AP42 Section:	1.1
Reference:	55
Title:	Electric Utility Trace Substances Synthesis Report, Volume 1, Report TR-104614, Electric Power Research Institute, Palo Alto, CA, November 1994.

1.1 #55

EPRI
Electric Power
Research Institute

EPRI TR-104614
Project 3081
November 1994

Electric Utility Trace Substances Synthesis Report

Prepared by
Electric Power Research Institute
Palo Alto, California

Electric Utility Trace Substances Synthesis Report

Volumes 1–4

A comprehensive evaluation of human health risks from trace substances in electric utility stack plumes was carried out for each of 600 U.S. power plants. Emissions estimates were based on measurements at 43 units. Under realistic assumptions of exposure and plant configuration, inhalation risks were well below one in one million for increased cancer likelihood to all individuals exposed to emissions from power plants. Mercury case studies at four power plants showed health risks lower than federal guidelines.

INTEREST CATEGORIES

Air quality—human health
Fossil plant air quality control
Risk analysis; management & assessment
Fuel quality

KEYWORDS

Air toxics
Risk assessment
Emissions
Health effects
Control systems
Mercury

BACKGROUND Research begun by EPRI in the past decade showed the inadequacy of published data for characterizing the concentrations of and risks due to trace substances in utility fuels, power plant process streams, plant stack emissions, and other discharges. Guided by initial screening-level risk assessments, EPRI field measurements began before Congress enacted the Clean Air Act Amendments of 1990, which require the USEPA to study utility trace substance emissions and associated health risks. These EPRI measurements, and the simultaneous development of risk assessment tools for inhalation and multimedia exposure, led to a comprehensive evaluation of electric utility trace substances.

OBJECTIVES

- To characterize the emissions of two dozen trace substances from fossil-fired utility stacks, based on fuel use and on plant and control operations.
- To quantify the potential health risks to surrounding communities from these emissions, using the best available information on health effects and human exposure.

APPROACH Field measurements were carried out at 43 operating and pilot plant sites by EPRI and the U.S. DOE to characterize trace substances emissions from power plants, and the fate of these substances within each power plant. Measurements of stack emissions were used to derive relationships associating fuel use and particulate emissions with emissions rates for each trace substance. These relationships were then applied to each of 1700 operating units at 600 power plants in the United States. For purposes of prediction, the power plants were configured based on two independent studies of industry operations in the year 2010 and after. Health risks from trace substances were based on mid-1994 federal guideline levels for exposure and dose; the exception was for arsenic, whose risk calculations employed new research yielding values of one-third the federal level.

RESULTS Emissions of most trace substances were found to be well below (in some cases, substantially below) levels estimated in previously published reports. For mercury, industry-wide emissions are estimated at one-half the level cited in earlier reports. Particle-bound trace substances are to a large extent removed by particulate controls at all power plants. More volatile trace substances, such as mercury, are less effectively removed by flue gas desulfurization systems. Mercury removal is highly variable, and not fully understood. Risks based on "worst-case" assumptions of human exposure, those typically used in regulatory proceedings, exceed risks based on more realistic exposure measures by a factor of 3 to 5. Under realistic exposure scenarios, inhalation risks of cancer are below one in one million for all power plants, and inhalation risks of

noncancerous toxic effects are well below federal threshold levels for all power plants. Population-weighted inhalation risks are insignificant. Case studies at four power plants found that multimedia risks, including risks from mercury, are below levels of concern for all health effects.

EPRI PERSPECTIVE EPRI work, begun well before passage of the Clean Air Act Amendments, was accelerated after 1990 to provide the most accurate technical information available on utility trace substances to the federal government and to the research community. This information was used to design an effective framework for assessing trace substance emissions and health risks for the entire utility industry. EPRI's comprehensive approach not only yielded the estimation of community risks due to emissions from every individual power plant in the nation, but also permitted risks due to emissions from multiple power plants to be estimated. In all cases, the resulting risks were insignificant, even under highly conservative assumptions. Several independently-derived scenarios of the industry in 2010 led to estimates of risk that did not vary greatly, demonstrating the robustness of the methodology.

Although additional work is needed to further characterize emissions, health effects, and resulting risks in particular cases, this national study provides strong evidence that utility emissions of trace substances do not pose a significant risk to individuals or communities.

PROJECT

RP3081; RP3508; RP3237; RP3177, RP3453

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Electric Utility Trace Substances Synthesis Report

Volume 1: Synthesis Report

TR-104614-V1

Research Project 3081
November 1994

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EXECUTIVE SUMMARY

Background and Overview

Title III of the 1990 Clean Air Act Amendments (CAAA) addresses human exposure to trace substances listed in the legislation as "hazardous air pollutants." This portion of the amendments contains specific provisions that require the U.S. Environmental Protection Agency (EPA) to conduct a study of the potential health risks posed by the emission of hazardous air pollutants from electric utility plants. The legislation explicitly directs EPA to focus on the risks to human health due to emissions from fossil fuel-fired electric utility steam generating units (with >25MW capacity) *after* utilities have complied with other provisions of the Clean Air Act (such as provisions related to emissions of sulfur and nitrogen oxides). The EPA administrator is to decide whether further regulation of electric utility steam generating units is "necessary and appropriate" based on the results of EPA's study.

This report by the Electric Power Research Institute (EPRI) is intended to provide information to electric utilities, and to EPA for its own utility study, as well as to the broader scientific and regulatory community. The key goal of the effort is to bring together the best scientific data and methods currently available to understand the potential magnitude and nature of human health risks due to trace substance emissions from electric utility steam-generating units in the United States. As such, the report summarizes the results of recent trace substance research, conducted by EPRI and others, addressing a range of critical issues such as trace substance emission rates from utility generating units, appropriate sampling and analytical methods for determining these rates, and the toxicity of specific substances found in utility emissions. Further, the report describes the risk assessment methodology developed to integrate this research and understand its implications with respect to a nationwide evaluation of potential human health risk.

The data, methodologies, and analysis results presented in this report provide an understanding of utility trace substance emissions and the risks associated with these emissions, consistent with the best data and methodologies available at this time. The results of this research and analysis indicate that trace substance emissions from fossil-

fired electric utility steam generating units, after compliance with other provisions of the Clean Air Act Amendments, will not pose significant long-term risks (either carcinogenic or noncarcinogenic) to human health.

Scope/Approach

Two major projects served as the foundation for EPRI's trace substance research and provided much of the input and direction for this analysis. These projects were the Power Plant Integrated Systems: Chemical Emissions Study (PISCES) project and the Comprehensive Risk Evaluation (CORE) project. PISCES conducted extensive method development and field measurement programs that provide a database for predicting airborne trace substance emissions throughout the utility industry. CORE developed the methodology to integrate the information from PISCES and other research results on emissions, fate, and health effects, into a nationwide assessment of health risks.

As an initial step, these projects identified 16 trace substances as critical to include in an industry-wide risk assessment. The 16 were selected based on an evaluation of those trace substances (1) most likely to be found in utility stack emissions (based on a literature review and EPRI measurement data) *and* (2) for which health risk analysis could be readily performed using available toxicity factors. The substances identified as meeting both of these criteria include:

Arsenic	Chlorine	Lead	PAHs
Benzene	Chromium	Manganese	Radionuclides
Beryllium	Dioxins/Furans	Mercury	Selenium
Cadmium	Formaldehyde	Nickel	Toluene

The scope of the risk assessment presented in this report encompasses the potential chronic (i.e., long-term) health risks due to emissions of these 16 trace substances from approximately 600 power plants, encompassing roughly 1700 fossil-fired steam electric generating units. To predict emissions from these units, several scenarios were produced describing how the electric utility industry may be configured after all measures to achieve compliance with the SO_2 provisions of the CAAA are implemented (assumed to be the year 2010). These scenarios were augmented by additional data on particulate controls developed through an independent survey of utility operators regarding their plans to modify or upgrade particulate control equipment to meet other air pollution control requirements. These projections of power plant operations and control configurations were combined with characterizations of trace element concentrations in utility fuels to produce estimates of emissions in the year 2010 from the entire population of U.S. electric utility steam generating units (with >25 MW nameplate capacity).

Plant operating and design characteristics together with regional meteorological data were used to model the transport and dispersion of trace emissions and the resulting ground-level concentration within 50 kilometers of each plant. The EPA's Industrial Source Complex Long Term 2 (ISCLT2) model was selected for the dispersion modeling. The ISCLT2 model estimates annual-average concentrations, making it useful for evaluating chronic effects due to long-term exposures to chemicals, as well as allowing the efficient modeling of several hundred separate sources. In addition to evaluating the concentration levels due to individual plants, an "overlapping plume" analysis was performed to account for the total exposure of populations living within 50 kilometers of more than one power plant.

Population data from the 1990 United States census were used to assess the exposure levels of individuals living within 50 kilometers of electric utility generating units, based on electricity supply scenarios for the year 2010. Due to the initial atmospheric transport of these contaminants, the primary pathway evaluated for human exposure was through inhalation. The incremental increase in inhalation cancer risk due to utility emissions was computed for two exposure scenarios: the "Maximally Exposed Individual" (or MEI) and a "Reasonably Exposed Individual" (or REI). The MEI represents a conservative estimate of possible exposure, assuming an individual breathes outside air, in the location with the highest concentrations of trace substances due to a single power plant's emissions, 24 hours per day for a 70-year lifetime.

To provide a more realistic estimate of potential human exposures, and in keeping with recent recommendations by EPA and the National Academy of Sciences, EPRI developed the REI. The REI incorporates data on the amount of time individuals spend indoors and outdoors in various activities and on indoor reductions of outdoor concentration levels. In addition to the inhalation exposure assessments, several case studies were performed to evaluate potential multimedia exposures to utility trace emissions through all exposure pathways (i.e., inhalation, ingestion, and dermal contact).

Finally, the exposure assessments were combined with dose-response relationships to provide an estimate of potential public health risks. Due to the nature of the emissions (i.e., very low levels of substances emitted over a relatively long period of time), the primary health effects evaluated are potential increases in chronic cancer or non-cancer risks over a 70-year lifetime. For noncarcinogens, the predicted concentration levels due to generating unit emissions are compared to federal reference doses (RfDs) or reference concentrations (RfCs) for the substances of concern. These reference levels are defined as being levels of daily exposure that are likely to be without appreciable chronic deleterious effects, even for sensitive individuals in a population.

For most of the carcinogens, the relevant unit risk factor tabulated by the U.S. EPA as of mid-1994 was used to estimate potential incremental cancer risk due to utility emissions of trace substances. The unit risk factor represents a plausible upper-bound estimate of the increased probability of contracting cancer due to a 70-year lifetime exposure to an

inhalation concentration of 1 $\mu g/m^3$ of a given substance. In the case of arsenic, a revised unit risk factor was derived based on a re-analysis of existing and new occupational exposure data. The revised unit risk factor of 1.43×10^{-3} per $\mu g/m^3$ (one-third of that listed in 1994 in the EPA Integrated Risk Information System (IRIS) database) was used in this study to estimate arsenic inhalation cancer risks. In addition, risk estimates were carried out using the higher EPA IRIS value.

Synopsis of Results

The following highlights key research findings that contributed to the overall assessment of risks due to trace emissions from electric utility steam generating units, as well as the results of the risk assessment itself.

Concentrations of Trace Substances in Coal

A data set based on U.S. Geological Survey information and coal cleaning data was developed to characterize the concentrations of inorganic substances in coals, "as fired," at power plants. This database represents a significant improvement over previous coal characterizations in that it incorporates economic criteria (i.e., seam depth and quality) and the impact of coal cleaning processes in predicting "as-fired" coal properties.

In addition to this work, a measurement program was carried out to specifically examine mercury concentrations in "as-fired" coal. Approximately 150 samples of delivered coal, representing 20 major seams and all coal ranks, were analyzed by atomic fluorescence to determine mercury concentrations. This analysis showed that concentrations of mercury in "as-fired" or "as-received" coal are about half of earlier estimates based on "in-ground" coal samples.

Field Measurements and Data Correlations

EPRI initiated its Field Chemical Emissions Monitoring (FCEM) program to rectify the lack of adequate field measurement data on trace substance emissions from operating power plants. FCEM provided the first such data for trace substances in power plant process and discharge streams. Initiated in May 1990 as part of the PISCES project, the FCEM program used EPA-recommended sampling analysis protocols (making modifications, as necessary), and acquired measurement data from 35 utility sites representing different combinations of boiler, fuel type, and environmental control devices. In addition, the U.S. Department of Energy (DOE) also conducted sampling and measurement programs at 8 utility plants, using similar protocols. The EPRI and DOE data encompass measurements for each major fuel type and boiler configuration, as well as all current SO_2, NO_x, and particulate control technologies. The resulting database represents the most up-to-date, complete, and accurate data set currently available for estimating trace substance emissions from the national population of steam electric generating units.

For coal-fired units, guidelines were developed for extrapolating the measurement data to predict trace emissions from similar units. Three major groupings were defined in order to develop emission factors or correlations that serve as the basis for predicting stack emissions for all coal-fired generating units.

- Particulate-phase inorganic substances. Based on the field data, these substances (e.g., arsenic, chromium) are well controlled by a particulate control device. In general, reductions of greater than 90% from levels in the incoming coal were achieved. Correlations were thus developed to estimate stack emissions based on the inlet coal concentration of each substance and the level of total particulate emissions.
- Volatile inorganic substances. These substances (including hydrochloric acid, mercury, and selenium) tend to be more volatile and not efficiently captured by particulate control devices. Based on the measurement data, the emissions of these substances could not be correlated to any specific factors and were therefore estimated using average removal efficiencies for each substance and control configuration.
- Organic compounds. These compounds are formed at very low levels during combustion and emitted in concentrations of parts per billion or lower. Emissions of organic compounds were estimated using the geometric means of measured emission factors, calculated from the field data, for each substance.

For oil- and gas-fired power plants, available data are not yet adequate to estimate the trace substance concentrations in fuel burned at individual utility sites on a nationwide basis. Emissions for these plants were calculated using average emission factors (i.e., emissions per Btu of heat input), based on the field measurements, averaged across all measured units of the same configuration and fuel type. These data show that the emission factors for uncontrolled oil-fired power plants are about the same as for coal-fired plants with electrostatic precipitators.

Inhalation Exposure Assessment

As noted above, EPRI developed an alternative measure of human exposure designed to provide a more realistic estimate of potential human health effects than the traditional "Maximally Exposed Individual," or MEI, approach. The "Reasonably Exposed Individual," or REI, employed in this report still focuses on an individual living in the area of highest concentrations due to utility emissions. However, the REI methodology incorporates data on age-specific and activity-specific breathing rates and other exposure variables. In addition, the REI approach assumes that generation units do not continue to operate for a full 70 years from their respective start-up dates, but are replaced after an average operating span of 55 years with units in the same location that meet the EPA 1994 New Source Performance Standards (NSPS) for particulates.

In general, inhalation exposures to carcinogens using the REI methodology are 2% to 19% of the exposure levels computed for the MEI. This difference is due primarily to assumptions regarding the amount of time individuals spend indoors and the amount of time

spent residing in one location. For the noncarcinogens, REI exposures are 21% to 70% of those computed for the MEI. This difference is primarily due to assumptions regarding the amount of time spent indoors and the reduction in trace substance concentration levels in indoor environments.

Health Effects

Arsenic. Based on new analyses of occupational exposure data, EPRI computed a revised unit risk factor for estimating increased cancer risks due to inhalation exposures to arsenic. The revised unit risk factor (1.43×10^{-3} per $\mu g/m^3$), which is used as the base case throughout this study, is one-third of the current EPA value. The revised value reflects a re-analysis of existing and new occupational exposure data for smelter workers exposed to arsenic in copper smelter dust. EPRI used the standard EPA risk assessment methodology to calculate the revised unit risk.

Other important issues with respect to the toxicity of arsenic in power plant fly ash may not be addressed by the revised unit risk factor and remain the subject of ongoing research at EPRI and elsewhere. These issues include: (1) the importance with respect to health effects of differences in the valence state of arsenic found in copper smelter dust and that found in fly ash, (2) the comparative bioavailability of arsenic in fly ash vs. in other mixtures, and (3) the impact of metabolic detoxification processes at various arsenic exposure levels. Although research is underway on these topics, they currently remain unresolved.

Mercury. Recent data also may provide an improved basis for computing potential neurotoxic effects due to chronic exposures to mercury. EPA's current reference dose for methylmercury is based on an incident in Iraq involving acute exposures to very high methylmercury concentrations in grain. However, data sets based on populations exposed to mercury via fish ingestion may be more appropriate for evaluating health risks from utility mercury emissions in the United States. EPRI is currently assessing data on the neurological responses of children in New Zealand exposed to methylmercury via maternal fish ingestion to serve as an alternative basis for evaluating risk.

Inhalation Risk Assessment

The results of research in a number of areas were brought together in a nationwide assessment of the potential health risks associated with trace substance emissions from electric utility steam generating units. In general, the analysis indicates that the cancer and noncancer inhalation risks to the general public due to trace substance emissions from utility generating units are small, as described in the following discussion.

Cancer Risk. For the roughly 600 plants investigated, the expected increase in individual cancer risk, incorporating exposure assumptions associated with maximum exposure over a 70-year life span, did not exceed 1.7 in one million (1.7×10^{-6}). Out of this entire group of power plants, only 3 plants, or 0.5%, approach exposures leading to a cancer risk greater than one in one million (1×10^{-6}) for a maximally exposed individual (MEI).

Incorporating more reasonable assumptions regarding individual exposure patterns results in the increased cancer risks for all plants being less than one in one million, and all but 2 plants (0.3%) being less than one in ten million (1×10^{-7}). Figure ES-1 shows the distribution of increased cancer risk for both a "Reasonably Exposed Individual" (REI) and a "Maximally Exposed Individual" (MEI) due to utility trace substance emissions. As shown, the vast majority of plants (greater than 85%) are associated with increased individual risk levels that are, at most, below 1 in 100 million (1×10^{-8}) for the REI, and below 1 in 10 million (1×10^{-7}) for the MEI.

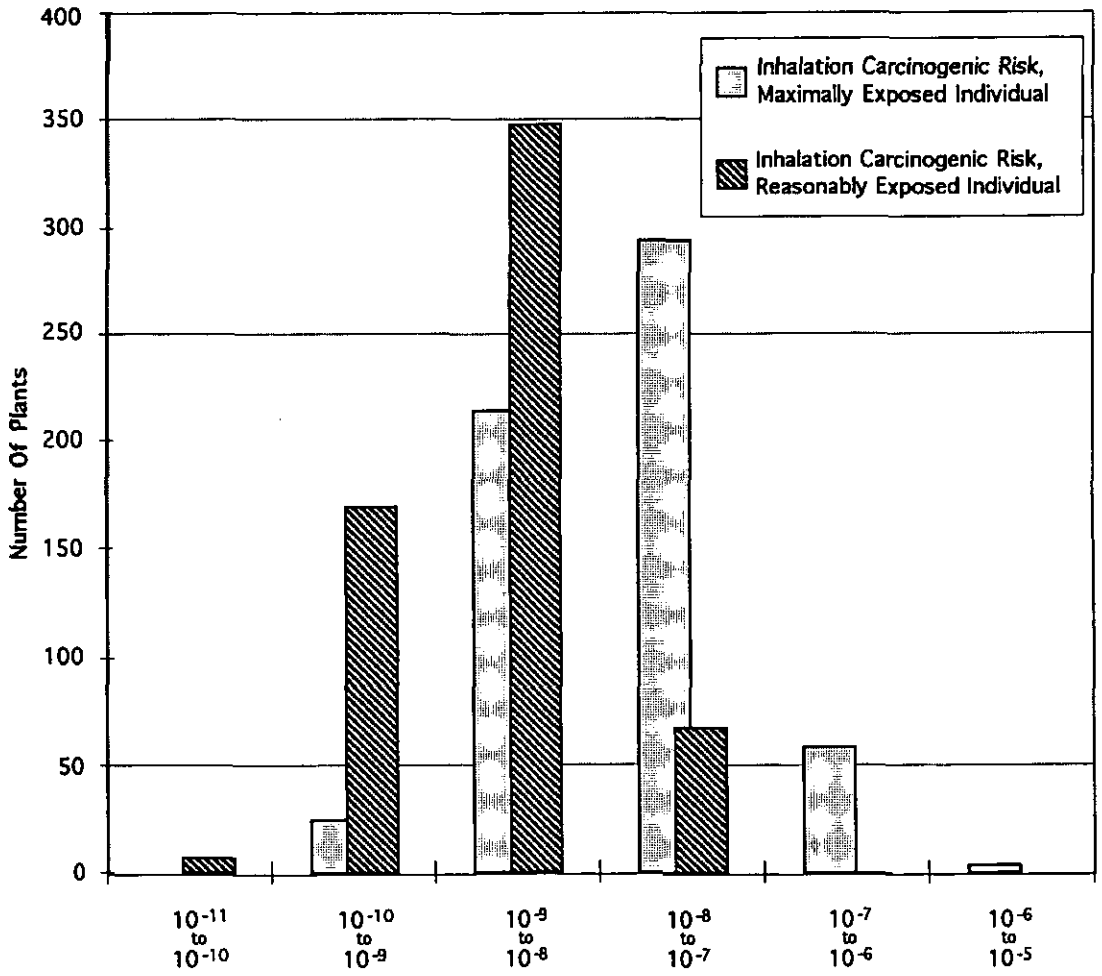

Figure ES-1.
Histograms of Inhalation Carcinogenic Risk

For coal- and oil-fired power plants, across all coal ranks (i.e., bituminous, subbituminous, and lignite) and control configurations, arsenic and chromium were found to be the largest potential contributors to inhalation cancer risks from utilities.

For plants fired only by gas, the median inhalation cancer risk is about an order of magnitude less than median risk levels for other fossil-fired plants, and the primary contributors to risk from gas plant emissions are chromium and formaldehyde.

Noncancer Risks. Noncancer risks were evaluated based on comparisons with EPA-defined reference doses (RfD) or reference concentrations (RfC). When no EPA-listed value was available, an RfD or RfC was developed based on other available health standards. These RfD and RfC values reflect thresholds below which no adverse health effects are anticipated, even over continuous long-term exposures. For all of the plants, inhalation exposures to all of the noncarcinogens examined (including mercury) were well below the recommended threshold levels.

Sensitivity and Scenario Analyses. Sensitivity analyses were conducted to clarify the impact of uncertainty in key parameters or modeling assumptions on the inhalation risk estimates. Based on extensive analyses, no single group of plants (as defined by plant configuration, operating characteristics, stack height, or fuel type) could be identified as consistently correlated with relatively high risk estimates. Rather, it was usually a unique combination of site- and plant-specific factors that led to higher relative risks for an individual plant.

Finally, although variations in assumptions about future scenarios (e.g., load, fuel type, control configuration, etc.) can influence risk estimates for individual plants and the relative risks across multiple plants, in the aggregate, the alternative scenarios did not significantly affect the risk estimates.

Multimedia Risk Assessment

EPRI conducted case studies of four power plants with measured emissions to estimate carcinogenic and noncarcinogenic multimedia risks from power plant emissions using the EPRI multimedia risk model, *TRUE* (Total Risk of Utility Emissions). Based on the four case studies, the estimated maximum incremental cancer risk due to exposures through all pathways (i.e., inhalation, ingestion, dermal contact) was below one in one million (1×10^{-6}) for all plants studied. For noncarcinogens, the multimedia analysis also showed all exposure levels to be below the relevant threshold levels (RfDs and RfCs) for adverse effects.

Mercury. A key focus of the multimedia risk assessment was the potential health effects due to mercury emissions. The case study results suggest that risks due to power plant emissions of mercury are primarily driven by exposure through ingestion of fish in which mercury has accumulated as methylmercury. The current EPA Reference Dose for mer-

cury is 0.3 μg per kg of body weight per day of methylmercury. Predicted methylmercury exposure levels due to mercury emissions from each of the four case study power plants are all less than 30% of federal reference levels.

Although these results incorporate the best current understanding of mercury-related risks, the complex biogeochemistry of mercury, how it is transformed in the atmosphere and ecosystems, and what exposure and dose levels ultimately result in health effects are still not fully understood. As new and ongoing research contributes to our understanding of this chemical, potential impacts on the risk assessment results should be considered.

Radionuclides. Exposures due to radionuclide emissions from fossil-fired generating units were also found to be small. Changes in the risk assessment methodology for radionuclides used by EPA resulted in risk levels below previous estimates made by EPA in its 1989 National Emissions Standards for Hazardous Air Pollutants (NESHAPS) for radionuclides. Specifically, improvements in estimating power plant radionuclide emissions (based on the average radioactivity of emitted particulate matter) resulted in predicted emissions that are one-third to one-tenth of previous estimates. Changes in modeled deposition rates further reduced predicted exposure levels.

Impact of Current Control Technologies

Trace metals in flue gas are normally condensed on fly ash particles and can be removed effectively by an efficient particulate collector. Mercury, present mainly in vapor form in the flue gas, is not collected effectively by particulate control devices such as electrostatic precipitators or baghouses. Studies to date to assess flue gas mercury removal methods, such as injection of activated carbon, show that the low mercury levels present in power plant flue gas are much more difficult to remove than the mercury emitted from waste incineration plants. Significantly more research is needed to evaluate these and other removal options in power plant settings.

Summary

This report is intended to provide insight into the best data and methods available for estimating health risks due to trace emissions from fossil fuel-fired steam-electric generating units. To meet this goal, EPRI conducted extensive research aimed at improving the state-of-the-art in a number of areas, including:

- More appropriate fuel composition data
- More accurately measuring trace substance emissions from power plant stacks
- Improved methods for estimating emissions for the national capacity of power plants

- Development of alternative scenarios of future industry operations
- Updated health impact data
- Development of reasonable measures of human exposure and health risks

Although the research presented in this report provides a considerable improvement over previously available data and methods, important uncertainties remain. For example, more complete data are needed on: speciation of arsenic, chromium, and mercury in stack emissions; the atmospheric chemistry of trace substances; and dose-response information incorporating bioavailability. However, the results presented herein suggest that trace substance emissions from electric utility steam generating units, after compliance with other provisions of the CAAA, will not pose significant long-term risks (either carcinogenic or noncarcinogenic) to human health.

ACKNOWLEDGEMENTS

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INTRODUCTION

1.1 Background

Title III of the 1990 Clean Air Act Amendments (CAAA) is intended to reduce human exposure to trace substances listed in the legislation as "hazardous air pollutants." This title of the amendments contains specific provisions that require the U.S. Environmental Protection Agency (EPA) to conduct a study of the potential health risks posed by the emission of hazardous air pollutants from electric utility plants. A number of the CAAA-listed trace substances may be present in emissions from some fossil fuel-fired power plants.

The EPA study must focus on the risks to public health that would continue to be posed by power plants after plant operators have complied with provisions of the CAAA. This means that, for purposes of the study, EPA must assume that strategies for reducing power plant emissions of sulfur dioxide (SO_2), nitrogen oxides (NO_x), and particulate matter are already in place. On the basis of the study results, the EPA administrator will decide whether further regulation of electric utility steam generating units is "necessary and appropriate." (Title III of CAAA defines an electric utility steam generation unit as a fossil-fuel-fired steam electric generating plant constructed to supply more than one-third of its potential electric output capacity and more than 25 megawatts electric output to a utility power distribution system.)

This report by EPRI is intended to provide information on trace emissions, by summarizing the most recent methodology and research results available. The report focuses on methods and findings from EPRI projects to assess the impacts of fuels, operating configurations, and control devices on trace substance emissions and the health risks from these emissions. The report not only assesses the health risks from projected emissions, but also provides additional insights into the key drivers of risk.

1.2 Objectives

The Electric Power Research Institute (EPRI) has conducted a number of research projects addressing the broad range of issues associated with utility trace substance emissions and their potential effects. This report on that research has three principal objectives:

- To provide a comprehensive overview of current research results and associated analyses related to utility emissions of airborne trace substances. In particular, the report highlights the use of the most recent measurement data from operating power plants to provide an improved basis for estimating trace substance emissions.
- To describe an approach that integrates the above information into a comprehensive assessment of potential health risks due to trace substance emissions from electric utility steam-generating units in the United States.
- To present the results of an industrywide application of the approach, including inhalation risks due to trace emissions from electric utility generating stations and risks due to exposure by both inhalation and non-inhalation routes.

The report brings together information on airborne trace substances, analytical methods, and results of recent and ongoing research from programs both within and external to EPRI. The results presented here are intended to provide insights into the magnitude and nature of trace substance emissions from electric utility steam generation units, as well as insights into the key factors that may influence the associated health risks.

1.3 Conceptual Framework

Two major projects served as the foundation for EPRI's trace substance research: the Power Plant Integrated Systems: Chemical Emissions Study (PISCES) project and the Comprehensive Risk Evaluation (CORE) project. PISCES has conducted method development and field measurement programs to characterize material flow and the rate of emissions of airborne trace substances from different types of power plants. This work provides the basis for estimating trace substance emissions throughout the utility industry. CORE developed the framework and methodology that were used to integrate existing information and new results from research by EPRI and others into an assessment of health risks.

This report describes EPRI's key research efforts, including the methodologies used, important results to date, and remaining technical issues. The report also describes the risk assessment methodology and how the results of the other research areas are integrated into a comprehensive assessment of the risks to public health due to utility stack emissions of trace substances.

Figure 1-1 depicts an organizing framework in which risk assessment is portrayed as a multi-step process:

- Study Definition;
- Source Characterization;
- Transport & Dispersion;

- Exposure Assessment; and
- Dose/Response.

The following section introduces the research elements that are incorporated into each of these steps and provides a brief description of each.

Figure 1-1.
Risk Assessment Approach

1.4 Trace Substances Research Overview

During the past decade EPRI has conducted over 40 projects aimed at improving understanding of trace substance emissions from electric utility power plants and the potential risks that these emissions may pose to human health. These projects have encompassed the full range of trace substance issues, from improved characterization of fuel composition, through sampling of power plant process streams, to stack emissions measurements, to modeling transport and transformations in the atmosphere, to assessing exposure patterns and potential human health effects. The resulting data and analytical models provide a foundation for understanding key processes and properties associated with trace substance emissions and their impacts.

Figure 1-2 summarizes each of the principal areas of research, and indicates how the research results contributed to the various steps in Figure 1-1. For example, research on the potential mix of future generation technologies and on expected SO_2 and particulate emission compliance strategies helped to characterize combinations of fuel, boiler type, and emission controls that may contribute to patterns of trace substance emissions. Likewise, field sampling programs and application of analytical methods provided an improved basis for estimating emissions for source characterization. The following sections summarize important information needs in each area, as well as the key research projects and results that have addressed these needs. The main questions and uncertainties that remain are also outlined.

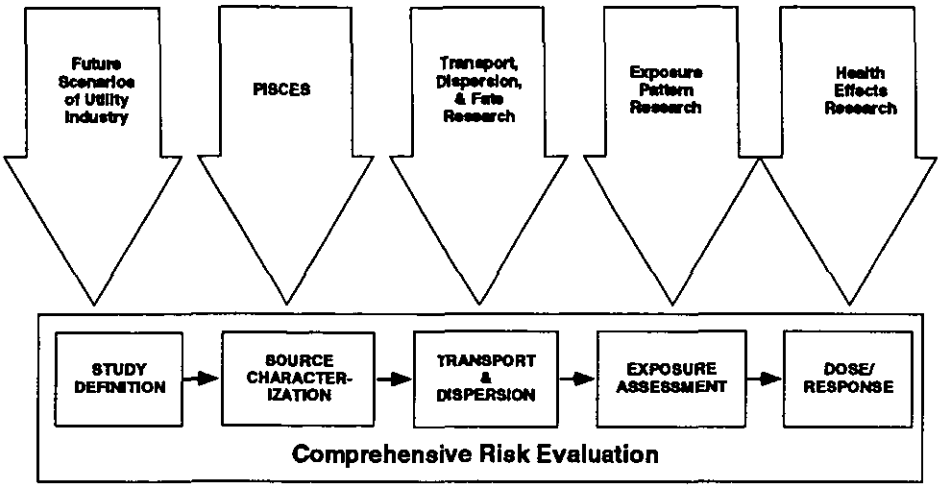

Figure 1-2.
Information Flow for Risk Assessment

1.4.1 Scope

EPRI has focused its trace substances research, and this report, on those substances known to be emitted by operational power plants and which may be potentially harmful to human health. The PISCES project provided key guidance in defining the scope of the overall trace substance research program. Through a preliminary literature survey, PISCES identified 24 substances of interest, some of these potentially toxic, as being most relevant for characterizing human health risks from utility emissions. These substances were identified as likely to be present in the liquid, solid, or gaseous discharges from United States power plants at levels representing potential concern, even after dispersion in the environment, and requiring an assessment of human health.

The PISCES focus differs from that of Title III of the CAAA, which contains a general list of 189 substances classified as "hazardous air pollutants," primarily because the PISCES literature survey indicated that utility sources emit only a small fraction of the CAAA-listed substances. The remaining CAAA-listed substances are assumed to be absent from power plant stack emissions or to be emitted in quantities that do not pose a significant human health risk when dispersed in stack plumes. Additionally, because the PISCES project is intended to address liquid and solid effluent streams in addition to stack emissions, the list of 24 substances addressed by the PISCES research includes some substances not listed in Title III.

Although field measurement data collected under the PISCES program address all 24 substances, the health risk assessment reported here focuses on a subset consisting of 16 chemicals. These substances encompass those that (a) were identified by PISCES as most

likely to be found in utility stack emissions and (b) have existing toxicity factors (for carcinogenic or non-carcinogenic effect, or both) that can be employed in carrying out a health risk assessment.

- Arsenic
- Benzene
- Beryllium
- Cadmium
- Chlorine
- Chromium
- Dioxins/furans
- Formaldehyde
- Lead
- Manganese
- Mercury
- Nickel
- PAHs
- Radionuclides
- Selenium
- Toluene

Not all of the 16 substances listed here were measured at every field site. For example, radionuclides and dioxins/furans were incorporated later in the program. Additionally, particular projects on trace substance environmental behavior focused on smaller subsets of this list of 16, or on a larger set of substances. These selections were primarily driven by lack of reliable information on physical, chemical, or biological properties of particular substances; such data are needed for a comprehensive understanding of their environmental transport, fate, and effects.

The analysis addresses the emissions of the selected trace substances and the ensuing risks to public health that would be posed by power plants after compliance with other provisions of the CAAA. Three scenarios were adapted for this study to project utility industry operations in a future scenario year, following compliance with all provisions of the 1990 CAAA. The scenarios originally involved SO_2 compliance modeling and a survey of planned modifications to particulate control equipment. For the base case scenario, an electric utility system-level SO_2 compliance scenario developed for EPA was disaggregated to the generating unit level. Two alternative scenarios were also used; these were developed for EPRI using different SO_2 modeling assumptions. All of the scenarios are described in Section 4.

1.4.2 Source Characterization

Source characterization involves understanding what substances are emitted from power plant stacks, in what forms, and at what rates. Prior to the PISCES project, reliable measurements of trace substances in emissions were not available for power plant fuel and emissions control configurations. A principal goal of PISCES is to understand and quantify how trace substances in fuels may be chemically transformed in the combustion process and partitioned through plant components into various gaseous, liquid, and solid discharge streams.

PISCES began with a thorough review of the literature on trace substance emissions and compiled a database of published emissions information. Much of the available data cited in the literature were gathered over several decades using a wide variety of dated, and sometimes ill-defined, sampling and analysis techniques, many of which have since been replaced with more accurate methods. Furthermore, relatively few literature citations included a full set of measurements of the fuel, control device removal efficiency, and emissions for the same plant.

The EPRI Field Chemical Emissions Monitoring (FCEM) program was begun as part of PISCES to improve available data on power plant emissions. In May 1990, the program began sampling for up to 24 substances in the process and discharge streams of selected power plants, representing different combinations of boiler, fuel type, and environmental control devices. By the end of 1993, FCEM had acquired measurements from 35 sites (including some measured under non-EPRI programs by one or more utilities). The U.S. Department of Energy (DOE) has conducted sampling and measurement programs at an additional 8 utility plants to date, using similar protocols.

The FCEM and corresponding DOE measurements constitute the best database currently available on trace substances released from power plants, and provide a credible empirical foundation for the balance of the risk assessment work. This database—in combination with information on trace substance content of fuels and projections of future utility industry operations—formed the basis for estimating emissions for U.S. electric utility steam generating units, as they might operate following compliance with all provisions of the 1990 CAAA.

Another key element of the PISCES program is the development and refinement of field sampling and analytical methods to measure trace substances at operating power plants. In some cases, early PISCES field tests used standard methods that were intended for use in less exacting environments. Problems with repeatability and high detection limits required reassessment of these procedures. On the basis of a review of sampling and analytical methods, and risk-based target detection limits, FCEM experimental field procedures were updated over time. Subsequent FCEM measurements reflect these improvements.

Although the PISCES data provide a sizable base for extrapolating emissions to unmeasured power plants, additional information was needed to characterize future emissions from these plants under plausible future operating scenarios. To understand the impact of future control technologies and other SO_2, NO_x, and particulate compliance strategies on emissions, a scenario of the industry was developed at a unit-specific resolution. To support the scenario, the FCEM site measurements of fuel trace substance composition were supplemented by broader sets of fuel analyses for substances of interest. The results of the fuel analysis research were combined with the PISCES field data and other research results in order to estimate utility emissions under projected future operating conditions.

1.4.3 Transport, Dispersion and Fate

EPRI research on the transport, dispersion, and fate of trace substances has focused on the chemical transformation and biogeochemistry of mercury, and intermedia transfer in general. For mercury, EPRI has carried out extensive research on the atmospheric chemistry, deposition, interaction with ecosystems, and aquatic dynamics.

To simulate the transport and fate of trace substances, EPRI used a combination of existing models and new EPRI research results. In order to simulate downwind concentrations due to stack emissions from more than 600 power plants, an atmospheric dispersion model was sought that could be used for repetitive runs with readily available data, and whose performance has been evaluated in field tests against observations. The Industrial Source Complex Long Term 2 (ISCLT2) model was selected to estimate long-term average concentrations of trace substances due to power plant emissions in an area surrounding each power plant.

Although ISCLT2 models the deposition of substances from the atmosphere to the ground level, it will not simulate transfers into ground and surface water systems, nor to crop plants or other entry points for human ingestion and other exposure pathways. EPRI instead developed a methodology for estimating the multimedia fate of substances emitted by power plants and their associated risks by all pathways. Under this approach, the EPRI multimedia model, TRUE (Total Risk of Utility Emissions) was used to assess the environmental transport and fate (and eventual exposure and risk) of trace emissions from several power plants.

1.4.4 Exposure

Estimating human exposure to trace substances requires combining ambient air concentration estimates and demographic data. The methodology and analyses presented in this report utilize exposure measures consistent with guidelines issued by the EPA and, more recently, by the National Academy of Sciences. The two principal measures that were evaluated were the maximum population-wide exposure across the population to average concentrations, and the exposure of a maximally exposed individual (MEI), that is, an individual who spends 70 years outdoors in the populated location with the highest concentrations due to power plant emissions. These measures incorporate sets of highly conservative assumptions about human presence and behavior that impact calculated exposures, doses, and risks.

The methodology used here also includes a more detailed assessment of exposure for the reasonably exposed individual (REI), as defined in Section 5. This assessment assumes plant replacement or modification after 55 years of operation and incorporates available data on "reasonable" human behavior (for example, time spent in indoor environments, which reduces exposure to some substances originating in utility sources outdoors). The methodology utilizes available information about where people live, the movement of

sensitive populations through specified indoor and outdoor environments, human activity levels in each such environment (a determinant of breathing rates), and the variation of trace substance concentrations for each such environment.

1.4.5 Health Effects

Health effects research addresses both the mechanisms and the likelihood of adverse health effects for a given dose of a chemical. Scientific information on the health effects of trace substance emissions from power plants is limited. Where health data on a particular chemical compound exist, the species, valence state, or physical matrix tested may differ significantly from the characteristics of that substance in power plant emissions. EPRI, EPA, and others have studies under way to develop a better understanding of the pharmacokinetic mechanism and bioavailability of emitted trace substances. One goal of the EPRI research is to improve the information base for species-specific dose-response models. To date, EPRI's program has focused on arsenic and mercury, where uncertainties in health effects are considered most critical for understanding the potential impacts due to electric utility emissions.

1.4.6 Risk Assessment

To guide the assessment of trace substance emissions, fate, effects, and risks, an integrating framework was developed. This framework (Figure 1-1) was used to synthesize results from a wide array of research programs, to evaluate the overall impacts of utility industry trace emissions on human health, and to assess how uncertainties in the available information impact the results.

EPRI's CORE and TRUE projects provide the overall framework and methodology for the risk assessment. CORE developed a method for assessing the nationwide cancer and noncancer health risks due to exposure by inhalation of trace substances. TRUE provides more detailed methods for assessing multimedia risks, addressing exposure to trace substances through air, food, and water via inhalation, dermal contact, and ingestion.

The risk assessment estimated trace substance emissions of 16 classes of substances (listed above) from electric utility steam generating units and the ensuing potential human health risks. The human health effects of concern for the selected chemicals, at the doses likely to arise due to utility emissions, are chronic or long-term in nature. Therefore, the risk assessment estimated noncarcinogenic and carcinogenic risks resulting from long-term inhalation exposure to the selected substances.

Risks were assessed for the estimated emissions of U.S. electric utility steam generating units over a 70-year timeframe, beginning in 2010. In general, the analysis included units expected to be operating in the year 2010, and assumed that they would continue to operate in the manner projected for 2010 throughout the 70-year timeframe. For the REI

measure, the analysis assumes that units are replaced after 55 years of operation with units that meet EPA's 1994 New Source Performance Standards (NSPS) for electric utility boilers.

Figure 1-3 depicts the scope of the analysis involved in each step of the risk assessment. As shown, the study focused on stack emissions of a specific set of trace substances, given scenarios describing the industry after compliance with other provisions of CAAA. The transport, dispersion and exposure assessment steps addressed the population within a 50-km radius of each plant, and focused on the inhalation pathway. Only those potential health risks associated with utility sources were considered.

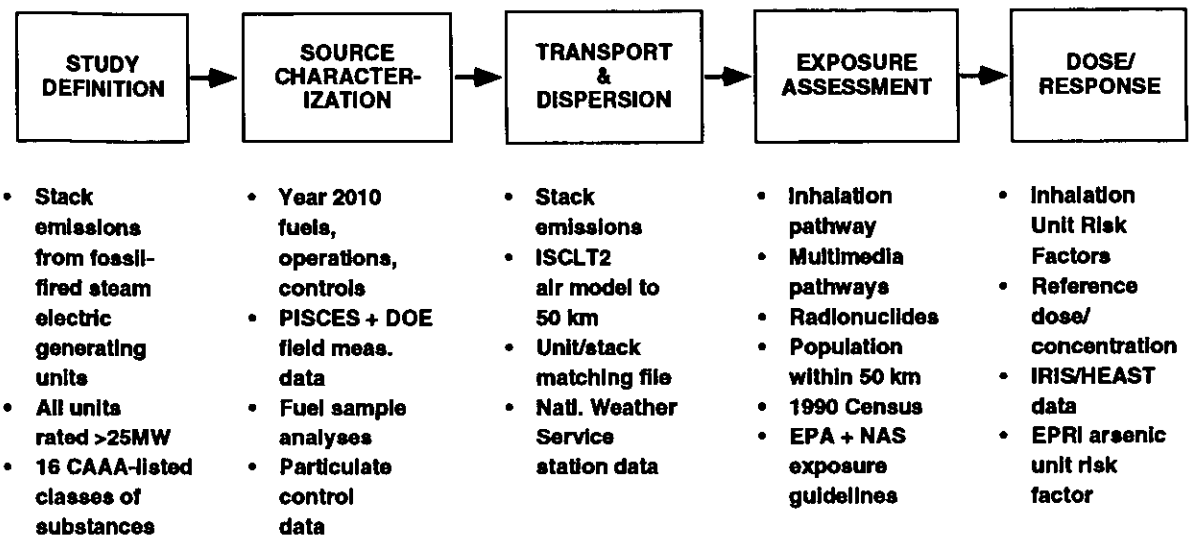

Figure 1-3.
Scope of Risk Assessment

The key data sources and models that were used in conducting the risk assessment are also shown in Figure 1-3. The primary source of data for emissions was the PISCES field measurements, which constitute the best available database on trace substance emissions from power plants; these were supplemented by available data from DOE plant measurements. Additional data used in the subsequent steps of the risk assessment were derived from national databases (e.g., census and meteorology data) or other published sources, such as the EPA's Integrated Risk Information System (IRIS) and Health Effects Assessment Summary Tables (HEAST).

To reconcile the need to carry out a broad range of analyses with the limited time available for the assessment, an overall risk assessment approach was developed that relies as much as possible on existing data and methods. The approach was structured to incorporate additional results that may emerge from ongoing research. The risk assessment methodology summarized in this report builds on the set of data and methodological

research results available as of early 1994. The methodology was designed to be iterative, beginning with a preliminary screening-level analysis based on a limited set of data. This analysis helped to guide development of the full methodology and to highlight the most important research needs. A key component of the assessment is estimation of ranges of uncertainty in the data and methods used. The report evaluates the sensitivity of the risk results to these uncertainties.

1.5 Overview of This Report

The following summarizes the contents of each remaining section of the main body of the report, and the attached technical appendices:

Main Body of Report

Section 2. Sampling and Analytical Methods

Describes the methods for trace substance sampling and sample analysis in operating power plants. Limitations due to field conditions, method detection limits, and other factors are discussed.

Section 3. Site Test Results: Field Data Presentation and Correlations

Describes available measurements; derives substance-specific emission factors for individual power plants measured under the PISCES project; and performs a parametric analysis of the field data, enabling the prediction of emissions from non-measured power plants.

Section 4. Industry Emissions Assessment

Describes the methodology used to combine the results of Section 3 with additional fuel analyses and unit-specific projections of power plant operations, in order to estimate emission rates for power plants in a future scenario year.

Section 5. Inhalation Exposure Assessment

Describes plume modeling of power plant stack emissions, demographic analyses to determine where people live in relation to atmospheric concentrations, and the methods for characterizing their exposure.

Section 6. Health Effects

Presents the current understanding of human health effects from key utility trace substance emissions, including interpretation of applicable regulatory guidelines, and provides information on ongoing health effect research efforts.

Section 7. Inhalation Risk Assessment

Describes the approach for integrating technical findings into an overall inhalation risk assessment, and presents the results from an industry-wide application of the approach.

Section 8. Multimedia Risk Assessment and Uncertainty Evaluation

Describes structure of the EPRI multimedia risk model, application of the model to multimedia carcinogenic and noncarcinogenic risks (including associated uncertainties), mercury exposure assessment, and the approach for radionuclide risk assessment.

Section 9. Insights and Conclusions

Summarizes the research conducted in support of the risk assessment effort, describes how the research was incorporated into the risk assessment, and summarizes the results and conclusions obtained from the risk assessment.

Technical Appendices

Appendix A. Sampling and Analytical Methods

Provides details of sampling and analytical methods for different types of substances and the derivation of risk-based detection limits.

Appendix B. Site Test Results: Field Data Presentation and Correlations

Provides detailed information on field test sites, presents results of field measurements for trace substances at coal, oil, and gas sites, and discusses calculations of emission factors, removal efficiencies, and correlation parameters.

Appendix C. Industry Emissions Assessment

Discusses allocation of coal data to individual generating units, and calculation of particulate emissions rates. Also compares modeled and measured emission rates, presents alternative industry scenarios, and provides sample emission calculations.

Appendix D. EPRI Mercury in Coal Study

Presents results of a study addressing new analytical methods for determining mercury concentrations in coal and analyzing "as-fired" coal samples for mercury content using this method.

Appendix E. Alternative Exposure Methodology

Provides detailed discussion on the approach, and assumptions and limitations of the calculation of the Reasonably Exposed Individual (REI).

Appendix F. Complex Terrain Considerations

Discusses an analysis carried out to indicate the approximate magnitude of the difference between concentrations due to dispersion in flat terrain and those in rolling (complex) terrain.

Appendix G. EPRI Health Research

Summarizes EPRI research in health effects due to arsenic and mercury, discussing issues such as valence state, pharmacokinetic models, and bioavailability.

Appendix H. Unit Risk for Arsenic

Presents the EPRI report on an updated calculation of the arsenic unit risk factor, reflecting recent re-analyses of data.

Appendix I. Inhalation Risk Assessment

Provides details on the computational framework of the risk assessment, covering MEI and REI risk and hazard quotient calculations, and a detailed analysis of the plants with risk outcomes.

Appendix J. TRUE Multimedia Risk Assessment Model

Provides detailed TRUE model description.

Appendix K. TRUE Multimedia Risk Assessment Case Studies

Presents details of the screening-level multimedia health risk assessments associated with emissions of four power plants.

Appendix L. Radionuclides

Discusses revisions to the EPA radionuclide risk assessment methods, source terms, and calculation of radionuclide risks through the food chain.

Appendix M. Uncertainty Analysis

Provides detailed discussion of the approach to probabilistic uncertainty analysis and its application to two coal-fired power plants for carcinogenic chemicals and mercury.

Appendix N. Exposure to Mercury Via Fish Consumption

Reviews the mercury levels found in seafood, and estimates human exposure to mercury via consumption of fish and shellfish.

Appendix O. Mercury in the Environment

Presents this separate draft EPRI report which discusses mercury sources, mercury in the atmosphere and in aquatic systems, health effects of mercury, mercury risk assessment, and possible mercury controls.

Appendix P. Toxicology Profiles

Provides a summary of technical literature on the health effects of the 16 primary substances that are covered in this study.

2

SAMPLING AND ANALYTICAL METHODS

This section presents the sampling and analytical methods used in EPRI's Power Plant Integrated Systems: Chemical Emissions Studies (PISCES) Field Chemical Emissions Measurement (FCEM) program as well as those used in the similar DOE field program. It is intended to describe the methods used, their inherent limitations, the modifications made, and the quality and uncertainty associated with data obtained.

2.1 Overview

Initially, the FCEM project was designed to determine the distribution and fate of selected chemicals in power plant process streams, and to achieve a mass balance for the chemicals across the power plant system. After enactment of the 1990 Clean Air Act Amendments, the focus of the program was shifted to assess trace substance emissions from power plant stacks. The trace substance sampling and analysis methods initially chosen for the program were the most current EPA-recommended methods and were perceived to be appropriate techniques based on precision, bias, repeatability, and agency and industry acceptance. In many cases, these methods had not been validated (as defined by EPA Method 301 validation protocol [1]) for power plant streams where trace substances are found in very low concentrations. Further, while some of these chosen techniques had been used regularly in other applications, they had to be modified for fossil-fuel-fired utility conditions. Many sampling and analytical methods used for the field data collection have been pushed to their limits of capability. In all cases, the data obtained met traditional quality assurance (QA) and quality control (QC) criteria. DOE undertook their field sampling program in 1993, to complement EPRI's FCEM program and to expand the database of field results. The sampling and analytical methods of the DOE program were essentially the same as those used earlier in the EPRI FCEM program.

2.2 Sampling and Analysis Methodology

2.2.1 Summary of the Methods Used

The methods initially used for sampling utility power plant process streams, and the techniques used to preserve, prepare, and analyze the resulting field samples are summarized in this section. The sampling and analytical protocols were reviewed and approved by EPA. In the EPRI and DOE field programs, attention was focused on measurement of

chemicals in the fuel as well as in combustion flue gas and stack gas. Chemical composition of collected ash was used only in limited instances to derive mass balances for certain chemicals when difficulties were encountered in fuel or flue gas chemical measurements.

2.2.1.1 Fuels.

Samples of fuel were collected, divided as appropriate, and prepared for several types of inorganic constituent analyses. Whenever possible, time-averaged composite samples were collected from the fuel delivery system. Coal samples were collected with an automatic sampler that met ASTM guidelines or by grab sampling with a scoop from either the conveyor system or feed lines around the coal pulverizers. Fuel oil was typically collected as grab samples from taps on the pipeline to the burners. (Details of the general preparation and analytical steps used for fuels are presented in Figure A-1 of Appendix A).

Measurement of trace elements in coal is difficult because of the variability of major and trace element content in different coals and the difficulty in measuring small quantities of trace elements in the coal matrix [2,3]. A number of analytical techniques were used at some sites to obtain the required analytical detection limits and accuracy for all elements of concern. Table 2-1 shows the required sample preparation procedures for each analytical technique, and the analytes measured by each technique. Some sample preparation techniques (e.g. SW-846 Method 3050 [4]) used at some of the DOE-tested sites may not provide a complete digestion of the coal ash material; the procedure involves open digestion with HNO_3 and may result in a loss of mercury. Results obtained by analysis of the digested solution by CVAAS (SW-846 Method 7471 [5]) may therefore be biased low.

Table 2-1.
Analytical Techniques for the Analysis of Inorganics in Fuel

Technique	Analytes[a]	Required Preparation	Notes
Instrument Neutron Activation Analysis (INAA)	As, Ba, Cd, Cr, Co, Cu, Mn, Mo, Ni, Se, V, Al, Ca, Fe, Mg, K, Na, Ti	Grinding may be required.	Detection limits for INAA are often equivalent to other multi-element techniques.
Inductively Coupled Plasma Emission Spectrophotometry (ICP-AES)	Be, Ba, Co, Cu, Cl, Mn, Mo, V, Al, Ca, Fe, Mg, K, Na, Ti	Fuel dissolution into an acid matrix is performed prior to analysis. Ashing followed by acid digestion has also been used.	This technique provides adequate sensitivity (although, in general, AA detection limits are lower).

Table 2-1.
Analytical Techniques for the Analysis of Inorganics in Fuel (Continued)

Technique	Analytes[a]	Required Preparation	Notes
Atomic Absorption Spectrophotometry—Graphite Furnace (GFAAS), Cold Vapor (CVAAS), Hydride Generation (HGAAS)	As, Cd, Pb, Hg, Ni, Se	Fuel dissolution into an acid matrix is performed prior to analysis. Alternately, the fuels are combusted and the vapor concentrated prior to analysis. This is usually accomplished using an oxygen bomb technique for ashing prior to digestion.	AA provides much lower detection limits than are available for multi-element techniques. In addition, it is less susceptible to interference by high concentrations of other analytes in the matrix.
Ion Chromatography (IC) or Potentiometry by Specific Ion Electrode (SIE)	Cl, F	Closed oxygen bomb is used to dissolve the analytes in an aqueous matrix. The combustion ash and bomb rinses are then analyzed.	This analytical technique results in detection limits of between 20 and 100 μg/kg for lower sensitivity. A deionized water preparation is sometimes used.

[a] These are the analytes that have been tested as a part of the PISCES project, not necessarily all of the analytes the analytical technique is capable of detecting.

2.2.1.2 Flue Gases.

Four primary classes of hazardous air pollutants are measured in flue gas, each requiring separate sample collection and preparation steps. The four classes are: (1) inorganic analytes (metals, HCl, HF), (2) volatile organic compounds, (3) semivolatile organic compounds (including polychlorinated dibenzodioxins and dibenzofurans (PCDD/PCDF), and (4) aldehydes. Details of the flue gas sampling and analytical methods are provided in Appendix A.

Most of the sample collection techniques used for gas streams were standard reference methods designed for general combustion sources. Table 2-2 specifies the sampling and analytical methods initially employed for the trace substances in fuel and flue gas. Radian Corporation and Carnot Corporation together performed the vast majority of the EPRI FCEM program measurements presented in this report, with Radian performing most of the coal-site measurements and Carnot most of the oil-site measurements. For some chemicals, such as PAHs in flue gas, Carnot employed a method endorsed by the California Air Resources Board (CARB) while Radian employed the corresponding EPA method. This choice resulted from Carnot's greater familiarity and experience with CARB methods. Both approaches yielded similar results.

Table 2-2.
FCEM Sampling and Analytical Methods Summary*

Matrix	Analytes	Sampling Approach (Reference)	Analytical Technique (Reference)
Coal-Fired Plants			
Fuel	As, Ba, Be, Cd, Ca, Cr, Co, Hg, Pb, Mn, Mo, Ni, P, Se, Ti, and V	ASTM auto sampler or alternative ASTM D 2234 [1] and D 2013; Grab-Composite Sampling (Method S007 [2]).	As (SW-846 7060) [3]; Cd (SW-846 7131) [4]; Pb (SW-846 7421) [5]; and Se (SW-846 7740) [6]; Hg (DGAA) [7]; other metals by ICPES (SW-846 6010) [8]; metals determined after ASTM D 3683-78 digestion [9].Hg (SW-846 3050 [10], 7471 [11]); As, Se (ASTM D4606 [12]); As, Cd, Se (ASTM D 3864 [13]); SW-846 7060 [14], 7131 [15], 7740 [6]); INAA [16]; Be, Pb, P (ASTM D 3863 [17]; SW-846 6010 [8]); Hg (CVAAS [19]).
	Ash Mineral Analysis		XRF ASTM D 4326 [20].
	Cl, F, PO_4		Cl (ASTM D 4208-88) [21]; F (ASTM D 3761-91) [22]; F (CONSOL prep. [23] /IC); Cl (LECO Cl350 analyzer [24], ASTM D 4208 [21]); PO_4 (ASTM D 3682-78 [25]; SW-846 3050; [10]; ICP; colorimetric).
	S		ASTM D 3177-89 [26]; ASTM D 3177.
	C, H, N, O, moisture, ash, HHV (ultimate and proximate)		Ultimate (ASTM D 3176 [27]), Proximate (ASTM D 3172 [28], D 5142-90 [29]]); HHV (ASTM D 1989-91 [31], D 2015 [32]); C, H, N (ASTM D 5373 [33]); Ash (ASTM D 3174 [34]).
	Radionuclides		Alpha Spectrometry (Method 114) [35]; DOE Pb-01, Po-01, U-02; LANL ER200 [36]; EPA 904.0 [37], 908.1 [38].

Table 2-2.
FCEM Sampling and Analytical Methods Summary* (Continued)

Matrix	Analytes	Sampling Approach (Reference)	Analytical Technique (Reference)
Coal-Fired Plants (Continued)			
Flue Gas	As, Ba, Be, Cd, Ca, Cr, Co, Cu, Pb, Mn, Hg, Mo, Ni, P, Se, Ti, and V	Isokinetic Sampling Train - Liquid Sorbents (Multi-Metals Train) [39]; Draft Method 29 [40]; Carbon adsorption (HEST[41]).	Inductively Coupled Plasma Emission Spectrophotometry—ICPES. Ba, Be, Ca, Cr, Co, Mn, Mo, Ni, P, Ti, and V (SW-846 6010 [8]). Thin layer XRF [20].
			Graphite Furnace Atomic Absorption Spectrophotometry — GF-AAS. As (SW-846 7060 [14]); Cd (SW-846 7131 [15]); Pb (SW-846 7421 [5]); Ni (EPA-600 249.2 [42]); and Se (SW-846 7740 [6]).
			Cold Vapor Atomic Absorption Spectrophotometry—CVAA. Hg (SW-846 7470 [19]).
			SW846 3010 [43], 3020m [44], 7470 [19]; M29 [40]; INAA [16].
	Formaldehyde and Acetaldehyde	Isokinetic Sampling Train —Liquid Sorbent (Derivation of Method 5); Method 0011; EPA M5 [46].	High Performance Liquid Chromatography—HPLC (BIF Reference 0011 [47]); Method TO-5 (HPLC/UV [48]).
	Polycyclic Aromatic Hydrocarbons 5-methyl chrysene, 7H-dibenzo(c,g)carbazole, acenaphthene, acenaphthylene, anthracene, benz(a)anthracene, benzo(a)pyrene, benzo(b,j,k)fluoranthenes, benzo(g,h,i)perylene, chrysene, dibenz(a,h)acridine, dibenz(a,h)anthracene, dibenz(a,i)acridine, dibenzo(a,e)pyrene, dibenzo(a,h)pyrene, dibenzo(a,i)pyrene, fluoranthene, fluorene, indeno(1,2,3-cd)-pyrene, phenanthrene, pyrene	Isokinetic Sampling Train—Solid Sorbent— Modified Method 5 (Method 0010 [49]); (Adaptation of Method 23 [54]).	Gas Chromatography/High-Resolution Mass Spectrometry — GC/HRMS (adaptation of SW-846 Method 8290 [53] and Method 23 [54]); GC/MS (Method 8270 [55]).

Table 2-2.
FCEM Sampling and Analytical Methods Summary* (Continued)

Matrix	Analytes	Sampling Approach (Reference)	Analytical Technique (Reference)
Coal-Fired Plants (Continued)			
Flue Gas (Continued)	Volatile Organic Compounds (specifically benzene, toluene, xylene)	Nonisokinetic Sampling Train—Solid Sorbent—VOST (Method 0030 [52])	Gas Chromatography/Mass Spectroscopy —GC/MS (SW-846 Method 5041 [57]/8240, Method 5040/8240 [58]).
	Acid gases (HCl, SO_2/SO_3, HF)	Isokinetic Sampling Train—Liquid Sorbents—Anions Train (Adaptation of Method 5; Method 26A).	Cl^-, SO_4^- Ion Chromatography —IC (EPA 300.0 [59]) , Method 9056); F^- Specific Ion Electrode-SIE (EPA 340.2 [60]).
	Ammonia and Cyanide	Isokinetic Sampling Train - Liquid Sorbents - (Adaptation of Method 5).	NH_3, (EPA 350.3 [61]); CN^- (EPA 9010 [62]).
	Mercury Speciation—[Hg(0), Hg(II), CH_3HgX]	Nonisokinetic Sampling Train—Solid Sorbents (Proprietary Developmental Method of Frontier Geosciences) [63]; Iodated Carbon Sorbent.	Cold Vapor Atomic Fluorescence Spectroscopy (Proprietary Developmental Method of Frontier Geosciences) [63]).
	Dioxins (PCDDs) and Furans (PCDFs)	Isokinetic Sampling Train —Solid Sorbent (Method 23 [54])	High Resolution Gas Chromatography/Mass Spectrometry—GC/HRMS (Method 23).
	Radionuclides (Particulate)	Isokinetic Sampling (EPA Method 5; Method 17 [37]).	Alpha Spectrometry (Meth. 114 [35]); Gamma Spectrometry.
Solid Feedstocks, Ashes, and Other Solid By-products	As, Ba, Be, Cd, Ca, Cr, Co, Cu, Pb, Mn, Hg, Mo, Ni, P, Se, Ti, and V	ASTM autosampler[h] or alternative; Grab-Composite Sampling (S007 [2]).	Microwave Digestion (SW-846 3051 [64] - Mod.). Acid Digestion (SW-846, 3050 [10]) for limestone sorbents and gypsum solids only; not for ash.
	Cl, F		Cl (ASTM D 4208-88); F (ASTM D 3761-84), or EPRI FGD Handbook Digestion [65] followed by IC (EPA-300.0) or SIE (EPA 340.2).

Table 2-2.
FCEM Sampling and Analytical Methods Summary* (Continued)

Matrix	Analytes	Sampling Approach (Reference)	Analytical Technique (Reference)
Oil-Fired Plants			
Fuels	As, Ba, Be, Cd,, Cr, Co, Cu, Pb, Mn, Hg, Mo, Ni, P, Sb, Se, and V.	Grab-Tap Composite Sampling (Method S004 [66]).	ICPES (SW-846 6010 [6]); Graphite Furnace Atomic Absorption Spectrophotometry- GFAAS; Pb (SW-846 7421 [5]); INAA; Hg (CVAAS).
	Cl, F		Cl (ASTM D 808-87 [68]).
	S		ASTM D 129-64 [69], ASTM D 4294 [70].
	Composition and Heating Value		Water (ASTM D 95 [7 1]); Ash (ASTM D 482 [72]); HHV (ASTM D 240-87 [73]).
Flue Gas	As, Ba, Be, Cd, Co, Cr, Cu, Hg, Mn, Mo, Ni, P, Pb, Se, and V.	Isokinetic Sampling Train (Multi-Metals Train).	Inductively Coupled Plasma Emission Spectrophotometry - ICPES. Ba, Be, Cd, Cr, Co, Cu, Mn, Mo, Ni, P, Ti, and V (SW-846 6010); Cr (Flame AAS - SW-846 7190 [73]).
	Formaldehyde	Isokinetic Sampling Train (CARB 430 [74]).	CARB 430 [74].
	Particulate, SO_2, Cl^-, F^-, PO_4^{3-}	Isokinetic Sampling Train (EPA Method 5/8 or CARB 421 [75]).	CARB 421.
	Polycyclic Aromatic Hydrocarbons	Isokinetic Sampling Train (CARB 429 [76]).	High Resolution Gas Chromatography/Mass Spec. (CARB 429).
	Volatile Organic Compounds; benzene and toluene	Tedlar bags (CARB 410A [77]).	CARB 410A, preconcentration as part of 410A.
	Mercury Speciation	Proprietary Development Method, Frontier GeoSciences [63].	Cold Vapor Atomic Fluorescence Spectroscopy.

Table 2-2.
FCEM Sampling and Analytical Methods Summary* (Continued)

Matrix	Analytes	Sampling Approach (Reference)	Analytical Technique (Reference)
Oil-Fired Plants (Continued)			
Flue Gas (Continued)	Dioxins/Furans	Isokinetic Sampling Train Solid Sorbent (Method 23).	High Resolution Gas Chromatography Mass Spectrometry —GC/HRMS (Method 23).
Solid Feedstocks, Ashes, and Other Solid By-Products	As, Ba, Be, Cd, Ca, Co, Cr, Cu, Hg, Mn, Mo, Ni, P, Pb, Ti, Se, and V	Composited Grab Samples (Method S007).	Ba, Be, Cd, Ca, Cr, Co, Cu, Mn, Mo, Ni, P, Ti, and V (SW-846 6010); As (SW-846 7060); Pb (SW-846 7420 [78]); Hg (SW-846 7471); and Se (SW-846 7740).
	Polycyclic Aromatic Hydrocarbons[d]		HRGC/MS (CARB 429); GC/MS (SW-846 8270 [55]).
	Cl^-, F^-, PO_4^{3-}, and S		Cl^-, F^-, IC (EPA 300.0)- SIE (EPA 340.2); S (LECO SC-132, ASTM D 4239 [80]).
	Radionuclides		Alpha Spectrometry (Method 114 [35]).
Natural Gas-Fired Plants			
Fuel	Trace Elements	Grab Sampling.	Gas Chromatography/Atomic Emission Detector - GC/AED.
	Major Hydrocarbons		Gas Chromatography/Flame Ionization Detector - GC/FID (ASTM D 1945 [81]), Thermal Conductivity Detector - GC/TCD (ASTM D 1946 [82]).
	Non-Hydrocarbons (0_2, H_20, He, H_2, N_2, CO_2, CO)		Gas Chromatography/Flame Ionization Detector - GC/FID (ASTM D 1945 [81]), Thermal Conductivity Detector - GC/TCD (ASTM D 1946 [82]).
	Nitrogen Compounds		Gas Chromatography/Atomic Emission Detector - GC/AED.
Natural Gas-Fired Plants (Continued)			

Table 2-2.
FCEM Sampling and Analytical Methods Summary* (Continued)

Matrix	Analytes	Sampling Approach (Reference)	Analytical Technique (Reference)
Fuel (Continued)	Sulfur Compounds		Gas Chromatography/Flame Photometric Detector - GC/FPD
	Oxygenated Compounds		Gas Chromatography/Atomic Emission Detector - GC/AED; High Performance Liquid Chromatography - HPLC.
	Halocarbons		Gas Chromatography/Electron Capture Detector - GC/ECD.
	C5 + Hydrocarbons		High Resolution Gas Chromatography/Flame Ionization Detector - HRGC/FID.
Flue Gas	As, Ba, Be, Cd, Cr, Co, Cu, Pb, Mn, Hg, Mo, Ni, P, Se, and V	Isokinetic Sampling Train 0 Liquid Sorbents (Multi-Metals Train [18]); Draft Method 29.	Inductively Coupled Plasma Emission Spectrophotometry - ICPES. Ba, Be, Cd, Cr, Co, Cu, Mn, Mo, Ni, P, Ti, and V (SW-846 6010).
			Graphite Furnace Atomic Absorption Spectrophotometry - GF-AAS. Pb (SW-846 7421).
			Hydride Generation Atomic Absorption Spectrophotometry - HG-AAS. As (SW-846 7061 [83]); and Se (SW-846 7741 [84]).
			Cold Vapor Atomic Absorption Spectrophotometry - CVAA. Hg (SW-846 7470 [19]).
	Formaldehyde	Non-Isokinetic Sampling Train (CARB 430).	High Performance Liquid Chromatography - HPLC (CARB 430).

Table 2-2.
FCEM Sampling and Analytical Methods Summary* (Continued)

Matrix	Analytes	Sampling Approach (Reference)	Analytical Technique (Reference)
Natural Gas-Fired Plants (Continued)			
Flue Gas (Continued)	Polycyclic Aromatic Hydrocarbons[d] and Polychlorinated Biphenyls. naphthalene, acenaphthene, acenaphthylene, anthracene, benzo(a) anthracene, benzo(a)pyrene, benzo(b,k)fluoranthenes, benzo(g,h,i)perylene, chrysene,dibenzo(a,h)anthracene, 7,12-dimethylbenz(a)anthracene, fluoranthene, fluorene, 3-methylchloranthrene, 2-methylnaphthalene, phenanthrene, indeno(1,2,3-cd)-pyrene, pyrene	Isokinetic Sampling Train (CARB 429, CARB 428 [85]).	High Resolution Gas Chromatography/Low Resolution Mass spectrometry with Selective Ion Monitoring - HRGC/LRMS-SIM (CARB 429, CARB 428).
	Volatile Organic Compounds (specifically benzene and toluene)	Tedlar bags (CARB 410A).	Gas Chromatography/Mass Spectroscopy - GC/MS (CARB 410A, EPA TO-14 [86]).

*References cited in this table are listed at the end of Technical Appendix A.

It is useful to know the speciation (valence state) of some elements, particularly mercury, arsenic, and chromium, in flue gas emissions because toxicity by a given route of exposure and via a given mode of action depends on the valence state of the substance. However, the required speciation methods for these measurements have not been fully developed and tested.

Speciation of mercury emissions provide improved understanding of the wet and dry deposition patterns (primarily the downwind distance to deposition) associated with mercury emissions from power plants, as well as the potential for effectiveness of various control devices for removal from the gaseous stream. At some of the field test sites, a solid sorbent method (MErcury Speciation Adsorption, or MESA, method) that is under development by Frontier Geosciences [6] was used to measure mercury speciation (i.e., elemental mercury and oxidized mercury in vapor phase) in flue gas. Further details of this method are provided in Appendix A. Total mercury concentrations (sum of the different vapor phase mercury species) from the speciation method have also been used to provide a comparison with mercury measurements from the EPA (Draft) Method 29 [7] (multi-metals method). Sorbent temperature appears to be a critical parameter in the MESA method; ongoing developmental work on the method continues to better define the effects of sampling trap temperature on mercury speciation. Careful work needs to be done to examine and address the potential for mercury species conversion during the sample extraction and capture steps of EPA Method 29, as well as for other methods.

Chromium can exist in multiple valences states. The trivalent state is the most stable form, found in nearly all chromium ores. The hexavalent state is relatively unstable due to its strong oxidizing potential; this form is currently of interest as an EPA-listed inhalation carcinogen. Quantitative measurements of hexavalent chromium in utility flue gas has been difficult because of reactivity of the species. Limited hexavalent chromium measurements in utility flue gas have been made in the EPRI FCEM program using EPA Method 0013 [8]. This method is based on EPA Method 5 with some modifications. The primary such modification is the use of a recirculating train in which the impinger reagent (potassium hydroxide) is continuously recirculated to the nozzle to minimize reduction of Cr(VI) to Cr(III) between the nozzle and the impingers.

However, a recent effort [9] to validate the method (according to the protocol for EPA Method 301) for chromium speciation in an oil-fired utility stack gas was largely unsuccessful; this was likely caused by the lack of precision in measuring the very low concentrations of Cr(VI) at this source. Further work needs to be carried out to develop and validate a suitable chromium speciation method for utility stack gas emissions.

No chemical speciation of arsenic in combustion flue gas was performed during the EPRI or DOE field tests because no reliable methods for doing so were available. However, studies were performed on speciation of arsenic in captured fly ash particles to assess the speciation and physical form for future health assessments. These studies are described in Section 6 and in Appendix G.

2.2.1.3 Difficulties in FCEM Sampling and Analysis

Several complications were encountered in sampling and analysis during the course of the FCEM program.

- There are few validated procedures for sampling large-volume solid streams such as coal and ash that are non-homogeneous. For example, when an auto-sampler was not available, small grab samples had to be collected that might not have been representative of the whole stream.
- Some gas sampling locations did not meet the requirements of EPA Method 1 [9]. For instance, when two boilers shared a common stack, samples were collected in one of the ducts prior to the stack. These samples were difficult to collect and might not have been representative of the boiler tested.
- Because matrices of process streams may differ across a control device (e.g., the inlet to a particulate control device is high in dust loading, while the outlet has much lower levels of dust), inlet and outlet concentrations for a given chemical may not be directly comparable.
- Extended gas sampling times (i.e., large volumes) had to be used to enable the multi-metals train to collect sufficient samples to detect the low levels of trace substances found in flue gas. This resulted in overloading of the particulate filter as well as in high permanganate consumption. In some cases, these adversely affected the measurement results.

2.2.1.4 Method Modifications

Modifications were made to various sampling and analytical methods to (1) overcome some of the difficulties experienced, (2) address method limitations, and (3) to improve performance. Two examples are:

- Sample collection and handling procedures for EPA Method 5 were modified when sampling a gas stream with a high particulate loading. Collecting samples at the inlet to a particulate control device necessitated an EPA Method 17 [11] approach using a thimble-shaped filter placed in-stack, increasing the capacity for collecting solids and allowing the specified sampling period to be completed.
- Sampling periods specified in the standard methods were often increased to three hours at the inlet and/or more at the outlet to increase the gas volume sampled and to provide lower detection limits for elements of interest. The associated greater moisture condensate volumes required that an additional impinger be added to collect overflow from the first impinger in the sampling train.

Table 2-3 describes the important method modifications that were made along with the rationale for those modifications. In many cases the implementation of these modifications depends on the judgment of the field sampling crew, increasing the need for an experienced and knowledgeable team on-site and extensive documentation of all work.

Table 2-3.
Sampling and Analytical Method Modifications for Fossil-Fuel-Fired Power Plant Application

Stream	Analyte(s)	Method		
			Difficulty / Limitation / Need for Improvement	Modification & Rationale
Coal-Fired Plants				
Fuels	All	Sample Collection	Few validated or accepted procedures available for representative sampling of large volume, non-homogeneous solid streams such as coal.	Preferred approach is the use of an ASTM autosampler. If this is not available at the site, samples can be collected by scoop, from a pile, from a drop point, or from a moving belt. If an autosampler is not available, best efforts need to be made to get a representative sample.
			Excessive quantities of sample collected.	Sample collection frequency reduced.
	Hg	Bureau of Mines Double Gold Amalgamation Cold Vapor Atomic Absorption	Interference from combustion products resulting in low Hg recoveries.	$SnCl_2/H_2SO_4$ scrubber. between combustion bomb and gold wire. Concentrates Hg on gold while scrubbing potential chemical interferants.

Table 2-3.
Sampling and Analytical Method Modifications for Fossil-Fuel-Fired Power Plant Application (Continued)

Stream	Analyte(s)	Method	Difficulty / Limitation / Need for Improvement	Modification & Rationale
Coal-Fired Plants (Continued)				
Flue Gases	As, Ba, Be, Cd, Ca, Cr, Co, Pb, Mn, Hg, Mo, Ni, P, Se, Ti, V	Multi-Metals (Sampling)	Need greater detection sensitivity.	Reduce impinger rinse volumes. Provides maximum method sensitivity.
			Difficulty in calculating the total mercury , when the first permanganate impinger has detectable concentrations and the second impinger and rinses have non-detect levels.	Combine all mercury-only permanganate fractions and rinses. Minimizes the possibility of having to mathematically combine detected values (from first impinger) with non-detect results (from the second impinger).
	Volatile and Semi-volatile organics, particulate matter.	0030 (VOST); 0010/23 - MM5; M5 (PM, Rads etc.)	High SO_2 levels.	Included additional H_2O_2 impinger to protect equipment.
	Metals, acid gases, particulate matter	M29 Multimetals; M5 (PM, Rads, etc.); M26 (HCl, HF); M13 - BIF (Cr^{+6})	High SO_2 levels.	Modified impinger solutions (used higher concentrations of peroxide solutions) to account for SO_2.
	Metals, Semi-Volatile organics, and PCDD/ PCDF.	M29; 0010/23 - MMT	High particulate loading.	Added glass cyclone upstream of filter.
	Acid gases	M26	Excess moisture due to increased sample volume.	Used larger impingers.
			Necessary levels of quantitation are very low, and background levels of filter media are significant.	More analysis of blank filters. Multiple blank filter analyses provides statistically-defensible blank correction.

Table 2-3.
Sampling and Analytical Method Modifications for Fossil-Fuel-Fired Power Plant Application (Continued)

Stream	Analyte(s)	Method		
			Difficulty / Limitation / Need for Improvement	Modification & Rationale
All Gases and Process Solids	Hg	SW-846 7470, 7471	1) Insufficient permanganate (solution) used in method to consume the excess peroxide that interferes with Hg analysis.	1) Solid $KMnO_4$ added for excess. Solid $KMnO_4$ consumes excess H_2O_2 in HNO_3/H_2O_2 impingers without significantly increasing sample volume.
			2) Need improvement in heating samples.	2) Samples treated in oven instead of water bath. More samples can be heated as efficiently as with water bath.
	Semivolatile organics, metals, acid gases, and particulate matter	Semivolatiles (0010); Metals (M29); PM/ Anion/Rads	Need to enhance in-stack pollutant detection limits.	Sampling periods at baghouse outlet increased.
	Semi-volatile organics	Semivolatiles (0010)	Need lower analytical detection limits.	Used HRGC/HRMS instead of HRGC/LRMS.
All Gases	All "isokinetic" analytes (metals, PAH, acid gases, etc.)	Multi-Metals (Sampling), Semi-VOST, Method 23, Formaldehyde, etc.	Sampling locations are frequently difficult to reach, and require vertical sampling.	Use Teflon transfer line before impinger train. Use of Teflon transfer line eases sampling difficulty, and results in satisfactory data quality with a cost savings.
Hi-dust Gases	All "isokinetic" analytes (metals, PAH, etc.)	Multi-Metals, Semivols, VOST, etc.	Particulate loads on hi-dust streams (e.g., inlet to a baghouse) make sampling with a standard train configuration very difficult.	Use thimbles, in-stack filters, or pre-sampling filters, as necessary. Modification allows the use of sampling apparatus designed to deal with higher particulate loading.

Table 2-3.
Sampling and Analytical Method Modifications for Fossil-Fuel-Fired Power Plant Application (Continued)

Stream	Analyte(s)	Method	Difficulty / Limitation / Need for Improvement	Modification & Rationale
Coal-Fired Plants (Continued)				
All Gases	All "isokinetic" analytes (metals, PAH, etc.)	Multi-Metals, Semivols, VOST, etc.	Collection of sample at locations not meeting the minimum criteria of EPA Method 1. Sampling locations too close to flow disturbances, and / or multiple traverses not possible.	More sampling points are added , when possible, to address potential stratification. When multiple traverses not possible, choose sampling point as close to center of flow as possible.
	All "isokinetic" analytes (metals, PAH, etc.).	Multi-Metals, Semivols, VOST, etc.	Need lower detection limits for analytes of interest.	Sampling periods specified in the method were often increased to three hours. This sometimes required the addition of an impinger to collect condensate. This provided lower detection limits for analytes of interest.
	As, Pb, Cd, Se	Digestion SW 846 3020	This digestion used for GFAA metals. Hydrochloric acid causes fracturing of graphite tubes during combustion heating, shortening the tube life.	Use nitric acid only. No hydrochloric acid. Hydrogen peroxide replaces hydrochloric acid as an oxidizer in the digestion.
	Ba, Be, Ca, Cr, Co, Mn, Mg, Ni, P, Ti, V	SW-846 3005	Desirable to have more QA/QC samples, and enhanced safety.	Modification uses 50 ml sample. Smaller aliquots allow more QA/QC replicates. HNO_3 and HCl acids at 1:1 aqueous dilution not concentrated. 1:1 acids safer with same digestion efficiency.
	PAH	CARB 429 High Resolution GC/ MS	Potential interference from the use of internal spike material that may exist in the stack gas.	Use deuterium-spiked compounds. Method is a modification of SW-846. Deuterated compounds are available for internal and surrogate spikes.

Table 2-3.
Sampling and Analytical Method Modifications for Fossil-Fuel-Fired Power Plant Application (Continued)

Stream	Analyte(s)	Method	Difficulty / Limitation / Need for Improvement	Modification & Rationale
Oil-Fired Plants				
Flue Gases	Metals are As, Ba, Be, Cd, Cr, Co, Cu, Pb, Mn, Hg, Mo, Ni, P, Se, and V	Multi-Metals Method 29	Improve method sensitivity.	Reduced impinger rinse volume.
	Metals are As, Ba, Be, Cd, Cr, Co, Cu, Pb, Mn, Hg, Mo, Ni, P, Se, and V	Multi-Metals Method 29	Improve method sensitivity.	Combine particulate and HNO_3/H_2O_2 impinger digestates for analysis.
	Formaldehyde	CARB 430	Prevent particulate from collecting in the impingers.	Use glass fiber filter.
	Benzene and toluene	CARB 410A	Prevent particulate from collecting in Tedlar bags.	Use glass wool plug in probe.
	All analytes except benzene and toluene	—	Very large ducts require very long probes. Glass is too fragile to be effectively used.	Use Teflon liner at ESP inlet locations.

2.2.1.5 Evaluation of Flue Gas Mercury Measurement Methods

As noted earlier, many of the sampling and analytical methods used in the field sampling programs had not been formally validated for use with power plant streams. Recognizing the need for such validation, EPRI and EPA have recently sponsored an effort to formally evaluate EPA (Draft) Method 29 for mercury measurement in the stack gas of a coal-fired utility boiler. The tests were performed according to the "analyte spiking" procedure of EPA Method 301 protocol for the field validation of stationary source emission measurements. Several other mercury measurement methods were also employed during the tests to provide a comparison to the Method 29 measurements. Two solid sorbent methods were also used. The first, the MESA (Mercury Sorbent Adsorption) method, utilizes iodated carbon traps in series with soda lime traps [6]. The second method uses activated charcoal for sampling followed by neutron activation analysis [12]. The other methods were the Hazardous Element Sampling Train (HEST) [13] and EPA Method 101A. The mercury measurements by EPA Methods 29 and 101A and by the HEST train included particulate mercury, while the other methods measured only vapor phase mercury. Flue gas samples for all the methods were collected, in each of the eight runs, using quadruplet sampling trains (quad trains) located in adjacent ports in the vertical run of a duct leading from the outlet of an electrostatic precipitator (ESP) to the stack. Details of the test program and results can be found elsewhere [14]. Based on the test data, the EPA (Draft) Method 29 appears to meet the precision and bias criteria of EPA Method 301 protocol (i.e., precision not to exceed 50% relative standard deviation, and bias correction factor, if any, to be in the range of 0.7 to 1.3). Figure 2-1 shows a comparison of the flue gas total mercury concentrations measured by the different methods. For Method 29, the figure shows average concentration of the unspiked train. The results indicate reasonably good agreement among mercury measurements from the different methods.

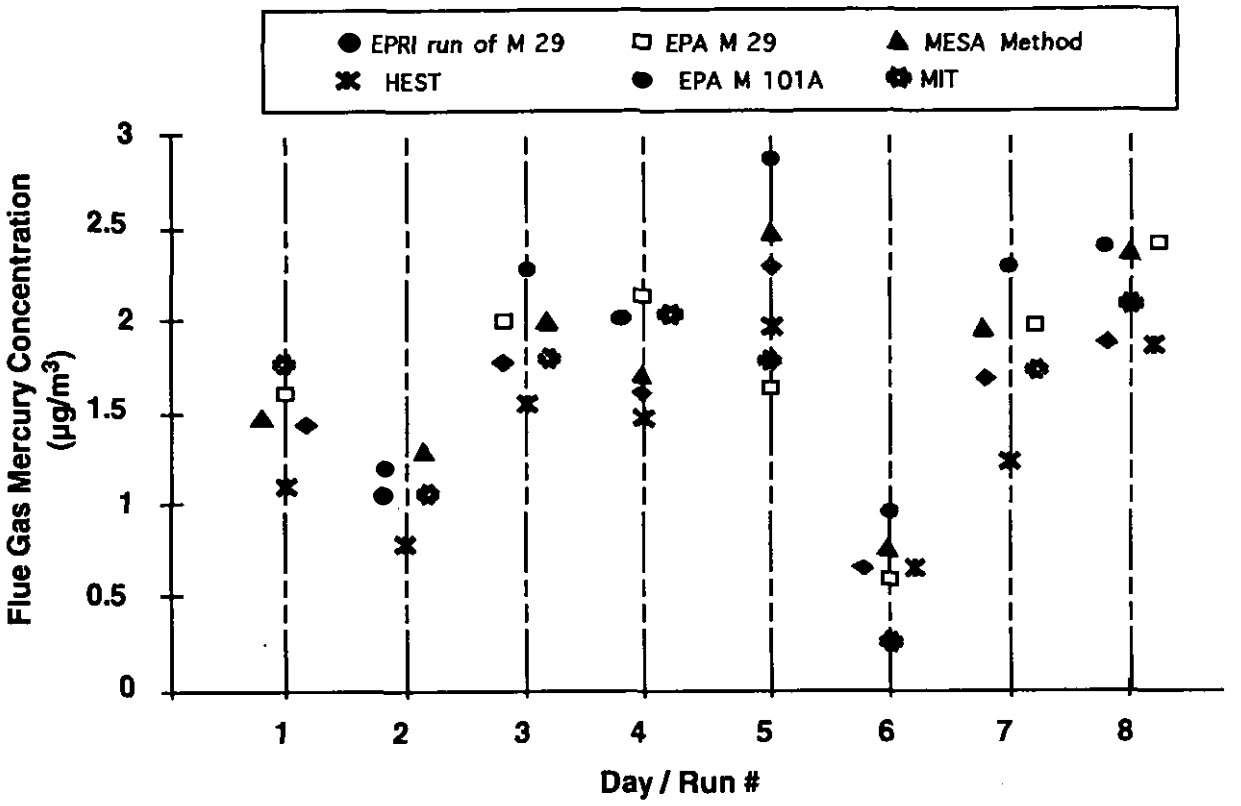

Figure 2-1.
Measurement of Total Mercury by Different Methods in Stack Gas of a Bituminous Coal-fired Plant

2.2.2 Uncertainty

Every measurement taken during the field program has an uncertainty associated with it. This uncertainty includes variability and errors in both the sampling and analysis components of the measurement. Additionally, the reported value is intended to be representative of a process or emission stream that has its own inherent variability.

Uncertainty is typically described in terms of precision (variability) and bias. Precision describes the repeatability of the measurement, while bias reflects a consistent deviation from the "true" value for the measurement (as determined by comparison with a standard). Table 2-4 presents the multiple performance measures, for both precision and bias, for each of the analytical parameters addressed during the field programs. The ranges of precision and bias shown in Table 2-4 reflect what might be expected for these methods under very carefully controlled sampling and analytical methods such as those followed during the FCEM program. It is easier to routinely obtain a measure of precision and bias

under controlled laboratory conditions than under exacting field conditions. A discussion of the approach to assessing and/or controlling precision and bias of the sampling and analytical measurements is provided below in Section 2.3.

Table 2-4.
FCEM Bias and Precision Objectives

Analytical Parameters	Bias[1]		Precision[2]	
	Tools	Example Range	Tools	Example Range
Metals	Laboratory Check Standards (LCS)	80-120% Recovery	LCS Duplicates	25% RPD[3]
	Matrix Spikes (MS)	70-130% Recovery[4]	MS Duplicates	20% RPD[5]
	Analysis of Standard Reference Materials	80-120% Recovery	LCS/LCS Duplicates	15% RPD
Anions	Laboratory Check Standards	80-120% Recovery	LCS Duplicates	25% RPD
	Matrix Spikes	75-125% Recovery[8]	MS Duplicates	15% RPD
	Independent Standard	90-110% Recovery	Replicate Analysis	15% RPD
Volatile Organics	Matrix Spikes	50-150% Recovery	MS Duplicates	15% RPD
	Surrogate Spikes	50-150% Recovery[6]	MS/MS Duplicates	50% RPD[7]
Semivolatile Organics	Matrix Spikes	50-150% Recovery	MS Duplicates	40% RPD
	Surrogate Spikes	70-130% Recovery	MS/MS Duplicates	30% RPD
Radionuclides	Laboratory Check Standards	70-130% Recovery	Replicate Analyses	20% RPD
Dioxins/ Furans	Surrogate Spikes	40-130%	Replicate Analyses	50% RPD
PAHs (solids)	Surrogate Spikes	90-130% Recovery	Replicate Analyses	50% RPD
PAHs (gas)		40-120% Recovery		-
Metals (coal)	Standard Reference materials	80-120% Recovery	Replicate Analyses	70% RPD
Aldehydes	Laboratory Check Samples	80-120% Recovery	Replicate Analyses	20% RPD
	Matrix Spikes	50-150%	MS Duplicates	50% RPD

Table 2-4.
FCEM Bias and Precision Objectives (Continued)

Analytical Parameters	Bias[1]		Precision[2]	
	Tools	Example Range	Tools	Example Range
HHV	Standard Reference Materials	90-110% Recovery	Replicate Analyses	20% RPD
Ultimate/ Proximate Analysis	Standard Reference Materials	90-110% Recovery	Replicate Analyses	10% RPD

1 Blanks were also used to evaluate potential bias. Field, method and reagent blanks were analyzed for each test program. Blank corrections were not usually made unless the value of the blank was equal to or greater than 50% of the native concentration. Some values were flagged if excessive blank contamination was found; however, the flagging practice varied from site to site.

2 Precision of measurements made very near the detection limit exhibited a larger relative percent difference than when analyte concentrations were well above the MDL.

3 RPD = Relative percent difference.

4 Range for FCEM is 80-120%.

5 Value for FCEM is 15%.

6 Range for FCEM is 70-130%.

7 Value for FCEM is 30%.

8 Range for FCEM is 75-125%.

Estimates of precision and bias of specific process streams were used to calculate confidence intervals from the raw results according to standard statistical techniques. Error propagation analysis, based on ANSI/ASME [15] protocols, was also used to determine the separate contributions of process, sampling, and analytical variability and measurement bias to the overall uncertainty of the results. These calculations produced results which may be used in risk assessment models and applications that require statistical validation of input data.

2.2.3 Detection Limits

Many trace substances are present in very low concentrations in fossil power plant streams. The sampling and analytical methods used to measure these substances must have a detection limit at least an order of magnitude lower than the stream concentrations to ensure that the data obtained are reliable. In the FCEM program, this was accomplished by a combination of extended sampling times for collection of larger sample volumes (flue gas) and employment of alternate, more sensitive analytical techniques.

For the FCEM program, the detection limit (DL) is calculated by multiplying the MDL (Method Detection Limit) [16] by any dilution or concentration factors for the sample being analyzed. The MDL is laboratory-specific, instrument-specific, and matrix-specific,

and includes variability in both sample preparation and analysis. The MDL is similar to an instrument detection limit (IDL) but differs in that the MDL includes variability associated with the preparation of the sample. Thus, for a given method, the DL can vary with each individual analysis. Likewise, DLs vary among laboratories, instruments, and analysts, all using the same method. Appendix A discusses the derivation of risk-based detection limits.

2.3 Quality Assurance/Quality Control

Several data quality objectives (DQOs) were established for the FCEM project to serve as benchmarks for referencing confidence in the associated results. The precision and bias objectives stated in Table 2-4 are examples of the DQOs for the FCEM project. Additionally, DQOs were developed for levels of background contamination, desired analytical detection limits, and mass balance closures. Data that did not meet the DQOs were not *a priori* considered unsatisfactory. Rather, the DQOs served as benchmarks for comparison to the precision and bias actually achieved by a particular measurement.

A variety of quality control (QC) activities were used to assess and control the sampling and analytical measurement process. These QC activities are summarized in Table 2-5. They include multiple measures of precision and bias. They also address uncertainty associated with both sampling and analytical procedures as well as the actual process and emission streams. Each of these performance measures can be compared to the stated DQO and an evaluation made of the fitness for use of the associated data. Data points that did not fall within the desired range of the stated DQOs (and for which there were no clear explanations) were not necessarily considered unacceptable. However, the data were noted as having potential limitations, and were generally not used in further calculations.

Table 2-5.
Types of Quality Control Samples

QC Activity	Characteristic Measured
Precision	
Replicate samples collected over time under the same conditions	Total variability, including process or temporal, sampling, and analytical but not bias.
Duplicate field samples collected simultaneously	Sampling plus analytical variability at the actual sample concentrations.
Duplicate analyses of a single sample	Analytical variability at the actual sample concentrations.
Matrix- or media-spiked duplicates	Sampling plus analytical variability at an established concentration.
Laboratory control sample duplicates	Analytical variability in the absence of sample matrix effects.

Table 2-5.
Types of Quality Control Samples (Continued)

QC Activity	Characteristic Measured
Accuracy (including bias and precision)	
Matrix-spiked samples	Analyte recovery in the sample matrix, indicating possible matrix interferences and other effects. In a single sample, includes both random error (imprecision) and systematic error (bias).
Surrogate-spiked samples	Recovery of compounds in the sample matrix that are chemically similar to compounds of interest. Used as an indicator of analytical efficacy.
Media-spiked samples	In this case, blank media is spiked. Used where a matrix-spiked sample is not feasible, such as certain stack sampling methods.
Laboratory control samples (LCS)	Analyte recovery in the absence of actual sample matrix effects. Used as an indicator of analytical control.
Standard Reference Material	Analyte recovery in a matrix similar to the actual samples.
Blank Effects	
Field blank	Total sampling plus analytical blank effect, including sampling equipment and reagents, sample transport and storage, and analytical reagents and equipment.
Trip blank	Blank effects arising from sample transport and storage.
Method blank	Blank effects inherent in analytical method, including reagents and equipment.
Reagent blank	Blank effects from reagents used.

In addition to the above activities, other data quality assessment and/or control measures included:

- Documenting the representativeness of the plant operations as compared to "normal" (based on information provided by plant personnel);
- Documenting the representativeness of the sampling location relative to the system being sampled;
- Documenting the calibration of field sampling equipment and adherence to standard operating procedures during the sampling process; and
- Documenting data consistency through material balances (mass flow rates).

As with the above activities, these qualitative and quantitative measures could also be compared to the DQOs for the project, and a comprehensive assessment of data quality made regarding the sampling and analytical data obtained.

2.4 Lessons Learned

This section discusses several important lessons that have been learned to date in trace substance sampling and analysis during the FCEM project. Some of the lessons are general while others involved specific sampling and analytical procedures. Among the more important lessons are:

1. *Site differences require flexibility in the execution of each test program.* Detailed test plans were generated prior to each field test that incorporated the appropriate sampling and analytical procedures and any modifications needed to characterize the specific site.
2. *All measurement program teams have to go through a "learning curve" before they are able to successfully do sampling and analyses needed to produce meaningful results.* This is because the sampling and analytical methods used for FCEM data collection are being pushed to their limits of capability. The situation is made more difficult because several different sampling/analysis organizations had to be used to perform the field work at the large number of sites in the FCEM program in the required timeframe. Even within one sampling/analysis organization, differences among field personnel between sites can result in significant differences in the quality of results.
3. *The sampling and analysis process is an iterative one.* During the course of the FCEM program, several method modifications were made to overcome practical difficulties and method limitations. Re-tests at some of the earlier field sites were carried out using modified methods to obtain more meaningful results.
4. *Severe matrix effects complicate analytical procedures.* Coal composition (major components such as sulfur, chloride, ash, and trace element concentrations) varies from one region of the country to another. Therefore, a single standard measurement method was not always applicable from one plant to another. Test samples and additional QC procedures were often needed to determine the appropriate analytical technique for each particular type of coal and analyte, resulting in extra time and cost for analysis. Samples were often analyzed for the same parameters by several different techniques.
5. *Background interference problems may be significant.* Risk assessment data needs prompted measurements at very low concentrations at which background interferences became a very troublesome issue. For instance, improved analytical instrumentation in the form of GC/HRMS provided better sensitivity by two or three orders of magnitude for semivolatile organic compounds. However, while the improved analytical tools greatly enhanced sensitivity they also added to the costs, and required still lower background contamination concentrations. Background contamination problems were minimized by the use of ultra-pure or select-grade reagents, though at increased cost.
6. *Modification of measurement techniques requires careful implementation.* The impacts and ramifications of a specific change were not easily anticipated. For example, sensitivity of the multi-metals method for some gas stream trace substances was improved by collecting a larger sample over a longer time period.

But, collecting a larger sample entailed the following additional complications:

- More particulate material was collected, plugging the filter and causing excessive pressure drop in the sampling train.
- More moisture was collected, so the impinger train needed to be changed at mid-run or additional impingers had to be added to collect overflow.
- Analytical interferents which were below detection limits on lower-volume samples became a problem as larger samples were collected.
- More time was required to collect the sample, which sometimes required a second shift or longer testing days.

7. *Vapor-phase mercury measurements in flue gas by the EPA Method 29 were biased low (in early field tests) due to the failure to consume the excess peroxide that interferes with the mercury analysis.* This occurred, during initial field tests, when insufficient potassium permanganate ($KMnO_4$) solution was added to the nitric acid/hydrogen peroxide impingers. The method calls for the addition of a solution of $KMnO_4$. During subsequent tests (as well as during the re-tests of earlier sites for mercury), approximately 2.5 grams of solid $KMnO_4$ were added to the nitric acid/peroxide impingers to consume excess hydrogen peroxide. The addition of solid rather than aqueous $KMnO_4$ minimizes sample dilution and assures the consumption of excess peroxide. Proper sample preparation and analysis techniques have since been verified for vapor-phase mercury.
8. *Teflon® was found to be inappropriate for volatile organic sampling train (VOST) probe liners.* The VOST methodology requires the use of a non-contaminating probe liner material. Teflon® liners were initially chosen based on the non-contaminating characteristics of the material and interpretation of the method protocol. However, Teflon® was found to be permeable to volatile organics present in the surrounding heat tape. Glass liners, though more fragile and expensive, were used at subsequent sites to prevent background contamination.

2.5 Summary and Future Work

The sampling and analytical methods initially chosen for the FCEM program were EPA-recommended methods and were perceived to be appropriate techniques based on precision, bias, and agency and industry acceptance. These methods were at their limits of capability (e.g., detection sensitivity) in terms of their application to trace substance measurements in fossil-fuel power plant streams. In several instances, modifications had to be made to overcome difficulties encountered in sampling and analyzing the very low trace substance concentrations present. The FCEM sampling and analytical effort has been an iterative process. Using modified methods, some of the earlier field sites were re-tested to obtain more meaningful results. In the FCEM program, the best methods available to date have been used to obtain the most credible field data.

Still, further sampling and analytical methods development is needed to refine methods and formally validate them. Work is underway or planned in the following areas:

- Formal and rigorous evaluation of the capabilities of the EPA (Draft) Method 29 and other methods for flue gas mercury speciation in terms of oxidized and elemental forms of mercury.
- Evaluation of conversion among species or physical form in sample extraction and capture processes of Method 29 as well as other methods, with respect to mercury.
- Investigation of flue gas temperature effects on the particulate mercury (mercury caught on the filter within the Method 29 train).
- Investigation of "artifacts" in the preparation/digestion procedure for soda lime sorbents in the MESA mercury speciation method that resulted in overestimation of flue gas methylmercury concentrations at some sites.
- Investigation to improve measurement of flue gas Se concentrations (there was a wide spread in measurements by the various DOE field contractors).
- Development and formal evaluation of methods for speciation of other important trace metals such as As, Cr, and Ni.

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3

SITE TEST RESULTS: FIELD DATA PRESENTATION AND CORRELATIONS

Over the past several years, EPRI and the U.S. Department of Energy (DOE) have sponsored programs to conduct a thorough characterization of trace substance emissions from power plants. These measurement programs provide a comprehensive set of data that can be used to estimate emissions from similar, untested facilities. In this section, data are presented and analyzed, resulting in correlations and emission factors that are then used in the work described in Section 4 to estimate emissions for all plants. Specific highlights of this section are as follows:

- EPRI and DOE have measured emissions of hazardous air pollutants from 48 test sites at fossil fuel-fired steam generating operating power plants. The tests encompass each major fuel type and boiler configuration as well as SO_2, NO_x, and particulate control technologies. The resulting database represents the best data set currently available to estimate emissions from steam-electric fossil fuel-fired power plants.
- The results have been quite variable from unit to unit, with emissions of a specific hazardous air pollutant (HAP) ranging over several orders of magnitude. For some HAPs, the data are not normally distributed; therefore, an arithmetic average is not an appropriate estimator. When appropriate, the results were subdivided into smaller subsets to account for variables such as fuel type and SO_2 and particulate control technologies.
- The HAPs were divided into three major groupings to develop emission factors for estimating emissions. The correlations or average emission factors suggested in this Section are appropriate for estimating emissions from the utility industry. They are not precise enough for use in developing permit conditions for individual units. For coal-fired units, the recommended approaches are:
  - *Particulate-Phase Metals.* These trace metals are components of the fly ash and are effectively captured by a particulate control device, generally with greater than 90% reduction from their coal concentration levels. The recommended estimation approach for each particulate-phase trace metal is a correlation that incorporates the inlet concentration in the coal and the total particulate emission rate. These element-specific regressions integrate the data from the various control technologies and coal types.
  - *Volatile Inorganics.* These inorganic substances in the fuel (such as chloride which forms hydrochloric acid, mercury, and selenium) are more volatile and, thus, are not consistently captured in a particulate control device. Removal efficiencies have

varied significantly from site to site. A correlation to adequately predict removal efficiencies could not be developed, thus the recommended emissions estimates for these compounds are average removal efficiencies dependent on the type of control device.

- *Organic Compounds.* These compounds are formed at trace levels during the combustion process. Concentrations of organics measured are quite variable. The recommended emissions estimates are geometric means for each substance. VOCs (benzene, toluene) and aldehydes are found at the level of pounds per 10^{12} Btu (levels similar to trace metal emissions from coal plants with particulate controls only). Emissions of PAHs and dioxins/furans are about 3 and 6 orders of magnitude lower than that of VOCs, respectively.

- For oil- and gas-fired power plants, the recommended emissions estimates for each group of HAPs are geometric or arithmetic mean emission factors.

3.1 Test Programs Overview

EPRI began the Field Chemical Emissions Measurement (FCEM) project in early 1990 to gather information of consistent quality on power plant emissions. Emphasis was placed on selecting test sites which were representative of the utility industry as a whole and on using the most current EPA draft or approved sampling and analytical protocols (see Section 2).

The results and experience gained at the initial test sites were used to modify some of the sampling and analytical methods for later sites. These changes, which have led to improved procedures and to a greater understanding of the distribution of trace substances in power plant streams, were discussed in Section 2. This experience also allowed the program to focus the sampling efforts on the trace substances of highest interest.

In parallel with EPRI's interest in trace substance emissions, the Department of Energy has two initiatives which have collected similar data. The Clean Coal Technology program, in which advanced technologies are demonstrated, has incorporated the measurement of emissions as project objectives at several sites, sometimes in collaboration with EPRI. This program has often provided data from both a "baseline" configuration, and then after a control technology has been implemented. The second DOE initiative was the Comprehensive Assessment of Emissions project, carried out for eight coal-fired field sites. This program conducted sampling in the summer of 1993. The combined data from the EPRI and DOE test sites has been used to develop emissions estimates for coal-fired units in Section 3.5.

3.2 Scope

The primary purpose of this section is to present appropriate methods of estimating trace substance emissions from fossil-fuel power plants. The large variability of conventional control device performance indicates that trace substance emissions will also be variable. Estimation procedures that incorporate the variability of the control device are used when statistically significant correlations can be developed. In other instances, emission factors developed from recent measurements are used.

Sampling studies and data evaluation have been completed at a total of 48 (27 coal-, 12 oil-, and 9 gas-fired boilers) sites. Up to 23 substances were measured at each field test site. However, this section presents the emission estimates only for the following trace substances of greatest interest for risk assessment:

- Arsenic, beryllium, cadmium, chromium, cobalt, manganese, mercury, lead, nickel, and selenium;
- Hydrogen chloride (HCl);
- Radionuclides;
- Benzene, toluene, and formaldehyde;
- Polycyclic aromatic hydrocarbons (PAHs); and
- Dioxins/furans.

Radionuclides and dioxin/furans were sampled at only selected test sites (primarily at the DOE test sites), thus the data available are limited.

3.3 Test Sites

In late 1989, EPRI profiled the utility industry by categorizing steam-electric power plants in terms of fuel type and emission controls. Some sixteen major groupings were developed. For example, bituminous coal-fired units with ESPs make up about 40% of the total fossil-fuel generating capacity. For many of these categories, candidate test sites with units to be measured were identified. A goal in the site selection process was to obtain a data set for the major configurations of fuel type and emission control. In Appendix B, the fuel, site, and measurement characteristics about the sites that have been tested are presented. The type of information obtained at each site has varied, depending on the specific goals of each test program and the level of funding available.

Figure 3-1 compares the fuel types of all the commercial units (the 1,750 + individual boilers larger than 25 MW reported in the Utility Data Institute's Power Statistics Database in operation, construction, or planned) and the number of data sets available. In Figure 3-1, the left and right vertical scales maintain a 40:1 ratio. When the industry and data set bar height are the same, 2.5% of that industry classification has been tested. Proportionally

more subbituminous- and oil-fired sites have been tested. However, potential fuel switching strategies from medium- and high-sulfur bituminous coals to low-sulfur subbituminous coals may change the future industry profile. The large number of oil sites listed are due to the seven sites tested as part of the State of California Air Toxics "Hot Spots" Information and Assessment Act of 1987 (AB2588). A more detailed comparison (i.e. plant age, size, control devices, etc.) of the test sites and commercial units is provided in Appendix B. In general, all major classifications are equally represented. This indicates that the results obtained should represent the current emission levels of the electric utility industry, and provide a basis for projecting these measurements to use with scenarios of the future industry (for current measured technologies).

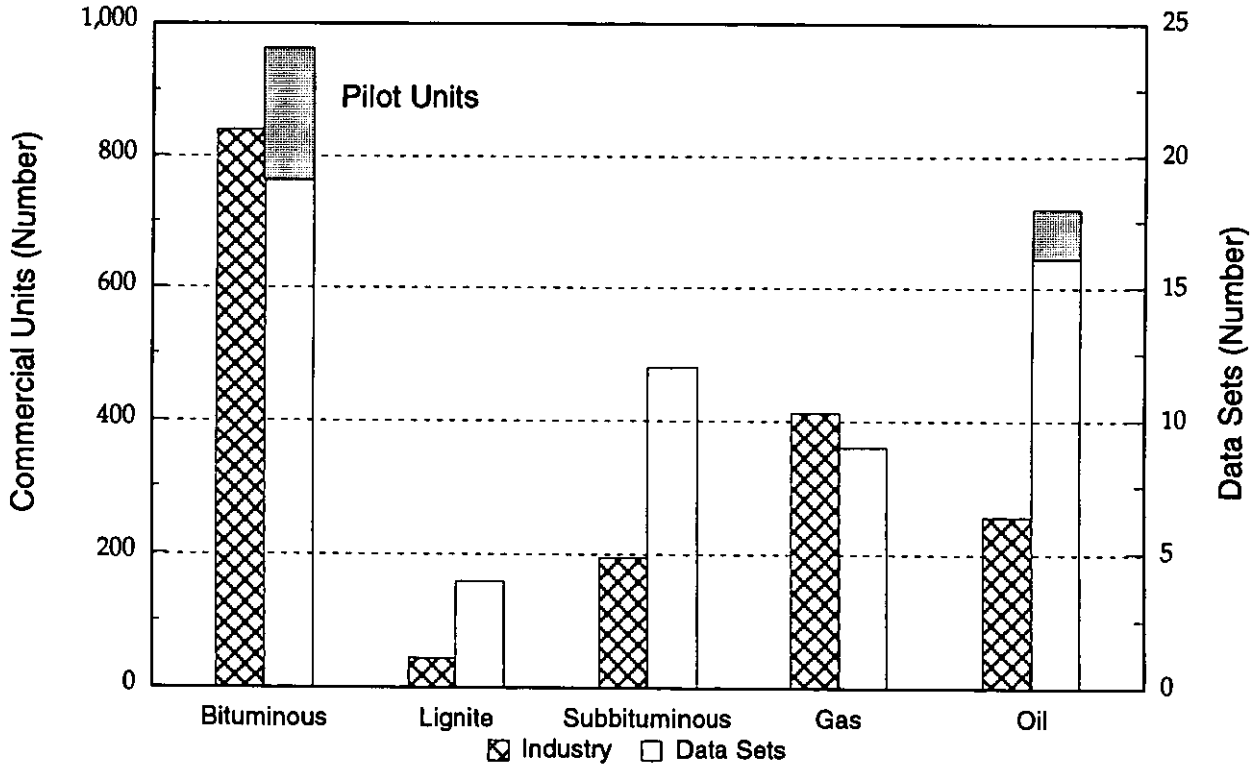


Figure 3-1.
Fuel Distribution

3.4 Trace Substance Emissions Estimation Approach

Results from the FCEM and DOE sampling programs were voluminous. To effectively utilize the information from these programs, it was necessary to define the purpose to which they would be applied. Within the scope of this section, site data have been used to develop emission estimates for risk assessments for power plants both with and with-

out unit test results. The recommended emissions estimation technique is presented for each class of compounds and used in subsequent sections to estimate source terms for the risk assessment.

There are many ways to use the current data for emissions estimation. The approach presented here incorporates experience gained during the sampling and analysis effort (i.e., which streams and substances can be measured accurately) and an assessment of what information is readily available for units that have not been tested (i.e., what terms should be independent parameters in the correlations). Inherent in all these activities is the actual measurement variability associated with trace quantities of substances (i.e., the degree of sophistication should be consistent with the measurement uncertainty). For metals emissions from coal-fired units, a correlation with coal composition and particulate emission rates incorporates the variability seen across many of the sites as independent parameters. For the volatile elements in coal, average percentage removals for various equipment and coal types are the recommended approaches to estimate emissions. For all other substances and fuels, emission factors have been derived based upon the data set for the respective measured sites representing those operating conditions.

The field data are discussed below by fuel type: coal, oil, and gas. From each site test reports, a database was created that contains the mean concentration of the trace substances of interest. The mean value is typically the result of three successive measurements, often on consecutive days. Solid and liquid fuel streams are typically composited over each flue gas sampling period. Therefore, the resulting mean value is a "snapshot" of the site operation. Statistically estimating the long- term average emissions, based on three samples, typically produces a large confidence interval about the mean. This uncertainty must be considered when estimation of emissions is performed, so that an inappropriate degree of accuracy is not inferred from a calculated value.

3.5 Results From Coal-fired Field Sites

Of the target substances, few logical groupings exist for presenting emission estimates. Many of the elements partition to the solid phase (at particulate control temperatures) and are effectively controlled by a conventional particulate control device. Other elements (mercury, chlorine, and selenium) are relatively volatile at stack gas conditions. Volatile organic substances are created during the combustion process and typically not effectively reduced by air pollution control devices at power plants. Radionuclides are present in the solid phase and can be directly related to the amount of particulate matter present in the stack gas. These four groupings are used in the following discussion.

This section will present recommended correlations and emissions factors that will be used in Section 4 to estimate emissions for all units. A more detailed analyses of the results is provided in Appendix B.

3.5.1 Particulate Phase Metals

The particulate phase metals are, by definition, less volatile and tend to be associated with the fly ash emissions. These elements are: antimony, arsenic, beryllium, cadmium, chromium, cobalt, lead, manganese, and nickel. These metals are well controlled by existing particulate control technologies. Particulate phase metals tend to behave like what we have always referred to as "particulate emissions."

There are many ways to use the current EPRI and DOE data to estimate particulate-phase metals for all coal-fired units. A mechanistic model that incorporates furnace type, particle size distributions, ash resistivity, and control equipment design parameters (specific collection areas, air to cloth ratios, etc.) would be a more ideal approach. However, the quantity of detailed information that would be necessary to develop and to use a mechanistic model is not available for most of the test units, and certainly is not available for all the coal-fired units for which emission estimates are needed. After evaluating several alternative approaches, a relatively simple empirical approach was adopted. The empirical approach is based on statistical correlations among the measured data. Specifically, measured particulate-phase metals are correlated with measured particulate emissions and trace metal concentrations in the coal ash.

The correlation is in the form of the following power-law relationship:

$$E_i = a_i \left[(coal_i / ash\ fraction) * PM \right]^{b_i}$$

where:

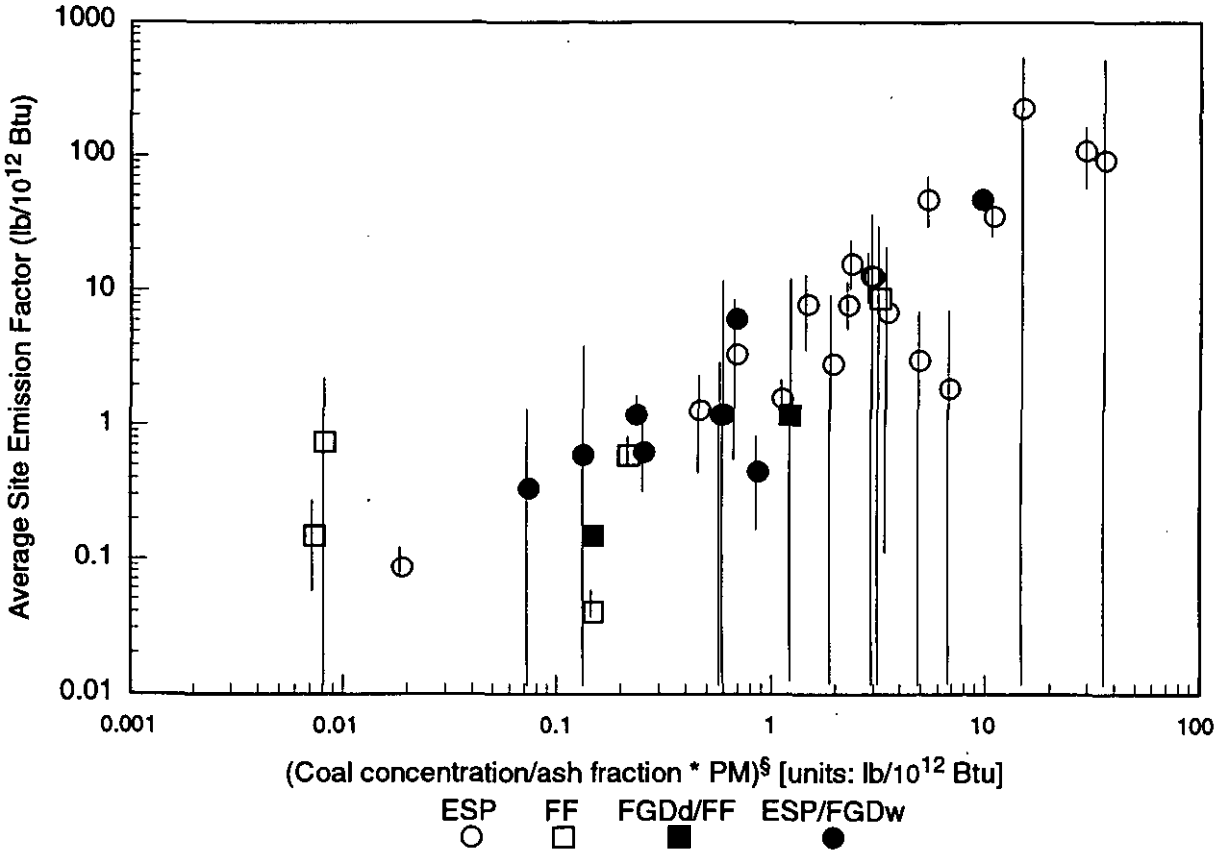
E_i	=	Emission of trace substance "i" ($lb/10^{12}$ Btu)
$coal_i$	=	Concentration of trace substance "i" in coal (ppm)
ash fraction	=	Fraction of ash in coal (dimensionless-fraction)
PM	=	Emission factor for total particulate matter ($lb/10^{6}$ Btu)
a_i , b_i	=	Correlation coefficients for trace substance "i."

These empirical correlations incorporate the degree of particulate control efficiency with the particulate emission rate; it also indirectly includes potential enrichment and particle size distribution

While these nine metals all partition to the solid phase, the data set is robust enough to statistically differentiate the power-law fit for each of the metals. Arsenic data are presented below as an example of how the existing site data can be used to estimate emissions from untested coal-fired plants.

3.5.1.1 Arsenic

Figure 3-2 plots the measured emissions as a function of coal trace substance concentration, coal ash, and particulate emission. The vertical lines through each average site emission spans the 95% confidence limit about the mean value. At many sites, although the mean value is large, the confidence interval includes zero, indicating large uncertainty in the calculated site mean.



§ **Coal concentration:** concentration of substance in coal [parts per million by weight]; **ash fraction:** fraction of coal that is ash [dimensionless]; **PM:** particulate matter emissions [lbs per 10^6 Btu]

Figure 3-2.
Arsenic Correlation Data

Also note that the data points do not show any marked difference for the various types of control technologies (i.e., although most ESP/FGD and fabric-filter sites have lower arsenic emission levels, the data points line up with the ESP emission data). This simply indicates that the nominal composition of particulate matter exiting a control device is primarily dependent on the fuel concentration, and that any differentiation of chemical composition by particle size is relatively small when data from many sites are aggregated. Conducting the regression analysis on the data shown in Figure 3-3 yields the following:

$$E_i = 3.1\ [(coal_i/\text{ash fraction}) * PM]^{0.85}$$

Figure 3-3 shows the regression of the data. The confidence intervals about each site average have been removed for clarity of presentation. The correlation coefficient (r^2) is 0.72. Because the regression includes over 30 data points, the correlation is significant at the 99.9% probability level (i.e., there is a one in one thousand probability that a set of numbers would show this relationship from chance alone). This equation predicts the long-term average emission level of arsenic from a typical coal-fired unit for a constant coal concentration and particulate emission level. Emissions measured at a specific plant at a given time may vary considerably from the predicted value. Two additional statistical parameters are also shown on the figure. The outer dashed lines are the 95th percentile confidence bands for the site mean values. This band is where the average of triplicate measurements for a site should lie 95% of the time.

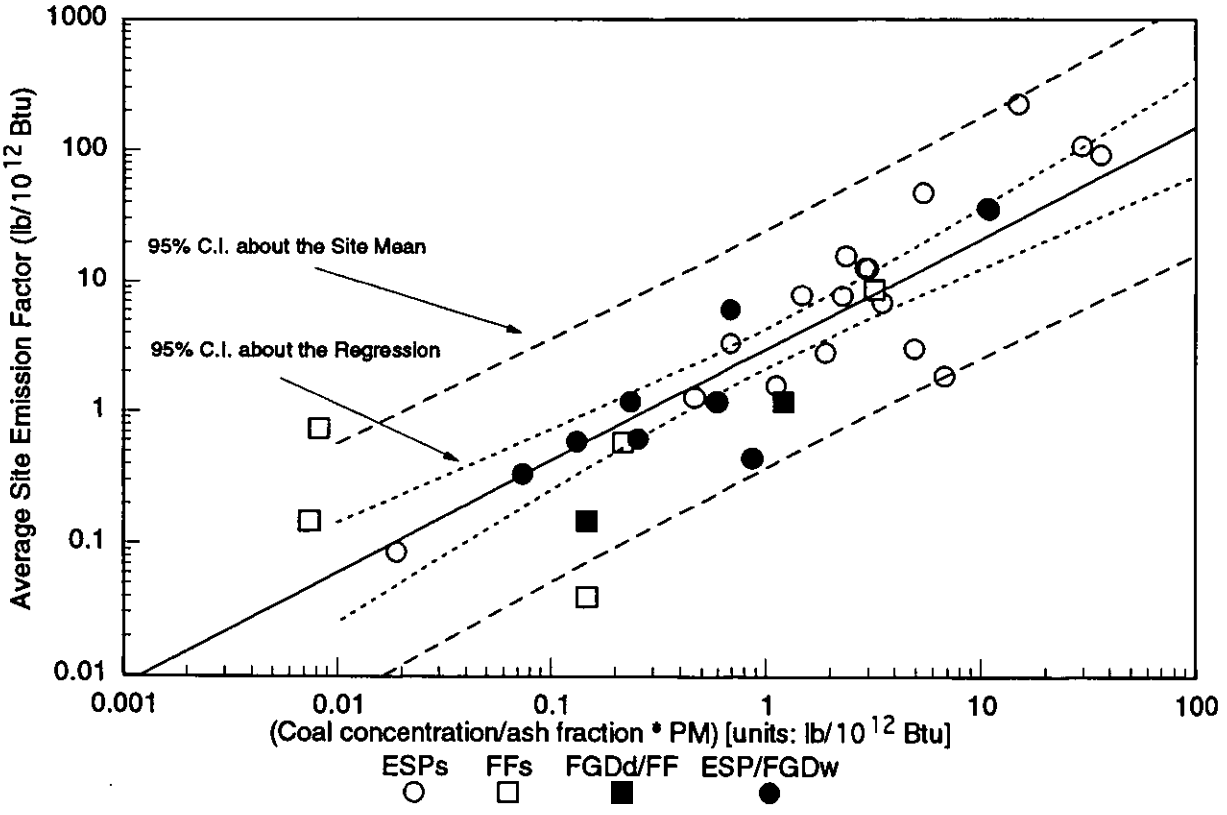


Figure 3-3.
Arsenic Emission Correlation

The inner dotted lines represent the upper and lower 95% confidence intervals about the regression. It is within this range that the true average value is expected to occur, 95% of the time.

3.5.1.2 Other Elements

Similar correlations and figures have been developed for the other eight metals. The correlations are presented in Table 3-1. The correlation coefficient (r^2) indicates the model relationship is statistically significant for these particulate-phase metals. As with arsenic, the variability of the predicted emission level can be expressed with confidence intervals. Appendix B presents the figures and the statistical information needed to calculate these values for all nine metals.

Table 3-1.
Summary of Particulate-Phase Emission Equations, lb/10^{12} Btu

Analyte	Average Predicted Emissions	Data Pairs	r^2
Antimony	$(0.92) \times^{0.63}$	8	0.65
Arsenic	$(3.1) \times^{0.85}$	34	0.72
Beryllium	$(1.2) \times^{1.1}$	17	0.83
Cadmium	$(3.3) \times^{0.5}$	9	0.78
Chromium	$(3.7) \times^{0.58}$	38	0.57
Cobalt	$(1.7) \times^{0.69}$	20	0.57
Lead	$(3.4) \times^{0.80}$	33	0.62
Manganese	$(3.8) \times^{0.60}$	37	0.57
Nickel	$(4.4) \times^{0.48}$	25	0.51

x: Coal ppm/ash fraction * PM.
r^2: Correlation coefficient for the regression.

Figure 3-4 shows the calculated average emission rate for these elements on a logarithmic scale for the ranges of independent levels seen at the test sites. The elements traditionally thought to be more volatile (lead and arsenic) show higher emissions for a given input. Chromium, magnesium, and nickel behave nearly identically. Antimony, beryllium and cobalt are the least volatile elements. All measured and predicted values of antimony, beryllium, and cadmium are below 10 lb/10^{12} Btu of fuel heat input.

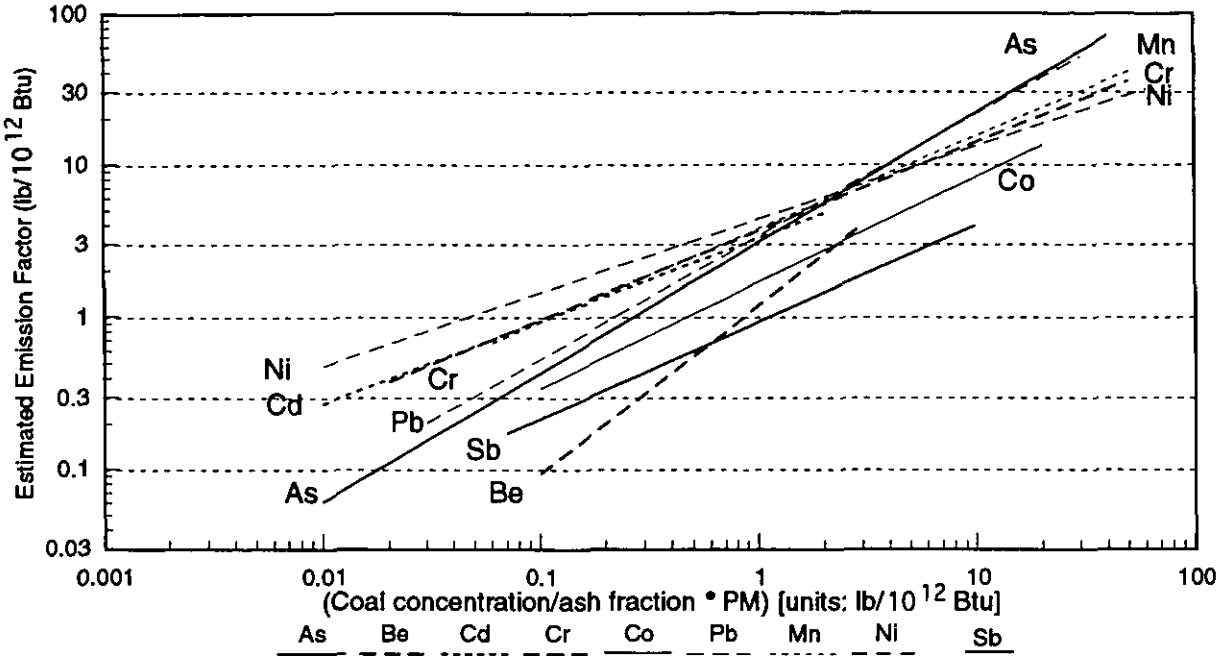


Figure 3-4.
Predicted Metal Emission Factors

3.5.1.3 Metal Speciation

The oxidation state of trace metal emissions can be of importance due to health risk concerns. For arsenic, chromium, mercury, and nickel, one oxidation state is of greater concern that the other. Speciation methods for these trace metals are developmental. Mercury speciation tests were conducted at a number of sites, and the results from these tests are discussed in Section 3.5.2.2. Sampling for arsenic, chromium, and nickel speciation were conducted at select sites. Thus limited data are available and additional effort is required to better quantify the oxidation state of these trace metal emissions.

Chromium. Chromium speciation sampling was conducted at four coal-fired power plants. At two sites, stack gas levels of hexavalent chromium [Cr(VI)] were less than field blank levels (i.e., the blank correction was greater than any measured result). At a third site, the high dust gas entering a fabric filter was sampled. An average of 4% of the total chromium was present as Cr(VI). The stack gas from an ESP was sampled at the fourth site. The average Cr(VI) level was 25% in this latter sampling. At both of these sites, the three replicate values had significant variability and corrections for background level were made; these were often 50% to 80% of the initial measured value.

At an oil-fired site with a poorly performing ESP, two sets of measurements were conducted (upstream and downstream of the ESP) using isotopic Cr(VI) spiked sampling trains. At the conclusion of sampling, about 50% to 60% of the isotopic Cr(VI) had con-

verted to the trivalent state. This conversion factor was applied to the measured levels to obtain average hexavalent concentrations. The mean Cr(VI) levels were 4% and 3% of the total chromium for the ESP inlet and outlet locations, respectively.

Additional data are required to better quantify Cr(VI) concentrations in flue gas stack emissions. Based on the developmental nature of the sampling and analytical method and the limited data available, a nominal value of 5% (based on the literature) of the total chromium emission level is assumed to be Cr(VI) for the risk assessment calculations in subsequent sections.

Nickel. Nickel speciation sampling was attempted at one oil-fired site using modified EPA Method 5 sample train and analyzed using sequential leaching procedures. Samples were taken at the ESP inlet and outlet. The particulate phase nickel consisted of 8% sulfidic nickel (such as Ni_3S_2, NiS, Ni_3S_4) at both the ESP inlet and outlet. The mean oxidic nickel was 54% and 27% of the total nickel at the ESP inlet and outlet, respectively. Soluble nickel (water soluble salts such as nickel sulfate and nickel chloride) represented 37% and 64% of the total nickel, respectively. Metallic nickel was measured at 1% at the ESP inlet and below detection limit at the ESP outlet.

Arsenic. No chemical speciation of arsenic combustion flue gas was performed in the EPRI or DOE field tests. Studies were performed on speciation of fly ash samples. The results of these analyses are described in Section 6.

3.5.2 Volatile Elements

Three inorganic substances found in coals are present primarily in the vapor phase of combustion flue gases and are not typically removed effectively by particulate control devices. Mercury, selenium, and hydrochloric acid measurement results and emission estimation techniques are discussed below.

3.5.2.1 Mercury

Figure 3-5 presents the sets of paired data for mercury (coal and flue gas measurements) for various control devices. As can be seen, for a number of ESP sites (and one wet FGD system), the normalized concentration in the gas stream is higher than the coal measurement. This obviously represents sampling and analytical variability. A correlation could not be developed to incorporate the variability in mercury removal efficiency. Table 3-2 presents the removal efficiency calculated for plants with various types of control devices based on the current data set. Also shown is the 95% confidence interval about the multiple-site average reduction (not about all of the data). The large confidence interval indicates that the average value lacks precision, as expected based on Figure 3-5. Furthermore, the small increase in control seen at sites with ESP/wet FGD systems indicates that reduction of mercury emissions at power plants is not currently well-understood. Based

on the current data set, it is recommended that mercury emissions from plants with ESPs and fabric filters be estimated as 70% of the coal level. For wet and dry FGD systems, 55% of the fuel level is recommend.

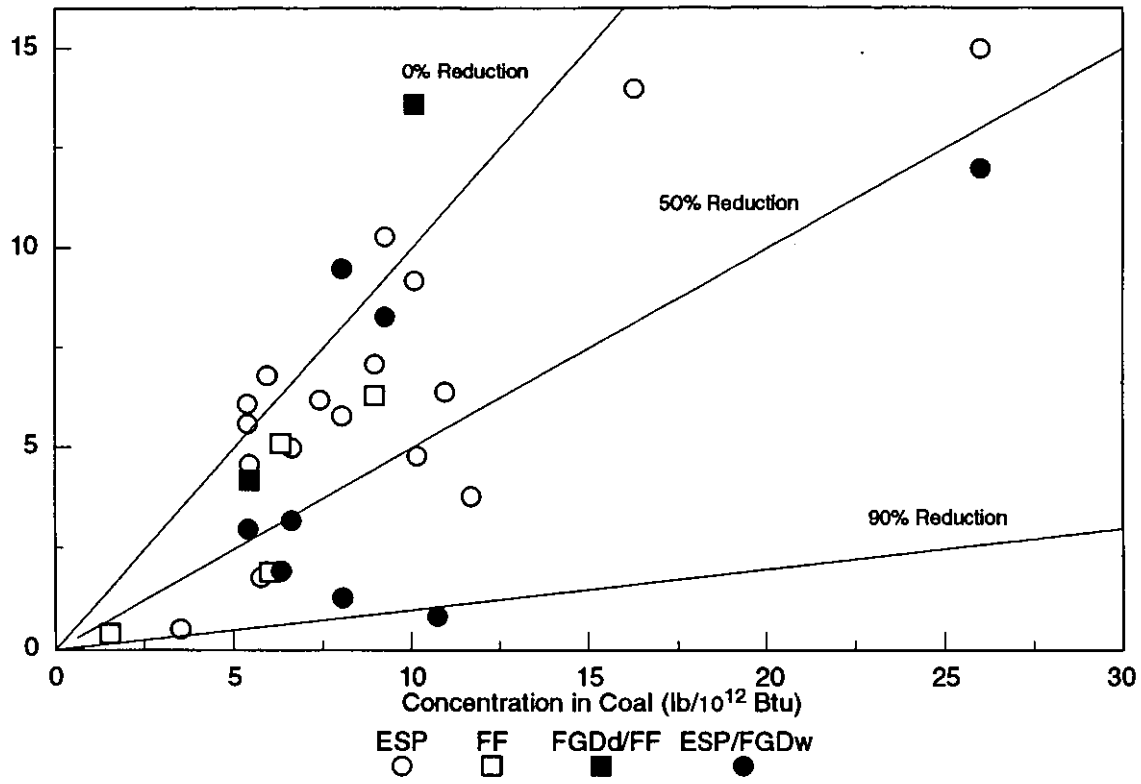


Figure 3-5.
Mercury Emissions from Coal Units

Table 3-2.
Mercury Reduction by Control Devices, All Coal Types

Control Device	Number of Sites	Average Reduction	95% C.I.[a]	Recommended Emission Factor Percent of Coal Input
ESP	17	26%	±14%	70%
Fabric Filter	5	39%	±38%	70%
ESP & Fabric Filter	22	29%	±13%	70%
FGD Systems	9	45%	±27%	55%

[a] 95% confidence interval about the multiple-site average reduction, not about all the site results.

3.5.2.2 Mercury Speciation

Mercury emissions may be present in at least two valence states—elemental [Hg(0)] and oxidized [Hg(II)]. This has significance for several reasons. The chemical form of the mercury may affect the degree of removal, as well as atmospheric fate, health effects, and calculated risk.

EPRI has applied two sampling methods to quantify mercury emissions—the EPA multi-metals train (EPA draft Method 29) and the sorbent speciation train now called the MESA method, see below [Bloom 1992] . EPRI has also used both methods to provide some estimate of oxidized and elemental mercury. It is important to note that neither method has been validated for mercury speciation, and the multi-metals method was not designed to speciate mercury. Mercury speciation results from the two methods were quite variable and a definitive relationship between oxidized mercury and chloride concentration in the coal could not be determined. These results are based on use of the EPA Method 29 train, however, both the MESA method and Method 29 require additional development and evaluation. The mercury speciation results are summarized in Table B-5 in Appendix B.

In extending these speciation results to power plant units for which direct measurements are unavailable, some cautions are noted. Because of the uncertainties in the understanding of mercury chemistry, an arithmetic mean value is recommended for estimating the oxidized fraction. For dry particulate devices, 70% of the emission level is estimated to be in the oxidized state. After scrubbing, 45% of the mercury is estimated to be in the oxidized state.

3.5.2.3 Selenium and Hydrochloric Acid

Some particulate controlled sites exhibit high levels of selenium and hydrochloric acid (HCl) reduction; these sites predominantly are burning coals with high levels of alkaline ash (10-20% CaO). Most of the wet FGD systems show high removal as well. Tables 3-3 and 3-4 present the recommended emission factors for coal-fired units, based on coal rank, or the presence of an FGD system. Although the average removal seen in fabric filters is higher than that found in ESPs, the difference is presumably due more to the sample population size than the inherent capability of fabric filters. Most of the tested units with fabric filters tested burn sub-bituminous alkaline ash coals.

Table 3-3.
Selenium Reduction by Coal Type and FGD System

Coal Type [a]	Number of Sites	Average Reduction	95% C.I. [b]	Recommended Emission Factor Percent of Coal Input
Bituminous, with ESP	15	45%	±13%	55%
Lignite, with ESP	1	use bituminous		55%
Subbituminous, ESP or FGD	5	97%	±4%	3%
Bituminous or Lignite, with FGD System	8	88%	±12%	12%

[a] For any coal type controlled by ESP or fabric filter.
[b] 95% confidence interval about the multiple-site average reduction, not about all the site results.

Table 3-4.
HCl Reduction by Coal Type and FGD System

Coal Type [a]	Number of Sites	Average Reduction	95% C.I. [b]	Recommended Emission Factor Percent of Coal Input
Bituminous	15	-1%	±13%	100%
Lignite	1	use bituminous		100%
Subbituminous	7	79%	±14%	20%
All, with FGD System	5	97%	±2%	3%

[a] For any coal type controlled by ESP or fabric filter.
[b] 95% confidence interval about the multiple-site average reduction, not about all the site results.

Selenium. For units burning bituminous and lignite coals, 55% of the coal level is recommended for the selenium emission factor. For sub-bituminous coal, 3% of the coal value is emitted. Units with FGD systems emit an average of 12% of the fuel level.

Hydrochloric Acid. Units burning bituminous and lignite coals show 100% emissions; subbituminous coals emit 20% of the fuel level. Units with FGD systems emit 3% of the fuel level.

3.5.3 Organic Substance Emissions

Five substances/classes of organic compounds are found in combustion flue gases that are on the CAAA list of hazardous air pollutants. The FCEM and DOE programs have collected data on volatile organics, aldehydes, semivolatile organics, and dioxin/furans. Each class of organics consists of a number of substances. For example, the GC/MS analytical procedure for volatile organics is able to discriminate among over 40 compounds. For the sake of brevity, selected results for each class of organics are presented. Specifically, benzene, toluene, formaldehyde, benzo(a)pyrene equivalents, and 2,3,7,8-p-tetrachloro-p-dioxin equivalents are of greatest interest from a health perspective.

Unlike the trace elements present in coal, the organic substances do not correlate well with particulate emissions or with type of control device. Organics may be formed due to incomplete combustion. However, data on CO levels, O_2 concentrations, unburned carbon, etc. are not available for many of the test sites. Furthermore, the measurement variability of trace organic substances often exceeds the mean value, making correlations impractical. High and low values were measured at field sites with both dry particulate controls and FGD systems. Therefore, all of the average site values have been pooled to estimate mean emission factors and confidence intervals. Log-normal distributions are appropriate for describing these sets of data. The inter-site data population is not normally distributed. The data appeared to fit a log-normal distribution.

Dioxin/furans and PAHs were sampled at only a few sites, thus the database for these compounds is still limited. The dioxin/furan data were highly variable from site to site, yielding significant uncertainty about the mean. This uncertainty may be due to the measured results being near the method detection limit. In addition, at some sites, the field blanks were equivalent to the measured stack emissions. Dioxin and PAH equivalency factors were calculated using the Method 23 protocol of multiplying the detected congeners (or PAH) concentration by the weighted equivalency factors per site. Table 3-5 presents the recommended emission factors for the five organic substances and classes for coal-fired power plants.

Table 3-5.
Organic Substance Emission Factors for Coal-fired Units, lb/10^{12} Btu

Organic Substance/Class	Measurements	Geometric Mean	95% C.I.[a]
Benzene	23	3.8	1.6 - 8.8
Toluene	21	1.4	0.7 - 3
Formaldehyde	22	3	1.5 - 6
Benzo(a)pyrene equivalent	11	0.0018	0.0004 - 0.0082
2,3,7,8-tetrachloro-p-dioxin equivalent	9	0.000002	0.0000004 - 0.00001

[a] 95% confidence interval is about the geometric mean, not about all the data

3.5.4 Radionuclides

Table 3-6 presents the geometric mean and confidence interval for eight radionuclides in coal and emitted particulate matter using appropriate data from the FCEM and DOE test sites and from the Utility Air Regulatory Group (UARG) report [1]. These data are internally consistent and are recommended as appropriate emissions estimates if the coal composition is not known for a particular plant. The radionuclide emission rate can be estimated by multiplying the particulate emission concentration (grams/gas volume) by the activity (pCi/gram).

Table 3-6.
Coal Radionuclide Emission Values, pCi/gram

	Coal			Emitted Particulate Matter		
Isotope	Number of Values	Geometric Mean	95% CI	Number of Values	Geometric Mean	95% C.I.[a]
Pb^{210}	20	0.6	0.3 - 1	7	10	4 - 23
Po^{210}	12	0.2	0.1 - 0.4	11	15	12 - 20
Ra^{226}	19	0.3	0.1 - 0.5	10	1.0	0.3 - 3.4
Ra^{228}	15	0.4	0.2 - 0.8	3	0.15	
Th^{228}	11	0.2	0.1 - 0.4	10	3.6	2 - 6
Th^{230}	12	0.4	0.2 - 0.7	10	6.2	3 - 13
Th^{232}	12	0.2	0.1 - 0.4	8	3.8	2.3 - 6.4
U^{234}	4	0.3	0.1 - 0.8	2	7	1 - 80
U^{235}	12	0.1	0.02 - 0.8	1	<0.3	
U^{238}	15	0.2	0.4 - 1.0	10	5.7	4.1 - 7.9

[a] 95% confidence interval is about the geometric mean, not about all the data

3.6 Results From Oil-fired Units

Only about 25% of the oil-fired units employs particulate control in the form of mechanical collectors or ESPs. Most of the field data measurements are on units without current controls.

3.6.1 Uncontrolled Oil Units

Most substances found in fuel oil are emitted in approximately the same quantity from the stack. Consequently, the estimation of emissions from untested units for these substances could be based on 100% of the fuel oil analysis. Considering that (1) the concentration of substances in fuel oil varies by country of origin and degree of refining; (2) a nationwide database of oil compositions similar to the USGS coal database does not exist; and, (3) oil consumers often purchase fuel oil on spot markets rather than through long-term contracts, an emissions estimation approach that does not rely on oil analysis is more desirable. The obvious choice is the use of average emission factors for trace substances. This approach is suitable for estimating emissions for the entire utility industry, but emissions at a specific unit may vary considerably from the sample-average value.

The data sets for specific substances were examined to determine if they were normally distributed. For all substances except HCl, the substance specific emission factors were not normally distributed. Therefore, geometric means and confidence intervals were calculated for both elemental and organic substance emissions from uncontrolled oil-fired units. As an example of the log-normal distribution, Figure 3-6 presents the average arsenic emissions per site, rank ordered, and the cumulative probability distribution for the statistical parameters from the data set. Table 3-7 presents the geometric average emission factor and confidence interval values about the geometric mean for the substances of interest. For the organic substances and the volatile elements (mercury, selenium, and HCl), the data from the four other oil sites with particulate controls were included in the distribution and statistics. The values reported for these substances at the outlet of a control device are similar to the uncontrolled emissions. Table 3-7 shows the total number of site average values, number of sites where the substance was detected, and number of data sets used in the statistics for a substance.

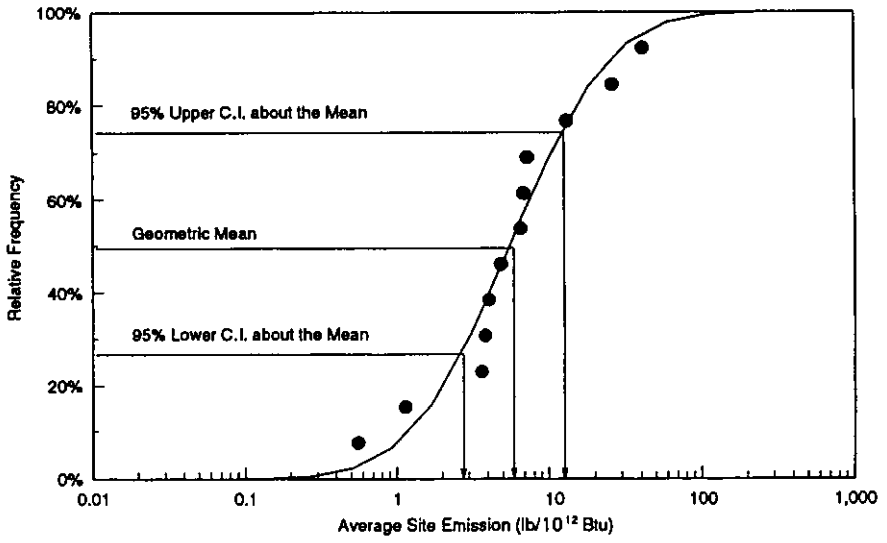


Figure 3-6.
Arsenic Emissions Distribution for Uncontrolled Oil-fired Boilers

Table 3-7.
Emission Factors for Oil-fired Units, lb/10^{12} Btu

Substances	Sites Tested	Sites Detected[a]	Sample Size[b]	Geometric Mean	95% C.I.[c]
Arsenic	12	11	12	5.5	2.6 - 12
Beryllium	12	5	10	0.20	0.07 - 0.56
Cadmium	12	11	12	1.3	0.5 - 3.6
Chromium	13	12	13	5.2	3.0 - 8.9
Cobalt	6	6	6	37	16 - 86
HCl	11	11	11	2400	1900 - 3100
Lead	12	10	12	7.0	3.4 - 14
Manganese	12	12	12	13	7.9 - 23
Mercury	16	9	11	0.46	0.20 - 1.04
Nickel	13	13	13	720	460 - 1140
Selenium	16	10	12	2.0	0.7 - 5.5
Benzene	17	11	13	1.1	0.7 - 1.5
Formaldehyde	17	12	17	20	7.5 - 56
Toluene	11	11	11	9.9	4.8 - 20
Radionuclides	1	1	1	1.9 pCi/gram	
Benzo(a)pyrene equivalents	15	4	4	0.0038	0.0005 - 0.026
2,3,7,8-TCDD equivalents	4	3	3	0.0000083	0.0000014 - 0.00012

[a] Number of times the substance was quantified.
[b] The number of measured values used to calculate the mean and confidence interval. (Individual values with high detection limits [>2x the highest quantified value] were not included in the mean.)
[c] 95% confidence interval is about the geometric mean, not about all the data.

3.6.2 Emissions From Oil Units with Particular Controls

Three oil sites tested had normally operating ESPs (the other two sites employed pilot-scale SCR and PJFF units respectively). With this limited data set, the pairs of detected fuel/emission data and ESP inlet/emission data were used to calculate removal efficiencies for the nine trace metals of interest. The average reduction was about 40% between inlet and outlet gas streams. Therefore, for these nine metals, 60% of the values in Table 3-7 should be used to estimate emissions from oil units with ESPs. For organic substances and volatile elements, the values in Table 3-7 are appropriate.

3.7 Results From Gas-fired Units

Limited test data are available from the FCEM program on emissions from gas-fired units. Some eight sites have been tested. At six sites, only benzene and formaldehyde measurements were made. A complete sampling and analytical effort was performed at two sites. With these limited data, only arithmetic average values are presented, except for formaldehyde, in Table 3-8. Most of the metal values reported had significant background levels in the filters, making these values questionable. Note that for some substances, no current measurements exist, however, significant levels of those substances are not expected in gas-fired flue gas, based on the typical composition of natural gas.

Table 3-8.
Emission Factors for Gas-fired Units, lb/10^{12} Btu

Substances	Sites Tested	Sites Detected [a]	Sample Size [b]	Arithmetic Mean
Arsenic	2	2	2	0.23
Beryllium	2	0	0	<0.01
Cadmium	2	1	1	0.04
Chromium	2	2	2	1.1
Cobalt	2	1	1	0.08
HCl	0			NV [c]
Lead	2	2	2	0.4
Manganese	2	2	2	0.4
Mercury [d]	2	1	2	0.0008
Nickel	2	2	2	2.4
Selenium	2	0	0	<0.02
Benzene	8	2	5	0.8
Formaldehyde [e]	9	8	9	34(7-150)
Toluene	2	2	2	10
Radionuclides	0			NV
Benzo(a)pyrene equivalents	2	0	0	ND [f]
2,3,7,8-TCDD equivalents	1	1	1	0.0000012

[a] Number of times the substance was quantified.
[b] The number of site values used to calculate the mean and confidence interval. (Individual values with high detection limits [>2x the highest quantified value] were not included in the mean.)
[c] NV = No value.
[d] Based on natural gas analysis, not detected in stack gas at higher concentration.
[e] Geometric mean and confidence interval.
[f] ND = Not detected.

Emission Factors for Oil-fired and gas-fired Boilers

3.8 Summary

Taken in the aggregate, the recent field sampling results present a relatively consistent set of information. For any given site, the individual measurement results are generally within 50% of the average value. This level of variability appears to be caused by a combination of day-to-day fluctuations and sample gathering, since the laboratory QA/QC characterizing the analytical procedure is generally very good. When comparing samples among different sites, the absolute magnitude of the measurements may vary by orders of magnitude. This indicates that for industry-wide models, log-normal statistical estimates are more appropriate estimators of emissions than normal statistical estimates. For metallic elements, a good correlation among fuel concentrations, particulate emissions, and metal emissions exists for coal-fired units.

For the purposes of estimating emissions from untested plants, the regressions and values developed here are appropriate and typically agree with actual measured values within an order of magnitude. Considering the uncertainty associated with actual measurements, this variability was deemed acceptable in light of the sensitivity analysis conducted as part of the risk assessment (see Section 7). Until the behavior of trace substances is better understood (and daily and sampling variability eliminated), any statistical analysis will predict values that differ from measured values, either under- or over-predicting the emission for a particular site. However, as an industry-wide estimate, the methodologies employed here are believed to be the best available.

The data are still somewhat sparse in some areas that could use additional research. Additional data are needed to better quantify the species of various metals, including arsenic, chromium, and mercury. For example, emissions from more old/poorly controlled units could place upper bounds on the particulate models. In addition, there are relatively few controlled oil plant data sets and fewer gas test sites.

3.9 Reference

Roberson, R.L. and T.E. Eggleston, 1983. *Characterization of Radionuclide Emissions from Coal-Fired Utility Boilers*. Report No. 83-180-06F. Prepared for the Utility Air Regulatory Group by KEA (July 1983).

4

INDUSTRY EMISSIONS ASSESSMENT

Section 3 discussed the analyses of the PISCES field measurement data, representing measurements from about 3% of U.S. electric utility steam generating units. The purpose of the industry emissions assessment is to develop an estimate of trace substance emissions from the entire population of United States electric utility steam generating units as they will operate in a future "scenario" year, following compliance with all provisions of the 1990 CAAA.

The assessment of industry-wide emissions has three components:

1. Projections of power plant control technologies, fuel usage, and electricity generation in a future scenario year (Section 4.1)
2. Characterizations of trace element concentrations for utility fuels (Section 4.2)
3. Procedures for estimating trace substance emission rates as a function of various plant characteristics and operating conditions (Section 4.3).

4.1 Future Industry Operations

Industry scenarios were developed that project key characteristics of the future generating unit population in the year 2010 by reviewing information on the present status of the industry and then projecting compliance with the SO_2 provisions of the CAAA. Although the industry may experience additional changes due to competition and other factors that accelerate the replacement of older, less-efficient units with cleaner, more-efficient technologies, such factors were not addressed in the emissions assessment. The scenario also incorporates available information on planned modifications to particulate control equipment. The remainder of Section 4.1 describes how changes in the electric utility industry are represented by the future scenarios used in this study.

4.1.1 Future Scenarios

Title III of the CAAA specifies that EPA must project risks for the utility industry as it will be operating following compliance with other sections of the 1990 CAAA. Scenarios for assessment of future trace substance emissions must project the design and operations of electric utility steam-generating units for such a future time. Of the other provisions of the CAAA, Phases I and II of the 1990 SO_2 compliance provisions in Title IV are likely to have the greatest impact on trace emissions. The second and final phase of the SO_2 compliance

provisions begins in the year 2000. It is assumed that most SO_2 compliance strategies will be in place within a decade thereafter, and the industry will have stabilized with respect to SO_2 emissions. Therefore, 2010 was selected as the start year for the future scenarios used in this study. The future scenarios characterize the evolution of the industry, primarily in response to the requirements of the CAAA, over the 1995-2010 time frame. The future scenarios:

- are driven primarily by SO_2 compliance
- project changes in SO_2 control technology and fuels (Section 4.1.2)
- reflect planned modifications to particulate control technology (Section 4.1.3)
- project retirements of particular units by 2010.

The future scenarios do not focus on changes in the industry due to various other factors (e.g., penetration of advanced combustion technologies, competition, etc.). However, the base case scenario does account for redirection of some gas-fired steam-electric capacity to gas-fired combined cycle or combustion turbine capacity by 2010.

The future scenarios characterize steam-electric generating units with respect to the factors that impact trace substance emissions. These factors include:

- *The amount, source and composition of fuel burned annually in individual units.* Key considerations here are the amount of fuel switching anticipated under the CAAA; the relative roles of Powder River Basin, Central Appalachian and other low-sulfur coals; and specific or representative coal seams likely to be relied upon from different regions. Projections of fuel type and usage are based on SO_2 compliance modeling (Section 4.1.2).
- *The configuration of environmental controls at individual units.* The fraction of trace substances that is ultimately emitted depends primarily on the fraction captured by particulate control devices and wet FGD systems. The choice of SO_2 and particulate control devices will be influenced by CAAA compliance strategies and local particulate emission limits. Estimated FGD system retrofits (between 1990 and 2010), based on earlier studies by EPRI [1], range from about 20 GW, including 13 GW to be installed for Phase I SO_2 compliance, to 40 GW or more for Phase II SO_2 compliance. The base-case scenario provides unit-by-unit information concerning the type and performance of projected SO_2 and particulate control equipment. Projections of SO_2 control equipment are based on SO_2 compliance modeling (Section 4.1.2). Projections of particulate control equipment are based on a separate particulate survey (Section 4.1.3).

4.1.2 SO_2 Compliance Modeling—Base Case Scenario

The goals of the SO_2 compliance modeling for the base case scenario were to provide (1) a plausible "base case" scenario of the operation of electric utility steam generating units after implementation of the CAAA and (2) a common basis for EPRI and EPA to evaluate

the potential risks due to trace emissions from power plants. To meet these goals, this effort disaggregated a system-level base case scenario developed for EPA [2] to the unit level.

The SO_2 compliance modeling was performed by ICF Resources Incorporated using their Coal and Electric Utilities Model (CEUM). CEUM is an industry model based on engineering relationships and uses a linear programming optimization technique. The model incorporates current Phase I compliance plans (including current scrubber installation plans), as well as key assumptions affecting electric utility fuel consumption and emissions. The most significant assumptions with respect to trace substance emissions are electricity demand growth rate assumptions and existing coal plant availability, which influence in large measure the degree to which existing coal-fired units are operated.

Starting with the system-level results of a scenario developed for the EPA, CEUM was used to forecast the fuel use in 2010 of electric utility steam generating units. Unit-level forecasts were developed for (1) boilers that were on-line by 1991 and are not forecast to retire by 2010 and (2) boilers that are planned to be built before 2010 [3,4]. For coal-fired units, the projections include the rank, origin by supply region, and sulfur content of one or more types of coal, the heat input of each coal type, and type of SO_2 control, if any. The SO_2 controls include wet scrubbers, integrated gasification combined cycle (IGCC), and fluidized bed boilers. For oil- and gas-fired units, the operation projections include the heat inputs for oil and gas. The fuel usage and SO_2 control equipment projections of individual units were used in the base case scenario.

The scenario includes unidentified coal-fired capacity that is forecast by the model (rather than currently scheduled) to be built by 2010, since currently existing and announced capacity may be insufficient to meet future electricity demand. For each state with unidentified capacity, the base case scenario specifies the rank of coal to be used, origin by supply region, sulfur content of one or more types of coal, and the heat input of each coal type. All of the unidentified coal-fired capacity is assumed to be equipped with wet scrubbers. The unidentified capacity covers 23 states and represents a total heat input of about 2600×10^{12} Btus. Of the total heat input for the unidentified capacity, bituminous coal firing supplies about 90% and subbituminous coal supplies the remaining 10%.

Table 4-1 compares the projected heat input of electric utility steam generating units in 2010 to their actual heat input in 1990. The table reflects currently existing and announced electric utility steam generating units, and unidentified steam electric capacity. The percentage columns present the fraction of the total heat input supplied by each fuel. In both 1990 and 2010, coal supplies more than 80% of the total steam electric heat input. From 1990 to 2010, the coal heat input of units with scrubbers increases about 50%. The 2010 total steam electric heat input is about 23% greater than the 1990 total heat input, representing an annual growth rate for total fossil fuel-fired steam electric heat input of about 1%. The growth is primarily due to the projected 35% increase in steam electric coal heat

input industry-wide from 1990 to 2010. Oil and gas steam-electric heat inputs are projected to decrease between 1990 and 2010 by 45% and 20%, respectively. The decrease in gas steam-electric heat input results from two factors:

Table 4-1.
Heat Input by Fuel and SO_2 Control – Base Case Industry Scenario

Year	Heat Input (10^{12} Btu)				Percentage (%)			Heat Input (10^{12} Btu)
	Coal	Oil	Gas	Total	Coal	Oil	Gas	FGD Scrubber
1990	16,059	1,153	2,503	19,715	81	6	13	4,131
2010	21,629	630	1,999	24,258	89	3	8	6,163
Change	+5,570 (+35%)	-523 (-45%)	-504 (-20%)	+4,543 (+23%)				2,032 (+49%)

- First, the majority of gas steam-electric heat input, upon retirement, is assumed to be replaced with combined cycle and combustion turbine technology (for which data on trace substance emissions are still unavailable, and which are beyond the scope of the present study). These technologies are not included in the 2010 scenario used here, since they are not steam-electric generation capacity.
- Second, for the remaining gas steam-electric capacity expected to be replaced by units of the same type, the replacement gas units are excluded from the 2010 scenarios. This is because the contribution of gas plants to the SO_2 emissions for which the scenarios were originally developed was minimal. (As noted in Section 7 below, the inhalation risks from gas plants are also minimal.)

Of the 2010 steam electric coal heat input, 88% represents currently existing or announced capacity with sufficient data for risk calculations, 0.2% represents announced capacity with insufficient data for risk calculations, and 12% represents unidentified steam electric capacity. All of the 2010 steam-electric oil and gas heat input represent currently existing or announced capacity with sufficient data for risk calculations.

4.1.3 SO_2 Compliance Modeling—Alternative Scenarios

While the primary focus of the assessment was on the base case scenario, trace substance emissions also were evaluated for two alternative scenarios of fossil plant operations for the year 2010. The alternative scenarios provide a perspective independent from the base case. The alternative scenarios were developed by extending recent analyses conducted for EPRI of utility compliance with the SO_2 provisions of the 1990 CAAA, and relied on the Emissions Reduction Analysis Model (ERAM) and Utility Fuel Consumption Model [1].

The alternative scenarios include utility units burning coal and those burning residual oil. The alternative scenarios exclude units expected to burn only natural gas, which have very low emissions of most trace substances of concern and are forecast to include considerable future utility and non-utility capacity additions at locations not currently possible to identify.

Table 4-2 compares the two alternative scenarios evaluated in the industry emissions assessment and risk assessment. Appendix C discusses the alternative scenarios in detail, and compares them with recent government and industry forecasts. The "government trend" (GT) scenario is patterned on the 1994 Energy Information Administration (EIA) Reference Case [5], which has lower coal- and oil-fired generation and faster retirements than some major industry forecasts. In contrast, the "high trend" (HT) scenario projects higher coal- and oil-fired generation, more in the mid-range of industry forecasts. Including only announced retirements and limited unplanned additions, the HT scenario reflects high utilization of existing fossil units.

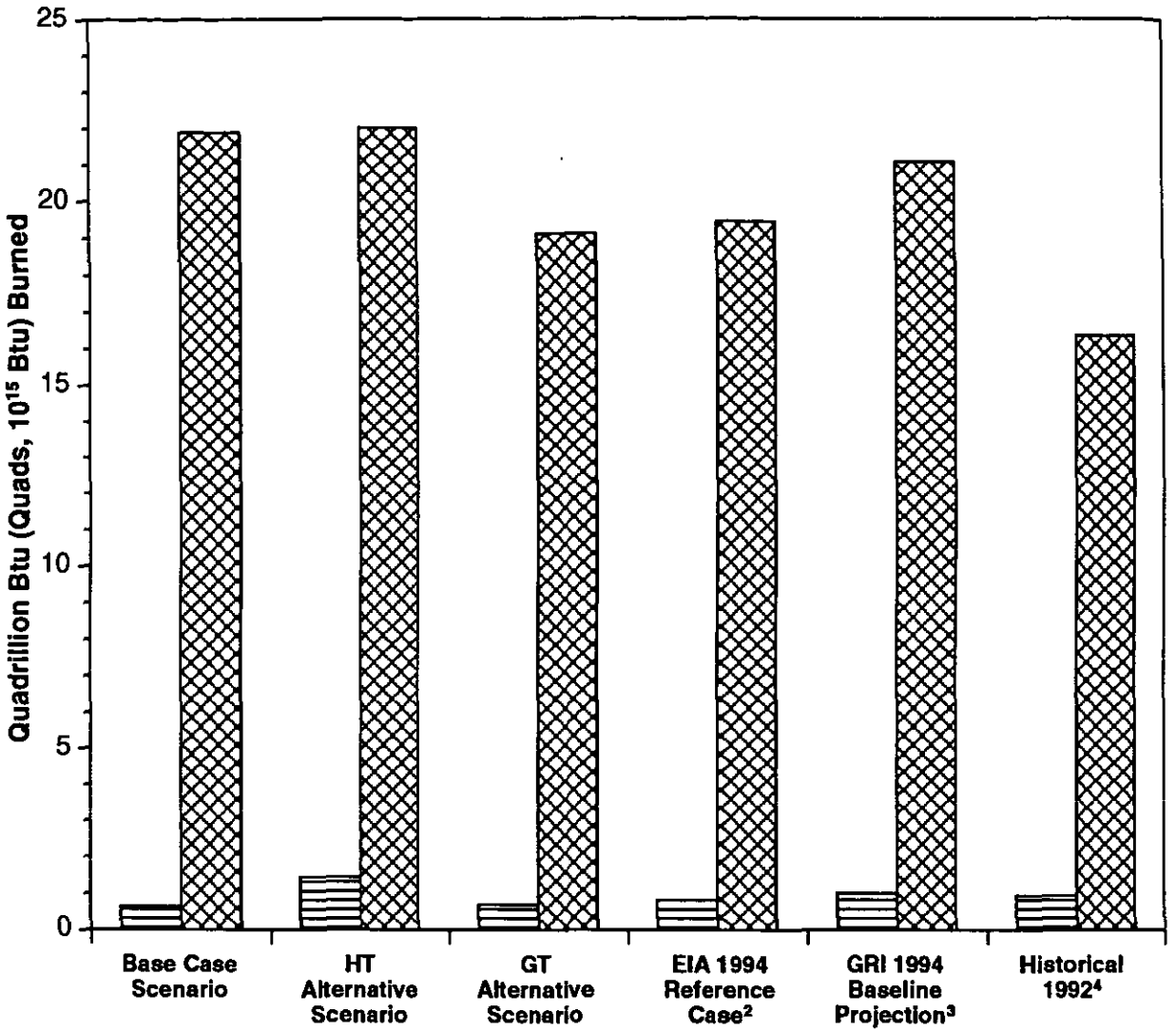
Table 4-2.
Alternative Fossil Plant Scenarios

Scenario	Total Generation	Oil-Fired Generation	Capacity Replacement	Allowance Trading
Government Trend	Low	Low	Fast	Constrained
High Trend	High	High	Slow	Perfect

Assumptions about the efficiency of the future SO_2 allowance market substantially influence projections of fuel switching and technology retrofits, and thus the potential for emissions of trace substances. The HT scenario incorporates perfect trading of SO_2 allowances, resulting in avoidance of costly fuel switches and retrofits of flue gas desulfurization (FGD) equipment. Recognizing that the trading of allowances is likely to be less than economically perfect, the GT scenario constrains inter-utility trading such that allowance purchases only occur when cost savings (relative to internal compliance) exceed 25% of the projected cost of the allowances.

4.1.4 Comparison of Future Scenarios

Figure 4-1 shows that, relative to the base case scenario, the HT scenario has similar overall coal consumption (although more concentrated in existing units) and the GT scenario has lower coal consumption. The figure also shows that the GT and HT scenarios have about 120% and 240%, respectively, of the base case residual oil consumption.



1 In addition, projected coal consumed at non-utility units ranges from 2-7% of utility coal consumption.

2 Energy Information Administration, *Annual Energy Outlook 1994 with Projections to 2010*, DOE/EIA-0383(94), January 1994 (after subtracting estimated 0.5 x 10^{15} Btus at non-utility units).

3 Holtberg, P., Woods, T., Lihn, M., and Koklauner, A., *Baseline Projection Data Book, 1994 Edition of the GRI Baseline Projection of U.S. Energy Supply and Demand to 2010*. Gas Research Institute, 1993.

4 Energy Informationn Administration, *Annual Energy Outlook 1994 with Projections to 2010*. DOE/EIA-0383(94), January 1994.

Figure 4-1.
Projected Year 2010 Coal and Oil Consumptions by Electric Utility Steam Generating Units[1]

With respect to the base case scenario, the alternative scenarios generally project higher consumption of Northern Appalachian and Powder River coals, and lower consumption of Illinois Basin, western bituminous, and, especially, Central Appalachian coals. These

differences reflect different assumptions regarding future coal prices, coal sulfur premiums, unplanned coal plant additions and their coal selections, future capacity factors, and the relative economics of coal switching versus FGD retrofits on a unit by unit basis.

Table 4-3 presents the portion of coals burned in scrubbed units by coal type. The portion of coals burned in scrubbed units depends on assumptions regarding long-term additions of new, scrubbed capacity in the western United States. There is general agreement among the scenarios that only about one-quarter of the Powder River coal will be burned at scrubbed units.

Table 4-3.
Projected Percent of Coal Burned at Units with FGD[1] in Year 2010, by Region (10^{12} Btu)

	Alternative Scenarios		Base Case
Coal Region	HT	GT	
N. Appalachian	73	61	62
C. Appalachian	20	21	12
S. Appalachian	15	15	7
Illinois Basin	80	74	87
Interior West	91	94	100
Rocky Mountain	57	61	74
Southwestern	81	82	64
S. Wyoming	40	35	83
Powder River	20	29	19
Gulf Lignite	77	81	71
Plains Lignite	67	80	68
All Coals	42	43	40

1. FGD includes wet scrubbers and spray dryers. It excludes sorbent injection, fluidized bed boilers (about 1.3 GW in alternative scenarios), and integrated coal gasification-combined cycle units (about 0.5 GW in alternative scenarios).

4.1.5 Particulate Survey

As explained in Section 3, particulate emissions are a key factor for estimating emissions of particulate-phase inorganic substances. The particulate emissions of plants that undergo modifications to their particulate control equipment are likely to be lower in 2010

than in 1990. To gain information on the current status of control equipment and planned modifications, the Utility Data Institute (UDI) conducted a survey of operators of all U.S. utility steam electric generating units [6].

The survey response was 68% on the basis of nameplate capacity. No response was received from operators of 831 units (representing 150,700 MW), while operators of 1,215 units (284,800 MW) reported that no modifications are planned. Operators of 132 units (39,730 MW) reported that some equipment or fuel modifications are either planned or have been recently completed. The information from the survey was used to determine the particulate controls of individual units for the base case scenario and the alternative future scenarios.

4.2 Characterization of Trace Substances in Utility Fuels

4.2.1 Introduction

This section describes the sources of fuel composition data used in the estimation of industry-wide emissions. The fuel analyses available from the PISCES project and other sources are heavily weighted toward coal, reflecting that coal capacity accounts for about 70% of fossil fuel generating (nameplate) capacity and about 80% of the fossil generation. Many of the non-PISCES coal analyses, however, reflect "in-the-ground" coals rather than "as-fired" coals. To augment the available coal analyses, EPRI researchers estimated the concentrations of trace substances in "as-fired" coals and measured the mercury concentrations in delivered coals for use in the base case scenario. EPRI also developed a database of proprietary "as-shipped" coals for the alternative scenarios. Diverse sources were used to obtain information on fuel composition.

For oil and natural gas, PISCES data were used. An additional special study was conducted of "as-burned" oil and coal samples to ascertain mercury concentrations.

4.2.2 Coal

4.2.2.1 Coal Characterizations Based on Non-Proprietary Data (Base Case Scenario)

The source of coal data for this scenario was an adjusted subset of the U.S. Geological Survey (USGS) COALQUAL data set. EPRI used the same subset provided EPA in 1992. This subset, made up of approximately 3,300 samples, included information on the top 50 coal-producing seams for 1991. The USGS data include core and channel coal samples and represent the entire height of a coal seam, often including interbedded rock and minerals. The data are representative of "in-the-ground" coal quality, not necessarily "as-shipped" or "as-burned" coal. The data set as maintained by USGS hence may not be representative

of utilities burning eastern and midwestern coals, because about 75% of those coals are cleaned or washed before combustion, reducing the concentration of many trace substances [8,9].

To develop information more representative of "as-shipped" or "as-fired" coals for the base case scenario, the COALQUAL data set was reviewed and screened in cooperation with USGS. Based on the screening criteria in Table 4-4, USGS removed entries representing coal seams too thin or deep to be mined economically, as well as obvious samples of interbedded rock and minerals (partings), reducing the data set from about 3,300 samples to roughly 2,700 samples. A preliminary examination of the screened data shows a moderate decrease in trace substance concentrations.

Table 4-4.
Screening Criteria for the Raw USGS Coal Seam Database for Economically Unrecoverable Coal Samples

General Criteria for Uneconomic Coal (Covers All US including Appalachia Coal)	Seam Height	Seam Depth
Weathered channel or grab sample	all	all
Too thin for underground or surface mining	<9"	
Too deep for underground mining (Central & Southern Appalachia)		>3500'
Too deep for underground mining (Northern Appalachia)		>2000'
Too deep for surface mining and too thin for underground mining	<24"	≥400'
Illinois Basin Coal		
Too thin for underground or surface mining	<12"	
Too deep for underground mining		>1500'
Too deep for surface mining and too thin for underground mining	<42"	>200'
Powder River Basin Coal		
Too thin for surface mining	<60"	
Too deep for surface mining		>300'

Table 4-4.
Screening Criteria for the Raw USGS Coal Seam Database for Economically Unrecoverable Coal Samples (Continued)

General Criteria for Uneconomic Coal (Covers All US including Appalachia Coal)	Seam Height	Seam Depth
Lignite Coal		
Too thin for surface mining	<36"	
Too deep for surface mining		>200'
San Juan Basin Coal		
Too thin for surface mining	<324"	
Too deep for surface mining		>300'
Rocky Mtn Coal (Uinta, Green River, Raton Mesa)		
Too thin for surface mining	<18"	
Too deep for surface mining and too thin for underground mining	<42"	>300'
Interior Basin Coal		
Too deep for underground mining	>500'	
Too deep for surface mining and too thin for underground mining	<30"	>200'

Algorithms were then developed to allow refinement of the screened data set from USGS to be more representative of "as-shipped" coal quality [10]. These algorithms are based on the limited amount of published data from industry and EPRI research, and include material balances around several configurations of coal cleaning plants. The algorithms were applied to selected entries in the refined COALQUAL data set to develop a data set more representative of "as-fired" coals.

Table 4-5 presents trace substance concentrations (in $lb/10^{12}$ Btu) of 22 "as-fired" coals, based on applying the algorithms to the refined COALQUAL data set. As described in Appendix C, these coals were used to characterize the concentrations of ten trace substances (except for mercury) for the base case scenario. Of the 22 coals, 18 have more than ten samples. The bituminous coal data cover 15 regions in 14 states, the subbituminous coal data cover five regions in four states, and the lignite data cover two regions in two states.

Table 4-5.
Trace Substance Concentrations of "as-fired" Coals from USGS COALQUAL Database

State	Coal Supply Region	Rank	Sample Size	Concentration (lb/10^{12} Btu)									
				As	Be	Cd	Cl	Cr	Hg	Mn	Ni	Pb	Se
Alabama	Southern Appalachian	BIT	1	1,712	155	4	30,569	1,710	13	3,560	1,194	420	142
Colorado	Green River	BIT	26	54	58	5	8,685	234	2	798	176	371	70
Illinois	Eastern	BIT	47	519	121	81	91,031	1,096	7	3,473	1,024	1,667	150
Indiana	Eastern	BIT	80	532	205	24		997	6	2,880	1,108	587	149
Kentucky	Central Appalachian	BIT	337	1,460	241	4	86,877	1,083	11	1,324	1,051	488	293
Kentucky	Eastern	BIT	116	927	192	22	28,037	1,484	11	3,941	1,294	727	193
Maryland	Northern Appalachian	BIT	38	1,275	122	8	21,916	1,476	20	881	1,124	478	183
New Mexico	San Juan River	BIT	3	41	114	9	8,786	434	2	1,705	613	522	108
Ohio	Northern Appalachian	BIT	492	1,398	194	9	50,980	1,081	16	2,515	1,067	493	287
Pennsylvania	Northern Appalachian	BIT	539	1,769	175	6	53,199	1,289	17	1,729	1,199	603	224
Tennessee	Central Appalachian	BIT	12	2,231	79	7	3	740	13	1,037	577	267	218
Utah	Uinta	BIT	22	40	30	4	251	302	2	366	193	206	98
Virginia	Central Appalachian	BIT	52	722	117	4	41,490	805	8	1,305	707	344	196
West Virginia	Central Appalachian	BIT	280	569	211	7		1,021	9	1,087	936	463	271
West Virginia	Northern Appalachian	BIT	101	851	134	5		1,037	11	1,760	767	352	229
		Median		851	134	7	29,303	1,037	11	1,705	1,024	478	193

Table 4-5.
Trace Substance Concentrations of "as-fired" Coals from USGS COALQUAL Database (Continued)

State	Coal Supply Region	Rank	Sample Size	Concentration (lb/10^{12} Btu)									
				As	Be	Cd	Cl	Cr	Hg	Mn	Ni	Pb	Se
Colorado	Green River	SUB	1	65	60	6	6,470	125	1	1,150	92	506	110
Montana	Powder River	SUB	95	335	49	7	2	302	7	2,515	401	291	62
New Mexico	San Juan River	SUB	104	371	307	21	4,986	752	10	5,197	527	4,984	254
Wyoming	Green River	SUB	2	289	29	9		806	23	4,099	315	616	143
Wyoming	Powder River	SUB	141	290	48	8	5,773	667	13	1,902	455	281	95
		Median		290	49	8	5,380	667	10	2,515	401	506	110
North Dakota	Fort Union	LIG	56	946	39	11	534	571	19	10,284	322	479	95
Texas	Texas	LIG	54	288	152	9	1	1,037	17	12,396	714	470	584
		Median		617	95	10	267	804	18	11,340	518	475	340
		Min		40	29	4	1	125	1	366	92	206	62
		Median		550	122	8	8,736	901	11	1,831	711	479	166
		Mean		758	129	12	24,422	866	11	2,996	721	710	189
		Max		2,231	307	81	91,031	1,710	23	12,396	1,294	4,984	584

Of all substances analyzed, chlorine has the highest median concentration across all samples, at 8,700 lb/10^{12} Btu, followed by manganese at 1,800 lb/10^{12} Btu. Median concentrations of chromium, nickel, and arsenic are between 450 lb/10^{12} Btu and 900 lb/10^{12} Btu, and the median concentrations of all other substances are less than 200 lb/10^{12} Btu.

To provide more definitive information on the mercury content of "as-fired" coals, 154 samples of domestic "as-received" coal provided by utility operators were analyzed by atomic fluorescence. The data set comprises 106 bituminous coal samples, 37 subbituminous samples, and 12 lignite samples, representing at least 20 major seams. As described in Appendices C and D, these samples were used to characterize the concentrations of mercury in coal for the base case scenario. Table 4-6 compares mercury concentrations in Pittsburgh seam coals and other bituminous coals from the mercury study and the power plant measurements discussed in Section 3 with those from the USGS data. The data indicate that the average mercury concentration in "as-shipped" or "as-received" coal (as represented by the EPRI study of mercury in coal) is about 50% lower than in "in-the-ground" coal represented by the USGS data [11]. Appendix C summarizes the mercury concentrations in coal by state and rank.

Table 4-6.
Comparison of Mercury Concentration in Selected Coal Data

	USGS[a]	Revised USGS Data Set[b]	EPRI Mercury Study[c]	PISCES FCEM[d]
Bituminous Coals				
Average (ppm)	0.21	0.19	0.09	0.12
No. of Samples	3,500	2,300	106	47
Pittsburgh Seam Coal				
Average (ppm)		0.21	0.1	
No. of Samples		29	13	

a EPA, "Locating and Estimating Air Emissions from Sources of Mercury and Mercury Compounds," EPA-454/R-93-023, Sept. 1993.

b Akers, D. November, 1993, personal communication of information from revised USGS database.

c Baker, S.S., 1994. "EPRI Mercury in Coal Study: A Summary Report for Utilities that Submitted Samples," Systems Applications International, Morrisville, N.C.

d Section 3

4.2.2.2 Coal Characterizations—Alternative Scenarios

The alternative fossil plant scenarios used data on trace substance concentrations from a different set of coal samples drawn from the same regions as the samples in the base case scenario. The alternative coal characterization is based on analyses of 1,530 samples of "as-shipped" or "as-received" coal from major coal suppliers, as well as from utilities. The data are considered proprietary [12]. Trace substance concentrations in this dataset were weighted by the corresponding coal production volumes to create a new adjusted set of concentration data. It is important to note that the trace substance concentrations in the base case scenario were not production weighted. In each case, coal concentrations were matched to particular coal seams, and then to particular boilers and generating units employing coal from those sources.

This analysis found that higher-sulfur and higher-ash coals from each region generally contain higher levels of trace elements. Therefore, the alternative coal characterization methodology distinguishes coals within each region partly on the basis of sulfur content. This approach improves the estimation of future trace element levels in burned coals, especially as influenced by coal switching to reduce SO_2 emissions. When combined with the use of as-shipped coal samples weighted by production volumes, this provides estimates of trace element levels in burned coals that are lower than the coals in the base case. Table 4-7 shows that the alternative characterization reflect lower as-shipped mercury and arsenic levels for most of the major coal regions than those in the base case scenario.

Table 4-7.
Projected Arsenic and Mercury Levels in Coals Burned: Base Case versus Alternative Scenarios[1]

Region	Arsenic ($lb/10^{12}$Btu)		Mercury ($lb/10^{12}$ Btu)	
	Base Case	Alternative	Base Case	Alternative
Northern Appalachia	1584	484	11.4	10.1
Central Appalachia	1188	396	7.3	5.1
Illinois Basin	770	396	6.4	7.0
Gulf Coast Lignite	484	44	28.2	5.1
Powder River Basin	330	121	7.5	7.3

[1] For each region, levels represent averages over coal subcategories weighted by consumption (measured in Btus). For the alternative scenarios, averaging also is done over the different alternative industry scenarios.

4.2.3 Oil

Emissions factors for oil-fired plants were used in the emissions assessment, since the number of data sets currently available for oil is extremely limited. Only 12 oil sites were tested under the PISCES FCEM projects, and detailed fuel analyses were not available for all sites. All 12 tested sites fired residual oils. In the 1990 Power Statistics database, a specific type of oil was designated for 361 of 1122 units that have the capability to fire with oil. Of these 361 units, residual oil (e.g., fuel oil #6) was specified for 87%. Several other fuel oils, including distillate oil (e.g., fuel oil #2), were specified for the remaining 13%. The future scenarios used in Section 4.1 did not specify oil type, hence data for residual oil were used throughout. Since #2 distillate oil burns cleaner than #6 residual oil, the use of emission factors based on residual oil measurements results in conservative risk estimates if some of the oil burned in the future scenario is, in fact, #2.

Additional analyses of No. 6 fuel oil samples were conducted. Twenty-two oil samples were collected by cooperating utilities from fuel feed lines into boilers; these samples were then split and archived, with one split being sent to EPA for radionuclide analysis. Supplemental analyses were then conducted for other trace constituents, and the results were compared to those of the field measurements [13]. The supplemental analyses were generally consistent with the field measurements with the exception of beryllium, cadmium, mercury, selenium, and to a lesser extent arsenic. If the oils burned in the 2010 through 2080 assessment period are similar to those measured in the supplemental analyses, the assessment may overstate the emissions of these substances.

4.2.4 Natural Gas

The data on natural gas are limited by the number of sites tested and the difficulty in measuring trace elements in natural gas. Although the PISCES FCEM data sets include nine natural gas-fired boilers, only two gas-fired plants were fully tested. Furthermore, many of the measurements were at or below detection limits, particularly for trace composition of the gas fuel. Consequently, trace substance concentrations for gas-fired units were based on emission factors rather than fuel composition, for all such units.

4.3 Emissions Estimation Procedure

Using the unit data from the future scenarios, plant measurements, and fuel analyses, EPRI developed a procedure for estimating power plant emissions of trace substances in future years. This procedure integrated information from scenarios of industry operations, data on trace substances in utility fuels, and the recommended emission-estimating equations derived from the field data (see Section 3).

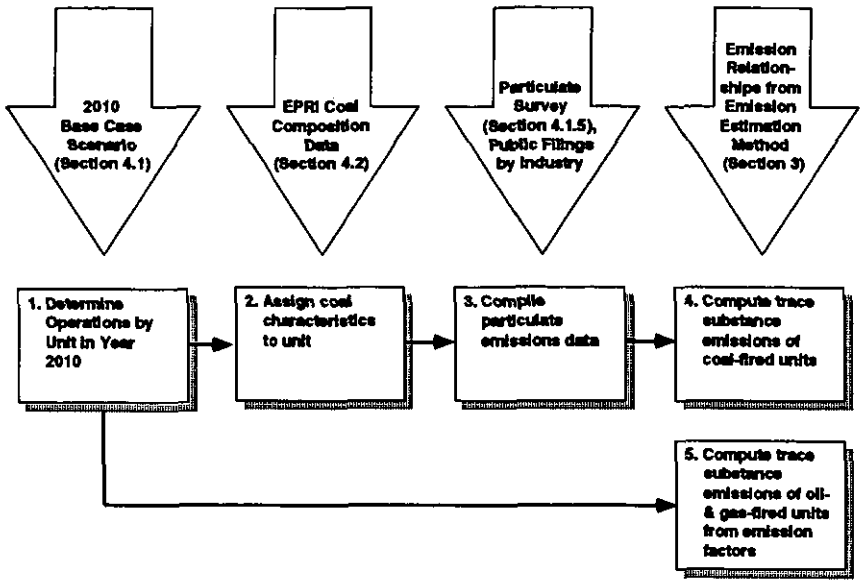


Figure 4-2.
Steps of the Industrywide Emissions Estimation Procedure

As shown in Figure 4-2, the emission estimation procedure had five steps:

1. *Information from the future scenario (Section 4.1) was used to determine the operations of individual units in 2010.* The operations data include projections of fuel types and sources, average annual fuel consumption for each fuel, retirement year, control technologies, and particulate emission rate.
2. *Coal characteristics were assigned to coal-fired units based on coal composition research.* The important coal characteristics are average concentrations of trace substances and average heat content in representative coal supplies. Coal characteristics from the adjusted COALQUAL database were assigned to units based on the rank and origin of the coals (e.g., Wyoming subbituminous coal from the Powder River basin) specified for each unit in the future scenario for the base case. Appendix C discusses how coal characteristics were assigned to units in the base case scenario.
3. *Particulate emissions of coal-fired units were compiled.* Based on the particulate survey, the base case scenario, and data contained in publicly-available filings by individual utility operators, particulate emissions were determined for each unit firing coal. Appendix C provides details on the particulate emission determinations.
4. *Trace substance emissions for coal-fired units were calculated.* Based on the projected control configuration, specific sets of emissions relationships were assigned to individual coal-fired units. For particulate-phase metals, statistical correlations relate trace substance concentration in the coal and particulate emission rates to trace substance emission rates. For mercury, selenium and chlorine, a specific fraction of the trace substance input to the boiler, depending on the coal rank and/or control equipment, was assumed to be emitted. For organic compounds, emission factors by plant type

were used. Using the appropriate equations along with projections of future operations from Step 1, fuel characteristics from Step 2, and particulate emissions from Step 3, estimates of trace substance emissions for individual units were calculated.

5. *Trace substance emissions for oil- and gas-fired units were calculated.* Based on projected control configuration, specific sets of emissions relationships were assigned to individual oil- and gas-fired units. For oil- and gas-fired generation, emission factors (mass of substance per unit heat rate) were used for all substances. Using the emissions factors along with projections of future operations from Step 1, estimates of trace substance emissions for individual units were calculated.

This approach was applied to all units in the base case and alternative scenarios, including those measured under the PISCES FCEM project. The result was a set of trace substance emission estimates for each unit in the base case scenario and similar sets of emission estimates for units in the alternative scenarios, for the year 2010.

4.4 Comparison of Modeled and Measured Emission Rates

The data needed to model emissions using the methodology described in Section 4.3 include the trace substance and ash content in coal, and the particulate emission rate. For the industry-wide assessment, these data are obtained from databases. For selected units, however, the PISCES program has obtained specific emission measurements during coal-firing. Since the input data that the modeling methodology requires has also been measured for these selected units, it is possible to model emission rates using field data for these plants and compare the results.

In general, emissions modeled by using field data input (that is, fuel concentrations measured contemporaneously with PISCES emissions measurements) yield more accurate estimates for the period in which field measurements were conducted than emissions modeled with database values that may not be representative of the measurement period. Using field data, modeled arsenic emissions were within or extremely close to the 95% confidence interval for all eight of the detected measurements. Using database values, modeled emissions were higher than the upper bound of the 95% confidence interval for six of the nine detected measurements. The median estimate using field data input is 70% of measured emissions, while the median estimate using database values is 340% of measured emissions. These results suggest that the database coal concentration and particulate emission values are not representative of the period during which the field tests were conducted.

Based on the comparisons, it appears that the modeling method in Section 3 is sufficiently accurate for use in the risk analysis. It also appears that emissions estimates based on field data input are better suited for estimating emissions that were measured during the time period the field tests were conducted. Field data, however, are based on a very short period of time (generally 3 to 4 days), so it is not clear to what extent they are representative of long-term operations. The field data may, in fact, be less representative of long-

term operations than the database values. The industry-wide assessment relied on database values, since measured values cannot be demonstrated to be applicable to future long-term operation of those same units, and since measured values are not available for the entire nationwide set of units or plants. The accuracy of emission estimates used in the risk assessment will depend heavily upon the extent to which database values are representative of future operations.

4.5 Emission Rates for Coal Plants

Table 4-8 summarizes estimated emission rates of inorganic substances from coal-fired units. (Figure 4-3 presents the same information in graphical format.) For each unit firing coal, the emission rates of inorganic substances were modeled using Steps 1 through 4 of the procedure described in Section 4.3. The emission rates serve as an intermediate calculation for the emissions input to dispersion modeling in the next section. (The emission rate for a particular substance in lb/10^{12} Btu multiplied by the annual heat input equals the annual emissions in lb/year.)

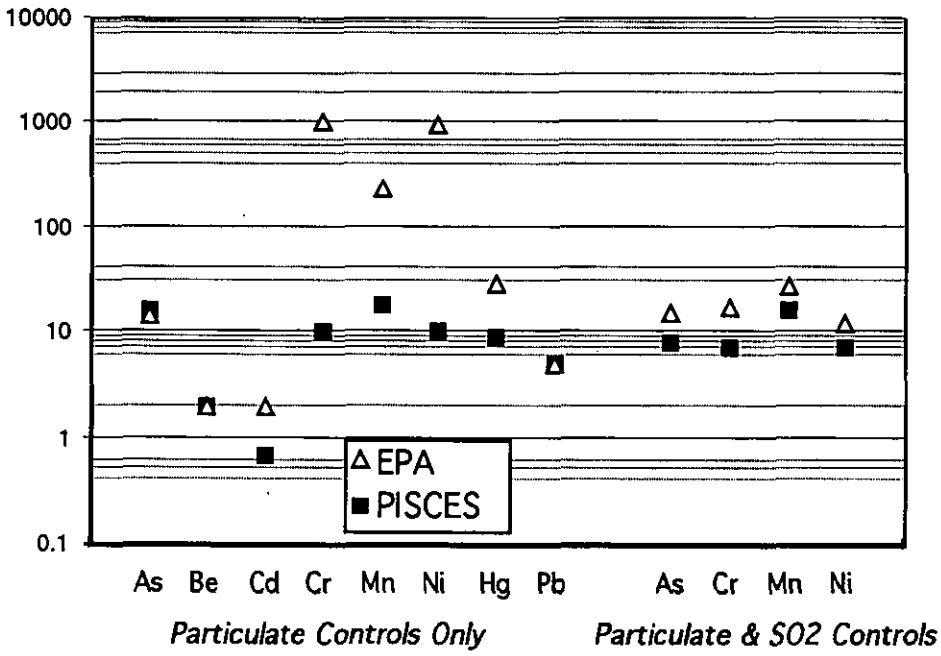


Figure 4-3.
Comparison of Emission Factors Based on PISCES Field Measurements with Values in EPA (1989) (Coal-fired Units Only, lbs/10^{12} Btu)

Table 4-8.
Emissions Rate Summary for Units Burning Only Coal (lb/10^{12} Btu)

		Modeled		Reported by EPA*	
		Particulate Controls Only	Particulate & SO2 Controls	Particulate Controls Only	Particulate & SO2 Controls
As	median	10.	4.		
	mean	16.	8.	14.	15.
	high	131.	120.	242.	
	low	0.15	0.33	0.35	
Be	median	0.84	0.35		
	mean	2.	0.69	2.	
	high	20.	14.	32.	
	low	0.02	0.01	0.11	
Cd	median	0.61	0.57		
	mean	0.7	0.72	2.	
	high	4.	4.	53.	
	low	0.1	0.08	0.2	
Cr	median	9.	6.		
	mean	10.	7.	1,007.	17.
	high	41.	56.	7,970.	
	low	1.	1.	1.6	
Mn	median	14.	13.		
	mean	18.	16.	232.	28.
	high	91.	154.	9,240.	
	low	2.	1.	1.	
Ni	median	9.	5.		
	mean	10.	7.	976.	12.
	high	31.	35.	5,760.	
	low	1.	1.	70.	
Pb	median	6.	6.		
	mean	9.	8.	29.	
	high	48.	77.	91.	
	low	0.58	0.27	6.	
HCl	median	47,973.	1,402.		
	mean	46,885.	2,062.		
	high	140,953.	50,156.		
	low	0.59	0.06		

Table 4-8.
Emissions Rate Summary for Units Burning Only Coal (lb/10^{12} Btu) (Continued)

		Modeled		Reported by EPA*	
		Particulate Controls Only	Particulate & SO2 Controls	Particulate Controls Only	Particulate & SO2 Controls
Hg	median	6.	4.		
	mean	5.	4.	5.	
	high	15.	12.	22.	
	low	1.	1.	0.115	
Se	median	117.	23.		
	mean	100.	28.		
	high	451.	142.		
	low	3.	3.		

* EPA reports emission factors in Estimating Trace Substance Emissions from Coal and Oil Combustion Sources, EPA-450/2-89-001, U.S. EPA, Office of Air and Radiation, Research Triangle Park, NC, April, 1989. Mean values are averages of average emission factors over different coal types. Median, high, and low values are for the particular power plant at that point in the emissions distribution.

The median and mean emission rates of inorganic substances from coal-fired units with particulate controls-only are higher than those for coal plants with particulate and SO_2 controls. For 8 of the 10 substances, the emission rates for plants with particulate controls only are up to twice the emission rates of plants with both particulate and SO_2 controls. For both types of plants, the mean and median emission rates are less than 20 lb/10^{12} Btu for all substances except chloride and selenium. Selenium emission rates (for particulate controlled plants) are about an order of magnitude larger, at about 100 lb/10^{12} Btu, while median chloride emission rates are about 1,400 and 48,000 lb/10^{12} Btu for particulate/SO_2 controlled plants and particulate controlled plants, respectively.

Table 4-8 also presents emission rates reported by an earlier EPA report. While 5 of 8 inorganic substance emission factors are comparable to the modeled emission rates, 3 substances have substantially different values. Average emission factors for chromium and nickel reported by the EPA report are two orders of magnitude higher than the modeled emission rates, and average EPA manganese emission factors are one order of magnitude higher. The modeled emission rates generally have narrower ranges than those reported by EPA.

4.6 Industry Emissions Results

Table 4-9 summarizes results of the industry emissions assessment. For each electric utility steam generating unit, the emissions of each trace substance was estimated using the procedure described in Section 4.3. The unit-level emissions estimates, aggregated to the stack serving each unit, and then aggregated to the power-plant level, served as the emissions input to the dispersion modeling in the next section. Although emissions levels influence the ultimate risk estimates, these estimates are also influenced by other factors such as dispersion characteristics and dose-response functions.

Table 4-9.
Plant Emissions Summary by Plant Group[1] (lb/year, by plant)

		Coal		Oil		Gas	Mixed
		particulate controls only	particulate & SO2 controls	uncontrolled	controlled		
As	median[3]	126.	88.	16.	9.	0.69	5.
	mean[2]	230.	178.	20.	22.	1.	10.
	high[3]	3,965.	1,852.	85.	90.	13.	111.
	low[3]	0.96	3.	3.	0.99	0.09	0.35
Be		11.	8.	0.6	0.34	0.03	0.19
		23.	16.	1.	1.	0.05	1.
		506.	188.	3.	3.	0.56	12.
		0.11	0.14	0.1	0.04	4.0×10^{-3}	0.01
Cd		7.	16.	4.	2.	0.12	1.
		12.	19.	5.	5.	0.19	2.
		93.	93.	20.	21.	2.	8.
		0.53	0.69	0.65	0.23	0.02	0.08
Cr		115.	145.	16.	10.	3.	8.
		168.	189.	19.	21.	5.	12.
		1,415.	1,060.	81.	85.	61.	77.
		7.	11.	3.	0.94	0.44	0.42
Mn		174.	299.	39.	22.	1.	13.
		303.	461.	46.	51.	2.	21.
		3,621.	3,602.	202.	213.	22.	88.
		8.	12.	7.	2.	0.16	0.82
Ni		107.	142.	2,094.	1,170.	7.	380.
		156.	180.	2,558.	2,793.	11.	878.
		1,218.	790.	11,161.	11,794.	133.	4,192.
		7.	11.	360.	130.	0.96	43.

Table 4-9.
Plant Emissions Summary by Plant Group[1] (lb/year, by plant) (Continued)

	Coal		Oil		Gas	Mixed
	particulate controls only	particulate & SO2 controls	uncontrolled	controlled		
Pb	84. 145. 1,944. 4.	140. 186. 875. 4.	21. 25. 109. 4.	12. 28. 115. 1.	1. 2. 22. 0.16	7. 11. 57. 0.46
HCl	378,712. 763,918. 5,622,419. 1.	17,528. 38,710. 396,554. 0.31	6,960. 8,520. 37,200. 1,200.	6,480. 15,504. 65,520. 720.	. . . .	2,160. 16,811. 582,874. 240.
Hg	60. 102. 1,013. 3.	87. 140. 933. 6.	1. 2. 7. 0.23	1. 3. 13. 0.14	$2.4x10^{-3}$ $3.8x10^{-3}$ 0.04 $3.2x10^{-4}$	0.41 3. 36. 0.05
Se	896. 1,704. 22,766. 5.	507. 845. 5,911. 21.	6. 7. 31. 1.	5. 13. 55. 0.6	0.06 0.09 1. 0.01	2. 31. 1,101. 0.2
Benzene	47. 68. 328. 2.	100. 110. 365. 5.	3. 5. 17. 0.63	4. 8. 30. 0.33	2. 4. 44. 0.32	3. 6. 27. 0.19
Formaldehyde	37. 54. 259. 1.	79. 87. 288. 4.	69. 100. 327. 13.	72. 166. 546. 6.	102. 161. 1,890. 14.	86. 149. 581. 5.
PAH	0.02 0.03 0.16 $8.6x10^{-4}$	0.05 0.05 0.17 $2.2x10^{-3}$	0.01 0.01 0.06 $1.9x10^{-3}$	0.01 0.02 0.1 $1.1x10^{-3}$	. . . .	$3.1x10^{-3}$ 0.01 0.03 $3.8x10^{-4}$
Dioxins/ Furans	$2.5x10^{-5}$ $3.6x10^{-5}$ $1.7x10^{-4}$ $9.6x10^{-7}$	$5.3x10^{-5}$ $5.8x10^{-5}$ $1.9x10^{-4}$ $2.4x10^{-6}$	$2.4x10^{-5}$ $2.9x10^{-6}$ $1.3x10^{-4}$ $4.2x10^{-6}$	$2.2x10^{-5}$ $5.4x10^{-5}$ $2.3x10^{-4}$ $2.5x10^{-6}$	. . . .	$6.6x10^{-6}$ $1.4x10^{-5}$ $7.5x10^{-5}$ $8.3x10^{-7}$

Table 4-9.
Plant Emissions Summary by Plant Group[1] (lb/year, by plant) (Continued)

	Coal		Oil		Gas	Mixed
	particulate controls only	particulate & SO2 controls	uncontrolled	controlled		
Toluene	17.	37.	33.	36.	30.	29.
	25.	41.	44.	75.	47.	50.
	121.	135.	158.	270.	556.	191.
	0.67	2.	6.	3.	4.	2.

1 Plant group: all plants with a given fuel and control device configuration.
2 Mean is based on aggregating emissions of a given trace substance across all plants in a single plant group, then normalizing by the number of plants in that group.
3 Median, high, and low entries are calculated emissions of a given substance for the single power plant at that point (middle, high, or low) in the distribution, for the approximately 600 plants in the industry population.

Therefore, one cannot rely *a priori* solely on emissions to infer the absolute or relative level of risk from a plant. The inhalation risk assessment in Section 7 provides further perspective on the relationship between the level of emissions and the calculated health risks.

To provide insight into the level and variability of emissions from different types of plants, the substance-specific emissions were aggregated by plant group; here, "plant group" refers to all power plants of a particular of fuel type (coal, oil, gas, or a mix of fuels) and control devices. The table shows the mean value across all plants of a given group, and the values for the particular plants in each group with the median, highest, and lowest long-term emissions of each substance.

The median emission values in Table 4-9 are lower than the mean values, reflecting that the distributions are positively skewed (that is, most plants lie below the average value for each substance). Coal plants (both those with only particulate controls and those with both particulate and SO_2 controls) tend to have the highest total emissions, relative to other plant groups. However, oil plants (controlled and uncontrolled) show the highest nickel emissions, and relatively high emissions of organic compounds (e.g., formaldehyde, dioxins/furans, and toluene) compared to other plant groups. Gas plants, while exhibiting low inorganic emissions, show high emissions of organic compounds, relative to other plant groups.

4.7 Summary

The industry-wide emissions assessment uses data from a variety of sources: SO_2 compliance modeling, the UARG particulate survey, coal composition information, industry databases, and emission estimation procedures developed in Section 3. The method uses input from industry databases rather than field data, because the field data are available

for only about 50 units. Application of the procedures yielded a set of trace substance emission estimates for electric utility steam generating units as they are projected to operate in 2010. The emission estimates were generally found to be within or beyond the upper 95% confidence interval of measured emissions of PISCES sites, indicating that the estimates are generally conservative compared to measurements. The emission estimates are a key input for the dispersion modeling in the next section, and the risk assessment.

4.8 References

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5

INHALATION EXPOSURE ASSESSMENT

Inhalation exposure assessment provides the link between environmental releases of substances, estimated in Section 4, and the assessment of human health impacts, described in Sections 6 and 7. This section summarizes the methodology for assessing inhalation exposure to trace substances emitted by power plants. The exposure methodology involves both (1) modeling the dispersion of trace substances emitted from the power plant and (2) identifying groups of individuals exposed to various atmospheric concentration levels of substances emitted from the power plant (refer to Figure 1-1). The exposure methodology also involves reasonably modeling the level of exposure for typical individuals due to nearby power plants.

The inhalation exposure assessment was guided by the following conventions:

- The base case assessment estimates inhalation exposure to substances emitted from power plants for individuals who reside in the vicinity of power plants for 70 years (from 2010 to 2080). The base case assumes that each unit will continue to operate for 70 years with the configuration and fuel usage projected for 2010.
- The inhalation exposure assessment estimates two standard measures of exposure, from which risk measures can be derived: the exposure of the maximally exposed individual (MEI) and the average population exposure. These exposure measures are based on conservative assumptions about individual behavior that may affect exposure and risk.
- An additional inhalation exposure measure was derived, the reasonably exposed individual (REI), to accommodate finer time and space resolution, and to provide insight on how patterns of human activity may impact exposure. In addition, the REI measure deviates from the base case by assuming that units are replaced after 55 years of operation with units that meet the current New Source Performance Standards (NSPS) for particulate emissions.

Deriving exposure measures requires information on atmospheric concentrations, and patterns of residence with respect to these concentrations. Within this section, descriptions of the basic concepts of exposure, exposure modeling, and exposure measures used in this assessment are followed by an explanation of the specific methods used for dispersion modeling and exposure assessment. Section 5.4 describes the dispersion modeling, utilizing EPA's Industrial Source Complex Long Term 2 (ISCLT2) model, that provided atmospheric concentration estimates of trace substances due to power plant emissions at

locations within 50 km of each plant. Section 5.5 describes analyses of U.S. Census data, used to determine patterns of individual residence relative to the patterns of atmospheric concentrations of utility-emitted trace substances.

5.1 Background

Exposure and dose assessment are concerned with how substances in various media enter the body, cross absorption barriers, and then react within the body to adversely affect health. The extent of exposure depends on the concentrations of the substance in the various environmental media and on the duration of contact. The dose depends on the quantity of each substance that crosses the outer boundary of the body and the extent to which the substance is available to sites within the body. This section defines the terminology used in the exposure assessment.

5.1.1 Exposure

Current EPA guidelines define *exposure* as the contact of a substance with the outer boundary of an individual [1]. The magnitude of exposure depends on the substance concentration to which the individual is exposed and the duration of the exposure. Exposure may occur continuously or intermittently.

5.1.2 Dose

The EPA guidelines define the *dose* as the amount of substance available for interaction with metabolic processes or with biologically significant receptors after exposure occurs. Substances cross the outer boundary either through *intake* or *uptake* processes. In intake processes, substances cross the boundary without passing an absorption barrier. Examples of intake processes are inhalation of substances contained in the ambient air and ingestion of substances in food or liquid. In uptake processes, substances cross an absorption barrier and are absorbed into the body. An example of an uptake process is the dermal absorption of substances from ambient air, water, or soil.

5.2 Exposure Modeling

Although dose levels within the body will tend to be lower than exposure levels at the body surface (due to differences among substances in solubility and tissue absorption), research is still underway on these particular properties of utility-emitted trace substances. For that reason, this study assumes that internal dose equals external exposure.

Many methods exist for modeling exposure which involve derivation of one or more exposure measures. A common inhalation exposure measure is lifetime average daily dose (LADD). For purpose of this risk assessment, the terms "exposure" and "dose" are used interchangeably. EPA suggests using LADD with linear, non-threshold carcinogenic

dose-response models (e.g., potency slope factor) [1], and with noncarcinogenic risk measures as well. The units of the LADD are milligrams of the substance per kilogram of body weight per day, assumed the average value over a lifetime. To calculate the inhalation LADD, assumptions must be made about its key components: inhalation rate, duration of exposure (or dose), body weight, and lifetime, as well as the dose concentration, assumed equal to the exposure concentration.

The inhalation rate is the rate at which the individual inhales ambient air. The exposure duration is the time period over which exposure occurs (or is being evaluated). Body weight is important because the linearized dose-response relationships for carcinogens (e.g., potency slope factor) assume that the expected chronic health response to a given dose of a substance is inversely proportional to the weight of the individual. The exposure concentration is the average lifetime concentration of the substance in the medium to which the individual is exposed.

The EPRI base case exposure assessment utilized standard EPA assumptions for a maximally exposed individual (MEI), including an inhalation rate of 20 m^3/day, a body weight of 70 kilograms, and a lifetime of 70 years. To obtain exposure concentrations, atmospheric dispersion modeling was used to generate estimates of ground-level concentrations within 50 km of each power plant (Section 5.4) and demographic analyses were employed to determine where population resides in relation to concentrations (Section 5.5). This analysis also extended the standard EPA approach to incorporate key parameters (such as residential and activity patterns) needed for a more "realistic" assessment of lifetime exposure, the reasonably-exposed individual (REI) measure. This was done by assigning values to the key components of exposure, such as inhalation rate, to reflect a more typical community exposure.

5.3 Exposure Measures

Several exposure measures were defined for the inhalation exposure assessment: maximally exposed individual (MEI), the "reasonably" exposed individual (REI), and average population exposure. These are described below:

- The MEI is defined as an individual exposed to outdoor air for 70 years in the populated location with the highest concentrations of trace substances emitted by a power plant.
- The REI is defined as an individual with typical activity and residential moving patterns, who lives in the populated location with the highest concentrations of trace substances emitted by a power plant. The REI computations in the risk assessment (Section 7) reflect the activity patterns of an outdoor worker with all activities near the plant.

- Average population exposure combines MEI exposure assumptions with population-weighted average concentrations due to power plant emissions, yielding a high-end inhalation exposure estimate for an average individual in the population surrounding each plant.

Table 5-1 compares the assumptions contained in these risk measures. The MEI and the average population exposure are similar, and share standard EPA exposure assumptions; these measures are conservative (tend to overestimate values) because they assume inhalation of outdoor air concentrations, at a moderate breathing rate, for 70 years.

Table 5-1.
Assumptions Embodied in Various Exposure Measures

Assumption	MEI	REI	Average Population Exposure
Exposure Time	70 years	Average amount of time living in one area (approx. 19 years)	70 years
Power Plant Replacement	none	After 55 years of operation, unit is replaced with similar unit that meets 1994 NSPS for particulates (0.03 lb/10^6 Btu)	none
Concentrations	The highest outdoor concentration due to plant emissions in a populated area within 50 km	The highest outdoor and indoor concentrations due to plant emissions in a populated area within 50 km (indoor concentrations are approx. 60% of outdoor concentrations for particle-bound material)	The population-weighted outdoor concentration due to plant emissions within 50 km
Time Spent Outdoors	24 hrs/day	Fraction of day based on activity pattern data	24 hrs/day
Breathing Rate	20 m^3/day	Varying by activity; age-weighted	20 m^3/day
Susceptibility to Health Effects	average	average	average

For the average population exposure, the average concentration across receptor locations is weighted by the number of residents associated with each receptor location. These measures incorporate standard EPA exposure assumptions consistent with corresponding EPA dose-response functions. The exposure assessment developed plant-specific concentration estimates corresponding to each measure. Sections 5.4 and 5.5 describe how these concentration estimates were developed.

The REI is fundamentally different from the other exposure measures used, as it does not utilize standard EPA exposure assumptions. Instead, the REI relies on population-wide data on breathing rates, activity levels, commuting patterns, etc. Since it uses these data rather than standard default assumptions, it is not as excessively conservative as the MEI.

Whereas the MEI is a bounding estimate of exposure, the REI is a central estimate of a typical individual's exposure. While EPA recommends use of a different measure (the Reasonable Maximum Exposure, or RME) as an alternative, the use of an MEI and REI are consistent with EPA guidelines on exposure by inhalation.

5.3.1 Reasonably Exposed Individual (REI) Exposure Assessment

The inhalation exposure assessment estimated the inhalation exposure to trace emissions from power plants for a reasonably exposed individual (REI) living near each power plant. The assessment incorporated available data for several key factors that affect REI exposure, including activity patterns, indoor/outdoor concentrations, and population mobility. The assessment also accounted for the replacement of aging units with units that meet New Source Performance Standard (NSPS) for electric utility boilers. The remainder of this section summarizes the methodology for estimating REI exposure. Appendix E provides further detail on the methodology.

Assessing REI exposure requires outdoor and indoor trace substance concentrations to estimate exposure levels for the time an individual spends "near the plant" (defined to be within 50 km). The highest outdoor concentrations in a populated cell for each plant (determined in the demographic analysis discussed below) were adjusted by indoor/outdoor ratios which reflect the extent to which specific trace substances (from outdoor sources) penetrate indoors, yielding the corresponding indoor concentrations. The assessment assumed indoor/outdoor ratios of 65% for particles and reactive gases, and 100% for nonreactive gases (i.e., complete penetration) [8,9,10].

REI exposure depends on the time spent indoors near the plant (i.e., the time an individual spends indoors at home or inside another structure within 50 km of the plant) and the time spent outdoors near the plant (i.e., the time an individual spends outdoors at home or outdoors at another location within 50 km of the plant). The two key time parameters are (1) the average time spent in each location per day of exposure and (2) the number of days of exposure in a lifetime.

The average time spent indoors and outdoors near the plant per day is estimated from information about activity patterns. Human activity patterns specify the time sequence of an individual's movement among various micro-environments (e.g., home, outdoors) and their associated level of physical activity (e.g., light exercise). Such patterns are important determinants of exposure to trace substances emitted from power plants. The location during each activity interval determines the concentration levels of trace substances in the air individuals breathe, while the activity level affects the rate at which the individual breathes in the contaminated air.

To capture differences in behavior patterns across demographic groups, the assessment used activity pattern data compiled by EPRI [11] to evaluate the time spent (and inhalation rates) indoors and outdoors for eight population subgroups:

- non-workers with all activities near the plant
- non-workers with all activities (outside the home) far from the plant
- indoor workers who work near the plant
- indoor workers who work far from the plant
- outdoor workers who work near the plant
- outdoor workers who work far from the plant
- indoor workers who commute into the area near the plant
- outdoor workers who commute into the area near the plant.

Non-workers were defined to be individuals who never report holding a paid position over the 70 year time horizon. Indoor workers were defined to be individuals who have jobs in low-exposure environments for their entire work life. Outdoor workers were defined to be individuals who have jobs in high-exposure environments for their entire work life. The REI computations in the risk assessment estimate exposure for an outdoor worker working near the plant.

The best estimate of number of days exposed in a lifetime is based on the years nearby residents are expected to live near a power plant, considering the likelihood that they will move in and out of that area. Based on data from the 1991 American Housing Survey [12], a Monte Carlo sampling approach was used to simulate the sequential moves of an individual away from and back to the area near the plant. The simulation was repeated 2000 times to provide a distribution of residence time in the vicinity of a power plant. The median value of residence time was 19 years. The median value was assumed to be applicable for a REI near any power plant.

REI exposure also depends on outdoor and indoor inhalation rates. The assessment estimated exercise-level weighted average indoor and outdoor inhalation rates using standard inhalation rates by exercise level and the amount of time spent in different exercise levels.

REI exposure also requires body weight over a lifetime. Body weight is important because linear dose-response relationships accepted by EPA (e.g., potency slopes) assume that the expected chronic health response to a given dose of a substance is inversely proportional to an individual's weight. The standard assumption for adult body weight (independent of sex) in risk assessment is 70 kilograms. Since this assessment considers exposure over a lifetime, it used a best estimate of 62.5 kilograms based on body weight data for different ages specified in EPA's Exposure Factors Handbook [13]. The best estimate is a time-

weighted average of mean values over 70 years for males and females combined. The best estimate is only appropriate for use in analyses which address exposure over an entire lifetime.

The key factors that affect REI exposure were combined to derive best estimates of exposure scaling factors for each substance. Exposure scaling factors are dimensionless adjustment factors that account for the differences between MEI exposure assumptions and more reasonable exposure assumptions. A substance exposure scaling factor multiplied by the corresponding exposure of the MEI yields an estimate of reasonable exposure to that substance for the typical individual living in the area with highest concentrations from the plant.

For REI exposure, the emissions input was adjusted to account for retirement and replacement of older units. It was assumed that units are replaced after 55 years of operation with units that meet or exceed the current NSPS for particulate emissions, 0.03 $lb/10^{12}$ Btu. Operationally, two emissions runs were performed. To project emissions for the years from 2010 to the unit's 55th year in operation, the first run used the projected particulate emissions for 2010. To project emissions for the unit's 56th year in operation to 2080, the second run used the minimum of the NSPS standard for particulates and the projected particulate emissions for 2010. The emissions input to the dispersion modeling is the average emissions from the two runs weighted by the number of years each run is applicable.

5.3.2 Comparison of REI and MEI Exposure

The comparison of REI and MEI exposure provides insight into the impact of using more reasonable assumptions on the likely exposure and dose levels due to power plant emissions. Noteworthy is that the REI assessment did not account for the bioavailability of substances in the body or the respirable fraction of particles, which would further reduce REI exposure and dose levels.

Figure 5-1 presents carcinogenic and noncarcinogenic exposure scaling factors for arsenic by population subgroup. These dimensionless factors are the ratio of REI exposure (reflecting all key factors other than plant replacement) to MEI exposure. Since plant replacement assumptions impact each plant differently, they were not incorporated in the exposure scaling factors. The impact of plant replacement assumptions on exposure is discussed below.

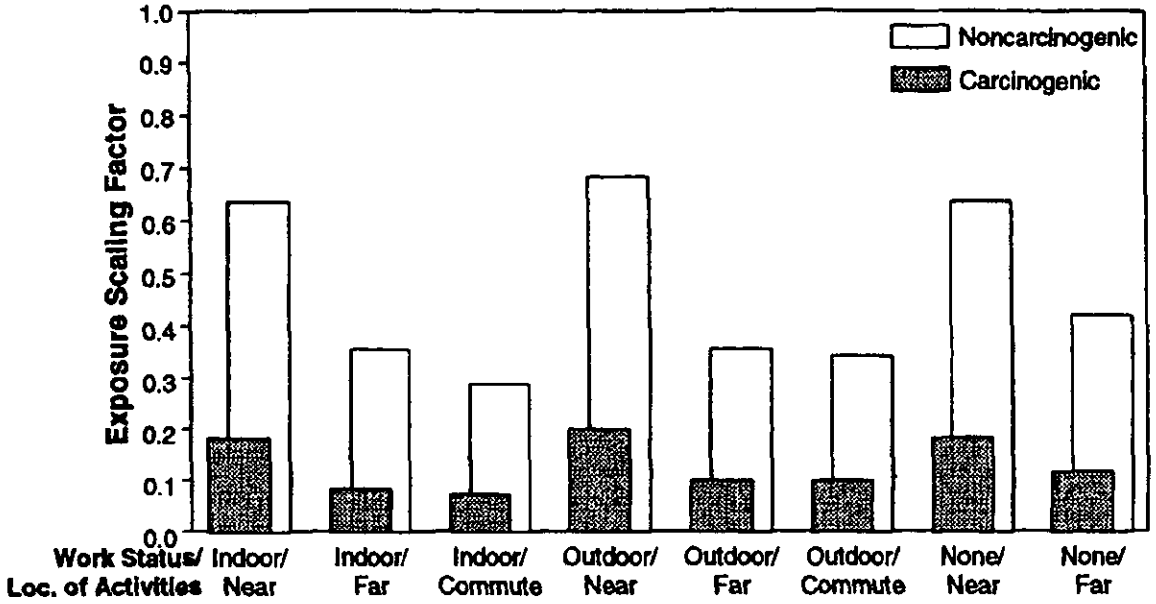

Figure 5-1.
Exposure Scaling Factors for Arsenic

The impact of using reasonable exposure assumptions on REI vs. MEI exposure to noncarcinogenic substances is the same as for carcinogenic exposure, with the exception of residence time. Residence time has no effect on exposure measures to noncarcinogens since the exposure period for chronic, noncarcinogenic effects is assumed to be on the order of years, rather than the decades required for carcinogenic effect at low exposures.

The arsenic carcinogenic exposure scaling factors for each population group are between 0.08 and 0.18, suggesting that, for a given concentration level, analyses based on MEI exposure assumptions overestimate arsenic risks for typical individuals by a factor of about 5 to 12. Exposure scaling factors are relatively high for groups of individuals with all activities near the plant. The group with the highest exposure scaling factor is outdoor workers with all activities near the plant; this factor was used in REI risk computations. Exposure scaling factors are lowest for groups of individuals living far from the plant and commuting to the area near the plant.

The arsenic noncarcinogenic exposure scaling factors for each population group are between 0.29 and 0.66, suggesting that, for a given concentration level, analyses based on MEI exposure assumptions overestimate risks for average individuals by a factor of about 2 to 3.

Since bioavailability and the respirable fraction of particles were not incorporated in the REI assessment, all of the trace substances except non-reactive gases have the same exposure scaling factors as arsenic. The carcinogenic exposure scaling factors for the non-reactive gases, benzene and toluene, are about 20% to 70% larger than those of the other trace substances, and range as high as 0.25.

Figure 5-2 shows how reasonable exposure assumptions account for differences between carcinogenic MEI and REI exposure. Activity patterns, inhalation rates, and indoor/outdoor ratios are considered together as one set of "activity-related" exposure assumptions, as they all contribute to modeling activity patterns. This set of assumptions makes REI exposure overall some 20% to 75% lower than the corresponding MEI exposure, depending on the power plant. The amount of the difference is specific to the population group and the characteristics of the trace substance.

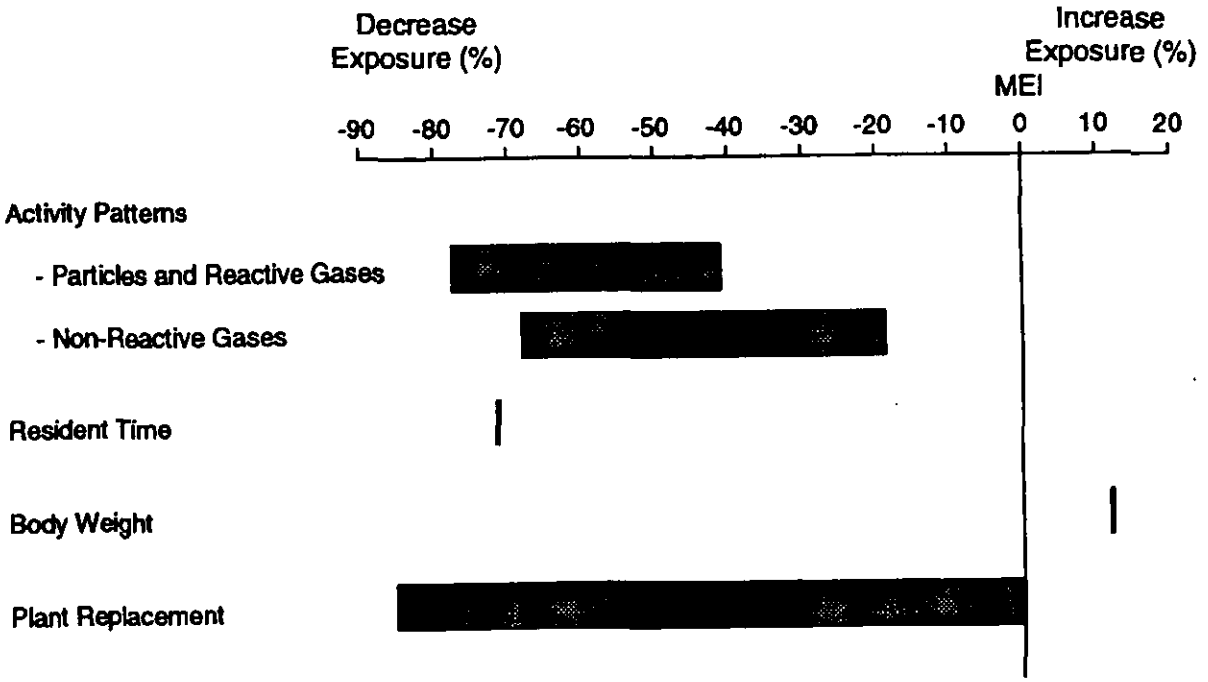

Figure 5-2.
Impact of Assumptions on Exposure

REI exposure to particle-bound trace substances and reactive gases is lower than that of non-reactive gases. This reflects the assumption that the indoor concentrations of particle-bound substances and reactive gases are 60% lower than their corresponding outdoor concentrations, whereas indoor and outdoor concentrations are assumed to be the same for non-reactive gases. The contribution of each plant's trace substance emissions to the REI/MEI ratio thus is a function of the particular proportions of individual classes of trace materials emitted.

Residence-time assumptions contribute about a 75% decrease between MEI and REI exposure for each plant, reflecting a decrease in exposure time from 70 years for the MEI to 19 years for the REI. This residence time assumption reflects an analysis of a study by the American Housing Survey (U.S. Department of Commerce) on the number of years householders live in a given unit. Appendix E discusses this analysis. Body weight assumptions alone would lead to REI exposure being about 10% higher than MEI exposure, reflecting a decrease in body weight from about 70 to 62.5 kilograms when age-weighting is used.

Although plant replacement assumptions are not incorporated in the exposure scaling factors, they too imply lower exposure levels. The plant replacement assumptions lower the particulate emissions of coal-fired units whose projected 2010 particulate emission exceed the current NSPS, reducing the units' average trace substance emissions over the 70-year time period (2010 to 2080) and the subsequent exposure of the REI. On average, these assumptions make REI exposure 14% lower than MEI exposure. Across all plants, these assumptions result in REI exposures up to 85% lower than corresponding MEI exposure, depending on a unit's projected particulate emissions for 2010 and its expected start-up date within a given power plant.

5.4 Atmospheric Plume Modeling

5.4.1 Plume Dispersion Modeling

Atmospheric dispersion modeling was used to generate estimates of ground-level concentrations of the substances emitted to the atmosphere. Venkatram and Seigneur [2] reviewed mathematical models suitable for simulating the atmospheric dispersion of airborne chemicals in the context of public health risk assessment. They recommended the EPA-approved ISC (Industrial Source Complex) model for screening-level analysis [3]. ISC employs an assumption that cross-wind profiles of concentration in the plume are described by a Gaussian (normal) distribution. Several studies have been performed to evaluate the performance of ISC [4,5,6]. The model has two versions, one each for simulation of short-term or long-term concentration patterns respectively. The current long-term version, ISCLT2, estimates annual-average concentrations using joint frequency distributions of atmospheric stability and wind direction and speed. Since the primary focus of the risk assessment is chronic effects due to long-term exposure to chemicals, ISCLT2 was selected for modeling ground-level concentrations due to power plant emissions.

Running ISCLT2 requires assembly of appropriate input data fields. These include source characteristics, meteorological data, and model option settings appropriate to the environment being simulated. Discussion of these inputs follows.

For simulating elevated point sources, ISCLT2 requires source height (in this case, physical stack height) and diameter, and the exhaust gas temperature and velocity. These data are used to calculate fluxes of heat, buoyancy, and mass and to determine effective stack height. The data are available in the UARG stack file, a modified subset of the information in the 1991 UDI database. The 1991 UDI database was derived from operator filings with the U.S. Energy Information Administration Form EIA-767, reporting on plant operations during 1991.

ISCLT2 requires as input a number of different types of local meteorological data, some readily available and others not. The readily available data include STability ARray (STAR) summaries with five or six atmospheric stability categories [7] for selected National Weather Service (NWS) stations. The STAR data are joint frequency distributions of wind speed and direction by stability class, and reflect long-term average meteorological data. The STAR data in the 1983 tabulation for the closest NWS station were assigned to each power plant, although these data may not always be the most representative of the location of the power plant (e.g., in mountainous regions).

For many locations, key meteorological data (e.g., atmospheric boundary layer depth) are not readily available. Table 5-2 presents model default values that were used for all plants (as well as the "regulatory default" values recommended in the ISCLT2 documentation for other environmental variables).

Table 5-2.
ISCLT2 Model Settings

Parameter	Name	Value
Mixing Heights	HM	stability class A: 2100 m stability class B: 1400 m stability class C: 1400 m stability class D: 1163 m stability class E: 450 m stability class F: 0 m
Weather Station Recording Height	ZR	10 m
Wind Direction	THETA	starts at 0°, every 22.5°
Number of Particulate Size Categories	NVS	1
Settling Velocity	VS	1 cm/s
Mass Fraction of Particles	FRQ	1
Surface Reflection Coefficient	GAMMA	1 (complete reflection)

The model also requires that the user specify a number of input parameters or model settings that govern the implementation of model calculation options. To tailor the dispersion runs to power plants, the analysis specified the configuration of the dispersion grid and the use of urban or rural dispersion coefficients. Due to lack of readily available information, the analysis did not specify building dimensions or terrain elevations.

A radial dispersion grid was specified around each power plant. Each grid extends 50 km from a plant with radial sector boundaries at one km intervals and angular boundaries every 22.5 degrees. For each sector, ISCLT2 estimates the concentration at its center. The dispersion grid defines a domain that captures the locations of the maximum concentration for all the plants modeled and covers the majority of populations that may be exposed to the highest concentrations due to plant emissions.

Simulating stack plume dispersion requires a choice of "urban" or "rural" dispersion parameters for each source location. Urban parameters generally result in higher ground-level concentrations near the source than do rural parameters. EPA guidance for running ISCLT2 [6] calls for evaluating either population density or land use within 3 km of the source to determine whether the urban or rural setting is most appropriate. Since national land use data are not readily available, the risk assessment used the population-density approach to assign dispersion coefficients to individual plants. According to EPA guidance, this method characterizes areas with a population density greater than $750/km^2$ within a 3-km radius of the source as urban, and other areas as rural. The analysis used the latitude and longitude of each power plant from the UARG stack file and the 1990 U.S. Census block population data to evaluate the population density within 3 km of each power plant. Rural parameters were thereby assigned to about 80% of the plants.

ISCLT2 also allows the user to specify receptor terrain elevations that are below physical stack height, and to include (or exclude) the effect of downwash due to any nearby large structures. Because of the great number of sources being modeled and the lack of detailed site information on each, the no-downwash and flat-terrain options were selected for all locations. However, stack-tip downwash, which modifies plume height calculations to account for stack wake turbulence, is incorporated in the parameterizations of the "regulatory" option selected.

Using the data inputs and model settings described above, ISCLT2 simulations were performed for all plants. For each plant, ISCLT2 output provided concentration estimates for every receptor in the dispersion grid, the receptor-average concentration, and the maximum concentration due to the plant's emissions. It was assumed that no atmospheric chemical transformations occur.

5.4.2 Overlapping Plume Modeling Analysis

EPRI conducted analyses to gain an understanding of the extent to which overlapping plumes from multiple sources in a densely-populated area have the potential to increase individual exposure in the surrounding population. A screening-level analysis was conducted to examine the extent to which plumes from U.S. electric utility generating plants have the potential to overlap. The screening-level analysis indicated that New York City has the greatest number of plants in a contiguous area.

A detailed analysis was performed for the greater New York City area to determine a likely upper bound on the impact of overlapping plumes of U.S. power plants. The detailed analysis estimated emissions from 28 elevated point sources in the greater New York City area. The sources represent stacks associated with a combination of utility and non-utility generating sources, and additional fossil fuel-fired boilers. The analysis used the ISCLT2 model to estimate the total concentration in New York City due to all of these sources. Although use of this model was consistent with the industrywide study described throughout this report, it was not the most appropriate atmospheric dispersion model for a setting such as the metropolitan New York City area. Because of the complicated boundary layer structure, and the similarity in height between many modeled sources and adjacent structures, other, more sophisticated models would be technically more appropriate for a detailed assessment. The findings below should be viewed in that light.

The analysis results indicated that one source contributed about 75% of the total concentration due to modeled sources in the location with the maximum modeled concentration. Overlapping plumes from other nearby sources (8 within 5 miles) had a relatively minor contribution to the maximum modeled concentration in this same location. Since New York City has the greatest number of plants in a contiguous area and overlapping plumes have relatively minor impacts, it is unlikely that overlapping plumes in other parts of the country have major impacts on individual exposure levels. Based on this analysis, it was assumed that, in any given area, the total concentration to which an individual is exposed can be modeled as the concentration due to emissions from the nearest plant. However, plumes of individual plants were summed in the analysis of total population risks nationally, discussed in Section 7, to account for overlapping plumes.

5.5 Demographics

Demographic data were used to determine where individuals lived in relation to atmospheric concentrations of trace substances generated in the previous section. National population data from the 1990 U.S. Census were mapped to the sectors of the polar grid surrounding individual power plants. These sectors map directly to those used in the dispersion modeling grid, providing information on the number of individuals exposed to various concentration levels.

As with the dispersion modeling grid, the area within 50 km of the plant was modeled as a radial grid with sector boundaries at 1 km intervals from the plant and 22.5 degrees angular resolution. The location of most plants was obtained from the UARG stack data set. For plants without location data in the UARG stack file, the analysis used information from EIA Form EIA-767 and, when necessary, EPA's Aerometric Information Retrieval System (AIRS) database.

For every population grid, the total population in a sector is the sum of the populations with census block centroids in the sector. The analysis matched each plant's population by sector to the corresponding concentration levels from the dispersion runs, providing "population/concentration" information for every plant. The population/concentration information consists of the sector number, the distance from the plant, the orientation with respect to the plant (e.g., NW), the population in the sector, and the estimated concentration per unit emissions for the receptor at the "center" of the sector.

Using the population/concentration information for each plant, the EPRI exposure assessment determined the highest concentration in a populated cell for the MEI and REI calculations. The exposure assessment also computed the population-weighted average concentration for the average population exposure calculation.

5.6 Summary

The inhalation exposure assessment estimated MEI and average population exposure by incorporating standard EPA exposure assumptions. To obtain these measures, the exposure assessment estimated the appropriate annual average concentrations due to plant emissions to which groups of individuals may be exposed. Dispersion simulations were performed using the EPA-developed plume dispersion model, ISCLT2, to calculate ground-level concentration estimates within 50 km of each plant. Demographic data were used to determine where people lived in relation to the concentration estimates.

The inhalation exposure assessment also used a "reasonable" measure of exposure for residents in the populated area with the highest concentrations due to each plant's emissions. This measure, the REI, incorporates relatively fine time and space resolution with respect to human activity patterns, such as extent and duration of exposure to outdoor and indoor concentrations resulting from outdoor levels of substances emitted by power plants. It also assumes that aging units are replaced with units that meet the 1994 New Source Performance Standards (NSPS) for particulates.

5.7 References

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6

HEALTH EFFECTS

6.1 Overview

Assessing risks to health due to emissions from electric power plants involves collecting and interpreting many different kinds of information. As this report indicates, emissions data, whether measured directly or inferred from measurements of fuel and the performance of pollution control equipment, provide a source term estimate. Modeling the transport of emitted materials through the air, including deposition on the ground and into food, provides the basis for calculating exposure point concentrations. These concentrations, coupled with demographic information on the location and activities of people (e.g., on the fraction of time spent indoors) can produce an exposure estimate. Based on the exposure estimate and various toxicological and epidemiological data, health risks can be estimated. Finally, uncertainties can be analyzed and described.

This section addresses the biological information that, when combined with the exposure assessment, can be used to estimate risks to health. For the most part, the risks in this report are based on a screening risk assessment approach. For screening risk assessment, simple models are used, often because the information needed to justify the use of more complex and realistic models is not available. For carcinogens, it is common to use a screening model in which the risk due to any particular chemical is proportional to the emissions of that chemical. Such a model assumes that exposure is directly proportional to emissions and that risk is directly proportional to exposure. For carcinogens, this standard method focuses on assessing a plausible upper bound on risk. For noncarcinogens, risks are calculated and compared with a reference level to establish levels of exposure at which there is high confidence that little chance exists for adverse health effects.

The health assessment methods provided in U.S. EPA guidance are sometimes referred to as default assumptions—that is, assumptions used in the absence of more reliable information. Because the biological impact of every chemical cannot be tested, it is frequently necessary to base a risk estimate for a family of chemicals (e.g., all arsenic compounds) on experience with one form of the chemical. Similarly, extrapolation from high doses to low doses can be based on an understanding of the behavior of the chemical in the body in terms of where the chemical goes, how rapidly it is removed or metabolized, and the toxicity and behavior of the metabolic products. Such information is referred to as pharmacokinetic information. But pharmacokinetic information is often unavailable, so a

standard default method of extrapolating to low dose effects is used. This method is intentionally designed to be conservative—that is, more likely to overestimate risk than to underestimate it.

This section addresses how health effects information is treated in the risk assessment of utility air toxics. As noted, these methods may be conservative, but they are necessary when information is not readily available to support alternative models and assumptions. The focus of the health effects work is on two substances, arsenic and mercury, which are known to be present in some power plant emissions.

As was noted in Section 1, the EPRI research on potentially hazardous emissions from power plants began with 16 substances chosen for study because they were known to be emitted by power plants in measurable concentrations and they may also be potentially harmful to human health. Descriptions of the toxicological profiles and chemical characteristics for each of these 16 compounds are contained in Appendix P.

The focus of the health effects work discussed in this section is on two substances, arsenic and mercury. The reason for choosing these two substances, out of the 16 substances listed in Section 1.4.1 is that, of all the substances emitted from power plants, arsenic appears to be the most significant contributor to potential inhalation cancer risk (based on earlier screening inhalation risk assessments, used to provide guidance to the PISCES field sampling program). A possible exception are radionuclide emissions; these are discussed separately. Mercury was selected for discussion because it was found to be one of the comparatively more significant contributors to non-cancer risk, and because mercury is identified in the Clean Air Act Amendments and in ongoing EPA work as a substance of current interest.

Because these chemicals are important to the assessment of risk from steam-electric power plants, it is appropriate to explore whether information can be developed that gives a more solid scientific basis for the risk assessment than do the default assumptions and methods. Ongoing EPRI research to develop the information needed to support a more technically sound risk assessment for arsenic and mercury is described in Section 6.3.

6.2 Regulatory Guidance for Risk Assessment

The Integrated Risk Information System (IRIS) is a database containing U.S. EPA consensus scientific positions on potential adverse human health effects that may result from chemical exposures. The information in IRIS is EPA's chosen source of toxicity information for risk assessment.

The standard default assumptions and methods for risk assessment are selected to provide a plausible upper bound for risk; this means that these assumptions are likely to overestimate the actual risks that are present. One reason that the default method may be conservative is that there are several important mechanisms that are not included in a

simple linear model. For example, detoxification and repair mechanisms can often handle low doses of particular chemicals, but these mechanisms can be overwhelmed by high doses. Other conservative assumptions are often made under a default approach. For example, in some of the air toxic risk assessment results described here, it is assumed that maximally exposed individuals are exposed for 70 years at an unchanging outdoor location.

6.2.1 Carcinogenic Effects

Cancer risks are assessed based on estimates of exposure and on cancer slope factors or unit risk factors. Cancer slope factors, usually expressed in units of $(mg/kg\text{-}day)^{-1}$, define the lifetime cancer risk from an average dose of 1 mg per day per kilogram of body weight. Cancer slope factors are typically used to assess risks where the exposure is through ingestion. Unit risk factors define the estimated lifetime cancer risk from an inhalation exposure to a concentration of 1 μg per cubic meter of air. These risk factors are derived from the results of chronic animal bioassays or human epidemiology studies. While it is desirable to base slope factors on studies involving exposure by ingestion and unit risk factors on inhalation studies, such information is not always available. It is possible to derive slope factors from unit risk factors, or vice versa, based on the assumption that an average person weighs 70 kg and inhales 20 cubic meters of air per day.

Animal bioassays are usually conducted at doses much higher than those likely to be encountered by humans, in order to detect possible adverse effects in the small test populations used in these studies. Because humans are generally exposed to lower doses than the animals in these studies, data are adjusted using mathematical models. A model is typically fitted to data from animal studies to obtain a dose-response curve. Upper statistical confidence limits for points on the curve are then subjected to various adjustments, including application of an interspecies scaling factor, to derive a cancer potency factor for humans. Dose-response data derived from human epidemiological studies are fitted to dose-time-response curves on an *ad hoc* basis.

Whether based on human or animal data, conservative assumptions are applied so that the models provide plausible estimates of the upper limits on lifetime risk. Due to conservative assumptions used in quantifying estimated risks, the actual risks associated with exposure to a potential carcinogen are not likely to exceed the quantitatively estimated risks, and may be much lower. As noted above, available EPA-derived slope factors for chemicals are presented in IRIS.

In order to estimate the theoretical excess lifetime carcinogenic risk associated with exposure to a chemical, the product of the medium-specific cancer slope factors and the lifetime average daily dose estimated for the exposure pathway of concern is determined. For inhalation exposures, the product of the unit risk factor with the lifetime average air concentration provides the risk estimate. For ingestion, the product of the cancer slope factor and the lifetime average daily dose provides the excess lifetime cancer risk.

6.2.2 Noncarcinogenic Effects

Noncancer effects encompass a wide range of responses, including adverse effects on specific organs or organ systems, effects on reproductive capacity, the viability and structure of developing offspring, and survival. Unlike carcinogens, chemicals posing health risks other than cancer are often considered to have thresholds below which there is no risk. The current practice of estimating risk for non-cancer health effects starts with a "no-observed-adverse-effect-level" (NOAEL) or a "lowest-observed-adverse-effects-level" (LOAEL), which is determined from human or animal studies.

The NOAEL or LOAEL is then divided by one or more numerical uncertainty factors (typically these dimensionless factors are 3 or 10) to produce the desired level of protectiveness. Factors are commonly used to provide for a margin of safety and to account for the uncertainties inherent in the extrapolation from a LOAEL or NOAEL or from laboratory animals to humans. Specific factors are often used to adjust from occupational exposures to healthy workers for 40 hours per week to the general population exposed continuously, to account for the variability in human sensitivity to chemical exposures, or to account for deficiencies in the available data. The objective is to identify a dose level or range that will protect the most sensitive subgroups in the population.

The result of this process is an estimate of a level of exposure that is virtually safe for continuous lifetime exposures. This estimate is termed a reference dose (RfD). Similarly, inhalation reference concentrations (RfC) are defined as the concentration of a chemical in air that is safe. Virtually all of the uncertainty adjustments for noncarcinogenic oral RfDs and inhalation RfCs have incorporated large "safety factors," often of several orders of magnitude, to account for the uncertainties noted above. The RfD or RfC is defined as a level of exposure, surrounded by an uncertainty band of perhaps an order of magnitude, that provides a virtually safe level of lifetime exposure.

Adverse noncarcinogenic effects are evaluated by comparing the estimated daily intake of a chemical to its associated RfC or RfD. Doses less than the RfC or RfD are not likely to be associated with any adverse health effects and are, therefore, not of regulatory concern. However, doses that exceed the RfC or RfD are considered to present the potential for adverse health effects. The relationship is expressed numerically using a parameter known as the Hazard Quotient (HQ). The HQ is obtained by dividing the average daily dose by the RfC or RfD. The dose calculation for each chemical will have a distinct HQ. For mixtures of chemicals, such as are found in power plant air emissions, the HQs for the various components are summed to yield a hazard index (HI). An HI value of less than one indicates that an adverse effect would not be anticipated. Conversely, an HI equal to or greater than one indicates that there is a potential for a noncarcinogenic health effect to occur as a result of the chemicals released.

The use of HQs and HIs for the evaluation of exposures to hazardous materials is recommended or required in many EPA guidance documents. Perhaps the most relevant of these documents requiring use of these measures for this evaluation of power plant emissions is EPA's *Methodology for Assessing Health Risks Associated with Indirect Exposures to Combustor Emissions.*

6.3 Substances Selected for Research Focus

This section briefly describes results from ongoing EPRI research on arsenic and mercury. Appendix G provides greater technical detail on studies underway on these trace elements. The purpose of this research is to provide data to improve the basic understanding of the chemical state of these compounds and their interactions with biological systems in order to more accurately assess potential risks from possible exposure to these materials that may occur as a result of power plant operations.

As mentioned earlier in this section, Section 1.4.1 describes EPRI's rationale for focusing general research on 16 substances most relevant to utility emissions. In brief, the 16 substances listed in Section 1.4.1 were chosen for study since they are known to be emitted by power plants in measurable concentrations and they may also be potentially harmful to human health. Descriptions of the toxicological profiles and chemical characteristics for each of these 16 compounds are contained in Appendix P.

As described in detail in Section 7 of this report, EPRI conducted an inhalation risk assessment to estimate potential health risk due to inhalation of trace substances emitted from electric utility steam-generating units. In this risk assessment framework, health effects were characterized by using dose-response information tabulated by EPA and other sources. For carcinogenic effects, the current U.S. EPA unit inhalation risk factors were used, with one exception. The exception is arsenic, for which recent alternative findings were used; the EPA IRIS number is treated as a sensitivity study. For non-carcinogenic effects, the analysis used inhalation reference concentrations which are based on EPA reference doses, defined as being levels of daily exposure that are likely to be without appreciable chronic deleterious effects. In general, the dose-response information used was from EPA's Integrated Risk Information System (IRIS) database.

Using assumptions in the risk assessment framework as described in Section 7, arsenic was shown to consistently contribute the majority of inhalation carcinogenic risk among the 16 trace substances chosen for study and emitted from both coal- and oil-fired power plants. Therefore, EPRI's major health effects research effort has focused principally on this compound.

Health concerns for mercury are based on potential neurotoxic effects related to ingestion of methylmercury as described in Appendices N and O. The current Reference Dose used by EPA for methylmercury is derived from human high acute exposure data that are not likely to be relevant to the situation of low chronic exposure that may be associated with

power plant emissions. Recently-undertaken health research that is relevant to a low-level chronic exposure situation for methylmercury is described in Section 6.3.3 and Appendix G.

6.3.1 Arsenic

The current U.S. EPA unit risk factor for arsenic as a carcinogen by the inhalation route is 4.29×10^{-3} $(\mu g/m^3)^{-1}$ and is based on the assumption that all chemical forms of arsenic are equally carcinogenic. This means that the lifetime cancer risk associated with inhalation of air containing arsenic at a concentration of 1 $\mu g/m^3$ is estimated by EPA to be 4.29×10^{-3}. This factor is based on findings of significantly increased risk for lung cancer among workers at two United States smelters, the Anaconda Copper Smelter in Montana and the ASARCO Copper Smelter in Tacoma, Washington.

In addition to the reassessment of exposures and risks to smelter workers discussed below, EPRI research is underway to address other factors relevant to the assessment of arsenic risks from power plant emissions. A central objective of these studies is to determine how applicable the results from studies in copper smelters are to the estimation of community risks from arsenic in fly ash. At least three major default assumptions that were used to derive the current EPA inhalation unit risk factor for arsenic are addressed in new EPRI research: (1) relevance of valence state of arsenic in copper smelter dust compared with that present in fly ash, (2) comparative bioavailability of arsenic from copper smelter dust compared with fly ash given chemical composition and matrix differences between the two complex mixtures, and (3) extrapolation of high occupational exposure to copper smelter dust to the situation of low community exposure to arsenic in fly ash. More detailed methodology and results from EPRI research underway to address these three assumptions are contained in Appendix G.

6.3.1.1 Proposed Revision of the IRIS Inhalation Unit Risk for Arsenic

Recently, the original authors of the Tacoma smelter study have published updated exposure/dosimetry estimates for that study indicating that the workers were much more highly exposed than had been previously thought when EPA completed its risk assessment in 1984 [1]. In addition, results from a recent Swedish smelter study have been described in the literature [2]. The Swedish study is of workers at the Ronnskar smelter, who were employed for more than three months during the period from 1928 to 1967 and who were followed through 1981. The resulting cohort of 3916 workers has extensive industrial hygiene and medical surveillance data available.

In current EPRI work, standard EPA risk assessment methodology has been used to recalculate estimated risk using the newly revised exposure estimates from the Tacoma smelter study. Based on these data, the unit risk estimate is calculated as 1.28×10^{-3}. In addition, a unit risk of 0.89×10^{-3} was derived, again using standard EPA methodology,

from recent findings in the Swedish cohort. Pooling these two new unit risk estimates with EPA's estimate from Montana smelter worker data yields a composite new unit risk of 1.43×10^{-3} which is one-third of the value of 4.29×10^{-3} that appears in the IRIS database as of mid-1994. Based on this work, EPRI formally requested a change in the IRIS database for the inhalation unit risk for arsenic, and has used a unit risk factor of 1.43×10^{-3} in the analyses reported here. A manuscript describing this work has been accepted for publication in *Regulatory Toxicology and Pharmacology* [3]. (The full text of the accepted manuscript is included in this report as Appendix H).

6.3.1.2 Arsenic Research

Results to date from EPRI research to address the three principal areas of uncertainty listed in Section 6.3.1 and are briefly described here. More technical detail on this research can be found in Appendix G.

6.3.1.2.1 Arsenic Speciation in Coal Fly Ash

As indicated in Section 3 (Section 3.5.1), arsenic may volatilize from fuels in the combustion process, but subsequently does re-condense on fly ash particles at normal stack gas temperatures of about 300° F. Volatile forms of the compound thus are not emitted from the stack, therefore the concentration and characterization of compounds as present in emitted particulate fly ash are the important parameters to study with regard to potential human exposure and health effects.

The default assumption currently used for arsenic cancer risk assessment for exposure by the inhalation route assumes that arsenic in any valence state or chemical form is equally carcinogenic. As discussed in Appendix G (Section G.2), this is unlikely to be so. Therefore, EPRI has conducted research on the valence state(s) and chemical form(s) of arsenic in fly ash as well as the implications of these differences in chemical state for other relevant toxicological endpoints. Current results from this work are described in the following sections.

Results from chemical speciation studies performed on coal fly ash using at least four different analytical approaches indicate that arsenic is present in coal fly ash in the arsenic (V) valence state. Copper smelter dust is thought to contain principally arsenic (III) as arsenic trioxide. It is known from a body of published work that arsenic (III) is approximately 10 times as acutely toxic as is arsenic (V).

Calcium arsenate (arsenic (V)) has been identified as the major arsenic compound in coal fly ash. This compound has been found consistently whether the coal fly ash sample originates in bottom ash, in hopper ash, in samples collected from high in the stack, in particles collected isokinetically on filters in the stack, or on filters from high volume ambient air samplers. Solubility studies further confirm these findings.

6.3.1.2.2 Lung Retention and Urinary Excretion Kinetics of Arsenic.

Results from an animal study conducted to assess lung retention and excretion kinetics of arsenic using the respiratory route of exposure suggest that lung retention of arsenic is slightly higher for copper smelter dust compared with coal ash, and that arsenic that is bioavailable (absorbed) from copper smelter dust seems to be excreted in urine at a slightly slower rate than arsenic that is bioavailable from coal ash. This implies that the lung residence time for arsenic from copper smelter dust may be slightly longer and that absorbed arsenic from this dust may not be methylated (detoxified) quite as rapidly as that from coal fly ash.

For both copper smelter dust and coal fly ash, arsenic clearance from the lung in current experiments can be described as a typical two-phase nonlinear process, in which the majority of arsenic is cleared from the lung in an early rapid phase, followed by a much longer phase during which the remaining relatively low concentration is cleared. It is currently unclear whether this relationship holds under conditions of relatively low particulate exposure as occurs in ambient community air.

Studies of soluble arsenic compounds (sodium arsenite (III) and sodium arsenate (V)) show that virtually no arsenic is retained in the lung at one day after exposure and that sodium arsenate is more efficiently excreted immediately after exposure than is sodium arsenite. These results indicate that soluble arsenic compounds of either valence state are readily bioavailable.

More recent results comparing the behavior of pure calcium arsenate with two coal fly ashes–an ESP sample containing about 2000 mg/kg arsenic and a pulse jet fabric filter (PJFF) ash sample containing 200 mg/kg arsenic shows that two days after treatment, lung retention is higher and urinary excretion rate is lower for the PJFF fly ash than for either of the other two particulates. Due to the differing arsenic concentrations in the ash samples, ten times more total particulate material from the PJFF sample had to be administered than for the ESP sample in order obtain equal treatment doses of arsenic. The number and mass of particles in the lung may affect arsenic lung clearance rate such that arsenic in some fly ash samples is retained in the lung slightly longer than arsenic from pure calcium arsenate when there is higher particle lung loading with fly ash. Studies are now underway to assess the relative importance of particle lung loading to arsenic lung clearance rates.

Other important results from these studies suggest that coal hopper ash produces less lung inflammatory reaction than does copper smelter dust. This is of potentially great importance since inflammation is thought to be an important step in progression in the development of cancer.

Studies are underway to clarify the possible effects of lung particle load on arsenic bioavailability from coal fly ash; studies are also planned to succinctly describe the nature and magnitude of the lung inflammatory response for copper smelter dust compared with coal fly ash.

6.3.1.2.3 Occupational Exposure to Arsenic in Coal Fly Ash.

A study to assess arsenic bioavailability in humans during occupational exposure to coal fly ash during a planned outage is underway. The power plant is located in the Slovak Republic in an area where lignite coal of very high arsenic content is used as the principal fuel. Mean daily breathing zone air concentrations of arsenic during outage operations were documented over a wide range; in addition, community air concentrations of arsenic were determined both inside and outside residences in three nearby villages. Daily urine samples were collected from workers and community referents. Preliminary results show that detectable amounts of arsenic and its two major metabolites are present in urine of workers. This study is designed to describe exposure, bioavailability and excretion kinetics of arsenic from coal fly ash over a wide range of exposures; results are to be applied in the validation of the arsenic PBPK model. Results may also indicate whether bioavailability and exposure are linearly related.

6.3.1.2.4 Detoxification of Arsenic in Humans: Evidence for Non-linearity.

A population study was undertaken in two villages in Mexico in order to assess bioavailability and methylating capacity under conditions of chronic exposure to arsenic over a wide range of chronic exposure that occurs due to ingestion of water containing arsenic. The mean arsenic concentration in well water from the "high" village ranged from 0.375 to 0.392 mg/L whereas that in the "low" village ranged from 0.019 to 0.026 mg/L. (The current drinking water standard in the United States is 0.05 mg/L; therefore these village levels represent values both below and considerably above this standard and allow assessment of effects over a wide range of exposures that bracket the current standard). A total of sixty persons (thirty from each village) were enrolled in the study; participants were matched on age and sex. Urinary concentrations of inorganic arsenic, and the two major metabolites, monomethyl arsenic acid (MMA) and dimethyl arsonic acid (DMA) were determined.

An issue of ongoing interest is whether methylation (detoxification) remains linear over a wide range of arsenic exposure concentrations or whether at high exposures, efficiency of the methylation steps decreases, resulting in a nonlinear process. Results from urinary analyses show that for the most highly exposed village significantly less DMA (55.5% vs. 80.6%) and more MMA (12.1% vs. 7.8%) was excreted in urine than in the low exposure village controlling for other factors such as age and sex (see Appendix G, Section G.3.3 and Figure G-4).

Although there are possible alternative hypotheses, results suggest that the second methylation step is impaired in the most highly exposed individuals. Similar results have been shown very recently in a pilot study of a small group of individuals chronically exposed to arsenic in drinking water in the same area [4]. Additionally, a similar pattern of excretion was seen in a recent animal study in which acute oral exposure to sodium arsenate was studied [6]. Taken as a whole, these results imply that non-linearity in the detoxification process may occur at both high acute and chronic exposures. These findings have important implications for risk assessment, since the default assumption for cancer risk assessment is that a linear relationship exists between exposure and response at all exposure concentrations.

6.3.1.2.5 Pharmacokinetic Model for Arsenic.

An operational multi-compartment physiologically based pharmacokinetic (PBPK) model has been developed for arsenic [5]. Studies are underway to further validate and refine the model and then to apply it to various relevant exposure scenarios. In this way, the effect on uptake and bioavailability of varying parameters such as valence state, chemical compound, matrix, particle size and particulate lung loading among others can be studied. In addition, effects on bioavailability and kinetics of metabolism and excretion can be compared between conditions of high occupational exposure and low community ambient exposure.

6.3.2 Mercury

Almost all concern about potential health effects of mercury in the environment centers on methylmercury exposure. People are exposed to methylmercury when they eat mercury-contaminated fish. Methylmercury bioaccumulates in fish muscle tissue, is eliminated very slowly, and can cause neurological problems in adults who consume the fish, and psychomotor retardation in children exposed before birth.

Since mercury is ubiquitous in the environment and enters the food chain in the form of methylmercury, those seeking to protect public health must determine a safe chronic level, or Reference Dose (RfD), of methylmercury ingestion. The current RfD for methylmercury is 0.3 μg/kg (body weight)-day and is based on published studies of methylmercury poisoning in Iraq where more than 100 people suffered temporary or permanent nervous system damage when they ate bread baked with flour mistakenly milled from seed grain treated with a methylmercury fungicide [3, 1]. It is important to note that the RfD is set at an exposure level designed to protect the "most susceptible individual," likely to be the unborn fetus exposed during a critical period in brain development [17].

Because the present RfD has been calculated using data from the Iraqi poisoning episode, it is based on short-term exposure to high doses of methylmercury. Thus, the present RfD for methylmercury may be very conservative, and an RfD is needed for people exposed at low doses over longer periods of timea scenario more typical of possible exposure related to utility emissions.

Population studies relating maternal dose during pregnancy to children's responses on a variety of neurological and performance tests have been completed in Iraq [11, 12, 13], Canada [19, 14], and New Zealand [15, 16]. Researchers have used information from these studies to assess the health risks of methylmercury employing the health risk assessment estimates derived from statistical modeling. To model health risk, researchers must describe dose-response functions rigorously to relate levels of exposure to changes in health resulting from neurotoxicity.

This task is difficult because characterization of the neurotoxicity of methylmercury is complex. There are different endpoints reflecting multiple biological effects, such as changes in sensory or motor function or cognition. Some effects, such as paresthesia, increase in severity with increasing dose, while others occur only at higher doses and may reflect damage at a different neurological site or by a different mechanism. Moreover, adults may recover from clinical symptoms when exposure ceases. Finally, as we have seen, the fetus is very sensitive to methylmercury if exposure occurs during critical stages of neurogenesis. Thus, any dose-response model used must accommodate the complexity of methylmercury neurotoxicity. Long-term developmental studies being conducted to address these problems are described in more detail in Appendix G and in Appendix O.

As a basis for better understanding the effects that chronic low doses of methylmercury can have on the central nervous system in children, EPRI has developed a physiologically based pharmacokinetic (PBPK) model to describe the fate of methylmercury in the body. Using information from this model, in conjunction with new statistical procedures, EPRI researchers are reanalyzing available epidemiology data on populations that consume fish, to improve dose-response estimates, especially for assessing risks borne by children of exposed mothers. For example, EPRI's Benchmark Dose analysis [4] suggests that the best estimate of the NOAEL for the most sensitive indicator of developmental effects (a grammar understanding test) in six-year-old New Zealand children exposed to methylmercury *in utero* occurs at approximately 17 ppm mercury in maternal hair. At that NOAEL, analysis using the PBPK model indicates that fetal brain tissue concentrations of methylmercury are on the order of 50 ppb (μg/L). According to the model, this concentration in fetal brain tissue would result from a maternal dietary intake of methylmercury ranging from 0.8 to 2.5 μg/kg (body weight)-day. This broad range of intakes corresponding to a target maternal hair concentration of 17 ppm mercury reflects the high degree of variability among hair-to-intake ratios seen in human studies. These results suggest that the current USEPA RfD for methylmercury of 0.3 μg/kg (body weight)-day adequately protects against developmental effects [8].

EPRI's preliminary statistical modeling research using the Iraqi data set [5] indicates that the results from threshold models are very model-dependent and there is a large range for the 95% confidence interval of the threshold. Thus, the Benchmark Dose method is probably a better model to use. EPRI's analysis also reveals inconsistencies in the Iraqi data that make other data sets based on long-term exposure at low dose a more suitable basis for setting standards for prolonged human exposure to methylmercury.

PBPK modeling and statistical investigations are described in more detail in Appendix G and Appendix O. Ultimately, EPRI will use these new findings in a national health risk assessment for methylmercury.

6.4 Summary

- Using standard EPA analysis methods, and published revision by the original investigators of exposure estimates for copper smelter workers, a revised inhalation unit risk for arsenic of 1.43×10^{-3} was calculated. This information was submitted to the IRIS database administrators (see Appendix G, Section G.1 and G.2).
- Results from arsenic research to date show that coal fly ash contains exclusively pentavalent arsenic (V) and that the major compound present in coal fly ash is most likely calcium arsenate (Appendix G, Sections G.2.1 through G.2.3). This is in contrast to copper smelter dust in which trivalent arsenic (III) is the likely predominant species—present principally as arsenic trioxide—and which was used to establish the IRIS inhalation unit risk for cancer. For an in-depth discussion of the rationale and importance of these and other findings, refer to Appendix G1, Section G.2.
- Arsenic from copper smelter dust is somewhat more highly retained in the lung and excreted more slowly in urine than is arsenic from coal fly ash (see Appendix G, Section G.3). The lower solubility of arsenic trioxide compared to calcium arsenate may partially account for the above observation.
- Copper smelter dust may produce more inflammation in the lung than does coal fly ash; this finding has potentially important health effect implications since inflammation is thought to be an important step in progression to cancer.
- Results from two observational studies of a population chronically exposed to arsenic via ingestion of drinking water imply that non-linearity in the detoxification process (methylation) may occur at high chronic exposure concentrations (see Appendix G, Section G.3.3 and Fig. G-4). These results and others imply that use of a linear model to extrapolate cancer risk from very high exposures to very low exposures as is currently done for arsenic cancer risk assessment is probably not appropriate.
- Results to date from an occupational study in Slovakia (see Appendix G, Section G.3.2) indicate that exposure has been documented over a wide range of arsenic concentrations. These data are likely to be very important for high-exposure to low-exposure extrapolations for arsenic in coal fly ash and to further validate the arsenic PBPK model with information relevant to coal fly ash (see Appendix G, Sections G.4).

- A physiologically-based pharmacokinetic model of arsenic has been developed and is being validated (see Appendix G, Section G.4.1). This model is critical for extrapolation of high exposure scenarios (as might occur in an occupational setting) to lower exposure scenarios (as might occur in a community setting).
- The primary environmental health concerns for mercury are those associated with methylmercury exposures to pregnant women and young children. The concerns are based on observations of methylmercury levels in the environment near those that have been linked with biological responses. The main route of exposure to methylmercury is through the food chain. Exposure to other forms of mercury is not significant in the ambient environment.
- A physiologically based pharmacokinetic (PBPK) model has been developed, validated, and used to estimate fetal dose of methylmercury given measurements of the chemical in pregnant mothers' hair. The model provides a critical link between maternal intake and fetal dose (see Appendix G, Section G.3).
- The existing RfD for methylmercury is based on information from an acute poisoning episode in Iraq. This data set may not be suitable for assessing possible health risks from utility emissions because (see also Appendix G, Section G.3 and Appendix O, Section 5): (1) it represents short-term exposure to high doses of methylmercury rather than longer-term exposure to low doses, (2) its health endpoints are based on subjective recall rather than on objective measurements, and (3) alternative statistical methods for analyzing the data set may be more appropriate than the method used to set the current RfD.
- Analyses of more appropriate data sets will be available shortly. These include EPRI's re-analysis of the New Zealand data set, and long-term developmental assessments of children in the Seychelles Islands and the Faroe Islands (see Appendix G, Section G.3 and Appendix O, Section 5).

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7

INHALATION RISK ASSESSMENT

To assess the potential human health impacts due to inhalation of trace substance emitted from power plants, EPRI conducted a risk assessment that integrates emissions estimates, transport and dispersion modeling results, and exposure analyses with standard potency information. For key substances emitted, the risk assessment estimated both carcinogenic and noncarcinogenic risks associated with inhalation exposure over a 70-year time frame, based on projections of the future industry generation mix for the year 2010. EPRI performed a set of deterministic risk analyses for each plant, and additional scenario analyses and sensitivity analyses to examine the impact of varying parameters and alternative modeling assumptions on the risk estimates.

This section focuses on potential risks due to exposure to trace substances by inhalation. Multimedia risk assessment methodologies appropriate for mercury and other chemicals, and methods for radionuclide risk assessment are described in Section 8.

7.1 Measures of Risk

The purpose of the inhalation risk assessment is to estimate potential risks due to inhalation of trace substances emitted from the stacks of electric utility steam-generating units. The risk measures of primary importance correspond to the exposure measures defined in Section 5.3: maximally exposed individual (MEI), the *reasonably exposed individual* (REI), and the average population exposure. The types of carcinogenic and noncarcinogenic risks are described below.

- Individual lifetime risk is the increase in likelihood that an occurrence of cancer will take place as a result of exposure to air concentrations due to the sources studied, over a 70 year human lifetime. Individual lifetime risks are used to compute risks for the MEI and REI.
- Annual population risk is the expected number of excess cancer occurrences (per year) in a given population exposed to average concentrations due to a given source. It is the lifetime risk corresponding to assuming the entire population around a power plant is maximally-exposed to average concentrations, multiplied by the number of individuals living within 50 km of a power plant, and then divided by 70 years.
- The *hazard index* uses the definition provided by EPA [3]; it is the sum, across substances, of the calculated *hazard quotient* [3] for each substance. The hazard quotient is the concentration for each substance divided by the appropriate concentration of concern, usually the federally-defined reference concentration [4]. The hazard quotient re-

quires cautious interpretation. It indicates the proximity to noncarcinogenic criteria limits, or the extent to which these limits are exceeded. As the quotient approaches unity, concern for occurrence of a potential hazard increases. Hazard quotients, and their sum across all substances, the hazard indexes, are used for noncarcinogenic assessment of risk to the MEI and REI.

The MEI scenario overestimates actual exposure because it combines a number of extremely conservative exposure assumptions. EPA exposure guidelines [5] stipulate that the MEI, as a bounding estimate, lies outside the range of actual exposures that might be experienced by any individual, hence both MEI and REI exposure assumptions have been adopted in this study.

7.2 Inhalation Risk Assessment Framework

This section describes the conceptual framework that EPRI has developed for examining inhalation risks due to trace substance emissions from the national capacity of power plants. Appendix I provides details on the actual computations. The conceptual framework addresses impacts to the MEI, the REI, and the population.

7.2.1 Emissions Assessment

This step estimates trace substance emissions from all U.S. electric utility steam-generating units in 2010. As discussed in Section 4, the assessment of industry-wide emissions involves (1) projections of power plant control technologies, fuel usage, and electricity generation in 2010, (2) characterizations of trace element concentrations in utility fuels, and (3) statistical correlations for estimating trace substance emission rates for various plant characteristics and operating conditions. The statistical correlations are derived from the PISCES and other field measurement data. The result is a set of trace substance emissions for every power plant in the United States with units greater than 25 megawatts nameplate capacity as estimated for the year 2010.

7.2.2 Dispersion Modeling

As discussed in Section 5, long-term ground-level atmospheric concentrations within 50 km of each plant were modeled for each plant in the study. This domain captures the calculated maximum concentrations of all plants and covers the majority of populations likely to be exposed to emissions from one or more plants. The ISCLT2 model was used with the "regulatory default" options. The analysis incorporated source characteristics of individual stacks and meteorological data representative of the region around each plant. Each plant was modeled using either "urban" or "rural" dispersion coefficient options, as appropriate for the population density around each plant. Because of the large number of

sources, other default modeling options were used, such as assuming flat terrain and no plume downwash due to nearby structures. The dispersion modeling generated concentration estimates for trace substances within a 50-km radial grid around each plant.

7.2.3 Inhalation Exposure Assessment

Also discussed in Section 5, inhalation exposure is based on estimated atmospheric concentrations. A key part of the inhalation exposure assessment is determining where people reside with respect to estimated concentrations due to power plant emissions. For each plant, the assessment mapped 1990 census data onto the 50-km radial domain used in the dispersion modeling, calculated population-averaged concentrations, and identified both the overall maximum concentration and the maximum concentration in a *populated* grid cell.

The inhalation exposure assessment also developed a measure of exposure, the REI, that is intended to be more realistic than the MEI. As with the MEI, the REI scenario focuses on an individual living at the point of maximum concentration around a power plant. In this case, however, activity data, breathing rates, and indoor/outdoor concentration ratios were used to refine the conservative assumptions incorporated in the MEI exposure measure.

7.2.4 Health Effect Characterization

As discussed in Section 6, this step uses dose-response information tabulated by EPA as well as by other sources to estimate the potential health effects resulting from exposure to trace substances. Table I-1 (Appendix I) presents the dose-response information used in the risk assessment and its sources. For carcinogenic effects, the analysis used inhalation unit risk factors. For noncarcinogenic effects, the analysis used inhalation reference concentrations, which are based on EPA reference doses (RfD). If no value was listed in the EPA IRIS and HEAST sources, other sources of information (e.g., the Threshold Limit Values established by the American Conference of Government Industrial Hygienists) were used. The RfD values are defined as being levels of daily exposure that are likely to be without appreciable chronic deleterious effects. In general, the dose-response information is from the EPA's Integrated Risk Information System (IRIS) database. For arsenic carcinogenicity, the analysis used the revised unit risk factor based on recent EPRI research instead of the IRIS unit risk factor.

The International Toxicity Equivalent Factors adopted by U.S. EPA [2] were used for calculating risks from the dioxin/furan congeners. For PAHs, carcinogenic factors relative to benzo(a)pyrene were taken from Krewald, et al. [1]

7.2.5 Inhalation Risk Assessment

This step builds on and integrates the other elements described above to estimate inhalation cancer risks and hazard quotients for each chemical and plant. For each plant, the cancer risks are summed across carcinogens. (This summation is used as a default assumption, in the absence of strong data about synergistic or antagonistic effects among the chemicals studied.) As a screening method, the inhalation hazard quotients were summed across noncarcinogens for each plant, yielding screening-level inhalation hazard indexes. Although screening-level hazard indexes reflect the sums of hazard quotients for different health endpoints, screening-level values sufficiently less than 1 indicate that adverse effects due to inhalation exposure would not be anticipated [1].

Sensitivity analyses were carried out to identify the modeling parameters that most influence the risk estimates, reflecting the sensitivity of the risk estimates to changes in a parameter and the level of uncertainty in the parameter.

7.3 Carcinogenic Risk Results

This section presents the carcinogenic inhalation risk results for the base case. The base case risk results reflect the base case 2010 operations discussed in Section 4.1 and the emissions estimation procedure as discussed in Section 4.3. Data were available to compute risks for 594 plants.

7.3.1 Population Incidence

The total annual population inhalation carcinogenic risk for the population exposed to emissions from the 594 plants in the base case scenario is 0.08 excess cancer occurrences per year. According to the 1990 census, the total population of the United States is about 249 million. In addition to the 594 plants and their base case capacity of $18{,}950 \times 10^{12}$ Btu, the base case scenario projects $2{,}600 \times 10^{12}$ Btu of unidentified capacity (by state, fuel, and control configuration) and 50×10^{12} Btu of announced capacity with insufficient data for risk calculations, for a total of $21{,}600 \times 10^{12}$ Btu. Assuming that the unidentified capacity and announced capacity with insufficient data have population risks equal to the average population risk for similar plants (based on heat input per unit), the total annual population inhalation carcinogenic risk for the projected 2010 capacity is 0.09. Across the 594 defined plants, the mean annual population risk is 0.0001 per year per plant. An analysis of the 30 plants with the highest annual population carcinogenic risks showed that about three-quarters of these plants have at least 1 coal-fired unit, and all but 4 of these plants have more than one million people residing within 50 km. Of these 30 plants, 12 are also among the highest 30 plants for individual (MEI) carcinogenic inhalation risk. Details of this analysis can be found in Appendix I.

7.3.2 Individual Inhalation Carcinogenic Risks

Figure 7-1 shows a cumulative distribution of inhalation carcinogenic risk for the MEI and the REI. For 34% of the plants, the MEI risk occurs in a location that does not have the highest concentration. For these plants, the highest concentrations occur in locations with no surveyed resident population (1990 Census data). The MEI risk for those plants would increase only slightly (about 4% on average across all plants) if individuals were to live in locations with the highest concentrations that are currently not inhabited. (Note that some of these uninhabited locations are over water bodies or otherwise uninhabitable.) The REI risk is for an individual living in the populated area with the highest concentrations from the plant, and working outdoors in that area. The REI reflects "reasonable" exposure and plant replacement assumptions. The REI risks range from 2% to 19% of the corresponding MEI inhalation risks.

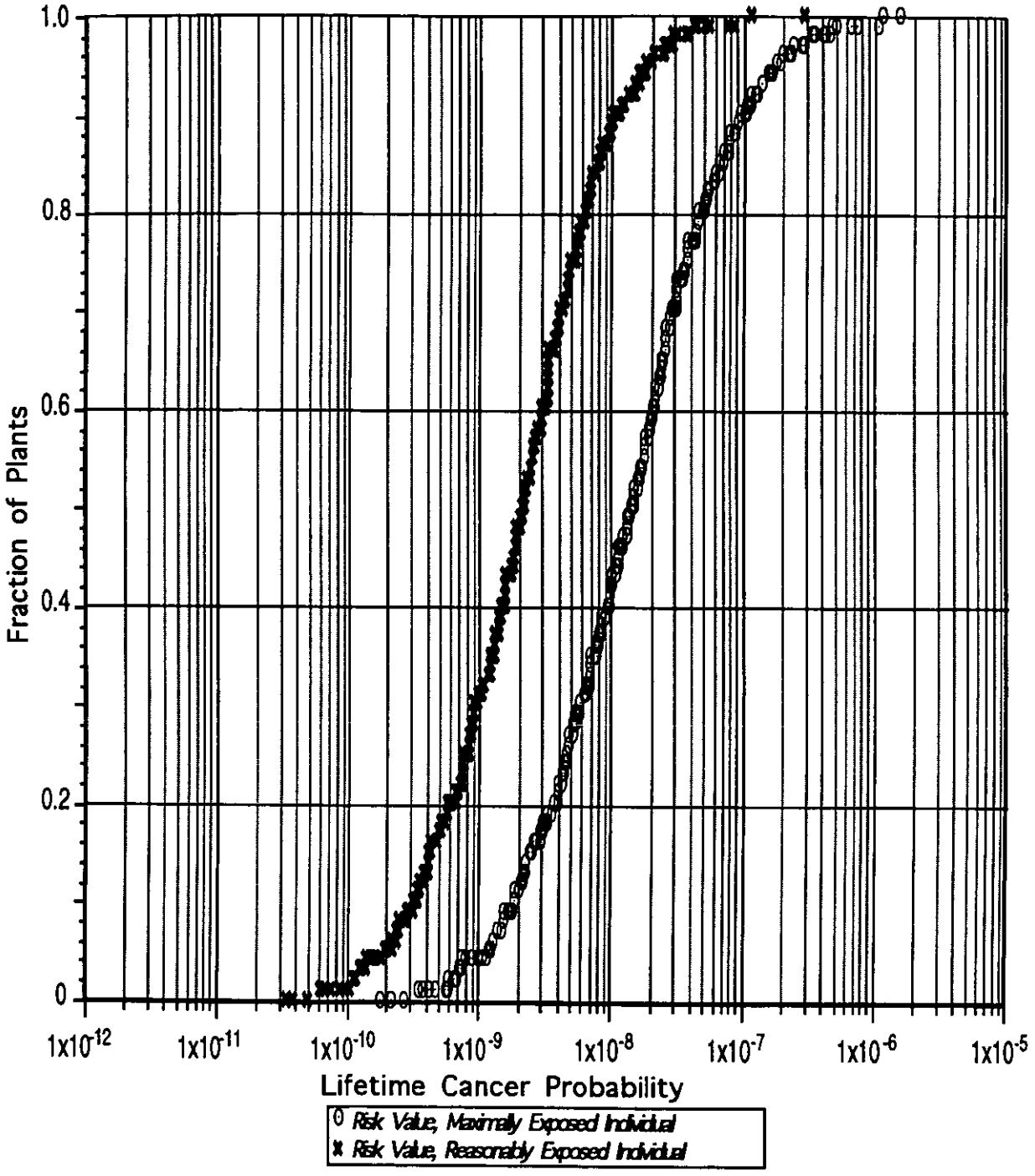


Figure 7-1.
Distribution of Inhalation Carcinogenic Risks, MEI & REI

Figure 7-2 displays the MEI and REI risks by the number of plants in each risk decile. The highest MEI risk across all plants is 1.7×10^{-6}. Of the 594 plants, only three have MEI inhalation risks greater than 10^{-6}, while 62 (10%) have MEI inhalation risks between 10^{-6} and

10^{-7}. None of the REI risks are greater than 10^{-6}, only two (0.3%) of the plants have REI risks greater than 10^{-7}(with the highest REI value at 3×10^{-7}), and the remainder of the plants have risks less than 10^{-7}.

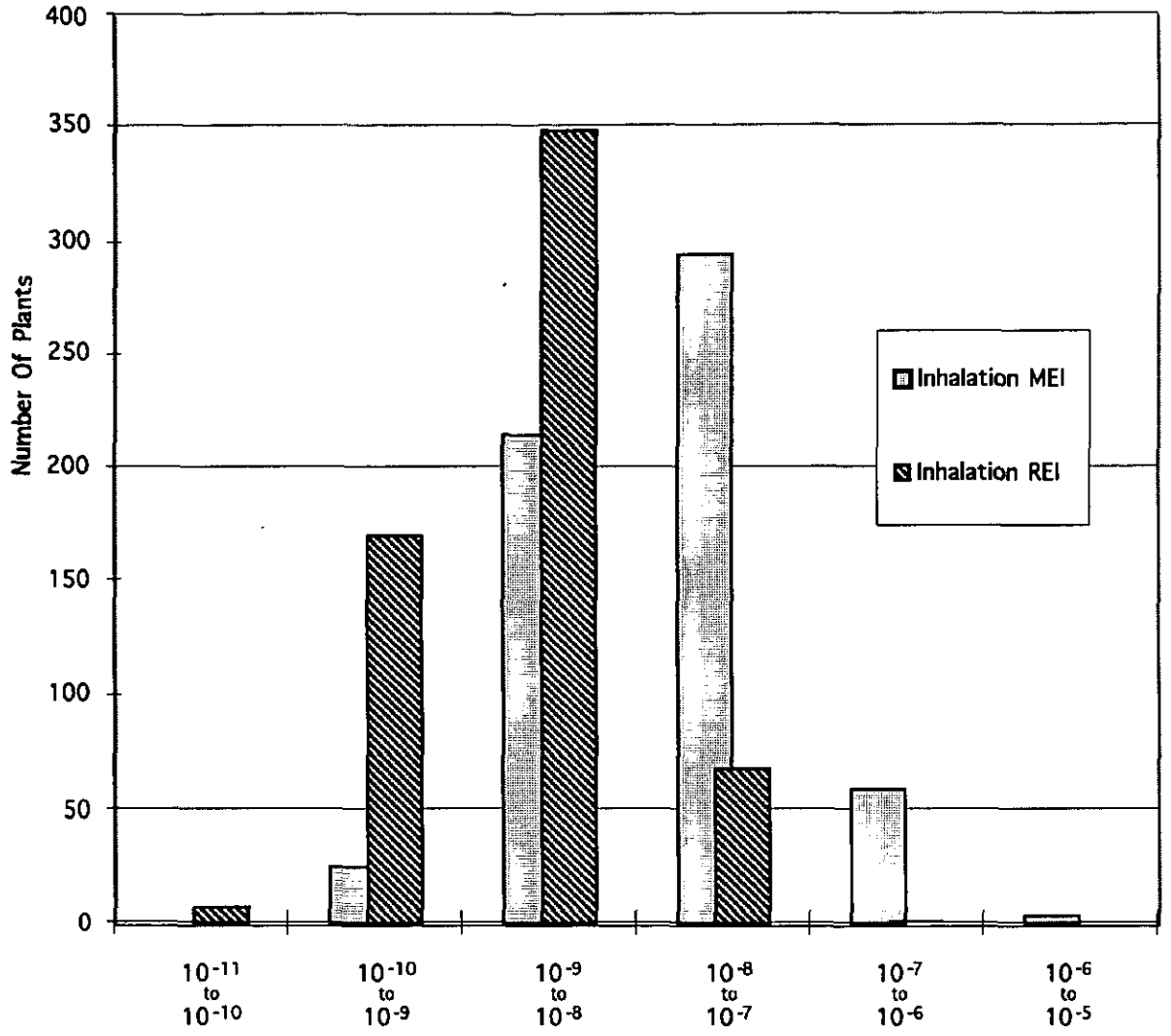


Figure 7-2.
Histograms of Inhalation Carcinogenic Risk

An analysis of the 30 plants with the highest MEI inhalation carcinogenic risks shows that these plants tend to have poor dispersion characteristics compared to the plants with the highest population risk. Details of this analysis are in Appendix I. Figure 7-3 presents the median and range of MEI inhalation risks by plant group. Plant groups are based on type of fuel and control equipment, and are identical to those developed for the emissions correlations (Section 3). The highest MEI inhalation risks are for individual uncontrolled oil plants and for coal-fired plants with particulate controls only. Coal plants with particulate controls only and coal plants with particulate and SO_2 controls have the highest median

MEI risks as groups, at about 3×10^{-8} and 2×10^{-8}, respectively (even though these median values are quite low nonetheless). Gas-only plants have the lowest median MEI risks at about 4×10^{-9}. Since most of the unidentified capacity and the announced capacity with insufficient data is coal-fired with particulate and SO_2 controls, the plants that ultimately make up this capacity are likely to have extremely low risk levels.

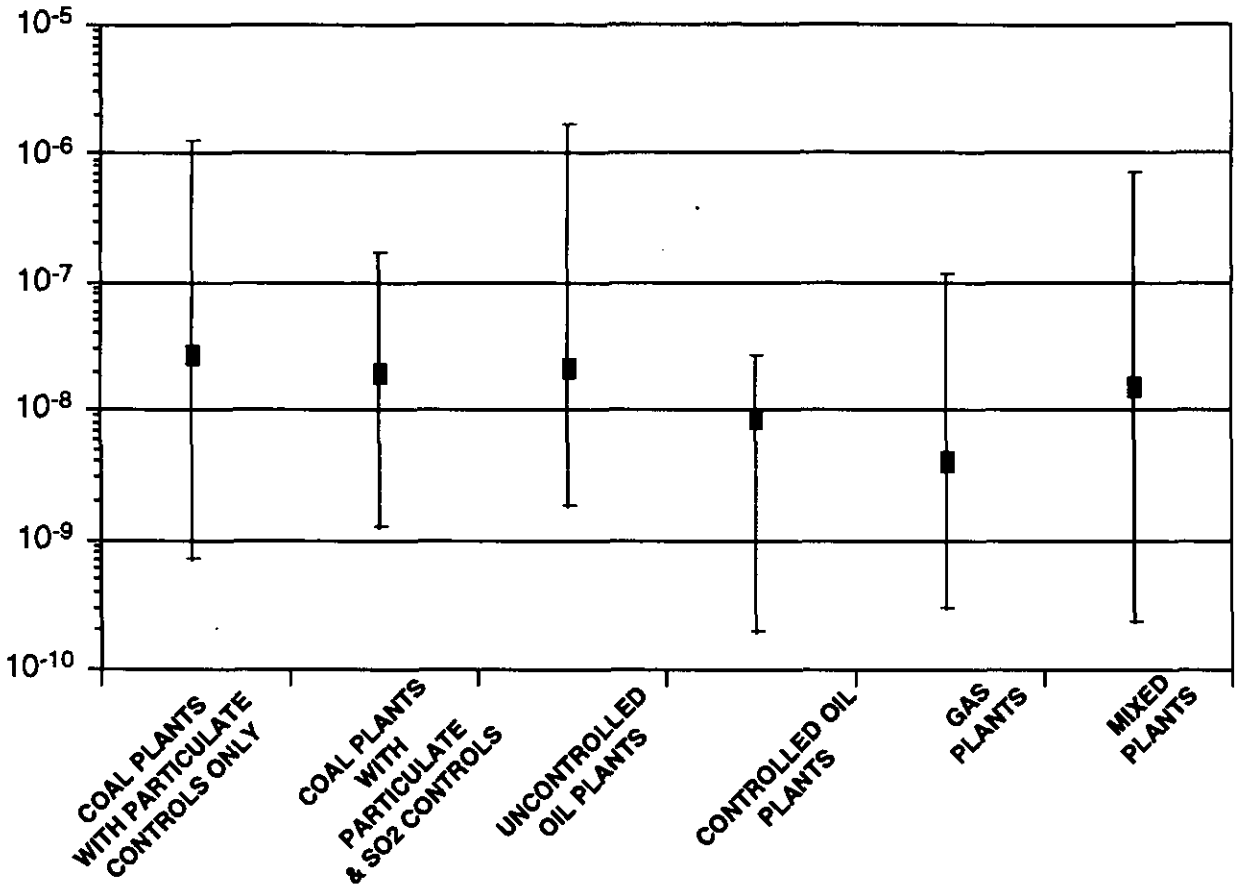
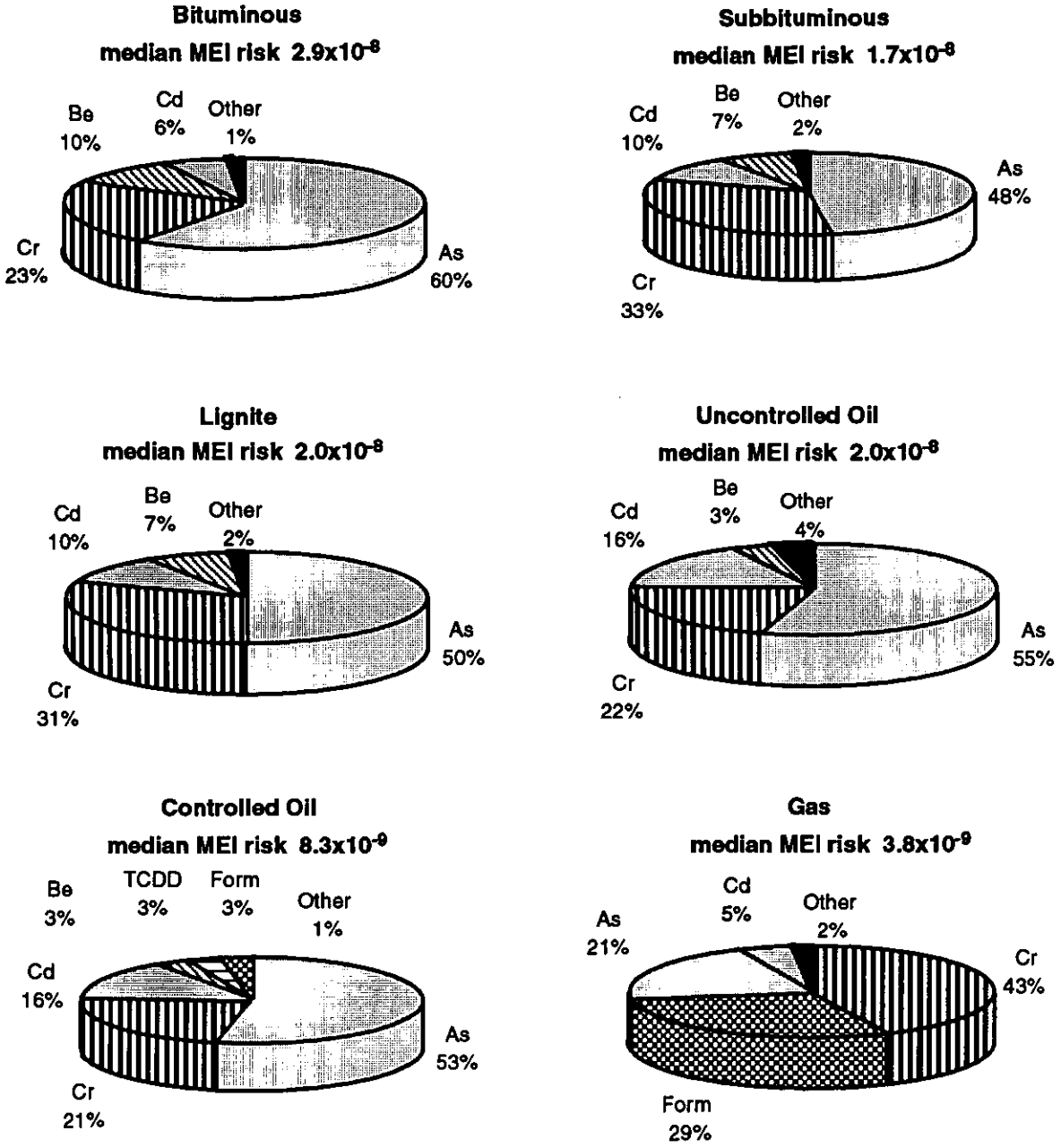


Figure 7-3.
MEI Inhalation Carcinogenic Risk, by Plant Group

Figure 7-4 shows the contribution to MEI inhalation risk by chemical for each fuel. For plants fueled by the three grades of coal categorized, arsenic is the largest contributor to MEI risk with contributions ranging from 48% to 60% of risks. The next highest contributor to MEI risks for coal-fired plants was hexavalent chromium, making up 23% to 33% of the median MEI risk. For controlled and uncontrolled oil, arsenic and chromium contributed about 55% and 20% of the MEI risk, respectively. Median MEI risks for controlled and uncontrolled oil plants are 8.3×10^{-9} and 2×10^{-8}, respectively. For gas-only plants, for which the median MEI risk is 3.8×10^{-9}, chromium and formaldehyde contributed 43% and 29% of the MEI risk, respectively.



- mixed-fuel units and plants are excluded here, since emissions by fuel vary widely from plant to plant

Figure 7-4.
Contributions of Individual Substances to MEI Inhalation Carcinogenic Risk, Median Plant by Fuel Type*

7.4 Noncarcinogenic Risk Results

The section contains the noncarcinogenic inhalation risk results for all trace substances in the analysis. As with the carcinogenic results, the noncarcinogenic results reflect base case assumptions and methods, including 2010 operations and the emissions estimation procedure.

7.4.1 Hazard Indexes

There are no plants for which downwind concentrations exceed the respective reference concentration for any single substance, and no plant has a screening-level inhalation hazard index greater than or equal to 1. Figure 7-5 shows the distributions of inhalation hazard indexes across all plants. The highest MEI hazard index (0.5). The plant with the highest MEI hazard index also exhibits the highest hazard quotient for a single substance, hydrochloric acid, with an inhalation hazard quotient of 0.31. The second highest hazard quotient for a single substance occurs at the plant with the highest chromium hazard quotient (0.30). The maximum hazard quotient across all plants for each remaining substance is significantly lower than these two values; none of the remaining hazard quotients exceeds 0.035. The REI hazard indexes across all plants range from 21% to 70% of the corresponding MEI inhalation hazard indexes.

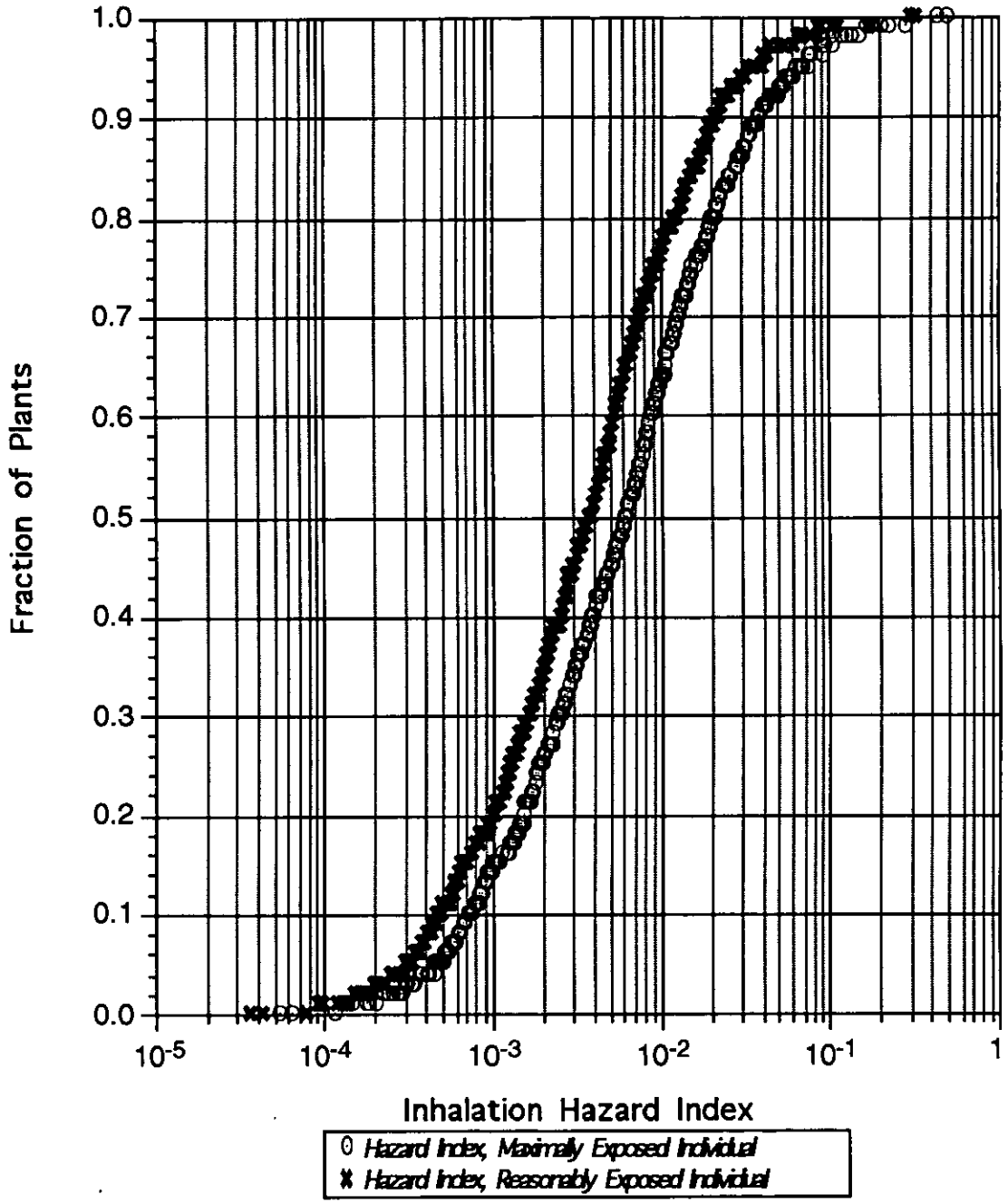


Figure 7-5.
Distributions of Inhalation Hazard Index

Figure 7-6 aggregates the hazard indexes into a histogram of the number of plants at each hazard index decile. Fewer than 2% of the plants have MEI hazard indexes greater than 0.1, and about 32% have MEI hazard indexes between 0.01 and 0.1. Fewer than 1% of the plants have REI hazard indexes greater than 0.1, and about 19% have REI hazard indexes between 0.01 and 0.1.

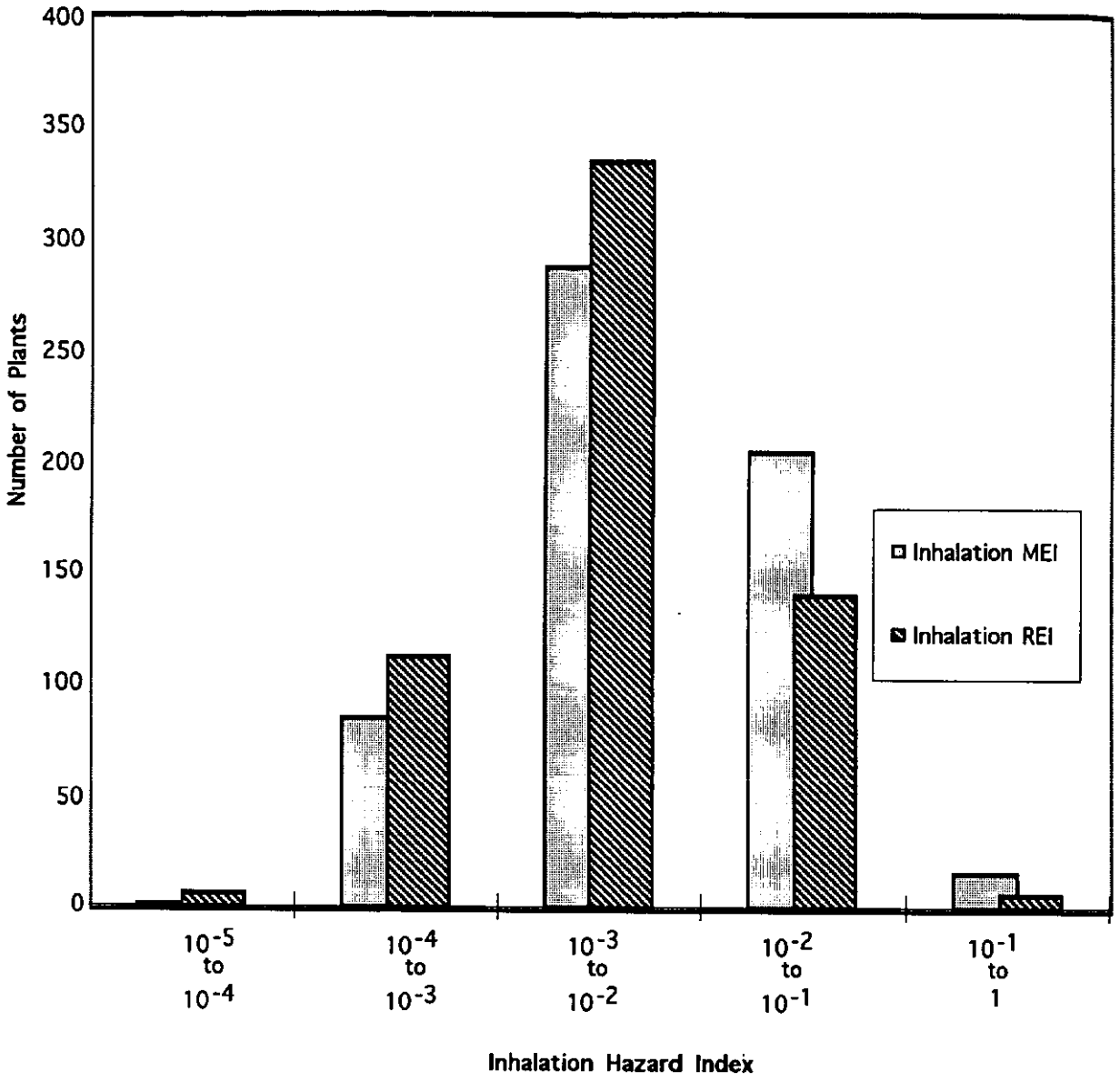


Figure 7-6.
Histograms of Inhalation Hazard Index

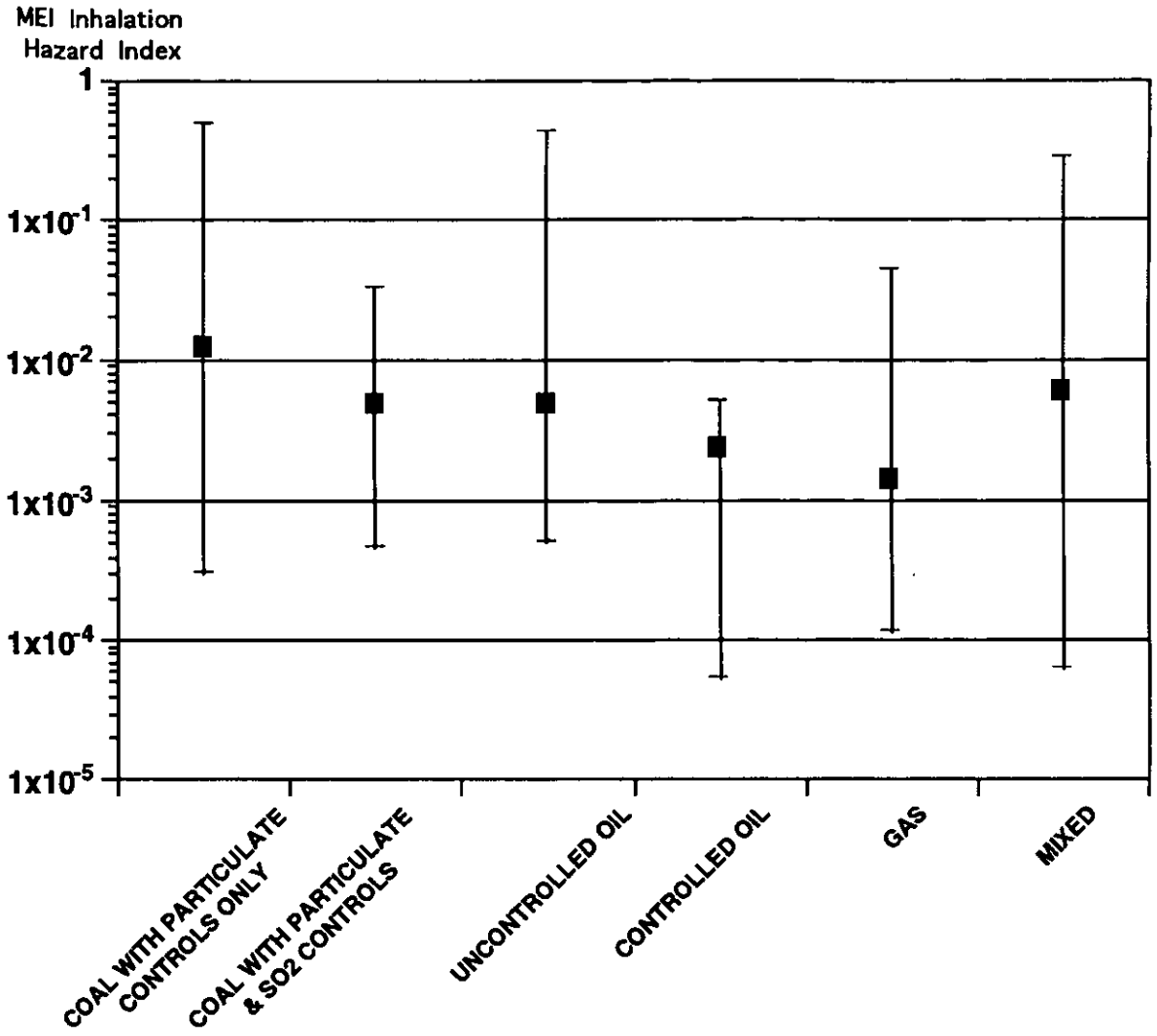


Figure 7-7.
MEI Inhalation Hazard Index by Plant Group

Figure 7-7 shows how the MEI hazard indexes range among different groups of plants. Coal plants with particulate controls only exhibit the highest MEI hazard indexes of all plant groups, followed by uncontrolled oil-fired plants with mixed units. Controlled oil-fired plants and plants with mixed units exhibit the lowest MEI hazard indexes. Coal plants with particulate controls only have the highest median MEI hazard index. The median hazard indexes for other plant groups range from 12% to 47% of the median hazard index for coal plants with particulate controls only.

Figure 7-8 shows the contribution to MEI inhalation hazard index by chemical for various plant groups. The median MEI hazard indexes for the plant groups range from 1.5×10^{-3} for gas plants to 1.3×10^{-2} for bituminous-fired coal plants. Total chromium contributes

from 43% to 90% of the MEI inhalation hazard index for all plant types. Hydrochloric acid (HCl) accounts for 45% of the hazard index from bituminous coal-fired plants. The median HCl emissions rate of the bituminous coal-fired plants exceeds that of each of the other plant groups by a factor of 20 or more. For the other plant groups, HCl accounts for no more than 14% of the hazard index. All other substances have contributions of less than 15%.

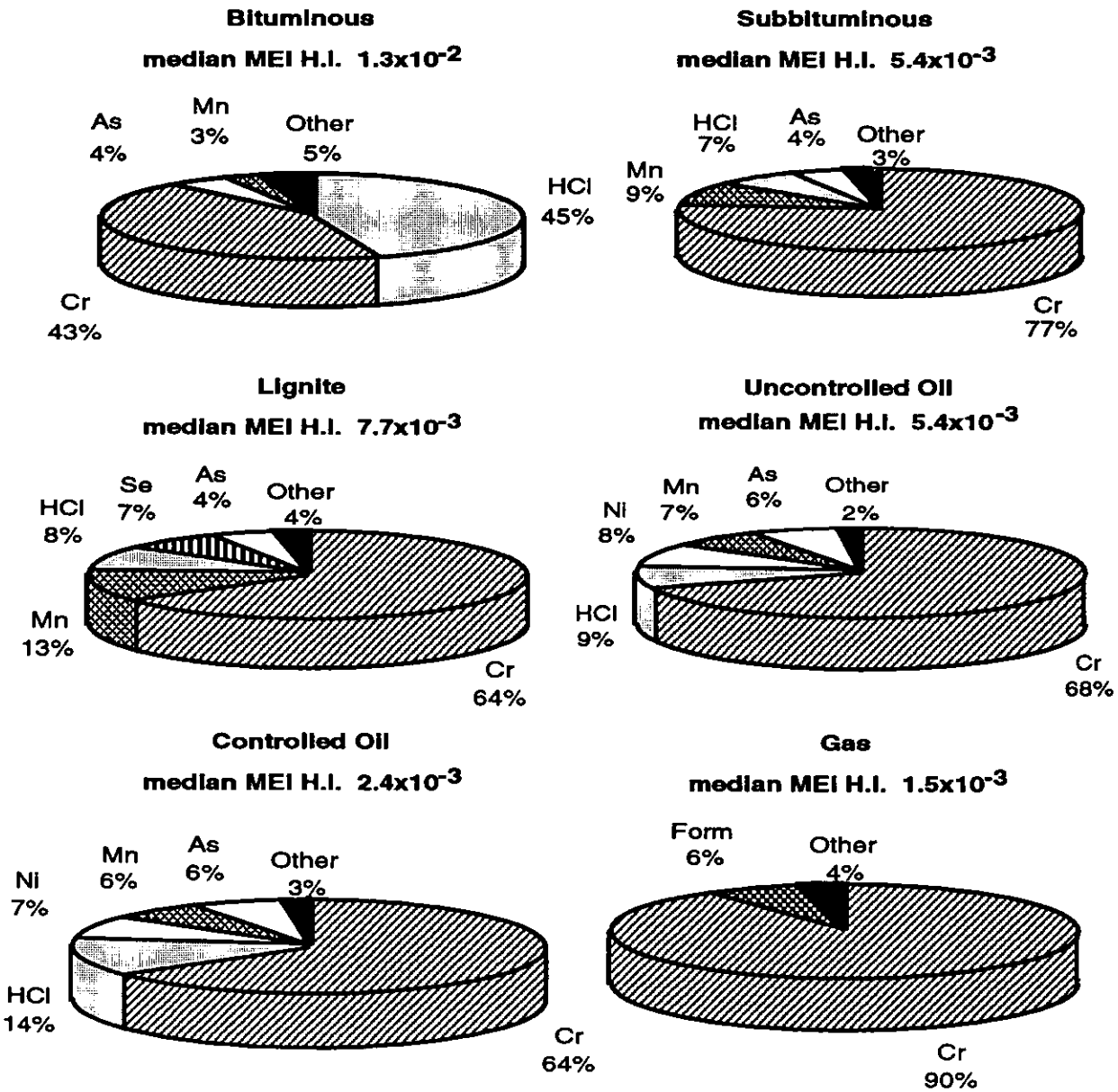


Figure 7-8.
Contributions of Individual Substances to MEI Inhalation Hazard Index

7.5 Assessment Scenario Analysis

Alternative assessment scenarios (groups of assumptions used in the risk assessment) were evaluated to gain insight into the risk implications of alternative modeling assumptions and additional projections of the future utility industry. These assessments of alternative scenarios addressed carcinogenic inhalation risks only.

7.5.1 Alternative Modeling Scenarios

Two scenarios involving emissions and health effects are used to represent alternative hypotheses from the base case risk modeling assumptions. These are:

- *Arsenic.* In mid-1994, the value listed by EPA for the carcinogenic unit risk factor for arsenic by inhalation is 4.3×10^{-3} $(\mu g/m^3)^{-1}$. As discussed in Section 6, EPRI has developed an alternative unit risk factor for arsenic of 1.43×10^{-3} $(\mu g/m^3)^{-1}$. Following EPA protocols, EPRI updated the unit risk factor to reflect revisions to the exposure estimates by the original researchers. The base case scenario discussed above used the updated unit risk factor for arsenic. This "arsenic scenario" examined the implications of using the EPA unit risk factor for arsenic.
- *Particulate emission limits.* This scenario examined the risk implications of having all electric utility steam generating units meet EPA's Subpart *Da* New Source Performance Standard (NSPS) for electric utility boilers. The 1994 NSPS limit for particulate emissions is 0.03 lb/10^6 Btu. The scenario sets the annual particulate emissions rate (for 2010 to 2080) to the minimum of the projected 2010 emissions or 0.03 lb/10^6 Btu.

7.5.2 Alternative Future Scenarios

Two additional scenarios were employed to provide a reasonable range of estimates for sensitivity of risk results to assumptions about industry operations in the future. These two scenarios represent alternative future industry operations, reflecting different SO_2 compliance modeling methods and assumptions than those for the base case. These scenarios are described below and in Appendix C.

- *High trend.* This analysis examined an alternative scenario for the year 2010 that reflects similar coal generation and considerably higher residual oil generation. This scenario also reflects relatively slow announced retirements and replacement of fossil units.
- *Government trend.* This analysis examined an alternative scenario for the year 2010 that reflects lower coal generation and higher residual oil generation than the base case scenario. It also includes considerable unannounced retirements and replacement of fossil-fired units.

As mentioned in Section 4.1, all the future industry scenarios (i.e., "high trend" and "government trend"), including the base case, have several common characteristics that have significant implications with respect to health risk estimates. These characteristics and their implications for health risk estimates include:

- The scenarios project changes in SO_2 control technology and fuels. By 2010, many plants are projected to retrofit FGD scrubbers or switch to subbituminous coals in response to the SO_2 provisions of CAAA. The field measurements indicate that FGD scrubbers, on average, reduce the emissions of particulate-bound trace substances by 75%. They also reduce the emissions of volatile inorganic substances (e.g., mercury, HCl). EPRI research on coal indicates that, at the median, switching to or blending subbituminous coals will reduce the amount of trace substances entering and forming in the boiler per unit heat input.
- The scenarios reflect planned modifications to particulate control technology. About 125 coal-fired units plan (for completion by 2010) or have recently completed modifications to their particulate control equipment. Such modifications will likely reduce the level of particulate emissions, which will lead to reductions in the emissions of particulate-bound inorganic substances.
- The scenarios project retirements by 2010. By 2010, many existing units will be more than 50 years old, which may be approaching the economic lifetime of a unit. The older units tend to have higher particulate emissions, and therefore higher risks per unit generation. Some of the these units are projected to be retired by 2010.
- The scenarios do not project retirements (or replacement) after 2010.

7.5.3 Results of Scenario Analyses

Table 7-1 compares the carcinogenic risk results of the alternative scenarios to those of the base case scenario. The results in this table (including carcinogenic population incidence) reflect the existing units and planned units with sufficient data for risk calculations in each scenario. The arsenic scenario has higher population risk and higher risks for individual plants than the other scenarios. The particulate emission limit scenario leads to slightly lower risk estimates for individual plants than the base case scenario, as shown in Table 7-1, as well as slightly lower aggregate population risks.

Table 7-1.
Impact of Alternative Assessment Scenarios on MEI Inhalation Carcinogenic Risk

Scenario	No. of Plants	Carcinogenic Population Incidence	No. of Plants with MEI Risk Exceeding 10^{-6}	No. of Plants with MEI Risk Exceeding 10^{-7}	Highest Carcinogenic MEI Inhalation Risk	Median Carcinogenic MEI Inhalation Risk
Base Case	594	0.08	3	62	1.7×10^{-6}	1.5×10^{-8}
Alternative Arsenic Use of EPA IRIS unit risk factor	594	0.17	11	130	3.5×10^{-6}	3.1×10^{-8}
Particulate Emissions All sources are at NSPS particulate standard	594	0.04	1	27	1.7×10^{-6}	1.2×10^{-8}
High Trend Alternative 2010 Industry Scenario	532	0.06	0	49	9.1×10^{-7}	1.8×10^{-8}
Government Trend Alternative 2010 Industry Scenario	485	0.04	0	25	5.6×10^{-7}	1.2×10^{-8}

Because the alternative future scenarios do not address a number of the oil and gas plants, Figure 7-9 compares the inhalation REI risks of coal-fired plants for the base case and the alternative scenarios. As represented in Figure 7-9, both the government trend and the high trend scenarios tend to have lower risk estimates for individual plants than the base case scenario. Though a specific plant's risk estimate for the government trend or high trend may exceed its risk estimate for the base case scenario, the interpretation of this figure reveals that the fraction of plants exceeding any inhalation REI risk level that is chosen will be greater for the base case scenario than for either of the other industry scenarios.

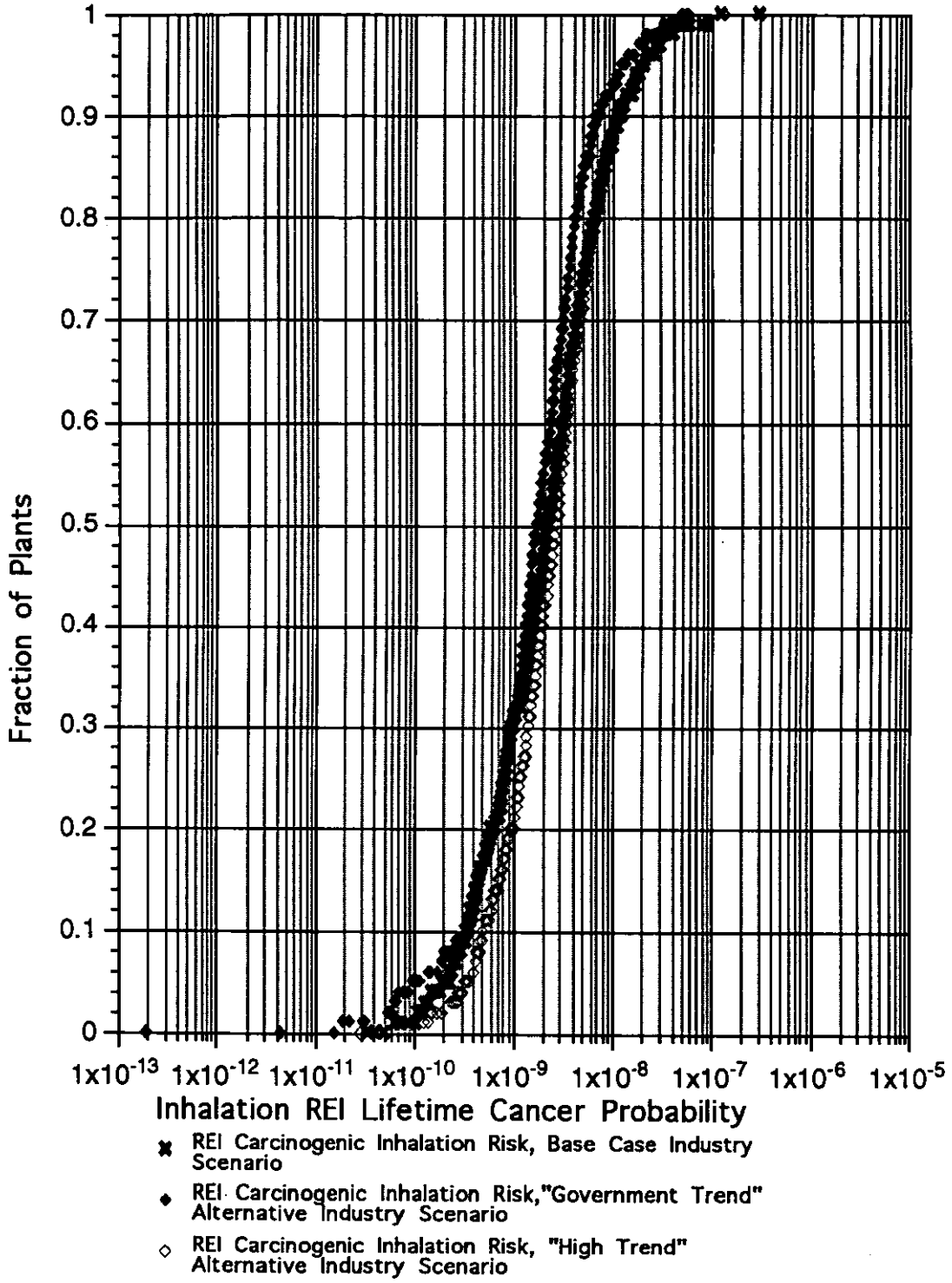


Figure 7-9.
Distributions of Inhalation REI Carcinogenic Risk for Base Case and Alternative Future Industry Scenarios

7.6 Sensitivity Analysis

Sensitivity analysis was used to examine the implications of uncertainties in key parameters on cancer risk and hazard index estimates. By identifying the range of uncertainty in each parameter and assessing the sensitivity of cancer risk and hazard index to that parameter, sensitivity analysis was used to estimate the implications of uncertain data on cancer risk and hazard index estimates. The estimated 95% upper and lower confidence intervals served as the range of uncertainty. Emission-related parameters were the primary parameters considered in the sensitivity analysis. Other parameters, such as exposure parameters and unit risk factors, were considered in the REI analysis and scenario analyses (described above). The emission-related parameters fall into categories based on individual substances, type of fuel fired, and type of control technology at the plant, since the emission correlations (Section 3) vary along these dimensions. The sensitivity analysis included uncertainties in the following parameters.

- *Coal-fired plants (all types of controls).* Content of trace substances in coals, correlation parameters for nonvolatile inorganic substances in coals (which are used to estimate emissions of these substances), penetration rates for volatile substances through control devices, emission factors of organics, and particulate emissions. Although trace substance concentrations and correlation parameters are specific to each plant, the uncertainty ranges used here are aggregated over coal type and control type. This allows for the same uncertainty range for trace substance concentration and correlation parameter to be applied to any plant within a group in order to gain a general understanding of the sensitivity of the risk results to input variability.
- *Oil-fired plants (controlled and uncontrolled).* Emission factors for all trace substances.
- *Gas-fired plants.* Emission factors for all trace substances.

Particulate emissions from coal plants are not included in this sensitivity analysis because uncertainty data were unavailable. However, particulate emissions may have substantial impact on risk and hazard index estimates. Because uncertainty ranges for emission factors for substances from controlled oil plants and gas plants were unavailable, the analyses for these plant types involve sensitivity only, and assume ranges of uncertainty for the parameters. The uncertainty ranges of emission factors for controlled oil plants are assumed to be the same as those for uncontrolled oil plants (i.e., the uncertainty in the arsenic emission factor for controlled oil plants is assumed to be the same as that for uncontrolled oil plants, which ranges from 53% to 118% of the nominal value). Since the only uncertainty range available for gas plants is for the formaldehyde emission factor, the sensitivity analysis assumed uncertainty ranges of ±100% for all substances. Therefore, the gas plant results indicate which parameters most affect the carcinogenic inhalation risk estimates assuming equivalent levels of uncertainty.

Since the sensitivity analysis provides insight into which uncertainties affect the risk estimates the most, it helps identify which parameters may warrant further research to reduce the uncertainty. This sensitivity analysis does not, however, provide information

on the sensitivity of risk to compound uncertainties in more than one parameter (e.g., high end uncertainty of trace substance content combined with that of emissions correlation parameters).

7.6.1. Results of Sensitivity Analysis of Carcinogenic Inhalation Risk

Figure 7-10 shows the range in carcinogenic inhalation risk for the median plant implied by the uncertainty in the key parameters shown, for plants grouped by fuel type. Coal plants are grouped by coal rank, as the key uncertain parameters vary more along this dimension. For each plant group, the key uncertain parameters are listed. The range of risk shown in this figure applies to the *median* risk in the group and should not be assumed as the range of risk due to uncertainty for the *high* or *low* risk plants in the group. That is, Figure 7-10 shows a reasonable range of uncertainty around a plant at the middle of the distribution, not the most extreme high and low plants.

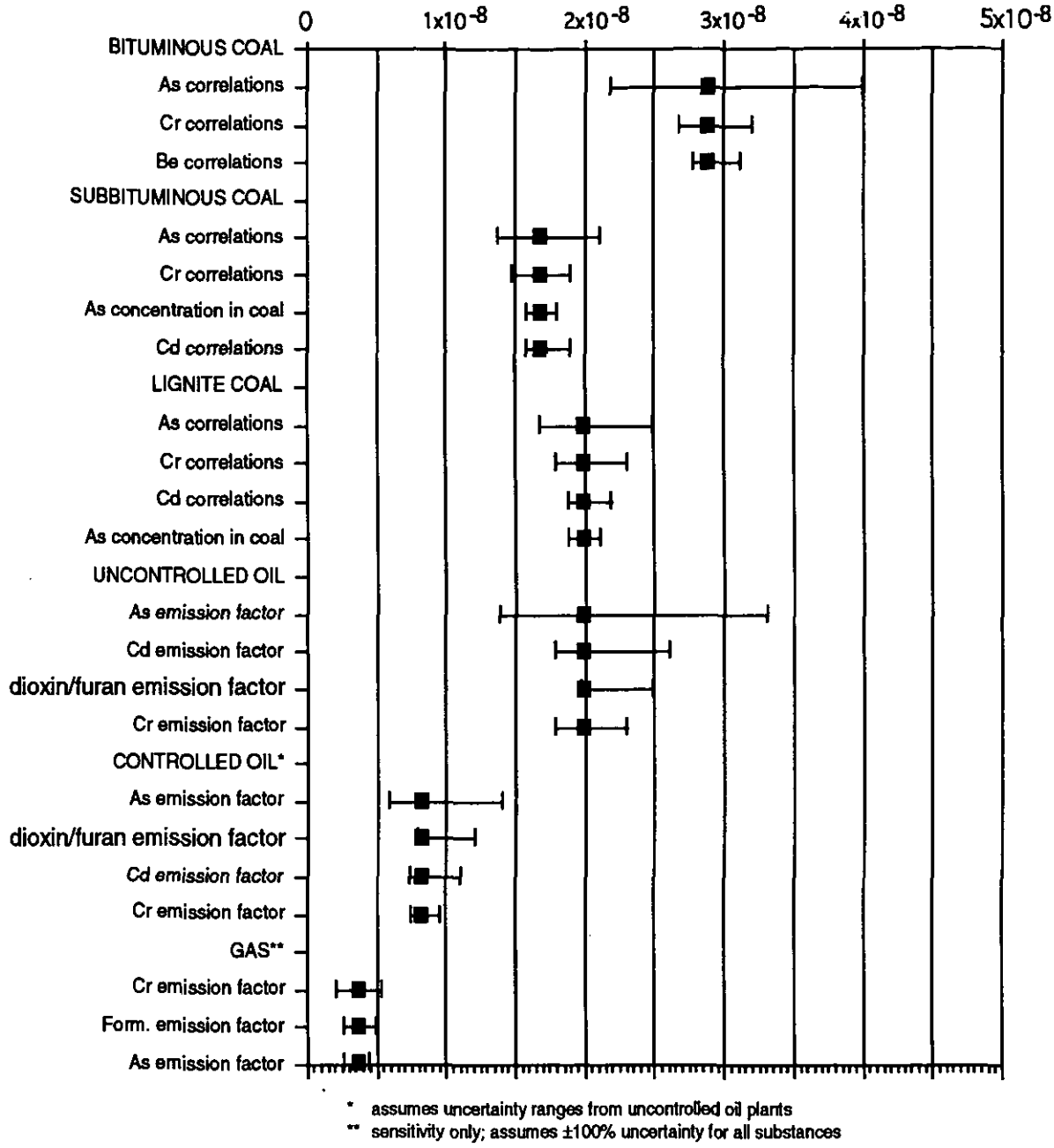


Figure 7-10.
Sensitivity of Median Cancer Risk Estimates to Uncertainty in Key Parameters (by Plant Group)

Excluding gas plants, the ranges of carcinogenic inhalation risk shown in Figure 7-10 are largest in percentage terms for oil plants, which reflects larger uncertainty ranges for key oil plant parameters than for other plants' parameters. Estimates for oil plants range from

30% lower to 90% higher than the median carcinogenic inhalation risk estimate, while estimates of carcinogenic inhalation risk for coal plants range from approximately 20% lower to 60% higher than the median carcinogenic inhalation risk estimate.

Across coal plants, carcinogenic inhalation risk estimates are consistently most sensitive to uncertainties in the correlation parameters for arsenic and chromium, while also somewhat sensitive to the correlation parameters for beryllium and cadmium, and the concentration of arsenic in coal. These parameters represent the four substances with the largest contributions to carcinogenic MEI inhalation risk for coal plants. Of all the parameters, carcinogenic inhalation risks for coal plants are most sensitive to uncertainty in arsenic correlation parameters. Although risk estimates are probably sensitive to particulate emissions, these were not included in the sensitivity analysis due to lack of data to establish uncertainty limits.

The carcinogenic inhalation risk estimates for uncontrolled oil plants show the most sensitivity to emission factors for arsenic, cadmium, dioxins/furans and chromium. Arsenic, cadmium, and chromium are the three largest contributors to carcinogenic inhalation risk for oil plants. Dioxins/furans, while contributing only 2% of the carcinogenic inhalation risk, have a highly uncertain emission factor. The sensitivity analysis for controlled oil plants (which assumes the uncertainty ranges for emission factors from uncontrolled oil plants) shows generally the same results as those for uncontrolled oil plants. Estimates of carcinogenic inhalation risk for oil plants are most sensitive to the arsenic emission factor.

Carcinogenic inhalation risk estimates for gas plants show highest sensitivity to chromium, formaldehyde, and arsenic emission factors. This reflects only the relative contribution of these substances to carcinogenic inhalation risk, since uncertainty ranges on the emission factors were unavailable.

7.6.2 Results of Hazard Index Sensitivity Analysis

For each plant group, Figure 7-11 shows the sensitivity of the median hazard index to uncertainty in parameters used to calculate the hazard index. Excluding gas plants, inhalation hazard indexes vary more in percentage terms for oil plants than for coal plants due to the uncertainty levels in key parameters. While inhalation hazard indexes for coal plants range from ±30% of the median estimate, inhalation hazard indexes for oil plants range from 30% lower to 50% higher than the median estimate.

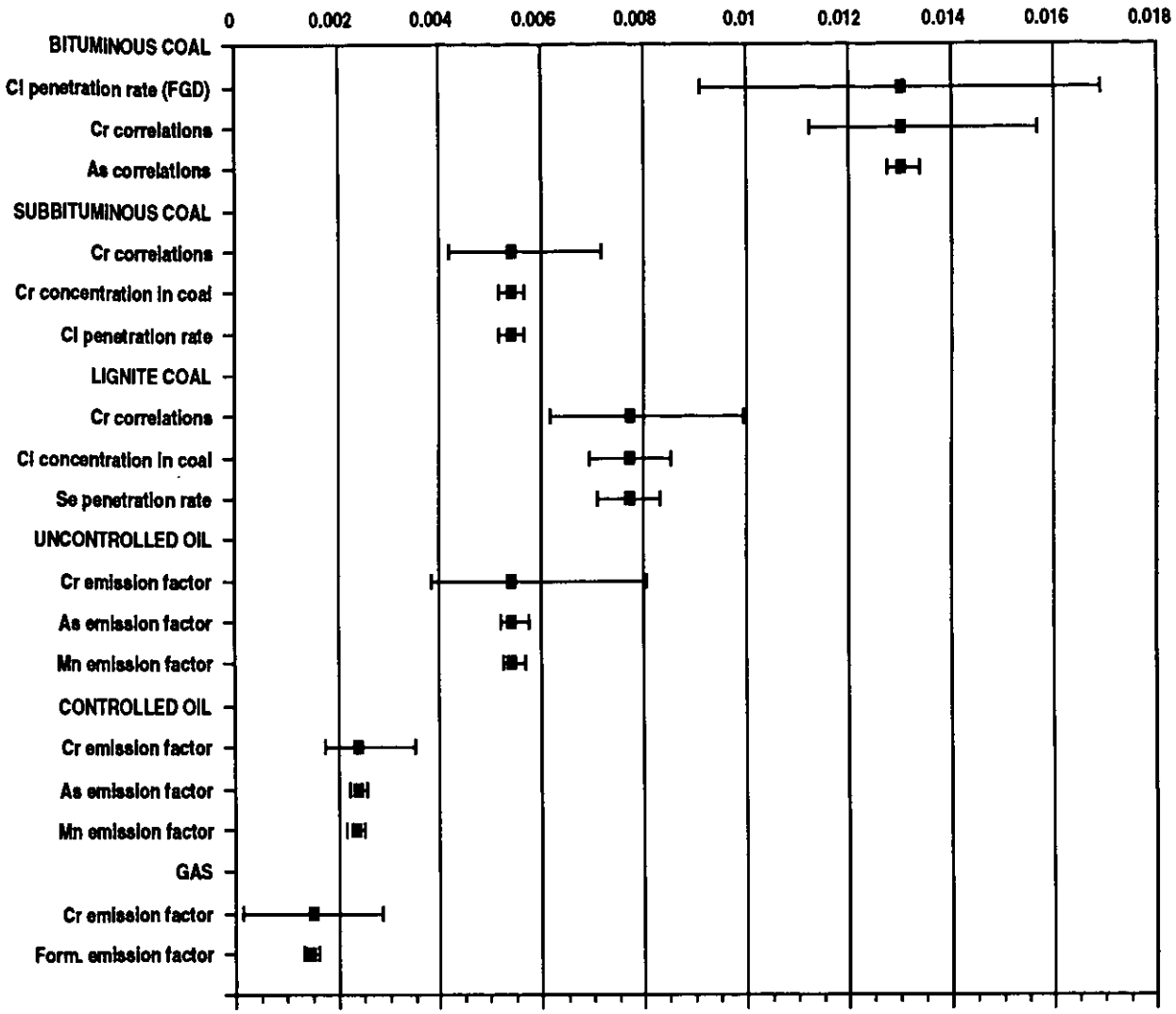


Figure 7-11.
Sensitivity of Median Hazard Index Estimates to Uncertainty in Key Parameters (by Plant Group)

The inhalation hazard index tends to be most sensitive to uncertainties in the parameters associated with the substances that contribute most to inhalation hazard indexes. Estimates of hazard indexes for coal plants are sensitive in general to parameters associated with HCl and chromium, which are the two largest contributors to inhalation hazard index for coal plants. While the removal efficiency for HCl affects only inhalation hazard indexes of bituminous plants with particulate controls-only by ±6%, it affects the estimates for bituminous plants with particulate and SO_2 controls by ±30%.

Uncertainty in the chromium emission factor has the most impact on estimates of inhalation hazard indexes for controlled and uncontrolled oil plants. The contribution of chromium to inhalation hazard index and the uncertainty in the chromium emission fac-

tor are both substantially higher than those for other substances. Again, in the absence of controlled plant data, the sensitivity analysis assumes that uncertainty in emission factors for controlled plants equals that for uncontrolled plants.

Inhalation hazard indexes for gas plants show highest sensitivity to chromium, and formaldehyde emission factors. As with the carcinogenic inhalation risk sensitivity analysis, this reflects only the relative contribution of these substances to inhalation hazard index, since uncertainty ranges on the emission factors were unavailable.

Although uncertainty in organic compound emission factors is relatively high, neither carcinogenic inhalation risk estimates nor hazard index estimates are sensitive to these uncertainties for any plant type, due to the small contribution of organic compounds to cancer risk estimates and hazard index estimates.

7.7 Summary

Over the total population (within 50 km of a given power plant), and using MEI assumptions, the annual cancer incidence to the inhalation of utility-emitted trace substances for the base case 2010 industry scenario is 0.08 cancer occurrences per year. Coal-fired plants with relatively large populations within 50 km tend to have the highest associated inhalation carcinogenic population risks, relative to other plant types and settings. However, the population risk is very small for all plants.

The highest inhalation MEI cancer risk is 1.7×10^{-6}, found for an uncontrolled oil-fired plant with extremely poor stack plume dispersion characteristics. For this one plant, arsenic and chromium contribute 55% and 22% of the risk, respectively. Coal-fired plants with particulate controls but no SO_2 controls tend to have the highest inhalation MEI risks. For these plants, arsenic and chromium contribute 59% and 23% of the risk, respectively. The REI living and working outdoors in the populated area with the highest concentrations from each plant is well under 10^{-6} for all plants. The substances contributing to the REI inhalation risk are the same as for the MEI inhalation risks.

Inhalation hazard quotients (characterizing potential risks due to noncarcinogenic modes of action for trace substances) are less than 1 for all individuals in the U.S. population. This holds for all analyzed substances from all power plants, suggesting that no significant inhalation-pathway noncarcinogenic health risks are associated with emissions of these substances. The highest inhalation MEI hazard quotient for any one plant and any one substance is 0.3, for hydrochloric acid from a bituminous coal-fired plant with only particulate controls. The inhalation hazard quotients for the REI across all plants is from 21% to 70% of the corresponding MEI inhalation hazard quotient.

Although variations in assumptions about future scenarios (e.g., load, fuel type, controls, etc.) can influence risk estimates for individual plants and relative risks across multiple plants, the alternative assessment scenarios did not significantly affect the risk estimates

overall. The arsenic scenario (use of the EPA unit risk vs. the more recent EPRI unit risk value) led to higher population risk and higher MEI risks for individual plants, while the particulate emissions limit scenario (assuming all units met NSPS standards) lead to slightly lower risk estimates for individual plants and for population risks.

Sensitivity analysis shows that uncertainty in any particular parameter does not increase the inhalation cancer risk estimates by more than about 90% or decrease it by more than about 80%, for single-parameter variability. Multivariate sensitivity analysis would, however, lead to greater variability ranges in the risk estimates. Larger uncertainty ranges for key parameters for oil plants result in higher sensitivity of cancer risk estimates for oil plants compared to that of coal plants. These same conclusions hold for sensitivity analysis of oil plant hazard quotients.

7.8 References

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2. U.S. EPA, 1990, "Cancer Risk from Outdoor Exposure to Air Toxics," EPA-450/1-90-0004a, Washington, D.C.
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4. U.S. EPA, 1987. The Risk Assessment Guidelines of 1986, EPA/600/8-87/045, U.S. Environmental Protection Agency, August 1987.
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8

MULTIMEDIA RISK ASSESSMENT

8.1 Overview

The previous chapter addresses human health impacts due to the inhalation pathway. Fully characterizing risks due to all pathways requires "multimedia" risk assessment. Assessing risks by all pathways, however, tends to be a complex process, requiring site-specific data as well as a number of assumptions for which qualifying data are often poor. To provide a standard framework for multimedia risk assessment, EPRI developed the "Total Risk of Utility Emissions" (*TRUE*) model, along with methodological extensions to that model to address uncertainty. Applications of the *TRUE* model and the uncertainty extensions provide insight into the multimedia pathways and consequent risks due to trace substance stack emissions from power plants. Because different modeling tools are typically used to assess the potential health risks of radionuclides and of substances acting by chemical routes, radionuclide risk modeling is the subject of a separate discussion.

Following a brief overview and a review of chemicals with multimedia effects, this chapter covers the following key areas:

- multimedia *TRUE* model and case study risk assessments
- radionuclide risk modeling
- uncertainty analysis for carcinogenic and mercury risk analyses
- current mercury research results.

Since material emitted from stacks may later deposit to waterways or ground surfaces, it is necessary to simulate a variety of physical, chemical and health-related processes that include: (1) the transport and fate of chemicals in the atmosphere, surface water, soil, groundwater and biota (i.e., food chain); (2) the exposure of the public to those chemicals; (3) the absorbed dose of those chemicals; and (4) the associated health response. Table 8-1 presents an overview of these different components of the health risk assessment.

Table 8-1.
Media and Pathways Analyzed by TRUE Model

Environmental Media		Exposure Pathways		
Individual	Intermedia	Inhalation	Ingestion	Dermal Absorption
AIR	AIR-SOIL –Dry deposition –Wet deposition	AMBIENT AIR –Gases –Particulates	SOIL	SOIL CONTACT
SOIL			WATER –Drinking –Swimming	WATER CONTACT –Swimming –Showering
SURFACE WATER	SOIL-SURFACE WATER –Overland Runoff			
GROUNDWATER	SOIL-GROUND-WATER –Infiltration		FOOD CHAIN –Produce –Fish –Beef –Dairy Milk –Mother's Milk	

8.2 Chemicals with Multimedia Pathways

For most trace substances, inhalation is the major route of entry to the human body. Some chemicals emitted as gases (e.g., benzene) may have negligible deposition rates. Without significant deposition, a chemical will not enter the soil and water bodies in significant amounts, and its effect on the food chain will be insignificant. Also, some chemicals are considered to have carcinogenic effects only via inhalation. For other chemicals that have significant atmospheric deposition rates, bioconcentration characteristics, and multipathway toxicity, non-inhalation pathways may be dominant.

A total of 20 chemicals or categories of chemicals was selected for study in EPRI's multimedia risk assessments associated with power plant emissions. These 20 substances included all of those listed in Section 1 and analyzed for the inhalation risk assessment of Section 7. (Although later PISCES sites have included measurements of radionuclides, these data were not available for the sites modelled for multimedia risks to date.) The substances studied under the multimedia assessments include 13 metals (arsenic, beryllium, cadmium, chromium (VI), chromium (III), copper, lead, manganese, mercury, molybdenum, nickel, selenium, and vanadium), polycyclic aromatic hydrocarbons (PAHs),

dioxins/furans, 2 inorganic gases (chlorine compounds and fluorine compounds), and 3 organic gases (benzene, formaldehyde, and toluene). Of these, only 8 chemicals are considered by EPA to have carcinogenic effects. Noncarcinogenic effects, however, are considered for all 20 chemicals.

Of the eight chemicals with carcinogenic effects, two—cadmium and hexavalent chromium—are considered to be carcinogenic only through inhalation. The remaining six (arsenic, beryllium, PAHs, dioxins/furans, benzene, and formaldehyde) may have carcinogenic effects via either the inhalation or non-inhalation pathways. Of these, PAHs and dioxins/furans are of greatest interest due to their complex set of non-inhalation pathways and their tendency to bioconcentrate in produce, fatty tissue, and milk.

Dioxins/furans were not measured at the units sampled during these PISCES programs. Dioxins/furans measurements were available at only one site for a different unit. Because of their potential importance to carcinogenic risks due to their ability to bioconcentrate in the environment, dioxins/furans were nevertheless considered in our analysis. Because of the paucity of sampling data, it was necessary to use modeled emissions at three of the four sites considered. Consequently, the carcinogenic health risks due to dioxins/furans is treated separately from the risk due to the other seven measured carcinogenic chemicals, to emphasize the difference of methodology used to characterize the emissions.

For noncarcinogenic effects, mercury is the chemical of greatest interest. Although mercury vapor presents an inhalation noncarcinogenic risk, mercury compounds are of greater interest primarily due to their ingestion pathway and the tendency of organic mercury compounds to bioaccumulate in biota, particularly in fish. Mercury exposure is expected to be particularly sensitive to the characteristics of receiving water bodies that are used for subsistence fishing. A more detailed discussion of human exposure to mercury via fish consumption can be found in Appendix N.

8.3 *TRUE* Model

Mathematical modeling of the health risk is carried out by combining appropriate models of transport and fate, exposure, dose, and health risk. The selection of these models is critical because a balance must be maintained between the availability of data needed to perform the modeling and the accuracy desired for the health risk estimates.

A comprehensive treatment of all these processes would require a multimedia transport model with fine spatial and temporal resolutions in all media, two-way intermedia transport, description of population exposure in a variety of microenvironments, and the development of accurate dose-response relationships. The level of detail provided by such a model may be unnecessary since the total health risk is likely to be dominated by exposure to a few chemicals transported through a few dominant routes. Consequently, it appears preferable to use a two-level approach to the modeling of the health risk.

In a screening analysis, the health risks can be estimated with a relatively simple assessment model. Such a screening-level analysis is intended to provide a moderate overestimate of the health risks using an easily manageable amount of data and readily available models. If the results of this screening analysis show that the estimated health risks for particular substances or pathways warrant further analysis, a more detailed assessment can be conducted. Such detailed analyses are designed to refine the modeling of important pathways, exposure routes, and health effects. These detailed calculations can then focus on the chemicals, transport pathways, exposure routes, and health effects that contribute the bulk of the calculated risk.

The *TRUE* model [1] is a tool for multimedia health risk assessment that can handle both screening-level and detailed site-specific analyses. The model combines a number of individual models to handle the transport and fate of chemicals in the atmosphere, surface water, surface soil, groundwater, and the food chain. Major intermedia transport processes are also included. Chemical concentrations calculated by the fate and transport models are used by exposure-dose models to calculate the individual doses, which are then used to calculate health risks.

TRUE takes as input power plant emissions, physical characteristics of the environmental media, food and water consumption information, and health effect parameters for the chemicals of interest, and provides an output of environmental concentrations, exposure doses, and human health risks.

A brief description of the *TRUE* model, including input requirements and output information, is provided in Appendix J.

8.4 TRUE Case Studies

8.4.1 Overview

The TRUE model was used to perform screening-level multimedia health risk assessments associated with the emissions of four fossil-fueled power plants. This section provides a summary of the findings of these assessments. A more detailed description of the sites, modeling approach and results can be found in Appendix K.

These four plants were chosen because of the variety of environmental settings represented, the availability of good unit emission measurement data, and the representativeness of the plants for substance emissions rate data in comparison to all plants modeled. (There is no way to judge the representativeness of risk results for these four plants, however, without doing a similar set of risk assessments for all plants in all locations.) For example, three of the four plants studied range from the 34th to 79th percentile of all (modeled) plants for arsenic emissions (the fourth had no detected arsenic emissions), and between the 38th and 98th percentile ranked on mercury emissions.

The emissions data for the 20 chemicals of concern (Section 8.2) were based on field measurements at operating power plants. In the case where measurements were not available for mercury and dioxins/furans, the 2010 modeled emissions that were used in the inhalation risk assessment (see Section 7) were selected. This was the case at one site for mercury and at three sites for dioxins/furans. For the chemicals that were measured at these sites, health effects were estimated only for chemicals sampled by PISCES at levels above the corresponding detection limit. The emission rates were scaled to represent the overall annual emission rates of the power plants in 2010 as described in Section 4.

The discussion of the results of the TRUE multimedia assessments is intended to:

- provide a quantitative characterization at a screening level of the potential health effects associated with power plant chemical emissions
- demonstrate the dependence of multimedia risks on the specific details of the source locale
- demonstrate the spatial variability of multimedia risks for a single site
- identify substances, exposure pathways, and environmental media driving the risk estimates.

8.4.2 TRUE Multimedia Health Risk Assessments—MEI Scenario

The four power plants modeled include three coal- and one oil-fired facilities. The coal-fired power plants are: (1) PISCES Site 12 in a rural area; (2) power plant Site A in a rural area; and (3) Site B in the vicinity of a large city. The oil-fired plant is designated Site C, and is located in a coastal environment. These power plants were selected to represent a variety of emission characteristics (i.e., different fuel and control equipment) as well as different environmental characteristics. Although other combination of emissions and environmental characteristics can be considered, the four case studies presented here cover a large range of such characteristics.

The exposure assumptions used in the four assessments correspond to those of an MEI.

The areas surrounding the four facilities exhibit significant differences with respect to the meteorological, climatological, hydrological, and hydrogeological characteristics, as well as water and fish supply sources. These differences result in estimated risk levels that vary significantly both among the sites studied, and among different locations surrounding the same site.

For Sites 12, A, and C, the calculated cumulative carcinogenic risks from each plant individually were less than forty in a billion (4×10^{-8}). Risks for Site B ranged from seven in a billion to less than one in a million, depending on the particular receptor area considered. The calculated cumulative hazard indexes for all sites remained below one in all subregions. Tables 8-2, 8-3, and 8-4 summarize the major findings of these assessments with

respect to carcinogenic risk, total noncarcinogenic health effects and mercury noncarcinogenic health effects including: dominant chemicals, dominant pathways, risk and hazard index ranges, and cancer burdens.

Table 8-2.
TRUE Case Studies — Carcinogenic Health Effects

	Site 12	Site A	Site B	Site C
Dominant Chemicals[1]	Arsenic (57%) Chromium (VI) (21%) Cadmium (21%)	Arsenic (68%) Chromium (VI) (16%) Cadmium (14%)	Arsenic (90%) Beryllium (9%)	Beryllium (95%) Cadmium (3%)
Dominant Pathways[1]	Inhalation (48%) Ingestion (52%)	Inhalation (36%) Ingestion (64%)	Inhalation (2%) Ingestion (97%)	Inhalation (11%) Ingestion (87%)
Risk Range (probability)	1.5×10^{-9} to 1.4×10^{-8}	5.5×10^{-9} to 4.0×10^{-8}	7.3×10^{-9} to 6.0×10^{-7}	1.7×10^{-9} to 2.0×10^{-8}
Total Population	645,896	225,419	2,127,210	6,614,776
Population Cancer Incidence (annual)	2.2×10^{-5}	4.9×10^{-5}	2.0×10^{-3}	6.8×10^{-4}

[1] Percentage contributions indicated correspond to subregion of maximum *total* risk. Contributions in other subregions varied.

The carcinogenic risk due to dioxins/furans was estimated to be 0.009, 1.9, 0.014, and 0.09 in one million for Sites 12, A, B and C, respectively. These risk estimates, however, are based on modeled emissions at three of the four sites since measurements were available only for Site C.

Two methods were used to speciate mercury emissions at Site A. The EPA speciation method were used for the non-carcinogenic health risks reported in Tables 8-3 and 8-4. The mercury speciation using the MESA method leads to slightly lower mercury health risk with a maximum mercury hazard index of 0.15 compared to the value of 0.28 reported in Table 8-4.

Table 8-3.
TRUE Case Studies — Non-Carcinogenic Health Effects

	Site 12	Site A	Site B	Site C
Dominant Chemicals[1]	Chromium (III) (68%) Chlorine comp. (11%) Mercury (7.6%) Lead (4.9%)	Mercury (97%)	Chromium (III) (37%) Fluorine comp. (28%) Chlorine comp. (22%) Mercury (4%)	Nickel (51%) Chromium (III) (23%) Vanadium (17.6%)
Dominant Pathways[1]	Inhalation (69%) Dermal Absorption (17%) Ingestion (14%)	Ingestion (98%)	Inhalation (76%) D. Absorption (16%)	D. Absorption (58%) Inhalation (33%)
Range of the Hazard Index	7.1×10^{-4} to 4.3×10^{-3}	6.1×10^{-3} to 2.9×10^{-1}	3.0×10^{-3} to 3.4×10^{-2}	2.8×10^{-3} to 1.5×10^{-2}

[1] Percentage contributions indicated correspond to subregion of maximum *total* hazard index. Contributions in other subregions varied.

Table 8-4.
TRUE Case Studies: Breakdown of Mercury Risks at Location of Maximum Mercury Hazard Index (Speciation Using EPA Method 29)

	Hazard Quotient			
Pathway	Site 12	Site A	Site B	Site C
Ingestion	3.2×10^{-4} (98%)	2.8×10^{-1} (100%)	3.6×10^{-3} (99.8%)	1.8×10^{-4} (99.5%)
Inhalation	6.2×10^{-6} (2%)	2.2×10^{-5} (<0.1%)	8.7×10^{-6} (0.2%)	7.9×10^{-7} (0.4%)
Dermal Absorption	1.5×10^{-7} (<0.1%)	6.4×10^{-7} (<0.1%)	1.3×10^{-6} (<0.1%)	1.1×10^{-7} (0.1%)
Total Hg Hazard Index	3.3×10^{-4}	2.8×10^{-1}	3.6×10^{-3}	1.8×10^{-4}

Note: Absolute risks and percentage contributions indicated correspond to the subregion of maximum Hg hazard index. Contributions in other subregions varied.

8.4.3 TRUE Case Studies—Alternative REI Scenario

The primary scenario considered in the four TRUE risk assessments described above was based on worst-case exposure assumptions characterizing a "Maximally Exposed Individual" (MEI). As a sensitivity analysis, an alternative scenario was evaluated utilizing more realistic assumptions for a "Reasonably Exposed Individual" (REI). The exposure parameters for these two scenarios were described in Section 5. The same exposure assumptions were used for the inhalation risk as in the inhalation risk assessment (see Section 7). For the ingestion risk and dermal absorption risk, the exposure assumptions affecting the REI were the change in body weight and the residence time. REI calculations were performed for carcinogenic risks only.

The alternative REI scenario resulted in carcinogenic risks which ranged between 24% and 30% of the values of the corresponding MEI risks. A summary of the estimated carcinogenic risks for the two alternative scenarios is provided in Table 8-5 for each individual case study.

Table 8-5.
Comparison of REI to MEI Carcinogenic Risk for TRUE Case Studies

	Risk (Lifetime Cancer Probability)			
Exposure Scenario	Site 12	Site A	Site B	Site C
MEI[a]	1.4×10^{-8}	4.0×10^{-8}	6.0×10^{-7}	2.0×10^{-8}
REI[b]	3.4×10^{-9} (24%)[c]	1.0×10^{-8} (26%)[c]	1.8×10^{-7} (30%)[c]	5.0×10^{-9} (25%)[c]

[a] MEI = Maximally Exposed Individual
[b] REI = Reasonably Exposed Individual
[c] Number expresses REI risk as a percentage of the corresponding MEI risk.
(The exposure assumptions used for the MEI and REI scenarios are presented in Section 5.)

8.4.4 TRUE-MCM Combined Analysis

Due to the complexity of mercury (Hg) behavior in the environment, and the significance of the aquatic environment in the resulting Hg concentrations in fish, a reliable assessment of mercury risks requires comprehensive treatment of its cycling in surface water bodies. For that purpose, EPRI's "Mercury Cycling Model" (MCM) [2] was combined with TRUE for the evaluation of noncarcinogenic risks associated with Hg power plant emissions. MCM takes as input Hg loads, hydraulic and chemical lake characteristics, and provides an output of Hg concentrations in lake water and fish. A more detailed description of MCM is provided in Appendix O.

The combined approach was used to evaluate the multimedia risks associated with the Hg emissions of Sites 12 and C. These two sites were selected because there were lakes in their general vicinity for which sufficient information was available to apply MCM. These lakes were part of a set of 15 lakes nationally used to evaluate MCM results for a variety of hydrologic conditions. In this exercise, all characteristics of the sites were maintained as in their actual environments, with the exception of the water bodies, which were substituted by alternative lakes for which the necessary data for the MCM analyses were available. For each site, two alternative lakes were analyzed to evaluate the sensitivity of Hg risks to the characteristics of the aquatic environment.

Table 8-6 summarizes the relevant hydrologic and water quality characteristics for the four lakes considered. The selection of the lakes to be used with each power plant was performed on the basis of geographic proximity to the site. Lakes I and II (as listed in Table 8.6) were used in the analysis of Site 12, whereas Lakes III and IV were used with Site C.

Table 8-6.
Characteristics of the Lakes Used in the TRUE-MCM Combined Analysis

		Site 12		Site C	
		Lake I[1]	Lake II[1]	Lake III[2]	Lake IV[2]
Hydrologic Parameters	Hydrologic Status	Drainage	Reservoir	Reservoir	Drainage
	Surface area (km^2)	166	5.4	0.49	0.56
	Watershed area (km^2)	2027	1937	2.8	3.6
	Mean depth (m)	54	6.4	2.4	3.5
	Residence time (yr)	9.5	0.05	0.68	0.8
Water Quality Parameters	Nutrient status pH	Mesotrophic 8.2	Eutrophic 8.0	Hypereutrophic 7.5	Mesotrophic 4.9
	DOC (µg/l)	2.6	6.8	4.8	0.67
	POC (µg/l)	0.8	1.7	1.0	0.07
	Cl- (µeq/l	1363	1207	422	112
	Ca++ (µeq/l)	2092	2227	371	34
	SO4= (µeq/l)	719	1119	159	128
	Chl a (µg/l)	1.4	25.9	24.0	0.2

[1] Lakes I and II were used with site 12
[2] Lakes III and IV were used with site C

The resulting mercury concentrations in water, fractions of methylated Hg, and methylmercury water-to-fish BAFs varied among the different lakes, depending on their hydraulic and water quality characteristics, thus causing an equivalent variation in the resulting risks. The results of the MCM simulations for each individual site and lake are provided in Table 8-7.

Table 8-7.
MCM–TRUE Combined Analysis — Predicted Hg Concentrations and Methylmercury Water-to-Fish Bioconcentration Factors

	Site 12		Site C	
	Lake I	Lake II	Lake III	Lake IV
Concentrations in Water (µg/l)				
Hg(II)	2.6×10^{-9}	4.2×10^{-7}	4.6×10^{-8}	1.5×10^{-7}
Methylmercury	1.6×10^{-9}	5.7×10^{-8}	1.6×10^{-8}	1.2×10^{-8}
Hg0	4.8×10^{-8}	1.5×10^{-7}	8.5×10^{-9}	0.0
Methylmercury BAF[1] (l/kg)	3.7×10^{4}	1.5×10^{4}	1.3×10^{5}	3.6×10^{6}
Methylmercury Concentrations in Fish (ppm)	5.9×10^{-5}	8.5×10^{-4}	2.0×10^{-3}	4.1×10^{-2}

[1] Methylmercury BAF values provided are based on the *fresh* weight of fish.

The estimated multimedia hazard indexes were significantly less than 1 for both sites and all alternative lakes evaluated. Table 8-8 summarizes the corresponding breakdown of the mercury multimedia hazard indexes among the various pathways considered.

Table 8-8.
MCM–TRUE Combined Analysis: Predicted Mercury Health Risk for the Locations of Maximum Hazard Index for Mercury

	Hazard Quotient or Hazard Index			
	Site 12		Site C	
Pathway	Lake I	Lake II	Lake III	Lake IV
Ingestion				
Produce	2.3×10^{-4}	2.3×10^{-4}	1.7×10^{-4}	1.7×10^{-4}
Soil	3.2×10^{-7}	3.2×10^{-7}	2.3×10^{-7}	2.3×10^{-7}
Drinking water	5.0×10^{-6}	6.0×10^{-5}	6.7×10^{-6}	1.6×10^{-5}
Swimming water	6.1×10^{-9}	7.4×10^{-8}	8.2×10^{-9}	1.9×10^{-8}
Fish	1.3×10^{-4}	5.6×10^{-3}	4.0×10^{-3}	7.5×10^{-2}
Subtotal	3.7×10^{-4}	5.9×10^{-3}	4.2×10^{-3}	7.5×10^{-2}
Inhalation	6.2×10^{-6}	6.2×10^{-6}	7.6×10^{-7}	7.6×10^{-7}
Dermal				
Water	5.5×10^{-8}	6.6×10^{-7}	7.3×10^{-8}	1.7×10^{-7}
Soil	1.5×10^{-7}	1.5×10^{-7}	1.1×10^{-7}	1.1×10^{-7}
Subtotal	2.0×10^{-7}	8.1×10^{-7}	1.8×10^{-7}	2.8×10^{-7}
Total	3.7×10^{-4}	5.9×10^{-3}	4.2×10^{-3}	7.5×10^{-2}

8.5 Radionuclides

8.5.1 Introduction

This section addresses the assessment of radionuclide emissions from fossil fuel-fired power plants. Only a short summary of results is provided here; a more detailed description is included as Appendix L.

Unlike the analyses for conventional chemical emissions, risk calculations for all U.S. power plants have not been made by EPRI. For this reason, this section and Appendix L focus more on the results of calculations made for representative plants (those analyzed by EPA in its 1989 NESHAPS analysis) and on methodological issues associated with assessment of risks from radionuclide emissions.

This section addresses two aspects of the analysis of radionuclide risks from coal-fired power plants: the radionuclide source term and the indirect exposure pathway analysis.

8.5.2 Radionuclide Source Terms

A key aspect of the assessment of risks from radionuclide emissions from fossil-fueled power plants is the estimation of the emission source term. In the 1989 NESHAPS, emissions were estimated based on an average emission factor in the units of radionuclide emissions per unit combustion energy (e.g., pCi per 10^6 Btu). Enrichment factors were used to account for the tendency of some radionuclides to preferentially collect in or on small particles.

An alternative approach is taken here, in which radionuclide emissions are assumed to be directly proportional to particulate emissions. Revised source terms were calculated using such an approach for the eight NESHAPS plants, using the same particulate emission factors developed for the chemical risk assessment. This analysis produced comparatively small changes in the estimated source terms for "typical" plants, i.e., the smaller of the plants considered. For these four plants, two showed slightly higher emissions under this method, two somewhat lower emissions. The difference between the two methods for estimating emissions was more significant for the "large" plants, due to the fact that large plants tend to be newer and better controlled than average plants.

8.5.3 Results

The calculated maximum individual risks and population risks for the eight NESHAPS plants are indicated in Appendix L on Figures L-1 and L-2. These results indicate that CAP93-PC calculates a range for the maximum lifetime individual risk, using a source term estimate based on fly ash emissions, of between 3.6×10^{-7} and 2.6×10^{-6}, with an average individual risk of 1.2×10^{-6}. These risk estimates correspond to an estimated annual effective dose equivalent of less than 0.016 to 0.185 mrem per year for the fly ash source term (the average MEI dose is 0.08 mrem/yr). For the source term used by EPA in its 1989 NESHAPS assessment, the lifetime risks ranged from slightly below 10^{-6} to 1.6×10^{-5}, with an average value of 5.4×10^{-6}. This range corresponds to an annual dose to the maximum exposed individual of between 0.075 and 1.13 mrem per year, with an average for the eight NESHAPS plants of 0.32 mrem per year.

As an indication of the significance of such doses, it is noteworthy that the National Commission on Radiation Protection and Measurements (NCRP) has defined a negligible individual dose rate at a level of 1 mrem per year, (NCRP 116, 1993), and that EPA has not considered such exposure levels to be significant in other regulatory contexts. The individual radiation doses are low by conventional standards for radiation protection. However, it is customary in radiation protection to also consider whether population doses might be significant. For the eight NESHAPS plants using a fly ash-based source term, the population risk in terms of cancer mortality per year is estimated to range from 1.8×10^{-5} to 1.4×10^{-3} cancer fatalities per plant per year, with an average value of

3.9×10^{-4} per plant per year. For a population of roughly 600 power plants, this would correspond to a national estimate of about 0.2 cases per year in the United States. See the technical appendix for a fuller discussion of these results.

8.5.4 Exposure Pathways

The exposure pathways of greatest significance for individual risk are not the same pathways that make the major contribution to population risk. For individual risk, exposures and risks are dominated by direct external exposure to radionuclides on the ground surface, and, to a lesser extent, to ingestion of radionuclides in food. For the eight plants analyzed, the ground surface exposure pathway contributed about 83% of the total risk, with risks from ingestion contributing about 16% of the total. Inhalation was not a significant contributor to individual risk. For population risk, inhalation is the dominant pathway, averaging about 84% of the total, with the remaining population risk from ground surface exposures (10%) and from ingestion (5%). This surface exposure term incorporates very long-term build-up of deposited particulate matter in each location, with small removal terms due to such natural processes as surface water runoff.

As is noted in the technical appendix, the methods by which exposures through food consumption are calculated for radionuclides include more conservative assumptions than do the standard methods for chemical risk assessment. The major reason for the differences is apparently that the radionuclide model assumes greater consumption than do the other models, particularly for produce.

The population risk estimate is largely determined by the calculated inhalation doses. This analysis is less uncertain than the calculation for individual risk for several reasons. The inhalation dose is not dependent on a calculation of the buildup of past releases, nor is the result as sensitive to the precise location of individuals. Both analyses make certain conservative assumptions (e.g., that those exposed live outdoors at one location for 70 years).

8.5.5 Comparison With Chemical Risk Analysis Results

Although it may be desirable to calculate the sum of risks from emissions of both radioactive and chemically toxic substances from power plants and to evaluate their relative significance, there are several methodological problems with combining the results of CAP93-PC with those from a chemical risk assessment, at least for calculations of individual risk. The comparison is only valid to the extent that comparable analytical methods and assumptions have been applied, which is not the case. An important limitation is that the maximum individual risk, as calculated by CAP93-PC, is likely to be at a different location than the point of maximum chemical risk. The location of the MEI in CAP93-PC is determined by the ground surface exposure term; this pathway has no chemical analog. Because these MEI risks are not at same place, they are not calculations of risk to the same

individual and therefore should not be added. Because population risk estimates are determined largely from inhalation exposures, comparisons of radionuclide and chemical risks to population should be more reliable.

These limitations notwithstanding, there appear to be several observations that can be made based on analyses done of the NESHAPS plants for both chemical and radionuclide risk. First, it appears that the risk to the most exposed individual from radiation, taking all exposure pathways into account, is greater than the risk to the most exposed individual from the inhalation of chemical toxicants. In the calculations reported here (and using the fly ash-based source term for radionuclides), it appears that the population risks from the eight plants evaluated for radionuclide risk have a population risk that is, on average, about equal to that calculated for chemicals due to inhalation risk alone.

8.5.6 Conclusions

As this section notes, the calculated risks from radionuclide emissions appear to be lower than indicated by the analysis made in the 1989 NESHAPS. This is due both to an improved method for calculating the source term and from corrections made to the model used for this analysis. This section also notes that the multipathway analysis made for radionuclides gives higher exposure estimates than other common methods for risk analysis, by a factor ranging from 2.3 to 6.8. These limitations notwithstanding, a full reading of the radionuclide and chemical assessment indicates that, on average, the population risks from radionuclides (dominated by inhalation exposure) and from chemical carcinogens by inhalation exposure only are about equal. However, this conclusion about the relative significance of radionuclide emissions and risks from power plants, in comparison to chemically toxic materials, is uncertain.

8.6 Uncertainty Analysis

Assessment of human health risks due to trace emissions from power plants should include explicit treatment of uncertainty. Such uncertainties arise in both models and data. These uncertainties need further investigation. Following a brief overview on uncertainties in health risk assessment, this section describes extensions to the *TRUE* model which are used to characterize the uncertainty in health risks, and applies the approach to two example power plants.

8.6.1 Overview

Uncertainties in health risk assessments arise in: (1) the formulation of the models used, and (2) the estimation of the parameter values used as input to these models.

The uncertainty due to model formulation can be reduced to some extent by using models that provide a more comprehensive treatment of the relevant physical and chemical processes. Another source of uncertainty arises from the assumptions and simplifications made when the chosen models are linked together. These uncertainties need further investigation. The focus in this discussion is on the uncertainties due to the input parameters for a given health risk assessment model. For that reason, and since the model uncertainties are not included, the relative magnitude of results (expected values versus point-estimate values) is more significant than their absolute magnitude.

By characterizing the uncertainties (or variability) in model input parameters and studying the effects of variation in these parameters on the model predictions, an estimate can be made of the part of the uncertainty in the predictions that is due to uncertainty in the inputs.

8.6.2 Approach

The methodology for the uncertainty analysis of health risk estimates consists of the following 6 steps:

- Step 1: *Sensitivity analysis of the health risk assessment model.* This analysis allows one to determine the influential parameters of the model, that is, those that have the most significant effect on the model output.
- Step 2: *Estimation of parameter uncertainty.* This task involves the derivation of "uncertainty indexes" which quantify the uncertainty in the individual parameter values. Combination of the uncertainty indexes with the results of the sensitivity analysis leads to the selection of the critical parameters, that is, those that need to be included in the final uncertainty analysis.
- Step 3: *Construction of parameterized models with critical parameters as the sole variables.* This reduces the number of parameters in the analysis, simplifying the uncertainty propagation process.
- Step 4: *Selection of probability distributions for the critical parameters.*
- Step 5: *Propagation of the parameter uncertainties.* This task is performed with the parameterized version of the model and provides the uncertainties in the model outputs.
- Step 6: *Analysis of the probability distribution of the risk estimates.*

A more detailed description of each individual step of the methodology can be found in Appendix M.

8.6.3 Example 1—Uncertainty Analysis of Carcinogenic Health Risk Estimates

The case study presented here, an uncertainty analysis of carcinogenic health risk, was carried out for case study power plant Site 12. The above-described methodology is applied to the lifetime carcinogenic risk for the maximally exposed individual due to stack emissions of all carcinogenic chemicals detected at that site (chromium (VI), arsenic, cadmium, and benzene).

Sensitivity analysis performed for the various model components resulted in the selection of 22 critical parameters (out of a total of 52 examined) to be included in the final uncertainty analysis. Appropriate probability distributions were then selected for these parameters on the basis of available statistical data, literature value ranges, and personal judgment.

A probabilistic analysis with 10,000 iterations on a parameterized version of the model resulted in a probability distribution of the carcinogenic risk for arsenic, cadmium, and chromium (VI) together. The distribution was positively skewed (i.e. right tail) with a mean (i.e. expected value) of 1.2×10^{-5}. The risk value calculated in the deterministic assessment (i.e., 1.4×10^{-5}) was estimated to be at the 80th percentile of the derived probability distribution.

The major sources of uncertainty were the exposure duration and the cancer potency slope factor for ingestion.

A more detailed description of this application can be found in Appendix M.

8.6.4 Example 2—Uncertainty Analysis of Mercury Health Risk Estimates

The case study presented here corresponds to the stack mercury emissions at case study power plant Site A. The uncertainty analysis was carried out for the subregion in the plant modeling domain with maximum risk from all chemicals and all pathways due to exposure to air emissions.

Uncertainties in the estimation of mercury health risks are involved in all parts of the risk assessment procedure including the modeling of environmental and food chain fate and transport, the estimation of individual exposure, and the prediction of health risks. Of the several environmental processes involved in modeling mercury transport, fate, and health effect, particular attention was devoted to: (1) atmospheric chemical transformations, (2) dry deposition, (3) wet deposition, and (4) aquatic chemical transformations.

Sensitivity analysis performed on the various components of the *TRUE* model resulted in the selection of 28 critical parameters. These included: mercury atmospheric chemistry and physical morphology, dry and wet deposition processes, methylation of mercury in

the aquatic environment, food chain bioconcentration factors, and toxicology. However, as discussed in Appendix M, uncertainties in reference doses were not addressed in the probabilistic analysis.

A probabilistic analysis with 10,000 iterations on a simplified version of the model resulted in a probability distribution of the mercury hazard index. The distribution was positively skewed with a mean of 0.022. The hazard index value calculated in the deterministic assessment (0.28) was estimated to fall beyond the 95th percentile of the derived distribution. The major sources of uncertainty were the fish ingestion rate, the water-to-fish bioconcentration factor for methylmercury, and the deposition velocity for gaseous divalent mercury.

A more extensive discussion of the uncertainties involved in the estimation of mercury health risks and a more detailed description of this application can be found in Appendix M.

8.7 Summary

The results of modeling risk by multiple exposure pathways for four selected case study power plants indicate that the substances that are the key drivers of risk, including mercury, do not pose significant health risks for these instances. Although the power plants modeled span a wide range of substance emission rates when compared to other power plants for the same substances, there is no current procedure for gauging how representative the risk results are for the entire national population of power plants and their environmental settings. Nevertheless, by inclusion of site-specific information, both screening-level analyses and more detailed analyses involving modeling of the aquatic pathway for mercury indicate that mercury risks are relatively small due to emissions from these power plants.

Results to date for radionuclides indicate that the way in which physical processes are modelled is critical to the calculated magnitude and location of the resulting risks. These processes for particle-phase material (the predominant form for radionuclides from power plants) require additional investigation to be well-represented in future modelling efforts. In addition, more site measurement data on radionuclide fuel content for oil plants, and emissions from those plants, are required for accurate modeling of the resulting risks.

8.8 References

1. Constantinou, E. and C. Seigneur "A Mathematical Model for Multimedia Health Risk Assessment" *Environ. Software*, Vol. 8, pp. 231-246, 1993.
2. Hudson, R.J.M., S.A. Gherini, C.J. Watras, and D.P. Porcella, "Modeling the Biogeochemical Cycle of Mercury in Lakes: The Mercury Cycling Model (MCM) and its Application to the MTL Lakes." Proceedings of the 1992 Conference on Mercury as a Global Pollutant. 1994.

9

INSIGHTS AND CONCLUSIONS

This section summarizes the key results and insights gained from the research and analyses presented in this report. First, the section summarizes the key research findings in the development of data and methodologies for assessing trace substance emissions and their potential impacts on human health. Then, the section summarizes key results and conclusions from the inhalation risk assessment, and insights gained from the multimedia risk assessment case studies. Taken as a whole, this research suggests that trace substance emissions from electric utility steam generating units do not pose significant risks to human health.

9.1 Summary of Contributing Research

As part of the study of trace emissions from power plants, EPRI, in cooperation with the Utility Air Regulatory Group (UARG), the U.S. Department of Energy (DOE), and other organizations, developed new methods and information necessary for understanding the nature of these emissions and their potential health risks. A summary of the key insights gained from this research follows.

9.1.1 Concentrations of Trace Substances in Coal (Section 4.2)

- A data set was developed on the concentrations of inorganic substances in coals "as-fired" at power plants. The data set selected only economically usable coals from the USGS COALQUAL database and then simulated the effect of coal cleaning processes on the concentrations of many trace substances in those coals. To augment these data, a measurement program determined the concentration of mercury in "as-fired" coals based on approximately 150 samples. Together, these data provide improved estimates of the actual concentrations of trace substances in as-fired coals. A key result is that concentrations of mercury in coal based on as-fired samples are about half of those values using earlier estimates based on "in-ground" samples.

9.1.2 Sampling and Analytical Methods (Section 2)

- EPA-recommended sampling and analytical methods were initially chosen for the field program as appropriate and technically-accepted techniques. However, critical limitations of these techniques were discovered when they were applied to measuring trace substance concentrations in field conditions at operating power plants. In particular, it was very difficult to acquire reliable measurements in very high flow rate/low

concentration streams such as flue gas conditions. Several modifications were made to the initial methods in order to overcome practical difficulties and method limitations. Re-tests at some of the earlier field sites were carried out using the modified methods to obtain more meaningful results.

- Further developmental work to improve and validate sampling and analytical methods is planned or in progress (e.g., determination of speciation of mercury and other metals in utility combustion gases). Low mercury concentrations in utility flue gas are difficult to measure, and EPRI's measurement methods as well as those for speciating mercury in flue gas have yet to receive formal EPA validation.

9.1.3 Field Measurements and Data Correlations (Section 3)

- EPRI and DOE have tested units at nearly 45 fossil fuel-fired power plants in order to characterize emissions of hazardous air pollutants. The recent field tests include plants representing each major fuel type and boiler configuration, as well as the range of existing SO_2, NO_x, and particulate control technologies. The resulting database represents the best dataset currently available to estimate trace emissions from electric utility stacks, and the health impacts of those emissions.
- The results of these field tests were quite variable, with emissions of a specific substance (expressed as mass emitted normalized by heat input) ranging over several orders of magnitude across all plants. Therefore, the results were subdivided into smaller subsets in order to account for a number of potential variables such as fuel type and SO_2 and particulate control technologies. The results for these subsets could then be used to project emissions for other plants with similar fuel, control, and operating characteristics.
- The trace substances were divided into three major groupings in order to develop emission factors or correlations for estimating emissions for the entire capacity of electric utility steam-generating plants. The correlations or average emission factors suggested in this report are appropriate for estimating emissions for individual units across the entire utility industry, for units aggregated into power plant configurations, or for sets of grouped power plants. They are not precise enough, however, for estimating emissions from any particular power plant for the purpose of site-specific studies. For example, their use in developing permit conditions for individual units would be inappropriate.
- For coal-fired power plants, the following guidelines were developed for extrapolating the measurement data to predict trace emissions from similar units:
  - Particulate-phase inorganic substances. Based on the field data, these substances (e.g., arsenic, chromium) are well controlled by a particulate control device. In general, reductions of greater than 90% from levels in the incoming coal were achieved. Correlations were thus developed to estimate stack emissions based on the inlet coal concentration of each substance and the level of total particulate emissions.

  - Volatile inorganic substances. These substances (including hydrochloric acid, mercury, and selenium) tend to be more volatile and not consistently captured by particulate control devices. Based on the measurement data, the emissions of these substances could not be correlated to any specific factors and were therefore estimated using average removal efficiencies for each substance and control configuration across sampled units.
  - Organic compounds. These compounds are formed at very low levels during combustion, and emitted in concentrations of parts per billion or lower. Emissions of organic compounds were estimated using the geometric means of measured emission factors, calculated from the field data, for each substance.

- For oil- and gas-fired power plants, available data are not yet adequate to estimate the trace substance concentrations in fuel burned at individual utility sites on a nationwide basis. Emissions were estimated using sample-averaged emission factors, based on the field measurements, across all power plants of the same configuration and fuel type. These data show that the emission factors (i.e., emissions per Btu of heat input) for uncontrolled oil-fired power plants are about the same as for coal-fired plants with ESPs.

9.1.4 Future Scenarios of the Electric Utility Industry (Section 4.1)

- Three separate scenarios describing how the electric utility industry will be configured and will operate after compliance with SO_2 provisions of the Clean Air Act Amendments of 1990 were produced. One such scenario, originally produced for EPA by contractors, was further disaggregated for this study to the level of individual boiler units. Two additional scenarios of the future industry were independently derived by EPRI researchers.
- Additional data on planned and projected particulate controls were incorporated into these scenarios for use in the trace substances study. UARG independently surveyed utility operators on their plans for modifying, upgrading, or retrofitting particulate control equipment at their plants. Of the responding operators, 10% indicated that they have plans to make changes in particulate control equipment for one or more units before the year 2010. These data were used to augment the future scenarios which had focused primarily on SO_2 compliance projections.

9.1.5 Inhalation Exposure Modeling (Section 5)

- In addition to standard assumptions for a maximally exposed individual (MEI), EPRI developed an exposure methodology for estimating the risks to a reasonably exposed individual (REI) living in the area with the highest air concentrations from plant stack emissions. This methodology accounts for human activity patterns, indoor concentrations due to outdoor sources, residential moves, and power plant operating lifetimes. This REI estimate is intended to more closely approximate typical risks to an individ-

ual than does the standard MEI measure, which is considered an upper bound on plausible exposure. REI exposure estimates for carcinogens are about an order of magnitude less than the corresponding MEI exposure estimates.

9.1.6 Health Effects—Arsenic and Mercury (Section 6.3)

Arsenic

- EPRI research has led to a revised inhalation unit risk factor for arsenic. This revised estimate was developed using EPA-standard risk assessment methods to examine new and previously-analyzed occupational exposure data. This revised unit risk factor, 1.43×10^{-3} $(\mu g/m^3)^{-1}$, is one-third that currently listed in the EPA IRIS database. The revised value reflects reassessment of exposure levels by the original researchers whose studies formed the basis for the EPA-listed unit risk value, as well as results from a recently published Swedish study.
- The current EPA inhalation unit risk for arsenic is based upon observation of increased risk of lung cancer among copper smelter workers. However, copper smelter dust may produce more inflammatory response in the lung than does coal fly ash. If substantiated, this finding has potentially important implications since inflammation is thought to be a critical step in the progression to cancer. Also, arsenic from copper smelter dust is more highly retained in the lung and excreted more slowly in the urine than is arsenic from coal fly ash, indicating that coal fly ash arsenic may be less bioavailable than arsenic in copper smelter dust. These issues with respect to arsenic toxicity remain the subject of ongoing research at EPRI and elsewhere.

Mercury

- Recent data also may provide an improved basis for computing potential neurotoxic effects due to chronic exposures to mercury. EPA's current reference dose for methylmercury is based on an incident in Iraq involving acute exposures to very high methylmercury concentrations in grain. However, data sets based on populations exposed to mercury via fish ingestion may be more appropriate for evaluating health risks from utility mercury emissions. EPRI is currently assessing data on the neurological responses of children in New Zealand exposed to methylmercury via maternal fish ingestion.

9.1.7 Radionuclide Research (Section 8.3)

- Based in part on comments from EPRI and UARG, EPA revised several aspects of the radionuclide risk assessment methodology used in its 1989 report on National Emissions Standards for Hazardous Air Pollutants (NESHAPS) for radionuclides (including coal-fired power plants). These changes tend to lead to lower risk estimates from the model. Important changes include:

Revised emission estimation method. EPA's Office of Radiation and Indoor Air accepted a revised method for estimating radionuclide emissions based on the average radioactivity of emitted particulates rather than on total coal consumption. For large coal-fired plants, this results in predicted radionuclide emissions that are one-third to one-tenth as great as in the 1989 NESHAPS study. These earlier findings were, in themselves, considered to represent insignificant health risks.

CAP93-PC Model. EPA replaced the primary exposure and dose model used in the NESHAPS study, the CAP88-PC model, with the CAP93-PC model, which corrects the calculation of wet deposition, and estimates somewhat lower MEI risks and substantially lower population risks.

9.2 Summary of Risk Results and Insights

9.2.1 Population Inhalation Risks and Health Effects

The base case scenario (see Section 7) provided the following results concerning population impacts from the inhalation of utility-emitted trace substances:

- For the total population (living within 50 km of a power plant), the estimated annual cancer incidence due to the inhalation of trace substances emitted from electric utility steam generating units is 0.09 cancer incidences per year. This estimate includes exposure by individuals to emissions from multiple power plants, and assumes exposures occur 24 hours per day for 70 years. The units assessed include existing and announced units plus future capacity for which particular sites cannot now be designated.
- Coal-fired plants with relatively large populations in 50 kilometers tend to have the highest inhalation population risks, relative to other plants. However, the population risk is very small for all plants.
- Inhalation hazard indexes (characterizing potential noncarcinogenic risks due to trace substance emissions) for all individuals in the United States are less than 1 for all analyzed substances from power plants. This result suggests that there are no inhalation-pathway noncarcinogenic health risks associated with emissions of these substances.

9.2.2 Individual Inhalation Carcinogenic Risks

The base case scenario provided the following results about individual cancer risks:

- For the roughly 600 power plants investigated, the expected increase in individual cancer risk, incorporating exposure assumptions associated with maximum exposure over a 70-year life span, did not exceed 1.7 in one million (1.7×10^{-6}). Out of this entire population of plants, only 3 plants, or 0.5%, approach exposures leading to a cancer risk greater than one in one million (1×10^{-6}) for a maximally exposed individual.

- The highest inhalation MEI risk is 1.7×10^{-6}, and corresponds to an uncontrolled oil-fired plant with extremely poor dispersion characteristics. For this plant, arsenic and chromium contribute 55% and 22% of the risk, respectively.
- Reflecting more reasonable exposure and plant replacement assumptions, the inhalation risks for the Reasonably Exposed Individual (REI) living and working outdoors in the populated area with the highest concentrations from the plant ranges from 2% to 19% of the MEI inhalation risks. The maximum REI risk for all plants is less than one in one million, and risks for all but 2 plants are less than one in ten million (1×10^{-7}).

9.2.3 Individual Inhalation Noncarcinogenic Risks

The base case scenario provided the following results and insights regarding non-cancer risks to individuals due to the inhalation of trace substance emissions from power plants:

- The highest inhalation MEI hazard quotient is 0.3, and corresponds hydrochloric acid emitted from to a bituminous coal-fired plant with particulate controls only.
- Reflecting more reasonable exposure and plant replacement assumptions, the inhalation hazard quotients for the REI living and working outdoors in the populated area with the highest concentration due to the plant's emissions ranges from 21% to 70% of the MEI inhalation hazard quotients.

9.2.4 Factors That Influence Inhalation Risk Estimates

A number of data and modeling factors influence the risk estimates. These factors include:

- type(s) of control equipment
- concentrations of trace substances in the fuel
- fuel-burning load
- particulate emissions
- emissions estimation procedure and data
- dispersion coefficients (i.e., urban or rural)
- source characteristics (e.g., stack height)
- the location and magnitude of nearby residential populations
- future scenarios of industry operations.

Based on extensive sensitivity analyses, no single group of plants (as defined by plant configuration, operating characteristics, stack height, or fuel type) could be identified as consistently correlated with relatively high risks compared to the distribution of risks across all plants. Rather, it was usually a unique combination of site- and plant-specific factors that led to higher relative risks for an individual plant.

Although variations in assumptions about future scenarios (e.g., load, fuel type, controls, etc.) can influence risk estimates for individual plants and relative risks across multiple plants, in the aggregate, the alternative scenarios did not significantly affect the risk estimates.

9.2.5 Multimedia Risk

- EPRI conducted screening case studies of four power plants with measured emissions to estimate carcinogenic and noncarcinogenic multimedia risks from power plant emissions using a multimedia risk assessment model, *TRUE* (Total Risk of Utility Emissions). The estimated maximum incremental cancer risk due to exposures through all pathways (i.e., inhalation, ingestion, dermal contact) was below one in one million (1×10^{-6}) for all plants studied. For noncarcinogens, the multimedia analysis also showed all exposure levels to be below the relevant threshold levels (RfDs and RfCs) for adverse effects.
- EPRI developed a methodology to carry out an uncertainty analysis and applied the methodology to two of the multimedia risk case study plants. At one power plan, a distribution was developed for multimedia carcinogenic risks associated with the plant emissions. At a second plant, noncarcinogenic risks associated with mercury emissions from the plant were examined in greater detail. In both cases, the results indicate that the assumptions made in the point-estimate multimedia risk assessments are highly conservative.

9.3 Summary

This report is intended to provide insight into the best data and methods available to estimate health risks due to trace emissions from fossil fuel-fired steam-electric units. To do this, EPRI conducted and integrated the results of a number of research projects aimed at generating more appropriate coal composition data, accurately measuring trace substance emissions from power plant stacks, developing sound methods to estimate emissions for the national capacity of power plants, developing scenarios of future industry operations, improving health effects data, and assessing reasonable measures of health risks. The data, methodologies, and analysis results presented in this report provide an understanding of risks associated with power plant trace emissions, and related issues that are consistent with the best data and methodologies currently available.

Although the research reported herein provides a considerable improvement over previously available data and methods, considerable uncertainties remain. As additional data become available, these results can be further refined. However, the data and analyses presented herein suggest that, following power plant compliance with provisions of the 1990 CAAA, trace substance emissions from electric utility steam generating units will not pose significant risks (either carcinogenic or noncarcinogenic) to human health.

GLOSSARY

Absorbed Dose	The amount of a substance (e.g., a chemical) that is assimilated into the body of an exposed organism.
ACGIH	American Conference of Governmental Industrial Hygienists
Acute Exposure	Exposure to an agent for 24 hours or less; a one-time or short-term, often high, exposure that may or may not cause a health effect.
Additive Effect	Refers to situations where the combined effect of two or more substances (e.g., two or more chemicals) is equal to the sum of their individual effects.
Additivity	The characteristic property of a mixture of toxicants that exhibits a total toxic effect equal to the arithmetic sum of the effects of the individual toxicants.
Adherence	The amount of soil, sediment, or other solid-phase material stuck to the skin that could be absorbed into the body, usually expressed as a surface density in mg/cm^2.
Ambient	Naturally-occurring background amounts of a substance in a particular environmental medium; may also refer to existing amounts in a medium regardless of source.
Analytical Sampling	Chemical or physical analyses of environmental samples, such as groundwater or effluent discharge samples.
ANOVA	Analysis of variance
ANSI	American National Standards Institute
Anthropogenic Sources	Sources of chemicals in the environment that are human-created, as opposed to sources that occur naturally in the environment. Can refer to either contemporaneously operating or historical sources.
Aqueous	Pertaining to a water solution.

Aquifer	An underground formation, group of formations, or part of a formation that contains sufficient saturated permeable material to yield economical quantities of water to wells and springs.
Aromatic	In organic chemistry, compounds that contain one or more benzene rings.
ASME	American Society of Mechanical Engineers
Assay	A test for a particular substance or effect.
ASTM	American Society for Testing and Materials
Atmospheric Dispersion	The turbulent diffusion of chemicals in the atmosphere due to atmospheric eddies.
Background Level	Normal ambient environmental concentration of a substance (e.g., a chemical). Also may be referred to as *Background Concentration.*
Bias	In experimental design, an inadequacy that leads to results or conclusions not representative of the population under study.
BIF	Boiler and Industrial Furnace Regulations
Bioaccumulation	The build-up of chemicals in living tissues through direct and food uptake.
Bioavailability	The state of being capable of being absorbed and available to interact with the metabolic and physiological processes of an organism.
Bioconcentration	The accumulation of a substance (e.g., a chemical) in tissues of an organism (such as fish) to levels that are greater than the level in the medium (such as water) to which the organism is exposed. See *Bioaccumulation.*
Bioconcentration Factor (BCF)	The ratio of the chemical concentration in an organism (such as fish) divided by the concentration in the surrounding medium (the water body).
BOOS	Burners out of service (a NOx reduction technique).
Brownian Motion	Random movement of small particles due to thermal motion in the carrying medium.

Btu	British thermal unit, the amount of energy required to raise the temperature of one pound of water one degree at 60°F.
CAAA	Clean Air Amendments of 1990
Cancer	The uncontrolled, invasive growth of malignant cells. Cancerous cells can metastasize, or break away from the original tumor, relocate, and grow elsewhere in the body.
Cancer Burden	The increased number of cancer cases above background cancers that have occurred in a population as a result of exposure to chemical substances (or other cancer-causing agents).
Cancer Potency Slope Factor (q_1^*)	An indication of a chemical's human cancer-causing potential derived using animal studies or epidemiological data on human exposure. It is based on extrapolating high-dose levels over short periods of time to low-dose levels and a lifetime exposure period through the use of a linear model.
Cancer Slope Factor (CSF)	Ratio of the probability that an individual will contract cancer as a result of lifetime exposure to chemicals, to the chemical dose thought to cause such cancer.
CAPCOA	California Air Pollution Control Officers Association
CARB	California Air Resources Board
Carcinogen	An agent capable of inducing cancer.
Carcinogenic	Producing or inciting a cancer response.
Carcinogenic Risk	The quantitative measure for evaluating the lifetime probability of contracting cancer from exposure to carcinogenic chemicals.

Carcinogens, EPA Classification of	Category/Criterion *A:* Human carcinogen, with sufficient evidence from epidemiological studies. *B1:* Probable human carcinogen, with limited evidence from epidemiological studies. *B2:* Probable human carcinogen, with sufficient evidence from animal studies and inadequate evidence or no data from epidemiological studies. *C:* Possible human carcinogen, with limited evidence from animal studies in the absence of human data.
Chemical Transformation	Changes in the valence state or chemical compound of a substance due to reaction.
Chronic	Of long duration. Chronic exposure usually refers to long-term, low-level exposure. Chronic toxicity refers to the effects produced by such exposure. Chronic exposure may cause latent damage that does not appear until later.
Chronic Exposure	An exposure to an agent for 90 days or more; a long-term exposure often for a major portion of the lifetime usually to a relatively low concentration that may or may not cause a health effect.
4	4
Compound	A substance formed by the union of two or more elements.
Concentration	The quantity of a substance per unit volume or weight. Examples: amount of a chemical in drinking water or air; amount of toxicant relative to an organism (for example, amount per unit of blood volume).
Confidence Interval	Specifies a range and the probability that an uncertain quantity falls within this range if the process of measuring the values is repeated some number of times. Expressed as a percent confidence interval.
Congeners	Of the same kind; i.e., all dioxin and furan compounds that contain chlorine in the 2, 3, 7, and 8 positions.

Correlation Coefficient	A number between 0 and 1 that indicates the degree of correlation between two data sets. 0 indicates no correlation, 1 indicates a perfect positive correlation.
Critical Parameters	Influential parameters that also have significant uncertainties. Such parameters should be considered in a model uncertainty analysis.
Cumulative Exposure	The summation of exposures of an organism to a substance (e.g., a chemical) over a period of time.
Cumulative Probability Distribution	A curve or mathematical expression that yields the probability that a variable's outcome will be less than or equal to any specific value.
CVAAS	Cold Vapor Atomic Absorption Spectroscopy
Decay	The first-order conversion reaction rate of a chemical.
Demography	The study of the characteristics of human populations, such as size, growth, density, distribution, and vital statistics.
Deposition	The process by which airborne chemicals are deposited on surfaces (ground, plants, buildings, etc.) and thereby removed from the atmosphere; the process by which particles are deposited in different portions of the lung according to their mass median aerodynamic diameters (based on particle size and density).
Dermal	Pertaining to the skin.
Dermal Contact or Exposure	Contact between a chemical and the skin.
Detection Limit	A level of chemical concentration below which the particular sampling and/or analytical method cannot detect; the minimum concentration at which a chemical may be identified, depending on the chemical, procedure, and equipment.
Deterministic Analysis	An analysis that is based on specific values of input parameters and consequently provides a single value (so-called point estimate) of the output variable.
DGAAS	Double Gold Amalgam\Cold Vapor Atomic Absorption Spectroscopy

Dispersion — In air or water modeling, the process of turbulent diffusion of an emission flow stream after it has left a source, resulting in lower plume concentrations (typically over a broader area) farther from the emission source.

DOE — U.S. Department of Energy

Dose — (1) A quantitative measure of the amount of chemical intake of exposed individuals. It is generally expressed as the mass of chemical per unit weight of an individual per unit time (e.g., mg/(kg-day), and, consequently, should be referred to as a dose rate.

(2) The quantitative amount of a chemical agent that reflects the magnitude of exposure modified by a series of intervening processes including inhalation or ingestion, transfer across membranes and uptake by tissues.

(3) The quantity of a chemical (amount administered, absorbed, or believed to be effective) to which an organism is exposed via ingestion, inhalation, or dermal contact.

Dose-Response — The quantitative relationship between the dose of a chemical and the probability of an effect caused by the chemical.

Dose-Response Assessment — A component of risk assessment that describes the quantitative relationship between the amount of exposure to a substance and the resulting biological effect; it may include the likelihood and extent of injury or disease.

Dose-Response Curve — A graphical representation of the quantitative relationship between the dose of a chemical or agent, and a specific biological response to that agent.

Dose-Response Functions — Dose-response functions specify the fraction of a population that will incur a biological effect (acute or chronic), given a specific dose. A variety of functions are commonly used to represent the relationship between dose and effects, including: threshold, linear, one-hit, multistage, and logit. The unit risk is a special case of a linear dose-response function, where the dose is specified as the lifetime cumulative (70-year) dose, and the response curve is linear through the origin (i.e., no threshold).

DQO — Data Quality Objective

Dry Deposition	The transport of chemicals from the atmosphere to the earth's surface through dry processes such as gravitational settling and interaction with terrain features.
Duration	Length of time over which an exposure is incurred, or a substance is released from a source.
Effluent	Treated or untreated waste material discharged into the environment. Generally refers to water pollution.
Electrostatic Precipitator (ESP)	A control device installed between the boiler and the stack to reduce the emissions of particulate fly ash from flue gas by means of electrically charged fields.
Emission	The release of contamination from a source. May be quantified in terms of volume or mass flow rate or concentration of hazardous material in a flow stream.
Emission Control Equipment	Device used to lower chemical emissions from facilities.
Emission Factor	The amount of a trace substance emitted from a specific unit, expressed in terms of mass per unit of energy input to the boiler (i.e., pounds per trillion, or lb/1012, Btu)
Empirical	Originating in or based on observation or experience.
Endpoint	A biological effect used as an index of the effect of a chemical on an organism, e.g., carcinogenicity, mutagenicity, neurotoxicity, etc.
Environmental Fate	The destiny of a substance (e.g., a chemical) after release to the environment. Involves considerations such as transport through air, soil, and water; bioaccumulation; and degradation.
Environmental Risk Analysis	Encompasses both risk assessment and risk management.
EPA	U.S. Environmental Protection Agency
Epidemiological Studies	Statistical studies that investigate elements contributing to a disease or toxic effects in human populations.
Epidemiology	The study of patterns of disease in groups of people.
EPRI	Electric Power Research Institute

Equivalency Factors — Relative factors for expressing congener or homologue toxicity effects for a specific substance.

Expected Value — Probability-weighted average of an uncertain variable or outcome, calculated by weighing each possible outcome value by its probability of occurrence and then summing across all possible outcomes.

Exposure — (1) Initial contact of an agent with a biological entity of interest, e.g., skin, lungs, etc.

(2) Contact between a receptor organism and the substance of concern. In general, human exposure to environmental pollutants can occur through three different physiological processes or routes: dermal penetration, ingestion, and inhalation.

Exposure Assessment — The determination or estimation (qualitative or quantitative) of the magnitude, frequency, duration, route, and extent (number of people) of exposure to a substance.

Exposure Concentration — The concentration of a chemical in its transport or carrier medium at the point of contact.

Exposure Pathway — The course a chemical or physical agent takes from a source to an exposed organism. Each pathway includes a source or release from a source, an exposure point, and an exposure route.

Exposure Point — A geographical location of potential contact between an organism and a chemical agent or physical agent.

Exposure Scenario — A set of conditions or assumptions about sources, exposure pathways, concentrations of toxic chemicals, and populations (numbers, characteristics, and habits) that aid the investigator in evaluating and quantifying exposure in a given situation.

Extrapolation — (1) An estimation of the numerical value of an empirical function at a point outside the range of data that establishes the function.

(2) The estimation of an unknown value by extending or projecting from known values.

Fabric Filter (Baghouse)	A particulate matter control device that uses filter bags to remove fly ash from flue gas
Fate and Transport Modeling	A mathematical process for simulating the behavior of contaminants in various environments to predict contaminant concentration and mobility. Models range from relatively simple analytical solutions to complex numerical models.
FCEM	Field Chemical Emissions Monitoring; EPRI Research Program to conduct field studies to characterize trace substance emissions.
Flue Gas Desulfurization (FGD)	The processes used to remove SO2 from flue gas.
Foliar Uptake	Plants' absorption of chemicals through their leaves.
Frequency	In exposure modeling, the number of exposure events per time period (e.g., per day, year, or lifetime).
Gas	A substance that is in a vapor state (above its vapor point) under normal temperature and pressure.
GC/HRMS	Gas Chromatography/High Resolution Mass Spectrometry
GC/MS	Gas Chromatography/Mass spectrometry
Geometric mean	The nth root of the product on n numbers, e.g., the average value obtained from the logarithms of a distribution, expressed as a base10 number.
GFAAS	Graphite Furnace Atomic Absorption Spectroscopy
Gravitational Settling	Deposition of chemicals onto the earth's surface as a result of gravity.
Hazard Index	The sum of all hazard quotients over several exposure pathways and/or several chemicals.

Hazard Quotient	The ratio of an exposure dose to either the appropriate reference dose. A quantitative measure for evaluating non-cancer health effects due to exposure to a single chemical through a single pathway. It is mathematically defined as the ratio of the exposure dose to a threshold dose — the reference dose or concentration (RfD of RfC), acceptable intake chronic (AIC), or acceptable intake subchronic (AIS) value) — or the ratio of the exposure concentration to a reference concentration.
Hazardous Air Pollutants (HAPs)	Any of 189 compounds listed in Title III of the Clean Air Amendments of 1990.
Health Hazard	*Acute:* Immediate toxic effects *Chronic*: Persistent or prolonged injury *Delayed*: Toxic effect occurring after a lapse of time
Health Risk	The likelihood of harmful health effects from exposure to chemicals.
HEAST	Health Effects Assessment Summary Tables, U.S. EPA
Hexavalent Chromium	Chromium compound that is in the oxidation state of six (Cr(VI)). It is considered to be the most toxic form of the metal because of its carcinogenicity.
HGAAS	Hydride Generation Atomic Absorption Spectroscopy
HHV	Higher Heating Value
HPLC	High Performance Liquid Chromatography
Human Health Risk	The likelihood (or probability) that a given exposure or series of exposures may have damaged or will damage the health of individuals experiencing the exposures.
Hydrological Balance	A budget that accounts, quantitatively, for the input, transport, and output of water in a locale.
IC	Ion Chromatography
ICPES	Inductively Coupled Plasma Emissions Spectroscopy
INAA	Instrumental Neutron Activation Analysis

Incidence (of Disease)	The number of new cases of a disease, usually expressed as an incidence rate—the number of new cases occurring in a population during a specified time period divided by the number of persons exposed to the disease during that period.
Independence (or *Probabilistic Independence*)	The relationship between two or more events when knowledge that one event has occurred does not alter the probability that the other event will occur. For uncertain quantities (random variables), knowledge of the outcome for one does not alter the probability distribution for the other.
Individual Lifetime Risk	The estimated incremental lifetime risk of an adverse effect incurred by an individual owing to exposure to a specified concentration of risk agent for a given time period (usually 70 or 75 years for lifetime risk).
Influential Parameters	Input parameters to which model outputs are most sensitive. These parameters are identified through a sensitivity analysis.
Ingestion	Type of exposure through liquids or solids entering via the body the mouth, as with food or beverages.
Inhalation	Type of exposure through gases or aerosols entering the nose or mouth, as in breathing.
Intake	(1) The process by which a substance crosses the outer boundary of an organism. (2) Amount of material inhaled, absorbed through the skin, or ingested during a specified period of time.
Intermedia Transport	Transport of chemicals between different media (e.g., between the atmosphere and the soil).
IRIS	Integrated Risk Information System, U.S. EPA
Latin Hypercube	In a probabilistic analysis, a sampling method that divides a parameter's probability distribution into intervals of equal probability. A random number is then generated for each interval.
LCI	Lower Confidence Interval
LCS	Laboratory Control Sample

Lifetime Exposure	Total amount of exposure to a substance that a human would receive in a lifetime (usually assumed to be 70 years).
Likelihood	Statistical probability that an event (such as harm or injury) may occur as a result of exposure to a risk agent.
Linear Relationship	Straight-line. When the statistical relationship between two variables increases on a direct unit-for-unit basis, this relationship, when plotted on a chart, will form a straight line.
Linearized Multistage Procedure	A sequence of steps in which (a) the multistage statistical model is fitted to tumor incidence data, (b) the maximum linear term consistent with the data is estimated, (c) the low-dose slope of the dose-response function is equated to the coefficient of the maximum linear term, and (d) the resulting slope is then equated to the upper bound of potency. This is the default procedure recommended by the U.S. EPA to estimate cancer risks from animal studies.
LNB	Low NOx Burner (a NOx control device)
Lognormal Distribution	A data set that exhibits a normal distribution when the logarithms of the data are plotted.
Low NOx Burners	Burners specifically designed to minimize the formation of nitrogen oxides.
Lowest Observed Adverse Effect Level (LOAEL)	The lowest dose in an experimental study at which a statistically or biologically significant adverse effect was observed.
Maximally Exposed Individual (MEI)	(1) An individual, sometimes hypothetically placed, who resides at the location where the highest health risk occurs. (2) A Maximally Exposed Individual is the person most at risk due to chemical release or contamination. Often the MEI is hypothetical. Calculation of the risk posed to the MEI is very conservative and assumes that the persons activities occur at the point of maximum concentration.
MDL	Method Detection Limit
Medium	A material or substance that serves as the transport matrix for a contaminant (e.g., surface soil, groundwater, indoor air).

Mercury Cycling Model (MCM)	A mathematical model that simulates the transformations and food chain uptake of mercury in an aquatic environment.
Methylation	A chemical process during which methyl radicals become attached to an atom, in this case mercury, or arsenic.
Model Parameterization	A method of keeping certain variables in a mathematical model constant in order to reduce the magnitude of the computations.
Modeling	Use of mathematical equations to simulate and predict real events and processes.
Modifying Factor	An uncertainty factor, greater than zero and less than or equal to 10; its magnitude reflects professional judgment regarding aspects of the data used for the assessment.
Monte Carlo Analysis or Simulation	A simulation modeling technique in which outcomes of events or variables are determined by selecting random numbers, subject to a defined probability law, from within a given range to predict the probability distributions of risk outcomes.
Multimedia	More than one environmental media (e.g., atmosphere, soil, and surface water).
MWe	Plant output, expressed in terms of electrical megawatts.
NAAQS	National Ambient Air Quality Standard
Neurotoxic	Exerting a destructive or poisonous effect on nerve tissue or nervous system.
Neurotoxin	A chemical that may lead to harmful health effects to one's neurological system.
Nm^3	Dry Normal Cubic Meter (0^o C, 1 atm)
No Observed Adverse Effect Level (NOAEL)	The highest experimental dose at which there was no statistically significant increase in any monitored adverse health or physiological endpoint.
Non-threshold Effects	Associated with exposure to chemicals that have no safe exposure levels (i.e., cancer).

Noncarcinogenic Risk	The non-cancer health effects resulting from exposure to chemicals. It is often expressed quantitatively by a hazard quotient or a hazard index.
Normal Distribution	The Gaussian bell curve distribution for probability density in which the x axis scale is linear.
OFA	Overfire Air (a NOx reduction technique).
PAH	Polynuclear Aromatic Hydrocarbon
Pathway	The means through which one is exposed to chemicals (e.g., inhalation of contaminated air, ingestion of contaminated food, skin contact with a contaminant).
PCDD	Polychlorinated Dibenzodioxins
PCDF	Polychlorinated Dibenzofurans
pCi	picoCurie, a unit of measure of nuclei disintegration equal to 130 events per hour.
PEL	Permissible Exposure Level, Occupational Safety and Health Administration (OSHA)
Pharmacokinetics	The study of the time course of absorption, distribution, metabolism, and excretion of a foreign substance in an organism.
Physiologically Based Pharmacokinetic Model (PBPK)	A mathematical model used to describe the time relationship between exposure to a chemical, and absorption, distribution, metabolism, and excretion using species specific physiological parameters.
PISCES	Power Plant Integrated Systems: Chemical Emission Studies; EPRI research program to characterize the source, distribution, and fate of trace substances in power plants.
Plume	(1) The volume of an environmental medium which encompasses the chemicals released from a source. (2) An emission flow stream in the ambient atmosphere or water body beyond the exhaust discharge point; alternatively, the bounded volume of the emission flow stream considered over a specified time interval.
Point Source	A stationary location where pollutants are discharged, usually by an industrial facility.

Polycyclic	In chemistry, pertaining to organic compounds whose molecules contain more than one circular structure (ring).
Population Risk	An estimate of the increase in the number of cancer cases (or other effects) in a population as a result of exposure to a toxic chemical.
Potency	The level of toxicity of a substance, normally a measure of the amount of the substance necessary to produce a given effect or risk.
Potential Carcinogen	Any substance, or combination of substances, which causes an increased incidence of benign and/or malignant neoplasms or a decrease in the latency period between exposure and onset of neoplasms in humans or in experimental animals. This definition also includes any substance which is metabolized into one or more potential carcinogens by mammals.
pound/trillion Btu	Common emission factor units (one pound of substance emitted per trillion, or 10^{12}, Btus of heat input).
ppm	Parts per million. A unit of measure of concentration. Measures the ratio of the mass (or volume in the case of air contamination) of a contaminant media (e.g., soil, water, or air). One (1) ppb of benzene in water would indicate that every kg of water would contain 1 mg of benzene.
ppmw	Part per million by weight, equivalent to one milligram per kilogram.
Probability	The likelihood of an event occurring expressed as a fraction (or decimal equivalent) between zero and one where an event with a probability of zero will not occur and an event with a probability of one will certainly occur.
Probability Density Function (PDF)	A function that describes the likelihood of all possible values for a variable. The value of the function is proportional to the likelihood of the input variable value. Also, the area under the function between defined limits represents the probability of the values within those limits.
Probability Distribution	A mathematical representation of the probabilities that a given variable will have various values.

Quality Assurance/ Quality Control (QA/QC)	A system of procedures, checks, audits, and corrective actions to ensure that technical activities are of the highest achievable quality. In risk assessments, technical activities include environmental monitoring and sampling as well as modeling, assumptions, and calculations.
Radionuclides	Isotopes of elements that spontaneously emit alpha, beta, or gamma particles
Reasonable Maximum Exposure (RME)	A Reasonable Maximum Exposure analysis of health risk includes a series of specific conservative assumptions for chemical concentration, exposure, and dose-response which lead to a very conservative estimate of risks. The Superfund process requires use of RME analyses for each identified receptor group.
Receptor	(1) In risk assessment, an organism (human, animal, or plant) that receives, may receive, or has received environmental exposure to a substance (e.g., a chemical). (2) In biochemistry, a specialized molecule in a cell that binds a specific chemical with high specificity and high affinity.
Reference Concentration (RfC)	Similar to the RfD. The RfC specifies an air concentration below which would not cause serious health risk, even with continuous exposure.
Reference Dose (RfD)	(1) An estimate of the daily exposure to the human population that is likely to be without appreciable risk of deleterious effect during a lifetime. (2) An estimate of the daily exposure, via ingestion or inhalation, to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effect during a lifetime. There are two estimates of daily exposure, chronic or subchronic. A chronic estimate is for a full lifetime, while subchronic refers to a portion of a lifetime. The RfD is expressed in units of mg/kg body wt/day.
Regression Analysis	A determination of an empirical relationship with two or more correlated variables using measured data for the purpose of estimating values of one variable when given values of other variables.

Respirable — Refers to material capable of being inhaled and small enough (in terms of effective size aerodynamically) to enter the bronchial system.

Respiratory System — All structures through which air enters the body; consists principally of nasopharynx and tracheo-bronchial region, and lungs.

Response Surface — A simplified version of the actual model which can be used to approximate the model results with considerably less computation (see Model Parameterization).

Risk — The potential for adverse consequences (i.e., injury, damage, or loss) from an event or activity. Risk has two components: probability (i.e., how likely it is that an adverse incident will occur) and severity (i.e., the magnitude of the consequences if such an incident does occur). Risk is typically expressed as the damage likely to occur in a given time period as a result of certain events or activities.

Risk Analysis — Comprised of the full range of scientific and policy evaluation activities included in the risk assessment, risk management, and risk communication processes.

Risk Assessment — (1) The scientific procedure in evaluating the toxic properties of a chemical and the condition of human exposure to it in order to both ascertain the likelihood that exposed humans will be adversely affected, and to characterize the nature of the effects they may experience.

(2) The qualitative or quantitative evaluation of the environmental, health, and/or operational risks that may result from exposure to a pollutant or activity. It combines exposure assessment results with toxicity assessment results to characterize risks.

Risk Characterization — The final phase of the risk-assessment process that involves integration of the data and analysis involved in hazard identification, source/release assessment, exposure assessment, and dose-response assessment to estimate the nature and likelihood of adverse effects.

Risk Estimate — A description of the probability that organisms exposed to a specified dose of a substance (e.g., chemical) will develop an adverse response (for example, cancer).

Root Uptake	Plants' absorption of chemicals through their root system.
Route	The way a chemical or pollutant enters an organism after exposure, e.g., by ingestion, inhalation or dermal absorption.
RPD	Relative Percent Difference
RSD	Relative Standard Deviation
Sampling	Selecting a portion of a group of data in order to determine the accuracy or propriety or other characteristics of the whole body of data.
Saturated Zone	The zone in a soil column below the water table under which soil is saturated with water.
Scavenging Rate	The rate of removal of a chemical by precipitation.
Sensitivity Analysis	A method used to examine the behavior of a model by measuring the variation in its outputs resulting from changes to its inputs.
Sensitivity Index	The ratio of the change in the model output to the perturbation in an input parameter.
SIE	Specific Ion Electrode
Simulation Model	Mathematical model that simulates physical, chemical, and biological processes.
Slope Factor	(1) The slope of the upper bound dose extrapolation model at doses approaching zero. (Also called cancer potency factor.) (2) The slope of the upper-bound dose-response extrapolation model at low doses. The slope factor may also refer to slopes calculated by alternative models. Current slope factors are available in EPA's on-line toxicological database, the Integrated Risk Information System (IRIS) or in EPA's Health Effects Assessment Summary Tables (HEAST)
Source Term	A quantitative measure of emissions of materials at the source of release.

Source/Release Assessment — A step in risk assessment that estimates the amounts, frequencies, and locations of the introduction, release, or escape of risk agents into occupational, residential, or outdoor environments.

Standard — Any established measure of extent, quantity, quality or value. Any type, model, or example for comparison; a criterion of excellence.

Statistically Significant — Experimental results that are "not likely" to have occurred by chance. "Significant with 0.05 probability" means there is only a 5 percent probability that the results were attributable to chance and a 95 percent probability that the results were attributable to the experiment.

Student- t test — A probability density function used to test the equivalence of data sets.

Substance — Refers to chemicals and other external, non-living sources of potential hazard, such as ionizing radiation and microwaves.

Synthetic Simulations — Simulations using specified probability distributions of parameters to generate a set of synthetic model results. Synthetic simulations represent the effect of various combinations of possible input parameter values on the output of a model.

Threshold — (1) The lowest dose (exposure) at which a defined biological effect occurs.

(2) Refers to the lowest dose of a chemical at which a specified measurable effect is observed and below which it is not observed.

TLV — Threshold Limit Value, American Conference of Governmental Industrial Hygienists

Toxic — Pertaining to, due to the nature of the poison.

Toxicity — The quality or degree of being poisonous or harmful to plant, animal, or human life.

Toxics — Those pollutants that have a toxic effect on living organisms. The CWA Section 307(a) "priority" pollutants are a subset of this group of pollutants.

Transport	Movement of contaminants through the environment, including air, groundwater, and surface water media.
UARG	Utility Air Regulatory Group, a membership group of electric utilities and utility industry organizations.
UCI	Upper Confidence Interval
Uncertainty Analysis	An analysis that relates the uncertainty in input variable(s) to the resulting uncertainty in the output variable.
Uncertainty Factor	The factor(s) used to derive the reference dose (RfD) from experimental data to account for the variation in sensitivity among humans, the uncertainty in extrapolating animal data to humans, and the uncertainty in extrapolating from data obtained in a study that is of less that lifetime exposure.
Uncertainty Index	Measure of the uncertainty associated with a parameter. The uncertainty index is sometimes defined as the ratio of the standard deviation of the parameter distribution to the mean value of the parameter.
Unit Risk	(1) The incremental upper bound lifetime risk estimated to result from lifetime exposure to an chemical agent at a concentration of 1 microgram per cubic meter. (2) (Also known as the Unit Cancer Risk) The unit risk provides a quantitative estimate of carcinogenic potency expressed as the incremental chance of contracting cancer from a 70-year lifetime exposure to a concentration of 1 $\mu g/m^3$ of a given substance. Unit risk values published by EPA's Carcinogenic Assessment Group (CAG) are intended to yield "plausible upper-bound" (based on a 95% upper confidence level) estimates of risk at low doses.
Upper-Bound Exposure (Dose) Estimate	A plausible estimate of individual exposure or dose for those persons at the upper end of an exposure or dose distribution, conceptually above the 90th percentile, but not higher than the individual in the population who has the highest exposures or dose.
Upper-Bound Estimate	Estimate not likely to be lower than the true risk.

Vadose Zone	The soil zone containing water at levels below saturation. The zone is limited above by the land surface and below by the surface of the zone of saturation, that is, the water table. This zone is also referred to as the unsaturated zone.
Vapor	Visible, fine particles of a liquid suspended in air, or the gaseous phase of something that is usually a liquid or a solid.
Volatile Organic Chemical (VOC)	Any of a group of easily vaporizable compounds. VOCs often refers to the group of light aromatics made up of benzene, toluene, xylene, and ethylbenzene.
VOST	Volatile Organic Sampling Train
Washout Coefficient	The fraction of chemical removed per unit time due to precipitation. Also referred to as the scavenging coefficient.
Water Table	The surface between the vadose zone and the groundwater.
Weathering Elimination Rate	The rate of removal of chemicals from surfaces due to weathering phenomena (e.g., precipitation).
Wet Deposition	The removal and subsequent deposition of chemicals from the atmosphere to the earth's surface by precipitation, fog, and cloud droplets.
XRF	X-ray Fluorescence

Electric Utility Trace Substances Synthesis Report

Volume 2: Appendices A through N

TR-104614-V2

Research Project 3081
November 1994

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FIGURES

TABLES

APPENDIX A
SAMPLING AND ANALYTICAL METHODS

A.1 Details of the Sampling and Analytical Methods

Fuels. Figure A-1 shows a schematic of the general preparation and analytical steps for fuels.

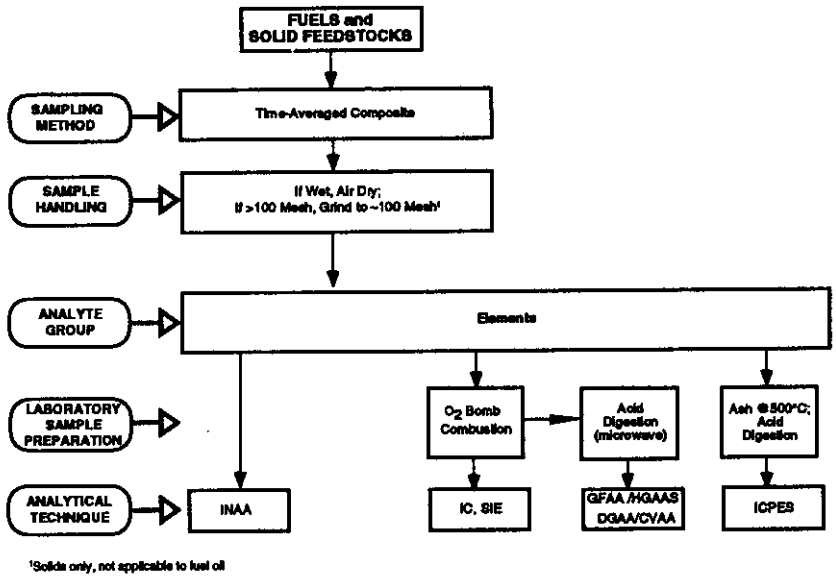

Figure A-1.
Overview Flow Chart of Current Sampling and Analytical Methods for Fuels and Solid Feedstocks

Flue Gases. A schematic of the general treatment of flue gas samples is shown in Figure A-2. Four primary classes of hazardous air pollutants are analyzed in flue gas, each requiring separate sample collection and preparation steps. The four classes are:

- Inorganic analytes
- Volatile organic compounds
- Semivolatile organic compounds [polychlorinated dibenzodioxins and dibenzofurans (PCDD/PCDF) are included under semivolatile organics]
- Aldehydes

Chlorine and fluorine, which occur in the flue gas stream predominantly as the acid gases HCl and HF, are included in the class of inorganic analytes. Most of the sample collection techniques used for gas streams were standard reference methods designed for general combustion sources. Some modifications to the standard methods are required, in particular, to deal with the high particulate loadings at some locations in coal-fired power plants. Sampling and analytical techniques for each of the four pollutant classes present in flue gases are discussed separately.

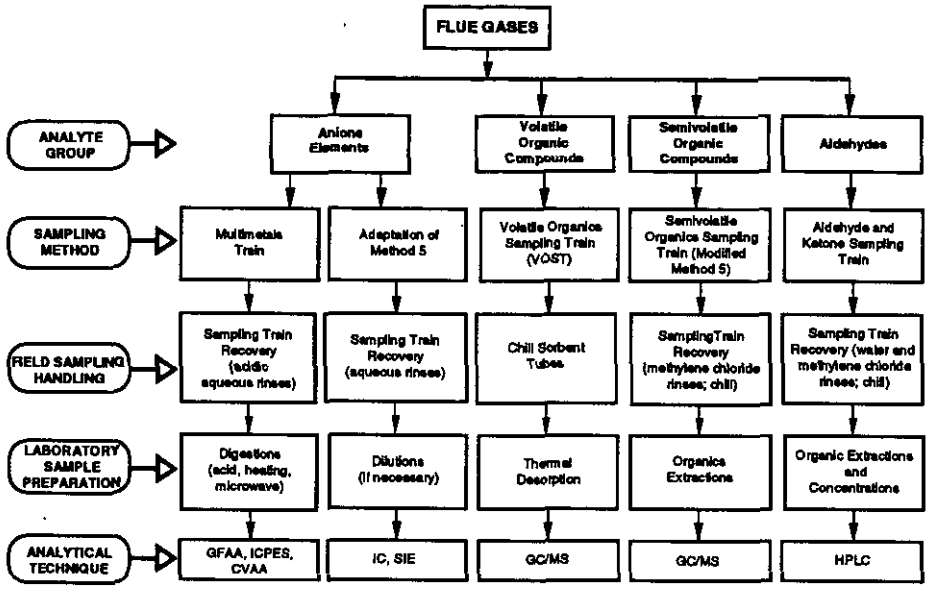

Figure A-2.
Overview Flow Chart of Current Sampling and Analytical Methods for Flue Gases

Inorganic Analytes. Inorganic analytes in flue gases were collected using EPA's "Methodology for the Determination of Metals Emissions in Exhaust Gases from Hazardous Waste Incinerators and Similar Combustion Processes" [1]. Samples were collected isokinetically (at the same flow rate as the gas stream), filtered, and passed through a series of four impingers containing strong acid reagents designed to oxidize vapor-phase metals and maintain them in solution. Sample recovery involved separate rinsing, handling and digestion procedures for each component subsystem of the sampling train. Spectrophotometric analysis of these samples provided metals concentrations for both particulate and vapor phases.

Acid gas components of the flue gases were collected in a separate sampling train using a hydrogen peroxide solution buffered with a mixture of sodium carbonate and bicarbonate. The sampling train used was an adaptation of EPA Method 5 [2], in which isokinetic samples were filtered and the carbonate-buffered impinger solutions collected acid gases as ionic species suitable for analysis by ion chromatography and potentiometry. Hydrogen chloride, oxides of sulfur, and hydrogen fluoride were analyzed as chloride, sulfate,

and fluoride ions, respectively by EPA Methods 300.0 [3] and 340.2 [4]. Phosphate was analyzed directly by ICP-AES in the nitric acid impinger solutions from the multi-metals sampling train.

Mercury Speciation. As has been mentioned in Chapter 2, a solid sorbent method that has been under development by Frontier Geosciences was used to determine mercury speciation (i.e., elemental mercury, ionic mercury, and methylmercury). In the Frontier Geosciences method [5], flue gas is drawn non-isokinetically through a heated sampling system (95-100°C) comprised of KCl/soda-lime traps followed by iodated carbon traps. This method does not attempt to measure particulate mercury. The vapor phase mercury (total and elemental) collected on iodated carbon traps is determined by SnCl2 reduction of small aliquots of acid digests, purging and pre-concentration on gold, followed by cold vapor atomic fluorescence spectrometry (CVAFS) detection. To obtain chemical speciation information, soda-lime traps are first dissolved in acetic acid. Ionic mercury and methylmercury are determined by aqueous phase ethylation, purging onto carbon trap, cryogenic GC separation, and CVAFS detection. Methylmercury is determined as methylethyl mercury, while ionic mercury is determined as diethyl mercury. Recently, questions have arisen regarding the accuracy of methyl mercury measurements by this method. It appears that methyl mercury can be produced during the acetic acid digestion of soda lime, giving rise to 'false positive' detection of methyl mercury. This is apparently caused by reaction of acetic acid with SO3—in the flue gas that forms methyl groups which, in turn, react with Hg (II). Work is currently underway to investigate this phenomenon, and to modify the method for more accurate methyl mercury measurements in flue gas.

Volatile Organic Compounds. Volatile organic compounds (VOCs) were collected from the vapor phase of the gas stream using EPA Method 0030 [6] volatile organic sampling train (VOST) or using a Tedlar bag (CARB Method 401A). The VOST train uses solid sorbents (Tenax and charcoal) to trap VOCs in the sample. The sorbent traps are recovered and kept chilled throughout transit and storage to minimize any potential loss of volatile organic compounds before thermal desorption and analysis by gas chromatography/mass spectrometry (GC/MS) using EPA method 5041 [7]. Method modifications are not necessary for the current application of these methods to utility power plant flue gases. However, these methods are not well applied to ESP inlet flue gases. This is due primarily to inefficient gas collection on the resin.

Semivolatile Organic Compounds. Semivolatile organic compounds were collected using EPA Method 0010, with a Modified Method 5 (MM5) Sampling Train [8]. The method specifies isokinetic collection of particulate matter and vaporous organic compounds in a Method 5 collection train consisting of a particulate filter and sorbent resin. An XAD-2 resin was used to capture organic compounds penetrating the filter. The gas was chilled before it contacted the resin, which removed organic compounds from the cooled gas and from the aqueous condensate penetrating the resin. This sampling method requires no modifications for utility emissions monitoring. The resins were retained for analysis and the collection train components were rinsed with methylene chloride to

recover any organic material they may have trapped. The filtered solids, XAD-2 resin sorbent, and rinses were chilled to 4°C until extraction and analysis by GC/MS using EPA Method 8270 or by GC/HRMS. This method allowed quantitation at lower levels. Alternately, CARB Method 429 was used to collect and analyze semivolatile compounds (primarily at the oil-fired sites). CARB 429 uses the same resin and extraction procedures as Method 8270. Detection and identification is made by HRGC/MS which is 100 times more sensitive than GC/MS used by 8270. A slightly different compound target list is used in CARB 429. CARB 429 uses deuterated internal standards for mass resolution and quantitation.

Aldehydes. Aldehydes were collected from the flue gas using EPA Method 0011 [9]. This method was applied to both aldehyde and ketone emissions and used an impinger solution of acidic dinitrophenylhydrazine (DNPH—to convert these compounds into a stable, water insoluble form). The DNPH derivative was extracted from the impinger solution in methylene chloride and subsequently analyzed by high-performance liquid chromatography (HPLC). No modifications were necessary for flue gas applications. Aldehydes are assumed to be present in the vapor phase, and samples have been collected non-isokinetically at a single point of average velocity.

Polychlorinated Dibenzodioxins and Dibenzofurans. Polychlorinated dibenzodioxins and dibenzofurans were measured using EPA Method 23 [10]. This sampling approach is very similar to Method 0011, discussed above. The recovery used was somewhat more aggressive, using more solvents. Final analysis was accomplished using GC/HRMS.

A.2 Derivation of Risk-Based Detection Limits

Even with application of the best sampling and analytical techniques, in some cases it may not be possible to provide the sensitivity required to perform meaningful risk assessment. Substances whose sampling and analysis yield concentrations below method detection limits are normally discarded from risk assessments. Table A-1 demonstrates that target detection limits can be substantially improved—that is, made more attainable with available methods—when site-specific environmental data are used for modeling source impacts. This approach uses a screening health risk assessment to determine appropriate detection limits prior to any field sampling program. The health risk assessment allows target detection limits to be calculated based on site and source characteristics and allows risks to be assessed with more sensitivity to conditions actually prevailing at a source and its site.

Table A-1.
Target Detection Limits[1] (μg/m³)—(Example for a Given Site)

Chemical	Screening Generic[2]	Refined Generic[3]	Refined Site-Specific[4]
Arsenic	1.3×10^{-2}	6.3×10^{-1}	5.1
Beryllium	4.1×10^{-3}	1.9×10^{-1}	4.9
Cadmium	9.6×10^{-1}	$4.5 \times 10^{+1}$	$4.5 \times 10^{+1}$
Chromium (Vi)	1.4×10^{-1}	6.8	6.8
Benzene	$2.1 \times 10^{+2}$	$9.8 \times 10^{+3}$	$1.0 \times 10^{+4}$
Formaldehyde	$1.3 \times 10^{+2}$	$6.2 \times 10^{+3}$	$6.2 \times 10^{+3}$
PAH (as B(a)P)	6.0×10^{-4}	2.8×10^{-2}	4.9×10^{-1}

1 Concentrations that would lead to a lifetime excess cancer risk of 10-7 if one-half the detection limit is used in risk assessment.

2 Used worst-case meteorology, worst-case multimedia factors.

3 Used site meteorology but worst-case multimedia factors.

4 Used site meteorology and site-specific multimedia factors. Target DLs changed only for chemicals with significant non-inhalation carcinogenicity (i.e., Be, As, benzene, formaldehyde, PAH).

The emission rate of a given chemical will in turn determine maximum ambient ground-level air concentrations via dependence on meteorology, terrain, and source characteristics. The use of appropriate exposure parameters in combination with a potency factor or reference dose allows the carcinogenic or noncarcinogenic health risk due to inhalation of the chemical to be calculated. For a chemical presenting health effects through non-inhalation pathways (e.g., ingestion), the calculation extends to alternative pathways.

Because chemical emissions from fossil fuel-fired power plants consist of a variety of compounds, the combined health effects from all compounds need to be considered for the determination of appropriate detection limits. The target detection limits are calculated such that, if none of the chemicals is detected, the total carcinogenic or chronic noncarcinogenic health risk will be less than the selected target.

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APPENDIX B
SITE TEST RESULTS: FIELD DATA PRESENTATION AND CORRELATIONS

Appendix Section B.1 presents a summary description of the EPRI FCEM and DOE field test sites. This includes a comparison of the recent field sites with the number of commercial units.

Section B.2 provides a more detailed presentation of the field data and analyses of the correlations and emission factors.

Sections B.3 through B.7 present the procedures used in analyzing the field test site data from the various plant configurations that have been characterized in the EPRI FCEM and DOE programs. In addition, graphical and tabular results of data analysis are presented.

Section B.8 discusses the effect of existing pollutant controls on trace substance emissions. The EPRI and DOE field results are used to assess the amount of trace substances removed in conjunction with criteria pollutant control.

B.1 Test Sites

B.1.1 Selection Rationale, Site Characteristics, and Measurements

In late 1989, the information in the PISCES database was evaluated and compared with the profile of the utility industry, by categorizing steam-electric power plants in terms of fuel type and emission controls. Some sixteen major groupings were developed. For example, bituminous coal-fired units with ESPs make up about 40% of the total fossil-fuel generating capacity. For many of these categories, candidate test sites were identified. A goal in the site selection process was to obtain a data set for the major configurations of fuel type and emission control.

The first set of field tests was conducted in 1990. As the program evolved, EPRI actively pursued additional sites in order to best characterize the U.S. electric utility industry. Most of the sites were chosen based on the host utilities' interest in participating in the FCEM project. In addition, some utilities conducted testing and provided their results to

EPRI to be included in the PISCES study. EPRI has also collaborated with the DOE Clean Coal Technology program. In 1992, DOE began the comprehensive site assessment project, which involved the characterization of eight commercial units.

Tables B-1 through B-3 present fuel, site, and measurement characteristics about the units that have been tested. All the data sources have site code numbers, including those tested by DOE.

Table B-1.
FCEM and Other Test Sites: Fuel Characteristics

Site #	Configuration	Fuel Characteristics				
		Source	Type	% Ash	% Sulfur	HHV
12	ESP/FGD	West Virginia	Coal-Bit	9.0%	2.8%	13,700
12	ESP	West Virginia	Coal-Bit	9.0%	2.8%	13,700
14	SD/FF	West Virginia	Coal-Bit	9.0%	2.8%	13,700
15	ESP	Appalachia	Coal-Bit	13.0%	1.5%	13,000
16	ESP	Virginia/Kentucky	Coal-Bit	10.0%	1.6%	13,700
16	ESP/LNB	Virginia/Kentucky	Coal-Bit	9.5%	1.7%	13,800
18	ESP	Virginia/Kentucky	Coal-Bit	13.0%	0.9%	13,400
18	ESP/pPJFF	Virginia/Kentucky	Coal-Bit	13.0%	0.9%	13,400
19	ESP	Virginia/Kentucky	Coal-Bit	9.0%	0.8%	13,500
21	ESP/FGD	West Virginia	Coal-Bit	6.0%	1.6%	14,000
110	ESP	Kentucky	Coal-Bit	9.0%	2.9%	11,900
110	ESP/LNB	Kentucky	Coal-Bit	9.0%	2.9%	12,000
114	ESP	Lamar, IN	Coal-Bit	9.0%	1.2%	11,090
114	ESP/Reburn	Lamar, IN	Coal-Bit	9.0%	1.2%	11,090
116	ESP	Ohio	Coal-Bit	12.0%	3.5%	13,200
116	pSNRB	Ohio	Coal-Bit	12.0%	3.5%	13,200
122	ESP	Illinois #5	Coal-Bit	9.3%	2.0%	13,400
125	ESP/FGD	Western Kentucky	Coal-Bit	14.0%	2.0%	13,000
DOE2	ESP	Ohio	Coal-Bit	12.0%	2.8%	12,900
DOE2	FF	Ohio	Coal-Bit	12.0%	2.8%	12,900

Table B-1.
FCEM and Other Test Sites: Fuel Characteristics (Continued)

Site #	Configuration	Fuel Characteristics				
		Source	Type	% Ash	% Sulfur	HHV
DOE3	ESP	Illinois	Coal-Bit	12.0%	3.4%	12,500
DOE4	ESP	Illinois #5 & #6	Coal-Bit	13.8%	3.3%	14,300
DOE4	ESP/FGD	Illinois #5 & #6	Coal-Bit	13.8%	3.3%	14,300
DOE5	ESP	Pitt. #8	Coal-Bit	11.4%	3.2%	13,100
20	ESP	Wilcox, Texas	Coal-Lig	21.0%	2.2%	10,000
20	ESP/FGD	Wilcox, Texas	Coal-Lig	21.0%	2.2%	10,000
DOE6	ESP	North Dakota	Coal-Lig	17.0%	1.0%	9,970
DOE6	ESP/FGD	North Dakota	Coal-Lig	17.0%	1.0%	9,970
10	FF	Salt River	Coal-Sub	21.0%	0.5%	11,000
11	ESP	Powder River	Coal-Sub	6.0%	0.4%	11,900
11	ESP/FGD	Powder River	Coal-Sub	6.0%	0.4%	11,900
22	ESP	Powder River	Coal-Sub	6.8%	0.4%	12,000
101	FF	New Mexico	Coal-Sub	25.0%	0.8%	11,000
101	FF/FGD	New Mexico	Coal-Sub	25.0%	0.8%	11,000
102	ESP	Powder River/Coke	Coal-Sub	9.0%	1.0%	12,200
111	SD/FF	Utah/Wyoming	Coal-Sub	14.0%	0.6%	10,020
115	FF	Western	Coal-Sub	10.0%	0.4%	12,600
115	FF/Urea	Western	Coal-Sub	10.0%	0.4%	12,600
DOE7	SD/FF	New Mexico	Coal-Sub	22.9%	0.7%	10,500
DOE8	FF	Powder River	Coal-Sub	11.2%	0.9%	11,700
103			Gas			
104			Gas			
105			Gas			
106			Gas			
107			Gas			
108			Gas			

Table B-1.
FCEM and Other Test Sites: Fuel Characteristics (Continued)

Site #	Configuration	Fuel Characteristics				
		Source	Type	% Ash	% Sulfur	HHV
109			Gas			
120			Gas			
121			Gas			
13	Uncontrolled		Oil	0.06%	0.3%	19,000
13	BOOS		Oil	0.06%	0.3%	19,000
13	pPJFF		Oil	0.06%	0.3%	19,000
103	Uncontrolled		Oil	NM	0.20%	19,200
104	Uncontrolled		Oil	0.03%	0.35%	18,800
105	Uncontrolled		Oil	0.25%	0.40%	19,000
106	Uncontrolled		Oil	0.02%	0.15%	19,000
107	Uncontrolled		Oil	0.02%	0.18%	19,200
108	Uncontrolled		Oil	0.05%	1.5%	18,300
109	Uncontrolled		Oil	NM	0.20%	18,900
112	Uncontrolled		Oil	0.03%	0.9%	18,600
112	ESP		Oil	0.03%	0.9%	18,600
117	Uncontrolled		Oil	0.09%	1.4%	18,600
117	pSCR		Oil	0.09%	1.4%	18,600
118	Uncontrolled		Oil	0.06%	0.75%	18,800
118	ESP		Oil	0.06%	0.75%	18,800
119	Uncontrolled		Oil	0.03%	0.9%	18,600
119	ESP		Oil	0.03%	0.9%	18,600

Table B-2.
FCEM and Other Test Sites: Site Configuration

Site No.	Configuration	Site Information							Start Year
		Boiler Type	MW	NO_x Control [a]	Particulate Control [a]	FGD Reagent	Absorber	Bypass	
12	ESP/FGD	Opposed	690	LNB	ESPc	Limestone	Open Tower	5%	1984
12	ESP	Opposed	690	LNB	ESPc				1984
14	SD/FF	Opposed	4	LNB	FFrg	Lime	Spray Dryer	0%	1988
15	ESP	Tangential	565		ESPc				1970
16	ESP	Front Wall	500	OFA	ESPc				1970
16	ESP/LNB	Front Wall	500	LNB/OFA	ESPc				1993
18	ESP	Tangential	350		ESPc/SO3				1965
18	ESP/pPJFF	Tangential	4		ESP/FFpj				1991
19	ESP	Opposed	1080		ESPc/SO3				1974
21	ESP/FGD	Opposed	4	LNB	ESPc	Limestone	Open Tower	0%	1988
110	ESP	Tangential	175		ESPh,c				1965
110	ESP/LNB	Tangential	175	LNB/OFA	ESPh,c				1992
114	ESP	Cyclone	114		ESP				1959
114	ESP/Reburn	Cyclone	114	Reburn	ESP				1992
116	ESP	Wall	63		ESPc				1944
116	pSNRB	Wall	5	pilot SCR	FFpj	H_2SO_4			1992
122	ESP	Cyclone	330		ESPc				1958
125	ESP/FGD	Cyclone	704		ESPc	Limestone	Venturi	0%	1963

Table B-2.
FCEM and Other Test Sites: Site Configuration (Continued)

Site No.	Configuration	Site Information							Start Year
		Boiler Type	MW	NO_x Control [a]	Particulate Control [a]	FGD Reagent	Absorber	Bypass	
DOE2	ESP	Cyclone	100		ESP				1954
DOE2	FF	Cyclone	35		FF				1954
DOE3	ESP	Cyclone	568		ESPc				1973
DOE4	ESP	Tangential	100		ESPc				1950
DOE4	ESP/FGD	Tangential	100		ESPc	Limestone	CT121	0%	1992
DOE5	ESP	Cell	615		ESPc				1967
20	ESP	Opposed	720	LNB	ESPc				1985
20	ESP/FGD	Opposed	720	LNB	ESPc	Limestone	Open Tower	5%	1985
DOE6	ESP	Tangential	550	OFA	ESP				1980
DOE6	ESP/FGD	Tangential	550	OFA	ESP	Lime			1980
10	FF	CFBC	110		FF shake	Limestone	CFB		1987
11	ESP	Tangential	720	OFA	ESPc				1980
11	ESP/FGD	Tangential	720	OFA	ESPc	Limestone	Open Tower	25%	1980
22	ESP	Wall Fired	700		ESPc				1982
101	FF	Front Wall	818	LNB	FFrg				1969
101	FF/FGD	Front Wall	818	LNB	FFrg	Lime	Open Tower	25%	1969
102	ESP	Cyclone	598		ESPc				1968
111	SD/FF	Opposed	290	LNB	FFrg	Lime	Spray Dryer	0%	1985
115	FF	Roof	100	LNB/OFA	FFrg	Ca,Na inj.	Fabric Filter	0%	1955

Table B-2.
FCEM and Other Test Sites: Site Configuration (Continued)

Site No.	Configuration	Site Information							Start Year
		Boiler Type	MW	NO_x Control [a]	Particulate Control [a]	FGD Reagent	Absorber	Bypass	
115	FF/Urea	Roof	100	LNB/OFA/ Urea	FFrg	Ca,Na inj.	Fabric Filter	0%	1955
DOE7	SD/FF	Tangential	425	LNB/OFA	FF	Lime	Spray Dryer	15%	1989
DOE8	FF	Wall	69		FF				1960
103		Front Wall	136	OFA/BOOS					1962
104		Opposed	345	OSF					1962
105		Opposed	750	OFA/FGR					1967
106		Opposed	495	OFA/FGR					1966
107		Face Fired	175	LNB/OFA					1955
108		Front Wall	54						1958
109		Opposed	230	BOOS					1965
120		Tangential	700	OFA/FGR/B					1972
121		Opposed	330	BOOS/OFA/FG					1965
13		Front Wall	376						1959
13	BOOS	Front Wall	376	BOOS					1959
13	pPJFF	Front Wall	4		FFpj				1991
103		Front Wall	136	OFA/BOOS					1962
104		Opposed	345	OSF					1962
105		Opposed	750	OFA/FGR					1967
106		Opposed	495	OFA/FGR					1966

Table B-2.
FCEM and Other Test Sites: Site Configuration (Continued)

Site No.	Configuration	Site Information							Start Year
		Boiler Type	MW	NO_x Control [a]	Particulate Control [a]	FGD Reagent	Absorber	Bypass	
107		Face Fired	175	LNB/OFA					1955
108		Front Wall	54						1958
109		Opposed	230	BOOS					1965
112	Uncontrolled	Tangential	387		ESPc				1967
112	ESP	Tangential	387		ESPc				1967
117	Uncontrolled	Front Wall	1						1975
117	pSCR	Front Wall	1	pilot SCR					1993
118	Uncontrolled	Front Wall	902	OFA/FGR					1980
118	ESP	Front Wall	902	OFA/FGR	ESPc				1980
119	Uncontrolled	Tangential	387						1967
119	ESP	Tangential	387		ESPc				1967

[a] Acronyms: LNB = Low NO_x Burners; OFA = Overfire Air; SCR = Selective Catalytic Reduction; BOOS = Burners out of service; OSF = Off-stoichiometric firing; FGR = Flue Gas Recirculation; LEA = Low Excess Air; ESPc = Cold-side ESP; FFrg = Reverse gas flow fabric filter; FFpj = Pulse Jet Fabric Filter

Table B–3.
FCEM and Other Test Sites: Measurements

Site No.	Configuration	Sampling Contractor	Test Period	Streams Sampled	Analyte Groups
12	ESP/FGD	Radian	Jul 90, Dec 92	Comprehensive[a]	Metals, Anions, VOST, Aldehydes, Semivols
12	ESP	Radian	Jul 90, Dec 92	Comprehensive	Metals, Anions, VOST, Aldehydes, Semivols
14	SD/FF	Radian	Jul 90	Comprehensive	Metals, Anions, Aldehydes, Semivols
15	ESP	Radian	Oct 90	Comprehensive	Metals, Anions, VOST, Aldehydes, Semivols
16	ESP	Radian	Mar 91	Comprehensive	Metals, Anions, VOST, Aldehydes, Semivols
16	ESP/LNB	Radian	May 93	Comprehensive	Metals, Anions, VOST, Aldehydes, Semivols
18	ESP	Radian	Feb 92	Coal, Stack	Metals, Anions
18	ESP/pPJFF	Radian	Feb 92	Coal, Gas In/Out	Metals, Anions
19	ESP	Radian	Mar 92	Coal, Gas In/Out	Metals, Anions
21	ESP/FGD	Radian	Aug 92	Coal, Gas In/Out	Metals, Anions, Semivols
110	ESP	SRI	Sep 91	Comprehensive	VOST, Semivols, Metals, Anions, Aldehydes
110	ESP/LNB	SRI	Jan 92	Comprehensive	VOST, Semivols, Metals, Anions, Aldehydes
114	ESP	Acurex	Nov 92	Comprehensive	Metals, Anions
114	ESP/Reburn	Acurex	Nov 92	Comprehensive	Metals, Anions
116	ESP	Battelle	May 93	Comprehensive	Metals, Anions
116	pSNRB	Battelle	May 93	Comprehensive	Metals, Anions
122	ESP	SRI	May 93	Coal, Stack	Metals
125	ESP/FGD	SRI	Aug 93	Coal, Stack	Metals, Anions

Table B–3.
FCEM and Other Test Sites: Measurements (Continued)

Site No.	Configuration	Sampling Contractor	Test Period	Streams Sampled	Analyte Groups
DOE 2	ESP	Battelle	Jul 93	Comprehensive	Comprehensive[b]
DOE 2	FF	Battelle	Jul 93	Comprehensive	Comprehensive
DOE 3	ESP	Weston	Jul 93	Comprehensive	Comprehensive
DOE 4	ESP	Radian	Jun 93	Comprehensive	Comprehensive
DOE 4	ESP/FGD	Radian	Jun 93	Comprehensive	Comprehensive
DOE 5	ESP	EER	Jun 93	Comprehensive	Comprehensive
20	ESP	Radian	Jun 93	Comprehensive	Metals, Anions
20	ESP/FGD	Radian	Jun 93	Comprehensive	Metals, Anions
DOE 6	ESP	Battelle	Jun 93	Comprehensive	Comprehensive
DOE 6	ESP/FGD	Battelle	Jun 93	Comprehensive	Comprehensive
10	FF	Radian	May, Dec 90	Comprehensive	Metals, Anions, VOST, Aldehydes, Semivols
11	ESP	Radian	Aug 90, Feb 92, Apr 93	Comprehensive	Metals, Anions, VOST, Aldehydes, Semivols
11	ESP/FGD	Radian	Aug 90, Feb 92, Apr 93	Comprehensive	Metals, Anions, VOST, Aldehydes, Semivols
22	ESP	Radian	Jul 93	Comprehensive	Metals, Anions, PAHs, Dioxins

Table B–3.
FCEM and Other Test Sites: Measurements (Continued)

Site No.	Configuration	Sampling Contractor	Test Period	Streams Sampled	Analyte Groups
101	FF	Radian	Jan 94	Comprehensive	Metals, anions
101	FF/FGD	Radian	Jan 94	Comprehensive	Metals, Anions, VOST
102	ESP	Interpol	May 91	Comprehensive	Metals, Dioxins
111	SD/FF	Clean Air Engr	Dec 91	Comprehensive	Metals, Anions, VOST, PAH
115	FF	Carnot	92/93	Comprehensive	Metals, Anions, Radionuclides
115	FF/Urea	Carnot	92/93	Comprehensive	Metals, Anions, Radionuclides
DOE 7	SD/FF	SRI	Jun 93	Comprehensive	Comprehensive
DOE 8	FF	Weston	Jun 93	Comprehensive	Comprehensive
103		Carnot	Apr 90	Stack	Benzene, Formaldehyde
104		Carnot	Mar 90	Stack	Benzene, Formaldehyde
105		Carnot	Feb 90	Stack	Benzene, Formaldehyde
106		Carnot	Mar 90	Stack	Benzene, Formaldehyde
107		Carnot	Feb 90	Stack	Benzene, Formaldehyde
108		Carnot	Mar 90	Stack	Benzene, Formaldehyde
109		KVB	Nov 90, Jan 91	Stack	Benzene, Formaldehyde
120		Carnot	Apr 93	Gas, Stack	Metals, VOC, PAHs
121		Carnot	Apr 93	Gas, Stack	Metals, VOC, PAHs, PCBs
13		Radian	Mar, Apr 91	Comprehensive	Metals, Anions, VOST, Aldehydes, Semivols

Table B–3.
***FCEM and Other Test Sites:* Measurements (Continued)**

Site No.	Configuration	Sampling Contractor	Test Period	Streams Sampled	Analyte Groups
13	BOOS	Radian	Mar, Apr 91	Comprehensive	Metals, Anions, VOST, Aldehydes, Semivols
13	pPJFF	Radian	Mar, Apr 91	Comprehensive	Metals, Anions, VOST, Aldehydes, Semivols
103		Carnot	Apr 90	Oil, Stack	Metals, PAH, Benzene, Formaldehyde
104		Carnot	Mar 90	Oil, Stack	Metals, PAH, Benzene, Formaldehyde
105		Carnot	Feb 90	Oil, Stack	Metals, PAH, Benzene, Formaldehyde
106		Carnot	Mar 90	Oil, Stack	Metals, PAH, Benzene, Formaldehyde
107		Carnot	Feb 90	Oil, Stack	Metals, PAH, Benzene, Formaldehyde
108		Carnot	Mar 90	Oil, Stack	Metals, PAH, Benzene, Formaldehyde
109		KVB	Nov 90, Jan 91	Oil, Stack	Metals, PAH, Benzene, Formaldehyde
112	Uncontrolled	Carnot	Jul 92	Comprehensive	Metals, Anions, PAH, VOC
112	ESP	Carnot	Jul 92	Comprehensive	Metals, Anions, PAH, VOC
117	Uncontrolled	Carnot	Jan 93	Comprehensive	Metals, Anions
117	pSCR	Carnot	Jan 93	Comprehensive	Metals, Anions
118	Uncontrolled	Carnot	Jan 93	Comprehensive	Metals, Anions
118	ESP	Carnot	Jan 93	Comprehensive	Metals, Anions
119	Uncontrolled	Carnot	Mar 93	Comprehensive	Metals, Anions, VOC, Radionculides, Dioxins
119	ESP	Carnot	Mar 93	Comprehensive	Metals, Anions, VOC, Radionculides, Dioxins

[a] Comprehensive streams are input, output, and major intermediate streams.

[b] Comprehensive analytes include metals, anions, VOC, PAHs, aldehydes, dioxins, furans, and radionuclides.

The type of information obtained at each site has varied, depending on the specific goals of each test program and the level of funding available. The initial list of 23 target species were not measured at each field site. As noted earlier, the radionuclides and dioxins/furans were initially not on the list of target species. As the field results from the early sites were evaluated, the sampling and analytical plans became more focused on the trace substances of highest concern from the standpoint of public health. At other sites, some internal streams were not measured because a suitable sampling location was not available. Results are presented later for only the target group previously listed.

B.1.2 Test Site Representativeness

To provide a reference for the test site characteristics, a series of comparisons have been done between the number of commercial units (the 1,750 + individual boilers larger than 25 MW reported in the Utility Data Institute's Power Statistics Database in operation, construction, or planned) and the number of data sets available. The following section briefly compares the configuration of units tested with commercial units for the following parameters:

This comparison is presented in a series of figures. In these figures, the left and right vertical scales maintain a 40:1 ratio. When the industry and data set bar height are the same, 2.5% of that industry classification has been tested.

Unit Age/Start-up Year. The comparison of the number of available data sets with the actual or scheduled startup year for commercial units in operation, construction, or planned is presented in Figure B-1. Tested sites have start-up dates within each decade from the 1930s to the present. The majority of commercial units (about 1,000) have start-up dates from 1950 to 1979. About 35 of the data sets are units in this age range. Proportionally more units with start-up dates from 1980 to the present have been tested. This is due in part to the number of pilot units and retrofits tested. The test dates for the pilot units or retrofits are used as the start-up date. Thus, many of the "1980–1989 start-up year" units and all of the "1990–1999 start-up year" units tested represent either possible "future" configurations" (i.e., pulse-jet fabric filters and other pilot unit technology) or retrofits of older units (i.e., the addition of low-NO_x burners). The focus of this study is plants operating in the year 2010. Thus, the large number of "future" configurations may represent components of the utility industry in 2010.

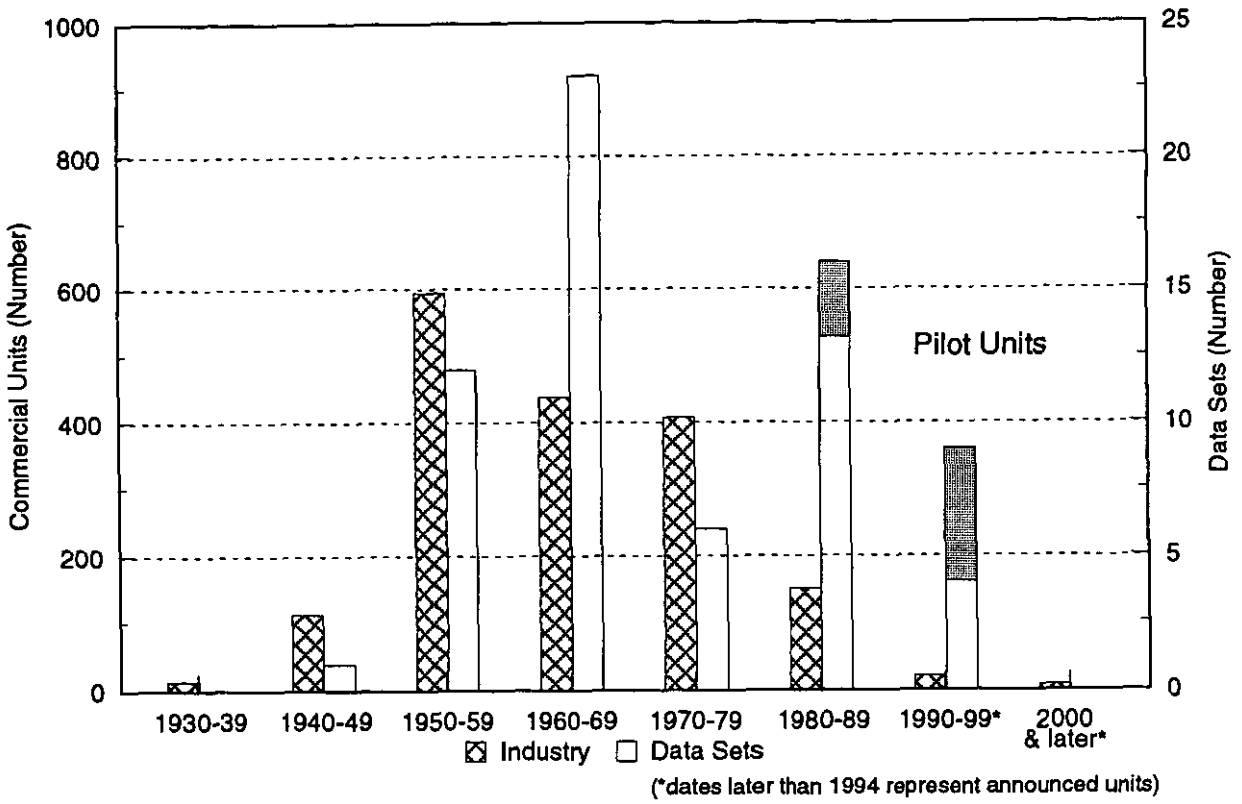


Figure B-1.
Unit Start-Up Year

Unit Size. Figure B-2 provides the distribution of unit size in 200 MW increments for the industry and the test sites. The pilot unit test data are included in the count of units under 200 MW, although they are actually less than 25 MW since they are treating a portion of gas from large units. While proportionally more units larger than 200 MW have been tested, the unit size is expected to have a relatively small effect on the concentration of trace substances emissions.

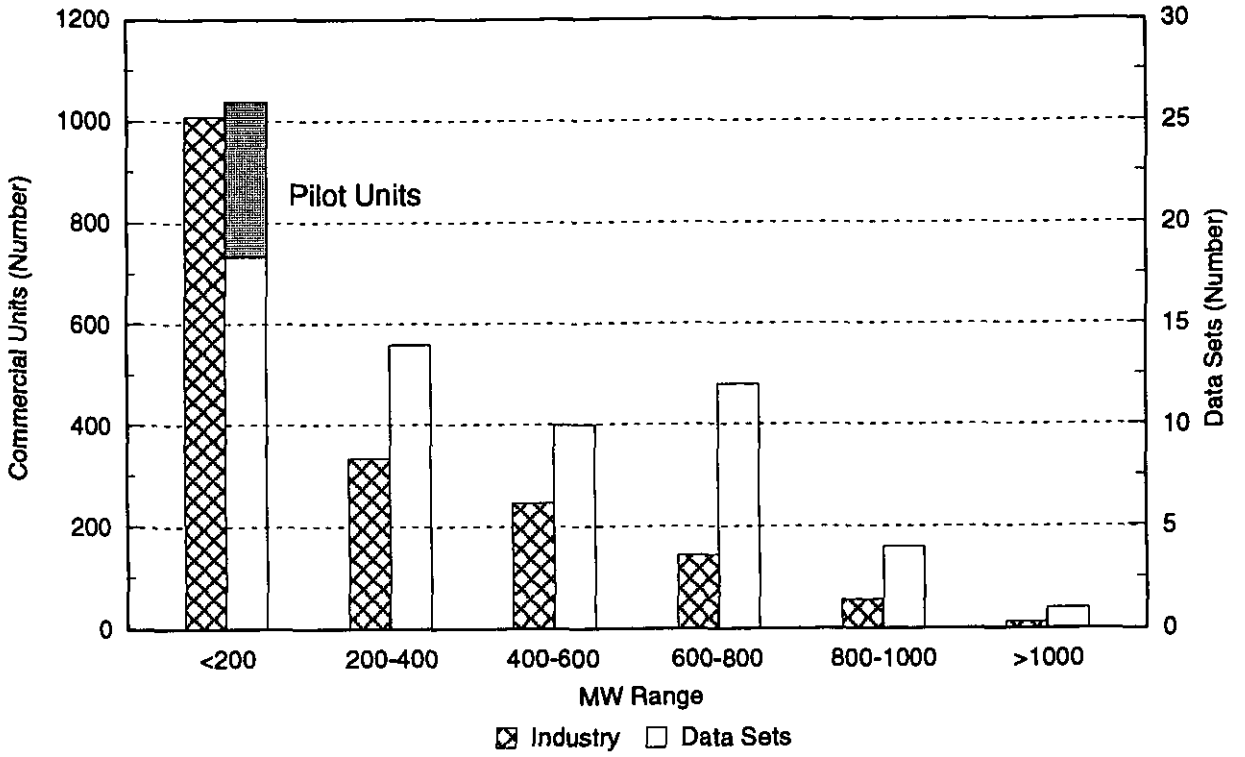


Figure B-2.
Unit Size

Fuel Type. Figure B-3 compares the fuel types of all the commercial and tested units. Proportionally more subbituminous and oil units have been tested. However, potential fuel switching strategies from medium- and high-sulfur bituminous coals to low-sulfur subbituminous coals may change the future industry profile. The large number of oil units listed are due to the seven sites tested as part of the State of California Air Toxics "Hot Spots" Information and Assessment Act of 1987 (AB2588).

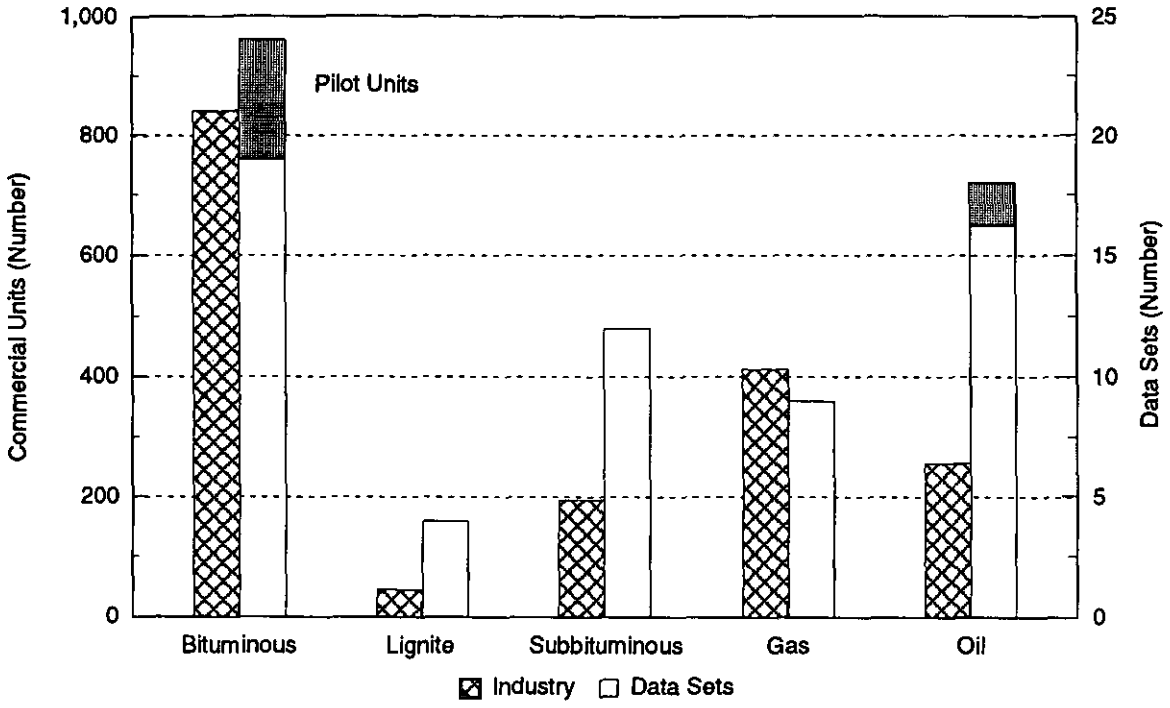


Figure B-3.
Fuel Distribution

Particulate, SO_2, and NO_x Control Technology. Figures B-4, B-5, and B-6 show the industry and test site distribution of particulate, SO_2, and NO_x control technologies for coal-fired units. Proportionally more fabric filters and flue gas desulfurization (FGD) systems were tested than are currently used; however, the Phase I and Phase II requirements of Title IV of the CAAA will likely result in the retrofit of additional FGD systems. Relatively few coal-fired units incorporate NO_x controls, and the proportion of sites tested with NO_x controls is greater than the percentage in the industry. Similarly, the CAAA may result in more NO_x control retrofits in the future.

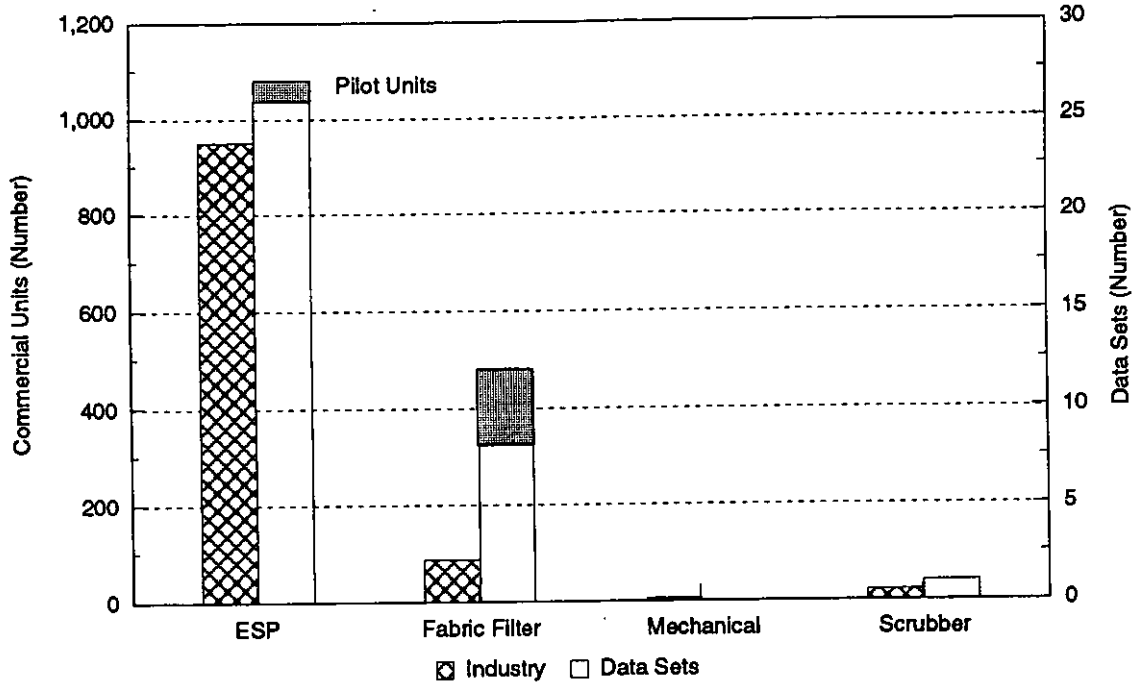


Figure B–4.
Particulate Control at Coal Units

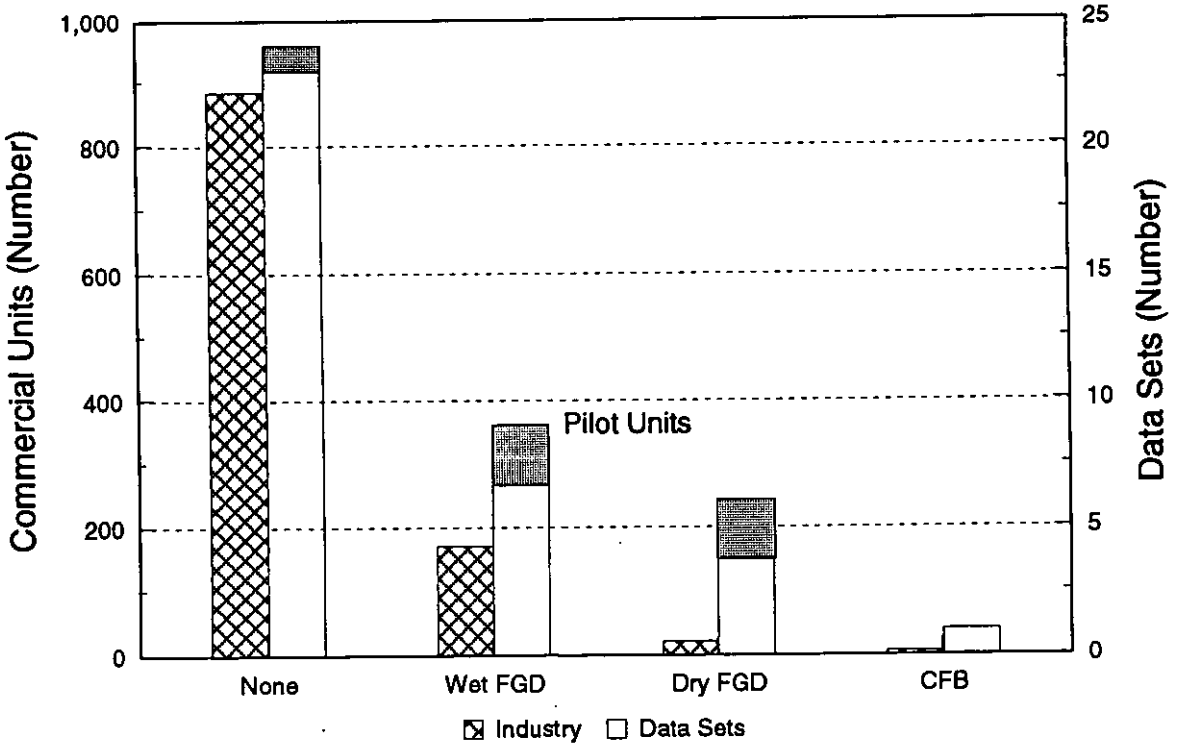


Figure B-5.
SO_2 Control at Coal Units

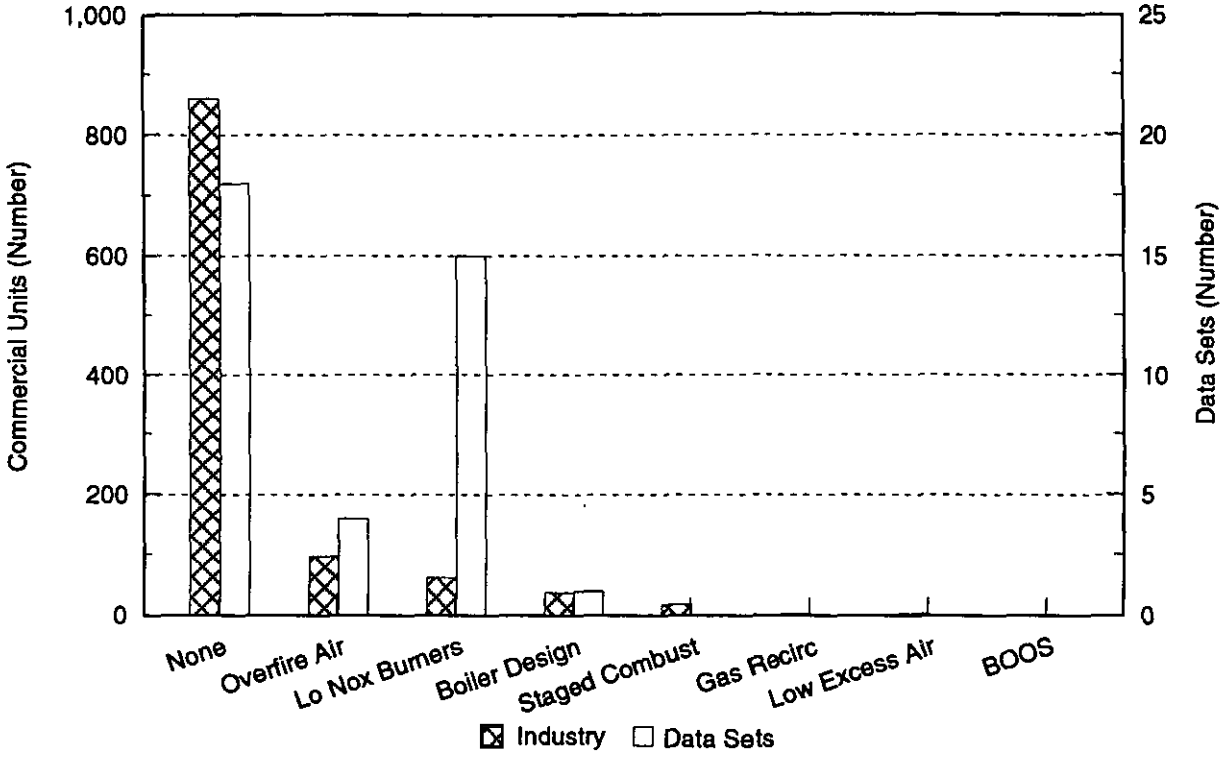


Figure B-6.
NO_x Control at Coal Units

Figures B-7 and B-8 show the type of NO_x controls found in oil- and gas-fired units. A greater percentage of oil and gas units employ some type of NO_x control relative to coal-fired boilers. About one-half of the oil units also use mechanical collectors or ESPs for particulate control. Less than 10% of the gas units have these devices.

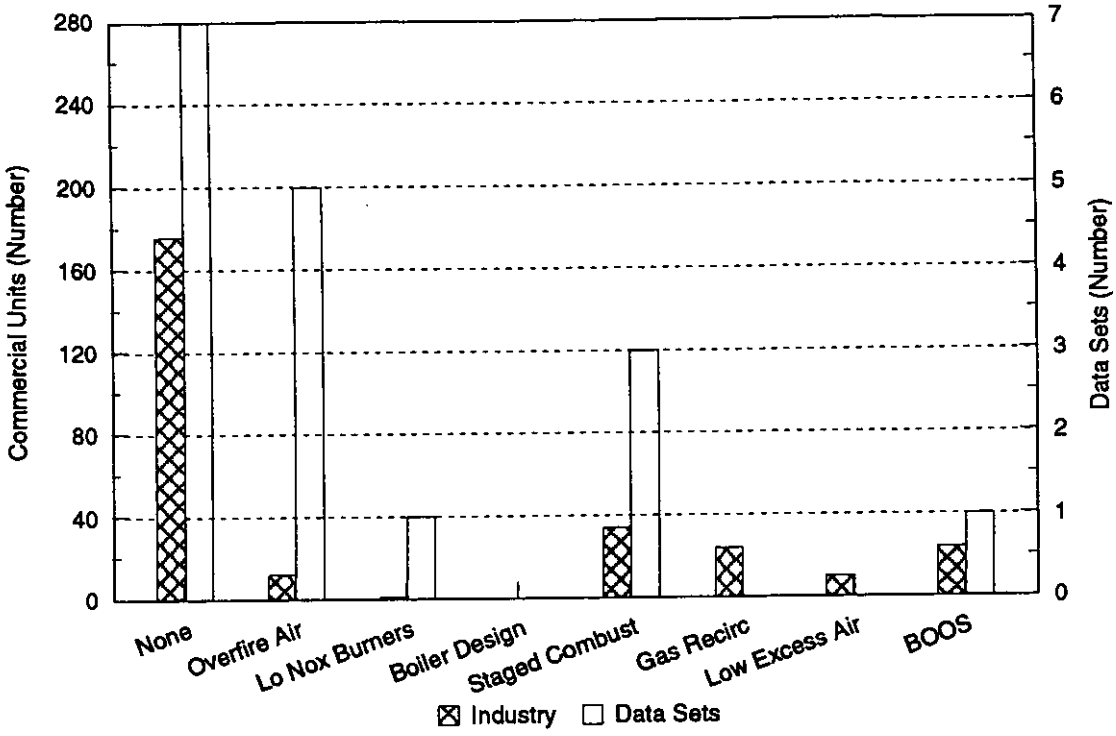


Figure B–7.
NO_x Control at Oil Units

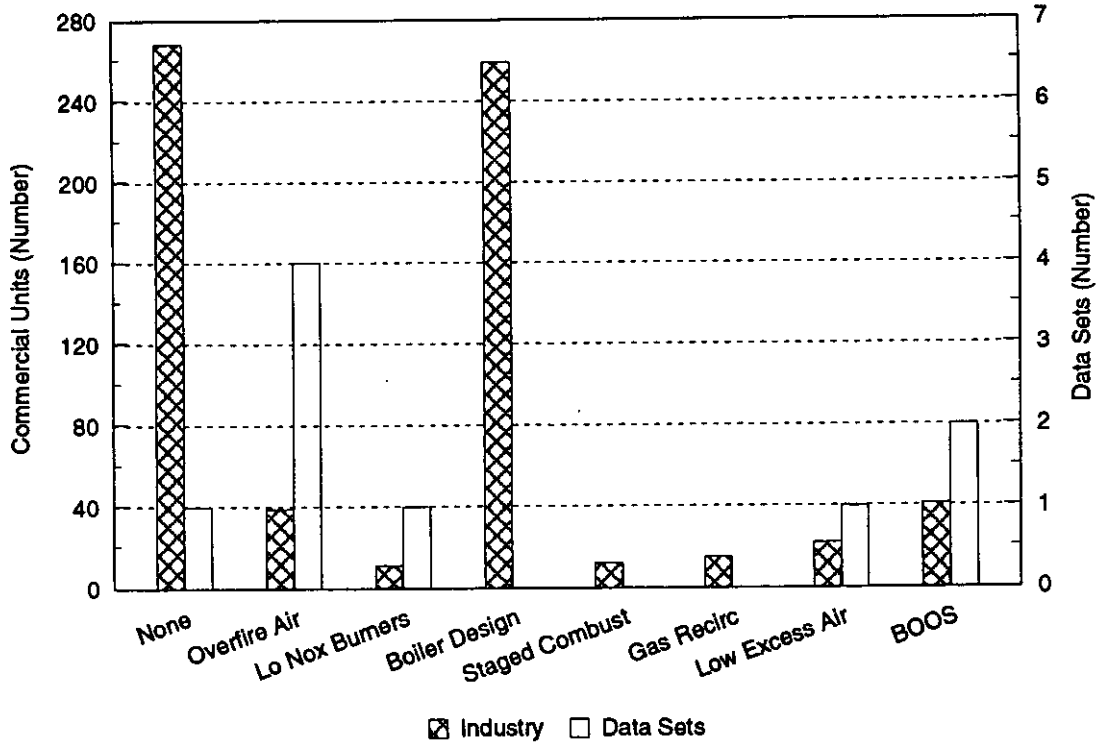


Figure B–8.
NO_x Control at Gas Units

Summary. Some type of recent trace substances measurements have been performed on about 3% of the utility boiler population. This testing has encompassed each major class of fuel, plant configuration, and unit size and age. Thus, the information obtained from the test sites provides a useful representation of the current and future emission levels of the electric utility industry.

B.2 Field Data Presentation and Correlations

B.2.1 Coal-Fired Results

Of the target substances, several logical groupings exist for presenting results. Many of the elements partition to the solid phase (at particulate control temperatures) and are effectively controlled by a conventional particulate control device. Other elements (mercury, chlorine, and selenium) are relatively volatile at stack gas conditions. The volatile organic substances are created during the combustion process and typically not effectively reduced by air pollution control devices at power plants. Radionuclides are present in the solid phase and can be directly related to the amount of particulate matter present in the stack gas. These four groupings are used in the following discussion.

B.2.1.1 Particulate-Phase Elements

Nine metallic elements, found at varying levels in coal, are listed as Hazardous Air Pollutants in Title III of the CAAA of 1990. These elements are: antimony, arsenic, beryllium, cadmium, chromium, cobalt, lead, manganese, and nickel. During combustion, many of these elements volatilize and subsequently re-condense on fly ash particles. At normal stack gas temperature of about 300°F, little, if any, of these elements are collected in the impingers of a multi-metals train (i.e., they are collected by the filter). Consequently, one would expect to see a strong dependence between total particulate emissions and the emissions of a specific trace metals. Since the emissions of metallic elements are affected by both the fuel concentration and the total amount of particulate matter emitted, it is reasonable to correlate the available emissions data with these two parameters. Coincidentally, these parameters are either available or can be estimated for plants that have not been tested.

While these nine metals all partition to the solid phase, the data set is robust enough to statistically differentiate the correlation coefficients for each of the metals. Arsenic data are presented below as an example of how the existing site data can be used to estimate emissions from untested coal-fired plants.

B.2.1.1.1 Arsenic.

At nearly all of the sites tested, the removal of arsenic exceeded 90% of that present in the coal. Figure B-9 presents the potential uncontrolled emission (the coal concentration divided by the heating value) in equivalent units (pounds of substance per 10^{12} Btu) against the average site emission factor for all of the data sets. The data plotted in this figure include emissions from ESPs, fabric filters, dry and wet FGD systems for all types of coals. Seven of the data sets were produced by cyclone boilers, the rest are either wall-fired or tangential-fired. For this data set, the average removal in arsenic is 96 percent. As seen in this figure, the absolute average emission level varied considerably for a given fuel concentration. The sources of this variability include the performance of the particulate control devices, daily fuel variability, and measurement uncertainty. Since arsenic is found in the particulate phase at stack temperatures, it is reasonable to include particulate matter as a term in a correlation. This will permit the data to be differentiated by particulate emission levels for a given fuel concentration at each site.

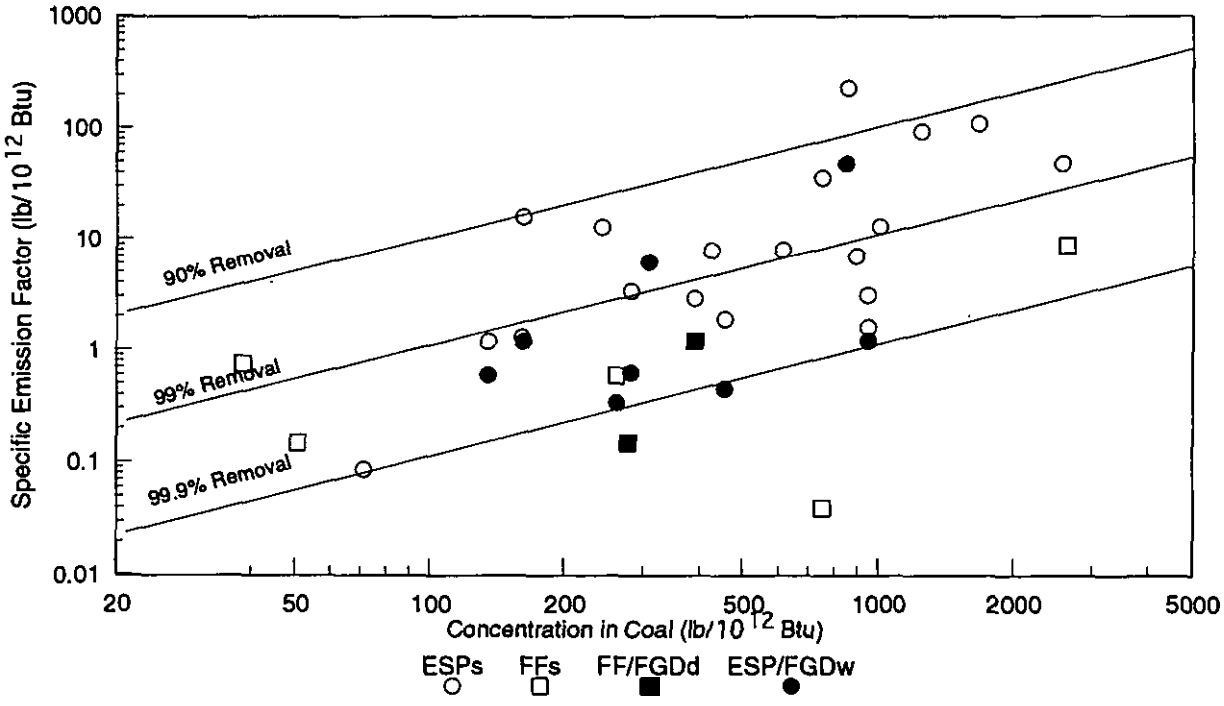


Figure B–9.
Arsenic in Coal vs. Emission Levels

Thus, it is reasonable to correlate particulate-phase metal emissions with the inlet coal composition and particulate matter emissions. Because the ultimate purpose of this analysis is to predict trace substance emissions for the utility industry, it is prudent to use readily available information for the independent parameters. Since both the coal compo-

sition and particulate matter emission can be easily estimated for power plants, correlating trace substance emissions with coal composition and particulate matter emission levels would be suitable for estimating emissions for the utility industry.

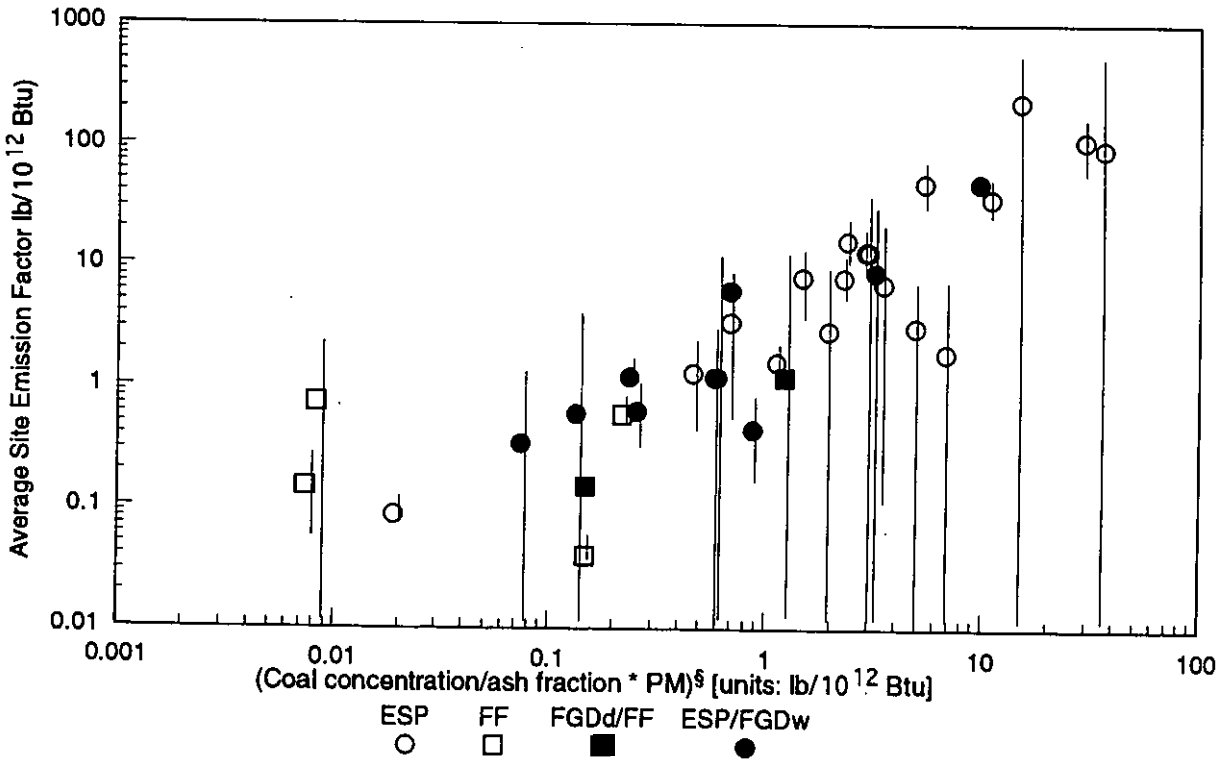
The data can be expressed in the following form:

$$E_i = f\,[(Coal_i / Ash\ Fraction)*PM]$$

where:

E_i	=	Emission of substance "i" (lb/10^{12} Btu)
$Coal_i$	=	Trace substance "i" concentration in coal (ppm)
Ash Fraction	=	Fraction ash in coal (lb ash/lb coal)
PM	=	Total particulate matter emission (lb/10^{6} Btu)

Figure B-10 plots the measured emissions as a function of coal trace substance concentration, coal ash, and particulate emission.



§ **Coal concentration**: concentration of substance in coal [parts per million by weight]; **ash fraction**: fraction of coal that is ash [dimensionless]; **PM**: particulate matter emissions [lbs. per 10^{6}Btu]

Figure B–10.
Arsenic Correlation Data

The vertical lines through each average site emission spans the 95% confidence limit about the mean value. At many sites, although the mean value is large, the confidence interval includes zero, indicating large uncertainty in the calculated site mean.

An examination of Figure B-10 suggests that a power relationship of the following form may fit the data:

$$E_i = a_i [(coal_i / ash\ fraction) * PM]^{b_i}$$

where a_i and b_i are correlation coefficients for trace substance "i."

Also note that the data points do not show any marked difference for the various types of control technologies (i.e., although most ESP/FGD and fabric-filter sites have lower arsenic emission levels, they line up with the ESP emission data). This simply indicates that the nominal composition of particulate matter exiting a control device is primarily dependent on the fuel concentration, and that any particle size/chemical composition relationships are small when data from many sites are aggregated. Conducting the regression analysis on the data shown in Figure B-10 yields the following:

$$E_i = 3.1 [(Coal_i/\text{Ash Fraction})*PM]^{0.85}$$

Figure B-11 shows the regression of the data. The confidence intervals about each site average have been removed for clarity of presentation. The correlation coefficient (r^2) is 0.72. Because the regression includes over 30 data points, the correlation is significant at the 99.9% probability level (i.e., there is a one in one thousand probability that a set of numbers would show this relationship from chance alone). This equation predicts the long-term average emission level of arsenic from a typical coal-fired plant for a constant coal concentration and particulate emission level. Actual emissions measured at specific plants may vary considerably from the predicted value. Two additional statistical parameters are also shown on the figure. The outer dashed lines are the 95th percentile confidence bands for the site mean values. This band is where the average of triplicate measurements for a site should lie 95% of the time.

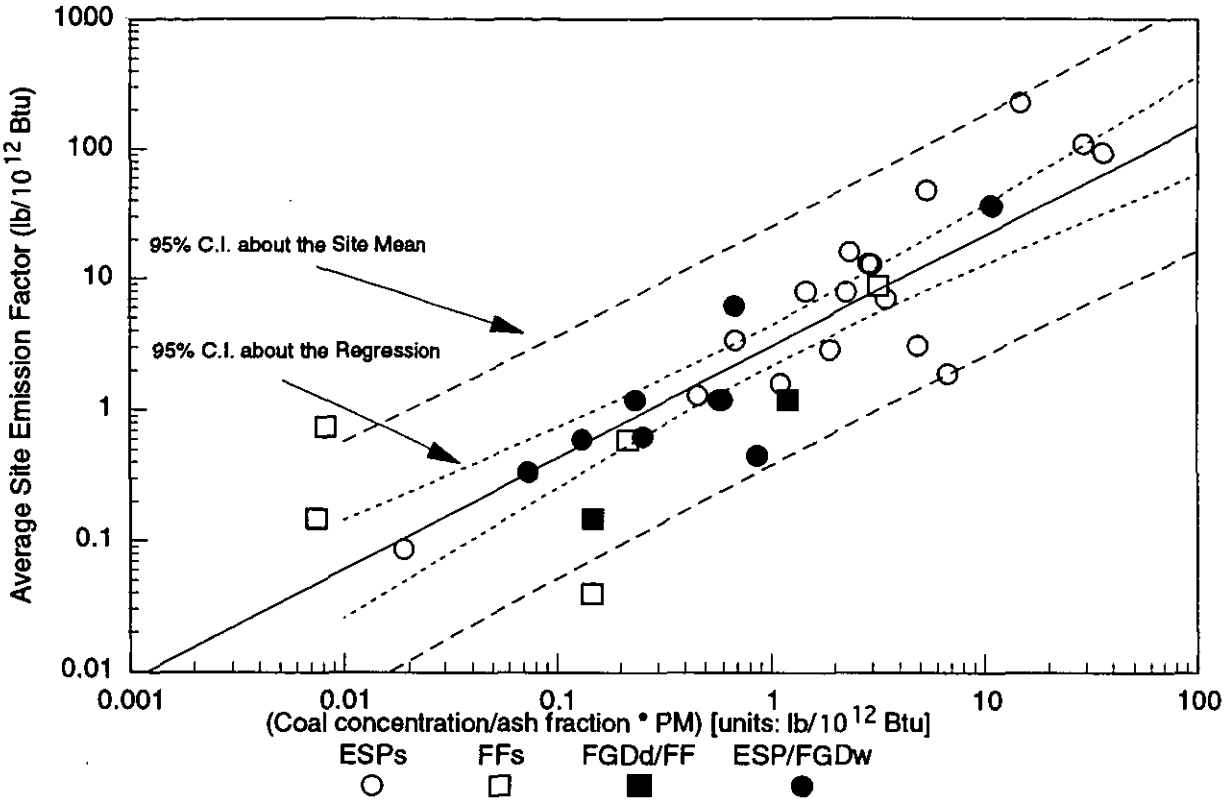


Figure B–11.
Arsenic Emission Correlation

The inner dotted lines represent the upper and lower 95% confidence intervals about the regression. It is within this range that the true average value is expected to occur, 95% of the time.

B.2.1.1.2 Other Elements.

Because this approach uses data from all of the coal-fired plants for a specific substance and permits the use of relatively easy to obtain input parameters, it is an appropriate method to estimate the emissions from plants that have not been tested. It is favored over more simplistic approaches, such as average removal efficiencies or constant emission factors since it incorporates logical input parameters. Given the variability present in the measurement at a single site, more refined approaches are probably unjustified unless many input parameters are available. Therefore, similar correlations and figures have been developed for the other eight metals. The correlations are presented in Table B-4. The correlation coefficient (r^2) indicates the model relationship is statistically significant for these particulate-phase metals. As with arsenic, the variability of the predicted emission level can be expressed with confidence intervals. Appendix B.3 presents the figures and the statistical information needed to calculate these values for all nine metals.

Table B-4.
Summary of Particulate-Phase Emission Equations, lb/10^{12} Btu

Analyte	Average Predicted Emissions	Data Pairs	r^2
Antimony	$(0.92) \times^{0.63}$	8	0.65
Arsenic	$(3.1) \times^{0.85}$	34	0.72
Beryllium	$(1.2) \times^{1.1}$	17	0.83
Cadmium	$(3.3) \times^{0.5}$	9	0.78
Chromium	$(3.7) \times^{0.58}$	38	0.57
Cobalt	$(1.7) \times^{0.69}$	20	0.57
Lead	$(3.4) \times^{0.80}$	33	0.62
Manganese	$(3.8) \times^{0.60}$	37	0.57
Nickel	$(4.4) \times^{0.48}$	25	0.51

x = Coal ppm/ash fraction • PM.
r^2: Correlation coefficient for the regression.

Figure B-12a and Figure B-12b show the calculated average emission rate for these elements on both logarithmic and linear scales for the ranges of independent levels seen at the test sites. The elements traditionally thought to be more volatile (lead and arsenic) show higher emissions for a given input. Chromium, magnesium, and nickel behave nearly identically. Antimony, beryllium and cobalt are the least volatile elements. All measured and predicted values of antimony, beryllium, and cadmium are below 10 lb/10^{12} Btu.

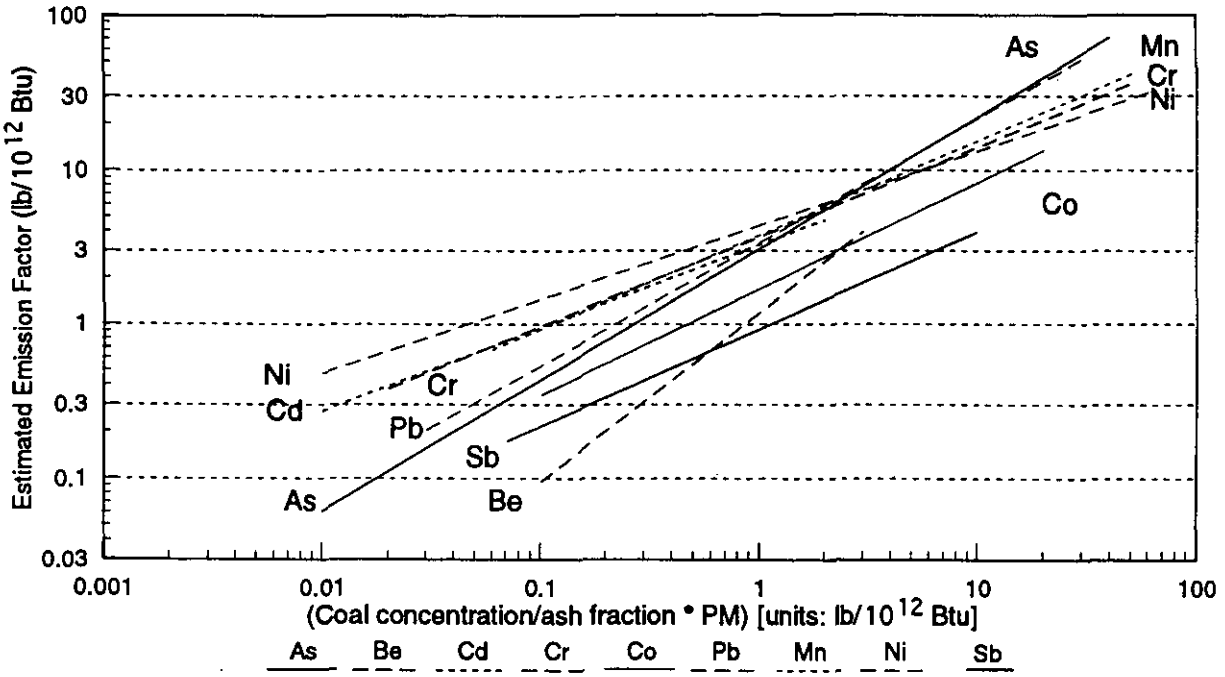


Figure B–12a.
Predicted Metal Emission Factors, Logarithmic Scales

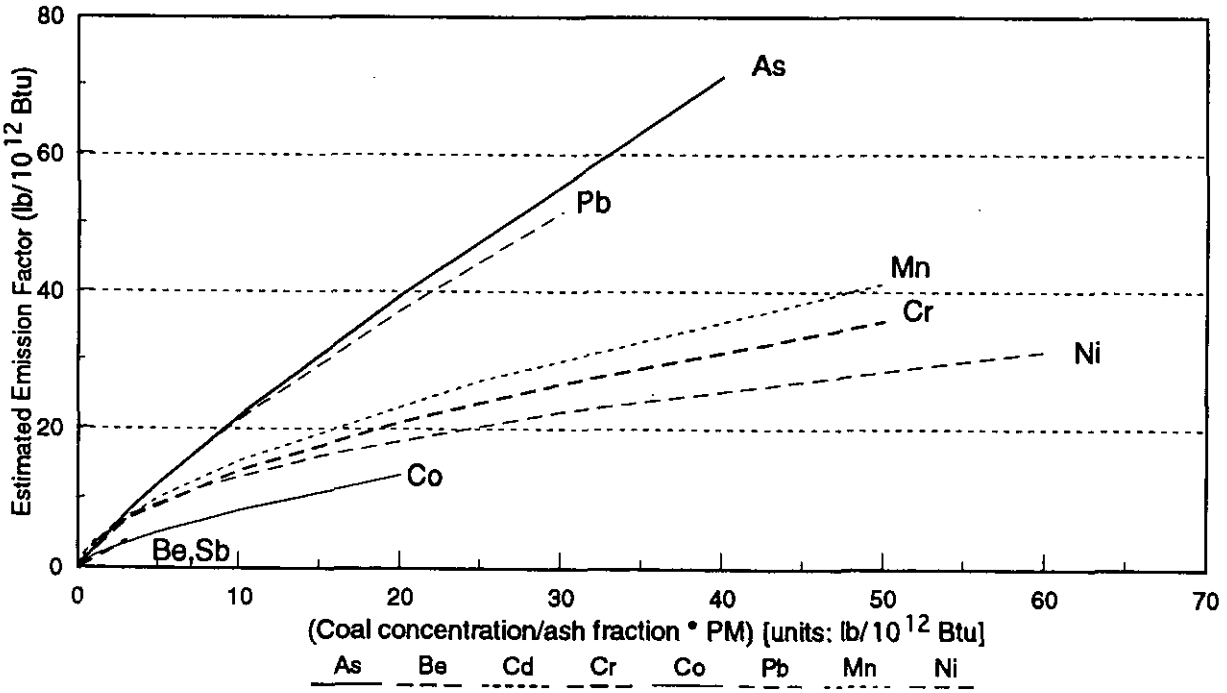


Figure B–12b.
Predicted Metal Emission Factors, Linear Scale

Figure B-13 compares the emission factors obtained from the recent sampling efforts with those published in the literature [1] for utility boilers with ESPs. As can be seen, for each substance, the arithmetic average is lower for the current data set. This may be due to 1) a boiler population that has fewer "poor performing" units and/or 2) the use of better sampling equipment. Prior to the development of the multi-metals train (which uses glass nozzles and probe liners) significant chromium and nickel contamination was often encountered which makes older literature values suspect.

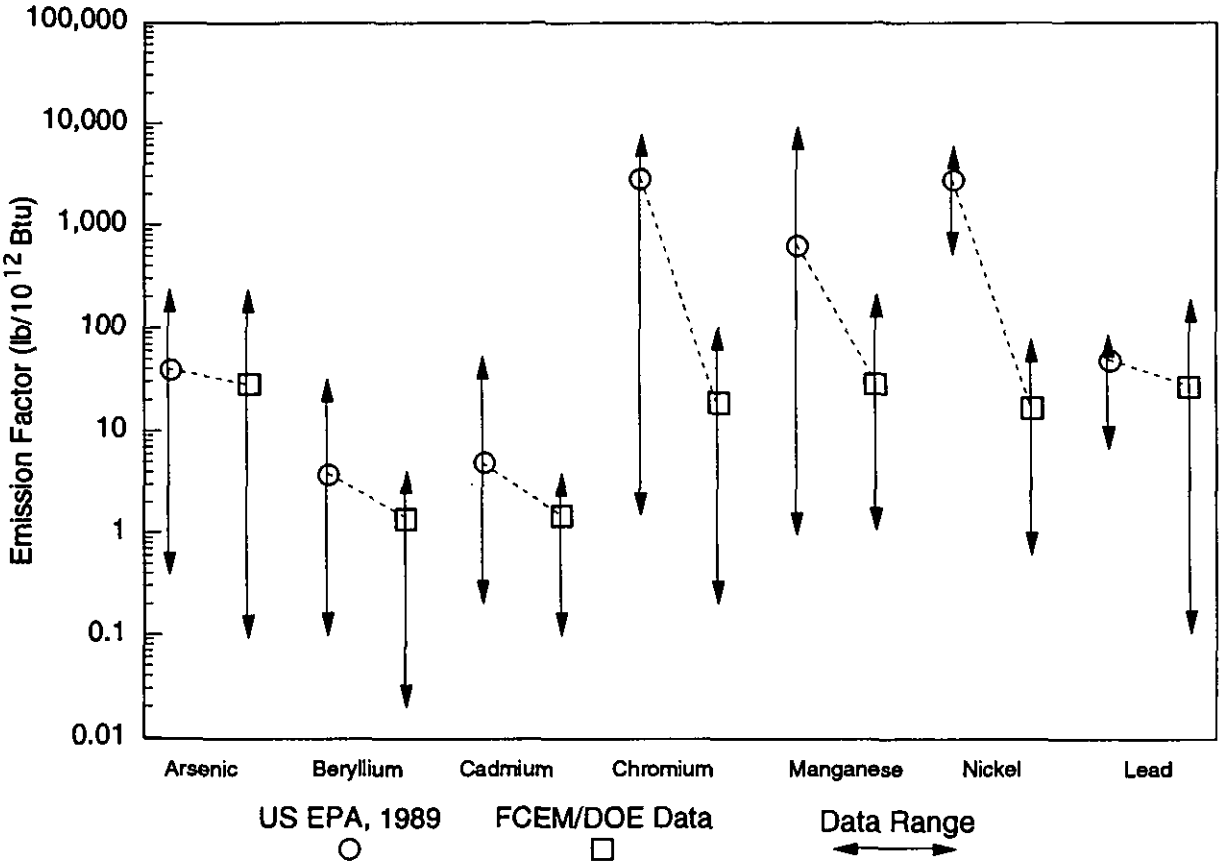


Figure B-13.
Comparison of Emission Factors from Literature Citations with Those from Recent Emissions Measurements

B.2.1.2 Volatile Elements

Three inorganic substances found in coals are present primarily in the vapor-phase of combustion flue gases and are not typically removed effectively by particulate control devices. Mercury, selenium, and hydrochloric acid measurement results and emission estimation techniques are discussed below.

B.2.1.2.1 Mercury.

Of all the inorganic substances listed on the HAPs list, mercury is generally present in coal at the lowest levels. Concentrations in coal typically vary from 0.02 to 0.15 mg/kg. This low level approaches analytical detection limits (approximately 0.02 mg/kg), creating uncertainty in the reported fuel concentrations. The non-homogeneity of coal creates additional variability. The sampling methods for flue gas mercury use concentration techniques, in which a large volume of gas is pulled through absorbing solutions or solids. This usually produces sufficient quantities of mercury in the sample so that detection level uncertainty is typically not a concern. The impact of this variability is that the mass balance comparison of the fuel and stack gas is often indeterminate. At several sites, the calculated mass flow rate of mercury exiting the plant in the flue gas exceeds that entering with the coal. As long as there is not a gross disparity, this type of result just reflects the variability inherent in measuring trace substances. Figure B-14a presents the sets of paired data for mercury (fuel and gas emissions) for various control devices. As can be seen, for a number of ESP sites (and one wet FGD system), the normalized concentration in the gas stream is higher than the coal measurement. This appears to represent sampling and analytical variability.

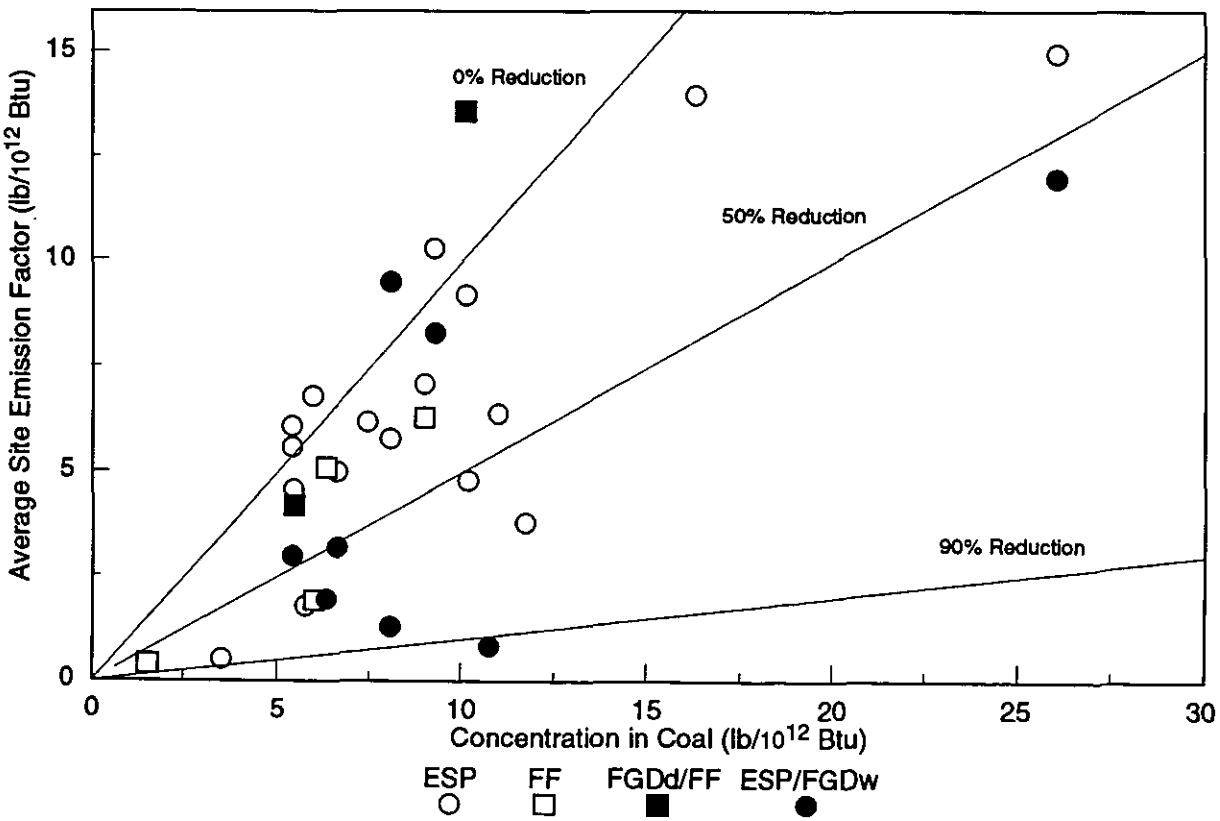


Figure B–14a.
Mercury Emissions, Coal Units

Table B-5 presents the removal efficiency calculated for plants with various types of control devices based on the current data set. Also shown is the 95% confidence interval about the multiple-site average reduction. The large confidence interval indicates that the average value lacks precision, as expected based on Figure B-14a. Furthermore, the small increase in control seen at sites with ESP/wet FGD systems indicates that effective methods of controlling mercury emissions at power plants are not currently understood. Preliminary research indicates that ionic forms of mercury are removed to a greater degree in both wet and dry control devices; however, the mechanism or conditions under which mercury exists in an ionic state are not known. Although the "Student-t" test does not indicate that the distribution of removals seen at dry particulate control sites is different from those observed at plants with FGD systems, measurements before and after FGD systems consistently indicate some reduction in mercury emissions. Based on the current data set, it is recommended that mercury emissions from plants with ESPs and fabric filters be estimated as 70% of the fuel level. For wet and dry FGD systems, 55% of the fuel level is recommended.

Table B-5.
Mercury Reduction by Control Devices, All Coal Types

Control Device	Number of Sites	Average Reduction	95% C.I.	Recommended Emission Factor Percent of Coal Input
ESP	17	26%	±14%	70%
Fabric Filter	5	39%	±38%	70%
ESP & Fabric Filter	22	29%	±13%	70%
FGD Systems	9	45%	±27%	55%

B.2.1.2.2 Mercury Speciation.

Although mercury is present at relatively low concentrations in coal and combustion gases, its bioaccumulation is of concern. Because it has a relatively high vapor pressure at stack gas temperatures, emissions reduction require approaches not commonly employed at power plants. To further understand the physical and chemical properties of mercury, attempts have been made to measure the types of mercury present in combustion gases.

There are two sampling methods that have been used to speciate mercury emissions—the EPA multi-metals train (draft Method 29) and the sorbent speciation train of Bloom, basis for the MESA method [2]. Both methods should provide some indication of the degree of oxidation of mercury, although neither has been validated for speciation applications. Method 29 has been validated for total mercury measurements. Method 29 involves the collection of an isokinetic volume-proportioned sample of gas. The MESA sampling

apparatus uses a stationary probe without a nozzle and, therefore, does not obtain a representative particulate sample. The MESA method requires the gas be well mixed and the mercury be present in the vapor phase.

Method 29 mercury analysis are based on three portions of the sampling train—the filter and probe and nozzle rinse, two nitric acid/hydrogen peroxide impingers, and two potassium permanganate/sulfuric acid impingers. The mercury captured in the first two stages (the filter and nitric acid) is assumed to be an ionic form, presumably mercuric chloride. This assumption is made because elemental mercury is highly insoluble in aqueous solutions and the filter operates at 250°F (so mercury should not condense). It has been demonstrated that the $KMnO_4$ impingers collect any mercury that passes through the nitric impingers. This mercury is, therefore, considered to be in the elemental state.

The MESA method uses solid sorbent traps to collect the various species of mercury. A soda-lime sorbent is used in the first series of traps to remove oxidized mercury, followed by an iodated activated carbon trap for elemental mercury capture. Mercury is recovered by several techniques that offer the possibility of further differentiating the oxidized species that may be present. Both of these methods must be considered experimental in terms of mercury speciation.

Table B-6 presents the speciation data available from the FCEM and DOE field sites. Shown on this table for each site is the coal analysis, control devices employed at the test sites, and the percent of mercury calculated to be in an ionic form based on the multi-metals train or the solid sorbent procedure. Gas samples were obtained from three locations at power plants: upstream of the particulate control device (high dust gas), downstream of the particulate control device (low dust gas), and downstream of a flue gas desulfurization unit, if present (scrubbed gas). Figure B-14b shows the direct comparison of the Method 29 and MESA oxidized fraction at sites where both procedures were used. These results show two general observations. First, the MESA method has, on the average, lower oxidized values than Method 29. This could mean the soda lime trap does not collect all of the ionic mercury (a temperature effect has been observed), or the nitric impingers collect some elemental mercury (the multi-metals train is not designed for speciation). Additional research is ongoing attempting to resolve these issues. Second, as the combustion gas passes through particulate control devices, the percent of oxidized mercury present remains constant at nearly 70 percent. This is unexpected, since the data in the prior section show about 30% of the mercury is removed (based on the fuel concentration). If only ionic mercury is removed at stack temperatures, the ionic percentage should decrease across an ESP or fabric filter. This effect is seen to a small degree when the gas passes through an FGD system (the nominal oxidized fraction is 45% of the total mercury present).

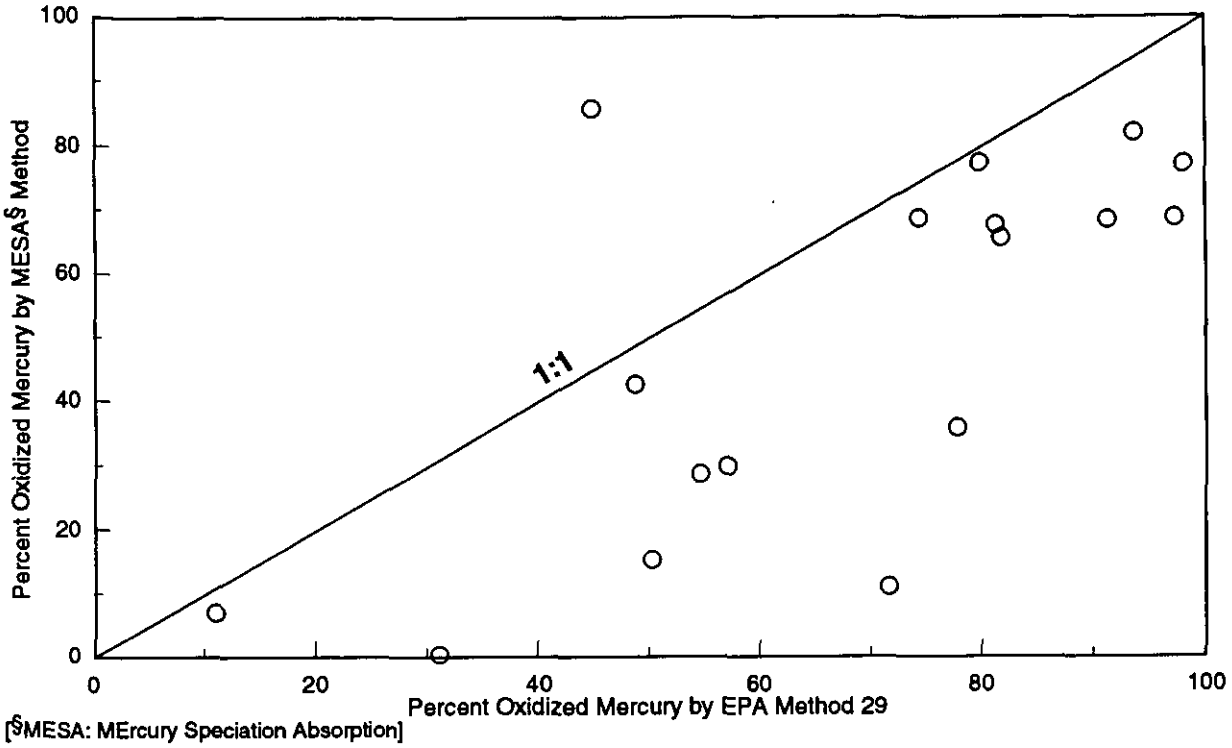


Figure B–14b.
Comparison of Mercury Speciation Data Across Methods

Table B-6.
Mercury Speciation Data for Coal-Fired Sites

	Coal Analysis				Control Systems			Percent of Mercury as Oxidized					
								High Dust Gas		Low Dust Gas		Scrubbed Gas	
SITE ID No.	Rank	S%	Hg ppm	C.I. ppm	ESP	Fabric Filter	FGD System	M29	MESA	M29	MESA	M29	MESA
Site 122	Bit	2.0	0.079	2500	Y					43	63		
Site 110	Bit	2.9	0.06	1850	Y								
Site 110 OFA	Bit	2.9	0.08	1700	Y								
DOE 4	Bit	2.5	0.08	1400	Y		Y	97	69	82	66	50	15
Site 15	Bit	1.6	0.14	860	Y								
Site 12R	Bit	2.6	0.10	830	Y		Y	75				46	
Site 21	Bit	2.6	0.10	800	Y		Y	91	69			71	11
DOE 3	Bit	2.9	0.07	790	Y			78	36	74	69		
Site 125	Bit	3.0	0.09	740	Y		Y		14		35		19
Site 19	Bit	0.9	0.10	700	Y					93			
Site 16	Bit	1.6	0.15	410	Y					82			
DOE 7	Sub	0.7	0.03	400		Y	Y	56				14	

Table B-6.
Mercury Speciation Data for Coal-Fired Sites (Continued)

	Coal Analysis				Control Systems			Percent of Mercury as Oxidized					
								High Dust Gas		Low Dust Gas		Scrubbed Gas	
SITE ID No.	Rank	S%	Hg ppm	C.I. ppm	ESP	Fabric Filter	FGD System	M29	MESA	M29	MESA	M29	MESA
Site 18 PJFF	Bit	1.1	0.12	400		Y		82		84			
Site 18	Bit	1.1	0.12	400	Y					73			
Site 116 SNRB	Bit	3.5	0.13	370		Y							
Site 116	Bit	3.4	0.13	370	Y								
Site 16L	Bit	1.7	0.14	340	Y					66	45		
Site 101	Sub	0.8	0.06	290		Y	Y			94	82	81	68
Site 11R	Sub	0.6	0.11	280	Y		Y			57	30	54	29
Site 115 Urea	Bit	0.5	0.02	238		Y							
DOE 8	Sub	0.7	0.08	190		Y		49	43	80	77		
Site 20	Lig	2.2	0.26	130	Y		Y			45	86	11	7
Site 22	Sub	0.4	0.14	<100	Y					31	0		
Site 114 RB	Bit	1.7	<0.11	82	Y								

Table B-6.
Mercury Speciation Data for Coal-Fired Sites (Continued)

	Coal Analysis				Control Systems			Percent of Mercury as Oxidized					
								High Dust Gas		Low Dust Gas		Scrubbed Gas	
SITE ID No.	Rank	S%	Hg ppm	C.I. ppm	ESP	Fabric Filter	FGD System	M29	MESA	M29	MESA	M29	MESA
Site 114	Bit	1.7	<0.12	69	Y								
Site 115	Bit	0.5	0.02	22		Y			77		50		
DOE 2	Bit	3.8	0.21	4	Y			85		86			
DOE 2 FF	Bit	2.8	0.26	4		Y		78		86			
DOE 6	Lig	1.1	0.08	0.4	Y			31		64		34	
Hg Valid.	Bit	1.0	0.02		Y					98	77		
DOE 5	Bit	3.1	0.05		Y			0.37		0.50			
Mean		1.9	0.10	560				66	51	69	57	45	25
95% C.I.		0.4	0.02	220				19	26	13	15	21	23

Note also in Table B-6 that the data are sorted in decreasing concentration of coal chloride levels. The relationship between the oxidation state and the chloride concentration is under investigation. Because of the uncertainties in the understanding of mercury chemistry, an arithmetic mean value is recommended for estimating the oxidized fraction. For dry particulate devices, 70% of the emission level is estimated to be in the oxidized state. After scrubbing, 45% of the mercury is in the oxidized state. These results are based on Method 29 and should be considered as an upper bound.

B.2.1.2.3 Selenium.

The behavior of selenium in power plants is somewhat similar to sulfur in that it forms an acid gas during combustion (SeO_2) and can be neutralized and absorbed by alkaline substances. SeO_2 sublimes at 650°F; therefore, as flue gas is cooled, it should re-condense on fly ash particles. However, to a large degree selenium remains vaporized. The majority of selenium is typically found in the vapor phase, collected in impingers in a multi-metals sampling train instead of the quartz filter.

Figure B-15a presents the matched sets of data for coal and gas emissions at various plant configurations. Some particulate-controlled sites exhibit high levels of removal; these sites predominantly are burning coals with high levels of alkaline ash (10-20% CaO). Most of the wet FGD systems show high removal also. The one wet FGD system showing slightly negative removal is designed primarily for particulate control; the mass transfer area for gas-phase species is probably very small. Although the average removal seen in fabric filters is higher than seen in ESPs, the difference is presumably due more to the sample population than the inherent capability of fabric filters. Plants where most of the fabric filters tested burn subbituminous alkaline ash coals. The two fabric filter data points that show low removal are at bituminous coal boilers. Figure B-15b shows the same data set, ordered by coal rank, and FGD systems. The presence of alkaline ash or an FGD system reduces selenium emissions to a great degree. Table B-7 presents the recommended emission factors for coal plants, based on coal rank, or the presence of an FGD system. Again, a multiple site average value and confidence interval has been computed. For units burning bituminous and lignite coals, 55% of the coal level is recommended for the emission factor. For units burning subbituminous coal, 3% of the coal value is emitted. Units with FGD systems emit an average of 12% of the fuel level.

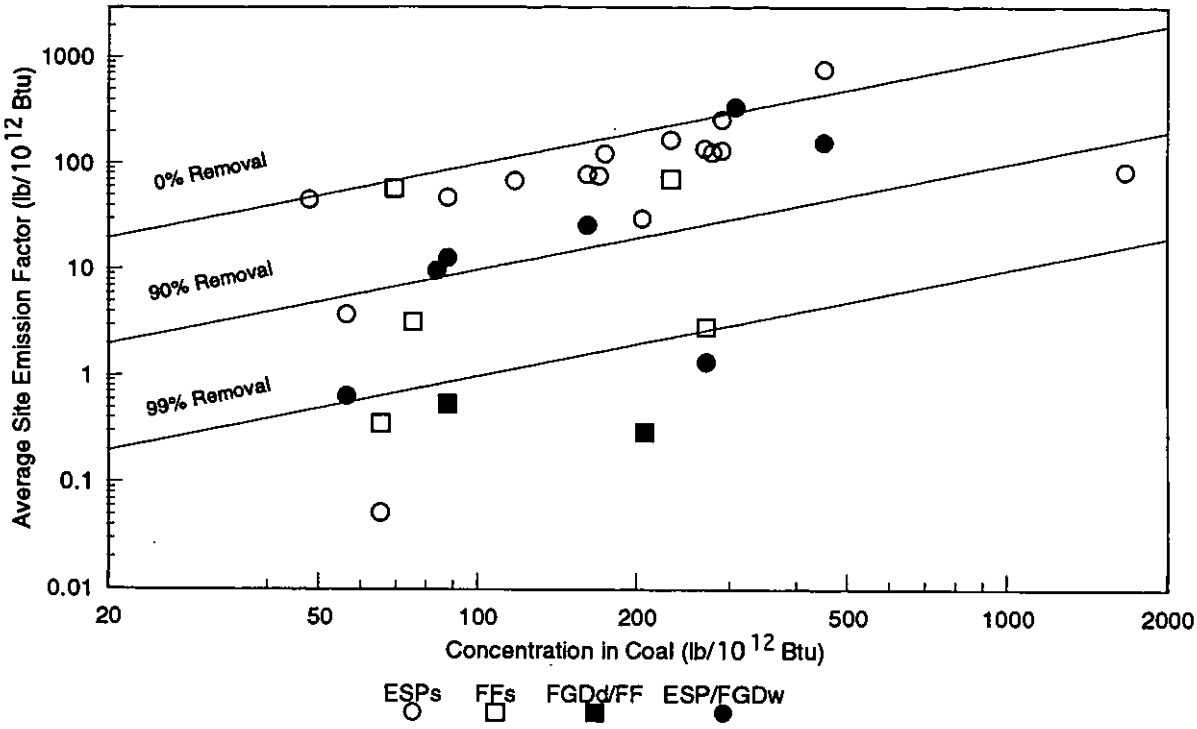


Figure B–15a.
Selenium Emissions, by Control Device

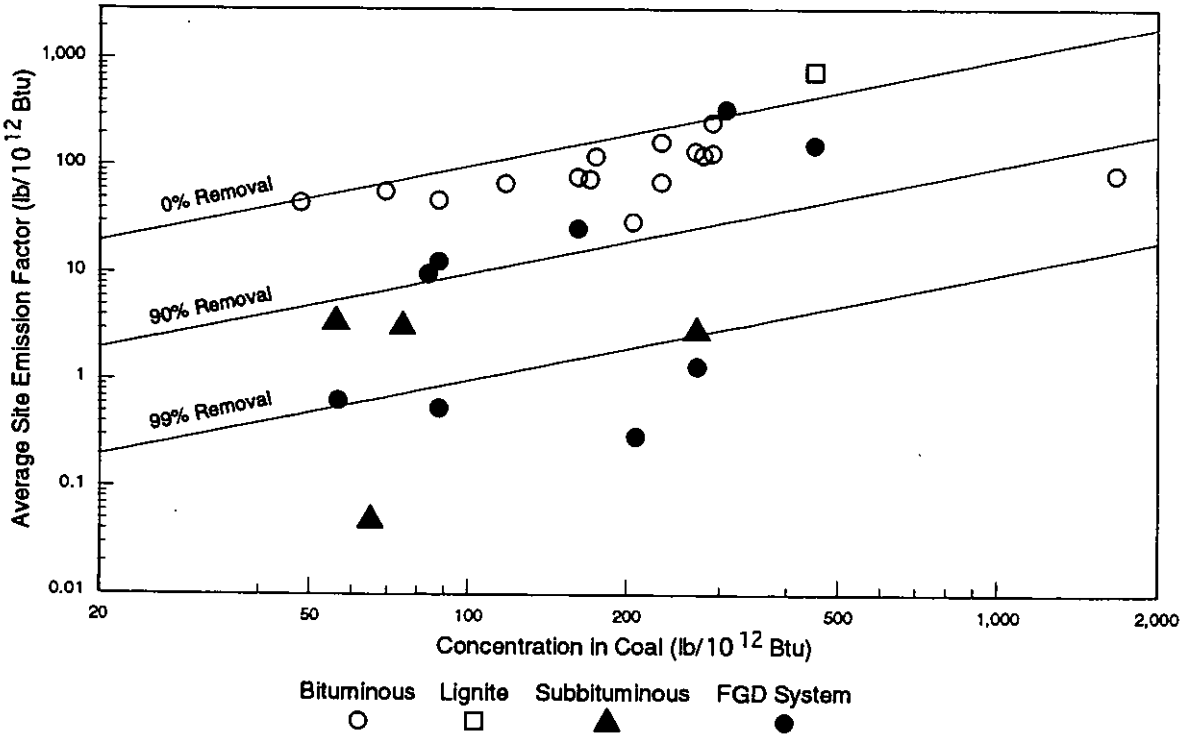


Figure B–15b.
Selenium Emissions, by Coal Type

Table B-7.
Selenium Removal by Coal Type and FGD System

Coal Type [a]	Number of Sites	Average Removal	95% C.I.	Recommended Emission Factor Percent of Coal Input
Bituminous	15	45%	±13%	55%
Lignite	1	use bituminous		55%
Subbituminous	5	97%	±4%	3%
Bituminous or Lignite, with FGD System	8	88%	±12%	12%

[a] For any coal type controlled by ESP or fabric filter.

B.2.1.2.4 Hydrochloric Acid.

Like selenium, chloride present in coals typically forms a gas phase species, HCl, during combustion. Of all the HAPs substances, chloride concentrations in coal are at the highest levels, ranging from 100 to several thousand mg/kg. Gas-phase concentrations are on the order of 10 to 100 mg/Nm^3. HCl is readily absorbed in aqueous solutions and is neutralized also by alkaline materials. Figure B-16 presents the data pairs for HCl and coal chloride as a function of coal rank, again demonstrating the effect of alkaline ash and scrubbing. Table B-8 presents the range of removals and the recommended value for emissions estimation. Although the one lignite data set shows fairly high removal on Figure B-16, the fly ash from this site contained no chloride; therefore, the removal value is suspect and low alkalinity lignite fuels should be considered with bituminous coals for estimation purposes. Units burning bituminous and lignite coals show 100% emissions; subbituminous coals emit 20% of the fuel level. Units with FGD systems emit 3% of the fuel level.

Table B-8.
HCl Reduction by Coal Type and FGD System

Coal Type [a]	Number of Sites	Average Removal	95% C.I.	Recommended Emission Factor Percent of Coal Input
Bituminous	15	-1%	±13%	100%
Lignite	1	use bituminous		100%
Subbituminous	7	79%	±14%	20%
All, with FGD System	5	97%	±2%	3%

[a] For any coal type controlled by ESP or fabric filter.

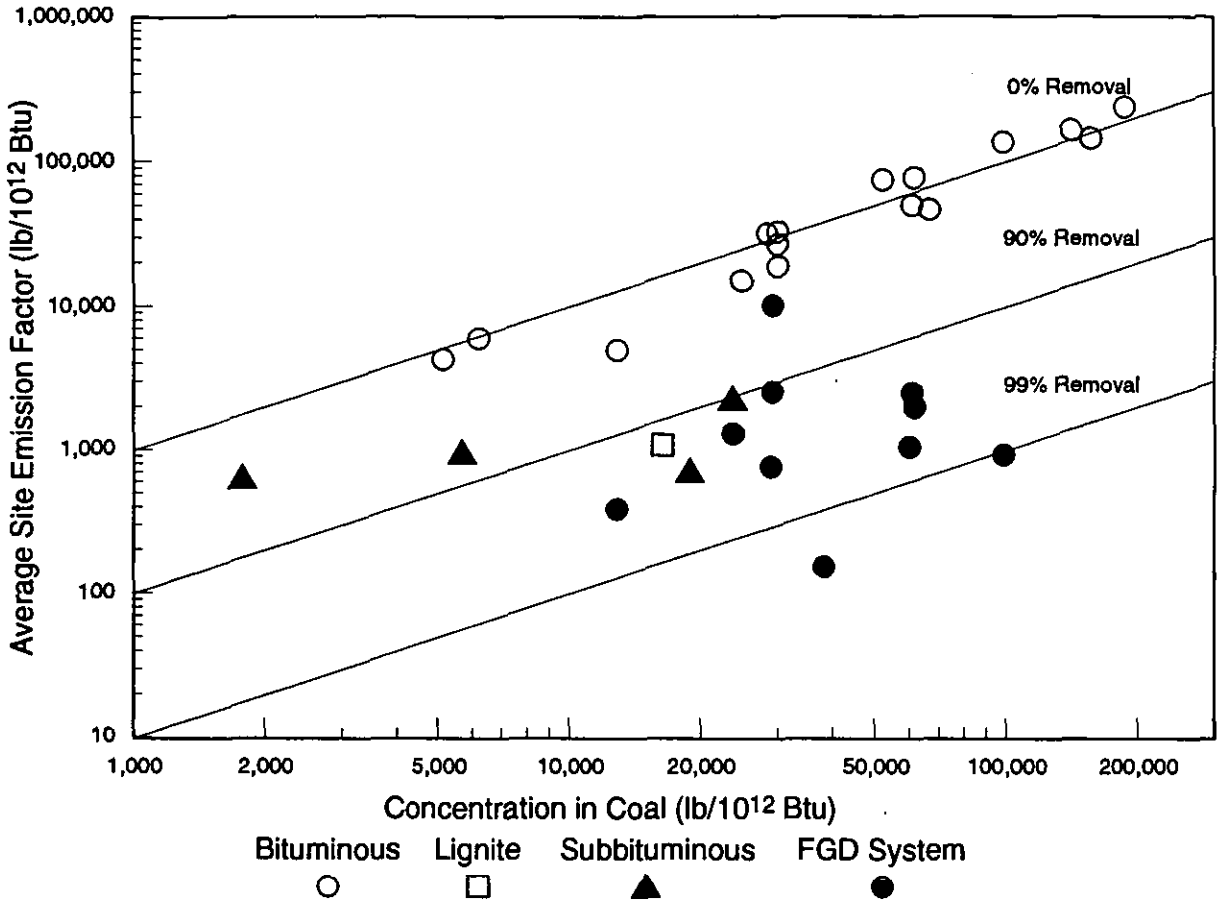


Figure B–16.
HCl Reduction, by Coal Type

B.2.1.3 Organic Substance Emissions

Five substances/classes of organic compounds are found in combustion flue gases that are on the CAAA list of hazardous air pollutants. The FCEM and DOE programs have collected data on volatile organics, aldehydes, semivolatile organics, and dioxin/furans. Specifically, benzene, toluene, formaldehyde, benzo(a)pyrene equivalents, and 2,3,7,8-p-tetrachloro-p-dioxin equivalents are of interest from a health perspective. Data from the site test reports have been compiled to permit the estimation of emissions from untested units.

Unlike the trace elements present in coal, the organic substances do not correlate well with particulate matter or control devices. (Data on CO levels, O_2 concentrations, etc. are not available for many of the test sites. Furthermore, the measurement variability of trace organic substances often exceeds the mean value, making correlations impractical.) Both

dry particulate controls and FGD systems report high and low values. Therefore, all of the average site values have been pooled to estimate mean emission factors and confidence intervals. Log-normal distributions are appropriate for describing these sets of data.

Figure B-17 shows the rank-ordered emission factors of benzene from 22 sites. All reported and detection level values (using half of the detection limit) were used to obtain the sample population. Also shown on this figure is the cumulative frequency distribution calculated for benzene using the geometric mean and standard deviation for the measured benzene values. (The inter-site data distribution is not normally distributed. It cannot be demonstrated that it is not a log-normal distribution.) The geometric mean and its 95% confidence interval are also shown. The geometric mean value for benzene is 3.8 lb/10^{12} Btu. The 95% lower and upper estimates of the industry average are 1.6 and 8.8 lb/10^{12}Btu. Toluene exhibits a similar distribution. The geometric mean is 1.4, within a confidence interval of 0.7 to 3 lb/10^{12}Btu.

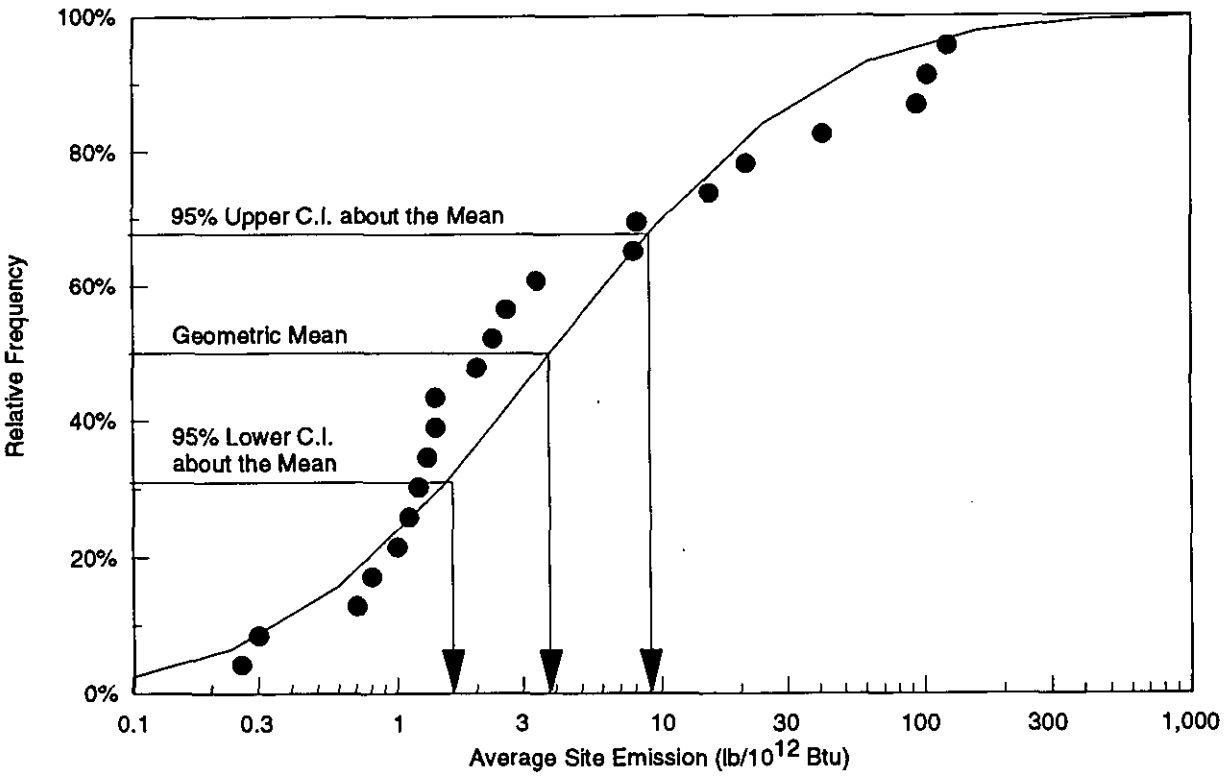


Figure B-17.
Benzene Emissions Distribution

Although it has been observed that FGD systems can absorb formaldehyde, the geometric means for particulate control-only units versus those with FGD systems were 2.8 and 3.7, respectively, with large confidence intervals. Therefore, these data were combined to provide a mean value of 3 lb/10^{12}Btu.

Figure B-18 presents the distribution of emission factors for 2,3,7,8-tetrachloro-p-dioxin equivalents in a similar fashion. Dioxin and PAH equivalency factors were calculated using the Method 23 protocol of multiplying the detected congeners (or PAH) concentration by the weighted equivalency factors per site. The International Toxicity Equivalent Factors adopted by US EPA [3] were used for the dioxin/furan congeners. For PAHs, carcinogenic factors relative to benzo(a)pyrene were taken from Krewald, et al. [4]. Table B-9 presents the recommended emission factors for the five organic substances and classes for coal-fired power plants.

Table B-9.
Organic Substance Emission Factors for Coal, lb/10^{12} Btu

Organic Substance/Class	Measurements	Geometric Mean	95% C.I.
Benzene	23	3.8	1.6 - 8.8
Toluene	21	1.4	0.7 - 3
Formaldehyde	22	3	1.5 - 6
Benzo(a)pyrene equivalent	11	0.0018	0.0004 - 0.0082
2,3,7,8-tetrachloro-p-dioxin equivalent	9	0.000002	0.0000004 - 0.00001

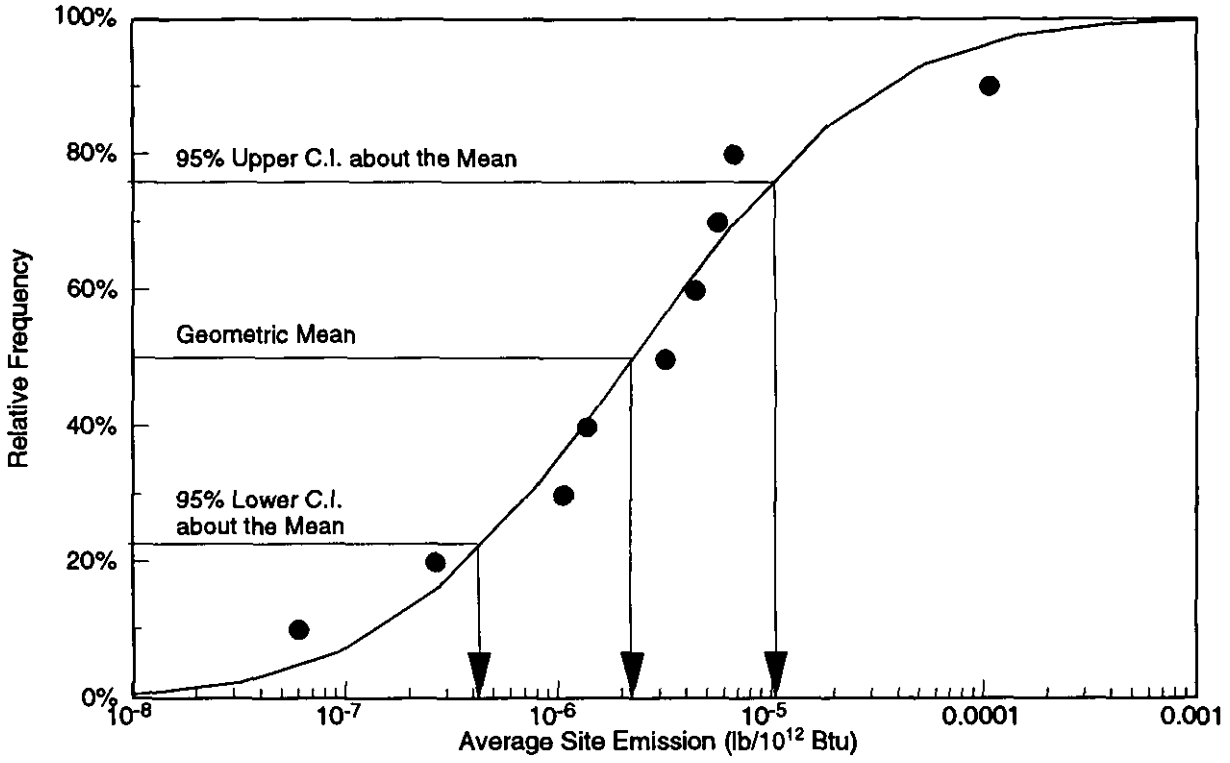


Figure B–18.
Dioxin Emissions Distribution (2,3,7,8–TCDD equivalents)

B.2.1.4 Radionuclides

Radionuclides were not initially included in the FCEM program for several reasons. First, during the 1980s, EPA and the industry conducted a detailed study of radionuclide emissions from coal-fired units and the risk to public health posed by these emissions. In 1989, EPA concluded that radionuclide emissions from coal-fired power plants "represent a level of risk that protects the public health with an ample margin of safety." [5] Only two FCEM sites and six DOE sites have been tested for radionuclides. The results from many of these sites exhibit high detection levels and, in some cases, high blank results.

Another reason that the FCEM program did not emphasize radionuclides is that a relatively large amount of radionuclide emission data exist because of activities related to the earlier EPA rulemaking. For example, during EPA's gathering of radionuclide data, stack fly ash samples were collected from five coal-fired power plants. Using the analytical results presented in the test report[6], a geometric mean U^{238} concentration of 5.2 pCi/g can be calculated for particulate matter in the stack.

In 1982–1983, the Utility Air Regulatory Group (UARG) sponsored a radionuclide sampling program during which stack fly ash samples were collected and analyzed from eight coal-fired plants[7]. These plants were selected to represent the range of commercial

coal-fired plants with respect to coal characteristics, furnace design, and particulate control technology. The generating capacities of the plants ranged from 100 to 750 MWe, and the time in commercial service at the time of sampling ranged from less than 1 to approximately 30 years. Uranium concentrations in the coal burned at these plants ranged from 0.45 to 2.4 mg/kg and averaged approximately 1 mg/kg.

Table B-10 presents the geometric mean and confidence interval for eight radionuclides in coal and emitted particulate matter using appropriate data from the FCEM and DOE test sites and from the UARG report. These data are internally consistent and are recommended as appropriate emissions estimates if the coal composition is not known for a particular plant. The radionuclide emission rate can be estimated by multiplying the particulate emission concentration (grams/gas volume) by the activity (pCi/gram). As can be seen, the average coal composition exhibits good secular equilibrium among the isotopes. The fine fraction of fly ash present in the stack gas shows greater enrichment of lead and polonium, relative to radium, thorium, and uranium.

Table B-10.
Coal Radionuclide Emission Values, pCi/gram

	Coal			Emitted Particulate Matter		
Isotope	Number of Values	Geometric Mean	95% C.I.	Number of Values	Geometric Mean	95% C.I.
Pb^{210}	20	0.6	0.3 - 1	7	10	4 - 23
Po^{210}	12	0.2	0.1 - 0.4	11	15	12 - 20
Ra^{226}	19	0.3	0.1 - 0.5	10	1.0	0.3 - 3.4
Ra^{228}	15	0.4	0.2 - 0.8	3	0.15	
Th^{228}	11	0.2	0.1 - 0.4	10	3.6	2 - 6
Th^{230}	12	0.4	0.2 - 0.7	10	6.2	3 - 13
Th^{232}	12	0.2	0.1 - 0.4	8	3.8	2.3 - 6.4
U^{234}	4	0.3	0.1 - 0.8	2	7	1 - 80
U^{235}	12	0.1	0.02 - 0.8	1	<0.3	
U^{238}	15	0.2	0.4 - 1.0	10	5.7	4.1 - 7.9

B.2.2 Results from Oil-Fired Units

Only about 25% of the oil-fired units employ particulate control in the form of mechanical collectors or ESPs. Most of the field data measurements are on uncontrolled units. The following discussion presents the field results by uncontrolled and then controlled test sites for the major classes of substances.

B.2.2.1 Uncontrolled Oil Sites

For units without particulate or SO_2 control devices, the emissions of trace substances should consistently match the mass rate present in the fuel, less whatever deposition occurs on the furnace walls and surfaces. Organic substance emissions are a function of operating conditions and not easily related to fuel measurements.

Figure B-19 presents a plot of the average fuel and emissions measured at the uncontrolled oil test sites for those substances present in detectable quantities in both the fuel oil and stack gas (this plot includes the metallic substances, as well as mercury, selenium and HCl). As can be seen, there is a fair degree of scatter for some data pairs. However, most substances found in the fuel are also emitted in approximately the same quantity. Consequently, the estimation of emissions from untested units for these substances could be based on 100% of the fuel oil analysis. Considering that 1) the concentration of substances in fuel oil varies by country of origin and degree of refining; 2) a nationwide database of oil compositions similar to the USGS coal database does not exist; and, 3) fuel oil consumers often purchase oil on spot markets rather than through long-term contracts, an emissions estimation approach that does not rely on oil analysis is more desirable. The obvious choice is the use of emission factors for trace substances.

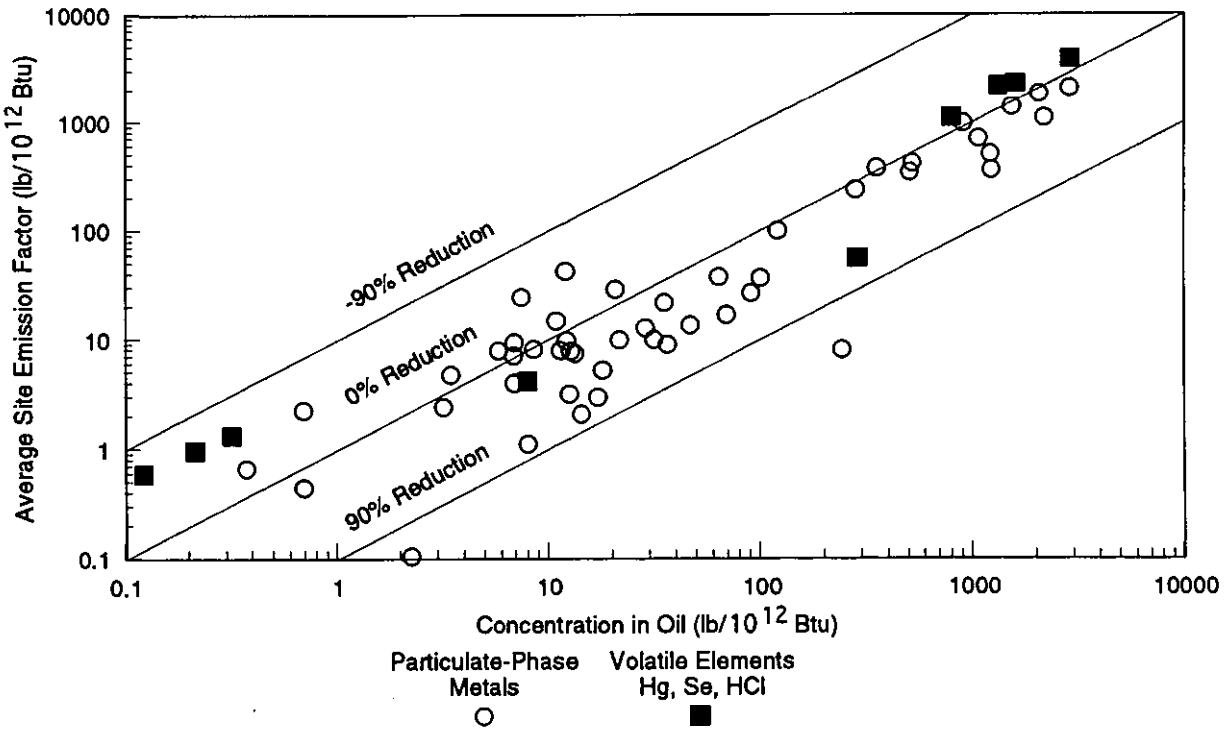


Figure B–19.
Uncontrolled Oil Unit Emissions

For this study, emission factors were developed using the following approach:

- Site-specific mean concentrations listed in the test reports were used to calculate the statistical parameters. For substances reported as not detected, one-half of the detection limit was used in the calculations.
- If one-half of the reported detection limit was greater than the highest quantified level for a substance, that result was not included in the calculation. This was done to prevent the average from being biased artificially high by abnormally high detection limits.[8]

The data sets for specific substances were examined to determine if they were normally distributed. For all substances except HCl, the substance specific emission factors were not normally distributed. Therefore, geometric means and confidence intervals were calculated for both elemental and organic substance emissions from uncontrolled oil-fired power plants. As an example of the log-normal distribution, Figure B-20 presents the average arsenic emissions per site, rank ordered, and the cumulative probability distribution for the statistical parameters from the data set. Table B-11 presents the geometric average emission factor and confidence interval values for the substances of interest. For the organic substances and the volatile elements (mercury, selenium, and HCl), the data from the four other oil sites with particulate controls were included in the distribution and statistics. The values reported for these substances at the outlet of a control device are similar to the uncontrolled emissions. Table B-11 shows the total number of site average values, number of sites where the substance was detected, and number of data sets used in the statistics for a substance.

Table B-11.
Oil-Fired Unit Emission Factors, lb/10^{12} Btu

Substances	Sites Tested	Sites Detected [a]	Sample Size [b]	Geometric Mean	95% C.I.
Arsenic	12	11	12	5.5	2.6 - 12
Beryllium	12	5	10	0.20	0.07 - 0.56
Cadmium	12	11	12	1.3	0.5 - 3.6
Chromium	13	12	13	5.2	3.0 - 8.9
Cobalt	6	6	6	37	16 - 86
HCl	11	11	11	2400	1900 - 3100
Lead	12	10	12	7.0	3.4 - 14
Manganese	12	12	12	13	7.9 - 23
Mercury	16	9	11	0.46	0.20 - 1.04
Nickel	13	13	13	720	460 - 1140

Table B-11.
Oil-Fired Unit Emission Factors, lb/10^{12} Btu (Continued)

Substances	Sites Tested	Sites Detected [a]	Sample Size [b]	Geometric Mean	95% C.I.
Selenium	16	10	12	2.0	0.7 - 5.5
Benzene	17	11	13	1.1	0.7 - 1.5
Formaldehyde	17	12	17	20	7.5 - 56
Toluene	11	11	11	9.9	4.8 - 20
Radionuclides	1	1	1	1.9 pCi/gram	
Benzo(a)pyrene equivalents	15	4	4	0.0038	0.0005 - 0.026
2,3,7,8-TCDD equivalents	4	3	3	0.0000083	0.0000014 - 0.00012

[a] Number of times the substance was quantified.

[b] The number of site values used to calculate the mean and confidence interval. (Individual values with high detection limits [>2x the highest quantified value] were not included in the mean.)

Addendum to Table B-11.
Emission Factors for Oil-Fired Unit, lb/10^{12} Btu
(400 MW residual oil unit with multiclones; data not received in time for inclusion in analyses)

	Oil (ppm)		Emission Factor	
Substances	Mean	95% C.I.	Mean	95% C.I.
Arsenic	<0.2		2.3	2.9
Beryllium	<0.002		<0.06	
Cadmium	0.0054	0.0057	0.3	0.15
Chromium	0.29	0.088	290	260
Cobalt	0.6	0.17	13.3	7.3
HCl	37	6.7	2500	910
Lead	1.6	1.9	34	14
Manganese	0.44	0.37	23	19
Mercury	0.0039	0.0021	0.95	0.78
Nickel	24.3	8.8	540	310
Selenium	0.115	0.058	6.9	12
Benzene			0.74	0.73
Formaldehyde			8.5	7.2
Toluene			25	2.7
Benzo(a)pyrene equivalents			<0.004	
HHV, Btu/lb	18217		34	
Sulfur (%)	1.5		0.3	
Ash (%)	0.05		0.014	

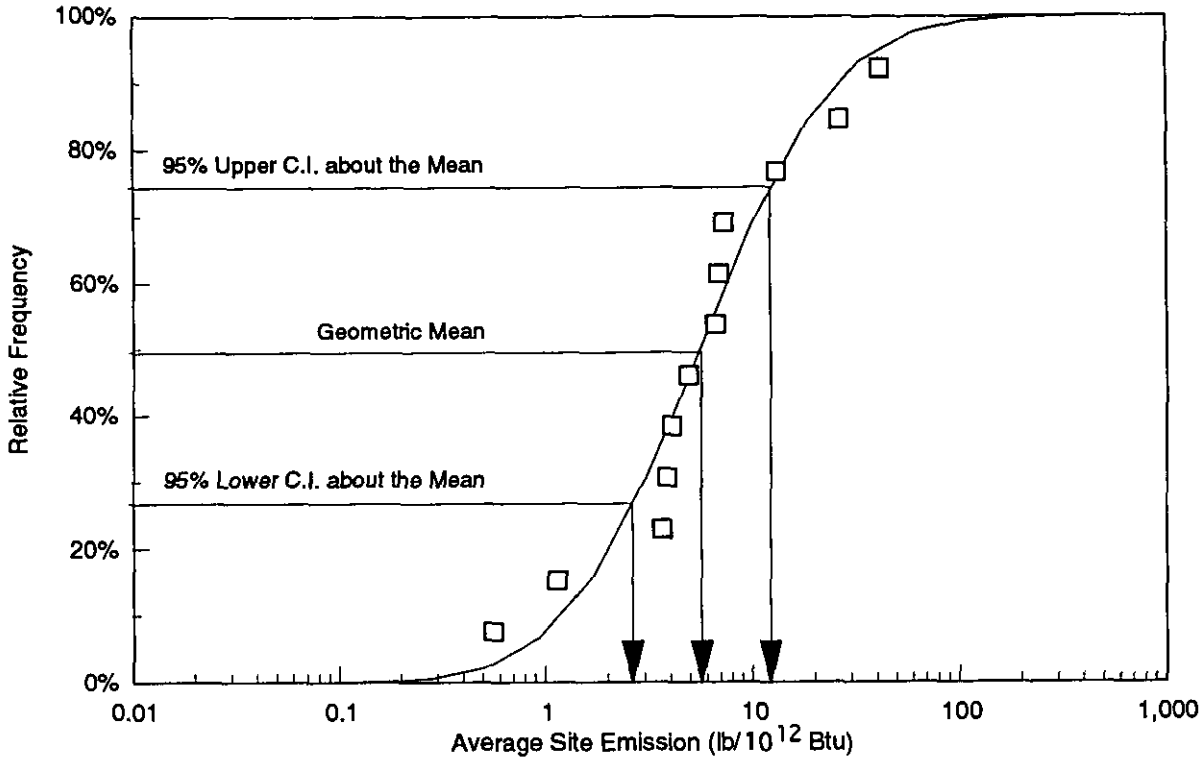


Figure B–20.
Arsenic Emissions Distribution for Uncontrolled Oil-Fired Boilers

B.2.2.2 Emissions from Particulate Controlled Oil Units

Three oil sites tested had normally operating ESPs (the other two sites employed pilot-scale SCR and PJFF units respectively). With this limited data set, the pairs of detected fuel/emission data and ESP inlet/emission data were used to calculate removal efficiencies for the nine trace metals of interest. The average reduction was about 40% between inlet and outlet gas streams. Therefore, for these nine metals, 60% of the values in Table B-8 should be used to estimate emissions from oil units with ESPs. For organic substances and volatile elements, the values in Table B-11 are appropriate.

B.2.3 Results from Gas-Fired Units

Limited test data are available from the FCEM program on emissions from gas-fired units. As shown in Table 3-1, some eight sites have been tested. At six sites, only benzene and formaldehyde measurements were made. A complete sampling and analytical effort was performed at two units. With these limited data, only arithmetic average values are presented, except for formaldehyde, in Table B-12. Most of the metal values reported had significant background levels in the filters, making these values questionable. Note that

for some substances, no current measurements exist, however, significant levels of those substances is not expected in gas-fired flue gas, based on the typical composition of natural gas.

Table B-12.
Gas-Fired Unit Emission Factors, lb/10^{12} Btu

Substances	Sites Tested	Sites Detected [a]	Sample Size [b]	Arithmetic Mean
Arsenic	2	2	2	0.23
Beryllium	2	0	0	<0.01
Cadmium	2	1	1	0.04
Chromium	2	2	2	1.1
Cobalt	2	1	1	0.08
HCl	0			NV [c]
Lead	2	2	2	0.4
Manganese	2	2	2	0.4
Mercury [d]	2	1	2	0.0008
Nickel	2	2	2	2.4
Selenium	2	0	0	<0.02
Benzene	8	2	5	0.8
Formaldehyde [e]	9	8	9	34(7-150)
Toluene	2	2	2	10
Radionuclides	0			NV
Benzo(a)pyrene equivalents	2	0	0	ND [f]
2,3,7,8-TCDD equivalents	1	1	1	0.0000012

[a] Number of times the substance was quantified.
[b] The number of site values used to calculate the mean and confidence interval. (Individual values with high detection limits [greater than twice the highest quantified value] were not included in the mean.)
[c] NV = No value.
[d] Based on natural gas analysis, not detected in stack gas at higher concentration.
[e] Geometric mean and confidence interval.
[f] ND = Not detected.

B.3 Data Treatment

The purpose of this data analysis was to develop suitable methods of estimating emissions from untested units so that risk assessments can be performed. Three types of estimates were used, depending on the substance of concern. Some substance emissions varied over many orders of magnitude. For these, correlations with input parameters proved appropriate. Other substances showed that certain coal types or control devices provided reasonable groupings within which average removal efficiencies could be estimated. Finally, some substances (notably the organic compounds) did not exhibit any functionality; therefore, average (either geometric or arithmetic) values were calculated from the data set. Discussed below are the general procedures followed in the development of the emission estimates.

B.3.1 Site Report Review

Most of the sites tested under the EPRI and DOE programs have been sampled three times for specific classes of substances. A single sample event typically requires a whole day therefore, three runs are indicative of plant operation on three days. If the data from all three runs is deemed valid (based on quality control results and other considerations) the best estimate of the plants emission is the arithmetic average of these three runs, bracketed by a suitable confidence interval. The three results include input, process, sampling, and analytical uncertainty. Often, the uncertainty in the mean value may be high, but unless there is a legitimate reason to discredit the information, it is used. Described below are the guidelines followed for:

- Combining analytical results of the multi-metal train; and,
- Averaging the results of replicate runs.

Several conventions have been developed for treating the test data and developing average concentrations of substances in the various streams.

To determine the total gas concentration for each run, the solid- and vapor-phase contributions were considered. However, the absence of some reportable concentrations in either (or both) phases required that conventions be developed for dealing with these data and formulating emission factors. These conventions are summarized below.

For each substance, there are three possible combinations of vapor- and solid-phase concentrations in the emitted gas stream. These are:

Case 1: The concentrations in both the solid and vapor phases are above detection limits.

Case 2: The concentrations of both the solid and vapor phase are below the detection limits.

Case 3: The concentration in one phase is above the detection limit, and the concentration in the other phase is below the detection limit.

For constituents of interest other than HCl, selenium, and mercury, the flue gas stream data indicates that most of the material is present in the solid phase and that only a small fraction is generally found in the vapor phase. Thus, the following conventions were selected for defining the total gas stream concentrations:

For Case 1, the total concentration is the sum of the concentrations in the vapor and solid phases.

For example, the total chloride concentration in the stack gas could be calculated as follows:

Cl in solid phase = 66 $\mu g/Nm^3$

Cl in vapor phase = 683 $\mu g/Nm^3$

Total Cl in stack gas = 749 $\mu g/Nm^3$

For Case 2, the total concentration is considered to be the detection limit in the solid phase.

For example, the total beryllium concentration in the stack gas may be as follows:

Be in the solid phase = ND(0.023) $\mu g/Nm^3$

Be in the vapor phase = ND(0.091) $\mu g/Nm^3$

Total Be in the stack gas = ND(0.023) $\mu g/Nm^3$

For Case 3, the total concentration is considered to be the one above the detection limit, regardless of which phase this represents.

For example, the arsenic concentration in the stack gas could be as follows:

As in solid phase = 0.11 $\mu g/Nm^3$

As in vapor phase = ND(0.11) $\mu g/Nm^3$

Total As in stack gas = 0.11 $\mu g/Nm^3$

The above conventions also are in accordance with guidance provided by EPA (*Technical Implementation Document for EPA's Boiler and Industrial Furnace Regulations*, U.S. Environmental Protection Agency, Office of Solid Waste, Washington, D.C., March 1992).

Testing at several sites has shown that HCl, selenium, and mercury are present primarily in the vapor phase. For Case 2, then, the total concentration is considered to be the reporting limit in the vapor phase. For Cases 1 and 3, the methods are unchanged from those described above.

- The following criteria were used to average the results of different runs:
- When all values for a given variable were above the method detection limit, the mean concentration was calculated as the true arithmetic mean.
- For results that include values both above and below the reporting limit, one-half the detection limit was used to calculate the mean. For example:

Analytical Values	Calculation	Mean Value
10, 12, ND(8)	[10+12+(8/2)]/3	8.7

By convention, the calculated mean is not allowed to be smaller than the largest detection limit value. In the following example, using one-half the detection limit would yield a calculated mean of 2.8. This mean value is less than the highest detection level obtained, so the reported mean is ND(4).

Analytical Values	Calculation	Mean Value
5, ND(4), ND(3)	[5+(4/2)+(3/2)]/3 = 2.8	ND(4)

- When all analytical results for a given variable are below the detection limit, the mean is reported as ND(x), where x is the largest detection limit. The bias estimate (used to calculate confidence intervals for other parameters) is one-half the detection limit, and no confidence interval is reported.

B.3.2 Calculated Streams

When data from multiple sites is compared, it must be done on an equivalent basis. Stack gas sampling involves the collection of a known volume of gas and the determination of the total mass of a substance by the collection media. The resulting concentration can be expressed as a mass per unit of gas volume. Unfortunately, the gas volumes used are not consistent. They vary from the actual stack gas conditions, to standard conditions, to Nm^3 conditions, to constant O_2 levels, depending on the report. By expressing the emission on a mass per unit of input energy, all of the gas volume inconsistencies are eliminated. Therefore, all mean emission levels from the site reports were collected on a unit-energy basis, typically in pounds per 10^{12} Btu heat input. (10^{12}, or one trillion, Btus is about 40,000 tons of bituminous coal.)

In addition, at many test sites, gas streams other than the stack gas were sampled. If these streams were equivalent to stack gas streams at other locations, their data was also compiled. For example, gas exiting an ESP and entering a FGD system at one plant is

comparable to gas from another plant controlled only be an ESP. If necessary, the gas concentrations reported for this type of internal stream may have been calculated on a unit-energy basis for inclusion.

B.3.3 PAH/Dioxin Equivalents

The suite of dioxin/furan congeners and the carcinogenic PAH compounds have been expressed in "equivalent" concentration form, relative to 2,3,7,8-tetrachlorodibenzo(p)dioxin and benzo(a)pyrene, respectively. These equivalent concentrations were developed by multiplying the average substance concentration at each site by its weighing factor and then summing the weighted concentrations. Table B-13 presents the dioxin/furan factors while Table B-14 presents the PAH factors.

Table B-13.
International Toxicity Equivalence Factors for Chlorinated Dioxins and Furans

Substance	International Toxicity Equivalent Factor
2,3,7,8-tetrachlorodibenzo(p)dioxin	1.0
1,2,3,7,8-pentachlorodibenzo(p)dioxin	0.5
1,2,3,4,7,8-hexachlorodibenzo(p)dioxin	0.1
1,2,3,6,7,8-hexachlorodibenzo(p)dioxin	0.1
1,2,3,7,8,9-hexachlorodibenzo(p)dioxin	0.1
1,2,3,4,6,7,8-heptachlorodibenzo(p)dioxin	0.01
Octachlorodibenzo(p)dioxin	0.001
2,3,7,8-tetrachlorodibenzofuran	0.1
1,2,3,7,8-pentachlorodibenzofuran	0.05
2,3,4,7,8-pentachlorodibenzofuran	0.5
1,2,3,4,7,8-hexachlorodibenzofuran	0.1
1,2,3,6,7,8-hexachlorodibenzofuran	0.1
1,2,3,7,8,9-hexachlorodibenzofuran	0.1
1,2,3,4,6,7,8-heptachlorodibenzofuran	0.01
1,2,3,4,7,8,9-heptachlorodibenzofuran	0.01
Octachlorodibenzofuran	0.001
Source: EPA 1989	

Table B-14.
PAH Equivalence Factors

Substance	Equivalence Factor
Benzo(a)anthracene	0.145
Benzo(b)fluoranthene	0.14
Benzo(j)fluoranthene	0.061
Benzo(k)fluoranthene	0.066
Benzo(a)pyrene	1.0
Chrysene	0.0044
Dibenzo(a,h)anthracene	1.11
Indeno(1,2,3-cd)pyrene	0.232
Source: Krewald, et al. (1985).	

B.3.4 Statistical Calculations

B.3.4.1 Means and Confidence Intervals for Stream Concentrations

The mean concentration and 95% confidence interval (C.I.) about the mean were calculated for each target substance in the fuel and gas stream. The means were calculated according to the conventions listed above. Example calculations are presented here for arsenic in the stack gas.

The concentration data (in $\mu g/Nm^3$) assumed for arsenic are:

	Run 1	Run 2	Run 3
Solid Phase	0.107	0.109	0.0932
Vapor Phase	ND(0.11)	ND(0.11)	ND(0.11)
Total	0.107	0.109	0.0932

The mean is calculated from the individual run totals (N=3):

Mean = (0.107+0.109+0.0932)/3

= 0.103

The sample standard deviation of the individual run totals is calculated:

$$S_p = \sqrt{\left[(0.107-0.103)^2 + (0.109-0.103)^2 + (0.0932-0.103)^2\right]/(N-1)}$$
$$= 0.00860$$

The standard deviation of the average is calculated for N = 3:

$$S_p = \frac{0.00860}{\sqrt{N}}$$
$$= 0.00497$$

The bias error is found by root-sum-squaring the product of the bias error and the sensitivity from each run. According to the conventions listed in above, no bias error is assigned to values above detection limits, whereas a bias error of one-half the detection limit is assigned to values below detection limits. The sensitivity of the mean to each run in this case is 1/N.

$$\beta_r = \sqrt{(1/3\times 0)^2 + (1/3\times 0)^2 + (1/3\times 0)^2}$$
$$= 0$$

The total uncertainty in the result is found calculated below, where the t statistic represents the 95% confidence limit for N=3 samples:

$$U_r = \sqrt{\beta^2 + \left(t\times S_p\right)^2}$$
$$= \sqrt{0^2 + (4.3\times 0.00497)^2}$$
$$= 0.02$$

Thus, the result is reported as $0.11 \pm 0.02\ \mu g/Nm^3$.

B.3.4.2 Test for Normal Distribution

For several substances, the range of data measurements span several orders of magnitude. Data with this variability, if it cannot be shown to be dependent upon an input parameter, is typically not normally distributed. Therefore, arithmetic averages and standard deviations are not the appropriate statistical descriptors of the data. When there are relatively few numbers for analysis (less than 50), statistical methods are employed to determine the probability that a data distribution does not fit a certain classification.

The W test of Shapiro and Wilk [9] can be used to demonstrate that the organic substance emission data are not normally distributed with a degree of confidence. Furthermore, when the W test is applied to the log of the organic emissions, the test does not disprove the hypothesis that the data are log-normal. Therefore, log-normal statistics were used to describe the emissions population. The log-normal technique permits the inclusion of several suspiciously high benzene values (hundred pounds per 10^{12} Btu) that have not been specifically refuted by QA/QC data in the site reports without significantly changing the average value.

B.3.4.3 Linear Regression and 95% Confidence Interval Calculation

For the particulate phase metal data, a linear regression in the log domain was used to develop the regression. The log of the ash-basis coal concentration multiplied by the particulate matter emission was used as the independent term, while the log of the measured emission factor was the dependent term. The additional statistical information in Table B-15 can be used to calculate the upper and lower confidence intervals for each data set and the regression. The equations below are used to calculate the upper and lower predictive bands. Definitions of these terms are in Table B-15. The confidence interval about the regression is calculated with these equations by not adding "1" in the exponential term.

$$Upper\ bound = y_i * 10^{t*RMSE*\sqrt{1+\frac{1}{N}+\frac{(\log x_i - \bar{x}\log x)^2}{SS_{\log x}}}}$$

$$Lower\ bound = y_i \Big/ 10^{t*RMSE*\sqrt{1+\frac{1}{N}+\frac{(\log x_i - \bar{x}\log x)^2}{SS_{\log x}}}}$$

Table B–15.
Summary of Models

Analyte	Predicted Emissions	R^2	N	Root MSE	t-Value	$xbar_{log}$	SS_{logx}
Antimony	$(0.92)x^{0.63}$	0.65	8	0.37	2.45	-0.30	3.8
Arsenic	$(3.1)x^{0.85}$	0.72	34	0.44	2.04	-0.006	27
Beryllium	$(1.2)x^{1.1}$	0.83	17	0.29	2.13	-0.26	4.8
Cadmium	$(3.3)x^{0.5}$	0.78	9	0.24	2.37	-0.55	7.8
Chromium	$(3.7)x^{0.58}$	0.57	38	0.40	2.03	0.31	23
Cobalt	$(1.7)x^{0.69}$	0.57	20	0.42	2.10	0.016	8.3
Lead	$(3.4)x^{0.80}$	0.62	33	0.48	2.04	0.061	18
Manganese	$(3.4)x^{0.60}$	0.57	37	0.39	2.03	0.70	18
Nickel	$(4.4)x^{0.48}$	0.51	25	0.49	2.07	0.28	25

x=Coal ppm/ash fraction * PM.
R^2: R-squared for the regression.
Root MSE: Square root of the mean squared error of the regression.
$xbar_{log}$: Mean of the log(xi)'s,
SS_{logx}: Sum of Squared Deviations of the log(xi)'s

B.4 Coal Site Test Data

A tabular summary of all coal data used is presented at the end of this section.

B.4.1 Metals

Figures B-21 through B-29 present the emissions of inorganic substances as a function of the independent parameter [(ashed coal concentration) times (particulate matter)]. Shown on these figures are the mean regression and confidence intervals about the data set and about the mean, calculated using the values in Table B-16.

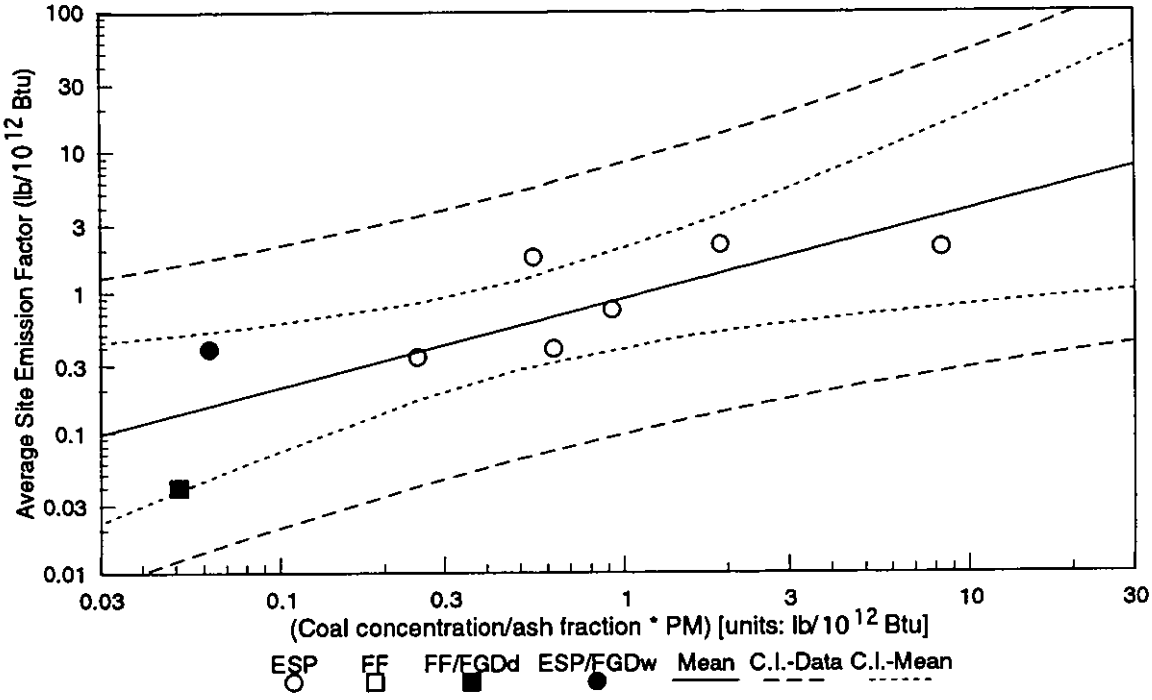


Figure B–21.
Antimony Correlation

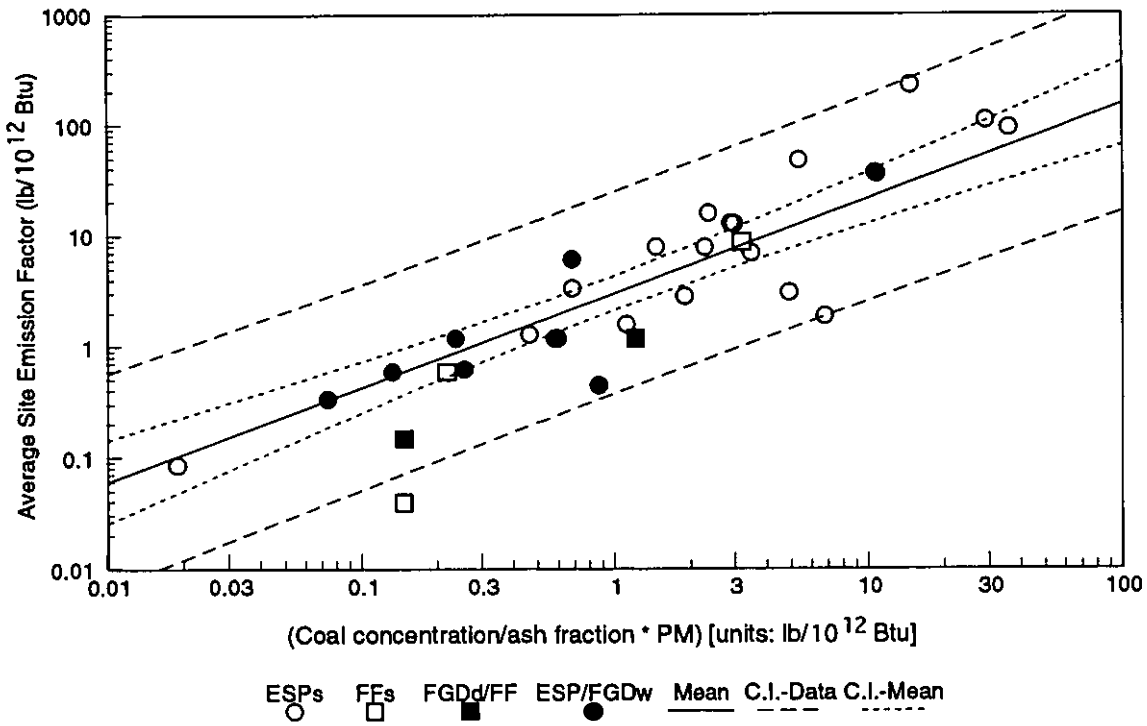


Figure B–22.
Arsenic Correlation

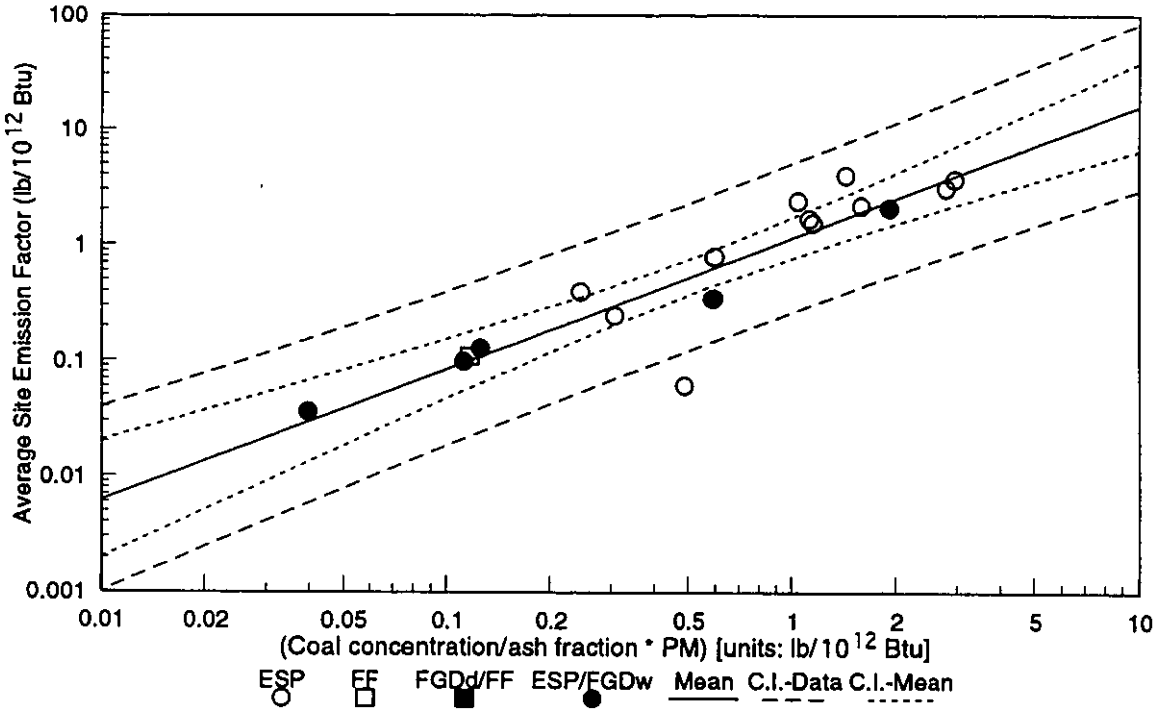


Figure B–23.
Beryllium Correlation

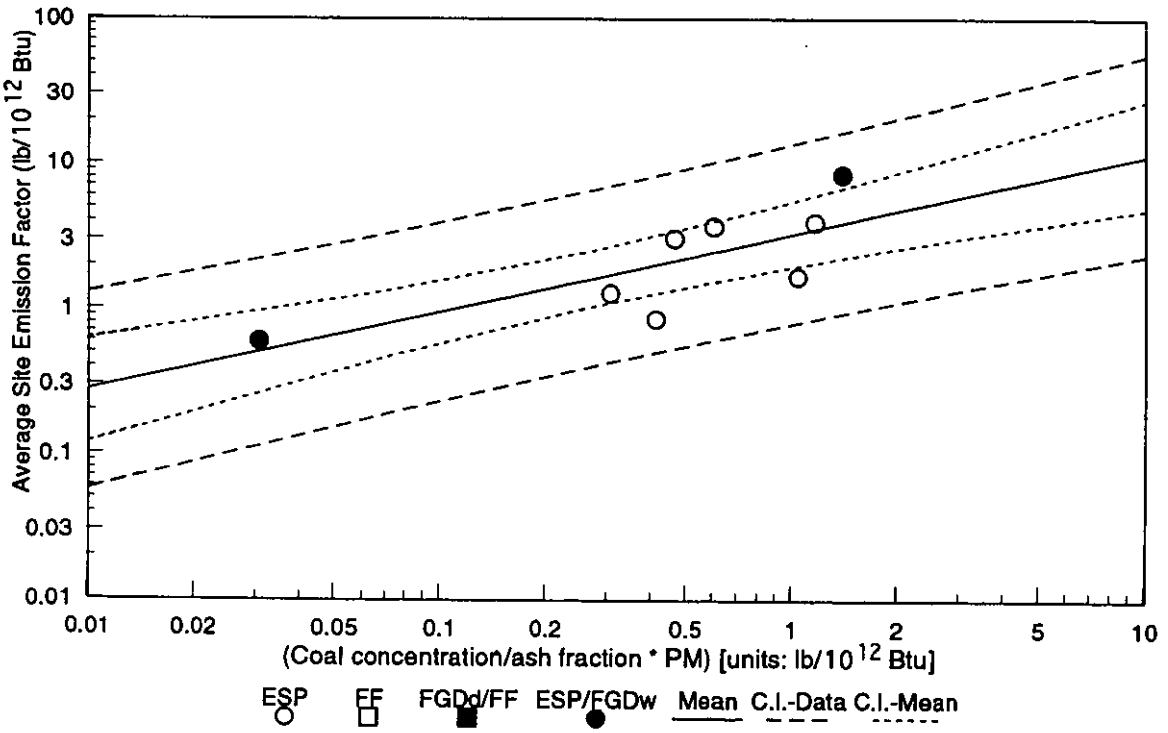


Figure B–24.
Cadmium Correlation

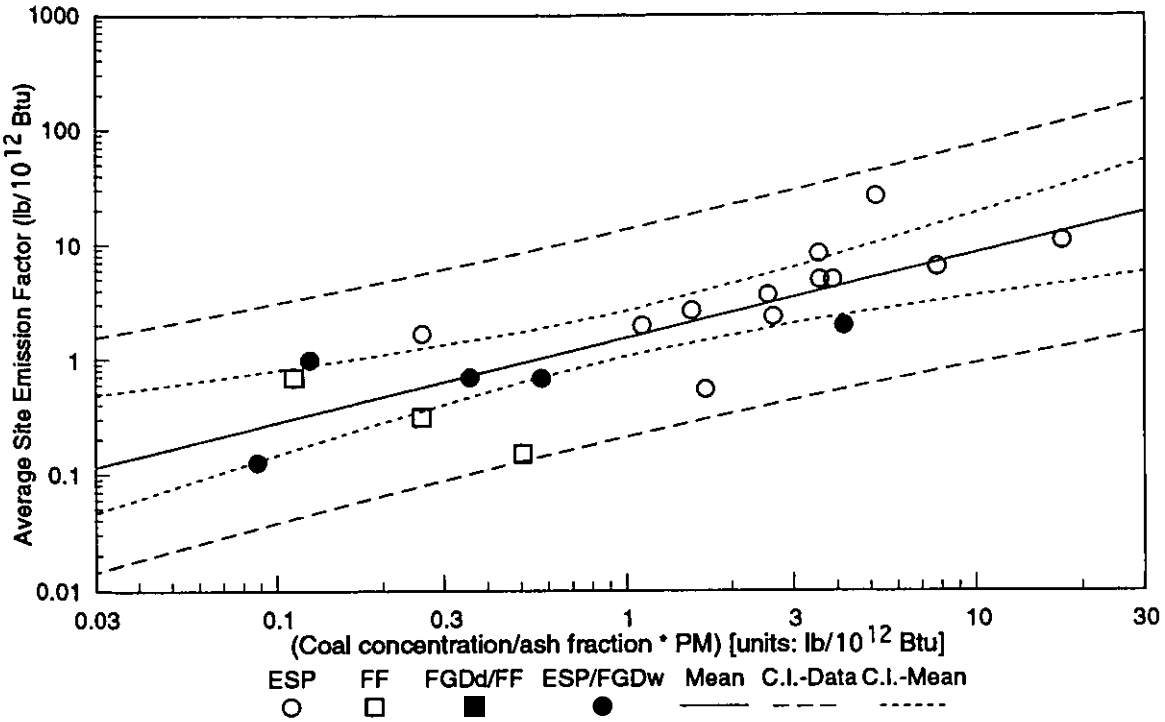


Figure B–25.
Cobalt Correlation

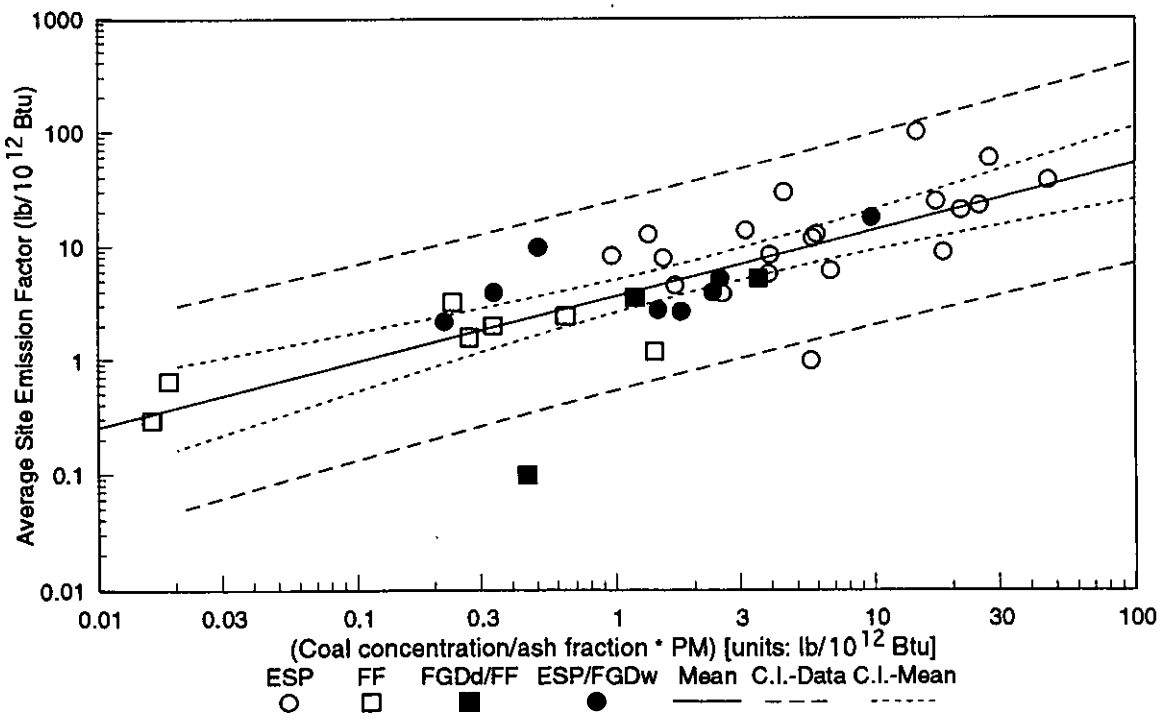


Figure B–26.
Chromium Correlation

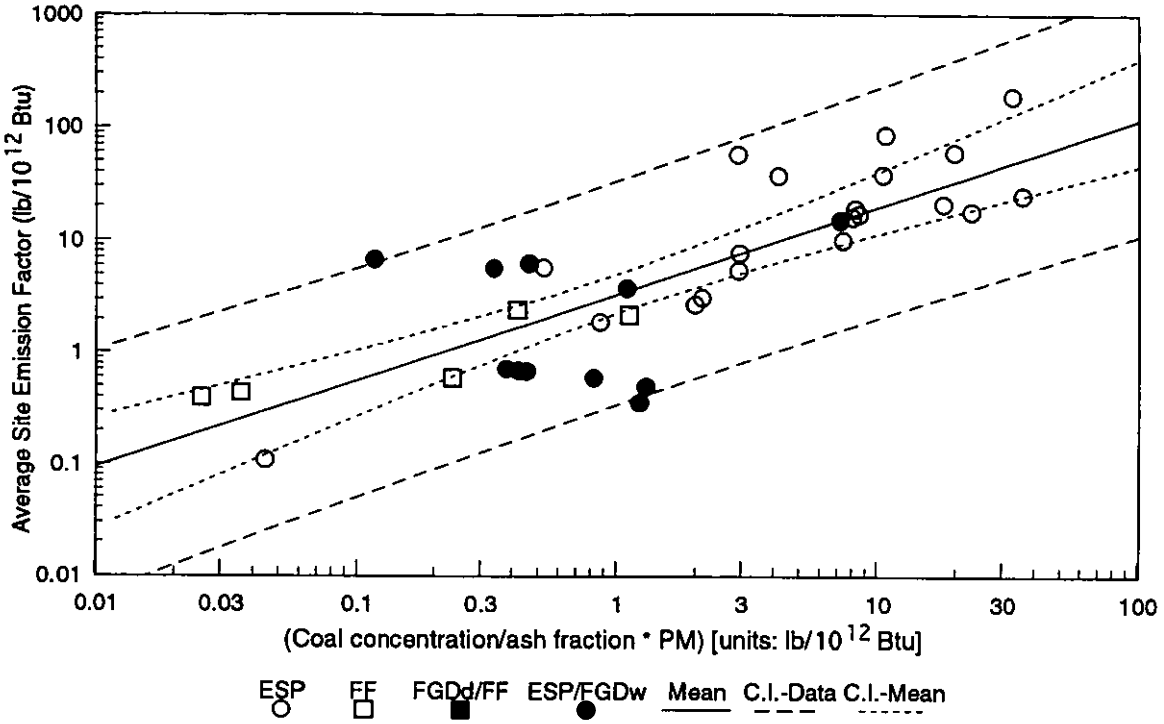


Figure B–27.
Lead Correlation

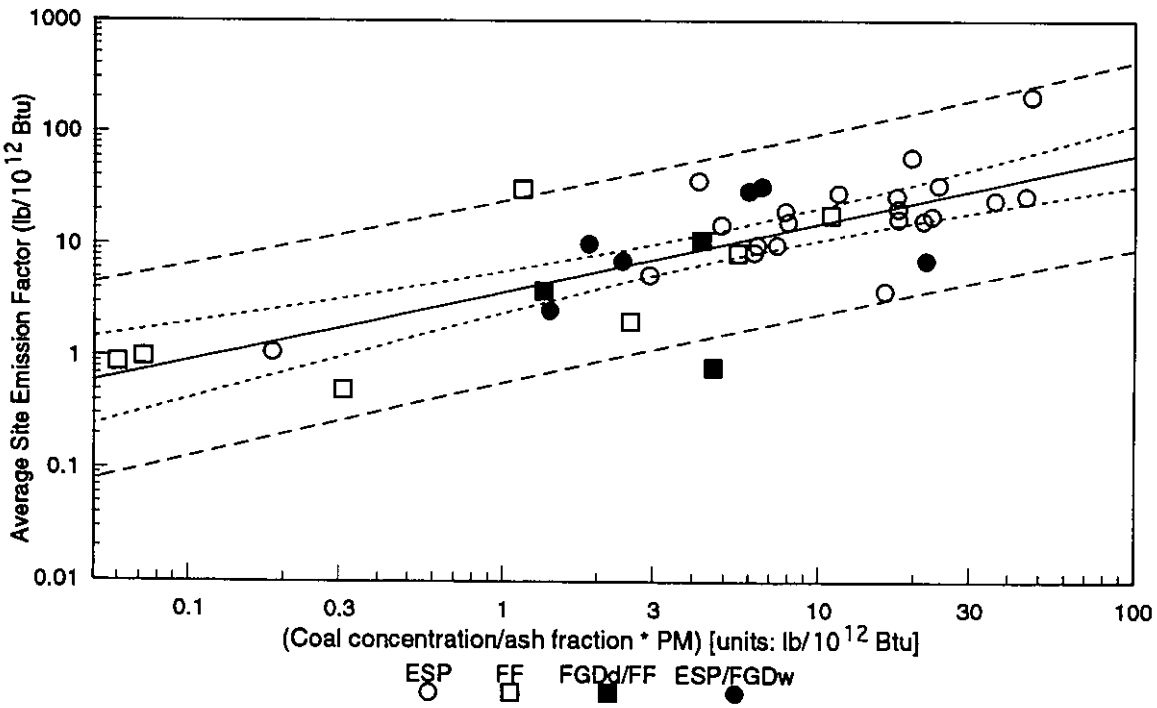


Figure B–28.
Manganese Correlation

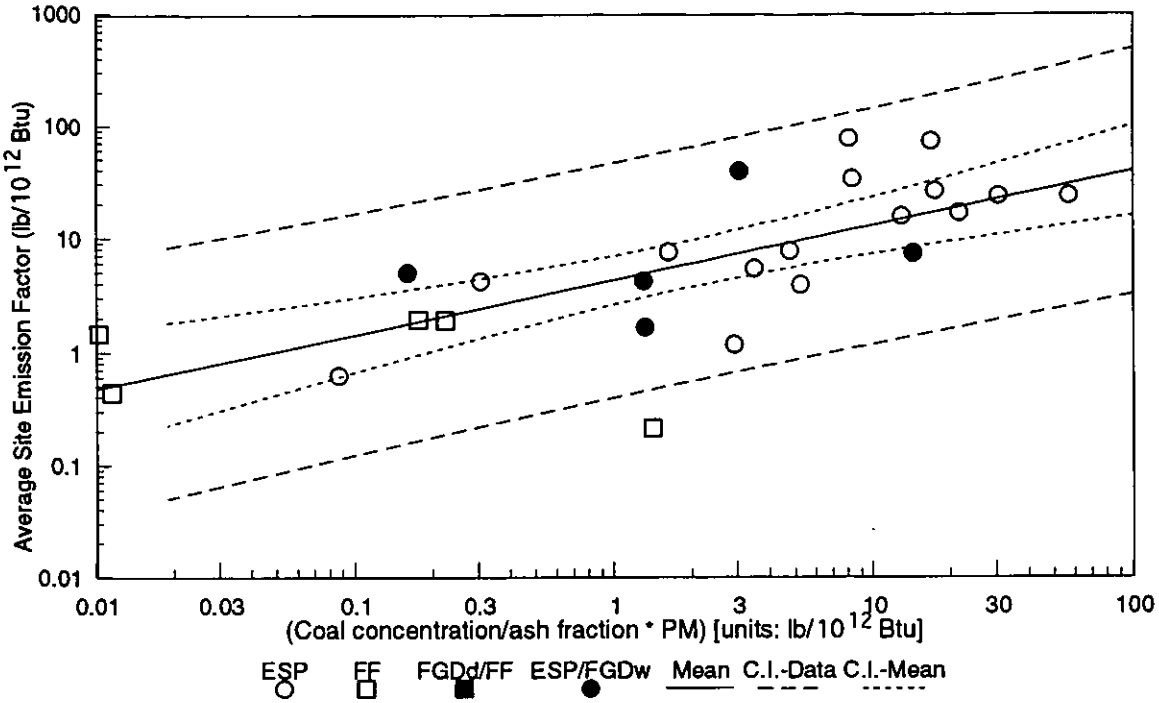


Figure B–29.
Nickel Correlation

B.4.2 Volatile Constituents

For mercury, HCl and selenium, nominal removal efficiencies are plotted on Figures B-30, B-31, and B-32. The average of the these removal levels has been used in Section 3 as the recommended emission factors.

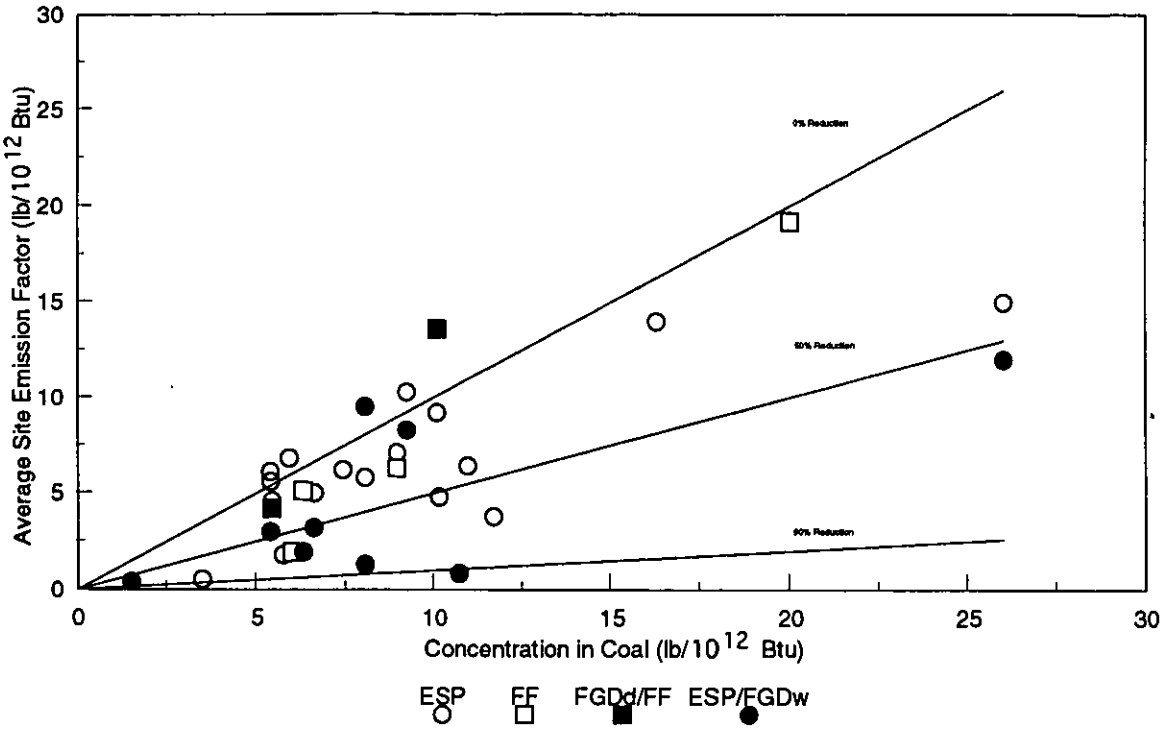


Figure B–30.
Mercury Data

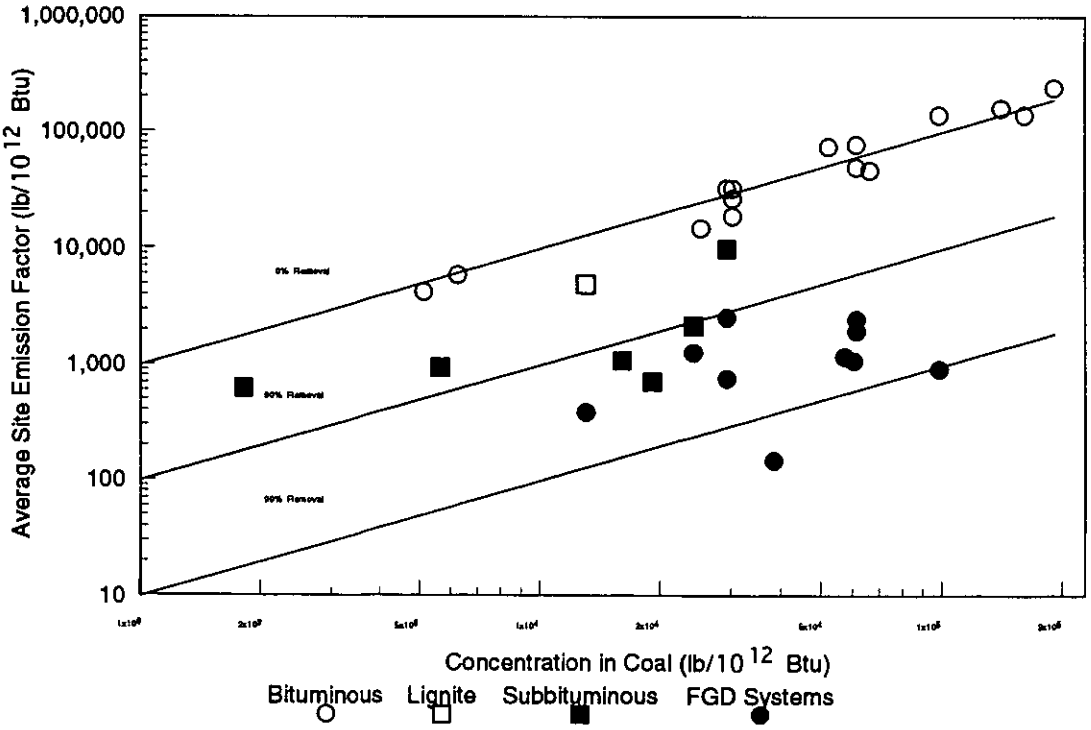


Figure B–31.
HCl Data

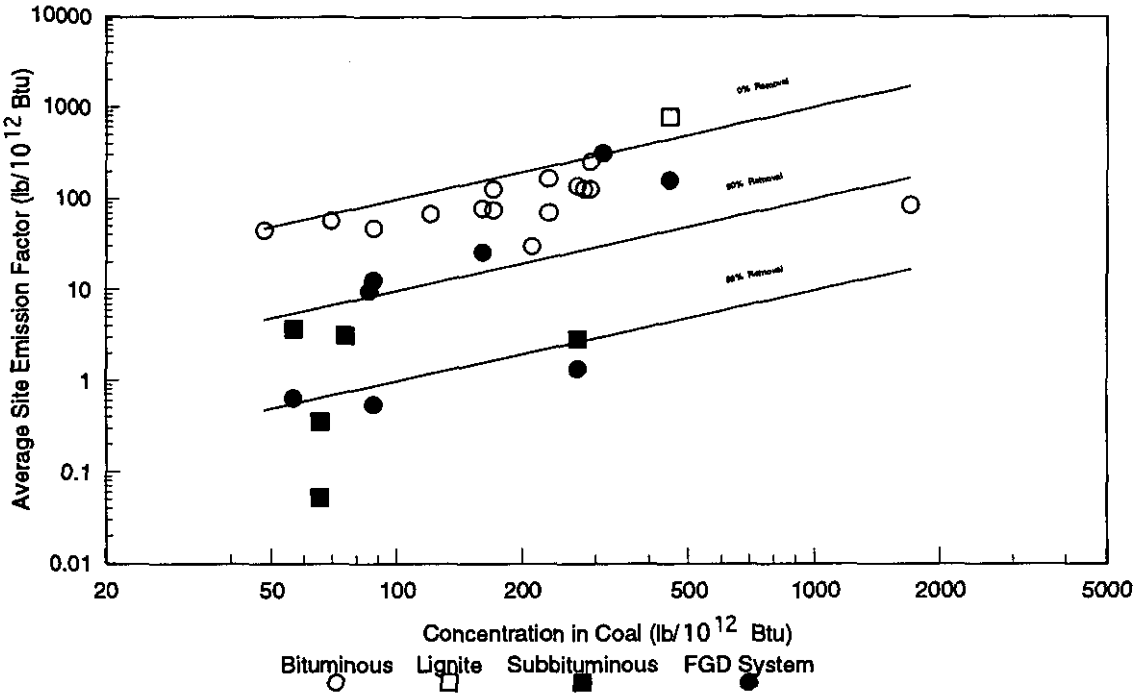


Figure B–32.
Selenium Data

B.4.3 Organic Substances

Figures B-33 and B-34 show the emission levels measured on a relative frequency plot for volatile organic substances (benzene, toluene, and formaldehyde) and benzo(a)pyrene/ 2,3,7,8-tetrachloro-p-dioxin equivalents, respectively. The geometric mean and standard deviation were used in the calculation of the emission factors for Section 3.

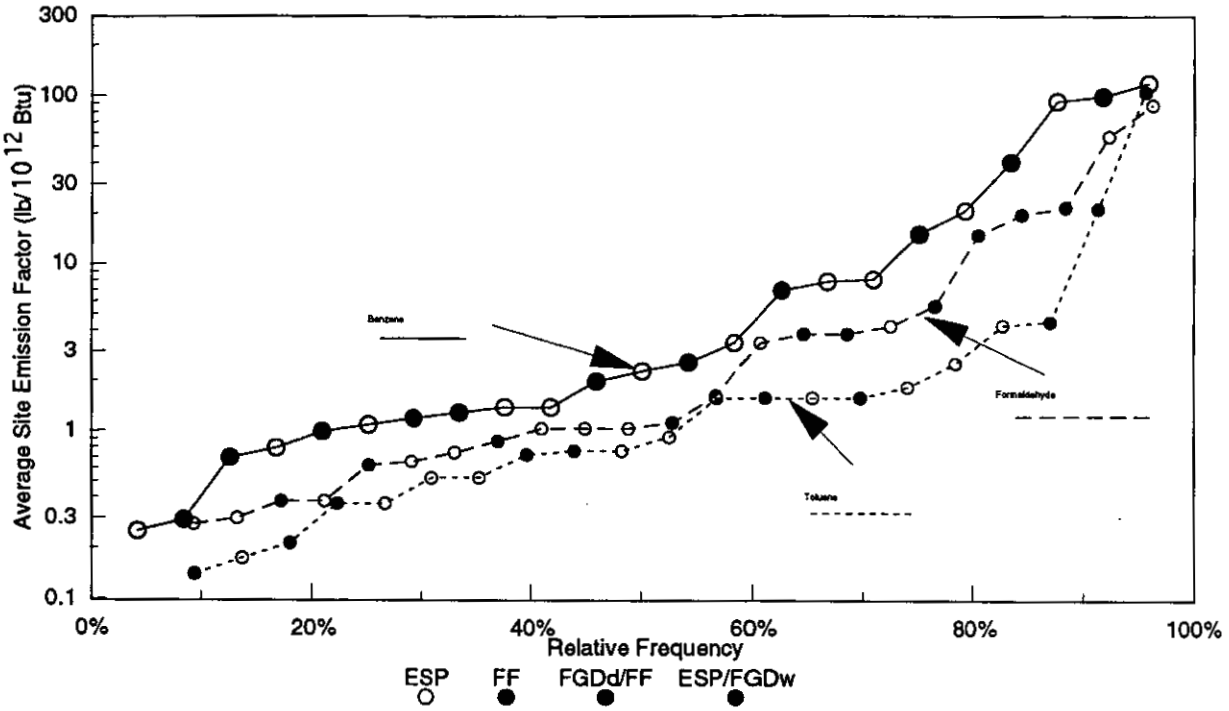


Figure B–33.
VOC Emission Distribution

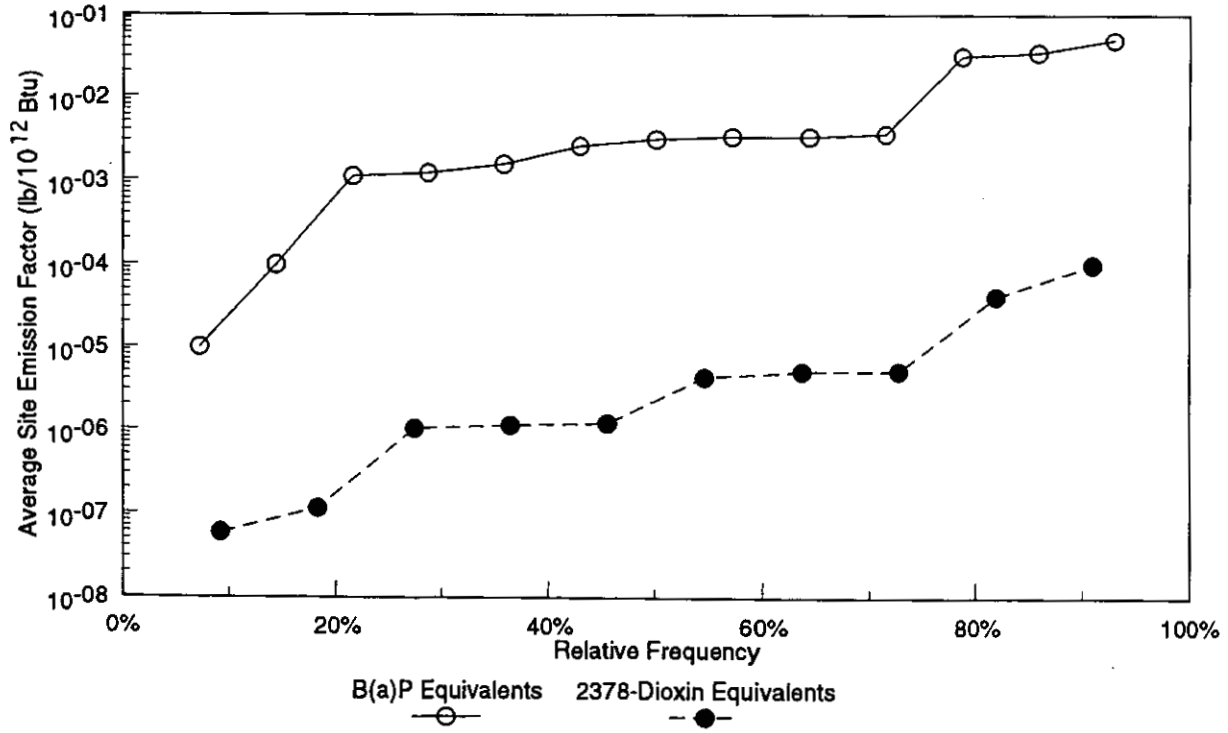


Figure B–34.
SVOC Emissions Distribution

B.4.4 Site Test Data

Table B-16 presents information from the individual site reports which has been used in this report to develop the recommended emissions estimation procedures presented in Section 3. Many of the columns in this table are common to multiple substances; however, they are presented to aid the reader in examining this information. The inorganic substance data are grouped in the following hierarchy: substance, final control device, coal rank, coal concentration. Mean values from the site reports are used in the data analysis. The emission factor is expressed in terms of pounds of substances per 10^{12} Btus of fuel input. The volatile organic substances of interest are presented in increasing order of emission level. The semivolatile organics (carcinogenic PAHs and dioxins/furans) are grouped by test site.

Table B-16.
Coal Site Measurements

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 20	Antimony	NM		<5.5		L	21.0%	10,000	2.2%	0.046	0.051
FGDw	Site 20	Antimony	NM		<5.2		L	21.0%	10,000	2.2%	0.017	0.019
ESP	Site 22	Antimony	0.13	0.06	<3.8		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 11	Antimony	0.13	0.07	<7.3		S	6.2%	11,900	0.6%	0.033	0.023
FGDw	Site 11	Antimony	0.13	0.01	<9.0		S	6.2%	11,900	0.6%	0.0063	0.0051
ESP	Site 12	Antimony	0.28	0.07	<7.3		B	9.4%	13,700	2.8%	0.12	0.10
FGDw	Site 12	Antimony	0.28	0.09	<8.5		B	9.4%	13,700	2.8%	0.014	0.013
FGDd	Site 14	Antimony	0.3	0.1	<9.0		B	9.3%	13,700	2.9%	0.0076	0.0073
FF	DOE 8	Antimony	0.5	0.1	<0.68		S	11.2%	11,700	0.9%	0.011	0.009
ESP	DOE 3	Antimony	0.57	0.05	1.8	0.36	B	12.0%	12,900	3.4%	0.15	0.11
FF	Site 101	Antimony	0.6	0.27	<0.26		S	25.0%	10,000	0.8%	0.024	0.021
FGDw	Site 101	Antimony	0.6	0.27	2.8	30	S	25.0%	10,000	0.8%	0.0083	0.0070
ESP	DOE 4	Antimony	0.61	0.16	0.40	0.1	B	13.8%	14,300	3.3%	0.14	0.14

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDw	DOE 4	Antimony	0.61	0.16	0.40	4.0	B	13.8%	14,300	3.3%	0.015	0.014
ESP	Site 19	Antimony	0.63	0.29	0.35	0.18	B	9.1%	13,500	0.9%	0.039	0.036
ESP	Site 15	Antimony	0.73	0.6	<3.4		B	12.7%	13,000	1.6%	0.037	0.028
FF	Site 18 FF	Antimony	0.85	0.23	<0.038		B	13.0%	13,400	1.1%	0.0017	0.0019
ESP	Site 18	Antimony	0.85	0.23	0.76	0.040	B	13.0%	13,400	1.1%	0.13	0.14
FF	DOE 2 FF	Antimony	0.9	0.7	<0.34		B	12.0%	13,000	2.6%	0.014	0.011
ESP	Site 116	Antimony	<1		0.10	0.090	B	12.0%	12,900	3.5%	0.056	0.046
FGDd	Site 116 SNRB	Antimony	<1		0.76	9.6	B	12.0%	12,900	3.5%	0.031	0.029
ESP	DOE 2	Antimony	1.1	0.88	<0.54		B	12.0%	12,900	2.8%	0.025	0.019
ESP	Site 16	Antimony	1.4	1.2	<25		B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 16L	Antimony	1.5	0.69	2.2	0.5	B	9.5%	13,800	1.7%	0.14	0.12
FGDd	DOE 7	Antimony	1.5	2	0.04	0.1	S	22.9%	10,500	0.7%	0.017	0.012

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 110B	Antimony	<2		<10		B	9.1%	11,900	2.9%	0.030	0.022
ESP	Site 110L	Antimony	<4		3.6	8.0	B	8.3%	12,100	2.9%	0.056	0.041
ESP	Site 102	Antimony	<10		<2.0		S	8.8%	12,200	1.0%	0.082	0.058
ESP	DOE 5	Antimony	22	50	2.1	3.2	B	11.4%	13,800	3.2%	0.047	0.043
FGDw	DOE 6	Antimony	<37		0.18	0.20	L	17.0%	9,970	1.1%	0.0094	0.0104
ESP	DOE 6	Antimony	<37		<1.1		L	17.0%	9,970	1.1%	0.020	0.020
FF	Site 10	Antimony	69	NA	<0.010		S	21.0%	11,000	0.5%	0.011	0.008
FF	Site 115	Arsenic	0.48	0.19	0.75	1.4	S	11.2%	12,565	0.6%	0.0025	0.0019
FGDd	Site 111	Arsenic	0.6	0.11	<0.21		S	14.0%	10,020	0.6%	0.011	0.041
FF	Site 115U	Arsenic	0.64	0.37	0.15	0.10	S	10.5%	12,638	0.5%	0.0015	0.0012
ESP	Site 22	Arsenic	0.85	0.18	0.087	0.019	S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 102	Arsenic	<1.3		2.9	1.3	S	8.8%	12,200	1.0%	0.082	0.058

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 11	Arsenic	1.6	0.5	1.2	10	S	6.2%	11,900	0.6%	0.033	0.023
FGDw	Site 11	Arsenic	1.6	0.5	0.60	3.0	S	6.2%	11,900	0.6%	0.0063	0.0051
FF	DOE 8	Arsenic	1.8	0.6	<0.32		S	11.2%	11,700	0.9%	0.011	0.009
ESP	Site 110B	Arsenic	1.9	0.51	1.3	0.90	B	9.1%	11,900	2.9%	0.030	0.022
ESP	Site 110L	Arsenic	<2		9.5	6.9	B	8.3%	12,100	2.9%	0.056	0.041
FGDw	DOE 4	Arsenic	2.3	1.4	1.2	0.20	B	13.8%	14,300	3.3%	0.015	0.014
ESP	DOE 4	Arsenic	2.3	1.4	16	6.0	B	13.8%	14,300	3.3%	0.14	0.14
FF	Site 101	Arsenic	2.6	0.8	0.59	0.39	S	25.0%	10,000	0.8%	0.024	0.021
FGDw	Site 101	Arsenic	2.6	0.8	0.34	0.85	S	25.0%	10,000	0.8%	0.0083	0.0070
FF	Site 10	Arsenic	2.8	NA	<1.0		S	21.0%	11,000	0.5%	0.011	0.008
ESP	Site 20	Arsenic	2.8	2	3.4	2.9	L	21.0%	10,000	2.2%	0.046	0.051
FGDw	Site 20	Arsenic	2.8	2	0.63	0.34	L	21.0%	10,000	2.2%	0.017	0.019
FGDd	DOE 7	Arsenic	2.9	0.5	0.15	0.6	S	22.9%	10,500	0.7%	0.017	0.012

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 3	Arsenic	3.1	0.9	13	22	B	12.0%	12,900	3.4%	0.15	0.11
FGDw	Site 21	Arsenic	4.3	3	6.2	0.80	B	6.7%	14,000	1.6%	0.013	0.011
ESP	Site 116	Arsenic	5	2.5	2.9	5.8	B	12.0%	12,900	3.5%	0.056	0.046
FGDd	Site 116 SNRB	Arsenic	5	2.5	1.2	12	B	12.0%	12,900	3.5%	0.031	0.029
ESP	Site 19	Arsenic	5.7	1.7	7.9	2.9	B	9.1%	13,500	0.9%	0.039	0.036
ESP	Site 12	Arsenic	6.2	1.4	1.9	4.8	B	9.4%	13,700	2.8%	0.12	0.10
FGDw	Site 12	Arsenic	6.2	1.4	0.45	0.30	B	9.4%	13,700	2.8%	0.014	0.013
FGDd	Site 14	Arsenic	6.3	4.9	<0.50		B	9.3%	13,700	2.9%	0.0076	0.0073
ESP	Site 114R	Arsenic	8.1	9	8.0	4.6	B	8.9%	13,300	1.8%	0.020	0.016
ESP	DOE 6	Arsenic	9.4	2	1.6	0.8	L	17.0%	9,970	1.1%	0.020	0.020
FGDw	DOE 6	Arsenic	9.4	2	1.2	1.5	L	17.0%	9,970	1.1%	0.0094	0.0104
FF	Site 18 FF	Arsenic	10	3.4	0.031	0.036	B	13.0%	13,400	1.1%	0.0017	0.0019

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 18	Arsenic	10	3.4	36	12	B	13.0%	13,400	1.1%	0.13	0.14
FGDw	Site 125	Arsenic	11	2	48	10	B	9.8%	13,043	3.0%	0.12	0.09
ESP	Site 122	Arsenic	11	0.7	230	310	B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 114B	Arsenic	12	23	7.0	13	B	8.7%	13,500	1.8%	0.040	0.025
ESP	DOE 5	Arsenic	13	10	3.1	3.5	B	11.4%	13,800	3.2%	0.047	0.043
ESP	Site 15	Arsenic	13	5	13	5.3	B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 16	Arsenic	17	8	94	430	B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 16L	Arsenic	23	2.3	110	56	B	9.5%	13,800	1.7%	0.14	0.12
ESP	DOE 2	Arsenic	33	3.8	48	20	B	12.0%	12,900	2.8%	0.025	0.019
FF	DOE 2 FF	Arsenic	34	33	8.8	20	B	12.0%	13,000	2.6%	0.014	0.011
FGDw	Site 11	Beryllium	0.15	0.13	<0.20		S	6.2%	11,900	0.6%	0.0063	0.0051
ESP	Site 11	Beryllium	0.15	0.13	<1.0		S	6.2%	11,900	0.6%	0.033	0.023
ESP	Site 22	Beryllium	0.2	0.02	<0.031		S	6.8%	11,980	0.4%	0.0018	0.0015

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FF	Site 115	Beryllium	0.22	0.04	<0.020		S	11.2%	12,565	0.6%	0.0025	0.0019
FF	DOE 8	Beryllium	0.24	0.07	<0.13		S	11.2%	11,700	0.9%	0.011	0.009
FGDd	Site 14	Beryllium	0.45	0.56	<0.20		B	9.3%	13,700	2.9%	0.0076	0.0073
FF	Site 115U	Beryllium	0.55	0.59	<0.020		S	10.5%	12,638	0.5%	0.0015	0.0012
ESP	Site 110B	Beryllium	0.56	0.33	<0.50		B	9.1%	11,900	2.9%	0.030	0.022
FF	Site 10	Beryllium	0.6	NA	<0.20		S	21.0%	11,000	0.5%	0.011	0.008
ESP	Site 12	Beryllium	0.67	0.2	<0.14		B	9.4%	13,700	2.8%	0.12	0.10
FGDw	Site 12	Beryllium	0.67	0.2	<0.16		B	9.4%	13,700	2.8%	0.014	0.013
ESP	Site 116	Beryllium	0.67	0.38	<13		B	12.0%	12,900	3.5%	0.056	0.046
FGDw	DOE 6	Beryllium	0.67	0.15	<1.6		L	17.0%	9,970	1.1%	0.0094	0.0104
ESP	DOE 6	Beryllium	0.67	0.15	<1.7		L	17.0%	9,970	1.1%	0.020	0.020
FGDd	Site 116 SNRB	Beryllium	0.67	0.38	<15		B	12.0%	12,900	3.5%	0.031	0.029

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDw	Site 21	Beryllium	0.79	0.07	0.13	0.030	B	6.7%	14,000	1.6%	0.013	0.011
ESP	Site 102	Beryllium	0.9	0.1	<4.4		S	8.8%	12,200	1.0%	0.082	0.058
FGDw	DOE 4	Beryllium	1.1	0.01	0.10	0.040	B	13.8%	14,300	3.3%	0.015	0.014
ESP	DOE 4	Beryllium	1.1	0.01	1.7	NC	B	13.8%	14,300	3.3%	0.14	0.14
ESP	Site 122	Beryllium	1.1	1.3	4.0	1.5	B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 15	Beryllium	1.1	0.5	0.40	0.030	B	12.7%	13,000	1.6%	0.037	0.028
FGDd	DOE 7	Beryllium	1.2	0.25	<0.040		S	22.9%	10,500	0.7%	0.017	0.012
ESP	DOE 3	Beryllium	1.2	0.12	1.6	0.20	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 5	Beryllium	1.3	1	0.062	0.070	B	11.4%	13,800	3.2%	0.047	0.043
ESP	Site 110L	Beryllium	1.3	1.1	<0.60		B	8.3%	12,100	2.9%	0.056	0.041
FGDw	Site 101	Beryllium	1.4	0.4	0.036	0.014	S	25.0%	10,000	0.8%	0.0083	0.0070
FF	Site 101	Beryllium	1.4	0.4	0.11	0.04	S	25.0%	10,000	0.8%	0.024	0.021
ESP	Site 16	Beryllium	1.4	0.93	3.7	3.8	B	10.0%	13,700	1.6%	0.25	0.21

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 2	Beryllium	1.9	0.8	0.25	0.033	B	12.0%	12,900	2.8%	0.025	0.019
FGDw	Site 125	Beryllium	2.2	0.4	2.1	0.1	B	9.8%	13,043	3.0%	0.12	0.09
ESP	Site 16L	Beryllium	2.2	0.72	3.1	1.5	B	9.5%	13,800	1.7%	0.14	0.12
FF	DOE 2 FF	Beryllium	2.3	0.6	<0.064	0.033	B	12.0%	13,000	2.6%	0.014	0.011
ESP	Site 114R	Beryllium	3.3	0.4	0.80	1.3	B	8.9%	13,300	1.8%	0.020	0.016
ESP	Site 114B	Beryllium	3.6	0.4	2.4	2.0	B	8.7%	13,500	1.8%	0.040	0.025
FGDw	Site 20	Beryllium	6.5	3.9	0.35	0.18	L	21.0%	10,000	2.2%	0.017	0.019
ESP	Site 20	Beryllium	6.5	3.9	2.2	3.1	L	21.0%	10,000	2.2%	0.046	0.051
FF	Site 115	Cadmium	<0.05		0.12	0.24	S	11.2%	12,565	0.6%	0.0025	0.0019
FGDd	DOE 7	Cadmium	<0.05		0.026	0.05	S	22.9%	10,500	0.7%	0.017	0.012
FF	Site 115U	Cadmium	<0.06		<0.070		S	10.5%	12,638	0.5%	0.0015	0.0012
FF	DOE 8	Cadmium	0.08	0.02	<0.65		S	11.2%	11,700	0.9%	0.011	0.009

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDw	Site 101	Cadmium	<0.1		0.40	0.8	S	25.0%	10,000	0.8%	0.0083	0.0070
FF	Site 101	Cadmium	<0.1		0.53	1.2	S	25.0%	10,000	0.8%	0.024	0.021
FGDw	Site 21	Cadmium	<0.1		0.57	0.29	B	6.7%	14,000	1.6%	0.013	0.011
ESP	Site 22	Cadmium	<0.1		0.16	0.27	S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 16	Cadmium	<0.11		0.50	2.7	B	10.0%	13,700	1.6%	0.25	0.21
ESP	DOE 5	Cadmium	<0.13		0.80	2.8	B	11.4%	13,800	3.2%	0.047	0.043
FGDw	Site 20	Cadmium	<0.14		0.70	0.68	L	21.0%	10,000	2.2%	0.017	0.019
ESP	Site 20	Cadmium	<0.14		0.52	0.18	L	21.0%	10,000	2.2%	0.046	0.051
ESP	DOE 4	Cadmium	0.3	0.01	1.3	NC	B	13.8%	14,300	3.3%	0.14	0.14
ESP	Site 116	Cadmium	<0.3		<13		B	12.0%	12,900	3.5%	0.056	0.046
FF	DOE 2 FF	Cadmium	<0.3		<0.056		B	12.0%	13,000	2.6%	0.014	0.011
ESP	DOE 2	Cadmium	<0.3		<0.14		B	12.0%	12,900	2.8%	0.025	0.019
FGDw	DOE 4	Cadmium	0.3	0.01	0.60	0.80	B	13.8%	14,300	3.3%	0.015	0.014

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDd	Site 116 SNRB	Cadmium	<0.3		0.60	6.0	B	12.0%	12,900	3.5%	0.031	0.029
ESP	Site 19	Cadmium	<0.4		0.13	0.20	B	9.1%	13,500	0.9%	0.039	0.036
FF	Site 18 FF	Cadmium	0.43	0.52	0.23	0.13	B	13.0%	13,400	1.1%	0.0017	0.0019
ESP	Site 18	Cadmium	0.43	0.52	3.1	6.3	B	13.0%	13,400	1.1%	0.13	0.14
ESP	DOE 3	Cadmium	0.63	0.14	3.7	0.26	B	12.0%	12,900	3.4%	0.15	0.11
FGDw	DOE 6	Cadmium	<0.9		<3.0		L	17.0%	9,970	1.1%	0.0094	0.0104
ESP	DOE 6	Cadmium	<0.9		<3.0		L	17.0%	9,970	1.1%	0.020	0.020
ESP	Site 122	Cadmium	0.9	0.7	4.0	1.5	B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 102	Cadmium	<1		<0.20		S	8.8%	12,200	1.0%	0.082	0.058
FGDw	Site 125	Cadmium	1.6	0.5	8.5	1.8	B	9.8%	13,043	3.0%	0.12	0.09
ESP	Site 110B	Cadmium	1.7	0.59	0.86	0.50	B	9.1%	11,900	2.9%	0.030	0.022
FGDw	Site 11	Cadmium	<2		1.3	5.3	S	6.2%	11,900	0.6%	0.0063	0.0051

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 11	Cadmium	<2		1.5	1.9	S	6.2%	11,900	0.6%	0.033	0.023
FF	Site 10	Cadmium	<2		<0.40		S	21.0%	11,000	0.5%	0.011	0.008
ESP	Site 110L	Cadmium	2.1	0.11	1.7	3.0	B	8.3%	12,100	2.9%	0.056	0.041
ESP	Site 16L	Cadmium	<2.6		3.6	2.4	B	9.5%	13,800	1.7%	0.14	0.12
FGDd	Site 14	Cadmium	<3		1.0	3.0	B	9.3%	13,700	2.9%	0.0076	0.0073
ESP	Site 12	Cadmium	<3.4		1.8	3.7	B	9.4%	13,700	2.8%	0.12	0.10
FGDw	Site 12	Cadmium	<3.4		1.2	1.1	B	9.4%	13,700	2.8%	0.014	0.013
FGDd	Site 111	Cadmium	<4		<2.1		S	14.0%	10,020	0.6%	0.011	0.041
ESP	Site 15	Cadmium	<8		3.1	9.0	B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 114B	Cadmium	39	160	1.8	5.0	B	8.7%	13,500	1.8%	0.040	0.025
ESP	Site 114R	Cadmium	47	89	0.40	1.3	B	8.9%	13,300	1.8%	0.020	0.016
ESP	DOE 5	Chlorine (as chloride)	NM		23,000	7,200	B	11.4%	13,800	3.2%	0.047	0.043

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 6	Chlorine (as chloride)	0.4	1.3	2,900	1,700	L	17.0%	9,970	1.1%	0.020	0.020
FGDw	DOE 6	Chlorine (as chloride)	0.4	1.3	1,300	710	L	17.0%	9,970	1.1%	0.0094	0.0104
FF	DOE 2 FF	Chlorine (as chloride)	3.9	4	150,000	9,800	B	12.0%	13,000	2.6%	0.014	0.011
ESP	DOE 2	Chlorine (as chloride)	3.9	2.4	130,000	9,800	B	12.0%	12,900	2.8%	0.025	0.019
FF	Site 115	Chlorine (as chloride)	22	7.6	630	190	S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 10	Chlorine (as chloride)	62	NA	960	NA	S	21.0%	11,000	0.5%	0.011	0.008
ESP	Site 114B	Chlorine (as chloride)	69	100	4,300	740	B	8.7%	13,500	1.8%	0.040	0.025
ESP	Site 114R	Chlorine (as chloride)	82	26	6,000	1,400	B	8.9%	13,300	1.8%	0.020	0.016
ESP	Site 22	Chlorine (as chloride)	<100		730	220	S	6.8%	11,980	0.4%	0.0018	0.0015
FGDw	Site 20	Chlorine (as chloride)	128	83	390	610	L	21.0%	10,000	2.2%	0.017	0.019
ESP	Site 20	Chlorine (as chloride)	128	83	5,000	9,900	L	21.0%	10,000	2.2%	0.046	0.051
FF	Site 101	Chlorine (as chloride)	140	30	9,800	8,700	S	25.0%	10,000	0.8%	0.024	0.021

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FF	DOE 8	Chlorine (as chloride)	190	90	1,100	1,200	S	11.2%	11,700	0.9%	0.011	0.009
FF	Site 115U	Chlorine (as chloride)	238	24	720	210	S	10.5%	12,638	0.5%	0.0015	0.0012
ESP	Site 11	Chlorine (as chloride)	280	130	2,200	890	S	6.2%	11,900	0.6%	0.033	0.023
FGDw	Site 11	Chlorine (as chloride)	280	130	1,300	2,000	S	6.2%	11,900	0.6%	0.0063	0.0051
FGDw	Site 101	Chlorine (as chloride)	290	30	2,500	800	S	25.0%	10,000	0.8%	0.0083	0.0070
ESP	Site 16L	Chlorine (as chloride)	340	200	15,000	2,400	B	9.5%	13,800	1.7%	0.14	0.12
ESP	Site 116	Chlorine (as chloride)	373	87	33,000	5,000	B	12.0%	12,900	3.5%	0.056	0.046
FGDd	Site 116 SNRB	Chlorine (as chloride)	373	87	770	1,200	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	DOE 7	Chlorine (as chloride)	400	130	150	NA	S	22.9%	10,500	0.7%	0.017	0.012
ESP	Site 18	Chlorine (as chloride)	400	440	27,000	18,000	B	13.0%	13,400	1.1%	0.13	0.14
FF	Site 18 FF	Chlorine (as chloride)	400	440	33,000	27,000	B	13.0%	13,400	1.1%	0.0017	0.0019
ESP	Site 16	Chlorine (as chloride)	410	230	19,000	2,400	B	10.0%	13,700	1.6%	0.25	0.21

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 19	Chlorine (as chloride)	700	310	75,000	53,000	B	9.1%	13,500	0.9%	0.039	0.036
FGDw	Site 125	Chlorine (as chloride)	740	460	1200	300	B	9.8%	13,043	3.0%	0.12	0.09
ESP	DOE 3	Chlorine (as chloride)	790	90	78,000	830	B	12.0%	12,900	3.4%	0.15	0.11
FGDd	Site 14	Chlorine (as chloride)	817	110	1,100	2,600	B	9.3%	13,700	2.9%	0.0076	0.0073
ESP	Site 12	Chlorine (as chloride)	830	190	50,000	7,100	B	9.4%	13,700	2.8%	0.12	0.10
FGDw	Site 12	Chlorine (as chloride)	830	190	2,500	730	B	9.4%	13,700	2.8%	0.014	0.013
FGDw	Site 21	Chlorine (as chloride)	860	110	2,000	880	B	6.7%	14,000	1.6%	0.013	0.011
ESP	Site 15	Chlorine (as chloride)	864	220	47,000	7,600	B	12.7%	13,000	1.6%	0.037	0.028
ESP	DOE 4	Chlorine (as chloride)	1,200	100	140,000	63,000	B	13.8%	14,300	3.3%	0.14	0.14
FGDw	DOE 4	Chlorine (as chloride)	1,200	100	930	850	B	13.8%	14,300	3.3%	0.015	0.014
ESP	Site 110L	Chlorine (as chloride)	1,700	160	160,000	13,000	B	8.3%	12,100	2.9%	0.056	0.041
ESP	Site 110B	Chlorine (as chloride)	1,850	140	140,000	52,000	B	9.1%	11,900	2.9%	0.030	0.022
ESP	Site 122	Chlorine (as chloride)	2,500	300	240,000	16,000	B	9.3%	13,370	2.0%	0.17	0.12

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FF	Site 115	Chromium	1.1	0.6	0.66	0.48	S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 115U	Chromium	1.4	1.4	0.30	0.19	S	10.5%	12,638	0.5%	0.0015	0.0012
ESP	Site 22	Chromium	4	0.1	<0.50		S	6.8%	11,980	0.4%	0.0018	0.0015
FGDw	Site 11	Chromium	4.1	2	4.0	21	S	6.2%	11,900	0.6%	0.0063	0.0051
ESP	Site 11	Chromium	4.1	2	8.0	90	S	6.2%	11,900	0.6%	0.033	0.023
FF	DOE 8	Chromium	4.2	0.7	2.0	1.1	S	11.2%	11,700	0.9%	0.011	0.009
ESP	Site 110B	Chromium	5.5	2	13	9.0	B	9.1%	11,900	2.9%	0.030	0.022
ESP	Site 102	Chromium	6	1	8.5	3.5	S	8.8%	12,200	1.0%	0.082	0.058
FF	Site 10	Chromium	7.1	NA	1.6	NA	S	21.0%	11,000	0.5%	0.011	0.008
FF	Site 101	Chromium	7.7	3.8	2.5	1.3	S	25.0%	10,000	0.8%	0.024	0.021
FGDw	Site 101	Chromium	7.7	3.8	2.2	4.3	S	25.0%	10,000	0.8%	0.0083	0.0070
FGDd	Site 111	Chromium	8	1.4	<4.3		S	14.0%	10,020	0.6%	0.011	0.041
ESP	DOE 6	Chromium	8.2	4	8.4	11	L	17.0%	9,970	1.1%	0.020	0.020

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDw	DOE 6	Chromium	8.2	4	10	78	L	17.0%	9,970	1.1%	0.0094	0.0104
FGDd	DOE 7	Chromium	8.9	0.75	0.10	0.15	S	22.9%	10,500	0.7%	0.017	0.012
ESP	Site 110L	Chromium	9	0.86	30	30	B	8.3%	12,100	2.9%	0.056	0.041
ESP	Site 114R	Chromium	9.4	2	4.6	5.5	B	8.9%	13,300	1.8%	0.020	0.016
ESP	Site 114B	Chromium	11	12	14	13	B	8.7%	13,500	1.8%	0.040	0.025
ESP	Site 122	Chromium	11	4	100	89	B	9.3%	13,370	2.0%	0.17	0.12
FGDw	Site 125	Chromium	11	2	18	12	B	9.8%	13,043	3.0%	0.12	0.09
FGDw	Site 21	Chromium	11	0.6	2.7	0.40	B	6.7%	14,000	1.6%	0.013	0.011
FGDd	Site 116 SNRB	Chromium	15	2.9	5.3	65	B	12.0%	12,900	3.5%	0.031	0.029
ESP	Site 116	Chromium	15	2.9	1.0	2.0	B	12.0%	12,900	3.5%	0.056	0.046
FF	DOE 2 FF	Chromium	15	2.5	1.2	1.6	B	12.0%	13,000	2.6%	0.014	0.011
FGDd	Site 14	Chromium	15	5.7	3.6	7.0	B	9.3%	13,700	2.9%	0.0076	0.0073

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 19	Chromium	15	3.8	13	5.0	B	9.1%	13,500	0.9%	0.039	0.036
FF	Site 18 FF	Chromium	16	14	3.3	12	B	13.0%	13,400	1.1%	0.0017	0.0019
ESP	Site 20	Chromium	16	2.3	5.8	3.6	L	21.0%	10,000	2.2%	0.046	0.051
FGDw	Site 20	Chromium	16	2.3	2.8	1.8	L	21.0%	10,000	2.2%	0.017	0.019
ESP	Site 18	Chromium	16	14	25	2.8	B	13.0%	13,400	1.1%	0.13	0.14
ESP	DOE 2	Chromium	16	2.5	3.9	1.6	B	12.0%	12,900	2.8%	0.025	0.019
ESP	Site 16L	Chromium	17	8.7	21	7.5	B	9.5%	13,800	1.7%	0.14	0.12
ESP	Site 12	Chromium	17	4	9.0	80	B	9.4%	13,700	2.8%	0.12	0.10
FGDw	Site 12	Chromium	17	4	4.0	20	B	9.4%	13,700	2.8%	0.014	0.013
ESP	DOE 5	Chromium	18	10	6.2	15	B	11.4%	13,800	3.2%	0.047	0.043
ESP	Site 16	Chromium	22	8.6	38	32	B	10.0%	13,700	1.6%	0.25	0.21
ESP	DOE 4	Chromium	25	2.9	23	NC	B	13.8%	14,300	3.3%	0.14	0.14
FGDw	DOE 4	Chromium	25	2.9	5.3	50	B	13.8%	14,300	3.3%	0.015	0.014

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 15	Chromium	26	9	12	15	B	12.7%	13,000	1.6%	0.037	0.028
ESP	DOE 3	Chromium	29	4	59	14	B	12.0%	12,900	3.4%	0.15	0.11
FGDw	Site 21	Cobalt	NM		4.1	2.7	B	6.7%	14,000	1.6%	0.013	0.011
ESP	Site 110L	Cobalt	<0.5		3.0	0.40	B	8.3%	12,100	2.9%	0.056	0.041
FF	Site 115	Cobalt	0.9	0.30	<0.22		S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 115U	Cobalt	1.3	0.46	<0.23		S	10.5%	12,638	0.5%	0.0015	0.0012
FF	DOE 8	Cobalt	1.4	0.15	0.70	0.5	S	11.2%	11,700	0.9%	0.011	0.009
ESP	Site 22	Cobalt	1.4	0.30	<0.70		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 11	Cobalt	1.5	1.2	<0.80		S	6.2%	11,900	0.6%	0.033	0.023
FGDw	Site 11	Cobalt	1.5	1.2	1.0	7.1	S	6.2%	11,900	0.6%	0.0063	0.0051
FF	Site 10	Cobalt	1.6	NA	<0.80		S	21.0%	11,000	0.5%	0.011	0.008
FGDw	DOE 6	Cobalt	2.2	1.3	<1.5		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDd	Site 14	Cobalt	2.2	1	<1.0		B	9.3%	13,700	2.9%	0.0076	0.0073

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 6	Cobalt	2.2	1.3	<1.6		L	17.0%	9,970	1.1%	0.020	0.020
FGDd	Site 116 SNRB	Cobalt	2.3	1.4	<18		B	12.0%	12,900	3.5%	0.031	0.029
ESP	Site 116	Cobalt	2.3	1.4	<16		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 12	Cobalt	3	2.2	<3.5		B	9.4%	13,700	2.8%	0.12	0.10
FGDw	Site 12	Cobalt	3	2.2	<1.0		B	9.4%	13,700	2.8%	0.014	0.013
FF	Site 101	Cobalt	3.1	1.8	0.32	0.04	S	25.0%	10,000	0.8%	0.024	0.021
FGDw	Site 101	Cobalt	3.1	1.8	0.13	0.16	S	25.0%	10,000	0.8%	0.0083	0.0070
FGDd	DOE 7	Cobalt	3.3	0.83	<0.30		S	22.9%	10,500	0.7%	0.017	0.012
ESP	DOE 4	Cobalt	3.5	1.9	5.0	NC	B	13.8%	14,300	3.3%	0.14	0.14
FGDw	DOE 4	Cobalt	3.5	1.9	0.70	0.80	B	13.8%	14,300	3.3%	0.015	0.014
ESP	DOE 3	Cobalt	3.7	0.07	8.4	0.76	B	12.0%	12,900	3.4%	0.15	0.11
ESP	Site 122	Cobalt	4	2	27	7.2	B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 102	Cobalt	4	1	2.4	1.5	S	8.8%	12,200	1.0%	0.082	0.058

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 5	Cobalt	4.5	10	0.55	0.40	B	11.4%	13,800	3.2%	0.047	0.043
FGDw	Site 125	Cobalt	4.8	1.5	<2.0		B	9.8%	13,043	3.0%	0.12	0.09
ESP	Site 15	Cobalt	5	2	2.0	0.80	B	12.7%	13,000	1.6%	0.037	0.028
FF	DOE 2 FF	Cobalt	5.4	1.3	0.15	0.4	B	12.0%	13,000	2.6%	0.014	0.011
ESP	Site 16L	Cobalt	6.1	1.7	6.5	2.9	B	9.5%	13,800	1.7%	0.14	0.12
FGDw	Site 20	Cobalt	6.3	3.3	0.69	0.54	L	21.0%	10,000	2.2%	0.017	0.019
ESP	Site 20	Cobalt	6.3	3.3	2.7	2.8	L	21.0%	10,000	2.2%	0.046	0.051
ESP	DOE 2	Cobalt	6.3	3.8	<0.21		B	12.0%	12,900	2.8%	0.025	0.019
ESP	Site 19	Cobalt	6.4	0.64	3.7	2.0	B	9.1%	13,500	0.9%	0.039	0.036
ESP	Site 16	Cobalt	8.3	1.4	11	11	B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 110B	Cobalt	16	0.92	5.0	10	B	9.1%	11,900	2.9%	0.030	0.022
FGDw	Site 11	Lead	1.4	0.7	6.8	65	S	6.2%	11,900	0.6%	0.0063	0.0051
ESP	Site 11	Lead	1.4	0.7	5.8	11	S	6.2%	11,900	0.6%	0.033	0.023

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 22	Lead	2	0.1	0.11	0.010	S	6.8%	11,980	0.4%	0.0018	0.0015
FGDd	Site 14	Lead	2	0.6	<0.30		B	9.3%	13,700	2.9%	0.0076	0.0073
FF	Site 115	Lead	2.1	1.2	0.44	0.090	S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 115U	Lead	2.2	1.3	0.40	0.050	S	10.5%	12,638	0.5%	0.0015	0.0012
ESP	Site 12	Lead	2.4	0.09	9.7	7.0	B	9.4%	13,700	2.8%	0.12	0.10
FGDw	Site 12	Lead	2.4	0.09	5.7	32	B	9.4%	13,700	2.8%	0.014	0.013
FGDw	Site 21	Lead	2.9	0.35	6.3	6.0	B	6.7%	14,000	1.6%	0.013	0.011
ESP	Site 102	Lead	3	1	2.7	1.6	S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 15	Lead	4	1.3	4.3	4.1	B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 110B	Lead	<5		16	26	B	9.1%	11,900	2.9%	0.030	0.022
ESP	Site 16	Lead	5.1	1.1	33	34	B	10.0%	13,700	1.6%	0.25	0.21
FF	DOE 8	Lead	5.2	1.2	2.4	2.7	S	11.2%	11,700	0.9%	0.011	0.009
ESP	Site 116	Lead	5.3	1.4	<0.20		B	12.0%	12,900	3.5%	0.056	0.046

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDd	Site 116 SNRB	Lead	5.3	1.4	0.50	5.0	B	12.0%	12,900	3.5%	0.031	0.029
FF	Site 10	Lead	6.1	NA	0.60	NA	S	21.0%	11,000	0.5%	0.011	0.008
ESP	DOE 5	Lead	6.8	46	3.8	2.4	B	11.4%	13,800	3.2%	0.047	0.043
ESP	DOE 6	Lead	7.3	1	1.9	3.5	L	17.0%	9,970	1.1%	0.020	0.020
ESP	Site 16L	Lead	7.3	2.9	11	5.1	B	9.5%	13,800	1.7%	0.14	0.12
FGDw	DOE 6	Lead	7.3	1	0.69	0.50	L	17.0%	9,970	1.1%	0.0094	0.0104
ESP	DOE 4	Lead	8	2.5	19	NC	B	13.8%	14,300	3.3%	0.14	0.14
FGDw	DOE 4	Lead	8	2.5	0.60	0.60	B	13.8%	14,300	3.3%	0.015	0.014
FGDd	DOE 7	Lead	8.2	2	0.70	0.5	S	22.9%	10,500	0.7%	0.017	0.012
FGDw	Site 125	Lead	8.2	1.5	15	14	B	9.8%	13,043	3.0%	0.12	0.09
ESP	DOE 3	Lead	11	3.5	38	5.4	B	12.0%	12,900	3.4%	0.15	0.11
FGDw	Site 20	Lead	12	9.9	3.8	2.9	L	21.0%	10,000	2.2%	0.017	0.019
ESP	Site 20	Lead	12	9.9	7.7	3.5	L	21.0%	10,000	2.2%	0.046	0.051

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 2	Lead	13	4.3	3.1	1.8	B	12.0%	12,900	2.8%	0.025	0.019
FF	DOE 2 FF	Lead	13	4.3	0.36	1.8	B	12.0%	13,000	2.6%	0.014	0.011
FF	Site 101	Lead	13	1.4	2.2	0.6	S	25.0%	10,000	0.8%	0.024	0.021
FGDw	Site 101	Lead	13	1.4	0.72	1.4	S	25.0%	10,000	0.8%	0.0083	0.0070
ESP	Site 114R	Lead	16	10	57	32	B	8.9%	13,300	1.8%	0.020	0.016
ESP	Site 110L	Lead	17	1.6	17	27	B	8.3%	12,100	2.9%	0.056	0.041
ESP	Site 122	Lead	25	10	190	170	B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 114B	Lead	37	40	86	83	B	8.7%	13,500	1.8%	0.040	0.025
FGDw	Site 21	Manganese	NM		15	16	B	6.7%	14,000	1.6%	0.013	0.011
FF	Site 115	Manganese	4.2	3.6	1.0	1.2	S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 115U	Manganese	5.2	3.3	0.89	2.8	S	10.5%	12,638	0.5%	0.0015	0.0012
ESP	Site 19	Manganese	7.3	8	5.4	120	B	9.1%	13,500	0.9%	0.039	0.036

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 22	Manganese	8.3	3.8	1.1	0.90	S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 16L	Manganese	14	0.8	21	4.9	B	9.5%	13,800	1.7%	0.14	0.12
ESP	Site 110L	Manganese	16	1.5	16	11	B	8.3%	12,100	2.9%	0.056	0.041
ESP	Site 16	Manganese	17	11	25	18	B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 11	Manganese	17	6	10	84	S	6.2%	11,900	0.6%	0.033	0.023
ESP	Site 110B	Manganese	17	3.5	37	72	B	9.1%	11,900	2.9%	0.030	0.022
FGDd	Site 14	Manganese	17	2.9	3.9	5.6	B	9.3%	13,700	2.9%	0.0076	0.0073
FGDw	Site 11	Manganese	17	6	2.6	7.0	S	6.2%	11,900	0.6%	0.0063	0.0051
ESP	Site 12	Manganese	18	4	60	45	B	9.4%	13,700	2.8%	0.12	0.10
FGDw	Site 12	Manganese	18	4	<1.6		B	9.4%	13,700	2.8%	0.014	0.013
ESP	Site 116	Manganese	19	2.4	10	40	B	12.0%	12,900	3.5%	0.056	0.046
FGDd	Site 116 SNRB	Manganese	19	2.4	0.80	6.0	B	12.0%	12,900	3.5%	0.031	0.029

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FF	Site 18 FF	Manganese	21	7.6	0.51	1.1	B	13.0%	13,400	1.1%	0.0017	0.0019
ESP	Site 18	Manganese	21	7.6	18	1.9	B	13.0%	13,400	1.1%	0.13	0.14
ESP	DOE 4	Manganese	23	3.3	34	3.5	B	13.8%	14,300	3.3%	0.14	0.14
FGDw	DOE 4	Manganese	23	3.3	7.2	48	B	13.8%	14,300	3.3%	0.015	0.014
FGDw	Site 125	Manganese	25	6	7.3	2.2	B	9.8%	13,043	3.0%	0.12	0.09
ESP	Site 114B	Manganese	27	7	20	25	B	8.7%	13,500	1.8%	0.040	0.025
FF	DOE 2 FF	Manganese	27	11	2.1	3.0	B	12.0%	13,000	2.6%	0.014	0.011
ESP	Site 114R	Manganese	27	13	15	17	B	8.9%	13,300	1.8%	0.020	0.016
ESP	Site 102	Manganese	27	6	17	6.2	S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 15	Manganese	28	5	8.6	5.4	B	12.7%	13,000	1.6%	0.037	0.028
FF	Site 10	Manganese	30	NA	31	NA	S	21.0%	11,000	0.5%	0.011	0.008
ESP	Site 122	Manganese	36	16	210	290	B	9.3%	13,370	2.0%	0.17	0.12

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 3	Manganese	47	2.2	27	2.4	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 5	Manganese	57	180	16	60	B	11.4%	13,800	3.2%	0.047	0.043
FGDw	Site 101	Manganese	66	24	10	70	S	25.0%	10,000	0.8%	0.0083	0.0070
FF	Site 101	Manganese	66	24	8.5	1.8	S	25.0%	10,000	0.8%	0.024	0.021
ESP	Site 20	Manganese	72	29	27	28	L	21.0%	10,000	2.2%	0.046	0.051
FGDw	Site 20	Manganese	72	29	33	78	L	21.0%	10,000	2.2%	0.017	0.019
FGDd	DOE 7	Manganese	83	25	11	11	S	22.9%	10,500	0.7%	0.017	0.012
ESP	DOE 6	Manganese	97	20	29	19	L	17.0%	9,970	1.1%	0.020	0.020
FGDw	DOE 6	Manganese	97	20	30	60	L	17.0%	9,970	1.1%	0.0094	0.0104
ESP	DOE 2	Manganese	100	1	3.9	3.0	B	12.0%	12,900	2.8%	0.025	0.019
FF	DOE 8	Manganese	136	28	18	26	S	11.2%	11,700	0.9%	0.011	0.009
FF	Site 115U	Mercury	0.019	0.1	0.41	0.59	S	10.5%	12,638	0.5%	0.0015	0.0012
FF	Site 115	Mercury	0.021	0.018	<0.35		S	11.2%	12,565	0.6%	0.0025	0.0019

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 5	Mercury	0.048	0.06	0.53	0.66	B	11.4%	13,800	3.2%	0.047	0.043
FGDd	DOE 7	Mercury	0.057	0.02	4.2	1.8	S	22.9%	10,500	0.7%	0.017	0.012
FGDw	Site 101	Mercury	0.063	0.014	1.9	1	S	25.0%	10,000	0.8%	0.0083	0.0070
FF	Site 101	Mercury	0.063	0.014	5.1	4	S	25.0%	10,000	0.8%	0.024	0.021
ESP	Site 110B	Mercury	0.064	0.007	6.1	1.2	B	9.1%	11,900	2.9%	0.030	0.022
FF	DOE 8	Mercury	0.07	0.025	1.9	1.8	S	11.2%	11,700	0.9%	0.011	0.009
ESP	DOE 3	Mercury	0.07	0.01	4.6	1.5	B	12.0%	12,900	3.4%	0.15	0.11
ESP	Site 102	Mercury	0.07	0.02	1.8	1.0	S	8.8%	12,200	1.0%	0.082	0.058
FGDw	DOE 4	Mercury	0.077	0.029	3.0	0.30	B	13.8%	14,300	3.3%	0.015	0.014
ESP	DOE 4	Mercury	0.077	0.029	5.6	1	B	13.8%	14,300	3.3%	0.14	0.14
ESP	Site 122	Mercury	0.079	0.04	6.8	21	B	9.3%	13,370	2.0%	0.17	0.12
FF	Site 10	Mercury	<0.08		<0.60		S	21.0%	11,000	0.5%	0.011	0.008
ESP	DOE 6	Mercury	0.08	0.025	5.8	3.8	L	17.0%	9,970	1.1%	0.020	0.020

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDw	DOE 6	Mercury	0.08	0.025	9.5	19	L	17.0%	9,970	1.1%	0.0094	0.0104
ESP	Site 110L	Mercury	0.08	0.06	5.0	2.3	B	8.3%	12,100	2.9%	0.056	0.041
FGDw	Site 125	Mercury	0.086	0.012	3.2	1	B	9.8%	13,043	3.0%	0.12	0.09
ESP	Site 19	Mercury	0.1	0.014	6.2	1.5	B	9.1%	13,500	0.9%	0.039	0.036
ESP	Site 114R	Mercury	<0.11		3.8	4.8	B	8.9%	13,300	1.8%	0.020	0.016
FGDw	Site 11	Mercury	0.11	0.02	8.3	12	S	6.2%	11,900	0.6%	0.0063	0.0051
FGDw	Site 12	Mercury	0.11	0.03	1.3	1.1	B	9.4%	13,700	2.8%	0.014	0.013
ESP	Site 11	Mercury	0.11	0.02	10	0.60	S	6.2%	11,900	0.6%	0.033	0.023
FF	Site 18 FF	Mercury	0.12	0.05	6.3	0.70	B	13.0%	13,400	1.1%	0.0017	0.0019
ESP	Site 18	Mercury	0.12	0.05	7.1	4.8	B	13.0%	13,400	1.1%	0.13	0.14
ESP	Site 114B	Mercury	<0.12		4.5	7.0	B	8.7%	13,500	1.8%	0.040	0.025
FGDd	Site 116 SNRB	Mercury	0.13	0.04	14	29	B	12.0%	12,900	3.5%	0.031	0.029

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 116	Mercury	0.13	0.04	9.2	2.5	B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 22	Mercury	0.14	0.0008	3.8	0.40	S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 16L	Mercury	0.14	0.07	4.8	2.0	B	9.5%	13,800	1.7%	0.14	0.12
ESP	Site 15	Mercury	0.14	0.04			B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 16	Mercury	0.15	0.08	6.4	1.1	B	10.0%	13,700	1.6%	0.25	0.21
FGDw	Site 21	Mercury	0.15	0.03	0.84	0.10	B	6.7%	14,000	1.6%	0.013	0.011
ESP	DOE 2	Mercury	0.21	0.13	14	5.3	B	12.0%	12,900	2.8%	0.025	0.019
ESP	Site 20	Mercury	0.26	0.04	15	7.2	L	21.0%	10,000	2.2%	0.046	0.051
FF	DOE 2 FF	Mercury	0.26	0.2	19	5.3	B	12.0%	13,000	2.6%	0.014	0.011
FGDw	Site 20	Mercury	0.26	0.04	12	2.6	L	21.0%	10,000	2.2%	0.017	0.019
FGDd	Site 111	Mercury	<0.48		<67		S	14.0%	10,020	0.6%	0.011	0.041
FF	Site 115	Nickel	0.6	0.5	1.5	6.1	S	11.2%	12,565	0.6%	0.0025	0.0019
FGDw	Site 101	Nickel	<1		2.9	8	S	25.0%	10,000	0.8%	0.0083	0.0070

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FF	Site 115U	Nickel	1	0.1	0.45	0.060	S	10.5%	12,638	0.5%	0.0015	0.0012
FF	Site 101	Nickel	<1		2.0	0.5	S	25.0%	10,000	0.8%	0.024	0.021
ESP	DOE 6	Nickel	2.6	3.2	4.3	7	L	17.0%	9,970	1.1%	0.020	0.020
FGDw	DOE 6	Nickel	2.6	3.2	5.1	35	L	17.0%	9,970	1.1%	0.0094	0.0104
FF	DOE 8	Nickel	2.8	0.2	2.0	2.7	S	11.2%	11,700	0.9%	0.011	0.009
ESP	Site 22	Nickel	3.9	2	0.64	1.1	S	6.8%	11,980	0.4%	0.0018	0.0015
FGDd	DOE 7	Nickel	5	0.25	<0.30		S	22.9%	10,500	0.7%	0.017	0.012
ESP	Site 110B	Nickel	6.7	1.6	7.7	11	B	9.1%	11,900	2.9%	0.030	0.022
ESP	Site 110L	Nickel	6.8	1.3	<5.0		B	8.3%	12,100	2.9%	0.056	0.041
FGDd	Site 116 SNRB	Nickel	8	2.5	49	500	B	12.0%	12,900	3.5%	0.031	0.029
ESP	Site 116	Nickel	8	2.5	<16		B	12.0%	12,900	3.5%	0.056	0.046
FGDw	Site 21	Nickel	8.3	0.86	1.7	0.40	B	6.7%	14,000	1.6%	0.013	0.011

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FF	Site 10	Nickel	<10		<2.0		S	21.0%	11,000	0.5%	0.011	0.008
ESP	Site 11	Nickel	<10		2.6	23	S	6.2%	11,900	0.6%	0.033	0.023
FGDw	Site 11	Nickel	<10		3.0	15	S	6.2%	11,900	0.6%	0.0063	0.0051
FF	Site 18 FF	Nickel	12	5	2.0	7.1	B	13.0%	13,400	1.1%	0.0017	0.0019
ESP	Site 19	Nickel	12	0.1	7.9	4.0	B	9.1%	13,500	0.9%	0.039	0.036
ESP	Site 18	Nickel	12	5	16	2.0	B	13.0%	13,400	1.1%	0.13	0.14
ESP	Site 122	Nickel	13	8.4	74	67	B	9.3%	13,370	2.0%	0.17	0.12
ESP	DOE 5	Nickel	14	4	4.0	11	B	11.4%	13,800	3.2%	0.047	0.043
ESP	Site 20	Nickel	14	4.8	5.5	6.0	L	21.0%	10,000	2.2%	0.046	0.051
FGDw	Site 20	Nickel	14	4.8	4.3	2.1	L	21.0%	10,000	2.2%	0.017	0.019
FGDd	Site 14	Nickel	<15		2.3	2.6	B	9.3%	13,700	2.9%	0.0076	0.0073
FF	DOE 2 FF	Nickel	15	3	0.22	1.7	B	12.0%	13,000	2.6%	0.014	0.011
FGDw	Site 125	Nickel	17	3	7.5	1.4	B	9.8%	13,043	3.0%	0.12	0.09

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 16L	Nickel	17	12	17	4.5	B	9.5%	13,800	1.7%	0.14	0.12
ESP	DOE 2	Nickel	18	8	1.2	1.7	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 3	Nickel	18	2.3	27	2.8	B	12.0%	12,900	3.4%	0.15	0.11
ESP	Site 15	Nickel	<20		5.9	3.3	B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 16	Nickel	27	3.1	24	19	B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 114B	Nickel	28	50	78	64	B	8.7%	13,500	1.8%	0.040	0.025
ESP	DOE 4	Nickel	30	6.4	24	5.9	B	13.8%	14,300	3.3%	0.14	0.14
FGDw	DOE 4	Nickel	30	6.4	40	440	B	13.8%	14,300	3.3%	0.015	0.014
ESP	Site 102	Nickel	31	12	340	210	S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 12	Nickel	<32		2.3	1.6	B	9.4%	13,700	2.8%	0.12	0.10

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDw	Site 12	Nickel	<32		4.0	60	B	9.4%	13,700	2.8%	0.014	0.013
ESP	Site 114R	Nickel	46	20	34	52	B	8.9%	13,300	1.8%	0.020	0.016
FGDd	Site 111	Nickel	<120		<5.3		S	14.0%	10,020	0.6%	0.011	0.041
ESP	Site 110B	Selenium	0.57	0.03	46	17	B	9.1%	11,900	2.9%	0.030	0.022
ESP	DOE 2	Selenium	<0.6		62	65	B	12.0%	12,900	2.8%	0.025	0.019
ESP	Site 102	Selenium	<0.6		<0.14		S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 11	Selenium	0.67	0.21	3.8	31	S	6.2%	11,900	0.6%	0.033	0.023
FGDw	Site 11	Selenium	0.67	0.21	0.65	0.65	S	6.2%	11,900	0.6%	0.0063	0.0051
ESP	Site 22	Selenium	0.78	0.30	0.053	0.036	S	6.8%	11,980	0.4%	0.0018	0.0015
FF	Site 115	Selenium	0.82	0.25	0.36	0.46	S	11.2%	12,565	0.6%	0.0025	0.0019
FGDd	DOE 7	Selenium	0.86	1.6	<0.038		S	22.9%	10,500	0.7%	0.017	0.012
FF	DOE 8	Selenium	0.88	0.2	3.2	0.3	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 2 FF	Selenium	0.9	1.7	58	65	B	12.0%	13,000	2.6%	0.014	0.011
ESP	Site 114R	Selenium	<1.1		150	340	B	8.9%	13,300	1.8%	0.020	0.016
FGDw	Site 12	Selenium	1.2	0.08	13	1.2	B	9.4%	13,700	2.8%	0.014	0.013
FGDd	Site 14	Selenium	1.2	0.5	0.55	1.4	B	9.3%	13,700	2.9%	0.0076	0.0073
FGDw	Site 21	Selenium	1.2	0.4	9.9	4.1	B	6.7%	14,000	1.6%	0.013	0.011

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/MBtu
ESP	Site 114B	Selenium	<1.2		240	350	B	8.7%	13,500	1.8%	0.040	0.025
ESP	Site 12	Selenium	1.2	0.08	48	40	B	9.4%	13,700	2.8%	0.12	0.10
ESP	DOE 6	Selenium	<1.3		8.7	1.5	L	17.0%	9,970	1.1%	0.020	0.020
FGDw	DOE 6	Selenium	<1.3		8.3	19	L	17.0%	9,970	1.1%	0.0094	0.0104
FF	Site 10	Selenium	1.4	NA	<16		S	21.0%	11,000	0.5%	0.011	0.008
FF	Site 115U	Selenium	1.5	1.9	<0.060		S	10.5%	12,638	0.5%	0.0015	0.0012
ESP	Site 122	Selenium	1.6	1	70	100	B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 110L	Selenium	2.1	0.5	130	58	B	8.3%	12,100	2.9%	0.056	0.041
ESP	Site 15	Selenium	2.2	0.6	77	38	B	12.7%	13,000	1.6%	0.037	0.028
ESP	DOE 4	Selenium	2.3	1.4	80	120	B	13.8%	14,300	3.3%	0.14	0.14
FGDw	DOE 4	Selenium	2.3	1.4	27	58	B	13.8%	14,300	3.3%	0.015	0.014
ESP	Site 116	Selenium	2.7	1.4	31	96	B	12.0%	12,900	3.5%	0.056	0.046
FGDd	Site 116 SNRB	Selenium	2.7	1.4	<0.30		B	12.0%	12,900	3.5%	0.031	0.029
FGDw	Site 101	Selenium	2.7	0.6	1.4	4	S	25.0%	10,000	0.8%	0.0083	0.0070
FF	Site 101	Selenium	2.7	0.6	2.9	1.4	S	25.0%	10,000	0.8%	0.024	0.021
ESP	Site 18	Selenium	3.1	0.63	170	280	B	13.0%	13,400	1.1%	0.13	0.14

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FF	Site 18 FF	Selenium	3.1	0.63	72	43	B	13.0%	13,400	1.1%	0.0017	0.0019
ESP	Site 16L	Selenium	3.7	0.8	140	120	B	9.5%	13,800	1.7%	0.14	0.12
ESP	DOE 3	Selenium	3.7	0.38	130	100	B	12.0%	12,900	3.4%	0.15	0.11
ESP	Site 16	Selenium	3.8	1.2	130	33	B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 19	Selenium	3.9	0.46	260	350	B	9.1%	13,500	0.9%	0.039	0.036
FGDw	Site 125	Selenium	4	1.2	320	140	B	9.8%	13,043	3.0%	0.12	0.09
ESP	Site 20	Selenium	4.5	2.1	780	220	L	21.0%	10,000	2.2%	0.046	0.051
FGDw	Site 20	Selenium	4.5	2.1	160	65	L	21.0%	10,000	2.2%	0.017	0.019
ESP	DOE 5	Selenium	23	46	86	80	B	11.4%	13,800	3.2%	0.047	0.043
ESP	Site 16L	Benzene			<0.51		B	9.5%	13,800	1.7%	0.14	0.12
FGDw	Site 101	Benzene			<0.60		S	25.0%	10,000	0.8%	0.0083	0.0070
FGDw	Site 12	Benzene			0.70	0.40	B	9.4%	13,700	2.8%	0.014	0.013
ESP	Site 15	Benzene			0.80	3.0	B	12.7%	13,000	1.6%	0.037	0.028
FGDd	DOE 7	Benzene			1.0	0.4	S	22.9%	10,500	0.7%	0.017	0.012
ESP	Site 114R	Benzene			1.1	0.70	B	8.9%	13,300	1.8%	0.020	0.016
FGDw	Site 11	Benzene			1.2	1.0	S	6.2%	11,900	0.6%	0.0063	0.0051
FGDw	DOE 4	Benzene			1.3	0.30	B	13.8%	14,300	3.3%	0.015	0.014

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 4	Benzene			1.4	0.40	B	13.8%	14,300	3.3%	0.14	0.14
ESP	Site 16	Benzene			1.4	3.0	B	10.0%	13,700	1.6%	0.25	0.21
FF	Site 10	Benzene			2.0	2.0	S	21.0%	11,000	0.5%	0.011	0.008
ESP	Site 114B	Benzene			2.3	2.6	B	8.7%	13,500	1.8%	0.040	0.025
FF	Site 115	Benzene			2.6	2.2	S	11.2%	12,565	0.6%	0.0025	0.0019
ESP	DOE 5	Benzene			3.4	6	B	11.4%	13,800	3.2%	0.047	0.043
FGDd	Site 116 SNRB	Benzene			7.0	7.7	B	12.0%	12,900	3.5%	0.031	0.029
ESP	DOE 2	Benzene			7.9	5.5	B	12.0%	12,900	2.8%	0.025	0.019
ESP	Site 122	Benzene			8.1	6	B	9.3%	13,370	2.0%	0.17	0.12
FF	DOE 2 FF	Benzene			15	24	B	12.0%	13,000	2.6%	0.014	0.011
ESP	Site 116	Benzene			26	9	B	12.0%	12,900	3.5%	0.056	0.046
FGDw	DOE 6	Benzene			41	95	L	17.0%	9,970	1.1%	0.0094	0.0104
ESP	DOE 6	Benzene			94	110	L	17.0%	9,970	1.1%	0.020	0.020
FF	DOE 8	Benzene			100	180	S	11.2%	11,700	0.9%	0.011	0.009
ESP	DOE 3	Benzene			120	330	B	12.0%	12,900	3.4%	0.15	0.11
FGDw	Site 11	Toluene			<0.40		S	6.2%	11,900	0.6%	0.0063	0.0051

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 16	Toluene			<0.49		B	10.0%	13,700	1.6%	0.25	0.21
FGDd	DOE 7	Toluene			0.50	0.43	S	22.9%	10,500	0.7%	0.017	0.012
FGDw	Site 101	Toluene			<0.60		S	25.0%	10,000	0.8%	0.0083	0.0070
ESP	Site 114R	Toluene			0.70	0.17	B	8.9%	13,300	1.8%	0.020	0.016
ESP	Site 16L	Toluene			0.70	0.21	B	9.5%	13,800	1.7%	0.14	0.12
ESP	Site 114B	Toluene			1.0	0.30	B	8.7%	13,500	1.8%	0.040	0.025
ESP	Site 116	Toluene			<1.0		B	12.0%	12,900	3.5%	0.056	0.046
FGDw	Site 12	Toluene			1.0	0.40	B	9.4%	13,700	2.8%	0.014	0.013
ESP	DOE 4	Toluene			1.2	1.0	B	13.8%	14,300	3.3%	0.14	0.14
FGDd	Site 116 SNRB	Toluene			<1.9		B	12.0%	12,900	3.5%	0.031	0.029
ESP	Site 122	Toluene			2.0	1.7	B	9.3%	13,370	2.0%	0.17	0.12
FGDw	DOE 4	Toluene			2.0	1.0	B	13.8%	14,300	3.3%	0.015	0.014
ESP	DOE 3	Toluene			2.0	1.7	B	12.0%	12,900	3.4%	0.15	0.11
FF	DOE 2 FF	Toluene			<4.0		B	12.0%	13,000	2.6%	0.014	0.011
ESP	DOE 2	Toluene			<4.6		B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 5	Toluene			5.2	9	B	11.4%	13,800	3.2%	0.047	0.043

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FF	DOE 8	Toluene			5.5	1.8	S	11.2%	11,700	0.9%	0.011	0.009
ESP	DOE 6	Toluene			<6.3		L	17.0%	9,970	1.1%	0.020	0.020
FGDw	DOE 6	Toluene			24	53	L	17.0%	9,970	1.1%	0.0094	0.0104
FF	Site 115	Toluene			110	84	S	11.2%	12,565	0.6%	0.0025	0.0019
ESP	DOE 4	Formaldehyde			0.40	1.0	B	13.8%	14,300	3.3%	0.14	0.14
ESP	Site 122	Formaldehyde			<0.74		B	9.3%	13,370	2.0%	0.17	0.12
FGDd	Site 116 SNRB	Formaldehyde			<1.0		B	12.0%	12,900	3.5%	0.031	0.029
ESP	Site 116	Formaldehyde			<1.0		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 16L	Formaldehyde			1.3	2.4	B	9.5%	13,800	1.7%	0.14	0.12
FF	DOE 2 FF	Formaldehyde			<1.6		B	12.0%	13,000	2.6%	0.014	0.011
ESP	DOE 3	Formaldehyde			<1.7		B	12.0%	12,900	3.4%	0.15	0.11
FF	DOE 8	Formaldehyde			<2		S	11.2%	11,700	0.9%	0.011	0.009
ESP	DOE 6	Formaldehyde			<1.9		L	17.0%	9,970	1.1%	0.020	0.020
FGDw	DOE 6	Formaldehyde			<2.2		L	17.0%	9,970	1.1%	0.0094	0.0104
ESP	Site 114R	Formaldehyde			<2.6		B	8.9%	13,300	1.8%	0.020	0.016
ESP	Site 114B	Formaldehyde			<2.6		B	8.7%	13,500	1.8%	0.040	0.025

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 2	Formaldehyde			4.0	9.0	B	12.0%	12,900	2.8%	0.025	0.019
ESP	Site 16	Formaldehyde			<4.0		B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 15	Formaldehyde			<5.0		B	12.7%	13,000	1.6%	0.037	0.028
FGDd	DOE 7	Formaldehyde			6.0	13	S	22.9%	10,500	0.7%	0.017	0.012
FGDw	Site 12	Formaldehyde			<9.0		B	9.4%	13,700	2.8%	0.014	0.013
FGDw	Site 11	Formaldehyde			<9.0		S	6.2%	11,900	0.6%	0.0063	0.0051
FF	Site 10	Formaldehyde			<13		S	21.0%	11,000	0.5%	0.011	0.008
FF	Site 115	Formaldehyde			17	20	S	11.2%	12,565	0.6%	0.0025	0.0019
FGDw	DOE 4	Formaldehyde			24	33	B	13.8%	14,300	3.3%	0.015	0.014
ESP	DOE 5	Formaldehyde			60	230	B	11.4%	13,800	3.2%	0.047	0.043
ESP	Site 12	Formaldehyde			90	300	B	9.4%	13,700	2.8%	0.12	0.10
ESP	DOE 2	Benzo(a)anthracene			0.0037	0.0098	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	Benzo(a)pyrene			<0.0024		B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	Benzo(b,j&k)fluoranthene			0.0071	0.025	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	Benzo(g,h,i)perylene			<0.0030		B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	Chrysene			0.0090	0.021	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	Dibenzo(a,h)anthracene			<0.0030		B	12.0%	12,900	2.8%	0.025	0.019

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 2	Indeno(1,2,3-cd)pyrene			<0.0030		B	12.0%	12,900	2.8%	0.025	0.019
FF	DOE 2 FF	Benzo(a)anthracene			0.0045	0.0080	B	12.0%	13,000	2.6%	0.014	0.011
FF	DOE 2 FF	Benzo(a)pyrene			0.0014	0.0042	B	12.0%	13,000	2.6%	0.014	0.011
FF	DOE 2 FF	Benzo(b,j&k)fluoranthene			0.0035	0.0064	B	12.0%	13,000	2.6%	0.014	0.011
FF	DOE 2 FF	Benzo(g,h,i)perylene			<0.0017		B	12.0%	13,000	2.6%	0.014	0.011
FF	DOE 2 FF	Chrysene			0.0059	0.0080	B	12.0%	13,000	2.6%	0.014	0.011
FF	DOE 2 FF	Dibenzo(a,h)anthracene			<0.0017		B	12.0%	13,000	2.6%	0.014	0.011
FF	DOE 2 FF	Indeno(1,2,3-cd)pyrene			0.0022	0.0020	B	12.0%	13,000	2.6%	0.014	0.011
ESP	DOE 3	Benzo(a)anthracene			<0.0012		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	Benzo(a)pyrene			<0.0005		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	Benzo(b)fluoranthene			<0.0026		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	Benzo(k)fluoranthene			<0.0013		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	Benzo(g,h,i)perylene			<0.0011		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	Chrysene			<0.0021		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	Dibenzo(a,h)anthracene			<0.0003		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	Indeno(1,2,3-cd)pyrene			<0.0011		B	12.0%	12,900	3.4%	0.15	0.11
FGDw	DOE 4	Benzo(a)anthracene			<0.16		B	13.8%	14,300	3.3%	0.015	0.014

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDw	DOE 4	Benzo(a)pyrene			<0.14		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	Benzo(b,j&k)fluoranthene			<0.20		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	Benzo(g,h,i)perylene			<0.20		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	Chrysene			<0.20		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	Dibenzo(a,h)anthracene			<0.19		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	Indeno(1,2,3-cd)pyrene			<0.26		B	13.8%	14,300	3.3%	0.015	0.014
ESP	DOE 6	Benzo(a)anthracene			0.098	0.07	L	17.0%	9,970	1.1%	0.020	0.020
ESP	DOE 6	Benzo(a)pyrene			0.0042	0.0003	L	17.0%	9,970	1.1%	0.020	0.020
ESP	DOE 6	Benzo(b,j&k)fluoranthene			0.15	0.09	L	17.0%	9,970	1.1%	0.020	0.020
ESP	DOE 6	Benzo(g,h,i)perylene			<0.0013		L	17.0%	9,970	1.1%	0.020	0.020
ESP	DOE 6	Chrysene			2.3	1.4	L	17.0%	9,970	1.1%	0.020	0.020
ESP	DOE 6	Dibenzo(a,h)anthracene			<0.0013		L	17.0%	9,970	1.1%	0.020	0.020
ESP	DOE 6	Indeno(1,2,3-cd)pyrene			<0.0013		L	17.0%	9,970	1.1%	0.020	0.020
FGDw	DOE 6	Benzo(a)anthracene			0.0021	0.0018	L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	Benzo(a)pyrene			0.00078	0.0015	L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	Benzo(b,j&k)fluoranthene			0.0046	0.0052	L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	Benzo(g,h,i)perylene			0.00049	0.0093	L	17.0%	9,970	1.1%	0.0094	0.0104

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDw	DOE 6	Chrysene			0.0053	0.0049	L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	Dibenzo(a,h)anthracene			0.00064	0.0054	L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	Indeno(1,2,3-cd)pyrene			0.00054	0.0010	L	17.0%	9,970	1.1%	0.0094	0.0104
FF	DOE 8	Benzo(a)anthracene			0.0047	0.0139	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Benzo(a)pyrene			<0.00021		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Benzo(b)fluoranthene			0.0027	0.0012	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Benzo(k)fluoranthene			0.00033	0.0072	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Benzo(g,h,i)perylene			<0.00052		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Chrysene			0.012	0.031	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Dibenzo(a,h)anthracene			<0.00012		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Indeno(1,2,3-cd)pyrene			<0.00035		S	11.2%	11,700	0.9%	0.011	0.009
FF	Site 10	Benzo(a)anthracene			<2.0		S	21.0%	11,000	0.5%	0.011	0.008
FF	Site 10	Benzo(a)pyrene			<2.0		S	21.0%	11,000	0.5%	0.011	0.008
FF	Site 10	Benzo(b,j&k)fluoranthene			<2.0		S	21.0%	11,000	0.5%	0.011	0.008
FF	Site 10	Benzo(g,h,i)perylene			<2.0		S	21.0%	11,000	0.5%	0.011	0.008
FF	Site 10	Chrysene			<2.0		S	21.0%	11,000	0.5%	0.011	0.008
FF	Site 10	Dibenzo(a,h)anthracene			<2.0		S	21.0%	11,000	0.5%	0.011	0.008

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FF	Site 10	Indeno(1,2,3-cd)pyrene			<2.0		S	21.0%	11,000	0.5%	0.011	0.008
ESP	Site 11	Benzo(a)anthracene			<2.0		S	6.2%	11,900	0.6%	0.033	0.023
ESP	Site 11	Benzo(a)pyrene			<2.0		S	6.2%	11,900	0.6%	0.033	0.023
ESP	Site 11	Benzo(b,j&k)fluoranthene			<2.0		S	6.2%	11,900	0.6%	0.033	0.023
ESP	Site 11	Benzo(g,h,i)perylene			<2.0		S	6.2%	11,900	0.6%	0.033	0.023
ESP	Site 11	Chrysene			<2.0		S	6.2%	11,900	0.6%	0.033	0.023
ESP	Site 11	Dibenzo(a,h)anthracene			<2.0		S	6.2%	11,900	0.6%	0.033	0.023
ESP	Site 11	Indeno(1,2,3-cd)pyrene			<2.0		S	6.2%	11,900	0.6%	0.033	0.023
FGDw	Site 11	Benzo(a)anthracene			<2.0		S	6.2%	11,900	0.6%	0.0063	0.0051
FGDw	Site 11	Benzo(a)pyrene			<2.0		S	6.2%	11,900	0.6%	0.0063	0.0051
FGDw	Site 11	Benzo(b,j&k)fluoranthene			<2.0		S	6.2%	11,900	0.6%	0.0063	0.0051
FGDw	Site 11	Benzo(g,h,i)perylene			<2.0		S	6.2%	11,900	0.6%	0.0063	0.0051
FGDw	Site 11	Chrysene			<2.0		S	6.2%	11,900	0.6%	0.0063	0.0051
FGDw	Site 11	Dibenzo(a,h)anthracene			<2.0		S	6.2%	11,900	0.6%	0.0063	0.0051
FGDw	Site 11	Indeno(1,2,3-cd)pyrene			<2.0		S	6.2%	11,900	0.6%	0.0063	0.0051
FGDd	Site 111	Benzo(a)anthracene			0.0090	0.090	S	14.0%	10,020	0.6%	0.011	0.041
FGDd	Site 111	Benzo(a)pyrene			<0.0040		S	14.0%	10,020	0.6%	0.011	0.041

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDd	Site 111	Benzo(b,j&k)fluoranthene			0.0080	0.040	S	14.0%	10,020	0.6%	0.011	0.041
FGDd	Site 111	Benzo(g,h,i)perylene			0.0040	0.030	S	14.0%	10,020	0.6%	0.011	0.041
FGDd	Site 111	Chrysene			<0.0040		S	14.0%	10,020	0.6%	0.011	0.041
FGDd	Site 111	Dibenzo(a,h)anthracene			<0.0040		S	14.0%	10,020	0.6%	0.011	0.041
FGDd	Site 111	Indeno(1,2,3-cd)pyrene			0.0040	0.030	S	14.0%	10,020	0.6%	0.011	0.041
FF	Site 115	Benzo(a)anthracene			<0.014		S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 115	Benzo(a)pyrene			<0.021		S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 115	Benzo(b)fluoranthene			<0.011		S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 115	Benzo(k)fluoranthene			<0.014		S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 115	Benzo(g,h,i)perylene			<0.030		S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 115	Chrysene			<0.014		S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 115	Dibenzo(a,h)anthracene			<0.044		S	11.2%	12,565	0.6%	0.0025	0.0019
FF	Site 115	Indeno(1,2,3-cd)pyrene			<0.037		S	11.2%	12,565	0.6%	0.0025	0.0019
ESP	Site 116	Benzo(a)anthracene			0.013	0.023	B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	Benzo(a)pyrene			0.020	0.038	B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	Benzo(b)fluoranthene			0.019	0.026	B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	Benzo(k)fluoranthene			0.019	0.026	B	12.0%	12,900	3.5%	0.056	0.046

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 116	Benzo(g,h,i)perylene			0.0090	0.0080	B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	Chrysene			0.040	0.13	B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	Dibenzo(a,h)anthracene			0.0060	0.0050	B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	Indeno(1,2,3-cd)pyrene			0.0090	0.014	B	12.0%	12,900	3.5%	0.056	0.046
FGDd	Site 116 SNRB	Benzo(a)anthracene			0.0084	0.0071	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	Benzo(a)pyrene			0.013	0.0078	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	Benzo(b)fluoranthene			0.022	0.015	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	Benzo(k)fluoranthene			0.022	0.015	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	Benzo(g,h,i)perylene			0.013	0.016	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	Chrysene			0.015	0.0047	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	Dibenzo(a,h)anthracene			0.0090	0.019	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	Indeno(1,2,3-cd)pyrene			0.011	0.013	B	12.0%	12,900	3.5%	0.031	0.029

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 12	Benzo(a)anthracene			<2.0		B	9.4%	13,700	2.8%	0.12	0.10
ESP	Site 12	Benzo(a)pyrene			<2.0		B	9.4%	13,700	2.8%	0.12	0.10
ESP	Site 12	Benzo(b,j&k)fluoranthene			<2.0		B	9.4%	13,700	2.8%	0.12	0.10
ESP	Site 12	Benzo(g,h,i)perylene			<2.0		B	9.4%	13,700	2.8%	0.12	0.10
ESP	Site 12	Chrysene			<2.0		B	9.4%	13,700	2.8%	0.12	0.10
ESP	Site 12	Dibenzo(a,h)anthracene			<2.0		B	9.4%	13,700	2.8%	0.12	0.10
ESP	Site 12	Indeno(1,2,3-cd)pyrene			<2.0		B	9.4%	13,700	2.8%	0.12	0.10
FGDw	Site 12	Benzo(a)anthracene			<2.0		B	9.4%	13,700	2.8%	0.014	0.013
FGDw	Site 12	Benzo(a)pyrene			<2.0		B	9.4%	13,700	2.8%	0.014	0.013
FGDw	Site 12	Benzo(b,j&k)fluoranthene			<2.0		B	9.4%	13,700	2.8%	0.014	0.013
FGDw	Site 12	Benzo(g,h,i)perylene			<2.0		B	9.4%	13,700	2.8%	0.014	0.013
FGDw	Site 12	Chrysene			<2.0		B	9.4%	13,700	2.8%	0.014	0.013
FGDw	Site 12	Dibenzo(a,h)anthracene			<2.0		B	9.4%	13,700	2.8%	0.014	0.013
FGDw	Site 12	Indeno(1,2,3-cd)pyrene			<2.0		B	9.4%	13,700	2.8%	0.014	0.013
FGDd	Site 14	Benzo(a)anthracene			<2.0		B	9.3%	13,700	2.9%	0.0076	0.0073
FGDd	Site 14	Benzo(a)pyrene			<2.0		B	9.3%	13,700	2.9%	0.0076	0.0073
FGDd	Site 14	Benzo(b,j&k)fluoranthene			<2.0		B	9.3%	13,700	2.9%	0.0076	0.0073

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDd	Site 14	Benzo(g,h,i)perylene			<2.0		B	9.3%	13,700	2.9%	0.0076	0.0073
FGDd	Site 14	Chrysene			<2.0		B	9.3%	13,700	2.9%	0.0076	0.0073
FGDd	Site 14	Dibenzo(a,h)anthracene			<2.0		B	9.3%	13,700	2.9%	0.0076	0.0073
FGDd	Site 14	Indeno(1,2,3-cd)pyrene			<2.0		B	9.3%	13,700	2.9%	0.0076	0.0073
ESP	Site 15	Benzo(a)anthracene			<8.0		B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 15	Benzo(a)pyrene			<8.0		B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 15	Benzo(b,j&k)fluoranthene			<8.0		B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 15	Benzo(g,h,i)perylene			<8.0		B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 15	Chrysene			<8.0		B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 15	Dibenzo(a,h)anthracene			<8.0		B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 15	Indeno(1,2,3-cd)pyrene			<8.0		B	12.7%	13,000	1.6%	0.037	0.028
ESP	Site 16	Benzo(a)anthracene			<5.0		B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 16	Benzo(a)pyrene			<5.0		B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 16	Benzo(g,h,i)perylene			<5.0		B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 16	Chrysene			<5.0		B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 16	Dibenzo(a,h)anthracene			<5.0		B	10.0%	13,700	1.6%	0.25	0.21
ESP	Site 16	Indeno(1,2,3-cd)pyrene			<5.0		B	10.0%	13,700	1.6%	0.25	0.21

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 16L	Benzo(a)anthracene			0.0070	0.027	B	9.5%	13,800	1.7%	0.14	0.12
ESP	Site 16L	Benzo(a)pyrene			<0.0041		B	9.5%	13,800	1.7%	0.14	0.12
ESP	Site 16L	Benzo(b,j&k)fluoranthene			0.0015	0.0038	B	9.5%	13,800	1.7%	0.14	0.12
ESP	Site 16L	Benzo(g,h,i)perylene			<0.0031		B	9.5%	13,800	1.7%	0.14	0.12
ESP	Site 16L	Chrysene			0.0018	0.0030	B	9.5%	13,800	1.7%	0.14	0.12
ESP	Site 16L	Dibenzo(a,h)anthracene			<0.0037		B	9.5%	13,800	1.7%	0.14	0.12
ESP	Site 16L	Indeno(1,2,3-cd)pyrene			<0.0027		B	9.5%	13,800	1.7%	0.14	0.12
FGDw	Site 21	Benzo(a)anthracene			0.0013	0.0012	B	6.7%	14,000	1.6%	0.013	0.011
FGDw	Site 21	Benzo(a)pyrene			0.0018	0.0020	B	6.7%	14,000	1.6%	0.013	0.011
FGDw	Site 21	Benzo(b,j&k)fluoranthene			0.0070	0.0080	B	6.7%	14,000	1.6%	0.013	0.011
FGDw	Site 21	Benzo(g,h,i)perylene			0.0012	0.0012	B	6.7%	14,000	1.6%	0.013	0.011
FGDw	Site 21	Chrysene			0.0070	0.0080	B	6.7%	14,000	1.6%	0.013	0.011
FGDw	Site 21	Dibenzo(a,h)anthracene			<0.0030		B	6.7%	14,000	1.6%	0.013	0.011
FGDw	Site 21	Indeno(1,2,3-cd)pyrene			0.0015	0.0015	B	6.7%	14,000	1.6%	0.013	0.011
ESP	Site 22	Benzo(a)anthracene			0.0010	0.0020	S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	Benzo(a)pyrene			0.0011	0.0021	S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	Benzo(b)fluoranthene			0.0027	0.0042	S	6.8%	11,980	0.4%	0.0018	0.0015

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 22	Benzo(g,h,i)perylene			0.0022	0.0013	S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	Chrysene			0.0025	0.0042	S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	Dibenzo(a,h)anthracene			<0.00030		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	Indeno(1,2,3-cd)pyrene			0.0086	0.0050	S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	DOE 2	2,3,7,8-Tetrachlorodibenzo-p-dioxin			<2.6x10^{-06}		B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			<3.5x10^{-06}		B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			<4.1x10^{-06}		B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			3.0x10^{-06}	8x10^{-06}	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			2.9x10^{-06}	9x10^{-06}	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	1,2,3,4,6,7,8-Heptachlorodibenzo- p-dioxin			1.7x10^{-05}	5x10^{-05}	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	Octachlorodibenzo-p-dioxin			1.9x10^{-05}	8x10^{-05}	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	TotalTetrachlorodibenzo-p-dioxin			7.3x10^{-06}	2x10^{-05}	B	12.0%	12,900	2.8%	0.025	0.019

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 2	TotalPentachlorodibenzo-p-dioxin			2.6x10^{-06}	7x10^{-06}	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	TotalHexachlorodibenzo-p-dioxin			1.1e-05	4e-05	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	TotalHeptachlorodibenzo - p-dioxin			2.7x10^{-05}	7x10^{-05}	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	2,3,7,8-Tetrachlorodibenzofuran			4.8x10^{-06}	1x10^{-05}	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	1,2,3,7,8-Pentachlorodibenzofuran			<4.1x10^{-06}		B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	2,3,4,7,8-Pentachlorodibenzofuran			3.2x10^{-06}	6x10^{-06}	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	1,2,3,4,7,8-Hexachlorodibenzofuran			9.7x10^{-06}	3x10^{-05}	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	1,2,3,6,7,8-Hexachlorodibenzofuran			4.0x10^{-06}	1x10^{-05}	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	1,2,3,7,8,9-Hexachlorodibenzofuran			6.6x10^{-06}	1x10^{-05}	B	12.0%	12,900	2.8%	0.025	0.019

Shaded data not included in statistical calculations.

Table B-16.

Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 2	1,2,3,4,6,7,8-Heptachlorodibenzofuran			$1.7x10^{-05}$	$5x10^{-05}$	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	1,2,3,4,7,8,9-Heptachlorodibenzofuran			$3.7x10^{-06}$	$9x10^{-06}$	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	Octachlorodibenzofuran			$2.0x10^{-05}$	$3x10^{-05}$	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	Total Tetrachlorodibenzofuran			$1.7x10^{-05}$	$7x10^{-05}$	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	Total Pentachlorodibenzofuran			$2.0x10^{-05}$	$7x10^{-05}$	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	Total Hexachlorodibenzofuran			$2.3x10^{-05}$	$9x10^{-05}$	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 2	Total Heptachlorodibenzofuran			$2.0x10^{-05}$	$7x10^{-05}$	B	12.0%	12,900	2.8%	0.025	0.019
ESP	DOE 3	2,3,7,8-Tetrachlorodibenzo- p-dioxin			$<2.5x10^{-06}$		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	1,2,3,7,8-Pentachlorodibenzo- p-dioxin			$<4.2x10^{-07}$		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			$<4.2x10^{-07}$		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			$<3.2x10^{-07}$		B	12.0%	12,900	3.4%	0.15	0.11

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 3	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			<4.2x10^{-07}		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	1,2,3,4,6,7,8-Heptachlorodibenzo- p-dioxin			<1.3x10^{-06}		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	Octachlorodibenzo-p-dioxin			<8.9x10^{-06}		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	TotalTetrachlorodibenzo-p-dioxin			1.3x10^{-06}	2.2x10^{-07}	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	TotalPentachlorodibenzo-p-dioxin			<7.4x10^{-07}		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	TotalPentachlorodibenzo-p-dioxin			1.1x10^{-05}	7.2x10^{-06}	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	TotalHexachlorodibenzo-p-dioxin			9.6x10^{-07}	1.4x10^{-06}	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	TotalHeptachlorodibenzo-p-dioxin			2.5x10^{-06}	2.9x10^{-06}	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	2,3,7,8-Tetrachlorodibenzofuran			<1.3x10^{-06}		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	1,2,3,7,8-Pentachlorodibenzofuran			<7.4x10^{-07}		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	2,3,4,7,8-Pentachlorodibenzofuran			1.8x10^{-06}	1.4x10^{-06}	B	12.0%	12,900	3.4%	0.15	0.11

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 3	1,2,3,4,7,8-Hexachlorodibenzofuran			2.4x10^{-06}	1.4x10^{-06}	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	1,2,3,6,7,8-Hexachlorodibenzofuran			8.0x10^{-07}	3.6x10^{-07}	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	1,2,3,7,8,9-Hexachlorodibenzofuran			<3.2x10^{-07}		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	2,3,4,6,7,8-Hexachlorodibenzofuran			<1.5x10^{-06}		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	1,2,3,4,6,7,8-Heptachlorodibenzofuran			1.5x10^{-06}	2.2x10^{-07}	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	1,2,3,4,7,8,9-Heptachlorodibenzofuran			<4.6x10^{-07}		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	Octachlorodibenzofuran			4.2x10^{-06}	5.8x10^{-06}	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	TotalTetrachlorodibenzofuran			<3.8x10^{-06}		B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	TotalPentachlorodibenzofuran			1.8x10^{-05}	2.2x10^{-06}	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	TotalPentachlorodibenzofuran			4.0x10^{-06}	5.8x10^{-06}	B	12.0%	12,900	3.4%	0.15	0.11
ESP	DOE 3	Total Hexachlorodibenzofuran			5.6x10^{-06}	7.2x10^{-06}	B	12.0%	12,900	3.4%	0.15	0.11

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 3	Total Heptachlorodibenzofuran			$3.2x10^{-06}$	$2.2x10^{-06}$	B	12.0%	12,900	3.4%	0.15	0.11
FGDw	DOE 4	2,3,7,8-Tetrachlorodibenzo- p-dioxin			$<3.3x10^{-06}$		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	1,2,3,7,8-Pentachlorodibenzo- p-dioxin			$<4.7x10^{-06}$		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			$<1.5x10^{-05}$		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			$<9.9x10^{-06}$		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			$<1.2x10^{-05}$		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	1,2,3,4,6,7,8-Heptachlorodibenzo- p-dioxin			$<2.6x10^{-05}$		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	Octachlorodibenzo-p-dioxin			<0.00013		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	TotalTetrachlorodibenzo-p-dioxin			$7.0x10^{-06}$	$8x10^{-06}$	B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	2,3,7,8-Tetrachlorodibenzofuran			$<3.3x10^{-06}$		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	1,2,3,7,8-Pentachlorodibenzofuran			$<3.2x10^{-06}$		B	13.8%	14,300	3.3%	0.015	0.014

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDw	DOE 4	2,3,4,7,8-Pentachlorodibenzofuran			<3.2x10^{-06}		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	1,2,3,4,7,8-Hexachlorodibenzofuran			<1.6x10^{-05}		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	1,2,3,6,7,8-Hexachlorodibenzofuran			<5.8x10^{-06}		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	1,2,3,7,8,9-Hexachlorodibenzofuran			<8.8x10^{-06}		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	2,3,4,6,7,8-Hexachlorodibenzofuran			<1.6x10^{-05}		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	1,2,3,4,6,7,8-Heptachlorodibenzofuran			<2.3x10^{-05}		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	1,2,3,4,7,8,9-Heptachlorodibenzofuran			<1.5x10^{-05}		B	13.8%	14,300	3.3%	0.015	0.014
FGDw	DOE 4	Octachlorodibenzofuran			<0.00013		B	13.8%	14,300	3.3%	0.015	0.014
ESP	DOE 5	1,2,3,4,6,7,8-Heptachlorodibenzo- p-dioxin			2.3x10^{-06}	0.0000008	B	11.4%	13,800	3.2%	0.047	0.043
ESP	DOE 5	2,3,7,8-Tetrachlorodibenzofuran			6.6x10^{-07}	0.0000006	B	11.4%	13,800	3.2%	0.047	0.043
ESP	DOE 5	1,2,3,4,6,7,8-Heptachlorodibenzofuran			1.6x10^{-06}	0.0000014	B	11.4%	13,800	3.2%	0.047	0.043

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	DOE 5	Octachlorodibenzofuran			1.1x10^{-05}	0.0000023	B	11.4%	13,800	3.2%	0.047	0.043
ESP	DOE 5	TotalPentachlorodibenzofuran			2.8x10^{-06}	0.0000022	B	11.4%	13,800	3.2%	0.047	0.043
FGDw	DOE 6	2,3,7,8-Tetrachlorodibenzo-p-dioxin			<2.1x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			<2.5x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			<2.2x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			<3.3x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			<3.4x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	1,2,3,4,6,7,8-Heptachlorodibenzo- p-dioxin			3.2x10^{-06}	0.000004	L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	Octachlorodibenzo-p-dioxin			1.5x10^{-05}	1.7x10^{-05}	L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	TotalHeptachlorodibenzo-p-dioxin			<1.1x10^{-05}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	2,3,7,8-Tetrachlorodibenzofuran			1.0x10^{-05}	0.0000056	L	17.0%	9,970	1.1%	0.0094	0.0104

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDw	DOE 6	1,2,3,7,8-Pentachlorodibenzofuran			<4.5x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	2,3,4,7,8-Pentachlorodibenzofuran			<3.8x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	1,2,3,4,7,8-Hexachlorodibenzofuran			<4.0x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	1,2,3,7,8,9-Hexachlorodibenzofuran			<5.5x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	1,2,3,6,7,8-Hexachlorodibenzofuran			<3.2x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	2,3,4,6,7,8-Hexachlorodibenzofuran			<2.0x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	1,2,3,4,6,7,8-Heptachlorodibenzofuran			<2.4x10^{-05}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	1,2,3,4,7,8,9-Heptachlorodibenzofuran			<4.0x10^{-06}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	Octachlorodibenzofuran			<1.0x10^{-05}		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	Total Tetrachlorodibenzofuran			ND		L	17.0%	9,970	1.1%	0.0094	0.0104
FGDw	DOE 6	Total Pentachlorodibenzofuran			ND		L	17.0%	9,970	1.1%	0.0094	0.0104

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDd	DOE 7	2,3,7,8-Tetrachlorodibenzo-p-dioxin			<3.0x10^{-06}		S	22.9%	10,500	0.7%	0.017	0.012
FF	DOE 8	2,3,7,8-Tetrachlorodibenzo-p-dioxin			8.1x10^{-07}	1.0x10^{-06}	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			<1.4x10^{-06}		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			<2.8x10^{-06}		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			<1.4x10^{-06}		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			<1.4x10^{-06}		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	1,2,3,4,6,7,8-Heptachlorodibenzo- p-dioxin			<1.4x10^{-06}		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Octachlorodibenzo-p-dioxin			<1.1x10^{-05}		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	TotalTetrachlorodibenzo-p-dioxin			9.3x10^{-06}	0.0000021	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	TotalPentachlorodibenzo-p-dioxin			4.6x10^{-06}	0.0000008	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	TotalHexachlorodibenzo-p-dioxin			2.1x10^{-06}	0.0000023	S	11.2%	11,700	0.9%	0.011	0.009

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FF	DOE 8	TotalHeptachlorodibenzo -p-dioxin			<1.9x10^{-06}		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	2,3,7,8-Tetrachlorodibenz ofuran			6.0x10^{-06}	0.0000052	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	1,2,3,7,8-Pentachlorodibe nzofuran			5.6x10^{-06}	0.0000034	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	2,3,4,7,8-Pentachlorodibe nzofuran			5.1x10^{-06}	0.0000039	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	1,2,3,4,7,8-Hexachlorodib enzofuran			6.5x10^{-06}	0.0000039	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	1,2,3,6,7,8-Hexachlorodib enzofuran			2.2x10^{-06}	0.0000039	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	1,2,3,7,8,9-Hexachlorodib enzofuran			<1.4x10^{-06}		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	2,3,4,6,7,8-Hexachlorodib enzofuran			2.3x10^{-06}	0.0000039	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	1,2,3,4,6,7,8-Heptachlorod ibenzofuran			4.6x10^{-06}	0.000002	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	1,2,3,4,7,8,9-Heptachlorod ibenzofuran			<2.8x10^{-06}		S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Octachlorodibenzofuran			1.9x10^{-06}	0.0000019	S	11.2%	11,700	0.9%	0.011	0.009

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FF	DOE 8	Total Tetrachlorodibenzofuran			6.0x10^{-05}	0.0000171	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Total Pentachlorodibenzofuran			4.7x10^{-05}	0.0000019	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Total Hexachlorodibenzofuran			2.2x10^{-05}	0.000004	S	11.2%	11,700	0.9%	0.011	0.009
FF	DOE 8	Total Heptachlorodibenzofuran			7.0x10^{-06}	0.0000024	S	11.2%	11,700	0.9%	0.011	0.009
ESP	Site 102	2,3,7,8-Tetrachlorodibenzo- p-dioxin			<3.5x10^{-06}		S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	1,2,3,7,8-Pentachlorodibenzo- p-dioxin			<7.0x10^{-06}		S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			<1.8x10^{-05}		S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			<1.4x10^{-05}		S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			<1.8x10^{-05}		S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	1,2,3,4,6,7,8-Heptachlorodibenzo- p-dioxin			1.4x10^{-04}	1.1x10^{-04}	S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	Octachlorodibenzo-p-dioxin			2.5x10^{-04}	2.5x10^{-04}	S	8.8%	12,200	1.0%	0.082	0.058

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 102	2,3,7,8-Tetrachlorodibenzofuran			6.1x10^{-05}	5.6x10^{-05}	S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	1,2,3,7,8-Pentachlorodibenzofuran			<5.6x10^{-06}		S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	2,3,4,7,8-Pentachlorodibenzofuran			4.9x10^{-05}	7.0x10^{-06}	S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	1,2,3,4,7,8-Hexachlorodibenzofuran			5.3x10^{-05}	5.8x10^{-05}	S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	1,2,3,6,7,8-Hexachlorodibenzofuran			<1.4x10^{-05}		S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	1,2,3,7,8,9-Hexachlorodibenzofuran			<1.8x10^{-05}		S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	2,3,4,6,7,8-Hexachlorodibenzofuran			1.7x10^{-05}	5.6x10^{-05}	S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	1,2,3,4,6,7,8-Heptachlorodibenzofuran			2.4x10^{-04}	3.7x10^{-05}	S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	1,2,3,4,7,8,9-Heptachlorodibenzofuran			6.0x10^{-05}	6.0x10^{-05}	S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 102	Octachlorodibenzofuran			6.0x10^{-05}	6.0x10^{-05}	S	8.8%	12,200	1.0%	0.082	0.058
ESP	Site 116	2,3,7,8-Tetrachlorodibenzo-p-dioxin			3.4x10^{-06}	1.1x10^{-05}	B	12.0%	12,900	3.5%	0.056	0.046

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 116	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			<3.3x10^{-07}		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			<3.3x10^{-07}		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			<3.3x10^{-07}		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			<4.1x10^{-06}		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	1,2,3,4,6,7,8-Heptachlorodibenzo- p-dioxin			9.4x10^{-06}	0.0000025	B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	2,3,7,8-Tetrachlorodibenzofuran			<2.5x10^{-06}		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	1,2,3,7,8-Pentachlorodibenzofuran			<3.3x10^{-07}		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	2,3,4,7,8-Pentachlorodibenzofuran			<4.9x10^{-06}		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	1,2,3,4,7,8-Hexachlorodibenzofuran			<4.9x10^{-06}		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	1,2,3,6,7,8-Hexachlorodibenzofuran			2.6x10^{-06}	0.0000012	B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	1,2,3,7,8,9-Hexachlorodibenzofuran			2.5x10^{-06}	0.0000037	B	12.0%	12,900	3.5%	0.056	0.046

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 116	2,3,4,6,7,8-Hexachlorodibenzofuran			<2.5x10^{-07}		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	1,2,3,4,6,7,8-Heptachlorodibenzofuran			<1.1x10^{-06}		B	12.0%	12,900	3.5%	0.056	0.046
ESP	Site 116	1,2,3,4,7,8,9-Heptachlorodibenzofuran			<8.2x10^{-06}		B	12.0%	12,900	3.5%	0.056	0.046
FGDd	Site 116 SNRB	2,3,7,8-Tetrachlorodibenzo-p-dioxin			<1.9x10^{-06}		B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			1.8x10^{-05}	7x10^{-05}	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			3.8x10^{-05}	0.0002	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			4.4x10^{-05}	0.0002	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			8.5x10^{-05}	0.0004	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	1,2,3,4,6,7,8-Heptachlorodibenzo- p-dioxin			0.00052	0.0023	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	2,3,7,8-Tetrachlorodibenzofuran			3.8x10^{-05}	0.0002	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	1,2,3,7,8-Pentachlorodibenzofuran			3.3x10^{-05}	0.0001	B	12.0%	12,900	3.5%	0.031	0.029

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
FGDd	Site 116 SNRB	2,3,4,7,8-Pentachlorodibenzofuran			6.1x10^{-05}	0.0003	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	1,2,3,4,7,8-Hexachlorodibenzofuran			3.4x10^{-06}	NC	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	1,2,3,6,7,8-Hexachlorodibenzofuran			1.1x10^{-06}	NC	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	1,2,3,7,8,9-Hexachlorodibenzofuran			0.00022	0.0009	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	2,3,4,6,7,8-Hexachlorodibenzofuran			1.4x10^{-05}	6x10^{-05}	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	1,2,3,4,6,7,8-Heptachlorodibenzofuran			0.0011	0.0048	B	12.0%	12,900	3.5%	0.031	0.029
FGDd	Site 116 SNRB	1,2,3,4,7,8,9-Heptachlorodibenzofuran			0.00016	0.0007	B	12.0%	12,900	3.5%	0.031	0.029
ESP	Site 122	2,3,7,8-Tetrachlorodibenzo-p-dioxin			ND		B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			ND		B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			ND		B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			ND		B	9.3%	13,370	2.0%	0.17	0.12

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 122	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			ND		B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	1,2,3,4,6,7,8-Heptachlorodibenzo- p-dioxin			3.7x10^{-06}	NA	B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	Octachlorodibenzo-p-dioxin			ND		B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	2,3,7,8-Tetrachlorodibenzofuran			ND		B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	1,2,3,7,8-Pentachlorodibenzofuran			ND		B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	2,3,4,7,8-Pentachlorodibenzofuran			1.5x10^{-06}	NA	B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	1,2,3,4,7,8-Hexachlorodibenzofuran			ND		B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	1,2,3,6,7,8-Hexachlorodibenzofuran			1.5x10^{-06}	NA	B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	1,2,3,7,8,9-Hexachlorodibenzofuran			ND		B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	2,3,4,6,7,8-Hexachlorodibenzofuran			1.5x10^{-05}	NA	B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	1,2,3,4,6,7,8-Heptachlorodibenzofuran			2.2x10^{-05}	NA	B	9.3%	13,370	2.0%	0.17	0.12

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 122	1,2,3,4,7,8,9-Heptachlorodibenzofuran			ND		B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 122	Octachlorodibenzofuran			ND		B	9.3%	13,370	2.0%	0.17	0.12
ESP	Site 22	2,3,7,8-Tetrachlorodibenzo-p-dioxin			$<3.3x10^{-06}$		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			$<3.9x10^{-06}$		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			$<4.8x10^{-06}$		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			$<4.2x10^{-06}$		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			$<4.2x10^{-06}$		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	1,2,3,4,6,7,8-Heptachlorodibenzo- p-dioxin			$<7.7x10^{-06}$		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	Octachlorodibenzo-p-dioxin			$5.5x10^{-05}$	$3x10^{-05}$	S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	2,3,7,8-Tetrachlorodibenzofuran			$<3.6x10^{-06}$		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	1,2,3,7,8-Pentachlorodibenzofuran			$<2.2x10^{-06}$		S	6.8%	11,980	0.4%	0.0018	0.0015

Table B-16.
Coal Site Measurements (Continued)

Control Device	Unit I.D.	Chemical Substance	Coal ppmw		Emission Factor		Coal composition				Particulates	
			Mean	95% C.I.	Mean	95% C.I.	Rank	Ash %	HHV	S %	g/Nm^3	lb/ MBtu
ESP	Site 22	2,3,4,7,8-Pentachlorodibenzofuran			<4.2x10^{-06}		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	1,2,3,4,7,8-Hexachlorodibenzofuran			<6.6x10^{-06}		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	1,2,3,6,7,8-Hexachlorodibenzofuran			<1.2x10^{-06}		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	1,2,3,7,8,9-Hexachlorodibenzofuran			<4.1x10^{-06}		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	2,3,4,6,7,8-Hexachlorodibenzofuran			<4.5x10^{-06}		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	1,2,3,4,6,7,8-Heptachlorodibenzofuran			<4.2x10^{-06}		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	1,2,3,4,7,8,9-Heptachlorodibenzofuran			<4.5x10^{-06}		S	6.8%	11,980	0.4%	0.0018	0.0015
ESP	Site 22	Octachlorodibenzofuran			4.2x10^{-06}	3x10^{-06}	S	6.8%	11,980	0.4%	0.0018	0.0015

B.5 Oil Site Test Data

Table B-17 presents the compiled data from the oil site test reports. This information is in a similar format to the coal data, however, many of the test sites have no control device (the first column is blank). The grouping of the data follows the same hierarchy as for coal data. Because of the simplicity of the recommended emission factor approach for oil-fired sites presented in Chapter 3, no figures are presented here.

Table B-17.
Oil Site Measurements

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	112	Arsenic	0.065	0.70	4.8	4.3	18,900
	13	Arsenic	0.13	0.019	7.2	1.2	19,000
	117	Arsenic	0.13	0.030	4.0	6.3	18,900
	118	Arsenic	0.15	0.23	1.1	0.38	18,800
	107	Arsenic	<1.0		13	17	19,200
	104	Arsenic	<1.1		6.5	6.4	18,800
	106	Arsenic	<1.1		26	34	19,000
	103	Arsenic	<1.1		3.6	7.4	19,100
	105	Arsenic	<1.1		41	3.2	19,000
	108	Arsenic	<1.2		6.8	10	18,300
	109	Arsenic	1.3	2.3	<1.1		18,900
BOOS	13	Arsenic	0.11	0.019	3.8	1.3	19,000
ESP	112	Arsenic	0.065	0.70	<2.4		18,900
ESP	118	Arsenic	0.15	0.23	0.55	0.35	18,800
PJFF	13	Arsenic	0.13	0.019	0.040		19,000
SCR	117	Arsenic	0.13	0.030	2.9	2.9	18,900
	118	Beryllium	0.0070	0.0030	0.68	0.15	18,800

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	13	Beryllium	0.010		<0.26		19,000
	117	Beryllium	0.013	0.0060	2.3	0.76	18,900
	112	Beryllium	0.060	0.017	2.4	1.1	18,900
	109	Beryllium	<0.080		<0.50		18,900
	107	Beryllium	<0.20		<0.10		19,200
	108	Beryllium	<0.20		<0.030		18,300
	104	Beryllium	<0.20		0.17	0.33	18,800
	105	Beryllium	<0.22		<0.36		19,000
	106	Beryllium	<0.22		0.15	0.20	19,000
	103	Beryllium	<0.23		<0.24		19,100
BOOS	13	Beryllium	<0.010		<0.20		19,000
ESP	118	Beryllium	0.0070	0.0030	<0.062		18,800
ESP	112	Beryllium	0.060	0.017	0.58	0.80	18,900
PJFF	13	Beryllium	<0.010		<0.010		19,000
SCR	117	Beryllium	0.013	0.0060	1.9	1.3	18,900
	118	Cadmium	<0.0070		<0.19		18,800
	112	Cadmium	0.013	0.0070	0.45	0.33	18,900

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	117	Cadmium	0.042	0.13	0.11	0.14	18,900
	109	Cadmium	<0.14		3.1	6.9	18,900
	104	Cadmium	<0.20		0.62	0.39	18,800
	108	Cadmium	<0.20		4.0	6.0	18,300
	107	Cadmium	<0.20		1.6	2.0	19,200
	106	Cadmium	<0.22		1.2	1.0	19,000
	105	Cadmium	<0.22		0.69	0.58	19,000
	103	Cadmium	0.24	0.55	3.2	5.1	19,100
	13	Cadmium	<0.53		14	45	19,000
BOOS	13	Cadmium	<0.41		11	41	19,000
ESP	118	Cadmium	<0.0070		<0.18		18,800
ESP	112	Cadmium	0.013	0.0070	0.33	0.90	18,900
PJFF	13	Cadmium	<0.53		<0.0052		19,000
SCR	117	Cadmium	0.042	0.13	0.13	0.17	18,900
	119	Chromium	0.13	0.15	9.5	8.0	18,900
	118	Chromium	0.16	0.10	8.3	5.3	18,800
	109	Chromium	<0.17		11	10	18,900

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	107	Chromium	0.22	0.060	8.0	23	19,200
	117	Chromium	0.23	0.070	9.9	6.1	18,900
	103	Chromium	<0.23		3.5	4.4	19,100
	112	Chromium	0.24	0.10	7.9	3.9	18,900
	105	Chromium	0.27	0.13	2.1	1.7	19,000
	104	Chromium	0.32	0.51	3.0	2.6	18,800
	108	Chromium	0.40	0.60	<1.2		18,300
	106	Chromium	0.41		10	47	19,000
	13	Chromium	0.69	0.63	9.1	2.3	19,000
BOOS	13	Chromium	0.29	0.17	3.0	1.3	19,000
ESP	119	Chromium	0.13	0.15	17	25	18,900
ESP	118	Chromium	0.16	0.10	3.3	1.9	18,800
ESP	112	Chromium	0.24	0.10	3.7	4.3	18,900
PJFF	13	Chromium	0.69	0.63	1.7		19,000
SCR	117	Chromium	0.23	0.070	20	42	18,900
	103	Cobalt	0.60	0.01	10	4.4	19,100
	117	Cobalt	1.2	0.25	38	8.4	18,900

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	118	Cobalt	1.7	1.2	27	14	18,800
	112	Cobalt	1.9	2.3	37	6.7	18,900
	13	Cobalt	2.3	0.055	100	50	19,000
BOOS	13	Cobalt	2.1	0.31	69	7.1	19,000
ESP	118	Cobalt	1.7	1.2	1.9	1.2	18,800
ESP	112	Cobalt	1.9	2.3	10	14	18,900
PJFF	13	Cobalt	2.3	0.055	<0.052		19,000
SCR	117	Cobalt	1.2	0.25	27	20	18,900
	118	Lead	0.25	0.13	7.5	8.3	18,800
	112	Lead	0.39	0.060	29	15	18,900
	117	Lead	0.54	0.31	13	11	18,900
	107	Lead	<1.0		<2.0		19,200
	103	Lead	<1.1		3.7	1.2	19,100
	106	Lead	<1.1		28	9.0	19,000
	105	Lead	<1.1		9.0	19	19,000
	104	Lead	<1.1		<1.6		18,800
	108	Lead	<1.2		10	16	18,300

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	109	Lead	1.3	6.1	17	59	18,900
	13	Lead	4.6	10	8.2	5.4	19,000
BOOS	13	Lead	1.1	1.1	4.7	5.2	19,000
ESP	118	Lead	0.25	0.13	1.8	2.5	18,800
ESP	112	Lead	0.39	0.060	6.0	7.0	18,900
PJFF	13	Lead	4.6	10	0.026		19,000
SCR	117	Lead	0.54	0.31	9.1	4.6	18,900
	13	Manganese	0.11	0.034	8.0	1.3	19,000
	118	Manganese	0.14	0.11	25	33	18,800
	108	Manganese	0.20	0.01	15	30	18,300
	103	Manganese	<0.23		15	12	19,100
	106	Manganese	0.23		43	84	19,000
	112	Manganese	0.34	0.10	5.3	4.3	18,900
	107	Manganese	<0.40		10	17	19,200
	105	Manganese	<0.43		4.0	1.1	19,000
	104	Manganese	0.66	5.7	22	170	18,800
	109	Manganese	<0.80		58	240	18,900

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	117	Manganese	0.88	0.60	14	8.4	18,900
BOOS	13	Manganese	0.081	0.010	4.8	0.68	19,000
ESP	118	Manganese	0.14	0.11	19	10	18,800
ESP	112	Manganese	0.34	0.10	15	19	18,900
PJFF	13	Manganese	0.11	0.034	0.85		19,000
SCR	117	Manganese	0.88	0.60	21	22	18,900
	109	Nickel	5.3	2.3	240	300	18,900
	106	Nickel	6.7		380	74	19,000
	103	Nickel	9.7	1.1	350	21	19,100
	107	Nickel	10	0.1	420	33	19,200
	117	Nickel	17	9.0	990	400	18,900
	118	Nickel	20	10	710	350	18,800
	104	Nickel	23	13	360	760	18,800
	105	Nickel	23	13	510	58	19,000
	108	Nickel	28	6.0	1,400	450	18,300
	13	Nickel	39	3.4	1,900	530	19,000
	112	Nickel	41	3.5	1,100	170	18,900

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	119	Nickel	54	3.7	2,100	1600	18,900
BOOS	13	Nickel	36	5.2	1,400	150	19,000
ESP	118	Nickel	20	10	46	35	18,800
ESP	112	Nickel	41	3.5	300	400	18,900
ESP	119	Nickel	54	3.7	2,200	1900	18,900
PJFF	13	Nickel	39	3.4	1.6		19,000
SCR	117	Nickel	17	9.0	840	990	18,900
	119	Chlorine (as chloride)			2,700	1100	18,900
	13	Chlorine (as chloride)	15	2.7	1,100	780	19,000
	117	Chlorine (as chloride)	25	9.0	2,200	1700	18,900
	112	Chlorine (as chloride)	30	11	2,300	900	18,900
	118	Chlorine (as chloride)	54	5.0	3,900	1300	18,800
	108	Chlorine (as chloride)	78	670			18,300
	109	Chlorine (as chloride)	<90				18,900
	103	Chlorine (as chloride)	130	230			19,100
	104	Chlorine (as chloride)	130	1			18,800
	107	Chlorine (as chloride)	130	310			19,200

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	106	Chlorine (as chloride)	150				19,000
	105	Chlorine (as chloride)	720	1100			19,000
BOOS	13	Chlorine (as chloride)	19	3.8	2,700	920	19,000
ESP	119	Chlorine (as chloride)			2,100	950	18,900
ESP	112	Chlorine (as chloride)	30	11	3,600	2,400	18,900
ESP	118	Chlorine (as chloride)	54	5.0	3,600	1,100	18,800
PJFF	13	Chlorine (as chloride)	15	2.7	1,600	2,700	19,000
SCR	117	Chlorine (as chloride)	25	9.0	1,800	710	18,900
	117	Mercury	0.0023	0.0017	0.60	1.3	18,900
	118	Mercury	0.0040	0.0020	0.98	0.58	18,800
	112	Mercury	0.0060	0.010	1.3	2.1	18,900
	13	Mercury	<0.040		0.23	0.14	19,000
	103	Mercury	<0.090		<3.6		19,100
	106	Mercury	<0.10		<5.0		19,000
	107	Mercury	<0.10		<37		19,200
	104	Mercury	<0.10		12		18,800
	105	Mercury	<0.10		<4.7		19,000

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	108	Mercury	<0.10		<32		18,300
	109	Mercury	<0.90		1.8	9.1	18,900
BOOS	13	Mercury	<0.030		0.16	0.076	19,000
ESP	118	Mercury	0.0040	0.0020	0.50	0.55	18,800
ESP	112	Mercury	0.0060	0.010	0.24	0.24	18,900
PJFF	13	Mercury	<0.040		<0.066		19,000
SCR	117	Mercury	0.0023	0.0017	0.49	0.76	18,900
	112	Selenium	0.050	0.080	<7.1		18,900
	13	Selenium	<0.090		<0.65		19,000
	117	Selenium	0.15	0.20	4.2	1.9	18,900
	107	Selenium	<1.0		<2.0		19,200
	105	Selenium	<1.0		2.8	7.2	19,000
	106	Selenium	<1.0		4.1	4.0	19,000
	103	Selenium	<1.1		0.27	0.11	19,100
	104	Selenium	<1.1		<3.5		18,800
	108	Selenium	<1.2		15	13	18,300
	109	Selenium	<5.0		3.7	16	18,900

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	118	Selenium	5.4	7.4	57	17	18,800
BOOS	13	Selenium	<0.073		2.2	1.6	19,000
ESP	112	Selenium	0.050	0.080	<4.9		18,900
ESP	118	Selenium	5.4	7.4	2.7	3.0	18,800
PJFF	13	Selenium	<0.090		<0.026		19,000
SCR	117	Selenium	0.15	0.20	3.6	1.7	18,900
	108	Benzene			<2.8		18,300
	106	Benzene			<4.0		19,000
	107	Benzene			<4.0		19,200
	104	Benzene			<2.3		18,800
	109	Benzene			<9.7		18,900
	112	Benzene			0.60	0.50	18,900
	117	Benzene			<0.40		18,900
	105	Benzene			<2.5		19,000
	118	Benzene			0.56	2.3	18,800
	119	Benzene			1.8	1.2	18,900
	103	Benzene			<4.4		19,100

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	13	Benzene			1.4	1.0	19,000
BOOS	13	Benzene			1.0	0.84	19,000
ESP	112	Benzene			2.4	0.30	18,900
ESP	119	Benzene			2.0	1.4	18,900
ESP	118	Benzene			0.59	0.45	18,800
PJFF	13	Benzene			2.1	0.34	19,000
BOOS	13	Formaldehyde			<6.9		19,000
ESP	118	Formaldehyde			6.0	3.0	18,800
ESP	112	Formaldehyde			13	39	18,900
ESP	119	Formaldehyde			2.4	5.4	18,900
PJFF	13	Formaldehyde			36	16	19,000
	119	Toluene			5.5	1.5	18,900
	117	Toluene			42	19	18,900
	118	Toluene			6.0	38	18,800
	112	Toluene			16	9.0	18,900
	13	Toluene			5.3	13	19,000
BOOS	13	Toluene			2.3	1.0	19,000

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
ESP	119	Toluene			7.7	6.0	18,900
ESP	112	Toluene			80	80	18,900
ESP	118	Toluene			8.3	9.0	18,800
PJFF	13	Toluene			2.7	3.1	19,000
SCR	117	Toluene			23	12	18,900
	112	Formaldehyde			20	4.0	18,900
	108	Formaldehyde			<20		18,300
	119	Formaldehyde			5.5	8.5	18,900
	118	Formaldehyde			7.5	6.8	18,800
	109	Formaldehyde			400	210	18,900
	117	Formaldehyde			11	1.0	18,900
	106	Formaldehyde			<20		19,000
	105	Formaldehyde			620	850	19,000
	104	Formaldehyde			170	380	18,800
	103	Formaldehyde			<18		19,100
	107	Formaldehyde			600	1900	19,200
	13	Formaldehyde			<8.4		19,000

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	103	Benzo(a)anthracene			<0.0060		19,100
	103	Benzo(a)pyrene			<0.0060		19,100
	103	Benzo(b+k)fluoranthene			<0.0060		19,100
	103	Benzo(g,h,i)perylene			<0.0060		19,100
	103	Chrysene			<0.0060		19,100
	103	Dibenzo(a,h)anthracene			<0.0060		19,100
	103	Indeno(1,2,3-c,d)pyrene			<0.0060		19,100
	104	Benzo(a)anthracene			<0.010		18,800
	104	Benzo(a)pyrene			<0.010		18,800
	104	Benzo(b+k)fluoranthene			<0.010		18,800
	104	Benzo(g,h,i)perylene			<0.010		18,800
	104	Chrysene			<0.010		18,800
	104	Dibenzo(a,h)anthracene			<0.010		18,800
	104	Indeno(1,2,3-c,d)pyrene			<0.010		18,800
	105	Benzo(a)anthracene			0.030	0.11	19,000
	105	Benzo(a)pyrene			<0.0070		19,000
	105	Benzo(b+k)fluoranthene			0.016	0.054	19,000

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	105	Benzo(g,h,i)perylene			0.010	0.030	19,000
	105	Chrysene			<0.0060		19,000
	105	Dibenzo(a,h)anthracene			<0.0060		19,000
	105	Indeno(1,2,3-c,d)pyrene			0.010	0.025	19,000
	106	Benzo(a)anthracene			<0.010		19,000
	106	Benzo(a)pyrene			<0.010		19,000
	106	Benzo(b+k)fluoranthene			<0.010		19,000
	106	Benzo(g,h,i)perylene			<0.010		19,000
	106	Chrysene			<0.010		19,000
	106	Dibenzo(a,h)anthracene			<0.010		19,000
	106	Indeno(1,2,3-c,d)pyrene			<0.010		19,000
	107	Benzo(a)anthracene			0.10	1.2	19,200
	107	Benzo(a)pyrene			<0.010		19,200
	107	Benzo(b+k)fluoranthene			0.040	0.30	19,200
	107	Benzo(g,h,i)perylene			0.030	0.30	19,200
	107	Chrysene			0.060	0.60	19,200
	107	Dibenzo(a,h)anthracene			0.010		19,200

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	107	Dibenzo(a,h)anthracene			0.010	0.060	19,200
	107	Indeno(1,2,3-c,d)pyrene			0.030	0.27	19,200
	108	Benzo(a)anthracene			<0.0080		18,300
	108	Benzo(a)pyrene			<0.0080		18,300
	108	Benzo(b+k)fluoranthene			<0.0080		18,300
	108	Benzo(g,h,i)perylene			<0.0080		18,300
	108	Chrysene			<0.0080		18,300
	108	Indeno(1,2,3-c,d)pyrene			<0.0080		18,300
	109	Benzo(a)anthracene			0.0088	0.0069	18,900
	109	Benzo(a)pyrene					18,900
	109	Benzo(b+k)fluoranthene					18,900
	109	Benzo(g,h,i)perylene					18,900
	109	Chrysene			0.021	0.020	18,900
	109	Dibenzo(a,h)anthracene					18,900
	109	Indeno(1,2,3-c,d)pyrene					18,900
	112	Benzo(a)anthracene			<0.070		18,900
	112	Benzo(a)pyrene			<0.021		18,900

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	112	Benzo(b+k)fluoranthene			<0.026		18,900
	112	Benzo(g,h,i)perylene			<0.024		18,900
	112	Chrysene			<0.074		18,900
	112	Dibenzo(a,h)anthracene			<0.053		18,900
	112	Indeno(1,2,3-c,d)pyrene			<0.021		18,900
ESP	112	Benzo(a)anthracene			<0.078		18,900
ESP	112	Benzo(a)pyrene			<0.012		18,900
ESP	112	Benzo(b+k)fluoranthene			<0.020		18,900
ESP	112	Benzo(g,h,i)perylene			<0.080		18,900
ESP	112	Chrysene			<0.027		18,900
ESP	112	Dibenzo(a,h)anthracene			<0.11		18,900
ESP	112	Indeno(1,2,3-c,d)pyrene			<0.069		18,900
	117	Benzo(a)anthracene			<0.0020		18,900
	117	Benzo(a)pyrene			<0.0080		18,900
	117	Benzo(b+k)fluoranthene			<0.0060		18,900
	117	Benzo(g,h,i)perylene			<0.0090		18,900
	117	Chrysene			<0.0030		18,900

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	117	Dibenzo(a,h)anthracene			<0.017		18,900
	117	Indeno(1,2,3-c,d)pyrene			<0.0080		18,900
SCR	117	Benzo(a)anthracene			<0.0080		18,900
SCR	117	Benzo(a)pyrene			<0.013		18,900
SCR	117	Benzo(b+k)fluoranthene			<0.0050		18,900
SCR	117	Benzo(g,h,i)perylene			<0.0070		18,900
SCR	117	Chrysene			<0.011		18,900
SCR	117	Dibenzo(a,h)anthracene			<0.0080		18,900
SCR	117	Indeno(1,2,3-c,d)pyrene			<0.010		18,900
	118	Benzo(a)anthracene			<0.010		18,800
	118	Benzo(a)pyrene			<0.012		18,800
	118	Benzo(b+k)fluoranthene			<0.0060		18,800
	118	Benzo(g,h,i)perylene			<0.0060		18,800
	118	Chrysene			<0.018		18,800
	118	Dibenzo(a,h)anthracene			<0.013		18,800
	118	Indeno(1,2,3-c,d)pyrene			<0.010		18,800
ESP	118	Benzo(a)anthracene			<0.012		18,800

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
ESP	118	Benzo(a)pyrene			<0.017		18,800
ESP	118	Benzo(b+k)fluoranthene			<0.0080		18,800
ESP	118	Benzo(g,h,i)perylene			<0.0090		18,800
ESP	118	Chrysene			<0.020		18,800
ESP	118	Dibenzo(a,h)anthracene			<0.019		18,800
ESP	118	Indeno(1,2,3-c,d)pyrene			<0.015		18,800
	119	Benzo(a)anthracene			<0.050		18,900
	119	Benzo(a)pyrene			<0.24		18,900
	119	Benzo(b+k)fluoranthene			<0.090		18,900
	119	Benzo(g,h,i)perylene			<0.050		18,900
	119	Chrysene			0.11		18,900
	119	Dibenzo(a,h)anthracene			<0.040		18,900
	119	Indeno(1,2,3-c,d)pyrene			<0.040		18,900
ESP	119	Benzo(a)anthracene			<0.027		18,900
ESP	119	Benzo(a)pyrene			<0.016		18,900
ESP	119	Benzo(b+k)fluoranthene			<0.046		18,900
ESP	119	Benzo(g,h,i)perylene			<0.027		18,900

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
ESP	119	Chrysene			<0.030		18,900
ESP	119	Dibenzo(a,h)anthracene			<0.020		18,900
ESP	119	Indeno(1,2,3-c,d)pyrene			<0.020		18,900
	118	2,3,7,8-Tetrachlorodibenzo-p-dioxin			$<4.1\times10^{-6}$		18,800
	118	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			$<4.1\times10^{-6}$		18,800
	118	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			4.9×10^{-6}		18,800
	118	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			$<2.6\times10^{-6}$		18,800
	118	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			$<4.4\times10^{-6}$		18,800
	118	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin			7.8×10^{-6}		18,800
	118	Octachlorodibenzo-p-dioxin			1.6×10^{-5}		18,800
	118	2,3,7,8-Tetrachlorodibenzofuran			3.9×10^{-6}		18,800
	118	1,2,3,7,8-Pentachlorodibenzofuran			2.9×10^{-6}		18,800
	118	2,3,4,7,8-Pentachlorodibenzofuran			4.6×10^{-6}		18,800
	118	1,2,3,4,7,8-Hexachlorodibenzofuran			3.7×10^{-6}		18,800
	118	1,2,3,6,7,8-Hexachlorodibenzofuran			$<1.8\times10^{-6}$		18,800
	118	1,2,3,7,8,9-Hexachlorodibenzofuran			$<3.8\times10^{-6}$		18,800
	118	2,3,4,6,7,8-Hexachlorodibenzofuran			$<2.7\times10^{-6}$		18,800

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	118	1,2,3,4,6,7,8-Heptachlorodibenzofuran			$<9.2x10^{-6}$		18,800
	118	1,2,3,4,7,8,9-Heptachlorodibenzofuran			$<1.4x10^{-5}$		18,800
	118	Octachlorodibenzofuran			$<1.2x10^{-5}$		18,800
ESP	118	2,3,7,8-Tetrachlorodibenzo-p-dioxin			$<3.9x10^{-6}$		18,800
ESP	118	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			$<4.5x10^{-6}$		18,800
ESP	118	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			$<1.1x10^{-5}$		18,800
ESP	118	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			$<3.3x10^{-6}$		18,800
ESP	118	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			$<5.5x10^{-6}$		18,800
ESP	118	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin			$<1.7x10^{-5}$		18,800
ESP	118	Octachlorodibenzo-p-dioxin			$<2.1x10^{-5}$		18,800
ESP	118	2,3,7,8-Tetrachlorodibenzofuran			$<1.7x10^{-6}$		18,800
ESP	118	1,2,3,7,8-Pentachlorodibenzofuran			$<2.4x10^{-6}$		18,800
ESP	118	2,3,4,7,8-Pentachlorodibenzofuran			$<2.5x10^{-6}$		18,800
ESP	118	1,2,3,4,7,8-Hexachlorodibenzofuran			$<4.5x10^{-6}$		18,800
ESP	118	1,2,3,6,7,8-Hexachlorodibenzofuran			$<2.4x10^{-6}$		18,800
ESP	118	1,2,3,7,8,9-Hexachlorodibenzofuran			$<6.4x10^{-6}$		18,800
ESP	118	2,3,4,6,7,8-Hexachlorodibenzofuran			$<4.5x10^{-6}$		18,800

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
ESP	118	1,2,3,4,6,7,8-Heptachlorodibenzofuran			$<1.3x10^{-5}$		18,800
ESP	118	1,2,3,4,7,8,9-Heptachlorodibenzofuran			$<2.5x10^{-5}$		18,800
ESP	118	Octachlorodibenzofuran			$<1.4x10^{-5}$		18,800
	119	2,3,7,8-Tetrachlorodibenzo-p-dioxin			$6.5x10^{-6}$		18,900
	119	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			$6.5x10^{-6}$		18,900
	119	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			$<1.3x10^{-5}$		18,900
	119	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			$<5.7x10^{-6}$		18,900
	119	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			$<8.0x10^{-6}$		18,900
	119	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin			$1.2x10^{-5}$		18,900
	119	Octachlorodibenzo-p-dioxin			$3.9x10^{-5}$		18,900
	119	2,3,7,8-Tetrachlorodibenzofuran			$7.3x10^{-6}$		18,900
	119	1,2,3,7,8-Pentachlorodibenzofuran			$5.8x10^{-6}$		18,900
	119	2,3,4,7,8-Pentachlorodibenzofuran			$6.5x10^{-6}$		18,900
	119	1,2,3,4,7,8-Hexachlorodibenzofuran			$5.8x10^{-6}$		18,900
	119	1,2,3,6,7,8-Hexachlorodibenzofuran			$3.6x10^{-6}$		18,900
	119	1,2,3,7,8,9-Hexachlorodibenzofuran			$<5.4x10^{-6}$		18,900
	119	2,3,4,6,7,8-Hexachlorodibenzofuran			$<4.8x10^{-6}$		18,900

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
	119	1,2,3,4,6,7,8-Heptachlorodibenzofuran			$9.4x10^{-6}$		18,900
	119	1,2,3,4,7,8,9-Heptachlorodibenzofuran			$<1.0x10^{-5}$		18,900
	119	Octachlorodibenzofuran			$<1.0x10^{-5}$		18,900
ESP	119	2,3,7,8-Tetrachlorodibenzo-p-dioxin			$<2.6x10^{-6}$		18,900
ESP	119	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			$3.0x10^{-6}$		18,900
ESP	119	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			$7.7x10^{-6}$		18,900
ESP	119	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			$8.0x10^{-6}$		18,900
ESP	119	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			$9.7x10^{-6}$		18,900
ESP	119	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin			$5.8x10^{-5}$		18,900
ESP	119	Octachlorodibenzo-p-dioxin			0.00025		18,900
ESP	119	2,3,7,8-Tetrachlorodibenzofuran			$<7.0x10^{-6}$		18,900
ESP	119	1,2,3,7,8-Pentachlorodibenzofuran			$7.8x10^{-6}$		18,900
ESP	119	2,3,4,7,8-Pentachlorodibenzofuran			$6.0x10^{-6}$		18,900
ESP	119	1,2,3,4,7,8-Hexachlorodibenzofuran			$9.3x10^{-6}$		18,900
ESP	119	1,2,3,6,7,8-Hexachlorodibenzofuran			$4.3x10^{-6}$		18,900
ESP	119	1,2,3,7,8,9-Hexachlorodibenzofuran			$<2.8x10^{-6}$		18,900
ESP	119	2,3,4,6,7,8-Hexachlorodibenzofuran			$2.9x10^{-6}$		18,900

Table B-17.
Oil Site Measurements (Continued)

Control Device	Unit Name	Chemical Substance	Oil (ppmw)		Emission Factor		Oil HHV Btu/lb
			Mean	95% C.I	Mean	95% C.I.	
ESP	119	1,2,3,4,6,7,8-Heptachlorodibenzofuran			$2.0x10^{-5}$		18,900
ESP	119	1,2,3,4,7,8,9-Heptachlorodibenzofuran			$<8.5x10^{-6}$		18,900
ESP	119	Octachlorodibenzofuran			$<2.0x10^{-5}$		18,900

B.6 Gas Site Test Data

Table B-18 presents the compiled data from the gas site test reports. This information is in a similar format to the oil data, however, none of the test sites have emission control devices. Only two sites have been tested extensively. Benzene and formaldehyde were measured at a number of sites after more extensive measurements in an oil-firing mode were performed. The grouping of the data follows the same hierarchy as for oil data. Because of the simplicity of the recommended emission factor approach for gas-fired units presented in Chapter 3, no figures are presented here.

Table B-18.
Gas Site Measurements

Unit Name	Chemical Substance	Fuel (ppmv)		Emission Factor		Natural Gas	
		Mean	95% C.I.	Mean	95% C.I.	HHV	lb/10^{12} Btu
Site 120	Arsenic	<0.0006		0.23	0.42	1,002	<0.12
Site 121	Arsenic	<0.0012		0.23	0.30	998	<0.25
Site 120	Beryllium			<0.01		1,002	
Site 121	Beryllium			<0.01		998	
Site 120	Cadmium			<0.03		1,002	
Site 121	Cadmium			0.05	0.42	998	
Site 120	Chlorine	<0.2				1,002	<20
Site 121	Chlorine	<0.2				998	<20
Site 120	Chromium			1.1	0.70	1,002	
Site 121	Chromium			1.1	5.5	998	
Site 120	Cobalt	<0.1		0.12	0.12	1,002	<16
Site 121	Cobalt	<0.1		<0.10		998	<16
Site 120	Lead	<0.1		0.27	0.04	1,002	<58
Site 121	Lead	<0.1		0.58	0.09	998	<58
Site 120	Manganese			0.38	0.18	1,002	
Site 121	Manganese			0.44	0.60	998	
Site 120	Mercury	$<1.1 \times 10^{-6}$		<0.35		1,002	<0.0006

Table B-18.
Gas Site Measurements (Continued)

Unit Name	Chemical Substance	Fuel (ppmv)		Emission Factor		Natural Gas	
		Mean	95% C.I.	Mean	95% C.I.	HHV	lb/10^{12} Btu
Site 121	Mercury	$2.2x10^{-6}$		<0.40		998	0.0012
Site 120	Nickel	<0.05		3.6	2.1	1,002	<8.2
Site 121	Nickel	<0.05		1.2	4.2	998	<8.2
Site 120	Selenium	<0.002		<0.02		1,002	<0.44
Site 121	Selenium	0.002		<0.03		998	0.44
Site 103	Benzene			<4.4			
Site 104	Benzene	210		<2.3		1,036	46,000
Site 105	Benzene	160		<1.0		1,043	35,000
Site 106	Benzene			<4.0		947	
Site 107	Benzene			<4.0		957	
Site 108	Benzene	280		<2.2		984	62,000
Site 120	Benzene	6		<0.41		1,002	1,300
Site 121	Benzene	7		1.4	1.6	998	1,500
Site 120	Toluene	6		7.3	22	1,002	1,400
Site 121	Toluene	6		13	2.7	998	1,400
Site 103	Formaldehyde			<18			
Site 104	Formaldehyde			25	90	1,036	
Site 105	Formaldehyde			600	410	1,043	

Table B-18.
Gas Site Measurements (Continued)

Unit Name	Chemical Substance	Fuel (ppmv)		Emission Factor		Natural Gas	
		Mean	95% C.I.	Mean	95% C.I.	HHV	lb/10^{12} Btu
Site 106	Formaldehyde			82	82	947	
Site 107	Formaldehyde			800	320	957	
Site 108	Formaldehyde			<12		984	
Site 109	Formaldehyde			46	9.0		
Site 120	Formaldehyde			4.7	4.2	1,002	
Site 121	Formaldehyde			5.9	1.9	998	
Site 120	Benzo(a)anthracene			<0.002		1,002	
Site 120	Benzo(a)pyrene			<0.001		1,002	
Site 120	Benzo(b,j&k)fluoranthene			<0.002		1,002	
Site 120	Benzo(g,h,i)perylene			<0.001		1,002	
Site 120	Chrysene			<0.002		1,002	
Site 120	Dibenzo(a,h)anthracene			<0.001		1,002	
Site 120	Indeno(1,2,3-cd)pyrene			<0.002		1,002	
Site 121	Benzo(a)anthracene			<0.004		998	
Site 121	Benzo(a)pyrene			<0.006		998	
Site 121	Benzo(b,j&k)fluoranthene			<0.003		998	
Site 121	Benzo(g,h,i)perylene			<0.003		998	
Site 121	Chrysene			<0.005		998	

Table B-18.
Gas Site Measurements (Continued)

Unit Name	Chemical Substance	Fuel (ppmv)		Emission Factor		Natural Gas	
		Mean	95% C.I.	Mean	95% C.I.	HHV	lb/10^{12} Btu
Site 121	Dibenzo(a,h)anthracene			<0.003		998	
Site 121	Indeno(1,2,3-cd)pyrene			<0.004		998	
	2,3,7,8-Tetrachlorodibenzo-p-dioxin			<0.000003			
	1,2,3,7,8-Pentachlorodibenzo-p-dioxin			<0.000006			
	1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin			<0.000008			
	1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin			<0.000004			
	1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin			<0.000006			
	1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin			<0.000006			
	Octachlorodibenzo-p-dioxin			0.000004			
	2,3,7,8-Tetrachlorodibenzofuran			<0.000003			
	1,2,3,7,8-Pentachlorodibenzofuran			<0.000004			
	2,3,4,7,8-Pentachlorodibenzofuran			<0.000004			
	1,2,3,4,7,8-Hexachlorodibenzofuran			<0.000003			
	1,2,3,6,7,8-Hexachlorodibenzofuran			<0.000002			
	1,2,3,7,8,9-Hexachlorodibenzofuran			<0.000003			
	2,3,4,6,7,8-Hexachlorodibenzofuran			<0.000003			
	1,2,3,4,6,7,8-Heptachlorodibenzofuran			0.000122			

Table B-18.
Gas Site Measurements (Continued)

Unit Name	Chemical Substance	Fuel (ppmv)		Emission Factor		Natural Gas	
		Mean	95% C.I.	Mean	95% C.I.	HHV	lb/10^{12} Btu
	1,2,3,4,7,8,9-Heptachlorodibenzofuran			<0.000007			
	Octachlorodibenzofuran			<0.000006			

B.7 Radionuclide Test Data

Table B-19 presents the coal (and two oil sites) data for radionuclide measurements in the fuel and gas emission stream. Various reports have presented the results on either a μg/Nm^3 or pCi/Nm^3 basis. Since the isotopes of interest are all nonvolatile metals, it is convenient to convert all the emission levels to a specific activity basis (pCi/gram of particulate matter). As discussed in Chapter 3, at a number of the recent test sites analytical procedures used were not sensitive enough to actually detect the levels present. These "high" non-detect values are shaded in Table B-19 and were not used in developing the recommended emission factors in Chapter 3. Because this detection issue restricts the data set severely, additional data from an earlier study [10] has been included. This study examined coal and stack gas particulate samples from nine coal-fired units. The plant sizes ranged from 100 to 750 MW and were from old and new (Subpart D) units. As can be seen in Table B-19, these data complement the recent data well. This table is organized by isotope, in increasing fuel concentration. The oil data at the end of the table is from two sites and indicates a larger degree of variability.

Table B-19.
Radionuclide Data

Control Device	Control Device	Isotop	Coal pCi/g	Concentration		Specific Activity pCi/gram	Coal		PM g/Nm^3	Coal Ash pCi/g	Enrich. Ratio
				µg/Nm^3	pCi/Nm^3		Rank	Ash %			
FGDw	UARG 900	Pb^{210}	0.19			30	S	20%	0.19	1.0	31.6
ESP	UARG 500	Pb^{210}	0.20			<5	S	20%	0.0025	1.0	<5
ESP	UARG 700	Pb^{210}	0.23			36	S	10%	0.059	2.3	15.7
ESP	UARG 800	Pb^{210}	0.24			9.0	B	10%	0.18	2.4	3.8
ESP	UARG 600	Pb^{210}	0.40			8.0	B	10%	0.11	4.0	2.0
ESP	UARG 400	Pb^{210}	0.40			20	S	20%	0.076	2.0	10.0
ESP	Site 116	Pb^{210}	<0.50		300	<5,400	B	12.0%	0.0560	0.0	0.0
FGDd	Site 116 SNRB	Pb^{210}	<0.50		13	420	B	12.0%	0.0310	0.0	0.0
ESP	UARG 300	Pb^{210}	0.60			10	B	10%	0.21	6.0	1.7
FF	DOE 8	Pb^{210}	<0.60	<5.00x10^{-08}		<340	S	11.2%	0.011	0.0	0.0
FGDd	DOE 7	Pb^{210}	0.60			0	S	22.9%	0.017	2.6	0.0
ESP	DOE 3	Pb^{210}	<0.66	<5.00x10^{-07}		<260	B	12.0%	0.15	0.0	0.0
FF	Site 115	Pb^{210}	<0.69		0.75	<300	S	11.2%	0.0025	0.0	0.0
ESP	UARG 100	Pb^{210}	0.75			12	B	10%	0.15	7.5	1.5
ESP	DOE 6	Pb^{210}	0.76		290	<15,000	L	17.0%	0.020	4.5	0.0
FGDw	DOE 6	Pb^{210}	0.76		150	<16,000	L	17.0%	0.0094	4.5	0.0
ESP	DOE 4	Pb^{210}	1.3				B	13.8%	0.14	9.4	0.0

Table B-19.
Radionuclide Data (Continued)

Control Device	Control Device	Isotop	Coal pCi/g	Concentration		Specific Activity pCi/gram	Coal		PM g/Nm^3	Coal Ash pCi/g	Enrich. Ratio
				µg/Nm^3	pCi/Nm^3		Rank	Ash %			
ESP	DOE 2	Pb^{210}	1.7		450	<18,000	B	12.0%	0.025	14.2	0.0
ESP	DOE 5	Pb^{210}	2.0		180	3,800	B	11.4%	0.047	17.5	217
FF	DOE 2 FF	Pb^{210}	2.0		460	<33,000	B	12.0%	0.014	16.7	0.0
ESP	DOE 6	Pb^{211}	<0.85		310	<16,000	L	17.0%	0.020	0.0	0.0
FGDw	DOE 6	Pb^{211}	<0.85		150	<16,000	L	17.0%	0.0094	0.0	0.0
ESP	DOE 2	Pb^{211}	<1.5		653	<26,000	B	12.0%	0.025	0.0	0.0
FF	DOE 2 FF	Pb^{211}	<1.8		580	<41,000	B	12.0%	0.014	0.0	0.0
ESP	DOE 6	Pb^{212}	0.21		23	<1,200	L	17.0%	0.020	1.2	0.0
FGDw	DOE 6	Pb^{212}	0.21		14	<1,500	L	17.0%	0.0094	1.2	0.0
FF	DOE 2 FF	Pb^{212}	0.28		40	<2,900	B	12.0%	0.014	2.3	0.0
ESP	DOE 2	Pb^{212}	0.35		41	<1,600	B	12.0%	0.025	2.9	0.0
ESP	UARG 800	Po^{210}	0.05			15	B	10%	0.18	0.5	30.0
ESP	UARG 700	Po^{210}	0.10			6.6	S	10%	0.059	1.0	6.6
ESP	UARG 500	Po^{210}	0.12			29	S	20%	0.0025	0.6	48.3
FF	Site 115	Po^{210}	0.12		0.06	<24	S	11.2%	0.0025	1.1	<22
FF	DOE 8	Po^{210}	0.12	$<6.00 \times 10^{-11}$		<23	S	11.2%	0.011	1.1	<21
FGDw	UARG 900	Po^{210}	0.14			12	S	20%	0.19	0.7	17.1
FGDd	DOE 7	Po^{210}	0.20				S	22.9%	0.017	0.9	0.0

Table B-19.
Radionuclide Data (Continued)

Control Device	Control Device	Isotop	Coal pCi/g	Concentration		Specific Activity pCi/gram	Coal		PM g/Nm^3	Coal Ash pCi/g	Enrich. Ratio
				µg/Nm^3	pCi/Nm^3		Rank	Ash %			
ESP	DOE 3	Po^{210}	0.24	5.00x10^{-10}		16	B	12.0%	0.15	2.0	8.0
ESP	UARG 400	Po^{210}	0.29			17	S	20%	0.076	1.5	11.7
ESP	UARG 600	Po^{210}	0.41			24	B	10%	0.11	4.1	5.9
ESP	UARG 300	Po^{210}	0.49			24	B	10%	0.21	4.9	4.9
ESP	UARG 100	Po^{210}	0.50			14	B	10%	0.15	5.0	2.7
ESP	UARG 700	Ra^{226}	0.06			0.70	S	10%	0.059	0.6	1.2
ESP	UARG 500	Ra^{226}	0.08			<0.1	S	20%	0.0025	0.4	<0.25
ESP	UARG 800	Ra^{226}	0.11			1.5	B	10%	0.18	1.1	1.4
FGDw	UARG 900	Ra^{226}	0.14			0.60	S	20%	0.19	0.7	0.9
FGDd	DOE 7	Ra^{226}	0.20			0	S	22.9%	0.017	0.9	0.0
ESP	UARG 600	Ra^{226}	0.27			0.20	B	10%	0.11	2.7	0.1
ESP	Site 116	Ra^{226}	0.27		5.3	<95	B	12.0%	0.0560	2.3	0.0
FGDd	Site 116 SNRB	Ra^{226}	0.27		6.7	<220	B	12.0%	0.0310	2.3	0.0
FF	DOE 8	Ra^{226}	0.28	4.00x10^{-07}		35	S	11.2%	0.011	2.5	14.0
FF	Site 115	Ra^{226}	0.28		0.10	40	S	11.2%	0.0025	2.5	16.1
ESP	UARG 400	Ra^{226}	0.34			1.8	S	20%	0.076	1.7	1.1
ESP	DOE 6	Ra^{226}	0.37		61	3,100	L	17.0%	0.020	2.2	1,424
FGDw	DOE 6	Ra^{226}	0.37		17	<1,800	L	17.0%	0.0094	2.2	0.0

Table B-19.
Radionuclide Data (Continued)

Control Device	Control Device	Isotop	Coal pCi/g	Concentration		Specific Activity pCi/gram	Coal		PM g/Nm^3	Coal Ash pCi/g	Enrich. Ratio
				µg/Nm^3	pCi/Nm^3		Rank	Ash %			
ESP	DOE 3	Ra^{226}	0.43	<3.00x10^{-07}		<1.7	B	12.0%	0.15	3.6	<0.5
ESP	UARG 300	Ra^{226}	0.45			1.2	B	10%	0.21	4.5	0.3
ESP	UARG 100	Ra^{226}	0.45			2.7	B	10%	0.15	4.5	0.6
ESP	DOE 2	Ra^{226}	0.49		51	<2,000	B	12.0%	0.025	4.1	0.0
FF	DOE 2 FF	Ra^{226}	0.50		49	<3,500	B	12.0%	0.014	4.2	0.0
ESP	DOE 5	Ra^{226}	0.67		16	<340	B	11.4%	0.047	5.9	0.0
ESP	DOE 6	Ra^{228}	0.18		72	<3,700	L	17.0%	0.020	1.1	0.0
FGDw	DOE 6	Ra^{228}	0.18		29	<3,100	L	17.0%	0.0094	1.1	0.0
ESP	UARG 500	Ra^{228}	0.20			<0.5	S	20%	0.0025	1.0	<0.5
ESP	UARG 800	Ra^{228}	0.20			<0.1	B	10%	0.18	2.0	<0.05
ESP	DOE 2	Ra^{228}	0.22		132	<5,300	B	12.0%	0.025	1.8	0.0
ESP	Site 116	Ra^{228}	0.23		9.4	<170	B	12.0%	0.0560	1.9	0.0
FGDd	Site 116 SNRB	Ra^{228}	0.23		14	<450	B	12.0%	0.0310	1.9	0.0
FF	Site 115	Ra^{228}	0.24		0.84	340	S	11.2%	0.0025	2.1	159
FF	DOE 2 FF	Ra^{228}	<0.35		120	<8,600	B	12.0%	0.014	0.0	0.0
ESP	DOE 3	Ra^{228}	0.40	<2.00x10^{-08}		<24	B	12.0%	0.15	3.3	0.0
ESP	DOE 5	Ra^{228}	0.46		36	<770	B	11.4%	0.047	4.0	0.0
ESP	UARG 600	Ra^{228}	0.50			<0.5	B	10%	0.11	5.0	<0.1

Table B-19.
Radionuclide Data (Continued)

Control Device	Control Device	Isotop	Coal pCi/g	Concentration		Specific Activity pCi/gram	Coal		PM g/Nm^3	Coal Ash pCi/g	Enrich. Ratio
				µg/Nm^3	pCi/Nm^3		Rank	Ash %			
FF	DOE 8	Ra^{228}	0.74	<1.00x10^{-08}		<230	S	11.2%	0.011	6.6	0.0
ESP	DOE 4	Ra^{228}	1.2				B	13.8%	0.14	8.5	0.0
FGDd	DOE 7	Ra^{228}	<2.5			0	S	22.9%	0.017	0.0	0.0
ESP	UARG 500	Th^{228}	0.10			12	S	20%	0.0025	0.5	24.0
FGDw	UARG 900	Th^{228}	0.10			1.7	S	20%	0.19	0.5	3.4
ESP	UARG 700	Th^{228}	0.15			1.5	S	10%	0.059	1.5	1.0
ESP	UARG 800	Th^{228}	0.16			2.3	B	10%	0.18	1.6	1.4
FF	DOE 8	Th^{228}	0.17	<1.00x10^{-10}		<9.5	S	11.2%	0.011	1.5	<6
ESP	DOE 3	Th^{228}	0.18	<4.00x10^{-09}		<23	B	12.0%	0.15	1.5	<15
ESP	UARG 600	Th^{228}	0.28			2.5	B	10%	0.11	2.8	0.9
ESP	UARG 100	Th^{228}	0.35			2.8	B	10%	0.15	3.5	0.8
ESP	UARG 300	Th^{228}	0.43			5.0	B	10%	0.21	4.3	1.2
ESP	UARG 400	Th^{228}	0.47			5.8	S	20%	0.076	2.4	2.5
FGDd	DOE 7	Th^{228}	<0.60			0	S	22.9%	0.017	0.0	0.0
ESP	DOE 6	Th^{229}	<0.29		150	<7,700	L	17.0%	0.020	0.0	0.0
FGDw	DOE 6	Th^{229}	<0.29		62	<6,600	L	17.0%	0.0094	0.0	0.0
ESP	DOE 2	Th^{229}	<0.58		255	<10,000	B	12.0%	0.025	0.0	0.0
FF	DOE 2 FF	Th^{229}	<0.70		240	<17,000	B	12.0%	0.014	0.0	0.0

Table B-19.
Radionuclide Data (Continued)

Control Device	Control Device	Isotop	Coal pCi/g	Concentration		Specific Activity pCi/gram	Coal		PM g/Nm^3	Coal Ash pCi/g	Enrich. Ratio
				µg/Nm^3	pCi/Nm^3		Rank	Ash %			
ESP	UARG 500	Th^{230}	0.18			20	S	20%	0.0025	0.9	22.2
ESP	UARG 700	Th^{230}	0.18			2.8	S	10%	0.059	1.8	1.6
ESP	UARG 800	Th^{230}	0.18			3.5	B	10%	0.18	1.8	1.9
FF	Site 115	Th^{230}	0.20		0.17	<68	S	11.2%	0.0025	1.8	0.0
FGDw	UARG 900	Th^{230}	0.22			2.4	S	20%	0.19	1.1	2.2
FF	DOE 8	Th^{230}	0.25	<0.000004		<6.1	S	11.2%	0.011	2.2	<2.8
ESP	UARG 600	Th^{230}	0.44			3.8	B	10%	0.11	4.4	0.9
ESP	UARG 400	Th^{230}	0.52			7.4	S	20%	0.076	2.6	2.8
ESP	UARG 100	Th^{230}	0.57			6.2	B	10%	0.15	5.7	1.1
ESP	UARG 300	Th^{230}	0.70			6.5	B	10%	0.21	7.0	0.9
ESP	DOE 3	Th^{230}	0.82	0.0004		55	B	12.0%	0.15	6.8	8.0
FGDd	DOE 7	Th^{230}	1.1				S	22.9%	0.017	4.8	0.0
ESP	DOE 6	Th^{230}	<4.0		1,500	<77,000	L	17.0%	0.020	0.0	0.0
ESP	Site 116	Th^{230}	<4.0		230	<4,100	B	12.0%	0.0560	0.0	0.0
FGDd	Site 116 SNRB	Th^{230}	<4.0		320	<10,000	B	12.0%	0.0310	0.0	0.0
FGDw	DOE 6	Th^{230}	<4.0		680	<72,000	L	17.0%	0.0094	0.0	0.0
ESP	DOE 2	Th^{230}	6.2		2,420	<97,000	B	12.0%	0.025	51.7	0.0
FF	DOE 2 FF	Th^{230}	<7.1		2,600	<190,000	B	12.0%	0.014	0.0	0.0

Table B-19.
Radionuclide Data (Continued)

Control Device	Control Device	Isotop	Coal pCi/g	Concentration		Specific Activity pCi/gram	Coal		PM g/Nm^3	Coal Ash pCi/g	Enrich. Ratio
				µg/Nm^3	pCi/Nm^3		Rank	Ash %			
ESP	DOE 5	Th^{230}	<7.4		650	<14,000	B	11.4%	0.047	0.0	0.0
FF	Site 115	Th^{232}	0.10		0.11	<44	S	11.2%	0.0025	0.9	0.0
FGDw	UARG 900	Th^{232}	0.10			1.5	S	20%	0.19	0.5	3.0
ESP	UARG 500	Th^{232}	0.13			12	S	20%	0.0025	0.7	18.5
ESP	UARG 700	Th^{232}	0.15			1.8	S	10%	0.059	1.5	1.2
ESP	UARG 800	Th^{232}	0.16			2.4	B	10%	0.18	1.6	1.5
FF	DOE 8	Th^{232}	0.17	<0.98		<9.5	S	11.2%	0.011	1.5	<6
ESP	DOE 3	Th^{232}	0.18	<32		<22	B	12.0%	0.15	1.5	<15
ESP	UARG 100	Th^{232}	0.28			2.9	B	10%	0.15	2.8	1.0
ESP	UARG 600	Th^{232}	0.32			2.2	B	10%	0.11	3.2	0.7
FGDd	DOE 7	Th^{232}	0.40				S	22.9%	0.017	1.7	0.0
ESP	UARG 300	Th^{232}	0.44			4.9	B	10%	0.21	4.4	1.1
ESP	UARG 400	Th^{232}	0.45			5.3	S	20%	0.076	2.3	2.4
ESP	DOE 6	Th^{234}	1.1		270	<14,000	L	17.0%	0.020	6.5	0.0
FGDw	DOE 6	Th^{234}	1.1		97	<10,000	L	17.0%	0.0094	6.5	0.0
FF	DOE 2 FF	Th^{234}	2.7		460	33,000	B	12.0%	0.014	22.5	1,467
ESP	DOE 2	Th^{234}	2.8		340	<14,000	B	12.0%	0.025	23.3	0.0
FGDd	DOE 7	U^{234}	<0.10				S	22.9%	0.017	0.0	0.0

Table B-19.
Radionuclide Data (Continued)

Control Device	Control Device	Isotop	Coal pCi/g	Concentration		Specific Activity pCi/gram	Coal		PM g/Nm^3	Coal Ash pCi/g	Enrich. Ratio
				µg/Nm^3	pCi/Nm^3		Rank	Ash %			
FF	Site 115	U^{234}	0.13		0.06	<24	S	11.2%	0.0025	1.2	0.0
FF	DOE 8	U^{234}	0.35	<0.00002		<12	S	11.2%	0.011	3.1	<3.9
ESP	DOE 3	U^{234}	1.1	0.0002		8.8	B	12.0%	0.15	8.8	1.0
ESP	DOE 6	U^{234}	9.6		6,300	<320,000	L	17.0%	0.020	56.5	0.0
FGDw	DOE 6	U^{234}	9.6		3,000	<320,000	L	17.0%	0.0094	56.5	0.0
ESP	Site 116	U^{234}	<13		970	<17,000	B	12.0%	0.0560	0.0	0.0
FGDd	Site 116 SNRB	U^{234}	<13		1,300	<42,000	B	12.0%	0.0310	0.0	0.0
ESP	DOE 2	U^{234}	<22		10,250	<410,000	B	12.0%	0.025	0.0	0.0
ESP	DOE 5	U^{234}	<27		2,900	<62,000	B	11.4%	0.047	0.0	0.0
FF	DOE 2 FF	U^{234}	<29		11,100	<790,000	B	12.0%	0.014	0.0	0.0
FF	Site 115	U^{235}	0.01		0.06	<24	S	11.2%	0.0025	0.1	0.0
FF	DOE 8	U^{235}	0.02	<0.04		<7.6	S	11.2%	0.011	0.2	0.0
ESP	DOE 3	U^{235}	0.04	<0.048		<0.65	B	12.0%	0.15	0.3	<2
ESP	DOE 4	U^{235}	0.07				B	13.8%	0.14	0.5	0.0
ESP	DOE 6	U^{235}	0.09		67	<3,400	L	17.0%	0.020	0.5	0.0
FGDw	DOE 6	U^{235}	0.09		26	<2,800	L	17.0%	0.0094	0.5	0.0
FGDd	DOE 7	U^{235}	<0.10				S	22.9%	0.017	0.0	0.0
ESP	DOE 5	U^{235}	0.17		20	430	B	11.4%	0.047	1.5	288

Table B-19.
Radionuclide Data (Continued)

Control Device	Control Device	Isotop	Coal pCi/g	Concentration		Specific Activity pCi/gram	Coal		PM g/Nm^3	Coal Ash pCi/g	Enrich. Ratio
				µg/Nm^3	pCi/Nm^3		Rank	Ash %			
ESP	Site 116	U^{235}	<0.18		9.4	<170	B	12.0%	0.0560	0.0	0.0
FGDd	Site 116 SNRB	U^{235}	<0.18		13	<420	B	12.0%	0.0310	0.0	0.0
ESP	DOE 2	U^{235}	<0.22		1,010	<40,000	B	12.0%	0.025	0.0	0.0
FF	DOE 2 FF	U^{235}	<22		110	<7,900	B	12.0%	0.014	0.0	0.0
FF	Site 115	U^{238}	0.10		0.34	<140	S	11.2%	0.0025	0.9	0.0
ESP	UARG 700	U^{238}	0.15			3.0	S	10%	0.059	1.5	2.0
ESP	*UARG 500*	U^{238}	*0.16*			*7.0*	S	*20%*	*0.0025*	*0.8*	*8.9*
ESP	UARG 800	U^{238}	0.16			3.3	B	10%	0.18	1.6	2.1
FGDw	UARG 900	U^{238}	0.16			4.1	S	20%	0.19	0.8	5.1
FF	DOE 8	U^{238}	0.36	<0.35		<10	S	11.2%	0.011	3.2	<3
FGDd	DOE 7	U^{238}	<0.40				S	22.9%	0.017	0.0	0.0
ESP	UARG 600	U^{238}	0.47			4.4	B	10%	0.11	4.7	0.9
ESP	UARG 400	U^{238}	0.51			7.6	S	20%	0.076	2.6	3.0
ESP	UARG 100	U^{238}	0.57			7.2	B	10%	0.15	5.7	1.3
ESP	Site 116	U^{238}	<0.57		6.5	120	B	12.0%	0.0560	0.0	0.0
FGDd	Site 116 SNRB	U^{238}	<0.57		3.6	120	B	12.0%	0.0310	0.0	0.0
ESP	UARG 300	U^{238}	0.80			9.2	B	10%	0.21	8.0	1.2
ESP	DOE 3	U^{238}	1.0	5.4		12	B	12.0%	0.15	8.6	1.3

Table B-19.
Radionuclide Data (Continued)

Control Device	Control Device	Isotop	Coal pCi/g	Concentration		Specific Activity pCi/gram	Coal		PM g/Nm^3	Coal Ash pCi/g	Enrich. Ratio
				µg/Nm^3	pCi/Nm^3		Rank	Ash %			
ESP	DOE 5	U^{238}	4.0		230	4,900	B	11.4%	0.047	35.1	140
Fuel Oil											
ESP	Site 118	Pb^{210}	0.033			0	Resid	0.06%	0.0062	55.0	0.0
ESP	Site 119	Pb^{210}	<0.1		1.5	14	Resid	0.11%	0.105	0.0	0.0
ESP	Site 119	Po^{210}	0.13			0	Resid	0.11%	0.105	118	0.0
ESP	Site 118	Po^{210}	<0.14		0.07	11	Resid	0.06%	0.0062	0.0	0.0
ESP	Site 118	Ra^{226}	0.01		0.07	11	Resid	0.06%	0.0062	16.7	0.7
ESP	Site 119	Ra^{226}	<0.1		0.082	0.78	Resid	0.11%	0.105	0.0	0.0
ESP	Site 119	Ra^{228}	0.01		3.7	<35	Resid	0.11%	0.105	9.1	0.0
ESP	Site 118	Ra^{228}	0.03		0.86	140	Resid	0.06%	0.0062	50.0	2.8
ESP	Site 118	Th^{228}	0.02		0.26	42	Resid	0.06%	0.0062	33.3	1.3
ESP	Site 119	Th^{228}	0.03		1.2	11	Resid	0.11%	0.105	27.3	0.4
ESP	Site 118	Th^{230}	0.012		0.3	48	Resid	0.06%	0.0062	20.0	2.4
ESP	Site 119	Th^{230}	0.02		0.32	3.0	Resid	0.11%	0.105	18.2	0.2
ESP	Site 118	Th^{232}	<0.004		0.1	16	Resid	0.06%	0.0062	0.0	0.0
ESP	Site 119	Th^{232}	<0.01		0.074	0.70	Resid	0.11%	0.105	0.0	0.0
ESP	Site 118	U^{234}	<0.006		0.054	<8.7	Resid	0.06%	0.0062	0.0	0.0
ESP	Site 119	U^{234}	<0.01		0.18	1.7	Resid	0.11%	0.105	0.0	0.0

Table B-19.
Radionuclide Data (Continued)

Control Device	Control Device	Isotop	Coal pCi/g	Concentration		Specific Activity pCi/gram	Coal		PM g/Nm^3	Coal Ash pCi/g	Enrich. Ratio
				µg/Nm^3	pCi/Nm^3		Rank	Ash %			
ESP	Site 118	U^{235}	<0.003		0.054	<8.7	Resid	0.06%	0.0062	0.0	0.0
ESP	Site 119	U^{235}	<0.01		0.05	<0.48	Resid	0.11%	0.105	0.0	0.0
ESP	Site 118	U^{238}	<0.007		0.054	<8.7	Resid	0.06%	0.0062	0.0	0.0
ESP	Site 119	U^{238}	<0.01		0.19	1.8	Resid	0.11%	0.105	0.0	0.0
Shaded data not included in statistical calculations.											

One method of evaluating this data is to compare the particulate matter activity to the coal (ashed basis). Ratios above about 20:1 typically indicate some type of analytical inconsistency. Figure B-35 plots the paired data sets for five of the elements of concern. An EPA document [11] has published enrichment factors for these elements as follows: Lead: 5, Polonium: 5, Radium: 1.5, Thorium: 1, and Uranium: 2.

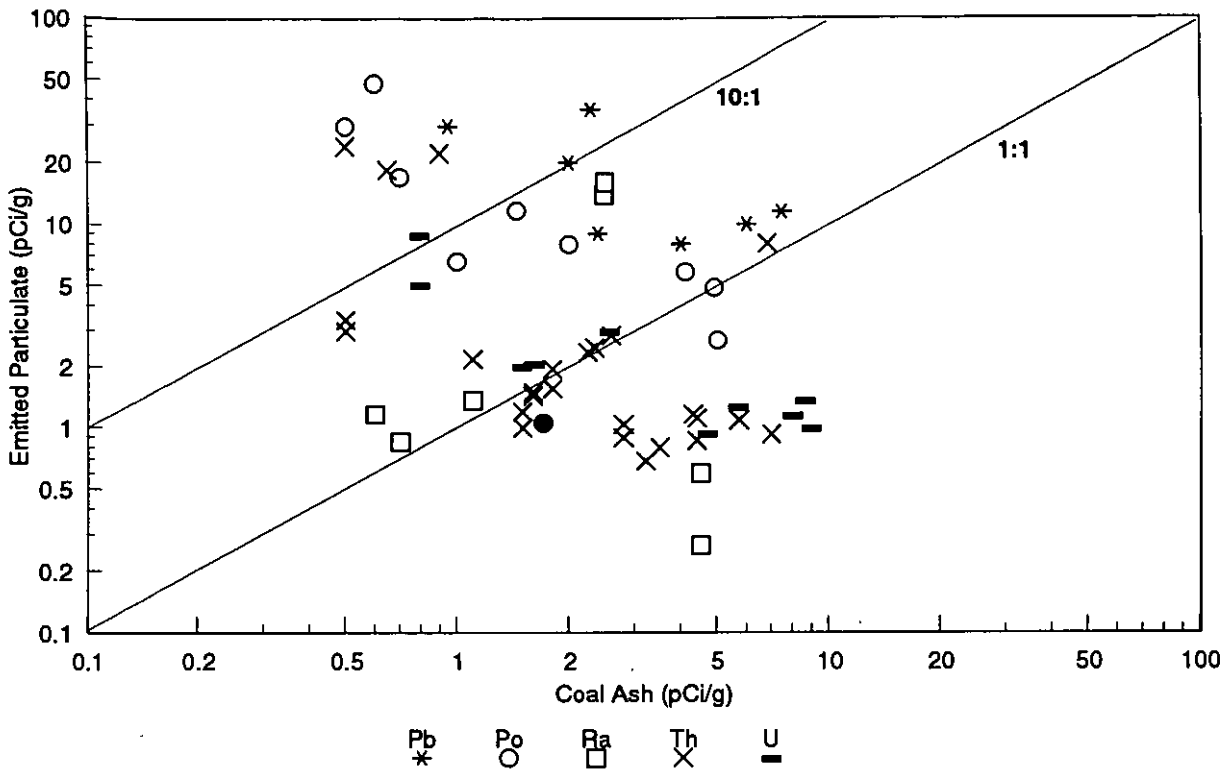


Figure B–35.
Radionuclide Enrichment

B.8 Impact on Trace Substance Emissions of Controls for Criteria Pollutants

In this section, the effects of existing criteria pollutants control (particulates, SO_2, and NO_x) on trace substance emissions will be presented. Field data obtained from the FCEM and DOE programs (as summarized in Section 3) are used to assess the amount of trace substances removed in conjunction with criteria pollutant control. The discussion is separated into specific sections for coal-, oil-, and gas-fired units.

B.8.1 Coal-Fired Boiler Controls

All modern utility boilers are regulated to varying degrees on the amount of ash, nitrogen oxide, and sulfur dioxide that can be emitted. Compliance with the emission limits can involve several strategies[1]. Ash removal from the flue gas is accomplished by three major

classes of technology. Electrostatic precipitators (ESPs) control particulates from about 90% of the coal-fired plants in the country. Fabric filters are used at 9% of the units; venturi scrubbers are used at about 1% of the plants. Most ESPs and fabric filters have particulate collection efficiencies >99% while venturi scrubbers are less efficient (<98%). Moderate levels of NO_x reduction (30–50%) are typically met by "low-NO_x" burner designs which stage the combustion process to minimize NO_x formation. More stringent reductions are achievable through the use of selective NO_x reduction techniques, either with or without catalysts. Sulfur dioxide limits are met by either combusting low-sulfur fuels or scrubbing the flue gas. Coal sulfur levels across the U.S. vary by about ten-fold in nominal sulfur content (0.35 to 3.5 percent); scrubbers can be designed for greater than 90% SO_2 reduction. About twenty percent of the coal-fired units use flue gas desulfurization systems, the remainder burn compliance coal. In addition, about 75% of the coal burned in the U.S. is subjected to some form of physical cleaning, which reduces sulfur and trace metal levels.

Based on the trace substance field data obtained to date, it is possible to classify trace substance emissions according to four different criteria pollutant control options (for boilers greater than 25 MWe):

- Units with ESPs;
- Units with ESPs and FGD systems;
- Units with fabric filters; and
- Units with fabric filters and FGD systems.

There are insufficient data to subdivide the information further to assess the effect of low-NO_x burners or selective catalytic and noncatalytic reduction of NO_x on trace substance emissions separately.

Figure B-36 presents the range of particulate control performance (expressed as mass removal efficiency ranges) for the four major technology groups represented in the FCEM data. The removal efficiency is an overall efficiency based on the input coal amount compared to the stack emissions rather than the actual device removal efficiency. The two values generally do not differ significantly except for cyclone units with a high percentage of bottom ash. Typical coal ash levels range from 5 to 10 lbs/106 Btu. Emission limits of 0.1 lb/106 Btu, therefore, equate to about 99% removal, while 0.03 lb/106 Btu requires greater than 99.5% removal. Using the recent field data, the ESP particulate mass control efficiency is classified into four removal ranges. All four tests sites with fabric filters are in the highest removal range. The one lower removal point for ESP/FGD systems is at a site where a venturi scrubber performs the majority of the particulate removal. Most of the tested sites have >99% particulate control.

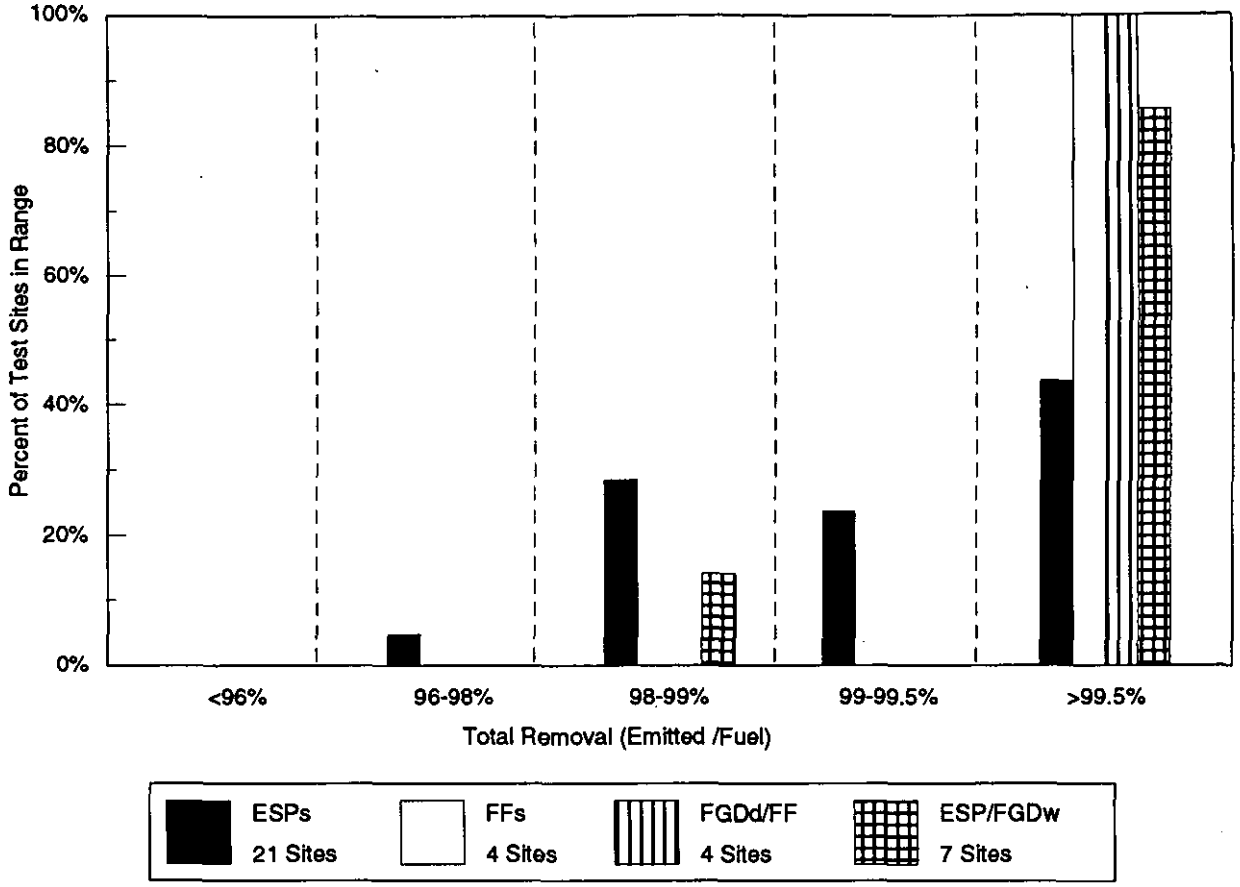


Figure B-36.
Particulate Removal Performance, by Control Technology (PISCES Measurement Sites)

Figures B-37 and B-38 present removal efficiencies for arsenic and chromium. Both of these figures show that the removal efficiency for arsenic and chromium are lower compared to overall particulate matter in Figure 9-1. Since most control systems have the poorest performance on the finest particle sizes, this lower removal efficiency is most likely due to the relative enrichment of fine particles of ash with respect to more volatile trace elements. However, the removal seen for these two metals is still quite high for all of the sites tested (90–99+%). Other metals such as lead, nickel, manganese, cadmium, beryllium, and cobalt behave similarly.

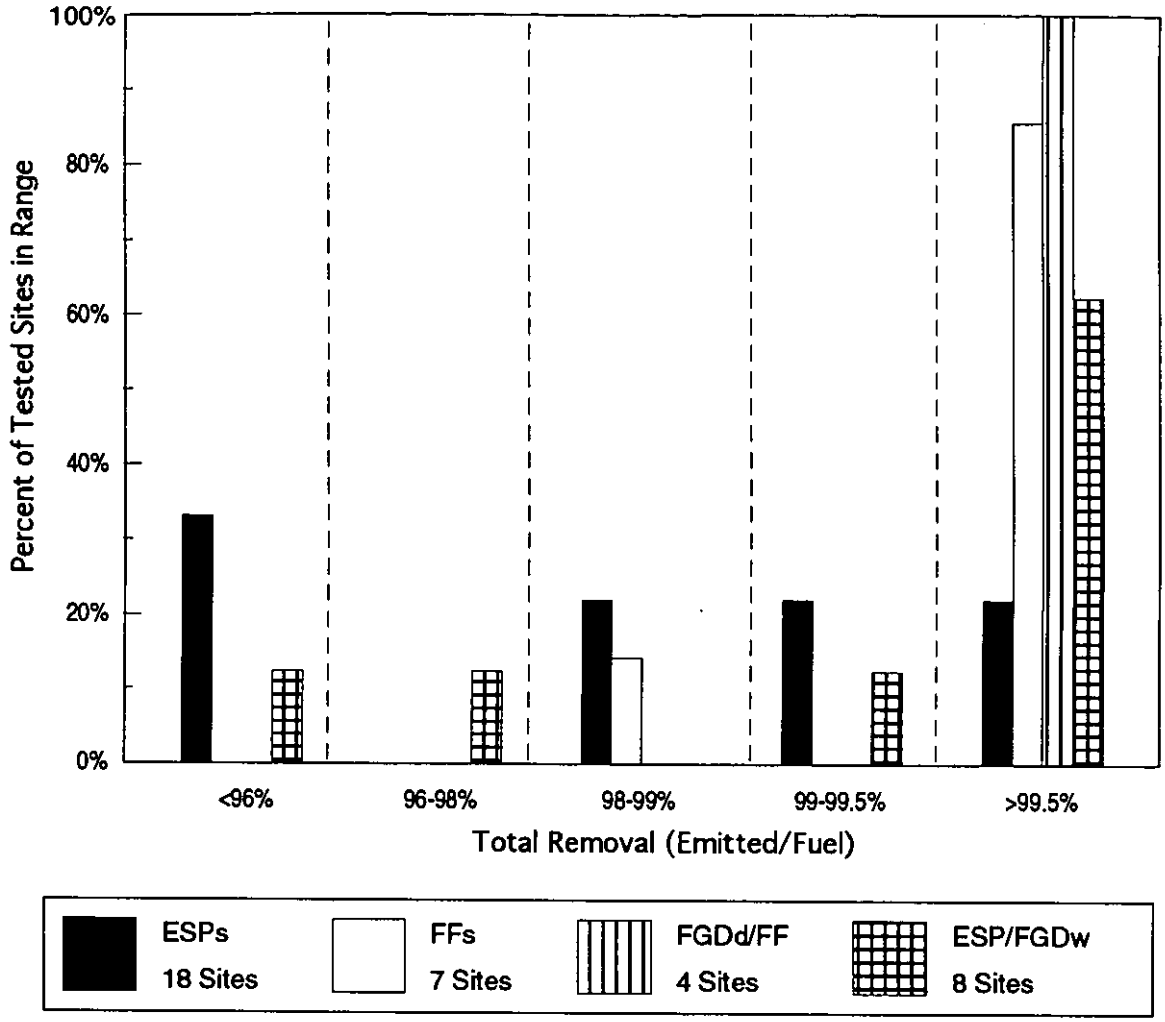


Figure B-37.
Arsenic Removal, by Control Technology, PISCES Measurement Sites

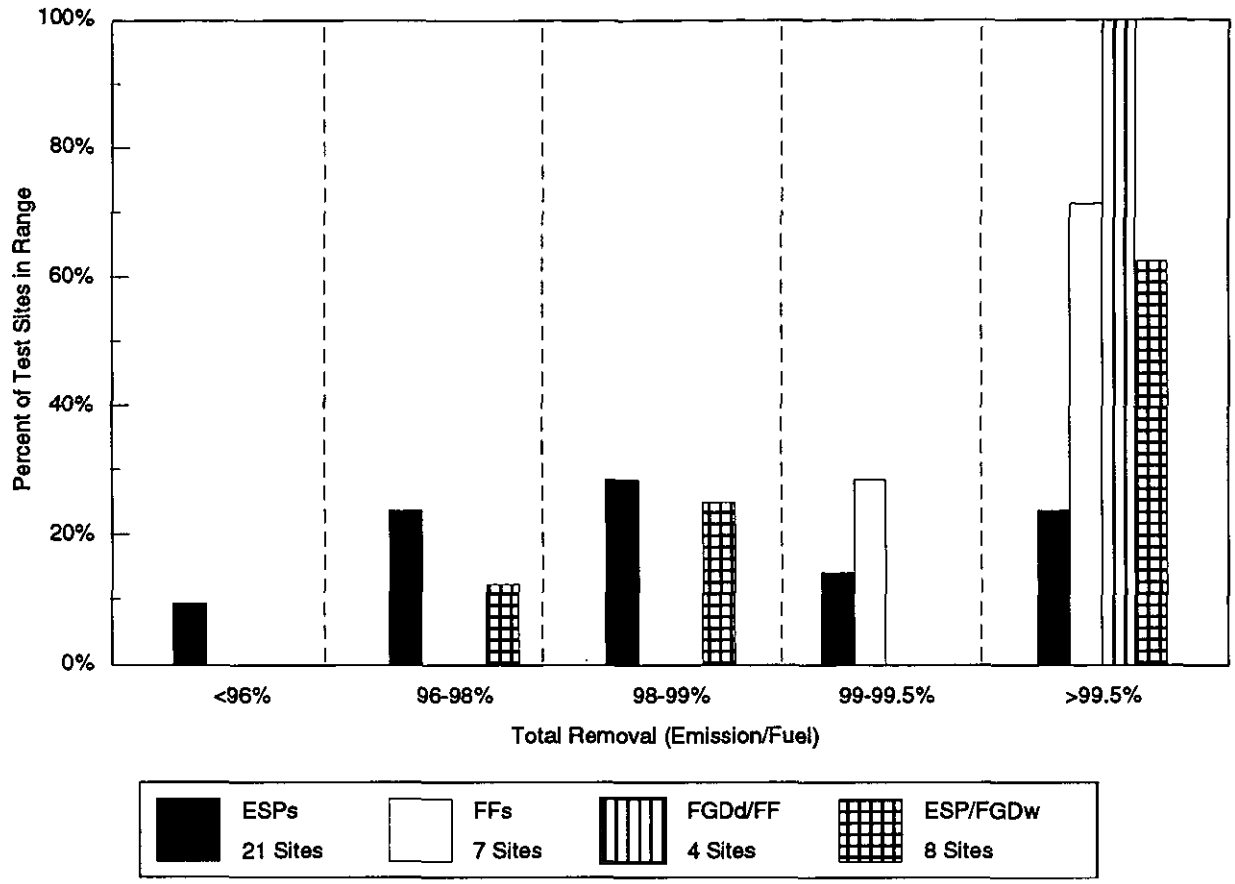


Figure B-38.
Chromium Removal, by Control Technology, PISCES Measurement Sites

Figures B-39, B-40, and B-41 show the removal efficiencies for mercury, selenium, and HCl. The removal ranges have been greatly expanded to demonstrate the wide variability of different substance/control technology combinations. Figure B-39 shows that mercury removal levels vary considerably. The sources of this variability are under study and are discussed further in this chapter. Figure B-40 shows a bimodal distribution for selenium control by ESPs. As discussed in the prior chapter, sub-bituminous coal, with highly alkaline ash, is very effective at reducing selenium in either ESPs or fabric filters. HCl is not controlled by ESPs, unless alkaline ash is present, but is easily removed by FGD systems.

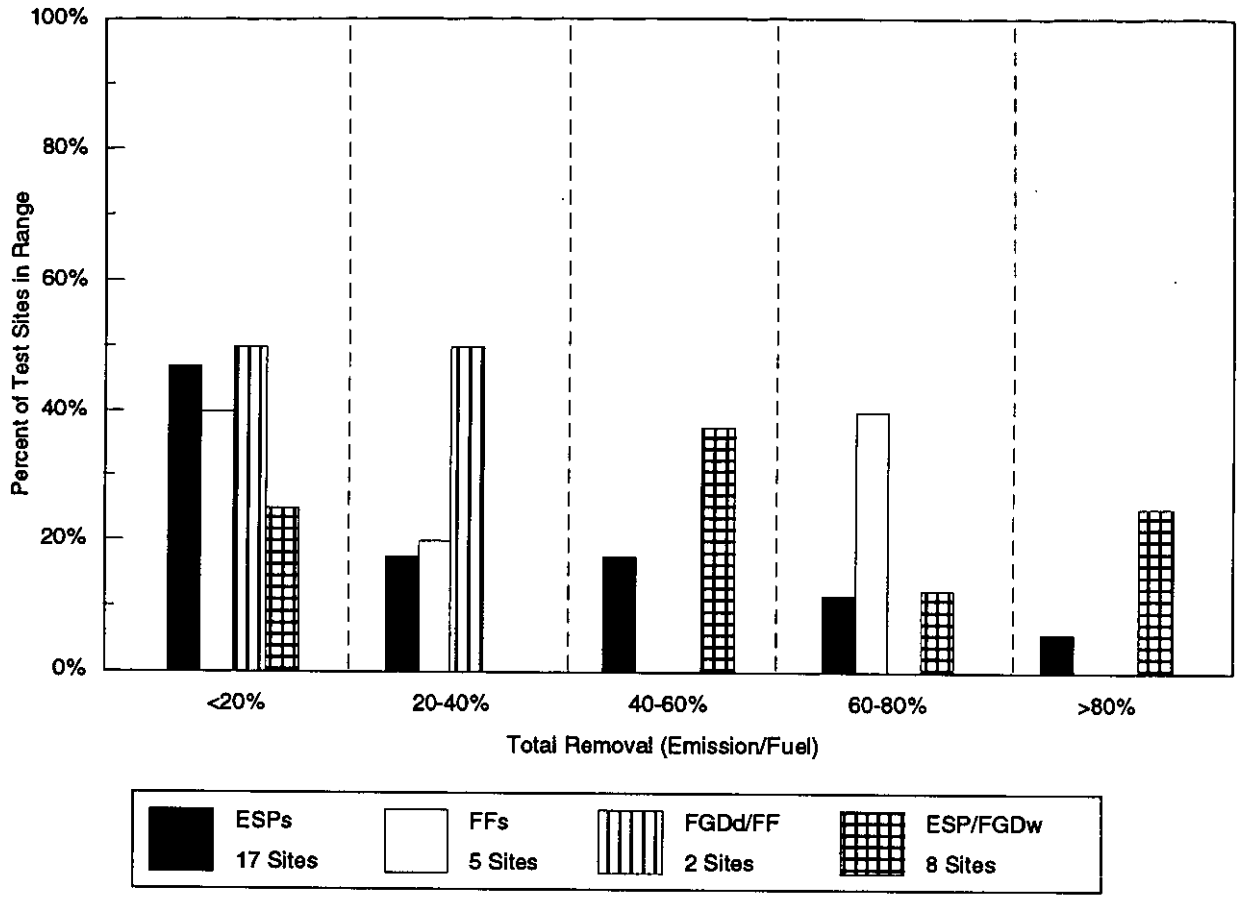


Figure B-39.
Mercury Removal, by Control Technology, PISCES Measurement Sites

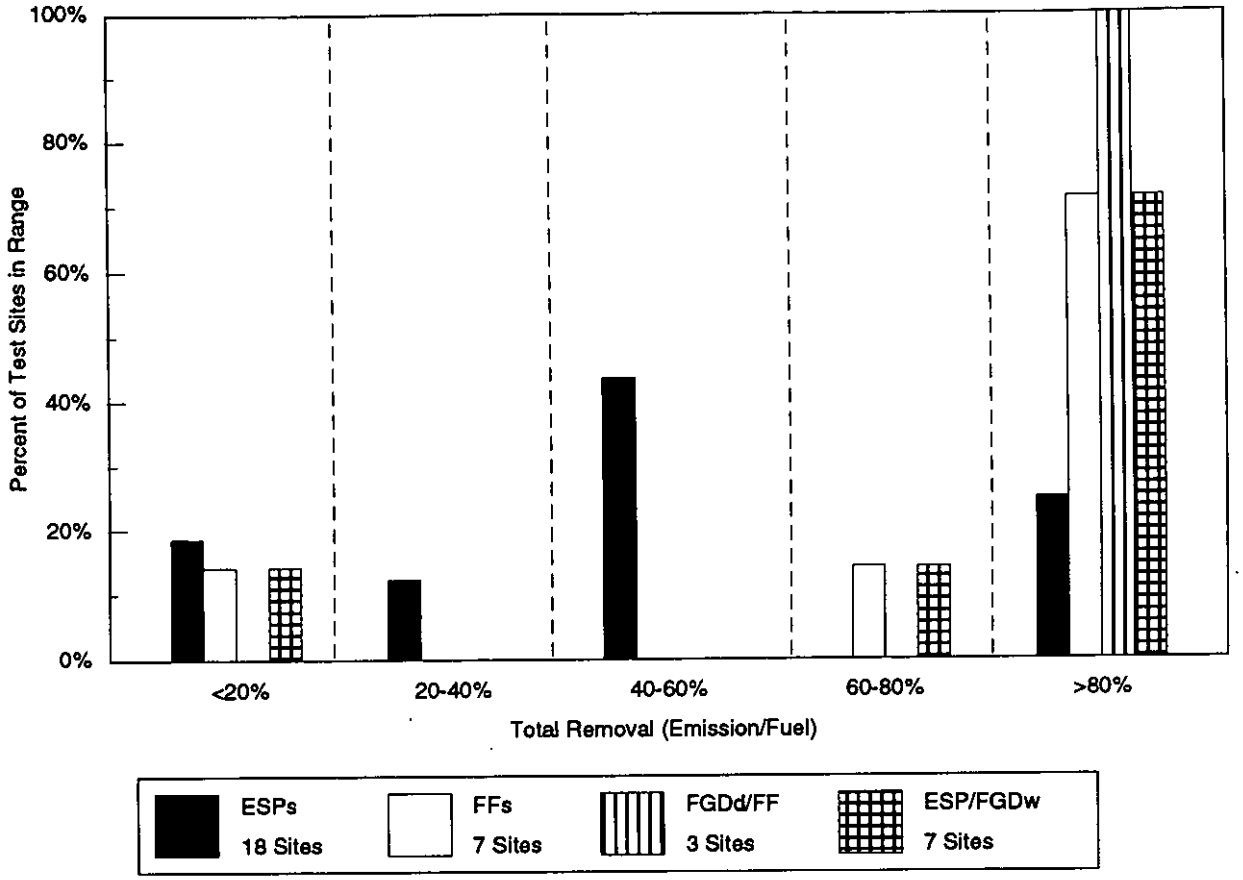


Figure B-40.
Selenium Removal, by Control Technology, PISCES Measurement Sites

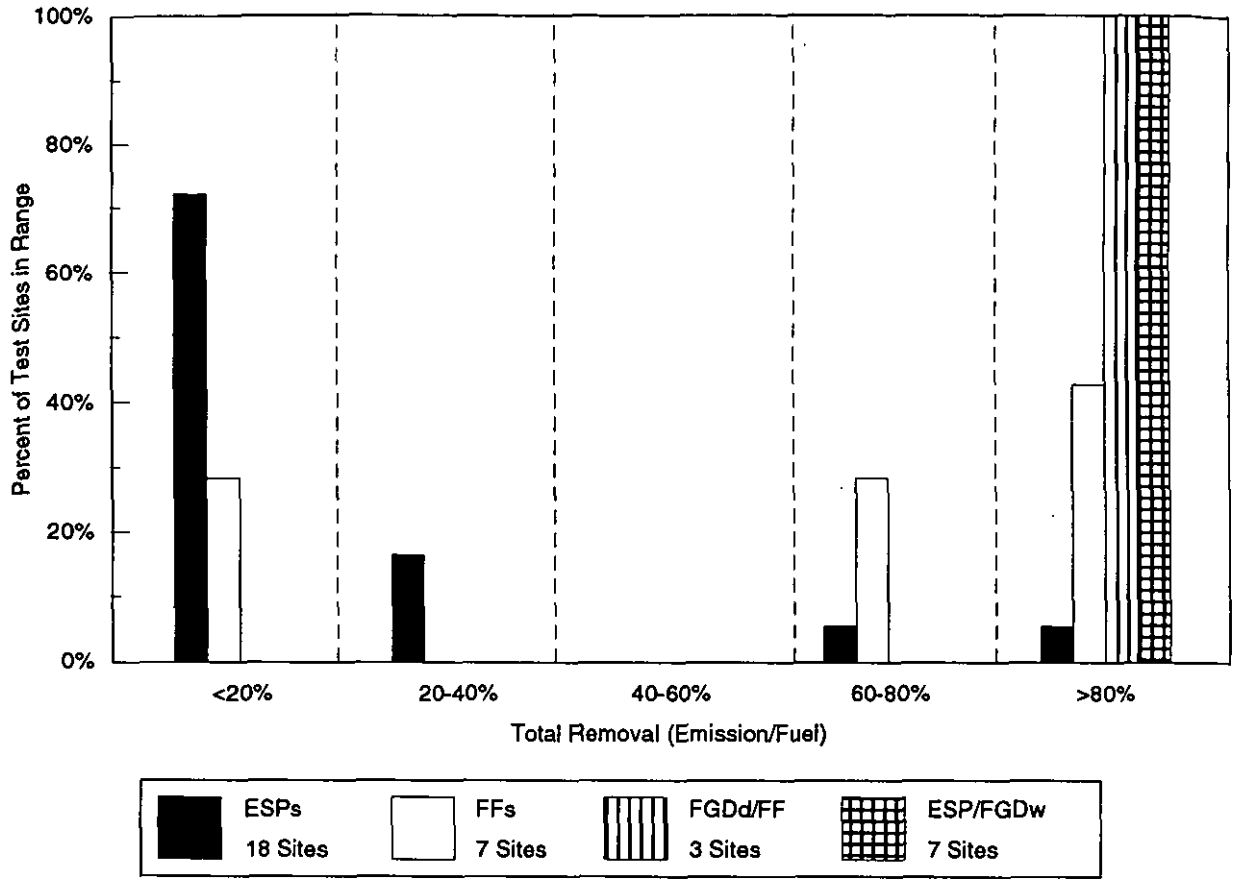


Figure B-41.
HCl Removal, by Control Technology, PISCES Measurement Sites

B.8.2 Oil-Fired Boiler Controls

Of the approximately 250 oil-fired units in the U.S., about 20% have mechanical particulate collectors and another 20% have ESPs. In many cases, particulate controls are not needed since uncontrolled emissions from oil units are low (Chapter 3). Some form of control is sometimes used to reduce opacity. Many of the units with ESPs are older plants that initially burned coal. In these plants, the ESP performance is often less than optimal and the particulate removal efficiencies vary widely. Various NO_x controls are used on oil-fired boilers; however, there are insufficient data to determine if these affect trace substance emissions. No commercial oil units use FGD systems; however, low sulfur oils are required in some metropolitan areas.

Figure B-42 presents the range of particulate removal efficiencies from oil-fired units with ESPs. Because of limited recent field data, the information includes other literature information. The performance measured at the three FCEM field sites with ESPs are also shown for comparison.

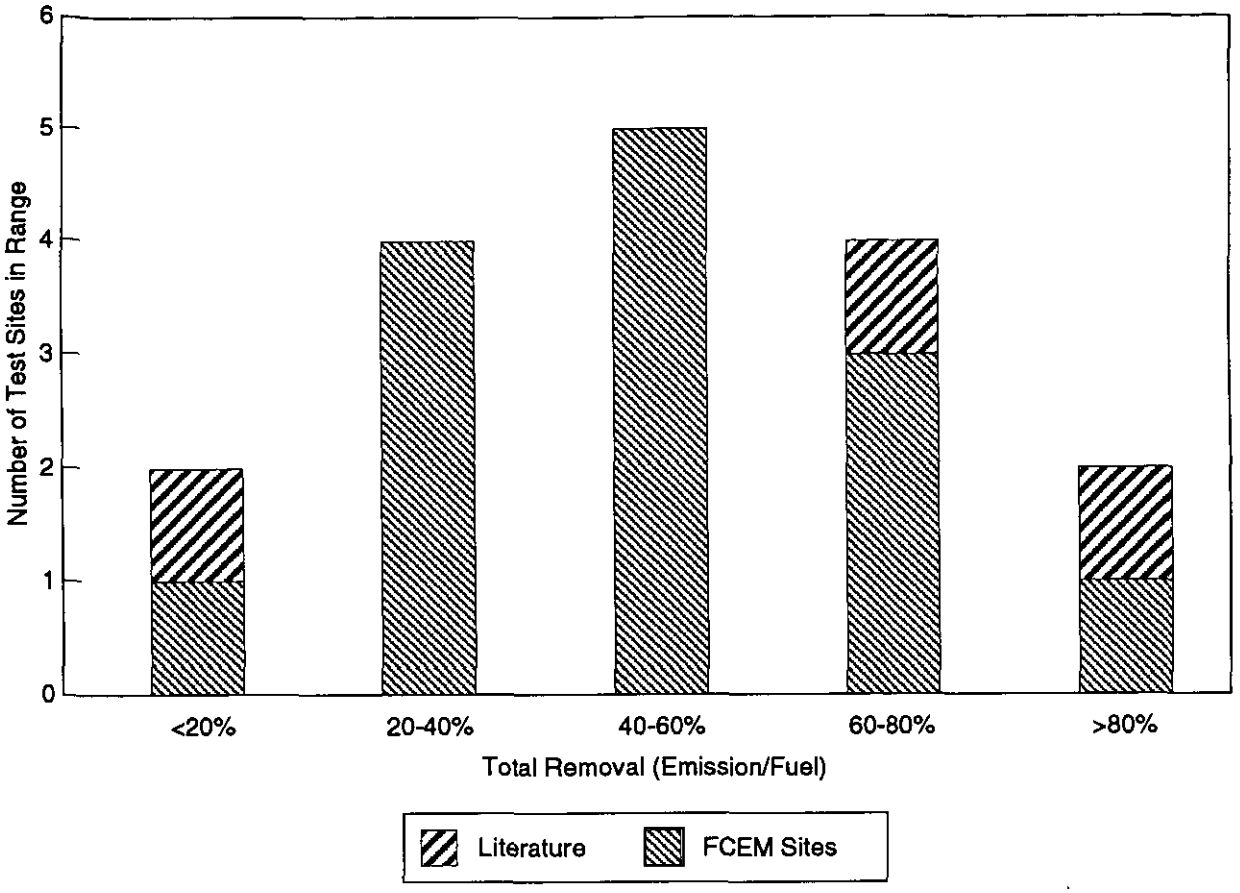


Figure B-42.
Particulate Removal Efficiencies, Oil Units with ESPs

Like the coal sites, the particulate removal efficiency measured at an oil-fired site is higher than the removal efficiency for trace metals. The average removal efficiencies for the three FCEM ESP sites for the nine metals of interest (As, Be, Cd, Cr, Co, Pb, Mn, Ni, and Sb) is about 40 percent.

B.8.3 Gas-Fired Boiler Controls

Only NO_x is normally controlled at gas-fired power plants. Of the 400+ units which use gas as the primary fuel, only 20 have some form of particulate control, most of which are mechanical collectors. The field data on gas plants confirm that trace substance emissions are lower than for any other fuel.

B.9 References

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5. *Federal Register* at 51672, Col. 1 (December 5, 1989).
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7. R.L. Roberson and T.E. Eggleston. *Characterization of Radionuclide Emissions from Coal-Fired Utility Boilers.* Report No. 83-180-06F. Prepared for the Utility Air Regulatory Group by KEA (July 1983).
8. U.S. Environmental Protection Agency. *Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual.* Interim final, OSWER Directive 9285.7-01a (September 29, 1989).
9. R.O. Gilbert. *Statistical Methods for Environmental Pollution Monitoring,* Van Nostrand Reinhold Co. Inc. (1987).
10. R.L. Roberson and T.E. Eggleston. *Characterization of Radionuclide Emissions from Coal-Fired Utility Boilers,* Report No. 83-180-06F, prepared for the Utility Air Regulatory Group by KEA (July 1983).
11. U.S. Environmental Protection Agency, Office of Radiation Programs. *Risk Assessments - Environmental Impact Statements for NESHAPS Radionuclides,* Volume 2, Background Information Document, EPA 520/1-89-006-1, Washington D.C. (September 1989).

APPENDIX C
COAL CHARACTERISTICS AND PARTICULATE EMISSIONS

C.1 Assigning Coal Characteristics to Units

The concentrations of inorganic substances in coal are a key input to the emissions estimation procedure in Section 4.3. As discussed in Section 4.2, EPRI developed a set of concentration data intended to be more representative of "as-fired" coal and a data set of mercury concentrations in delivered coal. Unfortunately, these coal samples were not directly linked to the base case scenario (Section 4.1.4). This section discusses the assignments of available coal concentration data to regions anticipated as coal supply regions in the year 2010.

Table C-1 shows the number of samples available for each coal basin and region from a subset of the USGS COALQUAL database. This subset represents data on the top 50 producing seams (as of 1991). It was adjusted to be more representative of "as-fired" coal quality. Sufficient data are available from COALQUAL for 17 of the 32 coal-origin regions in the base case industry scenario. For the remaining 15 regions anticipated to supply coal to utilities in the year 2010, analyses of surrogate coals are required. Finkelman (1994) suggested surrogates based on proximity and rank. For example, a composite of Powder River coals from North Dakota and Wyoming was used as a surrogate for Central West Basin coals. In this case, the concentration and heat content data are from different sources. The average of the mean concentrations across North Dakota and Wyoming coals was used along with the average heat content across the three Missouri coal samples in the "as-fired" database. In all other cases, the concentration and heat content data are from the same surrogate coal. Of the 15 coals requiring surrogates, 10 are burned by 10 units or fewer in the base case scenario.

Table C-1.
Coals in the EPRI Coal Database and Base Case Scenario

Basin	Coal Supply Region	Rank	No. of Samples	No. of Units Operating in 2010	Surrogate
Northern Appalachian	Pennsylvania (PN)	Bit	539	0	
	Pennsylvania, central (PC)		0	47	PN
	Pennsylvania, west (PW)		0	36	PN
	Ohio (OH)		492	30	
	Maryland (MD)		38	1	
	West Virginia (WN)		101	38	
Midwest	Illinois (IL)	Bit	15	54	
	Indiana (IN)		80	24	
	Kentucky, west (KW)		116	47	
Central West	Iowa (IA)	Bit	0	2	avg(MP,WP)
	Missouri (MO)		4	3	avg(MP,WP)
	Kansas (KS)		0	2	avg(MP,WP)
Gulf	Texas (TX)	Lig	54	16	
	Louisiana (LA)		0	1	TX
Central Appalachian	West Virginia, south (WS)	Bit	280	133	
	Virginia (VA)		52	82	
	Kentucky, east (KE)		337	232	
	Tennessee (TN)		12	8	
Southern Appalachian	Alabama (AL)	Bit	150	34	
Eastern Northern Great Plains	Montana, Powder River (MP)	Sub	95	57	
	North Dakota (ND)	Lig	56	13	
	Wyoming, Powder River (WP)	Sub	141	220	

Table C-1.
Coals in the EPRI Coal Database and Base Case Scenario (Continued)

Basin	Coal Supply Region	Rank	No. of Samples	No. of Units Operating in 2010	Surrogate
Rockies	Wyoming (WY)	Bit	0	7	avg(Rockies)
	Wyoming, Green River (WG)	Bit	0	9	avg(Rockies)
	Wyoming, Green River (WG)	Sub	2	10	avg(Rockies)
	Colorado, Green River (CG)	Bit	26	18	
	Colorado, Green River (CG)	Sub	1	0	
	Colorado, Raton (CR)	Bit	0	3	NS sub
	Colorado, Uinta (CU)	Bit	0	39	avg(Rockies)
	Colorado, Uinta (CU)	Sub	0	18	avg(Rockies)
	Utah, Uinta (UU)	Bit	22	0	
	Utah, central (UC)	Bit	0	17	avg(Rockies)
Southwest	New Mexico, San Juan (NS)	Bit	3	10	NS sub
	New Mexico, San Juan (NS)	Sub	104	12	
	Arizona (AZ)	Bit	0	5	NS sub

Table C-2 shows the number of coal samples available for each basin and region under the coal mercury sampling and analysis program. In this case, 22 of the 32 regions specified in the base case 2010 scenario have coal data available for characterization of mercury content. The remaining 10 require use of a surrogate coal. As above, surrogates were assigned based on proximity and rank. Of the 10 regions requiring surrogates, 8 are burned by 10 units or fewer in the base case scenario.

Table C-2.
Delivered Coals in the Base Case Scenario with Measured Mercury Values

Basin	Coal Supply Region	Rank	No. of Samples	No. of Units Operating in 2010	Surrogate	Hg Conc. (ppm)
Northern Appalachian	Pennsylvania, Central (PC)	Bit	4	46		0.250
	Pennsylvania, West (PW)		10	36		0.122
	Ohio (OH)		4	36		0.113
	Maryland (MD)		0	1	avg(PC,PW,WN, VA)	0.145
	West Virginia (WN)		10	41		0.093
Midwest	Illinois (IL)	Bit	11	56		0.064
	Indiana (IN)		4	35		0.107
	Kentucky, West (KW)		2	58		0.077
Central West	Iowa (IA)	Bit	0	2	avg(MP, WP)	0.054
	Missouri (MO)		0	3	avg(MP, WP)	0.054
	Kansas (KS)		0	3	avg(MP, WP)	0.054
Gulf	Texas (TX)	Lig	8	18		0.164
	Louisiana (LA)		0	1	TX	0.164
Central Appalachian	West Virginia, South (WS)	Bit	7	137		0.069
	Virginia (VA)		6	83		0.081
	Kentucky, East (KE)		15	235		0.110
	Tennessee (TN)		2	8		0.088
Southern Appalachian	Alabama (AL)	Bit	9	34		0.071
Eastern Northern Great Plains	Montana, Powder River (MP)	Sub	3	75		0.038
	North Dakota (ND)	Lig	3	14		0.065
	Wyoming, Powder River (WP)	Sub	19	230		0.070

Table C-2.
Delivered Coals in the Base Case Scenario with Measured Mercury Values (Continued)

Basin	Coal Supply Region	Rank	No. of Samples	No. of Units Operating in 2010	Surrogate	Hg Conc. (ppm)
Rockies	Wyoming (WY)	Bit	0	7	avg (Rockies)	0.033
	Wyoming, Green River (WG)	Bit	0	9	avg (Rockies)	0.033
	Wyoming, Green River (WG)	Sub	3	10		0.027
	Colorado, Green River (CG)	Bit	0	19	avg (Rockies)	0.033
	Colorado, Green River (CG)	Sub	1	0		0.067
	Colorado, Raton (CR)	Bit	0	3	avg (Rockies)	0.033
	Colorado, Uinta (CU)	Bit	2	41		0.018
	Colorado, Uinta (CU)	Sub	0	18	avg (Rockies)	0.033
	Utah, Central (UC)	Bit	7	21		0.022
Southwest	New Mexico, San Juan (NS)	Bit	10	10		0.049
	New Mexico, San Juan (NS)	Sub	5	15		0.036
	Arizona (AZ)	Bit	2	5		0.029

Table C-2 also shows the mercury concentrations from the mercury sampling project. Central Pennsylvania bituminous and Texas lignite coal samples have the highest mercury concentrations at 0.25 and 0.22 ppm, respectively. Samples from the Rocky Mountain region and the Southwest have the lowest mercury concentrations, ranging from 0.018 to 0.049 ppm.

C.2 Calculating Particulate Emissions

Unit-specific particulate emissions are a key input for estimating emissions of inorganic substances from unmeasured plants. Although particulate emission estimates for 2010 are unavailable, relevant information is available in EIA 767 forms, the Power Statistics database, and the UARG particulate survey. The EIA data were used because the entries are intended to represent long-term average operations. This section describes the approach utilized for estimating 2010 particulate emissions for individual units.

The approach used the best available data to calculate 2010 particulate emissions for individual units. It utilizes the set of equations shown in Table C-3, which prioritizes equations according to their expected accuracy. Given the available information, the approach calculated the unit's particulate emissions using the most desirable equation for which data are available. If the unit was projected to retrofit a wet scrubber by 2010, the calculated particulate emissions were reduced by the average wet FGD system removal percentage in the field measurements (74%). The calculated particulate emissions level was then compared to the total suspended particulate (TSP) emissions limit, and the lower value was used in subsequent emission calculations for inorganic substances. It was assumed that units will operate in compliance with their regulated TSP limit, and that calculated values greater than the TSP limit are a result of erroneous particulate emissions or removal efficiency data.

Table C-3.
Equations for Estimating Particulate Emissions (listed in order of use)

	Rank	Equation for 2010 Particulate Emissions
Modification	1	Pre_Mod_PM * (1 - Post_Mod_RE) / (1 - Pre_Mod_TRE)
Modification	2	Pre_Mod_PM * (1 - Post_Mod_RE) / (1 - Pre_Mod_DRE)
Modification	3	EIA_PM * (1 - Post_Mod_RE) / (1 - Pre_Mod_TRE)
Modification	4	EIA_PM * (1 - Post_Mod_RE) / (1 - Pre_Mod_DRE)
Modification	5	Ash_Input * Ash_Carryover * (1 - Post_Mod_RE)
No Modification	*1*	*UDI_Mod_PM*
No Modification	*2*	*EIA_PM*
No Modification	*3*	*Ash_Input * Ash_Carryover * (1 - Pre_Mod_TRE)*
No Modification	*4*	*Ash_Input * Ash_Carryover * (1 - Pre_Mod_DRE)*
No Modification	*5*	*Ash_Input * Ash_Carryover * (1 - UDI_DRE)*

Key for variables used in equations:
Pre_Mod_PM = pre-modification particulate emissions from particulate survey (lb/10^6Btu)
EIA_PM = particulate emissions from 1990 EIA 767 forms (lb/10^6Btu)
Post_Mod_RE = post-modification removal efficiency from particulate survey
Pre_Mod_TRE = pre-modification test removal efficiency from particulate survey
Pre_Mod_DRE = pre-modification design removal efficiency from particulate survey
UDI_DRE = design removal efficiency from the UDI Power Statistics database
Ash_Input = ash input calculated from ash and heat contents in EPRI "as-fired" database (lb/10^6Btu)
Ash_Carryover = 0.3 for cyclones boilers and 0.8 for other boilers based on field meas

The first five equations of Table C-3 (those labelled "modification" in the first column) represent post-modification particulate emissions, and were utilized in situations where modifications to particulate control equipment are planned or underway *and* sufficient data are available. The second set of five equations (those labelled "no modification") rep-

resent pre-modification particulate emissions, and were utilized when modifications are not planned or sufficient data are unavailable for the set of equations specified for plants undergoing modification.

For 13 units, the approach estimated higher post-modification particulate emissions than pre-modification particulate emissions. This occurs when the post-modification removal efficiency is lower than the pre-modification removal efficiency. Since a preliminary analysis indicated that these 13 units were not in the groups of plants with the highest inhalation MEI and population cancer risks, the approach for estimating particulate emissions was not altered to accommodate these units.

C.3 Comparison of Modeled and Measured Emission Rates for Coal-Fired Units

The data needed to model emissions using the methodology described in Section 4.3 include the trace substance concentration and ash content of the coal, and the particulate emission rate of the unit. For the industry-wide assessment, these data are obtained from databases. For selected units, however, the PISCES program has obtained specific emission measurements during coal-firing. Since the input data that the modeling methodology requires has also been measured for these selected units, it is possible to model emission rates using field data for these plants.

Table C-4 shows two different sets of modeled emissions results for arsenic, one using field measurements of arsenic concentrations in coal (along with ash content of the coal, and particulate emission rates), and the other using database values. The database values include ash content in coal from the 1990 Power Statistics database and 1991 EIA 767 Forms, particulate emissions from 1991 EIA forms and the UARG particulate survey, and trace substance concentration data from the adjusted subset of the COALQUAL database, and are intended to represent "current" (1990-era) operations. Emissions estimates for current operations provide a basis for comparison with recently-measured emissions.

Table C-4.
Comparison of Arsenic Emissions Estimates and Measured Values For PISCES Coal-Fired Sites (lb/10^{12} Btu)

Site No.	(95% C.I.)	Modeled Using Field Data Input*		Modeled Using Database Input**	
		Modeled	Modeled / Measured	Modeled	Modeled / Measured
10	< 1.0	0.5	—	16.3	—
11	1.2 (10)	0.6	0.5	11.3	9.4
12	1.9 (4.8)	2.7	1.4	9.6	5.1

Table C-4.
Comparison of Arsenic Emissions Estimates and Measured Values For PISCES Coal-Fired Sites (lb/10^{12} Btu) (Continued)

Site No.	(95% C.I.)	Modeled Using Field Data Input*		Modeled Using Database Input**	
		Modeled	Modeled / Measured	Modeled	Modeled / Measured
15	13 (5.3)	7.6	0.6	44.2	3.4
18	36 (12)	23.4	0.6	15.6	0.4
19	7.9 (2.9)	*6.2*	0.8	22.4	2.8
101	—			0.7	—
102	2.9 (1.3)			12.5	4.3
110	1.7 (1.1)	*2.0*	1.2	7.5	4.4
114	7.6 (13)	*9.0*	1.2	*4.2*	0.5
115	0.8 (1.4)	*0.1*	0.1	*0.7*	0.8
min		0.1	0.1	0.7	0.4
max		23.4	1.4	44.2	9.4
median		2.7	0.7	11.3	3.4

Italic signifies that the emissions estimate is within the 95% confidence interval of the measurements.
All modeled emissions use substance-specific correlation parameters.
* Modeled emissions (with field data input) are based on ash content and particulate emissions, and trace substance concentrations from field measurements.
** Modeled emissions (with database input) are based on ash content and particulate emissions from the 1990 Power Statistics database and 1991 EIA 767 Forms; trace substance concentrations found in the literature.

Both sets of modeled emissions results reflect uncertainty in the modeling method. Results using field data reflect uncertainty in measurements of the emissions values, and the results using database values reflect the extent to which the database values are representative of operating conditions when the unit was tested.

Using field data in the emission modeling process yielded relatively accurate emission estimates (when compared against measured emissions) for the period during which field tests were conducted; modeled emissions were usually within the 95% confidence interval for all eight of the detected emissions measurements. This suggests that the emissions parameterization method (described in Section 3) is a relatively good predictive tool for estimating emissions from individual utility plants.

Using database values in the emissions modeling process reduced the accuracy of the predictions of emissions for the period during which field measurements were conducted; modeled arsenic emissions were within the 95% confidence interval for only two of the

nine corresponding arsenic measurements above detection limits. Modeled emissions using database values were higher than the upper bound of the 95% confidence interval for seven of the nine corresponding detected measurements.

When using database values, most of the discrepancy between measured and modeled emissions is attributable to the difference between database values and values measured during the emissions field test period. During these field tests, measurements were conducted of arsenic concentrations in the coal and in particulate emissions. When the emissions parameterization equations of Section 3 were applied, it was found that the differences between database and measured values for ash content had negligible impact on the resulting arsenic emission rates. When considering arsenic concentrations in the coal, nine of the ten database values were larger than the corresponding field-measured values, and the mean database value was 85% larger than the mean measured value. For particulate emissions, eight of the ten database values were larger (that is, resulted in higher calculated emissions of arsenic) than the measured values, and the mean database value was 40% larger (producing higher arsenic emissions) than the mean measured value. For ash content, nine of the ten database values were smaller than the measured values, and the mean database value was 15% smaller (resulting in higher calculated emissions) than the mean measured value.

In general, modeling emissions using field data yields more accurate estimates for the period in which field measurements were conducted than modeling emissions with database values that may not be representative of that time period. The median estimate using field data is 70% of measured emissions, while the median estimate using database values is 340% of measured emissions. Using database values, the modeled emissions ranged from 40% to 940% of the corresponding measured values. The modeled emissions using database values reflect database coal concentration and particulate emission values that are not necessarily representative of generating unit operations during the period when the field tests were conducted.

Table C-5 presents the same comparison for measured and modeled emissions of chromium. Modeling emissions with field data yields chromium estimates that are relatively close to measured emissions concentrations during the field testing period. The median emissions using field data is 80% of the measured emissions, which is almost identical to the ratio for arsenic. The modeled chromium emissions using database input are closer to the measured chromium emissions than the corresponding modeled and measured arsenic emissions are to one another. For chromium, the median estimate is 140% of the measured value.

Table C-5.
Comparison of Chromium Emissions Estimates and Measured Values for PISCES Coal-Fired Sites (lb/10^{12} Btu)

PISCES Site No.	Measured (95% C.I.)	Modeled Using Field Data Input*		Modeled Using Database Input**	
		Modeled	Modeled / Measured	Modeled	Modeled / Measured
10	1.6	1.7	1.1	14.7	9.2
11	8 (90)	*2.0*	0.2	*11.0*	1.4
12	9 (80)	*6.1*	0.7	*9.9*	1.1
15	12 (15)	10.2	0.8	*17.3*	1.4
18	25 (2.8)	19.3	0.8	11.9	0.5
19	13 (5)	*10.4*	0.8	*13.0*	1.0
101	1.8			2.6	1.5
102	8.5 (3.5)	*8.2*	1.0	*11.8*	1.4
110	17 (13)	*5.0*	0.3	*13.6*	0.8
114	14 (13)	*7.3*	0.5	*2.8*	0.2
115	0.7 (0.5)	*0.7*	1.0	2.5	3.5
min		0.7	0.2	2.5	0.2
max		19.3	1.1	17.3	9.2
median		6.7	0.8	11.8	1.4

Italic signifies that the emissions estimate is within the 95% confidence interval of the measurements.
All modeled emissions use substance-specific correlation parameters.
* Modeled emissions (with field data input) are based on ash content and particulate emissions, and trace substance concentrations from field measurements.
** Modeled emissions (with database input) are based on ash content and particulate emissions from the 1990 Power Statistics database and 1991 EIA 767 Forms; trace substance concentrations found in the literature.

Based on these comparisons, it appears that the modeling method in Section 3 is appropriate for use in this generic industrywide risk analysis. It also appears that emissions estimates based on field data input are better suited for estimating emissions representative of the period that field tests were conducted than are industrywide data sets. Field data, however, are based on a very short series of measurements (generally fewer than 10 samples over 3 to 4 days), so it is not clear to what extent they are representative of long term operations. The industry-wide assessment relied on database values, since present-day measured values may be inapplicable to future long-term operation of units. The

accuracy of these emission estimates, for year 2010 operations, that are used in the risk assessment will depend upon the extent to which database values are representative of future operations.

C.4 Alternative Fossil Plant and Coal Quality Scenarios for Trace Substances Emissions Assessment

C.4.1 Overview: Purpose of Alternative Scenarios

This section describes alternative scenarios of fossil plant operations developed for the emissions estimation step of the utility trace substances study. The 1990 Clean Air Act Amendments require fossil plants to meet substantial new emission constraints between 1995 and 2010. For many plants this will result in accelerated retirement, altered levels of utilization, fuel switching, and/or emission control retrofits, all of which affect emissions of trace substances. The base case and alternative scenarios were employed to provide independent perspectives on future fossil plant operations, and to explore implications of some key assumptions that varied among the different scenarios.

This section describes

- the methodology used for developing four alternative scenarios of fossil plant operations in year 2010,
- major assumptions underlying and distinguishing these scenarios,
- characterization of trace substances in coal samples, developed independently from the base case characterization,
- key scenario projection results used for estimating trace substance emissions.

C.4.2 Alternative Fossil Plant Scenarios: Methodology

The two alternative future industry scenarios described in Section 4 were selected from among four scenarios assessed to provide a range of estimates of industry configurations and operations. These four scenarios of utility fossil plant operations in the year 2010 were developed to provide projections of fossil plant additions, retirements, generation, fuel selection and consumption, and emission controls. The methodology for constructing these scenario projections is described in this section. Characterization of trace substance levels in different coals was developed for use in converting scenario results to emissions estimates, and is described in section C.4.6 below.

The four alternative scenarios extend earlier studies conducted by EPRI to analyze nationwide utility strategies for compliance with the Title IV SO_2 emission reduction requirements of the 1990 CAAA [1]. The analyses included evaluation of future markets for fuels, control technologies, and emission allowances, and showed that there is likely to be a dynamic interaction among these markets that will enhance planning flexibility

and mitigate costs. The analyses concluded that compliance strategies and markets can be strongly influenced by allowance trading behavior and constraints, as well as other uncertainties regarding, for example, fuel prices, load growth, and fossil plant retirements.

The four alternative fossil plant scenarios were developed for the trace substances assessment using the Emissions Reduction Analysis Model (ERAM) that was developed for EPRI [1]. ERAM simulates each generating unit based on a detailed characterization of its emissions, emission controls, and fuels.

Key drivers for the four scenarios were:

- forecast capacity additions,
- forecast unit by unit generation levels and retirements,
- fuel switching options and their costs at individual units, including fuel composition, transportation costs, and switching penalties,
- models and data used to project unit-specific performance and costs for emission control technology retrofits, and
- assumed constraints on between-utility trading of SO_2 emission allowances.

Unit by unit retirements, capacity additions, and generation assumptions were developed using historical capacity factors, North American Electric Reliability Council (NERC) forecasts, forecasts using the EPRI Utility Fuel Consumption Model, and other generation and capacity forecasts. Inputs that were varied among the four different alternative scenarios included retirement dates and generation levels for individual units, capacity additions, prices for certain fuels, and assumed constraints on between-utility trading of emission allowances. Inputs and calculations were independent from the methods used to develop EPRI's base case scenario.

For each alternative scenario, projection results included over 100 variables physically and economically characterizing each unit's future generation, fuel(s), emissions, and emission controls. Scenario results also included each utility system's emissions, total and marginal emission reduction costs, and emission allowance sales and purchases. The few key scenario results subsequently used in the assessment's estimation of trace substances emissions were generating unit-level fuel selection, fuel consumption, and emission controls. These control scenarios projected both particulate controls and flue gas desulfurization. This alternative assessment also used a characterization of trace substance concentrations in coal that differed from that of the base case scenario; this characterization was used to calculate trace substance emissions for each generating unit under the alternative scenarios.

Development of alternative scenarios proceeded in two steps. First, the least-cost method of meeting each of up to eleven different potential SO_2 emission levels at each unit was identified and characterized in detail after simulating numerous fuel switching, fuel blending, and control technology options. In this process, the cost and performance of several different SO_2 control technologies were estimated using parameterized models based on recent studies conducted by EPRI, taking into account a number of unit-specific parameters, including retrofit factors based on site visits. The technologies modeled included wet scrubbing, spray drying, and several sorbent injection technologies for SO_2 control, along with electrostatic precipitator and fabric filter particulate controls.

To complete the alternative scenarios of future fossil plant operations, strategies were projected for each utility system to comply with its CAAA SO_2 emission cap, based on that system's projected year 2010 allowance allocation plus any announced allowance trades. This involved selecting emission reduction options at various units in each system to minimize systemwide compliance costs, also considering allowance trading among utilities. Allowance trading can substantially affect projected fuel and emission control choices.

The alternative fossil plant scenarios included individual existing and announced utility coal-fired and residual oil-capable steam units greater than 25 MW, plus sufficient generic coal plant additions in each North American Electric Reliability Council (NERC) region to achieve capacity levels forecast for year 2010 under each scenario. Essentially no additions of utility plants burning residual oil are forecast. Which oil/gas steam units were projected to burn substantial amounts of residual oil sometimes differed between the different alternative and base case scenarios.

While included in the SO_2 strategy projections, non-utility coal plants were excluded from the alternative scenarios provided for the trace substances assessment because these plants (1) were similarly excluded from the assessment's base case scenario, (2) are generally projected to account for only about five percent of coal consumption for electric generation by year 2010, and (3) include many small projects often with uncertain prospects and design. Also, about 3.5 GW of utility coal units below 25 MW were excluded because these units are not subject to the SO_2 caps under the 1990 CAAA. About 0.3 GW of this 3.5 GW of small utility coal units are included in the assessment's base case scenario.

The alternative scenarios included steam units burning residual oil, accounting for 90 percent or more of forecast total utility oil consumption. Besides being used in lesser quantities, distillate oil contains lower levels of most trace substances. Combustion turbine and combined cycle units burning natural gas or distillate fuel were excluded from the alternative scenarios. These units were also excluded from the base case scenario and, with a few exceptions, from the recent EPRI SO_2 analyses. Natural gas burned in utility gas and dual oil/gas steam units was not incorporated into the alternative scenarios. In contrast, the base case scenario did include gas consumption in utility steam units,

accounting for 2.1 quadrillion Btus (quads) of projected gas consumption, compared to the 4-5 quads of total utility plus gas consumption by non-utility generators (roughly 75% of utility levels), forecast for year 2010 [2,3].

C.4.3 Differences Among Alternative Fossil Plant Scenarios

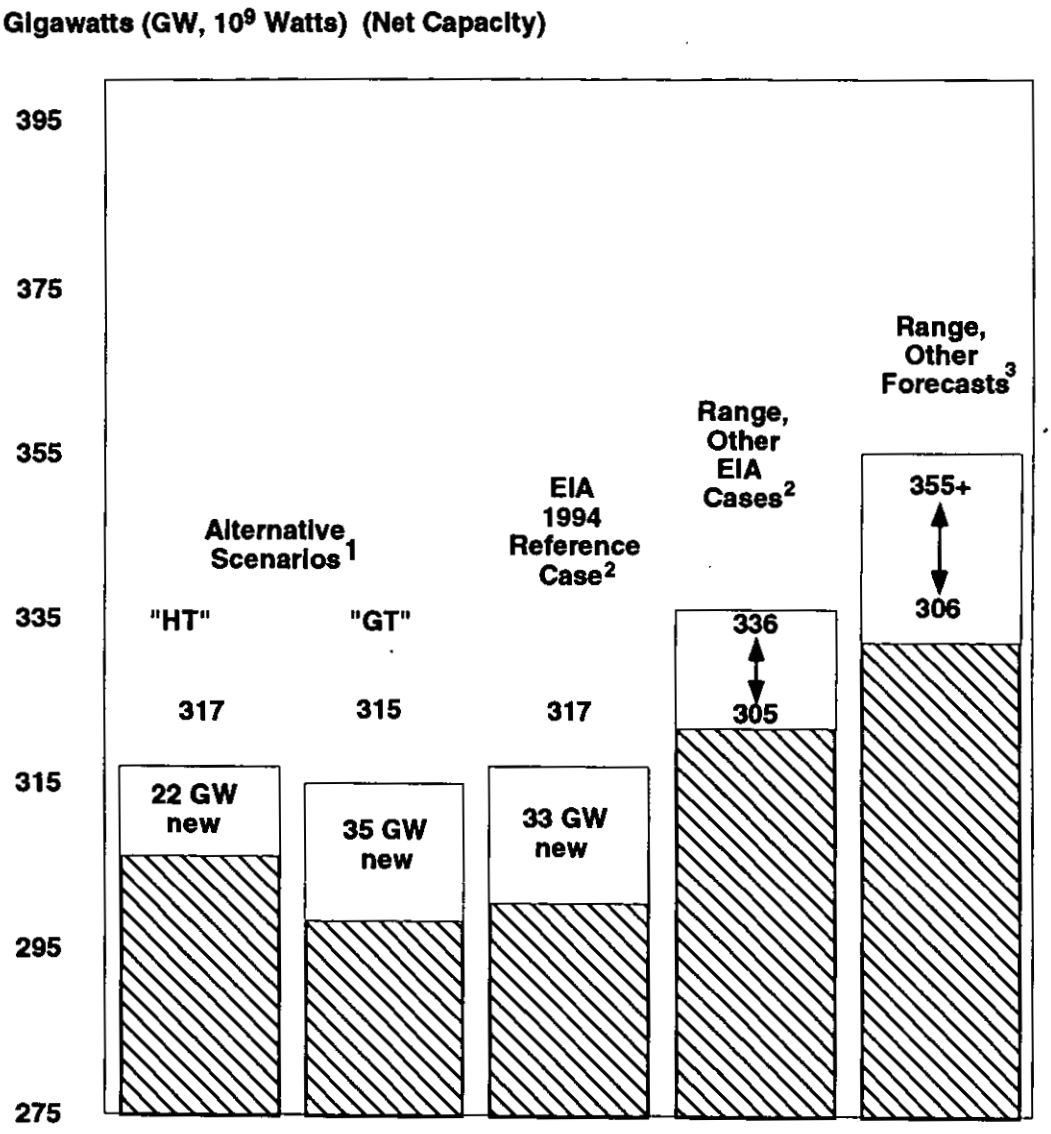
Projecting utility fossil plant capacity and generation out to the year 2010 requires assumptions regarding, for example, economic and load growth, demand side management, competition and non-utility generation, fuel prices including oil-gas price differential, nuclear plant availability, technological advance, plant life extension, and long term unannounced capacity additions and retirements. Different forecasts use different methodologies and assumptions, resulting in considerable divergence among forecasts at the national level and even greater divergence at the regional and system levels. For example, different forecasts predict different distributions of coal-fired generation among different NERC regions and between new versus existing units. Projections of residual oil consumption vary widely, being sensitive to the assumed oil versus gas price differential and also to the magnitude of marginal loads projected to remain after full dispatch of resources with lower energy costs, such as coal, nuclear, and renewable energy plants.

Two of the four alternative scenarios incorporated utility coal and residual oil-based capacity and generation patterned on the 1994 EIA Reference Case described in the Annual Energy Outlook 1994 and its Supplement [2,4]. These "Government Trend" (GT) scenarios (Table C-6) assume that all fossil plants below 100 MW are retired at an age of 45 years, as are oil and gas-fired plants above 100 MW unless located in regions heavily dependent on oil and/or gas (New England, New York, New Jersey, Florida, Louisiana, Arkansas, Oklahoma, Texas, California).

Table C-6.
Alternative EPRI Fossil Plant Scenarios

Scenario GT_p	*Scenario* HT_p*
GT forecast (low generation & oil consumption, faster capacity replacement), plus perfect allowance trading	HT forecast (higher generation & oil consumption, slow capacity replacement), plus perfect allowance trading
Scenario GT_c*	*Scenario* HT_c
GT forecast (low generation & oil consumption, faster capacity replacement), plus constrained allowance trading	HT forecast (higher generation & oil consumption, slow capacity replacement), plus constrained allowance trading
* Scenarios used in subsequent emissions and risk calculations.	

In these GT scenarios, utility coal-fired capacity falls roughly in the mid-range of other forecasts or slightly lower, with faster retirements than projected in many other forecasts (Figure C-1). Like the 1994 EIA Reference Case, the GT scenarios include considerable long term additions of generic (not yet announced) coal capacity, especially in the Southwest Power Pool (SPP) and Western States Coordinating Council (WSCC) NERC regions. Essentially no additions of utility units burning residual oil are included in any major forecasts, so that differences among forecasts regarding future residual oil-burning capacity mainly reflect different assumptions regarding retirements, which are relatively high in the GT scenarios and in the EIA reference case.



1. Additionally, 5-15 GW of non-utility capacity plus a roughly equivalent amount of coal-fired cogeneration are forecast.
2. EIA, Annual Energy Outlook, 1994.
3. References:

   Department of Energy/Energy Information Administration. *Annual Energy Outlook 1994 with Projections to 2010* (January 1994), and *Supplement* (March 1994)

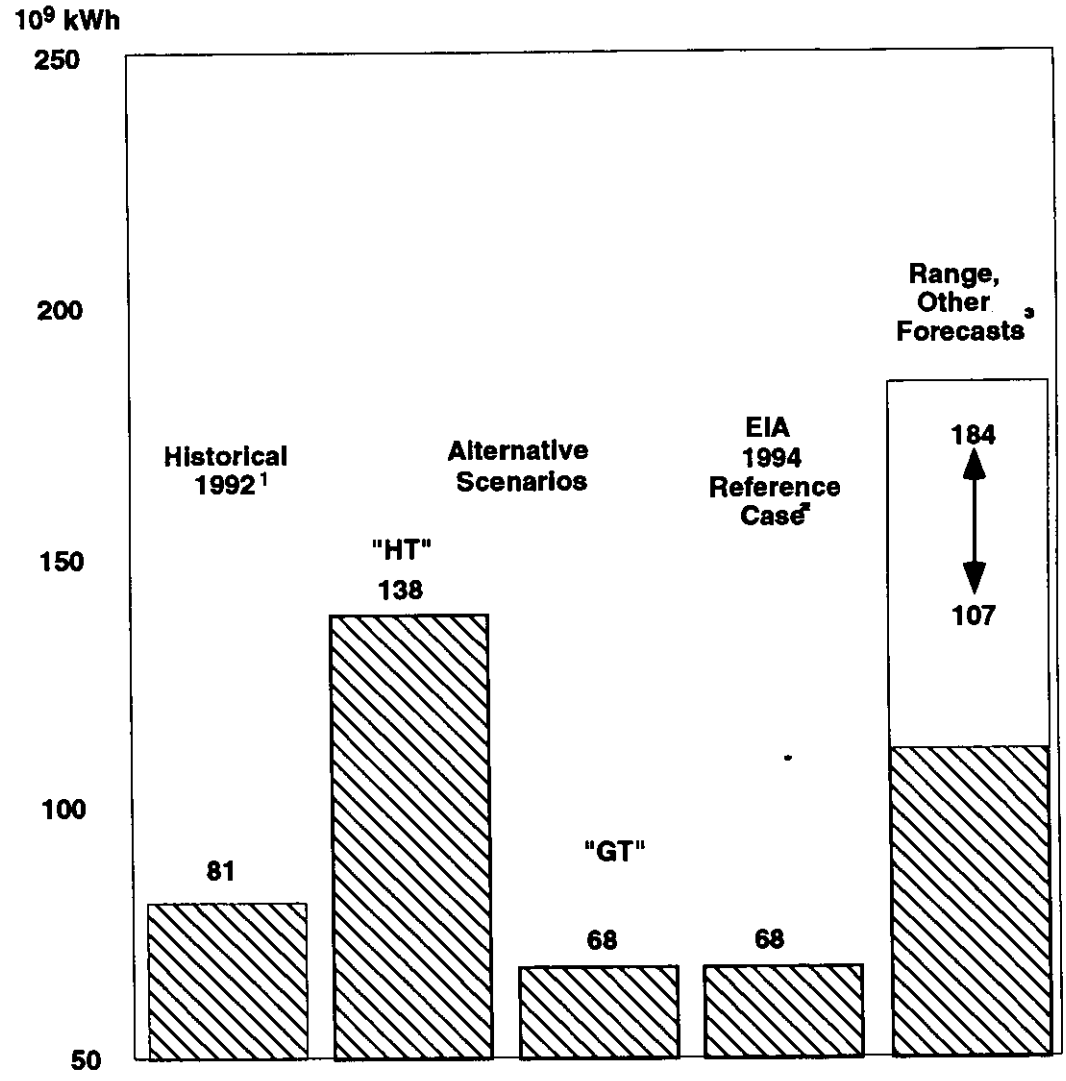
   Gas Research Institute, *GRI Baseline Projection of U.S. Energy Supply and Demand to 2010, 1994 Edition* August 1993)

   DRI/McGraw-Hill, *Energy Review* (Third Quarter 1993)

   Energy Ventures Analysis, Inc., forecast for EPRI (Fall, 1993)

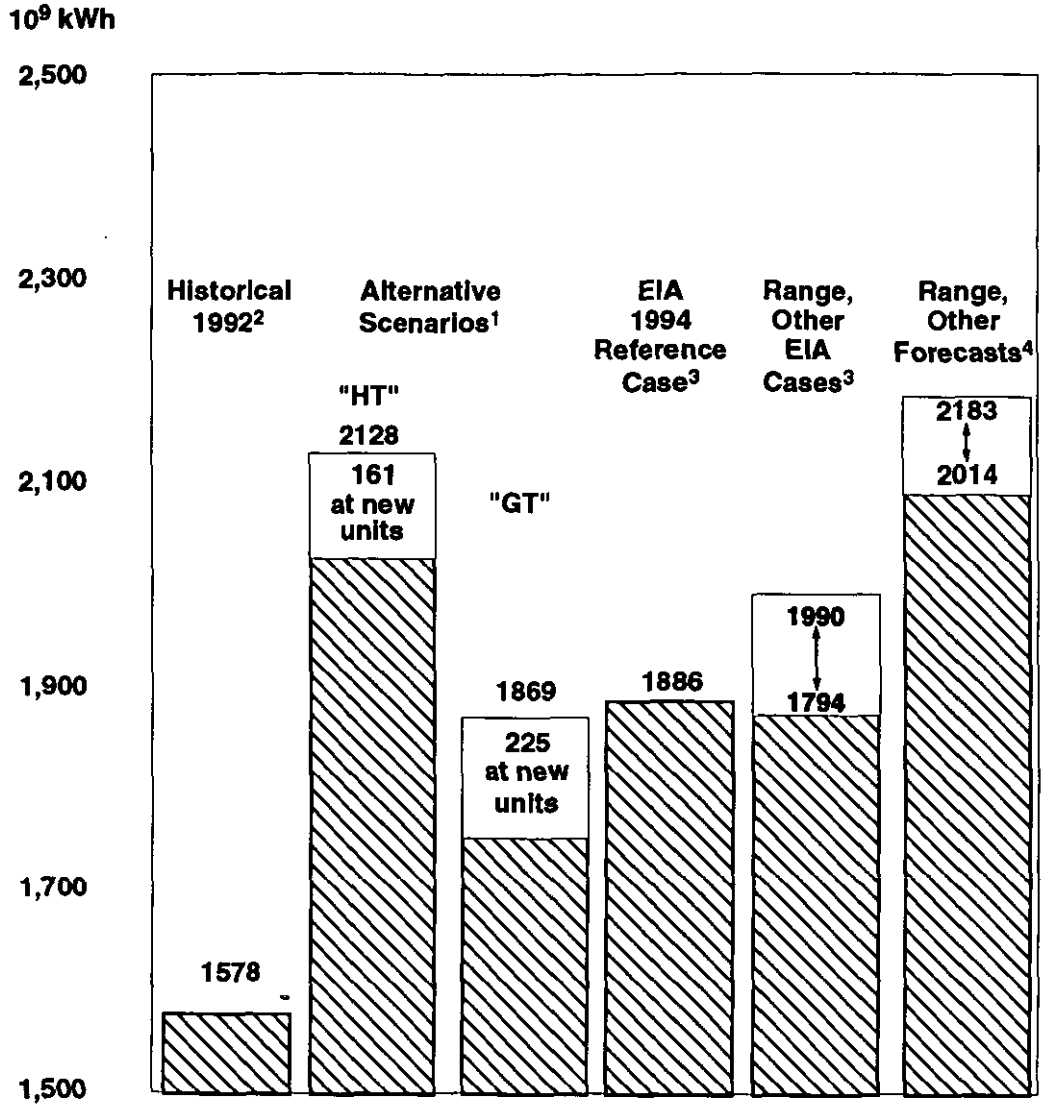
Figure C-1.
Forecasts of Utility Coal-Fired Generating Capacity for Year 2010
("new": begins operation after 1990)

For generation from coal and residual oil, the GT scenarios follow the EIA 1994 Reference Case's projections for individual NERC regions and subregions. This gives forecast national utility residual oil and coal-based generation on the low end of the forecast spectrum (Figures C-2 and C-3), although forecast coal-based generation is actually relatively high for the SPP and WSCC NERC regions.



1. FERC FORM 759.
2. EIA, Annual Energy Outlook, 1994.
3. See Figure C-1. Based on residual oil consumed or oil steam unit generation where available; otherwise 90% of projected total oil generation.

Figure C-2.
Forecasts of Utility Generation: Residual Oil, Year 2010



1. Additionally, roughly 30-100 x 10^9 kWh are forecast from non-utility coal generation.
2. FERC Form 759.
3. EIA, Annual Energy Outlook 1994.
4. See Figure C-1.

Figure C-3.
Forecasts of Utility Generation: Coal, Year 2010 ("new": begins operation after 1990)

The other two alternative fossil plant scenarios (Table C-6) are the "HT" (high trend) scenarios, which project higher residual oil and coal-based generation, more in the mid-range of other recent forecasts (Figures C-2 and C-3). In these HT scenarios, utility coal-based generation for year 2010 is about 35% above the historical 1992 level and about 14% above the level in the GT scenarios. While residual oil-based generation is about 70% above the 1992 level and about twice the level in the GT scenarios, it is still only in the mid-range of recent forecasts (Figure C-2), and roughly equivalent to the high oil

consumption of 1989, when the gas versus oil price differential peaked. To provide further contrast with the GT scenarios, the HT scenarios assume no fossil unit retirements beyond announced retirements, and include relatively low levels of fossil unit additions, limited largely to current announcements (Figure C-1). The HT ("high trend") scenarios are so named because they result in high utilization of existing fossil units.

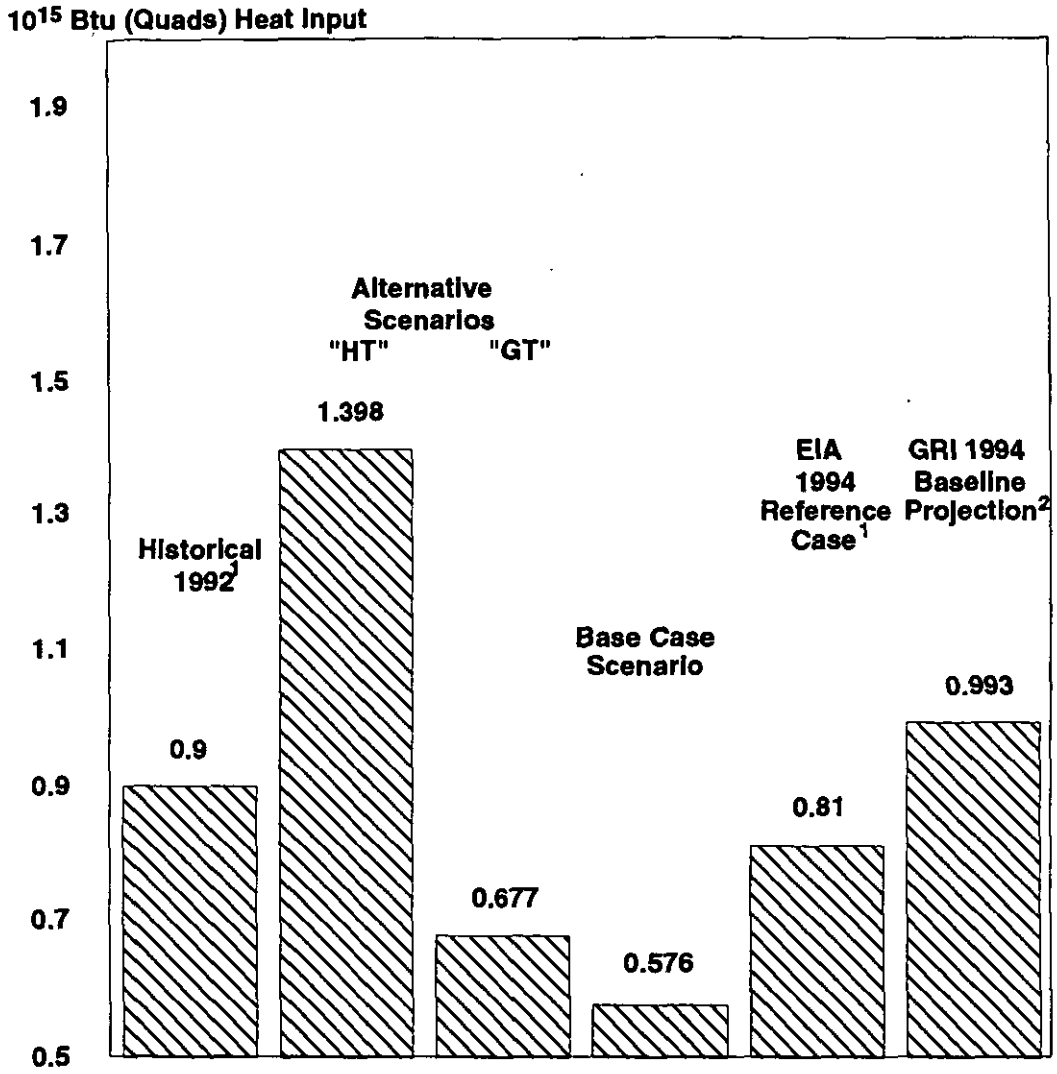
Besides generation and capacity replacement levels, the alternative scenarios explore the implications of two different levels of SO_2 allowance trading. Allowance trading can affect emissions of trace substances by influencing fuel switching and technology retrofits for compliance with SO_2 caps set under the 1990 CAAA. Constraining trading generally increases the projected amount of high cost emission controls that must be implemented in some systems, while simultaneously decreasing projected low cost overcontrol by other systems to generate surplus allowances for sale.

One GT and one HT scenario incorporated perfect economic trading of SO_2 allowances among utilities, minimizing costs nationwide (Table C-6). This is the kind of trading incorporated in the base case scenario used in this study. Such perfect trading will most likely not occur, and therefore one GT and one HT scenario incorporated "constrained" trading. Trading was constrained via a trading "hurdle" under which each utility system was assumed to substitute purchased allowances for more costly within-system emission reductions only where the resulting cost savings exceeded 25 percent of the cost of the purchased allowances.

Of the four scenarios summarized in Table C-6, scenarios GTc (low fossil generation, constrained trading) and HTp (high fossil generation, perfect trading) were considered to provide the most complete bracketing of likely future conditions, and their unit by unit results were used in the succeeding steps of the industrywide assessment of emissions, exposure, and health risk under the alternative industry scenarios.

C.4.4 Scenario Results: Amounts and Types of Fuels Burned

For estimation of trace substance emissions from residual oil-burning units, the key scenario results were the amount of residual oil burned and its distribution among units. This is because the trace substances emissions assessment did not have sufficient data to distinguish among different kinds of residual oils, and the alternative fossil plant scenarios did not generally project fuel switching and emission control retrofits for oil-fired units. As noted, the projected levels of residual oil-based generation in the GT and HT scenarios were at the low and mid-range, respectively, of various recent forecasts (Figure C-2). This results in projections of residual oil consumption that are somewhat higher (GT scenarios) and much higher (HT scenarios) than the consumption given by the industry base case scenario (Figure C-4).

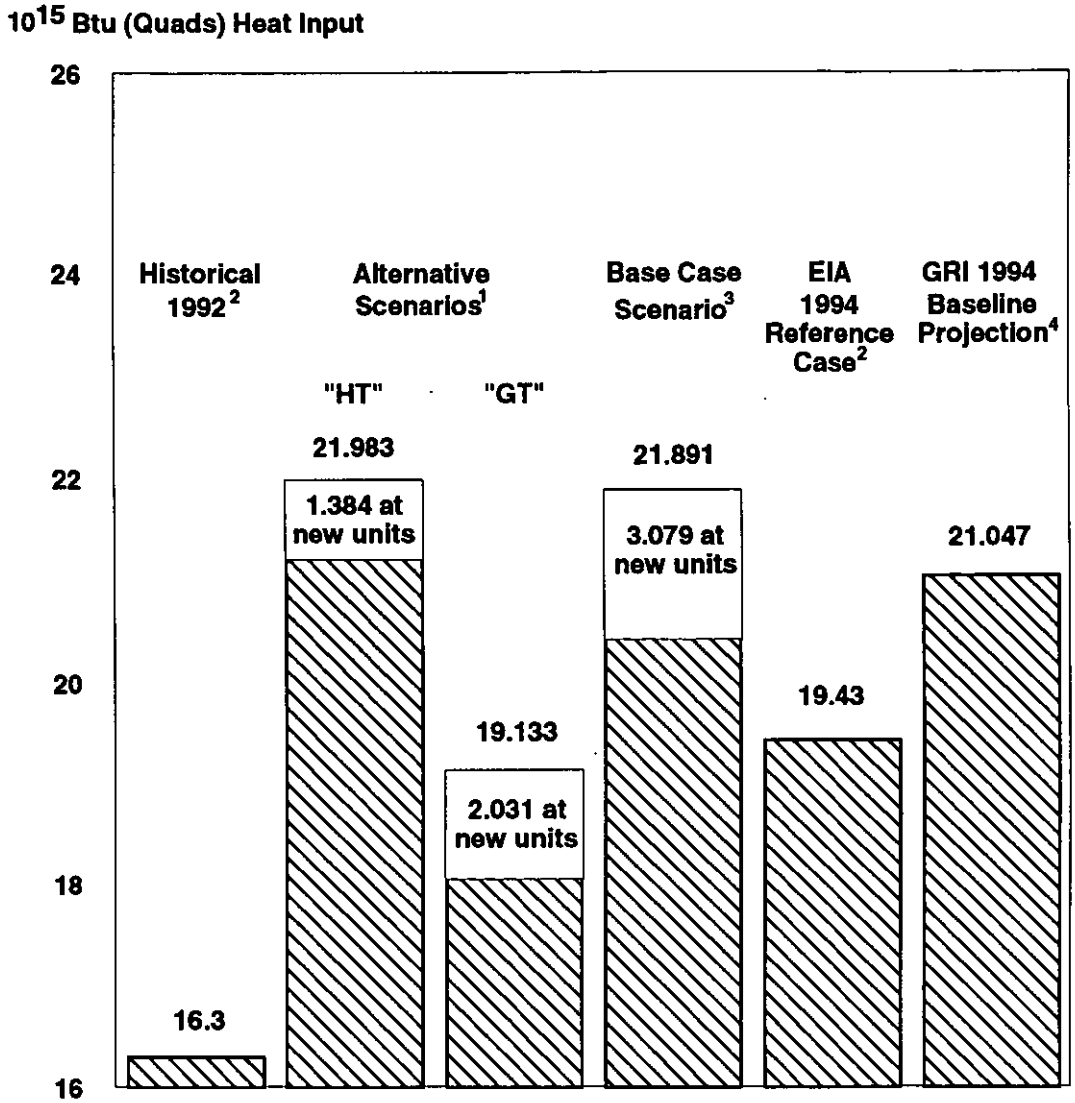


1. EIA, Annual Energy Outlook 1994.

2. Baseline Projection Data Book, 1994 Edition.

Figure C-4.
Projected Utility Residual Oil Consumption Year 2010

For projected coal firing, the GT and HT scenarios fall in the low and middle ranges, respectively, of the forecast spectrum and give a total amount consumed that is roughly 13 percent less than that burned under the base case scenario (for the GT alternative scenario), or equal to that of the base case scenario (for the HT alternative scenario) (Figure C-5). However, relative to the base case, the alternative scenarios and especially the HT scenarios have a greater portion of projected consumption occurring in existing units, often without scrubbers, and with specific locations for which risk can be quantified.



1. In addition, projected coal consumption at non-utility units ranges from 2-7% of utility consumption.
2. EIA, Annual Energy Outlook 1994.
3. After subtracting estimated 0.5 quads at non-utility units.
4. Baseline Projection Data Book, 1994 Edition.

Figure C-5.
Projected Utility Coal Consumption Year 2010 ("new": begins operation after 1990)

It should be noted, as discussed in Section C.4.6 below, that there is sometimes considerable variation in trace substance levels within, as well as between, the different coal seams and basins. Trace substance concentrations vary widely within and between coal seams. Therefore, in addition to the amount of coal burned, the kinds of coal burned can strongly

affect trace substance concentrations. Many uncertainties affect the projected mix of coals burned much more than they affect the projected amount of coal burned. To simplify discussion of the projected mix of coals burned, coals are grouped into regions (Table C-7).

Table C-7.
Projected 2010 Utility Coal Utilitization by Region:
Alternative Scenarios vs. Base Case Scenario (10^{12} Btu)

Coal Region	Base Case	Alternative Scenarios			
		HT_p	HT_c	GT_p	GT_c
N. Appalachian	2715	3093	3332	2841	2950
C. Appalachian	6619	5912	5719	4864	4929
S. Appalachian	505	357	360	355	359
Illinois Basin	2508	2445	2407	2045	1907
Interior West	72	11	64	86	36
Rocky Mountain	1269	1053	1046	1162	1190
Southwestern	852	749	762	809	809
Southern Wyoming	612	429	463	506	583
Powder River Basin	5499	6254	6180	5223	5254
Gulf Lignite	873	945	943	750	750
Plains Lignite	364	372	372	284	284
Pacific Coast	-	91	91	91	91
Imports	-	247	208	116	39
Anthracite	-	25	25	0	0
All	21,891	21,983	21,972	19,133	19,181

The likely effects on coal markets of compliance with the SO_2 cap established by the 1990 CAAA have been examined in a recent EPRI study [1]. The amount of western, especially Powder River, coals employed is expected to be much higher than recent levels, the consumption of Central Appalachian coal somewhat higher, and the consumption of Northern Appalachian and Illinois Basin coal substantially lower. However, the relative contributions of the different regions to overall utility coal utilization vary only slightly among the four alternative scenarios (Table C-7). The higher projected contribution of

western bituminous coal in the GT scenarios largely reflects high generation growth projected for the west relative to the nation overall in the 1994 EIA Reference Case upon which the GT scenarios were patterned.

Constraints on between-utility trading of SO_2 allowances are projected to increase the consumption of Northern Appalachian coals (Table C-6), largely by increasing FGD retrofits in the northeastern part of the country and thus making the higher sulfur local coals more attractive. Constrained trading is projected to have a smaller and less clear-cut effect on the consumption of Central Appalachian and Powder River coals. The amount of switching to very low sulfur Central Appalachian coals will depend strongly on how those coals are priced relative to higher sulfur eastern coals.

While there is only moderate variation among the alternative scenarios regarding the projected mix of coals burned, the contrast with the base case is greater (Table C-6). The alternative scenarios project greater relative contributions of Northern Appalachian and Powder River coals and lower contributions from some other coal basins, mainly Central Appalachia. Underlying differences among scenarios regarding projected nationwide utility coal consumption are some considerable differences in projected amounts and kinds of coal consumed at individual units.

C.4.5 Scenario Results: Flue Gas Desulfurization

Among the factors likely to affect trace substance emissions, the most striking difference among the four alternative fossil plant scenarios is the wide variation in projected flue gas desulfurization (FGD) retrofits (Table C-8). Here, FGD is defined as wet scrubbing or spray drying.

Table C-8.
Projected Utility Coal Unit Flue Gas Desulfurization (FGD)[1] Capacity and Associated Coal Consumption

	Alternative Scenarios[2]				Base Case
Category	HT_p	HT_c	GT_p	GT_c	
10^3 MW OF CAPACITY WITH FGD					
Existing, 1991	67.3	67.3	66.9	66.9	
CAAA FGD retrofits	41.6	53.0	20.3	26.4	

Table C-8.
Projected Utility Coal Unit Flue Gas Desulfurization (FGD)[1] Capacity and Associated Coal Consumption (Continued)

New Units after 1991	19.2	19.2	30.7	30.7	
All Coal Steam Units	128.2	139.5	117.8	123.9	
10^{15} BTU OF COAL CONSUMED					
Existing Units (On Line in 1991)					
All Existing Units	20.60	20.60	17.10	17.10	18.81
With pre-CAAA FGD	5.04	5.04	4.56	4.56	5.773
With post-CAAA FGD	2.88	3.68	1.34	1.74	
Units Operating After 1991, with FGD	1.33	1.33	1.99	1.99	2.91
All Units[4]	21.98	21.97	19.13	19.18	21.89
All Units with FGD	9.25	10.05	7.89	8.29	8.68

1. FGD defined as wet scrubbers or spray dryers, but not sorbent injection SO2 controls. "FGD" also excludes fluidized bed boilers (about 1.3x103 MW in alternative scenarios) and integrated coal gasification-combined cycle units (about 0.5x103 MW in alternative scenarios).
2. See Table C-6.
3. Pre- and post-CAAA FGD combined.
4. Including small amounts of coal consumbed in fluidized bed and gasification-combined cycle units.

The percentage of total coal consumed in units with FGD depends on the relative amounts of new and existing coal-fired capacity and also on projected utility strategies for CAAA compliance under different levels of allowance trading. Although not explored in the scenarios, adding more nonconventional coal plants such as fluidized bed and gasification combined cycle plants to the assumed mix of future coal capacity additions could substantially alter the projected amount of coal burned in units with FGD.

Projected FGD retrofits to meet the CAAA SO_2 caps are about 20-25 GW higher under the high-generation HT scenarios than under the GT scenarios (Table C-8). This reflects greater SO_2 reduction requirements under the HT scenarios, due to higher coal consumption and also due to slower retirements and consequent greater utilization of existing, currently unscrubbed units. However, the total scrubbed capacity is only about 10-15 GW, not 20-25 GW, higher in the HT scenarios, due to assumed faster addition of new scrubbed capacity in the GT scenarios.

Constraining allowance trading by imposing minimum criteria for cost savings per ton for allowance purchases increases projected FGD retrofits by about 6-10 GW (Table C-8). The effect is greatest in the HT scenarios with their high utilization of existing, unscrubbed capacity. The lowest level of projected FGD retrofits among the alternative fossil plant scenarios is about 20 GW, under low generation and perfect allowance trading (scenario GTp, Table C-8). This is only 7 GW beyond FGD retrofits already committed for Phase I SO_2 compliance, and represents a low plausible bound on FGD retrofits, barring dramatic changes in circumstances.

The four alternative fossil plant scenarios project a range of 41 to 46 percent of utility coal consumption to occur in scrubbed units, with the lower figure representing the "low bound" FGD retrofits. This compares closely with the 40 percent projected under the base case scenario. However, the base case includes higher unplanned coal plant additions, all with FGD, so that among "existing" units (those operating in 1991), the base case scenario projects just under 31 percent of coal consumption to occur at scrubbed units, versus 38 to 42 percent for the HT scenarios that have essentially the same overall coal generation as the base case, and 34 to 37 percent for the low-generation GT scenarios. Thus, the base case scenario appears to represent a very low level of FGD at existing units.

Since trace substance concentrations vary considerably in coal, the impact of FGD on emissions depends on how much of which types of coal is burned in scrubbed units. In general the base case and alternative scenarios agree that only about one-fourth of the Powder River coal consumption will be burned in scrubbed units (Table C-9). Much of the consumption of Southwestern, lignite, and, to a lesser extent, Rocky Mountain coal will occur in scrubbed units. The amount of the various western coals projected to be burned at scrubbed units depends on what is assumed regarding the amount of unannounced future capacity in the west.

Table C-9.
Projected Percent of Coal Consumption at Units with FGD[1] in Year 2010, by Coal Mining Region (10^{12} Btu)

Coal Region	Base Case	Alternative Scenarios[1]			
		HT_p	HT_c	GT_p	GT_c
N. Appalachian	62	73	79	51	61
C. Appalachian	12	20	28	21	21
S. Appalachian	7	15	15	0	15
Illinois Basin	87	80	88	62	74
Interior West	100	91	100	97	94
Rocky Mountain	74	57	57	63	61
Southwestern	64	81	80	82	82
S. Wyoming	83	40	45	41	35
Powder River	19	20	19	28	29
Gulf Lignite	71	77	77	81	81
Plains Lignite	68	67	77	80	80
All Coal Consumed	40	42	46	41	43

1. FGD defined as wet scrubbers or spray dryers, but not sorbent injection SO_2 controls. "FGD" also excludes fluidized bed boilers (about 1.3 GW in alternative scenarios) and integrated coal gasification-combined cycle units (about 0.5 GW in alternative scenarios).
2. See Table C-6.

For eastern coals, a higher portion of Appalachian coals are projected to be burned at scrubbed units under the alternative scenarios (Table C-9), especially with higher generation and/or constrained allowance trading. This is important because Appalachian and especially Northern Appalachian coal generally has higher than average trace substance concentrations, and the alternative scenarios project Northern Appalachian coal to make a higher contribution to total utility coal consumption than does the base case scenario (Table C-7). The dynamic interaction between coal selection and FGD retrofits for SO_2 compliance also affects all emissions. Where trace substance concentrations are positively

correlated with coal sulfur levels, SO_2 compliance considerations can result in the coals with the highest trace substance levels being most likely to be burned in scrubbed units, reducing these coals' potential to produce trace substance emissions.

C.4.6 Trace Substance Concentrations in Coal

An alternative characterization of trace substance concentrations in different coals was developed independently from the characterization used with the base case scenario. A simplified portrayal of this alternative characterization is provided in Table C-10; values represent weighted averages across different coals in each region, the weighting based on the total Btu equivalent of each coal projected to be consumed. This alternative characterization is based on analysis of 1,530 samples of "as-shipped" and "as-received" coals from major coal suppliers and utilities, generally weighting samples by the coal production volumes they represent. These data are considered proprietary currently [5].

Table C-10.
Weighted Trace Substance Concentrations for Coal-producing Regions Under Alternative Scenario HTp (High Trend with Perfect Allowance Trading (units: lb/10^{12} Btu)

	Sb	As	Be	Cd	Cr	Co	Pb	Mn	Hg	Ni	Se	Cl	F
Northern Appalachia	112	499	71	21.3	1,136	362	474	1,338	10.6	670	131	91,092	4,677
Central Appalachia	62	391	148	14.3	1,099	395	567	844	5.0	990	192	73,632	5,456
Southern Appalachia	91	828	149	40.8	1,204	743	420	2,158	2.3	1,224	83	15,868	5,494
Illinois Basin	68	391	148	35.7	1,668	215	614	3,448	7.0	1,409	176	57,223	5,405
Powder River Basin	87	120	23	44.3	428	147	309	1,405	7.3	350	63	19,064	6,053
Rockies	55	85	44	11.6	419	111	335	789	4.1	236	78	11,880	6,449
South Wyoming	149	18	23	16.0	64	79	96	3,398	1.5	230	17	20,213	23,197
Arizona-New Mexico	131	137	74	8.2	600	136	262	2,151	3.7	253	134	19,360	39,087
Other Western	128	120	60	36.9	604	81	400	4,894	4.1	646	53	31,223	25,402
Lignite	124	243	69	31.7	1,764	127	790	12,202	6.6	297	128	26,108	19,322
PA Anthracite	27	1,328	386	21.1	3,845	278	1,268	9,471	56.1	2,077	616	120,001	3,000

Table C-10.
Weighted Trace Substance Concentrations for Coal-producing Regions Under Alternative Scenario HTp (High Trend with Perfect Allowance Trading (units: lb/10^{12} Btu) (Continued)

	Sb	As	Be	Cd	Cr	Co	Pb	Mn	Hg	Ni	Se	Cl	F
Average, all regions, weighted on a Btu basis	85	301	86	27.1	950	262	464	2,143	6.7	691	127	49,869	7,836

Weighted average trace element levels. Weighted by projected Btu utilitzation for the different coal categories in each region.

This contrast between the base case trace element characterization and the alternative characterization is attributable to the following:

- The base case trace element characterization uses sample-population averages for trace substances within a region. The alternative trace substance characterization uses a different averaging method. the average trace substance concentration is weighted by projected Btu utilization, by coal category and region.
- Some mercury and arsenic are associated with the pyritic sulfur in coal. Many of the samples used for the base case coal quality characterizations have higher sulfur contents than coal "as-shipped" to utilities and likewise have higher arsenic and mercury levels. For example, the samples used for the base case characterization of Pennsylvania coals are concentrated in the Freeport/Kittanning seams which have higher pyrite and trace substance concentrations than the higher-producing Pittsburgh seam. This situation is reflected in Table C-11, which compares average calculated mercury and arsenic levels for the base case and alternative (all four averaged) scenarios, based on each scenario's projected mix of coals burned.

Table C-11.
Projected Arsenic and Mercury Levels in Coals Consumed: Base Case versus Alternative Scenarios

Coal Producing Region	Arsenic (lb/10^{12} Btu)		Mercury (lb/10^{12} Btu)	
	Base Case	Alternative	Base Case	Alternative
Northern Appalachia	1584	499	11.4	10.1
Central Appalachia	1188	396	7.3	5.1
Illinois Basin	770	396	6.4	7.0
Gulf Coast Lignite	484	44	28.2	5.1
Powder River Basin	330	121	7.5	7.3

- "As-shipped" coals reflect selective mining techniques practiced in the coal industry in which some seams (or partings) bearing high trace element concentrations (reflected in high ash content) may be bypassed.

C.4.7 Discussion

Key assumptions in four major areas impact the potential emissions of trace substances by utility fossil generation plants in a year 2010 scenario:

- the amount of fossil fuel burned,
- the mix of fuels consumed,
- trace element concentrations in the fuels, and
- future emission control measures and generation technologies.

A few points about broader uncertainties in each of these areas are noted below.

Amounts of Coal and Oil Burned: As noted earlier, many factors affect the future levels of coal and oil burned, and forecasts vary widely. Technological or political developments could conceivably drive electricity demand and fossil fuel consumption considerably higher or lower than was considered in scenario development. For example, there could be rapid penetration of electric vehicles or demand side management, or additional costly emission restrictions. Such developments could drive coal generation outside of the roughly 400,000 GWh/year band encompassed by most forecasts for year 2010 (Figure C-3).

Mix of Fossil Fuels: Since trace substance concentrations vary considerably among different fuels, and by region and by seam among coals, one method of lowering trace element emissions is fossil fuel source selection. The amount of projected oil consumed is very sensitive to the assumed oil-gas price differential. If the price of residual oil were to drop by $0.25/MMBtu relative to natural gas, the amount of residual oil consumption could nearly double.

C.5 Sample Emissions Calculations

As a demonstration of the emissions estimation method, this section presents a sample calculation for the New York State Electric and Gas Company Kintigh plant. The emissions estimation procedure characterizes plants based on their design and operating characteristics, the amount of fuel burned, and the characteristics of the fuel burned. In the base case scenario, Kintigh was assumed to burn bituminous coal from central Pennsylvania, and to have an electrostatic precipitator (ESP) and a wet scrubber.

The following calculation of arsenic emissions for the Kintigh plant illustrates the method used for particulate-phase inorganic substances such as arsenic and chromium. The emissions estimation method requires unit-specific information from industry databases,

including fuel heat input data, the ash content in the fuel, and the particulate emissions rate through the control device. Other input data include the trace substance concentration in fuel (Section 4), as well as correlation coefficients and exponents from Section 3.

$$\begin{matrix}\text{Arsenic} \\ \text{emissions}\end{matrix} = \begin{matrix}\text{coefficient} \\ \text{for Arsenic}\end{matrix} \times \left[\frac{\begin{matrix}\text{concentration} \\ \text{of As in coal}\end{matrix}}{\begin{matrix}\text{ash content} \\ \text{of coal}\end{matrix}} * \text{Particulate emissions} \right]^{\begin{matrix}\text{Exponent} \\ \text{for As}\end{matrix}} \times (\text{coal heat input})$$

or, substituting,

$$\begin{matrix}\text{Arsenic} \\ \text{emissions}\end{matrix} = 3.1 \times \left[\frac{(26.04 \frac{lb\,As}{10^6\,lb\,coal})}{(0.1234 \frac{lb\,ash}{lb\,coal})} * \left(0.013 \frac{lb\,ash}{10^6\,Btu} \right) \right]^{0.85} \times (52.2 \frac{10^{12}\,Btu}{year})$$

or, finally,

$$\begin{matrix}\text{Arsenic} \\ \text{emissions}\end{matrix} = 381\,lb\,As / year$$

The emissions estimation method for volatile inorganic substances (e.g., mercury, selenium) requires unit-specific fuel heat input data. Other input data include the trace substance concentration in fuel and the percent of coal level emitted (i.e., the fraction of the volatile inorganic substance that passes through the control device) from Tables 3-5 to 3-7, and fuel heat content. The fraction of the volatile inorganic substance that passes through the control differs according to the substance and control configuration. The following calculation of mercury emissions for the Kintigh plant illustrates the method used for volatile inorganic substances.

$$\begin{matrix}\text{Mercury} \\ \text{emissions}\end{matrix} = \left[\begin{matrix}\text{concentration} \\ \text{of Hg in coal}\end{matrix} * \left(\frac{\begin{matrix}\text{coal} \\ \text{heat input}\end{matrix}}{\begin{matrix}\text{coal} \\ \text{heat content}\end{matrix}} \right) * \begin{matrix}\text{Fraction of Hg} \\ \text{input emitted,} \\ \text{with particulate /} \\ SO_2 \text{ controls}\end{matrix} \right] \times 10^6$$

or, substituting,

$$\begin{matrix}\text{Mercury} \\ \text{emissions}\end{matrix} = \left[\left(0.250 * \frac{lb\,Hg}{10^6\,lb\,coal} \right) \frac{\left(52.2 \frac{10^{12}\,Btu}{year} \right)}{\left(12900 \frac{Btu}{lb\,coal} \right)} * 0.55 \right] \times (10^6)$$

or, finally,

$$\underset{emissions}{Mercury} = 557\,lb\,Hg\,/\,year$$

The emissions estimation method for organic compounds requires unit-specific fuel heat input and substance-specific emission factors from Table 3-8. The following calculation of benzene emissions illustrates the method used for organic compounds.

$$\underset{emissions}{Benzene} = \left(\begin{array}{c} Coal\,emissions \\ factor\,for \\ benzene \end{array} * \begin{array}{c} Coal \\ heat\,input \end{array} \right)$$

or, substituting,

$$\underset{emissions}{Benzene} = \left(3.8 \frac{lb}{10^{12}\,Btu}\right) * \left(52.2 \frac{10^{12}\,Btu}{year}\right)$$

or, finally,

$$\underset{emissions}{Benzene} = 198\,lb\,benzene\,/\,year$$

C.6 References

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2. Energy Information Administration, *Annual Energy Outlook 1994 with Projections to 2010*, DOE/EIA-0383(94), January 1994.
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4. Energy Information Administration, Supplement to the Annual Energy Outlook 1994, DOE/EIA-0554(94), March 1994.
5. Energy Ventures Analysis, Inc., Trace Elements in Fuels: Challenges in Estimating Trace Element Concentrations Entering Into Utility Boilers, prepared for the Electric Power Research Institute, November 1993 (draft).

APPENDIX D
EPRI STUDY OF MERCURY IN COAL

EPRI Mercury in Coal Study:
A Summary Report for Utilities
That Submitted Samples
Update

Prepared for

Utility Air Regulatory Group
Hazardous Air Pollutants Committee

Frank McDowell and Scott Osbourn, Cochairmen

June 1994

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Executive Summary

Due to the questionable quality of historical mercury in coal data and the failure to obtain material mass balance closure in some recent power plant testing projects, EPRI decided to foster the development of a new analytical method for determining mercury concentrations in coal and to analyze a number of coal samples for mercury content using this new method. In the first round of the study, approximately 100 coal samples that represent a significant portion of the current and anticipated future coal supplies for 26 electric utilities were analyzed under the direction of Dr. Nicolas Bloom, the developer of the new analytical method for mercury in coal. The first approximately 100 samples analyzed included no samples from several coal-producing states (Arizona, Montana, Utah, and Washington), however, and coal supplies for several other major coal-producing states (Colorado, Indiana, Ohio, Texas, Virginia, and Wyoming) were under-represented in the sample pool. To fill in gaps and improve the overall representativeness of the database, it was decided to obtain and analyze approximately 50 additional samples of coal from the unrepresented and under-represented states.

The accuracy of the method used in the study ranges from approximately 2.8% when judged by results for analysis of South African reference material coal, to approximately 11.8%, when judged by results for analysis of National Bureau of Standards standard reference material coal. The precision of the method, as indicated by the relative standard deviation for multiple analyses of the same field samples, is approximately 8.5%, ranging from 0.9% to 22.8% across samples over the two rounds of the study.

In the first round of the study, Dr. Bloom analyzed a total of 87 bituminous, 8 subbituminous, and 6 lignite coal samples from 53 counties in 14 states, including over 20 major coal seams and 82 different supplies. Over the two rounds, a total of 106 bituminous, 37 subbituminous, and 11 lignite samples were analyzed. These include samples of coal from a total of 76 counties in all 18 major coal-producing states in the U.S. In aggregate, the results from the two rounds of the study are believed to provide an even more broad-based representation of the mercury content of U.S. coals than was provided by the previously reported results from the first round of the study.

The overall average mercury concentration for the approximately 100 samples analyzed in the first round of the study was 0.089 ppm, with a standard deviation of 0.070 ppm. In the second round, the overall average mercury concentration was very similar (0.078 ppm), with a standard deviation of 0.080 ppm. Over the two rounds of the study, the overall average mercury concentration was 0.085 ppm, with a standard deviation of 0.074 ppm. The average mercury concentration for all bituminous coal samples analyzed in the two rounds (0.087 ppm) was almost the same as the overall average and had a similar standard deviation of 0.070 ppm. This average value for bituminous coal is approximately one half of the average value of 0.21 ppm that is reported for bituminous coal for the U.S. Geological Survey database.

Only 8 subbituminous coal samples were analyzed in the first round of the study. These samples averaged 0.044 ppm, with a standard deviation of 0.015 ppm. In the second round, 29 additional

subbituminous coal samples were analyzed, thereby substantially increasing the breadth of the database for subbituminous coals. Over the two rounds of the study, the average mercury concentration for the subbituminous samples was 0.053 ppm, with a standard deviation of 0.027 ppm, which is not significantly different from the first round results.

In the first round of the study, the average mercury concentration for three Texas lignite samples was 0.116 ppm and the average mercury concentration for three North Dakota lignite samples was 0.075 ppm, resulting in an average concentration of 0.095 ppm for lignite, which did not differ significantly from the average for the bituminous coals (0.092 ppm). In the second round of the study, however, the average mercury concentration for five additional Texas lignite samples (0.274 ppm) was about two and one-half times the average concentration for Texas lignite in the first round, resulting in an average concentration of 0.215 ppm for the eight Texas lignite samples analyzed in the two rounds. Due to the high concentration of mercury for the Texas lignite samples analyzed in the second round, the average mercury concentration for the eight Texas and three North Dakota lignite samples analyzed over the two rounds (0.177 ppm) is considerably higher than the 0.095 ppm average for the lignite samples analyzed in the first round and is also considerably higher than the average for all bituminous (0.087 ppm) and subbituminous (0.053 ppm) coal samples analyzed in the two rounds.

Introduction

As a task under its Field Chemical Emissions Monitoring (FCEM) project, the Electric Power Research Institute (EPRI) authorized Frontier Geosciences (FGS), under the direction of Dr. Nicolas S. Bloom, to determine the mercury content for a number of samples of coals burned by electric utilities. Mr. Paul Chu succeeded Dr. Donald Porcella as the EPRI Project Manager for this work. At EPRI's request, the Utility Air Regulatory Group (UARG) Hazardous Air Pollutants (HAP) Committee agreed to assist by helping obtain the samples to be analyzed and by preparing a summary report of the analytical results for the companies that submitted samples. A principal reason for UARG HAP Committee involvement was to ensure that companies could submit samples without the source of the samples being know outside of UARG. The HAP Committee Cochairmen asked Systems Applications International (SAI) to coordinate the Committee's assistance to EPRI and to prepare this report.

During the fall of 1993, 101 samples of domestic coal were analyzed for mercury content under this project. The results for these samples were reported in the initial version of this report. While the results for these 101 samples provide a broad-based representation of the mercury content of U.S. coals, no samples had been obtained for coal from several coal-producing states (Arizona, Montana, Utah, and Washington), and coal supplies for several other major coal-producing states (Colorado, Indiana, Ohio, Texas, Virginia, and Wyoming) were under-represented in the sample pool. To fill in gaps and improve the overall representativeness of the database, it was decided to obtain and analyze approximately 50 additional samples of coal from the unrepresented or under-represented states. This report presents the results for these additional samples, along with the combined results for all samples analyzed.

Background

The electric utility industry is interested in obtaining accurate information on the mercury content of coal for several reasons. First, due to the quality of the analytical methods and laboratory quality assurance measures used in previous studies, the accuracy of some historical measurements of mercury concentration in coal is questionable, making it impossible to judge the accuracy of emission estimates derived from these historical data. Second, even today some mercury in coal measurements are of questionable accuracy. For example, in some recent trace metal testing projects at power plants, as much as 50% or more of the reported mercury in coal cannot be accounted for in the emissions. A positive bias in the mercury in coal measurements due to sample contamination or other causes is one possible explanation. The questionable quality of historical mercury in coal data, and the failure to obtain material mass balance closure in some recent power plant testing projects, were the principal impetuses for EPRI to develop a new analytical method for determining mercury concentrations in coal and to analyze a number of coal samples for mercury content using this new method. A new analytical method, based on cold vapor atomic fluorescence spectrometry after wet oxidation of samples using perchloric acid, has been developed by Dr. Nicolas Bloom and colleagues [1]. This new method was used in the EPRI mercury in coal study summarized in this report.

Coal Samples for the Study

To obtain the initial samples for the EPRI mercury in coal study, the UARG HAP Committee wrote a letter to utilities represented on the Committee, asking them to submit samples for analysis. Recipients of the letter were asked to submit samples that represent a significant portion of their company's current and anticipated future coal supplies. To obtain the second round of samples from the unrepresented or under-represented states, the HAP Committee asked SAI to contact companies by telephone that were believed to use coal from the target states. For both the first and second round of samples, participants were requested to provide some basic information for each sample submitted, including: (1) short proximate analysis data; (2) any additional available analytical data for the sample, such as results of ultimate or trace metal analyses; (3) information on the origin of the coal, including state, county, seam, and mine; (4) coal rank; and (5) other miscellaneous information, including lot size and sampling method. Participants were asked to assign each sample a company identification (ID) number and submit the samples to SAI for assignment of a study ID number and for forwarding of selected samples to FGS for analysis.

In response to the initial HAP Committee letter, 26 companies submitted a total of 198 coal samples and one limestone sample[1]. Since 100 analyses had been set by EPRI as the approximate initial limit for the study, SAI selected and forwarded 102 coal samples (including one sample of a non-domestic coal) and the one limestone sample to FGS for analysis. The following criteria were used in selecting the initial samples for analysis. First, at least one sample submitted by each participating company was selected. Next, at least one sample was selected for each different county and coal seam represented in the sample pool. Preference was given to single-seam samples over samples for multiple-seam blends. Several samples that had already been analyzed for mercury content by laboratories other than FGS were selected. Finally, replicate samples[2] of several coals were included to investigate within-seam variability.

In response to the telephone contacts, 19 companies (including 4 of the initial 26, which were recontacted) submitted a total of 56 samples from the target states. Three of these samples were received after the sample submittal deadline. The remaining 53 were forwarded by SAI to FGS for analysis. Over the two rounds, 154 domestic coal samples were analyzed for mercury content by FGS.

Accuracy and Precision of the Method

A formal evaluation of the accuracy and precision of the method developed by Dr. Bloom has not been performed. Nonetheless, quality control results reported by Dr. Bloom for the first and

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[1] Submitted for analysis to investigate whether mercury in limestone might significantly contribute to mercury emissions for atmospheric fluidized bed combustion systems or for power plants equipped with wet flue gas desulfurization systems.

[2] Samples of coal from the same mine collected at different times.

second round of samples can be used to assess the accuracy and precision of the method.

Accuracy. The results reported by Dr. Bloom for analyses of reference material coals can be used to assess the accuracy of the method [2,3]. These results are shown in Table 1.

Table 1. Summary of FGS Results for Reference Material Samples

	FGS Results			Difference from Certified Value	
NT S-1630	Number of Analyses	Mean (ppm)	Std. Dev. (ppm)	(ppm)	%
First Round	14	0.114	0.0045	0.013	10.2%
Second Round	9	0.110	0.0106	0.017	13.4%
Combined	23	0.112	0.0078	0.015	11.8%
SARM-20					
First Round	13	0.240	0.013	0.010	4.0%
Second Round	8	0.248	0.005	0.002	0.8%
Combined	21	0.243	0.011	0.007	2.8%

The apparent accuracy of the method is approximately 2.8% when judged by results for analysis of South African Reference Material (SARM) coal (certified value = 0.250 ppm; 95% confidence interval 0.180 to 0.270 ppm). When judged by results for analysis of National Bureau of Standards (NBS) standard reference material (SRM) coal (certified value = 0.127 ppm; standard deviation 0.013 ppm), the apparent accuracy of the method is approximately 11.8%. The FGS results were always lower than the certified value for SRM-1630. Dr. Bloom notes that other laboratories (presumably using other methods) have also reported results on the low end of the certified range for NBS SRM-1630, possibly bringing into question the certified value, which was established 15 years ago [2].

Precision. Dr. Bloom reports that during analysis of the first 101 samples, the relative standard deviation for triplicate analyses of the same field sample averages approximately 10.2% and ranges from 5.4% to 22.8% across samples. For the second round of samples, the relative standard deviation for multiple analyses of the same field samples ranges from 0.9% to 12.6% across samples, and averages 7.4%. Overall, the relative standard deviation for multiple analyses of field samples averages 8.5%. These results are shown in Table 2.

Table 2. Summary of FGS Results for Multiple Analyses of Field Samples

	Field Sample Number	Number of Analyses	Mean (ppm)	RSD
First Round	BHAB250401	3	0.081	6.8%
	LA270103	3	0.086	22.8%
	BHA050501	3	0.099	5.6%
	BM150101	3	0.331	5.4%
Second Round	BHB125901	6	0.019	12.1%
	SA290301	3	0.018	3.6%
	SC310101	7	0.042	6.0%
	SA240301	3	0.010	9.0%
	BHA370101	6	0.029	12.6%
	LA380501	6	0.419	0.9%
Combined		43	0.113	8.5%

Results

The following subsections summarize the mercury concentrations reported by FGS for the 101 first round samples and 53 second round samples. Combined results for the two rounds are also summarized. Results from analysis during the first round of a sample of Venezuelan coal (0.051 ppm Hg) and a limestone sample (0.003 ppm Hg) are not included in the following discussions of results. All concentrations are on an as-received moisture basis.

Mercury concentrations by state of origin of samples. Of the 53 second round samples, a total of 14 were for four states (Arizona, Montana, Utah, and Washington) for which no samples had been obtained in the first round. The remaining 39 second round samples were for six major coal-producing states (Colorado, Indiana, Ohio, Texas, Virginia, and Wyoming) that were under-represented in the first round. These six states were considered to be under-represented in the first round for the following reason: While coal production from these six states represented 42.3% of the production in 1992 from the top 16 coal producing states, samples from these states represented only about 10% of all samples that were analyzed in the first round of the EPRI study.

Figures 1 through 4 illustrate the geographic distribution of the coals analyzed in the two rounds of the study. Figure 1 shows the counties in the 48 contiguous states where coal was mined in 1989 [4]. Figure 2 shows the 53 counties of origin of the 101 samples analyzed in the first round of the study. Figure 3 shows the 76 counties of origin of the 154 samples analyzed in the

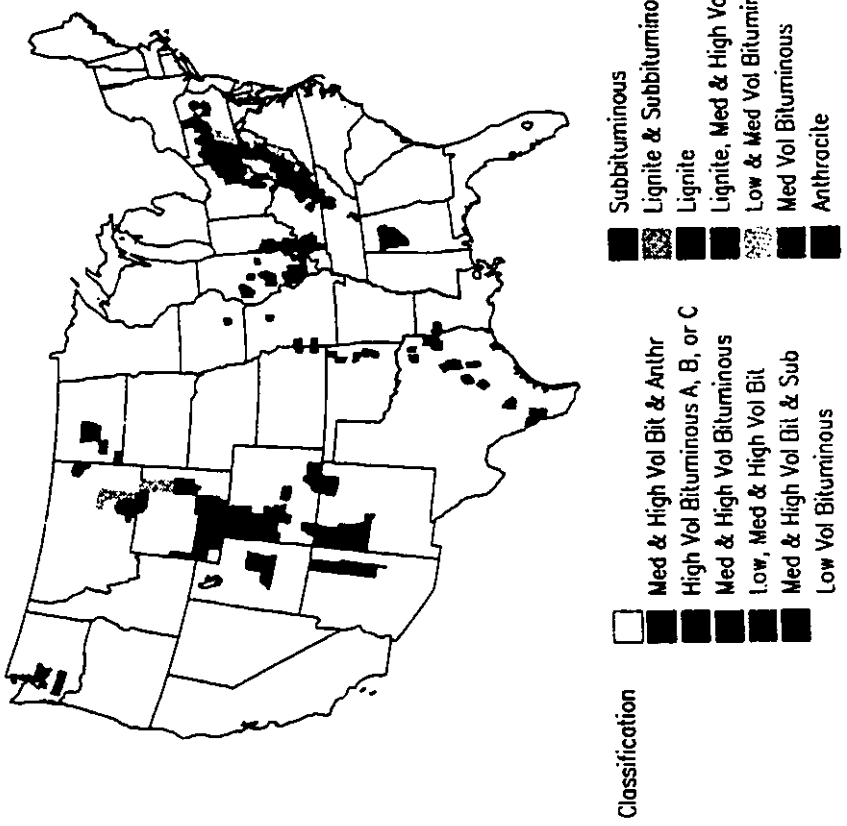

Figure 1. Map of counties where coal was mined in 1989.

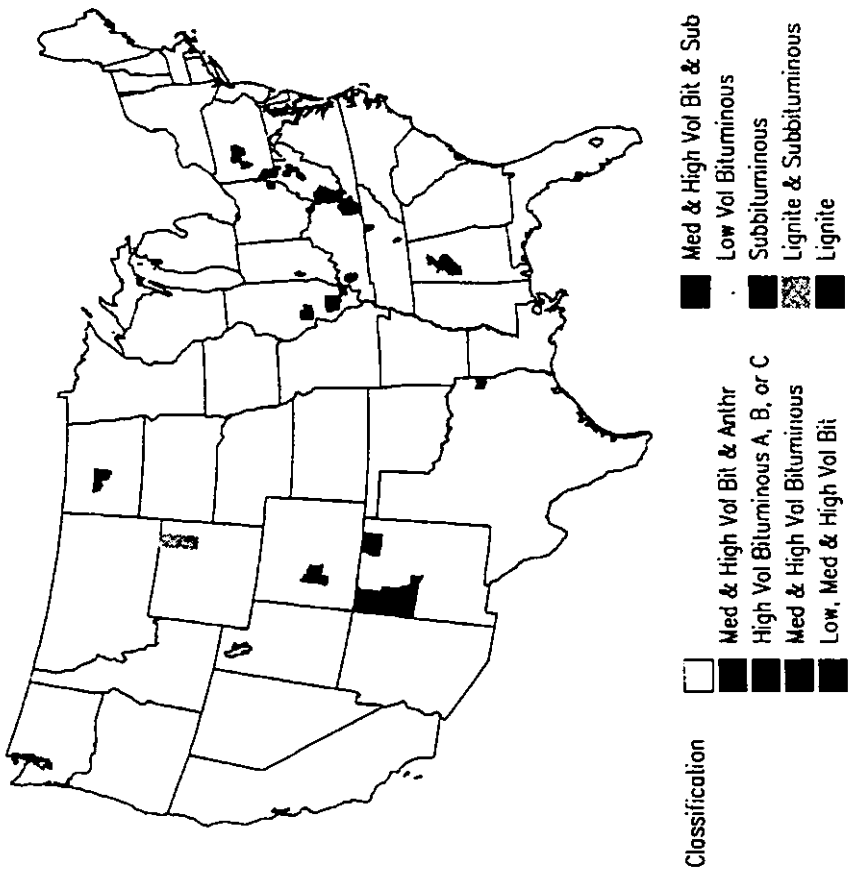

Figure 2. Counties of origin of initial coal samples.

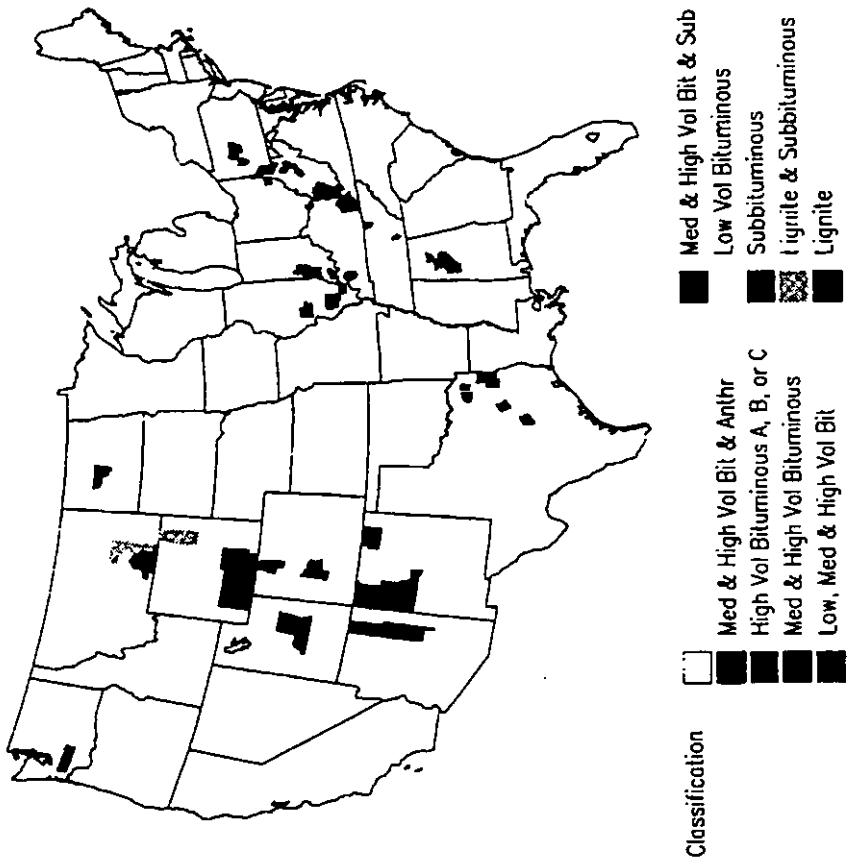

Figure 3. Counties of origin of all coal samples.

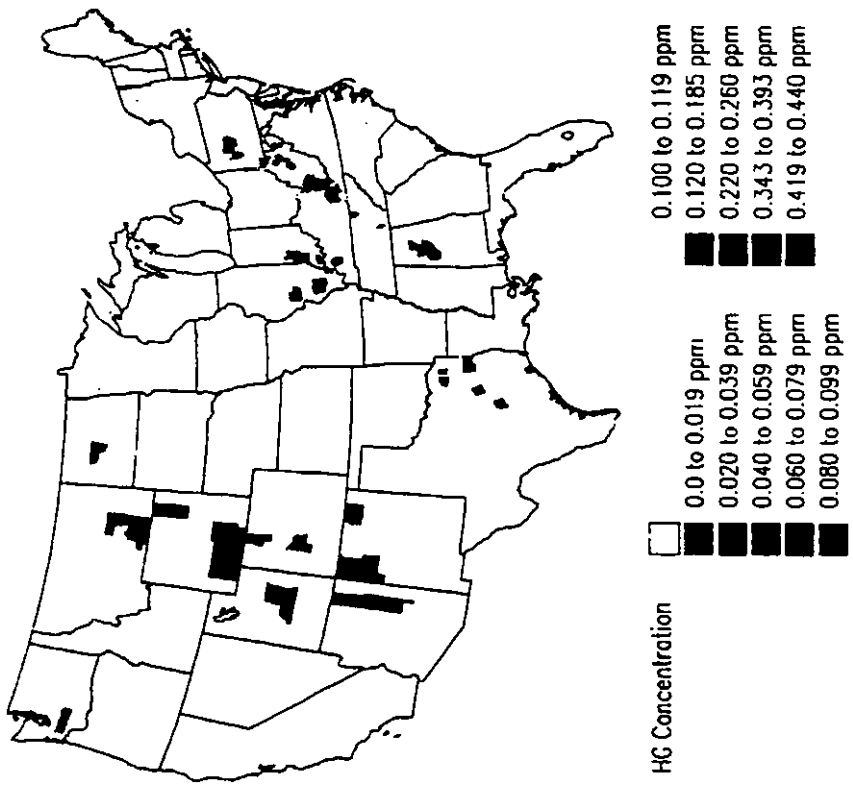

Figure 4. Mercury in coal by counties of origin of coal samples.

two rounds of the study. Figure 4 shows the average concentrations of samples analyzed in the two rounds of the study plotted by county of origin of the samples. The wider geographic distribution of coals covered by the study that was achieved through analysis of the second round of samples can be seen by comparing Figures 2 and 3.

Figures A1 through A3 in Appendix A of this report provide an overview of the breadth of the study in terms of the number of mines and tonnage production of mines for which samples were analyzed. Figure A1 shows the tonnage production of all mines in the states and the total tonnage production of the mines for which samples were analyzed. Figure A2 shows the approximate number of mines in each state, and the number of mines for which samples were analyzed. Figure A3 shows the number of mines and the tonnage production of mines for which samples were analyzed as a percent of the total number of mines in the state and the total tonnage production for the state.

Table 3 shows the mercury concentrations measured by FGS for the 101 first round samples, 53 second round samples, and for the combined 154 samples, summarized by the state of origin of the samples. It can be seen from Table 3 that based on the combined data for the two rounds of the study, the highest average mercury concentrations were for the Texas (lignite) samples. It can also be seen that, with the exception of Texas, for states where samples were analyzed in both rounds, the results for the two rounds are similar. Figure 5 shows the average mercury concentration for the samples from each state that were analyzed in the first round of the study. Figure 6 shows the average mercury concentration for all samples from each state analyzed in the two rounds of the study. The significant increase in the average mercury concentration for the Texas lignite samples from an average of 0.116 ppm in the first round to an overall average of 0.215 ppm for the two rounds can be seen from the two figures. The insignificant change in the average concentration for the other states that were under-represented in the first round (Colorado, Indiana, Ohio, Virginia, and Wyoming) can also be seen from the two figures.

One utility submitted mercury concentration data for two Wyoming coal samples and two Montana coal samples analyzed under the direction of Dr. Bloom for a different project. The average concentration for the two Wyoming samples is 0.071 ppm; the average concentration for the two Montana samples is 0.058 ppm. The average for the Wyoming samples is within one standard deviation of the mean for the Wyoming samples shown in Table 3 (0.064 $\pm$0.026 ppm), and the average for the Montana samples is just outside the range defined by the mean $\pm$ one standard deviation for Montana (0.038 $\pm$0.019 ppm) shown in Table 3.

Mercury concentrations by coal rank. Utilities were asked to identify the rank of the coal samples submitted according to ASTM D388 (Standard Classification of Coals by Rank) [4]. The D388 scheme for classifying coal according to rank is shown in Table B1 in Appendix B to this report. Table 4 shows the mercury concentrations measured by FGS for the 101 first round samples, 53 second round samples, and for the 154 samples combined, summarized by coal rank classified according to D388. The data presented in Table 4 for the 101 first round samples and for the 154 first and second round samples are shown in Figure 7.

Table 3. Mercury Concentrations by State of Origin of Samples

State	First Round			Second Round			Combined		
	Number of Samples	Mean (ppm)	Std. Dev. (ppm)	Number of Samples	Mean (ppm)	Std. Dev. (ppm)	Number of Samples	Mean (ppm)	Std. Dev. (ppm)
AL	9	0.071	0.078				9	0.071	0.078
AZ				2	0.029	0.002	2	0.029	0.002
CO	1	0.017		2	0.043	0.024	3	0.034	0.023
IL	13	0.063	0.025				13	0.063	0.025
IN	1	0.099		5	0.073	0.053	6	0.077	0.049
KY	17	0.106	0.067				17	0.106	0.067
MT				3	0.038	0.019	3	0.038	0.019
NM	15	0.044	0.019				15	0.044	0.019
ND	3	0.075	0.010				3	0.075	0.010
OH	1	0.103		3	0.116	0.003	4	0.113	0.006
PA	14	0.159	0.115				14	0.159	0.115
TN	2	0.088	0.045				2	0.088	0.045
TX	3	0.116	0.018	5	0.274	0.112	8	0.215	0.118
UT				7	0.022	0.009	7	0.022	0.009
VA	3	0.082	0.010	3	0.080	0.030	6	0.081	0.022
WA				2	0.019	0.005	2	0.019	0.005
WV	18	0.084	0.038				18	0.084	0.038
WY	1	0.065		21	0.064	0.027	22	0.064	0.026
TOTAL	101	0.089	0.070	53	0.078	0.080	154	0.085	0.074

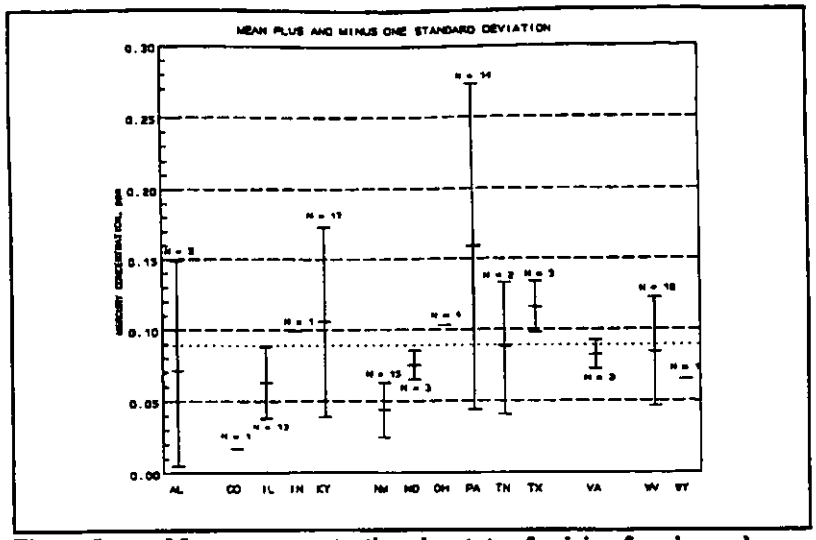

Figure 5. **Mercury concentrations by state of origin of coal samples — first round data.**

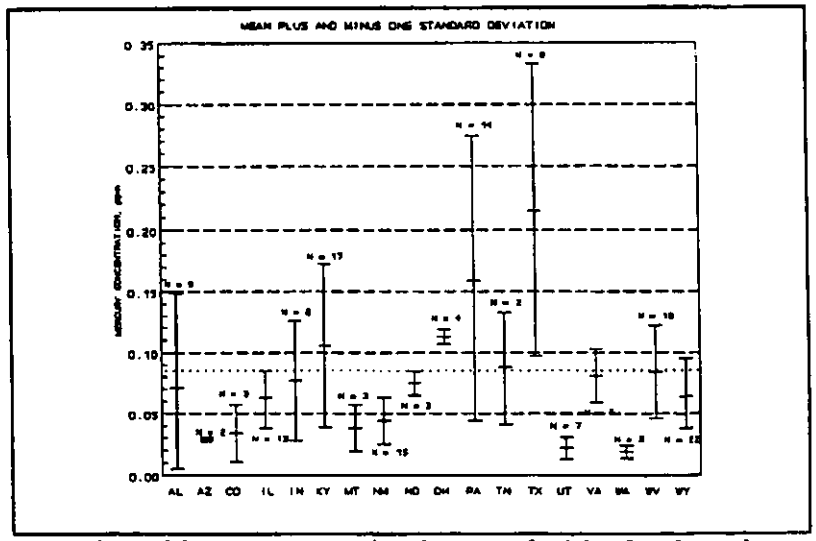

Figure 6. **Mercury concentrations by state of origin of coal samples — first and second round data combined.**

Table 4. Summary of Mercury Concentrations by Coal Rank

Coal Rank	First Round			Second Round			Combined		
Bituminous	Number of Samples	Mean (ppm)	Std. Dev. (ppm)	Number of Samples	Mean (ppm)	Std. Dev. (ppm)	Number of Samples	Mean (ppm)	Std. Dev. (ppm)
Low volatile	7	0.054	0.030				7	0.054	0.030
Medium volatile	9	0.130	0.107	3	0.109	0.036	12	0.125	0.095
High volatile	71	0.091	0.068	16	0.051	0.041	87	0.084	0.066
All bituminous	87	0.092	0.073	19	0.060	0.045	106	0.087	0.070
Subbituminous	8	0.044	0.015	29	0.055	0.029	37	0.053	0.027
Lignite	6	0.095	0.025	5	0.274	0.112	11	0.177	0.118
All ranks	101	0.089	0.070	53	0.078	0.080	154	0.085	0.074

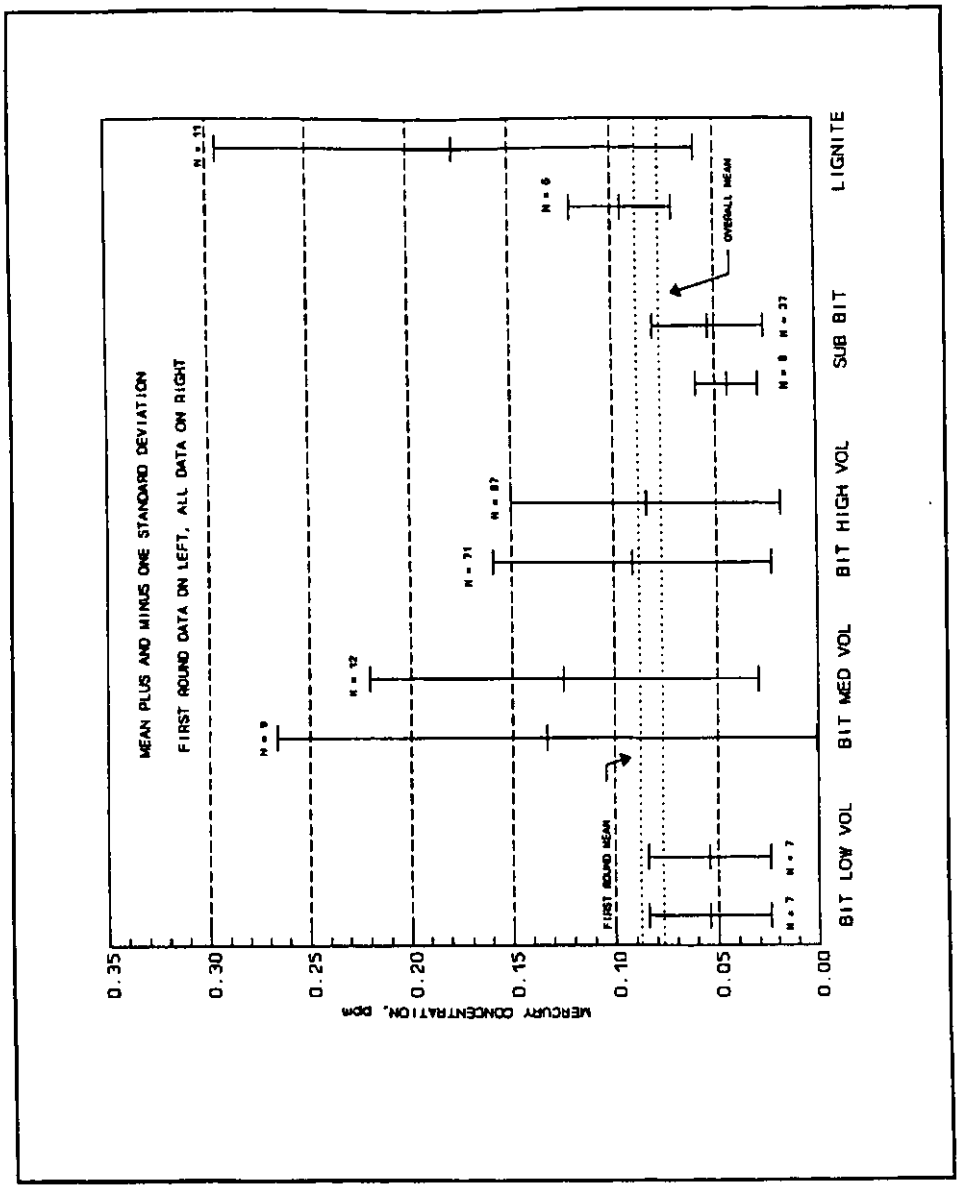

Figure 7. **Mercury concentrations by coal rank — first round data and combined first and second round data.**

As shown in Table 4, the average mercury concentration for the 87 bituminous coal samples in the first round of samples is 0.092 ppm, with a standard deviation of 0.073 ppm. In the second round, the average mercury concentration for bituminous coals is somewhat lower (0.060 ppm), with a smaller standard deviation of 0.045 ppm. The overall average mercury concentration for the 106 bituminous coal samples analyzed in the two rounds is 0.087 ppm, with a standard deviation of 0.070 ppm. This average value for bituminous coal is approximately one half of the average value of 0.21 ppm that is reported for bituminous coal for the U.S. Geological Survey (USGS) database [5]. In the first round, the average mercury concentration for the medium volatile bituminous samples (0.130 ppm) is about 50% higher than the average for all samples analyzed in the first round. The average mercury concentration for the medium volatile bituminous samples in the second round (0.109 ppm) is also about 50% higher than the average for all samples analyzed in the second round. No low volatile bituminous coal samples were analyzed in the second round. In summary, the mercury concentration for bituminous coal samples are similar for the two rounds; the overall average mercury concentration for the 106 bituminous coal samples analyzed in the two rounds is 0.087 ppm as compared to an average of 0.092 ppm for the 87 bituminous samples analyzed in the first round. The overall average value for bituminous coal is approximately one half of the value reported in the USGS database for bituminous coal.

In the first round, the average mercury concentration for the subbituminous samples (0.044 ppm) was about 50% lower than the average for all samples analyzed in the first round. The average mercury concentration for the subbituminous samples in the second round (0.055 ppm) was about a third lower than the average for all samples analyzed in the second round. The overall average mercury concentration for the 37 subbituminous coal samples analyzed in the two rounds (0.053 ppm) is slightly higher than the 0.044 ppm average for the 8 subbituminous samples analyzed in the first round.

In the first round, the average concentration for the six (three North Dakota and three Texas) lignite samples (0.095 ppm) was not substantially different from the average of all samples analyzed in the first round. In the second round, however, the average concentration for the five (Texas) lignite samples (0.274 ppm) was more than three times the overall average. The overall average mercury concentration for the 11 lignite samples analyzed in the two rounds (0.177 ppm) is considerably higher than the 0.095 ppm average for the lignite samples analyzed in the first round and is also considerably higher than the average for all bituminous (0.087 ppm) and subbituminous (0.053 ppm) coal samples analyzed in the two rounds.

Potential uncontrolled mercury emission factors by state of origin of samples. Utilities were asked to provide short proximate analysis data (moisture, sulfur, ash, and Btu) for samples submitted to the EPRI mercury in coal study. The Btu content of the sample was reported for 153 of the 154 samples analyzed in the two study rounds. The reported as-received Btu values were used to calculate potential uncontrolled emission factors in lb $Hg/10^{12}$ Btu for these 153 samples. These factors are presented in Table 5 and Figure 8 by state of origin of the samples.

Table 5. Mercury Potential Uncontrolled Emission Factors (lb Hg/10^{12} Btu) by State of Origin of Samples

		lb Hg/10^{12} Btu			
State	Number of Samples	Minimum	Maximum	Mean	Std. Dev.
AL	8	0.73	21.23	6.13	6.28
AZ	2	2.40	2.84	2.62	0.22
CO	3	1.45	6.39	3.15	2.29
IL	13	1.09	9.04	5.46	2.00
IN	6	0.93	14.04	6.89	4.26
KY	17	3.83	26.70	8.46	5.21
MT	3	2.48	7.56	4.38	2.26
NM	15	1.18	10.36	4.40	2.20
ND	3	9.91	13.86	11.91	1.61
OH	4	8.71	9.68	9.23	0.39
PA	14	3.34	33.27	12.56	9.34
TN	2	3.40	9.69	6.54	3.14
TX	8	13.97	69.00	33.65	20.75
UT	7	1.03	3.38	1.86	0.80
VA	6	3.00	9.10	6.21	1.85
WA	2	1.82	3.04	2.43	0.61
WV	18	2.43	12.68	6.57	2.92
WY	22	1.80	11.27	6.95	2.84
TOTAL	153	0.73	69.00	8.21	9.07

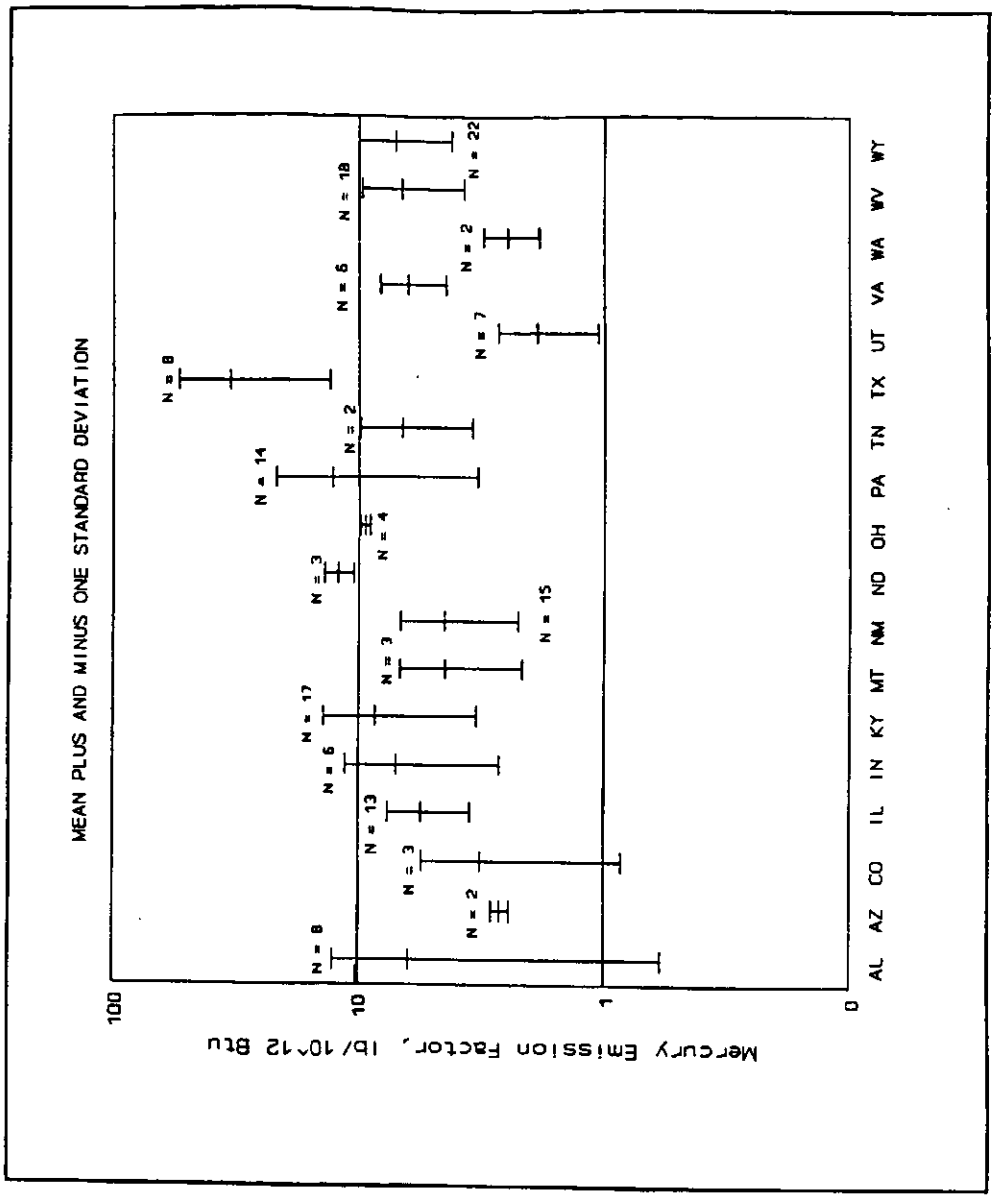

Figure 8. **Mercury emission factors by state of origin of samples — combined first and second round data.**

Across states, the average potential uncontrolled emission factors range from 1.86 lb Hg/10^{12} Btu for the seven samples of coal from Utah, to 33.65 lb Hg/10^{12} Btu for the eight Texas lignite samples. Although the mercury concentration for the Texas lignite samples is higher than the concentration for the samples from the other states by a factor ranging from about 1.4 to 11 (see Table 3), when the lower Btu content of lignite is taken into account it can be seen from Table 5 and Figure 8 that the potential uncontrolled emission factor for the Texas lignite samples (33.65 lb Hg/^{12} Btu) is higher by a factor ranging from about 2.7 to 18 than the emission factor for the samples from the other states.

Potential uncontrolled mercury emission factors by coal rank. Table 6 shows the mercury emission factors for the dif erent ranks of coal. The emission factor for the 11 (three North Dakota and eight Texas) lignite samples (27.72 lb Hg/10^{12} Btu) is almost four times the factor for the bituminous coal samples and about five times the factor for the subbituminous coal samples. The data in Table 6 are shown in Figure 9.

Table 6. Mercury Potential Uncontrolled Emission Factors (lb Hg/10^{12} Btu) by Coal Rank

		lb Hg/10^{12} Btu			
Coal Rank	Number of Samples	Minimum	Maximum	Mean	Std. Dev.
Bituminous					
Low volatile	7	0.73	6.62	4.03	2.13
Medium volatile	12	1.45	30.33	10.43	8.17
High volatile	86	1.03	33.27	6.83	5.04
All bituminous	106	0.73	33.27	7.05	5.54
Subbituminous	37	0.93	11.27	5.70	2.92
Lignite	11	9.91	69.00	27.72	20.19
All ranks	**153**	**0.73**	**69.00**	**8.21**	**9.07**

Seam-weighted mercury concentrations and potential uncontrolled emission factors. Utilities were asked to report the seam for coal samples submitted to the EPRI mercury in coal study. This information was used in calculating seam-weighted concentrations and potential uncontrolled emission factors for samples from the various states. Seam "weighting factors" were derived as follows. Total tonnage production, and percentage breakdown by seam, are reported in the Keystone Coal Industry Manual for many coal mines [6]. The tonnage production by seam was tabulated for a subset of the mines in each state, as illustrated in the following tabulation.

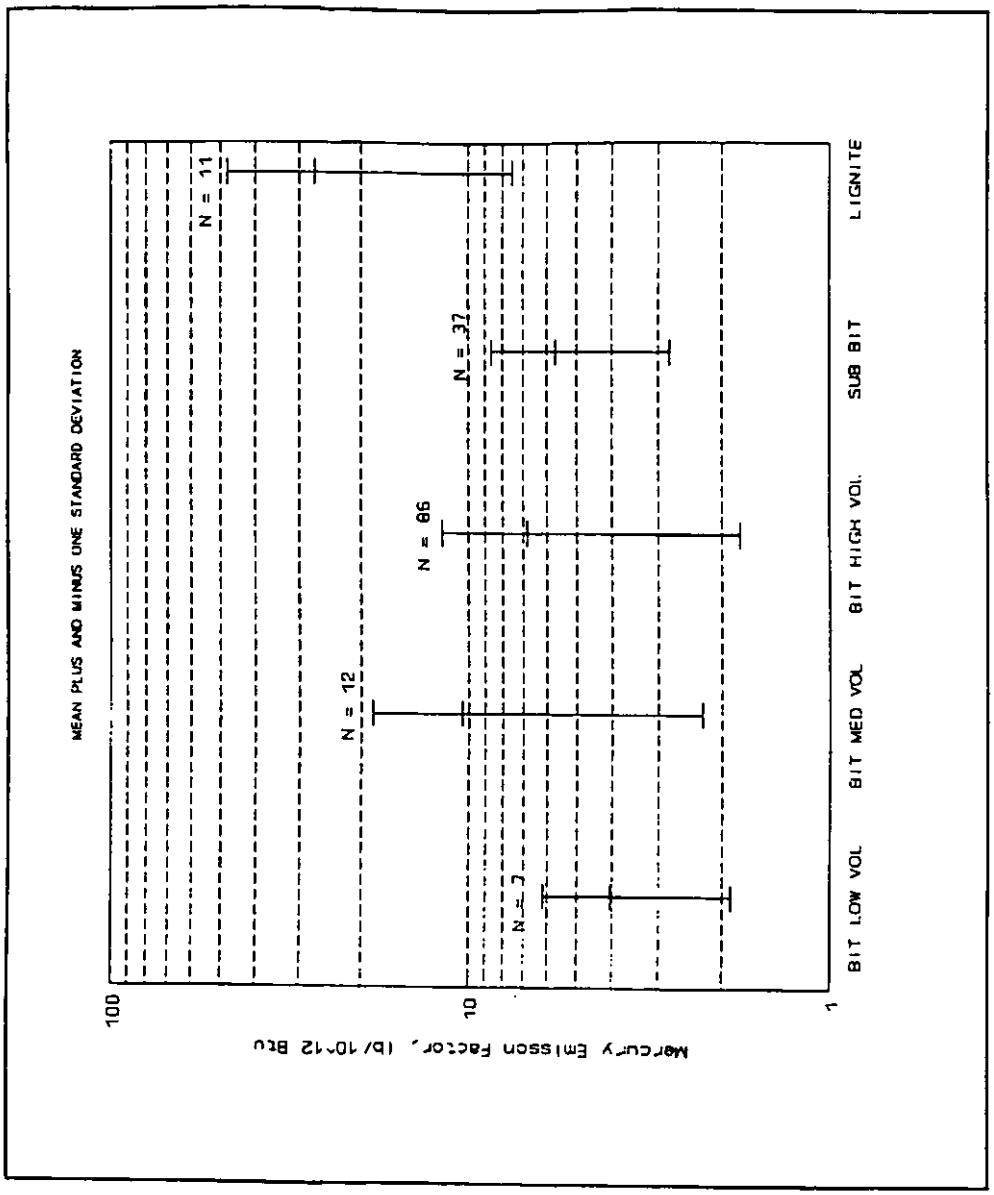

Figure 9. **Mercury potential uncontrolled emission factors by coal rank — combined first and second round data.**

Illustration of Derivation of Seam Weighting Factors

	Tonnage Production		Seam A	Seam B	Seam C	Seam D
Mine A	% by seam		100%			
	Total	10,000	10,000			
Mine B	% by seam		50%	50%		
	Total	20,000	10,000	10,000		
Mine C	% by seam			50%	25%	25%
	Total	30,000		15,000	7,500	7,500
Seam Tonnage		60,000	20,000	25,000	7,500	7,500
Weighting Factor			0.333	0.417	0.125	0.125

Seam weighting factors were calculated by dividing the total tonnage production for the seam (e.g., 20,000 T for seam A in the illustration) by the total tonnage production for all seams for the mines in the subset (e.g., 60,000 T in the illustration).

Data on the percentage breakdown of production by seam was not available for all mines in all states, and for some states there were too many mines to tabulate the production from all mines. Therefore, the tonnage production by seam was tabulated for only a subset of the mines in most states. The subset included all of the mines listed in the table of major U.S. coal mines in the Keystone Manual, along with numerous others. Table C1 in Appendix C of this report shows the seams represented by samples from each state. Table C2 in Appendix C shows the seam weighting factors and the "percentage of production surveyed" for each state. The column in Table C1 headed "Percentage of Production Surveyed," shows the total tonnage production for the mines in the subset divided by the total tonnage production for all mines in the state, expressed as a percent. This value indicates the percentage of the total tonnage coal production for the state that was used in calculating the seam weighting factors and thus provides a general indication of the margin for error in the factors.

Seam-weighted average mercury concentrations and potential uncontrolled emission factors were calculated according to the following equation:

$$Hg_w = \frac{\sum_{i=1}^{n} Hg_i \times F_i}{\sum_{i=1}^{n} F_i} \qquad \text{Eq. (1)}$$

Where:

Hg_w	=	seam-weighted average mercury concentration or emission factor, in ppm or lb $Hg/10^{12}$ Btu
Hg_i	=	mercury concentration or emission factor for the ith sample, in ppm or lb $Hg/10^{12}$ Btu
F_i	=	weighting factor for the ith sample.

Seam-weighted average mercury concentrations and potential uncontrolled emission factors are shown by state in Table 7. For purposes of comparison, Table 7 also includes unweighted concentrations from Table 3 and unweighted emission factors from Table 5.

In calculating the unweighted values, results for replicate samples of the same coals were treated as separate values, as opposed to being averaged prior to calculation of the state average. For purposes of comparison, Table 7 also includes values in the column headed "Replicates Averaged" that were calculated by averaging the results for replicate samples of the same coal and then calculating the state average. It can be seen from Table 7 that averaging the results for replicate samples prior to calculating the state average usually results in almost the same mean value for the state. Seam weighting, however, sometimes results in a significantly different mean concentration and potential uncontrolled emission factor for the state. For example, the unweighted concentration and emission factor for Alabama are 0.071 ppm and 6.13 lb $Hg/10^{12}$ Btu; whereas, the seam-weighted concentration and emission factor are substantially lower (0.043 ppm and 2.90 lb $Hg/10^{12}$ Btu). The unweighted concentration and emission factor for Pennsylvania are 0.159 ppm and 12.5 lb $Hg/10^{12}$ Btu; whereas, the seam-weighted concentration and emission factor are also substantially lower (0.109 ppm and 8.28 lb $Hg/10^{12}$ Btu).

Effect of coal cleaning on mercury concentrations and potential uncontrolled emission factors. Utilities were asked to report the ash content and extent of cleaning for coal samples submitted to the EPRI mercury in coal study. Table 8 summarizes this information by coal rank. The partially cleaned category in Table 8 includes samples consisting of some cleaned and some uncleaned coal, as well as samples where the coal was not fully cleaned. The lb $Hg/10^{12}$ Btu and ash data ($\pm$ 1 std. dev.) in Table 8 are shown in Figure 10. It can be seen that while ash content and potential uncontrolled mercury emission factor decrease with increasing extent of coal cleaning, there is a large amount of variability in the relationship, as indicated by the length of the standard deviation bars in Figure 8.

Comparison of results by other laboratories. Mercury concentrations determined by other laboratories were available for 22 of the samples analyzed in the first round and for 10 of the samples analyzed in the second round. In the first round, the mercury concentrations measured by FGS (0.084 ppm) averaged 0.036 ppm higher than the concentrations measured by the other laboratories (0.048 ppm). In the second round, however, the FGS results and the results for the other laboratories were virtually the same: FGS 0.056 ppm, other laboratories 0.054 ppm.

Table 7. Seam-Weighted Mercury Concentrations and Potential Uncontrolled Emission Factors (lb Hg/10^{12} Btu) for Combined Data

	All Samples			Replicates Averaged			Seam-Weighted		
State	Number of Samples	ppm	lb/10^{12} Btu	Number of Values	ppm	lb/10^{12} Btu	Number of Values	ppm	lb/10^{12} Btu
AL	9	0.071	6.13[a]	6	0.080	7.22	11	0.043	2.90
AZ	2	0.029	2.62	1	0.029	2.62	1	0.029	2.62
CO	3	0.034	3.15	3	0.034	3.15	4	0.063	5.94
IL	13	0.063	5.46	11	0.062	5.35	12	0.058	5.12
IN	6	0.077	6.89	5	0.061	5.46	10	0.065	5.79
KY	17	0.106	8.46	15	0.109	8.73	23	0.105	8.36
MT	3	0.038	4.38	3	0.038	4.38	3	0.041	4.76
NM	15	0.044	4.40	13	0.043	4.39	Insufficient information for calculation		
ND	3	0.075	11.91	1	0.075	11.91	1	0.075	11.91
OH	4	0.113	9.23	2	0.110	9.00	2	0.116	9.29
PA	14	0.159	12.56	12	0.174	13.74	13	0.109	8.28
TN	2	0.088	6.54	2	0.088	6.54	2	0.107	7.91
TX	8	0.215	33.65	6	0.248	39.24	7	0.308	49.64
UT	7	0.022	1.86	6	0.023	1.94	10	0.022	1.85
VA	6	0.081	6.21	5	0.079	6.21	9	0.085	6.09
WA	2	0.019	2.43	1	0.019	2.43	2	0.019	2.43
WV	18	0.084	6.57	15	0.089	6.87	19	0.093	7.21
WY	22	0.064	6.95	20	0.065	7.02	20	0.069	7.48
TOTAL	154	0.085	8.21	127	0.088	8.46	149	0.087	8.39

Table 8. Mercury Concentrations and Emission Factors by Coal Rank for Uncleaned, Partially Cleaned, and Fully Cleaned Samples

Coal Rank	Not Cleaned				Partially Cleaned				Fully Cleaned			
Bituminous	Samples	Ash (%)	ppm	lb/10^{12} Btu	Samples	Ash (%)	ppm	lb/10^{12} Btu	Samples	Ash (%)	ppm	lb/10^{12} Btu
Low volatile									7	9.19	0.054	4.03
Medium volatile	6	12.37	0.170	14.31	1	8.82	0.077	6.89	3	9.05	0.082	6.58
High volatile	13	9.24	0.037	3.05	13	10.16	0.101	8.09	43	8.78	0.094	7.52
All bituminous	19	10.22	0.079	6.62	14	10.06	0.100	8.00	53	8.85	0.088	7.01
Subbituminous	25	5.91	0.061	6.65					2	12.70	0.019	2.43
Lignite	11	12.8	0.177	27.72								
All ranks	55	8.78	0.090	10.85	14	10.06	0.100	8.00	55	8.99	0.086	6.84

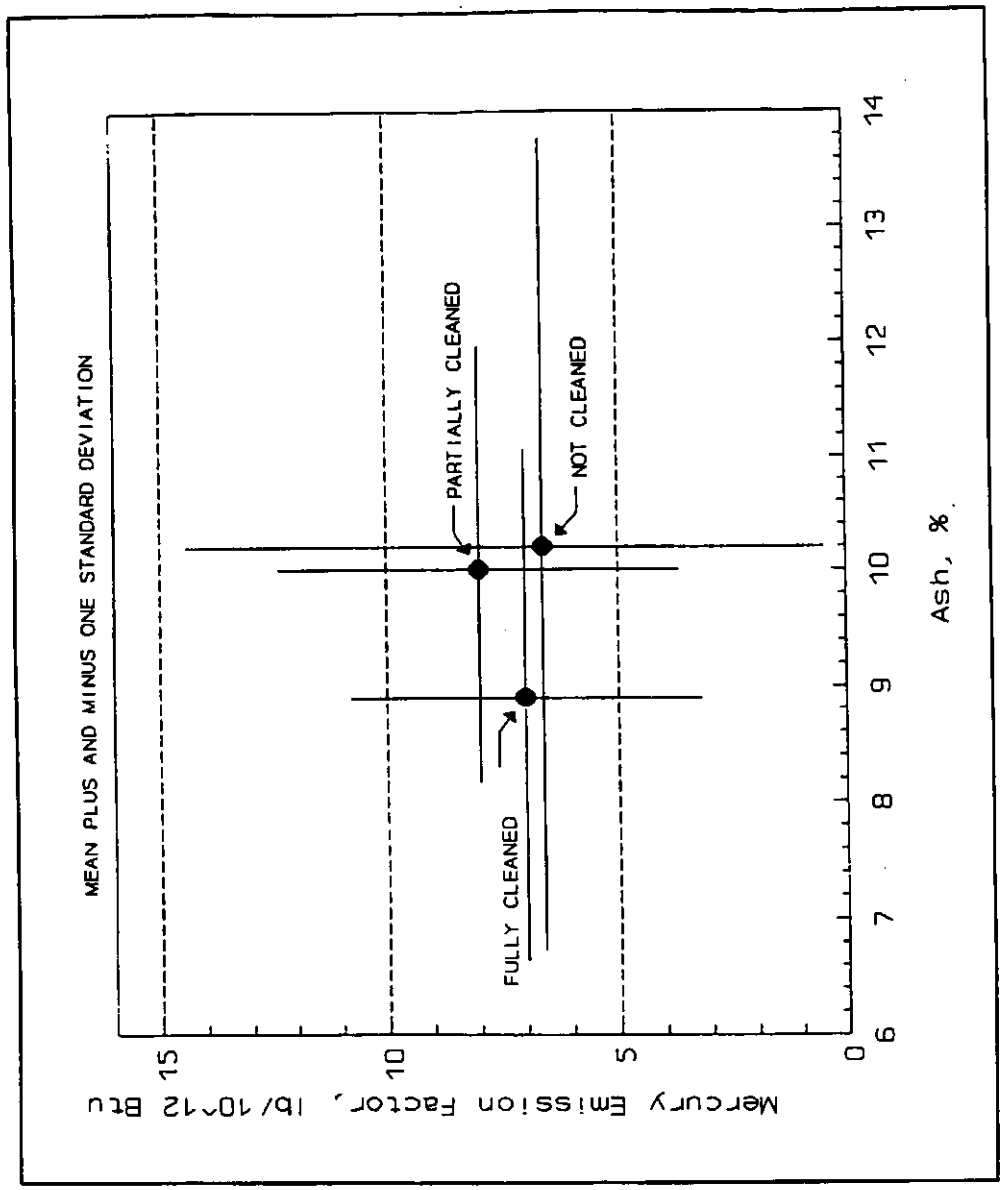

Figure 10. **Mercury potential uncontrolled emission factors for bituminous coal related to extent of coal cleaning — combined first and second round data.**

Over the two rounds, the mercury concentrations measured by other laboratories ranged from <0.02 ppm (the method detection limit) to 0.11 ppm, averaging 0.050 ppm. The mercury concentrations measured by FGS for the same samples ranged from 0.009 ppm to 0.276 ppm, averaging 0.075 ppm, or 0.025 ppm higher than the average for the other laboratories. The median difference between the FGS results and the results by other laboratories was 0.0185 ppm.

ASTM Method D 3684 (Standard Test Method for Total Mercury in Coal by the Oxygen Bomb Combustion/Atomic Absorption Method) was the method used to determine the mercury concentration for 28 of the 32 samples analyzed by other laboratories. The analytical method was not reported for the other 4 samples analyzed by other laboratories. The average mercury concentration was 0.075 for the 28 samples that were analyzed by ASTM Method D 3684. The average mercury concentration was 0.078 for the 4 samples for which the method was not reported; the average concentration measured by FGS for these 4 samples was virtually the same, 0.077 ppm. These results are summarized in Table 9. There is no readily apparent reason why the FGS measurements are higher, on average, than the results reported by other laboratories that used ASTM Method D 3684.

Table 9. Comparison of Mercury Concentration Measurements by FGS and Other Laboratories

	First Round			Second Round			Combined		
		Hg ppm			Hg ppm			Hg ppm	
Method	N	FGS	Others	N	FGS	Others	N	FGS	Others
D3684	18	0.086	0.041	10	0.056	0.054	28	0.075	0.046
Unknown	4	0.077	0.078	--	--	--	4	0.077	0.078
Combined	22	0.084	0.048	10	0.056	0.054	32	0.075	0.050

Tabulation of results in Appendix D. Analytical results for all samples, along with the state, county, and seam(s) of origin of the samples, are listed in Table D1 in Appendix D to this report. Replicate samples are identified in Table D1 by a sample ID number ending in 2 or 3. The rank of the samples (as classified by the submitter) is identified by the alphabetical characters in the ID number according to the following codes:

BHA - Bituminous high volatile A
BHB - Bituminous high volatile B
BHC - Bituminous high volatile C
BHAB - Mixed bituminous high volatile A and B
BL - Bituminous low volatile
BM - Bituminous medium volatile
LA - Lignite A
LS - Limestone
SA - Subbituminous A
SB-C - Mixed subbituminous B and C
SC - Subbituminous C

References

[1] Bloom, N.S., et al. 1993. Determination of Mercury in Fossil Fuels at the PPB Level by Cold Vapor Atomic Fluorescence Spectrometry, after Total Wet Oxidation with Perchloric Acid in Teflon Microwave Digestion Bombs. Conference on Managing Air Toxics: State of the Art. Washington, DC.

[2] Bloom, N.S. 1993. Survey of Mercury in American Coals. Internal Report to EPRI. Submitted for Publication.

[3] Bloom, N.S. 1994. Survey of Mercury in American Coals (Addendum). Internal Report to EPRI. Submitted for Publication.

[4] American Society for Testing and Materials (ASTM). 1992. Annual Book of ASTM Standards, Vol. 05.05. D 388: Standard Classification of Coals by Rank. Philadelphia, PA.

[5] Based on data in the U.S. Geological Survey National Coal Resources Data System presented by D.M. White et al. in EPA-600/7-84-066 as cited by R.C. Mead et al. in Summary of Trace Emissions from and Recommendations of Risk Assessment Methodologies for Coal and Oil Combustion Sources (1986).

[6] 1992 Keystone Coal Industry Manual. 1992. Maclean Hunter Publishing Co. Chicago, IL.

APPENDIX A
Number and Tonnage Production of Mines in Database

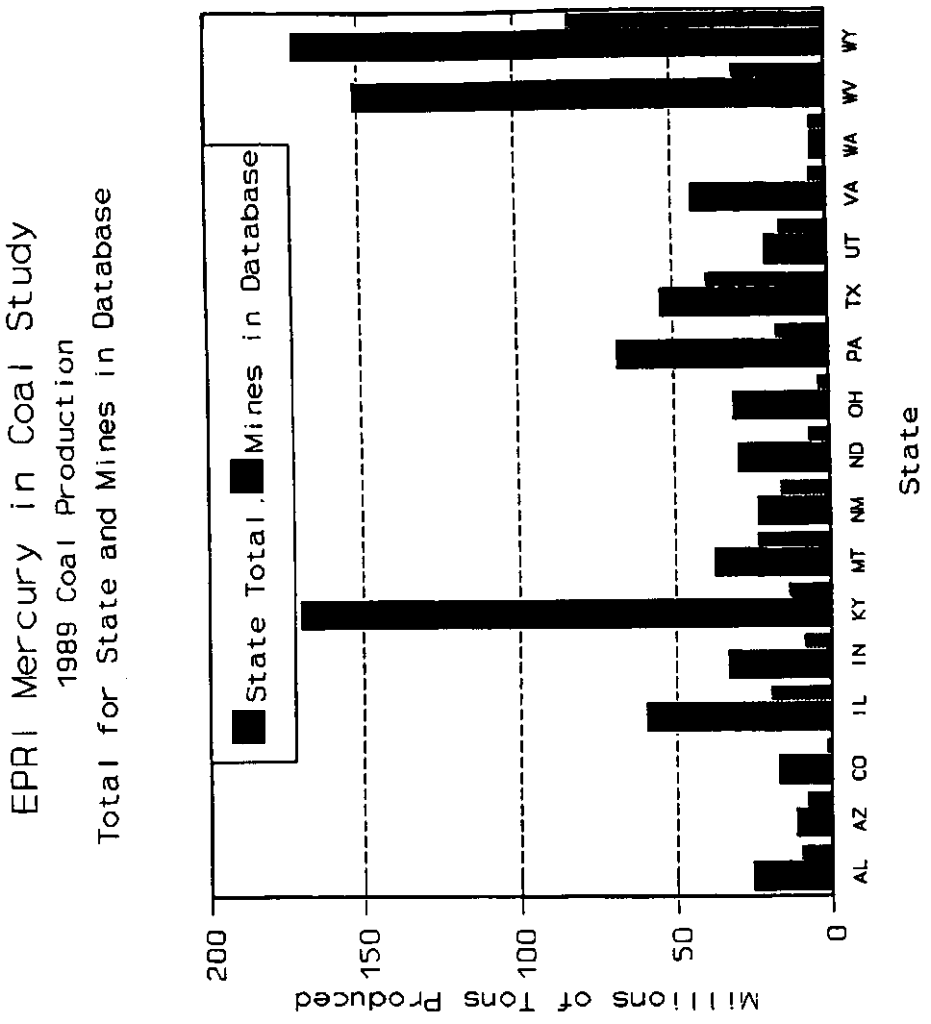

Figure A1. 1989 Tonnage Coal Production for Mines in Database

A-1

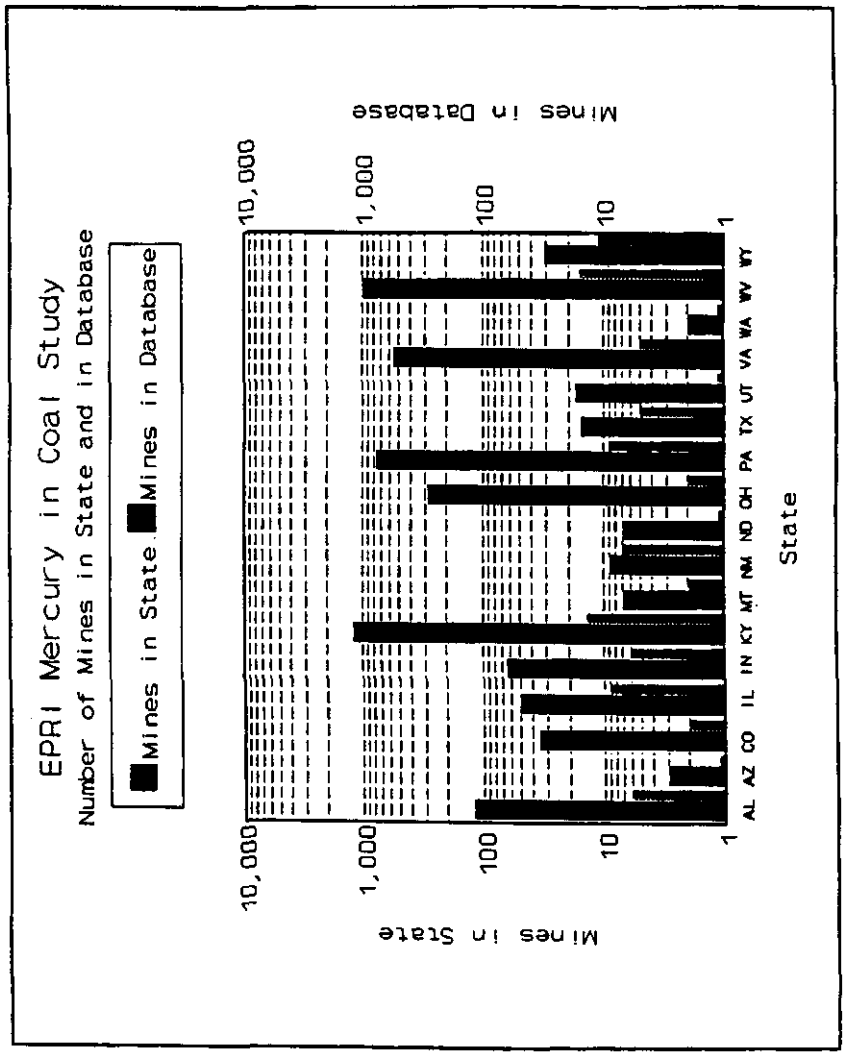

Figure A2. Number of Mines Represented in Database

A-2

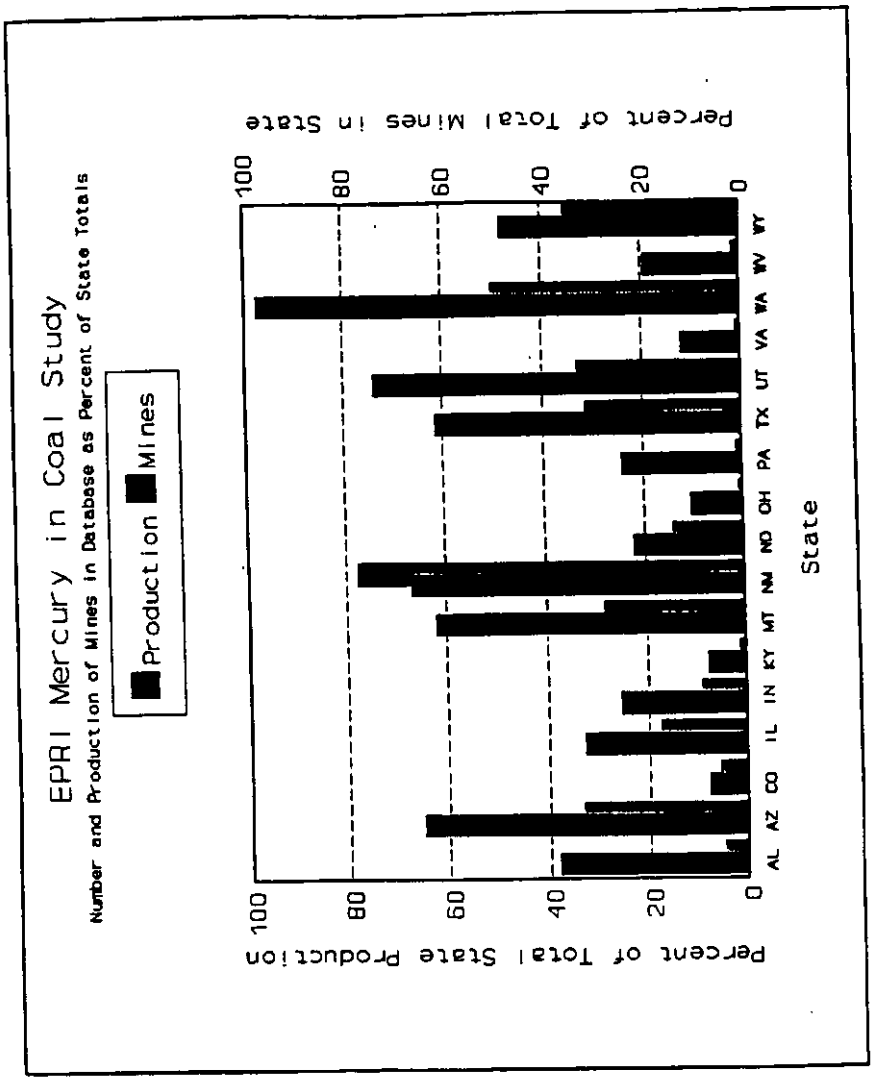

Figure A3. Number and Production of Mines Represented in Database as a Percentage of Totals for States

A-3

APPENDIX B
ASTM D388 Classifications of Coal by Rank

Table B1. ASTM D 388 Classifications of Coal by Rank

Coal Rank	Fixed Carbon[a]	Volatile Matter[a]	Btu/lb[b]	Agglomerating Character
Bituminous				
Low volatile	78% to 86%	14% to 22%		
Medium volatile	69% to 78%	22% to 31%		
High volatile A	< 69%	> 31%	> 14,000	Commonly agglomerating
High volatile B			13,000 to 14,000	Commonly agglomerating
High volatile C			11,500 to 13,000	Commonly agglomerating
			10,500 to 11,500	Agglomerating
Subbituminous			8,300 to 11,500	Nonagglomerating

a Dry, mineral-matter-free basis.
b Moist, mineral-matter-free basis.

APPENDIX C
Seams and Seam Weighting Factors

Table C1. Seams Represented by Samples

State	Seams							
AL	American	Black Creek	Blue Creek	Mary Lee	New Castle	Nickel Place	Pratt	
AZ	Blue	Brown	Green	Orange				
CO	B	Somerset	Wedge	Williams Fork				
IL	No. 5	No. 6						
IN	No. 5	No. 6	Lower Block	Upper Block	Staunton			
MT	Anderson	Rosebud						
NM	Fruitland	La Plata No. 1	La Plata No. 2	Pinion No. 8	Pinion No. 2	Left Fork	York Canyon	
ND	Hagel							
OH	Pittsburgh	Waynesburg						
PA	Clarion	Lower Kittanning	Middle Kittanning	Upper Kittanning	Pittsburgh	Sewickley		
TN	Jellico	Sewanee						
UT	Gilson	Hiawatha	Lower O'Connor	Upper O'Connor	Wattis			
VA	Clintwood	Lower Sandiford	Upper Sandiford	Lower Splint	Owl	Pocahontas No. 3	Upper Wilson	
WA	Big Dirty	Smith						
WV	Redstone	Coalburg	Hernshaw	Cedar Grove	Lower Kittanning	No. 5 Block	Pearlers	Pittsburgh
	Upper Freeport	Upper Stockton	Winifrede	Alma				
WY	62/63/65	Deadman	Smith	Wyodak	Wyodak-Anderson			

(Continued)

Table C1. Seams Represented by Samples (Continued)

State	Seams										
KY	Alma	Block	Clarion	Coalburg	Elkhorn	Hazard No.4	Path Fork	Princess No. 3	Stockton		
	Upper Harlan	W. Kentucky No. 9	W. Kentucky No. 11-14								
TX[a]	Freestone	Harrison	Hopkins	Milam	Panola	Rusk	Titus				

[a] County.

Table C2. Seam Weighting Factors

State	Percentage of Production Surveyed	Seam and Weighting Factor*								
AL	52.5%	American	Black Creek	Blue Creek	Mary Lee	New Castle	Nickel Place	Pratt		
		0.001	0.001	0.343	0.080	0.007	0.001	0.070		
CO	91.5%	B	Somerset	Wedge	Williams Fork					
		0.029	0.001	0.296	0.001					
IL	82.9%	No. 5	No. 6							
		0.190	0.733							
IN	62.7%	No. 5	No. 6	No. 7	Lower Block	Upper Block	Stanton			
		0.213	0.268	0.047	0.027	0.018	0.001			
MT	100.0%	Anderson	Rosebud							
		0.170	0.550							
OH	58.2%	Pittsburgh	Waynesburg							
		0.267	0.001							
PA	44.6%	Clarion	Lower Kittanning	Middle Kittanning	Upper Kittanning	Pittsburgh	Sewickley			
		0.016	0.086	0.021	0.037	0.679	0.001			
TN	26.9%	Jellico	Sewanee							
		0.081	0.032							
UT	48.4%	Gilson	Hiawatha	Lower O'Connor	Upper O'Connor	Wattis				
		0.077	0.432	0.082	0.192	0.134				
VA	59.2%	Clintwood	Lower Sandford	Upper Sandford	Lower Splint	Owl	Pocahontas No. 3	Upper Wilson		
		0.041	0.003	0.003	0.022	0.001	0.425	0.007		
WY	99.6%	62/63/65	Deadman	Smith	Wyodak	Wyodak-Anderson				
		0.006	0.070	0.186	0.271	0.220				

* Weighting factor of 0.001 indicates that the seam represents <0.1% of total production for state, based on percentage of production surveyed.

Table C2. Seam Weighting Factors (Continued)

State	Percentage of Production Surveyed	Seam and Weighting Factor[a]							
KY	36.1%	Alma	Block	Clarion	Coalburg	Elkhorn	Hazard No.4	Path fork	Princess No. 3
		0.075	0.013	0.013	0.039	0.036	0.140	0.001	0.001
		Stockton	Upper Harlan	W. Kentucky No. 9	W. Kentucky No. 11-14				
		0.027	0.044	0.145	0.341				
TX[b]	99.9%	Freestone	Harrison	Hopkins	Milam	Panola	Rusk	Titus	
		0.236	0.052	0.419	0.120	0.138	0.138	0.218	

[a] Weighting factor of 0.001 indicates that the seam represents <0.1% of total production for state, based on percentage of production surveyed.
[b] County.

APPENDIX D
Data Listing

	SAMPLE I.D. No.	Hg ppm Avg	Sample Origin			As-Received Short Proximate Analysis			
			State	County	Seam(s)	Ash (%)	Moisture (%)	Sulfur (%)	Btu/lb
1	BHA020101	0.108	West Virginia	Barbour	Redstone	8.44	4.64	1.56	13,246
2	BHA020301	0.037	West Virginia	Mingo	Lower Cedar Grove/Upper Alma	8.68	5.54	0.74	13,056
3	BHA020401	0.168	West Virginia	Barbour	Lower Kittanning	9.19	5.45	1.16	13,249
4	BHA020601	0.079	West Virginia	Braxton	Lower Kittanning	8.30	4.29	0.88	13,248
5	BHA040201	0.051	West Virginia	Braxton	Lower Kittanning	8.49	6.96	0.84	12,961
6	BHA040202	0.048	West Virginia	Braxton	Lower Kittanning	8.21	7.22	0.83	12,914
7	BHA050101	0.038	Illinois	Washington	Illinois No. 6	10.55	12.97	3.01	10,845
8	BHA050201	0.075	Illinois	Perry	Illinois No. 5 and 6	10.42	12.57	2.97	10,980
9	BHA050301	0.343	Kentucky	Perry	Hazard No. 4A	8.23	6.22	0.79	12,848
10	BHA050401	0.063	Kentucky	Letcher	Hazard No. 4A	10.93	8.16	0.72	11,996
11	BHA050501	0.099	Indiana	Clay	Upper and Lower Brazil Block	8.17	13.78	2.32	11,521
12	BHA050601	0.013	Illinois	Jefferson	Illinois No. 6	4.63	12.82	0.52	11,962
13	BHA080201	0.146	Pennsylvania	Washington	Pittsburgh	5.62	5.45	1.23	13,514
14	BHA080301	0.076	Pennsylvania	Greene	Pittsburgh	6.92	4.96	1.79	13,444
15	BHA090101	0.078	West Virginia	Boone	Kittanning	11.04	6.32	0.73	12,233
16	BHA110101	0.085	Kentucky	Martin	Clarion/Block/Coalburg/Stockton	9.38	6.87	0.95	12,186
17	BHA110102	0.095	Kentucky	Martin	Clarion/Block/Coalburg/Stockton	12.00	6.09	0.93	11,929
18	BHA110201	0.118	Kentucky	Harlan	Upper Harlan/Path Fork	10.59	6.57	1.20	12,451
19	BHA110301	0.180	Kentucky	Harlan	Upper Harlan/Path Fork	10.47	6.04	1.20	12,554
20	BHA110401	0.115	Kentucky	Harlan	Upper Harlan/Path Fork	11.21	5.59	1.31	12,364
21	BHA120501	0.114	Kentucky	Pike,Greenup	Alma/Princess No. 3	7.85	10.92	1.48	11,865
22	BHA120601	0.102	Alabama	Walker	Mary Lee	14.10	7.43	0.94	11,824
23	BHA120602	0.087	Alabama	Walker	Mary Lee	14.11	8.05	0.94	11,829
24	BHA121301	0.121	West Virginia	Wayne, Lincoln, Mingo, Logan	Coalberg/No. 5 Block/Winifrede	10.61	7.51	0.90	11,848
25	BHA121401	0.088	Kentucky	Harlan	Hazard No. 4	8.60	7.46	1.32	12,624
26	BHA121501	0.072	Kentucky	Pike	Elkhorn No. 3	7.58	7.34	1.59	12,754
27	BHA121502	0.065	Kentucky	Pike	Elkhorn No. 3	6.89	7.54	1.57	12,873
28	BHA121801	0.089	Illinois	Gallatin	Illinois No. 5	8.84	9.42	2.81	11,984
29	BHA121802	0.072	Illinois	Gallatin	Illinois No. 5	9.93	9.47	3.07	11,798
30	BHA122001	0.121	Kentucky	Knott	Elkhorn No. 2 and 3	10.54	5.03	1.49	12,581
31	BHA122401	0.107	Virginia	Wise, Lee	Multiple-Seam Blend	11.45	5.94	1.66	12,448
32	BHA122601	0.049	Kentucky	Harlan	Hazard No. 4	9.50	5.41	1.41	12,800
33	BHA122701	0.276	Alabama	Walker, Jefferson	Pratt/Nickel Place/American/New Castl	10.27	4.06	2.15	13,000
34	BHA123201	0.072	Kentucky	Hopkins	W. Kentucky No. 11, 12, 13, and 14	10.57	8.84	2.97	11,679
35	BHA123301	0.051	Kentucky	Leslie	Hazard No. 4	8.53	4.96	1.37	12,945
36	BHA123901	0.066	Illinois	Franklin	Illinois No. 6	9.31	9.12	2.33	11,746
37	BHA124701	0.025	Alabama	Walker, Cullman	Mary Lee/Blue Creek/Black Creek	•••	•••	•••	•••
38	BHA126201	0.111	Virginia	Wise	Upper and Lower Sandiford	7.64	5.68	1.40	12,192
39	BHA126301	0.089	Virginia	Wise, Lee	Upper Wilson/Lower Splint	12.75	6.54	1.45	12,242
40	BHA126401	0.040	Virginia	Wise	Upper Sandiford/Lower Splint/Owl	8.31	5.46	0.73	13,201

	SAMPLE I.D. No.	Hg ppm Avg	Sample Origin			As-Received Short Proximate Analysis			
			State	County	Seam(s)	Ash (%)	Moisture (%)	Sulfur (%)	Btu/lb
41	BHA140101	0.030	West Virginia	Boone	Upper Stockton	11.76	4.61	0.76	12,325
42	BHA140102	0.044	West Virginia	Boone	Upper Stockton	9.17	4.52	0.78	12,792
43	BHA150501	0.092	Pennsylvania	Washington	Pittsburgh No. 8	6.25	6.56	1.51	13,115
44	BHA160101	0.097	Pennsylvania	Greene	Pittsburgh No. 8	6.50	7.07	1.48	13,017
45	BHA160102	0.098	Pennsylvania	Greene	Pittsburgh No. 8	6.25	7.02	1.47	13,020
46	BHA160201	0.052	New Mexico	Colfax	Upper Left Fork/York Canyon	12.31	4.83	0.53	12,523
47	BHA160202	0.061	New Mexico	Colfax	Upper Left Fork/York Canyon	11.83	4.52	0.53	12,576
48	BHA160203	0.053	New Mexico	Colfax	Upper Left Fork/York Canyon	12.38	3.83	0.55	12,307
49	BHA160301	0.057	Illinois	Franklin, Jefferson	Illinois No. 6	6.35	11.10	1.65	11,909
50	BHA200201	0.014	New Mexico	San Juan	La Plata No. 1	12.21	6.72	0.51	11,860
51	BHA210101	0.084	Kentucky	Pike	Elkhorn	10.95	4.67	1.10	12,798
52	BHA210301	0.123	West Virginia	Boone	Pearless	7.53	5.00	1.47	13,384
53	BHA220301	0.091	Pennsylvania	Greene	Pittsburgh No. 8	6.30	7.00	1.60	13,000
54	BHA220501	0.051	Venzuela	Unknown	Unknown	5.60	6.50	0.53	13,120
55	BHA260101	0.118	West Virginia	Monongalia	Pittsburgh No. 8	11.05	4.89	2.50	12,779
56	BHA260201	0.106	West Virginia	Monongalia	Pittsburgh No. 8	7.78	6.07	2.03	13,145
57	BHA340101	0.112	Ohio	Belmont	Pittsburgh No. 8	9.41	6.30	4.22	12,486
58	BHA340102	0.117	Ohio	Belmont	Pittsburgh No. 8	6.77	10.24	4.73	12,254
59	BHA340103	0.119	Ohio	Belmont	Pittsburgh No. 8	9.59	7.03	4.25	12,366
60	BHA370101	0.029	Utah	Carbon	Lower Sunnyside/Gilson	11.10	7.57	0.56	11,598
61	BHA370201	0.015	Utah	Emery	Hiawatha	6.97	6.31	0.50	12,748
62	BHA370301	0.038	Utah	Sevier	Upper Hiawatha	8.41	10.91	0.35	11,256
63	BHA370401	0.016	Utah	Carbon	Wattis	8.56	9.29	0.43	12,045
64	BHA370501	0.016	Utah	Carbon	Upper and Lower O'Connor	8.87	8.63	0.38	11,721
65	BHAB250201	0.043	Tennessee	Sequatchie	Sewanee	11.80	5.10	0.72	12,651
66	BHAB250301	0.132	Tennessee	Scott	Jellico	4.60	5.30	1.91	13,624
67	BHAB250401	0.081	Kentucky	Webster	W. Kentucky No. 9	9.00	6.30	2.96	12,610
68	BHB125901	0.019	Colorado	Gunnison	B	8.60	9.13	0.46	11,818
69	BHB126801	0.013	Utah	Emery	Hiawatha	8.94	5.64	0.47	12,214
70	BHB200101	0.071	New Mexico	San Juan	Unknown	21.02	10.70	0.85	9,531
71	BHB200301	0.089	New Mexico	San Juan	La Plata No. 2	29.51	8.12	1.03	8,594
72	BHB200401	0.029	New Mexico	San Juan	Pinion No. 8	15.97	7.36	1.05	10,456
73	BHB200501	0.036	New Mexico	San Juan	Pinion No. 9	18.45	7.16	0.70	10,294
74	BHB230101	0.055	West Virginia	Monongalia	Pittsburgh	6.01	6.38	2.44	13,339
75	BHB230201	0.440	Pennsylvania	Armstrong	Clarion	6.11	6.47	1.77	13,225
76	BHB230301	0.228	Pennsylvania	Jefferson	Lower and Middle Kittanning/ Upper and Lower Clarion	9.53	5.87	1.46	12,990
77	BHB230501	0.098	Pennsylvania	Greene	Pittsburgh No. 8	6.33	6.55	1.54	13,109
78	BHC010101	0.046	New Mexico	McKinley	Unknown	15.60	14.10	0.49	9,600
79	BHC010102	0.034	New Mexico	McKinley	Unknown	14.50	14.20	0.52	9,840
80	BHC030101	0.041	Pennsylvania	Greene	Sewickley	12.91	6.13	1.27	12,273

	SAMPLE I.D. No.	Hg ppm Avg	Sample Origin			As-Received Short Proximate Analysis			
			State	County	Seam(s)	Ash (%)	Moisture (%)	Sulfur (%)	Btu/lb
81	BHC030102	0.045	Pennsylvania	Greene	Sewickley	13.34	5.25	1.35	12,243
82	BHC070201	0.054	Illinois	Macoupin	Illinois No. 6	8.11	15.91	3.29	10,757
83	BHC170101	0.107	Illinois	Saline	Illinois No. 6	8.40	10.61	2.68	11,832
84	BHC170201	0.097	Illinois	Saline	Illinois No. 5	6.53	11.23	1.47	12,708
85	BHC170301	0.037	Illinois	Jefferson	Illinois No. 6	12.65	11.55	0.79	11,021
86	BHC360101	0.025	Utah	Carbon	Upper and Lower O'Connor	9.71	10.41	0.47	11,220
87	BHC410101	0.026	Arizona	Navajo	Brown/Orange/Blue/Green	8.11	12.52	0.50	10,931
88	BHC410102	0.031	Arizona	Navajo	Brown/Orange/Blue/Green	8.49	12.62	0.52	10,856
89	BL040301	0.082	Virginia	Buchanan	Pocahontas No. 3	4.53	5.72	0.81	14,169
90	BL040302	0.094	Virginia	Buchanan	Pocahontas No. 3	4.57	5.52	0.81	14,192
91	BL120901	0.009	Alabama	Jefferson	Blue Creek	12.65	8.49	0.49	12,253
92	BL120902	0.016	Alabama	Jefferson	Blue Creek	10.77	9.20	0.50	12,457
93	BL121601	0.050	Alabama	Tuscaloosa	Blue Creek	11.77	8.93	0.68	12,359
94	BL121602	0.048	Alabama	Tuscaloosa	Blue Creek	10.98	8.84	0.66	12,483
95	BL130101	0.077	West Virginia	Grant	Upper Freeport	9.07	6.84	1.42	13,020
96	BM080101	0.129	West Virginia	Upshur, Harrison, Barbour	Redstone	7.55	4.42	2.01	13,342
97	BM100101	0.103	Ohio	Belmont	Waynesburg No. 11	13.01	5.85	2.75	11,823
98	BM123601	0.024	Alabama	Tuscaloosa	Blue Creek	10.24	8.3	0.66	12,693
99	BM150101	0.331	Pennsylvania	Clearfield	Upper Kittanning	23.74	4.66	2.05	10,901
100	BM150201	0.295	Pennsylvania	Clearfield	Upper Kittanning	9.12	5.92	0.87	13,128
101	BM150301	0.148	Pennsylvania	Clearfield	Upper Kittanning	13.84	7.38	0.65	12,107
102	BM170501	0.017	Colorado	Gunnison	Somerset/Williams Fork	9.52	8.57	0.48	11,744
103	BM210201	0.070	Virginia	Wise	Clintwood	9.86	5.70	1.19	12,794
104	BMZ20201	0.050	West Virginia	Boone	Winifrede/Stockshaw	7.50	7.50	0.83	13,250
105	BM330101	0.159	Indiana	Greene, Owen	Multiple-Seam Blend	8.13	15.04	2.26	11,310
106	BM330201	0.092	Indiana	Warrick, Vanderburgh	Indiana No. 5, No. 6, No.7	9.37	13.71	3.07	11,198
107	BM330301	0.077	Indiana	Daviess	Lower Block/Upper Block/Staunton	8.82	14.25	2.05	11,198
108	LA190101	0.139	Texas	Harrison	Green	7.96	37.31	0.54	6,997
109	LA190102	0.094	Texas	Harrison	Green	10.79	36.43	0.63	6,731
110	LA190103	0.114	Texas	Harrison	Green	9.03	37.09	0.93	6,899
111	LA270101	0.075	North Dakota	McLean	Hagel	10.69	37.84	0.75	6,270
112	LA270102	0.063	North Dakota	McLean	Hagel	10.39	37.75	0.80	6,356
113	LA270103	0.086	North Dakota	McLean	Hagel	11.49	36.94	0.82	6,231
114	LA380101	0.236	Texas	Freestone		14.52	31.76	0.80	6,812
115	LA380201	0.138	Texas	Rusk, Panola		11.60	35.92	0.92	6,660
116	LA380301	0.393	Texas	Titus		20.54	32.22	0.49	5,864
117	LA380401	0.185	Texas	Milam		15.88	30.50	1.18	6,749
118	LA380501	0.419	Texas	Hopkins		17.86	33.07	0.49	6,066
119	SA240101	0.056	Illinois	Montgomery	Illinois No. 6	8.94	19.31	1.26	10,129
120	SA240102	0.052	Illinois	Montgomery	Illinois No. 6	8.94	19.31	1.26	10,129

	SAMPLE I.D. No.	Hg ppm Avg	Sample Origin			As-Received Short Proximate Analysis			
			State	County	Seam(s)	Ash (%)	Moisture (%)	Sulfur (%)	Btu/lb
121	SA240201	0.028	Indiana	Knox	Indiana No. 7	9.73	17.70	2.72	10,282
122	SA240301	0.010	Indiana	Sullivan	Indiana No. 6	8.18	18.75	2.14	10,353
123	SA290301	0.067	Wyoming	Carbon	62,63,65	8.03	13.29	0.41	10,270
124	SA300101	0.018	Wyoming	Campbell	Wyodak	5.36	27.97	0.50	8,559
125	SB126001	0.083	Wyoming	Campbell	Wyodak	5.14	30.22	0.32	8,406
126	SB126101	0.092	Wyoming	Campbell	Wyodak	4.79	10.23	0.72	12,355
127	SB320101	0.073	Wyoming	Sweetwater	Smith	7.36	21.12	0.48	9,580
128	SB350101	0.019	Wyoming	Campbell	Wyodak	5.63	30.74	0.35	11,914
129	SB390101	0.066	Wyoming	Campbell	Wyodak-Anderson	7.58	5.00	0.39	11,201
130	SB-C060201	0.068	New Mexico	Cibola	Fruitland	14.39	16.08	0.60	9,594
131	SB-C060301	0.028	New Mexico	Cibola	Fruitland	14.57	16.12	0.62	9,548
132	SB-C060401	0.041	New Mexico	Cibola	Fruitland	14.54	16.34	0.66	9,514
133	SB-C060501	0.027	New Mexico	Cibola	Fruitland	15.43	16.17	0.66	9,338
134	SB-C060601	0.024	New Mexico	Cibola	Fruitland	17.65	15.65	0.70	9,101
135	SC126501	0.060	Wyoming	Campbell	Wyodak	5.09	27.97	0.27	8,652
136	SC126601	0.056	Wyoming	Campbell	Wyodak	5.40	30.82	0.30	8,142
137	SC126701	0.038	Montana	Big Horn	North Extension	5.45	24.59	0.41	9,249
138	SC170401	0.029	Wyoming	Campbell	Wyodak	4.64	27.77	0.18	8,673
139	SC240401	0.065	Colorado	Routt	Wedge	9.69	16.27	2.62	10,509
140	SC280101	0.021	Montana	Rosebud	Rosebud	8.59	27.44	0.65	8,443
141	SC280201	0.065	Montana	Rosebud	Rosebud	8.90	26.09	0.64	8,595
142	SC290101	0.096	Wyoming	Campbell	Wyodak-Anderson	4.50	31.61	0.34	8,487
143	SC290201	0.047	Wyoming	Campbell	Wyodak-Anderson	4.82	26.46	0.21	8,836
144	SC310101	0.042	Wyoming	Sweetwater	Deadman	9.91	18.77	0.57	9,751
145	SC310201	0.014	Washington	Lewis	Smith/Big Dirty	13.70	24.80	0.47	7,961
146	SC310202	0.025	Washington	Lewis	Smith/Big Dirty	11.70	25.80	0.45	8,065
147	SC350101	0.055	Wyoming	Campbell	Wyodak	5.02	28.34	0.34	8,615
148	SC350201	0.077	Wyoming	Campbell	Wyodak	4.76	29.69	0.26	8,500
149	SC360201	0.081	Wyoming	Campbell	Wyodak-Anderson	4.96	26.59	0.34	8,845
150	SC360301	0.088	Wyoming	Campbell	Wyodak-Anderson	5.00	30.25	0.34	8,285
151	SC390101	0.128	Wyoming	Campbell	Wyodak	6.67	5.12	0.50	11,344
152	SC400101	0.049	Wyoming	Campbell	Wyodak-Anderson	4.83	26.82	0.39	8,869
153	SC400102	0.030	Wyoming	Campbell	Wyodak-Anderson	4.16	27.86	0.17	8,820
154	SC400201	0.057	Wyoming	Campbell	Wyodak	6.00	27.89	0.59	8,668
155	SC400202	0.085	Wyoming	Campbell	Wyodak	5.25	28.34	0.25	8,633

APPENDIX E
ALTERNATIVE EXPOSURE METHODOLOGY

The inhalation exposure assessment estimated the inhalation exposure to trace emissions from power plants for a *reasonably exposed individual* (REI) living near each power plant. The assessment incorporated available data on several key factors that affect REI exposure, including activity patterns, indoor/outdoor concentrations, and population mobility. The assessment also accounted for the replacement of aging units with units that meet EPA's Subpart Da New Source Performance Standard (NSPS) for electric utility boilers. This appendix describes the REI assessment approach and the corresponding assumptions and limitations.

E.1 Approach

E.1.1 Step 1: Definition of Exposure Scenarios

The first step defines exposure scenarios that would lead to significant levels of exposure to power plant emissions for nearby residents. Each scenario represents in aggregate many activities that are likely to occur in locations with similar concentration levels. People residing near a power plant are assumed to spend their time in one or more of the scenarios. In Exposure Scenario 1, individuals spend time within a structure located "near the power plant"— that is, within 50 km of a plant. In Exposure Scenario 2, individuals spend time outdoors near a power plant. In Exposure Scenario 3, individuals spend time more than 50 km from a power plant, and are assumed not to be exposed to significant concentrations due to plant emissions.

E.1.2 Step 2: Estimation of Concentrations Near the Plant

For each plant, the second step determines the concentrations to which individuals are exposed for the "near-plant" exposure scenarios. Outdoors near the plant (i.e., Exposure Scenario 2), the best-estimate concentration is simply the highest concentration in a populated location within 50 km of the plant.

Indoors near the plant (i.e., Exposure Scenario 1), the concentrations are adjusted by chemical-specific indoor/outdoor ratios. The ratio between indoor and outdoor concentrations of a substance due to a particular outdoor source reflects the extent to which a trace substance emitted by an outdoor source penetrates indoors. For example, an

indoor/outdoor ratio of 50% for a specific chemical indicates that the indoor concentration due to a particular outdoor source will be 50% of the outdoor concentration due to the same source. Indoor/outdoor ratios differ depending on the characteristics of the chemical and the building structure. Recent research indicates that residences have indoor/outdoor ratios of about 65% for particles [1,2]. It has been hypothesized that reactive gases have indoor/outdoor ratios similar to those of particles and that nonreactive gases have indoor/outdoor ratios of essentially 100% (i.e., complete penetration) [3].

E.1.3 Step 3: Determination of Time Spent Indoors and Outdoors Near Plant

Step 3 determines time spent in indoor and outdoor environments in the near-plant exposure scenarios. The time spent indoors near the plant is the time an individual spends indoors at home or inside another structure within 50 km of the plant (Exposure Scenario 1). The time spent outdoors near the plant is the time an individual spends outdoors at home or outdoors at another location within 50 km of the plant (Exposure Scenario 2). The two key time parameters—the average time spent in the location per day of exposure and the number of days of exposure in a lifetime—are discussed below.

The average time spent indoors and outdoors near the plant per day is estimated from information about activity patterns. Human activity patterns specify the time sequence of an individual's movement among various micro-environments (e.g., home, outdoors) and their associated level of physical activity (e.g., light exercise). Such patterns are important determinants of exposure to trace substances emitted from power plants, since the location during each activity interval determines the concentration levels of trace substances in the air individuals breathe. The activity level affects the rate at which the individual breathes in the contaminated air.

As part of an EPRI study on exposure to acidic aerosols, researchers processed and reorganized raw time-diary activity records from five activity pattern databases [4]. The activity pattern databases were derived from studies performed between 1982 and 1990 that spanned several geographic regions and age groups. The individual studies were conducted in the State of California, and the metropolitan areas of Denver, Cincinnati, and Washington, D.C. Regional differences among these studies were not carried forward into the combined database. Data entries that passed a quality check were coded by microenvironment, exercise level, and demographic group.

To translate the activity pattern data to time (and inhalation rate) parameters for average individuals, the inhalation exposure assessment defined three *population classes:* non-workers, indoor workers, and outdoor workers. Non-workers were defined to be individuals who report never holding a paid position over the 70 year time horizon. Indoor workers were defined to be individuals who have jobs in low-exposure environments for their entire work life. Outdoor workers were defined to be individuals who have jobs in high-exposure environments for their entire work life.

To account for instances where individuals commute to locations far from the plant, the assessment split each population class into two subclasses based on where they spend time outside their residence. The result is six groups of individuals (3 classes x 2 subclasses each). These groups are

- non-workers with all activities near the plant
- non-workers with all activities (outside the home) far from the plant
- indoor workers who work near the plant
- indoor workers who work far from the plant
- outdoor workers who work near the plant
- outdoor workers who work far from the plant.

The assessment also created two additional groups to account for individuals who commute from residences far from the plant to the area near the plant. These groups are

- indoor workers who commute to the area near the plant
- outdoor workers who commute to the area near the plant.

Table E-1 presents the mean values (over a lifetime) for time spent indoors near the plant and time spent outdoors near the plant. The values for subgroups with all activities near the plant indicate that individuals spend the majority of their time indoors (over 80%). The values for the subgroups with non-home activities far from the plant indicate that individuals tend to spend about 16 hours a day at home. Non-workers and indoor workers spend slightly more time indoors than outdoor workers. Commuters spend far less time indoors near the plant and time outdoors near the plant than other population groups.

Table E-1.
Representative Amounts of Time in Plant Proximity Sectors, by Subgroup (hours/day)

Population Subgroup		Time Spent Indoors Near the Plant	Time Spent Outdoors Near the Plant
Work status	Activities		
Indoor	Near	20.9	3.1
Indoor	Far	16.1	0.6
Indoor	Commute	4.8	3.1
Outdoor	Near	20.0	4.0
Outdoor	Far	15.9	0.6
Outdoor	Commute	4.1	4.0
Nonworking	Near	21.1	2.9
Nonworking	Far	18.5	0.6
Source: Calculations based on activity pattern data			

The best estimate of number of days exposed in a lifetime is based on the years nearby residents are expected to live near a power plant, considering the likelihood that they will move in and out of that area. The approach simulates the sequential moves of an individual away from and back to the area near the plant. It uses Monte Carlo sampling to select from distributions of i) time between moves and ii) classification of moves with respect to Metropolitan Statistical Areas (MSAs) and to areas outside of MSAs (non-MSAs). These distributions can be estimated from the American Housing Survey for the United States in 1991, which contains interview data on the year in which residents moved into current units and the location of the previous unit from which they moved.

Table E-2 shows a distribution of the number of years householders have lived in their current unit. The distribution is based on information in the 1991 American Housing Survey [5] regarding the year in which householders moved into their current units. According to the survey, in 1991, the median year in which householders had moved into their units was 1986, which corresponds to a median length of time living in the current unit of about five years. The risk analysis used the length of time in the current unit to characterize the length of time between moves.

Table E-2
Years Householders Have Lived in Current Unit

Year householder moved into unit	Years lived in current unit	Percent of Residents	Cumulative Percent
1990 to 1991	0 to 1	26.3	26.3
1985 to 1989	2 to 6	29.0	55.3
1980 to 1984	7 to 11	11.4	66.7
1975 to 1979	12 to 16	10.1	76.8
1970 to 1974	17 to 21	6.7	83.5
1960 to 1969	22 to 31	8.5	92.0
1950 to 1959	32 to 41	5.1	97.2
1940 to 1949	42 to 51	1.9	99.0
1939 or earlier	>51	1.0	100
Source: Based on 1991 American Housing Survey [Department of Commerce,1993]			

Table E-3 shows the breakdown of moves generated from a summary of actual moves in the 1991 American Housing Survey [5]. (The breakdown considers Primary MSAs (PMSAs) as MSAs. PMSAs are large urbanized regions that demonstrate strong internal economic and social links and are located within an MSA of population greater than 1,000,000.) The breakdown presents the probabilities that moves originating from certain locations will end up in the same location and other locations. In order to generate a breakdown of moves that could be applied to the typical location, the following assumptions were used:

- each state has about five MSAs (the national average per state)
- moves from one MSA to any other MSA within the state are equally likely
- moves from one state to any other state are equally likely.

Table E-3
Breakdown Of Moves For Typical MSAs and Non-MSAs (%)

Destination		Original MSA		Different MSA/ Same State		Different MSA/ Different State		Non-MSA	
Origin		city	suburbs	city	suburbs	city	suburbs	same state	diff state
Original MSA	city	55.4	17.1	4.9	4.1	6.6	6.1	2.6	3.1
	suburbs	16.9	54.4	3.2	9.0	4.4	6.7	2.8	2.7
Diff MSA/ orig state	city	0.87	0.72	59.5	20.5	6.6	6.1	2.6	3.1
	suburbs	0.56	1.58	19.5	61.8	4.4	6.7	2.8	2.7
Diff MSA/ Diff state	city	0.02	0.02	0.12	0.11	66.9	27.2	0.06	5.6
	suburbs	0.01	0.02	0.08	0.12	24.4	70	0.05	5.4
Non-MSA	orig state	0.98	1.65	5.6	9.4	3.4	5.3	5.8	68.0
	diff state	0.01	0.02	0.06	0.09	9.9	16.2	1.4	72.4
Different nation		0.18	0.10	1.01	0.58	58.5	33.3	0.13	6.2

Source: EPRI calculations based on 1991 American Housing Survey [Department of Commerce,1993]

For example, of the total moves nationwide that occurred within identical MSAs, 2% (one out of fifty) were assumed to occur in each state. Of the total moves within any state, 0.4% (one-fifth of one-fiftieth) were assumed to occur in a typical MSA.

It was assumed that the breakdown of moves characterizes an individual's history of moves over an entire lifetime. It was also assumed that moves within the original location (e.g., non-MSA) or back to the original location result in similar exposures.

Each simulation starts at the appropriate origin (e.g., central city of a MSA) and randomly selects the number of years before the first move; the time at the original location and the simulation time are set to this value. The destination of each move is then selected at random (origin and destination can be identical for some moves). This pattern continues until the simulation time reaches 70 years. Then, the total residence time at the originating location is recorded as an estimated residence time near a power plant. The simulation was repeated 2000 times to provide a distribution of the residence time in the vicinity of a power plant. The median value of residence time was 19 years. The median value was assumed to be applicable for calculating an REI near any power plant.

E.1.4 Step 4: Estimation of Inhalation Rates

The fourth step determines the best estimate of outdoor and indoor inhalation rates for the near-plant scenarios. (The terms "exposure" and "dose" are used interchangeably in the risk assessment.) The indoors and outdoors inhalation rates are exercise-level weighted averages based on standard inhalation rates by exercise level [6] and the amount of time spent in different exercise levels for each near-plant exposure scenario [4].

Table E-4 presents the mean estimates (over a lifetime) of indoor and outdoor inhalation rates by subgroup. These rates correspond to near-plant activities. As expected, the outdoor inhalation rates are considerably higher than the indoor rates. In addition, the outdoor rates for the outdoor workers and the non-workers are slightly larger than those of the indoor workers; apparently outdoor workers and non-workers have more strenuous outdoor activities than indoor workers. The workers that commute into the area have higher indoor inhalation rates than the other populations, because their indoor near-plant activities do not include at-home activities (e.g., sleeping, watching TV) that tend to be associated with lower inhalation rates.

Table E-4
Representative Inhalation Rates by Subgroup (m^3/hour)

Population Group		Indoor Breathing Rate	Outdoor Breathing Rate
Work status	Activities		
Indoor	Near	0.61	0.97
Indoor	Far	0.57	0.97
Indoor	Commute	0.72	0.97
Outdoor	Near	0.61	1.05
Outdoor	Far	0.57	1.05
Outdoor	Commute	0.74	1.05
Non	Near	0.60	1.05
Non	Far	0.58	1.05

Source: Calculations based on activity pattern data and inhalation rate data from EPA Exposure Factors Handbook

E.1.5 Step 5: Estimation of Other Factors

Step 5 incorporates best estimates of several other factors that potentially impact the dose, as available. These factors include body weight, the bioavailability of specific chemicals in the human body, and the respirable fraction of emissions for specific types of power plants.

Body weight is important because linear dose-response relationships accepted by EPA (e.g., potency slopes) assume that the expected chronic health response to a given dose of a substance is inversely proportional to an individual's weight. The standard assumption for adult body weight (independent of sex) in risk assessment is 70 kilograms. Since this assessment considers exposure over a lifetime, it used a best estimate of 62.5 kilograms based on body weight data for different ages specified in EPA's Exposure Factors Handbook [6]. The best estimate is a time-weighted average of mean values over 70 years for males and females combined. The best estimate is only appropriate for use in analyses which address exposure over an entire lifetime.

EPA's exposure guidelines advocate the use of known bioavailability information for converting potential dose to applied dose [7]. The fraction of respirable particles determines the portion of suspended trace substances that can potentially enter the body through respiration. Due to the lack of available data, bioavailability and the fraction of respirable particles were not incorporated in the assessment.

E.1.6 Step 6: Estimation of Exposure Measures

This step derives a best estimate of exposure and explores the impacts of key factors on the level of exposure. Best estimates of factors from the previous steps are combined to derive best estimates of *exposure scaling factors* for each substance. Exposure scaling factors are dimensionless adjustment factors that account for the differences between MEI exposure assumptions and more realistic exposure assumptions. A substance exposure scaling factor multiplied by the corresponding exposure of the MEI provides an estimate of realistic exposure to that substance for the typical individual living in the area with highest concentrations from the plant. This step also examines the impact of individual factors from the previous steps on realistic exposure estimates.

E.1.7 Step 7: Incorporation of Plant Replacement

The final step adjusts the emissions input to account for retirement and replacement of older units. For the REI, it was assumed that units are replaced after 55 years of operation with units that meet or exceed the 1994 NSPS for particulate emissions, 0.03 lb/10^6 Btu. Two emissions runs were performed. To project emissions for the years from 2010 through a unit's 55th year in operation, the first run used the projected particulate emissions for 2010. To project emissions for a unit's 56th year in operation to 2080, the second run used the minimum of the NSPS standard for particulates and the projected particulate emissions for 2010. The emissions input to the dispersion modeling is the average emissions from the two runs weighted by the number of years each run is applicable.

E.2 Assumptions and Limitations

The extended exposure approach incorporated relevant data and information from a number of previous studies. To be consistent with rest of the risk analysis and to incorporate the available information, the inhalation exposure assessment relied on a number of assumptions. The major assumptions are described below along with their implications and potential limitations for interpreting the results.

- *Representative values are applicable to all locations in the United States.* The values of some parameters, however, vary by region. For example, people may spend less time outside in areas with extreme weather patterns (e.g., north-central state) than in areas with milder weather and indoor/outdoor ratios may be less for areas with extreme weather patterns because of more extensive weatherproofing. Due to lack of data, the extent to which activity patterns vary by region and its significance for exposure assessments are not well-known. One activity pattern data summary showed insignificant differences for urban and rural locations in the United States, but did not address regional differences [8]. Although the exposure assessment did not explicitly address regional differences, the preliminary ranges of the exposure parameters are likely to more than account for such differences.
- *Representative values reflect exposure levels for the average individual.* It was assumed that the representative values adequately represent the levels of exposure for a reasonably exposed individual.
- *The three population groups in the assessment provide the appropriate level of disaggregation for a national analysis.* Although the three groups only represent a fraction of the possible combinations of working indoors and outdoors and not working, they do provide the most extreme cases from which other cases can be extrapolated, as appropriate.
- *Indoor/outdoor ratios of all buildings are not significantly different from those of residences alone.* This assumption may lead to overestimates of indoor concentrations in buildings other than residences and subsequent exposure levels, as the "typical" building (e.g., job site) is likely to be better insulated than the "typical" residence and, furthermore, many buildings do not have windows that can be opened.
- *Potential dose is not significantly larger than biologically effective dose for the substances in the study.* Available data on factors that impact the biologically effective dose (i.e., the particle size distribution of emissions, absorption, and the bioavailability of trace substances in the human body) were insufficient for use in the risk assessment. Consequently, the exposure assessment is likely to overestimate the dose that ultimately contributes to adverse effects. Incorporating these factors could reduce the risk estimates significantly.
- *Residents are not exposed to concentrations due to emissions from other plants.* EPRI research indicates that this assumption may have minor effects on total exposure due to utility sources in urban areas with multiple power plants or areas in which multiple power plants have wind-aligned plumes.

- *The breakdown of moves for 1991 adequately represents the likely moves of an average individual over their lifetime.* This assumption probably adds a bias to underestimate the frequency with which individuals move back to an area. For example, someone who grew up in an area and subsequently moved away, would seem to be more apt to move back to the area in which they grew up than people who grew up in other areas.
- *The amount of time a person lives in one house (plus incremental time for moves in the same area) is a measure of the duration of an individual's exposure to emissions from the plant.* Since individual risk estimates are associated with individual plants, they do not account for the possibility that someone moves to another area in which they are exposed to emissions from another power plant. The population risk, however, is unaffected by population mobility; it was assumed that someone moves in whenever someone moves out.
- *Moving patterns within and to MSAs (and non-MSAs) are an adequate representation of moving patterns from and back to the area of elevated concentrations due to emissions from a power plant.* This assumption overestimates the moves back to the area of elevated concentrations because the area of elevated concentration is likely to be smaller than the typical MSA or non-MSA.
- *Moves within the area of elevated concentrations or back to the area of elevated concentrations are assumed to have random start- and end-points (with respect to concentrations due to plant emissions).* This assumption is unbiased with respect to exposure levels, as the location of a new home within the area of elevated concentrations due the plant is equally likely to have higher or lower concentrations than the location of the original home.

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APPENDIX F
COMPLEX TERRAIN CONSIDERATIONS

F.1 Introduction

The exposure assessment in Section 5 of this report assumed dispersion occurred for all sites under flat terrain conditions when calculating the maximum ground-level concentration (MGLC) and receptor concentrations. This section will examine the potential impact of adding terrain conditions to the dispersion calculation. The model used for the dispersion calculations in this study is U.S. EPA's Industrial Source Complex (ISC) Version 2, in the Long Term (LT) time averaging mode (ISCLT2)[1]. Changes in the ground-level concentrations will have proportionate changes in the risk for the individual cancer risk based on the maximum concentration.

A literature survey was conducted of the results of comparisons between "regulatory" Gaussian plume models, such as ISCLT2, and field tracer tests to evaluate those and other models [2-13]. The Gaussian models in general have limitations, but have been shown to be quite good for predictive purposes (that is, results within a factor of 2 to 4 of measurements) [14-21], although these have in general been qualification experiments on the short-term mode of the models. Since this type of model is semi-empirical, any severe disagreement with measurement could be accommodated by an "adjustment factor" based on measurements. In these cases, higher-order (parameterized nonlinear) models along with field measurements have been used to generate adjustment factors for the ISC results (see, e.g., [22]).

The measurements used for comparison also have limitations due to temporal and spatial variations. The ISC model has gained wide acceptance for analysis of chronic health impacts using annual concentrations [23-25]. Bowman's comparison [26] indicated "concentrations generally agree within 6%," but larger differences may occur. Recent results for current model results indicate agreement to within a few percent.

F.2 Dispersion Coefficients

The selection of urban vs. rural dispersion coefficients in the ISC model runs is site-dependent and may result in a large change in the estimated risk. In all cases for elevated sources, the urban dilution factor is greater than the rural dilution factor (that is, concentrations are higher in the former case than in the latter per unit emission rate, all else being

equal). For a range of modeled plants in a variety of settings, the average ratio of urban to rural dispersion parameter (χ/Q)is about 6 [27]. About 80% of power plant sites nationally are classified as rural by use of EPA population density criteria, although local regulatory agencies may specify use of preferred dispersion coefficients generally resulting in the more conservative results. The impact of the choice for the plant's dispersion coefficients is expected to be greater than the inclusion of terrain.

F.3 Terrain

In the context of air dispersion model analysis, terrain surrounding an elevated source can be viewed in two contexts: 1) the height the surrounding topography that is below the effective stack height and 2) the height of the surrounding topography that is above the effective stack height (*complex terrain* conditions). The effective stack height is based on the buoyant exit conditions of the stack gas. The dispersion of the emitted material about the centerline of the plume traveling downwind is modeled using empirically based dispersion coefficients. A key issue for calculating the MGLC is the modeled intersection of this centerline with the ground. Since boundary-layer air flow will result in "terrain-following" centerlines, these conditions require additional modeling capabilities not found in ISC. Currently, in many site-specific risk assessment calculations, dispersion is computed using the short term version of ISC (ISCST2) with truncated (below-stack) elevations or, alternatively, using models designed for this specific purpose (e.g., COMPLEX–I) [28,29]. Current EPA guidance for computing the concentration for a receptor array about a site refers to use of the following:

1. For ground elevations below the stack height, use ISC
2. For ground elevations above the stack height, but below the effective plume height (termed *"intermediate terrain"*) use both ISC2 and COMPLEX–I, selecting the maximum value calculated for each time period.
3. For ground elevations above the effective plume rise, use COMPLEX–I.

The additions of terrain to a dispersion calculation adds the large additional data requirement of the ground elevations associated with each site (16 compass directions/site x 50 radii/compass direction x 600 sites, or roughly 500,000 data points, for this assessment). Hence, flat terrain assumptions were used for all sites in the national power plant assessment. Other site-specific dispersion analyses have used ground elevations in regulatory calculations and limited studies are available, but in these analyses other conditions (such as the selection of dispersion coefficients and the effects of downwash) were also varied. Thus, the impact of terrain should be considered in conjunction with the other conditions that impact the MGLC and receptor concentrations.

Analysis of terrain effects on dispersion for a small number of power plant sites was carried out to indicate the approximate magnitude of the difference between concentrations due to dispersion in flat terrain and those in complex terrain. The results indicate that the

adjustments to accommodate terrain are highly specific to each site and that generalizations are difficult. Table F-1 presents the results of these calculations. For this small set of plant sites in complex terrain, the change from flat terrain concentrations calculated using ISCLT2 ranged between an increase of a factor of two and a decrease of a factor of two. The analysis then considered the magnitude of dispersion result changes for other conditions impacting dispersion. It was found that selection of dispersion coefficient (which produced increases in MGLC of up to a factor of 10) and inclusion of building downwash (increases of a factor of 6) for stacks below "good engineering practice" (GEP) represented larger potential increases than the inclusion of terrain. For a large number of modeled sites, such as in the current analysis, only a *very small fraction* will be underestimated by use of the ISCLT2 model and use of the flat terrain assumption. It should be noted that the ISCLT2 model is designed to provide conservative numbers (that is, to over-predict concentrations vis-a-vis actual conditions). The application of upper bound uncertainty factors to such a small number of sites, requiring site-specific information regarding building dimensions, land uses, population and topography, was beyond the scope of the present analysis.

Table F-1
Ratios of Maximum Ground Level Concentrations for Stacks in Complex Terrain (Rural Coefficients, Eastern U.S. Locations)

Site	Stack Condition	Concentration for Listed Condition Divided by MGLC with Terrain		
		without accounting for terrain	with downwash	with urban coefficients
1	<GEP	0.8	5.9	9.9
2	<GEP	0.5	3.1	6.1
4	<GEP*	1.0	4.4	8.9
5	<GEP*	2.1	4.4	3.5
6	<GEP*	0.5	3.3	3.8

* Calculations at less than 100% load GEP is Good Engineering Practice

F.4 Downwash

Downwash, as used here, refers to mechanical turbulence induced by aerodynamic wakes downwind of structures, resulting in increased turbulence and reduced plume rise in that wake region. In the current model treatments of downwash, adjustments are made for the dispersion coefficients [30] along with the plume rise [31] using the dimensions of nearby structures.

In general, downwash will increase the concentrations near the stack and decrease the concentrations far from the stack. Stacks designed to good engineering practice (GEP) will not be effected by nearby structures. Thus, downwash is of concern for a small number of sites containing shorter stacks. Such stacks may be eliminated by the year 2010. The impact of downwash for a limited number of sites in complex terrain for stacks below GEP is given in Table F-1. For stacks not meeting GEP, the upper bound uncertainty factor may be up to a factor of 6.

F.5 Elevated Receptors

Exploratory analyses were carried out to investigate how the assumption of flat terrain may impact the resulting concentration estimates. In one case, the effects of nearby tall residential structures were examined for a particular urban power plant. This analysis was performed with the use of the SCREEN model [34]. SCREEN is a very conservative model relying on a default set of worst-case meteorological conditions and conservative parameters. SCREEN has the ability to simulate receptors at heights equivalent to a high-rise building ("flagpole" receptors). The model calculates the worst-case, maximum hourly concentrations as a function of distance from the stack. The ratio of concentrations at various receptor heights divided by the ground-level concentration provides a representative scaling factor for the ISCLT2 results, most relevant to the highly conservative MEI exposure assumption.

SCREEN analysis for a tall (>500 ft.) stack in an urban location resulted in the maximum concentration for a receptor height of about 1200 ft. above the stack base. At this elevation, and at a distance from the stack of 1000 meters, the exposure would be 30 times as great as at ground level, and at 2000 meters distance, about 20 times as great. This means the MGLC (and thus the MEI) would seriously underestimate the risk to a resident in a tall apartment building near a stack. Assessing that risk requires detailed site-specific data applicable to only a few sites. It should be noted that the SCREEN model is designed to provide an overestimate of concentrations, to guide investigators to more detailed models for a particular site.

F.6 Short-Term vs. Long-Term Averaging Periods

In addition to its long-term averaging "LT" mode, the ISC model has a short-term "ST" version, requiring discrete hourly meteorological data. ISCST can be used to calculate an annual summary, but requires considerably more data, especially if short-term hazard quotients as well as chronic risks are computed in a risk assessment [29]. The long-term model uses several assumptions to reduce the large computational requirement of the short-term version, but the same equations are used for calculating the concentrations.

The long-term model uses meteorological data in a STability ARray (STAR) summary format. The STAR summary is a joint frequency of occurrence of wind-speed class and stability category for each 22.5 degree wind sector. Wind speeds are grouped into one of six classes for wind speeds of 0-3, 4-6, 7-10, 11-16, 17-21, and greater than 21 knots. The Pasquill-Turner methods is used to determine stability class. A temperature and mixing height are required for each stability class. The STAR program distributes calm winds over each stability class based on the frequency of occurrence of the lowest two wind-speed classes within each stability class.

A potential uncertainty in any ISC calculation is related to the amount and period of the meteorological data used in the dispersion calculation. An analysis of the Mt. Tom Power Station in New England compared results using one year of measured STAR data to those using 5 years of STAR data, and to results derived using hourly data from a nearby NWS weather station [33]. The uncertainty factor due to the choice of meteorology data was estimated at 1.3 (that is, the upper bound uncertainty factor is +30%).

F.7 Summary

The results applicable to this analysis are summarized below and in Table F-2. The consequence for the industry-wide analysis is that a very small number of sites may have risk estimates that are not conservative due to the assumptions of no structure downwash and flat terrain.

Table F-2.
Summary of Dispersion Analysis Impacts on Risk Assessment

Description	Air Quality Model Feature			
	Dispersion Coefficient	Terrain	Receptor	Structures
CORE Analysis	Uses population criteria consistent with EPA Guidelines	Flat terrain	Ground Level Concentration (GLC)	None
Impact	Small number of sites with underestimate of risks	Underestimate of risk for populations in areas with significant topographic features near source	Underestimate of risk for population in structures at or above stack height	Underestimate or over-estimate of risk based on relative location of receptors and morphology of nearby structures
Recommended treatment in sensitivity analysis	Use Urban in place of Rural coefficients only when non conservatism is of potential concern	Highly site-dependent based on location of receptors and population	Concern for sites when non-conservatism is an important issue. Treat specifics for small set of sites	Not considered in current effort. Simple uncertainty factors cannot be used; screening needed for determining effect at specific sites.

Table F-2.
Summary of Dispersion Analysis Impacts on Risk Assessment (Continued)

Description	Air Quality Model Feature			
	Dispersion Coefficient	Terrain	Receptor	Structures
Relative importance for site-specific Risk Assessment	High; Comparisons for the entire set of sites available	Low; may impact a very small number of sites but downwash and dispersion coefficient are more important	High for a very small number of sites with elevated receptors	Medium for a very small number of sites with stacks below GEP
Low: less than one order of magnitude change in the maximum concentration. Medium; 0.5 to 1 order of magnitude change in the maximum concentration. High: greater than 1 order of magnitude change in the maximum concentration.				

- *Selection of Air Quality Model for Dispersion.* Gaussian air quality dispersion models appear to match observations of ground-level concentrations only to within a factor of 2 (and that only for relatively short-term conditions); this level of uncertainty represents a small contribution to the overall uncertainty in the risk estimate (based on uncertainties in emissions estimates, discussed in Section 4). The choice of an air dispersion program (ISCLT2 vs. more specialized models) would thus tend to have a small impact on the overall risk calculation due to the conservative nature of ISCLT2.
- *Dispersion Coefficients.* The selection of urban vs. rural dispersion coefficients is site-dependent and may cause a large change in the risk estimate. In all cases, for elevated sources, the urban concentrations are higher than those for the rural assumption. The impact of the choice for the plant's dispersion coefficients is expected to be greater than the inclusion of terrain, but will only impact a very small number of sites.
- *Terrain.* The impact of terrain needs to be considered in conjunction with other conditions that contribute greater uncertainty to the inhalation risk calculations.
- *Downwash.* Site-specific calculations of structure downwash is beyond the scope of the current study because building dimensions would be required near each of the stacks at a site. This should not be a concern except for stacks below GEP.
- *Elevated Receptors.* In addition to site-dependent characteristics such as terrain and dispersion coefficients, downwash and elevated receptors (e.g. nearby residential or office buildings) are expected to have a greater impact than terrain on the dilution factor portion of the risk estimate. The *ground-level concentration* (GLC) is inappropriate for assessing impacts on elevated receptors.

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APPENDIX G
EPRI HEALTH RESEARCH

This section, which describes ongoing EPRI work, focuses on health research on arsenic and mercury. The purpose of much of this work is to address deficiencies in important areas specific to utility concerns where default assumptions (as described for arsenic in Section 6.3.1) are used in current risk assessments. Results from all of these studies are aimed at reducing uncertainties in these assumptions and thus eventually improving the accuracy of risk assessments for these compounds.

G.1 Arsenic

G.1.1 Proposed Revision of the IRIS Inhalation Unit Risk for Arsenic

The current U.S. EPA unit risk factor for arsenic as a carcinogen by the inhalation route is 4.29×10^{-3}. This factor is based on findings of significantly increased risk for lung cancer in epidemiology studies among workers at two U.S. smelters, the Anaconda Copper Smelter in Montana and the Asarco Copper Smelter in Tacoma, Washington. Recently, the original authors of the Tacoma smelter study have published updated exposure/dosimetry estimates for that study indicating that the workers were much more highly exposed than had been previously though when EPA completed its risk assessment in 1984. Further, results from a recent Swedish smelter study have been described in the literature. Based on these new data, EPRI has used standard EPA risk assessment methodology to re-calculate estimated risk. Pooling risks derived from these new data along with EPA's earlier estimate yields a composite new unit risk of 1.43×10^{-3} which is one-third the value of 4.29×10^{-3} that now appears in the IRIS database. Based on this work, EPRI is formally requesting a change in the IRIS database for the inhalation unit risk for arsenic. A manuscript describing this work has been accepted for publication in *Regulatory Toxicology and Pharmacology* and is attached in it entirety as Technical Appendix 6.2 (TA6.2).

G.1.2 Examination of Valence State, Chemistry, and Particle Size

The current default assumption used for cancer risk assessment for arsenic by the inhalation route assumes that arsenic in any valence state or chemical form is equally carcinogenic. Unit risk for arsenic as a carcinogen by the inhalation route is based principally on findings of significantly increased risk for lung cancer in epidemiology studies of copper smelter workers [1, 2, 3]. In these settings, workers were exposed to copper

smelter dust that contains high concentrations of arsenic trioxide in a complex mixture of other elements and compounds including cadmium and sulfur dioxide. Arsenic trioxide is the principal form of arsenic in copper smelter dust and is present in the As (III) valence state. As(III) is approximately 10 times more acutely toxic than is arsenic in the As(V) valence state and some portion of inhaled or ingested arsenic (V) is converted to As(III) in the body. The possible implications of these differences in valence state for cancer induction are unknown. It has been uncertain whether emitted coal fly ash contains arsenic principally in the form of As(III) or As(V). As previously stated, the current U.S. EPA cancer risk assessment for arsenic by the inhalation route is based chiefly on occupational exposure to the As(III) form [4]. Therefore, a goal of this research is to characterize the form of arsenic related to particles in coal fly ash and then compare this information with similar parameters in copper smelter dust.

Knowledge of arsenic's chemical form in different particle sizes is important because the particle size determines the differential deposition in the respiratory tract. In addition, the distribution of arsenic species in various particle size fractions may be incorporated into the physiologically based pharmacokinetic (PBPK) arsenic model currently under development. This will allow estimation of the inhaled fractions and chemical forms of arsenic in coal fly ash, its deposition characteristics in the lung, and bioavailability and distribution in the body. Scenarios can then be compared with similar information characterizing copper smelter dust.

G.1.2.1 Laser Microprobe Mass Analysis (LAMMA)

The distribution and speciation of arsenic present in individual fly ash particles has been studied by developing the Laser Microprobe Mass Analyzer (LAMMA) technique [5]. The LAMMA method can determine the composition of individual particles in the micrometer size range. Although it cannot obtain exact quantification of arsenic in each particle, the overall detection limit for arsenic is in the range of 0.1 to 1 ppm. Particles are shot with a laser beam and the ions generated are sorted by a mass spectrometer. Ion intensity ratios are determined and used to identify the form of arsenic present in the particle. Verification of results from LAMMA analysis were obtained by use of standard analytical methods for arsenic as described in the following section.

Mass spectral behavior under LAMMA conditions of the following compounds were investigated in order to apply this method to analysis of arsenic in coal fly ash: arsenic trioxide, arsenic pentoxide, sodium metaarsenite, sodium arsenate, potassium arsenate, calcium arsenite, calcium arsenate, several naturally occurring arsenates and phosphates and aluminum arsenate. The mass spectra of these compounds contain signals for As, AsO_3, AsO_2, and AsO. The negative-mode LAMMA spectra, through signals for metal-oxide fragments along with the positive-mode spectra, provide information about the

counter cations for the oxoanions of arsenic. Use of factor analysis of the mass spectral signals for As and AsO_x (x = 1, 2, 3) obtained from the reference arsenic compounds listed above also allows the identification of counter cations.

LAMMA analyses of at least ten different coal ash samples—including electrostatic precipitator (ESP) ash, isokinetically collected stack fly ash, ash collected high in the stack on a pulse-jet fabric filter (PJFF), hopper ash, bottom ash, and material collected on filters by high-volume sampling from ambient air—were conducted.

Analysis of a sample with a total arsenic concentration of 2200 mg/kg from the ESP of a power plant in Slovakia burning lignite coal of very high (about 800 ppm) arsenic content, showed that in this sample the element is present in about 5% of particles with diameters < 2 μm. Factor analysis of the spectral data indicated a calcium salt of an oxoanion of arsenic as the major component of these small arsenic-rich particles. Approximately 40% of the large silicate particles in this sample also gave mass spectra with a weak signal for AsO2 thus indicating that arsenic is also associated with these silicate particles.

LAMMA spectra from a coal fly ash sample containing 200 mg/kg total arsenic obtained from a PJFF in a U.S. coal-fired plant indicated that no small arsenic-rich particles were present, but approximately 20% of all particles gave a mass spectra with a weak arsenic signal. The total arsenic concentration in this sample is relatively high for coal fly ash from U.S. power plants.

Thus, in samples with very high total arsenic content as in the ESP sample above, arsenic is associated both with small non-silicate arsenic-rich particles (that contain calcium, as well as traces of phosphate, iron and aluminum) and to a lesser extent with large silicate particles. In samples with lower total arsenic concentration such as the PJFF sample (but yet a very high total arsenic concentration for a U.S. sample), arsenic is found only in association with silicate particles that make up the bulk of fly ashes.

Other methods were used to verify and extend the above findings as follows. Experiments with calcium arsenite revealed this compound to be unstable in air. Atmospheric oxygen oxidizes calcium arsenite rather quickly to calcium arsenate. These results provide strong evidence for calcium arsenate mixed with aluminum/iron arsenates and phosphates as the predominant arsenic compound in coal fly ash. This identification was corroborated by results of leaching studies with fly ash samples and the identification of arsenic compounds in the leachates by hydride generation and high performance liquid chromatography (HPLC) with arsenic-specific detection employing an inductively coupled argon plasma mass spectrometer (ICP-MS). Only arsenate was found in these leachates. Scanning electron microscopy with energy-dispersive X-ray fluorescence (SEM-EDX) identification of the elements in the arsenic-rich particles showed that the calcium signals are positively correlated with the arsenic signals. The recorded micrographs

of a number of samples indicate that arsenic-rich particles are agglomerated with much larger silicate particles. Thus, calcium arsenate (As V) has been identified as the major arsenic compound in coal fly ash.

Using the LAMMA technique, no nickel- nor chromium- containing particles were found in any samples of coal fly ash, however, the concentration of these elements in coal fly ash may be considerably below the limit of detection of the LAMMA method, especially if these elements are evenly distributed over all the silicate particles.

G.1.2.2 High Performance Liquid Chromatography with Inductively Coupled Plasma Mass Spectrometry (HPLC-ICP-MS)

Analysis of water soluble arsenic from the standard reference coal fly ash sample NIST-SRM 1633a at neutral pH showed concentrations in the range of 40-60 ppb, less than 0.1% of the certified amount in the sample. At pH 12 the amount of arsenic in solution increased to 2000 ppb while at pH 1 the amount of solubilized arsenic increased to about 3500 ppb.

Water soluble arsenic was speciated by HPLC using an ion exchange resin. Separation of standard sodium salts of As (III) and As (V) as well as the two primary metabolites monomethyl arsenic acid and dimethyl arsonic acid was accomplished with little or no interference due to argon or chloride. This methodology was applied to water soluble arsenic from the NIST SRM 1633a as well as several utility stack ashes and a copper smelting dust. At neutral pH, As (V) was the predominant species for all but one of the coal fly ashes and the smelter dust, accounting for 90 to 95% of the total arsenic. The exception had 15 ppb As (III) and 8 ppb As (V). At pH 2 As (III) was preferentially solubilized in all samples.

The data indicate that: (1) very little of the total arsenic is soluble in water, and (2) the arsenic that is soluble is predominantly As (V) at neutral pH.

G.1.2.3 Borohydride Reduction at pH 8-9 or pH 1

The relative amounts of As (III) and As (V) in micro-milled or ultra-sonicated samples of NIST SRM 1633a was determined by selective borohydride reduction for As (III) and As (V) respectively. The product was quantitated using I_3^- adsorption and catalytic wave voltammetry using an instrument specifically designed for this purpose. Effects of storage conditions and extraction ruled out any conversion of As (III) to As (V). Mass balances within 1% of the certified value were accomplished. The limit of sensitivity for this method is 3.9 ppb.

No As (III) was detected in the NIST SRM 1633a fly ash. These results were verified using the NIST SRM 1633b fly ash which differs in composition from the 1633a.

Therefore, using several different analytical and methodological approaches, it has been shown that the species of arsenic present in coal fly ash is As (V) and that the major arsenic compound present in coal fly ash is calcium arsenate (As (V)).

G.1.3 Bioavailability

It is unknown whether assumptions about the bioavailability of arsenic can be extrapolated from occupational epidemiology studies in copper smelters to community exposure of fly ash. An issue of concern is whether the arsenic present in fly ash is readily solubilized in the lung and then distributed throughout the body or whether some insoluble fraction is retained in the lung where it may then exert local toxic effects. The relevance of the complex exposure mixture in copper smelters to the exposure to arsenic in fly ash (another complex mixture) needs to be studied.

Assessment of the bioavailability of arsenic from fly ash by the inhalation route as observed directly in humans is also important to reduce uncertainties in this area. The occupational setting provides opportunity to measure breathing zone exposure to arsenic in fly ash and to assess urinary excretion kinetics of inorganic arsenic and organic metabolites.

G.1.3.1 Lung Retention and Bioavailability in Hamsters

Experimental animal studies are necessary to assess whether significant differences in bioavailability exist between arsenic in fly ash and arsenic in other sources including copper smelter dust. A study in hamsters has been conducted to determine lung retention and bioavailability as indicated by urinary excretion after respiratory exposure by intratracheal instillation. These parameters have been measured at time points after exposure to arsenic in fly ash and compared with those for arsenic in copper smelter dust and for the pure sodium salts of arsenic—arsenite [As(III)] and arsenate [As(V)]—with and without concomitant exposure to inert particles.

Results indicate that soluble sodium salts are cleared completely from the lung by the second day after exposure; simultaneous presence of an inert particle has no effect on clearance. In contrast, a portion of arsenic from both the Slovak coal ESP hopper ash and copper smelter dust are still retained in the lung at the second day after exposure with arsenic from copper smelter dust being somewhat more highly retained (Fig. G-1). This figure also illustrates that lung clearance follows a typical two-stage non-linear process. In addition, results from the urinary analyses indicate that arsenic that is bioavailable (absorbed) from copper smelter dust seems to be excreted at a slightly slower rate than arsenic that is bioavailable from coal ash.

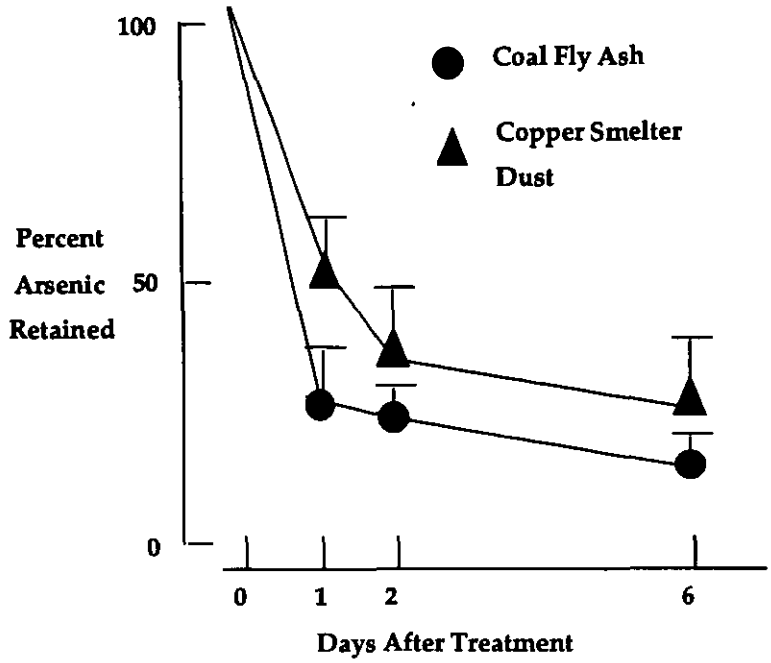


Figure G-1.
Comparative Lung Retention of Arsenic After Treatment with Coal Fly Ash or Copper Smelter Dust

A second series of experiments was conducted to compare the lung retention and urinary excretion kinetics of arsenic from a coal fly ash sample obtained from a U.S. power plant stack pulse-jet fabric filter (PJFF) with that of the Slovak ash sample as well as with soluble As (V) as sodium arsenate and less soluble arsenic in the form of pure calcium arsenate. The PJFF sample contained considerably less total arsenic at 200 mg/kg than that of the copper smelter dust at about 2000 mg/kg therefore ten times more total particulate material from the PJFF sample needed to be administered in order to treat animals with equal doses of arsenic.

Results show that the soluble As (V) compound is not retained in the lung and is rapidly excreted with about 65% of the dose excreted at 48 hours. In contrast, all three of the particulate preparations showed a two-phase non-linear clearance from the lung and even at six days post-exposure from 5-20% of the administered dose remained in the lung for all particulates compared with no measurable amount remaining for the soluble pentavalent arsenic. At 48 hours after exposure, however, arsenic from the PJFF sample was more highly retained than from either of the other two particulates. Approximately 45% of the dose from this sample was still present in the lung compared with about 20% of the dose retained for both calcium arsenate and the Slovak ESP fly ash sample. The difference in the lung retention for these three particulates was also reflected in the rate of urinary excretion of arsenic metabolites. The slower rate of arsenic lung clearance for the PJFF fly ash sample may be due to a greater dust load in the lung or to less solubilization than for either of the other particles or to a combination of these factors. Particle lung loading has

been observed to have a significant effect on lung clearance [6]. Further work is underway to standardize the lung dust load and then re-compare arsenic lung clearance for these three particulates to assess the effect of lung particle load on retention and clearance rates.

Initial macroscopic observations of the lung indicate that coal hopper ash does not produce a significant inflammatory reaction, but that copper smelter dust may be more aggressive. EPRI plans to rigorously assess possible differences in lung inflammatory response between arsenic containing coal ash and copper smelter dust. Such inflammatory response will be assessed by examining bronchoalveolar lavage fluid for different cell types as well as macromolecules (such as specific proteins, cytokines, and growth factors). Inflammatory response may be of considerable consequence since it may induce cell proliferation, which is thought to be important in the carcinogenic pathway.

G.1.3.2 Occupational Exposure Assessment and Bioavailability of Arsenic from Coal Fly Ash

A study of occupational exposure to arsenic in coal fly ash is being conducted in a coal-fired power plant in the Slovak Republic, where lignite coal of very high arsenic content is used as the principal fuel. Breathing-zone exposure has been characterized for a group of about 40 power plant workers estimated to be exposed to a wide gradient of fly ash concentrations during outage operations. Daily full-shift time-weighted average (TWA) personal breathing zone samples were collected on each of five consecutive days from each worker beginning on the first day of a planned maintenance outage after at least two days off from work. Cascade impactor samples were also collected to determine the concentration of arsenic in various particle size fractions. Total arsenic in particulate air samples was determined using atomic absorption spectrophotometry (AAS). Twenty persons from the surrounding community participated in the study as referents. Ambient air concentrations were estimated using high volume air samplers both inside and outside of the homes of the community reference population. First void urine samples were collected from all workers every morning for the five consecutive days of air sampling and also after the shift on the fourth and fifth work day. Analysis is under way using hydride generation coupled with AAS to determine the concentrations of inorganic arsenic and organic metabolites in urine samples from both workers and community referents. Questionnaires were administered to all participants to determine occupational and health history as well as lifestyle and dietary factors including fish consumption in order to control for possible confounders.

Results to date show that arsenic air concentrations did range widely from a mean of 0.03 $\mu g/m^3$ in both indoor and outdoor ambient air for community referents up to a weekly mean of 150 $\mu g/m^3$ for the most highly exposed group of workers (Fig. G-2). Preliminary results from cascade impactor samples indicates that about 80% of arsenic is contained in the large particle size fractions greater than 6 μm in diameter (Fig. G-3). These samples were collected from areas inside the boiler during cleaning operations early in the outage

and also some personal cascade impactor samples were collected. Work is continuing to speciate arsenic in urine samples from all subjects. Data from questionnaires is being assembled into a database before conducting statistical analyses with adjustments for potential confounders.

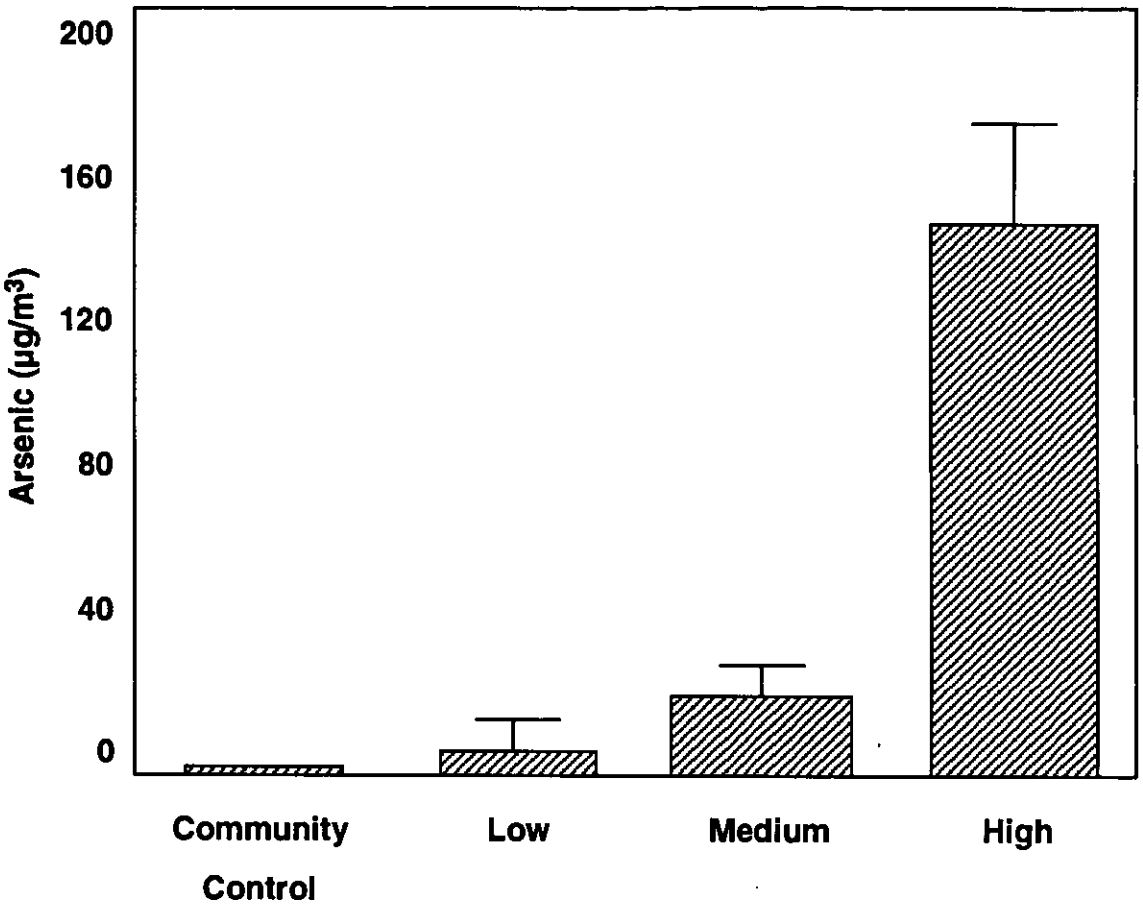


Figure G-2.
Weekly Mean Arsenic Concentration in Air, Per Group

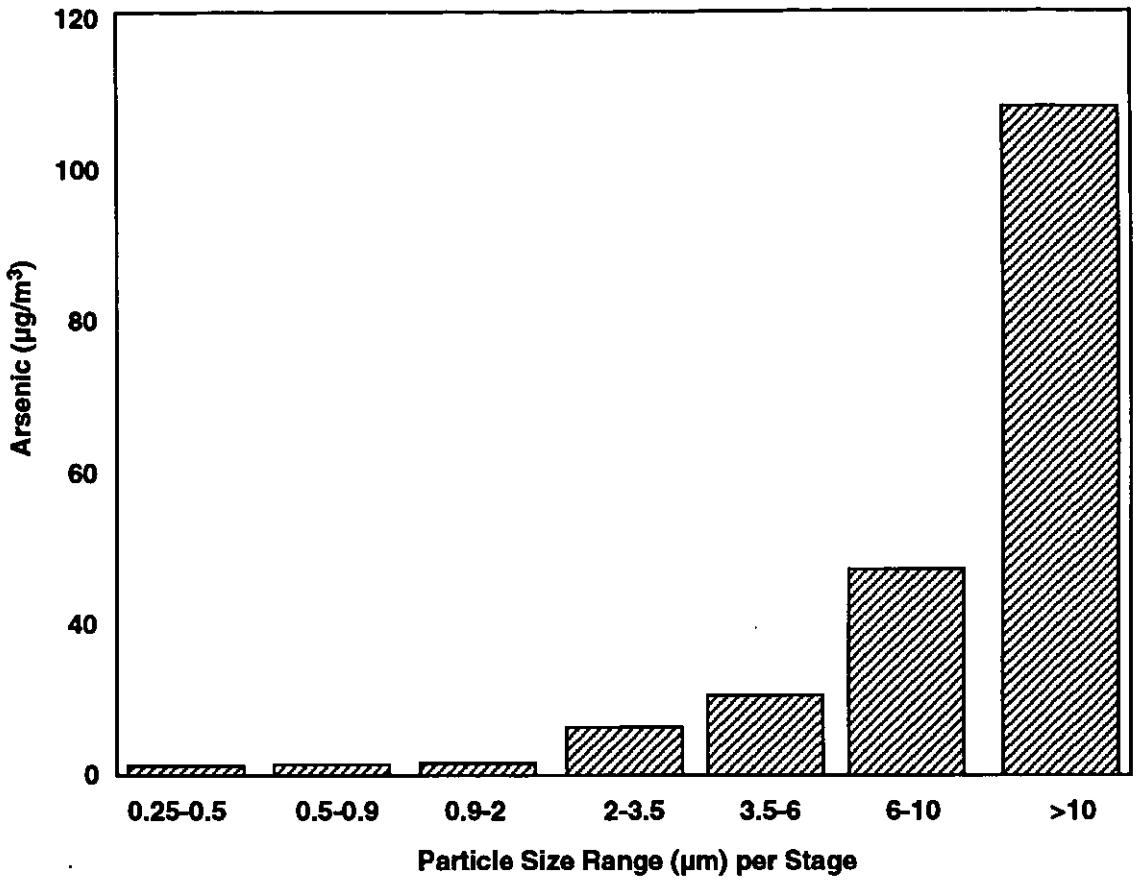


Figure. G-3.
Arsenic Concentration by Particle Size Fractions

A large gradient of exposure from high to low in this study may allow examination of differences in excretion kinetics of arsenic under different conditions of exposure. Thus, high-to-low-exposure effects might be studied in the context of one set of exposures to arsenic in coal fly ash. Data from this study may also be useful to validate the arsenic PBPK model described in Section 6.1.4.

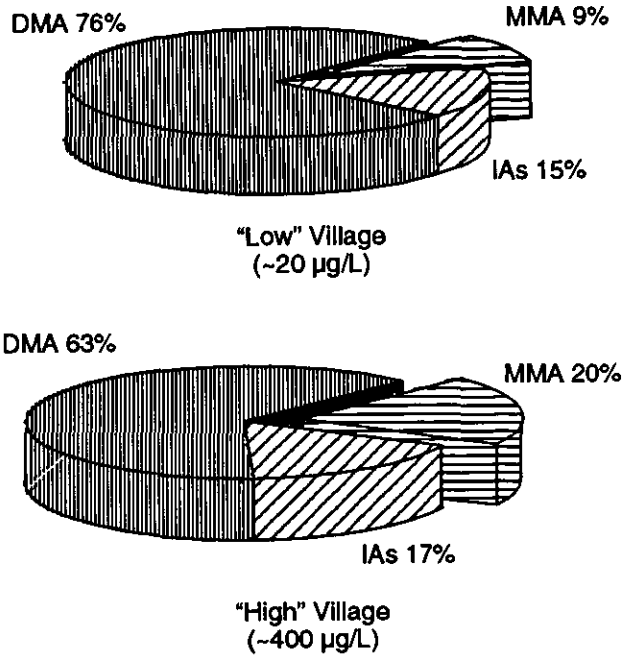
G.1.3.3 Bioavailability of Arsenic from Drinking Water in Mexico: Implications for Non-linearity

A study was undertaken in order to assess bioavailability and kinetics of arsenic under conditions of chronic exposure over a fairly wide range of exposures that occur due to ingestion of water containing arsenic. Two villages in the Comarca Lagunera of Mexico were selected. The levels of arsenic in drinking water in the "High" exposed village were between 0.375 and 0.392 mg/L whereas in the "Low" exposed village, arsenic concentrations measured during the past two years were between 0.019 and 0.026 mg/L. The current drinking water standard in the U.S. is 0.05 mg/L. The objective of the subject sam-

pling design was to select healthy individuals from the high exposed and low exposed communities so that factors such as medical treatments, alcoholism, and chronic disease would not confound the potential associations between arsenic exposure and disease. Persons with skin lesions attributable to arsenic exposure were preferentially sampled from the high exposed community to obtain a larger number of these individuals. Individuals from the low exposed community were frequency matched on age and sex to the sample obtained from the high exposed community. A questionnaire was administered to study participants that included questions on age, lifestyle, medical history, occupation, and water consumption. Urine and blood samples were collected from each participant for subsequent analyses. A total of 60 individuals were enrolled in the study; 30 from each of the two villages. Urinary concentration of inorganic arsenic, and the two major metabolites, monomethyl arsenic acid (MMA) and dimethyl arsonic acid (DMA) were determined by hydride generation AAS. A number of parameters are being measured in blood including arsenic concentration as well as cell proliferation and chromosomal studies.

The metabolism of arsenic involves reduction-oxidation reactions that inter convert As (V) and As (III) and sequential methylation of As (III) to yield the two major metabolites, MMA and DMA. Metabolism is generally considered to be a detoxification process since the organic metabolites are considerably less toxic than the inorganic species. An issue of ongoing interest is whether this process remains linear over a wide range of arsenic exposure concentrations or whether at high exposures to arsenic, efficiency of the methylation steps decreases, resulting in a non-linear process.

In order to assess the above, inorganic arsenic, MMA and DMA were calculated as a percent of the total urinary arsenic excreted for each subject and then the mean value of each of these parameters was calculated for each of the two villages. Results show that for the most highly exposed village, significantly less DMA (55.5% vs. 80.6 %) and more MMA (12.1 % vs. 7.8%) was excreted in urine than in the low exposed village controlling for other factors such as age and sex. Although there are possible alternative hypotheses, results suggest that the second methylation step is impaired in the most highly exposed individuals. Similar results have been shown very recently in a pilot study of a small group of individuals chronically exposed to arsenic in the same area (Fig. G-4) [7]. Additionally, a similar pattern of excretion was seen in a recent animal study in which acute oral exposure to sodium arsenate was studied; at high doses, the amount of MMA excreted was significantly increased, whereas the elimination of DMA was delayed and a lower amount was excreted [8]. Taken as a whole, these results imply that non-linearity in the detoxification process may occur at both high acute and chronic exposures.



Ref. Del Razo, et al., *Sci. Tech. Letter*, 1994 (In press).

Figure G-4.
Proportion of Arsenic Species in Urine of Individuals Chronically Exposed to Arsenic Via Drinking Water, Region Lagunera, Mexico

Demonstration of non-linearity in the exposure-response relationship has important implications for risk assessment, since the default procedure for cancer risk assessment is to carry out linear extrapolation from effects observed at very high exposures to assumed effects at very low estimated exposures. If that relationship is indeed non-linear, then linear extrapolation is not appropriate.

G.1.4 High-Dose to Low-Dose Extrapolation

In smelter work settings, exposure to high concentrations of arsenic trioxide [As (III)] occurred. In some studies, airborne daily time-weighted average concentrations of arsenic were estimated to range from 0.4 to 62 mg/m^3. Extrapolation from these high exposures to very low community ambient levels needs to be examined in light of multiple biological parameters that may deviate from linearity under conditions of high exposure relative to ambient levels. The ongoing power plant occupational study with community referents currently being conducted in the Slovak Republic (described in the previous section) also addresses the issue of high-exposure to low-exposure extrapolation for arsenic in coal fly ash.

G.1.4.1 Development of a PBPK (Physiologically Based Pharmacokinetic Model) for Arsenic

Development and validation of an arsenic PBPK model that is applicable to humans and other mammals is necessary to effectively summarize current knowledge about factors related to absorption, distribution, metabolism, excretion, and kinetics of the compound in the body. After appropriate validation, the model can then be applied to simulate various exposure scenarios to compare and contrast a number of potential differences including the effect of high dose versus low dose, route of exposure, peak versus chronic exposure, arsenic species, arsenic compound solubility, particle size distribution, and lung clearance rates, among others.

A multi-compartment PBPK model has been developed using information from the literature on physiological parameters and metabolic pathways specific for arsenic (Fig. G-5) [9]. This operational model encompasses oral and pulmonary exposure routes to arsenic compounds in both the As(III) and As(V) valence states. Keratin has been included in the skin compartment since arsenic [As(III)] is known to bind with the sulfhydryl groups in keratin. Different distribution equations were developed for the ionic compounds [As(V), MMA, and DMA] and for the non-ionic compound [As(III)]. Reduction and methylation rates (metabolism) were incorporated into the model. Preliminary validation of the model has been carried out for several animal species and for humans.

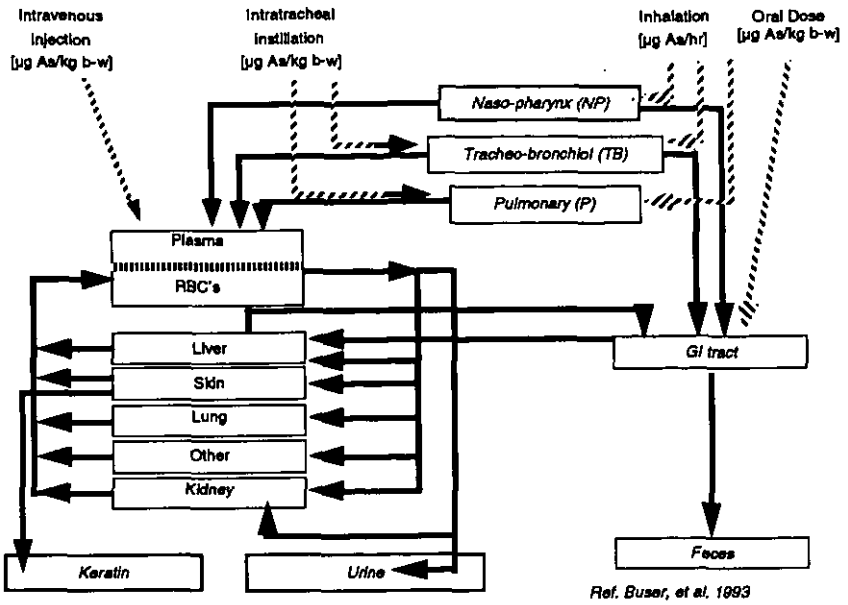


Figure G-5.
Schematic Presentation of the Arsenic PBPK Model: Absorption Routes, Tissues and Excretion Routes for Metabolites.

Various exposure scenarios have been tested in the model. These factors, if substantiated, may considerably refine assumptions concerning the uptake and kinetics of arsenic in the body under different conditions of exposure. Some preliminary results follow: comparison between the oral and inhalation route of exposure for As(III) versus As(V) indicates that route of exposure appears to have less effect on the proportion of metabolites excreted in urine than does arsenic species [As(III) versus As(V)]. Modeled exposure to As(III) compared with As(V) seems to result in higher liver concentrations of As(III) independent of route of exposure. There seems to be little effect on arsenic concentration in the lung when mechanical lung clearance rates in the model are changed by a factor of four. Less soluble arsenic compounds appear to produce higher lung concentrations for exposure by inhalation. Testing in the model of the effect of different size aerosols shows that aerosols with smaller mass median diameters give rise to lower concentrations of arsenic metabolites in all tissues and urine. This is likely due to lower deposition rates for particles of small aerodynamic diameter.

Work is under way to refine the model, for example, in order to attain a more physiological uptake from the gastro-intestinal (GI) tract, the absorption from the GI tract was changed from the plasma to the liver to take into account the first pass effect. The fecal elimination rate was obtained by using the rate of food passage in the small intestine by a first order mechanism. Adjustment of absorption rates was then accomplished and a refined fit of tissue affinity constants and the metabolic rate constants were made using published data. *In vitro* experiments are underway to generate independent data on tissue affinity constants.

EPRI plans to apply specific high-dose to low-dose extrapolation scenarios such as the examination of various parameters of absorption, distribution, metabolism, and excretion between high occupational exposure to arsenic, as experienced in copper smelters, and estimated low airborne community exposures. In general, EPRI plans to conduct simulation studies of specific bioavailability and toxicological problems related to inhalation exposure of fly ash compared to exposure to forms of arsenic present in copper smelter dust. Such simulation studies are likely to conserve both time and financial resources and may reduce uncertainties for a number of parameters in the current default assumptions used in arsenic risk assessment.

G.2 Mercury

To learn about the risks associated with typical environmental exposures, data sets based on prospective studies with rigorous experimental design are needed. These studies should use well-defined health endpoints (such as developmental effects measured by a test battery), exclude potential confounding factors (such as other chemical exposures and/or socioeconomic or cultural factors), and examine health effects in everyday long-term, low-level exposure settings.

G.2.1 Long-Term Developmental Studies

Several population studies relating maternal dose of methylmercury during pregnancy to children's responses on a variety of neurological and performance tests have been completed or are underway. For example, the Danish government is studying the neurobehavioral effects of intrauterine exposure to methylmercury among residents of the Faroe Islands located in the North Atlantic between Scotland and Iceland (Grandjean et al. 1992; Grandjean and Weihe 1993). They have collected blood and hair samples from 1000 mothers and the babies who were born to them over a two-year period at regional island clinics. Analysis of these samples has shown that maternal—hence fetal—methylmercury exposures are primarily determined by Faroese consumption of pilot whale meat containing an average of 3.3 μg/g total mercury, about half of which is methylmercury. The study is also examining confounding factors such as the influence of methylmercury on birth weight, the presence of selenium and PCBs in pilot whale meat, and maternal alcohol consumption during pregnancy. Faroese children born during the study are being followed and will be tested for developmental effects of methylmercury exposure. This study is important because it represents a prospective, long-term developmental assessment.

The NIEHS and the Ministry of Health, Republic of Seychelles, are co-sponsoring a study of 779 mother-infant pairs living in the Seychelles Islands located in the Indian Ocean off the coast of Africa. Seychelles Islanders consume a fish diet thought to be high in methylmercury. Researchers have tracked pre- and postnatal exposure of the children involved and have inferred body burdens of methylmercury from blood and hair sam-

ples. This long-term study has followed children up to 5.5 years of age and has evaluate their neuropsychological development using a battery of tests cross-culturally validated for Seychelles children and administered by trained personnel at a local Child Development Center (Davidson et al. 1993). Preliminary analyses of the study results will soon be available.

By focusing on island populations, both of these research programs automatically exclude many factors—such as differing genetic and cultural backgrounds and varying nutritional environments—that could confound the influence of methylmercury on human health. Both are well-designed long-term efforts that should yield valuable data sets for further analysis. However, neither of these exposed populations enjoys a lifestyle similar to that of the United States. For example, although the Faroe Islands are administered by the Danish government, citizens there eat few green vegetables, may drink large quantities of alcohol, and dine on cured meat. Therefore, extrapolating conclusions from these studies to U. S. populations will require careful analysis.

G.2.2 EPRI's Mercury Health Research Program

As a basis for better understanding the effects that chronic low doses of methylmercury can have on the central nervous system in children, EPRI has developed a physiologically based pharmacokinetic (PBPK) model to describe the fate of methylmercury in the body. Using information from this model, in conjunction with new statistical procedures, EPRI researchers are reanalyzing available epidemiology data to improve dose-response estimates, especially for assessing risks borne by children of exposed mothers.

EPRI's PBPK model describes the oral adsorption, distribution, metabolism, and excretion of methylmercury. The goal has been to create a model that will simulate the kinetics of methylmercury in different species simply by changing species-specific parameters. For example, because the model has separate compartments for red blood cells (RBCs) and plasma, it can predict changes in the kinetics of methylmercury related to RBC/plasma ratios that are unique to each species. The PBPK model has been validated with data for rats, monkeys, and people.

The adult PBPK model uses compartments to represent organs, specific tissues, and waste products. The compartments describe methylmercury transport (plasma, kidney, richly and slowly perfused tissues, brain-blood, placenta, liver, gut, RBCs, and brain) and methylmercury reabsorption and excretion (the intestinal lumen, hair, urine, and feces). Conversion of methylmercury to inorganic mercury by flora in the gut and subsequent elimination of inorganic mercury in the feces is the most important mechanism of excretion. (Some methylmercury is excreted in the feces, but most is reabsorbed.) Incorporation of methyl and inorganic mercury in the hair is also a significant mechanism of excretion. Finally, the adult model incorporates a fetal sub-model with four compartments that grow during the time of gestation (fetal plasma, RBCs, brain, and the

remaining fetal body). The fetal sub-model is particularly important since one of the purposes of the PBPK model is to describe exposure to the human fetus for developing a reference dose.

The PBPK model accurately describes both the long-term concentrations of methylmercury in specific organs and the clearance of methylmercury from the body following termination of exposure. To date, the model has successfully predicted plasma, red blood cell, brain, and hair levels of both methyl and inorganic mercury in monkeys who have sustained up to four years of continuous oral exposure to 10–300 μg/kg (body weight)-day of methylmercury. The model has made similar predictions for human volunteers exposed for varying lengths of time to a broad range of methylmercury doses. Thus, the PBPK model appears to reliably predict physiological changes that covary with exposure to methylmercury.

EPRI researchers have used the PBPK model to determine the relationship between maternal intake of methylmercury and measured maternal blood and hair concentrations. Armed with this information, they have reliably estimated fetal *in utero* exposure from maternal hair concentration. Such estimates allow them to reevaluate methylmercury doses for children in data sets from previous studies where maternal hair concentration is known. (Model extensions are planned to describe postnatal exposure, such as that occurring when breast fed children ingest mothers' milk.)

In collaboration with its principal investigators, EPRI is reanalyzing the data set from a New Zealand study of mothers and their prenatally exposed children. These mothers ate a steady diet of fish, and analyses revealed more than 6 mg of methylmercury per kg of their hair sampled during pregnancy. This level contrasts with mean values of 2.3 to 3.1 mg mercury per kg of hair that are typical of adults living in the Northern hemisphere (Airey 1983). Using the PBPK model, EPRI researchers have been able to estimate the dose for New Zealand children based on the concentration of methylmercury measured in their mothers' hair. So far, methylmercury exposure has failed to explain a significant part of the variability in scores among these children when they were assessed for cognitive disfunction on standardized tests such as those for IQ. Since socioeconomic factors may contribute much of that variability, EPRI researchers are obtaining socioeconomic profiles for the New Zealand population under study and incorporating those profiles in the reanalysis in cooperation with the principal investigators.

The current approach to assessing risk from methylmercury exposure defines a threshold—a LOAEL or NOAEL ("no-observed-adverse-effect-level") reduced by an uncertainty factor of 10. However, the USEPA is currently giving serious consideration to the use of a Benchmark Dose statistical method for setting the methylmercury RfD; it has already performed such an analysis on developmental endpoints (Marsh et al. 1987, 1981, 1980) from the Iraqi data set. In the traditional approach for estimating a NOAEL from animal data, responses at each dose are compared statistically with control responses, and the NOAEL is defined as the lowest dose showing no statistically significant difference.

In the case of human epidemiological data, it is necessary to group the observations into arbitrary categories by exposure in order to perform this analysis. In contrast, the Benchmark Dose method uses a statistical dose-response model to calculate a "benchmark dose" (BMD), the dose or exposure predicted to result in a specified amount of increased risk (the "benchmark risk" or BR). By fitting a dose-response model to all of the data, the Benchmark Dose method makes better use of the dose-response information inherent in the original sample and avoids arbitrary categorization of observations.

A statistical lower bound on the benchmark dose has been proposed as a replacement for the traditional NOAEL (USEPA 1990; Gaylor and Slikker 1990, Kimmel and Gaylor 1988). Table G-1 presents results from a study recently conducted for the USEPA (Allen et al. 1994; Faustman et al. 1994) comparing benchmark doses associated with differing benchmark risks to traditionally derived NOAELs for 424 sets of animal data. It is clear that use of 0.1 benchmark risk provides a statistical lower bound on the benchmark dose that corresponds most closely to the traditional NOAEL. However, use of 0.1 benchmark risk also makes the RfD more conservative by a factor of about 2 to 3 on average as compared to the RfD derived using the traditional NOAEL approach.

Table G-1.
Comparison of Benchmark Dose for Differing Levels of Risk with Traditional NOAEL

Benchmark Risk	Benchmark Dose vs. Traditional NOAEL
0.1	BMD < NOAEL by an average factor of 2.9 for 75% of the data sets
0.5	BMD < NOAEL by an average factor of 5.9 for 90–95% of the data sets
0.01	BMD < NOAEL by an average factor of 29 for 95+% of the data sets

EPRI researchers have developed a method for calculating benchmark doses and their statistical lower bounds (BMDLs) from continuous endpoints (Crump 1994) such as those represented by children's scores on test batteries. They have used this method to reanalyze psychological, behavioral and scholastic data from the study of New Zealand children described above.[1]

Table G-2 presents the results of this Benchmark Dose analysis. Depending on the values selected for P_0 (the percentage of control children who might be deficient on a given test) and BR, the Benchmark Dose analysis suggests that the NOAEL for the most sensitive indicator of developmental effects in six-year-old children occurs at approximately 10-31 ppm mercury in maternal hair, with an estimate based on the most reasonable choice of parameters (BR = 0.1 and $P_0 = 0.05$) of 17 ppm. The most sensitive indicator of effects (that is, the test producing the lowest BMDLs) was the grammar understanding section of the Test of Language Development.

Table G-2.
Benchmark Dose Analysis of Scores Attained on a Battery of Developmental Tests Administered to New Zealand Children Exposed to Methylmercury *in utero*

P_0 Fraction of Unexposed Population Affected	BR Benchmark Risk Level	BMD Maximum Likelihood Estimate (range*, ppm in maternal hair)	BMDL 95% Lower Bound (range*, ppm in maternal hair)
0.01	0.10	61–379	31–90
0.01	0.05	43–347	22–64
0.05	0.10	34–7221	17–50
0.05	0.05	20–4364	10–30

*Range of values obtained over the various tests used in the epidemiological study.

At a NOAEL of 17 ppm mercury in maternal hair, analysis using the PBPK model described above indicates that fetal brain tissue concentrations of methylmercury are on the order of 50 ppb (μg/L). According to the model, this concentration in fetal brain tissue would result from a maternal dietary intake of methylmercury ranging from 0.8 to 2.5 μg/kg (body weight)-day. This broad range of intakes corresponding to a target maternal hair concentration of 17 ppm mercury reflects the high degree of variability among hair-to-intake ratios seen in human studies. These results suggest that the current USEPA RfD for methylmercury of 0.3 μg/kg (body weight)-day adequately protects against developmental effects (Gearhart et al. 1994).

EPRI also continues to investigate the possible relevance of the Iraqi data set to health risk from utility emissions. Since the original analysis (Marsh et al. 1987), upon which the current RfD for methylmercury is based, other researchers have reanalyzed these data on late or retarded development in Iraqi children exposed to methylmercury *in utero.* One

[1]In this application, the researchers chose a nonlinear dose-response model

$$\mu(d) = \mu_0 + \beta d^k$$

where $\mu(d)$ is the mean of the responses associated with a specific dose, d; μ_0 is the mean of the responses for the controls; and β and k are the estimated parameters. When they compared the test scores of prenatally exposed children with those of unexposed controls, they also needed to account for the performance of children in the control group who might score badly on a test for reasons unrelated to methylmercury exposure. Therefore, the researchers estimated that either 5% ($P_0 = 0.5$) or 1% ($P_0 = 0.01$) of the control children would fall in the deficient category. Finally, the model assumed that test scores are normally distributed with a standard deviation, σ, independent of dose. Given these assumptions, choosing $P_0 = 0.01$ and BR = 0.1 is equivalent to defining the benchmark dose as the dose that results in a 10% change in the mean response relative to the standard deviation (that is, as the dose that satisfies $|\mu(d) - \mu_0| / \sigma = 0.1$).

reanalysis (Cox et al. 1989) used delayed walking and neurological scores for exposed children to calculate the "best statistical estimate" of the NOAEL as 10 ppm mercury in maternal hair, with a 95% range of uncertainty between 0 and 13.6 ppm. This threshold estimate is equivalent to an RfD of 0.07 μg/kg (body weight)-day (Stern 1993), much lower than the current USEPA RfD of 0.3 μg/kg (body weight)-day. However, preliminary research at EPRI (Crump et al. 1994) indicates that the results from threshold models are very model-dependent and there is a large range for the 95% confidence interval of the threshold. Furthermore, EPRI's analysis indicates that the Benchmark Dose method is probably the best model to use. Finally, the analysis reveals inconsistencies in the Iraqi data that make other data sets based on long-term exposure at low dose a more suitable basis for setting standards for prolonged human exposure to methylmercury.

Ultimately, EPRI will use these new epidemiological, experimental, pharmacokinetic, and statistical findings in a national health risk assessment for methylmercury.

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APPENDIX H
UNIT RISK FOR ARSENIC

REGULATORY TOXICOLOGY AND PHARMACOLOGY 20, 125–138 (1994)

Unit Risk Estimates for Airborne Arsenic Exposure: An Updated View Based on Recent Data from Two Copper Smelter Cohorts

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Received January 21, 1994

The current unit risk for airborne arsenic, 4.29×10^{-3}, was established by the EPA in 1984. Using updated results from a cohort mortality study on Tacoma smelter workers and recent findings from a cohort study of 3619 Swedish smelter workers, new unit risk estimates were developed for the respective cohorts. Methods were analogous to those used by the EPA in 1984, and all estimates were derived under an absolute risk model. A new unit risk 1.28×10^{-3}, was estimated for the Tacoma smelter cohort which was a factor of 5 less than the EPA's earlier estimate, and a direct result of radically revised exposure estimates. A unit risk of 0.89×10^{-3} was estimated from the Swedish study. Pooling these new unit risk estimates with the EPA's earlier estimates from the Montana smelter cohort yielded a composite unit risk of 1.43×10^{-3}. Based on this estimate, the present unit risk may overestimate the effects of airborne arsenic by a factor of 3. A need to update the unit risk for airborne arsenic and the collateral IRIS database is evident from the results.

INTRODUCTION

In 1984 the EPA established a unit risk of 4.29×10^{-3} for chronic lifetime exposure to airborne arsenic. This was principally based on dose–response relationships representing cumulative arsenic exposure and excess lung cancer mortality experience of workers employed in two U.S. copper smelters: the Anaconda smelter in Montana and the Asarco smelter in Tacoma, Washington (Table 1). A decade later, this remains the agency's best estimate for projecting excess lung cancer risk in the general population. Since 1984, updated analyses of the Tacoma smelter cohort have been published (Enterline *et al.*, 1987; Mazumdar *et al.*, 1989), incorporating an extensively revised assessment of arsenic exposure. These new exposure estimates show an upper range of cumulative exposure, that is about a factor of 10 greater than that used in earlier EPA analyses (Pinto, 1978; U.S. EPA, 1984).

Further, comprehensive analyses covering the lung cancer mortality experience of 3916 Swedish smelter workers were recently published (Jarup *et al.*, 1989). All workers ever employed in the Ronnskar smelter for three or more months during the period 1928–1967 were studied, and cohort mortality experience was determined through

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TABLE 1

EPA UNIT RISK ESTIMATES FOR AIRBORNE ARSENIC DERIVED FROM TACOMA AND MONTANA SMELTER STUDIES

Smelter population	IRIS reference[a]	Unit risk estimate: Study	Unit risk estimate: Cohort	Unit risk estimate: Summary
Montana	Brown and Chu, 1983	1.25×10^{-3}	2.56×10^{-3}	4.29×10^{-3}
	Feldstein, 1983	2.80×10^{-3}		
	Higgins and Welsh, 1982	4.90×10^{-3}		
Tacoma	Enterline and Marsh, 1982	6.81×10^{-3}	7.19×10^{-3}	
		7.60×10^{-3}		

[a] U.S. EPA (1992). The Integrated Risk Information System (IRIS): inorganic arsenic. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

1981. Exposure reconstruction took into account available air measurements, temporal changes in industrial hygiene practices, production levels, and processes through 1981. Temporal exposure patterns were further validated using medical surveillance and departmental morbidity data when available (Sandstrom *et al.*, 1989). In contrast to the Montana and Tacoma studies, this cohort included most workers ever employed in the facility, and workers with short-term exposure (about 19% worked less than a year) were well represented.

In this paper, the ramifications of these recent data on the present unit risk estimate are explored. For both the Swedish and Tacoma cohorts, potency estimates for arsenic-induced lung cancer were derived under an absolute risk model in keeping with the 1984 EPA paradigm. Cohort-specific unit risks were calculated from the respective potency estimates utilizing a life table based on 1976 all-cause and lung cancer mortality. Methods employed in this study were comparable to those used by EPA and afford a direct comparison with earlier estimates (EPA, 1984).

Summary unit risks were calculated from Swedish and Tacoma results alone and further combined with the 1984 unit risk from the Anaconda smelter cohort. Taken together these new summary estimates suggest a revised unit risk, perhaps a factor of 3–4 less than the present EPA estimate.

MATERIALS AND METHODS

The unit risk (UR) represents the excess probability of developing lung cancer, given continuous atmospheric exposure to 1 $\mu g/m^3$ of arsenic over a lifetime. Employing methods analogous to those used by the EPA in 1984, potency estimates summarizing the cohort-specific dose response were derived under a linear absolute risk model. This assumed that additional cancer mortality increased linearly with dose, was independent of age (Crump and Allen, 1985), and reflected the additional lung cancer mortality per $\mu g/m^3$/year resulting from occupational exposure. These were used as inputs into a life table to compute the corresponding unit risk, accounting for competing risks from all-cause mortality and making an adjustment for the differential between

occupational and continuous population exposure. The authors based the life table on 1976 age-specific all-cause and lung cancer mortality, and evaluated the unit risk assuming an average life expectancy of 76.5 years.

The dose-related excess absolute risk (EMR) was calculated from the difference between the observed and expected lung cancer mortality rates, based on observed and expected deaths, and person-years of observation stratified by cumulative dose, in each study. Maximum likelihood estimates of potency resulted from fitting the EMRs and cumulative arsenic exposure under the absolute model, by a Poisson regression for grouped data with external controls (Breslow and Day, 1987). These were fitted using GLIM (Numeric Algorithms Group, 1986), and model diagnostics were based on the uncorrected Pearson χ^2 and corresponding P value to assess the adequacy of model fit. The fit was considered unacceptable if the P value was less than 0.01.

Since the expected lung cancer deaths in each cohort were based on the age and calendar-time mortality experience of an external reference population, the authors estimated the potency with and without a fitted intercept. The intercept estimates the excess background cancer risk, a measure of the underlying comparability between the cohort and reference population (external control). In the author's analyses there was clear evidence that the baseline lung cancer rate in the Swedish cohort was greater than in the reference population, likely reflecting the greater frequency of smoking among cohort members, relative to the reference population.

RESULTS

Tacoma Smelter Cohort

Background. A unit risk, $\sim 7 \times 10^{-3}$, was developed by the EPA from data published by Enterline and Marsh (1982). In that study urinary arsenic concentration was used as a biomarker for airborne exposure, and the dose response for arsenic-related lung cancer mortality was expressed in terms of cumulative urinary arsenic exposure (μg/As/liter urine-years). Since workers might have used some means of respiratory protection, Enterline assumed that urinary arsenic was a more relevant measure of actual arsenic uptake.

For purposes of assessing airborne risk, however, equivalency between airborne arsenic and urinary concentration needed to be established. Urinary exposure was converted to equivalent air levels in $\mu g/m^3$ by the EPA from the association between air and urinary arsenic: $As_{air} = 0.304\ As_{urine}$ previously reported by Pinto *et al.* (1976). Cumulative air exposure was projected by multiplying the 1982 cumulative urinary arsenic exposure by 0.304. That relationship was based on Pinto's study of 24 workers exposed to low levels of arsenic during a 5-day period, when daily urinary and air arsenic concentrations were monitored.

In retrospect, limitations were apparent in Pinto's study. High baseline levels of urinary arsenic (about 150 μg/liter) related to prior arsenic exposure were not taken into account. Thus, Pinto's relationship did not reflect the true incremental change in urinary arsenic related to air exposure during the period of observation. Urinary analyses, also, did not account for dietary organic arsenic, and airborne arsenic exposure was, on average, very low (about 54 $\mu g/m^3$).

Because of these and other limitations in assessing exposure, a reanalysis of the earlier Tacoma study was undertaken (Enterline *et al.*, 1987). This assessment radically revised the dosimetry, which has been used to estimate the unit risk presented here. Regression analyses of urine and air measurements covering a range of smelter departments showed that the resulting power function: $As_{air} = 0.0064$ (urine arsenic)$^{1.942}$ best explained the air/urine relationship. The dramatic contrast between Pinto's earlier estimate and that developed by Enterline is shown in Fig. 1. As we discuss later, Enterline's results are highly consistent with more recent findings from other investigators.

Estimates. Potency estimates were developed for both 1982 and 1987 studies from the cumulative arsenic exposure and corresponding EMRs presented in Table 2. Standardized mortality ratios (SMR) were also tabulated since they reflect the association with exposure under a relative risk model. In either case the effects of the revised dosimetry are apparent. Contrasting the range of exposure between the 1982 and 1987 studies, the lower end of the dose range, gave new estimates, about 4 times those previously observed, while the upper range of dose varied over previous estimates by a factor of 10.

Table 3 contrasts the replication of the EPA's earlier findings with results using the revised exposure estimates. Both the 1982 and 1987 scenarios manifest a clear dose-response, even though the respective potency estimates are remarkably different. Similarly, those regressions fitted to the null, without intercept, adequately describe a dose response in either data set. This is consistent with the other regression results shown in Table 3, where an explicit intercept was estimated and found not to be statistically significant.

Based on the model goodness of fit summarized in Table 3, the regression utilizing 1987 arsenic dosimetry yielded a superior fit (P value of 0.60), with an estimated potency of 1.13×10^{-7}. This is about a factor of 5 less than shown in the alternative analysis based on earlier exposure estimates. The difference is further reflected in the corresponding unit risks which were 1.28×10^{-3} and 6.76×10^{-3}, respectively.

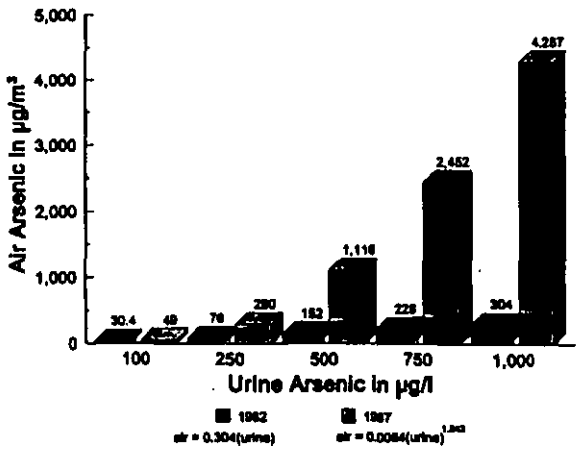

FIG. 1. Relation of air to urine arsenic in the 1982 and 1987 studies of the Tacoma smelter cohort.

TABLE 2

LUNG CANCER MORTALITY IN TACOMA COHORT BY LEVEL OF CUMULATIVE ARSENIC EXPOSURE AND STUDY

Arsenic $\mu g/m^3$-years	Enterline and Marsh 1982[a]				Arsenic μ/m^3-years	Enterline *et al.*, 1987[b]			
	Deaths	SMR	EMR	Person-years		Deaths	SMR	EMR	Person-years
91.8	8	202.0	3.71	10,902	424.5	9	136.4	1.47	16,277
263	18	158.5	3.07	21,642	1,370.1	15	169.9	3.95	14,611
661	21	203.3	7.30	14,623	2,955.0	19	184.0	6.47	13,394
1,381	26	184.1	8.55	13,898	5,784.5	21	204.9	9.29	11,568
4,091	31	243.3	19.43	9,398	11,412.0	23	221.0	13.36	9,423
—	—	—	—	—	29,558.2	13	264.0	22.96	3,519
—	—	—	—	—	57,375.0	4	338.5	41.96	672
Total	104	198.2	7.31	70,464		104	198.2	7.31	70,464

Note. EMR, the excess absolute lung cancer mortality risk $\times 10^4$.

[a] Based on Table 8 in Enterline and Marsh (1982).

[b] Based on Table 2 in Enterline *et al.* (1987).

TABLE 3

LUNG CANCER RISK IN TACOMA SMELTER COHORT: COMPARISON OF DOSE–RESPONSE ESTIMATES UNDER ABSOLUTE RISK MODEL

	Dose response		Model fit and unit risk		
Data source	Intercept[a]	Potency	$x^2(df)$	*P* value	Unit risk[b]
Enterline and Marsh, 1982	2.94×10^{-4}	4.15×10^{-7}	0.546 (3)	0.91	4.68×10^{-3}
	NS				
	-0-	6.00×10^{-7}	5.419 (4)	0.25	6.76×10^{-3}
Enterline *et al.*, 1987	2.52×10^{-4}	8.48×10^{-8}	1.263 (5)	0.94	0.96×10^{-3}
	NS				
	-0-	1.13×10^{-7}	4.612 (6)	0.60	1.28×10^{-3}

Note. -0-, Regressions fitted without intercept. NS, not statistically significant.
[a] Intercept: Estimate of cohort background risk and expressed as excess cancers per 10,000 person-years.
[b] Unit risk given as excess lung cancer deaths/1000 persons exposed to 1 μg of As for a lifetime.

In terms of model fit, the estimated potency and corresponding unit risk, derived from the 1987 study seem superior to earlier estimates. Since there was no further mortality follow-up, the differences in the unit risk estimates are a direct result of the choice of exposure metric. Figure 2 shows the revised dose response and observed EMRs with corresponding 95% confidence limits and demonstrates the excellent fit associated with the revised dosimetry.

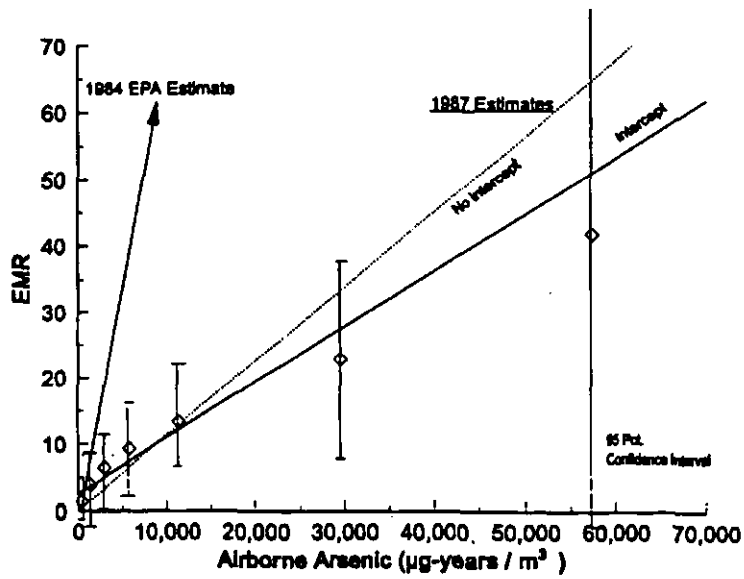

FIG. 2. Comparison of lung cancer risk under the absolute model for 1984 and 1987 exposure estimates, Tacoma smelter workers.

Ronnskar Smelter Cohort

Separate analyses, covering the total cohort and two subcohorts based on the year of first employment, were developed. Subcohort analyses accounted for broad temporal changes in exposure and demographics. In all there were 106 lung cancer deaths, occurring among the 3619 workers, while 27.5 were expected based on the local county population as an external reference. County disease-specific death rates by age and calendar time were applied to the respective cohort person-years to estimate expected deaths. The midpoint of each cumulative exposure level (Table 4), expressed in micrograms, was used as a measure of dose. Since the interval for cumulative exposure exceeding 100 mg-year/m^3 was open ended, the authors assumed that the median exposure in this group was 25% greater than the lower bound of the given interval.

Table 4 details observed deaths, standardized mortality rates (SMR), and the absolute risk (EMR) for lung cancer, stratified by level of cumulative arsenic exposure and subcohort. Workers employed after 1940 had significantly lower exposure then workers hired prior to World War II, and accounted for about 90% of the observed person-years among workers with cumulative exposure under 1000 μg-years/m^3. This resulted from the implementation of strict controls on worker exposure and the introduction of respiratory protection after 1945. Lung cancer mortality was roughly three times greater in the pre-1940 cohort, when measured by either the SMR or EMR.

Estimates. The resulting potency estimates and corresponding model fits are given in Table 5. Model fits covering all workers (total cohort) demonstrated a statistically significant background risk not accounted for by arsenic exposure. This is seen in the unacceptable model diagnostics when the regression was fitted to the null ($\chi^2 \sim 37$; $P < 0.001$) and related to a greater frequency of smokers in the cohort than that in the local reference population (Jarup *et al.*, 1989). It was known that the local reference population had a lower percentage of smokers compared to Sweden as a whole, and significantly less than among smelter workers.

TABLE 4

LUNG CANCER MORTALITY IN SWEDISH SMELTER WORKERS BY CUMULATIVE ARSENIC EXPOSURE AND PERIOD OF FIRST EMPLOYMENT[a]

Cumulative As exposure		Total cohort			First hired <1940			First hired 1940+		
Dose category mg-years/m^3	Range midpoint in μg-years/m^3	Deaths	SMR	EMR	Deaths	SMR	EMR	Deaths	SMR	EMR
<0.25	125	14	271	2.15	3	284	4.29	11	267	1.88
0.25–<1	625	13	360	3.85	3	603	11.88	10	319	3.08
1–<5	3,000	17	238	3.67	6	223	3.64	11	247	3.68
5–<15	10,000	15	338	7.50	10	285	5.89	5	537	13.26
15–<50	32,500	29	461	14.12	27	448	13.60	2	757	26.46
50–<100	75,000	6	728	24.87	6	728	24.87	—	—	—
100+	125,000	12	1137	43.94	12	1137	43.94	—	—	—
Total		106	372	6.17	67	428	10.98	39	302.3	3.25

Note. Person-years by ascending order of exposure: Total cohort: 41,152, 24,407, 26,884, 14,091, 16,083, 2081, 2491 (total = 127,189). <1940: 4533, 2106, 9088, 11,023, 15,427, 2081, 2491 (total = 46,747). Hired 1940+: 36,619, 22,301, 17,796, 3069, 656 (total = 80,441).

[a] Based on Tables 4–5 in Jarup *et al.* (1989).

TABLE 5

LUNG CANCER AND CUMULATIVE ARSENIC EXPOSURE IN THE SWEDISH SMELTER COHORT: DOSE-RESPONSE ESTIMATES BASED ON ABSOLUTE RISK MODEL

Employment cohort	Dose response		Model fit and unit risk		
	Intercept[a]	Potency	χ^2 (*df*)	*P* value	Unit risk[b]
Total	2.74×10^{-4}	3.43×10^{-6}	1.223 (5)	0.941	0.39×10^{-3}
	-0-	4.64×10^{-5}	37.44 (6)	<0.001	Lack of fit
First hired <1940	4.04×10^{-4}	2.94×10^{-6}	2.261 (5)	0.814	0.33×10^{-3}
	-0-	4.05×10^{-5}	16.51 (6)	0.011	0.46×10^{-3}
First hired 1940–1967	1.93×10^{-4}	8.53×10^{-6}	0.604 (3)	0.894	No association
	-0-	1.51×10^{-7}	13.09 (4)	0.011	1.71×10^{-3}

[a] All estimates of background were highly significant ($P < 0.01$).
[b] No association: Estimate of potency did not achieve statistical significance ($P < 0.05$).

In the authors' analysis the best fitting model for the total cohort yielded a potency estimate of 3.43×10^{-4} and a corresponding unit risk of 0.39×10^{-3}. This was more than a factor of 2 less than observed in the authors' previous estimate from the Tacoma study.

Subcohort analyses showed some deviation from the pattern seen in the cohort as a whole. Among workers hired prior to 1940, the model fit with intercept was excellent. The dose response (potency) was highly significant. Background risk was evident since the intercept achieved statistical significance. The corresponding unit risk was 0.33×10^{-3} which was comparable to that for the total cohort. Forcing the regression to the null gave a very poor model fit, but acceptable by the EPA's criteria, given the the p value was 0.011. The resulting unit risk was 0.046×10^{-3}.

Among workers employed after 1939, there was no statistical evidence of an association between arsenic exposure and increased lung cancer risk. When the model was fitted with intercept, the residual background risk (intercept) was statistically significant, as noted in other analyses. Although there was a positive trend in the dose response, the potency estimate was not statistically significant ($P < 0.15$). This is consistent with previous results presented by Jarup *et al.* (1989), where there was no clear association between arsenic exposure and lung cancer risk in those hired after 1939 based on a relative risk model. Fitting the model to the null yielded a very poor model fit, but acceptable by EPA criteria. The corresponding unit risk was 1.71×10^{-3}.

In the Swedish cohort, the significant background lung cancer risk is very likely related to smoking. The effects of smoking and potential interaction with arsenic has been well studied in the cohort (Jarup and Pershagen, 1991). Even though all analyses contained evidence of a residual background risk, representing the well-documented disparity in smoking pattern between the cohort and reference population, regressions without intercept yielded marginally acceptable model fits by EPA criteria. The authors used those potency estimates to compute the subcohort unit risks. This, undoubtedly, attributes smoking-related risk to arsenic exposure. Figure 3 shows the general features of the relationships reported here.

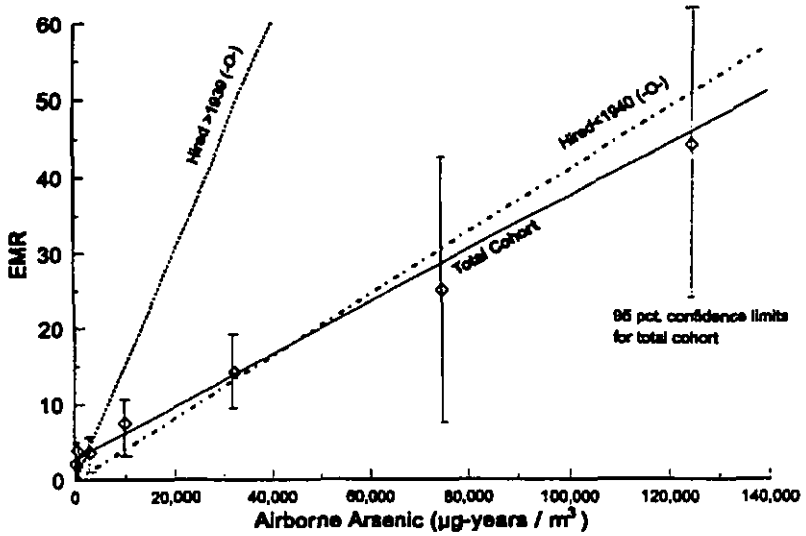

FIG. 3. Lung cancer risk and arsenic exposure in Swedish smelter workers: Estimates derived under the absolute model.

Summary Unit Risks

In this study the authors emphasized the sensitivity of the current global unit risk to the incorporation of new data. Pooled estimates were based on the geometric mean of the individual estimates making up the scenario of interest provided in Table 6. In Swedish analyses, the authors pooled the two subcohort estimates, giving a unit risk of 0.89×10^{-3} and a factor of 2 greater than the estimate for the total cohort (0.39×10^{-3}). This difference probably results from decomposing the cohort into smaller analytic units, while increasing the variability. For purposes of this investigation the authors accept 0.89×10^{-3} as the pooled result for the cohort. The actual risk is likely to be less.

The authors also take the view that there was sufficient reason to reject the 1984 unit risk estimate, developed by the EPA for the Tacoma cohort. If the dosimetry from the Pinto study were accepted, then the average arsenic exposure for Tacoma workers would be far below that observed in other smelter environments. Conversely, the 1987 dosimetry implies exposure intensity comparable to that observed elsewhere.

Three sets of pooled estimates are given in Table 6. Circumscribing the pooled estimate to findings in this study, the combined Ronnskar and Tacoma results produced a unit risk estimate of 1.07×10^{-3}, more than a factor of 4 less than the present IRIS unit risk. In scenario 2, the authors replicate the earlier EPA unit risk estimate, based on Tacoma and Montana, alone. Substituting the authors' new unit risk for Tacoma, and pooling this with the EPA's 1984 Montana estimate, gave a unit risk of 1.81×10^{-3}. This is about 40% less than the present unit risk estimate.

TABLE 6

UPDATED UNIT RISK ESTIMATES: POTENTIAL IMPACT ON THE CURRENT LUNG CANCER UNIT RISK OF 4.29×10^{-3}

Risk update	Smelter population	Estimated unit risk: Study	Estimated unit risk: Cohort	Pooled unit risk
Pooled estimate using updated Swedish and Tacoma cohorts	Tacoma, 1987	1.28×10^{-3}	1.28×10^{-3}	1.07×10^{-3}
	Ronnskar, 1989			
	Workers hired <1940	0.46×10^{-3}	0.89×10^{-3}	
	Workers hired 1940+	1.71×10^{-3}		
Updated Tacoma with original EPA estimates for Montana cohort	Tacoma, 1987			
	Updated results supercede earlier estimates		1.28×10^{-3}	1.81×10^{-3}
	Montana, 1984 (EPA)		2.56×10^{-3}	
	New estimates not available, 1984 EPA estimates apply			
Pooled across all smelter cohorts	Ronnskar, 1989		0.89×10^{-3}	1.43×10^{-3}
	Tacoma, 1987		1.28×10^{-3}	
	Montana, 1984 EPA		2.56×10^{-3}	

Finally, a pooled estimate from the combination of all studies yielded a unit risk of 1.43×10^{-3}, about a factor of 3 lower than the present estimate for airborne arsenic. Taken as a whole, the authors' analyses emphasize the importance of a realistic and complete exposure assessment, in developing quantitative estimates from epidemiologic data.

DISCUSSION

Incorporating information from the Ronnskar, Tacoma, and Anaconda smelter cohorts produced a risk estimate, a factor of 3 less than the present unit risk. This was attributable to the remarkable decline in the Tacoma risk estimate, based on revised arsenic dosimetry. This is a clear example of the importance of adequate exposure assessment and selection of the appropriate exposure metric in risk estimation. These new results indicate the serious needs to update the IRIS database and consider revision of the current unit risk for airborne arsenic exposure.

Although comparable estimates based on the multiplicative model were not presented, the unit risks were similar to those generated under the absolute model. In the EPA's earlier assessment of airborne arsenic, the model fit for the dose response (potency) was somewhat better under absolute risk assumptions. This observation is affirmed in the authors' analyses. In Swedish subcohort analyses using the relative risk model, unacceptable fits were evident ($P < 0.001$), when an estimate for background risk (intercept) was not included in the model. This is consistent with the smoking-related residual risk in this cohort. The unit risk based on the total cohort would have been about 0.87×10^{-3}, very comparable to the estimate derived under the absolute

risk model. The alternative estimate under the relative risk model for the Tacoma cohort was 0.97×10^{-3} and compares favorably to the 1.28×10^{-3} obtained under absolute risk assumptions.

The authors' analyses did not consider intrinsically nonlinear models for assessing risk. However, Enterline and Marsh (1987) demonstrated that a power fit of the SMR to dose (log–log regression) produced an excellent fit when applied to data tabulated in Table 2. The resulting fit, $SMR = 100 + 4.897\ (As)^{0.03499}$, explained about 98% of the variation in the SMR. Although not noted in the 1987 analysis, the corresponding linear regression performed equally well, with 93% of the variation in the SMR explained by the model $SMR = 169.1 + 0.0031\ (As)$. Both fits are remarkably good as confirmed by the corresponding $\chi^2_{(5\ df)}$ (0.799 and 0.138 for the linear and power models, respectively; $P < 0.97$ in both scenarios).

Applying the same models to data for the total Swedish cohort (from Table 4) demonstrated that the linear and power models, respectively, explained 97.8 and 64% of the variance in the SMR. The difference in the variance explained was statistically significant ($P < 0.05$) and the relatively poor performance of the power model was evident in the corresponding goodness of fit $\sigma^2_{(5\ df)}$ (1.75 and 9.14 for the linear and power models, respectively). Taking the results of both cohorts into account, little evidence to support an argument for nonlinear association as put forth by Enterline and Marsh (1982) is seen. This would also hold if the authors confined their view to the Tacoma cohort alone.

The authors did not explore new data on the Montana cohort in this study. This may be a limitation in terms of their present results. However, the authors concluded that major problems were still associated with exposure assessment for that cohort. The authors rejected consideration of a more recent study (Feldstein 1986, 1989) as the postulated upper range of exposure intensity would have been acutely toxic to many workers. Further, other significant risk factors have been identified as important determinants of lung cancer mortality in that cohort, independent of arsenic exposure, and need to be accounted for in any revision of the unit risk (Breslow, 1985). Still, in the context of the authors' estimates, the Montana unit risk stands as the upper bound for lung cancer risk.

Although the authors favor the new Tacoma unit risk over the previous estimate derived by EPA investigators, there are still questions that need resolution. Since there was no further mortality follow-up of this cohort, the difference in risk estimates relies, solely, on the veracity of the exposure metric. The new relationship established by Enterline *et al.* (1987) is highly consistent with results reported by others (Smith *et al.*, 1977; Vahter *et al.*, 1986; Pollisar *et al.*, 1990; Offergelt *et al.*, 1992). As shown in Table 7, the Enterline estimate is intermediate between the results of Vahter and

TABLE 7

NONLINEAR ESTIMATION OF THE RELATIONSHIP BETWEEN AIR AND URINE ARSENIC CONCENTRATION

Source	Regression
Enterline *et al.*, 1987	$As_{urine} = 13.48^{*}(As_{air})^{0.315}$
Vahter *et al.*, 1986	$As_{urine} = 18^{*}(As_{air})^{0.55}$
Offergelt *et al.*, 1992	$As_{urine} = 13.49^{*}(As_{air})^{353}$

Offergelt. These results are consistent with greater retention of arsenic with increasing dose, associated with diminished methylation capacity and increased half-life seen with higher arsenic body burdens.

In an experimental study similar to that of Pinto *et al.* (1976), Vahter *et al.* (1986) found it necessary to take account of worker hygiene, in assessing the relationship between air and urinary arsenic concentration. In a controlled study of 17 smelter workers where urinary and airborne arsenic were monitored, Vahter *et al.* noted that several workers had urinary excretion that exceeded levels attributable to arsenic uptake through inhalation alone. Indeed, ingestion through hand to mouth contamination was the main determinant in workers achieving relative urinary excretion rates exceeding those possible through inhalation. Following the same line of reasoning the authors looked at relative urinary arsenic excretion in the 24 workers studied by Pinto *et al.* (1976).

The authors assumed that workers breathed 10 m^3 of air during the work shift, that all arsenic inhaled was absorbed (100% uptake), and that urine excretion was 1.4 liters per day. Figure 4 shows the relative excretion of urinary arsenic with the individual workers average air arsenic concentration as reported by Pinto. Six of the 24 workers had a relative urinary excretion exceeding 100%. Clearly evident is the high relative excretion at very low exposure. As noted earlier, this could also result from the high background urinary arsenic concentration, from previous and unmeasured exposure. Relative excretion values substantially less than 50% would be expected from the results in other studies, and may be significantly less, depending on particle size, deposition, absorption, and half-life.

Many workers would have derived arsenic exposure by other pathways than through inhalation, reflecting organic arsenic from fish, and ingestion from hand to mouth contamination, while eating and smoking. Further, analytic methodology employed at the time might not have been sufficient to assess the association between urinary concentration and very low level air arsenic exposure. Preemployment urinary analyses on individuals with no known industrial exposure to arsenic, averaged about 53 μg/liter (Pinto *et al.*, 1976). This background concentration was about a factor of 5 greater

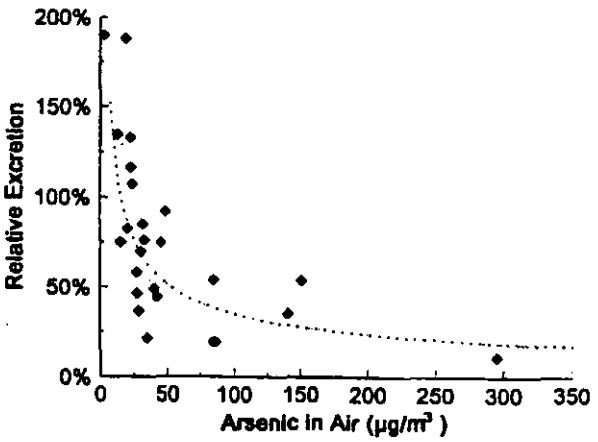

FIG. 4. Relative urinary arsenic excretion with average air concentration in 24 workers studied by Pinto.

than reported for other nonexposed populations (Vahter *et al.*, 1986; Farmer and Johnson, 1990) and a limiting factor in assessing the association between inhalation and urinary concentration at low exposure. In another context the background urinary concentration would be directly related to the notion of "enhanced bioavailability" of arsenic at a very low concentration as suggested by Enterline and Marsh (1982).

From a comparable analysis of the 1987 dosimetry, it is noted that the average relative excretion was about 20% for the complete range of exposure, with about 50% at an air concentration at or below 50 $\mu g/m^3$. This marked increase at low exposure is probably related to the increasing contribution of background to the measured urinary concentration, as airborne exposure decreased. Since the data represented air and urinary measurements covering the period 1948 through the mid-1970s, the background urinary arsenic concentration may have varied from well over 100 μg/liter (Pinto, 1953) to under 50 μg/liter depending on the analytic methods employed at the time.

In large part, these results confirm the insufficiency of the Pinto series for estimating the relationship between urinary arsenic and air exposure as used by the EPA, and caution against placing excessive confidence in the recent exposure data. It is likely that only explicit knowledge regarding metabolism and pharmacokinetics will resolve the association between inhaled arsenic and tissue bioavailability at low doses.

CONCLUSIONS

The primary evidence for arsenic-related carinogenisis comes from human epidemiologic studies. Using recent epidemiologic evidence from three smelter populations, this investigation provided updated estimates of the unit risk for arsenic-induced lung cancer resulting from lifetime exposure to airborne arsenic, Benchmarked against the earlier EPA estimates, a summary unit risk of 1×10^{-3} seems wholly consistent with the recently available epidemiologic data. This finding holds whether estimates were made under absolute or multiplicative risk assumptions, and is about a factor of 4 lower than that postulated by the EPA. This divergence in unit risk estimates can be directly attributed to the critical uncertainty in exposure estimation, evident in earlier analyses of the Tacoma and Montana cohorts.

In each case, the more recent exposure estimates suggest a range of exposure significantly greater than previously estimated. An appraisal of the 1987 Tacoma exposure estimates indicated reasonably good agreement with findings of other investigators. Still, the relationship between air and urinary arsenic was based on highly aggregated data, with some indication that the association at very low exposure was uncertain. New estimates from the Montana cohort have not been included in this study, since the most recent exposure profiles may grossly overestimate the upper range of exposure. It is likely, however, that the range of exposure was at least as great as seen that in the Swedish and Tacoma cohorts, implying significantly lower unit risks. Because of the critical impact of exposure on the underlying risk estimates, a detailed exposure reconstruction could be the best approach to resolving the uncertainties in future analyses. Finally the association between urinary and airborne arsenic needs further clarification in order to assess the relationship between inorganic arsenic and cancer of other sites, regardless of exposure pathway.

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APPENDIX I
INHALATION RISK ASSESSMENT: COMPUTATIONAL FRAMEWORK AND RESULTS

I.1 Overview

The computational framework was designed to efficiently generate risk estimates for hundreds of sources at a time. The framework directly estimates the aggregate impact of all chemicals, rather than estimating the impact of individual substances and then summing across substances to get aggregate results. For example, the framework allows for the direct calculation of total risk due to all 16 classes of substances from any given plant, however, it does not explicitly calculate the risk due the emissions of individual substances. [The average contribution values shown in the pie charts of contribution to risk by chemical (i.e., Figures 7-4 and 7-8) are based on each chemical's fraction of the total toxic-equivalent emissions of the plant. This approach assumes that the individual stacks of plant with multiple stacks have similar concentrations per unit emissions (χ/Q). This approach may not produce representative values when multiple stacks have differing χ/Q.] Since the CORE analysis would normally require about 10,000 dispersion runs (i.e., 1 run per substance, 16 substances per plant, about 600 plants), the computational framework provides an efficient means to generate aggregate results.

In the computational framework for the inhalation carcinogenic risk assessment, chemical-specific emissions estimates from the industry-wide emissions assessment (Chapter 4) are combined with chemical-specific potency values (in this case, unit risk factors) to produce a toxic-equivalent emission value for each unit. The toxic-equivalent emission value and source characteristics information (e.g., stack height and flue gas exit velocity) then serve as input for ISCLT2 dispersion simulations. Because the emissions input is a toxic-equivalent, the result of the simulation is a set of risk (rather than concentration) estimates. The next section describes the computations for inhalation MEI risk and hazard index. The two following sections describes the slight differences in the computations for the inhalation REI risk and hazard index. The final section describes the computation of the fuel index, a measure of the substances entering the boiler in the fuel.

I.2 Computations for Inhalation MEI Risk and Hazard Index

The ISCLT2 model requires as an input the chemical emission rates from one or more sources, and yields an estimate of the resulting ambient concentration at specified receptors. Operationally, ISCLT2 simulates the concentrations due to emissions from each source separately, and then sums them to get the total ambient concentration at each receptor. Because concentrations are proportional to emissions, ISCLT2 simulations can be used to determine the relationship between the emission rate of chemical i from a specific stack j ($Q_{i,j}$) and the resulting ambient concentration of chemical i at any receptor k ($\chi_{i,j,k}$):

$$\chi/Q_{i,j,k} = \chi_{i,j,k}/Q_{i,j} \quad (1)$$

This relationship, concentration per unit emissions (χ/Q), depends on the source characteristics, meteorology, terrain, and local dispersion characteristics. Since ISCLT2 does not incorporate any atmospheric chemistry, substance dispersion is based on purely physical mechanisms. Therefore, χ/Q is constant across chemicals, and the term reduces to $\chi/Q_{j,k}$.

Using the results of ISCLT2 simulations for each chemical, an indicator of total risk due to all chemicals is computed as the sum across chemicals of the product of each chemical's ambient concentration and a toxicity factor (T_i):

$$R_k = \sum_i (\sum_j (\chi_{i,j,k}) * T_i \quad (2)$$

Using the relationship between concentration and emission rate in Equation 1, a new expression is derived for the risk indicator:

$$R_k = \sum_i [\sum_j (Q_{i,j} * \chi/Q_{j,k}) * T_i] \quad (3)$$

For cancer risk calculations, the toxicity factors are unit risk factors. For hazard index calculations, the toxicity factors are the reciprocal of respective reference concentrations. Table I-1 shows unit risk factors and reference concentrations for the substances along with the sources of these data. Information on reference concentration values came from a variety of sources. The primary sources were the Integrated Risk Information System (IRIS) and the Health Effects Assessment Summary Tables (HEAST), both maintained by EPA. In the absence of IRIS or HEAST data, several alternate sources were used, including Threshold Limit Values (TLV) published by the American Conference of Governmental

Industrial Hygienists. The TLV values are based on occupational exposures and are translated for community exposures by scaling for exposure time differences between community and occupational exposure assumptions.

Table I-1.
Dose-Response Information and Sources

Substance	Carcinogenic		Non-Carcinogenic	
	Unit Risk $(\mu g/m^3)^{-1}$	Source	Reference Concentration (mg/m^3)	Source
Arsenic	$1.43x10^{-3}$	EPRI	$2.40x10^{-5}$	TLV
Beryllium	$2.40x10^{-3}$	IRIS	$4.76x10^{-6}$	TLV
Cadmium	$1.80x10^{-3}$	IRIS	$3.50x10^{-3}$	IRIS
Chromium	$6.00x10^{-4}$ *	IRIS	$2.00x10^{-6}$	CAPCOA
Manganese	—		$5.00x10^{-5}$	IRIS
Nickel	—		$2.38x10^{-3}$	TLV
Lead	—		$1.51x10^{-3}$	NAAQS
HCl	—		$7.00x10^{-3}$	IRIS
Mercury	—		$3.00x10^{-4}$	HEAST
Selenium	—		$5.01x10^{-4}$	CAPCOA
Benzene	$8.30x10^{-6}$	IRIS	$7.63x10^{-2}$	TLV
Formaldehyde	$1.30x10^{-5}$	IRIS	$8.82x10^{-4}$	TLV
PAH	$1.70x10^{-4}$	HEAST	$4.76x10^{-4}$	PEL
Dioxins / Furans	$3.30x10^{+1}$	HEAST	—	
Toluene	—		$4.00x10^{-1}$	IRIS

IRIS—Integrated Risk Information System, U.S. EPA
HEAST—Health Effects Assessment Summary Tables, U.S. EPA
TLV—Threshold Limit Value, American Conference of Governmental Industrial Hygienists (ACGIH)
CAPCOA—California Air Pollution Control Officers Association
PEL—Permissible Exposure Level, Occupational Safety and Health Administration (OSHA)
NAAQS—National Ambient Air Quality Standard
EPRI—Electric Power Research Institute
* Applies to hexavalent chromium

By reversing the order of the summations, Equation 3 can be rewritten as:

$$R_k = \sum_j [\sum_i (Q_{i,j} * T_i) * \chi / Q_{j,k}] \quad (4)$$

The CORE methodology combines stack emissions of all chemicals into a toxic-equivalent emission rate (TEQ_j) for input to ISCLT2. TEQ_j is the sum across chemicals of the product of the each chemical's stack emissions and unit risk factor:

$$TEQ_j = \sum_i (Q_{i,j} * T_i), \quad (5)$$

so that Equation 4 becomes:

$$R_k = \sum_j (TEQ_j * \chi / Q_{j,k}) \quad (6)$$

This approach requires only a single ISCLT2 simulation, supplemented by an algebraic manipulation of the ISCLT2 model input to account for chemical emission rates and their relative toxicity. Conceptually, the toxic-equivalent emissions (TEQ_j) can be thought of as "risk" units emitted from a stack, with the ISCLT2 simulation distributing the risk units to receptor points around the source.

I.3 Calculation of Inhalation REI Risk and Hazard Index

Estimating REI risks requires a slight adjustment to the computational framework to account for the impact of reasonable exposure assumptions. Along with stack emissions of all chemicals and potency values, the REI computation combines exposure multipliers (EMi, Appendix E) to obtain a toxic-equivalent emission rate (rTEQj) for reasonable exposure. rTEQj is the sum across chemicals of the product of the each chemical's stack emissions, its unit risk factor, and its exposure multiplier:

$$rTEQ_j = \sum_i (Q_{i,j} * T_i * EM_i) \quad (7)$$

Using this toxic-equivalent emissions measure in the ISCLT2 dispersion modeling produces REI risk estimates as outputs. As above, the toxicity factors for cancer risk calculations are unit risk factors and the toxicity factors for hazard index calculations are the reciprocal of reference concentrations. Also, there are different sets of exposure multipliers for carcinogenic and noncarcinogenic risks. Note that the emissions term (Qi,j) in the REI calculation accounts for plant replacement (Section E.7).

I.4 Analysis of Power Plant Inhalation Risk Results

This section provides additional information on the inhalation carcinogenic risk results presented in Section 7.3. The methods described were applied to results of the carcinogenic risk assessment for individual power plants, but could also be applied to noncarcinogenic risk results. A tabular ranking system is used to provide insight into the factors that may lead to risk estimates at the high end of all power plants analyzed, relative to the other power plants in the analysis. For the 30 plants with the highest overall risk estimates, the ranking system orders plants by their overall risk rank, ranks the plants with respect to components that drive overall risks (e.g., emission rates), and presents the values of key parameters that influence each component. For each component, the plants are ranked in descending order of the component's contribution to risk; for example, an emissions rank of 11 means the plant has the 11th highest emissions. This information is used to develop insight into the factors which tend to lead to high-end overall risk estimates, relative to other power plants (whether or not the risk estimates are high in an "absolute" sense).

The ranking system is designed to develop insights about relative risk levels and factors that influence risks. Since the emissions assessment and dispersion modeling rely on average substance-specific correlations and database information that may not be representative of long-term future operations of plants (e.g., particulate emissions), the risk estimates of any individual plant may reflect inaccuracies in the input data. Although the risk estimate of any given plant may be somewhat biased, it was assumed that the biases will balance in the aggregate results across plants.

I.4.1 Population Inhalation Carcinogenic Risk

Table I-2 displays the ranking system for the thirty plants with the highest annual population carcinogenic risks. The "Population Risk Rank" orders plants by their annual population risk, with 1 representing the plant with the highest risk, and is wholly determined by the population within 50 kilometers and the factors represented in the emissions and dispersion ranks.

Table I-2.
Characteristics of Plants with Highest Population Inhalation Carcinogenic Risk

Pop. Risk Rank	Fuel and Controls	Emissions Rank	Fuel Index	Est. Load (MWe)	Part. Emissions (lb/MMBtu)	Dispersion Rank	Dispersion Coefficient (R/U)	Approx. Height, Lowest Stack (ft)	Population Within 50 km
1	Coal-fired (bit / ESP) & controlled oil	48	3.2	440	0.10	207	U	330	HIGH***
2	Coal-fired with particulates only (bit / ESP)	6	3.3	360	0.38	219	U	550	MED
3	Coal-fired (bit / scrubber) & gas	138	1.6	600	0.04	91	U	250	HIGH
4	Coal-fired with particulates only (bit / ESP)	25	3.3	910	0.05	275	R	310	MED
5	Coal-fired with particulates only (bit / ESP)	225	1.8	300	0.03	30	U	170	MED
6	Coal-fired with particulates only (bit / ESP)	69	3.7	430	0.07	293	R	330	MED
7	Coal-fired (bit / ESP) & controlled oil	39	2.7	930	0.05	166	U	350	MED
8	Gas	328	0.0*	1430	—	88	U	300	HIGH
9	Coal-fired with particulates only (sub / ESP)	246	0.9	360	0.04	109	U	380	HIGH
10	Coal-fired (bit / ESP & sub / ESP)	107	3.2	250	0.07	273	R	240	MED
11	Coal-fired (bit / scrubber), controlled oil & gas	252	1.7	120	0.10	243	R	300	MED
12	Coal-fired with particulates only (bit / ESP)	22	1.8	530	0.15	336	R	250	MED
13	Coal-fired with particulates only (bit / ESP)	21	3.7	430	0.13	364	R	350	MED
14	Controlled oil & gas	336	0.0*	710	—	212	U	500	HIGH
15	Coal-fired with particulates only (bit / ESP)	192	3.7	50	0.19	27	U	230	LOW
16	Coal-fired with particulates only (bit / ESP)	15	1.8	1210	0.07	367	R	280	MED
17	Coal-fired with particulates only (bit / ESP)	58	3.3	410	0.08	401	R	260	MED

Table I-2.
Characteristics of Plants with Highest Population Inhalation Carcinogenic Risk (Continued)

Pop. Risk Rank	Fuel and Controls	Emissions Rank	Fuel Index	Est. Load (MWe)	Part. Emissions (lb/MMBtu)	Dispersion Rank	Dispersion Coefficient (R/U)	Approx. Height, Lowest Stack (ft)	Population Within 50 km
18	Coal-fired (bit / scrubber)	9	3.2	1490	0.05	532	R	600	MED
19	Coal-fired with particulates only (bit / ESP)	66	1.8	160	0.34	210	R	200	LOW
20	Coal-fired (bit / ESP) & gas	130	2.5	420	0.04	198	U	200	MED
21	Coal-fired (bit / scrubber)	18	2.7	1200	0.07	461	R	600	MED
22	Coal-fired (bit / ESP, sub / ESP & bit / scrubber)	29	2.3	1200	0.05	433	R	490	MED
23	Uncontrolled oil & gas	390	0.0	80	0.00	224	R	230	HIGH
24	Coal-fired with particulates only (sub / ESP)	221	0.9	360	0.05	112	U	400	HIGH
25	Coal-fired (bit / ESP), controlled oil & gas	102	2.7	460	0.06	298	R	260	MED
26	Coal-fired (bit / ESP & sub / ESP)	31	2.6	680	0.06	392	R	400	MED
27	Coal-fired with particulates only (sub / ESP)	315	0.8	210	0.04	136	U	450	HIGH
28	Coal-fired with particulates only (bit / ESP)	49	3.3	720	0.04	381	R	300	MED
29	Coal-fired with particulates only (sub / ESP)	100	0.8	890	0.05	440	R	450	MED
30	Coal-fired with particulates only (bit / ESP)	72	3.3	100	0.32	161	R	150	LOW
Statistical summary for all plants		median	1.8	230	0.04			270	
		mean	1.9	400	0.05			350	
		high	3.9	2350	1.1			1200	
		low	0.0*	2**	0.0017			40	

Table I-2.
Characteristics of Plants with Highest Population Inhalation Carcinogenic Risk (Continued)

Pop. Risk Rank	Fuel and Controls	Emissions Rank	Fuel Index	Est. Load (MWe)	Part. Emissions (lb/MMBtu)	Dispersion Rank	Dispersion Coefficient (R/U)	Approx. Height, Lowest Stack (ft)	Population Within 50 km
* Fuel index falls below rounding limit of 0.05.									
** Though nameplate capacity exceeds 25 MWe, estimated load reflects low capacity factor.									
*** Population classes: LOW: ≤1 million, MED: between 1 million and 5 million, HIGH: ≥5 million									
— Particulate emissions not used in emission calculations for oil and gas plants.									

The "Emissions Rank" column orders plants by a toxicity-weighted emissions measure. This measure is the sum, across substances emitted at each plant, of the emission rate of each substance, multiplied by the corresponding unit risk factor for that substance. This emissions measure accounts for the varying carcinogenicity of the different substances present in the emissions stream. The plant with the highest value of this measure is assigned an emissions rank of 1. Since this is a simple rank-ordering, there may be a number of plants grouped relatively quickly, and other similar plants with quite large gaps between them in the ordering. Factors that determine this emissions measure include carcinogenic fuel index (a measure of the equivalent toxicity of the mixture of trace substances emitted), estimated load, and particulate emissions. Section I.5 details the fuel index computations. The estimated load is based on the 2010 heat input and a nominal plant heat rate of 12,000 Btu/kWh. Particulate emissions are calculated from information in the particulate survey, the base case scenario, and public filings in industry databases.

The "Dispersion Rank" orders plants by their maximum concentration per unit emissions (χ/Q),with a value of one representing the plant with the highest χ/Q. χ/Q is the concentration of a substance in the air in a specific location resulting from a unit of emissions of that substance from the plant. Since χ/Q normalizes the concentration by emissions, it provides a basis for comparing concentrations of substances from plants of different size. Factors that influence χ/Q include (but are not limited to) whether urban or rural dispersion coefficients were assigned to the plant, and the plant stack height. The table displays the approximate height of the lowest stack at each plant.

About three-quarters of the plants with the highest population risks comprise at least one coal-fired unit, and have more than one million people residing within 50 kilometers. Typically, either their emissions or dispersion rank is in the highest 100 plants. For the (relatively) highest-risk plants as measured by population incidence, one or more of the three components that influence emissions (most frequently fuel index or load) tends to be significantly above the mean. The plants with the highest population risks tend to have rural dispersion coefficients, which tend to correspond to lower MEI risks, and the lowest

stack height at these plants tends to cluster around the average height across all plants. The emission rank tends to be higher than the dispersion rank for these plants, implying that emissions are usually the key driver for high population risks.

The plants with the highest annual population risks typically have relatively high population-weighted average risk, and a relatively large exposed population. All but four of the highest 30 population risk plants have over one million people within 50 kilometers, and 12 have over four million people within 50 kilometers. Of the thirty plants with the greatest population risks, 12 are also in the highest 30 for MEI risk.

I.4.2 Individual Inhalation Carcinogenic Risks

Table I-3 shows the characteristics of the thirty plants with the highest MEI inhalation carcinogenic risks. Like Table I-2, the "MEI Inhalation Carcinogenic Risk Rank" orders the plants by their MEI risk, with a value of one representing the plant with the highest risk. More than two-thirds of the high MEI risk plants have either an emissions or dispersion rank in the highest 100, and most plants have emission and dispersion ranks in the highest 300. For the highest-risk plants, one or more of the three components that influence emissions rank (fuel index, load, and particulate emissions) tends to be significantly above the average. Compared to the highest population risk plants in Table I-2, the high MEI risk plants tend to have poor dispersion. The dispersion coefficient of the plants with the highest MEI risks tends to be urban.

Table I-3.
Characteristics of Plants with Highest MEI Inhalation Carcinogenic Risk

MEI Inhalation Carcinogenic Risk Rank	Pop. Risk Rank	Fuel and Controls	Emissions Rank	Fuel Index	Est. Load (MWe)	Part. Emissions (lb/MMBtu)	Dispersion Rank	Dispersion Coefficient (R/U)	Approx. Ht of Lowest Stack (ft)
1	90	Uncontrolled oil	313	0.0*	210	—	1	U	140
2	2	Coal-fired with particulates only (bit / ESP)	6	3.3	360	0.38	220	U	550
3	15	Coal-fired with particulates only (bit / ESP)	192	3.7	50	0.19	27	U	230
4	5	Coal-fired with particulates only (bit / ESP)	225	1.8	300	0.03	30	U	170

Table I-3.
Characteristics of Plants with Highest MEI Inhalation Carcinogenic Risk (Continued)

MEI Inhalation Carcinogenic Risk Rank	Pop. Risk Rank	Fuel and Controls	Emissions Rank	Fuel Index	Est. Load (MWe)	Part. Emissions (lb/MMBtu)	Dispersion Rank	Dispersion Coefficient (R/U)	Approx. Ht of Lowest Stack (ft)
5	7	Coal-fired (bit / ESP) & controlled oil	40	2.7	930	0.05	167	U	350
6	3	Coal-fired (bit / scrubber) & gas	138	1.6	600	0.04	92	U	250
7	44	Coal-fired with particulates only (bit / ESP)	14	1.8	270	0.42	253	R	200
8	30	Coal-fired with particulates only (bit / ESP)	72	3.3	100	0.32	162	R	150
9	229	Uncontrolled oil	385	0.0*	60	—	4	U	160
10	43	Coal-fired with particulates only (sub / ESP)	157	0.9	540	0.05	91	U	200
11	1	Coal-fired (bit / ESP) & controlled oil	49	3.2	440	0.10	208	U	330
12	98	Coal-fired with particulates only (bit / ESP)	321	3.7	20	0.15	25	U	220
13	124	Coal-fired with particulates only (bit / ESP)	301	3.3	20	0.17	34	U	200
14	19	Coal-fired with particulates only (bit / ESP)	66	1.8	160	0.34	211	R	200
15	93	Coal-fired with particulates only (bit / ESP)	347	3.1	50	0.04	24	U	170
16	67	Coal-fired with particulates only (bit / ESP)	11	3.7	450	0.17	312	R	310
17	4	Coal-fired with particulates only (bit / ESP)	25	3.3	910	0.05	277	R	310

Table I-3.
Characteristics of Plants with Highest MEI Inhalation Carcinogenic Risk (Continued)

MEI Inhalation Carcinogenic Risk Rank	Pop. Risk Rank	Fuel and Controls	Emissions Rank	Fuel Index	Est. Load (MWe)	Part. Emissions (lb/MMBtu)	Dispersion Rank	Dispersion Coefficient (R/U)	Approx. Ht of Lowest Stack (ft)
18	39	Coal-fired (bit / ESP) & controlled oil	158	1.3	410	0.05	120	U	440
19	49	Coal-fired with particulates only (bit / ESP)	242	1.9	160	0.05	81	U	260
20	78	Coal-fired with particulates only (bit / ESP)	3	3.3	650	0.22	436	R	500
21	89	Coal-fired with particulates only (bit / ESP)	217	2.2	70	0.20	95	R	150
22	32	Coal-fired with particulates only (sub / ESP)	228	0.7	210	0.10	101	U	400
23	66	Coal-fired with particulates only (bit / ESP)	185	1.8	250	0.05	133	R	210
24	35	Coal-fired (bit / ESP), uncontrolled oil & gas	224	1.6	180	0.06	112	U	270
25	109	Coal-fired with particulates only (bit / ESP)	24	3.3	340	0.15	325	R	350
26	24	Coal-fired with particulates only (sub / ESP)	221	0.9	360	0.05	113	U	400
27	9	Coal-fired with particulates only (sub / ESP)	246	0.9	360	0.04	110	U	380
28	97	Coal-fired with particulates only (bit / ESP)	342	0.5	160	0.08	43	U	350
29	175	Coal-fired (bit / ESP & bit / scrubber)	63	2.7	780	0.05	282	R	400

Table I-3.
Characteristics of Plants with Highest MEI Inhalation Carcinogenic Risk (Continued)

MEI Inhalation Carcinogenic Risk Rank	Pop. Risk Rank	Fuel and Controls	Emissions Rank	Fuel Index	Est. Load (MWe)	Part. Emissions (lb/MMBtu)	Dispersion Rank	Dispersion Coefficient (R/U)	Approx. Ht of Lowest Stack (ft)
30	12	Coal-fired with particulates only (bit / ESP)	22	1.8	530	0.15	338	R	250
Statistical summary for all plants			median	1.8	227	0.04			270
			mean	1.9	404	0.05			350
			high	3.9	2345	1.1			1200
			low	0.0*	2**	1.7×10^{-3}			40

* Fuel index falls below rounding limit of 0.05.

** Though nameplate capacity exceeds 25 MWe, estimated load reflects low capacity factor.

— Particulate emissions not used in emission calculations for oil and gas plants.

An uncontrolled oil-fired plant and two bituminous coal-fired plants with ESPs have the highest MEI risks, which fall between 1×10^{-6} and 1.7×10^{-6}. Of the 30 plants with the highest MEI inhalation risks, 23 are coal-fired, five are fired with multiple fuels, and two are oil-fired. The plant with the highest MEI inhalation risk has the worst dispersion characteristics (partly due to its relatively short stacks and urban dispersion setting).

I.5 Calculation of Fuel Index

Table I-4 presents the carcinogenic and noncarcinogenic fuel index of coals used in the base case scenario, and Table I-5 presents corresponding fuel index data for coals used in alternative industry scenarios. The fuel index measures indicate the toxic-equivalent of trace substances entering or forming in the boiler per unit heat input. These measures are used in Tables I-2 and I-3 to indicate the relative toxicity of the fuels combusted in utility boilers.

Table I-4.
Fuel Index of "as-fired" Coals for the Base Case Scenario

				Fuel Index	
State	Coal Supply Region	Rank	Sample Size	Carcinogenic	Non-Carcin-ogenic
Alabama	Southern Appalachian	Bit	1	3.9	$1.0x10^{+6}$
Colorado	Green River	Bit	26	0.4	$1.5x10^{+5}$
Illinois	Eastern	Bit	47	1.8	$6.8x10^{+5}$
Indiana	Eastern	Bit	80	1.9	$6.2x10^{+5}$
Kentucky	Central Appalachian	Bit	337	3.3	$6.9x10^{+5}$
Kentucky	Eastern	Bit	116	2.7	$9.1x10^{+5}$
Maryland	Northern Appalachian	Bit	38	3.0	$8.4x10^{+5}$
New Mexico	San Juan River	Bit	3	0.6	$2.8x10^{+5}$
Ohio	Northern Appalachian	Bit	492	3.1	$7.0x10^{+5}$
Pennsylvania	Northern Appalachian	Bit	539	3.7	$8.0x10^{+5}$
Tennessee	Central Appalachian	Bit	12	3.8	$5.0x10^{+5}$
Utah	Uinta	Bit	22	0.3	$1.7x10^{+5}$
Virginia	Central Appalachian	Bit	52	1.8	$4.9x10^{+5}$
West Virginia	Central Appalachian	Bit	280	1.9	$6.0x10^{+5}$
West Virginia	Northern Appalachian	Bit	101	2.2	$6.2x10^{+5}$
			Median	2.2	*$6.2x10^{+5}$*
Colorado	Green River	Sub	1	0.3	$1.0x10^{+5}$
Montana	Powder River	Sub	95	0.8	$2.3x10^{+5}$
New Mexico	San Juan River	Sub	104	1.8	$5.6x10^{+5}$
Wyoming	Green River	Sub	2	1.0	$5.0x10^{+5}$
Wyoming	Powder River	Sub	141	0.9	$4.0x10^{+5}$
			Median	0.9	$4.0x10^{+5}$
North Dakota	Fort Union	Lig	56	1.8	$5.0x10^{+5}$
Texas	Texas	Lig	54	1.4	$8.0x10^{+5}$

Table I-4.
Fuel Index of "as-fired" Coals for the Base Case Scenario (Continued)

				Fuel Index	
State	Coal Supply Region	Rank	Sample Size	Carcinogenic	Non-Carcinogenic
			Median	1.6	$6.2x10^{+5}$
			Min	0.3	$1.0x10^{+5}$
			Median	1.8	$5.8x10^{+5}$
			Mean	1.9	$5.6x10^{+5}$
			Max	3.9	$1.0x10^{+6}$
		Uncontrolled Oil		$1.4x10^{-2}$	$3.8x10^{+3}$
			Gas	$1.5x10^{-3}$	$6.1x10^{+2}$

The carcinogenic fuel index of fuel f ($cTEC_f$) is defined as the sum, across inorganic substances, of the concentration of substance i in the fuel ($C_{i,f}$) multiplied by its unit risk factor (T_i)plus the sum, across organic compounds, of the emission factor of organic compound *o* ($EF_{o,f}$) multiplied by its unit risk factor (T_o):

$$cTEC_f = \sum_i (C_{i,f} * T_i) + \sum_o (EF_{o,f} * T_o) \quad (8)$$

The noncarcinogenic fuel index ($nTEC_f$) is the sum, across substances, of the concentration of the substance in the fuel divided by its reference concentration (RfC_i) plus the sum, across organic compounds, of the emission factor of the organic compound divided by its reference concentration (RfC_o):

$$nTEC_i = \sum_i (C_{i,f} / RfC_i) + \sum_o (EF_{o,f} / RfC_o) \quad (9)$$

Since noncarcinogenic and carcinogenic fuel indices address different types of endpoints, they are not meant to be compared.

For the base case, bituminous coals have the highest median carcinogenic fuel index at 2.2, 40% and 145% greater than the medians of lignite and subbituminous coals, respectively. For perspective, the carcinogenic fuel index of oil and gas are significantly lower at values of $1.4x10^{-2}$ and $1.5x10^{-3}$. This implies that, on a unit Btu basis, bituminous coals have higher concentrations of relatively-toxic trace substances than other fuels. Switching from the median bituminous to subbituminous coal will reduce trace substance input to the

boiler. The extent to which switching reduces trace substance emissions will depend upon the change in ash characteristics and their subsequent impact on the fraction of the substance that ends up as bottom ash and the performance of control devices.

As shown in Table I-4, of the coals with more than 10 samples tested, Tennessee and Pennsylvania bituminous coals have the highest carcinogenic fuel indices at about 3.8. Five other bituminous coals have carcinogenic fuel indices greater than 2. All of the subbituminous and lignite coals have carcinogenic fuel indices less than 2. Of the coals with more than ten samples, Wyoming and Montana Powder River coals have the lowest carcinogenic fuel indices at about 0.9.

The base case lignite coals have the highest median noncarcinogenic fuel index, 10% and 70% greater than the medians of bituminous and subbituminous coals, respectively. The noncarcinogenic fuel indices of oil and gas are significantly lower than coals at values of $3.8x10^3$ and $6.1x10^2$, respectively. Of the coals with more than 10 samples, Eastern Kentucky bituminous and Texas lignite have the highest noncarcinogenic fuel indices at $9.1x10^5$ and $8.1x10^5$, respectively. Utah Uinta, Colorado Green River, and Montana Powder River coals have the lowest noncarcinogenic fuel indices at values ranging from $1.5x10^5$ to $2.1x10^5$.

Table I-5 summarizes the average calculated indices for coals from the major mining regions based on the alternative coal quality characterization. In Table I-5, the fuel index of each coal category (several per region) is weighted by that coal's projected burn under the HTp alternative scenario. Note that this does not distinguish scrubbed versus unscrubbed units.

Table I-5.
Fuel Index of Major Coal Producing Regions For Alternative Industry Scenarios

		Fuel Index	
Coal Category	Sample Groupings	Carcinogenic	Non Carcinogenic
Northern Appalachia		1.6	$6.4x10^{+5}$
NA7	NA7- Avg	1.9	$7.5x10^{+5}$
NA6	Pittsburgh #8	1.7	$7.7x10^{+5}$
NA5	Pittsburgh #8	1.1	$5.8x10^{+5}$
NA4/2/1	NA4/2/1 Avg	1.8	$6.5x10^{+5}$
NA3	Pittsburgh #8	1.2	$5.1x10^{+5}$
Central Appalachia		1.6	$6.3x10^{+5}$
CA3	Mid Sulf Avg	2.2	$7.9x10^{+5}$

Table I-5.
Fuel Index of Major Coal Producing Regions For Alternative Industry Scenarios (Continued)

		Fuel Index	
Coal Category	Sample Groupings	Carcinogenic	Non Carcinogenic
CA2	Low Sulf Avg	1.7	$6.6x10^{+5}$
CA1	Compliance Avg	1.3	$5.5x10^{+5}$
CA0	Pocahontas #3	1.8	$4.0x10^{+5}$
Southern Appalachia		2.3	$7.1x10^{+5}$
SA3/4	Pratt	3.3	$5.3x10^{+5}$
SA2	MaryLee	2.3	$5.7x10^{+5}$
SA1	Blue Creek	1.8	$8.9x10^{+5}$
Illinois Basin		2.0	$9.6x10^{+5}$
IE5	IE5 Average	2.0	$1.1x10^{+6}$
IE4	WK #9	1.9	$8.5x10^{+5}$
IE3	INVII	2.1	$9.3x10^{+5}$
IE2	IE2 Total	1.9	$6.6x10^{+5}$
IE0	INVII	1.5	$6.6x10^{+5}$
Powder River Basin		0.6	$2.6x10^{+5}$
PR3	Wyodak-Low Btu	0.3	$1.8x10^{+5}$
PR2	Dietz	0.7	$4.3x10^{+5}$
PR1	Wyodak-High Btu	0.7	$2.5x10^{+5}$
PR4	Rosebud	0.5	$2.4x10^{+5}$
Rockies		0.5	$2.4x10^{+5}$
CW1	Rockies Avg	0.5	$2.4x10^{+5}$
South Wyoming		0.1	$1.1x10^{+5}$
SY1	SWY-Compl	0.3	$1.9x10^{+5}$
SY2	Deadman	0.1	9.4x10+4
Arizona-New Mexico		0.7	$3.7x10^{+5}$
SW1	AZ-Compl	0.6	$2.9x10^{+5}$
SW2	NM-Comp	0.8	$3.9x10^{+5}$

Table I-5.
Fuel Index of Major Coal Producing Regions For Alternative Industry Scenarios (Continued)

		Fuel Index	
Coal Category	Sample Groupings	Carcinogenic	Non Carcinogenic
SW3	AZ-Low Sul	0.8	$3.9x10^{+5}$
SW4	NM-LS	1.1	$4.9x10^{+5}$
Other Western		0.7	$4.2x10^{+5}$
PC1	WA/AK	0.1	$1.0x10^{+5}$
IW1/5	Missouri/Iowa	2.6	$1.3x10^{+6}$
Lignite		1.6	$1.2x10^{+6}$
NW	Beulah Zap	1.8	$6.2x10^{+5}$
GC	Wilcox	1.6	$1.4x10^{+6}$
Import			
IM2	South American Compliance	0.5	$2.7x10^{+5}$
IM1	Canadian Compliance	0.6	$2.8x10^{+5}$
Anthracite			
AN	PA Anthracite	5.2	$2.3x10^{+6}$

The fuel index calculations for the alternative scenarios give lower carcinogenic index nationally, and for Appalachian and Powder River coals in particular (Table I-5), than do the base case calculations (Table I-4), even without considering the higher projected scrubbing in the alternative HTp scenario. The main reason for the lower calculated carcinogenic fuel index is the lower arsenic levels assigned to most coals in the alternative characterization.

In contrast, the alternative scenario and coal characterization give a calculated nationwide noncarcinogenic fuel index roughly equal to the base case calculations. This is apparently due mainly to the higher chromium levels assigned to many coals under the alternative characterization.

APPENDIX J
TRUE MULTIMEDIA RISK ASSESSMENT MODEL

J.1 Introduction

EPRI's "Total Risk of Utility Emissions" (*TRUE*) model is a tool for multimedia health risk assessment that can be run at either a screening or detailed level [3]. An overview of the model was presented in Section 8.3 of this report. This Appendix provides a brief description of the model structure, input requirements, and output information.

J.2 Model Description

TRUE is structured around the WTRISK model [1]. WTRISK acts as the core program that stores and handles general information about the study area (e.g., subregion division, location of water bodies, etc.) and coordinates the subprograms performing the environmental transport, food chain, exposure/dose, and risk calculations. All environmental fate and transport model components are connected to the WTRISK core program as external subroutines.

The model study area is defined by a radius of up to 100 km around the power plant. In the case studies presented here, a radius of 50 km was used since such an area covered most of the water bodies of interest and was consistent with the inhalation risk assessment spatial resolution. The study area is divided into population subregions that are defined by angular sectors and radial divisions. Environmental concentration, exposure/dose, and health effect calculations for a hypothetical *Maximally Exposed Individual (MEI)* are performed within each subregion.

The atmospheric fate and transport model (ISCLT2) [4]handles the model study area as a whole, and calculates the steady-state atmospheric concentrations at receptors placed on a polar grid with 10 degree angular increments and at various radial spacings. The average concentration and deposition rate in each subregion is calculated, and the maximum atmospheric concentration in the subregion is selected to be transferred to the exposure/dose calculation subroutines. The amount of chemical deposited on the ground surface is distributed among overland runoff and soil infiltration.

A separate soil chemical fate and transport simulation is performed for each subregion in the study area. The vadose zone of each subregion represents a separate soil compartment, which takes a constant chemical load applied to its surface. The SESOIL [2] simulation is a transient simulation, starting with a clean environment. The simulation period is 70 years, which is assumed to be an upper bound on the lifetime of a power plant. An average surface soil concentration for the subregion is calculated over the 70-year period for later use by the exposure/dose calculations.

Transient chemical releases to groundwater calculated in SESOIL are used as input to the AT123D model [5] to predict transient groundwater chemical concentrations. For each subregion, a time-averaged groundwater concentration is calculated for the 70-year exposure period, and is transferred back to the WTRISK core program for later use by the food chain and exposure/dose subroutines.

A surface water fate and transport simulation is performed by the WTRISK surface water model for each surface water body in the study area. An average surface water concentration is calculated for each water body and is transferred back to the WTRISK core program for later use by the food chain and exposure/dose subroutines.

An average concentration of the general-use water is calculated for each subregion. This calculation is based on the fraction of the subregion's water consumption that is drawn from surface or groundwater sources, as well as the fraction of the surface water part that is drawn from each individual surface water body. All groundwater used in a subregion is assumed to be drawn from the aquifer portion underlying the same subregion.

A separate food chain chemical transport simulation is performed for each subregion using the environmental concentrations that were calculated for the subregion by the environmental fate and transport models as input.

The subregions' chemical concentrations in the different components of the food chain and the different environmental media are transferred to the exposure/dose subroutines, where the dose for each specific exposure pathway is calculated. The carcinogenic (risk) and noncarcinogenic (hazard index) health effects are calculated for all chemicals and pathways through the use of chemical- and pathway-specific cancer potency factors and reference doses.

J.3 References

1. Bolton, J. G., "User's Guide to the Water Emissions Risk Assessment Model (WTRISK)," Working Draft. WD-3725-3-EPRI, Electric Power Research Institute, Palo Alto, California. 1989.
2. Bonazountas, M., and J.M. Wagner, 1984. SESOIL—A Seasonal Soil Compartment Model. Designed for U.S. EPA. Contract 68-01-6271, Arthur D. Little, Inc., Cambridge, Mass.

3. Constantinou, E. and Seigneur C. "A Mathematical Model for Multimedia Health Risk Assessment" Environ. Software, Vol. 8, pp. 231-246, 1993.

4. U.S. Environmental Protection Agency. 1992. User's Guide for the Industrial Source Complex (ISC2) Dispersion Models. Volume 1—User Instructions. EPA-45014-92-008a. Office of Air Quality Planning and Standards, Technical Support Division. Research Triangle Park, NC.

5. Yeh, G.T., 1981. AT123D: Analytical Transient One-, Two-, and Three-Divmensional Simulation of Waste Transport in the Aquifer System. Publication No. 1439. ORNL-5602, Oak Ridge National Laboratory. Environmental Sciences Division. Oak Ridge, TN.

APPENDIX K
TRUE MULTIMEDIA RISK ASSESSMENT CASE STUDIES

K.1 Introduction

The TRUE model was used to perform screening-level multimedia health risk assessments associated with the emissions of four fossil-fueled power plants in the United States. These plants were selected from the set of plants measured by the EPRI PISCES program. A brief summary of the findings of these assessments was presented in Chapter 8 of this report. This Technical Appendix provides a more detailed description of the sites, modeling approach and results.

K.2 Case Studies

The four power plants discussed in this section include three coal and one oil-fired. The three coal-fired are: (i) PISCES Site 12 in upstate New York, (ii) Site A in a rural area in the Midwest, and (iii) Site B in the vicinity of a large city in the east. The oil fired one is PISCES Site C located in a coastal suburban environment in the northeast.

The four plants are all equipped with electrostatic precipitators (ESPs) for particulate control and have comparable stacks ranging from 183 to 229 m high. A comparison of the characteristics of the four facilities is provided in Table K-1. The corresponding stack air emissions, as measured in the PISCES program and scaled to 2010 power plant annual capacity, are presented in Table K-2.

Table K-1.
***TRUE* Case Studies — Facility Characteristics**

	Site 12	Site A	Site B	Site C
• Fuel	Bituminous Coal	Subbituminous Coal	Bituminous Coal	Residual Oil
• Particulate Control	ESP	ESP	ESP	ESP
• SO2 Control	Wet Limestone FGD	Wet Limestone FGD	Coal Blending	—

Table K–1.
***TRUE* Case Studies — Facility Characteristics (Continued)**

	Site 12	Site A	Site B	Site C
• Stack Characteristics				
Temperature (K)	323	359	415	444
Velocity (m/s)	13	22	26	17.3
ESP: Electrostatic Precipitator FGD: Fuel Gas Desulfurization				

Table K–2.
***TRUE* Case Studies — Chemical Emission Rates**

Chemical	Emission Rates (g/s)				
	Site 12	Site A	Site B	Site C	Nature of Emissions
Arsenic	3.10×10^{-4}	9.27×10^{-4}	5.61×10^{-3}	$<1.8 \times 10^{-3}$	P
Beryllium	$<1.1 \times 10^{-4}$	$<1.74 \times 10^{-3}$	1.71×10^{-4}	6.0×10^{-4}	P
Cadmium	7.98×10^{-4}	2.09×10^{-3}	1.34×10^{-3}	3.31×10^{-4}	P
Chromium (Total)	2.45×10^{-3}	6.95×10^{-3}	6.0×10^{-3}	4.8×10^{-3}	P
Chromium (VI)	NS(2)	NS	NS	$<5.69 \times 10^{-4}$	P
Copper	3.19×10^{-3}	$<1.74 \times 10^{-3}$	3.11×10^{-3}	6.70×10^{-3}	P
Lead	3.99×10^{-3}	1.39×10^{-2}	2.14×10^{-3}	4.6×10^{-3}	P
Manganese	1.11×10^{-3}	4.36×10^{-3}	4.09×10^{-3}	1.52×10^{-2}	P
Mercury	9.4×10^{-4}	1.28×10^{-2}	3.05×10^{-3}	2.4×10^{-4}	G or P(4)
Molybdenum	$<2.76 \times 10^{-3}$	$<4.65 \times 10^{-3}$	1.1×10^{-2}	6.1×10^{-3}	P
Nickel	3.06×10^{-3}	4.36×10^{-3}	2.5×10^{-3}	3.1×10^{-1}	P
Selenium	9.2×10^{-3}	1.85×10^{-3}	2.5×10^{-2}	$<4.1 \times 10^{-3}$	P
Vanadium	$<1.11 \times 10^{-3}$	2.27×10^{-3}	6.1×10^{-3}	$<2.5 \times 10^{-1}$	P
Chlorine Compounds	1.72	1.57	19.3	3.72	G or P(4)
Fluorine Compounds	1.79×10^{-2}	1.22×10^{-1}	1.71	4.8×10^{-1}	G or P(4)
PAHs	$<1.66 \times 10^{-3}$(2)	$<4.65 \times 10^{-3}$(2)	$<3.11 \times 10^{-3}$(2)	$<9.6 \times 10^{-4}$	P or G(4)
Benzene	4.90×10^{-4}	3.20×10^{-3}	6.0×10^{-2}	2.42×10^{-3}	G

Table K-2.
***TRUE* Case Studies — Chemical Emission Rates (Continued)**

Chemical	Emission Rates (g/s)				
	Site 12	Site A	Site B	Site C	Nature of Emissions
Formaldehyde	$<5.77 \times 10^{-4}$	$<1.16 \times 10^{-2}$	$<2.3 \times 10^{-3}$	1.38×10^{-2}	G
Toluene	7.40×10^{-4}	1.40×10^{-3}	7.9×10^{-2}	8.23×10^{-2}	G

These emission rates represent annual capacity power plant values.
<: Chemical was not detected; detection limit shown
NS: Not Sampled (i.e., no speciation sampling was performed for Chromium (VI)
P: Particulate
G: Gaseous

(1) In the application, total chromium emissions were considered to consist of 5% Cr (VI) and 95% Cr (III).
(2) The detection limit of PAHs corresponds to a single compound.
(3) The detection limit given for PAHs corresponds to the weighted sum of 7 carcinogenic compounds using their relative potency to BaP.
(4) The chemical nature (P,G) listed first was used in the model application.

The domain of each risk assessment was the area within 50 km of the corresponding facility. Each study domain was further subdivided into 40 subregions defined by 8 angular sectors of 45 degrees each, and five radial divisions from 10 to 50 km from the facility. Carcinogenic and noncarcinogenic health risk calculations were performed for each one of the subregions. The subregions can be referred to by their sector and radial indexes. The sectors are numbered in a clockwise direction from the north, and the radii are numbered from the facility outward. For example, subregion (2,3) represents sector 2 (i.e., northeast), radial division 3 (i.e., 20-30 km from the plant).

The areas surrounding the four facilities exhibit significant differences with respect to the meteorological, climatological, hydrological and hydrogeological characteristics, as well as water and fish supply sources. These differences result in the estimation of significantly variable risks both between different sites and between different locations (i.e., subregions as used in the TRUE analysis) at the same site. Schematic descriptions of the four study areas including the major hydrological features and subregion division are presented in Figures K-1 through K-4. Table K-3 provides a comparison of the major

characteristics of the environmental settings for the four sites. These characteristics include: prevailing wind direction, hydrological balance, surface water bodies, and groundwater aquifer dimensions.

Table K–3.
***TRUE* Case Studies – Characteristics of Environmental Media**

	Site 12	Site A	Site B	Site C
• **Wind Direction**	West/Southwest	South	Northwest	West
• **Hydrological Balance**				
- Precipitation (cm)	95.5	86.4	104.0	119.0
- Evapotranspiration (cm)	65.5	69.6	51.0	85.0
- Overland Runoff (cm)	14.0	16.5	19.0	14.0
- Infiltration (cm)	16.0	0.3	34.0	20.0
• **Surface Water Bodies**	1 large river 1 large lake	1 river 2 lakes 2 creeks	5 rivers 1 small reservoir	1 large river 1 small river 2 small reservoirs 1 marine area
• **Groundwater System**				
- Unsaturated zone (m)	N/A	8.0	8.5	11.0
- Saturated zone (m)		11.0	30.0	61.0

N/A = not applicable

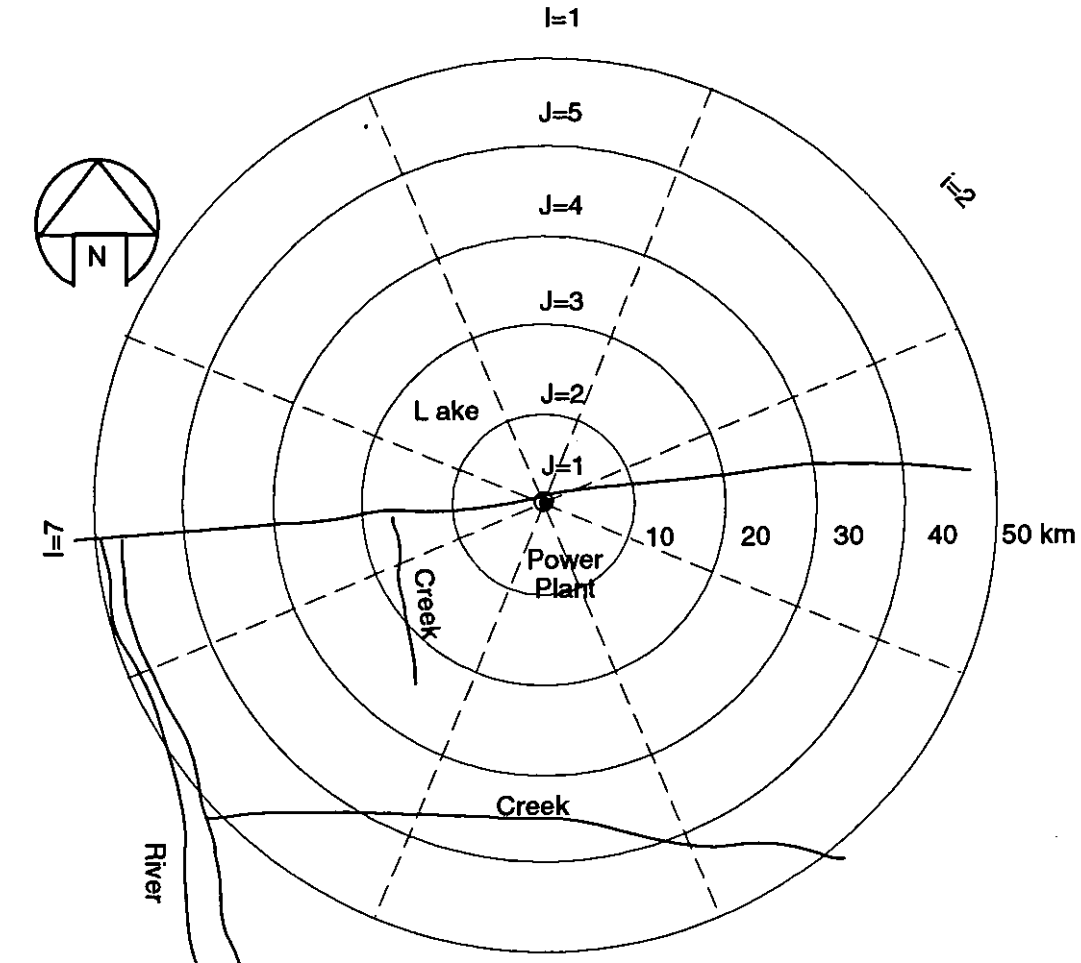

Figure K-1.
Model Study Domain—Site 12

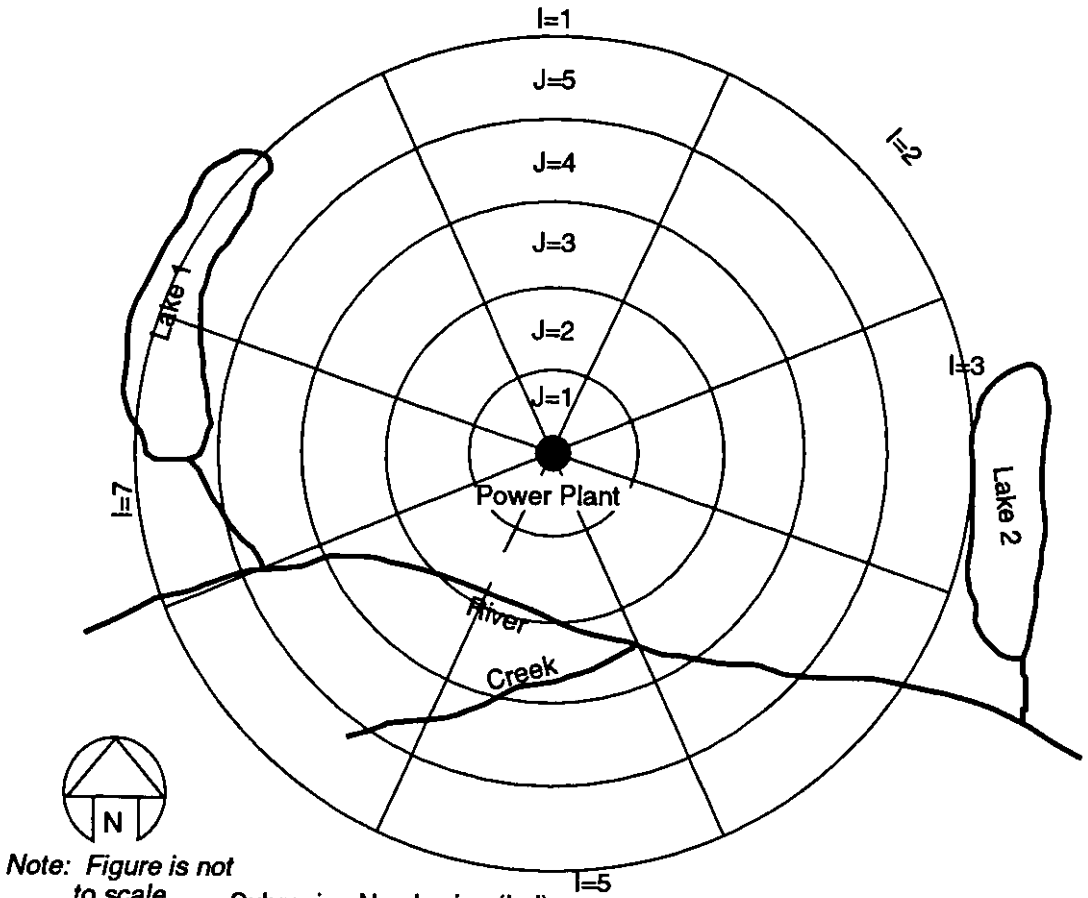

Figure K-2.
Model Study Domain—Site A

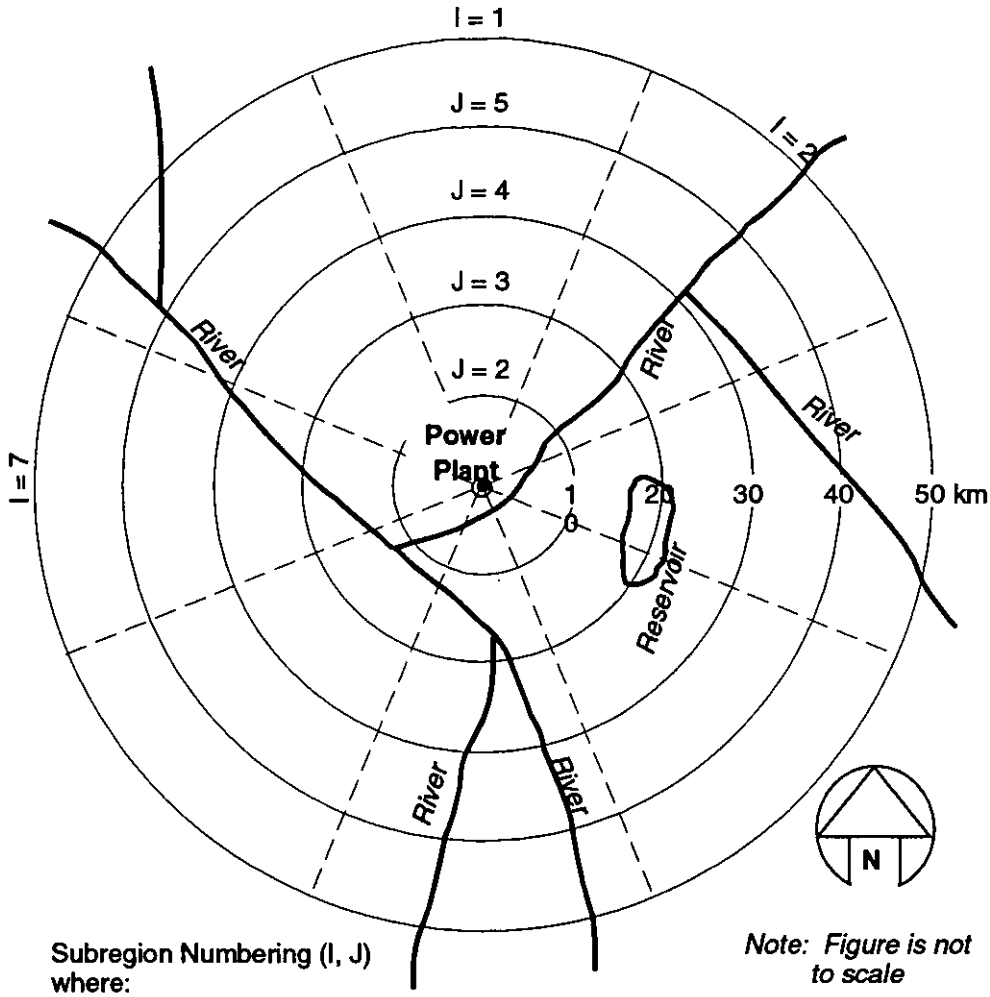

Figure K-3.
Model Study Domain—Site B

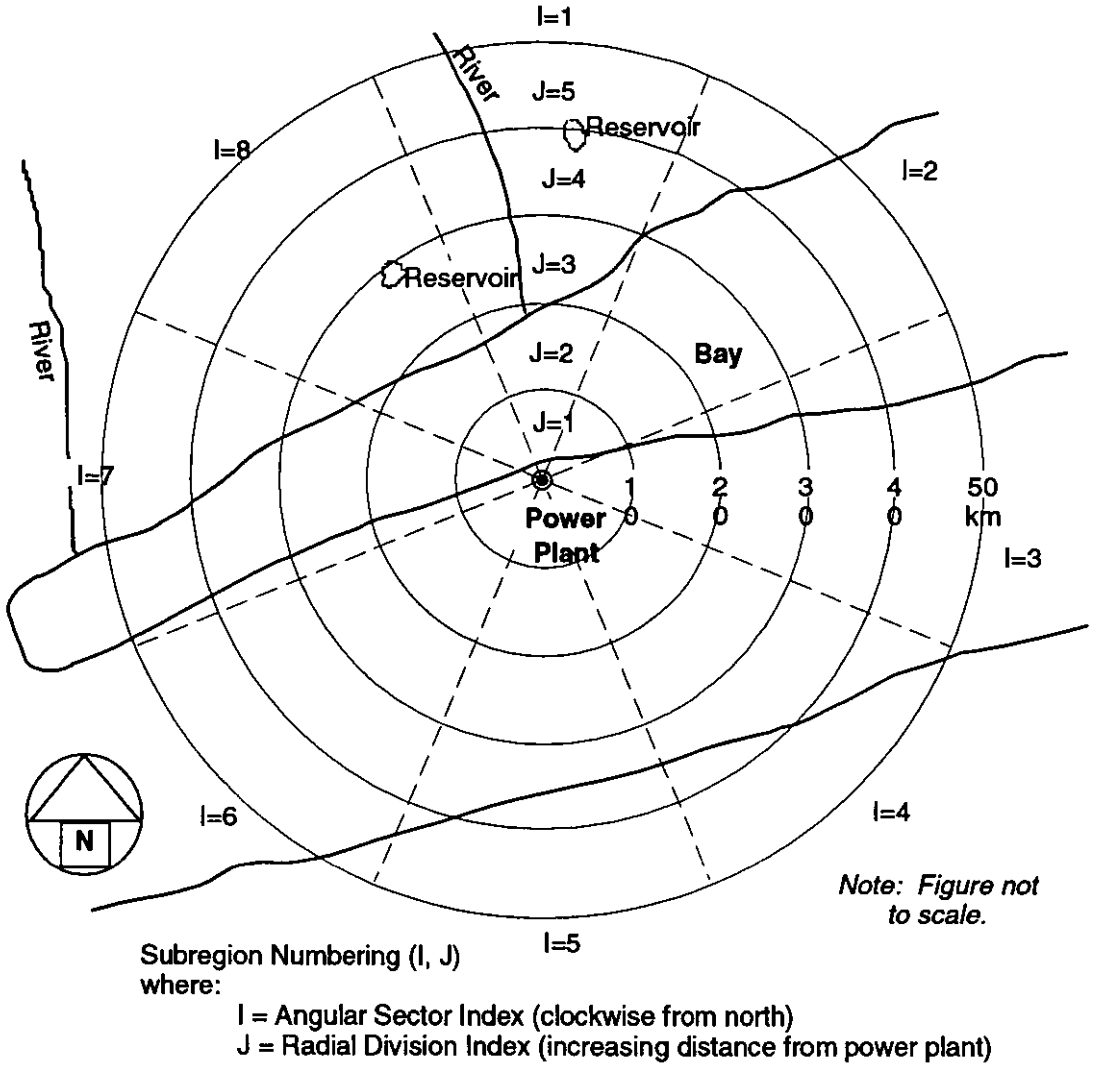

Figure K-4.
Model Study Domain—Site C

Ground-level chemical concentrations in the air were calculated for a total of 1500 receptors placed on a polar grid with 10-degree increments, and at radial spacings ranging from 100 to 2,000 m, depending on the proximity to the power plant. The maximum calculated concentration-to-emission rate ratios (χ/Q) for the four cases were comparable (within a factor of 2). A comparison of the selected options and results of the atmospheric transport modeling is presented in Table K-4.

Table K-4.
***TRUE* Case Studies - Atmospheric Transport Modeling Options/Results**

	Site 12	Site A	Site B	Site C
Environment	Rural	Rural	Rural	Rural
Terrain	Flat	Rolling hills	Rolling hills	Rolling hills
Max χ/Q				
— Location				
Direction	east	north	east	east
Distance	10 to 20 km	0 to 10 km	20 to 30 km	10 to 20 km
— Value (μg/m3)/(g/s)	2.0×10^{-3}	1.6×10^{-3}	2.9×10^{-3}	1.6×10^{-3}

Surface water concentration calculations in each case were performed for the water bodies identified as major contributors to the area's public water and fish supplies. The input chemical loads to these water bodies were due to overland runoff and were based on the previously calculated deposition rates and the corresponding drainage areas. The resulting chemical concentrations varied depending on the flow and geometric characteristics of the different water bodies.

A 70-year simulation of chemical infiltration was performed, and an average surface soil concentration over the simulation period was calculated for the top 5 cm. soil layer of each subregion. These soil concentrations were used by the food chain and health effect models for the estimation of the chemical concentration in produce, as well as for the estimation of the human exposure dose due to ingestion and dermal contact with soil.

The chemical releases to the water table were passed as input to the groundwater model. In all cases the resulting chemical concentrations in groundwater were very small.

Table K-5 summarizes the ranges of calculated concentrations in the different environmental media for an index chemical in each case study. The emission rates correspond to 2010 annual-average capacity of the power plants. The chemical selected as index in each case was the one identified as the major contributor to carcinogenic risk.

Table K–5.
***TRUE* Case Studies – Environmental Concentrations**

	Site 12	Site A	Site B	Site C
Index Chemical (Qe(g/s))	Arsenic (3.1×10^{-4})	Arsenic (9.3×10^{-4})	Arsenic (5.6×10^{-3})	Beryllium (6.0×10^{-4})
Air Concentration Range (µg/m3)	8.5×10^{-8} to 6.2×10^{-7}	2.2×10^{-7} to 1.5×10^{-6}	1.5×10^{-6} to 1.6×10^{-5}	1.2×10^{-7} to 7.4×10^{-6}
Soil Concentration Range (mg/kg)	2.7×10^{-6} to 7.5×10^{-5}	3.1×10^{-7} to 9.0×10^{-6}	3.1×10^{-5} to 1.9×10^{-3}	9.0×10^{-6} to 3.8×10^{-4}
Surface Water Concentration Ranges (mg/l)	• Niagara River 6.2×10^{-8} to 1.2×10^{-7} • Lake Ontario 3.9×10^{-6}	• Creeks 2.7×10^{-4} to 7.0×10^{-4} • River 9.3×10^{-7} to 1.1×10^{-4} • Lake #1 3.8×10^{-5} • Lake #2 1.8×10^{-4}	• River System 9.2×10^{-7} to 9.7×10^{-5} • Reservoir 1.3×10^{-2}	• Small River 2.5×10^{-6} to 5.2×10^{-5} • Large River 3.1×10^{-8} to 3.6×10^{-7} • Reservoir 1 3.4×10^{-5} • Reservoir 2 3.83×10^{-5} • Marine 5.4×10^{-7}
Groundwater Concentration Ranges (µg/l)	NA	10^{-13} to 10^{-11}	10^{-11} to 10^{-9}	10^{-10} to 10^{-8}
Qe = stack air emission rate of chemical NA: Groundwater is not being used in the area				

The calculated environmental concentrations of chemicals in the different subregions were used for the calculation of the corresponding concentrations in the different food chain components, which were subsequently used to calculate the chemical exposure dose of the MEI in each subregion. The values used for the parameters characterizing human exposure are summarized in Table K-6.

Table K–6.
Selected Dose Parameters[1]

Parameters	Selected Value
Exposure duration	70 years
Body weight	70 kg
Inhalation rate	20 m^3/day
Ingestion rate for drinking water	2 l/day
Ingestion rate for soil	100 mg/day
Ingestion rate for food	
Fish	0.037 kg/day
Meat	0.075 kg/day
Dairy Products	0.30 kg/day
Vegetables	0.05 kg/day
Fruit	0.028 kg/day

[1] A detailed discussion of the selection of these parameters and values of other dose parameters are presented in Liu (1994)

In all cases the calculated cumulative carcinogenic risks remained below a one-in-a million level, in all subregions. In the case of Site B, the risk in one subregion (receiving water supply from a small reservoir located in a high deposition area) was estimated to be 0.6 in one million.

The calculated cumulative hazard indexes for all cases remained well below the threshold value of one in all subregions.

APPENDIX L
RADIONUCLIDES

L.1 Introduction

This technical appendix addresses the assessment of radionuclide emissions from fossil fuel-fired power plants. Unlike the analyses for conventional chemical emissions, risk calculations for all U.S. power plants have not been made by EPRI. For this reason, this section focuses more on the results of calculations made for representative plants (those analyzed by EPA in its 1989 NESHAPS analysis) and on methodological issues associated with assessment of risks from radionuclide emissions and with specific aspects of EPA's CAP93-PC model, EPA's model of choice for assessing air emissions of radionuclides for the purposes of the air toxics study. This model is the successor to models used by EPA to evaluate air emissions of radionuclides from a variety of sources, including coal-fired power plants, over the past decade.

In past assessments, EPA has evaluated risks from radionuclide emissions by assessing the risks from representative plants, and has concluded that the risks from such emissions from power plants were sufficiently small that regulation was not required. In the 1989 NESHAPS, EPA evaluated eight plants: urban, suburban, rural, and remote plants in "large" and "typical" sizes. These analyses used actual plant characteristics such as capacity, heat rate, stack height, and location, including site specific consideration of meteorology and population.

The specific aspects of the analysis addressed here concern the significance of changes in EPA's analytical methods since the 1989 NESHAPS. In particular, this section addresses two aspects of the analysis, the estimate of the radionuclide source term and the indirect exposure pathway analysis.

L.2 Revisions to the EPA Radionuclide Risk Assessment Methods

For more than a decade, EPA has evaluated risks from releases of radionuclides to air through a set of analytical models, principally AIRDOS and DARTAB. As the capabilities of personal computers increased, EPA had a PC version of these models prepared that integrated the many calculations that are included in a radionuclide risk assessment. These calculations include radioactive decay and the in growth of decay products, air dispersion, deposition onto the ground, exposure to radionuclides from inhalation, ingestion

and direct shine off the ground surface, and conversions of these calculated exposures to doses and risks. The PC version of these models was published as CAP88-PC. A revised version of the model was released as CAP93-PC in 1993; this later version includes a notice that it is not to be used for regulatory compliance purposes. EPA has indicated that CAP93-PC, which differs from CAP88-PC principally in how wet deposition of radionuclides onto the ground surface is calculated, is an appropriate model for research purposes, including research within EPA on air toxic emissions.

The modification to CAP88-PC results in modest reductions in the calculated values of maximum individual risk and somewhat more significant reductions in the calculated risks to populations. Specific results for the eight plants studied in the 1989 NESHAPS are provided in the next section of this chapter.

L.3 Radionuclide Source Terms

A key aspect of the assessment of risks from radionuclide emissions from fossil-fueled power plants is the estimation of the source term. The source term is usually estimated based on measurements of radionuclides in the fuel, coal or oil, coupled with information or measurements about the plant and its operating characteristics. In the 1989 NESHAPS, emissions were estimated based on an average emission factor in the units of radionuclide emissions per unit combustion energy (e.g., pCi per 10^6 Btu). The emission factor was based on the measured radionuclide content of coal and of coal ash and on the estimated removal efficiencies of power plant particulate control systems. In addition, enrichment factors were used to account for the tendency of some radionuclides to preferentially collect in or on small particles.

Comments submitted by the industry in response to this approach in the 1989 NESHAPS noted that the use of this method could greatly overstate the emissions from well-controlled plants and underestimate emission from poorly controlled ones. An alternative approach was recommended in which radionuclide emissions are assumed to be directly proportional to particulate emissions. Revised source terms were calculated using such an approach by Ralph Roberson for the eight NESHAPS plants. This analysis produced comparatively small changes in the estimated source terms for "typical" plants, i.e., the smaller of the plants considered. For these four plants, two showed slightly higher emissions under this method, two somewhat lower emissions. The difference between the two methods was more significant for the "large" plants, due to the fact that large plants tend to be newer and better controlled than average plants.

The calculated maximum individual risks and population risks for the eight NESHAPS plants are indicated on Figures L-1 and L-2. These figures also indicate the differences in calculated risk between CAP88-PC and CAP93-PC.

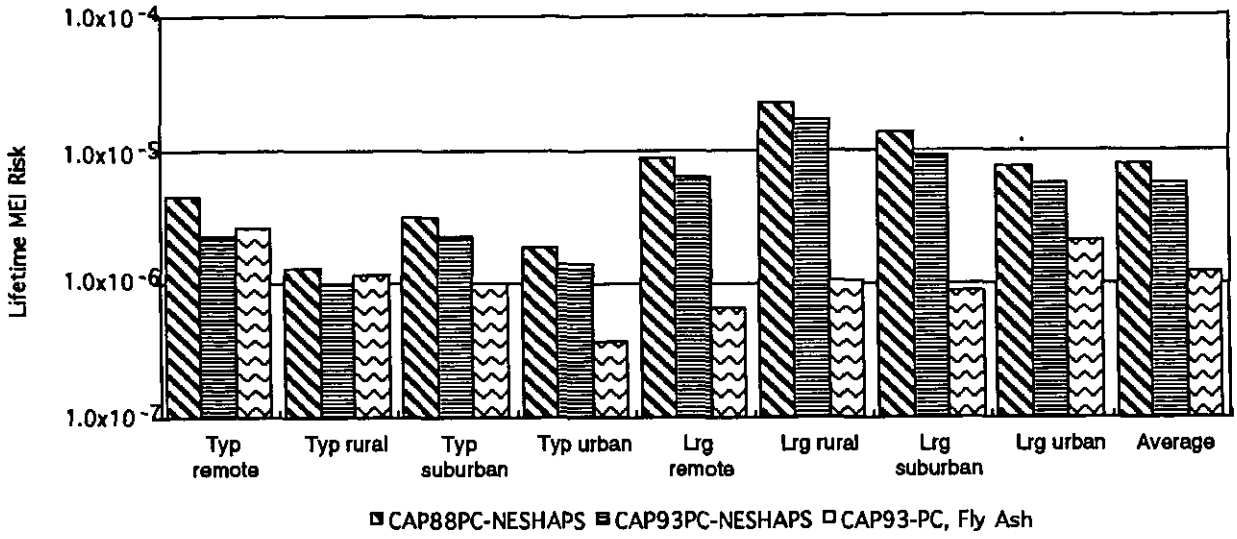

Figure L-1.
Individual Risk Based on CAP93/CAP88-PC NESHAPS vs. Fly-Ash Based Source Term

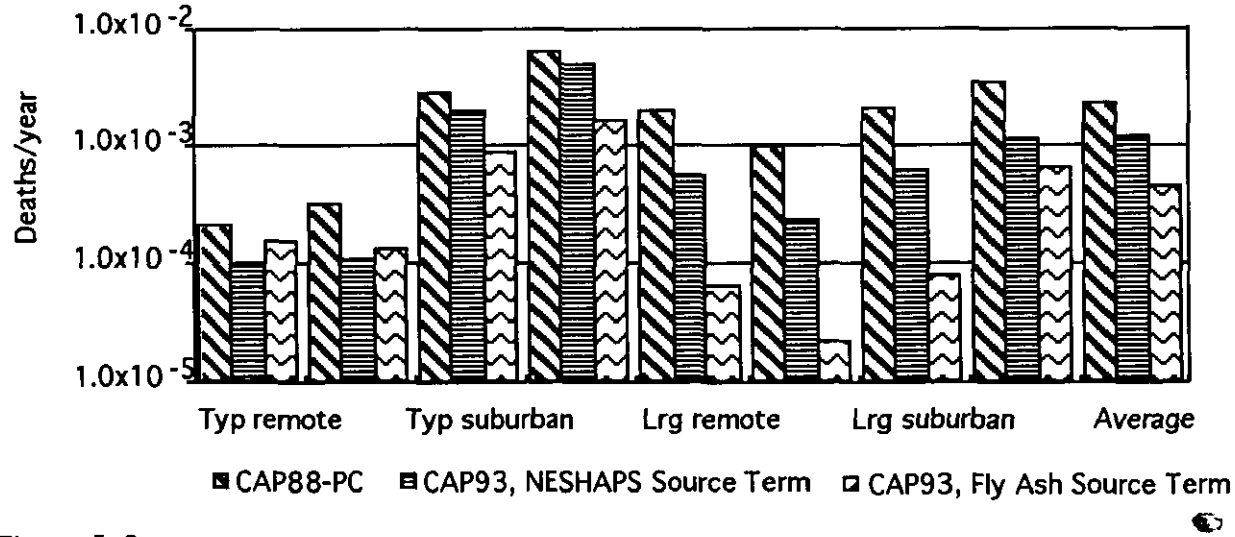

Figure L-2.
Population Risk Based on CAP88/CAP93-PC NESHAPS vs. Fly-Ash Based Source Term

The risk estimates for radionuclides indicated on these figures are based on the same estimates for particulate emissions used elsewhere in this Synthesis Report. Emission rates for Radon-220 and 222 are based on the emission factors per Btu used by EPA in the 1989 NESHAPS. The other radionuclide emission rates are calculated based on the emission factors per gram of particulate emitted indicated in Table L-1.

Table L-1.
Emission Factors for Coal-Fired Power Plant Radionuclide Risk Calculations

Nuclide	Emissions, pCi/g
U-238	5.7
U-234	5.7
Th-230	6.2
Ra-226	1.0
Pb-210	10.
Po-210	15.

Nuclide	Emissions, pCi/g
Th-232	3.8
Ra-228	5.7
Th-228	3.8
Ra-224	5.7
Pb-212	19.
Bi-212	3.8

L.4 Calculation of Radionuclide Risks Via the Food Chain

L.4.1 Introduction

Human exposure to radiation from radionuclide emissions from fossil-fueled power plants results not only from direct inhalation of radionuclides in ambient air but also indirectly from the ingestion of radionuclides present in soil particles, water and food, and from direct exposure from radionuclides on the ground surface. This chapter evaluates how EPA's CAP93-PC model assesses exposures via the food pathway. The basis for this assessment is a comparison of CAP93-PC with three other standard methods for environmental foodchain analysis. The CAP93-PC model analysis includes exposures to radionuclides from air deposition and transfer from contaminated soil on or into plants, the ingestion of cow's milk affected by the consumption of contaminated feedstocks, and the ingestion of meat from cattle feeding on contaminated feedstocks.

The comparison described here is based on a representative run of the CAP93-PC model in which it is assumed that an individual is exposed through consumption of locally produced food. It is also assumed that the feedstocks for milk and meat are also grown at the location of the individual. Calculations with all four models are based on the deposition rate flux calculated by CAP93-PC, measured in $pCi/m^2/sec$. Because all four models use an identical deposition rate, the calculations do not reveal differences that may exist between the models in how source terms or air dispersion or deposition are calculated. These calculations focus instead on assumptions that lead to differences in the calculated

concentrations of radionuclides in plants, milk, and meat and on the estimates of consumption of those foods. The detailed results are provided in the attached Tables L-2 through L-13, and in the summary table at the end of this chapter.

This comparative analysis is based on calculations with CAP93-PC and other models for emissions of Ni-59. Nickel-59 was selected as the radionuclide for this comparative analysis for several reasons:

- Ni-59 is on CAP93-PC's list of isotopes for which data are included for calculations to be run,
- Ni-59 has a sufficiently long half-life (76,000 years) that radioactive decay will not affect comparisons with chemical multipathway models in which decay is not considered.
- Nickel (and nickel compounds) are among chemicals considered in many chemical risk assessments, including in the multipathway analyses conducted by ENSR for EPRI.

The results of the comparison indicate that although an identical deposition rate was used for each of the four different mathematical models, the estimated exposures from consumption of plants, milk and meat varies from model to model. Considering the results of the combined total food intakes (vegetables, produce, meat, and milk), the CAP93-PC model gives the highest estimates for total exposure of the four models reviewed. The calculated exposures of the CAP93-PC model are higher than those of the other models by a factor of 2.3 to 6.8. The major factors in this difference are the assumptions used in CAP93-PC regarding consumption of produce and meat.

This comparison illustrates that, because the analytical assumptions used to calculate exposures from food pathways differ between these models, care should be taken to avoid reaching conclusions regarding the relative risks from radionuclide emissions versus chemical emissions. Only when the emissions are assessed in an analytically equivalent way can such conclusions regarding the relative significance of various emissions be drawn.

L.4.2 Comparison of Exposures Through Food Consumption

Starting with a fixed deposition rate, the chemical concentrations in each of the food components (vegetables, fruit, meat, and milk) were estimated using a series of mathematical algorithms. Then these concentrations were multiplied by the assumed consumption rates for each food. Mathematical models were derived from four reference sources:

1. *CAP93-PC–A Computerized Methodology for Estimating Environmental Concentrations and Dose to Man from Airborne Releases of Radionuclides;*
2. CAPCOA and Standard EPA and CAL-DTSC risk guidance;

3. EPA *Methodology for Assessing Health Risks Associated with Indirect Exposures to Combustor Emissions (IECE)*; and
4. The EPRI Total Risk and Uncertainty Evaluation Model (EPRI *TRUE*) This includes fate and transport models presented by Bonfiglio (ENSR) et. al. (1993), used in the detailed case studies in the EPRI Synthesis Report.

Each of these approaches present a similar but distinct method to derive chemical concentrations in food from deposition and in their assumptions regarding food consumption. The differences in the models are primarily dependent on their complexity. Complex models have more data requirements and generally require site-specific information. Concentrations of Ni-59 in food were estimated from the four different references in order to show the discrete differences, if any, of each of the models. The major purpose of this comparative analysis is to evaluate whether CAP93-PC uses assumptions and methods for calculating foodchain exposures that are comparable to the methods used for chemical exposure assessment.

Risk assessments typically use a term to account for emissions that contribute to a buildup of the soil concentration of a radionuclide or chemical pollutant before the risk calculation begins. This means that it is not assumed that exposures begin with "clean" soil in which crops are grown. The guidance for standard risk assessments is typically that the calculations should be based on the actual operating life of a facility, including both the time it has operated in the past and the time it is anticipated to operate in the future. The future operating lifetime is often taken to be 70 years for a generic facility. Because CAP93-PC assumes that soil concentrations are at the level that would be achieved through 100 years of prior operation, this soil concentration was also assumed for two of the other models.

Calculation of the radionuclide or chemical concentration in and on vegetation involves two separate aspects. The first is the analysis of direct deposition onto the exposed plant surface. This deposition pathway assumes that contaminants are deposited onto edible plant surfaces with a constant flux and are partially eliminated by weathering and cleansing. The second calculation is for pollutants that become mixed into surface soils and that are taken up by the roots and transferred to edible portions of the plant.

The concentration in meat and milk is estimated for cattle based on ingestion of plants (as above, both direct deposition and soil uptake into grazing crops are considered) and ingestion of soil while grazing. Concentrations in meat and milk are derived from the concentration of the pollutant in plants and soil, the quantity of plants and soil that animals consume, and the biotransfer factor of each type of animal tissue.

L.4.2.1 CAP93-PC.

Table L-2 presents the equations used by CAP93-PC to estimate the concentration in and on vegetables from deposition and uptake in picocuries per kilogram (pCi/kg). CAP93-PC uses the same parameters for vegetables and produce. In addition to a removal rate for physical losses by weathering, a washing factor of 0.5 is used to account for the

removal of radionuclides that are adhered to plant surfaces. The period for long term build up in soil is assumed to be 100 years. The chemical concentrations in vegetables utilizing the CAP93-PC method are presented in Table L-2.

Table L-2.
Estimating Intakes of Deposited Nickel by Ingestion of Vegetables —CAP93-PC Radionuclide Methodology

Cv/Cp = di [{R(1-exp(-Ei * te)) / (Yv * Ei)} * DDl + {Bv(1-exp(-^i * tb)) / (P * ^i)}] exp(-^i * th)		
deposition = di [{R(1-exp(-Ei * te)) / (Yv * Ei)} * DDl] exp(-^i * th)		
uptake = {Bv(1-exp(-^i * tb)) / (P * ^i)} exp(-^i * th)		
Parameters	**Value**	**Units**
Cv/Cp = concentration of nuclide in and on vegetable/produce	calculated	pCi/kg
di = ground deposition rate of radionuclide i	$2.52X10^{-05}$	pCi/m^2-hr
R = fraction retained on edible portions of crops	$2.00X10^{-01}$	unitless
^i = radioactive decay constant of nuclide	0	hr^{-1}
Ei = removal rate constant from crops by weathering	$2.90X10^{-03}$	hr^{-1}
te = time crops are exposed to nuclide	$1.44X10^{+03}$	hr
Yv = agricultural productivity yield	$7.16X10^{-01}$	kg/m^2
Bv = conc. uptake factor of nuclide from soil by edible parts of crops	$2.57X10^{-02}$	pCi/kg/pCi/kg
tb = long term build up in soil	$8.76X10^{+05}$	hr
P = effective density of top 15cm soil	$2.15X10^{+02}$	kg/m^2
th = holdup time between harvest and consumption of crops	$3.36X10^{+02}$	hr
DDl = fraction of radioactivity retained after washing	$5.00X10^{-01}$	unitless
Results:		
Uv = ingestion rate of produce	$1.76X10^{+02}$	kg/yr
Ul = ingestion of leafy vegetable	$1.80X10^{+01}$	kg/yr
fg = fraction of produce ingested grown in garden	1.00	unitless
fl = fraction of leafy vegetables grown in garden	1.00	unitless
assumed 100% of plant intake originated from contaminated soil		

The concentrations in meat and milk are dependent on the amount and contamination level of feed, pasture and soil consumed by cattle. Tables L-3 and L-4 present the equations used to estimate the radionuclide concentration in animal feed and in pasture grass which in turn are used to derive the concentration in animal tissues. Similar to the

approach used to estimate vegetable concentrations, the parameters used to estimate meat and milk concentrations were obtained from the parameter outputs generated from CAP93-PC. It was assumed that beef cattle are on open pasture for the same grazing periods as dairy cattle. Tables L-3 and L-4 present the estimated chemical concentrations in beef and milk.

Table L-3.
Estimating Intakes of Deposited Nickel by Ingestion of Milk —CAP93-PC Radionuclide Methodology

Cv = (fp*fs*Cp) + (1-(fp*fs)) Cs		
Cm = (Fm*Cv*Qf) * exp(-^i * tf)		
Parameters	**Value**	**Units**
Cv = concentration of nuclide in animal's feed	calculated	pCi/kg
Cm = concentration of nuclide in milk	calculated	pCi/liter
Cp = concentration of nuclide in pasture grass	$2.39X10^{-03}$	pCi/kg
Cs = concentration of nuclide in stored feed	$2.39X10^{-03}$	pCi/kg
fp = fraction of year animals graze on pasture	$4.00X10^{-01}$	unitless
fs = fraction of daily feed that is pasture grass when animals graze	$4.30X10^{-01}$	unitless
Fm = avg fraction of animal's daily intake of nuclide in milk	$1.00X10^{-03}$	days/liter
Qf = amout of feed animal consumes per day	$1.56X10^{+01}$	kg/day
tf = avg transport time of activity from feed into milk & to receptor	2.00	day
^i = radioactive decay constant of nuclide	0	day^{-1}
Dairy productivity = milk production of cow	$1.10X10^{+01}$	liters/day
Results:		
Um = ingestion rate of milk	$1.12X10^{+02}$	liters/yr
assumed 100% of intake originated from contaminated soil		

Table L-4.
Estimating Intakes of Deposited Nickel by Ingestion of Beef
—CAP93-PC Radionuclide Methodology

Cf = (Ff x Cv x Qf) exp(-^i x ts)		
Parameters	**Values**	**Units**
Cf = nuclide concentration in meat	calculated	pCi/kg
Ff = fraction of animal's daily intake of nuclide in each kg/flesh	$6.00X10^{-03}$	days/kg
Cv = Concentration of nuclide in animal's feed	$2.39X10^{-03}$	pCi/kg
Qf = amount of feed consumed by animal per day	$1.56X10^{+01}$	kg/day
^i = radioactive decay constant of nuclide	0	day^{-1}
ts = average time from slaughter to consumption	$2.00X10^{+01}$	days
Results:		
Uf = Ingestion rate of meat	$8.50X10^{+01}$	kg/yr
assumed 100% of intake originated from contaminated soil		

L.4.2.2 CAPCOA/EPA Risk Methodology

This set of models refers to EPA's Exposure factors Handbook, with additional elaboration provided by the California Air Pollution Control Officer's Association (CAPCOA) and other California EPA guidance. The deposition onto the plant surface is a function of the type of plant and its surface area. Per CAPCOA guidance, vegetables are divided into leafy vegetables, exposed produce, and protected produce (root vegetables). Direct deposition of particles onto edibles surfaces are therefore only applicable for leafy vegetables and exposed produce.

The deposition rate output from CAP93-PC was utilized to estimate the chemical concentration in vegetables and produce. A conversion factor (1 Ci Ni-59 = 12.35 g) was used to convert the radionuclide deposition rate to a chemical flux rate in the units of $mg/m^2/$ year. The interception fraction is the fraction of the deposited particles that fall onto the plant surfaces. An interception fraction of 0.19 and 0.078 were used for leafy vegetables and exposed produce, respectively, and were derived from Baes et al. (1984). The weathering rate constant describes the loss of particles from the plant surface and is based on a half-life of 14 days, as recommended by CAPCOA. An average growth/exposure period of 58 and 45 days were used for leafy vegetables and exposed produce, respectively.

The potential concentrations from root uptake were derived by the product of the estimated soil concentration and the plant uptake factor. Soil concentrations were estimated from the deposition rate, the soil buildup time, the mixing depth and soil bulk density. The soil buildup time was assumed to be 100 years; the mixing depth was estimated at 15 centimeters; and the bulk density was assumed to 1800 kg/m^3 for silty sandy soils. An uptake factor of 0.0213 was derived from Baes et al. (1984). Table L-5 presents the chemical concentrations in vegetables and produce from deposition and uptake.

Table L-5.
Estimating Intakes of Deposited Nickel by Ingestion of Vegetables —Standard Risk Methodology (U.S. EPA & CAPCOA Risk Guidance)

Cv or Cp = [{(D • R • CF) / (k • Y)} • (1-exp(-k*T)] + (Cs * Bv)
where: Cs = (D • tb) / (SD • BD)
deposition = [{(D • R • CF) / (k • Y)} • (1-exp(-k*T)]
root uptake = (Cs • Bv)

Parameters	Value	Units
Cv/Cp = concentration of chemical in and on vegetable/produce	calculated	mg/kg
D = ground deposition rate	$2.73X10^{-09}$	mg/m^2-yr
Rv = interception fraction (leafy vegetables) •	$1.91X10^{-01}$	unitless
Rp = interception fraction (exposed produce) •	$7.78X10^{-02}$	unitless
k = removal rate constant from crops,by weathering	0.0495	day^{-1}
Tv = time crops are exposed (leafy)	58	days
Tp = time crops are exposed (produce)	45	days
Y = agricultural productivity yield	2.50	kg/m^2
Bv = uptake factor from soil by edible parts of crops **	$2.13X10^{-02}$	unitless
tb = long term build up in soil (accumulation time)	100	years
CF = conversion factor	$2.74X10^{-03}$	yr/365 days
Cs = concentration in offsite soils from deposition	$1.01X10^{-09}$	mg/kg
SD = soil depth of mixing	$1.50X10^{-01}$	meters
BD = bulk density ***	$1.80X10^{+03}$	kg/m^3
IRv = Vegetable Ingestion Rate	0.08	kg/day
IRp = Produce Ingestion Rate	0.042	kg/day

Table L-5.
Estimating Intakes of Deposited Nickel by Ingestion of Vegetables
—Standard Risk Methodology (U.S. EPA & CAPCOA Risk Guidance) (Continued)

* interception fractions derived from Baes et. (1984) & EPA 1990
** standard average uptake factors for nickel on leafy, vine & root vegetables, Baes et al., 1984
*** assumed bulk density, range for silty sandy soils
**** assumed 100% of plant intake originated from contaminated soil
References:
Baes et al. 1984 Review and analysis of parameters for assessing transport of released radionuclides....
EPA 1989, U.S. EPA Exposure Factor Handbook, May 1989
EPA 1991, U.S. EPA Supplemental Standard Default Exposure Parameters, April 1991
Note: Recent risk guidance stipulates the use of "aboveground" and "belowground" vegetables. In addition, according to CAL-EPA, only chemicals with a log Kow greater than 3.0 should be evalauted because they are considered more likely to transfer pollutants from the root soils to the exposed (above ground) and protected (below-ground) layers of vegetation. Because nickel does not have a Kow and is used as our example, the old method was used to estimate the concentration of nickel in vegetables.

The estimation of chemical concentrations in meat and milk involves two steps. The concentration of nickel in feed, pasture and soil are first estimated, and then the transfer of the ingested chemicals to the tissues of the cow are considered. Tables L-6 and L-7 present the models used to estimate the chemical concentrations in feed and pasture for beef and dairy cattle, respectively. The concentration in soil was estimated using the same equation described above. According to CAPCOA guidance, the feed ingestion rates differ among beef (8 kg/day) and dairy cattle (16 kg/day). The chemical transfer coefficients from the diet to beef cattle and dairy cattle are 0.002 and 0.001, respectively and were obtained as recommended values from CAPCOA.

Table L-6.
Estimation of Nickel in Beef
—Standard Risk Methodology (CAPCOA Risk Guidance)

eq.	Model		Recommended Default Value	Reference
[1]	Cfa = (Feed ingestion + Pasture/Grazing ingestion + Soil ingestion) x Fi		$4.33X10^{13}$	
	Cfa	= Concentration in beef (mg/kg)	eq. [1]	
	Feed ingestion	= Dose through feed ingestion (µg/d)	eq. [2]	
	Pasture/Grazing ingestion	= Dose through pasture/grazing ingestion (µg/d)	eq. [3]	
	Soil ingestion	= Dose through soil ingestion (µg/kg)	eq. [4]	
	Fi	= Chemical transfer coefficient from diet to beef (d/kg)	$2.00X10^{03}$	CAPCOA '91 for Ni
[2]	Feed ingestion = (1 - %G) x FI x L x Cf		$1.08X10^{07}$	

Table L-6.
Estimation of Nickel in Beef
—Standard Risk Methodology (CAPCOA Risk Guidance) (Continued)

eq.	Model		Recommended Default Value	Reference
	%G	= % Diet provided by grazing	1	site-specific
	FI	= Feed ingestion rate (kg/d)	8.00	CAPCOA '91
	L	= % of locally grown feed that is not pasture	1	site-specific
	Cf	= Concentration in feed (µg/kg)	eq. [5]	
[3]	Pasture/Grazing ingestion = %G x Cf x FI		$1.08X10^{-07}$	
	Cf	= Concentration in pasture/grazing material (µg/kg)	eq. [5]	
[4]	Soil ingestion = SI x Cs		$4.04X10^{-10}$	
	SI	= Soil ingestion rate for cattle (kg/d)	eq. [6]	
	Cs	= Soil concentration (µg/kg)	TABLE 1	
[5]	Cf = Cdepv x BIO + Ctrans		$1.35X10^{-08}$	
	Cdepv	= Concentration due to direct deposition (µg/kg)	eq. [7]	
	BIO	= Bioavailability	1.00	assumed 100%
	Ctrans	= Concentration due to root translocation or uptake (µg/kg)	eq. [8]	
[6]	SI = [(1 - %G) x %Sf x FI] + %G x %Sp x FI		$4.00X10^{-01}$	
	%Sf	= Soil ingested as a % of feed ingested	$1.00X10^{-02}$	CAPCOA '91
	%Sp	= Soil ingested as a % of pasture ingested	$5.00X10^{-02}$	CAPCOA '91
[7]	Cdepv = [Dep x IF / (k x Y)] x (1 - exp[-kT])		$1.35X10^{-08}$	
	Dep	= Deposition on affected vegetation per day (µg/m2_/d)	$7.47X10^{-09}$	site-specific for Ni

Table L-6.
Estimation of Nickel in Beef
—Standard Risk Methodology (CAPCOA Risk Guidance) (Continued)

eq.	Model		Recommended Default Value	Reference
	IF	= Interception fraction	$2.00X10^{-01}$	crop-specific (leafy)
	k	= Weathering constant (1/d)	$4.95X10^{-02}$	CAPCOA '91
	Y	= Yield (kg/m2_)	2.00	CAPCOA '91
	T	= Growth period (d)	$4.50X10^{+01}$	CAPCOA '91 (45-90)
[8]	Ctrans = Cs x UF2		$5.05X10^{-11}$	
	UF2	= Root uptake factor	$5.00X10^{-02}$	CAPCOA '91
	IRb	= Ingestion Rate of meat (kg/day)	0.075	CAPCOA '91
	DD	= Intake rate of beef (mg/day)	$3.25X10^{-14}$	

Table L-7.
Estimation of Nickel in Milk—Standard Risk Methodology (CAPCOA Risk Guidance)

eq.	Model		Recommended Default Value	Reference
[1]	Cm = (Feed ingestion + Pasture/Grazing ingestion + Soil ingestion) x Fi		$4.33X10^{-13}$	
	Cm	= Concentration in milk (mg/kg)	eq. [1]	
	Feed ingestion	= Dose through feed ingestion (µg/d)	eq. [2]	
	Pasture/Grazing ingestion	= Dose through pasture/grazing ingestion (µg/d)	eq. [3]	
	Soil ingestion	= Dose through soil ingestion (µg/kg)	eq. [4]	
	Fi	= Chemical transfer coefficient from diet to beef (d/kg)	$1.00X10^{-03}$	CAPCOA '91 for Ni
[2]	Feed ingestion = (1 - %G) x FI x L x Cf		$2.16X10^{-07}$	

Table L-7.
Estimation of Nickel in Milk—Standard Risk Methodology (CAPCOA Risk Guidance) (Continued)

eq.	Model		Recommended Default Value	Reference
	%G	= % Diet provided by grazing	1	site-specific
	FI	= Feed ingestion rate (kg/d)	16.0	CAPCOA '91
	L	= % of locally grown feed that is not pasture	1	site-specific
	Cf	= Concentration in feed (µg/kg)	eq. [5]	
[3]	Pasture/Grazing ingestion = %G x Cf x FI		$2.16X10^{-07}$	
	Cf	= Concentration in pasture/grazing material (µg/kg)	eq. [5]	
[4]	Soil ingestion = SI x Cs		$8.08X10^{-10}$	
	SI	= Soil ingestion rate for cattle (kg/d)	eq. [6]	
	Cs	= Soil concentration (µg/kg)	TABLE 1	
[5]	Cf = Cdepv x BIO + Ctrans		$1.35X10^{-08}$	
	Cdepv	= Concentration due to direct deposition (µg/kg)	eq. [7]	
	BIO	= Bioavailability	1.00	assumed 100%
	Ctrans	= Concentration due to root translocation/uptake (µg/kg)	eq. [8]	
[6]	SI = [(1 - %G) x %Sf x FI] + %G x %Sp x FI		$8.00X10^{-01}$	
	%Sf	= Soil ingested as a % of feed ingested	$1.00X10^{-02}$	CAPCOA '91
	%Sp	= Soil ingested as a % of pasture ingested	$5.00X10^{-02}$	CAPCOA '91
[7]	Cdepv = [Dep x IF / (k x Y)] x (1 - exp[-kT])		$1.35X10^{-08}$	
	Dep	= Deposition on affected vegetation per day (µg/m2_/d)	$7.47X10^{-09}$	site-specific for Ni
	IF	= Interception fraction	$2.00X10^{-01}$	crop-specific (leafy)

Table L-7.
Estimation of Nickel in Milk—Standard Risk Methodology (CAPCOA Risk Guidance) (Continued)

eq.	Model		Recommended Default Value	Reference
	k	= Weathering constant (1/d)	$4.95X10^{-02}$	CAPCOA '91
	Y	= Yield (kg/m_u2_)	2.00	CAPCOA '91
	T	= Growth period (d)	0.45	CAPCOA '91 (45-90)
[8]	Ctrans = Cs x UF2		$5.05X10^{-11}$	
	UF2	= Root uptake factor	$5.00X10^{-02}$	CAPCOA '91
	IRm	= Ingestion Rate of milk (kg/day - L/day)	0.3	CAPCOA '91
	DD	= Intake rate of milk (mg/day)	$1.30X10^{-13}$	
Note: difference between beef cattle and dairy cattle is the chemical transfer coefficient and feed ingestion rate				

L.4.2.3 Methodology for Assessing Health Risks Associated with Indirect Exposures to Combustor Emissions (IECE)

According to the Methodology for Assessing Health Risks Associated with Indirect Exposures to Combustor Emissions (IECE), chemical concentrations in vegetation are estimated from the sum of the total pollutant contribution from deposition, root uptake and air-to-plant transfer. For the example compound used in this analysis, nickel, chemical concentrations from air-to-plant transfers were not applicable.

Interception fractions of 0.0266 and 0.0103 were recommended for leafy and exposed produce, respectively. An average crop yield of 0.32 kg/m^2 for all plant species, a plant surface loss coefficient of 18.07/yr, and an estimated soil loss constant of 0.003/yr were used as inputs. An uptake factor of 0.0213, identical to the one used in the CAPCOA method, was utilized.

IECE provides bioconcentration factors (uptake factors) for different plant species and for different chemicals. In lieu of plant-specific uptake factors for nickel, chemical concentrations for different plant species from root uptake were estimated using bioconcentration factors for cadmium (see Table L-8). The resulting chemical concentrations in grain, forage and silage were used along with the soil concentration to estimate the concentration of nickel in animal tissues. A plant and soil ingestion rates of 11.8 kg/day and 0.6 kg/day were provided for beef cattle. Conversely, a plant and soil ingestion rates of 16.9 kg/day

and 0.4 kg/day were provided for dairy cattle. The biotransfer factor for beef and dairy cattle are 0.003 days/kg and 0.0055 days/kg, respectively. Table L-9 provides the chemical concentrations for specific plant species.

Table L-8.
Estimating Intakes of Deposited Nickel by Ingestion of Vegetables

Cv/Cp = 1000 • [Dyd + (Fw*Dyw)] • Rp • {1-exp(-kp*Tp)] / Yp • kp + Cs • Br			
where: Cs = (Dyd + Dyw) • [1-exp(-ks • Tc)] • 100 / Z • BD • ks			
deposition (Pd) = 1000 • [Dyd + (Fw*Dyw)] * Rp • {1-exp(-kp*Tp)] / Yp • kp			
root uptake (Pr) = Cs • Br			
Parameters	**Value**	**Units**	**Comments**
Cv = conc. of pollutant due to direct deposition (leafy)	calculated	mg/kg	
Cp = conc. of pollutant due to direct deposition (produce)	calculated	mg/kg	
1000 = conversion factor	1000	kg/g/µg/g	
Dyd = yearly dry deposition rate	$2.57X10^{-13}$	g/m^2/year	
Fw = fraction of wet deposition that adheres to plant surfaces	0.1	unitless	
Dyw = yearly wet deposition rate	$2.49X10^{-12}$	g/m^2/year	
Rp = interception fraction of leafy vegetables	$2.66X10^{-02}$	unitless	
Rp = interception fraction of exposed produce	$1.03X10^{-02}$	unitless	
kp = plant surface loss coefficient	18.07	yr^{-1}	
Tp = length of exposure to deposition (leafy)	$1.64X10^{-01}$	years	
Tp = length of exposure to deposition (produce)	$1.64X10^{-01}$	years	
Yp = crop yield	0.32	kg/m^2	avg all groups
Cs = soil concentration after deposition	$8.80X10^{-10}$	mg/kg	
ks = soil loss constant	$3.00X10^{-03}$	yr^{-1}	estimated
Tc = total time in which deposition occurs	100	years	same as CAP93
0.1 = units conversion factor	0.1	unitless	
Z = soil depth	15	cm	
BD = bulk density	1.80	g/cm^3	
Br = Uptake (Bioconcentration) Factor for Nickel (assumed same as BAES)	$2.13X10^{-02}$	unitless	for all plants

Table L-8.
Estimating Intakes of Deposited Nickel by Ingestion of Vegetables (Continued)

IRv = Ingestion Rate of leafy vegetables	0.0084	kg/day	95th percentile
IRp = Ingestion Rate of exposed produce	0.0238	kg/day	95th percentile
IRp = Ingestion Rate of protected produce	0.3164	kg/day	95th percentile

* Exposed produce signifies exposure from both deposition and root uptake. Protected produce from root uptake only.
** assumed 100% of plant intake originated from contaminated soil
leafy produce = leafy vegetables & brassica
protected produce = grains, potatoes, root vegetables, & melons
exposed produce = legumes and other fruiting vegetables
Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (IECE), EPA, 1990, 1993

Table L-9.
Estimating Nickel Concentrations in Different Plants

Root Uptake (Pr):Pr = Cs • Br (assuming Brs for cadmium are the same for nickel; see reference examples)
Deposition (Pd):Pd = 1000 * [Dyd + (Fw*Dyw)] • Rp * (1-exp(-kp*Tp)] / Yp * kp
bioconcentration factors (Brs) = plant uptake (UFs)

Plant species	Br (unitless)	Rp (unitless)	Yp (kg/m≤)	Tp (years)
grains:	0.05	0	0.22	0.164
legumes:	0.24	0.010	0.43	0.164
potatoes:	0.09	0	0.43	0.164
root vegetables:	1.98	0	0.43	0.164
fruits & fruit vegetables:	1.16	0.010	0.26	0.164
leafy vegetables:	1.18	0.027	0.14	0.164
forage:	0.39	0.47	0.31	0.123
Average:	0.78	0.01	0.32	

Concentration of nickel in different plants:

Plant species	Pr (mg/kg)	Pd (mg/kg)	Total (mg/kg)	
grains:	$4.40X10^{-11}$	protected	$4.40X10^{-11}$	
legumes:	$2.11X10^{-10}$	$6.34X10^{-13}$	$2.12X10^{-10}$	
potatoes:	$7.92X10^{-11}$	protected	$7.92X10^{-11}$	
root vegetables:	$1.74X10^{-09}$	protected	$1.74X10^{-09}$	

Table L-9.
Estimating Nickel Concentrations in Different Plants (Continued)

Root Uptake (Pr):Pr = Cs • Br (assuming Brs for cadmium are the same for nickel; see reference examples)
Deposition (Pd):Pd = 1000 * [Dyd + (Fw*Dyw)] • Rp * (1-exp(-kp*Tp)] / Yp * kp
bioconcentration factors (Brs) = plant uptake (UFs)

Plant species	Br (unitless)	Rp (unitless)	Yp (kg/m²)	Tp (years)
fruits & fruit vegetables:	$1.02X10^{-09}$	$1.05X10^{-12}$	$1.02X10^{-09}$	
leafy vegetables:	$1.04X10^{-09}$	$5.04X10^{-12}$	$1.04X10^{-09}$	
forage:	$3.43X10^{-10}$	$3.79X10^{-11}$	$3.81X10^{-10}$	
TOTAL minus forage	$4.14X10^{-09}$	$6.73X10^{-12}$	$4.14X10^{-09}$	
TOTAL leafy			$1.04X10^{-09}$	
TOTAL protected produce			$2.08X10^{-09}$	
TOTAL exposed produce			$1.02X10^{-09}$	
Note: the bioconcentration factors used in this table are those available for cadmium.				

The chemical concentration in animal tissues is calculated by adding the concentration in animal tissue due to plant consumption with that due to soil ingestion by cattle, with consideration of the appropriate biotransfer coefficients. The results of the chemical concentrations in animal tissues are presented in Table L-10.

Table L-10.
Estimating Chemical Concentrations in Animal Tissues from: "Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (IECE)," EPA, 1990, 1993

Parameters	Value	Units	Comments
Aj = concentration of pollutant in cattle tissue group	calculated	mg/kg	mg pollutant/g animal tissue
Fij = fraction of the ith plant and eaten by cattle	1	unitless	100% contaminated, no purchased feed
Qpij = quantity of ith plant group eaten by beef cattle each day	11.77	kg/day	overall pasture/feed ingestion rate
Qpij = quantity of ith plant group eaten by dairy cattle each day	16.9	kg/day	overall pasture/feed ingestion rate
Pij = total concentration of pollutant in plants eaten by cattle	$8.06X10^{-10}$	mg/kg	grain, forage & silage
Qsj = quantity of soil eaten by beef cattle each day	0.6	kg/day	soil ingestion rate
Qsj = quantity of soil eaten by dairy cattle each day	0.4	kg/day	soil ingestion rate

Table L-10.
Estimating Chemical Concentrations in Animal Tissues from: "Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (IECE)," EPA, 1990, 1993 (Continued)

Parameters				Value	Units	Comments
Cs = soil concentration from deposition				$8.80X10^{-10}$	mg/kg	mg pollutant/g soil
BAj = biotransfer factor for beef cattle tissue group				0.003	d/kg	Belcher & Travis, 1989 in IECE
BAj = biotransfer factor for dairy cattle tissue group				0.0055	d/kg	Belcher & Travis, 1989 in IECE
Estimating nickel concentrations in animal tissues						
	beef cattle		dairy cattle			
	Qp/Qs	Conc	Qp/Qs	Conc		
Plant	(kg/day)	(mg/kg)	(kg/day)	(mg/kg)		
grain	0.47	$4.40X10^{-11}$	2.6	$4.40X10^{-11}$		
forage	8.8	$3.81X10^{-10}$	11	$3.81X10^{-10}$		
silage	2.5	$3.81X10^{-10}$	3.3	$3.81X10^{-10}$		
soil	0.6	$8.80X10^{-10}$	0.4	$8.80X10^{-10}$		
tissue conc		$3.00X10^{-11}$		$7.69X10^{-11}$		
assumed 100% of intake originated from contaminated soil						

L.4.2.4 EPRI TRUE Model

The following section refers to the multipathway method used in the EPRI case studies. The concentration of nickel in plants from deposition is based on the deposition rate of the chemical and three non-chemical-specific factors: interception fraction, crop density, and weathering half life. A weighted interception fraction of 0.045 for all fruits and vegetables in all plant groups was recommended. A crop density of 2.0 kg/m^2 and an elimination weathering rate that produces a half life of 14 days were used as input parameters. The concentration of nickel in plants from root uptake is based on the soil-to-plant bioconcentration factor (uptake factor) and the soil concentration. A bioconcentration factor of 0.06 was utilized (Baes et al. 1984).

Chemical concentrations in beef and milk were estimated using the same equation provided by IECE. The equation calculates the dose to the animal resulting from the ingestion of vegetation and soil and the result is multiplied by the bioconcentration factor in the animal tissue. However, the inputs used in the EPRI *TRUE* method differ slightly with the inputs from IECE. The crop ingestion rates employed are 10.1 kg/day and 11.1 kg/day

for beef and dairy cattle, respectively. The soil ingestion rates are 1.8 kg/day for beef cattle and 0.59 kg/day for dairy cattle. The bioconcentration factor for metals in beef liver is 0.004 (Rundle et al.). According to the Bonfiglio document, metal concentrations in milk are not appropriate because studies have shown that metals do not translocate in milk. However, for this analysis, a biotransfer coefficient of 0.0055 for metals in milk was assumed, as referenced in IECE guidance. The resulting chemical concentrations in beef and milk are presented in Table L-11. Table L-11 also presents the chemical concentrations from deposition and root uptake on the methods developed for EPRI *TRUE*.

Table L-11.
Estimating Chemical Concentrations in Vegetables/Produce, Meat, and Milk

PLANT PATHWAY		
Deposition of chemical on plant surface:	Cp = (DR x IF) / (Kel x CD)	
Parameter	**Value**	**Units**
Cp = Concentration in plant from deposition	$1.20X10^{-14}$	mg/kg
DR = deposition rate	$7.48X10^{-12}$	mg/m^3-day
IF = interception fraction	0.045	unitless
Kel = weathering elimination rate	14	day^{-1}
CD = crop density	2	kg/m^2
Root Uptake:	Cp = BCF x Cs	
Parameter	**Value**	**Units**
Cp = Concentration in plant from root uptake	$6.06X10^{-11}$	mg/kg
BCF = soil to plant bioconcentration factors	0.06	untiless
Cs = concentration in soil	$1.01X10^{-09}$	mg/kg
BEEF PATHWAY (applicable for crop and soil ingestion only, does not include air or water).		
Concentration in meat	Cb = DD x BCF	
Dose from crop ingestion	DDic = Cf x CR	
Dose from soil ingestion	DDis = Cs x SR	
Parameter	**Value**	**Units**
Cb = concentration in beef	$9.79X10^{-12}$	mg/kg
DDic = daily chemical dose from crop ingestion	$6.12X10^{-10}$	mg/day

Table L-11.
Estimating Chemical Concentrations in Vegetables/Produce, Meat, and Milk (Continued)

PLANT PATHWAY		
DDis = daily chemical dose from soil ingestion	$1.84X10^{-09}$	mg/day
Cf = chemical concentration in feed	$6.06X10^{-11}$	mg/kg
Cs = concentration in soil	$1.01X10^{-09}$	mg/kg
CR = crop consumption rate	10.1	kg/day
SR = soil ingestion rate	1.8	kg/day
DD = total dose from crop and soil ingestion	$2.45X10^{-09}$	mg/day
BCF = bioconcentration factors for metals in beef liver	0.004	unitless
MILK PATHWAY (applicable from crop and soil ingestion only, does not include air or water).		
Concentration in milk	Cm = DD x BTF	
Dose from crop ingestion	DDic = Cf x CR	
Dose from soil ingestion	DDis = Cs x SR	
Parameter	**Value**	**Units**
Cm = concentration in milk	$6.97X10^{-12}$	mg/kg
DDic = daily chemical dose from crop ingestion	$6.75X10^{-10}$	mg/day
DDis = daily chemical dose from soil ingestion	$5.92X10^{-10}$	mg/day
Cf = chemical concentration in feed	$6.06X10^{-11}$	mg/kg
Cs = concentration in soil	$1.01X10^{-09}$	mg/kg
CR = crop consumption rate	11.13	kg/day
SR = soil ingestion rate	0.59	kg/day
DD = total dose from crop and soil ingestion	$1.27X10^{-09}$	mg/day
BTF = biotransfer factors for metals in milk	0.0055	unitless
Concentration of chemical in food chain:		
Concentration of chemical in vegetables	$6.06X10^{-11}$	mg/kg
Concentration of chemical in produce	$6.06X10^{-11}$	mg/kg
Concentration of chemical in meat	$9.79X10^{-12}$	mg/kg
Concentration of chemical in milk	$6.97X10^{-12}$	mg/kg

Table L-11.
Estimating Chemical Concentrations in Vegetables/Produce, Meat, and Milk (Continued)

PLANT PATHWAY		
Ingestion rates for food: (1)		
Ingestion rate of vegetables	0.05	kg/day
Ingestion rate of produce	0.028	kg/day
Ingestion rate of meat	0.075	kg/day
Ingestion rate of dairy products	0.3	kg/day
Intake rates for food:		
Vegetables	$3.03X10^{-12}$	mg/day
Produce	$1.70X10^{-12}$	mg/day
Meat	$7.35X10^{-13}$	mg/day
Milk	$2.09X10^{-12}$	mg/day
total veggies	$4.73X10^{-12}$	mg/day
total beef and milk	$2.83X10^{-12}$	mg/day

(1) selection of ingestion rate parameters are presented in Liu via ENSR, 1993

Note: According to the document, "because many investigators have shown that inorganic metals do not translocate to into milk, metal concentrations are generally not assessed. For this analysis, it is assumed that metals translocate."

Table L-12.
Comparison of Parameters Used to Estimate Intakes of Deposited Nickel by Ingestion of Vegetables Using Separate Risk Methodologies

Methodology	CAP93PC	CAPCOA	IECE	EPRI *TRUE*	
Parameter	Value	Value	Value	Value	Units
Concentration of chemical in/on vegetable/ produce	calculated	calculated	calculated	calculated	mg/kg
Ground deposition rate	$2.73X10^{-09}$	$2.73X10^{-09}$	$2.73X10^{-09}$	$2.73X10^{-09}$	mg/m^3-yr
Interception fraction (leafy vegetables)	$2.00X10^{-01}$	$1.91X10^{-01}$	$2.66X10^{-02}$	$4.50X10^{-02}$	unitless
Interception fraction (exposed produce)	$2.00X10^{-01}$	$7.78X10^{-02}$	$1.03X10^{-02}$	$4.50X10^{-02}$	unitless
Radioactive decay constant of nuclide	0	NA	NA	NA	hr^{1}

Table L-12.
Comparison of Parameters Used to Estimate Intakes of Deposited Nickel by Ingestion of Vegetables Using Separate Risk Methodologies (Continued)

Methodology	CAP93PC	CAPCOA	IECE	EPRI *TRUE*	
Parameter	Value	Value	Value	Value	Units
Removal rate constant from crops by weathering	$6.96X10^{-02}$	$4.95X10^{-02}$	$4.95X10^{-02}$	$4.95X10^{-02}$	day^{-1}
Time crops are exposed (leafy)	60	58	60	NA	days
Time crops are exposed (produce)	60	45	60	NA	days
Agricultural productivity yield	$7.16X10^{-01}$	2.50	$3.18X10^{-01}$	2.00	kg/m^2
Uptake factor from soil by edible parts of crops	$2.57X10^{-02}$	$2.13X10^{-02}$	$2.13X10^{-02}$	$6.00X10^{-02}$	unitless
Long term build up in soil (accumulation time)	100	100	100	NA	years
Effective density of top 15cm soil	$2.15X10^{+02}$	NA	NA	NA	kg/m^2
Holdup time between harvest and consumption	$3.36X10^{+02}$	NA	NA	NA	hr
Fraction of chemical retained after washing	$5.00X10^{-01}$	NA	NA	NA	untiless
Soil loss constant	NA	NA	0.003	NA	yr^{-1}
Conversion factor (1)	NA	$2.74X10^{-03}$	NA	NA	yr/365 days
Conversion factor (2)	NA	NA	1000	NA	kg/g/ µg/g
Concentration in soils from deposition	NA	$1.01X10^{-09}$	$8.80X10^{-10}$	$1.01X10^{-09}$	mg/kg
Soil depth of mixing	NA	0.15	0.15	NA	meters
Bulk density of soils	NA	1800	1800	NA	kg/m^3
Vegetable Ingestion Rate	0.05	0.08	0.0084	0.05	kg/day
Produce Ingestion Rate	0.48	0.042	0.3402	0.028	kg/day
Concentration in/on leafy vegetables	$4.73X10^{-11}$	$3.24X10^{-11}$	$2.38X10^{-11}$	$6.06X10^{-11}$	mg/kg
Concentration in/on produce	$4.73X10^{-11}$	$2.57X10^{-11}$	$2.04X10^{-11}$	$6.06X10^{-11}$	mg/kg
Conc. of leafy vegetables from deposition	$1.47X10^{-11}$	$1.09X10^{-11}$	$5.04X10^{-12}$	$1.20X10^{-14}$	mg/kg
Conc. of produce from deposition	$1.47X10^{-11}$	$4.19X10^{-12}$	$1.68X10^{-12}$	$6.06X10^{-11}$	mg/kg
Conc. of leafy vegetables and produce from uptake	$3.26X10^{-11}$	$2.15X10^{-11}$	$1.87X10^{-11}$	$6.06X10^{-11}$	mg/kg

Table L-12.
Comparison of Parameters Used to Estimate Intakes of Deposited Nickel by Ingestion of Vegetables Using Separate Risk Methodologies (Continued)

Methodology	CAP93PC	CAPCOA	IECE	EPRI *TRUE*	
Parameter	Value	Value	Value	Value	Units
Intake through leafy vegetables	$2.33X10^{-12}$	$2.59X10^{-12}$	$2.00X10^{-13}$	$3.03X10^{-12}$	mg/day
Intake through produce	$2.28X10^{-11}$	$1.08X10^{-12}$	$5.93X10^{-12}$	$1.70X10^{-12}$	mg/day
TOTAL INTAKE	$2.51X10^{-11}$	$3.67X10^{-12}$	$6.13X10^{-12}$	$4.73X10^{-12}$	mg/day
RATIO OF CAP93PC TO OTHER METHODS	1	6.9	4.1	5.3	---

Table L-13.
Comparison of Parameters Used to Estimate Intakes of Deposited Nickel by of Meat and Milk Using Separate Risk Methodologies

Methodology	CAP93PC	CAPCOA	IECE	EPRI *TRUE*	
Parameter	Value	Value	Value	Value	Units
Concentration in chemical in meat	calculated	calculated	calculated	calculated	mg/kg
Concentration in chemical in milk	calculated	calculated	calculated	calculated	mg/kg
Fraction of pasture eaten by animal	1	1	1	1	unitless
Concentration of chemical in pasture grass	$2.95X10^{-11}$	$1.35X10^{-11}$	$8.06X10^{-10}$	$6.06X10^{-11}$	mg/kg
Concentration of nuclide in animal's feed	$2.95X10^{-11}$	$1.35X10^{-11}$	$8.06X10^{-10}$	$6.06X10^{-11}$	mg/kg
Amount of feed consumed by beef cattle per day	15.6	8	11.77	10.1	kg/day
Amount of feed consumed by dairy cattle per day	NA	16	16.9	11.13	kg/day
Concentration in soil from deposition	NA	$1.01X10^{-09}$	$8.80X10^{-10}$	$1.01X10^{-09}$	mg/kg
Amount of soil consumed by beef cattle each day	NA	0.4	0.6	1.8	kg/day
Amount of soil consumed by dairy cattle each day	NA	0.8	0.4	0.59	kg/day
Fraction of year animals graze on pasture	0.4	NA	NA	NA	unitless
Fraction of feed that is pasture grass when animals graze	0.43	NA	NA	NA	unitless

Table L-13.
Comparison of Parameters Used to Estimate Intakes of Deposited Nickel by of Meat and Milk Using Separate Risk Methodologies (Continued)

Methodology	CAP93PC	CAPCOA	IECE	EPRI *TRUE*	
Parameter	Value	Value	Value	Value	Units
Radioactive decay constant of nuclide	0	NA	NA	NA	day^{-1}
Fraction of daily intake in each kg/flesh (BTF)	0.006	0.002	0.003	0.004	day/kg
Fraction of daily intake in milk (BTF)	0.001	0.001	0.0055	0.0055	day/kg
Average time from slaughter to consumption	20	NA	NA	NA	days
Avg transport time of activity from feed to milk to receptor	2	NA	NA	NA	days
Dairy productivity = milk production of cow	11	NA	NA	NA	liters/day
Beef ingestion rate	0.233	0.075	0.041	0.075	kg/day
Milk ingestion rate	0.307	0.3	0.052	0.3	kg or L/day
Concentration in beef	$2.76X10^{-12}$	$4.33X10^{-13}$	$3.00X10^{-11}$	$9.79X10^{-12}$	mg/kg
Concentration in milk	$4.60X10^{-13}$	$4.33X10^{-13}$	$7.69X10^{-11}$	$6.97X10^{-12}$	mg/kg mg/L
Intake rate through beef	$6.43X10^{-13}$	$3.25X10^{-14}$	$1.23X10^{-12}$	$7.35X10^{-13}$	mg/day
Intake rate through milk	$1.41X10^{-13}$	$1.30X10^{-13}$	$4.00X10^{-12}$	$2.09X10^{-12}$	mg/day
TOTAL INTAKE	$7.85X10^{-13}$	$1.62X10^{-13}$	$5.23X10^{-12}$	$2.83X10^{-12}$	mg/day
RATIO OF CAP93PC TO OTHER METHODS	1	4.8	0.2	0.3	—
TOTAL INTAKE VEGETABLES/PRODUCE	$2.51X10^{-11}$	$3.67X10^{-12}$	$6.13X10^{-12}$	$4.73X10^{-12}$	mg/day
TOTAL INTAKE BEEF/MILK	$7.85X10^{-13}$	$1.62X10^{-13}$	$5.23X10^{-12}$	$2.83X10^{-12}$	mg/day
TOTAL ALL FOOD	$2.59X10^{-11}$	$3.83X10^{-12}$	$1.14X10^{-11}$	$7.55X10^{-12}$	mg/day
RATIO OF CAP93PC TO OTHER METHODS	1	6.8	2.3	3.4	—

L.4.2.5 Comparative Analysis

Tables L-12 and L-13 present a side by side comparison of the input parameters from each of the four different models used to estimate chemical concentrations and intakes for vegetables, produce, meat and milk. Employing the different mathematical models resulted

in a range of concentrations in vegetables and produce. The resulting intakes were likewise different between each methodology. The difference in chemical concentrations in vegetables and produce can be attributed to the different values recommended for crop yield, interception fractions and uptake factors. In each case, the chemical concentration from root uptake is the predominant contributor to the overall chemical concentration in plants. Consequently, the disparity in uptake factors resulted in a range of chemical concentrations. Similarly, each of the methodologies recommends a specific vegetable and produce ingestion rate.

The resulting chemical concentrations in beef and milk are also slightly different and are attributed to several factors. Because the methods used to estimate the concentration in crops and feed are similar to the ones used to estimate vegetable concentrations, the chemical concentrations in crops and feed are therefore also different. Furthermore, although there appears to be some similarity to the methods used to calculate chemical concentrations in animal tissue, the difference between the recommendations for feed and soil consumption rates by cattle resulted in a contrasting range of chemical concentrations.

The vegetable and produce intakes estimated using the CAP93-PC methodology resulted in a higher intake when compared to the other three models. Conversely, the estimated meat and milk intakes calculated from the EPRI TRUE method are the highest values. The meat and milk intakes from the CAP93-PC methodology was the third highest. Nevertheless, it is the intake of vegetables and produce that contribute the greatest amount to the total intake of food from deposition. The results of the combined total food intakes indicate that the CAP93-PC model is the most conservative methodology. The single most significant factor contributing to the higher result for CAP93-PC is the assumption regarding a daily consumption of 0.48 kg of produce. The chemical concentrations, the ingestion rates as recommended from each method, and the resulting intake rates are summarized in Table L-14.

Table L-14.
Summary of Indirect Exposure Calculations

Chemical Concentration	CAP93-PC	CAPCOA	IECE	EPRI *TRUE*	UNITS
Leafy vegetables	4.73×10^{-11}	3.24×10^{-11}	2.38×10^{-11}	6.06×10^{-11}	mg/kg
Produce	4.73×10^{-11}	2.57×10^{-11}	2.04×10^{-11}	6.06×10^{-11}	mg/kg
Beef	2.76×10^{-12}	4.33×10^{-13}	3.00×10^{-11}	9.79×10^{-12}	mg/kg
Milk	4.60×10^{-13}	4.33×10^{-13}	7.69×10^{-11}	6.97×10^{-12}	mg/kg
Assumed Ingestion Rates					
Leafy vegetables	0.05	0.08	0.008	0.05	kg/day

Table L-14.
Summary of Indirect Exposure Calculations (Continued)

Chemical Concentration	CAP93-PC	CAPCOA	IECE	EPRI *TRUE*	UNITS
Produce	0.48	0.042	0.3402	0.028	kg/day
Beef	0.233	0.075	0.041	0.075	kg/day
Milk	0.307	0.3	0.052	0.3	L/kg-day
Error! Bookmark not defined.Intake Rates of Ni-59					
Leafy vegetables	2.33×10^{-12}	2.59×10^{-12}	2.00×10^{-13}	3.03×10^{-12}	mg/day
Produce	2.28×10^{-11}	1.08×10^{-12}	5.93×10^{-12}	1.70×10^{-12}	mg/day
Beef	6.43×10^{-13}	3.25×10^{-14}	1.23×10^{-12}	7.34×10^{-13}	mg/day
Milk	1.41×10^{-13}	1.30×10^{-13}	4.00×10^{-12}	2.09×10^{-12}	mg/day
TOTAL INTAKE	2.59×10^{-11}	3.83×10^{-12}	1.14×10^{-11}	7.55×10^{-12}	mg/day
RATIO OF CAP93-PC TO OTHER METHODS	1	6.8	2.3	3.4	
The IECE consumption assumptions are described as 95th percentile estimates.					

L.5 Interpretation of the Analysis of Population and Individual Risks

The results presented in Figures L-1 and L-2 indicate that CAP93-PC calculates a range for the maximum lifetime individual risk, using a source term estimate based on fly ash emissions, of between 3.6×10^{-7} and 2.6×10^{-6}, with an average individual risk of 1.2×10^{-6}. These risk estimates correspond to an estimated annual effective dose equivalent of less than 0.016 to 0.185 mrem per year for the fly ash source term (the average MEI dose is 0.08 mrem/yr. For the NESHAPS source term, the lifetime risks range from slightly below 10^{-6} to 8.8×10^{-6}, with an average value of 5.4×10^{-6}. This range corresponds to an annual dose to the maximum exposed individual of between 0.075 and 1.13 mrem per year, with an average for the eight NESHAPS plants of 0.32 mrem per year. As an indication of the significance of such doses, it is noteworthy that the National Council on Radiation Protection and Measurement (NCRP) has defined a negligible individual dose rate at a level of 1 mrem per year, (NCRP 116, 1993), and that EPA has not considered such exposure levels to be significant in other regulatory contexts.

The individual radiation doses are low by conventional standards for radiation protection. However, it is customary in radiation protection to also consider whether population doses might be significant. For the eight NESHAPS plants using a fly ash-based source term, the population risk in terms of cancer mortality per year is estimated to range from 2.1×10^{-5} to 1.6×10^{-3} cancer fatalities per year, with an average value of 4.4×10^{-4} per plant per year. For a population of roughly 600 power plants, this would correspond to a

national estimate of about 0.2 cases per year in the U.S. There are methodological weaknesses with simply multiplying the average population risk per plant times the number of plants, (i.e., it does not consider actual population distributions around each plant) so this estimate is quite uncertain. Using the NESHAPS source term, the population risk estimate is, on average, about three times larger for the eight plants considered using the NESHAPS source term than that based on fly ash.

L.5.1 Consideration of Exposure Pathways

One interesting aspect of this analysis is that the exposure pathways of greatest significance for individual risk are not the same pathways that make the major contribution to population risk. For individual risk (see Figure L-3), exposures and risks are dominated by direct external exposure to radionuclides on the ground surface. For the eight plants analyzed, the ground surface exposure pathway contributed about 83% of the total risk, with risks from ingestion contributing about 16% of the total. Inhalation was not a significant contributor to individual risk (the average for the eight plants was that inhalation contributed 0.4% of the risk). This result was not sensitive to the use of the fly ash source term; similar results were obtained using the NESHAPS source term.

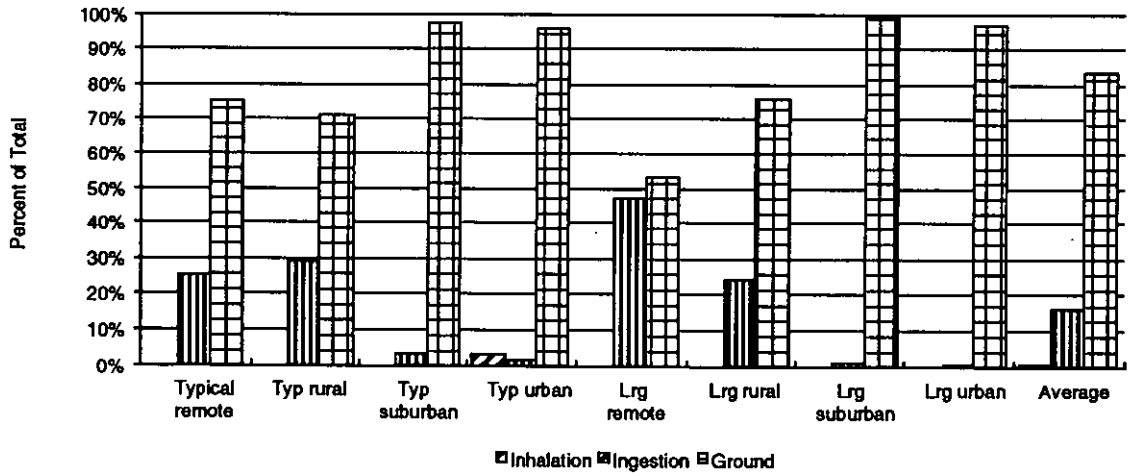

Figure L-3.
Maximum Individual Risk By Pathway Fly Ash Source Term with CAP93-PC

For population risk, inhalation is the dominant pathway. As indicated in Figure L-4, inhalation exposures make up about 84% of the total, with around 10% from ground surface exposures and 5% from ingestion. As with the calculation of individual risk pathways, these results are not sensitive to the use of the fly ash-based source term; similar results were obtained using the NESHAPS source term.

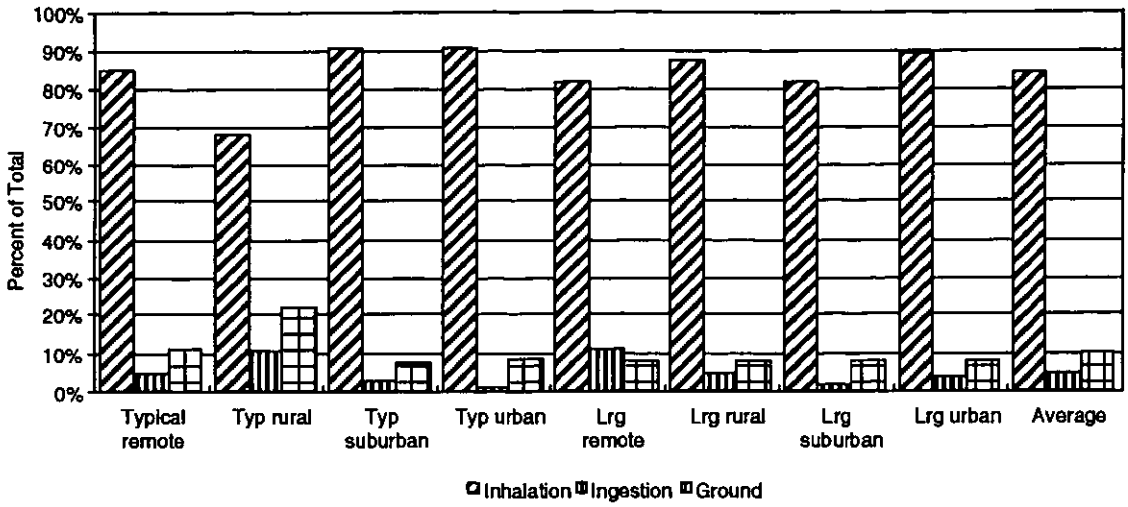

Figure L-4.
Population Risk By Pathway Fly Ash Source Term with CAP93-PC

There are several reasons for the differences in the relative importance of the three pathways. The maximum individual risk occurs at the closest occupied location, and, where the population pattern is relatively uniform, in the prevailing wind direction. The physical process that determines the maximum individual risk, at least in the case of the eight plants considered, is wet deposition of particulate radionuclides onto the ground surface and onto plants. Inhalation is not a significant contributor to risk at locations near the plants because with a tall stack and a buoyant plume, ground level concentrations are not typically affected inside a distance of several kilometers. The dose due to wet deposition rolls off fairly rapidly with distance, and is therefore sensitive to the location of the nearest individuals. The analytical method for calculating exposures from the ground surface involve the use of assumptions that appear to be quite conservative. Specifically, the exposure is based on the calculated dose one meter above an infinite flat plane onto which deposition has occurred for 100 years, after which a 70 year exposure period is computed. Allowance is made for radioactive decay and for a loss rate of 2% per year.

The population risk estimate is largely determined by the calculated inhalation doses. This analysis is less uncertain than the calculation for individual risk for several reasons. The inhalation dose is not dependent on a calculation of the buildup of past releases, nor is the result as sensitive to the precise location of individuals. Both analyses make certain conservative assumptions (e.g., that those exposed live outdoors at one location for 70 years).

L.5.2 Comparison with Chemical Risk Analysis Results

Although it may be desirable to describe the risks from emissions of both radioactive and chemically toxic substances from power plants, there are several methodological problems with combining the results of CAP93-PC with those from a chemical risk assessment, at least for calculations of individual risk. For purposes of making a comparison of the relative significance of risks from radionuclides versus chemical toxicants, the comparison is only valid to the extent that comparable analytical methods and assumptions have been applied. As the preceding analysis of the calculation of exposure through food consumption indicates, these assumptions are not comparable. An important limitation is that the maximum individual risk, as calculated by CAP93-PC, is likely to be at a different location than the point of maximum chemical risk. The location of the MEI in CAP93-PC is determined by the ground surface exposure term; this pathway has no chemical analog. Because these MEI risks are not at same place, nor would they apply to the same person, they should not be added.

There appear to be several observations that can be made based on the analyses done to date. First, it appears that the risk to the most exposed individual from radiation, taking the food and ground surface pathways into account, is greater than the risk to the most exposed individual from the inhalation of chemical toxicants. It may also be reasonable to conclude, to within the limits of uncertainty associated with both analyses, that the risks from radionuclides and chemicals are roughly equal. In any case, these results do not provide a basis to conclude that one is significantly greater than the other.

L.6 Conclusions

As this chapter notes, the calculated risks from radionuclide emissions appear to be lower than indicated by the analysis made in the 1989 NESHAPS. This is due both to an improved method for calculating the source term and from correction of the wet deposition analysis. This chapter also notes that the multipathway analysis made by CAP93-PC gives higher exposure estimates than other common methods for risk analysis. As the above summary table indicates, the ingestion exposure calculated by CAP93-PC is higher by a factor of 2.3 to 6.8 than that calculated by other models. For this reason, conclusions about the relative significance of radionuclide emissions and risks from power plants, in comparison to chemically toxic materials, may be misleading.

APPENDIX M
UNCERTAINTY ANALYSIS

Assessment of human health risks due to trace emissions from power plants should include explicit treatment of uncertainties. Such uncertainties arise in both models and data. A structured approach to the uncertainty analysis due to uncertainties (and variability) in data was developed. A brief description of this approach to uncertainty analysis and its application to two coal-fired power plants for carcinogenic chemicals and mercury was presented in Section 8.6. This Technical Appendix provides a more detailed description of both the approach and the applications.

M.1 Introduction

Uncertainties in health risk assessments arise in: (1) the formulation of the models used; and (2) the estimation of the values used as input to these models.

The uncertainty due to model formulation can be reduced to some extent by using models that provide a more comprehensive treatment of the relevant physical and chemical processes. Seigneur et al. (1992) provide some guidance on the selection of mathematical models for health risk assessment with various levels of accuracy in their formulation. The focus here is on the uncertainties due to the input parameters for a given health risk assessment model.

Uncertainties in parameter values arise for three reasons. First, the value may be based on measurements, with their associated imprecision. In the context of this report, however, errors of measurements are likely to be insignificant compared to other kinds of uncertainty. Second, the value may have been measured, but under circumstances other than those for which it must be applied. In this case, additional uncertainty arises from the variation of the parameter in time and space. Third, the value may not have been measured at all, but estimated from relationships with other quantities that are known or measured. In this case, uncertainty in the parameter of interest arises both from uncertainty in the quantities that are measured and from uncertainty about the estimating relationship.

By characterizing the uncertainties in model input parameters and studying the effects of variation in these parameters on the model predictions, we can estimate the part of the uncertainty in the predictions that is due to uncertainty or variability in the inputs.

Uncertainty can be characterized by a probability distribution. Sometimes such probability distributions can be usefully summarized by a few parameters, such as the mean and standard deviation. Uncertainties in the input parameters propagate through the model to produce probability distributions on the output parameters.

M.2 Approach

The following paragraphs describe a structured methodology for the parameter uncertainty analysis of health risk estimates and two applications of this methodology to the carcinogenic and mercury noncarcinogenic risks of Sites 12 and A, respectively. Site 12 was the first site for which a multimedia health risk assessment was conducted. Since both inhalation and ingestion were significant contributors to the carcinogenic health risk, this site was selected to demonstrate the application of an uncertainty analysis to a carcinogenic risk assessment. Site A presented the highest mercury health risk among the four case studies considered for multimedia assessment. Consequently, this site was selected for the application of the uncertainty analysis to mercury health risk. The proposed methodology involves: (1) a sensitivity analysis of the model used to perform the health risk calculations; (2) the determination of probability distributions for a number of selected input parameters (i.e., the ones identified as most critical to the output variable); and (3) the propagation of the uncertainties through the model.

In summary, this methodology is viewed as consisting of the following 6 steps:

- Step 1: *Sensitivity analysis of the health risk assessment model.* This analysis allows one to determine the influential parameters of the model, i.e., those that have the most significant effect on the model output.
- Step 2: *Estimation of Parameter Uncertainty.* This task involves the derivation of "uncertainty indexes" which quantify the uncertainty in the individual parameter values. Combination of the uncertainty indexes with the results of the sensitivity analysis leads to the selection of the critical parameters, i.e., those that need to be included in the final uncertainty analysis.
- Step 3: *Construction of response surface models with critical parameters as the sole variables.* A response surface model is a parameterized version of the original model that allows one, for a specific site, to reduce computational costs while maintaining reasonable accuracy. This approach also reduces the number of parameters in the analysis, simplifying the uncertainty propagation process.
- Step 4: *Selection of probability distributions for the critical parameters.*
- Step 5: *Propagation of the parameter uncertainties.* This task is performed with the parameterized version of the model and provides the uncertainties in the model outputs.
- Step 6: *Analysis of the probability distribution of the risk estimates.*

A more detailed description of each individual step of the methodology is presented in the following paragraphs.

M.2.1 Sensitivity Analysis

Some parameters have a greater impact on results than others. The uncertainty analysis should focus on those parameters to which the calculated health risks are most sensitive. The sensitivity analysis allows us to determine the parameters to which risks are most sensitive, termed *influential parameters.*

The sensitivity of the model output (i.e., the dependent variable) to a model input parameter can be measured by the ratio of the change in the model output to the perturbation in the input parameter. We define this ratio as the sensitivity index, SI. For parameter i:

$$SI_i = \frac{\Delta Y}{\Delta X_i} \tag{1}$$

where ΔX_i is the perturbation in the input parameter, and ΔY is the corresponding change in the model output. In order to compare the sensitivity indexes for various input parameters, it is appropriate to use a dimensionless representation of the sensitivity index:

$$SI_i^* = \frac{\frac{\Delta Y}{\overline{Y}}}{\frac{\Delta X_i}{\overline{X}_i}} = \frac{\Delta Y^*}{\Delta X_i} \tag{2}$$

where $\overline{X}_i$ and $\overline{Y}$ are the mean or some other reference values of the variables, X_i and Y, respectively; and ΔY^* and ΔX_i^* refer to relative normalized perturbations.

Two characteristics of the sensitivity index are:

- The value of the sensitivity index is a function of the value of and the perturbation in the input parameter except for cases where the relationship between the model output variable and the input parameter is linear.
- The value of the sensitivity index may be a function of the value of the other model input parameters except for cases where the relationship between the model output and the input parameters is linear.

M.2.2 Estimation of Parameter Uncertainty

Even though the sensitivity index, as defined above, sufficiently describes the effect on the model result for a given change in the input parameter, it does not provide a measure of the range of variation in the model output, given the expected range of variation of the input parameter. In other words, a parameter that has a high sensitivity index, may have little effect on the model output if that parameter can only have a very small variation. The height of a power plant stack is an example of a parameter that has a significant effect

on atmospheric ground-level concentrations but has a small uncertainty. For the case of the power plant studied here, a 100% change in stack height caused a 73% change in the resulting concentrations. Stack height uncertainty, however, is not expected to be more than ± 2%. Consequently, the actual influence of this parameter on the model result is very small.

We define the uncertainty index as a measure of the uncertainty associated with a parameter X_i:

$$U_i = \frac{\sigma_i}{\overline{X}_i} \tag{3}$$

where: σ_i is the standard deviation of the parameter distribution

$\overline{X}_i$ is the mean value of parameter X_i

This uncertainty index (coefficient of variation) provides a measure of the variation of parameter X_i.

The combination of the model sensitivity to a parameter, and the uncertainty in that parameter provides the information required to assess which parameters need to be included in the uncertainty analysis. We define a sensitivity/uncertainty index as follows:

$$I_i = (SI_i)(UI_i) = \frac{\Delta Y^*}{\Delta X_i^*}\frac{\sigma_i}{\overline{X}_i} \tag{4}$$

The sensitivity/uncertainty index, therefore, constitutes a measure of the effect that a parameter has on the model results and can be used to select the model *critical* parameters to be included in the uncertainty analysis.

Even though the concept of the standard deviation of a parameter was used in the definitions of the uncertainty and sensitivity/uncertainty indexes, it is rather unlikely that actual standard deviations will be available for all the parameters examined in the sensitivity analysis. Since the sensitivity analysis is a screening procedure whose goal is to minimize the number of parameters included in the final uncertainty analysis, it is generally appropriate to use other measures that are more readily available to characterize the variability of a parameter. For example, the expected range of variation can be used instead of an actual standard deviation.

M.2.3 Response Surface Construction

The TRUE multimedia model uses a large number of input parameters and comprises several individual models for simulating fate and transport, exposure, dose, and health effects. Such a model can be computationally very demanding and performing an uncertainty analysis for a large number of parameters may, therefore, not be feasible. It is, therefore, necessary to parameterize the various model components in order to reduce the magnitude of the computations. This model parameterization can be achieved by constructing response surfaces.

A response surface is a simplified version of the actual model which can be used efficiently in the uncertainty analysis as a replacement of the real model. In the case of simple analytical models in the form of a single equation (e.g., dose models) only minor simplifications need to be made for the construction of the response surface. Such simplifications can be accomplished by factoring out of each of the terms of the equation the selected critical parameters, and representing the remaining part of the term by a lumped parameter calculated from previous results of the model. So, the response surface can be of the following form:

$$rY = \sum_{i=1}^{m} (X_{i1}^{P_{i1}} \cdots X_{iK_i}^{P_{iK_i}}) A_i \qquad (5)$$

where:

m = Number of terms in dependent variable expression

K_i = Number of independent variables included in term i

A_i = Calculated lumped parameter of term i

P_{il} = Exponent of parameter X_{il}

In the case of complex models (e.g., environmental transport models) the response surface can be developed using the following procedure: For all critical parameters of a model component select a number K of parameter sets, $\mathbf{X_i} = (X_{i1}..X_{in})$ i = 1,K and perform experimental runs of the actual complex model. Then use the pairs of parameter sets $\mathbf{X_1}....\mathbf{X_k}$, and corresponding models results $Y_1...Y_k$ to construct the response surface.

It must be noted that a response surface is a parameterization of the model that is typically specific to a given model application. That is, some of the case study characteristics (e.g., meteorology, hydrology) are implicitly included in the constant parameters of the response surfaces.

M.2.4 Selection of the Probability Distributions for the Critical Parameters

Once the critical parameters have been identified, and the response surfaces for each model component constructed, probability distributions must be selected to represent each one of the parameters.

As was mentioned previously, a parameter value can be either directly measured or indirectly estimated through an estimation procedure which usually involves fitting of a curve through a set of experimental points.

In the case of a directly measured parameter, the uncertainty results from uncertainty in the measurement process, and this can sometimes be estimated from repeated measurements. Often, however, the amount of available data is not enough to produce meaningful histograms or probability plots. What is usually available is a range of values within which the true value of a parameter is expected to lie, and possibly a most likely, or range of most likely values for the parameter. In this case, it is left to our judgment and experience to decide what probability distribution is appropriate.

In the case of parameters estimated indirectly through curve fitting (e.g., bioconcentration factors, and cancer potency factors) uncertainty results both from statistical errors in fitting the curve, which can be estimated by statistical procedures, and uncertainty about the form of the curve, which is a matter of judgment.

A priori expert judgment can be combined with available direct or indirect measurements using the Bayesian method (Wadsworth, 1990). If the measurements are direct, precise and numerous enough to sufficiently describe the variation pattern of the parameters, then the *a priori* judgment may have little or no influence on the resulting probability distributions. Conversely, if the measurements are indirect and imprecise, then the *a priori* judgment may be of great importance.

M.2.5 Propagation of the Model Uncertainties

At this step, the response surfaces developed for the different model components can be combined in a single spreadsheet that performs the function of the overall risk assessment model in a simplified fashion for the case study considered. Several techniques exist to develop a probability distribution in the model output given probability distributions in the model input parameters. Monte-Carlo and Latin hypercube simulations are standard examples of such techniques (Decisioneering Inc., 1993). In the special cases where the probability distributions are similar and simple (e.g., normal), the probability distribution in the model output can be calculated analytically. For the general case where no simple analytical approach can be used, however, the spreadsheet model can then be coupled to one of several commercial software packages, which uses the specified probability distributions of the parameters together with the spreadsheet calculations to generate a set of synthetic model results.

M.2.6 Analysis of the Probability Distribution of the Model Health Risk Estimates

If the number of replications for the probabilistic synthetic simulations is large enough, the synthetic results can be statistically analyzed to yield a reliable probability distribution of the dependent variable (i.e., health risk). If the uncertainty analysis procedure was performed correctly, this probability distribution should represent a more realistic characterization of the anticipated health risks, as it provides a range of possible values accompanied by their corresponding likelihoods instead of a single, deterministic point estimate. Recall, however, that we do not capture the uncertainties associated with the model itself.

M.3 Power Plant Example 1—Uncertainty Analysis of Carcinogenic Health Risk Estimates

The case study presented in the following paragraphs corresponds to the emissions of Site 12, a deterministic analysis of which was presented in the "TRUE Case Studies" section of Section 8. The results subject to uncertainty analysis correspond to the carcinogenic health effects in the subregion of maximum risk. Noncarcinogenic risks are not addressed here.

Four chemicals with listed carcinogenic effects were detected in the emissions of the 200 m high stack of the facility: chromium, arsenic, cadmium, and benzene. The corresponding chemical emission rates for annual power plant capacity were estimated to be 2.45×10^{-3}, 3.10×10^{-4}, 7.98×10^{-4}, and 4.9×10^{-4} g/s, respectively. Since chemical speciation for chromium was not available, the corresponding deterministic health effect calculations were performed based on the assumption that total chromium emissions consisted of 5% Cr(VI) and 95% Cr(III). The cumulative carcinogenic lifetime risk from all chemicals and pathways in the subregion of maximum risk was calculated to be 1.4×10^{-8}.

Arsenic was calculated to be the major contributor to carcinogenic risk with a contribution of 57%. Chromium (VI) and cadmium followed with equal contributions of 21%. The contribution of benzene was insignificant. Among the three exposure pathways considered in the analysis, ingestion and inhalation were the two major contributors with comparable contributions of 52 and 48%, respectively. Dermal absorption had an insignificant contribution of 0.4%.

Produce was calculated to be the food chain component which contributed most to ingestion risk, with a contribution of 95%. Soil and fish ingestion had small contributions of 2.6 and 2.3%, respectively, and drinking water had an insignificant contribution of 0.1%.

It should be noted that among the four carcinogenic chemicals included in the analysis, only arsenic and benzene are considered to be carcinogenic through non-inhalation pathways. Since benzene's contribution was very small, benzene was not included in the uncertainty analysis. Even though arsenic is considered carcinogenic through the inges-

tion pathway, no cancer potency value has been tabulated since October, 1991 for this pathway in the Integrated Risk Information System (IRIS) database. The value used for this parameter in the deterministic health risk assessment was the most recent value that had been previously tabulated in IRIS.

M.3.1 Sensitivity Analysis

Sensitivity analysis of the individual model components as well as the overall multimedia health risk assessment model was performed to help identify the influential parameters. A total of 52 parameters were examined for potential inclusion in the final uncertainty analysis.

Sensitivity/uncertainty indexes of the input parameters were derived for each of the individual model components as well as for the overall risk assessment model. Based on the calculated sensitivity/uncertainty indexes of the 52 parameters, 22 (i.e., the ones with sensitivity/uncertainty indexes greater or equal to 0.05) were selected to be included in the final uncertainty analysis.

M.3.2 Response Surfaces

Response surfaces were constructed for each of the multimedia health risk assessment model components. In the case of simple models such as the food chain, exposure-dose, and risk models, the response surfaces were constructed manually by factoring out the influential parameters, and representing the remaining parts of the equations by constants calculated based on the model results. In the case of the more complex environmental transport models, additional sensitivity runs were performed by varying the critical parameters within their assumed range of variation.

In the case of the atmospheric transport model, ISC-LT, four critical parameters were identified: the chemical emission rate (Q_e), the stack exit velocity (V_s), the stack exit temperature (T_s), and the ambient air temperature (T_a). The influences of T_s and T_a were found to be correlated, as what affected the results was the difference between the stack and the ambient temperature, (T_s-T_a) and not their absolute values. Consequently, the two parameters were treated together as one, the temperature difference (T_s-T_a).

The equation describing the resulting simplified atmospheric transport model is:

$$C_a = aQ_e F_1 F_2 A_{11} \quad (6)$$

where:

$$F_1 = A_{12} + A_{13} V_s + A_{14} V_s^2 \quad (7)$$

$$F_2 = A_{15} + A_{16} (T_s-T_a) + A_{17} (T_s-T_a)^2 \quad (8)$$

where:

C_a = ground-level air concentration;

Q_e = Chemical emission rate;

a = Chemical speciation fraction (applies only to chromium case);

V_s = Stack exit velocity;

T_s = Stack exit temperature;

T_a = Ambient temperature;

A_{1j} = Constant j for model component 1

It should be noted that model components corresponding to media other than the atmosphere and pathways other than inhalation apply only to the arsenic case. Cadmium and chromium are not considered carcinogenic through non-inhalation pathways. Consequently, only the atmospheric transport, inhalation dose, and inhalation risk components apply to their case.

M.3.3 Probability Distribution Selection

Evaluation of the probability distributions of the 22 critical parameters of the model was performed on the basis of available statistical data, literature value ranges, and personal judgment. The selected probability distributions, and the information on which the distribution types and parameters where based are summarized in Table M-1.

Table M-1.
Uncertainty Analysis of Carcinogenic Health Risk Estimates Probability Distribution Selection

Influential Parameter Index	Parameter Symbol (Units)	Base Case Value	Probability Distribution		Primary Uncertainty Source	Comments
			Type	Parameters		
1	Qe1 — Arsenic (g/s)	3.1×10^{-4}	Lognormal	$\mu' = -8.05$ $\sigma' = 0.395$	U,V	Type suggested by existing literature data (Berkenpas and Rubin, 1992). Parameters calculated based on measurements.
2	Qe2 — Cadmium (g/s)	8.0×10^{-4}	Lognormal	$\mu' = -7.28$ $\sigma' = 1.06$	U,V	Type suggested by existing literature data (Berkenpas and Rubin, 1992). Parameters calculated based on measurements.
3	Qe3 — Chromium (g/s)	2.5×10^{-3}	Lognormal	$\mu' = -6.67$ $\sigma' = 1.30$	U,V	Type suggested by existing literature data (Berkenpas and Rubin, 1992). Parameters calculated based on measurements.
4	a—Chrom (VI) (—)	0.05	Normal	$\mu = 0.06$ $\sigma = 0.015$	U,V	Type assumed. Parameters calculated based on measured range.
5	Ts (K)	323	Uniform	$a = 318$ $b = 328$	V	Type assumed. Range based on personal judgement.
6	Vs (m/s)	13	Uniform	$a = 10.4$ $b = 15.6$	V	Type assumed. Range based on personal judgement.
7	Ta (K)	283	Uniform	$a = 278$ $b = 288$	V	Type assumed. Range based on personal judgement.
8	Vd (m/s)	0.005	Normal	$\mu = 0.005$ $\sigma = 0.0025$	U	Type assumed. Parameters calculated based on range reported in the literature (Zannetti, 1990).
9	dp (mm)	0.35	Lognormal	$\mu' = -1.1$ $\sigma' = 0.94$	V	Type and parameters estimated based on measurements.

Table M-1.
Uncertainty Analysis of Carcinogenic Health Risk Estimates Probability Distribution Selection (Continued)

Influential Parameter Index	Parameter Symbol (Units)	Base Case Value	Probability Distribution		Primary Uncertainty Source	Comments
			Type	Parameters		
10	J (mm/hr)	6.5	Normal	$\mu = 6.5$ $\sigma = 0.33$	V	Type assumed. Parameters calculated based on a ± 10% variation.
11	u(m/s)	9.2	Normal	$\mu = 9.2$ $\sigma = 0.46$	V	Type assumed. Parameters calculated based on a ± 10% variation.
12	ds (m)	0.05	Lognormal	$\mu' = -3.10$ $\sigma' = 0.75$	U,V	Type assumed. Parameters calculated based on assumed range 0.01-0.20.
13	ED (yr)	70	Lognormal	$\mu' = 2.24$ $\sigma' = 0.58$	V	Type suggested by EPA data (EPA, 1989). Parameters calculated based on EPA provided percentiles (EPA, 1989).
14	EST (yr)	0	Uniform	a = 0 b = 100	V	A 100 year power plant life was assumed.
15	BCFp (—)	0.04	Uniform	a = 0.01 b = 0.05	U	Type assumed. Ranged provided by Baes el al., 1993.
16	IR (m3/d)	20	Triangular	a = 14 $\mu = 22$ b = 30	V	Minimum, maximum, and most likely values provided by EPA, 1989.
17	INRp (kg/d)	0.08	Lognormal	$\mu' = -2.58$ $\sigma' = 0.61$	U,V	Type and parameters based on homegrown percentage consumption data provided by EPA, 1989.
18	BW (kg)	70	Lognormal	$\mu' = 4.2$ $\sigma' = 0.2$	V	Type suggested by Brainard and Burmaster, 1992, parameters based on data reported by the U.S. Public Health Service (National Health Survey, 1987).

Table M-1.
Uncertainty Analysis of Carcinogenic Health Risk Estimates Probability Distribution Selection (Continued)

Influential Parameter Index	Parameter Symbol (Units)	Base Case Value	Probability Distribution		Primary Uncertainty Source	Comments
			Type	Parameters		
19	CSF11 — Arsenic (kg-d/mg)	5.0	Uniform	a = 3.1 b = 9.4	U	Type assumed. Range based on the results of different studies (Viren and Silvers, 1994).
20	CSF21 — Arsenic (kg-d/mg)	1.75	Triangular	a = 0 μ = 1.75 b = 15.0	U	Type assumed. Parameters calculated based on a zero lower and a 15 upper bound. Upper bound corresponds to reported CSF for a Taiwanese population (EPA, 1984b).
21	CSF12 — Chromium (VI) (kg-d/mg)	42.0	Triangular	a = 10.5 μ = 42.1 b = 295.2	U	Type assumed. Lower bound, upper bound, and best estimate provided by EPA (1984c) based on uncertainties in the epidemiological study.
22	CSF13 — Cadmium (kg-d/mg)	6.3	Normal	μ = 6.3 σ = 2.93	U	Type and parameters based on data analysis for the CSF derivation provided by EPA, 1985.

Explanations: The above-listed probability distribution types are defined as follows:

- Uniform [a,b]
- Normal (μ,s)
  where: a = minimum value
  where: μ = distribution mean
  b = maximum values = distribution standard deviation
- Triangular [a,μ,b]
- Lognormal (μ′,σ′)
  where: a = minimum value where μ′ = mean of underlying normal distribution
  μ = most likely values ¢ = standard deviation of underlying normal distribution
  b = maximum value
- Primary Uncertainty Source:
  U: Uncertainty (i.e., inaccuracy in measurement or estimation method for the parameter value)
  V: Variability (i.e., temporal, spatial, or population variation of the parameter)

In a health risk assessment, the uncertainty associated with the health effect parameters (i.e., cancer potency slope factors in the case of the present application) is of major importance. The EPA recommended values for these parameters are usually derived based on limited animal or epidemiological studies, the conditions of which may differ significantly from the conditions for which these values will be applied in a risk assessment.

In the case of epidemiological studies, uncertainty is associated with high-to-low dose extrapolation and factors related to secondary exposures, diet, and hygiene of the population under study. The data are fitted by an assumed model, and the maximum likelihood estimate (MLE) is usually recommended for use by EPA.

In the case of animal studies, uncertainty is associated with interspecies as well as high-to-low dose extrapolations. Due to the additional uncertainty of interspecies extrapolation in this case an upper bound value (95^{th} percentile) is usually recommended for use by EPA.

The cancer potency slope factors (CSFs) for the three chemicals included in this application were all derived based on epidemiological studies.

In the case of arsenic inhalation, EPA currently recommends a CSF value of 15.0 $(mg/kg\text{-}d)^{-1}$. This value was derived utilizing data from two separate U.S. smelter worker populations in Tacoma and Montana [20]. A recent analysis performed by EPRI (Viren and Silvers, 1994) combining information from the above two smelters with recent findings from a Swedish study, led to a CSF value which is lower than that of EPA by a factor of 3. The primary cause of the decline in the value of this newly derived CSF was the reevaluation of the dosimetry in the case of the Tacoma population. The new value recommended by EPRI was derived by obtaining the geometric mean of the individual CSF values for the three populations. In this application, we chose to represent the uncertainty of the arsenic inhalation CSF by a uniform distribution extending over the range of values derived in the three different studies.

In the case of arsenic ingestion, the CSF derivation was based on an epidemiological study of a Taiwanese population exposed to high arsenic concentrations in drinking water [20]. Due to the uncertainties associated with its derivation, this CSF was removed from IRIS in October, 1991 until a more reliable value could be derived. In this application we chose to represent the uncertainty of the arsenic ingestion CSF by a triangular distribution with lower bound equal to zero, most likely value equal to the scaled value most recently listed in IRIS (September, 1991), and upper bound equal to the value originally derived from the Taiwanese population data.

In the case of cadmium inhalation, the CSF derivation was based on epidemiological data of a U.S. smelter worker population (EPA, 1985). The EPA-recommended value was determined by fitting a linear non-threshold model to these data. We recognize a great deal of uncertainty exists in the model; nonetheless, we assumed the model was appropriate for

this analysis. A 90% confidence interval based only on statistical consideration was constructed around the MLE. In this application we chose to represent uncertainty associated with the cadmium inhalation CSF by a normal probability distribution with mean equal to the maximum likelihood estimate and standard deviation calculated from the estimated confidence interval.

In the case of chromium (VI) inhalation, the CSF derivation was based on a U.S. population of chromate plant workers [21]. The EPA-recommended value was derived by fitting a two-stage model to the data. A lower bound and an upper bound were constructed around the MLE to account for the possibility of underestimation or overestimation of the exposure due to lack of consideration of poor hygiene, smoking habits, and chemical speciation in the analysis. In this application, we chose to represent the uncertainty associated with the chromium (VI) CSF by a triangular distribution defined by the best estimate, upper, and lower bounds.

M.3.4 Monte Carlo Analysis: Health Risk Probability Distribution

The derived response surfaces were combined in a simplified spreadsheet model which was coupled to the software package Crystal Ball (Decisioneering Inc., 1993) which performed the propagation of the input parameter uncertainties through the model. A Latin Hypercube analysis with 10,000 iterations of the simplified model was performed to produce a synthetic set of carcinogenic health risks associated with the studied coal-fired power plant. Statistical analysis of the synthetic results yielded a probability distribution for the risk. The risk value calculated in the deterministic risk assessment (1.4×10^{-8}) was estimated to be at the 80th percentile of the derived probability distribution. The statistical parameters of this distribution are summarized below:

- Mean (expected value) $\mu = 1.2 \times 10^{-8}$ (84% of the deterministic value)
- Mode (most probable value) $M_o = 1.9 \times 10^{-9}$
- Standard Deviation, $\sigma = 2.0 \times 10^{-8}$
- Skewness $= 6.9$ (positively skewed—right tail)
- Percentiles:
  - 5% $F_{0.05} = 1.2 \times 10^{-9}$
  - 25% $F_{0.25} = 3.0 \times 10^{-9}$
  - Median, 50% $F_{0.5} = 6.0 \times 10^{-9}$
  - 75% $F_{0.75} = 1.3 \times 10^{-8}$
  - 95% $F_{0.95} = 4.1 \times 10^{-8}$

The derived probability density plot for the carcinogenic risk is presented in Figure M-1.

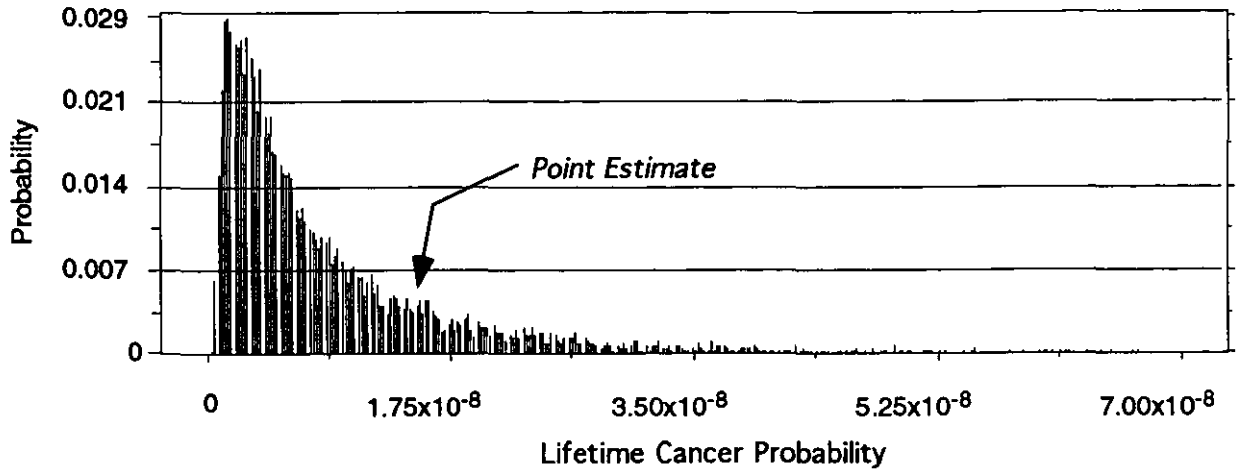

Figure M-1.
Distribution of Multimedia Risk for Arsenic, Cadmium, and Chromium, Case Study Site 12

The major parameters governing the uncertainty were the exposure duration and the cancer potency slope factor for ingestion of arsenic.

M.4 Power Plant Example 2—Uncertainty Analysis of Mercury Health Risk Estimates

Among the different chemicals emitted from fossil-fueled power plants, mercury requires special attention, as studies have shown mercury to exist at concentrations in some aquatic environments that may lead to potential ecological and health concerns. This section describes the key issues and uncertainties for assessment of mercury health risks and presents an application of the TRUE uncertainty analysis extensions for the mercury emissions of case study Site A. Ecological impacts are not considered in this analysis.

The estimation of public health effects associated with mercury emissions typically involves: (1) modeling of the fate and transport of mercury in the environment (i.e., via air, water, soil, and intermedia); (2) prediction of the corresponding concentrations in the food chain; and (3) estimation of the resulting human exposure doses and subsequent health effects.

Large uncertainties are involved in all three parts of the calculations. Such uncertainties arise from the formulation of the models used to perform the calculations, and from the estimation of the parameter values used as input to these models. Such parameters may include mercury's physical state in the plume, mercury atmospheric chemical speciation, dry deposition velocities and rain scavenging coefficients of gaseous and particulate mercury, methylated fraction of mercury in the aquatic environment, food chain bioconcentration factors, and toxicological parameters. Because epidemiological data are

presently being analyzed to develop reference doses for mercury and results from these analyses are not yet available, no attempt was made to include uncertainties in toxicological parameters.

The following paragraphs provide a discussion of the chemical speciation, environmental cycling and toxicological effects of mercury, and a quantitative estimation of the uncertainty in the health risk estimates associated with the mercury emissions of Site A, the deterministic analysis of which was presented in the "TRUE Case Studies" section of this chapter.

M.4.1 Chemical Forms

Mercury is primarily present in the environment in two oxidation states: elemental mercury, Hg^0, and divalent mercury, Hg (II); the divalent compounds can be present in the atmosphere in either gas, liquid, or solid phase.

Compounds of Hg (II) have the highest solubility, whereas elemental mercury has a high vapor pressure and lower solubility in water. Mercury exists both in the form of inorganic and organic compounds in the environment. The organic compounds of mercury, which are primarily found in aquatic environments, are of particular environmental interest because they have a tendency to bind to sulfhydryl groups on proteins, and can, thus, accumulate in biota and particularly in large fish.

The total concentration of mercury in the atmosphere is generally dominated by elemental mercury vapor (Seigneur et al., 1994). Lindberg et al. (1991) report a particulate (i.e., mainly Hg (II) inorganic compounds) to total mercury ratio of 0.5%, measured during a study at Walker Branch Watershed in Tennessee. Organic mercury is present in the atmosphere at very low concentrations.

M.4.2 Mercury Fate and Transport

Mercury emitted from a source undergoes a number of changes that govern its distribution and resulting chemical forms in the various environmental media. Some of these processes are poorly understood, however, thus increasing the uncertainty in the fate and transport models. Of these processes, we focus on the following four: (1) Atmospheric chemical transformations; (2) Dry deposition; (3) Wet deposition; and (4) Aquatic chemical transformations.

Atmospheric Chemical Transformations. Chemical transformations of mercury in the atmosphere may occur in the gaseous and the aqueous phases. Gas-phase mercury chemistry involves primarily oxidation of Hg (0) by common atmospheric species (e.g., Cl_2, O_3, HCl, H_2O_2, etc.), leading to the formation of Hg (II) inorganic compounds. Aqueous mercury reactions can occur in rain, cloud, or fog water as well as in the moisture associated with hygroscopic aerosols. These reactions involve the oxidation of Hg (0) to Hg (II) by ozone,

the reduction of Hg (II) to Hg (0), and the complexation/dissociation of various Hg (II) species. The oxidation of Hg (0) by organic acids, photolytic reduction of divalent mercury adsorbed on soot and the formation of organo-mercurials (e.g., methylmercury) is also possible. The chemical reaction mechanism of inorganic atmospheric mercury is fairly well understood; however, there are still large uncertainties regarding the kinetics of these reactions (Seigneur et al., 1994).

Mercury speciation studies have shown organic mercury to exist at relatively insignificant concentrations in the atmosphere. Fitzgerald et al. (1991) report methylmercury concentrations lower than 0.7% of the total gaseous mercury in a lacustrine region in north-central Wisconsin.

Based on the above information on chemical transformations and speciation studies, it is reasonable to assume that mercury atmospheric chemistry is mainly inorganic.

Dry Deposition. The term dry deposition expresses the uptake of chemicals at the earth's surface, and combines the effects of gravitational settling and interaction with terrain features. In mathematical models, dry deposition is often parameterized through the use of a deposition velocity, V_d, which represents the ratio between the deposition flux and the atmospheric concentration. Despite the use of the term "velocity," V_d is essentially an effective velocity, derived from dimensional analysis. The processes dominating dry deposition depend on the physical state of the chemical as well as on the particle size distribution, in the case of particulate bound chemicals.

In the case of large particles, dry deposition is usually dominated by gravitational effects (i.e., sedimentation), and can be characterized by an actual settling velocity, which is a function of particle size. In the case of small particles, dry deposition is dominated by Brownian motion, which allows rapid movements of the particles through the viscous sublayer of the atmospheric boundary layer [Zannetti, 1990]. In the case of intermediate size particles, both Brownian motion and gravitational settling are weak, and dry deposition is dominated by surface impaction and interception phenomena. Since such phenomena are difficult to quantify, the determination of a dry deposition velocity for these particles is highly uncertain.

Dry deposition of gaseous chemicals is probably the most complex, and inadequately characterized of all dry deposition cases. The dominating processes in this case are chemical interactions with the deposition surfaces. The determination of an effective deposition velocity is, therefore, dependent on the physical and chemical characteristics of these surfaces as well as the gas.

As mentioned earlier, most of the mercury in the atmosphere is found in the form of elemental mercury vapor. Dry deposition velocities of Hg (0) vapor have been measured and predicted by several investigators for different types of deposition surfaces. Barton et al. (1981) reported values ranging between 0.06 and 0.1 cm/s for a tall grass canopy, based

on field gradient measurements. Data from the chamber studies of Lindberg et al. (1979) suggest a range of dry deposition velocities between 0.03 and 0.1 cm/s for an alfalfa canopy. Also, Lindberg et al. (1991) report predicted mean weekly values of Hg (0) deposition velocity in a deciduous forest in Tennessee ranging between 0.006 and 0.12 cm/s.

Reported deposition velocities for particulate mercury are also low. Lindberg et al. (1991), in their deciduous forest study, predicted V_d values for particulate mercury ranging between 0.02 and 0.11 cm/s during the growing season, and a lower average of 0.003 cm/s during the dormant period.

Wet Deposition. Wet deposition is the removal and subsequent deposition of chemicals in the atmosphere by precipitation, fog, and cloud droplets. Unlike dry deposition, which occurs in the lower layers of the plume above the deposition surface, wet deposition affects the entire volume of the plume inside the fog, cloud, or precipitation layer.

In mathematical models, wet deposition due to precipitation is often parameterized through the use of a washout (or scavenging) coefficient, which is a function of the precipitation intensity, particle size (in the case of particulate matter), and solubility (in the case of gases). This washout coefficient can be used with the wet deposition flux to define a wet deposition velocity, V_w. Wet deposition phenomena due to clouds and fog are currently only poorly understood.

Aquatic Chemical Transformations. Mercury deposited or transported into surface water bodies may undergo a variety of chemical and biological transformations. An accurate quantitative characterization of these transformation processes has, however, not yet been achieved.

The transformation process of greatest concern in assessment of health impacts is the methylation of mercury. During the process of methylation, bacteria present in the aquatic environment transform dissolved inorganic mercury into methylmercury compounds. The reverse process (i.e., transformation of methylmercury back to inorganic) is also possible, and is called demethylation. The rates of methylation and demethylation in a given aquatic environment depend on a variety of environmental factors, including the amount of dissolved organic carbon present, the acidity of the water (low pH favors methylation), water temperature, nutrient levels, and available oxygen. Even though these factors have been qualitatively identified as having an effect on these processes, their effect has not yet been satisfactorily quantified.

Field measurement studies have shown organic mercury in aquatic environments to exist at relative (to inorganic) concentrations significantly higher than the corresponding ones in the atmosphere, even in remote water bodies whose only source of contamination is atmospheric deposition (Fitzgerald et al., 1991). This observation suggests that most organic mercury in water bodies is generated in place, from methylation of deposited inorganic mercury.

M.4.3 Food Chain Bioconcentration

Mercury in the environment may enter the different components of the food chain through a variety of pathways.

Mercury uptake by plants may occur through direct deposition from the atmosphere onto the plant surfaces, foliar uptake of mercury vapor from air, and root uptake of mercury accumulated in surface soil. The effect of direct deposition may be parameterized from the corresponding mercury deposition rates and the characteristics of the crop surface. Foliar uptake can be quantified using the mercury concentration in air and mercury's volatilization tendency (i.e., Henry's law constant). Root uptake is described through the use of a "bioaccumulation factor" (BAF). This soil-to-plant BAF expresses the ratio of the mercury concentration in plants to that in the soil around the plant roots. Values of the BAF depend on the specific mercury species and are usually derived experimentally.

Mercury contamination of fish may result from direct uptake of mercury in water through the gill membrane and ingestion of mercury with food including fish at lower trophic levels. The effects of these different pathways are typically lumped together in a bioconcentration factor, which relates the concentration of dissolved mercury in the water body to the resulting mercury concentration in fish. The value of the resulting BAF depends on the chemical form of mercury as well as the type and trophic level of the fish.

The results of existing studies suggest that methylmercury bioconcentrates to a greater degree than inorganic mercury. The U.S. EPA reports BAFs ranging from 130 to 10,000 for inorganic mercury, and 11,000 to 86,000 for methylmercury for different types of fish [19].

M.4.4 Mercury Toxicity

The different forms of mercury in the environment exhibit widely different toxicities and biological impacts. The elemental form of mercury exhibits primarily inhalation toxicity, causing impairment of the central nervous system, which in extreme form is known as the "mad hatter" syndrome. Mercury salts (i.e., inorganic forms of mercury) exhibit toxicity primarily through ingestion and dermal absorption, with symptoms exhibited in the gastrointestinal tract, kidneys, and liver. Finally, methylmercury, exposure to which happens mainly from ingestion of contaminated fish, may, in addition to impairment of the central nervous system cause severe birth defects. Epidemiological studies are presently in progress to develop reference doses for mercury exposure through ingestion but results are not yet available. Consequently, uncertainties in toxicological factors were not included in our uncertainty analysis.

M.4.5 Case Study

The case study presented here, corresponds to the mercury emissions of Site A. A deterministic health risk assessment associated with the emissions of this site was presented earlier in the "TRUE Case Studies" section of this chapter. The health risk estimates subject to uncertainty analysis correspond to the subregion of maximum mercury hazard index. The deterministic results for this subregion are summarized below:

- The mercury multimedia hazard index was estimated to be 0.28 (i.e., an estimated dose below the ingestion reference dose).
- Ingestion was the major contributing pathway to the total mercury hazard index.
- Fish was the major contributor to the mercury ingestion dose.

Public water and fish supply for this subregion were considered to originate from two different segments of a nearby creek.

Due to the poor quantitative characterization of certain phenomena in the mercury cycle, a number of assumptions were made prior to the performance of the uncertainty analysis and were, therefore, excluded from probabilistic characterization in the uncertainty analysis. The following assumptions were based on interpretation of available quantitative information:

- All mercury is emitted from the power plant as either Hg (0) (g) or Hg (II).
- No organic forms of mercury are being created in the atmosphere.
- Transfer of Hg (II) from the gaseous to the particulate phase is well-described by assuming equilibrium with the liquid phase of aerosols.
- Scavenging of Hg (0) (g) is considered negligible due to the very low solubility of that form of mercury.
- Scavenging of gaseous inorganic mercury, Hg (II) (g), occurs in a manner similar to that of other gases with comparable solubility.
- The particulate size distribution in the plume remains the same as that at the stack exit. Therefore, scavenging of particulate Hg (II) can be estimated based on that distribution.
- Since the only chemical form of mercury being deposited in significant quantities is Hg (II), all mercury in surface soil is considered to be of that form. No chemical transformations in soil are considered.
- Only inorganic mercury is taken up by plants.
- From 1 to 10% of the total inorganic mercury deposited and transferred into surface water bodies undergoes methylation.

M.4.6 Sensitivity Analysis

Sensitivity analysis was performed according to the methodology described above under the section on uncertainty analysis. The results of this sensitivity analysis were used in combination with uncertainty estimates for parameters, to select the critical parameters and construct response surfaces for the different components of the TRUE model.

M.4.7 Probability Distribution Selection

Selection of appropriate probability distributions for the identified influential parameters was performed on the basis of available statistical data, literature value ranges, and engineering judgment. The selected probability distributions, and the information used as the basis of their derivation are presented in Table M-2.

Table M-2
Uncertainty Analysis of Mercury Health Risk Estimates Probability Distribution Selection

Index	Parameter Symbol and Units	Point(1) Value	Description	Probability Distribution		Comments
				Type	Parameters	
1	Qe (g/s)	1.3×10^{-2}	Emission rate of plant	Point(2)	1.3×10^{-2}	No distribution assigned due to lack of information. Assumed same value as in deterministic risk assessment.
2	Vs (m/s)	22.0	Stack gas exit velocity	Uniform	a = 17.6 b = 26.4	Type assumed. Range based on personal judgement.
3	Ts (K)	359	Stack gas exit temperature	Uniform	a = 354 b = 364	Type assumed. Range based on personal judgement.
4	Ta (K)	287	Ambient temperature	Uniform	a = 283 b = 293	Type assumed. Range based on personal judgement.
5	α (—)	0.71	Fraction of Hg in plume as Hg (0)	Uniform	a = 0.46 b = 0.71	Range obtained from measurements using two different methods (MESA and EPA 29).
6	β (—)	0	Fraction of Hg (II) in plume in particulate phase	Uniform	a = 0 b = 0.03	Distribution assigned based on data and model simulations.
7	Vd2p (m/s)	0.01	Deposition velocity for Hg (II) particulates	Lognormal	$\mu' = -7.78$ $\sigma' = 0.74$	Type and parameters estimated using the measured particulate size distribution and available literature relationships (Zannetti, 1990).
8	Vd0 (m/s)	6×10^{-5}	Deposition velocity for Hg (0)	Uniform	a = 0 $b = 1.2 \times 10^{-3}$	Kim et al., 1994

Table M-2
Uncertainty Analysis of Mercury Health Risk Estimates Probability Distribution Selection (Continued)

Index	Parameter Symbol and Units	Point(1) Value	Description	Probability Distribution		Comments
				Type	Parameters	
9	Vd2g (m/s)	5.4×10^{-3}	Deposition velocity for HgII gas	Lognormal	$\mu = -5.2$ $\sigma' = 1.1$	Range obtained from Seinfeld, 1986 assuming similarity with HNO3
10	Λ2g (s-1)	2.1×10^{-4}	Scavenging coefficient for gaseous Hg (II)	Uniform	$a = 1.2 \times 10^{-4}$ $b = 3.7 \times 10^{-4}$	The simplest type of distribution assumed due to lack of information. Range estimated using literature reported values on the basis of compound solubility (Seinfeld, 1986)
11	Λ 2p (s-1)	4.3×10^{-4}	Scavenging coefficient for particulate Hg (II)	Lognormal	$\mu' = -7.2$ $\sigma' = 0.67$	Type and parameters estimated using the measured particle size distribution and precipitation intensity with an empirical relationship provided in the literature (Jindal and Heinold, 1991)
12	ds (cm)	5.0	Surface soil depth	Lognormal	$\mu' = 1.50$ $\sigma' = 0.75$	Type assumed. Parameters calculated based on a 0-20 cm range.
13	ED (yr)	70	Exposure duration	Lognormal	$\mu' = 2.24$ $\sigma' = 0.58$	Type and parameters suggested by EPA data (U.S. EPA, 1989)
14	ORf (—)	0.98	Fraction of deposited mercury attributed to overland runoff	Normal	$\mu = 0.9$ $\sigma = 0.05$	Type assumed. Parameters calculated based on assumed range.
15	qw,d (m3/s)	0.29	Discharge in portion of river used for drinking water	Normal	$\mu = 0.29$ $\sigma = 0.03$	Type assumed. Parameters calculated based on assumed range.
16	qw,f (m3/s)	1.0	Discharge in portion of river used for fishing	Normal	$\mu = 1.0$ $\sigma = 0.1$	Type assumed. Parameters calculated based on assumed range.

Table M-2
Uncertainty Analysis of Mercury Health Risk Estimates Probability Distribution Selection (Continued)

Index	Parameter Symbol and Units	Point(1) Value	Description	Probability Distribution		Comments
				Type	Parameters	
17	g (—)	0.0	Fraction of Hg in water in inorganic form	Normal	$\mu = 0.945$ $\sigma = 0.0225$	Type assumed. Parameters calculated based on assumed range (Tetra Tech, 1991).
18	BCFp (—)	0.90	Soil-to-plant bioconcentration factor for inorganic Hg	Lognormal	$\mu' = -0.11$ $\sigma' = 1.4$	Type assumed. Parameters estimated based on reported values (Baes, 1993) and information acquired through personal communication (Baes, 1993).
19	BCFf,1 (—)	0.0	Water-to-fish bioconcentration factor for inorganic Hg	Lognormal	$\mu' = 8.45$ $\sigma' = 0.85$	Type and parameters estimated using reported experimental values (U.S. EPA, 1984).
20	BCFf,2 (—)	33,000	Water-to-fish bioconcentration factor for organic Hg	Lognormal	$\mu' = 10.25$ $\sigma' = 1.0$	Type and parameters estimated using reported experimental values (U.S. EPA, 1984) and model estimates (Tetra Tech, 1991).
21	IR (m3/d)	20.0	Inhalation Rate	Triangular	$a = 14$ $\mu = 22$ $b = 30$	Minimum, maximum, and most likely values provided by U.S. EPA (1989).
22	BW (kg)	70.0	Body weight	Normal	$\mu = 71.5$ $\sigma = 17.0$	Type and parameters based on EPA data (U.S. EPA, 1989).
23	INRw (l/day)	2.0	Water ingestion rate	Lognormal	$\mu' = 0.65$ $\sigma' = 0.39$	Type and parameters based on reported data (U.S. EPA, 1984, Ershow and Cantor).

Table M-2
Uncertainty Analysis of Mercury Health Risk Estimates Probability Distribution Selection (Continued)

Index	Parameter Symbol and Units	Point(1) Value	Description	Probability Distribution		Comments
				Type	Parameters	
24	INRp (kg/d)	0.078	Plant ingestion rate	Lognormal	$\mu' = -2.58$ $\sigma' = 0.61$	Type and parameters based on homegrown percentage consumption data provided by (U.S. EPA, 1989).
25	INRf (g/d)	37.0	Fish ingestion rate	Lognormal	$\mu' = 0.92$ $\sigma' = 1.99$	Distribution and parameters extracted from the literature (Anderson et al., 1992).
26	RfD1 (mg/kg-d)	8.6×10^{-5}	Mercury inhalation reference dose	Point	8.6×10^{-5}	No distribution assigned. Assumed same value as in deterministic risk assessment.
27	RfD21 (mg/kg-d)	3.0×10^{-4}	Inorganic mercury ingestion reference dose	Point	3.0×10^{-4}	No distribution assigned. Assumed same value as in deterministic risk assessment.
28	RfD22 (mg/kg-d)	3.0×10^{-4}	Organic mercury ingestion reference dose	Point	3.0×10^{-4}	No distribution assigned. Assumed same value as in deterministic risk assessment.

Explanations: The above-listed probability distribution types are defined as follows:

- Uniform[a, b], where: a = minimum value, b = maximum value
- Triangular[a, μ, b], where: a = minimum value, μ = most likely value, b = maximum value
- Normal (μ,σ), where: μ = distribution mean, σ = distribution standard deviation,
- Lognormal(μ', σ'), where: μ' = mean of underlying normal distribution, σ' = standard deviation of underlying normal distribution

Note: All lognormal distributions are based on natural logarithms.

(1) point value indicates value of parameter that was used in the deterministic risk assessment.

(2) Uncertainty due to emission variation was not considered in the analysis due to lack of reliable information needed for the development of a probability distribution.

M.4.8 Latin Hypercube Analysis

The derived response surfaces were combined into a parameterized version of the multimedia health risk assessment model, which was coupled to the software package Crystal Ball (Decisioneering Inc., 1993) for the performance of Latin Hypercube synthetic simulations. Ten thousand iterations of the simplified model were performed and the results were used for the construction of a probability distribution for the mercury hazard index. The value of the hazard index calculated in the deterministic risk assessment (0.28) was estimated to be beyond the 95th percentile of the derived probability distribution. The statistical parameters of this distribution are summarized below:

- Mean (expected value) μ = 2.24×10^{-2} (8% of the deterministic value)
- Standard Deviation, σ = 9.2×10^{-2}
- Skewness = 23.7 (positively skewed—right tail)
- Percentiles: 5% $F_{0.05}$ = 3.94×10^{-4}

  25% $F_{0.25}$ = 1.22×10^{-3}

  Median 50% $F_{0.5}$ = 3.80×10^{-3}

  75% $F_{0.75}$ = 1.40×10^{-2}

  95% $F_{0.95}$ = 9.01×10^{-2}

The derived probability density plot for the mercury hazard index is presented in Figure M-2.

Sensitivity analysis indicated that the parameters which had the most important effect on the variability of the results were the fish ingestion rate (75% of the variance) and the water-to-fish bioconcentration factor for organic mercury (6%).

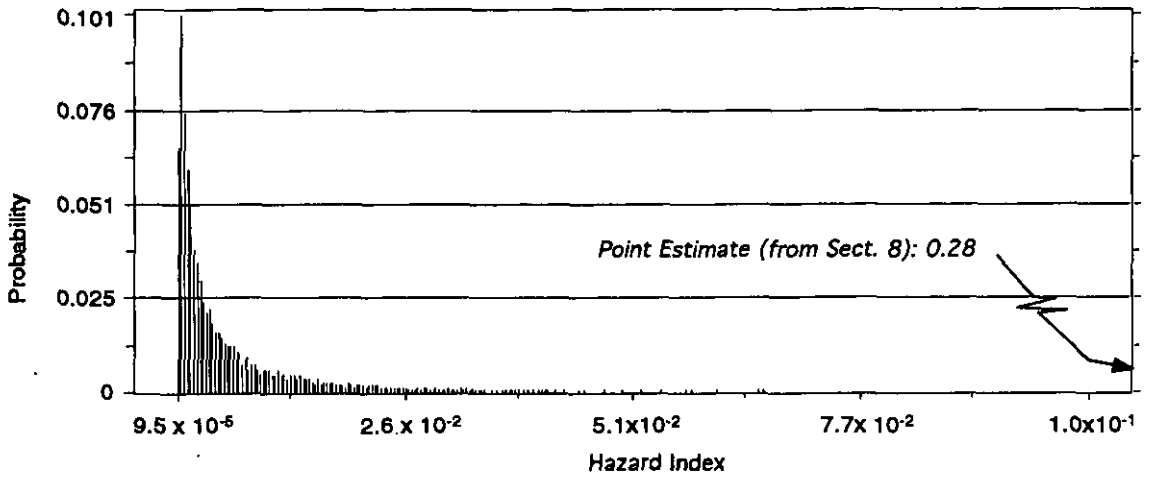

Figure M-2.
Distribution of Hazard Index for Mercury, Case Study Site A

M.5 References

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APPENDIX N
EXPOSURE TO MERCURY VIA FISH CONSUMPTION

N.1 Introduction

Mercury is released into the atmosphere by natural and man-made sources. Natural sources of mercury include the evaporation of mercury from the ocean surface; degassing of mineral mercury from the earth's crust, oceans and volcanic eruptions and microbial decomposition of surface soils and sediments with subsequent off-gassing. Man-made sources of elemental and inorganic mercury compounds have historically been derived from the manufacture of products for science laboratories and agriculture. Historically, the largest consumer of mercury has been the chlor-alkali industry (ICF, 1993). However, the use of mercury has been significantly reduced over the past decades but, elevated levels of mercury found in food, specifically seafood. It is currently believed that fish and shellfish consumption are the major sources of methylmercury exposure to the general population. This concept has caused great public concern and prompted several interest groups including the Food and Drug Administration (FDA), Environmental Protection Agency (EPA), National Marine Fisheries Service (NMFS) and the World Health Organization (WHO) to evaluate and monitor the levels of mercury in the environment and seafood products.

The purpose of this paper is to review the movement of mercury in the environment; to discuss the mercury levels found in seafood; and to estimate human exposure to mercury via consumption of fish and shellfish.

N.2 Mercury In the Environment

Mercury occurs in three general forms, elemental, inorganic and organic. The form or species dictates mercury's toxicity and behavior in the environment. Historically, organic mercury, particularly methylmercury, has caused the greatest concern with regards to human health and aquatic life.

Elemental mercury Hg (0) is also referred to as mercury vapor when in air, and metallic mercury, in the liquid form. Liquid mercury is used for gasoline manometers and other scientific equipment such as thermometers and barometers (Von Burg and Greenwood 1991). Inorganic mercury compounds such as mercuric chloride ($HgCl_2$) and mercurous chloride (Hg_2Cl_2) are formed from ionic mercury Hg (II) and Hg (I), are highly soluble

and often found in acidic and neutral environments (Von Burg and Greenwood 1991). As with elemental mercury, ionic mercury has many industrial uses including preparations of dental amalgams, catalysts, preservatives and explosives. Organic forms of Hg have also been used agriculturally as seed and cereal protectants and as fungicides. Of all forms of organic mercury, methylmercury (MeHg) is of the greatest concern due to its toxicity and potential to bioaccumulate (ICF 1993).

Organic mercury compounds are formed from inorganic mercury by both chemical and bacterial processes and all forms of mercury entering the aquatic environment can be converted to organic methylmercury (Tollefson and Cordle, 1986). These compounds include monomethylmercury (CH_3Hg) and dimethylmercury ($(CH_3)_2Hg$). Methylation can take place under the appropriate conditions. For example, methylation may occur by the interaction of ultraviolet light with surface waters (Summers and Silver 1978) or bacteria in lake sediments can methylate mercury from deposited inorganic mercury (Von Burg and Greenwood 1991). This process seems to occur to a greater extent under anaerobic conditions (without oxygen) rather than aerobic conditions (with oxygen).

Bacteria also degrade methylmercury into mercury and methane (Tonomura et al., 1968) by splitting the carbon-mercury bond. The released Hg^{+2} is then reduced to elemental mercury (Von Burg and Greenwood 1991). While methylation appears to be a key step in bio-geochemical cycling, demethylation has been suggested to maintain environmental methylmercury to a minimum (Spangler et al., 1973). The competing reactions of methylation and demethylation result in steady-state concentrations of ecological methylmercury (D'Itri 1991).

N.3 Fate and Transport

Air: The environmental cycle of mercury is characterized by natural and man-made releases into the atmosphere, atmospheric transport, wet and dry deposition of mercury and the adsorption of mercury to soil and sediments (ICF 1993). Mercury in the atmosphere is mostly in the form of elemental mercury vapor (Linqvist 1991) and generally has a long residence time. Inorganic mercury attached to particles or in the form of particles of $HgCl_2$ and HgO can undergo sedimentation (dry deposition), be collected by cloud droplets and dissolved into rain or snow (wet deposition) which deposits the mercury back onto the ground (ICF 1993). Organic mercury (monomethyl and dimethylmercury) has also been found to adhere to particulates in the atmosphere (Johnson and Braman 1974).

Soil: Mercury has a strong tendency to adsorb to soil or sediment with a strong affinity not only to organic materials but also to clays (Bodek et al., 1988, Porvari et al., 1992). Soil profile studies of mercury concentrations generally demonstrate that the highest Hg concentrations are found at the surface soil horizons with diminishing concentrations as depth increases (Adriano, 1986). The retention of Hg in soil is not only due to adsorption to organic and inorganic materials by ionic or covalent bonds but also due to the forma-

tion of carbonates, phosphates and sulfides with low water solubilities. As a result, although the leaching of Hg from municipal landfills has been reported (Fuller, 1978), the Hg content of soil leachates is generally low (EPA 1985).

In general, Hg is unstable in the environment because it is subject to a number of biological, chemical and photochemical reactions that yield elemental Hg which can volatilize and this pathway may be the dominant pathway for Hg loss from soil; more important than leaching or plant uptake (Gilmour and Miller, 1973).

Water: In water, deposited inorganic mercury can undergo oxidation-reduction reactions. Hg (0) can be oxidized to Hg (II) in the same manner as Hg (0) is thought to react with rain droplets in the atmosphere (EPA 1985). The ionic form of Hg can then form inorganic and organic complexes. Of considerable concern is the ability of the Hg (II) ion to become methylated to form the more toxic MeHg. This transformation is accomplished primarily by biological processes of anaerobic bacteria (NRCC, 1979; Beijer and Jernelov, 1979). The methylated Hg then becomes available for uptake by organisms and bioaccumulates. This results in the inclusion of Hg into the food chain where it is biomagnified through trophic levels, primarily fish, and presents a potential source of toxicity for humans.

The extent of MeHg formation depends primarily on the total amount of Hg available and environmental conditions. For example, although anaerobic conditions are more conducive to the methylation process, they also limit the methylation of Hg because anaerobic conditions also favor the formation of HgS; a relatively water insoluble compound, resistant to methylation. On the other hand, under aerobic conditions, sulfite converts to sulfate. This along with other cationic exchanges release Hg from a bound or insoluble state and make it available for methylation. Such mercury releases are seen with certain flood control practices. In South Florida, dredge and fill operations and lake drawdowns have mobilized the Hg in the sediment in this fashion and make it available for aquatic receptors (ICF, 1993).

N.4 Mercury in Fish

Methylmercury is absorbed by fish through their gills or by eating smaller organisms such as phytoplankton and zooplankton (EPRI 1991). Bloom (1992), after analyzing the methylmercury content in edible muscle of 229 freshwater and marine fish samples concluded that virtually all (99%) of the mercury found in fish muscle is MeHg. (Investigators continue to report inorganic Hg content in fish muscle in the range of 10-20%, calling into question the value of 100% methylmercury.) However, most other investigator believe the MeHg content in relations to the total Hg content is more on the order of 80–90%. Regardless of such details, it is generally believed that most of the Hg content in fish is in the form of MeHg (Von Burg and Greenwood, 1991). For the purposes of the present discussion, this assumption will be retained.

The excretion and regulation of MeHg in fish is slow (Evans et al., 1993) with a half-life on the order of approximately 2 years. This long half-life, coupled with a continual exposure results in a steady accumulation that never reaches steady state in the fish flesh during its life span (Beijer and Jernelov, 1978). As a result, the mercury concentration in fish is generally dependent upon the size, predation level and ecological niche and this mercury accumulation can eventually to such levels that it becomes lethal to the fish (Von Burg and Greenwood 1991).

Due to the accumulation and presence of MeHg in fish there is a need to characterize the levels of methylmercury in fish, fishery products and the patterns of fish consumption. In response to this need, several research efforts have attempted to assess the distribution of Hg levels in fish.

N.4.1 Marine Fish and Shellfish

Some early efforts to assess the distribution of Hg in fish were reported in the 1970s (Bligh and Armstrong, 1971; Doi and Ui, 1973) but a more extensive effort was reported by the National Marine Fisheries Service (NMFS) as part of a Microconstituents Program (NMFS, 1978). This program was a survey of trace elements in fin fish, mollusks and crustaceans from 198 sites around the coastal United States. Total concentrations for 15 elements including mercury were determined in the 204 aquatic species which represents approximately 94% of the U.S. commercial and sportfish catch volume. Mean mercury levels were below 0.3 ppm with an overall average weighted concentration of 0.11 μg Hg/g. Shellfish such as clams, crabs, oysters, scallops and shrimp averaged only 0.05 ppm. However, 31 species were found to exceed the FDA action level of 0.5 ppm but these species account for only 2% of the intended U.S. fish consumption. The results of this survey are presented in Table N-1.

Table N-1.
Summary of Mercury in Marine Fish

Species	Concentration (ppm)	Location
Starry Flounder	<0.002-0.156	Puget Sound, WA [1]
Rockfish	<0.002-0.079	
Sablefish	0.003-0.022	
Rock Sole	<0.002-0.009	
Walleye Pollock	<0.001-0.073	
Pacific Cod	0.003-0.090	
Pacific Hake	0.003-0.009	
Tomcod	0.003-0.008	
Albacore tuna	0.11-0.244	Pacific and Atlantic Ocean [2]
Bigeye tuna	0.44	

Table N-1.
Summary of Mercury in Marine Fish (Continued)

Species	Concentration (ppm)	Location
Cod	0.02-0.23	Atlantic Coast [3]
Flounder	0.07-0.17	
Haddock	0.07-0.10	
Herring	0.02-0.09	
Tuna	0.33-0.86	
156 Species including:		U.S. Coastal waters along natural and traditional boundaries [4]:
Bass	0.1-1.0	North and South Atlantic
Bonito	<0.1-2.0	California and Gulf of Mexico
Catfish	0.1-2.0	N. Atlantic and Gulf of Mexico
Cod	<0.1-0.2	N. Atlantic, Northwest and Alaska
Eel	<0.1-0.3	N. Atlantic
Flounder	<0.1-0.3	N. and S. Atlantic and Gulf of Mexico
Halibut	<0.1-0.4	Northwest and Alaska
Herring	<0.1-0.3	N. Atlantic, Northwest and Alaska
Mackerel	<0.1-3.0	N. and S. Atlantic, California and Gulf of Mexico
Mako	1.0-3.0	
Mullet	<0.1	S. Atlantic, Gulf of Mexico
Perch	<0.1-0.7	Hawaii
Pollock	<0.1-0.2	California
Rockfish	0.1-0.3	N. Atlantic, Northwest and Alaska
Sablefish	0.2-0.6	California and Northwest
Salmon	<0.1-0.2	California, Northwest and Alaska
Snapper	0.1-0.6	Alaska, Northwest and California
Sole	<0.1-0.3	S. Atlantic, Gulf of Mexico and Hawaii
Trout	<0.1-0.4	California and Northwest
Tuna	0.1-0.4	N. Atlantic, Hawaii and Gulf of Mexico

More recent information from NMFS and other studies (NOAA, 1987; San Diego Bay, 1990; Bloom, 1992) indicates that the mercury levels in the 1978 report have not changed significantly (Cramer, 1994). Analysis of 245 samples of canned tuna fish, reported as the most frequently consumed fish item, found a mean MeHg level of 0.17 ppm which is very similar to an earlier study on canned tuna that found 0.24 ppm total mercury (Yess, 1993). Essentially the range of Hg concentrations in comparable pelagic marine fish are very similar irrespective of the geography of the catch. Higher Hg values are reported for fish

at higher trophic levels such as tuna, sharks and billfish. For example, methylmercury levels of 1.3–1.5 ppm are routinely reported for sharks with some levels as high as 4.2 ppm (Cramer, 1994).

N.4.2 Freshwater Fish

Relative to marine fish, Hg levels reported in freshwater fish are found to be highly variable. The wide range of values result from a number of differences in the physical and chemical characteristics of the water body such as dissolved organic content (DOC), pH and proximity of natural or anthropogenic sources of Hg. Fish size, age, trophic level and ecological niche are also known to be major sources of variability (Lipfert et al. 1994). To demonstrate the variability of mercury in freshwater fish, examples of five independent studies conducted throughout the U.S. are briefly presented below and are summarized in Table N-2.

Table N-2.
Summary of Mercury Data on Freshwater Fish

Species	Concentration (ppm)	Location
Largemouth Bass	0.087-0.364	Midwestern Lakes including a contaminated site [7]
Yellow Perch	0.160-0.260	
Northern Pike	0.300-1.444	[4]
White Sucker	0.090-0.340	
Largemouth Bass	0.07-2.37	Florida lakes and streams [8]
Lake Trout	0.13-1.11	Eagle Lake and St. Froid Lake of Northern Maine [9]
Brook Trout	0.08-0.58	
Turbot	0.35-1.29	
Rainbow Smelt	0.28-0.59	
Lake Trout	0.10-0.23	Cliff Lake of Northern Maine
Brook Trout	0.12-0.21	
Yellow perch	0.01-2.37	Upper Peninsula of Michigan [10]
Northern pike	0.07-1.64	
White sucker	0.01-0.59	
Largemouth bass	0.06-1.00	
Walleye	0.11-0.42	
Smallmouth bass	0.02-0.39	

Table N-2.
Summary of Mercury Data on Freshwater Fish (Continued)

Species	Concentration (ppm)	Location
Walleye	0.58 ± 0.26	Great Lakes:
Perch	0.24 ± 0.14	Lake Erie [11]
White Bass	0.49 ± 0.31	
Smallmouth Bass	0.51 ± 0.19	
Perch	0.88 ± 0.75	Lake St. Claire
All Others	0.48 ± 0.32	
All Types	0.11 ± 0.11	Lake Michigan
All Types	0.30 ± 0.30	Lake Ontario
All Types	0.19 ± 0.11	Lake Huron
All Types	0.13 ± 0.11	Lake Superior
Northern Pike	0.18-1.52	Northern Minnesota Lakes [12]
Walleye	0.13-1.75*	

* Value calculated from other fish species.

1. Bloom, N. S. 1992: This investigator analyzed marine fish and several species of freshwater fish for total, monomethyl, and dimethylmercury in edible muscle by cold vapor atomic fluorescence spectrometry (CVAFS). Although emphasis was placed on the fraction of mercury in the methylated form instead of particular concentrations, the total mercury concentrations reported for several freshwater species were: bass (0.087–0.364 µg/Kg), perch (0.160–0.260 µg/Kg), northern pike (0.300–1.444 µg/Kg) and white sucker (0.0900–0.340 µg/Kg).

2. Florida Department of Environmental Regulation, 1990: In this study, mercury levels of 619 largemouth bass from 96 Florida stations (lakes, streams and canals) were analyzed. The period of investigation was from 1983 to 1989. Of the stations investigated, some areas were known to be polluted. Other areas considered to be "pristine" were analyzed to determine background concentrations. This statewide sampling program led the Florida Department of Health and Rehabilitative Services to issue several fish consumption advisories. Of the 22 lakes analyzed, the mercury levels found in the largemouth bass samples were 0.07, 0.44, 0.77 and 0.85 ppm for the 5th, 50th, 90th and 95th percentile. Stream fish levels were 0.22 ppm, 0.80 ppm, 1.41 ppm and 2.37 ppm for the 5th, 50th, 90th and 95th percentiles respectively. The mercury levels in seventy percent of the fish collected and analyzed exceeded 0.5 µg/kg and eight percent of the streams in south Florida had concentrations exceeding 1.5 µg/kg. The spatial distribution of mercury measured in Florida's bass tissue were also found to be elevated (0.5-1.5 µg/kg) in many of the pristine areas while very low mercury levels were found in highly polluted lakes suggesting that

the mercury concentrations are inversely proportional to the overall lake quality. However, as pH gets lower, mercury level in fish tissue can increase at a given trophic level and the mercury in the sediments becomes more available.

3. *Akielaszek and Haines, 1981:* Mercury levels in muscle tissue of brook trout and lake trout of three northern Maine lakes were examined to evaluate the effects of the trophic structure on mercury concentrations of top carnivores and the effects of the trophic level on mercury concentration. Fish mercury levels from Eagle Lake and St. Froid Lake, in the same drainage basin are compared with each other and in turn compared to fish from Cliff Lake of a different basin. Total mercury in fish muscle (wet weight) was found to range from 0.08 to 2.17 μg/Kg. Mercury concentrations above 0.50 μg/Kg were detected in 28% of the fish examined and Hg levels in fish from all three lakes were found to be higher than expected and the trout from Eagle and St. Froid Lakes had mercury levels similar to lakes near sources of Hg contamination in Maine (Table N-2). The authors were unable to determine if fish mercury levels had always been elevated in these lakes, but believe that the levels are atypical for remote, wilderness lakes. The authors suggest that several factors may be responsible for the elevated mercury levels: a). rainbow smelt which can accumulate substantial amounts of mercury are not present in Cliff Lake and rainbow smelt serve as an important food source for fish in Eagle and St. Froid Lakes; b). conditions for methylation is enhanced in the oligotrophic lakes because surface sediments were found to contain more Hg than deep sediments, suggesting an increase of mercury input in the lakes, and thus increased mercury availability for fish uptake; and c). acid precipitation may have reduced the buffering capacity of the lakes resulting in a lower pH which enhances methylation of mercury.

4. *U.S. EPA, 1990:* Forty-nine drainage and seepage lakes in the upper Michigan peninsula were sampled to explore the relationship between physical and chemical characteristics of the lakes and the mercury levels in fish. This study was conducted in conjunction with Phase II of the U.S. Environmental Protection Agency (US EPA) Eastern Lake Survey also known as ELS-II. Samples from 49 lakes were obtained from June 8 to August 30, 1987, and fish species from 37 lakes were analyzed for factors affecting their Hg concentrations. Correlation and simple regression analysis were used to determine length, weight and age of yellow perch, northern pike, white sucker and largemouth bass to their mercury levels. Subsequently, multiple stepwise regressions were conducted between mercury concentrations, lake chemistry and physical characteristics.

The results showed that there was a difference in mercury accumulation between drainage and seepage lakes. Although the Hg concentrations of yellow perch of ages 2 and 5 years were similar in both types of lakes, the drainage lakes had higher mercury levels in older perch. However, statistically significant Hg levels in fish from both lakes were only apparent in yellow perch of ages 1 and 7 year. In general, the seepage lakes had a wider range of Hg levels in fish while drainage lakes tended to have higher levels of Hg in the yellow perch population.

5. Sorensen et al., 1990: These authors created a comprehensive multimedia database for fish consumption advisories. Mercury concentrations in precipitation, lake water, sediment, zooplankton, and fish were measured and analyzed in relation to extensive watershed and lake chemistry data of 80 northern Minnesota Lakes. Fish mercury residue of northern pike and walleye in 65 lakes were measured at a standardized length and weight of 55 cm and 1.0 kg respectively. The Hg concentration was computed from the log-log regression equation for each lake. The average Hg level of northern pike at a standardized length and weight for these lakes were found to be 0.45 ppm with a range of 0.14–1.5 ppm. The highest correlation of mercury level to fish length were at lengths 55 and 39 cm for northern pike and walleye respectively.

N.5 Fish and Shellfish Consumption

Not only is there a good deal of variability in the levels of Hg in the fish but it can also be anticipated that there will be a good deal of variability in the species of fish consumed as well as total amount of seafood consumed by a different sociodemographic populations. The simplest approach to this problem estimates seafood consumption on a per capita basis based on the total fish products available for consumption.

N.5.1 Estimation by Fish production

The National Oceanic and Atmospheric Administration, NOAA (1993) reported the United States fish landings for 1992 to be approximately 9,637,303,000 pounds. Of the total 1992 annual catch, approximately 86% fall into eight major fish categories. The remaining 14% is assigned to a numerous assortment of fish (Table N-3). If the landing weight of shellfish were reduced by a factor of 0.49 (Landolt et al., 1987) due to the presence of the non edible carapace and other non-edible anatomical parts, the relative weight of shell fish is reduced to 716,929,000 pounds. Thus, the descending order of relative landing weight is Pollock > Shellfish > Salmon > Flounder > Cod > Tuna > Herring. According to NOAA, on the basis of landing weight, tuna fish is not in great demand compared to the other choices which contradicts the estimations of Javitz (1980). Javitz based his figures on the 1973-74 NPD Research data which indicated that on a weighted basis, 94% of the U.S. population indicated tuna fish to be in the highest demand (Cramer, 1994).

Table N-3.
Mercury in Major Fish Categories Contributing to the Total Fish Landings

Species	Estimated amount of the 1991 fish landings [13] (thousands of pounds)	Study	Mercury Concentrations ppm Average	No of samples
Salmon	715,828	[14] Salmon, Chinook (king)	<0.1	95
		Salmon, chum (keta)	<0.1-0.2	60
		Salmon, coho (silver)	<0.1	76
		Salmon, pink	<0.1	32
		Salmon, sockeye	<0.1	64
Flounder	645,829	[15][16]Flounder, fourspot	0.1-0.2	71
		Flounder, gulf	<0.1-0.2	41
		Flounder, southern	<0.1	44
		Flounder, summer	0.1-0.2	44
		Flounder, windowpane	0.1-0.2	18
		Flounder, winter	<0.1-0.3	125
		Flounder, witch	0.1-0.2	71
		Flounder, yellowtail	<0.1	90
			<0.1	88
		[15]Starry Flounder	<0.002	195
			0.156	200
			<0.002	199
			<0.002	198
Cod	611,810	[16]Cod, Atlantic	<0.1-0.2	150
		Cod, Pacific	<0.1-0.2	74
Herring	291,162	[16]Herring, Atlantic	<0.1-0.2	95
		Herring, Pacific	<0.1-0.3	1
		Herring, round	<0.1	44
				50
Tuna	577,382	[16]Tuna, albacore	0.1-0.2	44
		Tuna, bigeye	0.3-0.4	60
		Tuna, blackfin	0.3-0.4	1
		Tuna, bluefin	0.1-0.3	12
		Tuna, skipjack	0.1-0.2	78
		Tuna, yellowfin	0.1-0.3	106

Table N-3.
Mercury in Major Fish Categories Contributing to the Total Fish Landings (Continued)

Species	Estimated amount of the 1991 fish landings [13] (thousands of pounds)	Study	Mercury Concentrations ppm Average	No of samples
Pollock	2,967,973	[16]Pollock	<0.1-0.2	105
		Pollock, walleye (Alaska)	<0.1-0.2	50
		[17]Walleye Pollock	0.073	270
			0.008	266
			0.003	267
			0.006	269
			<0.001	268
			—	232
			0.006	231
Menhaden*	1,705,026	[16]Menhaden, Atlantic	<0.1-0.2	209
Total landings for the above fish categories	8,387,752			
Annual 1992 catch	10,235,020			
Catch intended for human consumption	7,618,000			

* Not generally used for human consumption
** Adjusted weight for non-edible parts

Estimates of fish consumption based on landing weight have been conducted by the USDA (1991) and is summarized in Table N-4. This summary table indicates that there has been a general upward trend in fish and shellfish consumption over the period of 1970–1991 amounting to approximately 4 grams per day. Of this total consumption, the most popular seafood species categories that represent almost 80% of the seafood consumed are presented in Table N-5.

Table N-4.
Fish and Shellfish per capita Consumption

Year	Pounds	grams/day
1970	11.7	14.54
1971	11.5	14.29
1972	12.5	15.5
1973	12.7	15.8
1974	12.1	15.0
1975	12.1	15.0
1976	12.9	16.0
1977	12.6	15.7
1978	13.4	16.7
1979	13.0	16.2
1980	12.4	15.4
1981	12.6	15.7
1982	12.4	15.4
1983	13.3	16.5
1984	14.1	17.5
1985	15.0	18.6
1096	15.4	19.1
1987	16.1	20.0
1988	15.1	18.8
1989	15.6	19.4
1990	15.0	18.6
1991	14.8	18.4

Source: USDA, Food Consumption, Prices, and Expenditures 1970-1990

Table N-5.
Most Popular Seafood Species Consumed per capita for 1992

Species	Average daily intake per capita (g/p-d)
Tuna	4.4
Shrimp	3.1
Alaska Pollock	1.5
Salmon	1.4
Cod	1.3
Catfish	1.1
Clams	0.8
Flatfish	0.6
Crabs	0.4
Scallops	0.3
Other	3.6
Total	18.5
(Modified from Cramer, 1994)	

Table N-6 presents a summary of these various attempts to estimate the seafood consumption of the U.S. general population, recreational anglers and subsistence anglers. As a result, a value of 18.5 grams per day (g/d) per capita has been estimated by the USDA. A value of 21 g/d has been estimated from the 1986 National Marine Fisheries Service (NMFS) data while 16 g/d is the estimated annual per capita fish consumption in Canada (CCAC, 1977). However, such values reflect consumption primarily of purchased fish and does not include recreational fish consumed. The New Jersey Department of Environmental Protection and Energy has estimated that the average consumption for an individual is 24.3 g/d including one 8 ounce meal of locally caught freshwater fish per week (excluding the winter months of December, January and February). The National Academy of Science estimated a 20 g/d per capita fish consumption based on the assumption that the average person consumes approximately 1 fish meal per week.

Table N-6.
Summary of Fish Consumption Estimate for the United States

Consumption Group	Consumption (g/person/day)		Source
	Average	RME	
General Population	21	—	National Marine Fishery Service, 1986
	24.3	—	New Jersey Dept. of Env. Health, 1992
	18.5	—	Cramer, 1994
	20	—	[17]National Academy of Sciences, 1978; UN Food and Agricultural Organization, 1977
	14.3	41.7	Javitz, 1980
	Ave: 19.6		
Recreational Anglers	36.9	224.8	Puffer et al. 1981
	23	54	Pierce et al. 1981
	31.2	—	Hickey, 1990
	11	—	Landlot et al. 1987
	30	140	[18]US EPA Fish Consumption Guidance, 1989
	Ave: 26.4	Ave: 139.6	
Subsistence Anglers	396[a]	—	Food and Drug Administration (FDA), 1990
	134[b]	—	
	162[c]	—	
	69[d]	—	
	16.2[e]	116	Richardson et al. 1993
	Ave: 190.2[*]		

RME Reasonable maximum exposure

— Not calculated.

a Fish and shellfish consumption of the most isolated population in Kodiak Island, Alaska.

b Fish and shellfish consumption of the less isolated population in Kodiak Island, Alaska.

c This survey was conducted for the village of Chenega Bay on Evans Island, Prince William Sound (Alaska Department of Fish and Game, Division of Subsistence between 1984 and 1986). Chenega Bay is accessible only by air and water and traditional fishing and hunting are used to obtain a major portion of their food.

d Fish consumption of subsistence angler and their families in the contiguous United States.

e The geometric mean fish consumption rate of Ontario Amerindians.

* The average does not include the value reported by Richardson et al. (1993).

There are a number of difficulties associated with accepting this approach for estimating either fish consumption or mercury exposure. On the one hand, such estimates may overestimate consumption because they presume consumption of whole fish weights, and include fish not consumed, discarded, and donated. Presumably, fish used for pet food, meal, oil or bait has been taken into consideration. On the other hand, such approaches may underestimate fish consumption because they do not consider freshwater fish consumption, sport fish consumption or aquaculture fish production (figures reported by NOAA are in numbers of fish and not pounds of fish). In addition, imported fish (either fresh or canned) or ethnic variations in fish preparations and socioeconomic patterns of fish consumption are also not considered. Yet again, it might be argued that the sum of these positive and negative influences on the estimate of fish consumption are self-canceling and such estimates are easy and practical. In any or all events, more accurate information would be necessary to resolve the conflicts.

N.5.2 Estimation by Surveys

The patterns for seafood consumption is extremely diverse and strongly dependent upon the ethnic and sociodemographic as well as to socioeconomic characteristics of the population under study. These facts, along with the inherent conflicts associated with estimates based on fish production data, have led investigator to attempt surveys. Table N-7 presents the diverse total seafood consumption reported from different countries. This table also suggests that the U.S. has one of the lowest fish consumption rates in the world with a U.S. EPA estimate of 20 grams/day as an average individual consumption rate.

Table N-7.
Reported Daily Fish Consumption by Selected Populations

Country	Average Consumption (g/p-d)	Reference
U.S. General Population	20	National Academy of Sciences, 1978 UN Food and Agricultural Organization, 1977
Sweden, Denmark, Spain	40 - 60	UN Food and Agricultural Organization, 1977
Japan	100 84	UN Food and Agricultural Organization, 1977 FAO-UN
Sardinia, New Guinea	200 - 300	Bernhard & Renzoni, 1977; NAS, 1978 Kyle and Ghani, 1982

Table N-7.
Reported Daily Fish Consumption by Selected Populations (Continued)

Country	Average Consumption (g/p-d)	Reference
Great Britain	200 - 300	Haxton et al., 1979 Sherlock et al., 1982
Minimata Japan	800 - 1500	Tsubaki and Irukayama, 1977
Western Europe	800	Bernhard & Rensoni, 1977
Canadian Population	450 - 1300	Barbeau et al., 1976
Gen Canadian Pop.	16	CCAC, 1977
Kuwait	7.5	Khordagui & Al-Ajmi, 1991
Sweden	low intake = 14.3 high intake = 142.9	Skerfving, 1974
Peru	110 - 660	Turner et al., 1980
Source: Inskip, M.J. and J.K. Piotrowski, 1985.		

General Population

As indicated above, there have been a number of attempts to address the patterns of seafood consumption in the United States which include the assumptions such as consuming one seafood meal per week, an estimate of per capita consumption based on the tonnage of seafood landed, and an estimate of the per capita consumption based on seafood purchased. Surveys, on the other hand, avoid such assumptions and can provide information regarding the sociodemographic characteristics, and fishing activities of the population under study (EPA 1992).

Based on a survey of 6,980 families representing the U.S population and conducted in 1973-74 by NPD Research, Inc., an average value of 14.3 grams per person per day (g/p-d) was estimated for the consumption of all seafood. To date, this survey represents the most ambitious and comprehensive study that specifically focused on the consumption of seafood. The study population was weighted on the basis of a number of census-defined controls, which included census region, family size, income, children, race and age. The head of the household completed a 1 month diary of fish purchases. The families answered questionnaires concerning the date of meals containing fish, type and quantity of fish, and whether the fish was recreationally caught or purchased. Meals eaten away from home were also included in this survey. The total number of fish consumers was 24,652, representing on a weighted basis, 94 percent of U.S. residents (US EPA Exposure Factors Handbook, 1989). Some of the key findings from this study are:

- 93% of the participants consumed seafood during the 1-month survey period.
- Individual from the Great Lakes Region have a higher average consumption of 20 g/p-d.
- 187 g/p-d is the highest consumption value recorded.
- 18.58 ounces per month was the average consumption of all seafood per individual (17.55 g/p-d). Slightly over 60% of the participants consumed tuna fish and had an average consumption of 5.6 g/p-d.
- 22% of the participants consumed shrimp, with the average consumption of 7.5 g/p-d.
- The 10 most popular fish consumed, in descending order, are:
- Tuna > Shrimp > Flounder > marine Perch > Salmon > Clams > Cod > Pollock > Haddock > Herring.

Using the consumption data from the NMFS survey and an average Hg residue level of 0.3 ppm, Cramer (1994) estimated the average consumption rate for the general seafood eating population to be 32 g/p-d (Table N-8).

Table N-8.
Estimate of the Average Daily Consumption of All Fish and Mercury

Age	Fish Consumption (g/p-d)		Hg Intake (µg/p-d)	
	Average	90th Percentile	Average	90th percentile
all ages	32	64	10	19
2-5 yrs	16	32	5	10
18-44 yrs	35	72	11	22
All ages (Monte Carlo)	—	—	4.4	10
Source: Cramer 1994.				

A national dietary survey of the Canadian population estimated that the national average consumption rate for fish and shell fish was on the order of 11 g/p-d (HWC, 1977; Conacher et al., 1989) with a national average consumption of freshwater fish at 1.2 g/p-d (Conacher et al., 1989). This survey suggests that Canadian fish consumption is less than the U.S. It would appear that the Indian population which subsists on fish consumption were not included in this survey.

However, a survey asks an individual to recall information about amounts, types and frequency of food consumed over some time period or ask the individual to maintain a diary. The accuracy of the recall information is strongly influenced by the length of time over which the individuals are asked to itemize their eating habits. The use of food diaries overcomes the inaccuracy of recall surveys they are labor intensive, expensive and may suffer from under reporting (Cramer, 1994).

Such a diary survey was conducted by Smith et al. (1985) on an adult population. Of the 2000 panel members, 1437 (71.8%) responded with a completed food consumption diary of at least 1 month, (4400 monthly diaries were collected), signed consent form and a hair sample. This population was distributed throughout the U.S., demographically balanced, and included 422 individuals that did not eat fish. Fish intake by species and amounts were recorded by the diaries and correlated with 1307 corresponding hair samples. Based on the correlation of hair samples with dietary intake, the estimate for the average fish consumption rate is 20 g/p-d.

Recreational Anglers

For recreational anglers, survey data for fish and shellfish consumption ranges between 36.9 and 224.8 g/p-d (Puffer, 1981) on the basis of interviews with 1,059 sport anglers from 12 fishing locations in the Los Angeles area. Each site was surveyed on the average of 3 times per month. The sample number of 1,059 sport anglers was extrapolated to estimate 91,606 anglers in the area. Finally, with the inclusion of family and living groups of the anglers, a population of 342,606 was estimated to consume locally caught fish. Calculations for fish consumption rates were conducted only for those anglers who indicated that they eat the fish they catch as follows:

Consumption = K x NW/E x F/365

where:

K = edible portions of fish

F = frequency of fishing per year

E = number of fish eaters per family

W = average weight of fish

N = number of fish in catch

Other assumptions associated with this survey attempt were:

1. The average weight and amount of fish per catch is constant.
2. The fishing frequency of each angler is constant throughout the year.

3. The number of family fish eaters is constant (>0).
4. All of the catch is eaten and 25 to 50 percent of the weight is edible.

The Asian-American group had a higher consumption of recreationally caught fish than the other ethnic groups interviewed (Table N-9) and the California halibut was consumed the most by the anglers at a median consumption of 143.1 g/p-d (Table N-10).

Table N-9.
Ethnic Group Distribution of Anglers and Their Families

Ethnic group	Percent interviewed	Median Consumption (g/p-d)
Caucasian	42	46.0
Black	24	24.2
Mexican-American	16	33.0
Oriental/Samoan	13	70.0
Other	5	—

Source: Puffer (1981); EPA Exposure Factors Handbook (1989)

Table N-10.
Median Consumption of Primary Fish by Sport Angler (n=1059)

Species	Median Consumption (g/p-d)
White Croaker	14.8
Pacific Mackerel	35.8
Pacific Bonito	63.6
Queensfish	7.8
Jacksmelt	9.4
Walleye Perch	5.4
Shiner Perch	2.0
Opaleye	16.1
Black Perch	8.1
Kelp Bass	3.9
California Halibut	143.1
Shellfish	10.0

Source: Puffer (1981)

In Tacoma, Washington, Pierce (1981) interviewed 304 sport anglers in the summer and 204 in the fall for 5 and 4 consecutive days respectively. The ethnic composition of this population was similar to the Los Angeles population except that Los Angeles had more Mexican-Americans which displaced primarily the Caucasian group (Table N-11). The

range of fish consumption was between 23-54 g/p-d, but only 56% of the individuals interviewed indicated that they ate the fish they caught and only 2% ate fish more than once a week. Heatwole and West (1984) interviewed shoreside anglers in the New York Bight area and found that only 21% intended to consume their catch.

Table N-11.
Ethnic Composition of Anglers in Tacoma, Washington

Ethnic group	Summer	Fall
White	58.9%	60.8%
Black	22.7%	15.2%
Oriental	15.5%	23.5%
Mexican	2.6%	0.5%
Indian	0.3%	0%

Source: Pierce et al. (1981): EPA Exposure Factors Handbook 1989

Landolt et al. (1987) also surveyed boat and shore anglers on Puget Sound, specifically Edmonds Bay and Commencement Bay. They reported no significance differences in consumption patterns due to ethnicity with frying the skinned fillet the preferred method of preparation (skinned fillets were preferred over unskinned fillets by 4:1). The average fish consumption rates across species were 14 g/p-d for Edmonds Bay and 8 g/p-g for Commencement Bay with a mean consumption rate for the population studied equal to 11 g/p-d. More than half (51.7%) of the shore anglers were unsuccessful catching fish while only 31.1% of the boat anglers were unsuccessful. The range of Hg in the fish fillets was 0.001–0.09 ppm wet weight.

Subsistence Anglers

The average consumption of subsistence anglers can be expected to be considerably higher but is subject to variability which can be attributed to demographics, socioeconomic status, the patterns of seafood preparation and consumption as well as success of the angler. An average value of 69 g/p-d is estimated on the basis of an average red meat consumption pattern in the non-subsistence population but an average value of 134 g/p-d and as high as 396 g/p-d have been reported for an isolated populations on Kodiak Island in Alaska. However, use of fish consumption estimates such remote and northern populations may not be appropriate for U.S. purposes.

The identification of a population sufficiently large to conduct a survey for fish consumption is a major problem with subsistence anglers. However, Richardson and Currie, (1993) studied the native Ontario Amerindians residing in 58 reserves across the Canadian province. A major portion of this Indian population subsists on freshwater fish as a component of their diet. Hair samples of 4,327 of the Amerindians studied were analyzed for mercury.

The concentration of Hg in the three most commonly consumed fish was also determined. From these two analytical end points, consumption rates were calculated. Increases in fish consumption were also found to parallel with age, increase with latitude and have a seasonal variation that peaked during the summer months but the geometric mean consumption rates for males was 18.9 g/p-d, 14.4 g/p-d for females and an overall average of 16.2 g/p-d. For a population that subsists on freshwater fish, this estimate appears to be excessively low as a fish consumption rate.

Pregnant Women

A search of the literature did not produce a "recall" or "diary" survey regarding pregnant women. It is apparently also difficult to assemble a suitably large enough population of pregnant women to conduct such a survey.. However, information on Hg exposure and fish consumption is available for several "pregnant" populations by investigators that used biological exposure indicators (see below).

N.5.3 Alternative Estimations for Fish Consumption Rates

Direct Estimation of Dietary Mercury Intake

An alternative method for estimating the fish consumption rate for a given population is through direct dietary analysis for Hg or MeHg. The FDA sponsors an annual program that purchases selected grocery items across the Unites States and analyses these foods, as composites, for essential minerals, pesticides, radionuclides, and industrial chemicals. Included in this study are four seafood products; tuna, shrimp, fish sticks (probably pollock) and cod/haddock fillets. Total Hg is included in this analysis and the FDA has determined that 84% of the total Hg in the 1986-1991 diet is derived from the four seafood products. From these analysis, the FDA has estimated that the total Hg exposure from the diet is relatively unchanged over the past decade at roughly 0.03–0.04 μg/kg b.w./day for individuals 14 years and older. For the standard 70 kilogram man this is equivalent to an average total Hg intake of 2.4 μg/d. Assuming that the majority of the population preferentially consumes primarily marine fish with an average concentration of 0.11 ppm, this calculation estimates the average consumption of fish to be on the order of 21.8 g/p-d. This number is almost identical to the Hg intake and fish consumption rates estimated by the Smith data.

This method of estimation of fish consumption can be favored because it is free from most of the deficiencies and assumptions necessary by other methods of estimation. It relies strictly on analytical data and an estimate of the average concentration of Hg in fish. Unfortunately, however, it cannot account for individual differences in consumption patterns.

Use of Biological Exposure Indicators

Successful indices for exposure to methylmercury can be provided by measurements of mercury levels in hair or blood. Detailed studies of human poisoning episodes have allowed the calculation of the half-life of Hg in humans (about 70 days) and the relationship between ingestion of methylmercury from contaminated fish and the levels of Hg in the hair or blood (Tollefson and Cordle, 1986: Grandjean et al., 1992; Matthews, 1983). A steady state body burden of methylmercury is generally achieved over the course of one year of continual exposure. Thus, at steady state, the blood level expressed in ng/ml (ppb) is approximately equal to the daily intake expressed in µg/d for a 70 Kg person (Clarkson, 1977) or the difference between the two values is 0.001 (ratio: 100:1). However, Skerfving (1974) reported a slightly different ratio (0.00066) with his correlation of an intake of 300 µg/d by a 70 kg person results in a blood concentration of 200 ppb.

General Population. Cramer (1994), using the fish consumption data from the MRCA 1982-87 survey, utilized Monte Carlo analysis to estimate the most likely Hg residue exposure and developed an average most likely exposure estimate (MLE) of 4.4 µg MeHg/p-d (Table N-8). Using data generated by Smith (1985), the back calculation from 4.4 µg/g results in a Hg concentration in the hair of 0.4 µg/g. This value, plus the additional background level of 0.17 ppm raises the Hg level in the hair to 0.57 µg/g hair; a value that is 60% larger than the mean hair levels reported in the Smith survey as a geometrical mean for the general population.

In the survey and study of the general population by Smith (1985;1994), the levels of Hg in hair were lognormally distributed with a geometric mean of 0.35 µg/g hair with an arithmetic mean of 0.57 µg/g. The non-fish eaters showed a hair concentration of 0.17 µg/g. As a result, this hair concentration cannot be correlated to fish consumption and can serve as a background or blank value. This suggests that actual fish consumption only contributes 0.18 µg/g to the hair. As a result, the hair correlates to an intake of 2 µg MeHg/day. Assuming an average MeHg concentration in fish of approximately 0.11 ppm, the arithmetic average daily consumption of fish amounts to 18 grams of fish/day; considerably less than the estimated 32 g/p-d from the MRCA calculated estimate proposed by Cramer (1994).

Recreational Anglers. It does not appear that mercury exposure of recreational anglers has been attempted with the use of Biological Exposure Indexes.

Subsistence Anglers. Richardson and Currie (1993) attempted to use biological indicators for estimating fish consumption rates in Amerindians. Hair samples were collected from 4,327 adult Amerindian Canadians; 1,789 males and 2,538 females. The calculations were based on the following assumptions:

- hair grows at the rate of 1 cm/month
- the Hg in fish is greater than 99% methylmercury

- trout, walleye and pike were the important fish species analyzed for Hg content
- the geometric mean for Hg concentrations in these fish species was used to estimate fish consumption rates
- the deposition of Hg into hair is a linear function of absorption from the gut.
- the value of 0.001 represents the ratio of steady-state blood Hg concentration to the daily dietary intake
- the ratio between blood and hair levels was 292.

The estimation of Fish Consumption was determined by the following relationships:

Hg intake (m/day) = fish consumption rate (g/day) x fish [Hg] (μg/g)

Blood [Hg] (μg/ml) = **a** x Hg intake (μg/day)

Hair [Hg] (μg/g) = **b** x blood [Hg] (μg/ml)

Thus,

Hair [Hg] (μg/g) = **a** x **b** x fish consumption rate (g/day) x fish [Hg] (μg/g)

Fish consumption rate (g/day) = hair [Hg] / (fish [Hg] x **a** x **b**)

Where:

a = The ratio of steady state blood Hg concentration to daily dietary intake of mercury estimated to be 0.0010 (WHO, 1976; Kershaw et al., 1980)

b = The ratio of hair [Hg] to blood estimated to be 250 in populations exposed to methylmercury (WHO 1976).

On the basis of these data and assumptions, the geometric daily fish consumption rates were 18.9 g/d for males, 14.4 g/d for females and 16.2 g/d for the combined sexes (Table N-12). Considering the fact that these Indian populations eat fish as a major source of dietary protein Richardson and Currie suggest that estimates for fish consumption rates for other population are excessive. Alternatively, the estimation of the Amerindian consumption rate may be too low and may be confounded by the double relationship used (i.e., hair/blood followed by blood/ingestion relationships).

Table N-12.
Estimation of Fish Consumption Rates of Amerindians in Canada

No of individuals/ GENDER	Hair Hg Geometric mean (range)	Fish Hg Geometric mean (range)	Fish consumption (g/d)
1789/M	2.7 µg/g (0.3 - 128.0)	0.39 µg/g (0.01 - 6.13)	19
2538/F	2.1 µg/g (0.2 - 38.4)	0.39 µg/g (0.01 - 6.13)	14
4327/total	2.3 µg/g	0.39 µg/g (0.01 - 6.13)	16 (64)*

Source: Richardson and Currie, 1993.

* recalculated "most likely value" based on the data of Smith, 1994.

A more direct approach of converting hair analysis to a fish consumption rate produces higher numbers. For example, Stern (1993) reports a relationship of 10 µg Hg/g hair is equivalent to a dose of 0.7 µg Hg/kg b.w./d. or 49 µg/p-d for a 70 kg male. With a geometric mean Hg concentration of 2.7 µg Hg/g hair in males (Table N-12) reported by Richardson and Currie (1993) it can be calculated that the daily intake of MeHg corresponds to 13.2 µg/d. At an average concentration of 0.39 ppm Hg in fish, a daily fish consumption rate of 33.9 g/d can be calculated; considerably greater than the estimates of Richardson and Currie (1993).

Using the hair/dietary intake relationship of Smith (1994), a hair concentration of 2.7 µg Hg/g would correspond most directly to a daily Hg intake of 30 µg (This value is sufficiently large that any background contribution is negligible). At an average fish Hg concentration of 0.39 µg/g, the consumption rate would be 77 g/d; considerably greater than Richardson's estimate of 19 g fish/p/d, double the estimate using Stern's relationship but closer to what might be expected as a fish consumption rate for a subsistence fishing population or one associated with high fish consumption rates seen in other countries (Table N-7).

Kyle and Ghani (1982) studied a New Guinea population at Lake Murray that depended on fish as their total dietary protein source. The giant perch is the predominant fish species while the catfish is the secondary fish species consumed. Chemical analysis showed a mean MeHg content of 0.49 ppm and 0.57 ppm total Hg (tHg) content of perch while catfish had a mean MeHg content of 0.17 ppm and tHg content of 0.20. The average content of MeHg in the 114 hair samples collected was 15.5 ppm (Table N-13). Using the Stern relationship of MeHg to dietary exposure, this hair concentration translates to a Hg ingestion rate of 76 µg/d associated with fish consumption. Assuming a 3:1 distribution of

perch to catfish, and that tHg equals MeHg content, the average Hg concentration in the fish is approximately 0.47 ppm. This translates to a consumption rate of 159 grams of fish per day.

Table N-13.
Concentrations of MeHg and Total Hg in Fish and Human Hair at Lake Murray

Analyte	MethylHg Geometric mean, ppm (range)	Total Hg Geometric mean, ppm, (range)	Sample SIZE
Giant Perch (Lates calcarifer)	0.049 ± 0.33 (0.19 -1.13)	0.57 ± 0.38 (0.23 - 1.40)	12
Catfish (Hexanematichthys latriostris)	0.17 ± 0.05 (0.09 - 0.23)	0.20 ± .06 (0.12 - 0.28)	6
Hair	15.5 ± 6.9 (3.2 - 50.5)	18.0 ± 7.8 (4.1 - 58.4)	114

Using the Smith relationships, the Lake Murray population is exposed to 172 µg/p-d and consumes 191 grams of fish. Again, either of these values can be considered to reflect the fish consumption rates for a population that eats fish 2–3 times a day.

Pregnant Women It is suspected that the fetus is more susceptible to methylmercury insult than the adult. As a result, there is growing concern about the levels of exposure experienced by pregnant women but data on the fish consumption rates for this subpopulation is scarce. Soria et al.,(1992), reported that the median maternal blood-total mercury concentration from 77 volunteers was 6.23 ppb. Using the relationships of Skerfving (1974) this blood level would correspond to a daily intake of 9.2 µg Hg/d. Assuming that the average concentration of Hg in the fish approximates 0.11 ppm, the calculated fish intake for these women averages 84.5 g/d.

Using the blood/diet relationship of 0.001 suggested by Clarkson, the daily Hg exposure rate would be 6.23 µg/d corresponding to a fish consumption rate of 56.6 g/p-d (0.11 ppm in fish). Although there is a 30% difference, either one of these numbers is in the range of the high average fish consumption rate for Spain (see Table N-7) which can be expected for and area likely to have to have a relatively high fish consumption rate.

Skerfving (1988), studied seven postpartum Swedish women. His data provides blood cell values and plasma ratios and indicates that ingestion of 0.3 µg/kg produces a plasma level of 5 ng/g (ppb) total Hg (WHO, 1976). Based on this relationship, a daily mercury exposure can be calculated. For example, nine mothers with an average plasma Hg levels of 3.8 ng/g have an average daily exposure to approximately 11.4 µg Hg. This assumes

an average body weight of 50 kg, a total intake of 15 μg (50 kg x 0.3 μg/kg) per day, yielding a plasma level of 5 ng/g. One individual did not eat fish but had a blood cell Hg level of 2 ng/g. This value may be considered a basal blood Hg level and must be attributed to Hg sources other than fish. Two individuals ate only commercial fish and they had a blood cell Hg level of 6 and 10 ng/g. Other individuals had blood cell Hg levels between 12-72 ng/g and ate coastal water fish as well as lake fish with a residue level that ranged from 0.3–0.8 μg/kg fish (it is assumed that the average value is 0.5 μg/kg). The calculations indicate that the fish consumption level in these post-partum women ranged from 25–35.5 g/d after correcting for background levels and are summarized in Table N-12. These estimations indicate that the fish consumption rate is well within the national range (Table N-7). However, plasma Hg levels may provide underestimates of fish consumption, particularly when levels exceed 3 meals per week since the highest consumers of fish (3 fish meals/ week) exhibited lower blood Hg levels than individuals consuming 2 fish meals/week (Svensson et al., 1992). The increase in blood Hg associated with increasing fish consumption followed by decreasing blood Hg levels with continued increase in fish consumption does not appear to have a ready explanation unless it can be correlated to the consumption of fish species with lower Hg levels. Alternatively, such data suggest that the designation of a high fish consumer by admission or by socioeconomic status is likely to be an overestimate.

Marsh et al., (1985) studied 413 pregnant women living in New Bedford Massachusetts. The hair level of MeHg was determined to be lognormally distributed with an arithmetic mean of 0.74 ppm. After correcting with the 0.17 background value (Smith, 1994), these women had a daily ingestion rate of 6.3 μg/d (Smith et al., 1985) which can be correlated to 57.6 grams of fish per day while the Stern correlation suggests a consumption rate of 33 g/p-d. Either of these values would correspond to the expected rates of fish consumption in a fishing community such as New Bedford. It also suggests that fish consumption in non-fishing communities are likely to have a be lower and similar to the national average rate.

N.6 Conclusions Regarding Mercury Exposure by Fish Consumption

It is apparent that attempts to determine either the fish consumption rate or mercury intake level of the U.S. population is fraught with difficulty, highly variable and based on a number of broad and not completely valid assumptions. Estimations of the fish consumption have been based on assumptions a tenuous as: The average U.S. citizen consumes one seafood meal per week; to a per capita consumption estimate based on the tonnage of seafood landed per year or a per capita consumption estimate based on the amount of seafood purchased per year to the gathering of data by any number of surveys. These attempts have produced a wide range of numbers, any of which can only be selected with a limited degree of confidence.

The most desirable method for estimating rates of fish consumption appears to be a method that avoids many of the generalizations associated with per capita estimates and is free from subjective response and recall inherent in surveys. A method appears based on a Biological Exposure Index (BEI) amenable to non-invasive sampling such as hair sampling or only minor invasion such as blood sampling is the most suitable. Chemical analysis of such samples will then provide an estimate of the exposure. Using the distribution ratios between body or kinetic compartments (e.g., hair/blood) will allow a more accurate estimation of exposure to Hg directly or indirectly through fish or grain consumption.

The Hg concentration in the dietary source also need further investigation and clarification. The recent report of Bloom (1992) claiming that all the Hg in fish is in the form of MeHg needs confirmation because such a concept is intuitively conflicting with established and known metabolic processes in biological systems.

With respect to fish ingestion, relating the BEI to a consumption rate also requires further investigation. Two potential values are currently available to related hair concentrations with ingestion. One value, presented by Stern (1993) estimates that the relationship between hair Hg and ingestion is 10 μg/g hair equivalent to 0.7 μg Hg/kg b.w./d. A second value of 1 μg Hg/g hair equivalent to 10 μg Hg ingested is provided by the data of Smith (1985;1994). This latter factor also has the advantage that it incorporates a "background" value which is important at low Hg exposure rates but becomes insignificant at higher Hg exposure rates. Although these two values differ by a factor of 2, either one appears to estimate fish consumption rate with a reasonable degree of accuracy. The resolution of these two differing values would provide greater accuracy and confidence in any fish consumption term.

From the current discussion, it would appear that the fish consumption rate for the average U.S. adult is in the range of 18–20 g/p-d. Quantitative values for fish consumption by recreational anglers are scarce and those that are available are highly variable and intuitively suspect. Quantitative fish consumption values associated with subsistence fishing are also highly variable. Estimated values by recall surveys range from 135 g/d to 396 g/d. Estimates using Biological Exposure indicators have an apparent range of 16 g/d to almost 200 g/d. Part of the variability for subsistence fishing may be related, in part, to physical and chemical composition of the water body generally fished, the species pursued (freshwater vs. marine) and the relative success of the angler as well as other less obvious parameters.

It has been suggested that the fetus is more susceptible to Hg toxicity than the adult. This has raised concern about the Hg exposure of pregnant women and spurred an effort to identify the amount of fish a pregnant woman might consume. Pregnant women are undergoing dramatic physiological changes which often adversely affect their feeling of well being. This suggests the possibility that pregnant women may not consume or decrease their consumption of fish. However, from the data and present calculations,

pregnant women do not significantly alter their fish consumption rate and overall, consume fish at the national average rate. Pregnant women in local communities appear to consume fish at the average rate of that community.

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Electric Utility Trace Substances Synthesis Report

Volume 3: Appendix O, Mercury in the Environment

TR-104614-V3

Research Project 3081, 3297
November 1994

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EPRI FOREWORD

The Electric Power Research Institute (EPRI) began funding of mercury research in 1983 with a project to measure environmental mercury. Over the last decade the direction of the work has expanded to include efforts in many of EPRI's divisions and programs focusing on fossil fuel analyses and emission measurements, studies of control technologies, atmospheric cycling, ecosystem cycling, health effects, and risk assessments. All of these areas have benefited from development of analytical and sampling methods that are more sensitive, reproducible, and accurate than older methods. Additional improvements are needed; though there is less uncertainty about mercury's cycling and effects than previously, there is still much to learn.

No obvious health effects from mercury have been observed in human populations, except in cases of gross exposure (such as industrial discharges into Minimata Bay, Japan, and grain contamination in Iraq). Furthermore, reducing electric utility emissions does not appear to provide much benefit in lowering the most obvious potential health hazard, mercury accumulation in fish. Should further study show that it is desirable to protect human populations from given levels of mercury exposure, new generation technologies to replace older and uneconomic technologies may offer opportunities for less-expensive and more effective removal of mercury and other trace substances. This report explores these complex issues by summarizing our current knowledge of the trace substance, mercury, and defining where important uncertainties still exist.

EPRI environmental science research is conducted through contracts with state agencies, universities, and other research institutions throughout the United States and in other countries. It is EPRI policy to encourage researchers to make the results of their investigations available to the scientific community, as well as to other interested parties, as expeditiously as possible. When the work is complete, results are published in the peer-reviewed literature.

This work presents the results gleaned from many projects sponsored by EPRI and others. The review was written by EPRI project managers, representing work in progress and published results. We hope this document will be useful to researchers, utility sponsors, and other interested agencies.

Ian Torrens, Issue Manager
Electric Utility Trace Substances

ACKNOWLEDGMENTS

This Mercury Synthesis Report borrows liberally from work performed and reported elsewhere by many investigators. However, the report was written by EPRI staff: Mary Ann Allan, Ramsay Chang, Paul Chu, Leonard Levin, Babu Nott, David Owens, Don Porcella, Abe Silvers, Barbara Toole-O'Neil, and Ron Wyzga.

The major work described in this Synthesis Report was funded by the Electric Power Research Institute (EPRI). Also, the Wisconsin Department of Natural Resources funded much of the lake work. The Empire State Electric Energy Research Corporation (ESEERCo) helped fund the lake studies in Michigan and funded most of the Adirondack atmospheric and lake mercury research. Cooperative funding by the U. S. Environmental Protection Agency (USEPA) provided essential support for initial studies of fish. Also, the U. S. Fish and Wildlife Service and the U. S. Geological Survey funded part of this research. Cooperative research funded through the National Acidic Precipitation Assessment Program by the USEPA provided an experimental manipulation and important ancillary information on Little Rock Lake. Furthermore, EPRI wishes to acknowledge the participation of Public Service Company of Colorado and the Environmental Control Technology Center co-sponsors: New York State Electric and Gas Corporation; ESEERCo; the United States Department of Energy (USDOE); Electric Power Development Company; and Babcock and Wilcox. Substantial coordinated and cooperative efforts have resulted from joint field studies of emissions with the agencies and personnel of USEPA, USDOE, the U. S. Department of Interior (USDI), the Tennessee Valley Authority (TVA), and many private researchers.

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ACRONYMS

BMD	Benchmark Dose
CAAA	Clean Air Act Amendments of 1990
CVAAS	Cold Vapor Atomic Absorption Spectrophotometry
CVAFS	Cold Vapor Atomic Fluorescence Spectrophotometry
DOC	Dissolved Organic Carbon
ECTC	Environmental Control Technology Center
EPRI	Electric Power Research Institute
ESP	Electrostatic Precipitator
ESEERCo	Empire State Electric Energy Research Corporation
FCEM	Field Chemical Emissions Measurement program
FGD	Flue Gas Desulfurization system
INAA	Instrumental Neutron Activation Analysis
IRIS	Integrated Risk Information System database
MCM	Mercury Cycling Model
MEI	Maximally Exposed Individual
MESA	Mercury Speciation Adsorption method
MTL	Mercury in Temperate Lakes project
MWe	Megawatts electric capacity
NAPAP	National Acidic Precipitation Assessment Program
NAS	National Academy of Sciences
NCRDS	National Coal Resources Data Set
NIEHS	National Institutes of Environmental Health Sciences
NOAEL	No-Observed-Adverse-Effect Level
PBPK	Physiologically Based Pharmacokinetic model
PISCES	Power Plant Integrated System: Chemical Emissions Study
REI	Reasonably Exposed Individual
RfC	Reference Concentration
RfD	Reference Dose
TPJ	Transportable Pulse-Jet baghouse
TRUE	Total Risk of Utility Emissions model
TVA	Tennessee Valley Authority
USDI	U. S. Department of Interior
USDOE	U. S. Department of Energy

USEPA	U. S. Environmental Protection Agency
USFDA	U. S. Food and Drug Administration
USGS	U. S. Geological Survey
WHO	World Health Organization

EXECUTIVE SUMMARY

There are numerous and diverse natural and anthropogenic sources of mercury. Mercury cycles through the atmosphere and deposits in ecosystems where it enters surface waters, is transformed, and is accumulated by fish almost exclusively as methylmercury.

Methylmercury represents a health risk—to fetal neurosystems in particular. Current understanding of mercury health risk from power generation is based on recent studies of power plant emissions of mercury, its atmospheric and ecologic cycle, and its effects on populations of human beings sensitive to mercury exposure. Quantification of health risk provides important information for decision makers to use in determining the need for emission controls.

The following statements summarize our current understanding of knowledge about mercury. Research needs are noted where further results would supply information critical to decision makers.

- Mercury emissions from U. S. electric power generation—dominated by coal combustion—are about 40 metric tons per year. This is less than half of the most recent previous estimates.

- U. S. electric power generation contributes less than 1 percent of the total mercury input to the global atmosphere and about 1 percent of the anthropogenic share of that input. Electric power generation contributes no more than 16 percent of total estimated mercury emissions to the atmosphere arising from U. S. sources. A more complete inventory of sources of input to the atmosphere is needed to make accurate estimates of both global and U. S. emissions.

- Temporal trends in emissions of mercury are not well-characterized. Although current deposition is 2–5 times pre-industrial estimates, there is strong evidence in Minnesota and the United Kingdom that atmospheric mercury deposition has decreased dramatically since about 1960. A broader-scale historical perspective is needed to understand these recent trends. More

extensive sampling of peat and lake sediment cores may provide such a perspective.

- Whether emissions will deposit locally, regionally, or enter the global atmospheric cycle remains somewhat uncertain although models have been used to apportion such deposition. Both atmospheric processes and mercury chemistry govern how much deposition comes from global, regional or local sources. For example, phase (particle versus gas) and chemical species [elemental mercury, Hg(0); inorganic mercury, Hg(II)] affect the transport and deposition of mercury. Additional research on conversion of Hg(0) to and from Hg(II), phase changes of Hg(II) from gas to particle form in emission plumes, and the fate of particles themselves is needed to obtain accurate models of the atmospheric fate of mercury emissions. Furthermore, aqueous-phase transformations of mercury are important to transformation rates.

- Fish accumulation of mercury is related to inputs, but no consistent relationship has been found. The proportion of deposited mercury transformed to methylmercury (CH_3Hg^+)—and available for accumulation in fish tissues—appears to be dominated by local factors. In fact, in areas presumably dominated by atmospheric deposition of mercury, fish mercury apparently has not changed while mercury deposition has decreased by a factor of 2–3 over the same timeframe. Understanding the processes of methylation and demethylation appears to be key to evaluating the link between deposition and accumulation. The Mercury Cycling Model can be used to assess the important variables that affect this link, and has predictive value in small-lake ecosystems. However, extrapolating the model to other environments awaits results from ongoing research.

- Risk assessments remain somewhat incomplete. However, initial assessments at four case study sites show the risks to humans to be quite small in comparison to federal reference doses. Assessment of mercury-related risks to humans from power plant emissions requires additional research, but to date, the most conservative assumptions at a limited number of case study sites indicate that effects are below current levels of concern—less than 30 percent for the worst cases.

Section 1
INTRODUCTION

Mercury Issues

The 1990 Clean Air Act Amendments (CAAA) require the U. S. Environmental Protection Agency (USEPA) to carry out an assessment of health and environmental effects caused by mercury emissions. Also, the law requires a separate assessment of health risk due to emissions of 189 trace chemicals (including mercury) from fossil-fuel-fired electric generation by utilities. Mercury is only one of the CAAA-listed trace chemicals potentially emitted into the atmosphere, but it was singled out for separate study and risk assessment because of its potential effects on human health. Mercury is not a carcinogen in humans, but appears to affect neural tissues, primarily.

Mercury is a crustal element that is mobilized by both natural and human activities, and cycles through atmospheric, aquatic, and terrestrial environments. Mercury cycling varies spatially and temporally, because natural and anthropogenic sources are located in different geographic areas and source strengths have varied historically. In addition, aquatic and terrestrial sources contaminated from previous human activities may contribute substantial emissions as part of so-called "background" emissions. Mercury undergoes transformations among its chemical forms, including elemental mercury [Hg(0)], inorganic or oxidized mercury [Hg(II)], and methylmercury (CH_3Hg^+). The chemical form affects transport through air, land, and water, as well as chemical and biological behavior.

The most important form of mercury for impact assessment is methylmercury. Methylmercury is a neurotoxin and is regarded as the most toxic chemical form of mercury. Biotic and abiotic methylation of inorganic mercury produces methylmercury, and fish accumulate methylmercury from water and their diet. Nearly all (more than 95 percent) of mercury in fish flesh occurs as methylmercury (Huckabee et al. 1979; Grieb et al. 1990; Bloom 1992a). Fish consumption by human populations raises health concerns, especially for developing neural systems of fetuses and young children (Clarkson 1990; Fitzgerald and Clarkson 1991). Similarly, methylmercury can adversely affect

developing neural tissue of mammals and birds that consume fish (for example, see review by Zillioux et al. 1993). Game fish consumption is the major route of mercury exposure to humans. Game fish are generally large, older fish that prey on other fish; consequently, they have higher concentrations of mercury, and the mercury concentration increases in fish as they age (Grieb et al. 1990). For humans and wildlife, the non-fish diet is relatively low in methylmercury.

To protect the health of humans and other sensitive organisms, regulatory agencies focus on fish as the target organism. For example, the U. S. Food and Drug Administration (USFDA) set an advisory standard of 1 ppm of total mercury wet-weight in fish flesh, and the World Health Organization recommends that the dose from all sources should not exceed 30 μg/day to protect adult humans. Many states use 0.5 ppm wet-weight in fish flesh to set fish-consumption advisories. Finding fish in a body of water that exceed established advisories leads health agencies to issue sport fish consumption limits to protect at-risk populations.

Mercury has been observed in a wide variety of environments, but attention has been extended recently to remote regions having dilute, relatively unproductive waters. Many people had assumed that the problem of mercury was solved by eliminating methylmercury discharges and methylmercury fungicides, and by reducing industrial mercury discharges to surface waters. The discovery of high levels of mercury in fish in areas remote from human activities disproved that assumption and, instead, implicated atmospheric deposition as a source (for example, Huckabee 1973; Lindqvist et al. 1984, 1991; Fitzgerald 1986; Rada et al. 1989). The atmospheric deposition has complex origins, coming from a variety of natural and anthropogenic sources, and from different spatially important scales. A significant fraction comes from the global background, as well as from local and regional sources. Because atmospheric cycling integrates many sources of mercury, control schemes are not straightforward.

Since 1983, the Electric Power Research Institute (EPRI) has sponsored research on environmental mercury to reveal the factors that may influence human health, including mercury in fish, and to determine the role of electric power generation in contributing to those factors. A key part of this research entailed the development of procedures for measuring mercury. This task was complex for

two primary reasons: (1) The concentration of mercury is extremely low in most media. Thus, not only are sensitive techniques needed, but contamination of sampling and analysis steps by background and laboratory mercury can lead to erroneously high results; and (2) Mercury occurs in nature in a variety of oxidation states [Hg(0), Hg(I), Hg(II)] and organic and inorganic chemical forms that complicate analytical procedures.

In the past, scientists inferred mercury's biogeochemical behavior by comparing fish mercury accumulation with water chemistry parameters and other limnological factors (Rudd et al. 1983; Håkanson 1980). Analytical problems forced this inference, because only sediments and biota contained sufficient concentrations for easy detection where sample contamination would have little effect. Recently, sample contamination problems have been overcome by development of ultraclean sampling and laboratory procedures along with more sensitive analytical techniques. These recent methods appear to provide accurate estimates of air and water concentrations of the different mercury forms (Fitzgerald and Gill 1979; Slemr et al. 1985; Gill and Fitzgerald 1987; Brosset 1987; Bloom and Fitzgerald 1988; Bloom 1989; Fitzgerald and Watras 1989; Gill and Bruland 1990; Iverfeldt 1990; Lindqvist et al. 1991; Munthe 1991; Fitzgerald et al. 1991; Porcella et al. 1992). Values of 1–2 ng/m^3 in air and less than 1–2 ng/l in clear lake water for ambient samples taken distant from known point sources are considered indicative of background concentrations. Concentrations in precipitation are considerably higher, requiring less sensitive techniques (5–25 ng/l) (Bloom and Watras 1989; Fitzgerald et al. 1991). The ultraclean methods for measuring mercury in these concentration ranges allow more accurate characterization of mercury biogeochemistry. Aside from the need for ultraclean sampling and analysis techniques for ambient air and water samples, nearly as much care is needed for sampling and analysis of fuels and emissions (Bloom 1992b). Application of these methods has formed the basis for studies reported in this document.

Objectives and Scope

The major research areas relevant to atmospheric mercury are emissions, biogeochemistry and accumulation in fish, and health risk from consumption of fish. More specifically, EPRI has sponsored research to characterize emissions to

the atmosphere from anthropogenic and natural sources, and to evaluate temporal and spatial patterns of mercury inputs to and outputs from the atmosphere. Biogeochemistry involves the factors affecting mercury cycling—including transformations and transport in the atmosphere—and the deposition of mercury onto watersheds and aquatic environments where further transformations and transport occur. Ecosystem compartments (fish and sediments are important compartments) then accumulate this mercury. Investigators apply methods gained from this ongoing research to assess human and animal health concerns and to evaluate risks. Not all of this research is complete; indeed some of it has barely begun—particularly studies to assess temporal and spatial patterns of natural and anthropogenic mercury emissions and deposition. Despite these gaps, the research effort has begun to provide answers to key scientific questions of relevance to the management of mercury.

In relation to these management issues, EPRI has identified the following questions whose answers provide information to decision makers for assessing the need to control power plant mercury emissions:

- What are the mercury emissions from electric power generation, today and what will they be in the future?
- How do these emissions compare with total emissions on regional and global scales?
- What are the historical trends in mercury emissions to the atmosphere?
- What are the spatial trends in mercury emissions to the atmosphere?
- How are emissions converted into deposition and input to ecosystems where health risk arises? In other words: Which factors govern and affect the atmospheric cycle of mercury, particularly its transport, transformation, and deposition?
- How much deposition at a site comes from global, regional, or local sources? Taking the question further: What are the spatial and temporal trends in the atmosphere and in deposition?
- What is the link between deposition and fish mercury levels? In other words: Which factors govern and affect the aquatic cycle of

mercury, particularly its transport, transformation, and bioaccumulation by fish in watershed, stream, lake, and wetland systems?

- Are there ecological risks associated with expected ambient concentrations of mercury?
- What are the human health risks, and what factors affect risk to humans?
- What are the mercury-related risks to human populations attributable to electric power generation?

Ongoing research has allowed scientists to frame these questions, and although answers are still being obtained, we have summarized known results in the following Sections: 2. Mercury Sources, 3. Atmospheric Mercury, 4. Mercury in Aquatic Ecosystems, 5. Health Effects of Mercury, 6. Mercury Risk Assessment, 7. Potential Mercury Control Approaches, and 8. Conclusions.

Section 2
MERCURY SOURCES

Sources of mercury to the atmosphere need to be considered at different spatial scales, because a site will receive deposition from global and regional, as well as local mercury sources.

Global Emissions

Nriagu and Pacyna (1988), using geometric means, made a broad-brush inventory of trace metals from a variety of sources on a global basis (Table 2-1), and they estimated that anthropogenic emissions of mercury accounted for more than half of the global atmospheric mercury cycle of about 6,000 metric tons/yr (Slemr et al. 1985; Fitzgerald 1986). Furthermore, Nriagu and Pacyna (1988) estimated that electrical generation on a worldwide basis accounted for about 13 percent of global anthropogenic sources. Lindqvist et al. (1991) estimated that, on a global basis, point sources emitted 3500 metric tons per year and diffuse sources (paint, landfills, etc.) emitted 1000 metric tons per year, with about 3000 metric tons per year attributed to natural sources, to total 7500 metric tons annually. There is considerable uncertainty in these estimates which may vary by a factor of 2–4 (Lindqvist et al. 1991). Mason et al. (1994) used the mean of these two anthropogenic estimates (4000 metric tons per year), and assumed that half of the anthropogenic emissions entered the global atmospheric cycle and half deposited on a local or regional scale. Although this assumption needs testing, these investigators have recognized clearly that mercury atmospheric cycling has a global dimension as well as local and regional dimensions.

Regional Emissions

USEPA compiled present-day estimates of mercury emissions from many sources to the atmosphere in the United States that amounted to about 300 metric tons per year in 1990 (MRI 1993) (Table 2-2). These data indicate two major sources of mercury to the atmosphere within the United States: coal combustion and solid waste combustion. Because many other historical sources of mercury emissions to the atmosphere have been controlled to a large extent (for example,

Table 2-1
Summary of Global Mercury Emission Sources
(Taken from Nriagu and Pacyna 1988)

Source Category	Geometric Mean of Range 1000 kg/yr	Range 1000 kg/yr
Coal—electric utility	290	155–542
Coal—other	1212	495–2970
Smelting—Pb, Cu, Zn	100	45–223
Waste incineration	579	155–2160
Other fuels	117	60–230
GLOBAL TOTAL	2300	910–6200 (median = 3560)

Table 2-2
Summary of U. S. Anthropogenic Mercury Emissions to the Atmosphere
(Data Compiled from MRI 1993)

Source Category	Metric Tons Per Year	Percent of Total
Industrial/Commercial	31.4	10
Electric utility: coal, oil, wood, geothermal	94.5	31
Other coal and oil uses	31.2	10
Waste combustion: medical, municipal, and sewage sludge	118.2	39
Paint emissions	13.2	4
Mobile sources	4.5	2
Smelting	8.2	3
TOTAL[a]	301.2	99

[a] Anthropogenic sources not included due to lack of information: refining (petroleum, oil shale), gas combustion from power plants, copper smelting, iron and other metal ore roasting, fugitive emissions (examples include landfills, mine spoils, mercury spills, dispersed mercury uses), mercury catalysts, by-product coke combustion. Total not 100 percent due to rounding.

chlor-alkali plants) or not accounted for, these two sources now appear as important contributors.

No evaluation of uncertainty occurred in the Midwestern Research Institute (MRI) report, but uncertainty greatly affects estimated utility emissions of mercury. For example, the waste combustion emissions were estimated using measured emission factors, but the coal combustion data were based on mercury measurements of the fuel. Furthermore, coal-fired power plant emissions were based on measurements of in-the-ground coal quality, not coal as burned at U. S. power plants. Measurements of in-the-ground coal quality overestimate mercury emissions because coal as burned at power plants is typically cleaned. This removes about 50 percent of the mercury before combustion. More recent estimates are discussed in the subsection, Mercury in Coal.

One important source of mercury not included in the MRI analysis is the roasting of ferrous and nonferrous ores whose minerals likely contain trace amounts of mercury. Even though the concentrations of mercury could be small, the amount of ore processed is substantial, and this processing could result in substantial mercury emissions, lowering the relative emissions from other sources. Furthermore, additional discrepancies exist that illustrate the uncertainty in emissions. The paint emissions listed in Table 2-2 of 13 metric tons per year for the United States contrast markedly with the diffuse mercury source input to the atmosphere estimated for the United States by Lindqvist et al. (1991) of 350 metric tons per year. In general, diffuse sources are not as well-characterized as point sources. Historical mercury use has not been assessed in the United States, but Sweden has shown that peak emissions occurred in about 1960 due largely to chlor-alkali plants (Lindqvist et al. 1991). Other emissions categories that affect the relative contributions of different sources include re-emissions of past anthropogenic mercury releases and mercury emissions from natural sources.

While total U. S. anthropogenic mercury emissions have been estimated to be on the order of 300 metric tons per year (Table 2–2), emissions from the 27 European states have been estimated at 726 metric tons per year (Pacyna et al. 1991). The former East Germany alone emitted about 330 metric tons per year.

Mercury in Fossil Fuels

The U. S. electric utility industry burns three major classifications of fossil fuels: coal, fuel oil, and natural gas. Figure 2-1 compares recently measured mercury emission factors with those in the literature. The recent measurements indicate that mercury levels in coal range from 0.02–0.25 ppm [corresponds to an emission factor range of 0.5–10 μg/MJ—micrograms per megajoule; multiply μg/MJ by 2.3 to obtain lb/10^{12} Btu]. Mercury levels in coal tend to be 1 to 4 orders of magnitude greater than in fuel oil and natural gas.

Mercury in Coal. Coal is the only fossil fuel for which sufficient data currently exist to perform detailed analyses of trace element concentrations, including mercury. Between 1973 and 1987 the U. S. Geological Survey (USGS) analyzed thousands of channel and core samples of coal for various coal quality parameters, including trace element content. These data form the National Coal Resources Data Set (NCRDS). Because channel and core samples in the NCRDS represent the entire height of a coal seam and include interbedded rock and minerals, they are representative of in-the-ground (not cleaned) coals.

Using the NCRDS, MRI (1993) estimated that coal-fired U. S. power plants emit 89 metric tons per year, or 94 percent of total power plant mercury emissions (Table 2-2). However, this approach will overestimate emissions for utilities burning eastern and midwestern coals because over 75 percent of these coals are cleaned or washed before combustion, reducing the concentration of many trace substances (USDOE 1993).

To develop information more representative of as-fired coals that have been cleaned, USGS and EPRI cooperated to screen and refine the subset of the NCRDS to be delivered to USEPA. First, they removed entries representing coal seams too thin or deep to be mined economically, as well as obvious samples of interbedded rock and minerals. Then, they developed algorithms to refine the screened data set (Akers 1993). These algorithms were based on published coal-mercury data from industry and EPRI research, and included material balances around several configurations of coal cleaning plants. Finally, they applied the algorithms to selected entries in the screened USGS data set to develop a refined data set more representative of as-fired coals (Akers 1994).

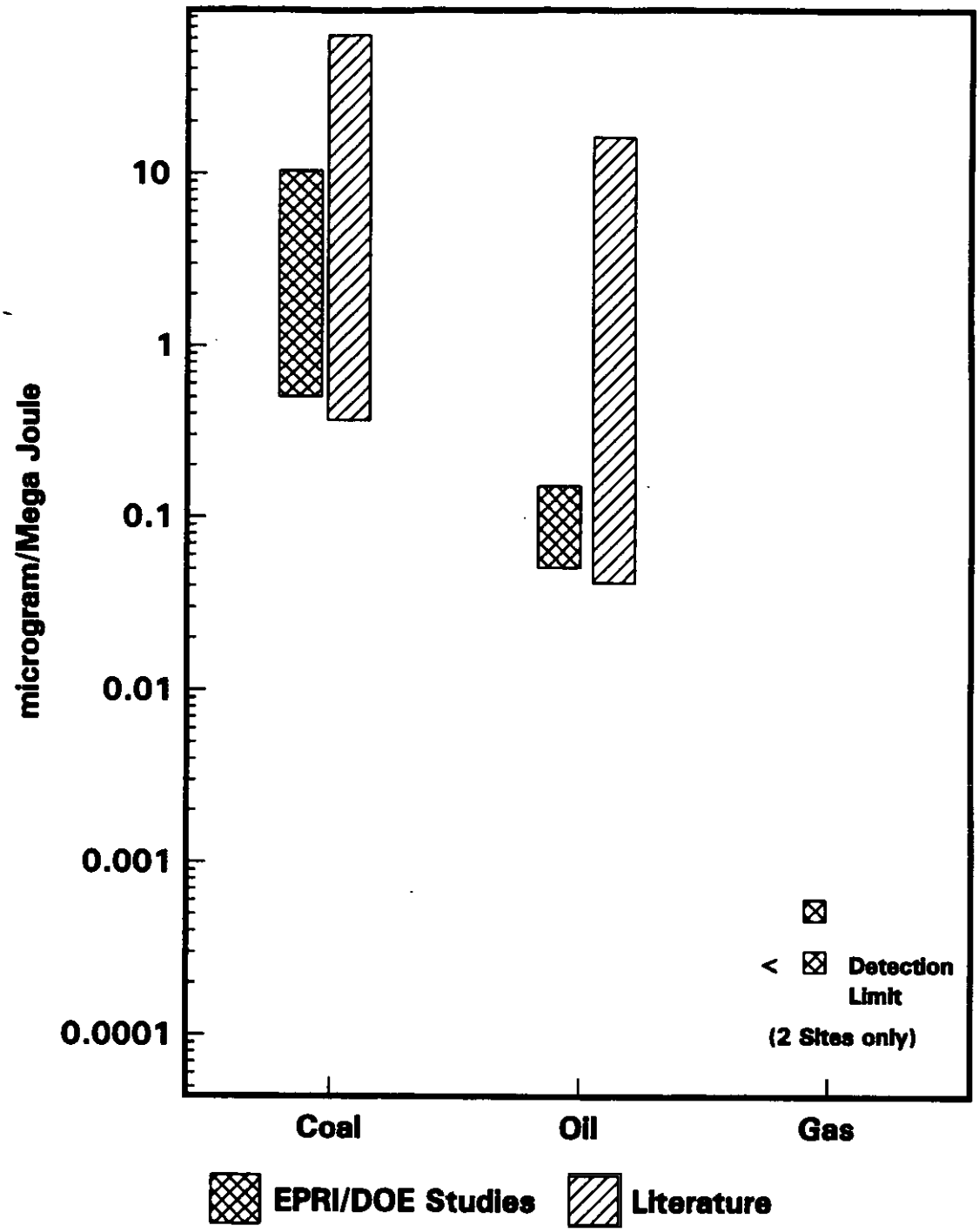

Figure 2-1
Improved sampling and analysis techniques show reduced uncertainty in mercury emission factors from fossil-fuel combustion

EPRI also analyzed 154 coal samples (31 were replicates) of as-fired or as-received coals (Bloom and Prestbo 1994; Baker 1994). The data set, including replicates, comprised 106 bituminous coal samples, 37 sub-bituminous samples, and 11 lignites, and represented the major coal seams (Baker 1994). A frequency distribution for the 123 as-fired coals shows that all but 11 percent had mercury concentrations below 0.15 ppm (Figure 2-2). The 123 samples averaged about 0.09 ppm, less than one-half of the concentrations used by MRI (1993) in their study. This compares closely to a mean value of less than 0.12 ppm in 21 non-U. S. (mostly European) coals (Bloom and Prestbo 1994).

The data shown in Table 2-3 indicate lower average mercury concentrations in as-fired coals as compared to the MRI (1993) calculation. These coals—represented in EPRI's Mercury in Coal study (Baker 1994) as well as EPRI's field measurements [the Field Chemical Emissions Measurement (FCEM) program of the Power Plant Integrated System: Chemical Emissions Study (PISCES)]—show concentrations about 50 percent lower than those of in-the-ground coals represented by USGS data. This result is supported by an analysis performed by USGS, who estimated that about 50 percent of the mercury in USGS coal samples would have been removed had the coal been washed. USGS further estimated that thorough washing removed 70 percent of the mercury in coal samples provided via EPRI (Finkelman, 1994).

Mercury in Fuel Oil and Natural Gas. At the EPRI fuel oil sites, mercury was initially not detected in most of the fuel oil samples. To obtain lower detection limits, INAA (instrumental neutron activation analysis) was used instead of CVAAS (cold vapor atomic absorption spectrophotometry). Using INAA, with standards and blanks to ensure no change in mercury, mercury was measured in the 0.002–0.008 ppm range (0.04–0.13 μg/MJ). Emission factors are 1 to 2 orders of magnitude less than in coal. Natural gas samples were analyzed for mercury at only two sites. Mercury was detected at 0.02 $\mu g/m^3$ (0.00056 μg/MJ) at one site and was below the detection limit (0.01 $\mu g/m^3$) at the other site.

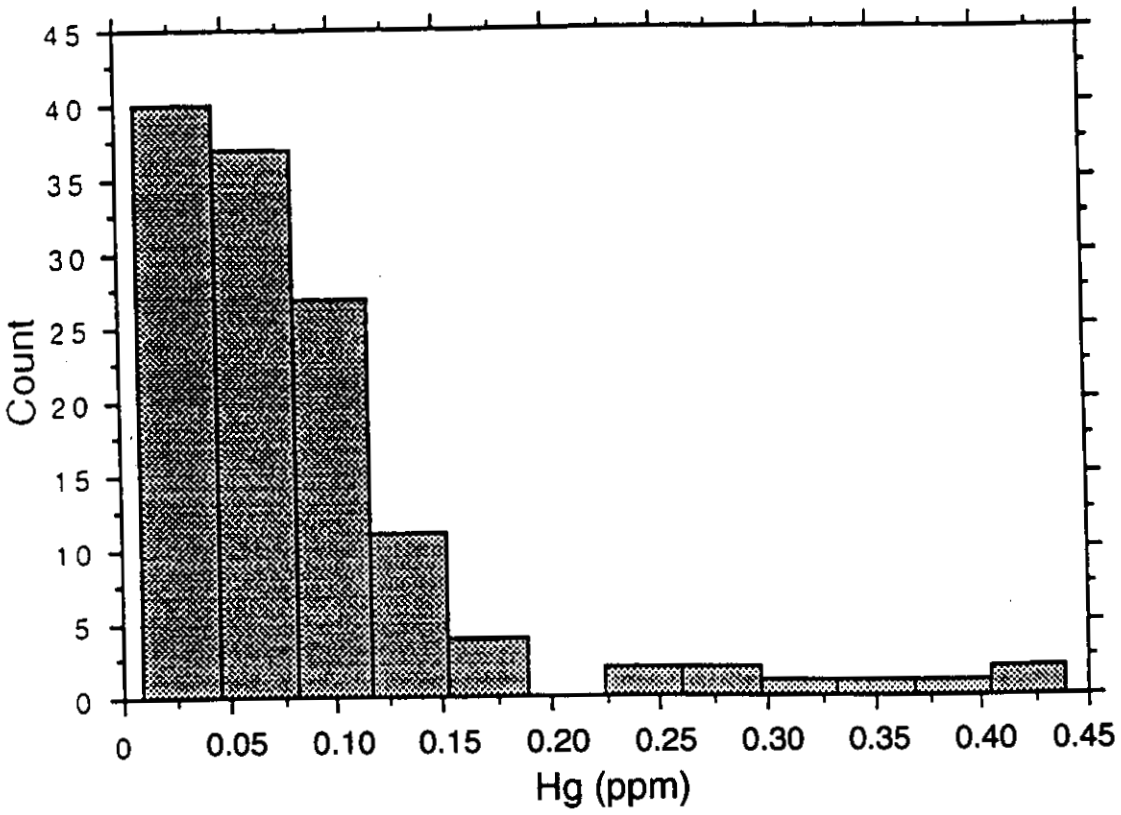

Figure 2-2
Frequency diagram for the concentration of mercury in 123 different as-fired U. S. coals (Bloom and Prestbo 1994)

Table 2-3
Comparison of Mercury Concentrations in Coal Samples

	USGS[a]	Revised USGS Data Set[b]	EPRI Mercury Study[c]	PISCES FCEM[d]
Bituminous Coals				
Average (ppm)	0.21	0.19	0.09	0.12
No. of Samples	3,500	2,300	106	47
Bituminous Subsample: Pittsburgh Seam Coal				
Average (ppm)		0.21	0.10	
No. of Samples		29	10	

[a]MRI 1993
[b]Akers 1993
[c]Baker 1994
[d]EPRI 1994

Emissions From Fossil-Fuel-Fired Power Plants

Measurement of trace elements like mercury require sensitive and robust methods to detect the low concentrations in flue gases and stack emissions. EPRI contractors have used USEPA-recommended methods and, in cooperation with others, have sponsored development of new methods as a way to ensure the most accurate estimates of power plant emissions. For example, Bloom (1992b) has shown the need for clean techniques when measuring fuels and emissions.

EPRI and USEPA have conducted joint tests for formal evaluation of USEPA Draft Method 29 (multi-metals method) for measurement of mercury and other metals in the stack gas of a coal-fired power plant. The tests were performed according to the analyte spiking procedure of USEPA Method 301 protocol for the field validation of stationary source emission measurements. Also, selected field samples collected by the contractors were split for inter-calibration analysis. Several other mercury measurement methods were employed for comparison with Method 29 results. These included USEPA Method 101A, the Hazardous Element Sampling Train (HEST), sorption on iodated carbon traps, and the solid sorbent series method using soda lime traps followed by iodated carbon traps. Flue gas samples for all the methods were collected in each of eight runs, using quadruplet sampling trains located in adjacent ports in the vertical run of a duct leading from the outlet of an electrostatic precipitator (ESP) to the stack. Based on the analyses of the samples collected by the EPRI contractor, USEPA Draft Method 29 appears to meet the precision and bias criteria of USEPA Method 301 protocol. The results further indicate fairly good agreement between mercury measurements by the different methods as shown in Figure 2-3. The results of this study indicate that estimates of mercury emissions can be obtained with a variety of carefully designed protocols that are properly applied. Also, the results show the wide variation in day-to-day concentrations of mercury in flue gases, supporting the need for integrated long-term studies.

Recent field measurements have been conducted by EPRI, the U. S. Department of Energy (USDOE), and individual utilities to characterize trace substance emissions from utility fossil-fuel-fired power plants. Tests have been conducted at plants burning bituminous, sub-bituminous, and lignite coals—as well as fuel oil and natural gas. In addition, these field tests have evaluated the various SO_2

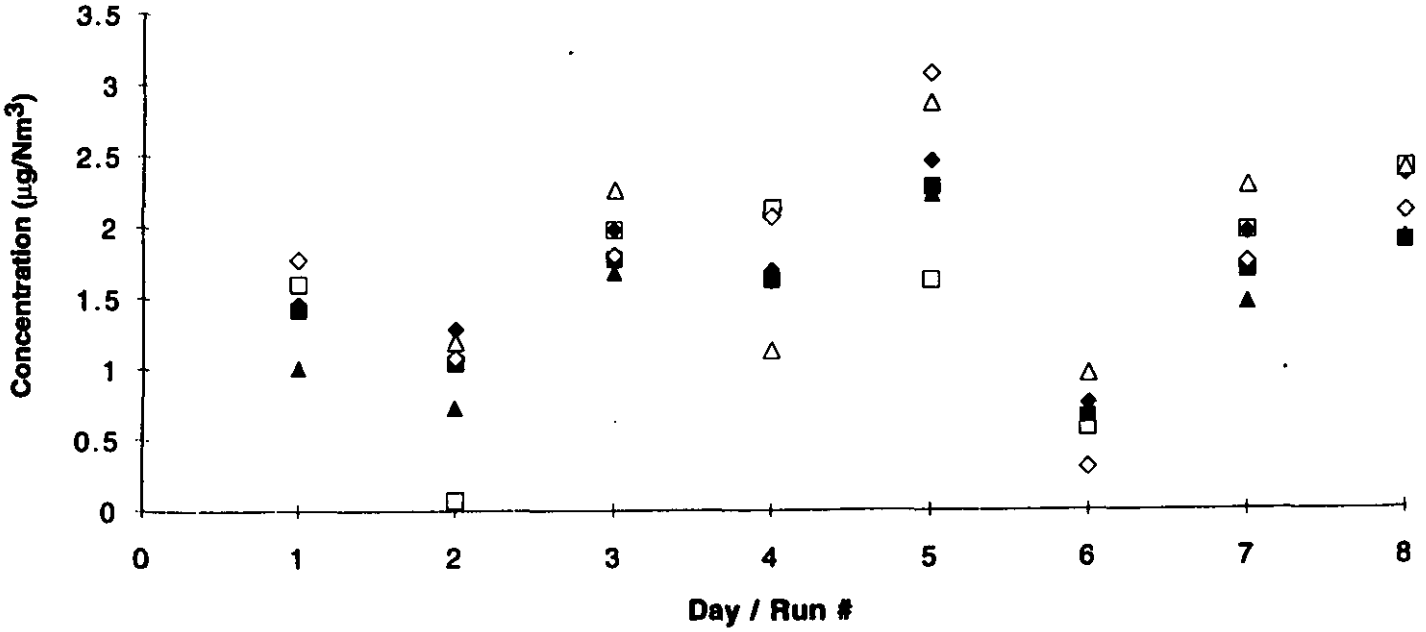

Figure 2-3
Stack gas mercury concentrations measured by six different methods. Concentration is expressed in normal (25°C at 1 atm) cubic meters (Nm^3). Each symbol represents a different laboratory and sampling/analysis method.

and particulate control technologies as potential control technologies. The results of these field studies provide a reasonable estimate of expected mercury emissions from utility power plants. However, because of the low concentrations of mercury and the chemical and physical nature of mercury, sampling and analysis have generally been difficult and have yielded significant uncertainty about total mercury emissions as well as the chemical form of the mercury in flue gases.

The measured mercury emission factors for fossil-fuel-fired power plants are summarized in Tables 2-4a and 2-4b. Because coal, oil, and gas have different levels of mercury in the fuel, it makes sense to discuss mercury emissions for each different fuel type. In addition, the particulate and SO_2 control systems may reduce mercury emissions. All coal-fired power plants employ some type of particulate control technology and some plants include a flue gas desulfurization (FGD) system. Only a fraction of the oil-fired plants have particulate control devices (results from oil plants with particulate controls are not included in Table 2-4b), and no oil-fired plants use an FGD system. Gas-fired power plants do not have controls because their emissions are so low.

Coal-Fired Power Plants. Because mercury is generally present in the vapor phase at particulate control temperatures (120–150°C; 250–300°F), mercury is not consistently well-controlled by an ESP or fabric filter. Mercury reduction varies among the test sites—and includes some sites where the outlet mercury is greater than the inlet coal mercury. This is likely due to sampling and analytical variability. By contrast, mercury reductions greater than 60 percent were measured at several sites, with the mercury being accounted for in the collected fly ash. However, an explanation could not be found why certain plants or coals yield more mercury in the particulate phase. The mean removal efficiency for all coal-fired plants with dry particulate controls is about 30 percent.

Mercury removal efficiencies for combined ESP (or fabric filter) and wet FGD systems have also been highly variable and difficult to correlate with the FGD design, coal composition, or measured mercury valence (oxidation) state. The mercury removal efficiencies for ESP/FGD systems range from as low as 0 percent to as high as 90 percent. Research has shown that oxidized mercury appears to be removed to a greater degree than elemental mercury. However, a

Table 2-4a
Mercury Emission Factors for Coal-Fired Power Plants[a]
(μg/MJ)[b]

	Dry Particulate Control[c]			Combined Particulate and FGD Systems[d]		
Coal Type	Range	Number	Mean	Range	Number	Mean
Bituminous	1.7–6	15	2.8	0.3–1.5	4	0.6
Subbituminous	< 0.2–4.4	7	1.4	0.8–3.6	3	2.1
Lignite	4.5–6.4	2	5.5	4.3–5.2	2	4.7

[a]Based on recent field measurements as part of EPRI's PISCES program and USDOE's field test efforts. Results obtained using the USEPA multi-metals train (USEPA Draft Method 29).
[b]Multiply by 2.3 to convert to $lb/10^{12}$ Btu.
[c]This includes both ESPs and fabric filters.
[d]This includes both wet and dry FGD systems.

Table 2-4b
Mercury Emission Factors for Oil- and Gas-Fired Power Plants
(μg/MJ)[a]

	No Controls		
Fuel Type	Range	Number	Mean
Fuel Oil	0.07–0.6	5	0.20[b]
Natural Gas	<0.00026 –0.00056	2	0.00034[c]

[a]Multiply by 2.3 to convert to $lb/10^{12}$ Btu.
[b]Mercury stack emission results for oil plants were assumed log normally distributed. This emission factor would also be an appropriate estimate for oil-fired plants with ESPs.
[c]Natural gas emission factors are based on analyses of mercury in the inlet natural gas, assuming that all mercury was emitted in the flue gas.

relationship has not been developed to predict flue gas concentrations of oxidized mercury or mercury removal efficiency by FGD systems. The mean mercury removal efficiency for the combined ESP/FGD system was about 45 percent. EPRI, USDOE, and other organizations are continuing work in this area to better understand the influence of mercury chemistry on removal mechanisms.

The mercury emission results for coal-fired plants are presented in Table 2-4a for the three major coal classifications as well as the two general air pollution control technologies—dry particulate control (ESP and fabric filters) and FGD systems (spray dryer absorbers and wet FGD systems). The database is quite small for most of the categories, and this should be considered when applying the results in Table 2-4a. For example, the average mercury emission factor for units burning subbituminous coal does not make good engineering sense: the average mercury emission factor for units with only particulate controls was actually less than the average mercury emission factor for the combined particulate and FGD systems. This inconsistency was probably due to the large uncertainty and the small number of units studied. Two of the FGD systems had less than 25 percent mercury removal, while four of the dry particulate controls sites achieved greater than 65 percent removal. Only two plants that burn lignite coal were tested; thus, the confidence interval around the average emissions for these units is broad. One plant burned a North Dakota lignite, while the other burned a Texas lignite.

Speciating Emissions From Coal-Fired Power Plants. Mercury in flue gases may be present in several valence states—mainly elemental [Hg(0)] and oxidized [Hg(II)]. This has significance for several reasons. The chemical form of the mercury may affect the degree of removal, as well as atmospheric fate, health effects, and risk assessment.

EPRI has applied two sampling methods to quantify mercury emissions—the USEPA multi-metals method (Draft Method 29) and the mercury speciation adsorption (MESA) method (Prestbo and Bloom 1995). The multi-metals method uses two sets of impingers to capture the vaporous mercury. The first set of impingers consists of nitric acid/peroxide (HNO_3/H_2O_2) and the second set consists of potassium permanganate ($KMnO_4$). The multi-metals method was

not designed to speciate mercury, but it has been suggested that only oxidized mercury is captured in the HNO_3/H_2O_2 impingers; thus all remaining mercury (expected to be elemental mercury) is captured in the $KMnO_4$ impingers. The MESA method works in a similar fashion, except it uses a different medium to capture the mercury. This method employs solid sorbent traps—consisting of soda lime and iodated carbon—to capture the oxidized and elemental mercury, respectively. Both methods are still experimental for mercury speciation.

The oxidized mercury concentrations from the multi-metals method (assuming that the mercury captured in the first impinger is oxidized mercury) generally appear to be higher than the oxidized mercury concentrations from the MESA method. Figure 2-4 compares the measured levels of oxidized mercury for these two methods. Because the two methods generally agree for total mercury (although some sites have large discrepancies), it would appear that one or both methods do not accurately quantify oxidized mercury. The purpose of the HNO_3/H_2O_2 impingers in the multi-metals method was to capture volatile trace metals (such as arsenic, chromium, and nickel), and not to selectively capture oxidized mercury. Thus, some elemental mercury may be captured in those impingers. Another possibility is that the MESA method does not efficiently capture all the oxidized mercury. Recent research has shown that the oxidized mercury capture efficiency in the soda lime traps is a function of the sampling temperature. Some of the early runs were not conducted at optimum temperatures and it is possible that some of the oxidized mercury was not captured in the soda lime traps, allowing the method to underestimate oxidized mercury. Samples obtained after trap temperature was standardized, however, still show a lower percentage of mercury than the multi-metals method. This suggests that the MESA method actually estimates a lower percentage of oxidized mercury. In addition, the MESA method was not designed to sample particulates isokinetically; thus the method does not obtain a representative particulate sample. However, this fact may be relatively unimportant since particulate-phase mercury has been measured at only select sites and, with a few notable exceptions yet to be explained, is generally below the detection limit.

For dry particulate controls (ESPs and fabric filters), the mean percentage of oxidized mercury removal based on the MESA and multi-metals methods

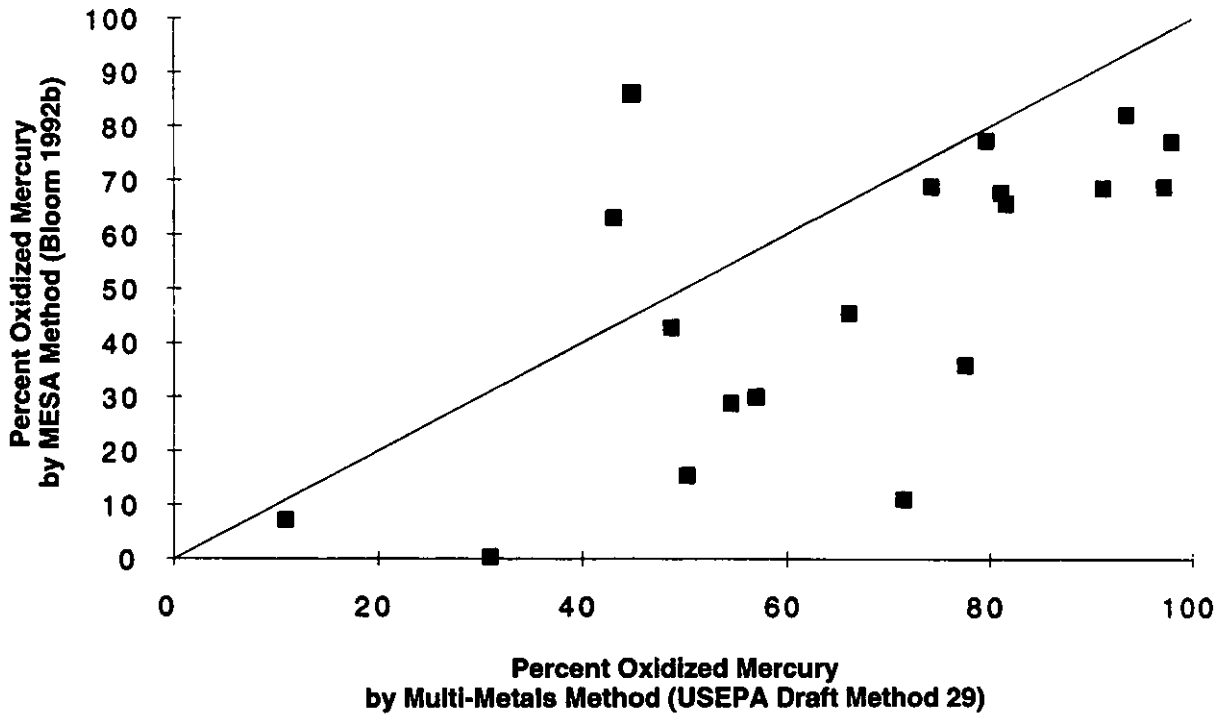

Figure 2-4
Comparison of levels of oxidized mercury measured by two different methods

was 55 and 70 percent, respectively. The mean percentage of oxidized mercury removal for particulate removal systems and for FGD systems was 25 and 45 percent, respectively.

The ratio of oxidized to elemental mercury in flue gas is potentially a function of coal type and composition, as well as flue gas conditions. Data were insufficient to determine any definitive correlations to predict levels of oxidized and elemental mercury; thus, utilities must rely on direct measurements. Figures 2-5 and 2-6 compare the concentrations of oxidized mercury [in $\mu g/Nm^3$—micrograms per normal (25°C, 1 atm) cubic meter] as a function of chloride in the coal for both the multi-metals and MESA methods. The results from the MESA method show a trend toward higher oxidized mercury concentrations with increasing chloride content in the coal. This trend was not apparent with the mercury speciation results from the multi-metals train, where some results appear to be "outliers" and there is significant scatter among the data. This may be due to other factors that affect mercury speciation which have not been completely evaluated. In addition, some of the scatter may be due to process variability as well as variability introduced by sampling and analytical methodologies.

Oil-Fired Power Plants. As part of the State of California AB2588 study, utilities attempted to measure mercury (as well as other trace substance) emissions from oil-fired power plants. The method detection limits were not sufficient to quantify the concentrations of mercury in either the fuel oil or the stack. At EPRI field sites, more sensitive analytical methods were used to achieve lower detection limits in both the fuel oil and stack measurements: instead of CVAAS, INAA was used to analyze the fuel oil.

With INAA, mercury in fuel oil was measured in the 0.002–0.008 ppm (0.04–0.13 $\mu g/MJ$) range. Assuming that all the mercury in the fuel oil is emitted through the stack, the mercury concentration in the flue gas would be approximately 0.1–0.4 $\mu g/Nm^3$. Because the mercury method detection limits (flue gas measurements) have ranged from 0.1–0.5 $\mu g/Nm^3$, these low levels of mercury have led to difficulties in quantifying the mercury concentration in flue gas for oil-fired power plants. Mercury stack emissions have ranged from 0.2–1.7 $\mu g/Nm^3$ (0.07–0.6 $\mu g/MJ$). The measured emission levels have been highly

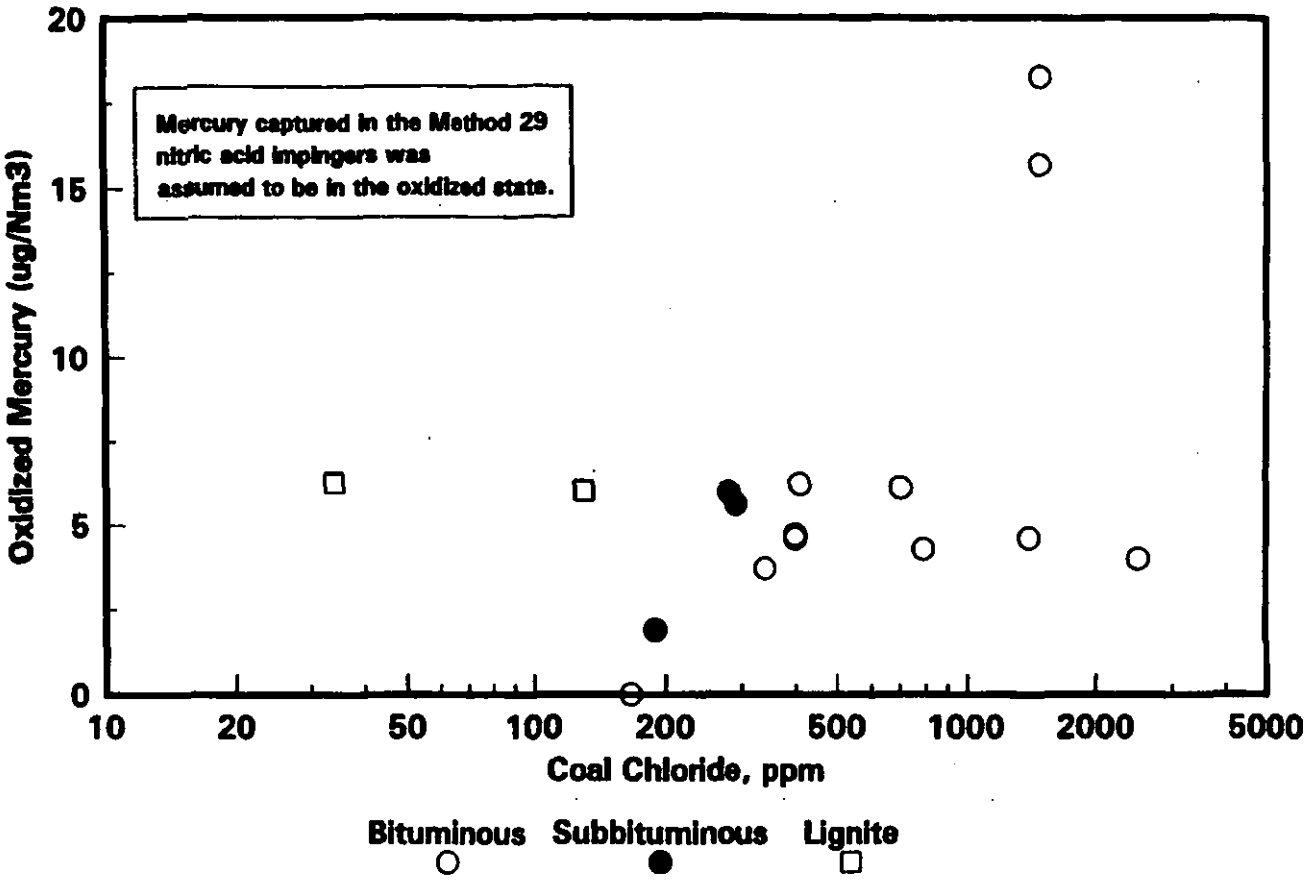

Figure 2-5
The multi-metals method (USEPA Draft Method 29) as an estimator of oxidized mercury shows no relationship with coal chloride concentrations

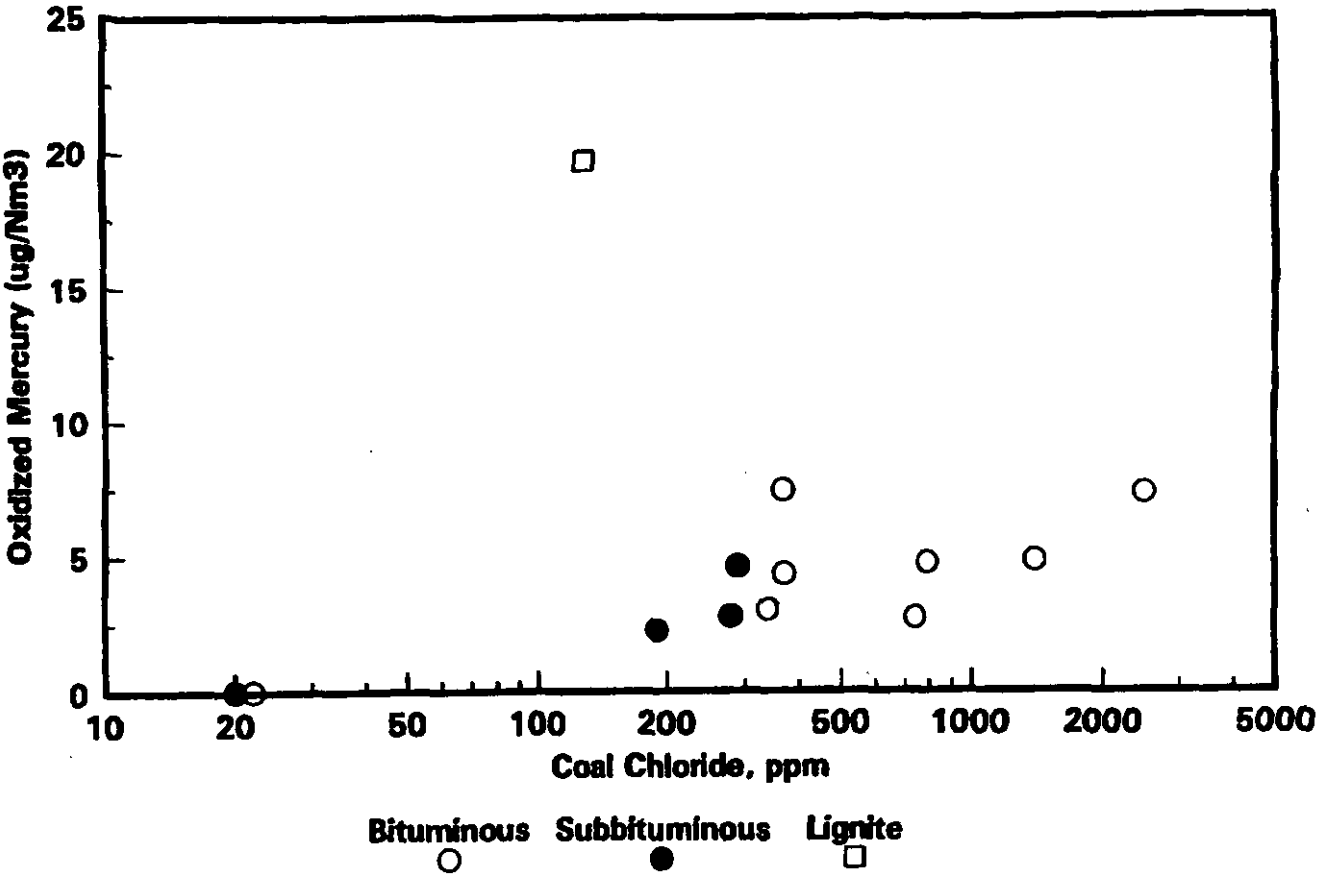

Figure 2-6
The MESA method suggests that oxidized mercury does relate to coal chloride concentrations

variable and have sometimes been much greater than the inlet fuel levels. The trace metals emissions data distribution from fuel oil plants appears to be log normal; thus a geometric mean for mercury appears to be more appropriate than an arithmetic mean. A geometric mean reduces the emphasis on the very high measurements which are likely due to sampling and analytical difficulties. The geometric mean was 0.2 μg/MJ; this emission factor is conservative since it is higher than one based on the average mercury levels in the fuel oil.

Gas-Fired Power Plants. Field tests were conducted at two electric utility gas boiler plants. Mercury was not detected in the stack at either site. The detection limit was about 0.5 $\mu g/Nm^3$ (0.17 μg/MJ) which was three orders of magnitude higher than the expected levels based on the natural gas fuel analyses. The mercury concentration in the natural gas was measured at 0.00056 μg/MJ (near the detection limit) at one boiler and was below the detection limit of 0.00027 μg/MJ at the other. This yields an average of 0.00034 μg/MJ (assuming half of the detection limit for the nondetected value). The best estimate for mercury emissions would be to use the natural gas analyses and assume all the mercury is emitted through the stack.

Summary: Mercury Emissions From Fossil-Fuel-Fired Power Plants. Based on these recent and relatively extensive fuel and stack measurements, two approaches, in addition to that of MRI (1993), were employed to estimate total electric utility emissions from fossil-fuel-fired power plants.

The more detailed approach incorporated the coal purchases for each individual power plant (EIA/USDOE 1993) and average mercury concentrations based on EPRI's Mercury in Coal study (Baker 1994; Bloom and Prestbo 1994) to estimate fuel mercury. Average removal efficiencies as calculated from the recent EPRI/USDOE studies were then applied to estimate mercury emissions at each power plant. The individual mercury emissions were then summed to yield about 39 metric tons per year for U. S. electric utility coal-fired plants. Mercury emissions from oil- and gas-fired utility plants based on heat input data (MRI 1993) and the average emission factors (Tables 2-4a and 2-4b) were less than 0.3 metric tons per year.

An alternative approach applied the average emission factors for each combination of fuel type and control technology from Tables 2-4a and 2-4b and the total heat input data for the U. S utility industry. The Utility Data Institute Power Statistics Database (1989) was used to calculate a weighted emission factor based on the capacity for each combination of fuel type and control technology. This simplistic approach is similar to the methods used by other surveys such as that of MRI (1993), and yields a mercury emission estimate similar to that of the detailed approach described above—41 metric tons per year for the coal-fired power plants.

The estimated mercury emissions by these two approaches are compared with MRI's estimates in Table 2-5. Both approaches provide estimates of utility mercury emissions on the order of 40 metric tons per year for the 1990 period—which is less than half of MRI's estimate. Thus, the revised U. S. total for mercury from all sources would be on the order of 250 metric tons per year (MRI 1993)—assuming the existing mercury emission data for all other sources were correct. The contribution from power plants would be no more than 16 percent of the U. S. total, and would decrease as newer generation technologies come on-line.

Table 2-5
Total Mercury Emissions From U. S. Fossil-Fuel-Fired Power Plants
(metric tons per year in 1990)

	MRI[a]	EPRI ESTIMATES	
Fuel Type		**Fuel/Removal Efficiencies Methodology[b]**	**Emission Factors Methodology[c]**
Coal	89	39	41
Fuel Oil	3.8	0.3[d]	0.3
Natural Gas	not estimated	0.001[d]	0.001
TOTAL	93	39.3	41.3

[a]Midwest Research Institute (1993).
[b]This methodology employs recent data on mercury in fuels and average removal efficiencies for ESP/fabric filters and ESP/FGD systems for coal-fired power plants.
[c]This methodology employs average emission factors and heat inputs. The emission factors were based on actual measured mercury concentrations in flue gases. This calculation uses the same approach as MRI (1993), but uses the recent emission factors in Table 2-4a for each coal type.
[d]Based on recent emission factors in Table 2-4b.

Section 3
ATMOSPHERIC MERCURY

Mercury in the Atmosphere

Because the presence of trace substances such as mercury in the atmosphere has become a concern only recently, far less is known about their sources, atmospheric reactions, ambient concentrations, and deposition rates than about substances like sulfur dioxide, particulate matter, and ozone where sustained research over the last two decades has created mature disciplines devoted to their geochemical cycling. Nonetheless, the experience with these more researched substances has created a rich repertoire of experimental methods, simulation methods and, above all, approaches for conceptualizing and structuring problems that are directly transferable to potentially hazardous trace substances.

This transferability of methods and approaches is of particular value when dealing with atmospheric mercury, which offers numerous challenges in understanding its composition and fate. Mercury is present in the atmosphere at relatively small concentrations (at levels of nanograms and picograms per cubic meter). It is found in both the gaseous and particulate phases, and in different valence states. It can both deposit and emit from surfaces, at concentrations that challenge our limits of detection. Mercury is mobilized from natural sources by human and natural processes and activities that are widely distributed across the globe. The historical record of anthropogenic effects on the natural cycle of mercury has not been well-characterized. In this Section, we summarize the current understanding of the occurrence, transformation, transport, and fate of atmospheric mercury.

Global Perspectives

Three spatial scales are relevant when addressing atmospheric mercury: global, regional, and local. (1) The global scale is dominated by Hg(0) and fine particulate Hg(II) (particles less than 1 micron in diameter) that have escaped local or regional scavenging. (2) The regional scale encompasses an area that requires a transport time of more than one diurnal cycle (from 100 to about

2000 km from a source). It describes areas sufficiently remote or distant from large emission sources that concentration fields are rather homogeneous, lacking steep gradients. (3) The local scale describes the area within which emissions can travel in one diurnal cycle (generally within 100 km of a source) and where concentration gradients generally are steep.

Whether mercury emissions enter the global cycle or deposit locally or regionally depends on the species emitted and the transformations occurring in the atmosphere. Natural and manmade emissions into the atmosphere are likely to enter the global cycle if they are in the form of Hg(0), but are more likely to deposit locally or regionally if they are oxidized [Hg(II)] (see Lindqvist et al. 1991). Supporting this view, mercury in the atmosphere tends to occur almost exclusively as Hg(0). Oxidized forms [Hg(II) and methylmercury] typically constitute less than 2 percent of the total concentration in air (Fitzgerald 1986, 1989). Virtually all deposition is oxidized forms (Fitzgerald et al. 1991).

Oceanic emissions to the atmosphere are Hg(0) (Fitzgerald 1986). Land and water emissions from reducing processes (whether biotic or abiotic) are largely Hg(0). Anthropogenic sources emit different mixes of Hg(0) and Hg(II). The oxidized forms [Hg(II)] may become associated with particles prior to, or soon after, emission. These particles will have varying atmospheric residence times depending on particle size, wind speed, cloud encounters, and other atmospheric conditions. Gaseous reduced mercury [Hg(0)] must be transformed to oxidized mercury [Hg(II)] to contribute substantially to mercury deposition. Apparently, gaseous Hg(II) has rapid dry deposition, and the lack of measurements has forced modelers to assume a dry deposition velocity similar to that of nitric acid (HNO_3). Moreover, mercury particle formation is poorly understood. Lindqvist et al. (1991) argue that soot particles play a strong role in cloud water transformations of Hg(0) to Hg(II), by sequestering Hg(II) on particles and preventing reduction back to Hg(0). The formation of particulate mercury may be a major key to understanding the contribution of local and regional sources to the global cycle. Tennessee Valley Authority (TVA) researchers are measuring mercury species in power plant plumes to provide results that will strengthen modeling of atmospheric emissions.

No broad-scale studies have accurately apportioned sources of mercury. The most recent depiction of the global mercury cycle (Mason et al. 1994) shows mercury exchange to be on the order of 2000 metric tons per year between ocean and atmosphere, and 3000 metric tons per year between land and atmosphere. This cycle assumes a steady state of 5000 metric tons in the atmosphere and an atmospheric residence time of 1 year. This picture differs slightly from previous analyses (Fitzgerald 1986, 1989; Lindqvist et al. 1991; Porcella 1994). The analysis by Mason et al. further assumes that 1000 metric tons per year come from nonanthropogenic (natural) sources and that half of the global anthropogenic emissions, or 2000 metric tons per year, become part of the global atmospheric cycle. The remaining fraction of the global cycle (40 percent) represents re-emissions of previously deposited mercury. As described in Section 2 (see Table 2-1), estimates of anthropogenic input vary considerably, but estimates of natural mercury evasion from the land are even more inaccurate and in present day budgets usually are determined by difference.

Open-ocean input of mercury to the atmosphere is the most credible estimate in the global budget (Fitzgerald 1986, 1989). In these studies, open ocean air concentrations varied between 1-2 ng/m^3, with the southern hemispheric concentrations being about half those of the northern hemisphere. Fitzgerald (1986) suggested that most anthropogenic sources were located primarily in the northern hemisphere, accounting for the observed difference in air concentrations because the atmosphere does not mix rapidly across the equator. A compilation of northern hemispheric concentrations measured at several locations over the land surface shows that air concentrations of gaseous mercury are remarkably uniform, but rain concentrations and particulate concentrations are less so (Table 3-1). These results show that particulate concentrations over land are considerably greater (by a factor of 10–30 times) than those over the open ocean, and that concentrations over midcontinental U. S. sites in nonurban areas vary within a factor of 2–3. The largest particulate mercury concentrations occur in the Nordic sampling network, and apparently reflect large regional sources.

Land sources have been determined by difference, equaling total minus oceanic and anthropogenic sources. To make the overall global mercury balance credible, independent estimates of evasion from land surfaces are needed that are

Table 3-1
Global Estimates of Mercury Deposition, and Atmospheric and Rain Concentrations

LOCALE	TOTAL MERCURY				
	Atmospheric		Rain	Wet Deposition	Reference
	Gas-phase ng/m^3	Particulate ng/m^3	ng/l	$\mu g/m^2 yr$	
North Pacific	1.77	<0.002	9	10	Fitzgerald et al. 1991
Wisconsin	1.57	0.022	10.5	6.8	Fitzgerald et al. 1991
Tennessee	2.15	0.030	10.7	14.2	Lindberg 1994
Nordic	2.5–2.8	0.060	18	4.5–8.0	Iverfeldt 1990, 1991
Florida	1.64	0.0015–0.008	11	13–25	Landing et al. 1994
Michigan	—	—	—	6.1–9.1	Keeler 1994
New York	—	0.051–0.089	—	—	Olmez et al. 1994
Ontario	—	—	10	10	Mierle 1990

based on accurate methods. Furthermore, one must assume that at least part of the oceanic and terrestrial inputs to the atmosphere is the re-emission of previously deposited mercury. The inaccurate estimates of global flux from human activities and land evasion add considerable uncertainty to the global cycle of mercury and any estimates of global contributions to risk from fish consumption.

Research from various parts of the northern hemisphere indicates that ambient concentrations of mercury are approximately the same everywhere but next to point sources, and are remarkably similar within each medium (that is, rainfall, dry deposition, and air concentration). This indicates a global-scale background occurrence of mercury and hence, the importance of developing a global model to constrain regional source areas (for example, Eastern Europe, Asia, northern and southern hemispheres) in relation to deposition patterns. A key assessment objective will be the relative contribution of U. S. utility emissions to global atmospheric mercury. Since mercury is mobilized from its terrestrial sources with and without human intervention, source-attribution studies need to establish how much of the atmospheric mercury produced "naturally" comes from deposits stemming from human activities and how much is naturally cycled.

Temporal Perspectives

Two important assumptions underlie the global mercury mass balance calculations. First, the system is assumed to be at steady state; that is, concentrations and fluxes are not changing appreciably. Second, the global anthropogenic emission estimates are assumed to be reasonable and relatively constant for the last decade. To evaluate whether mercury levels in the atmosphere are at steady state, historical estimates are needed. Some investigators have used sediment and peat cores to estimate present and pre-industrial mercury deposition as a predictor of atmospheric concentrations. These measurements indicate that the range of present-day mercury deposition is 2 to 5 times greater than pre-industrial deposition (Lindqvist et al. 1991). However, these before-after data do not provide any information on the intervening temporal pattern.

The temporal pattern of deposition inferred from cores taken in northern Minnesota does not suggest a monotonic increase in atmospheric mercury concentrations (see data of Benoit et al. 1994; Swain et al. 1992). Results from the Minnesota peat cores suggest that the peak deposition occurred prior to 1970 (Figure 1, Zillioux et al. 1993). Engstrom (1994) obtained similar patterns in lake sediment cores.

Previous uses of mercury may have introduced more mercury than present activities. Nriagu (1993a,b) argues that gold and silver extraction from the middle of the 16th to the end of the 19th centuries introduced nearly 160,000 tons of mercury to the atmosphere. This is comparable to the assumption by Mason et al. (1994) of 200,000 metric tons entering the global atmosphere since about 1890. The Virginia City mines of Nevada alone used more than 7000 metric tons of mercury in gold extraction prior to the turn of the century, and only about half has been accounted for by soil and water measurements (Cooper et al. 1985).

More recently, atmospheric inputs of mercury could have occurred from other major uses including gold mining in the Amazon River basin (Nriagu et al. 1992) and nuclear weapons manufacturing (for example, Union Carbide 1983). Other emissions, resulting from mercury use by U. S. battery manufacturers, fungicides in agriculture, paints, and chlor-alkali plants, have declined in the last decade (Neme, 1990). In addition, Lindqvist et al. (1991), clearly show that Swedish mercury emissions to the atmosphere peaked prior to 1960. Assuming that many industrialized countries used mercury in a similar fashion, atmospheric mercury emissions could have peaked prior to the most recent decades. These estimates have been obtained only from local geographic areas, making it difficult to extrapolate from them to the global atmospheric mercury cycle. Therefore, additional assessments are needed. Although Slemr and Langer (1992) provided data to show that mercury in the air had increased in mid-latitudes of the Atlantic Ocean, the evidence is equivocal because long-term continuous records at permanent stations are not available.

Concentrations in biota measured over time may be more indicative of the temporal pattern of mercury deposition. These results may be more relevant than historical air measurements, especially since fish represent the major potential source of human exposure. Most marine fish monitored show no time

trends in mercury concentrations for same-size fish (NAS 1978; Cramer 1994). Mercury concentrations in fresh-water fish measured over different time intervals have been reviewed, and only one increasing trend has been reported, that for Minnesota (Swain and Helwig 1989). Others have shown no change or a decline in fish mercury concentrations. As Swain and Helwig note, their data are too sparse to draw conclusions and are appropriate only for formulating hypotheses about mercury. If atmospheric deposition has decreased in Minnesota since the 1960s (see Zillioux et al. 1993; Benoit et al. 1994), it is surprising that a decreasing trend in fish mercury has not been observed. One possibility is that a large part of the mercury supply comes from watershed soils, and soils have not changed substantially from the effects of anthropogenic sources (Mason et al. 1994). Furthermore, as will be shown later in Section 4 when discussing mercury accumulation in fish, covarying factors such as water-quality variables can confound such reported trends, and research must account for these factors to obtain more accurate trends.

Newton et al. (1993) provide data from 25 years of measuring liver mercury concentrations in two raptor species and a fish-eating grey heron, collected from many locations in the United Kingdom. These data show that peak concentrations occurred prior to 1970. The precipitous decline in the early 1970s suggests that local sources, such as agricultural uses of mercury fungicides, may have led to elevated mercury levels two to three decades ago. The bird liver results are consistent with the patterns obtained from the Minnesota peat cores and the Swedish emission patterns, strongly suggesting that mercury deposition has decreased from peak levels in the 1960s. McIntyre et al. (1993) concluded that similar trends existed for mercury levels in eggs of the Common Loon collected from New York and New Hampshire.

Chemical Transformations

Chemical and physical transformations govern the atmospheric behavior of mercury and its environmental fate. Chemical form governs the phase state (that is, whether a substance exists in the gas, liquid or solid state); the valence state [for example, divalent Hg(II) and elemental Hg(0)]; and the chemical bonding (for example, inorganic versus organic compounds). These attributes of mercury

bear on its transport, its rate of removal from the atmosphere (that is, deposition), and thus, its zone of influence.

The wet and dry removal rates of mercury are substantially different for particulate and gaseous states. Elemental mercury is present primarily as a gas. Mercuric compounds can be present in the atmosphere either as gases [for example, $HgCl_2$, $Hg(OH)_2$], in the aqueous phase [for example, $HgCl_2$, $Hg(OH)_2$ or mercuric sulfites], or in the solid particulate phase (for example, HgO or HgS). In addition, mercury combines with organic compounds to form organomercuric compounds such as methylmercury. Although methylmercury is present at trace levels in air and rainfall (Bloom and Watras 1989), its source remains unclear. Typical atmospheric concentrations of major mercury species reported in the literature are given in Table 3-2.

A summary of the known major chemical processes that govern the atmospheric fate of inorganic mercury is given in Table 3-3. Lindqvist et al. (1991) considered reactions 7, 8, and 9 to be the important aqueous-phase (cloud-water) reactions that would dominate forward and back transformations of mercury. However, reaction 12 may dominate overall reaction rates due to mass transfer limitations. Further work is needed to clarify chemical interactions and transformations of mercury, as suggested by Seigneur et al. (1994). Our current understanding of the predominant phase and oxidation states in the atmosphere, based on literature review and simulations of mercury chemistry (Seigneur et al. 1994), is summarized in the following paragraphs.

- Gaseous elemental mercury is the dominant form of mercury in the atmosphere. The gas-phase reactions of mercury tend to convert Hg(0) to Hg(II). The fastest identified reactions include those with hydrogen peroxide, chlorine (over the oceans), and ozone. However, measurement uncertainties are associated with the kinetics of these reactions and their rates are upper limits; for example, recent laboratory data suggest that the gas-phase ozone reaction actually may be relatively slow. Based on the existing kinetic data, the half-life of Hg(0) is estimated to be on the order of hours (for reactions with chlorine (Cl_2) in a nocturnal marine air or for reactions with hydrogen peroxide (H_2O_2) or other oxidants in the ambient

Table 3-2
Typical Concentrations of Mercury Species in the Atmosphere
(Taken from Seigneur et al. 1994)

Mercury Species	Typical Gas-Phase Concentrations	Typical Liquid-Phase Concentrations	Reference
Hg(0)	2 to 5 ng/m^3	6 to 27 x 10^{-3} ng/l[a]	Schroeder et al. 1991
Hg(I)	not available	not available	—
Hg(II)	0.09 to 0.19 ng/m^3	3.5 to 13.3 ng/l	Brosset 1987

[a]Estimated from gas-phase air concentrations by means of Henry's law.

Table 3-3
Atmospheric Chemical Kinetic Mechanism of Mercury
(Taken from Seigneur et al. 1994)

Reaction	Equilibrium or Reaction Rate Parameter[a]
1. $Hg^0(g) + O_3(g) \rightarrow Hg(II)(g)$	$< 8 \times 10^{-19}$ cm^3 molec^{-1} s^{-1}
2. $Hg^0(g) + Cl_2(g) \rightarrow HgCl_2(g)$	$\leq 4.1 \times 10^{-16}$ cm^3 molec^{-1} s^{-1}
3. $Hg^0(g) + H_2O_2(g) \rightarrow Hg(OH)_2(g)$	$\leq 4.1 \times 10^{-16}$ cm^3 molec^{-1} s^{-1}
4. $Hg_2^{2+} \leftrightarrow Hg^0(aq) + Hg^{2+}$	2.9×10^{-9} M
5. $Hg^{2+} + SO_3^{2-} \leftrightarrow HgSO_3(aq)$	5.0×10^{12} M^{-1}
6. $HgSO_3(aq) + SO_3^{2-} \leftrightarrow Hg(SO_3)_2^{2-}$	2.5×10^{11} M^{-1}
7. $Hg(SO_3)_2^{2-} \rightarrow Hg^0(aq)$	1×10^{-4} s^{-1}
8. $HgSO_3(aq) \rightarrow Hg^0(aq) + SO_3^{2-}$	0.6 s^{-1}
9. $Hg^0(aq) + O_3(aq) \rightarrow Hg(II)(aq) + O_2(aq)$	4.7×10^7 M^{-1} s^{-1}
10. $Hg(OH)_2(aq) \leftrightarrow Hg^{2+} + 2\ OH^-$	10^{-22} M^2
11. $HgCl_2(aq) \leftrightarrow Hg^{2+} + 2\ Cl^-$	10^{-14} M^2
12. $Hg^0(g) \leftrightarrow Hg^0(aq)$	0.11 M/atm
13. $HgCl_2(g) \leftrightarrow HgCl_2(aq)$	1.4×10^6 M/atm
14. $Hg(OH)_2(g) \leftrightarrow Hg(OH)_2(aq)$	1.2×10^4 M/atm

[a]At 25°C except reaction (1) (22°C) and reaction (12) (20°C)

atmosphere); in the absence of these conditions, much longer half-lives (that is, months, years) are possible. The longer half-lives seem most plausible from the atmospheric data. Gas-phase reduction of Hg(II) to Hg(0) seems unlikely under typical atmospheric conditions where reductant concentrations are minimal.

- Aqueous-phase reactions appear to be the most important pathways for removal of elemental mercury from the atmosphere. The presence of an aqueous phase (cloud, fog, submicron particles) leads to an equilibrium between Hg(0) and Hg(II) as aqueous-phase oxidation of Hg(0) occurs by reaction with dissolved ozone and reduction of Hg(II) occurs by reaction with dissolved sulfur dioxide. The atmospheric liquid water content, the pH, the sulfur dioxide concentration, and the hydrogen chloride concentration are shown to influence this equilibrium. For a wide range of conditions simulated, Hg(0) concentrations exceeded Hg(II) concentrations by at least an order of magnitude. Hg(0) is weakly soluble in water, while Hg(II) would be subject to scavenging and washout during precipitation, leading to Hg(0) as the dominant atmospheric species.

- While organic mercury may be emitted from some sources, anthropogenic emissions appear to be primarily inorganic [Hg(0) or Hg(II)]. Organic forms are produced primarily through biogenic transformations within terrestrial and aquatic environments. There is no information on the reaction products and kinetics of atmospheric reactions that may transform organic mercury species in the atmosphere, nor on the possibility of significant atmospheric formation of organic mercury species. Many investigators have reported trace amounts of methylmercury in the air and rainwater (for example, Bloom and Watras, 1989; Mason and Fitzgerald 1990). While these reactions may be insubstantial, experimental studies are needed to confirm that this is so.

Atmospheric Transport and Deposition Mechanisms

Specific constituents that react chemically with mercury control its phase and species. However, atmospheric transport and deposition of mercury are dependent on the same atmospheric and meteorological processes and conditions as are other gases and aerosols. Consider the figure from Schroeder

and Lane (Figure 3-1) on the mercury emission-to-deposition cycle. Mercury in the troposphere, above the boundary layer, is believed to exist exclusively as gaseous Hg(0). As such, it is readily advected for long periods of time (months to years) until chemically transformed to a more soluble species or physically bound with an atmospheric aerosol. The most likely dominant deposition mechanism is via aqueous-phase chemical conversion of Hg(0) to Hg(II), with subsequent wet or dry deposition. Current estimates for gaseous Hg(II) assume a deposition velocity comparable to that of nitric acid (1–2 cm/s). However, there are no data to support this assumption. Fine particulate Hg(II) is expected to have a long half-life (weeks to months) in the atmosphere unless scavenged by precipitation.

Elemental mercury is dry-deposited to surfaces. Laboratory evidence suggests that plant uptake of Hg(0) is regulated by the ambient concentration: when the ambient air concentration exceeds a certain level, plants absorb Hg(0); at lower concentrations, Hg(0) may be emitted by the plants. This is similar to the behavior of plants with respect to ammonia (NH_3). The concentration of ammonia where the net flux in either direction is zero has been termed the "compensation point" (Hanson et al. 1994; Tjepkema et al. 1981). It seems likely that the compensation point for mercury is at concentrations greater than ambient air concentrations suggesting that vegetation may act as a mercury source. However, more experimental study is needed to better understand this mechanism, as well as the mercury fluxes from soils, water bodies, and other surfaces.

A clear definition of mercury dry-deposition processes (that is, particle settling, particle scavenging, foliage-ozone-mercury interactions) was limited by lack of technology to conduct accurate experiments. Under EPRI research, new methods were developed to perform such experiments and these methods have been deployed in the field (Kim and Lindberg 1994; Kim et al. 1993, 1994; Lindberg et al. 1992).

Atmospheric Mercury Modeling

The atmospheric transport, transformation, and fate of chemicals in power plant stack emission plumes depend on source characteristics, environmental

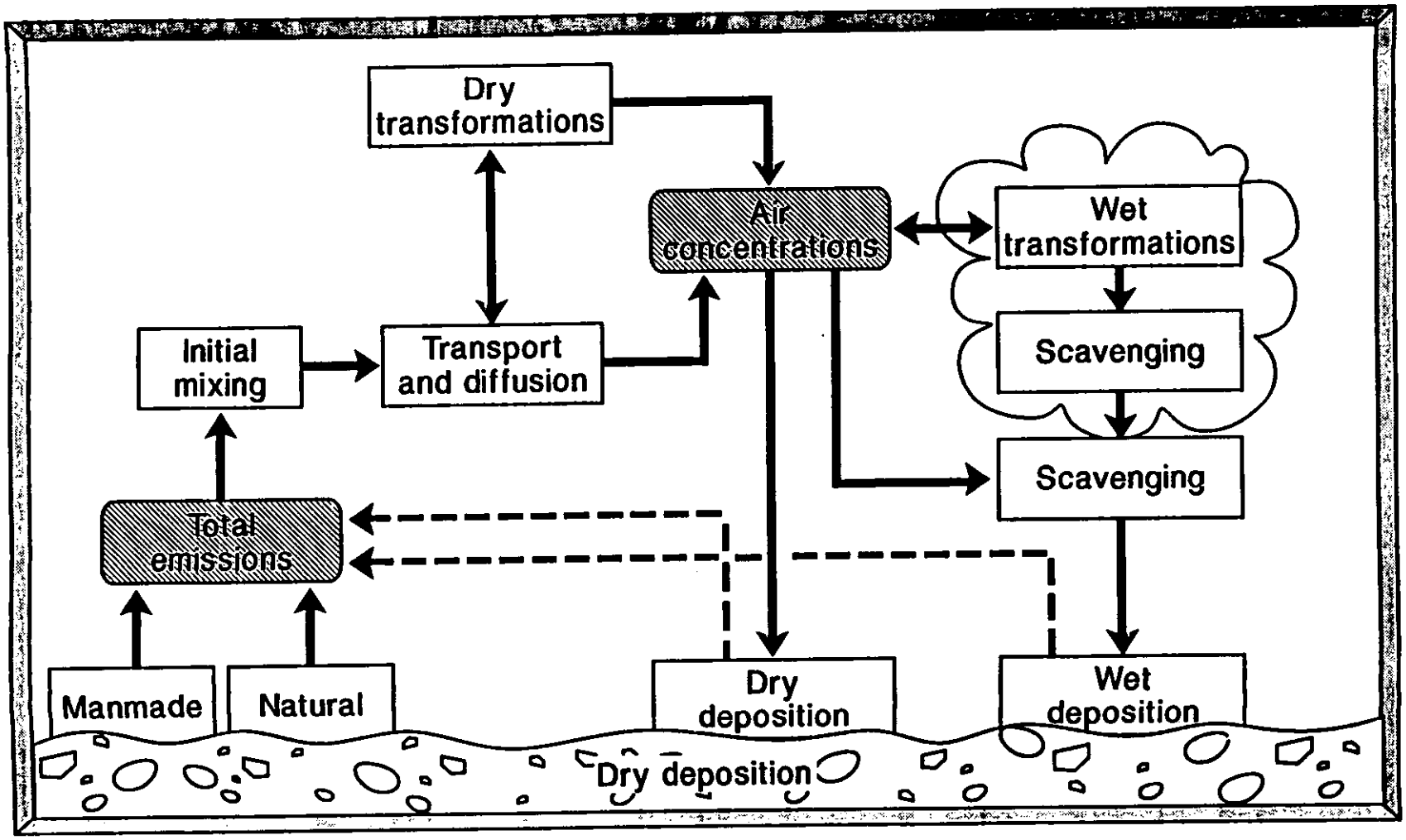

Figure 3-1
Emission-to-deposition cycle of mercury in the atmosphere (adapted from Schroeder and Lane 1988)

conditions (local and regional meteorology, terrain, nearby structures), as well as atmospheric chemistry, and removal and deposition processes (precipitation, dry deposition). An assessment of the impacts of power plant emissions on human health requires estimating utility contributions to ambient concentrations, as part of assessing impacts due to both nearby and far-upwind utility and other sources. These estimates of the relationship between source emissions and receptor concentrations are typically derived using computer models. The following information is needed to attribute a portion of receptor concentrations to specific emission sources:

1. Spatially resolved estimates of emission rates of mercury from all sources.

2. Observed atmospheric and precipitation concentrations of mercury for characterizing the prevailing exposure levels and for testing the realism of atmospheric models.

3. Rates of chemical transformations that bear on the transport and deposition rates.

4. Spatially resolved meteorological information (wind velocity as a function of atmospheric height, mixing depth, relative humidity, cloud cover, and precipitation rates) that govern the transport, dispersion, and removal of these emissions.

5. An atmospheric simulation model that synthesizes the data on emissions, chemistry, and meteorology by means of basic equations governing atmospheric dynamics.

Nearly all models being used to estimate mercury concentrations and deposition gradients were originally developed for sulfur dioxide/sulfate/acid rain concerns, and have been modified to varying degrees to capture mercury transformation and deposition rates. A summary of the atmospheric models being used to address mercury is given in Table 3-4.

The choice of modeling technique sophistication level is dictated by: (1) the precision of the input information, and (2) the desired precision of the results. The preceding information represents a first attempt to compile data about emissions, chemical transformations, and ambient concentrations of mercury; it is therefore suitable for use in a broad-brush exploratory characterization of long-term average (for example, annual) regional source-receptor relationships.

Table 3-4
Summary of Source-Based Mercury Atmospheric Models

Model	ADOM	ASTRAP	CAM	EMERC	PARADE	RELMAP	STATMOD
Sponsor(s)	AES/OMEE-Canada/ Germany	AES/DOE	SNV/ELFORSK	German Environmental Protection Agency	EPRI	EPA	EPRI
Developer(s)	AES/ENSR/OMEE	AES/Argonne Nat'l Laboratory	IVL	GKSS-Germany/ IVL-Sweden	EdF-France/ENSR	EPA	ENSR
Emissions (Sources)	Point and area sources.	Point and area sources.	Area sources.	Point and area sources.	Point sources.	Point and area sources.	Point and area sources.
Spatial Resolution (Horizontal & Vertical)	Grid size 127 km x 127 km; domain contains 33x33 grids; 12 vertical layers from 1 m to 10 km.	Trajectory model used to calculate air concentration and deposition on regional to continental scale.	Trajectory model.	Grid size 150 km x 150 km.	Trajectory (plume) model with 2-D grid (10x10 grids).	Horizontal: 1/2 deg. long. x 1/3 deg. lat. Vertical: 4 layers, up to 1500 m above ground level.	Statistical trajectory model with 1 vertical layer (mixed layer).
Transport	Eulerian. Horizontal advection and diffusion.	Lagrangian.	—	96-hour backwards trajectories.	Advection with mean wind trajectory.	Puff advection with vertically averaged wind field.	Straight-line trajectory using large scale, upper air wind rose. (Enhancement forthcoming)
Turbulent Diffusion	Vertical: advection and diffusion.	Horizontal: trajectory distribution Vertical: convective mixing.	Well mixed.	Horizontal: trajectory pathways. Vertical: well mixed planetary boundary layer.	1st order or 2nd order closure diffusion.	Horizontal: puff expansion. Vertical: mixed planetary boundary layer during day.	Horizontal: wind rose relative frequency) Vertical: mixed layer. (Enhancement forthcoming)

(cont.)

Table 3-4 (cont.)
Summary of Source-Based Mercury Atmospheric Models

Model	ADOM	ASTRAP	CAM	EMERC	PARADE	RELMAP	STATMOD
Mercury Transformation	Hg(0) oxidation by O_3 assumed balanced by reduction reactions; $HgCl_2$ scavenged using Henry's Law scavenging coefficients; Hg(part) scavenged by nucleation.	Slow net conversion of Hg(0) to Hg(part) at rate of 0.05%/hr.	Aqueous chemistry. Detailed chemistry of Hg, oxidants, and inorganic ligands.	No gas-phase transformations. Aqueous redox reactions; adsorption on soot particles.	Gas-phase and aqueous chemistry.	Hg(0) - HgII redox: O_3 oxidation and sulfite reduction; considers HgII adsorption by soot particles.	Gas-phase conversion; 1st order parameterization.
Dry Deposition	Deposition to forests.	Deposition velocity of Hg(II) and Hg(part). Includes elemental Hg.	No deposition for Hg. Deposition for reactants.	No deposition velocity for Hg(0). Dry deposition velocity for Hg(II) and Hg(part).	Deposition velocities for gases and particulates.	Deposition velocities for: Hg(0) gas, HgII gas, Hg(part), and carbon soot.	Deposition velocities.
Wet Deposition (Including Cloud & Precipitation)	(See Mercury Transformation above.)	Deposition to surface, and redistribution from mixed layer to free troposphere.	No.	Scavenging ratios for Hg(0), Hg(II), and Hg(part). Wet deposition proportional to rainfall amount.	Cloud microphysics, rainout and washout.	Precip. scavenging of HgII, Hg(part), and soot; clouds assumed at top 2 layers when precipitating.	Parameterized scavenging rate, proportional to rainfall amount.
Major Applications To Date	Eastern North America episodes; sensitivity analyses.	Great Lakes Region deposition loading.	Sensitivity analysis of chemistry.	North Sea/Baltic Sea deposition loading and deposition fluxes over Central/ Northern Europe.	Power plant plume simulations.	In progress.	Hg development in progress.
References	ERT 1984a,b.	Shannon 1985.	—	Petersen 1992a,b; Petersen and Iverfeldt 1993; Petersen et al. 1994a,b.	Joos and Seigneur 1994a,b; Seigneur et al. 1994.	Eder et al. 1986.	Venkatram et al. 1990, 1994.

Reactive Plume Model. The PARADE (Panache Réactif en Atmosphère Avec Dépôts) model, updated with current data and process knowledge (Joos et al. 1987, Joos and Seigneur 1994a,b), will incorporate treatments of: plume dynamics, including plume rise due to buoyancy and momentum, and plume dispersion due to plume turbulence and ambient turbulence; plume chemistry, including gas-phase, aerosol-phase, and droplet chemistry for ozone, nitrogen dioxide, and acid formation, as well as mercury chemistry; plume visual effects, including light scattering and absorption; and atmospheric removal processes.

Work is in progress to combine the knowledge on chemical transformation of mercury into a plume model with realistic treatment of dispersion (for example, eddy diffusivity and second-order closure) and chemistry of other plume constituents (for example, other air toxics). The critical step after this will be model evaluation, which will require gathering data on mercury concentrations in plumes, obtained via cooperative studies in the 1995–1996 timeframe.

Regional Models. The information on chemical transformation of mercury (described in the subsection on Chemical Transformations) is being combined with a reduced-form long-range transport model previously developed under EPRI sponsorship for estimating emission-deposition relationships for sulfate and nitrate. Along with emissions, and meteorological and air quality inputs, this outgrowth of the STATMOD model will be used to estimate the contribution of various source types and regions to atmospheric concentrations and deposition fluxes. We emphasize that extensive data on mercury ambient concentrations and deposition fluxes collected on a regional scale will be necessary to evaluate the performance of such models.

Estimating the Spatial Scale of Mercury Deposition: A Case Study

To investigate the spatial scale of mercury deposition, EPRI estimated the fraction of power plant emitted mercury that is deposited locally (in this case, within a radius of 50 km from the source). The calculations were done using the dispersion and deposition algorithms of EPRI's Total Risk of Utility Emissions (TRUE) model (Constantinou and Seigneur 1993). TRUE is a screening-level multimedia health risk assessment model, developed to predict the fate and

transport of chemicals in the different environmental media, and estimate the associated health risks (see Section 6).

In TRUE, the atmospheric fate and transport model is the USEPA-approved ISCLT2 that calculates the steady-state atmospheric concentrations at receptors placed on a polar grid with 10-degree increments and at various radial spacings. The model study area is defined in this case study by a radius of up to 50 km around the power plant. The average concentration and deposition rates in each subregion are calculated using chemical-specific deposition velocities for dry deposition and precipitation data with chemical-specific scavenging coefficients for wet deposition.

For this case study, four power plants with available dispersion characteristics were used. The calculations were based on the "extreme case" assumption that all mercury emitted is in the form of gaseous Hg(II), and that it remains in this form as the plume travels downwind resulting in higher scavenging by rain and high deposition during dry conditions. The plants all had relatively tall stacks ranging from 180 to 230 m. Therefore the results are representative only of power plants with comparable stacks, which includes a large fraction of the utility industry.

To perform the calculations, all the dispersion characteristics (that is, wind speed at plume height, percent time with rain, concentration-to-emission-rate ratio) were kept constant at their measured values, while the deposition characteristics (that is, dry deposition velocity and scavenging rate) were allowed to vary within a range of possible values. The range of possible deposition values was based on the assumption that gaseous Hg(II) is similar to HNO_3 (using dry deposition velocities reported in the literature ranging from 0.06 to 5 cm/s and scavenging rates of 0.003 to 0.05 s^{-1}). The calculations specifically evaluated the fraction of mercury emissions deposited within a 50-km radius of the source. The resulting ranges for the four case studies were:

Plant A:	min = 0.5%	max = 22%
Plant B:	min = 0.4%	max = 15%
Plant C:	min = 0.5%	max = 21%
Plant D:	min = 0.6%	max = 27%

The wide variation in the results is attributed primarily to the wide range of possible dry deposition velocities provided in the literature for HNO_3. Recent EPRI work by Kim et al. (1994) derived dry deposition velocities for Hg(0) over deciduous forest soils based on micrometeorological measurements. Their estimated deposition velocities were in the range of 0.03 ± 0.024 cm/s; this is less than the lowest values used for gaseous Hg(II). Thus, the results of the case study would approach maximum values of less than 5 percent if emissions were assumed to be all Hg(0).

In a second analysis, the case study calculations were performed assuming that all the mercury is in the particulate form (using a deposition velocity of 1 cm/s and a scavenging rate of 4×10^{-4} s^{-1}). Because indications are that Hg(II) occurs as particulate in the air, this calculation may be more realistic than the analysis above which assumes that mercury deposition is similar to that of nitric acid. With these alternative assumptions, the maximum fraction of mercury emissions deposited within a 50-km radius was estimated to be between 4 and 7 percent.

Recent plume modeling studies, using a state-of-the-art model (PARADE, Table 3-4; Constantinou et al. 1994), have indicated that the contribution of a hypothetical power plant to the atmospheric mercury deposition within 100 km of the plant is in the range of 1–4 percent of total deposition. The ratio of Hg(II) to Hg(0) and total emissions could alter the range to lower or higher fractions depending on site-specific conditions.

These estimates tend to indicate that a small to moderate fraction (1–10 percent) of emitted mercury is deposited near the source, and that a much larger fraction of the point-source emissions enters the regional (or global) regime.

Section 4
MERCURY IN AQUATIC ECOSYSTEMS

Mercury Biogeochemistry

Transforming Mercury. After deposition of mercury into a watershed or directly onto a surface water, natural processes transport and transform mercury. Methylmercury has a long biological half-life in fish, accounting for its bioaccumulation. Fish-eating birds, humans, and other fish eaters subsequently become subject to increased risk governed by the amount of fish ingested. Thus, methods are needed to calculate the chemical form and amounts of mercury accumulated by fish so that risk analysis methods can be used to estimate the effects of mercury deposition on risks to populations of interest. The primary purpose of EPRI's research on mercury in freshwater environments was the construction of a simulation model to calculate mercury accumulation by fish in relation to mercury inputs and to all the site specific factors that control the rate of accumulation. The model required an extensive array of field research to develop process concepts and the data for parameterizing process equations.

Lake Biogeochemistry. Previous sections of this report have focused on large spatial scales. Focusing on a smaller scale—a single lake—can provide useful insights about mercury biogeochemistry at those larger scales. Biogeochemical studies of lakes that contain fish with high mercury levels (Grieb et al. 1990; Spry and Wiener 1991), yet remote from point sources and mercury-containing geological strata, can delineate the extent and role of atmospheric exchange of mercury. The investigators for the Mercury in Temperate Lakes (MTL) project selected seven northern Wisconsin seepage lakes that were isolated from all but atmospheric sources to quantify mercury cycling and mass balances (Watras et al. 1994). The seepage lakes in northern Wisconsin represent the type of lakes most likely to show effects of atmospheric deposition. In these lakes, essentially all inputs of mercury come from the air. The lake waters have low nutrient concentrations, produce small amounts of biomass, and can be acidified easily because of low buffer capacity. The lakes are remote from point sources, small, and hydrologically simple, making them easier to assess than large lakes with large drainage basins and complex morphometry. Although these characteristics are typical of the region, they tend to reflect factors that generally enhance

bioaccumulation of methylmercury. In contrast to seepage lakes that do not have a watershed, drainage lakes receive inflows from their watersheds. These inflows contain atmospheric deposition from their watersheds along with mercury from geologic sources, mobilized by precipitation on the watershed.

MTL was coordinated with a National Acidic Precipitation Assessment Program (NAPAP) project to assess the effect of acidification on Little Rock Lake. Little Rock Lake was physically divided into two nearly equal parts, one of which was treated with acid and called the "treatment basin" while the other was retained as the "reference basin" (see Watras and Frost, 1989; Watras et al. 1994; Wiener et al. 1990; Bloom et al. 1991). This section summarizes a mass balance for Little Rock Lake treatment basin (Watras et al. 1994). In addition, a mass balance for a reservoir with an abandoned mercury mine in the watershed is discussed relative to Little Rock Lake (Gill and Bruland 1992). Also, implications of mercury biogeochemistry and the applications of a simulation model for mercury cycling are summarized.

Mercury Mass Balances

Total Mercury. The Little Rock Lake treatment basin is 10 hectares in volume, relatively shallow, and has a small hypolimnion (a layer of water isolated from the upper mixed layer because of density differences) that is lacking in the shallower reference basin (Watras and Frost, 1989). Measurements taken over a 3-year period quantified the mass balance for total mercury in the treatment basin (Figure 4-1). In round numbers, the standard deviation of the annual mean fluxes and pools varied from 5–30 percent. The atmospheric mercury concentration of 1.6 ng/m^3 and wet deposition input of 0.7 g/yr was typical of open ocean values in the northeastern Pacific and northwestern Atlantic (Fitzgerald et al. 1991; Slemr et al. 1985). Almost all (99 percent) of the atmospheric mercury was Hg(0), and particulate mercury (Hg(II)) was assumed to account for almost all of the mercury input as wet (measured) and dry (calculated from particle deposition velocities; Fitzgerald et al. 1991) deposition. Inputs of mercury in surface water and ground water were nil, supporting the assumption of a system dominated by atmospheric inputs. Evasion of mercury as Hg(0) from Little Rock Lake to the atmosphere (0.7 μg/m^2yr) was about

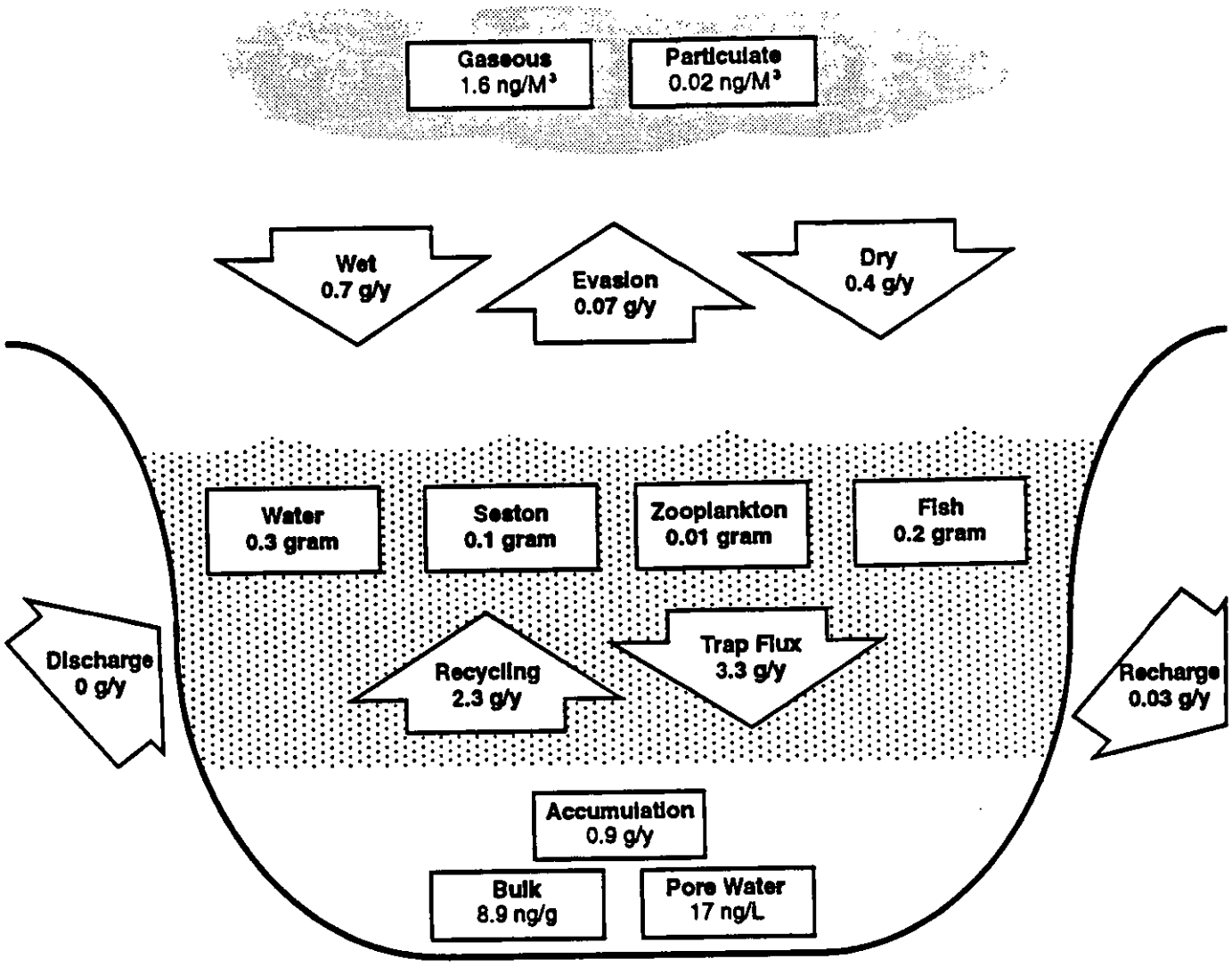

Figure 4-1
Mass balance for total mercury in Little Rock treatment basin showing that atmospheric sources can account for all mercury in fish (adapted from Watras et al. 1994)

5 percent of the deposited input. In other MTL lakes, evasion ranged from 10–50 percent of the total deposition (Watras et al. 1994).

Cycling of mercury occurred within the lake, with about as much mercury in the biota (seston—particulate organic matter that was mostly phytoplankton) as in the water column, an observation atypical of many other microconstituents in lakes. Considerable resuspension and settling occurred, but the bulk (more than 90 percent) of the deposited mercury settled into the sediment pool.

Atmospheric deposition was the major source of mercury to the lake. Deposition was balanced by sedimentation plus evasion and a small flux to the ground water. The annual atmospheric deposition of mercury was almost a factor of 4 greater than the total mercury in the biotic pool.

Methylmercury. The mass balance for methylmercury differed greatly from that for total mercury (Figure 4-2). Standard deviations varied from 5–100 percent of the mean fluxes and pools (Watras et al. 1994). Methylmercury varied more than total mercury, primarily due to seasonal variation in water column concentrations. Although detected in the atmosphere, methylmercury accounted for only about 1 percent of the total mercury input to the lake. Fish were the dominant methylmercury pool in the water column. The measured sediment trap flux was substantial, but sediment methylmercury concentration was only about 1 percent of total mercury. Unlike total mercury, the net flux of methylmercury to and from the sediments appeared to be zero in this lake. The mass balance showed that virtually all methylmercury was formed within the lake ecosystem.

Reservoir Comparison. A less detailed mass balance for total mercury was constructed for Davis Creek Reservoir in northern California. The dilute-water, low-pH Wisconsin lake described above provides a strong contrast to the more productive, high-pH California reservoir that also receives more mercury input. The reservoir receives substantial inflow from its watershed, which contains an abandoned and partly remediated mercury mine and smelter (Gill and Bruland 1992). Quantitatively unlike the evasion from Little Rock Lake, the Hg(0) evasion from Davis Creek Reservoir to the atmosphere is at least double the atmospheric deposition (calculated by Gill and Bruland 1992 from literature values) and about

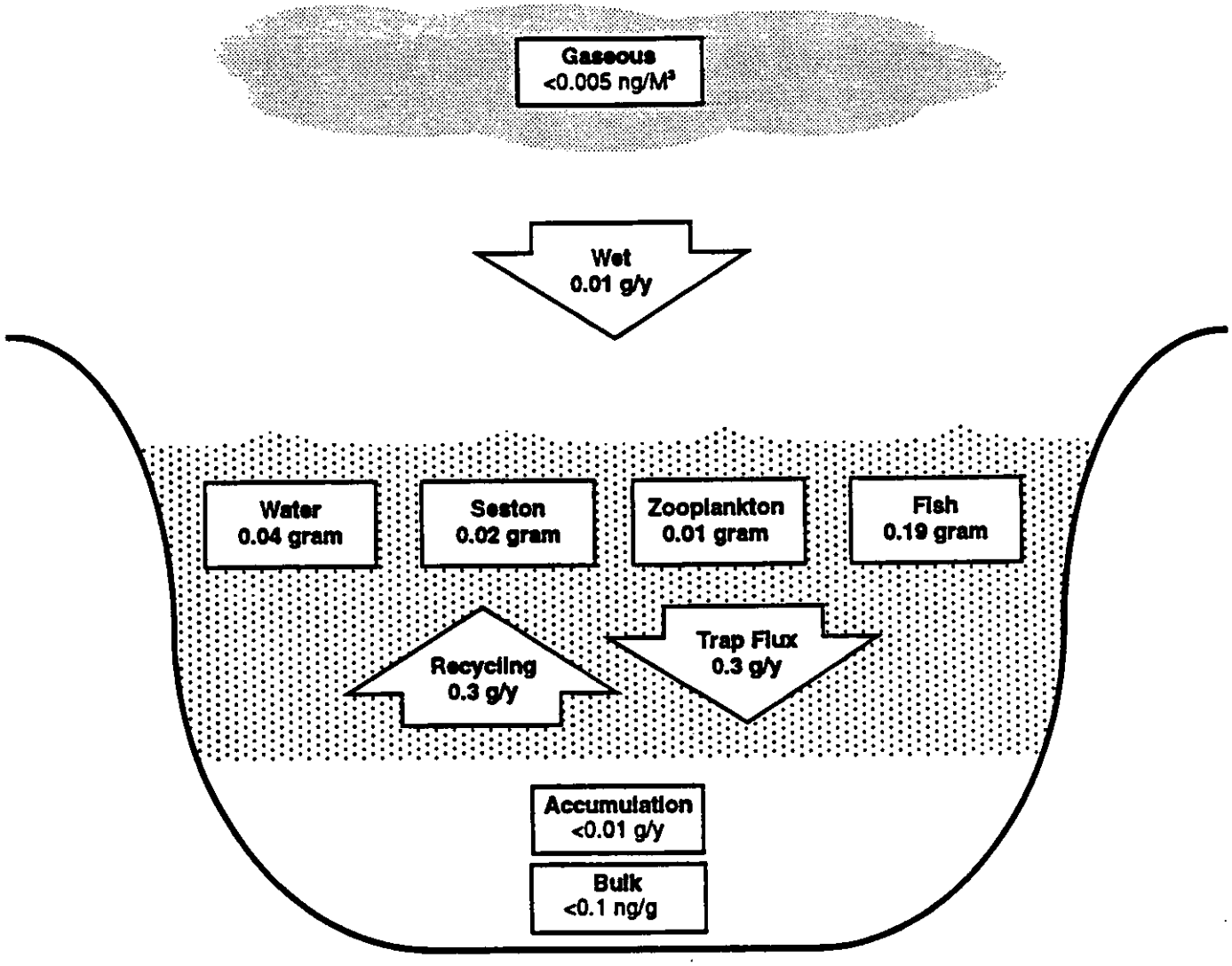

Figure 4-2
Mass balance for methylmercury showing that methylation within the lake ecosystem is the key process affecting mercury accumulation by fish (adapted from Watras et al. 1994)

equal to the watershed mercury loading to the lake. The median mass of dissolved mercury in the reservoir was less than that of the particulate form. Areal deposition rates were assumed equivalent to Wisconsin results (10 $\mu g/m^2yr$), but areal evasion rates of 25 $\mu g/m^2yr$ were calculated to be about 30 to 40 times the rate at Little Rock Lake treatment basin (0.7 $\mu g/m^2yr$). Although due in part to the higher input from the watershed, these results support the arguments of Fitzgerald et al. (1994) that higher pH favors production and subsequent evasion of elemental mercury. In laboratory studies, the evasion of elemental mercury in the experimental systems seemed to depend on biotic processes because no evasion occurred in sterile (autoclaved) systems (Regnell and Tunlid 1991). Outflow from Davis Creek Reservoir during the period of study removed about the same mass as was deposited, and assuming a steady state, the water column and sediment compartments would be in balance. Low rainfall due to drought occurred during the study, suggesting that additional mercury loading from the watershed would have occurred under more typical hydrologic conditions and would account for increased input of mercury to sediments during high rainfall years.

The Davis Creek Reservoir study also shows that other factors, such as manganese cycling, may control the availability of mercury and its speciation by binding Hg(II) and changing its availability for chemical and biological reactions. (Gill and Bruland 1992). It may be that manganese acts analogously to aluminum in its apparent, but undiscerned, role in mercury biogeochemistry in drainage lakes (Grieb et al. 1990). Concentrations and chemical forms of metals such as manganese and aluminum vary seasonally, and their binding of other trace constituents changes considerably and follows a repetitive annual cycle. Although this pattern is more obvious in the chemically richer Davis Creek Reservoir, even the relatively dilute systems of the MTL lakes may follow a similar annual cycle. Results from lakes in the Adirondacks strongly support a similar pattern (Driscoll et al. 1994).

Biogeochemical Processes

Previous research had suggested that pH and DOC (dissolved organic carbon) affected methylmercury accumulation in fish (see reviews in Grieb et al. 1990; Spry and Wiener 1991; Driscoll et al. 1994). Generally, low pH is associated with

high fish mercury; in drainage lakes high DOC is associated with high mercury levels in fish, while in seepage lakes there is no obvious DOC relationship with fish mercury. As discussed in the subsection on Production of Methylmercury, wetlands—which are an important source of DOC and often have low pH waters—appear to play a pivotal role in the production of methylmercury. Therefore, seven lakes within a relatively small area were selected for study in the MTL project, spanning a range of pH and DOC (Watras et al. 1994). If atmospheric deposition were the sole factor controlling fish mercury, the fish from these lakes would presumably have relatively similar mercury concentrations. In fact, the concentrations varied ten-fold showing conclusively that factors other than deposition have more effect on fish methylmercury accumulation (Figure 4-3). This result supports the observation that fish mercury seems to show no downward trend in Minnesota despite a two-to-three-fold decrease in deposition (see Zillioux et al. 1993; Temporal Perspectives in Section 3). It is not possible to explain the differences solely on the basis of pH and DOC, although pH is clearly involved (Grieb et al. 1990; Wiener, et al. 1994; Hudson et al. 1994). For example, Rada et al. (1993) showed that areal burdens of mercury in sediments varied strongly with pH and hypothesized that pH-related efflux of gaseous mercury (Hg(0)) from water to atmosphere was partly responsible. Other chemical factors such as chlorophyll *a*, sulfate, chloride, and calcium vary two-fold (Watras et al. 1994), and appear to be at least partly responsible for some of the differences (Hudson et al. 1994). Lake morphometry varies considerably, but no clear relationship between lake physical characteristics and methylmercury concentration exists.

Bioaccumulation of Mercury. Certain chemicals, such as mercury, show increased concentrations in organisms that feed at higher levels in the food web (Table 4-1). The increase, called biomagnification, occurs largely because the lower levels in the food web bioaccumulate much of the available mercury from the water. Then each succeedingly higher level takes in mercury from water and food and, because it consumes much more food than its mass during growth, its exposure to mercury increases over that of the preceding level.

Biomagnification of methylmercury occurred in the Little Rock Lake treatment basin (Table 4-1). Also, another process seemed to be involved in producing the high concentrations of methylmercury in higher trophic levels: a higher affinity

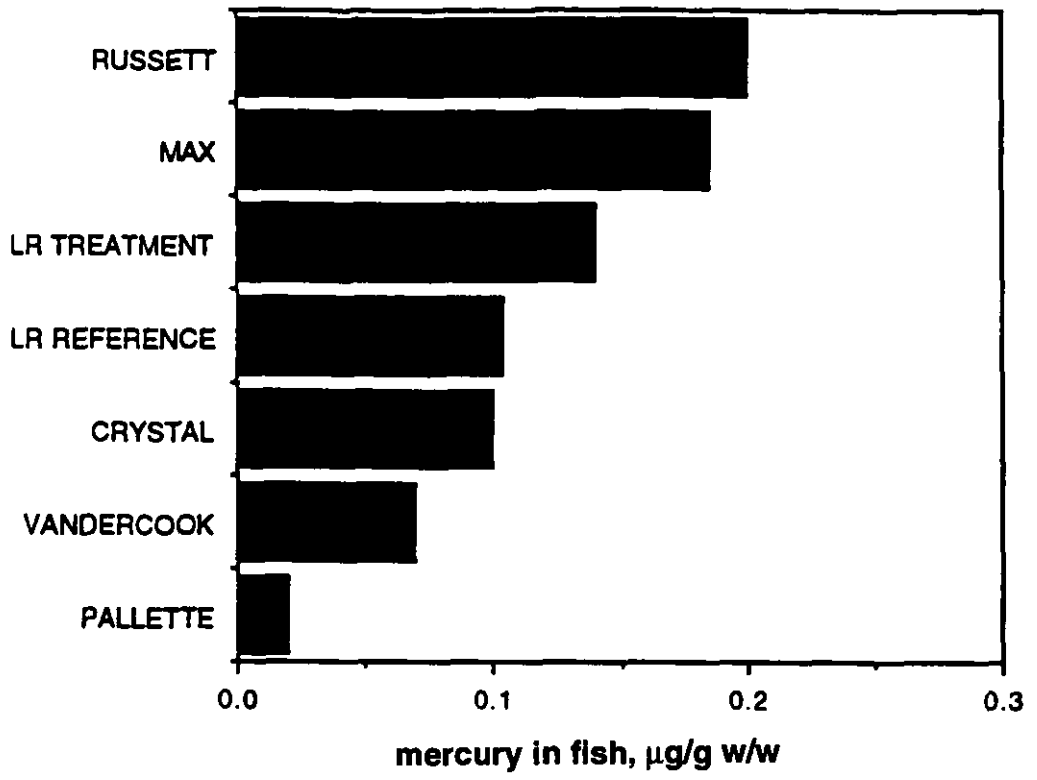

Figure 4-3
Order-of-magnitude differences in mercury content of one-year-old yellow perch from seven northern Wisconsin seepage lakes suggest that factors other than atmospheric deposition lead to differences (data from Watras et al. 1994).

Table 4-1
Biomagnification of Methylmercury
in the Aquatic Food Chain of Little Rock Lake (Treatment Basin)
(Modified from Watras and Bloom 1992)

	Relative Concentrations		
Compartment	Methyl-Hg	Non-Methyl-Hg	Percent Methyl-Hg
Water	1	10	10
Phytoplankton	100,000	500,000	15
Zooplankton	320,000	800,000	30
Fish	3,200,000	100,000	95

for non-methylmercury in organisms of lower trophic levels, and a higher affinity for methylmercury in fish, where affinity is defined as the net effect of water and food intake and release by secretion and excretion. This produces the long biological half-life of up to 2 years for mercury in fish (Spry and Wiener, 1991) as opposed to the biological half-life of about 70 days in humans (Clarkson, 1990).

By looking at younger one-year-old fish, some of the complexities caused by long-term accumulation are minimized, making it easier to interpret relationships. For example, older fish have greater mercury concentrations (for example, Grieb et al. 1990). For one-year-old yellow perch (*Perca flavescens*), 95 percent of the total mercury was methylmercury, a value consistent with other results (Grieb et al. 1990; Bloom 1992a). The overall percentage of methyl-mercury in young perch (one-year-old fish) was considerably higher than that in lower trophic levels in the treatment basin. The fish accumulated the methylmercury from both food sources and the water column, and the bioaccumulation factor (calculated from the ratio of total whole fish to measured aqueous methylmercury) was greater than 3 million (Table 4-1). This indicates that methylmercury was concentrated by a factor of 3 million times in whole fish relative to water in this dilute-water lake. These results emphasize the importance of methylation as a key process in mercury biogeochemistry, and suggest that fish mercury may bear a relationship to total mercury in the water.

Concentrations of methyl- and total mercury were correlated among the seven lakes (Figure 4-4). This result supports the contention that total Hg(II) serves as the substrate for methylation (see analysis by Hudson et al. 1994). Three methods were used to bound an estimate of the amount of total mercury transformed to methylmercury: regression, comparison of sediment trap measurements, and modeling. The regression slope was heavily influenced by a single point (Russet Lake); accepting that, the slope suggests that about 20 percent of the total mercury was transformed to methylmercury (Figure 4-4). The regression estimate was within a factor of 2 of the sediment trap measurements shown in the treatment basin mass balances (Figures 4-1 and 4-2), which suggests a 13 percent transformation (0.3 g/yr methylmercury recycled out of 2.3 g/yr of total mercury). Simulations with the Mercury Cycling Model (Hudson et al. 1994; also, see following subsection) suggest that less than

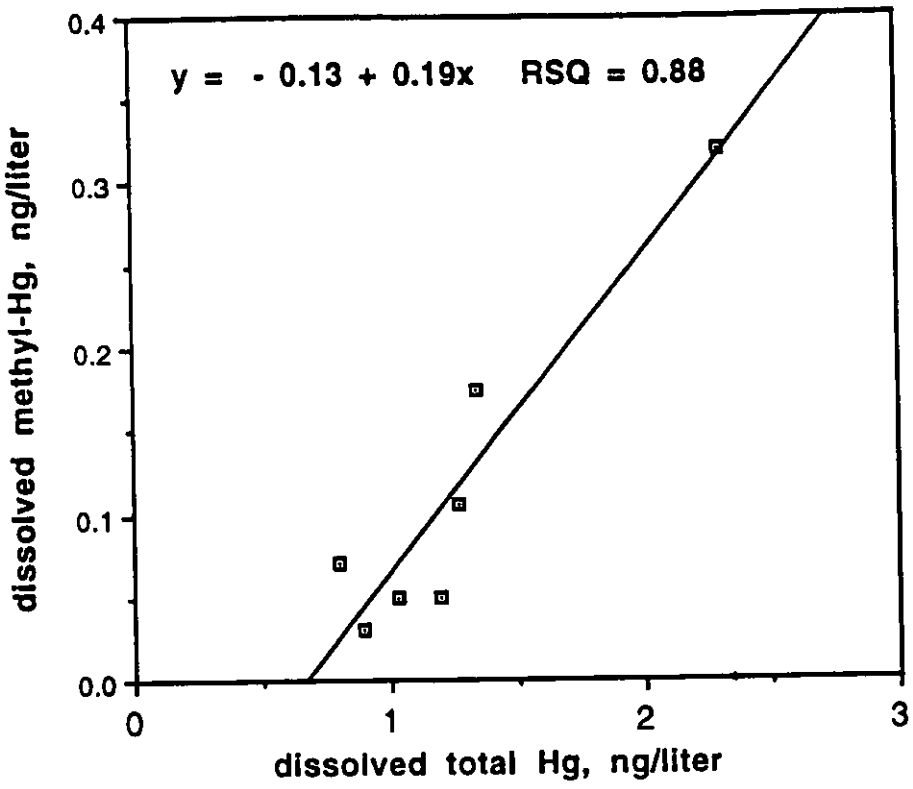

Figure 4-4
Dissolved methylmercury is highly correlated with total mercury in the waters of seven northern Wisconsin lakes (data from Watras et al. 1994)

5 percent of total mercury entering the lake was transformed to methylmercury (R. Hudson, 1992). The model, unlike the regression and mass balance calculations, accounted for temporal changes that probably occurred. Consequently, the 5 percent conversion estimate seems most likely. These results are consistent with similar ratios of methylmercury to total mercury in Sweden (0.02–0.06) tabulated in Lee and Iverfeldt (1991).

Relating mercury in one-year-old yellow perch to aqueous methylmercury yielded a lower, but significant, correlation (Figure 4-5). Gill and Bruland (1990) were the first to show a relationship between organic mercury and fish mercury concentrations. However, their data do not fit the same line shown in Figure 4-5, but instead have a slope of 0.05 ng/l of organic mercury per μg/g of total mercury in fish. Lee and Iverfeldt (1991) apparently found no correlation between fish mercury and methylmercury concentrations for eight Swedish lakes; they correlated fish mercury to the ratio of methylmercury to total mercury. Moreover, the eight Swedish lakes were drainage lakes receiving substantial organic matter from watershed wetlands where abiotic methylation could have occurred (Lee et al. 1985). Similarly, Driscoll et al. (1994) did not find a correlation between fish mercury and methylmercury. Driscoll et al. (1994) suggested that wetlands affect production of mercury and methylmercury, and these inputs would further complicate such a simple relationship as that shown in Figure 4-5. Differences in analytical procedures or fish species, or the effects of different nutrient and other water quality factors in the MTL lakes, may also help account for the different relationships seen in other lakes. Fish integrate exposure over a period of time (Grieb et al. 1990), and comparing fish concentrations to annual mean water concentrations could introduce bias. In the case of Figure 4-5, the relationship seemed appropriate only because one-year-old fish were used.

The slope of the relationship for the seven lakes shown in Figure 4-5 was equivalent to a bioaccumulation factor of 500,000, about an order of magnitude less than the factor shown in Table 4-1 for the treatment basin. A more likely bioaccumulation factor is the mean value for the seven lakes (2,000,000), because of the effect of the Russet Lake value on the regression. Given the slopes in Figures 4-4 and 4-5, a bioaccumulation factor of about 100,000 was calculated from the ratio of total fish mercury to total mercury in water (as is done for most

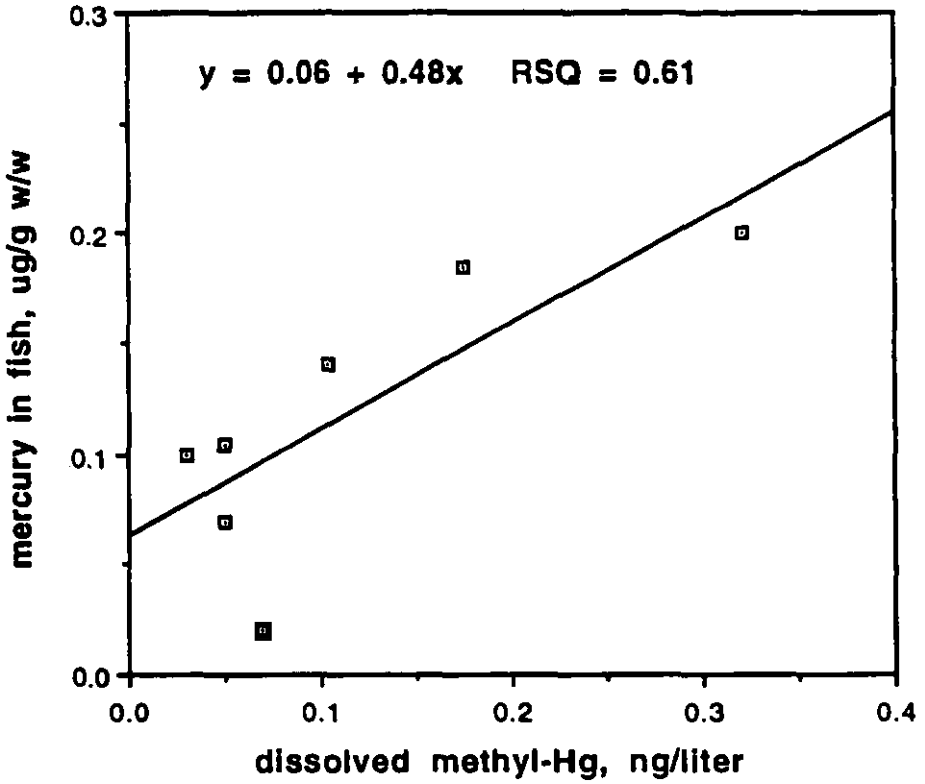

Figure 4-5
Mercury in one-year-old yellow perch from seven northern Wisconsin lakes is correlated with methylmercury in lake water (data from Watras et al. 1994)

studies because the cost of measuring methylmercury is prohibitive). This factor may change with water quality, as illustrated by the attempt to compare regression slopes with Gill and Bruland's (1990) data. Nutrient levels can substantially affect mercury concentrations in fish (for example, Rudd et al. 1983), and model analyses performed by Hudson (1992) show substantial decreases in fish mercury concentration from increased nutrients and consequent phytoplankton production. Moreover, large fish can have higher bioaccumulation because of age and food sources. Given these results, bioaccumulation factors do not seem applicable to a mercury risk assessment covering entire regions, but should be used only in a site-specific assessment (see discussion on the Mercury Cycling Model). Furthermore, the results re-emphasize the need to understand methylation processes [Hg(II) transformed to CH_3Hg^+].

Production of Methylmercury. Recent reviews describe methylation and demethylation processes in aquatic systems (Winfrey and Rudd 1990; Gilmour and Henry 1991; Olson 1991). Methylation appears to be a co-metabolic reaction with no known specific gene control (Summers 1986). In the seepage lake described above, biotic methylation appeared to produce almost all of the methylmercury. Sulfate-reducing bacteria appear to be important mediators of the methylation process (Winfrey and Rudd, 1990; Gilmour and Henry 1991; Compeau and Bartha 1985). Direct evidence for sulfate reduction being linked to methylation of mercury comes from estuarine studies using specific metabolic inhibitors (Compeau and Bartha 1985). Compeau and Bartha (1985) concluded that mercury methylating activity is fully expressed only when sulfate is limiting and appropriate energy sources are available. Gilmour et al. (1992), working with sediments from a freshwater lake with experimentally added Hg(II), showed that enrichments of sediment slurries up to about 100 micromoles of sulfate enhanced the production of methylmercury until sulfate was exhausted. Inhibition of sulfate reduction blocked almost all methylation. Evidence from this experiment suggests that methanogens (methane-producing bacteria) contribute little to mercury methylation, unlike previous suggestions (for example, Wood et al. 1968). Choi and Bartha (1993) showed that cobalamin (vitamin B_{12}) is the intermediate metabolite that methylates mercury. Although sulfate reduction is associated with methylation, fermentation produces the cobalamin and methylation is observed only during fermentation; methylation

ceases when fermentation stops, even though sulfate reduction continues. Not all sulfate reducers produce this compound, and mercury methylation appears to be incidental to the production of cobalamin.

In addition to biotic methylation, abiotic methylation can be an important wetland process (Lee et al. 1985); apparently, metals acting as catalysts and humic acids (organic matter) are all that are required for abiotic methylation of Hg(II) (Lee et al. 1985). Bacteria on plant surfaces could be responsible for the wetland methylation, and it is unclear whether—or how much—biotic or abiotic processes are involved.

High concentrations of methylmercury may be discharged from wetlands (for example, Lee and Hultberg 1990). St. Louis et al. (1994) showed that wetland portions of catchments yielded up to about 80 times more methylmercury (0.555 $\mu g/m^2$ yr) than purely upland catchments (0.007 $\mu g/m^2$ yr), and methylmercury was as much as 8 percent of the total mercury. In the seepage lake examples, abiotic methylation does not appear to be important. Driscoll et al. (1994) showed for 16 remote Adirondack lakes that total mercury of 1–6 ng/l increased roughly proportionally with methylmercury concentrations of 0.05–0.70 ng/l. Dissolved organic carbon correlated with percentage wetland in Adirondack watersheds and appeared to relate to the mercury species. These results suggest that wetlands may increase availability of total mercury as well as production of methylmercury.

The other half of the process that affects methylmercury concentrations is demethylation. Demethylation [CH_3Hg^+ transformed to Hg(II) and then to Hg(0)] is primarily enzyme mediated, taking place in a single cell and controlled by two genes (Summers 1986). There has been considerable effort to characterize demethylation processes (Oremland et al. 1991). Interestingly, the same types of organisms that methylate (sulfate reducers and methanogens) appear to be involved in anaerobic demethylation of freshwater sediments (Oremland et al. 1991). Aerobic demethylation occurs in estuarine sediments, but appears to be relatively unimportant in freshwaters (Oremland et al. 1991).

Net methylation (the net of methylation and demethylation processes) results in the methylmercury that bioaccumulates. Environmental factors affect

methylation and demethylation processes to increase or decrease net methylation (Winfrey and Rudd 1990). For example, Bodaly et al. (1993) hypothesized that higher temperature lake systems promoted higher mercury concentrations in fish based on a high correlation of lake temperature with fish mercury content. Also, experiments showed that methylation rates increased faster than demethylation rates as temperature increased (Bodaly et al. 1993). In addition to temperature, Wiener (1992) provided data showing that, although the well-known inverse relation between mercury fish concentration and pH was confirmed, it was difficult to ascertain organic matter relationships with fish mercury by measuring DOC; Wiener hypothesized that the latter relationship varies with many other factors affecting fish and mercury bioavailability. Driscoll et al. (1994) found a strong correlation of fish mercury with DOC in studies of 16 Adirondack drainage lakes.

As a first step in explaining the methylation process, concentration profiles in MTL Pallette Lake (Figure 4-6) indicate that redox conditions and sulfur cycling affect methylation and mercury dynamics (data from Bloom et al. 1991; Watras et al. 1994; graph provided by Hudson and Gherini 1992). Mercury species are shown in the left panel of Figure 4-6 and total sulfide [S(II)] and dissolved oxygen (O_2) are shown in the right panel. One key factor appears to be dissolved oxygen, which declined below 10 meters depth to near anoxia at 14 meters. At these same depths, sulfide increased from near zero to almost 40 mg/l due to sulfate reduction. Total mercury [almost 90 percent Hg(II)] generally declined with depth for the mixed layer profile (above 10 meters depth). However, the concentration of total mercury increased substantially to the range of 1.5–2 ng/l at 14 meters. At 10 meters, Hg(0) approached zero, suggesting that reduction of Hg(II) occurred only in the mixed layer. Although relatively constant and constituting less than 10 percent of the total mercury in the mixed layer, methylmercury concentration increased greatly below 10 meters, accounting for about 30 percent of the total mercury in the anoxic layer. This suggests that methylation was markedly greater in the anoxic zone than the oxygenated upper layers. Methylmercury did not account for the entire increase observed in the total mercury concentrations. Although transport of particulate material from the mixed layer may account for some of the methylmercury produced and for the increased total mercury concentrations, the increased relative concentration of methylmercury suggests that net methylation increased in the hypolimnion.

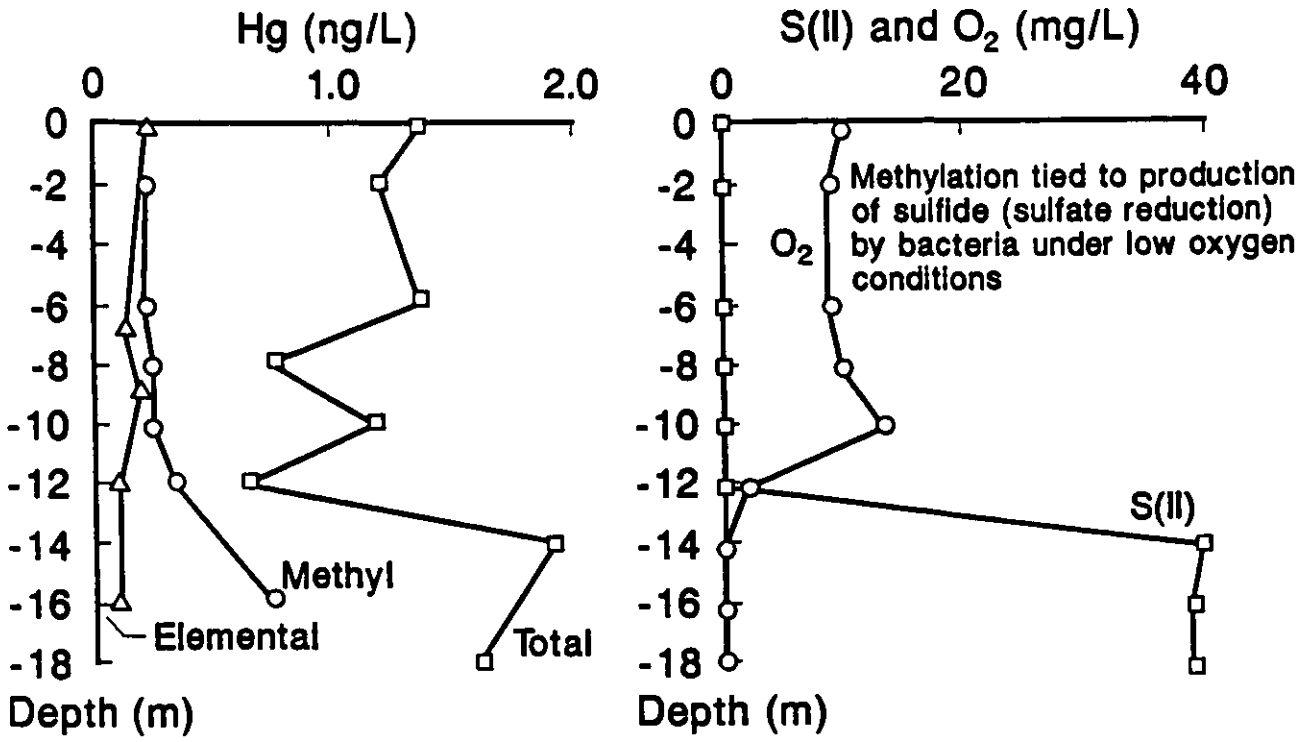

Figure 4-6
Late summer profiles in a stratified Wisconsin seepage lake support a linkage between sulfate reduction and net methylation of mercury (data from Bloom et al. 1991)

The results lend credence to the hypothesis that methylation is linked to sulfur cycling, perhaps via sulfate reduction (Compeau and Bartha 1985; Gilmour and Henry 1991).

Mercury Cycling Model (MCM, Version 1.0)

To further examine the large variety of processes and factors affecting mercury methylation and bioaccumulation in fish and to deal with the broad variety of aquatic environments, a simulation model incorporating the most recent understanding of mercury biogeochemistry was developed during the MTL project (Hudson et al. 1994). One of the most important uses of the MCM will be the prediction of methylmercury in fish (recall that fish consumption causes the major exposure of target organisms to methylmercury).

As presently formulated, the MCM is bounded by the atmosphere, the lake margins, and a deep sediment layer (Figure 4-7). Reactions in the watershed and the atmosphere are not modeled; volumetric inflows and concentrations are measured to provide mercury input at the model boundary. The MCM tracks all three major species of mercury [Hg(0), Hg(II), CH_3Hg^+] in three physical compartments: an upper mixed layer (epilimnion), a lower layer (hypolimnion), and the sediments. At one time dimethylmercury was considered as a possibly important chemical species, but so far it has been observed only at extremely low levels in marine environments (Mason and Fitzgerald 1990). The model defines four biotic compartments as occurring in the two aquatic layers: phytoplankton, zooplankton, a forage fish population, and a piscivorous fish population. Although simulation output can include any variable, the target of interest is mercury concentrations in piscivorous fish. The MCM has a monthly time step and runs on Macintosh™ computers. Additional information about the model can be found in Hudson et al. (1994).

According to model runs and field results from the seven Wisconsin seepage lakes, the major processes dominating fish uptake of mercury are methylation-demethylation (Figure 4-8) (Hudson et al. 1994). Calibration results for Little Rock Lake reference basin (Figure 4-9) indicate good agreement with biotic components (Hudson et al. 1994). Performing an even more difficult task, the

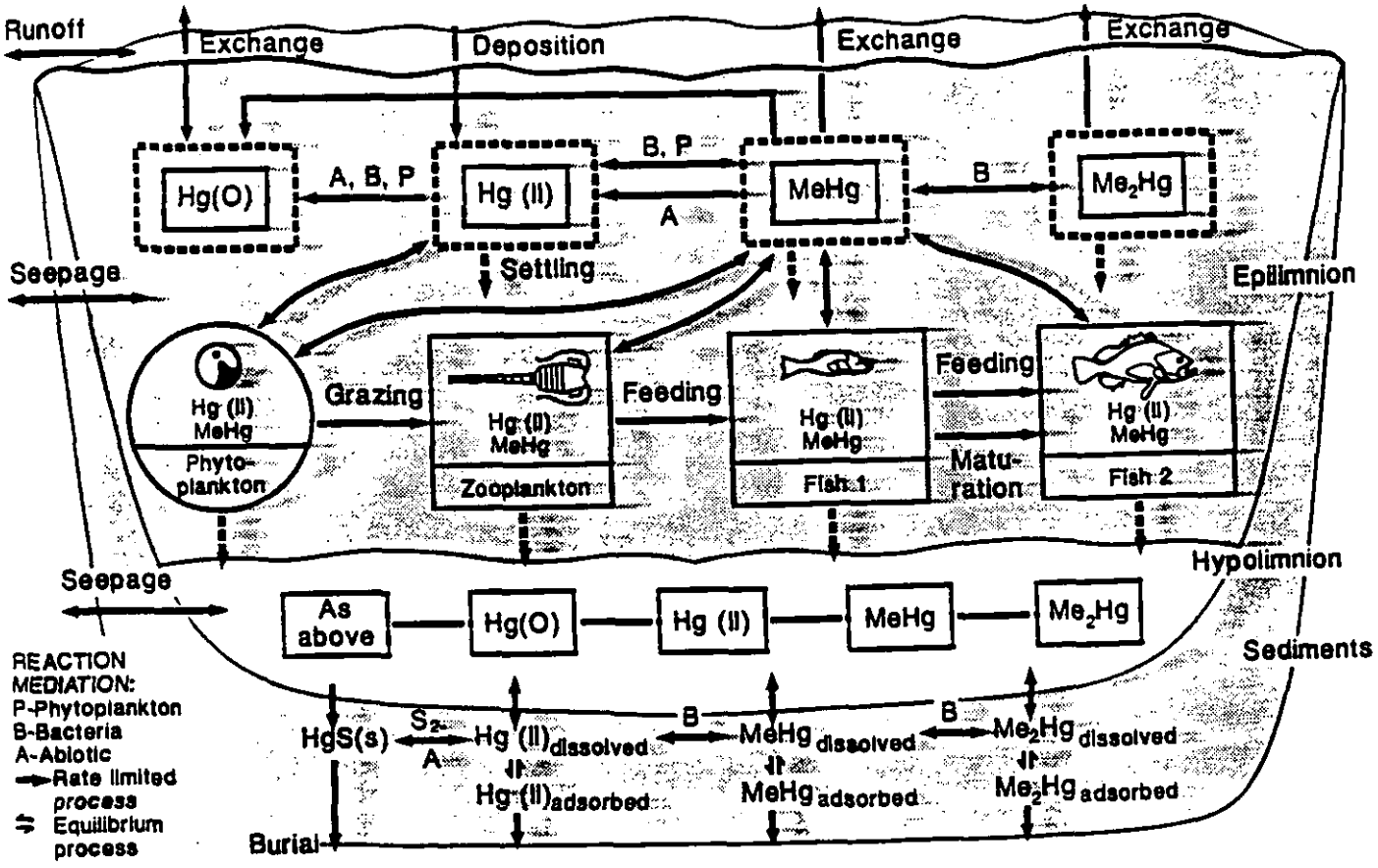

Figure 4-7
Schematic presentation of the Mercury Cycling Model (MCM v. 1.0)

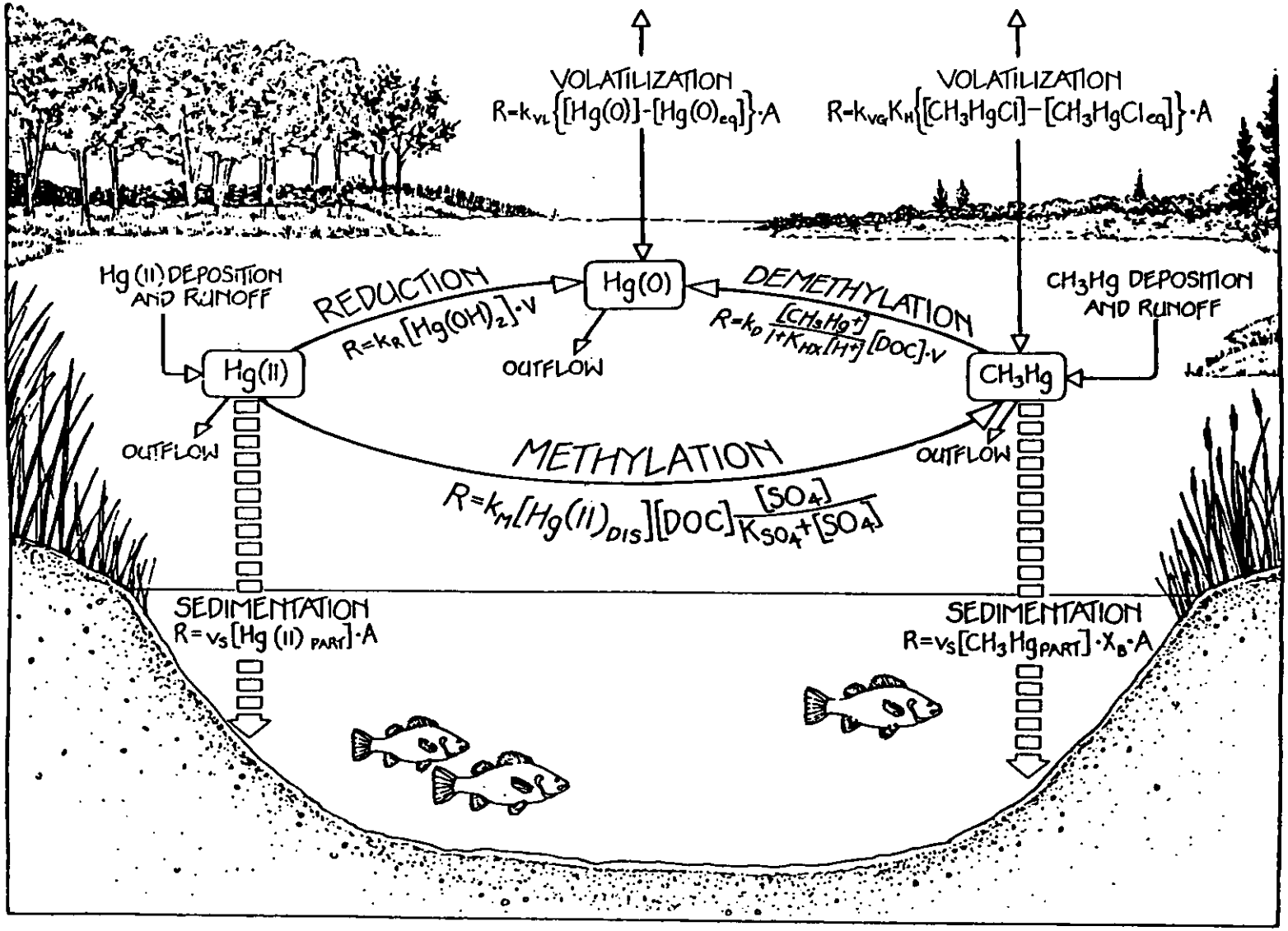

Figure 4-8
Schematic depiction of the lake mercury cycle in the Mercury Cycling Model. The rate laws for each major process derived from fitting the Mercury in Temperate Lakes study data are shown (data from Hudson et al. 1994).

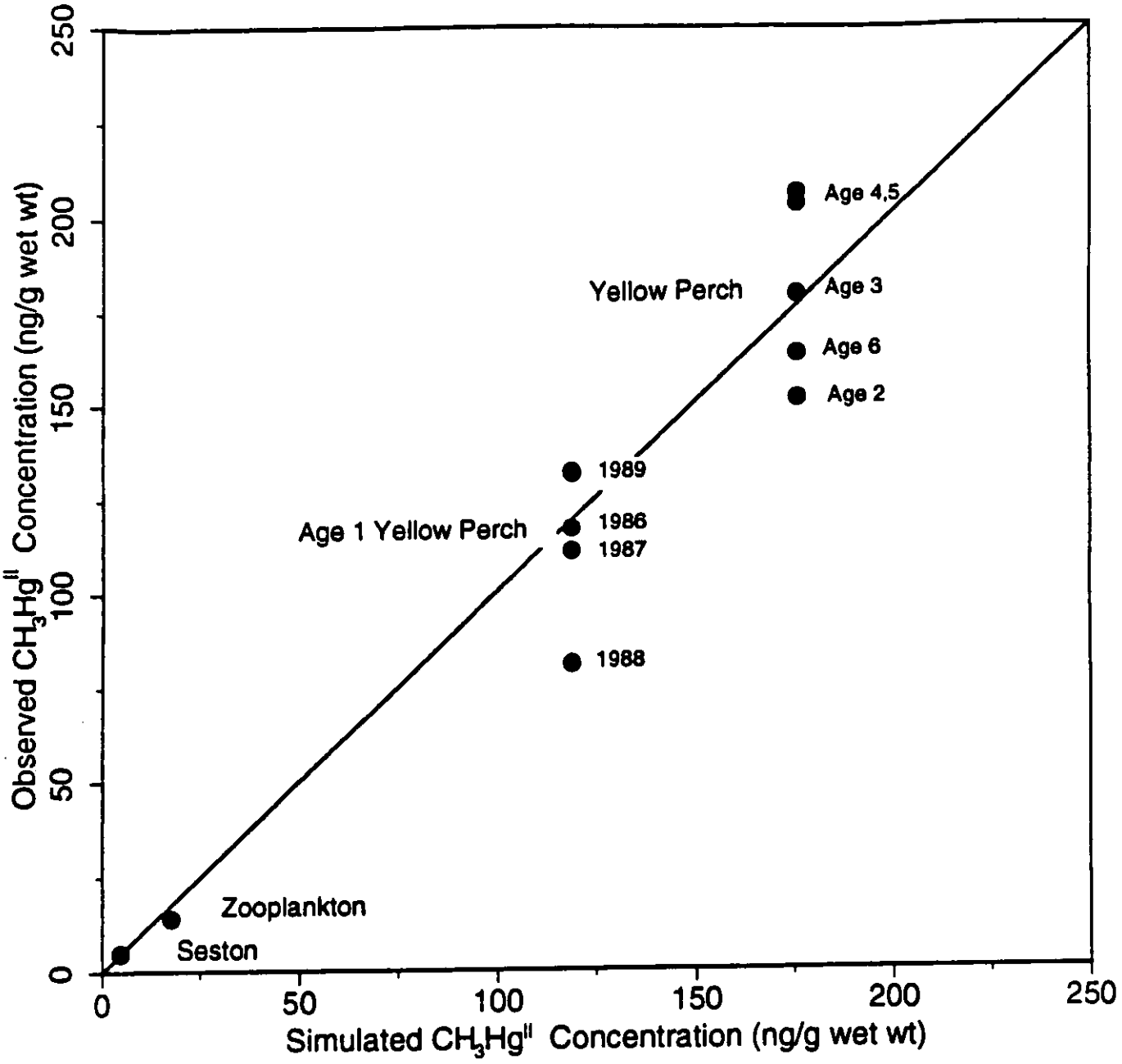

Figure 4-9
Correspondence of observed methylmercury concentrations in biota from Little Rock Lake reference basin and calibration values from the Mercury Cycling Model (from Hudson et al. 1994)

model simulates chemical speciation [Hg(0), Hg(II), CH_3Hg^+] for the seven lakes (Figure 4-10) (Hudson et al. 1994).

Four scenarios briefly illustrate how the MCM can predict methylmercury concentrations under different conditions (Figure 4-11). The graph shows how methylmercury in piscivorous fish responds over a 10-year period to changes of initial conditions in the Little Rock Lake treatment basin. The fish modeled in the treatment basin do not represent sport fish communities that contain larger and older fish species with higher mercury concentrations (Grieb et al. 1990). The base case for the simulation shows that fish mercury varies seasonally around a mean concentration of 0.18 ppm (wet); the MCM calculates concentrations from seasonally variable predictions of methylmercury in the piscivorous fish compartment and the predicted biomass, producing each annual cycle. If atmospheric mercury deposition were to be reduced by 5 percent, a small change in fish methylmercury would be first observed about eight years later, reflecting the small change in deposition and a long lag in response time of organisms with long life cycles.

The other two scenarios shown in Figure 4-11 describe conditions that affect the rate of methylmercury production. Detrital particles include clay and organic materials that compete with methylating processes for Hg(II). If the detrital particles were to decrease by a factor of 10, more Hg(II) would be available for methylation, and fish bioaccumulation would increase, as shown in the simulation. Analogously, an increase by a factor of 2 in the rate of demethylation lowers bioaccumulation in fish. In both cases, the simulated steady-state for the annual cycle does not occur until after year 10 due to the lag in response time associated with organisms of longer lifespan.

The illustration of model capabilities shows how risk assessment can be performed for different mitigation alternatives. As mercury inputs are changed with different mitigation alternatives or mercury controls, the MCM can calculate mercury concentration in fish at a specific site, and thereby simulate exposures for humans and fish-eating wildlife. More generic and broadly applicable assessments can be made with regionally averaged or quartile-range characteristics of lakes having regional deposition measurements. These approaches are discussed further in Section 6.

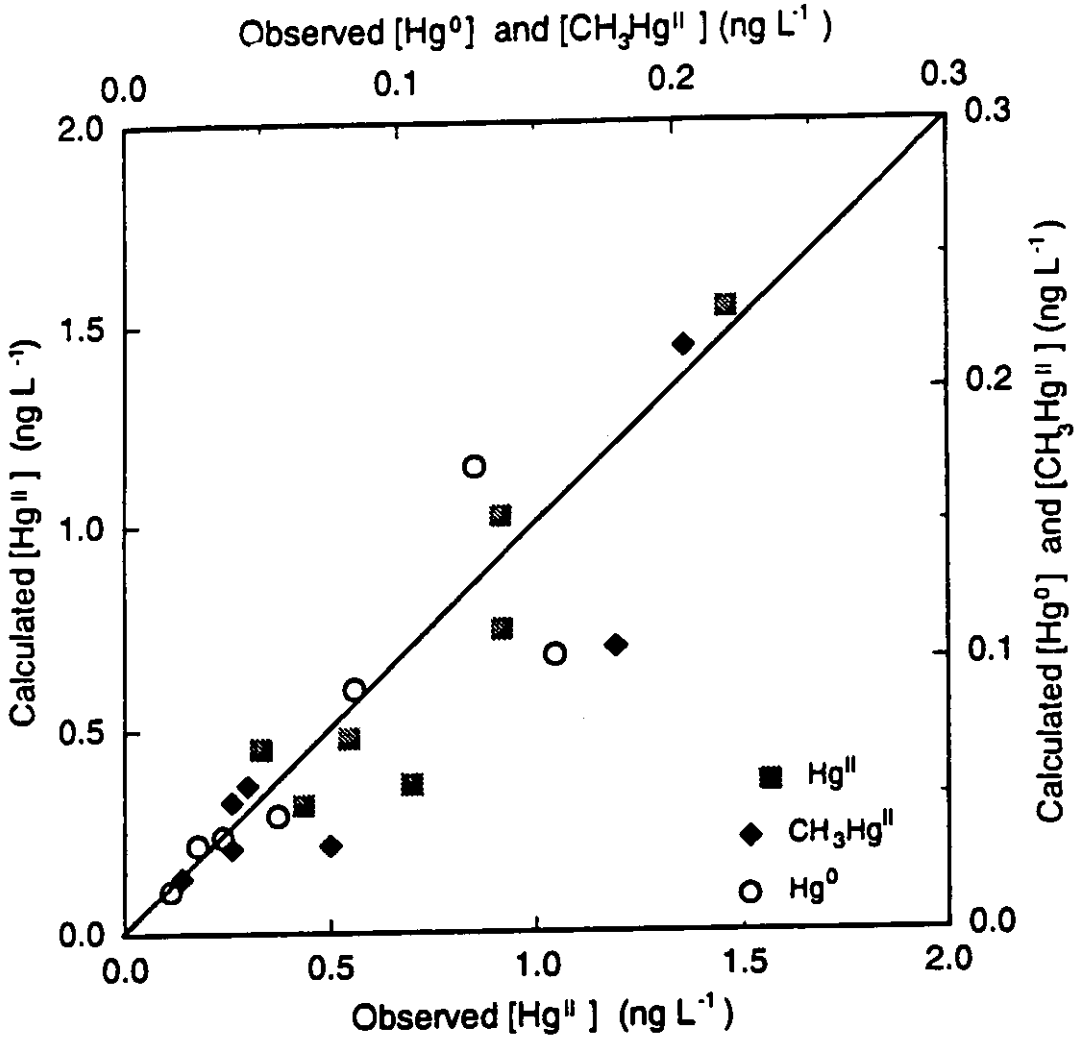

Figure 4-10
Comparison of average observed concentrations of dissolved mercury species with values calculated using the Mercury Cycling Model (from Hudson et al. 1994)

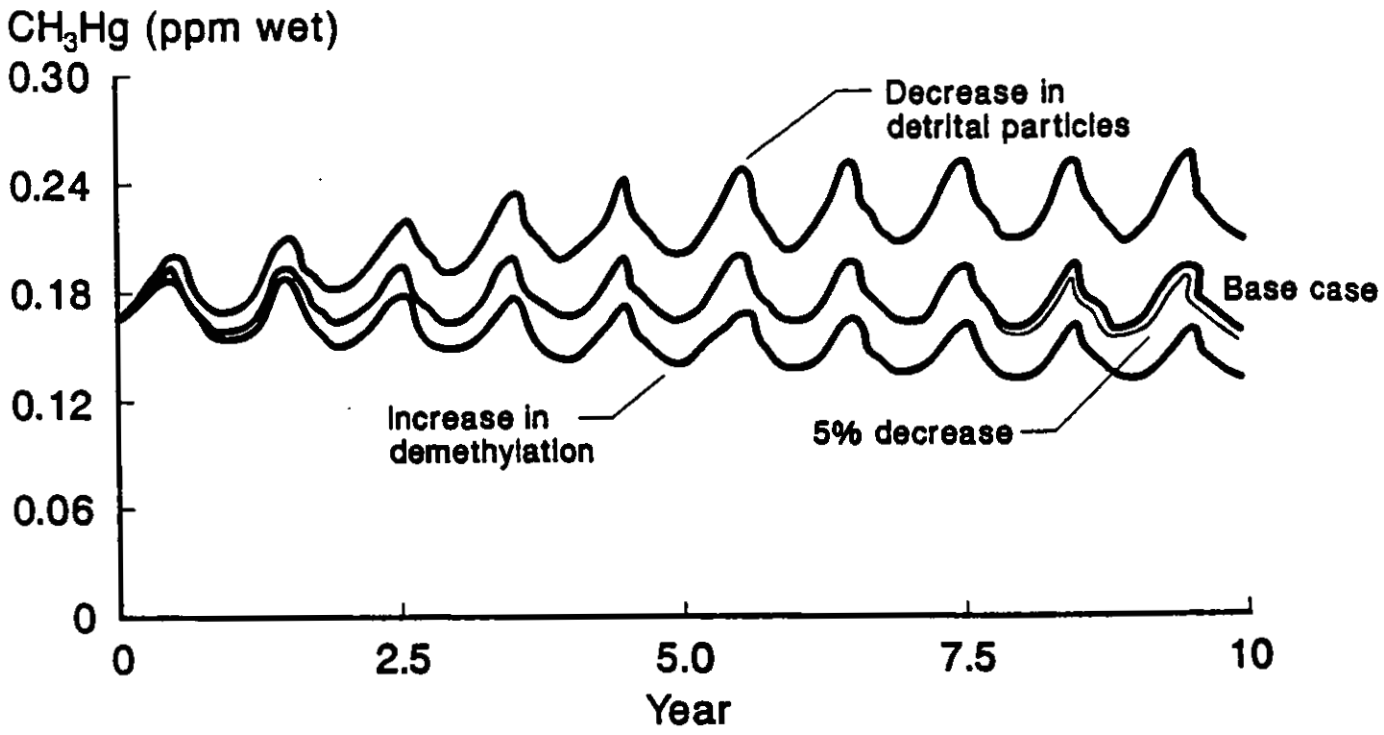

Figure 4-11
Illustration of Mercury Cycling Model simulation of different scenarios compared to the base case (Little Rock Lake reference basin): (1) the effect of a decrease in detrital particles by a factor of 10; (2) the effect of increased demethylation; and (3) the effect of a 5 percent decrease in atmospheric deposition

Effects

The risk to natural populations from atmospheric sources of mercury is not well-documented, and conventional wisdom suggests that aquatic organisms are not at risk (Table 4-2). Moreover, fish do not seem adversely affected until concentrations reach levels much higher than those that typically occur in natural waters at the present time (Wiener and Spry, 1994); a possible exception may be fish reproductive processes (Wiener and Spry, 1994). Piscivorous birds and other organisms that feed on fish may accumulate sufficient mercury to interfere with their survival (Zillioux et al. 1993).

Table 4-2
Mercury Effects on Freshwater Aquatic Biota and Birds
(Summarized from Zillioux et al. 1993)

Biotic Group	Hg Species	Thresholds of Effects
		Water Concentrations, ng/l
Algae	Organo-Hg	300-600
Planaria	Methyl-Hg	30-100
Cladocera	Hg(II)	5200-9300
Fish	Hg(II)	100-3000
	Methyl-Hg	40-1800
		Residues, μg/g
Birds		
Eggs	Methyl-Hg[a]	1-5
Liver	Methyl-Hg[a]	5

[a]Natural food assumed as Methyl-Hg.

Section 5
HEALTH EFFECTS OF MERCURY

Introduction

The National Institute of Environmental Health Sciences (NIEHS) is required by law to transmit to Congress "a study to determine the threshold level of mercury exposure below which adverse human health effects are not expected to occur. Such study shall include a threshold for mercury concentrations in the tissue of fish which may be consumed (including consumption by sensitive populations) without adverse effects to public health" (CAAA 1990).

Mercury exists in several chemical forms (species) that have varying human health impacts when encountered in high doses. Elemental mercury vapors at very high exposures can cause a neurotoxic response—the "mad hatter" syndrome—when inhaled. Inorganic mercury salts corrode tissues when touched or swallowed and damage the gastrointestinal tract, kidneys, and possibly the liver. Significant exposures to these forms of mercury have generally occurred only in occupational environments under conditions not found in modern industrial countries. Organic methylmercury can cause neurological problems such as paresthesia (numbness and tingling in the extremities) if ingested in toxic quantities by adults and can cause psychomotor retardation in children exposed before birth. Almost all concern about potential health effects of mercury in the environment centers on methylmercury exposure.

People are exposed to methylmercury primarily when they eat mercury-contaminated fish. Methylmercury bioaccumulates in fish muscle tissue, is eliminated very slowly, and can cause neurological problems in animals or humans who consume the fish. Accordingly, this section will focus on the health effects of ingested methylmercury.

The Basis for Health Studies of Methylmercury

Historical episodes of catastrophic exposure have shown that methylmercury ingestion can cause neurological damage. Industrial releases of effluent

methylmercury into Japanese fishing grounds in the 1950s and 60s resulted in deaths or neurological injury to several hundred people who ate fish contaminated by the chemical (Harada 1966). In 1973, thousands of Iraqis suffered temporary or permanent nervous system damage when they ate bread baked with flour mistakenly milled from seed grain treated with a methylmercury fungicide (Bakir et al. 1973).

Although these poisoning episodes have called attention to methylmercury toxicity, chronic exposure at very low doses is more common and, therefore, needs assessment. Since mercury is ubiquitous in the environment and enters the food chain in the form of methylmercury, those seeking to protect public health must determine a safe chronic level of methylmercury ingestion.

The concept of a Reference Dose (RfD) is used to define a safe level of exposure. According to the USEPA, an RfD is "an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious health effects during a lifetime." Calculating the current USEPA RfD for methylmercury involved several steps. Relying on published studies of methylmercury poisoning in the Iraqi incident (Bakir et al. 1973; Clarkson et al. 1975), USEPA officials first determined that central nervous system (CNS) disturbance was the critical effect of such poisoning . Then they calculated a lowest-observed-adverse-effect level (LOAEL) of exposure associated with CNS disturbance in the most susceptible individuals. This calculation assumed a threshold below which no adverse effects occur. Finally, USEPA reduced the LOAEL by an uncertainty factor of 10 to accommodate variability among individuals:

$$\text{RfD} = \text{LOAEL}/\text{Uncertainty Factor} = \text{mg/kg/day}$$

The current RfD for methylmercury is 0.3 μg/kg (body weight)-day, or 21 μg/day for a "standard man" weighing 70 kg. This value is somewhat lower than the exposure limit of 30 μg/day set by the World Health Organization (Clarkson et al. 1985).

Because the present RfD has been calculated using data from the Iraqi poisoning episode, it is based on short-term exposure to high doses of methylmercury. But that situation is not typical of the low-level long-term exposure experienced by most fish eaters. Thus, the present RfD for methylmercury may be very conservative, and an RfD is needed that is more relevant for people exposed at low doses over longer periods of time.

It is important to note that the RfD is set at an exposure level designed to protect the "most susceptible individual." Analysis of data from the Iraqi incident showed that the methylmercury ingested at high levels by mothers and circulating in their blood created a risk of psychomotor retardation to their unborn children (Marsh et al. 1981). This risk may be explained by neurophysiological evidence showing that methylmercury disrupts normal cell division and migration during a critical period of brain development (Sager et al. 1986). Thus, the unborn fetus is likely to be the most susceptible individual with respect to CNS effects of methylmercury exposure.

Population studies relating maternal dose during pregnancy to children's responses on a variety of neurological and performance tests have been completed in Iraq (Marsh et al. 1980, 1981, 1987), Canada (McKeown-Eyssen et al. 1983; Wheatley and Paradis 1994), and New Zealand (Kjellstrom et al. 1986, 1989). Researchers have used information from these studies to assess the health risks of methylmercury employing the health risk assessment estimates derived from statistical modeling. Incorporating epidemiological and toxicological data into statistical models involves uncertainties reflecting the limitations of the data and study design. The better the data and design, the more accurate the final health risk assessment.

Weaknesses of Methylmercury Health Studies

As noted above, the current RfD for methylmercury is based on a retrospective study relying on observations after the fact at a disaster site. The major developmental health endpoints in the Iraqi study were non-standard measures, such as time-to-talking or time-to-walking, reported by adults asked to recall their observations of children. However, if we want to learn about the risks associated with more typical environmental exposures, data sets based on

prospective studies with rigorous experimental design are needed. These studies should use well-defined health endpoints (such as developmental effects measured by a test battery), exclude potential confounding factors (such as other chemical exposures and/or socioeconomic or cultural factors), and examine health effects in everyday long-term, low-level exposure settings.

Current Research

An important unanswered question remains: What is the health risk associated with different levels of methylmercury exposure? To answer this risk-based question, researchers must describe dose-response functions rigorously to relate levels of exposure to changes in health resulting from neurotoxicity.

This task is difficult because the neurotoxicity of methylmercury is complex. There are different endpoints reflecting multiple biological effects, such as changes in sensory or motor function or cognition. Some effects, such as paresthesia, increase in severity with increasing dose, while others occur only at higher doses and may reflect damage at a different neurological site or by a different mechanism. Moreover, adults may recover from clinical symptoms when exposure ceases. Finally, as we have seen, the fetus is very sensitive to methylmercury if exposure occurs during critical stages of neurogenesis. Thus, any dose-response model used must accommodate the complexity of methylmercury neurotoxicity.

Long-Term Developmental Studies. Several research programs are beginning to address these problems (see Table 5-1 for a summary of major studies). For example, the Danish government is studying the neurobehavioral effects of intrauterine exposure to methylmercury among residents of the Faroe Islands located in the North Atlantic between Scotland and Iceland (Grandjean et al. 1992; Grandjean and Weihe 1993). They have collected blood and hair samples from 1000 mothers and the babies who were born to them over a two-year period at regional island clinics. Analysis of these samples has shown that maternal—hence fetal—methylmercury exposures are primarily determined by Faroese consumption of pilot whale meat containing an average of 3.3 μg/g total mercury, about half of which is methylmercury. The study is also examining confounding factors such as the influence of methylmercury on birth weight, the

Table 5-1
Summary of Studies Assessing Health Effects From Methylmercury Exposure

Exposed Population	Exposure to Methylmercury	Assessment Methods	Health Effects	Relevance to Health Risk From Utility Emissions	References
• several hundred Japanese living near Minimata Bay	• short-term exposure to high dose for those who ate fish and shcllfish contaminated by industrial chemical release	• retrospective observation	• neurological damage and, in some cases, death	• less relevant because exposure is short-term, dose is high, and information about health effects is based on after-the-fact observations	• Harada 1966
• 81 pregnant Iraqi mothers and their fetuses (the total exposed population included thousands of people and was therefore much larger than this study sample)	• short-term exposure to high dose for those who ate bread made with contaminated wheat flour • possible exposure during "critical period" of neurological development for the fetus	• retrospective study • measurement of peak mercury concentration in mothers' hair during pregnancy (surrogate for fetal dose) • neurological and performance tests • parent recall of time-to-walking and time-to-talking for children	• delayed motor and cognitive development in children	• less relevant because exposure is short-term, dose is high, the sample is small, and information about health effects is based on after-the-fact observations and parent recall • USEPA places "medium" confidence in its RfD of 0.3 μg/kg (body weight)-day based on these data	• Marsh et al. 1987, 1981, 1980 • Clarkson et al. 1975 • Bakir et al. 1973
		• reanalysis using parametric dose-response model	• estimates a NOAEL of 10 ppm mercury in maternal hair, corresponding to an RfD of 0.07 μg/kg (body weight)-day maternal intake	• less relevant because threshold models such as the one used give "model-dependent" results	• Cox et al. 1989

(cont.)

Table 5-1 (cont.)
Summary of Studies Assessing Health Effects From Methylmercury Exposure

Exposed Population	Exposure to Methylmercury	Assessment Methods	Health Effects	Relevance to Health Risk From Utility Emissions	References
• 38,571 indigenous peoples, such as the Cree, in Canada—includes pregnant mothers and their fetuses	• long-term exposure to low dose for those who ate contaminated fish or sea mammals • possible exposure during "critical period" of neurological development for the fetus	• retrospective study (adults) • prospective study (children) • measurement of methylmercury concentration in blood, including umbilical cord samples (surrogate for fetal dose) • measurements of methylmercury concentration in mothers' hair during pregnancy (surrogate for fetal dose) • neurological examination	• no definitive diagnosis of methylmercury poisoning in adults or in children exposed *in utero*	• more relevant because exposure is long-term, dose is low, and the sample is very large • less relevant because only those health effects that would appear on a neurological exam were considered, and neurological exams were offered only to the 608 people with very high exposure (methylmercury > 100 µg/l in blood)	• Wheatley and Paradis, 1994 • McKeown-Eyssen et al. 1983
• 1000 pregnant Faroe Island mothers and their fetuses	• long-term exposure to low dose for those who ate contaminated whale meat • possible exposure during "critical period" of neurological development for the fetus	• prospective study • measurement of mercury concentration in mothers' hair and blood during pregnancy (surrogate for fetal dose) • measurement of mercury concentration in children's hair and blood • assessment of confounding factors such as methylmercury influence on birth weight, selenium in whale meat, and maternal alcohol consumption during pregnancy • long-term developmental assessment of children	• results are not yet available	• more relevant because exposure is long-term, dose is low, the sample is large, and the study has a prospective design that considers confounding factors as well as a broad range of developmental health effects that may be seen over time • less relevant because the local population and lifestyle differ significantly from the population and lifestyle of the United States	• Grandjean and Weihe 1993 • Grandjean et al. 1992

(cont.)

Table 5-1 (cont.)
Summary of Studies Assessing Health Effects From Methylmercury Exposure

Exposed Population	Exposure to Methylmercury	Assessment Methods	Health Effects	Relevance to Health Risk From Utility Emissions	References
• 779 pregnant Seychelles Island mothers and their fetuses	• long-term exposure to low dose for those who ate contaminated fish • possible exposure during "critical period" of neurological development for the fetus	• prospective study • measurement of mercury concentration in mothers' hair and blood during pregnancy (surrogate for fetal dose) • measurement of mercury concentration in children's hair and blood • long-term assessment of children to 5.5 years of age • tests of neuropsychological development cross-culturally validated for Seychelles children and administered by trained local personnel	• results are not yet available	• more relevant because exposure is long-term, dose is low, the sample is large, and the study has a prospective design that considers a broad range of neuropsychological and developmental effects that may be seen from birth through 5.5 years of age • less relevant because the local population and lifestyle differ significantly from the population and lifestyle of the United States	• Davidson et al 1993

(cont.)

Table 5-1 (cont.)
Summary of Studies Assessing Health Effects From Methylmercury Exposure

Exposed Population	Exposure to Methylmercury	Assessment Methods	Health Effects	Relevance to Health Risk From Utility Emissions	References
• 296 pregnant New Zealand mothers and their fetuses	• long-term exposure to low dose for those who ate contaminated fish • possible exposure during "critical period" of neurological development for the fetus	• prospective study • measurement of methylmercury concentration in mothers' hair during pregnancy (surrogate for fetal dose) • standardized cognitive tests such as those for IQ	• methylmercury exposure fails to account for a significant part of the variability in children's test scores	• more relevant because exposure is long-term, dose is low, and the study has a prospective design that considers cognitive effects such as those revealed by scores on standardized IQ tests • less relevant because sample size is relatively small, and the Maori population and lifestyle differ significantly from the population and lifestyle of the United States	• Kjellstrom et al. 1989, 1986
		• EPRI reanalysis using PBPK model and Benchmark Dose statistical method	• estimates a NOAEL of 17 ppm mercury in maternal hair which corresponds to a fetal brain tissue concentration of methylmercury on the order of 50 ppb (µg/L) • this fetal brain concentration would result from a maternal dietary intake of methylmercury ranging from 0.8 to 2.5 µg/kg (body weight)-day.	• more relevant because the statistical analysis employed uses all of the information in the data set and the physiologically based pharmacokinetic model allows extrapolation from measured maternal hair concentration to fetal dose	• Crump 1994 • Gearhart et al. 1994

presence of selenium and PCBs in pilot whale meat, and maternal alcohol consumption during pregnancy. Faroese children born during the study are being followed and will be tested for developmental effects of methylmercury exposure. This study is important because it represents a prospective, long-term developmental assessment.

The NIEHS and the Ministry of Health, Republic of Seychelles, are co-sponsoring a study of 779 mother-infant pairs living in the Seychelles Islands located in the Indian Ocean off the coast of Africa. Seychelles Islanders consume a fish diet thought to be high in methylmercury. Researchers have tracked pre- and postnatal exposure of the children involved and have inferred body burdens of methylmercury from blood and hair samples. This long-term study has followed children up to 5.5 years of age and has evaluate their neuropsychological development using a battery of tests cross-culturally validated for Seychelles children and administered by trained personnel at a local Child Development Center (Davidson et al. 1993). Preliminary analyses of the study results will soon be available.

By focusing on island populations, both of these research programs automatically exclude many factors—such as differing genetic and cultural backgrounds and varying nutritional environments—that could confound the influence of methylmercury on human health. Both are well-designed long-term efforts that should yield valuable data sets for further analysis. However, neither of these exposed populations enjoys a lifestyle similar to that of the United States. For example, although the Faroe Islands are administered by the Danish government, citizens there eat few green vegetables, may drink large quantities of alcohol, and dine on cured meat. Therefore, extrapolating conclusions from these studies to U. S. populations will require careful analysis.

EPRI's Mercury Health Research Program. As a basis for better understanding the effects that chronic low doses of methylmercury can have on the central nervous system in children, EPRI has developed a physiologically based pharmacokinetic (PBPK) model to describe the fate of methylmercury in the body. Using information from this model, in conjunction with new statistical procedures, EPRI researchers are reanalyzing available epidemiology data to

improve dose-response estimates, especially for assessing risks borne by children of exposed mothers.

EPRI's PBPK model describes the oral adsorption, distribution, metabolism, and excretion of methylmercury. The goal has been to create a model that will simulate the kinetics of methylmercury in different species simply by changing species-specific parameters. For example, because the model has separate compartments for red blood cells (RBCs) and plasma, it can predict changes in the kinetics of methylmercury related to RBC/plasma ratios that are unique to each species. The PBPK model has been validated with data for rats, monkeys, and people.

The adult PBPK model uses compartments to represent organs, specific tissues, and waste products. The compartments describe methylmercury transport (plasma, kidney, richly and slowly perfused tissues, brain-blood, placenta, liver, gut, RBCs, and brain) and methylmercury reabsorption and excretion (the intestinal lumen, hair, urine, and feces). Conversion of methylmercury to inorganic mercury by flora in the gut and subsequent elimination of inorganic mercury in the feces is the most important mechanism of excretion. (Some methylmercury is excreted in the feces, but most is reabsorbed.) Incorporation of methyl and inorganic mercury in the hair is also a significant mechanism of excretion. Finally, the adult model incorporates a fetal sub-model with four compartments that grow during the time of gestation (fetal plasma, RBCs, brain, and the remaining fetal body). The fetal sub-model is particularly important since one of the purposes of the PBPK model is to describe exposure to the human fetus for developing a reference dose.

The PBPK model accurately describes both the long-term concentrations of methylmercury in specific organs and the clearance of methylmercury from the body following termination of exposure. To date, the model has successfully predicted plasma, red blood cell, brain, and hair levels of both methyl and inorganic mercury in monkeys who have sustained up to four years of continuous oral exposure to 10–300 μg/kg (body weight)-day of methylmercury. The model has made predictions for human volunteers exposed for varying lengths of time to a broad range of methylmercury doses. Thus, the PBPK model

appears to reliably predict physiological changes that covary with exposure to methylmercury.

In EPRI-sponsored research, Gearhart et al. (1994) have used the PBPK model to determine the relationship between maternal intake of methylmercury and measured maternal blood and hair concentrations. Armed with this information, they have estimated fetal *in utero* exposure from maternal hair concentration. Such estimates allow them to reevaluate methylmercury doses for children in data sets from previous studies where maternal hair concentration is known. (Model extensions are planned to describe postnatal exposure, such as that occurring when breast fed children ingest mothers' milk.)

In collaboration with its principal investigators, Gearhart et al. (1994) are reanalyzing the data set from a New Zealand study of mothers and their prenatally exposed children. These mothers ate a steady diet of fish, and analyses revealed more than 6 mg of methylmercury per kg of their hair sampled during pregnancy. This level contrasts with mean values of 2.3 to 3.1 mg mercury per kg of hair that are typical of adults living in the Northern hemisphere (Airey 1983). Using the PBPK model, EPRI researchers have been able to estimate the dose for New Zealand children based on the concentration of methylmercury measured in their mothers' hair. So far, methylmercury exposure has failed to explain a significant part of the variability in scores among these children when they were assessed for cognitive disfunction on standardized tests such as those for IQ. Since socioeconomic factors may contribute much of that variability, EPRI researchers are obtaining socioeconomic profiles for the New Zealand population under study and incorporating those profiles in the reanalysis in cooperation with the principal investigators.

As noted above, the current approach to assessing risk from methylmercury exposure defines a threshold—a LOAEL or NOAEL ("no-observed-adverse-effect-level") reduced by an uncertainty factor of 10. However, the USEPA is currently giving serious consideration to the use of a Benchmark Dose statistical method for setting the methylmercury RfD; it has already performed such an analysis on developmental endpoints (Marsh et al. 1987, 1981, 1980) from the Iraqi data set. In the traditional approach for estimating a NOAEL from animal data, responses at each dose are compared statistically with control responses,

and the NOAEL is defined as the highest dose showing no statistically significant difference. In the case of human epidemiological data, it is necessary to group the observations into arbitrary categories by exposure in order to perform this analysis. In contrast, the Benchmark Dose method uses a statistical dose-response model to calculate a "benchmark dose" (BMD), the dose or exposure predicted to result in a specified amount of increased risk (the "benchmark risk" or BR). By fitting a dose-response model to all of the data, the Benchmark Dose method makes better use of the dose-response information inherent in the original sample and avoids arbitrary categorization of observations.

A statistical lower bound on the benchmark dose has been proposed as a replacement for the traditional NOAEL (USEPA 1990b; Gaylor and Slikker 1990, Kimmel and Gaylor 1988). Table 5-2 presents results from a study recently conducted for the USEPA (Allen et al. 1994; Faustman et al. 1994) comparing benchmark doses associated with differing benchmark risks to traditionally derived NOAELs for 424 sets of animal data. It is clear that use of 0.1 benchmark risk provides a statistical lower bound on the benchmark dose that corresponds most closely to the traditional NOAEL. However, use of 0.1 benchmark risk also makes the RfD more conservative by a factor of about 2 to 3 on average as compared to the RfD derived using the traditional NOAEL approach.

EPRI researchers have developed a method for calculating benchmark doses and their statistical lower bounds (BMDLs) from continuous endpoints (Crump 1994) such as those represented by children's scores on test batteries. They have used this method to reanalyze psychological, behavioral and scholastic data from the study of New Zealand children described above.[1]

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[1] In this application, the researchers chose a nonlinear dose-response model

$$\mu(d) = \mu_0 + \beta d^k$$

where $\mu(d)$ is the mean of the responses associated with a specific dose, d; μ_0 is the mean of the responses for the controls; and β and k are the estimated parameters. When they compared the test scores of prenatally exposed children with those of unexposed controls, they also needed to account for the performance of children in the control group who might score badly on a test for reasons unrelated to methylmercury exposure. Therefore, the researchers estimated that either 5% ($P_0 = 0.5$) or 1% ($P_0 = 0.01$) of the control children would fall in the deficient category. Finally, the model assumed that test scores are normally distributed with a standard deviation, σ, independent of dose. Given these assumptions, choosing $P_0 = 0.01$ and BR = 0.1 is equivalent to

Table 5-2
Comparison of Benchmark Dose for Differing Levels of Risk With the Traditional NOAEL

Benchmark Risk	Benchmark Dose vs. Traditional NOAEL
0.1	BMD < NOAEL by an average factor of 2.9 for 75% of the data sets
0.5	BMD < NOAEL by an average factor of 5.9 for 90–95% of the data sets
0.01	BMD < NOAEL by an average factor of 29 for 95+% of the data sets

defining the benchmark dose as the dose that results in a 10% change in the mean response relative to the standard deviation (that is, as the dose that satisfies $|\mu(d) - \mu_0| / \sigma = 0.1$).

Table 5-3 presents the results of this Benchmark Dose analysis. Depending on the values selected for P_0 (the percentage of control children who might be deficient on a given test) and BR, the Benchmark Dose analysis suggests that the NOAEL for the most sensitive indicator of developmental effects in six-year-old children occurs at approximately 10-31 ppm mercury in maternal hair, with an estimate based on the most reasonable choice of parameters (BR = 0.1 and P_0 = 0.05) of 17 ppm. The most sensitive indicator of effects (that is, the test producing the lowest BMDLs) was the grammar understanding section of the Test of Language Development.

At a NOAEL of 17 ppm mercury in maternal hair, analysis using the PBPK model described above indicates that fetal brain tissue concentrations of methylmercury are on the order of 50 ppb (μg/L). According to the model, this concentration in fetal brain tissue would result from a maternal dietary intake of methylmercury ranging from 0.8 to 2.5 μg/kg (body weight)-day. This broad range of intakes corresponding to a target maternal hair concentration of 17 ppm mercury reflects the high degree of variability among hair-to-intake ratios seen in human studies. These results suggest that the current USEPA RfD for methylmercury of 0.3 μg/kg (body weight)-day adequately protects against developmental effects (Gearhart et al. 1994).

EPRI also continues to investigate the possible relevance of the Iraqi data set to health risk from utility emissions. Since the original analysis (Marsh et al. 1987), upon which the current RfD for methylmercury is based, other researchers have reanalyzed these data on late or retarded development in Iraqi children exposed to methylmercury *in utero*. One reanalysis (Cox et al. 1989) used delayed walking and neurological scores for exposed children to calculate the "best statistical estimate" of the NOAEL as 10 ppm mercury in maternal hair, with a 95% range of uncertainty between 0 and 13.6 ppm. This threshold estimate is equivalent to an RfD of 0.07 μg/kg (body weight)-day (Stern 1993), much lower than the current USEPA RfD of 0.3 μg/kg (body weight)-day. However, preliminary research at EPRI (Crump et al. 1994) indicates that the results from threshold models are very model-dependent and there is a large range for the 95% confidence interval of the threshold. Furthermore, EPRI's analysis indicates that the Benchmark Dose method is probably the best model to use. Finally, the analysis reveals inconsistencies in the Iraqi data that make other data sets based

Table 5-3
Benchmark Dose Analysis of Scores Attained on a Battery of Developmental Tests Administered to New Zealand Children Exposed to Methylmercury *in utero*

P_0 Fraction of Unexposed Population Affected	BR Benchmark Risk Level	BMD Maximum Likelihood Estimate (range,* ppm in maternal hair)	BMDL 95% Lower Bound (range,* ppm in maternal hair)
0.01	0.10	61–379	31–90
0.01	0.05	43–347	22–64
0.05	0.10	34–7221	17–50
0.05	0.05	20–4364	10–30

*Range of values obtained over the various tests used in the epidemiological study.

on long-term exposure at low dose a more suitable basis for setting standards for prolonged human exposure to methylmercury.

Ultimately, EPRI will use these new epidemiological, experimental, pharmacokinetic, and statistical findings in a national health risk assessment for methylmercury.

Section 6
MERCURY RISK ASSESSMENT

Purpose and Approach

Mercury emitted from stacks may later deposit to waterways or ground surfaces where it undergoes a variety of physical, chemical and biological processes that may result in health effects. These include: (1) the transport and transformation of mercury in the atmosphere, surface water, soil, groundwater, and biota (including the food chain); (2) exposure of the public to those chemicals; (3) the absorbed dose of those chemicals; and (4) the associated health response.

This Section summarizes work by EPRI that is reported in greater detail elsewhere; additional information on the mercury risk assessment is found in Chapter 8 and Technical Appendices J and K of the *EPRI Electric Utility Trace Substances Synthesis Report* (EPRI, 1994).

For most trace substances, inhalation is a major route of entry to the human body. For chemicals like mercury that have significant atmospheric deposition rates, bioconcentration characteristics, and multipathway toxicity, the noninhalation pathways (such as ingestion) are dominant. Although mercury vapor presents a noncarcinogenic inhalation risk, mercury compounds are of greater interest primarily due to their ingestion pathway and the tendency of organic mercury (methylmercury) compounds to bioaccumulate in biota, particularly in fish. Mercury exposure is expected to be particularly sensitive to the characteristics of receiving waters, especially those used by anglers who subsist on the fish taken there.

Fully characterizing human health risks of mercury from all exposure pathways requires "multimedia" risk assessment. Assessing risks by all pathways, however, tends to be a complex process, requiring a number of assumptions for which qualifying data are often poor. To provide a standard framework for multimedia risk assessment, EPRI developed the Total Risk of Utility Emissions (TRUE) model, along with model extensions to address uncertainty. Applications of the TRUE model and the uncertainty extensions provide insight

into the multimedia pathways and consequent risks due to stack emissions from power plants of mercury and other trace substances.

Inhalation Risk Assessment

To assess the potential human health impacts due to inhalation of the suite of trace substances emitted from power plants (EPRI 1994), EPRI conducted an inhalation risk assessment that integrates emission estimates, transport and dispersion modeling results, and exposure analyses with standard potency information. For mercury emissions, the risk assessment estimated (noncarcinogenic) risks associated with inhalation exposure over a 70-year timeframe, based on projections of the future industry generation mix for the year 2010. EPRI performed a set of deterministic risk analyses for each of 594 coal and oil plants, and additional sensitivity analyses to examine the impact of varying parameters and alternative modeling assumptions on the risk estimates.

Measures of Risk. The risk measures of primary importance correspond to the four primary exposure measures: the maximally exposed individual (MEI), the hypothetical MEI, the *reasonably exposed individual* (REI), and the average population exposure (EPRI, 1994). For each exposure measure, a hazard quotient is defined for each substance assessed, and a hazard index is the sum of all the hazard quotients for one source. The hazard quotient is calculated as the calculated exposure concentration divided by the appropriate concentration level of concern, usually the federally defined reference concentration (USEPA 1987). For multimedia exposure the hazard index is also summed. The hazard index requires cautious interpretation. It indicates the proximity to noncarcinogenic criteria limits, or the extent to which these limits are exceeded. As the index approaches unity, concern for occurrence of a potential hazard increases, and additional studies are called for to assess risks more accurately for a given instance. The MEI scenario overestimates actual exposure because it combines a number of extremely conservative exposure assumptions. USEPA exposure guidelines (USEPA 1992) stipulate that the MEI, as a bounding estimate, lies outside the range of actual exposures that might be experienced by any individual. To provide more accurate insights on mercury effects from power plants, both MEI and REI exposure assumptions have been evaluated in this study.

Risk Assessment Framework. Mercury emissions were estimated for every identifiable power plant (a total of 594 plants) in the United States with units greater than 25 MWe nameplate capacity predicted to be operating for the year 2010. Long-term ground-level atmospheric concentration patterns within 50 km of each plant were modeled for each plant in the study. These simulations capture the calculated maximum concentrations of all plants and cover the majority of populations likely to be exposed to emissions from one or more plants. The ISCLT2 model was used with the "regulatory default" options (USEPA 1987). The analysis incorporated source characteristics of individual stacks and meteorological data representative of the area around each plant. Each plant was modeled using either "urban" or "rural" dispersion coefficient options, as appropriate for the setting. In lieu of land use data, population data around each plant were used as the selection criteria for choosing the urban or rural option. Other default modeling options were used, such as assuming flat terrain and no plume downwash due to nearby structures. The dispersion modeling generated concentration estimates for trace substances at various locations within 50 km of each plant.

A key part of the exposure assessment was determining where people reside with respect to estimated concentrations due to power plant emissions. For each plant, the assessment mapped 1990 census data onto the 50-km radial domain used in the dispersion modeling, calculated population-averaged concentrations, and identified both the overall maximum concentration and the highest concentration in a *populated* grid cell.

The exposure assessment also developed a measure of exposure, the REI, that is intended to be more realistic than the MEI. The REI scenario focused on an individual living at the point of maximum concentration around a power plant. In this case, activity data, breathing rates, and indoor/outdoor concentration ratios were used to refine the conservative assumptions incorporated into MEI exposure.

The next step used dose-response information tabulated by USEPA as well as by other sources to estimate the potential health effects resulting from exposure to mercury, and to all trace substances (Section 5; also, EPRI 1994, Chapters 7 and

8). For noncarcinogenic effects, the analysis used inhalation reference concentrations, which are based on USEPA reference doses, defined as being levels of daily exposure that are likely to be without observable chronic, deleterious effects. In general, the reference doses are from USEPA's Integrated Risk Information System (IRIS) database.

Hazard Indexes. No plant exceeds the relevant reference concentration (RfC) for any single substance, including mercury, and all plants have a screening-level inhalation hazard index, for all trace substances evaluated,[1] of less than 1 (Figure 6-1). The highest MEI hazard index and highest hypothetical MEI hazard index for a single plant are approximately the same value (0.5). The highest REI hazard index (0.3), from the same plant, is about 65 percent of the highest MEI hazard index, and the REI hazard indexes across all plants range from 21 to 70 percent of the corresponding MEI inhalation hazard indexes.

Figure 6-2 is a distribution of hazard indexes by decile (factors of 10). Fewer than 2 percent of the plants have MEI hazard indexes greater than 0.1, and about 32 percent have MEI hazard indexes between 0.01 and 0.1.

Figure 6-3 shows how the MEI hazard indexes range among different groups of plants. Coal plants with only particulate controls exhibit the highest MEI hazard indexes of all plant groups.

Figure 6-4 shows the contribution to the MEI inhalation hazard index by chemical for various plant groups. The median MEI hazard indexes for the plant groups ranged from 1.5×10^{-3} for gas plants to 1.1×10^{-2} for bituminous-fired coal plants. Mercury contributes less than 15 percent to the median MEI hazard index for all groups of plants, and does not show up as a major contributor to inhalation risk for any of the plant groups.

No measurable effect of alternative future scenarios or sources of uncertainty could be assigned to mercury inhalation risk (see EPRI 1994 for other trace substances).

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[1] Arsenic, Beryllium, Cadmium, Chromium, Manganese, Nickel, Lead, HCl, Mercury, Selenium, Benzene, Formaldehyde, PAH, Tolueme.

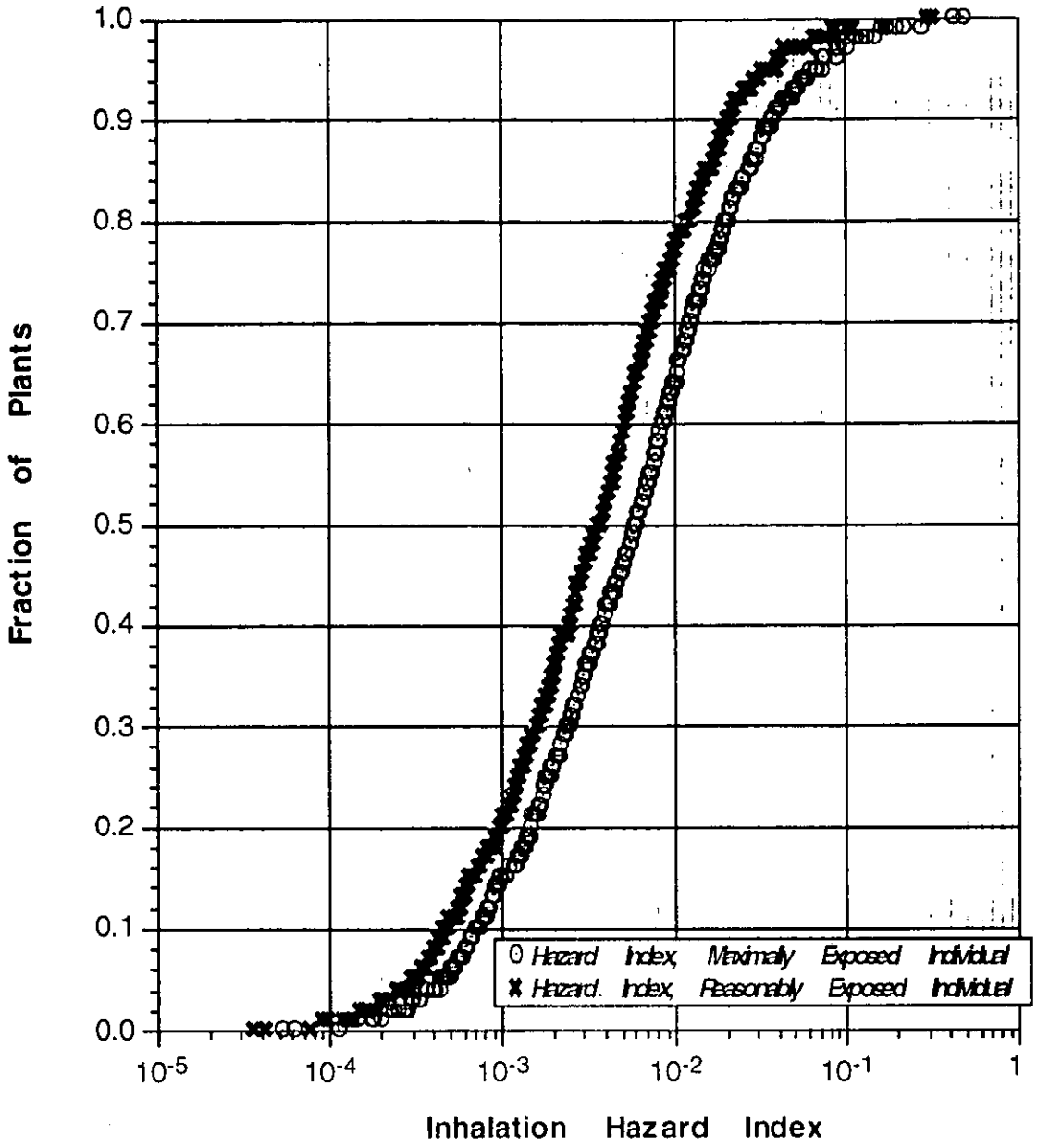

Figure 6-1
Distributions of inhalation hazard indexes for all PISCES trace substances

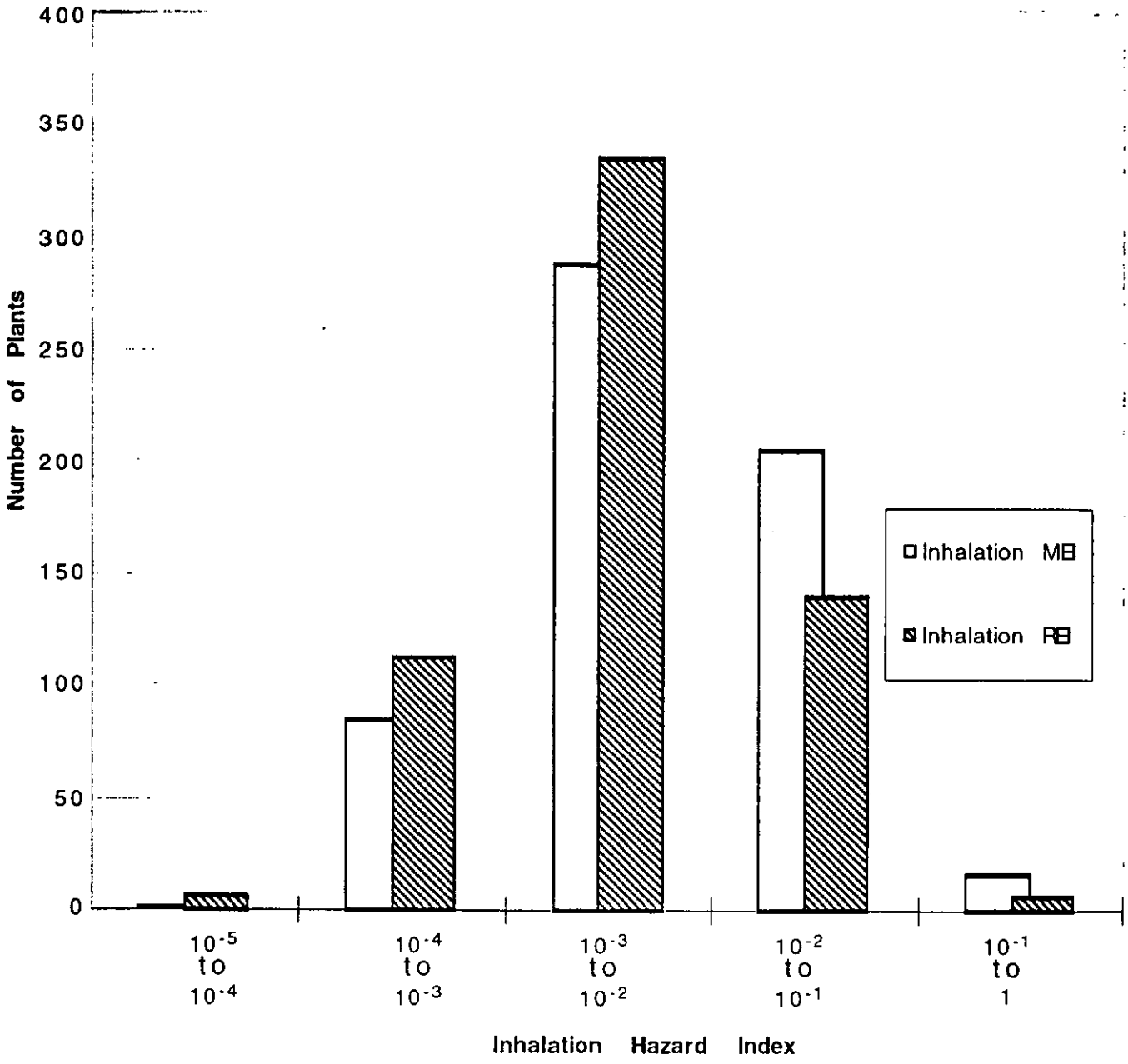

Figure 6-2
Distributions by decile of inhalation hazard indexes for all PISCES trace substances

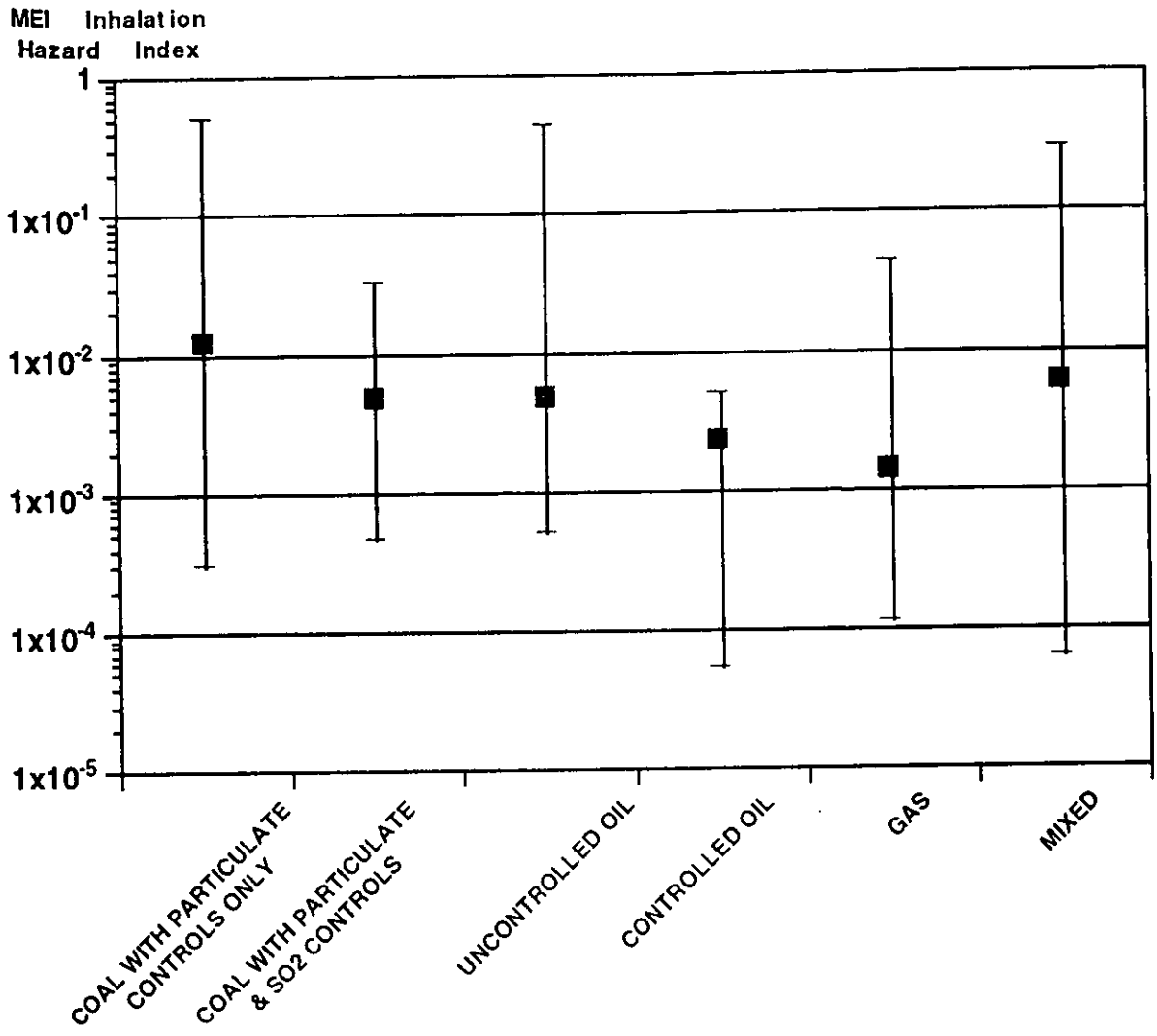

Figure 6-3
MEI inhalation hazard indexes for all PISCES trace substances for different groups of plants

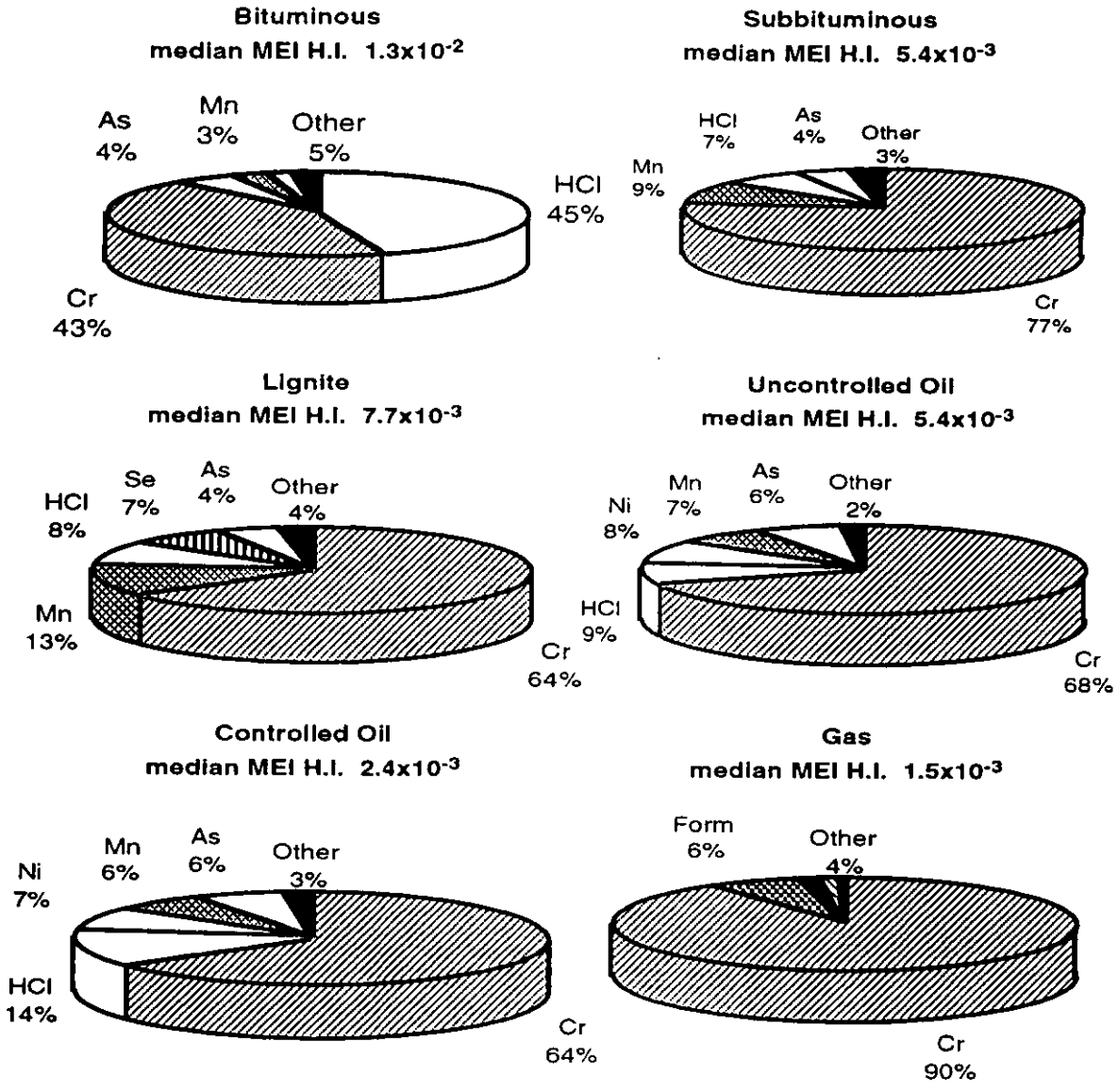

Figure 6-4
Contributions of individual substances to median MEI inhalation hazard indexes for plants grouped by fuel type

TRUE Multimedia Model

For individual power plants, mathematical modeling of the health risk is carried out by combining appropriate models of transport and fate, exposure, dose, and health risk. The selection of these models is critical because a balance must be maintained between the availability of data needed to perform the modeling and the accuracy desired for the health risk estimates.

A two-level approach to the multimedia modeling of health risk has been adopted for assessing mercury risks. In this approach, an initial screening analysis estimates health risks with a relatively simple assessment model. Such a screening-level analysis is intended to provide a moderate overestimate of the health risks using an easily manageable set of data and readily available models. If the results of this screening analysis show that the estimated health risks for particular substances or pathways warrant further analysis, a more detailed assessment can be conducted to refine the modeling of important pathways, exposure routes, and health effects.

The TRUE model is a tool for multimedia health risk assessment that can handle both screening-level and detailed site-specific analyses. The model combines a number of individual models to handle the transport and fate of chemicals in the atmosphere, surface water, surface soil, groundwater, and the food chain (Table 6-1). Major intermedia transport processes are also included. Chemical concentrations calculated by the fate and transport models are used with exposure-dose models to calculate the individual doses, which are then used to calculate health risks.

TRUE takes as input power plant emissions, physical characteristics of the environmental media, food and water consumption information, and health effect parameters for the chemicals of interest, and provides an output of environmental concentrations, exposure doses, and human health risks.

TRUE Model Screening Application to Mercury. The TRUE model was used in four case studies to perform screening-level multimedia health risk assessments associated with the emissions of fossil-fueled power plants (Table 6-2). This section provides a summary of the findings of these assessments. The

Table 6-1
Media and Pathways Analyzed by the TRUE Model

Environmental Media		Exposure Pathways		
Individual	**Intermedia**	**Inhalation**	**Ingestion**	**Dermal Absorption**
Air	**Air-Soil** —dry deposition —wet deposition	**Ambient Air** —gases —particulates	**Soil**	**Soil Contact**
Soil			**Water** —drinking —swimming	**Water Contact** —swimming —showering
Surface Water	**Soil-Surface Water** —overland runoff			
Groundwater	**Soil-Groundwater** —infiltration		**Food Chain** —produce —fish —beef —dairy milk —mother's milk	

Table 6-2
TRUE Case Studies—Facility Characteristics

	Site 12	Site A	Site B	Site C
Fuel	bituminous coal	subbituminous coal	bituminous coal	residual oil
Particulate Control	ESP	ESP	ESP	ESP
SO_2 Control	wet limestone FGD	wet limestone FGD	coal blending	—
Stack Characteristics				
—temperature (K)	323	359	415	444
—velocity (m/s)	13	22	26	17.3
Wind Direction	west/southwest	south	northwest	west
Hydrological Balance				
—precipitation (cm)	95.5	86.4	104.0	119.0
—evapotranspiration (cm)	65.5	69.6	51.0	85.0
—overland runoff (cm)	14.0	16.5	19.0	14.0
—infiltration (cm)	16.0	0.3	34.0	20.0
Surface Water Bodies	1 large river 1 large lake	1 river 2 lakes 2 creeks	5 rivers 1 small reservoir	1 large river 1 small river 2 small reservoirs
Groundwater System	N/A			
—unsaturated zone (m)		8.0	8.5	11.0
—saturated zone (m)		11.0	30.0	61.0
Environment	rural	rural	rural	urban
Terrain	flat	rolling hills	rolling hills	rolling hills
Max χ/Q				
—distance/direction (cm)	E/10-20	N/0-10	E/20-30	E/10-20
—value $(\mu g/m^3)/(g/s)$	2.0×10^{-3}	1.6×10^{-3}	2.9×10^{-3}	1.6×10^{-2}

ESP: Electrostatic Precipitator
FGD: Fuel Gas Desulfurization
N/A = not applicable

emission data for mercury were based on field measurements at the power plants by EPRI's PISCES research program (see Section 2 for background discussion).

The four plants rank approximately in the middle of the distribution of U. S. power plant mercury emissions. The range of mercury emissions (3–44 kg/yr) ranked at the 38th to 98th percentile of all mercury emissions from power plants (Table 6-3). Mercury deposited to the ground surface via calculated wet and dry deposition for each power plant location was transported via modeled surface and groundwater pathways to crop plants and aquatic ecosystems. At the screening level, the currently documented USEPA bioconcentration factor (of 33,000) was used to calculate fish-flesh concentration of total mercury. This in turn was compared to the current federal reference dose for mercury in food fish to compute the hazard index for mercury for each location. For the four units, the hazard index ranged between 0.00023 to 0.093. When additional site-specific information becomes available, this screening analysis can be combined with additional modeling to obtain more accurate risk assessments.

TRUE-MCM Combined Analysis. Due to the complexity of mercury behavior and the significance of the aquatic environment in the concentrations of mercury in fish, a reliable assessment of mercury-related risks requires comprehensive treatment of the cycling of mercury in surface water bodies beyond the screening-level bioconcentration factor. For that purpose, a steady-state version of EPRI's Mercury Cycling Model (see Section 4; also, see Hudson et al. 1994, and EPRI 1994, Technical Appendix N) was combined with TRUE for the evaluation of risks associated with mercury power plant emissions. The MCM input parameters include mercury loads and hydrologic and chemical lake characteristics, with output of mercury concentrations in lake water and in fish.

The combined approach was used to evaluate the multimedia risks associated with the mercury emissions of TRUE case study sites 12 and C (Table 6-4). In this exercise, all characteristics of the sites were maintained as in their actual environments, with the exception of the water bodies, for which alternative lakes were substituted. These were nearby lakes for which data necessary for the MCM analyses were available. For each site, two alternative lakes were analyzed to evaluate the sensitivity of mercury risks to the characteristics of the aquatic environment.

Table 6-3
Screening Studies:
Mercury Emissions for Power Plants—Case Studies

Site	Mercury Emissions		Percentile Rank Among 594 Coal-Fired Power Plants	Presence of Potential Fish Population[a]	Hazard Index for Mercury[b]
	kg/yr	g/s			
12	9.46	0.0003	42	river	3.3×10^{-4}
A	44.2	0.0014	77	no lakes	2.8×10^{-1}
B	23.0	0.00073	61	no local fishing	3.6×10^{-3}
C	2.93	0.000093	36	marine fish only	1.8×10^{-4}

[a]Sites 12 and C were the only sites assessed for fish ingestion because, to affect fish-related risk appreciably, a freshwater aquatic environment was required to be present within the 50-km radius.
[b]Calculated as the ratio of exposure divided by the mercury RfD (see EPRI 1994).

Table 6-4
Characteristics of the Lakes Used in the TRUE-MCM Combined Analysis

		Lake I[1]	Lake II[1]	Lake III[2]	Lake IV[2]
Hydrologic Parameters	hydrologic status	drainage	reservoir	reservoir	drainage
	surface area (km^2)	166	5.4	0.49	0.56
	watershed area (km^2)	2027	1937	2.8	3.6
	mean depth (m)	54	6.4	2.4	3.5
	residence time (yr)	9.5	0.05	0.68	0.8
Water Quality Parameters	nutrient status	mesotrophic	eutrophic	hypereutrophic	mesotrophic
	pH	8.2	8.0	7.5	4.9
	DOC (mg/l)	2.6	6.8	4.8	0.67
	POC (mg/l)	0.8	1.7	1.0	0.07
	Cl^- (µeq/l)	1363	1207	422	112
	Ca^{++} (µeq/l)	2092	2227	371	34
	$SO_4^=$ (µeq/l)	719	1119	159	128
	Chlorophyll*a*, (µg/l)	1.4	25.9	24.0	0.2

[1]Lakes I and II were used in the analysis of Site 12.
[2]Lakes III and IV were used in the analysis of Site C.

Table 6-5
MCM-True Combined Analysis—
Predicted Mercury Concentrations and Methylmercury Water-to-Fish BAFs

	Site 12		Site C	
	Lake I	**Lake II**	**Lake III**	**Lake IV**
Water Concentrations (mg/l)				
Hg(II)	2.6×10^{-9}	4.2×10^{-7}	4.6×10^{-8}	1.5×10^{-7}
Methylmercury	1.6×10^{-9}	5.7×10^{-8}	1.6×10^{-8}	1.2×10^{-8}
Hg(0)	4.8×10^{-8}	1.5×10^{-7}	8.5×10^{-9}	0.0
Methylmercury BAF_f[1] (l/kg)	3.7×10^{4}	1.5×10^{4}	1.3×10^{5}	3.6×10^{6}
Methylmercury Concentrations in Fish (ppm)	5.9×10^{-5}	8.5×10^{-4}	2.0×10^{-3}	4.1×10^{-2}

[1]Methylmercury BAF_f values provided are based on the *fresh* weight of fish. BAF is a bioaccumulation factor and includes uptake from water and food: BAF = fish concentration/water concentration.

Table 6-4 summarizes the relevant hydraulic and water quality characteristics for the four lakes considered. The lakes used with each power plant were selected on the basis of geographic proximity to the site. Lakes I and II (listed in Table 6-4) were used in the analysis of Site 12, whereas Lakes III and IV were used with Site C.

The resulting mercury concentrations in water, fractions of methylated mercury, and methylmercury water-to-fish BAFs varied among the different lakes, depending on their hydraulic and water quality characteristics, thus causing an equivalent variation in the resulting risks. The results of the MCM simulations for each individual site and lake are provided in Table 6-5.

The estimated multimedia hazard indexes were significantly less than 1 for both sites and all alternative lakes evaluated. Table 6-6 summarizes the corresponding breakdown of the mercury multimedia hazard indexes among the various pathways considered. With more detailed information for individual power plant sites, more accurate risk assessments with less conservative assumptions could be obtained.

Uncertainty Analysis

This section describes TRUE model results to characterize the uncertainty in health risks, and applies the approach to one power plant.

Uncertainties in health risk assessments arise in: (1) the formulation of the models used, and (2) the estimation of the parameter values used as input to these models. The focus in this discussion is on the uncertainties due to the input parameters for a given health risk assessment model. For that reason, and since the uncertainties due to model formulation are not included, the relative magnitude of results (most-likely values versus point-estimate values) is more significant than their absolute magnitude. By characterizing the uncertainties (or variability) in model input parameters and studying the effects of variation in these parameters on the model predictions, an estimate can be made of the part of the uncertainty in the predictions that is due to uncertainty in the inputs. A more detailed description of this application can be found in EPRI (1994).

Table 6-6
MCM-TRUE Combined Analysis —Predicted Mercury MEI Hazard Index

	Hazard Quotient/Hazard Index[1]			
	Site 12		Site C	
Pathway	Lake I	Lake II	Lake III	Lake IV
Ingestion				
produce	2.3×10^{-4}	2.3×10^{-4}	1.7×10^{-4}	1.7×10^{-4}
soil	3.2×10^{-7}	3.2×10^{-7}	2.3×10^{-7}	2.3×10^{-7}
drinking water	5.0×10^{-6}	6.0×10^{-5}	6.7×10^{-6}	1.6×10^{-5}
swimming water	6.1×10^{-9}	7.4×10^{-8}	8.2×10^{-9}	1.9×10^{-8}
fish	1.3×10^{-4}	5.6×10^{-3}	4.0×10^{-3}	7.5×10^{-2}
Subtotal	3.7×10^{-4}	5.9×10^{-3}	4.2×10^{-3}	7.5×10^{-2}
Inhalation				
Subtotal	6.2×10^{-6}	6.2×10^{-6}	7.6×10^{-7}	7.6×10^{-7}
Dermal				
water	5.5×10^{-8}	6.6×10^{-7}	7.3×10^{-8}	1.7×10^{-7}
soil	1.5×10^{-7}	1.5×10^{-7}	1.1×10^{-7}	1.1×10^{-7}
Subtotal	2.0×10^{-7}	8.1×10^{-7}	1.8×10^{-7}	2.8×10^{-7}
TOTAL	3.7×10^{-4}	5.9×10^{-3}	4.2×10^{-3}	7.5×10^{-2}

[1]Hazard Quotients for each pathway (Subtotals) and Hazard Indexes for each lake (Totals).

The case study presented here corresponds to the stack mercury emissions at a hypothetical coal-fired site. The parameters producing maximum risk from all chemicals and all pathways due to exposure to air emissions were identified and used in the uncertainty analysis.

Uncertainties in the estimation of mercury health risks are involved in all parts of the risk assessment procedure including the modeling of environmental and food chain fate and transport, the estimation of individual exposure, and the prediction of health risks. Of the several environmental processes involved in modeling mercury transport, fate, and health effects, particular attention was devoted to: (1) atmospheric chemical transformations, (2) dry deposition, (3) wet deposition, and (4) aquatic chemical transformations.

Sensitivity analysis performed on the various components of the TRUE model resulted in the selection of 26 critical parameters. These included: mercury atmospheric chemistry and physical morphology, dry deposition processes, methylation of mercury in the aquatic environment, food chain bioconcentration factors, and toxicology.

A Latin Hypercube analysis using 10,000 iterations on a simplified version of the TRUE model resulted in a probability distribution of the mercury hazard index. The distribution was positively skewed with a mean of 1.4×10^{-3} (a fraction of the federal reference dose). The hazard index value calculated in the deterministic assessment of MEI for this plant (9.0×10^{-2}) was estimated to fall beyond the 95th percentile of the derived distribution. The corresponding probability density plot is presented in Figure 6-5.

A more extensive discussion of the uncertainties involved in the estimation of mercury health risks and a more detailed description of this application can be found in Technical Appendix M of EPRI (1994).

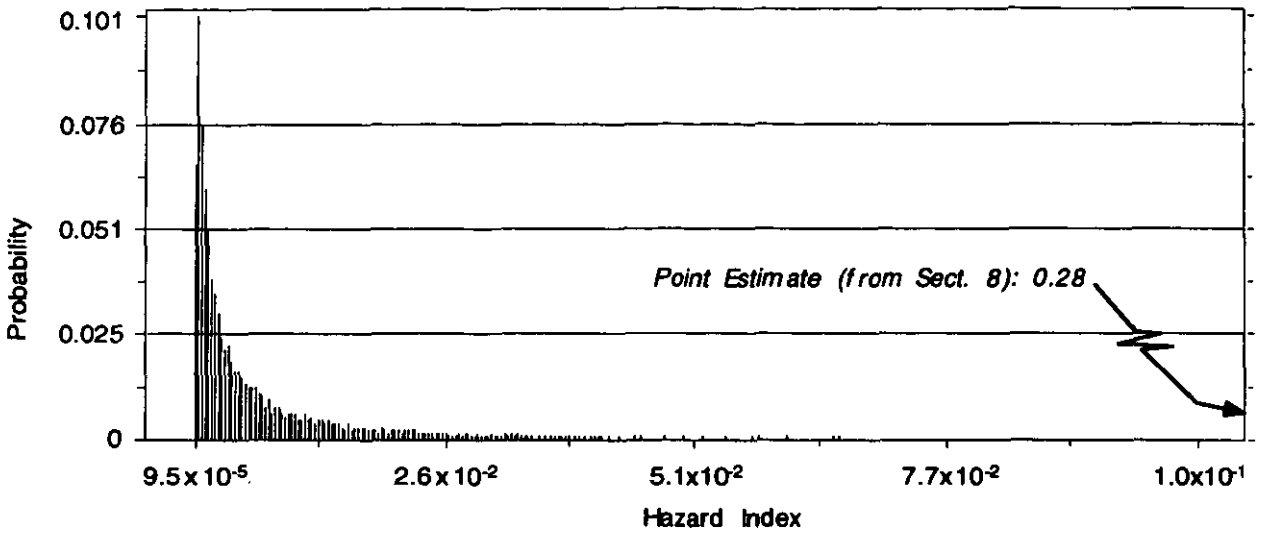

Figure 6-5
Probability distribution of mercury hazard indexes, hypothetical coal-fired case study site

Section 7
POTENTIAL MERCURY CONTROL APPROACHES

Developing Methods for Mercury Removal From Power Plants

Trace metals in flue gas are typically either in the particulate phase or condensed on fly ash particles and can be removed effectively by an efficient particulate collector. Mercury seems to be the most difficult to collect as it appears mainly in vapor form, much of which passes through particulate control devices such as baghouses and electrostatic precipitators.

Methods for mercury capture have been developed mainly for waste incineration plants. These include the direct injection of activated carbon and sodium sulfide, wet and dry scrubbing, and the use of activated carbon beds. Mercury concentrations in flue gas from utility boilers, however, are about two orders of magnitude less than those from waste incineration plants (1 to 10 versus 50 to 500 $\mu g/m^3$) and the gas compositions (including mercury species present) are very different. It is, therefore, uncertain whether any of these methods are transferable to power plants and what the cost implications would be. Compounding these difficulties is the problem of consistently making accurate measurements of the low mercury concentrations present in utility flue gas and the lack of validated methods to quantify different species of mercury present.

It is generally believed that the mercury vapor can exist as either elemental mercury (Hg^0) or as some form of oxidized mercury (e.g., mercuric chloride, $HgCl_2$). These different chemical forms of mercury have markedly different chemical and physical properties and can alter the effectiveness of specific mercury removal methods. Elemental mercury should not be absorbed significantly by water-based processes because it has a very low water solubility. On the other hand, the high water solubility of mercuric chloride suggests that this species should effectively be removed by wet scrubbing. The information that exists for mercury speciation in power plant flue gases has not been reliable. The reported speciation data were usually collected either by the MESA method (Bloom et al. 1993) or by USEPA's Draft Method 29 (USEPA 1990a). The MESA method was specifically designed for speciating mercury in flue gas. USEPA's Draft Method 29 was not designed to speciate mercury, but various researchers

have reported speciation data based on this method. Neither method has been thoroughly evaluated and validated for low flue gas mercury concentrations. In fact, an analytical artifact was found in the MESA method which invalidated initial reports of methylmercury measurements. Section 2 shows that mercury speciation varied significantly from site to site. Limited laboratory tests to date showed that the mercury species present in a specific flue gas will vary as the temperature changes, indicating that the mercury species can change as the flue gas travels from the boiler to the stack.

The development of work to date on mercury control technologies has found mercury removal effectiveness to be strongly dependent on the mercury species present, based on uncertified measurement techniques. Some initial results on two of the mercury removal technologies currently being assessed at EPRI, activated carbon injection and wet scrubbing, are discussed here. Development of other approaches has been very preliminary and is discussed only briefly.

Activated Carbon Injection

The direct injection of activated carbon into the flue gas stream of a utility boiler has been suggested as a relatively simple approach for controlling mercury, as the carbon can be collected in downstream particulate control equipment. This approach has been evaluated fairly extensively for waste incinerators and has been found to be capable of high mercury removal efficiencies. However, because power plant flue gas contains much lower mercury concentrations and because it differs significantly from incineration flue gas in composition and, perhaps, mercury speciation, the effectiveness of activated carbon injection for power plant flue gas mercury removal remains uncertain. For example, waste incinerators normally have high chlorine concentrations in the fuel resulting in generation of mercuric chloride species which are typically easier to remove.

Initial assessment of activated carbon injection was conducted at a 1 MWe transportable pulse-jet baghouse (TPJ) at Public Service Company of Colorado's (PSCCo) Comanche Station and at a bench-scale facility at the Energy and Environmental Research Center (EERC) in North Dakota. Tests were conducted with varying amounts of carbon, with different coals, and at different flue gas temperatures.

Results from the two test facilities indicate that removal of trace mercury levels in flue gas by activated carbon injection depends on flue gas temperature, coal type, mercury species, activated carbon properties and injection rate, flue gas composition, and operating conditions. While high mercury removal efficiencies were observed in some tests, low to moderate removals were measured at other typical power plant conditions. Some preliminary guidelines to improve mercury capture can be established but it is not yet possible to predict with confidence the level of flue gas mercury control achievable with activated carbon injection.

As an example of this difficulty, Figure 7-1 shows the results on total vapor-phase mercury removal from five tests (four at EERC and one at PSCCo) using activated carbon injection ahead of a pulse-jet baghouse (Chang et al. 1993; Miller et al. 1994). The amount of carbon used in each test is expressed as a carbon-to-mercury weight ratio (C:Hg, the weight of carbon injected per weight of mercury present). Lowering the temperature from 345 to 250°F (Tests 1 and 2) improved mercury removal efficiency from 0 to 37 percent. In earlier testing at PSCCo, 98 percent vapor mercury removal was measured at a temperature of 190°F (Chang et al. 1993). Most of these earlier tests were also conducted in flue gas with most of the fly ash removed by injecting activated carbon injection downstream of an existing baghouse and collecting the carbon in a second pulse-jet baghouse. It is uncertain whether this level of removal is achievable for other coals and at other sites. Further, such low temperatures are not normally encountered in boiler flue gas entering the electrostatic precipitator or baghouse. A normal temperature for flue gas at the precipitator or baghouse is 250 to 350°F. Cooling the flue gas to lower temperatures requires the use of large amounts of water injection which can cause significant problems with fly ash deposition and corrosion on the duct walls and the electrostatic precipitator or baghouse. Significant work remains to be done to develop proper spray cooling techniques to avoid some of these problems. The potential impact of moisture and low temperature on precipitator and baghouse operation will need assessment.

Increasing the amount of activated carbon used (Tests 2 and 3) from a C:Hg ratio of 3000:1 to 10,000:1 almost doubled the mercury removal efficiency. However, other tests show that the relation is not linear, and it is difficult to extrapolate

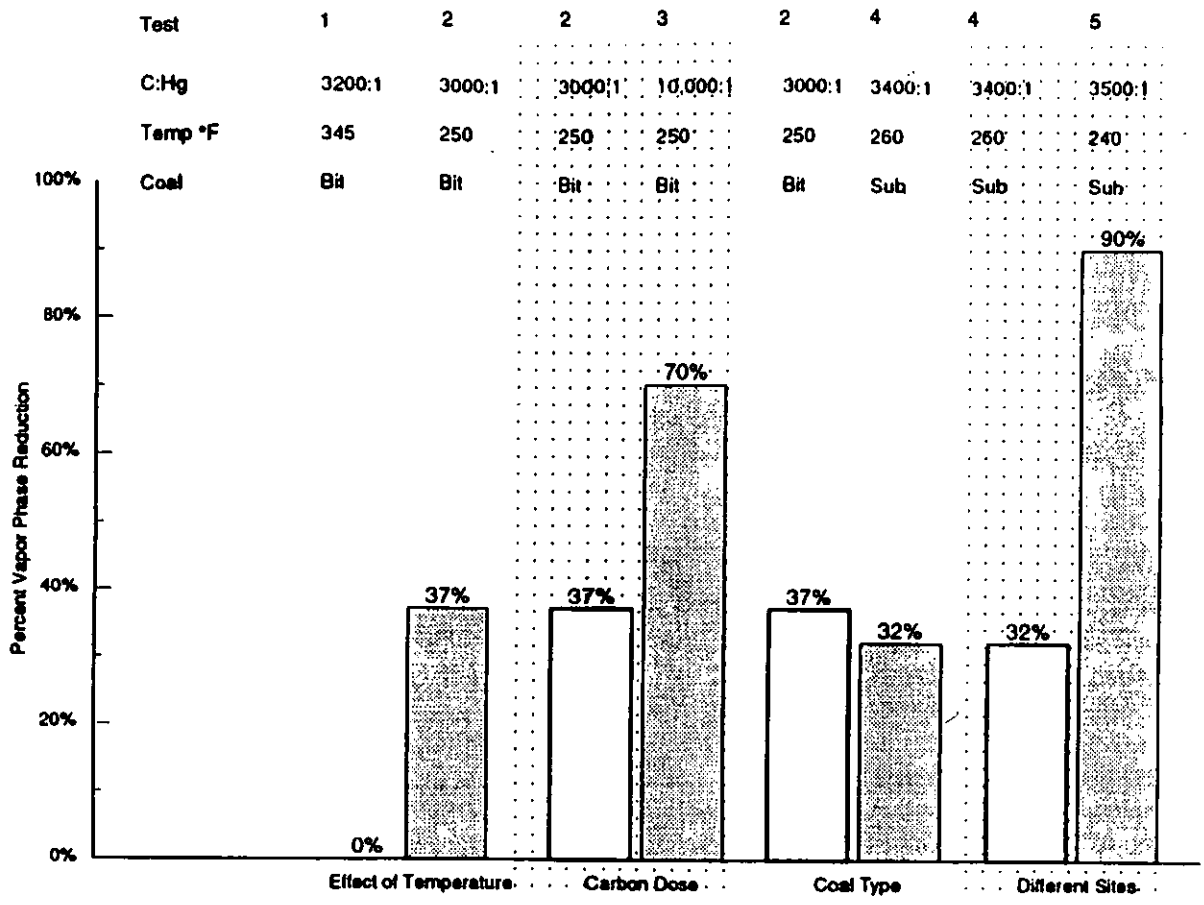

Figure 7-1
Test results for vapor-phase mercury control by activated carbon injection (Chang et al. 1993; Miller et al. 1994)

these limited results. Tests 2 and 4 show some differences resulting from coal type, while Tests 4 and 5 show variations in the results obtained from different sites. Test 4, conducted at EERC, had much lower mercury removal efficiencies compared to Test 5, conducted at PSCCo. The data were insufficient to determine whether this was a result of differences in the two types of low-sulfur coal used or other factors such as mercury speciation.

Judging from these preliminary test results at PSCCo and EERC, activated carbon injection appears to be able to remove mercury from flue gas, but its performance can vary considerably. Therefore, a better understanding of the different factors affecting mercury removal is necessary to determine if the method can be effective under the wide variety of conditions encountered in utility fossil-fuel-fired plants. The impact of injected carbon on existing equipment, such as the downstream ESP and baghouse, must be assessed carefully. Also, use of other sorbents (such as diatomaceous earth, zeolites, and various high-surface-area and chemically active materials) needs evaluation. Finally, the issue of waste disposal must be addressed to ensure that the mercury collected will not volatilize in a landfill or present solid or liquid waste problems.

Preliminary Cost Estimate. The research to date shows high variability in the mercury removal effectiveness under different conditions and at different sites. It is, therefore, difficult to estimate the cost effectiveness of activated carbon injection in controlling mercury since it is uncertain whether specific mercury removal levels are achievable at different conditions. It is possible, however, to prepare a preliminary cost based on the limited data available assuming different mercury removal efficiencies. The equipment needed for carbon injection does not vary much and consists of three major components: spray cooling, carbon injection, and a baghouse. Spray cooling is needed if the flue gas temperature is too high for effective mercury removal. It is assumed that an air atomized water spray system is used and that enough water is used to cool the flue gas by 100°F.

For carbon injection, it is assumed that a dry pneumatic conveying system is built to inject the dry powder from a storage silo directly into the gas stream. This system will include control and instrumentation to adjust the amount of carbon injected as a function of boiler load. A baghouse is added if the carbon is injected downstream of an existing particulate collector such as an ESP or a baghouse. If

the carbon is injected upstream of the primary particulate collector, it is assumed that another device will not be needed.

The cost analysis is based on vendor quotes, EPRI economic studies, and other contractor reports. Figure 7-2 provides a summary of the key results. The base case (Condition 1) assumes a 250-MWe pulverized-coal boiler with a flue gas temperature of 250°F and a mercury flue gas concentration of 10 $\mu g/m^3$. This equates to a mass rate of 145 pounds per year at the stack. Carbon is injected directly into the flue gas before the primary particulate collector and no cooling or additional baghouse is needed. A total mercury removal of 30 percent is assumed; results to date show vapor-phase mercury removal levels of 30 percent are possible. It is assumed that the carbon collected with flyash can be disposed as a nonhazardous waste. The levelized (capital and operating) cost per pound of mercury removed is estimated to be $14,400, including flyash/carbon disposal cost.

Condition 2 assumes that cooling is needed to reduce the temperature to 250°F. The analysis assumes a 100°F cooling is needed from a flue gas temperature of 350°F. The cost increases by about 57 percent to $22,700 per pound of mercury. In Condition 3, a baghouse is added after the primary particulate collector and carbon is injected before the baghouse with spray cooling. Mercury removal is assumed to improve to 50 percent. The waste carbon collected in the baghouse with any remaining flyash is assumed to be a hazardous waste with a disposal cost of $200/ton. The overall cost for mercury control is $38,200 per pound.

In Condition 4, carbon injection increases to 10,000:1 C:Hg and mercury removal increases to 70 percent. Both cooling and baghouse costs are included. The cost is estimated to be $36,800 per pound of mercury.

These conditions provide a range for mercury control cost effectiveness with the understanding that the cost estimates do not include potential items that can impact and increase the overall costs significantly. For example, the impact of large amounts of activated carbon injection (at >10,000:1 C:Hg) on the performance of an ESP is unknown. Spray cooling into the ductwork after the air heater and before the particulate collector can lead to corrosion and fly ash deposition due to the short residence time and the difficulty in atomizing the

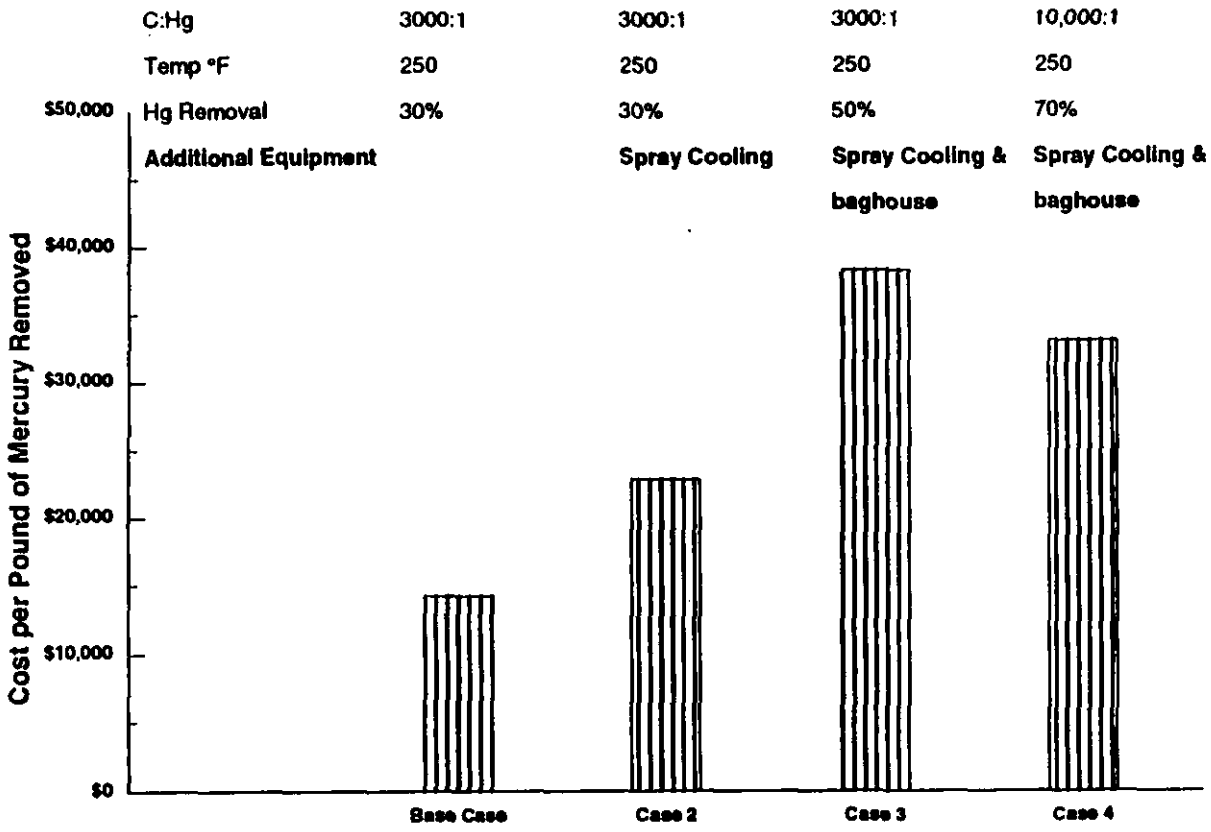

Figure 7-2
Cost-effectiveness of activated carbon injection

water droplets. There is usually very little duct length between the air heater and the ESP. The impact on ESP and baghouse performance is also not totally predictable. In some cases, moisture may create dustcakes that are difficult to remove. In others, the resistivity of the ESP may become too low and result in significant re-entrainment. Large quantities of water (300 gpm to cool flue gas from a 500-MWe plant by 100°F) are also needed. It is assumed that suitable wastewater from the plant is available, and no cost has been assigned to water usage.

For the carbon collected with fly ash, it is assumed that the total waste can still be classified as nonhazardous (the cost of disposal of carbon collected without fly ash downstream of the primary collector is treated as that of a hazardous waste at $200/ton). Otherwise, it can add significantly to the disposal cost. Some of the equipment, such as a downstream baghouse, may be difficult to retrofit because of space constraints that in some cases can double the installation cost requirements. The costs are also based on a 250-MWe boiler. Larger boilers will probably have a lower cost per pound of mercury removed due to economies of scale. Cost sensitivities to unit size and other factors will require further assessment.

Wet Scrubbing

Most of the recent measurements of mercury removal by wet FGD systems have been obtained in studies sponsored by EPRI. Two different approaches were used to investigate mercury removal. At EPRI's Environmental Control Technology Center (ECTC, formerly the High Sulfur Test Center), mercury removal across a pilot unit was evaluated under carefully controlled conditions. The test objectives were to identify chemical and physical variables that significantly affect mercury removal efficiency.

Mercury (and other air toxic) emissions have also been measured at full-scale electric utility sites as part of EPRI's FCEM and the USDOE Air Toxics programs. These programs have obtained full-scale data for mercury removal across conventional particulate control devices and FGD processes applied to a variety of boilers burning different coals.

Table 7-1 summarizes the mercury reduction observed from the fuel to the stack gas at the various full-scale sites that were equipped with wet FGD systems (EPRI 1994, Chapter 3). The data show:

- Nearly all mercury in coal was found in the flue gas exiting the boiler. Very small amounts of mercury have been found in bottom ash.
- The ESPs tested did not efficiently remove vapor-phase mercury.
- Mercury removal across wet limestone FGD systems ranged from 0 to 90 percent.
- Mercury speciation data, where available and assumed accurate, showed that the removal of oxidized mercury by FGD systems with one exception was generally high.
- Mercury removed by the FGD system was found in the scrubber sludge.

Because the mercury removal efficiency for wet FGD systems is not well-understood, EPRI has conducted pilot-scale studies at the ECTC (Peterson et al. 1994). As part of this test series, a method was devised to add elemental mercury to the flue gas upstream of the pilot unit. The objective of these changes was to explore how the proportion of elemental to oxidized mercury in the flue gas affected mercury removal by wet FGD systems.

Method 29 mercury measurement results showed that the oxidized mercury accounted for 95 percent of the total mercury in the unspiked flue gas. The results showed that 99 percent of the oxidized mercury was removed by the FGD system. In tests without elemental mercury spiking, the change in scrubber liquid-to-gas ratio (L/G) from 133 gallons/thousand actual cubic feet of gas to 45 gallons/thousand actual cubic feet reduced the oxidized mercury removal efficiency from 99 to 90 percent, suggesting that the removal of oxidized mercury is mass-transfer limited. Oxidized mercury removal also appeared to decrease slightly when the FGD system operation was changed from inhibited oxidation to forced oxidation (from 99 to 92 percent).

When elemental mercury was spiked into the flue gas, the concentrations of both oxidized and elemental mercury increased. These results suggest that either some of the spiked elemental mercury was converted to an oxidized form after it

Table 7-1
Mercury Removal Data for Full-Scale Sites With Wet FGD Systems

Site Name	Coal Sulfur (%)	Coal Type	Coal Hg (ppm)	Control Systems			Average Site Reduction (%)
				ESP	Fabric Filter	FGD Reagent	
DOE Site 4	2.5	Bituminous	0.08	Y		Limestone	44
DOE Site 6	1.1	Lignite	0.08	Y		Lime	-18
EPRI FCEM Site 101R	0.8	Sub-bituminous	0.06		Y	Lime	69
EPRI FCEM Site 11R	0.6	Sub-bituminous	0.11	Y		Limestone	10
EPRI FCEM Site 12R	2.6	Bituminous	0.10	Y		Limestone	84
EPRI FCEM Site 20	2.2	Lignite	0.26	Y		Limestone	54

mixed with the flue gas or that there was a bias in the mercury speciation as determined by Method 29.

During the mercury spiking tests, the oxidized mercury removal efficiency decreased significantly under otherwise constant operating conditions (from 99 to 89 percent under inhibited-oxidation conditions and from 92 to 80 percent under forced-oxidation conditions). This unexpected effect may indicate that the form of oxidized mercury during the spiking tests was different than the form of oxidized mercury normally present in the ECTC flue gas. If this hypothesis is true, it may explain why some full-scale FGD systems exhibit poor oxidized mercury removal efficiency. No elemental mercury removal was observed across the scrubber during the spiking tests.

Mercury removal across a wet scrubber can vary from 0 percent to greater than 90 percent depending on mercury species present and operating conditions. The mercury removal effectiveness under different flue gas and scrubber operating conditions is not totally understood. And, as in the activated carbon injection tests, accurately determining the mercury species present has been difficult, especially when mercury speciation varies along the flue gas pathway. A better understanding of the mercury species present and their removal across the wet scrubber will enable the design of more effective scrubbers. It will also provide information on which mercury species could be better removed across the scrubber and direct the development of methods that can influence the formation of specific mercury species upstream.

Preliminary Cost Estimate. For boilers with existing wet scrubbers, there is no incremental cost for the amount of mercury already controlled by the scrubber, assuming that any mercury removed in the scrubber does not require additional treatment or special disposal. The research to date, however, shows that the amount of mercury controlled by a wet SO_2 scrubber can vary significantly. Based on these limited data, it is assumed that a wet scrubber is built for mercury control and SO_2 removal is not required. A credit of $160 per ton of SO_2 is taken for the amount of sulfur dioxide removed.

Costs for FGD systems to control mercury are calculated for two different coal types, a low sulfur Powder River Basin coal and a high sulfur Illinois #6 coal.

The FGD systems in both cases are designed for 90 percent SO_2 removal. On the assumption that the oxidized mercury is of the form observed in pilot tests to be easily absorbed by the scrubber, this design would result in roughly 90 percent removal of the oxidized mercury.

For the low-sulfur coal (0.48 percent S) case, it is assumed that 75 percent of the inlet mercury is in the oxidized form and 90 percent removal of the oxidized mercury results in 68 percent total mercury removal at a cost of $116,000 per pound of mercury removed. In a more likely situation, if only 50 percent of the inlet mercury is in the oxidized form, 90 percent removal of the oxidized mercury (45 percent total mercury removal) results in a cost of $174,000 per pound of mercury removed.

In the high sulfur coal (4.0 percent) case, the costs for mercury control are $76,000 and $114,000 per pound of mercury for 75 percent and 50 percent of the inlet mercury in oxidized form, respectively. The cost of controlling mercury by wet scrubbing can increase dramatically if the SO_2 credits are lower or if the cost of scrubber sludge is treated the same as that of a hazardous waste.

Other Mercury Control Approaches

Three other potential mercury control approaches are mentioned briefly here but very few data exist for effectiveness of mercury removal from utility flue gas. A limited amount of data is available for mercury removal across spray dryer FGD systems treating coal-fired flue gas streams. Sources include pilot-scale data from EPRI's ECTC, full- and pilot-scale data published by Joy-Niro (Felsvang et al. 1993), and full-scale data from a USDOE study.

The pilot-scale spray dryer testing conducted at the ECTC showed that the system removed approximately 75 percent of the mercury during base-line operation (Davidson and Blythe 1993). When activated carbon was injected into the flue gas upstream of the spray dryer vessel, the removal efficiency increased to 95 percent. These results agree well with data reported by Niro. The Niro data also showed that the mercury removal across spray dryer systems depends on the amount of oxidized mercury in the flue gas. Niro was able to increase the relative amount of oxidized mercury in the flue gas by feeding sodium chloride

into the boiler along with the coal or by injecting HCl into the flue gas at boiler exit temperatures (1,000°F). However, the presence of chlorides may lead to increased fouling and corrosion in the boilers.

The USDOE full-scale data showed that essentially no mercury was removed across the spray dryer system. Sampling and analytical difficulties make it difficult to interpret the results from this study.

Activated carbon beds are used in Germany and Japan at waste incinerators as well as a few power plants as final-stage polishing units for the removal of SO_2, NO_x, volatile organics, and trace metals downstream of primary flue gas desulfurization units and particulate collectors. Due to the large amounts of activated carbon used in the beds, mercury removal has been very effective for these applications. However, there has been no work to date to develop carbon beds specifically for removal of mercury from utility flue gas.

Direct adaptation of the existing carbon bed technology to utility flue gas mercury removal is very costly because of the large flue gas volumes and the low mercury concentrations involved. One source estimated the cost to be $130,000 per pound of mercury removed (Radian 1993). Modifications to reduce the amount of carbon in the bed and the size of the beds are possible but will require a thorough engineering and economic analysis to determine feasibility. Furthermore, the effectiveness of the "modified" beds for mercury removal under different flue gas conditions will need testing.

Sodium sulfide injection has been shown to remove mercury from incinerator flue gas streams, presumably by reactions to form mercuric sulfide, which is then removed from the flue gas by the particulate control device (Guest and Knizek 1991). Because of limited testing and problems with sampling and analysis of mercury, there is some question about the results. This technology has not been tested on utility flue gas at full scale. Potential problems exist with storage and handling of the chemical, corrosion, and hydrogen sulfide formation.

Summary: Mercury Control

Development of suitable mercury control approaches has been complicated by the lack of reliable methods to measure and speciate the low mercury concentrations present in power plant flue gas. Current mercury control approaches have been applied to waste incineration plants where flue gas mercury concentrations are several orders of magnitude above those from steam electric plants, and thus may not be applicable to power plant flue gas. Tests of direct carbon injection into, and wet scrubbing of, power plant flue gas show the mercury removal efficiencies to be highly variable and dependent on flue gas conditions, coal type, fly ash, and gas composition, mercury speciation, residence time and reaction kinetics, and sorbent and scrubber properties. While high mercury removal efficiencies were observed in some tests, low to moderate removals were measured at other typical power plant conditions. Therefore, it is not yet possible to predict with confidence the level of flue gas mercury control achievable for power plant flue gas under all conditions. In addition, other issues, such as the impact of mercury control technologies on existing equipment and the proper disposal of the collected waste, will need to be addressed. Preliminary cost estimates to control 50 percent of the mercury emitted from utility power plants (assumed 40 metric tons/year, Table 2-5) in the United States ranged from $1 to $10 billion per year.

Section 8
CONCLUSIONS

Background

1. Mercury arises from both natural and human processes, and cycles through atmospheric, aquatic, and terrestrial environments. Forms of mercury that appear most important in these environments are elemental mercury, inorganic mercury, and methylmercury [Hg(0), Hg(II), and CH_3Hg^+, respectively]. The chemical form affects transport through air, land, and water, as well as chemical and biological behavior. Because of mercury's high vapor pressure, it has a global cycle, and this property increases the complexity of understanding its fate and effects in the environment.

2. The most important form for risk assessment is methylmercury, which is a neurotoxin, accumulates in fish, and is produced principally by microbiological processes. Fish typically have more than 95 percent of their total mercury as methylmercury, and are the major route of exposure for most fish-eating organisms.

3. Development of ultraclean sampling protocols and new analytical techniques have led to a broader understanding of mercury cycling, exposures, and effects than could be obtained previously. Even high concentration samples, like fossil fuels, sediments, and biota, require clean techniques and sensitive analytical methods to obtain meaningful results.

Mercury Sources and Emissions

4. Mercury is emitted around the globe, chiefly in the northern hemisphere, and sources include the aquatic and terrestrial environments as well as both historical and present-day natural and on-going human activities. Global perspectives of atmospheric cycling apply to mercury as do regional and local perspectives.

5. The global basis of mercury inputs to the atmosphere constrain the atmospheric cycle to 5000–6000 metric tons per year with an overall average

residence time of 1 year. An estimate of oceans as a source of mercury suggest that 2000 metric tons are emitted per year into the atmosphere. Anthropogenic activities have been estimated to produce between 2300 and 4500 metric tons per year. Uncertainty in the high-end estimates suggests a range of 2–4. Land sources, obtained by difference, amount to about 1000 to 2000 metric tons per year. Most uncertainty is associated with land mercury sources.

6. Mercury atmospheric inputs from fossil fuel combustion for electricity generation have been estimated as about 13 percent of global anthropogenic sources.

7. In 1990, emissions from U. S. anthropogenic sources amounted to about 250 metric tons per year, of which fossil fuel combustion for electricity generation comprised 40 metric tons per year or no more than 16 percent of total anthropogenic sources. U. S. electricity generation is about 1 percent of the global total anthropogenic emissions and about 0.8 percent of the annual atmospheric mercury cycle. Other global sources, such as the former East Germany, emitted about 330 metric tons per year in 1990, more than the total from all U. S. sources.

8. Older literature estimates of mercury emissions are suspect. Recent measurements of fossil fuel combustion sources have lower mean values and less variance, although the current range from high to low is about, or less than, a factor of 10 for coal, oil, and gas combustion. Coal combustion mercury emission estimates vary between 0.5 to 10 μg/megajoule (MJ), while oil and gas average 0.2 and 0.00034 μg/MJ, respectively. Coal emissions have the most variance, because coal type and emission controls vary broadly. The most common power plant coal combustion uses bituminous coal with electrostatic precipitators, averaging 2.8 μg/MJ, based on measurements at 15 plants.

9. Fuel measurements provide an independent way to constrain combustion emission estimates. A major data set obtained by measuring mercury in samples from coal seams provided a mean of 0.21 ppm. Using recent techniques on samples collected from coal-as-burned gave a mean of 0.09

ppm. This compares reasonable well with results for coals from non-U. S. sources, with a mean of 0.12 ppm. One reason for the difference between seam coal samples and coal-as-burned is the effect of washing, which can remove 50 percent of the mercury from high-mercury coals. Analysis of oil and gas samples required extremely sensitive techniques, with concentrations of 0.002 to 0.008 ppm in oil and 0.02 $\mu g/m^3$ in gas.

10. Measurements of emissions at a single coal-fired plant by five different investigators and collection methods provided results with excellent agreement. For total mercury, the emission factors for power plants appear to be accurate. The low concentrations in flue gases from modern power plants show significant variance among power plant types, fuels, and operation.

11. Contrary to previous literature stating that almost all input mercury escapes from the plant, the removal from coal-fired plants with dry particulate controls averages 30 percent. Further emission controls (flue-gas desulfurization) can reduce emissions by an additional 45 percent. Both processes are extremely variable, ranging from 0 to 90 percent removal for both processes in series.

12. Mercury emissions from combustion are generally classified as elemental [Hg(0)] or inorganic mercury [Hg(II)]. There is great uncertainty in these measurements, and attempts to speciate remain experimental. There does not appear to be any methylmercury in flue gases. Preliminary data (17 measurements from unconfirmed protocols) suggest that inorganic mercury averages less than 77 percent, with a most probable mean of 55 percent. The latter measurement is observed to relate to chlorine in coal. Higher chlorine is associated with higher inorganic mercury. The range appears to be from 0 to almost 90 percent inorganic mercury.

13. Based on older data, fossil-fuel-fired power plants have been estimated as emitting as much as 93 metric tons per year from U. S. electrical generation. However, two independent estimates using the most recent emissions and fuel mercury data provided remarkable agreement of 40 metric tons per year. Correcting total emissions for the difference (53 metric tons per year)

allows an estimate of the relative role of electrical generation as a source of mercury, which contributes no more than 16 percent of known U. S. anthropogenic emissions and about 1 percent of global anthropogenic emissions. As newer generation technologies come on-line, there is the promise of reduced emissions of mercury.

Atmospheric Mercury

14. One of the greatest sources of uncertainty in the atmospheric cycling of mercury concerns the transport and transformation of mercury. These processes are important because mercury deposits on local, regional, and global scales, depending on the chemical form of the mercury and local conditions.

15. Mercury in the atmosphere is virtually all elemental mercury, with only 2 percent or less present as inorganic mercury. Essentially all of the inorganic mercury is particulate (as aerosols), with the exception of source plumes where gaseous inorganic mercury might occur. Methylmercury is found in the atmosphere, but its source remains unknown at this time.

16. With the exception of areas affected by regional and local sources, elemental mercury is fairly uniform throughout the northern hemisphere, with Wisconsin, Florida, and the north-central Pacific Ocean all having equivalent concentrations.

17. Aerosol mercury varies by a factor of 10 to 30, with lowest concentrations in the north-central Pacific, and highest concentrations measured in the Nordic countries which are subject to large regional sources.

18. For the sites with data, average rainfall mercury concentration varies by less than a factor of 2, and wet deposition varies by a factor of 5. Dry deposition is not well-characterized, but dryfall, throughfall, and litterfall can contribute from 30 to 200 percent in addition to wet deposition.

19. Global scale contributions to local and regional deposition remain uncertain. Global calculations provide insight into processes, but a Global Mercury

Cycling Model (GMCM) under development may constrain the spatial deposition of mercury.

20. Emissions to the atmosphere from historical uses of mercury, particularly in precious metal extraction, apparently match emissions from anthropogenic activities during the industrial era. In Sweden, industrial uses of mercury peaked during the 1960s. Historical mercury use has not been well-characterized in other countries.

21. Based on peat and lake sediment cores, estimates of historical levels of deposition vary considerably. Present day mercury deposition is 2 to 5 times greater than pre-industrial deposition. However, in some areas the peak deposition occurred around 1960, with a two- to three-fold reduction to the levels found in the 1980s. In other areas, no such peak has been observed. Based on oceanographic surveys, increased mercury concentrations in air at mid-latitudes of the Atlantic Ocean contrast with relatively constant mid-Pacific Ocean concentrations, and suggest that historical mercury varies spatially as well as temporally. Better understanding of the multiple sources of historical mercury emissions will reduce uncertainty about the role of re-emissions from the ocean and land surfaces.

22. Analyses of mercury in biological samples collected over time are expected to reflect deposition of mercury. With one exception, fish mercury concentrations show no temporal trend. One assessment supported an hypothesis that fish mercury concentrations had increased over recent decades. These results occurred in the region where core samples indicated that deposition had decreased over the same time period. In the United Kingdom, bird livers showed a pattern of decrease since the late 1960s, supporting the idea that recent decades have seen decreasing mercury deposition.

23. Atmospheric mercury chemistry remains an area of great importance for assessing the fate of emissions, but few data are available to check laboratory studies and model simulations of mercury behavior in air. The most important reactions involve the transformations of Hg(0) and Hg(II) in

cloud water droplets, especially the oxidation of Hg(0) by ozone in the presence of sunlight, and the reduction of Hg(II) by aqueous sulfur dioxide. The mass transfer of weakly soluble Hg(0) (at equilibrium concentrations of picograms/l) is the probable rate-limiting step in the oxidation process.

24. Modeling of mercury atmospheric behavior depends on assumptions of mercury speciation that are not substantiated by field data. Currently, simulations assume that Hg(0) has an atmospheric half-life on the order of weeks to months, while gaseous Hg(II) deposits rapidly (half-life of hours). However, gaseous Hg(II) apparently attaches to fine particulates rapidly, and may not have time to deposit. Such fine particulate Hg(II) may have a long half-life, depending on whether or not rainfall scavenging removes the particles.

25. Despite the uncertainties about mercury transformations and transport in the atmosphere, investigators will use models to provide initial characterization of mercury distribution and deposition. Assuming all emissions are gaseous Hg(II), the maximum deposition within a 50-km radius of tall stacks would range from 0.4 to 27 percent due to uncertainty in deposition velocities. Assuming that all of the oxidized mercury is in particulate form provides a maximum deposition of between 4 and 7 percent. All Hg(0) would approach 5 percent as a maximum within 50 km. Recent plume modeling studies with a mixture of mercury species for a hypothetical power plant suggest the deposition lies between 1 and 4 percent of total emissions. These results suggest that mercury from power plants has a regional or global, rather than local, pattern of deposition.

Mercury in Aquatic Ecosystems

26. Research on mercury cycling and fish accumulation in northern Wisconsin seepage lakes was designed to look at ecosystems that would be dominated by atmospheric deposition, and fish mercury accumulation would approach a maximum because of the low productivity of the selected lakes, the low pH, and the range of DOC in the waters. The latter factors appear to enhance mercury accumulation in fish.

27. The mass balance of a single seepage lake in northern Wisconsin showed:

    - The amount of mercury deposited from the atmosphere was greater than the amount of mercury in the fishery.
    - Evasion and sedimentation were major sinks for mercury in lakes, but the transport to these sinks varied depending on lake water quality and mercury loading.
    - A large fraction of the methylmercury was formed within the lake ecosystem.
    - Net methylation was the key process affecting mercury in fish, where 95 percent of the mercury is methylmercury.

28. The amount of methylmercury accumulated by fish appears to be controlled by important factors other than just deposition. This result supports the observation that fish mercury levels did not respond to decreased deposition over the last few decades.

29. In one northern Wisconsin seepage lake, about 5 percent of the total deposited mercury was accumulated as methylmercury by fish.

30. For seven northern Wisconsin seepage lakes, fish mercury correlated with water mercury concentrations. However, the concept of bioaccumulation factors appears flawed when applied to a broad spectrum of lake types, adding uncertainty to risk assessment performed with such factors. For the seven lakes considered together, the average bioaccumulation factor was 100,000 (ratio of total mercury in fish to total mercury in water).

31. Field results supported the contention that sulfate-reducing bacteria are involved in methylation of mercury. The methylation/demethylation process rates in lakes depended on a variety of factors including DOC, sulfate, temperature, and nutrient levels. Wetlands may enhance the net production of methylmercury.

32. The results of the field studies and mechanistic concepts were used to develop the Mercury Cycling Model (MCM, v. 1.0), a user-friendly model

for desktop computers. The MCM simulation model can assess hypotheses about mercury behavior. Also, it provides an excellent tool for mercury risk assessment. The MCM can simulate mercury dynamics in the lake ecosystem and calculate concentrations in fish. Site-specific bioaccumulation factors can also be determined. The model has been applied to seepage and drainage lakes, as well as to Lake Superior and Lake Ontario. Also, the model has been incorporated into USEPA's WASP4 modeling framework as MERC4.

33. Simulation results of the MCM show that bioaccumulation factors vary, predatory fish take 5–10 years to respond to site changes at Little Rock Lake in Wisconsin, and small changes in atmospheric loading have negligible effects on fish mercury concentrations.

34. Most data suggest that ecological effects of mercury in aquatic organisms are not detectable at current levels. However, some data suggests that fish-eating birds and mammals may be at risk, and evaluation of these risk relationships is ongoing.

Health and Mercury

35. The primary environmental health concerns for mercury are those associated with methylmercury exposures to pregnant women and young children. The concerns are based on observations of methylmercury levels in the environment near those that have been linked with biological responses. The main route of exposure to methylmercury is through the food chain. Exposure to other forms of mercury is not significant in the ambient environment.

36. A physiologically based pharmacokinetic (PBPK) model has been developed, validated, and used to estimate fetal dose of methylmercury given measurements of the chemical in pregnant mothers' hair. The model provides a critical link between maternal intake and fetal dose.

37. The existing RfD for methylmercury is based on information from an acute poisoning episode in Iraq. This data set may not be suitable for assessing

possible health risks from utility emissions because: (1) it represents short-term exposure to high doses of methylmercury rather than longer-term exposure to low doses, (2) its health endpoints are based on subjective recall rather than on objective measurements, and (3) alternative statistical methods for analyzing the data set may be more appropriate than the method used to set the current RfD.

38. Analyses of more appropriate data sets will be available shortly. These include EPRI's reanalysis of the New Zealand data set, and long-term developmental assessments of children in the Seychelles Islands and the Faroe Islands.

Risk Assessment of Mercury From Power Plant Emissions

39. Assessment of emissions from each of 594 fossil-fuel-fired power plants showed no inhalation risk for the maximally exposed individual (MEI). The highest relative risk for a single plant was 0.5 (ratio of exposure to RfC).

40. Multimedia health risk assessments were obtained using EPRI's Total Risk of Utility Emissions (TRUE) model. The four plants studied ranked between the 38th and 98th percentiles when their mercury emissions were compared to those of all power plants. By modeling transport, deposition, and concentrations of methylmercury in fish flesh (using the USEPA bioconcentration factor of 33,000)—and comparing those concentrations to the current federal reference dose for mercury in food fish—researchers calculated a hazard index for mercury for each unit. These hazard indexes ranged from 0.00018 to 0.28.

41. A reliable assessment of mercury-related risks requires comprehensive treatment of mercury cycling in surface water bodies. For that purpose, a steady-state version of EPRI's Mercury Cycling Model (MCM) was combined with TRUE. Analysis of four lakes at two different sites showed variations in water mercury concentrations, fractions of methylated mercury, and methylmercury water-to-fish BAFs—and concomitant variations in risk—related to local hydrologic and water quality

characteristics. The estimated multimedia hazard indexes for mercury were significantly less than 1 for both sites and all alternative lakes evaluated.

42. To characterize the uncertainty in health risks related to mercury emissions, a sensitivity analysis based on simulation of one coal-fired power plant was performed using 10,000 iterations on a simplified version of the TRUE model. The analysis gave a probability distribution of the mercury hazard index that was positively skewed with a mean of 0.022 (as a fraction of the federal reference dose). A deterministic assessment of the MEI mercury hazard index for the same plant (0.28) was estimated to fall beyond the 95th percentile of the derived distribution.

Potential Mercury Control Approaches

43. Mercury is difficult to collect because it appears in flue gas mainly in vapor, rather than particulate, form. As vapor, it easily passes through particulate control devices such as baghouses and electrostatic precipitators. Low concentrations of mercury present in utility flue gas make collection even more difficult.

44. Because many wet flue gas desulfurization (FGD) installations are already in place to help coal-fired boilers meet SO_2 emissions regulations, there is significant interest in the capability of these systems to simultaneously remove SO_2 and trace chemicals, including mercury. The removal efficiency of wet scrubbers evidently depends on the species of mercury present in the flue gas. Field measurements of full-scale wet scrubbers show removal efficiencies from 0 to greater than 90 percent.

45. Mercury removal efficiencies with direct carbon injection or wet scrubbing are highly variable and depend on flue gas conditions, coal type, fly ash, gas composition, mercury speciation, residence time and reaction kinetics, and sorbent and scrubber properties. While some tests showed high removal efficiencies, others showed low to moderate removals at different, but typical power plant conditions. Thus, it is impossible to predict the level of mercury control achievable for power plant flue gas under all conditions.

46. Preliminary cost estimates to control mercury were developed for activated carbon and wet scrubbing processes. Activated carbon injection costs about $14,000 to $38,000 per pound ($31,000 to $84,000 per kg) removed. For a 250 MWe plant emitting 145 pounds of mercury per year, it would cost $1 to $3 million per year to remove 50 percent of the mercury present. On the other hand, wet FGD technology costs $76,000 to $174,000 per pound ($167,000 to $383,000 per kg) removed, including SO_2 allowances. For a 250 MWe plant emitting 145 pounds (66 kg) of mercury per year, it would cost $5 to $13 million dollars per year to remove 50 percent of the mercury present. These costs would increase if SO_2 allowances were lowered or scrubber sludge were declared hazardous waste. The total estimated cost to control 50 percent of the mercury emitted from U. S. utility power plants ranges from $1 to $10 billion per year.

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Electric Utility Trace Substances Synthesis Report

Volume 4: Appendix P, Toxicology Profiles

TR-104614-V4

Research Project 3081
November 1994

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APPENDIX P

TOXICOLOGY PROFILES OF KEY SUBSTANCES

This appendix presents a summary of scientific information on the key trace substances that are the subject of this Synthesis Report. The information is presented as supplemental material to the remainder of the report. Information on regulatory and other status of substances is current as of 1992.

TOXICOLOGY PROFILE
ARSENIC, ARSENIC TRIOXIDE AND ARSENIC COMPOUNDS

Synonyms: Black arsenic, grey arsenic, metallic arsenic

CAS NO: 7440-38-2 for metallic form 1327-53-3 for arsenic trioxide. Other forms have different CAS numbers.

Boiling point: Metallic form sublimes at 613°C. Arsenic trioxide sublimes at 193°C and boils at 457°C. Other forms have varying boiling points.

Color: Silver-grey, brittle, crystalline solid for the metallic form. Arsenic trioxide is white or transparent lumps or crystalline powder.

DOT designation: Poison

Flammable limits: Arsenic trioxide is non flammable
- autoignition: N/A
- flash point: Dust is flammable when exposed to heat or flame
- LEL: N/A
- UEL: N/A

Henry's law constant: N/A

Melting point: 817°C at 28 atm. for metallic arsenic
315°C at 1 atm. for arsenic trioxide.

Molecular formula: As (metallic)
AS2°3

Molecular weight: 74.9 for As

Odor:
- Threshold: <1 ppm
- Recognition: <1 ppm
- Characteristics: Arsine gas has a garlic-like odor

pH: No specific pH could be found for arsenic acid (H3AsO~).

Solubility water: Metal is insoluble in water. Arsenic trioxide is soluble at 21 g/liter of water at 25°C. Other forms of arsenic may be soluble to varying degrees.

K_{ow}: N/A

Other: Generally soluble in alcohols but solubility depends upon the form.

K_{oc}: N/A

Specific gravity: 5.727

Vapor density: N/A

Vapor pressure: Only arsenic trioxide and arsine gas are volatile, the other forms of arsenic are considered to be nonvolatile.

Viscosity: N/A

(HSDB, 1990; Hawley, 1987; RTECS, 1990)

COMPOSITION: Arsenic is an abundant element found in the earth's crust. It exists as a nonmetal or metalloid that may be present in the environment as a constituent of organic and inorganic compounds. The complexing of arsenic and organic compounds increases its solubility potential. Naturally - occurring arsenic can exist in four oxidation states: -3, 0 (the metallic state), +3, and +5. Arsenate (As^{5+}) is the dominant species in aquatic systems. Biological activity may reduce arsenate to arsenite (As^{3+}) and finally to methylated arsenicals (As^{3-}) (Callahan et al., 1979). Inorganic arsenic is most commonly present in the trivalent or pentavalent oxide form (CARB, 1990).

The toxicity of arsenic depends upon its chemical form as well as with the route, dose, and duration of exposure. In general, arsenites (As^{3+}) are potentially more toxic than arsenates; soluble arsenic compounds are potentially more toxic than insoluble ones and inorganic arsenic compounds are potentially more toxic than organic derivatives (EPA,1985) .

USES:

Arsenic is used in metallurgy for the hardening of alloys of copper and lead (Merck, 1983). Arsenic is used in insecticides, ant killers, weed killers, wallpaper, paint, ceramic and certain types of glass (Arena, 1986). Arsenic is also produced as a by-product of copper smelting operations (Proctor and Hughes, 1988).

ACUTE TOXICITY: Depending upon dose, arsenic is a potential irritant of the skin, mucous membranes, and the gastrointestinal tract. Acute toxicity from the ingestion of higher dose of arsenic may result in burning of the mouth and throat, gastric pain, vomiting, diarrhea with hemorrhage, hematuria, convulsions, increased capillary permeability, a severe drop in blood pressure, and cardiovascular effects (Vallee et al., 1960) .

The acute toxicity of arsenic varies with species and compound tested. Some of the reported acutely lethal values appear in Table 1.

Table 1.
Acute Lethal Dose Value

Species	*Value*	*Dose*	*Time*	*comments*
Rat	LD5o	763 mg/kg		oral
Mouse	LD50	145 mg/kg		oral
Rat	LD5o	605 mg/kg		oral
Mouse	LD50	120 mg/kg		oral
Rabbit	LD50	75 mg/kg		oral
Rabbit	LD10	300 mg/kg		oral
Guinea Pig	LD10	300 mg/kg		oral
Man	TD10	7857 mg/kg	55 years	oral
Man	TD10	76 mg/kg	12 years	oral

Ingestion:
The lethal dose for humans is reported to be 70 to 180 mg or 1.0 to 2.6 mg/kg-b.w. for a 70 kg adult (Vallee et al., 1960). Patients may describe a metallic taste in the mouth. A garlicky odor of the breath and stool may be noted (Deichmann, 1969). Ingestion of arsenic may result in burning of the mouth and throat, gastric pain, vomiting, diarrhea with hemorrhage, hematuria, increased capillary permeability, and hypotension (Vallee et al., 1960). Convulsions and coma may result from circulatory failure. If death is not immediate, jaundice, oliguria, or anuria may develop in 2 to 3 days (Arena, 1986).

No reports regarding the acute toxicity of arsenic to experimental animals could be located in the literature reviewed.

Inhalation:
Exposure to high concentrations of airborne arsenic trioxide results in severe irritation of the upper and lower respiratory tracts (EPA, 1984). Shortness of breath, cough with foamy sputum, cyanosis, and pulmonary edema may develop (Arena, 1986). Peripheral nervous system effects (numbness, paresthesias, weakness) have

followed single, high dose exposure to arsenic. Recovery of the peripheral nervous system is slow and may be incomplete. Reversible effects on the cardiovascular and hematopoietic systems may also follow acute exposure to arsenic (EPA, 1984).

Skin contact:
See chronic skin contact.

Eye contact:
Exposure to arsenic adsorbed to particulate matter (aerosols) may result in conjunctivitis (Proctor and Hughes, 1988) .

CHRONIC TOXICITY:
Chronic arsenic toxicity is characterized by weakness, anorexia, gastrointestinal disturbances, peripheral neuropathy and skin disorders but these effects are not commonly observed in occupational settings. Liver dysfunction has been seen in experimental animals (Proctor and Hughes, 1988). Moonshine is a possible source of chronic excess arsenic exposure in humans (Arena, 1986).

Ingestion:
Tay and Seah (1975) reported effects in patients in Singapore who had ingested arsenic-containing anti-asthmatic herbal preparations at doses of 2.5 mg As/day as arsenic oxide (As^{3+}) or 10.3 mg As/day as arsenic sulfides over a period of less than 67 months to 15 years. The major effects included: hyperpigmentation (melanosis), multiple arsenical keratoses, sensorimotor polyneuropathy, persistent chronic headache, lethargy, gastroenteritis, and mild iron deficiency anemia.

Chronic oral exposure of humans to inorganic arsenic compounds has been reported to cause skin lesions, peripheral vascular disease, and peripheral neuropathy at doses of 8.8 mg/day for 28 months (Silver and Wainman, 1952). The incidence of Blackfoot disease, a peripheral circulatory disease characterized by gangrene of the extremities, has reportedly been related to the presence of arsenic in the drinking water of residents of the southwest coast of Taiwan (Tseng, 1977). Tseng noted a dose response relationship between the prevalence of Blackfoot disease and arsenic exposure over a concentration range of 0.001-1.82 mg/l. Based upon this, a NOAEL (No observed adverse effect level) of 0.001 to 0.017 mg/l was reported by Tseng for Blackfoot disease.

Schroeder and Balassa (1967) studied the chronic oral toxicity of arsenic on growth and survival in mice. Ingestion of water containing As^{3+} at 5 mg/l over two years is reported to have resulted in decreased survival and reduced median life span in male and female mice.

Inhalation:
The symptoms of chronic inhalation exposure to arsenic compounds are similar to these associated with chronic oral toxicity. Inhaled arsenic compounds have been reported to be associated with skin lesions, cardiovascular and respiratory effects, and peripheral neuropathy (Stokinger, 1981; IARC, 1980; ACGIH, 1986). Workers exposed to arsenic trioxide were reported to develop ulceration and perforation of the nasal septum as well as conjunctivitis and pharyngitis (ACGIH, 1986). A direct relationship between the length and intensity of exposure to inhaled arsenic trioxide by smelter workers and peripheral nerve function damage was reported by Landau (1977) .

No information regarding chronic inhalation exposure of experimental animals to arsenic could be located in the available literature.

Skin contact
Skin contact with arsenic-containing dusts can result in irritation, arsenic dermatosis and skin carcinoma. Regardless of the route of administration, there may also be hyperpigmentation of the skin, eczema, scaling, desquamation and hyperkeratosis of palmar and plantar surfaces (Proctor and Hughes, 1988). Other skin manifestations of arsenic intoxication include brittle and deformed nails with transverse white lines (Aldrich-Mees' lines), loss of hair and nails, and localized edema of the eye lids (Arena, 1986). Gangrene of the fingers and toes secondary to impairment of the peripheral circulation (Blackfoot disease) has been reported (Tseng, 1977).

Arsenic exposure at certain doses may produce a pattern of skin disorders, hyperpigmentation, and keratosis that may develop into basal or squamous cell carcinoma (EPA, 1985). Multiple skin cancers were reported in humans who had used Fowler's solution, a medicinal trivalent arsenical (Cuzick et al., 1982).

SENSITIZATION:
Dermatitis attributable to sensitization has been reported (NIOSH, 1975).

TARGET ORGAN EFFECTS:
The principle tissues or organs affected by arsenic exposure are the skin, respiratory tract, gastrointestinal system, peripheral vascular system, and peripheral nervous system.

ABSORPTION-METABOLISM-EXCRETION:
Absorption from the gastrointestinal tract is dependent upon the solubility of the specific

arsenic compound and the dose. Solutions of As3+ or As5+ soluble inorganic compounds are reported to be most completely absorbed by rats (Coulson et al., 1935). Bettley and O'Shea (1975) have reported that greater than 95% of the inorganic arsenic that humans consume may be absorbed. Insoluble forms of arsenic tend to pass through the gastrointestinal tract (Mappes, 1977). In humans, the rate of arsenic excretion, which is dependent upon dose and oxidation state, is reported to be 50% to 90% within two to four days (Crecelius, 1977). Part of the unexcreted portion of ingested arsenic is deposited in skin, bone, hair and nails. It can be detected in the hair for weeks to months after exposure (Deichman and Gerarde, 1969).

Absorption from the respiratory tract depends upon the specific arsenic compound and the size of aerosols or dusts. Particles less than 1 to 2 μm may be absorbed through the respiratory epithelium; whereas, large particles are most likely deposited in the upper respiratory tract and ultimately swallowed. The amount of arsenic absorbed through inhalation has yet to be determined (EPA, 1984).

Toxicity data from rats cannot be extrapolated to man as the rat is able to store arsenic via binding to hemoglobin in red blood cells (Lanz et al., 1950). This binding results in extremely slow excretion of arsenic in rats as compared to other species (Mealey et al., 1959). For this reason, dogs have been used to obtain experimental toxicity information. Subchronic oral toxicity studies using dogs fed diets containing sodium arsenite or sodium arsenate report that arsenite is potentially more toxic than arsenate. The NOEL (no observed effect level) was reported to be 50 mg/kg-diet for both substances (Byron et al., 1967).

IMMUNOTOXICITY:

Arsenic is one of the metals that has been shown to alter tumor transplantation resistance in the mouse model (Murray and Dean, 1984). Animals treated with 100 ppm sodium arsenate in the feed had an increased survival time and decreased progressive tumor growth (Kerkvliet et al., 1980).

The role of inorganic arsenic as an immunosuppressant in man is mainly inferred from accumulated indirect data (NAS, 1977).

First, the therapeutic action of arsenicals, such as Fowler's solution in the treatment of steroid-responding disorders and as a lymphocytostatic agent, suggests action as an immunosuppressant.

Secondly, the occurrence of herpes simplex and chronic pulmonary infections associated with arsenic exposure, also suggest arsenic is an immunosuppressant (NAS, 1977). Histories of chronic cough and bronchitis in Chilean children exposed to arsenic in drinking water (Borgono et al., 1977) would tend to support such a role.

REPRODUCTIVE TOXICITY:

The teratogenic potential of arsenic has been investigated in animals. Matsumoto et al. (1973) reported decreased fetal weight after oral doses of up to 40 mg arsenate/kg b.w./day administered to pregnant mice for three consecutive days. Diets containing up to 100 mg arsenite/kg-diet, however, were reported to have had no effect on offspring (Kojima, 1974). Female hamsters injected with sodium arsenate on day eight of pregnancy showed a high incidence of fetal resorption and malformations (Holmberg and Ferm, 1969). No data regarding the teratogenicity of inhaled arsenic could be found in the literature.

Studies of occupational and non-occupational groups have found peripheral nervous system changes among those chronically exposed to arsenic. Effects include sensory changes (numbness, paresthesias) as well as motor neuron effects (weakness). Peripheral neuropathies have been associated with arsenic exposure via ingestion (water, medications) and well as via inhalation (employment or residence near smelters) (EPA, 1984).

NEUROTOXICITY:

Acute intoxications with arsenic have produced hyperexcitability, headache and dizziness (Deichman and Gerarde, 1969) ,. Some organic arsenicals, such as arsenilates, have a selective effect on the optic nerve and can cause blindness (HGS, 1964).

GENOTOXICITY:

Nearly all results of gene mutation studies for arsenic (III) and arsenic (V) compounds have been negative. Arsenite and arsenate also have been inactive in gene-specific mutation assays in yeast and cultured mammalian cells. In contrast, arsenic (III), arsenic (V), arsenite and arsenate have been found to result in chromosome aberrations and sister chromatic exchanges in cultured animal and human cells tested *in vitro* (ATSDR, 1987).

There is limited evidence that occupational exposure to arsenic may cause chromosome changes in humans (8eckman et al., 1977; Wen et al., 1981). Beckman et al. (1977) reported an increase in gaps, chromatic aberrations and chromosome aberrations from mine workers at a smelter in northern Sweden.

CARCINOGENICITY:

The majority of tests in which experimental animals were exposed orally to a variety of arsenic compounds produced negative results regarding carcinogenicity (Hueper and Payne, 1962; Byron et al., 1967). A few studies have, however, reported tumorigenic effects of arsenic treatment (Sommers and McManus, 1953; Fierz, 1965; Cuzick, et al., 1982). Mixed results were reported in which arsenic was administered intratracheally to laboratory animals studies (Ishinishi et al., 1976; Ivankovic et al., 1979).

An excess in respiratory cancer mortality has been documented consistently among smelter workers exposed to arsenic. The mortality experience of Anaconda smelter workers has been reported by Lee and Fraumeni (1969) with follow-up by Lee-Feldstein (1983 and 1986). The 8045 workers were categorized qualitatively by the highest exposure area worked in for at least 12 months, as well as by length of employment and year of first employment (1983). Industrial hygiene data were later incorporated into quantitative estimates of worker exposure (1986). An exposure-dependent increase in lung cancer was found in both analyses. The Standard Mortality Ratio (SMR) for respiratory cancer (full cohort) was 285, $p<0.01$. An eight to nine-fold increase in respiratory cancer was seen among those first hired before 1925 and with the highest estimated cumulative exposure (>500 mg/m^3-months).

Welch et al. (1982) followed a sub-cohort of the smelter workers studied by Lee and Frumeni (1969), extending the observation period by 14 years and applying a different exposure classification system. Industrial hygiene measurements gathered by department by 1943-1965 served as the basis of exposure classification. The sub-cohort of 1800 male workers included all those classified as "heavily" exposed by Lee and Fraumeni, as well as 20% random sample of other cohort members. A dose-dependent increase in lung cancer was observed when data were analyzed by ceiling arsenic exposure, time-weighted average exposure, and by cumulative exposure. The respiratory cancer SMR among those exposed to ceiling arsenic levels of 500-4999 µg/m^3 was 348 ($p<0.01$); for those exposed to ceiling arsenic levels >5000 µg/m^3 the respiratory cancer SMR was 662 ($p<0.01$). No significant elevation in respiratory cancer was noted among those exposed to ceiling levels <500 µg/m^3.

A dose-response relationship between the occurrence of skin cancer and arsenic consumption in the drinking water of Taiwanese was reported by Tseng et al. (1968 & 1977).

In 1987, Enterline et al. re-examined the 1982 data using both historical records of airborne arsenic as well as worker records of urinary arsenic levels. A nonlinear increase in lung cancer mortality with increasing dose was found using exposure estimates based on airborne arsenic levels. The relationship between urinary levels and lung absorption of arsenic may be proportionately greater at lower exposure levels (<10,000 µg/m^3/year) than higher levels.

Based upon epidemiological data, IARC and the U.S. EPA have classified arsenic as Human Carcinogen, therefore, neither an AIS (acceptable intake subchronic) nor an AIC (acceptable intake chronic) has been established. A cancer potency factor of 1.75 (mg/kg/day)$^{-1}$ for oral exposure to arsenic was derived, based on a correlation between skin cancer and arsenic ingestion (Tseng et al.,1968). A cancer potency factor of $1.51 \times 10^{+1}$ (mg/kg/day) 1 has been set for inhalation exposure based on epidemiological data from copper smelter workers from 2 different smelters. (Brown and Chu,1983 a,b,c; {re-analysis of Lee and Fraumeni 1969 data} Lee-Feldstein,1983; Welch et al.,1982; Enterline and Marsh, 1982; IRIS, 1990).

EPIDEMIOLOGY:

In a survey of 40,421 Taiwanese exposed to arsenic in well water, Tseng et al. (1977) found a dose-dependent increase in skin cancer, and the incidence of Blackfoot disease. Arsenic levels in well water ranged from 0.01 to 1.82 mg/l (mean near 0.5 mg/l). As estimated by EPA (1987), average daily intake of arsenic in the three exposure groups was 10.8, 29.9, and 50.9 µg/kg/day (males) and 6.8,18.8, and 32.0 µg/kg/day (females). The prevalence rates for skin cancer in the low, mid, and high exposure groups were 2.6, 10.1, and 21.4 per 1000, respectively. Epidemiological studies conducted in certain counties in California, Oregon, and Idaho where relatively high levels of arsenic have been found in drinking water supplies, have failed to correlate the incidence of skin cancer with arsenic in drinking water (Morton et al., 1976; Goldsmith et al., 1972) .

Arsenic is ubiquitous in the environment, with natural as well as manmade sources. Much of the arsenic produced via human activities is released into the atmosphere where it can be transferred to the ground by both wet and dry precipitation. Precipitated or applied arsenic in the soil may be transported to groundwater or surface water and adsorbed by sediments. The form of arsenic present in water depends on the pH, organic content, presence of suspended solids and sediment. Arsenic in the soil is usually bound to clay surfaces. Its mobility depends on

the pH of the soil, phosphate levels, iron and aluminum content and soil type (EPA, 1984). Chemical and biological transformations can make this cycle very complex.

ENVIRONMENTAL FATE:
Epidemiological studies focusing specifically on arsenic exposure and cancer are reviewed under CARCINOGENICITY.

ENVIRONMENTAL TOXICITY:
Arsenic is widely distributed in the environment and all humans are exposed to low levels from the food, air and water. A typical background exposure level is believed to be between 20-70 µg/day with most of the exposure attributable to the diet (ATSDR, 1987).

In air, the average 24 hour ambient arsenic level in the United States ranges from 2.6 ng/m³ to 10.9 ng/m3 but peaks as high as 78 ng/m³ have been recorded. It is mostly the trivalent and pentavalent forms of arsenic that are found in the air (ATSDR, 1987; CARB, 1990).

In soils, the range is between 0.1 and 80 ppm with an average value of 5-6 ppm (EPA, 1984).

The arsenic content of surface waters is, for the most part, below 10 µg/L. Drinking water supplies in the U.S. are generally below 2 µg/L and most drinking water supplies are well below the standard of 50 µg As/L. The chemical form of arsenic in drinking water is typically a mixture of arsenate and arsenite (ATSDR, 1987).

REGULATORY STATUS:

U.S. Environmental Protection Agency
In the 1984 Health Assessment Document for Inorganic Arsenic, the EPA presented a quantitative human health risk assessment. For respiratory cancer, the EPA used data presented in Lee-Feldstein 1983, Welch et al. 1982, Brown and Chu 1983 a,b,c, (a re-analysis of Lee-Feldstein, 1983) and Enterline and Marsh, 1982 (exposure periods lagged 0 and 10 years). Using an absolute-risk linear model, the lifetime lung cancer risk associated with continuous exposure to 1 µg/m³ arsenic was estimated to range from 1.25×10^{-3} to 7.6×10^{-3} with a weighted average estimate of 4.29×10^{-3}.

The EPA (1984) relied on data provided by Tseng et al. (1968) for development of the quantitative risk estimate for skin cancer. Using an absolute-risk linear model, the EPA estimated a lifetime skin cancer risk from drinking water containing 1 µg/L arsenic at 4.3×10^{-4}

The national water quality criteria to protect freshwater organisms is such that the average concentration of dissolved trivalent inorganic arsenic should not exceed 72 mg/l in 30 consecutive days; the maximum concentration should not exceed 140 mg/L; and the concentration may be between 72 and 140 mg/L for up to 96 hours. To protect saltwater organisms, the average concentration should not exceed 63 mg/l in 30 consecutive days; the maximum concentration should not exceed 120 mg/l; and the concentration may be between 63 and 120 mg/l for up to 96 hours.

State of California Department of Health Services
In 1987, the Air Unit Hazard Evaluation Section of the State of California Department of Health Services (DHS) developed a quantitative risk assessment for ambient exposures to arsenic (DHS, 1987). Calculations were based on data presented in Welch et al. (1982), Higgins et al. (1985) and Enterline et al. (1987). Lee-Feldstein (1986) data were not incorporated, as DHS staff considered the assumptions made in the quantitative exposure estimates to likely underestimate cancer potency.

Unit risks were developed for four smoking categories. The unit risk for lifetime exposure to 1 µg/m3 arsenic ranged from 0.3 per thousand (never smokers) to 7.3 per thousand (heavy smokers).

Occupational Safety and Health Administration (OSHA)
NIOSH (1985) recommended a 15-minute ceiling of 0.002 mg/m³. OSHA established a standard of 0.01 mg/m³ for airborne inorganic As (OSHA, 1989). U.S. EPA water quality criteria for human health are: 0 (2.2 ng/l) for the consumption of aquatic organisms and drinking water and 0 (25 ng/l) for drinking water only (EPA, 1986). Based on the non-threshold theory for potential carcinogens, zero concentration is recommended for maximum protection of human health, while the concentration in parentheses corresponds to a 104 excess lifetime cancer risk. The MCL (maximum contaminant level) for drinking water is 0.05 mg/l with a MCLG (maximum contaminant level goal) of the same value. The allowable food residue is 0.65 mg/lb (Arena, 1986) .

Table 2. Worker Permissible Exposure Limits

ACGIH TWA:	0.2 mg As/m^3
STEL:	NL
Ceiling:	NL
OSHA PEL:	CA
Ceiling:	10 µg/m^3
IDLH:	CA
NIOSH CEILING:	2 µg As/m^3
MSHA:	NA

CA = Carcinogenic
NA = Not Available
NL = Not Listed
(References: OSHA, 1989; NIOSH, 1985; ACGIH, 1988)

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TOXICOLOGY PROFILE

BENZENE

Synonyms: Benzol, Cyclohexatriene, Coal naphtha, Cyclohexatriene, Phenyl hydride, Polystream, Pyrobenzol

CAS No: 71-43-2

Boiling point: 80.1 °C

Color: Clear, colorless liquid, Rhombic crystals

Conversion Factors: 1 ppm = 3.242 mg/m^3 @ 20 °C 1 mg/m^3 = 0.3 ppm Blood/air partition coefficient = 7.8

DOT Designation: highly flammable liquid

Evaporation half life:
(from water): 2.7 - 3.5 hrs.

Evaporation rate: 2.8 (ether = 1)

Flammable limits:
autoignition: 1044 °F
flash point: -11 °C (12 °F)
LEL: 1.3%
UEL: 7.1%
burning rate: 6.0 mm/min

Henry's Law Constant: 5.59 x 10^{-3}atm-m^3/mole @ 20°C

Melting point: 5.5 °C

Molecular formula: C_6H_6

Molecular weight: 78.11

Odor:

Threshold: 4 - 5 ppm
Recognition: 34-119 ppm
Characteristics: strong aromatic

Solubility:
Water: 820 mg/l @ 22°C, 1000 mg/l @ 25°C
K_{ow} : 1.56 - 2.15
Other: Miscible in non aqueous solvents
K_{oc} : 0.3 - 100

Specific gravity: 0.875 g/cm^3 @ 20°C

Vapor density: 2.7 (air=1)

Vapor pressure: 60 mm Hg @ 15°C
76 mm Hg @ 20°C
100 mm Hg @ 26°C

Viscosity: 0.654 centipoise at 20°C.

(Source: HSDB, 1990; Merck 1989; ATSDR, 1989)

USES: Benzene is used primarily as a solvent, degreaser, fuel additive, and starting material for pharmaceutical and synthetic chemicals. It is used in the manufacture of a wide variety of consumer goods including plastic containers, radios, toys, sporting goods, furniture, appliances, automobiles, tires, lubricants, dyes, drugs and agricultural chemicals. Benzene also occurs in gasoline at concentrations between 1% to 5%. Most of the produced benzene remains in petroleum fuels such as gasoline (ATSDR, 1987).

The use of benzene in industrial processes and by consumers has been decreasing. In the past, benzene was used as a solvent in paints and paint strippers, arts and crafts supplies, rubber cement in tire patch kits, carburetor cleaners and denatured alcohol. Other solvents have now replaced benzene in these uses (ATSDR, 1987).

ACUTE TOXICITY: The characteristic pattern of acute benzene intoxication is central nervous system stimulation followed by respiratory depression, loss of consciousness and death (Goldstein, 1977). The onset and progression of these signs and symptoms is dose dependent.

The LD_{50s} for various species, as reported in RTECS (1990), are as follows:

Table 1.
Acute Toxicity Values for Benzene in Laboratory Animals

Species	*Route of Administration*	*Type*	*Test Concentration*
Rat	Oral	LD_{50}	3306 mg/kg
Rat	Intraperitoneal	LD_{50}	2890 µg/kg
Mouse	Oral	LD_{50}	4700 mg/kg
Mouse	Intraperitoneal	LD_{50}	340 mg/kg

Ingestion: Acute LD_{50} values for oral exposure to benzene in rats are age-dependant, and range from 0.87 g/kg to 5.6 g/kg (Kimura, et al., 1970). Benzene can be readily absorbed from the gastrointestinal tract following oral dosing. Following the oral administration of ^{14}C labelled benzene to rabbits, approximately 90% of the administered dose was recovered in the exhaled air and urine (Parke and Williams, 1953).

In humans, case studies of accidental or intentional ingestion also indicate that benzene is readily absorbed. The signs and symptoms following benzene ingestion include: a burning sensation of the mouth and esophagus, nausea, vomiting, dizziness, staggering gait, somnolence, loss of consciousness, and delirium. Other CNS effects include visual disturbances and convulsions, feelings of excitement and euphoria with sudden onset of weariness, fatigue and sleepiness followed by coma. Due to the volatility of benzene, there is also the danger of aspiration into the lungs during vomiting resulting in a chemical pneumonitis. Chemical pneumonitis is characterized by choking, shortness of breath, sensation of constriction in the chest and fever (Sandmeyer, 1981). Cardiovascular effects include shallow rapid pulse and pallor, followed by flushing. Progressive CNS depression, respiratory insufficiency, and ventricular fibrillation may result in death. The estimated fatal oral dose for humans has been reported to range between 9 - 30 grams or 129 mg/kg to 429 mg/kg (Sandmeyer, 1981).

Inhalation: Dogs and mice exposed to 600 to 1000 ppm (2,089–3,482 mg/m^3) of benzene via inhalation for 12 to 15 days developed leukopenia and fatal anemia, respectively (Hough and Freeman, 1944; Petrini, 1941). Rats, guinea pigs, rabbits, and monkeys also developed leukopenia when dosed via inhalation at 80 to 85 ppm (279 - 295 mg/m^3) for 136 to 187 times (Wolf et al., 1956).

Winek and Collum (1971) reported three fatal cases associated with "sniffing" episodes. One boy died as a result of sniffing rubber cement; another boy died after sniffing benzene. The last victim accidentally shot himself while in a euphoric state after sniffing glue. Blood and tissue analysis ranged as follows: blood - 0.094 - 6.5 mg%; fat - 2.23 mg%; brain - 3.9 mg%; kidney-0.55 - 1.9 mg%. At autopsy, inflammation of the respiratory tract, hemorrhages in the lungs, congestion in the kidneys, and cerebral edema were seen. Notably, there were no hematological effects. Sandmeyer (1981) estimated that exposure to 19,000 - 20,000 ppm for 5 to 10 minutes may prove fatal and the toxicity is most likely attributable to benzene directly rather than any of its metabolites.

Skin contact: Benzene was reported to be moderately irritating to the skin of rabbits. Repeated exposures (20 applications) produced erythema, edema, exfoliation, blistering, and moderate necrosis (Wolf et al., 1956).

Eye contact: Direct contact with undiluted benzene produces moderate eye irritation with slight transient corneal injury. Small areas of superficial corneal necrosis may also be noted (Wolf et al., 1956).

CHRONIC TOXICITY: Benzene has been associated with hematologic effects in occupationally-exposed humans. Adverse human effects include anemia, leukopenia and thrombocytopenia. Chronic benzene exposure may lead to a decrease in all circulating cells (pancytopenia) or failure to manufacture blood cells altogether (aplastic anemia) (Goldstein, 1977). These effects are hypothesized to be related to the metabolites of benzene acting on the precursors of circulating blood cells formed in the bone marrow. Because the toxic effect is difficult to simulate in experimental animals, the mechanism of this action has been difficult to determine.

Ingestion: In humans, chronic exposure to benzene by ingestion is rare. However, Schwarz (1932) reported the clinical picture and progress

of a 28-year-old male who ingested a gasoline/water mixture for several months as a self administered remedy for hypochondriacal gonorrhea. Severe weakness, muscular atrophy and polyneuritis with weak or absent reflexes developed. A slow recovery followed the discontinuation of gasoline ingestion, with hospital discharge after seven months. (It can be speculated that the benzene content of the gasoline at that time was probably between 5 - 8%. However, part of the neuromuscular effects noted in this case history might also be attributed, in part, to the n-hexane content of gasoline).

Inhalation: Chronic occupational exposure to benzene has resulted in adverse hematologic effects, including anemia, leukopenia, thrombocytopenia, aplastic anemia, and leukemia.

Lifetime inhalation exposure to 100 or 300 ppm (348 or 1,045) benzene by rats and mice resulted in lymphocytopenia, anemia, and decreased survival time (Snyder et al., 1978). Later evaluation of the same study showed preliminary evidence of carcinogenicity (U.S. EPA, 1987). There is a growing body of evidence that suggests that benzene produces leukemia in Sprague-Dawley rats (Goldstein et al., 1982).

Skin contact: Dermal absorption of benzene is considerably less than the absorption by inhalation. Hemotoxicity in humans or animals by dermal contact alone does not appear to have been reported (ATSDR, 1987).

Eye contact: see "Eye contact" under ACUTE TOXICITY.

SENSITIZATION: Eosinophilia, an indication of an allergic response, has been noted in several workers chronically exposed to benzene by a number of investigators (Aksoy et al., 1971).

TARGET ORGAN EFFECTS: The most significant effects of benzene are disruption of hematopoiesis, neurotoxicity, and increases in the risk of some forms of hematopoietic cancer.

ABSORPTION-METABOLISM AND EXCRETION: Benzene can be absorbed into the body by inhalation, ingestion or dermal contact. It is highly lipid soluble, and therefore tends to distribute to fatty tissues (U.S. EPA, 1987). After ingestion, approximately 90% of the administered dose is absorbed. Following inhalation, it is estimated that 50% of a continuous dose is absorbed (Nomiyama and Nomiyama, 1974a;b). Blank and McAuliffe (1985) calculated that a working adult would dermally absorb 1.5 µl/hr from whole body exposure ($2m^2$) to an atmosphere of 10 ppm benzene, while 7.5 µl/hr would be absorbed by the lungs under the same conditions. NIOSH calculated that a worker in direct contact with petroleum naphtha in tire manufacturing could absorb approximately 6 mg through the intact skin (US OSHA, 1985). In the rhesus monkey or hairless mouse, dermal absorption was reported to be <1% after a single direct application but did increase with repeated administration (Maibach and Anjo, 1981).

Both human and animal studies indicate that benzene must undergo metabolic transformation to exert its toxic effect on the hematopoietic system. The liver and the bone marrow contain the enzymes necessary for the metabolic transformation of benzene to benzene oxide, hydroquinone, phenol, catechol and trans, trans mucondialdehyde. The metabolites of catechol, p-benzoquinone, hydroquinone and phenol are known to cause lymphoid tissue suppression. Administration of benzene to rats, dogs, rabbits, and mice via inhalation and/or ingestion results in its rapid uptake and excretion, mainly via exhalation of unchanged benzene (Rickert et al., 1979; Parke and Williams, 1953; Andrews et al., 1977). In humans, the elimination of benzene is biphasic, with approximately 16% eliminated unchanged via exhalation within 5 hours (Nomiyama and Nomiyama, 1974a, 1974b). The remaining benzene is stored in fatty tissue and is excreted slowly .

REPRODUCTIVE TOXICITY: There is no evidence that benzene is teratogenic. However, benzene has been shown to be a growth inhibitor *in utero* (U.S. EPA, 1983) and show embryotoxic/fetotoxic effects at dose levels that are also maternally toxic (ATSDR, 1987). CD-1 mice exposed to 300 ppm benzene vapor , 6 hrs./day, 5 days/week for 13 weeks demonstrated bilateral ovarian cysts, decreased sperm counts with an increase in deformed sperm, and testicular atrophy and degeneration (Ward et al., 1985). Testicular degeneration was also noted in rabbits exposed to 80 ppm (Wolf et al, 1956) while the exposure of female rats to 210 ppm 10 days prior to mating and for 3 weeks after mating resulted in a complete absence of litters (Gofmekler, 1968).

Female workers exposed to levels in excess of 30 ppm developed menstrual disturbances and possibly decreased fertility (Barlow and Sullivan, 1982). The levels of exposure in this study exceeded the levels of exposure commonly seen in the present day workplace (ATSDR, 1987).

NEUROTOXICITY: Acute neurotoxic effects of benzene include: drowsiness, dizziness, headache, delirium, and perhaps loss of consciousness. These neurological effects are believed to be due directly to benzene and not any of its metabolites (ATSDR,1987).

Incident reports on low level, chronic exposures have suggested that benzene may produce a neurasthenic syndrome and polyneuritis. The neurotoxicity of benzene has not been extensively studied, perhaps owing to the fact that the chronic exposure to benzene and the associated hematoxicity may mask any neurotoxic effects (Sandmeyer, 1981).

IMMUNOTOXICITY:
Benzene-induced hematoxicity involves the erythroid, myeloid and lymphoid lineages of blood cells, of which the lymphoid line appears to be the most susceptible. Inhalation exposure of mice to 300 ppm, 6 hr/day for 115 exposures impaired the ability of T and B cells to respond to mitogenic stimuli with a marked reduction in the number of B-lymphocytes in the bone marrow and spleen and the T-lymphocytes in the thymus and spleen. B cells were considered to be more sensitive than T cells (Rosen and Snyder, 1985). These same authors also studied host resistance to infection with Listeria monocytogenes and concluded that benzene exposure delays the cell mediated immune response.

Other immunosuppressive effects attributed to benzene exposure include a depression of antibody formation in mouse spleen cells (Wierda et al., 1981) and a reduction in the number of circulating B-lymphocytes (Irons and Moore, 1980). In the spleen, benzene metabolites are cytotoxic and reduce the total number of cells as well as progenitor cells in the spleen and bone marrow.

GENOTOXICITY: Benzene has been shown to be non-mutagenic in Drosophila melanogaster and in several *in vitro* assays. However, benzene oxide, the presumed initial metabolite of benzene, is mutagenic without activation in the Ames Test (U.S. EPA, 1987). Morimoto and Wolff (1980) observed that hydroquinone and catechol increased the frequency of sister chromatid exchanges in human lymphocytes. Tunek et al., (1980) identified hydroquinone as the principal metabolite responsible for binding benzene in rat liver microsomes. Pfeiffer and Irons (1981) reported that hydroquinone and benzoquinone inhibited lymphocyte mitogenesis at concentrations that did not produce cytotoxicity.

CARCINOGENICITY: Benzene has been classified as a human carcinogen by IARC, NTP, EPA and OSHA based on the results of epidemiologic studies. Benzene is among the few substances given an "A" weight of evidence rating by the EPA indicating there is adequate/sufficient data from human studies to show that the compound is classified as a carcinogen.

An association between benzene exposure and lymphatic and hematopoietic cancer (particularly leukemia) has been documented in epidemiologic studies (McMichael, 1988). Acute myelogenous leukemia (AML) is most frequently associated with benzene exposure. Ott et al. (1978) examined long-term mortality in a cohort of 594 workers occupationally exposed to benzene at the Michigan Division of Dow Chemical Company. Estimates of cumulative benzene exposures were based on work histories and industrial hygiene records. Fifty-three individuals who experienced concomitant exposure to arsenicals, asbestos, or high vinyl chlorides were analyzed separately. Among the remaining cohort, observed deaths were less than expected based on U.S. white male mortality, with no statistically significant increases due to any cause of death. Three cases of leukemia were observed (two cases of acute myelogenous leukemia, one case of chronic myelogenous leukemia). Based on Third National Cancer survey data, the expected incidence of myelocytic leukemia was 0.8 cases. A clear dose-response relationship between benzene exposure and leukemia could not be established.

The mortality experience of workers exposed to benzene during the manufacturing of rubber hydrochloride has been reported by Infante et al. (1977a&b) and Rinsky et al. (1981). A five-fold increase in risk of developing leukemia of any type and a ten-fold excess in deaths attributable to myelogenous and monocytic leukemia were observed (Infante et al., 1977a). Two control populations were used as comparative standards: the general U.S. male population and a non-exposed working male (N = 1447) population.

After strong criticism of study methodology from Tabershaw and Lamm, Rinsky et al. (1981) re-analyzed their own data using Tabershaw and Lamm's suggested methodology. The re-calculated risk was 8.5- fold versus the original ten-fold risk for excess deaths due to myelogenous and monocytic leukemia. Rinsky et al. (1981) examined data from the occupational cohort of rubber workers followed by Infante et al. (1977a). Analysis was based on a 98% complete vital status follow-up. Although the Rinsky et al. study was limited in its characterization of exposure, the authors

concluded that a dose-response relationship between benzene exposure and leukemia exists. Because dermal exposure was not measured, and anecdotal evidence of significant dermal exposure exists (i.e., workers drenched in benzene during some of the operations), the authors may have underestimated benzene exposure and subsequently overestimated the quantitative leukemia risk (Wong, 1987).

Wong (1987) investigated mortality patterns among 7,676 chemical workers from seven plants exposed to benzene. As compared to chemical workers with no benzene exposure, workers continuously exposed to benzene experienced an increased risk of hematopoietic cancer (Standard Mortality Ratio = 320, $p < 0.05$).

EPIDEMIOLOGY: Epidemiologic studies of cohorts occupationally exposed to benzene have found increases in deaths due to lymphatic and hematopoietic cancer. Findings are reviewed under CARCINOGENICITY.

ENVIRONMENTAL FATE: Benzene in the atmosphere is not directly photolyzed but does react with photochemically produced hydroxyl radicals. The estimated half-life is of the order of a few days. The reaction time in polluted atmospheres which contain nitrogen oxides is estimated to be four to six hours (HSDB, 1989).

Benzene has been shown to undergo microbiological biodegradation both aerobically and anaerobically. Under aerobic conditions, effective biodegradation can occur within 7 days to several months. Anaerobic degradation is somewhat longer; reportedly 6 months to 2 years (ATSDR, 1987).

ENVIRONMENTAL TOXICITY: Benzene is ubiquitous in the environment. It has been identified in soils, water supplies, finished water, indoor and outdoor air, cigarette smoke and foods.

Outdoor ambient air levels range from 0.2 ppb to a high of 60 ppb in Tuscaloosa, Alabama and Pittsburgh, Pennsylvania. More common values for large cities are in the range of 10 to 30 ppb. Indoor air in Elizabeth and Bayonne, New Jersey have reached a maximum of 158 ppb.

Environmental lethal dose concentrations for aquatic species are presented in Table 2.

Table 2.
Environmental Lethal Dose Concentration

Species	*Value*	*Dose*	*Time*
Bluegill Sunfish	LD_{50}	29 mg/l	24-48 hr.
Bluegill Sunfish	LD_{100}	34 mg/l	24 hr.
Gold fish	LD_{50}	46 mg/l	24 hr.

(Reference: Verschueren, 1983)

REGULATORY STATUS: The U.S. EPA and IARC (International Agency for Research on Cancer, 1982) has classified benzene as a Group A (human carcinogen) in its weight of evidence classification system based on sufficient human evidence of carcinogenicity. The EPA Cancer Assessment Branch, using the data from three epidemiological studies (Rinsky et al., 1981; Ott et al., 1978 and Wong et al., 1983) and a linear dose-response model, has calculated a cancer potency factor of 2.90×10^{-2} $(mg/kg/day)^{-1}$ for both oral and inhalation routes of exposure (IRIS, 1986). These studies are based on historic reconstructions of exposure scenarios dating from approximately 1940 to 1971 and attempt to show a correlation between benzene exposure and increased incidences of myelogenous leukemia in occupationally exposed workers. Reconstruction of exposure scenarios are based on personnel data, job descriptions and other anecdotal reports. Since these investigations were not designed to support epidemiological studies but rather to identify problems with regulatory compliance, some data gaps exist. Results from these studies are inconclusive at this point.

The California Air Resources Board has established a range for the cancer potency factor of 2.65×10^{-2} to 1.85×10^{-1} $(mg/kg/day)^{-1}$ (CARB, 1989). The unit risk values for this range are 7.5×10^{-6} to 5.3×10^{-5} (CAPCOA, 1990). The cancer unit risk for drinking water is 8.3×10^{-7} per µg/L based on a one-hit extrapolation method and on the assumption that an adult human consumes 2 liters of water per day (IRIS, 1990). The unit

risk factor is defined as the additional risk of cancer death associated with drinking water containing 1 µg/L or breathing air containing 1 µg/m^3 of benzene for a human lifetime. The EPA has not established an RfD (IRIS, 1989). The exposure limit values listed or recommended by different agencies are presented in Table 3.

Table 3. Worker Exposure Limits

ACGIH TLV:	10 ppm (30mg/m^3) A-2 carcinogen
STEL:	N/A
OSHA PEL:	1 ppm (3.5 mg/m^3)
IDLH:	N/A
NIOSH REL:	N/A
MSHA:	N/A

N/A - not available
(References: ACGIH, 1988; NIOSH, 1985)

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TOXICOLOGY PROFILE

BERYLLIUM

Synonyms: Beryllium metallic, Beryllium-9, Glucinium, Glucinum, Beryllium dust, Beryllium (metal powder).

CAS No: 7440-41-7

Boiling range: 2970 °C

Color: Gray metal, Close packed hexagonal structure. A grayish-white, hard light metal.

DOT designation: Poison, flammable solid

Explosive limits: Beryllium forms explosive mixtures in air. Hazard is greater as the fineness increases.

Flammable limits:

Autoignition: Finely divided beryllium burns in air. Reaction between beryllium and vapors of phosphorus proceeds with incandescence.

Flash Point: N/A
LEL: N/A
UEL: N/A

Henry's law constant: NA

Melting point: 1287 °C

Molecular formula: Be

Molecular weight: 9.012

Odor: none

pH: 2.7 for isomolar beryllium sulfate

Solubility
water: Insoluble in cold water, mercury; slightly soluble in hot water.

K_{ow}: NA

other: Soluble in dilute acids and alkalies. Insoluble in nitric acid.

K_{oc}: NA

Specific gravity: 1.85 at 20° C

Vapor density: NA

Vapor pressure: 10 mm Hg @ 1860° C

Viscosity: N/A

Ca = carcinogen
NA = not available
N/A = not applicable

(References: ATSDR, 1987; CDC, 1988; HSDB, 1990)

COMPOSITION: Beryllium is usually found in localized deposits of beryl ($Be_3Al_2Si_6O_8$). Colored variants of beryl form the gemstones of emerald, and aquamarine. Ores are first converted to beryllium hydroxide, then

to the fluoride and finally reduced with magnesium to obtain the beryllium metal. The metal can then be reacted with carbon and melted with copper to obtain a master alloy (Reeves, 1986).

USES: Beryllium is a neutron moderator in nuclear weapons and test reactors, and a source of neutrons when bombarded with alpha particles. This reaction yields about 30 neutrons per million alpha particles. It is also used in manufacturing lightweight alloys. Beryllium is used as a heat sink material in aircraft brakes, in the manufacture of aerospace guidance systems and as a meteorite and heat shield material for spacecraft. In the form of hydrides, nitrides and carbides, it has been used as an experimental solid rocket fuel. It has also been used in the manufacture of mirrors used in space optics. It is used in the production of brass, as well as in navigational systems, aircraft/satellite structures, and missile parts. More conventional uses of beryllium include uses as a fluorescent lamp powder, ceramics and alloy in non sparking tools (ACGIH, 1990).

ACUTE TOXICITY: Beryllium is considerably more hazardous by inhalation than any other route of exposure since beryllium compounds are not easily absorbed through the skin or from the gastrointestinal tract. Beryllium oxide which imparts corrosion resistance to metals has a chemical reactivity and presumably a toxicity that is inversely related to the temperature of firing (Reeves, 1986). The direct cause of death in acute poisoning is believed to be due to hypoglycemia shock,spasms and respiratory paralysis (Reeves, 1986). Acute toxicity values of different beryllium salts are presented in Table 1.

TABLE 1. Lethal Values in Different Species

Compounds	Species	LC50 (mg/m3)	LC100 (mg/m3)	LD50 (mg/kg)
Beryllium sulfate	Several	0.5-2.0		
Beryllium sulfate	Mice		5.0 (12 days)	
High fired beryllium oxide	Several	30 (no effect)		
Low fired beryllium oxide	Rats & Dogs	3 (pulmonary damage)		
Beryllium chloride	Rats Mice			200 6.2
Be fluoride	Mice			18-20
Be sulfate	Rats Mice Guinea Pigs			120 1.5 1.5
Be nitrate	Mice			11.0

(adapted from Reeves, 1986)

Ingestion: All studies indicate that beryllium is poorly absorbed from the gastrointestinal tract. Therefore, ingestion is not considered a likely hazard (Hamilton and Hardy, 1974).

Inhalation: Short term exposure to beryllium in the air can produce substernal pain, weight loss, nonproductive cough, shortness of breath and irritation of the eyes, respiratory system and skin (CDC, 1988). Brief exposures to high concentrations of beryllium in occupational settings have resulted in rhinitis, pharyngitis, tracheobronchitis, and pneumonitis. The severity of pneumonitis increases with increasing level of exposure, and may lead to pulmonary edema and death. Symptoms from an acute exposure may appear in a few hours with generally complete recovery in 1 to 12 weeks. The typical signs of chemical pneumonitis are anorexia, weight loss, weakness and varying degrees of cyanosis. The physical signs include lowered vital capacity, fine to coarse sibilant rales, and rapid pulse. X-rays usually reveal haziness and punctuate infiltration throughout the lower lung fields, or in severe cases, consolidation. Pneumonitis may result from a single exposure to beryllium and is occasionally fatal. Fumes of beryllium in refining or

manufacturing produce metal fume fever, coryza, and bronchitis (CDC, 1988).

Rats exposed by nose-only apparatus to 800 $\mu g/m^3$ beryllium metal for 50 minutes demonstrates developed an acute, necrotizing, hemmorhagic pneumonitis and fibrosis. Two to 4 weeks after the exposure the inflammatory response quieted but later returned and progressed to a chronic inflammatory condition (Haley et al., 1990).

Skin contact: Contact dermatitis and a non-healing ulceration at the site of the injury, and/or subcutaneous nodules all may occur following beryllium exposure. Both allergic and irritant contact dermatitis have been associated with dermal exposure to soluble beryllium salts (Proctor et al., 1988).

Eye contact: Beryllium in contact with the eyes can produce a chemical conjunctivitis and corneal burns (Proctor et al., 1988). See also Eye contact under CHRONIC TOXICITY

CHRONIC TOXICITY:
Chronic beryllium disease is characterized by dyspnea, cough, and weight loss. It may sometimes be associated with systemic effects in the form of granulomas in skin and muscle as well as with effects on calcium metabolism.
Deaths from chronic beryllium disease (CBD) are often due to cor pulmonale. A long latency period is typical; 20 years may pass between the last exposure and the diagnosis of the disease (EPA, 1987). Out of 60 cases of chronic beryllium disease, 18 participants had died: 14 from cor pulmonale, 1 from respiratory insufficiency, 1 from cardiac arrest, 1 from virus pneumonia, 1 from renal insufficiency, and 1 from an unstated cause (HSDB, 1990).

Ingestion: Ingestion is not considered a likely route of chronic intoxication because beryllium is very poorly absorbed from the gastrointestinal tract (Hamilton and Hardy, 1974).

Inhalation: Chronic beryllium disease (berylliosis) may develop years after the last exposure to beryllium. The disease is characterized by cough, fatigue, weakness, weight loss, chest pain, and granulomatous lung disease. The disease is progressive, and may result in death due to cor pulmonale. An autoimmune basis for berylliosis has been postulated (EPA 1987), since the condition can occur with relatively low exposures to beryllium.

Pulmonary granulomatous disease may appear in 3 months to 15 years, often after short exposure to low concentrations. Weight loss and marked dyspnea is noted with a death rate of about 25% (HSDB, 1990). There is uncertainty as to complete recovery.

Eye contact: Exposure to beryllium dusts can irritate the eyes (CDC, 1988). Chronic exposure has produce a conjunctivitis that may be related to the production of a beryllium allergy (Reeves, 1986).

Skin contact: Accidental implantation of beryllium metal or crystals into the skin can produce tissue necrosis and ulceration. A large percentage of workers exposed to beryllium dusts experience erythematous, and vesicular rash. This metal may also cause eczematous dermatitis with maculopapular lesions (CDC, 1988).

SENSITIZATION: Hypersensitization may cause chronic beryllium disease in people following relatively low exposures. Beryllium can bind to lymphocyte membranes, which may explain the sensitizing properties of the metal. Hypersensitization can be detected with the lymphoblast transformation test (EPA, 1987).

TARGET ORGAN EFFECTS: The lung is the main target organ for toxicity following exposure to beryllium and its compounds. In humans, this toxicity is manifested in the form of acute pneumonitis or a more chronic form of lung disease (berylliosis) in which granulomas develop in the lung (EPA, 1986).

ABSORPTION-METABOLISM AND EXCRETION: Absorption of beryllium through intact skin is slight since it is bound tightly in the epidermis (EPA, 1987). Inhalation and ingestion are the main routes of intake. Soluble beryllium compounds are partly transformed to more insoluble forms in the lungs, resulting in long retention times following exposure to all types of beryllium compounds but the absorption from the lungs has been assumed to be complete (EPA, 1986). Immediately following exposure to beryllium sulfate aerosol, 67% of the retained dose was found in the lungs and 15 % was found in the skeleton. After 17 days, about 80% of the total body burden was found in the skeleton and about 18% in the lungs (Zorn et al., 1977).

In soft tissues, the highest concentrations are found in the liver (Reeves, 1965; Mullen et al, 1972) but detectable amounts are also found in the intestines, lungs, kidneys stomach and spleen (ATSDR, 1987).

Beryllium can be transferred from the lungs to the gastrointestinal tract, but only a minor portion is absorbed from the gut (probably less

than 1%). Of the deposited beryllium that is absorbed, part is rapidly excreted by the kidneys and part will be stored in the bone. Ingested and unabsorbed beryllium is eliminated via the feces (EPA, 1987).

REPRODUCTIVE TOXICITY: Clary et al. (1975) found no consistent effect on reproductive performance in rats treated intratracheally with beryllium oxide at 0.2 mg and allowed to mate repeatedly over a 15-month period (ATSDR, 1987).

IMMUNOTOXICITY: Early epidemiological studies suggested that berylliosis involved an immunological factor since patch testing appeared to be sensitizing and exacerbated the beryllium disease. It now appears well established that beryllium hypersensitivity is essentially a cell mediated disease and humoral antibodies are not produced. Sensitivity has been transferred to guinea pigs with lymphoid cells but serum transfer is ineffective. Furthermore, anti-lymphocyte serum can inhibit the cutaneous reaction to beryllium in guinea pigs (Reeves, 1986). It is currently believed that the beryllium ion acts as a hapten to provide the immunological reaction (Krivanek and Reeves, 1972).

Humans and dogs have been reported to develop beryllium-specific immune responses within the lung following acute beryllium inhalation exposure (Haley and Bice, 1988). Following an initial inflammation reaction, antigen induced immune granulomas consisting of macrophages, lymphocytes and plasma cells. At a later time, chronic proliferative lesions can develop (Freiman and Hardy, 1970; Haley et al., 1989). Beryllium specific immune responses do not appear to develope in the rat (Haley et al., 1990).

NEUROTOXICITY: Neither the central nervous system nor the peripheral nervous system appear to be direct target organ of beryllium toxicity. As a result, any neurological manifestations of toxicity would only be secondary to other more direct effects such as inhibition of enzymes associated with energy production.

GENOTOXICITY: The available literature indicates that beryllium has the potential to cause gene mutations, chromosomal aberrations and sister chromatid exchanges in cultured mammalian somatic cells (EPA, 1987).

CARCINOGENICITY: Several investigators have followed cancer mortality among beryllium manufacturing workers employed at Kawecki-Berylco Industries and Brush Beryllium Co. However, as reviewed in EPA (1987), findings are limited by study methodology and lack of consideration for confounding factors such as smoking. EPA concluded that the epidemiologic evidence of beryllium carcinogenicity among exposed workers is inadequate (EPA, 1987).

A study was undertaken of mortality patterns among white males entered into a beryllium case registry (BCR) while alive with a diagnosis of beryllium related non-neoplastic respiratory symptoms or disease. Analyses demonstrated an excessive risk of lung cancer among those subjects in the BCR who had been previously diagnosed with acute chemical pneumonitis or bronchitis secondary to short-term beryllium exposure. In evaluating the excessive lung cancer risk in this population, consideration should be given to the competing effects from the high case fatality rate of non-neoplastic respiratory disease. This excessive risk of lung cancer could not be explained on the basis of cigarette smoking per se. The findings of the present study using subjects in the BCR are consistent with results of animal studies that over 30 years ago first demonstrated beryllium to be a carcinogen and with numerous epidemiologic studies demonstrating an increased risk of lung cancer among workers occupationally exposed to beryllium and its compounds (Infante, 1980).

In a 1980 study of beryllium plant employees (Wagoner, et al, 1980), 47 deaths were observed among 3055 white males with a median employment duration of 7.2 months. The study followed 2068 of these employees for 25 years, and found a statistically significant increase in deaths from lung cancer (20 deaths). However, the number of expected lung cancer deaths was recalculated following the release of mortality data for 1969-1975, and the increase in Wagoner's study was found to be significant only among workers followed for 25 or more years. Furthermore, the increased incidence was found to be insignificant when the number of expected deaths was adjusted for smoking (EPA, 1986).

Schroeder and Mitchener (1975a) reported a slight, but statistically insignificant, increase in the cancer incidence among Long-Evans rats dosed at 5 ppm beryllium sulfate in drinking water for a lifetime. Males in the 5 ppm group had a slightly higher rate (9/33) than the control group (4/26). In another study, Schroeder and Mitchener (1975b) dosed Swiss mice with 5 ppm beryllium sulfate in drinking water for a lifetime. They found a statistically insignificant increase in lymphoma leukemia incidence in the females (9/25, compared to 3/47 in the controls).

At least 12 different studies reported that intravenous injections of beryllium compounds induced osteogenic sarcomas in rabbits. These cancers were also induced by intramedullary injection in at least four studies (EPA, 1987). Bone tumors were induced by beryllium oxide, zinc beryllium silicate, beryllium phosphate, beryllium metal, and beryllium silicate. No bone tumors were found following intravenous injections of beryllium oxide or zinc beryllium silicate in rats or guinea pigs (Gardner and Heslington, 1946).

EPIDEMIOLOGY: Epidemiologic studies have investigated cancer mortality among cohorts occupationally exposed to beryllium (Mancuso, 1979; 1980). Pertinent studies are reviewed under CARCINOGENICITY.

ENVIRONMENTAL FATE: The major source of the emission of beryllium into the environment is the combustion of coal and fuel oil which releases particles and fly ash containing beryllium into the atmosphere. Beryllium released in this way is usually removed by wet or dry deposition. The rate of dry deposition is a function of the particle size, wind speed and surface roughness. Beryllium on particulates smaller than 1 mm reportedly have remained aloft for about 10 days (Gladney and Owens, 1976). Beryllium is tightly adsorbed to sediments, clay and organic matter in soil and water because it displaces covalent cations which share common sorption sites. There is no evidence that beryllium is significantly biomagnified in the food chain (ATSDR, 1987).

ENVIRONMENTAL TOXICITY: The average beryllium content of urban air has ranged between 0.3 ng/m^3 in Boston to 3.0 ng/m^3 in Pittsburgh. However, most of this may have been contributed by the burning of coal which contains up to 3 ppm of beryllium. Agricultural soils and natural waters contain beryllium in the several ng/g range. Common trees such as the aspen, willow and birch can efficiently accumulate beryllium and act as indicators of exploitable ore deposits. Levels in the tissues of the tree can reach 3 ng Be/g. The beryllium content of food stuffs appears to depend on local growing conditions and concentrations. The following table (Table 2) lists two agricultural regions and the beryllium content in some representative food materials.

Table 2. Beryllium Content of Food Samples

Locale	Food	Beryllium Content
Australia	eggs	10-14 ng/g ash
	nuts	10-20
	vegetables	10-30
	seafood	100-230
		Ash Content = 0.65-15% of the fresh weight
West Germany	polished rice	80 µg/g dry substance
	potatoes	240
	green lettuce	330
	cigarettes	(moisture content = 90% fresh weight)
		0.47 - 0.74 µg/cigarette
		1.6 - 10% escaping in smoke

(adapted from Reeves, 1986)

It has been speculated that the difference in the food content between West Germany and Australia is due to past use of rocket propellants.

REGULATORY STATUS: The U.S. EPA considers beryllium to be a Group B2 carcinogen with sufficient evidence in animals but inadequate evidence in humans (IRIS, 1990). Based on the data of Wagoner et al. (1980), the EPA derived cancer potency factor for inhalation exposure is 8.4 (mg/kg/day)-1. However, this study when adjusted for smoking, did not produce a significant increase in deaths from cancer. Beryllium is not considered to be carcinogenic via the oral route. The EPA lists an oral cancer potency slope of 4.3 per mg/kg/day beryllium (IRIS, 1990).

The U.S. EPA derived oral RfD for beryllium is 5×10^{-3} mg/kg/day based on the NOAEL (5 ppm in drinking water) from a one dose chronic rat oral bioassay. An uncertainty factor of 100 was also used (EPA, 1988). The ambient water

quality criteria for the protection of human health from ingestion of fish and water is 6.8 x 10^{-3} mg/l which corresponds to a 1 x 10^{-6} incremental lifetime increase of cancer risk (ATSDR, 1987; IRIS, 1990).

The U.S. Clean Air Act requires ambient beryllium levels to remain below 10 ng/m^3 in inhabited places (Reeves, 1986).

The occupational exposure limit values available for beryllium are listed in the following table (Table 3).

TABLE 3. WORKER PERMISSIBLE EXPOSURE LIMITS

ACGIH TWA:	0.002 mg/m^3
STEL:	N/A
Ceiling:	5 µg/m^3
peak:	25 µg/m^3
OSHA-PEL:	2 µg/m^3
IDLH:	CA
NIOSH REL:	CA not in excess of 0.5 µg/m^3
MSHA:	NA

CA = carcinogen
NA = not available
(References: NIOSH, 1985; HSDB, 1990)

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TOXICOLOGY PROFILE

CADMIUM

Synonyms: Colloidal cadmium, Cadmium metal, Kadmium (German)

CAS No: 7440-43-9

Boiling point: 765°C

Color: Silver white, Blue-tinged, lustrous metal

DOT designation: NA

Corrosivity: *Highly corrosion resistant*

Flammable limits:

Autoignition: Cadmium dust ignites spontaneously in air and is flammable and explosive when exposed to heat, flame, or by chemical reaction. When heated strongly it emits toxic fumes of Cd.

Flash point: *Dependent on specific* compound

LEL: Dependent on specific compound

UEL: Dependent on specific compound

Henry's law constant: NA

Melting point: 321°C

Molecular formula: Cd

Molecular weight: 112.41

Odor: Odorless

Solubility:

water: Cadmium oxide and sulfide are almost insoluble in water. The arsenate, dichloride and sulfate forms are quite water soluble.

K_{ow}: NA

other: Soluble in acid, ammonium nitrate, and hot sulfuric acid

K_{oc}: NA

Specific gravity: 8.65 at 25°C

Vapor density: NA

Vapor pressure: NA

Viscosity: NA

NA = not available
(References: ATSDR, 1989; NIOSH, 1985; Sax and Lewis, 1989; EPA, 1986; HSDB, 1990.)

COMPOSITION: Cadmium is an element occurring naturally in the earth's crust and some cadmium has been found in all natural materials that have been analyzed (Friberg et. al., 1986). Cadmium is generally found combined with zinc, copper, lead ores, and phosphate deposits. Thus phosphate fertilization may be an important route of soil contamination.

USES: Metal plating, pigments, batteries, and plastic stabilizers, pesticides, alloys, chemical reagents, and/or intermediates (ATSDR, 1989).

ACUTE TOXICITY: It is a poison by ingestion, inhalation, and injection by intraperitoneal, subcutaneous, intramuscular, and intravenous routes. It is also an experimental carcinogen, tumorigen, and teratogen.

Ingestion: Acute toxicity may result from ingestion of relatively high concentrations of cadmium, as may occur in contaminated beverages or food. It is a powerful irritant of the gastrointestinal tract. Acute symptoms of oral exposure include nausea, vomiting, salivation, abdominal pain, cramps, and diarrhea. The concentration of cadmium in water that induces vomiting is about 15 mg/l, and a dose of 3 mg is considered to be an emetic threshold (ATSDR, 1989). Norberg and Nishiyama (1972) relates an instance in which nausea, vomiting, and abdominal pain occurred from consumption of drinks containing approximately 16 mg/l of cadmium.

Ingestion of several milligrams of cadmium per kilogram body weight of mice causes acute testicular necrosis followed by Leydig cell tumors (Piscator, 1981).

Inhalation: Acute pulmonary exposure of humans or experimental animals to cadmium in air may lead to marked bronchial and pulmonary irritation, but these effects are highly unlikely to occur outside the industrial environment (ATSDR, 1989). Brief high level exposure (>1 mg/m^3) to cadmium may result in pneumonitis, delayed pulmonary edema and death (EPA, 1985).

Eye contact: In contrast to many of the other metals, there is a noticeable lack of reported effects on the eyes even with exposures to dusts or fumes. As a result, eye contact with cadmium compounds is probably only mildly irritating and not likely to be severe enough to prevent exposures which may cause serious systemic toxicity (Grant, 1986).

Cadmium metal, when implanted into the eye of an experimental animal, produced a severe purulent intraocular inflammation and cataract (Grant, 1986).

Skin contact: Small quantities of cadmium may be absorbed through the skin, but dermal absorption is not normally a significant fraction of total cadmium absorption. Cadmium compounds have not been observed to cause significant health effects, acute or chronic, when exposure is by the dermal route (ATSDR, 1989).

CHRONIC TOXICITY:

Ingestion: Cadmium may be leached from food utensils and storage containers, resulting in nausea, vomiting, and diarrhea. Gastrointestinal symptoms have been associated with ingestion of >0.05 mg/kg in adults (EPA, 1985).

During World War II, Japanese rivers were polluted with wastes from metal smelting and refining operations. Ingestion of rice grown in water contaminated with cadmium, zinc, and copper produced a disease termed Itai-itai (ouch-ouch). Greater than 1 ppm cadmium was present in ingested rice (Friberg et al., 1974). Affecting predominantly post-menopausal women, the disease was characterized by severe back pain associated with demineralization of bone. Although community residents were exposed to a variety of pollutants, cadmium exposure is felt to have played the primary role in Itai-itai disease (EPA, 1985).

In a follow up study on 95 patients diagnosed as having Itai-itai disease, a 20 years mortality analysis demonstrated that cadmium exposure and renal damage significantly reduced the life span of patients as compared with matched controls (Nakagawa et al., 1991).

The most severe cases of oral cadmium toxicity have been associated with ingestion of foods or fluids which have been stored in cadmium-plated containers. Chronic oral exposure to cadmium has been suspected of causing a gastrointestinal malabsorption syndrome

referred to as cadmium enteropathy (ATSDR, 1989).

Inhalation: Chronic, low level exposure to cadmium (0.02-0.1 mg/m^3) in occupational settings is associated with the development of emphysema, loss of smell (anosmia), anemia, and yellow discoloration of the teeth. Renal insufficiency is a hallmark of chronic cadmium toxicity. Due to impaired renal tubular function, low molecular weight proteins (e.g., beta 2 macroglobulin) are not reabsorbed, yielding proteinuria. As the disease progresses, other essential components are also lost in the urine (glucose, other amino acids, calcium). Demineralization of bone secondary to renal disease may occur, with resultant bone pain.

Cadmium is present in cigarette smoke (1-2 mg per cigarette) and smokers add constantly to cadmium body burdens. The role of cadmium in the development of smoking-related chronic respiratory disease has not been clarified (EPA, 1985).

Chronic exposure to cadmium impairs lung function and causes dyspnea in workers. Emphysema has been reported in workers and rabbits exposed to cadmium after long-term exposure to levels of 0.1 mg/m^3 (ATSDR, 1989).

The principal long-term effects of low-level air exposure to cadmium are chronic obstructive pulmonary disease, emphysema and chronic renal tubular disease (Nomiyama, 1980).

Eye contact: See *ACUTE TOXICITY: Eye contact.*

Skin contact: See ACUTE TOXICITY: Skin contact.

SENSITIZATION: No reports of sensitization reactions to cadmium were located in the open literature.

TARGET ORGAN EFFECTS: Exposure to cadmium is associated with injury to a number of tissues or organs in both animals and humans, including the kidney, liver, cardiovascular system, skeleton, and immune system. At low-level chronic exposure, conditions most often experienced by humans, the kidney is generally recognized to be the critical organ. The renal cortex is the site of the highest cadmium concentration reached in the body. Parenteral injection of cadmium has been observed to cause severe acute pathological changes in the gonads of animals, especially males. Very large oral doses (100 mg/kg or higher) have also been reported to cause acute testicular damage similar to that following injection (ATSDR, 1989).

Besides the kidney, the next highest tissue levels of cadmium are found in the liver after both acute and chronic exposure. Painful bone disorders, including osteomalacia, osteoporosis, and spontaneous bone fracture, have been observed in some humans chronically exposed to high levels of cadmium (ATSDR, 1989).

ABSORPTION-METABOLISM AND EXCRETION: Variables which affect absorption include age, with neonates absorbing more than adults, and sex, with females absorbing more than males (ATSDR, 1989).

Cadmium is absorbed poorly from the gastrointestinal tract. A portion of an oral dose may be trapped in the intestinal mucosa without crossing into the blood or lymph. Absorption of cadmium from food is generally less than from milk, which in turn is less than from water. Cadmium is not known to undergo any direct metabolic conversions *in vivo* such as oxidation reduction, or alkylation. Cadmium does interact with the protein metallothionein (MT). Increased levels of MT increase intestinal trapping in the mucosa, decrease intestinal absorption, and increase accumulation of cadmium in MT-rich tissues such as the liver and kidney (ATSDR, 1989).

Cadmium is absorbed moderately well from the lungs. The major site of cadmium absorption is the alveoli. Small particles (0.1 μm) tend to penetrate the alveoli while larger particles (10 μm) tend to be deposited in the upper airway. Particle size and alveolar deposition are key determinants of cadmium absorption in the lung. Calculations based on an increased body burden in smokers compared to that in nonsmokers suggests that respiratory absorption in man is probably about 30 to 60% (ATSDR, 1989).

Differences in individual sensitivity to cadmium have not been systematically studied. The following factors may be important in determining above-average sensitivity: (1) the effect of cadmium on the kidney may be magnified by renal disease or other etiology, (2) genetic differences in the induction of metallothionein in response to cadmium exposure, (3) cadmium absorption from the gastrointestinal tract may increase due to dietary deficiencies in metal ions and/or protein, and (4) age, with the possibility of neonates or young children having higher gastrointestinal absorption rates than adults (ATSDR, 1989).

The principal excretory route for cadmium is urine. Excretion is low and reduces only a small

fraction of the total body burden. Because excretion is so slow, half-lives of cadmium in the body are correspondingly long (17 to 38 years), and cadmium accumulation over time is marked. Cadmium not absorbed from the gastrointestinal tract is excreted in feces (ATSDR, 1989).

GENOTOXICITY: Available data on cadmium compounds tested for mutagenic activity in a number of assay systems suggest that cadmium is mutagenic in mammalian cell culture assay systems. Cadmium has been demonstrated to be mutagenic both in the mouse lymphoma assay and in the Chinese hamster cell assay. Conflicting results for chromosomal aberration studies have been reported in human lymphocytes from exposed workers and in human and animal cell lines treated with cadmium *in vitro* (ATSDR, 1989).

IMMUNOTOXICITY: Exon and Koller (1986) have reviewed the findings on cadmium effects on the immune system. Koller et al. (1975) observed decreased levels of spleen plaque-forming cells in mice exposed to 0.6 mg/kg/day for 10 weeks. A dose-dependent suppression of the humoral immune response in mice exposed at 5 to 50 mg/l of cadmium for 3 weeks in drinking water was reported by Blakley (1985). Very low renal cadmium concentrations (0.3 µg/g to 0.6 µg/g) were associated with these functional alterations in the immune system. These concentrations are considerably lower than the suggested critical level of cadmium in the renal cortex in chronically ,exposed humans. While many of these findings are contradictory, it is relatively clear that low doses of cadmium can alter the immune response in experimentally dosed animals. Little evidence exists for suppression of the immune response in chronically exposed human populations (ATSDR, 1989).

NEUROTOXICITY: Parenteral administration of high doses of cadmium can cause lesions in the nervous system of experimental animals, but, evidence for neurologic effects following long-term oral or inhalation exposure of animals or man is limited. Oral cadmium administration in the rat increased passive avoidance behavior. Neurobehavioral changes have also been observed in rats following exposure to cadmium in utero (ATSDR, 1989). A 4.0 mg/kg dose of cadmium injected into adult rats did not produce any morphological changes but did cause lesions in the corpus callosum, caudate putamen, and cerebellum when injected into 4-day-old rats. It is suggested that the sensitivity of the newbom rats might result from an immature blood-brain barrier (ATSDR, 1989).

REPRODUCTIVE TOXICITY: In animal experiments, high parental doses of cadmium may cause severe injury to the gonads, especially in males, frequently resulting in reduced fertility or complete sterility.

Most studies involving oral exposure do not reveal significant effects on reproductive function. Reproductive effects of cadmium have not been reported in humans (ATSDR, 1989).

CARCINOGENICITY: Various epidemiological studies have been conducted to determine the relationship between inhalation exposure to cadmium and increased risk of cancer. Elinder et al. (1985) studied workers who had been exposed to cadmium for at least one year between 1940 and 1980 in a Swedish cadmium-nickel battery factory. Levels of cadmium oxide dust in air were estimated at 1 mg/m^3 before 1947, 0.3 mg/m^3 in 1947 to 1962, 0.05 mg/m^3 in 1962 to 1974, and 0.02 mg/m^3 since 1975. The standard mortality ratio (SMR) for both prostatic cancer and lung cancer tended to increase with estimated dose and duration of exposure but did not reach statistical significance. It was concluded that long-term high-level exposure to cadmium is likely associated with increased risk of lung cancer and there is insufficient evidence that cadmium exposure increases the risk for prostatic cancer in humans (ATSDR, 1989).

In contrast, Ades and Kazantzis (1988) observed a statistically significant lung cancer excess (SMR=124.5) in a study of a cohort of 4,393 men employed at a zinc-lead-cadmium smelter. The trend was toward an increased incidence as a function of employment duration. However, this risk did not correlate with estimated cumulative cadmium exposure levels, and it was concluded that cadmium was not responsible for the observed excess lung cancer (ATSDR, 1989).

There are presently no data to suggest that oral exposure of humans to cadmium is associated with increased risk of cancer. Mortality studies in areas of Japan and Europe, where oral cadmium exposure is elevated, have not revealed any observable increase in mortality from cancer, including prostatic cancer (ATSDR, 1989).

Studies conducted to date in animals have not shown cadmium to be carcinogenic by the oral route. In a study conducted by the Food and Drug Administration (FDA, 1977), groups of Charles River caesarean-originated, barrier-sustained Sprague-Dawley rats were fed diets containing 0, 0.6, 6, 30, 60, or 90 ppm cadmium for 103 weeks. No significant differences in the

tumor incidences of the control and treated groups were reported. Loeser (1980) also conducted a 2-year feeding study with Wistar rats. There were no significant differences in the incidence of tumors in Wistar rats fed diets containing 0, 1, 3, 10, or 50 ppm of cadmium chloride (ATSDR, 1989).

There is no evidence that dermal exposure to cadmium is carcinogenic in either animals or humans (ATSDR, 1989).

EPIDEMIOLOGY: There is limited but suggestive epidemiologic evidence that inhaled cadmium is associated with respiratory cancer. Thun et al. (1985) reported a dose-dependent increase in respiratory cancer among cadmium processing plant workers. The facility had produced cadmium compounds from 1925, but was formerly an arsenic smelter (1918-early 1920's). The 602 men were employed between 1940-1969 and following through 1978. Workers were grouped into one of three cumulative cadmium exposure categories corresponding to a 40-year TWA concentration of <40, 41-200, or >200 µg/m3 (<585, 585-2,920 and >2,920 mg-days/m3). The respiratory cancer SMRs (based on 20 deaths) for these categories were 53, 152, and 280, respectively. The overall respiratory cancer SMR was 265 ($p<0.05$). The dose-dependent trend remained after controlling for smoking and arsenic exposure.

The mortality experience of a subset of the workforce studied by Thun et al. (1985) has also been reported by Lemen et al. (1976) and Varner (1983). A dose-dependent effect for lung cancer was also observed in these earlier studies.

Other epidemiologic studies have noted increases in the risk of lung cancer among cadmium-exposed workers. Studies include: Kjellstrom et al. (1979), Holden (1980), Sorahan (1981), Anderson et al. (1982), Sorahan and Waterhouse (1983), and Armstrong and Kazantzis (1983). However, the findings in these studies are limited due to inadequacies in exposure classification or statistical power and analyses.

An increased risk of prostatic cancer has been reported among cadmium-exposed workers. Most reports have had major methodological problems, including a lack of appropriate control group, inadequate exposure assessments, concomitant exposures to known carcinogens, and limitations in analyses. Perhaps the best evidence supporting an association between cadmium exposure and prostatic cancer is found in Lemen et al. (1976). The study followed 292 cadmium production workers employed between 1940-1969 and followed through 1974. A 1973 industrial hygiene survey noted most cadmium 8-hour TWA exposures to be less than 1 mg/m^3; however, TWA concentrations up to 24 mg/m^3 were found. Four prostatic cancers were observed, 1.15 expected. Incorporating a 20 year latency period from first exposure, 4 prostatic cancers were observed vs. 0.88 expected, $p<0.01$. This Lemen study was later enlarged and updated by Varner (1983) and Thun et al. (1985). The finding of a statistically significant elevation in prostatic cancer was not replicated. The SMRs for prostatic cancer were slightly elevated, but did not reach statistical significance. Currently, there is insufficient evidence that cadmium exposure increases the risk of prostatic cancer in humans.

EPA has concluded that cadmium is a probable human carcinogen (Group B1) by the inhalation route based on limited carcinogenicity evidence from epidemiological studies and the finding of sufficient evidence of carcinogenicity in animals. Sufficient data to consider cadmium to be carcinogenic by the oral route does not exist (ATSDR, 1989).

In a study considered to supply only limited evidence of human carcinogenicity, an inhalation slope factor of 6.1 $(mg/kg/day)^{-1}$ has been derived for cadmium by inhalation based on limited human carcinogenic data (IRIS, 1990). A 2-fold excess risk of lung cancer was observed in cadmium smelter workers. The cohort consisted of 602 white males who had been employed in production work a minimum of 6 months during the years of 1940-1969. The population was followed to the end of 1978. Urine cadmium data that was available for 261 workers employed after 1960 suggest a highly exposed population.

ENVIRONMENTAL FATE: Cadmium enters the environment to a great extent from pollutant sources such as discarded metal-containing products, phosphate fertilizer, and fuel combustion. Cadmium particulates released into the atmosphere from the combustion of coal and petroleum products can be transported via wet or dry deposition providing an important route for environmental transfer. Typical atmospheric levels of cadmium are less than 3 $\mu g/m^3$ (ATSDR, 1989).

Cadmium is relatively mobile in the aqueous environment and the concentration of cadmium in water is usually inversely related to the pH value. Unlike mercury or arsenic, biological methylation or the formation of volatile compounds from cadmium does not occur in the aquatic environment. The concentration of cadmium in groundwater is relatively low due to several processes such as sorption by mineral

matter and clay, binding by humic substances, precipitation as cadmium sulfide and precipitation as the carbonate at relatively high alkalinities. Typical concentration in groundwater and drinking water are 1 µg/l or less. (ATSDR, 1989).

In soil, cadmium may be present as free compounds or as the Cd^{2+} ion dissolved in soil water. It may also be held to soil mineral or organic constituents by cation exchange, in which case, it is not readily leached by rainwater. The spread of cadmium to the environment may occur when soil particles containing bound cadmium are eroded into air or water. Cadmium levels in soils range between 0.01 to 0.7 ppm with an average value of 0.06 ppm (ATSDR, 1989).

Through both food and water, cadmium is strongly accumulated by all organisms. Cadmium concentrations in freshwater and marine organisms may accumulate hundreds to thousands of times higher than the concentrations found in water. Bioaccumulation of cadmium is strongly correlated with the cation exchange capacity of the soil in the organism's environment. Bioaccumulation of cadmium decreases as cation exchange increases. Bioconcentration in the aquatic environment is greatest for invertebrates like mollusks and crustaceans, followed by fish and aquatic plants. Typical concentrations of cadmium in the flesh of fish from non-polluted areas range from 1 to 100 µg/kg, and from 70 to 1200 µg/kg for shellfish. Higher levels of cadmium in beef and poultry have resulted from bioaccumulation of cadmium in feed crops, primarily a result of cadmium-containing fertilizers. In areas where soil levels are not elevated, typically levels of cadmium in leafy vegetables range from 12 to 450 µg/kg while values in grains or rice range from 5 to 130 µg/kg (ATSDR, 1989).

ENVIRONMENTAL TOXICITY: Cadmium can be incorporated into plants from the soil or water. In general, roots will contain a 10 fold higher concentration of cadmium than the rest of the plant. Cadmium uptake into fruit and nuts is generally less than the leafy portion of the plant (Stoeppler, 1984). Rice, wheat and tobacco are known to concentrate cadmium. Rice grown on fields irrigated with cadmium contaminated water is believed to have caused Itai-itai disease in Japan during the period 1967-1975. Fish and shellfish, particularly oysters, can accumulate fairly high levels of cadmium. In terrestrial animals, the accumulated cadmium is found primarily in the liver and kidneys (Friberg, et al., 1986).

REGULATORY STATUS: The WHO has recommended a drinking water guideline value for cadmium of 0.005 mg/l and a provisional tolerable weekly dietary intake of 0.4 to 0.5 mg per individual (~ 1 µg/kg/day) (ATSDR, 1989).

The EPA's ODW has promulgated an Interim Maximum Contaminant Level (MCL) for cadmium of 0.01 mg/l. Taking cost and feasibility into account, however, this MCL is based primarily on a consideration of health effects. ODW has proposed a Maximum Contaminant Level Goal (MCLG) of 0.005 mg/l for cadmium, based on the WHO and NAS guidelines. The EPA OWRS has set Ambient Water Quality Criteria to protect human health from potential adverse effects from the ingestion of water and/or edible aquatic organisms (fish and shellfish) from surface water sources. Based on toxic effects, the criterion for cadmium for ingestion of water and organisms is 0.01 mg/l. Since this value was identical to the existing drinking water standard, a criterion based on exposure from ingesting only organisms was not calculated (ATSDR, 1989).

Recommended and regulated worker exposure limit values are presented in Table 1.

Table 1. Worker Exposure Limit Values for Cadmium.

ACGIH TWA/TLV	0.05 mg/m^3 for dusts and salts
Ceiling	0.05 mg/m^3 for cadmium oxide fume
OSHA PEL	0.2 mg/m^3 for cadmium dust 0.1 mg/m^3 for cadmium oxide fume
Ceiling	0.6 mg/m^3 for cadmium dust 0.3 mg/m^3 for cadmium fume
IDLH	40 mg/m^3

(References: ACGIH, 1989; CFR, 1989)

dose-response: EPA has established a Reference Dose (RfD) of 0.0005 mg/kg/day for oral exposure to cadmium in water. This value is based on the estimate that if absorption is 4.5% and renal excretion is 0.01% per day, then ingestion of 0.35 mg/day would not exceed the critical renal concentration (200 µg/g) after 50 years of exposure. The proposed reference dose was derived by dividing by an uncertainty factor of 10 and taking 0.005 mg/kg/day as a NOAEL in humans. EPA has also established a separate Reference Dose of 0.001 mg/kg/day for cadmium in food because absorption of cadmium from the diet is approximately half of that from water (ATSDR, 1989).

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GLOSSARY

ACGIH: American Conference of Governmental Industrial Hygienists
EPA: Environmental Protection Agency
NAS: National Academy of Sciences
NIOSH: National Institute of Occupational Safety and Health
ODW: Office of Drinking Water
OSHA: Occupational Safety and Health Administration
OWRS: Office of Water Regulations and Standards
PEL: Permissible Exposure Limit
TLV: Threshold Limit Value
TWA: Time-Weighted Average (8 hours/day, 40 hours/week)
WHO: World Health Organization.

TOXICOLOGY PROFILE
CHLORINE

Synonyms: Bertholite, Molecular chlorine

CAS No: 7782-50-5

Boiling point: -34.6 °C

Color: Greenish-yellow, (diatomic gas)

DOT designation: Poison

Flammable limits: Nonflammable

Henry's law constant: NA

Melting point: -100.8 ° C

Molecular formula: Cl_2

Molecular weight: 70.906

Odor:
Threshold: 0.02-0.2 ppm
Recognition: 0.314 ppm

Characteristics: Pungent, irritating

pH: NA

Solubility:
water: 310 cm^3/100 cm^3 water at 10 °C
K_{ow}: NA
other: NA
K_{oc}: NA

Specific gravity: 1.5649 at -35 °C, 0.9949 atm; 1.4085 at 20 °C, 6.864 atm.

Vapor density: 2.5

Vapor pressure: 5 atm @ 10.3 °C

Viscosity: 0.385 cp at 0° C chlorine (liquid)

(References: HSDB, 1990; Sax and Lewis, 1987; Merck, 1989; Leonardos et al, 1969)

USES: Chlorine is used in the bleaching of fabrics, synthetic rubber and plastics. It is used for disinfecting drinking water. Chlorine is used in de-tinning and de-zincing of iron (Merck, 1989). Chlorine is an intermediate in the manufacture of chlorinated hydrocarbons, polyvinyl chloride, ethylene dichloride, hypochlorous acid, chlorobenzene, and chlorinated lime. It is used in processing of meat, fish, vegetables, and fruit (Sax and Lewis, 1987). Chlorine was used during World War I in combat as a poison gas (Clayton and Clayton, 1981).

ACUTE TOXICITY: Chlorine rapidly and spontaneously reacts with water in living tissues to form hypochlorous acid. Hypochlorous acid is capable of penetrating the cell membrane and forming N-chloro derivatives which are damaging to cellular integrity (Patton et al., 1972).

Chlorine is a gas at room temperature so inhalation is a primary route of exposure. Chlorine also reacts with water to form hydrochloric acid. The water solubility and reactivity of chlorine mean that the upper respiratory tract is affected first. Table 1 summarizes the acute toxicity of chlorine in several species.

Ingestion: Exposure by ingestion occurs when food or water treated with chlorine or chemicals which release chlorine are consumed (HSDB, 1990).

Ingestion of hydrochloric acid (hydrogen chloride in aqueous solution) results in severe burns of the mouth, esophagus, and stomach (Proctor et al., 1988). Pain, nausea, and vomiting may be followed by esophageal stricture.

Table 1. Summary of Acute Toxicity of Chlorine in Various Species

Species	Chlorine concentration mg/m^3 (ppm)	Exposure time (min.)	Effects
rat	2900 (1000)	53	50% mortality
mouse	2900 (1000)	28	50% mortality
dog	2200-2600	30	3-50% mortality
mouse	1100-2580	10	10-100% mortality
rat	850 (293)	60	50% mortality
mouse	368 (127)	30	50% mortality

(Reference: HSDB, 1990)

Inhalation: Chlorine and hydrogen chloride are potent irritants of the eyes and respiratory tract. Signs and symptoms include burning of the eyes and throat, cough, and a choking sensation. Should escape be prevented, profound respiratory effects may develop, including laryngeal spasm, tracheobronchitis, pneumonitis, and pulmonary edema.

Mild respiratory tract irritation may develop at 0.2-16 ppm, eye irritation at 7-8 ppm, and cough at 30 ppm. A few breaths at 1,000 ppm have resulted in fatal respiratory effects (Zenz, 1988). Persistent decrements in pulmonary function testing have been found among workers accidentally exposed to high concentrations of chlorine (Zenz, 1988).

In humans, moderate irritation of the upper respiratory track occurs at exposures of 5 to 15 ppm. At 30 ppm, exposures cough and chest pains result. Toxic pneumonitis and pulmonary edema occur at exposures of 430 ppm. Concentrations of 430 ppm are also lethal within 30 minutes. Concentrations of 1000 ppm are lethal within a few minutes (Ellenhorn and Barceloux, 1988).

The symptoms following the high acute exposures experienced by World War I soldiers could be divided into two phases. In the first phase which lasted 36 hours after exposure, the symptoms included a burning sensation in the throat, coughing, dyspnea, and aphonia. Death, when it occurred in this phase, was from pulmonary edema. In the second phase the pulmonary edema subsided but severe bronchitis developed. This was accompanied by headaches, nausea, vomiting, weakness, and diarrhea. The lungs of soldiers that died within

48 hours were characterized by gross swelling and a purplish red color. Mixed atelectasis and emphysematous patches with sticky membranous exudate on the trachea and bronchial mucosa were found (Gerchik, 1939)

Delayed effects which were found in chlorine-exposed soldiers included bronchopneumonia, lobar pneumonia, purulent pleurisy, and tubercular meningitis (Gilchrist and Matz, 1933).

Kaufman and Burkons (1971) studied 27 persons exposed by inhalation to chlorine from a leaking storage tank. There was no measurement of exposure levels. Two of the adults died from severe hemorrhagic pulmonary edema. Rales, dyspnea, and cyanosis were seen in the most heavily exposed victims. Subnormal vital capacity was observed in three patients initially. Reduction in forced expiratory volume was found in four patients. Residual volume was above normal levels in the most heavily exposed patients. In 30 to 90 days these abnormalities were less evident.

Leube and Kreiter (1971) studied 90 people acutely exposed to chlorine at a factory site. No estimate of exposure levels was available. Dyspnea was reported in 75% of the exposed patients, headaches in 66%, retrosternal pain in 47%, nausea in 44%, vertigo in 33% and vomiting in 11%. Ten of the exposed patients had signs of pulmonary edema when examined shortly after exposure. Two hours after exposure leukocytosis with white cells counts over 10,000 per cm^3 was found in 60 of the 68 patients tested. The following day only 6 patients had white cell counts over 10,000 cm^3. Elevated serum glutamic oxalic transaminase (SGOT) was found in 10 patients. The serum glutamic pyruvate transaminase (SGPT) was elevated in 27 of the patients. Liver biopsies were performed on two of patients. One patient had some individual swollen liver epithelia and a nuclear perturbation. No complications developed in any of the patients.

Numerous acute exposure animal studies have been performed with chlorine. The toxic effects of chlorine exposure which have been found in animal studies are similar to those found in humans and occur at similar doses (Weedon and Hartzell, 1940; WHO, 1982).

Mice which were exposed to 138 ppm which survived showed marked emphysema associated with an organizing exudate in the bronchioles, leading to bronchiolitis obliterans (Winternitz et al., 1920).

In another study dogs were exposed by inhalation to chlorine concentrations of 145-5,800 mg/m^3 for 30 minutes. The dogs that inhaled chlorine at the high range of exposure exhibited respiratory arrest and bronchoconstriction. The respiratory rate increased from 20 per minute to 35 per minute during the first hour after exposure. The pulse rate showed a decline following exposure but then increased to twice normal by ten hours after exposure. These respiratory and cardiovascular changes were accompanied by pulmonary edema. The dogs exhibited general excitement marked by restlessness, barking, urination, and defecation. Irritation of the eyes, sneezing, excess salivation, and vomiting were also observed. The progression of the pulmonary edema was marked by frothing at the mouth, labored respiration and finally death by asphyxiation. The pathological examination of these dogs revealed that necrosis of the epithelial lining of the respiratory tract had occurred. The destruction of the epithelial lining apparently allowed penetration of pathogenic organisms and a high incidence of pneumonia was found in dogs which survived the initial period after exposure. Chronic bronchitis, obliterative or organizing bronchiolitis, and fibrosis were seen in dogs dying or killed up to 6 months after chlorine exposure (Winternitz, 1920).
Due to the high water solubility of the gas, the upper lung is initially the more vulnerable target for chlorine toxicity.

Skin contact: Intense perspiration may be found following chlorine dermal exposure. Dermal exposure may cause pain, irritation, and erythema. Partial or total thickness burns of the skin may also result (Haddad and Winchester, 1983).

Skin exposure to chlorine gas, hydrogen chloride gas, or concentrated solutions of hydrochloric acid produces skin burns, with inflammation and vesicle formation. Exposure to weak solutions of hydrochloric acid may produce irritant contact dermatitis (Vernot et al., 1977; Proctor et al. 1988).

Eye contact: Chlorine is highly irritating to the eyes with exposures above 1 ppm (Clayton and Clayton, 1981).

CHRONIC TOXICITY:

Ingestion: Rats were given highly chlorinated drinking water (100 mg/l) for their entire lifetime for seven consecutive generations (Druckrey, 1968). No adverse effects on fertility, life span, growth pattern, hematology or histology were found. No increase in the incidence of malignant tumors was observed in the chlorine-treated rats.

No adverse effects were found in mice given drinking water with 200 mg/l chlorine for 33 days, or 100 mg/l for 50 days (Blabaum and Nichols, 1958). Only a limited number of parameters were monitored in this study.

Inhalation: An inhalation study was conducted with rats exposed to 0, 2.9, 8.7, or 26 mg/m^3 (0, 1,3 or 9 ppm) for 6 hours a day, 5 days a week for 6 weeks (Barrow et al., 1979). An increase in mortality occurred in the female rats which were exposed to 26 mg/m^3 (9 ppm). Body weight gain was reduced in the females exposed to 2.9, 8.7, and 26 mg/m^3, and in the males exposed to 8.7, and 26 mg/m^3. Both male and female rats showed clinical signs of ocular and upper respiratory tract irritation. These signs included lachrymation, hyperaemia of the conjunctiva, and nasal discharge. The rats exposed to 2.9 mg/m^3 showed occasional slight indications of irritation. All the chlorine-exposed groups had urinary staining of perineal fur. The urinary specific gravity was increased in all chlorine treated groups with exception of the low dose males.

Pathological examination of the rats exposed to 26 mg/m^3 chlorine levels revealed inflammation of the upper and lower respiratory tact. Focal to multifocal mucopurulent inflammation of the nasal turbinates and necrotic erosions of the mucosal epithelium were found. Epithelial hyperplasia and inflammation of the trachea and bronchiolar areas were observed. Inflammation and hypertrophy of the respiratory bronchioles and alveolar ducts was observed. Increased numbers of alveolar macrophage and isolated areas of atelectasis were found.

Slight degenerative changes were found in the renal tubules of the kidneys of the rats exposed to 26 mg/m^3. Elevated levels of blood urea nitrogen were found in these animals. Slight degenerative changes were found in the hepatocytes of the livers exposed to 8.7 and 26 mg/m^3. Elevated levels in serum levels of alkaline phosphatase, glutamic pyruvate transaminase (SGPT), and glutamic pyruvic transaminase (SGPT) were found in these animals (Schlagbauer and Henschler, 1967).

Skin contact: See skin contact under ACUTE TOXICITY.

Eye contact: Rats exposed to 8.6 and 26 mg/m^3 five days a week, for 6 hours a day for six weeks had signs of eye irritation such as hyperaemia of the conjunctiva and lacrimation (Barrow et al., 1979). The rats exposed to 2.9 mg/m^3 showed occasional signs of such irritation.

SENSITIZATION: No reports of sensitization from chlorine were located.

TARGET ORGAN EFFECTS: The mechanism of chlorine damage is thought to be mediated by the formation of hydrochloric acid (HCl), and hypochlorous acid (HOCl) from the reaction of chlorine with water. Hypochlorous acid is capable of penetrating the cell membrane and forming N-chloro derivatives which damage cellular integrity. This may be the cause of the pulmonary edema seen following high exposures. Hypochlorous acid also reacts with sulfhydryl groups in cysteine. Hypochlorous acid has been shown to inhibit various enzymes (WHO, 1982).

The primary target organ in exposures to gaseous chlorine is the lung. The water solubility and reactivity of gaseous chlorine cause the upper respiratory tract to be affected first. With increasing concentrations the lower respiratory tract becomes affected. Pulmonary edema can result from severe exposures. With both animal and human exposures, destruction of the epithelial lining of the trachea has been observed. The removal of mucociliary defense mechanism of the lung allows pneumonia and other infections to occur in survivors. Chronic bronchitis, obliterative or organizing bronchiolitis, and fibrosis have been seen in dogs which survived high acute exposures.

Inhalation of chlorine by anesthetized dogs and cats caused temporary cardiac arrest (Schultz, 1919). Severing the vagus abolished the effect. In humans, tachycardia followed by bradycardia may result from chlorine exposure. Hypertension may be found initially followed by hypotension. Cardiovascular collapse may result from severe exposure (HSDB, 1990).

Nephritis has been reported as a delayed effect following acute exposures in World War I (Clayton and Clayton, 1981) and slight degenerative changes have been reported in the kidney tubules of chronically exposed rats. Slight liver damage has been reported in biopsies of humans acutely exposed to high doses and in necropsies of chronically exposed rats. It is unclear whether the effects on the kidney and liver are secondary or primary effects.

ABSORPTION-METABOLISM-EXCRETION: As previously mentioned chlorine is readily soluble in water and thus tends to be absorbed first in the upper respiratory tract when inhaled. No quantitative estimates of the amount of an inhaled dose absorbed were found in the literature. The portion of the chlorine which reacted to form hydrogen chloride would enter the chloride ion pool and be largely

excreted in the urine. Adducts formed from the reaction of hypochlorous acids with macromolecules would presumably be eliminated through macromolecular turnover and subsequent xenobiotic metabolism through one of several pathways.

IMMUNOTOXICITY: Long term exposure to chlorine caused tuberculosis to develop sooner in guinea pigs injected with human tuberculosis. The guinea pigs were exposed to 5 mg/m^3 for 5 hours a day for 47 days prior to after the injection. The survival of the guinea pigs exposed before injection was lower than the controls or the guinea pigs exposed after injection (Arloing et al., 1940)

REPRODUCTIVE TOXICITY: In the multi-generational study conducted by Druckrey (1968) rats were exposed to 100 mg/liter chlorine in drinking water for their entire lifetime for seven generations. No adverse effects on fertility were found.

NEUROTOXICITY: Headache was reported to be a symptom accompanying respiratory irritation in people exposed in a storage tank accident. Autopsies on three person killed revealed "cerbrae purple" in the white matter of the brain (Baader, 1952). Headache is reported to result following exposures of 3 to 6 ppm (Clayton and Clayton, 1981).

GENOTOXICITY: Chlorine kill bacteria at concentrations of less than 1 mg/L (WHO, 1982). The bactericidal properties of the compound limit the usefulness of the Ames test in assessing the mutagenicity of chlorine.

Mickey and Holden (1971) reported that chlorine caused chromatid and chromosome breaks, translocations, dicentric chromosomes, and gaps in a human lymphocyte culture system. The concentrations of chlorine used were 2-20 times those found in drinking water.

CARCINOGENICITY: Rats were exposed to chlorine in drinking water 100 mg/l over the entire life span for 7 consecutive generations. The incidence of malignant tumors was the same in the control and experimental groups (Druckrey, 1968).

EPIDEMIOLOGY: An epidemiological study was performed on 332 workers exposed to chlorine. The time weighted average exposure of the workers ranged from 0.006 to 1.42 ppm with an average exposure time of 10.9 years. No evidence of permanent lung damage was found from exposure to chlorine at these levels. The parameters investigated include ventilatory capacity, maximal ventilatory volume and forced expiratory volume at 3 seconds. No increased incidence of tooth decay was found. No increase in the incidence of abnormal chest X-rays, or abnormal EKG was found in the exposed workers (Patil et al., 1970).

Several other less than conclusive epidemiological studies have been conducted but lack estimates of exposure and suffer from other methodological problems (NIOSH, 1976).

ENVIRONMENTAL FATE: Free chlorine is a strong oxidizing agent which readily reacts with inorganic compounds present in environmental waters. Free chlorine also reacts more slowly with organic compounds. Because of this reactivity, chlorine as the diatomic gas does not persist in the environment. It is rapidly photolyzed in the presence of sunlight. Some products of the reactions with organic compounds such as chloroform are of human health concern because they are carcinogenic and formed through chlorination of drinking water (HSDB, 1990).

ENVIRONMENTAL TOXICITY: Although chlorine is highly toxic to all organisms there is no potential for bioconcentration or bioaccumulation (HSDB, 1990). The following table gives LC_{50} for various aquatic organisms.

Table 2 Acute Lethal Concentration Values for Chlorine in Fish

Organism	LC_{50}(mg/l)	Exposure Conditions
Daphnia magna (water flea)	0.097 mg/l/30 min.	Not specified
Daphnia magna	0.063 mg/l/60 min.	Not specified
Gambusia affinis	1.59 mg/l/30 Min.	Not specified
Gambusia affinis	0.84 mg/l/60 min.	Not specified

Daphnia magna	0.017 mg/l/46 hr.	Not specified
Oncorhynchus kisutch	208 µg/l/1 hr.	Not specified
Daphnia pulex	0.49 mg/l/96 hr.	Not specified
Yellow perch	0.88 mg/l/1 hr.	Not specified
Micropterus salmoides (large mouth bass)	0.74 mg/l/1 hr.	
Lepomis macrochirus (bluegill sunfish)	0.44 mg/l/96 hr.	intermittent chlorination at 15 °C
Ictalurus punctatus (channel catfish) (fingerling)	0.07 mg/l/96 hr.	Not specified toxic effect: gill sodium uptake drastically impaired.
Emerald shiner (yearling)	0.23 mg/l/30 min.	test performed using Lake Superior water at 25 °C
Emerald shiner (adult)	0.28 mg/l/30 min.	test performed using Lake Superior water at 25 °C

(Reference: WHO, 1982)

REGULATORY STATUS: The ambient water quality criterion for chlorine in order to protect human health is recommended to be 10.0 mg/l (EPA, 1981)

For the protection of freshwater aquatic organism the EPA has proposed that the 4 day average concentration of total residual chlorine not exceed 11 µg/l more than once every 3 years on the average. As a short term limit the EPA has proposed that the 1 hour concentration not exceed 19 µg/l more than once every 3 years on the average (EPA, 1986).

The standard which EPA has proposed for salt water is that the 4 day average concentration of total residual chlorine not exceed 7.5 µg/l more than once every three years. The short term limit is that the 1 hour concentration not exceed 13 µg/l more than once every three years on the average (EPA, 1986)

Table 3. Worker Exposure Limit Values

ACGIH TWA:	0.5 ppm (1.5 mg/m^3)
STEL:	1 ppm, (3.0 mg/m^3)
Ceiling:	none
OSHA - PEL:	0.5 ppm (1.5 mg/m^3)
STEL:	1 ppm, (3.0 mg/m^3)
IDLH:	30 ppm
NIOSH REL:	0.5 ppm (115 mg/m^3) 15 min. ceiling
MSHA:	NA

NA = Not Available
(Reference: NIOSH, 1976;1985)

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TOXICOLOGY PROFILE
CHROMIUM AND HEXAVALENT CHROMIUM

Synonyms: Chrome, chrom

CAS No: 7440-47-3 (chromium metal)

Boiling point: 2672 °C (metal)

Color: Steel grey (metal)

DOT designation: Oxidizer (chromic acid)

Flammable limits: The metal is non flammable and non combustible.

Flash point: N/A

Henry's law constant: NA

Melting point: 1857 °C (metal)

Molecular formula: Cr

Molecular weight: 51.996 (metal)

Odor: odorless

pH: NA

Solubility:

water: The metal is insoluble. Hexavalent chromium (Cr^{6+}) compounds, such as chromium trioxide (chromic acid), calcium chromate, potassium chromate and dichromate, sodium chromate and dichromate are very soluble: whereas lead chromate and zinc chromate are virtually insoluble.

K_{ow}: NA
other: NA
K_{oc}: NA

Specific gravity: 7.20 (metal)
2.70 (chromic acid)

Vapor density: NA

Vapor pressure: 1 torr @ 1616°C

Viscosity: NA

NA = not applicable
N/A = not available
(References: OSHA, 1989; ACGIH, 1989; EPA, 1989)

COMPOSITION: Chromium is an element that exists in four valence states with the +3 and +6 states predominating. Cr^{6+} reacts with many other metals and metalloids to form chromates and dichromates.

USES: The three major industries that use chromium are metallurgical, refractory, and chemical. In the metallurgical industry, chromium is used in making steels, alloy cast irons, nonferrous alloys and miscellaneous other materials. Refractory uses include producing chrome and chrome-magnesite, magnesite-chrombrick, and granular chromite. In the chemical industry, chromium is used primarily in pigments, metal finishing, and wood treatment. (ASTDR, 1987)

ACUTE TOXICITY

Ingestion: An oral dose of 2-5 grams of Cr^{6+} (as a chromate compound) can be fatal to humans (Friberg, 1986). Toxic symptoms in individuals ingesting at least 5 grams of chromate compounds included gastrointestinal bleeding, massive fluid loss, and death from cardiovascular shock. Kidney and liver damage developing 1-4 days after exposure have been reported in individuals ingesting 2 grams or less of a chromate compound.

For sodium chromate and dichromate, potassium dichromate, and ammonium dichromate, oral LD_{50}s in rats range from 51 to 57 mg/kg body weight (Gad et al., 1986). In terms of chromium, the LD_{50}s ranged from 16.7 to 22.5 mg/kg body weight. For chromium trioxide (chromic acid), Kobayashi (1976) reported LD_{50}s ranging from

135-177 mg/kg body weight in rats (70-92 mg/kg as Cr) and from 80-114 mg/kg in mice (42-59 mg/kg as Cr).

Toxic effects observed by Gad and coworkers included pulmonary congestion, gastrointestinal edema, and erosion and discoloration of the gastric mucosa. Kobayashi reported diarrhea, cyanosis, tail necrosis, and gastric ulcers in the animals dosed with chromium trioxide.

Inhalation : Acute death due to inhalation of Cr^{+6} compounds has not been reported in humans. At concentrations of 0.01-0.024 mg/m^3, aerosols of unspecified chromate compounds were reported to irritate the nose when inhaled for a short period of time by human subjects (Kuperman, 1964).

LC_{50}s for sodium chromate and dichromate, potassium dichromate, and ammonium dichromate ranged from 33-65 mg/m^3 (as Cr) in male and female rats (Gad, et al., 1986). Toxic symptoms included respiratory distress and irritation.

Eye contact: Contact with metallic particles can cause physical abrasion and irritation of the eyes. Chromate salts and chromic acid are severe irritants and can lead to corrosion and ulceration (Proctor et al., 1988).

Skin contact: Following application of Cr^{6+}-containing anti-scabies ointment to the skin, 12 patients died (EPA, 1984). Toxic signs before death included nausea, vomiting, shock, and coma. The sites of application became necrotic. Albumin and blood were found in the urine. Autopsies revealed tubular necrosis and hyperemia of the kidneys.

Dermal LD_{50}s reported by Gad and coworkers (1986) for sodium chromate and dichromate, potassium dichromate, and ammonium dichromate range from 397 to 677 mg/kg (as Cr) in rats. Necrosis developed at the site of application of exposed rats. The rats also exhibited diarrhea, hypoactivity, and dermal edema and inflammation.

CHRONIC TOXICITY

Ingestion: The chronic effects of Cr^{6+} compounds in laboratory animals have been investigated in several studies. No effects have been observed at the administered doses. MacKenzie and coworkers (1958) provided male and female rats potassium dichromate at concentrations up to 25 mg/liter in drinking water for 12 months and observed no effects on blood chemistry, body weight or gross and microscopic pathology. The results of that study were used to calculate the oral reference dose for hexavalent chromium. Anwar and coworkers (1961) observed no effects in two dogs provided water containing up to 11.2 mg/liter of Cr^{6+} for four years. The parameters examined included urinalysis and gross and microscopic examination of the major organs. Maruyama (1982) provided mice with water containing up to 100 mg/liter of Cr^{6+} for one year; no adverse effects were observed. The study included blood chemistry, hematology, body weight measurements and analysis of several organs for iron, copper and zinc. A decrease in iron content was found in the liver, spleen, testes, and all other organs analyzed.

Inhalation: Cr^{6+} is a respiratory irritant. Observed effects in humans include perforation or ulceration of the nasal septum, inflamed nasal mucosa, papillomas of the oral cavity and larynx, impaired lung function, chronic rhinitis, chronic bronchitis, emphysema, chronic lung inflammation, and chronic pharyngitis. The no-observed-adverse-effect-level (NOAEL) for respiratory effects appears to be 0.2-1.2 μg/m^3. (ATSDR, 1987)

Following exposure to dusts of mixed Cr^{6+} compounds at concentration of 3-4 mg/m^3 5 hours/day 4 days/week for 50 months, rabbits and rats developed nasal perforations and granulomata (Steffee and Maetjer, 1965). No effects on the kidney, liver, or spleen were observed. Necrosis, atrophy, and hyperplasia of the bronchial epithelium occurred in mice exposed to 13 mg/m^3 of calcium chromate dust 5 hours/day, 5 days/week for 6 months to a lifetime. Other effects were bronchiolization of alveoli, alveolar proteinosis, atrophy of the spleen and liver, and ulceration of the stomach and intestinal mucosa.

Eye contact: See ACUTE TOXICITY, Eye contact.

Skin contact: Cr^{6+} compounds are powerful skin irritants. At high concentrations, they can causes necrotic lesions in the skin. At low concentrations, they are sensitizers (NAS, 1974). Chromium hypersensitivity has also been observed in guinea pigs (ASTDR, 1987).

SENSITIZATION: Cr^{6+} is a potent sensitizer. According to EPA (1984), chromium may be linked to asthmatic attacks in workers in the chromium industry and can cause allergic contact dermatitis. Immune system effects have been observed in laboratory animals. Glaser and coworkers (1985) found an increase in the phagocytic activity of macrophages, total serum immunoglobulin content, and antibody response to

injected sheep red blood cells in rats exposed to sodium dichromate at concentrations up to 200 mg/m^3 for 22 hours/day. An increase in the number of macrophages was observed in rabbits exposed to 0.9 mg/m^3 of Cr^{6+} for 6 hours/day, 5 days/week for 4-6 weeks (Johansson, et al., 1986).

TARGET ORGAN EFFECTS: Cr^{6+} targets the skin, upper respiratory tract and lower respiratory tract. Effects include dermatitis, nasal mucosa, inflammation and ulceration, nasal septum perforation, chronic inflammation of the lungs, emphysema, chronic bronchitis and chronic pharyngitis.

ABSORPTION-METABOLISM AND EXCRETION: Donaldson and Barreras (1966), measuring fecal excretion of radioactive Cr^{6+} in human volunteers, found the absorption of Cr^{6+} to vary by the method of oral administration. When ingested, 11% of soluble Cr^{6+} is absorbed; 50% is absorbed when administered intraduodenally.

Donaldson and Barreras (1966) found that absorption of Cr^{6+} by rats is also affected by the site of entrance to the gastrointestinal tract. When injected intragastrically, about 2 percent is absorbed; when injected into the jejunum about 25 percent is absorbed. Fasting increases chromium absorption via the gastrointestinal tract (Ogawa, 1976) and young animals absorb more Cr^{6+} than older ones (Sullivan, et al., 1984).

Cr^{6+} is absorbed through human skin (Mali, 1963). In studies using guinea pigs, dermal absorption of Cr^{6+} increased as the concentration of Cr^{6+} in the aqueous solution of sodium chromate applied to the skin increased and peaked when the concentration of sodium chromate was between 0.261 and 0.398 M. Peak absorption rate was 690-725 $\mu g\ mole/hr/cm^2$.

Cr^{6+} is absorbed from the lungs, as evidenced by the appearance of chromium in the urine, serum and red blood cells following inhalation of chromium by workers (Gylseth, et al., 1977; Cavalleri and Minoia, 1985). No information was found regarding the percentage of chromium absorbed by humans from inhaled air. From the results of studies with laboratory animals by Visek and coworkers (1953) and Wiegand and coworkers (1984), it appears that 53 to 85 percent of the chromium in Cr^{6+} compounds is absorbed via the lungs.

Absorption of trivalent chromium is equal to or less than that of Cr^{6+} (EPA, 1984).

Following pulmonary or gastrointestinal absorption of chromium, chromium concentrates in the lungs, lymph nodes, kidney, and liver. Chromium also concentrates in the bladder and bone following pulmonary absorption (IARC, 1980) and in the spleen and heart following gastrointestinal absorption (Teraoka, 1981). Distribution after dermal absorption does not appear to have been studied.

In vitro studies indicate that Cr^{6+} is reduced to trivalent chromium, with a pentavalent chromium intermediate formed in the process (Kitagawa, et al., 1982; Levis et al., 1978; Jennette, 1982). Reduction of Cr^{6+} to the trivalent form appears to occur through reaction with glutathione. Wiegand and associates (1984) incubated human red blood cells with an excess of Cr^{6+} and found a large reduction in glutathione. Three molecules of glutathione were needed to reduce one molecule of Cr^{6+}. The reaction was accelerated at pH less than 5 and when initial glutathione levels approximated those in the liver and red blood cells. *In vivo* studies also indicate that Cr^{6+} is reduced to the trivalent form before excretion (ATSDR, 1987).

Chromium absorbed via inhalation or ingestion is excreted primarily in the urine and feces (ATSDR, 1987) Cr^{6+} is converted to trivalent chromium before excretion (Cavalleri and Minoia, 1985; Sayato et al., 1980).

IMMUNOTOXICITY: See CHRONIC TOXICITY, Sensitization.

NEUROTOXICITY: There is no evidence that Cr^{6+} is neurotoxic in humans. However, studies suggest that Cr^{6+} may affect the central nervous system in animals. Diaz-Mayan and coworkers (1986) reported that rats provided water containing 700 mg/liter of Cr^{6+} became hypoactive. In rabbits given 2 mg/kg Cr^{6+} intraperitoneally every day for three or six weeks, changes observed by Mathur and coworkers (1977) included neuronal degeneration of the cerebral cortex, marked chromolysis, nuclear changes in the neurons, and meningeal congestion.

REPRODUCTIVE TOXICITY:

Hexavalent and trivalent chromium compounds may affect reproduction. The results of studies in which chromium was administered orally or dermally are inconclusive; however, reduced survival of embryos fertilized by sperm from male mice given a single intraperitoneal injection of potassium dichromate at 20 mg/kg or 21 daily injections at 2.0 mg/kg has been found (Paschin, et al., 1982). A reduction in testicular succinic dehydrogenase and adenosine triphosphatase activity in rabbits injected with

potassium dichromate at 2 mg/kg/day for 3 or 6 weeks has also been noted.

GENOTOXICITY: Chromosome aberration and SCE tests of Cr^{6+} compounds with Escherischia coli, Salmonella typhimurium, yeast (S. pombe and S. cerevisiae), hamster cells, and other mammalian cells, including some from humans, gave positive results (ATSDR, 1987). Positive findings have also been reported for dominant lethal and chromosomal aberration tests in intact laboratory animals; however, other results have been negative. Although chromosomal aberrations have been seen in workers exposed to chromium, the results of different studies are not consistent; hence, no conclusion can be made regarding the genotoxicity of chromium in humans.

CARCINOGENICITY: An increase in respiratory cancer deaths has been consistently observed among chromate production workers (ATSDR, 1987). Mancuso (1975) followed 322 workers exposed to both hexavalent and trivalent chromium compounds. Exposure assessments for cohort members (first employed between 1931-1937) were based on industrial hygiene data gathered in 1949. A dose-related increase in lung cancer mortality was observed, with highest rates found among those exposed to greater than 4.0 mg/m^3/year chromium.

The Mancuso 1975 data are consistent with findings of studies of chromate production workers in Japan, Great Britain, West Germany, and the United States (EPA, 1984).

Two studies of chrome pigment workers exposed to hexavalent chromium have found an association between chromium exposure and lung cancer (Langard and Norseth 1975, Davies 1978 and 1979). Three studies have evaluated the mortality experience of chrome plating workers. Royle (1975) found an association between exposure and lung cancer, while two other studies were inconclusive (Silverstein et al. 1981 and Okubo and Tsuchiya, 1979).

Despite ample evidence that Cr^{6+} causes lung cancer in humans when inhaled, inhalation studies with laboratory animals have produced negative or inconclusive results. Nettesheim and coworkers (1971) exposed 136 male and female mice to 13 mg/m^3 of calcium chromate (+6) for 5 hours/day, 5 days/week for a lifetime. They reported that the incidence of lung adenomas and adenocarcinomas was significantly higher in the treated group than in controls, but did not indicate whether or not their conclusion was based on statistical analysis. According to ATSDR (1987), IARC (1980) reviewed the study and concluded there were no excess tumors. Glaser and coworkers (1986) exposed 20 male rats to an aerosol containing up to 0.1 mg/m^3 sodium dichromate (+6) for 22-23 hours/day, 7 days/week for 18 months. In the high level group there were two adenomas and one adenocarcinoma of the lung and one malignant tumor of the pharynx. Statistical analysis was not performed.

Baetjer and coworkers (1959) and Steffee and Baetjer (1965) did not observed any significant carcinogenic effects in mice, rats, rabbits, and guinea pigs exposed to a chromium dust mixture containing mostly Cr^{6+} 4 hours/day, 5 days/week for an extended time. In another study (Steinhoff et al., 1986), rats were given 0.25 mg/kg of sodium dichromate intratracheally five times/week or 1.25 mg/kg one time/week for a lifetime. No lung tumors developed in the group exposed five times/week, but a total of 20 tumors (8 malignant and 12 benign) developed in the group dosed once/week. The authors concluded that sodium dichromate is a weak carcinogen.

Several Cr^{6+} compounds were investigated by Levy and coworkers (1975, 1983, 1986) for carcinogenic activity in rats. The compounds were administered intrabronchially or implanted in the lower left bronchus. Groups receiving strontium chromate, zinc chromate, or calcium chromate had a higher incidence of cancer than controls. The authors concluded that only sparingly soluble Cr^{6+} compounds are carcinogenic, while insoluble and highly soluble ones are not.

The carcinogenicity of Cr^{6+} administered orally has been studied in laboratory animals (Schroeder et al., 1964 and 1965; Ivankovic and Preussmann, 1975). None of the studies resulted in a significant increase in tumor incidence. No information was found on the carcinogenicity of dermally-applied Cr^{6+} or Cr^{3+}.

EPIDEMIOLOGY: Cross-sectional studies of workers exposed to chromate dust and to chromic acid during chrome plating have revealed increases in upper respiratory tract irritant symptoms, nasal septum ulceration and perforation, reversible decreases in lung function parameters, and lower respiratory tract effects (tracheitis, bronchitis).

Cohort mortality studies of chromate production workers have consistently shown an increase in respiratory cancer deaths. Cr^{6+} compounds have been implicated as the likely carcinogens (ATSDR, 1987). These studies are reviewed under CARCINOGENICITY.

ENVIRONMENTAL FATE: Chromium is a relatively rare, naturally-occurring element. In the United States, the chromium content of soil and rocks ranges from 1 to 2,000 mg/kg and averages 54 mg/kg (Shacklette and Boerngen, 1984). Chromium concentrations in unpolluted U. S. surface water and groundwater range up to 0.084 and 0.05 mg/liter, respectively (EPA, 1987). Chromium levels in fresh vegetables, nuts, grains and cereals are generally less than 0.05 mg/kg (ATSDR, 1987). In non-urban areas, the level of chromium in air is usually below detection limits. In urban areas, however, chromium concentrations exceeding 0.01 $\mu g/m^3$ have been reported (EPA, 1980)

In nature, chromium exists primarily in the trivalent state. In Hudson County, New Jersey, where chromite ore was processed into various hexavalent and trivalent chromium products for 60 years, only 2.6% of the total average soil chromium concentration was in the form of hexavalent chromium (Paustenbach et al., 1991).

Cr^{6+} in the environment comes primarily from industrial sources. It is a potent oxidizer and readily oxidizes organic matter. In the process, it is reduced to Cr^{3+}. Acidic conditions also reduce Cr^{6+} to Cr^{3+}. Cr^{6+} is not sorbed to any significant degree by clays or other soils (Callahan et al., 1979). In the atmosphere, Cr^{6+} us reduced to Cr^{3+} by reaction with vanadium, ferrous ions, bisulfuric ions and trivalent arsenic. The oxidation of Cr^{3+} to Cr^{6+} may occur via reaction of Cr^{3+} with trivalent and tetravalent manganese (Seigneur, 1986). In natural waters, Cr^{6+} and Cr^{3+} are readily interconvertible. Cr^{6+} is reduced by ferrous ions, dissolved sulfides and certain organic compounds with sulfhydryl groups. Cr^{3+} is oxidized to Cr^{6+} rapidly by reaction with MnO_2 and more slowly by reaction with dissolved oxygen (Schroeder and Lee, 1975). Chromium bioaccumulates in aquatic organisms. Hexavalent chromium does not accumulate in fish muscle (Stokes, et al., 1977; Fromm and Stokes, 1962), but does accumulate in the tissues of molluscs (EPA, 1980).

ENVIRONMENTAL TOXICITY: No information was found regarding the toxicity of Cr^{6+} to vegetation, terrestrial invertebrates, mammals, or birds. Acute toxicity estimates (LC_{50}) for Cr^{6+} are presented in the following table (Table 1) for selected aquatic species.

Table 1. Lethal Ecotoxicity values for Several Organisms.

Species Name	LC_{50} (mg/l)
Rotifer *(Philodina sp.)*	3.1-15
Snail *(Physa sp.)*	17.3-40.6
Water flea *(Daphnia magna)*	6.4
Scud *(Gammarus sp.)*	0.067
Midge *(Tanytarsus sp.)*	59.9
Rainbow trout *(Salmo gairdnerii)*	69
Brook trout *(Salvelinus fontinalis)*	59
Goldfish *(Carassius auratus)*	37.5-249
Fathead minnow *(Pimephales promelas)*	17.6-66
Bluegill *(Lepomis machrochirus)*	110-213
Polychaete worms *(several species)*	2-8
Mysid shrimp *(Mysidopsis sp.)*	2-4.4
Blue crab *(Callinectes sapidus)*	89-98
Sanddab *(Citharichthys stigmaeus)*	30
Atlantic silverside *(Menidia menidia)*	12.4-20.1

(Reference: EPA, 1980.)

An Expert Panel concluded that soils contaminated with 75 ppm of hexavalent chromium and 1000 ppm total chromium did not pose a significant health hazard to neighboring residents or workers. In addition, the residents of New Jersey that were living near the abandoned chromite ore mining and processing sites were exposed to a risk level of less than 10^{-6} (Paustenbach et al., 1991).

REGULATORY STATUS: The following table lists the U.S. Federal Standards that were located in the literature.

Table 2. U.S. Federal Standards.

Occupational Exposure Limits

OSHA PEL:
None established
OSHA ceiling: 0.1 mg/m^3 (OSHA, 1989)
ACGIH TWA: 0.05 mg/m^3 (ACGIH, 1989)
ACGIH STEL: None established

Drinking water standard: 0.05 mg/liter (EPA, 1976)

Proposed standard: 0.1 mg/liter (EPA, 1989)

Ambient water quality criteria

Health: 0.05 mg/liter (EPA, 1986)

Freshwater organisms: 16 µg/l, 1-hour (EPA, 1986)
11 µg/l, 4-hour (EPA, 1986)

Saltwater organisms: 1.1 mg/l, 1-hour (EPA, 1986)
50 µg/l, 4-hour (EPA, 1986)

Reportable quantity: 1 lb, metal (EPA, 1985)
1000 lbs, chromates, acid (EPA, 1985)

Reference dose (mg/kg/day)

Oral: 0.005 (IRIS, Feb. 1990). Based on a NOAEL of 25 mg/l, converted to 2.4 mg/kg/day, and an uncertainty factor of 500.

Inhalation: Pending (IRIS, 1990)

Cancer Potency

Oral: Not applicable
Inhalation: $4.1 \times 10^{+1}$ (IRIS, 1990) Based on occupational exposure study by Mancuso (1975).

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COMPOSITION: The polychlorinated dibenzodioxins (CDDs) and dibenzofurans (CDFs) form a class of closely related compounds with different numbers of chlorine atoms attached to two benzene rings. In the 75 different dioxins, the two rings are joined by two oxygen bridges, while in the 135 different furans, one oxygen bridge complements a direct carbon-carbon bond (see Figure 1). The individual members of the class are called congeners. The toxicity of the class is related to the coplanarity of the base dioxin or furan, and chlorine configurations that maintain coplanarity are generally more toxic. Table 1 lists the CDDs and CDFs that are generally considered most toxic, and includes their toxicity relative to that of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (see toxicity section).

Synonyms: Varies depending on specific congener. For example,

2,3,7,8-tetrachlorodibenzo-*p*-dioxin (CAS No. 1746-01-6) is also known as
2,3,7,8-tetrachlorodibenzodioxin;
2,3,7,8-tetrachlorodibenzo[b,e](1,4)-dioxin;
2,3,7,8-tetrachlorodibenzo-1,4-dioxin;
2,3,7,8-TCDD;
TCDD;
TCDBD;
Dioxin;
Dioxine;
Tetradioxin

2,3,7,8-tetrachlorodibenzofuran (CAS No. 51207-31-9) is also known as
2,3,7,8-TCDF;
TCDF

Table 1. International Toxicity Equivalence Factors for Chlorinated Dioxins and Furans

Chemical Name	Abbreviation	CAS Number	I-TEF[a]
Dioxins			
2,3,7,8-tetrachlorodibenzo-*p*-dioxin	2,3,7,8-TCDD	1746-01-6	1.000
1,2,3,7,8-pentachlorodibenzo-*p*-dioxin	1,2,3,7,8-PeCDD	40321-76-4	0.500
1,2,3,4,7,8-hexachlorodibenzo-*p*-dioxin	1,2,3,4,7,8-HxCDD	–	0.100
1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin	1,2,3,6,7,8-HxCDD	–	0.100
1,2,3,7,8,9-hexachlorodibenzo-*p*-dioxin	1,2,3,7,8,9-HxCDD	19408-74-3	0.100
1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin	1,2,3,4,6,7,8-HpCDD	–	0.010
Octachlorodibenzo-*p*-dioxin	OCDD	3268-87-9	0.001
Furans			
2,3,7,8-tetrachlorodibenzofuran	2,3,7,8-TCDF	51207-31-9	0.100
1,2,3,7,8-pentachlorodibenzofuran	1,2,3,7,8-PeCDF	57117-41-6	0.050
2,3,4,7,8-pentachlorodibenzofuran	2,3,4,7,8-PeCDF	57117-31-4	0.500
1,2,3,4,7,8-hexachlorodibenzofuran	1,2,3,4,7,8-HxCDF	70648-26-9	0.100
1,2,3,6,7,8-hexachlorodibenzofuran	1,2,3,6,7,8-HxCDF	–	0.100
1,2,3,7,8,9-hexachlorodibenzofuran	1,2,3,7,8,9-HxCDF	–	0.100
1,2,3,4,6,7,8-heptachlorodibenzofuran	1,2,3,4,6,7,8-HpCDF	–	0.010
1,2,3,4,7,8,9-heptachlorodibenzofuran	1,2,3,4,7,8,9-HpCDF	–	0.010
Octachlorodibenzofuran	OCDF	–	0.001

[a] International Toxicity Equivalency Factor, the ratio of the toxic potency of the congener to that for 2,3,7,8-TCDD. TEFs for all other congeners are set to zero, unless the congener is the same as one of the above through a reflection or rotation. The U.S. Environmental Protection Agency, which had earlier proposed somewhat different TEFs, adopted the I-TEFs in 1989. Source: EPA 1989.

The monochloro dibenzodioxins and dibenzofurans are sometimes not included as polychlorinated. They are designated by the prefix M: 2-MCDD is 2-chlorodibenzo-*p*-dioxin, for example.

The polychlorinated species are designated by the following symbols:

D	dichloro
Tr	trichloro
T	tetrachloro
Pe	pentachloro
Hx	hexachloro
Hp	heptachloro
O	octachloro

CAS No.: Differs according to specific congener. Refer to Table 1 for the most important congeners.

Boiling point: 412.2°C for 2,3,7,8-TCDD (estimated)

Color Colorless or white

Conversion factor: Depends on molecular weight. For 2,3,7,8-TCDD, is 1 ppb = 13.384 $\mu g/m^3$

Flammable limits: Unknown

Henry's law constant: 2.1×10^{-6} atm-m^3/mol (estimated)

Melting point: 305°C for 2,3,7,8-TCDD

Molecular formula: Dioxins: $C_{12}O_2Cl_zH_{8-z}$; Furans: $C_{12}OCl_zH_{8-z}$

Molecular weight: 202 to 460. 321.97 for 2,3,7,8-TCDD

Odor threshold: Unknown

Solubility

Water: 0.00000791 to 0.000317 mg/l for 2,3,7,8-TCDD at 20-25°C, according to different sources

log K_{ow}: 6.15-7.28 for 2,3,7,8-TCDD, according to different sources

log K_{oc}: 6.0-7.39 for 2,3,7,8-TCDD, according to different sources

Specific gravity: 1.827 g/ml for 2,3,7,8-TCDD (estimated)

Vapor pressure: 1.4×10^{-9} mm Hg for 2,3,7,8-TCDD at 25°C (estimated)
3.5×10^{-9} mm Hg for 2,3,7,8-TCDD at 30°C

(References: ATSDR, 1987; EPA 1989; EPA 1992a)

USES: The CDDs and CDFs have no known use except as research chemicals. They are produced, however, in a variety of processes involving chlorine-containing substances and organic chemicals. They are known to be formed in the production of chlorophenols and herbicides such as 2,4,5-T (a constituent of Agent Orange), in the chlorine bleaching of wood pulp, in incineration of municipal and certain industrial wastes, in combustion of wood in the presence of chlorine (including forest fires), in exhaust from automobiles using leaded gasoline with a chlorinated scavenger, and in improper disposal of certain chemical wastes (ATSDR, 1987). The CDFs are also known to be formed in the production and especially the combustion of polychlorinated biphenyls (PCBs). The formation of the CDDs from PCBs is not favored because of the necessity to break the carbon-carbon biphenyl bond.

ABSORPTION, METABOLISM, AND EXCRETION: The CDDs and CDFs can be absorbed into the body by inhalation, ingestion, and dermal contact. As they are highly soluble in lipids but not in water, the vast majority of the compounds will be found in fatty tissues.

A substantial fraction of ingested 2,3,7,8-TCDD is absorbed from the gastrointestinal tract: 66-93% in rats (Rose et al. 1976), 50% in guinea pigs (Nolan et al. 1979), 75% in hamsters (Olson et al. 1980), and 87% in one human observation (Poiger and Schlatter, 1986). Other CDDs and CDFs are also absorbed at rates that are congener-specific, nearing 100% for 2,3,7,8-TCDF (Birnbaum et al. 1980, Decad et al. 1981), but only about 2-15% for OCDD (Birnbaum and Couture, 1988). If the dioxins or furans are attached to soil or some other medium upon ingestion, they are usually substantially less bioavailable (EPA 1992b).

The CDDs and CDFs appear to be absorbed only slowly through the skin (EPA 1992b). Studies that followed absorption over time in rodents observed approximately first-order absorption at rates of about 0.5 to 1.0% per hour (Banks and Birnbaum, 1991a,b). Absorption rates also appear to decline as the applied dose increases (Brewster et al. 1989). Limited data suggest that absorption through human skin might be an order of magnitude slower than for rodent skin, and that a substantial portion of the dioxins that move into skin remain in the stratum corneum for some time (EPA 1992b), possibly sloughing off before absorption into body. Dioxins associated with soil or other lipophilic media are substantially less well absorbed through skin than the neat compound (Poiger and Schlatter, 1980).

Data are more limited for absorption of the CDDs and CDFs from the lung, but the available studies suggest that absorption of the neat compound from the lung is similar to that from the gastrointestinal tract, i.e., around 90% for 2,3,7,8-TCDD (Diliberto et al. 1992).

Following absorption, the CDDs and CDFs are probably distributed primarily by the lymphatic system, where binding to lipoproteins may alter its pharmacokinetics (EPA 1992b). It then moves rapidly into adipose tissue and the liver in all species investigated (EPA 1992b). The CDDs and CDFs are eliminated from the human body relatively slowly (half-life for 2,3,7,8-TCDD of the order of 6 years)(Poiger and Schlatter 1986) but more rapidly in other species. It is thought that metabolism of these compounds is required for elimination by conversion to polar metabolites such as hydroxy CDDs and CDFs that can be rapidly excreted via urine or bile (EPA 1992b). Conversion rates are species and congener specific. Metabolic conversion also appears to reduce toxicity.

The CDDs and CDFs also distribute to mother's milk during lactation, which can provide an exposure pathway for nursing infants (Korte et al. 1990). Typical concentrations in mother's milk are about 2-5 pg/g in the United States, with higher values seen where exposure was known to take place (Schecter et al 1988). Exposure via placental transfer is relatively less important (EPA 1992b).

TOXICITY OVERVIEW: The CDDs and CDFs are toxic to a wide variety of organ systems and can cause dermal toxicity, immunotoxicity, reproductive toxicity, and a variety of other chronic toxicities including cancer. The coplanar CDDs and CDFs with chlorines at three or four of the 2, 3, 7, and 8 positions appear to be most toxic. All of these toxicities are thought to be related to the same mechanisms (beginning with binding to the Ah receptor - see Carcinogenicity, below).

ACUTE TOXICITY: 2,3,7,8-TCDD is toxic to all mammalian species but large differences in toxic potency occur among species and among strains of the same species (EPA 1992c). The toxicity is manifested by a loss of body weight (the "wasting syndrome") and by toxicity in the liver, thymus, and lymphatic tissues as well as in other tissues in some species. For one-time acute exposures, the lethal doses range from 0.6 µg/kg body weight in male guinea pigs through 20-60 µg/kg in rats to 5500 µg/kg in hamsters (ATSDR 1987). If a lethal dose is incurred, deaths usually occur within two or three weeks after a single exposure.

CHRONIC TOXICITY: The CDDs and CDFs can affect a variety of organ systems after continuing exposure. Lethality from chronic exposure occurs in the same range of total dose as for acute exposures. For example, feeding 0.008 µg/kg-day for a total of 0.8 µg/kg was lethal to guinea pigs over a 90-day period (DeCaprio et al. 1986). Toxicities to specific organ systems are discussed below. Laboratory experiments with CDDs and CDFs typically involve exposure by injection, gavage, or ingestion with feed or water, except as indicated below. Systemic toxicities can presumably be caused by inhalation or dermal absorption as well.

Dermal Toxicity: The best documented human effect from exposure to the CDDs and CDFs is chloracne, a skin disease characterized by the formation of black lesions on the face and upper torso. Chloracne is also induced by other chlorinated aromatic compounds, but the CDDs and CDFs are particularly potent, although the exact dose needed to induce chloracne in humans is not known. Chloracne was frequently observed in children exposed to dioxin following the pentachlorophenol factory accident in Seveso, Italy, and in various occupationally exposed populations. Chloracne or similar conditions can be induced in monkeys, rabbits, and hairless mice at doses down to 0.08 µg.

Liver Toxicity: 2,3,7,8-TCDD induces hyperplasia and hypertrophy of liver parenchymal cells and therefore increases liver weight in all species investigated (EPA 1992c). These changes are accompanied by impaired liver function. At higher doses, frank liver necrosis is seen and can lead to death. A variety of biochemical and structural changes are also seen (EPA 1992c). Liver damage in rats and mice appears at lower doses than in guinea pigs and hamsters. Effects on the liver are not usually seen below total doses of 1 µg/kg body

weight. Porphyria in rats exposed to 2,3,7,8-TCDD appeared at a chronic exposure of 1 μg/kg per week over 45 weeks but not at 0.1 μg/kg per week (Cantoni et al. 1981).

Other organ systems: There is limited evidence that other organ systems are affected by exposure to the CDDs and CDFs. For example, ATSDR (1987) states that the human nervous system may be affected and that the kidney and digestive system have been affected in laboratory animals. Endocrine regulation alterations have been suggested both in humans and laboratory animals exposed to 2,3,7,8-TCDD (EPA 1992c). Effects on vitamin A storage and lipid peroxidation have also been discussed as signs of dioxin toxicity (EPA 1992c). Enzyme induction is the most universal marker of dioxin exposure at potentially toxic levels and has been reported at single doses as low as 0.002 μg/kg body weight (Kitchin and Woods, 1979).

SENSITIZATION: No reports of studies of CDDs and CDFs as sensitizers were located.

TARGET ORGAN EFFECTS: Although the CDDs and CDFs are clearly pluripotent toxicants, the most sensitive organ systems appear to be the liver (chronic toxicity and carcinogenicity), the skin (chloracne), the immune system, and the reproductive and developmental systems.

REPRODUCTIVE TOXICITY: 2,3,7,8-TCDD and similar CDDs and CDFs have been reported to cause reproductive toxicity in both female and male laboratory animals (EPA 1992d). In females, the effects reported include reduced fertility, reduced litter size, changes in the estrous/menstrual cycle, and effects on the gonads. In males, reported effects include reduced fertility and spermatogenesis, decreased sex organ weights, and abnormal testicular morphology.

In female rats, decreases fertility and litter size have been observed at doses of 0.01 μg/kg-day but not at 0.001 μg/kg-day (Murray et al. 1979) in continuous dosing studies. In female monkeys, 25 ppt 2,3,7,8-TCDD in diet caused problems in conception and gestation (Schantz and Bowman, 1989). In humans, 25 ppt in diet would be considerably less than 0.001 μg/kg-day. These effects do not seem to be explainable by changes in hormone levels, which have been observed only at higher exposure levels (Allen et al. 1979). Nor is antiestrogenic action clearly associated with the clinical effects (Shiverick and Muther 1983), although dioxin binding to the Ah receptor does appear to be involved in the antiestrogenic effect (Zacharewski et al. 1991).

Effects of dioxins on spermatogenesis in males may be secondary to other effects and appear to occur only at relatively high exposure levels (Kociba et al. 1976). 2,3,7,8-TCDD can affect androgen levels down to single doses of 15 μg/kg (EPA 1992d).

DEVELOPMENTAL TOXICITY: The effects of the CDDs and CDFs on growth and development have been studied in a variety of animal models as well as by human epidemiology. The reported effects can be divided into death/growth/clinical signs, structural malformations, and functional alterations (EPA 1992d).

In laboratory mammals, prenatal exposure to 2,3,7,8-TCDD causes mortality in the monkey and several species of rodent (EPA 1992d). The stage of gestation is very important in determining whether a given dose will be fetotoxic. Mortality of the fetus is usually accompanied by maternal toxicity in rodents but not necessarily in the monkey. Fetal mortality is seen at total maternal doses down to about 1 μg/kg; the complication of gestational period makes it difficult to state the threshold daily dose that would not cause fetal toxicity.

The signs of dioxin toxicity in mammalian embryos include decreased thymus development, blood alterations, and edema. In the mouse, cleft palate is frequently seen, while intestinal hemorrhage is seen in the rat (EPA 1992d).

As with most other effects of dioxin, the evidence for developmental toxicity in humans is marginal. Perinatal mortality and other signs of developmental toxicity (growth retardation, structural malformations, and organ dysfunction) were reported in pigmented babies born to mothers who had consumed rice oil contaminated with chlorinated furans (Hsu et al. 1985). However, PCBs were also present and the dose classification system was weak. The greatest prevalence was in mothers who showed signs of maternal toxicity (specifically, chloracne).

The principal structural abnormalities seen in laboratory animals exposed to CDDs and CDFs are cleft palate, hydronephrosis in the kidney, and retarded development of the thymus (EPA 1992d). Hydronephrosis appears to be the most sensitive endpoint and can occur in the 1 μg/kg range. Studies have indicated that the Ah receptor is probably critical to the development of cleft palate and hydronephrosis from the CDDs and CDFs.

Various post-natal effects of maternal exposure to CDDs and CDFs have also been reported. Among them are perinatal androgen deficiencies and associated sexual aberrations in male rats (Mably et al. 1991, 1992) and neurobehavioral aberrations in monkeys (Schantz and Bowman 1989). Rojan et al. (1988) also reported that children born to mothers who had eaten rice oil contaminated with chlorinated furans had behavioral problems.

IMMUNOTOXICITY: Numerous studies of the toxicity of the CDDs and CDFs to the immune system have been conducted in cell cultures, whole animals, and by epidemiologic observations in humans (EPA 1992e). At relatively high doses, they produce lymphoid tissue depletion, with the thymus the most sensitive tissue. Various immunological functions are affected by exposure to 2,3,7,8-TCDD and related substances, and in some cases the effects are clearly adverse, resulting in increased susceptibility to infectious disease at doses down to 1 μg/kg once a week for four weeks (equivalent to about 0.14 μg/kg-day) (Thigpen et al. 1975).

Both cell-mediated and humoral immune responses can be suppressed by 2,3,7,8-TCDD exposure, which has been interpreted to mean that multiple cellular targets may be at risk. The suppression of immune response to sheep red blood cells (SRBC) by 2,3,7,8-TCDD is seen in most species tested. The effects of the CDDs and CDFs on other organ systems may also have secondary effects on the immune system.

As with other toxic effects, the Ah receptor binding of the dioxin appears to contribute to immunotoxicity, although some effects do not seem to be Ah-dependent. Some of the observed effects of dioxin exposure on the immune system are clearly beneficial in the sense of protecting it against other immunotoxic agents; these are probably limited to low doses of the CDDs and CDFs. Other effects are not clearly beneficial or adverse, but may simply be adaptations to exposure. In guinea pigs (again one of the most sensitive species), immune system effects have been reported at doses down to 0.006 μg/kg-day (Vos et al. 1973).

As with other toxicities of the CDDs and CDFs, the epidemiologic evidence is subject to various interpretations. The consumption of rice oil contaminated with PCBs and polychlorinated furans was reported to be associated with increased rates of infections, decreased concentrations of some serum immunoglobulins, and increases and decreases in some categories of T cells (Lu and Wu, 1985). Similar changes were reported in Missouri residents with elevated levels of 2,3,7,8-TCDD in fat (Webb et al. 1989). These changes were not seen in the children exposed in Seveso (Mocarelli et al. 1986), but some lymph abnormalities in another cohort of children were reported by Tognoni and Bonaccorsi (1982). Confounding of exposures prevents developing any dose-response information for most of the studied cohorts, and conflicting evidence from different studies can be interpreted either as a lack of evidence of effect or as a caution about the sensitivity of the test methods. While it cannot be stated with certainty that immunologic effects of dioxin and furan exposures in humans have been demonstrated, neither can it be concluded that the observations are inconsistent with the observed immunotoxicity in laboratory animals.

GENOTOXICITY: The CDDs and CDFs exhibit very little direct mutagenicity in a variety of test systems, but some positive results have been reported (ATSDR 1987). Bronzetti et al. (1983) reported mutagenesis by 2,3,7,8-TCDD in *Saccharomyces cerevisiae* with enzyme activation as well as gene conversion in cytogenetic tests. Structural aberrations in marrow cells of mice (Loprieno et al. 1982) and rats (Green et al. 1977) have also been reported in cytogenetic tests of 2,3,7,8-TCDD. It is generally agreed that if the CDDs and CDFs are genotoxic at all, that toxicity is not very important to the primary risks of dioxin exposure.

CARCINOGENICITY: In rats, ingestion of 2,3,7,8-TCDD has been associated with an increase in liver tumors (and tumors in some other organs) at a very low level of daily dose (Kociba et al., 1978). The only other reported positive cancer bioassay of a dioxin or furan was for a mixture of 2,3,7,8-HxCDs (NTP 1980). On the basis of the Kociba data, the International Agency for Research on Cancer and the U.S. National Toxicology Program have declared the evidence for the carcinogenicity of 2,3,7,8-TCDD in experimental animals to be sufficient.

The evidence for the carcinogenicity of the CDDs and CDFs in humans is less well established. Several epidemiologic studies of populations potentially exposed to CDDs and CDFs have been reported as positive by their authors (e.g., Fingerhut, et al. 1991; Manz et al. 1991; Hardell et al. 1981), but all have weaknesses that prevent them from being definitive. In particular, most of the populations studied had exposures to other substances that might have been responsible for the observed associations. No official body has declared the evidence for human carcinogenicity sufficient. Therefore, 2,3,7,8-TCDD is generally

described as a suspect or presumed human carcinogen.

In spite of the lack of confirmatory experimental or epidemiological evidence, certain other CDDs and CDFs are assumed for policy purposes to be carcinogenic, based on the similarity of their effect on other types of toxicity and on biochemical responses such as enzyme induction. The most popular theory supporting this policy choice is that any of the tetra-, penta, hexa-, hepta-, and octa-chlorinated species with chlorines at the 2, 3, 7, and 8 positions will be coplanar and sterically similar to 2,3,7,8-TCDD and therefore potentially carcinogenic. If enzyme induction is part of the chain of carcinogenic effect, as is widely assumed, then the relative potency for induction of enzymes should predict the relative potency for carcinogenicity, perhaps modified by factors related to the kinetics and metabolism of the congener in the body as evidenced in differential non-cancer toxicity.

As a result of these policy assumptions, all of the 2,3,7,8-substituted congeners have been assigned "toxicity equivalence factors" or TEFs by a variety of regulatory agencies. The most widely accepted set of TEFs (the I-TEFs) was proposed by an international committee sponsored by NATO. The carcinogenic potency of specified congeners is calculated as the product of the TEF and the carcinogenic potency of 2,3,7,8-TCDD. The I-TEFs are shown in Table 1.

Ingestion: Groups of female rats administered 2,3,7,8-TCDD in diet at 0.01 and 0.1 μg/kg-day exhibited excess cancers of the liver, lung, tongue, hard palate, and nasal turbinates in comparison with a control group (Kociba et al., 1978). A group treated at 0.001 μg/kg-day did not show excess cancer and in fact tumor incidence in that group was lower than in the controls. The excesses were statistically significant in the liver for both of the higher exposures when adenomas were included in the tumor count. Male rats did not exhibit excess tumor incidence, and some organs in the female rats showed reduced tumor incidence at the lowest dose.

In another study of exposure to 2,3,7,8-TCDD by gavage conducted by the U.S. National Toxicology Program (1982), rats and mice exhibited statistically significant excess cancers in the liver and/or thyroid at doses of 0.007 μg/kg-day or above, but these data are not generally used to compute the carcinogenic potency of 2,3,7,8-TCDD. Van Miller et al. (1977) and Toth et al. (1979) also have reported increased cancers in laboratory rodents exposed to 2,3,7,8-TCDD.

Based on the Kociba data and further analyses, the U.S. Environmental Protection Agency (EPA) has established a cancer potency slope factor of 1.56×10^5 $(mg/kg\text{-}day)^{-1}$ to be used in risk assessments conducted by and for the agency. Similar values are used by the U.S. Occupational Safety and Health Administration, the U.S. Consumer Product Safety Commission, and the California Office of Environmental Health Hazard Assessment, all of which assume that the carcinogenicity of 2,3,7,8-TCDD scales with body surface area from rodents to humans. The U.S. Food and Drug Administration assumes that the carcinogenicity scales with body weight and uses a slope factor of 1.75×10^5 $(mg/kg\text{-}day)^{-1}$ for 2,3,7,8-TCDD.

A mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-HxCDD given by gavage increased liver tumors in both sexes of rats and mice at doses down to 0.18 μg/kg-day, with female rats again being the most sensitive (NTP, 1980). EPA (1992f) concluded on the basis of these data that HxCDD was approximately 1/20 as potent a carcinogen as 2,3,7,8-TCDD.

Inhalation: No studies of dioxin or furan carcinogenicity in laboratory animals by inhalation were found in the open literature. Many of the reported human epidemiological studies of exposures to CDDs and CDFs have some inhalation component (e.g., Fingerhut et al. 1991; Hardell et al. 1982), but the contribution of other pathways is often uncertain.

Skin Contact: The NTP (1982b) studied the carcinogenicity of 2,3,7,8-TCDD applied to the skin of Swiss mice. An increase of fibrosarcomas of the integumentary system was seen in female but not male mice. The evidence for carcinogenicity by dermal exposure is considered limited.

The evidence that 2,3,7,8-TCDD is a tumor promoter when applied to the skin following treatment with tumor initiators is mixed. Berry et al. (1978) reported no increase in tumor promotion in CD-1 mice after initiation with dimethylbenzanthracene (DMBA), but Poland et al. (1982) reported promotion after DMBA treatment in HRS/J hairless mice and concluded that susceptibility to promotion also had a genetic component. Protection by 2,3,7,8-TCDD against dermal carcinogenicity of known skin carcinogens has also been reported (Berry et al. 1979, Cohen et al. 1979).

Other Routes of Exposure: 2,3,7,8-TCDD has been reported to increase skin tumors in the facial region of golden hamsters following subcutaneous or intraperitoneal injection (Rao et

al. 1988). Intraperitoneal injections of 2,3,7,8-TCDD into mice also increased liver tumors and thymic lymphomas (Della Porta et al. 1987).

Mechanisms: Several hypotheses have been offered to explain the carcinogenicity of 2,3,7,8-TCDD and (presumably) of other CDDs and CDFs in laboratory animals. These mechanistic explanations also can be used to project their carcinogenic potential in humans. Among other findings, it is clear that the CDDs and CDFs are not directly genotoxic and therefore are not classic tumor initiators but strong tumor promoters (EPA 1992f).

The leading hypothesis (EPA 1992g,h) entails a cascade of events initiated by the binding of a dioxin or furan to an intracellular protein receptor known as the aryl hydrocarbon (Ah) or dioxin receptor. The binding is strongest for planar structures with at least 3 chlorines in the lateral (2, 3, 7, and 8) positions. Possibly after further transformations, including binding with another macromolecule called ARNT (Hoffman et al. 1991), the dioxin/receptor complex binds to DNA and modifies the genes that control transcription of proteins. Among other effects, CDDs and CDFs induce the aryl hydrocarbon hydroxylase (AHH) enzymes. Whether the AHH induction is the cause of the carcinogenicity or simply a signal of Ah receptor binding is not yet certain, but it is certain that the dioxin/Ah receptor complex can induce transcription of other genes, and that the various toxic effects of dioxin may trace back to different gene interactions. For example, dioxin can block binding of epidermal growth factor (EGF) to its receptor, allowing the excess EGF to stimulate cell division. Dioxin may also affect the storage and translocation of thyroid hormones (McKinney et al. 1985). The carcinogenicity of the CDDs and CDFs to different organ systems may be mediated by different gene effects.

No matter which series of events is necessary for cancer induction, overt organ toxicity may be necessary for the development of liver cancer by 2,3,7,8-TCDD. Sauer (1990) observed a high degree of correlation between liver toxicity and liver adenomas and carcinomas in the Kociba rats.

The hypothesized mechanisms for dioxin carcinogenicity imply that the incidence of tumors may not be linearly related to dioxin exposure. Because of the variety of co-factors for expression of the genetic transformations induced by dioxin, there may be considerable variation of susceptibility in the human population, both from genetic variability and from exposure to other chemicals.

EPIDEMIOLOGY: Epidemiological investigations of human populations exposed to CDDs and CDFs, usually with concomitant exposure to other potentially toxic substances, are described in the sections on dermal toxicity, immunotoxicity, developmental toxicity, and carcinogenicity, above. EPA (1993) has recently summarized the epidemiology and human data for TCDD. With respect to cancer, the evidence for an association of TCDD exposure with soft tissue sarcoma is best, with some evidence for malignant lymphomas, lung cancer, and a few other cancers. Many of the individual studies have either potentially confounding exposures or methodological difficulties, and firm conclusions about the potency of the CDDs and CDFs in humans are not yet possible. Although the evidence is good that chloracne, gamma glutamyl transferase activity, diabetes/serum glucose levels, and reproductive hormone levels are associated with exposure to TCDD, most other associations with non-cancer health effects are described as weak or needing further research (EPA 1993). For policy purposes, it is assumed that sufficient exposures to CDDs and CDFs would cause in humans most of the toxic effects seen in laboratory animals.

ENVIRONMENTAL FATE: Most of the available information on the environmental fate of the CDDs and CDFs comes from studies of 2,3,7,8-TCDD. Although general similarity is expected for other CDDs and CDFs, congener differences are also expected. In general, these compounds are very stable in the environment and relatively immobile except for mass transport when bound to suspended sediment or airborne dust (EPA 1992a).

2,3,7,8-TCDD is very insoluble in water and has a relatively low vapor pressure, so transport in dissolved form or in gas phase is not very important in comparison to transport with suspended sediment or dust. Leaching from soil to groundwater is relatively unlikely except in soils with very low organic carbon content and an organic co-solvent (ATSDR 1987).

Typical concentrations of CDDs and CDFs have been summarized by EPA (1992a). In North America, the toxic equivalent concentrations for CDDs and CDFs combined are 7.69 ppt for soil, 3.27 ppt for sediments, 1.37 ppt for fish, 8.36 ppq for surface water, and 0.051 pg/m^3 for air. Values for dairy products and beef are not reported for North America, but worldwide are reported as 0.0995 and 0.37 ppt, respectively.

Environmental degradation processes for the CDDs and CDFs are limited and usually slow. Although biodegradation by a fungus has been

reported (Bumpus et al. 1985), the only important degradation process is probably photodegradation, which can occur for dioxin in air, clear water, or deposited on surfaces exposed to sunlight. Once the dioxin is mixed into the soil or sediments, it is expected to be very persistent. The general environmental half life for 2,3,7,8-TCDD is not well known; it has been estimated to be of the order of 1 or 2 years on surface soils or in water, but 10 to 12 years in deep soils (ATSDR 1987). The latter range is usually used in risk assessments.

ENVIRONMENTAL TOXICITY:
The toxic effects of the CDDs and CDFs are exhibited in essentially all mammalian species with varying potencies that depend on the biochemical pathways for binding with receptors and with DNA, and on the subsequent gene products that can be induced. Among environmentally sensitive mammals, mink seem especially sensitive. Many of these toxic mechanisms also can occur in nonmammalian species. Because the dioxins are extremely lipid-soluble, they can bioconcentrate from aqueous media and biomagnify up the food chain. Top predators such as raptors are probably at greater risk, as in the case of PCBs and DDT. Much of the environmental toxicity of the dioxins may be expressed as reproductive or developmental toxicity.

The early life stages of fish appear to be more sensitive to the toxicity of the CDDs and CDFs than are adults. Arrested growth and development have been reported in several species exposed to 2,3,7,8-TCDD and the relative potencies for sterically similar CDD and CDF congeners appear to be similar to those reported for various mammalian endpoints (Walker and Peterson, 1991).

Similarly, bird embryos appear to be more sensitive to the CDDs and CDFs than adult birds. CDDs and CDFs injected into fertilized chicken eggs causes edema, liver lesions, inhibition of lymphoid development in the thymus, and various other abnormalities in the developing chick (EPA 1992d).

REGULATORY STATUS: 2,3,7,8-TCDD is considered a probable human carcinogen by the U.S. Environmental Protection Agency (Group B2), the U.S. National Toxicology Program, and the International Agency for Research on Cancer (Group 2B). EPA's IRIS system lists its carcinogenic potency as 1.56×10^5 kg-day/mg. The EPA (1989) also uses the International Toxicity Equivalence Factors to assess the carcinogenicity of other dioxin and furan congeners.

2,3,7,8-TCDD is listed as a substance subject to regulation under virtually every environmental stipulation. Under the Clean Water Act, it is a priority pollutant and its discharges to water must be regulated. Although the setting of ambient water quality standards is left to the States, EPA recommends an in-stream standard of 13 parts per quintillion. Under the Safe Drinking Water Act, drinking water supplies must contain no more than 50 parts per quadrillion. (This value is the practical quantitation limit.) It is listed as one of the 189 hazardous air pollutants that must be regulated under the Clean Air Act Amendments. Under the Comprehensive Environmental Restoration, Cleanup and Liability Act (Superfund), its Reportable Quantity is 1 pound. The Superfund Amendments and Reauthorization Act also requires industries to report discharges under Title 3. The Resource Conservation and Recovery Act designates it as a hazardous constituent of waste, and under the Federal Insecticide, Fungicide, and Rodenticide Act, it must be undetectable in the pesticides 2,4,5-T and Silvex and must meet a tolerance in hexachlorophene.

2,3,7,8-TCDD is also regulated by the U.S. Food and Drug Administration (through a detection limit for occurrence in food-producing animals and an advisory limit of 25 ppt in those fish sold in interstate commerce). It has been the subject of joint study by EPA, FDA, and the Consumer Product Safety Commission regarding its occurrence in products and wastes from the chlorine bleaching of wood pulp.

As an occupational hazard, 2,3,7,8-TCDD is regulated by the U.S. Occupational Safety and Health Administration under its Hazard Communication Standard and as a laboratory hazard. OSHA has not promulgated a permissible exposure level, but the U.S. National Institute for Occupational Safety and Health has recommended that dioxin exposure be reduced to the lowest feasible level.

(References: NTP 1991; HSDB 1993)

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[1]Although this document is marked "Review Draft (Do Not Cite or Quote), it is by far the most comprehensive summary of information on the toxicology of dioxin available at present; the statements attributed to it are backed up with extensive documentation to primary literature sources, many of which are also cited directly in this toxicity profile.

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TOXICOLOGY PROFILE

FORMALDEHYDE

Synonyms: Formalin, oxomethane, methanal, Formol, methaldehyde,

CAS No: 50-00-0

Boiling point: 19.5° C

Color: Colorless gas or liquid

Conversion factor: 1 mg/m^3 = 0.815 ppm

DOT designation:
Flammable/Combustible

Flammable limit:
Autoignition: 430° C
Flash point: 50 - 60° C (depending on methanol content)
LEL: 7.0%
UEL: 73%

Henry's law constant: 3.27 x 10^{-7} atm m^3/mole

Melting point: -92 °C

Molecular formula: H_2CO

Molecular weight: 30.0

Odor: pungent odor
Threshold: 0.07 mg/m^3 (0.05 - 1.0 ppm)
Recognition: 1.0 ppm
Characteristics: pungent

pH: 2.8 - 4.0

Solubility
water: miscible with water
K_{ow}: 0.35
other: miscible with alcohol and acetone
K_{oc}: NA

Specific gravity: 0.815 @ 4°C

Vapor density: 1.067

Vapor pressure: 10 mm Hg at -88°C; 3284 mm Hg @ 20°C

Viscosity: NA

NA = not available
(References: Hawley, 1987; Verschueren, 1983; Merck, 1983)

COMPOSITION: Pure formaldehyde is not available commercially because of its tendency to polymerize. The liquid is sold in aqueous solutions containing 30-50% formaldehyde and may contain 0 to 15% methanol as a stabilizing agent (Kirk-Othmer, 1984).

USES: Formaldehyde is used as a chemical sterilant, fixative in leather tanning, preservative, embalming fluid, and fumigant. It is also used in the manufacture of commercial products such as urea/formaldehyde and phenol/formaldehyde resins, wrinkle-proof fabrics, rubber products, dyes, textiles, plastics, paper products, cosmetics, plywood, particle board and foam insulation (HESIS, 1986). It is a component of gasoline engine exhausts (10 - 15

ppm) and tobacco smoke (37.5 - 73.1 μg/cigarette) (Verschueren, 1983; HSDB, 1990).

ACUTE TOXICITY: The acute toxicity values for different mammalian species are presented in Table 1.

Ingestion: Ingestion of formaldehyde produces severe abdominal pain, pain in the throat, headache, vomiting and diarrhea. This may be followed by gastritis with diffuse ulceration, fibrosis and contracture of the stomach. Effects may be severe enough to require gastrectomy. The urine is scanty and can contain red blood cells and casts. Death from shock may occur 1-2 days after ingestion. If recovery occurs, it may be rapid and complete (Arena, 1986). Part of the symptomatology following ingestion of formaldehyde may be attributed to the production of a metabolic acidosis (HSDB, 1990).

Inhalation: Formaldehyde gas may cause severe irritation of the mucous membranes of the respiratory tract. The threshold for upper airway irritation appears to be in the range of 0.1 - 2.0 ppm while the threshold for lower airway and other pulmonary effects among healthy individuals is in the range of 5 - 30 ppm (NRC, 1981). Higher concentrations may produce edema or spasm of the larynx. Severe obstructive tracheobronchitis may also result from inhalation of high concentrations (Sittig, 1985) and pulmonary edema, inflammation and pneumonia have been reported to occur at levels between 50 - 100 ppm (Porter, 1975).

Table 1. Acute toxicity values of formaldehyde for selected mammalian organisms.

Species	Value	Dose	Time	Comments
Guinea pig	LD_{50}	260 mg/kg		Oral dose
Rat	LD_{50}	0.1 - 0.8 g/kg		
Rat	LC_{50}	250 ppm 830 ppm	4 hr. 30 min.	
Rat	LD_{50}	800 mg/kg		Oral dose
Rat	LD_{50}	420 mg/kg		Subcutaneous
Rat	LD_{50}	87 mg/kg		Intravenous
Rat	LC_{50}	590 mg/m^3		Inhalation
Mouse	LD_{50}	42 mg/kg		Oral dose
Mouse	LD_{50}	300 mg/kg		Subcutaneous
Mouse	LC_{50}	400 mg/m^3/2h		Inhalation
Rabbit	LD_{50}	270 mg/kg		Skin
Cat	LC_{50}	650 ppm 200 ppm	8 hr. 3.5-4.0 hr. recovery by 6 days	Irritation
Mammal (species unspecified)	LD_{50}	92 mg/m^3		Inhalation

(Reference: RTECS, 1990)

Individuals with chronic respiratory disease may develop lower respiratory effects (cough, shortness of breath) at concentrations <5 ppm. Asthmatics may develop acute asthmatic attacks at formaldehyde concentrations of 0.25 - 5 ppm. Allergic contact dermatitis and urticaria have been observed following inhalation of formaldehyde gas (Sittig, 1985).

Formaldehyde has been reported to increase airway resistance in guinea pigs that inhaled 0.31 ppm (0.42 mg/m^3) of an aerosol (ACGIH, 1986).

Eye contact: Contact with vapors can produce eye irritation and conjunctivitis (Arena, 1986). Human testing indicates that definable eye irritation occurs between 0.05 and 0.5 ppm (NRC, 1981). Direct eye contact with liquid may cause severe eye irritation, permanent opacification, and loss of vision (Sittig, 1985). Testing of a 15% solution on experimental animals resulted in severe corneal damage, conjunctival edema and

iritis. Rabbits exposed to vapors of 40 -70 ppm showed slight tearing and eye discharge but not corneal injury (Grant, 1974).

Skin contact: Direct contact of the skin with formaldehyde solutions can cause brown discolorations, irritant contact dermatitis with pustulovesicular lesions and occasionally sloughing of the skin. Allergic contact dermatitis can result from contact with formaldehyde solutions or formaldehyde-treated products. Once sensitized, the individual may develop recurrent dermatitis after exposure to very low levels of formaldehyde (e.g. <0.3% solutions) (OSHA, 1987).

CHRONIC TOXICITY:

Ingestion: No data were located regarding the effects of chronic ingestion of formaldehyde.

Inhalation:
Workers chronically exposed to formaldehyde concentrations < 3 ppm have reported burning and dryness of the eyes, nose, and throat. Some level of tolerance can develop in individuals that are chronically exposed to formaldehyde (NRC, 1981). Headaches and lower respiratory effects (cough, chest discomfort) have also been noted (OSHA, 1987). Occupational asthma has been reported among workers exposed to formaldehyde. Formaldehyde exposure may also exacerbate respiratory symptoms among those with preexisting chronic respiratory disease.

Impairment in the mucociliary function of the nose and nasal squamous cell metaplasia were noted in one small study of formaldehyde-exposed workers (EPA, 1987).

Some concern has been raised over the level of formaldehyde in residential homes. The Consumer Product Safety Commission and Departments of Housing and Urban Development, in a 1 year period (1979-1980), received numerous calls and complaints from mobile home users and residents of conventional homes with foam insulation on ill health effects attributable to formaldehyde. Several studies during this time period indicate that the formaldehyde levels ranged from non-detectable to 2 ppm with fewer than 10% of the determinations over 1.0 ppm. The prevalence of symptoms in decreasing order were eye irritation, respiratory tract irritation, headache, nose irritation, nausea and drowsiness. Severity of the symptoms was not correlated with the formaldehyde exposure concentration (Breysse, 1978). Several studies reporting similar results are reviewed in NRC, (1981).

Animal studies have also associated formaldehyde exposure with nasal carcinomas (Klassen, 1986). These are reviewed under CARCINOGENICITY.

Eye contact: Refer to section on ACUTE TOXICITY.

Skin contact: Refer to section on ACUTE TOXICITY.

SENSITIZATION: Sensitization may develop after skin contact with formaldehyde, resulting in allergic contact dermatitis (Proctor, 1988). Cases of newly diagnosed occupational asthma associated with the inhalation of formaldehyde have also been reported. Guinea pigs are readily sensitized to challenges of formaldehyde but dermal sensitization to airborne formaldehyde has not been reported (NRC, 1981).

TARGET ORGAN EFFECTS: The most direct tissues or organs affected by formaldehyde exposure are the eyes, upper and lower respiratory tracts and skin. Other target organs include the liver, (hepatotoxicity and jaundice), the kidney (nephritis), and the hematopoietic system (hemolysis following hemodialysis accidents) (HSDB, 1990).

Large doses of formaldehyde have been reported to increase the blood pressure in mice (Egle, 1974) but dogs appear to respond in the opposite fashion perhaps owing to a sympathetic nervous system response (Tani et al., 1978).

ABSORPTION-METABOLISM-EXCRETION: Formaldehyde is well absorbed through inhalation. In dogs, the respiratory tract uptake is almost 100% Formaldehyde is rapidly oxidized to formic acid in various tissues including the liver and erythrocytes. The elimination half-life was estimated to be 1 - 2 minutes in monkeys, following intravenous infusion, with blood formic acid levels increasing concomitantly. Much of the formic acid is further oxidized to carbon dioxide and water in laboratory rodents. There can also be urinary excretion of significant amounts of formate, usually as a sodium salt. Metabolic conversion of some formic acid to labile methyl groups after activation by tetrahydrofolic acid also occurs (EPA, 1985).

Formaldehyde probably reacts with the mucosa of the alimentary and respiratory tracts. *In vitro* and *in vivo*, it has been shown to react with a variety of functional groups resulting in the formation of addition products or the initiation of polymerization reactions. Sulfhydryl reagents

antagonize the lethal effects of injected formaldehyde in rodents (Gosselin et al., 1984).

Formaldehyde is normal metabolite and vital ingredient in the synthesis of essential biochemical substances in man and animals.

NEUROTOXICITY: The severity of symptoms related to formaldehyde exposure are dose related. Neurologic effects are not noted at concentrations below 0.05 ppm. Between 0.05 and 1.5 ppm changes have been reported in optical chronaxie, EEG and the sensitivity of the dark-adapted eye to light (reviewed in NRC, 1981). At high concentrations, coma and death can result (HSDB, 1990).

REPRODUCTIVE TOXICITY:

Formaldehyde has not been reported to be teratogenic or to cause reproductive toxicity in animals (Clement, 1985). Pregnant dogs fed formaldehyde-containing diets at 125 and 375 ppm on days 4 - 56 of gestation did not show any teratogenic potential in the 212 pups that were littered (Hurni and Ohder, 1973).

Several rat studies have investigated the teratogenic potential of formaldehyde. In one of these studies, the rats were continuously exposed to 0 to 0.8 ppm of formaldehyde for 10 days prior to mating and 10 days after mating. There was an increase in gestation time and dose-related decreased in the number of pups littered. However, the formaldehyde exposed animals littered more pups than the controls. No explanation was offered for these effects (NRC, 1981).

GENOTOXICITY: Formaldehyde has been shown to be a weak mutagen, producing gene mutations in various test systems such as *E. coli. Pseudomonas fluorescens, Neurospora crassa* and *Aspergillus nidulans* but not in the Ames test on *Salmonella typhimurium*. In Drosophila, mutations were induced in larvae fed formaldehyde-containing food and adults given injections of aqueous solutions of formaldehyde (IARC, 1987). However, exposure of adults or larvae to formaldehyde vapors has not produced mutations (NRC, 1981).

Formaldehyde has produced an increased mutation frequency in the L5178Y mouse lymphoma assay but there was no clear dose response relationship (Gosser and Butterworth, 1977).

CARCINOGENICITY: Formaldehyde has been observed to be carcinogenic in rats following inhalation of vapors for 30 hours per week, for up to 24 months at concentrations of 2, 6, and 15 ppm (2.68, 8.04, 20.1 mg/m^3). Formaldehyde failed to produce tumors in mice that were similarly exposed. Reportedly, three rats developed nasal cavity squamous cell carcinomas after 12 months of exposure to 15 ppm (20.1 mg/m^3) formaldehyde. A total of 95 nasal cavity carcinomas had been reported by the end of the 24-month exposure at the 15 ppm (20.1 mg/m^3) level and some deaths occurred. It is unclear whether the lower dose groups developed any tumors (ACGIH, 1986).

Over the last 5 years, more than 30 epidemiologic studies have evaluated the potential human carcinogenicity of formaldehyde. Studies have focused on pathologists, morticians, garment workers, chemical production plant workers, particle board manufacturing workers and residents of mobile homes. Cohort studies conducted in the U.S. (Blair et al., 1986) and Britain (Acheson et al., 1984 a, b) noted an increase in lung cancer among exposed workers, but consistent dose-response relationships were not observed. Studies by Hayes et al. (1986), Vaughan et al.(1986 a, b), Olsen et al. (1984), and Blair et al. (1987) have found an association between nasopharyngeal cancer and formaldehyde exposure. Stayner et al. (1985) observed a significant increase in cancer of the buccal cavity among garment workers. Limited, inconsistent data are available regarding formaldehyde exposure and other cancer sites, including brain, colon, and leukemia.

Agencies, scientific panels, and researchers differ in the interpretation of studies evaluating formaldehyde as a human carcinogen. OSHA concluded that data indicate an excess risk of developing nasal cancer and a possible excess risk of lung cancer among formaldehyde-exposed workers (OSHA, 1987). EPA (1987) stated that there is limited epidemiologic evidence to indicate that formaldehyde is a human carcinogen. However, the Ad Hoc Panel of Health Experts (UAREP) concluded that 1) there is no convincing evidence of a relationship between formaldehyde exposure and any single malignancy in humans, and 2) if a relationship does exist, the excess risk, in absolute terms, must be small.

EPIDEMIOLOGY: Cross-sectional studies have noted increases in skin, eye, and respiratory tract irritant symptoms among formaldehyde-exposed workers. In addition, work-related decreases in pulmonary function testing have been found (OSHA, 1987; Malaka and Kodama, 1991).

There have been more than 30 different epidemiological studies over the past 5 years that have investigated the association of

formaldehyde exposure with human cancers. Epidemiologic studies focusing on cancer are reviewed under CARCINOGENICITY.

ENVIRONMENTAL FATE: Forest fires, burning of coal, oil or wood, animal waste, microbial products of biological systems and volatile plant compounds are all sources of environmental formaldehyde. Formaldehyde can also be formed in seawater by photochemical processes, with diurnal fluctuations that show the maximum production during the early afternoon hours (HSDB, 1990).

Formaldehyde is emitted in the atmosphere as a product of combustion. It is also a product of the atmospheric oxidation of organic compounds. It is readily photolyzed in the presence of sunlight and reacts rapidly with hydroxyl radical (Finlayson-Pitts and Pitts, 1986). Atmospheric concentrations of formaldehyde in many U.S. cities range between 2 to 27 ppb with median values between 14 to 20 ppb. During photochemical smog episodes in southern California, levels between 2 and 48 ppb were recorded. Peak levels were associated with automobile traffic patterns with increases in the late morning and again in the late afternoon (HSDB, 1990).

Table 2. Concentrations of formaldehyde in foods.

Species	*Method 1*	*Method 2*
Tomato	5.7 µg/g	7.3 µg/g
Apple	17.3	22.3

ENVIRONMENTAL TOXICITY: Table 3 presents LC_{50} values for various non mammalian species:

The boiling point of formaldehyde is at or slightly below normal room temperature, thus air is a major route of transport and exposure to formaldehyde. Photolysis of formaldehyde occurs in the lower troposphere producing carbon monoxide hydrogen and hydroperoxyl radicals. The estimated half-life of formaldehyde in sunlight is on the order of 1-2 hours (Clement, 1985). Additional quantities of formaldehyde are removed from the atmosphere by dry deposition, rain or dissolving in surface waters. One model predicts a dry deposition half life of 19 hours and a wet deposition of 50 hours (HSDB, 1990). The EPA (1986) reports a half-life in air of 0.8 day.

Formaldehyde can be biodegraded to low levels within a few days by aerobic and anaerobic processes. There is little adsorption to sediments and the adsorption to soil is presumed to be low due to the low K_{ow} value. Activated sludge and sewage degrade formaldehyde in 48–72 hours (HSDB, 1990).

Formaldehyde may be present naturally in food. Analysis of fruits and vegetables by two different methods showed the following:

Cabbage	4.7	5.3
Spinach	3.3	7.3
Green onion	13.3	26.3
Carrot	6.7	10.0
White radish	3.7	4.4

(Reference: IARC, 1985)

Table 3. Ecotoxicity LC_{50} Values for Formaldehyde

Species	Exposure Time	Type	Concentration
Rainbow trout (green egg)	96 hr	S	565-700 mg/l
Rainbow trout (eyed egg)	96 hr	S	198-435 mg/l
Rainbow trout (sac larvae)	96 hr	S	89.5-112 mg/l
Rainbow trout fingerlings	96 hr	S	61.9-106 mg/l
Rainbow trout	96 hr	S	440-618 mg/l
Rainbow trout	24 hr	S	214-7,200 mg/l
Rainbow trout fingerlings	96 hr	S	123-145 mg/l
Rainbow trout (green egg)	96 hr	S	1,020 mg/l
Rainbow trout	96 hr	FT	118 µl/l

Atlantic salmon	96 hr	FT	173 µl/l
Lake trout	96 hr	FT	100 µl/l
Black bullhead	96 hr	FT	62.1 µl/l
Channel catfish	96 hr	FT	65.8 µl/l
Green sunfish	96 hr	FT	173 µl/l
Bluegill	96 hr	FT	100 µl/l
Smallmouth bass *(Micropterus dolomieui)*	96 hr	FT	136 µl/l
Largemouth bass *(Micropterus salmoides)*	96 hr	FT	143 µl/l

S = Static
FT = Flowthrough
(Taken from Verscheren, 1983)

REGULATORY STATUS: The ACGIH (1986) has given formaldehyde an A-2 classification as a suspected human carcinogen and established a TLV-TWA of 1 ppm (1.5 mg/m^3) and a STEL of 2.0 ppm. The NIOSH Recommended Exposure Limit is 0.1 ppm as a 15 minute ceiling limit (NIOSH, 1985), and the OSHA TWA-TLV is 3.0 ppm, with a 5 ppm ceiling and a 10 ppm 30 minute ceiling limit. The OSHA IDLH is listed as 100 ppm.

The EPA (IRIS, 1990) has classified formaldehyde as a B1 carcinogen, meaning it is a "probable human carcinogen based on limited evidence in humans and sufficient evidence in animals." The cancer potency factor by the inhalation route is 4.5×10^{-2} $(mg/kg\text{-}day)^{-1}$ which is comparable to the CAPCOA unit risk value as converted to a cancer potency factor. However, a recent CIIT investigation using pharmacokinetic modeling for formaldehyde/DNA adduct formation indicates that use of the exposure dose overestimates the risk of formaldehyde. It is expected that EPA will decrease the potency of formaldehyde by a factor of 25 in the near future (EHL, 1990).

Due to uncertainties in human data, the estimation of cancer risk attributable to formaldehyde needs to rely on animal data. However, an impressive array of animal data indicates a significant non-linear relationship between rodent tumors and the administered dose. There is disproportionately less formaldehyde bound to the DNA of the respiratory epithelium at low airborne concentrations. Hence, using the more accurate tissue dose value, a multistage model analysis of the nasal tumor response produces estimates considerably lower than those using airborne formaldehyde concentrations. In addition, estimate on Rhesus monkey data are at least 10 fold lower than identically exposed rats (Starr, 1990). Such newer analysis therefore seriously questions the fundamental basis, rationale and EPA potency value for carcinogenicity of formaldehyde. The EPA has not established a reference dose (RfD) for noncarcinogenic effects.

Table 4. presents data for estimating average daily intakes of formaldehyde from different media.

Table 4. Average daily intake of formaldehyde by different media.

Media	Concentration	Daily Intake
Air intake	2 - 20 ppb	50-500 µg
Energy efficient houses	day 212 ppb night 114 ppb	4500 µg
Water intake	0 ppb	0 µg
Food	insufficient data	
Tobacco		50 µg

(Reference: Hemminki and Vainio, 1984).

Formaldehyde is a hazardous substance by the Clean Water Act and a hazardous waste under RCRA. As a valuable organic compound is subject to performance standards under the Clean

Air Act. It is a permissible indirect food additive only as a component of adhesives (FDA). Formaldehyde is exempted from the requirement of a tolerance (FIFRA) on certain grains, grasses or other animal feeds (HSDB, 1990).

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TOXICOLOGY PROFILE

LEAD, INORGANIC LEAD COMPOUNDS

Synonyms: Lead metal, Lead salts

CAS No: 7439-92-1

Boiling point: 1740°C

Color: Bluish-gray

DOT designation: None

Flammable limits: An alloy of 10%-70% zirconium plus lead will ignite when struck with a hammer.

Autoignition: NA

Explosive limits: Reacts explosively with fused ammonium nitrate below 200 °C.

Flash Point: NA

Henry's law constant: NA

Melting point: 327.5°C

Molecular formula: Pb

Molecular weight: 207.19

Odor: Odorless

pH: NA

Solubility: The metal is insoluble, the solubility of the salts is pH dependent with the greater solubility at pH extremes.

water: Insoluble to slightly soluble; organic lead is water-insoluble.

K_{ow}: Variable

other: Inorganic forms are considered to be essentially insoluble. Organic forms such as tetraethyl lead are soluble in benzene and other petroleum derived solvents (gasoline).

K_{oc}: NA

Specific gravity: 11.345 g/cm^3

Vapor density: NA

Vapor pressure: 1.0 mm Hg at 980°C

Viscosity: NA

NA = not applicable
(Reference: ASTDR, 1988)

COMPOSITION: Lead (Pb) is a naturally occurring metal commonly found as an ore. It is present in all parts of the environment (i.e., plants, animals, air, lakes, oceans, soil and dust). The inorganic chemistry of lead is dominated by the divalent (+2) oxidation state.

USES: Lead is primarily used in the manufacture of storage batteries. Other uses are production of ammunition; metal products and chemicals including the gasoline additives tetraethyl lead and tetramethyl lead.

ACUTE TOXICITY: Acute toxicity in animals varies with route of administration, moiety and species. Generally, organic acids such as oleate and naphthenate, reduce toxicity; however, acetate increases toxicity (Clayton and Clayton, 1981). Recent literature reviews have presented TD_{10} and LD10 values rather than LD_{50} values. Sax and Lewis (1989) define LD10 as the lowest aggregate fatal dose of a substance which can produce death given repeatedly in one or more doses.

The TD_{10} and LD10 values for several lead compounds are given in Table 1. The values for organic lead compounds are listed to illustrate the relative difference of toxicity between inorganic and organic lead compounds.

Ingestion: Lead poisoning usually occurs slowly, resulting from gradual accumulation of lead. Similar systemic effects occur whether exposure is via inhalation or ingestion. The progressive signs and symptoms of intoxication are loss of appetite, fatigue, malaise, insomnia, hypotension, headache, irritability, painful joints and muscles, flaccid paralysis without anesthesia, hallucination, gastritis and hepatic changes (Sax & Lewis, 1989). All salts of inorganic lead will cause symptoms of lead poisoning. Gastroenteric symptoms predominate after ingestion (Deichmann and Gerarde, 1969).

Among infants and children, the primary routes of exposure to lead are via ingestion of contaminated food, water, soil, and paint chips. Infants and children absorb more of the lead they ingest than do adults, and are particularly susceptible to the neurotoxic effects of lead. The most profound neurotoxic effect is encephalopathy which may follow acute, high level exposure or low level exposure over days, weeks, or months. Blood lead levels in children with encephalopathy are typically greater than 80 µg/dl. Adults are more resistant to the neurotoxic effects of lead and encephalopathy may develop with blood lead levels greater than 120 µg/dl (EPA, 1986). Acute hemolytic anemia and hemoglobinuria may occur along with direct kidney damage. The disease may progress rapidly, with convulsions, coma, and death within 48 hours. Survivors may be left with severe, irreversible brain damage (EPA, 1986). In instances of acute high level exposures, death may occur within 1 to 2 days following a large dose, and chronic toxic signs can appear if the individual survives (Goodman & Gilman, 1985).

Table 1. Acute Toxicity Values for Some Lead Compounds

Compound	Species	Determination	Value
Lead	oral human	TD_{10}	450 mg/kg
	inhalation human	TC_{10}	10 µg/m³
	i.p. rat	LD10	1000 mg/kg
	oral pigeon	LD10	160 mg/kg
Lead Acetate	oral dog	LD10	300 mg/kg
	i.v. hamster	TD_{10}	50 mg/kg
Lead Arsenate	oral rat	LD10	100 mg/kg
Lead Chloride	oral guinea pig	LD10	1500 mg/kg
		TD_{10}	
Lead Chromate	i.p. guinea pig	LD_{50}	400 mg/kg
Lead Fluoride	oral guinea pig	LD10	4000 mg/kg
Lead Nitrate	oral guinea pig	LD10	500 mg/kg
Lead Naphthenate	oral rat	LD10	5100 mg/kg
Lead Oleate	oral guinea pig	LD10	8000 mg/kg
Lead Oxide Red	oral guinea pig	LD10	1000 mg/kg
	i.p. guinea pig	LD_{50}	220 mg/kg
Lead Sulfate	oral dog	LD10	2000 mg/kg
	oral guinea pig	LD10	30000 mg/kg
Tetraethyl Lead	oral rabbit	LD10	30 mg/kg
	oral rat	LD_{50}	24 mg/kg
Tetramethyl Lead	oral rabbit	LD_{50}	24 mg/kg
	oral rat	LD_{50}	105 mg/kg

(References: Sax and Lewis, 1989; Clayton and Clayton, 1981)

Although children ingest less material than adults, they are more likely to ingest lead from high lead sources such as paint chips. As a result, they may have 2-3 times the exposure on a weight base than adults, with over 25% of a child's lead body burden in a soft tissue as compared to 5% for adults (EPA, 1984).

Other effects associated with lower blood lead levels are presented under CHRONIC TOXICITY. Effects vary with internal absorbed dose (as measured by blood lead) rather than with the duration or route of exposure. Therefore, at a given blood lead level, similar signs and symptoms appear regardless of the manner of exposure.

Inhalation: Severe poisoning is produced by exposure to fumes from lead furnaces and dust from drossing. Most inhalation studies involve dust or the fumes of insoluble lead oxide, sulfide or chromate. There is evidence that lead fume is less harmful than the dust of relatively soluble lead compounds.

Signs and symptoms of intoxication generally develop more rapidly from inhalation exposures than from an equivalent oral exposure (Sax and Lewis, 1989). Symptoms of sudden large exposures will disappear if the individual is removed from exposure (Kehoe, 1972). Older data indicates that signs and symptoms of lead intoxication via inhalation will not occur at levels below 0.2-0.15 mg/m³ (Johnstone & Miller, 1960). However, biochemical marker techniques show increased urinary coproporphyrin, stippling of blood cells and anemia with lead exposure levels of 0.12-0.14 mg/m³ (Tsuchiya and Harashima, 1965). In 1971 the TLV of lead was reduced from 0.2 mg/m³ to 0.15 mg/m³ in light of the improved biochemical marker methodologies.

Skin contact: Skin absorption of lead salts is poor because of their polarity. The acute

dermal toxicity of inorganic lead compounds may be considered to be greater than 2.5 g/kg. No data was located addressing primary skin irritation of inorganic lead.

Organolead compounds, however, can be absorbed through the skin (Deichmann and Gerarde, 1969). The dermal LD10 for tetraethyl lead in rabbits and guinea pigs is 830 mg/kg and 995 mg/kg, respectively. The dermal LD10 for tetramethyl lead for rabbits is 3391 mg/kg (Sax and Lewis, 1989).

Eye contact: Metallic lead metal foreign bodies in the eye or orbit in humans cause little reaction and rarely any toxic effect. Clinical experiences with various intraocular foreign bodies presented in detail with histologic studies indicated that metallic lead bodies caused minimal inflammatory reaction. A case is described in the literature where a small lead shot was allowed to remain in the vitreous humor, the vision returned to normal as blood in the vitreous absorbed lead or the shot settled in the course of a year. In one exceptional case, a patient with a lead shot behind one eye had impaired vision. This was assumed to be due to a toxic effect of lead. Whether this interpretation was correct or not, a significant improvement of vision was reported when systemic and topical treatment with 2, 3-dimercaptopropanesulfonate sodium was started 5 years after the injury (Grant, 1986).

CHRONIC TOXICITY: There is no clear distinction between acute and chronic symptoms. Acute effects can be delayed as long as 45 days (Gosselin, 1984).

Chronic lead poisoning starts with symptoms such as anorexia, muscle pain, malaise, headaches and a persistent metallic taste. Constipation or diarrhea may also occur. As intoxication progresses, intestinal spasms develop causing severe abdominal pain with anorexia and constipation becoming more apparent (Goodman and Gilman, 1985).

Chronic neuromuscular symptoms include weakness progressing to paralysis. The most active muscle groups are the most noticeably impaired, however, there is no indication of sensory involvement. Degenerative changes in motor neurons and their axons have been observed and may account for this effect (Goodman and Gilman, 1985).

Ingestion: Children may develop lead poisoning following chronic ingestion of lead-contaminated food, water, soil, or paint chips (Sax and Lewis, 1989). Encephalopathy may occur when blood lead levels exceed 80 µg/dl, with death reported at levels at and above 125 µg/dl.

Children with lead levels between 50-80 µg/dl can develop anemia, peripheral neuropathies, colic, other gastrointestinal problems, and chronic renal disease (ATSDR, 1988). Other effects such as learning and developmental delays, decreases in IQ scores, and reductions in growth are associated with blood lead levels below 40 µg/dl. Neurobehavioral effects and alterations in heme synthesis in children may occur at very low exposure levels, possibly at and below 15 µg/dl (ATSDR, 1988).

Adults may also develop adverse effects from chronic ingestion of lead-contaminated food and water. Signs and symptoms include gastrointestinal complaints (constipation, colicky pain), hypochromic, normocytic anemia with stippling of red blood cells, reductions in peripheral nerve conduction times, and mild central nervous system effects such as poor concentration, forgetfulness, depression. Effects may progress, with the development of wrist drop and foot drop.

Inhalation: Inhalation of lead is the principal route of exposure for occupational groups (ATSDR, 1988). For the general public exposure is approximately 50/50 between ingestion and inhalation (Sax & Lewis, 1989). Signs and symptoms of early lead toxicity are similar to those seen with ingestion. Reductions in peripheral nerve conduction times. Hematopoietic and peripheral nervous system effects such as reduced conduction times are associated with blood lead levels as low as 30-50 µg/dl, with no clear threshold evident (EPA, 1986). Peripheral motor nerve weakness with wrist drop and foot drop may develop with continued exposure to lead. Hypertension has been associated with chronic exposure among middle-aged males.

Chronic renal effects (chronic nephritis) have been reported among workers historically exposed to high levels of lead.

Skin contact: See ACUTE TOXICITY, Skin contact.

Eye Contact: See ACUTE TOXICITY, Eye contact.

SENSITIZATION: No information was found addressing the sensitization potential of inorganic lead compounds.

TARGET ORGAN EFFECTS: The central and peripheral nervous systems, the hematopoietic system, the kidney, the cardiovascular system,

and the male reproductive tract are the major target organ/systems for lead.

Hematopoietic Effects: Lead induces anemia by interfering with heme synthesis and reducing red blood cell life span. Lead directly interferes with heme synthesis by affecting the activity of three enzymes: delta-aminolevulinic acid synthetase (ALA-S); delta-aminolevulinic acid dehydratase (ALA-D); and ferrochelatase (Klaassen, et al., 1986). Inhibition of cytosolic ALA-D results in decreased production of porphobilinogen from ALA. Meanwhile lead indirectly stimulates mitochondrial ALA-S to form ALA, resulting in accumulation of ALA and inhibiting ALA-S activity via feedback depression. Lead also inhibits mitochondrial ferrochelatase, which catalyzes the incorporation of iron into the protoporphyrin to form heme. The end result is an accumulation of ALA and protoporphyrin IX in the circulating erythrocyte. As the cells circulate, zinc is chelated into the protoporphyrin ring to make zinc protoporphyrin (ZPP) (Moore and Goldberg, 1985).

The life span of erythrocytes is shortened by lead exposure. Although the mechanism is unknown, lead reduces the stability of the erythrocyte membrane. Inhibition of sodium and potassium dependent ATP-ases accompanies this effect (Hernberg, et al., 1967). Inhibition of pyrimidine-5-nucleotidase by lead results in accumulation of pyrimidine nucleotidase which alters cellular energetics and possibly reducing membrane stability (Angle, 1982).

Coulton showed decreased ALA-D activity in an unpublished controlled human inhalation study (ATSDR, 1988). Adult male volunteers were exposed to 3.2 or 10.9 µg Pb/m^3 for 23 hours per day for 3-4 months. Mean blood levels increased from 20 µg/dl to 26 µg/dl in the low dose group and from 19 µg/dl to 47 µg/dl in the high dose group. ALA-D activity was reduced to 15% of pre-exposure level in the low-dose group, while ALA-D activity in the high dose group was reduced to 44% of pre-exposed levels after 5 weeks of exposure. The source of this information is ambiguous as to whether both dose groups were measured at 5 weeks, or if only the high dose group was measured at 5 weeks (ATSDR, 1988).

Numerous studies show a correlation between blood lead levels and blood/urine ALA levels in industrially exposed workers, although the slope of the dose-response curve varies from study to study (ATSDR, 1988). Using erythrocyte protoporphyrin (EP) or zinc protoporphyrin (ZPP) as an indicator of lead exposure, the blood lead threshold in males for increasing EP/ZPP ranges 25-30 µg/dl (EPA, 1986). Women are somewhat more sensitive, with a threshold ranging from 15-20 µg/dl, regardless of whether the lead was ingested or inhaled (ATSDR, 1988). Urinary coproporphyrin has been used as an indicator of over exposure to lead, with 40 µg/dl as the lowest observed effect level (EPA, 1986). Based upon review of prior studies the EPA has determined that a blood level of 50 µg/dl is the threshold at which hemoglobin levels decrease, with the LOEL for frank anemia being at 80 µg/dl.

There are data showing that lead inhibits the formation of heme-containing cytochrome P-450 in children (Saenger, 1984). Children testing positive for lead exposure had significantly lower hydroxylation rates of cortisol, a reaction mediated by cytochrome P-450.

Renal Effects: Lead causes two primary forms of renal injury: reversible tubular disorder and irreversible interstitial nephropathy. Reversible renal tubular disorder occurs after acute exposure and is frequently observed in children, while irreversible interstitial nephropathy is most often observed in chronically exposed workers.

Lead nephrotoxicity impairs amino acid glucose and phosphate resorption, producing a Fanconi-like syndrome. Characteristic histopathology shows nonspecific interstitial fibrosis, internuclear inclusion bodies, tubular degeneration and glomerular and vascular changes in small arteries and arterioles (Morgan, 1966). Several case reports of renal adenocarcinoma in lead workers have been published (Goodman and Gilman, 1985).

Renal effects in rats are similar to those observed in humans. Effects observed include intranuclear inclusion bodies, mitochondrial swelling, impaired oxidative phosphorylation, and aminoaciduria. Rats, however, develop adenocarcinoma (Goyer, 1971) which has not been observed in humans.

Cardiovascular Effects: Lead has been shown to affect the cardiovascular system, producing high blood pressure, cardiac lesions and abnormal electrocardiographs (Kirkby and Gyntelberg, 1985). These findings have been limited to high level exposures of males in industrial settings. Although there was no apparent blood lead threshold for cardiovascular effects, controlling all potential contributing factors shows lead may have been responsible for a 1-2% increases in blood pressure (EPA, 1986). Lead causes increases in blood pressure in animals and is consistent with human data (ATSDR, 1988).

In addition, lead has been shown to impair the immune system (Kimber et al., 1986), interfere with vitamin D metabolism (Rosen and Chesney, 1983) and may retard growth (Johnson and Tenuta, 1979).

ABSORPTION-METABOLISM-EXCRETION: There are no data showing whether deposited lead particles accumulate in the lungs. All lead compounds deposited in the lungs are either absorbed or transported to the G.I. tract. The absorption rate of the deposited lead depends upon the physicochemical form of the lead (NRCC, 1973).

Only a very minor fraction of particles over 0.5 μm in mean maximal external diameter are retained in the lung. Most are cleared from the respiratory tract or swallowed. The percentage of particles less than 0.5 μm retained in the lung increases with reduction in particle size. About 90% of lead particles in ambient air that are deposited in the lung are small enough to be retained. Absorption of retained lead through alveoli is relatively efficient and complete.

Deposition rate is proportional to particulate diameter and ventilation rate, ranging from 5-63.2% for lead bearing particles (Kehoe, 1961). Volunteers inhaling lead particles (0.2-2 μm) in urban areas were observed to have deposition rates of about 50%. The same authors concluded the average deposition rate ranges from 30-50% (Friberg, 1986). There are differential deposition rates within the respiratory tract. Less than 8% of ^{212}Pb absorbed on aerosols was deposited in the tracheo-bronchial tree. The deposition rate for the entire respiratory tract ranged from 14-45% (Hursh, et al., 1969). The mucociliary mechanism removes a significant proportion of the deposited particles.

Approximately 10% of ingested lead is absorbed and is dependent upon the individual's age and fasting state (Kehoe, 1961). Children up to 8 years old may absorb up to 53% (Alexander et al., 1973), while fasting can increase absorption to 60% of the ingested lead at doses ranging from 4-400 μg Pb (Flanagan and Chamberlain, 1982). Ingestion of calcium and phosphate at background levels was found to inhibit lead absorption six-fold (Heard, 1982). Animal studies show lead absorption to average about 5-10% with younger animals absorbing up to 50% or more of trace doses administered (Schlipkoter and Pott, 1973).

Absorbed lead is transported by the blood, bound primarily to erythrocytes. Lead distributes into two compartments; an exchangeable compartment consisting of the blood and soft tissues and a storage compartment (bone). Individuals exposed to low levels store 90% of lead body burden in bones. Lead accumulates in bone throughout life, while its concentration in soft tissues stabilizes early in adulthood (Friberg, 1986). Unless mobilized, bone lead does not contribute to a toxicity syndrome (Goodman and Gilman, 1985).

After absorption, lead is distributed to the soft tissues, particularly to the liver and renal tubular epithelium. Lead is then later redistributed to bone, teeth and hair. However, some lead is accumulated in the brain, primarily in the gray matter and basal ganglia (Goodman and Gilman, 1985).

Blood lead levels are indicative of recent lead exposure. Ambient air lead levels ranging from 1-5 $\mu g/m^3$ show a direct correlation between air lead levels ($\mu g/m^3$) and blood lead levels ($\mu g/dl$) (WHO, 1977).

Approximately 90% of ingested lead is eliminated unabsorbed via the feces (Kehoe, 1961). From the absorbed lead, about 76% is eliminated in the urine, 16% by gastro-intestinal secretions and less than 8% by hair, nails and sweat (Rabinowitz, 1973). Lead excretion varies substantially among species. In primates, excretion is predominantly via the urine. In the rat and sheep, biliary and transmucosal excretion predominates (Castellino, 1966).

Biological half-life for lead in human bone is about ten years (EPA, 1979), while the blood lead half-life is approximately 28-36 days (Rabinowitz, 1976).

NEUROTOXICITY: Lead effects the peripheral nervous system, producing reductions in motor neuron function. The extensor muscles of the wrist are most commonly affected, resulting in wrist drop. Foot drop may also be observed. Lead also effects the central nervous system, producing decrements in concentration and memory. Children are particularly susceptible to the central nervous effects of lead, and may present with life-threatening encephalopathy. Learning and developmental delays have been documented among children chronically exposed to low levels of environmental lead.

Neurologic effect in young children include developmental effects lowered IQ and behavioral abnormalities without overt signs or symptoms. As exposure increases non-specific signs may be observed with encephalopathy, increasing the risk of permanent mental retardation, motor deficits and possibly death. The pathogenesis of lead encephalopathy is vague. Lead produces obvious changes in

cerebral spinal fluid dynamics and cellular hypoxia but also directly affects neurotransmission at asymptomatic blood lead levels of 30-50 µg/dl (Klaassen, 1986).

Based upon review of the data, the EPA has concluded that the Lowest Observed Effect Levels (LOELs) for neurological signs and symptoms in adults ranges from 40-60 µg/dl, with neurological signs and symptoms occurring at the same blood lead level as other conspicuous signs and symptoms of lead intoxication (EPA, 1986).

Studies have shown that the threshold for neurobehavioral effects is approximately 30 µg/dl. Pronounced effects ranging from disturbances in reaction time, visual motor performance, hand dexterity, cognitive performance, memory and IQ ratings were observed in individuals with blood levels ranging from 40-80 µg/dl. Exposure to lead also affects mood, causing depression, confusion, anger, nervousness, fatigue and tension (ATSDR, 1988).

Children are more susceptible to lead induced neurological effects than adults as indicted by responses at lower blood levels (ATSDR, 1988). Children exposed to lead have shown biphasic mental development, characterized by normal development during the first year to year and one half and followed by a steady decrease in speech and motor skills. Hyperkinetic, convulsive and aggressive behavior disorders have been observed as well. Hyperkinetic behavior and a significant decrease in IQ have been correlated to blood lead levels of 30-50 µg/dl (Goodman and Gilman, 1985). Landrigan et al., (1975) has also observed a correlation between blood lead levels and decreased intelligence and psychomotor skills (Landrigan et al., 1975).

Children's exposure to lead is largely via ingestion (ASTDR, 1988). Chisolm et al., (1956) observed, signs of encephalopathy (hyperirritability, ataxia, convulsions, stupor and coma) at blood lead levels ranging from 90-700 µg/dl, with the mean value at which death occurred being 327 µg/dL. Subsequent studies have shown that children most susceptible may develop encephalopathy at blood lead levels ranging from 80-100 µg/dL (EPA, 1986).

However, children may appear to be asymptomatic of encephalopathic effects, yet develop neurological effects. Based on numerous studies, the EPA has determined that asymptomatic children with blood lead levels ranging from 40-80 µg/dl consistently score lower on IQ cognitive function tests (EPA, 1986).

Neurobehavioral effects in animals approximate those observed in humans at roughly the same blood lead levels, although neuropathy is inconsistent between species (ATSDR, 1988). Blood lead levels in rats ranging from 15-20 µg/dl were associated with learning impairment (Cory-Slechta, et al., 1985). Similar experiments in monkeys produced effects analogous to those observed in human studies with a comparable dose-response relationship (ATSDR, 1988). Exposure to lead during the first year (mean blood level = 32 µg/dl) produced neurobehavioral effects that persisted through the fourth to fifth year (Bushnell and Bowman, 1979). Lead also affects the visual cortex, suprageniculate pathway, optic nerve and the ocular muscles (Grant, 1980).

Peripheral neuropathy from lead exposure is characterized by segmental demyelination and Schwann cell degeneration (Lampert and Schochet, 1968). Occasionally Wallerian degeneration of posterior roots of the sciatic and tibial nerves is observed, with motor nerves being much more sensitive than sensory nerves (Schlaepfer, 1969). Nerve conduction velocities have been shown to be affected at blood lead levels ranging from 50-70 µg/dl (Seppalainen, 1975).

REPRODUCTIVE TOXICITY: Several reproduction studies of the effects of subchronic oral exposure to lead in rats have been conducted (Grant *et al.*, 1980; Fowler *et al.*, 1980). These studies report that lead acetate administered in drinking water at various concentrations caused depressed body weights at 50 and 250 mg-Pb/l water, histological changes in the kidneys of offspring, cytokaryomegaly of the tubular epithelial cells of the inner cortex at concentrations greater than or equal to 25 mg/l and postnatal development delays at 50 to 250 mg/l. The reported LOAEL (Lowest-observable-adverse-effect-level) of 5 mg/l corresponds to a dose of 0.5 mg/kg/day.

Chronic oral exposure of female Long-Evans rats to lead (5 mg/Pb/l-water) reportedly resulted in slight effects on tissue excitability, systolic blood pressure, and cardiac ATP concentrations (Kopp et al., 1980a,b). This reported LOAEL corresponds to a dose of 0.5 mg/kg/day.

In utero exposure to lead in squirrel monkeys produced lesions analogous to human lead encephalopathy and growth retardation of the fetal cerebrum. Neurologic and behavioral symptoms persisted into adulthood (Logdberg et al., 1987). Orally administered lead in drinking water at levels of 50 to 250 mg/l reportedly resulted in postnatal developmental delays

(Kimmel et al., 1976). Higher oral doses of lead may result in decreased fertility and fetotoxic effects in a variety of species (Hilderbrand et al., 1973). A reduction in the number of offspring of rats and mice exposed to 25 mg Pb/l drinking water with a chromium deficient diet was reported by Schroeder et al. (1970).

Lead has been show to cross the placental barrier (Task Group on Metal Accumulation, 1973), with a correlation between maternal and fetal blood lead concentrations (Hower, 1975). Because there is no physiologic or metabolic barrier to placental uptake of lead by the fetus, maternal exposure results in fetal uptake. Furthermore, pregnancy induced stress may result in maternal bone lead release and compound fetal lead exposure.

Chronic lead ingestion caused abortion, miscarriage and reversible sterility in sheep (IARC, 1980).

Lead has been shown to have a direct adverse effect on gonads. A comparison of median sperm count between battery workers and cement workers showed a significantly lower ($p<0.025$) count in the battery workers. This correlated with significant elevations ($p<0.001$) of blood lead, urinary lead, semen lead and zinc protoporphyrin concentrations (Assennato, 1987).

Blood lead levels of 42 µg/100 ml have been shown to cause sexual dynamics disorders while concentrations greater than 52 µg/100 ml have impaired spermatogenesis (Lancranjan et al., 1975).

GENOTOXICITY: *In vitro* data shows that lead acetate induces cell transformation in Syrian hamster embryo cells (DiPaolo, et al. 1978). Other data show lead compounds are capable of producing chromosomal aberrations *in vivo* and *in vitro* (Grandjean, 1983). This supports data showing increased chromosomal defects in workers with blood lead levels above 60 µg/dl (Klaassen, 1986).

CARCINOGENICITY: The EPA concluded that the available epidemiologic literature provides no definitive evidence that there is a relationship between lead exposure and cancer (EPA, 1986). Thus, the EPA considers the evidence for lead and cancer in humans to be inadequate as does IARC (1987).

However, the EPA has determined that there is sufficient evidence to show lead is carcinogenic in animals (IRIS, 1990). More than 10 investigators using rats or mice have shown that lead salts, whether ingested or injected, are carcinogenic. The lead salts produced characteristic bilateral renal carcinoma. Lead subacetate produces lung adenomas in mice. No inhalation studies examining the carcinogenic potential of lead have been located.

EPIDEMIOLOGY: Much recent epidemiologic research has focused on the effects of lead on the growth and development of infants and children. Adverse effects on childhood growth and neurobehavioral development have been documented at blood lead levels in the 15-30 µg/dl range (EPA, 1986). A linear relationship between IQ and blood lead over the range of 6-47 µg/dl has been found among low income minority children (EPA, 1986).

Perinatal lead exposure (as determined by umbilical cord blood lead levels) and postnatal development have been evaluated in prospective studies. Infant mental development, length of gestation, and possibly other aspects of fetal development may be affected at cord blood levels of 10-15 µg/dl (EPA, 1986).

Data derived from the British Regional Heart Study and the U.S. NHANES II Study demonstrated an association between blood lead levels and increased blood pressure among middle-aged men (EPA, 1986). Similar findings have been reported in case series examining blood pressure among individuals occupationally exposed to lead (EPA, 1986). In a large cohort mortality study (Cooper et al. 1985). noted an increase in mortality due to chronic nephritis and hypertensive disease among workers heavily exposed to lead.

Epidemiologic studies have investigated cancer mortality patterns among lead battery workers, lead production workers, and lead-exposed smelter workers. Due to limitations in exposure information and data on confounding exposures (including smoking), EPA has concluded that little evidence regarding lead exposure and cancer in humans is available (EPA, 1986).

ENVIRONMENTAL TOXICITY: Lead has been shown to accumulate in freshwater fish and freshwater and marine invertebrates. In fish, lead accumulates in the epidermal mucous, thus limiting human exposure (EPA, 1986). The 50% lethal concentration (LC_{50}) of lead in several aquatic species is shown in the following table.

Table 2. Aquatic LC_{50} Values of Lead in Some Invertebrates.

Organism	LC_{50} (mg/l)
Oyster	2.45
Mussel	<40
Water Flea	0.3-5.89
Crayfish	0.9-3.3
Rainbow Trout	0.2-2.3

(Reference: WHO, 1989)

Although most plants do not absorb inorganic lead from the soil, some trees have the ability to accumulate large quantities of lead from contaminated soil. Most lead in plants is probably the result of atmospheric deposition on foliage (WHO, 1989).

ENVIRONMENTAL FATE: Lead is a naturally occurring element introduced into the atmosphere, soil and water by natural and human sources. Lead used in gasoline, would be discharged into the air as a component of automobile exhaust. It is also emitted to the air from iron and steel production facilities, lead acid battery manufacturing plants and the burning, weathering and welding of lead painted surfaces. It is found in drinking water from the leaching of lead pipes and connectors; in surface water from atmospheric dust and industrial waste water from lead manufacturing. Once deposited into water, lead adsorbs to sediment and little leaching occurs.

Lead is found in soil by deposition of atmospheric dust, waste disposal at land fill sites, depositing of paint chips and the spreading of fertilizers composed of sewage sludge.

When metallic lead is exposed to the atmosphere, oxidation of the surface produces a crust that resists corrosion. Metallic lead thus has limited environmental mobility. Most inorganic lead salts are also insoluble or virtually insoluble in water and they too have limited environmental mobility. Lead salts that have water solubility readily combine with carbonate or sulfate ions to form insoluble salts, or are adsorbed by ferric hydroxide (EPA, 1986). The solubility of lead salts is affected by pH and salinity. Dissolution is minimal at or near neutral pH and increases at higher or lower pH values (HSDB, 1990).

Under appropriate conditions sediment microorganisms may directly methylate specific inorganic lead compounds (EPA, 1986).

REGULATORY STATUS: Presently inorganic lead is classified as a Group B2 carcinogen based on eleven positive animal studies showing significant increases in renal tumors from ingestion or subcutaneous exposure to inorganic lead salts. However, human epidemiological data are insufficient to refute or prove inorganic lead is a human carcinogen.

Presently the EPA is considering changing the lead maximum contaminant level (MCL) from 10 ppb at the tap to 5 ppb at the plant. This proposal was published in the August 18, 1988 Federal Register and subsequently presented to the Health Science and Standards subcommittee of the National Drinking Water Advisory Council on August 24, 1989. The subcommittee rejected the proposal, claiming more data was needed showing that the former standard was inadequate. Thus the standard of 10 ppb is still in effect.

The tolerable intake of lead for preschool children should be less than 3 mg/week (WHO, 1977).

Table 3. Worker Exposure Limit Values for Lead

ACGIH TWA:	0.15 mg (Pb)/m^3
STEL:	NA
Ceiling:	NA
OSHA-PEL:	TWA 0.05 mg (Pb)/m^3
IDLH:	NA
NIOSH TWA:	<0.1 µg/m^3
MSHA:	NA

NA = not available
(References: ACGIH, 1989; NIOSH, 1985)

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TOXICOLOGY PROFILE

MANGANESE

Synonyms: Mangan, tronamang, magnacat

CAS No: 7439-96-5

Boiling point: 1962°C

Color: Reddish-grey or silvery; exists in 4 allotropic forms.
DOT designation: Combustible material, irritant.

Explosive limits: The minimum explosive concentration is 125 oz./1000 ft^3.

Flammable limits: Moderate flammability potential when exposed to flame. Autooxidizes when exposed to air.

Autoignition: 450°C for dust clouds

Flash point: NA

Henry's law constant: NA

Melting point temp.: 1244 °C

Molecular formula: Mn

Molecular weight: 54.94

Odor: None

Solubility:
water: Reacts slowly in hot or cold water.

K_{ow}: NA

other: Elemental Manganese is soluble in dilute acids.

K_{oc}: NA

Specific gravity: 7.26 - 7.47 depending on the allotrope

Vapor density: NA

Vapor pressure: 1 mm Hg at 1292°C

NA = not available
(Reference: Merck, 1989; HSDB, 1990).

COMPOSITION: Manganese occurs in a great variety of minerals widely scattered over the earth. It is a steel gray, lustrous, hard, brittle, highly reactive metal which exists in four allotropic forms: alpha, beta, gamma, and delta. The common valence states are 2, 4 and 7 while the more rare valence states are 1,3,5 and

6. Manganese will react with water or steam to produce hydrogen. Manganese metal is produced by acid leaching of the ore, precipitation or electrolysis processes. Manganese colors glass an amethyst color, and is responsible for color of true amethyst.

USES: The principal use of manganese is in metallurgy, which accounts for 95% of U.S. demand (Reidies, 1981; DeHuff and Jones, 1981). The majority of metallurgical use is in steel production as a reagent to reduce oxygen and sulphur. A variety of compounds of manganese are used in the chemical industry and battery manufacture. It is also used to produce extremely bright flares and lighting devices, and electrode coating in welding rods. Several manganese salts are used as driers for linseed oil, glass, and textile bleaching, and in dyeing, tanning of leather and also in fertilizers (HSDB, 1990). Some of the major uses of different manganese compounds are listed in Table 1.

Table 1. Major Uses of Selected Manganese Compounds

PRODUCT	FORMULA	USE
Electrolytic *manganese dioxide*	MnO_2	Dry-cell batteries ferrites
High purity manganese oxide	MnO	High-quality ferrites, ceramics; intermediate for high purity salts
60% manganese oxide	MnO	Fertilizer; feed additive intermediate for electrolytic manganese metal and dioxide
Manganese Sulfate	$MnSO_4$	Feed additive; fertilizer intermediate for many products
Manganese chloride	$MnCl_2$	Metallurgy; MMT synthesis brick colorant; dye dry cell batteries
Potassium permanganate	$KMnO_4$	Oxidant; catalyst; intermediate; water and air purifier
Methylcyclopentadienyl manganese tricarbonyl (MMT)	$CH_3C_5H_4Mn(CO)_3$	Fuel additive

(Reference: HSDB,1990).

ACUTE TOXICITY: The acute toxicity for manganese is greater for soluble compounds via the parenteral route. Divalent manganese (Mn^{2+}) is considered to be almost three times more toxic than the trivalent form. Although human toxicity occurs by inhalation of the dust or fumes, acute poisoning by manganese in humans is very rare (Clayton and Clayton, 1981).

Table 2. LD Values of Manganese for Different Mammalian Species.

Species/route of administration	Type	Test Concentration
oral - rat	LD_{50}	6750 mg/kg
rat	LD_{50}	4500 mg/kg
i.p. mouse	LD_{50}	53 mg/kg
ihl. - man	TC_{10}	2300 $\mu g/m^3$

(Reference: Sax and Lewis, 1989).

Ingestion: Very few poisonings have occurred from ingestion since the inorganic salts are poorly absorbed from the G.I. tract (HSDB, 1990). Gastric mucosal damage with resultant pyloric stenosis has been reported following ingestion of 3 grams of potassium permanganate. Fatal methemoglobinemia has also been associated with ingestion of this compound (EPA, 1984).

Inhalation: Inhalation of fumes with high concentrations of manganese and its oxides have been reported to cause metal fume fever. Symptoms include chills and fever, upset stomach, vomiting, dryness of the throat/cough, weakness, and aching of the head and body. These symptoms occur several hours after exposure and last for only a day or two (Browning, 1969; EPA, 1984).

Inhalation studies of pulmonary effects in animals show acute respiratory effects when the level of exposure exceeds 20 mg/m^3 of MnO_2 (HSDB, 1990).

Intratracheal administration of manganese dust or solutions to experimental animals produces an acute pneumonitis. Cytological examination of rats treated in this fashion showed shredded epithelial cells with intense mononuclear infiltrations of the alveoli and alveolar walls. In addition, there were adverse changes in the bronchial and alveolar epithelium of the rats treated in this fashion (Browning, 1969).

Skin contact: Absorption of manganese through the skin is negligible (ILO, 1983).

Eye contact: Mild irritation of the eyes has been noted in rodents and rabbits exposed to 500 mg Mn/24 h (CHEMINFO, 1990).

CHRONIC TOXICITY: Most of the toxicities reported for manganese are attributable to chronic exposures. The symptoms are generally manifested within 6 months to a year and consist of neurologic or respiratory abnormalities although exposure to heavy concentrations for as little as 3 months may also produce the condition (HSDB, 1990).

Ingestion: Manganese toxicity from excessive ingestion has not been reported except where industrial contamination has occurred (HSDB, 1990).

Repeated oral administration of manganese metal to animals for prolonged periods gave no evidence of adverse effects at moderate doses. At 100 ppm, Mn in the diet stimulated growth but at 600 ppm it was judged deleterious (Clayton and Clayton, 1981).

Inhalation: Chronic manganese poisoning is a clearly characterized disease which results mostly from the repeated inhalation of fumes or dust of manganese. The condition of "manganism" usually develops after 1-3 years of exposure. This disease involves primarily the CNS and is manifested by psychic and neurological disorders (Sax, 1989). Early symptoms include apathy, anorexia, languor, sleepiness, weakness in the legs with a spastic gait and a tendency to fall. A "Manganese Psychosis" may also be manifested at a latter stage where there is a stolid mask-like appearance of the face, salivation, speech disturbances such as slow and difficult articulation, incoherence or possibly muteness, excessive sweating, unaccountable and spasmodic laughter, impulsiveness, and sometimes sexual excitement followed by impotence (Clayton and Clayton, 1981). Chronic manganese poisoning is not fatal but the patient is disabled unless treated (Clayton and Clayton, 1981).

In experimental studies, rats received intratracheal injections of manganese dioxide and $MnCl_2$ to simulate the manganese pneumonitis seen in man. Characteristic histological changes in the lungs were produced. The $MnCl_2$ caused intense congestion and pulmonary edema which was often fatal (ACGIH, 1986).

Inhalation exposures of rabbits to MnO_2 dust 4 hours/day for 3 to 6 months at levels of 10 to 20 mg/m^3 resulted in decreased hemoglobin and erythrocytes in the blood (ACGIH, 1990).

Manganese compounds are common air contaminants. Exposure to dusts and fumes can possibly increase the incidence of upper respiratory infections and pneumonia (HSDB, 1990).

Skin contact: Dermal application of manganese causes a mild irritation in rodent/rabbits exposed to 500 mg/24 hr in the Standard Draize test (CHEMINFO, 1990).

Eye Contact: When the condition of "manganism" is manifested, nystagmus, oculogyric crisis or loss of Bell's phenomenon does not occur despite the Parkinsonism. With

the development of Manganese Psychosis, there is decreased movement of the eye lids and eyes but there is no paresis (Grant, 1986). Also see "Eye contact" under Acute Toxicity.

SENSITIZATION: No information on the sensitization potential of manganese was found in the literature.

TARGET ORGAN EFFECTS: The usual form of chronic manganese poisoning primarily involves the CNS (HSDB, 1990).

Degenerative changes and demyelination were seen histologically in the optic chiasma and spinal cord, particularly the posterior columns, in a monkey administered manganese aerosol. The Purkinje and granular layer of the cerebellum also showed some damage (Browning, 1969).

ABSORPTION METABOLISM EXCRETION:
Absorbed manganese is rapidly eliminated from blood and distributed mainly to liver, kidneys and endocrine glands, after parenteral and oral exposure, as well as inhalation. Minor amounts go to brain and bone as shown in mice, rats and monkeys. It preferentially accumulated in tissues rich in mitochondria (Friberg, 1986).

Manganese is widely distributed within the human and animal body in concentrations which are characteristic for individual tissues. The highest values of manganese in humans are found in liver , kidney and endocrine glands. Manganese has been shown to penetrate the blood-brain and placental barriers (HSDB, 1990). Manganese elimination from the body is accomplished mainly via feces with 20 to 25 % of the dose being excreted within 2 hours (Gregus and Klassen, 1986). In humans and in animals, urinary excretion is low. The total body clearance of manganese in humans can be described by a curve which is the sum of at least two exponential functions with half-times of 4 and 40 days, respectively (ILO, 1983).

Normal individuals excrete manganese (all routes) with a half-life of 37 days, whereas miners exposed to ore dust at TLV of 5 mg/m^3 excreted manganese with half-life of 15 days (Clayton and Clayton, 1981). Manganese is also excreted by sweat, hair, placenta, and milk (Friberg, 1986).

IMMUNOTOXICITY: No information was found on the immunotoxic potential of manganese.

REPRODUCTIVE TOXICITY:
Diminished libido or impotence have been the most common symptoms (Cook et al. 1974). There was no significant increase in deformities or malformations in pups in female rats given manganese in the diet during pregnancy at doses between 4 and 1004 mg/kg dry weight (Friberg et al., 1986).

NEUROTOXICITY: The effects of manganese intoxication produces signs and symptoms of CNS toxicity which can be divided into two broad stages, the first dominated by psychological disturbances which subside if manganese exposure is terminated and a second, predominantly neurological disturbance which occurs with continued manganese exposure and which is not reversible (Cook, 1974).

Chronic exposure to manganese at concentrations greater than 5 mg/m^3 may result in a central nervous system disorder referred to as manganism. Symptoms begin with headache and irritability. Hallucinations, psychotic behavior, somnolence followed by insomnia weakness, incoordination, speech impairment and impotence may then develop. In the advanced stage, the patient develops Parkinsonian-like symptoms with tremor, muscle rigidity, mask-like face and gait disorders. Once established, neurologic effects may progress even in the absence of continued exposure (Proctor et al., 1988). Manganese exerts these effects via disruption of the central nervous system neurotransmitter dopamine (EPA, 1984).

The neurochemical effects of chronic manganese exposure were first described by Cotzias (1962). Dopamine, norepinephrine and serotonin are depleted from certain brain regions, especially the caudate nucleus and substantia nigra. Manganese is most potent at depleting dopamine. This is supported by the fact that the signs and symptoms of chronic manganese toxicity resemble Parkinson's disease, a syndrome associated with selective dopamine depletion.

GENOTOXICITY: Manganese compounds have produced both positive and negative results with bacterial mutagenic assays. Sister chromatid exchanges have been recorded in human and hamster ovary cells. Negative results have been obtained with the dominant lethal mutation assay in male mice, mutations assays with Drosophila and gene conversions or recombination assays in yeast (REPROTEXT, 1990).

CARCINOGENICITY: There is inadequate data in animals and lack of any available data in humans to classify the carcinogenic potential of manganese (IRIS, 1990).

Stoner et al. (1976) tested manganous sulfate in a mouse lung adenoma screening bioassay. Groups of mice were intraperitoneally injected with 0, 6, 15, or 30 mg/kg manganous sulfate 3 times a week for 7 weeks (a total of 22 injections). The animals were observed for an additional 22 weeks. There was an apparent increase in the average number of pulmonary adenomas per mouse both at the mid and high doses as compared with the controls, but the increase was significant only at the high dose. Lung tumors were observed in all dosage groups. The percentage of mice with tumors was not significant at the highest dose level compared with controls. While the results suggest carcinogenicity, the data cannot be considered conclusive since the mean number of tumors per mouse was significantly increased at only one dose, and the evidence for a dose-response relationship was marginal.

EPIDEMIOLOGY: Cases of pneumonia have been reported following occupational exposure to greater than 5 mg/m^3 manganese. Chronic bronchitis has also been noted among exposed workers (EPA, 1984). Respiratory symptoms were found elevated among children residing near a ferromanganese plant. It has been postulated that manganese may interrupt the normal mechanisms of lung clearance, thereby increasing the susceptibility to pulmonary disease (EPA, 1984).

Cross-sectional studies of miners, ferromanganese alloy plant workers, ore crushing mill workers and other industrial workers have documented chronic neurologic effects (manganism) among those exposed to manganese. Effects have occurred after months to years of exposure but studies to date have not been able to determine the minimum dose that produces adverse neurological effects perhaps owing to different individual susceptibility.

Emara et al., 1971 studied 36 workers exposed to manganese dioxide dust in a factory manufacturing dry batteries. Average concentrations ranged from 6.8-42.2 mg Mn/m^3 in four areas. Eight workers (22%) exhibited symptoms of manganism. Concentrations at the main working areas for three of the cases ranged from 6.2-7.2 mg/m^3. The exposure was 1-16 years prior to diagnosis of chronic manganese poisoning.

There is a report of adverse effects from drinking water contaminated with an average concentration of 14 mg/l manganese in a small Japanese community. Sixteen cases of manganese poisoning, three of which were fatal were identified. The subjects exhibited psychological and neurological disorders associated with manganese poisoning, and high manganese and zinc levels were found in organs at autopsy (WHO, 1981).

ENVIRONMENTAL FATE: The principal sources of manganese in the atmosphere are natural processes including continental dust, volcanic gas and dust, and forest fires. The two main mechanisms that may determine the fate of atmospheric manganese are tropospheric chemical reactions and physical removal processes. Manganese aerosol may be removed from the air through dry fallout or wet precipitation. It has been estimated that the atmospheric residence time due to such physical removal processes is about 7 days (Cupitt, 1980). The fate of manganese in aquatic systems may be determined by its ability to undergo chemical and microbiological reactions. It may persist in aquatic systems for a long period. The residence time of aquatic manganese may be a few hundred years. Both chemical and microbiological interactions may cause speciation of manganese in soils. Soil pH and oxidation -reduction potential of soil may influence the speciation process. In acid water-logged soils, manganese passes freely into solution and may leach into groundwater. Also, manganese can be leached readily from waste burial sites and from other natural soils into groundwater (EPA, 1981).

The biological half-time for manganese is between 36 and 41 days (ILO, 1983).

ENVIRONMENTAL TOXICITY:

Manganese is not considered a priority pollutant (EPA, 1986). It is a vital nutrient for both plants and animals. Insufficient manganese produces a chlorosis (yellowed leaves) and failure of the leaves to develop properly. Domestic livestock with inadequate manganese will have low reproductive capabilities and deformed offspring. McKee and Wolf (1963) studied the effects of manganese ions on freshwater aquatic life and found that this metal was well tolerated at concentrations ranging from 1.5 to 1000 mg/l. Because manganese is rarely found at concentrations above 1 mg/l, manganese is not considered to be a problem in fresh waters. A primary water standard for manganese has not been proposed by EPA (1986) because its chemical and biological properties limit its toxicity and distribution in water. Permanganates are more toxic but these compounds are also rapidly reduced and detoxified (EPA, 1986).

REGULATORY STATUS: Manganese is a chronic toxin, a TLV of 5 mg/m^3 is recommended for manganese dust and compounds but

neurological symptoms have been reported at concentrations below this value (EPA, 1984). The regulatory Worker Exposure Limit Values are presented in Table 3.

Table 3. Worker Exposure Limit Values.

ACGIH TLV/TWA:	5 mg/m^3 dusts and compounds
	1 mg/m^3 fume
STEL:	3 mg/m^3
CEILING:	5 mg/m^3
OSHA PEL:	3 mg/m^3
CEILING:	5 mg/m^3
IDLH:	10,000 mg/m^3

(Reference: NIOSH, 1985; ACGIH, 1990).

Water Standards: The national secondary drinking water contaminant level for manganese for public water systems is 0.05 mg/l total manganese (CFR, 1989).

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TOXICOLOGY PROFILE

MERCURY

Synonyms: Metallic Mercury, liquid mercury, quicksilver, mercury salts, hydrargyrum, elemental mercury

CAS No: 7439-97-6

Boiling point: 356.6 °C

Color: Elemental mercury is a silver-white liquid metal; mercury salts vary in color, oxides, sulfides and iodides are yellow, while some sulfides may be black or brown.

DOT designation: Corrosive Material; 2809

Explosive limits: May explode on contact with 3-bromopropyne, methylazide, ethylene oxide, lithium, peroxyformic acid, chlorine dioxide, methylsilane or tetracarbonylnic. In oxygen, alkynes/silver perchlorate mixture may create explosive product upon contact with ammonia. Mercury vapor reacts violently with boron diiodophosphide, acetylenic compounds, aluminum, calcium, potassium, sodium, rubidium and nitromethane.

Flammable limits: Not flammable.

Henry's law constant: NA

Melting point: -38.87°C

Molecular formula: Hg

Molecular weight: 200.6

Odor: Mercury is odorless

Solubility:

water: Elemental mercury has a very low water solubility (0.002 g/100g water at 20 °C) but the vapor has a solubility in water of 60 mg Hg/l @ 20 °C. The solubility of the inorganic salts varies.

K_{ow}: Refer to specific inorganic salts.

other: Very soluble in alcohols; insoluble in ether; soluble in nitric acid; insoluble in hydrogen bromide; cold hydrogen iodide or dilute hydrogen chloride. Dissolves to some extent in lipids (5 - 50 mg/l).

K_{oc}: NA

Specific gravity: 13.59 at 20 °C

Vapor density: 18 mg/m^3 at 24 °C

Vapor pressure: 2 x 10^{-3} mm Hg at 25 °C

Viscosity: 15.5 millipoise at 25 °C

(References: HSDB, 1990; CRC, 1988, NIOSH, 1973)

COMPOSITION: Elemental mercury is a mobile, liquid metal above -38.87 °C. Inorganic mercury salts exists in two oxidative states as either a mercuric (Hg^{2+}) or mercurous (Hg^{1+}) form. The inorganic mercury salts generally exist as solids at room temperature. Mercury may also undergo biotransformation in aquatic environments to organic mercury compounds.

USES: Elemental and inorganic mercury is used in the chloralkali industry, in the manufacture of electrical equipment (switches, batteries, mercury lamps, x-ray tubes, solders) and

scientific equipment (thermometers, barometers). Other industrial uses include preparing dental amalgams, catalysts, preservatives and explosives. In the past mercury has been used in the plating, tanning and dying, textile, and pharmaceutical industries (Khayat, 1985).

Despite the wide variety of industrial uses and potential for release, the major source of mercury release into the environment is from the natural degassing of the earth's crust. Goldwater and Stopford (1977) estimate that the natural background release of mercury ranges from 25,000-150,000 tons per year. Friberg (1986) estimates that human activity results in the release of 20,000 tons of mercury annually. In the United States mercury production doubled between 1973 and 1986, from 1.17×10^6 kg to 3.49×10^6 kg (Bureau of Mines, 1987).

ACUTE TOXICITY: Elemental mercury has different toxicological effects than inorganic mercury salts based on differing physical properties. Thus, each is discussed independently under the following sections. The following table presents acute toxicity values of various mercury compounds.

Table 1.

Representative Acute Toxicity Values of Representative Mercury Compounds in Different Species of Animals

Hg_2Cl_2	Mercurous Chloride	rat	Oral	LD50	166 mg/kg
		rat	dermal	LD50	1500 mg/kg
		mouse	oral	LD50	180 mg/kg
$HgCl_2$	Mercuric Chloride	human	oral	LD10	29 mg/kg
		rat	dermal	LD50	41 mg/kg
$Hg(CN)_2$	Mercuric Cyanide	rat	oral	LD10	25 mg/kg
		mouse	oral	LD50	33 mg/kg
HgI	Mercuric Iodide	mouse	oral	LD50	110 mg/kg
Hg_2SO_4	Mercurous Sulfate	rat	oral	LD50	205 mg/kg
		rat	dermal	LD50	1175 mg/kg
		mouse	oral	LD50	152 mg/kg
$HgSO_4$	Mercuric Sulfate	rat	oral	LD50	57 mg/kg
		rat	dermal	LD50	625 mg/kg
		mouse	oral	LD50	25 mg/kg

(Adapted from Sax & Lewis, 1989)

Ingestion: Elemental mercury does not usually produce acute toxic effects when ingested, since G.I. absorption of the metallic form is low. However, volatilization in the G.I. tract followed by absorption of the vapor is the main mechanism of exposure although coating of the liquid with mercury sulfide can limit volatilization and absorption. Metallic mercury may present a toxicological problem when it is deposited in fistulas, diverticulas or abscesses in the G.I. tract, or as an aspiration hazard during emesis (Friberg et al., 1986; Geller, 1976).

If elemental mercury is not removed from the G.I. tract, the absorption of the vapors produces effects resembling mercury salt toxicity. These signs and symptoms include pneumonitis, lethargy or restlessness, fever, tachypnea, cough, chest pains, cyanosis, diarrhea, vomiting, atelectasis, emphysema, hemorrhage and pneumothorax. In mercury vapor intoxication, CNS toxicity is more prominent than with mercury salts because non-ionic elemental mercury passes through the blood-brain barrier more readily than the salt (Goodman and Gilman, 1980).

Mercury salts present a greater relative acute health hazard via ingestion than metallic mercury. It has been estimated that 1 to 4 grams of mercury chloride is fatal in adults, although fatalities have occurred with as little as 0.5 g. Mercury salts are also more corrosive than the metal and mercuric salts are more corrosive than the mercurous salts (Klaassen et al, 1986). The corrosiveness may enhance G.I. permeability and absorption rates and can be so rapid that a patient's prognosis is determined by events

during the first 10-15 minutes (Gosselin et al., 1984). Winek (1985) reported lethal blood levels to range from 0.4-22 µg/ml (0.04-2.2 mg%) in humans.

Signs and symptoms of mercury salt intoxication occur in two phases. Phase one is characterized by burning pain, in the chest, darkened discolorization of oral mucous membranes, severe G.I. pain, vomiting of mucoid material, bloody diarrhea, metallic taste, salivation, tachycardia, weak pulse, tachypnea, pallor, prostration, and possibly shock, circulatory collapse and death (Gosselin et al., 1984). If death does not occur, then phase two begins 1-3 days after ingestion. Phase two signs and symptoms include mercurial stomatitis, characterized by glossitis and ulcerative gingivitis, loosening of the teeth, jaw necrosis, proximal tubular necrosis resulting in transient polyuria, albuminuria, cylindruria, hematuria, anuria and renal acidosis. If death does not occur, recovery is usually within 10-14 days (Gosselin et al., 1984). Acute intoxication may also produce dysentery, tenesmus, colonic ulcerations, capillary damage, liver necrosis, occasionally tremors and peripheral neuropathies or other neurological effects. Death may occur from minutes to weeks after exposure (Gosselin et al., 1984).

Inhalation: Since elemental mercury has a toxicologically significant vapor pressure at room temperature, it presents an acute health hazard. Inhalation of mercury vapor can produce a potentially fatal corrosive bronchitis, pulmonary edema, interstitial pneumonia, and central nervous system effects which are commonly reversible (Klaassen et al., 1986), but McFarland and Reigel (1978) have reported irreversible effects. CNS effects from acute exposure may include tremors, paresthesia, memory loss, hyperexcitability, erythism and reduction in reflex and peripheral nerve conduction times (EPA, 1984b). Exposure of humans to mercury vapor ranging from 1.2-8.5 mg/m^3 caused coughing, chest pains, dyspnea, bronchitis and pneumonitis (NIOSH, 1978). Death results from respiratory compromise. An LC_{10} of 29 $mg/m^3/30$ hours was recorded in rabbits (Sax and Lewis, 1989). Although irreversible neurotoxicity is more commonly seen in long-term, chronic exposures, McFarland and Reigel (1978) reported six cases of chronic CNS effects after a single high concentration exposure to mercury vapor.

Inhalation of mercury salts is not a common pathway of exposure since mercury salts are generally solids at room temperature with low vapor pressures. However, upon heating, many salts decompose, liberating elemental mercury vapor which can be inhaled and cause toxic effects (Sax and Lewis, 1989).

Skin contact: Mercury salts have been shown to penetrate the skin. Absorption rate depends upon compound, concentration applied and integrity of the skin (Wahlberg, 1965). Vostal (1975) reported that animals absorbed up to 8% of mercury chloride within 5 hours. Unfortunately current data does not permit a comparison of the relative hazard of dermal absorption to inhalation. In general, mercury compounds are irritating, occasionally producing dermatitis, with or without vesication, discoloration of the nails, corrosion of mucous membranes and may cause severe system effects (Gosselin et al., 1984; Deichmann and Gerarde, 1969). DOT emergency guidelines state that skin contact to mercury compounds will cause corrosive burns (DOT, 1987).

Eye contact: Elemental mercury in direct contact with conjunctiva does not produce signs of irritation but is absorbed and detectable in urine (Grant, 1986). Eye contact with mercury vapor can produce inflammation and discoloration of the lens. Injecting droplets of elemental mercury into the anterior chamber or corneal stroma in rabbits causes a purulent formation around the droplet or in the cornea, culminating in expelling the droplet (Grant, 1986). Injection of elemental mercury into the vitreous humor of rabbits produced a purulent reaction, retinal detachment, shrinkage of the vitreous humor with retina and ocular atrophy (Grant, 1986).

No data are available discussing the acute effects of eye contact with mercury salts. Due to the general corrosivity of mercury salts, eye contact is likely to result in conjunctival irritation dependent upon compound, dose and duration of exposure. Gosselin et al. (1984) state that many mercury compounds cause ulceration of the conjunctiva and cornea. DOT emergency guidelines state mercury causes burns to the eye with eye contact (DOT, 1987).

CHRONIC TOXICITY: Chronic mercury toxicity typically arises from workplace exposure. Chronic toxicity to mercury salts is unlikely due to the probable generation of elemental mercury vapor and concurrent inhalation exposure (Friberg et al., 1986). Chronic mercury intoxication from exposure to organic mercury compounds via consumption of contaminated fish or bread has been observed (Goodman and Gilman, 1980).

Ingestion: See ACUTE TOXICITY: Ingestion for information addressing signs and symptoms of inorganic mercury toxicity.

Effects associated with ingestion of organic mercury (methylmercury) are well-documented. In 1968, Katsuna reported neurologic disease among residents of Minimata, Japan who consumed fish contaminated with organic mercury. Signs and symptoms included memory loss, personality changes, paresthesias, incoordination, depression, and insomnia. Methyl mercury is particularly toxic to the nervous system of the developing fetus (Matsumoto et al. 1965).

Methylmercury compounds have been applied to seed grain to prevent growth of fungi. Mass poisonings have resulted when treated seed grain is made into bread products. Cases have been reported in Sweden in 1952, and in Iraq in 1956, 1960, and 1972. Bakir et al. (1973) reported severe neurologic damage associated with the ingestion of bread prepared from methylmercury-contaminated seed grain in Iraq. Fetotoxicity was evident: severe brain damage developed among infants born with blood mercury levels greater than 250 µg/dl. As was noted in Minimata, the fetus is particularly susceptible to the effects of methylmercury. In a follow-up of Iraqi women exposed in 1971, Marsh et al. (1980) noted developmental delays and mild neurologic changes in children of women with low exposure (mercury levels in hair 0-11 and 12-85 µg/g).

Inhalation: Chronic inhalation of elemental mercury produces conspicuous central nervous system effects that are both neurological and psychiatric (Goodman and Gilman, 1980). The severity and reversibility of effects are dose-dependent (Friberg et al., 1986). The pathological basis of CNS dysfunction is unknown (Friberg et al., 1986) but there is mounting evidence that the mechanisms may include interference with protein synthesis and neuronal synaptic transmission (Manalais and Cooper, 1975).

Chronic neurologic disease has been reported among a variety of mercury-exposed groups, including dentists (Iyer et al., 1976), dental technicians (Uzzell and Oler, 1986), and chlor-alkali plant workers (Levine et al., 1982, Albers et al., 1982, Piikivi et al., 1984). A syndrome resembling amyotrophic lateral sclerosis (ALS) has also been reported among mercuric-oxide manufacturing workers (Barber, 1978) and cannery workers salvaging elemental mercury (Adams et al., 1983).

Effects associated with mercury toxicity may or may not be reversible. Increasing the duration and intensity of exposure decreases the likelihood of recovery (EPA, 1984b).

Chronic, low-level exposure to inorganic mercury may result in kidney effects, including acute nephrotic syndrome with proteinuria. Stewart et al. (1977) described renal effects in technicians in a histology laboratory exposed to mercuric chloride in a fixative solution. Elevations in urine mercury (median 265 nanomoles per 24 hours) and protein levels were found. Levels returned to normal after industrial hygiene improvements.

Skin contact: Mercurous salts are less corrosive and toxic than mercuric salts due to their lower solubility. Calomel, a mercurous chloride product, has been implicated as an agent acrodynia when it was used as a teething powder for infants. Acrodynia, or "pink disease", is probably a hypersensitive response to mercury salts characterized by vasodilation, fever, hyperkeratosis, swelling of the spleen, lymph nodes and fingers and excessive perspiration (Klaassen, 1986).

Chronic absorption of mercurous and mercuric salts via skin contact may produce renal effects in addition to CNS effects from inhalation of elemental mercury vapor. Other symptoms include increased salivation, and inflammation of the gums and black dental lines resulting from mercuric sulfide precipitation (Friberg et al., 1986).

Eye Contact: See ACUTE TOXICITY, Eye contact. Repeated non-ocular exposure of mercury to animals produces discoloration of the cornea, consisting of a grey ring in the cornea extending to the lens (Grant, 1986).

SENSITIZATION: Based upon skin prick tests, mercury salts, have been shown to cause hypersensitive responses. In a study of dental students, hypersensitive responses grew from 2% in entering students to 18% in senior students (White and Brandt, 1976). Mercury has also been reported to induce a renal autoimmune reaction (Klaassen et al., 1986).

TARGET ORGAN EFFECTS: Inorganic mercury adversely affects the central and peripheral nervous systems, the kidney, the eye and reproductive fitness. *In utero* exposure produces fetotoxic effects. Organomercury compounds produce similar effects, but are not within the scope of this toxicological profile.

Renal effects are primarily attributable to the divalent mercuric ion. Two types of renal damage are associated with mercury salt exposure; damage to the basal membrane of the glomeruli and tubular damage (Friberg et al., 1986). Glomeruli basal membrane damage is caused by an autoimmune reaction. Mercury

induced antibodies to glomerular tissue have been found in rats exposed to mercuric chloride (Sapin et al., 1977). Mercury also accumulates in distal tubules, impairing protein synthesis, thus compromising renal function. Sensitivity to mercury renal toxicity is sex-dependent, with males being more sensitive (Muraoka and Itoh, 1980).

ABSORPTION-METABOLISM-EXCRETION: Mercury vapor is readily absorbed across the alveolar membrane into the blood stream. An average of 25% of the mercury inhaled was absorbed from air concentrations that ranged from 2.9 to 26.18 mg/m^3 (Browning, 1969). Gastrointestinal and skin absorption of elemental mercury is very low, with less than 0.01% probably absorbed from the G.I. tract Once in the blood stream, mercury may be oxidized by red blood cells to the divalent cation, or transported by RBC's to other tissues where it is oxidized. Elemental mercury has greater lipid permeability than the ionized form and can pass though the blood-brain or transplacental barriers (Friberg et al., 1986). Rapid transfer of mercury across the blood-brain barrier creates a toxicologically significant differential distribution to the brain. A similar distributive process occurs in the fetus. Upon entering these tissues, mercury undergoes oxidation and is trapped in these compartments (Klaassen, 1986).

The primary routes of exposure for ionic mercury is via ingestion or skin contact (Klaassen, 1986). Although ionic mercury may be present as a respirable dust or adsorbed onto particulate matter, no literature addresses this potential route of exposure. Ionic mercury is corrosive and probably enhances absorption by altering permeability (Gosselin et al., 1984). Approximately 10% of the ingested dose is absorbed, while approximately 8% of the dermally applied dose is absorbed within 5 hours (Goodman and Gilman, 1980). Ionic mercury distributes approximately equally between plasma and RBC's probably binding to sulfhydryl groups on hemoglobin and possibly to glutathione (Friberg et al., 1986).

Ionic mercury does not readily penetrate either the blood-brain or transplacental barriers, but does accumulate in fetal membranes (Suzuki, et al., 1977). Except for differences in brain and fetal distribution, elemental and ionic mercury tissue distribution may be similar. In the brain, mercury accumulates in the cerebral cortex, particularly the occipital and cortical areas, the cerebellum, nuclei of the brainstem and the choroid plexus (Friberg et al., 1986). Mercury accumulates to the greatest extent in the kidney, particularly in the peripheral part of the renal cortex corresponding to the convoluted portion of the proximal tubuli (Taugner et al., 1966). Mercury in the kidney may either be soluble, bind to metallothionein or to proteins (Cherian and Clarkson, 1976).

The second largest depot of mercury deposition is the liver, with the greatest concentration near the periportal areas. Mercury has an affinity for ectodermal and endodermal epithelial cells such as the epithelial lining of the G.I. tract, squamous epithelium of the hair and skin, and glandular tissue, such as salivary glands, thyroid, pancreas, sweat glands, testicles and the prostrate.

Elimination of mercury, whether exposure is to vapor or ionic salts, is mainly in the ionic form, although some elemental mercury vapor is eliminated in exhaled air. The majority of ionic mercury is excreted in the feces and urine. Partition between these two routes is dose dependent, with the larger fraction being eliminated in urine as the dose increases. Elimination also occurs via sweat glands, lacrimal glands, mammary glands, salivary glands and bile. Mercury is taken up by the basal cells of the proximal tubuli from capillaries (Wessel, 1967), transported through the cell membranes of the tubular wall as amino acid complexes (Richardson et al., 1975) and transferred to the tubular lumen.

Mercury distribution and excretion can best be approximated as a multi compartment model with at least two excretion rates. The brain compartment has a long half-life that may exceed several years (Rossi et al., 1976). The second compartment; the rest of the body has an excretion rate with a half-life of approximately 60 days and accounts for 80% of the mercury body burden.

REPRODUCTIVE TOXICITY: No data were available addressing the teratogenic potential of inorganic mercury in humans.

Exposure to elemental mercury vapors caused reduced reproductive fitness and impairing fetal development from *in utero* exposure. Sager et al. (1986) states that mercury adversely affects both male and female reproductive capacity.

Adverse effects on human reproductive fitness was observed by Sikorski et al. (1987) in a study measuring reproductive failure of dental professionals in Poland which are customarily exposed to mercury vapor in the preparation of amalgams. The dental professionals were observed to have double the normal reproductive failure rate or 23.9% versus 11.1% in controls. The mercury content in scalp and pubic hair

samples of the exposed group correlated with reproductive failure.

In animals, a spontaneous abortion was observed in one monkey exposed to 0.5 mg/m^3 for 20 weeks (Clayton and Clayton, 1981). Mercury was found in all tissues and organs analyzed from the fetus, indicating mercury crossed the transplacental barrier. Both ionic and elemental mercury cross the placenta, although elemental mercury crosses it approximately twenty times more readily (Koos and Longo, 1976). Mercury has also been shown to accumulate in fetal liver and the placenta in concentrations greater than maternal liver or fetal or maternal blood (Yoshida, 1986).
Animal studies with ionic mercury have produced a variety of embryotoxic effects which include increased litter resorption, growth retardation, subcutaneous edema, exanaphaly, anophthalmia, other eye defects, increased frequency of malformations and fetal death (Koos and Longo, 1976; Rizzo and Furst, 1972; Miyoshi, 1959). Neonate exposure may also arise via nursing. Both ionic and elemental mercury have been found to transfer to breast milk (Mansour et al., 1973).

Organic mercury compounds such as methylmercury and phenylmercury, are also teratogens and reproductive toxins in humans and animals (Friberg et al., 1986), however, this class of mercury compounds is not within the scope of this profile.

NEUROTOXICITY: Inorganic mercury vapor is absorbed into the blood via inhalation or ingestion. The non-ionized mercury then passes through the blood-brain barrier, where it penetrates nerve cells (Friberg et al., 1986). Cassano et al. (1966) found intracellular mercury in nerve cells following mercury vapor exposure. The elemental mercury is believed to be oxidized to the divalent mercuric ion which then binds to sulfhydryl proteins, impairing cellular function. Mercury's most significant cellular effect is probably inhibiting protein synthesis, although it may interfere in neurotransmission (Manalais and Cooper, 1975). Pathological changes from exposure to mercury have been observed in Purkinje cells, granule cells in the cerebellum, cortical pyramidal cells and neurons in the substantia nigra, the nuclei olivaris and dentatus, and anterior horn cells of the spinal cord (Davis et al., 1974). CNS effects may be reversible, depending upon intensity and duration of exposure (Vigliani et al., 1953).

The peripheral nervous system may also be affected. Investigators have found that the neural conduction velocities are reduced in chronically exposed individuals (Vroom and Greer, 1972). Iyer et al. (1976) found differences between sensory and motor nerve response to chronic mercury exposure in one individual. Treatment with penicillamine reversed mercurial neurotoxicity in this individual. Ingestion of organic mercury via contaminated fish or seed grain has resulted in memory loss, personality changes, parathesias, incoordination, depression, and insomnia. Severe brain damage has been documented among children exposed in utero (Marsh et al., 1980; Bakir et al., 1973).

GENOTOXICITY: There is equivocal data indicating inorganic mercury may be genotoxic. Aneuploidy and other chromosomal aberrations were observed in whole blood cultures of individuals exposed to mercury amalgams (Verschauerve, 1979). Popescu et al. (1979) were unable to confirm that worker exposure to organic or inorganic mercury resulted in an increase in aneuploidy or chromosomal aberrations. Mercuric mercury (Hg^{2+}) failed to induce chromosomal aberrations in cultured mammalian cells (Umeda and Nishimura, 1979) or sister chromatid exchange in human lymphocytes (Ogawa, 1979). The aneuploidic or hyperploidic effect may be due to inhibition of mitosis in a colchicine-like fashion.

CARCINOGENICITY: Inorganic mercury is not known to be a human carcinogen, EPA classifies it as Group D: not classifiable as a human carcinogen (IRIS, 1990). Human epidemiology studies have found no correlation between exposure and cancer. Cragle et al. (1984) followed the mortality experience of 2133 workers exposed to mercury vapor from 1953 to 1963. An excess in lung cancer (SMR 1.34) was observed in both the exposed and control group, thus excess cancer deaths could not be attributed to mercury exposure.

EPIDEMIOLOGY: Cross-sectional studies have found neurologic disease and adverse renal effects among workers exposed to inorganic mercury. These studies are reviewed under Chronic Toxicity, Inhalation. Case studies have noted neurotoxicity among populations consuming organic mercury contaminated fish and seed grain. These studies are reviewed under Chronic Toxicity, Ingestion.

ENVIRONMENTAL FATE: Mercury enters the environment from natural and man-made sources in three possible forms, as elemental mercury, or a mercury salt or as an organo-mercury compound.

Inorganic mercury may exist as vapor in the atmosphere subject to soil and aquatic deposition, or as a liquid solid in soil water.

Background air concentrations may range from 1-4 ng/m^3, depending on industrial and volcanic activity. Typical aquatic concentrations of dissolved mercury are: 0.5-3 ng/l for open ocean; 2-15 ng/l for coastal sea water; and 1-3 ng/l for lakes and rivers. Background soil levels are typically less than 50 µg/kg, while ocean sediments range from 20-100 µg/kg (WHO, 1989). Most mercury deposited on land will revaporize within 1-2 days after deposition. The half-life of mercury in aquatic environments is estimated to be about one year. Bioaccumalation or transformation into a more volatile compound are the two primary routes of elimination (NRCC, 1979).

Inorganic mercury may be oxidized to the divalent cation, particularly in aquatic environments in the presence of organic matter. Both inorganic forms of mercury may undergo biotransformation into methyl mercury in aquatic environments. In addition, inorganic salts may absorb to sediments or soils, particularly those with high sulfur content, or may be reduced to inorganic elemental mercury and vaporize (Klaassen, 1986).

Microbial methylation is a slow detoxification response enabling microbes to eliminate heavy metals and depends on microbial population, environmental conditions, such as pH, temperature and redox potential (Klaassen, 1986). The best reported microbial conversion rate was 1.5% per month (Jensen and Jernalov, 1969).

Both organic and inorganic mercury accumulate in algae and fish. Methyl mercury is taken up much more readily and eliminated more slowly, resulting in greater accumulations than inorganic forms. The biological half-life of mercury, primarily methylmercury, in fish is approximately 2-3 years (EPA, 1984a). Callahan et al. (1979) found the following bioconcentration factors for mercury.

Table 2. Bioconcentration Factors for Mercury in Different Marine Species.

Species	Bioconcentration Factors
Marine Plants	1,000
Freshwater Plants	1,000
Marine Invertebrates	100,000
Freshwater Invertebrates	100,000
Marine Fish	1,670
Freshwater Fish	1,000

(Reference: HSDB, 1990)

Therefore, consumption of aquatic species, particularly those at the top of the food chain, can contribute to mercury body burdens because mercury bioconcentrates. Ingestion of mercury containing food particularly aquatic food species, is the primary source of human exposure (EPA, 1984a).

Terrestrial plants also have a limited ability to absorb and translocate mercury from the soil. Investigators have found that plant absorption is dependant on soil mercury concentration (NRCC, 1979). Marine plants have been found to contain 0.01-37 ppb (fresh weight) of mercury. Terrestrial plants have been found to contain 0-40 ppb (fresh weight) mercury, while terrestrial plants in the vicinity of mercury deposits have been found to contain 200-30,000 ppb (fresh weight) (Jonasson, 1972).

ENVIRONMENTAL TOXICITY: Mercury is toxic to microorganisms, plants, invertebrates and vertebrates. Aquatic mercury toxicity is dependent upon the chemical form. The organic forms are generally more toxic than inorganic forms. Aquatic mercury toxicity is also dependant upon temperature, pH, salinity and water hardness (WHO, 1989).

Inorganic mercury has produced adverse effects in microorganisms at levels as low as 5 µg/l, while organic mercury may produce effects at concentrations ten times lower. The mechanism of action in aquatic organisms is essentially the same as in higher forms of life; mercury binds to essential proteins, such as enzymes, and impairs cellular functions. Microorganisms have developed six protective responses to mercury exposure. Microorganisms may actively excrete, reduce, chelate, bind, precipitate or methylate mercury (WHO, 1989). The LC_{50} of mercuric chloride in *Chlorella vularis* was 400 µg/l (Rai, et al., 1981) with a NOEL of 50 µg/l (Den Dooren de Jong, 1965).

Aquatic plants are more sensitive to mercury than terrestrial plants. As in microorganisms, organic mercury compounds are more toxic than the inorganic compounds. Generally, aquatic plants are affected by inorganic mercury concentrations approaching 1 mg/l. Salinity reduces toxicity of both mercury forms in aquatic environments. Photosynthesis in isolated spinach chloroplasts was significantly inhibited with 0.005 µm Hg/mg chlorophyll (Brandeen et al., 1973). The 50% inhibition of growth rate for mercuric chloride in red alga was 1.0 mg/l at 6 hours; 0.5 mg/l at 12 hours and 0.25 mg/l at 24 hours. Exposure to mercuric chloride in concentrations ranging from 0.05-20 mg/l on

floating water cabbage reduced chlorophyll content, protein, dry weight, RNA, catalase and protease activities while increasing free amino acid content (De Anil, et al., 1985). Mercury phytotoxicity is affected by soil absorption which can produce a barrier that prevents mercury translocation from the roots to other regions of the plant (De Anil et al., 1985).

Seed germination and early growth in the 15 species tested was not significantly affected by exposure to saturated mercury vapor (14 µg/l), although prolonged exposure for up to 75 days caused necrosis (Siegel et al., 1984).

The table below contains acute ecotoxicity values for inorganic mercury in a variety of species.

Table 3.
Acute Ecotoxicity Values for Mercury in Different Species

Species	Parameters	Dose
Water flea (Daphania magna)	LC_{50sa}	5-4890 µg/l
Crayfish (Orconectes limosus)	LC_{50fb}	<2-50 µg/l
Freshwater Snail (Pila Globosa)	LC_{50s}	296-1108 µg/l
Freshwater Crab (Oziotelphusa sp.)	LC_{50s}	443-739 µg/l
Freshwater Mussel (Lamellidens sp.)	LC_{50s}	3690-7390 µg/l
Marine Snail (Nassarius osboletus)	LC_{50s}	700-32,000 µg/l
Marine Crab (Scyella serrata)	LC_{50s}	680-930 µg/l
Softshell clam (Mya arenaria)	LC_{50s}	4-5200 µg/l
American Lobster (Homarus amevicanus)	LC_{50s}	20 µg/l
Catfish (Sarotherodon sp.)	LC_{50s}	75-1700 µg/l
Rainbow Trout (Salmo giardneri)	LC_{50s}	220-650 µg/l
Striped Bass (Roccus saxatillus)	LC_{50s}	90-220 µg/l
Carp (cyprinus carpio)	LC_{50s}	180-330 µg/l
Japanese Quail (Conturnix conturnix japonica)	LC_{50c} LD_{50}	5086 mg/kg 42 mg/kg
Pheasant (Phesianus colchicus)	LC_{50}	3790 mg/kg
Mallard Duck (Anas platyrhynchos)	LC_{50}	>5000 mg/kg

(a)s = static system
(b)f = flow through system
(c) birds fed with a dosed diet for 5 days followed by regular diet for 3 days.
(Adapted from WHO, 1989)

Aquatic invertebrates vary in sensitivity to mercury compounds. Generally, organic mercury compounds are more toxic than inorganic mercury compounds and larval stages are more sensitive than adult stages. Temperature, oxygen content, salinity and water hardness affect toxicity. The LC_{50} for the most sensitive developmental stage for most species ranges from 1-10 µg/l with the lower values in flow through rather than static systems (WHO, 1989). The 96 hour static LC_{50} in freshwater species generally ranges from 33-400 µg/l for inorganic mercury which suggests that mercury may be absorbing to the exposure chamber and not provide realistic estimates of mercury toxicity (WHO, 1989). Mercury has also been shown to impair capture avoidance in prey organisms (Kraus and Kraus, 1986).

Mercury is toxic to freshwater and marine fish species. Inorganic mercury has also been shown to be a reproductive toxicant in freshwater fish. Mercury ingestion is toxic in birds, effecting renal function and pathology, cardiovascular function, immune response and blood parameters. Mercury has also been shown to impair growth and affect behavior (WHO, 1989).

REGULATORY STATUS: Mercury is used in a wide variety of applications, thus there are a wide variety of industries where human exposure and environmental releases occur. Consequently, mercury is subject to regulation under a variety of statutes, ranging from the Occupational Safety and Health Act to the Food, Drug and Cosmetic Act. The EPA (1984a) found mercury content of fish and shell fish to vary, averaging 100-200 ng Hg/g fish. Food additives containing mercury are also subject to regulation under FDA. Dietary intake of mercury on a daily average between 1982-85 was up to 3.7 µg for adults and up to 1.2 µg in toddlers/infants (EPA, 1987).

Mercury may also be ingested in water. EPA (1984b) estimated drinking water to contain 5-100 ng/l. Mercury is designated a toxic pollutant under section 307(a)(1) of the Clean Water Act (CFR, 1989) and has a maximum contaminant level (MCL) of 2 µg/l (CFR, 1989). EPA (1980) proposed 144 ng/l as protective of ambient water quality.

Workplace inhalation exposure is regulated under the Occupational Safety and Health Act (CFR, 1989). The NIOSH standard was based on concentrations causing erethism, rather than tremors (NIOSH, 1973).

Mercury is also subject to hazardous waste regulations under CERCLA and RCRA. Wastes containing mercury are subject to designation as hazardous waste following the Toxicant Extraction Procedure as set out in RCRA (CFR, 1989). One pound or 454 g of mercury is the reportable quantity under CERCLA (CFR, 1989)

Table 4. Worker Exposure Limits for Mercury

ACGIH TWA	0.05 mg/m^3 (vapor); 0.10 mg/m^3
STEL	<0.25 mg/m^3; 0.5 mg/m^3 for 30 min.
Ceiling	0.10 mg/m^3
OSHA PEL	0.05 mg/m^3
MSHA	N/A

N/A = not available
(Reference: NIOSH, 1973; NIOSH, 1985; HSDB, 1990).

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TOXICOLOGY PROFILE

NICKEL

Synonyms: nickel powder

CAS NO: 7440-02-0

Boiling point: 2920 °C for the elemental form

Color: silvery

Dot designation: combustible/irritant

Flammable limits
autoignition: NA
flash point: NA
LEL: NA
UEL: NA

Henry's law constant: NA

Melting point: 1455 °C for the elemental form

Molecular formula: Ni = element
$Ni(CO)_4$ = nickel carbonyl
$NiCl_2$ = nickel chloride
$Ni_{11}As_8$ or Ni_5As_2 = nickel subarsenide
Ni_3S_2 = nickel subsulfide
NiAsS = nickel sulfarsenide
$NiSO_4$ = nickel sulfate
NiO = nickel oxide

Molecular weight: 58.7

Odor: unknown

pH: NA

Solubility

water: The chloride, nitrate and sulfate salts are soluble in water. The elemental oxide and subsulfide form is insoluble in water.

K_{ow}: NA

other: The elemental form of nickel is considered to be insoluble in most solvents. Other compounds of nickel are soluble in alcohols and acids to varying degrees.

K_{oc}: NA

Specific gravity: 8.9 for the elemental form

Vapor denisity: Nickel carbonyl is 4 times heavier than air. Other nickel compounds found only as particulate matter.

Vapor pressure: Nickel carbonyl is highly volatile. Other nickel compounds are not volatile.

Viscosity: NA

(References: IARC,1984; ATSDR, 1987)

COMPOSITION: Nickel comes in four oxidation states; 0, +1, +2, and +3. The metal is found as sulfide or oxide ores (EPA, 1984) which undergo flotation, roasting and smelting to produce a "matte" that is composed of 15% nickel. This matte is treated to selectively oxidize iron sulfide to produce a material that contains about 50% nickel. A final electrolysis increases the purity of the nickel material. High purity nickel is also produced from nickel carbonyl. Nickel carbonyl is a volatile, colorless liquid with a boiling point of 43 °C and which decomposes at temperatures above 50 °C. Heating nickel carbonyl forms a high purity nickel and releases carbon monoxide (Norseth, 1986).

USES: About 40% of the produced nickel is used in the manufacture of steel and other alloys. Coins and kitchen utensils are common items that use nickel alloys. About 20% of the produced nickel, in the form of nickel sulfate, is used in electroplating and nickel hydroxide is used in the manufacture of nickel cadmium batteries. Nickel carbonate is used in electronic components such as vacuum tubes and transistors (Norseth, 1986).

ACUTE TOXICITY: Many nickel compounds are not considered to be very toxic on the basis of acute oral exposures in rats as can be seen in Table 1.

Excessive exposure to nickel can produce a variety of local effects but the only compound known to produce systemic effects is nickel carbonyl (Norseth, 1986). The predominant effect of exposure to nickel and its compounds is an allergic reaction (Proctor and Hughes, 1988). It is estimated that 5% of all eczemas are produced by nickel reactions (HSDB, 1990). Once sensitivity is acquired, it can be aggravated by the diet and is apparently not lost (Proctor and Hughes, 1988).

TABLE 1. Acute Toxicity Values of Several Nickel Compounds.

Compound	LD50 (mg compound/kg bw)	LD50 (mg Ni/kg bw)
Nickel acetate	355	118
Nickel hydroxide	1600	1021
Nickel sulfate hexahydrate	300	67
Nickel oxide	>5000	>3929
Nickel sulfide	>5000	>3233
Nickel subsulfide	>5000	3666

(References: ATSDR, 1987; Mastromatteo, 1986)

Ingestion: Limited human data are available regarding adverse health effects associated with the ingestion of nickel and nickel compounds. The only fatality by nickel via the oral route was reported by Daldrup et al., (1983). A two-and-a-half year old girl ingested 15 grams of nickel sulfate crystals, exhibited pulmonary rhonchi and died of cardiac arrest.

In another episode, 32 workers in an electroplating plant accidentally drank water contaminated with nickel sulfate and nickel chloride with a total nickel content of 1.63 g/L. Twenty workers promptly developed nausea, vomiting, abdominal discomfort, diarrhea, giddiness, lassitude, headache, cough, and shortness of breath which lasted from a few hours to several days. It was estimated that the workers drank between 0.5 to 2.5 grams of nickel. Serum concentrations of Ni were between 13 to 1,340 µg/l and urine concentrations ranged from 0.15 to 12 mg/g creatinine. Induced diuresis shortened the elimination half time to 27 hours

compared to the non-induced groups with an elimination half time of 60 hours. All the subjects recovered and returned to work within eight days (Sunderman et al., 1988).

Inhalation: The acute toxicity from exposure to nickel by inhalation includes headache, sore throat, hoarseness, and a non productive cough. This may be followed by tightness in the chest, dyspnea, and retrosternal pain. Late effects could include pulmonary edema and interstitial fibrosis (HSDB, 1990). Nickel carbonyl is the most acutely toxic nickel compound. In some cases of accidental occupational exposures, nephrotoxicity was manifested in the form of renal edema, hyperemia and parenchymatous degeneration (Carmichael, 1953).

Eye contact: Nickel and its compounds are considered irritating to the eye as well as other mucous membranes (NIOSH, 1977). Workers employed in nickel plating shops have reported developing conjunctivitis and sudden flood of tears when the ventilation was poor (Grant, 1986).

Skin contact: Workers exposed to nickel can develop a sensitization dermatitis called "Nickel Itch". The initial symptoms may be a sensation of burning or a pruritus which occurs several days before an erythematous or follicular skin eruption. This may be followed by superficial discrete ulcers which discharge and become encrusted or an eczema on the web of the fingers, wrist and forearm. In addition to the primary contact site, the eruptions may occur on the eyelids, sides of the face and neck and flexure of the elbow. Recovery from the dermatitis usually occurs within 7 days following cessation of exposure.

Application of nickel sulfate to the skin of rabbits indicated very little absorption of the applied material. However, application of similar dose to the abraded skin produced signs of toxicity such as convulsions, salivation and death (NIOSH, 1977).

CHRONIC TOXICITY: More recent clinical and epidemiological studies indicate that nickel sensitivity is not confined to the workplace since more and more people are encountering nickel containing commodities in their environment. Vandenberg and Epstein (1963) were able to sensitize 9% of their subjects to nickel.

Ingestion: The role of oral nickel in nickel dermatitis has been shown by a number of workers and is summarized by EPA (1986). Dietary nickel has been found to be related to nickel dermatitis flare-ups in about 50% of the clinical cases. A low nickel diet can control dermatitis.

Inhalation: Irritation of the upper respiratory tract, chronic rhinitis, sinusitis, asthma and an increased risk of respiratory tract infections have been reported among individuals occupationally exposed to nickel. Chronic exposure to high levels of nickel and its compounds may result in decreased sensation of smell and nasal septal perforation (ATSDR, 1987).

Eye contact: See Eye contact , ACUTE TOXICITY.

Skin contact: With chronic exposure, workers that have been sensitized to nickel can develop pigmented or depigmented plaques on the skin. One worker that had developed cutaneous sensitization also developed asthma from inhalation of nickel sulfate (Proctor and Hughes, 1988).

SENSITIZATION: The most prevalent effect in humans from exposure to nickel is contact dermatitis. Once sensitized, even minor contact with nickel containing metals will result in a reaction (ATSDR, 1987). Sensitization to nickel can occur by contact with cheap jewelry including ear ring posts, wristwatches buckles and buttons, as well as dental braces, scissors, and kitchen utensils. Occupational exposure to nickel aerosols has been associated with asthma, acute pneumonitis and contact hypersensitivity (Sunderman, 1977).

TARGET ORGAN EFFECTS: The skin is the principal target organ of toxicity to nickel although kidney effects and respiratory effects may also be involved. In addition, increased risk of lung and nasal cancers has been reported for workers in the nickel smelting and refining industry and electroplating shops.

ABSORPTION-METABOLISM-EXCRETION: In animals, 1 to 10% of the dietary nickel is absorbed by the gastrointestinal tract (Horak and Sunderman, 1973). Blood levels of nickel increase in fasted subjects when the nickel is administered in drinking water but when the nickel is administered with meals, the blood levels did not rise (ATSDR, 1987).

Absorption of nickel compounds by inhalation varies. Nickel carbonyl is readily absorbed in rats (Sunderman and Selin, 1968) but nickel oxide, as particulate material has negligible absorption in hamsters and rats (Leslie et al., 1976; Kodama et al., 1985). In mice, about 75% of

nickel chloride aerosol is cleared within 4 days (Graham et al., 1978).

Dermal absorption can also occur but the extent of absorption appears to be governed by the solubility of the compound. Fullerton et al., (1986) found that nickel chloride is absorbed approximately 50 times faster than nickel sulfate but only 0.23% of the nickel chloride penetrated in 144 hrs when the skin was not occluded. With occlusion, 3.5% of the applied dose was absorbed.

Once absorbed, soluble nickel compounds are distributed to the kidney, liver, and lungs in rabbits and rats. Insoluble nickel compounds are retained at the site of administration but particles can be phagocytized by the reticuloendothelial system (Oskarsson and Tjalve, 1979). Human autopsy examinations showed that the only age dependent accumulation of nickel in soft tissue occurs in the lungs (EPA, 1986).

GENOTOXICITY: Nickel and its compounds have been extensively tested for genotoxicity. Tests for gene mutations have produced equivocal results while tests for DNA damage, sister chromatid exchange, and chromosomal aberrations and transformations have been mostly positive. In contrast, *in vivo* genotoxic studies such as chromosomal aberrations, micronuclei formation and dominant lethality in mice and rats as well as lethality and mutations in Drosophila melanogaster have been negative (ATSDR, 1987).

IMMUNOTOXICITY: In mice, natural killer lymphocytes (NK cells) and T lymphocytes in the spleen were reported to be depressed within 24 hours following a single intramuscular injection of nickel chloride at a dose level of 18.3 mg/kg (Smialowicz, 1985). Parenteral administration of soluble nickel compounds decrease antibody forming cell response in mice (Figoni and Treagan, 1975), thymic involution in mice and rats (Knight et al., 1987) and spleen cell proliferative responses to mitogens in mice (Dieter et al., 1988).

Haley et al. (1990) obtained similar results with nickel compounds of varying solubility and inhalation exposures to mice and rats. The immunotoxic activity appeared to depend upon the form, solubility and dose of the compound with relative activity of $NiSO_4$ > Ni_3S_2 > NiO.

REPRODUCTIVE TOXICITY: In mice, nickel chloride in the drinking water at 1000 ppm during the period of gestation reduced the maternal weight gain, reduced fetal weight and increased the incidence of spontaneous abortions. At 500 ppm (NOAEL) no adverse effects were observed (Berman and Rehnberg, 1983).

In a two generation rat study, again with nickel chloride in the drinking water, statistically significant effects on maternal weight, pup mortality and incidence of short ribs, were only observed in the 500 ppm group. Therefore 250 ppm was taken as a NOAEL (RTI, 1987).

Inhalation of nickel sulfate by rats resulted in degeneration of the germinal epithelium at an exposure of 1.6 mg Ni/m^3. No damage was seen at 0.7 mg Ni/m^3. Mice similarly exposed did not show any effects (Benson et al,1988). However, both rats and mice showed testicular degeneration with exposure to nickel subsulfide at 1.8 mg Ni/m^3. The NOAEL was reported as 0.9 mg Ni/m^3 (Benson et al., 1987).

NEUROTOXICITY: Early symptoms after inhalation of nickel compounds are dizziness, giddiness and weakness (HSDB, 1990). Inhalation of nickel carbonyl has been reported to produce profound neuropathology including edema and diffuse punctate hemorrhages in the cerebrum, cerebellum and brain stem with degeneration of neural fibers (NAS, 1975).

CARCINOGENICITY: Most information regarding the potential carcinogenicity of nickel is derived from studies of nickel refinery workers. The largest and most comprehensive of these studies have followed workers in Wales, Canada, Norway, and the United States.

Doll et al. (1970 and 1977) presented the mortality experience of nickel refinery workers in Clydach, Wales. The 1977 study followed 967 men from 1934-1971. Significantly elevated risks of lung cancer and nasal cancer were found among those hired before 1930. The standard mortality ratio (SMR) for lung cancer was 623 (observed/expected deaths 137/21.98) and for nasal cancer 28,718 (observed/expected 56/0.195). No case of nasal cancer occurred among those entering employment after 1930, and lung cancer rates dropped steeply after this date (SMR 146, non-significant, for those hired 1930-1944).

The largest and most recent study of Canadian refinery workers was reported by Roberts et al. (1982). This study of 54,724 workers included data on Copper Cliff workers reported by Chovil et al. in 1981 and Port Colborne workers studied by Sutherland et al. (1969, 1971). Consistent with studies of Clydach workers, excesses in nasal and lung cancer were observed among the Canadian workers. Among sinter facility workers, the SMRs for nasal cancer

ranged from 1500-8000, while lung cancer SMRs ranged from 279-424.

Pedersen (1973) studied Norwegian refinery workers beginning employment between 1953-1961. Significant increases in nasal cancer (SMR 2800, observed/expected 14/0.5) and lung cancer (SMR 475, observed/expected 48/10.1) were found. After classifying workers by the longest-held job, the cancer risk was greatest for smelting, roasting, and electrolysis workers.

The Pedersen study was updated through 1970 by Magnus et al. (1982). Again, an excess in nasal cancer and lung cancer deaths were found: nasal cancer SMR=2800 (observed/expected deaths 21/0.753) and lung cancer SMR=380 (observed/expected deaths 82/21.85). While the risk of nasal cancer was noted to decline over time, no such declining risk of lung cancer was observed during the period 1930-1960.

Enterline and Marsh (1982) studied mortality patterns in 1,855 nickel refinery workers in Huntington, West Virginia. Workers were first employed before 1948 (while calciners were in operation) and followed 1948-1977. Concentrations of airborne nickel in areas where the copper-nickel matte was crushed and handled were estimated to range from 20-350 mg/m3; concentrations around the calciners were estimated to range from 5-15 mg/m^3. Four cases of nasal cancer were observed. Lung cancer mortality did not show a significant excess, but a slight trend of increasing lung cancer risk by cumulative nickel exposure (mg/m^3 nickel-months) was observed.

EPIDEMIOLOGY: Based on epidemiologic data, inhalation of nickel and nickel compounds has been associated with asthma, upper respiratory tract irritation, chronic rhinitis, sinusitis, and an increased risk of respiratory tract infections. Impairment of the sensation of smell and nasal septal perforation has been noted in cross-sectional studies of workers exposed to high levels of nickel and nickel compounds.

Cohort mortality studies of nickel refinery workers have found a consistent association between exposure to nickel refinery dust and nickel subsulfide and cancer of the lung and nasopharynx. These studies are reviewed under CARCINOGENICITY.

The carcinogenic potential of nickel compounds was reexamined with additional and up-to-date reviews by the International Committee on Nickel Carcinogenesis in man (Doll, 1990). This committee concluded that respiratory cancer risks are primarily related to exposure to soluble nickel compounds at concentrations in excess of 1 mg Ni/m^3. Exposure to the less soluble forms of nickel compounds at concentration greater than 10 mg/m3 are also associated with an excess cancer risk. However, there is no evidence for increased cancer risk from exposure to metallic nickel.

ENVIRONMENTAL FATE: The levels of nickel in seawater ranges from 0.1 to 0.5 µg/l. Nickel is seldom found in ground water and in the U.S. drinking water-supplies generally contain nickel levels below 20 µg Ni/l. However, in some nickel mining areas, drinking water levels have been reported to go as high as 200 µg/l (McNeeley et al., 1972).

At pH levels less than 6.5, most nickel compounds are relatively soluble. At pH values greater than 6.5 the nickel may exist predominantly as a hexahydride ion which is poorly absorbed by living organisms. In river and surface waters, half the nickel is in an ionic form and the other half exists as stable organic complexes which become adsorbed on bottom sediments. In addition, since many of the inorganic compounds of nickel are water insoluble, much of the nickel in water and soil is not bioavailable (ATSDR, 1987).

The elemental concentration of nickel in natural soils ranges from 5 - 500 ppm with an average value of 40 ppm (Shields, 1985). In farm soils, nickel concentrations range between 3 and 1000 ppm (Norseth, 1986).

Ambient air levels in highly industrialized areas and large cities can average 150 ng Ni/m^3. Urban areas may have yearly averages between 178 - 25 ng/m^3 while non urban areas are in the range of 6 ng/m^3 (NAS, 1975). Nickel carbonyl has a half life of about 100 sec at ambient air conditions (EPA, 1986).

ENVIRONMENTAL TOXICITY: Dietary intake of nickel typically contributes between 80 to 94% of the body burden of nickel. Nuts, seeds,cocoa and grains are typically high in nickel content with concentration levels up to 6-7 ppm. Fruits and vegetables range from 0.0 - 2.5 ppm and seafood from 0.3 to 1.7 ppm. The estimated daily intake of nickel in the U.S. is between 0.3 - 0.5 mg/d (Norseth, 1986).

Nickel is an essential nutrient in urease-rich plants such as jackbeans and soybeans. In these plants, nickel concentration can attain levels of 10 g/kg in leaves and 250 g/kg in the sap (ATSDR, 1987). Nickel has also been found to be an essential trace element in chickens, cows, goats, minipigs, rats and sheep. States of nickel deficiency have produced decreased weight

gain, liver damage, disturbed metabolism and decreased iron absorption efficiency (EPA, 1986).

Nickel is a component of cigarettes and tobacco smoke. The average nickel content of cigarettes is 2.2 - 2.3 µg/cigarette. About 10 - 20% of the nickel is released into the main-stream smoke and is in the gaseous phase. This and other data suggest that the nickel might therefore be in the carbonyl form (NAS, 1975; Norseth, 1986).

REGULATORY STATUS: The following table presents the recommended and enforceable standards for nickel in the workplace. The World Health Organization has not published a water quality limit for nickel but the Ambient Water Quality Criterion (AWQC) according to the EPA is 632 µg/l for the protection of human health and 4.77 mg/l for the protection of human health through contaminated aquatic species. The recommended RfD is 0.02 mg/kg/d based on a 5 mg/kg NOAEL; a 100 uncertainty factor and a 3 modifying factor. On the basis of epidemiological data, the Carcinogen Assessment Group (CAG) has calculated a unit risk value of 2.4×10^{-4} $(\mu g/m^3)^{-1}$ for nickel refinery dust and 4.8×10^{-4} $(\mu g/m^3)^{-1}$ for nickel subsulfide. IARC has classified "nickel refining" as a Group 1 carcinogen. Certain other nickel compounds such as nickel powder, subsulfide, oxide, hydroxide, carbonate, carbonyl, nickelocene, nickel iron sulfide matte, and nickelous acetate have been classified by IARC as Group 2A carcinogens which means the evidence in humans is limited but the evidence in animals is sufficient to support this classification (ATSDR, 1987).

TABLE 3. Worker's Exposure Limits

ACGIH TWA:	1 mg/m³ (soluble compounds) 0.1 mg/m³ (insoluble compounds)
STEL:	Ca
CEILING:	Ca
OSHA PEL:	1 mg(Ni)/m³
CEILING:	Ca
IDLH:	Ca
NIOSH REL:	10 H TWA 0.015 mg(Ni)/m³
MSHA:	1 mg/m³

Ca - carcinogen. Lowest feasible level should be maintained.
(Reference: ATSDR, 1987)

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TOXICOLOGY PROFILE

POLYNUCLEAR AROMATIC HYDROCARBONS

Synonyms: Varies depending on specific compound. For example,

Acenaphthene (CAS No 83-32-9) is also known as:

1,2-dihydroacenaphthylene;
1,8-dihydroacenaphthaline;
1,8-ethylenenaphthalene; $C_{12}H_{10}$.

Acenaphthylene (CAS No 208-96-8) is also known as:

Cyclopenta [d,e] naphthalene; $C_{12}H_8$.

Anthracene (CAS No 120-12-7) is also known as:

Anthracin; green oil; paranaphthalene; Tetra olive NZG, Anthracene oil; $C_{14}H_{10}$.

Benzo[a]anthracene is also known as:

Benz[a]anthracene; 1,2-benzanthracene; benzo[b]phenanthrene; 2,3-benzophenanthrene; tetraphene; $C_{18}H_{12}$.

Benzo[a]pyrene is also known as:

Benzo[d,e,f]chrysene; 3,4-benzopyrene; 3,4-benzpyrene; benz[a]pyrene; BP; B[a]P; $C_{20}H_{12}$.

Benzo[b]fluoranthene is also known as:

3,4-Benz[e]acephenanthrylene; 2,3-benzfluoranthene; 3,4-benzfluoranthene; 2,3-benzofluoranthene; 3,4-benzofluoranthene; benzo[e]fluoranthene; B[b]F; $C_{20}H_{12}$.

Benzo[k]fluoranthene is also known as:

8,9-benzfluoranthene; 8,9-benzofluoranthene; 11,12-benzo-fluoranthene; 2,3,1,8-binaphthylene; dibenzo[b,j,k]fluorene; $C_{20}H_{12}$.

Benzo[g,h,i]perylene is also known as:

1,12-benzoperylene; $C_{22}H_{12}$.

Chrysene (CAS No 218-01-9) is also known as:

1,2-Benzophenanthrene; benzo[a]phenanthrene; 1,2-benzphenanthrene; benz[a]phenanthrene; 1,2,5,6-dibenzo-naphthalene; $C_{18}H_{12}$.

Dibenz[a,h]anthracene is also known as:

1,2,5,6-Dibenz[a,h]anthracene; Dibenzo[a,h]anthracene, DB[a,h]A; DBA; 1,2:4,6-dibenz[a,h]anthracene; 1,2:5,6-dibenz[a]anthracene; $C_{22}H_{14}$.

Fluoranthene (CAS No 206-44-0) is also known as:

1,2-(1,8-naphthylene)benzene; 1,2-benzacenaphthene; 1,2-(1,8-naphthalenediyl)benzene; benzo[j,k]fluorene; $C_{16}H_{10}$.

Fluorene (CAS No 86-73-7) is also known as:

ortho-Biphenylene methane; diphenylenemethane; 2,2-methylene biphenyl; 2,3-benzidene; $C_{13}H_{10}$.

Indeno[1,2,3-c,d]pyrene is also known as:

1P; *ortho*-phenylenepyrene; 1,10-(*ortho*-phenylene)pyrene; 1,10-(1,2-phenylene)pyrene; 2,3-*ortho*-phenylene pyrene; indenopyrene; $C_{22}H_{12}$.

Phenanthrene (CAS No 85-01-8) is also known as:

Phenantrin, $C_{14}H_{10}$.

Pyrene (CAS No 129-00-0) is also known as:

B-pyrene; benzo[d,e,f]phenanthrene; $C_{16}H_{10}$.

CAS No: Refer to individual compounds.

Boiling point range: 96.2°C to 530°C.

Color: Colorless, white, yellow, slight blue fluorescence.

Conversion factor: For Benzo[a]pyrene, 1 ppm = 10.32 mg/m^3.

Flammable limits

autoignition: For anthracene, 540°C

flash point: For phenanthrene, 171°C; for anthracene, 250°F.

Henry's law constant: 5.34 x 10^{-8} - 1.02 x 10^{-3} atm-m^3/mole

Melting point range: 80°C to 270°C (generally increases with increasing molecular weight).

Molecular weight: 116 to 278

Odor threshold: For anthracene and phenanthrene, faint aromatic odor.

For acenaphthene, water = 0.08 ppm with a range of 0.02- 0.22 ppm; air = 8.0 x 10^{-2} ppm.

Solubility

Water: 0.0003 mg/l to 34 mg/l (generally decreases with decreasing molecular weight).

K_{ow}: 3.4 to 7.6 (generally increases with increasing molecular weight).

K_{oc}: 1000 ml/g to 5.5 x 10^6 ml/g

Vapor pressure: 10^{-10} to 10^{-2} mm Hg at 20°C (generally decreases with decreasing molecular weight).

(References: ATSDR, 1989; Clement, 1985.)

COMPOSITION: Polycyclic aromatic hydrocarbons (PAHs) consist of joined aromatic rings (benzene) that do not have carbon substitution within the ring structure of the side chains. For example, nitrogen substituted cyclic compounds are classified as amines and are not considered in the category of PAHs. The number of rings appears to afect carcinogenic potential. In general, it is the four aand five ring compounds that are likely to be carcinogenic while the two and three ring compounds or greater than five ring compounds are not likely to exhibit tumor production activity. However, as with all axioms, there can be exceptions.

USES: PAHs such as benzo[a]pyrene, pyrene, benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo-[g,h,i]perylene, dibenzo[a,h]anthracene, and indeno[1,2,3-c,d]pyrene have no known use except as research chemicals. Anthracene is used as an intermediate in dye production, for smoke screens, scintillation counter cocktails, and in organic semiconductor research. Acenaphthene is used as an insecticide, fungicide, and as a dye intermediate. Fluorene is used as a chemical intermediate in the formation of polyradicals and fluoranthene is used as a lining material to protect the interior of steel, ductile-iron drinking water pipes, and storage tanks. Phenanthrene is used in the manufacture of dyestuffs and explosives and in biological research (ATSDR, 1989).

TOXICITY: Polynuclear aromatic hydrocarbons (PAHs) can be classified into two categories of effects: potentially carcinogenic effects and noncarcinogenic effects. The toxicity section will discuss PAHs in these two categories.

CARCINOGENIC EFFECTS:

Initiation and/or promotion of cancer, either systemically, by ingestion or inhalation, or at a point of dermal contact is a toxic effect of a subset of PAHs. PAHs with carcinogenic effects generally tend to be high in molecular weight, have at least three aromatic rings (usually more), have low water solubility, are easily absorbed by humans, and have very low acute toxicity. The following PAHs have caused cancer in laboratory animals through ingestion, dermal contact, or inhalation:

benz[a]anthracene,
benzo[a]pyrene
benzo[b]fluoranthene
benzo[k]fluoranthene
chrysene
dibenzo[a,h]anthracene
indeno[1,2,3-c,d]pyrene

Reports of potential effects in humans show that individuals exposed by inhalation or dermal contact for long periods of time to mixtures of other compounds and PAHs such as soots and tars, may also develop cancer. Many of the toxicological studies cited in this profile did not focus on one specific chemical. Many of the studies involved two or more chemicals and evaluated potentially carcinogenic and noncarcinogenic PAHs together. Because of these mixtures in the studies, it may be difficult to determine exactly which chemical causes specific effects.

Ingestion: Mice fed benzo[a]pyrene in the diet exhibited gastric neoplasms at concentrations equivalent to 33.3 mg/kg/day. Lower concentrations of benzo[a]pyrene, equivalent to 13.3 mg/kg/day, administered for up to 7 days, however, did not produce forestomach tumors. Mammary tumors were induced by an extremely large single oral dose of 100 mg benzo[a]pyrene in 88% female Sprague-Dawley rats. After a single oral dose of 50 mg benzo[a]pyrene, a 77% mammary tumor incidence was observed in 90 weeks (ATSDR, 1987; 1989).

The subchronic administration of dietary benzo[a]pyrene at various doses up to 250 ppm for 30 to 197 days showed a relationship between the duration of oral exposure and the incidence of forestomach tumors in mice. As the dose increased, the tumor incidence increased also. In the same study, mice fed 250 ppm for periods of 1 to 7 days exhibited increased forestomach tumor incidences following 2 or more days of benzo[a]pyrene exposure, while mice fed 10 ppm for 110 days did not develop tumors. These findings suggest evidence that cumulative carcinogenic effects of benzo[a]pyrene or its metabolites in mice do not exist (ATSDR, 1987; 1989).

Various strains of mice receiving dibenzo[a,h]anthracene emulsions in mineral oil or olive oil in place of drinking water at a dose approximately equal to 25 mg/kg/day throughout their lifetimes developed lung adenomas, papillomas and squamous cell carcinomas in the fore-stomach. However, the animals did not tolerate the vehicle well and extensive emaciation and dehydration occurred but the results indicate that benzo[a]anthracene, benzo[a]pyrene, and dibenzo[a,h]anthracene, and possibly other PAHs, are potentially carcinogenic by the oral route of exposure (ATSDR, 1989). Chronic oral administration of a total dose of 4.5 g/rat anthracene in the diet to BD1 or B111 rats for 78 weeks did not produce tumors, suggesting that anthracene is noncarcinogenic in animals following chronic oral exposure (ATSDR, 1989).

Inhalation: Epidemiologic studies have shown increased mortality due to lung cancer in humans exposed to coke-oven emissions, roofing-tar emissions, and cigarette smoke. Benzo[a]pyrene, chrysene, benz[a]anthracene, benzo[b]fluoranthene, dibenzo[a,h]anthracene along with other potentially carcinogenic PAHs and other chemicals that may be tumor promoters, initiators, and cocarcinogens are present in these mixtures. Because of the complexity of these mixtures, it is impossible to evaluate the contribution of any individual PAH to the total carcinogenicity of these mixtures. Reports of this nature, despite their limitations, provide qualitative evidence that mixtures containing PAHs are carcinogenic (ATSDR, 1989).

One inhalation study for animals provides evidence of a dose-response relationship between inhaled benzo[a]pyrene particles (99% of the benzo[a]pyrene particles were between 0.2 and 0.54 microns in diameter) and respiratory tract tumorigenesis. For hamsters exposed to 9.5 mg/m^3 and 46.5 mg/m^3 for 109 weeks, respiratory tract tumors were induced in the nasal cavity, pharynx, larynx, and trachea in a dose-related manner. Following exposure to 46.5 mg/m^3, tumors were also observed in the esophagus and forestomach (ATSDR, 1987; 1989).

Rats exposed to 10 mg/m^3 benzo[a]pyrene in the presence of sulfur dioxide (SO_2) for 1 hour/day for 1 year exhibited increased incidences of squamous cell carcinomas in the lungs compared to rats exposed to 10 mg/m^3 benzo[a]pyrene alone. By itself, sulfur dioxide does not have carcinogenic effects. Benzo[a]pyrene, and possibly other PAHs, as indicated by these results, may be carcinogenic following inhalation exposure. Carcinogenicity is potentially enhanced by concurrent exposure to gases and particulates commonly found in the environment (ATSDR, 1987; 1989).

Skin Contact: Chronic dermal exposure to soots containing PAHs were associated with an increased incidence of scrotal cancers among 18th century British chimney sweeps (Shimkin, 1987).

Studies in laboratory animals have shown benz[a]anthracene, benzo[b]fluoranthene, benzo[a]pyrene, chrysene, and dibenzo[a,h]anthracene induce skin tumors following intermediate dermal exposure.

Anthracene, fluoranthene, fluorene, indeno(1,2,3-cd)pyrene, phenanthrene, and pyrene do not have carcinogenic effects (ATSDR, 1989). These conclusions are supported by data discussed in the following paragraphs for each chemical.

Anthracene: Swiss mice receiving 10% anthracene in acetone topically applied to their backs three times a week throughout a lifetime did not develop skin tumors after 20 months. Anthracene has also been found to be inactive as an initiating agent (ATSDR, 1989).

Benz[a]anthracene: For 50 weeks, graded concentrations of benz[a]anthracene in toluene or n-dodecane was topically applied to the backs of mice resulting in a dose-related increase in tumor incidence. When toluene was the solvent, malignant tumors were observed at dose levels of approximately 0.02% benz[a]anthracene. In comparison, when n-dodecane was the solvent, tumors were observed at concentrations of 0.0002% benz[a]anthracene (ATSDR, 1989).

Benz[a]anthracene has been observed to be a tumor initiator in a subchronic dermal study where CD-1 mice exhibited an increased skin tumor incidence (36%). Benz[a]anthracene at a concentration of 0.57 mg was topically administered followed by promotion with tetradecanoyl phorbol acetate (TPA) for 25 weeks (ATSDR, 1989).

Benzo[b]fluoranthene: A dose-response relationship for the dermal carcinogenicity of benzo[b]fluoranthene has been demonstrated over an order-of-magnitude dose range in Swiss mice receiving (0.01 to 0.5%) benzo[b]fluoranthene throughout their lifetime. Malignant tumors (90% carcinomas) appeared as early as 4 months in the high dose benzo[b]fluoranthene group. In the intermediate-dose group, papillomas and carcinomas (65 and 85%, respectively) appeared after five months. This study provides evidence that benzo[b]fluoranthene is carcinogenic following intermediate-duration exposure. The lowest dose at which benzo[b]fluoranthene elicited malignant tumors was 0.1%, or approximately an average daily dose of 1.2 mg/kg. Initiation doses of benzo[b]fluoranthene (10 to 100 µg) followed by TPA for 20 weeks dermally applied to the backs of CD-1 mice elicited a dose-related skin tumor incidence (ATSDR, 1989).

Benzo[a]pyrene: A dose-response relationship for skin tumors was exhibited in mice receiving 0.001 to 0.01% benzo[a]pyrene dermally applied to their backs throughout their lifetimes (ATSDR, 1987; 1989). Benzo[a]pyrene is an active tumor initiator using initiation/promotion protocols. An enhanced incidence (80-92%) of skin papillomas resulted from a topical application of a single initiation dose of benzo[a]pyrene to the backs of mice followed by promotion with TPA or croton oil. Ten doses of benzo[a]pyrene (0.1 mg/dose) topically applied to the backs of Swiss mice followed by promotion with croton oil (for 20 weeks) resulted in the development of skin tumors (ATSDR, 1987; 1989).

Benzo[a]pyrene is a potent experimental skin carcinogen and it is often used as a positive control in bioassays of other agents (ATSDR, 1987; 1989).

Benzo[k]fluoranthene: Although not as potent as benzo[b]fluoranthene, benzo[k]fluoranthene has been demonstrated to be a tumor initiator in mice. A single application of 11 mg of benzo[k]fluoranthene applied dermally to Swiss mice followed by promoting treatments with croton resin enhanced the incidence of papillomas and carcinomas (90 and 25%, respectively). However, no tumor induction was observed without a promoting agent as benzo[k]fluoranthene alone is not a complete carcinogen. In a different study, NMRI mice painted with up to 9.2 µg benzo[k]fluoranthene for a lifetime showed no significant increase in tumor incidence (ATSDR, 1989).

Chrysene: Topical application of chrysene in a n-dodecane/decalin solution to the skin of mice produced a significant increase in the carcinogenic potency of chrysene compared with that obtained using decalin alone; 26 and 63% of mice exhibited papillomas and carcinomas, respectively, at 49 weeks. Decaline and n-dodecane have been shown in other experiments to be noncarcinogenic in mice (ATSDR, 1989).

Initiating doses of chrysene followed by promotion with TPA or croton resin induced a dose-related papilloma incidence in mice. Ten daily treatments of chrysene to Swiss mice followed by TPA promotion for 20 weeks resulted in an enhanced incidence of papillomas and carcinomas (61%) compared to the chrysene treatment group. Chrysene as an initiating agent is noteworthy since it occurs in complex mixtures of chemicals that may include tumor promoters (ATSDR, 1989).

Dibenzo[a,h]anthracene: Swiss mice treated throughout their lifetimes with concentrations

of 0.001 - 0.1% dibenzo[a,h]anthracene applied to their backs exhibited dose-related papilloma and carcinoma incidences at the site of application at the two lowest doses. The lowest concentration was 0.001%, approximately equal to 0.012 mg/kg/day. In another chronic dermal study, a dose-related increase in skin carcinoma formation was observed, as well as decreased survival time and tumor latency period (ATSDR, 1989).

Fluoranthene: Chronic dermal application of up to 1% fluoranthene to the backs of mice did not induce skin tumors following a lifetime of application. Topically administered doses (10) of fluoranthene followed by promotion with croton oil did not exhibit initiation activity in Swiss mice (ATSDR, 1989).

Fluorene: Fluorene has been reported to be negative as a complete carcinogen and as a cocarcinogen with 3-methylcholanthrene. It is also inactive as a tumor initiator when an estimated total dose of 1 mg was applied prior to TPA application (ATSDR, 1989).

Indeno[1,2,3-c,d]pyrene: Chronic dermal application of indeno[1,2,3-c,d]pyrene in dioxane to mice did not produce an increased incidence of skin tumors. However, a dose-related increase in tumor incidence was observed at 9 months when acetone was used as the solvent. No tumor induction was reported after chronic topical application of up to 9.2 µg of indeno[1,2,3-c,d]pyrene in acetone was applied to the backs of mice. The induced carcinogenicity of indeno[1,2,3-c,d]pyrene appears to vary with the solvent employed for delivery (ATSDR, 1989).

Indeno[1,2,3-c,d]pyrene was observed to have tumor initiating activity at repeated doses of 250 µg followed by promotion with croton oil (ATSDR, 1989).

Phenanthrene: Phenanthrene tested negative as a complete carcinogen in two mouse studies that lacked adequate reporting (ATSDR, 1989). Phenanthrene was ineffective as an initiator and also was inactive as a tumor promoter (ATSDR, 1989).

Pyrene: Mice chronically administered a 10% pyrene solution throughout their lifetimes did not develop skin tumors. Pyrene has been shown to be inactive as an initiation agent (ATSDR, 1989).

RELATIVE POTENCY: Benzo[a]pyrene is the most intensely studied PAH and has been used as the surrogate PAH for evaluating other carcinogenic PAHs. However, only dibenz[a,h] anthracene has been shown to be more potent than B[a]P. All of the other PAHs which have available test data (genotoxici or carcinogenic) were found to be less potent. Several authors (Clement Associates, 1988; Rugen et al., 1989; Krewski et al., 1989) have ranked the relative potency of a number of carcingenic PAHs similar to the values presented in Table 1.

Table 1. Relative Potency of PAH Compounds

PAH	Relative carcinogenicity	Relative mutagenicity
Dibenz[a,h]anthracene	1.11	0.47
Benzo[a]pyrene	1.0	1.0
Anthanthracene	0.320	0.06
Indeno[1,2,3-cd]pyrene	0.232	0.14
Benz[a]anthracene	0.145	0.62
Benz[b]fluoranthene	0.141	0.20
Benz[k]fluoranthene	0.066	--
Benz[j]fluoranthene	0.061	--
Pyrene	0.081	0.20
Cyclopentadienol[cd]pyrene	0.023	0.26
Benzo[ghi]perylene	0.022	0.08
Chrysene	0.0044	0.37
Benzo[e]pyrene	0.004	0.42

(ref: Collins et al., 1991).

NONCARCINOGENIC EFFECTS:
Many PAHs have been assayed for carcinogenicity with negative results. Of those, naphthalene and fluoranthene are the most highly studied, and these are often used as surrogates to model the behavior and toxicity of other noncarcinogenic PAHs. PAH compounds which have not been demonstrated to be carcinogenic include:

Acenaphthalene
Acenaphthene
Anthracene
Benz[a]acridine
Benzo[a]fluorene
Benzo[b]fluorene
Benzo[c]fluorene
Benzo[g,h,i]perylene
Benzo[c]phenanthrene
Benzo[e]pyrene
Coronene
Dimethyl naphthalenes
1,4 Dimethylphenanthrene
Fluoranthene
Fluorene
1 methylchrysene
3 methylfluoranthene
1-methylphenanthrene
1-Methyl naphthalene
3-Methyl naphthalene
Naphthalene
Perylene
Phenanthrene
Pyrene
Triphenylene

(ref: IARC, 1987)

Many carcinogenic PAHs may also produce noncarcinogenic health effects through various routes of exposure. Typically, carcinogenic health effects take precedent over noncarcinogenic health effects when considering human health impacts from chemical exposure. Noncarcinogenic effects are considered below.

Ingestion: Minimal information is available on the gastrointestinal effects of human oral exposure to PAHs. In one study, humans that consumed anthracene-containing laxatives (no specific concentration) for prolonged periods of time were found to have an increased incidence of melanosis of the colon and rectum. However, no definite conclusions can be drawn because of study limitations (ATSDR, 1989).

Mice administered increasing dietary doses of pyrene ranging from 1000 mg/kg food up to 25,000 mg/kg food over a 25 day study exhibited dilation of the renal tubules in an unspecified number of mice. These effects were not observed until the highest dose was administered (ATSDR, 1989).

Benzo[a]pyrene, benz[a]anthracene, and chrysene are moderate inducers of hepatic carboxylesterase activity in rats intragastrically administered 50, 100, 150 mg/kg/day, respectively for 4 days (ATSDR, 1989).

The kidney microsomal carboxylesterase activity of rats was moderately induced by 50-150 mg/kg of benzo[a]pyrene following 4 days of intragastric administration. However, rats administered 100 mg/kg/day of anthracene or phenanthrene did not exhibit increased activity (ATSDR, 1989).

Inhalation: No studies were available in the references consulted regarding noncarcinogenic health effects in humans or animals following inhalation exposure to any of the PAHs discussed in this profile.

Skin Contact: Over a period of 4 months, 1% benzo[a]pyrene was applied to human skin up to 120 times causing regressive variceal to occur. Although this study is flawed in that no benzene control was evaluated, the changes were thought to represent neoplastic proliferation. Rhoads et al. (1954) reported similar epidermal changes in humans exposed to benzo[a]pyrene. For 4 consecutive days, human volunteers were painted on 1 cm^2 areas of the upper back. Nucleolar enlargement was observed (ATSDR, 1987; 1989).

Adverse dermal effects were observed in humans following subchronic dermal exposure to benzo[a]pyrene in patients with preexisting dermal conditions of pemphigus vulgaris and xeroderma pigmentosum. Local bullous eruptions characteristic of pemphigus resulted when a 1% benzo[a]pyrene solution was topically applied to patients with the disease. Only pigmentary and slight verrucous effects were observed from patients with xeroderma pigmentosum exposed to 1% benzo[a]pyrene (ATSDR, 1987; 1989).

An enhanced dermal inflammation resulted from an acute (96-hour) dermal application of anthracene to the backs of hairless mice followed by ultraviolet radiation (UVR) exposure for 40 minutes. However, this effect was reversed within 48 hours. Anthracene may be considered a photosensitizer in hairless mice by potentiating skin damage elicited by sunlight exposure (ATSDR, 1989).

SENSITIZATION: Specific subsections of the population may be susceptible to the toxic effects produced by exposure to PAHs which include the unborn, people who smoke, people

with a history of excessive sun exposure, and people with skin and liver diseases. People with numerous conditions such as genetically inducibility aryl hydrocarbon hydroxylase (AHH) activity, nutritional deficiencies, genetic diseases that influence the efficiency of DNA-repair, and immunodeficiency due to age or disease may also be susceptible to PAH exposure (ATSDR, 1989).

The unborn are susceptible to the toxic effects produced by maternal exposure to PAHs, such as benzo[a]pyrene because of an increased permeability of the embryonic and fetal blood-brain barrier and a decreased liver-enzyme conjugating function (ATSDR, 1987; 1989).

Certain nutritional deficiencies have been associated with an increased cancer incidence in PAH-exposed animals, including deficiencies in Vitamins A and C, iron, and riboflavin. The interaction between nutrition and PAH exposure by administrating benzo[a]pyrene to experimental animals has been studied. Nutritional factors either reduced the activity of AHH, reduced the amount of benzo[a]pyrene binding to DNA in rat liver or stomach tissue, or prevented or reversed genetic damage (ATSDR, 1989).

People exposed to PAHs in conjunction with particulates from tobacco smoke, fossil-fuel combustion, coal fly ash, and asbestos fibers are at an increased risk of developing toxic effects, primarily cancer. The adsorption of PAH onto particulates results in a synergistic action, transporting more efficiently through membranes, clearing from tissues more slowly, and distributing into tissues differently depending on the size and type of particulate matter (ATSDR, 1989).

People with significant exposure to ultraviolet (UV) radiation may be at an increased risk of developing skin cancer due to PAH exposure. A synergistic influence on PAH-induced skin cancer following dermal exposure may originate from UV radiation. Experimental animals were at a higher risk of skin tumor induction when exposed concurrently to chronic UV irradiation and PAHs (ATSDR, 1989).

Subsections of the population that suffer from liver and skin diseases may be at an increased risk of developing adverse effects due to PAH exposure. Increased hepatic toxicity may occur through exposure to PAHs by inducing enzyme alterations, preneoplastic changes in hepatocytes referred to as gamma-GT foci, hepatic regeneration, and increasing hepatic weight. Following exposure to some PAHs, people with pre-existing skin conditions and those with normal skin may be at an increased risk of developing adverse dermal effects ranging from rashes to cancer and exposure to more than one PAH may enhanced tumor development (ATSDR, 1989).

ABSORPTION:

Ingestion: Oral absorption of benz[a]anthracene in rats is rapid and efficient, reaching a maximum in the brain, liver, and blood within 1-2 hours after administration (ATSDR, 1989).

The intestinal absorption of PAHs is highly dependent on the presence of bile in the stomach. Conscious rats with bile duct and duodenal catheters were given isotopically labeled benzo[a]pyrene, phenanthrene, anthracene, 2,6-dimethyl naphthalene (DMN), and 7,12-dimethyl-benzo[a]anthracene (DMBA) in corn oil with or without exogenous bile. PAH absorption efficiency was estimated from the cumulative recovery of radioactivity in the bile and urine over 24 hours. The efficiencies without bile were: benzo[a]pyrene, 22.9%; phenanthrene 96.7%; anthracene 70.8%; DMN, 91.6%; DMBA 43.4%. Absorption of benzo[a]pyrene and DMBA was strongly dependent on the presence of bile in the intestinal lumen. The absorption of anthracene and phenanthrene differed with respect to their dependency on bile for efficient absorption which correlates with a difference in water solubility. Products with low water solubility are dependent on the creation of an intermediate phase of lipolysis and bile salt products. PAH absorption is enhanced when solubilized in a vehicle that is readily absorbed such as oils (ATSDR, 1989).

Inhalation: The size of particles on which benzo[a]pyrene is adsorbed affects pulmonary absorption and elimination. In one study, benzo[a]pyrene (0.6 µg/l) adsorbed onto Ga_2O_3 particles was administered as an aerosol to rats. A control study was conducted without the Ga_2O_3 particles at a concentration of 1 µg/l. The fraction of the total amount of aerosol particles deposited in the lung was approximately 20% for Ga_2O_3 and approximately 10% for the pure hydrocarbon aerosol after an exposure time of 30 minutes. Nearly all of the radioactivity was recovered over a period of 2 weeks or more, indicating complete absorption of the initially instilled hydrocarbon. A subsequent amount of benzo[a]pyrene coated on Ga_2O_3 particles was removed from the lungs by mucociliary clearance and subsequent ingestion. The pure benzo[a]pyrene particles retained in the lungs were removed by absorption into the blood

stream. The association of benzo[a]pyrene with the particles increased the relative amount of benzo[a]pyrene that was cleared by mucociliary action and subsequent ingested (ATSDR, 1987; 1989).

Dermal: The percutaneous absorption of anthracene (9.3 $\mu g/cm^2$) was estimated in rats. Radioactivity was measured in the urine, feces, and tissues over a six-day period with approximately 52.3% of the dose being absorbed. It is suggested that anthracene was dermally absorbed in a dose-dependent manner because the permeation of anthracene significantly decreased over time (ATSDR, 1989).

Percutaneous absorption through human skin is similar to that seen in the rhesus monkey and is dependent upon the vehicle employed. With an acetone vehicle, the absorption rate is approximately 50 %. However, when the matrix for BaP is soil, the absorption rate declines to approximately 13% (Wester et al., 1990).

DISTRIBUTION:

Ingestion: Following oral exposure, benzo[a]anthracene and chrysene were rapidly and widely distributed in the rat. Within 1-2 hours after administration, maximum concentrations in perfused tissues like the liver, blood, and brain were achieved. In 3-4 hours, maximum levels in lesser perfused tissues, such as adipose and mammary tissue, were reached (ATSDR, 1989).

Maximum tissue concentrations of orally absorbed dibenzo[a,h]anthracene was reached at 10 hours after administration. The highest tissue concentrations were found in the liver and kidneys, followed by adrenal glands, ovaries, blood, and fat. The removal rate from the liver and kidneys was very rapid and 3-4 days after administration, dibenzo[a,h]anthracene was distributed only in the adrenal glands, ovaries, and fat (ATSDR, 1989).

Inhalation: Results from a rat study in which benzo[a]pyrene was intratracheally administered demonstrated that the highest fractions were distributed to the lung, liver, kidney, gastrointestinal tract, and carcass. The benzo[a]pyrene concentration in the intestines increased with time, suggesting the occurrence of biliary excretion and enterohepatic recirculation. The radioactivity distribution in tissues was qualitatively similar in hamsters and guinea pigs (ATSDR, 1987; 1989).

Dermal: PAH distribution in animal tissue following dermal exposure is limited. Six days after administration, only 1.3% of an applied dose of anthracene (9.3 $\mu g/cm^2$) was detected in rat tissue (ATSDR, 1989).

METABOLISM: Metabolism of PAH occurs in all tissues. PAHs are metabolized via enzyme-mediated oxidative mechanisms and the metabolites formed for many PAHs have the capacity of leading to tumor formation (Lehr, et al. 1978). The metabolism of PAHs alters these chemicals both chemically and structurally, rendering them more water-soluble and more excretable. The metabolic process involves several possible pathways with varying degrees of enzyme activities. The activities and affinities of the enzymes in a given tissue determine which metabolic route will prevail (ATSDR, 1989).

EXCRETION:

Ingestion: One study reported that as the dose of chrysene increased in the diet of rats, the percentage of excreted hydrocarbon also increased. Approximately 79% of the chrysene dose was eliminated by the rat in the feces (ATSDR, 1989).

Isotopically labeled benzo[a]pyrene was administered to rats by gavage (0.04 µmol, 0.4 µmol, 4.0 µmol). Total excretion of radioactivity in the feces averaged 74-79% and 85% at 48 and 168 hours, respectively, following administration. The amount of parent compound excreted decreased as the dose increased (ATSDR, 1987; 1989).

Inhalation: A single intratracheal instillation of benzo[a]pyrene (2.5 mg/kg) cleared rapidly from the lungs of a mouse with a half life of approximately eight hours. Approximately 85% was cleared from the lungs 24 hours after instillation (ATSDR, 1987; 1989).

Dermal: A study in which rats receiving anthracene applied to the skin reported that 6 days after administration, the fraction of the dose in the urine and feces was 29.1 and 21.9%, respectively (ATSDR, 1989).

GENOTOXICITY: Oral exposure to a total dose of 10 mg/kg benzo[a]pyrene produced gene mutations in mice in the mouse coat color spot test (ATSDR, 1987; 1989). A single topical application of 100 mg benzo[k]fluoranthene or indeno[1,2,3-c,d]pyrene and 30 mg benzo[a]pyrene and benzo[b]fluoranthene were reported to bind to DNA in CD-1 mouse skin following dermal exposure. Covalent binding of chemicals to DNA can result in strand breaks and DNA damage, ultimately leading to mutations (ATSDR, 1989).

IMMUNOTOXICITY: Benzo[a]pyrene is immunogenic when applied dermally to the skin of animals. Acute application of 120 µg benzo[a]pyrene in mice elicited an allergic contact hypersensitivity in C3H mice which was antigen specific (ATSDR, 1987; 1989).

Immunocompetence is an important factor in lowering susceptibility of toxicity and disease due to exposure to environmental contaminants, including PAH. The immune system may be compromised due to age, genetic factors, or disease. Some PAHs depress the immune system and can therefore be considered as possible cofactors in the development of lymphoreticular cancers. Immunological effects, such as allergic contact, and hypersensitivity, which was antigen specific, was produced in mice exposed to PAHs (ATSDR, 1989).

REPRODUCTIVE TOXICITY: Data from three animal studies which evaluated developmental effects of benzo[a]pyrene in inbred strains of rats and mice indicate that prenatal exposure to benzo[a]pyrene produced reduced mean pup weight during postnatal developmental and a high incidence of sterility in the F1 progeny of mice. In rats, effects were reported following benzo[a]pyrene treatment during gestation (ATSDR, 1987; 1989).

When benzo[a]pyrene was administered daily during gestation to pregnant rats. Decreased maternal weight gain and hematological changes were reported along with reduced fetal weights and decreased fetal survival. Bladder dilation and hydronephrosis were reported in fetuses at all dose levels. Treatment during the early-and mid-gestation period also produced post-implantation losses and decreased fetal weights. However, no maternal toxicity was reported when benzo[a]pyrene was administered during early- and mid-gestation. These results suggest that benzo[a]pyrene produced adverse developmental effects in rats at doses that are not maternally toxic (ATSDR, 1987; 1989).

ENVIRONMENTAL FATE: Physical and chemical properties of PAHs determine to a great extent how these chemicals will be transported in the environment. In general, PAHs have low water solubilities and some of their transporting characteristics are roughly correlated to their molecular weights.

Particulate PAHs released into the atmosphere may be transported over great distances and removed by wet and dry deposition. PAHs can bind to particulates, biodegrade, oxidize, photodegrade, volatilize, and accumulate in aquatic organisms while present in surface water. PAHs can also biodegrade or accumulate in aquatic organisms and plants while present in soils and sediments (ATSDR, 1989).

PAHs are present in the atmosphere in the gaseous phase or sorbed to particulates. It has been estimated that a total of 23% of benzo[a]pyrene released to the atmosphere is deposited on soil and water surfaces. Dry deposition of benzo[a]pyrene adsorbed onto atmospheric aerosols accounts for most of the removal while wet deposition is less significant by a factor of 3 to 5 (ATSDR, 1987; 1989).

PAH compounds tend to be removed from the water column by volatilization to the atmosphere, binding to particulates or sediments, or by being accumulated by or sorbed onto aquatic biota. Sorption of PAHs to sediments and soils increases with increasing organic carbon content and is directly dependent on particle size (ATSDR, 1989). Either as a result of migration directly from surface waters or through the soil, PAHs have been detected in groundwater. PAHs have also been shown to be transported laterally within aquifers (ATSDR, 1989).

In air, PAHs can undergo photooxidation and can react in the atmosphere with ozone, sulfur dioxide, nitrogen oxides, and peroxyacetylnitrate. In water, the most important processes contributing to PAH degradation are photooxidation, chemical oxidation, and biodegradation by aquatic microorganisms. In soil, microbial metabolism is the major process for PAH degradation (ATSDR, 1989).

ENVIRONMENTAL TOXICITY: Due to the lipophilic nature of PAHs, bioconcentration occurs in plants and animals. These burdens can then be passed up the food chain, resulting in higher doses at higher tropic levels. Uptake of ingested or inhaled PAHs is rapid and extensive. Clearance of these compounds from the body appears to be dependent upon metabolism. Metabolism appears to be mediated by the aryl hydrocarbon hydroxylase (AAH) fraction of the mixed-function oxidase (MFO) system. Fish and animals with AHH have the ability to metabolize PAHs and show little tendency to bioaccumulate those compounds, while creatures lacking AHH (such as some molluscs) tend to bioaccumulate PAHs (EPA, 1984a; Nariagu and Simmons, 1983).

Aquatic Life Toxicology: Different PAHs are commonly found as complex mixtures. Therefore, they will be dealt with as a group instead of as separate entities. However, it is not accurate to assume equivalent toxicities for

different PAHs as LC_{50} values may differ by as much as 2 orders of magnitude. The PAH data base for aquatic life toxicology was limited for information on acenaphthene, fluoranthene, and naphthalene.

In a 96-hour EC_{50} study on algae with acenapthene and *Selenastrum capricornutum*, respective values of 530 and 520 µg/l were derived for chlorophyll *a* and cell numbers. EC_{50} values for this plant and fluoranthene were 54,600 µg/l for chlorophyll *a* and 54,400 µg/l for cell numbers. In a 48-hour study with naphthalene, *Chlorella vulgaris* experienced 50% reduction in cells at a concentration of 33,000 µg/l.

Forty-eight-hour EC_{50}s for the three aforementioned PAHs were determined for *Daphnia magna* (aquatic invertebrate). The acute values for naphthalene, acenapthene, and fluoranthene were 8,570, 41,200, and 325,000 µg/l, respectively. In a 96-hour LC_{50} study, the acute values for bluegill and acenapthene and bluegill and fluoranthene were 1,700 and 4,980 µg/l, respectively. Only the fathead minnow was tested for naphthalene sensitivity. At 14°C the 96-hour LC_{50} was 4,900 µg/l while at 24°C the acute value was 8,900 µg/l.

Using flow through methods, a 96-hour LC_{50} of 2,300 µg/l for naphthalene was determined in rainbow trout. Mosquitofish recorded a remarkably high concentration of 150,000 µg/l in an LC_{50} test with naphthalene.

The only freshwater species for which chronic testing has been performed is the fathead minnow. An embryo-larval test (flow through) determined a chronic value of 620 µg/l.

A 28-day exposure realized a bioconcentration factor (BCF) of 387 for acenapthene in fish. No BCFs were calculated for the other two chemicals.

REGULATORY STATUS: The WHO has set an European Standard for PAHs in Drinking Water at a level of 0.2 µg/l.

The U.S. EPA has derived Ambient Water Quality Criteria (AWQC) for the protection of human health from potential carcinogenic effects of PAH mixtures (Table 2).

Table 2. EPA Ambient Water Quality Criteria for Risk due to PAHs

Risk Level	Ingesting Organisms and Water (mg/l)	Ingesting Organisms Only (mg/l)
10^{-5}	2.8×10^{-2}	3.11×10^{-1}
10^{-6}	2.8×10^{-3}	3.11×10^{-2}
10^{-7}	2.8×10^{-4}	3.11×10^{-3}

Worker Exposure Limits: Increase risks of cancer of the lung, kidney and skin have been documented among workers exposed to coal tar pitch volatiles containing PAHs. Sources of exposure include emissions from coke ovens, from cooking of coal tar pitch and from Soderberg aluminum reduction electrolytic cells (Proctor et al., 1988; NIOSH, 1977).

ACGIH TLV-TWA - Includes anthracene, B[a]P, phenanthrene, acridine, chrysene, and pyrene at a level of 0.2 mg/m^3.

OSHA PEL - Includes anthracene, B[a]P, phenanthrene acridine, chrysene, and pyrene at a level of 0.2 mg/m^3.

NIOSH-TWA - For benzene soluble PAHs at a level of 0.1 mg/m^3.

NIOSH-IDLH - For coal tar pitch volatiles which include anthracene, benzo[a]pyrene, phenanthrene, acridine, chrysene, and pyrene at a level of 400 mg/m^3.

WHO - European Standard for Drinking Water at a level of 0.2 µg/l. Benzo[a]pyrene, benzo[b]fluoranthene, benzo[a]fluoranthene, benzo[k]fluoranthene, fluoranthene, and indeno[1,2,3,-c,d]pyrene were used as indicators to derive this standard.

EPA OWRS - Ambient Water Quality Criteria for Protection of Human Health for Ingesting Water and Organisms at a risk level of: 10^{-5}

with a value of 28 ng/l, 10^{-6} with a value of 2.8 ng/l, 10^{-7} with a value of 0.28 ng/l; organoleptic effects at a level of 20 µg/l; fluoranthene at a level of 42 µg/l. Specific chemicals used to derive these standards were not listed in the reference consulted (ATSDR, 1989).

For non-specific media, EPA's OERR has set a Reportable Quantity at a level of 1 lb (ATSDR, 1989).

Cancer Factor: The data of Neal and Rigdon (1967) were used to derive a potency slope for benzo[a]pyrene that is applied to all potentially carcinogenic PAH. Neal and Rigdon (1967) fed mice chow containing between 1 and 250 ppm benzo[a]pyrene and found that rats treated at higher levels developed stomach tumors against the control group. The increased tumor incidence was dose-dependent. After adjusting the doses to correct for presumed differences in mouse versus human metabolism, these data were used to calculate the upper 95% confidence interval on the slope of a dose-response line fitted to an equation modeling the assumed no threshold, multistage mechanism of chemical carcinogenesis. The potency slope derived is 11.5 $(mg/kg/day)^{-1}$ for ingestion exposures to benzo[a]pyrene. The potency slope indicates that an individual consuming 1 µg benzo[a]pyrene per kg body weight, daily, for life, might have a risk of contracting cancer of about 1 chance in 100,000 over that of the non-exposed individual (note that this is an upper bound on the estimate, the actual risk is likely to be lower). The potency slope for inhalation exposure to benzo[a]pyrene is based on the data of Thyssen et al, (1981). Using a similar dose-response extrapolation method, the EPA Cancer Assessment Group (CAG) determined the inhalation slope to be 6.10 $(mg/kg/day)^{-1}$. Because the dose-response relationship is presumed to be linear, simply multiplying the predicted lifetime daily intake of carcinogenic PAHs by the potency slope will give an upper bound estimate of excess cancer risk from PAHs.
Reference Dose: The U.S. EPA has not published a Reference Dose criteria for all non-carcinogenic PAHs (EPA 1984b).

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GLOSSARY

ACGIH = American Conference of Governmental Industrial Hygienists
B[a]P = benzo[a]pyrene
NIOSH = National Institute for Occupational Safety and Health
EPA = Environmental Protection Agency
IDLH = Immediately Dangerous to Life and Health
OERR = Office of Emergency and Remedial Response
OSHA = Occupational Safety and Health Administration
OWRS = Office of Water Regulations and Standards
PEL = Permissible Exposure Limit
REL = Recommended Exposure Limit
TLV = Threshold Limit Value
TWA = Time-Weighted Average
WHO = World Health Organization

TOXICOLOGY PROFILE

RADIONUCLIDES

COMPOSITION: A nuclide is any form of an element with a specific complement of protons and neutrons in the nucleus. It is usually identified by the name of its element and its atomic weight. For example, uranium-235 has 92 protons (as do all uranium nuclides) and 143 (235-92) neutrons.

A radionuclide is any nuclide that decays spontaneously with the emission of radiation. Most radionuclides belong to elements that also have stable nuclides. However, no element with atomic number greater than 82 (lead) has a completely stable nuclide, although bismuth (atomic number 83) has a nuclide (atomic weight 209) that is virtually stable.

In nature, radionuclides occur principally as members of the decay chains of the two primordial long-lived radionuclides uranium-238 (half-life, 4.5 billion years) and thorium-232 (half-life, 14 billion years) or as nuclides that are produced by the interaction of cosmic rays with stable elements (e.g., hydrogen-3 (tritium), carbon-14, potassium-40). Man-made radionuclides include fission products from nuclear weapon fallout and nuclear reactors, neutron-induced nuclides from reactors, and a variety of nuclides produced in accelerators. The radionuclides of principal concern for fossil-fuel-fired electric power plants are the naturally occurring radionuclides in the uranium and thorium series[1] and potassium-40.

Radionuclides can emit a variety of radiations, but the ones of most environmental importance are alpha, beta, and gamma radiation.

Alpha particles are essentially bare helium nuclei, consisting of two protons and two neutrons. Although relatively energetic, they interact quickly in matter and have a range in tissue of only one or two cell diameters. Therefore, alpha radiation external to the body is of little consequence because the particles cannot penetrate the stratum corneum, the dead layer of skin. Internally deposited alpha emitters, on the other hand, can irradiate and damage nearby viable cells and, in fact, alpha particles are considerably more biologically

[1] Another series descends from U-235 and includes isotopes of the odd-atomic-number elements protactinium and actinium as well as odd-atomic-weight isotopes of thorium, radium, radon, polonium, bismuth, and lead. The natural abundances of these materials are too low to be significant in the toxicity of power plant emissions.

damaging per unit dose than are beta and gamma radiation.

Beta radiation consists of energetic electrons that have intermediate penetrating power (about a centimeter in tissue). Although beta particles can cause burns to the skin, external beta radiation at environmental levels does not easily penetrate to organ systems where it can be damaging, and is therefore a threat mainly from internally deposited beta emitters. Internal beta radiation is less biologically damaging per unit dose than alpha radiation, but is essentially identical in effects to gamma radiation.

Gamma radiation consists of energetic photons of electromagnetic radiation that easily penetrate the body with little attenuation. External gamma emitters are therefore as important and often more important than internally deposited gamma emitters.

When radionuclides decay, usually either an alpha or beta particle is emitted first, forming a nucleus with a different atomic number. If an alpha particle is emitted, the resulting nucleus has two less protons and two less neutrons, and therefore has atomic number two less and atomic weight four less than the parent. If a beta particle is emitted, the resulting nucleus has one more proton and one less neutron, and therefore has an atomic number one greater than the parent but a virtually identical atomic weight. The resulting nuclide is often formed in an excited state and then decays to its ground level state by emission of gamma radiation. The resulting nuclides are often themselves radioactive and are called "daughters" or "progeny" of the parent radionuclide.

Many radionuclides can decay by two or more processes with different probabilities. Some progeny of radioactive decay are so short-lived or so infrequently formed that they are often not counted separately and their radiations are lumped with those of the longer-lived parent radionuclide. The propensity of a nuclide to decay is measured by its halflife, with shorter halflives associated with higher decay activity per unit time. The quantity of a radionuclide present in a medium is usually measured in becquerels (disintegrations per second) or in the older unit of curies (3.7×10^{10} disintegrations per second). Environmental levels are often expressed in picocuries (pCi), i.e., 10^{-12} Ci. The specific activity of a pure radionuclide is measured in Ci/g and is higher the shorter the halflife. The principal members of the radioactive decay chains of uranium-238 and thorium-232 are shown in Figures 1 and 2, respectively.

In this profile, only the radionuclides shown in Table 1 are included.

Synonyms: Varies depending on specific radionuclide. For example, uranium-238 (CAS No. 7440-61-1) is also known as:

U-238
^{238}U
Uranium I

Radon-220 is also known as:

Rn-220
^{220}Rn
Thoron

CAS No.: Differs according to specific radionuclide. Refer to Table 1 for the most important radionuclides.

Table 1. Selected Radionuclides

Radionuclide Name	Abbreviation	CAS Number	Half-life	Primary Radiation
Uranium Series				
uranium-238	U-238	7440-61-1	4.5 billion years	alpha
thorium-234	Th-234	7440-29-1	24 days	beta
uranium-234	U-234	7440-61-1	250 thousand years	alpha
thorium-230	Th-230	7440-29-1	80 thousand years	alpha
radium-226	Ra-226	7440-14-4	1600 years	alpha
radon-222	Rn-222	14859-67-7	3.8 days	alpha

radon daughters	Po-218, Pb-214, Bi-214, Po-214		all less than 27 minutes	alpha beta
lead-210	Pb-210	7439-92-1	21 years	beta
bismuth-210	Bi-210	7440-69-9	5 days	beta
polonium-210	Po-210		138 days	alpha
Thorium Series				
thorium-232	Th-232	7440-29-1	14 billion years	alpha
radium-228	Ra-228	7440-14-4	5.8 years	beta
thorium-228	Th-228	7440-29-1	1.9 years	alpha
radium-224	Ra-224	7440-14-4	3.6 days	alpha
radon-220	Rn-220	22481-48-7	55 seconds	alpha
thoron daughters	Po-216, Pb-212, Bi-212, Po-212, Ti-208		All less than 11 hours	alpha beta
Potassium				
potassium-40	K-40	7440-09-7	1.25 billion years	beta gamma

Boiling point: Uranium: 3818°C
Thorium: 4500°C (approx.)
Radium: 1140°C (approx.)
Radon: -61.8°C
Polonium: 962°C
Potassium: 766°C

Color Uranium: Silvery metal
Thorium: Silvery metal
Radium: Silver-white
Radon: Colorless
Potassium: Silvery white metal

Conversion factor: Depends on nuclide. For natural uranium (mixture of U-238, U-235, and U-234): 1 µg = 0.67 pCi
Thorium-232: 1 µg = 0.11 pCi
Thorium-230: 1 µg = 21 µCi
Thorium-228: 1 µg = 833 µCi
Radium-224: 1 µg = 162 mCi
Radium-226: 1 µg = 0.99 µCi
Radium-228: 1 µg = 275 µCi
Radon-222: 1 µg = 154 mCi
Radon-220: 1 µg = 932 Ci
Potassium-40: 1 µg = 7.1 pCi

Flammable limits: Uranium: Dust cloud can ignite at room temperature
Thorium: Dust cloud can ignite at room temperature

Henry's law constant: NA

Melting point: Uranium: 1132.3°C
Thorium: 1750°C
Radium: 700°C (approx.)
Radon: -71°C
Polonium: 254°C
Potassium: 63.2°C

Molecular formula: U for uranium
Th for thorium
Ra for radium
Rn for radon
Po for polonium
Bi for bismuth
Pb for lead
K for potassium

Molecular (atomic) weight: Uranium: 238.03
Thorium: 232.04
Radium: 226.03
Radon: 222
Polonium: 210
Bismuth: 209
Lead: 207.21
Potassium: 40

Odor threshold: Unknown

Solubility
Water: Radon: 510 cm^3/l at 0°C
224 cm^3/l at 25°C
130 cm^3/l at 50°C
Others: depends on chemical species

log K_{ow}: NA

log K_{oc}: NA

Specific gravity: Uranium: 19.05 g/ml
Thorium: 11.70 g/ml
Radium: 5.50 g/ml
Radon: 0.01 g/ml
Potassium: 0.86 g/ml

Vapor pressure: Uranium: 1 mm Hg at 2450°C
Thorium: NR
Radium: NR
Radon: 395.2 mm Hg at -71°C
Potassium: 8 mm Hg at 432°C

NA = Not applicable
NR = Not reported

(References: ATSDR 1990a,b,c,d; HSDB 1993a,b,c,d; NAS 1988)

USES: Uranium: The principal use of uranium is as a fissionable material in nuclear weapons and reactors. U-235 is more fissionable than the other isotopes. Depleted uranium (mostly U-238) is used in military projectiles, gyroscopic compasses, radiation shielding, and other applications because of its mass density. Uranium dioxide is used in the filaments of large incandescent lamps. A variety of other uranium compounds are used in photography; staining or dyeing of wood, leather, and some fabrics; ceramic glazes; and chemical catalysts.
Thorium: Although in principle thorium could be used as a fissionable material for nuclear energy, it is actually in very limited use. It is used in refractory applications, in mantles for kerosene lamps and in other lighting applications, in aerospace alloys and ceramics, in computer memories, in welding electrodes, and in nuclear weapon production. Formerly, it was used in Thorotrast, a contrast medium for medical radiography.

Radium: Almost all of radium's current and former uses have been based on its radioactivity. It is currently used as a source of radiation for cancer therapy and materials radiography and in various other measurement and research purposes. Formerly, it was extensively used in a variety of luminous devices (watch faces, instruments, etc.) and as a pharmaceutical. All three major isotopes (224, 226, and 228) were used in medicine.

Radon: Radon has been used for a variety of medical purposes and is still used in cancer therapy. Other uses are primarily in research.

Polonium: The principal uses for polonium are a result of its radioactivity: static elimination and production of neutrons by alpha-particle interaction with beryllium.

Bismuth and lead: Stable lead and quasi-stable bismuth-209 have a multitude of uses, but the radioactive isotopes have little use other than in research.

Potassium: Although there are many used for natural potassium, none for K-40 outside of research is known.

(References: ATSDR 1990a,b,c,d; HSDB 1993a,b,c,d; NAS 1988; Merck 1989.)

ABSORPTION, METABOLISM, AND EXCRETION: The absorption, distribution, and excretion of the radionuclides depends on their chemical, not radiological properties. It must be borne in mind, however, that radioactive decay from one nuclide to another occurs, with a corresponding change of chemical identity. For decay chains whose members have short radioactive halflives, these transformations can affect the patterns of absorption, distribution, and excretion. The following discussions focus principally on the behavior of the parent element, not on progeny created in the body.

Uranium: The absorption, distribution, metabolism, and excretion of uranium depends markedly on the chemical species in which the uranium exists in the exposure environment and on the physical size of the particles to which these species are attached. In power plant emissions, the principal forms will probably be the oxides UO_2, U_3O_8, and UO_3. Only the tetra- and hexavalent states are biologically important, and biological processing of uranium is especially dependent on the uranyl ion, UO_2^{2+} (NAS 1988).

After inhalation, particles containing the oxides typically remain in the lung or the associated lymph glands from weeks to years, with some clearance by the mucociliary pathway and some dissolution into body fluids. Radiation can occur either in the lungs or after absorption. Absorption from the lungs is probably in the range 0.2-5%, although in laboratory animals absorption of UO_3 as high as 23% has been reported (ATSDR 1990a).

Following ingestion, absorption is also low for most uranium species. Estimates of the fraction absorbed range from 0.2% for very insoluble compounds to as high as 20%. Most estimates are in the range 0.5-5%, with absorption rates declining somewhat with increasing dose.

Absorption from the gastrointestinal tract may also depend on whether food is present (ATSDR 1990a).

Although some uranium compounds can be absorbed through the skin, the relatively insoluble environmental species are probably absorbed little if at all (ATSDR 1990a).

After absorption, uranium is distributed principally to kidney and bones, with some in lymph nodes and fat. Uranium found in lungs is probably due to the immobility of inhaled uranium on particles. Most forms of uranium eventually react to form the uranyl ion, which binds to components of blood plasma and is eventually cleared in the urine (NAS 1988, ATSDR 1990a).

Thorium: As with uranium, the absorption, distribution, metabolism, and excretion of thorium depends markedly on the chemical species in which the thorium exists in the exposure environment and on the physical size of the particles to which these species are attached. In power plant emissions, the principal form will probably be the dioxide, ThO_2.

After inhalation, particles containing thorium dioxide typically remain in the lung or the associated lymph glands for long times (15-30% remaining after 21 months), with some clearance by the mucociliary pathway and some dissolution into body fluids. Radiation can occur either in the lungs or after absorption. Absorption of thorium from the lungs is probably under 5% (ATSDR 1990b).

Following ingestion, absorption is very low for most thorium species. Estimates of the fraction absorbed range from 0.02% to 1%. Based on data from rats, absorption of thorium from the gastrointestinal tract may be somewhat greater for infants than for adults (ATSDR 1990b).

Although some thorium compounds probably can be absorbed through the skin (ATSDR 1990a), thorium dioxide is probably absorbed little if at all.

After absorption, thorium is distributed principally to bone. Transferrin plays a major role in the distribution of thorium absorbed into the body. Thorium found in lungs and lymph nodes is probably due to the immobility of inhaled thorium on particles. Although thorium was found in the liver, spleen, and bone marrow of patients injected with Thorotrast, the colloidal nature of that material probably explains the different distribution from environmental thorium (NAS 1988, ATSDR 1990b).

Most ingested and inhaled thorium is excreted in the feces, having never been absorbed into the body. Although excretion of inhaled thorium via mucociliary clearance and the feces is relatively rapid (several weeks), absorbed thorium is cleared only slowly to the urine, with a biological halflife of at least 14 years (ATSDR 1990b). Whether chemical transformations are involved in clearance is not known.

Radium: As with other inorganic elements, the absorption, distribution, and excretion of radium depends on the chemical species of exposure. Radium in power plant emissions probably occurs as the element and the sulfate. In water, radium occurs principally as a divalent ion (ATSDR 1990c).

After an accidental inhalation exposure, radium sulfate was cleared from the lung with a halflife of about 120 days (Marinelli et al. 1953). How much was cleared without absorption was not reported, but substantial absorption either directly from lung or indirectly after mucociliary clearance and swallowing is assumed (ATSDR 1990b).

Following ingestion of radium sulfate, absorption was estimated to be of the order of 20% (Maletskos et al. 1969). The remainder of the radium was excreted via the feces. Absorption could be higher for more soluble species.

No studies were located on the absorption of radium through the skin.

Following absorption, radium is rapidly distributed throughout the body, with the majority being found in bone (radium is a calcium analogue). A relatively small proportion of the body burden (2-4%) is rapidly cleared via the urine in a few days, but the skeletal radium remains for a much longer time, with different time frames for surface and volume-deposited radium. Perhaps 1% of the absorbed radium remains after 30 years. Excretion is principally via the biliary tract and feces, but perhaps 2% of the excretion is via urine (ATSDR 1990c, NAS 1988).

Radon: Radon, as an inert gas, is distributed in the body on the basis of its solubility characteristics and readily diffuses across the linings of both the lungs and gastrointestinal tract (and presumably through the skin). After prolonged radon exposure, concentrations in body organs can reach 30-40% of those in inhaled air. In the GI tract, most of the radon is absorbed in

the stomach and small intestine before reaching the large intestine, although the contents of the stomach can influence the rate of absorption. With a halflife of approximately four days, most of the radon inhaled or ingested will escape the body, principally via the lungs, before decaying. As the biological halflife is measured in minutes to hours, less than 1% of absorbed radon will decay while in the body. Radon is more soluble in fat than in water and preferentially accumulates in fatty tissues such as the liver (ATSDR 1990d).

More important for the toxicology of "radon" are the properties of the radon progeny; for Rn-222 they are Po-214 and -218, Bi-214, and Pb-214. These substances are formed as radon decays in air and reach partial equilibrium with the radon levels. (Equilibrium occurs when the activities in pCi/l of radon and all its progeny are the same.) They typically attach to particulate matter in the air and are carried into the lung as an aerosol. Deposition of the particulates in the lung for even a short time provides a relatively high probability of decay in place, because the halflives of all these radionuclides are less than half an hour. They are thus not very likely to move directly into the body or up the mucociliary escalator and be swallowed. In any case, the absorption of radon progeny from the gastrointestinal tract does not appear to be very great, also in part because of the short halflives. The longer-lived decay product of the radon series, Pb-210, has been found in bone and teeth following radon exposure (ATSDR 1990d).

Polonium: Polonium is biologically important via the short-lived decay products (Po-214 and -218) of airborne radon and Po-210, the last member of the uranium-238 chain. Po-210 is produced by the beta decay of Pb-210 and Bi-210, and itself decays by alpha emission to stable Pb-206. Because Pb-210 has a halflife of 21 years, Po-210 can exist separately from a continuing source of radon. Its chemistry is unlike that of most of the other heavy radionuclide elements and resembles the rare earths (NAS 1988).

Not much is known about the absorption of polonium from lungs or gastrointestinal tract other than that it depends on the chemical species of the polonium. Once absorbed, polonium distribution depends on its tendency to form hydroxides and radiocolloids. As a colloid, it is phagocytized by cells and deposited in spleen, lymph nodes, bone marrow, and liver. Kidney is another major site of deposition. Without colloid formation, polonium is widely distributed in body tissues. Most of the polonium excreted is via the feces. Biological halflives for clearance appear to be measured in the range 4 to 40 days and depend on whether the dose is single or repeated (NAS 1988).

Bismuth: Except for quasi-stable Bi-209, all of the bismuth isotopes have short halflives and their biological properties are not very important for toxicity.

Lead: The biological properties of the radioactive lead isotopes (especially for long-lived Pb-210) are the same as for stable lead (see lead toxicity profile).

Potassium: The biological properties of the potassium-40 are the same as for stable potassium; as an essential element for life, it is easily and widely distributed in the body. Potassium in power plant emissions probably occurs as the chloride or as an aluminosilicate, although some of it could be oxidized to K_2O. Both potassium-40 and the non-radioactive isotopes of potassium in emissions from fossil-fueled electric power plants will generally follow the same distribution patterns as potassium from other sources.

TOXICITY OVERVIEW: All of the radionuclides treated in this profile produce ionizing radiation, either particles or photons with sufficient energy to ionize intracellular water and other chemical constituents of tissue. Radiation damage takes place either directly, in which ionized molecules such as DNA do not recombine exactly as they were before the ionization, or indirectly, in which ionized species (e.g., free radicals) attack DNA or other cellular constituents and cause chemical changes that can be deleterious. In extreme cases, physical damage due to the amount of energy deposited in a cell may also play a part. This radiation toxicity is virtually always much more important than the chemical toxicity of the radionuclides. The one exception is for uranium, particularly the low-specific activity U-238, which can cause kidney toxicity independent of its radioactive properties.

The principal concern regarding radiotoxicity is cancer, but high levels of radiation exposure can lead to reproductive and developmental abnormalities through both genetic toxicity and other damage. The affected organs differ from nuclide to nuclide principally through differences in the physical and chemical properties of the parent element as determined by the number of electrons surrounding the nucleus and not by the numbers of protons and neutrons in the nucleus. Differences also arise because of different halflives (which influence

the steady-state distributions of radionuclides in organs) and different radiations (more of the energy emitted by a beta-emitter will escape an organ in which it is deposited than for an alpha-emitter).

ACUTE TOXICITY: Very high acute exposures to uranium compounds can result in kidney toxicity and death within 14 days following exposure. The lethal concentration for 50% of exposed rats was reported to be 120,000 mg/m^3 for a two-minute exposure, declining to 12,000 mg/m^3 for a ten-minute exposure. Guinea pigs appear to be about twice as sensitive as rats (Leach et al. 1984).

While acute toxicity from the other radionuclides covered in this profile is possible, the risk of radiogenic cancers is much more important for human health.

CHRONIC TOXICITY: Kidney toxicity is also the primary finding for chronic exposure to uranium in both humans and laboratory animals. The renal injury in dogs, rats, guinea pigs, and rabbits is manifested by degeneration, necrosis, and regeneration of the distal portions of the proximal renal tubules (Stokinger et al. 1953). In humans, chronic exposure to uranium has increased deaths due to nephritis and renal sclerosis (Waxweiler et al. 1983) and at lower levels increased the excretion of b-2-microglobulin and 5 amino acids (Thun et al. 1985). These observations are consistent with the animal observations. Renal damage occurs after a threshold concentration of uranium in kidney is reached; estimates of the threshold range from less than 1 to 3 µg/g tissue (NAS 1988). At the higher figure, about 400-500 µg uranium per day would be excreted in urine (NAS 1988), implying that the steady-state threshold for absorbed uranium would also be about 6-7 µg/kg body weight/day. The threshold for exposure depends on the fraction of uranium absorbed from inhalation or ingestion and would be in the range 20 to 200 times higher.

Respiratory effects such as emphysema and fibrosis have been reported after human exposure to uranium (Waxweiler et al. 1983), but animal studies suggest that at least some of the effects may be due to confounding factors. Reported changes in hemoglobin and erythrocyte count in uranium miners (Vich and Kriklava 1970) may be a radiation rather than chemical effect. Neither of these effects nor any others that have been reported are likely to be as sensitive as end points for uranium exposure as kidney toxicity.

As with acute toxicity, the chronic non-cancer effects of the other radionuclides are not important in comparison with their potential for radiogenic cancer.

SENSITIZATION: No studies were found that suggest that any of the radionuclides are sensitizers.

TARGET ORGAN EFFECTS: Because of their chemical, physical, and radiological properties, the different radionuclides have differing potential to affect various organ systems. Except for uranium (as described above), the principal toxicity for all the radionuclides is carcinogenesis, covered in greater depth below. This section simply summarizes the target organs.

Uranium: Chemical toxicity to the kidney is the principal risk of exposure to natural uranium or depleted uranium containing mostly U-238 by weight. All of the uranium isotopes can in principle also cause radiogenic cancers, most likely in the lung after inhalation exposures or in bone after absorption from the lung or gastrointestinal tract. With natural or depleted uranium, these cancers have not been observed in humans or experimental animals (ATSDR 1990a). High-specific-activity U-232 and U-235, however, are able to induce bone cancers in laboratory animals (NAS 1988). Based on these data and other observations, Mays et al. (1985) estimated the risk of bone sarcoma to be 1.5 per million persons exposed to a constant 1 pCi/day of uranium in water or food.

Thorium: The most reported malignancies from thorium exposure are the liver tumors in the patients injected with Thorotrast. The Thorotrast patients also exhibited a variety of blood disorders, including anemias and leukemia (ATSDR 1990b). For exposures other than Thorotrast, little epidemiologic or experimental evidence suggests that natural thorium causes tumors. It is reasonable to suppose that radiation from any of the thorium isotopes is possible and could occur in lung, bone, liver, and hematopoietic tissues. Bone cancers have been induced by high-specific-activity Th-228 in dogs (Mays et al. 1969, Lloyd et al. 1986).

Radium: The most prominent effects of radium exposure are cancers of bone, paranasal sinuses, and mastoid air cells in the radium dial painters (Rowland et al. 1978) and patients treated with radium (Mays and Spiess 1984). Ra-224, -226, and -228 are all effective in producing tumors. Other effects of radium exposure are seen principally in humans or

animals injected with radium compounds and may not be relevant for environmental exposures. They include hematological abnormalities, liver cirrhosis, cataracts, and tooth breakage (ATSDR 1990c).

Radon: Exposure to radon progeny in uranium mines unambiguously produces excess lung cancer in humans (NAS 1988). Most of the toxicity arises from the progeny and not the parent radon itself. It is generally assumed that some of the ingested radon penetrating the walls of the gastrointestinal tract will decay before it is exhaled and that radiation from that decay and from subsequent decay of the progeny will also produce tumors. Other non-malignant respiratory diseases have also been associated with inhalation exposure to radon and its progeny in both humans and animals (ATSDR 1990d). Mild hematological effects, kidney damage, and skin cancer may also be associated with exposures to radon and progeny, but the evidence is not strong (ATSDR 1990c).

Polonium: In experimental animals, exposure to polonium isotopes has been associated with lymphomas, bone tumors, and various soft tissue tumors. Sclerotic changes in blood vessels, atrophy of the testes, atrophy of the thymus, spleen, and bone marrow, and effects on the kidney, adrenals, pancreas, lung, and circulatory system have also been reported at high doses. Most are assumed to be direct or indirect effects of radiation (NAS 1988).

Bismuth: The health effects of the short-lived bismuth isotopes are included with those of the longer-lived parent radionuclides. All except Bi-211 are also beta-emitters and therefore of somewhat lower radiotoxicity per unit activity.

Lead: All of the radioactive isotopes of lead are beta-emitters and therefore have less potential to cause health effects for a given level of activity than do the alpha-emitters treated above. They presumably have some carcinogenic potency in lead-concentrating organs, particularly bone.

Potassium: The biological effects specific to potassium have not been well studied. It is widely assumed that both internal beta radiation and internal and external gamma radiation from potassium can be carcinogenic to most if not all radiosensitive organs. Given the ubiquity of potassium in the body, no particular organ system stands out as most at risk.

REPRODUCTIVE TOXICITY: At high levels of exposure, all ionizing radiations have the potential to harm the reproductive system. Because short-range alpha particles deposit relatively large amounts of energy in small volumes, they could be reproductive hazards if deposited in the reproductive organs. Most of the radionuclides described in this profile have little affinity for the reproductive system. Available information on reproductive effects is summarized below by radioactive element.

Uranium: Little information is available to suggest that ordinary exposures to natural uranium affect reproduction (ATSDR 1990a). In one study (Paternain et al. 1989), mice gavaged with uranyl acetate dihydrate showed an increase in fetal resorptions after conception. In another, rats treated with soluble uranium compounds exhibited testicular lesions (Malenchenko et al. 1978).

Thorium: Natural thorium does not appear to pose much risk to the reproductive system. Only one study reported positive findings, edema of the seminiferous tubules and (at higher doses) some changes in sperm in male rats following application of thorium nitrate to abdominal and scrotal skin (Tandon et al. 1975). The effect might have been due to chemical toxicity rather than radiation.

Radium: No studies reporting reproductive effects of radium isotopes were located.

Radon: A change in the ratio of male to female children of male uranium miners has been suggested as possibly related to radon exposure, but it may depend on age of exposure and on concomitant exposures to other substances (ATSDR 1990d).

Polonium: At high doses, Po-210 was reported to affect the testes of rats (Casarett 1964). Russian workers who had incorporated 1-5 μCi of Po-210 also showed impairments in reproductive organs (Kauranen and Miettinen 1967).

Potassium: No studies reporting reproductive effects of potassium-40 were located.

DEVELOPMENTAL TOXICITY: Ionizing radiation also has the potential to affect development at relatively high doses to the fetus. In the offspring of women who were exposed to radiation from the atomic bombs in Japan, a marked increase in microcephaly (characterized by small head size and mental retardation) and other developmental abnormalities was observed (NAS 1990). In those cases, the primary radiation was external gamma radiation directly from the bombs. Some

effects have also been observed from exposure to internally deposited radionuclides (NAS 1988), and the pertinent information for each radioactive element is described below.

Uranium: Only one study (Domingo et al. 1989) was located that addresses the developmental toxicity of uranium. When mice were treated by gavage with 3 mg/kg-day or more of uranyl acetate dihydrate, reduced fetal weight and length, skeletal malformations, and external hematomas were observed in some of the pups. There was evidence of maternal toxicity (reduced weight gain and increased relative liver weight) at all dose levels.

Thorium: No studies reporting developmental effects of thorium isotopes were located.

Radium: The only report of developmental toxicity related to radium concerns children who were injected with radium-224 as a therapy for tuberculosis. The adult heights of these persons were significantly lower than for untreated controls (Spiess et al. 1985), and the effect appeared to be dose-related.

Radon: No studies reporting developmental effects of radon isotopes were located.

Polonium: No studies reporting developmental effects of polonium isotopes were located.

Potassium: No studies reporting developmental effects of potassium-40 were located.

IMMUNOTOXICITY: High doses of radiation compromise the immune system, as is seen in patients undergoing radiotherapy for cancer. Internally deposited radionuclides may also have the potential to damage the immune system, but the evidence is less clear-cut.

Uranium: Although some changes in lymph nodes have been reported after exposure to natural uranium, these changes have not been associated with changes in immune competence (ATSDR 1990a).

Thorium: No immune system effects of thorium have been reported other than for Thorotrast injections. Fibrosis of both the lymph nodes and the spleen was reported in human patients injected with Thorotrast (da Silva Horta 1967) and suppression of the immune response was reported for mice injected with Thorotrast, especially when administered intraperitoneally (Michael and Murray 1970).

Radium: Intraperitoneal injection of 22,000 μCi/kg of Ra-226 into mice decreased the number of peripheral white blood cells (Schoeters and Vanderborght 1983) and one case study reported similar loss of leukocytes in a chemist exposed occupationally to radium (ATSDR 1990c). Exposure of the bone marrow by deposition of radium in bone is a plausible explanation.

Radon: No studies reporting immunological effects of radon isotopes were located.

Polonium: A Russian study reported changes in the immunological system of laboratory animals exposed to Po-210 (Moroz and Parfenov 1972), but few details are available.

Potassium: No studies reporting immunological effects of potassium-40 were located.

GENOTOXICITY: Because ionizing radiation is known to affect genetic material, some genetic toxicity by internally deposited radionuclides is expected (NAS 1988). Even for highly exposed populations such as the Japanese survivors of the atomic bombs, genetic changes have been difficult to verify, let alone to quantify (NAS 1990). Humans are almost surely less susceptible to genetic damage from radiation than are mice, in which most of the mammalian genetic studies have been conducted.

Uranium: Chromosome aberrations have been reported in cultured lymphocytes of uranium miners (Brandom et al. 1978). No other reports of genotoxicity related to uranium were located.

Thorium: Chromosome aberrations have been reported in cultured lymphocytes of thorium processing workers (Hoegerman and Cummins 1983), monazite sand millers (Costa-Ribeiro 1975), and Thorotrast patients (Sasaki et al. 1987). No reports of other genotoxicity related to uranium were located.

Radium: No studies reporting genetic effects of radium isotopes were located.

Radon: The chromosome aberrations reported in uranium miners above could be related to radon exposure. Chromosome aberrations were also reported in spa-house personnel and residents exposed to environmental radon in Badgastein, Austria (Pohl-Rüling and Fischer 1979). Stimulation of DNA repair was

reported in persons occupationally exposed to 3000 pCi/l radon (Tuschl et al. 1980). Sister chromatid exchange in bone marrow cells of rats was reported at cumulative radon doses as low as 100 Working-Level-Months (Poncy et al. 1980). Radon in water may also increase chromosome aberrations (Stenstrand et al. 1979).

Polonium: No studies reporting genetic effects of polonium isotopes were located.

Potassium: No studies reporting genetic effects of potassium-40 were located.

CARCINOGENICITY: Carcinogenicity is far and away the most important concern for exposure to most of the radionuclides (with the exception of the kidney toxicity of natural uranium). Although direct evidence of radiation carcinogenesis is lacking for some of the radionuclides, it is generally assumed that all of them have the potential to cause human cancer given sufficient exposure. For policy purposes, it is further assumed that any exposure will increase the risk of cancer, usually in proportion to the level of exposure. Specific evidence for radiogenic cancers from the radionuclides is discussed below; it is also assumed that the other radionuclides of the uranium and thorium series and potassium-40 can induce cancer.

Ingestion:

Uranium: No unambiguous evidence exists for the induction of cancer by ingestion of natural or depleted uranium in humans or laboratory animals in the absence of uranium series progeny (ATSDR 1990a, NAS 1988).

Thorium: No evidence that thorium causes cancer via ingestion was found.

Radium: The radium dial painters who ingested luminous paints containing Ra-226 and -228 as the sulfate exhibited excess bone sarcomas and tumors of the mastoid air cells and paranasal sinuses (Rundo et al. 1986). The lowest total radium dose associated with a bone tumor has been estimated to be 1.03 μCi/kg (Rundo et al. 1986). The evidence for leukemia from ingested radium is much weaker and is confounded by the likelihood of external gamma radiation from the radium decay products (NAS 1988). One study reported an association of excess leukemia with radium in groundwater (Lyman et al. 1985), but the results do not appear to be consistent with other observations (NAS 1988). Limited evidence from experiments with rats (Evans et al. 1944) supports the association of radium ingestion with bone sarcomas.

Radon: Hess et al. (1983) found a positive correlation between levels of Ra-222 in drinking water in Maine and rates of lung cancer and all cancers combined. However, Hess et al. noted possibly confounding factors, and the correlation, even if causal, could be due to radon evaporating into the air for subsequent inhalation. No reports of cancer in laboratory animals from ingested radon were located.

Polonium: No studies are available documenting the carcinogenicity of polonium isotopes in humans separate from radionuclides further up in the decay chain of radium. Casarett (1964) describes various soft tissue tumors in rats administered single or multiple oral doses of Po-210.

Inhalation:

Uranium: No unambiguous evidence exists for the induction of cancer by inhalation of natural or depleted uranium in humans or laboratory animals in the absence of uranium series progeny (ATSDR 1990a, NAS 1988).

Thorium: Thorium workers employed for more than one year exhibited excess pancreatic cancer (Stehney et al. 1980) but some of the excess may be due to smoking (ATSDR 1990a). In a second study (Polednak et al. 1983), pancreatic cancer and lung cancer were elevated in comparison with controls, but the excesses were not statistically significant and smoking or thoron could have been involved (ATSDR 1990b). Rats exposed to thorium dioxide at levels yielding radiation doses of 3000 rem or more exhibited lung cancers: reticulosarcomas at the lower doses and carcinomas at the higher doses (Likhachev et al. 1973).

Radium: No studies reporting carcinogenicity from inhalation exposure to radium isotopes were located.

Radon: Many epidemiological studies of uranium miners and other miners exposed to Rn-222 occupationally have demonstrated that inhalation of the short-lived radon progeny Po-218, Pb-214, Bi-214, and Po-214 can cause lung cancers. The primary studies were reviewed and analyzed by a committee of the National Academy of Sciences (NAS 1988). Studies of non-miner populations exposed to radon and progeny have shown mixed results. Some reported as positive (e.g., Svensson et al. 1989) can be criticized for limited information on exposure, while those reported as negative are often insufficiently sensitive to reject the radon hypothesis. In general, the non-mining studies can be described as not inconsistent with the mining studies. There is limited evidence that the risk of cancer is higher in smokers than in

non-smokers (NAS 1988) and it is assumed for policy purposes that radon increases cancer risk in smokers by the same factor as in nonsmokers. Risk appears to depend on age of exposure and time since cessation of exposure (NAS 1988). Lifetime exposure to 1 pCi/l of radon and progeny in typical equilibrium fractions is estimated by the Environmental Protection Agency to increase lung cancer risk by about 7 in 1,000 for smokers and 4 in 10,000 for nonsmokers (EPA 1993). Experimental exposure of laboratory animals to radon and progeny generally confirms the human epidemiology (ATSDR 1990d). Little evidence is available on the carcinogenicity of Ra-220 (thoron).

Polonium: No studies reporting carcinogenicity from inhalation exposure to polonium isotopes were located.

Skin Contact:

Uranium: No evidence was found that uranium causes cancer via dermal exposure.

Thorium: No evidence was found that thorium causes cancer via dermal exposure.

Radium: No evidence was found that radium causes cancer via dermal exposure.

Radon: A statistically significant increase in basal cell skin cancers was reported for uranium miners exposed to about 3 pCi/l of radon in air (Sevcova et al. 1978). As this observation could be confounded by other exposures and is not reported in other radon cohorts, it is not considered proof of radon carcinogenicity via dermal exposure.

Polonium: No evidence was found that radium causes cancer via dermal exposure.

Other Routes of Exposure:

Uranium: U-232, U-233, U-234, and U-235 have all caused bone, lung, or kidney cancers in laboratory animals following intravenous or intratracheal administration (ATSDR 1990a).

Thorium: Patients injected with Thorotrast exhibit primarily excess cancers of the liver and leukemias (erythroleukemia and acute myelogenous leukemia) (citations summarized in ATSDR 1990b and NAS 1988). Other cancers reported include bone (Harrist et al. 1979), lung (Faber 1986), kidneys (Christensen et al. 1983), spleen (Levy et al. 1986), and pancreas (Mori et al. 1979). The cancers appear to arise from the radiation dose (typically 1000 rad to the liver) and not from chemical toxicity, but the colloidal nature of the Thorotrast probably influences the carcinogenicity of thorium, depositing it in organs where it might not otherwise concentrate (ATSDR 1990b).

Radium: Ample evidence exists that injection of Ra-224 (Spiess et al. 1989) or Ra-226 (Gustafson and Stehney 1985) causes bone sarcomas and head carcinomas. Other reported cancers associated with radium injection include breast and liver (ATSDR 1990c).

Radon: No studies reporting carcinogenicity from other routes of exposure to radon and progeny were located.

Polonium: Injection of Po-210 into mice induced lymphomas and bone cancers (Finkel and Hirsch 1954). The effects have been attributed to deposition in bone marrow (NAS 1988).

EPIDEMIOLOGY: Ample epidemiologic evidence exists for the induction of bone and head cancers from ingestion of radium (Ra-226 and -228) and injection of Ra-224 and -226, the induction of lung cancers by inhalation of radon (Ra-222) and progeny, and the induction of liver cancers, leukemias, and other tumors by injection of Thorotrast (colloidal thorium dioxide). The epidemiology for other cancers and other radionuclides is considerably weaker. Chromosome aberrations have been reported in a number of human populations exposed to various radionuclides. Only limited epidemiologic evidence for any developmental toxicity or reproductive toxicity of the uranium and thorium series radionuclides has been offered. People who were injected with radium as children were found to be shorter than their unexposed counterparts in adulthood. The effects of uranium exposure on the kidney are also unambiguous in human studies of workers exposed to uranium. The principal epidemiological studies to reach these conclusions are cited in the appropriate sections above.

ENVIRONMENTAL FATE: As elements, the radionuclides disappear from the environment only through radioactive decay with the half-lives shown in Table 1. All of the radionuclides treated in this profile are naturally occurring and are found in soil, surface and groundwater, and air as a result of natural dispersion. Distribution in the environment after disturbance by humans depends on the chemical form in which the element occurs. All of the radionuclides except the radon isotopes are solids at ordinary temperatures and will be discharged from fossil-fueled power plant stacks principally as particulate matter. These radionuclides are transported through the environment in that form and are subject to dry deposition by settling and impingement and wet

deposition by precipitation scavenging. Transport in aqueous environments may be either in dissolved form or via suspended sediment. The importance of dissolved form transport depends on the solubility of the chemical species and its affinity for soils or organic material. Most of the species emitted from power plants will be relatively insoluble.

Uranium: The fate of most uranium released to the atmosphere is probably in sediments following wash-off of deposited uranium-containing particulate matter. Leaching from soils to groundwater can occur but is inhibited by sorption to soils, particularly clays. Some anionic complexes may be relatively mobile in soil. Uranium is not expected to accumulate in biota because uptake in higher trophic levels is relatively low. Uranium in plant materials is more likely due to adherence after deposition than to uptake and distribution in the plant. Dissolved uranium will be present as a carbonate complex in alkaline waters but as an organic complex in acidic waters. Reference: ATSDR 1990a.

Thorium: The fate and transport of thorium in the environment (mostly as the dioxide) is similar to that for uranium with the following differences. Thorium compounds are in general even less soluble than uranium compounds and transport in dissolved form is minor. In alkaline water, thorium complexes with carbonate may be somewhat more soluble. Thorium bioconcentrates to some degree in aquatic species, but accumulation decreases at higher trophic levels. Reference: ATSDR 1990b.

Radium: Radium is generally more labile than uranium and thorium. In combustion of coal, the radium may volatilize and then recondense onto fly ash and therefore be somewhat more available than if bound into an inorganic matrix. Deposition from the atmosphere is by gravitational settling, impaction, and precipitation scavenging. Although radium can be soluble under certain high pH conditions, existing as a divalent ion, its adsorption to soils reduces its mobility via surface or ground water. Radium is readily absorbed from soils into plants and can be taken up by foraging animals. It is modestly bioconcentrated in aquatic species. Reference: ATSDR 1990c.

Radon: Before its radioactive decay, radon occurs as a noble gas with very little chemical activity. After formation from the parent radium, radon is ejected from its site of formation by alpha recoil, in which the momentum of the alpha particle must be balanced by that of the residual radon nucleus. After creation, radon moves in the environment like any other unreactive gas. In soils, it diffuses through pore spaces and is carried with mass flow of groundwater, in which it is sparingly soluble. Once in contact with the atmosphere or soil gas, it readily escapes from water and moves in the atmosphere via diffusion, turbulence, and mass flow. The atmosphere is the sink for radon, and concentrations are controlled by the balance between radon emanation and its radioactive decay. Little radon escapes the troposphere. Radon dissolved in domestic water from groundwater sources readily volatilizes into buildings for subsequent inhalation. Radon gas is passively absorbed by biota without any accumulation. Reference: ATSDR 1990d.

The toxicological implications of radon cannot be understood without consideration of the environmental properties of its short-lived progeny. After decay of the radon, these solid elements attach to particles in the air and to other surfaces. Only those that attach to particles of respirable size have any significant potential for damage to health or the environment. The particles with the radon progeny move in the environment as suspended particulates with some settling and (outdoors) precipitation scavenging. Because of their short half-lives, however, the potential for human or environmental exposures is limited to areas near the point of radon decay. Most progeny of radon released to the atmosphere probably decay in the atmosphere before deposition or inhalation.

Polonium: Polonium (specifically Po-210) in the environment occurs as the last radioactive member of the uranium radioactive decay series. Its occurrence is therefore influenced by the movement of the precursor radionuclides, especially mobile radon and relatively long-lived Pb-210. After formation, its fate is influenced only by processes that occur relatively rapidly, as its half-life is only 138 days. Deposition of polonium or polonium precursors from air onto plants (e.g., tobacco) and uptake by foraging animals is well documented. The occurrence in plants is probably due much more to deposition than to uptake from soil. Transport by slower water-mediated pathways is probably not very important. Reference: NAS 1988.

Potassium: Potassium is easily and widely distributed in the environment by both abiotic and biotic processes. Potassium from power plant emissions will quickly equilibrate with all other potassium in the environment, making it essentially impossible to identify any impact due to the emissions.

ENVIRONMENTAL TOXICITY: As with toxicity to humans, the environmental toxicity of the naturally occurring radionuclides covered in this profile is probably due mainly to radiation and not to any chemical properties. Even for uranium, whose chemical toxicity can be important for relatively highly exposed humans, the chemical toxicity from environmental levels of uranium is likely to be unimportant. As part of the atomic weapons and energy programs, the radioecology of fission products, transuranic elements, and enriched uranium has been extensively studied, but that of the natural radionuclides less so. Presumably, non-laboratory mammals have the same general susceptibility to diseases from internally deposited radionuclides as laboratory species and humans. Non-mammalian species typically have different radiosensitivities than mammals, often being less sensitive. Although exposures to high levels of radiation (e.g., from the cobalt-60 source at Brookhaven National Laboratory) have changed the species composition of ecosystems, it is less clear whether environmental levels of radioactivity can cause any changes of lasting significance. The International Atomic Energy Agency has reviewed the sensitivity of terrestrial and aquatic organisms and has concluded that, while effects on individual members of a species may occasionally occur, "chronic dose rates of 1 $mGy(d)^{-1}$ or less to even the most radiosensitive species in terrestrial ecosystems are unlikely to cause measurable detrimental effects in populations." (IAEA 1992) In aquatic ecosystems, the limiting dose rate is at least 10 $mGy(d)^{-1}$, according to the IAEA. 1 mGy per day is approximately 37 rems per year and is hundreds of times the radiation protection standards used to protect human populations.

REGULATORY STATUS: All radionuclides are considered to be human carcinogens by virtue of their radioactivity. Thorium dioxide is specifically designated as having sufficient evidence or human carcinogenicity by the U.S. National Toxicology Program. Radionuclides are designated as Group A (human) carcinogens. Exposure to radionuclides in air is regulated by the Environmental Protection Agency (EPA) through the National Emission Standards for Radionuclides under its Hazardous Air Pollutants program. EPA is in the final stages of issuing its Maximum Contaminant Levels for radionuclides in Drinking Water; proposed MCLs are shown in Table 2. Radionuclides are covered by the Comprehensive Environmental Restoration, Cleanup and Liability Act (Superfund). Their Reportable Quantities under CERCLA are shown in Table 2. The Superfund Amendments and Reauthorization Act also requires industries to report discharges of thorium dioxide under Title III. The Resource Conservation and Recovery Act specifically exempts "source, special nuclear, and byproduct materials," which in effect excludes most radionuclides from RCRA attention. The EPA does not directly regulate radon in indoor air, but advises remediation whenever measured concentrations exceed 4 pCi/l. The EPA's soil cleanup levels for radium at uranium mill tailing sites, 5 pCi/g averaged over the first 15 cm. and 15 pCi/g averaged over each succeeding 15 cm. depth, are widely used elsewhere as a "standard" not only for Ra-226 but for some other radionuclides as well. Similarly, the radon emanation rate limit for uranium mill tailings of 20 pCi/m^2-sec is sometimes used elsewhere. Radionuclides associated with nuclear power or weapons are regulated by the U.S. Nuclear Regulatory Commission (NRC[1]). Concentrations permitted by the NRC in off-site air and water are shown in Table 2. The U.S. Occupational Safety and Health Administration has set Permissible Exposure Levels for radionuclides in workplace air by reference to NRC standards for licensed facilities. Radionuclides are regulated in bottled water, food, drugs, cosmetics, and medical devices by the U.S. Food and Drug Administration and in consumer products by the U.S. Consumer Product Safety Commission. The U.S. Department of Transportation has stringent requirements for the shipping of radioactive materials, although some of the naturally occurring radionuclides have too low specific activity to be covered. DOT is also concerned about the potential flammability of metallic uranium and thorium.

(References: NTP 1991; HSDB 1993a,b,c,d)

[1] Not to be confused with the National Research Council whose reports are cited elsewhere in this profile.

Table 2
Regulatory Status of Selected Radionuclides

Radionuclide	Proposed Maximum Contaminant Level (pCi/l)	CERCLA Reportable Quantity (Curie)	NRC Limit[d]		OSHA PEL[a] (μCi/ml except where shown)
			Air (μCi/ml)	Water (μCi/ml)	
uranium-238	[a]	0.1	5×10^{-12}	4×10^{-5}	1×10^{-10}
uranium-235	[a]	0.1	4×10^{-12}	3×10^{-5}	1×10^{-10}
uranium-234	[a]	0.1	4×10^{-12}	3×10^{-5}	1×10^{-10}
thorium-232	[b]	0.001	1×10^{-12}	4×10^{-5}	3×10^{-11}
thorium-230	[b]	0.01	3×10^{-13}	3×10^{-5}	1×10^{-11}
thorium-228	[b]	0.01	2×10^{-13}	1×10^{-5}	6×10^{-12}
radium-228	20	0.1	1×10^{-12}	3×10^{-5}	4×10^{-11}
radium-226	20	0.1	2×10^{-12}	3×10^{-5}	5×10^{-11}
radium-224	[b]	10	2×10^{-11}	5×10^{-6}	7×10^{-10}
radon-222	300	0.1	3×10^{-9}	–	3×10^{-7}
radon-220	[b]	0.1	1×10^{-8}	–	3×10^{-7}
lead-210	[c]	0.01	8×10^{-12}	2×10^{-4}	2×10^{-10}
bismuth-210	[c]	10	2×10^{-10}	4×10^{-5}	6×10^{-9}
polonium-210	[b]	0.01	7×10^{-12}	3×10^{-5}	2×10^{-10}
total other alpha	15				
natural uranium	30		5×10^{-12}	3×10^{-5}	1×10^{-10}; $200\,\mu g/m^3$
natural thorium	[b]		2×10^{-12}	2×10^{-5}	6×10^{-11}
potassium-40	[c]	1	–	–	–

[a] Included in total uranium
[b] Not specifically given, but covered under total adjusted gross alpha limitation of 15 pCi/l
[c] Not specifically given, but covered under beta and photon emitter limitation to 4 mrem/yr
[d] For insoluble compounds. NRC also supplies values for soluble compounds.
Sources: EPA 1991, NRC 1991, HSDB 1993a,b,c,d, ATSDR 1990a,b,c,d

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TOXICOLOGY PROFILE

SELENIUM AND SOME OF ITS COMPOUNDS

Synonyms: Elemental selenium, gray selenium

CAS No: 7782-49-2 - elemental form
7783-08-6 - selenic acid
7446-08-4 - selenium dioxide
7488-56-4 - selenium sulfide

Boiling point: 685 - 690 °C

Color: As a liquid, selenium has a brownish-red color. The amorphous crystalline form has a red color while the vitreous crystalline form is black. The hexagonal crystalline form is the most stable form and is grey in color.

DOT designation: Poison

Flammable limits: Selenium burns in air with a bright blue flame and emits a horseradish like odor. Fires involving selenium can release toxic decomposition products.

Autoignition: NA
Flash point: NA

Henry's law constant: NA

Melting point temp.: 170-217 °C

Molecular Formula: Se

Molecular weight: 78.96

Odor: Odorless but selenium dioxide has a pungent sour smell and hydrogen selenide is said to simply have a disagreeable odor.

Threshold: 0.0002 for selenium dioxide
Recognition: NA
Characteristics: Sour; disagreeable

pH: NA

Solubility
water: Selenium oxides are soluble in water but the metallic and sulfide forms are considered to be insoluble.

K_{ow}: NA

other: Soluble in sulfuric acid, aqueous potassium cyanide, and aqueous alkaline

solutions. Also soluble in carbon disulfide and benzene.

K_{oc}: NA

Specific gravity: 4.26 - 4.81

Vapor density: NA

Vapor pressure: >0.001 mm Hg at 20 °C

Viscosity: NA

NA = not available
(References: Merck, 1989; NIOSH, 1985; HSDB, 1990)

COMPOSITION: There are no true deposits of selenium and on the average it is found only at concentration of 0.09 ppm in the earth's crust. Hence, selenium cannot be economically recovered directly. It is found in small quantities in pyrite but usually is found the sulfide ores of heavy metals (Merck, 1989).

USES: Selenium is used as a red pigment in the manufacture of glass, in the electronics industry the manufacture of photocells and rectifiers, a vulcanizing agent in processing of rubber, production of metal alloys, textiles, petroleum, medical therapeutic agents and a toning agent in photographic emulsions.

ACUTE TOXICITY: Elemental selenium has a general low order of toxicity but all selenium salts may produce toxicity by ingestion, inhalation and percutaneous absorption. The acute oral LD_{50}s in rats reported for different forms of selenium are presented in Table 1.

Table 1. LD_{50} values in the rat for different chemical species of selenium.

Species	LD_{50} or Lethal Value	Form
oral-rat	6700 mg/kg	metallic selenium
oral-rat	12.5 mg/kg	sodium selenite solution
oral-rat	7.0 mg/kg	sodium selenite shampoo

(Reference: CESARS, 1990.)

Ingestion: Ingestion of toxic doses of selenium can produce salivation, vomiting and a garlic-odor on the breath. Fatty degeneration of the liver and cirrhosis have also been reported as well as mild tubular degeneration in the kidney (HSDB, 1990). Chronic selenium toxicity was reported in individuals ingesting 3.2 - 6.7 mg/day. (HSDB, 1990). Recovery from acute selenium toxicity is generally rapid (Hogberg and Alexander, 1986).

Rats fed selenium showed slight liver damage at concentrations of 5.6 to 8.33 ppm dietary selenium. Swine fed this concentration did not show any effects except for an increase in glutathione peroxidase (Goehring et al., 1984). However, a condition of poliomylomalacia was induced in swine at a dose level of 52 ppm sodium selenite in the diet (Goehring et al., 1983).

Inhalation: Exposure to elemental selenium dusts can produce respiratory tract irritation manifested by nasal discharge, loss of smell, epistaxis and cough (Ellenhorn and Barceloux, 1988). There may also be difficulty in breathing, frontal headaches and dyspnea (HSDB, 1990). Pneumonitis and delayed pulmonary edema have been reported among workers with brief high level exposure to selenium dioxide (Buchan, 1947). The most toxic selenium compound found in ambient air and occupational settings is hydrogen selenide. Hydrogen selenide is a severe pulmonary irritant, resulting in bronchitis, pneumonitis, and pulmonary edema. Early signs of toxicity include dizziness, fatigue, metallic taste and garlic odor on the breath and have been reported to occur at levels of 0.066 mg/m^3 hydrogen selenide (Buchan, 1947).

Skin contact: Skin contact with selenium containing material can produce a dermatitis. Workers have reported dermatitis on the backs of their hands (Patty, 1981).

Selenium sulfide was applied to the shaved backs of the mice for 5 days/week for 13 weeks. Histopathological skin alterations were observed in animals that received 5 - 10 mg/kg. Interstitial nephritis developed in all but the lowest dose group and one animal from each of the two high dose groups developed a focal necrosis of the liver. No pathological changes were seen in the groups that was dosed with 1 mg/kg (NCI, 1980).

Eye contact: Acute industrial exposure irritates the mucous membranes and produces redness of the eyes (HSDB, 1990).

CHRONIC TOXICITY: The known human health effects due to chronic oral exposure to selenium compounds such as deformed fingernails, loss and brittle hair, excessive tooth decay and in extreme cases numbness and ataxia

are not considered to be specific to selenotoxicosis. As a result, blood levels need to confirm the condition of selenosis. However, blood levels can vary with populations. For example, the Finnish population averages 0.055 mg Se/l but the U.S. population shows an average of 0.206 mg Se/l (ATSDR, 1989).

Ingestion: In a series of toxic cases where the estimated ingestion rate ranged between 27 - 2,310 mg, nausea, vomiting, nail changes, fatigue and irritability were the most common manifestations of toxicity (Ellenhorn and Barceloux, 1988).

High dietary levels of selenium have been investigated in South Dakota (Smith et al., 1937). The most common symptoms signs were a high urinary excretion of selenium (0.20 - 1.98 µg/ml) along with gastrointestinal disturbances , icteroid discolorations of the skin, decayed teeth and less frequently, jaundice, dermatitis and pathological nails.

Chronic oral exposure to high levels of selenium in water or in foods grown in high selenium soil may be toxic. Hair and nail loss, dermatitis as well as a numbness , paralysis, and hemiplegia have been observed in an area in China with high levels of selenium in the soil (Yang et al., 1983). Daily intake of selenium in this area was estimated at 5.0 mg/day vs. 0.1 mg/day in unaffected regions.

Individuals may also self administer high doses of selenium in over-the-counter selenium preparations and develop adverse effects.

In domestic animals, a syndrome termed "blind staggers" can develop in livestock due to grazing on plants that accumulate selenium. Impaired vision, depressed appetite and a tendency to wander in circles is characteristic. The latter stages are characterized by paralysis and death from respiratory failure (Moxon and Rhian, 1943). In a more chronic form of selenotoxicosis termed "alkali disease" the animals show a loss of vitality, loss of appetite, emaciation, deformation and shedding of the hoofs, loss of long hair and erosion of the joints of the long bones. Advanced cases may also show signs of liver cirrhosis (Moxon and Rhian, 1943).

Inhalation: Long term inhalation of selenium dusts have produced bronchial irritation, gastrointestinal distress, nasopharyngeal irritation and persistent garlic odor on the breath. The odor is believed to be due to the excretion of dimethyl selenium. Other long term exposure studies report pallor, irritability, excessive fatigue, coated tongue, nervousness, and metallic taste in the mouth (Clayton and Clayton, 1981).

Studies of workers exposed to selenium fume have noted irritation of the eyes, nose, throat and respiratory tract. Workers have also reported skin rashes, garlic odor on the breath, headaches and indigestion associated with exposure to inhaled selenium fume (0.2 - 3.5 mg/m^3).

In an urban atmosphere, inhalation of selenium will not contribute significantly to the human body burden and accounts only for a small fraction of the organ concentration of selenium (Medinsky et al., 1985).

Skin Contact: Chronic ingestion of high selenium diets have reportedly produce icteroid discoloration of the skin, dermatitis and pathological nails (Smith et al., 1937).

Skin contact with selenium dioxide may produce burns and dermatitis (Glover, 1970).

SENSITIZATION: Airborne selenium dioxide can produce an allergic reaction in the eyes manifested by conjunctivitis. Occupational long term exposure may cause a condition of "pink eye" where the eyelids become puffy and discolored. Occasionally, skin contact with selenium dioxide has been reported to produce a urticaria (Glover, 1970) while selenite reportedly produced a contact dermatitis (Senff et al., 1988) with a papilla-follicular morphology (Richter et al., 1987).

TARGET ORGAN EFFECTS: Although animal studies indicate that the liver is a target organ of chronic excess selenium, liver effects in humans have not yet been documented (ATSDR, 1989). Under conditions of selenium deficiency, this element is preferentially distributed to the brain, the reproductive organs and the other endocrine glands (Behne et al., 1988).

ABSORPTION-METABOLISM-EXCRETION: Whole blood selenium levels in individuals range between 1.33 to 7.5 µg/ml. Normal level in the U.S.A. are approximately 0.1 µg/ml (HSDB, 1990).

The rate of absorption of elemental selenium or selenium sulfide is low but water soluble selenium compounds are effectively absorbed from the gastrointestinal tract. Thus more than 90% of administered selenite or better than 80 % of some organoselenium compounds are absorbed by rats. The efficiency of selenite absorption in humans may be in the range of 40 -80 % but the absorption of selenomethionine may be around 75-90% (Friberg, 1986).

Inhalation studies in rats show that 94% of deposited selenious acid or 57% of elemental selenium is absorbed from the lung within four hours (Friberg, 1986).

There is rapid distribution of water-soluble selenium compounds to the organs but after 24 hours, the selenium remains primarily in the liver and kidneys. In the liver, selenium is biotransformed into excretable metabolites. Excretion is principally by the urine in the form of trimethylselenide. However, the feces, sweat and breath can also contribute to excretion. At toxic doses, the respiratory pathway of excretion can become significant and the selenium is eliminated in the form of dimethylselenide (Clayton and Clayton, 1981). When compared to other metals, the elimination of selenium from the body is at a moderate rate (Gregus and Klaassen, 1986).

On the basis of six human subjects, a tracer dose of ^{74}Se showed 84% absorption with 65% retention after 12 days and 35% retention after 90 days. The retained portion appears to reside in the general tissue pool (Patterson et al., 1989).

IMMUNOTOXICITY: There are a number of limited studies on the beneficial and adverse effects of selenium compounds on the immune system. However, the data are inconclusive (ATSDR, 1989). Selenium appeared to increase the host resistance of mice towards subsequent Klebsiella pneumonia infection even though it may have had a detrimental effect on the antibody response. Thus, it remains to be established what immunotoxic consequence acute or chronic exposure to selenium may have in man (Laschi-Loquerie et al. 1987).

NEUROTOXICITY: Dizziness, decreased reflexes, CNS depression and coma have been reported to be associated with selenium exposure (ATSDR, 1989).

REPRODUCTIVE TOXICITY: Selenium has been shown to exert a protective action against cadmium induced testicular damage in mice (Sugawara, et al., 1989), but when fed to rats at a level of 12.5 ppm in wheat, it produced testicular weight reduction and interrupted spermatogenesis (Parshad and Sud, 1989).

The concomitant feeding of selenium inhibited the fetotoxicity induced by methylmercury in mice (Nishikido et al., 1987).

Selenium has been shown to cross the placenta of dogs and mice and is transferred to suckling offspring by milk in dogs and rats. High selenium levels in soil and water in the Kesterson Reservoir area of the San Joaquin Valley in California is believed to account for the gross deformities seen in the local game birds (ATSDR, 1989). Sodium selenite at a level of 10 ppm in the diet reduced the hatchability of mallard duck eggs (Hoffman and Heinz, 1988).

GENOTOXICITY: Human whole blood cultures were exposed to selenium in different valence states. The ability of Se compounds to induce sister chromatid exchanges in decreasing order of effectiveness were elemental selenium, selenium dioxide, sodium selenide, sodium selenite, and sodium selenate (Ray and Altenburg, 1980). Selenite was able to induce single strand breaks in the DNA of at hepatocyte cultured *in vitro* (Garberg et al., 1988).

Selenium was found to be non-mutagenic in the Ames Assay, reduce the mutagenic potential of cigarette smoke (Chortyk et al., 1988) and inhibit the mutagenic potential of Benzo (a)pyrene, 3 methylcholanthrene and 3 methylcholanthrene (Prasanna et al., 1987).

CARCINOGENICITY: Available data provide no suggestion that selenium is carcinogenic to man (IARC, 1987). Instead, studies have suggested that selenium may be protective against cancer. Using an ecologic design (studying groups rather than individuals), researchers noted elevated rates of cancer in regions with low levels of selenium in the soil and crops grown for livestock (Shamberger and Frost, 1969; Schamberger and Willis, 1971). However, results of ecologic studies are limited by the lack of information on individual risk factors that may impact on cancer rates. Based on current evidence, there is insufficient data regarding the role of selenium in the prevention of human cancer.
In mice, arsenic and selenium, when administered alone were reported to reduce the size of pulmonary adenoma tumors induced by the administration of urethane (Blakley, 1987). Selenium was also protective against tumor production by other known carcinogenic agents (Balansky et al., 1983) and as little as 1 ppm in the diet was protective towards the development of mammary cancer in mice (Whanger, 1983).

EPIDEMIOLOGY: Cross-sectional studies of workers have reported irritant effects on the skin, eyes and upper respiratory tract associated with exposure to selenium. Epidemiological studies using an ecologic design have found elevated cancer rates among communities with

low levels of selenium in soil. These studies are cited under CARCINOGENICITY.

ENVIRONMENTAL FATE: Selenium is associated with volcanic effluent, igneous rocks (0.05 ppm); shales 0.- 0.6 ppm; sandstone (0.0 - 0.05 ppm); limestones (0.08 ppm and soils (0.2 ppm). As a result, this element finds its way into surface waters and drinking water supplies (HSDB, 1990). The form of selenium in the soil depends on the pH, and redox potential. At equilibrium, most soil selenium should be elemental selenium (Parr et al. 1983). In acid or neutral soils, the biologically active selenium will steadily decline due to incorporation into plants. Agricultural and industrial activity may accelerate this decline. In dry areas with alkaline soils, and oxidizing conditions, the weathering of rocks can maintain biologically active selenium in the soil (HSDB, 1990).

In the atmosphere, selenium is predominantly associated with particulate materials. Up to 90% of the selenium content in ambient air is emitted during the burning of fossil fuels in the form of elemental selenium and selenium dioxide. Selenium dioxide reacts with water or sweat to form selenious acid which is an irritant (Clayton and Clayton, 1981).

The bioavailability of inorganic selenium is highest for highly soluble sodium selenate and lowest for insoluble elemental selenium and selenium sulfide. Sodium selenate is readily taken up by plants and organic selenium compounds like those found in plants, fish and shellfish are highly bioavailable (ATSDR, 1989).

The reported bioconcentration factor for selenium ranges from 150 to 1,850 while the bioaccumulation factor ranges from 1,746 to 3,975 (Lemly, 1985). The potential for selenium to bioaccumulate in aquatic and terrestrial organisms is vividly presented in the account of the Kesterson Reservoir agricultural drain in California (Fan et al. 1988).

In plants, the primary bioaccumulators consist of grasses and grains and can contain 100 - 100,000 mg Se/kg of plant tissue (Rosenfeld and Beath, 1964). Fruits and vegetable are generally low in selenium content (Schubert et al., 1987).

ENVIRONMENTAL TOXICITY: Selenium is a trace element which is essential in low amounts for adequate human nutrition and development. The safe and adequate range of daily intake of selenium for adults is 50 -200 µg (EPA, 1980). The high total daily intake of selenium which did not produce selenosis was 1.5 mg/d. A form of heart disease termed "Keshan Disease", occurring among women and children in China, has been associated with long-term ingestion of selenium-deficient diets.

Excessive exposure to selenium can produce lethality. Table 2 presents the LC 50 values for various vertebrate and invertebrate species.

REGULATORY STATUS: The Food and Nutrition Board of National Research Council has suggested a range of 50 - 200 µg/day as an adequate dietary intake. The upper limit of 200 µg/day is a conservative one. Observations in Japanese fisherman suggests that intakes of selenium from 10 - 200 times normal have not been shown to produce toxic effects. Ingestion of 31 mg/day for 11 days produced symptoms of toxicity.

Table 2. Ecotoxic values of selenium for non mammalian organisms.

Species	Duration	LC50 (mg/l)	Type	Form
goldfish (Carassius auratus)	168	8.78	--	sodium selenate
rainbow trout (Salmo gairdneri)	96	1.8-4.5	static	sodium selenate
rainbow trout	96	8.8-12.5	flow-through	sodium selenate
goldfish (Carassius auratus)	168	12		selenium dioxide
rainbow trout (Salmo gairdneri)	120	2.7-2.75		sodium selenite
Northern pike (Exos lucius)	76	11.1		sodium selenite

creek chub (Semotilus atromaculatus)	48	12.0		selenium dioxide
brook trout (Salvelinus fontinalis)	96	10.2	flow-through	selenium dioxide
goldfish (Carassius auratus)	96	26.1	flow-through	selenium dioxide
fathead minnow (Pimephales promelas)	96	1.7-11.3	static	sodium selenite
fathead minnow (Pimephales promelas)	96	.62-.970	flow-through	selenious acid
white sucker (Catostomus commersoni)	96	29-31.4	flow-through	sodium selenite
striped bass (Marone saxatilis)	96	1.3-2.4	static	sodium selenite
channel catfish (Ictalurus punctatus)	96	13.6	flow-through	selenium dioxide
channel catfish (Italurus punctatus)	96	66	static	sodium selenate
flagfish (Jordanella floridae)	96	6.5	flow-through	selenium dioxide
mosquito fish (gambusia affinis)	96	12.6	static	sodium selenite
bluegill (Lepomis macrochirus)	96	12	static	sodium selenite
bluegill (Lepomis macrochirus)	96	28.5	flow through	selenium dioxide
bluegill (Lepomis macrochirus)	96	63	static	sodium selenate
yellow perch (Perca flavescens)	96	11.7	flow through	sodium selenite
rainbow trout (Salmo gairdneri)	96	24	static	sodium selenate
rainbow trout (Salmo gardneri)	96	5.5-47	flow-through	sodium selenate
fathead (Pimphales promelas)	96	11-12.5	static	sodium selenate
Invertebrates				
amphipod (Gammarus pseudolimnaous)	96	1.7-4.3	static	sodium selenite
amphipod (Gammarus pseudolimnaous)	96	0.34	flow-through	sodium selenite
amphipod (Gammarus pseudolimnaous)	96	0.75	static	sodium selenate
amphipod (Hyalella azteca)	96	0.76	flow-through	sodium selenate
midge (chironomus plumosus)	96	24.15-27.85	static	sodium selenite
midge (chironomus plumosus	96	42.5	flow-through	selenium dioxide
midge (paratanytarsus parthenogenetic)	96	20	static	sodium selenate

ostracod (Cyclocypris sp.)	48	130		sodium selenite
snail (Lymnaea stagnalis)	144	15		sodium selenate
snail (Aplexa hypnorum)	96	193	static	sodium selenate
snail (Aplexa hypnorum)	96	23-53	static	sodium selenite
hydra (Hydra sp.)	96	7.3	static	sodium selenate
hydra (Hydra sp.)	96	1.7	static	sodium selenite
leech (nephelopsis obscura)	96	442	static	sodium selenate
leech (Nephelopsis obscura)	96	203	static	sodium selenite
cladoceran (Daphnia magna)	48	.21-5.3	static	sodium selenate
Cladoceran (Daphnia magna)	48	.45-2.37	static	sodium selenite
cladoceran (Daphnia magna)	48	.43-1.22	static	selenious acid

(Reference: CESARS, 1990.)

Selenium is considered to be a toxic pollutant pursuant to section 307 (a) (1) of the Clean Water Act and is subject to effluent limitations. The MCL in drinking water is .01 mg/l. Exposure standards for occupational settings are presented in the following table (Table 3).

TABLE 3. Worker Exposure Limits

ACGIH TWA: 0.1 mg/m^3
STEL: 0.3 mg/m^3
Ceiling: NA
OSHA-PEL: 0.2 mg/m^3 as selenium
IDLH: 100 mg/m^3

(References: NIOSH, 1985; HSDB, 1990)

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TOXICOLOGY PROFILE

TOLUENE

Synonyms: Toluol, methylbenzene, methacide, phenylmethane.

CAS No: 108-88-3

Conversion factor: 1 ppm - 3.76 mg/m^3

Boiling point: 110.625 °C

Color: Colorless

DOT designation: Flammable liquid

Flammability limits
- **Autoignition temp.:** 480°C
- **Flash point:** 40
- **LEL:** 0.9%
- **UEL:** 9.5%

Henry's law constant: 6.37E-03

Melting point: 139°C

Molecular formula: C_7H_8

Molecular weight: 92.13

Odor:
- **Threshold:** 2.9 ppm, v/v
- **Recognition:** Same
- **Characteristics:** Sweet, pungent

pH: NA

Solubility
- **Water:** 535 mg/l
- **K_{ow}:** 2.79
- **other:** Miscible with alcohol, ether, acetone, chloroform, carbon disulfide and glacial acetic acid.
- **K_{oc}:** 2.47

Specific gravity: 0.87

Vapor density: 3.14 (air = 1)

Vapor pressure. 28 mm @ 25°C

Viscosity: 0.587 mN-s/(m)^2 (milli-Newton second per square meter) @ 20°C.

NA = not applicable
(References: ACGIH, 1989; OSHA, 1989; Lange, 1985)

COMPOSITION: Toluene consists of seven carbon atoms and 8 hydrogen atoms forming a 6 carbon ring and one methyl group.

USES: Toluene is a component of crude oil. In the United States, about 70 percent of the toluene produced is used to make benzene; 15 percent is used as a feedstock; 15 percent is used in the production of other chemicals; and the balance is used directly as a component of gasoline (6-7% by volume) or as a solvent for paints and coatings (EPA, 1980; Verschueren, 1983).

ACUTE TOXICITY:

Ingestion: Toluene exhibits relatively low acute oral toxicity. Oral LD50s in laboratory animals range from 2,000 to 7,000 mg/kg body weight (EPA, 1980). In humans, acute oral exposure may include a burning sensation in the oropharynx and stomach, abdominal pain, hematemesis, nausea, vomiting, and weakness. Other possible symptoms include dizziness, euphoria, headache, tightness in the chest, and staggering. Symptoms of more severe exposure include blurred vision, tremors, shallow and rapid breathing, ventricular irregularities, paralysis, unconsciousness, and convulsions (Driesbach, 1966). Most of these symptoms are indicative of central nervous system toxicity.

Inhalation: Most of the toxicological investigations on toluene have focused on inhalation exposure. In humans, symptoms of acute inhalation toxicity are the same as those associated with acute oral toxicity. Toluene has been implicated in the death of "glue sniffers"and other solvent abusers in England (Anderson, et al.,1982, 1985). In most of the cases, death was caused by cardiac arrhythmias, central nervous system depression, asphyxia, and hepatic and renal failure. In rats, Carpenter and coworkers (1976) obtained an inhalation LC50 of 8,800 ppm for 4 hours of exposure.

Eye contact: Vapors may cause irritation of the eyes (Von Burg, 1981; Carpenter et al., 1946) at air concentrations greater than 200 ppm although at 800 ppm the irritation is still considered to be mild. Tearing may also occur. Von Oettingen et al. (1942) noted slowed pupillary reflexes and dilation of the pupils in volunteers at exposures above 200 ppm. Exposure to the liquid produced rapid cloudiness of the lens (Clayton and Clayton, 1981).

Skin contact: The acute lethality of toluene by dermal application was reported to be 14.1 g/kg (Smyth et al., 1969). In humans, dermal contact with toluene may irritate or damage the skin by its degreasing action. Wolf and coworkers (1956) observed slight to moderate irritation of the skin on the ears and body of rabbits after repeated application of toluene. When applied to the eyes of rabbits, toluene caused slight irritation of the conjunctival membranes, but no corneal injury (Hazelton Laboratories, 1962). Carpenter and Smyth (1946), however, observed severe eye injury in rabbits following direct application of toluene.

CHRONIC TOXICITY

Ingestion: In a recent review of the literature on the toxicity of toluene, the Agency for Toxic Substances and Disease Registry (ASTDR, 1988) reported that no studies were found regarding the respiratory, cardiovascular, musculoskeletal, or gastrointestinal effects of orally administered toluene.

Renal and hepatic effects were reported by Wolf and coworkers (1956) in rats administered toluene by gavage 5 days/week for 6 months. The NOAEL was 590 mg/kg/day. Neurological effects were observed by Kostas and Hotchin (1981) in mice receiving an oral dose of toluene of 98.3 mg/kg/day. This effect was reflected in reduced open field activity.

Inhalation: In humans and laboratory animals, toluene acts primarily on the central nervous system. Studies supporting this statement have been reviewed by ASTDR (1988). Effects on the central nervous system occur most rapidly when toluene is inhaled. The effects are discussed in the section on neurotoxicity.

In humans, the only observed effect of chronically inhaled toluene on the respiratory tract is irritation. Respiratory tract irritation was observed in workers exposed for several years to 200-800 ppm of the compound (Parmeggiani and Sassi, 1954); however, a single 7 to 8 hour exposure to 800 ppm did not cause respiratory tract irritation in human volunteers (Von Oettingen et al., 1942).

Lung irritation occurred in rats exposed to 600 ppm of toluene for 5 weeks and rats exposed to 2500 and 5000 ppm developed pulmonary lesions (von Oettingen, et al, 1942). However, no signs of respiratory distress or histological abnormalities of the lung or heart were observed in mice exposed to toluene at concentrations of 4,000 to 12,000 ppm for 8 weeks (Bruckner and Peterson, 1981b). In a study by CIIT (1980), no histopathological lung or heart lesions were found in mice exposed to 300 ppm of toluene for 24 months.

A moderate increase in heart rate was observed in rats exposed to near-lethal concentrations of toluene (Vidrio, et al., 1986). These results, coupled with those of Bruckner and Peterson (1981a) and CIIT (1980) suggest that toluene does not seriously affect cardiac function even though the cardiac arrhythmia has been cited as a cause of death of solvent abusers.

Toluene may affect the blood elements; however, the picture is not clear. Decreased leukocyte counts have been observed in laboratory animals exposed to toluene (Hobara, et al., 1984; Horiguchi and Inoue, 1977) and reduced hematocrit and mean corpuscular hemoglobin levels were reported by CIIT (1980) in female rats exposed to 100-300 ppm of toluene.

Although similar effects have been observed in occupationally exposed individuals, the occurrence of effects in workers has not been consistent. Tahiti and coworkers (1981) reported that some of the workers in a tarpaulin factory had reduced leukocyte counts. These workers had been exposed to toluene concentrations ranging from 20 to 200 ppm for several years. On the other hand, studies by Yin and coworkers (1987), Banfer (1961) and Masushita and coworkers (1975) failed to uncover any hematological effects among workers exposed to toluene.

Although liver and kidney failure has been cited as one of the causes of death among solvent abusers, studies with humans and laboratory animals have not produce evidence that the liver or kidney are critical target organs for toluene fatality (ATSDR, 1988).

Eye contact: see Acute toxicity: Eye contact.

Skin contact: Due to its volatile properties, incidental contact with toluene without an occlusive cover will not result in appreciable dermal absorption (Low et al., 1988). However, due to the lipid solubility properties, it can be envisioned that repeated and prolonged dermal contact can produce a dry and fissured dermatitis (Key et al., 1977).

SENSITIZATION: There is very little evidence that toluene is an allergic sensitizer. Mice exposed for five days to 2.5 to 500 ppm of toluene exhibited increased sensitivity to respiratory infection by *Streptococcus zooepidemicus* (Aranyi, et al., 1985). Workers exposed to toluene, benzene and xylene had significantly lower serum IgG and IgA levels (Lange et al., 1973).

TARGET ORGAN EFFECTS: The major target organ for toluene is the brain.

ABSORPTION-METABOLISM-EXCRETION: Toluene is readily absorbed from the respiratory and gastrointestinal tracts and to a lesser degree through the skin.

In a study with human volunteers, Astrand and coworkers (1972) detected toluene in the arterial blood within 10 seconds after the volunteers were exposed to 100 and 200 ppm of toluene in air. Toluene levels in the blood rose rapidly in the first few minutes, then rose more slowly during the remainder of each 30 to 60 minute exposure session. The average arterial blood toluene levels appeared to approach equilibrium between 20 and 30 minutes of exposure time. At equilibrium, blood toluene levels in individuals exposed to 100 or 200 ppm toluene were 1 and 2 µg/ml, respectively.

Absorption from the gastrointestinal tract occurs more slowly than from the lungs. Pyykko and coworkers (1977) reported that peak toluene levels in rat tissues occur in 15-30 minutes following a 10 minute inhalation session. However, peak tissue levels did not occur for 2- 3 hours following gastric intubation and were lower than the peak levels observed after inhalation. Dutkiewicz and Tyras (1968) applied toluene to the forearm of human volunteers and measured the rate of toluene disappearance from a closed chamber covering the exposed skin. They calculated an absorption rate of 14 to 23 $mg/cm^2/hr$.

Paterson and Sarvesvaran (1983) and Takeichi and coworkers (1986) measured toluene concentrations in selected tissues of individuals who died from toluene exposure. They found measurable levels of toluene in the brain, blood, lungs, and liver. In a study with mice, Bergman (1979) found high levels of radioactive toluene in the body fat, bone marrow, spinal nerves, spinal cord and white matter of the brain immediately after inhalation exposure. High levels of radioactivity were found in the medullary region of the kidney immediately after cessation of exposure.

The metabolism of toluene in humans and laboratory mammals has been well characterized. According to ATSDR (1988), toluene is initially converted primarily to benzyl alcohol by cytochrome P-450 enzymes in the liver. In turn, benzyl alcohol is metabolized to benzaldehyde and subsequently to benzoic acid. Benzoic acid is activated by liver enzymes to form a coenzyme A derivative, which reacts with glycine to form hippuric acid, which is excreted in the urine. Benzoic acid also reacts with glucuronic acid to form benzoyl glucuronide. Toluene is also converted by P-450 enzymes to o- and p- cresol, both of which are conjugated with sulfate or glucuronic acid and excreted in the urine.

In humans and laboratory mammals, 60-70 percent of the toluene absorbed from the lungs is excreted as hippuric acid via the urine, and excretion occurs within 12 hours of exposure (Ogata, et al., 1970). Excretion after oral or dermal exposure is unknown.

IMMUNOTOXICITY: Workers exposed to toluene, benzene and xylene had significantly lower serum IgG and IgA levels (Lange et al., 1973).

REPRODUCTIVE TOXICITY: API (1985) investigated the effects of toluene on reproduction and reported that the compound had no effect on reproduction in mice exposed to concentrations as high as 2,000 ppm in air. API (1981) also reported that exposure of male mice to 400 ppm of toluene for 8 weeks did not induce dominant lethal mutations in sperm cells.

CIIT (1980) exposed rats to 300 ppm of toluene for 24 months and reported finding no histopathological lesions of the ovaries or testes that could be attributed to toluene exposure.

Toluene has been shown to adversely affect fetal development both in humans and laboratory animals when inhaled. Syrovadko (1977) reported that a group of women occupationally exposed to toluene and other solvents through use of varnishes exhibited a relatively high incidence of menstrual disorders. Newborn children of these women were reported to experience more frequent fetal asphyxia, to be more often underweight, and nurse more poorly than "normal" children. Matsushita and coworkers (1975) reported that women chronically exposed to 60-100 ppm of toluene in shoe manufacturing operations complained frequently of dysmenorrhea.

In laboratory animals, inhalation exposure to toluene has caused increase in the incidence of skeletal anomalies, signs of retarded skeletal development and low fetal weights (Courtney, et al., 1986; Ungvary and Tatrai, 1985; API, 1985). Effects on fetal development appear to be related to dose. At doses of 100 to 400 ppm, toluene had no affect on fetal development (API, 1978); however at a concentration of 2,000 ppm, the compound caused significant inhibition of growth of the offspring for two generations (API, 1985). It is possible that growth in the offspring was inhibited indirectly through malnourishment of the mothers. At 2,000 ppm toluene would probably cause central nervous system depression.

Nawrot and Staples (1979) found a significant increase in embryo mortality and decrease in fetal weight in mice when mothers were given toluene by gavage on days 6-15 of gestation at doses ranging from 0.3 to 1.0 mg/kg. The offspring of mothers on the 1.0 ml/kg dose also had a significant incidence of cleft palate, which was not related to retardation of growth rate. Hudak and Ungvary (1978) reported that toluene reduced body weight gain and skeletal growth in the fetuses of rats exposed continuously to 399 ppm of toluene on days 1 to 8 of pregnancy; however, the compound did not produce teratogenic effects. Roche and Hine (1968) also concluded on the basis of the results of studies with rats and chick embryos that toluene is not teratogenic. Pregnant mice receiving 2,350 mg/kg of toluene orally produced normal litters (Smith, 1983).

NEUROTOXICITY: The primary effect of toluene is neurotoxicity. In humans, the major effects of acute exposure to high concentrations of toluene are central nervous system dysfunction and narcosis. At low to moderate concentrations the compound impairs neuromuscular function and cognitive functions. Toluene also affects behavior and hearing and alters brain neurochemistry and electrical processes.

Studies of solvent abusers indicate that certain effects of toluene on the central nervous system are permanent and result in ataxia, tremors, and impaired speech, hearing and vision (Devathasan, et al., 1984; King, et al., 1981; Suzuki et al., 1983).

Rats continuously exposed to toluene for 30 days have decreased total brain weights and decreased weight of the cerebral cortex. There was also a decrease in total phospholipid content of the cerebral cortex (Kyrklund, et al., 1987).

GENOTOXICITY: Whether or not toluene is genotoxic is unclear. Schmid and coworkers (1985) and Bauchinger and coworkers (1982) reported finding an increased incidence of sister chromatid exchange in individuals occupationally exposed to toluene. Other investigators (Haglund, et al., 1980; Maki-Paakkanen, et al. 1980) found no correlation between chronic occupational exposure to toluene and increased incidence of chromosomal aberrations. Studies with laboratory animals have been negative (ATSDR, 1988; HSDB, 1990).

CARCINOGENICITY: Toluene has not been shown to be carcinogenic in any *in vitro* mutagenicity/carcinogenicity bioassay system; nor has it been shown to be carcinogenic in humans or laboratory animals (ATSDR, 1988). The most recent NTP study (1990) reported no important increases in either non-neoplastic lesions in mice or neoplastic lesions in rats and mice.

EPIDEMIOLOGY: Due to the central nervous system effects associated with toluene exposure, a number of researchers have attempted to measure subtle changes by neurobehavioral tests and measures. Cherry et al. (1985) could not detect any significant differences between exposed workers and the controls in tests such as trail making, dotting, visual search, digit symbol, block design, grooved peg board, simple reaction time, memory

or reading tests. However, Foo et al., (1990) claim differences in workers that were exposed to levels of toluene at or near the TWA and lower level exposed controls. Statistically significant differences were seen in manual dexterity (grooved peg board), visual scanning (trail making, visual reproduction), digit symbol and verbal memory (digit span) but not in finger tapping and simple reaction time.

ENVIRONMENTAL FATE: Toluene is a volatile liquid, therefore , it can be expected to enter the atmosphere where it would be oxidized primarily by reaction with hydroxyl radicals. Products of the atmospheric reactions of toluene includes benzaldehyde, nitrotoluene, acetaldehyde, formaldehyde, glyoxal and methylglyoxal (Seinfeld, 1986). The atmospheric lifetime of toluene is of the order of several hours; about 5 hours in a highly polluted atmosphere (Finalyson-Pitts and Pitts, 1986). The primary human exposure to toluene is from gasoline exhaust in urban air, gasoline vapors around filling stations or from occupational exposures to toluene based solvents (HSDB, 1990).

The compound has an aqueous solubility of 535 mg/liter at 25°C. Its volatilization half life in water has been estimated to be 5.18 hours (Callahan, et al., 1979). With a $\log(K_{ow})$ of 2.79 and a K_{oc} equal to a range of 37 - 250, it has a moderate propensity to sorb to organic matter but it is fairly rapidly evaporated or destroyed by bacterial action. If it is transported to groundwater, microbial degradation is insignificant (HSDB, 1990).

Some species of bacteria are capable of using toluene as a carbon source (Callahan, et al., 1979). The metabolic process is oxidative and involves hydroxylation of the aromatic ring to cresols and catachols, which are metabolized further to acetic acid and pyruvic acid.

ENVIRONMENTAL TOXICITY: No information was found regarding the toxicity of toluene to vegetation, terrestrial invertebrates and vertebrates, and birds. Acute toxicity estimates for toluene and selected aquatic species are presented in Table 1.

TABLE 1.
Acute Ecotoxicity Values of Toluene for Various Species.

Species	LC50 (mg/l)	Comments
Water flea *(Daphnia magna)*	60-313	LD_0 - LD_{50}
Goldfish *(Carassius auratus)*	22.8-58	24 - 96 hr LC_{50}
Fathead minnow *(Pimephales promelas)*	34.2-56	TLm 24 - 96 hr.
Bluegill *(Lepomis machrochirus)*	12.7-24	TLm 24 - 96 hr.
Pacific oyster *(Crassostrea gigas)*	1,050	n.o.s.
Mysid shrimp *(Mysidopsis bahia)*	56.3	n.o.s.
Bay shrimp *(Crago franciscorum)*	3.7 - 4.3	96 hr LC_{50}
Grass shrimp *(Paleomonetes pugio)*	9.5	96 hr LC_{50}
Striped bass *(Moroni saxitalis)*	6.3	96 hr LC_{50}

n.o.s. = not otherwise specified
(Ref: Verschueren, 1983).

REGULATORY STATUS:

Table 3.
Table of Critical Regulatory Values.

Ambient Air Quality Standard (NAAQS):
None

National Emissions Standard (NESHAP):
None

Occupational Exposure Limits

OSHA PEL: 200 ppm
STEL: 500 ppm
ceiling: 300 ppm
IDLH: 2000 ppm
ACGIH TLV: 100 ppm (375 mg/m^3) **STEL:** 150 ppm

(560 mg/m^3) **NIOSH TWA:**
100 ppm
10 min. ceiling: 200 ppm

Drinking Water Standard:
2.0 mg/l, proposed

Ambient Water Quality Criteria Health: 14.3 mg/l
Freshwater organisms:
None established

Saltwater organisms: None established

Reportable Quantity: 1000 lb

Cancer Potency: None

Reference Dose:
Oral - 0.2 mg/kg/day based on a NOAEL of 312 ppm for increased liver and kidney weights in rats converted to 223 mg/kg/day, and an uncertainty factor of 1000. The LOAEL in this study was 625 mg/kg (IRIS, 1990).

Inhalation - None established.

(References: NIOSH, 1985; EPA, 1985a;b; IRIS, 1990; EPA, 1989)

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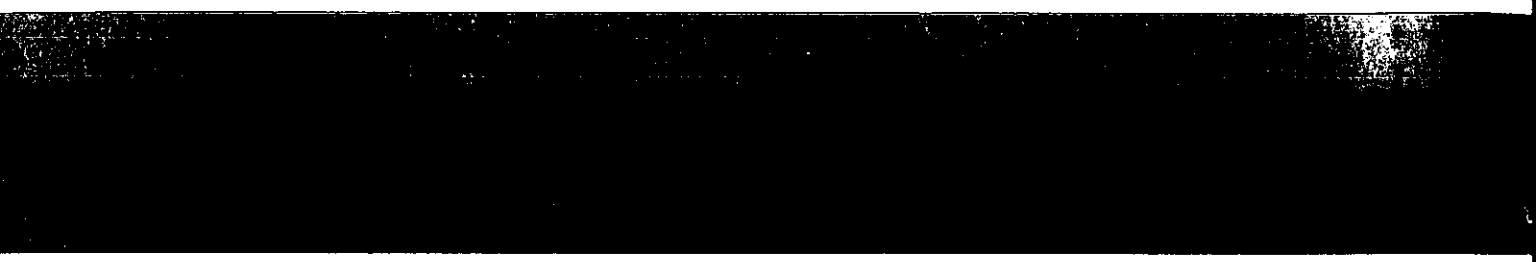

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