	249.9	3.8	1.76	3.4	1.10	3.2	0.69
1000	266.2	266.2	3.9	2.11	3.6	1.31	3.3	0.82
1500	306.0	306.0	4.2	3.05	3.8	1.99	3.5	1.23
2000	331.8	331.8	4.4	3.90	4.0	2.54	3.7	1.56

Table R-2 Estimated Box Model Blood Lead Levels Associated With Yard Soil Cleanup Action Criteria (continued)

Osburn

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	418.6	493.0	5.3	8.25	4.8	5.67	4.4	3.90
400	142.5	493.0	4.0	2.39	3.7	1.56	3.4	0.97
600	204.7	493.0	4.3	3.45	3.9	2.25	3.6	1.39
800	263.2	493.0	4.6	4.70	4.2	3.05	3.8	1.99
1000	295.4	493.0	4.7	5.32	4.3	3.45	4.0	2.25
1500	334.1	493.0	4.9	6.03	4.5	4.15	4.1	2.70
2000	348.9	493.0	5.0	6.42	4.5	4.42	4.1	2.87
Actual	418.6	418.6	5.1	6.84	4.6	4.70	4.2	3.05
400	142.5	142.5	2.9	0.37	2.7	0.22	2.5	0.14
600	204.7	204.7	3.4	0.97	3.1	0.61	2.9	0.37
800	263.2	263.2	3.9	1.99	3.5	1.31	3.3	0.77
1000	295.4	295.4	4.1	2.70	3.8	1.76	3.5	1.10
1500	334.1	334.1	4.4	3.90	4.0	2.54	3.7	1.66
2000	348.9	348.9	4.5	4.42	4.1	2.87	3.8	1.87

Side Gulches

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	367.5	695.0	5.7	10.61	5.2	7.28	4.7	5.00
400	123.6	695.0	4.6	4.42	4.2	2.87	3.8	1.87
600	195.6	695.0	4.9	6.03	4.5	4.15	4.1	2.70
800	266.2	695.0	5.2	7.75	4.7	5.32	4.3	3.45
1000	296.8	695.0	5.4	8.79	4.9	6.03	4.4	3.90
1500	344.7	695.0	5.6	9.96	5.1	6.84	4.6	4.70
2000	354.8	695.0	5.6	10.61	5.1	7.28	4.7	5.00
Actual	367.5	367.5	4.7	5.00	4.3	3.24	3.9	2.11
400	123.6	123.6	2.7	0.25	2.5	0.16	2.3	0.09
600	195.6	195.6	3.3	0.87	3.1	0.55	2.8	0.33
800	266.2	266.2	3.9	2.11	3.6	1.31	3.3	0.82
1000	296.8	296.8	4.1	2.87	3.8	1.76	3.5	1.16
1500	344.7	344.7	4.5	4.15	4.1	2.70	3.8	1.76
2000	354.8	354.8	4.6	4.42	4.2	3.05	3.8	1.87

Table R-2 Estimated Box Model Blood Lead Levels Associated With Yard Soil Cleanup Action Criteria (continued)

Kingston

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	256.8	466.0	4.5	4.15	4.1	2.70	3.7	1.66
400	135.9	466.0	3.9	2.11	3.6	1.39	3.3	0.82
600	151.8	466.0	4.0	2.39	3.6	1.47	3.3	0.92
800	161.2	466.0	4.0	2.39	3.7	1.56	3.4	0.97
1000	164.7	466.0	4.0	2.54	3.7	1.66	3.4	1.03
1500	187.7	466.0	4.1	2.87	3.8	1.87	3.5	1.16
2000	193.4	466.0	4.2	2.87	3.8	1.87	3.5	1.23
Actual	256.8	256.8	3.8	1.87	3.5	1.16	3.2	0.73
400	135.9	135.9	2.8	0.33	2.6	0.20	2.4	0.12
600	151.8	151.8	2.9	0.43	2.7	0.28	2.5	0.16
800	161.2	161.2	3.0	0.52	2.8	0.33	2.6	0.19
1000	164.7	164.7	3.1	0.55	2.8	0.35	2.6	0.20
1500	187.7	187.7	3.2	0.77	3.0	0.49	2.8	0.29
2000	193.4	193.4	3.3	0.87	3.0	0.52	2.8	0.31

Lower Basin

Yard Soil Remediation Concentration (µg/kg)	Communtity Soil Concentration (µg/kg)	Community Dust Concentration (µg/kg)	Geo Mean 9 to 24 mos. (µg/dL)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dL)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dL)	% to exceed 10 µg/dl 0 to 84 mos.
Actual	109.5	301.0	3.2	0.77	3.0	0.49	2.8	0.28
400	68.2	301.0	3.0	0.52	2.8	0.33	2.6	0.19
600	72.2	301.0	3.0	0.52	2.8	0.33	2.6	0.20
800	73.1	301.0	3.0	0.55	2.8	0.33	2.6	0.20
1000	74.1	301.0	3.1	0.55	2.8	0.35	2.6	0.20
1500	80.2	301.0	3.1	0.58	2.9	0.37	2.6	0.21
2000	84.5	301.0	3.1	0.61	2.9	0.37	2.7	0.22
Actual	109.5	109.5	2.6	0.18	2.4	0.12	2.3	0.07
400	68.3	68.3	2.2	0.06	2.1	0.04	2.0	0.02
600	72.2	72.2	2.3	0.07	2.1	0.04	2.0	0.02
800	73.1	73.1	2.3	0.07	2.1	0.05	2.0	0.02
1000	74.1	74.1	2.3	0.07	2.2	0.05	2.0	0.03
1500	80.2	80.2	2.3	0.09	2.2	0.06	2.1	0.03
2000	84.5	84.5	2.4	0.10	2.2	0.06	2.1	0.03

Table R-3 Estimated Community Blood Lead Levels and Health Risk Associated With Various Yard Soi Action Criteria

Mullan

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)
EPA Default							
Actual	627.8	985.0	11.3	58.29	10.2	50.13	9.3
400	127.9	381.4	5.3	8.25	4.8	5.67	4.4
600	158.8	411.1	5.7	10.61	5.1	7.28	4.7
800	199.4	446.9	6.1	13.64	5.5	9.96	5.1
1000	230.4	471.4	6.4	16.45	5.8	12.03	5.3
1500	299.9	525.3	7.2	22.39	6.5	16.45	5.9
2000	413.1	607.1	8.2	32.10	7.4	25.29	6.8
Box Model							
Actual	627.8	985.0	7.6	26.86	6.9	19.81	6.3
400	127.9	381.4	3.6	1.39	3.3	0.87	3.0
600	158.8	411.1	3.8	1.99	3.5	1.23	3.2
800	199.4	446.9	4.1	2.87	3.8	1.87	3.5
1000	230.4	471.4	4.4	3.67	4.0	2.39	3.7
1500	299.9	525.3	4.9	5.67	4.4	3.90	4.0
2000	413.1	607.1	5.6	10.61	5.1	7.28	4.7

Burke/Nine Mile

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)
EPA Default							
Actual	679.1	879.0	11.0	55.50	9.9	47.56	9.0
400	102.8	353.7	4.9	6.42	4.5	4.15	4.1
600	118.8	371.1	5.2	7.28	4.7	5.00	4.3
800	164.8	416.5	5.7	11.30	5.2	7.75	4.7
1000	195.4	442.7	6.1	13.64	5.5	9.36	5.0
1500	282.1	511.8	7.0	21.06	6.3	15.45	5.8
2000	401.4	598.9	8.1	30.26	7.3	23.80	6.7
Box Model							
Actual	679.1	879.0	7.5	25.29	6.8	19.81	6.2
400	102.8	353.7	3.4	0.97	3.1	0.61	2.9
600	118.8	371.1	3.5	1.23	3.2	0.77	3.0
800	164.8	416.5	3.9	2.11	3.6	1.31	3.3
1000	195.4	442.7	4.1	2.7	3.8	1.76	3.5
1500	282.1	511.8	4.7	5.32	4.3	3.45	3.9
2000	401.4	598.9	5.5	9.96	5.0	6.84	4.6

Table R-3 Estimated Community Blood Lead Levels and Health Risk Associated With Various Yard Soi Action Criteria (continued)

Wallace

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)
EPA Default							
Actual	771.5	1004.0	12.0	61.13	10.8	52.78	9.9
400	111.5	364.7	5.1	6.84	4.6	4.70	4.2
600	142.0	395.1	5.5	9.36	5.0	6.42	4.5
800	180.8	430.7	5.9	12.03	5.4	8.79	4.9
1000	258.6	493.4	6.7	18.62	6.1	13.64	5.6
1500	388.8	590.0	8.0	30.26	7.2	22.39	6.6
2000	514.3	678.4	9.1	40.37	8.2	32.10	7.5
Box Model							
Actual	771.5	1004.0	8.2	32.10	7.4	25.29	6.8
400	111.5	364.7	3.5	1.10	3.2	0.69	2.9
600	142.0	395.1	3.7	1.56	3.4	1.03	3.1
800	180.8	430.7	4.0	2.39	3.7	1.56	3.4
1000	258.6	493.4	4.6	4.42	4.2	2.87	3.8
1500	388.8	590.0	5.5	9.36	5.0	6.42	4.5
2000	514.3	678.4	6.3	15.45	5.7	10.61	5.2

Silverton

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)
EPA Default							
Actual	351.9	557.0	7.6	26.86	6.9	19.81	6.3
400	137.4	390.3	5.4	8.79	4.9	6.03	4.5
600	194.6	442.7	6.1	13.64	5.5	9.36	5.0
800	249.9	487.2	6.7	18.62	6.0	12.81	5.5
1000	266.2	499.6	6.8	19.81	6.2	14.52	5.6
1500	306.0	529.7	7.2	22.39	6.5	17.50	5.9
2000	331.8	548.9	7.5	25.29	6.8	18.62	6.2
Box Model							
Actual	351.9	557.0	5.2	7.75	4.7	5.32	4.3
400	137.4	390.3	3.7	1.56	3.4	0.97	3.1
600	194.6	442.7	4.1	2.70	3.8	1.76	3.5
800	249.9	487.2	4.5	4.15	4.1	2.70	3.8
1000	266.2	499.6	4.6	4.70	4.2	3.05	3.9
1500	306.0	529.7	4.9	6.03	4.5	4.15	4.1
2000	331.8	548.9	5.1	6.84	4.6	4.70	4.2

Table R-3 Estimated Community Blood Lead Levels and Health Risk Associated With Various Yard Soi Action Criteria (continued)

Osburn

Yard Soil Remediation Concentration (mg/kg)	Communtity Soil Concentration (mg/kg)	Community Dust Concentration (mg/kg)	Geo Mean 9 to 24 mos. (µg/dl)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dl)
EPA Default							
Actual	418.6	493.0	7.6	25.29	6.8	19.81	6.2
400	142.5	396.1	5.5	9.36	5.0	6.42	4.5
600	204.7	451.1	6.2	14.52	5.6	9.96	5.1
800	263.2	497.3	6.8	19.81	6.1	13.64	5.6
1000	295.4	521.6	7.1	22.39	6.4	16.45	5.9
1500	334.1	550.3	7.5	25.29	6.8	18.62	6.2
2000	348.9	561.2	7.6	26.86	6.9	19.81	6.3
Box Model							
Actual	418.6	493.0	5.3	8.25	4.8	5.67	4.4
400	142.5	396.1	3.7	1.66	3.4	1.03	3.1
600	204.7	451.1	4.2	3.05	3.8	1.87	3.5
800	263.2	497.3	4.6	4.70	4.2	3.05	3.8
1000	295.4	521.6	4.8	5.67	4.4	3.67	4.0
1500	334.1	550.3	5.1	7.28	4.6	4.70	4.2
2000	348.9	561.2	5.2	7.75	4.7	5.32	4.3

Side Gulches

Yard Soil Remediation Concentration (µg/kg)	Communtity Soil Concentration (µg/kg)	Community Dust Concentration (µg/kg)	Geo Mean 9 to 24 mos. (µg/dL)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dL)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dL)
EPA Default							
Actual	367.5	695.0	8.5	34.02	7.7	26.86	7.0
400	123.6	377.3	5.2	7.75	4.8	5.32	4.3
600	195.6	443.5	6.1	13.64	5.5	9.36	5.0
800	266.2	499.6	6.8	19.81	6.2	14.52	5.6
1000	296.8	523.0	7.1	22.39	6.4	16.45	5.9
1500	344.7	558.3	7.6	26.86	6.9	19.81	6.3
2000	354.8	565.6	7.7	26.86	6.9	21.06	6.3
Box Model							
Actual	367.5	695.0	5.7	10.61	5.2	7.28	4.7
400	123.6	377.3	3.6	1.31	3.3	0.82	3.0
600	195.6	443.5	4.1	2.70	3.8	1.76	3.5
800	266.2	499.6	4.6	4.70	4.2	3.05	3.9
1000	296.8	523.0	4.8	5.67	4.4	3.90	4.0
1500	344.7	558.3	5.2	7.28	4.7	5.00	4.3
2000	354.8	565.6	5.2	7.75	4.8	5.32	4.3

Table R-3 Estimated Community Blood Lead Levels and Health Risk Associated With Various Yard Soi Action Criteria (continued)

Kingston

Yard Soil Remediation Concentration (µg/kg)	Communtity Soil Concentration (µg/kg)	Community Dust Concentration (µg/kg)	Geo Mean 9 to 24 mos. (µg/dL)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dL)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dL)
EPA Default							
Actual	256.8	466.0	6.5	17.50	5.9	12.81	5.4
400	135.9	389.3	5.4	8.79	4.9	6.03	4.5
600	151.8	404.6	5.6	9.96	5.1	6.84	4.6
800	161.2	412.9	5.7	10.61	5.2	7.28	4.7
1000	164.7	416.5	5.7	11.30	5.2	7.75	4.7
1500	187.7	435.9	6.0	12.81	5.4	8.79	4.9
2000	193.4	441.0	6.0	13.64	5.5	9.36	5.0
Box Model							
Actual	256.8	466.0	4.5	4.15	4.1	2.70	3.7
400	135.9	389.3	3.6	1.47	3.4	0.97	3.1
600	151.8	404.6	3.8	1.76	3.5	1.16	3.2
800	161.2	412.9	3.8	1.99	3.5	1.23	3.2
1000	164.7	416.5	3.9	2.11	3.6	1.31	3.3
1500	187.7	435.9	4.0	2.54	3.7	1.66	3.4
2000	193.4	441.0	4.1	2.70	3.7	1.76	3.4

Lower Basin

Yard Soil Remediation Concentration (µg/kg)	Communtity Soil Concentration (µg/kg)	Community Dust Concentration (µg/kg)	Geo Mean 9 to 24 mos. (µg/dL)	% to exceed 10 µg/dl 9 to 24 mos.	Geo Mean 0 to 60 mos. (µg/dL)	% to exceed 10 µg/dl 0 to 60 mos.	Geo Mean 0 to 84 mos. (µg/dL)
EPA Default							
Actual	109.5	301.0	4.6	4.70	4.2	3.05	3.8
400	68.2	311.2	4.4	3.90	4.1	2.54	3.7
600	72.2	316.7	4.5	4.15	4.1	2.70	3.8
800	73.1	318.1	4.5	4.15	4.1	2.87	3.8
1000	74.1	319.4	4.5	4.42	4.1	2.87	3.8
1500	80.2	327.3	4.6	4.70	4.2	3.05	3.9
2000	84.5	332.4	4.7	5.00	4.3	3.24	3.9
Box Model							
Actual	109.5	301.0	3.2	0.77	3.0	0.49	2.8
400	68.2	311.2	3.1	0.55	2.8	0.35	2.6
600	72.2	316.7	3.1	0.58	2.9	0.37	2.7
800	73.1	318.1	3.1	0.61	2.9	0.37	2.7
1000	74.1	319.4	3.1	0.61	2.9	0.39	2.7
1500	80.2	327.3	3.2	0.69	2.9	0.43	2.7
2000	84.5	332.4	3.2	0.73	3.0	0.46	2.7

l Cleanup

% to exceed 10 µg/dl 0 to 84 mos.
42.68
3.67
5.00
6.84
8.25
12.03
18.62
14.52
0.52
0.77
1.16
1.47
2.54
5.00

% to exceed 10 µg/dl 0 to 84 mos.
40.37
2.70
3.45
5.32
6.84
11.30
18.62
14.52
0.37
0.46
0.82
1.10
2.25
4.70

l Cleanup

% to exceed 10 µg/dl 0 to 84 mos.
47.56
3.05
4.42
6.03
9.96
17.50
25.29
18.62
0.41
0.61
0.97
1.87
4.42
7.75

% to exceed 10 µg/dl 0 to 84 mos.
15.45
4.15
6.84
9.36
10.61
12.81
14.52
3.45
0.58
1.10
1.76
1.99
2.70
3.05

l Cleanup

% to exceed 10 µg/dl 0 to 84 mos.
14.52
4.42
7.28
9.96
12.03
14.52
15.45
3.90
0.65
1.23
1.99
2.54
3.24
3.45

% to exceed 10 µg/dl 0 to 84 mos.
21.06
3.67
6.84
10.61
12.03
14.52
15.45
5.00
0.49
1.10
1.99
2.54
3.45
3.67

l Cleanup

% to exceed 10 µg/dl 0 to 84 mos.
8.79
4.15
4.70
5.00
5.32
6.42
6.42
1.66
0.58
0.69
0.77
0.82
1.03
1.10

% to exceed 10 µg/dl 0 to 84 mos.
1.99
1.66
1.76
1.76
1.87
1.99
2.11
0.28
0.20
0.22
0.22
0.22
0.25
0.28

Table R-4 Individual Blood Lead Levels and Health Risk at Potential Action Levels

Method	Yard Soil Remediation Concentration (mg/kg)	Geometric Mean 0 to 84 mos. (µg/dl)	% to exceed 10 µg/dl 0 to 84 mos.	% to exceed 15 µg/dl 0 to 84 mos.
Mullan				
EPA Default	400	5.6	10.0	1.7
	600	6.6	17.5	3.7
Box Model	600	4.9	5.7	0.8
	800	5.6	10.61	1.8
	1000	6.4	15.5	3.2
	2000	9.7	45.1	16.5
Burke/Nine Mile				
EPA Default	400	5.4	9.4	1.5
	600	6.4	15.5	3.2
Box Model	600	4.8	5.3	0.7
	800	5.6	9.96	1.7
	1000	6.3	15.5	3.1
	2000	9.6	45.1	16.5
Wallace				
EPA Default	400	5.5	9.4	1.6
	600	6.5	16.5	3.5
Box Model	600	4.8	5.7	0.7
	800	5.6	9.96	1.7
	1000	6.4	16.5	3.2
	2000	9.8	45.1	17.5
Silverton				
EPA Default	400	5.6	10.6	1.8
	600	6.7	18.6	4.2
Box Model	600	4.9	6.0	0.8
	800	5.7	11.3	1.9
	1000	6.4	16.5	3.2
	2000	9.5	42.7	15.5
Osburn				
EPA Default	400	5.7	10.6	1.8
	600	6.8	18.6	4.2
Box Model	600	4.9	6.4	0.9
	800	5.7	11.3	1.9
	1000	6.5	16.5	3.5
	2000	9.6	42.7	15.5
Side Gulches				
EPA Default	400	5.6	10.0	1.7
	600	6.7	18.6	4.2
Box Model	600	4.9	6.0	0.8
	800	5.7	11.3	2.0
	1000	6.5	16.5	3.5
	2000	9.6	42.7	15.5
Kingston				
EPA Default	400	5.6	10.6	1.8
	600	6.5	17.5	3.7
Box Model	600	4.8	5.7	0.8
	800	5.6	9.96	1.7
	1000	6.2	14.5	2.9
	2000	9.3	42.7	14.5
Lower Basin				
EPA Default	400	5.2	7.8	1.2
	600	6.1	13.6	2.5
Box Model	600	4.6	4.7	0.6
	800	5.3	8.25	1.31
	1000	6.0	12.8	2.4
	2000	9.1	40.4	13.6

APPENDIX S

Heavy Metal Concentrations in Water Potatoes in the Coeur d'Alene Basin, Idaho

2360.5

Heavy Metal Concentrations in *Sagittaria spp.* Tubers (Water Potato) in the Coeur d'Alene Basin, Idaho

FINAL DRAFT

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Litigation Sensitive -- Not for Distribution

Abstract

The Coeur d'Alene Basin in northern Idaho provides extensive wetland habitat, along the lower Coeur d'Alene and lower St. Joe rivers, for migrating and resident waterfowl. The lateral lakes and wetlands of the lower Coeur d'Alene River, a major portion of this habitat, have been impacted by metals-contaminated sediments related to mining activities. For years waterfowl have died in the Coeur d'Alene River wetland areas, reportedly due to lead poisoning from contaminated sediments or vegetation. *Sagittaria spp.* tubers are one of the major food sources for waterfowl in the Coeur d'Alene Basin. These tubers are also a traditional subsistence item for the Coeur d'Alene Tribe, whose aboriginal homeland is the Coeur d'Alene Basin. Tribal members gather tubers annually from the non-contaminated wetlands along the lower St. Joe River. In 1994 we sampled *Sagittaria* tubers from 14 wetlands along the lower Coeur d'Alene and St. Joe rivers to determine if tubers in Coeur d'Alene River wetlands expose waterfowl eating the tubers to metals (primarily lead), and to determine what metals concentrations tribal members would be exposed to if they were to eat tubers from Coeur d'Alene River wetlands. We also evaluated metals concentrations associated with tuber skins vs. the interior portion of the tuber. In addition, we investigated differences in mean metals concentrations in tubers between Coeur d'Alene River wetlands, and looked for relationships between tuber metals concentrations and sediment metals concentrations in wetlands from which the tubers were sampled. We found that waterfowl feeding on tubers from Coeur d'Alene River wetlands are exposed to significantly higher levels of lead, cadmium, manganese and zinc than those feeding on tubers from St. Joe River wetlands. Mean lead concentrations from Coeur d'Alene River wetlands which were

sampled ranged from 3.6 to 86.3 ppm ww, for tubers with skins. Lead was not detected in St. Joe River wetland tubers. Lead in tubers from Coeur d'Alene River wetlands exists primarily in the tuber skins, and is most likely due to sediment adhering to skins or trapped in fibrous material on the skins. If tribal members were to gather and eat tubers from Coeur d'Alene River wetlands they would likewise be exposed to significantly higher levels of lead, cadmium, manganese and zinc. Lead exposure from ingesting tubers could be greatly reduced by removing skins, however potential exposure to lead and other metals from sediment contact during the gathering process should be considered.

Introduction

Sagittaria latifolia and *Sagittaria cuneata* are found in lakes and wetlands throughout the Pacific northwest United States (California, Oregon, Washington and Idaho), as well as locations on the Atlantic coast and throughout the U.S.A. (Hitchcock and Cronquist, 1973). These wetland plants produce large starchy tubers, commonly known as water potato, which serve as a major food source for waterfowl and a traditional subsistence item for the Coeur d'Alene Tribe (SiJohn, per comm.) and other native American tribes. *Sagittaria spp.* is found throughout the Coeur d'Alene Basin (CDAB), including the Coeur d'Alene (CDA) and St. Joe (STJ) River basins, of northern Idaho. This area serves as major habitat for thousands of migrating waterfowl, including ducks, Canada geese (*Branta canadensis*) and tundra swans *(Cygnus columbianus*), in

the spring and to a lesser extent in the fall. Biologists have observed waterfowl feeding on *Sagittaria spp.* tubers in wetlands in the CDAB and have identified the tubers in ingesta removed from waterfowl found dead in the basin (Audet et al., 1999). The CDAB is also the home land of the Coeur d'Alene Tribe, whose members gather the tubers annually for human consumption.

The lateral lakes wetland complex is an approximately 40 km stretch of lakes and associated wetlands along the lower CDA River between the towns of Cataldo and Harrison. This area has been contaminated with heavy metals resulting from more than 100 years of mining activity upstream in the Silver Valley, a historic mining district near the town of Kellogg. Horowitz et al. (1993) estimated that 75 million tons of metal enriched sediments associated with mining activities in upstream locations of the CDARB have been deposited in the lakebed of Lake CDA. In addition, the floodplain, associated wetlands and lateral lakes of the CDA River contain large volumes of trace element-rich material, which have been released by mining activities in the upper basin and transported via the CDA River downstream to these areas. Elevated levels of hazardous substances such as lead (Pb), zinc (Zn) and cadmium (Cd), as well as other metals have been documented in the lower CDA River system (Horowitz et al., 1993; Hornig et al.,1988; Maxfield et al, 1974). Deaths of waterfowl in the lateral lakes area have been reported since 1924 (Blus et al., 1991; Neufeld, 1987; Chupp and Dalke, 1964; Chupp, 1956), many of them caused by Pb poisoning due to ingestion of Pb contaminated vegetation or sediments (Blus et al., 1991; Krieger, 1990). Elevated Pb concentrations have been found in kidney and liver tissues from waterfowl found dead in the CDA River Basin (CDARB) (Audet, 1997; Blus et al., 1991; Krieger, 1990). Neufeld (1987) suggests that the most significant cause of swan mortality

in the CDARB is ingestion of Pb contaminated sediments deposited on and retained by aquatic vegetation, particularly after high water, and direct consumption of the sediments for use as grit. Lead and Zn concentrations, as well as other metals, have been reported in both submergent and emergent vegetation collected from the CDARB (Audet, 1997.; Krieger, 1990). Lead concentrations were found to be higher in equisetum samples with small quantities of CDARB sediment adhering to them than in equisetum cleaned with deionized water (Beyer et al. 1997). Audet (1997) also found Pb concentrations to be higher in sediments than in vegetation collected from the same areas.

Sagittaria spp. tuber contamination in the CDARB may exist as the result of sediments clinging to the tuber skins as well as from metals incorporated into the tissue of the tuber. Therefore, the tubers may serve as a pathway of exposure to heavy metals for waterfowl feeding in contaminated wetlands. According to observations of feeding behavior, the birds dig into the sediment after the tubers, potentially ingesting large amounts of sediment as well as the tubers. Beyer et al. (1998) found that waterfowl ingest as little as 2% and up to 22% sediment as diet, possibly depending on the season, the plant species and other environmental factors. Audet (1997) reported swan sediment ingestion rates in the CDAB ranging from 0 - 70%. Beyer et al. (1997) also determined that Pb exposure of wood ducks in the CDARB depended primarily on the amount of Pb- contaminated sediment ingested rather than Pb in food items.

The tubers may also present a human health concern to tribal members gathering water potatoes for consumption, as exposure may occur via ingestion of tubers with fine sediment adhering to or

incorporated into skins, or from metals uptake into the pulp of the tubers. In addition tribal members may be exposed during the harvesting process, by handling sediments and inadvertently ingesting them, or by inhaling airborne particulates. The harvest of water potatoes occurs during the fall season. Water potatoes are typically collected by accessing a water potato patch (either by walking from the shore and then wading in the shallow waters/mud of the marsh, or by canoe or boat). Once the patch has been reached a digging implement is used to dig approximately 6 to 10 inches down into the sediment. Digging implements range from shovels and pitchforks to sturdy sticks. Once the sediment has been penetrated to the depth of 6 to 10 inches, the shovel full of sediment is brought to the surface. The sediment is then sorted through by hand to obtain the potatoes. Those that use sticks simply loosen the sediments with the stick and then use their hands to probe the sediment for water potatoes. Some persons wear gloves while others do not. The potatoes are then put in a sack and the process continues. Tribal members still gather water potatoes annually in wetland locations along the lower STJ River, which lies within current reservation boundaries, and has not been significantly impacted by mining activity. Although extensive *Sagittaria spp.* habitat is known to exist in the lateral lakes wetland complex along the lower CDA River, tribal members do not currently gather there because of the contamination that exists and the concern of heavy metals exposure.

Objectives

Due to the importance of this natural resource as both a food item to waterfowl and a cultural subsistence item to tribal members, it was determined that this investigation be designed with multiple objectives, and two different but compatible levels of sampling. Concentrations in

CDARB tubers vs. STJ River Basin (STJRB) reference area tubers, as well as metals concentrations associated with tubers collected in different wetlands in the CDARB, were evaluated to help determine which wetlands may pose a greater exposure risk to feeding waterfowl. In addition, sampling was designed to investigate the pathway of exposure, i.e., the tubers vs. the sediment in which the tubers are located. Therefore tubers from each location were analyzed both with skins (and potentially small quantities of adhering sediment) and without skins/sediment. Metals concentrations in tubers collected from individual wetlands were also compared to mean metals concentrations in sediments (Campbell et al. 1999) collected from the respective wetlands. To address human health concerns sampling was designed to simulate two different food preparation techniques used by tribal members, skinning tubers vs. eating tubers whole, to determine if removal of skins before consumption would potentially reduce exposure. In addition tubers were collected throughout the documented habitat in the CDARB and STJRB to evaluate metals concentrations associated with different potential gathering areas. Specific study objectives were as follows:

- determine if differences exist in metals concentrations in tubers between the CDARB and the STJRB;

- determine if differences exist in metals concentrations in tubers among individual wetlands in the CDARB and STJRB;

- determine if differences exist in metals concentrations between skinned and

unskinned tubers from CDARB and STJRB wetlands.

- determine if a correlation exists between metals concentrations in sediments from individual wetlands in the CDARB and STJRB (Campbell et al., 1999) and metals concentrations in tubers collected from the same wetlands.

Methods

Site Description

The CDAB (Figure 1) extends from the westernmost boarder of Montana across the panhandle of northern Idaho to the Spokane River. It includes the CDARB (CDA River, tributaries, and lateral lakes wetland complex), the STJRB (STJ River and its tributaries) and Lake CDA. Major sources of metals entering the lateral lakes originate approximately 25 km upstream, from the Silver Valley mining district.

The primary *Sagittaria spp.* habitat is located in palustrine emergent areas of the lateral lakes along the CDA River, and the palustrine emergent areas of the lake system adjacent to the lower STJ River (downstream from the town of St. Maries). The lateral lakes palustrine emergent habitat encompasses approximately 5900 acres, and this stretch of STJ River palustrine emergent habitat encompasses approximately 440 acres. The habitat inventory and sample collection

conducted for this investigation focused on these areas. The STJRB was selected as a reference area because of the lack of mining activity (and associated elevated metals concentrations in sediment) in the basin, the presence of similar wetland vegetation and waterfowl use, and tribal cultural concerns and use of the basin as a water potato gathering area.

Study Design

In the fall of 1994, personnel from the U. S. Fish and Wildlife Service (USFWS), Coeur d'Alene Tribe and the Bureau of Land Management conducted an inventory of *Sagittaria spp.* in the CDARB and STJRB for the purpose of mapping the distribution of the vegetation. Both *Sagittaria latifolia* and *Sagittaria cuneata* were included in the inventory, since no visual differences were observed in the tubers of the two species, both exist in waterfowl feeding habitat and both are gathered as water potato by the Tribe. Locations of patches that were identified to be a minimum of 1.5 m by 1.5 m were recorded in field logs and placed on a field map. Dimensions of each patch were also noted in field logs. Patches were physically marked where practical. The inventory was conducted via ground surveys on foot or from watercraft, as well as visual surveys from vehicles. There were limitations to mapping the distribution of the plant, therefore the total area mapped was considered a conservative estimate. These limitations included:

- physical inaccessibility (i.e., some areas could not be accessed via boat, canoe, hovercraft or kayak)
- waterfowl hunting activity in wetland areas

- only easily discernable patches (no less than 2.25 square meters) were mapped
- areas requiring access to private land were not inventoried
- ability to identify the decaying vegetative component of the plant at the time of the inventory (October); only plants that could be confirmed as *Sagittaria spp.* were included in the inventory

Sampling design was based on total habitat string for each river basin, i.e., the total length of all patches documented for each basin. A target number of samples (100 in the CDARB and 50 in the STJRB) would be collected at regular intervals along each habitat string, beginning at random starting points. A total of 4540 meters of habitat string were documented in the CDARB, including patches in Harrison Slough, Thompson Marsh, Thompson Lake, Anderson Lake, Blue Lake, Cave Lake, Medicine Lake, Moffit Slough, Hidden Marsh, Campbell Marsh, Killarney Lake and Strobl Marsh. A total of 555 meters were documented in the STJRB, including patches in Lake Chatcolet, Benewah Lake and Round Lake (Figure 2). For the purposes of this study, the Plummer Creek wetland was included as part of Lake Chatcolet, since single patches of *Sagittaria spp.* extended from one wetland into the other.

Before field collection began in each basin, patches were listed from upstream to downstream, with their respective lengths, to simulate the habitat string. A collection interval was generated by dividing habitat string length by target number of samples to be collected (100 in the CDARB, 50 in the STJRB); thus the interval was 45.4 meters for the CDARB and 11.0 meters for the STJRB. The first sampling point in the first interval was randomly generated between 0

and 45.1 meters for the CDARB and between 0 and 10.7 meters for the STJRB. Random numbers were selected within a range 0.3 meters short of the length of the sampling interval to ensure that the first sampling location fell within the first interval. Subsequent sample locations were identified every 45.4 meters and 11.0 meters, respectively, from upstream to downstream along the habitat string, i.e., sampling points were assigned to patches in an upstream to downstream order. The direction of sampling along the long axis within each patch was determined by flipping a coin.

Field Collection

Sagittaria spp. tubers were collected in November and December of 1994 from 11 of the 12 CDARB wetlands and all 3 of the STJRB wetlands (Figure 2) where *Sagittaria spp.* habitat was mapped. Anderson Lake was not sampled because only one patch was mapped there and the sampling interval was such that no sampling location fell within the patch. At each sample location within a wetland, tubers were collected along a 1 meter wide transect running perpendicular to the long axis of the patch and running the entire width of the patch (Figure 3). In general, the transect was divided into five approximately equal sections and each section excavated with a shovel until two whole tubers were located. One tuber was placed in a ziplock bag to be analyzed whole and the other placed in a ziplock bag to be analyzed with skins removed. Tubers were collected from each section until each bag contained approximately five tubers, or a total of ten tubers per sample location. If tubers were less than average size (approximately 6 cm in length, 4 cm diameter) enough were collected from each transect (a representative number from each section) to provide sufficient weight for analysis

(approximately 10 grams). If tubers were difficult to locate and fewer than ten were collected from the transect, adjacent transects were excavated at one meter, two meters, etc. from the original transect on alternating sides of the original transect until ten tubers were collected. If ten tubers were not collected by the time the transect extended five meters from the original transect then the number obtained was submitted for analysis. During field collection, staff avoided collecting tubers that had come in contact with the shovel. Tubers that were damaged by the shovel, chewed on by wildlife or otherwise damaged so that sediment was embedded in the pulp of the tuber, or decayed were not collected. Each bag was labeled in the field with date, location, patch number, transect number and the collectors initials. The bags of tubers were frozen at approximately 5° F upon return from the field, and stored frozen until processed.

Prior to shipment to the laboratory all tubers were thawed and cleaned with deionized water. One portion of each sample was skinned and the other left whole, in order to simulate two different tribal food preparation techniques, as well as to provide waterfowl exposure and pathway information. The skinned tuber scenario was intended to simulate a common tribal preparation technique where tubers are peeled before cooking or canning. In addition, the skinned tuber analysis may help to better define the pathway of heavy metals exposure to waterfowl (exposure via the tuber vs. exposure via sediment adhering to the skin). The whole tuber (skins left on) scenario was intended to simulate another common tribal preparation technique where tubers are simply washed before cooking . The whole tubers would also represent the form of tuber that would be ingested by waterfowl. This scenario was considered a conservative representation of sediment ingestion by waterfowl, since birds feeding on tubers in

the wild would likely ingest more sediment than was left on the prepared whole tuber samples.

Tubers to be skinned were thoroughly rinsed with deionized water and lightly scrubbed with a soft bristled brush until no sediment was visible on the surface. This was done to prevent introduction of sediment particles to the pulp of the tubers when the skin was removed. A 1 to 2 mm layer of skin was then peeled from the tubers with a plastic knife, removing all exposed surface material. The tubers were again rinsed with deionized water to remove any adhering skin or other particles. Tubers to be analyzed whole were dipped in a deionized water bath then sprayed lightly with deionized water, but not scrubbed, to remove visible sediment. Shoots and rhizomes were removed but all skin was left intact, along with any attached fibrous material. After cleaning each sample was placed in a clean, labeled bag, weighed, then frozen pending shipment to the laboratory.

A total of 145 tuber samples (95 from the CDARB and 50 from the STJRB) were submitted for analysis with skins on and 143 (93 from the CDARB and 50 from the STJRB) were submitted for analysis with skins off. One tuber sample, BL11-T9, collected from Blue Lake, was an unidentified tuber other than *Sagittaria latifolia* or *Sagittaria cuneata*. Although the plant associated with this tuber was not included in the habitat inventory, the sample was included in the metals analyses because this species of tuber has been observed in the ingesta contents of waterfowl found dead in the CDAB (J. Campbell, per observ.). This sample was smaller than the average *Sagittaria spp.* sample and therefore no skinned portion was submitted. Two other *Sagittaria spp.* tuber samples from Killarney Lake (sites KL4-T7 and KL4-T8) were

inadvertently mixed during the skinning process. Therefore the skinned portions of these samples were composited and submitted as a single sample (KL4T7/8S). The unskinned portions of these samples were submitted as unique samples (KL4-T7U and KL4-T8U).

Analytical Methods

Tubers were shipped on dry ice to Research Triangle Institute (RTI) for metals analysis. At the laboratory samples were preserved frozen until preparation for analysis. Due to the non-homogeneous nature of the tubers, the laboratory was instructed to homogenize the entire sample in a food processor prior to subsampling. A portion of the sample was then removed, freeze dried, ground in a mill and digested in a microwave with nitric acid. Sample digestates were analyzed for six metals, including aluminum (Al), Cd, iron (Fe), manganese (Mn), Pb and Zn, by Inductively Coupled Plasma (ICP) atomic emission spectroscopy. Metals values were reported in parts per million (ppm) on a wet weight (ww) and dry weight (dw) basis. Detection limits varied from sample to sample, likely due to the variable matrix (i.e., variable amounts of fibrous material and potential sediment on the tuber exteriors). Wet weight detection limits were generally in the ranges of 2.5 - 4.0 ppm for Al, 0.04 - 0.08 ppm for Cd, 4 - 9 ppm for Fe, 0.2 - 0.3 ppm for Mn, 0.40 - 1.8 ppm for Pb and 0.45 - 0.80 ppm for Zn. Dry weight detection limits were generally in the ranges of 8 - 10 ppm for Al, 0.15 - 0.25 ppm for Cd, 15 - 23 ppm for Fe, 0.6 - 0.9 ppm for Mn, 1.5 - 4.0 ppm for Pb and 1.5 - 2.1 ppm for Zn.

Field Quality Control Samples

Field duplicates were collected at an approximate frequency of 6% (nine duplicate pairs for the set of 145 unskinned samples and nine duplicate pairs for the set of 143 skinned samples), to measure field variability. Since it was not practical to homogenize this matrix and split samples, field duplicates were generated as sample replicates instead of split samples, as described in USFWS and EcoChem, Inc. (1995). Duplicates were generated by collecting four tubers from each of the five transect sections, thus generating two bags for skinned analysis and two for whole analysis. Field duplicates were prepared in an identical manner to the original samples and submitted blind to the laboratory. Relative percent difference (RPD) values were calculated for metals concentrations greater than ten times the respective detection limits and compared with the 50% criteria specified in the project Quality Assurance Plan (QAP) (USFWS and EcoChem, Inc., 1995). RPD values for two of the nine unskinned duplicate pairs were above the 50% criteria for Zn concentrations. RPD values for one of the same pairs were also above criteria for Cd, Fe, and Mn concentrations. The Zn RPD value for one of the nine skinned duplicate pairs and the Mn RPD value for another skinned duplicate pair were also above the 50% criteria. Field duplicates are an indicator of variability in field sampling as well as sample matrix. Due to the replicate collection procedure, increased variance in sample measurements was expected. Since the majority of the RPD values were within the 50% criteria for all six metals, and only one duplicate pair had more than one metal outside of criteria, there was no indication of problems with field sampling relative to any specific analyte of interest (e.g., there is no indication that sampling technique is inappropriate for reproducible measurements of Pb). Therefore, there were no expected data precision problems as indicated by field duplicates. The

consistently high RPD values for the one unskinned duplicate pair is more indicative of the potential for matrix variability, particularly associated with the skins of the tubers. This is likely due to the variable amounts of fibrous material found on tuber skins, which could entrap variable amounts of sediments. Since only one out of nine unskinned pairs (and no skinned pairs) demonstrated this variability, little impact on overall data quality was expected.

A sample of the deionized water used to clean the tubers prior to shipment to the laboratory was also submitted for analysis, to verify that no metals contamination occurred from the water source during sample processing. No positive result was reported by the laboratory for Al, Cd, Fe, Mn, Pb or Zn in this water sample.

Data Validation

Analytical data was validated for six metals of interest, including Al, Cd, Fe, Mn, Pb and Zn according to quality control (QC) criteria specified in the project QAP (USFWS and EcoChem, Inc., 1995) and to Environmental Protection Agency (EPA) guidelines for inorganic data review (USEPA, 1994). A portion of the data was subjected to 100% (full) validation in order to evaluate general data quality and to identify any analytical issues which may have affected it. The remainder of the data received limited validation which included review of any issues of concern identified in the full validation. Data validation was performed by EcoChem, Inc.

In general, all data was evaluated to be acceptable for its intended use (EcoChem, Inc., 1997a; EcoChem, Inc.,1997b) as specified in the study objectives. A number of analytical issues were

reported which resulted in qualification of the data. Background concentrations of several analytes were identified in some laboratory preparation blanks, which were prepared and analyzed with the tuber samples. This indicated potential background contamination of laboratory systems. Data which may have been affected were identified and qualified as non-detected at the reported concentrations, since these concentrations may be largely attributable to laboratory contamination in addition to, or instead of, metals actually in the samples. Methods for analyzing non-detected data are discussed in statistical methods below. Additional data was qualified as estimated due to slight biases in accuracy. General high biases existed for Fe and Zn data for unskinned tubers, and for Pb data for unskinned tubers in the assessment area only, when matrix spike recoveries were evaluated.

Post-digestion spikes (analyzed to measure matrix effects and used to generate a correction factor to compensate for matrix effects) indicated a low but variable bias, particularly for unskinned tuber data. The laboratory applied a correction factor to the data to compensate for potential low recoveries indicated by the post-digestion spike data. Since post-digestion spike recoveries were as low as 53% (unskinned data) and as high as 86% (skinned data) comparisons were run on Pb data to see if variable recoveries impacted data usability, i.e., if significant differences existed between CDARB data and STJRB data, both with and without correction factors applied. An extreme-case comparison was made between CDARB unskinned data without Pb correction factors and STJRB unskinned data with Pb correction factors applied. A significant difference existed between CDARB and STJRB data, both in the extreme test and in a comparison of the corrected data sets. Therefore it was determined that post-digestion spike recoveries did not

impact data usability and correction factors were left in place.

The standard reference material (SRM) associated with unskinned tubers also indicated a low bias for Pb, although the SRM matrices (citrus leaves and peach leaves) were not as representative of the sample matrix as were the matrix spike and post-digestion spike QC samples.

Finally, high variability existed in Pb data for unskinned tubers, as indicated by laboratory duplicate samples, resulting in qualification of data as estimated. This was attributed to the non-homogeneous nature of the exterior of the tuber, as some tubers had large amounts of fibrous material on the skin (which could also trap more sediment) and others did not. The laboratory reported inability to homogenize the unskinned tubers. Tubers with skins removed provided a more homogeneous matrix, and better precision data was generated with the skinned tuber data set.

Although these issues were identified in the data review process as potentially imposing some bias to the data, it was determined that none had impact on data usability. Data values were adjusted when appropriate, i.e., to compensate for laboratory background contamination and for low post-digestion spike recoveries due to matrix effects. Other QC checks (including SRM recovery outliers and laboratory duplicate outliers) warranted qualification of additional data to advise of potential bias for interpretive purposes only, but did not warrant adjusting data values. In general, laboratory difficulties in meeting some QC criteria can be attributed to the variable

nature of this sample matrix. The fibrous component (and associated sediment component) of tuber exteriors varied from sample to sample, potentially contributing to variability in sample to sample measurements. In addition, this material created difficulties in sample homogenization, potentially creating within sample variability. In order to evaluate the potential impacts of this matrix on study results, we ran statistical comparisons on the most conservative data scenario and saw no differences in results. We therefore concluded that analytical issues had no impact on conclusions of this investigation. Since overall laboratory performance and data quality was considered acceptable to meet all study objectives (EcoChem, Inc., 1997a; EcoChem, Inc., 1997b) we accepted the data as reported by the laboratory for use in our analyses.

Statistical Methods

Statistical comparisons were run on unskinned and skinned tuber data for Al, Cd, Fe, Mn, Pb and Zn to compare :

- metals concentrations in unskinned tubers from the assessment area to those from the reference area;
- metals concentrations in unskinned tubers from each wetland to sediment data for that wetland;
- metals concentrations in skinned tubers from the assessment area to those from the reference area;
- metals concentrations in skinned tubers from each wetland to sediment data from that wetland; and

- metals concentrations in unskinned tubers to metals concentrations in skinned tubers for each wetland.

Inference from these data are to the *Sagittaria spp.* habitat identified prior to sample collection. Inferences to the identified habitat patches are made under the assumption that specimens collected are a random sample of specimens from the identified habitats. We also assume that the habitats identified prior to sampling are a representative sample from all available *Sagittaria spp.* habitats. All analyses were conducted using ww ppm values except where tuber concentrations were compared to sediment concentrations, in which case dw ppm was used for both.

Statistical comparisons of metal levels between STJRB and CDARB and between each CDARB wetland and the entire STJRB were made using Wilcoxon's two sample rank sum test (Lehmann, 1975; Hollander and Wolfe, 1973; Mann and Whitney, 1947; Wilcoxon, 1945). Wilcoxon's two sample rank sum test was performed by ranking observations from both samples, replacing any ties (note treatment of concentrations below analytical detection limits, below) with the average rank, and summing ranks over one of the samples. One-tailed significance of the rank sum was assigned using the normal approximation given by Lehmann (1975). The null hypothesis in each case was that the distribution of concentrations in the CDARB was stochastically lower or equal to the distribution of concentrations in the STJRB. The alternative hypothesis was that the CDARB distribution was stochastically higher than that of the STJRB. Reported p-values in the range 0.90 to 1.0 can be considered significant in the opposite direction (i.e., the STJRB was stochastically higher than the CDARB). All computations were carried out using S-PLUS and

their testing routine wilcox.test (S-PLUS version 3.3 for Windows, © 1995 MathSoft, Inc.).

Correlation and regression analyses were conducted to investigate the relationship between mean metal (Cd, Fe, Mn, Pb and Zn) concentrations in tubers and in sediments (Campbell et al., 1999). Mean tuber and sediment concentrations were calculated from sample data within each of 14 wetlands where tubers were collected. The resulting 14 pairs of mean concentrations in tuber and sediment samples were then plotted and simple linear regression models were fit for each metal using tuber concentration as the dependent variable and sediment concentrations as the independent variable. A separate model was fit for each analyte. Fitted regression lines were plotted on the same axes. Certain metal concentration levels were reported as "below detection limit" (BDL) by the laboratory and in an effort to reduce the influence of BDL observations on the correlation analyses, calculated means were also ranked and the Spearman rank correlation coefficient was calculated for each analyte.

To analyze the effects of skinning, skinned tuber samples were paired with unskinned tuber samples collected at the same location and differences in metal concentrations were assessed using Wilcoxon's signed rank procedure (Lehmann, 1975; Hollander and Wolfe, 1973). Signed rank tests were computed for all samples combined across all wetlands in the reference and assessment areas and for each wetland from which two or more paired tuber samples were collected. There were three sites from which either the skinned or unskinned value could not be matched with a corresponding sample (BL11-T9, no skinned value; KL4-T7 and KL4-T8, no pairing because skinned tubers were inadvertently mixed). In total there were 92 paired samples

from the assessment area and 50 from the reference area. Exact significance levels were computed if no (non-zero) ties existed within a pair and if there were fewer than 25 pairs, otherwise a normal approximation was used to assign significance and, in the presence of ties, the normal approximation given by Lehmann (1975) was used. All computations were carried out in S-PLUS using their routine wilcox.test with the option paired=T (S-PLUS version 3.3 for Windows, © 1995 MathSoft, Inc.). Raw paired differences were plotted to aid interpretation.

In cases where concentrations of metals were reported as BDL the laboratory reported the detection limit of the test and a qualifier specifying that the true level was below this limit. When reporting average concentrations and for graphing purposes, a value of one-half the detection limit was used in place of all BDL observations. For statistical comparisons using Wilcoxon's two sample procedure, all values (both above and below detection) in the two samples which were below the maximum detection limit of the two samples were treated as tied. For each paired test comparing metal concentrations in skinned and unskinned tubers, the largest detection limit among those values reported as BDL in both samples was computed and any value (reported as BDL or not) which was below this maximum was replaced with this maximum. For analyses correlating metal concentrations in tubers with metal concentrations in sediments (Campbell et al., 1999), concentrations reported as below analytical detection limit were replaced with a uniform random number between zero and the detection limit. Means used in the correlation analysis were then calculated including the random uniform numbers. Use of a uniform random number for values below detection accurately represents both location and variance of the resulting wetland mean, a requirement for the correlation analysis. Again, the

means were also ranked and the Spearman rank correlation coefficient was calculated in order to reduce the influence of BDL observations.

Results

Unskinned Tubers (CDARB vs. STJRB)

Mean and median metal levels in unskinned samples from each basin appear in Table 1, along with the number and percentage of BDL values. Overall Pb, Zn, Cd, Fe and Mn levels were significantly higher in unskinned tuber samples collected in the assessment basin compared to those collected in the reference basin (all p-values <0.001, Wilcoxon test, Table 2). Aluminum levels were significantly lower in the assessment basin compared to the reference basin ($p > 0.999$, Wilcoxon test, Table 2).

Tests comparing the distribution of metals in unskinned tuber samples from assessment wetlands to the entire sample from the reference basin appear in Table 2. Lead, Zn, Cd, and Mn distributions in all assessment wetlands was significantly higher than the corresponding metal distribution in tubers from the reference area (largest p among 44 tests = 0.048, Table 2). In general, the distribution of Al was lower in the assessment wetlands when compared to the corresponding reference distribution ($p > 0.90$ for all wetlands except Thompson Marsh ($p=0.62$) and Cave Lake ($p=0.16$), Table 2). Tests involving Fe were less conclusive with Blue Lake,

Campbell Marsh, Hidden Marsh, Harrison Slough, Killarney Lake, and Strobl Marsh having higher concentrations than the reference area (all p-values < 0.054) while the distribution of Fe in Medicine Lake was lower than in the reference area ($p = 0.945$). Box plots representing the distribution of concentrations in unskinned tubers from every wetland under study appear in Figure 4, 5, 6, 7, 8, and Figure 9. Note that a single sample was taken from Thompson Marsh, Cave Lake, and Medicine Lake in the assessment area and Benewah Lake in the reference area.

Skinned Tubers (CDARB vs. STJRB)

Mean and median metal levels, as well as number and percent of BDL values, for skinned tuber samples from each basin appear in Table 1. Overall Cd, Mn, and Zn levels were significantly higher in skinned tuber samples from the assessment basin (all $p < 0.001$, Wilcoxon test, Table 2). Aluminum and Fe levels were higher in skinned tubers from the reference basin (both $p > 0.999$, Wilcoxon test, Table 2). The distribution of Pb in skinned samples was marginally higher in the assessment area ($p=0.185$, Wilcoxon test, Table 2). Due to the large proportion of values below analytical detection limits, the test for Pb, to a large extent, is testing for differences between the two detected values from the assessment basin and the maximum detection limit (recall all values below the maximum detection limit were tied in this analysis).

Tests comparing the distribution of metals in skinned tuber samples from assessment wetlands to the entire sample from the reference basin appear in Table 2. Every wetland in the assessment basin contained higher levels of Cd, Mn, and Zn in skinned tubers relative to all tubers from the reference basin (largest $p = 0.055$, Table 2). Nearly every wetland in the assessment basin

contained lower levels of Al and Fe in skinned tubers relative to all tubers from the reference basin (all $p > 0.90$) with the exception of Cave Lake (Al $p=0.798$, Fe $p=0.62$), Medicine Lake (Al $p=0.368$), Thompson Lake (Al $p=0.874$), and Thompson Marsh (Al $p=0.798$). Several relatively large readings of Al and Fe were obtained from Lake Chatcolet and Round Lake, both of which are reference wetlands (Figure 10 and Figure 12). Lead concentration distributions in skinned tubers from every assessment wetland were virtually identical to that from the reference basin with the exception of the two wetlands where values above the maximum detection limit were obtained; Blue Lake and Moffit Slough ($p=0.081$ and 0.028 respectively). Box plots representing the distribution of concentrations in skinned tubers from every wetland under study appear in Figure 10, 11, 12, 13, 14, and Figure 15.

Unskinned vs. Skinned Comparisons

Significance levels of the Wilcoxon signed rank tests comparing metal concentrations in unskinned tubers to metal concentrations in skinned tubers appear in Table 3. Overall, skinned tubers contained significantly lower concentrations of all six metals than unskinned tubers (all $p<0.001$, last row Table 3). If the same comparison is repeated on each wetland, those wetlands with five or more skinned-unskinned sample pairs showed lower concentrations of metals in skinned tubers when compared to unskinned tubers (all $p<0.062$ for all metals, except Killarney Lake Cd $p=0.116$, Table 3). Those wetlands where comparisons were not statistically significant also had small sample sizes (< 5 skinned-unskinned sample pairs). Lack of significant differences does not indicate that metals concentrations in skinned and unskinned samples from these wetlands are similar, but simply that there is insufficient data to determine if differences

exist. In several cases, the concentration of Pb in skinned tuber material was more than 100 ppm lower than the concentration of Pb in whole tubers collected at the same location. Similarly, in several cases the amount of Zn in skinned tuber material was more than 60 ppm lower than in whole tubers collected at the same location.

Tuber vs. Sediment

Iron, Mn and Pb concentrations in unskinned tubers were found to be positively correlated with corresponding sediment concentrations ($p < 0.007$ Spearmans rank correlation, Table 4). Although not strongly correlated, Zn concentrations in tubers were also positively related to Zn concentrations in sediment ($p=0.31$, Table 4). Similarly, regression coefficients for Fe, Mn, and Pb calculated using parametric statistical analyses were positive ($p < 0.04$, Table 5) indicating the same positive relation between metals concentrations in tubers and corresponding sediments. A summary of the analyses for unskinned tubers can be found in Tables 4 and 5, and the data are plotted in Figure 16. Manganese, Pb and Zn concentrations in skinned tubers were positively correlated with corresponding sediment metals concentrations (Table 4 and Table 5). The strength of correlation for Mn was statistically significant at the $p=0.04$ level (Spearman's, Table 4). A summary of the analyses for skinned tubers can be found in Tables 4 and 5, and the data are plotted in Figure 17.

Discussion

Waterfowl Exposure to Lead

We found that waterfowl feeding in *Sagittaria spp.* habitat in the CDARB have greater exposure to Pb from eating the tubers than waterfowl feeding in the STJRB. This is demonstrated by the significantly higher Pb concentrations in unskinned CDARB tubers compared to unskinned STJRB tubers, for every CDARB wetland sampled. Documented sediment Pb concentrations are significantly higher in the CDARB than in the STJRB (Campbell et al., 1999). Beyer et al. (1998) determined that waterfowl in the CDAB ingest substantial quantities of sediment as part of their diet (mean sediment ingestion rates up to 22%). Tundra swan sediment ingestion rates in the CDAB have been documented as high as 70% (Audet, 1997). In addition, biologists observations have reported that birds ingest *Sagittaria spp.* tubers (with skins and adhering sediment). These findings again indicate that waterfowl feeding on *Sagittaria spp.* tubers in the CDARB have greater exposure to Pb from eating the tubers than waterfowl eating tubers in the STJRB.

Lead distributions in tubers from the CDARB were only marginally higher than in the STJRB when skins were removed. There is also an overall significant difference between Pb concentrations in skinned and unskinned tubers. This indicates that Pb is associated primarily with the tuber skins, and is not taken up by the tuber pulp to any great extent. In addition, Pb concentrations in unskinned tubers were found to be strongly correlated with sediment Pb

concentrations (Campbell et al., 1999) in the wetlands from which they were collected. Lead concentrations in skinned tubers were also positively correlated with Pb concentrations in sediments from corresponding wetlands, however the strength of the correlation was weak. These findings support the conclusions of Beyer et al. (1997) that sediment is the primary source of Pb and the pathway of exposure. Tuber skins have considerable amounts of fibrous material which is in contact with the sediment while in the ground, and which could entrap various amounts of sediment, even after being rinsed. Neufeld (1987) supports this by attributing the retention of sediment on equisetum (and thus the significant increase of Pb in swan diets) to the rough surfaces of the plant. To more closely simulate what a bird would ingest, unskinned tubers analyzed for this investigation were rinsed but not scrubbed, and fibrous material was left intact, possibly along with adhering sediment. This was considered a very conservative representation of Pb exposure, since birds feeding on the tubers potentially ingest much larger quantities of sediment than were left on the study tubers.

Mean Pb concentrations for unskinned tubers collected in the CDARB ranged from 3.6 ppm ww in Cave Lake to 86.3 ppm ww in Harrison Slough. The highest tuber Pb concentration in any CDARB wetland was 126.8 ppm ww, in Moffit Slough. In comparison, (Neufeld, 1987) reported Pb in *Sagittaria spp.* tubers from the CDARB up to 400 ppm. There was, however, no indication of tuber cleaning procedures and it is possible that sediment removal from the tuber skins was not equivalent. Krieger (1990) reported Pb concentrations in tubers up to 810 ppm. Based on this report's description of sample preparation, Krieger's cleaning procedures were approximately equivalent to those used in this investigation. It is possible that the tubers with

the most elevated metals concentrations were sampled from areas with the most elevated sediment metals concentrations within the CDARB wetlands. In general, mean tuber Pb concentrations from the Krieger study were elevated when mean sediment Pb concentrations from the same sites were elevated, although most mean sediment concentrations were at least 10X higher than the mean tuber concentrations from the same sites. Neufeld (1987) also reports Pb uptake by tubers, although no skinned vs. unskinned analyses was reported. As stated previously, the skinned vs. unskinned analyses performed as a part of this investigation indicate that there is no significant uptake of Pb by the tuber pulp, but that Pb concentrations are primarily associated with tuber skins and adhering sediment.

Lead concentrations in *Sagittaria spp.* tubers were evaluated as part of a Pb uptake study at various wildlife refuges in the United States (Behan et al., 1978). In this investigation *Sagittaria spp.* tubers along with other aquatic plants were collected from Pb shot areas at Squaw Creek and Ravalli National Wildlife Refuges. *Sagittaria latifolia* tuber concentrations were averaged with concentrations in river bullrush (*Scirpus fluviatilis*) and American lotus (*Nelumbo lutea*) for a mean concentration of 12.0 ppm dw. This value is comparable to the mean unskinned tuber concentration from Cave Lake (11.7 ppm dw, based on an average unskinned tuber percent moisture of 69.1%), but well below other CDARB wetland means, on a dw basis. *Sagittaria cuneata* was also collected in this study from a Pb shot seeded area and a control area. Mean concentrations for the seeded area were 7.4-7.5 ppm dw, comparable to the lowest CDARB wetland means. The control area mean concentration was <1.2 ppm dw, comparable to the STJRB unskinned tuber range of 0.94-1.1 ppm dw. Behan et al. (1978) also concluded that

uptake by aquatic plants of Pb from sediments containing Pb shot or metallic Pb is not sufficient to cause Pb poisoning, and that Pb exposure of waterfowl is due primarily to the source of Pb (Pb shot or leaded sediment).

Waterfowl Exposure to Other Metals

Zinc, Cd and Mn concentrations were also significantly higher in unskinned tubers from all CDARB wetlands where tubers were sampled than in unskinned tubers from the STJRB, indicating that waterfowl feeding on the tubers in the CDARB have greater exposure to these metals than waterfowl feeding in the STJRB. However, there are still significant differences in concentrations of these metals between the CDARB and STJRB when the tubers are skinned. This indicates that there is uptake of Zn, Cd and Mn to some extent by the tuber from the sediment, and that these levels do not normally exist in the tubers. It appears as though the tubers themselves serve as a pathway of exposure to Zn, Cd and Mn, as well as the sediment (as there are still overall significant differences between unskinned and skinned tubers for all of the metals).

In general, Al concentrations are higher in both unskinned and skinned tubers from the STJRB than in unskinned and skinned tubers from the CDARB. This may be due to sediment Al concentrations which have been documented as being higher in the STJRB (and in pre-mining CDARB sedimentary layers) than in sediment deposits resulting from mine tailings from the Silver Valley (Box, personal comm.). The data indicate that tubers uptake some Al, although concentrations are significantly reduced when tubers are skinned. Tests with Fe concentrations

were less conclusive. Overall, Fe concentrations were found to be significantly higher in unskinned tubers collected from the CDARB than in unskinned tubers collected from the STJRB, and significantly lower in skinned CDARB tubers than in STJRB tubers.

Tribal Significance

From a human consumption standpoint, these data suggest that eating tubers from wetlands in the CDARB without removing skins would present exposure to significantly higher levels of Pb and other metals than eating unskinned tubers from the STJRB.

We found that exposure to metals via ingestion of tubers collected in the CDARB could be significantly reduced by skinning the tubers. Detectable Pb levels were found in only two skinned samples from two wetlands in the CDARB, and as stated above, mean Pb concentrations from these wetlands were greatly reduced by skinning. Overall, Pb concentration distributions in the CDARB were the same as for the STJRB for skinned samples.

If cleaned reasonably well with metals-free water, no real benefit related to Pb exposure would be derived from skinning tubers collected in the STJRB, since no detectable levels of Pb were found in any tubers, skinned or unskinned, from the wetlands sampled in that basin.

It should be considered that concentrations of Pb and other metals in sediments in CDARB wetlands have been found to be significantly higher than in STJRB wetlands (Campbell et al., 1999). Exposure to Pb and other metals via sediment contact may be increased during the

process of gathering tubers in the CDARB vs. gathering tubers in the STJRB, even if tubers are to be skinned prior to eating. In general, gathering tubers from wetlands in the CDARB for consumption with or without removing skins would present exposure to significantly higher levels of Zn, Cd and Mn than eating tubers from the STJRB. However, removing skins of tubers gathered in the CDARB does significantly reduce the concentrations of all three metals overall.

Conclusions

Numerous studies have reported Pb poisoning as a cause of extensive waterfowl mortalities in the CDARB (Blus et al., 1991; Krieger, 1990; Neufeld, 1987; Chupp and Dalke, 1964). These studies suggested contaminated vegetation or sediments as the source of Pb exposure. *Sagittaria* tubers are a major food item for waterfowl in the CDARB and in the STJRB. They are also gathered by Coeur d'Alene tribal members, currently in the STJRB. This investigation was conducted to evaluate exposure of CDARB waterfowl which feed on *Sagittaria* tubers to Pb and other metals, and to evaluate metals concentrations that tribal members would be exposed to if they consumed tubers from the CDARB. We found that:

- waterfowl feeding on CDARB *Sagittaria* tubers have greater exposure to Pb, Cd, Mn and Zn than waterfowl feeding on STJRB tubers. Mean tuber Pb concentrations in the CDARB wetlands we sampled ranged from 3.6 - 86.3 ppm ww;
- lead concentrations detected in CDARB *Sagittaria* tubers are primarily associated with the skins of the tubers, and our findings suggest that sediments (probably adhering to skin

or fibrous material) are the primary source of Pb. There is some uptake of Al, Cd, Mn and Zn into the pulp of CDARB tubers, although concentrations of these metals are still higher in tubers with skins than tubers without skins;

- humans would be exposed to significantly higher levels of Pb, Cd, Mn and Zn by eating CDARB *Sagittaria* tubers with skins, as opposed to eating STJRB tubers with skins. Lead exposure from ingestion of tubers from most CDARB wetlands can be minimized to levels comparable to STJRB tubers, by removing skins prior to eating. However the potential for human exposure to Pb and other metals, via contact with sediments during the process of gathering CDARB tubers, will exists as long as sediment metals concentrations remain elevated in the CDARB.

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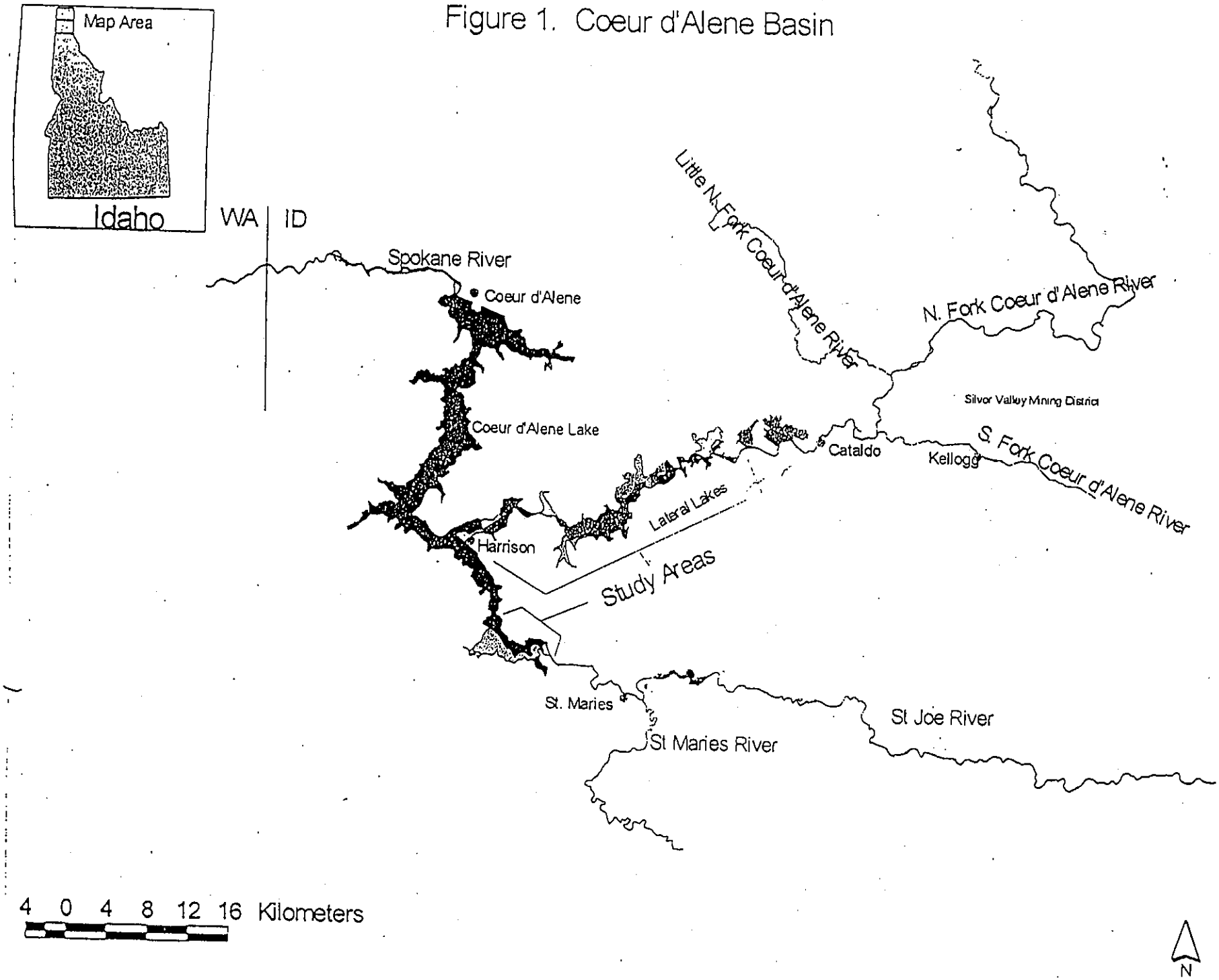
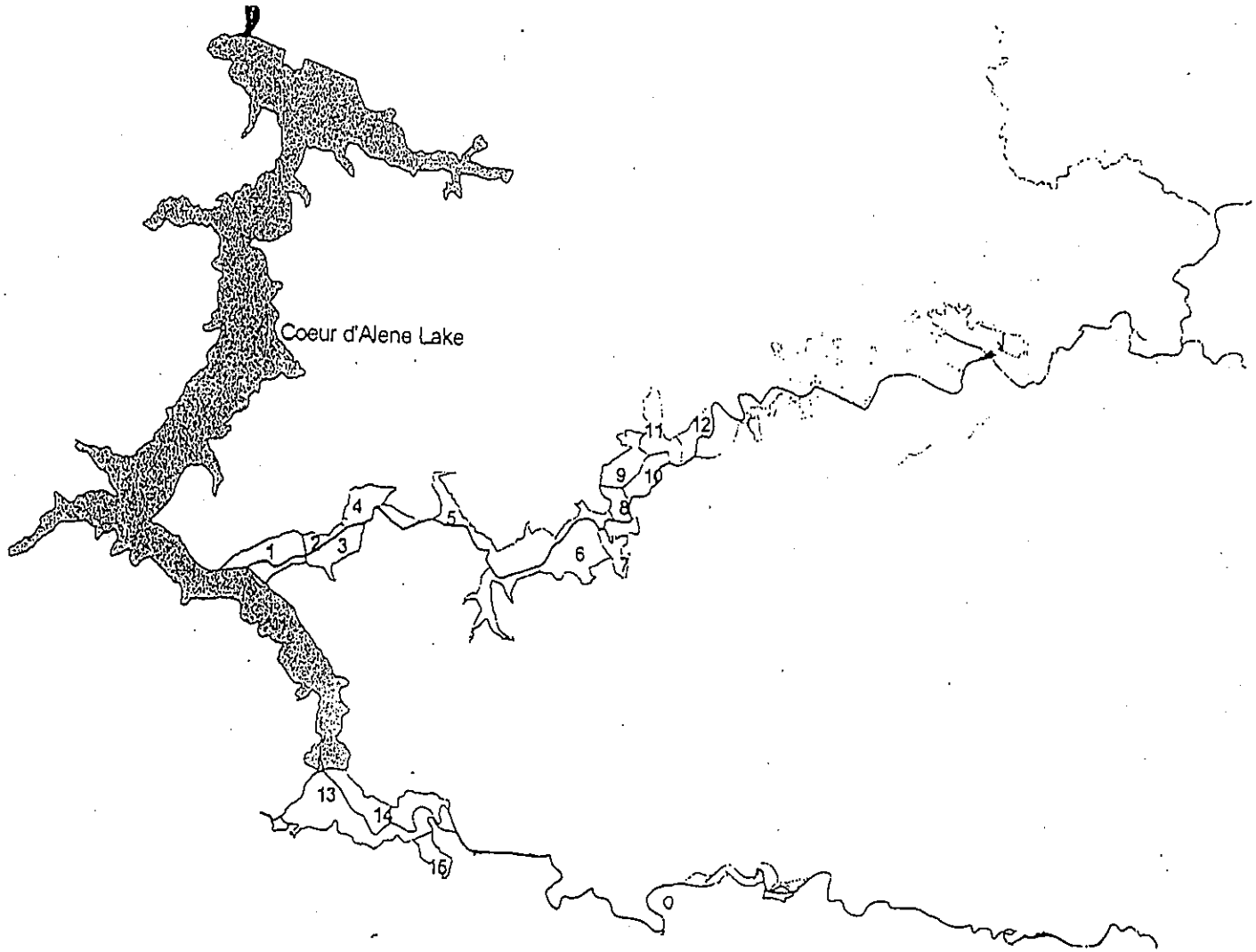


Figure 1. Coeur d'Alene Basin

Figure 2. Wetland units included in 1994 Sagittaria habitat inventory -- Coeur d'Alene and St. Joe River Basins



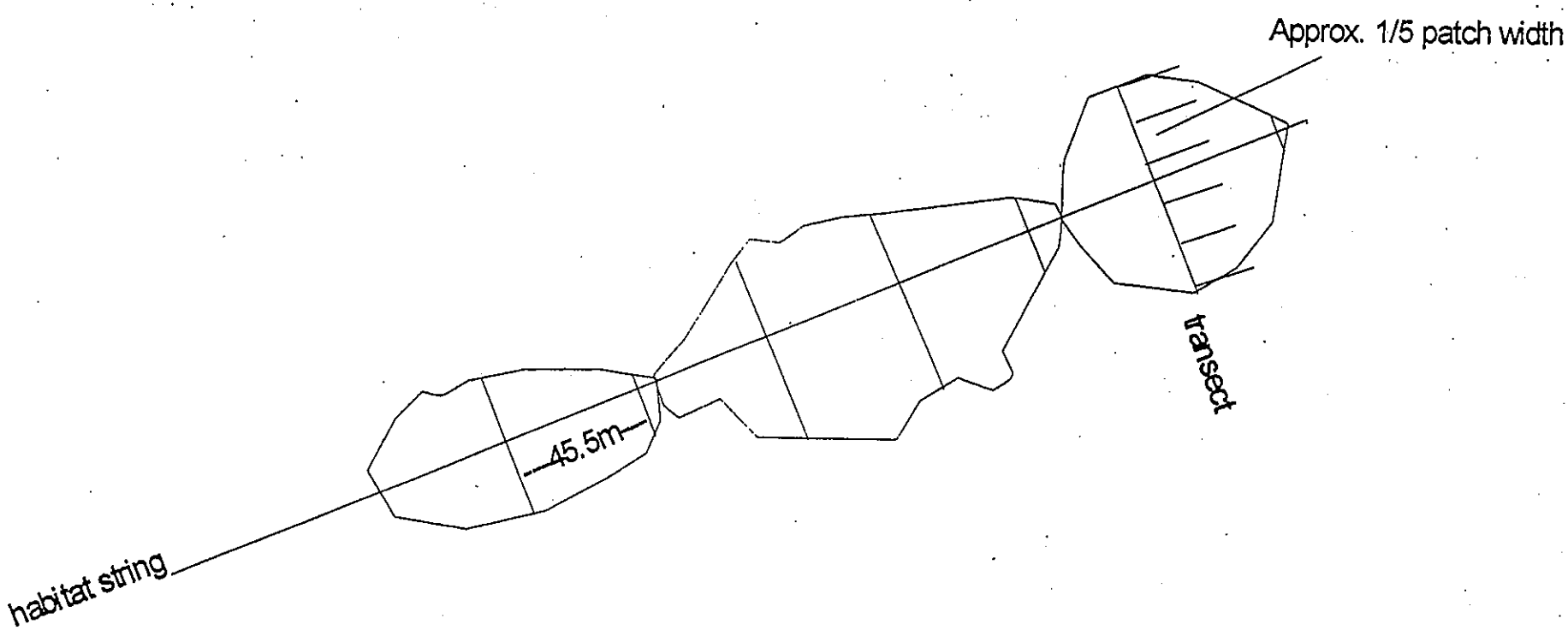
Wetland Units

1. Harrison Slough
2. Thompson Marsh
3. Anderson Lake
4. Thompson Lake
5. Blue Lake
6. Cave lake
7. Medicine Lake
8. Moffit Slough
9. Hidden Marsh
10. Campbell Marsh
11. Killarney Lake
12. Strobl Marsh
13. Lake Chatcolet
14. Round Lake
15. Benewah Lake

2 0 2 4 6 8 Kilometers

N
W
E
S

Figure 3. Sample design for tuber collection (e.g. CDARB)



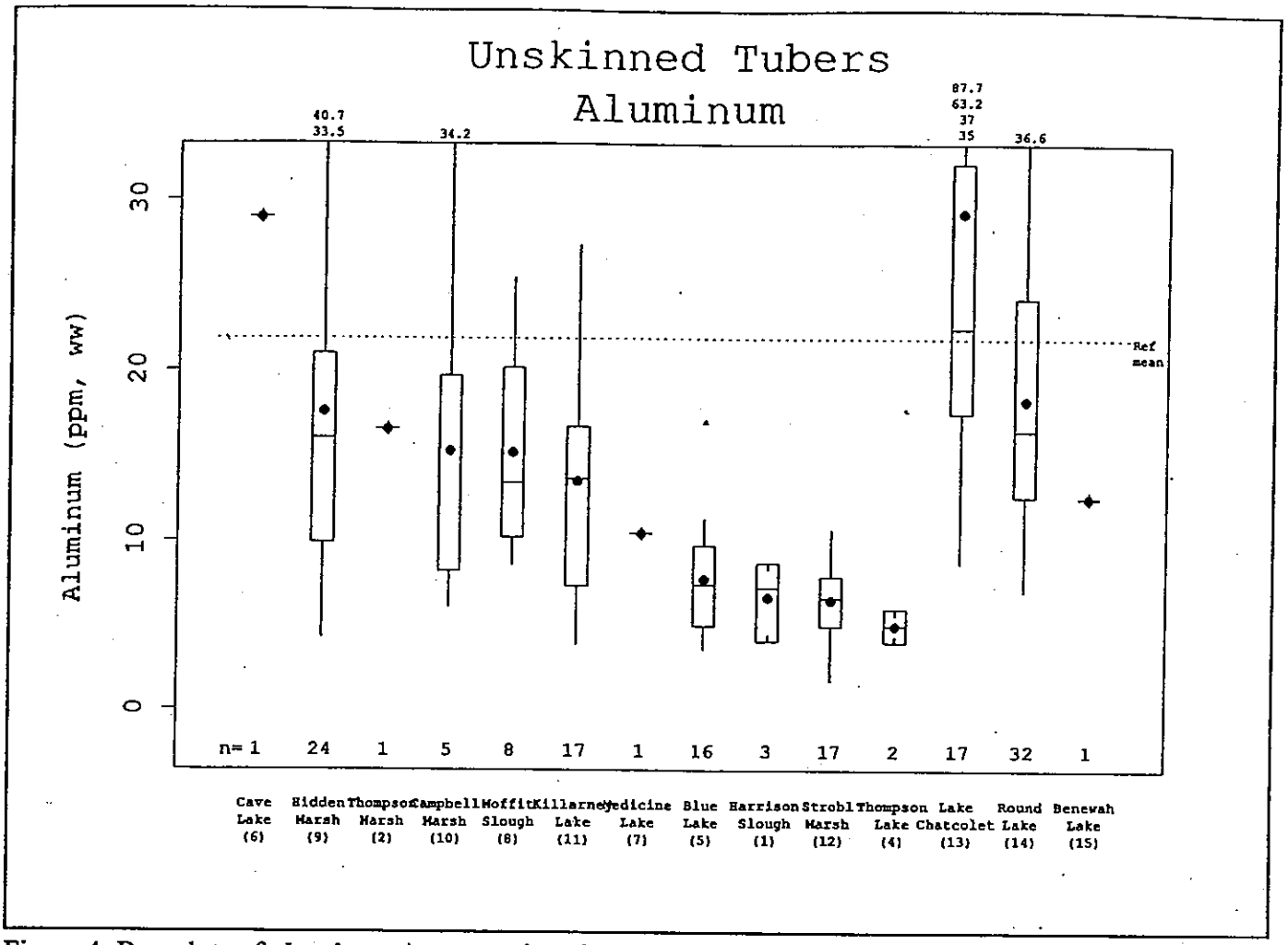


Figure 4: Box plots of **aluminum** concentrations in **unskinned** tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

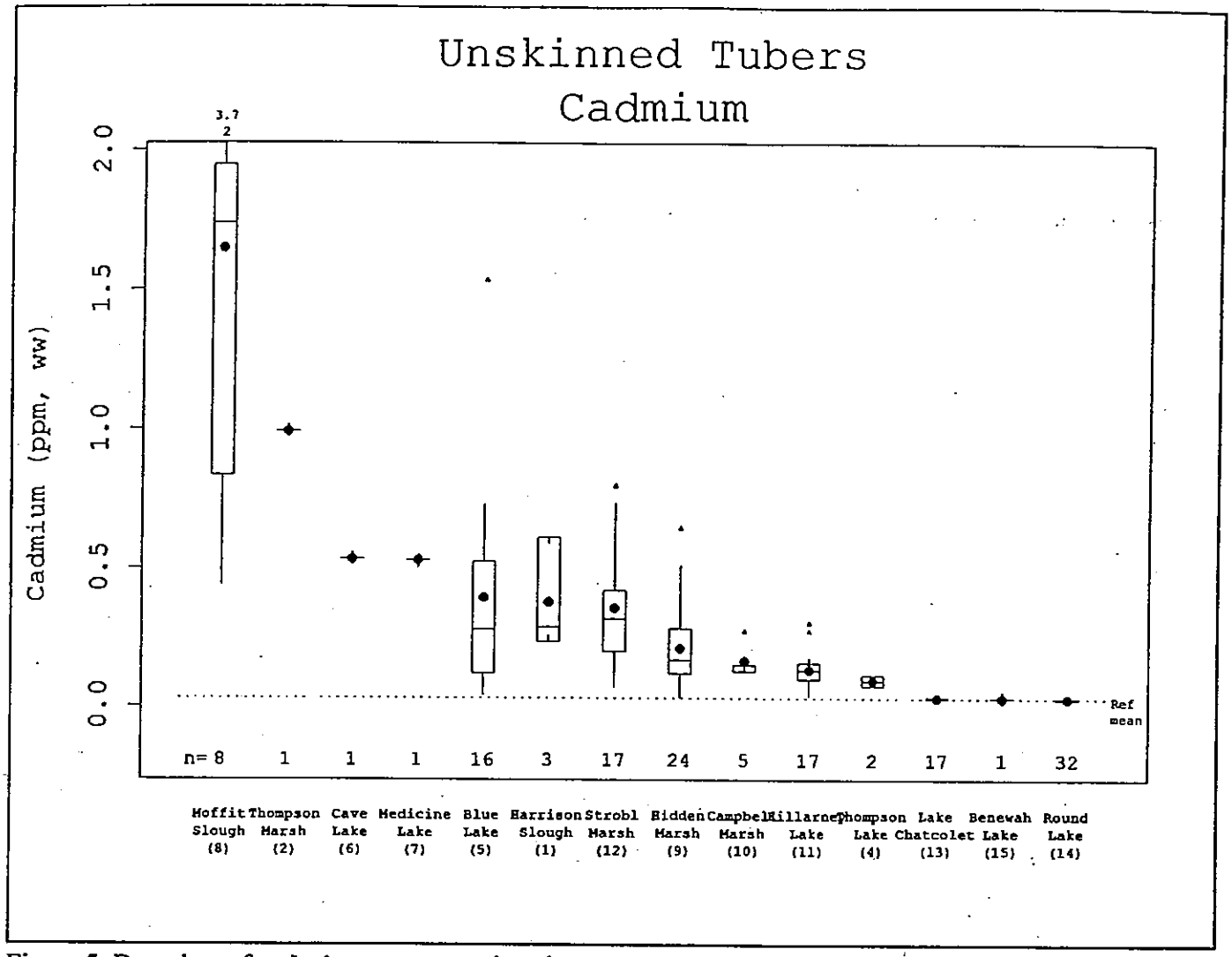


Figure 5: Box plots of **cadmium** concentrations in **unskinned** tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

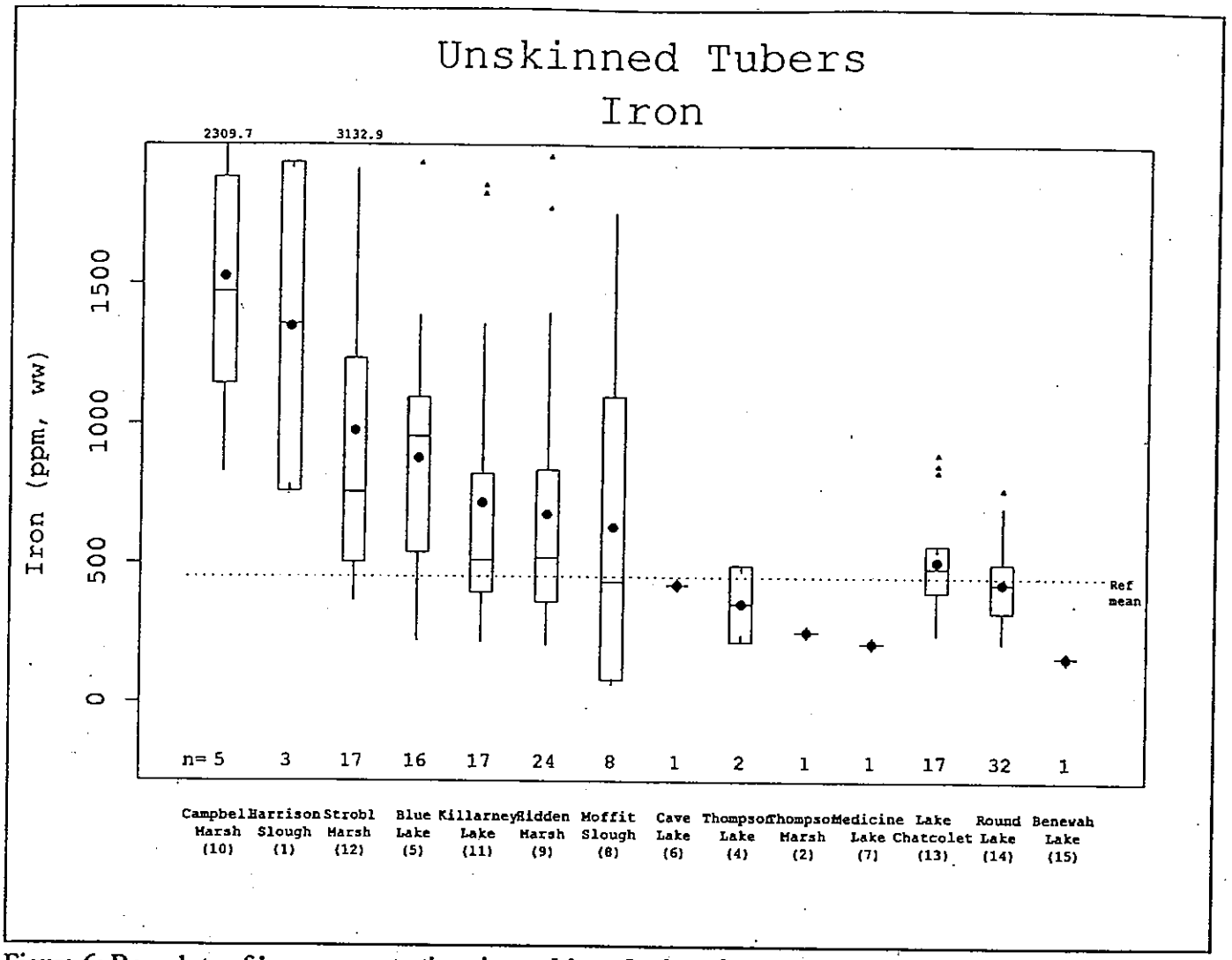


Figure 6: Box plots of **iron** concentrations in **unskinned** tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

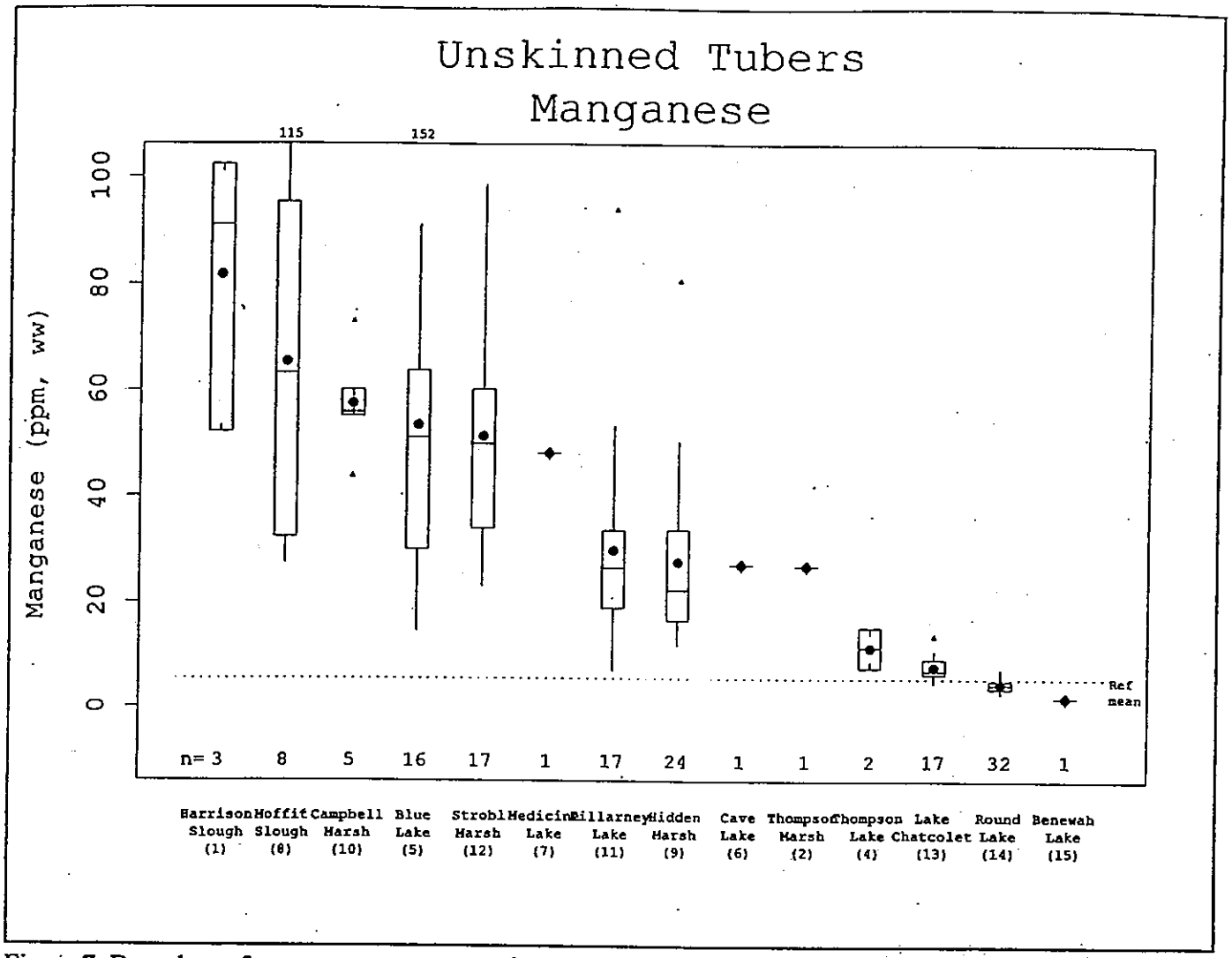


Figure 7: Box plots of **manganese** concentrations in **unskinned** tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

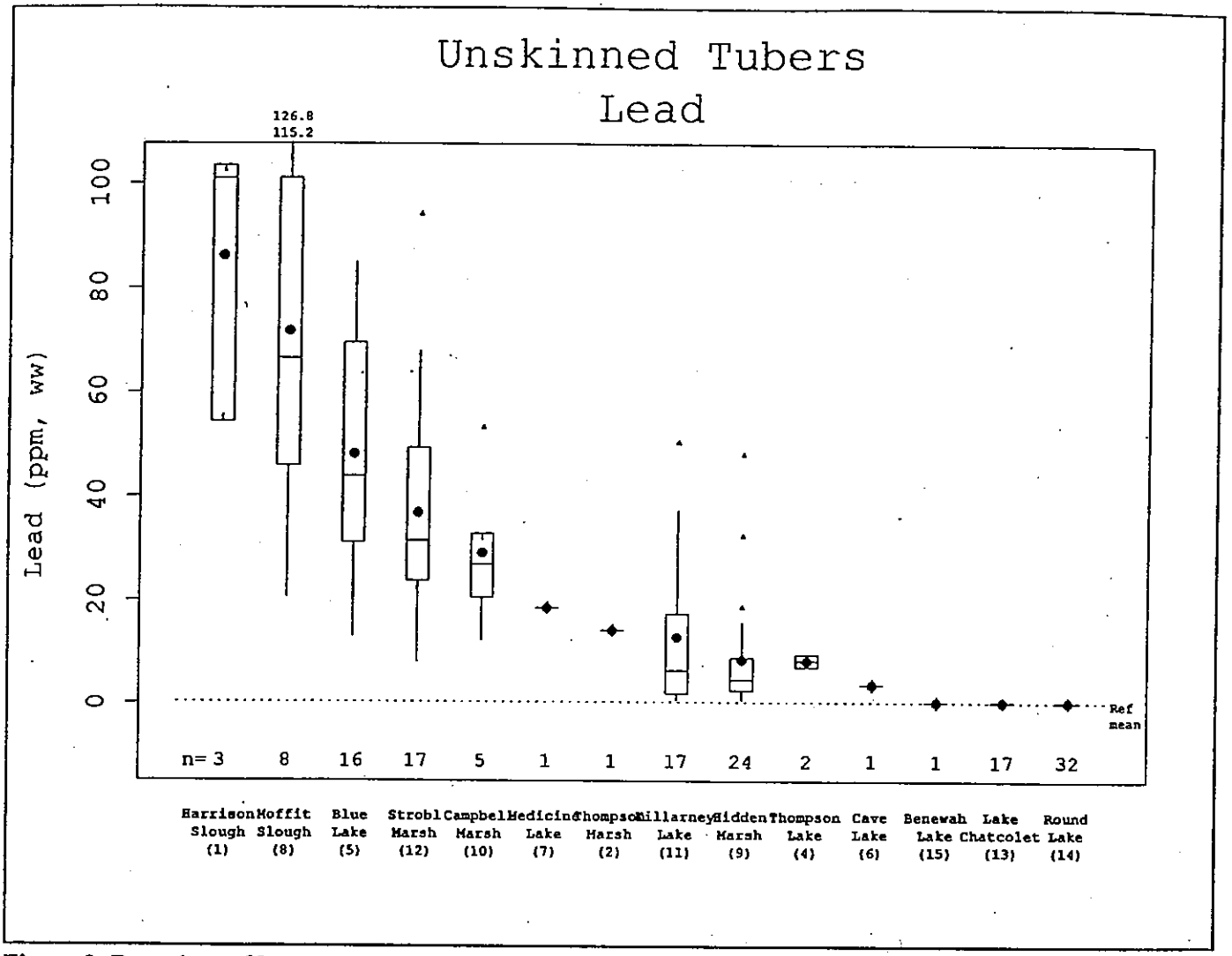


Figure 8: Box plots of **lead** concentrations in **unskinned** tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

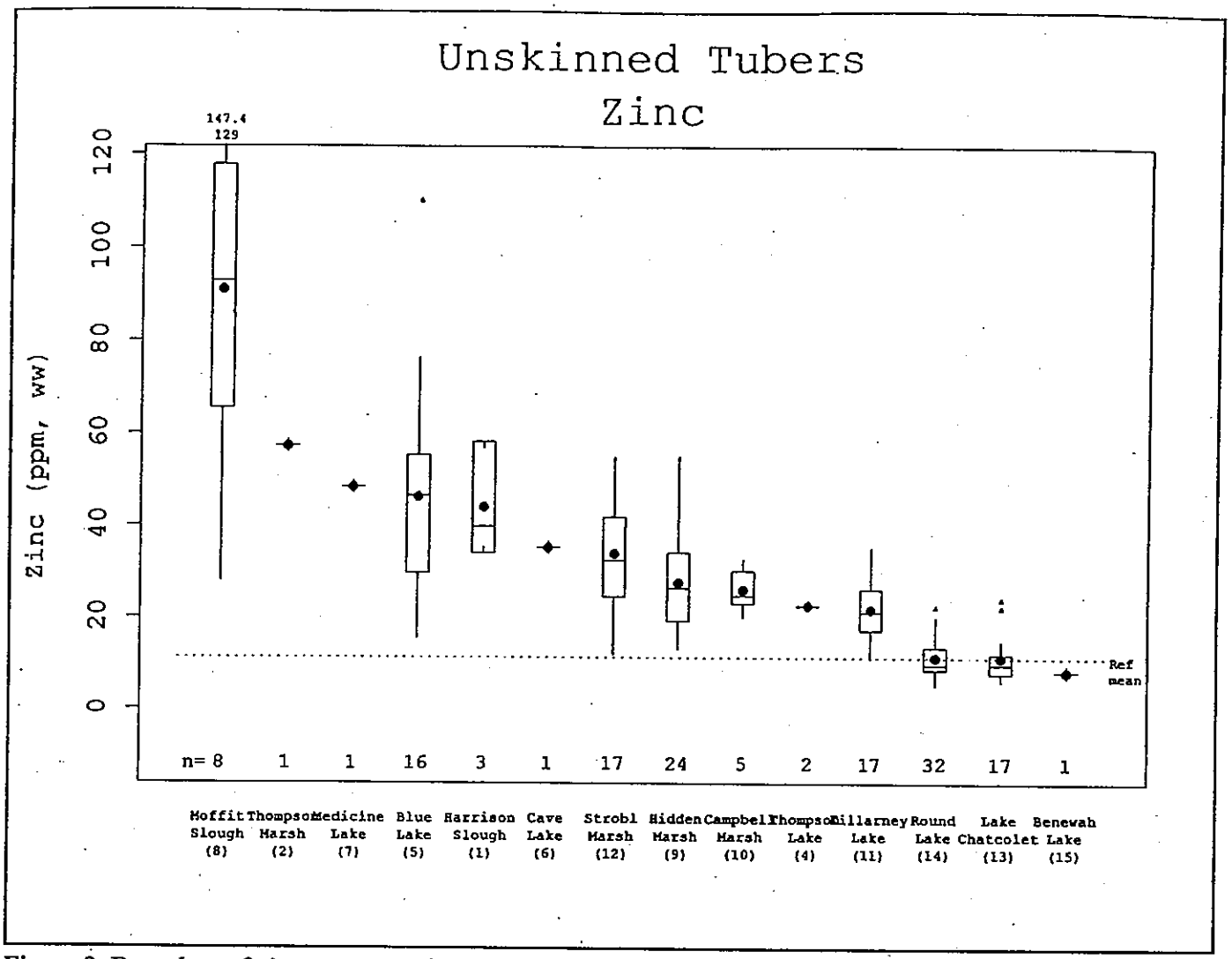


Figure 9: Box plots of **zinc** concentrations in **unskinned** tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

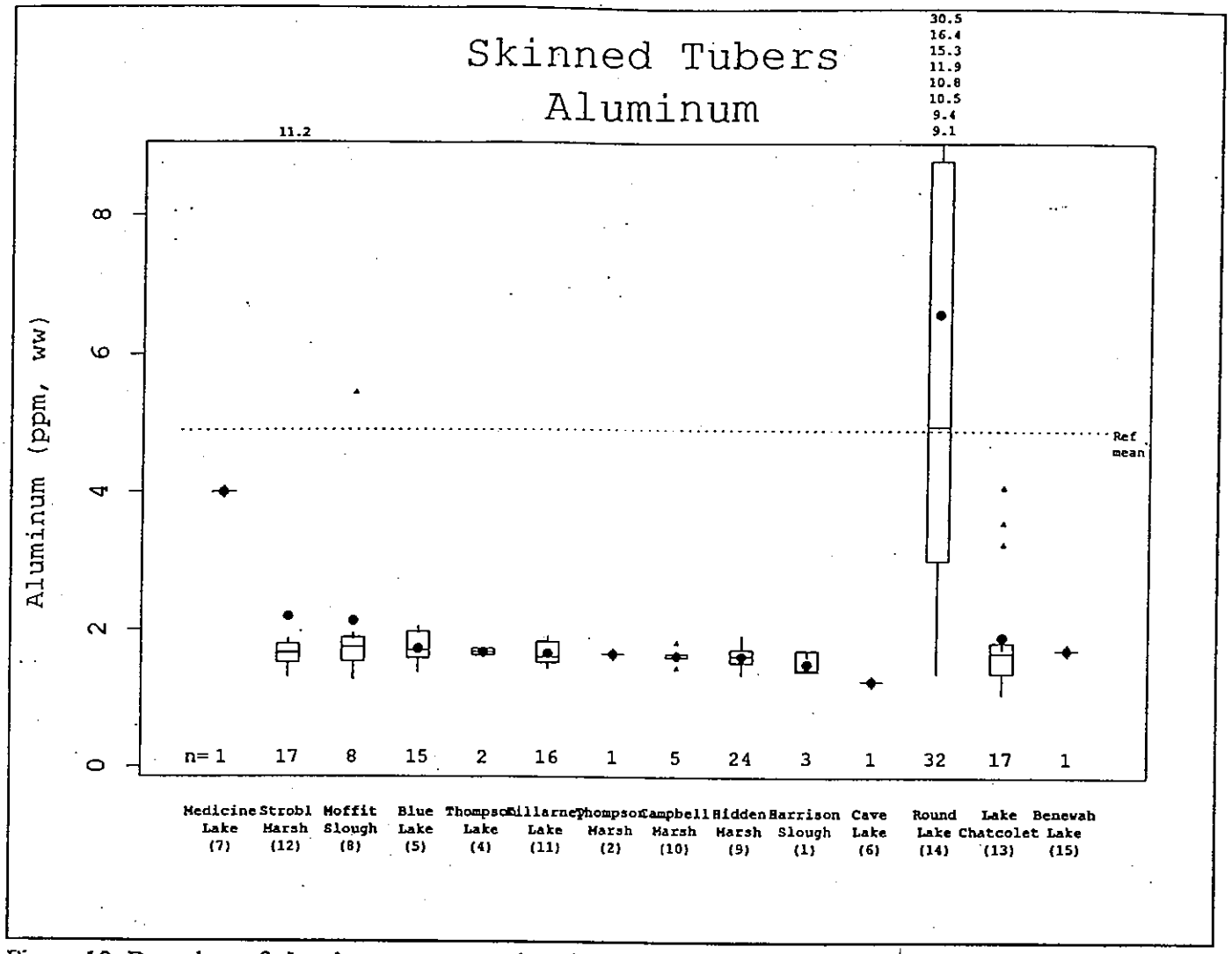


Figure 10: Box plots of **aluminum** concentrations in **skinned** tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

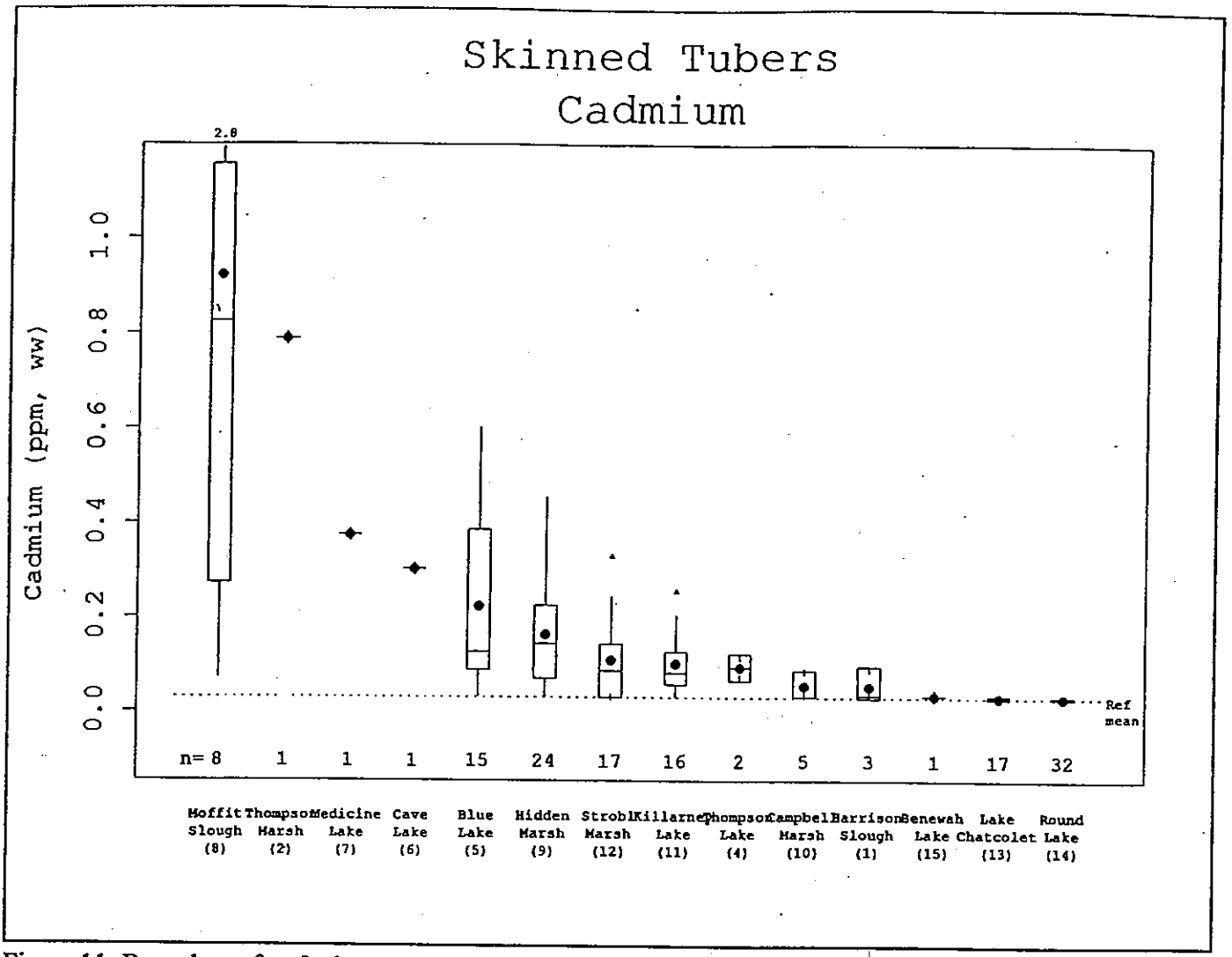


Figure 11: Box plots of **cadmium** concentrations in **skinned** tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

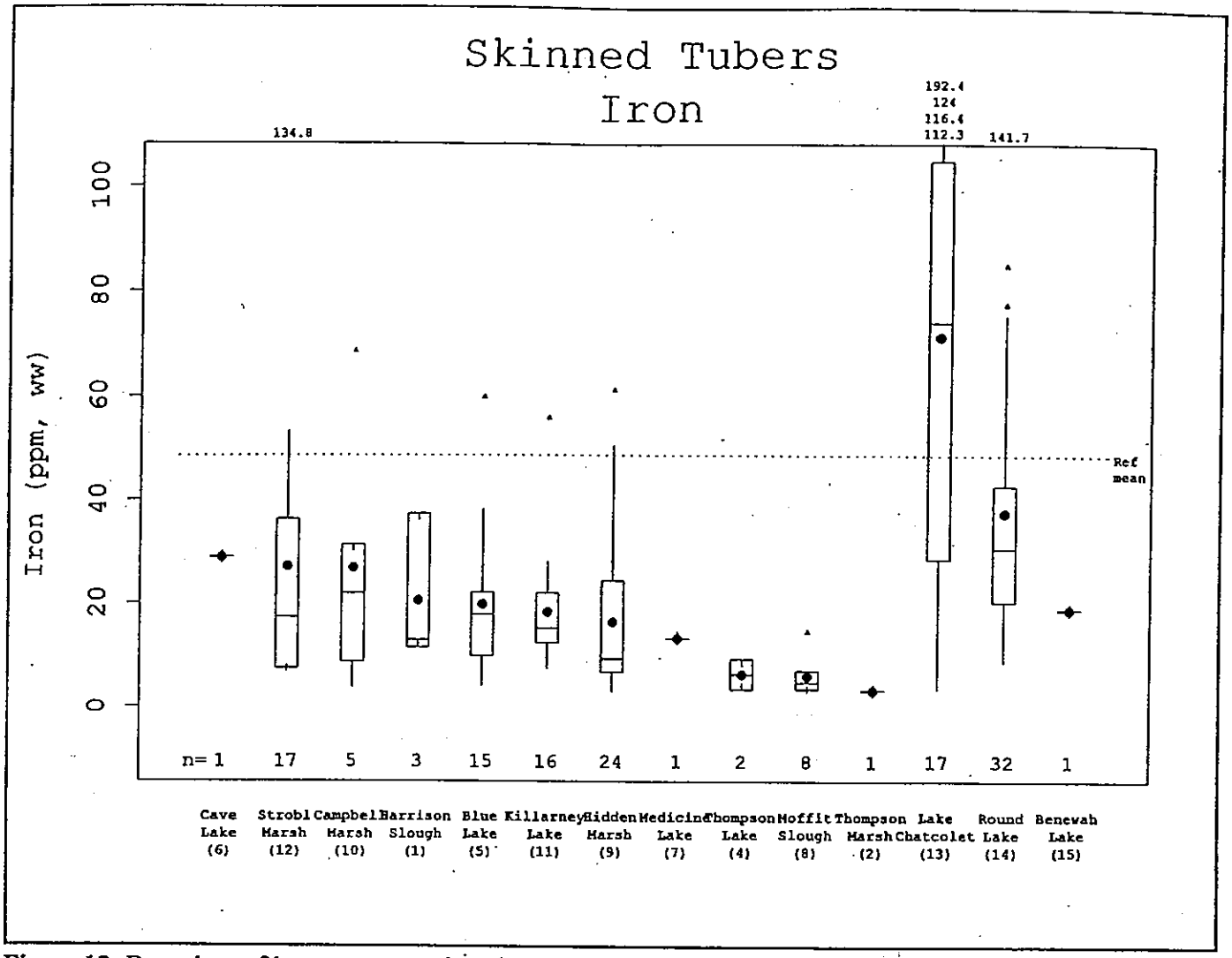


Figure 12: Box plots of **iron** concentrations in **skinned** tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

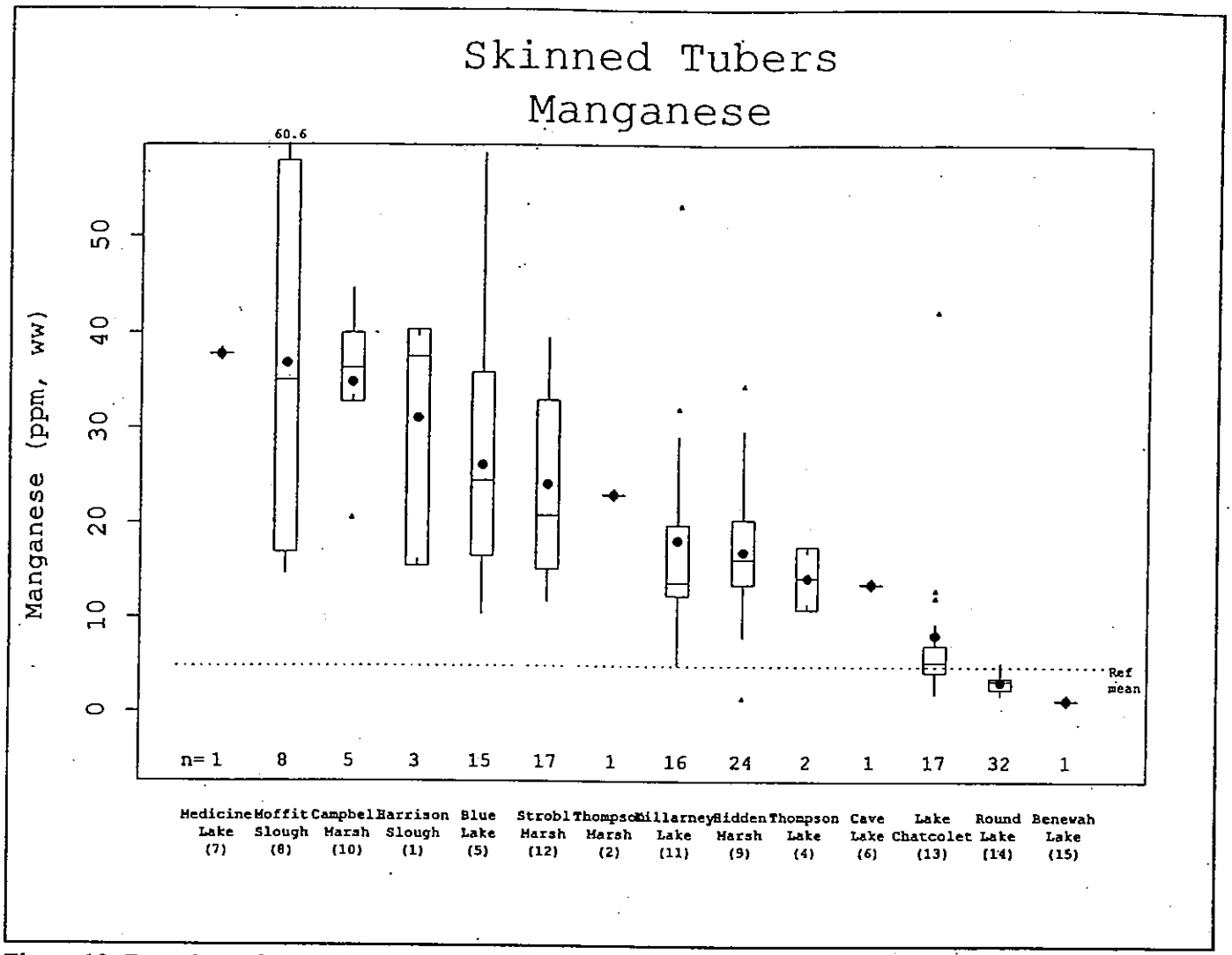


Figure 13: Box plots of **manganese** concentrations in **skinned** tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

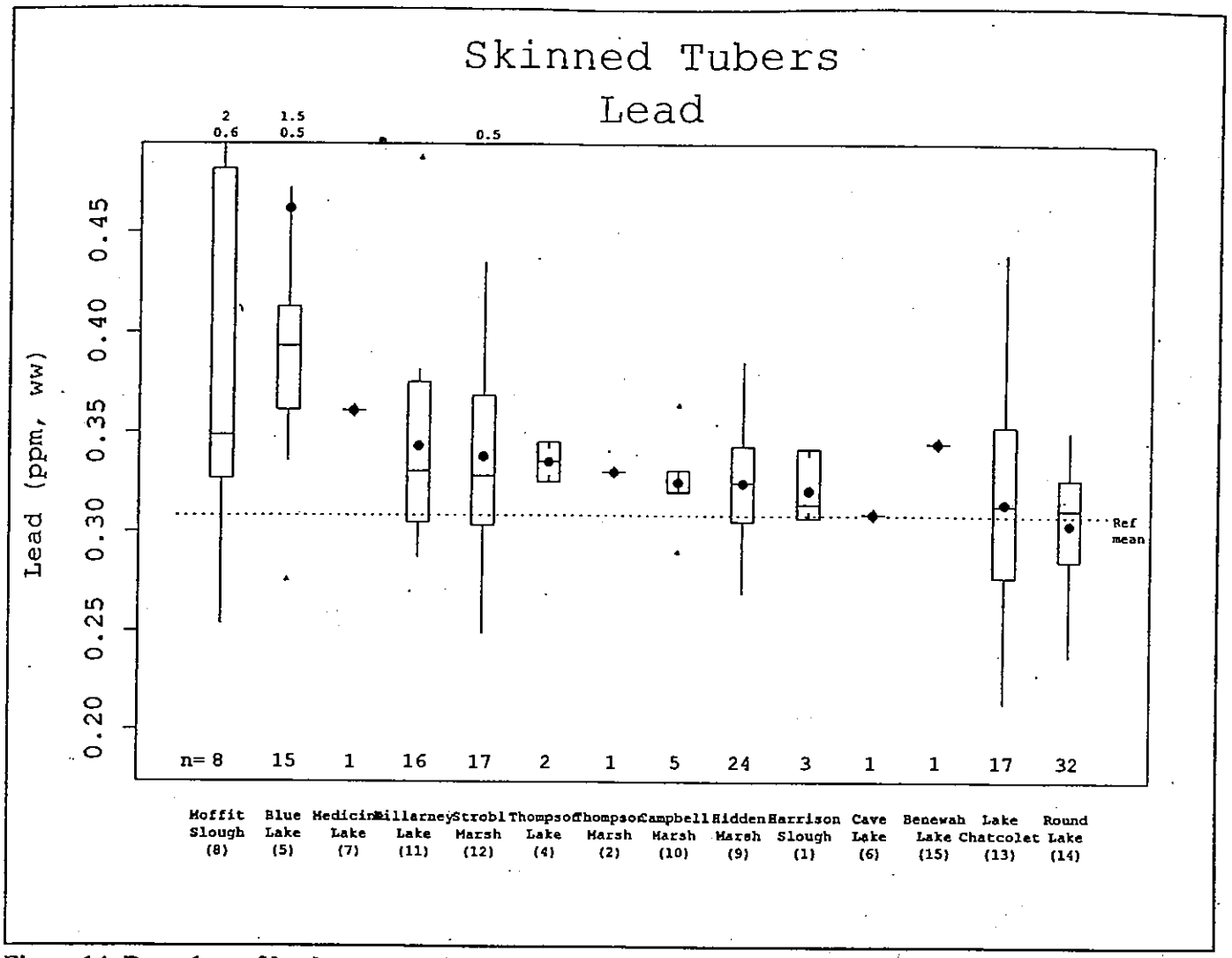


Figure 14: Box plots of lead concentrations in skinned tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

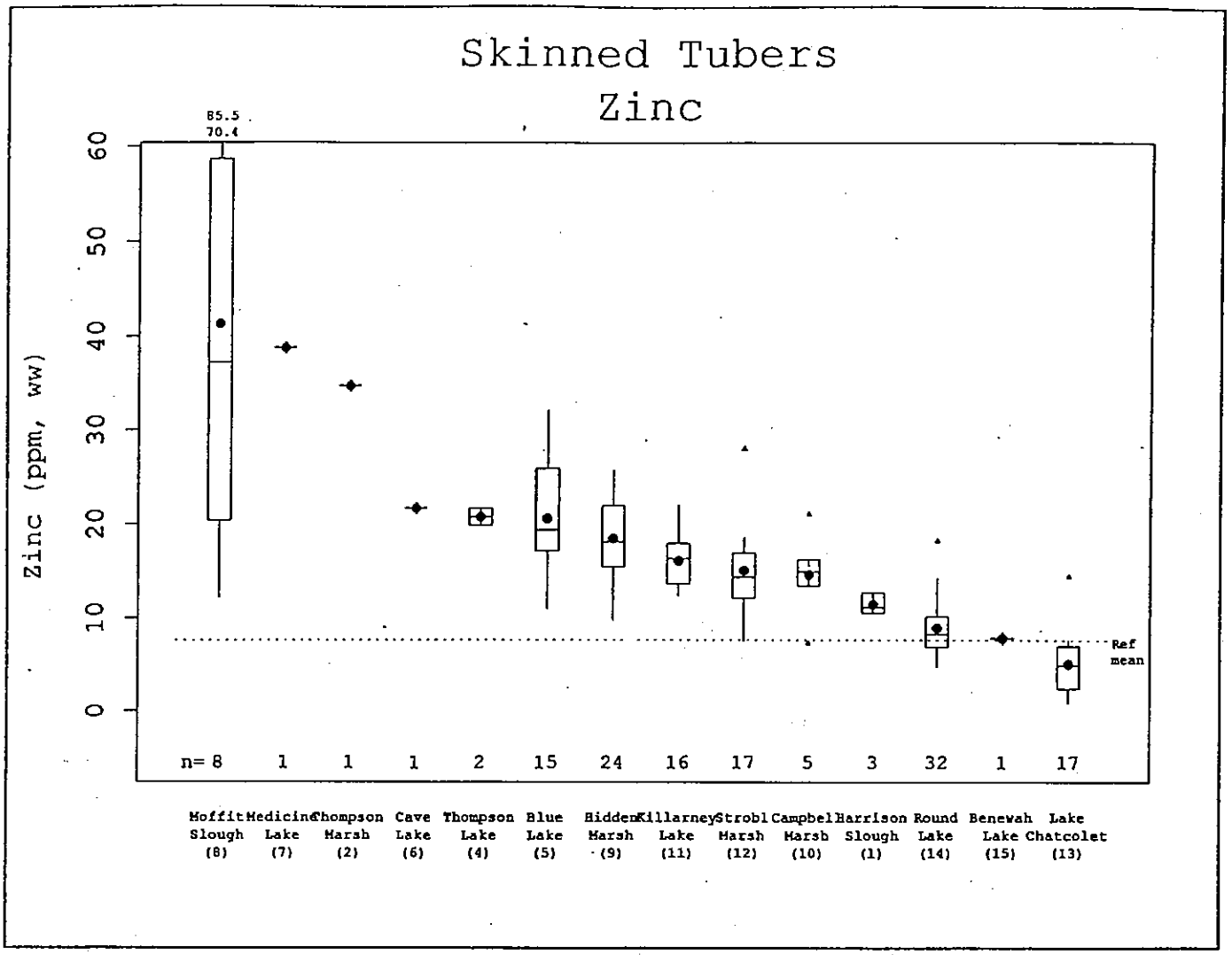


Figure 15: Box plots of **zinc** concentrations in **skinned** tubers for each wetland under study. Wetlands sorted by decreasing mean. Numbers at bottom inside box are sample sizes. Numbers below wetland name refer to location in Figure 2. Filled circles denote means. Box covers 25th and 75th percentiles. Line inside box denotes median. Whiskers (vertical lines) extend to observations at most 1.5 times length of box away from end of box, or to the top of the graph, which ever is smallest. Small triangles denote observations not covered by whiskers. Numbers above the graph are observed concentrations outside the range of the y-axis.

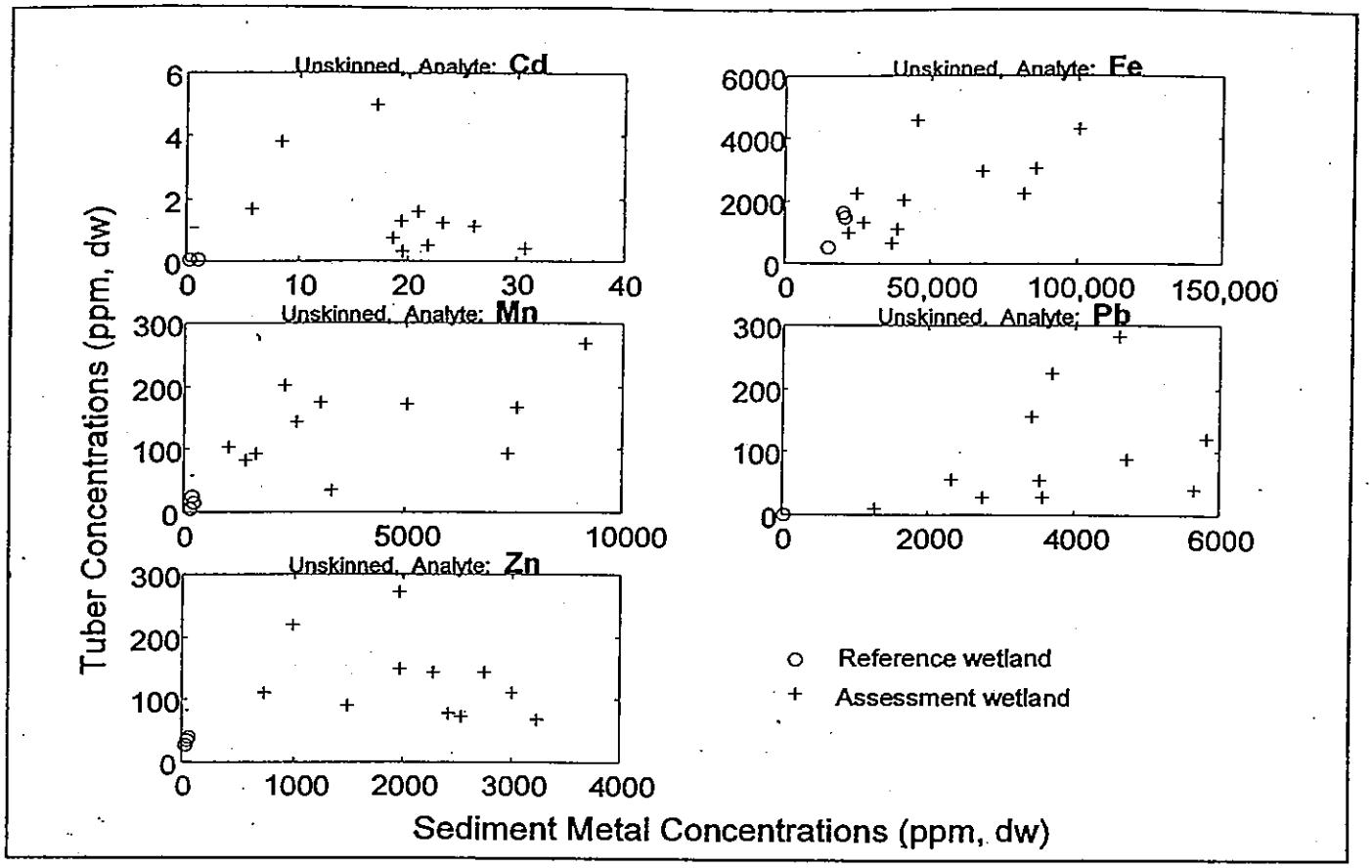


Figure 16. Metal concentrations in unskinned tubers versus metal concentrations in sediment.

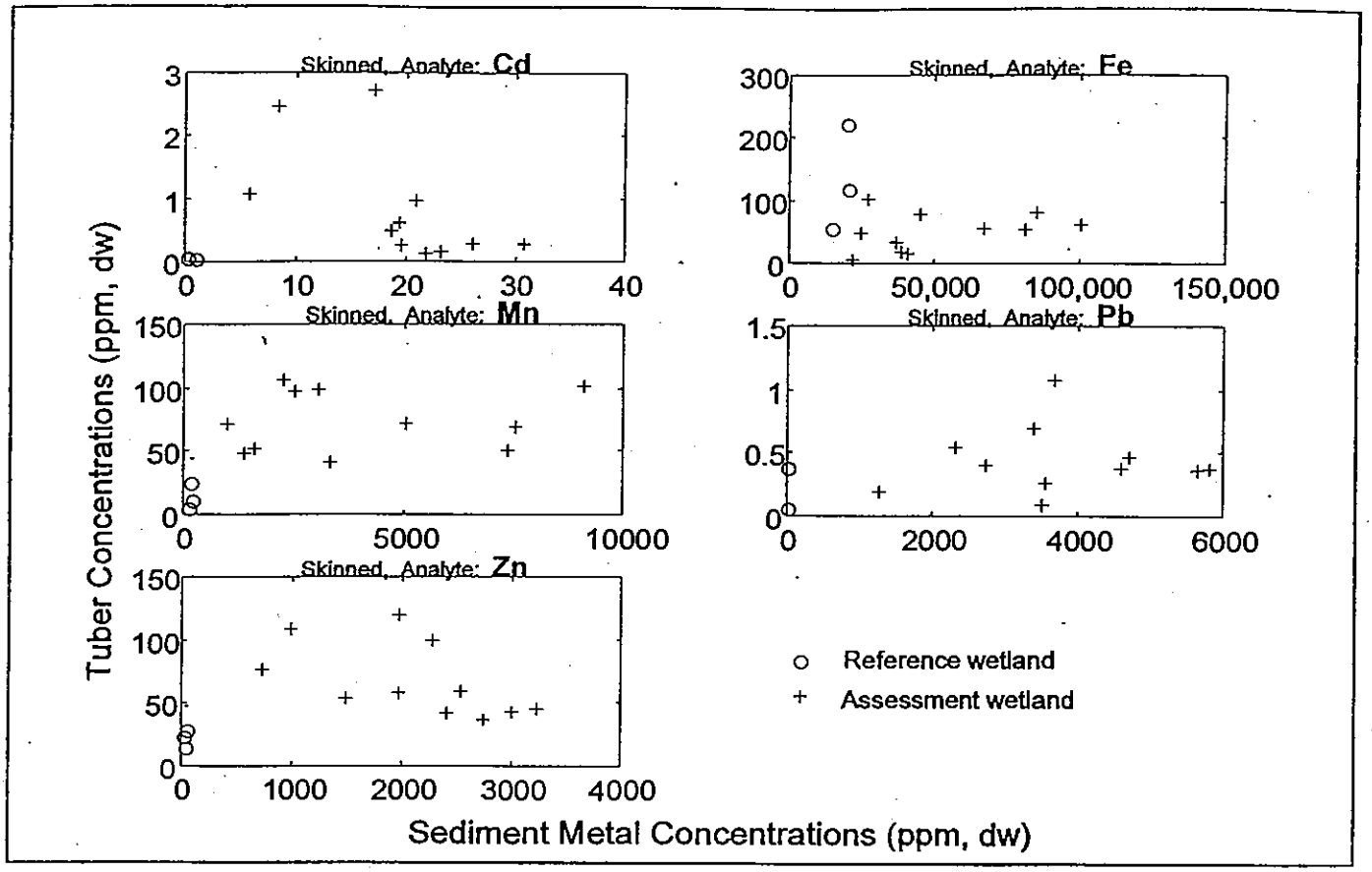


Figure 17. Metal concentrations in skinned tubers versus metal concentrations in sediment.

Table 1: Number of samples below detection and metal concentrations (ppm ww) in tuber samples collected in the assessment and reference basins.

UNSKINNED TUBERS						
Metal	Assessment (N= 95)			Reference (N= 50)		
	#<LOD[a] (%)	Mean	Median	#<LOD (%)	Mean	Median
Aluminum	1 (1%)	12.3	9.47	0 (0%)	21.89	18.07
Cadmium	7 (7%)	0.39	0.22	50 (100%)	0.03	0.03
Iron	0 (0%)	813.66	582.47	0 (0%)	451.11	428.77
Manganese	0 (0%)	42.92	33.84	0 (0%)	5.55	5.01
Lead	5 (5%)	30	20.3	50 (100%)	0.3	0.29
Zinc	0 (0%)	37.08	29.66	0 (0%)	11.28	9.72

SKINNED TUBERS						
Metal	Assessment (N= 93)			Reference (N= 50)		
	#<LOD (%)	Mean	Median	#<LOD (%)	Mean	Median
Aluminum	90 (97%)	1.81	1.64	21 (42%)	4.9	3.3
Cadmium	23 (25%)	0.22	0.11	50 (100%)	0.03	0.03
Iron	13 (14%)	18.75	12.84	1 (2%)	48.6	32.22
Manganese	0 (0%)	23.22	19.03	0 (0%)	4.84	3.43
Lead	91 (98%)	0.37	0.34	50 (100%)	0.31	0.31
Zinc	0 (0%)	19.76	17.08	7 (14%)	7.59	7.32

[a] LOD = Level of Detection

Table 2: One-sided significance levels for comparison of each assessment wetland with all reference samples based on Wilcoxon's two sample rank sum test (z test). All values below maximum detection limit were treated as tied. Small p-value implies distribution of concentrations in assessment wetland (or all) is larger than that in the reference area.

Unskinned Tubers						
Assessment Wetland	Metal					
	Aluminum	Cadmium	Iron	Manganese	Lead	Zinc
Blue Lake	>0.999	<0.001	<0.001	<0.001	<0.001	<0.001
Campbell Marsh	0.93	<0.001	<0.001	<0.001	<0.001	<0.001
Cave Lake	0.162	<0.001	0.594	0.048	<0.001	0.048
Hidden Marsh	0.901	<0.001	0.025	<0.001	<0.001	<0.001
Harrison Slough	0.998	<0.001	0.003	0.002	<0.001	0.002
Killarney Lake	0.997	<0.001	0.054	<0.001	<0.001	<0.001
Moffit Slough	0.958	<0.001	0.496	<0.001	<0.001	<0.001
Medicine Lake	0.918	<0.001	0.945	0.048	<0.001	0.048
Strobl Marsh	>0.999	<0.001	<0.001	<0.001	<0.001	<0.001
Thompson Lake	0.992	<0.001	0.784	0.022	<0.001	0.013
Thompson Marsh	0.62	<0.001	0.883	0.048	<0.001	0.048
All	>0.999	<0.001	<0.001	<0.001	<0.001	<0.001

Skinned Tubers						
Assessment Wetland	Metal					
	Aluminum	Cadmium	Iron	Manganese	Lead	Zinc
Blue Lake	0.998	<0.001	>0.999	<0.001	0.081	<0.001
Campbell Marsh	0.961	<0.001	0.928	<0.001	0.531	0.003
Cave Lake	0.798	<0.001	0.62	0.055	0.569	0.047
Hidden Marsh	>0.999	<0.001	>0.999	<0.001	0.514	<0.001
Harrison Slough	0.917	0.001	0.908	0.003	0.54	0.016
Killarney Lake	0.998	<0.001	>0.999	<0.001	0.517	<0.001
Moffit Slough	0.936	<0.001	>0.999	<0.001	0.028	<0.001
Medicine Lake	0.368	<0.001	0.907	0.055	0.569	0.047
Strobl Marsh	0.992	<0.001	0.997	<0.001	0.517	<0.001
Thompson Lake	0.874	<0.001	0.989	0.015	0.549	0.009
Thompson Marsh	0.798	<0.001	0.955	0.055	0.569	0.047
All	>0.999	<0.001	>0.999	<0.001	0.185	<0.001

Table 3: Two-sided significance levels comparing metal concentrations in **skinned and unskinned** tubers collected at the same location. Significance levels assigned using Wilcoxon's signed rank procedure. All values below maximum detection limit were assigned the maximum detection limit before differencing in the signed rank procedure. Wetlands containing a single skinned-unskinned pair were excluded.

Wetland	N Pairs	Metal					
		Aluminum	Cadmium	Iron	Manganese	Lead	Zinc
Blue Lake	15	0.001	0.017	<0.001	<0.001	<0.001	<0.001
Campbell Marsh	5	0.062	0.062	0.062	0.062	0.062	0.062
Hidden Marsh	24	<0.001	0.023	<0.001	<0.001	<0.001	<0:001
Harrison Slough	3	0.25	0.25	0.25	0.25	0.25	0.25
Killarney Lake	15	<0.001	0.116	<0.001	<0.001	0.001	<0.001
Lake Chatcolet[B]	17	<0.001	[A]	<0.001	0.045	[A]	<0.001
Moffit Slough	8	0.008	0.008	0.008	0.008	0.008	0.008
Round Lake[B]	32	<0.001	[A]	<0.001	<0.001	[A]	0.005
Strobl Marsh	17	0.006	0.001	<0.001	<0.001	<0.001	<0.001
Thompson Lake	2	0.5	1	0.5	0.5	0.5	0.5
All	142	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

[A] All values below maximum detection limit. By implication, p=1.
[B] Wetland in the reference area.

Table 4: Spearman's rank correlation coefficient and statistical significance between wetland average metals concentration in tubers and wetland average sediment concentrations for N=14 wetlands in the reference and assessment areas.

Analyte	Unskinned Tubers		Skinned Tubers	
	Correlation Coefficient	Statistical Significance	Correlation Coefficient	Statistical Significance
Cd	0.19	0.5126	0.14	0.63
Fe	0.71	0.0041	-0.06	0.84
Mn	0.71	0.0048	0.54	0.04
Pb	0.74	0.0070	0312	0.29
Zn	0.29	0.3100	0.20	0.50

Table 5: Regression coefficient and statistical significance using parametric statistical analysis for regression of wetland average metals concentration in tubers and wetland average metals concentration in sediment for N=14 wetlands in the reference and assessment areas.

Analyte	Unskinned Tubers		Skinned Tubers	
	Regression Coefficient	Statistical Significance	Regression Coefficient	Statistical Significance
Cd	0.012	0.78	-0.0025	0.92
Fe	0.031	0.01	-0.0003	0.57
Mn	0.018	0.01	0.0055	0.08
Pb	0.025	0.04	0.00003	0.35
Zn	0.017	0.32	0.0059	0.48

Appendix 1. Documents used in 1994 *Sagittaria spp.* Study

1019.4004	Water Potato Sampling Protocol for Coeur d'Alene Natural Resource Damage Assessment
1019.3701	Sample Documentation and Custody Procedures (SOP)
1019.3707	Decontamination of Equipment (SOP)
1019.3727	Sample Handling and Storage-Guidance (SOP)
1019.3728	Disposal of Waste Chemicals/Samples (SOP)
1019.3734	Map and Compass Use (SOP)
1019.3746	Federal Regulations for Shipping Biological Samples (SOP)
1019.3748	Lab Processing of *Sagittaria latifolia* Tubers (SOP)
1019.3850B	Coeur d'Alene Basin NRDA Wildlife Injury and Biological Pathways Studies Quality Assurance Plan

APPENDIX T

Masked Basin HHRA Lead Data

Categorical Statistical Summaries

This sheet contains the categorical summary information for the blood lead/environmental exposure data set on the following sheet.

Blood Lead Categorical Summary (ug/dl)

Category	Arith. Mean	Maximum	Minimum	St. Dev.	Geomean	GSD	Frequency
_LT_3_	1.6975	2.0000	1.0000	0.4613	1.6217	1.3768	119.0000
_LT_5_	3.4534	4.0000	3.0000	0.4994	3.4180	1.1545	161.0000
_LT_7_	5.4711	6.0000	5.0000	0.5012	5.4484	1.0957	121.0000
_LT_10	7.7692	9.0000	7.0000	0.7452	7.7347	1.0993	65.0000
_LT_15	11.6364	14.0000	10.0000	1.3652	11.5603	1.1228	33.0000
_GE_15	18.1200	29.0000	15.0000	3.1665	17.8898	1.1715	25.0000

Yard Soil Lead Categorical Summary (mg/kg)

Category	Arith. Mean	Maximum	Minimum	St. Dev.	Geomean	GSD	Frequency
NODATA							80.0000
_LT_75__	46.4725	68.1000	15.4000	18.2458	42.2110	1.6063	40.0000
_LT_100_	86.1324	96.5000	75.6000	7.1303	85.8394	1.0880	34.0000
_LT_150_	123.5971	149.8000	100.4000	17.6497	122.3845	1.1531	34.0000
_LT_200_	172.2909	199.0000	150.0000	17.9545	171.4056	1.1093	22.0000
_LT_250_	229.2789	248.0000	207.0000	15.5755	228.7696	1.0712	19.0000
_LT_350_	307.4129	342.8000	256.0000	25.8306	306.3144	1.0910	31.0000
_LT_500_	433.3852	494.0000	376.6000	43.8949	431.1862	1.1074	54.0000
_LT_650_	578.2025	647.0000	509.2000	45.2034	576.4375	1.0830	40.0000
_LT_900_	734.7222	895.9000	650.0000	59.7339	732.4116	1.0831	45.0000
_LT_1100	1008.6677	1086.7000	913.4000	55.7519	1007.1542	1.0575	31.0000
_LT_1500	1208.3103	1430.0000	1100.0000	93.2792	1204.9424	1.0784	29.0000
_LT_2200	1775.8886	2195.0000	1510.0000	229.2569	1762.0699	1.1339	35.0000
_GE2200_	3824.7767	7350.0000	2286.0000	1235.9273	3647.9224	1.3630	30.0000

Vacuum Dust Lead Categorical Summary (mg/kg)

Category	Arith. Mean	Maximum	Minimum	St. Dev.	Geomean	GSD	Frequency
NODATA							319.0000
_LT_150_	99.4407	147.0000	67.8000	28.8818	95.6723	1.3224	27.0000
_LT_350_	266.0588	341.0000	154.0000	59.7616	258.4401	1.2911	34.0000
_LT_550_	445.9912	546.0000	351.0000	60.3618	442.0373	1.1452	34.0000
_LT_750_	628.3241	707.0000	557.0000	45.3797	626.7569	1.0744	29.0000
_LT_1000	879.3167	958.0000	764.0000	65.3190	876.8937	1.0792	30.0000
_LT_1500	1182.0000	1380.0000	1030.0000	123.4825	1175.8519	1.1096	26.0000
_GE_1500	2745.1200	5800.0000	1500.0000	1145.4722	2542.0513	1.4823	25.0000

Dust Mat Lead Categorical Summary (mg/kg)

Category	Arith. Mean	Maximum	Minimum	St. Dev.	Geomean	GSD	Frequency
NODATA							355.0000
_LT_350_	200.3500	330.8000	54.7000	103.4109	171.6212	1.8378	18.0000
_LT_550_	436.7435	537.5000	374.0000	55.4059	433.4683	1.1329	23.0000
_LT_850_	729.4500	846.0000	551.5000	92.7562	723.4096	1.1426	30.0000
_LT_1000	915.8433	974.1000	851.1000	45.4481	914.7510	1.0510	30.0000
_LT_1500	1254.9259	1471.0000	1013.0000	166.0000	1244.2195	1.1434	27.0000
_LT_3250	2016.8462	3159.0000	1515.0000	513.0706	1959.0191	1.2736	26.0000
_GE_3250	7928.5333	27600.0000	3440.0000	7121.6855	6295.2170	1.8676	15.0000

Blood Lead/Environmental Exposure Data

This sheet provides categorical values for each of the 524 blood lead observation in the Basin HHRA data set. Categorical statistical summaries are found on the preceeding sheet.

Year	Area	Yard Soil	Vacuum Dust	Dust mat	Blood Lead	Age
YRBLPB	SUBAREA	SPBCAT	HDPBCAT	HMPBCAT	BLPBCAT	AGECAT
1996.0000	SIDE GUL	_LT_650_	_NODATA_	_LT_1000	_LT_3_	_5-6_
1997.0000	OSBURN	_LT_900_	_NODATA_	_LT_1500	_LT_3_	_7-9_
1996.0000	LOWER BA	_LT_75__	_NODATA_	_LT_350_	_LT_3_	_7-9_
1998.0000	LOWER BA	_LT_75__	_NODATA_	_LT_350_	_LT_3_	_7-9_
1998.0000	SILVERTO	_LT_250_	_LT_350_	_LT_550_	_LT_3_	_7-9_
1998.0000	SILVERTO	_LT_500_	_LT_550_	_LT_550_	_LT_3_	_7-9_
1996.0000	LOWER BA	_LT_75__	_NODATA_	_LT_550_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_250_	_LT_1000	_LT_550_	_LT_3_	_5-6_
1999.0000	OSBURN	_LT_250_	_LT_1000	_LT_550_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_250_	_LT_1000	_LT_550_	_LT_3_	_7-9_
1999.0000	SIDE GUL	_LT_200_	_NODATA_	_LT_850_	_LT_3_	_3-4_
1999.0000	SIDE GUL	_LT_200_	_NODATA_	_LT_850_	_LT_3_	_7-9_
1998.0000	OSBURN	_GE2200_	_NODATA_	_LT_850_	_LT_3_	_7-9_
1999.0000	KINGSTON	_NODATA_	_GE_1500	_NODATA_	_LT_3_	_5-6_
1999.0000	SILVERTO	_LT_250_	_LT_1500	_NODATA_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_200_	_LT_150_	_NODATA_	_LT_3_	_7-9_
1998.0000	LOWER BA	_LT_75__	_LT_150_	_NODATA_	_LT_3_	_7-9_
1999.0000	LOWER BA	_LT_75__	_LT_150_	_NODATA_	_LT_3_	_7-9_
1998.0000	KINGSTON	_LT_150_	_LT_350_	_NODATA_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_650_	_LT_350_	_NODATA_	_LT_3_	_3-4_
1999.0000	OSBURN	_LT_650_	_LT_350_	_NODATA_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_350_	_LT_550_	_NODATA_	_LT_3_	_7-9_
1999.0000	NINE MIL	_LT_900_	_LT_550_	_NODATA_	_LT_3_	_7-9_
1996.0000	NINE MIL	_LT_1100	_LT_750_	_NODATA_	_LT_3_	_7-9_
1996.0000	LOWER BA	_GE2200_	_NODATA_	_NODATA_	_LT_3_	_3-4_
1999.0000	KINGSTON	_LT_100_	_NODATA_	_NODATA_	_LT_3_	_3-4_
1996.0000	OSBURN	_LT_1500	_NODATA_	_NODATA_	_LT_3_	_7-9_
1998.0000	WALLACE	_LT_2200	_NODATA_	_NODATA_	_LT_3_	_7-9_
1998.0000	WALLACE	_LT_2200	_NODATA_	_NODATA_	_LT_3_	_7-9_
1999.0000	SIDE GUL	_LT_75__	_NODATA_	_NODATA_	_LT_3_	_7-9_
1998.0000	KINGSTON	_LT_900_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1999.0000	KINGSTON	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_3-4_
1999.0000	KINGSTON	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1999.0000	KINGSTON	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1999.0000	SILVERTO	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_200_	_LT_1000	_GE_3250	_LT_3_	_7-9_
1998.0000	SILVERTO	_LT_1100	_LT_1000	_LT_1000	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_1100	_LT_350_	_LT_1000	_LT_3_	_7-9_
1999.0000	SIDE GUL	_LT_650_	_NODATA_	_LT_1000	_LT_3_	_7-9_
1998.0000	NINE MIL	_LT_1500	_NODATA_	_LT_1500	_LT_3_	_5-6_
1997.0000	NINE MIL	_LT_1500	_NODATA_	_LT_1500	_LT_3_	_7-9_
1998.0000	NINE MIL	_LT_1500	_NODATA_	_LT_1500	_LT_3_	_7-9_
1999.0000	SILVERTO	_LT_900_	_LT_1000	_LT_1500	_LT_3_	_5-6_
1996.0000	SILVERTO	_LT_900_	_LT_1000	_LT_1500	_LT_3_	_3-4_
1999.0000	SILVERTO	_LT_900_	_LT_1000	_LT_1500	_LT_3_	_7-9_
1999.0000	SILVERTO	_LT_900_	_LT_1000	_LT_1500	_LT_3_	_3-4_

Year	Area	Yard Soil	Vacuum Dust	Dust mat	Blood Lead	Age
YRBLPB	SUBAREA	SPBCAT	HDPBCAT	HMPBCAT	BLPBCAT	AGECAT
1996.0000	OSBURN	_LT_900_	_NODATA_	_LT_1500	_LT_3_	_7-9_
1998.0000	OSBURN	_LT_900_	_NODATA_	_LT_1500	_LT_3_	_7-9_
1997.0000	SILVERTO	_LT_500_	_LT_1000	_LT_1500	_LT_3_	_5-6_
1996.0000	LOWER BA	_LT_75__	_NODATA_	_LT_350_	_LT_3_	_3-4_
1999.0000	LOWER BA	_LT_150_	_NODATA_	_LT_350_	_LT_3_	_7-9_
1996.0000	KINGSTON	_LT_75__	_NODATA_	_LT_350_	_LT_3_	_3-4_
1996.0000	LOWER BA	_LT_100_	_NODATA_	_LT_350_	_LT_3_	_3-4_
1996.0000	LOWER BA	_LT_75__	_NODATA_	_LT_350_	_LT_3_	_7-9_
1996.0000	SIDE GUL	_LT_100_	_NODATA_	_LT_350_	_LT_3_	_1-2_
1996.0000	LOWER BA	_LT_75__	_NODATA_	_LT_350_	_LT_3_	_7-9_
1997.0000	SILVERTO	_LT_500_	_LT_550_	_LT_550_	_LT_3_	_7-9_
1999.0000	SILVERTO	_LT_500_	_LT_550_	_LT_550_	_LT_3_	_7-9_
1996.0000	SIDE GUL	_LT_250_	_NODATA_	_LT_850_	_LT_3_	_7-9_
1999.0000	WALLACE	_LT_900_	_LT_1000	_LT_850_	_LT_3_	_7-9_
1998.0000	OSBURN	_GE2200_	_NODATA_	_LT_850_	_LT_3_	_7-9_
1999.0000	MULLAN	_LT_900_	_GE_1500	_NODATA_	_LT_3_	_7-9_
1998.0000	NINE MIL	_LT_1500	_GE_1500	_NODATA_	_LT_3_	_7-9_
1999.0000	SILVERTO	_LT_150_	_GE_1500	_NODATA_	_LT_3_	_7-9_
1999.0000	MULLAN	_LT_500_	_GE_1500	_NODATA_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_500_	_LT_1500	_NODATA_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_250_	_LT_150_	_NODATA_	_LT_3_	_1-2_
1999.0000	OSBURN	_LT_250_	_LT_150_	_NODATA_	_LT_3_	_3-4_
1999.0000	NINE MIL	_LT_1500	_LT_150_	_NODATA_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_200_	_LT_150_	_NODATA_	_LT_3_	_3-4_
1998.0000	LOWER BA	_LT_75__	_LT_150_	_NODATA_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_650_	_LT_350_	_NODATA_	_LT_3_	_5-6_
1999.0000	KINGSTON	_LT_150_	_LT_350_	_NODATA_	_LT_3_	_5-6_
1999.0000	KINGSTON	_LT_150_	_LT_350_	_NODATA_	_LT_3_	_7-9_
1998.0000	OSBURN	_LT_650_	_LT_350_	_NODATA_	_LT_3_	_3-4_
1999.0000	OSBURN	_LT_650_	_LT_350_	_NODATA_	_LT_3_	_7-9_
1998.0000	OSBURN	_LT_650_	_LT_350_	_NODATA_	_LT_3_	_7-9_
1999.0000	KINGSTON	_LT_350_	_LT_350_	_NODATA_	_LT_3_	_7-9_
1998.0000	LOWER BA	_LT_75__	_LT_350_	_NODATA_	_LT_3_	_7-9_
1998.0000	OSBURN	_LT_350_	_LT_550_	_NODATA_	_LT_3_	_7-9_
1999.0000	SIDE GUL	_LT_75__	_LT_550_	_NODATA_	_LT_3_	_5-6_
1999.0000	KINGSTON	_LT_100_	_LT_550_	_NODATA_	_LT_3_	_7-9_
1999.0000	KINGSTON	_LT_100_	_LT_550_	_NODATA_	_LT_3_	_7-9_
1998.0000	SILVERTO	_LT_150_	_LT_550_	_NODATA_	_LT_3_	_3-4_
1999.0000	SILVERTO	_LT_150_	_LT_550_	_NODATA_	_LT_3_	_5-6_
1996.0000	NINE MIL	_LT_1100	_LT_750_	_NODATA_	_LT_3_	_1-2_
1997.0000	NINE MIL	_LT_1100	_LT_750_	_NODATA_	_LT_3_	_7-9_
1998.0000	NINE MIL	_LT_1100	_LT_750_	_NODATA_	_LT_3_	_7-9_
1996.0000	MULLAN	_GE2200_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_100_	_NODATA_	_NODATA_	_LT_3_	_5-6_
1999.0000	KINGSTON	_LT_100_	_NODATA_	_NODATA_	_LT_3_	_1-2_
1999.0000	NINE MIL	_LT_100_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1996.0000	OSBURN	_LT_1100	_NODATA_	_NODATA_	_LT_3_	_7-9_
1996.0000	SILVERTO	_LT_200_	_NODATA_	_NODATA_	_LT_3_	_3-4_
1999.0000	SILVERTO	_LT_200_	_NODATA_	_NODATA_	_LT_3_	_5-6_
1996.0000	WALLACE	_LT_2200	_NODATA_	_NODATA_	_LT_3_	_7-9_

Year	Area	Yard Soil	Vacuum Dust	Dust mat	Blood Lead	Age
YRBLPB	SUBAREA	SPBCAT	HDPBCAT	HMPBCAT	BLPBCAT	AGECAT
1998.0000	WALLACE	_LT_2200	_NODATA_	_NODATA_	_LT_3_	_5-6_
1999.0000	WALLACE	_LT_2200	_NODATA_	_NODATA_	_LT_3_	_7-9_
1996.0000	OSBURN	_LT_500_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1999.0000	OSBURN	_LT_500_	_NODATA_	_NODATA_	_LT_3_	_3-4_
1999.0000	OSBURN	_LT_500_	_NODATA_	_NODATA_	_LT_3_	_3-4_
1999.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_3_	_5-6_
1996.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_3_	_3-4_
1996.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1997.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1996.0000	NINE MIL	_LT_900_	_NODATA_	_NODATA_	_LT_3_	_1-2_
1996.0000	MULLAN	_LT_900_	_NODATA_	_NODATA_	_LT_3_	_5-6_
1996.0000	MULLAN	_LT_900_	_NODATA_	_NODATA_	_LT_3_	_5-6_
1999.0000	KINGSTON	_LT_900_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1996.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_1-2_
1999.0000	SILVERTO	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1997.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1998.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1998.0000	KINGSTON	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_3-4_
1999.0000	KINGSTON	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_1-2_
1999.0000	SIDE GUL	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1999.0000	SIDE GUL	_NODATA_	_NODATA_	_NODATA_	_LT_3_	_7-9_
1998.0000	SILVERTO	_LT_500_	_NODATA_	_LT_1000	_LT_5_	_7-9_
1996.0000	SILVERTO	_LT_500_	_NODATA_	_LT_1000	_LT_5_	_7-9_
1996.0000	MULLAN	_LT_2200	_NODATA_	_LT_1000	_LT_5_	_5-6_
1996.0000	MULLAN	_LT_2200	_NODATA_	_LT_1000	_LT_5_	_7-9_
1999.0000	SIDE GUL	_LT_650_	_NODATA_	_LT_1000	_LT_5_	_5-6_
1997.0000	NINE MIL	_LT_1500	_NODATA_	_LT_1500	_LT_5_	_3-4_
1998.0000	OSBURN	_LT_350_	_LT_1500	_LT_1500	_LT_5_	_7-9_
1996.0000	WALLACE	_LT_2200	_NODATA_	_LT_1500	_LT_5_	_7-9_
1998.0000	SILVERTO	_LT_900_	_NODATA_	_LT_1500	_LT_5_	_7-9_
1998.0000	SILVERTO	_LT_900_	_NODATA_	_LT_1500	_LT_5_	_7-9_
1996.0000	SILVERTO	_LT_500_	_LT_1000	_LT_1500	_LT_5_	_5-6_
1996.0000	MULLAN	_LT_1100	_NODATA_	_LT_3250	_LT_5_	_5-6_
1996.0000	WALLACE	_LT_2200	_NODATA_	_LT_3250	_LT_5_	_7-9_
1996.0000	LOWER BA	_LT_150_	_NODATA_	_LT_350_	_LT_5_	_5-6_
1998.0000	LOWER BA	_LT_150_	_NODATA_	_LT_350_	_LT_5_	_1-2_
1996.0000	KINGSTON	_LT_500_	_NODATA_	_LT_350_	_LT_5_	_3-4_
1999.0000	SIDE GUL	_LT_150_	_NODATA_	_LT_350_	_LT_5_	_5-6_
1998.0000	KINGSTON	_LT_900_	_LT_350_	_LT_550_	_LT_5_	_7-9_
1999.0000	KINGSTON	_LT_900_	_LT_350_	_LT_550_	_LT_5_	_7-9_
1996.0000	SILVERTO	_LT_150_	_NODATA_	_LT_550_	_LT_5_	_5-6_
1996.0000	SIDE GUL	_LT_250_	_NODATA_	_LT_550_	_LT_5_	_7-9_
1999.0000	LOWER BA	_LT_150_	_NODATA_	_LT_550_	_LT_5_	_5-6_
1996.0000	SIDE GUL	_LT_900_	_NODATA_	_LT_550_	_LT_5_	_7-9_
1996.0000	WALLACE	_LT_1100	_NODATA_	_LT_850_	_LT_5_	_7-9_
1996.0000	OSBURN	_LT_350_	_NODATA_	_LT_850_	_LT_5_	_7-9_
1998.0000	OSBURN	_GE2200_	_NODATA_	_LT_850_	_LT_5_	_7-9_
1998.0000	NINE MIL	_LT_1500	_GE_1500	_NODATA_	_LT_5_	_7-9_
1999.0000	NINE MIL	_LT_75__	_GE_1500	_NODATA_	_LT_5_	_7-9_

Year	Area	Yard Soil	Vacuum Dust	Dust mat	Blood Lead	Age
YRBLPB	SUBAREA	SPBCAT	HDPBCAT	HMPBCAT	BLPBCAT	AGECAT
1999.0000	MULLAN	_LT_500_	_GE_1500	_NODATA_	_LT_5_	_5-6_
1999.0000	NINE MIL	_GE2200_	_LT_1000	_NODATA_	_LT_5_	_5-6_
1999.0000	MULLAN	_LT_250_	_LT_1000	_NODATA_	_LT_5_	_7-9_
1999.0000	OSBURN	_LT_350_	_LT_1500	_NODATA_	_LT_5_	_7-9_
1999.0000	WALLACE	_LT_1500	_LT_1500	_NODATA_	_LT_5_	_7-9_
1998.0000	WALLACE	_LT_2200	_LT_1500	_NODATA_	_LT_5_	_7-9_
1999.0000	OSBURN	_LT_500_	_LT_150_	_NODATA_	_LT_5_	_7-9_
1999.0000	SIDE GUL	_LT_150_	_LT_350_	_NODATA_	_LT_5_	_5-6_
1999.0000	SIDE GUL	_LT_150_	_LT_350_	_NODATA_	_LT_5_	_5-6_
1998.0000	KINGSTON	_LT_150_	_LT_350_	_NODATA_	_LT_5_	_3-4_
1999.0000	KINGSTON	_LT_75__	_LT_350_	_NODATA_	_LT_5_	_7-9_
1998.0000	OSBURN	_LT_650_	_LT_350_	_NODATA_	_LT_5_	_7-9_
1999.0000	SILVERTO	_LT_650_	_LT_350_	_NODATA_	_LT_5_	_1-2_
1999.0000	OSBURN	_LT_1100	_LT_550_	_NODATA_	_LT_5_	_7-9_
1999.0000	OSBURN	_LT_500_	_LT_550_	_NODATA_	_LT_5_	_5-6_
1999.0000	KINGSTON	_LT_100_	_LT_550_	_NODATA_	_LT_5_	_7-9_
1999.0000	KINGSTON	_LT_100_	_LT_550_	_NODATA_	_LT_5_	_7-9_
1998.0000	SILVERTO	_LT_150_	_LT_550_	_NODATA_	_LT_5_	_1-2_
1999.0000	SILVERTO	_LT_150_	_LT_550_	_NODATA_	_LT_5_	_1-2_
1997.0000	NINE MIL	_LT_1100	_LT_750_	_NODATA_	_LT_5_	_3-4_
1998.0000	SILVERTO	_LT_2200	_LT_750_	_NODATA_	_LT_5_	_7-9_
1999.0000	SIDE GUL	_LT_75__	_LT_750_	_NODATA_	_LT_5_	_7-9_
1998.0000	SIDE GUL	_LT_100_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1996.0000	SIDE GUL	_LT_100_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1998.0000	SIDE GUL	_LT_100_	_NODATA_	_NODATA_	_LT_5_	_5-6_
1999.0000	SIDE GUL	_LT_100_	_NODATA_	_NODATA_	_LT_5_	_5-6_
1998.0000	SILVERTO	_LT_100_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1999.0000	OSBURN	_LT_100_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1999.0000	OSBURN	_LT_100_	_NODATA_	_NODATA_	_LT_5_	_5-6_
1996.0000	OSBURN	_LT_1100	_NODATA_	_NODATA_	_LT_5_	_5-6_
1996.0000	OSBURN	_LT_1500	_NODATA_	_NODATA_	_LT_5_	_3-4_
1997.0000	OSBURN	_LT_1500	_NODATA_	_NODATA_	_LT_5_	_5-6_
1998.0000	SIDE GUL	_LT_1500	_NODATA_	_NODATA_	_LT_5_	_7-9_
1998.0000	SILVERTO	_LT_200_	_NODATA_	_NODATA_	_LT_5_	_5-6_
1997.0000	OSBURN	_LT_250_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1996.0000	OSBURN	_LT_500_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1999.0000	OSBURN	_LT_500_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1996.0000	OSBURN	_LT_500_	_NODATA_	_NODATA_	_LT_5_	_5-6_
1999.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1999.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_5_	_1-2_
1998.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1996.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1996.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1997.0000	LOWER BA	_LT_75__	_NODATA_	_NODATA_	_LT_5_	_7-9_
1999.0000	LOWER BA	_LT_75__	_NODATA_	_NODATA_	_LT_5_	_5-6_
1998.0000	KINGSTON	_LT_900_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1999.0000	KINGSTON	_LT_900_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1998.0000	KINGSTON	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1998.0000	SIDE GUL	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1998.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_5-6_

Year	Area	Yard Soil	Vacuum Dust	Dust mat	Blood Lead	Age
YRBLPB	SUBAREA	SPBCAT	HDPBCAT	HMPBCAT	BLPBCAT	AGECAT
1999.0000	OSBURN	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1998.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_5-6_
1999.0000	SILVERTO	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_5-6_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_5-6_
1999.0000	MULLAN	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1999.0000	MULLAN	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1999.0000	OSBURN	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1999.0000	MULLAN	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1998.0000	SILVERTO	_LT_1100	_LT_1000	_LT_1000	_LT_5_	_3-4_
1998.0000	SILVERTO	_LT_1100	_LT_1000	_LT_1000	_LT_5_	_1-2_
1997.0000	OSBURN	_LT_2200	_NODATA_	_LT_1000	_LT_5_	_5-6_
1997.0000	OSBURN	_LT_2200	_NODATA_	_LT_1000	_LT_5_	_7-9_
1998.0000	OSBURN	_LT_2200	_NODATA_	_LT_1000	_LT_5_	_7-9_
1998.0000	SIDE GUL	_LT_650_	_NODATA_	_LT_1000	_LT_5_	_7-9_
1996.0000	SILVERTO	_LT_900_	_LT_1000	_LT_1500	_LT_5_	_1-2_
1997.0000	SILVERTO	_LT_500_	_LT_1000	_LT_1500	_LT_5_	_3-4_
1996.0000	MULLAN	_LT_1100	_NODATA_	_LT_3250	_LT_5_	_7-9_
1996.0000	NINE MIL	_LT_900_	_LT_1500	_LT_3250	_LT_5_	_5-6_
1999.0000	SIDE GUL	_LT_650_	_NODATA_	_LT_3250	_LT_5_	_7-9_
1999.0000	NINE MIL	_LT_2200	_NODATA_	_LT_3250	_LT_5_	_7-9_
1999.0000	NINE MIL	_LT_2200	_NODATA_	_LT_3250	_LT_5_	_7-9_
1999.0000	MULLAN	_GE2200_	_GE_1500	_LT_3250	_LT_5_	_7-9_
1999.0000	LOWER BA	_LT_75__	_NODATA_	_LT_350_	_LT_5_	_5-6_
1999.0000	SIDE GUL	_LT_150_	_NODATA_	_LT_350_	_LT_5_	_3-4_
1998.0000	SIDE GUL	_LT_150_	_NODATA_	_LT_350_	_LT_5_	_3-4_
1999.0000	LOWER BA	_LT_150_	_LT_550_	_LT_550_	_LT_5_	_7-9_
1996.0000	KINGSTON	_LT_100_	_NODATA_	_LT_550_	_LT_5_	_3-4_
1999.0000	KINGSTON	_LT_100_	_NODATA_	_LT_550_	_LT_5_	_7-9_
1999.0000	KINGSTON	_LT_100_	_NODATA_	_LT_550_	_LT_5_	_3-4_
1999.0000	OSBURN	_LT_350_	_NODATA_	_LT_850_	_LT_5_	_7-9_
1999.0000	SIDE GUL	_LT_350_	_LT_550_	_LT_850_	_LT_5_	_7-9_
1999.0000	WALLACE	_LT_900_	_LT_1000	_LT_850_	_LT_5_	_5-6_
1998.0000	OSBURN	_GE2200_	_NODATA_	_LT_850_	_LT_5_	_7-9_
1999.0000	OSBURN	_LT_650_	_LT_750_	_LT_850_	_LT_5_	_3-4_
1999.0000	OSBURN	_LT_650_	_LT_750_	_LT_850_	_LT_5_	_3-4_
1999.0000	NINE MIL	_LT_1500	_GE_1500	_NODATA_	_LT_5_	_7-9_
1999.0000	WALLACE	_LT_1500	_GE_1500	_NODATA_	_LT_5_	_7-9_
1999.0000	MULLAN	_LT_500_	_GE_1500	_NODATA_	_LT_5_	_7-9_
1998.0000	NINE MIL	_LT_100_	_GE_1500	_NODATA_	_LT_5_	_1-2_
1999.0000	SILVERTO	_LT_1500	_LT_1000	_NODATA_	_LT_5_	_7-9_
1999.0000	LOWER BA	_GE2200_	_LT_1500	_NODATA_	_LT_5_	_1-2_
1999.0000	OSBURN	_LT_350_	_LT_1500	_NODATA_	_LT_5_	_7-9_
1998.0000	WALLACE	_GE2200_	_LT_1500	_NODATA_	_LT_5_	_7-9_
1998.0000	WALLACE	_GE2200_	_LT_1500	_NODATA_	_LT_5_	_7-9_
1999.0000	KINGSTON	_LT_150_	_LT_150_	_NODATA_	_LT_5_	_1-2_
1999.0000	OSBURN	_LT_650_	_LT_350_	_NODATA_	_LT_5_	_3-4_
1999.0000	KINGSTON	_LT_350_	_LT_350_	_NODATA_	_LT_5_	_3-4_
1999.0000	SILVERTO	_LT_150_	_LT_350_	_NODATA_	_LT_5_	_7-9_

Year	Area	Yard Soil	Vacuum Dust	Dust mat	Blood Lead	Age
YRBLPB	SUBAREA	SPBCAT	HDPBCAT	HMPBCAT	BLPBCAT	AGECAT
1999.0000	KINGSTON	_LT_75__	_LT_350_	_NODATA_	_LT_5_	_5-6_
1998.0000	OSBURN	_LT_650_	_LT_350_	_NODATA_	_LT_5_	_7-9_
1999.0000	KINGSTON	_LT_350_	_LT_550_	_NODATA_	_LT_5_	_7-9_
1998.0000	SILVERTO	_LT_900_	_LT_550_	_NODATA_	_LT_5_	_5-6_
1999.0000	OSBURN	_LT_500_	_LT_550_	_NODATA_	_LT_5_	_1-2_
1999.0000	OSBURN	_LT_500_	_LT_550_	_NODATA_	_LT_5_	_7-9_
1999.0000	SIDE GUL	_LT_100_	_LT_550_	_NODATA_	_LT_5_	_1-2_
1999.0000	SIDE GUL	_LT_100_	_LT_550_	_NODATA_	_LT_5_	_3-4_
1999.0000	MULLAN	_LT_650_	_LT_750_	_NODATA_	_LT_5_	_7-9_
1999.0000	MULLAN	_LT_2200	_LT_750_	_NODATA_	_LT_5_	_7-9_
1999.0000	SILVERTO	_LT_1500	_LT_750_	_NODATA_	_LT_5_	_1-2_
1999.0000	WALLACE	_LT_1500	_LT_750_	_NODATA_	_LT_5_	_5-6_
1996.0000	SIDE GUL	_LT_100_	_NODATA_	_NODATA_	_LT_5_	_1-2_
1998.0000	NINE MIL	_LT_100_	_NODATA_	_NODATA_	_LT_5_	_5-6_
1998.0000	WALLACE	_LT_1100	_NODATA_	_NODATA_	_LT_5_	_5-6_
1996.0000	SILVERTO	_LT_150_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1996.0000	SIDE GUL	_LT_200_	_NODATA_	_NODATA_	_LT_5_	_1-2_
1996.0000	WALLACE	_LT_2200	_NODATA_	_NODATA_	_LT_5_	_1-2_
1999.0000	SIDE GUL	_LT_350_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1996.0000	OSBURN	_LT_500_	_NODATA_	_NODATA_	_LT_5_	_1-2_
1999.0000	OSBURN	_LT_500_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1998.0000	SIDE GUL	_LT_500_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1999.0000	SIDE GUL	_LT_500_	_NODATA_	_NODATA_	_LT_5_	_3-4_
1998.0000	OSBURN	_LT_500_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1999.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1997.0000	LOWER BA	_LT_75__	_NODATA_	_NODATA_	_LT_5_	_1-2_
1998.0000	SIDE GUL	_LT_900_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1998.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1998.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1999.0000	OSBURN	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1999.0000	KINGSTON	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_5_	_7-9_
1998.0000	WALLACE	_GE2200_	_LT_1000	_GE_3250	_LT_7_	_7-9_
1996.0000	KINGSTON	_LT_250_	_NODATA_	_GE_3250	_LT_7_	_5-6_
1999.0000	OSBURN	_LT_500_	_LT_550_	_GE_3250	_LT_7_	_5-6_
1999.0000	OSBURN	_LT_500_	_LT_550_	_GE_3250	_LT_7_	_7-9_
1999.0000	NINE MIL	_LT_650_	_NODATA_	_LT_1000	_LT_7_	_5-6_
1998.0000	OSBURN	_LT_2200	_NODATA_	_LT_1000	_LT_7_	_5-6_
1996.0000	MULLAN	_LT_900_	_NODATA_	_LT_1000	_LT_7_	_1-2_
1996.0000	SILVERTO	_LT_500_	_NODATA_	_LT_1000	_LT_7_	_3-4_
1996.0000	SILVERTO	_LT_500_	_NODATA_	_LT_1000	_LT_7_	_3-4_
1999.0000	OSBURN	_LT_1100	_LT_350_	_LT_1000	_LT_7_	_5-6_
1996.0000	MULLAN	_LT_350_	_NODATA_	_LT_1000	_LT_7_	_5-6_
1996.0000	MULLAN	_LT_350_	_NODATA_	_LT_1000	_LT_7_	_7-9_
1996.0000	OSBURN	_LT_250_	_NODATA_	_LT_3250	_LT_7_	_1-2_
1999.0000	NINE MIL	_LT_900_	_LT_1500	_LT_3250	_LT_7_	_7-9_
1996.0000	SILVERTO	_LT_150_	_NODATA_	_LT_550_	_LT_7_	_1-2_
1999.0000	SIDE GUL	_LT_350_	_LT_550_	_LT_850_	_LT_7_	_3-4_
1998.0000	SILVERTO	_LT_350_	_NODATA_	_LT_850_	_LT_7_	_3-4_

Year	Area	Yard Soil	Vacuum Dust	Dust mat	Blood Lead	Age
YRBLPB	SUBAREA	SPBCAT	HDPBCAT	HMPBCAT	BLPBCAT	AGECAT
1996.0000	OSBURN	_GE2200_	_NODATA_	_LT_850_	_LT_7_	_5-6_
1996.0000	OSBURN	_GE2200_	_NODATA_	_LT_850_	_LT_7_	_7-9_
1998.0000	NINE MIL	_LT_1500	_GE_1500	_NODATA_	_LT_7_	_5-6_
1999.0000	SILVERTO	_LT_150_	_GE_1500	_NODATA_	_LT_7_	_1-2_
1998.0000	NINE MIL	_GE2200_	_GE_1500	_NODATA_	_LT_7_	_1-2_
1998.0000	OSBURN	_LT_1100	_LT_1000	_NODATA_	_LT_7_	_5-6_
1999.0000	OSBURN	_LT_1100	_LT_1000	_NODATA_	_LT_7_	_7-9_
1999.0000	WALLACE	_LT_1500	_LT_1000	_NODATA_	_LT_7_	_7-9_
1999.0000	WALLACE	_LT_1100	_LT_1000	_NODATA_	_LT_7_	_7-9_
1999.0000	OSBURN	_LT_500_	_LT_1500	_NODATA_	_LT_7_	_1-2_
1999.0000	LOWER BA	_LT_75__	_LT_150_	_NODATA_	_LT_7_	_7-9_
1999.0000	NINE MIL	_LT_1500	_LT_150_	_NODATA_	_LT_7_	_5-6_
1998.0000	OSBURN	_LT_500_	_LT_150_	_NODATA_	_LT_7_	_7-9_
1999.0000	KINGSTON	_LT_200_	_LT_350_	_NODATA_	_LT_7_	_3-4_
1998.0000	SILVERTO	_LT_900_	_LT_550_	_NODATA_	_LT_7_	_3-4_
1999.0000	SIDE GUL	_LT_75__	_LT_550_	_NODATA_	_LT_7_	_1-2_
1999.0000	NINE MIL	_LT_1100	_LT_750_	_NODATA_	_LT_7_	_5-6_
1998.0000	NINE MIL	_LT_1100	_LT_750_	_NODATA_	_LT_7_	_1-2_
1999.0000	NINE MIL	_LT_100_	_LT_750_	_NODATA_	_LT_7_	_5-6_
1999.0000	KINGSTON	_LT_500_	_LT_750_	_NODATA_	_LT_7_	_1-2_
1999.0000	OSBURN	_LT_900_	_LT_750_	_NODATA_	_LT_7_	_5-6_
1999.0000	WALLACE	_LT_1500	_LT_750_	_NODATA_	_LT_7_	_1-2_
1998.0000	SILVERTO	_LT_2200	_LT_750_	_NODATA_	_LT_7_	_3-4_
1999.0000	SIDE GUL	_LT_75__	_LT_750_	_NODATA_	_LT_7_	_1-2_
1998.0000	NINE MIL	_LT_100_	_NODATA_	_NODATA_	_LT_7_	_5-6_
1999.0000	WALLACE	_LT_1100	_NODATA_	_NODATA_	_LT_7_	_1-2_
1998.0000	WALLACE	_LT_1100	_NODATA_	_NODATA_	_LT_7_	_3-4_
1998.0000	WALLACE	_LT_1100	_NODATA_	_NODATA_	_LT_7_	_7-9_
1997.0000	OSBURN	_LT_1500	_NODATA_	_NODATA_	_LT_7_	_3-4_
1998.0000	SILVERTO	_LT_200_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1998.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1996.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_7_	_5-6_
1996.0000	NINE MIL	_LT_75__	_NODATA_	_NODATA_	_LT_7_	_7-9_
1999.0000	LOWER BA	_LT_75__	_NODATA_	_NODATA_	_LT_7_	_3-4_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1998.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1998.0000	OSBURN	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1999.0000	MULLAN	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_1-2_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_1-2_
1999.0000	MULLAN	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1999.0000	SIDE GUL	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_5-6_
1999.0000	KINGSTON	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1999.0000	OSBURN	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1999.0000	NINE MIL	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1999.0000	NINE MIL	_LT_650_	_NODATA_	_LT_1000	_LT_7_	_5-6_
1999.0000	NINE MIL	_LT_650_	_NODATA_	_LT_1000	_LT_7_	_7-9_
1998.0000	SILVERTO	_LT_500_	_NODATA_	_LT_1000	_LT_7_	_5-6_

Year	Area	Yard Soil	Vacuum Dust	Dust mat	Blood Lead	Age
YRBLPB	SUBAREA	SPBCAT	HDPBCAT	HMPBCAT	BLPBCAT	AGECAT
1998.0000	SILVERTO	_LT_500_	_NODATA_	_LT_1000	_LT_7_	_3-4_
1999.0000	LOWER BA	_LT_75__	_NODATA_	_LT_350_	_LT_7_	_1-2_
1999.0000	KINGSTON	_LT_900_	_LT_350_	_LT_550_	_LT_7_	_3-4_
1996.0000	NINE MIL	_LT_75__	_NODATA_	_LT_850_	_LT_7_	_7-9_
1998.0000	OSBURN	_LT_350_	_NODATA_	_LT_850_	_LT_7_	_7-9_
1998.0000	SIDE GUL	_LT_350_	_LT_550_	_LT_850_	_LT_7_	_5-6_
1998.0000	SILVERTO	_LT_350_	_NODATA_	_LT_850_	_LT_7_	_1-2_
1999.0000	SILVERTO	_LT_150_	_GE_1500	_NODATA_	_LT_7_	_3-4_
1999.0000	NINE MIL	_LT_100_	_LT_1000	_NODATA_	_LT_7_	_5-6_
1997.0000	NINE MIL	_LT_150_	_LT_1500	_NODATA_	_LT_7_	_7-9_
1999.0000	MULLAN	_LT_1500	_LT_1500	_NODATA_	_LT_7_	_1-2_
1999.0000	SILVERTO	_LT_250_	_LT_1500	_NODATA_	_LT_7_	_1-2_
1998.0000	WALLACE	_LT_75__	_LT_1500	_NODATA_	_LT_7_	_5-6_
1998.0000	WALLACE	_GE2200_	_LT_1500	_NODATA_	_LT_7_	_5-6_
1999.0000	MULLAN	_LT_500_	_LT_1500	_NODATA_	_LT_7_	_5-6_
1999.0000	LOWER BA	_LT_75__	_LT_150_	_NODATA_	_LT_7_	_3-4_
1999.0000	KINGSTON	_LT_150_	_LT_150_	_NODATA_	_LT_7_	_3-4_
1999.0000	OSBURN	_LT_500_	_LT_150_	_NODATA_	_LT_7_	_5-6_
1998.0000	OSBURN	_LT_500_	_LT_150_	_NODATA_	_LT_7_	_5-6_
1999.0000	OSBURN	_LT_500_	_LT_150_	_NODATA_	_LT_7_	_5-6_
1999.0000	KINGSTON	_LT_350_	_LT_350_	_NODATA_	_LT_7_	_3-4_
1998.0000	SILVERTO	_LT_900_	_LT_550_	_NODATA_	_LT_7_	_7-9_
1999.0000	WALLACE	_LT_1500	_LT_750_	_NODATA_	_LT_7_	_7-9_
1998.0000	NINE MIL	_LT_100_	_NODATA_	_NODATA_	_LT_7_	_1-2_
1998.0000	SILVERTO	_LT_100_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1999.0000	OSBURN	_LT_100_	_NODATA_	_NODATA_	_LT_7_	_1-2_
1996.0000	OSBURN	_LT_1100	_NODATA_	_NODATA_	_LT_7_	_5-6_
1999.0000	SIDE GUL	_LT_1100	_NODATA_	_NODATA_	_LT_7_	_7-9_
1999.0000	SIDE GUL	_LT_1100	_NODATA_	_NODATA_	_LT_7_	_7-9_
1997.0000	SILVERTO	_LT_200_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1998.0000	SILVERTO	_LT_200_	_NODATA_	_NODATA_	_LT_7_	_5-6_
1999.0000	SILVERTO	_LT_200_	_NODATA_	_NODATA_	_LT_7_	_5-6_
1999.0000	WALLACE	_LT_2200	_NODATA_	_NODATA_	_LT_7_	_5-6_
1999.0000	NINE MIL	_LT_2200	_NODATA_	_NODATA_	_LT_7_	_5-6_
1998.0000	SILVERTO	_LT_500_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1999.0000	WALLACE	_LT_500_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1999.0000	NINE MIL	_LT_500_	_NODATA_	_NODATA_	_LT_7_	_5-6_
1999.0000	OSBURN	_LT_650_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1999.0000	LOWER BA	_LT_75__	_NODATA_	_NODATA_	_LT_7_	_5-6_
1997.0000	LOWER BA	_LT_75__	_NODATA_	_NODATA_	_LT_7_	_3-4_
1996.0000	NINE MIL	_LT_900_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1999.0000	OSBURN	_LT_900_	_NODATA_	_NODATA_	_LT_7_	_5-6_
1999.0000	NINE MIL	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1998.0000	SIDE GUL	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_5-6_
1998.0000	MULLAN	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_1-2_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_3-4_
1998.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_5-6_
1998.0000	MULLAN	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_5-6_
1998.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_1-2_

Year	Area	Yard Soil	Vacuum Dust	Dust mat	Blood Lead	Age
YRBLPB	SUBAREA	SPBCAT	HDPBCAT	HMPBCAT	BLPBCAT	AGECAT
1999.0000	MULLAN	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_5-6_
1999.0000	NINE MIL	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1999.0000	KINGSTON	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1999.0000	MULLAN	_NODATA_	_NODATA_	_NODATA_	_LT_7_	_7-9_
1996.0000	KINGSTON	_LT_250_	_NODATA_	_GE_3250	_LT_10	_1-2_
1997.0000	OSBURN	_LT_2200	_NODATA_	_LT_1000	_LT_10	_1-2_
1996.0000	SILVERTO	_LT_500_	_LT_1000	_LT_1500	_LT_10	_1-2_
1999.0000	SIDE GUL	_LT_500_	_NODATA_	_LT_3250	_LT_10	_1-2_
1999.0000	KINGSTON	_LT_150_	_NODATA_	_LT_3250	_LT_10	_3-4_
1998.0000	SIDE GUL	_LT_150_	_NODATA_	_LT_350_	_LT_10	_1-2_
1998.0000	KINGSTON	_LT_900_	_LT_350_	_LT_550_	_LT_10	_1-2_
1998.0000	OSBURN	_LT_350_	_LT_350_	_LT_850_	_LT_10	_1-2_
1999.0000	OSBURN	_LT_350_	_LT_350_	_LT_850_	_LT_10	_1-2_
1999.0000	OSBURN	_LT_500_	_NODATA_	_LT_850_	_LT_10	_5-6_
1999.0000	NINE MIL	_LT_100_	_LT_750_	_NODATA_	_LT_10	_5-6_
1998.0000	OSBURN	_LT_150_	_NODATA_	_NODATA_	_LT_10	_1-2_
1999.0000	SIDE GUL	_LT_350_	_NODATA_	_NODATA_	_LT_10	_3-4_
1999.0000	MULLAN	_LT_350_	_NODATA_	_NODATA_	_LT_10	_7-9_
1996.0000	MULLAN	_LT_500_	_NODATA_	_NODATA_	_LT_10	_7-9_
1999.0000	SILVERTO	_LT_500_	_NODATA_	_NODATA_	_LT_10	_3-4_
1998.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_10	_3-4_
1998.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_10	_3-4_
1999.0000	NINE MIL	_LT_650_	_NODATA_	_NODATA_	_LT_10	_7-9_
1999.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_10	_7-9_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_10	_3-4_
1998.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_10	_3-4_
1998.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_10	_5-6_
1998.0000	MULLAN	_NODATA_	_NODATA_	_NODATA_	_LT_10	_7-9_
1999.0000	SIDE GUL	_NODATA_	_NODATA_	_NODATA_	_LT_10	_1-2_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_10	_1-2_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_10	_7-9_
1996.0000	KINGSTON	_LT_250_	_NODATA_	_GE_3250	_LT_10	_5-6_
1998.0000	NINE MIL	_LT_2200	_NODATA_	_GE_3250	_LT_10	_7-9_
1996.0000	WALLACE	_LT_2200	_NODATA_	_LT_1500	_LT_10	_5-6_
1999.0000	NINE MIL	_GE2200_	_NODATA_	_LT_3250	_LT_10	_7-9_
1996.0000	NINE MIL	_LT_2200	_NODATA_	_LT_3250	_LT_10	_7-9_
1996.0000	NINE MIL	_LT_75__	_NODATA_	_LT_850_	_LT_10	_7-9_
1999.0000	NINE MIL	_LT_100_	_LT_1000	_NODATA_	_LT_10	_5-6_
1998.0000	WALLACE	_LT_75__	_LT_1500	_NODATA_	_LT_10	_3-4_
1999.0000	NINE MIL	_LT_1500	_LT_150_	_NODATA_	_LT_10	_3-4_
1999.0000	OSBURN	_LT_200_	_LT_150_	_NODATA_	_LT_10	_1-2_
1999.0000	OSBURN	_LT_500_	_LT_150_	_NODATA_	_LT_10	_1-2_
1998.0000	SILVERTO	_LT_900_	_LT_550_	_NODATA_	_LT_10	_1-2_
1996.0000	SILVERTO	_LT_1500	_NODATA_	_NODATA_	_LT_10	_3-4_
1999.0000	SILVERTO	_LT_1500	_NODATA_	_NODATA_	_LT_10	_5-6_
1999.0000	SILVERTO	_LT_1500	_NODATA_	_NODATA_	_LT_10	_7-9_
1999.0000	SILVERTO	_LT_200_	_NODATA_	_NODATA_	_LT_10	_1-2_
1996.0000	WALLACE	_LT_2200	_NODATA_	_NODATA_	_LT_10	_1-2_
1999.0000	KINGSTON	_LT_250_	_NODATA_	_NODATA_	_LT_10	_7-9_
1999.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_10	_3-4_

Year	Area	Yard Soil	Vacuum Dust	Dust mat	Blood Lead	Age
YRBLPB	SUBAREA	SPBCAT	HDPBCAT	HMPBCAT	BLPBCAT	AGECAT
1998.0000	OSBURN	_LT_900_	_NODATA_	_NODATA_	_LT_10	_5-6_
1997.0000	SILVERTO	_NODATA_	_NODATA_	_NODATA_	_LT_10	_7-9_
1999.0000	NINE MIL	_NODATA_	_NODATA_	_NODATA_	_LT_10	_7-9_
1999.0000	NINE MIL	_NODATA_	_NODATA_	_NODATA_	_LT_10	_3-4_
1998.0000	MULLAN	_NODATA_	_NODATA_	_NODATA_	_LT_10	_7-9_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_10	_3-4_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_10	_5-6_
1998.0000	NINE MIL	_GE2200_	_GE_1500	_GE_3250	_LT_10	_7-9_
1999.0000	OSBURN	_LT_1100	_LT_350_	_LT_1000	_LT_10	_7-9_
1999.0000	WALLACE	_LT_900_	_NODATA_	_LT_1500	_LT_10	_7-9_
1996.0000	NINE MIL	_GE2200_	_NODATA_	_LT_1500	_LT_10	_7-9_
1999.0000	NINE MIL	_GE2200_	_NODATA_	_LT_3250	_LT_10	_1-2_
1999.0000	NINE MIL	_GE2200_	_NODATA_	_LT_3250	_LT_10	_5-6_
1996.0000	NINE MIL	_LT_75__	_NODATA_	_LT_850_	_LT_10	_3-4_
1999.0000	SIDE GUL	_LT_500_	_GE_1500	_NODATA_	_LT_10	_1-2_
1999.0000	LOWER BA	_GE2200_	_LT_1500	_NODATA_	_LT_10	_5-6_
1999.0000	WALLACE	_LT_650_	_NODATA_	_NODATA_	_LT_10	_5-6_
1999.0000	MULLAN	_LT_900_	_NODATA_	_NODATA_	_LT_10	_1-2_
1999.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_10	_3-4_
1996.0000	LOWER BA	_GE2200_	_GE_1500	_GE_3250	_LT_15	_5-6_
1999.0000	MULLAN	_GE2200_	_GE_1500	_LT_3250	_LT_15	_7-9_
1996.0000	NINE MIL	_LT_2200	_NODATA_	_LT_3250	_LT_15	_5-6_
1996.0000	LOWER BA	_LT_75__	_NODATA_	_LT_850_	_LT_15	_7-9_
1999.0000	OSBURN	_LT_500_	_NODATA_	_LT_850_	_LT_15	_1-2_
1999.0000	SILVERTO	_LT_200_	_NODATA_	_NODATA_	_LT_15	_3-4_
1996.0000	SILVERTO	_LT_200_	_NODATA_	_NODATA_	_LT_15	_3-4_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_15	_3-4_
1996.0000	WALLACE	_LT_500_	_NODATA_	_LT_1000	_LT_15	_1-2_
1998.0000	MULLAN	_GE2200_	_GE_1500	_LT_3250	_LT_15	_7-9_
1996.0000	NINE MIL	_NODATA_	_NODATA_	_LT_3250	_LT_15	_7-9_
1996.0000	LOWER BA	_LT_350_	_NODATA_	_LT_550_	_LT_15	_5-6_
1999.0000	OSBURN	_LT_500_	_LT_550_	_NODATA_	_LT_15	_3-4_
1999.0000	MULLAN	_LT_2200	_LT_750_	_NODATA_	_LT_15	_1-2_
1999.0000	KINGSTON	_LT_250_	_NODATA_	_NODATA_	_LT_15	_3-4_
1999.0000	OSBURN	_LT_900_	_NODATA_	_NODATA_	_LT_15	_1-2_
1998.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_15	_1-2_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_LT_15	_1-2_
1997.0000	NINE MIL	_LT_900_	_GE_1500	_GE_3250	_LT_15	_7-9_
1997.0000	LOWER BA	_GE2200_	_LT_1500	_NODATA_	_LT_15	_3-4_
1999.0000	LOWER BA	_LT_75__	_LT_150_	_NODATA_	_LT_15	_1-2_
1999.0000	SILVERTO	_LT_350_	_LT_150_	_NODATA_	_LT_15	_5-6_
1999.0000	MULLAN	_LT_2200	_LT_750_	_NODATA_	_LT_15	_3-4_
1996.0000	OSBURN	_LT_250_	_NODATA_	_LT_3250	_LT_15	_7-9_
1999.0000	NINE MIL	_GE2200_	_NODATA_	_LT_3250	_LT_15	_5-6_
1998.0000	WALLACE	_LT_350_	_LT_1000	_LT_3250	_LT_15	_3-4_
1998.0000	NINE MIL	_LT_100_	_NODATA_	_NODATA_	_LT_15	_3-4_
1998.0000	WALLACE	_LT_1100	_NODATA_	_NODATA_	_LT_15	_1-2_
1998.0000	LOWER BA	_NODATA_	_NODATA_	_NODATA_	_LT_15	_3-4_
1996.0000	NINE MIL	_GE2200_	_NODATA_	_GE_3250	_LT_15	_7-9_
1999.0000	LOWER BA	_LT_75__	_LT_150_	_NODATA_	_LT_15	_1-2_

Year	Area	Yard Soil	Vacuum Dust	Dust mat	Blood Lead	Age
YRBLPB	SUBAREA	SPBCAT	HDPBCAT	HMPBCAT	BLPBCAT	AGECAT
1999.0000	KINGSTON	_LT_200_	_LT_750_	_NODATA_	_LT_15	_1-2_
1998.0000	SIDE GUL	_LT_350_	_LT_750_	_NODATA_	_LT_15	_7-9_
1996.0000	NINE MIL	_LT_2200	_NODATA_	_GE_3250	_GE_15	_7-9_
1996.0000	NINE MIL	_GE2200_	_NODATA_	_LT_1500	_GE_15	_7-9_
1999.0000	SILVERTO	_LT_350_	_LT_150_	_NODATA_	_GE_15	_1-2_
1999.0000	KINGSTON	_LT_900_	_NODATA_	_NODATA_	_GE_15	_1-2_
1998.0000	WALLACE	_LT_350_	_LT_1000	_LT_3250	_GE_15	_1-2_
1996.0000	KINGSTON	_LT_200_	_NODATA_	_LT_550_	_GE_15	_5-6_
1999.0000	LOWER BA	_LT_75__	_LT_150_	_NODATA_	_GE_15	_5-6_
1999.0000	KINGSTON	_LT_200_	_LT_750_	_NODATA_	_GE_15	_3-4_
1999.0000	SIDE GUL	_LT_350_	_LT_750_	_NODATA_	_GE_15	_7-9_
1996.0000	SILVERTO	_LT_200_	_NODATA_	_NODATA_	_GE_15	_1-2_
1999.0000	KINGSTON	_LT_900_	_NODATA_	_NODATA_	_GE_15	_3-4_
1996.0000	NINE MIL	_LT_900_	_GE_1500	_GE_3250	_GE_15	_7-9_
1996.0000	LOWER BA	_GE2200_	_NODATA_	_LT_3250	_GE_15	_1-2_
1999.0000	NINE MIL	_LT_150_	_LT_1500	_NODATA_	_GE_15	_3-4_
1999.0000	LOWER BA	_LT_75__	_LT_150_	_NODATA_	_GE_15	_3-4_
1997.0000	NINE MIL	_LT_2200	_NODATA_	_GE_3250	_GE_15	_7-9_
1998.0000	NINE MIL	_LT_2200	_NODATA_	_LT_1500	_GE_15	_1-2_
1999.0000	WALLACE	_LT_1100	_GE_1500	_LT_3250	_GE_15	_3-4_
1997.0000	NINE MIL	_LT_150_	_LT_1500	_NODATA_	_GE_15	_1-2_
1999.0000	NINE MIL	_LT_2200	_LT_1500	_LT_1500	_GE_15	_1-2_
1998.0000	NINE MIL	_LT_2200	_NODATA_	_LT_1500	_GE_15	_5-6_
1998.0000	NINE MIL	_LT_150_	_LT_1500	_NODATA_	_GE_15	_3-4_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_GE_15	_3-4_
1999.0000	SILVERTO	_LT_1500	_NODATA_	_NODATA_	_GE_15	_1-2_
1999.0000	WALLACE	_NODATA_	_NODATA_	_NODATA_	_GE_15	_1-2_

APPENDIX U

Cancer Rate Analysis in Shoshone County

COUNTY PROFILES

A fact sheet from the Cancer Data Registry of Idaho, Idaho Hospital Association.

SHOSHONE COUNTY

Cancer Incidence 1993-1997
Cancer Mortality 1993-1997
BRFSS 1989-1998

CANCER

Cancer is a group of more than 100 different diseases, each characterized by uncontrolled growth and spread of abnormal cells. Cancer risk increases with age, and varies by gender and race. As the average age of the population increases, the incidence of cancer will increase as well.

It is generally accepted that 65-80% of all cancers are related to personal lifestyle or environmental factors, such as smoking and diet, and are therefore preventable. Other factors such as age, gender, and family history of specific cancer are also associated with cancer and aid in the identification of people at high risk.

For some cancers, effective treatment is available. For these cancers, early detection saves lives. For example, early detection of breast cancer in women 50 years of age and older has decreased breast cancer mortality by 30%. These patterns indicate opportunities for disease control and for reducing the number of cancer deaths through prevention, early detection, and treatment of the disease. Access to detection services is a key consideration.

RISK FACTORS AND INTERVENTIONS

Aging: Because the population is aging, the number of new cancer cases and cancer deaths that occur each year will continue to increase unless the trend is reversed by significant improvements in prevention, early detection, and treatment.

Smoking: Smoking and the use of smokeless tobacco are responsible for the majority of all cancers of the lung, trachea, bronchus, larynx, pharynx, oral cavity, and esophagus. Smoking is the leading cause of preventable death in the United States.

Diet: The U.S. Department of Agriculture recommends the following dietary guidelines for managing a healthy diet: eat a variety of foods; maintain a healthy weight; choose a diet low in total fat with plenty of fruits, vegetables, and grain products; limit the use of sugar, salt, and sodium; and minimize alcoholic beverage consumption.

Screening: Early detection is extremely important for those cancers that can be cured and which can be discovered early. Breast cancer is a good example of this, as stage at diagnosis is the strongest predictor for survival from breast cancer.

FOR MORE INFORMATION

Cancer Data Registry of Idaho
802 West Bannock, Suite 500
P.O. Box 1278, Boise, ID 83701
208-338-5100 ext. 213

National Cancer Institute
Cancer Information Services
1-800-4CANCER

American Cancer Society
2676 South Vista Avenue,
Boise, ID 83705
(208) 343-4609

CANCER INCIDENCE 1993-1997

During the five-year period 1993-1997, 26,046 cases of cancer were diagnosed among residents of the state of Idaho, 505 among Shoshone County residents. It is estimated that four in ten Idahoans will develop cancer during their lifetime.

Cancer Incidence 1993-1997	Shoshone County	State of Idaho
All Sites/Types	505	26,046
Prostate	65	3,823
Female Breast	62	3,365
Lung	98	2,936
Colon	33	1,750

The table, *CANCER INCIDENCE 1993-1997, COMPARISON BETWEEN SHOSHONE COUNTY AND THE REMAINDER OF THE STATE OF IDAHO*, shows for Shoshone County the number of observed cases, person-years, crude rates, age and sex-adjusted rates, expected number of cases based upon age and sex-specific rates in the remainder of Idaho, and p-values for tests comparing the number of observed and expected cases. The table also shows the number of observed cases, person-years, and crude rates for the remainder of the state of Idaho. Comparisons were made for all cancers combined, and 24 specific cancer types. Separate comparisons for males, females, and both sexes combined are included.

As the table shows, the crude rate of cancer incidence in Shoshone County was 723.5 cases per 100,000 person-years for the years 1993-1997. Compared with the crude incidence rate for the remainder of Idaho (445.4), this gives an estimate of the burden of disease in Shoshone County.

The age- and sex-adjusted incidence rate of invasive cancer in Shoshone County, all sites combined, was 555.7 cases per 100,000 persons per year for the years 1993-1997. There were statistically significantly more cases of cancer in Shoshone County (505) than expected (405) based upon rates in the remainder of the state ($p<.001$).

There are many reasons why cancer incidence rates differ by county, related to smoking, other personal behaviors, socioeconomic status, and other factors. For some cancers, such as breast cancer, incidence rates are higher in areas with higher rates of cancer screening.

CANCER MORTALITY 1993-1997

Cancer is the second leading cause of deaths in Idaho and in the United States. From 1993-1997, 9,783 persons in Idaho died from cancer, 208 in Shoshone County. The majority of cancer deaths are from four sites: lung, colon, prostate, and female breast.

Mortality 1993-1997	Shoshone County	State of Idaho
All Deaths	884	42,865
Cancer Deaths % of All Deaths	208 23.5%	9,783 22.8%
Lung	74	2,474
Colon	10	804
Prostate	12	789
Female Breast	16	783

The table, *CANCER MORTALITY 1993-1997, COMPARISON BETWEEN SHOSHONE COUNTY AND THE REMAINDER OF THE STATE OF IDAHO*, shows for Shoshone County the number of observed deaths, person-years, crude rates, age and sex-adjusted rates, expected number of deaths based upon age and sex-specific rates in the remainder of Idaho, and p-values for tests comparing the number of observed and expected cases. The table also shows the number of observed deaths, person-years, and crude rates for the remainder of the state of Idaho. Comparisons were made for all deaths, all cancer deaths, and 22 specific cancer types. Separate comparisons for males, females, and both sexes combined are included.

The age- and sex-adjusted cancer mortality rate for Shoshone County, all sites combined, was 221.9 deaths per 100,000 persons per year for the years 1993-1997, compared with 167.0 for the remainder of the state. There were statistically significantly more deaths from cancer in Shoshone County (208) than expected (157) based upon rates in the remainder of the state ($p<.001$).

Statistical Note: Rates and percentages based upon 10 or fewer cases or deaths (numerator) should be interpreted with caution.
Data Note: Mortality data may differ slightly from published official statistics from the Center for Vital Statistics and Health Policy.

CANCER INCIDENCE 1993-1997
COMPARISON BETWEEN SHOSHONE COUNTY AND THE REMAINDER OF THE STATE OF IDAHO

Cancer Site/Type	Sex	Shoshone County						Remainder of Idaho		
		Observed Cases	Person Years	Crude Rate (1)	A.A.I. Rate (1,2)	Expected Cases (3)	P-Value (4)	Observed Cases	Person Years	Crude Rate (1)
All sites combined	Total	505	69,797	723.53	555.69	404.8	0.000 >>	25,541	5,734,062	445.43
All sites combined	Male	254	34,906	727.67	540.11	211.4	0.005 >>	12,878	2,864,948	449.50
All sites combined	Female	251	34,891	719.38	572.79	193.4	0.000 >>	12,663	2,869,114	441.36
Bladder	Total	27	69,797	38.68	28.26	18.1	0.061	1,089	5,734,062	18.99
Bladder	Male	17	34,906	48.70	35.33	14.4	0.558	857	2,864,948	29.91
Bladder	Female	10	34,891	28.66	21.53	3.8	0.011 >>	232	2,869,114	8.09
Brain	Total	1	69,797	1.43	1.22	5.3	0.065	367	5,734,062	6.40
Brain	Male	1	34,906	2.86	2.41	3.0	0.400	207	2,864,948	7.23
Brain	Female	-	34,891	-	-	2.3	0.207	160	2,869,114	5.58
Breast	Total	63	69,797	90.26	70.61	51.6	0.135	3,314	5,734,062	57.79
Breast	Male	1	34,906	2.86	2.13	0.2	0.330	11	2,864,948	0.38
Breast	Female	62	34,891	177.70	138.90	51.4	0.164	3,303	2,869,114	115.12
Cervix	Female	6	34,891	17.20	15.22	3.2	0.205	231	2,869,114	8.05
Colon	Total	33	69,797	47.28	34.75	28.4	0.438	1,717	5,734,062	29.94
Colon	Male	15	34,906	42.97	31.42	14.3	0.915	856	2,864,948	29.88
Colon	Female	18	34,891	51.59	38.12	14.2	0.370	861	2,869,114	30.01
Endometrium	Female	8	34,891	22.93	17.52	10.2	0.617	642	2,869,114	22.38
Esophagus	Total	6	69,797	8.60	6.32	3.2	0.214	194	5,734,062	3.38
Esophagus	Male	4	34,906	11.46	8.45	2.5	0.482	151	2,864,948	5.27
Esophagus	Female	2	34,891	5.73	4.16	0.7	0.326	43	2,869,114	1.50
Hodgkin"s Lymphoma	Total	5	69,797	7.16	6.81	1.9	0.096	152	5,734,062	2.65
Hodgkin"s Lymphoma	Male	3	34,906	8.59	8.20	1.1	0.183	83	2,864,948	2.90
Hodgkin"s Lymphoma	Female	2	34,891	5.73	5.43	0.9	0.445	69	2,869,114	2.40
Kidney and Renal Pelvis	Total	15	69,797	21.49	16.33	7.7	0.026 >>	483	5,734,062	8.42
Kidney and Renal Pelvis	Male	13	34,906	37.24	28.22	5.0	0.004 >>	312	2,864,948	10.89
Kidney and Renal Pelvis	Female	2	34,891	5.73	4.38	2.7	0.978	171	2,869,114	5.96
Larynx	Total	5	69,797	7.16	5.25	2.9	0.333	174	5,734,062	3.03
Larynx	Male	2	34,906	5.73	4.16	2.4	1.000	142	2,864,948	4.96
Larynx	Female	3	34,891	8.60	6.61	0.5	0.030 >>	32	2,869,114	1.12
Leukemia	Total	9	69,797	12.89	10.28	9.9	0.934	650	5,734,062	11.34
Leukemia	Male	2	34,906	5.73	4.48	5.9	0.130	381	2,864,948	13.30
Leukemia	Female	7	34,891	20.06	16.42	4.0	0.221	269	2,869,114	9.38
Liver	Total	4	69,797	5.73	4.43	1.8	0.226	116	5,734,062	2.02
Liver	Male	3	34,906	8.59	6.72	1.1	0.195	70	2,864,948	2.44
Liver	Female	1	34,891	2.87	2.18	0.7	1.000	46	2,869,114	1.60
Lung and Bronchus	Total	98	69,797	140.41	102.28	47.4	0.000 >>	2,838	5,734,062	49.49
Lung and Bronchus	Male	54	34,906	154.70	111.28	29.1	0.000 >>	1,718	2,864,948	59.97
Lung and Bronchus	Female	44	34,891	126.11	93.73	18.3	0.000 >>	1,120	2,869,114	39.04
Melanoma of the Skin	Total	12	69,797	17.19	14.12	14.2	0.678	958	5,734,062	16.71
Melanoma of the Skin	Male	4	34,906	11.46	9.16	8.2	0.176	539	2,864,948	18.81
Melanoma of the Skin	Female	8	34,891	22.93	19.52	6.0	0.508	419	2,869,114	14.60
Multiple Myeloma	Total	7	69,797	10.03	7.40	4.1	0.252	251	5,734,062	4.38
Multiple Myeloma	Male	3	34,906	8.59	6.32	2.3	0.793	137	2,864,948	4.78
Multiple Myeloma	Female	4	34,891	11.46	8.50	1.9	0.241	114	2,869,114	3.97
Non-Hodgkin"s Lymphoma	Total	17	69,797	24.36	18.85	14.7	0.621	937	5,734,062	16.34
Non-Hodgkin"s Lymphoma	Male	11	34,906	31.51	24.54	7.7	0.316	494	2,864,948	17.24
Non-Hodgkin"s Lymphoma	Female	6	34,891	17.20	13.22	7.0	0.897	443	2,869,114	15.44
Oral Cavity and Pharynx	Total	8	69,797	11.46	8.73	9.9	0.686	620	5,734,062	10.81
Oral Cavity and Pharynx	Male	5	34,906	14.32	10.80	7.3	0.530	451	2,864,948	15.74
Oral Cavity and Pharynx	Female	3	34,891	8.60	6.75	2.6	0.973	169	2,869,114	5.89
Ovary	Female	6	34,891	17.20	13.73	7.1	0.871	466	2,869,114	16.24
Pancreas	Total	11	69,797	15.76	11.58	7.7	0.309	464	5,734,062	8.09
Pancreas	Male	7	34,906	20.05	14.54	4.2	0.257	248	2,864,948	8.66
Pancreas	Female	4	34,891	11.46	8.55	3.5	0.936	216	2,869,114	7.53
Prostate	Male	65	34,906	186.21	132.62	64.3	0.963	3,758	2,864,948	131.17
Rectum & Rectosigmoid	Total	21	69,797	30.09	22.32	11.1	0.011 >>	678	5,734,062	11.82
Rectum & Rectosigmoid	Male	16	34,906	45.84	33.82	6.7	0.003 >>	403	2,864,948	14.07
Rectum & Rectosigmoid	Female	5	34,891	14.33	10.72	4.5	0.924	275	2,869,114	9.58
Stomach	Total	5	69,797	7.16	5.37	5.3	1.000	324	5,734,062	5.65
Stomach	Male	3	34,906	8.59	6.43	3.4	1.000	210	2,864,948	7.33
Stomach	Female	2	34,891	5.73	4.32	1.8	1.000	114	2,869,114	3.97
Testis	Male	3	34,906	8.59	9.20	1.8	0.551	160	2,864,948	5.58
Thyroid	Total	2	69,797	2.87	2.62	4.4	0.375	329	5,734,062	5.74
Thyroid	Male	-	34,906	-	-	1.1	0.675	79	2,864,948	2.76
Thyroid	Female	2	34,891	5.73	5.29	3.3	0.721	250	2,869,114	8.71

Notes: 1. Rates are expressed as the number of cases per 100,000 persons per year (person-years).
2. Age and sex-adjusted incidence (A.A.I.) rates for county used age and sex-specific crude rates for the remainder of the state as standard.
3. Expected cases are based upon age and sex-specific rates for the remainder of the state of Idaho (compare to observed).
4. P-values compare observed and expected cases, are two tailed, based upon the Poisson probability distribution.
"<<" denotes significantly fewer cases observed than expected, ">>" denotes significantly more cases observed than expected (p=.05).

Statistical Note: Rates based upon 10 or fewer cases (numerator) should be interpreted with caution.

CANCER MORTALITY 1993-1997
COMPARISON BETWEEN SHOSHONE COUNTY AND THE REMAINDER OF THE STATE OF IDAHO

Cause of Death Cancer Site/Type	Sex	Shoshone County Observed Deaths	Person Years	Crude Rate (1)	A.A.M. Rate (1,2)	Expected Deaths (3)	P-Value (4)	Remainder of Idaho Observed Deaths	Person Years	Crude Rate (1)
All Causes of Death	Total	884	69,797	1,266.53	973.54	664.8	0.000 >>	41,981	5,734,062	732.13
All Causes of Death	Male	460	34,906	1,317.83	1,014.57	350.3	0.000 >>	22,137	2,864,948	772.68
All Causes of Death	Female	424	34,891	1,215.21	932.55	314.5	0.000 >>	19,844	2,869,114	691.64
All Malignant Cancers	Total	208	69,797	298.01	221.85	156.6	0.000 >>	9,575	5,734,062	166.98
All Malignant Cancers	Male	103	34,906	295.08	217.26	86.9	0.101	5,254	2,864,948	183.39
All Malignant Cancers	Female	105	34,891	300.94	227.14	69.6	0.000 >>	4,321	2,869,114	150.60
Bladder	Total	8	69,797	11.46	8.44	3.7	0.071	224	5,734,062	3.91
Bladder	Male	2	34,906	5.73	4.18	2.7	1.000	160	2,864,948	5.58
Bladder	Female	6	34,891	17.20	12.98	1.0	0.001 >>	64	2,869,114	2.23
Brain	Total	1	69,797	1.43	1.15	4.4	0.138	286	5,734,062	4.99
Brain	Male	1	34,906	2.86	2.27	2.5	0.566	164	2,864,948	5.72
Brain	Female	-	34,891	-	-	1.8	0.321	122	2,869,114	4.25
Breast	Total	17	69,797	24.36	18.80	12.2	0.222	772	5,734,062	13.46
Breast	Male	1	34,906	2.86	2.06	0.1	0.162	5	2,864,948	0.17
Breast	Female	16	34,891	45.86	35.37	12.1	0.325	767	2,869,114	26.73
Cervix	Female	2	34,891	5.73	4.71	1.1	0.566	71	2,869,114	2.47
Colon	Total	10	69,797	14.33	10.58	13.1	0.489	794	5,734,062	13.85
Colon	Male	4	34,906	11.46	8.41	7.1	0.324	429	2,864,948	14.97
Colon	Female	6	34,891	17.20	12.80	6.0	1.000	365	2,869,114	12.72
Endometrium	Female	3	34,891	8.60	6.46	0.7	0.067	43	2,869,114	1.50
Esophagus	Total	8	69,797	11.46	8.44	3.5	0.055	213	5,734,062	3.71
Esophagus	Male	7	34,906	20.05	14.79	2.8	0.048 >>	169	2,864,948	5.90
Esophagus	Female	1	34,891	2.87	2.11	0.7	1.000	44	2,869,114	1.53
Hodgkin's Lymphoma	Total	-	69,797	-	-	0.4	1.000	27	5,734,062	0.47
Hodgkin's Lymphoma	Male	-	34,906	-	-	0.3	1.000	16	2,864,948	0.56
Hodgkin's Lymphoma	Female	-	34,891	-	-	0.2	1.000	11	2,869,114	0.38
Kidney and Renal Pelvis	Total	4	69,797	5.73	4.24	3.8	1.000	229	5,734,062	3.99
Kidney and Renal Pelvis	Male	3	34,906	8.59	6.35	2.8	1.000	169	2,864,948	5.90
Kidney and Renal Pelvis	Female	1	34,891	2.87	2.13	1.0	1.000	60	2,869,114	2.09
Larynx	Total	2	69,797	2.87	2.06	1.0	0.503	57	5,734,062	0.99
Larynx	Male	1	34,906	2.86	2.06	0.7	1.000	41	2,864,948	1.43
Larynx	Female	1	34,891	2.87	2.06	0.3	0.475	16	2,869,114	0.56
Leukemia	Total	3	69,797	4.30	3.28	6.6	0.206	416	5,734,062	7.25
Leukemia	Male	1	34,906	2.86	2.16	4.0	0.186	246	2,864,948	8.59
Leukemia	Female	2	34,891	5.73	4.47	2.7	1.000	170	2,869,114	5.93
Liver and I/H Bile Duct	Total	3	69,797	4.30	3.19	2.2	0.764	135	5,734,062	2.35
Liver and I/H Bile Duct	Male	2	34,906	5.73	4.24	1.3	0.712	76	2,864,948	2.65
Liver and I/H Bile Duct	Female	1	34,891	2.87	2.13	1.0	1.000	59	2,869,114	2.06
Lung and Bronchus	Total	74	69,797	106.02	76.99	40.2	0.000 >>	2,400	5,734,062	41.86
Lung and Bronchus	Male	36	34,906	103.13	74.21	25.0	0.044 >>	1,474	2,864,948	51.45
Lung and Bronchus	Female	38	34,891	108.91	80.31	15.3	0.000 >>	926	2,869,114	32.27
Melanoma of the Skin	Total	1	69,797	1.43	1.12	2.5	0.580	160	5,734,062	2.79
Melanoma of the Skin	Male	1	34,906	2.86	2.27	1.7	1.000	109	2,864,948	3.80
Melanoma of the Skin	Female	-	34,891	-	-	0.8	0.892	51	2,869,114	1.78
Multiple Myeloma	Total	4	69,797	5.73	4.22	3.6	0.963	217	5,734,062	3.78
Multiple Myeloma	Male	2	34,906	5.73	4.23	1.9	1.000	114	2,864,948	3.98
Multiple Myeloma	Female	2	34,891	5.73	4.22	1.7	1.000	103	2,869,114	3.59
Non-Hodgkin's Lymphoma	Total	8	69,797	11.46	8.59	7.3	0.880	447	5,734,062	7.80
Non-Hodgkin's Lymphoma	Male	4	34,906	11.46	8.58	4.1	1.000	249	2,864,948	8.69
Non-Hodgkin's Lymphoma	Female	4	34,891	11.46	8.60	3.2	0.800	198	2,869,114	6.90
Oral Cavity and Pharynx	Total	1	69,797	1.43	1.06	2.3	0.664	139	5,734,062	2.42
Oral Cavity and Pharynx	Male	1	34,906	2.86	2.08	1.5	1.000	89	2,864,948	3.11
Oral Cavity and Pharynx	Female	-	34,891	-	-	0.8	0.898	50	2,869,114	1.74
Ovary	Female	5	34,891	14.33	10.74	4.7	1.000	288	2,869,114	10.04
Pancreas	Total	12	69,797	17.19	12.62	7.7	0.178	462	5,734,062	8.06
Pancreas	Male	9	34,906	25.78	18.77	4.2	0.054	249	2,864,948	8.69
Pancreas	Female	3	34,891	8.60	6.37	3.5	1.000	213	2,869,114	7.42
Prostate	Male	12	34,906	34.38	25.21	12.9	0.946	777	2,864,948	27.12
Rectum and Rectosigmoid	Total	4	69,797	5.73	4.30	2.4	0.450	149	5,734,062	2.60
Rectum and Rectosigmoid	Male	3	34,906	8.59	6.43	1.3	0.265	77	2,864,948	2.69
Rectum and Rectosigmoid	Female	1	34,891	2.87	2.16	1.2	1.000	72	2,869,114	2.51
Stomach	Total	4	69,797	5.73	4.28	3.1	0.763	192	5,734,062	3.35
Stomach	Male	2	34,906	5.73	4.29	1.9	1.000	115	2,864,948	4.01
Stomach	Female	2	34,891	5.73	4.26	1.3	0.717	77	2,869,114	2.68

Notes: 1. Rates are expressed as the number of cases per 100,000 persons per year (person-years).
2. Age and sex-adjusted mortality (A.A.M.) rates for county used age and sex-specific crude rates for the remainder of the state as standard.
3. Expected cases are based upon age and sex-specific rates for the remainder of the state of Idaho (compare to observed).
4. P-values compare observed and expected cases, are two tailed, based upon the Poisson probability distribution.
"<<" denotes significantly fewer cases observed than expected, ">>" denotes significantly more cases observed than expected (p=.05).

Statistical Notes: Rates based upon 10 or fewer cases (numerator) should be interpreted with caution.
Mortality statistics presented differ from CVSHP official statistics due to differences in methodology.

Data Source: Center for Vital Statistics and Health Policy (CVSHP), Division of Health, Idaho Department of Health and Welfare, 7/99.

Cancer Risk Factors and Screening: Behavioral Risk Factor Surveillance System (BRFSS)

The Center for Vital Statistics and Health Policy (CVSHP), Division of Health, Idaho Department of Health and Welfare, under a cooperative agreement with the Centers for Disease Control and Prevention, has conducted telephone Behavioral Risk Factor Surveys since 1984 of random samples of adult Idahoans to measure population prevalences of risk factors for the major causes of death - including cancer. The CVSHP provided a data set containing aggregated BRFSS data from 1989 through 1998 to CDRI staff, who performed the analyses descibed in these *County Profiles*. Data were weighted by probability of selection, and poststratified to 1997 Idaho population estimates by age group, sex, and health district. A minimum of 30 respondents was required to generate county-level statistics.

BRFSS: Cancer Risk Factors and Screening Prevalence Estimates, 1989-1998

Residence	BRFS Respondents	Clinical Breast Exam	Mammogram	Pap Test	Smoking Current	Smoking Ever	Use Smokeless Tobacco Ever
State of Idaho	25,973	75.8%	62.9%	77.5%	20.5%	44.6%	20.8%
Health District 1	3,556	76.4%	63.2%	78.2%	24.5%	53.9%	21.4%
Health District 2	2,819	78.3%	68.0%	79.6%	19.7%	46.2%	26.8%
Health District 3	3,568	73.3%	59.8%	75.7%	21.3%	44.6%	19.2%
Health District 4	5,601	80.2%	68.9%	81.6%	21.5%	46.7%	21.8%
Health District 5	3,469	72.6%	60.8%	74.4%	19.3%	43.4%	20.6%
Health District 6	3,528	74.5%	54.4%	76.5%	19.4%	38.0%	18.9%
Health District 7	3,432	71.1%	62.0%	73.2%	15.3%	35.2%	17.4%
SHOSHONE	442	69.3%	54.4%	74.2%	29.4%	55.6%	30.5%

Clinical Breast Exam (CBE) - 1990 to 1998
Statewide, 75.8% of women aged 18 and older reported having a CBE in the past 2 years. Screening differed significantly by race/ethnicity, with 76.0% of white non-Hispanic women, compared to 72.0% of women of other racial and ethnic backgrounds, reporting having a CBE in the past 2 years. CBE utilization differed significantly by age of respondent, with 82.6% of women aged 25-34, but only 67.9% of women aged 65 and older, being screened. There was no trend by year of survey. CBE utilization differed significantly by county, with a range in screening of 58.2% (Madison County) to 88.3% (Blaine County). Results showed that women in counties with low rates of breast cancer may not be getting screened with CBE as well as women in other areas of Idaho.

Mammogram - 1989 to 1998
Statewide, 62.9% of women aged 50 and older reported having a mammogram in the past 2 years. Mammography utilization differed significantly by age of respondent, with 67.7% of women aged 50-54, and 60.4% of women aged 65 and older, being screened. There was no significant difference in screening by race/ethnicity. Mammography rates increased significantly over time, from 50.8% in 1989 to 69.4% in 1998. Mammography rates differed significantly by county, with a range in screening of 44.5% (Custer County) to 78.9% (Blaine County). Results showed that women in counties with low rates of early stage breast cancer may not be getting screened with mammograms as well as women in other areas of Idaho.

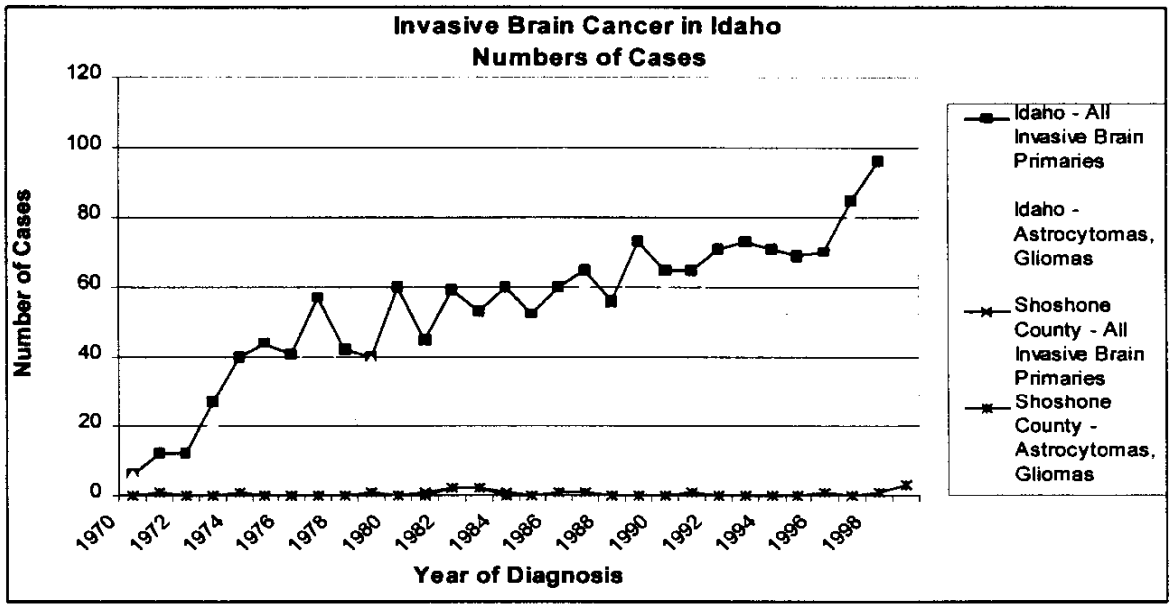
Pap Test - 1989 to 1998 (except 1990)
Statewide, 77.5% of women aged 18 and older (with intact cervix) reported having a pap test in the past 2 years. Pap screening differed significantly by age of respondent, with 86.8% of women aged 25-34, but only 59.3% of women aged 65 and older, screened in the past 2 years. Pap screening did not differ significantly by race/ethnicity, and there was no trend by year of survey. Pap screening differed significantly by county, with a range of 53.6% (Madison County) to 94.2% (Teton County).

Current Smoking - 1989 to 1998
Statewide, 20.5% of adults aged 18 and older were current smokers. Smoking prevalence differed significantly by age of respondent, with 24.2% of persons aged 35-44, and 10.7% of persons aged 65 and older, reporting current smoking. Twenty-two percent of males (21.8%) and 19.2% of females were current smokers, and smoking prevalence was lower among white non-Hispanics (20.3%) than among other race/ethnic groups (24.5%). There was no trend by year of survey. Smoking prevalence differed significantly by county, with a range of 5.2% (Madison County) to 29.4% (Shoshone County). Results showed that counties with higher rates of current smoking have higher rates of lung cancer.

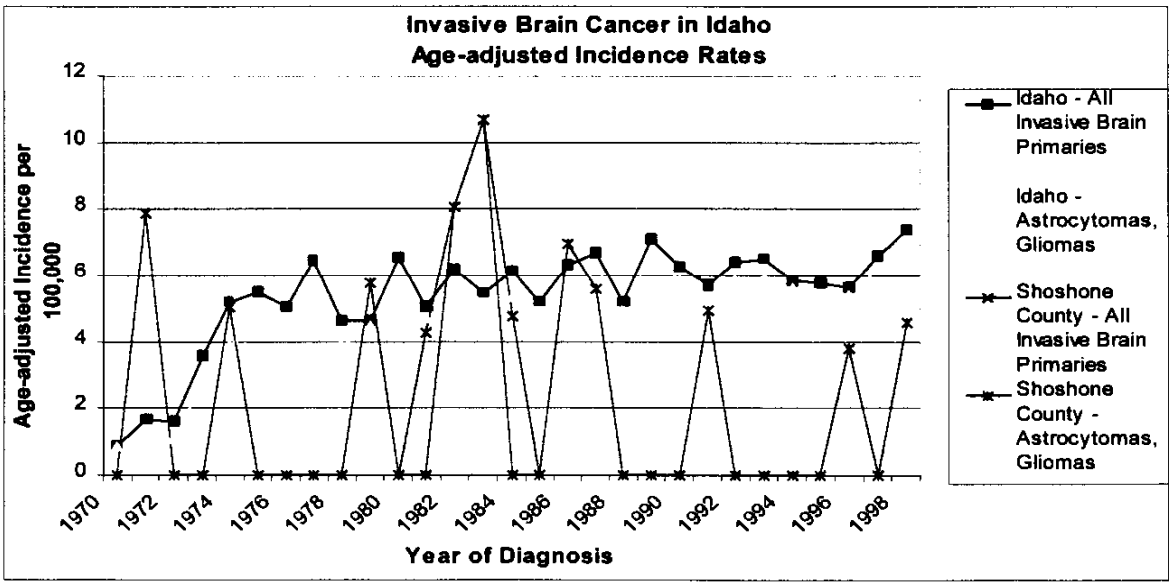
Smokeless Tobacco Use - 1992 to 1998 (except 1997)
Statewide, 20.8% of adults aged 18 and older ever used smokeless tobacco. Smokeless tobacco use differed significantly by age group, ranging from 30.6% of persons aged 18-24 to 10.8% of persons aged 65 and older. Thirty-eight percent of males (37.9%) and 4.2% of females ever used smokeless tobacco. There was no difference in smokeless tobacco use by race/ethnicity or year of survey. Ever using smokeless tobacco differed by county, with a range of 10.2% (Washington County) to 34.1% (Boise County). Results showed that counties with higher rates of smokeless tobacco use have higher rates of cancer of the oral cavity and pharynx.

Brain Cancer in Shoshone County

From 1970-1998, there were 1,569 cases of invasive brain cancer diagnosed among Idaho residents, 1,402 (89%) of which were astrocytoma/glioma. During this same time period, there were 14 cases of invasive brain cancer diagnosed among Shoshone County residents, 12 (86%) of which were astrocytoma/glioma (three additional cases have been diagnosed in 1999-2000). The graph below shows the numbers of cases by year of diagnosis for Shoshone County and Idaho.



During this time period, the average age-adjusted rates of invasive brain cancer and astrocytoma/glioma in the State of Idaho were 5.6 and 5.0 per 100,000, respectively. The rates in Shoshone County were less than half this, at 2.5 and 2.2 per 100,000.



From 1970-2000, the number of invasive brain cancer cases per year in Shoshone County has vacillated from 0-2, with 0 cases in 17 of the years, 1 case in 11 of the years, and 2 cases in each of three years. Given the average number of cases of 0.55 per year, there would need to be 3 or more cases in one calendar year for the count to be statistically significantly different from the county average. To date, this has not occurred. In addition, the Scan Test, a test of clustering for a single time series, has not revealed a time cluster with these data. The table below shows the numbers of cases of brain cancer that would need to occur in a calendar year for the resulting count to be statistically significant at the given p-value. Counts of three and higher have p-values less than 0.05, which would be considered to be statistically significantly different from the average number of cases expected.

SHOSHONE COUNTY BRAIN CANCER CASE COUNTS AND P-VALUES

CASES EXPECTED	COUNT	P VALUE
0.55	0	1.00000
0.55	1	0.84610
0.55	2	0.21146
0.55	3	0.03693
0.55	4	0.00493
0.55	5	0.00053
0.55	6	0.00005
0.55	7	0.00000
0.55	8	0.00000
0.55	9	0.00000
0.55	10	0.00000

Conclusion:

The incidence of brain cancer in Shoshone County has historically been lower than expected based upon rates in the remainder of Idaho. The new cases reported to date for diagnoses in 1999 and 2000 do not represent a statistical cluster in time. The incidence of brain cancer in Shoshone County is not higher than expected, but does warrant periodic reanalysis.

CANCER CLUSTER FACT SHEET

Cancer is a term that includes more than 100 different diseases, each characterized by the uncontrolled growth and spread of abnormal cells. A **CANCER CLUSTER** is the occurrence of a greater than expected number of cases of cancer within a small area or within a short period of time.

Cancer is one of America's greatest public health concerns. Four in ten persons in the United States will be diagnosed with cancer sometime in their life. Cancer is the second leading cause of death in both the United States and Idaho. In Idaho, cancer accounted for over 23 percent of deaths in 1998. When someone is diagnosed with or dies from cancer, family, friends and neighbors sometimes learn of other cases of cancer in their community. This apparent clustering of cancers is often reported to health departments or the media. However, closer inspection usually reveals that these "suspected" clusters involve several different ages, sexes and occupations. These cancer cases often have little or nothing in common (for example, some may have recently moved into the area), and are therefore not a "real" cancer cluster.

When several cancers occur within a limited area, this may represent a real cluster, but it may not be the result of an increased community risk of cancer. For example, in Idaho there are 44 counties. Every year, about half of the counties have rates of cancer that are above the average county value, and about half have rates that are below the average value. Counties may have above average rates one year and the next year the same counties may have rates below the average. This variation in expected and is more pronounced as the population being studied gets smaller (county, city, ZIP Code, neighborhood). Investigations of hundreds of reports of cancer clusters over many years by numerous states have shown approximately 15 percent of reported cancer clusters to be real clusters, based upon statistical evidence.

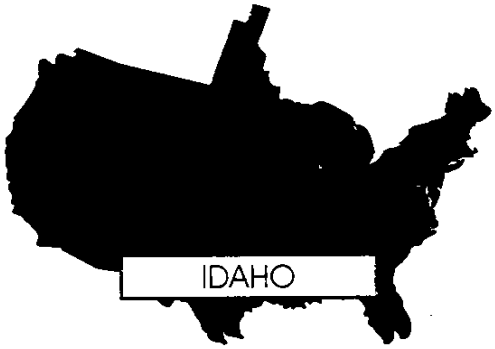
Cancer clusters that are a public health concern are the ones that represent a group of people who are at unusually high risk of cancer due to some factor or exposure that they have in common. A study of these clusters is sometimes necessary for the prevention of further cancers and to help understand more about specific risks for cancer. Understanding the reasons why the cancer risk is elevated may take months or longer, and the reasons are not always resolved. Less than 5 percent of all cluster reports fall into this category of "meaningful" cluster.

Cancer cluster investigations require data on the total number of residents and the number of diagnosed cancer cases in the area to be reviewed. At present time, the Cancer Data Registry of Idaho is able to investigate cancer incidence for several levels of geography: county, ZIP Code, Census Tract, and Census Block Group.

For more information regarding cancer clusters, contact:

Cancer Data Registry of Idaho
802 W. Bannock
Suite 500, PO Box 1278
Boise, Idaho 83701
(208) 338-5100 x213

Cancer Data Registry of Idaho



Your Source for Cancer Incidence and Survival Data for the State of Idaho

Cancer Data Registry of Idaho
P.O. Box 1278
Boise, ID 83701-1278
(208) 338-5100

CDRI
CANCER DATA REGISTRY OF IDAHO

CDRI

The Cancer Data Registry of Idaho (CDRI), a program of the Idaho Hospital Association (IHA), is a statewide cancer registry that collects incidence and survival data on all cancer patients who reside in the state of Idaho or who are diagnosed and/or treated for cancer in the state of Idaho. The goals of CDRI are to:

* determine the incidence of cancer in the state of Idaho with respect to geographic, demographic, and social characteristics;
* monitor trends and patterns of cancer incidence over time;
* identify high risk populations;
* provide a database and serve as a resource in conducting epidemiologic studies, and;
* provide data to assist public health officials, hospital administrators, and physicians to effectively plan services, prioritize health resource allocations and develop and measure prevention and intervention strategies

HISTORY AND FUNDING

The Idaho Hospital Association (IHA) contracts with the Idaho Department of Health and Welfare, Division of Health, to provide a statewide cancer surveillance system. CDRI was established in 1969 and became population-based in 1971. The Idaho State Legislature has provided guidelines for the establishment, requirements, and funding of the statewide cancer registry. The operations of the registry are mandated by Idaho Code 57-1703 through 57-1707. Funding is appropriated in Idaho Code 57-1701 and 63-2520 which delineates one percent of the cigarette tax to be dedicated to fund the statewide cancer registry. Additional funding has been awarded to CDRI from the Centers for Disease Control and Prevention with a federal grant (National Program of Cancer Registries).

CONFIDENTIALITY OF DATA

Idaho state law ensures the protection of confidential data and limits the release of identifying data. Only aggregate data are published. The same law protects report sources from liability for reporting data to CDRI.

COLLECTION OF DATA

Each Idaho hospital, out-patient surgery center, and pathology laboratory is responsible for the complete ascertainment of all data on cancer diagnoses and treatments provided in the facility within six months of diagnosis. Sources for identifying eligible cases include:

* hospitals,
* out-patient surgery centers,
* free-standing radiation centers,
* physicians,
* death certificates, and
* other state cancer registries reporting an Idaho resident with cancer (as negotiated).

When a cancer case is reported from more than one source the information is consolidated into one record.

REPORTABLE CASES

All in-situ or malignant neoplasms are reportable to CDRI. The database includes all cases of carcinoma, sarcoma, melanoma, lymphoma, and leukemia, diagnosed by histology/cytology, radiology, laboratory testing, clinical observation, and autopsy. Also reportable are benign tumors of the brain, meninges, pineal gland, and pituitary gland. Basal and squamous cell carcinomas of the skin are excluded except when occurring on a mucous membrane or if the stage group is II, III, or IV.

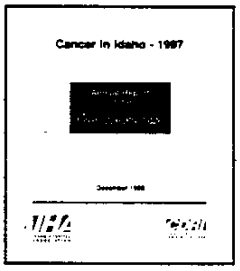
QUALITY ASSURANCE

To assure validity and reliability of data, CDRI uses software that checks the content of data fields against an encoded set of acceptable possible contents and flags the acceptability of coded data. Records are also routinely checked for duplicate entries.

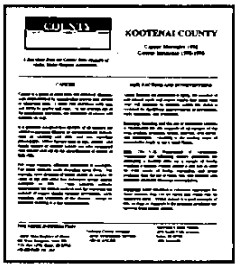
CDRI RESOURCES

Publications and More

You have access to an abundance of aggregate data through CDRI. You may request any of the following at no cost.

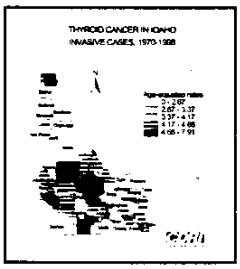


"Cancer in Idaho"
This annual publication contains summary data for one particular year on the most common 24 cancer types and all cancers combined. Data on cancer incidence and mortality are illustrated for the state as well as by health district. The publication contains statistics by cancer type, gender, and geographic area represented by stage of disease, age-adjusted incidence rates, age-specific rates, observed numbers of cases, and expected numbers of cases during the given year. Risk factors for each cancer type are also included.



"County Profiles"
These publications serve as fact sheets for each county in the state of Idaho. Each fact sheet contains incidence data for a five-year time period and mortality data for a one-year time period. Facts regarding risk factors and interventions as well as county-specific data by cancer type and gender are included. Data included are observed cases, crude rates, age-adjusted incidence rates, expected cases, and p-values; and, for comparison, observed cases and crude rates for the remainder of the state of Idaho. Mortality data are presented for each county compared with the state of Idaho.

"Data on the Web"
You are invited to visit our web site at:
HTTP://WWW.IDCANCER.ORG
Connecting with CDRI has never been easier than through our Web site. When you are online with CDRI, you have access to a wealth of resources including annual reports, background data on the cancer registry, our most recent special reports and common links to other useful Web sites.



"Special Reports"
Many reports are available for specific types of cancer such as breast cancer, thyroid cancer, or pediatric cancer. Special reports usually contain greater statistical and geographic detail.

"Cancer Cluster Fact Sheet"
This one page fact sheet defines a "cancer cluster" and how CDRI handles requests to conduct cancer cluster investigations.

Other Types of Data Analysis
If the listed publications do not meet your data needs, CDRI can perform data analysis specific to your needs. Just call a CDRI staff person and together we can design a report helpful to you.

HOSPITAL SERVICES

As a program of the Idaho Hospital Association, CDRI provides services to hospitals to help them comply with cancer reporting laws. The following services are provided:

- annual follow-up of cancer patients through physicians
- computerized monthly updates to hospital cancer registry database
- educational workshops for hospital cancer registrars
- on-site training of new registrars
- computer software training and on-going support
- video library for continuing education
- abstracting service to assist with backlog
- access to abstracting consultants for facilities with small caseloads
- quarterly newsletter to cancer registrars
- quality control of submitted data and feedback on error rates
- current documentation on minimum data set and data dictionary and abstracting procedures
- cancer data provided to assist with patient care evaluation studies, facility-specific annual reports, planning services, and evaluating early prevention and intervention strategies among served populations
- research of technical questions

RESOURCES

When you need cancer data, use this quick directory to contact CDRI, knowing your request will receive a timely and thoughtful response.

Address
Cancer Data Registry of Idaho
802 W. Bannock Street, Suite 500
P.O. Box 1278
Boise, ID 83701-1278

Phone
(208) 338-5100

FAX
(208) 338-7800

Web Site Address
http://www.idcancer.org

STAFF

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APPENDIX V
IEUBK Input Parameters

APPENDIX V

IEUBK Input Parameters

This appendix contains the input parameters and assumptions used in the IEUBK modeling presented in Chapter 6.

Table V-1. Batch Mode IEUBK Input Parameters Used to Create Table 6-49 and Tables 6-50a-c
(Current and Future Residential Exposure Scenario)

Exposure Medium	Model Specific Parameters	EPA-Default	Box-Model (40:30:30)
Air	Outdoor lead Conc.	0.10 ug/m^3	0.10 ug/m^3
	Indoor lead Conc. (% of outdoor)	30%	30%
	Time spent outdoors (0-1yr olds) hr/day	1	1
	Time spent outdoors (1-2yr olds) hr/day	2	2
	Time spent outdoors (2-3yr olds) hr/day	3	3
	Time spent outdoors (3-4yr olds) hr/day	4	4
	Time spent outdoors (4-5yr olds) hr/day	4	4
	Time spent outdoors (5-6yr olds) hr/day	4	4
	Time spent outdoors (6-7yr olds) hr/day	4	4
	Ventilation rate m^3/day (0-1 yr olds)	2	2
	Ventilation rate m^3/day (1-2 yr olds)	3	3
	Ventilation rate m^3/day (2-3 yr olds)	5	5
	Ventilation rate m^3/day (3-4 yr olds)	5	5
	Ventilation rate m^3/day (4-5 yr olds)	5	5
	Ventilation rate m^3/day (5-6 yr olds)	7	7
	Ventilation rate m^3/day (6-7 yr olds)	7	7
	Lung absorption %	32%	32%
Diet	Intake ug/day (0-1yr olds)	5.53	5.53
	Intake ug/day (1-2yr olds)	5.78	5.78
	Intake ug/day (2-3yr olds)	6.49	6.49
	Intake ug/day (3-4yr olds)	6.24	6.24
	Intake ug/day (4-5yr olds)	6.01	6.01
	Intake ug/day (5-6yr olds)	6.34	6.34
	Intake ug/day (6-7yr olds)	7.00	7.00
	Bioavailability	50%	50%
Alternate Sources	Total lead intake ug/day (0-1yr olds)	0.00	0.00
	Total lead intake ug/day (1-2yr olds)	0.00	0.00
	Total lead intake ug/day (2-3yr olds)	0.00	0.00
	Total lead intake ug/day (3-4yr olds)	0.00	0.00
	Total lead intake ug/day (4-5yr olds)	0.00	0.00
	Total lead intake ug/day (5-6yr olds)	0.00	0.00
	Total lead intake ug/day (6-7yr olds)	0.00	0.00
	Bioavailability	0%	0%
Drinking Water[1]	Lead Conc. In Drinking Water (ug/L)	Observation Specific*	Observation Specific*
	Bioavailability	50%	50%
	Water consumption L/day (0-1yr olds)	0.20	0.20
	Water consumption L/day (1-2yr olds)	0.50	0.50
	Water consumption L/day (2-3yr olds)	0.52	0.52
	Water consumption L/day (3-4yr olds)	0.53	0.53
	Water consumption L/day (4-5yr olds)	0.55	0.55
	Water consumption L/day (5-6yr olds)	0.58	0.58
	Water consumption L/day (6-7yr olds)	0.59	0.59
Soil/Dust[1]	Lead Conc. In Soil/Dust (mg/kg)	Observation Specific*	Observation Specific**
	Soil Dust Ingestion Weighting Factor (% soil)	45%	60%
	Bioavailability (soil)	30%	18%
	Bioavailability (dust)	30%	18%
Maternal-to-Newborn	Mother's blood lead at time of birth (ug/dL)	2.5	2.5
Computation Options	Iteration time step for numerical integration (hrs)	4	4
	Geometric Standard Deviation (unitless)	1.6	1.6

[1]Table 6-48 summarizes the observational specific data used in the batch mode IEUBK runs; because these data are confidential, observational specific data cannot be presented but are reported in ranges in Appendix T.

*Observational specific data were used; if not available then the geometric mean for the area was used; if yard soil data was missing, then the observation was deleted; the geometric mean used for water was a combination of both purged and 1996 well water summarized in Tables 6-11, separately.

**Weighted average for each individual soil observation was calculated using 30% yard soil and 30% mean community soil; if yard soil data was missing, then the observation was deleted.

Table V-2. Community Mode IEUBK Input Parameters Used to Create Tables 6-47a-c

(Current and Future Residential Exposure Scenario)

Exposure Medium	Model Specific Parameters	EPA-Default	Box-Model (40:30:30)
Air	Outdoor lead Conc.	0.10 ug/m^3	0.10 ug/m^3
	Indoor lead Conc. (% of outdoor)	30%	30%
	Time spent outdoors (0-1yr olds) hr/day	1	1
	Time spent outdoors (1-2yr olds) hr/day	2	2
	Time spent outdoors (2-3yr olds) hr/day	3	3
	Time spent outdoors (3-4yr olds) hr/day	4	4
	Time spent outdoors (4-5yr olds) hr/day	4	4
	Time spent outdoors (5-6yr olds) hr/day	4	4
	Time spent outdoors (6-7yr olds) hr/day	4	4
	Ventilation rate m^3/day (0-1 yr olds)	2	2
	Ventilation rate m^3/day (1-2 yr olds)	3	3
	Ventilation rate m^3/day (2-3 yr olds)	5	5
	Ventilation rate m^3/day (3-4 yr olds)	5	5
	Ventilation rate m^3/day (4-5 yr olds)	5	5
	Ventilation rate m^3/day (5-6 yr olds)	7	7
	Ventilation rate m^3/day (6-7 yr olds)	7	7
	Lung absorption %	32%	32%
Diet	Intake ug/day (0-1yr olds)	5.53	5.53
	Intake ug/day (1-2yr olds)	5.78	5.78
	Intake ug/day (2-3yr olds)	6.49	6.49
	Intake ug/day (3-4yr olds)	6.24	6.24
	Intake ug/day (4-5yr olds)	6.01	6.01
	Intake ug/day (5-6yr olds)	6.34	6.34
	Intake ug/day (6-7yr olds)	7.00	7.00
	Bioavailability	50%	50%
Alternate Sources	Total lead intake ug/day (0-1yr olds)	0.00	0.00
	Total lead intake ug/day (1-2yr olds)	0.00	0.00
	Total lead intake ug/day (2-3yr olds)	0.00	0.00
	Total lead intake ug/day (3-4yr olds)	0.00	0.00
	Total lead intake ug/day (4-5yr olds)	0.00	0.00
	Total lead intake ug/day (5-6yr olds)	0.00	0.00
	Total lead intake ug/day (6-7yr olds)	0.00	0.00
	Bioavailability	0%	0%
Drinking Water	Lead Conc. In Drinking Water (ug/L)	4	4
	Bioavailability	50%	50%
	Water consumption L/day (0-1yr olds)	0.20	0.20
	Water consumption L/day (1-2yr olds)	0.50	0.50
	Water consumption L/day (2-3yr olds)	0.52	0.52
	Water consumption L/day (3-4yr olds)	0.53	0.53
	Water consumption L/day (4-5yr olds)	0.55	0.55
	Water consumption L/day (5-6yr olds)	0.58	0.58
	Water consumption L/day (6-7yr olds)	0.59	0.59
Soil/Dust	Lead Conc. In Soil (mg/kg)	Geometric yard soil means from Tables 6-11a-h	Geometric yard soil means from Tables 6-11a-h
	Lead Conc. In Dust (mg/kg)	Geometric vacuum dust means from Tables 6-11a-h	Geometric vacuum dust means from Tables 6-11a-h
	Soil Dust Ingestion Weighting Factor (% soil)	45%	60%
	Bioavailability (soil)	30%	18%
	Bioavailability (dust)	30%	18%
Maternal-to-Newborn	Mother's blood lead at time of birth (ug/dL)	2.5	2.5
Computation Options	Iteration time step for numerical integration (hrs)	4	4
	Geometric Standard Deviation (unitless)	1.6	1.6

Table V-3. Community Mode IEUBK Input Parameters Used to Create Tables 6-51a-b

(Incremental Upland Parks Exposure Scenario - CT and RME)

Exposure Medium	Model Specific Parameters	EPA-Default	Box-Model (40:30:30)
Air	Outdoor lead Conc.	0.10 ug/m^3	0.10 ug/m^3
	Indoor lead Conc. (% of outdoor)	30%	30%
	Time spent outdoors (0-1yr olds) hr/day	1	1
	Time spent outdoors (1-2yr olds) hr/day	2	2
	Time spent outdoors (2-3yr olds) hr/day	3	3
	Time spent outdoors (3-4yr olds) hr/day	4	4
	Time spent outdoors (4-5yr olds) hr/day	4	4
	Time spent outdoors (5-6yr olds) hr/day	4	4
	Time spent outdoors (6-7yr olds) hr/day	4	4
	Ventilation rate m^3/day (0-1 yr olds)	2	2
	Ventilation rate m^3/day (1-2 yr olds)	3	3
	Ventilation rate m^3/day (2-3 yr olds)	5	5
	Ventilation rate m^3/day (3-4 yr olds)	5	5
	Ventilation rate m^3/day (4-5 yr olds)	5	5
	Ventilation rate m^3/day (5-6 yr olds)	7	7
	Ventilation rate m^3/day (6-7 yr olds)	7	7
	Lung absorption %	32%	32%
Diet	Intake ug/day (0-1yr olds)	5.53	5.53
	Intake ug/day (1-2yr olds)	5.78	5.78
	Intake ug/day (2-3yr olds)	6.49	6.49
	Intake ug/day (3-4yr olds)	6.24	6.24
	Intake ug/day (4-5yr olds)	6.01	6.01
	Intake ug/day (5-6yr olds)	6.34	6.34
	Intake ug/day (6-7yr olds)	7.00	7.00
	Bioavailability	50%	50%
Alternate Sources*	Total lead intake ug/day (0-1yr olds)	50% and 95% intake values from Table 6-35b	50% and 95% intake values from Table 6-35b
	Total lead intake ug/day (1-2yr olds)	50% and 95% intake values from Table 6-35b	50% and 95% intake values from Table 6-35b
	Total lead intake ug/day (2-3yr olds)	50% and 95% intake values from Table 6-35b	50% and 95% intake values from Table 6-35b
	Total lead intake ug/day (3-4yr olds)	50% and 95% intake values from Table 6-35b	50% and 95% intake values from Table 6-35b
	Total lead intake ug/day (4-5yr olds)	50% and 95% intake values from Table 6-35b	50% and 95% intake values from Table 6-35b
	Total lead intake ug/day (5-6yr olds)	50% and 95% intake values from Table 6-35b	50% and 95% intake values from Table 6-35b
	Total lead intake ug/day (6-7yr olds)	50% and 95% intake values from Table 6-35b	50% and 95% intake values from Table 6-35b
	Bioavailability	30%	18%
Drinking Water	Lead Conc. In Drinking Water (ug/L)	4	4
	Bioavailability	50%	50%
	Water consumption L/day (0-1yr olds)	0.20	0.20
	Water consumption L/day (1-2yr olds)	0.50	0.50
	Water consumption L/day (2-3yr olds)	0.52	0.52
	Water consumption L/day (3-4yr olds)	0.53	0.53
	Water consumption L/day (4-5yr olds)	0.55	0.55
	Water consumption L/day (5-6yr olds)	0.58	0.58
	Water consumption L/day (6-7yr olds)	0.59	0.59
Soil/Dust	Lead Conc. In Soil (mg/kg)	Geometric yard soil means from Tables 6-11a-h	Geometric yard soil means from Tables 6-11a-h
	Lead Conc. In Dust (mg/kg)	Geometric vacuum dust means from Tables 6-11a-h	Geometric vacuum dust means from Tables 6-11a-h
	Soil Dust Ingestion Weighting Factor (% soil)	45%	60%
	Bioavailability (soil)	30%	18%
	Bioavailability (dust)	30%	18%
Maternal-to-Newborn	Mother's blood lead at time of birth (ug/dL)	2.5	2.5
Computation Options	Iteration time step for numerical integration (hrs)	4	4
	Geometric Standard Deviation (unitless)	1.6	1.6

*Intake does not vary by age group

Table V-4. Community Mode IEUBK Input Parameters Used to Create Tables 6-52a-b
(Incremental Neighborhood Stream Recreational Exposure Scenario - CT and RME)

Exposure Medium	Model Specific Parameters	EPA-Default	Box-Model (40:30:30)
Air	Outdoor lead Conc.	0.10 ug/m^3	0.10 ug/m^3
	Indoor lead Conc. (% of outdoor)	30%	30%
	Time spent outdoors (0-1yr olds) hr/day	1	1
	Time spent outdoors (1-2yr olds) hr/day	2	2
	Time spent outdoors (2-3yr olds) hr/day	3	3
	Time spent outdoors (3-4yr olds) hr/day	4	4
	Time spent outdoors (4-5yr olds) hr/day	4	4
	Time spent outdoors (5-6yr olds) hr/day	4	4
	Time spent outdoors (6-7yr olds) hr/day	4	4
	Ventilation rate m^3/day (0-1 yr olds)	2	2
	Ventilation rate m^3/day (1-2 yr olds)	3	3
	Ventilation rate m^3/day (2-3 yr olds)	5	5
	Ventilation rate m^3/day (3-4 yr olds)	5	5
	Ventilation rate m^3/day (4-5 yr olds)	5	5
	Ventilation rate m^3/day (5-6 yr olds)	7	7
	Ventilation rate m^3/day (6-7 yr olds)	7	7
	Lung absorption %	32%	32%
Diet	Intake ug/day (0-1yr olds)	5.53	5.53
	Intake ug/day (1-2yr olds)	5.78	5.78
	Intake ug/day (2-3yr olds)	6.49	6.49
	Intake ug/day (3-4yr olds)	6.24	6.24
	Intake ug/day (4-5yr olds)	6.01	6.01
	Intake ug/day (5-6yr olds)	6.34	6.34
	Intake ug/day (6-7yr olds)	7.00	7.00
	Bioavailability	50%	50%
Alternate Sources*	Total lead intake ug/day (0-1yr olds)	50% and 95% intake values from Table 6-36b	50% and 95% intake values from Table 6-36b
	Total lead intake ug/day (1-2yr olds)	50% and 95% intake values from Table 6-36b	50% and 95% intake values from Table 6-36b
	Total lead intake ug/day (2-3yr olds)	50% and 95% intake values from Table 6-36b	50% and 95% intake values from Table 6-36b
	Total lead intake ug/day (3-4yr olds)	50% and 95% intake values from Table 6-36b	50% and 95% intake values from Table 6-36b
	Total lead intake ug/day (4-5yr olds)	50% and 95% intake values from Table 6-36b	50% and 95% intake values from Table 6-36b
	Total lead intake ug/day (5-6yr olds)	50% and 95% intake values from Table 6-36b	50% and 95% intake values from Table 6-36b
	Total lead intake ug/day (6-7yr olds)	50% and 95% intake values from Table 6-36b	50% and 95% intake values from Table 6-36b
	Bioavailability	30%	18%
Drinking Water	Lead Conc. In Drinking Water (ug/L)	4	4
	Bioavailability	50%	50%
	Water consumption L/day (0-1yr olds)	0.20	0.20
	Water consumption L/day (1-2yr olds)	0.50	0.50
	Water consumption L/day (2-3yr olds)	0.52	0.52
	Water consumption L/day (3-4yr olds)	0.53	0.53
	Water consumption L/day (4-5yr olds)	0.55	0.55
	Water consumption L/day (5-6yr olds)	0.58	0.58
	Water consumption L/day (6-7yr olds)	0.59	0.59
Soil/Dust	Lead Conc. In Soil (mg/kg)	Geometric yard soil means from Tables 6-11a-h	Geometric yard soil means from Tables 6-11a-h
	Lead Conc. In Dust (mg/kg)	Geometric vacuum dust means from Tables 6-11a-h	Geometric vacuum dust means from Tables 6-11a-h
	Soil Dust Ingestion Weighting Factor (% soil)	45%	60%
	Bioavailability (soil)	30%	18%
	Bioavailability (dust)	30%	18%
Maternal-to-Newborn	Mother's blood lead at time of birth (ug/dL)	2.5	2.5
Computation Options	Iteration time step for numerical integration (hrs)	4	4
	Geometric Standard Deviation (unitless)	1.6	1.6

*Intake does not vary by age group

Table V-5. Community Mode IEUBK Input Parameters Used to Create Tables 6-53a-b
(Incremental Public Beach Exposure Scenario - CT and RME)

Exposure Medium	Model Specific Parameters	EPA-Default	Box-Model (40:30:30)
Air	Outdoor lead Conc.	0.10 ug/m^3	0.10 ug/m^3
	Indoor lead Conc. (% of outdoor)	30%	30%
	Time spent outdoors (0-1yr olds) hr/day	1	1
	Time spent outdoors (1-2yr olds) hr/day	2	2
	Time spent outdoors (2-3yr olds) hr/day	3	3
	Time spent outdoors (3-4yr olds) hr/day	4	4
	Time spent outdoors (4-5yr olds) hr/day	4	4
	Time spent outdoors (5-6yr olds) hr/day	4	4
	Time spent outdoors (6-7yr olds) hr/day	4	4
	Ventilation rate m^3/day (0-1 yr olds)	2	2
	Ventilation rate m^3/day (1-2 yr olds)	3	3
	Ventilation rate m^3/day (2-3 yr olds)	5	5
	Ventilation rate m^3/day (3-4 yr olds)	5	5
	Ventilation rate m^3/day (4-5 yr olds)	5	5
	Ventilation rate m^3/day (5-6 yr olds)	7	7
	Ventilation rate m^3/day (6-7 yr olds)	7	7
	Lung absorption %	32%	32%
Diet	Intake ug/day (0-1yr olds)	5.53	5.53
	Intake ug/day (1-2yr olds)	5.78	5.78
	Intake ug/day (2-3yr olds)	6.49	6.49
	Intake ug/day (3-4yr olds)	6.24	6.24
	Intake ug/day (4-5yr olds)	6.01	6.01
	Intake ug/day (5-6yr olds)	6.34	6.34
	Intake ug/day (6-7yr olds)	7.00	7.00
	Bioavailability	50%	50%
Alternate Sources*	Total lead intake ug/day (0-1yr olds)	50% and 95% intake values from Table 6-37b	50% and 95% intake values from Table 6-37b
	Total lead intake ug/day (1-2yr olds)	50% and 95% intake values from Table 6-37b	50% and 95% intake values from Table 6-37b
	Total lead intake ug/day (2-3yr olds)	50% and 95% intake values from Table 6-37b	50% and 95% intake values from Table 6-37b
	Total lead intake ug/day (3-4yr olds)	50% and 95% intake values from Table 6-37b	50% and 95% intake values from Table 6-37b
	Total lead intake ug/day (4-5yr olds)	50% and 95% intake values from Table 6-37b	50% and 95% intake values from Table 6-37b
	Total lead intake ug/day (5-6yr olds)	50% and 95% intake values from Table 6-37b	50% and 95% intake values from Table 6-37b
	Total lead intake ug/day (6-7yr olds)	50% and 95% intake values from Table 6-37b	50% and 95% intake values from Table 6-37b
	Bioavailability	30%	18%
Drinking Water	Lead Conc. In Drinking Water (ug/L)	4	4
	Bioavailability	50%	50%
	Water consumption L/day (0-1yr olds)	0.20	0.20
	Water consumption L/day (1-2yr olds)	0.50	0.50
	Water consumption L/day (2-3yr olds)	0.52	0.52
	Water consumption L/day (3-4yr olds)	0.53	0.53
	Water consumption L/day (4-5yr olds)	0.55	0.55
	Water consumption L/day (5-6yr olds)	0.58	0.58
	Water consumption L/day (6-7yr olds)	0.59	0.59
Soil/Dust	Lead Conc. In Soil (mg/kg)	Geometric yard soil means from Tables 6-11a-h	Geometric yard soil means from Tables 6-11a-h
	Lead Conc. In Dust (mg/kg)	Geometric vacuum dust means from Tables 6-11a-h	Geometric vacuum dust means from Tables 6-11a-h
	Soil Dust Ingestion Weighting Factor (% soil)	45%	60%
	Bioavailability (soil)	30%	18%
	Bioavailability (dust)	30%	18%
Maternal-to-Newborn	Mother's blood lead at time of birth (ug/dL)	2.5	2.5
Computation Options	Iteration time step for numerical integration (hrs)	4	4
	Geometric Standard Deviation (unitless)	1.6	1.6

*Intake does not vary by age group

Table V-6. Community Mode IEUBK Input Parameters Used to Create Tables 6-54a-b
(Incremental Waste Pile Exposure Scenario - CT and RME)

Exposure Medium	Model Specific Parameters	EPA-Default	Box-Model (40:30:30)
Air	Outdoor lead Conc.	0.10 ug/m^3	0.10 ug/m^3
	Indoor lead Conc. (% of outdoor)	30%	30%
	Time spent outdoors (0-1yr olds) hr/day	1	1
	Time spent outdoors (1-2yr olds) hr/day	2	2
	Time spent outdoors (2-3yr olds) hr/day	3	3
	Time spent outdoors (3-4yr olds) hr/day	4	4
	Time spent outdoors (4-5yr olds) hr/day	4	4
	Time spent outdoors (5-6yr olds) hr/day	4	4
	Time spent outdoors (6-7yr olds) hr/day	4	4
	Ventilation rate m^3/day (0-1 yr olds)	2	2
	Ventilation rate m^3/day (1-2 yr olds)	3	3
	Ventilation rate m^3/day (2-3 yr olds)	5	5
	Ventilation rate m^3/day (3-4 yr olds)	5	5
	Ventilation rate m^3/day (4-5 yr olds)	5	5
	Ventilation rate m^3/day (5-6 yr olds)	7	7
	Ventilation rate m^3/day (6-7 yr olds)	7	7
	Lung absorption %	32%	32%
Diet	Intake ug/day (0-1yr olds)	5.53	5.53
	Intake ug/day (1-2yr olds)	5.78	5.78
	Intake ug/day (2-3yr olds)	6.49	6.49
	Intake ug/day (3-4yr olds)	6.24	6.24
	Intake ug/day (4-5yr olds)	6.01	6.01
	Intake ug/day (5-6yr olds)	6.34	6.34
	Intake ug/day (6-7yr olds)	7.00	7.00
	Bioavailability	50%	50%
Alternate Sources*	Total lead intake ug/day (0-1yr olds)	50% and 95% intake values from Table 6-38b	50% and 95% intake values from Table 6-38b
	Total lead intake ug/day (1-2yr olds)	50% and 95% intake values from Table 6-38b	50% and 95% intake values from Table 6-38b
	Total lead intake ug/day (2-3yr olds)	50% and 95% intake values from Table 6-38b	50% and 95% intake values from Table 6-38b
	Total lead intake ug/day (3-4yr olds)	50% and 95% intake values from Table 6-38b	50% and 95% intake values from Table 6-38b
	Total lead intake ug/day (4-5yr olds)	50% and 95% intake values from Table 6-38b	50% and 95% intake values from Table 6-38b
	Total lead intake ug/day (5-6yr olds)	50% and 95% intake values from Table 6-38b	50% and 95% intake values from Table 6-38b
	Total lead intake ug/day (6-7yr olds)	50% and 95% intake values from Table 6-38b	50% and 95% intake values from Table 6-38b
	Bioavailability	30%	18%
Drinking Water	Lead Conc. In Drinking Water (ug/L)	4	4
	Bioavailability	50%	50%
	Water consumption L/day (0-1yr olds)	0.20	0.20
	Water consumption L/day (1-2yr olds)	0.50	0.50
	Water consumption L/day (2-3yr olds)	0.52	0.52
	Water consumption L/day (3-4yr olds)	0.53	0.53
	Water consumption L/day (4-5yr olds)	0.55	0.55
	Water consumption L/day (5-6yr olds)	0.58	0.58
	Water consumption L/day (6-7yr olds)	0.59	0.59
Soil/Dust	Lead Conc. In Soil (mg/kg)	Geometric yard soil means from Tables 6-11a-h	Geometric yard soil means from Tables 6-11a-h
	Lead Conc. In Dust (mg/kg)	Geometric vacuum dust means from Tables 6-11a-h	Geometric vacuum dust means from Tables 6-11a-h
	Soil Dust Ingestion Weighting Factor (% soil)	45%	60%
	Bioavailability (soil)	30%	18%
	Bioavailability (dust)	30%	18%
Maternal-to-Newborn	Mother's blood lead at time of birth (ug/dL)	2.5	2.5
Computation Options	Iteration time step for numerical integration (hrs)	4	4
	Geometric Standard Deviation (unitless)	1.6	1.6

*Intake does not vary by age group

Table V-7. Community Mode IEUBK Input Parameters Used to Create Tables 6-55a-b

(Incremental Homegrown Vegetable Exposure Scenario - CT and RME)

Exposure Medium	Model Specific Parameters	EPA-Default	Box-Model (40:30:30)
Air	Outdoor lead Conc.	0.10 ug/m^3	0.10 ug/m^3
	Indoor lead Conc. (% of outdoor)	30%	30%
	Time spent outdoors (0-1yr olds) hr/day	1	1
	Time spent outdoors (1-2yr olds) hr/day	2	2
	Time spent outdoors (2-3yr olds) hr/day	3	3
	Time spent outdoors (3-4yr olds) hr/day	4	4
	Time spent outdoors (4-5yr olds) hr/day	4	4
	Time spent outdoors (5-6yr olds) hr/day	4	4
	Time spent outdoors (6-7yr olds) hr/day	4	4
	Ventilation rate m^3/day (0-1 yr olds)	2	2
	Ventilation rate m^3/day (1-2 yr olds)	3	3
	Ventilation rate m^3/day (2-3 yr olds)	5	5
	Ventilation rate m^3/day (3-4 yr olds)	5	5
	Ventilation rate m^3/day (4-5 yr olds)	5	5
	Ventilation rate m^3/day (5-6 yr olds)	7	7
	Ventilation rate m^3/day (6-7 yr olds)	7	7
	Lung absorption %	32%	32%
Diet	Intake ug/day (0-1yr olds)	5.53	5.53
	Intake ug/day (1-2yr olds)	5.78	5.78
	Intake ug/day (2-3yr olds)	6.49	6.49
	Intake ug/day (3-4yr olds)	6.24	6.24
	Intake ug/day (4-5yr olds)	6.01	6.01
	Intake ug/day (5-6yr olds)	6.34	6.34
	Intake ug/day (6-7yr olds)	7.00	7.00
	Bioavailability	50%	50%
Alternate Sources*	Total lead intake ug/day (0-1yr olds)	50% and 95% intake values from Table 6-40a	50% and 95% intake values from Table 6-40a
	Total lead intake ug/day (1-2yr olds)	50% and 95% intake values from Table 6-40a	50% and 95% intake values from Table 6-40a
	Total lead intake ug/day (2-3yr olds)	50% and 95% intake values from Table 6-40a	50% and 95% intake values from Table 6-40a
	Total lead intake ug/day (3-4yr olds)	50% and 95% intake values from Table 6-40a	50% and 95% intake values from Table 6-40a
	Total lead intake ug/day (4-5yr olds)	50% and 95% intake values from Table 6-40a	50% and 95% intake values from Table 6-40a
	Total lead intake ug/day (5-6yr olds)	50% and 95% intake values from Table 6-40a	50% and 95% intake values from Table 6-40a
	Total lead intake ug/day (6-7yr olds)	50% and 95% intake values from Table 6-40a	50% and 95% intake values from Table 6-40a
	Bioavailability	50%	50%
Drinking Water	Lead Conc. In Drinking Water (ug/L)	4	4
	Bioavailability	50%	50%
	Water consumption L/day (0-1yr olds)	0.20	0.20
	Water consumption L/day (1-2yr olds)	0.50	0.50
	Water consumption L/day (2-3yr olds)	0.52	0.52
	Water consumption L/day (3-4yr olds)	0.53	0.53
	Water consumption L/day (4-5yr olds)	0.55	0.55
	Water consumption L/day (5-6yr olds)	0.58	0.58
	Water consumption L/day (6-7yr olds)	0.59	0.59
Soil/Dust	Lead Conc. In Soil (mg/kg)	Geometric yard soil means from Tables 6-11a-h	Geometric yard soil means from Tables 6-11a-h
	Lead Conc. In Dust (mg/kg)	Geometric vacuum dust means from Tables 6-11a-h	Geometric vacuum dust means from Tables 6-11a-h
	Soil Dust Ingestion Weighting Factor (% soil)	45%	60%
	Bioavailability (soil)	30%	18%
	Bioavailability (dust)	30%	18%
Maternal-to-Newborn	Mother's blood lead at time of birth (ug/dL)	2.5	2.5
Computation Options	Iteration time step for numerical integration (hrs)	4	4
	Geometric Standard Deviation (unitless)	1.6	1.6

*Intake does not vary by age group

Table V-8. Community Mode IEUBK Input Parameters Used to Create Tables 6-56a-b
(Incremental Recreational Fish Exposure Scenario - CT and RME)

Exposure Medium	Model Specific Parameters	EPA-Default	Box-Model (40:30:30)
Air	Outdoor lead Conc.	0.10 ug/m^3	0.10 ug/m^3
	Indoor lead Conc. (% of outdoor)	30%	30%
	Time spent outdoors (0-1yr olds) hr/day	1	1
	Time spent outdoors (1-2yr olds) hr/day	2	2
	Time spent outdoors (2-3yr olds) hr/day	3	3
	Time spent outdoors (3-4yr olds) hr/day	4	4
	Time spent outdoors (4-5yr olds) hr/day	4	4
	Time spent outdoors (5-6yr olds) hr/day	4	4
	Time spent outdoors (6-7yr olds) hr/day	4	4
	Ventilation rate m^3/day (0-1 yr olds)	2	2
	Ventilation rate m^3/day (1-2 yr olds)	3	3
	Ventilation rate m^3/day (2-3 yr olds)	5	5
	Ventilation rate m^3/day (3-4 yr olds)	5	5
	Ventilation rate m^3/day (4-5 yr olds)	5	5
	Ventilation rate m^3/day (5-6 yr olds)	7	7
	Ventilation rate m^3/day (6-7 yr olds)	7	7
	Lung absorption %	32%	32%
Diet	Intake ug/day (0-1yr olds)	5.53	5.53
	Intake ug/day (1-2yr olds)	5.78	5.78
	Intake ug/day (2-3yr olds)	6.49	6.49
	Intake ug/day (3-4yr olds)	6.24	6.24
	Intake ug/day (4-5yr olds)	6.01	6.01
	Intake ug/day (5-6yr olds)	6.34	6.34
	Intake ug/day (6-7yr olds)	7.00	7.00
	Bioavailability	50%	50%
Alternate Sources*	Total lead intake ug/day (0-1yr olds)	50% and 95% intake values from Table 6-41a	50% and 95% intake values from Table 6-41a
	Total lead intake ug/day (1-2yr olds)	50% and 95% intake values from Table 6-41a	50% and 95% intake values from Table 6-41a
	Total lead intake ug/day (2-3yr olds)	50% and 95% intake values from Table 6-41a	50% and 95% intake values from Table 6-41a
	Total lead intake ug/day (3-4yr olds)	50% and 95% intake values from Table 6-41a	50% and 95% intake values from Table 6-41a
	Total lead intake ug/day (4-5yr olds)	50% and 95% intake values from Table 6-41a	50% and 95% intake values from Table 6-41a
	Total lead intake ug/day (5-6yr olds)	50% and 95% intake values from Table 6-41a	50% and 95% intake values from Table 6-41a
	Total lead intake ug/day (6-7yr olds)	50% and 95% intake values from Table 6-41a	50% and 95% intake values from Table 6-41a
	Bioavailability	50%	50%
Drinking Water	Lead Conc. In Drinking Water (ug/L)	4	4
	Bioavailability	50%	50%
	Water consumption L/day (0-1yr olds)	0.20	0.20
	Water consumption L/day (1-2yr olds)	0.50	0.50
	Water consumption L/day (2-3yr olds)	0.52	0.52
	Water consumption L/day (3-4yr olds)	0.53	0.53
	Water consumption L/day (4-5yr olds)	0.55	0.55
	Water consumption L/day (5-6yr olds)	0.58	0.58
	Water consumption L/day (6-7yr olds)	0.59	0.59
Soil/Dust	Lead Conc. In Soil (mg/kg)	Geometric yard soil means from Tables 6-11a-h	Geometric yard soil means from Tables 6-11a-h
	Lead Conc. In Dust (mg/kg)	Geometric vacuum dust means from Tables 6-11a-h	Geometric vacuum dust means from Tables 6-11a-h
	Soil Dust Ingestion Weighting Factor (% soil)	45%	60%
	Bioavailability (soil)	30%	18%
	Bioavailability (dust)	30%	18%
Maternal-to-Newborn	Mother's blood lead at time of birth (ug/dL)	2.5	2.5
Computation Options	Iteration time step for numerical integration (hrs)	4	4
	Geometric Standard Deviation (unitless)	1.6	1.6

*Intake does not vary by age group

Table V-9. Batch Mode IEUBK Input Parameters Used to Create Tables 6-61a-f and Tables 6-62a-f
(Current and Future Residential Exposure Scenario - Soil Remediation Concentrations)

Exposure Medium	Model Specific Parameters	EPA-Default	Box-Model (40:30:30)
Air	Outdoor lead Conc.	0.10 ug/m^3	0.10 ug/m^3
	Indoor lead Conc. (% of outdoor)	30%	30%
	Time spent outdoors (0-1yr olds) hr/day	1	1
	Time spent outdoors (1-2yr olds) hr/day	2	2
	Time spent outdoors (2-3yr olds) hr/day	3	3
	Time spent outdoors (3-4yr olds) hr/day	4	4
	Time spent outdoors (4-5yr olds) hr/day	4	4
	Time spent outdoors (5-6yr olds) hr/day	4	4
	Time spent outdoors (6-7yr olds) hr/day	4	4
	Ventilation rate m^3/day (0-1 yr olds)	2	2
	Ventilation rate m^3/day (1-2 yr olds)	3	3
	Ventilation rate m^3/day (2-3 yr olds)	5	5
	Ventilation rate m^3/day (3-4 yr olds)	5	5
	Ventilation rate m^3/day (4-5 yr olds)	5	5
	Ventilation rate m^3/day (5-6 yr olds)	7	7
	Ventilation rate m^3/day (6-7 yr olds)	7	7
	Lung absorption %	32%	32%
Diet	Intake ug/day (0-1yr olds)	5.53	5.53
	Intake ug/day (1-2yr olds)	5.78	5.78
	Intake ug/day (2-3yr olds)	6.49	6.49
	Intake ug/day (3-4yr olds)	6.24	6.24
	Intake ug/day (4-5yr olds)	6.01	6.01
	Intake ug/day (5-6yr olds)	6.34	6.34
	Intake ug/day (6-7yr olds)	7.00	7.00
	Bioavailability	50%	50%
Alternate Sources	Total lead intake ug/day (0-1yr olds)	0.00	0.00
	Total lead intake ug/day (1-2yr olds)	0.00	0.00
	Total lead intake ug/day (2-3yr olds)	0.00	0.00
	Total lead intake ug/day (3-4yr olds)	0.00	0.00
	Total lead intake ug/day (4-5yr olds)	0.00	0.00
	Total lead intake ug/day (5-6yr olds)	0.00	0.00
	Total lead intake ug/day (6-7yr olds)	0.00	0.00
	Bioavailability	0%	0%
Drinking Water	Lead Conc. In Drinking Water (ug/L)	Observation Specific*	Observation Specific*
	Bioavailability	50%	50%
	Water consumption L/day (0-1yr olds)	0.20	0.20
	Water consumption L/day (1-2yr olds)	0.50	0.50
	Water consumption L/day (2-3yr olds)	0.52	0.52
	Water consumption L/day (3-4yr olds)	0.53	0.53
	Water consumption L/day (4-5yr olds)	0.55	0.55
	Water consumption L/day (5-6yr olds)	0.58	0.58
	Water consumption L/day (6-7yr olds)	0.59	0.59
Soil/Dust	Lead Conc. In Soil/Dust (mg/kg)	Observation Specific[1]	Observation Specific[2]
	Soil Dust Ingestion Weighting Factor (% soil)	45%	60%
	Bioavailability (soil)	30%	18%
	Bioavailability (dust)	30%	18%
Maternal-to-Newborn	Mother's blood lead at time of birth (ug/dL)	2.5	2.5
Computation Options	Iteration time step for numerical integration (hrs)	4	4
	Geometric Standard Deviation (unitless)	1.6	1.6

*Observational specific data were used for all houses sampled whether or not a blood lead observation existed at that property or not; if not available then the geometric mean for the area was used; if yard soil data was missing, then the observation was deleted; purged and well water data used are summarized in Tables 6-11a-h.

[1]Observational specific data were used for all houses sampled whether or not a blood lead observation existed at that property or not; if yard soil data was missing, then the observation was deleted; if the yard soil concentration was greater than or equal to the soil cleanup level examined in each respective run (i.e., 2000, 1500, 1000, 800, 600, and 400 mg/kg) then that specific observation was given a post-remedial yard soil lead concentration of 100 mg/kg.

[2] Weighted average for each individual soil observation was calculated using 30% yard soil and 30% mean community soil; if yard soil data was missing, then the observation was deleted; all houses having a yard soil lead concentration were used for these analyses whether or not a blood lead observation existed at that property or not; if the yard soil concentration was greater than or equal to the soil cleanup level examined in each respective run (i.e., 2000, 1500, 1000, 800, 600, and 400 mg/kg) then that specific yard soil observation was given a post-remedial yard soil concentration of 100 mg/kg; each community mean was calculated using the post-remedial yard soil concentration (for each soil cleanup level examined, a new community mean was calculated for each area).

APPENDIX W

Comments and Responses on
Public Review Draft, July 2000

Comments and Responses on:

Public Review Draft
Human Health Risk Assessment for the
Coeur d'Alene Basin Extending from Harrison to
Mullan on the Coeur d'Alene River and Tributaries
Remedial Investigation/Feasibility Study
July 2000

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CONTENTS

Volume A
1.0 DOCUMENT ORGANIZATION
2.0 GENERAL RESPONSES TO COMMENTS
3.0 DR. PAUL MUSHAK'S RESPONSES TO COMMENTS
- 3.1 The Technical Review Workgroup for Lead (TRW), Citizens Against Rails-to-Trails (CART), and Local Governments
- 3.2 Public Comments
- 3.3 Mining Industry Comments

4.0 COMMENTS FROM THE TECHNICAL REVIEW WORKGROUP FOR LEAD (TRW)

Volume B
5.0 STATE/USEPA'S RESPONSES TO INDIVIDUAL COMMENTS
- 5.1 Mining Industry Comments
- 5.2 Local Governments
- 5.3 The Lands Council
- 5.4 Shoshone Natural Resource Coalition (SNRC)
- 5.5 Citizens Against Rails-to-Trails (CART)
- 5.6 Spokane Tribe
- 5.7 Private Individuals
- 5.8 Community Leaders for EPA Accountability Now (CLEAN)

SECTION 1.0
DOCUMENT ORGANIZATION

1.0 DOCUMENT ORGANIZATION

The following document contains comments and responses to the *Public Review Draft of the Human Health Risk Assessment for the Coeur d'Alene Basin Extending from Harrison to Mullan on the Coeur d'Alene River and Tributaries.* State/EPA received comments from several organizations and individuals. This document is a compilation of comments and response from various individuals and organizations. This section summarizes the contents and organization of this document, as follows:

- Section 2 contains a general response to comments that address global issues rather than specific comments.
- Section 3 contains Dr. Paul Mushak's responses to comments. Dr. Mushak provided an independent peer evaluation of the comments and is a consultant to EPA Region X.
- Section 4 contains the Technical Review Workgroup for Lead's (TRW) comments on the risk assessment. The TRW is an interoffice workgroup convened by the USEPA Office of Solid Waste and Emergency Response/Office of Emergency and Remedial Response (OSWER/OERR). Its goal is to support and promote consistent application of the best science in the field of lead risk assessment at contaminated sites nationwide.
- Section 5 contains responses to specific comments from each of the commentors. The comments were responded to by State/EPAs "Lead Contractor," Terragraphics (TG), or State/EPAs "Nonlead Contractor," URSCorp (URS). The comments and responses are arranged as follows:

  Each row contains a specific comment and it's response. The comments are in the left-hand column and the responses are in the right hand column. At the top of each row will be the Comment ID, the Comment Date, the commentor and his organization, the response due date, the respondent, and the status of the response (i.e., accepted, partially accepted, or not accepted). In general, an accepted response will trigger revisions to the next draft of the document; a partially accepted comment is one with which the State/EPA partially agrees, but may or may not be addressed in the revisions to text in the next draft of the document (the individual comment response notes whether text changes will be made); and a comment that is not accepted is one with which the State/EPA disagrees, no changes will be made to the document, and an explanation is provided in the responses.

SECTION 2.0
GENERAL RESPONSES TO COMMENTS

Human Health Risk Assessment for the Coeur d'Alene Basin Extending from Harrison to Mullan on the Coeur d'Alene River and Tributaries - Public Review Draft , July 2000

Response to Comments

The various written comments received and notes taken regarding discussions and questions during public meetings and presentations have been entered into a database as specific comments and concerns. Individual responses to each of the specific written comments received from the public and interested parties are included in the database. Those comments together with particular concerns expressed during meetings, conference calls, group discussions, and presentations have been summarized below in eleven general categories. A general response is provided following each comment category.

Also included as part of the response to comments are i) an independent peer evaluation of comments prepared by Dr. Paul Mushak, consultant to EPA Region X, and ii) the Technical Review Workgroup for Lead (TRW) evaluation of the HHRA.

Public Comment and General Response Categories

1 Comments Related to Population Demographics and Socio-economic Factors
- ***1a General Response Regarding Demographics and Socio-economic Factors***

2 Comments Related to Blood Lead and Exposure Surveys in the Basin
- ***2a General Response Regarding Blood Lead and Exposure Surveys***

3 Comments Related to the Use of Site-Specific Data
- ***3a General Response Regarding the Site-Specific Data Analysis***
- ***3b General Response Regarding Use of Blood Lead Data in Site-specific Analysis***
- ***3c General Response Regarding Use of Soil Lead Data in Site-specific Analysis***
- ***3d General Response Regarding Use of House Dust Data in Site-specific Analysis***

4 Comments Related to the Quantitative Site-specific Analysis of Blood and Dust Lead Levels
- ***4a General Response Regarding the Quantitative Site-specific Analysis***

5 Comments Related to Incremental Exposures
- ***5a General Response regarding the Characterization of Incremental Exposures***
- ***5b General Response regarding the Approach to Quantifying Incremental Exposures***
- ***5c General Response regarding the Combined Baseline and Incremental Exposures***
- ***5d General Response regarding Incremental Exposures associated with Rails-to-Trails***

6 Comments Related to Subsistence Exposures
- ***6a General Response regarding Subsistence Exposures***

7 Comments Related to Site-specific Exposure Parameters
- ***7a General Response regarding Site-specific Exposure Area Parameters***
- ***7b General Response regarding Use of Public Input in Developing Site-specific Exposure Parameters***

8 Comments Related to Exposure Pathways

8a General Response regarding Exposure Pathways
9 Comments Related to the Applicability of the IEUBK "Box" and EPA Default Models
9a General Response regarding the Applicability of the IEUBK "Box" and EPA Default Models
9b General Response regarding Bioavailability Estimates used in the IEUBK Model
9c General Response regarding the GSD used in the IEUBK Model
9d General Response regarding Observed and Predicted Blood Lead levels from the IEUBK Model
10 Comments Related to Interpretation and Discussion of Applicable Rules, Regulations and Guidance
10a General Response regarding Risk Assessment versus Risk Management Issues
10b General Response regarding Compliance with the NCP and Risk Assessment Guidance and Policy
10c General Response regarding Applicable or Relevant and Appropriate Requirements (ARARs)
10d General Response regarding Data Quality Objectives Guidance
11 Comments related to the Adult Exposures to Lead in Soil Model
11a General Response regarding Adult Exposures to Lead in Soil Model
11b General Response regarding Default Values

1 Comments Related to Population Demographics and Socio-economic Factors: Several comments addressed the socio-economic and demographic aspects of lead exposure. Comments included comparisons of the incidence of high blood lead levels to other populations with similar socio-economic characteristics, considerations in evaluating children with high blood lead levels, and how poverty-related factors might influence risk management decisions. Comments noted the incidence of poverty among children in the Basin was twice the State average, that socio-economic factors may influence many of the assumptions and parameters used in quantitative analysis of lead exposure, that the importance of the mining industry in the local economy was understated, that the potential for tourism was overstated, and suggested probable difficulties with developing new businesses during an extended Superfund project. Other comments urged that risk managers pay more attention (than believed typical for Superfund projects) to socio-economic issues in formulating risk-reduction strategies. Some comments indicate that the situation with respect to blood lead levels in the area is "relatively good" in consideration of the poverty levels, long history of mining activities, and comparison to other economically disadvantaged areas in the nation. Many comments addressed future risk reduction strategies questioning whether poverty initiatives can significantly reduce blood lead levels, that excess absorption is not isolated to disadvantaged families as not-so-poor children are also observed with high blood lead levels, and that health intervention efforts might be more effective than source cleanup in addressing lead poisoning. Other comments, conversely, suggest that those same poverty-related factors could make intervention efforts less effective, and that more source cleanup might be required.

1a General Response Regarding Demographics and Socio-economic Factors: *Comments received regarding demographic data generally did not dispute the data presented, but did provide suggestions regarding the interpretation of this information for risk assessment and eventual management activities. Demographic characteristics for the geographic portion of the Basin addressed in the HHRA are discussed in Section 3.1.2. The primary references were the 1980 and 1990 Census and updates, Idaho Department of Commerce publications, Idaho Kids Count: Profiles of Child Well-Being, and data provided by Public School Districts 391, 392 and 393. This area's economy has undergone significant changes in the last two decades that have had major impacts on local demographic factors that influence lead exposure. Total employment, according to census data in Shoshone County, for example, is down by 27% from 9126 jobs in 1980 to 6663 in 1996. Mining jobs decreased from 27% of total employment in 1980 (2465 jobs) to less than 10% (or 642 jobs) in 1996. These changes have been followed by out-migration of young families that has resulted in a continuing loss of young children. Overall population decreased by 30% from 1980 to 1990. The median age of the population changed from 27 years in 1970 to 39 years in 1998. Since 1990, the number of preschool children in the County has decreased by 12% compared to a 7.7% decrease among older children. Little new housing construction has occurred in the last two decades and median age of housing in several census tracts pre-dates World War II.*

Similar to many other lead contaminated sites, these and other socio-economic factors play an important role in the prevalence and degree of lead poisoning in the Basin. The HHRA notes that nearly one-third of the children in the Basin are growing up in poverty by federal government definitions. Poverty and lead poisoning interact in several ways. Children may have lowered nutritional status and live in poorer quality housing. Parents may experience more difficulties in managing the home and children and are less able to provide a stimulating and healthy home environment. Behavioral and home and child hygiene co-factors can lead to increased ingestion rates of soils and dusts. Yard soils and house dust can be more contaminated due to deteriorating lead paint, proximity to industrial sources, and lesser quality maintenance of the home, yard and local infrastructure. The age of housing in the Basin is problematic due to the frequent use of lead paint and accumulation of contaminated dusts throughout the last century. As a result, poor children ingest more soil and dust that has a higher lead content. These children tend to absorb more of the ingested lead than more nutritionally sound children, resulting in higher blood lead levels. In addition, poor children are then more vulnerable to adverse health effects resulting from their lower general health status, reduced access to quality health care, and early childhood educational opportunities.

Several comments urged that risk reduction efforts, that could address problems associated with poverty in the area, be considered in the development of risk management strategies. Many, if not most, of these lead source and socio-economic factors are common to the BHSS, located in the center of the Basin. Experience gathered at the BHSS and through the Lead Health Intervention Program in the Basin can be useful in assessing these children. These results suggest that following the cleanup efforts in the "Box", the children with high blood lead levels are young, are often exposed to sources outside the home environment, and have accompanying

socio-economic difficulties. In the 2000 Lead Health surveys, only six children (<3%) over 3 years of age showed blood lead levels greater than 10 ug/dl in the "Box". In the Basin, 7% or 8 children in this age range showed high levels. However, in children aged 1-3 years, 11% in the Box and 14% in the Basin showed excess absorption in 2000.

The greatest incidence of levels exceeding 10 ug/dl in 2000 was in the youngest 9-24 month aged group. This result suggests exposure to house dust levels in the principal activity areas of the home and home yard, or soil/dust exposures in locations away from the residence. Both sources seem to be indicated in these children's follow-up investigations. In most cases, these exposures are aggravated by socio-economic factors related to poverty. Overall, these findings suggest the most effective strategy to reduce risk for these young children should target reducing dust lead loading in the home and those socio-economic factors that aggravate lead absorption for these disadvantaged families. House dust studies in the Box and in the Basin show dust lead loading is related to several factors including overall community soil lead levels, home yard lead levels, interior paint condition and paint lead concentration, home hygiene practices, and exterior soil cover. Several of these factors are inter-related to socio-economic conditions that exacerbate lead exposure and absorption problems. Some comments urged risk managers consider poverty-related factors in developing cleanup plans. Potential risk reduction remedies that have been applied at other sites to reduce dust lead loadings and help to alleviate socio-economic co-factors that influence blood lead levels include:

- *Soil Abatement*
  - *Includes yard soil, community soil areas and specific fugitive sources removals to reduce direct exposure and a primary source of lead to house dust.*
- *Paint Abatement*
  - *Includes stabilizing interior and exterior lead based paint in homes of poor condition.*
- *Household Assistance/Select Cleaning*
  - *Includes providing vacuum cleaners loaners to homes without adequate vacuums, assistance in certain circumstances, and possible one-time cleaning.*
- *Screening*
  - *Includes voluntary blood lead screening services with provision for self-initiated testing at any time.*
- *Public Nursing*
  - *Includes follow-up services for all high blood lead level children, general advice and consultation and specialized assistance on a case-by-case basis.*
- *Access to other programs*
  - *Includes referrals to other social, housing and medical assistance programs that offer complimentary services for needy families.*
- *Community Development Poverty Initiatives*
  - *Includes assistance to and support of community development initiative to attract new industry and business to the Basin.*

-Job Training

- Includes assistance to job training programs to ensure local hiring for cleanup activities and enhance employment opportunities for Basin residents.

- Early Periodic Screening and Diagnostic Testing(EPSDT)

- Includes addition of blood lead screening and lead health related testing and diagnostic services to Medicaid programs among local service providers.

-Medicaid

- Includes assistance in securing appropriate Medicaid benefits for eligible young families throughout the Basin.

-Young Family Programs

- Includes development of educational programs for young parents alerting them to available health, educational, housing, and income resources.

-Preferential Hiring

-Includes adoption of appropriate rules and regulations to ensure local preference in hiring for any publicly funded cleanup activities.

-Developing clean play areas

- Includes cleanup of existing, and developing new recreational areas that will be accessed by young children to residential cleanup criteria.

-Water Subsidies

- Includes community and individual subsidies to provide water for dust control and maintenance of vegetative cover.

-Community Greening

- Includes grants and subsidies to promote vegetative cover throughout local communities.

-Housing Initiatives

- Includes support of paint abatement programs, assistance with remodel and cleanup activities that reduce lead health risk through home/yard improvement.

-City/County Public Works Assistance

- Includes assistance to communities that operate and maintain infrastructure critical to dust and storm water runoff control.

-Supplemental Water Source Development

- Includes assisting communities to develop supplemental water supplies to ensure dust control and vegetative barrier maintenance during drought periods.

-Curb/Gutter Storm water Infrastructure

- Includes curb and gutter installations to reduce dust generation, right-of-way contamination, and enhance dust control efforts.

- Includes supplementing existing storm water collection and containment systems to enhance dust and sediment control in communities.

- Includes assistance with facilities and operation and maintenance of snow removal activities to reduce aggravation of contaminated dust and sediment problems in communities.

-Parks/Playgrounds/Pools

- Includes development of alternate play areas and water-based recreation facilities to encourage families with young children to recreate in clean areas.

-Community Resource Centers

- Includes development of community resource centers for young parents that can dispense social and health services in an encouraging and constructive atmosphere.

2 Comments Related to Blood Lead and Exposure Surveys in the Basin: Several comments addressed the low turnout of children in the exposure and blood lead surveys conducted in the last four years. These comments indicate that the results of these surveys may misrepresent the extent and degree of lead intoxication in the Basin. Many of those comments contend that socio-economic biases may be inherent in the self-selected population presented for blood lead sampling. However, comments differed in opinions and conclusions with regard to the potential bias. Some reviewers believe solicitation incentive programs (paying $20-$40 per child for blood) result in a disproportionate number of poor individuals participating. These arguments suggest that socio-economic factors are well-correlated with blood lead levels, mask the true effects of contaminated media, and exaggerate the incidence of lead poisoning in the community. Other comments suggest that those that volunteer for blood lead testing are more informed regarding lead poisoning issues, more inclined to benefit from the intervention services, more attentive to health issues and, as a result, show lower blood lead levels than those that were not tested. Some comments also point out that socio-economic conditions in the area over the past two decades have led to age biases in the population that influence the degree of lead intoxication noted in the health surveys. These comments note that there has been a 12% decrease in young children in the past ten years and this age-group is greatly under-represented in blood lead surveys.

2a General Response Regarding Blood Lead and Exposure Surveys: *Several comments speculated that poverty-related factors influence and, possibly, distort the incidence of high blood lead levels in the Basin. Available data indicate that about one-in-four children under two years of age have blood lead levels of 10 ug/dl or greater, and the age adjusted incidence of excess blood lead levels is 16.2% for 1-6 year-old children. This incidence of high blood lead levels is a health concern for these children. There are divergent opinions as to how well the health surveys represent the non-participants and whether comparisons to other national and State populations are appropriate. Comparison of blood lead data for the Basin to other sites and national or State-wide surveys, for the purpose of determining whether these findings are "relatively good or bad", is problematic. Such large data sets (i.e., National Health and Nutrition Examination Survey (NHANES)), for various technical reasons, cannot be used to compare and draw conclusions about the relative degree of health hazard existing for children in the Basin communities. Scientific designs of the NHANES surveys, are constructed in a way that does not allow simple comparisons with results of blood lead distributions for a single community. NHANES data provide a current snapshot for numerous national subsets or strata,*

that may not be appropriate for any single community. An explicit warning on technical grounds against making such comparison is in the Executive Summary of ATSDR's 1988 report to Congress on childhood lead poisoning in America (ATSDR 1988). Additionally, the purpose and design of the Basin surveys were conducted in a manner that does not match the organization of the various demographic and socioeconomic strata in the NHANES III survey reports (e.g., race/ethnicity, income, housing age).

With regard to the Basin-wide survey, selection bias may have occurred related to individual family decisions to participate. One argument suggests that the incidence of lead poisoning is likely greater among non-participants, as families that did participate are more attentive to lead poisoning and have benefitted from the local health department's efforts to assist parents in reducing exposures. A counter argument suggests that paying each child $40 as an incentive in the 1999 survey favored low-income participation. Because potentially high exposures are associated with poverty-related factors, higher than average blood lead concentrations would be expected among the participants. The HHRA did not draw a conclusion relative to these arguments as there are not sufficient data to test either hypothesis. These issues are discussed in Sections 6.2.2 and 7.4.1, 8.8, and 8.11.2 and reflect most of the comments offered by reviewers.

Several comments requested additional characteristics regarding the number of children contained in the blood lead database for the Basin. A total of 524 blood lead observations were compiled in four surveys from 1996 to 1999. There were 424 individual children from 247 households. Eighty-one (81) children from 57 homes were tested more than once. Sixty-five (65) of those children were tested twice, 13 were tested three times and 3 were tested in each of the four years. Of those children tested more than once, 11 had levels greater than 10 ug/dl and received intervention services from the local public health program. Seven (7) of these children had lower blood lead levels in subsequent testing, 1 had the same level, and 3 had higher levels. The children tested more than once tended to have lower than average levels for children in their age group on the first test and similar levels on subsequent testing. Of the 81 children tested more than once, 21 had higher than average blood lead levels for their age group, 51 had lower, 9 had average levels. These results would indicate that some observations used in the analysis were lower than might be obtained in a random sampling of the population. It is estimated that there are between 1000 and 1100 children from 9 months to 9 years of age in the Basin area. In 1999, 272 or slightly more than 25% of these children were tested. In other years less than 20% of eligible children participated. The Bunker Hill Superfund Site participation rates have been estimated from 51% to 58% of the population annually over the last decade. Table 1 summarizes the participation rates.

Comments have suggested that the socio-economic co-factors describing the blood lead population be compared to that of the overall population. There is not, however, a complete socio-economic database for these children that would allow for such a comparison of risk co-factors. However, the environmental source characteristics can be compared, as shown in Table 2. These results indicate that the source variables for the blood lead population are similar to

that of the general population. Table 2 also shows that there is disparity with respect to the number of paired observations by geographic sub-area.

3 Comments Related to the Use of Site-Specific Data: There were several comments regarding the use and analysis of site-specific data in the HHRA. These comments addressed the definition and characterization of site-specific data and analysis, whether ingestion and contact rates appropriate to the Coeur d'Alene Basin were employed in the HHRA, whether appropriate dust sampling techniques were employed, and critiques regarding specific relationships and model parameters. Some comments were critical of the combination of soil contamination data from different surveys, the sieve size used in processing soil and dust samples, and the relationship between paint, soil, dust and blood lead levels. Comments were also received noting that confidentiality requirements necessary to protect individuals participating in public health service programs preclude independent evaluation of the findings of the HHRA with respect to site-specific analysis and assessment of individual behaviors and lifestyles contributing to high blood lead levels.

3a General Response Regarding the Site-Specific Data Analysis: *Site-specific analysis, as defined for this HHRA, involves the collection of actual blood lead data from the resident population and relating those observations to measured concentrations in environmental media. Existing blood lead data are summarized in Section 6.2 and site-specific quantitative analysis is presented in Section 6.4. Site-specific analysis of risk for this particular HHRA was conducted using the data available from recent surveys and investigations supplemented by additional sampling efforts conducted in November of 1999. This analysis was undertaken in response to Potentially Responsible Parties (PRP), State and local government, and public requests that actual blood lead levels and environmental data from the Basin be considered in assessing risk and identifying risk reduction strategies. The PRPs and local public interest groups also requested that specific evaluation of lead paint data previously obtained, but not analyzed in the 1996 Exposure Study, be recovered and used in these evaluations. The site-specific analysis helps to establish that percentage of the population that is actually experiencing lead poisoning and characterize the direct link between lead in blood and the various sources. This is generally accomplished by conducting well controlled investigations that collect both blood lead and environmental source data and relating those through statistical techniques. In this case, only the 1996 Basin Exposure Study was designed to support such analysis. Although a large residential environmental sample and exposure database was obtained in 1996, few children provided blood lead samples. Based on the results of the 1996 study, considerably more children were solicited in subsequent blood lead surveys and a strategy was developed of combining these results and conducting supplemental environmental sampling to complete a paired blood lead/environmental exposure data set to support site-specific analysis. This approach has been used at other Superfund sites and is consistent with EPA guidance. EPA guidance does require that any site-specific analysis be based on compelling scientific evidence, collected in controlled investigations that are representative of the population of concern, the contaminated media, and the routes and pathways of lead exposure that are, or could be, occurring in the future. A non-confidential form of the paired blood lead and environmental exposure data set will be included*

as an Appendix to the final document. All individual identifiers have been removed and categorical values developed to replace actual concentrations to protect the confidentiality of the data and privacy of families supplying information.

3b General Response Regarding Use of Blood Lead Data in Site-specific Analysis: *Many of the comments received suggest that the blood lead data are not representative of that portion of the population that did not participate in the blood lead surveys. The representativeness of the blood lead data set is discussed in General Comment # 2a The site-specific dose-response analysis conducted in the HHRA are reflective of those paired observations of blood lead and environmental exposure in the assembled database. As a result, the findings and conclusions are applicable to the population studied. The environmental exposures in the site-specific database are believed to be representative of the typical conditions throughout the Basin. There has been speculation in various comments that the blood lead information may be biased. Arguments have been presented for both high and low biases. The effect of these biases, if any, is unknown.*

3c General Response Regarding Use of Soil Lead Data in Site-specific Analysis: *With respect to environmental data, the effects of combining soil metal concentration results from different surveys were assessed in Appendix N to the HHRA. This analysis concluded that there were not substantive differences in surface soil sampling results from different surveys. Approximately 2400 homes in the Basin were estimated in the 1996 survey census to have potential residential soil contamination associated with mineral industry releases. Approximately, 40 % or 1020 of these homes have been sampled. Comments were received objecting to the use of surface soil contamination data in exposure characterization. Some comments indicated that sub-surface results should be used, as children are known to dig in play activities. Sub-surface soils are characterized in the HHRA in Section 6.6. These concentrations are used in assessing occupational risk to workers that might be involved in excavation activities. Surface soil concentrations are used in all residential and recreational exposure estimates as these are the soils most likely to be encountered in these activities and the soils most susceptible to transport and migration. This approach is consistent with current guidance and is routinely applied in risk assessments throughout the country. Risk managers are cautioned to remember that children do dig during play activities and to consider remedies protective of this potential pathway in developing risk reduction strategies.*

There are potential differences in soil lead levels between the Basin results and those from other sites on the basis of the sieve size used to process soil samples. Comments were received criticizing the HHRA for using soil samples sieved to minus 175 micron. Some comments favored the more coarse 250 micron sieve recommended in recent EPA guidance and others favored a finer sieve used for sediment characterization by the U.S. Geological Survey (USGS) in ecological/transport evaluations. The 175 micron mesh sieve technique was adopted in 1974 for the original lead health studies conducted in the area and has been used for all residential soil samples collected in the Basin RI/FS and all previous health and exposure studies. The procedure was developed to reflect the range of soil particle size most likely to adhere to children's hands and be involved in hand-to-mouth activities. Subsequent research has continued

to show that this size-range is applicable. The selection of this standard pre-dates either recommendation from federal agencies, and the State Department of Health and Welfare has elected to maintain consistent soil and dust measurement techniques throughout the course of these investigations. The EPA has concurred in that determination. No data have been collected to assess the difference, if any, among these size fractions for residential soils or house dust. Evidence and experience from other sites would suggest that smaller particle size fractions could exhibit higher concentrations of lead and other metals. If this were the case, lead concentration in soil and dust measured in the Basin might be lower using the sieve size suggested by the EPA or higher using that suggested by USGS. Assuming any concentration effect due to sieving is proportional, the use of lower value (as suggested with EPA's larger sieve size) could result in an increased dose response coefficient in the site-specific analysis. That is, the per unit effect of soil or dust lead concentration on blood lead levels would be greater. This would be interpreted as indicating higher bioavailability of soil and dust or lesser intake is occurring in the population. Using higher concentrations that might result from smaller sieve sizes would produce the opposite effect and conclusions. Provided the effect is proportional, it is of little consequence in the empirical site-specific analysis, as the coefficients relating blood lead to environmental variables would adjust accordingly. In relation to applications of the IEUBK model and comparisons to other sites, differences could be significant. It is important that risk managers use equivalent procedures in developing and designing risk reduction strategies involving measurement or evaluation of soil and dust lead concentrations.

An additional comment questioned combining flood plain soil and sediment data in the Lower Basin, suggesting that soil and sediment could be independent data sets and do not have sufficient statistical similarity to justify combining. For some sites, upland soil and beach sediment data may be statistically different for some contaminants. This may be an important issue in defining the nature and extent of contamination. However, due to the assumptions used in calculating risk, any differences in sample means is accounted for in the estimation of exposure point concentrations. The soil and sediment data were appropriately combined for the Lower Basin neighborhood and public receptors for several reasons. The "upland" areas had all been impacted by previous flood events, and experienced a mixing of soil and sediment materials. For this reason, this material was identified as "flood plain soil/sediment" and refers to materials within the approximately 1-mile wide flood plain area. The "upland" areas and the "beach" areas of the Lower Basin CUAs are in close proximity to one another. Lastly, a receptor is presumed to spend an equal amount of time in upland areas as in beach areas. It is also assumed that receptors will have an equal probability of visiting one CUA in the Lower Basin as another. As a result, the data were combined and an average concentration representing the aggregate exposure in the Lower Basin is appropriate, as specified by EPA risk assessment guidance (1989, 1992, 1996).

3d General Response Regarding Use of House Dust Data in Site-specific Analysis: *Comments were received that the dust and lead loading measurement methodology employed in the Basin was unconventional and may not represent actual exposures to children in the area. Dust lead exposures were measured by two independent techniques. Samples were collected from home*

vacuum cleaners, if these were available and had not been used outside or in the family car, and by entryway mats. The former technique measures lead concentration in the minus 175 micron fraction of vacuum cleaner dust. This method has also been continuously monitored in the BHSS since 1974 and has been a significant correlate of both blood lead and soil lead levels at the Superfund site. The second technique measures both dust lead concentration from the same size fraction, and the accumulation rate of both dust and lead on the mat. The accumulation rate of lead, or lead loading rate on these mats was the single strongest environmental source correlate with blood lead in the site-specific analysis (r=0 .63). Blood lead is also significantly correlated with interior and exterior paint lead (r=0.341 and 0.407, respectively), yard soil lead concentration (r=0.158), and community-wide soil lead concentration (r=0.116).

Divergent opinions were offered in comments as to whether these were "poor, good or significant" correlations. The significance criteria used was the p=0.05 level. Whether the correlation coefficients are "poor or good" is a matter of opinion. A low correlation coefficient does not necessarily imply an unimportant or absent relationship. The variables may be directly related in a non-linear manner or dependent on the effect of other variables. Reviewers familiar with these types of studies have concluded that these correlations are typical and provide useful information in assessing such situations. The relatively strong and consistent correlations among these variables suggest that dust lead loading is an important factor in the environmental exposure and lead absorption situation in the Basin.

Comments were received that suggested that soil should not have been used as a surrogate for house dust concentration data for metals other than lead. These comments suggested this was inconsistent with the lead methodology and that there were sufficient data to characterize non-lead metals for house dust data in the risk assessment equations. The HHRA concluded that insufficient data were available for <u>each geographic subregion</u> because paired soil-dust data were not available for every home. The primary reason the data were not used in the risk and hazard calculations was because the uncertainty in predicting dust concentrations from soil concentrations was considered more problematic than the uncertainties of using the soil data as a surrogate. Paired soil and dust data for lead were available for over 800 homes compared to 84 homes for non-lead. As a result, actual dust data were used for lead and the soil-dust relationship for lead was reasonably well characterized and compared to results at other sites. Similar data from other sites is not available for non-lead contaminants. The HHRA acknowledged the uncertainties associated with using yard soil as a surrogate for house dust concentrations and indicated whether exposure point concentrations for various non-lead metals might be over or under estimated (see discussion on pages 7-14 through 7-16 of the HHRA). These concerns do not apply to lead risk assessment as observed dust lead concentrations were utilized.

4 Comments Related to the Quantitative Site-specific Analysis of Blood and Dust Lead Levels: There were several comments regarding the site-specific quantitative analysis relating blood lead levels to environmental variables and exposure factors. Comments disagreed

regarding what constitutes "good", "high" or "significant" correlations or multi-variate regression relationships. Comments also failed to agree on the interpretation of the results. Of particular concern was the paint-soil-dust-blood lead relationship. Most comments agreed that the strongest relationship with blood lead was dust lead loading rate as indicated by the entryway mat. Because dust lead loading is, in turn, most highly correlated with yard soil lead concentrations, some reviewers see this as evidence that soils are a primary contributor to dust lead and blood lead levels. Others speculate that entryway mat lead loading reflects lead paint sources on porches, doorways and exterior surfaces. Others indicate that the influence of dust loading is indicative of home hygiene practices that are, in turn, reflective of lower socio-economic status and associated personal and family hygiene practices. Other reviewers suggest that entryway mat lead levels are not indicative of lead levels within the home. Several reviewers pointed out that the robustness of paint lead blood lead correlation is decreased or eliminated by inclusion of community mean soil lead levels in step-wise regression analysis. Some comments interpret this to mean that older communities have higher soil lead levels due to the long mineral industry history and, coincidentally, more lead paint due to the age of the housing. As a result, the significance of paint lead diminishes after accounting for community-wide soil lead. Others conclude that the higher community soil lead levels are related to deterioration of the exterior paint. Some comments suggest deleting homes and blood lead observations with known or suspected paint lead exposures from the analysis.

4a General Response Regarding the Quantitative Site-specific Analysis: *These relationships were assessed using multi-variate analysis in Section 6.4.2 of the HHRA. With respect to blood lead levels, backward selection step-wise regression analysis indicated that dust lead loading rate alone explained nearly 40% of the variance in the dependent variable. Other environmental variables were significant in combination with dust lead loading rate. Those variables were yard soil lead levels, median exterior paint XRF reading, and interior paint condition. Together with age of the child, these variables explain 60% of the variance in blood lead levels. It is well established in the lead health literature that there is an inherent variance in blood lead response among individuals in a population. Considering that this regression model does not address this inherent variance, accounting for 60% of the variation in observed blood lead levels must be considered a strong relationship. The interpretation of these results in the HHRA was that contaminated soils, house dust, and lead based paint are all related to excess absorption. Overall this suggests complex exposure pathways, with blood lead levels most related to dust lead loading in the home, followed by independent effects of yard soil lead, interior paint lead condition, and exterior paint lead content. The dust lead pathway is most influenced by outdoor soils, augmented by paint contributions in older homes, especially those in poor condition.*

Differing interpretations were offered by reviewers. Few comments addressed the blood lead environmental exposure model. Most comments addressed the model explaining dust lead loading rate. Multi-variate regression analysis using dust lead loading rate as the dependent variable indicated four significant variables including yard soil lead concentration, interior paint condition, the maximum interior lead paint XRF reading in the home, and the mean soil concentration in the local community. The dust lead loading rate is calculated by multiplying the

total dust accumulation rate by the lead content of that dust. Results of step-wise regression analysis, also shown in Section 6.4.2 show that dust lead content on these mats is most related to yard soil lead concentration followed by interior paint lead condition. The next most significant variable by backwards elimination is the community mean soil lead level at the p=0.0001 level. No other variables are significant at the p=0.1 level in the presence of these factors. If community mean soil concentration is eliminated from the selection, the maximum interior paint lead XRF reading and the exterior median paint lead XRF reading are significant at the p=0.02 and 0.03 level, respectively. Vacuum bag lead concentration is related to the mat lead concentration (p=0.001), yard soil concentration (p=0.01), and maximum interior paint lead XRF reading (p=0.03). Vacuum bag lead content typically exhibits about a 30% to 40% lower concentration than mat lead content. Some comments speculate that the elevated mat lead concentrations are due to paint lead contributions from the entryway areas and are not reflective of dust lead exposures to children. The significance of the paint lead variables in the mat dust lead concentration model is suggestive of this effect. However, the non-significance of these variables in the presence of the mean community soil lead concentration, could imply that these effects are related to community-wide lead levels that could, in turn, be a surrogate for the age of the community. Those towns showing the highest soil and dust lead levels also have the oldest housing stock, highest lead paint levels, and longest history of industrial pollution.

Suggestions were made regarding the inclusion of socio-economic variables and development of lead-paint condition interactive factors or cross products in these analyses. However, as was noted for the proposed socio-economic characterization of the blood lead data set, insufficient data are available to perform these adjustments. Suggestions were also made to perform separate analysis of homes with and without paint hazards. This analysis would also be difficult as most homes, other than trailer homes, have lead paint. The primary indicator of paint condition (peeling/chipping/chalking paint) has been shown in the parent 1996 Basin Exposure Study to be highly correlated with home hygiene and socio-economic status. As a result, it is not clear whether the significance of this variable is reflective of the paint source of lead, socio-economic status, personal and family behavior, home hygiene practices, or dust loading.

In summary, several comments have offered additional speculation regarding the interpretation of site-specific analysis. Most comments agree that house dust lead loading is strongly correlated with blood lead levels. Comments do not agree with respect to the source of lead on these mats. Some believe the evidence is supportive of paint lead sources, others believe outdoor soils from both the yard and community are primary sources. The HHRA concluded that both sources are likely significant, but there is uncertainty regarding paint sources due to the relationship between paint condition and socio-economic status that cannot be unraveled with these data. That conclusion remains unchanged. These findings are consistent with the follow-up reports from public health nurses investigating children with high blood lead levels and results from other sites including the nearby BHSS. As a result, risk managers should consider both sources potentially important to lead poisoning in the Basin.

5 Comments Related to Incremental Exposures: A number of comments were received regarding incremental exposures. Some of these comments noted that the presentation of incremental exposures was confusing and that some of the descriptive terminology was ambiguous. Many of the comments were contradictory. Some comments indicated that it was inappropriate to combine various incremental pathways, as it is unlikely that any individual would engage in all these behaviors. Similar comments objected to double counting of exposures or the failure to discount residential exposure from the typical baseline intake for children recreating outside the home environment. Other comments indicated that this approach resulted in multiplying various conservative assumptions and safety factors, resulting in unrealistic scenarios and over-stating risk. Others, conversely, noted that the document fails to indicate that all of these pathways could be active for some individuals. Similar comments objected to assuming average or typical baseline residential exposures for those children that might engage in the recreational activities. These comments argue that the children with high intake rates at home are more likely to have high intake rates during recreational activities, that risks are understated, and that no margin of safety is provided in the analysis for these children. Comments were also received indicating that mixed age bands were applied in the analysis in that some young children are unlikely to engage in certain activities. Several comments objected to the averaging periods employed for the various behaviors and the allocation of those intakes over an annual period in the application of the IEUBK model. Comments objected to applying RME parameters to lead analysis indicating that the IEUBK analysis inherently accounts for high exposure rates. Others objected to the use of the IEUBK model to assess periodic or episodic behaviors, citing EPA policy statements. These comments indicated that the averaging times used overstate risk, and that longer averaging periods should be utilized. EPA's Technical Review Workgroup for lead (TRW), that reviewed the document, conversely indicated that the model is applicable, that shorter averaging periods should be used, and the document likely understates risk. Some comments were received regarding the inappropriateness of certain assumptions used in the intake estimates. Comments objected to contact rates at waste rock piles, as these areas have little exposed fines, and the use of disturbed water samples, those with significant levels of suspended sediment, to represent beach activities. Several comments were received regarding the Rails-to-Trails conversion of the existing railroad right-of-way through the Basin. Comments were submitted that the HHRA failed to consider potential exposures associated with the trail, that the trail would invite people into contaminated areas, that the risk management plan adopted for the trail in earlier EE/CA actions was insufficient, that contamination in areas outside those geographic areas considered in the HHRA were ignored, that demographics and private property throughout the Basin were not adequately addressed in the HHRA, and that wastes in the Lower Basin were miscategorized as to source and description.

5a General Response regarding the Characterization of Incremental Exposures: *Incremental exposures are introduced in Section 6.5.3 of the HHRA as estimated lead intake rates. Incremental lead intake rates refer to the amount of lead taken into the body during activities in which only certain members of the population engage. These individuals either consume more soil, dust, water, or food than the general population, or those media have higher lead content. Incremental intake rates were developed both for the typical (Central Tendency (CT)) and*

reasonable maximum exposure (RME) members of the population. Estimating the intake rates is a relatively straight-forward procedure utilizing exposure factors developed elsewhere in the document. Generally, these factors are linear and intake estimates are proportional to exposure point concentrations, contact times, and exposure frequencies. Should risk managers disagree with the underlying assumptions or wish to consider alternative factors, the incremental intake rates can be adjusted accordingly. This option is discussed in more detail in General Response to Comment # 7b.

Estimation of potential blood lead increments and the increased risk of exceeding critical toxicity levels associated with these intakes, however, presents additional challenges. Incremental exposures should be evaluated as a cumulative effect added to exposures received in the home environment. This is accomplished by adding the incremental intake of lead to the baseline (or residential) intake. There is a significant question as to characterizing the baseline exposure to which the increments should be added. Should the baseline reflect the typical child residing in the Basin, a child from outside the Basin, or the child most at risk within the Basin? Should this analysis be performed for current conditions, or for projected post-remedial lead levels, following cleanup in the residential areas? There is a question as to whether the baseline should be discounted to avoid double-counting intake during incremental activities (i.e., the child is not at home when at the beach). However, most children do not engage in these behaviors and discounting the population baseline would underestimate their risk. Other questions are related to combining incremental behaviors. All children will not engage in all incremental activities, but some children will engage in one or more activity, and a few might engage in all the activities considered. Which combinations of potential incremental intakes should be assessed? Other concerns are related to application of the IEUBK model to incremental exposures. In the IEUBK model, mean blood lead estimates are developed for typical children assuming a uniform annual exposure. RME characterizations are estimated by applying a distribution of responses around the mean. Because various members of the population will have different exposures, depending on which incremental behaviors they engage in, estimates of the number of children to exceed critical toxicity levels are difficult to interpret.

There are concerns related to averaging periods for the incremental exposures. Should the incremental intake be averaged over the period of exposure, the season, or the year? How should these average intakes be input to the IEUBK model? The IEUBK is not designed to address episodic behaviors, but near equilibrium responses are achieved in a few months and the model has been successfully used to assess seasonal inputs. The EPA's Technical Review Workgroup (TRW) recommends using 2 to 3 month averaging periods for these applications. The TRW concludes that the exposure duration is sufficient to include in IEUBK analysis, but believes the risk may be understated, by about 35%, as the exposure should be averaged over the seasonal exposure duration rather than the 365 day year. Additionally, the recreational ingestion rates are applied only for those hours in the day during which the activity occurs, although the rates used could be considered event related. For example, the 300 mg/day RME soil ingestion rate for upland parks is applied for 7 hours/day, or 50% of waking hours, resulting in 150 mg of soil ingested while recreating. An alternative interpretation of the ingestion rate would be 300

mg/day per even , resulting in doubling the recreational soil intake. As a result, risk managers may want to consider risk potentially underestimated for incremental behaviors.

Finally, the concept of RME is problematic with these exposures. Extreme responses in the population can be estimated by applying an appropriate GSD to the mean blood lead estimate from the IEUBK model, although this is difficult to interpret as noted. This technique requires that typical, or CT intake rates be input to the model for both the baseline and incremental exposure. The extreme responses estimated by applying the GSD reflect the biokinetic variation in the population and the variation inherent in the typical exposure. However, there are environmental extremes in the potential incremental exposures to consider in addition to the bio-kinetic response and typical baseline exposure factors. Some children, for example, may always play at the most contaminated beaches, rather than at the typical or average concentration. The RME scenarios used in the IEUBK reflect CT ingestion rates for both the baseline and incremental exposure applied at 95th percentile contact concentration.

5b General Response regarding the Approach to Quantifying Incremental Exposures:
Comments received addressing several of these questions were variously characterized as overestimating or underestimating risk, depending on the understanding and perspective of the reviewer. The IEUBK model, if appropriately applied, is capable of estimating risk and providing useful information to risk managers for nearly all of the situations noted above. However, the application of the model must be precisely described and interpretation of the results limited to the particular situation evaluated. For example, assessing the incremental risk for a child visiting a contaminated recreational area on a seasonal basis requires identifying the baseline situation from which the child originates, the estimated incremental intake from the recreational activity, and the estimated intake from any other incremental behaviors. Determining these intakes requires specifying where the children live, and where and for how long they engage in these activities. The intake estimates must then be reconciled to a common exposure period, or averaged over either the seasonal exposure period of the incremental activity or the annual baseline exposure period. The IEUBK model is then run for both the baseline situation and the combined baseline and incremental behavior. This can be accomplished either by developing a time-weighted intake average incorporating both the baseline and incremental behaviors, or by inputting the incremental intake as an additional source. The results can then be compared and the blood lead increment interpreted as the increase predicted for the typical child exposed to this particular situation. The difference in the predicted percentage of children to exceed 10 ug/dl would be the interpreted as the increased probability that an individual child from that baseline situation would have an excessive blood lead level as a result of the incremental behavior(s).

These results would apply only to those children from that particular residential area engaging in those particular activities, or similar communities and activities. However, there are hundreds of possible combinations of baseline and incremental exposure situations that could be of interest in the Basin. Assessing each situation individually would require a substantial effort to accomplish and would produce a large amount of results to interpret, much of which would be

superfluous to risk management considerations and decision-making. As a result, the HHRA assessed a limited number of scenarios under current baseline conditions and introduced a methodology by which other scenarios could be evaluated during risk management activities.

In the HHRA, incremental lead intake rates were determined for a variety of potential activities that could significantly add to the amount of lead taken into the body. These rates were developed on an activity specific basis for both a typical (CT) or a worst case (RME) estimate. Initial soil, dust, food, and water ingestion and inhalation values used for these intake calculations correspond to those developed for the non-lead risk assessment. These intake rates are compared to baseline intake in Section 6.5. Risk managers can assess the incremental and total intake, and compare the relative increase. In Section 6.6 those intakes are input to the community mode IEUBK model as an additional source. The community mode was selected because the mean blood lead and percent to exceed estimates represent the most likely value for the typical child in each community. This estimate is most representative of the overall exposure situation for each community, as the batch mode data set is limited to those individuals for which blood lead levels are available. The community mode also facilitates the estimate of the percent of children to exceed specified toxicity levels. The resulting mean blood lead estimate from this procedure should be interpreted as the estimated blood lead level for the typical child from each community that engaged in the selected incremental activity. The percent to exceed estimate should be interpreted as the likelihood that the typical child will exhibit an unacceptably high blood lead level. These results are compared to the baseline estimates without the incremental exposure and the difference can be attributed to the incremental activity.

5c General Response regarding the Combined Baseline and Incremental Exposures:
Reviewers and those using these estimates must consider several of the concerns noted above. The baseline estimates included in the combined runs are not discounted for the time spent in the incremental behavior. This leads to an overestimation of risk, albeit small for the current baseline situation. For a child, about 5% of the baseline intake would be double counted at typical soil concentrations. However, as noted below, accounting for this reduction in baseline in the post-remedial environment will be important. In the HHRA, the incremental intakes are averaged over the year to correspond with the exposure period inherent in the residential baseline exposure estimate. This results in a probable underestimation of risk, in that blood lead levels reflect the annual average, whereas it is likely that blood lead levels will be higher during the exposure season.

It is also important to note that the estimated blood lead increment is dependent on the baseline blood lead level. As a result, because these estimates are developed at the current baseline that is unacceptably high, the same incremental intake applied to an acceptable, lower post-remedial blood lead level will result in a larger blood lead increment. This is because the dose-response relationship is non-linear and the intake at lower baseline blood lead levels will result in a greater response. The probability of exceeding the 10 ug/dl toxicity threshold then depends on both the baseline blood lead level and the increment. At acceptable post-remediation blood lead levels, the accounting for time away from the baseline for incremental activities becomes critical.

If there is no margin of safety incorporated in the residential baseline exposure, then the incremental intakes must not exceed the reduction in baseline intake due to the time away from home engaging in the incremental behavior.

For this reason, no post-remedial examples of potential cleanup criteria were provided for risk assessors as was accomplished in Section 6.7 for the residential or baseline situation. The examples provided should be reviewed as indicators of whether these activities result in substantial or significant increases in intake or blood lead estimates. However, using these results to effect remedial strategies must be done with caution. Potential cleanup estimates would not be relevant until risk managers suggest or provide criteria for residential exposure reductions. Should risk managers select a minimal residential cleanup criteria, (i.e., the highest allowable intake rates and corresponding soil and dust lead concentrations), then the corresponding criteria for incremental exposures would be that which results in offsetting the post-remedial residential intake during the time away from home engaging in the incremental activity. This result could be calculated without employing the IEUBK, requiring only a net balance of lead intake be achieved. These calculations could also include other considerations, such as local input regarding adjustment of exposure factors, consideration of age-specific responses, and institutional or intervention techniques that could reduce ingestion rates. If however, risk managers elect to provide some margin of safety in the residential criteria, then higher intake rates could be considered for recreational activities and higher criteria could be developed and assessed through the use of the IEUBK.

Risk assessment for the non-lead metals did not add risks from different receptor groups (i.e., residential with public recreational) but additional sources of exposure were expressed as potential incremental risks above the residential baseline and the possibility of "double counting" was noted. Because of the many sources of metals exposures possible for individuals in the Basin, the added health risks from certain activities outside the home should be acknowledged so that appropriate risk management decisions can be made on a location-specific basis. There is an example of the residential and neighborhood recreational scenarios combined in Section 5 in a qualified manner. This analysis was provided primarily to illustrate the potential for additional exposures outside the home, and to demonstrate that risks could increase over baseline residential risks if residents also engage in recreational activities.

5d General Response regarding Incremental Exposures associated with Rails-to-Trails:
Consideration of potential recreational and occupational exposures associated with the Rails-to-Trails conversion are subsumed under the recreational and occupational scenarios considered in the HHRA. The types of activities anticipated for trail users and workers are accounted for in the scenarios addressed in the HHRA. Those include upland park activities, public beach activities, neighborhood sediment activities and nominal recreational activities associated with the residential scenario. The HHRA recognizes that public beaches and other common use areas throughout the Basin, including railroad right-of-way, are routinely used by members of the public. That was one criteria for sampling these areas for HHRA assessment. Incidents of excess lead absorption have been attributed to common use areas in the Lower Basin. There are

numerous public access areas throughout the Basin that will be assessed in the development of a Proposed Plan for cleanup. This effort will include properties on, adjacent to, and remote from the railroad right-of-way. All of these properties can be evaluated by the same methodology provided in the HHRA, and described above. Incremental exposures were characterized using typical parameters that are specified in the HHRA. Intakes are calculated in a straight-forward manner proportional to those parameters and media contaminant concentrations. Should risk managers elect to modify these parameters to site-specific concerns, intake rates can be adjusted proportionately. Blood lead increments can then be estimated by IEUBK applications or intake offset calculations can be performed to determine appropriate cleanup criteria. These calculations can be performed for sites on or off the right-of-way. The HHRA is familiar with the risk management plan adopted for the trail. That plan addresses the areas likely to be accessed on the railroad right-of-way. Within 1000 feet of any residence the entire right-of-way will be provided with a clean surface. This addresses the nominal aspects of recreation associated with the residential scenario. At all major access points, sidings, and select oasis location, similar right-of-way cleanup will occur and large oases are strategically placed along the trail to provide clean rest and stop-and-view areas. In remote contaminated areas warning signs will be posted to alert trail users to areas presenting excessive risk similar to warnings to avoid local hazards in numerous venues. The signage is provided to both advise users to avoid undesirable areas and to identify safe areas to recreate. The proposal was extensively reviewed by a number of public agencies and governments including the EPA, Panhandle Health District, State of Idaho, Coeur d'Alene Nation, several federal trustee agencies, and the Agency for Toxic Substance and Disease Registry. All have found the risk management and cleanup plan to be compliant with pertinent rules and regulations and protective of public health.

6 Comments Related to Subsistence Exposures: Contradictory responses were also received with respect to exposure estimates associated with Native American subsistence activities. Some comments suggested that inclusion of subsistence scenarios was inappropriate as these are largely hypothetical lifestyles that don't currently exist and are unlikely to occur in the future. Comments noted that foodstuff contamination data submitted by tribal representatives did not include collection and analytical methodologies. Other comments indicated that high-end or excessive intake assumptions were employed regarding aboriginal activities, and that combination of these activities resulted in highly unlikely estimates. Other comments suggest that the HHRA is not comprehensive with respect to subsistence scenarios in several regards. These reviewers believe that the geographic areas evaluated were based on arbitrary political boundaries that distort the risk assessment. These comments note that most of the Coeur d'Alene Lake and Spokane River in Idaho was screened out of the HHRA based on preliminary studies. The comments note that these studies concluded in Idaho that no further data collection was warranted, while health officials in Washington State concluded that fish in the Spokane River present an unacceptable risk. These reviewers believe that the screening assessment was flawed because assumptions protective of subsistence pathways were not included in the screening and that the Preliminary Remediation Goals (PRGs) used for comparison were ill-conceived with respect to subsistence requirements. The comments also note that dioxins, PCBs, herbicides, poly-aromatic hydrocarbons and other contaminants potentially significant to subsistence

pathways were not addressed. These comments also note that several media of potential significance were also not addressed including crustaceans, amphibians, mollusks, natural building materials used in lodge construction, mosses, herbs, ash, bark, and plant material used in clothing, medicines, and ceremonial practices. As a result, these comments conclude that the HHRA was not comprehensive with respect to potential subsistence pathways and underestimates risk. Other comments suggested that those exposure factors derived from literature values attributed to Tribes near the Hanford Nuclear Reservation were inappropriate to the Coeur d'Alene Tribe in the Basin environment.

6a General Response regarding Subsistence Exposures: *Subsistence scenarios and relevant exposure factors were developed in cooperation with Coeur d'Alene Tribe representatives. The Traditional and Current Subsistence scenarios were requested by the Tribe as representing possible future uses of the area. Exposure factors were derived specifically for the Coeur d'Alene Tribe. Scenarios and exposure factor analysis were patterned after the development of similar scenarios for the Columbia River Tribes. A cultural anthropologist, working for the Coeur d'Alene Tribe, reviewed and suggested appropriate modifications for each of the exposure factors. Each pathway was characterized individually. Risk managers can combine pathway results as considered appropriate to estimate total intake rates. However, it should be remembered, as noted in the HHRA, that intake from dietary sources may be more highly absorbed than those from soil and sediment sources. Numerous potential pathways and contaminants were not addressed due to lack of data. These pathways are discussed in Section 3.2. Little data exist for organic contaminants. The HHRA addresses the geographic area extending from Harrison to Mullan. The area of investigation was determined jointly by the EPA, State and Coeur d'Alene Tribe. The results and conclusions of the HHRA should not be extended to Coeur d'Alene Lake or the Spokane River except as explicitly noted.*

With respect to recreational, occupational and residential exposures to the resident population, most of Coeur d'Alene Lake and Spokane River areas were excluded based on the earlier screening risk assessment. However, neither screening risk assessment addressed high levels of exposure associated with subsistence lifestyles. The HHRA concluded that achieving appropriate levels of risk for subsistence lifestyles would require levels of environmental contamination comparable to background concentrations (Executive Summary and Sections 6.7.6 and 8.2.6). Although concentrations of metals surrounding Coeur d'Alene Lake occurred below screening levels, risks for subsistence lifestyles may remain unacceptable. With regard to the upper Spokane River, the Washington Department of Health recommended against consumption of whole fish. Beaches sampled along the Spokane River located downstream from the Upriver Dam were found to have metals concentrations comparable to naturally occurring background levels. Sample results in the upper Spokane river were also near background. Samples collected by the EPA and USGS showed decreasing metals concentrations from east to west along the Spokane River.

Harrison beach and Blackwell Island were retained for additional consideration in the HHRA. A determination was made that insufficient data were available to assess sport or subsistence

fishing in Coeur d'Alene Lake and downstream tributaries in Idaho. No evaluation of subsistence lifestyles, including the screening level risk assessment, has been accomplished for Coeur d'Alene Lake or Spokane River areas.

7 Comments Related to Site-specific Exposure Parameters: Comments were received regarding the use of ingestion and frequency of contact assumptions for soil sources that were developed from guidance and reviews of studies and literature from locations outside the Silver Valley. Some comments suggested that these parameters should be adjusted to reflect the comments of local respondents to questionnaires circulated prior to development of the HHRA. Many of these comments pointed out that soil contact was seasonally dependent due to snow cover in the winter, that many of the potential recreational and waste pile sites included in these surveys were little used or unknown to locals, that many of the sites listed in the HHRA were waste rock piles with little accessible fine material, and were unattractive as recreational areas. Other comments suggested that "high-end" ingestion estimates were used for several potential intake rates. Some comments also addressed a perceived lack of definition of exposure areas.

7a General Response regarding Site-specific Exposure Area Parameters: *Typically, risk assessment is designed to over-estimate, rather than under-estimate health risks in order to make appropriate risk management decisions that err on the side of protecting public health. The estimates used in the risk calculations for neighborhood and public receptors are weighted upwards in part to protect the very high frequency outdoor exposure of some children. Some comments suggest that the exposure frequencies and duration likely overestimate exposure for recreational receptors. Exposure frequency times used in the recreational scenarios, in terms of hours per day, are recommendations from EPA's 1997 Exposure Factors Handbook containing national information. Children in the more rural areas of the Basin would not be expected to spend less time outside than times estimated from the national information that includes urban children. Information from the Panhandle Health District's (PHD) lead intervention program does indicate that many children do spend very large amounts of time outdoors, particularly in summer (12 hours a day for some children).*

Exposure to soil both by ingestion and dermal contact continues during the winter inside the home, although likely at a reduced rate, because soil continues to be a component of indoor dust in the winter. It is reasonable to expect that some reduction in exposure to soil-borne contaminants may occur during times of snow cover. However, exposure attributable to soil continues as snow cover is not impervious to contaminated soils, snow cover may become contaminated with soil contaminants, winter footwear may enhance soil tracking into the home, snow may create muddy conditions that increase tracking of soils, and children have increased exposure to interior dusts during the winter. Rather than adjusting soil rates downward and dust rates upward in the winter months, both the soil and house dust components of "dirt" ingestion are averaged for the year in lead risk assessment . For non-lead exposures the RME scenario did not adjust contact downward for winter while the CT scenario assumed no contact. These two assumptions potentially bound the actual amounts ingested/absorbed. Data from waste rock piles were evaluated only for the populations of Mullan, Nine Mile, and Canyon Creeks. The

waste pile data included in the HHRA were collected from piles near residential homes. Data from piles were evaluated separately, and the data were not mixed with other media. The fine material in waste piles is present in the top inch and this material would adhere to children's hands and be ingested; however an insufficient amount was present for laboratory analysis. Samplers collected material to a depth of 6 inches. The assumption is that the concentration found in the 0-6 inch depth is representative of the concentration in the top inch.

The HHRA agrees with comments that the dermal surface areas used for the 4 to 11 year old age group were excessive for the exposure period. Preliminary estimates indicate neighborhood risks and hazards will decrease by less than 10% if skin surface areas are reduced. Risks and hazards for combined neighborhood exposures are not risk drivers and dermal exposures were a relatively low percentage of the total neighborhood risks (35% to 17% for arsenic). Modifying skin surface areas for neighborhood exposures does not affect the conclusions of the risk assessment or potential risk management strategies. Table 3 summarizes the change in risk and hazard estimates if reduced skin surface areas are used in the calculations.

With regard to exposure area definitions, the Coeur d'Alene Basin is an extremely large and complex area. Early in the planning process, in order to meet public requests, the HHRA was placed on an accelerated schedule to be completed in parallel with the RI/FS. It was recognized that, with the associated time and budget constraints, sampling efforts would be limited. Decisions were made to utilize existing data to the maximum extent practicable, fill major data gaps with focused sampling efforts, and not address all possible data gaps and exposure pathways. As a result, the data organization effort used the Idaho Department of Health and Welfare and Panhandle Health District lead health investigation and databases to define exposure areas for residential risk assessment. The HHRA relied on the Conceptual Site Model (CSM) approach of the RI/FS to incorporate efforts from federal environmental and ecological studies. The HHRA acknowledges that human exposures would only occur in a portion of the large CSM "exposure areas" identified on the maps in Section 3 and would focus on population centers. In general, the majority of the data used in the HHRA was at or near a home and/or population center and does represent reasonable human exposure potential (see Figures 3-12 through 3-26 in Section 3 for non-residential sample locations). Neighborhood exposures for elementary-aged school children are likely limited to areas reasonably close to home and in general, the sample locations used to evaluate neighborhood exposures are close to at least a few residences. Because much of the population is decentralized and the Basin is very large, a potential play area close to one home will be far from another within the same geographical region. The HHRA elected to address this complexity by determining average exposures to sediments, surface water, and waste piles in potential play areas within a region and evaluate whether such behavior might be "risky". Risk management decisions will be on a site-by-site basis and will likely require additional sampling and consideration of site-specific use patterns and proximity to residences.

7b General Response regarding Use of Public Input in Developing Site-specific Exposure Parameters: *A summary of the public comments received during the development of the HHRA*

will be included in the Appendices to the final document . Substantial efforts were made in the HHRA to address the concerns and input from the local citizens. Many comments were submitted regarding the applicability of the exposure factors developed and assumptions made to the Silver Valley. Comments provided indicate that residents are concerned that these factors were developed at other sites with conditions that do not apply to their town. The HHRA addresses these concerns by relying to the extent possible on observed blood lead levels from the Basin. The follow-up results from the Panhandle Health District's investigations of lead poisoned children were carefully examined and reviewed. The site-specific analysis was conducted to examine the relationships between observed blood lead levels and environmental exposures. The lessons learned at the Bunker Hill Superfund Site (BHSS) were applied to the extent possible. In particular, IEUBK modeling results have been carried out by both the EPA Default model that is generically applied to sites and the Box Model used at the BHSS and the results of both models are compared to observed blood lead levels in the Basin. All residential, recreational and occupational exposure factors utilized are appropriate to northern temperate climates.

Several comments refer to poor people or socio-economically disadvantaged families that possibly make up a disproportionate number of children with high lead blood lead levels. Others indicated that other sources of lead, aside from the mining industry, may be responsible for the high levels. In response, the HHRA provides a detailed description of Basin demographics, poverty levels and indicators, and discussions of the relationships between poverty and lead poisoning. The HHRA does address lead paint and the relationship between lead in blood, soil, paint, and dust in detail.

Many comments were received regarding distrust of the federal EPA, the desire to not be listed as a Superfund site, to let the State implement and manage the cleanup, accomplish the fastest possible cleanup, or not to have any cleanup. In response, the HHRA provides and contrasts current EPA policy, the federal Centers for Disease Control (CDC) guidance on lead poisoning in children, and the Remedial Action Objectives (RAOs) that have been employed at the BHSS. The HHRA also discusses, to the extent practicable, potential risk management tools and potential cleanup limits, as a vehicle to facilitate addressing these items in the overall process.

There were several comments regarding the use and obscurity of the various Common Use Areas (CUAs) listed in the survey. Many respondents said that they had lived in the Basin for their entire lives and either didn't know of these areas as recreational sites or never knew of anyone to use the site for recreation. In response, the HHRA developed intake rates for these sites as incremental exposures based on assumed frequencies. In this way the impacts of these sites can be assessed in addition to (or incrementally) to other sources of lead. Should risk managers believe that less time is spent at these areas, the increment can be adjusted proportionately. In this manner each site can be evaluated individually, should risk managers or the public deem that appropriate.

There were numerous requests from the public asking that they not be lectured to, that they be provided with the details of the assumptions being made, but that too many technical terms were

being used. Some feel they are not being provided with appropriate details and uncertainties with the assumptions made, etc. Others want the bottom line in plain language. These frustrations seemed to indicate that different levels of communication will be required to effectively inform the public about these issues. In response, the HHRA was produced with three levels of summary, a lengthy uncertainty discussion, and an immense Appendix with numerous details and support information. The document itself contains many figures and tables to illustrate the main points with a significant level of detail. Each section is individually summarized. Much of these summaries is repeated in the Section 8 Summary and Conclusions, that was circulated as a stand alone document for general public review. There is also an Executive Summary that provided the highlights of the HHRA for those wishing the shortest version. For those interested in complete detail, a 1400 page Appendices was provided on CD. Section 7 of the document is a lengthy discussion of the many uncertainties associated with a project of this type. As a result, the HHRA is repetitive, but provided a level of detail appropriate to the various audiences.

8 Comments Related to Exposure Pathways: Several comments referred to pathway considerations. Some comments indicated that lead paint-related pathways were over-emphasized in both graphics and in the analysis. Others felt that paint lead exposure was under-represented and the document was biased toward soil as the primary source of lead to children in the Basin. Comments were received indicating that secondary sources of lead to the home, such as soil tracked into the home from construction workers, those employed in the minerals processing industries, and older children and adult's recreational activities were ignored. Some comments indicated that the effects of soil on blood lead levels were understated, as the document ignored the soil-to-house dust-to-blood lead pathway. These comments suggested that blood lead impacts attributed to dust, are, in fact due to soil, manifested through dust. Others argued that the same effects were, in turn, due to deteriorating paint. There were also contradictory comments regarding the contribution of dietary lead to observed blood lead levels. Comments were received noting that dietary lead is absorbed at a higher rate than lead from soil and dusts. Other comments suggested that recent work by Manton et al. suggests that dietary lead plays a minor role in contemporary lead exposure, and that blood lead reductions noted in the 1990s were due to reduction in dietary lead throughout the U.S. One comment objected to the presentation of a single age-group in the section of the document illustrating relative contributions to estimated total lead intake from different pathways.

8a General Response regarding Exposure Pathways: *Most of the comments received regarding pathways suggested alternative interpretations of the results of the site-specific and IEUBK analysis. These comments, for the most part, indicated that various pathways should be emphasized to a greater or lesser extent. General Responses 3a-d and 9a-d discuss the site-specific and IEUBK analysis, respectively. Many of the pathways-related comments address dust and speculate on the various sources of lead to house dust. It is important to note that actual observed house dust lead levels were used in both the site-specific and IEUBK model analysis that relate blood lead levels as a dependent variable to environmental dust concentrations. Except in the IEUBK application, where mean community dust lead*

concentrations were substituted for missing observations, these were paired observations from individual homes. As a result, the sources of lead to dust, such as paint, yard soils, materials tracked in by workers, fugitive dusts, etc. are inherent in the analysis. Dependent blood lead levels are directly related to house dust and other environmental sources as independent variables in either the empirical or mechanistic model derived analysis.

In either case, house dust is identified as the largest source of lead, particularly to young children. Any significant effects in addition to dust from soil or paint are similarly independent and likely represent primary source pathways exclusive of house dust. However, outdoor soils from the home yard and neighborhood, deteriorating paint, occupational or recreational contaminants on clothes and shoes, windblown dusts, and others are potential sources of lead to dust. In this sense, dust is a secondary source of lead to children, the lead ultimately coming from the other sources. It is important to quantify these effects in order to develop remedial strategies that will reduce dust lead concentrations. These soil and paint-to-dust-to-blood relationships can be evaluated simultaneously through pathways, or structural equations analysis, as some comments suggest. This was accomplished for the BHSS, using an accumulated data set of several thousand observations collected over a decade. There were insufficient data available to undertake structural equations analysis for the Basin data set. As a result, blood lead and dust lead were evaluated as dependent variables in separate regression models. These results were discussed in General Response to Comment # 3. The findings showed little difference from the same models applied to the BHSS data set, as discussed in General Response to Comment # 4e. The same source variables were significant and the regression coefficients or slope values were similar.

As a result, the findings regarding pathways explored through structural equations analysis at the BHSS are useful to consider in relation to the comments offered for the HHRA. These models indicate that about 40% of the blood lead absorbed from soils and dusts is through house dust with about 30% from community-wide soils and 30% from the home yard and immediate neighborhood. These relative contributions agree with findings of earlier studies conducted in the early 1980s and analysis used to develop the cleanup criteria for the BHSS. The same structural equation models suggest that community-wide soils contribute between 50% and 60% of the soil lead component in house dust with the neighborhood and home yard contributing about 20% each. This results in soils overall contributing about 80% of lead to house dust in the pre-remedial environment and an estimated 50% to 60% post-remedial, the remainder coming from other sources including lead paint.

Analysis of the relationship between house dust and soil lead levels show that dust lead concentrations demonstrate large variation. Soil lead levels are significantly correlated with ($p=0.0001$), but explain only 20% of the variability in house dust lead concentration. Approximately half the soil contribution to lead in house dust is attributable to the greater community, with the remainder coming about equally from the home yard and the immediate neighborhood of the home. Review and analysis of dusts collected from mats placed in BHSS homes and associated questionnaire data, show that much of the variation in house dust lead

levels is associated with housing, socio-economic, behavioral, family and occupational and recreational related factors. Although several of these factors do not affect dust lead concentration, as much as 25%-30% of the variability in dust and lead loading rates is explained by these factors.

Socio-economic status plays a complex role in dust loading relationships. Home or housing related factors (home age, yard covering, inside and outside paint condition); socio-economic factors (own/rent, occupancy time, number of residents); personal habit or behavior factors (use of mats, children's outside play frequency, general household hygiene, pets), and occupational/hobby related factors (mill worker, carpenter, landscaper, sanding within the home) influence dust loading rate or the amount of dust in the home. These variables are indices of complex social structure with many inter-related factors that influence both dust and lead loading in this community. In the presence of active sources of lead (i.e., contaminated soils or paint), this can result in higher dust lead loading rates. Many of the factors relate to both lead sources and dust loading. Paint condition can influence both dust and lead loading rates as a contaminant source and as an indicator of household hygiene and socio-economic status. Grass cover of the yard and general household hygiene are significant factors in that both contain lead sources and affect dustiness.

The relationships are complicated and often not straightforward. Older, more established homes may have higher lead paint content, but tend to have better paint condition and yard cover. The number of children and regular visitors and time spent outside positively correlate with owner/renter and length of occupancy status, and dust loading rate. Comparable background house dust studies were conducted in socio-economically similar communities to those in the BHSS. These communities located in northern Idaho were remote from the mineral processing industry and were selected to represent similar housing conditions. Background results suggest that concentrations in similar socio-economic and housing conditions are near 200 mg/kg lead.

9 Comments Related to the Applicability of the IEUBK "Box" and EPA Default Models:
Several comments were received regarding the applicability of these models to the Basin situation and the use of the IEUBK in risk assessment and risk management activities. Comments from some reviewers indicated that IEUBK is generic and simplistic and is inappropriate for risk assessment uses in the Basin. Reviewers also offered suggestions for alternative models that they believed to be more appropriate. Other reviewers supported the use of the IEUBK, but felt the EPA Default Model was the most appropriate to apply in evaluating risk in the Basin. These reviewers felt there was insufficient evidence to justify applying the Box Model outside of the Bunker Hill Superfund Site and believed that any cleanup predicated on the results of the Box Model would not be protective of children "in the tail of the EPA Default Model". Other reviewers indicated that the EPA Default Model clearly overestimated risk in the Basin. Other comments questioned the applicability of site-specific information and the results of site-specific analysis being incorporated into the model. Comments received suggested that inclusion of such data would result in a cleanup that was not protective, others suggested that it would result in unnecessarily over-protective cleanup. Other comments indicated the IEUBK

analysis was appropriate and, in combination with the thorough discussions of uncertainties, added to the base of knowledge necessary to make sound risk management decisions. Several specific technical comments addressed the use of the 18% bioavailability absorption rate in the Box Model, the inclusion of soil lead estimates from outside the home environment in the Box Model, the selection of the geometric standard deviation (GSD) in both models, and the comparison of model results to observed blood lead levels in the Basin. Comments were made both supporting and discounting the selections applied for these factors in the HHRA. Other comments noted the differences in model performance among the geographic areas, some indicating that this was valuable information, some suggesting this was evidence of the inapplicability of the model, and others arguing that this supported the use of the EPA Default Model in risk assessment and applying different risk management strategies in these areas. Comments were received suggesting that it was inappropriate, according to EPA guidance, to evaluate short-term exposures using the IEUBK and that, if employed, longer averaging times should be used. Reviewers from EPA's Technical Review Workgroup for lead (TRW) disagreed and, conversely, suggested shorter averaging periods for these exposures.

9a General Response regarding the Applicability of the IEUBK "Box" and EPA Default Models: *The IEUBK model is a series of mathematical equations that simulates the behavior of lead taken into the body. The model has an intake component that estimates how much of the lead is taken into the body from five main routes of exposure. Those exposure routes include lead from the diet, drinking water, soil and dust, air and other incidental ingestion sources, such as paint or consumer items. The uptake portion of the model then estimates how much of the intake lead is absorbed in the body, and the bio-kinetic portion distributes that absorbed lead among body tissues and excretes the remainder. The model provides predictions of mean blood lead levels for 0-7 year-old children in one year age increments. The mean blood lead estimate can be interpreted as the most likely response expected for the typical child exposed to those particular environmental conditions. In a follow-up step, probabilistic methods are used to estimate the distribution of blood lead levels for a population exposed to those conditions. In comparison to risk assessment methodologies used for other contaminants, such as that accomplished for several metals in Section 5 of the HHRA, the IEUBK is neither simplistic nor generic. The model allows for input of site-specific environmental data, exposure factors and route specific absorption rates; integrates the effects of lead deriving from different routes; and relates the biological response directly to toxicity criteria on an age-specific basis. This procedure is considerably more complex than that applied in non-carcinogenic risk assessment, and results in more precise and less uncertain estimates of effect, than is typically obtained. As a result, lower margins of safety are employed in sub-chronic lead risk assessment than in the methods used for other metals.*

The IEUBK has been extensively reviewed by the EPA, including reviews by the Science Advisory Board (SAB), and subsequent guidance reflecting these reviews was issued approving the IEUBK for sub-chronic risk assessment for lead in children. These guidance documents are provided in Appendix O. None of the other bio-kinetic simulation models suggested by reviewers have been similarly reviewed, nor has any guidance been issued regarding use of these alternate

techniques. EPA guidance does recognize site-specific empirical modeling of blood lead levels and dose-response as a useful tool to supplement IEUBK analysis. That analysis was accomplished in the HHRA and is discussed in General Response to Comments # 3 and 4. EPA guidance also recognizes that site-specific parameters can be input to the IEUBK model, provided those parameters are representative of site conditions. Four applications of the IEUBK were presented in the HHRA, to provide reviewers and risk managers with alternative analysis for consideration in developing risk management strategies. Those applications included both the community and batch mode versions of the model using nationally representative assumptions for soil and dust ingestion rates and absorption factors in the EPA Default Model application. Similar applications of the site-specific Box Model developed at the BHSS were also presented in the HHRA. The Box Model uses site-specific parameters developed at the BHSS.

The most significant differences in the EPA Default and Box Model is that the Box Model considers that a significant portion of the soil typically ingested by children derives from the local community and neighborhood and assumes that 18% of the lead in soil and dust is absorbed. The EPA Default Model assumes that all soil derives from the home yard and that 30% of the soil/dust derived lead is absorbed. With respect to pathways, the parameters in the Box Model were derived from structural equation analysis conducted on more than a decade of data collected at the BHSS. The inclusion of soil sources from outside the home yard is frequently applied in IEUBK analysis, if there is reason to believe that children access these soils. In the BHSS, both structural equations and general linear multiple regression analysis indicate that community and neighborhood soils are significantly related to blood lead levels. Pathways analysis indicates that about 40% of soil and dust exposure derives from house dust, with 30% coming from the home/immediate neighborhood and 30% from the greater community. Follow-up investigations of children with high blood lead levels indicate that neighborhood and community soils are important sources in children's exposure profiles.

Several reviewers question whether these same pathways are applicable to the Basin. Site-specific quantitative regression analysis relating blood lead and soil and dust lead levels indicate similar levels of significance and coefficient, or slope, values for the Basin and BHSS populations. These analyses, found in Section 6.4, suggest that the same pathways are involved in both areas at similar magnitudes. Additional discussions are included in General Response # 3a-d, 4a, 7a-b, and 8a. There do seem to be differences between the upper and Lower Basin with respect to this question. The primary sources, communities, demographics and socio-economic factors suspected to influence lead exposures in the Box have more in common with the upper Basin than in the Lower Basin. Most of the children in the upper Basin live in communities of similar size, history, housing characteristics and infrastructure to that of the BHSS area. Mineral industry activities were common throughout the history of the BHSS and the upper Basin influencing both the sources present and the character and economies of the local communities. Relatively high levels of community and residential soil and household dust lead are noted for the upper Basin.

The Lower Basin is more rural in character than the BHSS and upper Basin, with homes being located in the countryside or small unincorporated villages with limited infrastructure. Residential soil and dust lead concentrations are low for homes outside the Coeur d'Alene River floodplain. Residents of the Lower Basin have been more dependent on agriculture, and natural resource industries other than mining. The age of housing, income and poverty indices noted for Shoshone County and the upper Basin are not so evident in the Lower Basin and Kootenai County. Follow-up evaluations of children with high blood lead levels indicate potentially high exposures related to residential soils and dust in upper Basin communities, as opposed to extended recreational activities in the Lower Basin. Both of these observations are consistent with plausible interpretations of the results of IEUBK model assessments. Residential soil and dust exposures largely explain excess lead absorption in the upper Basin in a manner similar to that observed in the BHSS. High blood lead levels in the Lower Basin are explained by incremental recreational activities.

9b General Response regarding Bioavailability Estimates used in the IEUBK Model:
Reviewers have also questioned the appropriateness of applying the 18% bioavailability factor from the Box Model to the Basin. The dose/response relationship between blood lead and soil and dust exposures at the BHSS has long been noted to be reduced from that inherent in the default assumptions of the IEUBK model. In previous analysis, for practical purposes, the reduced effect has been attributed to lowered bioavailability of the soils and dust. The 18% absorption factor also results from analysis of the last twelve years of paired blood lead and environmental exposure data from the BHSS. The value was derived by estimating absorbed lead levels, or uptake, from observed blood lead levels using bio-kinetic factors common to the IEUBK Model. Bioavailability was determined empirically by relating uptake to estimated intake rates calculated by multiplying soil and dust lead concentrations by ingestion rates, also consistent with the IEUBK model. This method has the effect of attributing the reduced dose/response to lowered bioavailability. It is also possible, however, that children in the BHSS may exhibit lower ingestion rates than those assumed in the IEUBK model. There is reason to believe that ingestion rates may be depressed due to the ongoing long-term intervention and education programs that have sought to reduce lead intake through behavioral modification. If that were the case, then bioavailability would be greater than 18%. Similarly, the practice of using a 175 micron sieve for soils and dusts could potentially increase soil and lead concentrations above that expected from 250 micron sieve recommended for input to the IEUBK. This also would result in higher bioavailabilty being calculated for the BHSS. As a result, for the BHSS the 18% figure for bioavailability should be regarded as a minimum. The actual value could be higher if ingestion rates are less than the default assumptions. It is less likely that the effective bioavailability is lower than the 18% estimate.

A similarly reduced dose/response relationship is noted between observed blood lead levels and measured soil and dust lead concentration in the Basin. Some reviewers have suggested that Basin-wide ingestion rates may also be suppressed due to the same intervention and education efforts being extended to the Basin and the general knowledge of lead-related hazards in the area. The 175 micron sieve is also used to prepare soil and dust samples in the Basin. Both of

these factors would suggest that bioavailability may be higher than 18% in the Basin as well. However, there are other plausible arguments for lower bioavailability in the Basin than the BHSS. Soil and dust contamination in the BHSS was influenced by pyro-metallurgically processed ores from the smelter complex. These ores were released to the environment in predominantly lead oxide forms, whereas, the majority of ores released from mining and milling operations were released as lead sulfides. Generally, lead sulfide species are less soluble and less bioavailable than lead oxides.

However, there are also reasons to suspect that mill and mine tailings releases have undergone secondary mineralization in the environment and are no longer sulfide minerals. Milling practices conducted through most of the twentieth century did not effectively capture oxidized lead in the concentrating process. As a result, oxidized forms of lead in the original ores, present in near surface ore bodies, were preferentially discharged. The soil and dust particles that children access are generally small, in the <150 micron range, and more available for ingestion because of frequent hand-to-mouth activity. That is, smaller particles adhere to hands and are ingested orally. These small particles have been in the environment for several decades, many being discharged a century ago. There has been mechanical abrasion and much reworking of tailings due to human and stream activities. This decreases particle size and increases surface area to volume ratios providing increased exposure to air and more reaction sites for oxidation to occur. During this time, those tailings that reach children, i.e., the smallest particles at the ground surface, have been exposed to the atmosphere and aerobic hydrologic conditions. As a result, the particles or the surface fraction have become oxidized and are more bioavailable to children. Electron microscope images of lead particles in this size range often show oxidized surface inclusions that can be dissolved even on chemical species that are otherwise relatively unsoluble. As time goes on, these particles will only become more oxidized and more bioavailable unless confined to anaerobic conditions. As a result of all these factors, it is unlikely that the particles ingested by children are purely a sulfide form and are less likely to be in the future. This conclusion is not inconsistent with the results of the swine studies conducted in Region VIII of the EPA. Several tailings and sulfide ore wastes were found to be bioavailable. The overall results of those investigations suggest that average bioavailability of all wastes and soils tested reflects the 30% bioavailability default advocated by EPA. The 18% used in Idaho is actually on the low side of bioavailability observed across the range of potential sources. These studies are summarized in Appendix O.

9c General Response regarding the GSD used in the IEUBK Model: *Comments were also received regarding the appropriateness of the geometric standard deviation (GSD) employed in predicting the distribution of blood lead estimates relative to the mean predicted by the IEUBK model. The HHRA used the default GSD value of 1.6 recommended by current EPA guidance. That GSD is applied to the mean blood lead estimate determined by the IEUBK and reflects the variation in outcome blood lead levels for the exposed population. The result, if applied to a population, can be presented and interpreted as the percent of children expected to exceed critical toxicity or blood lead levels. If applied to a particular exposure situation, the result can be presented and interpreted as the probability that an individual would exceed that criteria. In*

the community mode IEUBK application, the GSD is applied to a mean blood lead estimate resultant from, among other factors, the mean community-wide soil and dust lead concentrations. The GSD that is applied should reflect both the inherent individual variation in response and the variation in exposure. In the batch mode application, mean blood lead estimates and probabilities are determined for each individual situation and the results are aggregated to estimate a community mean and percent to exceed criteria for the community. In this case, the overall GSD for the community is calculated from the aggregate risk, and represents both the inherent individual and exposure-related variation. In the batch mode, the mean blood lead level and probability to exceed toxicity criteria can also be determined and applied to the individual situation. For the individual situation, the GSD reflects only the inherent variation in response among individuals. The default GSD recommended by the EPA is representative of a number of investigations with varying degrees of exposure variation inherent in results. Applying the typical GSD value of 1.6 to individual situations could overestimate the probability of exceedance for the individual. Risk managers may want to consider the application of the 1.6 GSD in the batch mode application as an additional margin of safety when considering the probability of an individual exceeding toxicity criteria. This consideration would not apply to the community-wide estimates of the percent of the population to exceed these criteria.

9d General Response regarding Observed and Predicted Blood Lead levels from the IEUBK Model: *Several comments were received regarding the ability of the IEUBK to predict observed blood lead levels. Several comparisons of predicted and observed blood lead levels were included in the HHRA. Many comments were received regarding the representativeness of the observed blood lead data set. This issue was discussed in detail in Sections 7.4 and 8.11 of the HHRA and additional discussion is included in General Response to Comments # 2. There are several considerations with regard to what are the appropriate comparisons between the model projections and observed blood lead levels. One comment was received suggesting that observed and predicted blood lead levels are totally unrelated data sets and any comparison is improper. This conclusion is incorrect. The relationship between blood lead levels and environmental exposures is examined throughout the HHRA by a variety of methods. In regression analysis, such as the site specific quantitative models discussed in General Response to Comment # 4, it is common practice to compare dependent blood lead levels predicted from independent exposure variables to observed concentrations. In IEUBK analysis, the same independent exposure variables are input to a mechanistic model and outcome blood lead levels are predicted. It is also common to compare these predictions to observed blood lead levels. In both cases, the blood lead and environmental levels are related. Both the dependent and independent variables come from the same home and community and the purpose of the analysis is to investigate and quantify any relationship between the variables. The regression analysis discussed above shows a relatively strong relationship, that is consistent with plausible environmental and biological processes, and is similar to the findings of investigations at other sites including the BHSS. As a result, it is appropriate to compare predicted and observed blood lead levels in both empirical and mechanistic procedures.*

However, it is important to note that the IEUBK model predicts the mean, or most likely response, to the exposures represented in the model input. The model also provides the probability that higher or lower blood lead levels may be observed. As a result in comparing individual blood lead levels to IEUBK projections, the distribution of probable results must be considered. An individual observed blood lead may reasonably fall within the overall distribution of probable blood lead levels. For example, for a mean blood lead prediction of 5 μg/dl with a GSD of 1.6, approximately 68% of children so exposed would be expected to have a blood lead level between (5/1.6=)3.1 μg/dl and (5x1.6=)8.0 μg/dl, and 95% of children would have levels between (5/1.6/1.6=)2.0 μg/dl and (5x1.6x1.6=)12.8 μg/dl. In comparing observed and predicted levels from the IEUBK, it is necessary to compare the mean projection and distribution of responses. These comparisons are made in Section 6.7 of the HHRA. Examination of these results show that the observed mean blood lead levels in the upper Basin are best described by the Box Model and in the Lower Basin by the EPA Default Model. Similarly, the percent to exceed 10 μg/dl is best described by the batch mode of the same IEUBK models. Finally, it is important to note that neither the Box Model or the EPA Default model were calibrated or otherwise adjusted in response to, or to reflect, Basin blood lead levels. The site-specific or default parameters used in these models were developed from either analysis of data from the BHSS or national observations, respectively. One disadvantage of this methodology is that the number of observations that can be directly compared is limited to the number of communities examined, or for the Basin, eight comparisons.

The record for the BHSS is large with 5 communities being observed for 13 years. Appendix Q to the HHRA summarizes the results of applying the Box and Default models to the BHSS and figures are included showing predicted and observed mean blood lead levels for the entire period. These figures, the supporting discussions and analysis in Appendix Q suggest that the Box Model has effectively predicted blood lead levels in the BHSS for more than a decade. The EPA Default model has been nearly as effective a predictor in recent years, but tended to over-predict in earlier years.

Determining which model performs most effectively for the Basin depends on several of the issues discussed in the HHRA, reviewers comments and response to those comments. The Box Model effectively predicts both mean blood lead levels and percent of children to exceed 10 μg/dl in the upper Basin, in a manner consistent with its performance in the Box over the last decade. Risk managers could consider the Box Model appropriate to characterize risk in the Basin provided that similar pathways and dose-response relationships are involved and that the blood and environmental lead levels evaluated in the model are representative of the Basin population. There are questions as to whether the observed blood lead levels are representative of the overall Basin population. Regression analysis relating blood lead and environmental lead levels suggest similar pathways, with somewhat lower slope values for soil and dust concentrations in the Basin. There are many similarities among the BHSS and upper Basin communities from size, socio-economic, history, industry, economy, size, infrastructure, and demographic perspectives. There are reasons to suspect that a somewhat lower dose/response

rate with soils could be associated with mine-related to smelter-related sources of lead in the community.

10 Comments Related to Interpretation and Discussion of Applicable Rules, Regulations and Guidance: Several comments were received regarding interpretation and discussion of EPA and other federal guidance in the HHRA. Some comments indicated that the HHRA provides inappropriate risk management discussions and that those presentations bias the document toward particular risk management decisions. Some comments suggest that the HHRA is pre-disposed toward soil cleanup and ignores intervention-directed risk management strategies. The same comments also indicated that multiple-safety factors incorporated into the analysis and federal guidance requirements result in pre-determined cleanup levels beyond the level of diminishing returns. Other comments indicate that the HHRA is biased toward use of the Box Model in eventual cleanup decisions, and as a result, is not protective. These reviewers believe federal guidance requires the use of the EPA Default Model and that there is insufficient site-specific data to justify other models or risk management methods. These reviewers suggest that EPA guidance requires primary (i.e., preventative) risk management responses and that intervention activities are secondary measures non-compliant with guidance. Several comments referred to the EPA's guidance regarding protection of individual children versus percentages of the general population and there were differing views of the appropriateness and interpretation of Public Health Service and federal Centers for Disease Control (CDC) guidance on these matters. Some reviewers felt the HHRA was over-protective, while others felt too many children were left at risk under these interpretations. Comments were also received regarding inconsistencies in federal lead exposure and risk assessment/risk management requirements. These reviewers point out that federal OSHA standards require maintenance of adult occupationally exposed female blood lead levels less than 30 μg/dl as protective of the fetus, as opposed to the Public Health Service and EPA's adoption of the 10 μg/dl criteria. Comments also pointed out that federal Housing and Urban Development (HUD) guidance advises soil cleanup levels as high as 5000 mg/kg lead in contrast the EPA Default Model, de facto, 400 mg/kg level. Other comments pointed out that Tribal risk assessment policies that are more stringent than EPA guidelines have been ignored in the HHRA. Comments were also received regarding the appropriateness of lead toxicity discussions in both the HHRA and EPA guidance. Some reviewers felt the document failed to differentiate among potential adverse effects with respect to different levels of exposure. These reviewers objected to terminology used with respect to "lead poisoning" and "rates of toxicity" in the population and believed that all potential effects should be discussed in relation to the blood lead levels observed in the Basin. These reviewers also indicated that the discussions do not reflect the likelihood and severity of effects consistent with Basin blood lead levels, and as a result overstate the risk of adverse effects. Conversely, other reviewers believe that the risk and severity of effects associated with lead are understated because the chronic effects of lead poisoning are not considered additive to other contaminant risks in non-carcinogenic assessments under current EPA policy.

10a General Response regarding Risk Assessment versus Risk Management Issues: *Numerous comments addressed eventual risk management issues associated with the HHRA. The purpose*

and objectives of the HHRA are to assess the potential risk of adverse human effects associated with contaminated environmental media in the Coeur d'Alene Basin. Risk assessment identifies those contaminants, media, pathways, sources of contamination, routes of exposure, and potential for human intake that could pose unreasonable risk. The risk assessment process does not determine cleanup strategies or criteria for contaminated media. In situations similar to the Basin, however, public health authorities have found excess absorption to be occurring and preventative actions are in place. Risk management activities are already underway in the form of a lead health intervention program being locally implemented and focused remedial actions being conducted under emergency authority. The adjacent Bunker Hill Superfund Site (BHSS) has been implementing a variety of cleanup actions and risk reduction measures for more than a decade, many of which were the genesis of Basin-related activities. As a result, the HHRA does, to the extent possible, consider and review the information obtained, the relationships observed and lessons learned in the numerous efforts to eliminate lead poisoning among the children of the Silver Valley over the last three decades. Actual measurements of blood lead data paired with environmental exposures from a substantial portion of the population were used to identify site-specific factors that influence absorption in the Basin. Quantitative regression analysis was accomplished to identify site-specific relationships among blood and environmental lead levels. The follow-up records of dozens of children with high blood lead levels were gathered. Clues to both environmental sources and factors that influence the potential for excess absorption were identified and discussed. Finally, a site-specific bio-kinetic model that has accurately predicted blood lead levels and responses to remedial activities at the BHSS was utilized and the results contrasted with conventional risk assessment techniques.

These efforts have resulted in a lengthy, complex and comprehensive HHRA. This information has been presented, analyzed, evaluated, and discussed in Sections 1 through 6. Section 6 also provides discussions and analysis of the various factors and relationships that help to define the potential blood lead absorption problems in the Basin for the consideration of risk managers. Both qualitative and empirical relationships are derived and presented. This analysis was developed at the request of State of Idaho health authorities to aid in developing a comprehensive risk management strategy for the Basin. Section 7 discusses the uncertainties associated with the entire HHRA. In whole, the HHRA provides risk managers, the public, reviewers and other interested parties with information to independently evaluate developing effective risk management strategies. Determinations regarding particular elements of the eventual strategies and compliance with applicable State, federal or Tribal ARARs is a determination that is addressed in the Feasibility Study (FS), risk management process, and Proposed Plan for the site.

10b General Response regarding Compliance with the NCP and Risk Assessment Guidance and Policy: *With respect to guidance specific to the risk assessment process, the methods employed are compliant with applicable EPA guidance and the National Contingency Plan. EPA guidance on conducting risk assessments specific to human health hazards associated with lead were included in Appendix O in the HHRA and as Appendix D to the December 2000, Human Health Alternatives Technical Memorandum. EPA risk assessment guidance includes the 1994*

OSWER Directive #9355.4-12 Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities and the subsequent 1998 OSWER Directive #9200.4-27P Clarification to the 1994 Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. These directives recommend the IEUBK Model as a primary tool for lead risk assessment, describe the objective of limiting individual risks of having a blood lead level of 10 μg/dl or greater to no more than a 5% probability and focusing action preventing exposure rather than intervention after an exposure has occurred. Additionally, these directives clarify shared objectives and distinctions in the CERCLA, RCRA, and TSCA approaches to lead risk assessment and remedial response. A common objective is prevention of exposure to lead, based on use of the IEUBK model to predict risks from environmental sampling data, rather than relying on elevated blood lead levels as a guide to response actions. Specific considerations of the use of observed blood lead information in concert with modeling results are discussed in detail.

Excerpted from OSWER Directive #9355.4-12

> *In all three of these programs (CERCLA, RCRA, and TSCA Section 403), the Agency's approach is to consider soil lead in the context of other lead sources that may be present and contribute to the total risk. For example, TSCA Section 403 specifically requires the hazards posed by lead-based paint and lead contaminated interior dust, as well as lead-contaminated soil. Likewise, the OSWER Soil Directive includes evaluation of other lead sources as part of site assessment / investigation procedures. In addition, the primary focus of the three programs is primary prevention — the prevention of future exposures from the source(s) being remediated.*

Excerpted from OSWER Directive #9200.4-27P

> *At lead contaminated residential sites, OSWER seeks assurance that the health of the most susceptible population (children and women of child bearing age) is protected and promotes a program that proactively assesses and addresses risk. OSWER believes that predictive tools should be used to evaluate the risk of lead exposure, and that cleanup actions should be designed to address both current and potential future risk.*
>
> *While health studies, surveys and monitoring can be valuable in identifying current exposures and promoting improved public health, these are not definitive tools in evaluating potential risk from exposure to environmental contaminants. In the case of lead exposure, blood lead monitoring programs can be of critical importance in identifying individuals experiencing potential negative health outcomes and directing education and intervention resources to address those risks. However, CERCLA 12(b) requires EPA to select cleanup approaches that are protective of human health and the environment and that utilize permanent solutions to the maximum extent practicable. To comply with the requirements set forth in CERCLA 12(b), OSWER will generally require selection of cleanup programs that are proactive in mitigating risk and that do not simply*

rely on biological monitoring programs to determine if an exposure has already occurred.

To meet these objectives, OSWER will seek actions that limit exposure to soil lead levels that a typical child or group of similarly exposed children would have an estimated risk of no more than 5% of exceeding 10 μg/dl blood lead level. If lead is predicted to pose a risk to the susceptible population, OSWER recommends that actions be taken to significantly minimize or eliminate this exposure to lead.

Prevention of lead exposure is critical because adverse health effects resulting from lead exposures to young children persist into adulthood.

10c General Response regarding Applicable or Relevant and Appropriate Requirements (ARARs): *Comments were received referring to HUD soil cleanup guidelines indicating that HUD and EPA guidance exists for soil lead action levels from 400-5000 mg/kg, that this guidance allows methods other than soil abatement as a remedy, and that EPA sanctioned biological monitoring and reactive intervention strategies in lieu of soil remediation at the Leadville site in Colorado. Presumably, these comments are in reference to activities in regulatory programs other than Superfund and how these regulations are applied in CERCLA (Comprehensive Environmental Response Compensation and Liability Act), RCRA (Resource Conservation and Recovery Act), and TSCA (Toxic Substances Control Act). With respect to the soil action criteria, consideration of interim Title IV º403 of TSCA is described in various EPA-OSWER directives. These regulations are not considered to be ARARs by the EPA. TSCA º403 guidance should be evaluated separately from the OSWER program guidance as the former mainly addresses concurrent soil abatement associated with lead paint removal programs in largely urban areas. Three EPA OSWER directives address consideration of this guidance in CERCLA and RCRA actions: (1) #9355.4-12, EPA/540/F-94/043, August 1994; (2) #9200.4-27, EPA/540/F-98/030, August 27, 1998; (3) # 9200.4-29, EPA 540-F-98-061, December 1, 1998. These documents are included in Appendix O to the HHRA. The 12/1/98 guidance discusses soil clean-up levels addressed in Title IV TSCA º403 and both CERCLA and RCRA sites:*

[par. 1 of Directive] "...questions have been raised about the relationship between the proposed TSCA º403 rule [proposed June 3, 1998] and the Office of Solid Waste and Emergency Responses' Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Facilities (OSWER Directive #9200.4-27P, August 27, 1998)." [p. 2, par. 1 of Directive] "EPA has proposed a 2,000 ppm hazard standard for lead in soil at which children's exposures will be associated with a greater certainty of harm...The hazard standard was intended as a "worst first" level that will aid in setting priorities to address the greatest risks promptly. The proposed º403 regulations and the accompanying guidance are to be used by Federal, State, and Tribal lead paint programs, as well as by the industry performing inspections and risk assessments."

[p. 2, par. 3, "OSWER's Soil Lead Directive"] "The OSWER soil lead directive that

provides guidance for the cleanup of lead-contaminated sites under the CERCLA and RCRA laws is unaffected by this proposal. CERCLA and RCRA soil lead clean-ups should follow the approach in the 1998 directive...The TSCA º403 proposed 2,000 ppm hazard level should not be treated as an Applicable or Relevant and Appropriate Requirement (ARAR), "to be considered" or TBC or media cleanup standard (MCS). As recognized in the TSCA º403 rule, lead contamination at levels below 2,000 ppm may pose a serious health risk based upon a site-specific evaluation and may warrant timely response actions. Thus, the 2,000 ppm proposed standard under TSCA º403 should not be used to modify approaches to addressing Brownfields, RCRA sites, National Priority List (NPL) sites, Federal CERCLA removal actions, and CERCLA non-NPL facilities."

[p. 3, 1st full par.] "In the absence of site-specific information, EPA believes that levels above 400 ppm may pose a health risk to children through elevated blood lead levels. The 400 ppm screening level identified in the OSWER soil lead guidance is consistent with the "level of concern" identified in the preamble to the proposed TSCA º403 rule."

With regard to cleanup levels established at the Leadville site, the remedial action is an actively monitored pilot program that is specifically intended to set no precedent nor serve as an example for response actions at other sites. The record of Decision (ROD) for the site specifically states that "... Because this decision will result in hazardous substances remaining on-site, above health based levels, five-year reviews of this response action will be required." The Leadville cleanup is experimental and the efficacy of this ROD will be evaluated as a pilot project at prescribed interval by a group of outside reviewers.

Some comments pointed to an apparent discrepancy in federal blood lead standards for reproductive aged females between EPA public health policy of 10 µg/dl and the Occupational Health and Safety Administration (OSHA) worker protection 30 µg/dl requirement. OSHA standards are not considered ARARS by the EPA. There are two principal reasons for the treatment of OSHA standards as non-ARARs in the NCP. First, Congress appears to have intended that certain OSHA workplace standards apply directly to all CERCLA response actions. Second, EPA believes that OSHA is more properly viewed as an employee protection law rather than an "environmental" law, and thus the process in CERCLA section 121(d) for the attainment or waiver of ARARs would not apply to OSHA standards (55 FR 8679).

Moreover, OSHA does also state that "the blood lead levels (BLL) of workers (both male and female workers) who intend to have children should be maintained below 30 µg/dl to minimize adverse reproductive health effects to the parents and to the developing fetus." However OSHA then goes on to say that, "There is a wide variability of individual response to lead, thus it is difficult to say that a particular BLL in a given person will cause a particular effect. BLL measurements show the amount of lead circulating in your blood stream, but do not give any information about the amount of lead stored in your various tissues. BLL measurements merely show current absorption of lead, not the effect that lead is having on your body or the effects that past lead exposure may have already caused." [57 FR 26627, May 4, 1993, as amended at 58 FR 34218, June 24, 1993]

The 10 μg/dl value is a default value used in the Adult Blood Lead Model recommended by the Technical Review Workgroup for Lead (TRW). The basis for using 10 μg/dl is that fetuses and neonates are a highly sensitive population with respect to the adverse effects of lead on development, and 10 μg/dl is considered to be a blood lead level of concern from the standpoint of protecting the health of sensitive populations, such as the developing child (U.S. EPA, 1986, 1990; NRC, 1993). The basis for selecting the value of 10 μg/dl is based upon a combination of reviews by the Environmental Protection Agency, the Agency for Toxic Substances and Disease Registry, and the Centers for Disease Control. [see proposed rulemaking for 403 for details]

The purpose of the adult blood lead model is to predict PRGs (preliminary remediation goals) and not govern blood lead levels monitored in the workplace. The value of 10 μg/dl used in the model is based on the need to protect the health of children, and is therefore, used as a risk-based value in the model. Also, OSHA does not have a direct conflict with EPA's practice of its adult model for setting PRGs.

Pertinent memoranda are cited and/or included in the Appendices to the document.

10d General Response regarding Data Quality Objectives Guidance: *Three EPA guidance documents on data sampling and analysis were not cited in the HHRA and comments questioned whether appropriate guidance had been followed. The three documents cited were: 1993, Data Quality Objectives (DQO) Process guidance; 1998, Data Quality Assessment (DQA) guidance; and 1992, Data Usability in Risk Assessment. All three guidance documents were followed and are discussed in varying degrees in the individual FSPAs and the RI/FS. The following is a brief summary of how these guidance documents were applied to the Baseline HHRA.*

A. The purpose of the 1994, Guidance for the Data Quality Objectives Process is to provide general guidance to organizations on developing data quality criteria and performance specifications for decision making. The DQO process is a strategic planning approach that provides a systematic procedure for defining the criteria that a data collection design should satisfy, including when, where, and how many samples to collect and the tolerable level of decision errors for the study. The seven step DQO process was considered and documented in the Draft Technical Work Plan (URS Greiner and CH2M HILL 1998), and considered further and documented in varying degrees in the individual FSPAs developed from 1997 through 2000. Each FSPA and USGS task was developed to address specific data gaps identified after reviewing available historical data and results of previous sampling and analysis efforts. The purpose of each data collection effort was to investigate areas potentially impacted by mining related activities. Due to the large geographic extent of the study area, it was not possible to fully characterize all areas. As all data gaps were not addressed, subsequent studies of specific areas identified for remedial actions may be needed to support remedial design efforts.

B. The DQA Process is a comparison of the implemented sampling approach and the resulting analytical data against the sampling and data quality requirements specified by the DQOs.

Results of the DQA are used to determine whether data are of adequate quality and quantity to support the decision-making process. The data quality assessment performed for this study includes evaluation of the quality of the analytical data generated for each of the field sampling efforts and evaluation of the adequacy of the data set in meeting the intended data uses. To provide a high level of quality for the analytical data collected during this study, samples were submitted to commercial laboratories or to EPA for analysis using the EPAs contract laboratory program (CLP) methods or the EPA SW-846 methods. High quality is maintained in these programs through the use of on-site audits, performance evaluation samples, quarterly performance reports, fraud detection mechanisms, performance based scheduling and continuous inspection of laboratory data. Additionally, all analytical data were validated according to EPA data validation guidance (EPA 1994). Following validation, the data set was further reviewed for proper application of data qualifiers. Data identified during validation as being unacceptable for project uses were not carried forward in the assessment.

The sampling plans were designed to provide data to be used in determining if areas are impacted and to support risk assessment activities. Because all surveys have an associated measurement error and risk assessment requires a high degree of certainty, field sampling and analysis plans are designed with a known confidence level (95 or 99 percent) for the majority of common use area sampling. The majority of residential soil concentration data, however, was developed from pre-existing surveys and volunteer results from individual requests to be evaluated for emergency removal programs. These surveys did not have pre-specified confidence levels. The methodology employed was developed to support the 1996 IDHW/ATSDR exposure study conducted in the Basin. This methodology mirrored the sampling procedures used at the BHSS for the last two decades. These methods were developed in compliance with DQO and DQA requirements and have been extensively reviewed. Utilizing these data and the associated site-specific blood lead observations obtained through IDHW/ATSDR/PHD health response programs was a fundamental precept to conducting the risk assessment in parallel with the RI/FS. Comparisons of the results of the sampling methodologies combined to develop the data base supporting site-specific analysis is presented in Appendix N. Uncertainties associated with using the information generated in the earlier Basin investigations and combining data from different sampling efforts is discussed in Section 7 of the document.

Specifying confidence levels in advance of sampling is important when defining sites where it is difficult to determine if an area has been impacted by contamination (average concentration close to screening values). Where historical information clearly indicates areas are contaminated (average concentration much greater than screening value), specifying confidence intervals prior to sampling is not necessary because the probability of falsely characterizing the area are low. Residential soil lead levels in the Basin have been shown to be impacted and exhibit substantially higher lead concentrations than communities with similar demographic and socio-economic conditions. High blood lead levels among children has been documented and health response activities have been undertaken with respect to soil exposures. Subsequent sampling events have confirmed the initial assumption.

C. The 1992, Final Guidance for Data Usability in Risk Assessment provides practical guidance on how to obtain an appropriate level of quality of all environmental analytical data required for Superfund human health risk assessments. This guidance complements and builds upon other Agency guidance documents such as the 1989, Risk Assessment Guidance for Superfund (RAGS) and the DQO guidance mentioned above. The four data application questions requiring an answer for risk assessment from the 1992 guidance are as follows:
1. What contamination is present, and at what levels? - The extent of contamination in the Basin is addressed in Section 2 of the HHRA which describes sample collection methods, data analysis procedures (metals), and notes where samples were collected specifically for human health needs versus other uses. The vast majority of the data used in the HHRA was collected based on human health considerations and fulfills the requirements of risk assessment guidance described in RAGS and in the 1992 document. For the relatively small amount of data used that was not collected for HHRA use (sediment and surface water data in the South Fork, Canyon Creek, and Nine Mile Creek), the uncertainties associated with these data are discussed in both Section 2 and in Section 7 of the report. Other than the data noted above and the special case of waste piles, all samples were collected using a randomized or systematic sample design appropriate for risk assessment evaluations.
2. Are site concentrations different from background? -- Section 2 presents background concentrations for applicable media (except groundwater) and selected COPCs based on concentrations exceeding background levels and health levels.
3. Are all exposure pathways identified and examined? -- Exposure pathways were discussed and conceptual site models by human health geographic areas were presented in Section 3.
4. Are all exposure areas fully characterized? - Human health exposure areas were discussed in Section 3. However, they were not explicitly defined in many cases due to the large and complex area of the Basin. Additional data may be required to support remedial design and remedial action activities on a site-by-site basis for individual sub-areas of the Basin.

11 Comments related to the Adult Exposures to Lead in Soil Model: Several comments were received pertaining to uncertainty associated with using default values in the model. Other comments regarding the Adult Lead Methodology (ALM) suggested that data supporting the GSD for adults is limited and will result in overestimating risk. Comments also indicated that the 12% absorption default value used in the model should be lowered. A few comments also questioned the blood lead value of 10 μg/dl used in the model when OSHA monitors for blood leads of 30 μg/dl in pregnant woman working in lead contaminated areas. The TRW review also provided comments regarding the uncertainty associated using the ALM for infrequent exposure periods, the averaging time used, the soil weighting factor, and explorations into the use of site-specific blood lead levels.

11a General Response Regarding Adult Exposures to Lead in Soil Model: *The TRW recommends that the ALM not be used for exposure frequencies less than 3 months. The CT occupational exposure scenario assumes a two month construction period where the EF is 8.7 weeks per year for 5 days a week. This EF is the only scenario not meeting ALM criteria and will result in greater uncertainty, possibly overestimating risk. However, the averaging time*

used in the ALM was for exposure over a 365-day period instead of over the exposure duration, which would result in higher PRGs, or underestimating risk. The averaging time of a 365-day period was used for all (i.e., both occupational and the 4 recreational) scenarios. As a result, for the recreational and RME occupational scenarios, risk may be underestimated. The TRW also suggested to explore alternative assumptions for baseline blood lead input to the model, given the blood lead data collected at the site. The guidance to the ALM states, "Ideally, the value(s) for PbB(adult, 0) used in the methodology should be estimated in the population of concern at the site. This requires data on blood lead concentrations in a representative sample of the adult women who are not exposed to nonresidential soil or soil-derived dust at the site...The sample must be of sufficient size to yield statistically meaningful estimates of PbB(adult, 0)" (TRW 1996). The geometric mean female adult blood lead level was 2.0 μg/dl and ranged in the 8 geographic areas from 1.6-2.6 μg/dl. If the scenarios were applied to each geographic area as was done for children in the IEUBK, the total number of samples from each area was not of sufficient size (n=12-41, see Table 6-8b) to yield statistically meaningful estimates able to change the default value. A national value of 1.7 μg/dl was used in the risk estimates. If the Basin-wide geometric mean of 2.0 μg/dl were applied, higher blood lead levels and lower PRGs would be estimated. As a result, risk managers may consider risk somewhat underestimated for all but the short-term construction scenario. The TRW also pointed out that a soil weighting factor of 1 was used in equation 2 of the ALM (Tables 6-57 through 6-60b) and the HHRA agrees that the two approaches do not differ. These columns in Tables 6-57 through 6-60b will be deleted in the final document.

11b General Response Regarding Default Values: *One comment stated that the model uses all default values, resulting in high uncertainty. The HHRA actually uses site-specific exposure frequencies and soil ingestion rates for consistency with the non-lead portion of the document. Blood lead values (as described in 11a) were not changed because site-specific values were not significantly different. The uncertainty associated with the model is discussed in Section 7.4 of the HHRA. One comment suggested that the default GSD value is estimated from limited adult data and will overestimate risk. The default GSD values used in the ALM are 1.8-2.1. Site-specific GSD values range from 1.6-2.2 in the geographic areas, and are not dissimilar to the default values. One commenter suggested that the infrequent exposure periods would increase uncertainty and overestimate risk. As discussed in 11a, the HHRA agrees that uncertainty may be increased, but there are other factors used in the model that tend to underestimate risk.*

Finally, there were a few comments regarding the use of the default value of 10 μg/dl in the model versus the OSHA standard of 30 μg/dl. The OSHA standard reads as follows:

> *D. "Permissible Exposure": The permissible exposure limit (PEL) set by the standard is 50 micrograms of lead per cubic meter of air (50 μg/m(3)), averaged over an 8-hour workday.*
>
> *E. "Action Level": The interim final standard establishes an action level of 30 micrograms of lead per cubic meter of air (30 μg/m(3)), averaged over an 8-hour workday. The action level triggers several ancillary provisions of the standard such as exposure monitoring, medical surveillance, and training. (3) "Health protection goals of*

the standard". Prevention of adverse health effects for most workers from exposure to lead throughout a working lifetime requires that a worker's blood lead level (BLL, also expressed as PbB) be maintained at or below forty micrograms per deciliter of whole blood (40 µg/dl). The blood lead levels of workers (both male and female workers) who intend to have children should be maintained below 30 µg/dl to minimize adverse reproductive health effects to the parents and to the developing fetus. [57 FR 26627, May 4, 1993, as amended at 58 FR 34218, June 24, 1993]

OSHA states that "the blood lead levels of workers (both male and female workers) who intend to have children should be maintained below 30 µg/dl to minimize adverse reproductive health effects to the parents and to the developing fetus." However OSHA then goes on to say that "There is a wide variability of individual response to lead, thus it is difficult to say that a particular BLL in a given person will cause a particular effect. BLL measurements show the amount of lead circulating in your blood stream, but do not give any information about the amount of lead stored in your various tissues. BLL measurements merely show current absorption of lead, not the effect that lead is having on your body or the effects that past lead exposure may have already caused." [57 FR 26627, May 4, 1993, as amended at 58 FR 34218, June 24, 1993]

The 10 µg/dl value is a default value used in the Adult Blood Lead Model recommended by the Technical Review Workgroup for Lead (TRW). The basis for using 10 µg/dl is that fetuses and neonates are a highly sensitive population with respect to the adverse effects of lead on development, and 10 µg/dl is considered to be a blood lead level of concern from the standpoint of protecting the health of sensitive populations, such as the developing child (U.S. EPA, 1986, 1990; NRC, 1993).

The purpose of the adult blood lead model is to predict PRGs (preliminary remediation goals) and not govern blood lead levels monitored in the workplace. The value of 10 µg/dl used in the model is based on the CDC guidelines to protect the health of children, and is therefore, used as a risk-based value in the model. The purpose of the OSHA blood lead value is to protect workers at Brownfield sites. Also, OSHA does not have a direct conflict with EPA's practice of its adult model for setting PRGs. See also General Response to Comment # 10c.

SECTION 3.0
DR. PAUL MUSHAK'S RESPONSES TO COMMENTS

PB Associates

Memorandum

From: Paul Mushak

To: Sharon Quiring, Sean Sheldrake, Marc Stifelman, Ian von Lindern

Date: 12/15/00

Re: Part 1 Responses: TRW, Dr. Coomes

Part 1: Responses to Public and EPA-TRW Comments on the Draft HHRA

Paul Mushak, Ph.D.

Responses to comments on the draft HHRA are given below and are organized by each commenting group. General responses to the commenter's statements, criticisms and concerns are provided as well as specific responses following the sequence in the comment documents.

I. EPA's TRW for Lead Comments on the Public Draft HHRA

EPA's TRW for Lead review has been extremely helpful and constructive to the purposes and thoroughness of the draft HHRA. The authors should give serious thought to using some of the suggestions. I would, however, like to note the need for elaboration on one matter that the TRW had not adequately dealt with.

The incremental lead intakes portion of the HHRA, to include risk characterizations associated with incremental intakes and Pb-B increases therefrom, appear in TRW, Sec. 2.5. pp. 20-21. The TRW correctly states, 1st full par., p. 21, that the method of incremental risk estimation done in the HHRA underestimates incremental risk because of how the calculations were done. This TRW concern is not only valid but requires further elaboration.

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As the principal co-author of the Introduction and Lead portions of Ch. 7, the uncertainty chapter, I would like to have the following paragraphs added to the addendum of changes in Ch. 7. The best place for addition would be Sec. 7.4.4., p. 7-42, Incremental Blood Levels

"The means by which we first estimate baseline residential scenario blood leads and then use these estimates in combination with runs for incremental, non residential lead intakes in the IEUBK model runs can drive potential differences in the magnitude of the resulting incremental increase in children's blood lead levels and distributions. These are further dependent on methodology when there is remediation of the residential but not those community lead sources that provide the Pb-B increment. The authors estimate incremental intakes of lead and any associated Pb-B increases by looking at the difference in the estimates for combined residential/baseline plus additional (non-baseline) intakes, minus the residential baseline. However, the pre-remediation incremental amount of Pb-B derived in this way (total intake - background) can be different from, and in fact lower than, the incremental amount that would be estimated post-residential remediation. The overall impact of incremental sources outside the child's home can be demonstrably attenuated, that is, underestimated in the modeling, when one looks only at combined significant intakes before any clean-up actions.

"The general biokinetic curve depicting Pb-B versus daily lead intake, or versus such other surrogate independent measures as level of lead in some medium such as soil, is curvilinear downward across a broad intake range (USEPA, 1986a, Ch. 10). As the intake increases, the relative incremental increase across the spectrum in Pb-B for an identical added intake becomes less and less. Because the model is constructed to reflect this curvilinearity, combining any high baseline lead intakes in the residential scenarios with an incremental intake, i.e., adding incremental lead intakes to those already far up the curve of Pb-B vs. Pb intake, would obviously underestimate what that incremental intake might produce in Pb-B if it had actually entered the simulations further down the curve of Pb-B versus total lead intake and in the steeper slope (more rectilinear) portion of the Pb-B vs. intake curve.

"What this means, in essence, is that one can't accurately quantify any residual risk to children after residences but not community lead sources are cleaned up if one estimates contributions of such lead to Pb-B by combining residential and non-residential contributions prior to remediation, especially if residence contamination is already significant. A more accurate depiction of that "incremental" contribution to Pb-B and therefore remaining child health risk after residential remediation would be provided by adding that lower range of soil leads likely to encompass any clean-up level for the residence to whatever the incremental lead intakes are.

"These comparisons can be depicted using several simple IEUBK runs for children 0-84 months of age. In runs with all parameters at default settings except the resulting soil and dust lead (using the default dust Pb = 0.7 x soil Pb), the incremental Pb intakes were entered via the alternate source setting and with a bioavailability of 30%. One can then compare the "% to exceed 10 µg/dl" using various simulations with and without incremental Pb intakes.

"For an incremental daily lead intake of 100 µg/d in both cases and using residential, baseline soils at either a pre-cleanup value of 2000 ppm or a value at 400 ppm which might lie within a clean-up range, there is a sizeable difference in "% to exceed 10" for children 0-84 mos. The high, baseline soil lead-associated Pb-B exceedence rate is increased only 12.3/100 children with a 100 µg/d increment, while the corresponding exceedence level at 400 ppm of baseline soil is 57.2%. That is, almost five-fold more children can be seen to exceed the 10 unit cut-off at the lower vs. the higher soil lead baseline. This is because the Pb-B vs. Pb intake curve is rectilinear and steeper at Pb intakes linked to a soil Pb value of 400 ppm than further up the curve, at 2000 ppm."

A second suggestion made by TRW that should be followed would be to use some sensitivity analysis runs for the various intake parameters, specifically the intake and the bioavailability runs, in the respective parts of Ch. 7.4.4., anywhere in pp. 7-39 et seq. These runs would be useful in resolving for the interested reader whether uncertainty in some model parameter is or is not a major matter. That is, critical comments from readers of the public draft may or may not be justified in critiquing uses of parameters that don't really make major impacts on the estimated Pb-B outputs, as G.M. values and/or distributions. To avoid hassles with new tables and figures, summary text describing sensitivity analysis inputs/results can be used.

II. C.A.R.T. Comments on the Public Draft HHRA.

It is my understanding that all of the C.A.R.T. comments are being collectively responded to by EPA Region 10's counsel and that I do not need to respond to these in any way.

III. Dr. R.M. Coomes/Basin Community Comments on the Public Draft HHRA

I offer both general and specific responses to Dr. Coomes' comments on the public draft of the HHRA. Based on his report's cover, Dr. Coomes represents the collective positions of various Panhandle Idaho communities in the Basin: City of Coeur d' Alene, City of Harrison, City of Post Falls, and Kootenai County, Idaho. Hereafter, these comments are headed as "Dr. Coomes."

IIIA. General Responses to Dr. Coomes' Comments

Overall, I found the general and detailed comments of Dr. Coomes to be a very mixed bag of criticisms. In some places within his submission, comments appeared to be appropriate or had at least superficial plausibility. However, in most of the other portions, his comments were confused and confusing, being quite vague and problematic as to their accuracy and comprehension in addressing many of the main issues. His comments were often misleading in terms of taking material out of context. For example, the geostatistical sampling minimum — seven versus ten — was covered in the HHRA, but Dr. Coomes' comments suggest that there is an unexplained inconsistency. He also drew distinctions among matters in the HHRA that appeared to pose little or no epidemiological or toxicological difference.

Especially troubling, he does not cite scientific underpinning for his criticisms and assertions, and no references are provided to back up his conclusions. This largely voids his comments where they run opposite to known science and history, owing to the real need for clear substantiation.

Dr. Coomes applies generalities to specifics inappropriately. For example, he cites a Science Advisory Board report from 1993 that purports to show that one can protect children adequately against non-cancer effects of contaminants such as those found in the Basin by using a child/adult lifetime exposure approach rather than those for children alone.

However, that report is misused by the commenter in that the approach advocated is only generally endorsed in certain situations. In the case of the Basin, there are exceptions that call into question applicability of that recommendation. Furthermore, SAB reports need to be updated with current science. No SAB report, nor any other report for that matter, is immutable over time. The SAB report being referred to is dated, in that we now know that for a number of contaminants that affect children and adults, in terms of non-cancer effects, little protection is afforded by using chronic adult "safe" exposures. For example, the recent 1999 NAS arsenic report notes that children are a population of likely higher vulnerability to arsenic's toxic effects. They cited examples of this.

Arsenic is a major contaminant of concern in the Basin, and these vulnerabilities are not captured in an adequate way by inclusion of the current RfD arsenic value of 0.3 ug/kg-day assumed for lifetime protection from lifetime exposures. I refer Dr. Coomes to recent research from South America that shows statistically significant in-utero, developmental adverse impacts of maternally-ingested arsenic on fetal development and early infant survival rates.

Ref. Hopenhayn-Rich C, Browning SR, Hertz-Picciotto I, Ferreccio C, Peralta C, Gibb H. Chronic arsenic exposure and risk of infant mortality in two areas of Chile. Environ. Health Perspect. 108: 667-773 (2000).

In the case of the main driver for risk assessment in the Basin, lead, children are scientifically and administratively the only focus. The case of mercury, which can be biomethylated in the aquatic compartment to produce the potent neurodevelopmental toxicant, methylmercury, is one where there is little or no protective margin in the RfD for developmental toxicity in fetuses and the early infant; nor would these age bands benefit from any reference to adult lifetime "safe" levels.

His comments are of questionable substance in other places. For example, seemingly trivial inconsistencies are cited in isolation as though they are determinative for risk. Finally, his comments include erroneous statements because they are uninformed as to basic technical issues and their correct interpretation.

Statistical Analysis Issues

Areas in Coomes' comments that are particularly misleading, vague as to precision and even seemingly uninformed are his multiple critiques about the various statistical analyses results and interpretations that form a significant end product in the draft HHRA. These include but are not limited to a cluster of comments in his Executive Summary (e.g., p. 2, p. 5) dealing with the various degrees of statistical associations for relationships among various parameters measured and processed in the draft HHRA, with particular reference to how one characterizes these associations and to what uses they are to be put. First, Dr. Coomes offers vague comments to bolster an assertion that if and when "correlations" were below 0.5 in the HHRA, they were not "significant." A comment also is made about a lower correlation coefficient offering poor "predictability." He goes on to say "...predicting or explaining one value in terms of the other is the goal of correlation coefficients," followed by "A correlation coefficient less than 0.5 means that at least fifty percent of the variability in the data cannot be accounted for in the analysis." Collectively, his comments on statistical issues as used here strike the informed reviewer as unfocused, confused as to terminology, and erroneous as to validity of application to risk characterization portions of the HHRA. Consequently, his comments often cannot be taken as valid criticisms of statistical analyses done in this HHRA.

A coefficient, especially a regression coefficient, for some inter-variable relationship in a large database with many variables and with many inclusions of controls for confounding variables can be numerically small but still be highly statistically significant. This is how one does statistical association analysis, testing for statistical significance level regardless of the value of the coefficient. There are

examples of this in numerous articles on the environmental epidemiology of environmental contaminants, including metals, published in various open scientific journals. Those papers also make it clear that there is no magic number which an association has to reach or exceed in order to offer interpretive value. Secondly, it is not the case as he states that a “coefficient” less than 0.5 is not indicative of a "significant" association. Whether some value is or is not hinges not only on the level of statistical significance but also on the particular statistical design or statistical model being used.

For example, soil lead can impart its impact on lead on children's hands via various pathways, each having coefficients which depend on the model, e.g., the structural equation modeling approach for sorting out pathways. He indicates that a correlation coefficient (r) of 0.5 for an association means that 50% is unexplained and therefore the association is presumably meaningless for drawing inferences in this particular case of Basin lead contamination or other metal contamination. He apparently means the R-square statistic, i.e., explained variance via goodness-of-fit, not "r" per se. R^2 is the proportion of variance of the dependent variable y that can be directly explained by the variable x. Intrinsic in this, furthermore, is the overall existence of a regression line, not a simple correlation line. Dr. Coomes appears to invite the inference by the reader that other factors are therefore more important than lead. It is also curious that a number of the expressions of explained variance in the regression models that were used actually exceeded 50%. It's not even the case that all sections in the statistical results portion are at issue.

In the typical practice in epidemiological studies with complex biostatistical components, even very good associations in population studies can be less than "0.5", especially if the particular association being tested has been over-controlled for confounders that subsume within their controlling an environmental lead component. Anyone familiar with the environmental epidemiology of lead exposures of risk populations would know this. That is, if the remaining "0.5" has within it a basic association with the principal variable as well, then the crude association understates the overall contribution to the endpoint. Furthermore, the authors of the HHRA recognize in their statements on p. 6-21, Sec. 6.4.2, 3rd par. that explained variance in their Basin regression analyses are high for site studies of the type done in the Basin, and including earlier assessment of the BH communities "in the Box." The latter comparison is interesting since no one would deny that communities in the Box have their lead exposures clearly linked to the historical extractive industry lead contamination.

Dr. Coomes also seems to indicate his confusion about the difference between a correlation coefficient and a regression coefficient (see above my comments on the R-squared parameter). He states (see above quote) that predictions are the goal of correlation coefficients!! Correlation coefficients do not have predictive function; regression coefficients do. Many of the inferential statistical

analyses carried out by the authors of the HHRA as well as by authors of the 1996 Idaho/ATSDR study have to do with regression analysis, including multi-regression analysis. For example, Sec. 6.4.1, pp. 6-20,6-21 in the HHRA sets forth some correlation matrices that have as their only purpose providing some crude idea of what associations are valid candidates for regression analysis. Sec. 6.4.2 deals with regression analyses themselves, the major analyses done for the HHRA draft report.

A regression coefficient from some regression analysis equation has predictive value, since the independent variable is clearly established in the empirical statistical relationship, thereby fixing the direction of a potentially causal relationship, and is expected to generally predict the dependent outcome for some value of the independent variable in any given set of circumstances. The size of the regression coefficient can be low and still have predictive value when there is statistical significance. The larger the study population and the larger the complexity of the study design, the higher the likelihood of more modest regression coefficients.

I refer Dr. Coomes to the various NHANES-type studies carried out over the years that include regression associations of lead in blood with such outcomes as systolic and diastolic blood pressure and blood biochemistry biomarkers of vasoactivity and kidney function. These regression coefficients seen in peer-reviewed reports from the NHANES databases are quite modest but significantly predictive. They are also universally accepted in the informed public health community as demonstrating a causal relationship for cardiovascular and cerebrovascular morbidity and mortality. As an added example, one can have a modest regression coefficient for a statistically significant effect on blood lead of lead intake from soil ingestion when applied in a regression analysis to hundreds of subjects in a human study population. We would also say that ingested lead from lead-contaminated soil causes elevated Pb-B; elevated Pb-B does not cause elevated soil lead. That is, the direction of potentially causal relationship when there is regression analysis of environmental lead as the independent variable in environmental epidemiological relationships is always unidirectional.

By contrast, a correlation coefficient has no predictive value since it merely depicts the relationship between two sets of data, neither of which need have a causal relationship to the other. Both sets can be viewed as being the "dependent" variable. Statisticians differentiate between the two by describing a regression relationship as being unsymmetrical and correlations as being symmetrical. That is, in regression analysis we observe the change in one variable as the other is permitted to change. For example, when a laboratory tests a new method for lead measurement, it is common practice to run results of the new method for a set of samples with data for the same samples using an established or "reference" method. Here, one derives a simple correlation coefficient, one which would have to be quite high in order to support adoption of the new methodology as being as

accurate and precise as the method being used for reference. However, we don't say that the reference method has a potentially causal, independent relationship to the new method. The reference numbers say what they say as test results for any cluster of tested samples, and the method being tested likewise says what it says. How well the new method says what it in fact should say is assessed in the correlation analysis. A second correlation relationship example often encountered in lead epidemiology and toxicology is the level of lead in various experimental animal or human post-mortem tissues resulting from lead exposure. For example, lead exposure simultaneously elevates the levels of lead in both kidney and in liver, causing a significant correlation between kidney lead levels and liver lead levels. We do not speak of kidney lead causing liver lead or vice-versa. Nor do we say that the elevations track each other such that one closely "predicts" the other.

A persisting confusion between regression and correlation, cause and effect and basic mechanistic principles of lead exposure in risk populations appears to also occupy most of p. 5 of Dr. Coomes's Executive Summary. This portion dealt with modeled Pb-B levels, including a bizarre discussion of predicted vs. measured Pb-B levels. But it's not clear what the basis or the end result of his confusion is. The outcome seems to be that he holds any good agreement between modeled Pb-Bs via the IEUBK model and measured Pb-Bs as being due to chance. Making matters worse, he appears to have set up an irrelevant straw issue over the relationship of predicted versus measured Pb-B levels in terms of concordance and then proceeded to try to demolish that straw issue by saying there's no necessary cause-effect here by illustrating with an irrelevant example, the price of rum in Havana vs. salaries of ministers in Massachusetts.

This section of the Executive Summary is largely uninformed and meaningless to any serious dialogue between a reviewer and a reviewed work. No one is saying that there's anything more to the predicted vs. observed relationships than that compelled by known causative or other relationships between environmental lead intakes and predicted blood lead, especially when taken in tandem with causative relationships between environmental lead intakes and predicted blood lead. The set of source-pathway relationships to child exposure are depicted in Figure 6-6 in the draft HHRA.

There is an enormous global literature that is definitive and formed the basis of constructing the IEUBK model. This vast database compels a single conclusion of lead intake increasing blood lead, and arises from a huge literature for lead epidemiology in children, numerous compelling experimental animal results, and equally numerous biomolecular mechanistic studies showing lead to be a potent childhood poison, operating through its entry to and exit from blood. That is, these are toxic effects of lead exposure via lead intake from sources that operate through well-established dose-response relationships. I refer Dr. Coomes to any of many authoritative expert consensus documents, including the 1993 NAS/NRC study on

lead exposure, the several CDC Statements on childhood lead poisoning, EPA's 4-volume compendium and peer-reviewed document, and the 1995 World Health Organization criteria document on inorganic lead. Lead sources produce lead exposure in children and this exposure manifests itself in various body compartments which not only lend themselves to measurement, e.g., Pb-B, but also show quite tight dose-response relationships to childhood toxicity.

The IEUBK model is called a mechanistic, biokinetic model for the simple reason that the model takes lead sources known to be significant contributors to child exposure and predictors of children's blood lead and processes them by in-vivo kinetic processes also well known in the open scientific literature. That is, when children are in a highly lead-contaminated setting, they will sustain lead exposure, with resulting elevation in a reliable biomarker for lead, Pb-B. One can assess this contribution of intake lead via various sources with a model or one can take measured blood lead levels and do inferential statistics to show what in fact these various sources and pathways contribute to Pb-B. Alternatively, by use of a well-validated model, one can input known sources and pathways of lead and generate Pb-B G.M.s and Pb-B distributions. The IEUBK model has been validated and has also been calibrated against a large number of site-specific applications. When we look for concordance between measured and modeled Pb-Bs for children at some Superfund or other site, we are likely not dealing with chance associations or spurious relationships. I find these particular comments of Coomes' in his ExSum to be quite remarkable.

Uses of Available Data With Reference to Their Diversity/Similarity

It is not clear that Dr. Coomes readily follows the uses of various lead-containing media for statistical analysis. In various paragraphs in the Executive Summary, for example, he repeatedly objects to the combining of data sets. This issue of combining data sets was heavily thrashed out in task group discussions as well as in the text of the HHRA. Again, it strikes me that either he has not read the document carefully or does not recognize answers to questions in his review already present in the HHRA. These criticisms do not merit serious response.

There are eight different areas identified for detailed assessment within the overall Basin, each of which was isolated for discrete statistical analyses. It is not true, as implied by Dr. Coomes, that the HHRA is trying to make a global statement about the Basin. The document makes it clear that there is considerable variability as to some parameters. That's why the HHRA deals with eight areas. In some cases, sediment/soil figured more heavily for typically defined soils, while in the Lower Basin there are no clear demarcations between what's sediment and what's soil. In addition, suspended or settled sediment under water is treated as an additional medium for lead exposure. So long as mixed soils and sediments have

similar bioavailability, and intakes adjusted appropriately, it's not clear what the problem is.

Dr. Coomes' Comments on Sampling and Sampling Design Issues.

Dr. Coomes appears to misconstrue the caveats in EPA guidance for screening data not being permissible for baseline risk assessment with what's in the HHRA. What exactly does he mean, when we look at the criticism closely? Secondly, a number of the analyses in terms of their sampling geostatistical design were not, technically speaking, "screenings." That is, we are not uniformly dealing with screening-level data transformed somehow into more refined evaluations. There also seems to be some confusion underlying Dr. Coomes' comments as to what's a screening and what's a survey. He appears to refer to the expedited screening done for common-use areas. For example, EPA's RAGS, Vol. 1, HHEM, 1989, describes on pp. 4-20 the nature and valid uses of field screening analyses quite clearly and notes what is to be done to do further assessment.

He is also vague on the point of what are valid statistical analyses for data sets. For example, he notes on p. 2, ExSum that environmental dust data sets, i.e., 83 and 74 soil, mat, and house dust data points, are identified without apparent reason in the HHRA as not being amenable to statistical analysis. First, it's not clear what's precisely being referred to in terms of cited text of the HHRA. He may mean non-lead contaminants measured late in the 1996 to 1999 data pathway projects. The mat dust medium is itself a house dust contaminant measure, either as dust lead loading or dust lead concentration. The second measure is vacuum bag dust. The mat and vacuum bag dust is analyzed for lead in Sec. 6.4 in terms of concentration and lead loading, i.e., amount of lead per unit area measure. What is the discrete mention made of "dust" and how different from "mat" dust?

The HHRA makes ample use of statistical analyses for lead in dust pathways. I would note that, unlike lead, the soil-dust relationships for other contaminants occurring at sites is not well understood or characterized. Nor is Dr. Coomes accurate in implying that soil-dust contaminant relationships have been well studied at other sites in the case of non-lead metals and metalloids. They have not been. Some few other sites where this was attempted for arsenic, cadmium, etc. support the notion that we have a way to go for non-lead elements. One issue is that of standardizing what type of dust collection is appropriate. We have a good idea of what type is appropriate for lead.

Misstatements and Erroneous Statements

Examples of misstatements and erroneous statements by the commenter are present throughout the ExSum and the detailed comments. A good example is

found in the ExSum discussion of samplings of, and on, the waste piles. First, there could not be a ready random selection of the waste piles within the entire Basin since they were heterogeneously distributed spatially and in terms of their relative accessibility to children. Those of the eight areas further up the Basin were more problematic for waste pile exposures. These especially included tailing ponds and waste piles such as those at Burke/Nine Mile.

Secondly, owing to apparent misreading of the general literature and EPA guidance on waste piles and specific assessment discussion of waste piles by HHRA authors, Dr. Coomes' comments are not tenable. First, EPA guidance would treat a waste pile as a "hot spot" for sampling assessment. Waste piles typically have the highest levels of lead and other contaminants when compared to other media. Such piles include waste in the form of tailings, weathered/weathering slags, disintegrating mine overburden, etc. EPA's 1989 RAGS document treats hot spots in a prescribed way. Hot spot discussions are contained in RAGS at pp. 4-10 to 4-12, 4-17, 4-19, 5-27, 6-24 and 6-28. On p. 6-28, last par., left column:

"In some cases, contamination may be unevenly distributed across a site, resulting in hot spots (areas of high contamination relative to other areas of the site). If a hot spot is located near an area which, because of site or population characteristics, is visited or used more frequently, exposure to the hot spot should be assessed separately."

This says that assessment is to be site-specific and focused on the specifics of the interactions occurring between human receptors and the waste pile. Furthermore, one does not merely test the top inch or so of material at an extractive industry waste pile. There is a simple reason for this, but one that appears to have escaped the commenter. That is, children who are infants and toddlers will only typically encounter the surface of contaminated materials, such as their home play area soils. With waste piles, we don't have infants and toddlers crawling up, over and down waste pile surfaces and engaging in mouthing activity. Even somewhat older children are not interacting with waste piles in this behavioral fashion. What we have are older, mobile children interacting with waste piles in diverse ways, such as peddling their bikes on the piles, running up and down and, thereby breaking through the surface and contacting deeper strata in doing so. They would likely take their pets with them, providing another recognized vehicle for picking up waste particles at varying depth. The HHRA was correct in taking into account deeper sampling depths for piles, although its reasons for doing so differ from the above rationale.

IIIB. Specific Responses to Dr. Coomes' Comments

Executive Summary

p. 1, par. 1 This comment in the last sentence is too vague to respond to. The HHRA draft makes it clear that there is a bit of a structural distinction between CSM models 1-5 and the eight areas in the Basin selected because of human vs. ecological impact. However, as noted throughout early parts of the HHRA, the rationale for overlays of FSAP protocols with the CSMs do not actually make it difficult to merge the two approaches. Given the vagueness of the comment, and its marginal value even if clarified, it ought to be minimally responded to or not accepted.

Ibid., par. 2 Again, too much ambiguity to be useful for response or to be a candidate for acceptance. What exactly in the FSAPs or the early parts of the HHRA dealing with data sets selected for the HHRA refer to the inadvisability of combining data? He does not elaborate. The topic area Dr. Coomes refers to is really that of multimedia exposures, and multimedia exposures that reflect the highest likelihood of occurrence for the most vulnerable child receptor away from the residence, i.e., the more mobile child, 4-11 years old. The 4-11-year-old child is still into oral exploratory behavior at the younger end of the range, 4 and 5 years old, so as to consider soil surface contact with contaminant levels but mobile enough at the older age end of the age band to show significant contact with sediment. How does Dr. Coomes make his point averring that the data for sediment vs. upland soil were not meant to be combined??

Ibid., par. 3 This paragraph can be rejected as a non-relevant criticism as to variability of contamination in the Basin somehow being such that one can't do Basin assessment. The commenter ignores the fact that the Basin was specifically divided into eight areal segments to minimize any problems of excessive heterogeneity frustrating geostatistical and exposure analyses. The problem was recognized and dealt with by the authors and the HHRA team before Dr. Coomes was provided a copy.

Ibid., par. 4 plus carryover, p. 2. This criticism is misleading or what EPA says is misunderstood in terms of EPA RAGS guidance. It is not valid and needs no response of any substance. See the previous example on this very matter in the above general comments. What's more, it's not clear that in every case, field "screenings," even when confused with Basin surveys as occurred here with Dr. Coomes' interpretation, automatically require prescribed added assessment. The actual nature of the samplings and their statistical nature determine what, if anything further, needs to be done to do a "baseline risk assessment."

p. 2, 1st full par. This comment was dealt with as an example in my general response provided earlier. In light of that response, and given any citation for which SAB report he could possibly be referring to, the comment requires rejection with my explanation provided.

Ibid, 2nd par. This can arguably be rejected. I note responses to this in my earlier general responses.

Ibid, 3rd par. This par. has a number of confused and confusing, seemingly erroneous, statements. There's little to respond to and it cannot be accepted. See my multiple responses above in the general responses.

Ibid, 4th, 5th par. Cannot be accepted for reasons given already in my general responses.

Ibid, last par. & onto p. 3, top It is my interpretation of the eight different areas that the residential, baseline scenario for the segment is for baseline, not incremental, scenarios. Construction workers would fall under the latter. Dr. Coomes may have some problem with what's residential and what's incremental in each of the eight areas. Response to this by the HHRA authors should clarify the distinction to prevent any confusion.

p. 3, 1st full par. This comment is not tenable owing to ambiguity and unsubstantiated evidence for the seasonal band April through November being too broad. The authors may wish to emphasize the range in temperature and other climate conditions that justify the number of months selected for minimal-clothing, dermal exposures.

Ibid, par. 2 I really don't understand this comment. It would be difficult to respond to, since its point is unclear. The exposure point concentrations and the applicability of these values for the different clearly identified risk groups are spelled out as to specific areas in terms of both current and plausible future activity or future site use scenarios. The HHRA in several sections of Ch. 5 and such parts of Ch. 6 as Sec. 6.5 estimates, via multiple tables, various scenario lead and non-lead levels. For example, Dr. Coomes seems to believe that if there's no current construction activity going on in any of the eight areal segments in the Basin for assessment of residential/neighborhood baseline and incremental occupational scenarios then they should not be included. I see in this par. a misunderstanding of a baseline HHRA as being open-ended as to time frames. That is, future scenarios as well as current ones are the purpose of the HHRA.

Ibid, Par. 3 I suggest that the authors of the HHRA consider what he is claiming and they respond to it. Something does not make sense here. For one thing, are

these in reference to the lead, non-lead, or both portions as captured in Ch. 3 and later sections?

Ibid, last par. & follow on to p. 4 The comment is quite vague as to what is its meaning for contaminant frequency of exposure. Of course, every time a young child or even older child mouths his fingers or otherwise ingests soil, there would be dermal contact preceding ingestion. However, this is not what one quantifies in dermal exposure. The small surface area of the child's hands and the histological composition of finger/palm epidermis makes applicability of dermal exposure here of little consequence. Dermal exposures can occur more frequently than soil ingestion within the meaning of both the exposure factors recommendations of EPA in its Exposure Factors Handbook, 1997 version, and the various scenarios for contact in the three settings identified by Dr. Coomes. Dermal exposure in swimming is higher and more frequent as to occurrence in those older receptors where the frequency of actual incidental ingestion of soil is inverse, i.e., dermal exposures for a number of activities occur more frequently and involve more body surface area with age and less mouthing or incidental ingestion.

p. 4, 1st full par. This par. does not require acceptance because its premise is flawed. In the sections in Ch. 4 and Ch. 5 dealing with arsenic, for example, there is a discussion of the range of cancer risks as a preliminary cancer risk goal as well as some discussion of how high an individual estimate within that range need be before remediation. More to the point for a contaminant like arsenic with well-documented skin and internal cancer risks and with wide acceptance in regulatory quarters nationally and internationally as a very potent carcinogen in human populations, the notion that a risk less than E-4 does not require serious consideration for remediation action is a misstatement of EPA guidance and recommendations to risk managers.

Ibid, 2nd par. This par. so grossly misstates what the uses and limits of the IEUBK model are generally and for use by the authors of the HHRA that it cannot be seriously responded to. First, intermittent exposures of sufficient time to allow operation of steady-state lead kinetics in-vivo can be handled by the model. Intermittent exposures in the context used in the HHRA does not mean we are speaking here of acute exposures of a day or a week or so. Dr. Coomes is confusing acute exposures of several days or weeks with exposures that can be both intermittent but of sufficient length in those intermittent occurrences that they obey the requirement for steady-state kinetics to apply: for example, recreational exposures occurring in warmer seasons. That is, exposure is long enough for use of the IEUBK model even though unceasing, chronic exposure may not be occurring. I refer Dr. Coomes to the comments made by the TRW for lead, which I think will appear eventually as part of an Addendum to the public draft.

Ibid, 3rd. par. I can't respond to this since it is not clear precisely what he is referring to. Are the two day groups in reference to the lead and non-lead portions of the exposure factors sections in the HHRA? The IEUBK model selection of time intervals was clearly spelled out in the portion of Ch. 6 dealing with predicted children's and worker/adult Pb-B values.

Ibid, last par. Dr. Coomes seems to be setting up a straw issue here in terms of rigid protocols for DQOs and actual uses of data in a any particular risk assessment. This issue appeared to be addressed adequately for lead and non-lead sampling portions as applied to a BRA. I leave this particular matter to URS and TG data analysts to address the specifics of the three bullets as given in this par.

p. 5, all This page does not merit acceptance, because of its flaws of fact and interpretation of the huge database. A comprehensive response was provided to this criticism and its highly problematic nature in my earlier comments dealing with statistical analysis issues. That rebuttal also provided comments on the matter of what predicted Pb-B values using biokinetic models mean.

Responses to Dr. Coomes' Detailed Comments

p. 6 , first par. Dr. Coomes seems to have overlooked the fact that the CSMs were simply included for comparison purposes with the eight discrete demographic study areas within the Basin. This was not hard to do, and I in fact noted in a number of earlier review comments that outside readers will get things mixed up. That appears to have happened here. While the CSMs were developed for ecological assessments, this would not materially impact the HHRA since this HHRA focuses on the eight discrete demographic segments. One can simply read the HHRA's chapters and see that the entire assessment is done on the eight areas occupied by some level of human activity. It is not driven by theoretical constraints arising from original depictions of the CSMs. The latter do not even appear in the numerous tables and figures in the risk assessment portions of this HHRA. The criticism can be rejected as irrelevant.

Ibid, 1st bullet. It is not clear that this comment can be accepted as it was stated and as it would typically be interpreted. The first bullet presumes on the definition of chronicity of exposure in the EFH as applying here and also whether adjustment or normalizing for total seasonal or annual time exposures was factored into Dr. Coomes' critique or not. I do not believe the commenter has accurately characterized what went into coming up with the final daily times adjusted accordingly. The authors should, in their own responses to Coomes, spell out a repeat of the rationale and assumptions going into the time intervals of exposure.

Ibid, 2nd bullet. This comment is even murkier than the first bullet above. Somehow, we are to conclude from a sign and some vague assertion of available "evidence"

that there is current use. Again, it is not a necessary condition that there be actual present use, but that there can be use. Secondly, I don't see what's offered as substantiation for the criticism that it is tenable. The HHRA authors can respond as they wish in terms of accepting part of the comments.

p. 7, 1st full par. There is nothing in the 1989 RAGS document on field screening efforts that would say that the survey data for all but the expedited area is even at issue. As to the expedited screening level assessment for this one segment of the Basin, I leave it to the HHRA's URS-Greiner contractor authors, who would be clearly acting within EPA Region 10 guidance, as to whether the amount of exposure that needed to be "modified" was so substantially different from what was done that it required further assessment before any inclusion in the HHRA.

p. 7, bullet, Ch. 3 I have already responded to Dr. Coomes comment in the ExSum about SAB endorsement of using a combination child/adult approach. As I noted, this approach does not really provide protection from developmental effects of arsenic. In addition, there are concerns about neurobehavioral effects of As being more pronounced in childhood exposure.

p. 8, 1st bullet Sec. 3.2.2. only has a short paragraph on dermal uptake of COPCs, including a general statement on use of soluble vs. insoluble/partially insoluble substances and the statement that the tap water and surface water routes were not quantified in the HHRA. Dr. Coomes grossly misstates this short paragraph as "extensive discussion." He also misstates significance of the paragraph. That is, since no quantification occurred even though this pathway was a complete one, it is difficult to see any relevance to the remark or the usefulness of any required reconciliation with other sections.

Ibid, 2nd bullet He's correct in noting a cross-reference in error. The section at issue is 2.4.5, p. 2-17, subsection on Air, bottom of page.

Ibid, 3rd bullet. Dr. Coomes states that the HHRA deals with dermal absorption of metals as being more significant than stated in Sec. 3.2.3. Section 3.2.3 is correct as written. He does not identify what exactly is inconsistent with 3.2.3 text. The other sections on dermal exposures do provide details on dermal uptake, but it's not indicated that dermal uptake vs., say, oral ingestion, would be a major route. HHRA authors will wish to deal with this. I don't find the criticism substantiated and requiring acceptance.

Ibid, 4th bullet I thought the HHRA dealt with any inconsistencies with minimal samples to be taken for geostatistical adequacy in terms of confidence limits and representativeness. I leave response to the URS-Greiner contractor authors and Region 10 to sort out.

Ibid, last bullet See my general comments dealing with this issue. Again, it's not clear that the commenter is up on the literature for soil-dust relationships and those limitations for non-lead contaminants.

p. 9, 1st bullet It's not at all clear to me from what the HHRA says about uncertainty between yard soil and house dust, or from what the actual statistical analysis methods being used in the regression results in the HHRA indicate, that one has to use nonparametric testings to resolve uncertainty and variability. It is also not the case that nonparametric techniques are equal to the task for two data sets with complex association(s). The commenter recommends the Wilcoxon Rank Sum (WRS) non parametric test, basically equivalent to the Mann-Whitney (U) test. In fact, in his comment submission, he encloses such an analysis. The WRS is a nonparametric analogue of the t test for two independent sets of samples wherein one simply replaces actual measurement values with rank scores. This has the virtue, like all nonparametric approaches, of not shoehorning data into an assumption of normality to the distribution. It does, however, assume that both data sets have the same underlying distribution.

If one does so, however, one encounters limitations for the dust contaminant-soil contaminant relationship. In more complex statistical relationships, nonparametric testing becomes less efficient and less useful. What's more, the type of test suggested by the commenter is, like other nonparametric tests, mainly probing significance and not elaboration of confidence limits. Put differently, one does not resolve uncertainty in statistical relationships by using a quick-and-dirty nonparametric significance test that hides more uncertainty than it reveals.

I also believe he has mischaracterized what the HHRA is saying about variability and uncertainty in the soil-dust relationships across the eight areas. For example, the very dusty conditions at Burke/Nine Mile were recognized as possibly spelling somewhat different robustness of relationship than in others of the eight subareas. Also, the role of housing age and the level of severe deterioration were ascertained and the relative contribution of deterioration in lead-painted surfaces across the eight areas will affect the nature of distribution of dust lead vs. soil lead values. However, these area-specific differences have been examined for each of the areas.

Ibid, 2nd bullet The first sentence does not make any sense, and at best is a mischaracterization. Is he saying that it is somehow only because of uncertainty bounds in the analyses that the relationship of soil to dust becomes significant? And who is he saying has concluded this, he or the authors?? He then moves on to uncertainty as defined by EPA guidance. The detailed uncertainty discussion in Ch. 7 for both lead and non-lead contaminants seems to have been ignored. Based on vagueness and irrelevance, I would not accept the point of the statements, even when they are translated.

Ibid, 3rd bullet I agree that the term "exact" is misleading and in any event inappropriate. I would suggest deleting "exact" and substitute with "adequately characterizable" or similar wording.

Ibid, last bullet with carryover, top p. 10 This comment can be dismissed. I have rebutted it in my general comments above and the statements can be referred to.

p. 10, full bullet This is really a matter for URS-Greiner and TerraGraphics to respond to, as to details of sampling plans and QA/QC.

p. 11, to top of p. 12 The commenter seems to accord EPA guidance the status of compulsory adherence and to require cookbook approaches to any and all risk assessments without any professional judgment. That's not true! Whether stirring or not stirring sediment into a water column has prescriptive information in EPA guidance or not, it is a path of human exposure that makes quite good sense for the beach setting identified and the application of professional judgment. EPA does not hold out its guidance to be always inclusive of each and every conceivable exposure pathway scenario under the sun and applicable forever. Second, what EPA does or does not consider the "same" site is not defined by either EPA or the commenter for each and every possible pairing of samplings.

Dr. Coomes seems to be offering a booby trap, but a largely visible one. That is, he holds that EPA says that one has to sample something called the same site for all components of that mixed medium. However, if you do that, you are then overcounting. First, the mechanism for exposure to any intact sediment has little to do with orally ingested sediment particles in suspensions encountered by someone, say, wading and swimming in an area where a number of individuals are stirring up the water and where suspended particles become laterally mobile, especially for riparian or lateral lake beaches. The respective contact areal spreads are not identical. I considered the respective discussions in the HHRA on the above points to have been reasonably differentiating.

p. 12, 1st full bullet Dr. Coomes takes issue with the wording. He also asserts one cannot simply change the wording to solve the matter. How so, pray tell? Unless Dr. Coomes knows exactly what the authors meant in statistical terms as to what is their worst case, he can't second-guess whether one can revise or not. This comment is meaningless. I assume the original statement was more under the statistical rubric of a 95%-RME scenario, in which case worst case would be ambiguous. I leave it to the authors who drafted this portion to refine what they actually meant.

p. 12, 2nd bullet Dr. Coomes' caveat seems to have surface plausibility in the case where construction activities are simply confined to a highly localized intrusion into otherwise undisturbed topographical features within the Basin. But other scenarios

apply as well. I see some virtue in clarifying this text on p. 3-41 to respond to his concerns.

p. 12, 3rd bullet Dr. Coomes is concerned that while the small sampling may be o.k. for the residential baseline scenario, how does one characterize construction activity? I have several responses to that, but I will defer to the authors to respond as to the extent to which the actual likely construction areas in the Lower Basin are handled by the sampling data already in hand.

p. 12, last bullet I am not sure this quantity could be correct??? Without digging into the EFH in its 1997 update, the indicated average value of 71.8 kg for men and women versus the conventional ICRP Reference Man handbook values, with its 70 kg male adult and 50 kg female adult (for an average body mass of 60 kg) could not have changed that much. This figure should be checked for accuracy by the authors.

p. 13, 1st bullet The commenter takes exception to the time allocated for worker exposures. I don't see the stretch being made by him between the loss of population and any decreases in construction time. First, the loss in population is not equally distributed socioeconomically or demographically. Unless he can say that population trends affect that segment of likely future construction workers, then I see no merit. In fact, with the specific case of long-term, large-scale soil and other remediation efforts in the Basin having to draw on fewer workers, then by Coomes' logic, the exposure times would greatly increase, not decrease.

Ibid, 2nd bullet This is silly nitpicking on the part of Dr. Coomes, and is based on unfounded speculation offered in rebuttal about what people inadvertently ingest as water. I would ignore the comment. A volume of 30 ccs is a bit over an ounce of water or one-sixteenth of what a child splashes at another, i.e., a pint. This amount could easily and reasonably be imbibed with water play and vigorous splashing among children, and hardly requires submersion behavior for much of that hour.

Ibid, 3rd bullet Like the previous bullet, there is only undocumented speculation as to how cold ambient conditions get up and down the Basin on a monthly basis. The inference Dr. Coomes wishes to be drawn by the reader would be that kids would be tearing up bare feet on ice, not to mention one could never go about without being bundled up. I find it hard to accept this fanciful, if evocative, problem of kids freezing from April through November. What has Dr. Coomes evaluated among weather data and climatological distributions seasonally to make that kind of statement?

Ibid, 4th bullet Again, there is unfounded speculation about what would happen with particles adhering to skin post-beach play. Maybe people will be in the water, maybe they won't. The HHRA had to anticipate likely activities for all receptors

collectively, but one cannot say that some specific receptor engages in the same activities as all others. I do not find an adherence factor of 0.2 to be excessively protective in a risk assessment. For example, for the young infant, playing in beach waterline sediment/dry beach soil, the level of skin contact vs. body skin area contacting these media would be much higher than for older children, since the infant remains out of the water and involves more of his/her body surface area with medium contact.

Adherence is a function of particle size as well. To say 0.2 adherence is excessive is to say that not only will the particles not remain for any time, but that the fraction of small, adherable particles is less than 20% of the particle distribution, i.e., <20% with particles < 250 microns. This is not convincing.

Ibid, 5th bullet I agree here with Dr. Coomes. The actual percentile can be stated. The text already indicates that one can ingest up to that amount, so one might be led to believe that this %-ile is more like 99% than 95%.

Ibid, last bullet with carryover to p. 14 Dr. Coomes misrepresents or misstates what the relevance of the OSHA Pb-B limit is to protecting workers at brownfield sites. I believe Region X needs to be the main responder here. The practice of employers has been to remove women from the worksite as soon as they are known to be pregnant. Until the U.S. Supreme Court weighed in, in the Johnson Controls case in the early 1990s, it was assumed that pregnancy per se was the "medical removal" trigger, not some Pb-B value. Nor does OSHA have a direct conflict with EPA practice via its adult model for setting PRGs. EPA is exercising its area of legal authority. Dr. Coomes is referred to the 1996 document on the development, uses, and context for the adult Pb model when applied to worksite women of child-bearing age.

pp. 14-15 Dr. Coomes offers an unsubstantiated set of comments about his general problem or problems with the definitions and characterizations used for setting the exposure areas for the various receptors. For example, for children 4-11 years old, he notes that movement more than two miles from the residence is unlikely. What is his evidence for this? He repeatedly attempts to rebut risk parameters in this HHRA with undocumented speculations as to what is "likely" or "unlikely", or more appropriate, etc. This HHRA is not a dartboard for Dr. Coomes to toss a speculation wherever he wishes. I am inclined to ignore these many bullets focused primarily on dissecting and challenging the exposure factors portion of the HHRA. He offers little technical published material to support the comments.

p.15, Future Land Use This has little credence. It is not at all clear how current declines in one subset of the population, one among a number of risk populations, would materially color future land use considerations. The HHRA, furthermore, notes that the rate of decline has attenuated. What's more, there appears to be a

potential in-migration of the more high-risk segments of the child population: that is, those of low socioeconomic status and those also unaware of the extent of the exposure problems. The discussion in the commenter's submittal on the matter of groundwater seemed unfocused and, what's more, unsubstantiated. What evidence does Dr. Coomes have that there would be zero likelihood of side canyon development? The authors can weigh in here.

pp. 15 to 16, CSMs, 3-3 to 3-11 It seems that Dr. Coomes is unclear as to the fact that the CSM maps are provided for completeness, the populated areas within the eight geographic segments of the Basin being the spatial, demographic and environmental discriminator. The comments here reinforce my concern noted in several internal reviews that the overlapping of CSMs and geographic areas would create considerable confusion. The authors may wish to reinforce the relative status of the eight areas vs. the CSMs in the various maps in Ch. 3.

p. 16, Tables Ditto in the large table, Table 3.19, for comments offered above for CSM figures. Authors can respond as they wish. The authors also can respond to comments on Table 3-21.

Ibid, Comments on Table 3-22 The commenter has a point regarding how the frequency of tap water drinking would be different for any other residence-based exposure frequency as presently presented for CT and 95%-RME in Table 3-22. This should be clarified by the URS-Greiner and TG authors. As presented now, it appears tap water intake CT frequency is only 90%, 234 d/y, of the yard soil CT frequency, 260 d/y.

Ibid, last par. on age-based water intake rates The commenter is simply wrong about there being uniformity of water intake rates in children vs. adults. Children are well and widely known to consume water at a higher rate per some anthropometric value compared to adults. They therefore consume contaminants at a higher rate, regardless of how water needs are indexed. The caloric requirement per unit body weight is higher than adults, and water intake is linked to caloric requirements and physical activity. Generation of more water intake is also indicated by ventilation rate and oxygen intake requirements. Whatever the metric, kids have a higher water requirement.

I refer Dr. Coomes to the paper by Calderon and colleagues in a 1999 EHP article dealing with age-based water intakes in the U.S. and, because of this, increased arsenic intakes in children as a function of body mass. It shows U.S. children consume much more water than adults on a body mass basis.

Ref:

Calderon RL, Hudgens E, Le XC, Schreinmachers, Thomas DJ. Excretion of arsenic in urine as a function of exposure to arsenic in drinking water. Environ. Health Perspect. 107: 663-667 (1999).

I also refer Dr. Coomes to the EPA ODW/OGW 6/22/00 Federal Register notice [[65(121) FR 38888, 2000]], on proposed As MCL rulemaking, which includes statements that the early, bottle-feeding infant age band is a clear risk group for arsenic is because of the high daily water volume intake per body mass. These two citations and many others, such as the 1984 EPA health assessment document for arsenic, show that there is an inverse relationship between water volume intake and unit body index, e.g., kg body mass, such that the younger the individual, the higher the intake rate.

p. 18, Tables 3-23, 3-24 I responded to these issues earlier and one can refer to what's noted there.

Ibid, Sec. 5.0 comments on cancer risks for non-lead contaminants I have already responded to this assertion by the commenter and the responses can be found above.

p. 19, Section 6.0, 1st par. This concern about what is or is not intermittent exposure has already been addressed by me. See above general comments.

Ibid, Sec. 6.0, par. 2 onto p. 20 Dr. Coomes clearly is not aware that OSWER guidance for use of the IEUBK model does permit breaking out exposure modeling into, first, the residential unit and then incremental exposures. That is the whole basis of the uses of the IEUBK model in the HHRA and for the eight Basin areas. I refer Dr. Coomes to the OSWER directives on uses of the IEUBK model appearing in 1994 and with a confirmation of proper uses in August 1998.

p. 20, 1st full par. The HHRA authors should double check Dr. Coomes' calculations here to ascertain accuracy.

Ibid, Statistical Correlation I have already addressed the multiple flaws in Dr. Coomes' comments about statistical analyses in the general comments above.

p. 22, top, Summary Baseline... The rationale for use of the 4-year-old child can be provided by the authors. The HHRA authors can also address the following three bullets. The commenter statements are basically computational or editorial fine-tuning.

pp. 22-23, Recommendations I leave responses to recommendations to the authors, since the recommendations involve Coomes' views about what needs revising.

Dr. Coomes' Figures and Tables As I read these submissions, they can be easily addressed by the URS-Greiner and TG authors. I would note, however, several technical responses. First, the correlations between As and Pb, when we take into account the fact that these two elements can have differing vertical migration rates in soil over time, and that associations between elements presupposes some stability to the soil/sediment strata over time, appear convincing as to having the same geochemical emission origin. What's more, there is nothing in the Basin that would comprise an alternative source for As. As is not in interior or exterior paint, and there is no historical support for its use as an agrichemical in the Basin, etc. However, As is a common co-occurring element in extractive industry wastes.

Disturbances in surface soil strata from one geographical area to another and alterations that intrude to various depths, and have impacts with respect to various correlations, are understandable. They have some variability as to the degree of soil surface disturbance over the decades. Simple logic says that areas within the Basin that have more residential density or any agricultural uses over the decades will reflect different depth-linked ratios and concentrations than those minimally disturbed by various anthropogenic activities.

PB Associates

Memorandum

From: Paul Mushak

To: Sharon Quiring, Sean Sheldrake, Marc Stifelman, Ian von LIndern

Date: 12/15/00

Re: Part 2, Response to Public Comments

Part 2: Responses to Public Comments on the Draft HHRA

Paul Mushak, Ph.D.

I have general and specific responses to submissions for public comments on the draft HHRA. These comprise Part 2 of my responses, Part 1 having been submitted earlier, and address comments from the Shoshone Natural Resource Coalition (hereafter SNRC), the Lands Council, and the Coeur d' Alene Chamber of Commerce (hereafter CDACC).

IV. SNRC Comments

General and Specific Responses

The SNRC offers five general items, within which are a number of specific comments on the draft HHRA.

Item 1 The comments here are really more to the issue of views of economic development and the validity of different approaches to handling clean-ups in the Basin. As such, they are beyond the scope of the HHRA, its authors, or its advisors.

Item 2. Comments on Summary and Conclusions, p. 8-5 The set of questions indicates that the SNRC sees answers to its bullet questions about various sources of lead, especially amounts of paint lead vs. extractive industry lead being most hazardous to children. That is, those children requiring intervention should be looked at with reference to what's the most pressing source of child lead exposure. While these questions have plausible surface purpose, they ignore the simple fact that the nature of the health intervention program does not permit one to draw broad conclusions about the Basin itself. The sample size, N=50, is too small for meaningful statistical analysis, and the nature of the children being evaluated

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did not lend itself to specific inferential statistical analysis techniques. In brief, whatever the association of housing age or soil-dust lead specifics with the 50 children, it is not technically permissible to use any attempted analyses and draw Basin-wide conclusions. Such conclusions would be meaningless for the Basin.

Item 3. Comments on Risk Factors. The comments in Item 3 seem to indicate that the risk factors mentioned in the Item somehow get lead off the hook because it is purportedly those factors, not lead contamination, causing the problem. The comments have it backwards. Simply put, when there are risk factors that enhance lead exposure/poisoning problems in a community, there is every reason to take even more pains to minimize risks to child health by minimizing the extent of lead exposure by adequate lead remediation.

The commenters have a problem with use of a simple HHRA model to seemingly over-involve yard soil lead vs. other Pb sources. What's more, this is all foregone, in their view. The principal mode of assessing risk in the Basin is that dictated by EPA OSWER guidance, to not only attenuate the kind of questions arising with survey Pb-B data representativeness, but also open-ended applications for any future scenario. The authors of these comments appear to be unclear in their own minds as to what is the best approach for assessing risk, and the best approach for risk assessment that helps risk managers taking the long view.

In addition, the letter writer misstates what went on in the HHRA. The HHRA used yard baseline scenarios combined with incremental risks from non-residential exposures. This comment was selective and based on things taken out of context.

Item 4. Blood Lead Assessments The comments about the Panhandle Health Dept. screening efforts don't make much sense. All children should be screened in August, whether they are tested by PHD or not. The PHD assessment is for a specific purpose, which is a valid one. However, other studies such as the 1996 ATSDR/State study are more representative. What's more, one can make the argument that screenings and surveys in the Basin can either under-represent or over-represent risk. The contributors to bias in the Pb-B results were reasonably well handled in the draft HHRA ExSum and Ch. 8 Summary and Conclusions.

Item 5. Excessive Use of Safety Factors Item 5 first presents five bullets that collectively argue that the HHRA has seemingly built in over-protection, i.e., too many safety factors. That is not true, nor are the criticisms relevant. The criticisms show considerable ignorance about widely accepted environmental epidemiological and toxicological aspects of lead.

Bullet 1 seems to take exception to using August sampling to maximize survey data. That's more than a bit silly. We know from an extensive, widely accepted scientific and clinical literature that it is important to be able to monitor maximum Pb-B values, not values less than maximum that occur in other seasons. Why? Because dose-response relationships for lead exposure and lead poisoning are based on the concentration of Pb-B achieved, so that

the maximum toxicity risk in exposed children is only validly ascertainable by testing when Pb-B is at the maximum.

If the point of the criticism is that Pb-B values are lower and there is therefore less risk at other times of the year, the logic is meaningless. We do not know mechanistically what is the minimal time period for children at some maximum August Pb-B value to sustain toxic harm. A maximum Pb-B achieved for several summer months would be assumed to be sufficient to produce maximal harm. Secondly, neurodevelopmental harm in children is irreversible. Harm produced in August at maximum Pb-B value does not reverse at lower Pb-B values in other seasons. Simply put, one cannot average out Pb-B values throughout all seasons and use that in a dose-response relationship. That's simply not how it works.

Bullet 2 asserts that high-end ingestion rates were used for all scenarios. This is hardly the case. Based on current information about how much soil and dust children ingest, including recent and ongoing studies of Calabrese and Stanek, the values chosen in the HHRA are not all at RMEs. See for example, the newly-published paper by these authors in the October Risk Analysis:

Ref: Stanek EJ, Calabrese EJ. Daily soil ingestion rates for children at a Superfund site. Risk Analysis 20: 627-635 (2000).

They are likely somewhere between CT and 95%-RME values.

Bullet 3. This point of this statement is erroneous, or at best, misleading. The whole fish scenario is applicable for traditional subsistence practices of the CdA Tribe in the Basin. Whole fish describe the traditional dietary habit of these people. They did not fillet fish like the Eurocentric settlers or current residents.

Bullet 4. This point about using shallow well scenarios is ambiguous. Relative to deep wells, shallow wells invariably carry a higher contamination or likelihood of this. I don't believe the HHRA indicated anything more than this in the use of shallow-well groundwater exposures.

Bullet 5. EPA can respond to how the adult modeling assumptions for contaminated soil workplace settings jibe with the OSHA value and what is the legal basis for use of 10 µg/dl as the LOC. Secondly, the comment confuses a long-obsolete standard that is woefully out of date with current accepted science for fetal dose-toxic response relationships. What’s more, the numerical value has often been bypassed by the practice of removing women from the exposure setting when pregnancy occurs, at least subject to challenge under the U.S. Supreme Court ruling in the Johnson Controls case in 1991. As noted in many expert consensus documents of public agencies, such as the 1993 NAS/NRC report on sensitive population lead exposures, maternal Pb-B values at 10 units or even less are linked to threats to fetal development.

Bullet 6. The points about waste piles mischaracterize the approaches for dealing with waste piles. First, the way children interact with waste piles means that various depths of piles produce exposures. Older children, especially, encounter waste piles at potentially diverse depths: surface and subsurface. Secondly, these waste piles have the highest levels of contaminants of all media encountered by Basin children. Therefore, there are more negative consequences for child protection with any failure to be adequately protective with exposure factor assumptions. Underestimating intakes of fine tailing particles that contain 10,000-20,000 ppm lead, for example, can have great consequences in terms of toxic harm. I refer the commenters to my responses to Dr. Coomes on this very topic.

The commenters are correct that 25% was the best outcome. However, the comment is misleading in that the ExSum makes clear that the level of participation can either underestimate or overestimate best likely estimate of Basin-wide exposures. It is incorrect to assume or assert that the level of participation as occurred would only have somehow overestimated risk. The opposite could have occurred.

The last comment on p. 2 shows a total unawareness of the nature of blood lead measurements, the role of the IEUBK model at waste sites, and the interplay between the two. Commenters are referred to the August 1998 EPA OSWER directive on these matters. That should clarify any confusion.

The first paragraph, p. 3, is contradictory on its face and circular as to its logic. The only evidence that the SNRC or anyone else has to show how safe or unsafe the Basin is, is the HHRA. The commenters offer nothing equivalent to the HHRA to rebut the HHRA. Until that appears on the scene, the current HHRA is it.

The last paragraph challenges the HHRA to look beyond the "status quo." In point of fact, looking beyond the status quo, i.e., looking beyond current status of exposure, is the rationale behind agencies in general and the HHRA in particular using the IEUBK model for risk characterization for lead to anticipate future-use scenarios as well as current exposure settings, i.e., the "status quo."

V. Lands Council Comments

The Council comments generally support the draft HHRA. The Council, however, does advocate use of the more conservative default model versions vs. the Box model. The selection eventually becomes a risk management issue, which is where a number of the Council comments of this nature need to be directed at some future point.

VI. Coeur d'Alene Chamber of Commerce Comments

The Chamber of Commerce (C of C) comments are highly focused and brief in length, comprising three pages of a letter submission. They largely challenge the HHRA on points that were addressed fairly and at length in the HHRA. In that sense, the commenters

have either misunderstood or mischaracterized the material in the HHRA and the HHRA's interpretation of the data for overall risk characterization of lead and non-lead contaminants.

p. 1, 2nd par. The C of C mischaracterizes the nature and implications of national vs. local Pb-B distributions as a function of socioeconomic and demographic strata. Secondly, the nature and interpretation of Pb exposure data gathering by the Panhandle Health Department is distinct from the overall and broader nature and needs of the HHRA. Site-specific conditions were in fact used to a fare-thee-well in the HHRA.

pp. 2 and 3, Bullets and sub-bullets

Bullet 1 is a general comment about there having been progress. There appears to be no comment on the HHRA as such. No response is necessary.

Bullet 2 misstates the relationship of the HHRA to current and future risk scenarios. How exactly is anything "hidden" in terms of gains? One cannot color the interpretation of the level of current risk by declaring, well, things were worse years ago so let's let it go at that. I don't see the existence of any guidance or comments useful to the HHRA authors. Secondly, the average Pb-B values are tempered in the use of the IEUBK model by having to be the G.M. value corresponding to a 95%-ile value of 10 µg/dl.

Bullet 3, sub-bullet 1 misstates or misunderstands what the HHRA says about lead risks in the overall Basin. It is clear that the residential scenarios for the principal geographical areas all show significant exceedence of the LOC in Pb-B, 10 µg/dl. Secondly, education is no substitute for the physical remedying of soil and other media lead by abatements. We know this, and the topic is treated in the HHRA. Citations by the commenters of such material as the ATSDR information ignore all the qualifications and caveats about such information stated in the HHRA. Furthermore, the HHRA notes that the main lake cannot be characterized as to a fish consumption risk based on the lateral lake data.

Ibid, sub-bullet 2 does not in any way present evidence that the assumptions for child contact with exposure media April through November are unrealistically over-protective. One can't simply claim such assumptions to be inappropriate.

Bullet 4 is largely meaningless, since it ignores the fact that national data for lead exposure cannot be applied to a small area like the Basin. This was noted in a federal document back in 1988, the ATSDR report to Congress on childhood lead poisoning, and in the caveats discussed in the HHRA. The commenters insist on misrepresenting this basic bit of information. As to the second point, lead paint was heavily covered in the HHRA, more so in a number of statistical respects than soil lead. Everything that could be done to give lead paint a thorough assessment was done in the HHRA.

Bullet 5 is grossly uninformed or misinformed as to lead mechanisms of toxicity, lead dose-response relationships, the underlying purpose of a Pb-B measurement, the best time to do

a measurement, and why we do this in terms of dose-response relationships. The commenters here have the same problem as comments on this topic from the SNRC. My responses are the same as those for the SNRC set. Refer to those responses. For example, one cannot average out Pb-B values rather than get a maximum expression during late summer, since maximum toxic harm is related to the maximum Pb-B, the toxic harm is irreversible, and one cannot ignore this toxicological mechanistic reality for some vague and irrelevant statistical purpose.

The last paragraph is essentially what the C of C considers a statement of purpose with reference to pervasive Basin contamination rather than comments on HHRA sections. No response is required.

PB Associates

Memorandum

From: Paul Mushak

CC: Sharon Quiring, Marc Stifelman, Sean Sheldrake, Ian von Lindern

Date: 12/15/00

Re: Part 3. Responses to Mining Industry Comments on the Draft HHRA

Part 3: Responses to Mining Industry Comments on the Draft HHRA

Paul Mushak, Ph.D.

VII. Submissions from Hecla Mining and ASARCO

This is the last set of responses to public and other comments on the draft HHRA, those provided by Hecla Mining and ASARCO and collectively referred to as mining industry comments. The comments being addressed were those provided in hard copy via fax. This consisted of the cover letter of transmittal from the law firm HellerEhrman along with a set of comments comprising 48 pages, inclusive of references.

I provide both general and specific responses to comments provided below. My general comments are pegged to the general comments of the mining companies, and my specific comments are directed to the mining companies' specific comments.

VIIA. General Comments

The general evaluation of the HHRA in the industry comments includes basically two themes in critiques of the draft HHRA for lead. First, the comments aver that the HHRA authors and advisors overestimated community-wide toxicity risks in the Basin. Secondly, the HHRA steers risk management decisions as to clean-up strategies based on risk overestimates that the mining interests would judge Draconian and too demanding of resources, according to these comments.

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Pages 1-13 in the industry comments present what they first summarize on pp. 1-2 as their "Fundamental Concerns." They are grouped as "A. Inappropriate Modeling," "B. Potential for High Bias to Blood Lead Data," "C. Preferability of Community Health Intervention Approach.", and " D. Exaggeration of Arsenic Risks."

p. 2 et seq., A. Inappropriate Modeling. The mining industry comments pose the notion that the modeling of lead exposure and risk in the Basin was simplistic. In doing so, however, they largely misrepresent what was done in the HHRA in terms of modeling in the form of IEUBK modeling of children's exposures, the nature of the model, and the implications of the model for risk amelioration in the Basin.

p. 2, 1st full par. The comments challenge the limitations of the model in ways that are incorrect or, at best, misleading. For example, the assertion that paint lead is largely excluded is rebutted by the fact that one can accommodate paint lead in either the multi-source dust lead mode or one can use the alternate source mode, where daily lead intake as paint can be employed. The comments appear to seek to create the erroneous impression that the IEUBK model is basically a soil lead model for children's predicted exposures and any site-specific data showing other sources cannot be accommodated. The above is all that need be said on that point if the authors wish to respond.

p. 2, last par. onto p. 3, top The comments here seem to be a set of rambling, contradictory critiques of the inability of both versions of the IEUBK model as used in the Basin to match the blood lead data. This presents the industry view with a contradictory duality, in my opinion. They seem to be first saying that the modeling does not match the measurements, but at the same time are also asserting that the measurements of Pb-B do not best represent the level of lead exposure of Basin children. One has to ask, which is it that's the problem, the modeling or the measurements!? As I noted in my detailed paper in the 1998 Suppl. 6 issue of Environmental Health Perspectives, discordances in prediction vs. measurement can have various causes.

Ref: Mushak P. Uses and limits of empirical data in measuring and modeling human lead exposure. Environ. Health Perspect. (Suppl. 6) 106: 1467-1484 (1998).

The claim that no Basin-specific model was developed does not ring true. All the environmental measures that were available and quantifiable were used as environmental inputs; the bioavailabilities were ascertained as two values, one for the Box and one for default; and these bracket or approach the likely uptake rate. In addition, accommodations in the overall risk characterization scheme were done for lead paint, augmenting the principal model uses.

The comments state that the model does not show the low average Pb-B values. The statements misrepresent the Pb-B data. They are not "low" in terms of what the tabulations in the HHRA show in Ch. 6 in terms of the number of exceedences of the LOC, 10 µg/dl. Their experts need only read what's available. First, the comments seemingly set up a straw misstatement, i.e., blood leads are low in the Basin, and then attempt to demolish use of the model by noting that modeling says the Pb-Bs are not low.

The comments also attempt to set up a straw issue in terms of the intended uses of the model via EPA-OSWER guidance in the Basin or at any other site: open-ended modeling of lead exposures, factoring in changes in demographics, changes in future land use, etc.

It is not clear what the comments mean by the assertion that smelter emissions and their impact on lead intake and uptake may not apply to the rest of the Basin. They offer no evidence, however, to show why or if, in fact, inclusion or exclusion of "smelter emissions" can be done or should be done. Smelter emissions can in fact be mobilized by a variety of mechanisms for fate and transport that would/ could operate in the Basin. This is broadly discernible, first, in the aggregated environmental flow scheme for lead, first presented in the 1986 EPA lead criteria document, Fig. 7-1, p. 7-2, vol. II of this 4-volume document. Secondly, documented major flooding events have also produced documented measurements by the USGS of suspended and mobilized particles that were mobilized downstream. This would have necessarily included particulate from historical smelter emissions that were (1) dispersed first by air to fallout onto soils, (2) brought into surface runoff carrying suspended smelter fallout particles to the Coeur d'Alene River, and (3) subjected to flood events mobilizing sediments comprised in part of original smelter particulate arriving via (1) and (2).

p. 3, 1st bullet, to top of p. 4 I don't see that there is a big discrepancy here in terms of soil ingestion rates. However, it's something that can be responded to by the HHRA authors. I would note that the comments offer no evidence that "camping" entails no "outdoor" exposures and therefore would not entail as much contact with outside soils. This is a bizarre statement and one that, in any event, entails unsubstantiated speculation.

p. 4, 1st Bullet The commenters are incorrect that waste piles are so remote from contact by mobile (or even relatively immobile) children that they should not be figuring in soil level estimates. The comments indicate no awareness of actual conditions up and down the Basin. For example, Hecla's abandoned concentrator at Burke is across the street from residences, so tailing particles with high contaminant content are free to be transported a very short distance and to pile up by the side of the road. Such close impact likely explains why Pb-B values for young children in Burke/Nine Mile are especially high.

Ibid, 2nd bullet The commenters draw attention to the HHRA assumptions and tabulations indicating that areas away from home pose much of the lead exposure problems for the Lower Basin. However, the commenters note in challenge to that HHRA assumption that it is the home-bound infant and toddler who has the higher Pb-B, compared to older children. This should be responded to by the authors, keeping in mind that the commenters are vague and potentially misleading in this claim and ignore some basic activities among exposed populations. For example:

-- The Pb-B measurements for all children at various years were gathered in Summer, exactly the time period when families with both older children and very young children will be frequenting recreational areas with their children. During the recreational scenario operation, families have their infants and toddlers with them; they are not left at the residence.

--Contaminated beaches will typically be a play area with relatively intense contact exposure via mouthing and other pathways for infants and toddlers under parental and other family member observation when the family is at the beach and other recreational areas; this is what families do at beaches with their youngest children.

--Older children can also ultimately produce increased exposures for their younger siblings at other than beach times by the take-home contaminant dust pathway, by playing at recreational areas and bringing particles home on shoes, clothing, bikes, pets, etc.

p. 5, bullet The commenters dismiss the subsistence scenario as never being more than purely hypothetical. In support of their conclusion, the commenters take the position that the Tribal areas are unlikely to ever practice any traditional subsistence in the future whatever the status of contamination, remediation, or amounts of money spent for remediation. This logic ignores some historical points. The HHRA authors described traditional subsistence scenario as a hypothetical scenario, but they also, correctly, did not state that the scenario's behavioral characteristics for tribe members would never translate into any such future practice, even if only ritually, if the contamination and associated risks were removed. It is the current level of pervasive contamination which the HHRA takes as the reason for the traditional subsistence practices being largely hypothetical. Surely, the Coeur d'Alene tribe would take issue with the notion of their traditional lifestyle being gone forever. The reason the traditional subsistence lifestyle was abandoned in the first place was a very prudent recognition that a century or more of upstream mining waste emissions made continuation of such a practice an unacceptable hazard to life and health. The whole thrust and conceptual basis of the Coeur d'Alene tribal lawsuit presumably resides in this simple behavioral cause-effect reality.

Ibid, 1st full par. The basic premise is in error in these comments. The premise is that when we have any discordance between the model and measurement(s), the model is flawed. This shows ignorance about uses of blood lead surveys, their nature, and how and why EPA employs the IEUBK model vs. isolated Pb-B measurements. The commenters are referred to the August 1998 OSWER directive on use of models vs. blood lead measurements, and the role of each for risk interventions. Again, there is a contradictory stance between what's expressed here and elsewhere. That is, the submission questions the Pb-B measurements, then turns around and claims the model is at odds with these measurements that the commenters criticize.

p. 6: B. Potential for High Bias to Blood Lead Data This section is a series of mischaracterizations of sections on Pb-B measurements in the HHRA. I note these below.

Ibid, par. 1 The participation rate was low. However, the ExSum and Ch. 8 in the HHRA note that any impact of this could be in either direction. The commenters here prefer to assume that the impact would be to overestimate risk.

Ibid and p. 7, pars. 2, 3, 4 in Section The comments here misstate the caveats in the HHRA section dealing with this. The commenters seem to believe that any biasing is to the high end, rather than the low end. This is stated despite the fact that the HHRA makes it clear that there are a number of reasons why the Pb-Bs are likely to be lower. Those reasons for biasing to the low end are rather convincing to anyone informed on the environmental epidemiology of lead and the associated behavioral interactive factors operating with the key parameters for such analyses. Curiously, the commenters in the last par. of B acknowledge the existence of major factors driving to the low end in bias but then ignore it.

p. 7 et seq: C. Preferability of Community Health Intervention Approach

p. 8, 1st par. This par. claims that HUD and EPA guidance exists for soil Pb levels 400-5000 ppm that permits methods other than soil Pb abatement to be used. The commenters actually misread the relevant guidance and even to what that guidance applies. The guidance statements at issue actually deal with interim Title IV §403 of TSCA as described in various EPA-OSWER directives. TSCA §403 guidance should be evaluated separately from the OSWER program guidance and the two should not be confused, the former mainly having to do with lead paint programs in largely urban areas. The distinctions, in their major features, are collectively captured in three OSWER directives: (1) #9355.4-12, EPA/540/F-94/043, August 1994; (2) #9200.4-27, EPA/540/F-98/030, August 27, 1998; (3) # 9200.4-29, EPA 540-F-98-061, December 1, 1998.

It is a misreading of the December 1, 1998 OSWER guidance to claim that remediation options above 400 ppm do not have to include soil lead abatement until soil Pb reaches 2000 or 5000 ppm at legally established CERCLA sites (the commenters note both values).

The 12/1/98 guidance from the AA for OPPTS, Lynn R. Goldman, clarifies confusion about soil clean-up levels addressed in Title IV TSCA §403 and both CERCLA and RCRA sites. This OSWER directive also rebuts the inaccurate statements in this comment paragraph. For example:

[p. 1, par. 1 of Directive] *"...questions have been raised about the relationship between the proposed TSCA §403 rule [proposed June 3, 1998] and the Office of Solid Waste and Emergency Responses' Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Facilities (OSWER Directive #9200.4-27P, August 27, 1998)."*

[p. 2, par. 1 of Directive] *"EPA has proposed a 2,000 ppm hazard standard for lead in soil at which children's exposures will be associated with a greater certainty of harm...The hazard standard was intended as a "worst first" level that will aid in setting priorities to address the greatest risks promptly. The proposed §403 regulations and the accompanying guidance are to be used by Federal, State, and Tribal lead paint programs, as well as by the industry performing inspections and risk assessments."*

[p. 2, par. 3, "*OSWER's Soil Lead Directive"*] *"The OSWER soil lead directive that provides guidance for the cleanup of lead-contaminated sites under the CERCLA and RCRA laws is unaffected by this proposal. CERCLA and RCRA soil lead clean-ups should follow the approach in the 1998 directive...The TSCA §403 proposed 2,000 ppm hazard level should not be treated as an Applicable or Relevant and Appropriate Requirement (ARAR), "to be considered" or TBC or media cleanup standard (MCS). As recognized in the TSCA §403 rule, lead contamination at levels below 2,000 ppm may pose a serious health risk based upon a site-specific evaluation and may warrant timely response actions. Thus, the 2,000 ppm proposed standard under TSCA §403 should not be used to modify approaches to addressing Brownfields, RCRA sites, National Priority List (NPL) sites, Federal CERCLA removal actions, and CERCLA non-NPL facilities."*

[p. 3, 1st full par.] *"In the absence of site-specific information, EPA believes that levels above 400 ppm may pose a health risk to children through elevated blood lead levels. The 400 ppm screening level identified in the OSWER soil lead guidance is consistent with the "level of concern" identified in the preamble to the proposed TSCA §403 rule."*

p. 8, 2nd par.

It is a misreading of the August 1998 OSWER directive # 9200.4-27, which clarifies interventional methods, to say that all approaches are equally useful or equally permissible for assessment and use. That directive notes (pp. 5,6; Appendix Fact Sheet) the requirement of a tiered approach, ignored in the industry comments:

"IV. Determining Appropriate Response Actions at Lead Sites

"In selecting site management strategies, it is OSWER's preference to seek early risk reduction with a combination of engineering controls (actions which permanently remove or treat contaminants, or create reliable barriers to mitigate the risk of exposure) and non-engineering response actions...

"As a given project progresses, OSWER's goal should be to reduce reliance on education and intervention programs to mitigate risk. The goal should be cleanup strategies that move away from reliance on long-term changes in community behavior to be protective; behavioral changes may be difficult to maintain over time. The actual remedy selected at each site must be determined by application of the NCP remedy selection criteria to site-specific circumstances. However, this approach recognizes the NCP preference for permanent remedies and emphasizes the use of engineering controls for long-term response actions.…"

The comments also mix together various programs in use at different sites giving an indiscriminate grab-bag of modalities that have had differing success rates. The Butte approach essentially permitted the PRPs to get a less stringent level of soil lead cleanup in exchange for paint lead reduction steps. In the case of the Basin, for example, the HHRA indicates that only one in five residences have lead painted surfaces deteriorated enough to produce any likely benefit from such intervention, while the remaining 80% still have elevated soil lead levels absent deteriorating lead paint surfaces.

In the Trail program, there is a joint program with the community and the company, with financial support from Cominco, the historical emitter. However, few or no permanent soil lead abatements appear to have been done, so long-term efficacy of the approach remains unknown. Secondly, the impact zone is relatively contained in Trail, versus the huge impact zone of Basin contamination. Thirdly, the socioeconomic heterogeneity is quite low as is population mobility, unlike the Basin's demographic and socioeconomic profiles for those subsets of the population likely to be maximally at risk. That is, maintaining public education and caregiver awareness in the Basin, with people coming in and out, would be difficult. The Bunker Hill "Inside the Box" Pb-B data indicates that children moving into the Box are potentially at higher risk.

Leadville's approach as seen in its ROD and cover declaration is still largely, in the words of the R-8 Administrator, a "pilot project" approach — requiring close

oversight and with no clear or recommended relevance to, or a precedent for, any other site — to include five-year reviews and the pilot project to be "evaluated by a group of outside scientists."

In the words of the Region 8 administrator, the Leadville cleanup level was not intended to be one based or driven by health risk numbers. In fact the number chosen was admitted to be above health-based cleanup. For example, in the Leadville OU #9 ROD's declaration of September 2, 1999, Region VIII's Administrator office notes in the Statutory Determinations paragraph of the ROD that "... *Because this decision will result in hazardous substances remaining on-site, ABOVE HEALTH BASED LEVELS [upper case used for emphasis], five-year reviews of this response action will be required."*

Collectively, the Leadville ROD cleanup value was a purely experimental policy-driven exercise, not health risk driven, and therefore hardly an encouraging science-based, objective model for other sites, including the Basin. Here, again, contradictory stances by the commenters. Science alone is seemingly demanded by the commenters for reliable risk assessment in the Basin, while simultaneously touting for application to the Basin the non-scientific, purely experimental policy-driven Leadville OU 9 ROD cleanup level. What's more, it’s a choice of a level that by the Region 8 Administrator's own admission, is not health-driven.

I do not see, in any of the examples, any modality that comports with what the paragraph says they are, or whether the site characteristics of the Basin allow these to be used.

There appear to be contradictory positions here as well. First, there is insistence on solely site-specific information (see earlier response text) and then simultaneously a reaching out indiscriminately to quite different off-site areas, including one in Canada, to decide what's best for the Basin.

p. 9, par. 1 This par. first offers an unsubstantiated premise and then proceeds to lever further arguments with it. The HHRA makes it clear that it is extractive industry contamination that is the dominant contamination source. This is evident from the various statistical and other analyses described in detail in Ch. 6 of the HHRA. Secondly, while the contamination in the Basin may be stable, as the commenters claim, the impacted populations are not. The HHRA makes it clear that any in-migration is predominantly lower-income, less aware families who would be disproportionately at risk at the present time and with future demographic trends, absent any alteration of this contamination in place for "at least the last 75 years."

p. 9, par. 2 onto top, p. 10 This par. states that dust mat lead can originate from either interior or exterior sources. First, no evidence is presented, only speculation, to bolster the commenters' assertion. This speculation also is rebutted by evidence

from other studies showing that in fact mats collect particles from shoes when individuals enter their residences. See, for example, the results of the University of Cincinnati portion of the EPA three-city soil lead abatement demonstration project as described in both the UCinn portion of the study and the final, 1996 EPA Integrated Report of the project describing the use of mats in entryways and their use in assessing dust lead mats. Finally, simple logic as well as the study results dictate that mats collect most of their lead loadings and lead concentrations from exterior soils, since people wipe their dirt-laden shoes on the entry mat. It would be somewhat difficult to argue that people also typically wipe their feet on entry mats when exiting their residence.

Ref: U.S. EPA. Urban soil lead abatement demonstration project: EPA integrated report.EPA 600/P-93/001aF. Research Triangle Park, NC:Environmental Protection Agency, 1996.

The commenters state that direct blood lead to soil lead relationships are low. They then go on to note that the HHRA did not fully assess the allegedly significant role of paint. The commenters seem to be unaware of the fact that soil lead imparts effects on blood lead through various pathways, direct but mainly indirect, each of which has its own contribution to blood lead. The pathways therefore have to be evaluated for what would be the TOTAL contribution. In addition, one can also assess other lead sources contributing to dust pathways. Typical of a number of other studies, the relative size of the direct associations are tempered by the need to do structural equation modeling (SEM), developed by the UCinn group, and employed by that group to show sizeable indirect contributions of soil lead to blood lead. It is difficult to understand why the commenters would not acknowledge these Cincinnati studies in this section, since they cite the December, 1998 paper of Succop et al. (see references) that summarizes all the Western extractive industry sites studied by the group. These authors, developers of SEM, interestingly note that soil lead is more often a more robust source for dust lead than is lead paint in their exposure models applied to Western sites.

In addition to the Cincinnati group, US EPA has evaluated soil lead-dust lead relationships at Superfund sites using SEM. I refer the commenters to the 1995 EPA statistical analysis report by EPA's Dr. Alan Marcus, done for EPA Region 5 using data from the Taracorp/NL Industries Superfund site in Granite City, Madison County, Illinois. Detailed SEM analyses were employed to tease out very effectively a total robust soil lead input to blood lead, even though a direct association was found to be relatively modest in an earlier statistical analysis by authors of a 1994 assessment of lead exposures in children at this site.

Refs:

Marcus AH. Statistical analysis of data from the Madison County Lead Study and implications for remediation of lead-contaminated soil. Attachment 4: Decision Document/ Explanation of Significant Differences: NL Industries/ Taracorp Site. U.S. Environmental Protection Agency Region V, Chicago, IL, 1995. Available from EPA Region V: Waste Management Division, Chicago IL.

Illinois Department of Public Health. Madison County Lead Exposure Study. Granite City, Illinois. Springfield, IL, 1994.

p. 10, 1st full par. The commenters claim that those children with the high Pb-B levels identified in screening efforts were those also having high soil levels. First, the commenters seem to misunderstand the methodology underlying use of inferential statistics to link Pb-B and environmental media lead levels. It is not valid scientifically to use a small or pre-selected set of children identified via a screening program and health department intervention to draw conclusions about their accompanying soil lead levels, since this entails apples and oranges, statistically speaking. One cannot examine soil lead levels vs. Pb-B in children without using the full epidemiological cohorts across the four years of study, i.e., use of all the blood-soil lead pairs to do a full spectrum analysis. This is basic in lead epidemiology. The high blood lead children were identified in a different context statistically and for a different purpose.

Ibid, 2nd full par. The commenters seem to be claiming that the reason there's a health problem in the Basin is because there is low socioeconomic status. That observation, true or not, merely identifies a risk factor for lead exposure, it is not a surrogate explanation for why lead contamination causes both lead exposure and lead toxicity. When one has both amplifying factors for lead exposure and for lead toxicity, it is more necessary to be stringent about lead control, not less. The logic here appears to be a variation of blame-the-exposed-victim.

p. 11, 1st par. The commenters incorrectly and misleadingly claim that it is only the model that invokes a role for dust and soil lead in blood lead elevations. The commenters assert that an expert witness for the government in the litigation phase of this site’s actions agreed that children’s blood lead levels are not associated with soil lead in the Basin.

Several responses are merited here. First, all the public comments were made to the administrative record for the RI/FS in terms of public comments on an HHRA. The HHRA is not a litigation/court document per se being fought over by expert witnesses. Therefore, the commenters' reference to Dr. Landrigan's deposition and whether he said or did not say something regarding soil lead and blood lead is not directly relevant to the issues at hand for objective review of this HHRA, nor is it appropriate that responses to this comment be focused on Dr. Landrigan's testimony in the separate matter of litigation in the Basin. I am quite

familiar with Dr. Landrigan's clinical expertise in pediatric lead poisoning. It's not clear what is Dr. Landrigan's actual published expertise in the areas of exposure pathway analysis, multi-regression analyses using structural equation modeling, valid use of discrete sampling vs. single composites in some geostatistical design framework, etc. or for that matter, the actual context in which his deposition testimony said something or did not say something.

The soil lead-blood lead direct relationship as assessed in the 1999 report of the 1996 Idaho/ATSDR study is tempered by some simple technical problems. First, soil lead operates through several pathway mechanisms to have an impact on Pb-B. This is obvious from the EPA Integrated Report document cited above. I refer the commenters to my response showing that entry mats, with loadings and lead levels linked to Pb-B values, would mainly reflect soil and exterior dusts for obvious reasons. The best statistical analysis to tease out what was going on in this 1996 study in terms of soil lead getting to blood via all well-accepted pathways was not employed. Secondly, the study entailed inappropriate use of basically single or dual sampling per residential unit without prior detailed sampling to establish the presence of heterogeneity in the soil lead levels and their distribution in the Basin yards. Without any knowledge of the nature of the lead distributions in these residential yards, or their heterogeneity, one had to first use multiple discrete samplings to assess whether single or double samples per yard were even adequate.

I refer the commenters to what EPA's RAGS 1989 document says in a number of places about the need to ascertain hotspots and the inherent limitations of single sampling of an exposure unit. These responses also appear in my responses to Dr. Coomes. EPA's 1989 RAGS document treats hot spots in a prescribed way. Hot spot discussions are contained in RAGS at pp. 4-10 to 4-12, 4-17, 4-19, 5-27, 6-24 and 6-28. On p. 6-28, last par., left column:

"In some cases, contamination may be unevenly distributed across a site, resulting in hot spots (areas of high contamination relative to other areas of the site). If a hot spot is located near an area which, because of site or population characteristics, is visited or used more frequently, exposure to the hot spot should be assessed separately."

Absent any prior documentation as to the nature of lead contamination heterogeneity and lead distribution within the yards in the Basin, it would be inappropriate to take one or two samples of yard soil for testing and then say this is a reflection of lead distribution.

In the May, 1999 draft final report of the Idaho/ATSDR study, the investigators found in their statistical analyses additional results that provide good evidence that the soil lead-blood lead relationship would be stronger than indicated in a simple direct analysis.

On p. 38 of the 5/99 Idaho/ATSDR draft final, the summary bullet notes:

" • Children less than ten years of age who played outdoors most frequently on dirt or sand surfaces (including sandbox) had significantly higher log blood lead values than did children who played outdoors most frequently on grass or other surfaces. The proportion of children with elevated blood lead levels differed significantly by outdoor playing surface. Thirty-eight percent (37.5%) of children who played outdoors most frequently on dirt or sand surfaces had elevated blood lead levels, compared with 4.8% of children who played outdoors most frequently on grass or other surfaces."

The above refutes the notion, as do my other responses, that there was no relationship between lead in soil and lead in young children's blood. The more available the outside bare soil was to children, i.e., the soil/sand surface, the higher the blood lead. The less the contact with bare soil/sand, the lower the Pb-B.

p. 11: D. Exaggeration of Arsenic (As) Risks

1st par. et seq., pp. 11-13 The commenters claim that the RfD for ingested arsenic, involving non-cancer effects, is applicable only for a lifetime of exposure, and is therefore inappropriate for the child age band. This can be rejected on a number of grounds, given recent studies. First, we cannot say that the RfD as derived would be inappropriate, since it was developed for cardiovascular and dermatopathological lesions before new data emerged showing that children are at higher risk than adults for As non-cancer effects and that As is handled metabolically by the child differently than by the adult. The use of an RfD for less than lifetime risk is quite appropriate for an age band narrower than lifetime when there is increased risk within that band. That's what we have with children.

The commenters are referred to the NAS/NRC 1999 authoritative document on drinking water As for discussion of children as being at special risk. Discussions include one on p. 232 of the NAS report. Studies show that children don't biomethylated As as well as adults, although the precise role of biomethylation is not clear given current research. Two papers in particular were reviewed and their results basically accepted by the NAS report authors, those of Concha et al., 1998 and Kurttio et al., 1998. They collectively show that impaired biomethylation continues across a broad childhood band. That is to say, the risk band is broader than just infants and toddlers.

The use of the RfD for exposures that occur well into adulthood and certainly for the childbearing years in women, for the specific purpose of protecting against developmental toxicity of the fetus, is called for as well. A very recent paper by Hopenhayn-Rich et al., studying pregnancies and early infant outcomes in Chilean

mother-infant pairs, showed that increases in maternal As exposures are related to increases in infant mortality rates in these women.

Ref: Hopenhayn-Rich C, Browning SR, Hertz-Picciotto I, Ferreccio C, Peralta C, Gibb H. Chronic arsenic exposure and risk of infant mortality in two areas of Chile. Environ. Health Perspect. 108: 667-673 (2000).

The commenters claim that the low dose As relationship to cancer is sub-linear. That claim is clearly contradicted by the conclusions of the NAS 1999 report on As, which the commenters cite in their reference list. The NRC report authors note that the available models for low-dose extrapolation do not permit ruling out linear extrapolation. Quoting from its Executive Summary, p. 7, Risk Characterization, par. 3:

"Information on the mode of action of arsenic and other available data that can help to determine the shape of the dose-response curve in the range of extrapolation are inconclusive and do not meet EPA's stated criteria for departure from the default assumption of linearity. Of the several modes of action that are considered most plausible, a sublinear dose-response curve in the low-dose range is predicted, although linearity cannot be ruled out."

The NRC report made it clear that it considered the nature of the low-dose relationship to be driven by the mechanism of carcinogenic action of As. Since the NRC report appeared, additional data have appeared showing that a linear model at low dose would in fact be reasonable. Mass and coworkers, in work described in an SOT abstract, show that direct interaction of arsenic as the trivalent monomethyl metabolite with DNA was seen in tandem with various measures of DNA damage. Damage included: unwinding (nicking) of DNA and production of double-stranded breaks, and/or induction of alkaline labile sites at levels well below inorganic As levels. A number of other measures of damage were positive. These results show methyl-As (III) being genotoxic via DNA interaction.

Ref: Mass MJ, Tennant A, Roop B, Kundu B, Brock K, Kligerman A, DeMarini D, Wang C, Cullen W, Thomas D, Styblo M. Methylated arsenic (III) species react directly with DNA and are potential proximate or ultimate genotoxic forms of arsenic. The Toxicologist (2001, in press): Proc. Soc Toxicol 40th Annual Meeting, San Francisco, CA, March 25-29, 2001.

Ibid, 2nd par. onto top of p. 12 The commenters take issue with the soil ingestion rate for the recreational scenario, duplicating the criticism earlier for lead. Again, they offer no evidence for quantifying an alternative ingestion rate, only unsubstantiated speculation.

VIIB. Specific Responses to the Mining Industry's Specific Comments on the HHRA

A, Data Collection, p. 14 The commenters are superficially correct in noting that the mat at the entry way may not be fully integrating all dust that may arise and distribute internally. However, the fact remains that the mat loading and concentration were found to be particularly useful as a robust marker for interior dust reservoirs actually affecting Pb-B in earlier studies by the Cincinnati group. EPA, using the SEM approach of multiple regression analysis, in its Integrated report (referenced earlier) noted on p. 1-14, 15, Sec. 1.2.3, Cincinnati Study, that:

"...this integrated report concludes, through a detailed structural equation analysis, that there is a strong relationship between entry dust and interior dust in this subset of the Cincinnati study, where the impact of lead-based paint was minimized."

Not all of the residences tested in the Basin in 1996 had lead paint that was deteriorating. The actual value was about 20%. That is, 80% had a minimal input from deteriorating paint lead surfaces.

p. 15: Waste pile sample collection The commenters claim that the absence of fines on waste pile surfaces reduces the actual exposure impact of children playing thereon. This is simply misleading in its main thrust for risk characterizations away from home. I have addressed this issue in responses to other commenters, and commenters are referred to these statement. In summary, waste piles engage children, especially older children, in ways other than simple hand-surface contact where presence of fines would be an issue. The latter applies only to infants and toddlers, not likely to play on piles away from home. Older, mobile children climb waste piles, burrow in and around waste piles, ride bikes over the piles, etc., activities resulting in breaking through and exposing lower depths of piles. This justifies use of deeper depths and mitigates the need for surface fines. In other case, there are plenty of fines. As noted earlier, furthermore, absence of fines is not necessarily widespread. The waste piles around the defunct Burke concentrator have mobile tailings that are very close, across the street, from residences.

pp. 15-17: B. Data Interpretation

Geographic sub-area selection Again, as noted before, there is confusion as to integrating geographic sub-areas and the original five CSM units. The authors need to clarify this.

Background...in surface...and groundwater This may or may not be a valid concern by the commenters. It depends on the relative fraction of total metal in the samples that is dissolved. Relevant data for background levels showing the proportion of total metals that dissolved metals comprise would be helpful.

Screening arsenic concentrations in surface water We certainly know the forms of As in seafood are mainly present as arsenobetaine and/or arsenocholine and the authors should check the species-specific biotransformation processes in freshwater fish as well. However, there may be some confusion here on the part of the commenters. But given that fact, it's not obvious or clear that occurrence of biotransformation processes and transformed metabolites of As in biota preclude assessment via measurement of an empirical, quantitative relationship worked backwards from biota to limnological levels of original As. This relationship of a BCF to water levels is often followed, for example with methylmercury despite there being conversion from inorganic Hg to MeHg in lower trophic organisms, by biomagnification as one works up the trophic ladder. If some level of As, in whatever form, is mathematically linked to an original level of inorganic As in an aquatic system, why does the form of the biotic As preclude the computation?? The only possible way the matter might get muddled is if one cannot measure all forms as to core As content equally well. That could be a problem in a different context, such as if speciation itself for purposes of toxic potency were the focus. In the latter case, see the discussions in Mushak and Crocetti, cited below:

Ref: Mushak P, Crocetti AF. Risk and revisionism in arsenic cancer risk assessment. Environ. Health Perspect. 105: 103: 684-689 (1995).

Yard soil collection results This is a valid concern, and I found the section dealing with validity of combining data sets a bit confusing for yard soils. However, the HHRA does note that statistical tests of compatibility for sample sets collected different ways were done and merging was permissible except for the one case. There was also a reference to an Appendix. The authors might wish to clarify this portion some more. However, I reject the notion that there is such a huge biasing between 175 microns and 250 microns, the ceiling for IEUBK model testing, that one can't use 175 micron fractions. This is nonsense if one reads the scientific evidence. There may, in fact, be a very small fraction of the total sized between 175 and 250 microns in the total particles in a bulk sample. There appears to be some sense in the comment that particles somehow follow a homogeneous particle size distribution, such that one can simply interpolate the missing fraction between 175 and 250. With anthropogenic particles, there is typically a huge fraction below 100 microns, and especially below 10-50 microns. There may also be little between 250 and 175, at least not enough to question the results. This can be discerned in various studies. See, e.g., the plotted histogram data of Duggan et al. for general play areas for children:

Ref: Duggan MJ, Inskip MJ, Rundle SA, and Moorcroft JS. Lead in playground dust and on the hands of school children. Sci. Total Environ. 43: 65-79 (1985).

The case for geochemical waste materials from extractive industry sites is also especially informative. It permits us to say that little in the way of relative lead mass or total particle size fractions lies between 175 and 250. For example, EPA Region 8 reports particle size distributions for all of its study reports involving all those Superfund sites being tested in the young pig bioavailability testing protocol for lead. The protocol included sieving at 250. One can readily see that for the sub-250 particles, which of course would let through 175 particles as well, essentially 100% of the particles are below 175, with no evidence of particles of any substantive amount being 175 to 250 microns.

In the case of the Region 8 report for the Smuggler Mountain NPL site, Aspen, CO, Table 2.2 shows that 100% of the particles below 250 microns are at 125 microns or below. The highest particle size measured was 125 microns, for cerussite.

The Region 8 study of the Jasper County, MO Superfund site is especially telling, since the characterization of the media being studied for bioavailability included data not only on yards and the smelter site, but also mill tailings. The yard and mill tailings size distribution data have relevance to the media types in the Basin. In Table 2.2 of that report, we see that for the mill samples the fractions of particles between 175 and 250 were quite low, with the vast majority of the particles having a maximum size of 110 microns. For yard sample particles, the maximum particle size encountered was 100 microns.

Refs:

Casteel SW, Weis CP, Henningsen GM, Hoffman E, Brattin WJ, Hammon TL. Bioavailability of lead in soil samples from the Smuggler Mountain NPL Site Aspen, Colorado. (Region VIII). May, 1996. USEPA Region VIII Document File, Document Control No. 04800-030-0160.

Casteel SW, Weis CP, Henningsen GM, Hoffman E, Brattin WJ, Hammon TL. Bioavailability of lead in soil samples from the Jasper County, Missouri Superfund Site (Region VII). May, 1996. USEPA Region VIII Document File, Document Control No. 04800-030-0161.

pp. 17-22: C. Exposure Assessment Parameters

p. 17, Assessment of surface water exposures The commenters present a battery of purely speculative statements as to how the parameters selected for the HHRA in exposure assessment would militate against the HHRA choices. One can't rebut plausible assumptions by the HHRA with alternatives that offer little plausibility or credibility.

p. 17 onto p. 18, Waste pile exposures I have addressed this issue in my earlier comments.

p. 18, Exposure frequency this question arose in submissions of early commenters. The authors should clarify and reconcile with the climate record and behavior of the receptors at various times of the year.

Ibid, the recreational scenario soil ingestion rate This seems like a concern that can be responded to readily by the authors.

Dermal exposure pathway I would agree that the dermal pathway for some of the metals may not rival ingestion as to relative impact on risk. However, all pathways contribute to integrated intakes and integrated risks. We are not stratifying risks as simply "higher than" or "lower than."

I do take exception to what the Ruston, WA child data say about intakes based on biomonitoring of urine As. First, the sample size was relatively small especially when stratified as to distance from the site. Second, we don't know how As is handled in the bodies of children vs. adults, in terms of half-lives. Furthermore, we would expect that with a relative half-life for As in the human body being less than that for lead, i.e., several days, then a single-shot As screening could show a large effect on biomarkers for As where any alteration in that child's exposure interactions from parental intervention, etc., occurred. The vulnerability of a urine As to the artifact of parental awareness and control of children's activities is biologically and biokinetically higher than even for lead. Ruston, WA is a community that has been aware of, and concerned about, the ASARCO Tacoma smelter's impacts on the community for many years.

p. 20, to top p. 21, Homegrown vegetable...pathway The statements here misstate and selectively cite the available evidence. First, it is not the case that uptake of lead and arsenic by crops is low. Whether the uptake is low or not depends on a number of soil and phytochemical characteristics, and in any event will depend on soil metal concentrations as well as any uptake factors.

Hattemer-Frey et al., for example, described studies with a cluster of metals typically found at Superfund sites. The uptake rates varied enormously with various soil chemical characteristics and other factors. One cannot simply use, for example, static factors such as that developed by the USDA (Beyes, 1984) for all situations.

Ref: Hattemer-Frey HA, Krieger GR, Lau V. An evaluation of the effect of some soil properties on root uptake of four metals. In: (KB Hoddinott, ed.) Superfund Risk Assessment in Soil Contamination Studies (v. II), No. ASTME STP 1264. Philadelphia, PA: American Society for Testing and Materials.

Some studies of the role of garden vegetables at Superfund or related sites show limited risk, others show considerable risk. It depends on the thoroughness and effectiveness of the study design. A number of studies which simply tested available gardens ad-hoc had limited useful data, and this is not surprising from a statistical outcome standpoint. Such studies are limited by the availability of opportunity to study existing gardens.

Few investigators have systematically studied real-world garden plots in terms of valid statistical design and study execution. Where that was done, it is clear from the results that contaminated soils pose such a health risk that remediation and other intervention modalities typically urge residents not to plant gardens. In some cases, residential soil levels of contaminants are so high, nothing will grow. This is hardly reassuring from the standpoint of net human exposures via other pathways, however. The most thoroughly studied Superfund site in terms of systematic garden plot studies under multi-season, real world conditions by expert teams in this area were the studies carried out at Palmerton, PA, a community heavily impacted with multi-element contamination from two zinc smelters and associated facilities. The results, collectively, led to strong recommendations not to plant gardens or consume any garden crops whatsoever. The details of all these studies are in the EPA Region III Administrative Record for the Palmerton site, in Philadelphia.

The question of the chemical form of As in foods other than seafood has been an area of some contention. The debate over vegetables began with the detailed critique of the topic by Mushak and Crocetti in the arsenic paper cited in its entirety earlier.

p. 21, Use of house dust data The concern about non-lead soil vs. dust relationships is partly correct. The sources of lead in house dust are largely outside soil. The question of missing dust vs. soil relationships for non-lead should be addressed by the HHRA authors.

Ibid, Combination of exposure parameters Again, as was done repeatedly in this submittal and other commenter submissions I reviewed earlier, the commenters attempt to substitute reasonably protective scenario exposure parameters with alternatives that are only rooted in ungrounded speculations.

p.22: D. Exposure Scenarios

p. 22, Subsistence scenarios This compound comment consists of a number of bullets that indicate the commenters are unfamiliar with a number of studies in various publications. The commenters offer the purely arbitrary bit of speculation that the traditional subsistence scenario will never be achieved. I have responded to this in detail earlier and one can refer to these responses.

All the bullets are off the mark, since they indicate unawareness of the key study by Harris and Harper in Risk Analysis several years ago, supporting the figure of 300 mg/d for soil, 300 mg for sediment, etc. The commenters need to consult that paper, since they have not cited it. The paper is:

Ref: Harris SG, Harper BL. A Native American exposure scenario. Risk Analysis 17: 789-795 (1998).

It's also clear that the commenters are not aware that the 1997 update of the Native American tribal factors portions of the EPA Exposure Factors Handbook (EFH) supports the assumptions of exposure factors that amplify those factors based on a typical Eurocentric suburban or rural scenario. This set of comments is, in my opinion, woefully uninformed.

EPA EFH Ref: U.S. EPA. 1997. Exposure Factors Handbook. Volumes I-III. [An Update to Exposure Factors Handbook: EPA/600/8-89/043, 3/89]. Report No. EPA/600/R-97/006, 12/96: Washington, DC: Office of Solid Waste and Emergency Response.

p. 24, Combinations of exposure scenarios Again, the commenters substitute ungrounded speculations as to what they call the problem of overcounting since incremental exposures in their view are somehow concurrent with the baseline scenario. However, they fail or appear to fail to grasp that there are different subsets of receptors who are variably impacted by the residential baseline and the incremental scenarios. We don't double count children when we examine frequency of access to waste piles for older children along with infants and toddlers having exposures obviously restricted to the home, i.e., restricted mobility versus older children.

Besides the matter of double-counting, the commenters don't offer any useful documented alternatives to those factors selected for use in risk analysis in the HHRA. The comments here and elsewhere are riddled with subjective criticisms trying to have the reader draw an inference of implausibility without evidence.

p. 25: E. Characterization of Lead Health Risks This section starts with five paragraphs on lead toxicity characterization.

p. 25, 1st par. The commenters have a reasonable concern here in that the vast amount of accepted scientific evidence for dose-response relationships for lead are largely absent here. I had and still have problems here as well, since the information is there in huge abundance. I would suggest the authors paraphrase any of a number of dose-response tables and text from such authoritative sources as the 1993 NAS report on lead exposure, the 1997 and 1991 CDC Statements on lead poisoning in children, etc.

Ibid, 2nd par. over to p. 26 The commenters misrepresent the strength of low-level lead effects as an accepted body of science in the clinical and public health mainstream. The Stu Pocock paper is not accepted by regulatory agencies or among informed lead scientists and health professionals as showing trivial low-level effects (see, e.g., the 1993 NAS document). The commenters tend to represent a minority view in this, if not an actual fringe view. The commenters should consult: the 1993 NAS report on lead, the 1997 and 1991 CDC statements, the February, 1991 statement on elimination of childhood lead poisoning, the huge section in the 1986 EPA lead criteria document, i.e., Chapters 12 and 13 of Volume 4, and the 1995 WHO-IPCS lead criteria document.

p. 26, 1st full par. This par. is merely an added call to state dose-response relationships. Of course, this is useful, as I noted above. It is a bit of a disconnect, however, that the commenters cite the two CDC statements on childhood lead poisoning but don't communicate the clear conclusions in those statements that low-level lead poisoning in children is an important public health problem for health professionals.

p. 26, last par. over to p. 27 There is a terminology in the HHRA being referred to that is lifted from the 1991 CDC Statement. In the summary portion of that document, lead "poisoning" is noted to not occur below 10 µg/dl. The precise quotation is on p. 3, Table 1-1.

"Table 1-1. Interpretation of blood-lead results...

Class	*Blood lead concentration (µg/dL)*	*Comment*
I	*≤ 9*	*A child in Class I is not considered to be lead-poisoned."*

p. 27, 1st full par. Agreed. I suggested earlier that summary dose-response text for young children can be easily added from any of a number of the current, cited documents.

pp. 27-29,Site-specific Blood Lead and Environmental Exposure Analysis

p. 27 to top, p. 28, Role of socioeconomic status The commenters are not clear as to what point they are trying to make with comments on socioeconomic status. It is well known that SES status is a risk factor in the severity of lead poisoning risk, not the occurrence of risk. The occurrence of risk is there because of the lead contamination. The ExSum and Ch. 8 of the HHRA already make it clear with

explicit text that SES aggravates poisoning risk. The commenters cannot argue, obviously, that the reason there's lead in the environment of poor people — and therefore poor people have lead poisoning — is because they are poor; i.e., if they were not poor, they would not have lead poisoning. The premise is clearly untenable and illogical on its face.

p. 28, 29: four paragraphs on paint

1st par. The point's largely misleading. Paint will have a role only if it's present and present in a deteriorating condition or otherwise accessible to children. A vast literature for lead paint and lead epidemiology makes this clear. The commenters then state some simple statistical issues that the HHRA has neither rejected nor is not aware of. So, what's its point? Also, the commenters offer without substantiation the point that lead paint will be affecting the mat lead to the same extent as mining-related yard soil lead.

2nd par. The commenters offer comments about how paint lead is handled in the statistical analyses that are at odds with how detailed the paint lead analyses were. Furthermore, the commenters are referred to the Succop et al. paper in the 12/98 Supplement issue of EHP, describing in detail their findings as to how paint lead plays out in statistical models in Western mining sites, of which this group studied about 11 or so. Those studies showed that, in fact, there were a number of sites where lead paint was not as robust a source as yard soil lead largely arising from extractive industry contamination.

The commenters claim that the HHRA analyses would not sort out a case of paint entry from past activities. They then posit a historically undocumented set of scenarios, whereby former lead paint surface activity could have occurred, even if the current surfaces are intact. First, the soil samples taken for lead analyses were statistically gathered not next to the house, where exterior paint would fall, based on many earlier published studies, but apparently nearer a center point in each yard. Secondly, on purely statistical grounds, the amount of soil next to the house and POTENTIALLY containing lead paint particles as a fraction of total soil encountered by feet would be relatively small. Thirdly, the statistical analyses described earlier in my responses for the 1996 State/ ATSDR study show that blood lead is elevated in those bare soil areas that comprise play areas. Few of these areas, obviously, would be at the drip line.

3rd. par. Arsenic is not present in paint at levels rivaling those in geochemical media, if at all. Its use as a pigment was highly unlikely based on the available literature on paint technology, while geochemical As is a strong correlator with geochemical lead and cadmium. Second, the finding that arsenic is lower in house dust than in outside soil is the finding of a very limited, site-specific situation. The commenters cannot, obviously, extrapolate from one finding to a universal

statement. There is, again, a contradiction here. Commenters in parts of the submission demand use of only site-specific data and then indiscriminately throw in isolated findings from sites having nothing to do with the Basin.

4th par. I don't understand exactly what the points of the comments are? A general observation about a relationship not pursued in their comments as to a specific statistical purpose is then combined with a different risk factor in the lower Basin and then claimed collectively to explain the real problem. This is something the authors can readily deal with.

pp. 29, 30: two pars on correlations, Pb-B to environmental Pb

1st par. The commenters assert that the wording in the HHRA as to relative strengths of correlations, Pb-B vs. media, is misleading as to impact of yard soils. I will leave that to the authors as to what all went into formulating that statement, e.g., both direct and indirect pathways from soil to blood lead.

2nd par. The commenters are being simply misleading or misinformed when they note that the soil lead/ Pb-B slope factor seen in the Basin is much lower than assumed by the Model. First, this section in the HHRA discusses the DIRECT relationship of soil to blood, and does not take into account the strong role of soil lead adding to dust lead, and then the dust lead from soil lead having a robust relationship to Pb-B. This requires use of structural equation modeling. This point was also introduced and responded to above, in my earlier comments where I cite the 1995 structural equation modeling analysis used at the NL Industries/ Taracorp site as a reanalysis of the earlier report, a report in which the yard soil to blood lead relationship was, like here, modest. When soil lead acting through dust lead in the SEM was tested for, that connection had a very robust statistical relationship.

pp. 30-31, Source identification, 2 pars.

First par. First, the commenters posit a purely hypothetical, not substantiated role for paint lead for all the Basin children's lead exposures, and then note that this Pb-B increment has to be subtracted from what they imply is a sum of both geochemical lead from mining and paint lead. Nothing is offered as hard evidence or as their own analysis, merely speculations.

2nd par. The commenters attempt to link Bunker Hill Box lead exposures to the children's Pb-B data in the Basin. Quite aside from the obvious fact that it is the same lead source at issue throughout the Basin, the comments indicate a misunderstanding of the actual time points for lead biokinetics, mothers to infants. The *in-utero* damage done by lead is apparently permanent and that's not in dispute. But when the babies are born, maternal, prenatal Pb-B values lose linkage rapidly with the infants' Pb-B values. It is largely irrelevant to seemingly claim that

lead exposures measured in the Basin over the last four years are actually occurring in the BOX and are somehow imported into the rest of the Basin. That's a stretch, spatially and epidemiologically.

pp. 31 et seq: F. Application of the IEUBK Model

This section covers about six pages of comments about various parameters associated with uses of the IEUBK model for childhood lead exposures in the Basin.

p. 31, 1st par. The commenters assert that uses of the IEUBK model in the Basin by HHRA authors largely use default assumptions rather than site-specific data. The commenters then list their specific problems with the model's use. Responses are to those more specific comments.

p. 32 to top of p. 33, Geometric standard deviation The commenters offer a largely unsubstantiated or theoretical set of reasons why there should be a differentiation between individual GSDs and community GSDs, arguing that a group or community-wide basis for a GSD weaves in more than inter-individual variability. First, there is their question as to the extent to which inter-individual behaviors are already subsuming environmental heterogeneity by virtue of behavior dictating children "sampling" this heterogeneity in the community. Secondly, they argue that the model describes a hypothetical person whose inter-individual variability likewise "samples" the heterogeneity. It's not clear what sort of a straw issue the commenters first set up and then attempt to demolish.

The authors, I am sure, have a number of responses to this and they should offer them.

p. 32, 2nd par. The commenters here draw upon the GSDs found at other sites to make an argument for this site. Superfund sites differ greatly as to empirical calculation of a GSD. The commenters only cite, quite misleadingly, those which are less than the default 1.6. This is not being aboveboard. Other sites have had GSDs greater than the 1.6. For example, the Bornschein report for the Midvale, UT/ Sharon Steel site reported in 1990-91 that the Midvale community around the abandoned mill and large tailing pond had a GSD more like 1.8.

Secondly, the commenters have argued that the Pb-B surveys and screenings have been flawed. Now, however, they wish the reader to draw the inference that the HHRA should consider use of the empirical Pb-B data set to calculate an empirical GSD for the Basin. There is, here again, more than a whiff of contradiction.

p. 33, Bioavailability

1st Par. The first par. of comments mischaracterizes what the HHRA says about the relationship between measured and predicted Pb-B values up and down the Basin. Actually, in some cases the default was a closer predictor than the Box model.

2nd Par. This par. basically offers a collage of speculations as to, first, why different parts of the Basin will have different geochemical media from extractive industry waste streams, and then to argue why there is variability in level of measured vs. predicted concordance in the HHRA findings. This needs no response, unless the commenters can come up with hard evidence showing geochemically distinct media that in fact differ enough to be associated with different bioavailabilities. It is not enough to say that media may be chemically different, it must be shown that these are not distinctions without a biokinetic or toxicological difference.

3rd. par. The commenters make the blanket statement that Western mining sites are linked to media with lower bioavailabilities. This is both misleading and incorrect, as is the Ruby summary referred to. Also, the *in-vitro* testing has yet to be widely accepted, if at all, at this time. Again, to say otherwise is to be either misinformed or uninformed.

Many studies done jointly by EPA Region 8 and various academic research groups, and using the scientifically valid young swine bioavailability testing model, show relatively high bioavailabilities for diverse geochemical wastes from quite a number of sites. In fact, the Region 8 studies are now the most comprehensive set of valid and credible scientific studies of Western site bioavailabilities anywhere.

A number of these Region 8 studies were cited above in a different context. In addition, there are a number of others, including the 1997 Casteel et al. paper in the peer-reviewed literature.

Refs:

Casteel SW, Brown LD, Dunsmore ME, Weis CP, Henningsen GM, Hoffman E, Brattin WJ, Hammon TL. Bioavailability of lead in soil samples from the New Jersey Zinc NPL site, Palmerton, Pennsylvania, 1996. Doc Control No 04800-030-0159. Region VIII, U.S. Environmental Protection Agency, Denver, CO.

Casteel SW, Brown LD, Dunsmore ME, Weis CP, Henningsen GM, Hoffman E, Brattin WJ, Hammon TL. Bioavailability of lead in slag and soil samples from the Murray Smelter Superfund Site, 1996. Doc Control No 04800-030-0163. Region VIII, U.S. Environmental Protection Agency, Denver, CO.

Casteel SW, Cowart RP, Weis CP, Henningsen GM, Hoffman E, Brattin WJ, Guzman RE, Starost MF, Payne JT, Stockham SL, Becker SV, Drexler JW, Turk

JR. Bioavailability of lead to juvenile swine dosed with soil from the Smuggler Mountain NPL Site of Aspen, CO. Fund Appl Toxicol 36: 177-187 (1997).

p. 34, top The commenters misstate the Maddaloni et al. study, its conclusions and its relevance for epidemiology. The Maddaloni et al. paper merely used a Bunker Hill sample to show, via stable isotopic labeling, what is the effect of meal patterns in adults, NOT CHILDREN, on lead uptake for essentially one individual. This study was not statistically designed to serve as a human epidemiological study of lead bioavailability in the Basin under the full range of exposure scenarios at issue in the HHRA. It is also irrelevant to say we don't have uptake data in the Basin, since the Region 8 studies make it clear that the two bioavailabilities (uptakes) used in the HHRA are not only appropriate, but may even underestimate uptake in some cases.

p. 34, Soil/dust relationship The HHRA used the IEUBK model within all of OSWER's guidance for its use and within all the advisories provided by EPA's Technical Review Workgroup for Lead. That is countered by the commenters by their noting that against this framework, there is some analysis data out there showing a statistical association. The commenters also ignore that, in the HHRA, it is stated that paint's role in association with Pb-B is itself attenuated to non-significance when we use community lead levels in the modelings. This suggests that the "paint" measure in these statistical analyses is a surrogate variable for something else going on in lead pathways. Otherwise, why would "paint" be subject to broader soil lead in the community???

p. 34, Dietary lead intake The commenters are creating a straw issue, in arguing that the default diet intakes need to be (moderately) adjusted downward. They then proceeded to try to demolish what’s done in the HHRA modeling. This is nonsense. First, a single study by Griffin et al. chose to use an estimate, and this is not an EPA policy. Secondly, and more important, diet defaults are a minor part of the total lead intakes and uptakes, especially with soil and dust lead levels at issue here. One can substantially change a minor input and still not have a major impact on output. This is misleading in the extreme. Here again, we have contradiction. The commenters argue that diet has to be fine-tuned, yet offer a blanket rejection earlier (see my earlier responses) about garden crops having any role in the diet.

p. 35, Maternal blood lead concentrations

1st par. This large paragraph is erroneous, and its use by the commenters is hard to take seriously. That is, it attempts to use national data to characterize what women's Pb-B values should be in the Basin. The HHRA points out why one can't use national data. The commenters either did not read this passage or they are deliberately ignoring it, an objectionable practice. I refer the commenters to that section and the basis of the statements, in the ATSDR report to Congress on childhood lead poisoning. The statements in the ATSDR report were assembled

and reviewed by the National Center for Health Statistics, an agency of biostatisticians who were also the principal architects of the NHANES surveys, and are therefore a credible group whose statements are certainly more authoritative than those of the commenters.

p. 35, Combinations of exposure pathways and scenarios

1st par. Comments about over-counting do not appear credible, since they appear to imply that different-aged kids and different receptors in and out of families can have different exposures than younger kids, but exposures that do occur for older kids somehow comprise over-counting exposures for younger kids. These assertions don't seem to be clear or logical. The HHRA does not appear to invoke scenarios, baseline vs. incremental, that have any substantive hazard of over-counting.

2nd par. The comments about inappropriate use of RMEs for any IEUBK use. because the model estimates a CT in Pb-B, etc., appears (as they state the problem) to be a theoretical misreading of the model's uses. One can use any set of environmental media lead for input lead to the model as would be deemed appropriate. What the model basically says is that when you input either a community mean (GM or AM) or whether you use pairs of data for each of the residents, or whether you use a segment of some distribution of environmental lead levels in whatever media, the output is a CT for whatever that input is. If one selects a group of children at the high end of a distribution of soil leads, and then asks the question, what is the distribution of outputs given that input, the model answers the question accordingly.

p. 36, Application of the adult lead model The commenters simply offer a set of comments in general, vague terms without offering anything to respond to.

p. 37, Absorption fraction of lead from soil These comments are incomprehensible, in that they misstate what the IEUBK model's default bioavailabilities are and misinterpret other points as well. First, the soil lead uptake default for the model is 30%, based on 60% relative bioavailability for soil compared to 50% assumed absolute bioavailability for diet and water, i.e., 50% X .6 = 30%. Where does the 12%, i.e., 20% x .6, come from? See my comments earlier about the Maddaloni et al. study.

p. 37, Geometric standard deviation This topic was already introduced and stated in the mining industry comments for children, with little specific for adults. See my responses to the earlier GSD section.

p. 38, Timing of exposure The comments are a set of speculative observations which do not rebut the reasonable assumptions about timing and frequency in the HHRA. They require no response.

p. 38. Characterization of modeling results. Again, the comments for the discordance or concordance of model vs. measured results are largely qualitative, speculative comments that require no response.

p. 39, Other modeling The commenters ask that other models be considered. The Griffin et al. 1999 model is just a modeling group's use of probabilistic technique. EPA has not replaced the point-estimate approach for the IEUBK model, nor does it appear at this early point that PRA offers any superior alternative to what was done in the Basin for model-driven risk characterizations.

pp. 39 et seq. F. Characterization of Non-Lead Health Risks

This section principally deals with various aspects of arsenic contamination, exposure and risk characterization. The responses are organized by the individual topics presented by the commenters, except for those instances where I have already made detailed responses.

p. 40, pars 1,2: Use of chronic RfD for childhood exposures to arsenic I have already responded at length on this topic.

p. 40, bottom to top of p. 41, Observations of arsenic health effects in U.S. populations The gist of this par. is to say that if As were so potent in terms of cancer potency then we should be having a lot of discernible cancers. However, this has been refuted, whatever the surface appeal underlying this comment, by one having to first calculate all the water As and food As distributions and then estimate, using population densities, what the risk rates, or for that matter, prevalences and incidences, should be. Papers by Smith and coworkers at Berkeley include discussions that address this issue a number of times. These workers have been heavily involved in the epidemiology and biostatistics of population studies of As exposure and mortality/morbidity. The earlier citations are included in the paper cited earlier, Mushak and Crocetti, 1995 in EHP. A newer citation of Smith et al. that has discussions relevant to this issue are:

Ref: Smith AH, Goycolea M, Haque R, Biggs ML. Marked increase in bladder and lung cancer mortality in a region of northern Chile due to arsenic in drinking water. Am. J. Epidemiol. 147: 660-669 (1998).

The Lewis data was highly biased against applicability to the general U.S. population. Unlike the Utah study cohort, the U.S. population is largely not Mormon, not as non-smoking, not as non-drinking, not as healthy, in terms of SES and

associated health risk factors, potentially all risk factors that affect the expression of adverse effects of contaminants. However, the commenters appear to ignore that what the Lewis et al. data show is that As levels in these Utah communities are associated with cardiovascular risks.

p. 41, Risks associated with low dose levels The NRC 1999 report, like NRC reports in general, is more authoritative on matters of arsenic cancer models than an isolated EPA meeting four years earlier. That NRC report stated explicitly one could not reject linear low-dose extrapolations for cancer risks from low As intakes. The commenters strangely attempt to elevate the lesser of two sources of authority and to attenuate the more authoritative view. Science is at odds with this strategy. See my earlier responses on this topic.

p. 41, Risk calculations The calculations are straightforward, unlike the impression the commenters wish to convey.

p. 42, Risk characterization results One cannot say, at this point, whether individual effects from COPC metals will be additive or not. Additivity or its rejection requires knowledge of the mechanisms of toxic action for these Basin contaminants. Knowledge to reject inter-organ or inter-tissue toxic interactions is not known by science or the commenters. At this point, additivity is not far-fetched.

The issue of using as child risk reference for As an RfD intended for lifetime exposures was addressed in my earlier comments.

Last par. The issue of tribal subsistence exposure factors and their use for the HHRA were discussed in my earlier responses.

SECTION 4.0
COMMENTS FROM THE TECHNICAL REVIEW WORKGROUP FOR LEAD

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

Transmittal Memorandum
Technical Review Workgroup for Lead (Pb)

An interoffice workgroup convened by
Office of Solid Waste and Emergency Response

Date: October 19, 2000

Subject: Review of Human Health Risk Assessment for the Coeur d' Alene Basin

From: Mark Maddaloni
Kevin Koporec
Co-Chairpersons of the Technical Review Workgroup

To: Sean Seldrake, EPA Region 10

The TRW greatly appreciates the opportunity to review the *Human Health Risk Assessment for the Coeur d'Alene Basin*. Please find attached a compilation of the TRW's comments. Please note that the attached report has not undergone a full review by the TRW. The report was prepared by a sub-committee of the TRW and, after discussion of the review in a TRW teleconference, and in consideration of the urgency of delivering the report to Region 10, it was decided that the report would be forwarded directly to Region 10 without full TRW review.

Please do not hesitate to contact either Mark Maddaloni or Kevin Koporec if there is any way that the TRW can be of further assistance to Region 10.

Respectfully,

Mark Maddaloni, co-Chair, Region 2

Kevin Koporec, co-Chair, Region 4

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

TECHNICAL REVIEW WORKGROUP FOR LEAD

Technical Memorandum

Review of Human Health Risk Assessment for the Coeur d'Alene Basin

Prepared for

Sean Sheldrake
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October, 2000

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U.S. Environmental Protection Agency

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Table of Contents

1.0 Introduction 1

2.0 Major Comments 3
2.1 Relative Merits of Using the IEUBK Model in Community-mode or Batch-mode . 3
2.2 Evaluation of Alternative Approaches to IEUBK Modeling 4
2.3 Use of Blood Lead Survey Data 9
2.4 Use of the EPA Adult Lead Methodology (ALM) 15
2.5 Assessment of Incremental Lead Intakes and Associated Health Risks to Children 19
2.6 Environmental Data Sampling and Quality Assurance 21

3.0 Comments and Recommendations in Response to Region 10 Priority Issues 23

4.0 References 31

Attached Figure

Appendix A

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1.0 INTRODUCTION

1.1 Charge to the TRW

This report summarizes comments of the EPA Technical Review Workgroup for Lead (TRW) on the *Human Health Risk Assessment for the Coeur d'Alene Basin Extending from Harrison to Mullan on the Coeur d'Alene River and Tributaries Remedial Investigation Feasibility Study* (July 2000, Public Review Draft) (referred to in this report as the CDAB HHRA, or the HHRA). EPA Region 10 requested this review *to ensure that the HHRA is technically sound and consistent with EPA policies* (August 1 memorandum from Region 10 to TRW). The Region requested that the TRW give attention to the following priorities related to the assessment of lead risks:

- Is the Risk Characterization transparent, clear, consistent, and reasonable?
- Does the Uncertainty Discussion provide context for the risk results?
- Do the predicted house dust concentrations associated with various yard soil action levels support subsequent blood lead predictions and Preliminary Remediation Goals derivations?
- Does discussion of blood sampling methods, participation rates, and age distribution (which changed over time) help to interpret the blood lead screening results?
- Is the discussion of the results from the two modeling approaches sufficient to support risk management decisions protective for human health risks from lead?

The CDAB HHRA included an extensive assessment of exposures and risk associated with chemicals other than lead. These portions of the HHRA were not the subject of the TRW review.

1.2 Documentation and Data Reviewed

Documents provided to the TRW for this review included the CDAB HHRA report (July 2000 Public Review Draft) and various supporting memoranda and data tabulations provided by the Region at the request of the TRW, usually in response to requests for clarification of portions of the HHRA or to supplement knowledge of the historical background of the Basin assessment. Within the CDAB HHRA are contained the following types of information which the TRW reviewed:

- summaries of blood lead, soil, and dust lead measurements made during sampling events that occurred in the period 1996 - 1999;
- summaries of the results of correlation and regression analyses of PbB and

environmental exposure levels of lead;

- summaries of results of simulations run with the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK model), both community and residence batch runs;

- summaries of the results of applications of the EPA Adult Lead Methodology (ALM);

- results of a sensitivity analyses and risk reduction predictions;

- an uncertainty assessment.

Actual data inputs used in IEUBK model runs were not available to the TRW and, therefore, could not be reviewed, and predictions made using alternative inputs could not be compared with those in the HHRA.

2.0 MAJOR COMMENTS

2.1 Relative Merits of Using the IEUBK Model in Community-mode or Batch-mode

Section 6.6.1 of the CDAB HHRA presents child risk estimates that are based on community-mode and batch-mode IEUBK model runs. In the community mode, geometric mean exposure levels for house dust and yard soils for a given Conceptual Site Model Unit (CSMU) were used as input to the model to predict the geometric mean blood lead concentration and P_{10} for the CSMU. In the batch mode, house dust and yard soil lead levels for each residence were used as input and a geometric mean blood lead concentration and P_{10} were predicted for each residence. The corresponding CSMU values were calculated as the arithmetic means of the individual residence values.

The TRW supports the HHRA in not relying on the results of the community-mode runs to estimate community risk at CSUs or to estimate clean up levels. It also recognizes the utility of the uses of the community-mode runs in the HHRA as part of an exploration of the potential impacts of community yard soil and house dust exposure on risk, and in an analysis of the sensitivity of the model to variations in soil and dust lead levels, as a precursor to using the batch-mode runs to estimate soil clean-up levels (see Section 6.7.6, p 6-55 of the HHRA).

However, the TRW strongly agrees with Section 7.4.4 (p 7-39) of the CDAB HHRA which states the major limitations of the community-mode approach:

> *Use of the community mean input approach and subsequent estimation of community blood lead level means and blood lead level distributions is the least computationally and conceptually desirable of the various approaches that can be employed. The community approach subsumes too much uncertainty simply because it attenuates heterogeneity of lead exposures, and understates the most revealing depictions of blood lead distributions. For this reason, the IEUBK model's user manual (USEPA 1994a, b) discourages use of the model at this insensitive, gross level.*

EPA guidance stresses that, for the purpose of supporting remedial decisions for residential contamination, risk assessment approaches should focus on children who receive their principal lead exposures in the immediate vicinity of their homes (U.S. EPA, 1994). The batch-mode is the preferred approach to this end, because it ensures that risks at each residence are integrated into the site risk estimate.

While EPA guidance focuses on the need to evaluate risks for children at their homes, guidance also recognizes that other exposure scenarios can be important and should be considered where non-residential sources may make an important contribution to lead exposures in a community. In populations where young children spend a large amount of

time at locations other than their homes (e.g., neighboring yards, homes of relatives, etc), risk estimates based only on exposure of individual children at their homes may not accurately capture risks associated with each child's actual exposure. At such sites, it may be desirable to include exposures from these community areas in the batch-mode runs. This could be accomplished, for example, by using the multiple source dust model in the batch mode (not in the community mode). Alternatively, activity of the child could be distributed between home yard and community areas having different mean soil lead concentrations, and a time-weighted average used as input in the batch mode. This approach is represented in the HHRA in the application of the IEUBK Box model, although there are other issues associated with this model (see Section 2.2 of this report for further discussion of the Box model).

The community-mode approach was explored in the HHRA as a method for capturing community-wide residential exposures in the risk estimates. However, as suggested in Section 7.4.4 of the HHRA, the results obtained from the community approach should be interpreted with caution, as there may not be any children in the community that are exposed to the actual calculated mean (geometric or arithmetic) soil and dust lead concentrations. Only if a children randomly accesses all yards within the community equally could we expect over time the average exposure concentration for any child to be represented by the community mean exposure level. Random accessing of all yards over a given year (the exposure time step of the IEUBK model) would represent an extreme scenario at many sites, but may reflect the activity patterns of children in the relatively small communities within the CDAB. If this is not the case, then risks at any individual residence may be underestimated or overestimated by community-mode predictions, depending on whether the exposure levels at that residence are lower or higher, respectively, than the community average. The estimates may also be affected by other variables. For example, the relative contributions of home or community exposures may depend on the age of the child in a given home, the presence of older siblings, the geography of the community, or local activity patterns and social customs of the community

2.2 Evaluation of Alternative Approaches to IEUBK Modeling

2.2.1 General Comments

Two approaches were used to estimate lead risks in the CDAB. One approach used the IEUBK model with site-specific exposure inputs and all other parameters kept at default values. In the HHRA and in this report, this model is referred to as the IEUBK default model. A second approach used the IEUBK model with site-specific exposure inputs, an adjusted bioavailability factor (18% total percent available), and a time-weighted soil lead contribution from the residential yard and neighborhood (dust: home yard soil: community yard soil ratio, 40:30:30). These adjustments were based on calibration exercises conducted as part of a Five-year Review of the of the Bunker Hill Superfund Site (BHSS,

TerraGraphics, 2000). The adjusted IEUBK model is referred to in the HHRA and in this report as the IEUBK Box model, to distinguish it from the IEUBK default model. The HHRA presents risk estimates, as well as assessments of post-remediation risks assuming various clean-up action levels, based on both the IEUBK default and Box models.

The TRW supports HHRA in not relying exclusively on the IEUBK Box model to estimate pre-remediation risks in the CDAB (i.e., percentage of children exceeding 10 µg/dL, P_{10}). The Box model was calibrated to agree with the downward trend in post-remediation blood lead concentrations observed at the BHSS. Factors that may have affected this downward trend (e.g., decreased soil and dust intakes resulting from intervention and educational efforts) may not be operating or may not be as important in the CDAB. Ideally, if adjustments were to be made to the IEUBK model for its application to the CDAB, such adjustments should be based on the available information about exposures and blood lead concentrations in the CDAB and not at the BHSS. However, the extensive experience at the BHSS could be applied to the CDAB if there were a better understanding of the exposure factors that contributed to the downward trend in the blood lead concentrations at the BHSS, and whether or not these same factors affect blood lead concentrations to the same degree in the CDAB.

Aside from the extensive data base on the presence of lead contamination in the CDBA, the HHRA does not present site-specific data applicable to estimating specific parameters of the IEUBK model (see further discussion below). In the absence of data to estimate specific parameters, consideration of non-default choices can be useful for range-finding and sensitivity investigations. The blood lead concentrations and risk estimates based on the Box model represent an example of this, in that the Box model imposes certain assumptions that are thought to be valid at the BHSS, and the differences between the predictions made with the default and Box models show the impact of these assumptions. For example, if the fractional absorption of lead is lower than the default values, and there is a 50% contribution of community yard soil to soil lead intake in the CDAB, then the predicted blood lead concentrations will be lower than those based on the default model. The results of the Box model runs are interpreted from this perspective in the HHRA (see Section 7.4.4, p. 7-41, HHRA). At this time, there does not appear to be an adequate basis for determining which of the two models provide more accurate risk predictions in the CDAB. However, the differences in the predictions from the two models are not large, given uncertainties associated with both models, and it could be readily argued that actual risks fall within the range of predictions from the two models. Comparisons of the mode predictions with observed blood lead concentrations do not completely resolve this issue because of uncertainties regarding the representativeness of the blood lead data. These uncertainties are discussed at length in the HHRA (Section 7.4.1) and in this report (see Section 2.3 of this report). However, uncertainties not withstanding, the blood lead data do not exclude predictions from either model as being applicable to the CDAB.

It is also important to note that the soil and dust measurements used in the IEUBK model represent the 175 µm fraction, rather than the 250 µm fraction that is more commonly used

in CERCLA site assessments. While, as is explained in the HHRA (Section 7.4.2, p. 7-29, HHRA), the smaller particle size fraction may better represent the fraction that adheres to the hands of children, it also is likely to have been enriched with lead, relative to the 250 µm fraction. The TRW has recently provided clarification and further guidance on this issue (U.S. EPA, 2000). Therefore, risk estimates based on the 175 µm fraction would be expected to be higher than those based on the 250 µm fraction from the same samples. This introduces an additional conservative (health protective) bias into the risk estimates. Another way to view this, is that, had the 250 µm fraction been used as the basis for the soil and dust concentration terms, the risk estimates based on the IEUBK model would have been lower by some unknown degree. The use of the 175 µm soil and dust fractions also has relevance to the interpretation of the bioavailability adjustment used in the IEUBK Box model (see below).

2.2.2 Bioavailability Adjustment

The bioavailability value of 18% was applied as an alternative to the model default of 30%. No data specific to the bioavailability of lead in soil and dust at CDAB are discussed in the HHRA and such data apparently have not been generated at the site. The TRW's short sheet, *IEUBK Model Bioavailability Variable* (U.S. EPA, 1999a), discusses methods that can be used to study bioavailability of lead and which have been used in practical applications for other Superfund sites. The TRW recommends that bioavailability studies of soil and dust, or other relevant data, should be used to support a site-specific bioavailability value for the CDAB. However, as a means to provide information regarding the sensitivity of model predictions to this parameter, consideration of alternate bioavailability values, such as that used in the Box model, can provide useful information.

The TRW understands the intent in the HHRA in interpreting the bioavailability adjustment as a surrogate for adjustments in one or more of several variables that relate soil and dust exposure levels to the amount of lead taken up into the blood (see Section 7.4.4., p. 7-41, HHRA). However, the TRW does not endorse use of the bioavailability term in this way. Segregating the various factors that may affect lead uptake would allow one to consider the potential effects of these factors that may influence uptake of lead by children in the CDAB. For example, the CDAB lead concentration data are based on samples screened to a 175 µm sieve size. This may provide relatively conservative estimates of the lead concentration compared to a more common practice of using a 250 µm sieve size. To the degree that concentration estimates tend to be conservative, so would estimates of lead uptake in the model runs (see below). There is also a potential for some decrease in the soil and dust ingestion rates for children in households where health concerns about lead may have caused parents to use increased care in cleaning and supervision of children's activities.

Another uncertainty in extrapolating a bioavailability factor for the CDAB from BHSS data is that it is possible that exposures in the CDAB may be a mix of lead from the smelter and lead from mine wastes, or other sources, which may have different absorption fractions.

The relative contribution of these sources may change with location in the CDAB (e.g., with upwind or downwind from the smelter, or up or down gradient from the smelter), and may change with remediation. For example, at some locations in the CDAB, historic smelter emissions may contribute more to lead in house dust than in yard soils. If lead in smelter dust has a different fractional absorption than lead from other sources, removal of yard soil may change the absorption fraction of the lead to which children would be exposed at that location. There is some support for this possibility in the BHSS, where the calculated bioavailability factor which resulted in better agreement between the IEUBK model predictions and observed blood lead concentrations changed (increased) over time as the remediation proceeded (see Appendix Q, HHRA).

Since the bioavailability adjustment had a pronounced impact on predicted blood lead concentrations and risk estimates, it would be informative to more directly display in the assessment the effects of changes in bioavailability (either directly or as a surrogate modifying lead uptake) on lead risk predictions. This might take the form of graphs and tables that show a range of choices for the parameter value and resulting changes in risk. Given the lack of information specific to bioavailability, such presentations could show the effect of a potential site-specific modification to lead uptake through undetermined mechanisms. An example of this is provided in the attached Figure 1 which shows the impact of various assumptions about lead enrichment in the 175 µm fraction relative to the 250 µm fraction on lead risk. The TRW notes that the IEUBK modeling assumptions regarding bioavailability (or more generally lead uptake) need not be linked exclusively to the multi-source soil exposure scenario presented in the Box model.

2.2.3 Partitioning of Source Contributions to Soil Dust Ingestion

Exposures for children at sites other than their homes were incorporated into the Box model results (using the batch mode calculations) by assigning to each a child a fraction of total soil exposure at home and a fraction of total exposure to an *average* community yard soil concentration (i.e., house dust: yard soil: community soil ratio, 40:30:30). This scenario would have particular relevance for those (often older) children who would spend much of their time away from home playing at a variety of residences, parks, or other areas in the community.

The basis for the 40:30:30 ratio derives from structural equation modeling of the data from the BHSS, which indicated a significant effect of community yard soils on blood lead concentrations (Appendix Q, HHRA). The use of this ratio in modeling lead risks in the CDAB assumes a similar community yard soil contribution in the BHSS and CDAB. The HHRA concludes that this is the case from a stepwise regression analysis of the CDAB data (Section 6.4.2, p. 6-23), This, together with the experience at the BHSS and the expected similarities in the Basin communities, in terms of behavior patterns of children, were the empirical bases for retaining the 40:30:30 ratio in the application of the IEUBK Box model to the CDAB. Although this is a major conceptual change from the default model, the impact of use of the 40:30:30 ratio on risk estimates appears to be relatively minor; the

difference between the predicted blood lead concentrations when the default ratio of 55:45 or the 40:30:30 (or a 75:18:7) ratio were assumed in the model was relatively small (Table 4-28, Appendix Q of the HHRA). Thus, from a risk assessment perspective, the modification is of minor consequence.

The concept of including a community contribution to soil lead intake deserves further comment because of its potential utility at other sites. EPA guidance has encouraged the consideration of alternate sources of dust lead intake, other than that occurring at the home. This is the rationale for including the alternate dust source option in the IEUBK model. In the HHRA, the community average of yard soils was used to represent the soil lead concentration of the fraction of the soil lead exposure occurring away from home. There are many sources of dust in a typical community, such as deposition from industrial activity and vehicular traffic, that are not derived from soil. Consequently, an aggregate of individual property soils cannot fully represent community dust exposure. Nevertheless, the use of soil data in the absence of data from these other sources has the effect of assuming that the concentration in the unmeasured sources is the same as the aggregate community soil, not that the unmeasured sources do not exist. The community average serves as a reasonable central estimate in the absence of any information on additional sources of community dust or the behavior patterns of specific children. As an example of the potential utility of this measure, highly mobile children who lived at residences with *clean* soil (e.g., after yard remediation) may still have elevated risks due to access to lead at other yards in the community. The TRW would caution, however, that a community *average* concentration term is a non-specific measure. Risk calculations in which a child's exposure is assumed to be represented by a time-weighted average of home and community average values, may serve to indicate the importance of community wide lead sources for a highly mobile child. However this approach is of limited value in supporting clean-up decisions for specific non-home properties, for example daycare centers, schools, roadsides, and other public areas. A more useful alternative for these types of exposures would be to model results specific to contamination levels at specific schools, daycare locations, local parks (see Section 2.5 of this report for further discussion).

A specific technical concern pertains to how community *average* concentration values were calculated for use in IEUBK modeling (as applied in the Box model and in *community* mode calculations with the default model). As noted above, a rationale for use of a community average concentration term is that an (idealized) highly mobile child would be exposed to contamination throughout a community and the summation of these many events of contacting different concentrations would be equivalent to exposure to a lead concentration equal to the community average values. Under the circumstances where this scenario is applicable, explicitly calculating the summation of exposures will lead to the use of an arithmetic mean and not a geometric mean exposure concentration term. This may be illustrated with an example. Suppose that on three days, a child is exposed at three different locations with lead concentrations of 30, 300, and 3000 ppm. Further, assume that on each day the child ingests 0.1 g of soil at the exposure location. Therefore on the three days, the child has a lead intake from soil of 3 µg, 30 µg, and 300 µg, respectively (30 µg/g

x 0.1 g/d x 1 d = 3 µg, etc.) The daily average lead intake for this period is 111 µg/d. For comparison, the arithmetic mean soil concentration at these locations is 1110 ppm and the geometric mean concentration is 300 ppm. Daily intake rates calculated using the arithmetic mean soil concentration value reproduce the daily lead intake level for the child (1110 µg/g x 0.1 g/d = 111 µg/d). However, an intake calculation using the geometric mean understates daily lead intake by more than a factor of three (300 µg/g x 0.1 g/d = 30 µg/d).

2.3 Use of Blood Lead Survey Data

Substantial efforts have been made to collect data on blood lead levels on children and adults living in the CDAB. The HHRA reports that through a combination of efforts in 1996-1999, 524 blood samples representing 424 children under age 9 years of age living in 843 households were collected in the Basin.

The blood lead data in CDAB were collected as a public health service provided to Basin residents and have been utilized by local public health authorities (Idaho Department of Health and Welfare) to provide advice and assistance to children found to have elevated blood lead levels. The HHRA reports that 50 children received follow-up assistance due to the detection of blood lead levels above 10 µg/dL. The majority of children re-screened after public health intervention showed a reduction of blood lead levels from their prior elevated levels indicative of a benefit of this intervention program.

The effort to screen children for elevated blood lead levels in the CDAB comports with CDC recommendations. CDC guidance succinctly describes the value of blood lead screening programs:

> *Blood lead screening is an important element of a comprehensive program to eliminate childhood lead poisoning. The goal of such screening is to identify children who need individual interventions to reduce their BLLs [blood lead levels].*

Blood lead screening may or may not provide data that is representative of the population of concern.

The blood lead screening data in the CDAB also serves the important purpose of demonstrating the presence of continuing risks of lead exposures to the Basin children. Basin wide, 12.5% of tested children up to 7 years of age had blood lead levels above 10 µg/dL (see Table 6-4c, HHRA). In some communities in the Basin, the risks were higher: 22%, Burke/Nine Mile; 19%, Wallace; 14%, Kingston; and 25%, Lower Basin. Risks of elevated blood lead levels were also higher in the younger groups of screened children. Basin wide, 19-26% of tested children one to three years of age had blood lead levels above 10 µg/dL. In some communities, in the Basin the risks were higher in this age group: 50%,

2-3 years, Burke/Nine Mile; 22-40%, 83% 1 years; Wallace; 2-3 years; Kingston; 20-50%, 1-3 years, Lower Basin (the smaller numbers of children make these figures less accurate) (Table 6-5, HHRA). These results serve to demonstrate the need for further attention to reduce sources of lead exposure in the Basin and the need to continue screening and interventions to reduce lead exposures.

However, in interpreting these data it is important to recognize that blood lead screening efforts were not intended to constitute a research investigation of subjects living in the Basin. Individuals were not randomly or systematically chosen for screening as part of a statistical study. Therefore, the screening data must primarily be interpreted as information regarding the children and families who desired screening. It should not be assumed, in advance of careful examination, that the data on screened children is also representative of the majority of children who did not participate in the screening programs. This issue is discussed in some detail below.

Blood lead data collected in the CDAB were used in the HHRA in three ways: 1) to characterize age-related and geographic patterns of excessive blood lead concentrations; 2) blood lead concentrations predicted from the IEUBK model were compared to observed blood lead concentrations in order to assess the effectiveness of various assumptions made in the model for describing current blood lead concentrations; and 3) blood lead data were used in correlation and regression analyses to evaluate relationships between environmental levels and blood lead concentrations in the Basin.

The HHRA takes great care in discussing the limitations of the blood lead data for the above three uses in the risk assessment (see Section 7.4.1, HHRA). The TRW supports the uncertainty assessment of the blood lead data that is presented in the HHRA. In reviewing the documentation for the blood lead data in the HHRA, the TRW arrived at a similar conclusion; that several issues limit interpretations of both the empirical comparisons and the regression analyses. These include: 1) representativeness of the data with respect to the Basin community; 2) sampling bias; and 3) the potential effect of intervention on blood lead concentrations in the community (see detailed discussion below). The TRW concluded that the information presented in the HHRA that relate to these issues suggests that the data do not provide an adequate basis for reliably estimating central tendency blood lead concentrations, percentiles or the percent above 10 µg/dL, or other population parameters. Therefore, the data should be used with great caution and with appropriate consideration of the uncertainties associated with the method of solicitation of participants in the survey, particularly if it is used to characterize blood lead levels in the community. This has particularly important implications for extrapolating any results of these analyses to areas of the CDAB not sampled, or to extrapolations over time, such as post-remediation blood lead concentrations. In view of the limitations of the blood lead data, the TRW supports the approach adopted in the HHRA of basing risk estimates on the results of the IEUBK model runs. This approach is consistent with EPA Office of Solid Waste and Emergency Response (OSWER) guidance (U.S. EPA, 1994). The observed blood lead concentrations support the general outcomes of model runs, that the risk of exceeding a

blood lead of 10 µg/dL is greater than 5% is substantial for children who live on many of the properties in the CDAB. A more detailed discussion of the blood lead data are provided below.

2.3.1 Representativeness of the Data

General Issues Concerning Representativeness

The TRW supports the HHRA in its conclusion regarding the blood lead survey data (Section 7.4.1, p. 7-23, HHRA):

> *The nature of this turnout* (i.e. participation in the blood lead surveys) *raises questions about the reliability of using these data in the HHRA and subsequent remedial decisions.*

Blood lead data can provide information on relationships between environmental exposures and blood lead concentrations of individuals in the sample group; however, if such analyses are to be extrapolated to the general population of interest, in this case, residents in the Basin, the blood lead data must represent the entire CDAB population. A sample is likely to be representative if non-biased sampling methods are employed, such as random sampling (equal probability of selection of any individual or home) or stratified random sampling (probability of selection of any individual or home depends upon which strata to which they are assigned. If the sample is not random, it may have a bias which may result in the sample mean not reflecting the CDAB mean (this also applies to other descriptive variables of the sample and corresponding CDAB population parameters). A biased sample may still be used to estimate CDAB parameters, however, to do this, an understanding of the nature and quantitative effect of the bias is needed so that sample estimates can be adjusted to account for bias.

From the outset, the collection of blood lead data in the CDAB was never intended to provide a random sample for an epidemiological study. Blood lead data were collected as part of a public health service provided to CDAB residents. Thus, it would only be fortuitous if the sample turned out to approximate a random sample. Furthermore, data were not collected to specifically evaluate biases in the sample, although some data were collected that may be useful for this purpose.

The lack of a random sampling design in the blood lead program presents challenges for use of the data in the risk assessment, however, it should not preclude all use *a priori*, as the data do provide valuable information on a substantial number of children. In evaluating the data, all factors that might contribute to bias in the estimates need to considered and potential biases need to be identified and quantitatively explored, if possible. An exploration of information available to evaluate and adjust for sample bias is provided in the HHRA (Section 7.4.1, HHRA) and potential approaches are described in Section 2.3.2 of this report.

CDAB Sampling and Sample Size

If the sample is random, it can adequately represent the population even if it contains a relatively small fraction of population. However, concern for the representativeness of the sample increases as the fraction sampled becomes small. One of the concerns about the CDAB sample is that it captured a relatively small fraction of the target population. Child blood lead data used in the HHRA derive from surveys conducted during four consecutive summers, 1996-1999. In 1996, a CDAB-wide survey was conducted which attempted to capture all potentially impacted homes within one mile of the Coeur d'Alene River (essentially the entire flood plain), excluding the BHSS. In 1996, there were approximately 6252 homes in the CDAB. Among these, 2700 homes were identified as potentially subject to lead or other metal exposures and residents at 843 homes agreed to participate in the survey; blood samples were obtained from 98 children (ages 9 m-9 yr), or approximately 9% of the estimated number of children in the CDAB in identified impacted areas (1025-1120, p 6-9 of HHRA). Approximately 200 additional homes were sampled in subsequent sampling years. In 1997, samples from 26 children were collected in the impacted areas, 11 of whom had been sampled in 1996. In 1998, samples from 128 children were collected and 272 children provided samples in 1999. Thus, the total number of samples available for the assessment was 524. Approximately 100 children were sampled twice, therefore, the total number of children represented in the sample was approximately 424. This represents an unknown fraction of the population of children that lived in the CDAB over the four-year sampling period, including children who may have entered (included births) or left the area since 1996. The fraction of the children sampled may have varied across communities in the Basin.

In addition to differences in sample size, there were other notable differences in the four surveys. The HHRA does not provide much information on the sampling approach used in 1997 or 1998, for example, the extent to which it may have been targeted to certain groups of people, or geographically distributed within the Basin. The 1999 survey offered a cash incentive for participation, and was more aggressively promoted within the community (p. 6-9, HHRA).

2.3.2 Sources of Bias and Approaches to Evaluating Bias

Given the sampling objectives and approach, and the relatively small fraction of the population sampled, bias is a concern in extrapolations made from the sample to the CDAB population for the following reasons. Among the sampling data, the 1996 study came closest to being a systematic effort to capture all residences in the CDAB. However, because blood samples were obtained from only 9% of the potentially impacted children CDAB in 1996, there is no assurance that this study was representative of the community.

In the 1997 -1999 screening efforts, community residents were asked to take the initiative to bring their children into clinics for blood sampling. While a higher participation (272

children) was achieved in the 1999, the entirely self-selected nature of the participants reduces confidence that this sample would be representative of the non-sampled members of the community. It should also be noted that the later screening efforts did not limit participation to children from areas likely to be impacted by metals contamination, as was the case in 1996. As a result, that the numerically greater number of participants in1999, relative to1996, may have included a larger fraction of children who lived in areas that had lower potential for contamination.

Other data may be available to help judge the likelihood that data for screened children would be likely to be representative of the community as a whole. Relevant information would include consideration of factors that may be associated with lead risks such as age, residence in more contaminated locations, residence in properties in poor repair, and socioeconomic status. Data to allow a comparison of demographic characteristics of screened children and the community as a whole are unfortunately very limited. Data on factors such as socioeconomic status were not collected for screened children (unless a high blood lead value triggered a home intervention) and, therefore, cannot be compared with the larger community. However, age is one significant risk factor for which there is comparative data, and unfortunately, the younger groups of children that are at highest risk are substantially under represented in the group of screened children. This indicates that, taken as a whole, the screened group may be at somewhat lesser risk of elevated blood lead levels than the community at large. The deficit of young children in the screened group also indicates that the factors that motivated parents to participate in screening were not reflective of lead risks as they would be evaluated by public health professionals.

The HHRA discusses different hypotheses that have been offered concerning the potential biases in the available blood lead data (Section 7.4.1, p. 7-22, HHRA). One set of arguments suggests that parents with a greater level of concern about lead risks elected to have their children participate in screening. Such parents would be likely to act on their health concerns so as to limit their children's exposures to lead (e.g., limiting places of play, more contentious cleaning of dust at home or attention to hand washing and other hygiene measures). The TRW believes that this proposal has plausibility and that it corresponds with concerns of TRW about potential biases in some blood lead investigations conducted at other sites.

Alternately it has been contended that in the 1999 screening event, where the participation rate was greatest, the payment of a 40 dollars compensation to participants would have resulted in a disproportionate participation by lower income families. It is then argued that children in lower income families would have greater risks of elevated blood lead levels. In this regard, the TRW observes that, while socioeconomic variables have been shown to have correlations with lead risks in some other studies, caution needs to be exercised to avoid over interpretation of this issue. First, it is not clear that the payment of compensation to participants was the predominant factor in securing the somewhat larger participation rate in 1999. Considerable additional effort was invested in 1999 to inform and encourage participation in the 1999 survey. Secondly, to the extent that children in

lower income families may have increased risks of elevated blood lead, such a correlation would be expected to result from more fundamental underlying factors, not monetary income itself. Some (not all) families experiencing economic hardship may also lack time or resources to provide for as much supervision of children as they would desire. Therefore, it is not clear that parents in families under such stress would have the option of dropping other commitments to take children in for screening. The TRW does not believe that it is appropriate to make the assumption that parents with lower incomes would provide less attention to environmental risks to their children.

Potential sources of bias can be proposed, and then an evaluation made as to whether or not data are adequate for quantitatively assessing the direction and/or strength of the bias. Examples of potential sources of bias include:

- Neighborhood clustering could result in certain areas of the CDAB being under-represented in the sample (spatial bias).

- Parents with younger children might have been less inclined to provide blood samples from their children. This would result in an age bias in the sample.

- The inclination to allow samples may have been influenced by duration of residence which could have affected knowledge and perceptions of the extent or importance of the problem.

- Differences in socioeconomic status (SES) could affect the inclination to allow sampling; for example, lower SES residents may have placed a higher or lower priority to lead as an issue for their families than higher SES residents.

- Information about environmental lead levels or blood lead levels could have influenced participation in the survey. For example, parents who more strongly suspected that there was a lead problem in their community may have been more motivated to participate.

- Cash incentives for participation (discussed above).

The above examples can be translated into a series of specific queries directed at the existing data to determine if available data suggest or do not suggest bias in selection, or an unequal probability of response. Examples of these that could be explored include:

- Were the sample statistics stable over time?

- Were the responders equally distributed geographically in the CDAB and within the CSUs?

- Did the response rate vary across communities?

- Are SES scores similar in the sample and CDAB?

- Are other demographic variables similar in the sample and CDAB (e.g., age, age of housing, residence time)?

Despite a rather large variation in the level of participation in the blood lead monitoring study over the 4-year period (26-272 children per year), minimum, maximums, arithmetic and geometric means and standard deviations of the sample blood lead measurements remained remarkably constant from year to year (see Table 6-1, HHRA). This would suggest that, if there was a strong bias, it may have been relatively constant from year to year. This outcome would also be expected if the samples were indeed representative of a stable population. On the other hand, the percent participation in the blood lead survey varied with age (see HHRA Table 6-4a). This would suggest a possible age bias or under representation of younger children relative to older children.

2.3.3 Potential Effects of Intervention on Blood Lead Concentrations

Another time-related consideration is the impact of community awareness on the time course of blood lead concentrations within the CDAB. Community awareness can and does play a role in affecting short-term behaviors, through temporary decreases in contact with lead sources and consequent transient decreases in blood lead concentrations. Questioning about hygiene and home conditions at a time preceding blood sample collection may promote actions that would tend to reduce risks of elevated blood lead levels. Since there is evidence that individual level contact with parents is important to the success of intervention efforts (Kimbrough, 1994), such studies may implicitly include an important individual level intervention component. This was most likely the case in the CDAB where the blood lead and environmental surveys were specifically intended as part of public health service to the community residents.

In the CDAB, nurses visited homes where blood lead concentrations were considered to be elevated (greater than 10 µg/dL). Blood lead measurements taken in homes after a nurse visited that home may reflect the impact of the nurse-visit, and may not represent the blood lead that would be expected in that exposure scenario, had the nurse-visit not taken place (e.g. a new resident of the home). It is not clear from the HHRA whether blood lead measurements taken after a nurse-visit were excluded from or included in analyses reported in the HHRA. However, the TRW was advised by Region 10 that, if a second blood lead sample was collected as part of or as a follow-up to a nurse-visit, these data were excluded from the analyses. Therefore, nurse-visits are likely to be less of a factor in analyses of blood lead concentrations measured within a given sampling year. However, it is possible that blood samples may have been obtained from children who lived in homes that received a nurse-visit in previous years.

2.4 Use of the EPA Adult Lead Methodology (ALM)

The ALM was used in the HHRA to estimate Preliminary Remediation Goals (PRGs) for adult non-residential exposures, including occupational exposures and recreational exposures at upland parks and other Common Use Areas (CUAs). The EPA ALM includes algorithms that can be used to predict adult blood lead concentrations associated with site soil lead exposures or soil PRGs (U.S. EPA, 1996, 1999b).

PRGs were estimated based on central tendency and reasonable maximum exposure (RME) assumptions about exposure frequency and soil ingestion rate (see Section 6.5.2, pages 6-31 – 6-33, Tables 6-31 – 6-33 of HHRA). All other inputs to the ALM were default values from U.S. EPA (1996). The central tendency exposure frequency for the occupational scenario was 43 day per year which represented a 5 day per week construction project having a 2-month duration. The RME estimate was 195 days per year, representing a 5 day per week, 9-month (39 week) construction season. For CUAs, the corresponding central tendency and RME frequencies were 16 days per year and 32 days per year, respectively. For upland parks, the corresponding central tendency and RME frequencies were 15 days per year and 30 days per year, respectively. Soil ingestion rates for the three scenarios were as follows (central tendency, reasonable maximum): occupational, 0.1, 0.2; CUAs, 0.05, 0.1; upland parks, 0.05, 0.1.

The TRW supports the HHRA in the decision to calculate PRGs for non-residential soils based on the EPA ALM and supports the general approach used in applying the ALM at the site. However, several details in the application of the methodology were inconsistent with guidance developed by the TRW (U.S. EPA, 1996) and may have resulted in increased uncertainty in the risk estimates (Section 6.6.3, p. 6-46, Tables 6-57 – 6-60, HHRA) . These include the following:

- The EPA ALM should not be used to estimate PRGs for exposures that are less the three months in duration or less frequent than one exposure episode per week. Shorter exposure durations and lower exposure frequencies are not sufficient to achieve a quasi-steady state blood lead concentration, which is a required assumption for use of the ALM for predicting either PRGs for blood lead concentrations. The derivation of several of the parameters in the ALM (biokinetic slope factor and the absorption fraction) is based on steady-state observations. Furthermore, the relevance of the health criterion (10 µg/dL) to short-term exposures is less certain than it is for chronic exposures.

- The averaging time used in the EPA ALM should reflect the actual exposure duration. In the HHRA, the averaging time was the number of exposure days per year divided by the number of days in the year, even when the assumption made in the HHRA was that the exposure occurred over a shorter interval (e.g., 2 months in the occupational scenario). Time-averaging the exposure over a 365-day period,

rather than over the exposure duration, results in higher calculated PRGs.

- In the HHRA, PRGs were calculated with the EPA ALM using the standard (integrated soil and dust pathway) and discrete soil and dust pathway approaches, however, in the later, a value of 1 was assumed for the soil weighting factor. This assumption effectively converts the discrete approach into the standard approach, since it represents a scenario in which there is no dust ingestion. Thus, the calculated PRGs will always be the same for the two approaches if the values of all other parameters the same.

- The PbB_0 parameter in the EPA ALM was assigned a value of 1.7 µg/dL, a value recommended by the TRW to represent non-Hispanic, white adult females, based on national survey data. The use of 1.7 µg/dL is consistent with TRW recommendations for sites where site data are not adequate to support site-specific estimates of PbB_0. However, the HHRA does not quantitatively explore alternative assumptions that could have been made, given the blood lead data collected at the site.

These topics are discussed in greater detail below.

2.4.1 Use of the EPA ALM for Short-term Exposures

The TRW has recommended a minimum exposure frequency of 1 day per week for a continuous duration of 3 months for applications of the ALM (U.S. EPA, 1996). This recommendation is based on the minimum exposures required to achieve a quasi-steady state blood lead concentration. A quasi-steady state is a required assumption in the methodology because the recommended values for the absorption factor and biokinetic slope factor were based on an analyses of data relating lead exposure to quasi-steady state blood lead concentrations. Furthermore, the relevance of the health criterion10 µg/dL) to short-term exposures is less certain than it is for chronic exposures. ALM-based predictions of adult or fetal blood lead concentrations associated with very short exposure durations or infrequent exposures would be highly uncertain and are discouraged for use in risk assessment. In the HHRA, exposure durations of two months for the occupational scenario do not meet these minimum criteria.

2.4.2 Averaging Time in Relation to Exposure Duration

The averaging time used in the ALM should reflect the exposure duration (U.S. EPA, 1996). This allows for a better assessment of a peak exposure period which may result in adverse health effects, and is more consistent with the biokinetics of lead (deposition and release) in the body. For example, if the assumed exposure season (e.g., warm weather construction season) is considered to be 39 weeks, and the exposure frequency is 5 days per week, or 195 days, a more appropriate averaging time would be 39 weeks x 7 days per

week, or 273 days. Similarly, for a short term (3 month) construction project, the concern would be for the peak blood lead achieved during that time period. In this case, 64 day exposure period would be averaged over 90 days. In the HHRA, the averaging time was the number of exposure days per year divided by the number of days in the year. This effectively distributes the lead intake and uptake equally over a one-year period, even when the assumption made in the HHRA was that the exposure occurred over a shorter interval (e.g., 2 months in the occupational scenario). Time-averaging the exposure over a 365-day period, rather than over the exposure duration, results in higher calculated PRGs, which may not provide adequate protection to workers whose activities result in contact with soil.

2.4.3 Use of Soil/Dust Weighting Factor in ALM

The TRW has made recommendations regarding how to use the ALM to calculate PRGs when information is available to quantify discrete intake pathways from soil and dust (U.S. EPA, 1996). The methodology incorporates additional terms for the concentrations of lead in soil and dust (AF_S, AF_D), the mass fraction of soil in dust (K_{SD}), the absorption fraction for ingested dust (AF_D), and the fraction of the total soil plus dust ingestion rate contributed by soil (W_S, soil weighting factor).

In the HHRA, PRGs were calculated using the standard (integrated soil and dust pathway) and discrete soil and dust pathway approaches; however, in the latter, a value of 1 was assumed for the soil weighting factor. This assumption effectively converts the discrete approach into the standard approach, since it represents a scenario in which there is no dust ingestion. Thus, the calculated PRGs will be the same for the two approaches if the values of all other parameters are the same, and therefore, there is no justification for presenting the discrete pathway calculations.

2.4.4 Site-Specific Baseline Blood Lead (PbB_0) in an Uncertainty Analysis

The ALM includes a parameter that represents the blood lead concentration in adults expected at the site if the non-residential soil lead exposure of interest had not occurred. Ideally this should be estimated from blood lead measurements in women of child-bearing age who experience all exposures at the site with the exception of the non-residential exposures of interest, in this case, occupational, and recreational exposures. In reality, obtaining such a sample at a site, and in particular, identifying a representative subset of the population whose blood lead concentrations are not impacted by the non-residential exposures of interest is not always possible. As a result, the PbB_0 parameter is usually assigned a value based on data on other populations, such as national estimates.

In the HHRA, the PbB_0 parameter was assigned a value of 1.7 µg/dL, a value recommended by the TRW to represent non-Hispanic, white adult females, based on national survey data (U.S. EPA, 1996). The use of 1.7 µg/dL is consistent with TRW recommendations for sites where site data are not adequate to support site-specific estimates of PbB_0. However, the HHRA does not quantitatively explore alternative assumptions that could have been

made, given the blood lead data collected at the site. As part of the HHRA, blood lead data were collected in 1996 on 667 adults in the CDBA. Based on the population data presented in Table 3-4 of the HHRA, this would appear to represent approximately 16% of the 4200 adults of ages 15-44 years. Table 6-8b indicates that blood lead samples were obtained from 151 women of child-bearing age, defined as 17-45 years of age. If the sex ratio of this age range in the CDAB was approximately 50:50 (see Table 3-4, HHRA), then the sample would represent approximately 7% of the of women of child bearing age in the CDAB (i.e., 151/2100). The HHRA presents the summary statistics of the blood lead concentrations in this group of adult women, and concluded that the geometric means were 2.0 or less in all areas except Burke/Nine Mile (2.4 µg/dL) and Wallace (2.6 µg/dL). Use of the national estimate of 1.7 µg/dL is reasonable in this case because it would be difficult to make a convincing argument that the blood lead sample was representative of women of child bearing age at the site who did not experience soil lead exposures at recreational sites or from occupational activities. Nevertheless, because the geometric mean blood lead concentration of the sample was higher than the national estimate, it would have been informative to explore the implications of a higher site-specific PbB_0 on the estimates of the PRGs as part of the uncertainty assessment. If a site-specific value for PbB_0 were to be used in ALM, it would have been within the range 1.6-2.6. Most of this range would have yielded lower calculated PRGs if used in the ALM in place of the national estimate of 1.7 µg/dL. This would suggest the possibility that the PRGs may need to be lower than those predicted when national estimates of PbB_0 are applied to the site. A similar type of uncertainty assessment could have been applied to the geometric standard deviation (GSD) parameter in the ALM, based on the observed GSD in the sample of women of child bearing age.

2.4.5 Use of Other Input Parameter Values

The construction scenario is usually considered to be a high-end exposure in a risk assessment; therefore, it is usually not necessary to evaluate both central tendency and RME scenarios. However, it is always useful to evaluate the impacts on both the risk and the PRG when the sensitive parameters are varied. These parameters are usually those relating to the intake and to the exposure frequency and duration. In the HHRA, both the ingestion rate and the exposure duration were varied. The TRW has recommended the use of a soil intake in the range of 100 mg/day for a worker with direct contact with soil and dust, however, a range of values could be explored in an uncertainty analysis. However, because the averaging time for a non-carcinogenic contaminant is usually the time over which the exposure occurs, not much change will be seen in risk estimates or the projected PRGs when this parameter is changed. A reasonable scenario that meets the pseudo-steady state criterion and allows evaluation of a range of soil ingestion rates, is probably the most useful, especially in developing a protective PRG for an outdoor worker in the CDAB.

2.5 Assessment of Incremental Lead Intakes and Associated Health Risks to Children

The HHRA includes an assessment of incremental lead intakes and risks associated with recreational exposures of children to lead at neighborhood areas, upland parks and other CUAs (Section 6.6.2, p 6-43, HHRA). The TRW recognizes the importance of evaluating the *incremental* sources of lead exposure that may affect children and adults in the CDAB (e.g., waste piles and contaminated sediments) and supports the HHRA in including these assessments as an important component of the CDAB risk assessment. The HHRA, however, does not clearly indicate how the estimated increments were used in the IEUBK model. The HHRA should more clearly describe that the increments were input in addition to residential sources, and that the incremental blood lead concentration associated with a given recreational activity was (apparently) defined as the difference between the blood lead concentrations predicted when the incremental intakes were included or not included in the model. More importantly, however, the TRW believes that the reported incremental risks of elevated blood lead attributable to recreational exposures may have been underestimated, for several reasons discussed below.

First, exposure estimates for shorter-term exposures should not be averaged over the entire year, for use in the IEUBK Model. The IEUBK model is relevant for continuous exposure periods that are of sufficient duration to produce a quasi-steady state blood lead concentration. The TRW considers the minimum exposure duration to be three months. In order to predict the quasi-steady state that could occur during a shorter (less than a year) period, the soil exposure is not averaged across the year. The HHRA presented a number of assumptions regarding exposure frequencies for these recreational scenarios, which ranged over a period of 168 to 238 days per year. These periods should be long enough to attain a quasi-steady state concentrations if the incidents occur at least once per week.

An additional source of underestimation of risk is use of current environmental lead levels as the baseline for the incremental estimates. Once residences and other frequently used areas are remediated to lower lead concentrations, the incremental risk attributable to exposure at additional recreational areas, if not also remediated, will be greater than suggested in the HHRA, by a substantial amount in some cases.

Another factor qualifying the usefulness of the projected incremental exposures is the appropriate estimates of incremental soil ingestion. The HHRA reported increments estimated from total daily soil ingestion rates reduced by the proportion of waking hours spent at the site. The two components of these increments are the amount of soil ingestion associated with the recreational exposures, and any appropriate weighting. The TRW was not certain whether the intention was to assume that part of the total daily ingestion would occur at the recreational area, or whether the ingestion associated with recreational exposure was expected in addition to typical ingestion rates at more commonly frequented locations (home, school, daycare, etc.). The HHRA calculation resulted in a greater than default amount of daily soil ingestion, which may be quite reasonable. Even higher ingestion may result at a wet site, such as those involving sediments. However, the more representative weighting of soil ingestion is the proportion of <u>outdoor</u> time spent at the site,

not the proportion of waking hours.

The approach taken in the HHRA is very similar to that recommended by the TRW, however, the HHRA does not calculate cumulative risks (e.g., P_{10}) associated with the various recreational exposures, but instead, calculates the incremental intakes and incremental central tendency blood lead concentrations. Calculation of the cumulative risks associated with each scenario, or a combination of scenarios would be informative in terms of showing the potential impacts of recreational exposures when combined with residential exposures. This type of analysis is also likely to show that, when recreational exposures are considered, the risk of exceeding a 10 µg/dL blood lead concentration will exceed 5% at all CSUs, when estimated with either the IEUBK default or Box models.

The TRW has made recommendations regarding approaches to utilizing the IEUBK model in assessing cumulative risks from residential and recreational exposures (see Attachment A of this report). This approach was implemented in the risk-based screening assessment of the CUAs in the Lower Basin and a detailed description of the approach is provided in Appendix B of the HHRA.

2.6 Environmental Data Sampling and Quality Assurance

2.6.1 Use of Floor Mats to Collect Residential Dust Samples

A novel feature of the CDAB HHRA was the use of floor mats to collect residential dust samples Section 2.2.1, p. 2-7, HHRA). The dust mat data were not used as input to the IEUBK model runs; dust inputs were derived from vacuum bag samples. The TRW has recommended the use of floor dust samples for estimating house dust lead concentrations and input into the IEUBK model and recognizes that there is very little information available on vacuum cleaner bag samples and floor mat samples and the use of this data in risk estimation at lead sites. However, because the dust mat approach is currently being explored by other researches in the lead field, and because it is an approach that the EPA has no comparable experience, the following observations are offered in this report.

The 1996 sampling event was the first application of door mats for collecting residential indoor dust to assess exposure at a Superfund site. Dust mats were placed in approximately 500 homes in 1996, with no indication of whether vacuum bags and dust mats were collected from the same homes. Vacuum bag samples were collected from approximately 320 homes. Mats were placed inside the home in a *high traffic area* and as close to the main entry as possible. The mats were collected three weeks after placement. Instructions given to the residents of the homes were that the mats should be walked on, but were not to be used as a shoe cleaning mat. If mats were handled in a way that violates the protocol, the mat was excluded from the data set. The HHRA notes that two mats collected in 1999 were excluded from the data analysis. Although vacuum cleaner bag contents were collected, the HHRA does not specify how long the bags were in use, or how such

information might have been obtained. It does indicate that efforts were made to verify with the homeowner that the vacuum had not been used outside of the home since previous bag change.

The CDAB HHRA provides comparisons of the dust lead concentrations estimated from the dust mat and vacuum bag samples. Arithmetic and geometric mean dust mat concentrations were higher than vacuum dust concentrations at all of the CSUs. A statistical comparison of the results from the two sampling approaches was not provided in the HHRA. It is unlikely that the unpaired group means presented in the summary tables (Table 6-11 of the HHRA) are significantly different (a paired comparison is not discussed in the HHRA).

2.6.2 Water Sampling

Water samples were collected from homes that were not on community water supplies. In the 1996 sampling event, samples were collected as close to the well head as possible. In subsequent years, flushed and first-draw samples were collected from the tap. The samples collected near the well head may not reflect drinking water exposures. Although this approach to sampling may be useful for detecting potential lead exposures from the water supply, it is not the most desired approach to developing inputs for the drinking water pathway in the IEUBK model because it may not provide a good estimate of actual exposures to children in home. Piping and solder in the home can contribute to lead in tap water. This contribution will vary during the day with use of the home water system, being higher after the water stands for a period and lower after flushing of the pipe system. It will also vary with the hardness or softness, and pH of the water. In order to ensure that this variability is represented in the estimates of drinking water lead concentrations, samples should be collected from the tap of each home, or a representative sample of homes, after the water has been allowed to stand in the pipes (e.g., first flush) and after the pipes have been flushed.

3.0 COMMENTS AND RECOMMENDATIONS IN RESPONSE TO REGION 10 PRIORITY ISSUES

3.1 *Is the risk characterization transparent, clear, consistent, and reasonable?*

The CDAB HHRA is a complex document that demands a careful and thorough reading if it is to be understood in its entirety. This is not surprising given the complexity and history of the site, and the wealth of data that was evaluated in the assessment, including analyses of data from the BHSS. Whether the risk characterization is clear and transparent will be determined only after it has had a wider readership. The sheer complexity of the assessment is likely to result in a wide range of opinions on this, determined, in part, by the background of individual readers and their willingness to give the entire report a complete and thoughtful reading.

From a technical perspective, the TRW found the risk characterization to be consistent and reasonable, in terms of the major outcomes of the assessment. That is, the individual parts of the assessment strongly support the dominant findings that: 1) lead risks to children in the CDAB are unacceptably high; 2) to achieve a reduction of risk to acceptable levels, the site will have to achieve soil lead levels of 400-800 ppm; and 3) the major uncertainties in the latter estimates are the magnitude of the impact of soil lead reductions on house dust lead levels, and the impact of education and intervention on soil and dust ingestion. That an assessment of this complexity can arrive at such a strongly supported set of conclusions, including strong support for a fairly narrow range in the soil clean up level, is remarkable, and a compliment to the architects of and contributors to the assessment.

The HHRA presents the results of three approaches that provide information about lead risk in the CDAB: 1) blood lead screening data gathered over a 4-year period, which may be biased to some unknown degree; 2) the IEUBK default model, which has worked well at other lead sites when data for children who were known to be exposed primarily at their homes were used in the model (Hogan et al. 1998; White et al. 1998), but for which only limited site-specific data to evaluate parameter estimates are available for the CDAB; and 3) IEUBK Box model, which was calibrated to agree with nine years of blood lead survey data, during which environmental and blood lead levels have been decreasing, and for which applicability to the CDAB has not been adequately assessed.

In general, blood lead surveys are the least desirable approach to estimating lead risks, unless the survey is convincingly representative of the population at the site, which does not appear to be the case at the CDAB from the perspective of the TRW. The blood lead screening data for the CDAB do, however, provide important data that show that there is a substantial problem with environmental lead exposures for children in the Basin. In view of the limitations of the blood lead data, many of which are discussed in the Uncertainty Discussion (Section 7.4.1, HHRA), the TRW supports the approach adopted in the HHRA of basing risk estimates on the results of the IEUBK model runs. This approach is

consistent with OSWER guidance (U.S. EPA, 1994). Nevertheless, the blood lead measurements and the IEUBK default and Box models yield reasonably consistent information that support the same conclusion, that Basin-wide residential lead risks are above acceptable levels. The blood lead survey indicates that 13% of the screened children between the ages 9-84 months had a blood lead $\geq$10 µg/dL; the IEUBK default and Box models yield P_{10}s of 27% and 10.4%, respectively (for all parts of the Basin combined, 9-84 months). A reasonable estimate of Basin-wide residential risk is within this range and, risks may be higher by 5-10% if incremental risk from recreational exposures are considered. The risk estimates based on default assumptions may be somewhat conservative because of the use of the 175 µm fractions of soil and dust, which may have been enriched in lead relative to the 250 µm fractions that are more commonly measured at CERCLA sites.

This consistency in the outcome of various analyses could be emphasized to a greater extent in the HHRA. Indeed, some readers of the report may be left with a stronger impression of the differences in the outcomes of the three approaches rather than their similarities. The similarities of outcomes are a main strength in the Risk Characterization.

In addition to the above general comments related to consistency and reasonableness, the TRW offers several other suggestions that would strengthen both aspects of the Risk Characterization:

- More emphasis should be placed on estimates of residential lead risk that are based on the batch mode IEUBK model runs, in which risks are estimated at each individual residence, and not on community mode runs. The batch-mode approach is consistent with EPA policy that emphasizes that, for the purpose of supporting remedial decisions for residential contamination, risk assessment approaches should focus on children who receive their principal lead exposures at their homes (U.S. EPA, 1994). The analyses termed “community mode” in the HHRA utilize an inappropriate simplifying assumption that all children within a community are exposed to the same average lead concentrations. The batch-mode is the preferred approach for site assessment, because it ensures that risks at each residence are integrated into the site risk estimate.

- Information that would allow a more complete assessment of the degree to which the blood lead samples reflect the CDAB population would facilitate the interpretation of the blood lead data, particularly the interpretations of comparisons between observed and predicted blood lead concentrations and regression analysis of relationships between exposures and blood lead concentrations. Such information might include the geographic distribution of the sampling within the CDAB and within CSUs, the distribution of response rates across communities, SES scores within the sample compared to those of the CADB and various comparisons of various demographic variables in the sample and CDAB (e.g., age, age of housing, residence time).

- Comparisons between the blood lead concentrations predicted with the IEUBK model and those observed in the CDAB (p. 6-29, HHRA) should not be relied on as the sole basis for evaluating the accuracy of model to represent exposures and blood lead concentrations in the CDAB. In order for this type of comparison to be correctly interpreted, the HHRA would have to provide more evidence that the observed blood lead concentrations adequately represent the CDAB population and that the exposure assumptions adequately represent the individual children sampled. The blood lead comparisons (Appendix Q, Tables Q4.26, HHRA) using alternative assumptions about the dust:soil ratio are useful only as a sensitivity analysis, but not as a basis for adjusting the model, because there is no real basis for attributing a *better fit* between predicted and observed blood lead concentrations to any given variable or set of variables. Also, there is uncertainty regarding factors that may have biased the blood lead observations.

- The IEUBK Box model should not be used as the basis for estimating pre-remediation risks in the CDAB (p. 6-39, HHRA). The Box model was calibrated to agree with the downward trend in post-remediation blood lead concentrations observed at the BHSS. Factors that may have affected this downward trend (e.g., decreased soil and dust intakes resulting from intervention and educational efforts) may not be operating or may not be as important in the CDAB. If adjustments were to be made to the IEUBK model for its application to the CDAB, such adjustments should be based on the available information about exposures and blood lead concentrations in the CDAB. The experience at the BHSS could be applied to the CDAB by gaining a better understanding of the exposure factors that contributed to the downward trend in the blood lead concentrations at the BHSS, and whether or not these same factors can be expected to affect blood lead concentrations in the CDAB to the same degree.

- The concept of separating yard and neighborhood soil contributions to lead intake is a potentially useful one, in particular when applied to predicting the soil lead cleanup levels (p. 6-29, HHRA). If supporting data were available, a similar approach could be extended various potential sources of dust lead exposure. However, Appendix Q of the HHRA does not provide support for use of the 40:30:30 ratio of dust: yard soil: community soil. Appendix Q suggests that there was little difference in predicted blood lead concentrations when either of three dust:soil ratios (55:45, 40:30:30, 75:18:7) were assumed in the model (see Appendix Q, Table 4-26 4-27, HHRA), which leads to an inconsistency in the HHRA.

- In representing the community soil lead levels, the arithmetic mean, rather than the geometric mean is generally preferred (p. 6-39, HHRA).

- The discussion of the bioavailability adjustment in Appendix Q (p. Q-10/2, HHRA)

seems to lump the absorption and intake terms in the IEUBK model into a single bioavailability term. These are actually separate parameters in the model that can be affected independently by site factors. Segregating these factors would allow one to consider the potential effects of changes in lead intake or absorption on risk estimates. The assumption that the bioavailability of lead in soil and dust is less than the IEUBK model default model (approximately 30% at low lead intakes) is not adequately justified to support adjustment of the IEUBK model for application to the CDAB (p. 6-39, HHRA). This assumption would be more strongly supported with evidence in animals or humans that the bioavailability of ingested lead in CDAB soil and/or dust is actually lower than the default values or lower than lead in soils from other mining/smelting sites.

- Inclusion of more detailed documentation on the IEUBK model runs would allow the reader to understand exactly how the model was implemented (p. 6-38, HHRA). Ideally, a file containing the inputs to the batch model runs would be important documentation that would enable a third party to reproduce the model runs described in the HHRA.

- The EPA ALM should not be used to estimate PRGs for exposures that are less the three months in duration or less frequent than one exposure episode per week (6-46, Tables 6-57 – 6-60, HHRA). The averaging time used in the EPA ALM should reflect the actual exposure duration.

3.2 *Does the Uncertainty Discussion provide context for the risk results?*

The uncertainty discussion is very comprehensive and does provide excellent context to the risk assessment. However, in some cases, the discussion may be interpreted as being in conflict with the Risk Characterization. For example, the Uncertainty Discussion states that the community-mode IEUBK model runs are of limited value for estimating risks (7.4.4, p 7-39, HHRA) A conclusion with which the TRW concurs. However, risk estimates based on community-mode runs are nevertheless included in the Risk Characterization. The Uncertainty Assessment discusses the limitations in the blood lead data collected in the CDAB and the implications these limitations place on interpreting comparisons with model predictions and in making remedial decisions (Section 7.4.1, p. 7-23, HHRA). However, these data are used in the Risk Characterization, and the outcomes of comparisons with model predictions are described in terms of *over predictions* or *under predictions*, suggesting a greater confidence in the blood lead data than is actually reflected in the Uncertainty Discussion. These inconsistencies are not major problems if the HHRA is thoroughly read and understood, but may lead to misunderstandings or misperceptions for a more casual reader.

The Uncertainty Discussion is largely qualitative and certain conclusions could be more strongly supported by more quantitative sensitivity analyses. For example, certain

assumptions for which there is great uncertainty could have been varied in model runs, similar to the approach that was taken in the sensitivity analysis of soil and dist lead levels in the estimate of clean up goals (Section 6.7.6, p. 6-55, HHRA). An example of this is also included in this report as it pertains to the sieving fraction (see attached Figure 1). Assumptions about bioavailability and soil and dust ingestion rates could also have been quantitatively explored. A more quantitative uncertainty analysis, in which the more sensitive model parameters were allowed to vary according to their respective uncertainty ranges, may also have been of added benefit. Such an analysis would have shown, most likely, that the apparent differences in the predictions of the IEUBK default and Box models are actually well within an overlapping range of model predictions, when uncertainty is considered. This would have supported a convergence, rather than a divergence, of the model outcomes. The above suggestions, if feasible, would have complimented the HHRA, but are not needed to support the conclusions of the HHRA or remedial decisions that might follow.

3.3 *Do the predicted house dust concentrations associated with various yard soil action levels support subsequent blood lead predictions and Preliminary Remediation Goals derivations?*

The goal of the approach taken in the HHRA of estimating post-remediation house dust lead concentration from the regression relationship between pre-remediation soil lead and house dust lead is reasonable, given the options available. However, the applicability of the outcome of such an analysis to the post-remediation conditions is uncertain. It should be recognized that when there is substantial noise in the data (e. g., in the lead contamination estimates for specific residences), regression models have a tendency to under-predict the strength of the true relationship between the variables. In this context, it is plausible that cleanup of yard soil will have a larger impact in the reduction of indoor dust levels in residences than is predicted by the regression equations developed in the HHRA. At this point there is insufficient data to determine the magnitude or kinetics of the impact of soil remediation on house dusts at the CDAB site. Numerous factors could result in the post-remediation dust lead levels having a very different relationship to soil lead levels than in the pre-remediation condition.

The dust lead projection will remain an important variable in any projection of post-remediation risks or estimation of clean up levels. This is demonstrated clearly in the sensitivity analysis presented in the HHRA (Section 6.7.6, p. 6-55, HHRA). The effectiveness of soil remediation in lowering blood lead concentrations will depend on the degree to which house dust lead levels decrease in response to changes in soil lead levels. A program in which dust lead levels in the homes were monitored before and after remediation would provide data to develop additional analyses at the site that may allow a more certain quantitation of the impacts of remediation on house dust lead levels.

3.4 *Does discussion of blood sampling methods, participation rates, and age distribution (which changed over time) help to interpret the blood lead screening results?*

The discussion of the blood lead data, in particular, that which appears in the Uncertainty Discussion (Section 7.4.1, HHRA), is very helpful. However, the TRW noted certain details that would have helped if emphasized, but which were absent or difficult to glean from the HHRA. (Ultimately, this information was made available to the TRW via conversations with Region 10).
These include:

- Additional information on the sampling approaches used in 1997 or 1998, for example, the extent to which the sampling was targeted or geographically distributed, would be useful for assessing the representativeness of the data, and whether or not the data should be combined with data collected in other sampling events.

- Additional information on the timing of the blood samples with respect to the timing of environmental samples, noting that all blood lead samples were collected in August and within one or two months of the collection of environmental samples. This is an important positive aspect of the sample design in that it alleviates variables that might otherwise affect interpretations of relationships between the blood lead concentrations and environmental lead levels at individual residences

- Because of the potential effects of health intervention activities in soil and dust ingestion and blood lead concentrations, it would be useful to indicate whether or not blood lead data collected after intervention (e.g,. nurses visits) were used in the various blood lead analyses. As it turns out these data were not used in the risk estimates.

In addition to the above, certain other information and analyses would be helpful, if feasible to provide. These would include the geographic distribution of the sampling within the CDAB and within CSUs, the distribution of response rates across communities, SES scores within the sample compared to those of the CDAB and various comparisons of various demographic variables in the sample and CDAB (e.g., age, age of housing, residence time). Such information might be useful, if available, for exploring further the existence and quantitative significance of biases in the blood lead measurements.

3.5 *Is the discussion of the results from the two modeling approaches sufficient to support risk management decisions protective for human health risks from lead?*

The discussion of the results from the IEUBK default and Box modeling approaches in the batch mode will support risk management decisions. The TRW considers the use of the IEUBK default model to be the preferred approach for decision-making, based on the

results of previously reported empirical comparisons (Hogan et al., 1998). These empirical comparisons showed satisfactory agreement between observed blood lead concentrations and IEUBK model predictions for children with environmental lead exposure measurements that characterized the majority of their exposure (approximately 90%-100%), and was relatively stable (that is, not decreasing over time), as the model was designed to be used. This review has discussed a number of reasons why the blood lead data collected in the CDAB, while very helpful for the children surveyed, may not be suitable for calibrating IEUBK predictions for decision-making:

- incomplete information about children's exposures (admittedly, this information is difficult to obtain; typically, about 50% have exposures away from their residences);
- possible enrichment of the residential soil and dust lead concentrations in the 175 µm soil and dust fractions relative to the measurements the IEUBK model was formally calibrated with;
- the non-steady-state nature of the lead contamination, due to on-going clean-up efforts; and
- the on-going community awareness of the lead problem, possibly lowering (temporarily) dust and soil ingestion rates.

The first factor has a unknown impact on the correspondence of observed and predicted blood lead levels, while the last three logically tend toward higher IEUBK predictions relative to observed blood lead levels, due to the design of the IEUBK model. For decision-making, the primary intended use of the IEUBK model, the TRW recommends considering the default dust/soil ingestion rate to estimate risk for future children populations, when environmental lead levels will have finally equilibrated after the last clean-up and behavioral interventions may let up under the presumption that there is no remaining *hazard.*

Nevertheless, the uncertainties discussed in the HHRA and this review argue against completely dismissing risk estimates based on the Box model. Parameter assumptions in the box model are within a range that can reasonably be considered in a sensitivity analysis of IEUBK risk estimates for this site. For the most recent years of data, there are indications that the calibrated (Box) model tends to underestimate some of the risks and that the default model tends to overestimate some risks. In the absence of any strong scientific basis for excluding either model from consideration, the residential clean up levels can be bracketed by using the two models and accounting for 1) recreational exposure-related increments in blood lead, 2) additional uncertainty introduced by the relatively high blood lead concentrations observed in the Lower Basin, given the relatively low soil and dust lead concentrations there; and 3) consideration of the possible effects of lead enrichment in the 175 µm fraction on the risk estimates. These considerations would support a relatively narrow clean up range, for example, 400–800 ppm. The difference between the extremes of the range, although highly significant in terms of potential clean up costs, would be well within the range of uncertainty bounds for each model if uncertainty were to be quantitatively introduced into the modeling results.

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Comments and Responses on:

Public Review Draft
Human Health Risk Assessment for the
Coeur d'Alene Basin Extending from Harrison to
Mullan on the Coeur d'Alene River and Tributaries
Remedial Investigation/Feasibility Study
July 2000

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CONTENTS

Volume A
1.0 DOCUMENT ORGANIZATION
2.0 GENERAL RESPONSES TO COMMENTS
3.0 DR. PAUL MUSHAK'S RESPONSES TO COMMENTS
- 3.1 The Technical Review Workgroup for Lead (TRW), Citizens Against Rails-to-Trails (CART), and Local Governments
- 3.2 Public Comments
- 3.3 Mining Industry Comments

4.0 COMMENTS FROM THE TECHNICAL REVIEW WORKGROUP FOR LEAD (TRW)

Volume B
5.0 STATE/USEPA'S RESPONSES TO INDIVIDUAL COMMENTS
- 5.1 Mining Industry Comments
- 5.2 Local Governments
- 5.3 The Lands Council
- 5.4 Shoshone Natural Resource Coalition (SNRC)
- 5.5 Citizens Against Rails-to-Trails (CART)
- 5.6 Spokane Tribe
- 5.7 Private Individuals
- 5.8 Community Leaders for EPA Accountability Now (CLEAN)

SECTION 5.0
STATE/USEPA'S RESPONSES TO INDIVIDUAL COMMENTS

Mining Industry Comments

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
131	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A1		Hecla Mining and ASARCO	1-Section 1	p.8-25		Not Accepted

Comments>

The HHRA uses a simplistic and generic predictive model and a statistically inadequate set of actual blood lead data to finger soil lead levels in parts of the Basin as a primary source of childhood lead exposure. In so doing, EPA and its contractors ignore the facts that the overall situation in the Basin for children s exposure to lead is a reasonably good one, given the long history of mining and metallurgical activities in the Basin. and that the situation is improving with time. Although the HHRA acknowledges that lead-based paint in aging housing stock and other factors significantly contribute to childhood blood lead levels in the Basin--as well as in urban areas throughout the Nation--the document nonetheless suggests that further soil cleanup in the Basin to levels below the "EPA residential soil screening level of 400 mg/kg" could conceivably be justified. See HHRA at 8-25. The HHRA reaches this ill-supported determination on the basis of blood lead data from a cohort too small to be representative. Moreover, it fails to account for numerous, relevant site-specific factors. As a result, the HHRA fails to recognize that a community health intervention approach in the Basin makes more sense than further extensive soil remediation.

Response>>

The HHRA disagrees with this comment. In comparison to risk assessment methodologies used for other contaminants, the IEUBK is neither simplistic nor generic. The model allows for route specific absorption rates, integrates the effects of lead coming from different routes, and relates the biological response directly to toxicity criteria, on an age-specific basis. This procedure is considerably more complex than that applied in non-lead, non-carcinogenic risk assessment, and results in more precise, and less uncertain, estimates of effects than is typically obtained. As a result, lower margins of safety are employed in sub-chronic lead risk assessment than in the methods used for other metals. Comparison of blood lead data for the Basin to other sites and national or State-wide surveys, for the purpose of determining whether these findings are "relatively good or bad", is problematic. Selection bias may have occurred related to individual family decisions to participate. The HHRA did not draw a conclusion relative to these arguments as there are not sufficient data to test either hypothesis. These opinions are discussed in Sections 6.2.2 and 7.4.1, 8.8, and 8.11.2 and reflect most of the comments offered by reviewers. Community health intervention activities are acknowledged as an effective short-term remedy for children experiencing excess absorption and may have an effect in reducing dose/response in the Basin. However, health intervention is not recognized as a primary prevention remedy under current EPA guidance. Extensive analysis and discussions of site-specific factors are included in the HHRA and the document has been specifically fashioned to provide risk managers with methods to additionally consider site-specific exposure factors. See also General Response to Comments, #9a through #9d and #10a through #10c.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
132	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A2		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

The HHRA employs EPA's integrated exposure uptake/biokinetic (IEUBK) model as the primary basis for quantifying potential exposures and health risks associated with exposures of young children to lead. The IEUBK model was used to predict children's exposures associated with a baseline residential scenario and with various other potential lead exposure sources, such as recreational contacts with soil, including mine wastes. EPA's guidance for modifying its models, including the IEUBK model, allows only a limited ability to incorporate all of the site-specific environmental data, especially when the environmental conditions vary widely across a large area. The models also do not generally consider other non-environmental factors that influence an individual's health risks. For example, the IEUBK model is very focused on the effect of soil on a predicted blood lead concentration and is not typically used to identify other sources. Incorporation of additional lead exposure from paint, for example, is generally not included because the use of the model to calculate cleanup levels for soil will just indicate that more soil must be remediated to address paint. Care should be taken in selecting input assumptions, however, to make sure that lead concentrations in other media (e.g., indoor dust) are not solely attributed to soil. Such a distinction is generally not made and indoor dust concentrations are automatically assumed to be attributed to soil. The HHRA's approach for application of the lead exposure model is to assume that house dust lead concentrations are due to soil and that paint is not a factor, which is inconsistent with the empirical data.

Response>>

The HHRA disagrees with this comment. In the IEUBK analysis, observed soil and dust lead concentrations are used. No consideration of the source of the lead in dust is inherent in the analysis. The IEUBK is equally capable of predicting the effects of changes along any pathway, and was originally developed to assess required changes in air lead levels, prior to being approved for use in CERCLA and RCRA programs. See also General Response to Comments, #3, #4, and #9.

Misc. Input>>

133	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A3		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

The HHRA makes little effort to incorporate site-specific data (other than soil lead and dust mat lead concentrations at house entry ways) for refining the modeling approach. Most importantly, adequate thought is not given to interpreting the modeling results in light of actual observations. EPA and the state have already performed detailed environmental and blood lead studies and a risk assessment within the BHSS that could serve as a useful starting point for the development of site-specific models or interpretation of similar empirical data from the Basin. Rather than serving as a starting point for exposure models for the Basin, the model developed for the BHSS (the "Box model") is used primarily as a point of contrast for the EPA model results based on default inputs. No effort was made to develop a site-specific model for the Basin that would address the differences between the BHSS and the surrounding Basin (e.g., to reflect the lesser importance of smelter emissions outside of the BHSS).

Response>>

The HHRA disagrees with this comment. Site-specific analysis for the Basin was conducted as a major component of the HHRA. Observed soil and dust lead concentrations are used and incremental intakes evaluated are developed on a site-specific basis. See General Response to Comment # A4 and A5. This analysis indicates pathways and dose/response relationships similar to the BHSS for, at least, the upper Basin. As a result, it is not unexpected that the Box Model accurately predicts blood lead levels and percent to exceed the 10 ug/dl criteria for this population, and that the site-specific input values are appropriate. There are questions, however, as to whether the model is representative of members of the population that did not participate in the blood lead surveys. The HHRA was unable to quantify any bias with respect to participation rates and drew no conclusion in regard to this question. See also General Response to Comments, #9a through #9d.

Misc. Input>>

134	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A4		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

In the end, neither the Box model nor the EPA default model explains the low average blood lead levels observed within the Basin.

Response>>

The HHRA disagrees with this comment. The Box Model accurately predicts average blood lead levels in the upper Basin and under-predicts average blood lead levels in the Lower Basin.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
135	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A5		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

In addition, neither of the HHRA models attempts to address the likely variation that exists among the identified exposure areas for site conditions that are likely to affect the IEUBK modeling results (e.g., age of housing, proximity to mine wastes, etc.).
Such critical site-specific issues must be appropriately addressed in the risk analyses so that remediation decisions focus on efforts that will effectively benefit overall public health in addition to meeting the needs of potentially sensitive sub-groups.

Response>>

The HHRA disagrees with this comment. The IEUBK models utilize observed soil and dust lead concentrations that are reflective of the sources on-going for the baseline exposure in the Basin. Incremental exposure assessments are developed in a manner that can be evaluated on a site-specific basis by risk managers. See also General Response to Comments, #3, #4 and #5 .

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
136	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS and TG
A6		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Examples of highly inappropriate modeling assumptions and exposure scenarios and their possible effects on risk management decisions include the following: The soil ingestion rate used for the recreational scenario is greater than that assumed for a reasonable maximum exposure (RME) scenario. The HHRA's soil intake rate for recreational exposure is based on an estimate of the intake for children 1 to 5 years old who are camping in summer, yet this rate is applied to children 4 to 11 years old. Children in this older age group would not have a soil ingestion rate as high as the younger children (consistent with the difference in RME soil ingestion rates for children over 6 years, 100 mg/day, and under 6 years, 200 mg/day). In addition, the types of recreational activities evaluated in the HHRA are daytime activities that would be associated with lower exposure than camping because children would not be living, sleeping and eating outdoors. Unrealistic assumptions about soil ingestion during recreational activities exaggerates the potential risks associated with common-use areas relative to other sources of health risk.

Response>>

Non Lead:

The 300 mg/day value is EPA Region 10's default value for children and adults engaged in intermittent recreational exposures. We used this value for the RME neighborhood scenario's soil and sediment ingestion rates, and it is larger than the RME residential value. The primary references are: (1) the Van Winjen study (1990) based on a 3-5 day exposure study of children aged 0 to 5 years while camping, which provided an upper percentile ingestion rate of 300 mg/day; and (2) a more recent study by Stanek et al (1997) which provides an upper percentile ingestion rate of approximately 300 mg/day for adults engaged in routine day-to-day activities over a 4-week period. Although the Stanek study population was small, its results suggest that adults (and therefore older children) could potentially have upper-bound soil ingestion rates within the vicinity of 300 mg/day. Soil ingestion is likely event-driven and likely occurs at a higher rate during outdoor activities than the average annual value of 200 mg/day. The most important aspect of choosing this contact rate over 200 mg/day is that it represents a short exposure duration in a relatively contact-intensive situation. Thus, this value was deemed appropriate for the 4-11 year old age group for intermittent recreational exposures.

Stanek, EJ III, E Calabrese, R Barnes, and P Pekow. 1991. Soil Ingestion in Adults-Results of a Second Pilot Safety. Ecotoxicology and Environmental Safety. 36:249-257.

Lead:

The HHRA disagrees with this comment. RME ingestion rates are not used in the models. RME rates are provided for risk managers convenience in comparing potential intake rates. See General Response to Comments, #5.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
137	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS and TG
A7		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Examples of highly inappropriate modeling assumptions and exposure scenarios and their possible effects on risk management decisions include the following: The metals concentrations of waste rock piles were used together with other data to compute a community average concentration in soil for use in evaluating recreational exposures of residents and visitors using common areas. The areas where waste piles are present are often distant from residential areas, and the use of waste pile concentrations to predict the average exposure in neighborhood common-use areas misrepresents their potential contribution to recreational exposure. This approach also artificially links elevated blood lead levels to recreational exposure on waste rock piles that, according to Basin residents, is not significant in most areas of the Basin (TerraGraphics 2000).

Response>>

The reviewer is incorrect. Data from waste rock piles were evaluated only for the population of Mullan, Ninemile and Canyon Creeks. The waste pile data included in the HHRA were collected from piles near residential homes. Data from waste piles were analyzed separately - the data were not mixed with other media - and were used to evaluate exposure to children who may play on the piles.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
138	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A8		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Examples of highly inappropriate modeling assumptions and exposure scenarios and their possible effects on risk management decisions include the following: Through the exposure assumptions adopted by the HHRA, elevated blood lead levels in Lower Basin children are attributed to their recreational activities in tailings-deposited beaches and other areas distant from their residential yards. However, the blood lead data from this area indicate that children from 1 to 2 years old have the highest blood lead levels of those tested from the Lower Basin. These children are not likely to have significant exposure to sources of lead outside the home or a daycare provider's home/business. If lead sources in common/recreational areas outside the home are the cause of elevated blood lead levels in Lower Basin children, then the older children in this area should have higher blood lead levels relative to those in other areas. In fact, of the 55 blood lead measurements from the Lower Basin, the 3 with results above 15 µg/dL are from children ages 2, 3 and 5. Only 2 of the 27 test results from children who were more than 5 years old had blood lead levels greater 10 µg/dL. Consequently, alternative explanations for elevated blood lead levels should be considered.

Response>>

The HHRA disagrees with the comment. Follow-up information for these 2-5 year old children with high blood lead levels do indicate extended beach and camping activity with their parents in the Lower Basin.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
139	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A9		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Examples of highly inappropriate modeling assumptions and exposure scenarios and their possible effects on risk management decisions include the following: Subsistence lifestyles are identified under populations of potential concern. However, there are no individuals, let alone populations, practicing "subsistence lifestyles," as defined by the HHRA, or even relying on the natural resources for the majority of their food. Therefore, this exposure scenario is purely hypothetical, and although it is possible, it is also highly improbable. If risk managers do not realize that this scenario is improbable then they will consider and may recommend costly, large-scale actions directed at calculated exposures and risks. If implemented, such actions may have little effect on actual risks for current or future Basin residents.

Response>>

The HHRA disagrees with the comment. Subsistence scenarios and relevant exposure factors were developed in cooperation with Coeur d'Alene Tribe representatives. The Traditional and Current Subsistence scenarios were requested by the Tribe as representing possible future uses of the area. Exposure factors were derived specifically for the Coeur d'Alene Tribe. Scenarios and exposure factor analysis were patterned after the development of similar scenarios for the Columbia River Tribes used at the Hanford Nuclear reservation. A cultural anthropologist reviewed and suggested appropriate modifications for each of the exposure factors. Each pathway was characterized individually, risk managers can combine pathway results as considered appropriate to estimate total intake rates. No blood lead modeling was performed for subsistence lifestyles. See also General Response to Comments, #6.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
140	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A10		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

This reliance on hypothetical model results, even where such results are unsupported by empirical observations, is contrary to standard scientific practice in model development and validation and is likely to lead to erroneous remediation decisions that may not effectively benefit public health. The observed discrepancies between the modeling and real-world conditions consists of both over predictions and under predictions, which vary in magnitude and are, in some cases. quite substantial. Such discrepancies indicate that the model structure has failed to adequately characterize those factors determining lead exposure and risk within the Basin. In contrast to the HHRA statements downplaying the importance of empirical observations in assessing potential health risks associated with lead exposures and in making remedial action decisions. such observations are critical for understanding the degree to which the model predictions reflect actual exposures and for identifying the most significant sources of exposure and resulting health risk.

Response>>

The HHRA disagrees with the comment. Site-specific analysis for the Basin was conducted as a major component of the HHRA. Observed blood, soil, paint and dust lead concentrations are analyzed on a home-specific basis. This analysis indicates pathways and dose/response relationships similar to the BHSS for, at least, the upper Basin. As a result, it is not unexpected that the Box Model accurately predicts blood lead levels and percent to exceed the 10 ug/dl criteria for this population, and that the site-specific input values are appropriate. See General Response to Comments, #3 and #9.

Misc. Input>>

141	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS and TG
A11		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

In summary, the HHRA s inclusion of overly conservative assumptions, improbable exposure scenarios and over-emphasis on negligible sources of exposure (e.g., garden vegetables) results in an inaccurate characterization of existing risks that is of little value for identifying and developing strategies that will be effective in reducing the actual risks.

Response>>

Non-lead:
We disagree that garden vegetables were over-emphasized, particularly for arsenic, see discussion on Page 7-16 and 7-17 of the report which acknowledges the semi-quantitative nature of the vegetable data and recommends further study if risk managers wish to reduce the uncertainty in this area. In addition, the results of the vegetable pathway were not added into the total risk values for residents. However, text will be added to sections 5, 7, and 8 to clarify garden vegetable exposures.

Lead:
The HHRA is a comprehensive evaluation of site-specific information. It includes surveys of a substantial portion of the Basin childhood population, and a review and follow-up identifying individual risk profiles for more than 90% of the children identified with high blood lead levels. The data base contains paired blood lead and environmental exposures for more than 400 observations. Quantitative analysis of this data by regression techniques explain 60% of the variation in observed blood lead levels. In consideration of the well documented individual variance in blood lead levels, this is a strong and compelling finding. The dose response relationships are similar to those based on thousands of observations at the adjacent BHSS. The baseline Box Model developed for the BHSS performs well in predicting both the mean and the distribution of blood lead levels in the upper Basin. Blood lead levels in the Lower Basin are consistent with risk estimates for reported recreational activities noted in the follow up reports. Garden vegetables and other negligible sources are not included in the baseline or recreational blood lead model estimates. See the General Response to Comments for additional details.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
142	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A12		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

B. Potential for High Bias to Blood Lead Data

The HHRA s discussion of the empirical observations from blood lead and environmental lead concentration data is deficient in a number of respects. The HHRA does not adequately characterize the degree to which these data are representative of the entire area under study. The blood lead concentration results are based on a small sample size. The level of participation is extremely low for accurately characterizing exposure pathways for the entire population in the Basin, especially because the data were not collected using stratified random sampling to attempt to characterize all aspects of the community. The blood lead data are particularly uncertain for some subareas with very low sample sizes (e.g., the Lower Basin).

The HHRA states that there is a 25 percent participation level in the blood lead testing program (based primarily on the 1999 blood lead samples), yet when the data over the four years used in the HHRA are considered, the actual percentage of the population participating is much lower. Since there were 574 blood lead samples from children over the 4 years and all children could have been sampled each year, the eligible population over 4 years is more like four times the 1,100 children per yearly cohort based on the HHRA. The actual participation rate over the 4-year period is more like 574/4400 or less than 15%. The HHRA has not obtained a true "experimental," i.e., representative, sampling cohort or an observational cohort of sufficient size and participation rate. Consequently, the data may reflect biases introduced by random factors as well as more systematic sampling biases.

Response>>

The HHRA disagrees with the comment. Blood lead levels in the Basin were tested for public health screening purposes to identify children that could benefit from follow-up intervention services. Although it was not collected for purposes for which it is applied in the HHRA, the site-specific blood lead data base is substantial. Appropriate analysis and presentation of this information, in combination with the individual risk profiles developed for high risk children by the local health department and the massive experience at the adjacent BHSS, provides risk managers with insights atypical for a Superfund site. Uncertainty issues associated with these data, analysis and the issues noted in this comment are extensively discussed in Section 7 of the document. The calculation regarding aggrigate screening of children for the four years is incorrect. There are approximately 1,000 children from 9 months through 9 years of age in the Basin. Over 4 years, this group includes about 1,300 individuals, of which 424, or about 1/3 of the population has been screened at least one time. See also General Responses to Comments and response to Comment A13.

Misc. Input>>

146	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A13		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Due to the low participation in the voluntary blood lead testing program, there are a number of potential biases in these data. Because multiple test results may be included for individual children, then blood lead data may be biased by the presence of repeat tests from families with a child with an elevated blood lead level. These families are more likely to be encouraged to return for retesting by the program operators and may also be more motivated to have the child as well as any siblings retested. Conversely, when a child has a low blood lead level, parents tend to be less concerned and are less likely to bring this child or any siblings in for future testing. Families that may have moved to the Basin from within the BHSS may have also received higher past exposures to lead and may be more inclined to take part in blood lead sampling programs. As noted by the HHRA, the $40 cash incentive in the 1999 monitoring program may have also attracted lower income families. These factors may introduce a high bias to the blood lead data set. In addition, given the small sample sizes from each of the geographic sub areas defined within the Basin, the blood lead data from sub areas may easily be biased by random factors (e.g., many children from one family with high lead exposures). Such biases may inflate estimates of the overall ratio of children with elevated blood lead levels.

Response>>

The HHRA agrees with this comment, but disagrees with the conclusion. Selection bias may have occurred related to individual family decisions to participate. The HHRA did not draw a conclusion relative to the potential biases, as there are insufficient data available to evaluate the question. With regard to repeat testing and siblings, eighty-one (81) children from 57 homes were tested more than once. Sixty-five (65) of those children were tested twice, 13 were tested three times and 3 were tested in each of the four years. Of those children tested more than once, 11 had levels greater than 10 ug/dl and received intervention services from the local public health program. Seven (7) of these children had blood lower blood lead levels in subsequent testing, 1 had the same level, and 3 had higher levels. The children tested more than once tended to have lower than average levels for children in their age group on the first test and similar levels on subsequent testing. These results would indicate that some observations used in the analysis were lower than might be obtained in a random sampling of the population. See also Sections 6.2.2 and 7.4.1, 8.8, and 8.11.2 of the HHRA and General Response to Comments, #2a.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
147	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A14		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

In an effort to address these uncertainties, the HHRA suggests that the observed blood lead concentrations are likely to be artificially low because of current community awareness of and efforts to mitigate lead exposures in the neighboring BHSS. The fourth paragraph on page 6-2 notes that site-specific monitoring data accurately describe blood lead levels and that its predictive value for the future may be contingent on continuing public health intervention activities to monitor and reduce blood lead levels. Nevertheless, blood lead monitoring and environmental intervention activities are relatively new to the portions of the Basin outside of the BHSS. Blood lead levels away from the BHSS area of smelter influence have, over the monitoring period from 1996 to 1999, shown no obvious decreases (also stated on page 6-9, fourth paragraph). Blood lead levels during the current economically depressed state of the Basin are actually likely to be worst case and would probably decrease in the future with economic improvement.

Response>>

The HHRA agrees with this comment. Improvement in socio-economic conditions in the Basin would be beneficial to risk reduction programs. See General Response to Comments, #1a.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
148	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A15		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

C. Preferability of Community Health Intervention Approach

When all of the available information from the Basin is viewed collectively and objectively, other risk reduction strategies, such as community health intervention, education and monitoring programs focusing on individual exposures are clearly more appropriate than extensive further soil excavation. Such programs, and their remediation components, are consistent with EPA's guidance for addressing soil lead hazards attributable to lead-based paint in that they address individual risk factors and focus on significant sources of lead exposure. The existing EPA and HUD guidance (FR Vol. 60, No. 175, 47248-47257) for lead in soil calls for a range of response actions where blood lead levels are elevated and soils have lead concentrations between 400 and 5,000 ppm. The actions range from monitoring and education to a variety of interim physical measures. Soil removal and replacement is not specified as a response action until concentrations are greater than 5,000 ppm (2,000 ppm in recently proposed rule). This guidance recognizes the inherently conservative approach used to identify soil lead hazards as it relates to the cost of soil removal and to the corresponding benefits from reductions in child blood lead levels (i.e., the conservative IEUBK modeling approach used to identify the 400 ppm EPA screening level).

Outreach, intervention, and education programs have proven effective at a number of sites (e.g., Butte, Montana; Leadville, Colorado; and Trail, British Columbia) where they are currently being implemented. These programs may include remediation of soil, as well as other sources of lead, and can be used to target those at risk based on known site-specific risk factors. Educational programs focusing on related health issues such as pre-natal care, nutrition, hygiene and early childhood development may also be instituted. These programs combine intervention efforts for actual lead exposure sources with proactive measures and thereby provide more effective public health protection at lower cost than widespread remedial actions. In the case of the Coeur d'Alene Basin, the mining companies have enlisted the aid of individuals involved with successful intervention programs, as well as recognized experts in the fields of lead toxicology and medicine, to develop a comprehensive program consistent with the "real world data." The Child Health Intervention Program (CHIP) is detailed in the enclosed material. Based on all the existing information, such a program is the most sensible approach for addressing elevated blood leads within the Basin.

As described below, the HHRA offers no adequate empirical basis to recommend disruptive soil excavation and replacement activities over other methods for risk reduction. In fact, the available information supports the type of actions contained in the CHIP. The Basin population has been exposed to multiple sources of lead, including but not limited to mining-related sources, that have been present for at least the last 75 years. Given these long-term conditions, blood lead levels in existing residents provide the best indicator of the baseline health risk from lead and can be especially useful when appropriately paired with environmental lead concentration data for identifying dominant sources of lead exposure. However, when interpreting data for the Basin population to identify sources of lead exposure, it is necessary to recognize the potential for biases in the blood lead data set (see previous comment) and to acknowledge the potential effects

Response>>

The HHRA disagrees with the interpretation of federal guidance. Current directives discourage reliance on behavioral modification programs. Current guidance included in Appendix O states:

"In selecting site management strategies, it is OSWER's preference to seek early risk reduction with a combination of engineering controls (actions which permanently remove or treat contaminants, or create reliable barriers to mitigate the risk of exposure) and non-engineering response actions... As a given project progresses, OSWER's goal should be to reduce reliance on education and intervention programs to mitigate risk. The goal should be cleanup strategies that move away from reliance on long-term changes in community behavior to be protective; behavioral changes may be difficult to maintain over time. The actual remedy selected at each site must be determined by application of the NCP remedy selection criteria to site-specific circumstances. However, this approach recognizes the NCP preference for permanent remedies and emphasizes the use of engineering controls for long-term response actions.…"

Current directives with respect to HUD and TSCA Title IV-403 guidance states: "The TSCA 403 proposed 2,000 ppm hazard level should not be treated as an Applicable or Relevant and Appropriate Requirement (ARAR), "to be considered" or TBC or media cleanup standard (MCS). As recognized in the TSCA 403 rule, lead contamination at levels below 2,000 ppm may pose a serious health risk based upon a site-specific evaluation and may warrant timely response actions. Thus, the 2,000 ppm proposed standard under TSCA 403 should not be used to modify approaches to addressing Brownfields, RCRA sites, National Priority List (NPL) sites, Federal CERCLA removal actions, and CERCLA non-NPL facilities."

Health intervention and behavioral modification programs have been successfully applied to reduce lead exposure reductions. The Lead Health Intervention Program (LHIP) at the BHSS has been found to be an effective interim method to assist parents and principal care-givers in reducing blood levels of children suffering from excess absorption. From 70% to 80% of children with high blood lead levels receiving follow-up services respond positively with reduced blood lead levels. The LHIP has not been demonstrated as an effective substitute for cleanup. Cleanup of active sources, yard soil removals, and the associated reductions in community-wide soil and dust lead levels have been identified as the most effective measures in reducing blood lead level at the BHSS. Implementing a new program or expanding the LHIP over the entire Basin would offer considerable logistic challenges. See also General Response to Comments, #1 and #10.

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
	of diverse residential settings, environmental conditions and family circumstances that exist for that population.					
	Misc. Input>>					
149	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A16		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted
	Comments>			**Response>>**		
	In an attempt to evaluate community-wide exposures, the HHRA performed multivariate statistical analyses of paired blood lead and environmental lead concentration data to identify significant correlations that may be indicative of an exposure/response relationship. Of the various environmental media examined for correlations with blood lead concentrations, the lead loading rate on entry mats had the highest correlation. Dust on entry mats may originate from both interior and exterior sources as the lead loading rate only describes the amount of dust and not its source. The next highest correlations were observed for various factors for interior or exterior paint condition and interior-paint lead concentration. Interestingly, these variables were more strongly correlated with blood lead level than entry mat dust lead concentration, which indicates that the prior correlation with lead loading is most closely tied to the amount of dust as opposed to the concentration of lead. The effects of housing age and exterior paint condition on blood lead were not examined, and because information on socioeconomic status was not obtained, the effects of this risk factor could not be evaluated for the data set used in the HHRA. However, it is most important to note that yard soil and community soil lead had among the lowest correlations with blood lead. The correlation coefficients reported for soil are low and do not signify "high correlations or strongly correlated" data sets as stated in the text (page 6-20 and page 6-24). Although these results demonstrate that in-home sources of lead, including lead-based paint, can be more significant contributors to blood lead levels than soil, this conclusion was not given adequate consideration when evaluating the risks from lead in soil.			The HHRA disagrees with this comment. The analyses referred to were accomplished by stepwise regression where the significance of the variables is determined by entry into, and exit out of the model after accounting for previously selected variables, not correlation coefficients. Dust lead loading is clearly the most significant variable with respect to blood lead levels. With respect to either dust lead loading or dust mat lead concentration, yard soil lead concentration is the most significant source variable. For vacuum bag lead content, dust mat loading is most significant, followed by yard soil lead concentration, and interior maximum XRF paint lead loading. These findings suggest pathway effects similar to those noted at the BHSS and other sites, i.e., soil and paint lead contribute to childhood house dust exposures, with soil lead also acting as an independent source through direct contact. The HHRA concluded that both sources are likely significant, but there is uncertainty regarding paint sources due to the relationship between paint condition and socio-economic status that cannot be unraveled with these data. That conclusion remains unchanged. These findings are consistent with the follow-up reports from public health nurses investigating high blood lead levels and results from other sites including the nearby BHSS. See also General Response to Comments, #1a, #3, #4a and #9.		
	Misc. Input>>					

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
150	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A17		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

The case-by-case follow-up studies of individual children from the Basin with blood lead concentrations greater than 10 µg/dL also indicate that many factors contribute to the elevated blood lead level (e.g., dusty homes exposure to soil lead concentrations >2,000 ppm, and other sources such as lead-based paint or remodeling exposures). When soil lead concentrations are mentioned as a contributing actor in the follow-up discussions, the concentrations typically are very high (e.g., greater than 2,000, 2,000-3,000, or greater than 4,000 mg/kg). These levels are considerably in excess of those identified later in the report as potential action levels (e.g., 400 to 800 mg/kg). In addition, there is no mention of low socioeconomic status as a risk factor, even though previous reports for this site (IDHW 1999) and the BHSS (TerraGraphics l997) have specifically mentioned the effects of socioeconomic status on blood lead levels.

The IDHW/ATSDR (1999) study of the Basin confirmed that socioeconomic status is an important risk factor for higher blood lead levels in young children. Low socioeconomic status is often related to factors that may increase lead exposure and absorption such as poor home upkeep, nutrition, hygiene, and child supervision. Socioeconomic factors also need to be identified when comparing Basin blood lead statistics to national and state statistics. The only such factor that is tracked in comparisons from the HHRA is age of housing. The relatively high percentage of the Basin population that is considered low income compared to national and state levels is not taken into account. Middle-class to upper-class children in properly renovated old homes are not comparable to the majority of the population in the Basin.

Response>>

The HHRA agrees with this the first part of this comment, but disagrees with the conclusion and mis-characterization of the Basin residents. Exposure to contaminated soils and dusts in excess of 2000 mg/kg lead is a common factor noted in follow-up of lead poisoned children. The HHRA notes in several sections that adverse socio-economic conditions are frequently identified as contributing factors to excess absorption. A discussion of socio-economic status and its relationship to lead poisoning is provided in Section 8.8. Current childhood poverty rates in the Silver Valley are near 30%, about twice the state-wide rate, and certainly not in the majority. Potential lead paint problems are noted for 20 to 25% of the homes, with 2 to 3% exhibiting clearly observable hazards. The majority of families in the area enjoy middle and upper socio-economic status and live in safe and adequate housing. Also see discussion under General Response to Comments, #1.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
151	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A18		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Contrary to the available information, only the model applications and conservative inputs support an explanation that soil and house dust containing soil are the primary cause of elevated blood lead levels in children from the Basin. This position is especially difficult to uphold in light of results presented in the 1999 IDHW/ATSDR Environmental Health Exposure Assessment Report that found no significant correlation of blood lead to the concentration of lead in yard soil. The accuracy of this finding has been confirmed by the testimony from government's own expert witness, Dr. Philip Landigran (deposition transcript attached), who stated that the conclusions reached in the IDHW/ATSDR report regarding the lack of correlation between blood lead levels in young children and lead levels in yard soil were accurate.

Response>>

The HHRA disagrees with this comment. The HHRA concluded that contaminated soils, house dust, and lead based paint are all related to excess absorption along complex exposure pathways, with blood lead levels most related to dust lead loading in the home, followed by independent effects of yard soil lead, interior paint lead condition, and exterior paint lead content. The dust lead pathway is most influenced by outdoor soils, augmented by paint contributions in older homes, especially those in poor condition. The conclusions regarding the role and relative importance of contaminated soils and dusts in childhood lead poisoning in the Basin are supported by numerous factors in addition to modeling analysis. Several studies and investigations are cited in the HHRA or support materials for federal guidance have noted similar results. Follow-up investigations by public health personnel have specifically identified soil and dust as the principal source of high blood lead levels observed in children in the Basin and BHSS. More than two decades of information, analysis, and observations at the nearby BHSS have indicated the importance of these sources in children's lead poisoning. Elimination of these sources has been shown to be the principal factor in reducing children's blood lead levels in the last decade. The 1999 IDHW/ATSDR report identifies dust lead loading as that factor most associated with blood lead levels and notes a strong relationship between outdoor soils and dust lead loading. That study also notes that the proportion of children with elevated blood lead levels differed significantly by outdoor playing surface. Thirty-eight percent (37.5%) of children who played outdoors most frequently on dirt or sand surfaces had elevated blood lead levels, compared with 4.8% of children who played outdoors most frequently on grass or other surfaces. These are the same findings that the HHRA indicates. The analysis, results and conclusions of the HHRA and the 1999 IDHW/ATSDR studies are consistent and are in agreement. This is not unexpected, as both studies arise from the same database. The site-specific analysis included in the HHRA employs the same exposure measurements as the IDHW/ATSDR analysis, augmented by quantitative paint exposure estimates and blood lead levels collected from subsequent years from the same homes. See also General Response to Comments, #2, #3, and #4.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
152	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A19		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

The highest risks identified for non-lead metals are associated with ingestion of soil by children. This result is not unexpected given the high soil ingestion rates used to compute the non-cancer hazard quotients and cancer risks. For the residential scenarios, soil ingestion is assumed to occur year-round (350 days a year), even though the Basin experiences winter conditions and snow cover for months of each year. For the neighborhood recreational scenario, the high soil ingestion rate for 1 to 5 year olds while camping is assumed to occur 1 to 2 times a week for 34 weeks a year, and the high surface water and sediment intakes (via swimming/wading) are assumed to occur 4 times a week for 24 weeks a year. The results of a questionnaire completed by Basin residents regarding their use of recreational areas show that nearly all of the respondents recognized that the soil ingestion rates (computed from the estimated number of days of exposure to soil) and frequencies of exposure to surface water and sediment used by the HHRA were unreasonably high given the local climate.

Response>>

Exposure to soil both by ingestion and dermally continues during the winter inside the home, although likely at a reduced rate, because soil continues to be a component of indoor dust in the winter; however, it is not clear how much reduction would occur. Therefore, the RME scenario did not adjust contact downward for winter while the CT scenario assumed no contact. These two assumptions potentially bound the actual amounts ingested/absorbed. We acknowledge that the exposure frequencies and duration likely overestimate exposure for the majority of recreational receptors; however, 1) information from the Panhandle Health District's lead intervention program indicates that many children do spend very large amounts of time outdoors (12 hours a day for some children), particularly in summer; and 2) the exposure times, in terms of hours per day, are from EPA's 1997 Exposure Factor's Handbook containing national information. Children in the more rural areas of the Basin would be expected to spend more time outside than that estimated from the national information which includes urban children. The RME estimates used in the risk calculations are weighted upwards in part to protect the very high frequency outdoor exposure of some children, and in part to fulfuill the requirements of an HHRA "reasonable maximum" exposure scenario to ensure the public is protected.

Soil residential ingestion rates used in the HHRA are EPAs default values from EPA (1991) Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors" (OSWER Dir 9285.6-03). Neighborhood recreational soil ingestion rates are EPA Region 10's default RME value for intermittent recreational exposure. See also response to Comment A6.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
153	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A20		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

The chronic RfD for lifetime arsenic exposure was used to evaluate the non-cancer risks associated with arsenic ingestion during childhood. A general principal of toxicology is that the dose controls the effect and that the dose is a function of both the amount of daily chemical intake and the length of exposure. Thus, a chemical dose tolerated for a short period of time might cause problems over a longer period of time (chronic exposure) because of cumulative effects. The exposure time for young children is limited to their early childhood (6 years). Reviews of the available data by EPA Region VIII (Benson 1995; 2000) and Exponent (Tsuji et al. 2000) have not found children to be more sensitive than adults to arsenic exposure, except at high doses when acute poisoning occurs. At the lowest doses, the population most at risk is older individuals. Therefore, there is no technical rationale for applying the lifetime chronic RfD to a childhood exposure scenario or for an assumption that children may be more sensitive at low doses than older age groups who have had exposure since birth.

Response>>

The issue of child-specific risk evaluations is an area of ongoing research. While some studies indicate children are not a more sensitive population to arsenic than adults, other studies indicate that they might well be (e.g., Concha et al, 1998 as cited in the NRC report on arsenic in drinking water, 1999). The NRC report concluded that the issue of sensitivity needs more study and in the absence of definitive information, recommended a health-protective approach. We agree and thus did not use the Benson (2000) estimate of a sub-chronic RfD for exposures of less than 10 years. In addition, the Benson (2000) estimate for a subchronic RfD is currently undergoing peer review, and we also considered use of his estimate in advance of peer review to be premature. Therefore, use of the chronic RfD was assumed to be the best available current estimate for use in the child non-cancer calculations. In the absence of peer-reviewed subchronic RfD's, EPA Region 10 (USEPA Region 10, 1999: Region 10 Supplemental Guidance, Assessing Childhood Exposures for Noncarcinogens) policy is to utilize the chronic RfD. Region 10 further states, regarding the use of chronic RfDs for childhood exposures:
"..this risk assessment policy advocates a prudent public health approach of not allowing children's exposures to exceed those allowed for adults when there is no Agency accepted child-specific toxicity value which is specifically developed to be protective of children's health. It's possible that the use of the chronic non-cancer toxicity values may not be protective of children's health. " See also Dr. Paul Mushak's comments regarding the use of the RfD for childhood exposures.

Misc. Input>>

154	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A21		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

The estimated cancer risks from arsenic were between 1 x 10-4 and 1 x 10-6 for the child/adult (0 to 30 years) scenario. However, these risks were identified using a linear slope factor to describe the arsenic dose-response relationship, even at low doses. As stated in the HHRA, this approach assumes that there is no threshold for the initiation of toxic effects, such that no dose, no matter how low, is without some risk of cancer. Because the dose-response relationship has not been observed at low doses and is based on extrapolation of high dose observations, the cancer risk predicted for low doses may be grossly over-estimated.

Response>>

We agree that there is some evidence that arsenic-induced carcinogenic responses have a threshold. However, specific modes of action as discussed in EPA's 1996 Cancer Guidelines have not yet been identified for arsenic. Until this occurs we agree with the National Research Council (NRC) recommendation that it is prudent not to rule out the possibility of a linear response.
While a discussion of arsenic toxicity issues is appropriate for the uncertainty section of the risk assessment (which currently notes these issues), evidence is insufficient to change the quantitative calculations and any future risk management decisions based on arsenic. See also Dr. Paul Mushak's comments regarding potential for a threshold response for arsenic.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
155	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A22		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Based on the factors described above, the HHRA's characterization of arsenic risks for the evaluated exposure scenarios very likely over-estimates the potential risks from arsenic exposure in the Basin and could lead to the selection of an overly conservative action level for arsenic in soils. Although risk-based action levels for arsenic are not identified in the HHRA, the draft includes a discussion of the potential for background levels of arsenic to contribute significantly to the arsenic-related health risks described for the Basin. This discussion prompts the question whether the risk assessment methods would identify non-cancer health hazards or unacceptable cancer risks when the arsenic concentrations used in the various exposure scenarios are within the range of background conditions.

Response>>

We agree that risks are likely over-estimated; however, erring on the side of over, rather than under, estimation is necessary to fulfill EPA's mandate to protect human health with an adequate margin of safety. The discussion on Page 7-19 of the HHRA indicates that risks greater than 10E-4 (i.e., "unacceptable") would not occur at background arsenic soil concentrations; however, whether or not there are risks at background concentrations is irrelevant. Superfund cleanups do not address background risks and do not remediate below background levels. Background concentrations and incremental risks above background are taken into consideration when risk management decisions are made.

Misc. Input>>

156	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A23		Hecla Mining and ASARCO	2-Section 2	p.2-7		Not Accepted

Comments>

House dust sample collection methodology

An unconventional methodology was used to collect house dust samples. The section on house dust (beginning on page 2-7) notes that vacuum bag samples give a general representation of lead concentration in the home, while dust mats provide lead concentration, dust loading rate and lead loading rate. The text should also clarify that the dust mats were placed just inside the entryway of the home and therefore are representative of dust deposition at the entryway. Collection of dust data from mats in this way differs from the methodology used at most sites to characterize house dust. The dust on a mat just inside an entryway of a home may not accurately reflect indoor dust exposure in a house. The dust mat may be indicative of dirt that is initially tracked in, but it can also contain more lead originating from deteriorating lead-based paint on porches, doors and door frames, which can receive considerable wear. Because many of the homes in the area are quite old, lead-based paint is likely to be present. The lead paint that could have been applied in these high-use areas may also have been a more durable type which historically contained a higher percentage of lead. Similarly, sampling of residents' vacuum bags is also not the most accurate method employed at sites, since the vacuum cleaner can be used on non-living areas such as the car. The preferred method for sampling would be to collect calibrated vacuum floor samples from living areas of the house that are frequented by young children, such as a child's bedroom, the living room and kitchen. This method can give both lead concentration and loading data that are more representative of the indoor dust contacted regularly by the occupants.

Response>>

The HHRA disagrees with this comment. Dust lead exposures were measured by two independent techniques. Samples were collected from home vacuum cleaners, if these were available and had not been used outside or in the family car, and by entryway mats. Both techniques measure lead concentration in the minus 175 micron fraction of dust. This vacuum bag method has also been continuously used in the BHSS since 1974 and has been a significant correlate of both blood lead and soil lead levels. The second technique measures both dust lead concentration from the same size fraction, and the accumulation rate of both dust and lead on the mat. The accumulation rate of lead or lead loading rate on these mats was the single strongest environmental source correlate with blood lead in the site-specific analysis (r=0.634). Both methods have also been employed at socio-economically and demographically similar homes outside the Basin. These results do show that older homes have higher dust lead concentrations and loading rates, but at significantly lower levels than observed in the upper Basin. See also General Response to Comments, #3d.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
157	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS and TG
A24		Hecla Mining and ASARCO	2-Section 2	p.2-9		Partially Accepted

Comments>

Waste pile sample collection

Waste piles are coarse-grained, rocky and typically contain few fines. As noted on page 2-9, because of the rocky nature of the waste piles, not enough fines could be collected from the 0- to 1-inch interval for sampling. Instead, samples were collected from the 0- to 6-inch interval and sieved prior to analysis. The difficulty in collecting fines at the exposure point implies that children would have little exposure to fines on waste piles and that the waste pile exposures presented in the HHRA are likely to be over-estimated.

Response>>

Fine material is present in the top inch and this material would stick to children's hands and be ingested; however an insufficient amount was present for laboratory analysis. The assumption is that the concentration found in the 0-6 inch depth is representative of the concentration in the top inch.

Incremental exposures for lead at waste piles do not distinguish among waste pile types and surface characteristics. Incremental intake rates were developed for both members of the population, one for the typical (Central Tendency (CT)) and one for the reasonable maximum exposure (RME). Estimating the intake rates is a relatively straight-forward procedure utilizing exposure factors developed elsewhere in the document. Generally, these factors are linear and intake estimates are proportional to exposure point concentrations, contact times, and exposure frequencies. Should risk managers disagree with the underlying assumptions or wish to consider alternative factors, the incremental intake rates can be adjusted accordingly. This option is discussed in more detail in General Response to Comments, #5a. See also Dr. Muchak's comments on this issue.

Misc. Input>>

158	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A25		Hecla Mining and ASARCO	2-Section 2	p.2-5		Accepted

Comments>

Water potato collection methodology

The methodology used to collect samples of water potatoes was not provided in the HHRA. The methodology used by the Coeur d'Alene Tribe to collect and process water potato samples (noted on page 2-5) should be stated in the report or referenced. In particular, the report should identify whether proper chain of custody and quality assurance/quality control (QA/QC) procedures were used. In addition, control samples should have been taken from an area unaffected by tailings to distinguish the incremental amount of exposure due to increased lead in sediments.

Response>>

The HHRA agrees with this comment. The report regarding collection of water potato data will be included in the Appendix of the final HHRA.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
159	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS and TG
A26		Hecla Mining and ASARCO	2-Section 2			Partially Accepted

Comments>

Geographic sub-area selection

The selection of geographic sub areas presented in the HHRA does not represent reasonable human exposure potential. Basin geographical sub areas, or conceptual site model units, are introduced in the HHRA in the beginning of this section. These sub areas, however, were selected on the basis of defining the conceptual site model for the Ecological Risk Assessment and are organized based on stream drainage areas and morphology. However, this method of geographical division has little relevance for human exposures. For example, several of the stream segments lack human populations. Also, in some cases, the sub areas encompass unrelated communities on either side of the river. A more appropriate division would focus on communities or populations with similar characteristics.

Response>>

The selection of geographic sub-areas for human health exposure purposes is described in detail in Appendix N. The maps and divisions in Section 2 were intended to provide a transition from the divisions used in the RI and the EcoRA to the HHRA. The divisions used in the HHRA are explained in Section 3. We acknowledge that human exposures would only occur in a portion of the large "exposure areas" identified on the maps in Section 3 and would be centered around homes and recreation areas. In general, the majority of the data used in the HHRA was at or near a home and/or population center and does represent reasonable human exposure potential (see the figures in Section 3). The HHRA identified certain activities that could be "risky" depending on actual concentrations and frequency of use at a specific site, e.g., recreational activities in the Lower Basin. Remedial actions will be made on a home-by-home basis and will not occur without sampling (if there is no data). Common use area remedial activities would be determined on a site-by-site basis and involve the local communities. See also response to Comment B28. The text will be amended to clarify this issue.

Misc. Input>>

160	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A27		Hecla Mining and ASARCO	2-Section 2	p.2-15 and p.2-16		Accepted

Comments>

Background concentrations in surface water and groundwater

Background concentrations of surface water and groundwater appear to reflect dissolved concentrations; however, total concentrations were used for site data. Pages 2-15 and 2-16 note that background samples of surface water and groundwater were measured for dissolved metals. If these samples are compared to the site data as total metal concentrations, this comparison represents an inappropriate use of the background data. The total metal concentrations in the background samples are the appropriate data for such comparisons.

Response>>

Background surface water concentrations used in the risk assessment were in fact total concentrations, not dissolved concentrations. The discussion on page 2-15 will be amended to clearly explain this.

The only background groundwater concentrations that were available were for dissolved metals. We agree that comparison of these dissolved concentrations with the total measured concentrations is not appropriate. Therefore, these background values were provided for informational purposes only and are not included on any of the tables or in any of the calculations. This is discussed on page 2-16, but further discussion will be added to clarify this point.

Misc. Input>>

161	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A28		Hecla Mining and ASARCO	2-Section 2			Not Accepted

Comments>

Screening arsenic concentrations in surface water

Use of the ambient water quality criteria (AWQC) to screen arsenic concentrations in surface water is highly inaccurate. It should be recognized that very little of the arsenic present in fish is in the form of inorganic arsenic as is assumed by the calculations that underlie the AWQC. The AWQC also incorporates a bioconcentration factor of 44 that is biased upward greatly by considering the bioconcentration factor for bivalves, a factor which was found to be 350 times greater than that for fin fish (USEPA 1980). Since few edible bivalves are available in the Coeur d'Alene River, any calculations of arsenic uptake into edible aquatic organisms should use the bioconcentration factor for fin fish of 1.

Response>>

We acknowledge that the AWQC for arsenic does not represent exposures in the Basin. However, as a screening tool the AWQC is widely used and simply selects arsenic as a chemical of potential concern (COPC). Arsenic would still have been selected as a COPC based on its exceedences over the drinking water Maximum Contaminant Level (MCL) if we had not used the AWQC. The risk calculations assumed a reasonable maximum exposure to surface water while swimming and/or playing in surface water.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
162	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A29		Hecla Mining and ASARCO	2-Section 2	p.2-6		Accepted

Comments>

Yard soil collection results

Discrepancies were observed in yard soil collection results. Page 2-6 notes that a strong correlation was observed between the results of soil sampling surveys conducted by IDHW together with the Agency for Toxic Substances and Disease Registry (ATSDR) and those conducted by EPA; however, the lead concentrations determined by the EPA protocols may be higher than those observed in the IDHW/ATSDR survey. As a result, the data were combined from the two surveys for all metals except lead. The text should note how lead was handled as a result of this discrepancy.

Response>>

The HHRA agrees with this comment. The combining of soil survey data for HHRA purposes is described in Appendix N. This information will be added to the text in the final HHRA. See also General Response to Comments, #3c.

Misc. Input>>

163	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A30		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Exposure frequency

For arsenic and other metals, 350 days per year of exposure to soil was assumed. Given that the ground surface is frozen and/or snow covered during winter months, this exposure frequency is unrealistically high and should be adjusted to reflect climate conditions in the Coeur d'Alene Basin.

Response>>

See response to Comment A19.

Misc. Input>>

164	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A31		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Soil ingestion rate

The soil ingestion rate used for the recreational scenario is greater than that assumed for an RME scenario. The recreational soil intake rate is assumed to be 300 mg/day which is the 90th percentile for young children ages 1-5 years old while camping in summer (Van Winjen et al. 1990). This rate is assumed for children between the ages of 4 and 11 years for 34 days per year (i.e., once per week for 34 weeks). EPA Region X guidance specifies this rate for recreational activities that might occur for part of a week; during a few weeks of the year because of the lack of averaging over a longer time period. Thirty-four days spread out over April through October from ages 4 to 11 years is a sufficiently long time period for averaging. Children in this older age group would also not have the soil ingestion rate of the 90th percentile of children ages 1-5 years. The RME soil ingestion rate for children older than age 6 is 100 mg/day and for those from 0 to 6 years is 200 mg/day.

Response>>

We acknowledge that EPA's Region 10 guidance memorandum refers to "a few weeks," and that a specific length of time for which the higher ingestion rate should apply is not defined and requires professional judgement. We disagree that the averaging time is sufficiently long to use the average yearly ingestion rate which is based on 350 days per year and selected the more health-protective approach. The days per year of exposure for neighborhood receptors were all less than 10% of the yearly value. Neighborhood park use was assumed to be 34 days per year, waste pile exposure was 17 days per year, and floodplain soil/sediment exposure assumed 21 days per year. See also response to Comment A6.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
165	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A32		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Dermal exposure pathway

Including the dermal pathway in the quantitative risk analyses is unnecessary for metals. Quantitative analyses are unnecessary for the protection of health because the EPA reasonable maximum exposure (RME) assumptions for the soil ingestion pathway are sufficiently conservative to more than adequately make up for the relatively small amount of dermal absorption of metals in soil. Evidence in support of this conclusion includes biomonitoring studies of total inorganic arsenic exposure compared to RME dose calculations via soil ingestion and the low relative bioavailability of arsenic in mine waste via the dermal pathway. Comparison of RME estimates of dose from soil with biomonitoring data from the Tacoma smelter site indicated that the soil ingestion assumptions were consistent with the 95th percentile urinary arsenic value (EPA Region X risk assessment for the Ruston Community; Glass and SAIC, 1992). The urinary arsenic data used in this comparison likely reflected more than soil exposure because these measurements were taken during and shortly after smelter operation when arsenic air levels and dust concentrations were higher. Thus, the RME estimates likely over-estimate actual exposures.

A similar examination for the Anaconda site in Montana also showed that the EPA soil ingestion assumptions for central tendency and RME estimates characterized the majority of urinary levels and the upper percentile levels, respectively, in the community (Walker and Griffin 1998). Calculations for both of these sites also over-estimate the arsenic dose resulting from soil because the amount of exposure due to inorganic arsenic in the diet was not adequately accounted for. Consequently, these studies have not found under-estimation of exposure that might be due to another pathway such as dermal absorption.

Response>>

We acknowledge that there is considerable uncertainty in the dermal pathway. We also note that this pathway is not a risk driver. In the absence of more information, we make the health-protective assumption to consider the potential contribution of the dermal pathway to total risks for arsenic and cadmium. We also acknowledge that the dermal pathway may have been over-estimated for the neighborhood recreational exposures because of the highly conservative estimates of skin surface area. A table will be provided which shows how alterations in skin surface area will affect calculated risks and hazards. See also response to Comment B15.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
166	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A33		Hecla Mining and ASARCO	3-Section 3	p.3-47		Not Accepted

Comments>

Dermal absorption of arsenic

In the HHRA, the dermal absorption of arsenic from soil is estimated based on data for soluble arsenic. The dermal exposure pathway (page 3-47, fourth paragraph) assumes 3 percent dermal absorption of arsenic based on studies of soluble arsenic freshly added to soil and applied to monkey skin (Wester et al. 1993). As recognized by the detailed discussion on gastrointestinal absorption (next four paragraphs on page 3-47), arsenic in soil from mining sources is likely to be less soluble and to have a lower bioavailability than arsenic from other sources (e.g., 60 percent gastrointestinal bioavailability is assumed as a default value for mining materials versus 80 percent bioavailability assumed as a default gastrointestinal absorption value for arsenic from other sources). At a minimum, the same relative correction factor should be applied to the assumed 3 percent dermal absorption for soluble arsenic (i.e., 0.75, or 0.6/0.8). The resulting absorption factor would be 2.25 percent. This adjusted value is still likely to over-estimate actual dermal absorption for these materials based on the results from human cadaver skin found by Wester et al. (1993) which were lower (0.8 percent) than the in vivo results observed for monkey skin. Because the dermal absorption of arsenic from weathered soil would be less than that from soluble arsenic in soil, the resulting risk estimates are very likely to over-estimate the magnitude of this exposure pathway.

Response>>

We agree that the absorption factor of 3% may overestimate the amount of mining-derived arsenic in soil entering through the skin. However, in the absence of more studies, this value is selected as health-protective. We do not agree that the value should be adjusted by 75%. The 60% factor is from a gastrointestinal absorption study in pigs and it is not known whether gastrointestinal absorption in pigs is comparable to absorption through monkey skin.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
167	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS and TG
A34		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Homegrown vegetable exposure pathway

Ingestion of homegrown vegetables is an exposure pathway of little concern for arsenic and lead and should be excluded from the quantitative risk analyses. Uptake ratios for arsenic and lead into vegetables have been found to be low (Glass and SAIC 1992; ISSI 2000), and biomonitoring data from many sites, including the Basin (IDHW 1999), have not indicated that ingestion of homegrown vegetables contributes to elevated lead and arsenic exposure in residents (Polissar 1987; Polissar et al. 1990; Hwang et al. 1997; University of Cincinnati 1997a,b; Advanced Geoservices 1996; ATSDR 1994; Bornschein et al. 1991; ATSDR 1992; Colorado Department of Health 1994; BSBDH and University of Cincinnati 1992). In fact, IDHW (1999) showed that the Basin residents who consumed vegetables had lower blood lead levels than those who did not. This empirical data contradicts the HHRA risk prediction that nearly all children from all communities would have a blood lead level over 10 µg/dL if they have incremental exposure to lead from homegrown vegetables.

Much of the arsenic in vegetables such as potatoes, tomatoes, carrots, beans and onions is in the relatively non-toxic organic form (Yost et al. 1998; Schoof et al. 1999). Only the percentage of arsenic that is in the inorganic form should be used in calculating health risks using the arsenic toxicity criteria.

Response>>

Arsenic

We agree this is likely not an important pathway for arsenic. However, there is some contribution to total arsenic exposure from the garden pathway and the risk assessment looked at all potential exposures, given the many sources that are present in the Basin, to assist the risk managers in making the most informed decisions. The HHRA noted that the vegetable pathway is semi-quantitative and did not include the calculation results in the total risk and hazard estimates. We acknowledge that arsenic is not 100% in the inorganic form in all foods. However, our reading of the Yost and Schoof papers cited here indicates that 100% of the arsenic in some terrestrial foods could be inorganic and that ranges were 25% to 100%. The Schoof paper concludes that rice and produce are likely to be significant contributors to dietary inorganic arsenic intake. In addition, the work cited in the Schoof and Yost papers was a survey of market produce, not produce grown on arsenic-contaminated soils. The Schoof and Yost papers suffer the shortcomings of the analytical method which was used, where the extraction efficiency (recovery), mass balance and/or integrity of species during sample handling were not evaluated or reported. Controlled experiments indicate that edible produce can accumulate high concentrations of inorganic arsenic when the contaminant is present. In the absence of site-specific speciated data, the 100% assumption is not unreasonable. See also Dr. Paul Mushak's comments on garden vegetable issues.

Lead

"Eating root or leafy vegetables from the household garden or another local garden in the twelve months prior to the study was associated with lower blood lead levels in children less than ten years of age. Better nutrition in this group or socioeconomic factors, such as increased health awareness in the gardening group, may explain this finding. Local vegetables were
not tested for lead content in this study" (ATSDR 2000). The HHRA risks were based on samples collected from local gardens and associated intake rates calculated using typical exposure factors.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
168	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A35		Hecla Mining and ASARCO	0-Executive Summary	p.3-38		Partially Accepted

Comments>

Use of house dust data

House dust data for non-lead metals were ignored and house dust was assumed to be 100 percent soil. Page 3-38 states, "soil is assumed to be a major contributor to indoor concentrations of chemicals in dust. However, yard soil concentrations may be good predictors of some, but not all chemical concentrations in dust." Given that statement, it seems illogical for the calculations in the HHRA to incorporate the assumption that house dust metals concentrations are equal to soil concentrations. Moreover, it is inappropriate for the analyses to be conducted ignoring all of the house dust data for non-lead metals while retaining the house dust data for lead. the metal most likely to have biased house dust data due to non-soil sources (e.g., paint). Data from other sites without active air emissions sources have shown that indoor house dust concentrations for arsenic for example, are lower than outdoor soil concentrations (CDM 1996; University of Cincinnati 1997).

Response>>

We acknowledge that there is uncertainty associated with the assumption that yard soil concentrations are an adequate surrogate for house dust concentrations. We direct your attention to pages 7-14 through 7-16 of the HHRA where this issue is discussed further. The primary reason the data were not used in the risk and hazard calculations was because of the uncertainty of the relationship between soil and dust for the non-lead metals, making a quantitative prediction of dust concentrations where we did not have data highly uncertain. The uncertainty in predicting dust concentrations from soil concentrations was considered more problematic than simply using the soil data. In addition, the majority of risk assessments to date do not have indoor dust concentrations for chemicals other than lead, thus using the soil data as a surrogate has precedence throughout the country. Paired soil and dust data for lead were available for over 800 homes. Therefore, the lead risk assessment, in addition to having a great deal more information for each of the 8 geographic subareas, did not have to predict a relationship in the absence of data. In addition, unlike lead, the soil-dust relationships for other contaminants occurring at sites is not nearly as well understood or characterized. The text will be revised to clarify the non-lead metals dust issues.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
169	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A36		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Combination of exposure parameters

Many of the exposure pathways quantified in the risk assessment include multiple exposure parameters in the risk calculations. In interpreting the results of these calculations, the technical basis and reasonableness of each individual parameter as well as the implications of the combined parameters must be considered. For example, calculations for the dermal exposure pathway for soil and sediment include numerous assumptions including those regarding the frequency of exposure, the potentially exposed skin surface area, the adherence of soil to skin, and the dermal absorption of the chemicals of potential concern (COPCs) from soil or sediment. Using the typical reasonable maximum exposure(RME) approach to such calculations, the combined assumptions for these parameters generally assume that an individual has extensive contact with soil or sediment over the entire possible assumed skin surface area on every day that contact with soil or sediment occurs. The calculations also inherently assume that the soil or sediment remains in contact with the skin sufficiently long for the skin to absorb the entire absorbable fraction of the COPC from the soil or sediment. In actuality, contacts with soil or sediment will vary from event to event. For example, the extent of skin coverage by soil or sediment will differ from event to event, and bathing or other contact with water may remove some or all of the soil or sediment before the entire absorbable fraction has been absorbed.

The combinations of conservative assumptions used to develop exposure scenarios result in even greater over-estimates of actual exposures that are highly improbable. For example, as discussed in more detail below, the combined exposure assumptions for the subsistence exposure scenario yield a total exposure level that is highly unlikely to actually occur. The highly conservative nature of such calculations should be acknowledged in interpreting the risk assessment results.

Response>>

Generally, we agree with the commenters that risks are likely over-estimated. The reasonable maximum scenario (RME) is designed to over estimate risks for most of the population so that any interventions will err on the side of being health protective. While the RME scenarios appropriately use high-end estimates of exposure for many parameters in order to be health protective, some exposure parameters are averages rather than high-end (body weight and skin surface areas). The combination of high-end and average assumptions results in reasonable maximum estimates of health risks.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
170	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS and TG
A37		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Subsistence scenarios

As previously stated in the general comments, inclusion of tribal exposure scenarios that do not presently exist and not are likely to exist in the future is inappropriate for the baseline risk assessment. In addition, both the individual assumptions and the combinations of assumptions used for characterizing tribal exposure are unreasonable. For example, for the traditional scenario, tribal members are assumed to camp on the river all year long and have the following exposures every day of the year from birth to 70 years (except for dirty surface water and sediment exposure as noted):

- Ingest 300 mg/day of soil, which was measured by Van Winjen et al. (1990) as the 90th percentile soil intake rate for 1-5 year old children while camping in summer

- Ingest an additional 300 mg/day of sediment

- For seven months of the year, have the whole body covered with sediment in a layer that is four to eight times thicker (i.e., 0.8 mg/sq.cm) than the amount assumed for the RME residential scenario

- For 365 days per year, have arms, head, forearms, hands, lower legs, and feet of 0-6 year old children covered with soil and have face, hands, and forearms of adults covered also with the thicker layer of soil as assumed for sediment (i.e., 0.8 mg/sq.cm). (This set of unrealistic factors assumes that children wear short sleeve shirts, short pants, and no shoes, even in winter. The total amount of soil and sediment coverage on these exposed surfaces for seven months of the year would be 1.6 mg/sq.cm. The thickness of this layer may exceed the thickness of the monolayer of soil on the skin within which absorption occurs, thus impeding complete chemical absorption from these materials.)

- Drink 30 ml/day of dirty water in the river from freshly kicked up sediments seven months of the year

- Consume their entire vegetable and fruit intake as water potatoes (which may not be peeled) grown in sediment

- Consume 540 g of possibly whole fish (including gills and liver which contain more metals) per day. (This amount is considerably higher than the 170 g/day assumed for the current subsistence scenario, which is based on the 95th percentile consumption rate for four Columbia River tribes. The Coeur d'Alene River is much smaller than the Columbia River and may not support this level of fish intake.)

- Drink 150 percent more surface water from the Coeur d'Alene River than the RME residential consumption rate during every day regardless of season.

Response>>

The HHRA disagrees with this comment. A baseline risk assessment appropriately includes potential future use scenarios. The two subsistence scenarios were requested by the Coeur d'Alene Tribe to represent possible future uses of the area. The specific exposure factors were developed in cooperation with Coeur d'Alene Tribe representatives. A cultural anthropologist, working for the Coeur d'Alene Tribe, reviewed and suggested appropriate modifications for each of the exposure factors. While some of the pathways are likely over-estimated, numerous other potential pathways could not be addressed because of lack of data (see discussion in Section 3.2.4, pages 3-28 - 3-34). See also General Response to Comments, #6.

We refer the commenters to the key study by Harris and Harper in Risk Analysis several years ago, supporting the figure of 300 mg/d for soil, 300 mg for sediment, etc. The paper is:

Ref: Harris SG, Harper BL. A Native American exposure scenario. Risk Analysis 17: 789-795 (1998).

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
171	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS and TG
A38		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Subsistence scenarios

As previously stated in the general comments, inclusion of tribal exposure scenarios that do not presently exist and not are likely to exist in the future is inappropriate for the baseline risk assessment. In addition, both the individual assumptions and the combinations of assumptions used for characterizing tribal exposure are unreasonable. With the exception of the fish and water potato ingestion scenarios, the current subsistence scenario has the same high exposure rates as the traditional scenario except that it assumes 61 days per year of exposure instead of 365 days per year of exposure.

Response>>

We disagree that the exposure assumptions are unreasonable. See response to Comment A37. Note that the differences in exposure duration and fish and water potato ingestion rates between the modern and traditional subsistence scenarios result in risks and hazards that are approximately 80% lower for the modern scenario than for the traditional scenarios. See also General Response to Comments, #6.

Misc. Input>>

172	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A39		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Combinations of exposure scenarios

The HHRA calculates exposures and risks for a baseline residential exposure scenario as well as for a variety of “incremental” exposure scenarios, e.g., recreational exposure scenarios. The potential exposures and risks for individuals participating in activities covered in different exposure scenarios (e.g., a resident who takes part in the evaluated recreational activities) are then calculated by adding the risk estimates derived for the chosen incremental scenario to those calculated for the baseline scenario. These calculations are not adjusted to account for “double-counting” of exposures, and thus result in over estimates of exposure and risk. For example, when adding a recreational scenario to the residential scenario, the incidental soil ingestion that is assumed to occur as part of the residential exposure scenario is assumed to be unaffected by the additional soil or sediment ingestion that is assumed to occur during the recreational activities. Because some of the incremental exposure scenarios assume relatively frequent and extensive exposures to recreational areas and relatively high contact rates during these activities, it is likely that the baseline residential exposures would decrease if the assumed levels of recreational activity were in fact occurring. Ideally, the results of the risk calculations should be adjusted to account for the double-counting of exposure. The likely over-estimate of risk reflected in the current procedure for combing risk calculations for multiple scenarios must be accounted for when interpreting the risk assessment results.

Response>>

Only the residential and neighborhood recreational scenarios were combined in one table in Section 5 where they were combined in a very qualified manner, primarily to illustrate the potential for additional exposures outside the home, and the potential "double-counting" was acknowledged. They were combined to demonstrate that risks might increase over baseline residential risks if residents also engaged in recreational activities. The last paragraph on page 5-11 acknowledges that the risks calculated from this combination of exposures is likely overestimated because of "double counting". However, more text will be added to clarify this point. See also General Response to Comments, #5.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
173	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A40		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Characterization of lead toxicity

In the toxicity profile for lead, as well as elsewhere in the document, the HHRA provides little information regarding how the nature and severity of effects associated with lead exposure vary with the extent of lead exposure.

In fact, as is typical of most dose-response relationships, both the severity of effect and the strength of the evidence linking exposures and effects decrease as the degree of exposure decreases (see, e.g., ATSDR 1999). For example, the cognitive effects of lead are less at lower blood lead levels (e.g., 10-14 µg/dL) than at higher blood lead levels (e.g., 20-30 µg/dL). In addition, the evidence linking low blood lead concentrations and specific adverse effects is weaker than the evidence associated with higher blood lead concentrations (Pocock et al. 1994). Frequently, conclusions regarding the effects of low blood lead concentration are derived from studies of large numbers of children over a range in blood lead levels. Although a linear statistical relationship can sometimes be extrapolated from the blood lead and cognitive test data compiled in such studies, conclusions regarding the potential impacts of low blood lead concentrations are rarely based on empirical observations at those concentrations. As a result, questions exist regarding whether such a relationship exists at low blood lead levels, the form of any such relationship, and the potential persistence of any such effects. Moreover, at low blood lead concentrations, the effects of lead exposure on health endpoints such as cognitive function are difficult to distinguish from the effects of other important influences such as socioeconomic status or nutrition. All of these factors make it important to distinguish between the magnitude of the effects associated with various blood lead levels and exposure durations, and the degree of certainty regarding the likelihood that such effects may occur or persist.

The importance of distinguishing among various lead exposure levels (as reflected in blood lead concentrations) is reflected in the Centers for Disease Control (CDC) guidance for screening children's blood lead concentrations (1991, 1997). This guidance identifies actions required for various blood lead concentration ranges based on the anticipated severity of the health effect as well as the likely effectiveness of the suggested intervention. At low blood lead concentrations in the 10-14 µg/dL range, education and follow-up testing are the primary actions recommended. Characterizing this concentration range as a "border zone," CDC does not recommend other interventions because of concerns regarding the precision of laboratory results at these concentrations and because "it is unlikely that there is a single predominant source of lead exposure for most of these children" with blood lead concentrations in this range. More active interventions such as environmental investigation and lead hazard control are not recommended until blood lead concentrations are in the 20-44 µg/dL range. For children with blood lead concentrations associated with frank and severe toxicity (i.e., >45 µg/dL), active interventions including medical treatment are begun on an urgent basis.

Response>>

The HHRA disagrees with this comment. The comment misrepresent the strength of low-level lead effects as an accepted body of science in the clinical and public health mainstream. A figure or text will be inserted into the final document citing the 1997 and 1991 CDC Statements on lead poisoning in children and the relationships among different blood lead levels and observed health effects.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
174	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A41		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

For example, the toxicity profile discusses the types of health effects associated with lead exposures in a very general way, providing little information regarding specific blood lead concentrations that have been associated with specific categories or severity of effects.

Response>>

See response to Comment A40.

Misc. Input>>

175	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A42		Hecla Mining and ASARCO	0-Executive Summary			Accepted

Comments>

Similarly, the toxicity profile also notes in several cases that no threshold is known for some categories of effects; however, the profile fails to discuss differences in the severity of effects or the strength of the evidence of effects associated with different degrees of exposure.

Response>>

Dose-response relationships for lead will be cited in the final report.

Misc. Input>>

176	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A43		Hecla Mining and ASARCO	0-Executive Summary			Accepted

Comments>

The HHRA also includes a number of statements suggesting that serious adverse health effects associated with lead exposure can be associated with “relatively short-term exposures on the order of months.” Again, little context is provided regarding the severity and persistence of the effects associated with various exposure durations. This approach leaves the misleading impression that all of the types of effects discussed in the toxicity profile are associated with lead exposures at any level or for any duration.

Response>>

Effects of lead associated with various exposure durations will also be discussed in the final report.

Misc. Input>>

177	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A44		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

The HHRA also discusses the health effects associated with low level lead exposures using terminology that implies that such exposure levels are linked with the more severe effects associated with higher blood lead concentrations. For example, the risk characterization section for lead refers to the analyses conducted in the HHRA (i.e., the evaluations of the potential for blood lead concentrations to exceed 10 µg/dL) as having assessed “[t]he risk of lead poisoning,” a term typically equated in common usage with the more serious effects associated with high level lead exposures.

Response>>

There is a terminology in the HHRA being referred to that is lifted from the 1991 CDC Statement. In the summary portion of that document, lead "poisoning" is noted to not occur below 10 µg/dl. The precise quotation is on p. 3, Table 1-1, "A child in Class I is not considered to be lead-poisoned."

Misc. Input>>

178	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A45		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

In another portion of the risk characterization discussion, the report uses the term “toxicity rates” to refer to the percentages of children with blood lead concentrations exceeding target blood lead concentrations of 10, 15, or 20 µg/dL. This term has no scientific validity and again provides a misleading perspective on the potential impacts of the blood lead concentration ranges under discussion.

Response>>

This might be confusing terminology, however, again toxicity is referring to "poisoning" as cited in CDC (see Comment A44). There are known health effects associated with certain blood lead levels, which is why the rates or number of children with those blood lead levels is presented. Again, a figure or text will be inserted in the final HHRA showing blood lead levels and the associated health effects; this will help reduce any "...misleading perspectives on the potential impacts of the blood lead concentration ranges under discussion."

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
179	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A46		Hecla Mining and ASARCO	0-Executive Summary			Accepted

Comments>

To provide more useful information to the community and to risk managers, the HHRA should present information regarding lead toxicity that clearly distinguishes among the types and severity of effects associated with different blood lead concentrations. In particular, the HHRA should present information that is relevant for the types of exposure levels observed in residents of the Basin. This context is necessary to identify the nature of the actual health risks that residents of the Basin may encounter and to make informed decisions regarding appropriate remedial measures.

Response>>

The HHRA agrees with this comment. See response to Comment A40.

Misc. Input>>

180	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A47		Hecla Mining and ASARCO	0-Executive Summary			Accepted

Comments>

In addition, the data from the Basin are not completely presented to allow for independent verification of the comparison results or development of alternate analyses.

Response>>

Confidentiality of these data are protected under Idaho State Law. Censored and masked data sets have been developed to release the data in a format that does not compromise individual confidentiality. These data will be included in an Appendix of the final HHRA.

Misc. Input>>

181	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A48		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Role of socioeconomic status

A factor for socioeconomic status should have been included in the model assessing potential relationships among lead exposure sources. This factor has been shown to be highly significant and well-correlated with blood lead concentrations at this site (IDHW 1999) and others (Succop et al. 1998). This factor is also associated with some of the types of effects associated with elevated lead exposures. The true effect of the various environmental media cannot be identified without correction for socioeconomic status. If a correction factor is not included for socioeconomic status, other factors may become surrogates for this factor. For example, lead loading on dust mats (which is a function of dust loading in addition to lead concentration) may be indicative of home hygiene and house upkeep and age (e.g., older houses have more lead-based paint). Lead in soil and dust is affected by the age and condition of the paint, which are also related to socioeconomic status.

Response>>

The HHRA agrees with this comment. Socio-economic status is an important factor in childhood lead poisoning. However, there is not sufficient data available to include family-specific socio-economic variables in the site-specific analysis. With respect to sources of lead, soil and paint remain the most significant sources manifesting effects through house dust regardless of social status. Socio-economic factors can influence the strength and relative contribution of these sources, contributing to increased media concentrations, ingestion rates, and absorption. See General Response to Comments, #1a.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
182	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A49		Hecla Mining and ASARCO	6-Section 6	p.6-23, Figure 6-10a		Not Accepted

Comments>

Role of paint

Paint has an important and often hidden influence on dust and soil. Dust mat lead concentration was correlated to yard soil, community geometric mean soil, and paint lead levels. One should recognize, however, that these correlations may be greatly influenced by the effect of eroding paint on soil, house dust, and the dust mat. When an independent variable affects several dependent variables, correlations will appear among the dependent variables. Although yard soil likely affects dust mats and house dust by tracking, the strength of this correlation will be increased by the influence of an independent variable such as paint on both soil and dust. As noted at the end of the first paragraph on page 6-23, homes with poor paint condition also show increased mat dust lead concentrations.

The effect of paint on blood lead levels is a function of the paint concentration and the condition of the paint. Both of these factors need to be combined in the analysis. Thus, although the scatter plots (Figure 6-10a) of paint concentration or condition separately considered relative to concentrations of lead in blood or dust show some variability, it is misleading to conclude that paint has less influence on blood lead levels than other factors. A combined correlation coefficient for the effect of paint concentration and condition should be considered. In addition, the soil concentration near the house (which could affect the dust mat lead concentration) may have been elevated by historical erosion of paint or removal before repainting of the house. Thus, even though the current paint condition may be good, lead paint may be present at the property in the soil and dust. This effect of lead-based paint would not be apparent from the analysis used by the HHRA. Although the HHRA does not show the correlation between paint factors and lead in soil, the summary of the various environmental parameters by geographic sub area indicates that such a correlation may exist.

Given these relationships between lead-based paint, paint condition and dust lead, substantial problems exist in identifying the effects of paint on environmental and blood lead concentrations. It is particularly difficult to evaluate the influence of paint concentrations on the ratio between the lead concentrations in dust and those in soil. As a result, the available data for arsenic or other metals without strong residential sources should be considered as a means for evaluating the relationships between indoor dust and outdoor soil concentrations. CDM (1996) and University of Cincinnati (1997) have noted that arsenic levels are generally lower in indoor dust than in soil relative to lead concentrations. These researchers have also noted that arsenic concentrations show a more accurate relationship between soil that is tracked or blown into the house and yard soil than do lead levels in these media.

Response>>

The HHRA disagrees with this comment. It is important to note that actual observed soil and house dust lead levels were used in both the site-specific and IEUBK model analysis that relate blood lead levels as a dependent variable to environmental dust concentrations. As a result, the sources of lead to soil and dust, such as paint, mineral industry wastes, yard soils, materials tracked in by workers, fugitive dusts, etc. are inherent in the analysis. Dependent blood lead levels are directly related to soils, house dust and other environmental sources as independent variables in either the empirical or mechanistic model derived analysis. Any significant effects in addition to dust from soil or paint are similarly independent and likely represent primary source pathways exclusive of house dust. The result is that lead in soils and dusts represent the primary risk, with house dust being most important to young children. Quantitative analysis of the dust lead pathway in the HHRA concluded dust lead loading is most influenced by outdoor soils, augmented by paint contributions in older homes, especially those in poor condition. The cross product of paint condition and lead concentration was not significant with relation to blood lead levels. Stratification of the database into homes by paint condition yielded insufficient observations to support rigorous analysis, although soil lead remained significant in both subsets. The conclusion of the combined analysis of blood-dust-soils-paint relationship is that, for young children in particular, house dust lead is the primary source of exposure followed by yard soils. Effective risk management strategies need to reduce house dust lead loading. Effectively reducing house dust lead loading requires addressing the principal sources of lead to house dust. Those sources are yard soils, community-wide soils, and paint lead in poor condition homes. These sources also present risk along independent pathways (i.e., direct contact) in addition to their role in dust lead. See also General Response to Comments, #3 and #4.

The HHRA agrees that additional analyses could provide more information for risk managers to consider in developing cleanup strategies. Suggestions were made regarding the inclusion of socio-economic variables and development of paint lead-paint condition interactive factors or cross products in these analysis. However, as was noted for the proposed socio-economic characterization of the blood lead data set, insufficient data are available to perform these adjustments. Suggestions were also made to perform separate analysis of homes with and without paint hazards. This analysis would also be difficult as most homes, other than trailer homes, have lead paint. The primary indicator of paint condition (peeling/chipping/chalking paint) has been shown in the parent 1996 Basin Exposure Study to be highly correlated with home hygiene and socio-economic status. As a result, it is not clear whether the significance of this variable is reflective of the paint source of lead, socio-economic status, personal and family behavior, home hygiene practices, or dust loading.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
183	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A50		Hecla Mining and ASARCO	6-Section 6	Figure 6-7, Table 6-13		Not Accepted

Comments>

The HHRA also presents a misleading perspective of the paint hazards within the specific geographic sub areas that were evaluated. Specifically, Figures 6-7a,b (Geometric Mean Interior and Exterior Paint Lead Concentration by Geographic Area) provide a misleading representation of potential paint hazards for the Burke/Nine Mile area. This area has the second highest median interior, exterior, and mat location paint concentrations behind Wallace (Table 6-13). Thus, a considerable number of homes in Burke/Nine Mile have elevated lead concentrations in paint. Combined with the likely lower socioeconomic conditions in this area and the dust loading in the homes, elevated blood lead levels here in response to such conditions are not surprising.

Response>>

The HHRA disagrees with this comment. The geometric mean paint lead variable was selected to illustrate paint lead concentrations consistent with the use of geometric means for other media, and was not intended to be misleading. Complete statistical summaries including arithmetic and geometric means, minimums, maximums, and medians are included in Table 6-13 in the HHRA, as noted. Each of these variable forms was included as candidate variables in the step-wise regression analysis.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
184	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A51		Hecla Mining and ASARCO	0-Executive Summary	p.6-20,6-24		Partially Accepted

Comments>

Correlations between lead concentrations in blood and environmental media

Results presented in the HHRA indicate that the lead loading rate on entry mats had a high correlation with blood lead levels whereas yard soil concentrations had low correlations with blood lead concentrations. Of the various environmental media examined for correlations with blood lead concentrations, the lead-loading rate on dust mats had the highest correlation (r = 0.63). The next highest correlations were observed for various factors for interior or exterior paint condition and concentration (r = 0.34 to 0.48), followed by mat lead concentration (r = 0.40). Yard soil and community soil had among the lowest correlations (r = 0.16 and 0.12, respectively). The correlation coefficients for soil and blood cannot be considered "high correlations or strongly correlated" as stated in the text (2nd paragraph, page 6-20; 2nd paragraph, page 6-24).

The more limited contributions of soil lead concentrations to blood lead concentrations are also supported by the analyses of the slope factor relating incremental increases in blood lead concentrations to increases in soil lead concentrations. Specifically, blood lead concentrations were found to increase by 0.7 ug/dL per 1000 mg/kg lead in home yard soil. This effect of soil lead on blood lead is lower than has been observed in the BHSS and at other sites (Succop et al. 1998), and may be suggestive of lower bioavailability of lead in soils in the Basin. This effect of soil on blood lead level is also 10 times lower than the relationship assumed by the IEUBK lead model. For a soil concentration change from 0 to 1,000 mg/kg, this model predicts about a 7 µg/dL change in blood lead and over 17 percent increase in risk of exceeding 10 ug/dL. Thus, the site data are at great variance with the IEUBK model predictions.

Response>>

The HHRA agrees with much of the discussion in this comment, however, there is disagreement with the conclusion. The accumulation rate of lead on entryway mats, or dust lead loading rate, was the single strongest environmental source correlate with blood lead in the site-specific analysis (r=0.63). Blood lead is also significantly correlated with median interior and exterior paint lead (r=0.341 and 0.407, respectively), yard soil lead concentration (r=0.158), and community-wide soil lead concentration (r=0.116). With respect to blood lead levels, regression analysis indicated that dust lead loading rate alone explained nearly 40% of the variance in the dependent variable. Other environmental variables were significant in combination with dust lead loading rate. Those variables were yard soil lead levels, median exterior paint XRF reading, and interior paint condition. Comparison of standardized regression coefficients indicate that soil lead and paint have similar effects on blood lead levels, somewhat less than dust lead loading. Both soil and paint, likely, manifest the greatest effect through the house dust pathway. Similar regression analysis indicate that dust lead content on these mats is most related to yard soil lead concentration. The next most significant variable is the community mean soil lead level at the p=0.0001 level followed by interior paint lead condition. No other variables are significant at the p=0.05 level in the presence of these factors. If community mean soil concentration is eliminated from the selection, the maximum interior paint lead XRF reading and the exterior median paint lead XRF reading are significant at the p= 0.02 and 0.03 level, respectively. Vacuum bag lead concentration is related to the mat lead concentration (p=0.001), yard soil concentration (p=0.01), and maximum interior paint lead XRF reading (p=0.03). Vacuum bag lead content typically exhibits about a 30% to 40 % lower concentration than mat lead content. The interpretation of these results in the HHRA was that contaminated soils, house dust, and lead based paint are all related to excess absorption. Overall this suggests complex exposure pathways, with blood lead levels most related to dust lead loading in the home, followed by independent effects of yard soil lead, interior paint lead condition, and exterior paint lead content. The dust lead pathway is most influenced by outdoor soils, augmented by paint contributions in older homes, especially those in poor condition. The slope values for soil and blood level are similar to, but somewhat less than, the BHSS, as noted. These relationships are not inconsistent with IEUBK dose/response relationships as the Box Model has accurately predicted blood lead levels at the BHSS for more than 10 years. These same regression coefficients were used in developing the site-specific parameters for that model. See also General Response to Comments, #7, #8, #9 and Appendix Q of the HHRA.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
185	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A52		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

The more limited contributions of soil lead concentrations to blood lead concentrations are also supported by the analyses of the slope factor relating incremental increases in blood lead concentrations to increases in soil lead concentrations. Specifically, blood lead concentrations were found to increase by 0.7 µg/dL per 1000 mg/kg lead in home yard soil. This effect of soil lead on blood lead is lower than has been observed in the BHSS and at other sites (Succop et al. 1998), and may be suggestive of lower bioavailability of lead in soils in the Basin. This effect of soil lead on blood lead is lower than has been observed in the BHSS and at other sites (Succop et al. 1998), and may be suggestive of lower bioavailability of lead in soils in the Basin. This effect of soil on blood lead level is also 10 times lower than the relationship assumed by the IEUBK lead model. For a soil concentration change from 0 to 1,000 mg/kg, this model predicts about a 7 µg/dL change in blood lead and over 17 percent increase in risk of exceeding 10 µg/dl. Thus, the site data are at great variance with the IEUBK model predictions.

Response>>

The HHRA agrees with the statement, but disagrees with the conclusion. The lower blood to soil slope is similar to that obtained at the BHSS and could be indicative of lower bioavailability or reduced ingestion rates. The Box model assumes lower bioavailability, although the discussions, uncertainty analysis, conclusions and qualifiers repeatedly acknowledge that lower ingestion rates associated with ever-present intervention efforts are also a likely explanation of the reduced response rate. However, the comparison of the slope values from the regression and the IEUBK is inappropriate, as it ignores the pathway effects noted in the studies referenced. These soil and dust data applied at 18% effective bioavailibility in the IEUBK model effectively describe observed blood lead levels in both the "Box" and the upper Basin. See also General Response to Comments, #9.

Misc. Input>>

186	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A56		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Geometric standard deviation

The geometric standard deviation (GSD) value is used to estimate the distribution of blood lead concentrations associated with the mean blood lead concentration calculated by the IEUBK model. The GSD is described by EPA as intended to reflect individual variability in blood lead concentrations that might result from a specified level of exposure. The default GSD value used in Version 0.99d of the IEUBK model, however, is based on review of community GSDs reflecting the variability observed in various studies of community blood lead concentrations. Community GSDs tend to be greater than individual GSDs because, in addition to the physiological and biological variability reflected in an individual GSD (e.g., due to differences in lead absorption in different individuals), the community GSD also reflects variability due to differences among individuals in the degree and types of lead exposures that they have (e.g., differing levels of exposure to deteriorated lead-containing paint). As demonstrated in many of the calculations presented in the HHRA and in other analyses (e.g., Bowers 1994), comparisons of predictions of the IEUBK model with observed concentrations in certain communities indicate that the IEUBK model over-estimates the number of children that may exceed a specified target blood lead concentration.

Response>>

The HHRA agrees with much of the discussion in this comment, however, there is disagreement with the conclusion. In the batch mode, the mean and probability to exceed toxicity criteria can also be determined and applied to the individual situation. For the individual situation, the GSD reflects only the inherent variation in response among individuals. The default GSD recommended by the EPA is representative of a number of investigations with varying degrees of exposure variation inherent in results. Applying the typical GSD value of 1.6 to individual situations could overestimate the probability of exceeding 10 ug/dl for the individual. Risk managers may want to consider the application of the 1.6 GSD in the batch mode application as an additional margin of safety when considering the probability of an individual exceeding toxicity criteria. This consideration would not apply to the community-wide estimates of the percent of the community to exceed these criteria. See General Response to Comments, #9c.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
187	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A57		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

In addition, the default GSD is greater than the community GSDs observed in some communities. For example, at the Sandy and Murray smelter sites in Utah, a GSD of 1.4 was calculated based on site-specific blood lead concentration data (U.S. EPA 1995a, Griffin et al. 1999). Similarly, at the Bingham Creek Channel site, a site-specific GSD of 1.43 was derived (U.S. EPA 1995b). In its evaluations of these sites using Version 0.99d of the IEUBK model, EPA used the site-specific GSDs rather than the default value. These factors suggest that the default GSD represents a conservative estimate of this value and is likely to be one of the reasons that the blood lead concentrations predicted by the model are generally substantially greater than the observed blood lead concentrations.

Response>>

The HHRA notes these observations. However, because the Box Model does accurately predict observed GSDs and the percent to exceed 10 ug/dl, the comment seems superfluous.

Misc. Input>>

188	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A58		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Bioavailability

The EPA default version of the IEUBK modeling presented in the HHRA assumes that the bioavailability of lead in soil and dust is 30 percent, the default model value. This value was modified to 18 percent in the “Box model” version of the IEUBK modeling presented in the HHRA, based on site-specific analyses performed for the BHSS located in the center of the Basin. As discussed in the HHRA, the blood lead concentrations predicted using the Box model (which included other site-specific adjustments) more closely paralleled the observed concentrations than did the concentrations predicted using all default assumptions. This observation suggests that a lower bioavailability assumption may more accurately predict actual lead uptake, at least in some of the modeled exposure areas.

Because the concordance between the modeled and observed results varied from area to area, however, it is likely that the bioavailability of these materials also varies in different areas. Materials that may be present in various portions of the Basin include mine waste from different mines, milling residue, and smelter-derived materials. These materials are likely to differ with regard to several factors that can influence bioavailability such as composition, particle size distribution, and weathering. To make the IEUBK modeling results more useful, differences among the potential exposure sources need to be more carefully examined and incorporated into the modeling efforts. A better understanding of the contributions of various potential sources to total lead exposures will allow remedial decision-making to be more effectively focused on actual exposure sources.

Information from many other mining sites in the Western U.S. indicates that lead forms at these sites are generally lower than the model default (summary by Ruby et al. 1999). Simple, easy-to-use laboratory tests are also available to estimate lead bioavailability in soils. This in vitro method is also showing promising results in comparison with the EPA swine model for measuring bioavailability (Ruby et al. 1999). The systematic determination of bioavailability is strikingly absent from the HHRA and is a significant methodological problem, particularly when mini-dose human assays are available that have been performed on Bunker Hill materials (Maddaloni 1998).

Response>>

The HHRA disagrees with the comment. Although, in-vitro bioavailability data could be a useful addition to the information base considered by risk managers, the laboratory assay has not yet been accepted or validated. Dr. Chris Weis, the EPA PI, has advised against its current application, as the results are still preliminary. No bioavailability data has been collected in the Basin or the BHSS. Please see Appendix O of the HHRA for more information.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
189	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A59		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Soil/dust relationship

The IEUBK modeling presented in the HHRA assumes that indoor lead concentrations in dust are due to lead in soil, despite the empirical data shown elsewhere in the report that paint is another contributor to lead in house dust. The report also assumes that the dust mat lead concentrations are equivalent to indoor dust lead concentrations. As noted previously under data collection, dust mat lead concentrations are not necessarily equivalent to indoor house dust concentrations because lead from lead-based paint used on porches, doors and door frames can also be present on entry mats.

Response>>

The HHRA disagrees with this comment. In the IEUBK analysis, observed soil and dust lead concentrations are used. No consideration of the source of the lead in dust is inherent in the analysis. The model uses vacuum dust lead concentrations for house dust input and clearly states that vacuum dust lead levels are typically less than mat concentrations. Vacuum bag lead concentration is related to the mat lead concentration (p=0.001), yard soil concentration (p=0.01), and maximum interior paint lead XRF reading (p=0.03).

Misc. Input>>

190	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A60		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Dietary lead intake

The default dietary lead intake values applied in the IEUBK model primarily reflect data collected by the U.S. Food and Drug Administration (FDA). Lead levels in food sources have been gradually decreasing over time as a result of various reductions in lead contamination of food, including reductions in the use of lead-containing solder in cans used for food packaging. The default assumptions applied in Version 0.99d of the IEUBK model reflect data collected by FDA during the early 1990s. In a recent application of the IEUBK model, EPA further reduced the dietary intake assumptions to 70 percent of the default values to reflect reductions in dietary lead intake that have likely occurred since the FDA data were collected (Griffin et al. 1999). Because the analyses presented in the HHRA do not account for reduced lead intake from this pathway, use of the default assumptions for this pathway will over-estimate lead exposure and blood lead concentrations. Data published by Manton et al. (2000) also convincingly demonstrated that dietary lead was not a contributor to the measured childhood blood levels. As the BLLs in the Basin continue to fall, the modeled relative contribution of dietary sources becomes more important and leads to further over estimation unless the HHRA continues to “back-titrate” the bioavailability parameter to account for errors in the default entry fields.

Response>>

The HHRA acknowledges the comment, but notes that dietary lead intake ranges from 10% to 20% of estimated total lead intake under current conditions. A thirty percent reduction in dietary intake estimates would have little impact in assessing the baseline situation. The effect, however, could be significant in formulating cleanup criteria, and could be considered a margin of safety for risk managers.

While dietary lead reductions have likely occurred since the data was referenced by the IEUBK, the EPA has not yet recommended how much lead intakes have declined, although the trend of declining dietary lead is generally accepted. The Griffin action has not been reviewed or accepted by the EPA TRW. There have been problems with some recent dietary lead data because non-detects have been reported as zero values in FDA summaries.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
191	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A61		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Maternal blood lead concentrations

The default maternal blood lead concentration used in Version 0.99d of the IEUBK model reflects the observed decrease in blood lead concentrations that has been observed in all age ranges of the U.S. population (see, e.g., Pirkle et al. 1994). This decrease has been attributed to the substantial reductions that have occurred in exposures to lead through such previously common lead exposure sources as residential paint, gasoline (and lead-bearing vehicle emissions), and solder used in food storage cans and drinking water pipes. National data indicate that decreases in the blood lead concentrations of women of childbearing age are likely to continue into the future. For example, data from the National Health and Nutrition Examination Surveys (NHANES) reported geometric mean blood lead concentrations in women between the ages of 20 and 49 years of 1.7 µg/dL (for non-Hispanic white women), 2.2 µg/dL (for non-Hispanic black women), and 2.0 µg/dL (for Mexican-American women) (Brody et al. 1994). This comprehensive, national study of a variety of health-related parameters was conducted during 1988 to 1991. For the next younger age range (12-19 years), the geometric means were 1.0, 1.8, and 1.5 µg/dL, respectively. Geometric mean concentrations in the next younger age range (6-11 years) were even lower. These data suggest that as these girls and young women mature, the mean blood lead concentrations in women of childbearing age will continue to decrease. Because this factor was not accounted for in the modeling presented in the HHRA, it again contributes to the over-estimates of blood lead concentrations that are likely to occur using the model default values.

Response>>

It is speculation that women of child bearing age would have decreasing blood lead levels as young women mature, based on the geometric means of these young women. The HHRA agrees that national data indicate decreases in blood lead levels in the past. However, if changes in lead exposure do not change from the group of women of child bearing age and the group of "young women", then it is only speculation that decreases in blood lead levels would be observed in the future. The HHRA used default blood lead levels in the Adult Model, although the average for the women of the Basin in 1996 was 2.0 ug/dl (default value = 1.7 ug/dl). Therefore, this would cause an underestimation of risk for the Basin residents. Please also see General Response to Comments, #11a.

Misc. Input>>

192	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS and TG
A62		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Combinations of exposure pathways and scenarios

The exposure and risk calculations include a baseline residential scenario as well as various additional incremental exposure sources (e.g., recreational exposures). As noted previously, the process of combining the baseline and incremental exposures did not include any adjustments to account for double-counting of exposure.

Response>>

The HHRA agrees with the comment. The baseline estimates included in the combined runs are not discounted for the time spent in the incremental behavior. This leads to an overestimation of risk, albeit it small for the current baseline situation. For a child recreational scenario, for example, there would be an unaccounted 5% decrease in time at the baseline residence. The difference in intake would depend on media concentration. Accounting for this reduction in baseline will be important in developing combined residential/recreational clean-up criteria. See also response to Comment A39 and General Response to Comments, #5.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
193	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A63		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

In addition, some of the combinations presented in the lead analyses appear to mix dissimilar exposure populations without adequately addressing the impacts of such calculations on the assessment results. In particular, the analyses for exposures to waste piles and neighborhood sediments are calculated for children between the ages of 4 and 11 years. The incremental intakes associated with these exposures are compared, however, to the baseline intakes calculated for 4-year-old children using the IEUBK model. Because the baseline intake of lead would be expected to decrease with age, the use of the baseline lead intake of 4-year-old children will over-estimate the baseline intake of children more than 4 years old.

Response>>

The HHRA disagrees with this comment. Four year old intake rates are provided as an example representing the mid-range of age-specific estimates. All IEUBK analysis is performed for age groups 1-7 years of age.

Misc. Input>>

194	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A64		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

In addition, application of the IEUBK model to assess potential lead exposures and blood lead concentrations in children older than the age range included in the model (i.e., 0-6 years) would also tend to over-estimate exposures and blood lead concentrations in the older age range.

Response>>

The HHRA agrees with the comment. IEUBK analysis are conducted for children aged 1-7 years. Risk calculations are pertinent to these age groups. Older children would likely have lower blood lead levels for the same intake rate.

Misc. Input>>

195	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A65		Hecla Mining and ASARCO	6-Section 6	Table 6-56b		Not Accepted

Comments>

In some cases, it also appears that the HHRA combines baseline exposures with incremental lead exposures for other activities calculated with RME input parameters (e.g., Table 6-56b). The use of RME parameters is inappropriate for this model, which requires the population's central tendency (geometric mean) and then uses the geometric standard deviation to estimate risks for the upper percentile of the population.

Response>>

The HHRA disagrees with this comment. Extreme responses in the population can be estimated by applying an appropriate GSD to the mean blood lead estimate from the IEUBK model, although this is difficult to interpret as noted. This technique requires that typical, or CT intake rates be input to the model for both the baseline and incremental exposure. The extreme response is estimated by applying the GSD reflect the biokinetic variation in the population and the variation inherent in the typical exposure. However, there are environmental extremes in the potential incremental exposures to consider in addition to the bio-kinetic response and typical baseline exposure factors. Some children, for example, may always play at the most contaminated beaches, rather than at the typical or average concentration. The RME intake estimates, used in the IEUBK estimates in the HHRA reflect CT ingestion rates for both the baseline and incremental exposure applied at 95th percentile contact concentration. See also General Response to Comments, #5.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
196	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A66		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Application of adult lead model

The HHRA uses EPA's interim guidance for modeling adult exposures to lead (U.S. EPA 1996). As with the modeling of children's exposures to lead, the analyses for adult exposures to lead presented in the HHRA focus primarily on EPA's default assumptions and do not incorporate site-specific data or considerations. When interpreting the results of the adult model, it should be considered that the model predictions have never been validated against empirical observations. While the individual default assumptions have varying degrees of technical support, the validity of the results yielded by the combined default model assumptions is highly uncertain.

Response>>

See General Response to Comments, #11.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
197	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A67		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Absorption fraction of lead from soil

EPA's default soil lead absorption factor (0.12) reflects two components. First, the absorption of soluble lead is assumed to be 20 percent (0.2). Second, the absorption of lead from soil is assumed to be 60 percent (0.6) of the absorption of soluble lead. By multiplying these two factors, an absolute absorption fraction for soil lead of 0.12 is derived. Based on model validation efforts for a physiologically-based model for adult lead uptake, other research has indicated that the mean absorption value for dietary lead sources may be closer to 8 percent (O'Flaherty 1993). Because lead in soil would be expected to be less well absorbed than dietary lead, these data suggest that the typical absorption fraction for lead in soil may be less than 8 percent (i.e., an absorption fraction of 0.08) and less than EPA's default assumption. Results from studies of lead bioavailability from soil using adult volunteers also suggest lower absorption than indicated by EPA's default estimate (Maddaloni et al. 1998). Taking into account the likely relative timing of incidental soil ingestion and consumption of meals, these studies resulted in mean estimates of absorption fraction that ranged from 3 to 14 percent depending on the number of meals assumed to be consumed per day and the assumed soil ingestion pattern.

Response>>

See General Response to Comments, #11 and #9b.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
198	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A68		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Geometric standard deviation

As discussed above for the IEUBK model, the GSD is intended to reflect individual variability in blood lead concentrations that might result from a specified degree of exposure. Because data regarding actual GSD values reflecting individual variability are limited for adult populations (as for children), GSD values based on community variability are typically applied to estimate individual variability. Because GSDs for community variability are greater than those reflecting individual variability, use of GSD values reflecting community variability will tend to over-estimate predicted blood lead concentrations at various percentiles for a specified set of exposure conditions.

Response>>

See General Response to Comments, #9c.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
199	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A69		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Timing of exposure

Uncertainty in the results of the adult lead model increases because of the use of the model under some of the exposure circumstances examined in the HHRA. The model is most appropriately applied to evaluate sites under an occupational scenario where it is assumed that adult workers have ongoing, frequent exposures to a relatively constant level of lead in soil. Such conditions would allow lead intake and resulting blood lead concentrations to reach a steady-state condition. Where exposures are infrequent or occur for only a short period of time, the degree of uncertainty in the model predictions increases.

Response>>

Infrequent exposures (i.e., less than 1 day per week) over a minimum duration of 90 days would be expected to produce oscillations in blood lead concentrations associated with the absorption and subsequent clearance of lead from the blood between each exposure event. The TRW recommends that the methodology should not be applied to scenarios in which the exposure factor is less than 1 day/week or less than 3 months in duration (TRW 1996).

The adult recreational scenario in the HHRA uses exposure frequencies equal to 1 full day/week for 15-32 weeks/year (or 105-224 days/year) (Table 6-31). Occupational exposure frequencies used are for 5 days/week for 8.7-39 weeks/year (or 61-273 days/year). The guidance for the Adult Exposures to Lead in Soil model states "...the TRW recommends that this methodology should not be applied to scenarios in which EFs is less than 1 day/week." The adult recreational and occupational RME scenarios used in the HHRA are consistent with guidelines recommended by the TRW and uncertainties associated with the model are discussed in Section 7.0 and more specifically in Section 7.4.4. The occupational CT scenario is less than 3 months in duration and, therefore, will contain more uncertainty. Please see General Response to Comments, #11.

Misc. Input>>

200	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A70		Hecla Mining and ASARCO	6-Section 6	p. 6-41 and 6-43		Not Accepted

Comments>

Characterization of modeling results

As previously discussed above, the default model performs particularly poorly in comparison with the empirical data. The evaluation of IEUBK default- and Box-model results (beginning on page 6-41) concludes that the EPA default model consistently overpredicts and the Box model underpredicts the percent of children likely to exceed the 10 µg/dL criteria relative to the empirical data. Whether the Box model overpredicts the observed data, however, depends on the geographic subarea. The default model overpredicts in all areas except Kingston and the Lower Basin. In the Upper Basin, the Box model overpredicts blood lead levels for some subareas and is generally close for the others. The default model highly overpredicts blood lead levels in the Upper Basin. Interestingly, this area has the most paint exposure as well. Without the paint exposure, the empirical data would be even lower than observed, further signifying that both models overpredict exposure.

The poor match in the Lower Basin with the observed data is likely due to the factors mentioned above regarding the sample size in the lower Basin. Also as noted on page 6 43 for the Lower Basin, “A small number of children are exhibiting much greater blood lead levels than expected under any scenario from the Baseline intakes.” These results therefore indicate both models and particularly the default model are not useful for setting remedial goals to address the elevated blood lead levels observed.

Response>>

The HHRA disagrees with this comment. Both the Box Model and the EPA Default Model use observed soil and dust lead levels. Paint contributions are inherent in these input values. Without paint exposure, dust lead levels would be lowered as would predicted blood lead levels. Sample size may be a factor in the Lower Basin, however, baseline blood lead predictions are low because residential soil and dust lead concentrations are low. Both the exposure assessment and follow-up reports of children experiencing high blood lead levels in the Lower Basin identify extended recreational exposures away from the home as probable sources. The baseline IEUBK analysis indicates that residential exposure reductions are called for where soil and dust lead levels are high, and are not necessary where levels are low.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
201	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	TG
A71		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Other modeling options

Other options for modeling lead exposure exist and should be considered in conducting the analyses for the Basin. In particular, the Integrated Stochastic Exposure (ISE) Model for Lead (Griffin et al. 1999) is similar to the IEUBK model, except that it uses probability density functions rather than point estimates as inputs for most concentrations and exposure parameters. The distributions are combined using Monte Carlo probabilistic techniques to predict a distribution of absorbed doses for different members of the exposed population. The biokinetic portion of the IEUBK model is then used to generate the geometric mean and predicted distribution of blood lead levels rather than applying a single point estimate for the GSD. Unlike the IEUBK model, soil lead concentrations well over 1,000 ppm are required for the predicted greater than 5 percent risk of exceeding a 10 µg/dL blood lead level.

Response>>

The HHRA disagrees with this comment. The IEUBK has been extensively reviewed by the EPA, including reviews by the Science Advisory Board (SAB) in 1991-1992, and subsequent guidance reflecting these reviews was issued approving the IEUBK for sub-chronic risk assessment for lead in children. These guidance documents are provided in Appendix O. None of the other bio-kinetic simulation models suggested by reviewers have been similarly reviewed, nor has any guidance been issued regarding use of these alternate techniques. EPA guidance does recognize site-specific empirical modeling of blood lead levels and dose-response as a useful tool to supplement IEUBK analysis. That analysis was accomplished in the HHRA and is discussed in General Response to Comments, #3, #4, and #9a through #9d.

Misc. Input>>

202	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A72		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Use of chronic RfD to assess childhood risks to arsenic

The chronic RfD for arsenic ingestion is overly conservative to assess childhood risks associated with arsenic ingestion. The chronic RfD is based on a no-effect level in populations exposed to arsenic for most of their lifetimes. Whether the safe dose for children and adults is the same or different depends on whether children are uniquely more sensitive to arsenic than adults.

Reviews of the available studies by EPA Region VIII (Benson 1995) and Exponent (Tsuji et al. 2000) have not found children to be more sensitive than adults except at high doses when acute poisoning occurs. Lower exposure to arsenic appears to delay the onset of health effects such that, at the lowest doses, those showing health effects in populations are not young children, but older individuals. Although few studies have quantified health effects at low doses in very young children, the available studies for children up to 9 to 15 years of age indicates that a no-effect level dose and the RfD for childhood exposure would be higher than that for chronic lifetime exposure. EPA Region VIII (Benson 1995) proposed a subchronic reference dose for children up to 15 years old of 0.006 mg/kg/day, which is 20 times higher than the chronic RfD for arsenic. This subchronic RfD has been used by EPA Region VIII to assess short-term risks to children (ISSI 2000). The subchronic RfD is currently undergoing national review and may be increased from this level depending on the uncertainty factor that is ultimately applied to the no-effect level.

Response>>

EPA Region 10 is awaiting the outcome of the national peer review prior to making any quantitative changes to risk calculations. See also response to Comment A20.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
203	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A73		Hecla Mining and ASARCO	0-Executive Summary	p.4-6		Partially Accepted

Comments>

Observations of arsenic health effects in U.S. populations

The HHRA fails to adequately acknowledge U.S. data regarding arsenic health risks. U.S. studies have shown few of the health effects associated with arsenic that have been observed in studies of overseas populations. The Utah study cited on page 4-6 of the HHRA (Lewis et al. 1999) found very few significant increases in diseases. The few significant effects found were not among the most noted toxic effects of arsenic and no dose-response relationship was found, which calls into question whether arsenic was the cause. The most prevalent endpoints for arsenic toxicity consistently noted in other studies from foreign countries (i.e., cancers of the skin, lung, and bladder, and effects on the skin) were not found in the Utah population. Because of the large number of disease endpoints examined, the results are also vulnerable to multicomparison errors in which significant associations may result by chance. Prostate cancer, which is common in older men, may also be more prevalent in this Mormon population because of lack of early deaths due to other common diseases associated with smoking and drinking.

Response>>

In the absence of more agreement in the scientific community on these issues, the HHRA takes a health-protective approach. We direct the reviewers attention to papers by Smith and coworkers at Berkeley which include discussions that address the problems with evaluating arsenic health risks in the United States. These researchers have been heavily involved in the epidemiology and biostatistics of population studies of arsenic exposure and mortality/morbidity. The newer citation of Smith et al. that has discussions relevant to this issue are:

Ref: Smith AH, Goycolea M, Haque R, Biggs ML. Marked increase in bladder and lung cancer mortality in a region of northern Chile due to arsenic in drinking water. Am. J. Epidemiol. 147: 660-669 (1998).

See also Dr. Mushak's comments on this issue. We agree with reviewers comments regarding the Lewis study. However, the Lewis data was highly biased against applicability to the general U.S. population. Unlike the Utah study cohort, the U.S. population is largely not Mormon, not as non-smoking, not as non-drinking, not as healthy, in terms of SES and associated health risk factors, potentially all risk factors that affect the expression of adverse effects of contaminants.

An expanded discussion will be added to the uncertainty section on these points.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
204	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A74		Hecla Mining and ASARCO	4-Section 4	p. 4-7		Not Accepted

Comments>

Risks associated with low dose levels

Arsenic risks at low doses are likely over-estimated by the HHRA. NRC's (1999) conclusion that the arsenic cancer risk at the maximum contaminant level(MCL) in water could be 1 in 100 (see HHRA page 4-7) was not accepted by all authors of this panel. This calculation is very controversial and assumes that arsenic risk can be extrapolated from high doses to low doses below which no effects have been observed. This estimate of 1 in 100 does not consider the findings of EPA's expert review panel on the mechanisms of arsenic carcinogenicity. The findings of this panel were that all possible mechanisms for arsenic carcinogenicity would have a non-linear dose-response relationship (Eastern Research Group 1995). Thus, cancer risks at low doses may well be lower than predicted based on the model used by NRC (1999).

Response>>

We acknowledge that there is disagreement in the scientific community regarding these issues. In the absence of agreement, we chose a health-protective approach. See also response to Comment A21.

We considered the NRC 1999 report as more authoritative on matters of arsenic cancer models than the EPA meeting four years earlier cited by the commenters because the NRC reviewed additional scientific information and all parts of the NRC document underwent peer review. The NRC report considered it prudent not to reject linear low-dose extrapolations for cancer risks from low arsenic intakes. The NRC report made it clear that it considered the nature of the low-dose relationship to be driven by the mechanism of carcinogenic action of arsenic. Since the NRC report appeared, additional data have appeared showing that a linear model at low dose would in fact be reasonable. Mass and coworkers, in work described in an SOT abstract, show that direct interaction of arsenic as the trivalent monomethyl metabolite with DNA was seen in tandem with various measures of DNA damage. Damage included: unwinding (nicking) of DNA and production of double-stranded breaks, and/or induction of alkaline labile sites at levels well below inorganic arsenic levels. A number of other measures of damage were positive. These results show methyl-arsenic (III) being genotoxic via DNA interaction.

Ref: Mass MJ, Tennant A, Roop B, Kundu B, Brock K, Kligerman A, DeMarini D, Wang C, Cullen W, Thomas D, Styblo M. Methylated arsenic (III) species react directly with DNA and are potential proximate or ultimate genotoxic forms of arsenic. The Toxicologist (2001, in press): Proc. Soc Toxicol 40th Annual Meeting, San Francisco, CA, March 25-29, 2001.

Misc. Input>>

205	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A75		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Risk calculations

The documentation of detailed calculations performed to obtain the numerical risk estimates for non-lead metals presented in the document is generally inadequate to perform a thorough review. The specific sets of assumptions and combinations of exposure pathways and scenarios that were used to derive risk estimates are difficult to identify based on the information presented in the HHRA. A more detailed "road map" of the calculations performed to support the risk assessment should be provided in the documentation.

Response>>

We are uncertain as to what additional information the reviewers suggest should be provided. Section 3 discusses in depth the processes and equations used to calculate chemical intakes and provides tables of all input parameters used. Section 4, Table 4-1, provides the toxicity criteria used in the risk calculations and Section 5 discusses in depth the process and equations used to calculate risks. Appendix A contains EPA's RAGS Part D tables which walk someone through all steps to each calculation completed in the HHRA. Appendices E and F provide the raw data and summary statistics that were used to calculate the exposure point concentrations (EPCs).

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
206	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A76		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Risk characterization results

The results of the risk calculations for the non-lead chemicals provide a misleading perspective on the degree of risk associated with exposures to these COPCs at this site. As noted above, the conservative nature of many of the individual exposure assumptions and the combinations of exposure pathways and scenarios yield highly conservative estimates of the potential risks associated with the site.

Response>>

As noted in earlier responses, RME risk estimates are intended to over-estimate risks, i.e., be a "reasonable maximum" in order to be health protective. The Central Tendency (CT) risks are intended to represent a more average situation and CT risks are also presented and discussed in Section 5. Text will be added to expand the CT results discussion. See previous comments on this issue, A39 and A36.

Misc. Input>>

207	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A77		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

The total hazard index for all metals over-estimates non-cancer risk because the effects of these metals are not likely to be additive. Non-cancer hazard quotients added over pathways and constituents are not likely to be additive. In fact, several of these metals are likely to compete for absorption in the gastrointestinal tract thereby reducing their combined toxicity. For example, iron and zinc are well known to antagonize the absorption and effects of other metals.

Response>>

We agree that iron and zinc are well known to antagonize the effects of other metals and this issue is discussed specifically in the HHRA on pages 5-4, 7-20, and 7-21. We acknowledge that the focus of subsequent discussions in Section 5 stills assumes additivity. However, some individual effects from COPC metals may be additive. Additivity or its rejection requires knowledge of the mechanisms of toxic action for these Basin contaminants. Toxic action mechanisms have not been fully characterized for all COPCs; thus knowledge to reject inter-organ or inter-tissue toxic interactions is not currently available. However, we will add more discussion to Section 5 concerning what the hazards would be if additivity is not assumed..

Misc. Input>>

208	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A78		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Non-cancer and cancer risks for soil are highest for the child scenario because of the conservative approach of combining the chronic RfD for arsenic ingestion with high childhood doses. As noted above, an RfD that is more appropriate for assessing childhood non-cancer risks should be used rather than the chronic reference dose. Non-cancer hazards of arsenic for chronic 30-year exposure duration are a low concern.

Response>>

See previous response to Comment A20.

Misc. Input>>

209	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A79		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Subsistence risks are greatly over-estimated. As described above, subsistence populations are assumed to eat a high amount of soil and sediment, to be completely covered over their whole body with a coating of sediment for seven months of the year and an additional coating of soil on exposed parts of the body for 365 days per year, and to drink 150 percent of the RME water ingestion rate from the river. It is not surprising that such assumptions led to dermal absorption, soil/sediment ingestion, and water ingestion being among the exposure pathways that contributed the most to a total cancer risk for this scenario of 4 x 10-3. This scenario combines so many worst-case and improbable assumptions, however, that the calculated risks have little relevance for risk management decisions.

Response>>

See response to Comment A37 and General Response to Comments, #6.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
210	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A80		Hecla Mining and ASARCO	0-Executive Summary			Accepted

Comments>

Presentation of results

Given the number of individual risk calculations and combinations of calculations provided in the HHRA, clear and consistent presentation of the risk calculation results is essential to aid review and synthesis of the results. In any risk characterization, it is important to present the numerical results; provide benchmarks for interpreting the results (e.g., target risk levels); identify primary chemicals, exposure pathways, and scenarios contributing to total exposures; and discuss major factors influencing the uncertainty of risk assessment results. While these issues were generally addressed in the risk characterization and summary sections of the HHRA, they were not always addressed in a consistent way. In particular, in the summary section of the report, the discussion of the cancer risk assessment results clearly identified those risk estimates that exceeded EPA's acceptable risk range. For risk estimates that did not exceed the range, however, the comparison with the risk range was not always discussed. Instead, the summary of these pathways frequently focused on those subcomponents of the total risk estimate that were associated with the highest risk or that were the primary contributors to risks. This approach gives the misleading impression that all of the exposure pathways and scenarios were associated with elevated and unacceptable risk levels.

Response>>

Comment noted. This section will be revised as appropriate to provide more consistency.

Misc. Input>>

211	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		0/30/2000	URS
A81		Hecla Mining and ASARCO	0-Executive Summary			Accepted

Comments>

Similarly, the summary of the non-cancer risk assessment results highlighted potentially unacceptable risk estimates in a misleading way. Specifically, the presentation of the hazard indices began with a review of the results obtained by combining the hazard quotients for all COPCs, regardless of health endpoint of concern. Such an approach is only to be used as an initial screening analysis and should have been clearly identified as such at the beginning of the discussion of these results. Only after an extensive discussion of the total hazard indices, the HHRA presented a limited discussion of the technical deficiencies in such an approach, particularly for the risk analyses for the Basin where there is limited overlap in health endpoints among the COPCs. This subsequent discussion then noted briefly that some of the hazard indices identified as exceeding the target level in the initial analyses would not, in fact, exceed the target when the more technically appropriate approach was used.

Response>>

Comment noted, see response to Comment A77. We will provide additional discussion regarding the hazard results if additivity is not assumed.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
313	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		1/02/2000	TG
A82		Hecla Mining and ASARCO	2-Section 2	p.2-7		Partially Accepted

Comments>

Soil samples were also sieved to particle sizes <175 µm prior to sampling which, as noted on page 2-7, is a smaller size fraction than the fraction sampled for the calibration of EPA's IEUBK model for lead. This would tend to bias the sample concentration result upward with respect to the size fraction that is typically incorporated into IEUBK modeling. This size fraction (<175 µm) is also not the standard for assessing bioavailability of metals in soil (I.e., <250 µm, Ruby et al. 1999).

Response>>

The HHRA agrees with this comment. The 175 micron mesh sieve technique was adopted in 1974 for the original lead health studies conducted in the area and has been used for all residential soil samples collected in the Basin RI/FS and all previous health and exposure studies. The procedure was developed to reflect the range of soil particle sizes that most likely adhere to children's hands which are then transferred by hand-to-mouth activities. Subsequent research has continued to show that this size-range is applicable. The selection of this standard pre-dates either recommendation from federal agencies, and the State Department of Health and Welfare has elected to maintain consistent soil and dust measurement techniques throughout the course of these investigations. The EPA has concurred in that determination. Assuming any concentration effect due to sieving is proportional, the use of a lower value (as suggested might occur with EPA's larger sieve size) would result in an increased dose response coefficient in the site-specific analysis. That is, the per unit effect of soil or dust lead concentration on blood lead levels would be greater. This would be interpreted as indicating higher bioavailability of soil and dust or lesser intake is occurring in the population. See also General Response to Comments, #3c and #9b.

Misc. Input>>

314	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		1/02/2000	URS
A83		Hecla Mining and ASARCO	2-Section 2	p.2-10		Not Accepted

Comments>

Assessment of surface water exposures

Use of data from the surface water samples that included artificially elevated sediment concentrations over-estimates typical recreational exposures. Sediment was deliberately kicked prior to surface water sampling to simulate disturbed conditions, and surface water samples contained "large amounts of suspended sediment" (page 2-10). The HHRA assumes regular ingestion of 30 ml of this water a day for the warmer months of the year. Ingestion of such water may occasionally occur when young children play near the shore but would not likely occur on a regular basis and would be very unlikely for older children and adults. The HHRA, however, assumes that all ages may ingest this water regularly (up to 70 years for the traditional subsistence scenario). For developing ambient water quality criteria. EPA (1998) assumes a water ingestion rate during swimming of 30 ml/hr or 10 ml/day. One would expect that wading in muddy water would result in less gulping of water than swimming. In addition, sediment in water is less likely to cling to hands than soil would. Therefore, hand-to-mouth activity by children is not likely to result in the same intake rate as assumed for swimming.

Response>>

The HHRA assumed that adults and children using recreational areas in the Lower Basin could swallow water containing suspended sediments during the warmer months (public receptors and the special subgroup of subsistence receptors). For other areas of the Basin, adults were not assumed to be exposed to surface water, only children aged 4 to 11. For public receptors, the assumption used was that for 32 days of the year 30 ml of water was swallowed. This represents a couple of mouthfuls (about one ounce of water) that could certainly be ingested during swimming/water play activities. While it may be an overestimate we do not consider the overestimate to be unreasonable.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
315	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		1/02/2000	URS
A84		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Waste pile exposures

Exposure to waste piles appears limited. The section under Upland Soil EPCs (page 3-39) notes that, in many cases, waste piles are not adjacent to residential homes (as in the case of the Nine Mile and Mullan areas) and sieved surface soil samples were not available because of lack of fines in the upper one inch due to the rocky nature of the piles. Nevertheless, the available waste pile data that could be collected are assumed to be representative of this type of exposure throughout the Basin. Given these facts, frequent visits and ingestion of large amounts of soil on waste piles (i.e., the 300 mg/day that is assumed, a value that is more than the residential RME soil ingestion rate) are not realistic assumptions. As a result, the waste pile risk calculations presented in the HHRA over-estimate the likely exposures and risks from waste piles and should be modified to reflect more realistic exposure assumptions.

Response>>

We agree that waste pile exposures are likely only an issue for elementary-aged school children where the piles are relatively close to residences and only these types of piles were evaluated in two areas of the HHRA. In addition, we agree that waste pile concentrations might be variable and these two things, location and concentration, would need to be taken into consideration for any risk management decisions regarding potential remedial actions at a waste pile. Many waste piles that are accessible on the valley floor and some of the side canyons do receive heavy use by teenagers and adults, although the pile may not be easily accessible by younger children. Therefore, many of the more remote piles do provide exposure and will be evaluated during the remedial design phase although the risk assessment did not quantitatively evaluate the older children/adult pathway. Text will be added to clarify this issue. See also response to Comment A24.

Misc. Input>>

316	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		1/03/2000	TG
A53		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

Source identification

To understand the contributions of mining sources to elevated blood lead concentrations, blood lead statistics should ideally be recalculated excluding the data for children whose blood levels are affected by non-mining sources. For example, children with known paint exposures should be removed from the database for comparison with IEUBK model results. Because of the bias introduced by paint, which is unrelated to lead in soil from mining activities, and the inability of the model to adequately account for this source of lead separate from other sources, children and houses with known lead-based paint exposures should be removed from the database.

Response>>

The HHRA disagrees with this comment. The HHRA evaluates the potential human health risks associated with contaminated environmental media. With regard to lead, the analysis examines the effects of soil and dust lead on blood lead levels in concert with dietary and other sources. There is little indication of direct ingestion of paint particulate aside from that lead paint incorporated in soil and dust pathway. The site-specific model analysis uses observed soil and house dust lead levels. As a result, the sources of lead to dust, such as paint, yard soils, materials tracked in by workers, fugitive dusts, etc. are inherent in the analysis. The influence of lead paint on these pathways is examined by regression analysis. The interpretation of these results was that contaminated soils, house dust, and lead based paint are all related to excess absorption. Separating out the homes with known paint hazard would be difficult as most residences, other than trailer homes, have lead paint. The primary indicator of paint condition (peeling/chipping/chalking paint) has been shown in the parent 1996 Basin Exposure Study to be highly correlated with home hygiene and socio-economic status. As a result, it is not clear whether the significance of this variable is reflective of the paint source of lead, socio-economic status, personal and family behavior, home hygiene practices, or dust loading. See also General Response to Comments, #1a.

Misc. Input>>

317	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		1/03/2000	TG
A54		Hecla Mining and ASARCO	0-Executive Summary			Accepted

Comments>

Similarly, if children's exposure is thought to arise due to exposures from areas other than their own property (as noted in case follow-up records), these children should be removed from the database if comparisons are being made to IEUBK model results for residential yards.

Response>>

The HHRA agrees with this comment. A small number of children that have been identified as having exposures outside the home were included in the analysis. Appropriate discussion will be included in the final document.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
320	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		1/03/2000	TG
A85		Hecla Mining and ASARCO	0-Executive Summary			Partially Accepted

Comments>

Evaluations of potential sources should also consider that blood lead concentrations for females of reproductive age and their children may be affected by exposure from the BHSS. Blood lead concentrations and the body burden of lead of females in the Basin could be affected by past exposures to greatly elevated levels of lead in the BHSS. This body burden would also contribute to prenatal exposure to their children. Older children who have accumulated lead from past exposures in the BHSS may also have moved to the Basin. No attempt appears to have been made to account for this possible source of elevated lead exposure in the Basin.

Response>>

The HHRA agrees with this comment. Reproductive-aged female blood lead levels are greater than national norms as indicated in the HHRA. The observed geometric mean female adult blood lead level was 2.0 ug/dl and ranged from 1.6-2.6 ug/dl in the 8 geographic areas. A national default value of 1.7 ug/dl was used in the risk estimates, as the total number of samples from each area was not of sufficient size (n=12-41, see Table 6-8b) to yield statistically meaningful estimates. Similarly, national default estimates were used for estimated maternal contribution to infant blood lead levels. Use of a higher maternal contribution would result in slightly greater blood lead estimates for young children. Risk managers may want to consider risk underestimated for these individuals. Little data are available to evaluate whether the elevations noted in Basin women are due to the BHSS, Basin-wide contaminant sources, occupational exposures, or other factors.

Misc. Input>>

321	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		1/03/2000	TG
A86		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

F. Application of the IEUBK model

As discussed above in the general comments section, the IEUBK calculations presented in the HHRA are based primarily on EPA's default exposure assumptions. Little effort is made in the HHRA to incorporate site-specific data or considerations in the modeling analyses, to refine the modeling approach to reflect site-specific information or to interpret the modeling results in light of actual observations. In particular, the HHRA modeling does not attempt to address the likely variation that exists among the identified exposure areas in factors that are likely to affect the IEUBK modeling results.

Response>>

The HHRA disagrees with this comment. Site-specific data were used for input into the IEUBK. Please see General Response to Comments, #3, #4, #8 and #9.

Misc. Input>>

322	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		1/03/2000	TG
A87		Hecla Mining and ASARCO	0-Executive Summary			Not Accepted

Comments>

In addition, in several cases, the default assumptions reflect highly conservative or outdated scientific information that also calls into question the validity of the model results.

Response>>

The HHRA disagrees with this comment. The default assumptions used in the analysis are recommended by current EPA guidance, and represent scientific consensus based on rigorous examination of national and international experience. The applicability of the various assumptions was extensively reviewed in the HHRA process and the applicability of these assumptions to the Basin and associated uncertainties are discussed in the document.

Misc. Input>>

323	10/13/2000	Hecla Mining and ASARCO	Public Draft - July 2000		1/03/2000	TG
A88		Hecla Mining and ASARCO	0-Executive Summary			Accepted

Comments>

Appropriateness of input assumptions

The various input assumptions and data used in the model runs should be more clearly presented in the report. Even if many default assumptions are used, these should be listed in a summary table. As presented, the assumptions used for the various model runs are difficult to reconstruct. Specific comments on input assumptions are discussed below.

Response>>

The HHRA agrees with this comment. All the assumptions are included in various Tables throughout the document. A summary table will be added to the final document to consolidate the presentation.

Misc. Input>>

Local Governments

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
212	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B1		Local Governments	0-Executive Summary			Not Accepted

Comments>

Conceptual Site Models (CSMs) appear to be based on ecological concerns and not human health issues.

Response>>

The fate and transport of mining materials does not differ between ecological and human receptors. The applicable human receptor pathways are included on the CSM figures in Section 3.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
213	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B2		Local Governments	0-Executive Summary			Partially Accepted

Comments>

The CSMs in the Report are very different than in Field Sampling and Analysis Plan (FSAP) Addendum 05, which was used to plan data collection.

Response>>

The CSMs in the HHRA contain much more detail regarding release mechanisms and also included other receptor groups than just "recreational." The CSM in FSP05 focused only on recreational receptors and contained no detail under "mechanisms." While FSP05 listed several different types of recreational activities that could be practiced partly because both lower and upper basin areas were sampled, the HHRA did not quantify all possible recreational activities, but selected exposure parameters protective of all recreational activities within a given geographical region and only quantified one risk for recreational activities per receptor group per area. For example, only one risk/hazard for each COPC was calculated for public recreational risks in the Lower Basin. The risk/hazard estimate reflects all types of recreational activities by the public in this area assuming equal exposure time to the different media and that any of the individual recreational areas are as likely to be used as any other. Therefore, only one recreational receptor "box" was included for each of the HHRA area-specific CSMs. Text will be added to clarify this issue.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
214	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B3		Local Governments	0-Executive Summary			Not Accepted

Comments>

It is not clear that data collection meets the needs for the CSMs in the Report.

Response>>

The human exposure routes identified on the CSMs and quantitatively evaluated in the report had sufficient data to calculate risks. In a few cases data that was collected for ecological concerns was used in the report and the implications of using it were discussed in Section 7 of the HHRA.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
215	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B4		Local Governments	0-Executive Summary			Partially Accepted

Comments>

Data from upland soil, collected at a depth of 0 to 1-inch and sediment collected at 0 to 6-inch and 0 to 12-inch depths were combined in the Report to evaluate potential risk to one receptor, a 4 to 11 year-old child. The data were collected to estimate exposure to campers (0 to 1-inch) and swimming and wading children (sediment). These data were collected for different uses and were not intended to be combined. A statistical analysis of the Blackwell Island data (the only area for which sufficient data exists to make the analysis) shows that the data cannot be combined (they are independent data sets and do not have sufficient statistical similarities to justify combining. Combining these data sets is similar to combining the weights of apples and oranges, there are too many differences to make the results meaningful. The review identified many similar data management situations.

Response>>

We acknowledge that upland soil and beach sediment data may be statistically different for some chemicals for some sites. However, while this may be an important issue for nature and extent issues, any differences that may exist in sample means is not relevant in this HHRA because of the exposure assumptions used in calculating risk. Therefore, the soil and sediment data were appropriately combined for the Lower Basin neighborhood and public receptors for the following reasons: 1) The "upland" areas and the "beach" areas of the Lower Basin are in close proximity to one another. 2) The "upland" areas have all been impacted by previous flood events and therefore experienced a mixing of soil and sediment materials. For this reason, this material was identified as "floodplain soil /sediment" and refers to materials within the approximately 1-mile wide flood plain area. 3) Lastly, a receptor is presumed to spend an equal amount of time in upland areas as in beach areas. It is also assumed that receptors will have an equal probability of visiting one CUA in the Lower Basin as another. Therefore, the data was appropriately combined for the Lower Basin neighborhood and public receptors and an average concentration across the entire Lower Basin is an appropriate use of the data.

See Appendix E where the details including sample number, depth, and media, are provided for all recreational data (except Blackwell Island which was inadvertently not included--it will be added), and Appendix F which contains all data used in the calculations.

With respect to Blackwell Island, however, we acknowledge that soil and sediment data are not statistically the same and because Blackwell Island's upland and beach areas are spatially separated (unlike in the Lower Basin), the assumption of equal time spent between the upland and beach areas may not be as valid in this instance. Therefore, separate risk evaluations may have been more appropriate. An evaluation of this data shows that the higher sediment concentrations are driving the risks and hazards for Blackwell Island. This will be taken into consideration during risk management decisions.

Misc. Input>>

216	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B5		Local Governments	0-Executive Summary			Not Accepted

Comments>

Evaluation of soil/sediment data for the Lower Basin identified significant variability over the entire Lower Basin and within small areas of the Lower Basin. It is not appropriate to evaluate a large area (make risk-based decisions) when there is highly variable data from even small portions of the entire area.

Response>>

The risk estimates for Lower Basin use assume any of the Lower Basin areas are as likely to be used as any other; therefore, combining the data is appropriate. Risk management decisions regarding Lower Basin remediation will be made on a site-by-site basis (i.e., by common use area, not for the entire Lower Basin as a whole) as described in the Technical Memorandum for Human Health Alternatives. Risk management decisions for non-lead chemicals are not addressed in the document. See also response to Comment B4.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
217	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B6		Local Governments	0-Executive Summary			Not Accepted

Comments>

When a screening-level risk assessment suggests a potential risk, for example Blackwell Island, EPA guidance states that exposure point concentrations and exposure parameters should be modified for the Baseline Risk Assessment. The modifications may include additional data collection or modifying exposure parameters to be more representative of actual land use. Exposure parameters in the Baseline Risk Assessment are identical to those in the Screening Level Risk Assessment and additional data have not been collected for Blackwell Island or Harrison Beach. Although Harrison Beach was identified for further evaluation, it is not evaluated in the Report. The assumption that the quantity and quality of data collected for Screening Level Risk Assessment is appropriate for Baseline Risk Assessment requires discussion in the Report. The acceptable uncertainties in screening level evaluations are much greater than those for a Baseline Risk Assessment.

Response>>

Sites in the screening level risk assessments were screened using a hazard quotient of 0.1 rather than 1 which added a level of protectiveness to the screening level document that is not incorporated into the baseline risk assessment. After review by the HHRA stakeholder team, it was decided that the exposure assumptions regarding frequency of site use used in the screening-level document were appropriate for the RME exposure scenario in the baseline RA for Blackwell Island. In addition to the RME scenario, a CT scenario was added in the Baseline RA.

Harrison Beach was evaluated in the Baseline RA as part of the Lower Basin and is first specifically noted as included in Section 2.1.3. Also the title of the HHRA is "...from Harrison to Mullan..."

Over 400 samples of floodplain soil and sediment were collected from 33 Lower Basin recreational areas using a randomized sampling scheme within each area (approximately 15-20 miles of river). This quantity of data is considered sufficient to evaluate recreational use in this area. Data quality (laboratory analysis) met all requirements for Baseline RA's.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
218	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B7		Local Governments	0-Executive Summary			Not Accepted

Comments>

Evaluating the child-only exposure scenario to determine noncarcinogenic risk is “overly conservative”, based on EPA’s Science Advisory Board’s (SAB) evaluation. SAB indicates that an adult/child scenario is sufficiently conservative and is recommended to determine hazard quotients.

Response>>

We disagree with this comment. The White House issued a policy statement on April 27th, 1997 regarding health risks to children which states "It is the policy of the USEPA to consider the risks to infants and children consistently and explicitly as part of risk assessments...the Agency will develop a separate assessment of risks to infants and children...".

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
219	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B8		Local Governments	0-Executive Summary			Not Accepted

Comments>

Statistical approaches used to determine the number of samples required to estimate exposure point concentrations are different in the Report and the Field Sampling and Analysis Plan (FSAP). The FSAP indicated that seven samples were adequate to support human health risk assessment, but the Report states that ten samples are required. This is confusing, because the FSAP was designed to collect samples to support risk assessment. An explanation is required in the Report.

Response>>

An explanation of the reasons for 7 versus 10 samples is provided in Section 3.3.1, in the last paragraph on page 3-35.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
220	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B9		Local Governments	0-Executive Summary			Not Accepted

Comments>

The report calculates correlation coefficients to a high confidence level and concludes that the variables are “significantly correlated”. Few correlation coefficients were greater than 0.5. Actually, correlation coefficients below 0.5 are poor indicators of data predictability (predicting or explaining one value in terms of the other is the goal of correlation coefficients). A correlation coefficient less than 0.5 means that at least fifty percent of the variability in the data cannot be accounted for in the analysis. This is not a “significant” correlation.

Response>>

The HHRA disagrees with this comment. Correlations that were considered significant were based on that correlation's p-value. A correlation was considered significant if the p-value was 0.05 or less. There is also confusion as to the difference between an r and an R-squared. R-squared values used for regression analyses explain variation in the dependent variable, not an r. Numerous articles from scientific journals on environmental epidemiology of environmental contaminants explain that there is no "magic number" which an association has to reach or exceed in order to offer interpretive value. Secondly, it is not the case that a "coefficient" less than 0.5 is not indicative of a "significant" association. Whether some value is or is not hinges not only on the level of statistical significance but also on the particular statistical design or statistical model being used. In the typical practice in epidemiological studies with complex biostatistical components, even very good associations in population studies can be less than "0.5", especially if the particular association being tested has been over-controlled for confounders that subsume within their controlling an environmental lead component. Please see General Response to Comments, #3d.

Misc. Input>>

221	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B10		Local Governments	0-Executive Summary			Partially Accepted

Comments>

There are 83 and 74 matched data sets for soil, floor mats, and house dust, but it is stated there are insufficient data to statistically analyze. This is difficult to understand, based on the characteristics of statistical test parameters. The Report should provide the analysis and identify the potential correlation for the data sets, similar to that done in Section 6.4.1.

Response>>

The text states that there is insufficient data for each geographic subregion for statistical analysis and that there is not paired soil-dust data for every home for the non-lead metals. For six of the eight geographical areas, less than 10 house dust samples were available, too few for statistical analysis given the large variability of concentrations in dust (explained on page 3-38 and in detail on page 7-14). The primary reason the data were not used in the risk and hazard calculations was because of the uncertainty of the relationship between soil and dust, making a quantitative prediction of dust concentrations where we did not have data highly uncertain. The uncertainty in predicting dust concentrations from soil concentrations was considered more problematic than just using the soil data. In addition, while the soil-dust relationships for lead are reasonably well characterized, the soil-dust relationship for non lead contaminants is not. The majority of risk assessments to date do not have indoor dust concentrations for chemicals other than lead, thus using the soil data as a surrogate has precedence throughout the country. Paired soil and dust data for lead were available for every home, so the lead risk assessment did not have to predict a relationship in the absence of data. Additional text will be added to Section 3 to clarify these issues.

The assumptions and reasons for not using the house dust data are discussed on pages 3-38 to 3-39 and further discussed in Section 7, page 7-14, table 7-1. Statistical correlations are provided in Appendix I.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
222	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B11		Local Governments	0-Executive Summary			Partially Accepted

Comments>

The sampling strategy used to collect waste pile samples does not appear to be appropriate to support risk assessment, based on EPA guidance. The samples appear to be from a biased or judgmental sampling strategy and were collected to characterize the waste piles. They do not appear to have been collected to estimate potential exposure (random sampling).

Response>>

The sampling strategy was appropriate per page 6-28 of EPA, 1989 RAGS Part A: "In some cases, contamination may be unevenly distributed across a site, resulting in hot spots (areas of high contamination relative to other areas of the site). If a hot spot is located near an area which, because of site or population characteristics, is visited or used more frequently, exposure to the hot spot should be addressed separately."

Waste piles close to residential homes were sampled along Canyon and Ninemile Creeks and in Mullan specifically to estimate exposure. Children were observed playing on these piles or were reported as having played on them; therefore, piles were sampled "purposively". Discussion will be added to Section 2 to clarify waste pile sampling and use.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
223	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B12		Local Governments	0-Executive Summary			Not Accepted

Comments>

In addition, it is stated that the 0 to 1 inch depth was not be sampled because sufficient fine material was not present at this depth. However, the fine material in the 0 to 1-inch depth interval is important for risk assessment, because children are exposed to this interval.

Response>>

Fine material is present in the top inch and this material would stick to children's hands and be ingested; however an insufficient amount was present for laboratory analysis. The assumption is that the concentration found in the 0-6 inch depth is representative of the concentration in the top inch.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
224	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B13		Local Governments	0-Executive Summary			Not Accepted

Comments>

Exposure areas are not physically defined. For example, a residential yard is generally assumed to be ¼ acre. The Report infers that the Lower Basin risk levels apply to the entire Lower Basin area. The estimated risk actually applies to the small individual areas adjacent to the Coeur d'Alene River that were sampled or to the four residences that were sampled. When the exposure area is not defined, it is difficult to identify 1) the area that requires remedial action and 2) whether sufficient data were collected to support the decision. Defining exposure area is a critical part of data collection and risk assessment, but the Report does not perform this important task.

Response>>

The CdA Basin is large and complex and it is neither possible nor necessary to sample all of it. We acknowledge that human exposures would only occur in a portion of the large "exposure areas" identified on the maps in Sectoion 3. The RA identified certain activities that could be "risky" depending on actual concentrations and frequency of use, e.g., recreational activities in the Lower Basin. Remedial actions will be made on a home-by-home basis and will not occur without sampling (if there is no data). Common use area remedial activities would be determined on a site-by-site basis. It is highly unlikely that any remedies will be applied wholesale to large areas where variable concentrations and human use patterns exist.
Data from 13 residences are included in the non-lead EPC values for the Lower Basin, not 4, see Table 3-21.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
225	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B14		Local Governments	0-Executive Summary			Partially Accepted

Comments>

The exposure area for a construction worker in the Lower Basin is much greater than a few feet from the bank of the ten-mile stretch of the Coeur d'Alene River that was characterized in the Lower Basin. The assumption that this small area represents the construction worker exposure area may result in over estimated risk and inappropriate risk-management decisions (is active remediation required to protect construction worker health?). It is recommended that exposure areas are defined for all receptors in all locations evaluated for the risk assessment.

Response>>

We acknowledge that risks to construction workers in the Lower Basin are likely over-estimated. If a construction worker is disturbing flood plain soil/sediment in the Lower Basin, then some potential for adverse health effects may be possible and the communities need to be aware of this. In reality, if there is a possibility that a construction project will disturb soils and sediments that have been impacted by mining, then samples should be collected and steps taken to protect the worker. This scenario is already part of the Institutional Control Program in the Upper Basin. The HHRA identifies that mining-contaminated soils could be a problem for construction workers but due to the large area of the Basin every "safe" or "unsafe" area cannot be explicitly identified by the HHRA.

Misc. Input>>

226	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B15		Local Governments	0-Executive Summary			Accepted

Comments>

Dermal exposure for children (no shoes, shorts, short-sleeved shirts) from April through November does not agree with local climatic conditions. The number of months for this exposure frequency needs to be reduced to reflect reality. Assuming children are bare foot April through November in the Coeur d'Alene Basin is not likely to pass a "smile test" and leads to public questioning of the "professional judgement" used in the risk assessment.

Response>>

We agree that the dermal surface areas used for the 4 to 11 year old age group were too large for the exposure period. Text and a table will be added to the report which demonstrate how calculated risks and hazards will be affected by alterations in surface area. Preliminary estimates indicate neighborhood exposure estimates will drop by <10%. Risks and hazards for combined neighborhood exposures do not drive the overall risks and dermal exposures were a relatively low percentage of the total neighborhood risks in the report (35% to 17% for arsenic). Hazard indices only slightly exceeded 1 for neighborhood receptors for two areas: Side Gulches (which was based on Elk Creek Pond, since remediated), and Burke/Ninemile (which included waste piles). Therefore, changing skin surface areas for neighborhood exposures will not affect the conclusions of the risk assessment or potential risk management strategies.

Misc. Input>>

227	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B16		Local Governments	0-Executive Summary			Not Accepted

Comments>

The Report does not include the analytical data in a way that allows relationships between contaminants and exposure areas to be reviewed. For example, the exposure point concentrations are very different for construction workers, recreational children, and residents. Where are these exposure areas? They should be defined on maps that identify sampling locations. Because this information is not included, the exposure point concentrations and estimated risk cannot be confirmed by the public.

Response>>

The details of the analytical data used to calculate the recreational EPCs (except Blackwell Island which will be included) are presented in Appendix E. The 15 maps in Section 3 show the approximate sample locations for all data except residential. Some of the common use areas have sample numbers combined by area in the interests of consolidating the map requirements. Appendix F presents all of the data used to quantify risks by exposure area and it also includes the residential data and Blackwell Island. Section 3, pages 3-38 to 3-41 describes the data used to calculate each EPC. Appendix A contains every input to every risk calculation with formulas.

Misc. Input>>

228	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B17		Local Governments	0-Executive Summary			Not Accepted

Comments>

The detection limits for non-detects and analytes that were not analyzed are not reported.

Response>>

The mining-related chemicals have been correctly identified and agree with historical assessments. Detection limits for other chemicals, not mining-related, would not affect the risk assessment. In addition, detection limits are reported in the Field Sampling Plans.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
229	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B18		Local Governments	0-Executive Summary			Partially Accepted

Comments>

Note that the Lower Basin had only four sample locations and 25 to 28 individual analyses to estimate the exposure point concentration. It is not clear that the quantity of data is sufficient to support decision-making over such a large area.

Response>>

There were 13 homes sampled in the Lower Basin for non-lead. See Table 3-21. Therefore, thirteen locations with a total of 28 samples were used in the calculations. Risk management decisions will not be made for the entire area, they will be made on a house-by-house basis and never in the absence of location-specific data.
The summary table in Appendix E included only EPA data, not the additional residential data from the State used in the non-lead EPCs. We will delete any reference to residential data from the Appendix E Table. All residential data is included in Appendix F by geographical area.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
230	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B19		Local Governments	0-Executive Summary			Not Accepted

Comments>

Analytical data are not identified by sample number in the report. Only statistical summaries are provided (they list individual analytical results) and the summaries often combine different media (upland soil and sediment of various depths). The actual data for metals other than lead should be provided by sample number and depth.

Response>>

Appendix E lists all the data used to calculate EPCs (except the residential EPCs and Blackwell Island) by media. The sample number and depth (where applicable) are both identified in these tables. Appendix F contains this same data as well as the residential data.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
231	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B20		Local Governments	0-Executive Summary			Partially Accepted

Comments>

There are inconsistencies between exposure frequency for incidental soil ingestion and dermal contact for residential, neighborhood recreational, and public recreational receptors. These exposure frequencies must be identical for a given receptor. For example, the public recreational receptor cannot be exposed to soil by skin contact for 68 days and incidentally ingest soil for 30 days. Incidental soil ingestion is due to hand-to-mouth transfer of soil and any time there is skin exposure; there is incidental ingestion. This inconsistency requires correction for several receptor populations evaluated in the risk assessment.

Response>>

The exposure frequencies for dermal absorption and ingestion are the same. The units for dermal exposure, however, are "hours per event" while the units for ingestion are "hours per day". The ingestion exposure frequency was normalized to 14-waking-hour days. This is explained in the table notes for Tables 3-23 and 3-24. However, a discussion will be added to the report to clarify this point and a table will be added to show more explicitly the steps that were taken to arrive at the exposure frequencies.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
232	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B21		Local Governments	0-Executive Summary			Accepted

Comments>

The Report should clearly state the differences between EPA's "acceptable cancer risk range" and the level of risk that warrants remedial action. The 1.0E-4 to 1.0E-6 range (a range of one additional cancer in 10,000 exposed individuals to one additional cancer in 1,000,000 exposed individuals) is a Superfund cleanup goal for sites that undergo remedial action. A 1.0E-04 cancer (one in 10,000 exposed individuals) risk is the level at which remedial action may be warranted, assuming no other adverse environmental impacts.

Response>>

Discussion will be added to the report which notes these differences. We also note that risks over 10-4 are not the only time action is warranted. The NCP allows actions at lower risk levels depending on site-specific conditions.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
233	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B22		Local Governments	0-Executive Summary			Not Accepted

Comments>

Based on EPA guidance, EPA's Integrated Exposure Uptake Biokinetic (IEUBK) Model, which is used to predict child blood lead levels, is not appropriate to evaluate periodic or episodic exposure to lead. However, the Report evaluates periodic lead exposure with the IEUBK Model. Because the Report "force fits" an episodic or intermittent exposure scenario into the IEUBK Model, there is disagreement with EPA guidance and the basis of the Model.

Response>>

The HHRA disagrees with this comment. The IEUBK model is relevant for continuous exposures that are of sufficient duration to produce quasi-state blood lead concentrations. The incremental exposures evaluated by IEUBK analysis should not be characterized as episodic. The exposures evaluated are seasonal in nature, occurring over 6 to 8 month periods, with event frequencies of at least once per week. The TRW comments at Section 2.5 provide additional discussion regarding this topic. See also General Response to Comments, #5a, #5b, and #9a.

Misc. Input>>

234	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B23		Local Governments	0-Executive Summary			Not Accepted

Comments>

The exposure assumptions used in the IEUBK Model for the Upland Parks and Schools recreational receptors assume an exposure frequency of 238, rather than 68 days per year (twice a week for 34 weeks). This overestimates the potential lead intake and blood lead levels by a factor of 238/68 (three and one-half times). A similar overestimate of exposure frequency is made for other receptors and exposure scenarios that occur away from the yard.

Response>>

The HHRA disagrees with this comment as it is inconsistent with the methodology employed in the HHRA. The Central Tendency (CT) Exposure Frequency is a 7 hour/day event, one-day per week, for 34 weeks. The RME factor is the same for two-days per week. The total number of hours spent on the recreational activities by children at Upland Parks are equivalent to 17 and 34 days, respectively, adjusted for waking hours. The RME exposure does occur on 68 days over a 238 day season. The HHRA averaged the 17 days of equivalent exposure over 365 days for inclusion in IEUBK model. The TRW comments on Section 2.5 provide additional discussion regarding this topic. The TRW concludes that the exposure duration is sufficient to include in IEUBK analysis, but believes the risk may be understated, by about 35%, as the exposure should be averaged over 238 days, rather than 365 days. See also General Response to Comments, #5a and #5b.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
235	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B24		Local Governments	0-Executive Summary			Partially Accepted

Comments>

The Data Evaluation Section of the Report simply reviews the laboratory data quality and does not demonstrate that: the data are appropriate for risk assessment. The report does not compare the collected data with criteria identified in EPA's Data Usability in Risk Assessment Guidance (EPA 1992).

Response>>

While not explicitly noted in the text, the four data application issues from the 1992 guidance were met and are as follows:

1. What contamination is present at what levels? -- Adequately addressed in Section 2 which describes sample collection methods, data analysis procedures (metals), and notes where samples were collected specifically for human health needs versus other uses. The vast majority of the data used in the HHRA was collected based on human health considerations and fulfills the requirements of risk assessment guidance described in EPA's 1989 Risk Assessment Guidance for Superfund and in the 1992 document. For the relatively small amount of data used that was not collected for HHRA use (sediment and surface water data in the South Fork, Canyon Creek, and Ninemile Creek), the uncertainties surrounding this data are discussed in both Section 2 and in Section 7 of the report. Other than the data noted above and the special case of waste piles, all samples were collected using a randomized or systematic sample design appropriate for risk assessment evaluations.
2. Are site concentrations different from background? -- Adequately addressed in Section 2 which presented background concentrations for applicable media (except groundwater) and selected COPCs based on concentrations exceeding background levels and health levels.
3. Are all exposure pathways identified and examined? -- Adequately addressed in Section 3 where exposure pathways were exhaustively discussed and conceptual site models by human health geographic area were presented.
4. Are all exposure areas fully characterized? – Human health exposure areas were discussed in Section 3. However, they were not explicitly defined in many cases due to the large and complex area of the Basin. This lack will be addressed in documents addressing remediation which will select individual locations on an area-by-area basis. See previous response to Comments B5, B13, B14 on exact exposure area definitions versus risk management practices.

Text will be added which briefly discusses the data usability guidance and the existing discussions mentioned above will be identified as fulfilling the appropriate data application issue from the 1992 guidance.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
236	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B25		Local Governments	0-Executive Summary			Partially Accepted

Comments>

The Data Evaluation Section of the Report simply reviews the laboratory data quality and does not demonstrate that: the Data Quality Objectives used to plan data collection, were met (DQO Process guidance, EPA 1993). The Report should compare the quantity and quality of the collected data with that planned in the Field Sampling and Analysis Plan (FSAP) to ensure that collected data support making decisions identified in the FSAP at the specified confidence levels, and

Response>>

See General Response to Comments regarding DQO/DQA issues and responses to Comments B3 and B6. In general, the data that was collected for use in the HHRA was of the same quality and quantity and at the specified confidence levels (either 95 or 99 percent) as that planned in the FSPAs. We note that FSPAs 6,7, and 12 were residential samplings and sampled only on a volunteer basis. The risk assessment discusses the limitations of using volunteer data in the uncertainty section. However, for the lead risk assessment over 800 homes in the basin were sampled. Leading the human health risk assessment team to believe that this data set is sufficient to adequately evaluate risks. As discussed in the General Response to the DQO comments, the DQO process was considered and documented to varying degrees in each of the FSPAs. Therefore, for further discussion see the specific FSPAs and their alterations reports. Text will be added to Section 2.3 which briefly discusses the DQO process and how it was followed and a reference for Section 4.2 of Part 1 of the RI will be added which refers the reader to further discussion on the DQO process.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
237	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B26		Local Governments	0-Executive Summary			Partially Accepted

Comments>

The Data Evaluation Section of the Report simply reviews the laboratory data quality and does not demonstrate that: the data are of the quality identified as needed to support decisions that were identified. This data quality analysis would follow EPA's DQA Guidance (EPA 1998).

Response>>

See General Response to DQO comments and responses to Comments B3, B24, and B25. We are unclear what decisions the reviewer is referring to. The purpose of the Risk Assessment is to quantitatively evaluate risks and to provide qualitative discussions of the uncertainties associated with use of the available data. We believe that the data used in this risk assessment adequately supports the risk conclusions that were made. Text will be added to Section 2.3 which briefly discusses the DQA process and how it was followed and a reference for Section 4.2 of Part 1 of the RI will be added which refers the reader to further discussion on the DQA process.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
238	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B27		Local Governments	0-Executive Summary			Not Accepted

Comments>

The Report identifies good agreement between measured and modeled blood lead values. This leads the public to believe that the model predictions are appropriate to protect their children from over exposure to lead. Unfortunately, the comparison made in the Report is simply comparing the two data sets (measured and modeled blood lead levels) a technique that does not support a conclusion that the model is accurately predicting blood lead. Causality between modeled and measured values is assumed and not supported. A "nonsense" example of a "spurious correlation" that is a statistical fact is this: "There is a close relationship between the salaries of Presbyterian ministers in Massachusetts and the price of rum in Havana." Which is the cause and which the effect? In other words, are the ministers benefiting from the rum trade or supporting it? [Taken from "How to lie with Statistics, D. Huff, 1954]. Agreement between modeled and measured blood lead levels must be evaluated for individuals that have measured blood lead levels and yards that have been characterized. The Report indicates that individuals were modeled in the batch mode, which means all data are entered to predict each individual's blood lead, based on the analytical data for the individual's environment. These individual predictions can be compared to the measured blood lead values as paired data. If the model accurately predicts the blood lead, a scatter plot of modeled versus measured data will show a high correlation coefficient and most of the predicted high blood levels will correlate with high measured levels. The scatter plot will identify the number of children at risk (blood lead greater than 10 ug/dl) that are not identified by the IEUBK Model and the number of children predicted by the Model to exceed 10 ug/dl that actually do not. This information will help the public and the risk manager to understand the uncertainty in IEUBK Model results in terms of protecting children's health. If there is a significant difference between the individual measured and Modeled blood lead levels, it is recommended that the issue be reviewed by EPA's Lead Technical Review Workgroup, as suggested by EPA guidance.

Response>>

The HHRA disagrees with this comment. The relationship between blood lead levels and environmental exposures is examined throughout the HHRA by a variety of methods. In regression analysis, it is common practice to compare dependent blood lead levels predicted from independent exposure variables to observed concentrations. In the IEUBK analysis, the same independent exposure variables are input to a mechanistic model and outcome blood lead levels are predicted. It is also common to compare these predictions to observed blood lead levels. Both the dependent and independent variables come from the same home and community and the objective of the analysis is to investigate and quantify any relationship between the variables. The regression analysis discussed above shows a relatively strong relationship, that is consistent with plausible environmental and biological processes, and is similar to the findings of investigations at other sites including the BHSS. As a result, it is appropriate to compare predicted and observed blood lead levels in both empirical and mechanistic procedures. The HHRA has been extensively reviewed by the EPAs Technical Review Workgroup (TRW) for Lead and the review is attached.

Misc. Input>>

239	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B28		Local Governments	2-Section 2			Not Accepted

Comments>

Conceptual Site Models (CSMs) appear to be based on the ecological risk assessment needs and concepts. CSMs are not geographical areas as defined in Section 2. The CSM graphic and supporting text simply defined (based on existing knowledge) how chemicals are transported from a source to a locations where receptors are exposed. The CSM identifies the sources being investigated, the release mechanisms of chemicals from identified sources, the transport mechanism of released chemicals from the source to the exposure point, and the intake routes of media containing transported chemicals at exposure points. The CSMs in the Report do not focus identifying the problem and data needs for human health risk assessment. The Data Quality Objectives Process Guidance (EPA 1993) indicates that identifying the problem is fundamental to determining the data needs and the decisions that are to be supported by those data.

Response>>

The fate and transport of mining materials does not differ between ecological and human receptors. The applicable human receptor pathways are included and the data exists to support evaluation of the quantified pathways. Section 2 defines CSM Units which are geographical areas that were defined for RI/FS purposes. The purpose of this section was to link the study areas of the HHRA and the EcoRA. Section 3.2.1 describes the CSM diagrams (Figures 3-3 to 3-11) and how they are applied in selecting media and pathways of concern to human health. See also response to Comment A26.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
240	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B29		Local Governments	2-Section 2			Partially Accepted

Comments>

The amount of time that 4-11 year-olds are assumed to be outdoors playing in various recreational activities, 10 hours on beach areas and 7 hours twice a week for upland soil exposure does not appear to be supported by EPA's Exposure Factors Handbook (EFH). Tables 14-1 through 14-9, of the EFH list mean and median exposure times outdoors, and for total recreation do not appear to support the exposure times listed in the Report. Documentation that is more specific is suggested. Because the exposure assumptions are over relative long periods of time (ranging from 16 to 34 weeks), it is assumed the exposure is chronic in nature. For a chronic outdoor exposure time EPA estimates the average 3-11 year-old child spends less than 4.7 hours each day (Table 14-12, Exposure Factors Handbook). This value is significantly lower than the exposure times given in the Report, which appear to be undocumented professional judgement. Professional judgement requires documentation or support for these rather significant differences from EPA's guidance.

Response>>

Chapter 14 in the 1997 EFH is the breast milk chapter, not the activity factors chapter; therefore, we were not able to refer to the tables listed in this comment. However, the exposure frequencies used for the 4-11 year old age group are values from the 1997 EPA Exposure Factors Handbook, Chapter 15. This document is cited as the source of the exposure frequencies. Specifically, information from tables 15-104, 15-108, 15-110, 15-132, 15-135, and 15-176 were reviewed and used to select RME values. A value of 10 hours per day was not used for neighborhood exposures, but only public exposures which assumed people would be traveling some distance to get to the recreational area and would spend the entire day there. Neighborhood exposure times were either 7 hours per day (EFH Table 15-176 recommended weekend time) or 3 hours per day (EFH Table 15-132 50th percentile time for 5-11 yr olds, rounded up from 2 and 1/2 hours to 3 hours, also consistent with assumptions made for the Bunker Hill Superfund site RA).

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
241	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B30		Local Governments	2-Section 2			Not Accepted

Comments>

Blackwell Island is to be developed into a recreational area through the efforts of the county, state agencies, and the public. This is written on a sign at the entrance (north of highway) to the area. There is a "no swimming" sign, apparently to warn of unacceptable metal concentrations on the south side of the highway. However, there is little or no evidence that receptors are frequenting the area on the south side of the highway. Figure 1 is a photo of the area does not show evidence of recent use.

Response>>

The risk assessment covers future as well as current use assuming no remediation efforts are made (baseline conditions). No evidence of current use does not mean there will be no use in the future, particularly as Blackwell Island is an identified recreational area.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
242	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B31		Local Governments	2-Section 2			Not Accepted

Comments>

The trail on the north side of the highway leads to the water. Figure 2 is an overview of the area north of the highway and documents the lack of significant "beach" areas. There is a small area of course sand or fine gravel approximately 15 feet long and 4 feet wide along the river's edge, which is shown in Figure 3. All other areas have vegetation growing to the water's edge as shown in Figure 2. It is not clear from the Report whether sample locations represent the recreational receptor exposure area.

Response>>

Areas to be sampled were selected by local individuals familiar with the recreational use patterns of the area. Sample locations can be found in Appendix B, Figures B-17A and B-17B. See also response to Comment B30.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
243	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B32		Local Governments	2-Section 2			Not Accepted

Comments>

Sampling locations should be identified for Blackwell Island. Recreational activities observed in the early morning included a jogger and a fisherman at the channel that causes Blackwell Island to be an island. It is recommended that the receptor populations hypothesized for Blackwell Island are reevaluated and supportive rationale provided for the selection.

Response>>

The intent of the HHRA is to provide risk estimates under reasonable maximum exposure scenarios. We acknowledge that "reasonable maximum" likely overestimates site use for many individuals; however, the overestimate is consistent with EPA risk assessment policy which makes health protective assumptions to protect public health. See response to Comment B4 and B30. Sample locations can be found in Appendix B, Figures B-17A and B-17B.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
244	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B33		Local Governments	2-Section 2			Partially Accepted

Comments>

The relationship between the identified exposure area and the sample locations should be described in the Report. FSAP 05 indicated that photos were taken of the area sampled and that the beach area was estimated in the field by pacing or stepping the area. This information should be included in the Report. Based on field observations, it appears that the risk evaluation and any remedial action would be limited to the small 15 by 4-foot area.

Response>>

See response to Comment B4 and B30.
Discussion will be added to the report to clarify the Blackwell Island beach area and a citation will be added to refer the reader to the photo in Appendix B, page A-5 and the diagrams in Appendix B, Figure B-17A and B-17B.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
245	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B34		Local Governments	2-Section 2			Not Accepted

Comments>

Future development by the county, state, and the public will dramatically change the existing metal concentrations. This should be considered in evaluating future potential risk.

Response>>

See response to Comment B30.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
246	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B35		Local Governments	2-Section 2			Not Accepted

Comments>

EPA's risk assessment guidance (EPA 1989, page 25) states "if a screening level approach suggests a potential health concern, the estimates of exposure should be modified to reflect more probable exposure conditions." It is not clear how the exposure estimate was modified for the Baseline Risk Assessment, compared to the expedited screening level risk assessment for Coeur d'Alene beach areas. The differences should be identified.

Response>>

See response to Comment B6.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
247	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B36		Local Governments	3-Section 3			Not Accepted

Comments>

Evaluating a child-only exposure scenario for non-carcinogenic health effects is overly protective (EPA's Science Advisory Board, 1993). SAB concluded that evaluating child exposure in combination with chronic toxicity criteria is overly protective. However, they noted that the approach may be appropriate for chemicals with chronic reference doses (RfDs) based on health effects that are specific to children (e.g. health effects related fluoride and nitrates in children) or where the dose-response curve is steep (i.e., the difference between the no-observed-adverse-effect level (NOAEL) and the adverse effect level is small. It is recommended that child/adult exposure be included in the potential risk assessment of metals. SAB concluded that the child/adult exposure scenario was sufficiently conservative for risk-based decision-making.

Response>>

See response to Comment B7.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
248	10/03/2000	R. Merril Coomes	Public Draft - July 2000	3.2.2	0/31/2000	URS
B37		Local Governments	3-Section 3			Not Accepted

Comments>

There is considerable discussion in Section 3.2.2 explaining that dermal uptake from water was limited to dissolved metals. However, the recreational beach exposure scenario apparently evaluates dermal uptake of surface water containing "stirred" or suspended sediment. It is not clear, how the beach exposure scenario for dermal exposure to suspended sediment is in agreement with the discussion in Section 3.2.2 and should be explained.

Response>>

Section 3.2.2 discusses pathways excluded from quantification. The dermal pathway for water was not quantified.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
249	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B38		Local Governments	3-Section 3	p.3-25		Accepted

Comments>

Page 3-25 states that inhalation Screening Values (SVs) were estimated using the particulate emission factor (PEF), as discussed in Section 2.4.1. Section 2.4.1 does not discuss PEF or how they are applied in this risk assessment.

Response>>

Page 3-25 contains a typographical error. Section 2.4.5 should be referenced rather than 2.4.1. The error will be corrected in the next version of the HHRA.

Misc. Input>>

250	10/03/2000	R. Merril Coomes	Public Draft - July 2000	3.2.3	0/31/2000	URS
B39		Local Governments	3-Section 3			Accepted

Comments>

Section 3.2.3 indicates that dermal absorption of metals is very slow and that "available data indicate that the contribution of dermal soil exposure to overall risk is typically small". However, the risk assessment appears to be inconsistent with this concept and identifies the dermal contact pathway as a significant contributor.

Response>>

See also response to Comment B15. Discussion will be added to the report to indicate that the dermal contribution to residential exposures is small, but that dermal contribution for neighborhood exposures can be higher because of the increased skin surface area assumptions. However, changing skin surface areas for the neighborhood exposures has little overall affect on risks. A table will be provided to show the dermal contributions to total risks and hazards based on reduced skin surface area.

Misc. Input>>

251	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B40		Local Governments	3-Section 3	p.3-35		Partially Accepted

Comments>

Page 3-35. It is indicated that the 95 percent upper confidence limit (95 UCL) was calculated only when the number of samples was greater than 10. FSAP Addendum 05, page 8, which based statistical rationale on the median, identified 7 samples as sufficient to estimate the 95 UCL. What caused the difference in the planned analysis and the Report analysis? This should be discussed in the data evaluation Section, following EPA's DQA guidance. An EPA citation and statistical rationale are recommended to support the position that 10 samples are too few to calculate a 95 UCL for human health risk assessment use. This position does not agree with Basin Sampling and Analysis Plans or, in general, with other EPA risk assessments. For example, 95 UCL values were calculated for the Spokane River risk assessment, where seven samples were collected at each site. If the decision that supported less than 10 samples were not risk-based, it is possible that the sample results are not appropriate to evaluate potential human risk. This would become clear if the Data Evaluation section used the DQA Process and Data Usability in Risk Assessment Guidance (guidance that is not cited or used in the report).

Response>>

The discussion on page 3-25 explains that a minimum of 7 samples was sufficient to calculate a UCL on beaches because of the relative homogeneity of the beach materials. The number of minimum samples was increased to 10 in the HHRA for non-beach areas because the EPCs were not all from relatively homogenous materials from the same source, as is consistent with the 1992 Supplemental Guidance to RAGS: Calculating the Concentration Term. Some discussion will be added to the text explaining that in only nine of the 49 EPCs calculated was the maximum concentration used because there were fewer than 10 samples, for these nine cases there were also fewer than 7 samples.

Misc. Input>>

252	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B41		Local Governments	3-Section 3	p.3-38		Not Accepted

Comments>

Page 3-38 states that there are insufficient data to statistically compare metal concentrations for analyzed soil floor mats and house dust. There are 83 matched data sets for soil and floor mats and 74 matched data sets for soil and house dust. If the hypothesis is whether or not there is a statistical relationship between the soil and floor mat or soil and house dust, there is sufficient information to make a comparison. Note statistics used to support decisions that data sets are the same, for example the Students T statistic, change very little after a sample number of about 30. The reviewer recommends performing the statistical tests and then explaining the resulting relationship.

Response>>

See response to Comment B10. Statistical evaluations were included in Appendix I and discussed in Section 7. There was insufficient data by geographic area, not overall.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
253	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B42		Local Governments	3-Section 3			Not Accepted

Comments>

The Report expressed concern about the relationship between yard soil and house dust for smaller geographical areas. If smaller areas are of concern and a relationship between soil and indoor dust is expected, the sub area could be tested to determine whether there is a difference between sub areas using nonparametric statistical tests such as the Wilcoxon Rank Sum. There appear to be sufficient data to estimate the uncertainty associated with this assumption.

Response>>

See response to Comment B10. We do not agree that there is enough data in each geographic area to make results of a Wilcoxon Rank Sum test meaningful. In addition, there are other issues regarding the relationship between yard soil and house dust that contributed to the decision not to use the data in the risk calculations. These reasons are discussed in Section 3 and 7.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
254	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B43		Local Governments	3-Section 3			Partially Accepted

Comments>

The Report assumes that soil is the major contributor to indoor concentrations of chemicals in dust because of the uncertainty in the soil/house dust data relationship. EPA RI/FS guidance states that the objective is not to eliminate uncertainty, but to make confident decisions with acceptable uncertainty in the data. Risks Assessment Guidance states that the quantitative risk assessments are performed when the uncertainty in the estimate is known (EPA 1992). It is recommended that the uncertainty in the relationship between soil and house dust be explained in a manner the public can interpret.

Response>>

The uncertainty in an estimate of concentration change between the soil and house dust data is not known. The HHRA used soil as a surrogate and some of the implications of that choice were discussed. Page 3-38 of the HHRA states "Using soil concentrations as surrogates for house dust concentrations has the potential to either underestimate or overestimate human health risks." Further discussion of this issue was provided in Section 7. The text of Section 3 will be reviewed and clarified.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
255	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B44		Local Governments	3-Section 3	p.3-39		Partially Accepted

Comments>

Page 3-39 States “The uncertainties regarding the exact relationship between dust and soil concentrations for metals other than lead make predicting a house dust concentration from a soil concentration problematic.” This statement appears to assume that lead is transported differently in the environment than other metals and that an exact relationship is required. The acceptable uncertainty for exact should be identified. Differences in transport of lead compared to other metals, should be supported with Conceptual Site Models (and supporting text) that identify the differences between the transport pathways from yard soil to house dust for lead compared to other metals. Note that EPA guidance does not require making exact relationships known. Because paired data are not presented in the Report, the potential relationship between yard soil and house dust, for other metals, cannot be reviewed.

Response>>

We agree that an "exact" relationship is not required. The reference to lead was meant to refer to the fact that lead dust issues have been more extensively studied and there is considerably more data for lead in dust than for other metals. We are not assuming that soil-to-dust transport mechanisms would necessarily be different for lead than other metals.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
256	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B45		Local Governments	3-Section 3	p.3-39		Partially Accepted

Comments>

Page 3-39 waste pile samples. Exposure to surface material is limited to the top inch of surface material. This boundary condition is described in many parts of the Report and is a basis for considerable data collection related to human exposure. However, it is stated that waste piles contained little or no fine material within the top one-inch and this is the only material (top one-inch) that would be ingested by hand-to-mouth transfer. Because there was insufficient fine material in the top one-inch, bulk "source" samples were apparently taken of the waste rock pile. EPA's risk assessment guidance cautions about using source characterization data for risk assessment. The guidance (EPA 1989, page 6-28) states that care must be taken in using such data (obviously contaminated soil or hot spot areas) to estimate exposure concentrations. EPA indicates that when a source area is included in a risk assessment, that a random sampling plan be used to obtain "source" data for the risk assessment. Data from the waste piles are apparently source characterization samples and are not likely to represent the potential for exposure. The reviewer does not have the FSAP for waste pile characterization. Were the samples randomly collected from the waste pile or judgmentally selected? Does the FSAP indicate that the samples will be used to support risk assessment? If it does, what are the identified uncertainties of using these data? These issues need to be documented in the Report. This comment assumes the samples were judgmentally selected for source characterization. If this is true, these data may not be appropriate for risk assessment. The limitations of using these data should be discussed in the exposure assessment and uncertainties sections.

Response>>

See response to Comment B11 and B12. Samples from waste piles were collected specifically to support the HHRA (FSPA08).

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
257	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B46		Local Governments	3-Section 3	p.3-40		Accepted

Comments>

Page 3-40. The Report describes floodplain soil/sediment samples as two intervals, the top 12 inches and 0-1 inch. However, data included in Table 1.1, Appendix E and the FSAP, identifies that data were also collected from the 0-6" depth (three individual sample depths).

Response>>

Text will be amended to include discussion of all three depth intervals as are noted in Section 2.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
258	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B47		Local Governments	3-Section 3	p.3-40		Partially Accepted

Comments>

Although the FSAP provided rationale to support collecting separate depth interval data (evaluating different receptor populations such as campers) the data were combined in the risk assessment for a single receptor. Difference between the rationale used in the Report and that used for data collection should be explained in the Report. The rationale to collect the data separately was provided in the FSAP, but the rationale to support combining the data is not discussed.

Response>>

Text will be added to clarify the reasons data were combined for a single receptor group. See also response to Comment B2.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
259	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B48		Local Governments	3-Section 3	p.3-40		Accepted

Comments>

Because the actual data are not provided in the Report by depth, it cannot be determined for all cases that combining the data is appropriate. However, a statistical analysis of the data for Blackwell Island is possible because the screening level risk assessment provided the average, maximum, and minimum values for arsenic. Using that information (Pages 49 and 50 of the November 30, 1998 Expedited Screening Level Risk Assessment), the soil and sediment values for different depths can be identified and are shown below:

Blackwell Island Data for Arsenic

Sediment, 0 to 6” and 0 to 12”	Upland Soil, 0 to 1”
Max = 83.4	Max = 20.3
Min = 19.8	Min = 9.7
19.8	9.7
37.2	12.4
38.8	12.5
39.8	14.2
45.8	15.7
52.2	16.7
53.4	20.3
56.7	Average 14.5
59.2	
59.3	
63.5	
74	
77	
83.4	
Average 54.3	

A Wilcoxon Rank Sum test demonstrates the two data sets are from different populations (data sets that cannot not be statistically combined) at the 99 percent confidence level. The available data do not allow distinguishing between the 0 to 6’ and 0-12” sediment data. The Report combines these data sets and then proves statistically that the distribution is not log normal and is a normal distribution. The analysis is not meaningful when the two data sets are from different populations. There is concern that sediment and soil data sets have been inappropriately combined for all recreational soil/sediment exposure scenarios. It is recommended that the actual data be summarized in the report to allow reviewers to confirm that combing the data sets is appropriate.

Response>>

Actual data for Blackwell Island were provided in Appendix F; however, the detail with sample number and depth appears to have been inadvertently left out of Appendix E. This information will be added. See also response to Comment B19.

Misc. Input>>

260	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B49		Local Governments	3-Section 3	p.3-40		Accepted

Comments>

In addition, the difference between the FSAP and Report, related to data evaluation and receptors, should be explained in the Report.

Response>>

Text will be amended to describe the differences. Also, see response to Comment B2.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
261	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B50		Local Governments	3-Section 3	p.3-40		Not Accepted

Comments>

Page 3-40 surface water sampling. Two methods were used, one to collect surface water and the other to collect “stirred” surface water. Risk assessment guidance states to evaluate surface water and sediment exposure that both media are collected from the same location. Risk assessment guidance does not identify a “stirred” sampling approach to evaluate exposure. The guidance does indicate that exposure to surface water and sediment occurs at the same time and place. Therefore, sediment and surface water samples should be collected from identical locations at the same time. The stirred sample data may “double count” the risk contribution made by sediment.

Response>>

Sediment and stirred surface water were collected from the same place at the same time. The sediment swallowed in the water represents a separate exposure in addition to the "usual" sediment ingestion during land activities. Therefore, we disagree that there was double counting.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
262	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B51		Local Governments	3-Section 3	p.3-40		Partially Accepted

Comments>

Page 3-40. It is stated that the fish tissue concentrations used in the risk assessment “likely represents somewhat of a “worst case” for human consumption. EPA policy specifically states that risk assessments are not intended to evaluate worst case exposure scenarios. It is recommended that the exposure scenario be modified to be in agreement with EPA policy. Note that simply changing the description of “worst case” is not appropriate. The exposure parameters or estimated concentrations should be modified for the fish tissue exposure scenario.

Response>>

Fish from the lateral lakes may represents somewhat of a worst case when considering fish from other areas (such as Lake CdA). However, if a person fishes in the Lateral Lakes area, EPCs and exposure parameters are representative of RME exposures. The text will be clarified.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
263	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B52		Local Governments	3-Section 3	p.3-41		Partially Accepted

Comments>

Page 3-41 Construction site soil. Using all soil data from all depth intervals within a geographical area is not appropriate. This approach dilutes the potential hot spot areas, lowering the estimated risk. In addition, the approach increases the estimated risk at “clean” locations within a geographical area that actually do not pose unacceptable risk. This problem is widespread in the risk assessment for all receptors and adds confusion to risk-management decision-making.

Response>>

We agree with the reviewer that the approach dilutes potential hot spots and overestimates risks to "clean" areas. The goal of risk assessment is to estimate "average" exposures (with an adequate margin of safety) for a specific activity. Construction workers could be moving dirt in both clean and dirty areas; therefore, the estimates are an appropriate representation of potential risks. As we described in our responses to Comment B13 and B14, all areas with potential mining wastes have not been sampled in the Basin; thus, our approach was to examine risk and hazards from exposure to both clean and dirty areas to get an idea of whether the over-all activity might be "risky" or not. Any individual, specific, construction project in the Basin must follow the institutional controls program (ICP) already in place and must collect samples if there is no data. The text will be amended to clarify this point.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
264	10/03/2000	R. Merril Coomes	Public Draft - July 2000	Appendix E	0/31/2000	URS
B53		Local Governments	3-Section 3			Partially Accepted

Comments>

Residential exposure for the Lower Basin appears to have been based on 4 samples, three from Cataldo and one from Harrison (Table in appendix E). A second table, identified as "Lower Basin Resid SS (1).xls (6/3/00) page 1 through 6" in Appendix A identifies 25 to 28 samples used to estimate the exposure point concentration, depending on the analyte. The sufficiency of these data to support a risk assessment that represents an area of more than 100 square miles (the assumed exposure area for a construction worker) is not clear. The area for decision-making is very large compared to the area actually sampled. It appears that the amount of data collected over the exposure area may be insufficient to evaluate construction worker risk and will result in significant uncertainties related to potential risk and the need for remedial action in the Lower Basin. The Report should identify the uncertainties in the risk estimate based on the limited number of samples.

Response>>

See response to Comment B18. The summary table in Appendix E only provides the EPA-collected data. This table will be modified. The residential data can be found only in Appendix F and is summarized on Table 3-21. 13 homes (not 4) were sampled in the Lower Basin. See also response to Comment B13. The data are representative of the areas sampled and remediation activities will need to collect site-specific data for areas which do not have data. The lack of data for the entire area is due to the size and complexity of the site. Data will have to continue to be collected to support risk-management decision-making.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
265	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B54		Local Governments	3-Section 3	p.3-46		Partially Accepted

Comments>

Page 3-46. The most recent average body weight for adult men and women is 71.8 kg (EPA, 1997). It is recommended that the more recent body weight be considered in the risk calculations.

Response>>

The use of 70 kg for average adult body weight is appropriate for the arsenic cancer risk calculations because 70 kg body weight was assumed in the derivation of cancer slope factors and unit risks. For the non-cancer risk calculations, however, we acknowledge that 71.8 kg is the newly recommended body weight in the 1997 EFH and use of 70 kg is a slight overestimation of actual risks. However, the most recent Region 10 RCRA guidance (1998) recommends use of the 70kg body weight and thus, this recommendation was followed in this risk assessment. We note that use of new 71.8 kg body weight would only minimally affect the risk calculations and would not affect the risk conclusions. A discussion will be added to the uncertainty section regarding how the higher body weight might affect risk estimates.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
266	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B55		Local Governments	3-Section 3	p.3-47		Not Accepted

Comments>

Page 3-47. The relationship between the average time a worker spends at one job and the central tendency exposure time for a construction worker is not clear. Would it be better to identify 6 years as an exposure assumption, without rationalizing the value? Because the area is experiencing a population decrease, is it likely that construction activities would be less than national average assumptions? Please discuss this issue in the exposure assessment.

Response>>

The 6.6 years is EPA's recommended average time a person spends in one job (Table 15-176 from EFH) while 25 years is the recommended time for RME scenarios. These exposure assumptions are identified as national averages and are not Basin-specific. It is unclear what is meant by "rationalizing the value."

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
267	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B56		Local Governments	3-Section 3	p.3-50		Not Accepted

Comments>

Page 3-50. Explain how a water intake of 30 ml/hr is appropriate for "playing" in water. This default intake, 30 ml/hr, actually represents a swimming scenario that assumes the swimmer's head is under water a significant percentage of the time. This value may be excessive for water play. A discussion is appropriate.

Response>>

We agree the value may be an overestimate for water play as opposed to swimming. However, because the risk calculations did not identify this pathway as exceeding health goals no additional discussion was provided in the uncertainty section. No changes to text.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
268	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B57		Local Governments	3-Section 3	p.3-51		Accepted

Comments>

Page 3-51. The exposure duration of 34 weeks (neighborhood recreational exposure to upland parks and schools), April through November is not consistent with the dermal exposure assumptions that assume shorts, bare feet, and short-sleeved shirts for children ages 4 to 11 years old. The climate in April and November (perhaps October) make the dermal exposure assumptions improbable. Snow and ice are tough on bare feet.

Response>>

See response to Comment B15. Discussion and a table will be added to the uncertainty section indicating how risks and hazards would be lowered by reducing skin surface areas.

Misc. Input>>

269	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B58		Local Governments	3-Section 3	p.3-51		Accepted

Comments>

A similar issue is related to the paragraph on page 3-50, Skin Surface Area. Local climate must be considered, especially since it is assumed in the previous paragraph that exposure to yard soil is 270 days each year. The skin exposure frequencies appear to be approaching a "worst case" estimate and are higher than a site-specific upper-bound estimate (RME) recommended in EPA risk assessment guidance.

Response>>

See response to Comment B15. Text will be added, no changes to risk calculations will be made.

Misc. Input>>

270	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B59		Local Governments	3-Section 3	p.3-51,2-52		Accepted

Comments>

Page 3-51 and 2-52. A soil (sediment) adherence factor of 0.2 is likely to overestimate the sediment adherence during beach play. The child or adult is in the water part of the time and the water activities would "wash off" the sediment. The exposure times for sediment and water contact are identical and it is likely assumed the exposures are concurrent. Please discuss this issue in the exposure assessment and the uncertainty section.

Response>>

The exposure frequencies for water and sediment differ, being 1 hour for water and 3 or 10 hours for sediment depending on whether neighborhood or public receptors are being considered. However, these exposures are anticipated to occur on the same day. The adherence factor of 0.2 was considered appropriate, particularly for public exposures, because of the likelihood that the majority of the time is spent out of the water. We agree the adherence factor may overestimate exposure for a beach scenario. No additions to the uncertainty section are planned because the pathway did not exceed target health goals or contribute significantly to over-all site risks and hazards.

Misc. Input>>

271	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B60		Local Governments	3-Section 3	p.3-53		Accepted

Comments>

Page 3-53. Soil Ingestion Rate. This paragraph indicates that about 300 mg/day is an upper percentile ingestion rate based on the cited study. What is the percentile represented by the "upper percentile" identified in the cited study (90 95, or 99 percentile)? What range of values is included in the qualifier about?

Response>>

Text will be added.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
272	10/03/2000	R. Merril Coomes	Public Draft - July 2000	3.4.2	0/31/2000	TG
B61		Local Governments	3-Section 3			Not Accepted

Comments>

Section 3.4.2 Adult Lead Model. It is stated that "The developing fetus is the most sensitive population for adult worker exposure". This is true, but EPA is responsible for "incidental or unknowing" exposure to lead that is not related directly to their jobs. The Occupational Safety and Health Administration (OSHA) is responsible for protecting worker health when the exposure is directly related to the work performed. OSHA has an acceptable and enforceable blood lead standard, which is 30 ug/dl blood lead. OSHA believes this concentration is protective of the fetus in the workplace. This issue should be discussed to ensure that the public understands that female workers, who are knowingly exposed to lead, are regulated by an OSHA blood lead level of 30 ug/dl. It should be clear for the public that EPA does not have authority over the blood lead levels of workers when knowingly working with lead-containing materials and that the acceptable blood lead level is different than currently discussed in the report.

Response>>

The purpose of the adult blood lead model is to predict PRGs (preliminary remediation goals) and not govern blood lead levels monitored in the workplace. The value of 10 ug/dl used in the model is based on the CDC guidelines to protect the health of children, and is therefore, used as a risk-based value in the model. Also, OSHA does not have a direct conflict with EPA's practice of its adult model for setting PRGs. See also General Response to Comments, #11and #10c.

Misc. Input>>

273	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B62		Local Governments	3-Section 3			Not Accepted

Comments>

The exposure assessment section does not discuss or define exposure areas for the receptor populations evaluated. For example, the residential exposure area is approximately ¼ acre or the size of an average residential yard. This value can be site-specifically adjusted depending on the characteristics of the demographic area being evaluated. EPA's SSL guidance evaluates exposure areas of ½ acre and identifies this area as the area to which a decision applies.

Response>>

See response to Comment B13 and others.

Misc. Input>>

274	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B63		Local Governments	3-Section 3			Not Accepted

Comments>

The exposure area for the "neighborhood recreational 4 to 11 year-old" should also be defined. For example, it is unlikely that this exposure area would include locations more than two (pick a number for the Report) miles from the residence. As the distance increases from the child's residence, the fraction of time exposed to the evaluated area would be expressed as "FI" (Fraction Ingested from the Contaminated Source) in the exposure algorithm.

Response>>

We agree that neighborhood exposures are likely limited to areas fairly close to home and in general, the sample locations used to evaluate neighborhood exposures are within 2 miles or less of a residence. Because the population is very spread out and the Basin is very large, a potential play area close to one home will be far from another within the same geographical region. The strategy selected was to look at average exposures to sediments, surface water, and waste piles in potential play areas within a region and get an estimate of whether such behavior might be "risky" or not. Adding an FI term under these conditions does not make sense. As described in the response to Comment B13 and B14, actual risk management decisions will be on a site-by-site basis and will take into consideration the concentrations at that spot and the number of residences in close proximity to the area.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
275	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B64		Local Governments	3-Section 3			Not Accepted

Comments>

Although FI is a critical concept when performing a risk assessment, it is not discussed in the report. Using appropriate FI values would result in a document that is in agreement with EPA guidance and would minimize the existing overestimates of potential exposure. A discussion of FI, as it relates to the defined exposure area for each receptor is strongly recommended in the revised report. A confident risk-management decision cannot be supported without using EPA's FI risk assessment concept.

Response>>

We disagree for this particular risk assessment, see response to Comment B63.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
276	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B65		Local Governments	3-Section 3			Not Accepted

Comments>

For example, a construction worker in the Lower Basin Area is likely to work in at least a ten by ten mile area (100 square miles). However, data have been collected only for a narrow corridor ten miles long adjacent to the Coeur d'Alene River. Assuming conservatively that collected data represents a width of one mile (note that all sample locations are actually adjacent to the river), only ten square miles have been characterized. Using these assumptions, a FI value for the construction worker would be 0.1.

Response>>

We disagree for this particular risk assessment, see response to Comments B14 and B63.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
277	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B66		Local Governments	3-Section 3			Not Accepted

Comments>

The Lower Basin risk assessment covers an exposure area that extends approximately 10 miles along the Coeur d'Alene River. If the risk is not acceptable in the Lower Basin along the river corridor and an active remedial alternative was selected, the area requiring active remediation would be the entire area, all 10 miles. Based on the limited data collection and combing sediment and surface soil data, it appears that the confidence associated with applying the results to the entire 10 mile area is low and the decision may not be defensible. There would be a high level of uncertainty in the decision that all of the area requires identical remedial action. Exposure area definition would assist in improving the confidence of the decision for each exposure area that was sampled, but would not provide additional confidence concerning decisions for the entire 10 mile stretch. It is concluded that the risk assessment, as currently structured is appropriate and/or sufficient to support risk-management decision-making for specific and isolated areas of the Lower Basin, but these isolated areas have not been defined or evaluated in the Report.

Response>>

No such decisions treating all of the area the same would be made. See response to Comment B13. The "isolated areas" for the Lower Basin are the common use areas already sampled and identified on the maps currently in the RA.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
278	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B67		Local Governments	3-Section 3	Figure 3-2		Not Accepted

Comments>

Figure 3-2 shows a declining population of students in the Coeur d'Alene River Basin. This should be taken into consideration when the "future use scenario" is considered. For example, groundwater in side canyons is not likely to become drinking water sources because there is little, if any, population growth pressure to develop those resources. Population decline considerations should be developed and included in the exposure assessment.

Response>>

We do not agree that population decline considerations should be included in the HHRA evaluation.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
279	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B68		Local Governments	3-Section 3	Figure 3-2		Not Accepted

Comments>

The assumption that groundwater will be used as a drinking water source should be documented using the procedures and rationale given in OSWER Directive 9355.7-04. Based on that Directive, it is unlikely that groundwater use in isolated areas would provide input to risk-based decision-making. If this scenario is to be included in the revised Report, please evaluate the probability of the scenario using guidance from OSWER 9355.7-04 in the Report.

Response>>

OSWER Directive 9355.7-04 does not apply to groundwater as it specifically states at the top of page four "Consideration of future ground water use for CERCLA sites is not addressed in this document." Groundwater is presently being used as a drinking water source in many homes in the Basin, and thus the media is appropriately evaluated under both current and future conditions as part of the residential scenario. In some cases, emergency response actions have been taken by the EPA and local health officials to address groundwater contamination over MCLs being drunk in people's homes. We do not agree that declining population issues affect the risk assessment evaluation of risks and hazards through drinking water although these issues may affect risk management decisions. Risk management decisions are discussed and documented in other reports than the risk assessment.

Groundwater near sources areas in Ninemile and Canyon Creek that is not currently being used as drinking water was also evaluated as a possible future scenario. The results of this evaluation were kept separate from the "baseline" residential risks and presented as potential additional incremental risk.

Misc. Input>>

280	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B69		Local Governments	3-Section 3			Not Accepted

Comments>

The Exposure Assessment should discuss each chemical transport pathway and medium identified in the CSM. The discussion of the CSM figures is limited to one brief paragraph at the bottom of page 3-34, which is section 3.2.5. The CSM is central to the discussion of exposure, but is not used in this entire section.

Response>>

The CSM figures are primarily discussed in Section 3.2.1, page 3-24. The discussion on page 3-34 is supplemental. However, the bulk of the fate and transport discussion is in the RI portion of the Basin RI/FS documents and it was not the intent of the HHRA to reproduce those details in the report.

Misc. Input>>

281	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B70		Local Governments	3-Section 3	p.3-21,3-22, 3-28 through 3-34		Not Accepted

Comments>

The subsistence lifestyle discussion on pages 3-21,3-22 and 3-28 through 3-34 requires separate CSMs to ensure the public understands subtle differences.

Response>>

We are not clear what "subtle differences" need to be addressed in separate CSM figures for residential and subsistence receptors. We consider the CSMs to sufficiently identify the source of contamination, exposure route, and exposure pathway for each receptor type. The discussions in section 3 mostly distinguish between differences in exposure durations and contact rates, neither of which are appropriately depicted on CSM figures.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
282	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B71		Local Governments	3-Section 3	Figures 3-3 through 3-11		Not Accepted

Comments>

These figures require modification to meet human health risk assessment objectives and be consistent with EPA human health risk assessment guidance. CSM developed on geographical or ecological considerations are not appropriate for evaluating human health. Currently the CSMs include information that is not discussed in the report, resulting in very complex CSMs. It is recommended that all information in the figures to the left of the Affected media and Secondary Sources "column" should be removed, with the exception of waste rock piles, which should be include as discussed below. While information included in the CSMs may be useful in the RI Report or during sample collection planning (DQO Process) for the RI/FS, it is not useful in human health risk assessment. Only information that is discussed in the Report should be included in the Figures. If this information is "required", all of the "boxes and arrows" should be discussed in sufficient detail for the public to understand why each "box" is included.

Response>>

We acknowledge that the fate and transport section of the CSM figures contains a great deal of information and that the text summary provided in Section 3.2.1 is relatively brief. We disagree that the figures require modification per EPA risk assessment guidance and no citation as to what specific guidance is being referred to was provided by the reviewer. HHRA guidance (USEPA, 1989, RAGs Part A) does not make any references to the complexity of information that should or should not be reproduced in an HHRA but simply requires that human health pathways be shown from source to receptor. The CSM figures adequately perform this task.

Misc. Input>>

283	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B72		Local Governments	3-Section 3	Figures 3-3 through 3-11		Not Accepted

Comments>

Media that are evaluated in the risk assessment should be in the "Affected media" column, which would be more clearly labeled "source media" for risk assessment.

Response>>

Comment noted. We are leaving the media evaluated in the risk assessment in the "exposure routes" column as sufficiently clear for the purposes of this risk assessment.

Misc. Input>>

284	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B73		Local Governments	3-Section 3			Partially Accepted

Comments>

Data were collected for upland soil in the lower basin, but that medium is not connected to a receptor in the Figure. However, the risk assessment evaluates those data for exposure to recreational receptors.

Response>>

The arrow is missing that connects the "upland soil & flood plain deposits" box under affected media to the soil/sediment box under exposure routes on Figure 3-3. This error will be corrected. See the response to Comment B2 and B4.

Misc. Input>>

285	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B74		Local Governments	3-Section 3			Not Accepted

Comments>

The media being evaluated in the risk assessment includes residential soil, mining waste rock, Upland soil (0 to 1 inch) sediment (0 to 6 inches), sediment (0 to 12 inches), tap water, surface water, groundwater, surface water with "stirred" sediment, homegrown vegetables, fish, and household dust (there may be others). The CSMs indicate that household dust originates from wind erosion of tailings and "concentrates and other wastes". However, the risk assessment assumes the house hold dust is equal to yard soil concentrations. The tailings, concentrates and other waste sources were not analyzed statistically in Section 6.4.1 to validate the CSM assumptions. Because the CSM is a document that changes with additional data collection, it appears that the pathways should be confirmed statistically or removed from the CSM as significant pathways. The risk assessment assumes that household dust is equivalent to residential yard soil. The CSM is not consistent with the pathways evaluated in the risk assessment.

Response>>

The CSMs in Section 3 all show that the sources for household dust are residential soil and fugitive dust. It is true that the sources for fugitive dust are listed as "concentrates and other wastes" and "wind erosion." However, fugitive dust is also listed as an insignificant pathway, hence our statement that soil is the major source of chemicals in house dust.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
286	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B75		Local Governments	3-Section 3	Table 3-19		Not Accepted

Comments>

The information included in, Table 3-19 identifies the receptors, exposure media and exposure points, but this information is not clearly included in the CSMs. The CSMs would communicate issues more clearly to the public, if they contained only the information included in Table 3-19 (plus transport pathways, release mechanisms and incomplete pathways, as appropriate).

Response>>

We disagree. Although Figures 3-3 - 3-11 are more detailed than one typically sees in a risk assessment, the risk assessment team considers these figures to appropriately reflect the information that is contained on Table 3-19. See also response to Comment B71.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
287	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B76		Local Governments	3-Section 3	Table 3-20		Partially Accepted

Comments>

Appendix E does not provide residential chemical data and therefore, the exposure point concentrations provided in Table 3-20 cannot be confirmed. This omission of data does not meet EPA's policy of clear and transparent risk assessment reports. How does the public confirm that the exposure point concentrations have been calculated appropriately?

Response>>

Appendix F contains the residential data. Text will be reviewed and may be amended to clarify the location of the data.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
288	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B77		Local Governments	3-Section 3	Table 3-21		Accepted

Comments>

Table 3-21 indicates that 13 homes were sampled for the risk assessment for the Lower Basin. However, Appendix E Table "Summary of Site Data by Geographical Area for the Baseline Risk Assessment" identifies four residential locations: R007, R025, and R115 in Cataldo, and R144 in Harrison.

Response>>

The Appendix E Table only included EPA data. The table will be corrected. See previous responses on this issue.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
289	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B78		Local Governments	3-Section 3	Table 3-22		Partially Accepted

Comments>

Table 3-22. Residential Exposure Factors. The reasonable maximum exposure (RME) frequencies for soil ingestion, dermal contact with soil and ingestion of drinking water are 350 days/year. However, the exposure frequencies are 260 days per year for soil ingestion and dermal contact, and 234 days per year for ingestion of drinking water in the central tendency exposure scenario. This is not consistent. If a resident is at home eating yard soil, they are likely drinking household tap water.

Response>>

The methods used to arrive at the CT estimates for these two pathways were different and do not imply that people are in their house for different numbers of days. The soil CT estimate is calculated based on the ground being frozen for that period of time while the water CT value assumes that people spend 8-hours a day away from home (the water value is 2/3 the RME value of 350 days). This issue will be clarified in the text of Section 3.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
290	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B79		Local Governments	3-Section 3	Table 3-22		Not Accepted

Comments>

Note that 260 days/year exposure frequency for the central tendency scenario is due to snow covering the ground and/or frozen soil that is resistant to sticking to the skin dermally. It is not clear, why this site-specific information does not modify the default RME exposure scenario. Snow cover and frozen ground are reality. The RME scenario becomes a "worst case" exposure scenario when the site-specific factors are not included. Worst case scenarios are not in agreement with EPA risk assessment policy. It is recommended that the RME exposure duration for incidental soil ingestion and dermal contact with soil is 260 days and the average scenario would be less.

Response>>

Exposure to soil both by ingestion and dermally continues during the winter inside the home because soil is still tracked into the house for much of the winter; however, it is not clear how much reduction would occur. Therefore, the RME scenario did not adjust contact downward for winter while the CT scenario assumed no contact. These two assumptions likely bound the actual amounts ingested/absorbed. See also response to Comment A19.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
291	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B80		Local Governments	3-Section 3			Not Accepted

Comments>

The Report proportions water intake between adults and children over the 30 year period they are assumed to live in one location. This is similar to the proportioning of soil intake where is assumed the child exposure is for 6 years and the adult exposure is for 24 years...However, proportioning water intake between adults and children is not supported by EPA risk assessment guidance. The Report supports proportioning of water intake to be "consistent" with EPA' soil ingestion policy. The EPA policy on soil ingestion is based on documented higher soil intakes and lower body weights for children compared to adults. The ratio of water intake to body weight for children and adults is relatively uniform compared to incidental soil ingestion intake. In addition, water intake for children is not well characterized. Please provide documentation of the rationale to combine adults and children for water intake. The discussion should evaluate the uncertainty associated with estimated child drinking water intake. Should water intake proportioning be included in a revised draft risk assessment, the IEUBK default water ingestion rates for children aged 6 months through 6 years, which ranges from 0.2 to 0.59 liters/day, should be discussed and considered in the Report.

Response>>

Proportioning water intake is used by EPA Regions 9 and 3 when developing PRGs. The HHRA followed those protocols. The EPA PRG calculations reflect the most recent information on childhood exposures, an on-going area of research.
We disagree that there is uniformity of water intake rates in children vs. adults. Children are known to consume water at a higher rate, regardless of how water needs are indexed . The caloric requirement per unit body weight is higher than in adults, and water intake is linked to caloric requirements and physical activity. Generation of more water intake is also indicated by ventilation rate and oxygen intake requirements. We refer the commenter to the paper by Calderon and colleagues in a 1999 EHP article dealing with age-based water intakes in the US and, because of this, increased arsenic intakes in children as a function of body mass. It show US children consume much more water than adults on a body mass basis. We also refer the commenter to the EPA ODW/OGW 6/22/00 Federal Register notice [[65(121)FR38888, 2000]], on proposed arsenic MCL rulemaking, which includes statements that the early , bottle-feeding infant age band is a clear risk group for arsenic is because of the high daily water volume intake per body mass. These two citations and many others, such as the 1984 EPA health assessment document for arsenic, show that there is an inverse relationship between water volume intake and unit body index, e.g., kg body mass, such that the younger the individual, the higher the intake rate.

Ref: Calderon RL, Hudgens E, Le XC, Schreinmachers, Thomas DJ. Excretion of arsenic in urine as a function of exposure to arsenic in drinking water. Environ. Health Perspect. 107:663-667 (1999).

Misc. Input>>

292	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B81		Local Governments	3-Section 3	Table 3-23		Partially Accepted

Comments>

Table 3-23, Neighborhood Recreational Exposure Factors. There are inconsistencies in the exposure durations for soil ingestion and dermal contact. Incidental soil ingestion occurs by hand-to-mouth contact. Children and adults eat dirt by licking their fingers. If dermal contact did not occur, incidental soil ingestion would not occur. However, if dermal contact occurs then it is assumed that incidental soil ingestion occurs. The point of this discussion is explained below for the RME scenario. The Waste Pile exposure parameters assume 17 days for soil ingestion, but 34 days for dermal contact. The Upland Soil ingestion exposure parameters assume 34 days for soil ingestion, but 68 days for dermal contact. The Floodplain exposure parameters assume 21 days for soil ingestion, but 96 days for dermal contact and 96 days exposure to surface water. The report needs to explain how the receptor can be dermally exposed to soil and not experience hand-to-mouth, incidental, soil ingestion. The current exposure parameters overestimate dermal exposure by a factor of two compared to soil ingestion intake.

Response>>

See response to Comment B20.

Misc. Input>>

293	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B82		Local Governments	3-Section 3	Table 3-23		Accepted

Comments>

A typographical error in Table 3-23; the exposure duration for the central tendency ingestion of surface water should be 48, not "I".

Response>>

The table will be amended.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
294	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B83		Local Governments	3-Section 3	Table 3-24		Partially Accepted

Comments>

Table 3-24, Public Recreational Exposure Factors. The same incidental soil ingestion/dermal exposure frequency issue is present for these receptors. For the RME scenario, for example in Parks and Schools, the soil ingestion exposure duration is 34 days each year and the dermal exposure duration is 68 days per year. The floodplain soil/sediment ingestion exposure durations are identical at 32 days, which is consistent.

Response>>

See response to Comment B20.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
295	10/03/2000	R. Merril Coomes	Public Draft - July 2000	5.2	0/31/2000	URS
B84		Local Governments	5-Section 5			Partially Accepted

Comments>

Section 5.2 Methodology for Assessing Cancer Risk.

The last paragraph is confusing and does not appear to agree with EPA's risk assessment guidance and the National Contingency Plan (NCP). The paragraph indicates that cancer risks within the 1.0E-06 to 1.0E-04 (one in 10,000 to 1 in 1,000,000) range will be further evaluated in the FS where risk management decisions will be considered. The reviewer cannot locate this interpretation in the NCP or EPA guidance. The NCP indicates that the goal for cancer risk, when a site is remediated, is the range of 1.0E-06 to 1.0E-04 and the point of departure is 1.0E-06. OSWER Directive (Use of Risk Assessment in Superfund Remedial Actions) states that when the cancer risk is less than 1.0E-04, remediation is not generally warranted unless there is evidence of harm to the environment (EPA OSWER Directive 93355.0-30). The risk characterization section discusses the relationship between estimated cancer risk and the "acceptable cancer risk range". This comparison should be removed from the risk characterization section because this range is only appropriate when discussed as a goal for the cleanup of an area that is being remediated. EPA guidance states that remedial action is not warranted unless the risk is greater than 1.0E-04 (assuming there are acceptable environmental effects). The discussion should compare estimated cancer risk to a risk of 1.0E-04, to be in agreement with EPA guidance and policy. If there are conflicting EPA guidance documents that discuss this issue, please incorporate all guidance in the discussion.

Response>>

Remedial activities may be warranted if risks are less than 10E-4 per both the NCP and the OSWER directive. The commenter notes that the goal of the NCP is cancer risks "within" the 10E-6 to 10E-4 range. The text of the OSWER directive states that "when the cancer risk is less than 1.0E-04, remediation is not generally warranted unless there is evidence of harm to the environment (EPA OSWER Directive 93355.0-30)." The directive includes the word "generally" in this statement to allow for site-specific decisions to be made. The text will be clarified regarding EPA guidance and target risk goals.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
296	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B85		Local Governments	6-Section 6			Not Accepted

Comments>

Using the Integrated Exposure Uptake Biokinetic (IEUBK) Model to Estimate Periodic Exposure Scenarios
A concern about the methodology used to evaluate blood lead levels using the IEUBK Model is modifying the model to accommodate intermittent exposure. EPA states:

"The IEUBK Model was designed for application to exposure scenarios in which there are long periods of relatively steady exposure, not to acute or relatively rapid sub-chronic exposure scenarios, so that only the slowest transfer compartments affect kinetics on the time scales of interest."(EPA 1994a, page 9-16, Report Reference Section 9.0).

The Report simply identifies this issue but does not explain the rationale that supports using the Model although this limitation is clearly stated. It is recommended that a discussion be included in the Report identifying the limitations of the IEUBK Model when attempting to evaluate periodic exposure.

Response>>

See response to Comment B22 and General Response to Comments, #9a, #5a, and #5b.

Misc. Input>>

297	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B86		Local Governments	6-Section 6			Not Accepted

Comments>

Lead Intake from Other Sources

OSWER Directives 9355.4-12 and 9200.4-27P indicate that other lead sources should be evaluated. The directives identify interior and exterior lead based paint, air, water, interior lead-based paint etc. The Directives also indicate that sources that may contaminate soil if the soil were remediated should be considered. It is not clear that the Directives provide guidance to evaluate sources of lead that are "remote" from the yard. For example, beach soil/sediment for campers and water sports does not appear to have high potential to contaminate yard soil for much of the Basin. It is not clear that it is appropriate to combine these sources with residential yard evaluation. This issue should be addressed in the Report. It is clear that specific areas (beaches) should be evaluated on their own to determine the potential for unacceptable exposure.

Response>>

Two types of soil exposure outside the home yard are considered in the lead portion of the HHRA. Community-wide soils are considered in the Box Model as part of the residential baseline. These are soils in the neighborhood or larger community that children access in the course of their everyday activities. Other soils outside the residential baseline are considered as incremental exposures. Incremental lead intake rates refer to the amount of lead taken into the body during activities in which only certain members of the population engage. These individuals either consume more soil, dust, water, or food, than the general population or those media have higher lead content. These activities take place outside of the baseline residential environment. Both the consideration of soils from sources other than the yard and the analysis of incremental exposures is consistent with OSWER guidance. These items are reviewed in the Technical Review Workgroup for Lead comments. The Technical Review Workgroup for Lead endorses the methodology but believes incremental exposures are underestimated due to the averaging time used in the analysis. See also General Response to Comments, #3, #5, and #8.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
298	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B87		Local Governments	6-Section 6	Table 6-32b, 6-33b		Not Accepted

Comments>

The calculation of yearly average lead intake overestimates the annual daily lead intake for receptors. An example is explained below for the soil ingestion intake for children at upland parks. Table 6-33b calculates the child intake from these potential source areas for the Reasonable Maximum Exposure scenario. The exposure scenario for the Upland Parks indicates that a child would be present two times each week for 34 weeks. This is a total exposure of 68 days in one year. The Upland Parks calculation in Table 6-33b for lead at 6,000 mg lead per kg of soil (mg/kg) shows a daily intake of 257 ug (micrograms)/day. This value is used to estimate the intake averaged over the year, which is 168 ug/day in the table. An annual intake of 168 ug/day actually assumes that the number of days at the Upland Park was approximately 238 or 7 days/week for 34 weeks in the year. The correct formula to estimate the average daily lead intake on an annual basis is:

Average Annual Daily Lead Intake (µg/day)Ave = Daily Intake (µg/day) X Number of Days at Upland Parks per Year Divided by the Number of Days in a Year

Because the child's daily lead intake, at 6,000 mg lead per kg of soil (mg/kg) is 257 ug/day and the exposure is 68 days per year at the Upland Park area, the average annual daily lead intake is:

Average Annual Daily Lead Intake = (257 µg/day) * (68 days at Upland Parks/Year) divided by 365 days/year = 47.9 micrograms lead per day (ug/day).

This value is more than three times less than the 168 ug/day given in Table 6-32b. Similar overestimates are given in the tables for beach sediment, waste piles, and neighborhood sediment. For example, the average annual lead intake for waste piles, assuming 6,000 mg/kg lead, is 2.4 ug/day, rather than 17 ug/day given in Table 6-32b.

Response>>

The HHRA disagrees with this comment as it is inconsistent with the methodology employed in the HHRA. The Reasonable Maximum Exposure (RME) Frequency is a 7 hour/day event, two-days per week, for 34 weeks. This totals 476 hours of exposure or 34 equivalent days at 14 waking hours per day. The ingestion rate while engaged in these activities is 300 mg/day. A total of 300 mg/day x 34 days = 10200 mg (10.2 grams) of soil ingested per season or year. At a concentration of 6000 ug/g, (6000 ug/g x 10.2 g) = 61200 ug Pb per year, or 168 g/day as reported in the HHRA. The TRW comments at Section 2.5 provide additional discussion regarding this topic. The Technical Review Workgroup for Lead concludes that the exposure duration is sufficient to include in IEUBK analysis, but believes the risk may be understated, by about 35%, as the exposure should be averaged over 238 days, rather than 365 days. See also General Response to Comments, #5a and #5b.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
299	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B88		Local Governments	6-Section 6			Not Accepted

Comments>

Statistical Analysis and Observations

Paint condition is correlated with blood lead, but paint condition appears to be a qualitative variable and not a continuous function. How this affects the analysis and predictability of the correlation should be explained in the Report.

With the large number of samples, it is not surprising that correlation coefficients are statistically significant. However, the calculated correlation coefficients and regression relationships do not imply a causal relationship between the variables. The regression relationships at best explain only half of the variance in the data (R2 = 0.63 2 = 0.49).

The correlation coefficients may be statistically significant (i.e., the confidence interval does not include zero), but the "r" values suggest that the resulting predictive relationship is not significant, because the values are so low (0 < r < 05). In addition, it appears that for a least a few of the correlations, a few high values drive the correlation, which results in relatively high correlation coefficients (for example blood lead (BLBP) versus lead loading rate (LEADLD). It is obvious that, individually, the relationships do not adequately explain variability in blood lead level. Further, because the correlations are so low, none of the resulting regression equations is appropriate for predictive purposes.

Regressions performed using median values for the x-variable should be examined carefully. The Report should discuss whether the median value is a good predictor of the variable it represents.

Correlations in log-space are appropriate only so long as the variables remain as logarithms. If any of the relationships are retransformed to base 10 the correlation coefficient, equation, and other statistics need to be transformed carefully. The relationships cannot be simply transformed.

Overall, the fact that many of the correlations are statistically significant is not a compelling argument. The small r-values, highly scattered data, and low predictive capability of the resulting relationships suggest that the individual variables evaluated are not good predictors for lead blood level.

Response>>

The HHRA disagrees with this comment. The relationship between blood lead levels and environmental exposures is examined throughout the HHRA by a variety of methods. With respect to blood lead levels, regression analysis indicated that dust lead loading rate alone explained nearly 40% of the variation in the dependent variable. Other environmental variables were significant in combination with dust lead loading rate. Those variables were yard soil lead levels, median exterior paint XRF reading, and interior paint condition. Together with age of the child, these variables explain 60% of the variation in blood lead levels. It is well established in the lead health literature that there is an inherent variance in blood lead response among individuals in a population. Considering that this regression model does not address this inherent variance, accounting for 60% of the variation in observed blood lead levels must be considered a strong relationship. See also General Response to Comments #4a and #3a through #3d.

Misc. Input>>

300	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B89		Local Governments	6-Section 6			Not Accepted

Comments>

The multiple regression analysis, which evaluates five variables simultaneously, is somewhat stronger. The analysis of five variables explains 60 percent of the variability in the blood level data is explained with the regression equation. However, 40 percent of the variance remains unexplained.

Response>>

The HHRA disagrees with this comment. See response to Comment B88.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
301	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B90		Local Governments	6-Section 6	p.6-20, Figures 6-10a-d		Not Accepted

Comments>

It is not clear that the implications of the statistical assumptions and limitations that have been made in the statistical analyses in the Report would be clear to the public.
To minimize confusion between the discussion of correlation for the various parameters discussed on page 6-20 and plotted in Figures 6-10a through 6-10d, it is recommended that the plots contain the correlation coefficient and the line representing the relationship.

Response>>

The HHRA disagrees with this comment. The correlation coefficient (r) can be added to the scatter diagrams, although this is not typically accomplished for presentation of simple correlations and scatter plots and the r values can be found in Tables 6-19 and 6-20. Adding a line to represent the relationship between these variables would more typically be a regression line. However, this may not be appropriate for multiple regression analysis as many of these individual relationships may be secondary in nature.

Misc. Input>>

302	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B91		Local Governments	6-Section 6	p.6-30		Not Accepted

Comments>

Page 6-30, Summary Baseline Intake Rates. Explain the rationale for using only the 4-year old child data. The IEUBK Model identifies the intake for each year of age. If this discussion provides meaningful input to the risk assessment, it would be clearer to discuss the entire spectrum of intake than to judgmentally select a specific age group.

Response>>

Four year old intake rates are provided as an example representing the mid-range of age-specific estimates. Other age group estimates are shown in Appendix P and are used in all age-specific analysis in the HHRA.

Misc. Input>>

303	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B92		Local Governments	6-Section 6	p.6-32		Accepted

Comments>

Page 6-32, third complete paragraph, discusses the “seasonal” and annual intake of lead and refers to Tables 6-29a-b. The descriptor seasonal is not included on the tables, making interpretation difficult. The text refers to medium exposure rather than central tendency, and this is easily confused with the “medium” to which the receptor is exposed.

Response>>

In the incremental exposure analysis for lead, intakes were estimated for seasonal activities and averaged over the year for input to bio-kinetic models. The EPA TRW review disagreed with this approach in Section 2.4 and 2.5 of the TRW review. The TRW believed that the increment should be averaged over the season. This results in risk being underestimated, according to the TRW, by about 35%. See also General Response to Comments, #5a and #5b. The term medium exposure is potentially confusing and does refer to Central Tendency estimates. The terminology will be modified in the final document.

Misc. Input>>

304	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B93		Local Governments	6-Section 6	p.6-31, first para		Not Accepted

Comments>

Page 6-31, first paragraph, provides details about adult blood lead levels and presents data in a table, but then states that the data are not used. Eliminate this table and discussion from the text, and simply state that actual measured blood lead levels are used (Table 6-8b).

Response>>

The table referred to in this comment (Table 6-26) shows adult baseline lead intake rates and not blood lead levels. This table and discussion was added to the HHRA for comparison purposes and to establish that most adult soil/dust intake would be coming from dust.

Misc. Input>>

305	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	TG
B94		Local Governments	6-Section 6	p.6-31		Accepted

Comments>

Page 6-31 under Occupational Intake Rates identifies three classifications of exposure, nominal, medium, and intensive, and then goes on to state that nominal exposure scenarios are not evaluated in this risk assessment. This is confusing because risk assessments are based on central tendency and reasonable maximum exposure scenarios. Please revise text to conform to EPA exposure scenario definitions.

Response>>

See response to Comment B92.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
306	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B95		Local Governments	0-Executive Summary			Not Accepted

Comments>
Create two CSMs for the lower Basin, one for the subsistence exposure scenarios and one for other receptors

Response>>
See response to Comment B70.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
307	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B96		Local Governments	0-Executive Summary			Not Accepted

Comments>
Add waste piles to the risk assessment "source media"

Response>>
Soil in waste piles is already on CSM figures 3-9 and 3-10 (the areas where it was evaluated) as an exposure pathway and the box is connected to various source media such as "waste rock" "tailings", etc. We do not think its addition to source media is necessary.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
308	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B97		Local Governments	0-Executive Summary			Not Accepted

Comments>
Fugitive dust (as a source medium) has a release mechanism – wind erosion – to provide the rationale for an inhalation intake route.

Response>>
Wind erosion is listed on the CSMs as a release mechanism leading to fugitive dust which is inhaled.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
309	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B98		Local Governments	0-Executive Summary			Not Accepted

Comments>
Use the conceptual site models in the Report that were used to design data collection. The conceptual site models shown in Figures 3-3 through 3-11 do not agree with conceptual site models used in the July 24, 1998 Field Sampling Plan, Addendum 05. For example, there is not a common use area conceptual site model in the Report, but Figure 1 in the FSAP, addendum 5 clearly shows the chemical transport and receptor relationships used to plan data collection. The CSM shown in Figure 3-4 of the Report and Figure 2 of the FSAP are significantly different, although they have the same objective, which is to collect data and use the data to estimate potential risk for recreational beaches.

Response>>
See previous response to Comment B2 on this issue.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
310	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B99		Local Governments	0-Executive Summary			Not Accepted

Comments>
Discuss the details of the conceptual site model in the text. The text should discuss chemical transport pathways, define exposure points, define the exposure area, and discuss intake routes. This text supports the figure and clearly explains exposure assessment issues to the public.

Response>>
See response to comment B69.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
311	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B100		Local Governments	0-Executive Summary			Partially Accepted

Comments>
Deviations from planned data use must be explained in the Report. The planned data use for the "wet" sediment was apparently to evaluate potential exposure to waders and swimmers (FSAP, Addendum 05). The planned data use for soil data, identified as unvegetated Beach/Playground in FSAP, Addendum 05, was to evaluate potential exposure to visitors and campers.

Response>>
The HHRA working group made the decision to evaluate only one recreational receptor rather than several different receptors for these areas. The assumption used was that the receptor would spend equal amounts of time exposed to the sediment and soil. This assumption is considered to be adequately protective given the exposure parameters used to quantify Lower Basin recreational risks. See response to Comment B2.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
312	10/03/2000	R. Merril Coomes	Public Draft - July 2000		0/31/2000	URS
B101		Local Governments	0-Executive Summary			Not Accepted

Comments>

Discuss and define exposure area(s) in the Report for all receptors.

A more detailed analysis of potential risk by exposure area is recommended in the Lower Basin. EPA guidance relates the exposure area to the Scale of Decision Making for surface and subsurface soil (EPA 1996). This means that the risk assessment results contribute to making a decision to select a remedial alternative over a defined area (scale of decision making). These concepts are not identified in the Report and this omission does not allow the risk manager to make an informed and defensible decision.

Response>>

See previous responses on this issue, particularly Comment B13 and B14.

Misc. Input>>

The Lands Council

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
127	10/16/2000	Michelle Nanni	Public Draft - July 2000		0/30/2000	URS
C1		The Lands Council	0-Executive Summary			Accepted

Comments>

First of all, we support that the assessment addresses current as well as FUTURE potential health risk scenarios in the basin.

Response>>

No response required

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
128	10/16/2000	Michelle Nanni	Public Draft - July 2000		0/30/2000	URS
C2		The Lands Council	0-Executive Summary			Accepted

Comments>

We also support the approach of evaluating health risks to both typical Central Tendency (CT) as well high risk Reasonable Maximum Exposures (RME) populations in the basin, including tribal/subsistence populations. We believe that health risks should be remedied and decreased for both the general population as well as those most exposed and at risk.

Response>>

No response required.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
129	10/16/2000	Michelle Nanni	Public Draft - July 2000		0/30/2000	TG
C3		The Lands Council	0-Executive Summary			Partially Accepted

Comments>

With respect to existing blood lead data - gathered throughout the basin in 1996 by the Idaho Department of Health and Welfare and federal ATSDR, along with subsequent blood lead data gathered between 1996 and 1999 - it is apparent that there are significant public health impacts occurring in the basin due to heavy metals contamination. Also, given that the assessment states that approximately 25% of eligible children in the basin participated in the surveys, it appears that the blood lead data set provides an adequate foundation, at this time, for determining health risks and potential remediation scenarios.

Response>>

The HHRA agrees with this comment. The existing blood lead data base is the most reliable information for current risk assessment purposes. However, there are legitimate concerns as to whether these data are representative of non-participants. See General Response to Comments, #2a and #3b.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
130	10/16/2000	Michelle Nanni	Public Draft - July 2000		0/30/2000	TG
C4		The Lands Council	0-Executive Summary			Not Accepted

Comments>

Therefore, based on the existing blood lead data and goals of the assessment, The Lands Council supports the use of the stricter soil cleanup levels for lead (400 mg/kg) estimated by the EPA Default Model, versus the Bunker Hill Superfund Site (BHSS) "Box Model," based on the following points:

* The assessment presents strong reasons why the blood lead data set may actually UNDER-estimate wider childhood exposures in the basin, which justifies a more conservative cleanup level to be achieved in the basin;

* It is unlikely that the consistent high rates of lead poisoning measured among children are due to repeat testing of the same children year to year, because health intervention measures usually correct the problem after a high blood lead level is detected ? therefore further supporting the data set and more protective cleanup levels;

* The assessment concludes that background or pristine environmental concentrations would be required for all media to safely support Native American subsistence activities;

* The assessment concludes that soils with lead concentrations near 500 mg/kg could result in a greater than a 5% probability for blood lead levels higher than 10 µg/dl, for intensive/RME exposures relating to landscapers, farmers, and agricultural workers, as well as remediation and construction workers;

* The BHSS "Box Model" uses site-specific data based on 1000 mg/kg residential yard cleanup level for lead, but a 350 mg/kg COMMUNITY-WIDE average overall for soils - which is contrary to the cleanup levels proposed by the Box Model for the rest of the basin in this assessment;

* The assessment shows that the Box Model underpredicts actual blood lead levels for most geographical sub-areas in the basin, particularly the Lower Basin area.

In summary, given the above points and the strong evidence of high rates of health impacts occurring in the basin, we strongly urge that the more conservative health cleanup thresholds predicted by the EPA Default Model be applied in the basin for remediation purposes. For the reasons stated above, we do not find sufficient evidence that the Box Model predictions and recommendations would provide adequate protection for public health in the basin ? for present or future populations.

Response>>

The HHRA disagrees with this comment. It is not known if the available blood lead data over-estimate, under-estimate or accurately portray the non-participants in the health surveys. The Box accurately predicts observed blood lead levels in the upper Basin. The Box Model has performed well at the BHSS, and there is reason to believe that similar pathways and dose/response relationships could apply in the upper Basin. Risk managers should not discount the potential applicability of this model in their consideration. See also General Response to Comments, #2a, #3b, #4a and #9.

Misc. Input>>

Shoshone Natural Resource Coalition (SNRC)

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
109	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D1		SNRC	8-Section 8	p.8-3,8-4		Partially Accepted

Comments>

1. In the Summary and Conclusions, pages 8-3,4:

The presentation of economic data overemphasizes tourism and understates the importance of mining in the economy of Shoshone County. The reported 58% decline in mining employment between 1990 and 1996 reported does not reflect the 250 jobs created at Coeur Silver Valley Operations and the Lucky Friday Mine between 1995 and 2000. Mining is still responsible for 40% to 50% of the economic activity in Shoshone County. Tourism presents an opportunity to increase the economic base and should not be portrayed as a replacement of industrial wage employment.

Response>>

The HHRA agrees with this comment.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
110	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D2		SNRC	8-Section 8	p.8-3,8-4		Partially Accepted

Comments>

Also we would like to note the difficulty of growing any business segment during a 25 year Superfund project. The State approach of a 25 to 30 year Public Works Project will be much more beneficial to development of a local economy capable of maintaining the remedy.

Response>>

The HHRA agrees with this comment.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
111	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D3		SNRC	8-Section 8	p.8-5		Partially Accepted

Comments>

1. In the Summary and Conclusions, page 8-5:

- In the July 1999 follow-up of 50 children with blood lead > 10 ug/dl: How many (%) of the children's homes were built before 1960 and before 1940?

Response>>

The July 1999 nurse visit follow-up targeted 28 children (see Table 6-1), with 25 completed because three children moved out of the area. There were a total of 58 children targeted for follow-ups, and 50 follow-up surveys completed for all years combined. Of those 25 completed in 1999, 11 (44%) of the children lived in homes built before 1960, and of those 11, 7 (28%) lived in homes built before 1940. The reasons for high blood leads for those 11 children were mostly attributed to high soil and dust and not necessarily due to lead based paint.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
112	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D4		SNRC	8-Section 8	p.8-5		Not Accepted

Comments>

1. In the Summary and Conclusions, page 8-5:

- In the July 1999 follow-up of 50 children with blood lead > 10 ug/dl: What was the correlation between yard soil, house dust and blood lead?

Response>>

Using the 58 blood lead observations above 10 ug/dl, there was no significant correlation with yard soil lead concentrations. This result is expected as this subset of the population represents children with high blood lead levels and high soil lead exposures. Correlations with other variables showed some significance, however, the number of matching observations was very low (n is less than or equal to 20). This sample size is too low to yield statistically meaningful estimates.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
113	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D5		SNRC	8-Section 8	p.8-5		Partially Accepted

Comments>

1. In the Summary and Conclusions, page 8-5:

- In the July 1999 follow-up of 50 children with blood lead > 10 ug/dl: What was the mean income of the families?

Response>>

The survey was a health based survey that was used to help identify possible exposure pathways. Family income level was not a question asked during the survey.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
114	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D6		SNRC	0-Executive Summary	P.8-5		Partially Accepted

Comments>
The HHRA and Risk Managers need to pay more attention to social economic factors than a traditional Superfund project, the end goal must be a Lead Safe community, not necessarily a lead free community (see page 3 of the executive summary).

Response>>
The HHRA agrees with this comment.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
115	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D7		SNRC	8-Section 8	p.8-4		Partially Accepted

Comments>
The Silver Valley has twice as many pre-1940 housing units as the State average (see page 8-4 of the Summary and Conclusions)

Response>>
The HHRA agrees with this comment.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
116	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D8		SNRC	8-Section 8			Partially Accepted

Comments>
The percentage of children living in poverty is twice as high as the state average

Response>>
The HHRA agrees with this comment.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
117	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D9		SNRC	8-Section 8			Not Accepted

Comments>
The Silver Valley demographics are similar to national demographics associated with childhood blood lead levels 3 times the clean-up goals.

Response>>
The HHRA disagrees with this comment. It is not possible to make direct comparisons between the Silver Valley and demographic stratifications included in the NHANES national blood lead data base. It is, however, clear that demographic and socio-economic factors play an important role in the degree and incidence of lead poisoning in both the Silver Valley and the nation. See the discussion in Section 6.2.2 of the HHRA. See also General Response to Comments, #1a.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
118	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D10		SNRC	8-Section 8			Not Accepted

Comments>
A traditional Superfund approach with the focus on yard removal is much too narrow in scope to make the Valley lead safe. The HHRA uses a simplistic model to justify what appears to be a predetermined conclusion that yard removal is the exclusive answer to elevated blood lead levels in children.

Response>>
The HHRA disagrees with this comment. Analysis conducted in the HHRA suggest that yard soils are a primary source of lead absorption among children both through direct contact and as a contributor to lead in house dust. Other sources, including lead paint, are also identified as sources to both blood and dust lead. The HHRA concludes that both sources present excessive risk and provides example analysis regarding potential cleanup criteria.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
119	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D11		SNRC	8-Section 8	p.8-18		Not Accepted

Comments>
The testing data is not considered representative. Panhandle Health is trying to find children with highest blood leads for health intervention reasons, that's why they test in August when the highest levels are expected. (page 8-18) Blood lead levels are known to have a seasonal relationship and the highest occur in August when the sampling is scheduled

Response>>
The HHRA disagrees with this comment regarding the representativeness of the blood lead data. These data are believed to be representative of the participating population, as the peak seasonal blood lead period is purposefully sampled. It is unknown if these data are representative of those Basin residents who did not participate. See also General Response to Comments, #2a.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
120	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D12		SNRC	0-Executive Summary			Not Accepted

Comments>

The HHRA includes multiplying safety factors: All these safety factors combine and when applied to only one remedy, soil removal, require lower and lower action levels for soil, well beyond the level of diminishing returns.

Response>>

The HHRA disagrees with this comment. Lead health risk assessment applies fewer safety factors or lower margins of safety than typical sub-chronic non-carcinogenic risk assessment methods for other metals. The HHRA does not suggest a single remedy. See response to Comment D10.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
121	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	URS and TG
D13		SNRC	0-Executive Summary			Not Accepted

Comments>

- High end ingestion rates have been assumed for all scenarios
- Whole fish scenarios
- Shallow well scenarios

Response>>

The HHRA disagrees with this comment. These are reasonably expected scenarios for particular populations.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
122	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D14		SNRC	0-Executive Summary			Not Accepted

Comments>

Using the 10 ug/dl level for adult occupations (constructions, earthwork) when the OSHA standard is 30.

Response>>

Please see response to Comment B61 and General Response to Comments, #10.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
123	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	URS and TG
D15		SNRC	0-Executive Summary			Not Accepted

Comments>

Exposure to waste rock piles is greatly overestimated because of the way samples were collected, the lack of silt sized materials and the outrageously high assumptions of exposure time for children.

Response>>

Incremental exposure factors for waste piles do not distinguish among waste pile types and surface characteristics. Incremental intake rates were developed for both members of the population, one for the typical (Central Tendency (CT)) and one for the reasonable maximum exposure (RME). Estimating the intake rates is a relatively straight-forward procedure utilizing exposure factors developed elsewhere in the document. Generally, these factors are linear and intake estimates are proportional to exposure point concentrations, contact times, and exposure frequencies. Should risk managers disagree with the underlying assumptions or wish to consider alternative factors, the incremental intake rates can be adjusted accordingly. This option is discussed in more detail in General Response to Comments, #5 and #5a.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
124	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D16		SNRC	0-Executive Summary	p.ES-3		Partially Accepted

Comments>

In the executive summary (ES-3) it is inferred that 25% of the eligible children participated in the blood lead surveys when in fact 1999 is the only year that level of participation was even approached.

Response>>

The HHRA agrees with this comment. It is estimated that there are between 1000 and 1100 children from 9 months through 9 years of age in the Basin area. In 1999, 272 or slightly more than 25% of these children were tested. In four years, 424 of approximately 1300 eligible children have been tested at least once, or about 33%. In other years, less than 10-20% of children were tested. In the Bunker Hill Superfund Site participation rates of the eligible population have been estimated from 42% to 58% annually over the last decade. See also General Response to Commments, #2a.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
125	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D17		SNRC	0-Executive Summary			Not Accepted

Comments>

If the IEUBK model doesn't reasonably represent the observed data, it should not be used to set action levels. Why not use observed results? The action level used within the Bunker Hill Box is on track to achieve the clean up goals of less than 5% of the at-risk population >10 ug/dl and none above 15 ug/dl. Also note that the average yard lead levels in Kellogg, Wardner and Smelterville were 3 to 4 times higher than those observed outside the "Box" before remedial action.

Response>>

The HHRA disagrees with this comment. The Box Model effectively predicts both mean blood lead levels and percent of children to exceed 10 ug/dl in the upper Basin, in a manner consistent with its performance in the BHSS over the last decade. Risk managers could consider the Box Model appropriate to characterize risk in the Basin provided that similar pathways and dose-response relationships are involved and that the blood and environmental lead levels evaluated in the model are representative of the Basin population. There are questions as to whether the observed blood lead levels are representative of the overall Basin population. Site-specific regression analysis relating blood lead and environmental lead levels suggest similar pathways, with somewhat lower slope values for soil and dust concentrations in the Basin, compared to the BHSS. See also General Response to Comments, #9.

Misc. Input>>

126	10/15/2000	Kathy Zanetti	Public Draft - July 2000		0/30/2000	TG
D18		SNRC	0-Executive Summary			Partially Accepted

Comments>

Let us be clear and unequivocal. We live in the study area, and nothing is more important to the members of the Shoshone Natural Resources Coalition than the health and welfare of our families, friends and neighbors. We have generations of observations to support our conclusion that the vast majority of the residential areas outside of the Bunker Hill Box are safe, healthy places to raise a family. We simply can't reconcile the extrapolated, projected conclusion, with layer upon layer of safety factor, presented in the HHRA, with the reality of life in most of the Silver Valley.

We challenge you to look beyond the status quo approach and work with the community towards meaningful risk management.

Response>>

The HHRA recognizes the communities commitment to public health and agrees that the involvement and acceptance of the community is critical to the success of any risk management strategy that might be adopted. The HHRA disagrees that inappropriate "safety factors" have been applied. The methodologies employed for lead risk assessment are considerably more complex than those applied in non-carcinogenic risk assessment for other contaminants, and results in more precise, and less uncertain, estimates of effects than is typically obtained. As a result, lower margins of safety are employed in sub-chronic lead risk assessment than in the methods used for other metals. See also General Response to Comments, #9a.

Misc. Input>>

Citizens Against Rails-to-Trails (CART)

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
5	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E1		Citizens Against Rails-to-Trails	0-Executive Summary			Not Accepted

Comments>

The HHRA is a flawed document which states clearly that heavy metals contamination is very dangerous to human health, yet there is no data to support conversion of the contaminated Union Pacific right-of-way (ROW) into a recreational trail. In fact, HHRA data documenting dangers to human health from contaminants (lead, arsenic, cadmium, zinc) suggests that humans should avoid such exposure, since "safe" levels are difficult to determine, and would be, ideally, no exposure at all.

Response>>

The HHRA disagrees with this Comment. Consideration of potential recreational and occupational exposures associated with the Rails-to-Trails conversion are subsumed under the recreational and occupational scenarios considered in the HHRA. The types of activities anticipated for trail users and workers are accounted for in the scenarios addressed in the HHRA. An extensive discussion of demographics and land use is included in Section 3.1, additional characterization can be found in the RI/FS. See also General Response to Comments, #5.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
6	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E2		Citizens Against Rails-to-Trails	0-Executive Summary			Not Accepted

Comments>

Further, HHRA descriptions of resident population, land-use, and ownership are distorted, omitted entirely, or skewed to support use of the abandoned UP ROW as a recreational trail.

Response>>

See response to Comment E1.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
7	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E3		Citizens Against Rails-to-Trails	0-Executive Summary			Not Accepted

Comments>

Additionally, the HHRA is a confusing document, rife with double-talk and allusions to solutions that will be dealt with under "Risk Management" or to problems that will be assessed and cleaned up as they are discovered. We assert strongly that this remedy is totally unsatisfactory, particularly along the abandoned Mullan to Plummer UP ROW.

Response>>

See response to Comment E1.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
8	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E4		Citizens Against Rails-to-Trails	0-Executive Summary			Not Accepted

Comments>

The HHRA documents and underscores the inescapable, hypocritical paradox C.A.R.T. members have been asking EPA to explain for years: How can EPA demand rigid cleanup in the Superfund area and then invite the public to recreate on contaminated land to which they would otherwise not come?

We, members of C.A.R.T., continue to assert that our voices have not been heard, and our right to responsible CERCLA cleanup of contamination left by a known PRP, Union Pacific, has been blatantly compromised. The HHRA essentially denies C.A.R.T. members, as well as the general public, the right to protection of the public welfare guaranteed by EPA. Although the HHRA documents that it is, indeed, extremely dangerous for people (particularly children and pregnant women) to be near contaminants like lead, arsenic, cadmium, and zinc, the Governments continue to invite the public and Basin residents to recreate in a highly contaminated area. This is preposterous! The major fallacy, inviting the public to an area that they would otherwise avoid, is not addressed in the HHRA, nor has it been addressed in any previous documents endorsed by EPA. The increased contamination exposure risk to local residents inherent in the proposed recreational trail has not been adequately addressed. In short, the HHRA, like all other EPA sanctioned documents, circumvents the serious contamination caused by Union Pacific.

Response>>

The HHRA disagrees with this Comment. The government does not invite people to recreate on contaminated properties. The risk management plan adopted for the trail addresses the areas likely to be accessed on the right-of-way. Within 1000 feet of any residence the entire right-of-way will be provided with a clean surface. This addresses the nominal aspects of recreation associated with the residential scenario. At all major access points, sidings and select oasis locations are scheduled for a right-of-way-wide clean up, and large oases are strategically placed along the trail to provide clean rest and stop-and-view areas. In remote contaminated areas, warning signs will be posted to alert trail users of areas presenting excessive risk similar to warnings to avoid local hazards in numerous venues. The signage is provided to both advise users to avoid undesirable areas and to identify safe areas to recreate. See also General Response to Comments, #5.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
9	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E5		Citizens Against Rails-to-Trails	0-Executive Summary			Not Accepted

Comments>

The HHRA, further, relies upon hypothetical future scenarios while omitting reference to the critical danger involved in current scenarios which invite the public to recreate on land so highly contaminated that warning signs and other "institutional controls" must be posted along the proposed trail.

Response>>

The HHRA disagrees with this Comment. The government does not invite people to recreate on contaminated properties. The HHRA recognizes that public beaches and other common use areas throughout the Basin and including railroad right-of-way are routinely used by members of the public. That was one criteria for sampling these areas for the HHRA assessment. Incidents of excess lead exposure have been attributed to common use areas in the Lower Basin. There are numerous public access areas throughout the Basin that will be assessed in the development of a Proposed Plan for clean up. In remote contaminated areas warning signs will be posted to alert trail users of areas presenting excessive risk similar to warnings to avoid local hazards in numerous venues. The signage is provided to both advise users to avoid undesirable areas and to identify safe areas to recreate. See also General Response to Comments, #5.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
10	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E6		Citizens Against Rails-to-Trails	0-Executive Summary			Not Accepted

Comments>

The inadequacy of signs has been acknowledged by the Governments, yet this "control" is expected to keep people on the 10-foot wide strip of asphalt, away from the unremediated contamination bladed off to either side. This contamination will be redistributed during seasonal flood events. Airborne distribution, through dust particles, will further expose the public to contaminants.

Response>>

The HHRA disagrees with this Comment. The governments believe the signage proposed for the trail to be adequate. Contaminated ballast will be graded under the asphalt cap. Some areas of the right-of-way will be subject to flooding as will several other common use areas in the Lower Basin. In areas near residences, sidings, oases, access points and developed recreational areas will be right-of-way-wide, not ten feet wide. Access controls and signage are proposed to warn people of the potential hazards.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
11	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E7		Citizens Against Rails-to-Trails	0-Executive Summary			Not Accepted

Comments>

Landowners along the ROW in the lower CdA Basin between Black Lake and Chatcolet have documented levels of lead as high as 10,300 ppm, arsenic as high 310 ppm, cadmium as high as 35 ppm, and zinc as high as 18,000 ppm in 27 samples taken at their own expense. Additionally, landowners in the Cataldo area found lead as high as 6,620 ppm, arsenic as high as 161 ppm, cadmium as high as 21 ppm. These samples, taken in areas not tested nor planned for contamination removal, are within the subembankment of the UP ROW below lake or river level. These areas, apparently, are not a concern to the Governments, yet the Army Corps of Engineers (partners with EPA and DEQ) state that "Arsenic exposure can increase cancer risk, cause skin problems, and blood and disorders." Further, the Corps report states that "Cadmium at high levels can severely damage the lungs while lower levels can lead to kidney disease." It further states that "Zinc exposure can cause stomach and digestive problems. It may also interfere with the immune system." The HHRA acknowledges the potential problems associated with recreational exposure, although the detrimental amounts of contaminants considered harmful are unclear. Very likely, no amount of any carcinogen can be considered safe. Yet the HHRA carefully avoids any mention of contaminants south of Harrison, on the Reservation, along the UP ROW. Why is this area omitted from mention?

The ROW in Harrison has been documented to have over 50,000 ppm lead, and this is directly adjacent to the public beach that is located well within the ROW. Yet, no remediation has been planned for this area, and no data exist as to the dangers to humans (particularly children, the most vulnerable) from recreational exposure there. Signs posted in the area (documented by C.A.R.T. photographs) are not a deterrent, and small children continue to make mud pies, build sand castles, swim, and generally play right in this highly contaminated area. What about the arsenic? The cadmium? The zinc? The proposed trail plan states that the ballast contamination above Harrison will be bladed to the side, and a 10-foot strip of asphalt will contain the remaining contaminants. This is an absurd remedy, and the Governments are shirking their responsibility to demand that the PRP, Union Pacific, fulfills its CERCLA obligation. Inviting the public to recreate upon a highly contaminated trail (to which they would not otherwise come) is a violation of the duty to protect public welfare. In fact, the lower Basin child mentioned in C.A.R.T.'s Ombudsman testimony with a blood level of 27 ug/dcl lead in August, has recently tested a dramatically lowered level of 14 ug/dcl. The only variable accountable for the drop is that the child no longer recreates along the contaminated public use areas, including the UP ROW!

Response>>

Low levels of contamination consistent with background levels in northern Idaho communities outside the mining district were found in Harrison and southern Lake Coeur d' Alene residential areas. These concentrations were well below screening criteria. These areas as well as lower lake residences, the City of Coeur d'Alene, Post Falls, and Spokane River front homes were excluded from the human health risk assessment. North of Harrison the clean up plan proposes grading exposed ballast to the center of the right-of-way to be contained under the asphalt cap. South of Harrison ballast will be removed. One of the reasons for removing, rather than capping, contaminated materials south of Harrison is the decreased threat of recontamination from flooding outside the flood plain of the Coeur d'Alene River. Estimated post-remediation soil lead levels on the railroad right-of-way from Harrison to Heyburn State Park average 84 mg/kg. Periodic reviews of the effectiveness of the remedy is required.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
12	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E8		Citizens Against Rails-to-Trails	8-Section 8			Not Accepted

Comments>

The gross misrepresentation of land in the HHRA, particularly land south of Harrison on the Coeur d'Alene Reservation, is unconscionable. "The Coeur d'Alene Basin (CDAB) in northern Idaho includes Lake Coeur d'Alene and the St. Joe and Coeur d'Alene River drainages that are the ancestral home of the Coeur d'Alene Indian Tribe." (Page 8-1, Summary and Conclusion Section) The description omits mention of the people who own land and live on the Reservation. In fact, the Reservation area, south of Harrison, is all privately owned land, and is not, as characterized in EPA/Governments' documents, "natural resource/recreational use land." In fact, allusions to land south of Harrison or assessments of that land by EPA are non-existent. Rather, EPA/Governments' documents appear to intentionally mislead DOJ and any other agencies or people reading the documents into assuming that this Reservation land is uninhabited "with no statistics available for population density," as stated erroneously in the EE/CA, the primary document from which all subsequent planning emerged. In reality, population statistics are a matter of public record, as noted by CART in comments on the EE/CA and Proposed Consent Decree. These comments have never been acknowledged nor addressed by DOJ or the Governments. It is as if our comments were useless. In fact, our comments, like all comments we have submitted over the past nine years, are virtually ignored. Sending us form "thank you for your comment" letters does not in any way meet EPA objectives to work with the public, as well as insure public welfare.

The above description (located in the Summary and Conclusion, page 8-1) of the Coeur d'Alene Basin includes, rightfully, the lands south of Harrison on the Reservation. The HHRA Introduction, (page 1-1, 1-2, accompanying map) however, is ambiguous in specific reference to, or inclusion of, the Reservation lands. The Basin is described as "including Lake Coeur d'Alene and the St. Joe and Coeur d'Alene River Basins" (page 1-1) Yet on page 1-2, the HHRA states that "The Lower Basin area includes 11 lateral chain lakes and extensive wetlands, located adjacent to the main channel and within the CDA River's floodplain. These marshes and lakes provide an extensive recreational area between the town of Cataldo and Lake Coeur d'Alene. Camping, fishing, boating, swimming, hunting, and wildlife photography/observation are popular activities through out the lower CDAB." There is absolutely no mention of Harrison and the privately owned lands on the Reservation, or of the fact that this land is not open to public recreation ! Further, the HHRA Introduction continues by stating that: " There are no incorporated villages between Cataldo and Harrison at the mouth of the main River. However, there are a few small unincorporated village areas and several rural residences." Again, Harrison and the Reservation lands to the south—all privately owned—are not included in this description. The map on the next page, the "Site Location Map," stops below Medimont and does not include the Basin areas south of Harrison on the Reservation! This serious omission makes it appear to the HHRA reader that the Reservation land is non-populated, "public" land that is not part of the Basin. This area includes the Union Pacific right-of-way, currently proposed as part of the 72-mile recreational trail.

The HHRA continues to portray erroneously the land south of Harrison on the Reservation, and thus, infer that the proposed Mullan-Plummer recreational trail is not on or adjacent to privately owned land. For example, on page 8-2, absolutely no references are included to the land south of Harrison. Instead, the HHRA states: (page 8-2,

Response>>

The HHRA disagrees with this Comment. Extensive demographic information is included in Section 3.1 of the HHRA. Figure 3.1 shows the area included in the discussion including lands as far south as Benewah County. Little or no contamination has been noted on these properties. Estimated post-remediation soil lead levels on the railroad right-of-way from Harrison to Heyburn State Park are low. The proposed remedy was extensively reviewed by a number of public agencies and governments including the EPA, Panhandle Health District, State of Idaho, Coeur d'Alene Tribe, several federal trustee agencies, and the Agency for Toxic Substance and Disease Registry. All have found the risk management and clean up plan to be compliant with pertinent rules and regulations and protective of public health. See also General Response to Comments, #1 and #5d, and specific response to Comment E7.

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
	8-3) "Much of the Basin is rural, undeveloped land. Approximately 32% of Kootenai County and 75% of Shoshone County consist of federally managed lands, primarily National Forests. These areas are rich in natural resources including forests, wildlife, and a number of tributaries and streams that support a variety of aquatic organisms. However, many of these areas are inaccessible due to lack of roads, difficult terrain, or lack of services…….Tourism related to the use of these natural resource areas for recreational purposes has increased significantly over the last two decades and is one of the fastest growing contributors to the local economy." This generalized description is not indicative of the lands south of Harrison on the Reservation, which is all privately owned, nor is it representative of lands up to Cataldo, most of which is also privately owned. The grossly misleading depiction, endorsed by the Governments, justifies the recreational trail-related activities as outlined on page 8-3: "Recreational use of the abundant natural resource areas include riding off-road vehicles, snowmobiling, berry picking, mountain biking, fishing and floating down the CdA River, and cross-country and downhill skiing." What the HHRA fails to add is that these activities (although they do happen) are illegal on much or most of the posted "No Trespassing" private land. Readers of this HHRA are misled into thinking the land is not private land, and therefore, recreational trail-related activities are legal on these lands. The erroneous designation, apparently intentional, supports the conversion of the contaminated ROW into a recreational trail. It does not, however, support the legal rights of adjacent landowners to maintain the integrity of their private land.					
	Misc. Input>>					
13	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E9		Citizens Against Rails-to-Trails	8-Section 8			Not Accepted
	Comments>			**Response>>**		
	Related to this, the HHRA section 8.3 does not include population and demographic statistics for Kootenai County, (south of Harrison) already acknowledged as part of the Basin. Statistics are only included for Shoshone County. This omission suggests there are no homes or people in Kootenai, but this is a blatant falsehood, since every inch of this land (at least south of Harrison) is privately owned and has homes built throughout the area.			The HHRA disagrees with this Comment. See response to Comment E7 and E8 and General Response to Comments, #5d.		
	Misc. Input>>					
14	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E10		Citizens Against Rails-to-Trails	8-Section 8			Accepted
	Comments>			**Response>>**		
	And, relating to demographics, why is there no intense discussion of elevated cancer rates in Shoshone County? What about other diseases and conditions which, very likely, could be correlated to heavy metal contamination?			A recent analysis of cancer rates in Shoshone County completed since the initial draft of the HHRA has been added and will be included as an Appendix to the HHRA.		
	Misc. Input>>					

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
15	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E11		Citizens Against Rails-to-Trails	8-Section 8			Not Accepted

Comments>

Further, the almost non-existent references to the privately owned lands on the Reservation, and indeed the lack of testing done there, attest to the reality that EPA is not only misrepresenting the land, but also EPA exposes area residents and the general public to unknown risk due to the high levels of unremediated contamination from the Union Pacific right-of-way. Indeed, the "Exposure Subareas" on page 8-7 stop at Harrison. No pertinent data are included for areas on the Reservation except for Native American subsistence scenarios. No data is included nor anticipated relating to residential scenarios pertaining to children and adults who live south of Harrison on or adjacent to the proposed trail. This is an egregious oversight. Some of the most contaminated areas in the entire Basin (verified by samples paid for at landowner expense, since only one sample was taken south of Harrison before the Certificate of Interim Trail Use was issued) are in the abandoned Union Pacific right-of-way. Apparently, pregnant women and young children, the most vulnerable to lead, arsenic and metals poisoning, will not be protected from risk if they live south of Harrison.

Further, potential cancer risks as well as non-cancer illnesses related to metals and contaminants are not a concern on the abandoned right-of-way, where land owners have verified lead levels up to 10,300 ppm and arsenic levels up to 310 ppm. Yet all documents endorsed or created by the Governments agree that no level of arsenic can be considered "safe," and that lead levels must be below 400 or even lower, since no "acceptable" level has been determined by scientists and doctors!!

Response>>

The HHRA disagrees with this Comment. Native American scenarios were conducted only for the Lower Coeur d'Alene River flood plain. Sampling of gathering areas in the St. Joe River drainage showed background levels or no detects for native subsistence media. This sampling report will be an Appendix in the final report. Similarly media contaminant levels in Harrison (excluding the beach) and south on the reservation are low outside railroad right-of-way. See also specific response to Comment E7 and E8 and General Response to Comments #5d.

Misc. Input>>

16	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E12		Citizens Against Rails-to-Trails	8-Section 8			Not Accepted

Comments>

The HHRA public recreational scenario "pertains to children and adults who use developed parks and playgrounds, and undeveloped recreation areas, whether they are local residents or visitors from outside the area. Public recreational exposures were quantified separately from residential and neighborhood recreational exposures because of the potential for cross-over Basin travel and the possibility that visitors from outside the Basin could use the public areas." Yet, astoundingly, no data is included relating to landowners who live on or adjacent to the proposed trail! It is as if the Union Pacific land and the adjacent landowners do not exist! Why is the land contaminated by Union Pacific omitted from rigorous EPA accountability for cleanup when, in fact, the contamination levels far exceed those already remediated in Superfund areas, and far exceed EPA Early Action lead levels of 2,000 ppm lead? In addition, this land is not included in the Tribe's action plans for the Reservation as documented where the Tribe states that "the plan doesn't include metals pollution caused by historic mining activity, which is being assessed by the tribe and federal government." (Spokesman Review article by Julie Titone, August 28, 2000) There is absolutely no mention of Union Pacific pollution of lands within the abandoned right-of-way that are far away from the smelters and the mines! We live in these areas, and the HHRA does not contain any data relating how the railroad contamination relates to the intended recreational use along the proposed trail.

Response>>

The HHRA disagrees with this comment. Low levels of contamination consistent with background levels in northern Idaho communities outside the mining district were found in Harrison and southern Lake Coeur d'Alene residential areas. Estimated post-remediation soil lead levels on the railroad right-of-way from Harrison to Heyburn State Park average 84 mg/kg. These concentrations were well below screening criteria. These areas as well as lower lake residences, the City of Coeur d'Alene, Post Falls, and Spokane River front homes were excluded from the human health risk assessment. Extensive analysis of contamination data is from both private property and pubic properties and is included in the HHRA for the upper and Lower Basin. See also response to Comment E7 and E8 and General Response to Comments, #5d.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
17	10/06/2000	CART	Public Draft - July 2000		0/30/2000	TG
E13		Citizens Against Rails-to-Trails	0-Executive Summary			Not Accepted

Comments>

In closing, the HHRA is another one in the series of confusing, erroneous documents submitted by the Governments in support of the proposed trail which is a part of the CdA Basin cleanup. All of these documents have not included reference to the egregious damage done by Union Pacific. Instead, the documents refer to “mine waste,” to contaminants “from the smelters,” to contamination from “ore and mine tailings.” The omission of data documenting contamination directly from the railroad construction and/or the railroad operations and the risks this contamination presents to adjacent landowners and the public, is unconscionable. This is especially true since the stated main purpose of the HHRA is “to determine the extent of heavy metal contamination in environmental media that may expose current or future residents or visitors to the CdA Basin, to evaluate the potential human health risks associated with exposure to those contaminated media, and to provide information for risk managers to evaluate the need for remediated action and development of associated cleanup criteria.” Union Pacific has, clearly, received preferential treatment that leaves adjacent landowners, Basin residents, and the general public at increased risk of health problems from exposure to contaminants. The Governments clearly have placed recreation ahead of public welfare, potential tourism ahead of safety from heavy metal poisoning. C.A.R.T. will continue stringent opposition to the flawed trail plan and the accompanying political grandstanding which divert attention from the central issue: Union Pacific must be held accountable for rigid and thorough cleanup of the highly contaminated right-of-way to which they were granted an easement for railroad purposes only. The proposed trail is unconscionable, and the HHRA certainly lends credence and support to that contention.

Response>>

The HHRA disagrees with this Comment. The heavy metal contamination found in the railroad right-of-way has been identified as mine tailings used in the construction of the line as fill or ballast, ores spilled in transportation activities, or fluvial deposits of mineral industry wastes released to the environment. The risk management plan adopted to address these wastes focuses on areas likely to be accessed on the right-of-way. Within 1000 feet of any residence the entire right-of-way will be provided with a clean surface. This addresses the nominal aspects of recreation associated with the residential scenario. At all major access points, sidings and select oasis locations are scheduled for a right-of-way-wide clean up and large oases are strategically placed along the trail to provide clean rest and stop-and-view areas. In remote contaminated areas warning signs will be posted to alert trail users of areas presenting excessive risk similar to warnings to avoid local hazards in numerous venues. The signage is provided to both advise users to avoid undesirable areas and to identify safe areas to recreate. The proposal was extensively reviewed by a number of public agencies and governments including the EPA, Panhandle Health District, State of Idaho, Coeur d'Alene Nation, several federal trustee agencies, and the Agency for Toxic Substance and Disease Registry. All have found the risk management and clean up plan to be compliant with pertinent rules and regulations and protective of public health. See also General Response to Comments, #5d.

Misc. Input>>

Spokane Tribe

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
324	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F1		Spokane Tribe	0-Executive Summary			Not Accepted

Comments>

1. The title should reflect the geographic scope, technical scope, and the preliminary nature of its findings. The preliminary nature of such findings are rooted in the fact that the BHHRA falls short in evaluating risk to sensitive subgroups [NCP at 40CR300.430 (e)(2)(i)(A)(1)] from all contaminated media [NCP at 40CR300.430 (d)]. This short-fall is rooted in the fact that the nature and extent of contamination in pertinent media (e.g. ground water) has not been adequately delineated and the models used to predict current or baseline risk are inadequate. Both of these problems were foreseeable and should or could have been identified prior to performing the BHHRA.

Although the BHHRA identifies many assumptions employed to generate estimates of risk, EPA cannot assume away the types, quantities, and qualities of data critical to making sound decisions regarding remedy selection [NCP at 400.300.430 (a)(1)(i)]. It is foreseeable that if remedy selection proceeds while relying on this less than comprehensive BHHRA, risk will not have been characterized and irretrievable consequences are likely to be realized.

Response>>

The HHRA disagrees with this comment. The title of the HHRA does reflect the geographic area and scope of the document. The HHRA addresses the geographic area extending from Harrison to Mullan. The area of investigation was determined jointly by the EPA, State and Coeur d'Alene Tribe. Those scenarios, source areas, pathways and routes of exposure examined were comprehensively characterized, evaluated and presented in accordance with the NCP. The results and conclusions of the HHRA should not be extended to other areas or scenarios except as explicitly noted.

Misc. Input>>

325	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F2		Spokane Tribe	0-Executive Summary			Not Accepted

Comments>

2. The BHHRA appears to mix risk assessment and risk management concepts (see General Comment No.
1) This fact is demonstrated in discussions pertaining to background and PRGs. Risk communicators are not necessarily concerned with the source or party at fault. The public only requests to know the risks involved with specific behaviors or practices.

A better approach would be to characterize the “total risk” (pre-mining baseline risk, incremental risk associated with mining, and incremental risk associated with other anthropogenic actions). Total risk will give the public a better picture of the comprehensive risks from all significant and “insignificant” pathways associated with each scenario describing specific behaviors/practices. Incremental risk associated with past mine waste management practices can then be ascertained once these other equally important components of risk have been determined.

Response>>

The HHRA disagrees with this comment. The purpose and objectives of the HHRA are to assess the potential risk of adverse human health effects associated with contaminated environmental media in that portion of Coeur d'Alene Basin addressed. Risk assessment identifies those contaminants, media, pathways, sources of contamination, routes of exposure, and potential for human intake that could pose unreasonable risk. The risk assessment process does not determine clean up strategies or criteria for contaminated media. In situations similar to the Basin, however, public health authorities have found excess absorption to be occurring and preventative actions are in place. Risk management activities are already underway in the form of a lead health intervention program being locally implemented and focused remedial actions being conducted under emergency authority. The adjacent Bunker Hill Superfund Site (BHSS) has been implementing a variety of clean up actions and risk reduction measures for more than a decade, many of which were the genesis of Basin-related activities. As a result, the HHRA does, to the extent possible, consider and review the information obtained, the relationships observed and lessons learned in the numerous efforts to eliminate lead poisoning among the children of the Silver Valley over the last three decades. See also General Response to Comments, #10a.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
326	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F3		Spokane Tribe	0-Executive Summary			Not Accepted

Comments>

3. Based on historical accounts and technical information, the Spokane Tribe has always believed that the Bunker Hill Superfund facility as defined by CERCLA includes the Coeur d'Alene Basin as well as the shores and uplands of the Spokane River to its confluence with the Columbia River. Due to socioeconomic and political shortfalls (both of which are non-technical in nature), EPA has chosen to segregate the facility based on political boundaries with the BHHRA ending and the Spokane River HHRA starting at the Washington-Idaho boundary.

From purely a technical standpoint, EPA's approach of characterizing risk associated with the facility will only be satisfactory if the calculations of risk consider the entire list of COPCs, each RA scenario, and all pathways throughout the facility. However, today we see a much different approach being applied to the problem at hand. For example, the Draft BHHRA concludes that the Coeur d'Alene Tribal Scenario cannot be evaluated because consumption rates and/or concentrations of COPCs are so high that current Pb models employed to evaluate risks are invalid. The Principal Scientist conducting the BHHRA has stated that given the observed concentrations in only one medium (sediment), the Coeur d'Alene Tribal Scenario "pegs the needle" in the CSM. However, another contractor who is conducting a "Screening-Level" RA (a single medium, single RA scenario for "beach play") concludes that only a few problematic areas have been identified in Washington near the state line and that no further study or data gathering is warranted. In the meantime WADOE performed a two pathway scenario (fish consumption and sediment ingestion) RA which includes a broader suite of COPCs that were not evaluated in the BHHRA or the Spokane River SLHHRA, but probably are present in the basin. The result of the WADOE RA indicates that fish consumption is associated with an undesirable degree of risk.

To make matters worse, the Spokane Tribe commented several times verbally and in writing that EPA is using the Screening Level Risk Assessment Tool inappropriately to prematurely screen-out pathways and COPCs for further study. In short, we believe that results of RA modeling for a subsistence scenario designed for the Spokane Tribe and similar to the one used for the Coeur d'Alene Tribe will indicate unacceptable risk is associated with Tribal use of its natural resources.

Response>>

The HHRA disagrees with this comment. The HHRA is limited to the geographic areas and exposure pathways determined jointly by the EPA, State and Coeur d'Alene Tribe. The results and conclusions of the HHRA should not be extended to Coeur d'Alene Lake or the Spokane River except as explicitly noted. With respect to recreational, occupational and residential exposures to the resident population, most of Coeur d'Alene Lake and Spokane River areas were excluded based on the earlier screening risk assessment. Harrison beach and Blackwell Island were retained for additional consideration in the HHRA. A determination was made that insufficient data were available to assess sport or subsistence fishing in the Lake and downstream tributaries in Idaho. No evaluation of subsistence lifestyles, including the screening level risk assessment, has been accomplished for the Lake or Spokane River areas.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
327	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F4		Spokane Tribe	0-Executive Summary			Not Accepted

Comments>

4. As stated above in General Comment No. 1, the nature and extent of contamination in pertinent media has not been delineated. The result is that the BHHRA relies on assumptions to develop exposure point concentrations (EPCs). These EPCs are then used to perform calculations regarding HHR. This approach has lead EPA to use HHRA tools to identify the "nature and threat of contamination" (at points) prior to identifying the nature and extent contamination". It is almost as if the BHHRA was conducted to support an Extended Site Investigation or a Hazard Ranking Score.

Mixing risk assessment and risk management concepts as described in General Comment No. 2 above, further compounds delineation of the nature and extent of contamination by eventually setting cleanup levels in which the allowable amount of contamination (set by the HHRA) has been fully allocated to extra-background/residential sources (i.e. mine/mill wastes). The bottom line is that the true nature and extent of contamination from past mine/mill practices as well as other historical anthropogenic actions will need to be delineated in order to enable the stakeholders to determine PRGs. Such PRGs should be much lower than BHHRA Risk Based Concentrations with allowable source-derived allocation of incremental risk determined by policy makers of the involved governments.

Response>>

The HHRA disagrees with this comment. Please see response to Comments F2 and F3.

Misc. Input>>

328	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F5		Spokane Tribe	0-Executive Summary			Partially Accepted

Comments>

5. The screening step in the BHHRA does not use subsistence assumptions; therefore, contaminants and pathways that do indeed contribute substantial risk have been screened out.

Response>>

See response to Comment F22. Subsistence assumptions were not used in screening and some additional chemicals might have been selected if they were used. All pathways identified by the CdA Tribe for which data was available were quantified.

Misc. Input>>

329	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F6		Spokane Tribe	0-Executive Summary			Partially Accepted

Comments>

6. Lead effects were not added to effects from other contaminants, particularly the neurological effects. The lead goals are not based on a human NOAEL or even a human LOAEL, but rather on a definite and measurable effect in children. Therefore, it is even more important to factor in additional neurotoxicity from other inorganics. Also, we are not sure at this time of the Coeur d'Alene's policy pertaining to acceptable risk associated with lead exposure. However, the acceptable risk criteria in the BHHRA does not meet risk identification policy used by the Spokane Tribe.

Response>>

The HHRA agrees with this comment. Several potential sub-chronic effects of lead are not evaluated as possible additive effects in non-carcinogenic risk assessment for other metals, due to the lack of a reference dose for lead. The non-carcinogenic effects of lead per se, are accounted for in the IEUBK. As a result risk managers should note that risks to particular organ systems due to other metals should be considered a minimum, if lead presents similar end point risks.

Misc. Input>>

330	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F7		Spokane Tribe	0-Executive Summary			Not Accepted

Comments>

7. The BHHRA is not really comprehensive given the data gaps and the probability that PCBs and dioxins are also present in the fish and sediment (See General Comments No. 1 and 2, above).

Response>>

The HHRA disagrees with this comment. The HHRA is comprehensive and compliant with the NCP with regard to the geographic areas, exposure pathways scenarios and contaminants addressed. This HHRA does not address PCBs or dioxin. See also General Response to Comments, #10.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
331	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F8		Spokane Tribe	1-Section 1			Not Accepted

Comments>

Specific Comments

Purpose of the BHHRA

1. Page 1-1 says that the RA is a companion document to the RI. The purpose of the RI is to define the "degree and extent" of the contaminant release. The purpose of the RA is to determine the risks associated with "residual heavy metal contamination" in the entire CDARB (minus several units that are excluded without a clear rationale).

Response>>

The HHRA disagrees with this comment. Please see response to Comments F1 through F7.

Misc. Input>>

332	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F9		Spokane Tribe	1-Section 1			Not Accepted

Comments>

2. Page 1-4 says that "it is important that the HHRA be conducted comprehensively," which needs community definition because the RA is not really comprehensive. See General Comments and comments below regarding screening and the need to be comprehensive).

Response>>

The HHRA disagrees with this comment. Please see response to comments F1 through F7.

Misc. Input>>

333	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F10		Spokane Tribe	1-Section 1			Accepted

Comments>

Boundaries of the CDARB

1. Section 1.1.1. The Coeur d'Alene River Basin boundaries are incorrect. The Spokane River has been omitted. Other language in this section includes the Spokane River identified as CDARB CSM Unit 5. The reference for the separate analysis of the Spokane River needs to be provided.

Response>>

The Spokane River was not discussed in this section and the text will be revised. Section 2.1 discusses each basin geographical area in detail and includes the Spokane River. Section 2.1.5 specifically addresses the Spokane River and references the screening document which evaluated the Spokane River.

Misc. Input>>

334	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F11		Spokane Tribe	1-Section 1			Partially Accepted

Comments>

2. Page 1-3. The sediments and tailings in the Cataldo area also migrated past Lake Coeur d'Alene and into the Spokane River.

Response>>

The HHRA agrees with this comment.

Misc. Input>>

335	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F12		Spokane Tribe	1-Section 1			Partially Accepted

Comments>

3. Page 1-4, Para 4. The sediments did not "possibly" migrate into the Spokane River, they definitely did.

Response>>

The HHRA agrees with this comment

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
336	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F13		Spokane Tribe	1-Section 1			Partially Accepted

Comments>

4. The exclusion of segments for lack of data may result in an underestimation of the nature and extent of contamination as well as the risks (See General Comment No. 2).

Response>>

The HHRA acknowledges and discusses the lack of data in many areas. With regard to exposure areas, scenarios, and pathways addressed, the Coeur d'Alene Basin is extremely large and complex. Early in the planning process, in order to meet public requests, the HHRA was placed on an accelerated schedule to be completed in parallel with the RI/FS. It was recognized that, with the associated time and budget constraints, sampling efforts would be limited. Decisions were made to utilize existing data to the maximum extent practicable, fill major data gaps with focused sampling efforts, and not address all possible data gaps and exposure pathways.

Misc. Input>>

337	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F14		Spokane Tribe	1-Section 1			Partially Accepted

Comments>

5. Excluding data from the adits on the rationale that the fences will prevent entry of people for all time should be revisited. In fact, adits are attractive nuisances. Intruder risks need to be included, especially for teenagers.

Response>>

The HHRA agrees with this comment. These properties can be evaluated by the incremental methodology provided in the HHRA. Incremental exposures were characterized using typical parameters that are specified in the HHRA. Intakes are calculated in a straight-forward manner proportional to those parameters and media contaminant concentrations. Risk management decisions for recreational or trespasser scenarios will be made on a site-specific basis, that will likely require additional sampling and survey information regarding contaminant levels, access restrictions and ownership. Should risk managers elect to modify risk factor parameters to site specific concerns, intake rates can be adjusted proportionately. See also General Response to Comments, #5b.

Misc. Input>>

338	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F15		Spokane Tribe	1-Section 1			Not Accepted

Comments>

Environmental Data

1. A separate document is needed that examines all the available environmental data collected over time. The detection limits of methods changed over the decades. The GLP and test methods need to be reviewed. The data are presented in a confusing manner, scattered through several chapters.

Response>>

The HHRA disagrees with this comment. The parent documents referenced in the HHRA and the Appendices provide the information.

Misc. Input>>

339	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F16		Spokane Tribe	1-Section 1			Not Accepted

Comments>

2. The differences in sampling depths and sieving raises concerns. It appears that soil samples from 1", 6" or 12" were mixed and sieved before analysis, which will alter the results, perhaps dramatically. Since sediment is laid down in layers, mixing up to 12" could dilute the concentration. A study that evaluates the concentration of COPCs as a function of grain size distribution within each CSM is necessary.

Response>>

The sediment layers may have different concentrations; however, risks are calculated based on the average chemical concentrations in a particular media to which people are exposed. The assumption is that when people are using the beaches they are exposed equally to these different depths (due to digging play on the beaches). Therefore, the risk equations appropriately use an average sediment concentration that represents beach exposure to estimate health risks. See also response to Comment B4.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
340	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F17		Spokane Tribe	1-Section 1			Not Accepted

Comments>

3. The rationale for sieving needs to be explained further. Page 1-3 refers to highly mobile and dispersible fine materials (without definition of particle size), while the paper by Kissell et al. on the skin adherence of < 175 um particles is used to justify the method used for environmental sampling. The relation between particle size, concentration (or adsorption), skin adherence, resuspension as dust, mobilization and sedimentation in water, plant and animal uptake, ingestion of all particle sizes, and so on should be explained more clearly.

Response>>

Section 2.2.1, pages 2-6 to 2-7 of the HHRA provides in detail the rationale for sieving and the use of the <175 um particle size for human health risk assessment. Other grain sizes and considerations are used for ecological risk assessment and are considered in the RI and FS portions of the study documents for the Basin.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
341	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F18		Spokane Tribe	1-Section 1			Not Accepted

Comments>

4. The water data is not clearly defined as filtered or unfiltered, or total versus dissolved.

Response>>

All water samples were unfiltered and analyzed for total metal content as described on page 2-11, Section 2.2.1. Both disturbed and undisturbed surface water samples were collected for the subsistence scenarios.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
342	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F19		Spokane Tribe	1-Section 1			Partially Accepted

Comments>

5. The problems with lack of fish data (no samples in the main lake and rivers, and analysis for only 3 or 1 contaminant), garden vegetables tested for only 3 compounds, total lack of animal data, and so on will be problematic in the future if not addressed soon.

Response>>

The HHRA agrees that these media have not been fully characterized at this time.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
343	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F20		Spokane Tribe	1-Section 1			Not Accepted

Comments>

Contaminants of Concern and the Screening Process

1. The COC list includes antimony, arsenic, cadmium, iron, lead, manganese, and zinc, but lead is the only contaminant that carries through the selected exposure pathways. These omissions increase the uncertainty and underestimate the risks.

Response>>

The HHRA disagrees with this comment. All potential COCs for which data are available were assessed according to the pertinent federal guidance. See also General Response to Comments, #10.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
344	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F21		Spokane Tribe	1-Section 1			Not Accepted

Comments>

2. Page 1-2 lists lead, zinc, arsenic, cadmium, silver, gold, copper, cobalt, nickel, sulfuric acid, phosphoric acid and fertilizers as major products (there must be minor products as well). The mine tailings and other materials placed on the Cataldo flats include zinc, lead and cadmium wastes. Aluminum, antimony, and thallium are also mentioned. A table that shows the original 23 contaminants and the reasons they were screened out would be helpful.

Response>>

The chemical screening tables (Table 2 series) in Appendix A provide a complete list of all the analytes for each media. In each subsection of Section 2.5, we note how many analytes were examined for each media and we refer the reader to the applicable tables in Appendix A.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
345	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F22		Spokane Tribe	1-Section 1			Partially Accepted

Comments>

3. The definitions of SV and PRG should be presented in risk terms (i.e., the SV for a contaminant relative to its hazard quotient or 1E-6 cancer level given a particular set of assumptions) should be presented a table. The equation for the SV indicates that they are NOT based on subsistence exposure scenarios. If there were a subsistence-screening step, more contaminants would be COPCs.

Response>>

The SV and PRG were defined according to health risk goals in Section 2.4.5, page 2-17. They are presented on the Table 2 series in Appendix A. We agree that these values were based on residential land use and were not based on subsistence exposures. We acknowledge that the use of subsistence-based screening values might have selected additional chemicals. However, the subsistence pathways were clearly identified as "risky" in the HHRA (these pathways had the highest exceedences over target health goals) and selecting more chemicals to evaluate would not change the conclusions of the report. In addition, the HHRA has appropriately identified the risk drivers for the subsistence pathways. We will revise Section 2.4.5 and the Uncertainty Section to clarify chemical selection with respect to the subsistence pathways.

Misc. Input>>

346	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F23		Spokane Tribe	1-Section 1			Not Accepted

Comments>

4. The one-contaminant, one-pathway risk levels of 1E-6 or 0.1 may not be acceptable to the affected communities.

Response>>

The HHRA selected COPCs using screening values based on a 1E-6 risk level or an HQ of 0.1. We do not believe that chemicals which pose a risk to the general population were omitted from the evaluation. Once a chemical was selected, the HHRA evaluated risks using a multi-contaminant, multi-pathway approach.

Misc. Input>>

347	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F24		Spokane Tribe	1-Section 1			Not Accepted

Comments>

5. The arsenic MCL for drinking water will be lowered to 5 ug/L, but the reference dose has not changed yet.

Response>>

The process which has resulted in EPA proposing a lower MCL for arsenic is independent of the process by which the RfD was calculated. Therefore, MCL changes do not necessarily mean RfD changes would be required. The RfD for arsenic was appropriately used in this risk assessment. See other responses to comments on this issue as well as Dr. Paul Mushak's responses on arsenic's RfD.

Misc. Input>>

349	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F26		Spokane Tribe	1-Section 1			Not Accepted

Comments>

7. If this RA is supposed to be "comprehensive," then at least a few soil and biota samples need to be analyzed for PCBs, dioxins, forestry herbicides, lumber treatment materials (preservatives such as CCA and PCP, creosote and polyaromatics, and so on). With all the logging and mining activity, some gasoline or diesel spills would be expected, too.

Response>>

The scope of the CERCLA response was limited to mining-related contamination in the Silver Valley, specifically metals. Organic compounds that may be present around specific mining and mill sites may be evaluated in the future.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
350	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F27		Spokane Tribe	2-Section 2			Not Accepted

Comments>

8. The COPC concept (page 2-11 and following) is based on the rationale that contaminants are co-located and that cleaning up the major contaminants, originally conceived as excavation, results in excavation of the co-located contaminants. This concept was logical at the time 20 years ago, but it is not as relevant to sites where contaminants have had decades to migrate differentially. In addition, as we learn more about the toxicity of contaminants both individually and in combination, it is clear that eliminating contaminants based on their individual concentrations within individual pathways of exposure results in underestimation of risk, some times by a large amount. The RA as a whole, then, is far from "comprehensive."

Response>>

We disagree. COPCs are not selected based on co-location. The COPCs are selected based on their individual exceedences above background and health-based concentrations in each media of concern. Page 2-11 makes no references to co-location, nor are there any discussions regarding excavation on page 2-11 or subsequent pages in this section.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
351	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F28		Spokane Tribe	2-Section 2			Not Accepted

Comments>

9. It is inadequate and improper to use Region 9 soil PRGs as screening levels. The Region 9 PRGs assume no existing groundwater contamination, no future leaching to groundwater, no ecological impacts, no uptake into food, no inhalation of volatiles, and residential (not subsistence) exposure. The factors for dust resuspension may also be unsuitable for the "highly mobile fine material." Using soil PRGs as a screening tool, then, assumes that some pathways are nonexistent, which results in screening out contaminants that may in fact be posing considerable risk via the pathways that are omitted. They are certainly not suitable for use in evaluating risks to tribal members.

Response>>

We disagree. The use of the Region 9 PRGs appropriately selects chemicals for HHRA evaluation per US EPA guidance. Ecological impacts are evaluated in the ecological risk assessment and potential leaching to groundwater is evaluated in both the RI and FS reports. Uptake into food was examined for both garden vegetables and fish -- these media were not screened. No volatiles were evaluated in this HHRA, see response to Comment F26; however, we note that the Region 9 PRGs include the inhalation pathway for all chemicals. See also response to Comment F22 regarding subsistence exposures. Subsistence pathways were identified as a risk in the HHRA.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
352	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F29		Spokane Tribe	2-Section 2			Not Accepted

Comments>

10. If a screening process is used (and it is preferable not to screen out any contaminants that are detected), it should be on a risk basis, not a regulatory basis. In other words, a full multipathway CSM must be used and contaminants screened out if they contribute individual risk levels of some TBD fraction of a total cancer and non-cancer risk. It is likely that contaminants have been improperly been screened out of the analysis. Mercury should be included regardless of its relation to the SV or PRG.

Response>>

We disagree. Selection of COPCs followed USEPA guidance. Mercury was retained as a COPC in surface water and in fish where it was identified as a potential hazard. The potential underestimation of risk from screening out chemicals is discussed in Section 7. See response to Comment F22, F27, and F28.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
353	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F30		Spokane Tribe	3-Section 3			Not Accepted

Comments>

11. After screening out contaminants, the problem is further compounded by screening out entire pathways (section 3.2.2).

Response>>

Screening out pathways that will not make a significant impact on risks and/or which cannot be quantified for some reason follows US EPA guidelines for risk assessment. The reasons for the exclusion of some pathways are clearly explained in Section 3.2.2. Potential underestimation of risk from screening out pathways is discussed in Section 7.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
354	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F31		Spokane Tribe	2-Section 2			Not Accepted

Comments>

12. The entire background argument is irrelevant to risk. The receptor does not know whether exposure is background or source-derived. Background should never be subtracted from the risk assessment numbers. It is only relevant when risk management actions are chosen.

Response>>

We disagree. Background concentrations were appropriately used as part of the screening process to select COPCs. USEPA guidance requires a comparison to background as part of the selection of COPCs process. If chemical concentrations do not exceed background concentrations, then the chemical is not selected because concentrations have not been impacted by site activities (in this case, mining). The reviewer is incorrect in stating that background was "subtracted" from the risk assessment numbers. Once a chemical is selected, background concentrations are not "subtracted" from the risk equations.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
355	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F32		Spokane Tribe	2-Section 2			Not Accepted

Comments>

13. The relation of background, detection limits and risk-based values is important, but the discussion in Section 2.4.2 of medium-by-medium (or pathway-by-pathway) and contaminant-by-contaminant approach serves only to eliminate contaminants for the convenience of the assessor. The total (cumulative) risk goals from the receptor's perspective are not stated.

Response>>

We disagree. The HHRA appropriately followed US EPA risk assessment guidance which requires that risk assessments focus on the chemicals that will drive risks and thus provide relevant and crucial information to the risk manager. EPA's 1989 Risk Assessment Guidance for Superfund states on page 5-23:

"The objective of the screening procedure is to identify the chemicals in a particular medium that -- based on concentration and toxicity -- are most likely to contribute significantly to risks calculated for exposure to that medium, so that the risk assessment is focused on the "most significant" chemicals."

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
356	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F33		Spokane Tribe	2-Section 2			Not Accepted

Comments>

14. The selection of the screening process, the risk goals, and so on, should have been the subject of government to government discussion at the study design step. The result is a draft risk assessment that fails to consider tribal risks from start to finish.

Response>>

The HHRA disagrees with this comment. The area of investigation scenarios and exposure pathways addressed was determined jointly by the EPA, State and Coeur d'Alene Tribe. Subsistence scenarios and relevant exposure factors were developed in cooperation with Coeur d'Alene Tribe representatives. The Traditional and Current Subsistence scenarios were requested by the Tribe as representing possible future uses of the geographic area addressed in the HHRA. Exposure factors were derived specifically for the Coeur d'Alene Tribe. Scenarios and exposure factor analysis were patterned after the development of similar scenarios for the Columbia River Tribes. A cultural anthropologist, working for the Coeur d'Alene Tribe, reviewed and suggested appropriate modifications for each of the exposure factors.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
357	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F34		Spokane Tribe	1-Section 1			Partially Accepted

Comments>

Receptor Definitions

1. Page 1-5 (Section 1.2) identifies children as age 1-9, adults as females between 17-49, and everyone else as the remainder. This is not standard risk assessment identification, although for lead by itself it makes some sense. However, for a general risk assessment, additional categories need to be used: infants, children aged 1-6, elders, and so on.

Response>>

The HHRA agrees with this comment. References to and discussions regarding susceptible sub-populations are included in Sections 3.1 and 3.2 of the HHRA.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
358	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F35		Spokane Tribe	2-Section 2			Not Accepted

Comments>

2. Section 2.1.3. Did the Coeur d'Alene Tribe have an opportunity to define some of the human exposure areas? Did they agree that only two areas (the mouth of the C d'A River and the lower basin-chain lakes area) would be used with subsistence exposure factors?

Response>>

Please see response to Comment F33 and General Response to Comments, #6a.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
359	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F36		Spokane Tribe	3-Section 3			Not Accepted

Comments>

Subsistence Exposure Scenarios

1. The Current Subsistence scenario uses 61 days/year, assuming that the warmest two months are spent traditionally - does the Coeur d'Alene Tribe agree?

Response>>

Please see response to Comment F33 and General Response to Comments, #6a.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
360	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F37		Spokane Tribe	3-Section 3			Not Accepted

Comments>

2. The Current Subsistence scenario uses 170 g/d for fish consumption (and a similarly reduced number for children) - does the Coeur d'Alene Tribe agree with these consumption rates.

Response>>

Please see response to Comment F33 and General Response to Comments, #6a.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
361	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F38		Spokane Tribe	3-Section 3			Accepted

Comments>

3. The omission of pathways means that the risks are underestimated, not "conservative" (which usually refers to overestimation in the risk community; page 3-33, bottom).

Response>>

The HHRA agrees with this comment. The text will be modified in the final document.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
362	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F39		Spokane Tribe	6-Section 6			Partially Accepted

Comments>

Health Risks

1. Lead risks are treated entirely separately even though many metals have neurologic effects. This results in an understatement of risks.

Response>>

The HHRA agrees with this comment. Please see response to comment F6.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
363	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F40		Spokane Tribe	6-Section 6			Partially Accepted

Comments>

2. The CDC PbB recommendation (95% <10 ug/dL) is used as the threshold to identify risk potential risk. This threshold is much higher and therefore less protective than the threshold used by the Spokane Tribe.

Response>>

The HHRA is unaware of Spokane Tribe thresholds and has not included any that might differ from those of other governments in this document.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
364	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F41		Spokane Tribe	4-Section 4			Not Accepted

Comments>

3. The GI absorption needs to be carefully examined, particularly the bioavailability rates of arsenic (Section 4.3 and 7.3.2).

Response>>

GI absorption was assumed to be 100 percent for all chemicals except arsenic. Assuming 100 percent is health-protective. For arsenic, the EPA considers there is sufficient information to depart from the default assumption of 100 percent. This information was discussed in Sections 3, 4, 7, and in Appendix I which contains detailed toxicity profiles for each chemical of concern.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
365	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F42		Spokane Tribe	5-Section 5			Accepted

Comments>

4. Of the 7 COCs, only two were evaluated for dermal exposure.

Response>>

The HHRA agrees with this comment. These chemicals were selected on the basis of potential adverse effects by exposure route. The other COPCs were not considered to have significant adverse health effects by the dermal pathway for the conditions of exposure in the CdA basin.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
366	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F43		Spokane Tribe	4-Section 4			Not Accepted

Comments>

5. Section 4.1.2 says that only oral RfDs were used - does this mean that inhalation of resuspended dust was not included?

Response>>

Yes. See sections 2.5.6 and 3.2.2. Inhalation of fugitive dust was not quantitatively evaluated for the non-lead metals.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
367	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F44		Spokane Tribe	4-Section 4			Not Accepted

Comments>

6. The information in Section 4.3 on individual contaminants say that many are poorly absorbed, but the assumptions used in the RA are not given.

Response>>

The use of the gastrointestinal absorption factor is discussed on page 3-47. As the text states, a correction factor was used for arsenic only and a detailed discussion follows on why the 60% factor was chosen. For all of the other chemicals, no correction factor was used therefore 100% absorption was assumed. See also response to Comment F41.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
368	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F45		Spokane Tribe	4-Section 4			Accepted

Comments>

7. There is additional information on lead that is not included in Table 4-2, such as behavioral effects at low exposure levels. Also, please include information on population blood lead levels in uncontaminated areas (it is at or below 5 ug/dL) to avoid the implication that 10 ug/dL is a NOAEL.

Response>>

The HHRA agrees with this comment. Additions to the Table will be considered for the final document.

Misc. Input>>

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
369	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	URS
F46		Spokane Tribe	5-Section 5			Partially Accepted

Comments>

8. Section 5.3 should emphasize two additional points: that not all contaminants were carried through the assessments and that lead is not included in the noncancer hazard discussion on hazard index.

Response>>

Section 5 will be amended for the subsistence section to indicate the potential for additional chemicals of concern. The non-carcinogenic effects of lead are addressed in Section 6, not in Section 5 because lead lacks a reference dose. See also resonse to Comment F6.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
370 F47	11/20/2000	Fred Kirschner Spokane Tribe	Public Draft - July 2000 6-Section 6		2/07/2000	TG Not Accepted

Comments>

9. Separation of the lead exposure into that received from residence (yard, garden, commercial foods obtained from elsewhere, housepaint) from that received from additional incremental exposure through recreational and occupational exposure may be a problem if this information is used to allocate risk management goals. A more logical way to define baseline in this case might be to separate housepaint and commercial food obtained from non-local sources from all source-derived exposure. Then, the PRG would be set assuming that paint-food exposures are ubiquitous and uncontrollable, so the soil PRG must be more stringent.

Response>>

The HHRA disagrees with this comment. The HHRA evaluates the potential human health risks associated with contaminated environmental media. The lead analysis examines the effects of soil and dust lead on blood lead levels in concert with dietary and other sources. There is little indication of direct ingestion of paint particulate aside from that of lead paint incorporated in the soil and dust pathway. The site-specific analysis uses observed soil and house dust lead levels. As a result, the sources of lead to dust, such as paint, yard soils, materials tracked in by workers, fugitive dusts, etc. are inherent in the analysis. The influence of lead paint on these pathways is examined by regression analysis. The interpretation of these results was that contaminated soils, house dust, and lead based paint are all related to excess absorption. See also General Response to Comments, #4a, #5a, and #7a.

Misc. Input>>

371 F48	11/20/2000	Fred Kirschner Spokane Tribe	Public Draft - July 2000 6-Section 6		2/07/2000	TG Not Accepted

Comments>

10. Since Native American subsistence exposures were estimated to be too high for the IEUBK to run properly, how will this information be used to set PRGs, especially since tribal blood lead values currently are not available? At what point does the IEUBK certainty diverge from an acceptable level?

Response>>

IEUBK analysis of subsistence lead intake could become relevant at levels resulting in blood lead concentrations in the 20 ug/dl to 30 ug/dl range and below. This would occur at media concentrations substantially less than those observed today. Blood levels below health criteria are unlikely to occur until media concentrations approach background levels. However, this assumes that the bio-kinetic portion of the model is applicable to individuals practicing subsistence lifestyles. The HHRA is unaware of any data or investigations regarding absorption factors for subsistence lifestyles.

Misc. Input>>

372 F49	11/20/2000	Fred Kirschner Spokane Tribe	Public Draft - July 2000 6-Section 6		2/07/2000	TG Not Accepted

Comments>

11. Are we going to get into an argument about whether the Box or Default Model is more applicable in different parts of the Valley? How can this be prevented?

Response>>

Please see General Response to Comments, #9.

Misc. Input>>

373 F50	11/20/2000	Fred Kirschner Spokane Tribe	Public Draft - July 2000 3-Section 3		2/07/2000	TG Accepted

Comments>

Other Comments

1. The area use (page 3-2) does not mention tribal use. Other than this omission, the demographic description is much better than usual.

Response>>

The text will be amended for the final document.

Misc. Input>>

374 F51	11/20/2000	Fred Kirschner Spokane Tribe	Public Draft - July 2000 3-Section 3		2/07/2000	URS Partially Accepted

Comments>

2. Section 3.1.4. "some residents will be exposed to lower concentrations in their homes than others." How is the range of test results used? It would be useful to at least have a reference to the uncertainty section and to the results that are presented as ranges.

Response>>

The intent of this section is to indicate that concentrations in individual homes are variable and that risks for non-lead are estimated by geographical area and not by individual home. The only ranges of risks estimated are for the reasonable maximum exposure scenario (RME) versus the central tendency (CT) scenario. RME and CT results are both presented in Section 5. The text in Section 3 will be clarified.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
375	11/20/2000	Fred Kirschner	Public Draft - July 2000		2/07/2000	TG
F52		Spokane Tribe	3-Section 3			Accepted

Comments>

3. It would be useful to have tables showing the various locations, the relevant exposure scenarios, and the contaminant concentrations. Without this, it is hard to tell how the environmental data were used (average? maximum? distribution?) with which pathways and scenarios.

Response>>

The HHRA agrees with this comment. All the assumptions are included in various Tables throughout the document. A summary table will be added to the final document to consolidate the presentation.

Misc. Input>>

Private Individuals

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
376	12/08/2000	Justin Rice-Wallace	Public Draft - July 2000		2/08/2000	TG
G1		Private Individual	0-Executive Summary			Not Accepted

Comments>

After reviewing the Human Health Risk Assessment (HHRA) Executive Summary it doesn't appear to fairly represent what I see occurring in my community. I have lived here for many years, and I believe the risk from lead exposure is minimal. The Environmental Protection Agency and the HHRA have completely overestimated the exposure that my children and I face living in the Silver Valley.

I understand that the HHRA has not taken into consideration the amount of time children are actually exposed to lead in their environment. There is a great deal of speculation and many assumptions that went into preparing the HHRA. Did you take into consideration that children don't habitually play on waste rock piles, that there is snow on the ground for six months of the year, and that children don't eat large quantities of fish from the river, bones and all? You assume that local children are playing for days, weeks and months on the sandy beaches along the river. You assume that they eat garden vegetables on a regular basis that are full of lead.

Response>>

The HHRA disagrees with this comment. The assumptions used in the HHRA are based on consensus reviews of scientific research and collaboration with national experts in the field of lead, and human health risk assessment. The HHRA does consider that children do not habitually play on waste rock piles, and that many areas are not available during a large portion of the year due to snow and other variables. For example, the predicted Reasonable Maximum Exposures for children playing on Upland Parks was based upon two visits per week for 34 weeks per year. Default dietary intake rates representing the typical US market basket are included in both model forms. National default values are used for baseline dietary intake rates throughout these analyses. Discussion of incremental lead intake rates from home grown produce and recreational fish consumption is discussed in section 6.3.1. The fish ingestion pathway evaluated for the tribal scenarios is based on filleted tissue metals data from a limited number of species from the lateral lakes and whole fish from the Spokane River. Fish ingestion for the resident population is based on fillet data from the lateral lakes. These results are likely not representative of fish from
Lake Coeur d'Alene and extrapolation of hazards and risks to the Lake Coeur d'Alene fishery is not recommended. Garden vegetable lead levels are based on actual samples collected in the Basin. See also General Response to Comments, #5 and #7.

Misc. Input>>

377	12/08/2000	Justin Rice-Wallace	Public Draft - July 2000		2/08/2000	TG
G2		Private Individual	0-Executive Summary			Not Accepted

Comments>

What I really don't understand is why you assume that the exposure comes mainly from soils. What about the paint in my home and other homes in the Valley, most of which were built long before the 1970's Isn't that lead paint?

Response>>

The HHRA disagrees with this comment. Lead from paint is discussed in Section 6.3.4, and specific studies regarding lead-based paint in the Coeur d'Alene River Basin are cited. Table 6-13 shows summary statistics for lead-based paint by geographic subarea, and Figures 6-7a and 6-7b show mean interior and exterior paint lead concentrations by geographic area. Extensive site-specific analysis was conducted regarding the relationships between blood, soil, paint, and dust lead levels. The interpretation of these results in the HHRA was that contaminated soils, house dust, and lead based paint are all related to excess absorption. Overall this suggests complex exposure pathways, with blood lead levels most related to dust lead loading in the home, followed by independent effects of yard soil lead, interior paint lead condition, and exterior paint lead content. The dust lead pathway is most influenced by outdoor soils, augmented by paint contributions in older homes, especially those in poor condition. See also General Response to Comments, #3 and #4.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
378	12/08/2000	Justin Rice-Wallace	Public Draft - July 2000		2/08/2000	TG
G3		Private Individual	0-Executive Summary			Not Accepted

Comments>

I really don't understand why you can't just check all the children instead of these small sample groups that don't represent the entire Valley. The HHRA states that our communities are pretty impoverished. Yet, if you compare us on a national level, our children have the same blood lead levels as children who live in similar conditions. So why are we being singled out to look like we are worse off?

Response>>

Testing the blood lead of every child in the Basin is unrealistic as mandatory compliance could not be compelled under State law. However, the Panhandle Health District (PHD) has an extensive promotional effort to inform and encourage voluntary testing of all children (ages 9 months through 9 years) throughout the Basin. One week prior to field activities, the program and the project schedule are advertised in area newspapers and on the radio. A bulk mailing was also sent to every house in the basin advertising the blood lead program. A phone number is provided so those who wish to participate can contact the project office. However, not all parents will choose to have their child tested. See also General Response to Comments, #1a and #2a.

There is a divergence of opinions regarding the appropriate comparisons between the National and State-wide Lead absorption databases with the results of the HHRA. Comparisons are difficult for the following reasons: 1) scientific designs of the NHANES surveys are constructed in a way that does not permit valid comparisons with results of blood lead distributions for a given community, and 2) the design for gathering and organization of the Basin data was not for purposes of matching the various demographic and socioeconomic strata in the NHANES III survey reports. If the Basin data was divided into the numerous categories to allow such comparisons, it would produce so few children as to make comparisons with national data meaningless.

Misc. Input>>

379	12/08/2000	Justin Rice-Wallace	Public Draft - July 2000		2/08/2000	TG
G4		Private Individual	0-Executive Summary			Not Accepted

Comments>

Do you not understand that by playing with our communities as if we were science projects or experiments you paint us in a bad light? Who wants to live in the Valley or vacation here or develop businesses here if they are given a false impression of the health risks to their families? We are proud of our children and our educational system. We have in the past and continue to produce some pretty smart young men and women in this Valley. Instead of using models and tables and assumptions why don't you look around you and see what kind of people really live here. Maybe then you will understand why we are insulted by your actions and believe the HHRA and its supporters are not working in the best interests of the people of the Silver Valley.

Response>>

The HHRA is not intended to insult anyone, but is designed to identify potential pathways for lead and other heavy metal exposure, so that residents of the Basin can be aware of any associated risks, and future decisions can be made to protect human health throughout the Basin. A diversity of opinions has been received from citizens, some believing risk has been overstated, others believing risk is understated, and a few believe risk has been fairly portrayed.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
380	12/08/2000	Frank Frutchey	Public Draft - July 2000		2/08/2000	TG
H1		Private Individual	0-Executive Summary			Not Accepted

Comments>

Thank you for extending the comment period for the HHRA regarding the Coeur d'Alene Basin. After reviewing the information presented to the citizens at the citizens advisory committee, it seems to me the correlation between human blood lead levels in the CDA Basin and the concentrations of lead in the soil does not tie together very well. Children who live and/or play on highly contaminated soils do not necessarily have high blood lead levels; conversely, some children with high blood lead levels do not live on, or near contaminated soils. Therefore, the cause and effect relationship between soil lead levels and human blood lead levels is weak.

In order to more effectively break the pathway of inception, it would be more prudent to investigate the habits of the children with high levels to determine the most practical method of prevention. There may be other sources of lead besides only that in the soil; examples of other sources may be paint, lead containing solder in water pipes, leaking batteries and old tailings piles used as playgrounds.

Response>>

The relationship of soil lead concentrations to blood lead levels is both direct and indirect and exposure can occur away from the home. Indirect exposure to soils can occur when soil becomes entrained in the house dust. Quantitative analysis of these relationships show that yard soil is a major contributor to mat dust lead loading in the home suggesting that yard soils are moving into the house. Mat dust lead loading showed the strongest relationship with blood lead levels (Tables 6-19 and 6-20). See General Response to Comments, #3d and #4.

Children with blood lead concentrations greater than 10 ug/dl are followed up with a nurse visit to investigate all possible sources of exposure. These nurse visits have noted that the majority of the cases of high blood lead levels have been attributed to high soil and dust concentrations either in the residence or from recreational visits. Lead solder and leaking batteries are rarely found to contribute to these cases of high blood leads, whereas lead paint is sometimes a problem. The focus of the nurse follow-up is on the pathways of exposure as well as education about the risks of lead. The PHD's Lead Health Intervention Program provides information and educational tools on the risks of lead exposure and how to decrease a child's exposure to lead in the community.

Misc. Input>>

381	12/08/2000	Frank Frutchey	Public Draft - July 2000		2/08/2000	TG
H2		Private Individual	0-Executive Summary			Partially Accepted

Comments>

Good hygiene plus re-establishing a good, soil farming vegetative cap on top of leaded soils will in my experience result in lower blood lead levels more quickly and more cost effectively than trying to dig up and safeguard in a repository all the soils with lead levels above EPA's background level.

Also, we who have lived in the CDA Basin for any length of time, know that relatively large amounts of lead, will be moved around alluvially during episodic events. Therefore, it seems futile to me to address the problem of human exposure to lead by digging and removal since several episodic events can occur each decade. In situ treatment of heavy metals in the soil coupled with re-vegetation using an indigenous sod farming grass will in my experience grow up through silt deposited during an episodic event. It is better to work with nature, rather than to struggle against such natural processes.

Response>>

The HHRA agrees that the selection of remedial clean up alternatives should consider these points in developing the Proposed Plan for the Basin.

Misc. Input>>

Community Leaders for EPA Accountability Now (CLEAN)

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
382	10/16/2000	Dee Jameson	Public Draft - July 2000		2/08/2000	TG
I1		CLEAN	0-Executive Summary			Not Accepted

Comments>

Accordingly, we recognize the EPA allowed the State to have the lead on the HHRA, but with it came the Integrated Exposure Uptake Biokinetic Model (IEUBK). The model, based on national defaults does not accurately reflect the conditions present in the Coeur d'Alene River Basin today. It is widely believed the model is not appropriate for evaluating periodic exposure to lead, based in part on the fact that the model grossly overestimates the potential blood-lead level of Basin residents. As such, we respectfully ask the State to modify its plan to allow for actual site-specific conditions - based on the factual data compiled by the Panhandle Health District during the last two decades.

Response>>

The HHRA was accomplished in accordance with pertinent EPA policy and guidance in compliance with the National Contingency Plan. The most recent guidance regarding use of the IEUBK and site-specific blood lead data is found in Appendix O. Both types of analysis are accomplished in the HHRA and the "Box-model" uses site specific data from the Bunker Hill Superfund Site as an input into the IEUBK. In addition, the follow-up investigations on children with high blood lead levels have been summarized in Section 6.2.3 of the HHRA. All of these data and analyses are considered in reaching the findings and conclusions of the document. See also General Response to Comments, #9 and #10.

Misc. Input>>

383	10/16/2000	Dee Jameson	Public Draft - July 2000		2/08/2000	TG
I2		CLEAN	0-Executive Summary			Not Accepted

Comments>

Outside the BHSS, the State's contractor TerraGraphics is using a cumulative set of blood-lead level (BLL) results (1996-99) for the entire Basin. By doing so, the levels hide the gains made, year by year, during that time. In reality, the 1999 Basin BLL average of 5.3 mg/dl shows that the average blood lead levels in the Basin are already at or near the EPA's remedial action goal.

Response>>

The HHRA disagrees with this comment. Observed blood lead levels have shown little discernable difference in the last four years with respect to mean blood lead levels or the incidence of children to exceed 10 ug/dl. The data shown in Table 6-1 suggest no significant difference among the four years of data, although the poor turnout in some years precludes making valid comparisons. The cumulative data set was used to maximize the number of observations available to support the site-specific analysis and to use actual blood lead levels to the maximum extent practicable in assessing risk in the Basin. Available data indicate that about one-in-four children under two years of age have blood lead levels of 10 ug/dl or greater, and the age adjusted incidence of excess blood lead levels is 16.2% for 1-6 year-old children. This incidence of high blood lead levels is a health concern for these children. There are divergent opinions as to how well the health surveys represent the non-participants and whether comparisons to other national and State populations are appropriate. Comparison of blood lead data for the Basin to other sites and national or State-wide surveys, for the purpose of determining whether these findings are "relatively good or bad", is problematic.

Selection bias may have occurred related to individual family decisions to participate. These opinions are discussed in Sections 6.2.2 and 7.4.1, 8.8, and 8.11.2 and reflect most of the comments offered by reviewers. See also General Response to Comments, #2, #3a and #3b.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
384	10/16/2000	Dee Jameson	Public Draft - July 2000		2/08/2000	TG
I3		CLEAN	0-Executive Summary			Not Accepted

Comments>

The entire population in the Basin is not at risk for lead exposure. We believe only those who may have some prior exposure are at risk, in addition to pregnant mothers and children under two. Your Plan needs to recognize this fact and should apply one set of remedies necessary to the vast majority of the population while developing another set for those may be at risk.

As an example, education programs for those families at risk on how to avoid further contamination make much more sense than dramatic physical remedies. Also keep in mind, the Agency for Toxic Substances Disease Registry (ATSDR) has already determined that fish consumption does not need to be curtailed from fish caught in the lateral lakes of the Coeur d'Alene River basin.

Further, the IEUBK model is also based on exposure scenarios that are unrealistically conservative. For example, it is assumed that young children are bare-foot and wear shorts and short-sleeved shirts from April through November in the River Basin, consume 25g/day of fish caught locally year round, and consume 8 g/day locally grown garden vegetables year round.

Response>>

The HHRA assesses risk for a variety of sub-populations, pathways and exposure sources and excessive risk is identified on an age-specific basis. Remedial strategies and clean up criteria developed under risk management activities will consider this information and address those populations at unacceptable risk levels. Homegrown vegetable ingestion rates are explained on page 3-48. A wet-weight vegetable ingestion rate of 5.04 g per kg body weight per day for the RME case and 0.492 g/kg-day for the CT case were selected, based on the U.S. Department of Agriculture Nationwide Food Consumption Survey (NFCS). Ingestion rates for fish are explained on page 3-52. A fish ingestion rate of 46 g/day was selected based on national fish portion sizes (USEPA 1997a) and information from a local fish consumption survey (ATSDR 1989). Further response is covered in General Response to Comments, #8a and #9a.

Misc. Input>>

385	10/16/2000	Dee Jameson	Public Draft - July 2000		2/08/2000	TG
I4		CLEAN	0-Executive Summary			Not Accepted

Comments>

The Human Health Risk Assessment (HHRA) focuses primarily on soil-lead contamination without accepting other exposure scenarios, including lead-based paint in the Silver Valley's pre-1970's homes. It further fails to compare actual blood lead levels in the Silver Valley population with those expected to be found in any population of similar economic and housing characteristics.

For example, a comparison to national and state-wide blood lead levels show that the geometric mean of BLL's for the Basin in 1999 (1-6year olds) at 5.2 mg/dl is lower than National (1991-94) low-income, pre-1946 housing BLL's of 5.5 mg/dl. The same category for percentage of children equal or greater than the 10 mg/dl (CDC standard) is also below the national level.

Response>>

The HHRA disagrees with this comment. Analysis conducted in the HHRA suggest that yard soils are a primary source of lead absorption among children both through direct contact and as a contributor to lead in house dust. Other sources including lead paint are also identified as sources to both blood and dust lead. The HHRA concludes that both sources present excessive risk and provides example analysis regarding potential cleanup criteria. Lead from paint is discussed in Section 6.3.4, and specific studies regarding lead-based paint in the Coeur d'Alene River Basin are cited. Table 6-13 shows summary statistics for lead-based paint by geographic subarea, and Figures 6-7a and 6-7b show mean interior and exterior paint lead concentrations by geographic area.

There is a divergence of opinions regarding the appropriate comparisons between the National and State-wide Lead absorption data bases with the results of the HHRA. Making actual comparisons is difficult as the scientific designs of the NHANES surveys are constructed in a way that does not permit valid comparisons with results of blood lead distributions for a given community, and the design for gathering and organization of the Basin data was not for purposes of matching the organization of the various demographic and socioeconomic strata in the NHANES III survey reports. If the Basin data was divided into the numerous categories to allow such comparisons, it would produce so few children as to make comparisons with national data meaningless. See also General Response to Comments, #2.

Misc. Input>>

Comments Summary

ID	Date	Comment By/Org	Document Version/Section	SubSection/Add'l Ref	Response Due	Response By/Type
386	10/16/2000	Dee Jameson	Public Draft - July 2000		2/08/2000	TG
I5		CLEAN	0-Executive Summary			Not Accepted

Comments>

Basin Blood lead testing needs to be done more than once a year. By taking those tests only in August when exposure levels are the greatest, results are skewed high. There should be multiple testing periods each year to get a more accurate reflection of levels/averages. Further, the State should consider following the Shoshone Natural Resources Coalition effort of "finger-prick" testing methods to reduce parents' and children's fear or apprehension of getting tested.

Response>>

Blood lead testing was purposely accomplished during the peak season, as the objective of the program is to identify children with excessive blood lead levels. There are seasonal variations in blood lead levels, but it is important to identify children at risk during the peak exposure period as these levels are of health concern. Experience at the BHSS has shown that conducting winter screens has diminished the turnout during the subsequent year when children can most benefit from the service. See further discussion under General Resonse to Comments, #2.

Misc. Input>>