Review of the National Ambient Air Quality Standards for Lead:

Policy Assessment of Scientific and Technical Information

OAQPS Staff Paper – First Draft
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PREFACE

This document is part of the Environmental Protections Agency’s (EPA’s) review of the National Ambient Air Quality Standards (NAAQS) for lead. Based on the information contained in the Agency’s Air Quality Criteria Document for Lead (October 2006; available at http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr_cd.html), this draft Staff Paper includes assessments and preliminary analyses related to:

1. air quality characterization,
2. integration and evaluation of health information,
3. human exposure analysis and health risk assessment, and
4. evaluation and analysis of information on vegetation damage and other welfare effects.

This initial draft document does not include any conclusions or recommendations with regard to potential retention or revision of the lead NAAQS.

To date, the lead NAAQS review has followed our historic approach to reviewing NAAQS, including issuance of a criteria document and a first draft staff paper. The Agency is now moving forward to implement a new, more efficient process for conducting NAAQS reviews (http://www.epa.gov/ttn/naaqs/). EPA intends to transition to that new process during the course of the lead NAAQS review.
# Table of Contents

List of Tables ............................................................................................................................... vii

List of Figures ............................................................................................................................... ix

1 INTRODUCTION ................................................................................................................ 1-1
1.1 PURPOSE ................................................................................................................... 1-1
1.2 BACKGROUND ........................................................................................................... 1-2
  1.2.1 Legislative Requirements ................................................................................... 1-2
  1.2.2 History of Lead NAAQS Reviews ....................................................................... 1-4
  1.2.3 Current Lead NAAQS Review ............................................................................ 1-4
1.3 GENERAL APPROACH AND ORGANIZATION OF THE DOCUMENT ............... 1-6
REFERENCES .................................................................................................................. 1-8

2 CHARACTERIZATION OF AMBIENT LEAD .............................................................. 2-1
  2.1 INTRODUCTION/BACKGROUND ........................................................................ 2-1
  2.2 PROPERTIES OF AMBIENT LEAD ...................................................................... 2-3
    2.2.1 Fate and Transport of Pb Particles ................................................................. 2-3
  2.3 SOURCES AND EMISSIONS TO THE ATMOSPHERE ........................................... 2-5
    2.3.1 Data sources ................................................................................................ 2-5
    2.3.2 Confidence Level for Emission Estimates ..................................................... 2-6
    2.3.3 Trends in National Emissions: 1980 to 2002 ................................................. 2-7
    2.3.4 Source Categories with Largest National Total Pb Emissions ...................... 2-8
      2.3.4.1 Industrial/Commercial/Institutional/Process Heaters ................................. 2-10
      2.3.4.2 Utility Boilers .......................................................................................... 2-10
      2.3.4.3 Mobile Sources ....................................................................................... 2-10
      2.3.4.4 Iron and Steel Foundries ........................................................................... 2-11
      2.3.4.5 Hazardous Waste Incineration/Combustion Facilities ............................... 2-12
      2.3.4.6 Primary Lead Smelting ............................................................................. 2-12
      2.3.4.7 Secondary Lead Smelting ........................................................................... 2-13
      2.3.4.8 Military Installations ................................................................................. 2-13
      2.3.4.9 Mining ..................................................................................................... 2-13
      2.3.4.10 Integrated Iron & Steel Manufacturing .................................................... 2-13
      2.3.4.11 Municipal Waste Combustors: Small & Large ........................................ 2-14
      2.3.4.12 Pressed and Blown Glass and Glassware Manufacturing ....................... 2-14
      2.3.4.13 Electric Arc Furnace Steelmaking ............................................................. 2-15
      2.3.4.14 Lead Acid Battery Manufacturing ............................................................. 2-15
3 POLICY-RELEVANT ASSESSMENT OF HEALTH EFFECTS EVIDENCE ........... 3-1
3.1 INTRODUCTION ..................................................................................................... 3-1
3.2 INTERNAL DISPOSITION – BLOOD LEAD AS DOSE METRIC ....................... 3-2
3.3 NATURE OF EFFECTS............................................................................................ 3-6
  3.3.1 Developing Nervous System ........................................................................ 3-10
  3.3.1.1 Endpoint for risk quantitation ..................................................................... 3-11
  3.3.1.2 Metric and quantitative model for risk quantitation ............................... 3-12
  3.3.2 Cardiovascular System ................................................................................. 3-15
  3.3.3 Heme Synthesis ............................................................................................ 3-16
  3.3.4 Renal System ............................................................................................... 3-16
  3.3.5 Immune System ........................................................................................... 3-17
  3.3.6 Adult Nervous System ................................................................................. 3-17
3.4 LEAD-RELATED IMPACTS ON PUBLIC HEALTH .......................................... 3-19
  3.4.1 Potentially Susceptible or Vulnerable Subpopulations ................................ 3-19
  3.4.2 Potential Public Health Impact .................................................................... 3-21
3.5 SUMMARY AND CONCLUSIONS ...................................................................... 3-23
REFERENCES ................................................................................................................ 3-26

4 CHARACTERIZATION OF HEALTH RISKS......................................................... 4-1
4.1 INTRODUCTION ..................................................................................................... 4-1
  4.1.1 Overview of Risk Assessment from Last Review ......................................... 4-2
4.2 SCOPE OF PB EXPOSURE AND RISK ASSESSMENTS ..................................... 4-4
  4.2.1 Conceptual Model for Human Health Risk Assessment ............................ 4-4
  4.2.2 Selection of Health Endpoint, Study Population, Dose-Metrics and
       Associated Concentration-Response Function .............................................. 4-7
  4.2.3 Selection of Case Study Locations .............................................................. 4-7
    4.2.3.1 Primary Pb Smelter Case Study .............................................................. 4-8
    4.2.3.2 Additional Point Source (Secondary Smelter) Case Study .................... 4-10
    4.2.3.3 Near Roadway (Urban) Location Case Study ........................................ 4-10
  4.2.4 Air Quality Scenarios Covered in the Pilot Analysis .................................... 4-12
  4.2.5 Overview of the Exposure and Risk Modeling Approach Used in the
       Pilot Analysis ..................................................................................................... 4-12
    4.2.5.1 CASAC Consultation Regarding Human Exposure and Health Risk
            Assessment ..................................................................................................... 4-13
    4.2.5.2 Child Study Population........................................................................... 4-13
    4.2.5.3 Timeframe for Current Conditions ......................................................... 4-14
    4.2.5.4 Spatial Scale and Resolution .................................................................. 4-15
    4.2.5.5 Overview of Analytical Steps .................................................................. 4-16
5 THE PRIMARY LEAD NAAQS ................................................................. 5-1
  5.1 INTRODUCTION .................................................................................. 5-1
  5.2 APPROACH .......................................................................................... 5-1
  5.3 PRIMARY LEAD STANDARD ................................................................. 5-3
    5.3.1 Level ................................................................................................. 5-3
      5.3.1.1 Sensitive Population ..................................................................... 5-4
      5.3.1.2 Maximum Safe Blood Level ......................................................... 5-5
      5.3.1.3 Nonair Contribution ...................................................................... 5-6
      5.3.1.4 Air Pb Level ................................................................................... 5-6
      5.3.1.5 Margin of Safety .......................................................................... 5-7
    5.3.2 Averaging Time, Form, and Indicator .............................................. 5-7
  5.4 POLICY OPTIONS CONSIDERED IN THE LAST REVIEW ..................... 5-8
REFERENCES ................................................................................................. 5-11

6 POLICY RELEVANT ASSESSMENT OF WELFARE EFFECTS .................. 6-1
  6.1 INTRODUCTION ..................................................................................... 6-1
  6.2 EFFECTS IN TERRESTRIAL ECOSYSTEMS ........................................ 6-1
    6.2.1 Pathways of Exposure ...................................................................... 6-3
    6.2.2 Effects of Lead on Energy Flow and Biogeochemical Cycles .......... 6-3
    6.2.3 Tools for Identifying Ecotoxicity in Terrestrial Organisms ............... 6-4
    6.2.4 Effects on Plants .............................................................................. 6-5
    6.2.5 Effects on Birds and Mammals ....................................................... 6-6
    6.2.6 Effects on Decomposers and Soil Invertebrates .............................. 6-7
    6.2.7 Summary ......................................................................................... 6-7
  6.3 EFFECTS IN AQUATIC ECOSYSTEMS .................................................. 6-8
    6.3.1 Tools for Identifying Ecotoxicity in Aquatic Organisms ................... 6-8
    6.3.2 Effects in Marine/Estuarine Ecosystems .......................................... 6-9
      6.3.2.1 Pathways of Exposure ................................................................. 6-9
      6.3.2.2 Effects on Organisms and Communities ...................................... 6-9
    6.3.3 Effects in Freshwater Ecosystems .................................................... 6-10
      6.3.3.1 Pathways of Exposure ................................................................. 6-10
      6.3.3.2 Effects at an Ecosystem Level ...................................................... 6-10
      6.3.3.3 Effects on Algae and Aquatic Plants .......................................... 6-12
      6.3.3.4 Effects on Invertebrates ............................................................. 6-13
6.3.3.5 Effects on Fish and Waterfowl ................................................................. 6-14
6.3.4 Summary .................................................................................................... 6-15
6.4 SCREENING LEVEL RISK ASSESSMENT .................................................... 6-15
  6.4.1 Overview of Analyses .............................................................................. 6-15
  6.4.2 Measures of Exposure and Effect ............................................................ 6-19
  6.4.3 National-Scale Screen and Case Studies ................................................. 6-20
    6.4.3.1 National Scale Screen ........................................................................ 6-20
    6.4.3.2 Ecologically Vulnerable Location ...................................................... 6-21
    6.4.3.3 Primary Pb Smelter .......................................................................... 6-22
    6.4.3.4 Secondary Pb Smelter ....................................................................... 6-23
    6.4.3.5 Near Roadway Non-Urban Case Study ............................................ 6-23
  6.4.4 Screening Values ..................................................................................... 6-24
    6.4.4.1 Soil Screening Values ....................................................................... 6-24
    6.4.4.2 Surface Water Screening Values ...................................................... 6-25
    6.4.4.3 Sediment Screening Values ............................................................... 6-25
  6.4.5 Results for Case Study Locations and Comparison to Screening Levels ... 6-25
    6.4.5.1 National-Scale Surface Water Screen ............................................... 6-25
    6.4.5.2 National-Scale Sediment Screen ...................................................... 6-27
    6.4.5.3 Primary Pb Smelter .......................................................................... 6-29
    6.4.5.4 Secondary Smelter .......................................................................... 6-32
    6.4.5.5 Near Roadway Non-Urban Case Study ............................................. 6-33
  6.4.6 Discussion ............................................................................................... 6-33
  6.4.7 Uncertainty and Variability .................................................................... 6-33
    6.4.7.1 Primary Pb Smelter Case Study ....................................................... 6-34
    6.4.7.2 Secondary Pb Smelter Case Study .................................................... 6-35
    6.4.7.3 Near Roadway Non-Urban Case Study ............................................. 6-35
    6.4.7.4 National-Scale Surface Water Screen .............................................. 6-35
    6.4.7.5 National-Scale Sediment Screen ...................................................... 6-36
6.5 FUTURE ANALYSES .................................................................................... 6-37
6.6 THE SECONDARY LEAD NAAQS ............................................................... 6-37
  6.6.1 Introduction ............................................................................................. 6-37
  6.6.2 Approach ................................................................................................. 6-38
REFERENCES ..................................................................................................... 6-40
List of Tables

Table 2-1. Distribution of point sources within the 2002 NEI and associated estimated emissions. ................................................................. 2-6
Table 2-2. Trend in Pb emissions (tpy) from 1980 to 2002. ................................................................. 2-8
Table 2-3. Source categories emitting greater than 5 tpy of Pb in the 2002 NEI. ........................................ 2-9
Table 2-4. Point Sources with Pb emissions in 2002 NEI greater than or equal to 5 tpy. ... 2-20
Table 2-5. Correlation among different TSP site-level statistics, 2003-2005. ........................................ 2-34
Table 2-6. FRM sites with Pb concentrations above the level of the current NAAQS, based on maximum quarterly average, 2003-2005. 2-34
Table 2-7. Comparison of numbers of sites that exceed various TSP Pb levels using different averaging times or forms, 2003-2005. 2-37
Table 2-8. Pb concentrations (µg/m3), at four sites, in different PM size fractions: 2003-2005. 2-44

Table 3-1. Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Children (CD, Table 8-5) 3-8
Table 3-2. Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Adults (CD, Table 8-6) 3-9
Table 3-3. Population subgroups with characteristics that may contribute to increased susceptibility or vulnerability to Pb health effects. 3-22

Table 4-1. Timeframe (years) reflected in the characterizations of Pb media concentrations used in the pilot risk assessment. 4-15
Table 4-2. Case study approaches for estimating media concentrations. 4-26
Table 4-3. IEUBK input parameters and basis or derivation. 4-40
Table 4-4. Projected ambient air and inhalation exposure concentrations. a 4-47
Table 4-5. Projected outdoor soil concentrations. 4-47
Table 4-6. Projected indoor dust concentrations. 4-48
Table 4-7. Projected blood Pb levels (µg/dL) for primary Pb smelter case study. 4-51
Table 4-8. Projected blood Pb levels (µg/dL) for secondary Pb smelter case study. 4-52
Table 4-9. Projected blood Pb levels (µg/dL) for near roadway (urban) case study. 4-52
Table 4-10. Performance evaluation of approaches for ambient air concentrations. 4-54
Table 4-11. Performance evaluation of approaches for outdoor soil concentrations. 4-55
Table 4-12. Performance evaluation of approaches for blood Pb levels. 4-56
Table 4-13. Projections of IQ loss for the primary Pb smelter case study - current conditions. 4-62
Table 4-14. Projections of IQ loss for primary Pb smelter case study - NAAQS attainment. 4-63
Table 4-15. Projections of IQ loss for secondary Pb smelter case study - modeled soil Pb approach. ................................................................................................................................. 4-65
Table 4-16. Projections of IQ loss for secondary Pb smelter case study - hybrid soil Pb approach. ................................................................................................................................. 4-66
Table 4-17. Projections of IQ loss for near roadway (urban) case study ........................................ 4-68
Table 4-18. Modeling elements considered in the pilot sensitivity analysis (including summary of approaches used to derive alternate approaches/inputs).......................... 4-71
Table 4-19. Summary of Sensitivity Analysis Results. ................................................................................. 4-74

Table 6-1. Models and Measurements Used for Ecological Risk Screening Assessment. ........ 6-18
Table 6-2. Soil Screening Values for Pb for Ecological Receptors............................................................ 6-25
Table 6-3. Results of Aquatic Risk Screen - Locations at which Dissolved Pb Measurements Exceed AWQC, Excluding Mining Sites. .................................................. 6-27
Table 6-4. Concentrations of Total Pb in Sediments at Locations Near or Matching the 15 Sites at which Dissolved Pb Concentrations Exceeded the AWQC, Excluding Mining Sites. ........................................................................................................... 6-28
Table 6-5. HQs for Soils for Primary Pb Smelter Case Study. ................................................................. 6-30
Table 6-6. HQs Calculated for Surface Waters for Primary Pb Smelter Case Study.................... 6-31
Table 6-7. HQs Calculated for Sediments in Surface Waters for Primary Pb Smelter Case Study. ................................................................................................................................. 6-32
Table 6-8. HQs Calculated for Soils for Secondary Pb Smelter Case Study.a .................................. 6-33
Table 6-9. HQs Calculated for Soils Near Roadway Non-Urban Case Study ........................................ 6-33
List of Figures

Figure 2-1. Principal pathways of human and ecological exposure to Pb ........................................ 2-2
Figure 2-2. Emission Density of All Pb Sources in the 2002 NEI .................................................. 2-17
Figure 2-3. Emission Density of Mobile and Non Point Sources of Pb in 2002 NEI ........................ 2-18
Figure 2-4. Geographic distribution of point sources with >1 tpy Pb emissions in 2002 NEI ........... 2-19
Figure 2-5. Change in the number of Pb TSP monitoring sites from 1980 to 2005 ..................... 2-23
Figure 2-6. Pb TSP monitoring sites: 2003-2005 ....................................................................... 2-24
Figure 2-7. Pb PM2.5 (STN) monitoring sites .............................................................................. 2-25
Figure 2-8. Pb PM2.5 (IMPROVE) monitoring sites ................................................................. 2-26
Figure 2-9. Pb PM10 (NATTS) monitoring sites network ......................................................... 2-27
Figure 2-10. Airborne Pb (TSP) concentrations, averaged across continuously operating
monitoring sites: 1980-2002 ................................................................................................. 2-28
Figure 2-11. Distribution of TSP Pb concentrations (represented by 4 different statistics)
at monitoring sites, 2003-2005 ......................................................................................... 2-30
Figure 2-12. Distribution of monitor level TSP Pb annual mean concentrations for
source-oriented and not sourced-oriented monitors, 2003-2005 ........................................ 2-31
Figure 2-13. Site level TSP Pb, annual mean concentrations, 2003-2005 ................................. 2-32
Figure 2-14. Site level TSP Pb, maximum quarterly mean concentrations, 2003-2005 ................ 2-33
Figure 2-15. Monthly average TSP Pb concentrations at 4 example monitor sites, 2003-2005 2-36
Figure 2-16. Distribution of PM2.5 Pb concentrations (represented by four different
statistics) at STN sites, 2003-2005 .......................................................................................... 2-38
Figure 2-17. Site level ‘urban’ (STN) PM2.5 Pb annual means, 2003-2005 ............................... 2-39
Figure 2-18. Distribution of PM10 Pb concentrations (represented by four different
statistics), 2003-2005 ............................................................................................................ 2-41
Figure 2-19. Monitor level PM10 Pb annual means, 2002-2005 .............................................. 2-42
Figure 2-20. Comparison of national mean and median monitor level Pb, annual means
for different size cut PM networks, 2002-2005 ...................................................................... 2-43
Figure 2-21. Modeled soil concentrations of Pb in the South Coast Air Basin of
California based on four re-suspension rates (A) ....................................................................... 2-52
Figure 2-22. Pb concentrations in sediment samples in 12 Michigan lakes ............................... 2-54
Figure 2-23. Spatial distribution of dissolved lead in surface water (N = 3445).
[CD, Figure AX7-2.2.7] ........................................................................................................ 2-56
Figure 2-24. Spatial distribution of total lead in bulk sediment <63 µm (N = 1466).
[CD, Figure AX7-2.2.9] ........................................................................................................ 2-57

Figure 4-1. Conceptual model for Pb human health risk assessment ........................................... 4-5
Figure 4-2. Overview of analysis approach for the pilot analysis .............................................. 4-17
Figure 4-3. Procedure for Generating Population Blood Pb Distributions................................ 4-43

Figure 6-1. Overview of Ecological Screening Assessment...................................................... 6-17
1 INTRODUCTION

1.1 PURPOSE

This first draft Staff Paper, prepared by staff in the U.S. Environmental Protection Agency’s (EPA) Office of Air Quality Planning and Standards (OAQPS) (henceforth referred to as “staff”), contains an initial evaluation of the policy implications of the key studies and scientific information contained in the final document, *Air Quality Criteria for Lead* (USEPA, 2006a; henceforth referred to as the CD), prepared by EPA’s National Center for Environmental Assessment (NCEA). This first draft also presents and interprets results from several initial or pilot analyses (e.g., human exposure analyses, human health risk assessments and environmental assessments) that will inform EPA's current review of the national ambient air quality standards (NAAQS) for lead (Pb). This draft Staff Paper, however, is an initial product intended to provide an initial assessment of the issues for the Clean Air Scientific Advisory Committee (CASAC) and public review and comment and, further, to solicit comment on the pilot exposure and risk assessments, as well as on plans for the full-scale assessments. This draft document does not present any conclusions or recommendations with regard to potential retention or revision of the primary (health-based) and secondary (welfare-based) Pb NAAQS.

The policy assessment to be presented in the final version of this document is intended to help “bridge the gap” between the scientific assessment contained in the CD and the judgments required of the EPA Administrator in determining whether it is appropriate to retain or revise the NAAQS for Pb. In conducting this assessment, staff is aware of the dramatic alteration in the basic patterns of air lead emissions in the U.S. since the listing of Pb as a criteria pollutant and the 1978 promulgation of the Pb NAAQS. The reduction of Pb in gasoline has resulted in orders-of-magnitude reductions in airborne emissions of Pb, and a significant shift in the types of sources with the greatest Pb emissions. An additional circumstance that has changed since 1978 is the enactment in 1990 of the Clean Air Act Amendments, in which Pb compounds were listed as hazardous air pollutants under Section 112. Section 112, as amended in 1990, requires EPA to establish technology-based (or "MACT") emission standards for those listed source categories emitting Pb compounds, and to establish risk-based standards, as necessary, for those categories of sources for which EPA has issued MACT standards. Given the significantly changed circumstances since Pb was listed in 1976, we will evaluate the status of Pb as a criteria
pollutant\(^1\) in light of currently available information and assess whether revocation of the standard is an appropriate option for the Administrator to consider.

In evaluating the adequacy of the current standard and policy alternatives, in the next draft of this document, emphasis will be placed on identifying those conclusions and uncertainties in the available scientific literature that are most pertinent to the indicator\(^2\), averaging times, forms\(^3\), and levels for primary (health-based) and secondary (welfare-based) standards, which must be considered collectively in evaluating the health and welfare protection afforded by Pb standards. The final version of this document will evaluate the policy implications of the key studies and scientific information contained in the CD, identify the critical elements to be considered in the current review of the NAAQS for Pb, and present factors relevant to the evaluation of current primary and secondary Pb NAAQS, as well as conclusions and identification of options for the Administrator’ consideration.

While this draft Staff Paper should be of use to all parties interested in the Pb NAAQS review, it is written with an expectation that the reader has some familiarity with the technical discussions contained in the CD.

1.2 BACKGROUND

1.2.1 Legislative Requirements

Two sections of the Clean Air Act (Act) govern the establishment and revision of the NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify and list each air pollutant that “in his judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health and welfare” and whose “presence . . . in the ambient air results from numerous or diverse mobile or stationary sources” and to issue air quality criteria for those that are listed. Air quality criteria are to “accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in ambient air . . . “

Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate “primary” and “secondary” NAAQS for pollutants listed under section 108. Section 109(b)(1) defines a primary standard as one “the attainment and maintenance of which in the judgment of the Administrator, based on [air quality] criteria and allowing an adequate margin of safety, are

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\(^1\) Section 108 of the Clean Air Act states that the Administrator “shall, from time to time … revise” the criteria pollutant list.

\(^2\) The “indicator” of a standard designates the chemical associated with the standard.

\(^3\) The “form” of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains the standard.
requisite to protect the public health.” A secondary standard, as defined in Section 109(b)(2), must “specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air.”

The requirement that primary standards include an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified. *Lead Industries Association v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir 1980), *cert. denied*, 449 U.S. 1042 (1980); *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1186 (D.C. Cir. 1981), *cert. denied*, 455 U.S. 1034 (1982). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary standards that include an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree.

In selecting a margin of safety, the EPA considers such factors as the nature and severity of the health effects involved, the size of the sensitive population(s) at risk, and the kind and degree of the uncertainties that must be addressed. The selection of any particular approach to providing an adequate margin of safety is a policy choice left specifically to the Administrator’s judgment. *Lead Industries Association v. EPA*, *supra*, 647 F.2d at 1161-62.

In setting standards that are “requisite” to protect public health and welfare, as provided in section 109(b), EPA’s task is to establish standards that are neither more nor less stringent than necessary for these purposes. In so doing, EPA may not consider the costs of implementing the standards. See generally *Whitman v. American Trucking Associations*, 531 U.S. 457, 471, 475-76 (2001).

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4 The legislative history of section 109 indicates that a primary standard is to be set at “the maximum permissible ambient air level . . . which will protect the health of any [sensitive] group of the population,” and that for this purpose “reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group.” S. Rep. No. 91-1196, 91st Cong., 2d Sess. 10 (1970)

5 Welfare effects as defined in section 302(h) (42 U.S.C. 7602(h)) include, but are not limited to, “effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being.”
Section 109(d)(1) of the Act requires that “not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards... and shall make such revisions in such criteria and standards and promulgate such new standards as may be appropriate...” Section 109(d)(2) requires that an independent scientific review committee “shall complete a review of the criteria... and the national primary and secondary ambient air quality standards... and shall recommend to the Administrator any new... standards and revisions of existing criteria and standards as may be appropriate...” Since the early 1980's, this independent review function has been performed by the Clean Air Scientific Advisory Committee (CASAC) of EPA’s Science Advisory Board.

1.2.2 History of Lead NAAQS Reviews

On October 5, 1978 the EPA promulgated primary and secondary NAAQS for lead under section 109 of the Act (43 FR 46246). Both primary and secondary standards were set at a level of \(1.5 \mu g/m^3\) as a quarterly average (maximum arithmetic mean averaged over a calendar quarter). This standard was based on the 1977 *Air Quality Criteria for Lead* (USEPA, 1977).

The most recent review was initiated in the mid 1980s. The scientific assessment for that review is described in the 1986 *Air Quality Criteria for Lead* (USEPA, 1986a), the associated Addendum (USEPA, 1986b) and the 1990 Supplement (USEPA, 1990a). As part of the review, the Agency designed and performed human exposure and health risk analyses (USEPA, 1989), the results for which were presented in the 1990 Staff Paper (USEPA, 1990b). Based on the scientific assessment and the human exposure and health risk analyses, the 1990 Staff Paper presented options for the Pb NAAQS level in the range of 0.5 to 1.5 \(\mu g/m^3\), and suggested the second highest monthly average in three years for the form (USEPA, 1990b). After consideration of the documents developed during the review and the significantly changed circumstances since Pb was listed in 1976, as noted above, the Agency did not propose any revisions to the 1978 Pb NAAQS. In a parallel effort, the Agency developed the broad, multi-program, multimedia, integrated *U.S. Strategy for Reducing Lead Exposure* (USEPA, 1991). As part of implementing this strategy, the Agency focused efforts primarily on regulatory and remedial clean-up actions aimed at reducing Pb exposures from a variety of non-air sources judged to pose more extensive public health risks to U.S. populations, as well as on actions to reduce Pb emissions to air.

1.2.3 Current Lead NAAQS Review

EPA initiated the review of the air quality criteria for Pb on November 9, 2004 with a general call for information (69 FR 64926). A project work plan (USEPA, 2005a) for the preparation of the CD was released in January 2005 for CASAC and public review. EPA held a
series of workshops in August 2005, with invited recognized scientific experts, to discuss initial
draft materials that dealt with various lead-related issues being addressed in the Pb air quality
criteria document. These workshops helped to inform the preparation of the first draft CD
(USEPA, 2005b), which was released for CASAC and public review in December, 2005 and
discussed at the CASAC meeting held on February 28-March 1, 2006.

A second draft CD (USEPA, 2006b) was released for CASAC and public review in May
2006, and discussed at the CASAC meeting on June 28, 2006. A subsequent draft of Chapter 7 -
Integrative Synthesis (Chapter 8 in the final CD), released on July 31, 2006, was discussed at an
August 15, 2006 CASAC teleconference. The final CD was released on September 30, 2006
(USEPA, 2006a). While the CD focuses on new scientific information available since the last
review, it appropriately integrates that information with scientific criteria from previous reviews.

In February, 2006, EPA released the Plan for Review of the National Ambient Air Quality
Standards for Lead (USEPA 2006c) that described Agency plans and timeline for reviewing the
air quality criteria, developing human exposure and risk assessments and an ecological risk
assessment, preparing a policy assessment, and developing the regulatory proposal and final
rulemaking.

In May, 2006, EPA released for CASAC and public review a draft Analysis Plan for
Human Health and Ecological Risk Assessment for the Review of the Lead National Ambient Air
Quality Standards (USEPA, 2006d) which was discussed at the June 29, 2006 CASAC meeting.
CASAC panel members’ views were received at and subsequent to the meeting (Henderson,
2006), and considered in the implementation of the human health and ecological risk
assessments, the pilot phase of which is described in this first draft Staff Paper. As described in
the May 2006 plan, the risk assessments are being performed in two phases: 1) pilot and 2) full-
scale. With consideration of CASAC and public comments received on this document and the
analyses described within, staff plans to develop and perform full-scale assessments.6 The full-
scale assessments will be presented in the second draft of this document for public and CASAC
review. Based on the scientific and technical findings described therein, the second draft of this
document will present initial conclusions and alternative policy options regarding the Pb
NAAQS. Comments received during CASAC and public review of the second draft will be
considered in preparation of the final document.

The schedule for completion of this review is governed by a judicial order resolving a
lawsuit filed in May 2004, alleging that EPA had failed to complete the current review within the

6 As discussed in Section 6.1, we do not at this time anticipate having funding to perform additional
quantitative ecological risk assessment work for this review.
The order that now governs this review, entered by the court on September 14, 2005, provides that EPA will prepare the initial draft Staff Paper not later than January 1, 2007, and will finalize it no later than November 1, 2007. The order also specifies that EPA sign, for publication, notices of proposed and final rulemaking concerning its review of the Pb NAAQS no later than May 1, 2008 and September 1, 2008, respectively. EPA published a series of interim target dates in its Plan for Review of the Pb NAAQS (USEPA 2006c) that are designed to ensure that these deadlines will be met. With regard to the Staff Paper, this includes release of a second draft document in June 2007, followed by CASAC and public review, and a final document in September 2007. There is also an interim target date for a proposed rulemaking in February 2008.

1.3 GENERAL APPROACH AND ORGANIZATION OF THE DOCUMENT

The final version of this document will take into account the scientific evidence reviewed in the CD and will include: 1) the results of air quality analyses, human exposure and health risk assessments, and environmental assessments; 2) an overall evaluation of the adequacy of the current primary and secondary NAAQS; and 3) conclusions pertaining to a range of policy choices available to address public health and welfare effects associated with exposure to ambient Pb resulting from emissions to the ambient air. This first draft Staff Paper includes discussion of the scientific evidence reviewed in the CD, as well as preliminary quantitative analyses based on available emissions and air quality information, information on deposition and distribution of ambient Pb in other media, and estimated health and environmental risks related to exposure to ambient Pb concentrations resulting from Pb emitted into the ambient air.

Following this introductory chapter, this draft Staff Paper is organized into three main parts: the characterization of ambient Pb; Pb-related health effects and primary Pb NAAQS; and Pb-related welfare effects and secondary Pb NAAQS. The content of these parts is discussed more fully below.

The characterization of ambient Pb is presented in Chapter 2 and includes information on Pb properties, current Pb air quality patterns, historic trends, and background levels. In recognition of the multimedia nature of Pb and the distribution into other media of Pb emitted into the air, Chapter 2 also includes information on Pb in media other than air including outdoor dust, soil, surface water and sediment. This chapter provides a frame of reference for exposure and risk analyses and subsequent discussion of the Pb NAAQS and alternative forms of Pb standards.

Chapters 3 through 5 comprise the second main part of this document, dealing with human health and primary standards. Chapter 3 presents an overview of key policy-relevant
health effects evidence, major health-related conclusions from the CD, and an examination of
issues related to the quantitative assessment of health risks. Chapter 4 describes the scope and
methods used in conducting human exposure and health risk assessments and presents initial
results from those assessments. Chapter 5 includes a preliminary discussion of the current
primary standard. This first draft of the Staff paper begins the discussion of the current standard,
buts does not evaluate the standard in light of new information since the last review; that
discussion will be included in the second draft.

Chapter 6 comprises the third main part of this document. Chapter 6 presents a policy-
relevant assessment of Pb welfare effects evidence and describes the scope and methods used in
conducting environmental risk assessments, as well as initial results from those assessments.
This chapter also includes a preliminary discussion of the current secondary standard, but as with
the primary standard, an evaluation of the current secondary standard will be included in the
second draft of this document.
REFERENCES


U.S. Environmental Protection Agency. (2006c) Plan for Review of the National Ambient Air Quality Standards for Lead. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available online at:
http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr_pd.html

http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr_pd.html
2 CHARACTERIZATION OF AMBIENT LEAD

2.1 INTRODUCTION/BACKGROUND

As described in the CD, “The multimedia aspects of Pb exposure can be seen in that Pb emissions to the air contribute to Pb concentrations in water, soil and dusts; Pb in soil and dust also can make important contributions to Pb concentrations in ambient air” (CD, p. 3-1).

Exposures to Pb emitted into the air occur via multiple pathways. As illustrated in Figure 2-1, pathways that are directly relevant to a review of the NAAQS include both newly emitted Pb from currently operating sources, and Pb emitted in the past, either from currently operating sources or historic sources, which are collectively referred to as “policy-relevant sources”.

Consequently, this document considers both airborne Pb as it contributes to human exposures through direct inhalation of particles containing Pb, and also Pb deposited to dusts, soil and other environmental media as it contributes to human exposures through ingestion, and to various ecological exposures. Further, Pb, once deposited, may also be re-entrained into the air. In addition, Figure 2-1 shows that people and the environment are also exposed to Pb that originates from non-air sources, including Pb-paint or drinking water distribution systems. For purposes of this review, the Pb from these non-air sources is collectively referred to as “policy-relevant background”.

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1 This categorization of policy-relevant and background sources is not intended to convey any particular policy decision at this stage regarding the Pb standard. Rather, it is simply intended to convey an area of interest to this review.
Figure 2-1. Principal pathways of human and ecological exposure to Pb. Among the policy-relevant pathways, heavy arrows indicate the predominant human exposures.

This chapter generally characterizes airborne Pb and deposited atmospheric Pb in terms of physical and chemical properties, measurement methods, recent concentrations and trends, and relationships with human and ecological exposure. The information provided here is intended to provide context for information presented in subsequent chapters, including the human exposure and risk assessments (Chapter 4) and the ecological risk assessment (Chapter 6). Additionally, in the second draft of this document, the analyses presented in this chapter are intended to be informative to policy considerations regarding the primary and secondary Pb standards. The information presented in this chapter was drawn from the CD and additional analyses of data from various Pb monitoring networks, emissions inventories, and modeling activities.

Section 2.2 presents information on the basic physical and chemical properties of airborne Pb, including a discussion of environmental fate and transport. Section 2.3 presents information on the sources of atmospheric Pb, and their emissions, both on a cumulative national basis, and on an individual source basis. Section 2.4 presents information on the methods used to measure ambient Pb, and on Pb concentrations, trends, and spatial patterns in the U.S. Section 2.5 describes currently available information on atmospheric deposition of Pb. Sections 2.6 and
2.7 present information on Pb in other media pertinent to human and ecological exposure including outdoor dust, soil, surface waters and sediments.

2.2 PROPERTIES OF AMBIENT LEAD

Due to its physicochemical properties, Pb exists in the environment predominantly in solid form. Consequently upon emission into the air, Pb deposits onto surfaces or exists in the atmosphere as a component of atmospheric aerosol (CD, Section 2.1). The various Pb compounds that are naturally occurring in the environment or are introduced by anthropogenic activities include oxides, chlorides (or other halides), sulfates, and sulfides (see CD, Table 2-5). A more complete discussion of the physical and chemical properties of Pb and Pb compounds is provided in the CD (Section 2.1). The Pb NAAQS pertains to the Pb content of all Pb compounds that may be emitted to air (see Section 2.4.1 for discussion of collection and analysis methods).

The relative presence of Pb among the various environmentally occurring compounds influences its distribution within the environment, and the relative bioavailability of these compounds has implications for human and ecological exposures and risks (CD, Sections 4.2.1, 8.1.3 and 8.2.3). With regard to human exposures and risk, this is reflected in the exposure modeling described in Chapter 4. Lead speciation and bioavailability are discussed further with regard to environmental effects in Chapter 6.

2.2.1 Fate and Transport of Pb Particles

The atmosphere is the major environmental transport pathway for anthropogenic Pb (CD, p 2-52). Lead can be transported in the atmosphere and undergo secondary dispersal via the deposition and re-suspension of particles containing Pb. As described in the CD (Section 2.3.1) and in Section 2.4.3 of this document, airborne Pb particles generally have a bimodal distribution with the greater mass of Pb found in the fine fraction (CD, p. 2-52). Since small particles are much slower to deposit than larger particles, Pb can be transported great distances in the atmosphere. Thus, Pb is widely dispersed, as evidenced by detection of Pb even in the most remote places such as the arctic region (CD, pp. 2-52, 3-3).

Airborne concentrations of species emitted from a point source are frequently described by a Gaussian distribution. Gaussian models are, in general, reasonably accurate for small geographic scales, e.g., within ~50 km of the source (CD, p. 2-53). The rate and direction of dispersion are dependent both on pollutant characteristics and meteorological conditions. Important meteorological factors influencing dispersion include wind speed, surface roughness, inversion frequency, inversion duration, and temperature. Results are site specific. For long range transport modeling, Lagrangian trajectory or Eulerian grid models are commonly
employed. These models determine how a parcel of air moves relative to the moving fluid and a fixed coordinate system, respectively. Retrospective air mass trajectories based on hybrid models are also used. Results included a finding of airborne Pb in a less industrial country originating from emissions in several distant countries (CD, p. 2-54).

Wet and dry deposition are the ultimate paths by which Pb particles are removed from the atmosphere. Dry deposition is the process by which Pb particles are delivered from the atmosphere onto surfaces in the absence of precipitation. Factors that govern dry deposition are the level of atmospheric turbulence, especially in the layer nearest the ground, particle size distributions and density, and the nature of the surface itself, such as smooth or rough. In the commonly used model formulation for dry deposition, it is assumed that the dry deposition flux is directly proportional to the local concentration of the pollutant species, at some reference height above the surface (e.g., 10 m or less), multiplied by the deposition velocity (CD, p. 2-55). The concentration is computed by the dispersion models mentioned above, depending on local versus regional or global applications. Estimates of dry deposition velocity constitute the primary output of a large number of dry deposition models that have been developed during the past ten years and most of these rely on so-called “resistance schemes.” The advantage of this deposition velocity representation is that all the complexities of the dry deposition process are bundled in a single parameter, but the disadvantage is that because this parameter addresses a variety of processes, it is difficult to specify properly. A large range of Pb deposition velocities (0.05 to 1.3 cm/s) has been reported (CD, pp. 2-55 to 2-57 and Table 2-21).

Wet deposition, or the delivery of a pollutant to the ground in precipitation, is the process by which airborne pollutants are scavenged by precipitation and removed from the atmosphere. The flux of a depositing species can be defined as the product of the rate of precipitation and the concentration of the chemical species in the precipitation (CD, pp. 2-57 to 2-59). Wet deposition, is affected by: 1) nucleation scavenging (removal by direct incorporation into new cloud droplets); 2) in-cloud scavenging (removal by incorporation into existing cloud droplets); and 3) precipitation washout (removal by rain as it is falling to the ground). The size of particles can influence wet deposition rates. Large particles are scavenged by precipitation more efficiently than smaller particles (CD, p. 2-59). Lead, beyond the influence of individual sources, is found primarily in the submicron size range, and consequently does not undergo wet deposition as easily as many of the crustal elements (CD, p. 2-59). Models vary in how they treat wet deposition. Gaussian models focus solely on washout aspects, mainly because this process is dominant within the 50 km limit of model applicability. Regional and global models have more comprehensive treatment of wet deposition. Lead concentrations in precipitation have shown a pronounced downward trend from the 1970s into the 1990s, presumed primarily due to the phase-out of leaded fuel (CD, pp. 2-60 to 2-61 and Table 2-22).
The re-suspension of soil-bound Pb particles and contaminated road dust can be a significant source of airborne Pb (CD, Section 2.3.3, and p. 2-62). Studies of emissions in southern California indicate that Pb in re-suspended road dust may represent between 40% and 90% of Pb emissions in some areas (CD, p. 2-65). Lead concentrations in suspended soil and dust, however, vary significantly (CD, p. 2-65). In general, the main drivers of particle re-suspension are typically mechanical stressors such as vehicular traffic, construction and agricultural operations, and to a lesser extent, the wind. Understanding the physics of re-suspension from natural winds requires analyzing the wind stresses on individual particles and although this analysis can be accurate on a small scale, predicting re-suspension on a large scale generally focuses on empirical data for soil movement due to three processes: saltation, surface creep, and suspension (CD, pp. 2-62 to 2-63). Further, rather than a continuous process, re-suspension may occur as a series of events. Short episodes of high wind speed, dry conditions, and other factors conducive to re-suspension may dominate annual averages of upward flux (CD, p. 2-65). These factors complicate emissions estimates.

2.3 SOURCES AND EMISSIONS TO THE ATMOSPHERE

The purpose of this section is to describe the available information on sources of Pb into the ambient air. The section does not provide a comprehensive list of all sources of Pb, nor does it provide estimates of emission rates or emission factors for all source categories, since such information is available for only a limited number of sources. Rather, the discussion here is intended to identify the larger source categories, either on a national or local scale, and provide some characterization of their emissions and distribution within the U.S. The data sources for this information are described in Section 2.3.1. Limitations of and confidence in the information is discussed in Section 2.3.2. Section 2.3.3 summarizes temporal trends of major source sectors, and Section 2.3.4 summarizes estimates of 2002 national emissions totals for the larger source categories. And lastly, Section 2.3.5 describes the geographic distribution of emissions based on the 2002 estimates.

2.3.1 Data sources

The Pb emissions information presented here is drawn largely from EPA’s National Emissions Inventory (NEI). The NEI is based on inputs from State, Tribal and local air pollution agencies and data obtained during the preparation of technical support information for the EPA’s hazardous air pollutant regulatory programs. The Agency is currently developing version 2.1 of the NEI for 2002 (http://www.epa.gov/ttn/chief/net/2002inventory.html). The information presented in this draft document is based on that version (USEPA, 2006), and comments
received on the NEI and on this draft document will be considered in developing such
information for the second draft of this document.

There are some 13,000 industrial, commercial or institutional sources in the U.S. that
contain one or more processes that emit Pb to the atmosphere and whose Pb emission estimates
are included in the 2002 NEI (Table 2-1). Most of these sources emit < 0.1 tons per year (tpy).

There are approximately 1300 point sources of Pb in the NEI with estimates of emissions greater
than or equal to 0.1 tpy and these point sources, combined, emit 1037 tpy, or 94% of the Pb point
source emissions. In other words, 94% of Pb point source emissions are emitted by the largest
10% of the sources.

In the NEI, emissions estimates for some of the point sources are in terms of mass of Pb
compounds, whereas the non point source and mobile source emissions estimates are in terms of
mass of the Pb only. For the point sources, approximately 80% are reported as mass of Pb and
most of the other 20% are reported as mass of Pb compounds. The high molecular weight of Pb
(as compared to elements with which it is associated in Pb compounds), however, reduces the
impact of this reporting inconsistency.

Table 2-1. Distribution of point sources within the 2002 NEI and associated estimated
emissions.

<table>
<thead>
<tr>
<th>Emissions Range (tpy)</th>
<th>Number of Sources</th>
<th>Total Emissions (tpy)</th>
<th>Average Emissions per Source (tpy)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 0.1</td>
<td>11,822</td>
<td>73</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>0.1 to 1.0</td>
<td>1038</td>
<td>355</td>
<td>0.3</td>
</tr>
<tr>
<td>1.0 to 5</td>
<td>204</td>
<td>394</td>
<td>2</td>
</tr>
<tr>
<td>&gt; 5</td>
<td>26</td>
<td>288</td>
<td>10</td>
</tr>
<tr>
<td>Summary</td>
<td>13,087</td>
<td>1114</td>
<td></td>
</tr>
</tbody>
</table>

2.3.2 Confidence Level for Emission Estimates

The comprehensiveness of emission inventories depends upon what is known regarding
which source types emit Pb, their locations and their operating characteristics, as well as the
reporting of this information to the inventory. As described in Section 2.3.1, the NEI relies on
information that is available from a variety of sources for this information. There are numerous
steps, each with its own uncertainties, associated with the development of this information for
use in the emissions inventory. First, the categories emitting Pb must be identified. Second, the
sources’ processes and control devices must be known. Third, the activity throughputs and operating schedules of these sources must be known. Finally, we must have emission factors to relate emissions to the operating throughputs, process conditions and control devices. The process, control device, throughputs and operating schedules are generally available for each source. However, the emission factors represent average emissions for a source type and average emissions may differ significantly from source to source. More information on emission factors and the estimation of emissions is found in the introduction to EPA’s *Compilation of Air Pollutant Emissions Factors* (USEPA, 2006b). Further information on emission factors is available at: [http://www.epa.gov/ttn/chief/ap42/](http://www.epa.gov/ttn/chief/ap42/).

The process of identifying sources that emit Pb into the air has been ongoing since before the Clean Air Act of 1970. The NEI includes inventory estimates for Pb sources with some exceptions, including re-suspended road dust, biomass burning and trace levels of Pb in motor fuel and lubricating oil. For example, while Pb has not been added to automobile gasoline for two decades, there are still deposits of Pb near roads. This Pb can be entrained into the air by disturbance of soil near the roads or by burning of biomass materials that are near roads (CD, Sections 2.3.3 and 2.2.1). We have not yet developed estimates for the NEI of Pb emissions associated with re-suspension of Pb still residing in roadway dust and soil. Likewise, we have not developed NEI estimates for Pb emitted from burning of biomass materials near roads or how much of the Pb that accumulated away from roads due to transport and deposition is still subject to emissions from forest fires or mechanical disturbance of soils. As described in the CD (Section 8.2.2), re-suspension of soil bound Pb and contaminated road dust may be a significant source of airborne Pb, however, quantitative estimates for this process remains an area of significant uncertainty. Also, while Pb is no longer added to gasoline or diesel fuel, it is still present as a trace contaminant in both fuels and there are trace amounts of Pb in lubricating oils. These are not reflected in the emissions inventory.

### 2.3.3 Trends in National Emissions: 1980 to 2002

Table 2-2 shows a downward trend in the fuel combustion, industrial process and solid waste sectors from 1980 through 2002, as well as the dramatic reduction in Pb emissions in the transportation sector due to the removal of Pb from gasoline. While the most dramatic reductions occurred prior to 1990, Pb emissions were further reduced by 68% (from 5,000 to 1,600 tpy) between 1990 and 2002 (USEPA 1990; 2002 NEI). The greatest emission reductions were from mobile sources, primary and secondary Pb and copper smelting, pulp and paper manufacturing, inorganic paint pigment production and steel wire products. As discussed in the CD (Section 2.2.4), reductions in mobile sources include some associated with the latter period.
of the “phase-out” of leaded gasoline. From a national inventory perspective, the categories that have the largest emissions in the 2002 NEI are discussed briefly in Section 2.3.4.

Table 2-2. Trend in Pb emissions (tpy) from 1980 to 2002.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Transportation</td>
<td>64,706</td>
<td>18,973</td>
<td>1,197</td>
<td>564</td>
<td>392</td>
</tr>
<tr>
<td>Fuel Combustion</td>
<td>4,299</td>
<td>515</td>
<td>500</td>
<td>490</td>
<td>425</td>
</tr>
<tr>
<td>Industrial Processes</td>
<td>3,938</td>
<td>2,531</td>
<td>2,474</td>
<td>2,271</td>
<td>736</td>
</tr>
<tr>
<td>Solid Waste</td>
<td>1,210</td>
<td>871</td>
<td>804</td>
<td>604</td>
<td>87</td>
</tr>
<tr>
<td>Total</td>
<td>74,153</td>
<td>22,890</td>
<td>4,975</td>
<td>3,929</td>
<td>1,640</td>
</tr>
</tbody>
</table>


2.3.4 Source Categories with Largest National Total Pb Emissions

Table 2-3 shows the sources of Pb emissions estimated to emit more than 5 tons per year of Pb in the 2002 NEI. The main sources of emissions in the 2002 NEI are comprised primarily of combustion-related emissions and industrial process-related emissions. Point source emissions account for about 68% of the national Pb emissions in the 2002 NEI. The point source emissions are roughly split between combustion and industrial processes; non road sources (general aviation aircraft – leaded fuel) accounts for 24%.

Presence of a source category on this list does not necessarily provide an indication of the significance of the emissions from individual sources within the source category. A source category, for example, may be composed of many small (i.e., low-emitting) sources, or of just a few very large (high-emitting) sources. Such aspects of a source category, which may influence its potential for human and ecological impacts, are included in the following short descriptions of the largest source categories identified in Table 2-3.
### Table 2-3. Source categories emitting greater than 5 tpy of Pb in the 2002 NEI.

<table>
<thead>
<tr>
<th>Source Category Name</th>
<th>2002 Total Emissions (TPY)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALL CATEGORIES b</td>
<td>1,640</td>
</tr>
<tr>
<td>Mobile sources (Leaded Aviation Fuel)</td>
<td>392</td>
</tr>
<tr>
<td>Utility Boilers</td>
<td>221</td>
</tr>
<tr>
<td>Industrial/Commercial/ Institutional Boilers &amp; Process Heaters</td>
<td>191</td>
</tr>
<tr>
<td>Iron and Steel Foundries</td>
<td>110</td>
</tr>
<tr>
<td>Primary Lead Smelting</td>
<td>59</td>
</tr>
<tr>
<td>Secondary Lead Smelting</td>
<td>46</td>
</tr>
<tr>
<td>Mining</td>
<td>38</td>
</tr>
<tr>
<td>Military Installations</td>
<td>33</td>
</tr>
<tr>
<td>Municipal Waste Combustors</td>
<td>33</td>
</tr>
<tr>
<td>Electric Arc Furnaces (EAF)</td>
<td>32</td>
</tr>
<tr>
<td>Integrated Iron &amp; Steel Manufacturing</td>
<td>32</td>
</tr>
<tr>
<td>Pressed and Blown Glass and Glassware Manufacturing</td>
<td>31</td>
</tr>
<tr>
<td>Lead Acid Battery Manufacturing</td>
<td>25</td>
</tr>
<tr>
<td>Secondary Nonferrous Metals</td>
<td>23</td>
</tr>
<tr>
<td>Hazardous Waste Incineration</td>
<td>22</td>
</tr>
<tr>
<td>Solid Waste Incineration</td>
<td>22</td>
</tr>
<tr>
<td>Primary Copper Smelting</td>
<td>22</td>
</tr>
<tr>
<td>Portland Cement Manufacturing</td>
<td>22</td>
</tr>
<tr>
<td>Primary Metal Products Manufacturing</td>
<td>20</td>
</tr>
<tr>
<td>Pulp and Paper Production</td>
<td>10</td>
</tr>
<tr>
<td>Industrial Inorganic Chemical Manufacturing</td>
<td>10</td>
</tr>
<tr>
<td>Sewage Sludge Incineration</td>
<td>10</td>
</tr>
<tr>
<td>Synthetic Rubber Manufacturing</td>
<td>9</td>
</tr>
<tr>
<td>Secondary Aluminum Production</td>
<td>9</td>
</tr>
<tr>
<td>Farm Machinery and Equipment Manufacturing</td>
<td>8</td>
</tr>
<tr>
<td>Secondary Copper Smelting</td>
<td>8</td>
</tr>
<tr>
<td>Stationary Reciprocating Internal Combustion Engines</td>
<td>8</td>
</tr>
<tr>
<td>Industrial Machinery and Equipment: Finishing Operations</td>
<td>7</td>
</tr>
<tr>
<td>Nonferrous Foundries</td>
<td>7</td>
</tr>
<tr>
<td>Ferroalloys Production</td>
<td>7</td>
</tr>
<tr>
<td>Residential Heating</td>
<td>6</td>
</tr>
<tr>
<td>Miscellaneous Metal Parts &amp; Products (Surface Coating)</td>
<td>6</td>
</tr>
<tr>
<td>Primary Nonferrous Metals--Zinc, Cadmium and Beryllium</td>
<td>5</td>
</tr>
<tr>
<td>Engine Test Facilities</td>
<td>5</td>
</tr>
<tr>
<td>Coke Ovens</td>
<td>5</td>
</tr>
<tr>
<td>Surface Coating Operations (Auto Refinishing)</td>
<td>5</td>
</tr>
</tbody>
</table>

*Some values differ from those in the CD (Table 2-8) due to changes in the 2002 NEI subsequent to CD publication. Additionally, values just above 5 tpy have been rounded to 5.

*Includes emissions from 137 TPY emitted by 314 smaller categories (70 TPY in MACT categories and 67 TPY in non MACT).
2.3.4.1 Industrial/Commercial/Institutional/Process Heaters

Coal and/or other substances (e.g., oil, wood) are burned in boilers and process heaters to produce steam. With regard to boilers, the steam is used to produce electricity or provide heat, while process heaters are used in industrial processes. Given their use at a wide variety of facilities (e.g., refineries, chemical and manufacturing plants, etc), as well as in a “stand alone” mode to provide heat for large building complexes, there are thousands of these sources throughout the country, generally located in urban areas, and they range widely in size. Most coal-fired industrial boilers emit about 0.06 tpy while the larger ones emit about 0.07 tpy due to the use of high efficiency particulate matter (PM) control (ERG, 2002a). U.S. EPA promulgated a national emissions standard in 2004 for this category that will reduce Pb emissions (U.S. EPA, 2004).

2.3.4.2 Utility Boilers

This category includes boilers that burn coal, oil and natural gas (or, at times, other substances such as wood and petroleum coke) to produce steam to produce electricity or to provide co-generated heat for process operations. Lead is present as a natural trace metal in the fuel and is emitted to air following combustion. The extent of emissions depends on the concentration of Pb in the fuel, the quantity of fuel burned, and PM control devices applied. Most common PM control devices used in the U.S. (such as fabric filters and electrostatic precipitators) capture Pb relatively well. However, some devices work somewhat better than others. Coal-fired utilities have the highest Pb emissions among these boilers. Oil-fired plants emit somewhat lower amounts, and gas-fired plants emit very low levels of Pb (U.S. EPA, 1998). There are approximately 1,300 coal-fired electric utility boilers in the U.S. ranging in size from 25 to approximately 1,400 MWe. Based on emission factor calculations, a 325 MWe coal-fired boiler would be expected to emit approximately 0.021 tpy Pb, based on the use of an electrostatic precipitator for PM control (USEPA, 1998). Coal-fired utility boilers tend (there are exceptions) to be located in non-urban areas and have stack heights that meet standards of good construction practice.

2.3.4.3 Mobile Sources

Until 1995, when the phase-out of Pb in the nation’s motor vehicle gasoline supply was complete, leaded gasoline was the dominant source of Pb to the atmosphere. Currently, Pb is added to gasoline used in piston-engine aircraft and some types of race cars. Depending on the grade of aviation fuel, or “avgas,” the Pb content can range from 0.1 to 1.1 g of Pb per liter (Chevron, 2000). About 33 percent of general aviation aircraft use avgas, and the remainder use jet fuel, which does not contain Pb additives (CD, p. 2-51). Emissions of Pb from the use of avgas is the largest source of Pb to the air currently and is the only mobile source of Pb in the
2002 inventory (Figure 2-2). The underlying data used in calculating the emissions of Pb from avgas are being reviewed which might lead to a changes in the emissions estimate for this source. Emissions from the combustion of leaded fuel include submicron inorganic Pb halides, as well as larger, coarse fraction Pb compounds (Habibi 1973).

Vehicles used in racing are not regulated by the EPA under the Clean Air Act and can therefore use alkyl-lead additives to boost octane. EPA has formed a voluntary partnership with NASCAR with the goal of permanently removing alkyl-Pb from racing fuels used in the Busch, Nextel Cup (formerly known as Winston Cup), and Craftsman Truck Series (CD, p. 2-50). In January of 2006, NASCAR agreed to switch to unleaded fuel in its racecars and trucks beginning in 2008.

Lead is also present as a trace contaminant in gasoline and diesel fuel and is a component of lubricating oil. A range of Pb emission factors for motor vehicles is reported in the CD (pp. 2-46, 2-47, 2-49). Mobile sources of roadside Pb contamination include deposition of Pb in exhaust (largely originating from leaded gasoline), brake wear, tire wear, and loss of Pb wheel weights. Brake wear emissions are highly variable and depend on brake pad composition and driving patterns. Wheel weights can become dislodged during quick stops and although deposited pieces of wheel weights are quite large, Pb is very malleable and can be worn away into respirable particles by being run over by vehicles (CD, p. 2-50).

Lead measured in the vicinity of motor vehicle traffic is reported to have a bimodal size distribution; a submicron mode that likely originates largely as a product of combustion, and a larger mode with an approximate size range of 1.0 to 18 μm in diameter, which is likely a product of physical processes such as road dust re-suspension and tire or brake wear, with some contribution from exhaust (CD, p. 2-48).

2.3.4.4 Iron and Steel Foundries

Iron and steel foundries melt scrap, ingot, and other forms of iron and steel and pour the molten metal into molds for particular products. The largest Pb emission sources at iron foundries are large capacity furnaces, for which emissions release heights are on the order of 25-30 feet. Lead emissions from these furnaces generally range from about 0.3 to 3 tpy, depending on the throughput of the furnace, the type and operating characteristics of the emission control system, and the Pb content in the metal charged to the furnace. In the U.S., there are about 650 existing foundries, all located in 44 of the lower 48 states, and most of which are iron foundries operated by manufacturers of automobiles and large industrial equipment and their suppliers. Foundries may be located in cities or in rural areas. There is a concentration of foundries in the Midwest (IN, IL, OH, MI, WI, and MN) - roughly 40% of foundries with almost 60% of U.S. production (USEPA, 2002b). Various regulations affecting Pb emissions from this category
were promulgated in 2004. Compliance with select work practices were required by April 2005, while compliance with other emission limitations are required by April 2007. The combined impact of these actions is projected to reduce Pb emissions from this category by approximately 25 tpy (USEPA, 2004a).

2.3.4.5 Hazardous Waste Incineration/ Combustion Facilities

Hazardous waste combustors include hazardous waste incinerators, as well as boilers and industrial furnaces that burn hazardous waste for energy or material recovery (e.g., production of halogen acid from the combustion of chlorine-bearing materials). Industrial furnaces burning hazardous waste include cement kilns, lightweight aggregate kilns, and hydrochloric acid production furnaces. Lead is a trace contaminant in the hazardous waste, fossil fuels, and raw materials used in the combustors. In 2005, there were nearly 270 hazardous waste combuster sources in operation in the United States (70 FR at 59530). Approximately 40 percent of hazardous waste combustors are located in the states of Texas and Louisiana. The source categories with the largest number of combustors were boilers and incinerators with 116 and 107 sources, respectively. On October 12, 2005, EPA finalized standards implementing section 112(d) of the Clean Air Act by requiring all existing and new hazardous waste combustors to meet HAP emission standards reflecting the performance of the maximum achievable control technology (MACT) (70 FR 59402). EPA promulgated emission standards for Pb and sources must be in compliance with these standards by October 2008. Following compliance with the standards, EPA estimates that cumulative Pb emissions from hazardous waste combustors will be reduced approximately to 4.0 tons per year (USEPA, 2005). This represents a 95% reduction in Pb emissions from 1990 levels.

2.3.4.6 Primary Lead Smelting

At primary Pb smelters, Pb-bearing ore concentrates are smelted to produce Pb metal. The processes at a primary Pb smelter include: ore concentrate storage and handling; sintering of ore concentrates; sinter crushing and handling; smelting of sinter to Pb metal; drossing, refining, and alloying of Pb metal; and smelting of drosses. Lead is emitted from primary Pb smelters as process emissions, process fugitive emissions, and fugitive dust emissions (CD, p. 2-21). U.S. EPA promulgated a national emissions standard in 1999 for this category which includes an emissions limit for Pb (U.S. EPA 1999a). In the 1990s, there were three operating primary Pb smelters in the U.S: one in Montana and two in Missouri. In 2002, there were two in operation (estimated emissions shown in Table 2-3); one of the two had less than 1 tpy Pb emissions. As of 2005, there is only one operating primary Pb smelter in the U.S. which is located in Missouri. The estimate of Pb emitted from this facility in 2005 is 25 tons (CD, p. 2-20).
2.3.4.7 Secondary Lead Smelting

Secondary Pb smelters are recycling facilities that use blast, rotary, reverberatory, and/or electric furnaces to recover Pb metal from Pb-bearing scrap materials, primarily Pb-acid batteries. This category does not include remelters and refiners or primary Pb smelters. At secondary Pb smelters, Pb may be emitted from: (1) process emissions contained in the primary exhaust of smelting furnaces, (2) process fugitive emissions associated with charging and tapping of smelting furnaces and Pb refining kettles, and (3) fugitive dust emissions from wind or mechanically induced entrainment of dust from stockpile and plant yards and roadways. U.S. EPA promulgated a national emissions standard in 1997 for this category which includes an emissions limit for Pb (USEPA, 1997). In 2002, there were 15 secondary smelters operating in 11 states, most of which are in the eastern half of the U.S. The 2002 emissions estimates for the individual facilities indicate most having total emissions (fugitive and process) of less than 4 tpy, and one facility having total emissions on the order of 12 tpy (USEPA, 2006a; EC/R, 2006).

2.3.4.8 Military Installations

This source category includes sources that are military facilities. The types of sources contributing to Pb emissions from this category include, among others, rocket and engine test facilities, ammunition manufacturing, weapons testing, waste combustion and boilers. While there are over 300 military facilities in the NEI, only 10% emit over 0.1 tpy of Pb and only 3% emit over 1 ton per year. The two largest facilities (listed in Table 2-3) are a missile ammunition production plant and a weapons testing facility and these two facilities account for over 75% of the category emissions.

2.3.4.9 Mining

This category includes various mining facilities that extract ore from the earth containing Pb, zinc, copper and/or other non-ferrous metals (such as gold and silver), and/or non-metallic minerals such as talc and coal. This category does not include the smelting or refining of the metals and minerals. These facilities produce ore concentrates (such as Pb, zinc, and copper concentrates) that are transported to other facilities where further processes, such as smelting and refining take place. The 2002 NEI indicates 3 mining facilities in the U.S. emitting greater than 0.5 tpy Pb, one of which emits more than 5 tpy. This facility is in Missouri and produces Pb, zinc, and copper concentrates that are shipped to customers for further processing.

2.3.4.10 Integrated Iron & Steel Manufacturing

Integrated iron and steel manufacturing includes facilities engaged in the production of steel from iron ore. The processes involved include sinter plants, blast furnaces that produce iron, and basic oxygen process furnaces that produce steel, as well as several ancillary processes.
including hot metal transfer, desulfurization, slag skimming, and ladle metallurgy. There are currently 17 facilities. The range of Pb emissions is from 2 to 8 tpy per facility. Stack heights range from heights of 30 - 50 feet. The facilities are located in 9 states; mostly in the Midwest (USEPA, 2003a). U.S. EPA promulgated a national emissions standard in 2003 for this category which includes an emissions limit for PM (USEPA, 2003c).

2.3.4.11 Municipal WasteCombustors: Small & Large

Municipal waste combustors (MWCs) are units that incinerate municipal or municipal-type solid waste. Currently about 14% of the municipal waste generated in the US is incinerated. The amount of municipal waste incinerated has remained stable over the past decade. As described in the CD, the amount of Pb emitted from municipal waste combustors depends on the amount of Pb in the refuse, with typical sources including paper, inks, cans and other metal scrap and plastics (CD, pp. 2-35 to 2-36). The Clean Air Act of 1990 required MACT be applied to all new municipal waste incineration units and retrofitted to all existing municipal waste incineration units. The MACT retrofits at existing MWCs were completed by 2005 and national Pb emissions from municipal waste incineration are now less than 10 tons per year. This represents greater than a 97% reduction in national Pb emissions from these incinerators since 1990. There are currently 66 large MWC plants and 26 small MWC plants operating nationally. Current information indicates that individual large MWC plants general emit less than 0.1 tpy Pb, while small MWC plants generally emit less than 0.02 tpy Pb (ERG, 2002b,c; Stevenson, 2002).

2.3.4.12 Pressed and Blown Glass and Glassware Manufacturing

The pressed and blown glass and glassware manufacturing category includes manufacturers of flat glass, glass containers, and other pressed and blown glass and glassware. Lead is emitted primarily from the pressed and blown glass industry sector. Some container plants also make a leaded-glass product, but this is not typical of container glass plants. Lead may also be added to flat glass for use in microwaves and flat-screen TVs. Emissions from individual facilities may range from a few pounds per year up to several tons per year depending on Pb content of their glass and the level of control. Furnace stacks for these facilities are typically of the order of 35-60 feet high. As of 2005, about 22 tons of Pb is emitted from glass manufacturing annually. Glass plants are located in 35 States (RTI, 2006). U.S. EPA is currently developing a regulation for HAP emissions from this category, which is scheduled for promulgation in December 2007.
2.3.4.13 Electric Arc Furnace Steelmaking

In the steelmaking process that uses an electric arc furnace (EAF), the primary raw material is scrap metal, which is melted and refined using electric energy. Since scrap metal is used instead of molten iron, there are no cokemaking or ironmaking operations associated with steel production that use an EAF. There are currently 141 EAFs at 93 facilities. The total nationwide Pb and Pb compound emissions are approximately 80 tons, and the average per facility is approximately 0.75 tpy. Stack heights range from heights of 30 - 50 feet. The facilities are located in 32 states; mostly in the northeast and Midwest, with ninety percent of the facilities located in urban areas. This information is drawn from multiple sources (Lehigh, 1982; Calspan, 1977; RTI, 2005). U.S. EPA is developing a regulation for HAP emissions from this category, which is scheduled for promulgation in December 2007.

2.3.4.14 Lead Acid Battery Manufacturing

The Pb acid battery manufacturing category includes establishments primarily engaged in manufacturing storage batteries. Lead acid storage batteries are produced from Pb alloy ingots and Pb oxide. The Pb oxide may be prepared by the battery manufacturer or may be purchased from a supplier. There are currently 58 facilities operating (data obtained from the Battery Council International (BCI)); there is a general slow decline in the number of facilities. The total Pb and Pb compound emissions in the 2002 NEI from approximately 50 of the facilities included in the NEI were 25 tons. The range of facility specific Pb and Pb compound emissions is from 1 x 10^-5 to just below 10 tpy, with an average of 0.5 tpy. Facilities are located in both urban and rural areas. The facilities are located in 23 states and Puerto Rico (2002 NEI).

2.3.4.15 Primary Copper Smelting

This source category includes all industries which refine copper concentrate from mined ore to anode grade copper, using pyrometallurgical processes. Smelting includes the handling and blending of ore concentrate; the drying of copper concentrate; the smelting of concentrate to matte grade copper; the conversion of matte grade copper to blister grade copper; the refining of blister grade copper to anode grade copper; and the pouring of copper anodes. Seven primary copper smelters are currently operating in the U.S. Six of these seven smelters use conventional smelter technology which includes batch converter furnaces for the conversion of matte grade copper to blister copper, while the seventh uses a continuous flash furnace. Two of the three largest smelters are located in AZ, and the third is in Utah. The largest facility emitted an estimated 12.8 tons Pb in 2002. The estimated emissions for the other two large facilities are between 0.1 to 5 tpy. No other source in this category emits more than 0.1 tpy. In 2002, U.S. EPA promulgated a national emissions standard, including limits for PM, for this category (USEPA, 2002d).
2.3.4.16 Portland Cement Manufacturing

Portland cement manufacturing is an energy intensive process in which cement is made by grinding and heating a mixture of raw materials such as limestone, clay, sand, and iron ore in a rotary kiln, which is a large furnace that is fueled by coal, oil, gas, coke and/or various waste materials. Lead is a trace contaminant both of the raw materials and some fuel materials (e.g., coal). Thus, it is emitted in particulate materials from the kiln stacks, which range in height from near 10 meters to more than 100 meters, with relatively smaller releases from grinding, cooling, and materials handling steps in the manufacturing process. Portland cement facilities tend to be located in portions of the country with limestone deposits and in rural areas or near small towns. The largest numbers of facilities are in Pennsylvania and California, although a significant percentage of facilities are in the Midwest. Between 1990 and 2002 total industry capacity grew by 22 percent, although the number of facilities decreased slightly from 112 to 108, and as of 2004, there were 107 Portland cement plants in the U.S. (O’Hare, 2006). All but three facilities report less than 1 tpy of Pb emissions. The highest estimated Pb emissions for a facility in the 2002 NEI is 5.4 tpy. In 1999, U.S. EPA promulgated a national emissions standard, including a limit for PM, for this category (USEPA, 1999b).

2.3.5 Geographic Distribution of Sources

2.3.5.1 National Patterns in the Distribution of Pb Emissions

Figure 2-2 shows the geographic distribution and magnitude of Pb emissions in the U.S. from all sources identified in the 2002 NEI, in terms of emissions density (defined here as tons per area per county). This presentation indicates a broad distribution of the Pb emissions across the US, with perhaps a concentration of emissions in a broad swath from Indiana to southern New England, as well as in other scattered locations. Figure 2-3 shows the emission density specifically for mobile and non-point sources. Lastly, Figure 2-4 presents the geographic distribution of point sources in the 2002 NEI with Pb emissions estimates greater than 1 tpy.
Figure 2-2. Emission Density of All Pb Sources in the 2002 NEI.
Figure 2-3. Emission Density of Mobile and Non Point Sources of Pb in 2002 NEI.
2.3.5.2 Largest Pb Point Sources in the 2002 NEI

While Sections 2.3.3 and 2.3.4 focus on source categories that rank highest due to cumulative national Pb emissions, this section is intended to consider Pb emissions on the individual source level. As mentioned in Section 2.3.1 (see Table 2-1), the 2002 NEI includes 26 facilities with emissions estimated to be greater than or equal to 5 tons per year (Table 2-4). Most of these sources are metallurgical industries, followed by waste disposal facilities and manufacturing processes. The information presented in Table 2-4 is based on the current version of the NEI (USEPA, 2006a).
Table 2-4. Point Sources with Pb emissions in 2002 NEI greater than or equal to 5 tpy.

<table>
<thead>
<tr>
<th>Source Category Name</th>
<th>State</th>
<th>County Name</th>
<th>2002 Point Emissions (TPY)a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary Lead Smelting</td>
<td>MO</td>
<td>Jefferson County</td>
<td>59</td>
</tr>
<tr>
<td>Military Installation</td>
<td>OK</td>
<td>Pittsburg County</td>
<td>17</td>
</tr>
<tr>
<td>Mining</td>
<td>MO</td>
<td>Reynolds County</td>
<td>15</td>
</tr>
<tr>
<td>Secondary Nonferrous Metals</td>
<td>TX</td>
<td>Potter County</td>
<td>14</td>
</tr>
<tr>
<td>Primary Copper Smelting</td>
<td>AZ</td>
<td>Gila County</td>
<td>13</td>
</tr>
<tr>
<td>Electric Arc Furnaces</td>
<td>IL</td>
<td>Peoria County</td>
<td>13</td>
</tr>
<tr>
<td>Secondary Lead Smelting</td>
<td>MO</td>
<td>Iron County</td>
<td>12</td>
</tr>
<tr>
<td>Integrated Iron &amp; Steel Manufacturing</td>
<td>IN</td>
<td>Lake County</td>
<td>11</td>
</tr>
<tr>
<td>Pressed and Blown Glass and Glassware Manufacturing</td>
<td>TN</td>
<td>Madison County</td>
<td>11</td>
</tr>
<tr>
<td>Military Installation</td>
<td>PA</td>
<td>Franklin County</td>
<td>10</td>
</tr>
<tr>
<td>Hazardous Waste Incineration</td>
<td>AR</td>
<td>Union County</td>
<td>10b</td>
</tr>
<tr>
<td>Lead Acid Battery Manufacturing</td>
<td>KY</td>
<td>Madison County</td>
<td>10</td>
</tr>
<tr>
<td>Industrial and Commercial Machinery</td>
<td>KS</td>
<td>Marshall County</td>
<td>8</td>
</tr>
<tr>
<td>Synthetic Rubber Products</td>
<td>IN</td>
<td>Cass County</td>
<td>7</td>
</tr>
<tr>
<td>Commercial and Industrial Solid Waste Incineration</td>
<td>AR</td>
<td>Clark County</td>
<td>7</td>
</tr>
<tr>
<td>Iron and Steel Foundries</td>
<td>OH</td>
<td>Cuyahoga County</td>
<td>7</td>
</tr>
<tr>
<td>Integrated Iron &amp; Steel Manufacturing</td>
<td>IN</td>
<td>Porter County</td>
<td>7</td>
</tr>
<tr>
<td>Integrated Iron &amp; Steel Manufacturing</td>
<td>IN</td>
<td>Lake County</td>
<td>6</td>
</tr>
<tr>
<td>Mineral Products Manufacturing</td>
<td>NM</td>
<td>Socorro County</td>
<td>6</td>
</tr>
<tr>
<td>Commercial and Industrial Solid Waste Incineration</td>
<td>CT</td>
<td>Windham County</td>
<td>6</td>
</tr>
<tr>
<td>Ferroalloys Production</td>
<td>OH</td>
<td>Washington County</td>
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<tr>
<td>Nonferrous Foundries</td>
<td>NE</td>
<td>Nemaha County</td>
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<tr>
<td>Portland Cement Manufacturing</td>
<td>MD</td>
<td>Frederick County</td>
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<tr>
<td>Coke Oven</td>
<td>VA</td>
<td>Buchanan County</td>
<td>5</td>
</tr>
<tr>
<td>Iron and Steel Foundries</td>
<td>IA</td>
<td>Jefferson County</td>
<td>5</td>
</tr>
<tr>
<td>Mining</td>
<td>MO</td>
<td>Reynolds County</td>
<td>5</td>
</tr>
</tbody>
</table>

a (USEPA, 2006a)
bFollowing compliance with the MACT standards in 2008, Pb emissions are estimated to be 0.07 tpy.

2.4 AMBIENT AIR CONCENTRATIONS

The EPA has been measuring Pb in the atmosphere since the 1970s. For the most part, Pb concentrations have decreased dramatically over that period. This decrease is primarily attributed to the removal of Pb from gasoline, however, some isolated locations still Pb concentrations above the level of the NAAQS. The following sections describe the ambient Pb measurement methods, the sites and networks where these measurements are made, as well as how the ambient Pb concentrations vary geographically and temporally.
2.4.1 Ambient Pb Measurement Methods

A number of methods are used to collect Pb and measure Pb concentrations in the atmosphere, however, most methods use a similar sample collection approaches. Ambient air is drawn through an inlet for a predetermined amount of time (typically 24 hours) and the PM is collected on a suitable filter media. After the sample has been collected, the filter may be used to determine the mass of PM collected prior to then being used for determination of Pb. The filter is chemically extracted and analyzed to determine the Pb concentration in the particulate. The concentration of Pb found in the atmosphere, in μg/m³, is calculated based on the concentration of Pb in the volume extracted, the size of the collection filter, and the volume of air drawn through the filter.

The primary factors affecting the measurements made are the sampling frequency, duration of sampling, type of inlet used, and the method of analyzing the filter for Pb content. The following paragraphs describe how these factors affect the Pb measurements.

2.4.1.1 Sampling Frequency

The frequency of Pb sampling used in the U.S. varies between one sample every day (1 in 1 sampling) to the more common frequency of one sample every 6 days (1 in 6 sampling). Semi-continuous methods for the measurement of ambient metals (including Pb) are currently being explored which would allow for more frequent sampling (as frequent as 1 sample per hour), but more work is needed on these methods before they can be deployed in a network setting.

More frequent sampling reduces the uncertainty in estimates of quarterly or annual averages associated with temporal variations in ambient concentrations. However, the costs of sampling and analysis are directly tied to sample frequency. As such, it is necessary to evaluate the reduction in measurement error versus the increase in sampling and analysis costs when selecting the required sampling frequency. A discussion of the observed temporal variation of Pb measurements is given later in this section.

2.4.1.2 Inlet Design

In ambient air monitors, a number of inlet designs have been developed that allow certain particle size ranges to be sampled. The inlets use either impaction or cyclone techniques to remove particles larger than a certain size (the size cutpoint) from the sample stream. Three particle size cutpoints are used in ambient Pb measurements including total suspended PM (TSP), PM less than or equal to 2.5 μm in diameter (PM₂.₅), and PM less than or equal to 10 μm in diameter (PM₁₀). The TSP inlet is designed to allow as much suspended particulate into the sampling device as possible while protecting against precipitation and direct deposition on to the filter (nominally 25 to 45 micrometers) (USEPA, 2004c).
Sampling systems employing inlets other than the TSP inlet will not collect Pb contained in the PM larger than the size cutpoint. Therefore, they do not provide an estimate of the total Pb in the ambient air. This is particularly important near sources which may emit Pb in the larger PM size fractions (e.g., fugitive dust from materials handling and storage).

**2.4.1.3 Sample Analysis**

After the samples have been collected on filters and the filters have been weighed, the filters are analyzed for Pb content. A number of analytical methods can be used to analyze the filters for Pb content including x-ray fluorescence analysis (XRF), proton-induced x-ray emission (PIXE), neutron activation analysis (NAA), atomic absorption (AA), or inductively-coupled plasma mass spectrometry (ICP-MS) (CD, pp. 2-80 to 2-81). A detailed discussion of these methods was given in the 1986 CD, and the reader is referred to that document for more information on these analytical methods. A search conducted on the Air Quality System database shows that the method detection limits for all of these analytical methods (coupled with the sampling methods) are very low, ranging from 0.01 µg/m³ to as low as 0.00001 µg/m³, and are adequate for NAAQS compliance purposes.

**2.4.2 Pb Monitoring Programs**

Ambient air Pb concentrations are measured by four monitoring networks in the United States, all funded in whole or in part by EPA. These networks provide Pb measurements for 3 different size classes of airborne PM: TSP, PM₂.₅, and PM₁₀. The networks include the Pb NAAQS compliance network, the PM₂.₅ Speciation Trends Network (STN), the Interagency Monitoring of Protected Visual Environments (IMPROVE) network, and the National Air Toxics Trends Stations (NATTS) network. The subsections below describe each network and the Pb measurements made at these sites. Comparisons of the data from these monitoring networks will be discussed in section 2.4.4. Each network provides different types information on airborne Pb, with the NAAQS compliance network providing data (on TSP Pb) most pertinent to this review.

In addition to these four networks, various organizations have operated other sampling sites yielding data on ambient air concentrations of Pb, often for limited periods and/or for primary purposes other than quantification of Pb itself. Most of these data are accessible via the Air Quality System. In an effort to gather as much air toxics data, including Pb, into one database, the EPA and STAPPA/ALAPCO created the Air Toxics Data Archive. The Air Toxics Data Archive can be accessed at: [http://vista.cira.colostate.edu/atda/](http://vista.cira.colostate.edu/atda/).

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2 EPA’s Air Quality System can be accessed at http://www.epa.gov/ttn/airs/airsaqs/
2.4.2.1 NAAQS Compliance Network

This network is comprised of official state/local Pb monitoring stations which measure Pb in TSP, i.e., particles up to 25 to 45 microns, for the purpose of determining compliance with the Pb NAAQS. These stations use samplers and laboratory analysis methods which have either Federal Reference Method (FRM) or Federal Equivalence Method (FEM) status. The FRM and FEM method descriptions can be found in the U.S. Code of Federal Regulations, Section 40 part 50, Appendix G. Sampling is conducted for 24-hour periods, with a typical sampling schedule of 1 in 6 days. Some monitoring agencies “composite” samples by analyzing several consecutive samples together to save costs and/or increase detection limits.

The number of sites in the Pb NAAQS compliance network has decreased significantly since the 1980s (see Figure 2-5). The number of sites in the network reached its highest point in 1981 (946 sites). About 250 sampling sites operated during 2005. This decline in the number of Pb NAAQS compliance sites is attributable to the dramatic decrease in Pb concentrations observed since the 1980s and the need to fund new monitoring objectives (e.g., PM$_{2.5}$ and ozone monitoring). Lead NAAQS compliance sites in lower concentration areas were shut down to free up resources needed for monitoring of other pollutants such as PM$_{2.5}$ and ozone.

Figure 2-5. Change in the number of Pb TSP monitoring sites from 1980 to 2005.
The locations of sites in operation between 2003 and 2005 are shown in Figure 2-6. The state/local agencies which operate these sites report the data to EPA’s Air Quality System where they are accessible via several web-based tools. EPA’s series of annual air quality trends reports have used data from this network to quantify trends in ambient air Pb concentrations. The most recent Trends report for Pb can be found at [http://www.epa.gov/airtrends/lead.html](http://www.epa.gov/airtrends/lead.html).

A preliminary review of the Pb NAAQS Compliance Monitoring network's coverage of the highest Pb emitting sources (as identified in the current version of the 2002 NEI) was conducted as part of preparing this draft document. This review indicates that many of the highest Pb emitting sources in the 2002 NEI may not have nearby Pb NAAQS compliance monitors. This preliminary review suggests that only 2 of 26 facilities (both Pb smelters\(^3\)) identified as emitting greater than 5 tpy have a Pb NAAQS compliance monitor within 1 mile. We are currently completing a full review on the Pb NAAQS compliance network, including quality assurance checks on details associated with monitor locations and aspects of the NEI.

\(^3\) Primary and secondary smelters were the source types given particular priority at the time of the last Pb NAAQS review (USEPA, 1990; USEPA, 1991).
sources, to confirm locations where Pb NAAQS compliance monitors are and should be located to ensure adequate monitoring around significant Pb sources. The findings of the full review will be described in the second draft of this document.

2.4.2.2 PM$_{2.5}$ Speciation Trends Network

This is a U.S. network of about 200 PM$_{2.5}$ speciation sites. This network consists of 54 long-term trends sites [commonly referred to as the Speciation Trends Network (STN)] and about 150 supplemental sites. Most STN sites operate on a 1 in 3 day sampling schedule, while most supplemental sites operate on a 1 in 6 day sampling schedule. Nearly all of these state or locally operated sites are in urban areas, often at the location of highest known PM$_{2.5}$ concentrations. Sites in this network determine the Pb concentrations in PM$_{2.5}$ samples and, as such, do not measure Pb in the size fraction $>$2.5 µm in diameter. Lead is quantified via the XRF method. The standard operating procedure for metals by XRF is available at: http://www.epa.gov/ttnamti1/files/ambient/pm25/spec/xrfso.pdf. Data are managed through the Air Quality System. These sites generally began operation around 2000. The locations of these sites are shown in Figure 2-7.

Figure 2-7. Pb PM$_{2.5}$ (STN) monitoring sites.
2.4.2.3 IMPROVE Network – PM2.5 Speciation

In the Interagency Monitoring of Protected Visual Environments (IMPROVE) network, PM$_{2.5}$ monitors are placed in “Class I” areas (including National Parks and wilderness areas) and are mostly in rural locations. This network is administered by the National Park Service, largely with funding by EPA, on behalf of federal land management agencies and state air agencies that use the data to track trends in rural visibility. Lead in the PM$_{2.5}$ is quantified via the XRF method, as in the STN. Data are managed and made accessible mainly through the IMPROVE website (http://vista.cira.colostate.edu/IMPROVE/), but also are available via the Air Quality System. The oldest of these sites began operation in 1988, while many others began in the mid 1990s. The locations of these sites are shown in Figure 2-8. There are 110 formally designated “IMPROVE” sites located in or near national parks and other Class I visibility areas, virtually all of these being rural. Approximately 80 additional sites at various urban and rural locations, requested and funded by various parties, are also informally treated as part of the network. Samplers are operated by several different federal, state, and tribal host agencies on the same 1 in 3 day schedule as the STN.

Figure 2-8. Pb PM$_{2.5}$ (IMPROVE) monitoring sites.
2.4.2.4 National Air Toxics Trends Stations – PM$_{10}$ speciation

The National Air Toxics Trends Stations (NATTS) network of 23 sites in mostly urban, but some rural, areas. These sites are also operated by 21 state or local host agencies. All collect particulate matter as PM$_{10}$ for toxic metals analysis, typically on a 1 in 6 day sampling schedule. Lead in the collected sample is quantified via the ICP/MS method. The standard operating procedure for metals by ICP/MS is available at: http://www.epa.gov/ttn/amtic/airtox.html. These NATTS sites are relatively new, with 2004 being the first year in which all were operating. The Air Quality System can be accessed at http://www.epa.gov/ttn/airs/airsaqs/ (see Figure 2-9 for the locations of the NATTS monitoring sites).

Figure 2-9. Pb PM$_{10}$ (NATTS) monitoring sites network.

2.4.3 Ambient Pb Concentrations, Trends and Spatial Patterns

The assessment of the available air quality at the time of the last NAAQS review (in and just prior to 1990), described the dramatic changes in airborne Pb concentrations, primarily associated with the reductions in use of leaded gasoline (USEPA, 1990). Given that change in mobile source-related air concentrations, the focus of the last review was on areas near stationary sources of Pb emissions (USEPA, 1990). Since that time, as described in Section 2.4.2, there have been changes to the Pb TSP network and additional networks that produce Pb data have
been created. This section describes the available data that inform our current understanding of airborne Pb concentrations in U.S.

2.4.3.1 Pb in TSP

2.4.3.1.1 Historical Trend

Airborne concentrations of Pb in the United States have fallen dramatically over the last 30 years due largely to the phase-out of leaded gasoline additives. Figure 2-10 shows the trend in overall U.S. airborne TSP Pb concentrations for a subset of the NAAQS FRM monitoring sites from 1983 through 2002. The data are plotted in terms of average per year of the maximum arithmetic mean averaged over a calendar quarter (the form of the current NAAQS) per monitoring site and are shown in relation to the current NAAQS of 1.5 μg/m³ (maximum quarterly average). The monitors used in this analysis are typically population-oriented urban monitors that are not source-oriented. Since 1983, major declines over several orders of magnitude have been observed not only in urban areas, but also in rural regions and remote locations. The sharp decline through the 1980s has also been observed in Pb associated with fine particles (less than or equal to 2.5 microns) at remote and rural sites throughout the United States and have been attributed to the phase out of leaded gasoline (Eldred and Cahill, 1994).

![Figure 2-10. Airborne Pb (TSP) concentrations, averaged across continuously operating monitoring sites: 1980-2002.](image)
2.4.3.1.2 Current Concentrations

Lead concentrations at very few locations in the U.S. (see discussion later in this section) exceed the current maximum quarterly average NAAQS. National average concentrations of TSP Pb measured in the NAAQS compliance network (i.e., via the FRM) are well below the NAAQS level of 1.5µg/m³. The national composite average of annual means for all monitoring sites with at least one valid year of data was 0.08 µg/m³ for the 3-year period, 2003-2005; the corresponding national composite median of the monitor level annual means was 0.02 µg/m³.

The distribution of the monitor site annual means is shown in Figure 2-11. This figure also shows the national distributions of: monitor level maximum quarterly average Pb concentrations (i.e., the NAAQS metric); monitor level second maximum monthly average Pb concentration (i.e., a candidate NAAQS replacement metric discussed in the last NAAQS review); and monitor level average annual 98th percentile 24-hour concentration values (i.e., the average across the 3 years of each year’s 98th percentile value). The first three box-plots utilize the same set of TSP Pb (FRM) data for 224 monitors. The 98th percentile plot (198 total sites) excludes data reported in ‘composite’ form (26 sites).

To be included in these TSP Pb characterization analyses, a site needed at least one “complete” year consisting of at least three quarters of 6 or more observations. One hundred sixty one (161) of the 224 sites had complete data for all three years (2003-2005), 35 monitors had only two years of complete data; and 28 monitors had only one usable year of data. [Excluding the ‘composite’ data (198 total sites), 140 sites had three years of utilized data, 31 sites had two years of data, and 27 sites had only one year of data.]

For 2003-2005, the national composite average of maximum quarterly mean Pb concentrations was 0.16 µg/m³; the corresponding national composite median was 0.03 µg/m³. This median value is about fifty times lower than the 1.5 µg/m³ NAAQS level. For 2003-2005, the national composite average of second maximum monthly average Pb concentrations was 0.18 µg/m³ and the corresponding composite median was 0.03 µg/m³. The monitor average 98th percentile distribution is plotted on a different scale since those summary levels are much higher than the other three statistics. The national composite mean of average 98th percentile concentrations was 0.37 µg/m³ and the corresponding median was 0.04 µg/m³.
Figure 2-11. Distribution of TSP Pb concentrations (represented by 4 different statistics) at monitoring sites, 2003-2005.

For all four metrics plotted in Figure 2-11, the national means are substantially higher than the national medians. This is due to a small number of monitors with significantly higher levels. These monitors with higher concentrations are almost exclusively associated with industrial point sources. If source-oriented monitors were eliminated from the national level statistics shown in Figure 2-11, all of the national level statistics would be significantly lower and the means would be more comparable to the medians.

Figure 2-12 re-plots the monitor level maximum quarterly means and distinguishes between the source-oriented monitors and those not identified as such. This plot shows that 95 percent of all monitors not identified as being source-oriented had a maximum quarterly average of 0.15 µg/m³ or less (which is one tenth of the NAAQS level). Almost 25 percent of the sites

---

4 Box depicts inter-quartile range and median; whiskers depict 5th and 95th percentiles; asterisks identify composite averages.

5 Sites were classified as ‘source-oriented’ if they were within one mile of an facility emitting at least one ton of Pb per year and/or they were previously identified as such (using a 2003 reference file).
identified as being source-oriented had maximum quarterly averages of 0.75 µg/m³ or more (which is 50 percent of the current NAAQS level).

<table>
<thead>
<tr>
<th>Source-oriented sites</th>
<th># Sites</th>
<th>Maximum</th>
<th>95th Percentile</th>
<th>75th Percentile</th>
<th>Mean</th>
<th>Median</th>
<th>25th Percentile</th>
<th>5th Percentile</th>
<th>Minimum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Source-oriented sites</td>
<td>59</td>
<td>4.093</td>
<td>1.923</td>
<td>0.695</td>
<td>0.484</td>
<td>0.252</td>
<td>0.081</td>
<td>0.019</td>
<td>0.010</td>
</tr>
<tr>
<td>Not source-oriented</td>
<td>165</td>
<td>0.447</td>
<td>0.147</td>
<td>0.039</td>
<td>0.037</td>
<td>0.016</td>
<td>0.007</td>
<td>0.003</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Figure 2-12. Distribution of monitor level TSP Pb annual mean concentrations for source-oriented and not sourced-oriented monitors, 2003-2005.6

The monitor level values for two of the four discussed statistical metrics (annual average, and maximum quarterly mean) are mapped in Figures 2-13 and 2-14. As seen when comparing these figures, the locations of the high concentration levels for both metrics are generally the same. In fact, there is significant correlation among all four of the monitor level summary metrics discussed above; see Table 2-5.

6 Box depicts inter-quartile range and median; whiskers depict 5th and 95th percentiles; asterisks identify composite averages.
Figure 2-13. Site level TSP Pb, annual mean concentrations, 2003-2005.
Figure 2-14. Site level TSP Pb, maximum quarterly mean concentrations, 2003-2005.
In the past several years only about one or two FRM sites per year have had measured maximum quarterly average TSP Pb levels that exceeded the NAAQS level (1.5 µg/m³, as a maximum quarterly average). These sites are shown in Table 2-6. Two areas are officially designated as nonattainment for the Pb NAAQS: East Helena Area portion of Lewis and Clark Counties, Montana⁷; and the area within the city limits of Herculaneum in Jefferson County, Missouri (http://www.epa.gov/air/oaqps/greenbk/lnca.html).

Table 2-5. Correlation among different TSP site-level statistics, 2003-2005.

<table>
<thead>
<tr>
<th></th>
<th>Annual Mean</th>
<th>Maximum Quarterly Mean</th>
<th>2nd Max Monthly Mean</th>
<th>Annual 98th Percentile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annual Mean</td>
<td>1.00</td>
<td>0.85</td>
<td>0.86</td>
<td>0.94</td>
</tr>
<tr>
<td>Maximum Quarterly Mean</td>
<td>1.00</td>
<td>0.99</td>
<td>0.99</td>
<td>0.93</td>
</tr>
<tr>
<td>2nd Maximum Monthly Mean</td>
<td>1.00</td>
<td></td>
<td>1.00</td>
<td>0.93</td>
</tr>
<tr>
<td>Annual 98th Percentile</td>
<td></td>
<td></td>
<td></td>
<td>1.00</td>
</tr>
</tbody>
</table>

Table 2-6. FRM sites with Pb concentrations above the level of the current NAAQS, based on maximum quarterly average, 2003-2005.

<table>
<thead>
<tr>
<th>State</th>
<th>Area</th>
<th>Source type</th>
<th>Site</th>
<th>year</th>
<th>quarter</th>
<th>quarterly mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>AL</td>
<td>Pike County</td>
<td>Secondary Pb smelter</td>
<td>011090003</td>
<td>2003</td>
<td>4</td>
<td>1.92</td>
</tr>
<tr>
<td>IN</td>
<td>Delaware County</td>
<td>Secondary Pb smelter</td>
<td>180350009</td>
<td>2004</td>
<td>2</td>
<td>4.09</td>
</tr>
<tr>
<td>MO</td>
<td>Herculaneum City nonattainment area (in Jefferson County)</td>
<td>Primary Pb smelter</td>
<td>290990015</td>
<td>2005</td>
<td>2</td>
<td>1.61</td>
</tr>
</tbody>
</table>

2.4.3.1.3 Variability

Some seasonal variability is common for air Pb concentrations. However, the extent to which seasonal variability is present depends on precipitation trends, changes in wind direction, and mixing height variability for a given area. For monitors situated near Pb point sources, factors related to the facilities’ operations also contribute to temporal variability.

Figure 2-15 plots monthly TSP Pb averages for the 2003-2005 time period for four example sites. The two sites on the left, both source-oriented, have some of the highest

⁷ The source associated with this area closed in early 2001 and monitoring ceased in late 2001.
concentrations in the nation. The two sites on the right are presumably not source-oriented; their
annual average concentrations are much lower, in fact, close to the national median. Each of the
four sites has unique monthly patterns. The two sites on the top (one source-oriented and the
other not) appear to have recurring seasonal patterns. The monthly variation for the source site is
probably related to the nearby source’s operations. The variation in the other site may be due to
similar factors albeit from a smaller and/or further away emission source but there probably also
is more of a meteorological impact. The two sites plotted on the bottom have more random
variation in their monthly averages than the two on top. In general, source oriented sites (such as
the two on the left) typically have significantly more variation in their monthly averages than do
monitors that are not source oriented (such as the two on the right). Note the wide relative range
of scale for the two left plots compared to the tight range for the two right plots. This difference
in magnitude of variation is illustrated by the ratios of highest monthly average to lowest
monthly average over the 3-year period for the four sites, 43 (top left) and 26 (bottom left) for
the two source-oriented locations and 4 for the two non-source-oriented locations.
Figure 2-15. Monthly average TSP Pb concentrations at 4 example monitor sites, 2003-2005.
Monthly variation at near-source locations is better characterized by short-term averaging times (e.g., monthly) than longer-term averaging times (e.g., yearly). This is demonstrated in Table 2-7. This table shows the number of TSP monitors (in the 224 site database used above) that exceed average levels of 0.5 to 1.5 µg/m³ with averaging times or forms of 3-year, one year, quarterly, and maximum monthly, and second maximum monthly. For example, with a stated level equal to the current standard of 1.5 µg/m³, no sites in this database exceed with an averaging time of 3 years, 2 sites exceed with an averaging time of 1 year, 3 sites exceed with a quarterly averaging time, 4 sites exceed based on the 2nd maximum monthly average and 11 on the first maximum monthly average. Using the lowest level examined, 0.5 µg/m³, however, 10 sites would exceed that level with an averaging time of 3 year; 16 sites would exceed that level with an averaging time of one year; 19 sites would exceed that level with a quarterly averaging time; 21 monitors would exceed that level with their second highest monthly average, and 32 monitors would exceed that level with a maximum monthly average form.

Table 2-7. Comparison of numbers of sites that exceed various TSP Pb levels using different averaging times or forms, 2003-2005.

<table>
<thead>
<tr>
<th>Level (µg/m³)</th>
<th>3-year annual avg.</th>
<th>Max annual average</th>
<th>Max quarterly average</th>
<th>2nd max monthly average</th>
<th>Max monthly average</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>10</td>
<td>16</td>
<td>19</td>
<td>21</td>
<td>32</td>
</tr>
<tr>
<td>0.6</td>
<td>7</td>
<td>11</td>
<td>18</td>
<td>18</td>
<td>28</td>
</tr>
<tr>
<td>0.7</td>
<td>5</td>
<td>8</td>
<td>17</td>
<td>15</td>
<td>23</td>
</tr>
<tr>
<td>0.8</td>
<td>3</td>
<td>5</td>
<td>11</td>
<td>15</td>
<td>21</td>
</tr>
<tr>
<td>0.9</td>
<td>2</td>
<td>4</td>
<td>11</td>
<td>14</td>
<td>19</td>
</tr>
<tr>
<td>1.0</td>
<td>2</td>
<td>3</td>
<td>8</td>
<td>12</td>
<td>17</td>
</tr>
<tr>
<td>1.1</td>
<td>2</td>
<td>3</td>
<td>7</td>
<td>9</td>
<td>16</td>
</tr>
<tr>
<td>1.2</td>
<td>1</td>
<td>3</td>
<td>7</td>
<td>8</td>
<td>14</td>
</tr>
<tr>
<td>1.3</td>
<td>1</td>
<td>3</td>
<td>7</td>
<td>8</td>
<td>13</td>
</tr>
<tr>
<td>1.4</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>5</td>
<td>12</td>
</tr>
<tr>
<td>1.5</td>
<td>0</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>11</td>
</tr>
</tbody>
</table>

2.4.3.2 Pb in PM<sub>2.5</sub>

As noted in section 2.4.2 above, there are two national monitoring programs that collect ambient PM<sub>2.5</sub> Pb information. The EPA STN focuses mainly on urban areas and the IMPROVE network focuses mostly on rural environments, specifically those classified as “Class 1” areas (including National Parks and wilderness areas). Figure 2-16 shows the STN site level distributions of annual means, maximum quarterly means, second maximum monthly means and
annual 98th percentiles for 2003-2005. For this 3-year period, the national composite average of annual means for all sites with at least one valid year of data was 0.005 µg/m³; the corresponding national composite median of the monitor level annual means was 0.004 µg/m³. Both levels are more than an order of magnitude less than the similar TSP data illustrated in Figures 2-11.

Figure 2-16. Distribution of PM2.5 Pb concentrations (represented by four different statistics) at STN sites, 2003-2005.

Figure 2-13 maps the 2002-2005 STN site level annual averages.

---

8 To be included in PM2.5 Pb characterization analyses, a site needed at least one “complete” year consisting of at least three quarters of 11 or more observations. One hundred eighty two (182) of the 272 STN sites had complete data for all three years (2003-2005), 47 sites had only two years of complete data; and 43 sites had only one usable year of data.

9 Box depicts inter-quartile range and median; whiskers depict 5th and 95th percentiles; asterisks identify composite averages.
Figure 2-17. Site level ‘urban’ (STN) PM$_{2.5}$ Pb annual means, 2003-2005.
For PM$_{2.5}$ Pb measured in the IMPROVE program, levels are even lower. The 2003-2004 monitor level median and mean annual average levels are both less than 0.001 µg/m$^3$. Levels measured in the IMPROVE program are considerably lower than those obtained in the PM$_{2.5}$ STN network, reflecting the fact that speciation monitors are generally located in urban areas while the IMPROVE sites are in national parks and wilderness areas. Published studies have also reported that concentrations of airborne Pb are sometimes several orders of magnitude higher in urban areas compared to remote regions (Schroeder et al., 1987; Malm and Sisler, 2000). Rural areas tend to have Pb concentrations falling somewhere between those of urban and remote areas. Thus, urban populations are typically exposed to distinctly higher levels of airborne Pb than rural or remote residents.

2.4.3.3 Pb in PM$_{10}$

Figure 2-18 shows distributions of PM$_{10}$ Pb site level annual means, max quarterly means, second max monthly means, and 98th percentile concentrations for 2003-2005. For this 3-year period, the national composite average of annual means for all monitors with at least one valid year of data was 0.007 µg/m$^3$; the corresponding national composite median of the monitor level annual means was 0.006 µg/m$^3$.

---

To be included in these PM$_{10}$ Pb characterization analyses, a site needed at least one “complete” year consisting of at least three quarters of 11 or more observations. Five (5) of the 29 sites had complete data for all three years (2003-2005), 9 sites had only two years of complete data; and 15 sites had only one usable year of data.
Figure 2-18. Distribution of PM$_{10}$ Pb concentrations (represented by four different statistics), 2003-2005.\textsuperscript{11}

The PM$_{10}$ Pb site means are mapped in Figure 2-19.

\textsuperscript{11} Box depicts inter-quartile range and median; whiskers depict 5th and 95th percentiles; asterisks identify composite averages.
Figure 2-19. Monitor level PM$_{10}$ Pb annual means, 2002-2005.
2.4.4 Relationships among Different Particle-sized Pb Concentration

As described in Sections 2.4.1.2 and 2.4.2., airborne Pb concentrations are measured in three PM size fractions – TSP, PM$_{10}$ and PM$_{2.5}$ – by the various monitoring networks. Figure 2-20 summarizes the annual means for the various PM size fractions and networks. The TSP monitor averages for “all sites” and the “source-oriented” subset dwarf the other averages. An inset figure re-plots on a different scale the Pb averages for the TSP non source oriented monitors, the PM$_{10}$ NATS monitors, the PM$_{2.5}$ STN monitors, and the PM$_{2.5}$ IMPROVE monitors. The TSP non source oriented averages are about 5 times larger than the PM$_{10}$ averages; the PM$_{10}$ averages are about 1.5 times the PM$_{2.5}$ urban averages; and the PM$_{2.5}$ urban averages are about 4 times the PM$_{2.5}$ rural averages.

<table>
<thead>
<tr>
<th>Statistic</th>
<th>Obs</th>
<th>Maximum</th>
<th>95th Percentile</th>
<th>75th Percentile</th>
<th>Mean</th>
<th>Median</th>
<th>25th Percentile</th>
<th>5th Percentile</th>
<th>Minimum</th>
</tr>
</thead>
<tbody>
<tr>
<td>TSP - all sites</td>
<td>224</td>
<td>1.4502</td>
<td>0.3711</td>
<td>0.0431</td>
<td>0.0750</td>
<td>0.0148</td>
<td>0.0058</td>
<td>0.0021</td>
<td>0.0010</td>
</tr>
<tr>
<td>TSP - source-oriented</td>
<td>59</td>
<td>0.0931</td>
<td>1.9233</td>
<td>0.6963</td>
<td>0.4840</td>
<td>0.2523</td>
<td>0.0813</td>
<td>0.0193</td>
<td>0.0100</td>
</tr>
<tr>
<td>TSP not source-oriented</td>
<td>165</td>
<td>0.4467</td>
<td>0.1467</td>
<td>0.0386</td>
<td>0.0373</td>
<td>0.0157</td>
<td>0.0070</td>
<td>0.0031</td>
<td>0.0010</td>
</tr>
<tr>
<td>PM10 (NATS)</td>
<td>29</td>
<td>0.0203</td>
<td>0.0151</td>
<td>0.0088</td>
<td>0.0067</td>
<td>0.0059</td>
<td>0.0035</td>
<td>0.0022</td>
<td>0.0016</td>
</tr>
<tr>
<td>PM2.5 - STN (mostly urban)</td>
<td>272</td>
<td>0.0453</td>
<td>0.0106</td>
<td>0.0051</td>
<td>0.0049</td>
<td>0.0038</td>
<td>0.0033</td>
<td>0.0021</td>
<td>0.0010</td>
</tr>
<tr>
<td>PM2.5 - IMPROVE (Nat. park/rural)</td>
<td>167</td>
<td>0.0058</td>
<td>0.0031</td>
<td>0.0015</td>
<td>0.0012</td>
<td>0.0009</td>
<td>0.0005</td>
<td>0.0004</td>
<td>0.0003</td>
</tr>
</tbody>
</table>

Figure 2-20. Comparison of national mean and median monitor level Pb, annual means for different size cut PM networks, 2002-2005.
There are not many sites where Pb measurements are made in different PM size fractions at the same location and the same day (and where Pb values exceed the minimum detection limit). Lead is measured in all three PM size fractions in only a few locations in the United States. Table 2-8 shows Pb concentrations for 2003-2005 from four such monitoring sites: one located in Wayne Co., MI (Detroit); one located in St. Louis City, MO, and two located in Davis County, Utah (Ogden). None of these sites are known to be source-oriented. At all four sites, the majority of the TSP Pb appears to be in the PM$_{10}$ size cut. The first two sites have a slight majority of their PM$_{10}$ TSP in the fine-sized fraction, but the Ogden, UT sites have significantly more PM$_{10}$ Pb in the coarse-sized fraction.

Table 2-8. Pb concentrations (µg/m³), at four sites, in different PM size fractions: 2003-2005.

<table>
<thead>
<tr>
<th>Area</th>
<th>Site</th>
<th>Number of collocated obs</th>
<th>Average for common days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wayne, Co., MI</td>
<td>261630033</td>
<td>143</td>
<td>TSP - Pb: 0.023</td>
</tr>
<tr>
<td>St. Louis City, MO</td>
<td>295100085</td>
<td>53</td>
<td>PM$_{10}$ - Pb: 0.020</td>
</tr>
<tr>
<td>Davis County, UT</td>
<td>490110001</td>
<td>22</td>
<td>PM$_{2.5}$ - Pb: 0.008</td>
</tr>
<tr>
<td>Davis County, UT</td>
<td>490110004</td>
<td>11</td>
<td></td>
</tr>
</tbody>
</table>

Note: Although Pb was measured in TSP, PM$_{10}$, and PM$_{2.5}$ at the same site in each of the above three locations, different PM monitoring methods (collection and/or analysis) were used for the different PM size fractions, contributing to apparent anomaly of PM$_{10}$ Pb value being higher than TSP Pb value for one of the Utah sites.

In a combined analysis of data from all co-located monitoring sites, there is typically a good correlation between Pb measurements in TSP and PM$_{10}$ (average site level r of 0.96 at four sites with 10+ paired observations) and to a lesser extent between Pb measurements in PM$_{10}$ and PM$_{2.5}$ (average r = 0.62 for 18 sites with 10+ paired observations). The correlation between Pb measurements in TSP and PM$_{2.5}$ is generally quite lower (average r of = 0.38 for 35 sites with 10+ paired observations). There is substantial variability in the correlation between Pb concentrations in TSP and PM$_{2.5}$ samples at different sites. For those sites with at least 10 paired observations, the correlation coefficients range from 0.00 to >0.99.

As described in the CD, several studies have investigated Pb concentrations in different PM size fractions (CD, p. 3-13). For example, average Pb concentrations reported in a rural area in the southeastern U.S. were 6.11 ng/m³ in PM$_{2.5}$ and 15.04 ng/m³ in TSP samples, with the average total mass concentration of 9.5 µg/m³ and 19.1 µg/m³ for PM$_{2.5}$ and TSP, respectively (Goforth and Christoforou, 2006); thus, Pb constituted a similar very small proportion of particles in each size fraction. Another study included two areas in the Los Angeles basin (Singh et al, 2002). In Downey, a site where refineries and traffic contribute heavily to particle...
in concentrations, Pb was proportionally greater in the fine and ultrafine fractions of PM$_{10}$. In Riverside, which is considered a receptor site for particles transported from the Los Angeles basin and also has agricultural sources, Pb was proportionally greater in the coarse fraction of PM$_{10}$. In Boston, MA, Pb concentrations of 326 ng/m$^3$ and 75.6 ng/m$^3$ were reported from PM$_{2.5}$ and PM$_{10-2.5}$ (Thurston and Spengler, 1985). Overall, these findings indicate that for locations primarily impacted by combustion sources, Pb concentrations appear to be higher in the fine fraction of particles. However, at locations impacted by mining and material handling sources, Pb contained in the larger particles can be of significantly higher concentrations than those for the fine particles.

2.4.5 Modeling Estimates (NATA- National Scale Assessments)

As part of the Agency’s national air toxics assessment (NATA) activities, a national scale assessment of hazardous air pollutants including Pb compounds has been performed twice over the past few years (USEPA 2006c, 2002c, 2001a). These two assessments included the use of the NEI for the years 1996 and 1999, respectively, with atmospheric dispersion modeling to predict associated annual average Pb air concentrations across the country. A national scale assessment is not yet available based on the 2002 NEI. A number of limitations are associated with the 1996 and 1999 ambient concentration estimates (see Section 2.4.5.2) and the underlying emissions estimates (e.g., see Section 2.3.3). While the associated limitations handicap a reliance on the absolute magnitude of these estimates, they may prove informative with regard to relative patterns of concentrations across the country, and are presented in that light.

2.4.5.1 Methods

To develop national-scale estimates of annual average ambient Pb concentrations, EPA used the Assessment System for Population Exposure Nationwide (ASPEN) model. ASPEN uses a Gaussian model formulation and climatological data to estimate long-term average pollutant concentrations. The ASPEN model takes into account important determinants of pollutant concentrations, such as: rate of release, location of release, the height from which the pollutants are released, wind speeds and directions from the meteorological stations nearest to the release, breakdown of the pollutants in the atmosphere after being released (i.e., reactive decay), settling of pollutants out of the atmosphere (i.e., deposition), and transformation of one pollutant into another (i.e., secondary formation). ASPEN concentration estimates do not account for day-of-week or seasonal variations in emissions (USEPA, 2001a).

For each source, the model calculates ground-level concentrations as a function of radial distance and direction from the source at a set of receptors laid out in a radial grid pattern. For each grid receptor, concentrations are calculated for each of a standard set of stability class/wind speed/wind direction combinations. These concentrations are averaged together using the annual
frequency of occurrence of each combination (i.e., the climatology) as weightings to obtain annual average concentrations (USEPA, 2001a). For the 1999 NATA assessment, meteorological data for 1999 were used and the frequency distributions were also stratified by time of day into eight 3-hour time blocks. This along with similar emission rate stratification helps to preserve any characteristic diurnal patterns that might be important in subsequent estimation of population exposure. The resulting output of ASPEN is a grid of annual average concentration estimates for each source/pollutant combination by time block (USEPA, 2001a).

Annual average concentration estimates for grid receptors surrounding each emission source are spatially interpolated to the census tract centroids within the 50 kilometers impact zone, and contributions from all modeled sources are summed to give cumulative ambient concentrations in each census tract. By accounting for all identified source categories (including background concentrations, which are added to the ASPEN-calculated concentrations), the sum of the concentration increments yields an estimate of the overall Pb concentration within each census tract. For many pollutants modeled, total concentrations include a “background” component which includes concentrations due to natural sources, sources not in the emissions inventory, and long-range transport (USEPA, 2001a). In the case of Pb, however, a background concentration value of zero was used.

2.4.5.2 Findings and Limitations

Historical studies show that Gaussian dispersion models, such as ASPEN, typically agree with monitoring data within a factor of 2 most of the time. In the case of Pb in the NATA assessment, model estimates at monitor locations were generally lower than the monitor averages for Pb, suggesting that the modeling system (i.e., emissions estimates, spatial allocation estimates, dispersion modeling) may be systematically underestimating ambient concentrations. This may be particularly true for Pb as metals tend to deposit rapidly with distance from the source according to their particle size and weight. The model-to-monitor analysis is described in detail at http://www.epa.gov/ttn/atw/nata1999/99compare.html. The modeling system underestimation may also be due in part to a lack of accounting for emissions re-entrainment (these "re-entrained" particles may be observed by the monitors, but they are not accounted for in the emissions inventory, and thus would not contribute to the model estimate). For more details on the limitations of the 1999 NATA national scale assessment, see http://www.epa.gov/ttn/atw/nata1999/limitations.html.

Because higher Pb concentrations are associated with localized sources, which are not well-characterized by this modeling approach, national scale assessments such as this can only provide answers to questions about emissions, ambient air concentrations, exposures and risks across broad geographic areas (such as counties, states and the country) for that period. They are
also based on assumptions and methods that limit the range of questions that can be answered
reliably such as identifying Pb exposures and risks for specific individuals, or identifying
exposures and risks in small geographic regions such as a specific census tract.

Given the limitations of this analysis with regard to estimating Pb concentrations
nationally (see above), specific absolute ambient concentration estimates for Pb compounds
generated by this analysis are not presented here. The general pattern of results, presented
elsewhere (USEPA, 2006c), is consistent with the following conclusions: 1) there are Pb
concentrations projected in remote areas; 2) there are distinct geographical variations in ambient
Pb concentrations; concentrations in rural areas are generally much lower than in urban areas;
and, 3) there are areas with high Pb concentrations associated with localized sources with high
emissions. These results also support the general conclusion that more detailed source and site
specific analyses are needed when addressing Pb impacts.

2.4.6 Air Quality Summary

Ambient air Pb concentrations are measured by four monitoring networks in the United
States, all funded in whole or in part by EPA. These networks - the Pb NAAQS compliance
network, the PM$_{2.5}$ STN, the PM$_{2.5}$ IMPROVE network, and the PM$_{10}$ network – provide Pb
measurements for 3 different sizes of PM, and the PM$_{2.5}$ size is measured separately in urban and
remote locations.

Airborne concentrations of TSP Pb in the United States have fallen dramatically over the
last 30 years due largely to the phase out of leaded gasoline additives. Despite this decline, there
have still been a small number of areas that have not met the current Pb NAAQS over the past
few years. The sources of Pb in these areas are stationary sources (e.g. primary and/or secondary
smelters). Except for the monitors in a limited number of areas, TSP Pb averages are quite low
with respect to the NAAQS. The median monitor level maximum quarterly average for 2003-
2005 is about fifty times lower than the 1.5 µg/m$^3$ NAAQS level. However, there appears to be
significant ‘under-monitoring’ near known Pb emission point sources.

Some monthly variability is common for ambient Pb concentrations. The current form of
the standard (quarterly average) attempts to account for seasonal variability. As suggested
during the last review, a shorter averaging period (monthly) would better capture short-term
increases in Pb concentrations (USEPA 1990). Although there have only been 3 sites that
violated the 15. µg/m$^3$ max quarterly average NAAQS during the 2003 – 2005 period, 11 sites
violated that level with respect to a maximum monthly average.

There are not many sites that collect ambient Pb data in all three size ranges. Analyses of
co-located Pb size data indicate that TSP-sized Pb and PM$_{10}$-sized Pb are well correlated. If
further analyses corroborate this finding, specifically for source-oriented sites, PM$_{10}$ Pb measurements may be useful as a TSP Pb surrogate.

The NATA national scale assessment estimates based on 1999 NEI reflect the quantity and distribution of Pb emissions, with the highest estimates associated with point sources. For example, the census tract with the highest estimated Pb concentration is located in the county with the highest Pb emissions estimate in the 1999 NEI, and the second highest census tract is located in a county with a now-closed major Pb smelter. Limitations of the assessment, however, seem to contribute to uncertainty and potential underestimation of Pb concentrations.

### 2.5 ATMOSPHERIC DEPOSITION

As described in Section 2.2.2, deposition is the path by which Pb particles are removed from the atmosphere and transferred to other environmental media, and, as discussed further in Chapters 4 and 6, deposited Pb, plays a major role in human and ecological exposures. There are several approaches described in the literature for estimating atmospheric deposition, or transfer of Pb from the atmosphere to soil or water bodies. These include measurements of Pb in rainfall (wet deposition) and on collection surfaces during dry periods (dry deposition); dry deposition has also been estimated via measurements of airborne Pb particles coupled with estimates of deposition velocity (see CD, Section 2.3.2). Studies that measure Pb in sediment or soil cores, coupled with isotope dating methods (see CD, Sections 2.2.1 and 8.1.2), provide observations informative of atmospheric deposition rates and trends. As there are currently no nationwide Pb atmospheric deposition monitoring programs, the information in this section is drawn from a variety of sources as discussed in the CD.

#### 2.5.1 Temporal Trends

The available atmospheric studies of dry, wet and bulk deposition of Pb indicate a pronounced downward trend in Pb deposition in the U.S. during the 1980s to early 1990s, likely reflecting the reduction in atmospheric levels during that time period (CD, Section 2.3.2). As an example, Pirrone and others (1995) estimated an order of magnitude reduction in dry deposition from 1982 to 1991 in Detroit, Michigan (CD, Section 2.3.2). Measurements of Pb in rainfall in Lewes, Delaware (small town at mouth of Delaware Bay) have fallen from approximately 3 μg/L in the early 1980s to less than 1 μg/L by 1989 (CD, pp. 2-60 and AX7-35; Scudlark et al., 1994). Sediment core studies provide evidence of the larger historical pattern (CD, Section 2.3.1). For example, Jackson and others (2004) reported that deposition to the Okefenokee Swamp, Georgia, USA peaked during the period from 1940s through 1970s, followed by a period of steady decline into the 1990s (CD, Section 2.3.1).
2.5.2 Deposition Flux Estimates since the Last Review

Contemporary rates of total Pb loadings to terrestrial ecosystems are estimated at approximately 1 to 2 mg/m²/year (CD, p. AX7-36). In association with the Great Lakes Water Quality Agreement between the United States and Canada, a deposition monitoring network was established to estimate regional atmospheric inputs to the Great Lakes (Voldner and Eisenreich, 1989). Based on measurements from that network, total Pb deposition to the Great Lakes (Lakes Superior, Michigan and Erie) in the early 1990s was estimated to be on the order of 1.5 - 2 mg/m²-year (CD, pp. 2-57 and 2-60; Sweet et al., 1998).

For Lakes Superior and Michigan, dry deposition estimates were greater than those for wet deposition by a factor of 1.5 to 2, while dry deposition to Lake Erie was estimated to be less than 80% of wet deposition (CD, pp. 2-57 and 2-60; Sweet et al., 1998). In the mid-Atlantic region during the 1990s, dry deposition was estimated to be equal to or lower than wet deposition, contributing ≤50% of total deposition (CD, Section 2.3.2; Scudlark et al., 2005). Reports of wet deposition for this region during the 1990s range from nearly 400 to just over 600 µg/m²-year (CD, Section 2.3.2).

2.6 OUTDOOR DUST AND SOIL

Lead in outdoor dust and soil may be derived from a range of sources including current and historical air emissions sources, as well as miscellaneous non-air sources (e.g., land disposal of wastes and subsequent weathering). Both media may play a substantial role in human and ecological exposures. With regard to human exposures, contaminated soil can be a potential source of Pb exposure, particularly for children (CD, Section 3.2). Another source of children’s exposure, as discussed in the CD (Sections 3.2 and 4.4), is house dust, which may be derived from Pb in outdoor dust and soil as well as from ambient air Pb.

2.6.1 Outdoor Dust

Outdoor dust refers to particles deposited on outdoor surfaces. Lead in outdoor dust has been associated with active point sources as well as older urban areas. For example, a 50% reduction in dust Pb levels accompanied a 75% reduction in airborne Pb concentrations associated with replacement of a smelting facility in Canada (CD, pp. 3-23 to 3-24). Additionally, Caravanos and others (2006b) have described Pb in dust (particulate matter) deposited on surfaces in New York City. Lead levels have been found to be higher in dust on or near roadways, or in older urban areas as compared to newer or rural areas (CD, Sections 3.2.3 and 3.2.4; Caravanos et al 2006a,b). As with surface soil, contact with outdoor dust may contribute to incidental ingestion of environmental contaminants including Pb. Additionally, as stated in the CD (Section 2.3.3), the “re-suspension of soil-bound Pb particles and contaminated
road dust is a significant source of airborne Pb”. Re-suspension, thus, provides a pathway for Pb transport into residences and its contribution to Pb in house dust. As mentioned in Section 2.2.1, particles containing Pb may be resuspended into the air by a range of processes including wind and vehicular traffic, as well as other mechanical processes including pedestrian traffic, agricultural operations, and construction.

2.6.2 Soil

A reservoir of 0.5 to 4 g/m² gasoline additive-derived Pb is estimated to exist in U.S. soils (CD, p. AX7-36), with most contained in the upper soil horizons (O + A horizons). Studies have indicated that industrial Pb can be strongly sequestered by organic matter and by secondary minerals such as clays and oxides of Al, Fe, and Mn, (CD, pp. AX7-24 to AX7-39). Accordingly, migration (e.g. to groundwater) and biological uptake of Pb in ecosystems is considered to be relatively low, with variability of Pb mobility in different systems influenced by factors including elevation and climate, vegetation type, acidity, and soil composition (CD, Sections 2.3.5 and AX7.1.2.3). Generally then, forest floors are considered to currently act as net sinks for Pb, and burial or movement of Pb over time down into lower soil/sediment layers also tends to sequester it away from more biologically active parts of the watershed, unless later disturbed or redistributed (CD, p. AX7-36). In areas of exposed soil, however, there is potential for interaction with airborne Pb (as discussed in Sections 2.6.1 and 2.2.1).

As discussed below (Section 2.6.2.1), findings to date indicate those systems less influenced by point sources still responding to reduced Pb deposition rates associated with reduced atmospheric emissions of Pb, including those associated with the phase-out of leaded gasoline (see Section 2.3.3). Situations near point sources and those involving historically deposited Pb near roadways are less well characterized. Section 2.6.2.2 summarizes estimates of soil Pb concentrations since the time of the last review.

2.6.2.1 Temporal Trends

Variability among soil systems in characteristics influencing Pb mobility contributes to differences in current and projected temporal trends in soil concentrations (e.g., CD, pp. 3-18 to 3-19, Sections 3.2.1-3.2.2, and pp. AX7-33 to AX7-34).

Studies of forest soils have concluded that the time for soils to respond to reduced Pb deposition rates (e.g., associated with Pb gasoline phase-out) is shorter than previously believed. For example, Miller and Friedland (1994) projected that a 37% reduction in Pb concentration in northern hardwood and subalpine forest soils would occur within 17 years and 77 years, respectively. Kaste and Friedland (2003) traced atmospherically deposited Pb within forest soils in Vermont and found similar response times of 60 and 150 years for the two forest soils, respectively. They also concluded that the penetration of atmospherically delivered Pb in soils is
currently limited to the upper 20 cm and that the heterogeneous distribution of Pb in soils would seem to indicate that the release of Pb to groundwater will be dispersed thereby reducing the likelihood of a large pulse to groundwater. This study and those of Wang and Benoit (1997), Johnson et al. (1995), and Zhang (2003) conclude that forest surface soils do not act as sinks under current deposition rates for Pb and that a gradual migration into mineral soils is occurring, making the possibility of a large pulse to groundwater in the future from past Pb pollution unlikely. Studies of the role of acidification in Pb mobility in sandy soils (e.g., NJ pine barrens), however, suggest a greater risk of mobilization of Pb and organic matter into these mineral soils, with subsequent inputs to associated stream waters (CD, p. AX7-91).

Studies in urban areas of southern California, where Pb has accumulated from past sources, suggests an environment in which Pb may remain at the soil surface (and other surfaces), contributing to air concentrations via re-suspension for the near-term (CD, pp. 2-65 to 2-67 and 3-18 to 3-19). Figure 2-21 illustrates how the temporal trend in surface soil concentrations at a location is considered to be influenced by the rate of re-suspension. Harris and Davidson (2005) suggested that typical long-term values for re-suspension rate fall in the range of $10^{-11}$ to $10^{-7}$ per second, based on wind speeds, with the range of $10^{-11}$ to $10^{-10}$ proposed as a range appropriate to California’s south coast air basin. Under these assumptions, the model illustrated that the occurrence of re-suspension at this rate, would lead to little to no reduction in soil Pb concentration in southern California over the next few hundred years (CD, pp. 2-65 to 2-67 and 3-18 to 3-20).
Source: Reprinted from Harris and Davidson (2005). Units for re-suspension rate ($\Lambda$) are per second (/s).

Figure 2-21. Modeled soil concentrations of Pb in the South Coast Air Basin of California based on four re-suspension rates ($\Lambda$).

Temporal trends in surface soils near established point sources are not well characterized. Information described in the CD for areas surrounding smelters after implementation of pollution controls, although showing declines in Pb concentrations in outdoor dustfall, street dust and indoor dustfall, has not indicated a noticeable decline in soil Pb concentrations (CD, pp. 3-23 to 3-24). Further, Pb concentrations in “clean” soil placed in areas influenced by current sources have been demonstrated to exhibit increasing temporal trends (USEPA, 2006d). Concentrations of Pb in the very top layer of material (within the upper 1 inch of soil, analyzed using portable x-ray fluorescence) at locations less than a mile from a primary Pb smelter exhibited statistically significant increasing concentration over a four year period, with the average monthly change in Pb concentration ranging from 1 to 8 mg/kg (USEPA, 2001b, 2006d). Estimates of associated steady-state surface soil Pb concentrations or the expected longer-term temporal pattern for this situation have not been made.

2.6.2.2 Current Surface Soil Concentrations

Present concentrations of Pb in forest surface soils range from 40 to 100 mg/kg while natural background levels would be expected to be <1 mg/kg (CD, Section AX7.1.2.3). Urban and roadside soils and those in areas of long-term Pb emissions from point sources have much
higher concentrations of Pb, ranging up to hundreds to tens of thousands of mg/kg (CD, Section 3.2.1). For example, Pb surface soil concentrations near smelters has been found to range from 1000s of mg/kg (dry weight) within approximately 100-250 meters, dropping to 200 mg/kg and below by distances of approximately 3-5 km (CD, Table 3-4). Soil Pb concentrations of 500-800 mg/kg have been reported near U.S. mines that are no longer active (CD, Table 3-6).

2.7 SURFACE WATER AND SEDIMENT

The primary source of Pb in aquatic systems is atmospheric deposition. Lead is also carried into water bodies via wastewater effluent from municipalities and industry, stormwater runoff, erosion, and accidental discharges (CD, p. AX7-142). Most Pb occurring in aquatic systems is associated with particles, with the distribution between particle-bound and dissolved form being influenced by water chemistry as well as suspended sediment levels (CD, pp. AX7-117 to AX7-118; CD, Section AX7.2.2). The ratio of Pb in suspended solids to Pb in filtrate has been described to vary from 4:1 in rural streams to 27:1 in urban streams (CD, p. AX7-118).

Water columns have been described as “transient reservoirs” for pollutants (CD, p. 2-75). Once deposited to sediments, whether Pb is available for re-suspension back into the water column with potential transport further down a watershed versus being buried into deeper sediments depends on the aquatic system. In open ocean waters (generally characterized by depth and distance from continental sources), re-suspension to surface waters is unlikely. In more shallow systems, and additionally those influenced by land sources (e.g., stormwater runoff as well as point sources), re-suspension may play a significant role in water column concentrations. For example, studies in San Francisco Bay, the southern arm of which as an average depth of 2 m, have indicated that Pb particles may be remobilized from surface sediments into the water column (CD, AX7-141).

2.7.1 Temporal Trends

As discussed in the CD, many studies have investigated trends in Pb concentration in sediment and surface waters (CD, Section AX7.2.2), with declines documented in many systems and usually attributed to the phasing out of leaded gasoline.

Using sediment cores, temporal changes in Pb deposition and associated sediment Pb concentration have been documented. In sediment cores from the Okefenokee Swamp, Pb concentrations were approximately 0.5 mg/kg prior to industrial development, reached a maximum of approximately 31 mg/kg from about 1935 to 1965, and following passage of the Clean Air Act in 1970 concentrations have declined to about 18 mg/kg in 1990 (CD, p. AX7-141). Researchers investigating trends in metals concentrations (roughly from 1970-2001) in sediment cores from 35 reservoirs and lakes in urban and reference settings found that number of
lakes exhibiting decreasing trends in Pb concentration outnumbered increasing trends (83% versus 6%). Mass accumulation rates of Pb in cores, adjusted for background concentrations, decreased from the 1970s to the 1990s, with a median change of 246%. The largest decreases were found in lakes located in dense urban watersheds, although anthropogenic mass accumulation rates in dense urban lakes remained elevated over those in lakes in undeveloped watersheds, indicating that urban fluvial source signals can overwhelm those from regional atmospheric sources (CD, p. AX7-141; Mahler et al, 2006).

Figure 2-22 presents data on Pb concentrations in lake sediments from 12 lakes in the Great Lakes area. Consistent with other studies, this study showed a peak in Pb concentrations consistent with peak use of leaded gasoline in the U.S. in the mid 70’s and declining concentrations in most lake sediments through the mid 1990’s.

Source: Yohn et al. (2004).

Figure 2-22. Pb concentrations in sediment samples in 12 Michigan lakes. The concentrations are normalized by the peak Pb concentration in each lake; peak Pb concentrations ranged from approximately 50 to 300 mg/kg.
2.7.2 Current Concentrations

An analysis of data from the United States Geological Survey (USGS) National Water-Quality Assessment (NAWQA) program is described in the CD. The NAWQA data set encompasses data, collected over the past 15 years, on Pb concentrations in flowing surface waters, bed sediment, and animal tissue for more than 50 river basins and aquifers throughout the country (CD, Section AX7.2.2.3). Based on analysis of these data, the mean dissolved Pb concentration in ambient surface waters of the U.S. is estimated to be 0.66 μg/L (range 0.04 to 30 μg/L), as compared to a mean of 0.52 μg/L (range 0.04 to 8.4 μg/L) for the “natural” locations. The term ambient was used by NAWQA to describe the combined contribution of natural and anthropogenic sources, and a separate set of samples was identified for natural locations (e.g., “forest”, “rangeland”, and “reference” sites). The mean concentration of Pb in ambient bulk sediment (<63 microns, grain size) is 120 μg/g dry weight (range 0.5 to 12,000 μg/g), as compared to a mean of 109 μg/g dry weight (range 0.5 to 12,000 μg/g).

Geographic distribution of Pb concentrations in surface waters and sediments in this data set are presented in Figures 2-23 and 2-24 (CD, Figures AX7-2.2.7 and AX7-2.2.9). Areas with high surface water Pb concentrations were observed in Washington, Idaho, Utah, Colorado, Arkansas, and Missouri, with the maximum measured Pb concentration occurring at a site in Idaho with a land use classified as mining (CD, p. AX7-131). As was seen with surface water Pb concentrations, the highest measured sediment Pb concentrations were found in Idaho, Utah, and Colorado. And also similar to the surface water findings, of the top 10 sediment Pb concentrations recorded, 7 were measured at sites classified as mining land use (CD, p. AX7-133).
Figure 2-23. Spatial distribution of dissolved lead in surface water (N = 3445). [CD, Figure AX7-2.2.7.]
Figure 2-24. Spatial distribution of total lead in bulk sediment <63 µm (N = 1466). [CD, Figure AX7-2.2.9]

As described in the CD, dissolved surface water concentrations reported for lakes have been generally much lower than the NAWQA values for lotic waters (CD, AX7-138).
REFERENCES


O'Hare, A. 2006. Email to Michele Price from Andy O'Hare, Portland Cement Association. February 28.


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3 POLICY-RELEVANT ASSESSMENT OF HEALTH EFFECTS EVIDENCE

3.1 INTRODUCTION

This chapter assesses key policy-relevant information on the known and potential health effects associated with exposure to ambient lead (Pb). The presentation here summarizes the qualitative assessment of health evidence contained in the CD, as a basis for the evidence-based assessment of primary standards for Pb that will be presented in Chapter 5 of the second draft of this document. The focus is on health endpoints associated with the range of exposures considered to be most relevant to current exposure levels. This presentation also gives particular attention to those endpoints for which there is quantitative health evidence available in this review that provides a foundation for the quantitative health risk assessment discussed in Chapter 4 and used in the risk-based assessment of primary standards for Pb that will be presented in Chapter 5 of the second draft of this document.

The presentation in this chapter recognizes several key aspects of the health evidence for Pb. First, because exposure to atmospheric Pb particles occurs not only via direct inhalation of airborne particles, but also via ingestion of deposited particles (e.g., associated with soil and dust), the exposure being assessed is multimedia and multi-pathway in nature, occurring via both the inhalation and ingestion routes. Second, the exposure index or dose metric most commonly used and associated with health effects information is an internal biomarker (i.e., blood Pb). Additionally, the exposure duration of interest (i.e., that influencing internal dose pertinent to health effects of interest) may span months to potentially years, as does the time scale of the environmental processes influencing Pb deposition and fate. Lastly, the nature of the evidence for the health effects of greatest interest for this review is epidemiological data strongly supported by toxicological data that provide biological plausibility and insights on mechanisms of action.

At the time of the last review, Pb was recognized to produce multiple effects in a variety of tissues and organ systems across a range of exposure levels, with blood Pb levels of 10-15 μg/dL being recognized as levels of concern for impaired neurobehavioral development in infants and children (USEPA, 1990). The current CD recognizes the existence of a wide array of Pb-induced deleterious effects, including several in children and/or adults that are induced by blood Pb levels extending well below 10 μg/dL, to below 5 μg/dL and possibly lower (CD, Section 8.4).

In recognition of the multi-pathway aspects of Pb, and use of an internal exposure metric in health risk assessment, Section 3.2 describes our understanding of the internal disposition or distribution of Pb, and the use of blood Pb as an internal exposure or dose metric. Section 3.3
discusses the nature of Pb-induced health effects, giving emphasis to those with the strongest
evidence, particularly those associated with the range of current exposure levels. Potential
impacts of Pb exposures on public health, including recognition of potentially susceptible or
vulnerable subpopulations, is discussed in Section 3.4. Finally, Section 3.5 summarizes key
policy-relevant conclusions about Pb-related health effects.

3.2 INTERNAL DISPOSITION – BLOOD LEAD AS DOSE METRIC

The health effects of Pb (discussed in the CD and summarized in Section 3.3 below) are
remote from the portals of entry to the body (i.e., the respiratory system and gastrointestinal
tract). Consequently, the internal disposition and distribution of Pb is an integral aspect of the
relationship between exposure and effect. This section summarizes the current state of
knowledge of Pb disposition pertaining to both inhalation and ingestion routes of exposure (as
described in the CD).

Inhaled Pb particles deposit in the different regions of the respiratory tract as a function
of particle size (CD, pp. 4-3 to 4-4). Lead associated with smaller particles, which are
predominantly deposited in the pulmonary region, may, depending on solubility, be absorbed
into the general circulation or transported via phagocytic cells to the gastrointestinal tract (CD,
pp. 4-3). Lead associated with larger particles, that are predominantly deposited in the upper
respiratory tract (e.g., nasal pharyngeal and tracheobronchial regions), may be transported by
mucociliary transport into the esophagus and swallowed, thus making its way to the
gastrointestinal tract (CD, pp. 4-3 to 4-4), where it may be absorbed into the blood stream.

The absorption efficiency of Pb from the gastrointestinal (GI) tract varies with particle
size, as well as with the chemical form or matrix in which it is contained (CD, pp. 4-8 to 4-9).
One line of evidence for this comes from research using animal models to estimate relative
bioavailability (RBA) by comparing the absorbed fraction of ingested Pb for different test
materials relative to that for a highly water-soluble form of Pb. Relative bioavailability of Pb
from contaminated soils from different industrial sites (e.g., near Pb smelters, mines, etc), as
assessed in such models, have been found to differ markedly, with RBA values ranging from 6 to
100% (CD, pp. 4-8 to 4-10; Casteel et al., 2006). As stated in the CD, “variations in size and
mineral content of the Pb-bearing grains are the suspected cause of variations in the rate and
extent of GI absorption of Pb” occurring in soil from different contaminated locations (CD, p. 4-9).

In addition to characteristics associated with the ingested Pb, GI absorption of Pb also
varies with an individual’s physiology (e.g., maturity of the GI tract), and nutritional status (e.g.,
iron and calcium deficiency increases absorption), as well as the presence of food in the GI tract
(CD, Section 4.2.1, pp. 4-5 to 4-8). With regard to GI tract maturity, estimates of Pb GI
absorption reported in the past for young children (~40-50%) are higher than those reported for adults (CD, pp. 4-5 to 4-6). Several studies have reported that the presence of food in the GI tract reduces the absorption of water-soluble Pb (CD, p. 4-6). A contributing factor to this phenomenon is the presence of calcium, iron, and phosphate in the food, which depresses Pb absorption (CD, pp. 4-6 to 4-7). Animal studies have also indicated that Vitamin D, which regulates calcium absorption, enhances Pb absorption from the GI tract (CD, p. 4-7).

Once in the blood stream, where approximately 99% of the Pb associates with red blood cells, the Pb is distributed throughout the body, with the bone serving as a large, long-term storage compartment, and soft tissues (e.g., kidney, liver, brain, etc) serving as smaller compartments, in which Pb may be more mobile (CD, Sections 4.3.1.4 and 8.3.1.). Lead accumulates in the bone during childhood development, and this accumulation continues through adulthood. For example, more than 90% of the total Pb body burden in adults is stored in the bone, while the storage in bone accounts for approximately 70% of a child’s body burden (CD, Section 4.2.2).

As described in the CD, Pb is exchanged between blood and bone and blood and soft tissues (CD, Section 4.3.2). The exchanges between the blood and bone vary with “duration and intensity of the exposure, age and various physiological variables” (CD, p. 4-1). For example, resorption of bone (e.g., in pregnant or nursing women, or associated with osteoporosis in postmenopausal women), results in a mobilization of Pb from bone into circulation (CD, Sections 4.3.2.4 and 4.3.2.5). Past exposures that contribute Pb to the bone, consequently, may influence current levels of Pb in blood. Where past exposures were elevated in comparison to recent exposures, this influence may complicate interpretations with regard to recent exposure (CD, Sections 4.3.1.4 to 4.3.1.6). That is, higher blood Pb concentrations are not always indicative of higher body burdens or cumulative exposure, but they are generally indicative of higher exposures or Pb uptake over a somewhat recent past (CD, pp. 4-34 and 4-133). Bone measurements, as a result of the generally slower Pb turnover in bone, are recognized as providing a better measure of cumulative Pb exposure (CD, Section 8.3.2).

The bone pool of Pb is thought to be much more labile in children than in adults due to the more rapid turnover of bone mineral as a result of growth (CD, p. 4-27). As a result, “changes in blood Pb concentration in children are thought to more closely parallel changes in total body burden” (CD, p. 4-27). This is in contrast to adults, whose bone has accumulated decades of Pb exposures (with past exposures often greater than current ones), and for whom the bone may be a significant source long after exposure has ended (CD, Section 4.3.2.5).

In several recent studies investigating the relationship between Pb exposure and blood Pb in children (e.g., Lanphear and Roghmann 1997; Lanphear et al., 1998), blood Pb levels have been shown to reflect Pb exposures, with particular influence associated with exposures to Pb in
surface dust. Further, as stated in the CD “these and other studies of populations near active
sources of air emissions (e.g., smelters, etc.), substantiate the effect of airborne Pb and
resuspended soil Pb on interior dust and blood Pb” (CD, p. 8-22).

As mentioned earlier blood Pb is generally described as reflecting recent exposures (CD,
Section 4.3.1.4). Inhaled or ingested Pb quickly enters the blood, and Pb in the blood is available
for exchange with the soft and skeletal tissues, conceptually viewed as the fast (half-life of ~28
days) and slow (half-life may be decades in adults) turnover pools, respectively (CD, Section
4.3.1.4). Simulations using biokinetic models indicate that blood Pb levels in adults achieve a
new quasi-steady state within 75-100 days (approximately 3-4 times the blood elimination half-
life) subsequent to abrupt increases in Pb uptake (CD, pp. 4-25 to 4-26). Similar models indicate
a quicker response of blood Pb levels in children (CD, p. 4-27 and Figure 4-5). Additionally,
response of the blood to reduction of a relatively brief Pb exposure appears to be faster than for
an exposure of several years, with estimated half-lives of approximately 9 months as compared
to 30 months for the longer exposure response (CD, pp. 4-25 to 4-26).

Blood Pb levels are extensively used as an index or biomarker of exposure by national
and international health agencies, as well as in epidemiological (CD, Sections 4.3.1.3 and 8.3.2)
and toxicological studies of Pb health effects and dose-response relationships (CD, Chapter 5).
The prevalence of the use of blood Pb as an exposure index or biomarker is related to both the
ease of blood sample collection (CD, p. 4-19; Section 4.3.1) and by findings of association with a
variety of health effects (CD, Section 8.3.2). Accordingly, the U.S. Centers for Disease Control
and Prevention (CDC), and its predecessor agencies, have for many years used blood Pb level as
a metric for identifying children at risk of adverse health effects and for specifying particular
public health recommendations (CDC, 1991; CDC, 2005). In 1978, when the current Pb
NAAQS was established, the CDC recognized a blood Pb level of 30 μg/dL as a level warranting
individual intervention (CDC, 1991). In 1985, the CDC recognized a level of 25 μg/dL for
individual child intervention, and in 1991, they recognized a level of 15 μg/dL for individual
intervention and a level of 10 μg/dL for implementing community-wide prevention activities
(CDC, 1991; CDC, 2005). In 2005, with consideration of a review of the evidence by their
advisory committee, CDC revised their statement on Preventing Lead Poisoning in Young
Children, specifically recognizing the evidence of adverse health effects in children with blood
Pb levels below 10 μg/dL and the data demonstrating that no “safe” threshold for blood Pb had
been identified, and emphasizing the importance of preventative measures (CDC, 2005).¹

¹ With the 2005 statement, CDC identified a variety of reasons, reflecting both scientific and practical
considerations, for not lowering the 1991 level of concern, including a lack of effective clinical or public health
Since 1976, the CDC has been monitoring blood Pb levels nationally through the National Health and Nutrition Examination Survey (NHANES). This survey has documented the dramatic decline in mean blood Pb levels in the U.S. population that has occurred since the 1970s and that coincides with regulations regarding leaded fuels, leaded paint, and Pb-containing plumbing materials that have reduced Pb exposure among the general population (CD, Sections 4.3.1.3 and 8.3.3; Schwemberger et al., 2005). Although levels in the U.S. general population, including geometric mean levels in children aged 1-5, have declined, mean levels have been found to differ among children of different socioeconomic status (SES) and other demographic characteristics (CD, p. 4-21). The health effects associated with blood Pb levels are extensively discussed in the CD, while those of particular policy relevance for this review are summarized in subsequent subsections of this chapter of this document.

Blood Pb levels are used as the index of exposure (or exposure metric) for prediction of Pb associated health risk in the human exposure and health risk assessments performed for this review (described in Chapter 4). This use of exposure-response functions that rely on blood Pb (e.g., rather than ambient Pb concentration, if that were feasible) as the exposure metric in risk assessments provides a reduced uncertainty as to causality aspects of Pb risk estimates, yet imposes additional effort on identifying specific risk contributions associated with specific Pb exposure sources or pathways. For example, the blood Pb-response relationships developed in epidemiological (or toxicological) studies do not distinguish among different sources of Pb (e.g., inhalation, ingestion of dust, ingestion of dust containing paint, etc.) to the blood Pb concentration. In the exposure and risk assessments described in Chapter 4, exposure, dosimetry and empirical models are used to inform estimates of the contributions of Pb to blood Pb levels arising from ambient air related Pb versus other Pb sources.

The CD extensively discusses models in the peer reviewed literature that describe blood Pb levels associated with Pb exposure, including summaries regarding the two pharmacokinetic models that have been used in the human exposure assessment described in Chapter 4: the Integrated Exposure Uptake BioKinetic (IEUBK) model for Pb in children developed by EPA (1994a,b; White et al., 1998; CD, Section 4.4.5); and, the model developed by Leggett (1985, 1992a, 1992b, 1993) for the International Commission on Radiological Protection, which simulates Pb kinetics from birth through adulthood (CD, Section 4.4.6). The performance of these models has been evaluated with empirical data sets (CD, Sections 4.4.5.3, 4.4.6.2, 4.4.7.2).

Interventions to reliably and consistently reduce blood Pb levels that are already below 10 μg/dL, the lack of a demonstrated threshold for adverse effects, and concerns for deflecting resources from children with higher blood Pb levels (CDC, 2005).
The IEUBK model, which unlike the others has an exposure pathway interface “has gained widespread use for risk assessment purposes in the United States” in evaluating multimedia Pb exposure impacts on blood Pb levels and distribution of Pb to bone and other tissues in young children <7 years old (CD, p. 8-23 and Sections 8.3.4 and 4.4.5.3). Aspects of the Leggett model have been used in an ‘All Ages Lead Model’, currently being developed by EPA (CD, pp. 4-118 and 8-23).

3.3 NATURE OF EFFECTS

As described in the CD (Section 8.4.1), Pb has been demonstrated to exert “a broad array of deleterious effects on multiple organ systems via widely diverse mechanisms of action” (CD, p. 8-24). This array of health effects and the evidence associated with each effect is comprehensively described in the CD. This draft Staff Paper, however, is limited in focus to those effects associated with the lowest Pb levels of exposure or blood Pb (i.e., those most pertinent to ambient exposures). At the time of the last Staff Paper, the health effects of primary interest included the following (USEPA 1990):

- Heme biosynthesis and related functions;
- Neurological development and function;
- Reproduction and physical development;
- Kidney function; and
- Cardiovascular function.

As illustrated by extensive discussion in the CD, the evidence for these effects remains, and in most cases has been strengthened. Further, there has been substantial investigation of Pb immunotoxicity. There is also evidence of Pb carcinogenicity, primarily from animal studies, with limited human evidence of suggestive associations (CD, Sections 5.6.2, 6.7, and 8.4.10).2

As stated in the CD, neurotoxic effects in children and cardiovascular effects in adults are “currently clearly of greatest public health concern” (CD, p. 8-60). Further, the toxicological and epidemiological information available since the time of the last review “includes assessment of new evidence substantiating risks of deleterious effects on certain health endpoints being induced by distinctly lower than previously demonstrated Pb exposures indexed by blood-Pb levels extending well below 10 μg/dL in children and/or adults” (CD, p. 8-25). For example, the

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2 Lead has been classified as a probable human carcinogen by the International Agency for Research on Cancer, based mainly on sufficient animal evidence, and as reasonably anticipated to be a human carcinogen by the U.S. National Toxicology Program (CD, Section 6.7.2). U.S. EPA classified it in the past as a probable carcinogen (http://www.epa.gov/iris/subst/0277.htm).
overall weight of the available evidence, described in the CD, provides clear substantiation of
neurocognitive decrements being associated in young children with blood Pb levels in the range
of 5 to 10 μg/dL, and some analyses appear to show Pb effects on intellectual attainment of
young children ranging from 2 to 8 μg/dL (CD, Sections 6.2, 8.4.2 and 8.4.2.6). Table 3-1
summarizes those Pb induced health effects for children, that given their occurrence in the range
of current blood Pb levels, are most pertinent to the current review. Similar information for
adults is presented in Table 3-2 (CD, Tables 8-5 and 8-6). These tables indicate some health
effects associated with blood Pb levels that extend below 5 ug/dL, and use the notation "(???)" to
indicate that some studies have observed these effects at the lowest blood levels considered (i.e.,
threshold levels for these effects cannot be discerned from the currently available studies).
Table 3-1. Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Children (CD, Table 8-5)

<table>
<thead>
<tr>
<th>Lowest Observed Effect Blood Lead Level</th>
<th>Neurological Effects</th>
<th>Hematological Effects</th>
<th>Immune Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 µg/dL</td>
<td>Behavioral disturbances (e.g., inattention, delinquency)</td>
<td>Increased urinary δ-aminolevulinic acid</td>
<td>Effects on humoral (↑ serum IgE) and cell-mediated (↓ T-cell abundance) immunity</td>
</tr>
<tr>
<td>15 µg/dL</td>
<td>Erythrocyte protoporphyrin (EP) elevation</td>
<td>Altered electrophysiological responses</td>
<td></td>
</tr>
<tr>
<td>10 µg/dL</td>
<td>Effects on neuromotor function</td>
<td>Inhibition of δ-aminolevulinic acid dehydratase (ALAD)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>CNS cognitive effects (e.g., IQ deficits)</td>
<td>Pyrimidine-5′-nuclotidase (Py5N) activity inhibition</td>
<td></td>
</tr>
<tr>
<td>5 µg/dL</td>
<td>(???)</td>
<td>(???)</td>
<td></td>
</tr>
<tr>
<td>0 µg/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Arrows depict cases where weight of overall evidence strongly substantiates likely occurrence of type of effect in association with blood-Pb concentrations in range of 5-10 µg/dL, or possibly lower, as implied by (???). Although no evident threshold has yet been clearly established for those effects, the existence of such effects at still lower blood-Pb levels cannot be ruled out based on available data.

Source: Adapted/updated from Table 1-17 of U.S. Environmental Protection Agency (1986a).

December 2006 3-8 Draft – Do Not Quote or Cite
Table 3-2. Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Adults (CD, Table 8-6)

<table>
<thead>
<tr>
<th>Lowest Observed Effect Blood Lead Level</th>
<th>Neurological Effects</th>
<th>Hematological Effects</th>
<th>Cardiovascular Effects</th>
<th>Renal Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 µg/dL</td>
<td>Peripheral sensory nerve impairment</td>
<td>Erythrocyte protoporphyrin (EP) elevation in males</td>
<td></td>
<td>Impaired Renal Tubular Function</td>
</tr>
<tr>
<td>20 µg/dL</td>
<td>Cognitive impairment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15 µg/dL</td>
<td>Postural sway</td>
<td>Erythrocyte protoporphyrin (EP) elevation in females</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Increased urinary δ-aminolevulinic acid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 µg/dL</td>
<td></td>
<td>Inhibition of δ-aminolevulinic acid dehydratase (ALAD)</td>
<td>Elevated blood pressure</td>
<td></td>
</tr>
<tr>
<td>5 µg/dL</td>
<td></td>
<td></td>
<td></td>
<td>Elevated serum creatine</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(↓ creatine clearance)</td>
</tr>
<tr>
<td>0 µg/dL</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Arrows depict cases where weight of overall evidence strongly substantiates likely occurrence of type of effect in association with blood-Pb concentrations in range of 5-10 µg/dL, or possibly lower, as implied by (???). Although no evident threshold has yet been clearly established for those effects, the existence of such effects at still lower blood-Pb levels cannot be ruled out based on available data.

Source: Adapted/updated from Table 1-16 of U.S. Environmental Protection Agency (1986a).
The evidence for the health effects of greatest interest for this review (e.g., neurotoxic
effects in children and cardiovascular effects in adults) is a combination of epidemiological and
toxicological evidence. The epidemiological evidence is strongly supported by animal studies
that substantiate the biological plausibility of the associations, in addition to providing an
understanding of mechanisms of action for the effects (CD, Section 8.4.2).

3.3.1 Developing Nervous System

The nervous system has long been recognized as a target of Pb toxicity, with the
developing nervous system affected at lower exposures than the mature system (CD, Sections
6.2.1, 6.2.2, and 8.4). While blood Pb levels in U.S. children ages one to five years have
decreased notable since the late 1970s, newer studies have investigated and reported associations
of effects on the neurodevelopment of children with these more recent blood Pb levels (CD,
Chapter 6). Effects on the mature nervous system are discussed in a subsequent subsection of
this chapter (Section 3.3.6).

Functional manifestations of Pb neurotoxicity include sensory, motor, cognitive and
behavioral impacts. As stated in the CD, “extensive experimental laboratory animal evidence
has been generated that (a) substantiates well the plausibility of the epidemiologic findings
observed in human children and adults and (b) expands our understanding of likely mechanisms
underlying the neurotoxic effects” (CD, p. 8-25). Numerous epidemiological studies have
reported neurocognitive, neurobehavioral, sensory, and motor function effects in children at
blood Pb levels below 10 μg/dL (CD, Section 6.2). Studies with laboratory animals (discussed
in Section 5.3 of the CD) provide strong evidence with regard to the role of Pb in producing
these effects.

Effects on cognition observed in epidemiological studies have included decrements in
intelligence test results, such as the widely used intelligence quotient (IQ) score, and in academic
achievement as assessed by various standardized tests as well as by class ranking and
graduation rates (CD, Section 6.2.16). As noted in the CD with regard to the latter,
“Associations between Pb exposure and academic achievement observed in the above-noted
studies were significant even after adjusting for IQ, suggesting that Pb-sensitive
neuropsychological processing and learning factors not reflected by global intelligence indices
might contribute to reduced performance on academic tasks” (CD, pp 8-29 to 8-30).

Other cognitive effects in children have been associated with Pb exposures including
effects on attention, executive functions, language, memory, learning and visuospatial
processing. Attention and executive function effects have been associated with Pb exposures
indexed by blood Pb levels below 10 μg/dL (CD, Section 6.2.5 and pp. 8-30 to 8-31). The
evidence for the role of Pb in this suite of effects includes experimental animal findings
(discussed in CD, Section 8.4.2.1) of Pb effects on learning ability, memory and attention (CD, Section 5.3.5), as well as associated mechanistic findings. As stated in the CD, the “animal toxicology findings provide strong biological plausibility in support of the concept that Pb may impact one or more of these specific cognitive functions in humans” (CD, p. 8-31).

The evidence described in the CD provides strong support for the association of neurocognitive decrements in young children with blood Pb levels in the range of 5 to 10 μg/dL, with some analyses indicating Pb effects on intellectual attainment of young children at blood Pb levels ranging from approximately 2 to 8 μg/dL (CD, Sections 6.2, 8.4.2 and 8.4.2.6; Lanphear et al., 2005; Lanphear et al., 2000; Al-Saleh et al 2001). These studies have observed these effects at the lowest blood levels considered (i.e., threshold levels for these effects is not evident in the populations studied). Further, Pb-induced deficits observed in animal and epidemiological studies, for the most part, have been found to be persistent (CD, Sections 5.4 and 6.2.11).

Behavioral effects, including incidence of delinquent behavior, have been associated with bone Pb and with blood Pb levels above 10 μg/dL (CD, Sections 6.2.6 and 8.4.2.2). For example, the CD, based on results of several epidemiological investigations of the relationship between Pb exposure and delinquent and criminal behavior in large cities, concluded that “Pb may play a role in the epigenesis of behavioral problems in inner-city children independent of other social and biomedical cofactors,” although “the particular biological mechanisms that may underlie Pb’s effects on aggression, impulsivity, and poor self-regulation are not yet well understood” (CD, p. 8-32).

Sensory effects associated with Pb exposures during development have included those related to hearing and vision. The evidence has included findings from investigations with animal models, as well as a limited number of epidemiological studies assessing hearing thresholds and auditory processing (CD, Sections 6.2.7 and 7.4.2.3). In studies of children with median blood Pb levels of 7 or 8 μg/dL, significant associations were found for increased hearing thresholds with blood Pb levels extending below 10 μg/dL (CD, Sections 6.2.7 and 8.4.2.3, p. AX6-23).

In the few epidemiological studies that have examined neuromotor function, early Pb exposures, even those related to blood Pb levels below 10 μg/dL, have been associated with deficits in neuromotor function. Although from the animal studies “no clear pattern of Pb-induced effects on motor activity has yet emerged”, “many studies do point to an increase in activity, as seen with epidemiological findings” (CD, p. 8-36).

3.3.1.1 Endpoint for risk quantitation

Neurocognitive impact, specifically decrement in IQ in young children, is a focus of the quantitative risk assessment in this review (see Chapter 4) due to the strength of evidence for

December 2006  Draft – Do Not Quote or Cite
association with blood Pb levels below 10 μg/dL, and the strength of the dose-response
information at these exposure levels. As discussed in the CD (Section 8.4.2) and by Rice (1996),
while there is no direct animal test parallel to human IQ tests, “in animals a wide variety of tests
that assess attention, learning, and memory suggest that Pb exposure results in a global deficit in
functioning, just as it is indicated by decrements in IQ scores in children” (CD, p. 8-27). The
following statements from the CD (p. 8-44) summarize the consistency and complementary
nature of the animal and epidemiological evidence for this endpoint:

“Findings from numerous experimental studies of rats and of nonhuman primates, as
discussed in Chapter 5, parallel the observed human neurocognitive deficits and the
processes responsible for them. Learning and other higher order cognitive processes show
the greatest similarities in Pb-induced deficits between humans and experimental
animals. Deficits in cognition are due to the combined and overlapping effects of Pb-
induced perseveration, inability to inhibit responding, inability to adapt to changing
behavioral requirements, aversion to delays, and distractibility. Higher level
neurocognitive functions are affected in both animals and humans at very low exposure
levels (≤10 μg/dL), more so than simple cognitive functions.”

As stated in the CD, “epidemiologic studies of Pb and child development have
demonstrated inverse associations between blood Pb concentrations and children’s IQ and other
outcomes at successively lower Pb exposure levels” over the past 30 years (CD, p. 6-64). This is
supported by multiple studies performed over the past 15 years (see CD, Section 6.2.13), with
particularly compelling evidence for decrements in IQ at blood Pb levels below 10 μg/dL is
provided by a recent international pooled analysis of seven prospective studies (Lanphear et al.,
2005; CD, Section 6.2.13). For example, this pooled analysis estimated a decline of 6.2 points
(with a 95% confidence interval bounded by 3.8 and 8.6) in full scale IQ occurring with a change
in blood Pb level across the entire pooled data set (measured concurrent with the IQ test), from
<1 μg/dL to 10 μg/dL (CD, p. 6-76). This analysis (Lanphear et al., 2005) is relied upon in the
quantitative risk assessment for this endpoint discussed in Chapter 4.

3.3.1.2 Metric and quantitative model for risk quantitation

The epidemiological studies that have investigated blood Pb effects on IQ (see CD,
Section 6.2.3) have considered a variety of specific blood Pb metrics, including: 1) blood
concentration “concurrent” with the response assessment (e.g., with IQ testing), 2) average blood
concentration over the “lifetime” of the child at the time of response assessment (e.g., 6 or 7
years), 3) peak blood concentration during a particular age range and 4) early childhood blood
concentration (e.g., the mean of measurements between 6 and 24 months age). All four specific
blood Pb metrics have been correlated with IQ (see CD, p. 6-62; Lanphear et al., 2005). In the international pooled analysis by Lanphear and others (2005), however, the concurrent and lifetime averaged measurements were considered “stronger predictors of lead-associated intellectual deficits than was maximal measured (peak) or early childhood blood lead concentrations,” with the concurrent blood Pb level exhibiting the strongest relationship (CD, p. 6-29).

Using concurrent blood Pb level as the dose or exposure metric and IQ as the response from the pooled dataset of seven international studies, Lanphear and others (2005) employed mathematical models of various forms, including linear, cubic spline, the log-linear, and piece-wise linear, in their investigation of the blood Pb concentration-response relationship (CD, p. 6-29; Lanphear et al., 2005). They observed that the shape of the dose-response relationship is nonlinear and the log-linear model provides a better fit for the data than a linear one (CD, p. 6-29 and pp. 6-67 to 6-70; Lanphear et al., 2005). In addition, they found that no individual study among the seven drove the results (CD p. 6-30). Others have also analyzed the same dataset and similarly concluded that, within the ranges of the dataset’s blood Pb levels, a log-linear relationship was a significantly better fit than the linear relationship (p=0.009) with little evidence of residual confounding from included model variables (CD, Section 6.2.13; Rothenberg and Rothenberg, 2005).

A nonlinear exposure-response relationship is also suggested by several other studies that have indicated a dose-response relationship, in terms of estimated IQ decline per \( \mu g/dL \) increase in blood Pb, that may be steeper at blood Pb levels below 10 \( \mu g/dL \) than at higher levels (CD, pp. 8-63 to 8-64). While, as discussed in the CD, this may at first seem at odds with certain fundamental toxicological concepts, a number of examples of non- or supra-linear dose-response relationships exist in toxicology, and this non-linear dose-effect relationship also occurs for several Pb effects (CD, pp. 6-76 and 8-83 to 8-39). With regard to this particular endpoint (IQ), the CD states that it “is conceivable that the initial neurodevelopmental lesions at lower Pb levels may be disrupting very different biological mechanisms (e.g., early developmental processes in the central nervous system) than the more severe effects of high exposures that result in symptomatic Pb poisoning and frank mental retardation” (CD, p. 6-76). In comparing across the individual studies and the pooled analysis, it is observed that at higher blood Pb levels, the slopes derived for log-linear and linear models are almost identical, and for studies with lower blood Pb levels, the slopes appear to be steeper than those observed at higher blood Pb levels (CD, p. 8-78, Figure 8-7).

Given the evidence summarized here and described in detail in the CD (Chapters 6 and 8), and consistent with recommendations from CASAC on the risk assessment plan (Henderson, 2006), the assessment of children’s risk described in Chapter 4 relies on the log-linear functions.
presented by Lanphear and others (2005) that relate absolute IQ as a function of the log of concurrent blood Pb, and lifetime average blood Pb, respectively. As discussed above, the slope of the exposure-response relationship described by these functions is greater at the lower blood Pb levels (e.g., less than 10 μg/dL). The impact of the nonlinear slope is illustrated by the estimated IQ decrements associated with increases in blood IQ for different ranges of blood Pb level. The IQ changes were 3.9 (with 95% confidence interval, CI, of 2.4-5.3), 1.9 (95% CI, 1.2-2.6) and 1.1 (95% CI, 0.7-1.5), for increases in concurrent blood Pb from 2.4 to 10 μg/dL, 10 to 20 μg/dL, and 20 to 30 μg/dL, respectively (Lanphear et al., 2005).

As discussed in the CD, threshold blood Pb levels for these effects cannot be discerned from the currently available epidemiological studies, and the evidence in the animal Pb neurotoxicity literature does not identify a well-defined thresholds for any of the toxic mechanisms of Pb (CD, Sections 5.3.7 and 6.2). However, in recognition of a reduced confidence in the characterization of the quantitative blood Pb concentration-response relationship at the lowest blood Pb levels included in the current studies, as well as the possibility of a threshold at or below these levels, the staff has employed a hypothetical threshold or cutpoint in the pilot quantitative risk assessment described in chapter 4, below which it is assumed that there is no individual response. In this context, this cutpoint is not intended as a true biological threshold. Rather it is intended simply to reflect a potential or hypothetical inflection point at the lower end of the concentration-response relationship.

In selecting the cutpoint for pilot risk assessment, we considered particularly two studies (Lanphear et al., 2000 and Lanphear et al., 2005). In the study by Lanphear and others (2000) that found associations of cognitive deficits in children aged 6-16 years of age using NHANES III, the authors stratified their analyses into four blood Pb categories: <10 ug/dL (n=4,681); <7.5 ug/dL (n=4526); <5.0 ug/dL (n=4,043) and <2.5 ug/dL (n=2,467). The lowest blood Pb group was substantially smaller in size than the other groups, and additionally, although coefficients for that category are fairly similar to the ones in higher level categories (sometimes slightly larger), none of the coefficients were statistically significant, indicating a reduction in statistical power. Additionally, in the pooled analysis by Lanphear and others (2005), from which the exposure-response functions for the health risk assessment is drawn, the proportion of the pooled data set below 2.5 μg/dL (concurrent blood Pb) is quite small. For example, the level of 2.4 μg/dL, is the concurrent blood Pb level for the 5th percentile of the pooled data set, while 33.1 μg/dL is the 95th percentile. The 5th and 95th percentile values for lifetime average blood Pb are 6.1 and 47 μg/dL, respectively (Lanphear et al., 2005).
3.3.2 Cardiovascular System

Epidemiologic and experimental toxicology studies support the relationship between Pb exposure and increased adverse cardiovascular outcome, including increased blood pressure, increased incidence of hypertension, and cardiovascular morbidity and mortality (CD, Sections 5.5, 6.5 and 8.4.3).

The cardiovascular effect most frequently examined in epidemiological studies is increased blood pressure in adults, which has been repeatedly associated with Pb exposure (CD, Sections 8.4.3 and 6.5.7). The association has been observed with Pb levels in both bone and blood (including blood Pb levels below 10 μg/dL). This epidemiological evidence is supported by evidence in numerous animal studies of arterial hypertension (HTN) with low Pb exposures, an effect that persists in animals long after cessation of exposure (CD, Sections 5.5 and 8.4.3). A recent meta-analysis by Nawrot and others (2005), that included a range of blood Pb levels from 2.3 to 63.8 μg/dL, reported an association of increased systolic blood pressure and decreased diastolic pressure with increased blood Pb level, including levels below 10 μg/dL. The changes observed, on the order of 1 millimeter mercury increase in systolic pressure per doubling of blood Pb, have considerable significance at the population level (CD, p. 8-45, Section 8.6.3). Systolic blood pressure exerts a strong influence on more serious cardiovascular events by its role in hypertension and its adverse cardiovascular sequelae (CD, p. 8-83).

Multiple studies of blood pressure and hypertension have reported positive associations with bone Pb levels, highlighting the important role for cumulative past Pb exposure in development of cardiovascular health effects (Sections 6.5.2.3 and 6.5.7). Further, a study of young adults who lived as children in an area of high Pb exposures indicates the potential for childhood exposure to contribute to such effects later in life. In this study, higher bone Pb levels were associated with higher systolic and diastolic blood pressure (CD, p. 6-138), while current blood Pb levels (mean of 2.2 μg/dL) were not associated with blood pressure effects (CD, p. 6-124).

Several analyses of National Health and Nutrition Examination Survey (NHANES) cohorts, including some recently released, have collectively suggested a “significant effect of Pb on cardiovascular mortality in the general U.S. population” (CD, p. 8-88, Sections 6.5.3.2 and 8.6.3). For example recent analyses of NHANES blood Pb data from 1976 to 1980 and 1988 to 1994 provide supportive evidence for an increased risk of cardiovascular mortality, consistent with projected likely increases in serious cardiovascular events (stroke, heart attack) resulting from Pb-induced increases in blood pressure (CD, Section 8.6.3).
3.3.3 Heme Synthesis

It has long been recognized that Pb exposure is associated with disruption of heme synthesis in both children and adults. At blood Pb levels above 30 μg/dL, such disruption leads to notable reductions in hemoglobin synthesis, and, at blood Pb levels above 40 μg/dL, to frank anemia, a clinical sign of severe Pb poisoning (CD, p. 8-47). The evidence regarding effects on heme synthesis and other hematological parameters in animal and humans is strong, and includes documented quantitative relationships between exposure and effects in children and adults. Interference with heme synthesis was identified as one of the targets of low-level Pb toxicity in children during the time of the last NAAQS review (USEPA, 1990), and was the primary basis for the initial setting of the Pb NAAQS in 1978 (USEPA, 1978).

Mechanisms associated with Pb interference with heme synthesis include inhibition of the enzymes δ-aminolevulinic acid dehydratase (ALAD) and ferrochelatase (CD Sections 5.2.1, 6.9.1, 6.9.2). Inhibition of ALAD has been associated with increased blood Pb concentrations across the range of 5 to 150 μg/dL. This information and evidence regarding associated mechanisms is presented and discussed in detail in the CD (Sections 8.4.4, 5.2.1, 6.9.1 and 6.9.2).

3.3.4 Renal System

As described in the CD (Sections 5.7.3 and 8.4.5), Pb nephrotoxicity is mediated by alterations in the glomerular filtration rate. The animal literature has described the occurrences and mechanisms of Pb uptake by and accumulation in the kidney, and associated cellular alterations (CD, Section 5.7). A set of screening tests involving markers of nephrotoxic effects have been established for screening individuals exposed to Pb occupationally or environmentally (CD, Section 5.7.1). In the epidemiological literature, associations between blood Pb and indicators of renal function impairment (e.g., measures of glomerular integrity, such as creatinine levels in urine) have been found at blood Pb levels extending below 10 μg/dL, to as low as ~2 to 4 μg/dL (CD, Sections 6.4.4.1.5 and 8.4.5). Associations are also observed with cumulative Pb dose, assessed via bone Pb, and longitudinal renal function decline (CD, p. 6-94).

Although previous observations from occupational studies have indicated much higher Pb blood levels (e.g., >30-40 μg/dL) as affecting renal tubular function, the CD describes the recent findings in non-occupational populations as providing “strong evidence that renal effects occur at much lower blood Pb levels than previously recognized” (CD, p. 6-113). Exposure history may play a role in the differences in the two sets of evidence. For example, the CD recognizes that “the data available to date are not sufficient to determine whether nephrotoxicity is related more to such current blood-Pb levels, higher levels from past exposure, or both” (CD, p. 8-49). Additionally, the CD suggests that the studies in the general population likely had larger...
proportions of susceptible individuals than occupational cohorts, which may play a role in the
findings of lower Pb dose thresholds for Pb renal effects in environmental compared to
occupational research (CD, p. 6-107).

The findings regarding Pb exposures and renal effects are of particular concern with
regard to certain susceptible subpopulations as described in the CD (p. 6-113).

“At levels of exposure in the general U.S. population overall, Pb combined with other
risk factors, such as diabetes, hypertension, or chronic renal insufficiency from non-Pb
related causes, can result in clinically relevant effects. Notably, the size of such
susceptible populations is increasing in the United States due to obesity.”

That is, Pb is recognized as acting cumulatively with other renal risk factors to cause early onset
of renal insufficiency and/or a steeper rate of renal function decline in individuals already at risk
for renal disease (CD, p. 6-107).

### 3.3.5 Immune System

Since the time of the last review, there has been substantial research on the
immunotoxicity of Pb. As summarized in the CD, “studies across humans and a variety of
animal models are in general agreement concerning both the nature of the immunotoxicity
induced by Pb as well as the exposure conditions that are required to produce
immunomodulation” (CD, p. 5-244, Section 5.9). Lead is distinguished from other
immunotoxicants, however, by the fact that the most sensitive biomarkers of its immunotoxicity
are associated with specific functional capacities that influence risk of disease, as opposed to
being associated with changes in immune cell numbers or pathological changes of lymphatic
system organs (CD, Section 5.9.1). The main immune system targets of Pb are macrophages
and T lymphocytes, leading to a potential for increased tissue inflammation, reduced cell-
mediated immunity, and increased risk of autoimmunity (See CD, Figure 5-18, Section 5.9.11).
Additionally, Pb exposures in both animal and human studies are associated with increased
production of IgE, an immunoglobulin involved in allergic responses and asthma (CD, Section
5.9.3.2). These effects have been reported in epidemiologic studies of children, and supported
by evidence in neonatal/juvenile animals, at blood-Pb levels extending below 10 μg/dL (CD, p.
6-197 and Sections 5.9.10 and 8.4.6).

### 3.3.6 Adult Nervous System

As discussed in Section 3.3.1, the nervous system has long been recognized as a target of
Pb toxicity (CD Sections 5.3.1, 8.4.2). As described in the CD, a blood Pb concentration of ≥14
μg/dL in those chronically exposed in the workplace is a possible threshold for various
neurological effects including peripheral sensory nerve impairment, visuomotor and memory
impairment, and postural sway abnormalities (CD, p. 6-87). Past occupational exposure also

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increases the risk of developing amyotrophic lateral sclerosis (ALS) and motor neuron disease (CD, Section 6.3.5 and p. 6-87). Essential tremor is also associated with Pb exposures, with studies indicating that the subpopulation of individuals with the ALAD2 allele have a 30-fold greater increased risk than those with only ALAD1 gene (CD, Sections 6.3.5 and 6.3.6 and p. 6-86).

Epidemiological studies of elderly populations have also investigated associations between Pb exposures and impaired cognitive performance (CD, Section 6.3.3). While significant associations have not been consistently found in studies employing blood Pb as the exposure metric, significant associations have been reported with bone Pb levels as the exposure metric (CD, Section 6.3.3.1), perhaps indicating a role of cumulative and/or past Pb exposures (CD, p. 6-83). The studies involving bone Pb have utilized several large cohorts of older adults and reported associations with cognitive dysfunction in aging populations (CD, Section 6.3.3.1). These include the Normative Aging Study (Rhodes et al., 2003; Payton et al., 1998; Wright et al., 2003; Weisskopf et al., 2004), and the Study of Osteoporotic Fractures (Muldoon et al., 1996). The general finding of these studies of significant associations with bone Pb, but not blood Pb, suggests that long-term cumulative exposure, more than current exposure, may contribute to these neurotoxic effects in adults (CD, p. 6-83).

As discussed in the CD (Section 5.3.7), there is animal evidence supporting an increased vulnerability among the elderly to Pb effects on cognitive function. During the demineralization of bone that occurs during aging, Pb may be released into the blood, thus augmenting blood Pb associated with current ambient exposures (CD, Section 4.3.2.4). Research involving lifetime exposure has found that senescent animals exhibit an increased vulnerability to Pb due to this increased exposure from bone resorption and an apparently greater sensitivity to the biochemical effects of Pb (CD, Section 5.3.7). Additionally, animal studies indicate that cognitive function effects in the elderly may also be related to physiological effects of Pb exposures in early childhood (CD, p. 5-67). Laboratory animal research in rats and monkeys has demonstrated an effect of early life exposure to Pb on latent upregulation of the gene associated with the production of beta-amyloid precursor protein (APP). Increased expression of APP, which is thought to have a role in Alzheimer’s disease, was also observed. Upregulation of APP mRNA and of APP was not demonstrated with Pb exposures in later life (CD, p. 5-67; Basha et al 2005). Thus, early life exposure to Pb may contribute to neurocognitive effects later in life due to the redistribution of Pb body burden from bone to brain and by enhanced vulnerability caused by age-related degenerative changes in various organs, including brain (CD, p. 8-40).
3.4 LEAD-RELATED IMPACTS ON PUBLIC HEALTH

In addition to the advances in our knowledge and understanding of Pb health effects at lower exposures (e.g., using blood Pb as the index), there has been some change with regard to the U.S. population Pb burden since the time of the last Pb NAAQS review. For example, the geometric mean blood Pb level for U.S. children aged 1-5, as estimated by the U.S. Centers for Disease Control, declined from 2.7 μg/dL (95% CI: 2.5-3.0) in the 1991-1994 survey period to 1.7 μg/dL (95% CI: 1.55-1.87) in the 2001-2002 survey period (CD, Section 4.3.1.3).3 Blood Pb levels have also declined in the U.S. adult population over this time period (CD, Section 4.3.1.3). These observation however, should not be interpreted to mean that blood Pb levels declined in all communities, and by this amount. As noted in the CD, “blood-Pb levels have been declining at differential rates for various general subpopulations, as a function of income, race, and certain other demographic indicators such as age of housing” (CD, p. 8-21).

The following discussion draws from the CD to characterize subpopulations potentially at risk for Pb-related effects and potential public health impacts associated with exposure to ambient Pb.

3.4.1 Potentially Susceptible or Vulnerable Subpopulations

The CD summarizes information on factors affecting susceptibility to Pb toxicity, and also recognizes associated factors of susceptibility within the individual discussions of specific Pb effects. Such factors include both physiological conditions contributing to a subgroup’s increased risk of effects at a given blood Pb level, and physiological conditions that contribute to blood Pb levels higher than those otherwise associated with a given Pb exposure (CD, Section 8.5.3). The term vulnerability additionally encompasses situations of elevated exposure (e.g., residing in old housing with Pb-containing paint or near sources of ambient Pb), as well as socioeconomic factors (e.g., reduced access to health care or low SES) (USEPA, 2003) that can contribute to increased risk of adverse health effects from Pb.

Three particular physiological factors contributing to increased risk of Pb effects at a given blood Pb level are recognized in the CD (e.g., CD, Section 8.5.3). The first factor is age. As summarized in Section 3.5.1 of this document, and described in detail in the CD (e.g., Sections 6.2, 8.4, 8.5, 8.6.2), the susceptibility of young children to the neurodevelopmental effects of Pb is well recognized. A difficulty in identifying a discrete period of susceptibility has been that the period of peak exposure is around 18-27 months when hand-to-mouth activity is at

3 These levels are in contrast to the geometric mean blood Pb level of 14.9 μg/dL reported for U.S. children (aged 6 months to 5 years) in 1976-1980 (CD, Section 4.3.1.3).
its maximal (CD, p. 6-60). Earlier Pb literature described the first 3 years of life as a critical
window of vulnerability to the neurodevelopmental impacts of Pb (CD, p. 6-60), however, recent
epidemiologic studies have indicated a potential for susceptibility of children to concurrent Pb
exposure to extend to the reaching of school age (CD, pp. 6-60 to 6-64). It may be that the
influence of concurrent blood Pb (and exposures contributing to it) remains important until
school age with regard to the potential to affect cognitive development (CD, pp. 6-63 to 6-64;
Chen et al., 2005). Additionally, the existing evidence regarding Pb immunotoxicity, and in
particular, impacts on the immunoglobulin, IgE, also indicates an increased susceptibility of
children (CD, Sections 5.9.10, 6.8.3 and 8.4.6). Early childhood Pb exposures have also been
associated with increased risk of cardiovascular and neurodegenerative effects in adulthood (CD,
p. 8-74).

A second physiological factor contributing to increased risk of Pb associated effects is
health status. For example, subpopulations with pre-existing health conditions may also be at
increased susceptibility (as compared to the general population) for particular Pb-associated
effects, which is most clear for renal and cardiovascular outcomes. Those with higher baseline
blood pressure or hypertension may face a greater risk of adverse health impact from Pb-
associated cardiovascular effects, e.g., African Americans, as a group, have higher frequency of
hypertension than the general population or other ethnic groups (NCHS, 2005). As discussed in
the CD (Sections 6.4.7 and 8.4.5), those with diabetes, hypertension, and chronic renal
insufficiency have been shown to be at increased risk of Pb-associated reductions in renal
function. Such reductions have been reported at blood Pb levels ranging down to just below 5
μg/dL (CD, Section 6.4.4.1 and p. 8-72). Additionally, older age may be a risk factor for effects
on renal function (CD, p. 6-107). Consequently, particularly vulnerable subpopulations may be
those that are Pb exposed and also at increased risk for obesity, diabetes, and hypertension; as
stated in the CD, frequently exposures to Pb occur in the same lower SES groups as these other
risk factors (CD, p. 8-89).

A third physiological factor relates to genetic polymorphisms. Subpopulations defined
by particular genetic polymorphisms have also been recognized with regard to susceptibility to
Pb toxicity. For example, presence of the ALAD allele appears to increase the magnitude of Pb-
associated renal dysfunction (CD, p. 8-71, Section 6.4.7.3), and also may play a role in the risks
of developing ALS or motor neuron disease that have been associated with past occupational
exposures to Pb (CD, p. 8-71, Sections 6.3.5 and 6.3.6).

Several physiological factors pertain to susceptibility or sensitivity by contributing to
increased blood Pb levels over those otherwise associated with a given Pb exposure (CD, Section
8.5.3). These include nutritional status, which as recognized in Section 3.2, plays a role in Pb
absorption from the GI tract; polymorphisms such as those for the vitamin D receptor, which
studies suggest may contribute to increased Pb absorption from the GI tract; and bone
demineralization, such as that occurring during pregnancy, lactation, and aging, which appear to
influence the release of Pb from bone storage into the blood (CD, Sections 5.10.2.5, 4.3.2 and
8.5.3). An increased prevalence of certain polymorphisms contributing to increased blood Pb
levels may occur in particular subpopulations, increasing the sensitivity or vulnerability of that
group to Pb associated effects. One example of this occurs with the vitamin D receptor or VDR
gene, which is involved in calcium absorption through the gut. A study on blood Pb levels
related to distribution of the FF genotype for this gene indicated that children with this genotype
had the highest adjusted mean blood Pb concentrations at 2 years of age compared to children
with alternate genotypes for this gene (CD, p. 6-56; p. 8-41). As described by the CD, the “high
prevalence of FF genotypes in African-American children, compared to non-African American
children, may partially explain higher blood Pb concentrations often observed in African-
American children” (CD, p. 8-41). Additionally, the last two NHANES surveys support findings
of significantly higher blood Pb levels in African-American children than whites, even after an
adjustment for urban residential status and family income, indicating that African-American
children are at increased risk for elevated blood Pb levels compared to white children (CD, p. 6-
54).

Differences in blood Pb levels among subpopulations living in the same area have also
been identified that indicate an increased vulnerability to Pb exposure among some subgroups,
perhaps related to SES. For example, a study of populations residing in a mining area found
highest blood Pb levels among African-American, Mexican-American, and poor children (CD,
pp. 3-26 and 8-13).

3.4.2 Potential Public Health Impact

There are several potential public health impacts associated with the current range of
population blood Pb levels, including potential impacts on population IQ, heart disease, and
chronic kidney disease (CD, Section 8.6). The quantitative implications of potential Pb-related
population impacts related to these health impacts are discussed in the CD (Sections 8.6.2, 8.6.3
and 8.6.4). With regard to IQ, it is noted that, given a somewhat uniform manifestation of Pb-
related decrements across the range of IQ scores in a population, “a downward shift in the mean
IQ value is not associated only with a substantial increase in the percentage of individuals
achieving very low scores, but also with substantial decreases in percentages achieving very high
scores” (CD, p. 8-81). For example, for a population mean IQ of 100 (and standard deviation of
15), 2.3% of the population would score above 130, but a shift of the population to a mean of 95
results in only 0.99% of the population scoring above 130 (CD, pp. 8-81 to 8-82).
In emphasizing the need to recognize distinctions between population and individual risk, the CD notes that a “point estimate indicating a modest mean change on a health index at the individual level can have substantial implications at the population level” (CD, p. 8-77). For example, “the import of a decline for an individual’s well-being is likely to vary depending on the portion of the IQ distribution” such that “for an individual functioning in the low range due to the influence of developmental risk factors other than Pb”, a Pb-associated IQ decline of several points, might be sufficient to drop that individual into the range associated with increased risk of educational, vocational, and social handicap (CD, p. 8-77). Similarly, “although an increase of a few mmHg in blood pressure might not be of concern for an individual’s well-being, the same increase in the population mean might be associated with substantial increases in the percentages of individuals with values that are sufficiently extreme that they exceed the criteria used to diagnose hypertension” (CD, p. 8-77).

The magnitude of a public health impact is dependent upon the size of population affected and type or severity of the effect. As summarized in Section 3.4.1, there are several population groups that may be susceptible or vulnerable to effects associated with exposure to Pb. They include, young children, particularly those in families of low SES, as well as individuals with hypertension, diabetes, and chronic renal insufficiency. Although comprehensive estimates of the size of these groups residing in proximity to policy relevant sources of ambient Pb have not been developed, total estimates of these population subgroups within the U.S. are substantial (Table 3-3).

Table 3-3. Population subgroups with characteristics that may contribute to increased susceptibility or vulnerability to Pb health effects.

<table>
<thead>
<tr>
<th>Childrena Living in poverty</th>
<th>Adultsb w. hypertensionc</th>
<th>Adultsb w. Diabetes</th>
<th>Adultsb w. chronic kidney disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estimated # in U.S. populationd</td>
<td>4.8 million (20%)e</td>
<td>~50 million (25.6%)e</td>
<td>18 million (8.7%)e</td>
</tr>
<tr>
<td>Reference</td>
<td>DeNavas-Walt et al., 2006 NCHS, 2005 CDC, 2003 Coresh et al., 2005</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

aChildren less than 6 years of age.
bIndividuals greater than 20 year of age.
cHypertension, defined as blood pressure of 140/90 millimeters of mercury (mm Hg) or higher, using blood-pressure lowering medications, or having been told at least twice by a physician or other health professional that they had high blood pressure (medical history).
dNote that there may be overlap among some groups (i.e., individuals may be counted in more than one subgroup).
ePercent of age group.
As described in the CD, and investigated in the pilot exposure and health risk assessment presented in Chapter 4 of this document, subpopulations residing near some sources of Pb emissions may be at increased risk of Pb exposures and associated effects. The limited information available on air and soil concentrations of Pb indicates elevated concentrations near some stationary sources (as compared with remote from such sources), including primary and secondary Pb smelters (see Chapters 2 and 4). Using information from the 2000 U.S. Census and locations of currently operating primary and secondary Pb smelters, it is estimated that some 76,000 persons, including some 8600 children less than 7 years of age, reside within 2 kilometers of these sources. Emissions estimates described in Section 2.3 for individual sources (e.g., Table 2-4) suggest a variety of other source types that may emit Pb in the range of secondary Pb smelters. Population size estimates near other Pb stationary point sources, however, have not been developed for this first draft Staff Paper. Additionally, the potential for historically deposited Pb near roadways to contribute to increased risks of Pb exposure and associated risk to populations residing nearby is suggested in the CD and also investigated in Chapter 4 of this document. Although estimates of the number of individuals, including children, living within close proximity to roadways specifically recognized for this potential have not been developed, these numbers may be substantial.

### 3.5 SUMMARY AND CONCLUSIONS

Based on the available health effects evidence and the evaluation and interpretation of that evidence in the CD, summarized briefly above, the following conclusions have been drawn:

- Lead exposures occur both by inhalation and by ingestion. Ingestion of Pb-contaminated dust has a strong influence on blood Pb levels in children.

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4 Total population counts are based on 2000 U.S. Census, derived for census blocks falling within 2 km of facility. In lieu of block-level age-specific counts, subgroup counts were derived from total population counts based on block counts and subgroup representation in block groups within 2 km of a facility, and rounded to nearest 100.

5 The distance of 2 kilometers is consistent with estimates of distances associated with significant deposition or soil deposition for these types of sources in the pilot exposure and risk assessment described in Chapter 4.

6 For example, the 2005 American Housing Survey, conducted by the U.S. Census Bureau indicates that some 14 million (or approximately 13% of) housing units are "within 300 feet of a 4-or-more-lane roadway, railroad or airport" (U.S. Census Bureau, 2006). Additionally, estimates developed for Colorado, Georgia and New York indicate that approximately 15-30% of the populations in those states reside within 75 meters of a major roadway (i.e., a “Limited Access Highway”, “Highway”, “Major Road” or “Ramp”, as defined by the U.S. Census Feature Class Codes) (ICF, 2005).
Children, in general and especially low SES children, are at increased risk for Pb exposure and Pb-induced adverse health effects. This is due to several factors, including enhanced exposure to Pb via ingestion of soil-Pb and/or dust-Pb due to childhood hand-to-mouth activity.

Once inhaled or ingested, Pb is distributed by the blood, with long-term storage accumulation in the bone. Bone Pb levels provide a strong measure of cumulative exposure which has been associated with many of the effects summarized below, although difficulty of sample collection has precluded widespread use in epidemiological studies to date.

Blood levels of Pb are well accepted as an index of exposure (or exposure metric) for which associations with the key effects (see below) have been observed. In general, associations with blood Pb are most robust for those effects for which past exposure history poses less of a complicating factor, i.e., for effects during childhood.

Epidemiological studies have observed significant associations between Pb exposures and a broad range of health effects. Many of these associations have been found at levels of blood Pb that are currently relevant for the U.S. population, with children having blood Pb levels of 5-10 μg/dL or, perhaps somewhat lower, being at notable risk.

Pb exposure is associated with a variety of neurological effects in children, notably intellectual attainment and school performance. Both qualitative and quantitative evidence, with further support from animal research, indicates a robust effect of Pb exposure on neurocognitive ability at blood Pb levels levels in the range of 5 to 10 μg/dL, and some analyses appear to show Pb effects on intellectual attainment of young children ranging from 2 to 8 μg/dL.

The staff concludes that it is appropriate to use log-linear concentration-response models for the qualitative risk assessment (described in Chapter 4) for neurocognitive ability in young children.

For children, the evidence is also robust for Pb-induced disruption of heme synthesis at blood Pb levels of 20-30 μg/dL. At blood Pb levels on the order of 10 μg/dL, and slightly lower, associations have been found with effects to the immune system, resulting in altered macrophage function, increased IgE levels and associated increased risk for autoimmunity and asthma.

In adults, epidemiological studies have consistently demonstrated associations between Pb exposure and increased risk of adverse cardiovascular outcomes, including increased blood pressure and incidence of hypertension. These associations have been observed with bone Pb and, for some studies with blood Pb levels below 10 μg/dL. Animal evidence provides confirmation of Pb effects on cardiovascular functions. For these Pb effects, particularly susceptible subpopulations include those with a higher baseline blood pressure. For example, African Americans, as a group, have greater incidence of elevated blood pressure than other ethnic groups.

Renal effects in adults, evidenced by reduced renal filtration, have also been associated with Pb exposures indexed by bone Pb levels and also with blood Pb below 10 μg/dL,
with the potential adverse impact of such effects being enhanced for susceptible subpopulations including those with diabetes, hypertension, and chronic renal insufficiency.

- Other Pb associated effects in adults occurring at or just above 10 μg/dL include hematological (e.g., impact on heme synthesis pathway) and neurological effects, with animal evidence providing support of Pb effects on these systems and evidence regarding mechanism of action.
REFERENCES


4 CHARACTERIZATION OF HEALTH RISKS

4.1 INTRODUCTION

This chapter presents the human exposure and health risk assessments conducted in support of the current review (throughout the remainder of this chapter, the term "risk assessment" will be used to refer to both the human exposure and health risk assessments collectively, unless specific reference to either the human exposure or health risk assessments is required). This risk assessment is being completed in two phases. The first phase (the pilot) is reflected in this first draft document. The second phase (the full-scale assessment) will be reflected in the second draft of this document. The pilot assessment is intended primarily as a demonstration of the risk assessment methodology being developed for the current review. Consequently, exposure and risk results presented here are considered preliminary. The pilot assessment presents exposure and risk assessments for two air quality scenarios (current conditions and attainment of the current NAAQS).

The risk assessment characterizes exposure and risk resulting from exposure to policy-relevant sources of Pb (see Section 2.1). Accordingly, the risk assessment is multi-pathway in nature, including consideration both for direct inhalation and incidental ingestion of Pb in soil and indoor dust that was originally released into outdoor air. In addition, because of the need to consider total Pb exposure in predicting blood Pb levels, the analysis also includes consideration of policy-relevant background Pb exposure. As described in Section 2.1, exposure pathways comprising background include diet, drinking water and Pb paint (with Pb paint potentially impacting both outdoor soil and indoor dust). 1

As with the last review (see Section 4.1.1), this risk assessment utilizes a case-study approach wherein a set of specific locations associated with policy-relevant Pb exposures are evaluated in detail. For the pilot analysis, three case studies have been selected including (a) a primary Pb smelter (in Herculaneum Missouri), (b) a secondary Pb smelter (in Troy, Alabama) and (c) a near roadway (urban) location in Houston, Texas. 2 Additional case studies may be

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1 In the pilot assessment, the contribution to food from air pathways is not explicitly included, such that dietary Pb exposure is treated as policy-relevant background. Further, although paint is a policy-relevant background source, for this analysis, it may be reflected somewhat in estimates developed for policy-relevant sources, due to modeling constraints (see Section 4.4.3.3.2 and 4.5.2).

2 Note, that the near roadway (urban) case study comprises a 1.5 mile road segment and the residents living within 200m of that road segment. Consequently, this case study provides perspective on the near roadway exposure scenario but is not intended to estimate total population risk for a larger urban or metropolitan area. Such an area would likely include a large number of such road segments with buffered residential populations.
evaluated for the full-scale analysis. The case studies modeled for the pilot have been selected to provide a preliminary perspective on the nature and magnitude of air-sourced Pb exposures and risk. In addition, they provide a range of exposure scenarios in which to test the risk assessment methodology developed for the current review. Because of differences in the exposure scenarios and available data at each of the case study locations, the approach used for modeling exposure and risk differs among the case studies. Results from this pilot assessment, as well as comments received from the public and CASAC will inform staff decisions on the number and type of case studies to be included in the full-scale assessment.

The remainder of this chapter is organized as follows. Section 4.1.1 provides an overview of the human health risk assessment completed in the last review of the Pb NAAQS in 1990 (USEPA, 1990a). Section 4.2 provides an overview of the scope of the pilot exposure and risk assessment, covering such topics as the conceptual model used in designing the analysis (4.2.1), the selection of health endpoints and case study locations (4.2.2 and 4.2.3 respectively), a description of the air quality scenarios covered in the pilot (4.2.4), and an overview of the key components of the pilot exposure and risk assessment (4.2.5). Following the discussion of the scope of the analysis, separate sections are dedicated to the exposure analysis (4.3) and risk assessment (4.4). Specifically, Section 4.3 documents the methods and results of the human exposure assessment completed for the analysis, which follows the analysis through the estimation of blood Pb levels for child populations at the three case study locations. Section 4.4 presents the methods and results of the health risk assessment which characterizes the distribution of IQ loss resulting from Pb exposure in the modeled child populations. Separate sections are dedicated to performance evaluation (4.3.6) and sensitivity analysis and uncertainty (4.4.3). Finally, a summary of the risk results generated for the pilot, including discussion of uncertainty and the identification of areas for potential enhancement for the full-scale analysis is presented in Section 4.5.

Additional technical detail regarding both the exposure analysis and risk assessment completed for the pilot analysis (beyond that presented in this chapter) can be found in Lead Human Exposure and Health Risk Assessments and Ecological Risk Assessment for Selected Areas (ICF, 2006; henceforth referred to as the Risk Assessment Report).

4.1.1 Overview of Risk Assessment from Last Review

In the risk assessment conducted in support of the last review, air quality scenarios were compared in terms of their impact on the percentage of modeled populations that exceeded specific blood Pb levels chosen with consideration of the health effects evidence at that time (USEPA, 1990). The 1990 analysis focused on both children (birth through 7 years of age) and middle-aged men residing in three case study locations (two near secondary Pb smelters and one...
The analysis also introduced the use of pharmacokinetic blood Pb modeling for children, although it used empirically-derived slope models for adult men to relate changes in air Pb to changes in blood Pb.

The following discussion presents a more detailed overview of the approach used in the 1990 risk assessment. For children, the staff used blood Pb levels of 10 and 15 μg/dL to evaluate effects of alternate NAAQS. These values were chosen with consideration of the health effects evidence at that time. Staff then used dispersion modeling (the Industrial Source Complex (ISC) model) combined with source characterization data for each point source to generate Pb air concentrations for each case study area. Statistically-derived relationships based on data from other industrial locations, including Pb smelters, that linked concentrations of Pb in air to Pb in indoor dust and outdoor soil were then used to predict Pb in these media for the three case study locations, based on the modeled air Pb concentrations. An uptake/biokinetic model was also developed to predict child blood Pb levels. This model was used in place of a statistically-based regression slope model to allow consideration for the dynamic nature of Pb exposure in children. EPA combined model-derived central tendency blood Pb levels with an estimated geometric standard deviation (GSD) reflecting inter-individual variability in blood Pb levels, to generate population distributions of blood Pb levels. These distributions were then used to estimate the percentage of children at each case study location that exceeded the specified blood Pb levels of interest for children (i.e., 10 and 15 μg/dL).

For adult men, the 1990 assessment used blood Pb levels of 10 and 12 μg/dL to compare relative effects of alternate NAAQS. The same approach was used for generating media concentrations for the adult analysis as was used for the child assessment. However, for adults as noted above, the 1990 analysis used statistically-derived slope models to relate air Pb to blood Pb levels with two versions of the slope models being employed: (a) the aggregate model which predicts blood Pb in adults based solely on air Pb levels (here a single slope factor captures both the direct inhalation pathway as well as the more complex pathway of Pb deposition to soil and dust followed by incidental ingestion) and (b) the disaggregate model which uses media-specific slopes to predict blood Pb based on Pb concentrations in soil, dust and air. Since the projected blood Pb levels were mean population levels, a GSD term was included to develop population-level blood Pb distributions. The GSD estimates for adults and children were derived from information on observed blood Pb levels in these subgroups. These population-level distributions were then queried to identify the percentage of adult men at each case study location with modeled blood Pb levels exceeding the levels of interest for adults (10 and 12 μg/dL).

The primary difference between the risk assessment approach used in the current pilot analysis and the assessment completed in 1990 involves the risk metric employed. Rather than estimating the percentage of study populations with exposures above blood Pb levels of interest
as was done in the last review (i.e., 10, 12 and 15 μg/dL), the current pilot analysis estimates the
degree of health decrement in study populations exposed to Pb. Specifically, the pilot analysis
estimates the distribution of IQ loss associated with Pb exposure for child populations at each of
the case study locations with that IQ loss further differentiated between background Pb exposure
and policy-relevant exposures.

4.2 SCOPE OF PB EXPOSURE AND RISK ASSESSMENTS

This section describes the scope of the pilot analysis, including key elements in the
design of both the exposure and risk assessments.

4.2.1 Conceptual Model for Human Health Risk Assessment

This section presents the conceptual model (Figure 4-1) for the exposure and risk
assessment intended to illustrate the elements pertinent to evaluating public health risks
associated with environmental Pb exposures.

For the purposes of this risk assessment, as noted earlier, “background” refers to sources
of, and exposures to Pb associated with pathways that do not involve the release of Pb to ambient
outdoor air. Included among these would be pathways associated with indoor Pb paint, Pb in
drinking water, Pb introduced to food during processing, etc. Those elements considered
“background” for this analysis are shown in Figure 4-1 in non-bold (regular) type. As shown in
Figure 4-1, the pilot assessment will include contributions from all background sources. Those
sources considered policy-relevant (i.e., associated with the release of Pb to outdoor air) are
shown in Figure 4-1 in bold type. As noted earlier, the pilot assessment results for both blood Pb
levels and risk are differentiated as to contributions associated with background sources and
policy-relevant sources.

Based on recommendations from CASAC and consideration of information presented in
the CD, the pilot assessment was designed to focus on children as the study population, blood Pb
as the dose metric, central nervous system as the target for effects, and IQ decrement as the
Figure 4-1. Conceptual model for Pb human health risk assessment.

NOTES
1 Components with gray background and solid borders will be included in this assessment. Components depicted in gray but without borders were not included in the Pilot analysis, but are being considered for inclusion in the Full Scale analysis. Further, a distinction is made between components linked to exposure pathways involving ambient air (shown in bold) and components involving other pathways (i.e., background).
2 Includes contributions of historical sources, including (but not limited to) emissions from the use of leaded gasoline, historical emissions from stationary sources, and exterior leaded paints.
metric associated with risk quantitation. Consequently, some of the elements presented in Figure 4-1 are not reflected in the pilot analysis (e.g., adult populations, internal dose metric of bone Pb, potential cardiovascular effects in adults and blood pressure changes). Those elements in the conceptual model which are reflected in the pilot analysis have solid bolded borders and are shaded. Those excluded from the pilot analysis have light borders and are not shaded.

- **Sources:** The focus of the pilot analysis is on policy-relevant sources. This includes both current sources of new Pb emissions (e.g., ongoing industrial point source and fugitive emissions) and re-emission of historically deposited Pb (e.g., re-entrainment of Pb deposited historically near roadways). However, because of the importance of characterizing total Pb exposures in modeling blood Pb levels, other (“background”) sources of environmental Pb (e.g., diet and drinking water) are also considered to varying degrees in the analysis.

- **Pathways:** Figure 4-1 is intended to generally illustrate the many pathways by which Pb emitted into the environment becomes available for human exposure. Those not passing through ambient air are considered “background” for the purposes of this assessment.

- **Routes:** The ingestion and inhalation routes are considered the primary routes of human exposures to environmental Pb. The ingestion route (including incidental dust and soil ingestion) is expected to have a greater contribution to the risk estimates. Both routes are included in this assessment.

- **Exposed Populations:** The Pb exposed populations can be characterized and stratified based on a variety of characteristics. Figure 4-1 identifies groups based primarily on age or lifestage, which has an influence on behaviours that can influence exposure or susceptibility (see Section 3.2 and 3.4.1 for additional detail on susceptible populations). It is recognized that more specific factors (e.g., calcium deficiency) also influence susceptibility. However, currently available data do not generally support quantitative modeling that differentiates between subpopulations on the basis of enhanced or reduced susceptibility to Pb effects (e.g., concentration response functions for IQ loss that differentiate between populations that are calcium deficient and those that are not).

- **Internal Disposition:** While Pb is distributed throughout the body, bone is an established site of internal accumulation of Pb, while blood is an established internal dose metric for purposes of both exposure and risk assessment. The pilot analysis relies on blood Pb with corresponding dose-response functions. However, the tools employed in modeling blood Pb levels in study populations recognize the role of bone as a reservoir with the potential to act as both a source and storage site.

- **Endpoints:** Figure 4-1 generally identifies the wide variety of health endpoints recognized in the CD (CD, p. E-8) as associated with Pb exposures. As mentioned previously, the endpoint of interest for this assessment is neurological effects in children and more specifically, IQ decrement.
• **Metrics:** Figure 4-1 generally recognizes that there are many metrics that might be considered for risk assessment. Recognizing the need for the metrics used in this assessment to have sufficient support for use in quantifying population health risk, the pilot analysis uses IQ decrement in children as its primary risk metric.

#### 4.2.2 Selection of Health Endpoint, Study Population, Dose-Metrics and Associated Concentration-Response Function

As recognized in Chapter 3 (e.g., Section 3.3.1), the CD notes that recent epidemiological studies have strengthened the consensus that the developing nervous system is the most sensitive endpoint in young children and that neurobehavioral deficits, including IQ impacts, appear to occur at lower levels than previously believed (i.e. at levels < 10 μg/dL and possibly <5 μg/dL). Consequently, for the pilot risk assessment, we selected the child neuro-developmental endpoint, specifically focusing on the IQ loss metric (see Section 3.3.1.1). This assignment of priority to children’s IQ as the endpoint assessed reflects consideration for evidence presented in the CD as well as advice received during our consultation with CASAC on the *Analysis Plan for Human Health and Ecological Risk Assessment For the Review of the Pb National Ambient Air Quality Standards* (USEPA, 2006a) (hereafter referred to as the *Analysis Plan*) (Henderson, 2006).

As discussed in Section 3.3.1.2, a pooled analysis (Lanphear et al., 2005) was selected as the basis for defining the relationship between Pb exposure and IQ loss. Furthermore, the pilot analysis will use log-linear functions based on concurrent and lifetime average blood Pb metrics, as discussed in Section 3.3.1.2. This decision was made after considering a range of concentration-response functions provided in the pooled analysis (e.g., cubic-spline and log-linear models).

#### 4.2.3 Selection of Case Study Locations

In identifying the three case study locations modeled for the pilot analysis, the staff followed the approach presented in the *Analysis Plan*. The *Analysis Plan* included examples of ambient Pb emissions sources and exposure scenarios that should be considered in selecting case studies. These include a study area near a primary Pb smelter, one near another (or multiple) significant stationary Pb sources, and a study area near an urban roadway. During our consultation on the plan, CASAC generally expressed support for the approach, emphasizing the need for inclusion of a near roadway location to provide coverage for the potential impact of historically deposited (auto-sourced) Pb on public health (Henderson, 2006). The case studies we have included in the pilot reflect these three situations and also reflect consideration for three additional factors described in the *Analysis Plan*: (a) availability of site-specific monitoring data for ambient air Pb, (b) availability of measurement data for other environmental media (soil and...
indoor dust) and biomonitoring of Pb exposure (i.e., blood Pb levels), and (c) consideration for
demographics and socioeconomic factors related to Pb exposure and risk.

The three case study locations modeled for the pilot assessment are (a) a primary Pb
smelter (in Herculaneum Missouri), (b) a secondary Pb smelter (in Troy, Alabama) and (c) a near
roadway location in Houston, Texas. The Herculaneum primary Pb smelter represents a
relatively large point source that has been active for over a century and for which there exist a
large amount of site-specific data characterizing both media concentrations (soil, indoor dust,
outdoor air) and population blood Pb levels. The secondary Pb smelter represents a somewhat
smaller point source (compared to Herculaneum) with relatively less site-specific data
characterizing media concentrations and exposure levels. Finally, the near roadway (urban)
location meets the criteria that we identified for a near roadway exposure scenario (e.g., little
influence of point source emissions in the immediate vicinity, conditions which could contribute
to population exposure to near roadway-deposited Pb, and residential populations located near
the modeled road segment). We had also intended to identify a multiple-source case study
location (i.e., a location with multiple point and/or area sources with none being dominant).
However, identification of a satisfactory location proved challenging preventing inclusion of this
type of case study in the pilot. Efforts may continue, during the full-scale analysis, to identify a
suitable multiple-source location.

Each of the three case study locations modeled for the pilot is briefly discussed below
including: (a) details related to each location which may be relevant to exposure and risk, (b) (for
the two point source locations) the magnitude of reported emissions for the facility, (c) the
magnitude of ambient air Pb levels at monitors associated with the case study location in the
context of overall monitored levels in the U.S. and (d) the availability of site-specific data
characterizing key media and Pb exposures (e.g., soil, blood Pb level data). The approach used
to identify the near roadway (urban) case study location, which is fairly complex, is also
discussed in some detail below.

4.2.3.1 Primary Pb Smelter Case Study

The facility in Herculaneum Missouri is the only remaining primary Pb smelter operating
within the U.S. It has been in operation for over a century contributing to Pb contamination of
the area surrounding the facility.\textsuperscript{3} However, over the past decade or more, remediation of yard

\textsuperscript{3} Portions of this study area comprise an active Superfund site and are subject to ongoing evaluation under
the Superfund program administered by the Office of Solid Waste and Emergency Response. Methods used in
conducting the human health exposure and risk assessment for the pilot analysis have been selected to address policy
soils have been completed for a significant number of the residences near the facility and in addition, the facility has extended its site boundaries to encompass many of the more heavily impacted houses and lots. The remediation activity introduced a complication to the risk modeling, especially aspects involving characterization of the relationship of ambient air Pb and residential soil Pb to indoor dust Pb (see Section 4.3.2.4).

The U.S. Census estimates that, as of 2000, a total of 37,562 people live within 10 km of the facility (U.S. Census Bureau, 2005). Of these, 3,880 are children (0-7 years of age). Specifically, 171 children are within 2 km of the facility, 1,545 children are between 2 and 5 km and 2,164 children are between 5 and 10 km from the facility.

Total annual Pb emissions to ambient air for the Herculaneum facility are estimated at 25 tons/year for 2005 (see Section 2.3.4.6).

As of 2005, there are nine TSP air monitors located within 10 km of the Herculaneum facility (as identified in EPA’s Air Quality System database). Annual average concentrations of Pb recorded at these monitors for 2005 ranged from (0.057 to 1.56 μg/m³) (USEPA, 2006b). Of the nine TSP monitors located within 10 km of the facility, all fall within the top 30% of the 2005 annual average levels for monitors in the database, with four of the nine monitors falling in the top 10% (see Risk Assessment Report, Section 3.1.5 for additional detail).

The Herculaneum facility has more site-specific monitoring data available to support risk assessment then the other two case study locations, including residential yard soil, indoor dust and road dust Pb measurements collected in areas potentially impacted by the facility. In addition, the Agency for Toxic Substances and Disease Registry (ATSDR) has conducted a number of health consultations which involved the collection of blood Pb measurements for children (ATSDR, 2003). The area within the city limits of Herculaneum is designated non-attainment for the Pb NAAQS and a State Implementation Plan (SIP) was approved in 2002 (67 FR 18497). EPA determined the existing SIP to be inadequate to attain the current NAAQS in 2006 (71 FR 19432), and consequently a revised SIP is under development for the area. Air dispersion modeling will play a role in development of this plan (Note, we intend to consider that updated air quality modeling in conducting the full scale analysis for this case study).

The significant amount of site-specific data available for Herculaneum, paired with air dispersion modeling for the facility conducted in support of SIP development for Pb, provides a strong data set for this study area which enhances the modeling of exposure and risk. However, the Herculaneum case study location also has a number of attributes that add complexity to the modeling of Pb exposure and risk including (a) complex terrain and meteorology which questions relevant to the Pb NAAQS review and consequently, may differ from those used by the Superfund program.
complicates the modeling of Pb transport in ambient air, (b) a large and complex facility with significant opportunity for fugitive emissions which makes source characterization challenging, and (c) a history of remediation activities which has contributed to widely varying residential soil Pb concentrations across the town.

### 4.2.3.2 Additional Point Source (Secondary Smelter) Case Study

The secondary Pb facility in Troy, Alabama, is one of 15 secondary Pb smelters operating within the U.S. as of 2002 (see Section 2.3.4.7). Secondary Pb smelters produce Pb from scrap and provide the primary means for recycling Pb-acid automotive batteries.

According to the U.S. Census (US Census, 2005), as of 2000, a total of 17,901 people live within 10 km of the facility. Of these, 1,672 are children (0-7 years of age). Specifically, 187 children are within 2 km of the facility, 896 children are between 2 and 5 km and 589 children are between 5 and 10 km from the facility.

Total annual Pb emissions to ambient air for the Troy facility are estimated for 2002 at 4.56 tons/year (EC/R, 2006), which places the facility as the third highest emitter within its source category.

There are two TSP air monitors located within 1 km of the Troy facility, specifically located between 300 and 800 m from the facility (as identified in EPA’s Air Quality System database). Annual average concentrations of Pb recorded at these monitors for 2000 range from 0.198 to 0.383 μg/m³ (USEPA, 2006b). These TSP values fall within the top 15% of TSP annual average values for 2000 (see Risk Assessment Report, Section 3.2.5 for additional details).

In contrast to the Herculaneum facility, we have not identified soil or indoor dust Pb measurements for this case study location and we did not identified systematic blood Pb assessments for children in the area. This means that the exposure assessment conducted for the Troy facility is more dependent on modeling and consideration of measurements available for similar locations and there is less opportunity for rigorous performance evaluation of modeling due to the lack of site-specific measurement data.

### 4.2.3.3 Near Roadway (Urban) Location Case Study

In choosing a location for this case study, focus was placed on identifying a location with ambient monitors that would provide reasonable coverage for near roadway Pb entrainment. The team reviewed available air monitoring data from two sources (a) the EPA’s Air Quality System database, focusing on TSP monitoring (USEPA 2006b) and (b) a series of air monitors placed by Desert Research Institute (DRI) in support of an urban particulate matter speciation study (ICF, 2005). Ultimately, the DRI monitors were favored for the near roadway (urban) case study because of their particulate matter speciation data which may be used in the full-scale analysis to support more detailed source-apportionment of the Pb concentrations. In addition, in selecting...
the location for this case study, we also considered modeling conducted for the Agency’s 1999 NATA national-scale assessment (USEPA, 2006c). As described in Section 2.4.5.1, air dispersion modeling is conducted in this assessment at the census tract-level and includes modeling of all sources in the 1999 NEI. Because the NEI does not include re-suspension as an emissions source, instances where measured (monitored) air Pb levels exceed predicted ambient air Pb concentrations may indicate potential contributions from re-entrainment. Therefore, in choosing the near roadway (urban) case study location, we also favored those locations with a positive difference between monitored air Pb levels and the national-scale assessment modeled ambient air concentrations. And finally, we also, favored those DRI monitor locations that have relatively low area and point Pb emissions within 20 km, thus reducing the chance of Pb emissions besides re-entrainment having a significant impact on Pb air concentrations in the study area (i.e., increasing the chances that re-entrainment is the dominant Pb air source).

Ultimately, a case study location in Houston TX, comprising a 1.5 mile road segment and the adjacent residential population, was selected to represent the near roadway exposure scenario based on the above criteria. This location has the following characteristics, which contributed to its inclusion in this analysis: (a) it is located in a relatively dry location where resuspension is likely to occur, (b) DRI measured Pb concentrations in air are higher than the NATA modeled air concentration estimates, and (c) there are relatively few point sources of Pb emissions within 20 km of the site (no point sources within 5 km and only two within 20 km). Note, however that one of the two emissions sources is a large airport (airports are a potentially significant source of Pb emissions due to the continued use of Pb in some aviation fuels) (CD Section 2.2.4).

According to the U.S. Census (U.S. Census Bureau, 2005), as of 2000, a total of 1,950 people live within the 200m buffer area surrounding the modeled road segment. Of these, 320 are children (0-7 years of age). It is important to note, that the near roadway (urban) case study was developed primarily to demonstration test the exposure and risk characterization approach developed for this type of case study. It is likely that a far larger number of children would be associated with near roadway exposures if a study area similar to that used for the two point-source case studies were considered (i.e., a larger number of road segments similar to that evaluated here would be located within a 10 km-radius urban or suburban area).

The DRI monitors measured PM$_{10}$ and collected samples over three days in February, 2001. These resulted in an average air Pb concentration over this sampling period of 0.0030 $\mu$g/m$^3$. Compared with annual average Pb concentrations obtained from the 36 PM$_{10}$ monitors in the Air Quality System database (USEPA 2006b) the DRI monitor average falls around the lower 20th% (see Risk Assessment Report, Sections 3.3.4 and 4.3.2.1 for additional detail on monitored ambient air data used in this assessment).
As with the Troy case study location, the Houston near roadway location does not have any site-specific soil Pb data or monitored Pb exposure data in the form of blood Pb levels. This means that the exposure assessment conducted for the near roadway (urban) case study is more dependent on surrogate data collected from other industrial locations and there is less opportunity for rigorous performance evaluation of modeling due to the lack of site-specific measurement data.

**4.2.4 Air Quality Scenarios Covered in the Pilot Analysis**

The pilot analysis involved exposure and risk modeling for the current conditions and current NAAQS attainment air quality scenarios. Consideration for alternate NAAQS scenarios will be covered as part of the full-scale analysis.

The current NAAQS attainment air quality scenario only applies to the primary Pb smelter case study since monitors located within that study area have shown exceedances of the Pb NAAQS (USEPA, 2006b). Monitors associated with the other two case study locations have not had recent exceedances of the Pb NAAQS and consequently the current conditions and current NAAQS attainment air quality scenarios are identical for these two locations.

Consideration of the current NAAQS attainment scenario for the primary Pb smelter case study involved adjustment of modeled air quality results to reflect attainment of the current NAAQS (see Section 4.3.2.1.1 for discussion of air quality modeling for this case study). Specifically, any quarterly-average modeled air concentrations at receptor points within the study area estimated to exceed the NAAQS were reduced to the NAAQS level of 1.5 ppm. Annual average air concentrations were then recalculated for those receptor points based on these adjusted quarterly averages (as noted in Section 4.3.2, exposure modeling for the pilot analysis is based on annual average media concentrations).

**4.2.5 Overview of the Exposure and Risk Modeling Approach Used in the Pilot Analysis**

This section provides an overview of the modeling approach implemented for the pilot analysis including: (a) consideration for recommendations provided by CASAC regarding exposure and risk modeling, (b) description of the child study population evaluated for the pilot analysis, (c) spatial scale of the analysis and the type of spatial template used in modeling, (d) overview of key modeling steps (predicting media concentrations, modeling exposure, modeling risk), (e) performance evaluation completed in support of the analysis and (f) the approach used to address uncertainty.
4.2.5.1 CASAC Consultation Regarding Human Exposure and Health Risk Assessment

The staff consulted with the CASAC on the draft Analysis Plan in June, 2006 (Henderson, 2006). Some key comments provided by CASAC members on the Analysis Plan included: (a) placing a higher priority on modeling the child IQ metric than the adult endpoints (e.g., cardiovascular effects), (b) recognizing the importance of indoor dust loading by Pb contained in outdoor air as a factor in Pb-related exposure and risk for sources considered in this analysis, and (c) concurring with use of the IEUBK biokinetic model, suggesting that the linking of a probabilistic exposure module with IEUBK run in batch-mode to generate blood Pb estimates for individual modeled children (as described in the Analysis Plan) should be given lower priority by staff in the pilot phase.4

The above comments, together with information presented in the CD, are reflected in the design of the pilot analysis. Important ramifications of this decision include: (a) a focus on modeling exposure and risk (IQ loss) for the child cohort in the pilot analysis, (b) emphasis placed on explicitly considering the linkage between ambient air-borne Pb and Pb in indoor dust and (c) development of a GIS-based blood Pb modeling approach that combines IEUBK with spatially-differentiated exposure (dose) modeling, but still relies on the GSD-based approach for ultimately characterizing variability in blood Pb levels related to behavior and biokinetics.

4.2.5.2 Child Study Population

As mentioned earlier, the pilot analysis focuses on estimating IQ loss in children associated with exposure to Pb derived from policy-relevant sources within each study area. The risk assessment conducted for each case study uses a simulated child population that begins exposure in the same year and continues that exposure for 7 years (i.e., the study population is assumed to be a single group, which begins exposure in the study area all at the same age and

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4 Note, as discussed in Section 4.3.3.3, for the pilot we did implement a probabilistic population exposure model, however this model is based on coverage for inter-individual variability in behavior related to Pb exposure using the GSD approach described in the IEUBK Technical Support Document (USEPA 1994) and does not involve deterministic modeling of individual Pb exposure levels outside of IEUBK (i.e., the population-exposure modeling used in the pilot does not utilize the approach deemphasized by CASAC). Note also, that in relation to blood Pb modeling, CASAC has expressed support for the use of empirically-based regression models in addition to biokinetic models. As discussed in Section 4.2.5.5.2, the pilot analysis utilizes both categories of models with biokinetic blood Pb modeling being used to generate exposure and risk estimates in the analysis proper, and regression-based blood Pb modeling being considered as part of the sensitivity analysis completed for the pilot (see Section 4.4.3).
continues that exposure until all modeled children are 7 years of age). Furthermore, it is assumed that no migration or immigration of these children occurs during this simulation period (i.e., none of the children move out of the study area and no children move in). With regard to characterizing Pb concentrations in media as part of exposure analysis, it is assumed that the media concentrations, after being defined for the start period of the simulation (when the simulated children are 1 day of age), hold steady and do not change. Note, however, that a variety of exposure factors and physiological parameters used in blood Pb modeling are allowed to change as each simulated child ages. We are considering potential refinements to this somewhat simplified modeling approach to address the needs of the full-scale assessment.

**4.2.5.3 Timeframe for Current Conditions**

The current conditions scenario modeled for the pilot is generally described as reflecting conditions for the timeframe 2000 to 2005. As summarized in the last row of Table 4-1, 2000-2005 data were used to characterize Pb media concentrations for the primary Pb smelter. Information used in the air and soil modeling for the secondary Pb smelter was collected between 1997 and 2000. And finally, the information used to characterize Pb concentrations for the near roadway (urban) case study reflects conditions in 2001.

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5 Modeling of blood Pb levels for the child population includes contributions representative of prenatal Pb exposure.

6 While air concentrations of Pb exhibit notable temporal variation, particularly near point sources (see Section 2.4.3.1.3), less temporal variation is expected for other media. For the purposes of this pilot analysis, however, temporal variation (within or across years) is not simulated for any of the media.
Table 4-1. Timeframe (years) reflected in the characterizations of Pb media concentrations used in the pilot risk assessment.

<table>
<thead>
<tr>
<th>Modeling input</th>
<th>Primary Pb smelter</th>
<th>Secondary Pb smelter</th>
<th>Near roadway (Urban)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(post-2001 SIP emissions data)</td>
<td>(stack test data used in source characterization)</td>
<td>(DRI monitor data)</td>
<td></td>
</tr>
<tr>
<td>(timeframe for soil sampling at site)</td>
<td>(modeled based on air deposition)</td>
<td>(near roadway soil data collected in Corpus Christi, TX)</td>
<td></td>
</tr>
<tr>
<td>(given that indoor dust modeling relies on both outdoor air data and outdoor soil data, the timeframe for indoor dust modeling reflects data used in characterizing the other two media)</td>
<td>(estimated based on ambient air concentrations and outdoor soil concentrations)</td>
<td>(given that indoor dust modeling relies on both outdoor air data and outdoor soil data, the timeframe for indoor dust modeling reflects data used in characterizing the other two media)</td>
<td></td>
</tr>
</tbody>
</table>

4.2.5.4 Spatial Scale and Resolution

Exposure modeling conducted for the pilot is based on spatial templates customized for each case study location. These spatial templates divide the study area into smaller exposure zones which form the basis for exposure and risk modeling. For the point source-related case study locations (primary and secondary Pb smelters), these exposure zones are a combination of U.S. Census blocks and/or block groups. By contrast, the near roadway (urban) case study uses exposure zones running parallel to the selected road segment, reflecting the focus placed on re-entrainment of near roadway dust and the reduction in Pb concentrations with distance from the road segment.

7 US Census block groups vary in size from several city blocks in densely populated urban areas to many square miles in less populated rural areas. Their population count varies from 600 to 3000 people per block group with the typical block group in the U.S. containing 1,500 people. US Census blocks are more refined than block groups and typically contain several hundred people or less. Their size can vary from a single city block in urban areas to multiple square miles in less populated rural locations.
At all case study locations, fate and transport modeling and/or empirical data are used to characterize Pb media concentrations (e.g., outdoor air, soil and indoor dust) for each exposure zone. A central tendency estimate of concurrent and lifetime average blood Pb levels is derived for the children within each exposure zone. Inter-individual variability in blood Pb levels between children within a given zone is considered through the use of a statistically-derived GSD reflecting variability in blood Pb levels for children living in areas with similar levels of Pb contamination. Additional detail on the spatial templates used for each case study location can be found in Section 4.3.1.

4.2.5.5 Overview of Analytical Steps

As illustrated in Figure 4-2, the risk assessment generally includes four analytical steps: (a) characterization of the fate and transport of Pb released into outdoor air, including the dispersion of Pb away from the point of release and the deposition of Pb onto surfaces, (b) prediction of the resulting concentration of Pb in media of concern including outdoor air, outdoor soil and indoor dust, (c) use of these Pb concentrations together with estimates of Pb in background media including diet to model blood Pb levels using biokinetic modeling and (d) relating modeled blood Pb levels in children to degrees of IQ loss using concentration-response functions derived from epidemiology studies. Figure 4-2 identifies the key input data sets, modeling steps and intermediate model output in each of the four analytical steps. The first two steps are employed in estimating media concentrations, while the third step completes the exposure assessment and the fourth is the risk assessment step. Each of the key elements of each step is briefly described below. Details of the exposure and risk assessments, along with exposure and risk estimates, are described in Section 4.3 and 4.4, respectively.

Note, that the modeling approach discussed below is based on the analysis completed for the current conditions air quality scenario for the pilot analysis. The method used for the current NAAQS attainment scenario is virtually identical, except that several U.S. Census blocks within the primary Pb smelter case study area with air Pb levels exceeding the current NAAQS (1.5 μg/m³) had those levels reduced to values just meeting the NAAQS. Those adjusted ambient air values were then used in exposure and risk modeling.
Figure 4-2. Overview of analysis approach for the pilot analysis.

Characterizing ambient air levels and deposition to soil

- Ambient (near roadway) monitoring data combined with spatial gradients for roadway air concentrations identified in the literature (near-roadway)
- Air dispersion modeling (primary, secondary Pb smelters)
- Lead deposition rates across study area

Characterizing soil and indoor dust concentrations

- Multi-media fate/transport model (secondary Pb smelter)
- Outdoor residential soil concentrations
- Site-specific soil monitoring data combined with statistical extrapolation (primary Pb smelter)
- Surrogate soil data for near roadway locations (identified in the literature) (near-roadway)

Characterizing blood lead levels

- Background lead exposure levels
  - diet
  - drinking water
  - indoor paint (actually reflected in dust modeling)
- Demographics (distribution of children within study areas)
- Biokinetic blood lead modeling (all 3 case studies)
- Probabilistic modeling of blood lead levels for children within each case study location (all 3 case studies)
- GSD reflecting inter-individual variability in behavior related to lead exposure and biokinetics

Characterizing risk (IQ loss)

- Distribution of IQ loss for study populations (partitioned between background and NAAQS-relevant exposures)
- Health effects incidence estimation (all 3 case studies)
- Concentration-response function
  - log-normal distribution (Lanphear pooled analysis)
  - breakpoint reflecting confidence in CR function
  - effects estimation based on concurrent and lifetime average blood Pb metric
4.2.5.5.1 Characterizing Media Concentrations

As part of the exposure assessment, the staff has estimated Pb concentrations in ambient media and indoor dust using a combination of empirical data and modeling projections. We recognize that Pb in these media may be derived from newly or historically emitted Pb, both of which are considered policy-relevant. A combination of empirical data and modeling was used to characterize Pb concentrations in media for the three case studies. The use of empirical data brings with it uncertainty related to the potential inclusion of background source signals in these measurements (e.g., house paint contributions to indoor dust and outdoor soil Pb). Conversely, the use of modeling tools also introduces its own uncertainties (e.g., model and parameter uncertainties). Both of these uncertainties are recognized in Section 4.4.3. Specific approaches used at the three case study locations are briefly described below.

Characterization of Pb in ambient air at the three case study locations relies on (a) dispersion modeling of stack and fugitive emissions (for the primary and secondary Pb smelter case studies) and (b) the use of ambient monitor data (for the near roadway (urban) case study). For the near roadway (urban) case study, monitoring data in the vicinity of the roadway were combined with information characterizing the spatial gradient of particulate matter concentrations near roadways (see Section 4.3.1.3) to estimate Pb air concentrations in exposure zones extending out to 200m along both sides of the modeled road segment. Although source-apportionment methods are being considered for the near roadway (urban) case study as a means for identifying the re-entrained component of the monitored signal, that step has not been taken in the pilot phase of the assessment.

Characterization of Pb concentrations in outdoor soil, resulting from deposition of airborne Pb is based on the use of (a) existing site-specific measurements (primary Pb smelter), (b) surrogate measurements obtained from the literature for similar locations (near roadway (urban) case study) and (c) fate and transport modeling (secondary Pb smelter). In the case of the primary Pb smelter, soil Pb concentration data were available for a zone close to the facility and statistical extrapolation from the available empirical data was used to predict soil levels for portions of the study area beyond this sampling zone.

Pb in indoor dust may be derived from Pb in ambient air or soil (Adgate et al., 1998, Von Lindern, 2003). Mechanisms by which this occurs include penetration indoors of Pb entrained in ambient air, with subsequent deposition to indoor dust, and transport of outdoor soil indoors. To predict concentrations of ambient Pb in indoor dust, we have relied on empirical (regression) models that relate indoor dust to outdoor air Pb and/or outdoor soil Pb (USEPA, 1989).

Additional detail on methods used to characterize media Pb concentrations for each case study location can be found in Section 4.3.2.
4.2.5.5.2 Exposure Assessment

Exposure assessment, in the context of this Pb risk assessment, includes the prediction of blood Pb levels based on assessments of exposure to Pb contained in various media (e.g., ambient air, diet, water, indoor dust). For this analysis, blood Pb levels were predicted using both biokinetic models IEUBK and Leggett. The same fundamental approach was used to estimate population distributions of blood Pb levels at each of the three case study locations. This approach involved three steps:

- **Characterize exposure levels for both policy-relevant background and policy-relevant pathways for each exposure zone within a given study area:** Approaches discussed in Section 4.2.5.5.1 generated ambient air Pb, soil Pb and indoor dust Pb concentrations for each exposure zone. These policy-relevant media concentrations represent average levels contacted by children residing within each zone. In contrast, the characterization of background exposure levels was not site-specific and instead, is based on national estimates of Pb in the diet and drinking water for children (as noted earlier, potential exposure to Pb paint indoors was not explicitly modeled, but is likely captured as part of indoor Pb dust exposure, which is treated as policy-relevant in the pilot).

- **Use biokinetic models to predict central tendency blood Pb levels for children within each exposure zone:** Biokinetic blood Pb modeling is used to generate central tendency blood Pb levels for each modeling zone. Further, blood Pb levels are parsed into the fraction associated with policy-relevant background versus that associated with policy-relevant sources with the latter category being further subdivided into inhalation, soil ingestion and indoor dust ingestion pathway contributions.

- **Implement probabilistic exposure model to generate population-distribution of blood Pb levels for children in each case study location:** Apply a probabilistic model that generates a distribution of simulated blood Pb levels for the children in each study area based on consideration for three key factors: (a) the central tendency blood Pb levels generated for each exposure zone in the last step, (b) demographic data (distribution of children 0-7 years of age) across the zones comprising a given study area and (c) consideration for a GSD characterizing inter-individual variability in blood Pb (e.g., reflecting differences in behavior and biokinetics related to Pb).

The approach described above produces a distribution of modeled child blood Pb levels for each case study location. Each of the individual modeled child blood Pb levels is further differentiated to show the relative contribution of background and policy-relevant sources to total blood Pb. It is important to point out that the methods available for characterizing exposures to

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Although emphasis was placed on biokinetic models in predicting blood Pb levels for children in the pilot analysis, a statistical (regression) based blood Pb model (Lanphear et al., 1998) was included as part of the sensitivity analysis (see Section 4.4.3.1).
Pb in soil and indoor dust do not allow us, at this time, to differentiate between older historically-deposited Pb and Pb that is more recently emitted to the air (with subsequent impacts to soil and indoor dust). Specifically, for the pilot analysis, we could not quantify (with reasonable confidence) the fraction of blood Pb resulting from contact with Pb which was emitted to the air some time in the past, from that emitted more recently (currently). For purposes of this analysis, both categories of Pb emissions are considered part of policy-relevant sources and are reflected in aggregate in our exposure and risk results. This issue will continue to be researched as part of the full scale analysis.

Additional detail on exposure assessment completed for the pilot can be found in Section 4.3.

4.2.5.5.3 Risk Characterization

Risk characterization for the pilot assessment involves generating a distribution of IQ loss estimates for the set of children simulated in the exposure assessment (see Section 4.2.5.5.2). Specifically, estimated blood Pb levels are combined with a blood Pb concentration-response relationship for the health decrement of interest, in this case IQ loss. As discussed in Section 4.2.2, the concentration-response relationships used here are the log-normal concentration-response functions for concurrent and lifetime average blood Pb concentrations, respectively, from a pooled analysis of epidemiology studies focusing on IQ loss in children (Lanphear et al., 2005).

For this pilot assessment, we also selected cutpoints of 2.4 μg/dL (concurrent exposure metric) and 6.1 μg/dL (lifetime average exposure metric) as blood Pb levels below which IQ loss was not to be predicted. The selection of these cutpoints is based on consideration for the blood Pb level at which our overall confidence in being able to characterize the shape of the concentration-response functions diminishes significantly. For example, for the concurrent blood Pb concentration response function, <5% of the children from the pooled analysis had concurrent blood Pb levels below 2.4 g/dL (Lanphear et al., 2005), which suggests an increased uncertainty in the functional form below this level of exposure.

As with the exposure analysis results, we developed and present risk results (IQ loss estimates) that differentiate between policy-relevant exposure pathways and policy-relevant background. From the distributions of IQ loss generated for the pilot, we have produced two categories of risk metrics:

- Population risk percentiles: The degree of IQ loss associated with policy-relevant exposure pathways for specific percentiles of the child population (e.g., the 50\(^{th}\), 90\(^{th}\), 95\(^{th}\), 99\(^{th}\) percentile modeled child). This category of metric provides perspective on the distribution of IQ loss resulting from policy-relevant exposure pathways in each
study area, ranging from the typical or average child (50th percentile, mean) to children experiencing higher exposures (90th, 99th percentiles).

- Child frequency counts associated with specific risk percentiles: Number of children, for a given study area, associated with each of the population percentiles (e.g., 25 children in a particular study area population are predicted to have risk levels at or above the 99th percentile). This risk metrics provides a perspective on the number of children associated with various degrees of IQ loss for a particular case study.

Additional detail on the risk characterization completed for the pilot can be found in Section 4.4.1.

4.2.5.6 Performance Evaluation

To the extent possible, existing datasets, including both site-specific data as well as measurements for surrogate locations have been used to support performance evaluation of exposure modeling completed for the pilot. Specific modeling steps subjected to performance evaluation include:

- Air dispersion modeling (primary and secondary Pb smelter case studies): modeled air Pb concentrations have been compared with data from ambient air monitors within the study areas.
- Prediction of soil Pb levels (secondary Pb smelter): soil Pb levels generated using fate and transport modeling for the secondary Pb smelter case study have been compared with soil Pb data for similar locations identified in the literature.
- Modeled blood Pb levels (all three case studies): modeled blood Pb levels for the primary Pb smelter case study location have been compared with blood Pb data available for that study area. In addition, central-tendency estimates of blood Pb for the three case study locations have been compared with general U.S. population blood Pb levels (age group-matched) from NHANES IV(CD, Table 4-1).

The results of the performance evaluation steps described above, provide insights into the degree of confidence associated with a particular analytical step. Consequently, performance evaluation can be viewed as a component of the overall effort in the pilot analysis to consider the potential impact of sources of uncertainty on exposure and risk estimates.

Additional detail on the performance evaluation completed for the pilot can be found in Section 4.3.6.

4.2.5.7 Uncertainty/Sensitivity Analysis

For the pilot analysis, we used sensitivity analysis techniques to examine the issue of uncertainty and its impact on exposure and risk estimates. Specifically, we used a "one element
at a time elasticity analysis" approach, in which the full risk model was run with one of the selected modeling elements adjusted to reflect an alternate (bounding if possible) input value or modeling choice. The results of that run with the modified modeling element would then be compared to the "baseline risk" run to determine the magnitude of the impact on risk results generated by modifying that one modeling element.

The sensitivity analysis completed for the pilot analysis focused on those modeling elements (including input datasets and modeling steps) believed to have the greatest potential for significantly impacting exposure and risk modeling (e.g., oral uptake factor, inter-individual blood Pb variability GSD, biokinetic model, concentration-response function for IQ loss). Consequently, this initial step of selecting modeling elements to include in the sensitivity analysis does reflect professional judgment on the part of the staff.

Ultimately, this type of sensitivity analysis allows us to (a) determine which of the modeling elements included in the sensitivity analysis has the greatest impact on risk modeling (this can be used to guide future efforts to refine the overall risk model) and (b) obtain a semi-qualitative feel for the potential magnitude of overall uncertainty in the risk results. The latter point deserves additional discussion. Although, as noted earlier, this type of sensitivity analysis does not allow a rigorous assessment of probabilistically-defined confidence in specific risk percentiles to be quantified (that only being possible with a formal uncertainty analysis), by reviewing the magnitude of the impacts of key modeling elements on risk percentiles, it may be possible to gain perspective on the degree of overall uncertainty associated with the risk results (e.g., orders of magnitude, less than one order of magnitude etc).

Note, that in discussing uncertainty related to the pilot analysis, in addition to presenting the results of the sensitivity analysis, the results of the performance evaluation are also discussed.

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9 Ideally, an uncertainty analysis would utilize probabilistic simulation to generate a distribution of exposure and risk estimates reflecting overall confidence (uncertainty) in key modeling elements including input parameters and modeling steps. However, this type of comprehensive uncertainty analysis requires that each input parameter and modeling step be assigned a confidence distribution (for input parameters) or confidence weight (for different modeling options). We felt that sufficient information did not currently exist to derive these types of confidence distributions and weights for all key modeling elements used in the pilot analysis. Consequently, rather than implementing a probabilistic uncertainty analysis in the pilot analysis, we used the sensitivity analysis approach described here to gain insights into key sources of uncertainty.

10 For purposes of the sensitivity analysis, the "baseline run" was defined as a full risk run for the primary Pb smelter case study involving the following modeling choices: IEUBK biokinetic model and the concurrent blood Pb metric. It is important to emphasize that the defining of a baseline run does not place greater confidence in this particular combination of modeling elements, but rather reflects the need to have a set of risk results for use in gauging the magnitude of the impact of alternative modeling elements on risk results.
(since they identify potential areas of uncertainty). Additionally, a qualitative discussion of other potential sources of uncertainty, not addressed in the sensitivity analysis and performance evaluation, is also included.

Additional detail regarding consideration for uncertainty in the pilot (including the sensitivity analysis) can be found in Section 4.4.3.

4.3 HUMAN EXPOSURE ASSESSMENT

This section describes the methods and results of the exposure assessment for the pilot analysis, including the performance evaluation. The section begins with a description of the GIS-based spatial template that formed the basis for modeling population-level exposure at each case study with consideration for demographics and the spatial variability of media Pb concentrations.

4.3.1 Spatial Templates

4.3.1.1 Primary Pb Smelter Case Study

A combination of U.S. Census block groups and the more spatially-refined blocks were used as spatial units in the exposure assessment. The decision whether to differentiate a block group into its constituent blocks for purposes of exposure modeling was based on consideration for the range of air concentrations across the blocks comprising a block group. If the ratio of the maximum block-level air concentration (within a given block group) to the average air concentration for that block group exceeded 2.0, then that block group was divided into blocks. Note, that those blocks and block groups with zero child populations were excluded from modeling. This approach resulted in 22 block groups and 201 blocks being identified within the 10 km radius study area for the primary Pb smelter. Generally, as would be expected, the more differentiated blocks are located nearer to the facility where the air concentration gradient is larger. However, in some instances, larger blockgroups further from the facility included sufficient air concentration gradient to warrant modeling using smaller blocks.

11 Initially, we considered modeling exposure across the entire study area at the more-refined US Census block-level, but concerns over model run time (given the tight schedule for the pilot analysis) argued against this level of refinement unless necessary to generate defensible exposure and risk estimates. As noted here, consideration for the degree of variability in modeled air concentrations across the larger US Census block groups ultimately supported modeling exposure with a mixture of US Census blocks and block groups (i.e., a number of block groups had relatively little intra-unit spatial variability in air concentrations, making it unnecessary to differentiate them into blocks for purposes of exposure modeling).
4.3.1.2 Secondary Pb Smelter Case Study

The study area for the secondary Pb smelter was defined out to the distance where modeled air concentrations for the secondary Pb smelter facility reached approximately 50 percent of regional background for Pb in ambient air (see Section 4.2.1 of the Risk Assessment Report). This resulted in a study area extending out to 10 km from the facility.

Block groups were included in the study area if (a) they were located completely within the study area or (b) (for block groups only partially contained within the study area) the majority of their air concentration would come from the study area. All block groups within the secondary Pb smelter study area were modeled at the more spatially-refined block-level. This was possible because the smaller overall number of blocks involved with this case study (435), which allowed this more refined-level of spatial modeling.

4.3.1.3 Near Roadway (Urban) Case Study

The GIS-based spatial template developed for the near roadway (urban) case study is based on research conducted into the spatial gradients of diesel PM (and other air pollutants) in the vicinity of roadways in Houston, TX (Risk Assessment Report, Section 4.3.2.1). Literature searches conducted in support of the current review did not identify any research specifically focused on spatial gradients associated with re-entrained dust near roadways and consequently, this research focusing on diesel PM was used as a surrogate. This does introduce additional uncertainty into this case study, since the spatial gradient for re-entrained roadside dust may differ from that of auto-emitted diesel PM.

The research examining spatial gradients for diesel PM included the derivation of "enhancements ratios" based on detailed dispersion modeling results (see Section 4.3.2.1.3). These enhancement ratios were developed for a 0-75 meter (m) and 75-200 m band extending out from the modeled roadway and reflect a decay in air contamination with distance away from the roadway. These same bands formed the basis for GIS-based templates used in this case study (as augmented reflecting available soil data - see below).

The inner 0-75 m band was further transected into 0-12 m and 12-75 m bands reflecting the fact that surrogate data for near roadway soil Pb (obtained in Corpus Christi, TX - see Section 4.3.2.2) included measurements at 12 m from the road, thereby allowing what appears to be a relatively steep initial decrease in near roadway soil Pb levels to be captured for purposes of exposure modeling. Note, also that consideration for remote sensing imagery for the case study location selected in Houston suggests that residential yards may exist immediately adjacent to the road (i.e., supporting this initial 12 m wide band as a potential play area for children). Consequently, the GIS-based spatial template ultimately used for this case study included six
exposure bands paralleling the modeled road segment including 0-12 m, 12-75 m, and 75-200 m segments on either side of the road.

Child counts for each band were generated by overlaying U.S. Census block-level data and using area-weighted apportionment to calculate the fraction of each intersected block falling within each of the six bands.

4.3.2 Methods for Estimating Media Concentrations

As described in Section 4.2.5.2, media concentrations, once defined, are held constant for the full exposure period simulated with the blood Pb modeling. For ambient air, ambient soil and indoor dust, estimates of annual average concentration were used for this purpose for all three case study locations. Additionally, from the ambient air concentrations, annual average inhalation exposure concentrations were estimated with consideration for daily activity patterns by children and differences in outdoor (ambient) versus indoor air Pb levels (see Section 4.3.2.2). To estimate media Pb concentrations for the three case study locations, the staff used a combination of empirical data and fate and transport modeling, reflecting the different availability of Pb measurements for three locations (Table 4-2). At all three locations, concentrations were estimated for current conditions and for the primary Pb smelter case study, concentrations were additionally estimated for a NAAQS attainment air quality scenario. Monitors associated with the other two case study locations have not had recent exceedances of the Pb NAAQS and consequently the current conditions and current NAAQS attainment air quality scenarios are identical for these two locations.
**Table 4-2. Case study approaches for estimating media concentrations.**

<table>
<thead>
<tr>
<th>Modeling Step</th>
<th>Primary Smelter</th>
<th>Secondary Smelter</th>
<th>Near Roadway (Urban)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GIS-based spatial template</td>
<td>Combination of U.S. Census blocks and block groups out to a 10 km radius around the facility</td>
<td>U.S. Census blocks out to a 10 km radius around the facility</td>
<td>Bands extending 0-12m, 12-75m, and 75-200m on either side of the modeled road segment</td>
</tr>
<tr>
<td>Ambient air concentrations</td>
<td>Dispersion modeling</td>
<td>Dispersion modeling</td>
<td>Monitor data (DRI near roadway monitors) combined with spatial gradient data for PM near roadways (from literature)</td>
</tr>
<tr>
<td>Performance evaluation</td>
<td>Comparison to TSP monitor data from study area</td>
<td></td>
<td>NA (empirical data already used in estimating outdoor air concentrations)</td>
</tr>
<tr>
<td>Inhalation exposure concentrations</td>
<td></td>
<td></td>
<td>Estimates for all three case studies are based on ambient air concentrations and reflect the application of location-specific adjustment factors that account for (a) the time spent by children at different locations (and at various activity levels) and (b) differences between indoor and outdoor ambient air Pb levels</td>
</tr>
<tr>
<td>Outdoor soil concentrations</td>
<td>Sampling data in remediation zone (near facility) with regression model used to extrapolate to outer portions of study area</td>
<td>Multiple Pathways of Exposure (MPE) model used to predict soil levels based on deposition (a hybrid approach which included consideration of both modeling and empirical data was included as part of the sensitivity analysis)</td>
<td>Surrogate soil data obtained for another near roadway location used to derive representative values for this study area</td>
</tr>
<tr>
<td>Performance evaluation</td>
<td>NA (empirical data already used in characterizing soil levels)</td>
<td>Modeled estimates compared to surrogate data for other industrial (point source) locations</td>
<td>NA (estimates based on surrogate data)</td>
</tr>
<tr>
<td>Indoor dust concentrations</td>
<td>Combination of (a) site-specific regression model (based on air) for remediation zone near facility and (b) pooled analysis regression model (based on air plus soil) for remainder of study area</td>
<td>Pooled regression model (both an air-only and an air + soil dust model was used, with the latter included as part of a sensitivity analysis for this case study)</td>
<td>Pooled regression model (based on air and soil)</td>
</tr>
<tr>
<td>Performance evaluation</td>
<td>Site-specific sampling data used in deriving regression model for remediation zone (no relevant empirical data identified for outer portions of study area)</td>
<td>No relevant empirical data identified for use in conducting site-specific performance evaluation</td>
<td></td>
</tr>
</tbody>
</table>
4.3.2.1 Ambient Air Concentrations

Different methods were used for estimating annual average ambient air concentrations at the three case study locations. For the primary and secondary Pb smelter locations, air dispersion modeling of Pb emissions was performed, while a combination of Pb measurements and published information on particle dispersion near roadways was used at the near roadway (urban) location.

4.3.2.1.1 Primary Pb Smelter Case Study

Outdoor air concentration and deposition rates for Pb were modeled using the Industrial Source Complex – Plume Rise Model Enhancements (ISC-PRIME) dispersion model. Source characterization and emissions data used in the pilot were obtained from EPA Region VII and reflect the current State Implementation Plan (SIP) developed for the facility (Missouri Department of Natural Resources (MDNR), 2000). A total of 266 tons/year were modeled for the facility based on this SIP, reflecting (a) processes at the facility (e.g., stack emissions), (b) fugitive emissions from transferring materials, (c) fugitive emissions from storage at the slag pile, and (d) emissions associated with roadway dust. Particle size distribution information was included for each source, reflecting consideration for the types of controls in place for specific processes. EPA Region VII is currently in the process of reviewing source characterization data to enhance modeling conducted as part of SIP planning and consequently, the dispersion model runs completed for the pilot analysis should be considered illustrative only (modeling to be completed for the full-scale analysis will reflect the latest source characterization completed as part of the SIP revision due in 2007). Performance evaluation was conducted by comparing modeled annual averaged Pb air concentration estimates for receptor points located at existing TSP monitors within the study area to the measured values at those monitoring locations (see Section 4.3.6.1).

For the current conditions scenario, the annual average Pb concentration for each air modeling receptor point was derived from hourly estimates produced from the model. Additionally, for the purposes of developing the current NAAQS attainment scenario, hourly estimates were used to generate quarterly average concentrations. Any quarterly average concentrations that were greater than 1.5 μg/m³ were replaced with 1.5, and an annual average air concentration for each air modeling receptor point was derived from these adjusted quarterly averages.

4.3.2.1.2 Secondary Pb Smelter Case Study

Outdoor air concentration and deposition rates for Pb were modeled using the AERMOD dispersion model. AERMOD is the current preferred general purpose dispersion model for assessing criteria pollutants under the Clean Air Act (70FR No. 216. p. 68218). Source
characterization and emissions data were the same as those used in a Residual Risk Assessment conducted for the facility (EC/R, 2006), with emissions estimates derived from stack tests performed in December, 1997, November, 1999 and February 2000. A total of 4.56 tons/year were modeled, reflecting processes at the facility (e.g., stack emissions) and fugitive dust emissions from materials storage and handling and roadway dust.

As with the primary Pb smelter case study, annual average Pb concentrations for the secondary Pb smelter (for the current conditions scenario) were derived from hourly estimates produced for each air modeling receptor point by the dispersion model. As noted earlier, the current conditions and current NAAQS attainment air quality scenarios are identical for this case study.

### 4.3.2.1.3 Near Roadway (Urban) Case Study

Outdoor air concentrations for Pb were characterized using PM$_{10}$ monitoring data collected from the DRI monitoring site located within the study area (ICF, 2005). Sampling was conducted over three days in early 2001 and the average of the values over these three days was used as the basis for the ambient air estimate. The DRI monitoring site is located 115m from the road segment forming the basis for this case study and the enhancement ratios were applied accordingly to generate an ambient air concentration value for the 0-12, 12-75 and 75-200m bands along each side of the road. Note, that because a single enhancement ratio was developed for the 0-75m band, that same ratio was used for both the 0-12 and 12-75m bands (it is likely that a higher entrained signal would be found in the band immediately adjacent to the road relative to the second 12-75m band, but available data do not support quantifying this distinction).

### 4.3.2.2 Inhalation Exposure Concentrations

Inhalation exposure concentrations for Pb were estimated for the population of interest (young children) from the estimated ambient air concentrations using age-group- and location-specific relationships for Pb developed using the exposure modeling component of EPA’s 1999 NATA national-scale assessment (USEPA 2006c). These relationships account for air concentration differences indoors and outdoors and mobility or time spent in different locations (e.g., outdoors at home, inside at home etc.) for the population of interest.

The exposure modeling component of the NATA national-scale assessment generated detailed exposure profiles (i.e., inhalation exposure concentrations) for sets of modeled children for each U.S. Census tract (USEPA, 2006c). We used the ratio of these NATA-based inhalation exposure concentrations to the NATA national-scale assessment’s corresponding estimates of ambient air Pb concentration (i.e., matched by U.S. Census tract) to develop adjustment factors that could be used to scale our estimates of ambient air Pb concentration in pilot assessment study areas to derive inhalation exposure concentrations (see Risk Assessment Report Section...
4.1.2.2 for additional detail on this procedure). The adjustment factors (or ratios) for the 0 to 4 age group (the closest age group for which outputs are available to the age group of interest for this assessment) between NATA national-scale assessment Pb exposure concentrations and ambient air concentrations ranged from 0.37 to 0.42 for the Census tracts within the three case study areas. Use of these ratios for the 0 to 4 year old age group to represent the 0 to 7 year old age group modeled in the pilot contributes some uncertainty in the estimate of inhalation exposure concentrations.

4.3.2.3 Outdoor Soil Concentrations

Pb concentrations in outdoor soil where characterized for the three case studies using a combination of modeling and empirical data. For the primary Pb smelter, empirical data were used to estimate concentrations in the remediation zone closer to the facility, while statistical extrapolation based on measurement data was used for the remainder of the study area. For the secondary Pb smelter case study, fate and transport modeling was used to predict soil concentrations. In addition, as mentioned earlier, a second hybrid – model plus empirical - approach was used to estimate soil concentrations based on measurement data from a surrogate location. Soil concentrations for the near roadway (urban) case study are based on a combination of empirical data obtained from a surrogate location and a spatial gradient decay function obtained from the literature.

4.3.2.3.1 Primary Pb Smelter Case Study

Over the past 10 years, many of the residential yards closer to the primary Pb smelter have undergone remediation involving the removal of contaminated soil and replacement using "clean" soil. Extensive soil sampling has been conducted to support this remediation effort including the collection of pre- and post-remediation Pb measurements. In addition, soil measurements have been collected for locations outside of the remediation zone, which extends out to about 1.5 km from the facility.

Characterization of soil Pb concentrations for this case study uses a combination of measurement data (for blocks within the remediation zone) and statistically-based predictions beyond the remediation zone. Soil levels within the remediation zone are based on the most recent post-remediation measurements available for a given block (i.e., pre-remediation soil levels are not used in estimating soil levels within the remediation zone). This reflects the fact that extensive remediation has occurred within the remediation zone and therefore, the latest measurements from remediated yards are assumed representative of current conditions. Note, that studies have shown recontamination of remediated yards (USEPA, 2006e) and consequently, the remediation zone should be viewed as a dynamic zone with changing soil Pb levels.
Characterization of soil levels for blocks and block groups beyond the remediation zone are based on a regression model predicting soil Pb as a function of distance from the facility, which was fitted to pre-remediation soil measurement data (available closer to the facility) (Risk Assessment Report Section 4.1.3). The use of pre-remediation soil data in deriving the regression equation reflects the fact that little remediation has occurred in these more distant locations and consequently, spatial trends seen in the pre-remediation soil levels are more likely to be representative for these outer portions of the study area. The regression model used in these estimates has an R² of 0.92 which suggests a good fit and increases overall confidence in these statistical estimates. However, it should be noted that this increased confidence holds for areas of interpolation (i.e., areas with sampling data used to fit the model – out to about 2.3 km from the facility) more than for areas of extrapolation (areas without sampling data – beyond 2.3 km from the facility).

Because sampling data were used either in establishing soil concentrations (within the remediation zone) or in fitting the regression model (beyond the remediation zone), no performance evaluation was conducted for this step in the analysis. Note, however, that future efforts may consider performance evaluation based on such approaches as split-set validation (i.e., fitting the regression model with half of the measurement data and evaluating the performance of that model using the other half of the data).

4.3.2.3.2 Secondary Pb Smelter Case Study

As noted in Section 4.2.3.2, there are no soil sampling data for this case study, necessitating the use of either (a) surrogate soil data (from a similar type of facility and study area) or (b) fate and transport modeling to predict soil Pb levels. Surrogate soil data sufficient to provide coverage for the entire study area were not identified and consequently, fate and transport modeling was employed. Specifically, outdoor soil concentrations were calculated at each block analyzed for the secondary Pb smelter case study using the AERMOD deposition estimates and EPA’s Multiple Pathways of Exposure (MPE) methodology (USEPA, 1998). The MPE methodology represents the update of the Indirect Exposure Methodology, or IEM (USEPA 1990b; USEPA 1993). MPE consists of a set of multimedia fate and transport algorithms developed by EPA’s Office of Research and Development, including a soil mixing model which was used in this assessment to calculate the soil concentrations resulting from deposition at the Troy site. The Human Health Risk Assessment Protocol (HHRAP), which utilizes the same soil mixing algorithm, includes a database of input parameters which was used to parameterize the equation for this assessment (USEPA, 2006d).

In the MPE/HHRAP algorithms, cumulative soil concentration is calculated as a function of particle deposition, soil mixing depth, bulk density, and a soil loss constant. The soil loss
constant (in this case) was set up to be a function of loss due to leaching, erosion, and runoff. Concentration in the soil was calculated assuming constant deposition of Pb for the entire operating period of the facility (37 years).

A background soil Pb concentration of 15 mg/kg (based on Gustavsson et al., 2001) was added to all modeled concentrations to produce a "total" Pb soil estimate. In this context, "background" refers to Pb in soil resulting from sources other than this particular secondary Pb smelter. Note, in presenting both exposure and risk results for this case study, the background soil Pb concentration described here was treated as part of background source exposure with the other contribution to soil Pb (from the facility itself) contributing to policy-relevant exposures.

Modeled soil concentrations for the secondary Pb smelter were compared to empirical data obtained from a surrogate location (see Table 4-11). Based on this comparison, which suggested that modeled soil Pb concentrations for this case study might be significantly underestimated, we included a second characterization of soil concentrations besides the purely modeled approach described above. Specifically, measurement data from a surrogate secondary Pb smelter location were used to “scale” up the modeled surface generated for this case study location to more closely match the empirical data obtained from the surrogate location (at specified distances from the facility). This second hybrid approach to estimate soil concentrations was included to address uncertainty in estimating soil Pb concentrations for this case study (see Tables 4-10 and 4-18, respectively, for additional details on the comparison of modeled results for this case study against the empirical data from the surrogate location and the hybrid approach that resulted from that comparison).

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12 Note, this background value of 15 mg/kg represents natural soil Pb concentrations and general anthropogenic activity. An argument could be made for considering a background value that might more closely reflect an urbanized scenario with greater coverage for auto-emitted lead and other sources (e.g., Pb paint). We will consider this issue of background in relation to the secondary Pb smelter as we refine the risk assessment for the full scale analysis (see Section 4.5.2).

13 Explicit consideration for background soil Pb levels for the secondary Pb smelter case study reflect the fact that soil Pb levels for this case study area modeled. Consequently, they need to include (a) contributions from the facility of interest (captured in fate and transport modeling described here) and (b) background (non-facility related) Pb in soil (covered by the background value of 15 mg/kg discussed here). Because the other two case studies considered in the pilot use empirical data to characterize soil Pb levels, the contribution of background soil Pb to total soil Pb is implicitly reflected in the measured values and there is no need to add an additional background value as is done for the secondary Pb smelter.
4.3.2.3.3 Near Roadway (Urban) Case Study

Soil measurement data were not available for the DRI monitoring location in Houston and consequently, surrogate near roadway soil data were identified and used (together with an empirically-based decay function from the literature) to characterize potential soil Pb gradients for the six bands evaluated in the analysis.

Near roadway soil data were identified for a number of locations around the country (see Risk Assessment Report, Sections 3.3.4 for a discussion of these data). Ultimately, sampling data from Corpus Christi, TX (Turner and Maynard, 2003) were selected for use as surrogates for this case study, primarily because of their proximity and geographic similarity to the case study location. Turner and Maynard collected samples near the entrance ramp to Interstate 37, which has higher traffic flow compared with the road segment in Houston used for this case study. The Corpus Christi data included measurements at 2, 3, and 12 m from the road with values of 731, 766 and 214 mg/kg, respectively (all samples were taken between 0 and 5 centimeters from the surface).

The relatively sharp decrease in soil Pb levels with distance from the road reflected in these data (i.e., the drop from 700+ mg/kg adjacent to the road to 214 mg/kg at 12 m), is supported by other studies. Southerland and Tolosa (2000) reported that there is a linear relationship between the log of soil Pb concentrations and the log of distance from the road, suggesting a sharply decreasing soil concentration gradient. Similarly, Filipelli et al., (2005) and Hafen and Brinkmann (1996) have reported an exponential decay in soil Pb concentrations with increasing distance from the roadway. In addition, review of available near roadway data suggests that concentrations drop off to near urban background within a distance of about 50 m from the road. A reasonable background urban soil Pb level (given available data in the literature) is 100 mg/kg. This reflects a number of studies (Lejano and Ericson, 2005, and Chinereje et al., 2004) (see Risk Assessment Report, Section 4.3.3 for additional discussion).

The data described above were used to develop representative (surrogate) soil Pb levels for each of the three bands used in this case study. Specifically, for the 0-12 m band, an assumption of a log-log (linear) relationship between soil Pb and distance was assumed and a consequently, a log-log model was fitted using two data points from the Corpus Christi data (766 mg/kg at 3 m and the 214 mg/kg at 12 m). The mean value predicted across this distance using the fitted regression line (388 mg/kg) was used for the 0-12 m band. For the 12-75 m band, an assumption of a linear decay (between the 214 mg/kg datapoint at 12 m and a background concentration of 100 μg/kg at 75 m) was used. This reflects and assumption that the exponential decay in soil Pb levels has been largely realized by the 12 m distance, with decay becoming linear at that point. This produced an average value of 157 mg/kg for the 12-75 m zone. And
finally, urban background (100 mg/kg) was used for the 75-200 m band (see Risk Assessment Report Section 4.3.3 for additional details on the derivation of these soil concentrations).

4.3.2.4 Indoor Dust Concentrations

Pb in indoor dust can originate from a variety of sources including (a) outdoor soil which is tracked into the house, (b) Pb in outdoor soil which is entrained and subsequently transported indoors (c) Pb released directly into outdoor air through ongoing anthropogenic activity (e.g., industrial point emissions) which is transported indoors and (d) interior sources of Pb (e.g., paint, hobbies) (Adgate et al., 1998, Von Lindern, 2003). In the exposure assessment conducted for the last review, indoor dust Pb concentrations were predicted based on Pb concentrations in outdoor soil and ambient air (USEPA, 1989). This is also the case for the default approach in the exposure component of the IEUBK model (USEPA, 1994).

The importance of outdoor soil relative to outdoor air in influencing indoor dust Pb levels appears to depend on the nature of the Pb sources involved. Investigations in urban areas and contaminated waste sites with elevated soil Pb levels without active air point source emissions of Pb have indicated a greater dependency of dust Pb concentrations on soil Pb concentrations than on ambient air concentrations (e.g., Abgate, 1998 and Von Lindern, 2003). By contrast, investigations in areas with current point sources of Pb (e.g., active Pb smelters) have implicated ambient air Pb as an important source of Pb to indoor dust (Hilts, 2003). Contributions of ambient air Pb to indoor dust Pb levels have also been illustrated by a deposition study conducted in New York City (Caravanos et al., 2006). Caravanos and others described Pb deposition indoors resulting primarily from exterior environmental sources and not from interior Pb sources.

In light of these differences between areas with and without active Pb point sources, we have relied on different air, soil and dust Pb relationships for estimating Pb levels in indoor dust at the three case study locations.

4.3.2.4.1 Primary Pb Smelter Case Study

We used different regression models for predicting Pb concentrations in indoor dust in areas where soil has been remediated (see description in Section 4.2.3.1 for details on soil remediation) and where it has not. For the remediation zone near the facility, a regression equation was developed using dust Pb measurement data which had been collected from some of the houses within this area. (these data, while sufficient for supporting development of a site-specific regression model, did not have sufficient coverage to be used alone to represent indoor dust Pb levels for that portion of the study area). For the remainder of the study area, we employed the regression equation developed for the last review. We decided not to use the site-specific dust Pb model for the entire study area because the soil Pb concentrations in the

December 2006 4-33 Draft – Do Not Quote or Cite
remediation zone are significantly different from those in the remainder of the study area as a result of the remediation activity. The dataset used to develop the model for the remediation zone was based on indoor dust samples collected in 17 houses within the remediation zone. Independent variables included in the analysis were: (a) estimated annual average Pb concentrations in air at census block centroids located within 200 meters of each of the 17 houses, (b) road dust Pb measurements for locations within 300 meters of each house and (c) post-remediation residential soil Pb measurements for the yard of each house. Pre-remediation soil Pb concentrations were not included in the regression analysis since they were not expected to represent current conditions at the site. Multiple samples for each medium associated with a specific house within the dataset (e.g., reflecting multiple samples collected over time) were averaged to produce a "temporally-averaged" value. A number of regression models were evaluated, (see Risk Assessment Report, Section 4.1.4), and the H6 model was ultimately selected based on goodness of fit and other considerations. This model relates the natural log of indoor house dust to the natural log of ambient air Pb ($r^2=0.701$):

\[
\ln(\text{house dust, mg/kg or ppm}) = 8.3884 + 0.73639*\ln(\text{air Pb, } \mu g/m^3)
\]

Several points regarding the other variables considered for the remediation zone regression are noted here. For example, road dust Pb concentration was not found to have significant predictive power for indoor dust Pb. This may reflect the fact that the road dust Pb dataset does not provide significant coverage for homes located near to the truck haul routes. Additionally, yard soil Pb concentration was found to be slightly, and statistically significantly, negatively correlated with indoor dust Pb levels. This counter-intuitive finding may be a result of the existence within the remediation zone of a patchwork of remediated yards, such that the remediation activity may have interfered with any correlation between yard soil Pb levels, ambient air Pb levels and indoor dust Pb levels that might have existed previously. The resulting slight negative correlation of dust Pb levels with soil Pb levels led us to exclude soil Pb from the model. The y-intercept for the selected model may reflect a number of factors not correlated with ambient air or distance from the facility, such as a general level of soil Pb contamination in the area and indoor Pb paint.

For areas beyond the remediation zone, a regression equation developed during the last review from data collected during the 1970s and 1980s at a number of operational primary Pb smelters, including the smelter at Herculaneum (i.e., this case study location) was used (USEPA, 1989, Appendix B). This model (referred to as the "AGG" or "aggregate" model) predicts indoor dust Pb concentration from both outdoor soil and ambient air Pb concentrations. We have

December 2006 4-34 Draft – Do Not Quote or Cite
selected the AGG model for the non-remediation portion of the primary Pb smelter case study area since this area has not been subjected to extensive remediation and is therefore likely to resemble the locations included in the pooled dataset used in deriving this model. The AGG model, selected for areas beyond the remediation zone is the following:

\[
\text{GG pooled analysis model (air+soil version):} \\
\text{House dust (mg/kg or ppm) = 31.3 + 638*air Pb (μg/m}^3) + 0.364*\text{soil Pb (mg/kg)}
\]

### 4.3.2.4.2 Secondary Pb Smelter Case Study

A version of the same "AGG" model (USEPA, 1989) used for the primary Pb smelter was also used for the secondary Pb smelter case study. However, in the case of the secondary Pb smelter, an "air-only" version of the model (USEPA, 1989) was employed reflecting the reduced overall confidence associated with soil characterization at this case study (as noted above, soil concentrations at the secondary Pb smelter case study were modeled, while empirical data were available for characterizing soil at the primary Pb smelter). The "AGG" model for estimating indoor dust (USEPA, 1989) was derived in two forms including an air-only model that based indoor dust concentrations on outdoor ambient air Pb (without explicitly considering outdoor soil Pb levels) and an air+soil model which based estimates on both outdoor soil and ambient air Pb data. It is important to note, however, that the air-only model does reflect (implicitly) some consideration for the \textit{air-to-soil-to-indoor dust} mechanism in the air signal. Specifically, the larger air factor for the air-only model (relative to the air+plus dust model's air factor) reflects contribution of air Pb both directly to dust through penetration indoors and subsequent deposition to surfaces and indirectly to dust through deposition to outdoor soil which impacts indoor dust. (USEPA, 1989).\(^{14}\)

\[
\text{AGG pooled analysis model (air-only version):} \\
\text{House dust (mg/kg or ppm) = 60 + 844*air Pb (μg/m}^3)
\]

\(^{14}\) Note, that for the sensitivity analysis run focusing on the characterization of soil Pb concentrations at this case study, the alternate AGG soil+air dust model was used, rather than the AGG air-only dust model used in the run described above. The decision to use the AGG soil+air model for the sensitivity analysis reflects the desire to make sure that the sensitivity analysis considered the full impact of higher soil Pb concentrations around the facility, including their impact on indoor dust Pb levels (use of the AGG air-only model, would have meant that the increased soil Pb concentrations considered in this sensitivity analysis run would have only impacted exposure through soil ingestion and not through their impact on indoor dust).
The AGG model used for the secondary smelter was based on a number of studies focusing mainly on primary Pb smelters (a number of primary Pb smelters were operational at the time of model development). This does introduce additional uncertainty into indoor dust predictions generated for the secondary Pb smelter using this model since factors related to indoor dust loading (particle size profiles and nature of the entrained Pb compounds) might differ for primary versus secondary Pb smelters resulting in differing degrees of indoor dust loading from stack-emitted Pb.

**4.3.2.4.3 Near Roadway (Urban) Case Study**

The same version of the "AGG" model (soil+air regression model) (USEPA, 1989) used for the primary Pb smelter was also used for the near roadway (urban) case study.

AGG pooled analysis model (air+soil version):

$$\text{House dust (mg/kg or ppm)} = 31.3 + 638*\text{air Pb (μg/m}^3) + 0.364*\text{soil Pb (mg/kg)}$$

A number of considerations went into the decision to use a soil+air version of the AGG regression model for the near roadway (urban) case study. First, measurement data for a surrogate near roadway location is used to characterize soil Pb levels for bands within the study area which increases the overall confidence in soil characterization relative to the use of fate and transport modeling. Having increased confidence in the soil Pb levels supports use of a model that explicitly considers soil in predicting indoor dust (i.e., includes a soil factor in estimating indoor dust Pb). Second, long-term historical loading of near roadway soils has produced relatively elevated levels of soil Pb (certainly within the 50m zone adjacent to the road) which might contribute significantly to indoor dust levels. Even if entrainment is relatively low, resulting in a smaller contribution of soil Pb to indoor dust loading through this mechanism, other mechanisms (tracking of soil indoors) could provide a means for soil to impact indoor dust. Use of an AGG model which explicitly considers soil in predicting indoor dust will allow these mechanism (related to soil loading dust) to be considered as part of exposure and risk analysis. It is important to note, however, that the soil+air AGG model was developed primarily based on data collected near primary Pb smelters. Therefore, its use in predicting indoor dust levels for houses near roadways (in areas with little current industrial Pb emissions) does introduce uncertainty into the analysis.

**4.3.3 Methods for Estimating Blood Pb Levels**

This section presents the methodology used to estimate blood Pb levels in the child study populations. The section begins with an overview of the two biokinetic models used in this analysis (IEUBK and Leggett). Input parameters used in running both models are then described,
with emphasis on those parameters expected to either introduce significant uncertainty into
modeled blood Pb levels and/or those parameters which required more complex methods to
develop input values for. The probabilistic approach used to generate population-level
distributions of blood Pb levels for each study population is then described. The section ends
with a discussion of the GSD used to reflect inter-individual variability in behavior related to Pb
exposure and Pb biokinetics (a key component in modeling population-level blood Pb).

4.3.3.1 Blood Pb Models

The modeling of blood Pb levels is required for the pilot analysis for a number of
reasons: (a) measured blood Pb levels are only available for a small fraction of the study
population associated with the primary Pb smelter and are not available for either of the other
two case studies (necessitating the need to model blood Pb levels), (b) exposure, characterized
using blood Pb, needs to be apportioned between policy-relevant and background Pb exposures,
which necessitates modeling capable of parsing blood Pb resulting from different exposure
pathways and (c) potential changes in existing blood Pb level distributions need to be predicted
given reductions in ambient air Pb levels. As discussed in Section 4.4.1 of the CD, there are two
broad categories of blood Pb models available to support exposure and risk assessment:

- **Statistical (regression) models**, which attempt to apportion variance in measured blood
  Pb levels for a study population to a range of determinants or control variables (e.g.,
surface dust Pb concentrations, air Pb concentrations). The development of these
  models requires paired predictor-outcome data which restricts these empirical models
to the domain of their observations (i.e., to applications involving the study
  population(s) and exposure scenarios used in their derivation or at least to scenarios
  very similar to the original study conditions) (Section 4.4.1, CD).

- **Mechanistic models**, which attempt to model the process of transfer of Pb from the
  environment to human tissues. While these models are considerably more complex
  compared with the regression models (in terms of both the number of variables and
  their computational structure), by incorporating variables that vary temporally and
  spatially, or across individuals or populations, mechanistic models can be extrapolated
to a wide range of scenarios, including those outside of the original populations and
  exposure scenarios used to develop/parameterize the models (Section 4.4.1, CD)

Given concerns over applying regression models to populations and exposure scenarios
other than those used in their derivation, we decided to place emphasis on mechanistic models in
conducting the exposure analysis for the pilot, given their greater flexibility in application. Note,
however, that regression models have been included as part of the sensitivity analysis addressing
uncertainty in blood Pb modeling - see Section 4.4.3.1.
The CD (Section 4.4.1) highlights three mechanistic (biokinetic) models developed over the past several decades including IEUBK for modeling child Pb exposure and two models for simulating Pb biokinetics from birth through adulthood (Leggett, 1993 and O'Flaherty, 1993, 1995, 1998). All three models have the potential for application in Pb risk assessment and have been evaluated to varying degrees using empirical datasets (CD, Section 8.3.4).

For the pilot analysis, we used the IEUBK and Leggett models to generate child blood Pb distributions for all three case studies. Inclusion of the Leggett and IEUBK models (together with the regression-based blood Pb model mentioned above) represents an effort to consider uncertainty in modeling blood Pb levels and is reflected in the sensitivity analysis completed for the pilot (see Section 4.4.3.1). A brief overview of the IEUBK and Leggett models is presented below (for discussion of the regression-based model used in the sensitivity analysis, see Section 4.3.3.1).

**4.3.3.1.1 IEUBK**

A multi-compartmental pharmacokinetics model for children 0-7 years of age, which predicts average quasi-steady state blood Pb concentrations corresponding to daily average exposures, averaged over periods of a year or more. The exposure submodel provides average daily intakes of Pb (averaged over a 1 year time increment) for inhalation (air, including consideration for both outdoor and indoor) and ingestion (soil, indoor dust, diet and water) (Section 4.4.5.1 of the CD). The model is intended to be applied to groups of children experiencing similar levels of Pb exposure and will generate a representative central tendency blood Pb estimate for that group. Consideration for inter-individual variability in biokinetics and behavior (e.g., varying rates of dietary Pb ingestion) is typically accomplished through the incorporation of a GSD which, together with the IEUBK-generated average blood Pb level, can be used to characterize the distribution of blood Pb levels for a group of modeled children. Additional detail on the IEUBK model can be found in Section 4.4.5 of the CD.

**4.3.3.1.2 Leggett**

Originally developed from a model designed to simulate radiation doses for bone-seeking radionuclides, this biokinetic model has a temporal resolution of one day and can model exposure from infancy through adulthood. Note, that the day-level resolution in Leggett does allow more comprehensive treatment of the temporal pattern of exposure and its shorter-term impact on blood Pb levels than IEUBK, although for this analysis, which focuses on longer-term trends in Pb exposure, this functionality is not that relevant. The model does not include a detailed pathway-level exposure submodel as does IEUBK, instead taking (as inputs) total ingestion and inhalation exposure. However, it is possible to link the Leggett model to a more detailed pathway-level exposure model, thereby allowing a more detailed treatment of Pb...
exposure pathways and their impact on blood Pb. The use of this type of external exposure model including pathway-specific modeling of exposure levels was implemented for the pilot. As with IEUBK, Leggett is used to derive central tendency blood Pb levels for groups of similarly exposed children. The same GSD used for IEUBK is then used to produce estimates of the distribution of blood Pb levels within study populations. For additional details on the Leggett model see Section 4.4.6 of the CD.

As noted above, both models (IEUBK and Leggett) were used in the pilot analysis essentially in unmodified form except for inclusion of an external exposure model for Leggett as mentioned above. Note, however, that a number of the input parameters for both models have been adjusted to reflect the latest data on behavior, biokinetics and Pb exposure (key input parameters for both models are discussed in the next section) (see Risk Assessment Report Section 5.1 for addition detail on blood Pb modeling completed for the pilot).

4.3.3.2 Model Inputs

Both the IEUBK and Leggett models require the specification of a range of input parameters addressing such factors as inhalation rates, inhalation exposure concentrations, dietary consumption rates, incidental ingestion rates for soil and dust and route-specific absorption factors. In addition, as noted above, characterization of blood Pb levels using both models include the application of a GSD reflecting inter-individual variability in both exposure levels and biokinetic factors.

This section highlights a subset of the factors used in biokinetic blood Pb modeling for the pilot, focusing on those factors (a) whose derivation involved a relatively complex analytical process, thereby warranting discussion and/or (b) are subject to potentially significant uncertainty resulting in their inclusion in the sensitivity analysis.

4.3.3.2.1 IEUBK Input Parameters

Exposure modeling completed for the three case study locations has generally identified background exposure (diet and drinking water), incidental soil ingestion and incidental dust ingestion as the pathways contributing most to total blood Pb in children (see Section 4.4.2 for detailed pathway-specific results). Table 4-3 presents input parameters for IEUBK related to the modeling of these key pathways (including their values and their basis for derivation). Full documentation of input parameters used for IEUBK modeling in the pilot are presented in Section 5.1.4 of the Risk Assessment Report.
Table 4-3. IEUBK input parameters and basis or derivation.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Parameter value*</th>
<th>Basis/Derivation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>INGESTION - DRINKING WATER</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Water consumption (L/day)</td>
<td>0.34, 0.31, 0.31, 0.33, 0.39, 0.42</td>
<td>(USEPA, 2002a) Based on value for infants, 1-3 yr olds and value for 1-10 yr olds (with trend lines used to interpolate intermediate age ranges).</td>
</tr>
<tr>
<td>Water Pb concentration (µg/L)</td>
<td>4.61</td>
<td>Geometric mean of values reported in studies of U.S. and Canadian populations (residential water) (CD, Section 3.3 Table 3-10).</td>
</tr>
<tr>
<td>Absolute absorption (unitless)</td>
<td>0.5</td>
<td>Assumed similar to dietary absorption (see &quot;Total percent accessible&quot; under Ingestion-Diet below)</td>
</tr>
<tr>
<td><strong>INGESTION – DIET</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dietary Pb intake (µg/day)</td>
<td>3.16, 2.60, 2.87, 2.74, 2.61, 2.74, 2.99</td>
<td>Estimates based on (a) Pb food residue data from U.S. Food and Drug Administration Total Diet Study (USFDA, 2001), (b) food consumption data from NHANES III (USCDC, 1997), and (c) dietary consumption rates (defaults) used in the IEUBK model (USEPA, 1994). See Website for details on derivation and data used (Superfund recommendations - website: <a href="http://www.epa.gov/superfund/lead/ieubkfaq.htm/input">http://www.epa.gov/superfund/lead/ieubkfaq.htm/input</a>).</td>
</tr>
<tr>
<td>Total fraction accessible (unitless)</td>
<td>0.5</td>
<td>Alexander et al., 1973 and Ziegler et al., 1978, as cited in the CD (Section 4.2.1). These two dietary balance studies suggest that 40-50% of ingested Pb is absorbed by children (2wks to 8years of age).</td>
</tr>
<tr>
<td><strong>INCIDENTAL INGESTION - SOIL and DUST</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Soil/dust weighting factor (unitless)</td>
<td>45</td>
<td>This is the percent of total ingestion that is soil. Value reflects best judgment and consideration for Clausing (Clausing, et al., 1987, as cited in USEPA, 1989). The Clausing study looked at tracer studies of ingestion rates for rainy days and non-rainy days, and assumed rainy was all soil ingestion and non-rainy days was a combination of soil and dust with the delta representing soil.</td>
</tr>
<tr>
<td>Total dust + soil ingestion (mg/day)</td>
<td>85, 135, 135, 135, 100, 90, 85</td>
<td>USEPA 1989, based on multiple studies focusing on children</td>
</tr>
<tr>
<td>Total fraction accessible (soil and dust) (unitless)</td>
<td>- primary Pb smelter case study: 0.48 for soil and 0.26 for dust - secondary Pb smelter and near roadway: 0.30 (for both soil and dust)</td>
<td>- Site specific absorption factors for soil and indoor dust were derived for the primary smelter case study using relative bioavailability (RBA) estimates generated based on swine studies involving soil and dust samples collected in the study area (Casteel, 2005). These RBAs were converted to absolute bioavailability factors (i.e., total percent accessible values) by applying the absolute bioavailability factor for the control material (Pb acetate water solution also fed to the animals). - secondary Pb smelter and near roadway values: (USEPA, 1989) reflects evidence that Pb in dust and soil is as accessible as dietary Pb and that dust/soil ingestion may occur away from mealtimes (resulting in enhanced absorption relative to exposure during meal events).</td>
</tr>
<tr>
<td><strong>OTHER</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal blood Pb (µg/dL)</td>
<td>1.94</td>
<td>NHANES IV (national geometric mean for adult women - all nationalities) (Madeloni, 2005)</td>
</tr>
</tbody>
</table>

* When appropriate (i.e., when age-differentiation is required to capture variability and/or when sufficient data exist to support age differentiation), values are presented for 0-1, 1-2, 2-3, 3-4, 4-5, 5-6 and 6-7 year olds. Otherwise, when a single value is provided, it was not age-differentiated and was used for all age groups in IEUBK modeling.
4.3.3.2 Leggett Input Parameters

As noted earlier, the Leggett model does not include a detailed exposure module and instead accepts daily intake rates for the inhalation and ingestion routes. For the pilot analysis, the Leggett model has been linked to an external exposure module that allows us to model the contribution of specific pathways (e.g., dietary ingestion and indoor dust ingestion) to total ingestion and inhalation intake. Input parameters used both in the external exposure module and in the Leggett model proper, have been selected to match those used in the IEUBK model to the extent possible. Specifically, input parameters have been specified to insure as close a match as possible between the route-specific Pb uptake rates used in Leggett and those used in IEUBK. This reflects a desire that any differences in the performance between IEUBK and Leggett stem from fundamental differences in the way the two models treat the distribution and disposition of Pb within the body and not from differences in the Pb uptake rates provided to the two models. Because of the similarities in the input parameters used in IEUBK and Leggett, the parameters for Leggett will not be presented here. The reader is referred to Section 5.1.3.3 of the Risk Assessment Report for additional details on the input parameters used in the Leggett modeling.

4.3.3.3 Probabilistic Population Blood Pb Modeling Procedure

This section provides an overview of the probabilistic modeling used to generate distributions of blood Pb levels for children associated with each of the case study locations. As discussed in Section 3.3.1, recent epidemiological studies have identified the concurrent and lifetime average blood Pb metrics as most strongly correlated with IQ loss in children. Therefore, these two metrics have been used in generating IQ loss estimates for the pilot analysis and consequently, exposure modeling conducted for the pilot is designed to characterize the distribution of both concurrent and lifetime average blood Pb levels for study populations.

The goal of this probabilistic exposure modeling is to generate population-level distributions of blood Pb levels that allow (a) specific percentiles of exposure (e.g., 50th, 90th, 99th and mean) within a study population to be identified and (b) allow the total blood Pb levels associated with a given percentile to be further differentiated by exposure pathway (e.g., background versus policy-relevant with the latter further differentiated as to ambient air inhalation, indoor dust ingestion and outdoor soil ingestion). Therefore, for example, we might

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15 As noted earlier, the modeling approach used for the pilot does not allow exposures resulting from the ingestion of soil and indoor dust to be further differentiated reflecting the contribution from older historically deposited lead and lead that has been released to the air more recently. Consequently, policy-relevant exposures (including the ingestion of outdoor soil and indoor dust) reflect the combined impact of older historical lead emissions and more recent emissions.
have an estimate of exposure for the 99th percentile child at the primary Pb smelter, with that
developmental and policy-relevant pathways including ambient air, indoor dust, and outdoor
soil.

The probabilistic exposure modeling relied on information in three areas as summarized
below:

- **Central-tendency blood Pb levels for each exposure zone**: biokinetic blood Pb
  modeling described in the last section produces central-tendency blood Pb levels
  (concurrent and lifetime average) for each exposure zone in each case study area.
  These blood Pb levels represent the "average" blood Pb levels projected for children
  residing in each exposure zone. However, in reality, blood Pb levels are distributed
  across the children within each zone, with that distribution being based around this
  central-tendency estimate. The variability in blood Pb levels is addressed through the
  use of the GSD discussed in the last section (see third bullet below).

- **Demographics (child distribution within study areas)**: The distribution of 0-7 year old
  children within each case study area (represented as child counts within each exposure
  zone) is used to insure that the generation of population-level blood Pb level
  distributions for each case study reflects where children are located.

- **GSD reflecting inter-individual variability in blood Pb levels**: As discussed earlier, a
  GSD is used to reflect inter-individual variability for blood Pb levels in groups of
  similarly-exposed children. For the pilot, the GSD is combined with the central-
tendency blood Pb level estimates described in the first bullet to generate a distribution
  of blood Pb levels for the group of children located in each exposure zone. The same
  GSD (i.e., 1.6) is used for all three case studies (see section 4.3.3.4 for additional
  discussion of the GSD).

The step-wise procedure used to generate population-level blood Pb distributions for each
case study is illustrated in Figure 4-3 (Note, that the information described in the bullets above is
referenced in Figure 4-3 as input data used in population-level exposure modeling).

Several points related to the modeling approach presented in Figure 4-3 need to be
highlighted. For the pilot analysis, 10,000 simulated individuals were generated for each case
study location, in order to insure that the population-level blood Pb distributions generated met
target stability goals (see Risk Assessment Report, Section 5.2.2). For all three case studies,

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16 An analysis of stability in the probabilistic modeling used for the pilot, showed that simulation runs with
10,000 realizations achieved reasonable stability goals (i.e., average run-to-run variability of <10% for percentiles
up to the 99.5th, with the 99.9th having average run-to-run variability <15%) (see Risk assessment Report Section
5.2.2 for additional detail).
Figure 4-3. Procedure for Generating Population Blood Pb Distributions.

**Input distributions and datasets used in the Monte Carlo-based population-exposure modeling:**

- **Central-tendency blood Pb level dataset:** Blood Pb modeling (described in Section 4.3.3.1) generates a central-tendency blood Pb level for each exposure zone (e.g., block, or blockgroup) within each study area (results dimensioned on blood metric and blood Pb model).

- **Demographic dataset:** the child count (US Census 2000 children <7 yrs of age) for each exposure zone is used to support population-weighted sampling of exposure zones.

- **Adjustment factor distribution representing inter-individual variability in behavior and biokinetics related to Pb exposure:** The GSD of 1.6 has been centered on a median value of 1.0 to produce a distribution that can be used to obtain adjustment factors reflecting the behavior and biokinetics (related to Pb exposure) for a single simulated child.

**Monte Carlo-Based Population Blood Pb Level Modeling Procedure:**

1. **Step 1 – select a central-tendency blood Pb level for a specific exposure zone based on population-weighted random sampling:**
   The central-tendency blood Pb level dataset is combined with the demographic dataset to conduct population-weighted sampling of a central-tendency blood Pb level for a specific exposure zone (i.e., sampling proportional to child count within each exposure zone is used to select a specific zone and the central-tendency blood Pb level for that zone is chosen as the output form Step 1).

2. **Step 2 – select an adjustment factor reflecting inter-individual variability in behavior and biokinetics related to Pb exposure for a single simulated child:** A value is sampled randomly from the adjustment factor distribution described above. This single value represents the behavior and biokinetics (related to Pb exposure) for a single simulated child.

3. **Step 3 – generate a blood Pb level for a single simulated individual:** The central-tendency blood Pb level selected in Step 1 is multiplied by the adjustment factor in Step 2 to produce a blood Pb level for a single simulated child within the exposure zone selected in Step 1.

4. **Step 4 – place simulated child blood Pb level (output of Step 3) in pool of modeled blood Pb levels for that study area:** This pool of simulated child blood Pb levels represents the distribution of blood Pb levels across the study area and reflects (a) the demographic distribution of children across that study area (and their relation to Pb levels in contact media) and (b) inter-individual variability in behavior and biokinetics related to Pb exposure.

Repeat procedure 10,000 times to generate set of 10,000 simulated individuals (Note, this set can be weighted down to reflect the actual population of children (0-7 years) within each study area in generating population-count related risk metrics).
this simulation count obviously represents a higher total child count than actually is associated
with the study area. Using a higher number of simulated individuals was necessary to generate
blood Pb distributions with "stable" higher-end exposure estimates (if simulations matching the
actual population count at each case study had been conducted, the distributions that would have
resulted would have been "unstable" at higher percentiles). It is important to note, however, that
in presenting population counts associated with individual percentiles (e.g., the number of
children associated with a given population percentile), the population counts have been scaled
to reflect the actual child counts associated with the study areas.

A further point needs to be clarified regarding the differentiation of specific percentile
total blood Pb levels into pathway-specific fractions. All simulated individuals associated with a
given exposure zone, were assigned the same pathway-specific apportionment, reflecting
biokinetic modeling conducted for each zone. The simulation described in Figure 4-3 involves
generating a set of simulated individual for each exposure zone by considering (a) the central
tendency blood Pb level generated using biokinetic modeling for that zone and (b) the GSD of
1.6 reflecting blood Pb variability. While this approach will produce a set of simulated
individuals with a range of total blood Pb levels, it is assumed that all of them have the same
pathway-specific apportionment of those blood Pb levels (i.e., the same apportionment generated
for the central-tendency blood Pb level modeled for that exposure zone). In reality, it is likely
that pathway apportionment would vary across children with different blood Pb levels located in
the same exposure zone (e.g., the contribution of indoor dust exposure to total blood Pb would
differ for kids living near each other who demonstrate different total blood Pb levels). However,
the modeling approach used in the pilot does not account for this level of resolution in pathway
apportionment. Note, however, that pathway apportionment does differ across exposure zones
(i.e., each exposure zone has a different pattern of pathway apportionment for its simulated
children).

The modeling approach presented in Figure 4-3 and described above, generates
population-level distribution of total blood Pb levels with further pathway apportionment as
caveated above. These distributions can be used to generate several types of exposure metrics
including:

- **Population-weighted exposure percentiles**: total blood Pb levels (with pathway
  apportionment) for simulated individuals representing specific points along the
  population blood Pb distribution (e.g., 50, 90, 95, 99 and 99.5th percentile).
- **Incidence counts**: number of children within a given study area projected to experience
  a specific degree of Pb exposure (total blood Pb level).
4.3.3.4 GSD for Population Blood Pb Levels

Regarding the GSD used to reflect inter-individual variability in Pb exposure and biokinetics (i.e., the basis for the adjustment factor distribution described above and in Figure 4-3), a value of 1.6 has been used for the pilot analysis. This value reflects the distribution of blood Pb levels measured in children exposed to smelter emissions at the Midvale UT smelter (White et al., 1998). This value represents a reasonable central-tendency GSD for populations of children living in relatively small, defined areas where the sources and relative importance of different exposure pathways and media are similar across the exposed population. All three case study locations considered for the pilot include exposed child populations where the source of interest (i.e., primary Pb smelter, secondary Pb smelter, or near roadway deposited Pb) is expected to contribute significantly to overall exposure, relative to background (at least for areas closer to the policy-relevant source of interest). In this context, using a GSD reflecting the distribution of blood Pb levels for children residing near a primary Pb smelter (i.e., a dominant Pb source such as the Midvale UT smelter) seems appropriate in modeling the three case study locations. It is also worth noting that the pre-remediation blood Pb GSD for children participating in the Baltimore Urban Pb Soil Abatement Project was estimated at 1.5 (White et al., 1998), which adds support to using the GSD of 1.6 for the near roadway (urban) case study, which can be interpreted as being somewhat similar to a general urban scenario in terms of Pb exposure.

Recent surveys of blood Pb levels in children at the national-level (NHANES IV data for years 1999-2002), have found GSD values in the range of 2.03 to 2.23 (Hattis, 2005). These GSDs, which are considerably larger than the values used in the pilot, likely reflect the fact that, while blood Pb levels for the majority of children in the U.S. have decreased significantly over the last 1-2 decades, a small fraction of children still retains relatively elevated blood Pb levels due to continued exposure to Pb paint and other artifact sources. Consequently, as the median and mean blood Pb levels have dropped, the extreme upper tail of the distribution is still somewhat anchored by these high-exposure children, resulting in an increased GSD for the overall population. However, because the three case studies modeled for the pilot reflect exposure scenarios with dominant Pb sources (e.g., smelters or the more heavily contaminated near roadway bands), it is believed that a smaller GSD (i.e., 1.6) is more appropriate for these case studies (again, at least for portions of the study areas nearer to the sources of interest). Note, however, that alternate GSDs have been considered in the sensitivity analysis (see Section 4.4.3.1).
4.3.4 Projected Media Concentrations

This section presents summaries of the media concentrations generated by the methods described in Section 4.3.2 for the three case study locations. The complete set of media concentration estimates for each of the case studies is included in the Risk Assessment Report (Section 4.0). Table 4-4 presents summaries of projected annual average air concentration results along with inhalation exposure concentration results for the three case study locations. Table 4-5 and Table 4-6 summarize the projected outdoor Pb soil concentrations and indoor dust Pb concentrations, respectively, for the three study areas.

Several factors should be noted in reviewing these summarized results. Both the ambient air concentration and indoor dust concentration results are differentiated as to air quality scenario (i.e., "current conditions" and "current NAAQS attainment"), since these differ for the primary Pb smelter (note, that the indoor dust Pb concentrations, because they are based on air concentrations, as well as soil, will show a reduction under the "current NAAQS attainment" scenario). By contrast, summarized results for outdoor soil Pb are not differentiated by air quality scenario, since for the pilot, potential changes in outdoor soil associated with attaining the current NAAQS at the primary Pb smelter case study were not modeled.17

---

17 Given that the reduction in air concentration associated with the current NAAQS attainment scenario is small, little reduction in soil Pb concentration is anticipated.
Table 4-4. Projected ambient air and inhalation exposure concentrations. a

<table>
<thead>
<tr>
<th>Statistic b</th>
<th>Current Conditions</th>
<th>Current NAAQS Attainment b,c</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average annual Pb air concentration (μg/m³)</td>
<td>Inhalation exposure concentrations (μg/m³)</td>
</tr>
<tr>
<td>Primary Pb Smelter</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximum</td>
<td>2.73</td>
<td>1.14</td>
</tr>
<tr>
<td>95th percentile</td>
<td>0.662</td>
<td>0.277</td>
</tr>
<tr>
<td>Median</td>
<td>0.0221</td>
<td>0.00895</td>
</tr>
<tr>
<td>5th percentile</td>
<td>0.00845</td>
<td>0.00329</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.00541</td>
<td>0.00210</td>
</tr>
<tr>
<td>Secondary Pb Smelter</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximum</td>
<td>0.536</td>
<td>0.238</td>
</tr>
<tr>
<td>95th percentile</td>
<td>0.0178</td>
<td>0.008</td>
</tr>
<tr>
<td>Median</td>
<td>0.0047</td>
<td>0.0021</td>
</tr>
<tr>
<td>5th percentile</td>
<td>0.0009</td>
<td>0.0004</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.0005</td>
<td>0.0002</td>
</tr>
<tr>
<td>Near roadway (Urban)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximum</td>
<td>0.008</td>
<td>0.0033</td>
</tr>
<tr>
<td>95th percentile</td>
<td>0.008</td>
<td>0.0033</td>
</tr>
<tr>
<td>Median</td>
<td>0.005</td>
<td>0.0022</td>
</tr>
<tr>
<td>5th percentile</td>
<td>0.005</td>
<td>0.0022</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.005</td>
<td>0.0022</td>
</tr>
</tbody>
</table>

a The 223 blocks and block groups with non-zero population selected for analysis were used to create this summary. Note that in some of these blocks the 2000 U.S. Census indicates there are no children.

b The 5th and 95th percentile values for the current conditions and current NAAQS attainment scenario (for the primary Pb smelter case study) are identical because only two of 223 U.S. Census blocks within the study area had modeled outdoor air Pb levels exceeding the NAAQS. This means that differences in the two air quality scenarios are only evident at the extreme high-end of modeled media concentration distributions and associated exposure levels.

c Note that the “Average annual Pb air concentration” values presented here are the values used in modeling exposure for the pilot analysis. As discussed in Section 4.3.2.1, consideration for the current NAAQS attainment scenario required clipping of modeled quarterly average air concentrations values at the NAAQS level of 1.5 μg/m³, with subsequent recalculation of the annual-average air concentration values used in exposure modeling.

Table 4-5. Projected outdoor soil concentrations.

<table>
<thead>
<tr>
<th>Statistic</th>
<th>Projected average soil concentration (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Primary Pb smelter</td>
</tr>
<tr>
<td>Maximum</td>
<td>976</td>
</tr>
<tr>
<td>95th percentile</td>
<td>426</td>
</tr>
<tr>
<td>Median</td>
<td>84.0</td>
</tr>
<tr>
<td>5th percentile</td>
<td>23.6</td>
</tr>
<tr>
<td>Minimum</td>
<td>15.9</td>
</tr>
</tbody>
</table>

* Range reflects the two approaches used to characterize soil Pb levels for the secondary case study including a purely modeling approach and a hybrid (model-empirical) approach, in which the modeled surface is scaled up to match trends seen in soil Pb levels at a surrogate location (see Table 4-18).
Table 4-6. Projected indoor dust concentrations.

<table>
<thead>
<tr>
<th>Statistic</th>
<th>Projected average indoor dust concentration (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Primary Pb smelter</td>
</tr>
<tr>
<td></td>
<td>Current Conditions</td>
</tr>
<tr>
<td>Maximum</td>
<td>5,263</td>
</tr>
<tr>
<td>95th percentile</td>
<td>2,191</td>
</tr>
<tr>
<td>Median</td>
<td>47</td>
</tr>
<tr>
<td>5th percentile</td>
<td>37</td>
</tr>
<tr>
<td>Minimum</td>
<td>35</td>
</tr>
</tbody>
</table>

* Range reflects dust Pb levels estimated as part of the sensitivity analysis examining uncertainty in outdoor soil Pb prediction for this case study location (see Table 4-18). That sensitivity analysis involved two different approaches to characterizing soil Pb levels. These different soil Pb estimates for the study area translated into different indoor dust Pb estimates reflected in this table. Note also, that the sensitivity analysis used two different approaches for predicting indoor dust Pb (i.e., the AGG air-only approach and the AGG air+soil approach - see Section 4.3.2.4). The fact that the lower percentiles do not have a range reflects the fact that outdoor soil has a very small effect on indoor dust Pb at greater distances from the facility (with indoor dust Pb at those distances driving primarily by the intercept term in the dust Pb models - see Section 4.3.2.4).

4.3.5 Projected Blood Pb Levels

This section presents summaries of blood Pb modeling completed for the three case study locations in the form of population percentiles. These results are dimensioned as follows:

- **Air quality scenario:** The primary Pb smelter had projected exceedances of the Pb NAAQS, so projected air concentrations (and inhalation air concentrations) differ for the two air quality scenarios, resulting in different modeled blood Pb levels.

- **Characterizing Pb concentrations in soil:** Performance evaluation conducted for the secondary Pb smelter focusing on soil Pb suggested that modeled soil Pb concentrations for this case study might be under-estimated by a factor of 3. This resulted in the decision to include two scenarios for this case study in both the exposure and risk assessment including: (a) a model-only scenario where soil concentrations are modeled and (b) a hybrid (model/empirical) approach where modeled estimates for this study area are scaled up based on comparison to empirical soil Pb estimates obtained for a surrogate secondary Pb smelter (see Tables 4-11 and 4-18). Consequently, two sets of exposure estimates are presented for the secondary Pb smelter.

- **Blood Pb model:** Both the IEUBK and Leggett models were used to project blood Pb levels for the case studies.

- **Blood Pb metric:** Both concurrent and lifetime average blood Pb levels were modeled for the three case study locations.

- **Pathway apportionment:** Each of the simulated individuals has had their projected total blood Pb levels apportioned between contributing pathways. For the summary tables presented in this section, pathway apportionment information is presented at the more...
generalized level of (a) policy-relevant background (drinking water and diet) versus (b) policy-relevant exposures (air-inhalation, soil and dust ingestion). For more detailed pathway-specific breakdown of exposure results, refer to the risk results tables presented in Section 4.4.2.

Given the number of dimensions involved, results have been separated by case study, with Table 4-7 summarizing results for the primary Pb smelter, Table 4-8 the secondary Pb smelter and Table 4-9 the near roadway (urban) case study.

A number of observations can be made by reviewing the blood Pb results presented in Tables 4-7 through 4-9. Generally, the IEUBK model generates higher blood Pb levels than the Leggett model (as seen when comparing population percentile blood Pb estimates across the two models). Furthermore, the concurrent blood Pb levels are typically lower than the lifetime average blood Pb levels; this is expected given that the lifetime average values reflect contributions from earlier years of exposure when Pb exposure is typically higher, while the concurrent estimates represent modeled blood Pb at 7 years of age. When combined, these two factors mean that the highest blood Pb levels are typically seen for the combination of the IEUBK model and the lifetime average blood Pb metric, while the lowest levels are seen with the combination of the Leggett model and the concurrent blood Pb metric.

Speaking specifically to exposure estimates generated for individual case studies, modeled blood Pb levels for the primary Pb smelter range from 0.2 to 28.6μg/dL (1st percentile to the 99.9th percentile simulated individual) for the current conditions exposure scenario and from 0.2 to 24.9μg/dL for the current NAAQS attainment scenario. Central tendency blood Pb levels for this case study range from 0.7 to 1.9μg/dL (50th percentile for both the current conditions and current NAAQS attainment scenarios). The results for this case study demonstrate a clear trend regarding pathway apportionment, with higher blood Pb levels reflecting a larger contribution from policy-relevant sources relative to policy-relevant background. This is expected, since simulated individual with higher blood Pb levels are generally located closer to the facility where Pb concentrations in exposure media are higher, resulting in a higher proportion of overall Pb exposure coming from facility-related Pb.

Speaking now to results generated for the secondary Pb smelter case study, as noted earlier, two sets of exposure estimates are presented for this case study, one reflecting a model-only approach to estimating soil Pb concentrations and the other reflecting a hybrid (model+empirical) approach. Projected blood Pb estimates for this case study range from 0.2 to 6.3μg/dL (1st percentile to the 99.9th percentile simulated individual for the model-only approach and the hybrid approach). Unlike the primary Pb smelter case study, pathway apportionment is fairly constant across all of the population percentiles (i.e., there is no clear trend with higher
percentiles being dominated by policy-relevant sources). This likely reflects the fact (discussed below in Section 4.4.3) that the highest impact areas near to the facility do not have any children (according to US Census data for 2000) and therefore, these portions of the study area that would have shown the highest gradients in exposure are not reflected in the population-weighted blood Pb distributions. Consequently, the blood Pb distribution for this case study is dominated by simulated individuals with fairly consistent patterns of exposure, in terms of pathway-apportionment (with policy-relevant sources generally contributing about 25-40% of total Pb exposure).

The near roadway (urban) case study has modeled blood Pb levels ranging from 0.3 to 9.1 μg/dL (1st percentile to the 99.9th percentile simulated individual). As with the secondary Pb smelter, pathway apportionment estimates for this case study are fairly constant across the population percentiles and show that typically, 60-65% of total Pb exposure comes from policy-relevant sources. The absence of a clear trend in pathway apportionment likely reflects the fact that this case study was modeled with a relatively small number of bands (3) extending out from the modeled road segment which significantly reduces the opportunity for refined gradients in exposure (i.e., exposure will be "clustered" into three subpopulations reflecting individuals located within each of the three bands extending out from the road segment).
### Table 4-7. Projected blood Pb levels (μg/dL) for primary Pb smelter case study.

<table>
<thead>
<tr>
<th>Statistic</th>
<th>IEUBK (concurrent)</th>
<th>IEUBK (lifetime average)</th>
<th>Leggett (concurrent)</th>
<th>Leggett (lifetime average)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blood Pb level</td>
<td>% from Policy-Relevant Pathways*</td>
<td>Blood Pb level</td>
<td>% from Policy-Relevant Pathways*</td>
</tr>
<tr>
<td><strong>Current conditions exposure scenario</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99.9th</td>
<td>21.9</td>
<td>98%</td>
<td>28.6</td>
<td>98%</td>
</tr>
<tr>
<td>99.5th</td>
<td>12.4</td>
<td>96%</td>
<td>16.9</td>
<td>95%</td>
</tr>
<tr>
<td>95th</td>
<td>7.4</td>
<td>96%</td>
<td>10.6</td>
<td>95%</td>
</tr>
<tr>
<td>90th</td>
<td>3.7</td>
<td>78%</td>
<td>5.3</td>
<td>74%</td>
</tr>
<tr>
<td>Median</td>
<td>2.9</td>
<td>66%</td>
<td>4.1</td>
<td>71%</td>
</tr>
<tr>
<td>75th</td>
<td>2.0</td>
<td>55%</td>
<td>2.7</td>
<td>38%</td>
</tr>
<tr>
<td>25th</td>
<td>0.9</td>
<td>44%</td>
<td>1.2</td>
<td>44%</td>
</tr>
<tr>
<td>1st</td>
<td>0.4</td>
<td>46%</td>
<td>0.5</td>
<td>46%</td>
</tr>
<tr>
<td><strong>Current NAAQS attainment exposure scenario</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99.9th</td>
<td>18.4</td>
<td>97%</td>
<td>24.9</td>
<td>96%</td>
</tr>
<tr>
<td>99.5th</td>
<td>11.2</td>
<td>96%</td>
<td>15.5</td>
<td>95%</td>
</tr>
<tr>
<td>95th</td>
<td>7.9</td>
<td>87%</td>
<td>11.3</td>
<td>95%</td>
</tr>
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<td>90th</td>
<td>3.7</td>
<td>72%</td>
<td>5.4</td>
<td>91%</td>
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<td>60%</td>
<td>4.1</td>
<td>46%</td>
</tr>
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<td>71%</td>
<td>1.9</td>
<td>48%</td>
</tr>
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<td>0.9</td>
<td>55%</td>
<td>1.2</td>
<td>44%</td>
</tr>
<tr>
<td>1st</td>
<td>0.4</td>
<td>38%</td>
<td>0.5</td>
<td>56%</td>
</tr>
</tbody>
</table>

* Policy-relevant pathways include inhalation, soil-ingestion and indoor dust ingestion (and exclude background sources e.g., diet, drinking water).
### Table 4-8. Projected blood Pb levels (μg/dL) for secondary Pb smelter case study.

<table>
<thead>
<tr>
<th>Statistic</th>
<th>IEUBK (concurrent)</th>
<th>% from Policy- Relevant Pathways*</th>
<th>IEUBK (lifetime average)</th>
<th>% from Policy-Relevant Pathways*</th>
<th>Leggett (concurrent)</th>
<th>% from Policy-Relevant Pathways*</th>
<th>Leggett (lifetime average)</th>
<th>% from Policy-Relevant Pathways*</th>
</tr>
</thead>
<tbody>
<tr>
<td>99.9th</td>
<td>3.7</td>
<td>30%</td>
<td>4.7</td>
<td>22%</td>
<td>2.1</td>
<td>26%</td>
<td>3.0</td>
<td>22%</td>
</tr>
<tr>
<td>99.5th</td>
<td>3.0</td>
<td>30%</td>
<td>3.9</td>
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<td>1.6</td>
<td>30%</td>
<td>2.3</td>
<td>24%</td>
</tr>
<tr>
<td>99th</td>
<td>2.7</td>
<td>22%</td>
<td>3.5</td>
<td>27%</td>
<td>1.4</td>
<td>22%</td>
<td>1.9</td>
<td>26%</td>
</tr>
<tr>
<td>95th</td>
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<td>31%</td>
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<td>0.9</td>
<td>25%</td>
<td>1.2</td>
<td>23%</td>
</tr>
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<td>24%</td>
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<tr>
<td>Median</td>
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<td>26%</td>
<td>1.1</td>
<td>22%</td>
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<td>29%</td>
<td>0.7</td>
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<td>25th</td>
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<td>26%</td>
<td>0.5</td>
<td>22%</td>
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<td>24%</td>
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<td>26%</td>
<td>0.2</td>
<td>24%</td>
<td>0.2</td>
<td>23%</td>
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</tbody>
</table>

Model only approach for characterizing soil concentrations

<table>
<thead>
<tr>
<th>Statistic</th>
<th>Blood Pb level</th>
<th>% from Policy-Relevant Pathways*</th>
<th>Blood Pb level</th>
<th>% from Policy-Relevant Pathways*</th>
<th>Blood Pb level</th>
<th>% from Policy-Relevant Pathways*</th>
<th>Blood Pb level</th>
<th>% from Policy-Relevant Pathways*</th>
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</thead>
<tbody>
<tr>
<td>99.9th</td>
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<td>2.3</td>
<td>38%</td>
<td>3.3</td>
<td>37%</td>
</tr>
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<td>99.5th</td>
<td>3.7</td>
<td>38%</td>
<td>4.9</td>
<td>40%</td>
<td>1.9</td>
<td>39%</td>
<td>2.7</td>
<td>38%</td>
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<td>40%</td>
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<td>36%</td>
<td>2.7</td>
<td>37%</td>
<td>1.0</td>
<td>38%</td>
<td>1.5</td>
<td>39%</td>
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<td>75th</td>
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<td>38%</td>
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<td>41%</td>
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<td>38%</td>
<td>1.1</td>
<td>38%</td>
</tr>
<tr>
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<td>37%</td>
<td>1.5</td>
<td>38%</td>
<td>0.6</td>
<td>40%</td>
<td>0.8</td>
<td>39%</td>
</tr>
<tr>
<td>25th</td>
<td>0.8</td>
<td>39%</td>
<td>1.1</td>
<td>40%</td>
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<td>36%</td>
<td>0.6</td>
<td>37%</td>
</tr>
<tr>
<td>1st</td>
<td>0.4</td>
<td>36%</td>
<td>0.5</td>
<td>36%</td>
<td>0.2</td>
<td>38%</td>
<td>0.3</td>
<td>40%</td>
</tr>
</tbody>
</table>

Hybrid approach (model + surrogate data) for characterizing soil concentrations

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<th>Statistic</th>
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<th>% from Policy-Relevant Pathways*</th>
<th>Blood Pb level</th>
<th>% from Policy-Relevant Pathways*</th>
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<th>Blood Pb level</th>
<th>% from Policy-Relevant Pathways*</th>
</tr>
</thead>
<tbody>
<tr>
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<td>9.1</td>
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<td>4.2</td>
<td>56%</td>
<td>6.9</td>
<td>80%</td>
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<td>7.0</td>
<td>56%</td>
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<td>56%</td>
<td>4.9</td>
<td>56%</td>
</tr>
<tr>
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<td>4.4</td>
<td>56%</td>
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<td>56%</td>
<td>2.7</td>
<td>56%</td>
<td>4.1</td>
<td>56%</td>
</tr>
<tr>
<td>95th</td>
<td>3.1</td>
<td>56%</td>
<td>4.4</td>
<td>56%</td>
<td>1.9</td>
<td>56%</td>
<td>2.9</td>
<td>56%</td>
</tr>
<tr>
<td>90th</td>
<td>2.6</td>
<td>56%</td>
<td>3.6</td>
<td>56%</td>
<td>1.6</td>
<td>56%</td>
<td>2.4</td>
<td>65%</td>
</tr>
<tr>
<td>75th</td>
<td>1.9</td>
<td>56%</td>
<td>2.7</td>
<td>56%</td>
<td>1.2</td>
<td>56%</td>
<td>1.7</td>
<td>56%</td>
</tr>
<tr>
<td>Median</td>
<td>1.4</td>
<td>65%</td>
<td>1.9</td>
<td>65%</td>
<td>0.8</td>
<td>56%</td>
<td>1.2</td>
<td>65%</td>
</tr>
<tr>
<td>25th</td>
<td>1.0</td>
<td>65%</td>
<td>1.4</td>
<td>65%</td>
<td>0.6</td>
<td>56%</td>
<td>0.9</td>
<td>56%</td>
</tr>
<tr>
<td>1st</td>
<td>0.4</td>
<td>56%</td>
<td>0.6</td>
<td>56%</td>
<td>0.3</td>
<td>56%</td>
<td>0.4</td>
<td>56%</td>
</tr>
</tbody>
</table>

* Policy-relevant pathways include inhalation, soil-ingestion and indoor dust ingestion (and exclude background sources e.g., diet, drinking water). Note, that for this case study, background sources also included a fraction of soil Pb identified as background (see Section 4.3.2.3.2).

### Table 4-9. Projected blood Pb levels (μg/dL) for near roadway (urban) case study.

<table>
<thead>
<tr>
<th>Statistic</th>
<th>IEUBK (concurrent)</th>
<th>% from Policy-Relevant Pathways*</th>
<th>IEUBK (lifetime average)</th>
<th>% from Policy-Relevant Pathways*</th>
<th>Leggett (concurrent)</th>
<th>% from Policy-Relevant Pathways*</th>
<th>Leggett (lifetime average)</th>
<th>% from Policy-Relevant Pathways*</th>
</tr>
</thead>
<tbody>
<tr>
<td>99.9th</td>
<td>6.5</td>
<td>56%</td>
<td>9.1</td>
<td>56%</td>
<td>4.2</td>
<td>56%</td>
<td>6.9</td>
<td>80%</td>
</tr>
<tr>
<td>99.5th</td>
<td>5.0</td>
<td>56%</td>
<td>7.0</td>
<td>56%</td>
<td>3.1</td>
<td>56%</td>
<td>4.9</td>
<td>56%</td>
</tr>
<tr>
<td>99th</td>
<td>4.4</td>
<td>56%</td>
<td>6.2</td>
<td>56%</td>
<td>2.7</td>
<td>56%</td>
<td>4.1</td>
<td>56%</td>
</tr>
<tr>
<td>95th</td>
<td>3.1</td>
<td>56%</td>
<td>4.4</td>
<td>56%</td>
<td>1.9</td>
<td>56%</td>
<td>2.9</td>
<td>56%</td>
</tr>
<tr>
<td>90th</td>
<td>2.6</td>
<td>56%</td>
<td>3.6</td>
<td>56%</td>
<td>1.6</td>
<td>56%</td>
<td>2.4</td>
<td>65%</td>
</tr>
<tr>
<td>75th</td>
<td>1.9</td>
<td>56%</td>
<td>2.7</td>
<td>56%</td>
<td>1.2</td>
<td>56%</td>
<td>1.7</td>
<td>56%</td>
</tr>
<tr>
<td>Median</td>
<td>1.4</td>
<td>65%</td>
<td>1.9</td>
<td>65%</td>
<td>0.8</td>
<td>56%</td>
<td>1.2</td>
<td>65%</td>
</tr>
<tr>
<td>25th</td>
<td>1.0</td>
<td>65%</td>
<td>1.4</td>
<td>65%</td>
<td>0.6</td>
<td>56%</td>
<td>0.9</td>
<td>56%</td>
</tr>
<tr>
<td>1st</td>
<td>0.4</td>
<td>56%</td>
<td>0.6</td>
<td>56%</td>
<td>0.3</td>
<td>56%</td>
<td>0.4</td>
<td>56%</td>
</tr>
</tbody>
</table>

* Policy-relevant pathways include inhalation, soil-ingestion and indoor dust ingestion (and exclude background sources e.g., diet, drinking water).
4.3.6 Performance Evaluation

This section describes performance evaluation completed in support of the pilot analysis (i.e., the comparison of modeled results to empirical data for purposes of assessing the representativeness of a particular modeling step). Performance evaluation for the exposure assessment focused on projections of Pb in ambient air and outdoor soil (discussed in Section 4.3.2.1 and 4.3.2.3, respectively) and projections of Pb in blood (covered in Section 4.3.3). Those case studies for which media concentrations were estimated using empirical data as the basis, were not subjected to performance evaluation; only those estimates based directly on modeling were included.

Performance evaluation can provide insights into the degree of representativeness associated with specific elements of exposure modeling by identifying systematic trends in either over- or underestimation of modeled results relative to empirical data.

4.3.6.1 Media Concentrations

Table 4-10 describes the performance evaluation completed for modeled ambient air concentrations and presents a summary of the results of that assessment for each of the case studies.
Table 4-10. Performance evaluation of approaches for ambient air concentrations.

<table>
<thead>
<tr>
<th>Case study</th>
<th>Description of performance evaluation</th>
<th>Results of the performance evaluation</th>
<th>Implications for overall analysis</th>
</tr>
</thead>
</table>
| Primary Pb smelter    | Comparison of 2 yr-averaged modeled air concentrations with annual averaged Pb measurements (from 2001 through 2005) from TSP monitors located within study area. | - Closest 2 monitors to the facility (~300m) are under-predicted by modeling in 2001, but over-predicted by modeling (factor of 1.3 to 1.6). Overall trend is over-prediction by modeling for these monitors.  
  - Remaining 7 TSP monitors (800m to 3km from facility) are under-predicted by modeling for all five years (0.05 to 0.6). | Low/moderate overestimation for points close to facility (for years 2002-2005) may suggest similar degree of overestimation for ambient air concentrations used in inhalation modeling and indoor dust prediction. Similarly, underestimations for points further from facility (but still only out to ~3km) may suggest moderate underestimation of inhalation exposure. Note, performance evaluation for this case study is jeopardized somewhat by the fact that meteorological data used in air modeling is from 1997-1999, while monitored data used in performance evaluation are from later years (2001-2005). |
| Secondary Pb smelter  | Comparison of 2 yr-averaged modeled air concentrations with annual averaged Pb concentrations from TSP monitors (1999 and 2000) located within study area. | 2 monitors (400m and 650m from the facility) are located in study area. Modeled results (geographically matched to monitors) are approximately three times lower than the monitored values. Note, however that the monitors are not downwind from the facility and that the highest modeled values (not matched to the monitors) are in the range of the monitored values. | Although the results suggested a significant underestimation of actual measured ambient air Pb levels (based on comparison at the monitors), because monitored values fall within the range of the highest modeled values predicted across the study area (and because the monitors are not located downwind from the facility), concern over a significant underestimation is reduced somewhat (see Risk Assessment Report, Section 4.2.2.4 for additional discuss). Note, that the age of the monitor data used in this performance evaluation (1997-2000) reduces the overall utility of the evaluation. |
| Near Roadway (Urban) | Performance evaluation was not completed for the near roadway study area since the characterization of ambient air Pb levels for this case study is based on empirical data (augmented with modeling characterizing spatial gradients in air pollution near roadways). |                                                                                                           |                                                                                                     |

Table 4-11 presents the results of the performance evaluation completed for modeled outdoor soil concentrations. Note, that because the secondary Pb smelter is the only case study to have its soil concentrations generated directly using modeling (the other two case studies relying on direct, or surrogate empirical data), only the secondary smelter was subjected to performance evaluation of its outdoor soil modeling.
Table 4-11. Performance evaluation of approaches for outdoor soil concentrations.

<table>
<thead>
<tr>
<th>Case study</th>
<th>Description of performance evaluation</th>
<th>Results of the performance evaluation</th>
<th>Implications for overall analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary Pb smelter</td>
<td>Performance evaluation was not completed for the primary Pb smelter study area since soil Pb concentrations were based directly on measured data (for the remediation zone closest to the facility) and on statistically-extrapolated values for the remainder of the study area.</td>
<td>Modeled soil concentrations are approximately three times lower than measured soil concentrations suggesting the potential for a significant underestimation of soil Pb levels at this case study.</td>
<td>The potential for a significant underestimation of soil Pb levels translates into a potentially significant impact (downward bias) on exposure and risk results. This finding led to an investigation of this issue of soil modeling at the secondary Pb smelter as part of the sensitivity analysis. Specifically, we developed an alternate soil concentration coverage for the study area by combining the modeled results (used to characterize relative spatial variability in soil levels across the study area) with surrogate data from Kimbrough (to adjust the absolute soil Pb levels for the study area). (see Table 4-16).</td>
</tr>
<tr>
<td>Secondary Pb smelter</td>
<td>Modeled results for this case study were compared to soil concentration data collected near a secondary Pb smelter located in an urban area where there is the potential for Pb impacts from multiple sources. Note, Small et al., (1995) also presents data collected near a secondary Pb smelter in Pennsylvania, but that study area was subjected to significant remediation of residential areas near the source and consequently, measurements may be significantly biased down.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Near Roadway (Urban)</td>
<td>Performance evaluation was not completed for the near roadway study area since the characterization of ambient air Pb levels for this case study is based on empirical data (augmented with modeling characterizing spatial gradients in air pollution near roadways).</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

4.3.6.2 Blood Pb Levels

Performance evaluation for the blood Pb modeling involved comparing modeled blood Pb levels for children (0-7 years of age) generated for the three case study locations against two empirical data sets: (a) national-level central tendency blood Pb levels for children (0-5 years) obtained through the NHANES IV survey (completed for all three locations) (CD, Table 4-1) and (b) site-specific monitored blood Pb levels (available only for Herculaneum). Table 4-12 presents the results of performance evaluation completed for blood Pb modeling.
Table 4-12. Performance evaluation of approaches for blood Pb levels.

<table>
<thead>
<tr>
<th>Case study</th>
<th>Description of performance evaluation</th>
<th>Results of the performance evaluation</th>
<th>Implications for overall analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Case Study Populations (Part 1)</td>
<td>Comparison of modeled median blood Pb levels for the three case study populations against GM values obtained from NHANES IV. The purpose of this evaluation is to determine whether there appears to be a significant error in the characterization of central tendency blood Pb levels (e.g., whether modeled central tendency levels are shown to be significantly lower than corresponding national-levels, which would suggest potential under-estimate in modeling since we would expect central tendency levels within the study area to be equal to or higher than national central-tendency levels, given the presence of the primary Pb smelter and its impact on exposure).</td>
<td>Modeled median lifetime blood Pb modeled with IEUBK ranged from 1.2 to 1.9 μg/dL (average 1.7); Median concurrent blood Pb modeled with IEUBK ranged from 0.9 to 1.4 μg/dL (average 1.2). Leggett median lifetime blood Pb ranged from 0.8 to 1.2 μg/dL (average 1.0); Median concurrent blood Pb ranged from 0.5 to 0.8 μg/dL (mean 0.7). These compare with a GM value from NHANES IV (for 0-5 yr olds) of 1.7 (2001-2002) (CD, Table 4-1).</td>
<td>The relatively close match between the modeled median lifetime levels from IEUBK and the national GM value from NHANES IV (for 0-5 yr olds) suggests that the IEUBK model with the given set of exposure, intake, and uptake factors is neither significantly over- or underestimating exposures for the study population. Because “concurrent” blood Pb in this analysis is defined as the average blood Pb from age 6 to 7 years, when blood Pb levels are known to decline from values seen in younger children, the median concurrent IEUBK values appear to also generally consistent with population data. The median blood Pb estimates are insensitive to the relatively small number of high-exposure block groups in the primary and secondary Pb smelter case studies; thus the lower exposure experienced by the large majority of the exposed populations are dominating this metric. The Leggett model, however, with the selected exposure, intake, and uptake factors, appears to be underestimating the GM blood Pb statistics somewhat, compared to the national population. The reason for this is not clear, although it is possible that the Pb exposure levels of the NHANES population are actually lower than the combined background and air-related levels used in the case studies.</td>
</tr>
<tr>
<td>Case study</td>
<td>Description of performance evaluation</td>
<td>Results of the performance evaluation</td>
<td>Implications for overall analysis</td>
</tr>
<tr>
<td>----------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| Primary Pb smelter (Part 2)| Comparison of upper-bound (extreme high-end) range of modeled blood Pb levels against the set of site-specific measured blood Pb levels collected for children <6 years of age in 2002 (U.S.DHHS, 2003). The purpose of this comparison is to compare the high-end of the modeled blood Pb level distribution to the range of empirical values obtained for this segment of more highly exposed children living close to the facility. Ideally, the extreme high-end of the modeled distribution (perhaps >99th percentile) should be similar to the high-end of the sampled population (perhaps >90th percentile), reflecting the fact that the measured data cover highly-exposed children close to the facility, while the modeled population includes children further from the facility who are less exposed. | 58 site-specific blood Pb level measurements from 2001 yield the following percentiles:  
> 95th percentile is 20-29 μg/dL  
> 90th percentile is 10-19 μg/dL  
> 50th percentile is 0-9 μg/dL  
Here are percentile results generated from our site-specific modeling:  
> 99.9th percentile is 13-29 μg/dL  
> 99.5th percentile is 6-17 μg/dL  
> 99th percentile is 4-11 μg/dL  
> 95th percentile approaches 5 μg/dL | These results show that the 58 screened blood Pb levels from the DHHS study correspond to the extreme high-end of our modeled distribution. This provides support for our modeled results generating reasonable high-end estimates. It should be noted that the top 5% of the screened children have higher blood Pb levels (20-29 μg/dL) than the top 5% of our modeled children (>5 μg/dL), but this is expected since the screening analysis focuses on children located relatively close to the facility, while the children we modeled include children out to 10km from the facility (inclusion of children further out will dilute the overall blood Pb distribution with children that live further from the facility and are less exposed). |
4.4 HEALTH RISK ASSESSMENT

This section describes the approach used to characterize risk for the pilot assessment, including discussion of the modeling approach (4.4.1) and presentation of results (4.4.2). This section also includes the results of the sensitivity analysis (4.4.3).

4.4.1 Method for Risk Characterization

Risk characterization for the pilot analysis focuses on modeling IQ loss in children using a log-linear concentration-response function obtained from a pooled analysis of epidemiology studies (Lanphear et al., 2005). This concentration-response function is combined with the population-level blood Pb distributions generated for each case study (see Section 4.3.3 above) to produce a distribution of IQ loss estimates for each study population. It is also possible to apportion IQ loss between different exposure pathways using the pathway-apportionment information generated as part of the exposure analysis (see Section 4.3.3.3 above).

Three key elements of the risk methodology used for the pilot are described in greater detail below, including: (a) the IQ-loss concentration response function used in the analysis, (b) the cut-points or policy-thresholds (representing specific exposure levels below which IQ loss will not be estimated) and (c) the step-wise analytical procedure used to generate the IQ loss (risk) distributions.

4.4.1.1 Concentration Response Function

As discussed in Section 3.3.1.2, log-linear concentration response functions for IQ loss (for the concurrent and lifetime average blood Pb metrics) obtained from a large pooled study (Lanphear et al., 2005) were used in this analysis. Specifically, these functions were used to estimate IQ decrements associated with a specific increment of blood Pb exposure above cutpoints established for the pilot analysis (see 4.4.1.2 below). The specific functions used in the pilot include (see Risk Assessment Report Section 6.1.2 for additional detail on the concentration response functions used and the application of the cutpoint):

Concurrent blood Pb metric log-linear IQ loss model:
IQ loss = -2.7 * ln (concurrent blood Pb/concurrent cutpoint)

Lifetime averaged Pb metric log-linear IQ loss model:
IQ loss = -3.04 * ln (lifetime average blood Pb/lifetime average cutpoint)
4.4.1.2 Derivation of Cutpoint

For the purposes of this analysis, we identified a blood Pb level below which risks would not be projected. In this context, the term "cutpoint" will be used for the lower-bound blood Pb level, below which IQ loss will not be estimated. Specifically, we chose the lower 5th percentile of blood Pb measurements from Lanphear et al. (2005) as the cutpoint. This reflects our recognition of the small sample size below this blood Pb level and the associated decreasing confidence in characterization of the concentration-response function in this blood Pb range (see Section 3.3.1.2). This lower 5th percentile of the sample blood Pb levels in the study, translates into two separate cutpoints for the two concentration-response functions: 2.4 μg/dL for the prediction of IQ loss using concurrent blood Pb and 6.1 μg/dL for predictions using the lifetime average metric. Because the cutpoints used in the pilot analysis are not based on an established biological threshold, these cutpoints are considered policy- or hypothetical thresholds.

4.4.1.3 Projection of Population Risk

Risk characterization completed for the pilot essentially involves converting the population-level blood Pb distributions into population-level distributions of IQ loss, given consideration for the cutpoints discussed in the last subsection. Specifically, each of the 10,000 simulated blood Pb levels generated for a given case study, is compared against the cutpoint. If the simulated total blood Pb level is above the cutpoint, then an IQ loss estimate is generated using the appropriate log-linear concentration response function described in Section 4.4.1.1 (i.e., using either concurrent or lifetime average based on the blood Pb metric). If the simulated blood Pb level is less than the cutpoint, then an IQ loss estimate is not generated. Note, that the application of this cutpoint approach results in a large fraction of the simulated individuals modeled for each case study, not being assigned an IQ loss estimate because their Pb exposure results in a projected blood Pb level less than the relevant cutpoint.

The pathway-apportioned IQ loss estimates generated using this approach are pooled to form a population-level distribution of IQ loss for a given study area. The point raised in Section 4.3.3.3 regarding pathway apportionment at the exposure zone-level (i.e., all simulated

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18 Note, however, that as discussed in Section 3.3.1.2 of this document and in the CD, a threshold blood Pb level for neurocognitive effects, including childhood IQ, has not been established and effects have been associated with the lowest Pb levels investigated (CD, Sections 6.2, 8.5.1 and 8.6.2). Consequently, this threshold reflects concerns over being able to characterize the nature of the concentration-response function for IQ loss and does not reflect evidence of a true biological threshold.
individuals from a given zone having the same pathway apportionment) holds for the risk estimates as well.

Just as with the population-level exposure estimates discussed in Section 4.3.3.3, risk estimates generated using the approach outlined here can be used to generate several types of risk metrics including:

- **Population-weighted risk (IQ loss) percentiles**: Total IQ loss (with pathway apportionment) for simulated individuals representing specific points along the population risk distribution (e.g., 50, 90, 95, 99 and 99.5th percentile simulated individuals).

- **Incidence counts**: Number of children within a given study area projected to experience a specific degree of risk (i.e., total IQ loss).

### 4.4.2 Risk Estimates

In this section we present risk results (IQ loss) generated for each of the three case studies, presented as population percentiles, as well as the numbers of children associated with each percentile (i.e., “Pop” in Tables 4-13 through 4-17). For example, a Pop value of 5 for the 99th percentile risk estimate indicates a projection of 5 children with IQ loss at or above the 99th percentile for that study area. In addition to presenting results reflecting the aggregate Pb exposure from all pathways, the fraction of the aggregate values associated with policy-relevant background versus policy-relevant exposure pathways are also presented, with the latter category further differentiated among three specific pathways (i.e., inhalation of ambient Pb, incidental ingestion of outdoor soil and incidental ingestion of indoor dust).

Each of the risk results tables is also dimensioned on blood Pb model and blood Pb metric, with separates sets of results in each table being presented for permutations of these two modeling options including: (a) IEUBK (concurrent blood Pb level metric), (b) IEUBK (lifetime average blood Pb level metric), (c) Leggett (concurrent blood Pb level metric) and (d) Leggett (lifetime average blood Pb level metric). Inclusion of these four parallel sets of risk results reflects the fact that both issues - blood Pb modeling and the blood Pb metric used in estimating IQ loss - represent potentially important sources of uncertainty in the pilot analysis. The decision to include full sets of risk results dimensioned on these two key issues reflects the fact that no clear "favored" approach (for either blood Pb model, or blood Pb metric) has been identified and therefore, all four permutations of these modeling elements are given equal weight in presenting risk results. Additional dimension are also included for two of the three case studies, as described below (e.g., inclusion of results for two air quality scenarios for the primary Pb smelter case study).
4.4.2.1 Primary Pb Smelter Case Study

Risk results for the primary Pb smelter are further dimensioned on air quality scenario, with results for both the current conditions and current NAAQS attainment scenarios being presented. Results for the primary Pb smelter range from 0 IQ points lost to 6 IQ points lost (1st percentile to the 99.9th percentile simulated individual) for the current conditions air quality scenario (see Table 4-13). It is important to note that IQ point losses are only projected for between 1 and 10% of the modeled population at this case study (39 to 388 children), depending on the concentration-response function used (and associated cutpoint), with the remainder having projected blood Pb levels below the cutpoints used in the analysis. Risk results for the current NAAQS scenario are similar to those for the current conditions scenario, with IQ loss estimates ranging from 0 points to 6 points (1st percentile to the maximum simulated individual) (see Table 4-14).

As with the exposure estimates discussed in Section 4.3.5, risk results for this case study exhibit a trend in terms of pathway apportionment, with higher risk estimates reflecting a higher proportion of total Pb exposure coming from policy-relevant sources. For example, the current conditions scenario (IEUBK + concurrent blood Pb metric) (Table 4-13) has a 99.9th percentile IQ loss of 6 points, with approximately 98% of the Pb exposure coming from policy-relevant sources and the vast majority of that originating from the incidental ingestion of indoor dust containing Pb. It is worth noting that incidental indoor dust ingestion dominates policy-relevant exposures for higher risk percentiles, but that incidental soil ingestion becomes increasingly important (in terms of policy-relevant exposure) as risk and exposure decreases. This is expected, since indoor dust (driven by the air-to-dust pathway) likely dominates exposure close to the facility since many of the yards have been remediated, decreasing the importance of incidental soil-ingestion. However, as you move away from the facility towards lower-risk zones, soils have not been remediated and consequently, they become more important in determining overall exposure and risk (with incidental ingestion of indoor dust decreasing somewhat in terms of its relative contribution to overall Pb exposure).
Table 4-13. Projections of IQ loss for the primary Pb smelter case study - current conditions.

<table>
<thead>
<tr>
<th>Percentile</th>
<th>Pop</th>
<th>Total blood Pb level (μg/dL)</th>
<th>IQ loss</th>
<th>Pathway Contribution*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Diet</td>
</tr>
<tr>
<td>IEUBK (concurrent blood Pb metric)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99.9th</td>
<td>4</td>
<td>21.9</td>
<td>6</td>
<td>2%</td>
</tr>
<tr>
<td>99.5th</td>
<td>19</td>
<td>12.4</td>
<td>4</td>
<td>4%</td>
</tr>
<tr>
<td>99th</td>
<td>39</td>
<td>7.4</td>
<td>3</td>
<td>4%</td>
</tr>
<tr>
<td>95th</td>
<td>194</td>
<td>3.7</td>
<td>1</td>
<td>22%</td>
</tr>
<tr>
<td>90th</td>
<td>388</td>
<td>2.9</td>
<td>1</td>
<td>34%</td>
</tr>
<tr>
<td>75th</td>
<td>970</td>
<td>2.0</td>
<td>-</td>
<td>45%</td>
</tr>
<tr>
<td>Median</td>
<td>1,940</td>
<td>1.3</td>
<td>-</td>
<td>56%</td>
</tr>
<tr>
<td>25th</td>
<td>2,910</td>
<td>0.9</td>
<td>-</td>
<td>56%</td>
</tr>
<tr>
<td>1st</td>
<td>3,841</td>
<td>0.4</td>
<td>-</td>
<td>54%</td>
</tr>
<tr>
<td>IEUBK (lifetime average blood Pb metric)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99.9th</td>
<td>4</td>
<td>28.6</td>
<td>5</td>
<td>2%</td>
</tr>
<tr>
<td>99.5th</td>
<td>19</td>
<td>16.9</td>
<td>3</td>
<td>5%</td>
</tr>
<tr>
<td>99th</td>
<td>39</td>
<td>10.6</td>
<td>2</td>
<td>5%</td>
</tr>
<tr>
<td>95th</td>
<td>194</td>
<td>5.3</td>
<td>-</td>
<td>26%</td>
</tr>
<tr>
<td>90th</td>
<td>388</td>
<td>4.1</td>
<td>-</td>
<td>29%</td>
</tr>
<tr>
<td>75th</td>
<td>970</td>
<td>2.7</td>
<td>-</td>
<td>62%</td>
</tr>
<tr>
<td>Median</td>
<td>1,940</td>
<td>1.8</td>
<td>-</td>
<td>25%</td>
</tr>
<tr>
<td>25th</td>
<td>2,910</td>
<td>1.2</td>
<td>-</td>
<td>56%</td>
</tr>
<tr>
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<td>3,841</td>
<td>0.5</td>
<td>-</td>
<td>54%</td>
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<td>39</td>
<td>6.8</td>
<td>&lt;1</td>
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<td>388</td>
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<td>-</td>
<td>29%</td>
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<td>3,841</td>
<td>0.3</td>
<td>-</td>
<td>54%</td>
</tr>
</tbody>
</table>

* inh (inhalation), ing (ingestion), diet (includes drinking water) and BCK (total background = diet + drinking water).
Table 4-14. Projections of IQ loss for primary Pb smelter case study - NAAQS attainment.

<table>
<thead>
<tr>
<th>Percentile</th>
<th>Pop</th>
<th>Total blood Pb level (μg/dL)</th>
<th>IQ loss</th>
<th>Pathway Contribution*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Diet</td>
</tr>
<tr>
<td><strong>IEUBK (concurrent blood Pb metric)</strong></td>
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<td></td>
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<tr>
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<td>3%</td>
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<td>19</td>
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<td>4</td>
<td>4%</td>
</tr>
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<td>7.9</td>
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<td>13%</td>
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</tr>
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<td>29%</td>
</tr>
<tr>
<td>25th</td>
<td>2,910</td>
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<td>45%</td>
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<tr>
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<td>3,841</td>
<td>0.4</td>
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<td>62%</td>
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<td>9%</td>
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<td>388</td>
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<td>54%</td>
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<tr>
<td>75th</td>
<td>970</td>
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<td>-</td>
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<tr>
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<td>2,910</td>
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<td>3,841</td>
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<td>44%</td>
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<td>10%</td>
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<td>95th</td>
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<td>25%</td>
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<td>90th</td>
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<td>22%</td>
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<td>970</td>
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<td>56%</td>
</tr>
<tr>
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<td>1,940</td>
<td>0.7</td>
<td>-</td>
<td>54%</td>
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<tr>
<td>25th</td>
<td>2,910</td>
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<td>-</td>
<td>57%</td>
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<tr>
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<td>3,841</td>
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<td>-</td>
<td>59%</td>
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<td><strong>Leggett (lifetime average blood Pb metric)</strong></td>
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<td>10%</td>
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<tr>
<td>99th</td>
<td>39</td>
<td>7.1</td>
<td>&lt;1</td>
<td>5%</td>
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<tr>
<td>95th</td>
<td>194</td>
<td>3.1</td>
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<td>35%</td>
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<tr>
<td>90th</td>
<td>388</td>
<td>2.3</td>
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<td>11%</td>
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<tr>
<td>75th</td>
<td>970</td>
<td>1.6</td>
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<td>45%</td>
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<tr>
<td>Median</td>
<td>1,940</td>
<td>1.1</td>
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<td>56%</td>
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<td>25th</td>
<td>2,910</td>
<td>0.7</td>
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<td>62%</td>
</tr>
<tr>
<td>1st</td>
<td>3,841</td>
<td>0.3</td>
<td>-</td>
<td>59%</td>
</tr>
</tbody>
</table>

* inh (inhalation), ing (ingestion), diet (includes drinking water) and BCK (total background = diet + drinking water).
4.4.2.2 Secondary Pb Smelter Case Study

As mentioned earlier, two sets of risk results were generated for the secondary Pb smelter case study: (a) risk estimates for the current conditions air quality scenario using a model-only approach for characterizing soil Pb impacts (Table 4-15) and (b) risk estimates for the current conditions scenario using a hybrid (model+empirical data) approach for characterizing soil Pb (Table 4-16). Because there were no projected exceedances of the Pb NAAQS for this case study location, the current NAAQS attainment scenario is the same as the current condition scenario and is not presented separately here. Risk results for the model-only approach range from no adverse impact to 1 IQ points lost (1st percentile to the 99.9th percentile simulated individual). Risk results for the hybrid approach range from 0 IQ points to 2 IQ points lost. It is important to point out that whole IQ point losses are only projected for the top 1% of the modeled child population (17 children) at this case study.

The differences between exposure and risk estimates for the two scenarios reflect the higher soil concentrations associated with the hybrid (model + empirical data) approach (see Section 4.3.2.3.2). This can be seen in the higher percentage of total blood Pb attributed to soil ingestion for the hybrid scenario (~20%) versus model-only scenario (<10%). Interestingly, the fraction of total blood Pb associated with dust ingestion is fairly similar (across percentile results) for the two scenarios, suggesting that soil has little impact on indoor dust Pb concentrations (i.e., even though we have increased the soil concentration in the hybrid scenario relative to the model-only scenario and used a dust model in the hybrid scenario based in part of soil concentrations, the overall impact on indoor dust seems to be small, given that the fraction of total blood Pb coming from dust ingestion remains largely unchanged between the two scenarios).

Risk results for the secondary Pb smelter should be considered in light of the fact that the highest impact U.S. Census block within the study area (i.e., the block having the highest modeled air concentration and deposition values and associated soil Pb levels), while having adult residents did not have any children 0-7 years of age, according to the U.S. Census for 2000. It is likely that, had children been located within this block, the upper tail of the exposure and risk distributions would have been significantly elevated in terms of blood Pb levels and IQ loss, respectively. This issue is addressed in the sensitivity analysis conducted for the pilot (see Tables 4-18 and 4-19).
Table 4-15. Projections of IQ loss for secondary Pb smelter case study - modeled soil Pb approach.

<table>
<thead>
<tr>
<th>Percentile</th>
<th>Pop</th>
<th>Total blood Pb level (μg/dL)</th>
<th>IQ loss</th>
<th>Pathway Contribution*</th>
</tr>
</thead>
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<td></td>
<td></td>
<td>Diet</td>
<td>Air-inh</td>
<td>Soil-ing</td>
</tr>
<tr>
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<td></td>
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<tr>
<td>99.9th</td>
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<td>3.7</td>
<td>1</td>
<td>63%</td>
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<td>99.5th</td>
<td>8</td>
<td>3.0</td>
<td>1</td>
<td>63%</td>
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<tr>
<td>99th</td>
<td>17</td>
<td>2.7</td>
<td>&lt;1</td>
<td>71%</td>
</tr>
<tr>
<td>95th</td>
<td>84</td>
<td>1.9</td>
<td>-</td>
<td>63%</td>
</tr>
<tr>
<td>90th</td>
<td>167</td>
<td>1.7</td>
<td>-</td>
<td>71%</td>
</tr>
<tr>
<td>75th</td>
<td>418</td>
<td>1.2</td>
<td>-</td>
<td>71%</td>
</tr>
<tr>
<td>Median</td>
<td>836</td>
<td>0.9</td>
<td>-</td>
<td>68%</td>
</tr>
<tr>
<td>25th</td>
<td>1,254</td>
<td>0.7</td>
<td>-</td>
<td>70%</td>
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<tr>
<td>1st</td>
<td>1,655</td>
<td>0.3</td>
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<td>69%</td>
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<td>IEUBK (lifetime average blood Pb metric)</td>
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<td>71%</td>
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<td>1,254</td>
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<td>1,655</td>
<td>0.4</td>
<td>-</td>
<td>67%</td>
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<tr>
<td>Leggett (concurrent blood Pb metric)</td>
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<tr>
<td>99.9th</td>
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<td>2.1</td>
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<td>67%</td>
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<tr>
<td>99.5th</td>
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<td>63%</td>
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<td>99th</td>
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<td>Median</td>
<td>836</td>
<td>0.5</td>
<td>-</td>
<td>65%</td>
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<td>0.3</td>
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<td>67%</td>
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<td>0.2</td>
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<td>69%</td>
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<td>Leggett (lifetime average blood Pb metric)</td>
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<tr>
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<td>0.7</td>
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<td>70%</td>
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<tr>
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<td>1,254</td>
<td>0.5</td>
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<td>71%</td>
</tr>
<tr>
<td>1st</td>
<td>1,655</td>
<td>0.2</td>
<td>-</td>
<td>70%</td>
</tr>
</tbody>
</table>

* inh (inhalation), ing (ingestion), diet (includes drinking water) and BCK (total background = diet + drinking water + fraction of soil Pb attributable to background, see Section 4.3.2.2).
Table 4-16. Projections of IQ loss for secondary Pb smelter case study -hybrid soil Pb approach.

<table>
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<th>Percentile</th>
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<th>IQ loss</th>
<th>Pathway Contribution*</th>
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<td></td>
<td></td>
<td>Diet</td>
</tr>
<tr>
<td>IEUBK (concurrent blood Pb metric)</td>
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<td>54%</td>
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<td>84</td>
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<td>&lt;1</td>
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<td>-</td>
<td>54%</td>
</tr>
<tr>
<td>Median</td>
<td>836</td>
<td>1.5</td>
<td>-</td>
<td>57%</td>
</tr>
<tr>
<td>25th</td>
<td>1,254</td>
<td>1.1</td>
<td>-</td>
<td>55%</td>
</tr>
<tr>
<td>1st</td>
<td>1,655</td>
<td>0.5</td>
<td>-</td>
<td>58%</td>
</tr>
<tr>
<td>Leggett (concurrent blood Pb metric)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99.9th</td>
<td>2</td>
<td>2.3</td>
<td>-</td>
<td>57%</td>
</tr>
<tr>
<td>99.5th</td>
<td>8</td>
<td>1.9</td>
<td>-</td>
<td>55%</td>
</tr>
<tr>
<td>99th</td>
<td>17</td>
<td>1.7</td>
<td>-</td>
<td>53%</td>
</tr>
<tr>
<td>95th</td>
<td>84</td>
<td>1.2</td>
<td>-</td>
<td>57%</td>
</tr>
<tr>
<td>90th</td>
<td>167</td>
<td>1.0</td>
<td>-</td>
<td>57%</td>
</tr>
<tr>
<td>75th</td>
<td>418</td>
<td>0.8</td>
<td>-</td>
<td>56%</td>
</tr>
<tr>
<td>Median</td>
<td>836</td>
<td>0.6</td>
<td>-</td>
<td>55%</td>
</tr>
<tr>
<td>25th</td>
<td>1,254</td>
<td>0.4</td>
<td>-</td>
<td>58%</td>
</tr>
<tr>
<td>1st</td>
<td>1,655</td>
<td>0.2</td>
<td>-</td>
<td>56%</td>
</tr>
<tr>
<td>Leggett (lifetime average blood Pb metric)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99.9th</td>
<td>2</td>
<td>3.3</td>
<td>-</td>
<td>57%</td>
</tr>
<tr>
<td>99.5th</td>
<td>8</td>
<td>2.7</td>
<td>-</td>
<td>56%</td>
</tr>
<tr>
<td>99th</td>
<td>17</td>
<td>2.5</td>
<td>-</td>
<td>55%</td>
</tr>
<tr>
<td>95th</td>
<td>84</td>
<td>1.8</td>
<td>-</td>
<td>55%</td>
</tr>
<tr>
<td>90th</td>
<td>167</td>
<td>1.5</td>
<td>-</td>
<td>56%</td>
</tr>
<tr>
<td>75th</td>
<td>418</td>
<td>1.1</td>
<td>-</td>
<td>57%</td>
</tr>
<tr>
<td>Median</td>
<td>836</td>
<td>0.8</td>
<td>-</td>
<td>55%</td>
</tr>
<tr>
<td>25th</td>
<td>1,254</td>
<td>0.6</td>
<td>-</td>
<td>57%</td>
</tr>
<tr>
<td>1st</td>
<td>1,655</td>
<td>0.3</td>
<td>-</td>
<td>55%</td>
</tr>
</tbody>
</table>

* inh (inhalation), ing (ingestion), diet (includes drinking water) and BCK (total background = diet + drinking water + fraction of soil Pb attributable to background, see Section 4.3.2.2).
4.4.2.3 Near Roadway (Urban) Case Study

Risk results for the near roadway (urban) case study range from no adverse impacts to 3 IQ points lost (1st percentile to the 99.9th percentile simulated individual) (see Table 4-17). It is important to note that whole IQ point losses are projected for the top 1-5% of the modeled population at this case study (3 to 16 children), depending on the concentration-response function (and associated cutpoint), with the remainder having projected blood Pb levels that do not exceed the cutpoints used in the analysis. In considering the risk results generated for this case study, it is important to reiterate the fact that the study area includes a 1.5 mile long urban road segment and the residents living within 200m of that road segment. Actual near roadway exposures across an urban or metropolitan area would be comprised of many such road segments and associated residential buffer areas and consequently would involve far larger study populations.

As with the exposure estimates discussed in Section 4.3.5, risk results for this case study exhibit a fairly consistent pattern regarding pathway apportionment, with policy-relevant sources typically contributing about 55-65% of total Pb exposure and policy-relevant background contributing the rest (as noted in Section 4.3.5, this likely reflects the fact that this case study was modeled using a small number of exposure bands which reduces the specificity in capturing gradients in population-level Pb exposure near the roadway). Unlike the other two case studies, for the near roadway (urban) case study, policy-relevant source exposure is split almost evenly between the incidental ingestion of soil and indoor dust. This likely reflects the relatively high historical soil concentrations associated with this case study, which increase the importance of the incidental soil ingestion pathway. Furthermore, the relatively low ambient air concentrations (relative to the other case studies) mean that indoor dust loading will be reduced which will de-emphasize that pathway in driving overall Pb exposure.
Table 4-17. Projections of IQ loss for near roadway (urban) case study.

<table>
<thead>
<tr>
<th>Percentile</th>
<th>Pop</th>
<th>Total blood Pb level (μg/dL)</th>
<th>IQ loss</th>
<th>Pathway Contribution*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Diet</td>
</tr>
<tr>
<td>IEUBK (concurrent blood Pb metric)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99.9th</td>
<td>&lt;1</td>
<td>6.5</td>
<td>3</td>
<td>44%</td>
</tr>
<tr>
<td>99.5th</td>
<td>2</td>
<td>5.0</td>
<td>2</td>
<td>44%</td>
</tr>
<tr>
<td>99th</td>
<td>3</td>
<td>4.4</td>
<td>2</td>
<td>44%</td>
</tr>
<tr>
<td>95th</td>
<td>16</td>
<td>3.1</td>
<td>&lt;1</td>
<td>44%</td>
</tr>
<tr>
<td>90th</td>
<td>32</td>
<td>2.6</td>
<td>&lt;1</td>
<td>44%</td>
</tr>
<tr>
<td>Median</td>
<td>80</td>
<td>1.9</td>
<td></td>
<td>44%</td>
</tr>
<tr>
<td>25th</td>
<td>159</td>
<td>1.4</td>
<td>-</td>
<td>35%</td>
</tr>
<tr>
<td>1st</td>
<td>239</td>
<td>1.0</td>
<td>-</td>
<td>35%</td>
</tr>
<tr>
<td>IEUBK (lifetime average blood Pb metric)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99.9th</td>
<td>&lt;1</td>
<td>9.1</td>
<td>1</td>
<td>44%</td>
</tr>
<tr>
<td>99.5th</td>
<td>2</td>
<td>7.0</td>
<td>&lt;1</td>
<td>44%</td>
</tr>
<tr>
<td>99th</td>
<td>3</td>
<td>6.2</td>
<td>&lt;1</td>
<td>44%</td>
</tr>
<tr>
<td>95th</td>
<td>16</td>
<td>4.4</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>90th</td>
<td>32</td>
<td>3.6</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>75th</td>
<td>80</td>
<td>2.7</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>Median</td>
<td>159</td>
<td>1.9</td>
<td>-</td>
<td>35%</td>
</tr>
<tr>
<td>25th</td>
<td>239</td>
<td>1.4</td>
<td>-</td>
<td>35%</td>
</tr>
<tr>
<td>1st</td>
<td>316</td>
<td>0.4</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>Leggett (concurrent blood Pb metric)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99.9th</td>
<td>&lt;1</td>
<td>4.2</td>
<td>2</td>
<td>44%</td>
</tr>
<tr>
<td>99.5th</td>
<td>2</td>
<td>3.1</td>
<td>&lt;1</td>
<td>44%</td>
</tr>
<tr>
<td>99th</td>
<td>3</td>
<td>2.7</td>
<td>&lt;1</td>
<td>35%</td>
</tr>
<tr>
<td>95th</td>
<td>16</td>
<td>1.9</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>90th</td>
<td>32</td>
<td>1.6</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>75th</td>
<td>80</td>
<td>1.2</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>Median</td>
<td>159</td>
<td>0.8</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>25th</td>
<td>239</td>
<td>0.6</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>1st</td>
<td>316</td>
<td>0.3</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>Leggett (lifetime average blood Pb metric)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>99.9th</td>
<td>&lt;1</td>
<td>6.9</td>
<td>&lt;1</td>
<td>20%</td>
</tr>
<tr>
<td>99.5th</td>
<td>2</td>
<td>4.9</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>99th</td>
<td>3</td>
<td>4.1</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>95th</td>
<td>16</td>
<td>2.9</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>90th</td>
<td>32</td>
<td>2.4</td>
<td>-</td>
<td>35%</td>
</tr>
<tr>
<td>75th</td>
<td>80</td>
<td>1.7</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>Median</td>
<td>159</td>
<td>1.2</td>
<td>-</td>
<td>35%</td>
</tr>
<tr>
<td>25th</td>
<td>239</td>
<td>0.9</td>
<td>-</td>
<td>44%</td>
</tr>
<tr>
<td>1st</td>
<td>316</td>
<td>0.4</td>
<td>-</td>
<td>44%</td>
</tr>
</tbody>
</table>

* inh (inhalation), ing (ingestion), diet (includes drinking water) and BCK (total background = diet + drinking water).
4.4.3 Uncertainty Analysis (Sensitivity Analysis, Performance Evaluation and Other Considerations)

This section discusses uncertainty associated with the pilot risk analysis. As mentioned in Section 4.2.5.7, for the pilot analysis, we completed a sensitivity analysis focusing on the impact of uncertainty in individual modeling elements on risk results. In addition to the sensitivity analysis results, the results of the performance evaluation conducted for the pilot analysis (section 4.3.6) can also be used to gain insights into elements of the analysis that might be subject to significant uncertainty. Finally, there are a range of potential sources of uncertainty, that, while not formally included in the sensitivity analysis or performance evaluation (due to a lack of data), can still be discussed qualitatively. These sources are also addressed in this section.

4.4.3.1 Sensitivity Analysis Methodology

As mentioned in Section 4.2.5.7, the sensitivity analysis completed for the pilot involved a "one element at a time elasticity analysis" in which the full model was run with one of the selected modeling elements adjusted to reflect an alternate (bounding if possible) input value or modeling choice. The results of that run with the modified modeling element were then compared to the "baseline risk run" to determine the magnitude of the impact on risk results generated by modifying that one modeling element. This procedure was repeated for all of the modeling elements selected for coverage in the sensitivity analysis.

The determination of the degree of impact from a given modeling element on risk results was based on a comparison of specific risk percentiles between the baseline and sensitivity analysis runs, including results generated for the 50th, 90th, 95th, 99th, and 99.5th simulated individuals. For example, we might compare the 90th percentile risk for the baseline run with that from a sensitivity analysis run reflecting a different option for a specific modeling element (e.g., different input dataset or different modeling approach). The difference between these two

---

19 Alternate models or input datasets for use in the sensitivity analysis were selected to reflect the range of options identified for a particular modeling element (i.e., given several values for a particular input parameter, the value representing the high or low bound on the range for that input would be selected). This approach reflects our desire that the sensitivity analysis capture, to the extent possible, the full impact of uncertainty in a particular modeling element on risk results.

20 For purposes of the sensitivity analysis, the "baseline run" was defined as a full risk run for the primary Pb smelter case study involving the following modeling choices: IEUBK biokinetic model and the concurrent blood Pb metric. It is important to emphasize that the defining of a baseline run does not place greater confidence in this particular combination of modeling elements, but rather reflects the need to have a set of risk results for use in gauging the magnitude of the impact of alternative modeling elements on risk results.
estimates of the 90th percentile risk would then be assigned to that particular modeling element to represent its impact on risk (i.e., the sensitivity of risk results to that modeling element).

Results of the sensitivity analysis for a particular modeling element are presented both in terms of (a) their absolute impact on IQ loss estimates (associated with the 99.9th percentile population percentile – see below) and (b) in terms of the percent difference between the baseline risk estimate and the estimates generated with modification of the element under consideration. Inclusion of a percentile impact metric in the sensitivity analysis makes it easier to rank modeling elements in terms of their overall impact on risk results. Note, that for some modeling elements, rather than having two data points (the baseline risk estimate and a single alternative from the sensitivity analysis run), we actually have three alternatives (the baseline risk estimate and a higher- and lower-end risk estimate generated by considering options producing both lower risk and higher risk estimates relative to baseline). In this case, we present all three risk estimates as well as the percent difference between the low- and high-end risk estimate, with the baseline risk estimate encompassed within this percent range.

The majority of the sensitivity analysis is based on the primary Pb smelter study area. Specifically, both the baseline run as well as the sensitivity analysis runs examining alternate options for specific modeling elements were completed using the primary Pb smelter. There are two exceptions to this. In the first, an analysis was completed focused on the secondary Pb smelter case study which considered the impact on risk estimates of locating children in the census block possessing the highest Pb impact on modeled media including ambient air, soil and indoor dust. Secondly, a sensitivity analysis was completed (also for the secondary Pb smelter), focusing on the characterization of Pb concentrations in outdoor soil. The results of the sensitivity analysis based on the primary Pb smelter case study are generally applicable to the other two case study locations. However, the results of the two analyses focused on the secondary Pb smelter are relevant only for this case study since they are considering site-specific factors related to exposure and risk modeling at this specific location.

Table 4-18 lists the modeling elements included in the sensitivity analysis and presents a brief summary of the alternative modeling approaches/inputs used to represent each within the sensitivity analysis. As mentioned earlier, consideration for which modeling elements would be included in the sensitivity analysis was based on professional judgment by the staff as to which modeling elements were likely to have significant impacts on risk and on consideration for data availability. Those modeling elements expected to have a significant impact on risk results but for which available data did not support inclusion in the sensitivity analysis are discussed qualitatively.
### Table 4-18. Modeling elements considered in the pilot sensitivity analysis (including summary of approaches used to derive alternate approaches/inputs).

<table>
<thead>
<tr>
<th>Modeling Element</th>
<th>Description</th>
<th>Baseline Run</th>
<th>Sensitivity Analysis Run(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Media modeling (indoor dust)</td>
<td>Statistical model used to predict indoor dust concentrations based on outdoor soil and/or outdoor ambient air</td>
<td>Two models used (see Section 4.3.2.4.1): A) site-specific statistical (air-only) model used for remediation zone near facility. B) pooled analysis model (air + soil) used for areas further from facility.</td>
<td>Two options considered: A) use pooled analysis model (soil + air) across entire study area. B) use pooled analysis model (air only) across entire study area.</td>
</tr>
<tr>
<td>Indoor dust modeling</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood Pb modeling</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined assessment focusing on oral absorption for background sources: - absolute absorption factor (water) - absolute absorption factor (diet)</td>
<td>Used to estimate uptake of Pb following dietary consumption and drinking water ingestion (background sources).</td>
<td>Single value used for both pathways (50 percentile)</td>
<td>Alternate values based on study data not identified. Therefore, conducted simple test of mathematical elasticity using factors that are 10% higher and 10% lower (i.e., 40% and 60% against the baseline value of 50%).</td>
</tr>
<tr>
<td>Combined assessment of factors related to intake and uptake modeling of soil and indoor dust: - soil/dust weighting factor - total fraction accessible (soil and dust)</td>
<td>Factors used to (a) determine the fraction of total soil+dust ingestion that is for each media (soil/dust weighting factor) and then (b) model uptake of Pb from ingestion soil and dust (total fraction accessible)</td>
<td>- soil/dust weighting factor: 45% - total fraction accessible: 48% for soil and 26% for dust</td>
<td>Alternate values obtained from Von Lindern et al., 2003. - soil-dust weighting factor: 58% - total fraction accessible: soil and dust are both 18%</td>
</tr>
<tr>
<td>Blood Pb modeling</td>
<td>Biokinetic and statistical (empirical) models used to predict blood Pb in children</td>
<td>IEUBK (see Section 4.3.3.1.1)</td>
<td>Two alternate models considered: - an alternate biokinetic model: Leggett (see Section 4.3.2.1) - a statistical (empirical) model: Lanphear et al., 1998 (see Risk Assessment Report Section 6.3.1)²¹</td>
</tr>
</tbody>
</table>

²¹ The Lanphear empirical model was developed by relating blood Pb measurements in young children to a number of factors including: (a) Pb exposures in air, soil, house Pb loading, drinking water, (b) the presence/absence...
<table>
<thead>
<tr>
<th>Modeling Element</th>
<th>Description</th>
<th>Baseline Run</th>
<th>Sensitivity Analysis Run(s)</th>
</tr>
</thead>
</table>
| Geometric standard deviation (GSD) | Used to represent inter-individual variability in Pb biokinetics and behavior related to Pb exposure. | 1.6 (see Section 4.3.3.4) | Two alternate values considered:  
- 1.3 (lower bound of GSDs provided for children living near Pb smelters, USEPA, 1989)  
- 1.9 (reflects range of GSDs found in NHEXAS study, USEPA, 2004). |
| IQ loss modeling | Statistical fit of log-linear concentration-response function model | Confidence intervals associated with fit of the Lanphear pooled analysis log-linear model | Best fit of log-linear model used in baseline run | Upper and lower 95th % confidence intervals on the model used in sensitivity analysis |
| Form of concentration-response function | Actual form of the concentration response function (e.g., log-linear, linear) | Log-linear (Lanphear et al., 2005) | Linear model with breakpoint at 10 μg/dL (i.e., linear model fit to subset of sample data with blood Pb levels <10 μg/dL) (Lanphear et al., 2005) |
| Blood Pb metric | Type of blood Pb measurement used to represent exposure (i.e., concurrent, lifetime average, peak) | Concurrent | Two metrics considered:  
- lifetime average  
- peak annual averaged |
| Cutpoint | Exposure level below which there is insufficient confidence (in the form of the concentration-response function) to predict IQ loss | Concurrent cutpoint: 2.4 μg/dL | Given that data applicable in establishing lower cutpoints are limited, alternate cutpoint was set as 1/2 baseline cutpoints (i.e., 1.2 μg/dL) |

Sensitivity analyses focused on the secondary Pb smelter

- Location of children close to the facility in the U.S. Census block with the greatest Pb media impacts
- Secondary Pb smelter has a U.S. Census block located close to the facility with high air and soil Pb impacts and with adult residents, but with no child residents (this means it is not included in projections of child exposure and risk estimates for the study area)
- Baseline run does not consider this block in calculating risk since the U.S. Census data used in the analysis identify no child residents within that block
- Calculate child exposure and risk estimates for this high-impact block as if there were children living there. Specifically, calculate mean and high-end percentile exposure and risk estimates specifically for that block to gain perspective for the magnitude of risk which could exist if children did live in that block.

of Pb paint and (c) a range of socioeconomic variables. The best fitting regression model included Pb concentrations in soil, and Pb loadings in house dust. In order to apply the Lanphear model in the pilot, it was necessary to convert the estimated house dust exposure concentrations derived for each study area into dust Pb loadings. This was done using a regression relationship based on the same underlying 1997 national Pb housing survey data used in developing the Lanphear empirical blood Pb model (see Section 6.3.1 of the Risk Assessment Report for additional details).
<table>
<thead>
<tr>
<th>Modeling Element</th>
<th>Description</th>
<th>Baseline Run</th>
<th>Sensitivity Analysis Run(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Characterization of Pb concentrations in outdoor soil</td>
<td>Fate and transport modeling used to predict soil Pb levels based on modeled air concentration and deposition over the study area. Performance evaluation for soil modeling suggests under-prediction using MPE fate and transport modeling (compared with measured soil data at surrogate location - see Table 4-11).</td>
<td>Model-only approach: use of MPE fate and transport modeling to predict soil concentrations.</td>
<td>Hybrid model-empirical data approach: use of MPE modeling to generate a soil concentration surface characterizing the relative spatial profile of soil levels combined with surrogate measurement data for soil Pb near secondary smelters to &quot;scale up&quot; the modeled surface to match the surrogate data. (Note, inclusion of this hybrid model reflects consideration for surrogate data (Kimbrough et al., 1995) which suggests underestimation by model-only approach – see performance evaluation discussion in Table 4-10). This hybrid modeling approach resulted in soil concentrations approximately 3X higher than the model-only approach.</td>
</tr>
</tbody>
</table>

### 4.4.3.2 Sensitivity Analysis Results

The results of the sensitivity analysis are summarized in Table 4-19 (see Table 4-18 for detail on each modeling element included in the sensitivity analysis). Because of the potential importance of higher-end risk percentiles in decision making, the presentation of sensitivity analysis results here includes only the range of predicted 99.9th percentile risk estimates. However, the percentile range associated with each modeling element (i.e., the percent difference between the baseline and alternative option runs) reflects the results for the 90th-99.9th percentiles. For a more complete presentation of the sensitivity analysis results please refer to the Risk Assessment Report, Section 6.3.

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22 The hybrid (model-empirical data) approach considered in the sensitivity analysis for the secondary Pb smelter case study also includes a modification to the approach used in modeling indoor dust, compared with the baseline approach. While indoor dust for the baseline run (using soil data generated with the model-only approach) used the AGG air-only dust model, the sensitivity analysis run used the AGG air+soil dust model. As discussed in Section 4.3.2.4.2, the decision to switch to the AGG air+soil dust model reflects the fact that the increased soil Pb levels considered in the sensitivity analysis run required a dust model that factors those increased soil concentrations in predicting indoor dust in order to assess their full impact on modeled blood Pb levels (i.e., use of the AGG air-only dust model would have produced lower blood Pb levels since the impact of increased soil Pb concentrations on indoor blood Pb levels would not have been considered). Inclusion of the AGG air+soil dust model in the sensitivity analysis run does complicate interpretation of the results since they reflect the combined impact of the higher soil Pb levels as well as the effect of switching from the AGG air-only dust model to the AGG air+soil dust model.
Table 4-19. Summary of Sensitivity Analysis Results.

<table>
<thead>
<tr>
<th>Modeling Element</th>
<th>Risk (IQ loss) estimates for the 99.9th simulated individual (baseline run and sensitivity analysis run)*</th>
<th>Percentile difference (baseline vs alternative option) across population percentiles: 90th to 99.9th **</th>
</tr>
</thead>
<tbody>
<tr>
<td>Media modeling (indoor dust)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indoor dust modeling</td>
<td>Baseline run: -5</td>
<td>-35% to +30%</td>
</tr>
<tr>
<td></td>
<td>Sensitivity analysis runs: AGG (air-only): -5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>AGG (air+soil): -4</td>
<td></td>
</tr>
<tr>
<td>Blood Pb modeling</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined assessment:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- absolute absorption factor (water)</td>
<td>Baseline run: -5</td>
<td>-88% to +52%</td>
</tr>
<tr>
<td>- absolute absorption factor (diet)</td>
<td>Sensitivity analysis runs: AF diet, water (40%): -4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>AF diet, water (60%): -6</td>
<td></td>
</tr>
<tr>
<td>Combined assessment:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- soil/dust weighting factor</td>
<td>Baseline run: -5</td>
<td>-5 to -88%</td>
</tr>
<tr>
<td>- total fraction accessible (soil and dust)</td>
<td>Sensitivity analysis run: Van Lindern values: -4</td>
<td></td>
</tr>
<tr>
<td>Blood Pb models</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Baseline run: -5</td>
<td>-100 to +55%</td>
</tr>
<tr>
<td></td>
<td>Sensitivity analysis runs: Leggett: -5</td>
<td></td>
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<tr>
<td></td>
<td>Lanphear: -5</td>
<td></td>
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<tr>
<td>GSD</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Baseline run: -5</td>
<td>-51% to +124%</td>
</tr>
<tr>
<td></td>
<td>Sensitivity analysis runs: GSD (1.3): -5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>GSD (1.9): -6</td>
<td></td>
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<tr>
<td>IQ loss modeling</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Statistical fit of log-linear model</td>
<td>Baseline run: -5</td>
<td>-53% to +80%</td>
</tr>
<tr>
<td></td>
<td>Sensitivity analysis runs: 95th % LCL: -1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>95th % UCL: -8</td>
<td></td>
</tr>
<tr>
<td>Form of concentration-response function</td>
<td>Baseline run: -5</td>
<td>-12% to +102%</td>
</tr>
<tr>
<td></td>
<td>Sensitivity analysis run: Linear (10 μg/dL breakpoint): -9</td>
<td></td>
</tr>
<tr>
<td>Blood Pb metric</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Baseline run: -5</td>
<td>-15% to +395%</td>
</tr>
<tr>
<td></td>
<td>Sensitivity analysis runs: Lifetime averaged: -4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Highest annual (peak): -8</td>
<td></td>
</tr>
<tr>
<td>Modeling Element</td>
<td>Risk (IQ loss) estimates for the 99.9th simulated individual (baseline run and sensitivity analysis run)*</td>
<td>Percentile difference (baseline vs alternative option) across population percentiles: 90th to 99.9th **</td>
</tr>
<tr>
<td>------------------</td>
<td>-------------------------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Cutpoint</td>
<td>Baseline run: -5</td>
<td>+43% to +412%</td>
</tr>
<tr>
<td></td>
<td>Sensitivity analysis run: 1/2 baseline cutpoint: -8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>This sensitivity analysis departs from the others in focusing only on risk for the single block of interest and not for the entire study area.</td>
<td>Percentile difference is not applicable here because the baseline results is across the entire study area and the sensitivity results are for the high-impact block not included in the baseline run. But the results clearly demonstrate that the extreme tail of this case study's risk distribution would be pushed higher if children were located in this high-impact block.</td>
</tr>
<tr>
<td></td>
<td>Baseline run: Note, the baseline run has no children in this block, so there would be no IQ loss projected for the block with the highest Pb media concentrations. However, the 99.9th% risk level modeled for the entire study area ranges from -1 to -2 IQ points.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sensitivity analysis run: Risk levels for max-impact block (assuming children are there):</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-- mean: -2 to -5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-- 95th%: -4 to -7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-- 99th%: -4 to -8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Outdoor soil Pb modeling (implemented using the secondary Pb smelter case study)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Baseline run: -1</td>
<td>+100% to +300%</td>
</tr>
<tr>
<td></td>
<td>Sensitivity analysis run: (X3) soil concentrations: -2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>It is interesting to note that, while differences in blood Pb levels are only on the order of 30 to 50%, between the baseline and sensitivity analysis run (across percentiles) given the non-linearities in the concentration-response function near this lower range of blood Pb levels, the differences in IQ can be far greater, as seen here.</td>
<td></td>
</tr>
</tbody>
</table>

* As noted in text, the 99.9th% risk estimates was selected as the basis for presenting results in this table given the potential importance of higher-end risk results in supporting decision making. The sensitivity analysis did include results for a range of percentile risk metrics (see Risk Assessment Report Section 6.3 for additional detail).

** Percent difference reflects sensitivity analysis results seen across the 90th to 99.9th percentile simulated individuals (Note, that many of the highest sensitivity analysis results – those exceeding 100% - were seen for impacts on the 90th-95th percentile simulated individual results and not for higher percentiles).
The sensitivity analysis results presented in Table 4-19 result in the following conclusions regarding the sensitivity of risk results generated for the pilot to specific sources of uncertainty:

- **Modeling elements with the greatest impact**: Modeling elements with high impacts (relative to the entire set considered in the sensitivity analysis) include: (a) characterization of soil Pb levels (for the secondary Pb smelter case study), (b) the blood Pb model, (c) the blood Pb metric, (d) the GSD, (e) the concentration-response function and (f) the cutpoint.

- **Overall perspective on magnitude of uncertainty in high-end risk results**: Consideration of the range of impacts across modeling elements presented in Table 4-19 suggests that overall uncertainty (resulting from these factors) would likely be under one order of magnitude. Note, as stated earlier, this is only a qualitative assessment – true quantitative uncertainty estimates would require a formal probabilistic uncertainty analysis.

### 4.4.3.3 Additional Considerations

As the performance evaluation (described in Section 4.3.6) involved comparison of modeled results against available empirical data, we are using this in addition to the sensitivity analysis (described in Section 4.4.3.1) to characterize potential uncertainty associated with specific steps of the pilot analysis. This section also discusses potential sources of uncertainty that have not been quantitatively investigated, but still deserve qualitative discussion.

#### 4.4.3.3.1 Performance Evaluation

From the performance evaluation (presented in Section 4.3.6), we have drawn the following conclusions regarding potential uncertainty in projected media concentrations and blood Pb levels for the three case studies:

- **Outdoor air Pb concentrations for the primary Pb smelter**: Performance evaluation completed for air modeling at this case study suggests an overestimation of air Pb levels closer to the facility (in the range of perhaps 50-60%) with air concentrations further from the facility potentially underestimated. The potential overestimation of air concentrations near the facility is a potentially important factor since it could result in overestimations of exposure (through both direct inhalation and indirectly through indoor dust loading) for that portion of the study area likely to experience the highest risks.

- **Outdoor air concentrations for the secondary Pb smelter**: Performance evaluation completed for air modeling at this case study suggests the possibility of a potential underestimation of air concentrations. This is an important observation since it could results in under-predicted exposures and risks for this case study.

- **Outdoor soil concentrations at the secondary Pb smelter**: Performance evaluation of modeled outdoor Pb soil concentrations at the secondary Pb smelter suggests a potentially significant underestimation (up to a factor of 3.0). This finding from the
performance evaluation led to this issue being specifically addressed in the sensitivity analysis (see Tables 4-18 and 4-19) and this issue will not be discussed further here.

- **Blood Pb levels for the primary Pb smelter:** Two performance evaluations were conducted for blood Pb levels generated for this case study: (a) a comparison of the median modeled blood Pb level for the case study against the median blood Pb level for 1-5 yr olds in the U.S. (NHANES IV) and (b) a comparison of the high-end modeled blood Pb levels (95th-99.9th percentile) against the high-end percentiles from a blood screening study completed for children living near the smelter (US DHHS, 2003). The results of the fist assessment suggest that our modeling is not generating unreasonable central-tendency blood Pb levels for the study area. The second assessment (focusing on high-end exposure percentiles) suggests that our modeling is also generating reasonable high-end exposures for that subpopulation, given the measurement data that are available.

- **Blood Pb levels for the secondary Pb smelter and near roadway (urban) case study:** As with the primary Pb smelter, performance evaluation focusing on central-tendency blood Pb levels for this case study suggest that our modeling is not generating unreasonable central-tendency blood Pb levels.

### 4.4.3.3.2 Qualitative Discussion of Uncertainty

The staff has identified a number of modeling elements with the potential to introduce uncertainty into the risk results, that were not addressed quantitatively in the sensitivity analysis and performance evaluation, the results of which are discussed above. These elements and the potential uncertainty associated with them in the pilot analysis are discussed below, with the discussion organized by modeling step.

**Characterizing media concentrations**

Sources of potential uncertainty in characterizing media concentrations which are not explicitly considered in sensitivity analysis or performance evaluation are described here.

- **Source characterization:** The estimates of Pb emissions rates and particle size profiles for the two point source case studies remains an area of uncertainty. The uncertainty is greatest with regard to fugitive emissions, both on the facility property and any associated re-suspension outside the property.

- **Paint Pb as a component of indoor dust:** The models used in the pilot analysis to estimate indoor dust Pb (based on Pb in air and soil), may include some contribution from paint Pb in their estimates. Specifically, all of the statistical models used in the pilot to predict indoor dust Pb include intercept terms which represent that fraction of indoor dust Pb not related to variations in outdoor soil Pb or air Pb. Given the data sets from which regressions were derived, it is likely that these intercept terms reflect, to some extent, paint Pb. For the pilot, the total value predicted by these models for indoor dust Pb was treated as policy-relevant and no effort was made to identify the
fraction of predicted indoor dust Pb levels resulting from Pb paint. There is the potential, then, that the predictions of indoor dust Pb have over-estimated the policy-relevant component by not excluding the paint Pb signal.

- **Characterizing the spatial gradient of outdoor air concentrations for the near roadway (urban) case study:** The use of dispersion model results for highway-related diesel (and other PM component) emissions to derive spatial gradients for re-entrained near roadway air-bound Pb is subject to uncertainty.

### Exposure Analysis and Blood Pb modeling

Sources of potential uncertainty in characterizing exposure and estimating blood Pb levels which are not explicitly considered in sensitivity analysis or performance evaluation are described here.

- **Modeling of a static child population (in terms of media concentrations and residence time within the study area):** The risk results generated for the pilot assume that modeled child populations at all three case studies (a) come in contact with fixed media concentrations (i.e., they do not change over time) and (b) reside for their entire exposure period within the case study. There is uncertainty associated with these simplifying assumptions. For example, media concentrations may change with time; this has been observed with recontamination of remediated soil near the primary Pb smelter (USEPA, 2006e). With regard to residence time, it is likely that children residing in the study area reflect a range of residence times, some having lived for most, if not all of their lifetimes, while others may only spend only a fraction of their time living near the facility. The assumption that all of the modeled children live in the study area for their entire exposure period is conservative and will contribute to an overestimation of Pb exposure for the case study.

### 4.5 SUMMARY OF FINDINGS AND CONSIDERATIONS FOR THE FULL-SCALE ASSESSMENT

This section summarizes the risk results and analyses of uncertainty for the pilot assessment (Section 4.5.1) and discusses plans for the full-scale assessment (Section 4.5.2) that will be presented in the second draft of this document. The detailed risk results for individual case studies are presented in Sections 4.4.2.1, 4.4.2.2 and 4.4.2.3.

We note that the primary purpose of the pilot assessment was to test out methodologies and design features of the assessment, as well as to assess the availability of different types of information pertinent to this assessment. Consequently, the risk results associated with the pilot are not intended to reflect our best estimates of risk associated with these case studies. Rather, they reflect preliminary estimates, limited by the initial application of our modeling tools and information. We intend to build on our experience and findings associated with the pilot assessment in designing and implementing the full-scale risk assessment, the purpose of which is
to inform the Agency’s development of and consideration of NAAQS policy options with regard to policy relevant sources of ambient Pb.

4.5.1 Summary of Findings in the Pilot Assessment

Risk results generated for the three case studies suggest that individuals in the upper 10\textsuperscript{th} to 5\textsuperscript{th} percentile of exposure (depending on case study) have the potential for quantifiable IQ loss associated with projected Pb exposure with IQ decrements ranging from less than an IQ point to greater than six IQ points. Among the three case studies, the greatest IQ loss was projected for children living in the vicinity of the primary Pb smelter case study, followed by the near roadway (urban) case study and the secondary Pb smelter case study. For the primary Pb smelter, individuals in the upper 10\textsuperscript{th} percentile (with IQ losses ranging from <1 to 6 points) had exposures dominated by contributions from policy-relevant sources (~98%), with incidental ingestion of indoor dust being by far the dominant pathway. For the secondary Pb smelter, individuals in the upper 5\textsuperscript{th} percentile (with IQ losses ranging from <1 to 2 points) have between 20\% and 40\% of their exposure associated with policy-relevant sources (this range reflecting the model-only approach and the hybrid approach, respectively). Contributions to IQ loss from policy-relevant sources are estimated to split evenly between soil and dust ingestion for the model-only scenario, but becomes dominated by soil ingestion, for the hybrid scenario (as would be expected given the higher soil Pb concentrations associated with the hybrid scenario). For the near roadway (urban) case study, individuals in the upper 10\textsuperscript{th} percentile (with IQ losses ranging from <1 to 3 points) have about half of their exposure coming from policy-relevant sources, with the majority of this coming from indoor dust ingestion.\textsuperscript{23}

Risk results generated for the secondary Pb smelter should be carefully considered in light of the fact that areas near the facility with the highest projected Pb media concentrations, while having adult residents, do not have any child residents. This resulted in these areas of potentially higher exposures not contributing any risk for this case study. A sensitivity analysis examining this issue showed that IQ loss estimates for the closest (adult-only) U.S. Census block would range from 2 to 8 points if child residents were included in that block. This finding

\textsuperscript{23} Note, that the pilot analysis did not explicitly model paint Pb exposure. However, it is likely that modeling of Pb exposure resulting from indoor dust Pb ingestion, which is treated as policy-relevant in the pilot, does reflect some degree of paint Pb impact (see Section 4.2.6.5.1). Depending on the degree to which indoor dust Pb ingestion does reflect paint Pb, there is the potential that policy-relevant exposures and risk presented in the pilot may be over-stated. The issue of paint Pb impacts to indoor dust, and efforts to separate it out and treat it as background, is an area that will continue to be researched as part of the full scale analysis.
illustrates the potential impact of population demographics in this assessment. This factor will be considered in selecting additional case studies for the full-scale analysis.

Performance evaluation completed for the pilot analysis (described in Section 4.3.6) has indicated the following with regard to model predictions for Pb concentrations in environmental media: (a) air modeling results for the primary Pb smelter suggest a potential for moderate overestimation of levels near the facility and underestimation of values further out, and (b) air modeling and soil modeling results for the secondary Pb smelter suggest a potentially significant underestimation of Pb concentrations in both media. Of these, the more significant conclusion appears to involve the secondary Pb smelter case study. These findings may contribute to an overall low bias in exposure and risk results generated for this case study. This, combined with the finding regarding child residents in the census block with highest projected media concentrations, indicates the need to make improvements in our characterization of potential risk associated with intermediate scale point source facilities in the full-scale assessment.

The sensitivity analysis indicated that risk estimates for the 90th to 99.9th percentiles could vary by up to several hundred percent depending on the approach or parameters used for certain aspects of the analysis. Modeling elements identified as having potentially significant impacts on risk results include: (a) characterization of soil Pb levels (for the secondary Pb smelter case study), (b) the blood Pb model, (c) the blood Pb metric, (d) the GSD for blood Pb distributions (e) the concentration-response function, and (f) the cutpoint for the concentration-response function. All of these areas, and others discussed previously, will be considered in finalizing our plans for the full-scale assessment.

4.5.2 Potential Areas for Enhancement in the Full-Scale Analysis

The staff intends that the full-scale risk assessment will provide a quantitative risk characterization to inform consideration of policy options pertaining to policy relevant exposures to ambient Pb. Our plans for the full-scale analysis will reflect our critical assessment of the pilot and its results as well as consideration of comments provided by the public and CASAC.

For the full-scale analysis, we are planning to include the following:

- **Additional case studies:** We are considering including additional case studies, e.g., representative of additional ambient Pb exposure situations.
- **Additional air quality scenarios:** We will be considering additional air quality scenarios to inform evaluation of alternate policy options.
In addition, based on experience in the pilot assessment, we are considering the following potential enhancements for the full-scale assessment:

- **Consideration for alternate exposure periods**: We intend to consider the potential impact on risk results from modeling shorter exposure periods (i.e., periods shorter than the annual average values used in the pilot). Factors to consider in relation to this issue include response time for key risk-driving media (e.g., soil and indoor dust), given changes in ambient air Pb levels and the capability of blood Pb models for tracking shorter-term fluctuations in Pb exposure.

- **Aspects of air dispersion modeling**: We intend to review and refine, as feasible, aspects of the air dispersion modeling step. This will include source characterization, as well as the choice of air dispersion model.

- **Characterization of Pb re-suspension**: For the near roadway (urban) case study, we are considering the feasibility of using source apportionment analyses of available PM speciation data to estimate the fraction of airborne Pb associated with re-entrainment of previously deposited Pb.

- **Soil Pb modeling**: We are considering the use of a compartmental mass balance model (i.e., Total Risk Integrated Methodology Fate, Transport and Ecological exposure model, TRIM.FaTE) (USEPA, 2002b, 2002c) to characterize temporal changes in soil Pb associated with different Pb emissions and deposition situations. An additional related consideration is characterization of background soil Pb levels (e.g., alternatives to the 15 mg/kg background soil Pb level used in characterizing the secondary Pb smelter case study).

- **Indoor dust Pb modeling**: We intend to review and refine, as feasible, aspects of the indoor dust modeling step. This includes refining our prediction of policy-relevant source contributions, as differentiated from policy-relevant background sources (e.g., paint Pb).

- **Inhalation absorption estimates**: We are considering ways to update the Pb inhalation absorption factors used in the biokinetic blood Pb models with consideration of current information both with regard to Pb particle size distribution associated with exposures for the various case studies, and with regard to respiratory tract Pb particle deposition and absorption.

- **Stability in probabilistic population-level exposure modeling**: We will reexamine the stability criteria used to establish the number of realizations used in probabilistic exposure modeling for the pilot (10,000) to insure that we have sufficiently stable high-end results.
REFERENCES


December 2006                                     4-82                                     Draft – Do Not Quote or Cite


5 THE PRIMARY LEAD NAAQS

5.1 INTRODUCTION

This first draft chapter discusses the general approach (Section 5.2) that is intended to be used in considering the adequacy of the current standard and in identifying policy alternatives for the next draft of this document. The current Pb NAAQS and its derivation are summarized in Section 5.3, and conclusions from the Staff Paper prepared in the last review are presented in Section 5.4. Key uncertainties and research recommendations related to setting a primary lead standard will be identified in the next draft of this document.

The current standard is 1.5 \( \mu g \) Pb/m\(^3\), as a maximum arithmetic mean averaged over a calendar quarter, set to provide protection to the public, especially children as the particularly sensitive population subgroup, against Pb-induced adverse health effects (43 FR 46246). In identifying options for the Administrator’s consideration in this review, we note that the final decision on retaining or revising the current Pb standard is largely a public health policy judgment. A final decision should draw upon scientific information and analyses about health effects, population exposure and risks, as well as judgments about the appropriate response to the range of uncertainties that are inherent in the scientific evidence and analyses. Our approach to informing these judgments, discussed more fully below, is based on a recognition that the available health effects evidence generally reflects a continuum consisting of ambient levels at which scientists generally agree that health effects are likely to occur, through lower levels at which the likelihood and magnitude of the response become increasingly uncertain.

This approach is consistent with the requirements of the NAAQS provisions of the Act and with how EPA and the courts have historically interpreted the Act. These provisions require the Administrator to establish primary standards that, in the Administrator's judgment, are requisite to protect public health with an adequate margin of safety. In so doing, the Administrator seeks to establish standards that are neither more nor less stringent than necessary for this purpose. The Act does not require that primary standards be set at a zero-risk level but rather at a level that avoids unacceptable risks to public health, including the health of sensitive groups.

5.2 APPROACH

As indicated in Chapter 1, the policy assessment to be presented in the final version of this document is intended to inform judgments required by the EPA Administrator in determining whether it is appropriate to retain or revise the NAAQS for Pb. In evaluating whether it is appropriate to consider retaining the current primary Pb standard, or whether consideration of revisions is appropriate, we intend to adopt an approach in this review that
builds upon the general approach used in the initial setting of the standard, as well as in the last
review, and reflects the broader body of evidence now available. As summarized in section 5.3,
the 1978 notice of final rulemaking (43 FR 46246) outlined key factors considered in selecting
the elements of a standard for Pb: the Pb concentration (i.e., level); the averaging time; and the
form (i.e., the air quality statistic to be used as a basis for determining compliance with the
standard). Decisions on these elements were based on an integration of information on health
effects associated with exposure to ambient Pb; expert judgment on the adversity of such effects
on individuals; and policy judgments as to when the standard is requisite to protect public health
with an adequate margin of safety, which were informed by air quality and related analyses,
quantitative exposure and risk assessments when possible, and qualitative assessment of impacts
that could not be quantified.

In developing conclusions and identifying options for the Pb standard in this review, staff
intends to take into account both evidence-based and quantitative exposure- and risk-based
considerations. A series of general questions will frame our approach to reaching conclusions
and identifying options for consideration by the Administrator in deciding whether to retain or
revise the current primary Pb standard. Examples of questions that we intend to address in our
review include the following:

- To what extent does newly available information reinforce or call into question
evidence of associations with effects identified in the last review?

- To what extent has evidence of new effects and/or sensitive populations become
available since the last review?

- To what extent have important uncertainties identified in the last review been reduced
and have new uncertainties emerged?

- To what extent does newly available information reinforce or call into question any of
the basic elements of the current standard?

To the extent that the available information suggests that revision of the current standard may be
appropriate to consider, we intend to also address whether the currently available information
supports consideration of a standard that is either more or less protective by addressing questions
such as the following:

- Is there evidence that associations, especially likely causal associations, extend to air
quality levels that are as low as or lower than had previously been observed, and what
are the important uncertainties associated with that evidence?

- Are exposures of concern and health risks estimated to occur in areas that meet the
current standard; are they important from a public health perspective; and what are the
important uncertainties associated with the estimated risks?
To the extent that there is support for consideration of a revised standard, we will then identify ranges of standards (in terms of an indicator, averaging time, level, and form) that would reflect a range of alternative public health policy judgments, based on the currently available information, as to the degree of protection that is requisite to protect public health with an adequate margin of safety. In so doing, we would address the following questions:

- Does the evidence provide support for considering a different Pb indicator?
- Does the evidence provide support for considering different averaging times?
- What ranges of levels and forms of alternative standards are supported by the evidence, and what are the uncertainties and limitations in that evidence?
- To what extent do specific levels and forms of alternative standards reduce the estimated exposures of concern and risks attributable to Pb, and what are the uncertainties associated with the estimated exposure and risk reductions?

As noted in Chapter 1, staff will also evaluate removing Pb from the criteria pollutant list and assess whether revocation of the Pb NAAQS is an option appropriate for the Administrator to consider. Section 108 of the Clean Air Act states that the Administrator “shall, from time to time … revise a list which includes each pollutant -

(A) Emissions of which, in his judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare;
(B) The presence of which in the ambient air results from numerous or diverse mobile or stationary sources; and
(C) For which air quality criteria had not been issued before December 31, 1970, but for which he plans to issue air quality criteria under this section.”

In evaluating such an option, staff expects to consider, among other things, many of the same issues identified earlier in the section. Information about the kinds and types of sources of Pb emissions, as well as the quantities of emissions from those sources will also be important for consideration.

5.3 PRIMARY LEAD STANDARD

As mentioned earlier, the current primary Pb NAAQS was promulgated in 1978. The basis for its establishment is described below.

5.3.1 Level

The level of the current NAAQS is 1.5 μg/m³. EPA’s objective in setting the level of the current standard was “to estimate the concentration of lead in the air to which all groups within the general population can be exposed for protracted periods without an unacceptable risk to
health” (43 FR 46252). Consistent with section 109 of the Clean Air Act, the Agency identified a level for the current standard that was not considered to be at the threshold for adverse health effects, but was at a lower level in order to provide a margin of safety (see Section 5.3.1.5). As stated in the notice of final rulemaking (and further described in the following subsections),

“This estimate was based on EPA’s judgment in four key areas:

(1) Determining the ‘sensitive population’ as that group within the general population which has the lowest threshold for adverse effects or greatest potential for exposure. EPA concludes that young children, aged 1 to 5, are the sensitive population.

(2) Determining the safe level of total lead exposure for the sensitive population, indicated by the concentration of lead in the blood. EPA concludes that the maximum safe level of blood lead for an individual child is 30 µg Pb/dl and that population blood lead, measured as the geometric mean, must be 15 µg Pb/dl in order to place 99.5 percent of children in the United States below 30 µg Pb/dl.

(3) Attributing the contribution to blood lead from nonair pollution sources. EPA concludes that 12 µg Pb/dl of population blood lead for children should be attributed to nonair exposure.

(4) Determining the air lead level which is consistent with maintaining the mean population blood lead level at 15 µg Pb/dl [the safe level]. Taking into account exposure from other sources (12 µg Pb/dl), EPA has designed the standard to limit air contribution after achieving the standard to 3 µg Pb/dl. On the basis of an estimated relationship of air lead to blood lead of 1 to 2, EPA concludes that the ambient air standard should be 1.5 µg Pb/m³.” (43 FR 46252)

5.3.1.1 Sensitive Population

The assessment of the science that was presented in the 1977 CD (USEPA, 1977), indicated young children, aged 1 to 5, as the population group at particular risk from Pb exposure. Children were recognized to have a greater physiological sensitivity than adults to the effects of Pb and a greater exposure. In identifying young children as the sensitive population, EPA also recognized the occurrence of subgroups with enhanced risk due to genetic factors, dietary deficiencies or residence in urban areas. Yet information was not available to estimate a threshold for adverse effects for these subgroups separate from that of all young children. Additionally, EPA recognized both a concern regarding potential risk to pregnant women and fetuses, and a lack of information to establish that these subgroups are more at risk than young children. Accordingly, young children, aged 1 to 5, were identified as the group which has the lowest threshold for adverse effects of greatest potential for exposure (i.e., the sensitive population) (43 FR 46252).
5.3.1.2 Maximum Safe Blood Level

In identifying the maximum safe exposure, EPA relied upon the measurement of Pb in blood (43 FR 46252-46253). The physiological effect of Pb that had been identified as occurring at the lowest blood Pb level, was inhibition of an enzyme integral to the pathway by which heme (the oxygen carrying protein of human blood) is synthesized, i.e., delta-aminolevulinic acid dehydratase (δ-ALAD). The 1977 CD reported a threshold for inhibition of this enzyme in children at 10 μg Pb/dL. The 1977 CD also reported a threshold of 15-20 μg/dL for elevation of protoporphyrin (EP), which is an indication of some disruption of the heme synthesis pathway. EPA concluded that this effect on the heme synthesis pathway (indicated by EP) was potentially adverse. EPA further described a range of blood levels associated with a progression in detrimental impact on the heme synthesis pathway. At the low end of the range (15-20 μg/dL), the initial detection of EP associated with blood Pb, was not concluded to be associated with a significant risk to health. The upper end of the range (40 μg/dL), the threshold associated with clear evidence of heme synthesis impairment and other effects contributing to clinical symptoms of anemia, was regarded as clearly adverse to health. EPA also recognized the existence of thresholds for additional adverse effects (e.g., nervous system deficits) occurring for some children at just slightly higher blood Pb levels (e.g., 50 μg/dL). Additionally, EPA stated that the maximum safe blood level should not be higher than the blood Pb level recognized by the CDC as “elevated” (and indicative of the need for intervention). In 1978, that level was 30 μg/dL1.

Once identifying the maximum safe blood level in individual children, EPA next made the policy-based judgment regarding the target mean blood level for the U.S. population of young children (43 FR 46252-46253). With this judgment, EPA identified a target of 99.5 percent of this population to be brought below the maximum safe blood Pb level. This judgment was based on consideration of the size of the sensitive subpopulation, and the recognition that there are special high risk groups of children within the general population. The population statistics available at the time (the 1970 U.S. Census) indicated a total of 20 million children younger than 5 years of age, with 15 million residing in urban areas and 5 million in center cities where Pb exposure was thought likely to be “high”. Concern about these high risk groups influenced EPA’s determination of 99.5%, deterring EPA from selecting a population percentage lower than 99.5 (43 FR 46253). EPA then used standard statistical techniques to calculate the population mean that would place 99.5 percent of the population below the maximum safe level. Based on the then available data, EPA concluded that the blood Pb levels in the population of

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1 The CDC subsequently revised their advisory level for children’s blood Pb to 25 μg/dL in 1985, and to 10 μg/dL 1991. More details on this level are provided in Section 3.2.
U.S. children were normally distributed with a geometric standard deviation of 1.3. Based on standard statistical techniques, a thus described population in which 99.5 percent of the population has blood Pb levels below 30 μg/dL has a geometric mean blood level of 15 μg/dL.

5.3.1.3 Nonair Contribution

When setting the current NAAQS, EPA recognized that the air standard needed to take into account the contribution to blood Pb levels from Pb sources unrelated to air pollution. Consequently, the calculation of the current NAAQS included the subtraction of Pb contributed to blood Pb from nonair sources from the estimate of a safe mean population blood Pb level. Without this subtraction, EPA recognized that the combined exposure to Pb from air and nonair sources would result in a blood Pb concentration exceeding the safe level (43 FR 46253).

In developing an estimate of this nonair contribution, EPA recognized the lack of detailed or widespread information about the relative contribution of various sources to children’s blood levels, such that an estimate could only be made by inference from other empirical or theoretical studies, often involving adults. Additionally, EPA recognized the expectation that the contribution to blood Pb levels from nonair sources would vary widely, was probably not in constant proportion to air Pb contribution, and in some cases may alone exceed the target mean population blood Pb level (43 FR 46253-46254).

The amount of blood Pb attributed to nonair sources was selected based primarily on findings in studies of blood Pb levels in areas where air levels were low relative to other locations in U.S. The air levels in these areas ranged from 0.1 to 0.7 μg/m³. The average of the reported blood levels for children of various ages in these areas was on the order of 12 μg/dL. So 12 μg/dL was identified as the nonair contribution, and subtracted from the population mean target level of 15 μg/dL to yield a value of 3 μg/dL as the limit on the air contribution to blood Pb.

5.3.1.4 Air Pb Level

In determining the air Pb level consistent with an air contribution of 3 μg Pb/dL, EPA reviewed studies assessed in the 1977 CD that reported changes in blood Pb with different air Pb levels. These studies included a study of children exposed to Pb from a primary Pb smelter, controlled exposures of adult men to Pb in fine particulate matter, and a personal exposure study involving several male cohorts exposed to Pb in a large urban area in the early 1970s (43 FR 46254). Using all three studies, EPA calculated an average slope or ratio over the entire range of data. That value was 1.95 (rounded to 2 μg/dL blood Pb concentration to 1 μg/m³ air concentration), and is recognized to fall within the range of values reported in the 1977 CD. On the basis of this 2 to 1 relationship, EPA concluded that the ambient air standard should be 1.5 μg Pb/m³ (43 FR 46254).
5.3.1.5 Margin of Safety

In consideration of the appropriate margin of safety during the development of the current NAAQS, EPA identified the following factors: (1) the 1977 CD reported multiple biological effects of Pb in practically all cell types, tissues and organ systems, of which the significance for health had not yet been fully studied; (2) no beneficial effects of Pb at then current environmental levels were recognized; (3) data were incomplete as to the extent to which children are indirectly exposed to air Pb that has moved to other environmental media, such as water, soil and dirt, and food; (4) Pb is chemically persistent and with continued uncontrolled emissions would continue to accumulate in human tissue and the environment; and (5) the possibility that exposure associated with blood Pb levels previously considered safe might influence neurological development and learning abilities of the young child (43 FR 46255). Recognizing that estimating an appropriate margin of safety for the air Pb standard was complicated by the multiple sources and media involved in Pb exposure, EPA chose to use margin of safety considerations principally in establishing a maximum safe blood Pb level for individual children (30 μg Pb/dL) and in determining the percentage of children to be placed below this maximum level (about 99.5). Additionally, in establishing other factors used in calculating the standard, EPA used margin of safety in the sense of making careful judgment based on available data, but these judgments were not considered to be at the precautionary extreme of the range of data available at the time (43 FR 46251).

EPA further recognized that because of the variability between individuals in a population experiencing a given level of Pb exposure, it was considered impossible to provide the same size margin of safety for all members in the sensitive population or to define the margin of safety in the standard as a simple percentage. EPA believed that the factors it used in designing the standards provided an adequate margin of safety for a large proportion of the sensitive population. The Agency did not believe that the margin was excessively large or on the other hand that the air standard could protect everyone from elevated blood Pb levels (43 FR 46251).

5.3.2 Averaging Time, Form, and Indicator

The averaging time for the current standard is a calendar quarter. In the decision for this aspect of the standard, the Agency also considered a monthly averaging period, but concluded that “a requirement for the averaging of air quality data over calendar quarter will improve the validity of air quality data gathered without a significant reduction in the protectiveness of the standards.” As described in the notice for this decision (43 FR 46250), this conclusion was based on several points, including the following:
• An analysis of ambient measurements available at the time indicated that the distribution of air Pb levels was such that there was little possibility that there could be sustained periods greatly above the average value in situations where the quarterly standard was achieved.

• A recognition that the monitoring network may not actually represent the exposure situation for young children, such that it seemed likely that elevated air Pb levels when occurring would be close to Pb air pollution sources where young children would typically not encounter them for the full 24-hour period reported by the monitor.

• Medical evidence available at the time indicated that blood Pb levels re-equilibrate slowly to changes in air exposure, a finding that would serve to dampen the impact of short-term period of exposure to elevated air Pb.

• Direct exposure to air is only one of several routes of total exposure, thus lessening the impact of a change in air Pb on blood Pb levels.

The statistical form of the current standard is as a not-to-be-exceeded or maximum value. EPA set the standard as a ceiling value with the conclusion that this air level would be safe for indefinite exposure for young children (43 FR 46250).

The indicator is total airborne Pb collected by high volume sampler (43 FR 46258). EPA’s selection of total suspended particulate Pb as the indicator for the standard was based on explicit recognition both of the significance of ingestion as an exposure pathway for Pb that had deposited from the air and of the potential for Pb deposited from the air to become re-suspended in respirable size particles in the air and available for human inhalation exposure. As stated in the final rule, “a significant component of exposure can be ingestion of materials contaminated by deposition of lead from the air”, and that, “in addition to the indirect route of ingestion and absorption from the gastrointestinal tract, non-respirable Pb in the environment may, at some point become respirable through weathering or mechanical action” (43 FR 46251).

5.4 POLICY OPTIONS CONSIDERED IN THE LAST REVIEW

During the 1980s, EPA initiated a review of the air quality criteria and NAAQS for Pb. CASAC and the public were fully involved in this review, which led to the publication of a criteria document with associated addendum and a supplement (USEPA, 1986a, 1986b, 1990a), an exposure analysis methods document (USEPA, 1989) and a staff paper (USEPA, 1990b).

Total emissions to air were estimated to have dropped by 94 percent between 1978 and 1987, with the vast majority of it attributed to the reduction of Pb in gasoline. Accordingly, the focus of this review was on areas near stationary sources of Pb emissions. Although such sources were not considered to have made a significant contribution (as compared to Pb in gasoline) to the overall Pb pollution across large, urban or regional areas, Pb emissions from such sources were considered to have the potential for a significant impact on a local scale. Air,
and especially soil and dust Pb concentrations had been associated with elevated levels of Pb absorption in children and adults in numerous Pb point source community studies. Exceedances of the current NAAQS were found at that time only in the vicinity of nonferrous smelters or other point sources of Pb.

In summarizing and interpreting the health evidence presented in the 1986 CD and associated documents, the 1990 Staff Paper described the collective impact on children of the effects at blood Pb levels above 15 μg/dL as representing a clear pattern of adverse effects worthy of avoiding. This is in contrast to EPA’s identification of 30 μg/dL as a safe blood Pb level for individual children when the NAAQS was set in 1978. The staff paper further stated that at levels of 10-15 μg/dL, there was a convergence of evidence of Pb-induced interference with a diverse set of physiological functions and processes, particularly evident in several independent studies showing impaired neurobehavioral function and development. Further, the available data did not indicate a clear threshold in this blood Pb range. Rather, it suggested a continuum of health risks down to the lowest levels measured.2

For the purposes of comparing the relative protectiveness of alternative Pb NAAQS, the staff conducted analyses to estimate the percentages of children with blood Pb levels above 10 μg/dL and above 15 μg/dL for several air quality scenarios developed for a small set of stationary source exposure case studies. These analyses omitted young children, whom it was considered could not be substantially affected by any changes in atmospheric Pb emissions under different standards (e.g., those with excessive pica3 and/or living in overtly deteriorated Pb-paint homes). The results of the analyses of children populations living near two Pb smelters indicated that substantial reductions in Pb exposure could be achieved through attainment of the current Pb NAAQS. According to the best estimate analyses, over 99.5% of children living in areas significantly affected by the smelters would have blood Pb levels below 15 μg/dL if the current standard was achieved. Progressive changes in this number were estimated for the alternative monthly Pb NAAQS levels evaluated, ranging from 1.5 μg/m³ to 0.5 μg/m³.

The staff paper, in light of the health effects evidence available at the time, in addition to air quality, exposure and risk analyses, and other policy considerations, presented the following staff conclusions with regard to the primary Pb NAAQS (USEPA, 1990b, pp. xii to xiv):

1) “The range of standards … should be from 0.5 to 1.5 μg/m³.”

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2 In 1991, the CDC reduced their advisory level for children’s blood Pb from 25 μg/dL to 10 μg/dL.
3 Pica is an eating disorder typically defined by persistent cravings to eat non-food items.
2) “A monthly averaging period would better capture short-term increases in lead exposure and would more fully protect children’s health than the current quarterly average.”

3) “The most appropriate form of the standard appears to be the second highest monthly averages \textit{sic} in a 3-year span. This form would be nearly as stringent as a form that does not permit any exceedances and allows for discounting of one “bad” month in 3 years which may be caused, for example, by unusual meteorology.”

4) “With a revision to a monthly averaging time more frequent sampling is needed, except in areas, like roadways remote from lead point sources, where the standard is not expected to be violated. In those situations, the current 1-in-6 day sampling schedule would sufficiently reflect air quality and trends.”

5) “Because exposure to atmospheric lead particles occurs not only via direct inhalation, but via ingestion of deposited particles as well, especially among young children, the hi-volume sampler provides a reasonable indicator for determining compliance with a monthly standard and should be retained as the instrument to monitor compliance with the lead NAAQS until more refined instruments can be developed.”

After consideration of the documents developed during the review, EPA chose not to propose revision of the NAAQS for Pb. During the same time period, the Agency published and embarked on the implementation of a broad, multi-program, multi-media, integrated national strategy to reduce Pb exposures (USEPA, 1991). As part of implementing this strategy, the Agency focused efforts primarily on regulatory and remedial clean-up actions aimed at reducing Pb exposures from a variety of non-air sources judged to pose more extensive public health risks to U.S. populations, as well as on actions to reduce Pb emissions to air, particularly near stationary sources. EPA established standards for Pb-based paint hazards and Pb dust cleanup levels in most pre-1978 housing and child-occupied facilities. Additionally, EPA has developed standards for the management of Pb in solid and hazardous waste, oversees the cleanup of Pb contamination at Superfund sites, and has issued regulations to reduce Pb in drinking water (http://www.epa.gov/lead/regulation.htm). Beyond these specific regulatory actions, the Agency’s Lead Awareness Program has continued to work to protect human health and the environment against the dangers of Pb by conducting research and designing educational outreach activities and materials (http://www.epa.gov/lead/). Actions to reduce Pb emissions to air during the 1990s included enforcement of the NAAQS, as well as the promulgation of regulations under Section 112 of the Clean Air Act, including national emissions standards for hazardous air pollutants at primary and secondary Pb smelters, as well as other Pb sources.
REFERENCES


6 POLICY RELEVANT ASSESSMENT OF WELFARE EFFECTS

6.1 INTRODUCTION

This chapter presents information in support of the review of the secondary NAAQS for lead (Pb). Welfare effects addressed by the secondary NAAQS include, but are not limited to, effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being. Lead is persistent in the environment and accumulates in soils and sediments thereby providing long term exposures to organisms and ecosystems. Past emissions of Pb from the use of Pb additives in automobile fuel significantly contributed to the widespread increase of Pb concentrations in the environment, a portion of which remains today.

This chapter includes a summary of policy relevant information presented in the CD, with effects of Pb in terrestrial ecosystems discussed in Section 6.2 and aquatic ecosystems discussed in Section 6.3. For some criteria pollutants, key effects and concentration responses are much more fully understood. For Pb, it is difficult to generalize effects due to the nature of the data and the general lack of community or population level information on the effects of Pb.

Therefore, this chapter attempts to describe the effects of Pb on ecosystems by grouping known effects into categories of organisms and summarizing the limited anecdotal information that is available for broader ecosystem effects of Pb. Sections 6.4 and 6.5 describe the screening level analyses that were conducted in this assessment in support of the current NAAQS review. These analyses are intended to identify areas of exposure for which there is the potential for adverse effects from Pb and could be used to focus further analyses on those areas. At this time, we do not anticipate having funding to perform additional ecological risk assessment work for this review. That is, the focus for this review with regard to the secondary standard will be on what we have learned from this pilot phase, in addition to the science assessment in the criteria document.

6.2 EFFECTS IN TERRESTRIAL ECOSYSTEMS

Ecosystems near smelters, mines and other industrial sources of Pb have demonstrated a wide variety of adverse effects including decreases in species diversity, loss of vegetation, changes to community composition, decreased growth of vegetation, and increased number of invasive species. Apportioning these effects between Pb and other stressors is problematic since these point sources also emit a wide variety of other heavy metals as well as SO₂ which may cause toxic effects. There are no field studies which have investigated effects of Pb additions alone but some studies near large point sources of Pb have found significantly reduced species composition and altered community structures. While these effects are significant, they are
spatially limited: the majority of contamination occurs within 20 to 50 km of the emission source (CD, AX7.1.4.2).

By far, the majority of Pb found in terrestrial ecosystems was deposited from the long range transport of Pb including Pb additives used in gasoline in the past few decades. There is little evidence that sites exposed to long range transport of Pb have experienced significant effects on ecosystem structure or function (CD, AX7.1.4.2). Studies have shown decreasing levels of Pb in vegetation which seems to correlate with decreases in atmospheric deposition of Pb resulting from the removal of Pb additives to gasoline (CD, AX 7.1.4.2). Little work, however, has been done on the effect of residual long term, low-level metal concentration on species diversity.

As stated in the CD (Section 7.1), terrestrial ecosystems remain primarily sinks for Pb but amounts retained in various soil layers vary based on forest type, climate, and litter cycling. Once in the soil, the migration and distribution of Pb is controlled by a multitude of factors including pH, precipitation, litter composition, and other factors which govern the rate at which Pb is bound to organic materials in the soil (CD, Section 2.3.5).

Like most metals the solubility of Pb is increased at lower pH. However, the reduction of pH may in turn decrease the solubility of dissolved organic material (DOM). Given the close association between Pb mobility and complexation with DOM, a reduced pH does not necessarily lead to increased movement of Pb through terrestrial systems and into surface waters. Studies have shown that in areas with moderately acidic soil (i.e., pH of 4.5 to 5.5) and abundant DOM, there is no appreciable increase in the movement of Pb into surface waters compared to those areas with neutral soils (i.e., pH of approximately 7.0). This appears to support the theory that the movement of Pb in soils is limited by the solubilization and transport of DOM. In sandy soils without abundant DOM, moderate acidification appears likely to increase outputs of Pb to surface waters (CD, AX 7.1.4.1).

Forest harvesting and management practices have significant and lasting effects on organic matter cycling in forest ecosystems. Clear cutting, as well as other methods of tree removal, leads to decreased organic matter for several years after harvesting and organic matter remaining in soils is exposed to higher temperatures and moisture which tend to increase rates of decomposition. Despite these effects, studies have shown very little to no mobilization of Pb from soils to surface waters following clear cutting. On possible explanation for this is that mineral soils (those below the biologically active, organic layer of soil) are efficient in capturing and retaining mobilized Pb. Loss of Pb in particulate form due to runoff and erosion in clear cut areas remains a potential source of Pb to surface waters.

As described in Chapter 2 (Sections 2.5 to 2.7) and in the CD (Chapter 7 and the Chapter 7 Annex), Pb emitted anthropogenically into the atmosphere accumulates in surface soils and vegetation throughout the Unites States as a result of wet and dry deposition. The following
discussion relies heavily on information presented in Chapters 2, 7, 8 of the CD and the Chapter 7 Annex of the CD.

6.2.1 Pathways of Exposure

The main pathways of exposure to Pb for animals are inhalation and ingestion. Inhalation exposures, which would be limited to areas immediately surrounding point sources, are not thought to be common and little information is available about inhalation in wildlife. Ingestion constitutes the main pathway of exposure for most organisms whether by incidental ingestion or prey contamination. For higher organisms which may ingest either contaminated plants or soils/sediments, the form and species of Pb ingested influences uptake and toxicity as does the presence of other heavy metals. The relative toxicity of metal mixtures and their affects on Pb toxicity is complex and varies greatly between species and metal.

For plants, direct deposition onto surfaces and uptake of dissolved Pb by roots is the main exposure route (CD, Section 7.1.3). While the migration and biological uptake of Pb in ecosystems is relatively low compared to other metals, there are many factors which may affect the mobility of Pb, including elevation and climate, vegetation type, acidity, and soil composition. The bioavailability and accessibility of Pb to plants is determined largely by the soil pH, chemical form of Pb, presence of other metals, and source of the Pb in the ecosystem. Low pH soils enhance bioavailability to plants and Pb chlorides and acetates are more bioavailable than Pb oxides. These factors directly relate to the ability of Pb complexes to enter pore water in soils and sediments and thereby enter root tissues.

6.2.2 Effects of Lead on Energy Flow and Biogeoecycling

Lead in soils and leaf litter can have a significant adverse effect on energy flow in terrestrial ecosystems through reducing the rate of litter decomposition and by decreasing photosynthetic rates in plants, both of which alter the ecosystem carbon cycling and may reduce the ability of trees and other plants to obtain nutrients from the soil (CD, AX7.1.4.3). Recent studies have associated high Pb concentrations in soils, such as those found near point sources, with reduced fungal and bacterial activity. This can lead to interruptions in various metabolic pathways by either reducing symbiotic relationships between the roots of some types of plants and fungi and/or bacteria or by tying up nutrients needed for plant growth (CD, AX7.1.4.3).

In less contaminated areas removed from point sources, there is little evidence that Pb represents a threat to energy flow or carbon cycling or that large pulses of Pb are likely to enter surface waters. Recent studies have shown that atmospheric deposition of Pb has decreased dramatically (>95%) over the last three decades and residence times in soils (the time for Pb to move out of the biologically active layers of soil) range from about 60 years in deciduous forests to 150 years in coniferous stands (CD, AX7.1.2.2).
6.2.3 Tools for Identifying Ecotoxicity in Terrestrial Organisms

In recognition of a need by EPA’s Superfund Program to identify the potential for adverse effect from various pollutants in soils to ecosystems, a multi-stakeholder group, consisting of federal, state, consulting, industry, and academic participants developed Ecological Soil Screening Levels (Eco-SSLs) for various pollutants including Pb. Eco-SSLs describe the concentrations of contaminants in soils that would result in little or no measurable effect on ecological receptors (USEPA, 2005a). They are intentionally conservative in order to provide confidence that contaminants, which could present an unacceptable risk, are not screened out early in the evaluation process (intended to be a specific site under consideration of the Superfund Program). That is, at or below these levels, adverse effects are considered unlikely. These values are defined in the Ecological Soil Screening Levels for Lead (USEPA, 2005a) as “concentrations of contaminants in soil that are protective of ecological receptors that commonly come into contact with soil or ingest biota that live in or on soil.” They were derived separately for four general categories of ecological receptors: plants, soil invertebrates, birds, and mammals.

In the case of plants and soil invertebrates, Eco-SSLs are expressed as concentration of Pb in soil (mg Pb/kg soil) and were developed with consideration of characteristics affecting bioavailability (e.g., pH, organic content, etc). The development of Eco-SSLs for avian and mammalian wildlife involved a two step process: 1) derivation of a toxicity reference value (TRV) in mg contaminant per kg body weight per day from available literature, and 2) application of the TRV with information on soil intake, foraging habits, diet, contaminant uptake by prey for a single species to derive an Eco-SSL in mg Pb per kg soil. In general for avian and mammalian wildlife categories, a single TRV was developed (e.g., the reference dose for the most sensitive of the adverse ecological effects on birds) for all species in each category. However, default assumptions regarding incidental soil ingestion, foraging techniques, contaminant intake by prey, and overall diet composition generally resulted in different Eco-SSL values, expressed as soil concentrations, for the different species in each receptor category. The receptor category Eco-SSL was then set equal to the lowest species-specific Eco-SSL (USEPA, 2005a; ICF, 2006). The Eco-SSLs for Pb, as developed by EPA Superfund Program, for terrestrial plants, birds, mammals, and soil invertebrates are 120 mg/kg, 11 mg/kg, 56 mg/kg and 1700 mg/kg, respectively. Section 2.6.2.2 discusses current concentrations of Pb in soils. Values range from 40 to 100 mg Pb/kg soil in remote forests where historic deposition of Pb from gasoline would be presumed to be the major source to hundreds to tens of thousands of mg/kg near point sources.

By comparing known or modeled soil concentrations of Pb to the Eco-SSL value derived for each receptor group, Eco-SSL values can be used to identify locations for which further analyses are warranted to determine adverse effects from Pb. Soil screening values, including...
Eco-SSLs, were used in this way in the ecological screening analyses conducted for this assessment and are discussed more fully in Section 6.4 of this document.

### 6.2.4 Effects on Plants

As discussed in Section 7.3.1 of the CD, atmospheric deposition of Pb onto vegetation is the primary route of exposure to plants from atmospheric Pb. Lead enters plant tissues primarily through direct transport, whether by surface deposition or through the soil. There is some uptake through root cell walls via pore water but little Pb is translocated to other parts of the plant by this mechanism. Most Pb that does enter plant tissues is deposited in the roots.

Toxicity to plants occurs over a broad range of soil Pb concentrations (tens to thousands of mg/kg) due in part, to the interaction between various soil processes and the bioavailability of Pb to plants (CD, Section 7.1.4). Laboratory studies have shown great variation in toxicity to plants based on the route of exposure and the form of Pb to which the plants are exposed. Two main factors make it very difficult to determine concentration responses for plants in the field: 1) the large number of confounding factors that need to be controlled for, and 2) the lack of good field sites without multiple metal exposures. The 1986 CD (USEPA, 1986) indicated that most plants experience reduced growth when Pb concentrations in pore water exceed 2 to 10 mg/kg and when soil concentrations exceed 10,000 mg/kg under conditions of low bioavailability (e.g., high pH, oxide rather than acetate forms, etc.) Under increased bioavailability, Pb would cause reduced growth at much lower levels (e.g. <100 mg/kg). More recent studies have indeed indicated effects at much lower levels than 10,000 mg/kg in the laboratory. For example, at 2,800 mg Pb/kg dry weight of soil, adverse effects on growth were found for radish shoots when exposed to Pb-chloride in mildly acidic sandy loams and at 12,000 mg/kg for shoots under similar exposures to Pb-oxide (CD, Section AX7.1.4). Root cell elongation, another indicator of growth, was inhibited in ryegrass at <2.5 mg/kg Pb-chloride and absence of root growth was observed at 5 mg/kg. Elevated toxicity was also found for red spruce and ryegrass when exposed to Pb under low pH conditions (CD, Section AX7.1.3.1). There is a wide breadth of studies discussed in the CD for various plants in the laboratory which indicate that Pb in concentrations found in soils near point sources could reduce plant growth. Despite this information, there are very few reports of phytotoxicity from Pb exposure under field conditions. Indeed two studies cited in Section AX7.1.3.2 of the CD found no indication of toxicity in plants exposed to high soil concentrations of Pb and other heavy metals near mining sites despite relatively high concentrations of Pb in the vegetation (4000 µg/g in Leita et al., 1989). Overall, the phytotoxicity of Pb is considered relatively low because little Pb enters plants from soil and what Pb does enter into plant tissue is deposited in roots where it is either detoxified or sequestered.
6.2.5 Effects on Birds and Mammals

The primary source of Pb exposure to birds and mammals is through dietary intake of both contaminated food items and incidental ingestion of soils/sediments. Direct inhalation of Pb rarely accounts for more than 10 to 15% of daily exposures and drinking water exposure is not a significant source of Pb for most organisms (CD, AX7.1.3.1).

Physiological effects from Pb exposure in birds and mammals include increased lipid peroxidation (fat breakdown) and effects on blood component production (CD, Section AX7.1.2.5). Lipid peroxidation and fatty acid changes have been linked to changes in immune system response and bone formation. Other adverse effects may include changes in juvenile growth rates; delay of reproductive maturity; behavioral effects, such as decreased predator avoidance or lack of balance and coordination; and mortality. This cascade of effects has the potential to influence populations by reducing the number of organisms and the rate at which they are replaced, as well as altering food web composition.

Toxic effects to birds from Pb exposure have been observed over a wide range of doses in laboratory studies, usually measuring reproductive success, but little to no data are available on field populations. Studies have found few significant effects in birds below doses of 100mg/kg in the diet and there is evidence that wide ranges of effects levels may be expected. Even in studies focused on reproductive effects in the same species, effects from doses ranging from <1 to >100 mg Pb/kg bw/day have been observed (CD, AX7.1.3.5). This variation is also true for other effects (e.g. behavioral and physiological effects) which have been observed at lower doses. As described in Section AX7.1.3.3 of the CD, no data are available on inhalation exposures of birds and very little research has been done since the 1986 CD on toxicity from Pb to birds not exposed to sediment (waterfowl). A discussion of effects to waterfowl can be found in Section 6.3.2.4 of this document.

Soil Pb concentrations and potential toxicity to birds has been considered in the development of Eco-SSLs by EPA’s Office of Solid Waste and Emergency Response (USEPA, 2005b). As discussed in Section 6.2.3, a soil Pb concentration of 11 mg/kg dry weight of soil was derived as the Eco-SSL for birds (woodcock) (CD, Section AX7.1.4). This concentration is commonly exceeded in many areas including those not influenced by point sources (CD, Sections 3.2 and AX7.1.2.3).

Toxic effects to mammals from Pb exposure have also been observed over a wide range of doses in laboratory studies with little information available for field populations or exposures. Recent studies indicate that effects on wildlife survival would likely occur at higher doses than the 2 to 8 mg/kg-day reported in the 1986 CD. Several studies have recently reported no observed adverse effect levels (NOAELs) for survival ranging from 3.5 to as high as 3200 mg/kg-day (CD, AX7.1.3.3). No inhalation studies were found to evaluate endpoints in mammals and in those studies used to develop toxicity endpoints, organisms were dosed using
either ingestion or gavage (tube feeding) which may not necessarily simulate exposure levels in
the field.

A Pb Eco-SSL has been derived for mammals (shrews) at 56 mg/kg dry weight of soil
based in part on toxicity reference values established for reproductive and growth effects
(USEPA, 2005b). Soil concentrations exceeding 56 mg Pb/kg are not uncommon in
urban/industrial locations or near major roadways and may indeed also occur in areas influenced
by deposition of gasoline derived Pb without current Pb emission sources (CD, Section 3.2 and
AX7.1.2.3).

Several behavioral and physiological processes seem to alter the toxicity of Pb in birds
and mammals. Nutritionally deficient diets, especially those low in calcium, lead to increased
uptake of Pb from the diet. Studies have also shown that younger animals and females are
generally more sensitive to Pb, insectivorous animals may be more highly exposed than
herbivores, and higher trophic level organism are less exposed than lower trophic level
organisms.

### 6.2.6 Effects on Decomposers and Soil Invertebrates

Elevated concentrations of Pb in soils can lead to decreased decomposition rates either by
direct toxicity to specific groups of decomposers, by deactivating enzymes excreted by
decomposers to break down organic material or by binding with organic matter and making it
resistant to the action of decomposers. Direct adverse effects to invertebrates, such as
earthworms and nematodes, include decreased survival, growth and reproduction. Toxicity has
been observed in soil invertebrates and microorganisms at concentrations of hundreds to
thousands of mg Pb/kg soil with significant variation due to soil parameters such as pH and
amount of organic matter (CD, Section AX 7.1.2).

As discussed in Section 6.4.2 and CD Section 7.1.4, an Eco-SSL of 1700 mg/kg dry
weight of soil has been derived for soil invertebrates (USEPA, 2005). This concentration does
not appear to be commonly exceeded in areas not directly influenced by point sources (CD,
Sections 3.2 and AX7.1.2.3).

Several physiological mechanisms for reducing Pb toxicity have been found among
invertebrates and microorganisms. These include enzyme mediated detoxification in two species
of spider, Pb storage in waste nodules in earthworms and storage as an inert compound,
pyromorphite, in nematodes. Avoidance of contaminated substrates and reduced feeding has
also been observed in invertebrates.

### 6.2.7 Summary

Lead exists in the environment in various forms which vary widely in their ability to
cause adverse effects on ecosystems and organisms. Current levels of Pb in soil also vary widely
depending on the source of Pb but in all ecosystems Pb concentrations exceed what is thought to

*December 2006*
be natural background levels. The deposition of gasoline-derived Pb into forest soils has
produced a legacy of slow moving Pb that remains bound to organic materials despite the
removal of Pb from most fuels and the resulting dramatic reductions in overall deposition rates.
For areas influenced by point sources of air Pb, concentrations of Pb in soil may exceed by many
orders of magnitude the concentrations which are considered harmful to laboratory organisms.
Adverse effects associated with Pb include neurological, physiological and behavioral effects
which may influence ecosystem structure and functioning. Eco-SSLs have been developed for
Superfund site characterizations to indicate concentrations of Pb in soils below which no adverse
effects are expected to plants, soil invertebrates, birds and mammals. Values like these may be
used to identify areas in which there is the potential for adverse effects to any or all of these
receptors based on current concentrations of Pb in soils.

6.3 EFFECTS IN AQUATIC ECOSYSTEMS

Atmospheric Pb enters aquatic ecosystems primarily through the erosion and runoff of
soils containing Pb and deposition (wet and dry). While overall deposition rates of atmospheric
Pb have decreased dramatically since the removal of Pb additives from gasoline, Pb continues to
accumulate and may be re-exposed in sediments and water bodies throughout the U.S (CD,
Section 2.3.6).

Several physical and chemical factors govern the fate and bioavailability of Pb in aquatic
systems. A significant portion of Pb remains bound to suspended particulate matter in the water
column and eventually settles into the substrate. Species, pH, salinity, temperature, turbulence
and other factors govern the bioavailability of Pb in surface waters (CD, Section 7.2.2).

6.3.1 Tools for Identifying Ecotoxicity in Aquatic Organisms

Ambient Water Quality Criteria (AWQC) were developed by U.S. EPA to provide
guidance to states and tribes to use in adopting water quality standards. AWQC values are
available for freshwater and marine environments and for chronic and acute exposures. These
values vary with water hardness and are based on the amount of dissolved Pb in the water
column. They are derived from toxicity testing on aquatic organisms, including fish,
invertebrates and algae and are considered to be values below which no adverse effect is
anticipated (USEPA, 1993). Therefore these values are useful in identifying locations for which
there is the potential for adverse effect from Pb. Section 6.4 describes how these criteria were
used in the risk characterization for the ecological analyses that accompany this review.

A number of sediment ecotoxicity screening values have been developed to identify the
concentration of Pb in sediment at which the potential for adverse effects occur. EPA has
recently published an equilibrium partitioning method for sediment which incorporates the
bioavailability of Pb and allows for mixtures of metals but may not account for ingestion of
sediment by sediment dwelling organisms. There are other alternative approaches for deriving sediment criteria which are based more directly upon comparisons between concentrations of Pb in sediment and associated effects from toxicity tests. These methods do not account for bioavailability or metal mixtures but are compatible with data available from current water quality databases. One of these methods developed by MacDonald et al (2000) is used in the analyses described in Section 6.4.

6.3.2 Effects in Marine/Estuarine Ecosystems

This section gives a brief overview of the information available for Pb in marine and estuarine systems. Most Pb in marine systems is in the inorganic form, complexed with chloride and carbonate ions. Increasing salinity increases the amount of Cl⁻ and CO₃²⁻ complexation and reduces concentration of free Pb²⁺ thereby producing compounds with lower bioavailability. There is less data available for the effects of Pb on saltwater organisms and ecosystems but studies indicate lower concentrations of Pb in oceans and large lakes. Toxicity data as expressed in both the AQWC guidelines (USEPA, 1993) and CD, AX7.2.2, indicate a much higher threshold for effects in saltwater environments.

6.3.2.1 Pathways of Exposure

Sources of Pb to marine and estuarine ecosystems include runoff from contaminated watersheds, direct atmospheric deposition and turnover of contaminated sediment in areas of high turbulence. Lead is primarily found in the open ocean in the dissolved form and is available in sediment in a variety of complexed forms. Lead concentrations in oceans were found to be much lower than those measured in freshwater lotic environments and studies with estuarine organisms have also shown reduced toxicity with increasing salinity, most likely due to increased complexation with Cl⁻ ions thereby reducing bioavailability. Studies in the Pacific Ocean near Hawaii have found concentrations of total Pb between 5-11 ng/kg (CD, Section 7.2.2).

6.3.2.2 Effects on Organisms and Communities

Hematological and neurological responses, including red blood cell destruction, enzyme inhibition and spinal curvature, were the most commonly reported effects in aquatic vertebrates. Demonstrated effects in invertebrates include alteration of reproduction rates and reduced growth. Studies with marine protozoa indicate that at water column concentrations of 0.02 to 1.0 mg Pb/L, abundance, biomass and diversity are reduced. In an estuarine community, Pb was found to affect species abundance when sediment concentrations reached 1343 mg/kg dry weight. Inhibition of embryo development in commercial shellfish has been documented at water concentrations of 50µg/L (CD, AX 7.2.4.3).
The toxicity of Pb in the marine or estuarine environment is highly dependent on salinity. A study of mysid shrimp reported a lethal concentration for 50% of the test organisms (LC$_{50}$) of 1140 µg/L at a salinity of 5% and an LC$_{50}$ of 4274 µg/L at 25% salinity. There is also some evidence of gender sensitivity in that male copepods were more sensitive to Pb in sediment than females. Smaller fish have been shown to be more sensitive than larger fish of the same species. Studies on invertebrates have also shown that deposit feeders were most affected by elevated substrate concentrations.

6.3.3 Effects in Freshwater Ecosystems

This section gives a brief overview of information available for Pb in freshwater systems. Most Pb in freshwater systems is in the inorganic form. Speciation is important in bioavailability and is dependent upon factors such as pH, temperature and water hardness. In freshwater, Pb typically forms strong inorganic complexes with OH$^-$ and CO$_3^{2-}$ and weak complexes with Cl$^-$. Organic Pb compounds in freshwater, which may increase bioavailability, arise from both natural and anthropogenic sources. Concentrations of various forms of organic Pb complexes are largely dependent on pH and water hardness.

6.3.3.1 Pathways of Exposure

The bioavailability and accessibility of Pb to aquatic organisms is determined largely by the species of Pb that forms in the ecosystem. In an acidic environment (pH<4) the ionic form, which is the more toxic form, of most metals generally predominates. As pH increases, carbonate, oxide, hydroxide, and sulfide complexes usually predominate and tend to be less toxic. Water hardness also influences toxicity by providing competition in the form of calcium and magnesium to Pb binding sites on biological membranes. Therefore, Pb is least toxic in neutral to basic pH levels and at increased water hardness. A further discussion of speciation and toxicity can be found in Section AX7.2.2.1 of the CD.

The US Geologic Service (USGS) has developed the National Water Quality Assessment (NAWQA) program which is a nation wide monitoring program that contains data on Pb concentrations in surface water, bulk sediment, and biological tissues for samples collected in many watersheds throughout the U.S. While the data are not representative of the entire U.S. and the analytical method employed for Pb was not as sophisticated as current methods, it is the most comprehensive national database available. The mean concentration of Pb in U.S. surface waters was 0.66µg/L (ranging from 0.04 to 30) and in bulk sediment was 120.11µg/g (ranging from 0.5 to 12,000) for data collected between 1991 and 2003 (CD, AX7.2.2.2 and Section 2.2).

6.3.3.2 Effects at an Ecosystem Level

The effects of Pb in aquatic ecosystems have not been well studied for areas affected by atmospheric deposition rather than point source pollution. Aquatic ecosystems near smelters,
mines and other industrial sources of Pb have demonstrated a wide variety of effects including reduced species diversity, abundance and richness; decreased primary productivity, and alteration of nutrient cycling. Apportioning these effects between Pb and other stressors is problematic since these point sources also emit a wide variety of other heavy metals which may cause toxic effects in aquatic systems.

Lead exposure may adversely affect organisms at different levels of organization, i.e., individual organisms, populations, communities, or ecosystems. Generally, however, there is insufficient information available for single contaminants in controlled studies to permit evaluation of specific impacts on higher levels of organization (beyond the individual organism). Potential effects at the population level or higher are, of necessity, extrapolated from individual level studies. Available population, community, or ecosystem level studies are typically conducted at sites that have been contaminated or adversely affected by multiple stressors (several chemicals alone or combined with physical or biological stressors). Therefore, the best documented links between Pb and effects on the environment are with effects on individual organisms.

However, several recent studies have attributed the presence of Pb to reduced primary productivity, increased respiration, and alterations of community structure. Specifically, dissolved Pb at concentrations from 6 to 80 mg/L (concentrations higher than those found in the NAWQA database) was found to reduce primary productivity and increase respiration in an algal community. Laboratory microcosm studies have indicated reduced species abundance and diversity in protozoan communities exposed to 0.02 to 1 mg Pb/L (CD, Section AX 7.2.5). Field studies have associated the presence or bioaccumulation of Pb with reductions in species abundance, richness, or diversity, particularly in sediment-dwelling communities (CD, Section AX7.2.5). Most of the available data for Pb effects in aquatic ecosystems comes from either laboratory studies which focused on only a few aspects of the natural system thereby neglecting some of the factors known to influence bioavailability of Pb or from complex natural systems with many stressors and various sources of anthropogenic Pb, particularly direct mining waste inputs (CD, AX7.2.5.2). Thus, the effects of atmospheric Pb on aquatic ecological condition remain to be defined.

There is a paucity of data in the general literature that explores the effects of Pb in conjunction with all or several of the various components of ecological condition as defined by the EPA (Young and Sanzone, 2002). Recent studies have attributed the presence of Pb to adverse effects on biotic conditions such as abundance, diversity, reduced primary productivity, and alteration of community structure (CD, Section 7.2.5). It is difficult to apportion effects between Pb and other stressors, however, and these studies did not generally account for modifying factors that may mediate or exacerbate Pb effects.
Lead concentrations in sediment vary with depth and are attributable to increased anthropogenic inputs over the last few decades. Several studies have been undertaken to identify regional sources of Pb in eastern North America and the Great Lakes and have found positive correlations between Pb isotope ratios in the Great Lakes and known aerosol emissions from current and historic industrial sources in Canada and the U.S. These studies seem to indicate that current emissions are contributing somewhat to Pb in sediments (CD, AX7.2.2.3). Resuspension of historically deposited Pb in sediments may also constitute a source of Pb in some systems for the foreseeable future (CD, AX 7.2.2.3).

6.3.3.3 Effects on Algae and Aquatic Plants

As primary producers in aquatic systems, algae and aquatic plants are vital to ecosystem function and provide the foundation upon which the food web depends. Therefore impacts to these organisms can create a chain of effects that impacts the entire ecosystem. Algae and aquatic plants are exposed to Pb by either uptake from the water column or sediment. Pb is most bioavailable in the divalent form (Pb²⁺) and as such is adsorbed onto cell walls and accumulates in the cell wall or surface of the plasma membrane of aquatic plants and algae (CD, AX7.2.3.1). Bioconcentration of Pb, the accumulation of Pb inside an organism, may be quite high for both algae and aquatic plants and have made them effective in the remediation of contaminated areas. In aquatic plants as in terrestrial plants, Pb tends to be sequestered (bound and stored) in roots much more than in shoots although some wetland plants have been found to accumulate high levels of Pb in shoots as well. Within the plants the sequestered Pb tends to be metabolically unavailable until a certain concentration is reached which appears to be species specific.

Growth inhibition is exhibited by algae and aquatic plants over a broad range of Pb concentrations in water (1000 to >100,000 µg/L) due in part, to the interaction between various biochemical factors and the bioavailability of Pb to these organisms (CD, AX7.2.3.1). Clinical signs of Pb toxicity in algae include deformation and disintegration of cells, shortened exponential growth phase, and inhibition of pigment synthesis which may ultimately lead to cell death. As reported in the CD (Section AX7.2.3.1), studies have shown growth inhibition of Closterium acerosum, a freshwater algae, at concentrations of 1,000 µg/L Pb nitrate exposure and an effects concentration for 50% of the test population (EC₅₀) for growth inhibition of Scenedesmus quadricauda has been reported at 13,180µg/L. Other species of algae such as Synechococcus aeruginosus were much more tolerant and required concentrations in excess of 82,000 µg/L to elicit significant growth inhibition. In aquatic plants, toxicity studies have focused on the effects of Pb on plant growth, chlorophyll concentration and protein content. An EC₅₀ of 1,100 µg/L was reported for growth inhibition for Azolla pinnata, an aquatic fern, when exposed to Pb-nitrate for 4 days. Studies with duckweed, Lemna gibba, have reported an EC₅₀ of 3,750µg/L under the same conditions. These studies indicate the possibility of adverse impacts
to algae and aquatic plants at concentrations which may be found in the vicinity of direct
discharges from point sources but which would not be expected from ambient deposition.

There are two main mechanisms by which algae and plants may moderate Pb toxicity:
sequestration in roots or cell walls, and production of enzymes which complex Pb to make it
metabolically inactive. Studies have shown phytochelatins, polypeptides which chelate heavy
metal ions and make them biologically unavailable to the organism, may be synthesized in
response to exposure to heavy metals (CD, AX7.1.2.4).

6.3.3.4 Effects on Invertebrates

Aquatic invertebrates serve an important role in aquatic ecosystems as both consumers of
detrital material and as a prey source for many other organisms. Therefore, adverse impacts to
invertebrates can dramatically alter or reduce ecosystem function. Invertebrates may accumulate
Pb in tissue through ingestion of food and water and adsorption from water. Dietary Pb may
contribute significantly to the chronic toxicity of Pb through ingestion of food which has
accumulated Pb or by incidental ingestion of sediments. Studies which relate the effects of
dietary exposure and toxic effects in aquatic systems are rare; however, it may be assumed that
both dietary and waterborne exposures are important to overall Pb toxicity (CD, AX7.2.4.3).

Exposure to Pb can result in reduction of growth rates and reproductive rates as well as
cause increased mortality. As discussed in Section 6.3.2.1 of this document, both acute and
chronic toxicity of Pb can be significantly influenced by water hardness and pH. A study by
Borgmann et.al (2005) with *Hyalella azteca*, a freshwater amphipod, showed a 23-fold increase
in acute toxicity in soft water (18 mg CaCO$_3$/L) compared to hard water (124 mg CaCO$_3$/L).
The influence of pH on Pb toxicity varies between invertebrate species. Studies have reported
increasing mortality with decreasing pH in some bivalves, cladocerans, amphipods, gastropods
and mayflies while some crustaceans and gastropods have shown no relationship between pH
and mortality under identical conditions. For the amphipod *H. azteca*, the low-observed effect
concentration (LOEC) for survival in hard water at pH 8.27 was 192µg/L as dissolved Pb and
466µg/L as total Pb leading to the conclusion that both waterborne and dietary Pb contributed to
this reduced survival (CD, AX7.2.4.3). Overall, adverse effects for the most sensitive
invertebrates studied, amphipods and waterfleas, occurred at concentrations ranging from 0.45 to
8,000µg/L. Exposures to Pb in sediment can also produce toxic effects in sediment dwelling
invertebrates. Acute effects in the water flea, *Daphnia magna*, included reduced mobility after
exposure to 7,000 mg Pb/kg dw for 48 hours while chronic exposure of midges to sediments
containing 31,900 mg Pb/kg dw resulted in 100% mortality over 14 days (CD, AX7.2.4.3).
Overall, based on recorded Pb concentrations in the NAWQA database, there are some surface
waters and sediments in the U.S where effects on sensitive invertebrates would be expected but
apportioning these concentrations between air and non-air sources has not been done.
There are several mechanisms by which invertebrates detoxify Pb. Lead may be concentrated in some invertebrates by formation of granules which may be eventually excreted, sequestered within the exoskeleton and glandular cells, or bound to membranes in gills and other tissues. Avoidance behaviors have been documented for the aquatic snail, *Physella columbiana*, but few studies were found that reported avoidance behaviors in invertebrates. As neurological and behavioral effects may be important in determining the adverse effects of Pb, further research is needed in this area.

### 6.3.3.5 Effects on Fish and Waterfowl

Both the ingestion of contaminated sediment and prey items as well as direct absorption from water contributes to fish exposures to Pb. Dietary effects of Pb are not well studied in fish but evidence supports that higher tissue concentrations have been found in fish with direct contact with sediment. Gale et al. (2002) found a good correlation between sediment concentration and tissue concentrations in suckers and small sunfish, which feed directly from the sediment, but not in smallmouth bass, which feed at a higher trophic level. Bioconcentration does occur in freshwater fish and bioconcentration factors (BCFs) for brook trout and bluegill of 42 and 45, respectively, have been reported (CD, AX7.2.3.1). Studies have also shown that fish accumulate Pb more rapidly in low pH environments and when diets are calcium deficient.

Lead has been observed to have adverse effects on the production of some enzymes which affect locomotor function as well as adverse blood chemistry effects in some fish. Symptoms of Pb toxicity in fish include the production of excess mucous, spinal deformity, anemia, darkening of the dorsal region, degeneration of the caudal fin, destruction of spinal neurons, enzyme inhibition, growth inhibition, renal pathology, reproductive effects, and mortality (CD, AX7.2.4.3). As in other organisms, Pb speciation, water pH and water hardness play an important role in the toxicity of Pb. Spinal deformities were found to occur at much lower Pb concentrations in soft water than in hard water. Maximum acceptable threshold concentrations (MATC), the maximum concentrations at which no adverse effects were seen, have been reported (CD, AX7.2.4.3) for rainbow and brook trout in soft water as 4.1 to 7.6 µg/L Pb and 58 to 118 µg/L Pb respectively. A LC$_{50}$ of 810 µg/L was found using fathead minnows at a pH of 6-6.5 while at the same water hardness the LC$_{50}$ was >5,400 µg/L at a pH range of 7 – 8.5. Other studies have shown alterations in blood chemistry in fish from chronic and acute exposures ranging from 100 to 10,000 µg/L Pb (CD, Section AX8.2.3.3). Therefore, given the concentrations of Pb found in surface waters in the NAWQA database, there are likely adverse effects to fish populations in some locations of the U.S. It is not clear what the ambient air contributions of Pb are at these locations.

There are several physiological and behavioral mechanisms by which fish reduce exposure and absorption of Pb. While the avoidance response to Pb in fish has not been well
studied, it is known for other metals and is thought likely for Pb (CD, AX7.2.3.2). As in other organisms, gender and age are important variables in determining the adverse effects of Pb with females and young fish being more sensitive to Pb.

Incidental ingestion of contaminated sediment is the primary route of exposure for waterfowl. Studies by Beyer et al. (2000) in the Coeur d’Alene watershed near mining and smelting activity have shown a range of effects for waterfowl based on sediment concentrations and corresponding blood Pb levels. This study suggested that a NOAEL blood concentration of 0.20 mg/kg wet weight Pb corresponded to a sediment concentration of 24 mg/kg Pb. Sub-clinical poisoning (LOEL) occurred in swans when sediment concentration was 530 mg/kg Pb which corresponded to a 0.68 mg/kg blood Pb level. Some mortality may occur with sediment concentrations as low as 1800 mg/kg Pb and an LC50 was found in swans at 3,600 mg/kg Pb in sediment. While these values are somewhat site specific and are dependent on the bioavailability of the Pb as well as the overall health and diet of the animals, the correlation between blood Pb levels and effects should be applicable irrespective of location-specific variables. Given current concentrations of Pb in sediment, it is likely that some adverse effects are occurring in waterfowl exposed to point sources of Pb, whether through deposition or direct discharge.

6.3.4 Summary

Lead exists in the aquatic environment in various forms and under various chemical and physical parameters which determine the ability of Pb to cause adverse effects either from dissolved Pb in the water column or Pb in sediment. Current levels of Pb in water and sediment also vary widely depending on the source of Pb. Conditions exist in which adverse effects to organisms and thereby ecosystems may be anticipated given experimental results. It is unlikely that dissolved Pb in surface water constitutes a threat to ecosystems that are not directly influenced by point sources. For Pb in sediment, the evidence is less clear. It is likely that some areas with long term historical deposition of Pb to sediment from a variety of sources as well as areas influenced by point sources have the potential for adverse effects to aquatic communities. The long residence time of Pb in sediment and its ability to be resuspended by turbulence make Pb likely to be a factor for the foreseeable future. Criteria have been developed to indicate concentrations of Pb in water and sediment below which no adverse effects are expected to aquatic organisms. These values may be used to identify areas in which there is the potential for adverse effects to receptors based on current concentrations of Pb in water and sediment.

6.4 SCREENING LEVEL RISK ASSESSMENT

6.4.1 Overview of Analyses

The ecological risk assessment consisted of case studies and a national scale surface water and sediment screening assessment. In this analysis, activities for an additional case study
(ecologically vulnerable location) focused on identification and description and did not include risk analyses. The case study analyses were designed to estimate the potential for ecological risks associated with exposures to Pb emitted into ambient air for three case studies: primary Pb smelter, secondary Pb smelter, and near roadway non-urban location. Soil, surface water, and/or sediment concentrations, as available, were compared to ecological screening benchmarks to assess the potential for ecological impacts from NAAQS-relevant sources. Figure 6-1 provides an overview of the analyses undertaken for this assessment. These results are not definitive estimates of risk, but rather intended to focus attention on those locations at which there is the greatest likelihood for adverse effect. The national-level screening assessment evaluated the potential for ecological risks associated with the atmospheric deposition of Pb released into ambient air at surface water and sediment monitoring locations across the United States.

This overview shows the key types of information and models involved in each phase of the analysis and how they are related to each other. Table 6-1 summarizes the use of these information types and models for each case study. As indicated in these exhibits, the specific approach for each case study differed based on the nature of the case study (e.g., type of source, land use) and the site-specific measurements available. In cases where the available measurements were not sufficient to characterize the study area (e.g., due to insufficient spatial coverage), these data were used for performance evaluation.
Figure 6-1. Overview of Ecological Screening Assessment.
Table 6-1. Models and Measurements Used for Ecological Risk Screening Assessment.

<table>
<thead>
<tr>
<th>Location</th>
<th>Primary Pb Smelter</th>
<th>Secondary Pb Smelter</th>
<th>Near Roadway Non-Urban</th>
<th>National-Scale Aquatic Screen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Missouri</td>
<td>Missouri</td>
<td>Alabama</td>
<td>Corpus Christi, Texas</td>
<td>Surface water bodies in the U.S.</td>
</tr>
<tr>
<td>Atlee, Virginia</td>
<td></td>
<td></td>
<td>140-m section of road; 2 - 30 m from road</td>
<td></td>
</tr>
</tbody>
</table>

- **Spatial extent and resolution**
  - Missouri: Approximately 6km diameter, centered on point source
  - Alabama: U.S. Census blocks
  - Corpus Christi: single transect perpendicular to road; 0.5 - 4 m from road
  - Atlee: 140-m section of road; 2 - 30 m from road

### Exposure Assessment: Estimating Media Concentrations

<table>
<thead>
<tr>
<th>Deposition to soil</th>
<th>Models</th>
<th>Measurements</th>
<th>Exposure Assessment: Estimating Media Concentrations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n/a</td>
<td>AERMOD&lt;sup&gt;a&lt;/sup&gt;</td>
<td>n/a</td>
</tr>
<tr>
<td>Measurements</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
</tr>
</tbody>
</table>

- **Soil conc.**
  - Models: n/a
  - Measurements: Site-specific conc. of total Pb in soil samples (26 locations)
  - U.S. Census blocks

- **Surface water conc.**
  - Models: n/a
  - Measurements: Site-specific conc. of dissolved Pb in water column samples from eight water bodies/drainage areas (30 locations)

- **Sediment conc.**
  - Models: n/a
  - Measurements: Site-specific conc. of total Pb in sediment samples from five water bodies/drainage areas (69 sites)

### Ecological Risk Assessment

<table>
<thead>
<tr>
<th>Soil</th>
<th>Soil screening values</th>
<th>n/a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Freshwater – water column</td>
<td>U.S. EPA Pb freshwater AWQC for aquatic life derived based on site-specific measured water hardness (conc. of CaCO&lt;sub&gt;3&lt;/sub&gt;)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>n/a</td>
</tr>
<tr>
<td>Freshwater – sediment</td>
<td>Sediment screening values based on MacDonald et al. (2000) sediment quality assessment guidelines</td>
<td>n/a</td>
</tr>
</tbody>
</table>

- <sup>a</sup> American Meteorological Society/EPA Regulatory Model (AERMOD) (USEPA, 2004)
- <sup>b</sup> Multiple Pathways of Exposure (MPE) (USEPA, 1998)
- <sup>c</sup> These screening values are based on measured ecotoxicity data
6.4.2 Measures of Exposure and Effect

The measures of exposure for these analyses are total Pb concentrations in soil, dissolved Pb concentrations in freshwater surface waters (water column), and total Pb concentrations in freshwater sediments. These exposure concentrations were estimated for the three case studies and the national-scale screening analyses as described below:

- For the primary Pb smelter case study, measured concentrations of total Pb in soil, dissolved Pb in surface waters, and total Pb in sediment were used to develop point estimates for sampling clusters thought to be associated with atmospheric Pb deposition, rather than Pb associated with non-air sources, such as runoff from waste storage piles.

- For the secondary Pb smelter case study, concentrations of Pb in soil were estimated using fate and transport modeling based on EPA’s MPE methodology (USEPA, 1998) and data available from similar locations.

- For the near roadway non-urban case study, measured soil concentration data collected from two interstate sampling locations, one with fairly high-density development (Corpus Christi, Texas) and another with medium-density development (Atlee, Virginia), were used to develop point estimates of Pb contaminated soils from historically-deposited Pb for each location.

- The national-level surface water and sediment screening analyses compiled measurements of dissolved Pb concentrations in surface water and total Pb in sediment for locations across the United States from available databases. Emissions, point discharge, and land use data for the areas surrounding these locations were assessed to identify locations where atmospheric Pb deposition may be expected to contribute to potential ecological impacts. The exposure assessment focused on these locations.

The Hazard Quotient (HQ) approach was used to compare estimated media concentrations with ecotoxicity screening values in soils, surface waters, and sediments around the primary Pb smelter, for soils only around the secondary Pb smelter case study location and the near roadway non-urban case study locations, and for surface water and sediment in the national-level screen. The HQ is calculated as the ratio of the media concentration to the ecotoxicity screening value. The HQ is represented by the following equation:

\[
HQ = \frac{\text{estimated media concentration}}{\text{ecotoxicity screening value}}
\]

For each case study, HQ values were calculated for each location where either modeled or measured media concentrations were available. Separate soil HQ values were calculated for each ecological receptor group for which an ecotoxicity screening value has been developed (i.e.,
birds, mammals, soil invertebrates, and plants). HQ values less than 1.0 suggest that Pb concentrations in a specific medium are unlikely to pose significant risks to ecological receptors whereas HQ values greater than 1.0 indicate that the expected exposure exceeds the ecotoxicity screening value.

6.4.3 National-Scale Screen and Case Studies

This section provides an overview of the study locations selected for ecological screening risk assessment performed in support of the NAAQS review. A national scale screen was conducted to look at current Pb concentrations in freshwaters and sediments throughout the U.S. and three case study locations were selected: 1) primary Pb smelter, 2) secondary Pb smelter and 3) near roadway non-urban. The primary and secondary smelter case studies represent an extreme and moderate point source scenario while the near roadway non-urban location represents a more ubiquitous exposure from historic gasoline Pb emissions along major roadways.

6.4.3.1 National Scale Screen

A national scale assessment was performed using the NAWQA database to identify locations in the U.S. in which concentrations of Pb in surface water and/or sediment exceed established screening values and for which ambient air Pb is likely to be a major factor. These locations were identified using the methodology described below and in the risk assessment report (ICF, 2006).

6.4.3.1.1 Fresh Surface Waters

A screening-level ecological risk assessment for aquatic ecosystems was conducted for Pb concentrations in fresh surface waters of the United States to identify areas in which there are concentrations in excess of EPA recommended national ambient water quality criteria (AWQC), both chronic and acute, for the protection of freshwater aquatic life. In this assessment, we identified locations at which Pb concentrations exceeded the EPA AWQC and for which air sources are likely to be the major contributor to the Pb concentrations in the water (i.e., there are no other obvious sources of Pb to the water).

As the geographic coverage achieved in this surface water screen is based entirely on the geographic coverage of available measurements of dissolved Pb in the selected database, it was important that the most appropriate dataset be used. It was concluded that of the commonly available databases, including NWIS, STORET and NAWQA, the NAWQA data set is most appropriate for a nationwide aquatic risk screen for several reasons. The inclusion of dissolved Pb as an analyte is limited in all of the databases (total Pb is measured more often). None of the databases provide the co-located measurements of water hardness in the same records as the measurements of dissolved Pb. STORET and NWIS include samples from more locations in the
United States than does the NAWQA data set, but the sampling and reporting protocols represented in STORET and NWIS are less consistent from site to site. Data for dissolved Pb in NWIS are predominantly from the 1980s, and therefore do not represent current conditions. The NAWQA data set, on the other hand, provides representative (though not complete) coverage of the United States, with samples through 2004 included in the database. The NAWQA data set also provides a consistent approach to sampling and analysis of the elements using consistent quantitation limits across the country. Given the sampling design for NAWQA and the consistency of the data across the country, it is considered to be more appropriate for a national-level aquatic risk screen than the other two data sets and was therefore used for this screen.

6.4.3.1.2 Lead in Sediments

Possible risks to sediment dwelling organisms were also examined at locations identified in the surface water screen by comparing total Pb concentrations in sediments to ecotoxicity benchmarks for sediments, generally referred to as sediment quality criteria. The preferred approach for sediment data was to obtain it from surface water sampling locations in the NAWQA database. It was not always possible to sample sediments at locations where surface water samples are desired. Therefore, some of the sites of interest do not have sediment samples available from the same location. Where an exact match was not found, a nearby sampling location was identified on the basis of latitude, and longitude, and name of the site location.

6.4.3.2 Ecologically Vulnerable Location

A literature search was conducted to identify an acidified forest or non-urban acidified watershed ecosystem to which the following criteria could be applied:

- Potential for increased bioavailability of Pb due to soil and water acidification;
- Relatively distant from point sources of Pb emissions;
- Relatively high elevation which may be subject to comparatively higher deposition of Pb due to wind speed and precipitation as well as longer residence time; and
- Availability of data on trends (temporal, elevation, etc.) of Pb concentration in various environmental media.

Based on these criteria, we selected the Hubbard Brook Experimental Forest (HBEF) in the White Mountains of New Hampshire for the ecologically vulnerable case study. The HBEF was established by the USDA Forest Service in 1955 as a major center for hydrologic research in New England. The USDA proposed to use the small watershed approach at Hubbard Brook to study linkages between hydrologic and nutrient flux and cycling in response to natural and human disturbances, such as air pollution, forest cutting, land-use changes, increases in insect populations and climatic factors. The first grant was awarded by the National Science Foundation (NSF) to Bormann and Likens in 1963 to support the research at the HBEF. Since
that time there has been continuous support from the NSF and the U.S. Department of Agriculture (USDA) Forest Service. In 1988 the HBEF was designated as a Long-Term Ecological Research (LTER) site by the NSF. On-going cooperative efforts among diverse educational institutions, private institutions, government agencies, foundations and corporations have resulted in one of the most extensive and longest continuous data bases on the hydrology, biology, geology and chemistry of natural ecosystems in the United States. This historical record makes HBEF uniquely suited to the purpose of this review. As discussed earlier, an assessment of Pb related ecological risks for this case study location is not presented in the first draft of this document. Full development of this case study location including discussion of ecological exposures and risk would be a useful enhancement for any future analyses.

6.4.3.3 Primary Pb Smelter

The primary Pb smelter case study location is one of the largest primary Pb smelters in the world, is the only remaining operating Pb smelter in the United States, and is also the longest operating smelter in the world, sustaining nearly continuous operation since 1892. Further information on the surroundings and demographics in the vicinity of the primary smelter can be found in the risk report (ICF, 2006). Portions of this study area comprise an active Superfund site and are subject to ongoing evaluation under the Superfund program administered by the Office of Solid Waste and Emergency Response. Methods used in conducting ecological risk assessment for the analysis have been selected to address policy questions relevant to the Pb NAAQS review and consequently, may differ from those used by the Superfund program.

The primary Pb smelter property is bordered on the east by the Mississippi River, on the southeast by Joachim Creek, on the west and north-northwest by residential areas, and on the south-southwest by a slag pile. A large part of the slag pile is located in the floodplain wetlands of Joachim Creek and the Mississippi River.

Ecological features near the facility include the Mississippi River, streams, emergent and scrub-shrub wetlands, and successional and mature bottomland hardwood forest tracts (ELM, 2005). Bottomland hardwood forests and agricultural fields are present to the west, south, and east of the characterization area between the smelter’s slag storage area and Joachim Creek. The most mature bottomland hardwood forest is adjacent to Joachim Creek. Immediately south of the facility is a mixture of floodplain forest, emergent marsh, and scrub-shrub wetland habitat that is populated by willow trees. Throughout much of the year, migratory birds such as the red-tailed hawk, belted kingfisher, and great blue herons utilize the habitat near the facility. The federally threatened bald eagle has been spotted on-site at the facility, which is known to be within the habitat for the bird. The facility is also within the habitat of the Indiana bat, which is on the federal and state endangered species lists. In addition, the state and federally endangered pallid sturgeon has been identified in the Mississippi River adjacent to and downstream of the
facility. The pink mucket, scaleshell, and gray bat also occur in Jefferson County and are on both the state and federal endangered lists.

6.4.3.4 Secondary Pb Smelter

The secondary Pb smelter location falls within the Alabama Coastal Plain in Pike County, Alabama. It is located in an area of disturbed forests, and is less than 2 km from Big Creek, which is part of the Pea River watershed. Big Creek is located approximately 0.5 m south southeast from the center of the facility. The surrounding area includes emergent and scrub shrub wetlands, forests, freshwater creeks, ponds, rivers, croplands, pastureland, and developed urban areas. The Pea River watershed drains into the Gulf of Mexico. The watershed is underlain by coastal plain sediments, including sand, clay, and limestone; and the topography can be characterized as gentle to moderate rolling hills (CPYRWMA 2006). Diversity of terrestrial and aquatic animal species is relatively high. The Choctawhatee and Pea River basins, in which the secondary Pb smelter is located, contain 43 species of marine, estuarine, and freshwater fish species (Cook and Kopaska-Merkel, 1996). Anadromous fish species (i.e., saltwater fish that must spawn in freshwater) found in the Pea River basin include the following: the threatened Gulf sturgeon, Alabama shad, striped bass, and skipjack herring. The Pea River basin also provides habitat for 20 species of freshwater mussels (Cook and Kopaska-Merkel, 1996), as well as numerous species of snails, snakes, and other invertebrates. Terrestrial species supported in this region include a variety of birds, mammals, invertebrates, and vascular plants. Other terrestrial fauna found in the region include migratory birds, small mammals and invertebrate species. A total of 34 vascular flora from Pike County are listed by the Alabama Natural Heritage Inventory Program as state endangered, threatened, or of special concern (Alabama Natural Heritage Inventory 2001). According to NatureServe and the U.S. Fish and Wildlife Service (USFWS), no species in Pike County are on the federal endangered species list (Outdoor Alabama, 2003). A few species, however, are candidates for the federal list.

6.4.3.5 Near Roadway Non-Urban Case Study

The Houston, Texas, near roadway urban case study for the human health risk assessment used surrogate soil Pb concentration data measured at a sampling location in downtown Corpus Christi, Texas (Turer and Maynard, 2003). Air concentration data are not needed for this assessment of ecological risks therefore a search for a more ecologically-relevant case study location was conducted. Non-urban case study locations that provide soil concentration data were sought with the expectation that ecological receptors would be more likely to occur along roads in less developed areas compared to the downtown location evaluated in the human health risk assessment. Terrestrial wildlife could forage in Pb contaminated soils alongside highways, particular on roads traversing undeveloped areas.
From the literature search for studies of Pb in near roadway soils, two non-urban sites for which soil Pb levels are available were identified for use in the ecological risk assessment. These locations are: (1) Interstate 37 near oil refineries in Corpus Christi, Texas (Turer and Maynard, 2003) and (2) Interstate 95 in Atlee, Virginia, which connects to a moderately traveled, two-lane road (Speiran, 1998).

Land cover data from 1992 within 1 m of the Corpus Christi, Texas study location showed 59 percent industrial, 10 percent low intensity residential, and 25 percent high intensity residential (Vogelmann et al., 2001). The remaining 5 percent of the surrounding area includes shrubland, row crops, pasture, grasses, and forested upland, including evergreen forest and deciduous forest.

The 1992 land cover data within 1 m of the Atlee, Virginia, study locations showed 26 percent developed: 2 percent low-intensity residential and commercial and 24 percent industrial and transportation. The remaining 74 percent included 25 percent deciduous forest, 14 percent woody wetlands, and 12 percent pasture (Vogelmann et al., 2001). Smaller proportions of mixed forest, evergreen forest, row crops, and transitional (barren) areas were also found.

### 6.4.4 Screening Values

The following is a discussion of specific ecological screening values selected for use in the risk assessment. The main discussion of the development and derivation of these tools can be found in Section 6.2.3 of this document and in the risk report (ICF, 2006). This discussion outlines the ways in which the tools were used for this assessment to identify potential effect from Pb exposure to specific ecological endpoints in either localized case studies or in the NAWQA monitoring database.

#### 6.4.4.1 Soil Screening Values

In developing soil screening values for use in this assessment, assumptions inherent in the derivation of the Superfund Eco-SSLs were examined, and as appropriate, augmented or replaced with current species-specific information. For example, the assumptions employed for deriving the Eco-SSLs for avian and mammalian wildlife from the corresponding TRVs were examined (ICF, 2006). Soil screening values were derived for this assessment using the Eco-SSL methodology (described in Section 6.2.3) with the TRVs for Pb (USEPA, 2005b) and consideration of the inputs on diet composition, food intake rates, incidental soil ingestion, and contaminant uptake by prey. The soil screening values shown in Table 6-2 for plants and soil invertebrates are the Eco-SSL values (USEPA, 2005a) while the screening values for birds and mammals are based on the Eco-SSL methodology but with modified inputs specific to this assessment (ICF, 2006).
Table 6-2. Soil Screening Values for Pb for Ecological Receptors

<table>
<thead>
<tr>
<th>Ecological Receptor</th>
<th>Soil Concentration (mg Pb/kg soil dry weight)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plants ¹</td>
<td>120</td>
</tr>
<tr>
<td>Soil Invertebrates ¹</td>
<td>1700</td>
</tr>
<tr>
<td>Birds ²</td>
<td>38</td>
</tr>
<tr>
<td>Mammals ²</td>
<td>112</td>
</tr>
</tbody>
</table>

¹ Values obtained from *Ecological Soil Screening Levels for Lead, Interim Final* (USEPA, 2005a).
² Values obtained by refinement described in risk report (ICF, 2006).

6.4.4.2 Surface Water Screening Values

Specific screening values were calculated using the AWQC developed by EPA (1984) for the primary smelter case study location and the national scale screen based on site-specific water hardness data. AWQC values for chronic exposures are called the criterion continuous concentration (CCC) and for acute exposures are called the criterion maximum concentration (CMC), and they are available for freshwater and marine environments. For a CCC to be exceeded, a 4-day average water concentration must exceed the CCC more than once every three years (USEPA, 1984).

6.4.4.3 Sediment Screening Values

This risk screen uses sediment criteria developed by MacDonald et al (2000) which focuses on total Pb concentrations in sediment and identifies a threshold-effect concentration (TEC) and a probable-effect concentration (PEC) as 35.8 mg/kg and 128 mg/kg respectively. This methodology is described more fully in the risk assessment report (ICF, 2006).

6.4.5 Results for Case Study Locations and Comparison to Screening Value

To identify locations in which Pb concentrations in soil, water and/or sediment may potentially be harmful, each case study location was assessed using either empirical data or model results for each media. These concentrations were then compared to screening values as described in Section 6.4.2. The HQ approach was then used to compare estimated exposures for geographic areas around the case study sites with ecotoxicity benchmark values in each of three media; soil, surface water, and sediment. HQs less than or equal to one suggest that ecological risks are negligible. HQs greater than one indicate a potential for adverse effects and a more refined analysis of sensitive receptors may be needed.

6.4.5.1 National-Scale Surface Water Screen

Based on EPA’s re-evaluation of AWQC for metals (USEPA, 1993), the CCC for relatively soft water (50 mg/L CaCO₃) is 1.2 µg/L while increasing hardness resulted in higher
CCC values. Therefore, the initial screen of dissolved Pb concentrations in surface water looked for measurements equal to or greater than $1.2\mu g/L$. This resulted in 42 sampling locations for which one or more measurements exceeded that screening value. Data for each measurement of dissolved Pb at these stations are provided in the risk assessment report (ICF, 2006b). For a definitive risk assessment, representing a given location with a single sampling measurement of dissolved Pb would not be considered. However, for purposes of this risk screen, given the limited analyses for dissolved Pb, all 42 sampling locations were retained for analysis. Next, the location-specific CCC and CMC values were determined based on water hardness for those locations. A review of the data on water hardness in the NAWQA data set for 1994 to 2004 indicated that the initial screening value of $1.2\mu g/L$ was too high to identify all locations for which dissolved Pb concentrations exceeded the CCC for the protection of aquatic life. Many waters in the United States are softer than anticipated (i.e., measured CaCO$_3$ concentrations down to 1 mg/L).

A second screen was therefore conducted in which dissolved Pb measurements greater than the quantitation limit (QL) but less than $1.2\mu g/L$ were reviewed. In the second screen, for each sampling location with one or more measured dissolved Pb concentrations above the QL but less than $1.2\mu g/L$, collocated measurements of CaCO$_3$ were used to calculate a site-specific CCC as described above. To attempt to isolate those locations where air derived Pb is the major source of Pb to water; land use data was obtained from NAWQA for each location in which the derived HQ was greater than 1.0. The available categories of land use in the dataset separated mining sites but did not separate other activities which are likely to produce Pb (e.g. smelting sites were included in the industrial category). While it is likely that mining activities do produce air emissions of Pb (Section 2.3.4), the data is lacking to apportion air and non-air Pb at mining sites. Therefore, results for locations with mining as the land use category were separated from the other land use types. Table 6-2 summarizes the HQs for the 15 non-mining sites for which the chronic HQs exceed 1.0 in order of increasing HQ. These locations are in areas classified in the NAWQA database as urban and mixed, but also include forest, rangeland, and a “reference” site in Alaska. The highest HQ is for the Alaska reference site and is based on one measurement of dissolved Pb and one measurement of calcium carbonate. Thus, the uncertainty associated with this HQ is high (ICF, 2006).
Table 6-3. Results of Aquatic Risk Screen - Locations at which Dissolved Pb Measurements Exceed AWQC, Excluding Mining Sites. 

<table>
<thead>
<tr>
<th>Basin ID</th>
<th>State</th>
<th>Station ID</th>
<th>Land Use</th>
<th>Lead CCC (ug/L)</th>
<th>Pb Measurements</th>
<th>Hazard Quotient</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No. &gt; CCC / Total N</td>
<td>No. &lt; QL, which is &gt; CCC</td>
</tr>
<tr>
<td>45</td>
<td>NM</td>
<td>8331000</td>
<td>Mixed</td>
<td>2.9</td>
<td>1/12</td>
<td>0</td>
</tr>
<tr>
<td>44</td>
<td>CO</td>
<td>3.85E+14</td>
<td>Other/Mixed</td>
<td>0.89</td>
<td>1/4</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>CT</td>
<td>1127000</td>
<td>Mixed</td>
<td>0.36</td>
<td>3/22</td>
<td>14</td>
</tr>
<tr>
<td>46</td>
<td>WA</td>
<td>12422500</td>
<td>Urban</td>
<td>0.99</td>
<td>4/28</td>
<td>24</td>
</tr>
<tr>
<td>46</td>
<td>WA</td>
<td>12422000</td>
<td>Urban</td>
<td>0.99</td>
<td>2/20</td>
<td>18</td>
</tr>
<tr>
<td>46</td>
<td>ID</td>
<td>12392155</td>
<td>Forest</td>
<td>0.17</td>
<td>4/17</td>
<td>10</td>
</tr>
<tr>
<td>47</td>
<td>UT</td>
<td>4.05E+14</td>
<td>Rangeland</td>
<td>5.8</td>
<td>1/2</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>CT</td>
<td>1119375</td>
<td>Mixed</td>
<td>0.18</td>
<td>5/20</td>
<td>13</td>
</tr>
<tr>
<td>31</td>
<td>MO</td>
<td>7018100</td>
<td>Forest</td>
<td>0.33</td>
<td>1/2</td>
<td>0</td>
</tr>
<tr>
<td>58</td>
<td>HA</td>
<td>16212700</td>
<td>Mixed</td>
<td>0.17</td>
<td>1/2</td>
<td>1</td>
</tr>
<tr>
<td>1</td>
<td>RI</td>
<td>1112900</td>
<td>Mixed</td>
<td>0.44</td>
<td>3/3</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>CT</td>
<td>1124000</td>
<td>Mixed</td>
<td>0.30</td>
<td>11/23</td>
<td>9</td>
</tr>
<tr>
<td>46</td>
<td>ID</td>
<td>12419000</td>
<td>Mixed</td>
<td>0.37</td>
<td>2/26</td>
<td>16</td>
</tr>
<tr>
<td>31</td>
<td>AR</td>
<td>7050500</td>
<td>Mixed</td>
<td>2.6</td>
<td>1/8</td>
<td>0</td>
</tr>
<tr>
<td>31</td>
<td>AK</td>
<td>6.01E+14</td>
<td>Reference</td>
<td>0.11</td>
<td>1/1</td>
<td>0</td>
</tr>
</tbody>
</table>

* In order of increasing Hazard Quotient for the CCC aquatic toxicity benchmark. Additional information characterizing these locations is provided in the risk report (ICF, 2006).

When the 15 sampling locations in Table 6-2 are compared to NEI data, only three appear to be near facilities emitting relatively large quantities of Pb to the atmosphere (i.e., more than 1 ton per year): one is in Oahu, Hawaii, one in Jewett City, Connecticut, and one in Manville, Rhode Island. An additional two sampling locations appear to be within 50 km of facilities emitting relatively large quantities of Pb, both in Connecticut; however, whether these facilities are close enough to influence the Pb concentrations in the water column at these sampling sites is unknown. Of the three sampling locations within 20 km of facilities emitting more than 1 ton of Pb per year, the location in Rhode Island might also be receiving a large part of its Pb from upstream discharges from metal ore processing facilities (i.e., there are six such discharges out of 14 National Pollutant Discharge Elimination System (NPDES) permitted facilities upstream of this sampling location). More information on emissions for these 15 locations can be found in the risk report (ICF, 2006).

6.4.5.2 National-Scale Sediment Screen

Sediment characterization for the 15 sites identified in the AWQC screen was performed using the hazard quotient method, where measures of total Pb concentrations in sediments were compared with the sediment TEC and PEC values for the protection of sediment dwelling...
organisms. The first step involved attempting to find matching sediment sampling locations in the NAWQA database. It was not always possible to find collocated sediment and surface water samples. It was expected, therefore, that some of the 15 sites of interest would not have sediment samples available from the same location. Where an exact match was not found, a nearby sampling location on the same water body was identified.

Table 6-4 shows the HQs for measured total Pb concentrations in sediments at 12 of the 15 surface water locations for which data were available. The HQs are calculated by dividing the TEC and PEC for sediment dwelling organisms from the consensus-based approach to sediment quality criteria (MacDonald et al., 2000).

Table 6-4. Concentrations of Total Pb in Sediments at Locations Near or Matching the 15 Sites at which Dissolved Pb Concentrations Exceeded the AWQC, Excluding Mining Sites. a

<table>
<thead>
<tr>
<th>Basin ID</th>
<th>State</th>
<th>Land Use</th>
<th>Match</th>
<th>Total [Pb] (mg/kg dry sediment)</th>
<th>SW HQ b : max [Pb]/CCC</th>
<th>Pb Emissions (tons/year) (b)</th>
<th>No. Upstream NPDES permits for metals</th>
<th>Sediment Hazard Quotients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fac &lt; 20 km</td>
<td>Fac &lt; 50 km</td>
<td></td>
<td></td>
</tr>
<tr>
<td>45</td>
<td>RIOG</td>
<td>NM</td>
<td>Mixed</td>
<td>23</td>
<td>1.03</td>
<td>0.068</td>
<td>0</td>
<td>0.64</td>
</tr>
<tr>
<td>2</td>
<td>CONN</td>
<td>CT</td>
<td>Mixed</td>
<td>68</td>
<td>1.13</td>
<td>6.1</td>
<td>7.0</td>
<td>0.53</td>
</tr>
<tr>
<td>46</td>
<td>NROK</td>
<td>WA</td>
<td>Urban</td>
<td>47.3</td>
<td>1.14</td>
<td>0.39</td>
<td>0.43</td>
<td>1.3</td>
</tr>
<tr>
<td>46</td>
<td>NROK</td>
<td>ID</td>
<td>Forest</td>
<td>24.9</td>
<td>1.32</td>
<td>0.0</td>
<td>0.0</td>
<td>0.37</td>
</tr>
<tr>
<td>47</td>
<td>GRSL</td>
<td>UT</td>
<td>Rangeland</td>
<td>2900</td>
<td>1.45</td>
<td>0.0</td>
<td>0.36</td>
<td>1</td>
</tr>
<tr>
<td>31</td>
<td>OZRK</td>
<td>MO</td>
<td>Forest</td>
<td>2300</td>
<td>1.89</td>
<td>0.0</td>
<td>0.34</td>
<td>ND</td>
</tr>
<tr>
<td>58</td>
<td>OAHU</td>
<td>HA</td>
<td>Mixed</td>
<td>59</td>
<td>1.98</td>
<td>4.9</td>
<td>4.9</td>
<td>ND</td>
</tr>
<tr>
<td>1</td>
<td>NECB</td>
<td>RI</td>
<td>Mixed</td>
<td>240</td>
<td>2.51</td>
<td>4.1</td>
<td>11.7</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>CONN</td>
<td>CT</td>
<td>Mixed</td>
<td>68</td>
<td>2.53</td>
<td>0.081</td>
<td>11.3</td>
<td>0</td>
</tr>
<tr>
<td>46</td>
<td>NROK</td>
<td>ID</td>
<td>Mixed</td>
<td>1620</td>
<td>2.69</td>
<td>0.34</td>
<td>0.43</td>
<td>4</td>
</tr>
<tr>
<td>31</td>
<td>OZRK</td>
<td>AR</td>
<td>Mixed</td>
<td>28</td>
<td>3.46</td>
<td>0.0</td>
<td>0.01</td>
<td>0</td>
</tr>
<tr>
<td>31</td>
<td>COOK</td>
<td>AK</td>
<td>Reference</td>
<td>239</td>
<td>14.91</td>
<td>0.0</td>
<td>0.0</td>
<td>0</td>
</tr>
</tbody>
</table>

a Exhibit in increasing order of the surface water (SW) column risk hazard quotient (HQ). HQs exceeding 1.0 are highlighted in bold type.
b Data collected for corresponding surface water locations

Abbreviations: [Pb] = total Pb concentration in sediments (mg/kg dry sediment). CCC = Criterion Continuous Concentration (or chronic AWQC). TEC = threshold-effect concentration, and PEC = probable-effect concentration, both from the consensus-based sediment quality criteria approach published by MacDonald et al. (2000; 2003).

Table 6-4 presents the HQs for risks to benthic organisms at the 9 matching and 3 near-match locations at which dissolved Pb concentrations in the water column exceeded the CCC (i.e., chronic AWQC) for the protection of aquatic organisms in surface waters. Nine of the TEC-based HQs exceeded 1.0, and three were less than 1.0. The three sites with HQs less than 1.0 are unlikely to pose risks to benthic aquatic communities based on the available data. None
of these three sites were those likely to be affected by air emissions of Pb from point sources (i.e., Pb emissions were less than 0.07 tons per year at all three locations).

Five of the PEC-based HQs exceeded 1.0, indicating probable adverse effects. Three of these exceeded a PEC-based HQ of 10, indicating a very high probability of adverse effects, and possibly higher severity of effects than at the locations with lower HQ values. None of these three locations were likely to be affected, however, by air emissions. One in Idaho was downstream from several NPDES-permitted discharges of metals to surface waters (10th entry). The other two locations were found in Utah and Montana and it is possible that these concentrations reflect historical sediment contamination from mining operations.

Of the three locations for which air emissions of Pb from point sources appear to be more likely to be contributing to ongoing Pb contamination of surface water and sediments (i.e. locations in Connecticut, Hawaii, and Rhode Island, respectively), only one, the Blackstone River in Manville, Rhode Island, is also likely to receive significant current Pb inputs from upstream NPDES-permitted sites. In addition to Pb contamination of sediment through deposition of current air emissions to surface waters, sediment at these three locations may be contaminated by current and historic erosion of soils containing current and historic deposits of Pb, particularly from leaded gasoline. The Quienebaug River in Connecticut (a near match between the Jewett City and Clayville locations) and the water body at Waikakalaua Street near Wahiawa, Oahu, Hawaii, had no other obvious inputs of Pb in our assessment than the point sources within 20 km. Both of those locations, however, are in “mixed” urbanized areas, and therefore may also have historic Pb deposition from leaded gasoline and ongoing inputs of Pb to sediments from erosion of soils contaminated by leaded gasoline. A further discussion of methodology for the sediment screen can be found in the risk assessment report (ICF, 2006).

6.4.5.3 Primary Pb Smelter

A Characterization Area Investigation (CAI) was performed at the primary smelter facility by ELM Consulting in 2005. The investigation area included the smelter, slag areas, and several haul roads within a 2.1 km radius from the facility as well as two “reference areas”, presumed to be outside the area of influence of the smelter, 6 to 7 km south of the facility. The area was evaluated for the potential for ecological impacts to soil, sediment, and surface water from Pb originating from the facility. Data collected as part of the CAI were used.

To develop soil concentrations for this assessment, surface soil data were grouped into 3 geographic clusters: the west bank of Joachim Creek and two “reference areas”: Crystal City and Festus Memorial Airport. Surface water and sediment samples were taken from backwater and low flow areas along Joachim Creek both upstream and downstream of the facility 800 m, 1.6 km and 3.2 km from the smelter. Additional samples were taken from the Mississippi River and
a nearby pond. Details on the sampling methods used by ELM can be found in the risk assessment report (ICF, 2006).

HQs calculated for each of the sampling clusters developed for this case study are provided here: soil results are listed in Table 6-5, surface water results are presented for Table 6-6, and sediment results are presented in Table 6-7. HQs equal to or greater than 1.0 are bolded.

All three of the soil sampling clusters (including the “reference areas”) had HQs that exceeded 1.0 for birds. The west bank of the Joachim Creek samples had HQs greater than 1 for plants and mammals also. The surface water sampling clusters all had HQs less than 1.0 as results were all below the detection limit of 3.0µg/L. However, three sediment sample clusters in Joachim Creek (1, 2, and 3) had HQs ranging from 1.0 to 2.2 and the U-shaped pond and one drainage area had HQs greater than 3 but less than 5.

Table 6-5. HQs for Soils for Primary Pb Smelter Case Study.

<table>
<thead>
<tr>
<th>Location of Sample Cluster</th>
<th>HQ for Plants</th>
<th>HQ for Soil Invertebrates</th>
<th>HQ for Birds</th>
<th>HQ for Mammals</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 - West Bank of Joachim Creek</td>
<td>3.55</td>
<td>0.25</td>
<td>11.19</td>
<td>3.80</td>
</tr>
<tr>
<td>2 - Crystal City</td>
<td>0.54</td>
<td>0.04</td>
<td>1.70</td>
<td>0.58</td>
</tr>
<tr>
<td>3 - Near Festus Airport</td>
<td>0.40</td>
<td>0.03</td>
<td>1.28</td>
<td>0.43</td>
</tr>
</tbody>
</table>

¹Control samples taken outside perceived influence of the smelter.
Table 6-6. HQs Calculated for Surface Waters for Primary Pb Smelter Case Study.

<table>
<thead>
<tr>
<th>Sample Location and Cluster ID</th>
<th>HQ using CCC (Chronic)</th>
<th>HQ using CMC (Acute)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joachim Creek</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cluster 1</td>
<td>0.39</td>
<td>0.02</td>
</tr>
<tr>
<td>Cluster 2</td>
<td>0.40</td>
<td>0.02</td>
</tr>
<tr>
<td>Cluster 3</td>
<td>0.39</td>
<td>0.02</td>
</tr>
<tr>
<td>Cluster 4</td>
<td>0.41</td>
<td>0.02</td>
</tr>
<tr>
<td>Cluster 5</td>
<td>0.41</td>
<td>0.02</td>
</tr>
<tr>
<td>Mississippi River</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upstream</td>
<td>0.54</td>
<td>0.02</td>
</tr>
<tr>
<td>Near Facility</td>
<td>0.49</td>
<td>0.02</td>
</tr>
<tr>
<td>Downstream</td>
<td>0.48</td>
<td>0.02</td>
</tr>
<tr>
<td>Emission Deposition</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cluster 1</td>
<td>0.69</td>
<td>0.03</td>
</tr>
<tr>
<td>HRDDP</td>
<td>0.24</td>
<td>0.01</td>
</tr>
<tr>
<td>RRDP-02</td>
<td>0.47</td>
<td>0.02</td>
</tr>
<tr>
<td>DAMUP</td>
<td>0.40</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Table 6-7. HQs Calculated for Sediments in Surface Waters for Primary Pb Smelter Case Study.

<table>
<thead>
<tr>
<th>Location and Cluster Sample ID</th>
<th>Hazard Quotient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joachim Creek</td>
<td></td>
</tr>
<tr>
<td>Cluster 1</td>
<td>1.0</td>
</tr>
<tr>
<td>Cluster 2</td>
<td>1.6</td>
</tr>
<tr>
<td>Cluster 3</td>
<td>2.2</td>
</tr>
<tr>
<td>Cluster 4</td>
<td>0.84</td>
</tr>
<tr>
<td>Cluster 5</td>
<td>0.96</td>
</tr>
<tr>
<td>Mississippi River</td>
<td></td>
</tr>
<tr>
<td>Upstream</td>
<td>0.41</td>
</tr>
<tr>
<td>Near Facility</td>
<td>0.84</td>
</tr>
<tr>
<td>Downstream</td>
<td>0.34</td>
</tr>
<tr>
<td>Pond and Drainage Areas</td>
<td></td>
</tr>
<tr>
<td>U-shaped Pond Cluster</td>
<td>4.8</td>
</tr>
<tr>
<td>ED1</td>
<td>3.1</td>
</tr>
<tr>
<td>ED2</td>
<td>0.41</td>
</tr>
</tbody>
</table>
6.4.5.4 Secondary Smelter

For the secondary Pb smelter case study, as described in Section 4.2.3, two sets of modeled average Pb soil concentrations were used as exposure estimates for both the human health and the ecological risk assessment. The first set of concentrations was obtained by MPE modeling (Section 4.2.3.2). These modeled soil concentrations for the secondary Pb smelter were compared to empirical data obtained from a surrogate location. Based on this comparison, which suggested that modeled soil Pb concentrations for this case study might be significantly underestimated, we included a second characterization of soil concentrations besides the purely modeled approach. Specifically, measurements from a surrogate secondary Pb smelter location were used to “scale” up the modeled surface generated for this case study location to more closely match the empirical data obtained from the surrogate location (at specified distances from the facility). The averages for 1-, 5-, or 10-km interval distances from the secondary Pb smelter facility and the associated soil HQs calculated for each interval are presented in Table 6-8.

<table>
<thead>
<tr>
<th>Distance Range (m)</th>
<th>Modeled Soil Concentration Datasets (mg/kg)</th>
<th>HQ for Plants</th>
<th>HQ for Soil Invertebrates</th>
<th>HQ for Birds</th>
<th>HQ for Mammals</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total Pb Soil Conc. with Background</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>1000</td>
<td>86.6</td>
<td>0.72</td>
<td>0.05</td>
<td>2.28</td>
</tr>
<tr>
<td>1000</td>
<td>2000</td>
<td>20.7</td>
<td>0.17</td>
<td>0.012</td>
<td>0.54</td>
</tr>
<tr>
<td>2000</td>
<td>3000</td>
<td>17.3</td>
<td>0.14</td>
<td>0.010</td>
<td>0.46</td>
</tr>
<tr>
<td>3000</td>
<td>4000</td>
<td>16.3</td>
<td>0.14</td>
<td>0.010</td>
<td>0.43</td>
</tr>
<tr>
<td>4000</td>
<td>5000</td>
<td>15.8</td>
<td>0.13</td>
<td>0.009</td>
<td>0.42</td>
</tr>
<tr>
<td>5000</td>
<td>10000</td>
<td>15.4</td>
<td>0.13</td>
<td>0.009</td>
<td>0.41</td>
</tr>
<tr>
<td>10000</td>
<td>20000</td>
<td>15.1</td>
<td>0.13</td>
<td>0.009</td>
<td>0.40</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Scaled 3x Total Pb Soil Conc. with Background</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1000</td>
<td>260</td>
<td>2.17</td>
<td>0.15</td>
<td>6.84</td>
</tr>
<tr>
<td>1000</td>
<td>2000</td>
<td>62.3</td>
<td>0.52</td>
<td>0.037</td>
<td>1.64</td>
</tr>
<tr>
<td>2000</td>
<td>3000</td>
<td>51.8</td>
<td>0.43</td>
<td>0.030</td>
<td>1.36</td>
</tr>
<tr>
<td>3000</td>
<td>4000</td>
<td>48.9</td>
<td>0.41</td>
<td>0.029</td>
<td>1.29</td>
</tr>
<tr>
<td>4000</td>
<td>5000</td>
<td>47.3</td>
<td>0.39</td>
<td>0.028</td>
<td>1.24</td>
</tr>
<tr>
<td>5000</td>
<td>10000</td>
<td>40.2</td>
<td>0.39</td>
<td>0.027</td>
<td>1.22</td>
</tr>
<tr>
<td>10000</td>
<td>20000</td>
<td>45.4</td>
<td>0.38</td>
<td>0.027</td>
<td>1.19</td>
</tr>
</tbody>
</table>

HQ values greater than 1.0 are highlighted in bold type.

The modeled soil concentrations within 1 km of the facility showed HQs of greater than 1.0 for avian wildlife. All soil concentrations for locations greater than 1 km from the facility were associated with HQs less than 1.0 for this dataset. The three-times-higher-scaled soil concentration dataset, developed based on soil data from similar locations, resulted in avian HQs...
greater than 1.0 for all distance intervals evaluated, including the farthest interval modeled, 10 to
20 km from the facility. The scaled soil concentrations within 1 km of the facility also showed
HQs greater than 1.0 for plants, birds, and mammals.

6.4.5.5 Near Roadway Non-Urban Case Study

Table 6-9 presents the HQ calculated for the Corpus Christi, Texas, near roadside soil
concentration data. HQs for birds were greater than 1.0 at all but one of the distances from the
road. Mammalian HQs also were greater than 1.0 at the 2 m sampling distance from the
roadway. Finally, plants also had HQs ranging from 2.83 and 5.42 at the 2 m distance. However
at the further distance from the roadway (4 m), birds and mammals still had HQs greater than 1.

Table 6-9. HQs Calculated for Soils Near Roadway Non-Urban Case Study.

<table>
<thead>
<tr>
<th>Sample location – distance from roadway</th>
<th>Sample depth</th>
<th>Total Pb concentration (mg/kg)</th>
<th>HQ for Plants</th>
<th>HQ for Soil Invertebrates</th>
<th>HQ for Birds</th>
<th>HQ for Mammals</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 m</td>
<td>2.5 cm</td>
<td>340</td>
<td>2.83</td>
<td>0.20</td>
<td>8.95</td>
<td>3.04</td>
</tr>
<tr>
<td>2 m</td>
<td>10 cm</td>
<td>650</td>
<td>5.42</td>
<td>0.38</td>
<td>17.1</td>
<td>5.80</td>
</tr>
<tr>
<td>2 m</td>
<td>20 cm</td>
<td>15</td>
<td>0.13</td>
<td>0.019</td>
<td>0.395</td>
<td>0.13</td>
</tr>
<tr>
<td>4 m</td>
<td>2.5 cm</td>
<td>140</td>
<td>1.17</td>
<td>0.082</td>
<td>3.68</td>
<td>1.25</td>
</tr>
</tbody>
</table>

6.4.6 Discussion

The results presented in this section of the document represent initial screening results for
the three case study locations and the national-level screen. These results are only indicative of
the potential for effects to terrestrial and aquatic systems from ambient Pb. It seems clear,
however, from this initial screening assessment that more refined analyses would be necessary in
order to characterize risk to various receptors from ambient Pb.

6.4.7 Uncertainty and Variability

This section addresses uncertainties and limitations associated with the primary Pb
smelter case study, the secondary Pb smelter facility case study, the near roadway non-urban
case studies and associated with the national-level screening for risks to aquatic organisms from
Pb deposition from air to surface waters. Note that limitations for the ecotoxicity screening
values are described where they are introduced in Section 6.2.3.

Uncertainties that apply across case studies include, but are not limited to, the following:

- The ecological risk screen is limited to specific case study locations and other locations
  for which dissolved Pb data were available and evaluated in the national-level surface
water and sediment screen. Efforts were made to ensure that the exposure estimates were attributable to background level and air emissions of Pb; however, it is uncertain whether other sources might have actually contributed to the Pb exposure estimates.

- A limitation to using the selected ecotoxicity screening values is that they might not be sufficient to identify risks to some threatened or endangered species or unusually sensitive aquatic ecosystems.

- The database supporting the current AWQC for Pb is over 20 years old. There are data to indicate that Pb bioconcentrates to some extent in invertebrates (e.g., bioconcentration factors, or BCFs, of 500 to 1,700), and, to a lesser extent, in fish (e.g., BCFs of 42 to 45 in two species) in freshwater ecosystems. However, in 1984, data were insufficient to estimate Final Tissue Residue Levels associated with adverse effects in fish, and thus the BCFs did not influence the CCC value. Also, EPA is evaluating whether pH may be a better indicator of bioavailability compared to water hardness.

- No adjustments were made for sediment-specific characteristics that might affect the bioavailability of Pb in sediments in the derivation of the sediment quality criteria used for this ecological risk screen. Similarly, characteristics of soils for the case study locations were not evaluated for measures of bioavailability.

- Although the screening value for birds used in this analysis substituted more realistic parameters for diet composition and assimilation efficiency, it was based on a conservative estimate of the relative bioavailability of Pb in soil and natural diets compared with water soluble Pb added to an experimental pellet diet. A recent site-specific determination of a soil concentration protective of soil-invertebrate-consuming birds suggested that the values of 38 mg/kg or even 83 mg/kg are still overly conservative. This is possibly because the assimilation efficiency of Pb in soils and natural foods compared with the assimilation efficiency of Pb acetate added to pelleted diets is much less than 50 percent.

### 6.4.7.1 Primary Pb Smelter Case Study

The ELM Sampling and Analysis Plan (ELM, 2003) was designed to investigate possible ecological risks from all sources of Pb (and other contaminants) attributable to the primary Pb smelter without a need to attribute the source of Pb in ecologically sensitive areas (ELM, 2003; ELM, 2005). For purposes of the Pb NAAQS review, it is important to distinguish areas impacted primarily from current or historic air deposition of Pb from areas impacted primarily from other non-air sources (e.g. erosion of mining waste piles, surface runoff from exposed mining ores, direct waste discharges to water). While those areas impacted from these other non-air sources are likely to be impacted from air deposition as well, it is not usually possible to source apportion Pb in these areas. Therefore, these analyses attempt to focus on those areas where it may be possible to identify effects from policy relevant sources.
The soil sampling locations within a 2.1-km radius were all in areas that might have been subject to Pb inputs from Joachim Creek during flooding events. As such, the stations might not represent the concentrations of Pb in soils that result from air emissions from the smelter. This limitation may overstate the risks from deposition of Pb emitted from the facility.

6.4.7.2 Secondary Pb Smelter Case Study

The ecological risk screen used modeled rather than measured media concentration data because measured data were not available for the case study location. Data were available for similar locations and these data were compared to the modeled results. These results appeared to vary 3-fold therefore, scaled modeled data is also reported in this assessment. A full discussion of the modeling steps can be found in Section 4.3.2. Fate and transport modeling limitations and uncertainties are described in the risk assessment report (ICF, 2006).

6.4.7.3 Near Roadway Non-Urban Case Study

Few measured data were available to evaluate ecological impacts of contaminated soils near roadways in less developed areas where ecological receptors may be anticipated to occur. The measured soil data for the Corpus Christi, Texas location 2 m from the roadway ranged from 15 mg Pb/kg at 20-cm depths to 650 mg Pb/kg at 10-cm depths. The Pb concentrations selected at the Atlee, Virginia location ranged from 17 mg/kg 15 m from the roadway to 540 mg/kg 2 m from the roadway; both samples were collected from 7.5-to 15-cm soil depths. It is uncertain how representative of other roadways these data are.

The soil concentration data were measured at sampling locations between 2 to 30 meters away from intensely traveled roads, and the analysis did not evaluate the suitability of avian and mammalian wildlife habitat in close proximity to roadways. Without this evaluation, it is uncertain whether the assessment overestimates the ecological risks of Pb in roadway soils.

The assessment did not address surface water ecosystem impacts of Pb from near roadway runoff of Pb contaminated soils. This may underestimate risks to aquatic receptors via this exposure pathway.

6.4.7.4 National-Scale Surface Water Screen

The analysis revealed only two or three NAWQA sampling locations nationwide where there appear to be risks to the aquatic community from Pb that may have originated from atmospheric deposition. However, this is likely to be a large underestimate of the true number of such sites for several reasons:

- The NAWQA Study Units cover less than 50 percent of the land area of the United States.
• Dissolved Pb was an analyte at only 16 percent of all NAWQA sampling locations.
• Dissolved Pb was measured only once or twice at many locations.
• For waters with a hardness of less than 47 mg/L as CaCO$_3$, the CCC for dissolved Pb is less than the quantitation limit for dissolved Pb that was used until the fall of 2000 (i.e., 1 µg/L).
• Fewer than 15 percent of samples analyzed for dissolved Pb between 1994 and 2004 were assessed with the lower quantitation limit of 0.08 µg/L, which is a value that is sufficiently low to match the CCC for waters with a hardness as low as 4.7 mg/L CaCO$_3$.

The first two bullet points alone suggest that the number of such sites nationwide might easily be at least ten times higher than what was represented in the NAWQA database. In addition, where the land use around a sampling location was classified as “mining,” no investigation was conducted to determine whether air emissions from a nearby smelter might also be contributing to the Pb in the water.

There are many sources of uncertainty in the results presented for the sampling locations for which there were some data, including the following:
• Many sampling locations are represented by only one or two measurements of dissolved Pb.
• The water hardness for some sampling locations was not measured or is represented by only one or two measurements.
• Where there are multiple measures of both dissolved Pb and water hardness at a given location, no attempt was made to match sampling dates and times to develop time-specific CCC values. ¹
• The water hardness measured at some locations was less than the lowest value of 20 mg/L of CaCO$_3$ used to develop the equation to calculate a CCC. The CCC equation is not necessarily valid at values less than 20 mg/L CaCO$_3$.
• It is not known how quickly dissolved Pb concentrations changed at any of the locations.
• The database supporting the current AWQC for Pb is over 20 years old; new AWQC for Pb may be available in 2007.

6.4.7.5 National-Scale Sediment Screen
Results of this analysis cannot conclusively link any of the locations with probable adverse effects of Pb in sediments on benthic communities to ongoing air emissions of Pb. This analysis was limited to those 15 locations from the NAWQA database at which dissolved concentrations of Pb in surface waters exceeded the chronic AWQC for Pb. Those 15 locations are believed to represent a small fraction of surface waters in the U.S. for reasons given above.

¹ The coefficient of variation for water hardness measurements was less than 10 or 20 percent for many stations; however, at some locations, the coefficient of variation exceeded 50 percent, indicating higher fluctuations in water hardness measurements.
An additional limitation is that where the land use around a sampling location was classified as “mining”, no investigation was conducted to determine whether air emissions from a nearby smelter might also be contributing to the Pb in the water and sediments. It was assumed that direct runoff and erosion from the mining sites to the surface waters would have contributed to the bulk of the Pb in sediments.

Further limitations accrue from the sediment sampling data. There were only nine exact matches and three near matches between the 15 surface water sampling locations of interest and locations at which sediment samples also were analyzed. Furthermore, there was a single sediment sample at each of the locations of interest, some of which were taken in the early 1990s.

Finally, no adjustments were made for sediment-specific characteristics that might affect the bioavailability of Pb in sediments in the derivation of the sediment quality criteria used for this risk screen.

6.5 FUTURE ANALYSES

There are several expansions and refinements to this initial screening analysis that could be considered for any future analyses. Additional case study locations could be identified, particularly for near roadway scenarios, and a case study could be developed around the ecologically vulnerable location identified in this draft. Development of more refined exposure estimates for several of the case studies using ecosystem and habitat suitability models would allow for exposure estimates that result in body burdens for target organisms which could be directly compared to available concentration effects data. Broadening of the national-level screen to focus on locations with known large air emissions would allow for a better estimate of media concentrations in areas which are likely to be directly influenced by ambient air concentrations. Lastly, a more detailed discussion of the effect of Pb on ecosystem services and a discussion of research needs could be included.

6.6 THE SECONDARY LEAD NAAQS

6.6.1 Introduction

This first draft document discusses the general approach that is intended to be used in considering the adequacy of the current standard and in identifying policy alternatives in the next draft of this document. In addition, the next draft will include key uncertainties and research recommendations related to setting a secondary Pb standard.

The current secondary Pb standard is 1.5 μg Pb/m³, as a maximum arithmetic mean averaged over a calendar quarter, set equal to the primary standard (43 FR 46246). A final decision should draw upon scientific information and analyses about welfare effects, exposure and risks, as well as judgments about the appropriate response to the range of uncertainties that
are inherent in the scientific evidence and analyses. Our approach to informing these judgments, discussed more fully below, is based on a recognition that the available ecological evidence generally reflects a continuum consisting of ambient levels at which scientists generally agree that adverse ecological effects are likely to occur through lower levels at which the likelihood and magnitude of the response become increasingly uncertain.

This approach is consistent with the requirements of the NAAQS provisions of the Act and with how EPA and the courts have historically interpreted the Act. These provisions require the Administrator to establish secondary standards that, in the Administrator's judgment, are requisite to protect public welfare. In so doing, the Administrator seeks to establish standards that are neither more nor less stringent than necessary for this purpose. The Act does not require that secondary standards be set at a zero-risk level but rather at a level that avoids unacceptable risks to public welfare.

6.6.2 Approach

As indicated in Chapter 1, the policy assessment to be presented in the final version of this document is intended to inform judgments required by the EPA Administrator in determining whether it is appropriate to retain or revise the NAAQS for Pb. In evaluating whether it is appropriate to consider retaining the current secondary Pb standard, or whether consideration of revisions is appropriate, we intend to focus on the extent to which a broader body of scientific evidence is now available that would inform such decisions. As summarized in section 5.2, the 1978 notice of final rulemaking (43 FR 46246) outlined key factors considered in selecting the elements of a standard for Pb: the Pb concentration (i.e., level); the averaging time; and the form (i.e., the air quality statistic to be used as a basis for determining compliance with the standard). Decisions on these elements were made only so far as to indicate that due to a lack of relevant data at that time, the secondary standard should be set to be identical to the primary standard.

In developing conclusions and identifying options for the Pb standard in this review, staff intends to take into account both evidence-based and quantitative exposure- and risk-based considerations. A series of general questions will frame our approach to reaching conclusions and identifying options for consideration by the Administrator as to whether consideration should be given to retaining or revising the current secondary Pb standard. Examples of questions that we intend to address in our review include the following:

- To what extent has evidence of new effects and/or sensitive ecosystems become available since the last review and to what extent are we able to characterize these effects?
- To what extent does newly available information support or call into question any of the basic elements of the current standard?
• Is there evidence of associations, especially likely causal associations, in areas that meet the current standard? What are the important uncertainties associated with that evidence?

To the extent that there is support for consideration of a revised standard, we will then identify ranges of standards (in terms of an indicator, averaging time, level, and form) that would reflect a range of alternative public welfare policy judgments, based on the currently available information, as to the degree of protection that is requisite to protect public welfare.

As noted in Chapter 1, staff will also evaluate removing Pb from the criteria pollutant list and assess whether revocation of the Pb NAAQS is an appropriate option for the Administrator to consider. Section 108 of the Clean Air Act states that the Administrator “shall, from time to time … revise a list which includes each pollutant -

(A) Emissions of which, in his judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare;

(B) The presence of which in the ambient air results from numerous or diverse mobile or stationary sources; and

(C) For which air quality criteria had not been issued before December 31, 1970, but for which he plans to issue air quality criteria under this section.”

In evaluating such an option, staff expects to consider, among other things, many of the same issues identified earlier in the section. Information about the kinds and types of sources of Pb emissions, as well as the quantities of emissions from those sources will also be important for consideration.
REFERENCES

Alabama Natural Heritage Inventory. (2001) Alabama Inventory List: The Rare, Threatened and Endangered Species, Animals and Natural Communities of Alabama. Montgomery, AL: Privately printed by the Alabama Natural Heritage Program.


