July 1999

Human Health and Ecological Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes

Background Document

Prepared for

U.S. Environmental Protection Agency
Office of Solid Waste
401 M Street SW (5307W)
Washington, DC 20460

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Prepared by

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List of Acronyms

ACF	age correction factor
AQUIRE	Aquatic Information Retrieval
ACR	acute-to-chronic ratio
ADD	average daily dose
ADRI	average daily rate of intake
AFs	adjustment factors
AIR	adjusted ingestion rate
AML	Arc Macro Language
AP	air concentration of particles
APCD	air pollutant control device
ASCK	area source cement kiln
ASINC	area source incinerator
AV	air concentration of vapors
BAF	bioaccumulation factor
BCF	bioconcentration factor
BPIP	Building Profile Input Program
BSAFs	bio-sediment bioaccumulation factors
CAA	Clean Air Act
CAPMS	Criteria Air Pollutant Modeling System
CDC	Centers for Disease Control and Prevention
CDFs	cumulative distribution functions
CDP	combined wet and dry deposition of particles
CINC	commercial incinerators
CK	cement kilns
Cl_2	chlorine
COC	chemicals of concern
COPD	chronic obstructive pulmonary disease
CRITFC	Columbia River Inter-Tribal Fish Commision
CSCLs	chemical stressor concentration limits
CSFs	cancer slope factors
CT DEP	Connecticut Department of Environmental Protection
DDP	dry deposition of particles
DEM	digital elevation model
DLGs	digital line graphs
EC	effective concentration
ED	exposure duration
EFH	Exposure Factors Handbook
EPA	U.S. Environmental Protection Agency
ER-L	10th percentile effects conentration
ER-M	median effects concentration

List of Acronyms (continued)

FAV	final acute value
FCV	final concentration value
FDEP	Florida Department of Environmental Protection
GIRAS	Geographic Information Retrieval and Analysis System
GIS	geographic information system
GLWQI	Great Lakes Water Quality Initiative
GSD	geometric standard deviation
HAPs	hazardous air pollutants
HBL	health-based level
HCs	hazardous concentrations
HCl	hydrogen chloride
HI	hazard index
HQ	hazard quotient
HWC	hazardous waste combustor
HWC-SERA	hazardous waste combustor screening ecological risk analysis
IEM	Indirect Exposure Emissions Model
IEM-2	Indirect Exposure Model (1993 addendum)
IEM-2M	Indirect Exposure Model (modified for mercury)
IEUBK	Integrated Exposure Uptake Biokinetic (model)
IMOE	incremental margin of exposure
INC	incinerators
ISCST3	Industrial Source Complex Model - Short Term, Version 3
LADD	lifetime average daily dose
LEL	lowest exposure level
LOAEL	lowest observed adverse effects level
LOEC	lowest observed effects concentration
LRS	lower respiratory symptoms
LWAK	lightweight aggregate kiln
MACT	maximum achievable control technology
MRADs	minor restricted activity days
MRTC	*Mercury Study Report to Congress*
NAAQS	National Ambient Air Quality Standards
NAWQC	National Ambient Water Quality Criteria
NCHS	National Center for Health Statistics
NEL	no effects level
NESHAP	National Emission Standards for Hazardous Air Pollutants

List of Acronyms (continued)

NFCS	National Food Consumption Survey
NHANES	National Health and Nutrition Examination Survey
NOAA	National Oceanic and Atmospheric Administration
NODA	notice of data availability
NOEC	no observed effects concentration
NSTP	National Status and Trends Program
OINC-L	on-site large incinerator
OINC-S	on-site small incinerator
OSW	Office of Solid Waste
PbB	blood lead
PCDD	polychlorinated dibenzo(*p*)dioxins
PCDF	polychlorinated dibenzofurans
PDFs	probability density functions
PEL	probable effects level
PM	particulate matter
RADs	restricted activity days
RCRA	Resource Conservation and Recovery Act
RDA	recommended daily allowance
RF3	Reach File Version 3
RfC	reference concentration
RfD	reference dose
RRFs	resource recovery facilities
SAB	Science Advisory Board
SAV	secondary acute value
SCV	secondary chronic value
SCS	Soil Conservation Service
SMCV	species mean chronic value
TCDD	tetrachlorodibenzo(*p*)dioxin
TEFs	toxicity equivalents
TEL	threshold effects level
TEQ	toxicity equivalency quotient
TEqC	toxicity equivalency quotient concentration
TSS	total suspended solids
UF	uncertainty factors
UIR	unadjusted ingestion rates
URFs	unit risk factors
URS	upper respiratory symptoms

List of Acronyms (continued)

URTC	Utility Report to Congress
USDA	U.S. Department of Agriculture
USGS	U.S. Geological Survey
USLE	universal soil loss equation
VHG	air concentration of elemental mercury vapors
VHG2	air concentration of divalent mercury vapors
WDP	wet deposition of particles
WDV	wet deposition of vapors
WHB	waste heat boilers
WLDs	work loss days
WVHG	wet deposition of elemental mercury vapors
WVHG2	wet deposition of divalent mercury vapors

1.0 Introduction and Background

On April 19, 1996, the U.S. Environmental Protection Agency (EPA) proposed rules to revise standards for hazardous waste combustors, which include incinerators and hazardous waste-burning cement kilns and lightweight aggregate kilns. The rule was proposed under joint authority of the Clean Air Act (CAA), as amended, and the Resource Conservation and Recovery Act (RCRA), as amended. Hazardous waste combustors (HWC) emit hazardous air pollutants (HAPs) that are listed under Section 112(d) of the CAA. The EPA proposed National Emission Standards for Hazardous Air Pollutants (NESHAP) pursuant to Section 112(d) of the CAA that establish emission standards based on application of maximum achievable control technology (MACT). Hence, these standards are referred to as MACT standards.

These MACT standards are technology-based standards; they are not risk-based. These facilities, however, are also covered by RCRA in Sections 3004(a) and 3004(q), which require EPA to develop standards that are protective of human health and the environment. The risk analysis described in this report was conducted to satisfy RCRA's requirement in support of the MACT standard rulemaking for HWCs. EPA's express intent is to minimize duplication in regulations and regulatory actions. Accordingly, the MACT standards for incinerators, cement kilns, and lightweight aggregate kilns are being developed under CAA authority with due consideration of human health and ecological risks. Consideration of human heath and ecological risk at this time allows EPA to satisfy the requirements of both RCRA and CAA.

The risk assessment conducted for the final rule covers the same source categories evaluated in the April 19, 1996, proposed rules: incinerators, cement kilns, and lightweight aggregate kilns. For the final rule risk assessment, three subcategories were added for incinerators: commercial incinerators, on-site incinerators (small), and on-site incinerators (large). In addition, waste heat boilers, which are associated with some incinerators, were evaluated separately. Area sources, which under Section 112 of the CAA are defined as sources that emit less than 10 tons per year of any one HAP or less than 25 tons of any combination of HAPs, also were considered as a separate category.

The regulatory scenarios included in this background document include baseline and three MACT options. Baseline reflects emissions under current RCRA controls and covers the scenario in which no additional controls are promulgated. That is, it is the level of control that would be expected under existing RCRA regulation in the absence of MACT standards. The three regulatory scenarios evaluated include:

- MACT—Standard. This regulatory scenario represents emissions that are projected to occur under the final rule, as promulgated. It reflects a combination of "floor" and "beyond the floor" emissions controls.

- MACT—Floor. This regulatory scenario represents emissions that would be projected to occur under the minimum level of control that is permitted under Section 112(d) of the CAA.

- MACT—Beyond-the-Floor (ACI). This regulatory scenario represents emissions that would be projected to occur with more stringent controls for dioxins and mercury. It corresponds to a level of control that could be achieved using activated carbon injection (ACI).

Table 1-1 gives the emission standard levels that were used to project emissions for the floor, standard, and beyond-the-floor (ACI) MACT options described above. In a few instances, the MACT options for the final rule differ from the MACT options that were analyzed in the risk analysis. Therefore, Table 1-1 lists the level of the standard for each of the options for both the final rule and the risk analysis. For dioxins and low-volatility metals, there are no differences between the final rule and the risk analysis for any option, including the final MACT standards. For mercury, there are differences for the beyond-the-floor (ACI) option but not the floor or the final standards. For the remaining emission standards, there are also differences for the final MACT standards depending on the particular emission standard (semivolatile metals, total chlorine, or particulate matter) and source category to which the standard applies (cement kiln, incinerator, or lightweight aggregate kiln).

This risk analysis was a multimedia, multipathway assessment that addressed direct exposures to constituents released to the atmosphere by HWC units and indirect exposures due to movement of chemical constituents into the food chain. The risk assessment addressed both human health risks (cancer effects and noncancer effects) as well as ecotoxicological risks. Constituents assessed were 7 congeners of chlorinated dioxin and 10 congeners of chlorinated furan, 3 species of mercury, the 11 metals that were modeled for the proposed rule (antimony, chromium VI, chromium III, arsenic, lead, barium, nickel, beryllium, selenium, cadmium, silver, and thallium), three additional metals (cobalt, copper, and manganese), particulate matter, hydrochloric acid, and chlorine gas. To the maximum extent possible, this risk assessment followed the latest risk guidelines adopted by EPA and used the most recent data available.

The remainder of this document presents risk results and describes the risk assessment methodology.

- Section 2.0 presents the risk characterization. A complete set of risk results is presented in *Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes, Human Health and Ecological Risk Results, July 1999.*

- Section 3.0 provides an overview of the analytical approach used to evaluate risks from HWC. This section provides the reader with an understanding of the basic methodology and the steps used to calculate risk.

Table 1-1. Comparison of MACT Option Levels for Final Rule to Option Levels for Risk Analysis [a]

MACT Option	System Type	Dioxin TEQ[b] ng/dscm	Mercury µg/dscm		Semi-Volatile Metals[c] µg/dscm		Low-Volatility Metals[d] µg/dscm	Total Chlorine[e] ppmv		Particulate Matter gr/dscf [f]	
		Final Rule/Risk Analysis	Final Rule	Risk Analysis	Final Rule	Risk Analysis	Final Rule/Risk Analysis	Final Rule	Risk Analysis	Final Rule	Risk Analysis
Floor	CK	0.20 or 0.40 & 400 °F	120	120	650	650	56	130	130	**0.3** [g]	**0.030**
	INC	WHB: 12 & 400 °F Others: 0.20 or 0.40 & 400 °F	130	130	240	240	97	**77**	**80**	0.015	0.015
	LWAK	0.20 or 4.1 & 400 °F	47	47	1,700	1,700	110	1,500	1,500	0.025	0.025
Standard	CK	0.20 or 0.40 & 400 °F	120	120	240	240	56	130	130	**0.3** [g]	**0.03**
	INC	0.20 or 0.40 & 400 °F	130	130	240	240	97	**77**	**80**	0.015	0.015
	LWAK	0.20 or 0.40 & 400 °F	47	47	**250**	**240**	110	**230**	**150**	0.025	0.025
Beyond the Floor (ACI)	CK	0.20	25	25	240	240	56	130	130	**0.3** [g]	**0.03**
	INC	0.20	**20**	**10**	240	240	97	**77**	**80**	0.015	0.015
	LWAK	0.20	10	10	**250**	240	110	**230**	**150**	0.025	0.025

[a] Differences between Final Rule and Risk Analysis options levels appear in bold. All concentrations are corrected to 7% O_2.
[b] 2,3,7,8-TCDD Toxicity Equivalence
[c] Cadmium and lead.
[d] Arsenic, beryllium, chromium.
[e] Hydrogen chloride and chlorine gas.
[f] CK options for final rule in kg PM/Mg dry raw feed.
[g] A level of 0.3 lbs PM/ton dry raw feed equates to a stack gas equivalent concentration of approximately 0.03 gr/dscf.

- Section 4.0 provides information on the universe of facilities evaluated and the sampling methods used to select facilities for detailed modeling. The section also explains the development of engineering data for the facilities modeled, including emissions, and describes the methods used to delineate the environmental setting and populations surrounding the facilities modeled.

- Section 5.0 explains the air dispersion and deposition modeling, calculation of environmental media concentrations, and food chain concentrations.

- Section 6.0 presents the exposure assessment methodology. The human receptors and exposure scenarios included in this analysis are described, and exposure parameters, exposure parameter variability, calculation methods used in making exposure estimates, and data on background exposures experienced by the general population for dioxins, mercury, and lead are presented.

- Section 7.0 presents the human health effects data used to establish human health benchmarks and the benchmark values used to characterize human health risk.

- Section 8.0 explains the methods used to characterize risks to human health, including a presentation of risk descriptors and methods used to analyze individual risk and population risk.

- Section 9.0 presents the entire ecological risk assessment methodology. This section describes the analytical framework, selection of indicator species, development of benchmarks, and supporting stressor-response profiles.

Supplementary information and data are provided in the appendixes to this report, which include:

- Appendix A: Expanded Discussion of Specific Statistical Topics Related to Sampling

- Appendix B: Facility-Specific Input Data

- Appendix C: Direct and Indirect Exposure Equations

- Appendix D: Parameter Derivation and Citations

- Appendix E: Particulate Matter (PM) Risk Assessment for the Proposed Combustor Emissions MACT Standard

- Appendix F: Derivation of Total Mercury Water Column Concentration, Cwt, as Used to Model the Drinking Water Ingestion Pathway

- Appendix G: IEM-2M Mercury Modeling Parameters

- Appendix H: Concentration of Selected Constituents in Modeled Media

- Appendix I: Exposure Parameter Variability Analysis
- Appendix J: Ecotoxicological Profile for Ecological Receptors

2.0 Risk Characterization

This section summarizes the risk results generated for the final rule for both the human health and ecological components of the analysis. In addition to summarizing the key findings for a particular category of risk, each subsection presents a brief overview of the methodology used to generate the risk results, including the sources of variability considered in their calculation. Each subsection also discusses the uncertainty associated with the risk results and presents additional information useful in interpreting the results. Human health risk results are presented in Section 2.1. Ecological risk results are presented in Section 2.2. The complete set of risk results for all human receptors and for the ecological risk analysis are presented in a separate, three-volume document, *Human Health and Ecological Risk Results: Baseline and MACT*.

2.1 Human Health Risk Characterization

The HWC risk analysis completed for the final rule characterized risk for those human receptors who may experience significant exposure to constituents released from HWC facilities because of their proximity to these facilities and/or their behavior.[1] In addition to characterizing risks to human receptors residing within the vicinity of HWC facilities (termed "local" receptors), the analysis also assessed annual cancer incidence in the general population resulting from the ingestion of agricultural commodities that are produced within the vicinity of HWC facilities but distributed nationally for consumption.

The local human receptors included in this assessment are divided into five categories for purposes of summarizing risk results:

- **Commercial farmers** who produce agricultural commodities and are assumed to ingest a portion of the commodities they produce

- **Residents** who are exposed to constituents through incidental soil ingestion, drinking water ingestion, and inhalation but are not assessed for agricultural commodity ingestion

[1] The HWC risk analysis assessed risks for a variety of different receptors located in the vicinity of HWC facilities. Although these receptors display a wide range of exposures and risks, the term "significant exposure" is considered appropriate here because certain receptors were deleted from this analysis based on results of the analysis performed at proposal that projected low risks (e.g., the commercial poultry farmer).

- **Home gardeners** who engage in home gardening and are exposed to constituents through the consumption of home-produced fruits/vegetables in addition to the routes mentioned above for residents

- **Recreational fishers** who engage in recreational fishing activity at waterbodies located within their particular study area

- **Subsistence receptors** who obtain a significant portion of their diet through the consumption of home-produced food items (i.e., fish for the subsistence fishers and a wide variety of food items including meats, fruits, vegetables, and dairy products for the subsistence farmers).

These five categories of receptors form the basis for that portion of the HWC risk analysis that assessed risk to human receptors residing within the vicinity of HWC facilities. A variety of cancer and noncancer effects were assessed for these five categories of receptors. Both individual and population-level risk results were used to characterize these effects.

Although risk results generated for these five categories of receptors capture a significant portion of the risk resulting from the deposition/concentration of key constituents within the vicinity of HWC facilities, one important component of that risk was not fully captured—cancer risk resulting from the consumption of agricultural commodities containing dioxins/furans that are raised within the vicinity of HWC facilities. Although the consumption of these commodities by the farmers that produce them was assessed, consumption of commercially produced commodities by the general population was not. Much of the agricultural commodity production is distributed to the general population, including individuals residing outside of study areas. The annual excess cancer incidence resulting from consumption of agricultural commodities containing dioxins/furans that were raised within study areas but distributed nationally captures this component of risk. In addition, it should be noted that the HWC risk analysis did not characterize risk associated with the transport of chemical constituents beyond study areas (i.e., beyond 20 km of HWC facilities).

The human health risk characterization methodology used in this analysis includes a number of components designed to generate risk estimates that are representative of the receptors being modeled. Important components include: (1) use of a 16-sector study area template to refine exposure assessment; (2) use of population density data for key receptors to reflect actual location and density of receptors relative to modeled HWC facilities in characterizing risk, (3) use of realistic exposure scenarios designed to reflect typical or realistic exposure for modeled receptors, and (4) inclusion of an exposure parameter variability analysis for key exposure pathways to reflect individual intake variability in characterizing risk. Despite these refinements in exposure assessment, both individual risk and population-level risk characterization are impacted by sources of uncertainty. Sources of uncertainty that impact specific risk categories are discussed in each subsection; however, there are several sources of uncertainty that potentially impact more than one category of risk results. To avoid repetition, these sources are discussed in the following section.

2.1.1 Uncertainties and Limitations

Finkel (1990) classified all uncertainty into four types—decision rule uncertainty, model uncertainty, parameter uncertainty**,** and variability. Variability is not synonymous with uncertainty, although failure to accurately reflect variability in an analysis can introduce uncertainty.

A national level risk assessment of this type is inherently subject to many sources of uncertainty because it uses a variety of different mathematical models, each of which require the aggregation of data from a wide variety of different sources. The modeling system is comprised of individual models that have been assembled in a feed-forward manner—that is, one model's output is the input for the next model. The data sources used to develop values for the model parameters come from a wide variety of databases and references. Some of these data were compiled by extracting and formatting national data (e.g., U.S. Census data) to fit model input specifications. The models used and the manner in which they are combined and configured introduces decision and modeling uncertainty. The data used for model input parameter values are often subject to both uncertainty and variability.

Decision rule uncertainty is primarily of concern to risk managers, although it can have a significant impact on risk results because it often affects the underlying framework of the analysis. This type of uncertainty can arise out of the need to balance different social concerns when determining an acceptable level of risk. Many of the fundamental decisions that are made during the planning phases of a risk analysis fall into the category of decision rule uncertainty. Examples of decision rule uncertainty in the HWC risk analysis are selections made regarding what to include in the analysis: combustors categories, chemical contaminants, the human receptor populations, and the study areas for which risks were characterized. The decision to focus the assessment on stack emissions from HWCs rather than other sources of risk is another example of decision rule uncertainty. Because decision rule uncertainty often involves key components of an analysis, this category of uncertainty can have a significant impact on the analysis results. Unfortunately, the nature of decision rule uncertainty can make it difficult to fully characterize its magnitude; that is, in order to characterize this category of uncertainty, an alternate framework for the analysis often needs to be implemented, which is not always feasible.

Model uncertainty is associated with all models used in all phases of risk assessment. These include the use of animal models to study human health effects, dose-response models to extrapolate to lower dose, as well as the computer models used to predict the fate and transport of chemicals in the environment and to estimate human exposures. Computer models are simplifications of reality, requiring exclusion of some variables that may influence predictions but cannot be included because of increased model complexity, a lack of data, or failure to have a full understanding of the underlying processes. Excluded variables may be important in some instances and not in others. A similar problem can occur when a model that is applicable under one set of conditions is extrapolated to a different set of conditions. Also, choosing the correct model form is often difficult because conflicting theories seem to explain a phenomenon equally well.

Parameter uncertainty occurs when parameters appearing in equations cannot be measured precisely and/or accurately either because of technical/equipment limitations or

because the quantity being measured varies spatially or temporally and data are insufficient to capture that variation. Parameter uncertainty is often introduced into an analysis when it is necessary to interpolate missing data using data from a surrogate source or when a lack of site-specific data requires the use of default or regional-level parameters to represent a factor that can display site-to-site variation. Parameter uncertainty can also result from errors introduced either during statistical sampling of data for parameter characterization or during subsequent statistical analysis of those data to derive representative parameters (e.g., means, medians, or variability distributions). Random, or sample errors, are a common source of parameter uncertainty that is especially critical for small sample sizes. More difficult to recognize are nonrandom or systematic errors that result from bias in sampling, experimental design, or choice of assumptions. Parameter uncertainty is especially important in risk assessment because the data are often derived from secondary sources of information and are used for other than their original intended purpose.

Variability is often used interchangeably with the term “uncertainty,” but this is not strictly correct. Variability is tied to variations in physical, chemical, and biological processes. This variability cannot be reduced with additional research or information, although it may be known with greater certainty (e.g., age distribution of a population may be known and represented by the mean age and its standard deviation). “Uncertainty” is a description of the imperfection in knowledge of the true value of a particular parameter or its real variability in an individual or a group. In general, uncertainty is reducible by additional information-gathering or analysis activities (better data, better models), whereas real variability will not change (although it may be more accurately known) as a result of better or more extensive measurements (Hattis and Burmaster, 1994). However, if variability is not fully characterized within a risk analysis, uncertainty can be introduced into the estimates that are generated. In other words, overall uncertainty in risk estimates can be reduced by more completely characterizing variability.

Many of the modifications that have been made to the HWC risk analysis methodology implemented for the final rule are designed to more fully represent variability associated with key risk-related factors such as:

- The variety of different facilities comprising each of the combustor categories (specifically their emission characteristics)
- Environmental settings associated with the facilities
- The location, density, and behavior of human populations potentially exposed to combustor emissions, including exposure factor variability.

Because variability is not in itself a source of uncertainty and because sources of variability are described elsewhere in the background document along with the description of the methodologies or parameters that they affect, the topic of variability will not be explicitly addressed in this section. Instead, the discussion will focus on the three identified sources of uncertainty: decision rule, model, and parameter uncertainty.

Given the complexity of the overall analysis, it was not feasible to conduct a full quantitative analysis of the impact for many of the key sources of uncertainty that fall into the

three categories of decision rule, model, and parameter uncertainty. Instead, a qualitative discussion of these uncertainties including, when possible, an assessment of the direction and magnitude of impact on risk results has been developed. Table 2-1 lists the major sources of model and parameter uncertainty impacting the HWC risk analysis. Sources of decision rule uncertainty, which primarily impact the risk assessment framework of the analysis, are not presented in the table and are instead discussed within the text.

The remainder of this section discusses the primary sources of uncertainty that impact each of the components of the risk analysis process: (1) risk assessment framework, (2) characterization of model facilities, (3) fate and transport modeling, (4) human exposure and risk methodology, and (5) human health effects. The discussion within each of these subsections focuses on those sources of uncertainty that impact a broad range of risk results. Sources of uncertainty that impact specific categories of risk are described separately in the section of this document where those risk results are presented. Because of the emphasis placed on the characterization of risk associated with mercury exposure, uncertainty associated with the fate/transport of speciated mercury is presented as a separate discussion within the fate and transport modeling subsection. Uncertainty associated with the ecological risk component of the analysis is discussed separately along with the ecological risk results in Section 2.2.1, although it should be noted that many of the uncertainties discussed in Sections 2.1.1.1 through 2.1.1.3 (e.g., site characterization and fate/transport modeling) also impact the ecological risk results.

2.1.1.1 <u>Risk Assessment Framework</u>. This section describes uncertainty associated with the underlying framework and design of the HWC risk analysis.

Constituents Selected for Analysis. The selection of constituents to be included in the analysis represents an important source of decision rule uncertainty because it has a fundamental effect on the risk estimates generated for the analysis. The constituents modeled for the analysis include:

- 2,3,7,8- Chlorine-substituted dioxin and furan congeners
- Mercury (including methyl, divalent, and elemental)
- 14 additional metals
- PM (including PM_{10} and $PM_{2.5}$)
- Hydrochloric acid and chlorine gas.

See Section 3.1.2 for a full listing of modeled constituents. The HWC risk analysis included all constituents for which there was sufficient facility-specific emissions data to support risk modeling. Consequently, nondioxin products of incomplete combustion (PICs) were not included in the analysis because of insufficient facility-specific emissions data. Some PICs are highly lipophilic and may bioaccumulate in the food chain and cause exposures through the consumption of contaminated food. The effect of excluding these constituents from the analysis is to potentially underestimate the risks from HWC emissions.

Receptor Populations Evaluated. The decision was made that the HWC risk analysis should address risks to the entire exposed population within study areas, i.e., within a 20-km radius. This includes individuals who, due to their activities, could be at increased risk (e.g., farmers, fishers, and home gardeners) as well as individuals who could be exposed simply by

Table 2-1. Major Sources of Model and Parameter Uncertainty Impacting the HWC Risk Analysis[a]

Component of the risk analysis process	Model Uncertainty	Parameter Uncertainty
Characterization of Model Facilities		
Emissions characterization		Use of compliance and test report data, use of only the most recent data, assumption of continuous operation, imputation of missing data, treatment of nondetects Mercury—incomplete data characterizing speciation of Hg emissions
Site characterization	Nonrandom selection of waterbodies for modeling (waterbodies selected to favor more heavily impacted areas, drinking water sources, and potential recreational activity), limiting watershed delineation to 20 km	Use of default value for soil erodibility, use of quasi-site-specific LS values, currency of GIRAS remote sensing data used to characterize cover, management and supporting practice factors Use of default values for agricultural parameters related to crops and animal feeding practices Human and livestock population location/density - currency of U.S. Census and Census of Agriculture data, spatial resolution of data
Fate and Transport Modeling		
Air dispersion/ deposition modeling	Air dispersion assumed to be Gaussian, use of winds at stack top, simplified treatment of complex terrain All study areas treated as rural locations, although some are urban Limiting receptor distances to 20 km. Mercury—failure to consider long-range transport of Hg beyond HWC study areas Incomplete characterization of building wake effects and elevated terrain. The ISCST3 terrain grid pathway not used for deposition estimates Dry deposition of vapors and gases evaluated outside of ISCST3 (mass balance issue)	Meteorological data obtained from met stations, which can be distant from HWC facilities Distribution of dioxins/furans between particle and vapor phases Particle size distributions defined at combustor-category level (instead of facility-level) Wet scavenging rate of small particles used for vapors Assumed dry deposition velocities for dioxin/furan vapors Mercury—incomplete data on wet and dry removal rates

(continued)

Table 2-1. (continued)

Component of the risk analysis process	Model Uncertainty	Parameter Uncertainty
Waterbody modeling	Use of a steady-state assumption in the IEM2 waterbody model for all waterbodies Use of loading from IEM2 watershed model for year 30 of operational period Uncertainties attendant to use of universal soil loss equation (USLE) and sediment delivery ratio to estimate waterbody loadings Spatial and temporal effects not considered Failure to consider chemical and solids loading beyond the study area boundary Mercury–IEM2M model less reliable for unsteady environments such as streams	Use of hydrologic regional average TSS values in waterbody modeling Use of default values for organic carbon content of soils, sediments, and suspended solids Use of biota-sediment accumulation factors for dioxins/furans based on pooled, non-site-specific data Mercury—use of assumed values for methylation rates in flowing waters, use of median bioaccumulation factors for trophic level 3 and 4 fish.
Farm food-chain modeling	Use of biotransfer factors to characterize uptake, distribution, and accumulation in animal tissues Hog farms modeled as outdoor operations rather than automated indoor operations (affects soil ingestion rates for hogs)	Air-to-plant uptake factors are not plant-species-specific, values inferred from empirical data Use of default values for plant deposition parameters Biotransfer factors for beef based on lipid-adjusted biotransfer factors for milk Biotransfer factors for beef used to model pork
Human Exposure and Risk Characterization		
Exposure assessment	Assumption that all commercial farmers and home gardeners consume home-produced foods Assumption that commercial farmers consume only one type of home-produced agricultural commodity Inability to characterize recreational fishing activity at specific waterbodies	Regional differences in activity patterns not considered Child exposure durations not differentiated as to farm/nonfarm status Currency of NFCS dietary intake rate data, extrapolation/adjustment of home-consumption data, small sample sizes Variability of background exposures to lead not fully characterized

(continued)

Table 2-1. (continued)

Component of the risk analysis process	Model Uncertainty	Parameter Uncertainty
Exposure assessment (continued)	Inability to identify location and activity patterns for subsistence farmers and fishers Use of mean exposure factors for estimating risks, except for risk-driving pathways (see below) Use of blood lead from IEUBK model for year 5 to represent 0-5 year olds Mercury—background exposures not considered	Background exposures to dioxins/furans not age-specific, except for infants
Risk characterization	Use of toxicity equivalence approach for assessing risks from dioxins/furans Use of no adverse effects levels for assessing noncancer risks Assumption of response additivity for estimating route-specific cancer and noncancer risks from exposures to chemical mixtures Use of discrete approximation approach in reflecting range of exposure/risk at the sector level (failure to fully capture upper end risk) Inability to quantify individual risks beyond certain percentiles, e.g., 99th percentile	Use of nondevelopmental toxicity benchmarks for assessing risks in children
Human Health Effects (dose-response assessment)		
Cancer	Nonthreshold assumptions and extrapolation steps used in deriving cancer slope factors from animal and human data	Estimation of human equivalent dose from animal studies Use of the 95% upper confidence limit on the slope of the dose response curve

(continued)

Table 2-1. (continued)

Component of the risk analysis process	Model Uncertainty	Parameter Uncertainty
Noncancer	Extrapolation steps used in deriving RfDs and RfCs from animal toxicity and epidemiological study data	Estimation of human equivalent dose from animal studies Use of the lower 95% confidence limit on the ED_{10} for estimating no-effects levels

[a] This table focuses on those sources of uncertainty that are specific to the HWC risk analysis and result from the analytical approach developed specifically for this analysis; those sources of uncertainty that are commonly encountered in conducting risk analysis are not generally included but are mentioned in the text. This table also does not list sources of decision rule uncertainty; these are discussed in the text.

virtue of their residing within study areas (i.e., nonfarm residents).[2] Although Census data were used to determine the number and location of exposed individuals, there is some uncertainty about the number and location of persons who fish recreationally or who raise certain types of livestock or who have backyard gardens. Moreover, Census data provide no information on the prevalence of subsistence activities, such as subsistence farming and subsistence fishing, that could lead to substantially higher exposures and, therefore, higher risks.

Stratified Random Sampling of Facilities. An important issue often raised with regard to national level risk analysis is the degree to which risk estimates are representative of the industrial category they are designed to represent. The overall representativeness of a national level risk assessment is heavily dependent on the method used to select facilities from the universe for risk characterization. The use of stratified random sampling in selecting facilities represents a significant improvement over nonrandom sampling (i.e., the use of model facilities) because it allows clear statistical statements to be made regarding the representativeness of risk estimates. Consequently, stratified random sampling was chosen as the means for selecting facilities for the final rule (see Section 4.1.4 for additional detail on facility sampling). Although this method is favored for facility sampling, there is decision rule uncertainty associated with its use.

The primary criteria used to guide the stratified sampling used in the HWC risk analysis was that there be at least a 90 percent probability of having sampled at least one “high-risk” facility from each of the main combustor categories evaluated in the analysis. If, for a given combustor category, sampling did not include a high-risk facility (a 10 percent probability for each category), then the risk distribution generated for that combustor category might not

[2] An exception was made for poultry farmers for which the proposed rule risk assessment indicated were not at increased risk relative to other residents. This is because poultry that are produced commercially are raised indoors, are fed grains that are generally produced elsewhere, and are physically protected from uptake from air. Therefore, they are not exposed significantly to HWC emissions and were not included in the risk analysis for the final rule.

accurately reflect the range of risk to individuals residing in the vicinity of facilities constituting that category. Specifically, the upper end of the risk distribution for that category could be underrepresented. The magnitude of the underrepresentation would depend on the extent to which high-risk facilities differ from non-high-risk facilities with regard to both risk and population density (i.e., if high-risk facilities were not sampled and they had significantly higher risks and population densities than non-high-risk facilities, then the upper end of the risk distribution could be underrepresented). It is also possible that the random sampling of facilities for a given combustor category resulted in more than one high-risk facility being sampled. If this were to happen, then the risk distribution generated for that category could have over-representation in the upper tail that could result in overly conservative predictions of both upper-bound and possibly central tendency risk, depending on the size of the combustor category. It is important to note, however, that the use of stratified random sampling significantly reduces uncertainty in the overall analysis compared to the use of nonrandomly selected facilities and makes it possible to quantify the uncertainty associated with sample size (i.e., sampling error).

The majority of human health risk results generated for the final rule include confidence intervals that reflect uncertainty associated with sampling error.[3] Sampling error refers to the error introduced into the risk estimates generated for a given combustor category by not having modeled all of the facilities in that specific combustor category. Consequently, for a given risk percentile, the confidence interval identifies the range of potential risk values within which a risk estimate based on modeling of all sites would be located. The confidence intervals do not reflect other sources of uncertainty, which, in many cases, are expected to have a greater impact on risk results.

Use of 20-km Radius in Defining Study Areas. The HWC risk analysis study area is defined as a 20-km radius surrounding each modeled facility. Consequently, risks resulting from the transport of emissions farther than 20 km from a given facility were not considered in the analysis. This source of decision rule uncertainty suggests that the risk to individuals who come into direct contact with media impacted by emissions transported beyond the defined study areas (e.g., the inhalation of ambient air) may be underestimated.

This uncertainty is expected to have the greatest impact on those individuals who ingest agricultural commodities raised on farms located beyond the study areas or ingest recreationally caught fish from waterbodies located outside of the study areas. As chemicals are transported farther from the point of release, air dispersion can reduce their ambient concentrations, resulting in reduced risk from direct inhalation. However, if those chemicals have the potential to bioaccumulate, then even relatively low ambient air concentrations (and resulting vapor/ particulate deposition rates) can result in chemical levels within agricultural commodities or recreationally caught fish that pose a potential health concern. Consequently, the decision to use a 20-km-radius study area is expected to introduce the greatest uncertainty into risk estimates generated for dioxin/furans and mercury because these constituents have the potential to bioaccumulate up the food chain. Specifically, it is possible that population-level risk estimates generated for the commercial farmers (dioxin-TEQ) and recreational fishers (methylmercury) as

[3] Those categories of risk results for which confidence intervals were not developed include: (1) both individual- and population-level blood lead (PbB) results, (2) semiquantitative population risk statements for the recreational fisher, and (3) avoided incidence for particulate matter.

well as the consumption of agricultural commodities containing dioxin-TEQ by the general public may have been underpredicted.

The decision to use a 20-km study area also leads to a possible underestimation of risks within study areas. This results from limiting chemical loading to surface waters from soil erosion and runoff to that portion of the watershed that lies within 20 kilometers. The result of this approach is that modeled waterbody concentrations may be underestimated because loading that occurs outside of the study area is not considered and the flow rates (and consequently dilution) that are used in predicting waterbody concentrations reflect volume contributions from the entire watershed including the portion located beyond the study area. This results in an underestimation of media and food chain concentrations from the affected bodies of water. Also, failing to account for the solids loading to surface water from the watershed beyond 20 kilometers creates uncertainty in the partitioning of contaminants between the sorbed and dissolved phases and between the sediments and water column. In particular, individual risks to recreational fishers from methylmercury may have been underestimated for these bodies of water.

Restriction of the particulate matter (PM) analysis to within 20 km of HWC facilities may have also underestimated risk because fine particles are transported over long distances. In general, fine particles are considered a regional, rather than a local, air pollution problem. Other factors, however, could lead to an overstatement of risk (e.g., assumption of no threshold for PM health effects modeling) and the net effect of these and long-range transport is not known (see Section 2.1.4.3).

Use of 16-Sector Template to Provide Spatial Resolution. The HWC risk analysis used a 16-sector template to apportion both modeled media chemical concentrations and human receptor populations for purposes of predicting human exposure (see Section 4.3.1 for additional detail on the 16-sector study area template). Although use of the 16-sector template does provide significant spatial resolution in characterizing human exposure to chemicals within various media, its use represents a source of model uncertainty that can impact the overall analysis. Specifically, use of the 16-sector template as the spatial apportionment grid for the analysis, instead of a more refined grid, could result in failure to represent individuals residing in areas that have locally elevated chemical concentrations (i.e., hot spots). The use of a 16-sector grid could result in averaging locally elevated media concentrations (hot spots) to generate sector average media concentrations, which form the basis for risk estimates. The accurate characterization of hot spots within the study areas could affect upper-bound risk estimates generated for the analysis because, potentially, some individuals would be found to reside within the hot spot, thereby experiencing elevated exposure and risk. By including risk estimates generated specifically for these more highly exposed individuals, the upper tail of the risk distribution could be extended, which could result in higher risk estimates for the upper-bound risk percentiles (e.g., 90th, 95th, and 99th percentiles).

Incremental Facility Risk Characterization. The HWC risk analysis was primarily designed to characterize incremental risk from individual HWC facilities because this is the most effective means of assessing the overall benefits associated with emissions reductions for HWC facilities. Uncertainty is introduced by not considering background concentrations or aggregate impacts of other anthropogenic sources. The HWC risk analysis focused on the incremental risk

from routine stack emissions because these are the emissions that are covered by the rule. However, there are other sources of emissions from HWC facilities such as fugitive emissions from waste handling operations and emissions associated with startup, shutdown, or other operating periods during which emissions differ from those during routine operations. Risks associated with nonroutine and fugitive emissions are not assessed.

Individual Facility Risk Characterization. The risk analysis completed for the final rule does not evaluate the aggregate impact from multiple HWC facilities to individuals residing within areas impacted by two or more HWC facilities. Instead, the risk analysis evaluates the impact of each facility's emissions separately. Characterizing risk from individual HWC facilities has its limitations because approximately 15 percent of the individuals residing within HWC study areas are impacted by more than one HWC facility. Failure to model risk for these overlapping populations introduces decision rule uncertainty into the overall analysis. Specifically, this approach results in "double counting" of exposed individuals (i.e., the same overlap population is assessed separately for each facility) and a potential underprediction of individual risk for individuals in overlap areas because risk resulting from the aggregate impact of multiple facility emissions on the same group of individuals is not considered.

2.1.1.2 <u>Characterization of Model Facilities and Environmental Settings</u>. This section describes the uncertainties associated with defining the HWC universe, characterizing emissions for the subset of HWC facilities that were modeled, and characterizing the environmental settings surrounding those facilities.

Defining the HWC Facility Universe. The HWC facility universe that forms the basis for this analysis is intended to reflect industry conditions at the end of 1997. Toward that end, the facility universe defined at proposal was updated to reflect new information on facility closures and entrants to the market. In addition, in the fall of 1997, site visits were made to state environmental and EPA Regional offices to identify additional information that could be used to update the facility universe (see Section 4.1.1 for additional detail on facility definition). Despite these efforts, there is the potential that the universe, as defined, does not fully reflect the status of all HWC facilities as of 1997. Although the potential for misrepresenting the status of a facility is considered relatively low, it does exist, and, consequently, uncertainty is introduced into the analysis. Another source of potentially significant uncertainty is the assignment of HWCs to the categories of interest for the risk analysis. Specifically, difficulties were encountered in classifying HWC facilities as area sources and in identifying facilities that have combustion units that incorporate waste heat recovery boilers (WHBs) in their design. Area sources are facilities with total emissions of hazardous air pollutants that meet certain criteria. However, facility-wide emissions were not available to ascertain whether the criteria were met.[4] Also, information on the existence of waste heat recovery boilers was not available for many HWC facilities.[5]

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[4] This problem was addressed by assigning only commercial and U.S. Department of Defense incinerators that met the criteria to the area source category. Therefore, on-site incinerators that may have met the area source criteria were not considered in the HWC risk analysis.

[5] This problem was addressed by using information on the prevalence of waste heat recovery boilers among combustion units with emissions test measurement data to impute the existence of waste heat recovery boilers among combustion units without test data. The magnitude of the impact on risk results from having misrepresented the status of a facility would depend on the combustor category affected and whether that facility was a high-risk facility

30-Year Modeled HWC Facility Lifetime. Hazardous waste facilities were assumed to operate for 30 years for purposes of evaluating the fate and transport of chemicals including accumulation in environmental media (see Section 5.3.2.1.3). The decision to use a 30-year operational lifetime for the HWC risk analysis introduces decision rule uncertainty that can impact a number of different scenarios. To the extent that such facilities operate longer than 30 years, higher levels of contaminants could accumulate in soils (and surface waters) leading to higher risks. Conversely, if facilities operate for less than 30 years, lower levels of contaminants could accumulate in soils and surface waters, leading to lower risks. This assumption should have relatively little effect on exposure pathways that are driven primarily by air concentrations, including consumption of forage by grazing animals such as beef and dairy cattle, and relatively greater effect on exposure pathways that are driven by soil concentrations, including transport of eroded soils to surface waters and bioaccumulation in fish. This could impact risks from dioxins/furans and mercury associated with consumption of fish because these contaminants can accumulate in soils for long periods of time.

Emissions Characterization. Emissions concentration and flow rate data for the HWC facilities were obtained from trial burn and certificate of compliance test reports (see Section 4.2 for additional detail on emissions characterization). The purpose of trial burns and compliance tests is to establish the operating envelopes for HWCs and to certify that permitted emissions levels are not exceeded when operating within that envelope. In order to establish the widest possible operating envelope, facilities tend to operate under conditions that may result in higher emissions than would occur during normal operations. For example, some facilities may choose to spike certain metals during the tests. At the same time, test conditions may not reflect conditions that occur during upset conditions that could result in emissions that are higher than under normal operating conditions.

The HWC risk analysis uses an overall particle size distribution generated for each of the combustor categories rather than facility-specific size distributions generating PM^{10} and $PM^{2.5}$ emissions estimates for individual facilities (a lack of facility-specific data prevented the development of facility-specific particle size distributions). PM^{10} and $PM^{2.5}$ emissions estimates are generated by combining these particle size distributions with facility-specific PM emissions data. The use of particle size distributions defined at the combustor category level rather than the facility level introduces parameter uncertainty into the analysis.

HWC facilities were assumed to be operating continuously for 24 h/d, 365 d/y for purposes of calculating annual stack emissions. Therefore, annual emissions rates may not be representative of actual facility operations with regard to temporal fluctuation in emissions and actual hours of operation. The significance of parameter uncertainty introduced through these operating assumptions is not known.

Emissions that result from materials handling, fugitive releases, emergency safety valve releases, and disruptions in the normal combustion operation, including startups and shutdowns, were not considered in characterizing facility emissions. It is important to note, therefore, that

(high-risk facilities have the potential to have a disproportionate effect on the upper end of the risk distribution).

the emissions estimates used in the final rule risk analysis may not be representative of all operating scenarios. For this reason, they may not capture all of the risk associated with a given facility (especially risk to receptors located proximate to the facility, where such emissions are more likely to have an impact). Any such ancillary risks, however, are not affected by the HWC rule because the rule applies only to stack emissions and the combustion unit is actually burning hazardous waste. Therefore, the risk analysis focused on risks associated with routine stack emissions rather than on short-term emissions that may occur under other conditions or emissions from other sources.

Complete emissions data were not available for all of the facilities evaluated in the HWC risk analysis and an imputation procedure was used to fill in missing data (for additional detail on the imputation procedure, see U.S. EPA, 1999). Efforts were made to impute data for a given facility from a set of facilities with characteristics similar to that facility. Despite efforts to match facilities for purposes of imputation, parameter uncertainty was introduced into the analysis by using imputed data to characterize emissions from HWC facilities.

Selection of Modeled Waterbodies. From zero to four waterbodies were selected for each study area to characterize risk related to aquatic media (e.g., recreational fish ingestion, drinking water ingestion, and impacts to ecological receptors) (see Section 4.3.2.1). The selection of these modeled waterbodies was not random; instead, it was conducted to provide coverage for those waterbodies that are (1) likely to be more heavily impacted by facility emissions (i.e., those located relatively close and/or downwind from the facility), (2) identified as drinking water sources, and (3) likely to experience recreational activity. This selection strategy was used to ensure that key factors related to human health risk (i.e., the three criteria listed above) were reflected in the waterbodies that were selected. The use of a nonrandomized sampling strategy, however, introduces decision rule uncertainty into the HWC risk analysis since it is not possible to know how representative the modeled waterbodies are of those waterbodies in study areas surrounding HWC facilities. The potential exists for the waterbodies that were selected to produce overestimates of risk for aquatic scenarios because the selection strategy partially favored waterbodies located in more heavily impacted areas. However, the two additional criteria (of drinking water source status and recreational potential) could have resulted in the selection of waterbodies that were located in areas less impacted by facility emissions. To fully characterize this source of decision rule uncertainty, a randomized selection strategy for waterbodies would need to be conducted and the resulting risk for aquatic scenarios compared to the risk generated using the current methodology. Even a randomized sampling strategy, however, would not address significant uncertainties related to human contact with aquatic media. This is discussed in the following section.

Terrain Elevation. Terrain elevation around HWC facilities can influence air dispersion in that elevated terrain features generally result in higher ambient air concentrations. In the analysis for the final rule, terrain was considered only if terrain elevations in the proximity of the facility were higher than stack height. In all other cases, terrain height was not evaluated and terrain was assumed to be flat. Flat terrain introduces uncertainty that generally results in an underestimation of ambient air concentrations and deposition but the magnitude of this uncertainty is not known.

Watershed Universal Soil Loss Equation (USLE) Parameters. A combination of watershed-averaged (i.e., site-specific) and national level parameters was used along with USLE to model soil erosion losses from watersheds to waterbodies (see Section 4.3.2.4 for additional detail on erosion modeling). Parameter uncertainty is associated with a number of these parameters. For example, surface thaw and snow melt are not reflected in the rainfall and runoff factors used in the analysis, which could result in underestimated erosion loads for facilities located in areas experiencing significant snow fall. A single soil type and related soil erodibility factor (K) were used in modeling erosion losses for all facilities, a simplification that is mitigated by the fact that soil erodibility is the least variable of the parameters in the universal soil loss equation.

In establishing topographic factors (LS) for watersheds, it was possible to obtain watershed-averaged (i.e., site-specific) slope (S) values, but it was not possible to obtain corresponding site-specific length (L) value. Therefore, an equation relating length to slope (based on a set of national default LS values) was used to estimate an L value for each modeled watershed that corresponds to the site-specific S value identified for that watershed. This approach results in an LS value for each watershed that reflects site characteristics yet is not purely site-specific and therefore introduces parameter uncertainty into the analysis.

Cover and management/supporting practice factors were characterized at the site-specific level using land use data obtained from GIRAS (Geographic Information Retrieval and Analysis System). As with all GIS-based land use data, the accuracy of the data is largely dependent on its currency (i.e., the age of the remote sensing imagery data that forms the basis for the land use coverages). Land use data based on older remote sensing images could misrepresent areas that have experienced significant growth, thereby introducing parameter uncertainty into the analysis (specifically affecting the cover and management/supporting practice factors). The different sources of parameter uncertainty associated with the USLE model that are described above have an aggregate impact on erosion estimates (actually all of the parameters are linearly related to erosion loss for a specific watershed). The relationship of these parameters to specific risk estimates, however, is far more complex and nonlinear. At this time, neither the direction nor magnitude of the impact on risk results has been quantified, although it is clear that the use of site-specific data in characterizing the majority of the USLE parameters significantly reduces uncertainty especially when compared to the use of default values.

Total Suspended Solids (TSS) Values for Modeled Waterbodies. Water column concentrations are intrinsically based on TSS values and, consequently, it is preferable to use waterbody-specific TSS values in conducting waterbody modeling. However, limitations in the STORET database, the source of TSS values for this analysis, prohibited the identification of waterbody-specific TSS values. Therefore, a regional approach was adopted in which a given facility was assigned to a hydrologic region and a typical (or central tendency) TSS value was identified for that region and assigned to the waterbodies modeled for that facility (see Section 4.3.2.5 for additional detail). Although use of regional level TSS values in predicting waterbody concentrations does represent a significant improvement over the use of default, or national level TSS values, it does introduce parameter uncertainty into the analysis since waterbody-specific TSS values were not used.

Use of U.S. Census Data and Census of Agriculture Data in Characterizing Human Population Location/Density. The primary uncertainty related to the use of U.S. Census data and Census of Agriculture data stems from the fact that these data represent different spatial scales. The Census data are at the block group level and the agriculture data are at the county level. Both sets of data are used for determining the number and location of persons living on farms of different types. (See Sections 4.4.1 and 4.4.2 for additional detail on derivation of human and livestock population estimates, respectively.) The block group Census data were used to determine the number and location of farm households and the Agricultural Census data were used to determine the kind of farming activity that is occurring at farm households. Using county-level Agricultural data to further disaggregate block group farm household data generally means that the same distribution of farm types applies to multiple block groups. This combining of data of different spatial resolution introduces uncertainty into the location of farm population estimates, but the magnitude of the uncertainty is not known. Also, these data fail to identify the number and location of persons engaged in subsistence farming or fishing.

2.1.1.3 Fate/Transport Modeling. This section describes the uncertainties associated with projecting the fate and transport of chemicals released from HWC facilities through all of the modeled media compartments.

Air Dispersion/Deposition Modeling. Air dispersion modeling completed for the HWC risk analysis using ISCST3 is subject to a number of sources of uncertainty (see Section 5.1 for additional detail on air dispersion and deposition modeling). As with many aspects of the HWC risk analysis, the use of a 20-km-radius in delineating study areas introduces decision rule uncertainty into the air dispersion and deposition modeling that has been conducted (see Section 2.1.1.1.)

Meteorological information used to support air modeling was generally obtained from locations well removed from modeled facilities and, therefore, may not be representative of conditions in the immediate vicinity of the stack. For example, localized channeling of winds can occur in areas with significant terrain features, resulting in higher concentrations than in areas of relatively flat terrain. The magnitude of this source of parameter uncertainty cannot be easily quantified, although it is expected that potential errors could result in a combination of over- and underpredictions of ambient air concentrations at specific locations. Consequently, the impact of uncertainty in meteorological parameters on central tendency risk results could be minimal, although they might have a more significant impact on upper-bound risk results.

Although 9 of the 76 modeled HWC facilities are located in urban areas, all facilities were modeled assuming rural locations. Consequently, the increased dispersion typically seen in urban areas, which can lead to increased ground-level concentrations at nearby receptors, was not reflected in the air dispersion modeling conducted for the subset of facilities located in urban areas. Modeling urban facilities in this manner is somewhat mitigated by the fact that, except for the analysis of particulate matter health effects, long-term averages (annual averages) were calculated for use in the risk analysis and air concentrations were averaged over each of 16 sectors.

Several limitations related to characterizing terrain near the point of release may have resulted in an underprediction of risk for receptors located near or adjacent to the stacks of

modeled facilities. These limitations include failure to: (1) fully consider building wake effects, (2) accurately classify sites as to elevated terrain, and (3) use the terrain grid pathway, all of which introduce model uncertainty into the analysis. With regard to building wake effect, limited information was available on the size of structures located near or adjacent to stacks at the modeled facilities. Building downwash, which can result from the presence of such structures, may significantly increase ground-level ambient air concentrations, particularly at locations that are relatively close to the point of release.

Site classification regarding simple versus complex terrain was conducted using criteria similar, but not identical to, those used within ISCST3. The criteria used in the HWC analysis could have resulted in sites that were classified as intermediate or complex based on ISCST3 criteria being modeled as simple terrain sites. If this misclassification occurred, then both ground-level concentrations and deposition rates could be underpredicted in the vicinity of the stack. Also, simple terrain was modeled as flat terrain, which could have resulted in under-prediction of ground-level concentrations in locations with significant terrain features below stack height.

Finally, the terrain grid pathway, which can enhance the modeling of air concentration/dry deposition modeling in elevated terrain, was not used in this analysis. Exclusion of the terrain grid pathway can result in an overestimation of impacts to receptors on the far side of intervening terrain and underpredicted impacts for receptors on the near side.

These three sources of model uncertainty will affect both central tendency and upper-bound risk estimates because they could affect a broad range of modeled facilities to varying degrees. They will tend to result in an underrepresentation of risk at the sector level. The overall magnitude of the uncertainty on risk results, however, is not known, although the effects are likely to be localized and to have limited impact on the HWC risk results due to spatial (16-sector) and temporal (long-term) averaging.

Wet scavenging of vapors (with the exception of mercury) was assumed to occur at the same rate as small particles; therefore, the analysis did not account for the dependence of wet scavenging on gas solubilities and vapor pressures. This could have affected the deposition of dioxin/furan congeners.

ISCST3 does not model the dry deposition of gases and vapors; consequently, these processes were evaluated outside of the ISCST3 model framework. Because they were modeled outside of ISCST3, it was not possible to deplete the plume to reflect these loss processes and, consequently, mass was not conserved within the model. This source of model uncertainty would tend to overestimate risk especially for sectors located farther from the facility because overall plume depletion increases at greater distances from the point of release. However, the modeled dry deposition of vapors and gases is relatively small; thus, this source of uncertainty is not expected to influence modeling results significantly.

The partitioning of semivolatile dioxin and furan congeners between vapor and particulate phases following release from the stack has a direct impact on risk related to the consumption of agricultural commodities. This occurs because dioxin/furan congeners present in the vapor phase are predicted to be more readily absorbed into plants (including silage and

forage) than are congeners present in the particulate phase. The HWC risk analysis uses the Junge-Pankow equation to determine the distribution between vapor and particulate phases for each congener. Although separate values are generated for each congener, a site-specific approach was not taken to project the vapor/particulate phase distribution for each congener, which introduces parameter uncertainty into the analysis. Ambient conditions, including temperature, humidity, and particulate matter as well as the particle size distribution for stack emissions, all of which are facility-specific, can impact the extent to which dioxin/furan congeners are distributed between vapor and particulate phases. Although the overall magnitude of this uncertainty is not expected to be high compared with other uncertainties, it is noted here for completeness.

Farm Food Chain Modeling. There are a variety of uncertainties related to the modeling of food chain impacts from HWC emissions (see Section 5.4 for additional detail on calculation of food chain concentrations). Although a number of modeled chemicals have the potential to bioaccumulate up the food chain, the dioxin/furan congeners are of primary concern because they have high bioaccumulative potential within both plants and animals and because of their relatively high cancer potency. Food chain modeling requires a number of different modeling steps (e.g., projecting chemical concentrations in soil, projecting plant uptake, and estimating the bioconcentration of chemicals in livestock following grazing), all of which are subject to both model uncertainty and parameter uncertainty.

The modeling of chemical uptake into plants involves a number of different mechanisms, each of which is subject to parameter uncertainty. For example, a factor used in the analysis to project air-to-plant (Bv) biotransfer, while chemical-specific, is not further differentiated as to plant type. However, Bv can vary for different plant species, reflecting the importance of physical attributes (e.g., exposed surface area and the structure of exposed membranes) in determining uptake. Failure to differentiate between different plant types introduces parameter uncertainty into the analysis.

The modeling of chemical bioconcentration in livestock is subject to several sources of parameter uncertainty and model uncertainty. The HWC risk analysis uses a biotransfer factor to model the complex and dynamic processes of absorption, distribution, metabolism, bioaccumulation, and excretion in farm animals. As such, the biotransfer factor is a great simplification of reality. Furthermore, the biotransfer factor presumes that bioaccumulation depends on the intake of a chemical substance by an animal without regard to its size or metabolic rate.[6] Thus, significant uncertainty is introduced when biotransfer factors for one animal are applied to another. In the HWC risk analysis, the dioxin/furan biotransfer factors for beef are derived from the biotransfer factors for dairy cow's milk by adjusting for the relative differences in lipid content of beef and milk. However, because beef cattle are smaller and eat less than dairy cattle, the resulting biotransfer factor predicts that beef cattle will have adipose tissue concentrations that are three times lower than for dairy cows (even though dairy cows excrete significant amounts of dioxins in their milk). Also, because the same biotransfer factors that are used for beef are used for pork, the biotransfer factor for pork predicts that hogs will

[6] This is in contrast to a bioaccumulation factor approach in which the concentration of a chemical contaminant in animal tissues is related to the concentration in animal feed rather than the contaminant intake.

have adipose tissue concentrations that are half that for beef cattle and only one-fifth that for dairy cows. It is unclear whether such predictions are actually observed in farm animals.

It was also assumed in modeling chemical bioconcentration in pork that commercial hogs are raised outdoors. However, increasingly, hog farms are being automated, resulting in reduced incidental soil ingestion because the hogs are raised primarily indoors. The modeling of hog exposure assuming a significant amount of time outdoors (with associated soil ingestion), when the trend is toward indoor automated operations, introduces parameter uncertainty into the analysis—specifically in the incidental soil ingestion rates that are used. Regarding livestock modeling in general, the HWC risk analysis uses a single set of national level livestock exposure assumptions in modeling chemical bioconcentration in each of the livestock species modeled (e.g., a single set of assumptions concerning the distribution of the diet among silage, forage, and grain is used in modeling beef cattle exposure for all modeled facilities). However, in reality, regional differences may exist in the way beef cattle are raised including the distribution of diet among these different feed sources. Failure to reflect regional differences in modeling chemical bioconcentration in livestock introduces parameter uncertainty into the analysis because specific parameter values, such as the amount of feed that is consumed, may not fully reflect local practice.

BSAFs for Dioxin/Furan Congeners. The HWC risk analysis uses biota-sediment accumulation factors (BSAFs) to project the uptake of dioxin/furan congeners into fish. These BSAFs were derived from data collected by the Connecticut Department of Environmental Protection, including a combination of preoperational and operational facility data (see Section 5.4.1.6 for further detail on BSAF derivation). Using a combination of preoperational and operational facility data addresses concerns that have been raised concerning the use of data from locations that are no longer impacted by dioxin/furan emissions (e.g., BSAFs from Great Lakes studies where dioxin/furan impacts are largely historical). Studies conducted at locations experiencing ongoing dioxin/furan contamination have been shown to produce BSAFs that are as much as an order of magnitude higher than areas no longer being impacted. Because the HWC risk analysis is designed to characterize baseline risks for facilities that are currently operating, use of BSAFs based on data from waterbodies that are currently impacted is preferable to the use of BSAFs from waterbodies that are not currently impacted. Although the use of the Connecticut study data does address this issue of basing BSAFs on data from nonimpacted waterbodies, these BSAFs are still impacted by several sources of uncertainty. The underlying data sets collected for the Connecticut study are subject to statistical sampling error and analytical measurement error, both of which produce parameter uncertainty. For all congeners other than 2,3,7,8-TCDD, 2,3,7,8-TCDF, and 2,3,4,7,8-PCDF, only pooled data were available for derivation of BSAFs, which can introduce uncertainties where there are significant differences in fish and sediment concentrations across sampling locations. In addition, the BSAFs used in the HWC risk analysis were derived using data selected by a hierarchy of criteria that compared other congener-specific data sources to the data sets presented in the Connecticut study (this procedure also included removal of outliers). For example, for 1,2,3,4,7,8-HCDF and 1,2,3,7,8,9-HCDF, the mean (no median values were available) of the pooled data for preoperational conditions was substantially influenced by outlier values, resulting in fish tissue concentrations that were higher than those for operational data. For these congeners, only the operational data pool was used in developing the BSAF values. Even when such hierarchical decision structures are designed and implemented carefully, parameter uncertainty is introduced into the values that are ultimately generated.

Mercury Modeling. Mercury concentrations in the environment affect all receptor populations. Important among these are recreational fishers and subsistence fishers. The most important exposure pathway is the aquatic pathway (e.g., waterbody concentrations - fish tissue concentrations - human consumption of fish); however, other exposure pathways have been evaluated as well. These include drinking water consumption, terrestrial food chain pathways, and soil ingestion.

A number of uncertainties are introduced into the risk assessment by mercury modeling because of a lack of data or inability to capture real-world complexities in the model formulations (detailed discussions of mercury fate/transport modeling within different media are presented in Section 5). Because these uncertainties affect multiple receptor populations and pathways, they are discussed in this section. Exposure assumptions, however, are receptor-population-specific and are discussed in the individual sections that follow. Uncertainties in the application of mercury health benchmarks are discussed under the topic “Reference Doses” in this section.

Mercury was modeled based on facility-specific emission rates. The form of mercury emitted by a given facility is thought to be a determining factor in the fate and transport of mercury in the atmosphere. Only limited data are available on the form of mercury emitted from hazardous waste combustors; however, emissions measurement data that were available were used to estimate both divalent (both particle and vapor phase) and elemental mercury (vapor phase only) emission rates (see Section 4.2). In addition to uncertainties in the emission rates themselves, atmospheric dispersion and deposition modeling did not account for atmospheric processes that would alter the vapor/particle partitioning or the other transformations of the mercury species, which introduces uncertainties in mercury species air concentrations and deposition rates. There are other uncertainties related to deposition of mercury as well. These include the lack of direct measurements of wet and dry removal processes for divalent mercury (e.g., gas scavenging rates and gas deposition velocities) and the use of air model algorithms that are not fully mass conserving with respect to that portion of divalent mercury vapor that is dry deposited. For a more detailed discussion, see Section 5.1.

In addition, the mercury modeling did not consider long-range transport of mercury emissions, which can be especially important for certain mercury species, such as elemental mercury, which has a relatively long half-life in the atmosphere. Because the HWC risk analysis did not consider the potential for mercury released from HWC facilities to impact other HWC study areas via long-range transport or the potential for mercury released from other anthropogenic sources to impact HWC study areas, model uncertainty is introduced into the analysis. Although a quantitative uncertainty analysis has not been conducted to determine the impact of this source of model uncertainty on risk results, it is expected that long-range transport could have a significant impact since it is considered an important factor in determining the overall fate/transport of mercury following stack release. Failure to consider long-range transport of mercury could result in an underprediction of mercury exposures because mercury contributions to a given study area from other HWC facilities (as well as other emissions sources) following long-range transport are not considered.

The behavior of mercury species in the soil and water environments is complex. There are a variety of uncertainties related to the fate and transport of mercury in watershed soils and

surface water. Among these are uncertainties involving the transport of mercury deposited in upland areas of a watershed to surface water and transformation of mercury in soil and subsequent volatilization and release to the atmosphere. Also uncertain is the disposition of mercury in surface water, including methylation and demethylation processes, sequestering in the water column and sediments, and uptake in aquatic organisms. In particular, methylation rates are highly variable and depend on the characteristics of the particular waterbody. This is considered a key uncertainty in the mercury analysis. Modeling the aquatic food chain pathway was based on the mercury modeling techniques contained in the *Mercury Study Report to Congress*, or MRTC (U.S. EPA, 1997).[7]

Even though the MRTC (U.S. EPA, 1997) provides the best data and modeling techniques available for modeling mercury, many sources of uncertainty still exist. The MRTC modeling upon which the mercury modeling in this risk assessment is based was developed for lakes only. Many of the sources of uncertainty in this risk assessment stem from the fact that both flowing waterbodies and lakes are included and the mercury modeling techniques were modified to accommodate both types of waterbodies. Sources of uncertainty include the following:

- The model's calculations of average waterbody concentrations are less reliable for unsteady environments, such as streams, than for more steady environments such as lakes.

- Volatilization from flowing waterbodies is based on the same general principles as those for lakes and using these volatilization algorithms for flowing waterbodies introduces uncertainty into the modeling.

- Methylation rate for flowing waterbodies, in both the water column and benthic sediments, was assumed to be 10 percent of the value used for lakes because the more aerobic conditions expected in flowing waterbodies tend to result in lower methylation rates.

- Modeling abiotic solids dynamics in waterbodies introduces uncertainty because waterbody-specific data were not available for these terms. This uncertainty affects partitioning of mercury in the water column and the balance between dissolution and sorption to suspended solids, as well as the mercury mass balance between the water column and sediments that occurs via settling of suspended solids or mobilization of sediments into the water column.

- The simple linear BAF model relating methylmercury in fish to methylmercury in water masks a number of nonlinear processes leading to the formation of bioavailable methylmercury in the water.

For a more detailed discussion of these and other aspects of mercury modeling, see Section 5.3.3.2.

[7] Other pathways were modeled using other variants of IEM (see Appendix F).

2.1.1.4 <u>Human Exposure and Risk Methodology</u>. This section describes uncertainties associated with the exposure assessment and risk characterization components of the HWC risk analysis.

Human Exposure Parameters. Many if not all of the exposure parameters used to model chemical uptake by human receptors are subject to parameter uncertainty and variability. (Note: The issue of variability was discussed in the introduction to Section 2.1.1 and will not be addressed here; see Section 6.3.1 for additional detail on human exposure parameters.) The HWC risk analysis uses national level assumptions in modeling human exposure; regional differences in behavior and activity patterns are not reflected in the modeling. For example, a single rate for home gardening within residential populations is used to establish the number of home gardeners for the analysis. Because the rate of home gardening is likely to vary for different parts of the country depending on climate and demographic factors, parameter uncertainty is introduced into the analysis through the use of a single home gardening value.

A number of the exposure parameters are not specific to the particular receptor populations for which they are used. For example, no distinction is made between farm and nonfarm households in the consumption of home-produced fruits and vegetables. The same consumption rates are used for both produce farmers and home gardeners due to a lack of data to distinguish between them. Similarly, the exposure duration values established for the child age groups considered in the analysis are not differentiated as to farm versus nonfarm status, despite the fact that substantially different exposure duration values are clearly indicated for adult farmers versus nonfarming adults. The child exposure durations could not be differentiated because of a lack of data. Although the overall impact on risk results stemming from the use of these undifferentiated values is not likely to be great, it does introduce parameter uncertainty into the analysis.

The majority of the data used to develop dietary intake rates for human receptors were obtained from the 1987/1988 National Food Consumption Survey (NFCS). A combination of household utilization and individual dietary intake data were used to estimate consumption rates of home-produced foods. However, the number of households that reported consuming home-produced foods for which data are available are relatively small; therefore, relatively small data sets are available for estimating intake of home-produced foods, especially for certain food items such as milk. Also, because this study is based on data collected more than a decade ago, parameter uncertainty is introduced into the analysis because dietary patterns may have changed in the intervening period. Furthermore, gaps in the NFCS data required interpolation between different age groups to generate a full set of intake rates for all receptors and all age groups modeled in the analysis. Although care was taken during interpolation to match age groups and compensate for sample size, parameter uncertainty is introduced into the analysis when intake rates for one age group are interpolated from other age groups. In a number of instances, information on age-specific dietary intakes in the general population were used for extrapolating age-specific consumption rates for households consuming home-produced foods and this may have introduced additional uncertainty into the exposure estimates.

Exposure Parameter Variability. The effect of exposure parameter variability was incorporated into the HWC risk analysis for selected chemical constituents and health effects for certain risk-driving pathways. In particular, variability in exposure duration and beef and milk

ingestion rates were considered in assessing cancer risks from exposures to dioxins/furans from consumption of home-produced beef and milk, and variability in fish ingestion rates was considered in assessing the potential for developmental and neurological effects of methyl-mercury exposures from consumption of recreationally caught fish. In addition, variability in blood lead was considered in assessing risks from exposure to lead (see discussion of blood lead modeling below). For all other chemical constituents and exposure pathways, mean exposure factors were used. Therefore, uncertainty is introduced by not having included the variability of exposure factors for these other constituents and pathways. The magnitude of the uncertainty depends on the relative magnitude of the many sources of variability considered in the analysis that affect the concentrations to which individuals are exposed and the magnitude of the variability of the exposure factors. Because exposure factor variability was included for the risk-driving pathways, the uncertainty that is introduced in the remaining risk estimates is not expected to be significant with respect to the primary findings and conclusions of the HWC risk assessment.

The exposure parameter variability analysis implemented in the HWC risk analysis for risk-driving pathways used both a probabilistic (Monte Carlo simulation) and discrete approximation approach to integrate exposure parameter variability into sector-level risk estimates (see Section 8.2.3 for additional detail on the exposure parameter variability analysis). A discrete approximation approach was used in a statistical analysis that generates risk estimates and confidence intervals for specific percentiles of the risk distribution for a given combustor category. This statistical analysis requires 20 discrete risk values and associated population weights, which are produced using the discrete approximation approach. Uncertainty is introduced by the use of 20 intervals to approximate the 20 discrete values to approximate variability at the subsector level. The impact of this source of uncertainty on the overall analysis is expected to be minimal; however, because an analysis of the degree of underprediction for upper percentiles using the discrete approximation showed minimal effect (especially for larger combustor categories that are less susceptible to outlying risk values).

Another source of uncertainty is the dietary recall data used to estimate the variability in the consumption of home-produced beef and milk. These data consist of a household component with data on food utilization over a 7-day period and an individual dietary recall component with data over a 3-day period. As such, the data reflect both short-term (day-to-day or week-to-week) variability and longer-term variability (month-to-month or year-to-year). However, the HWC risk assessment focused on chronic health effects and lifetime exposures; therefore, it is only the variability in long-term consumption rates between individuals and not short-term variability over time that is of interest. Therefore, the distributional data for home-produced foods may overstate the variability, particularly at the tails of the distribution for foods that exhibit significant short-term variability. However, this source of variability and, therefore, uncertainty was minimized in the probabilistic analysis by truncating the distributions (which were fitted to a log-normal distribution) beyond three geometric standard deviations from the mean.

Consumption of Home-Produced Foods by Commercial Farmers. It was assumed in this analysis that commercial farmers associated with HWC facilities located across the nation consume the same amounts of home-produced foods (i.e., the potential for regional variation in ingestion rates was not considered). This assumption may introduce uncertainty into the risk results generated for commercial farmers if home-produced food consumption among these

receptors displays patterns of regional variation. Moreover, all farm households were assumed to consume home-produced foods. Uncertainty introduced by these assumptions, however, was minimized because only those farms that have houses were considered in evaluating risks to commercial farmers (farms without residences were excluded).

Commercial Farmers Who Produce More Than One Agricultural Commodity. The U.S. Census block-group-level data and Census of Agriculture data used in this analysis do not allow for the enumeration of individuals engaging in more than one kind of agricultural activity. Consequently, separate risk estimates were generated for each of the four categories of commercial farmer receptor evaluated in the analysis, but risks representing overlap between these agricultural activities could not be estimated (i.e., the commercial farmer who raises beef and dairy cattle). If there are farmers who raise a variety of agricultural commodities, individual-level risk characterization for this receptor may underestimate risk (i.e., that farmer would be exposed to both home-produced beef and pork, for example, when the analysis generates separate individual-level risk estimates for each commodity). However, because the subsistence farmer receptor was assessed for simultaneous exposure to all modeled agricultural commodities, the risks assessed for the subsistence farmer can be used as an upper bound for all categories of farming activity including commercial activity involving multiple agricultural commodities (although subsistence farms were located without respect to the location of farms).

Recreational Fisher. Risk estimates generated for the recreational fisher are subject to both model uncertainty and parameter uncertainty. Analysis of recreational fisher exposure and risk is heavily dependent on accurately characterizing fishing activity at specific waterbodies because modeled chemical concentrations (specifically dioxins/furans and methylmercury) can differ significantly among the waterbodies located within a given study area (see Section 6.2.2 for additional detail on the recreational fisher). Given available data characterizing recreational fishing behavior and spatial modeling techniques, it was not possible to accurately predict the level of recreational fishing activity at specific waterbodies. Consequently, it was assumed that recreational fishing activity was equally distributed between waterbodies based on waterbody surface area. Moreover, recreational fishing activity was restricted to the specific bodies of water selected for modeling. To the extent that individuals fish at other bodies of water that are less affected by HWC emissions, exposures are likely to be overestimated. An inability to predict fishing activity at specific waterbodies introduces significant model uncertainty into the analysis. Parameter uncertainty is also associated with the recreational fish ingestion rates and the particular studies of fishers from which they were derived and from the imputation and averaging procedures used to fill in missing age groups.

Subsistence Scenarios. In the absence of site-specific information characterizing both the location and behavior of subsistence farmers and subsistence fishers, both of which can display regional variation, each receptor was modeled using a single scenario that was applied equally to all of the modeled study areas. For the subsistence farmer, it was assumed that a farm was located in each of the 16 sectors comprising each study area (i.e., farming activity was fairly evenly distributed across the study areas). For the subsistence fisher, it was assumed that fishing activity by a subsistence fisher occurred exclusively at a single modeled waterbody (i.e., separate risk estimates were generated for each modeled waterbody assuming fishing activity by an individual exclusively at that waterbody—see Section 6.2.3 for additional detail on the subsistence farmer and fisher). The use of a single standardized scenario to model subsistence

activity introduces both model and parameter uncertainty into the analysis. In reality, subsistence farming activity probably occurs at specific locations and is not distributed across the entire study area, while subsistence fishing activity probably includes a mix of activity distributed between waterbodies including those selected for modeling as well as those not selected.

Blood Lead Modeling. Modeled blood lead (PbB) levels were generated for the analysis using a combination of site-specific media concentrations (i.e., soil, drinking water, and ambient air) and dietary intake rate data obtained from the Indirect Exposure Model. These data were processed using the Integrated Exposure Uptake Biokinetic (IEUBK) model to generate sector-level PbB estimates for each modeled 0- to 5-year-old age group. There are several sources of uncertainty associated with the use of the IEUBK model as applied in the HWC risk analysis.

An age of 60 months was used in conducting the IEUBK modeling for the HWC risk analysis (i.e., the IEUBK model was configured to generate lead exposure estimates for a 5-year-old child). Although this approach is reasonable because the lead analysis focuses on the 0- to 5-year age group, some uncertainty is introduced into the PbB estimates by using a single age in conducting lead modeling. In reality, the 0- to 5-year-old age group within any given sector is comprised of a mix of children ranging in age from newborn to 5 years of age. Consequently, these children will display a range of intake rates and exposure durations reflecting their varying ages. Intake rates for most media and dietary items (on a milligram media per kilogram body weight basis) are higher for the first few years of life than for the 5th year of life. Consequently, the assumption of 5 years of age for all children in this age group may underestimate exposure levels for some of the younger children. The overall impact on modeled PbB levels resulting from the use of a single age for the 0- to 5-year-old age group will depend on a number of factors, including specific differences in exposure levels for different ages and key factors related to pharmacokinetic modeling for lead such as clearance rates and half-lives. A quantitative analysis of uncertainty associated with using a single age (i.e., 5 years) to characterize the child cohort evaluated in the lead analysis has not be conducted.

As with other modeling in this risk analysis, blood lead modeling focused on modeling incremental lead exposures resulting from HWC facility emissions. No indoor sources of lead (e.g., lead paint) were considered. However, indoor dust lead concentrations result primarily from two sources: outdoor soil containing lead that is tracked indoors and lead deposited from indoor air onto indoor surfaces. Following guidance provided in the IEUBK documentation, lead concentrations in indoor dust were assumed to equal modeled lead concentrations in outdoor soil. That is, deposition of lead from indoor air to indoor surfaces was not considered. There is, however, uncertainty associated with using this approach as the basis for modeling indoor dust exposure. The relative importance of lead deposition from indoor air to household surfaces as a contributor to indoor dust concentrations depends partially on the amount of outdoor soil that is tracked indoors. As the amount of outdoor soil that is brought indoors decreases, the importance of indoor lead deposition to total incremental exposure increases. Because the blood lead level modeling approach does not consider the contribution from indoor deposition, the modeling approach may not accurately represent those study areas where tracking of soil into homes is minimal and indoor deposition is the primary source of lead loading to indoor dust.

Following guidance from the IEUBK documentation, a bioavailability factor of 0.5 was applied to all dietary items considered in the HWC risk analysis in modeling lead exposure

resulting from ingestion of modeled agricultural commodities. Because different dietary items will display a range of bioavailability factors, the use of a single bioavailability factor for all dietary items introduces uncertainty into the PbB analysis.

The HWC risk analysis used a geometric standard deviation (GSD) of 1.6 obtained from the IEUBK documentation to represent interindividual variability in incremental PbB levels resulting from differences in pharmacokinetic, individual behavior, and site-specific factors related to lead exposure (e.g., the range of lead concentrations in soil that children in a given neighborhood are exposed to). The HWC risk analysis used the GSD of 1.6 to represent individual variability in PbB levels for all modeled study areas evaluated in the analysis. The use of the same GSD introduces uncertainty into the PbB analysis because site-specific differences in housing and the socioeconomic composition of the modeled population could result in different levels of interindividual variability for different study areas.

Characterization of Background Exposure. Although the HWC risk analysis focuses on risks from incremental exposures to HWC emissions, risks do not occur in isolation from other sources of exposure. Such exposures can be significant for a number of the chemical constituents assessed in the HWC analysis, including dioxins/furans, lead, and mercury. Such exposures may be especially significant in those instances for which a threshold level of effect exists for a given health effect.

Both the incremental margin of exposure (incremental MOE) analysis conducted for dioxin/furans and the lead analysis used background exposure data to evaluate the potential for adverse health effects (see Sections 6.5.1 and 6.5.2 for additional detail on the derivation of background levels used in evaluating lead exposure and dioxin incremental MOE). Uncertainty associated with the background exposure levels for dioxin-TEQ are described along with the individual incremental MOE results in Section 2.1.1.2; uncertainty associated with background exposure estimates for lead are discussed along with the individual lead results in Section 2.1.1.3. For mercury, no explicit analysis of background exposures was conducted, and, for this reason, the risks associated with HWC mercury emissions may be understated (although information is summarized in Section 6.6.2 on background exposures to mercury in the general population, which can be compared with exposures from HWCs).

Assumption of Additivity Between Chemicals in Characterizing Risk. Both cancer and noncancer risk was evaluated on a chemical-specific basis within the analysis. However, to characterize overall risk to specific receptors resulting from multiple chemical exposure, aggregated cancer and noncancer risk estimates were generated for both the inhalation and ingestion routes by assuming additivity between the different chemical-specific risks (e.g., a single ingestion hazard index is generated for the adult resident by summing the chemical-specific hazard quotients generated for the ingestion route). Whether or not a particular chemical mixture poses an additive risk depends on the targets (tissue, organ, or organ system) and the mechanisms of action of the individual chemicals. Because these factors were not evaluated for each chemical prior to assuming additivity, model uncertainty is introduced into the aggregate risk estimates generated for both the inhalation and ingestion routes. Because chemical mixtures can display both synergistic and antagonist behavior with regard to risk, it is not possible to state whether the assumption of additivity would tend to result in an over- or underprediction of aggregate risk. However, the primary risk driving pathways in the HWC risk analysis are

associated with a single chemical constituent (e.g., methyl mercury from ingestion of fish) or suite of constituents thought to act by a similar mechanism (e.g., dioxins/furans from beef and milk ingestion) and, therefore, significance of mixture effects are expected to be minimized.

Risk Characterization for Child Scenarios. To better characterize the range of risk to child receptors, the HWC risk analysis generated a separate set of risk results for three distinct child age groups (0 to 5, 6 to 11, and 12 to 19 years old). In generating these risk results, a separate set of exposure parameters was used for each age group to reflect age-dependent differences in behavior related to risk. Although this approach allows differentiation of exposure for the different age groups, for the majority of chemicals that were evaluated, it was not possible to differentiate between the age groups with regard to dose-response following exposure because age-differentiated toxicity factors were not available. Consequently, the majority of risk estimates for the three age groups were generated using toxicity benchmarks largely developed to model cancer and noncancer risk in adults. This concern may be of particular relevance to cancer risks that stem from exposures during childhood when an individual may be more susceptible to the effects of a cancer-causing agent.

The inability to characterize risk in children using age-group-differentiated toxicity factors introduces significant model uncertainty into these risk estimates. The model uncertainty associated with these child age group estimates is in addition to the uncertainty that already exists as a result of the various extrapolation steps and uncertainty factors used in generating the toxicity benchmarks themselves (e.g., extrapolation of animal study data to humans, high- to low-dose extrapolation, and duration of study adjustments to reflect lifetime exposure). However, no additional uncertainty factor has been applied to the toxicity benchmarks used in the HWC risk analysis.

It should be noted that the HWC risk analysis did evaluate the potential for adverse noncancer effects following exposure to lead and dioxins/furans using approaches designed specifically for the infant/child. Specifically, the lead analysis models blood lead levels for children ages 0 to 5 years using pharmacokinetic data and exposure parameters representative of that age group. In the case of noncancer risk for dioxins/furans, the HWC risk analysis uses an incremental MOE approach wherein infant exposure to dioxin-TEQ resulting from breastmilk ingestion is modeled and the resulting dose levels are compared to typical background levels. Although both the lead and MOE analyses are subject to model and parameter uncertainties, overall uncertainty associated with these analyses is reduced by using an assessment approach that is tailored to children and infants.

Noncancer Risk Characterization. The HWC risk analysis used the hazard quotient as a risk descriptor for assessing the potential for noncancer health effects. This risk descriptor is defined as the ratio of the estimated exposure (represented either as a dose for oral exposures or an air concentration for inhalation exposures) to a health benchmark such as the reference dose or reference concentration. However, the hazard quotient is a very imprecise measure of risk because the health benchmarks used to estimate it represent no adverse effects levels rather than adverse effects levels. A hazard quotient of unity (1) simply means that the estimated exposure does not exceed a level that is believed to be without appreciable risk. Although a hazard quotient that exceeds unity may indicate a potential for risk that increases with increasing exposures, any conclusion about the magnitude of the risk is subject to considerable uncertainty.

A somewhat different issue arises for dioxin/furans for which the toxicity data do not permit the development of a toxicity benchmark using EPA's reference dose methodology. Instead, the HWC risk analysis used a variant of the MOE approach in which the estimated exposures are compared directly to background exposures as a measure of risk. Background exposures for this suite of compounds are relatively high (within an order of magnitude or two) compared to levels that have been found to cause adverse effects in laboratory animals. Implicit in this approach is the presumption that background exposures are associated with de minimis risk and that an MOE that is small should be of inconsequential significance. However, because background exposures are not a toxicity benchmark, interpretation of the MOE is uncertain. This uncertainty is above and beyond the uncertainty related to characterization of background exposures used in the comparison.

2.1.1.5 <u>Human Health Effects</u>. This section describes uncertainties associated with the toxicity values used in relating dose estimates to resulting cancer and noncancer risk.

Cancer Slope Factors. Cancer slope factors (CSFs) were derived as the 95 percent lower confidence limit of the slope of the dose-response curve using a linear, no-threshold dose-response model. (Section 7.3 presents a review of toxicity factors and underlying data for all chemicals evaluated in the analysis.) The cancer slope factor is, therefore, an upper bound estimate of the cancer risk per unit dose and, for this reason, may overstate the magnitude of the risk. In addition, the use of CSFs in projecting excess individual cancer risk introduces uncertainty stemming from a number of factors including:

- Limited understanding of cancer biology
- Variability in the response of animal models
- Differential response in animal models versus humans
- Difference between animal dosing protocols and human exposure patterns.

A key step in CSF development is high- to low-dose extrapolation. Depending on the model used to fit the data, extrapolations to the low dose range can vary by several orders of magnitude, reflecting the potential uncertainty associated with the cancer slope factor. There are uncertainties involving the carcinogenicity of TCDD that require special attention. TCDD carcinogenicity is known to involve the Ah receptor, which involves tumor promotion, not initiation, and is a nongenotoxic response. TCDD is associated with carcinogenicity at multiple sites and the evaluation of TCDD has frequently involved mortality from all cancers combined. Mechanisms of carcinogenicity are not clearly understood beyond the involvement of the Ah receptor. Due to the nongenotoxic nature of TCDD carcinogenicity and the lack of cancer mechanisms, low-dose linearity is not a given. There may be a threshold, although at very low levels (i.e., nonlinearity might exist if dose-response below a certain point incurred zero additional risk); therefore, extrapolation of response into the low dose range has inherent uncertainties.

Reference Doses and Reference Concentrations. Uncertainty and variability in the toxicological and epidemiological data from which reference doses (RfDs) and reference

concentrations (RfCs) are derived are accounted for by applying uncertainty factors. An RfD (or RfC) 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 effects during a lifetime" (U.S. EPA, 1998b). RfDs and RfCs are based on the no adverse effects level (NOAEL) or lowest observed adverse effects level (LOAEL) for the most sensitive effect in the most sensitive or most relevant species. A series of standard uncertainty factors are applied to the NOAEL or LOAEL to derive the RfD or RfC. The following uncertainty factors account for areas of scientific uncertainty:

- Intraspecies variation, accounts for variation in sensitivity among humans (including sensitive individuals such as children, the elderly, or asthmatics)
- Interspecies variation, accounts for extrapolating from animals to humans
- LOAEL to NOAEL extrapolation
- Subchronic to chronic, accounts for extrapolating from a subchronic NOAEL or LOAEL to a chronic NOAEL or LOAEL
- Incomplete database, accounts for the lack of data for critical endpoints (e.g., reproductive and developmental).

Uncertainty factors of 1, 3, or 10 are used. The default value is 10; however, an uncertainty factor of 3 may be used if appropriate pharmacokinetic data (or models) are available. In addition, a modifying factor may be applied to account for additional uncertainties in accordance with professional judgment. The default value for the modifying factor is 1. All uncertainty factors (UFs) and the modifying factor (MF) are multiplied together to derive the total uncertainty factor, with 3,000 being the maximum recommended value (U.S. EPA, 1994b). Therefore, the RfD (or RfC) is derived by using the following formula:

$$RfD = NOAEL/(UF \times MF).$$

The effect of applying uncertainty and modifying factors is to lower the estimate of the reference dose and increase the hazard quotient (HQ) for a given exposure.

The RfD for methylmercury, which was developed to be protective of exposures in utero (1E-04 mg/kg-d, see Section 7.3.15) was applied in this risk assessment not only to maternal exposures but also to nonmaternal adult and child exposures based on the assumption that this RfD would be protective of neurological and/or developmental effects in these populations as well. This assumption is reasonable given that the most sensitive subpopulation identified in the epidemiological study underpinning the RfD is the infant exposed during fetal development. This approach should generate risk estimates for the nonmaternal adult and child that include an additional margin of safety; however, it does introduce uncertainty associated with extrapolating the RfD to these nonmaternal receptor populations.

For methylmercury, the reference dose represents a "no-effects" level that is presumed to be without appreciable risk. In deriving the reference dose, EPA used an uncertainty factor of 10

to derive the RfD for methylmercury from a benchmark dose that represents the lower 95 percent confidence level for the 10 percent incidence rate of neurologic abnormalities in children.[8] Therefore, there is a relatively small margin of safety between the RfD and the level corresponding to the threshold for adverse effects, as indicated by the human health data.

The current RfD was derived from an epidemiological study of a population in rural Iraq accidently exposed to methylmercury through home-baked bread made with contaminated grain. Some of the limitations associated with this study include a relatively short exposure duration (2 to 3 months) and the assumptions used to estimate daily intake levels from hair concentrations of mercury measured in the mothers. Some concerns have been raised about the applicability of a dose-response estimate from a grain-consuming population rather than a fish-consuming population. However, there is no compelling evidence suggesting that ingesting methylmercury in grain would be different from ingesting methylmercury in fish (U.S. EPA, 1997). Furthermore, numerous data from experimental animals, including primates, are available that report subtle sensory, cognitive, and motor deficits following long-term exposures. Although the animal data generally support the findings from the epidemiological studies, they also point out the potential importance of considering more subtle neurological endpoints (e.g., scores from sensory-motor and neuropsychological tests) from the human data rather than relying on the traditional developmental milestones (e.g., walking, talking) (U.S. EPA, 1997). Two important human epidemiological studies are currently in progress. These studies are examining childhood development and neurotoxicity associated with exposure to methylmercury from ingesting fish and whales. As these data become available, they will provide important new information for reassessing the RfD.

Toxicity Equivalence for Dioxin/Furan Congeners. The primary sources of uncertainty associated with the toxicity equivalency factor (TEF) approach for evaluating risk from environmental exposure to dioxin/furan congeners are common to human toxicologic benchmarks used in all risk assessments. These include both intra- and interspecies differences in susceptibility and response and extrapolation from high-dose to low-dose exposure. Two major uncertainties have been identified: (1) TEFs are based on the assumption that the effects of dioxin and furan congeners are additive and, therefore, do not consider possible synergistic or antagonistic relationships between various congeners: (2) TEFs do not account for pharmacokinetic processes, which can influence the dose (i.e., the change in mixture composition related to elimination and in vivo transformation of congeners).

Individual congeners are not found alone in the environment, but rather occur as complex mixtures. The TEF scheme was developed to assess the risk of these complex dioxin/furan mixtures. The TEF approach, however, is based on the assumption that the combined effect is equal to the sum of the individual congener effects and that the effects are mediated via binding to the Ah receptor. Data describing the mechanisms of action beyond receptor binding and induction of cytochrome P450, however, are lacking. Nonadditive interactions, particularly with non-dioxin-like PCBs, have been identified. These interactions contribute to the uncertainty associated with the TEQ approach for dioxin risk assessment. Nevertheless, Van den Berg et al.

[8] The uncertainty factor is intended to cover three areas of uncertainty: lack of data from a two-generation reproductive assay; variability in the human population, in particular the wide variation in the distribution and biological half-life of methylmercury; and lack of data on long-term sequelae of developmental effects.

(1998) reported that the additive model for TEFs remains the most plausible and that errors in prediction due to interactions were unlikely to be large.

At present, there are large data gaps regarding the pharmacokinetic processes and toxicity of non-2,3,7,8-TCDD congeners. Adequate toxicity data to assess the risk of individual congeners are lacking (e.g., chronic carcinogenic bioassays). These data gaps contribute to the uncertainty associated with using TEFs. To more accurately estimate the total potential for risk, data on the yearly distribution, metabolism, half-lives, and other properties for each congener would be needed. Pharmacokinetic data are very important for determining potential differences in response following acute, subchronic, or chronic exposure or high-dose and low-dose exposure. Such data would reduce uncertainty by answering questions regarding the applicability of a TEF scheme across exposure levels and durations. Although there are uncertainties, the available scientific data generally support the TEF model as the most plausible and feasible method for assessing the risk of dioxin mixtures.

The remainder of this section addresses individual-level risks for enumerated receptors (including commercial farmers, residents, and home gardeners), individual-level risks for subsistence scenarios, and population-level risks for human receptors.

2.1.2 Characterization of Individual-Level Risk for Enumerated Receptors

Individual risk for the enumerated receptors (for most types of cancer and noncancer effects) was characterized through the use of cumulative risk distributions, which were constructed by weighting sector-level individual risk estimates by the number of individuals located in that sector and then pooling those weighted risk estimates. These pooled risk estimates were then ranked according to risk magnitude, and specific percentiles of interest were identified. These percentiles can be interpreted as representing the risk level experienced by the individual located at that point on the risk distribution (i.e., central tendency or high-end risk estimates can be identified). These cumulative risk distributions include a number of factors designed to make them representative of the receptors for which they were developed:

- They reflect the location and density of receptors across study areas.
- They are based on central tendency exposure parameters (key exposure pathways include exposure parameter variability analyses designed to incorporate this additional source of variability into the characterization of risk).
- They are based on a 16-sector template, which enhances resolution in assessing exposure.

A wide range of cancer and noncancer effects were characterized for the enumerated receptors considered in this analysis. Of these effects, a relatively small number have been selected for discussion here based on the potential significance of their findings.

2.1.2.1 <u>Dioxins/Furans (Cancer Risk)</u>. Dioxins/furans were identified at proposal as a potential risk driver for the HWC risk analysis. Health effects are presented in greater detail in Section 7. Dioxins have been shown to cause a variety of cancers, including cancer of the lung

and soft tissue sarcomas. Dioxin causes a variety of toxicities in test animals following exposure. Although the human data are less clear, they are qualitatively and quantitatively consistent with the animal findings. There are sufficient data to warrant concern that this compound will induce toxic effects in humans in the range of the experimental animal data. Although EPA felt that there were a sufficient number of dose-response curves consistent with linearity to warrant concern about nonlinear extrapolations, there is no way to disprove scientifically the existence of nonlinearity in the area below the experimental region.

These constituents can accumulate in agricultural commodities (primarily meats and dairy products), thereby resulting in increased cancer risk for the consumers of these products. The market basket approach used at proposal demonstrated that individual-level risks to the average consumer from the consumption of meats and dairy products that are raised within study areas and contaminated with dioxins/furans were found to be low. However, commercial farmers who raise beef cattle, dairy cattle, or hogs and engage in the consumption of home-produced agricultural commodities remain a potential concern due to their higher rates of consumption of contaminated commodities (i.e., home-produced agricultural commodities). Therefore, these commercial farmer receptors were evaluated for the final rule.

Families that raise dairy cattle and, therefore, consume home-produced milk represent the receptor population most exposed to dioxins/furans released from HWC facilities. This is because the dairy cattle concentrate dioxins/furans after ingesting forage, silage, and grain that is grown locally and has been contaminated with these constituents through direct deposition of particles and vapor transfer. Dairy cattle are also exposed to dioxins/furans through the incidental ingestion of soil. Children of dairy farmers demonstrate the greatest exposure to dioxins/furans due to their high consumption rate of milk on a per body weight basis relative to adults.

Carcinogenic risk for dioxins/furans is expressed as the incremental lifetime excess cancer risk that results from exposure to dioxins/furans that are released from HWC facilities. In generating these estimates, a dioxin toxicity equivalent (dioxin-TEQ) approach is used. The dioxin-TEQ approach is a unifying approach applicable to all 2,3,7,8-chlorine-substituted dibenzo(*p*)dioxin and dibenzofuran congeners that is based on a common mechanism of action[9]. The total intake of contaminant from all applicable exposure pathways was used to estimate the lifetime average daily dose (LADD) for a given human receptor. LADDs then were multiplied by the appropriate CSF for 2,3,7,8-tetrachlorodibenzo(*p*)dioxin (TCDD) (i.e., either oral or inhalation) to produce a lifetime excess cancer risk estimate.

Table 2-2 presents summary data for this category of risk results.

Risk results generated for the final rule (for the child of the dairy farmer) project high-end lifetime excess cancer risks for the cement kiln category of less than 1E-05. Although projected high-end risks for area source cement kilns range up to 1E-05 (99th percentile), no reductions in risk are projected under implementation of the MACT standards for this category.

[9] With the dioxin-TEQ approach, each of the modeled media concentrations generated for the 17 dioxin/furan congeners considered in the analysis first was converted to an equivalent concentration of 2,3,7,8-TCDD using congener-specific toxicity equivalency factors (TEFs). The equivalent concentrations were summed to produce a single TEQ.

Table 2-2. Lifetime Excess Cancer Risk from Incremental Exposures to Dioxins for 0- to 5-Yr-Old Children of Dairy Farmers (with 90% Confidence Intervals)[a]

Emissions	Percentile of the Cumulative Distribution (Population Weighted)			
	50%	90%	95%	99%
Cement Kilns				
Baseline	1E-07 *(9E-08, 2E-07)*	1E-06 *(1E-06, 2E-06)*	3E-06 *(3E-06, 3E-06)*	7E-06 *(7E-06, 7E-06)*
Final Standards	1E-07 *(7E-08, 1E-07)*	1E-06 *(9E-07, 1E-06)*	2E-06 *(2E-06, 2E-06)*	5E-06 *(5E-06, 5E-06)*
Area Source Cement Kilns				
Baseline	2E-07 *(1E-07, 3E-07)*	3E-06 *(2E-06, 3E-06)*	5E-06 *(5E-06, 6E-06)*	1E-05 *(1E-05, 1E-05)*
Final Standards	2E-07 *(1E-07, 3E-07)*	3E-06 *(2E-06, 3E-06)*	5E-06 *(5E-06, 6E-06)*	1E-05 *(1E-05, 1E-05*
Lightweight Aggregate Kilns				
Baseline	4E-07	4E-06	7E-06	2E-05
Floor	4E-07	4E-06	7E-06	2E-05
Final Standards	1E-07	2E-07	5E-07	7E-07
All Incinerators				
Baseline	1E-08 *(6E-09, 3E-08)*	7E-07 *(4E-07, 1E-06)*	2E-06 *(1E-06, 2E-06)*	8E-06 *(5E-06, 1E-05)*
Final Standards	8E-09 *(5E-09, 1E-08)*	1E-07 *(1E-07, 2E-07)*	3E-07 *(2E-07, 4E-07)*	1E-06 *(8E-07, 1E-06)*
Area Source Incinerators				
Baseline	2E-08 *(3E-09, 1E-07)*	1E-06 *(1E-07, 3E-06)*	3E-06 *(3E-07, 5E-06)*	1E-05 *(3E-06, 2E-05)*
Final Standards	1E-08 (3E-09, 2E-08)	2E-07 (7E-08, 3E-07)	3E-07 (2E-07, 6E-07)	1E-06 [b]
Commercial Incinerators				
Baseline	2E-08 *(4E-09, 2E-07)*	1E-06 *(2E-07, 3E-06)*	3E-06 *(6E-07, 5E-06)*	1E-05 *(4E-06, 2E-05)*
Final Standards	1E-08 *(3E-09, 3E-08)*	2E-07 *(8E-08, 3E-07)*	4E-07 *(2E-07, 6E-07)*	1E-06 *(8E-07, 2E-06)*
Large On-site Incinerators				
Baseline	2E-08 (1E-08, 4E-08)	6E-07 (2E-07, 1E-06)	1E-06 (7E-07, 2E-06)	5E-06 (4E-06, 8E-06)
Final Standards	2E-08 (9E-09, 3E-08)	2E-07 (1E-07, 3E-07)	4E-07 (2E-07, 5E-07)	1E-06 [b]
Small On-site Incinerators				
Baseline	4E-09 *(1E-10, 4E-08)*	5E-07 *(7E-08, 1E-06)*	1E-06 *(3E-07, 2E-06)*	7E-06 [b]
Final Standards	2E-09 *(1E-10, 7E-09)*	6E-08 *(3E-08, 1E-07)*	1E-07 *(7E-08, 2E-07)*	5E-07 *(2E-07, 8E-07)*
Waste Heat Boilers				
Baseline	3E-07 *(2E-07, 4E-07)*	3E-06 *(2E-06, 4E-06)*	6E–06 *(4E-06, 9E-06)*	2E-05 *(1E-05, 2E-05)*
Floor	3E-07 *(2E-07, 4E-07)*	3E-06 *(2E-06, 4E-06)*	6E–06 *(4E-06, 9E-06)*	2E-05 *(1E-05, 2E-05)*
Final Standards	2E-08 *(2E-08, 4E-08)*	2E-07 *(2E-07, 4E-07)*	5E-07 *(3E-07, 7E-07)*	1E-06 *(8E-07, 2E-06)*

[a] Includes cancer risk from incremental exposures to 2,3,7,8 chlorine-substituted dibenzo(*p*)dioxins and dibenzofurans, expressed as TCDD-TEQs.
[b] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

High-end risks for lightweight aggregate kilns (LWAKs) are estimated to range from 4E-06 to 2E-05 (90th and 99th percentiles, respectively). High-end risk reductions on the order of 97 percent are projected to occur for this combustor category with MACT standard implementation (i.e., 2E-05 for 99th percentile baseline to 7E-07 for 99th percentile MACT standard). Baseline central tendency risks (50th percentile) for LWAKs are estimated as 4E-07.

Although high-end risks estimated for incinerators (INC) as an aggregate group are below 1E-05, several of the incinerator categories have projected high-end risks above 1E-05.[10] These same categories also demonstrate high-end risk reductions with MACT standard implementation of approximately an order of magnitude. Area source incinerators have high-end projected risks ranging from 1E-06 to 1E-05 (90th to 99th percentiles, respectively). Risk reductions of approximately an order of magnitude are projected for this combustor category with MACT standard implementation (e.g., 1E-05 for 99th percentile baseline to 1E-06 for 99th percentile MACT standard). Central tendency risks at baseline are estimated at 2E-08 for area source incinerators. Commercial incinerators are also projected to have high-end risks in the range of 1E-05 (99th percentile) and risk reductions for MACT standard implementation near an order of magnitude (e.g., 1E-05 for 99th percentile baseline to 1E-06 for 99th percentile MACT standard). High-end risks for both the large on-site incinerator (OINC-L) and small on-site incinerator (OINC-S) categories are projected to fall below 1E-05 at baseline. High-end risks for waste heat boilers are estimated to range from 3E-06 to 2E-05 (90th and 99th percentiles, respectively). Risk reductions of an order of magnitude are projected for this combustor category with MACT standard implementation (although no risk reductions are projected for WHBs at the floor).

The distribution of lifetime excess cancer risk reflects variability in: (1) site-specific differences in factors related to air dispersion/deposition (e.g., facility emissions, facility parameters, and meteorological conditions), (2) the location and density of dairy farms relative to modeled HWC facilities, and (3) interindividual differences in duration of exposure and the amount of home-produced milk that is ingested. Factors not reflected in the distribution of risk results include regional variation in agricultural practices that could affect the level of dioxin/furan bioaccumulation in milk (e.g., the amount of grain consumed relative to the amount of forage and silage). However, given the high degree of variability attributable to the factors that are considered in the analysis, it is not clear that the factors that were excluded from consideration would have had a significant impact on the overall distribution of risk results for this receptor.

The distributions of lifetime excess cancer risk generated for dioxin/furan exposure for the commercial dairy farmer are subject to some uncertainty. This uncertainty results from factors discussed in Section 2.1.1 including: (1) averaging of air modeling results within sectors (possible exclusion of hot spots), (2) farm food chain modeling of dioxins/furans from plant uptake into silage/forage/grain through bioconcentration in cattle following grazing, (3) inability to consider commercial farmers who produce more than one agricultural commodity,

[10] Subsets of the incinerator category (e.g., waste heat boilers), when analyzed as separate combustor categories, display higher percentile risk estimates because the facilities constituting these categories have higher risks compared to facilities in other incinerator categories. Consequently, when the “higher risk” categories are aggregated with other incinerator facilities to form the “incinerator” category, the overall distribution of risk is altered by the larger pool of data at the high end.

(4) assumption of uniform consumption rates for home-produced foods by commercial farmers, (5) use of 30-year modeled HWC facility lifetime, and (6) cancer slope factor derivation. Other uncertainties include limitations of the data available to assess consumption of home-produced milk. Uncertainty is also introduced through the assumption of additivity of dioxin and furan mixtures that is inherent in the TEQ approach.

2.1.2.2 Dioxins/Furans (Noncancer). As with the characterization of individual cancer risk for dioxins/furans, the characterization of noncancer risk has also focused on commercial farmer receptors who are exposed to these constituents through the consumption of home-produced agricultural commodities.[11] In lieu of an RfD for dioxins/furans, exposure levels were compared to reported background exposure levels in the general population and a margin of exposure estimate was calculated as the ratio of predicted exposure to background. A value greater than 1 implies that exposure exceeds expected background exposure but does not necessarily imply that noncancer health effects are likely. Health effects of dioxins are discussed below and in Section 7.0. The major noncarcinogenic effect from exposure to dioxin is chloracne, a severe acne-like condition that develops within months of first exposure to high levels of dioxin. There are limited human data to suggest the doses at which chloracne is likely to occur. However, based on the available mechanistic information, it is likely that exposure to dioxins/furans could induce a broad spectrum of effects. These may include altered cellular function, changes in hormone levels, and enzyme induction that could occur within or near the current background range of human exposure (U.S. EPA, 1994a). The true clinical significance of these effects in humans is unknown and is an active area of research. Therefore, noncancer risk assessment for dioxins/furans remains controversial and highly uncertain.

Because the children of commercial dairy farmers represent the receptor with the greatest exposure to dioxins/furans, this receptor forms the basis for the discussion of noncancer risk characterization for dioxins/furans.

For noncancer effects resulting from exposure to dioxins/furans, it is not appropriate to develop a reference dose, or level that is without appreciable risk, using standard uncertainty factors. This is due to the high levels of background exposures in the general population and the low levels at which effects have been seen in laboratory animals. Instead, a margin of exposure approach is used in which the average daily dose from a given source is compared to the average daily dose in the general population. The ratio of the two represents the incremental margin of exposure (incremental MOE) and, as such, measures the relative increase in exposures over background. Background levels used in the incremental MOE analysis, which are generally presumed to pose minimal risk (although, as noted above, this is not certain for dioxins/furans), were selected to be representative of typical exposure conditions experienced by the general population (i.e., they reflect a combination of both natural and anthropogenic background). As in cancer risk characterization, the incremental MOE analysis for dioxins/furans was conducted using dioxin-TEQs derived from congener-specific TEFs (see Section 2.1.2.1).

Table 2-3 presents summary data for this category of risk results.

[11] Although residents are also potentially exposed to dioxins/furans through the consumption of locally produced agricultural commodities, the market basket analysis conducted at proposal showed this pathway to result in relatively low exposure of the residents to dioxins/furans.

Table 2-3. Incremental Margin of Exposure to Dioxins for 0- to 5-Yr-Old Children of Dairy Farmers (with 90% Confidence Intervals)[a, b]

Emissions	Percentile of the Cumulative Distribution (Population Weighted)[c]			
	50%	**90%**	**95%**	**99%**
Cement Kilns				
Baseline	1E-02 *(8E-03, 2E-02)*	4E-02 *(3E-02, 4E-02)*	5E-02 *(5E-02, 6E-02)*	9E-02 *(8E-02, 9E-02)*
Final Standards	9E-03 *(8E-03, 1E-02)*	2E-02 *(2E-02, 2E-02)*	3E-02 *(3E-02, 3E-02)*	6E-02 *(6E-02, 6E-02)*
Area Source Cement Kilns				
Baseline	2E-02 *(1E-02, 2E-02)*	6E-02 *(5E-02, 6E-02)*	9E-02 *(8E-02, 9E-02)*	2E-01 *(2E-01, 3E-01)*
Final Standards	2E-02 *(1E-02, 2E-02)*	6E-02 *(5E-02, 6E-02)*	9E-02 *(8E-02, 9E-02)*	2E-01 *(2E-01, 3E-01)*
Lightweight Aggregate Kilns				
Baseline	4E-02	9E-02	1E-01	2E-01
Floor	4E-02	9E-02	1E-01	2E-01
Final Standards	6E-03	1E-02	2E-02	3E-02
All Incinerators				
Baseline	9E-04 *(5E-04, 3E-03)*	3E-02 *(2E-02, 4E-02)*	4E-02 *(4E-02, 7E-02)*	1E-01 *(9E-02, 2E-01)*
Final Standards	8E-04 *(5E-04, 1E-03)*	4E-03 *(3E-03, 5E-03)*	7E-03 *(4E-03, 9E-03)*	1E-02 *(1E-02, 2E-02)*
Area Source Incinerators				
Baseline	9E-04 *(1E-04, 2E-02)*	4E-02 *(6E-03, 6E-02)*	7E-02 *(2E-02, 9E-02)*	2E-01 *(6E-02, 3E-01)*
Final Standards	9E-04 *(1E-04, 2E-03)*	4E-03 *(3E-03, 7E-03)*	7E-03 *(4E-03, 9E-03)*	2E-02 *(9E-03, 3E-02)*
Commercial Incinerators				
Baseline	9E-04 *(1E-04, 2E-02)*	4E-02 *(1E-02, 7E-02)*	7E-02 (3E-02, 9E-02)	2E-01 *(8E-02, 3E-01)*
Final Standards	9E-04 *(1E-04, 2E-03)*	6E-03 *(3E-03, 7E-03)*	8E-03 *(6E-03, 1E-02)*	2E-02 *(1E-02, 3E-02)*
Large On-site Incinerators				
Baseline	1E-03 *(7E-04, 3E-03)*	3E-02 *(1E-02, 4E-02)*	4E-02 *(3E-02, 4E-02)*	9E-02 *(4E-02, 9E-02)*
Final Standards	1E-03 *(7E-04, 2E-03)*	5E-03 *(3E-03, 9E-03)*	9E-03 *(4E-03, 1E-02)*	1E-02 *(8E-03, 1E-02)*
Small On-site Incinerators				
Baseline	3E-04 *(4E-06, 9E-03)*	1E-02 *(3E-03, 4E-02)*	4E-02 *(1E-02, 9E-02)*	1E-01 [b]
Final Standards	3E-04 *(4E-06, 8E-04)*	2E-03 *(9E-04, 3E-03)*	3E-03 *(2E-03, 4E-03)*	9E-03 *(5E-03, 1E-02)*
Waste Heat Boilers				
Baseline	3E-02 *(1E-02, 3E-02)*	7E-02 *(4E-02, 9E-02)*	1E-01 *(5E-02, 1E-01)*	3E-01 *(1E-01, 5E-01)*
Floor	3E-02 *(1E-02, 3E-02)*	7E-02 *(4E-02, 9E-02)*	1E-01 *(5E-02, 1E-01)*	3E-01 *(1E-01, 5E-01)*
Final Standards	2E-03 *(1E-03, 3E-03)*	5E-03 *(3E-03, 7E-03)*	8E-03 *(5E-03, 9E-03)*	2E-02 *(1E-02, 3E-02)*

[a] Includes incremental exposures to 2,3,7,8-chlorine-substituted dibenzo(*p*)dioxins and dibenzofurans, expressed as TCDD-TEQs.
[b] Relative to an average background exposure to the general population of 1.5 pg/kg-d.
[c] Percentiles do not reflect variability in the population of the amount of milk consumed.
[d] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

Risk results generated for the final rule (for the child of the dairy farmer) project high-end incremental MOE levels at baseline to be at or below 0.1 except at the 99th percentile for area source cement kilns, LWAKs, area source incinerators, commercial incinerators, and WHBs. For these combustor categories, the ratios are 0.2 for all but WHBs and 0.3 for WHBs at the 99th percentile. Further, under MACT, the 99th percentiles are reduced to below 0.1 for LWAKs, area source incinerators, commercial incinerators, and WHBs. However, all of the incinerator categories have projected reductions in incremental MOE levels with MACT standard implementation of approximately an order of magnitude (e.g., the WHB category has 99th percentile incremental MOE levels of 3E-01 at baseline and 2E-02 with MACT standard implementation). It should be noted that the cement kiln category demonstrates a small reduction in projected incremental MOE levels under MACT standard implementation.

The distribution of incremental MOE results for dioxins/furans reflects variability associated with site-specific differences in factors related to air dispersion/deposition (e.g., facility emissions, facility parameters, and meteorological conditions) and the location and density of dairy farms relative to modeled HWC facilities. Factors not reflected in the distribution include interindividual differences in the amount of home-produced milk that is ingested and regional variation in agricultural practices that could affect the level of dioxin/furan bioaccumulation in milk (e.g., the amount of grain consumed relative to the amount of forage and silage). Although the incremental MOE results for dioxins/furans do not reflect variability in either exposure parameters or regional variability in agricultural practices, it is believed that a significant portion of the variability affecting this category of risk results has been captured in the factors that have been considered in the analysis.[12]

The distributions of incremental MOE estimates generated for dioxin/furan exposure for the commercial dairy farmer are subject to uncertainty. Many of the same sources of uncertainty that were discussed under carcinogenic risk characterization for dioxins/furans apply here for noncancer risk characterization (e.g., fate and transport modeling used to project dioxin/furan concentrations in agricultural commodities and the potential exclusion from the analysis of farms located at hot spots due to averaging of air modeling results over sectors).

In addition, there is uncertainty associated with the background exposure value used in the incremental MOE calculation for the child of the dairy farmer. Because an accepted childhood background exposure value was not available, an adult value was used, which introduces uncertainty. Although not well characterized, background exposures in children are expected to be somewhat higher than those for adults because of the child's greater intake of virtually all "environmental media." Therefore, the child incremental MOE calculated with adult background will generally be biased upward and will tend toward the adult incremental MOE when the appropriate child-specific background value is used. In addition, the adult background exposure value for dioxin-TEQ is itself subject to uncertainty resulting from the approach used to generate the value. The adult background dose estimate for dioxin-TEQ used in the HWC risk analysis was generated using pharmacokinetic modeling and steady-state assumptions to backcalculate the dose estimate from an adult background body burden value. The steady-state

[12] In addition, for cancer risk (for dioxins/furans), inclusion of exposure factor variability increased the risk at the upper percentiles by less than a factor of 2 to a factor of 5 for the same receptor population and exposure pathway (i.e., milk consumption).

assumption used in this calculation implies that past exposure to dioxin-TEQ was constant. Current body burden levels, however, are likely to result from nonconstant (i.e., variable) exposure levels over past decades. When non-steady-state conditions are used in conducting pharmacokinetic modeling for purposes of backcalculating dose estimates from these background body burden values, the resulting dose estimates can be significantly lower than values generated assuming steady-state conditions (i.e., continuous exposure). Therefore, the generation of background dose estimates from background body burden values assuming steady-state conditions, as was conducted for the HWC risk analysis, could result in an overestimation of background dose levels, which, in turn, would result in an underprediction of incremental MOE. Taken together, uncertainty associated with using the adult background dose estimate to represent children (which will tend to overestimate incremental MOE) and uncertainty associated with the adult background dose estimate itself (which will tend to underpredict incremental MOE) work to counteract each other. However, the overall impact from these two sources of parameter uncertainty on incremental MOE results for the child of the dairy farmer has not been quantified.

There is also significant uncertainty associated with using an incremental MOE approach to characterize the potential for noncancer health effects. When an RfD is available for a given constituent, the risk results generated (i.e., hazardous quotients) can be viewed as identifying the potential for adverse effects in the modeled receptor. By contrast, incremental MOE results simply establish whether modeled exposure levels exceed typical background concentrations—they make no clear statement regarding the potential for adverse effects. Although some researchers have suggested that dioxin/furan concentrations near background levels may result in adverse effects, a toxicity factor reflecting noncancer effects has not been developed for this group of constituents. The use of the incremental MOE as a noncancer risk descriptor, similar to a hazard quotient, presumes implicitly that background exposures are associated with de minimis risks.

2.1.2.3 Lead. The potential for adverse effects resulting from exposure to lead was assessed in this analysis for children 0 to 5 years of age because this age group is known to be highly sensitive to lead exposure. Human health effects are discussed in detail in Section 7.0. Human studies are inconclusive regarding lead and an increased cancer risk; however, lead is classified as a probable human carcinogen. The primary effects in humans from chronic exposure to lead are to the nervous system; children are particularly sensitive to the neurotoxic effects of lead.

Separate risk estimates were generated for the 0- to 5-yr-old age group from each of the modeled receptors; however, the child of the home gardener has been selected as the basis for discussing the risk results for lead since this receptor experiences the highest exposure to lead. The home gardener's relatively high exposure results from deposition of airborne lead on fruits and vegetables and consumption of home-produced fruits and vegetables. (Note: Exposures for the home gardener are generally comparable to those for the commercial produce farmer, since both consume home-produced fruits/vegetables.)

Blood lead levels (PbB levels) are used as the exposure metric in assessing risk resulting from exposure to lead. The potential for adverse effects resulting from exposure to this metal is evaluated by comparing modeled PbB levels to the action level established for lead of 10 µg/dL.

The 10-µg/dL PbB level requires further explanation to aid in the interpretation of PbB results. This action level is the current level of concern as defined by the Centers for Disease Control (CDC) and EPA. The level of concern is used in conjunction with lead screening programs to determine if a child has elevated blood lead levels. Early child exposure resulting in blood lead levels in the 10- to15-μg/dL range are known to increase the risk of irreversible neurobehavioral deficits (CDC, 1991). This is not to say that 10 μg/dL is a threshold level below which no effects will occur. In fact, a threshold level for lead is not evident from the available studies. The most sensitive indicators of effects in children are psychomotor tests or mental development indices. For example, several studies have reported a 2- to 4-point IQ deficit for each μg/dL increase in blood lead levels between 5 and 35 μg/dL (Goyer, 1996).

Modeled PbB levels were generated using the IEUBK model, which combines modeled media concentrations for lead along with exposure parameter values reflective of the 0- to 5-yr-old age group to make PbB projections for the receptor being modeled. The IEUBK model, as used in this analysis, generates incremental PbB levels (i.e., PbB levels reflecting exposure to lead released from the facility under consideration). These PbB levels are then further adjusted to reflect interindividual variations in the intake of lead (e.g., through soil ingestion) as well as pharmacokinetic factors.

Modeled PbB levels can be compared with background exposures in the same age group, i.e., children ages 0 to 5 years, in the general population. The median blood lead level in children in the general population is 2.7 µg/dL, and 4.4 and 1.3 percent of children have blood lead levels that exceed 10 and 15 µg/dL, the levels at which community-wide prevention and individual intervention efforts, respectively, are recommended.[13] However, the percentages vary widely depending on such factors as race, ethnicity, income, and age of the housing units occupied. Children whose blood lead levels are already elevated are the most susceptible to further increases in blood lead levels.

Table 2-4 presents summary data for this category of risk results. (Note: Although total, background, and incremental lead results were generated for the HWC risk analysis, Table 2-4 presents only incremental results since these are of primary concern for decision making. However, background, incremental, and total results are included in the detailed set of risk results generated for the HWC risk analysis.)

Projected high-end incremental PbB levels (for the child of the home gardener) for all combustor categories at baseline ranged from less than 0.01 µg/dL to 1.19 µg/dL (99th percentile for small on-site incinerators and 99th percentile for large on-site incinerators, respectively). Projected central tendency incremental PbB levels at baseline ranged from less than 0.01 to 0.40 µg/dL (50th percentile for LWAKs, and small on-site incinerators and 50th percentile for large on-site incinerators and all incinerators, respectively). All combustor categories demonstrated large reductions in incremental PbB levels under MACT standard implementation (e.g., 0.50 µg/dL baseline to <0.03 µg/dL MACT standard—99th percentile cement kilns). The PbB reductions projected for the MACT standard should be considered in the context of the magnitude of the

[13] Data from the CDC's National Health and Nutrition Examination survey (NHANES III, phase 2) conducted from October 1991 to September 1994 (CDC, 1997).

Table 2-4. Blood Levels for Incremental Exposure to Lead for 0- to 5-Yr-Old Children of Home Gardeners (µg Pb/dL blood)[a]

Source	50%	90%	95%	99%
Cement Kilns				
Baseline	0.17	0.31	0.37	0.50
Floor	0.02	0.04	0.04	0.06
Final Standards	0.01	0.02	0.02	0.03
Area Source Cement Kiln				
Baseline	0.02	0.04	0.04	0.06
Floor	0.02	0.04	0.02	0.03
Final Standards	<0.01	0.02	0.02	0.03
Lightweight Aggregate Kilns				
Baseline	<0.01	0.02	0.02	0.03
Floor	<0.01	0.02	0.02	0.03
Final Standards	<0.01	<0.01	<0.01	<0.01
All Incinerators				
Baseline	0.40	0.73	0.85	1.17
Final Standards	0.01	0.02	0.02	0.03
Area Source Incinerators				
Baseline	0.07	0.13	0.15	0.21
Final Standards	<0.01	0.02	0.02	0.03
Commercial Incinerators				
Baseline	0.07	0.13	0.15	0.21
Final Standards	<0.01	0.02	0.02	0.03
Large On-site Incinerators				
Baseline	0.40	0.72	0.84	1.19
Final Standards	<0.01	<0.01	<0.01	<0.01
Small On-site Incinerators				
Baseline	<0.01	<0.01	<0.01	<0.01
Final Standards	<0.01	<0.01	<0.01	<0.01

[a] A blood level of 10 µg Pb/dL is the level at which community-wide lead poisoning prevention activities are indicated.

incremental PbB levels that are involved. Modeled incremental PbB levels for all combustor categories at both baseline and under MACT standard implementation are below the average background levels identified by the CDC (i.e., 2.7 µg/dL) (CDC, 1997).

The distribution of PbB levels for lead reflects site-specific differences in factors related to air dispersion/deposition (e.g., facility emissions, facility parameters, and meteorological conditions) and the location and density of home gardeners. In addition, interindividual variability in pharmacokinetic parameters is also reflected in the PbB results. Factors not reflected in the PbB modeling include intersite variability in background exposure to lead and certain components of exposure parameter variability. The IEUBK model considers interindividual variability in behavior related to lead exposure (i.e., ingestion rates). The most important of such behavior in generating PbB estimates is mouthing behavior by infants and very young children. However, the model does not explicitly consider variability for all parameters associated with the pathways assessed for the home gardener receptor (i.e., consumption of home-produced fruits/vegetables). Therefore, the PbB levels that are generated may not fully reflect interindividual variability in PbB levels that result from differences in exposure parameters.

There is significant uncertainty associated with the risk results generated for lead exposure. This uncertainty results from several of the factors discussed in Section 2.1.1, including: (1) averaging of air modeling results within sectors (exclusion of hot spots), (2) uncertainties related to air dispersion/deposition modeling, and (3) use of a 30-yr modeled HWC facility lifetime. Because the primary exposure pathway is ingestion of fruits and vegetables (which are contaminated primarily as a result of direct air deposition), accumulation of lead in soil over the life of the facility is not a significant source of uncertainty.

Additional uncertainty is introduced into the PbB analysis through the use of the IEUBK model to project PbB levels and the use of the 10-µg/dL action level as a health benchmark. Although the IEUBK model has been subjected to extensive scientific review and has been shown to work well when implemented correctly, like all models designed to characterize biological systems, there is inherent uncertainty in its output. In addition, for purposes of characterizing PbB levels at the sector-level using IEUBK, it was assumed that all children in the 0- to 5-year-old age group were 5 years of age. This simplifying assumption was used because it was not feasible to integrate age as a stochastic variable into the modeling structure.[14] The use of a single age in conducting IEUBK modeling introduces uncertainty into the PbB results, because, in reality, the 0- to 5-year-old age group within any given sector is comprised of a mix of children ranging in age from newborn to 5 years of age. Consequently, their PbB levels reflect a wide range of exposure durations from days (or less) to 5 years. Modeling all individuals within a given sector as 5-year-olds with regard to lead exposure probably results in an overestimation of PbB levels because individuals experiencing shorter exposure durations are not reflected in the results. However, the magnitude of this overestimation has not been quantified.

[14] To integrate age as a stochastic variable, a separate IEUBK modeling run would have had to be conducted for each iteration of the Monte Carlo analysis that is used to characterize the range of PbB levels for each sector (i.e., each modeled individual's PbB level would have had to be separately generated using IEUBK, instead of generating a single representative PbB level for the sector and applying the GSD for individual variability to characterize the range of PbB levels as was done in the current analysis).

Uncertainty is also associated with the use of the 10-µg/dL action level as a health benchmark. Unlike an RfD, which is intended to represent a threshold for adverse effects within an exposed population (and often includes an uncertainty factor to account for sensitive subpopulation), studies suggest that there may not be a threshold for adverse effects for lead exposure, with a wide range of exposure levels producing effects of varying severity. Because the lead action level does not represent a true threshold, interpretation of individual lead results generated for this analysis is significantly complicated.

Uncertainty is also associated with the approach used to characterize background exposure for lead. As noted above, only incremental lead results are presented in Table 2-4, however, the full set of detailed risk results generated for this analysis includes characterization of total lead exposure, which is comprised of background and incremental. The HWC risk analysis characterizes total lead exposure by adding a national level geometric mean for background exposure (3.6 µg/dL) to each sector-level modeled incremental exposure estimate generated using IEUBK (see Section 8.2.4 for additional detail on the method used to characterize total, background, and incremental lead exposure). To reflect interindividual variability in factors related to lead bioaccumulation, a GSD of 1.6 is then applied to this sector-level total lead exposure estimate to generate a sector-level distribution of total PbB levels in a given receptor population. The GSD that is used is the value specified in the IEUBK guidance document for reflecting interindividual variability in a site-level analysis. Although this GSD is appropriate for reflecting interindividual variability in factors related to lead bioaccumulation (including pharmacokinetics and behavior related to lead exposure), it is not intended to reflect **regional** variation in background exposure. Consequently, it may underpredict overall background variability across sites, which is dependent not only on individual differences in pharmacokinetics and behavior but also on background lead concentrations in different media.

Additional uncertainty in characterizing background lead exposure results from an inability to integrate newly released data on background lead levels into the HWC risk analysis. Subsequent to completing PbB modeling for the HWC risk analysis, the CDC released a report summarizing the background PbB level results generated by NHANES III (CDC, 1997). The CDC has conducted an ongoing series of national studies of the health of the civilian noninstitutionalized population. NHANES has been the primary source of monitoring blood lead levels in the U.S. population. Phase 2 of NHANES III, which was conducted from 1991 to 1994, identified a national level geometric mean for background exposure of 2.7 µg/dL, which is lower than the value used in the HWC risk analysis. In addition, a preliminary analysis of data contained in the CDC report suggests that the GSD for background lead exposure in children could be higher than 1.6 (this is in line with concerns expressed above that the IEUBK-based GSD might not have captured regional variations in background lead concentrations for different media). The use of a lower mean for background exposure would tend to shift the entire PbB distribution for total and background exposure down, while the use of a higher GSD would tend to stretch out both tails, resulting in higher upper-end PbB predictions for both total and background exposure.[15] It is difficult to clearly state what the overall effect of these two factors would be on specific PbB percentiles (e.g., how the 95th percentile total PbB level would change

[15] It is arguable that the GSD that would be derived from the CDC data should not be used in characterizing interindividual variability alone (i.e., in generating incremental PbB estimates, which are site-specific) because it incorporates variation in background levels.

if the new mean and GSD values derived from the CDC data were used). However, a comparison of the number of children with elevated blood lead levels (above 10 µg/dL) predicted using the HWC approach (less than 2 percent) and those predicted using the CDC mean and GSD (4.4 percent) suggests that background and total PbB exposure estimates would probably increase if the new CDC mean and GSD were used.

2.1.2.4 Arsenic (Cancer Risk). Risk results generated at proposal identified ingestion cancer risk for arsenic as a potential risk driver for the analysis. In addition, arsenic is the only metal, of the 14 metals assessed in this analysis, for which an ingestion cancer slope factor is available. Consequently, ingestion cancer risk for arsenic has been selected as a pathway of interest in summarizing risk results for the final rule.

Families that raise dairy cattle and are exposed to arsenic primarily through the ingestion of home-produced milk represent the receptor population most exposed to arsenic through the ingestion pathway. Arsenic concentrations in dairy milk result from the deposition of arsenic on forage and silage that is consumed by dairy cattle. Children of dairy farmers will have the greatest exposure to arsenic due to their high consumption rate of milk on a per unit body weight basis relative to adults.

Carcinogenic risk for arsenic is expressed as the lifetime excess cancer risk that results from exposure to this metal. Modeled arsenic concentrations for a specific medium are combined with the appropriate intake rates and averaged over a lifetime to produce an average lifetime daily dose for all exposure pathways combined. LADDs are then multiplied by the oral cancer slope factor for arsenic to produce a lifetime excess cancer risk.

Table 2-5 presents summary data for this category of risk results.

Risk results generated for the final rule project high-end lifetime excess cancer risks (for the child of the dairy farmer) for all combustor categories to be less than 1E-06. Both the cement kiln and LWAK combustor categories had less than a 25 percent reduction in high-end risk projected to result from implementation of the MACT standard (e.g., 4E-09 for 95th percentile cement kiln at baseline to 3E-09 with MACT standard implementation). Projected high-end risk reductions for all incinerator categories except large on-site incinerators, resulting from MACT standard implementation are in the range of 50 percent (e.g., 1E-09 for 99th percentile OINC-S at baseline to 5E-10 with MACT standard implementation). High-end risk estimates reflecting MACT implementation could not be generated for the large on-site incinerators due to small sample size or an insufficient spread of modeled risk values.

The distribution of lifetime excess cancer risk reflects variability in site-specific differences in factors related to air dispersion/deposition (e.g., facility emissions, facility parameters, and meteorological conditions) and the location and density of dairy farms relative to modeled HWC facilities. Factors not reflected in the distribution of risk results include exposure parameter variability (i.e., interindividual variability in intake of home-produced milk and duration of exposure). Although these factors were not explicitly considered for arsenic, information obtained from dioxin/furan modeling can provide some insight into the effect of exposure parameter variability. For dioxins, inclusion of exposure factor variability increased the

Table 2-5. Lifetime Excess Cancer Risk from Incremental Exposures to Arsenic for 0- to 5-Yr-Old Children of Dairy Farmers (with 90% Confidence Intervals)

Emissions	Percentile of the Cumulative Distribution (Population Weighted)[a]			
	50%	90%	95%	99%
Cement Kilns				
Baseline	1E-10 *(6E-11, 1E-10)*	1E-09 *(3E-10, 3E-09)*	4E-09 *(2E-09, 4E-09)*	5E-09 *(4E-09, 5E-09)*
Final Standards	1E-10 *(6E-11, 1E-10)*	9E-10 *(3E-10, 2E-09)*	3E-09 *(1E-09, 4E-09)*	5E-09 *(3E-09, 5E-09)*
Area Source Cement Kilns				
Baseline	2E-09 *(1E-09, 2E-09)*	4E-09 *(3E-09, 4E-09)*	4E-09 (4E-09, 4E-09)	4E-09 *(4E-09, 4E-09)*
Final Standards	1E-09 *(8E-10, 1E-09)*	3E-09 *(3E-09, 3E-09)*	3E-09 *(3E-09, 3E-09)*	3E-09 *(3E-09, 3E-09)*
Lightweight Aggregate Kilns				
Baseline	3E-10	6E-10	7E-10	1E-09
Final Standards	2E-10	6E-10	7E-10	1E-09
All Incinerators				
Baseline	2E-10 *(2E-11, 4E-10)*	4E-09 *(7E-10, 8E-08)*	8E-08 *(3E-09, 1E-07)*	2E-07 *(2E-08, 2E-07)*
Final Standards	7E-11 *(9E-12, 1E-10)*	3E-09 *(3E-10), 2E-08)*	2E-08 [b]	[b]
Area Source Incinerators				
Baseline	5E-10 *(4E-12, 2E-09)*	4E-09 *(8E-10, 4E-09)*	4E-09 *(2E-09, 4E-09)*	9E-09 *(4E-09, 1E-08)*
Final Standards	6E-11 *(4E-12, 2E-10)*	4E-09 *(9E-11, 4E-09)*	4E-09 *(2E-10, 4E-09)*	4E-09 *(2E-09, 4E-09)*
Commercial Incinerators				
Baseline	5E-10 *(4E-12, 1E-09)*	4E-09 *(8E-10, 4E-09)*	4E-09 *(2E-09, 4E-09)*	9E-09 *(4E-09, 1E-08)*
Final Standards	6E-11 *(3E-12, 2E-10)*	4E-09 *(1E-10, 4E-09)*	4E-09 *(2E-10, 4E-09)*	4E-09 *(2E-09, 4E-09)*
Large On-site Incinerators				
Baseline	6E-10 *(2E-10, 7E-10)*	1E-07 *(7E-10, 2E-07)*	2E-07 *(9E-10, 2E-07)*	2E-07 [b]
Final Standards	2E-10 *(9E-11, 4E-10)*	[b]	[b]	[b]
Small On-site Incinerators				
Baseline	8E-12 *(6E-10, 1E-10)*	3E-10 *(3E-11, 3E-10)*	3E-10 *(1E-10, 9E-10)*	1E-09 *(2E-10, 1E-09)*
Final Standards	7E-12 *(6E-12, 8E-11)*	1E-10 *(2E-11, 2E-10)*	2E-10 *(9E-11, 4E-10)*	5E-10 *(1E-10, 5E-10)*

[a] Percentiles do not reflect variability in the population of exposure factors such as the duration of exposure and milk consumption.
[b] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

risk at the upper percentiles by less than a factor of 2 to a factor of 5 for the same receptor population and exposure pathway (i.e., milk consumption).

The distributions of lifetime excess cancer risk generated for arsenic exposure for the commercial dairy farmer are impacted by several of the sources of uncertainty discussed in Section 2.1.1. These include uncertainties related to fate/transport modeling (e.g., air dispersion/deposition modeling and farm food chain modeling) and toxicity characterization (e.g., uncertainties related to the cancer slope factor for arsenic).

2.1.2.5 Inhalation Carcinogens (Arsenic, Beryllium, Cadmium, Chromium VI, Nickel, and Dioxin-TEQ). Estimates of the combined cancer risk associated with inhalation exposures to all inhalation carcinogens assumes additivity of the risks from individual compounds. Populations that have the highest inhalation exposures are adult farmers and nonfarm residents. Adults have the longest exposure duration relative to other age groups and adult farmers have less mobility and, therefore, longer durations of exposure than nonfarm residents. However, depending on the location of farms and nonfarm households, adult nonfarm residents can have lifetime average exposures that are as high as adult farm residents.

Under the MACT standards, high-end (99th percentile) lifetime excess cancer risk from inhalation exposures is below 6 in 10 million for all source categories.

Table 2-6 presents summary data for this category of risk results.

The risk distribution for inhalation carcinogens reflects variability in individual exposures due to site-specific differences in emissions, location of exposure, and other factors. However, it does not reflect differences between individuals in the length of exposure or other exposure factors. Therefore, risks at the upper percentiles may be underestimated to some extent.[16] A full exposure factor variability analysis was not carried out for inhalation carcinogens because the risks using mean exposure factors are comparatively low.

Projections of inhalation risks are subject to a number of uncertainties. Individuals spend a majority of their time indoors and it is uncertain how representative modeled outdoor ambient air concentrations are of concentrations indoors. Furthermore, the daily activities of individuals living in the vicinity of an emissions source will tend to moderate actual exposures compared to modeled exposures at a fixed location. Air modeling uncertainty is discussed in Section 2.1.1.3. Nevertheless, inhalation risks are projected to be sufficiently low that the uncertainties introduced by these factors are not likely to have an appreciable effect on the overall conclusions.

2.1.2.6 Hydrogen Chloride (Inhalation). Of the compounds evaluated that are not carcinogenic, the highest inhalation exposures are for hydrogen chloride (HCl). Human health effects are presented in detail in Section 7.0. The acute effects on humans exposed by inhalation to hydrogen chloride include coughing, choking, inflammation and ulceration of the respiratory tract, chest pain, and pulmonary edema. Oral exposure may result in corrosion of the mucous

[16] The precise extent of underestimation at the upper percentiles associated with variability in the duration of exposure is unknown but is expected to be a factor of 3 or less.

Table 2-6. Total Lifetime Excess Cancer Risk from Incremental Exposures to Inhalation Carcinogens for Adult Residents (with 90% Confidence Intervals)[a]

Emissions	Percentile of the Cumulative Distribution (Population Weighted)[b]			
	50%	90%	95%	99%
Cement Kilns				
Baseline	1E-09 *(1E-09, 2E-09)*	6E-09 *(5E-09, 8E-09)*	1E-08 *(1E-08 1E-08)*	4E-08 *(3E-08, 6E-08)*
Final Standards	1E-09 *(1E-09, 1E-09)*	3E-09 *(3E-09, 4E-09)*	5E-09 *(4E-09, 6E-09)*	2E-08 *(1E-08, 2E-08)*
Area Source Cement Kilns				
Baseline	1E-08 *(2E-09, 2E-08)*	9E-08 *(8E-08, 9E-08)*	1E-07 *(1E-07, 1E-07)*	3E-07 *(2E-07, 3E-07)*
Final Standards	5E-09 *(2E-09, 1E-08)*	4E-08 *(3E-08, 4E-08)*	5E-08 *(4E-08, 5E-08)*	1E-07 *(7E-08, 1E-07)*
Lightweight Aggregate Kilns				
Baseline	4E-09	2E-08	2E-08	3E-08
Final Standards	2E-09	8E-09	1E-08	2E-08
All Incinerators				
Baseline	3E-10 *(1E-10, 1E-09)*	2E-08 *(3E-09, 3E-08)*	4E-08 *(1E-08, 7E-08)*	2E-07 [c]
Final Standards	2E-10 *(1E-10, 5E-10)*	3E-09 *(1E-09, 7E-09)*	8E-09 *(3E-09, 1E-08)*	2E-08 *(9E-09, 3E-08)*
Area Source Incinerators				
Baseline	7E-10 *(5E-11, 2E-09)*	2E-08 *(1E-08, 2E-08)*	3E-08 *(2E-08, 3E-08)*	6E-08 *(5E-08, 8E-08)*
Final Standards	5E-10 *(5E-11, 2E-09)*	9E-09 *(8E-09, 1E-08)*	2E-08 *(1E-08, 2E-08)*	3E-08 *(2E-08, 4E-08)*
Commercial Incinerators				
Baseline	1E-09 *(7E-10, 2E-09)*	1E-08 *(1E-08, 2E-08)*	3E-08 *(2E-08, 3E-08)*	6E-08 *(5E-08, 8E-08)*
Final Standards	5E-10 *(3E-10, 1E-09)*	9E-09 *(8E-09, 9E-09)*	2E-08 *(9E-09, 2E-08)*	3E-08 *(2E-08, 4E-08)*
Large On-site Incinerators				
Baseline	1E-08 *(2E-09, 2E-08)*	7E-08 *(3E-08, 1E-07)*	1E-07 *(5E-08, 3E-07)*	4E-07 [c]
Final Standards	2E-09 *(1E-09, 3E-09)*	1E-08 *(6E-09, 2E-08)*	2E-08 *(8E-09, 2E-08)*	6E-08 *(2E-08, 6E-08)*
Small On-site Incinerators				
Baseline	1E-10 *(1E-10, 2E-10)*	1E-09 *(3E-10, 2E-09)*	2E-09 *(7E-10, 3E-09)*	6E-09 *(2E-09, 9E-09)*
Final Standards	1E-10 *(1E-10, 2E-10)*	7E-10 *(3E-10, 1E-09)*	1E-09 *(5E-10, 2E-09)*	3E-09 *(2E-09, 5E-09)*
Waste Heat Boilers				
Baseline	6E-10 *(4E-10, 2E-09)*	4E-09 *(1E-09, 9E-09)*	1E-08 *(3E-09, 2E-08)*	4E-08 *(2E-08, 9E-08)*
Final Standards	4E-10 *(4E-10, 8E-10)*	1E-09 *(1E-09, 3E-09)*	3E-09 *(1E-09, 5E-09)*	1E-08 *(5E-09, 4E-08)*

[a] Includes cancer risk from incremental inhalation exposures to arsenic, beryllium, cadmium, chromium (VI), nickel, and TCDD-TEQs, assuming additivity.
[b] Percentiles do not reflect variability in the population of the duration of exposure.
[c] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

membranes, esophagus, and stomach, with nausea, vomiting, intense thirst, and diarrhea. Dermal contact with hydrogen chloride can cause burns, ulcerations, and scarring. Cases of gastritis, chronic bronchitis, dermatitis, and photosensitization have been reported among individuals exposed occupationally to hydrogen chloride (NLM, 1999).

Risks are expressed here in terms of an inhalation hazard quotient, which is defined as the ratio of the modeled air concentration to EPA's RfC. Inhalation hazard quotients are the same regardless of age. The receptor population with the highest inhalation hazard quotients is variable and depends on site-to-site differences in the location of farm and nonfarm households and differences in emissions.

Under the MACT standards, inhalation hazard quotients are projected to be at or below 0.01 for hydrogen chloride across all source categories.

Table 2-7 presents summary data for this category of risk results.

The distribution of hazard quotients reflects variability in individual exposures due to site-specific differences in emissions, location of exposure, and other factors. However, it does not reflect individual differences in activity patterns or breathing rates nor does it reflect temporal variations in exposure. This is because exposure factors used in deriving the RfC are fixed, and the RfC is intended to be protective of long-term, chronic exposures over a lifetime.[17] In addition, the same uncertainties related to indoor versus outdoor concentrations and atmospheric dispersion modeling discussed previously for inhalation carcinogens are applicable to hydrogen chloride. See Section 2.1.1.3 for a general discussion of air modeling uncertainty. However, modeled air concentrations are sufficiently below health benchmarks that these uncertainties may not have an appreciable effect on the overall conclusions.

2.1.2.7 Mercury. The recreational fisher is the receptor population with the highest exposure to mercury due to consumption of fish containing methylmercury. Exposures to other forms of mercury are quite low for all receptor populations. Although risk characterization for the recreational fisher included all constituents considered in the HWC risk analysis, methylmercury exposure resulting from the ingestion of fish has been identified as the only pathway of potential concern. Consequently, this pathway will be the focus of the risk characterization discussion for the recreational fisher.

For methylmercury, the most exposed population are those individuals who ingest home-caught fish, including both recreational fishers and subsistence fishers. Recreational fishers are discussed here (see Section 2.1.3 for a discussion of risks generated for the subsistence fisher). In the case of both recreational fishers and subsistence fishers, all family members are potentially exposed to methylmercury through consumption of the fish that have been caught.

Recreational fisher exposure to methylmercury was modeled assuming that fishing activity was restricted exclusively to the set of modeled waterbodies selected for a given study

[17] Although short-term exposures to HCl and Cl_2 resulting from routine releases can be significantly higher than long-term exposures, such exposures are unlikely to be high enough to pose a health concern. This is because the threshold for acute effects is quite high in comparison to that for chronic effects.

Table 2-7. Hazard Quotients for Incremental Exposures to Hydrogen Chloride for Adult Residents (with 90% Confidence Intervals)

Emissions	Percentile of the Cumulative Distribution (Population Weighted)			
	50%	**90%**	**95%**	**99%**
Cement Kilns				
Baseline	5E-04 *(3E-04, 7E-04)*	2E-03 *(2E-03, 2E-03)*	*2E-03 (2E-03, 3E-03)*	4E-03 [a]
Final Standards	4E-04 *(3E-04, 6E-04)*	2E-03 *(1E-03, 2E-03)*	2E-03 *(2E-03, 2E-03)*	4E-03 *(3E-03, 4E-03)*
Area Source Cement Kilns				
Baseline	3E-05 *(2E-05, 3E-05)*	1E-04 *(9E-05, 1E-04)*	1E-04 *(1E-04, 2E-04)*	3E-04 *(2E-04, 3E-04)*
Final Standards	3E-05 *(2E-05, 3E-05)*	1E-04 *(9E-05, 1E-04)*	1E-04 *(1E-04, 2E-04)*	3E-04 *(2E-04, 3E-04)*
Lightweight Aggregate Kilns				
Baseline	3E-03	1E-02	2E-02	5E-02
Floor	3E-03	1E-02	2E-02	5E-02
Final Standards	1E-03	3E-03	4E-03	8E-03
All Incinerators				
Baseline	1E-05 *(3E-06, 4E-05)*	2E-04 *(7E-05, 4E-04)*	5E-04 *(2E-04, 6E-04)*	1E-03 *(8E-04, 2E-03)*
Final Standards	1E-05 *(1E-06, 3E-05)*	2E-04 *(6E-05, 2E-04)*	3E-04 *(1E-04, 5E-04)*	9E-04 *(5E-04, 1E-03)*
Area Source Incinerators				
Baseline	5E-06 *(1E-06, 3E-05)*	7E-05 *(3E-05, 1E-04)*	1E-04 *(5E-05, 1E-04)*	3E-04 *(1E-04, 3E-04)*
Final Standards	5E-06 *(1E-06, 2E-05)*	4E-05 *(3E-05, 5E-05)*	7E-05 *(5E-05, 8E-05)*	1E-04 *(1E-04, 2E-04)*
Commercial Incinerators				
Baseline	3E-05 *(3E-06, 9E-05)*	5E-04 *(2E-04, 7E-04)*	9E-04 *(5E-04, 1E-03)*	2E-03 *(1E-03, 2E-03)*
Final Standards	3E-05 *(3E-06, 7E-05)*	4E-04 *(1E-04, 5E-04)*	7E-04 *(3E-04, 9E-04)*	2E-03 *(8E-04, 2E-03)*
Large On-site Incinerators				
Baseline	5E-05 *(3E-05, 1E-04)*	5E-04 *(2E-04, 7E-04)*	9E-04 *(4E-04, 1E-03)*	4E-03 [a]
Final Standards	5E-05 *(3E-05, 1E-04)*	5E-04 *(2E-04, 5E-04)*	6E-04 *(3E-04, 1E-03)*	2E-03 [a]
Small On-site Incinerators				
Baseline	3E-06 *(3E-06, 3E-05)*	8E-05 *(1E-05, 2E-04)*	2E-04 *(3E-05, 5E-04)*	8E-04 *(1E-04, 2E-03)*
Final Standards	2E-06 *(1E-06, 2E-05)*	5E-05 *(8E-06, 1E-04)*	1E-04 *(3E-05, 2E-04)*	3E-04 [a]

[a] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

area. It was further assumed that recreational fishers distribute their fishing activity among these waterbodies based on the surface area of each waterbody (i.e., they do not fish exclusively at a specific waterbody).[18]

Consumption of fish is the risk-driving exposure pathway for methylmercury because methylmercury is readily formed in aquatic ecosystems and bioaccumulates in fish. Children have the highest exposures due to their higher consumption of fish relative to body weight compared to adults. Risks from exposures to methylmercury are expressed here in terms of an ingestion hazard quotient, which is defined as the ratio of the modeled average daily dose to EPA's RfD. As discussed in Section 2.1.1.5, risk for both nonmaternal adults and children is evaluated using an RfD developed to be protective of in utero exposure. Although this approach provides an additional margin of safety for the nonmaternal adult and child scenarios, it also introduces uncertainty into the analysis. A more complete discussion of uncertainty associated with the methylmercury RfD is provided in Section 2.1.1.5.

Although recreational fishers are an enumerated population, there are no ready means by which to determine at which waterbodies they fish. An assumption of uniform population distribution across sectors was used in assessing exposure for the recreational fisher (i.e., a single individual from each receptor population was assumed to reside in each of the 16 sectors within a given study area; see Section 4.4.1.2 for further details).

Table 2-8 presents risk results for the child of the recreational fisher.

Risk results generated for the child of the recreational fisher (for the final rule) project high-end ingestion hazard quotients for the cement kiln category of less than 1.0. High-end ingestion hazard quotients for the area source cement kiln category range up to 1.0 (95th percentile), with high-end risk reductions on the order of 10 percent projected to occur for this combustor category under MACT standard implementation (i.e., 1.0 for 95th percentile baseline to 9E-01 for 95th percentile MACT standard). A central tendency hazard quotient of 2E-01 (50th percentile) is projected for the area source cement kiln category at baseline.

High-end risk results for lightweight aggregate kilns (LWAKs) at baseline range up to 4E-02 (95th percentile), with no results exceeding a hazard quotient of 1.0.

High-end baseline ingestion hazard quotients of less than 1.0 are projected for all incinerator categories with risks ranging from 9E-03 (99th percentile for large on-site incinerators) to 2E-02 (99th percentile for commercial incinerators). Central tendency baseline ingestion hazard quotients for all incinerator categories are below 8E-05 (50th percentile commercial incinerators).

Table 2-9 presents risk results for the adult recreational fisher.

[18] Ideally, detailed information on the fishing activities of individual fishers, including the degree to which fishing activity is distributed among different waterbodies and the size of the catch taken from individual locations, would be used to better assess exposures from consumption of recreationally caught fish.

Table 2-8. Hazard Quotients for Incremental Exposures to Methylmercury for 0- to 5-yr-old Children of Recreational Fishers (with 90% Confidence Intervals)

Emissions	Percentile of the Cumulative Distribution[a]			
	50%	90%	95%	99%
Cement Kilns				
Baseline	1E-02 *(7E-03, 2E-02)*	2E-01 *(1E-01, 3E-01)*	3E-01 *(2E-01, 5E-01)*	8E-01 [a]
Final Standards	1E-02 *(6E-03, 2E-02)*	2E-01 *(1E-01, 2E-01)*	3E-01 *(2E-01, 3E-01)*	6E-01 [a]
Area Source Cement Kilns				
Baseline	2E-01 *(1E-01, 2E-01)*	8E-01 *(6E-01, 9E-01)*	1 *(9E-01, 1)*	[a]
Final Standards	1E-01 *(1E-01, 2E-01)*	6E-01 *(5E-01, 7E-01)*	9E-01 *(7E-01, 1)*	[a]
Lightweight Aggregate Kilns				
Baseline	2E-03	2E-02	4E-02	[a]
Final Standards	2E-03	2E-02	3E-02	[a]
All Incinerators				
Baseline	6E-06 *(2E-06, 2E-05)*	2E-03 *(6E-04, 3E-03)*	4E-03 *(2E-03, 8E-03)*	2E-02 [a]
Final Standards	6E-06 *(2E-06, 2E-05)*	1E-03 *(5E-04, 3E-03)*	4E-03 *(1E-03, 8E-03)*	2E-02 [a]
Area Source Incinerators				
Baseline	6E-06 *(3E-06, 2E-05)*	1E-03 *(6E-05, 3E-03)*	3E-03 *(6E-04, 6E-03)*	1E-02 *(2E-03, 2E-02)*
Final Standards	6E-06 *(3E-06, 2E-05)*	9E-04 *(5E-05, 3E-03)*	3E-03 *(2E-04, 6E-03)*	1E-02 *(1E-03, 2E-02)*
Commercial Incinerators				
Baseline	8E-05 *(6E-06, 4E-04)*	4E-03 *(2E-03, 7E-03)*	8E-03 *(4E-03, 1E-02)*	2E-02 [a]
Final Standards	4E-05 *(4E-06, 2E-04)*	3E-03 *(1E-03, 5E-03)*	5E-03 *(2E-03, 8E-03)*	1E-02 [a]
Large On-site Incinerators				
Baseline	8E-07 *(1E-07, 2E-05)*	7E-04 *(1E-04, 2E-03)*	2E-03 *(3E-04, 5E-03)*	9E-03 [a]
Final Standards	2E-07 *(5E-08, 2E-05)*	5E-04 *(1E-04, 2E-03)*	2E-03 *(3E-04, 5E-03)*	8E-03 [a]
Small On-site Incinerators				
Baseline	6E-06 *(2E-06, 2E-05)*	1E-03 *(2E-04, 4E-03)*	4E-03 *(7E-04, 1E-02)*	2E-02 [a]
Final Standards	5E-06 *(2E-06, 2E-05)*	1E-03 *(2E-04, 4E-03)*	4E-03 *(6E-04, 1E-02)*	2E-02 [a]

[a] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

Table 2-9. Hazard Quotients for Incremental Exposures to Methylmercury for Adult Recreational Fishers (with 90% Confidence Intervals)

Emissions	Percentile of the Cumulative Distribution[a]			
	50%	90%	95%	99%
Cement Kilns				
Baseline	5E-03 *(3E-03, 1E-02)*	9E-02 *(6E-02, 1E-01)*	2E-01 *(1E-01, 2E-01)*	4E-01 a
Final Standards	5E-03 *(3E-03, 9E-03)*	6E-02 *(5E-02, 8E-02)*	1E-01 *(8E-02, 1E-01)*	2E-01 a
Area Source Cement Kilns				
Baseline	7E-02 *(4E-02, 1E-01)*	4E-01 *(3E-01, 5E-01)*	6E-01 *(5E-01, 7E-01)*	a
Final Standards	5E-02 *(4E-02, 6E-02)*	2E-01 *(2E-01, 2E-01)*	3E-01 *(2E-01, 3E-01)*	a
Lightweight Aggregate Kilns				
Baseline	8E-04 a	1E-02 a	2E-02 a	a
Final Standards	6E-04 a	7E-03 a	1E-02 a	a
All Incinerators				
Baseline	3E-06 *(8E-07, 7E-06)*	7E-04 *(3E-04, 2E-03)*	2E-03 *(8E-04, 4E-03)*	9E-03 a
Final Standards	3E-06 *(7E-07, 7E-06)*	5E-04 *(2E-04, 1E-03)*	2E-03 *(5E-04, 4E-03)*	8E-03 a
Area Source Incinerators				
Baseline	3E-06 *(9E-07, 9E-06)*	6E-04 *(4E-05, 1E-03)*	1E-03 *(3E-04, 2E-03)*	4E-03 *(1E-03, 7E-03)*
Final Standards	3E-06 *(9E-07, 9E-06)*	4E-04 *(3E-05, 1E-03)*	1E-03 *(1E-04, 2E-03)*	4E-03 *(6E-04, 7E-03)*
Commercial Incinerators				
Baseline	4E-05 *(2E-06, 2E-04)*	2E-03 *(8E-04, 3E-03)*	3E-03 *(2E-03, 4E-03)*	8E-03 a
Final Standards	2E-05 *(2E-06, 9E-05)*	1E-03 *(4E-04, 2E-03)*	2E-03 *(8E-04, 3E-03)*	5E-03 a
Large On-site Incinerators				
Baseline	4E-08 *(6E-09, 8E-06)*	3E-04 *(5E-05, 1E-03)*	1E-03 *(1E-04, 2E-03)*	5E-03 a
Final Standards	1E-08 *(3E-09, 8E-06)*	2E-04 *(5E-05, 9E-04)*	9E-04 *(1E-04, 2E-03)*	4E-03 a
Small On-site Incinerators				
Baseline	3E-06 *(9E-07, 9E-06)*	6E-04 *(8E-05, 2E-03)*	2E-03 *(4E-04, 6E-03)*	1E-02 a
Final Standards	2E-06 *(8E-07, 8E-06)*	5E-04 *(7E-05, 2E-03)*	2E-03 *(3E-04, 6E-03)*	1E-02 a

[a] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

Risk results generated for the adult recreational fisher (for the final rule) project high-end ingestion hazard quotients for the cement kiln category at baseline of less than 1.0 (i.e., 4E-01 for the 99th percentile). High-end ingestion hazard quotients for the area source cement kiln category range up to 6E-01 (95th percentile) at baseline, with no results exceeding a hazard quotient of 1.0.

High-end risk results for lightweight aggregate kilns at baseline range up to 2E-02 (95th percentile) with no results exceeding a hazard quotient of 1.0.

High-end baseline ingestion hazard quotients of less than 1.0 are projected for all incinerator categories with risks ranging from 4E-03 (99th percentile for area source incinerators) to 1E-02 (99th percentile for small on-site incinerators). Central tendency baseline ingestion hazard quotients for all incinerator categories are below 4E-05 (50th percentile commercial incinerators).

The distribution of ingestion hazard quotients reflects variability in individual exposures due to site-specific differences in emissions, location of waterbodies, and other factors, as well as differences among individuals in the amount of fish consumed. Other factors, such as waterbody-specific differences in the extent of methylation of inorganic mercury and the age and species of fish consumed are not reflected in the risk distribution. Variability among waterbodies in bioaccumulation of methylmercury in fish is substantial and likely to be a significant factor in the overall uncertainty of the results. However, it is unclear whether such factors would have a large effect on the distribution given the high degree of variability that is attributable to the factors that are considered.

The assumptions used in characterizing exposure for the recreational fisher introduce significant uncertainty into the risk estimates generated for this receptor population (see Section 2.1.1.4). Restricting fishing activity only to modeled waterbodies may result in conservative exposure estimates because recreational fishers could also use waterbodies that are impacted to a lesser extent than the modeled waterbodies (i.e., waterbodies located outside of the study area or waterbodies located in less-impacted portions of the study area). The number of recreational fishers who fit this scenario (i.e., fish exclusively at modeled waterbodies) may be quite small (and could possibly be zero). The assumption that recreational fishing activity is distributed between modeled waterbodies based on surface area weighting and does not involve focused activity at a specific waterbody may result in either an over- or underestimation of exposure for a specific study area since risks associated with specific waterbodies will be “averaged out” (i.e., the use of a surface-area-weighted averaging for modeling fishing activity will result in the “averaging out” of both upper and lower-bound waterbodies). Taken together, the overall impact of these two assumptions on risk estimates generated for the recreational fisher is uncertain although it is expected that risks may be somewhat conservative.

In addition to the uncertainty introduced through assumptions regarding exposure, risk estimates for the recreational fisher are also impacted by uncertainties associated with modeling mercury fate/transport through key environmental compartments (see Section 2.1.1.3).

2.1.3 Individual-Level Risks for Subsistence Receptors

Risks associated with subsistence activities were assessed because persons involved in subsistence activities (i.e., subsistence farming and subsistence fishing) may be more highly exposed to modeled constituents than the general population. In modeling subsistence receptors, subsistence farmers were assumed to obtain essentially all of their dietary intake from home-produced foods, including meats, milk, poultry, fish, and fruits and vegetables, and subsistence fishers were assumed to obtain essentially all of their dietary intake of fish from self-caught fish. Data on the mean rate of consumption of home-produced foods in households that consume home-produced foods were used to estimate the average daily intakes from subsistence farming. For subsistence fishing, data were used on the mean rate of fish consumption among Native American tribes that rely on fish for a major part of their dietary intake (see Section 6.2).

Individual risk characterization for the subsistence receptors resembles that of the enumerated receptors in that cumulative risk distributions (presented as frequency distributions for the subsistence receptors) are used to identify specific percentiles of interest. However, because it is not possible to characterize the location and density of subsistence receptors, the assumption is made that they are evenly distributed across the sectors constituting HWC study areas (i.e., a single subsistence individual is modeled for each sector). Therefore, the frequency distributions used to characterize individual risk for the subsistence receptors must be interpreted in relation to the frequency of the modeled scenarios and not the likelihood of such exposures actually occurring.[19] The inability to characterize the distribution of subsistence receptors across HWC study areas introduces uncertainty into the characterization of risk for these receptors since actual patterns of subsistence location and activity are not reflected in either exposure assessment or the risk estimates that are generated.

2.1.3.1 <u>Dioxins/Furans (Cancer Risk—Subsistence Farmer)</u>. The subsistence farmer is exposed to dioxins/furans contained in the home-produced agricultural commodities that this receptor produces (i.e., beef, pork, milk, and fruits/vegetables). These agricultural commodities can concentrate dioxins/furans through a number of mechanisms including: (1) vapor uptake/ direct deposition to the locally grown feed consumed by livestock, (2) incidental ingestion of soil by livestock, and (3) vapor uptake/root uptake/direct deposition to fruits/vegetables. The subsistence farmer can also be exposed to dioxins/furans through incidental soil ingestion. Children of the subsistence farmer will have the greatest exposure to dioxins/furans due to their high consumption rate for these agricultural commodities on a per unit body weight basis relative to adults.

Carcinogenic risk for dioxins/furans is expressed as the lifetime excess cancer risk that results from exposure to these constituents. In generating these estimates, a dioxin-TEQ approach is used (see Section 2.1.1.1 for an overview of the dioxin-TEQ approach and the derivation of lifetime excess cancer risk estimates).

[19] Moreover, the modeled subsistence scenarios cannot be considered equally probable because the sectors in which farms were located are of unequal area, being much smaller closer to a facility and much larger farther away, and because any particular sector may be more or less likely to support farming activities depending on soils, precipitation, existing land uses, and other conditions. Similarly, the modeled waterbodies may be more or less likely to support intensive fishing activity depending on their size, productivity, and other characteristics.

Table 2-10 presents summary data for this category of risk results.

Risk results generated for the final rule (for the child of the subsistence farmer) project high-end lifetime excess cancer risks for the cement kiln category of 2E-05 (0.01 cumulative frequency at baseline). No reductions in risk are projected under implementation of the MACT standards for this category.

Baseline high-end risks for LWAKs are estimated to range from 2E-05 to 3E-05 (0.10 and 0.05 cumulative frequencies, respectively). High-end risk reductions on the order of 80 to 85 percent are projected to occur for this combustor category with MACT standard implementation (e.g., 3E-05 for 0.05 cumulative frequency at baseline to 5E-06 with MACT standard implementation). Central tendency risks for LWAKs at baseline are estimated to be 4E-06 (0.50 cumulative frequency).

The incinerator categories have high-end risk estimates at baseline ranging from 1E-06 to 8E-05 (baseline 0.10 cumulative frequency for OINC-S and baseline 0.01 cumulative frequency for WHB, respectively). Projected high-end risk reductions with MACT standard implementation for each of the incinerator categories are approximately an order of magnitude.

To summarize, under the final MACT standards, lifetime excess cancer risks from dioxin exposures associated with subsistence farming are projected to be below 1 in 100,000 for all categories of combustors, with the exception of cement kilns at the lowest frequency of occurrence. The lifetime excess cancer risk for cement kilns is 2 in 100,000 at a frequency of 0.01. This indicates that only 1 in 100 sectors is expected to have risks of this magnitude or greater, assuming that subsistence farms are located in all sectors at all hazardous waste burning cement kilns. However, because the sectors increase in size with increasing distance, the probability that a subsistence farmer would be exposed to this level of risk is less than 1 percent.

The distributions of lifetime excess cancer risk generated for dioxin/furan exposure for the subsistence farmer are subject to a high degree of uncertainty, primarily due to the lack of information on the location of subsistence farms (or even the occurrence of subsistence farms within the study area of a given facility) and the assumption that individuals engaged in subsistence farming obtain essentially their entire dietary intake from home-produced foods (see Section 2.1.1.4).

2.1.3.2 <u>Dioxins/Furans (Noncancer—Subsistence Farmer)</u>. The subsistence farmer was also assessed for noncancer effects resulting from exposure to dioxins/furans. The subsistence farmer is exposed to these constituents through the ingestion of a broad range of home-produced agricultural commodities. However, unlike the commercial farmer receptors, it is not possible to characterize either the population density or location for the subsistence receptor, which introduces significant uncertainty into exposure assessment for this receptor. The children of the subsistence farmer will have the greatest exposure to dioxins/furans due to their high consumption rate for these agricultural commodities on a per unit body weight basis relative to adults.

The incremental MOE approach is used to assess potential noncancer effects resulting from exposure to dioxins/furans (for an overview of this methodology, see Section 2.1.2.2).

Table 2-10. Lifetime Excess Cancer Risk for 0- to 5-Yr-Old Children from Incremental Exposures to Dioxins from Subsistence Farming (with 90% Confidence Intervals)[a]

Emissions	Cumulative Frequency (Greater Than)[b]			
	0.50	**0.10**	**0.05**	**0.01**
Cement Kilns				
Baseline	8E-07 *(5E-07, 1E-06)*	6E-06 *(5E-06, 7E-06)*	9E-06 *(8E-06, 1E-05)*	2E-05 *(2E-05, 2E-05)*
Final Standards	7E-07 *(5E-07, 9E-07)*	4E-06 *(3E-06, 5E-06)*	7E-06 *(6E-06, 7E-06)*	2E-05 *(2E-05, 2E-05)*
Area Source Cement Kilns				
Baseline	1E-07 (6E-08, 1E-06)	1E-05 (7E-06, 2E-05)	[c] [c]	[c] [c]
Final Standards	1E-07 (6E-08, 1E-06)	1E-05 (7E-06, 2E-05)	[c] [c]	[c] [c]
Lightweight Aggregate Kilns				
Baseline	4E-06	2E-05	3E-05	[c]
Floor	4E-06	2E-05	3E-05	[c]
Final Standards	9E-07	4E-06	5E-06	[c]
All Incinerators				
Baseline	6E-08 *(3E-08, 9E-08)*	5E-06 *(2E-06, 8E-06)*	1E-05 *(8E-06, 2E-05)*	4E-05 *(3E-05, 5E-05)*
Final Standards	5E-08 *(3E-08, 9E-08)*	1E-06 *(9E-07, 1E-06)*	2E-06 *(1E-06, 2E-06)*	4E-06 *(4E-06, 5E-06)*
Area Source Incinerators				
Baseline	2E-08 *(1E-08, 2E-07)*	9E-06 *(9E-07, 1E-05)*	2E-05 *(4E-06, 3E-05)*	4E-05 [c]
Final Standards	2E-08 *(1E-08, 1E-07)*	1E-06 *(3E-07, 2E-06)*	2E-06 *(9E-07, 3E-06)*	5E-06 [c]
Commercial Incinerators				
Baseline	1E-06 *(6E-07, 3E-06)*	2E-05 *(1E-05, 3E-05)*	3E-05 *(2E-05, 4E-05)*	6E-05 [c]
Final Standards	3E-07 *(2E-07, 4E-07)*	2E-06 *(2E-06, 3E-06)*	4E-06 *(3E-06, 4E-06)*	6E-06 *(5E-06, 7E-06)*
Large On-site Incinerators				
Baseline	1E-07 *(7E-08, 2E-07)*	3E-06 *(1E-06, 9E-06)*	9E-06 *(2E-06, 2E-05)*	3E-05 [c]
Final Standards	1E-07 *(7E-08, 2E-07)*	1E-06 *(9E-07, 2E-06)*	2E-06 *(1E-06, 3E-06)*	4E-06 *(3E-06, 5E-06)*
Small On-site Incinerators				
Baseline	1E-08 *(8E-09, 4E-08)*	1E-06 *(4E-07, 4E-06)*	7E-06 *(1E-06, 1E-05)*	3E-05 *(1E-05, 7E-05)*
Final Standards	1E-08 *(8E-09, 4E-08)*	5E-07 *(2E-07, 7E-07)*	9E-07 *(5E-07, 1E-06)*	3E-06 *(1E-06, 4E-06)*
Waste Heat Boilers				
Baseline	3E-06 *(2E-06, 5E-06)*	3E-05 *(2E-05, 3E-05)*	4E-05 *(3E-05, 5E-05)*	8E-05 *(4E-05, 1E-04)*
Floor	3E-06 *(2E-06, 5E-06)*	3E-05 *(2E-05, 3E-05)*	4E-05 *(3E-05, 5E-05)*	8E-05 *(4E-05, 1E-04)*
Final Standards	4E-07 *(3E-07, 5E-07)*	2E-06 *(2E-06, 3E-06)*	3E-06 *(3E-06, 4E-06)*	5E-06 *(4E-06, 6E-06)*

[a] Includes cancer risk from incremental exposures to 2,3,7,8-chlorine substituted dibenzo(*p*)dioxins and dibenzofurans, expressed as TCDD-TEQs.
[b] Equal to 1 minus the cumulative frequency less than the indicated frequency.
[c] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

Table 2-11 presents summary data for this category of risk results.

High-end incremental MOE levels are projected to range from 7E-02 to 4 (baseline 0.10 cumulative frequency for OINC-S and baseline 0.01 cumulative frequency for WHB, respectively). Central tendency incremental MOE levels for all combustor categories are below 1.0.[20]

The cement kiln category has baseline incremental MOE levels below 1 for all cumulative frequencies. A 30 percent reduction in high-end incremental MOE levels is projected for the cement kiln with MACT standard implementation.

A high-end incremental MOE level of 1.0 is projected for the LWAK category at baseline (0.05 cumulative frequency). A 80 percent reduction in high-end risk is projected for this combustor category with MACT standard implementation.

High-end incremental MOE levels for the incinerator categories range from 7E-02 to 4 (baseline 0.10 cumulative frequency for OINC-S and baseline 0.01 cumulative frequency for WHB, respectively). Each of the incinerator categories is projected to have reductions in high-end incremental MOE levels of approximately an order of magnitude with MACT standard implementation.

To summarize, the incremental MOE is projected to be reduced to 0.1 or below for incinerators under the final MACT standards except at the lowest frequency of occurrence (i.e., 0.01 or 1 percent, for which an incremental MOE of 0.2 is projected). However, the incremental MOEs for cement kilns and lightweight aggregate kilns are projected to remain above 0.1 at a frequency of 10 percent or greater (ranging up to 0.7 at a frequency of 1 percent). This indicates that more than 1 in 10 sectors are expected to have risks associated with noncancer effects from these sources that are within an order of magnitude of any (unknown) risks that may be attributable to background exposures. However, for the reasons stated previously, the probability that a subsistence farmer would be exposed to this level of risk is probably lower than indicated by the number of sectors.

The same sources of uncertainty that impact the dioxin-TEQ cancer estimates generated for the subsistence farmer impact the incremental MOE estimates discussed here (see Section 2.1.3.1). In addition, the use of the incremental MOE approach introduces additional uncertainty into the analysis (see Section 2.1.2.2).

2.1.3.3 <u>Methylmercury and Dioxins/Furans (Subsistence Fisher)</u>. In addition to assessing risk resulting from the ingestion of fish containing methylmercury for the recreational fisher, this analysis also evaluated this pathway for the subsistence fisher. The subsistence fisher receptor represents those individuals who engage in subsistence fishing activity and consequently obtain a significant portion of their dietary intake from home-caught fish. The exposure parameters used in modeling this receptor (e.g., fish ingestion rates and exposure durations)

[20] It was not possible to generate high-end risk results for several of the combustor categories because of a small sample size for the sector-level risk data and/or a small spread in the sector-level risk results.

Table 2-11. Incremental Margin of Exposure to Dioxins for 0- to 5-Yr-Old Children from Subsistence Farming[a, b] (with 90% Confidence Intervals)

Emissions	Cumulative Frequency (Greater Than)[c]			
	0.50	**0.10**	**0.05**	**0.01**
Cement Kilns				
Baseline	4E-02 *(2E-02, 6E-02)*	3E-01 *(2E-01, 3E-01)*	5E-01 *(4E-01, 6E-01)*	9E-01 *(9E-01, 9E-01)*
Final Standards	3E-02 *(2E-02, 4E-02)*	2E-01 *(1E-01, 2E-01)*	3E-01 *(2E-01, 3E-01)*	7E-01 *(7E-01, 7E-01)*
Area Source Cement Kilns				
Baseline	8E-03 *(3E-03, 9E-02)*	7E-01 *(3E-01, 9E-01)*	[d] [d]	[d] [d]
Final Standards	8E-03 *(3E-03, 9E-02)*	7E-01 *(3E-01, 9E-01)*	[d] [d]	[d] [d]
Lightweight Aggregate Kilns				
Baseline	2E-01	9E-01	1	[d] [d]
Floor	2E-01	9E-01	1	[d] [d]
Final Standards	4E-02	2E-01	2E-01	[d] [d]
All Incinerators				
Baseline	3E-03 *(1E-03, 5E-03)*	2E-01 *(1E-01, 4E-01)*	7E-01 *(4E-01, 9E-01)*	2 *(1, 2)*
Final Standards	2E-03 (1E-03, 4E-03)	6E-02 *(4E-02, 7E-02)*	9E-02 *(8E-02, 1E-01)*	2E-01 *(2E-01, 2E-01)*
Area Source Incinerators				
Baseline	1E-03 *(5E-04, 1E-02)*	4E-01 *(4E-02, 9E-01)*	9E-01 *(2E-01, 1)*	2 [d]
Final Standards	1E-03 *(5E-04, 7E-03)*	5E-02 *(1E-02, 9E-02)*	1E-01 *(4E-02, 1E-01)*	2E-01 [d]
Commercial Incinerators				
Baseline	9E-02 *(3E-02, 1E-01)*	9E-01 *(9E-01, 1)*	1 *(1, 2)*	3 [d]
Final Standards	1E-02 *(9E-03, 2E-02)*	1E-01 *(9E-02, 1E-01)*	2E-01 *(1E-01, 2E-01)*	3E-01 [d]
Large On-site Incinerators				
Baseline	7E-03 *(3E-03, 1E-02)*	1E-01 *(6E-02, 4E-01)*	4E-01 *(1E-01, 9E-01)*	[d] [d]
Final Standards	6E-03 *(3E-03, 9E-03)*	8E-02 *(5E-02, 9E-02)*	1E-01 *(8E-02, 1E-01)*	2E-01 *(1E-01, 3E-01)*
Small On-site Incinerators				
Baseline	8E-04 *(4E-04, 2E-03)*	7E-02 *(2E-02, 2E-01)*	3E-01 *(6E-02, 7E-01)*	1 *(7E-01, 3)*
Final Standards	8E-04 *(4E-04, 1E-03)*	2E-02 *(1E-02, 3E-02)*	5E-02 *(2E-02, 7E-02)*	1E-01 *(8E-02, 2E-01)*
Waste Heat Boilers				
Baseline	1E-01 *(9E-02, 2E-01)*	1 *(9E-01, 1)*	2 *(1, 2)*	4 *(2, 5)*
Floor	1E-01 *(9E-02, 2E-01)*	1 *(9E-01, 1)*	2 *(1, 2)*	4 *(2, 5)*
Final Standards	2E-02 *(1E-02, 2E-02)*	1E-01 *(9E-02, 1E-01)*	1E-01 *(1E-01, 2E-01)*	2E-01 *(2E-01, 3E-01)*

[a] Includes incremental exposures to 2,3,7,8-chlorine-substituted dibenzo(*p*)dioxins and dibenzofurans, expressed as TCDD-TEQs.
[b] Relative to an average background exposure to the general population of 1.5 pg/kg-d.
[c] Equal to 1 minus the cumulative frequency less than the indicated frequency.
[d] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

reflect fish ingestion rates that are characteristic of subsistence populations (i.e., Native Americans in the Pacific Northwest).

Although risk characterization for the subsistence fisher included all constituents considered in the HWC risk analysis, methylmercury and dioxin exposure resulting from the ingestion of fish have been identified as the only pathways of potential concern. Consequently, these pathways will be the focus of the risk characterization discussion for the subsistence fisher.

Individuals engaged in subsistence fishing were assumed to obtain all the fish they consume from a single waterbody. To the extent that individuals may fish at more than one waterbody, the effect of this assumption may be to exaggerate the risk from waterbodies having relatively high modeled fish concentrations. The results generated for the subsistence fisher are summarized in the form of frequency distributions of individual risk. The distributions must be interpreted in relation to the frequency of the modeled scenarios and not the likelihood of such exposures actually occurring. Moreover, the modeled scenarios cannot be considered equally probable because the modeled waterbodies may be more or less likely to support intensive fishing activity depending on their size, productivity, and other characteristics.

With regard to methylmercury, as explained in Section 2.1.2.7, fish ingestion is considered to pose the greatest risk for methylmercury because methylmercury is readily formed in aquatic ecosystems and bioaccumulates in fish. Dioxins/furans that are deposited to watersheds and directly to waterbodies can concentrate in aquatic systems resulting in bioaccumulation within fish. Children have the highest exposures to both methylmercury and dioxins/furans due to their higher consumption of fish relative to body weight compared to adults.

Risks from exposures to methylmercury are expressed here in terms of an ingestion hazard quotient, which is defined as the ratio of the modeled average daily dose to EPA's RfD. Although the RfD was developed to be protective of exposures in utero, the RfD is applied here not just to maternal exposures but also to nonmaternal adult and childhood exposures based on the presumption that the RfD is protective of neurological and/or developmental effects in these populations as well. Tables 2-12 and 2-13 present summary data for this category of risk results for the child of the subsistence fisher and the adult subsistence fisher.

Risks from exposure to dioxins/furans are expressed both in terms of incremental lifetime excess cancer risk and incremental MOE, the latter being used to assess noncancer effects (see Sections 2.1.1.1 and 2.1.1.2, respectively). Table 2-14 presents summary data for this category of risk results.

Under the final MACT standards, ingestion hazard quotients for the ingestion of fish containing methylmercury by the child of the subsistence fisher and the adult subsistence fisher are projected to be below 1.0 for all combustor categories. High-end ingestion hazard quotients under MACT standard implementation for the child of the subsistence fisher ranged from 2E-03 (0.05 cumulative frequency for small on-site incinerators) to 6E-01 (0.05 cumulative frequency for cement kilns). High-end ingestion hazard quotients under MACT standard implementation for the adult subsistence fisher ranged from 1E-02 (0.05 cumulative frequency for small on-site incinerators) to 3E-01 (0.05 cumulative frequency for cement kilns).

Table 2-12. Hazard Quotients for 0- to 5-Yr-Old Children from Incremental Exposures to Methylmercury from Subsistence Fishing (with 90% Confidence Intervals)

Emissions	Cumulative Frequency (Greater Than)[a]			
	0.50	**0.10**	**0.05**	**0.01**
Cement Kilns				
Baseline	5E-02 *(2E-02, 1E-01)*	5E-01 *(4E-01, 7E-01)*	8E-01 b	b
Final Standards	5E-02 *(2E-02, 1E-01)*	4E-01 *(4E-01, 4E-01)*	5E-01 *(4E-01, 6E-01)*	b
Area Source Cement Kilns				
Baseline	4E-01 *(2E-01, 5E-01)*	b	b	b
Final Standards	3E-01 *(2E-01, 3E-01)*	b	b	b
Lightweight Aggregate Kilns				
Baseline	8E-03	2E-01	b	b
Final Standards	7E-03	b	b	b
All Incinerators				
Baseline	2E-05 *(8E-06, 9E-05)*	7E-03 *(2E-03, 2E-02)*	4E-02 *(9E-03, 8E-02)*	4E-01 *(8E-02, 5E-01)*
Final Standards	2E-05 *(8E-06, 7E-05)*	7E-03 *(1E-03, 2E-02)*	4E-02 *(8E-03, 7E-02)*	b
Area Source Incinerators				
Baseline	2E-05 *(1E-05, 6E-05)*	6E-03 *(1E-04, 4E-02)*	3E-02 *(2E-03, 8E-02)*	b
Final Standards	2E-05 *(7E-06, 6E-05)*	6E-03 *(1E-04, 4E-02)*	3E-02 *(5E-04, 8E-02)*	b
Commercial Incinerators				
Baseline	9E-04 *(8E-05, 2E-03)*	3E-02 *(7E-03, 7E-02)*	7E-02 b	b
Final Standards	3E-04 *(9E-05, 9E-04)*	1E-02 *(3E-03, 6E-02)*	6E-02 b	b
Large On-site Incinerators				
Baseline	1E-05 *(9E-07, 1E-04)*	2E-03 *(9E-04, 6E-02)*	5E-02 *(2E-03, 2E-01)*	b
Final Standards	6E-06 *(4E-07, 1E-04)*	2E-03 *(8E-04, 5E-02)*	5E-02 *(2E-03, 1E-01)*	b
Small On-site Incinerators				
Baseline	1E-05 *(5E-06, 6E-05)*	7E-03 *(5E-04, 2E-02)*	2E-02 *(2E-03, 4E-02)*	b
Final Standards	1E-05 *(5E-06, 6E-05)*	7E-03 *(5E-04, 2E-02)*	2E-02 *(2E-03, 4E-02)*	b

[a] Equal to 1 minus the cumulative frequency less than the indicated frequency.
[b] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

Table 2-13. Hazard Quotients for Incremental Exposures to Methylmercury for Adult Subsistence Fishers (with 90% Confidence Intervals)

Emissions	Cumulative Frequency (Greater Than)[a]			
	0.50	**0.10**	**0.05**	**0.01**
Cement Kilns				
Baseline	3E-02 *(1E-02, 9E-02)*	4E-01 *(3E-01, 5E-01)*	6E-01 *(4E-01, 7E-01)*	b
Final Standards	4E-02 *(1E-02, 9E-02)*	3E-01 *(3E-01, 3E-01)*	3E-01 *(3E-01, 4E-01)*	b
Area Source Cement Kilns				
Baseline	2E-01 *(2E-01, 3E-01)*	b	b	b
Final Standards	2E-01 *(2E-01, 3E-01)*	b	b	b
Lightweight Aggregate Kilns				
Baseline	6E-03 b	1E-01 b	b	b
Final Standards	5E-03 b	b	b	b
All Incinerators				
Baseline	2E-05 *(5E-06, 6E-05)*	5E-03 *(2E-03, 1E-02)*	3E-02 *(6E-03, 6E-02)*	3E-01 *(5E-02, 4E-01)*
Final Standards	2E-05 *(5E-06, 5E-05)*	4E-03 *(1E-03, 1E-02)*	2E-02 *(6E-03, 5E-02)*	2E-01 *(5E-02, 3E-01)*
Area Source Incinerators				
Baseline	2E-05 *(6E-06, 5E-05)*	4E-03 *(1E-04, 3E-02)*	2E-02 *(2E-03, 6E-02)*	b
Final Standards	2E-05 *(6E-06, 5E-05)*	4E-03 *(1E-04, 3E-02)*	2E-02 *(4E-04, 6E-02)*	b
Commercial Incinerators				
Baseline	7E-04 *(7E-05, 1E-03)*	2E-02 *(5E-03, 5E-02)*	5E-02 b	b
Final Standards	2E-04 *(6E-05, 8E-04)*	8E-03 *(2E-03, 4E-02)*	4E-02 b	b
Large On-site Incinerators				
Baseline	4E-06 *(6E-08, 7E-05)*	2E-03 *(6E-04, 4E-02)*	4E-02 *(1E-03, 2E-01)*	b
Final Standards	4E-06 *(2E-08, 7E-05)*	2E-03 *(6E-04, 3E-02)*	4E-02 *(1E-03, 7E-02)*	b
Small On-site Incinerators				
Baseline	1E-05 *(4E-06, 5E-05)*	4E-03 *(3E-04, 9E-03)*	1E-02 *(2E-03, 4E-02)*	b
Final Standards	1E-05 *(4E-06, 5E-05)*	4E-03 *(3E-04, 9E-03)*	1E-02 *(1E-03, 4E-02)*	b

[a] Equal to 1 minus the cumulative frequency less than the indicated frequency.
[b] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

Table 2-14. Lifetime Excess Cancer Risk from Incremental Exposure to Dioxins for 0- to 5-Year-Old Children of Subsistence Fishers (with 90% Confidence Intervals) [a]

Emissions	Cumulative Frequency (Greater Than)							
	0.50		0.10		0.05		0.01	
Cement Kilns								
Baseline	3E-07	*(1E-07, 6E-07)*	2E-06	*(2E-06, 3E-06)*	3E-06	*(3E-06, 4E-06)*	b	b
Final Standards	2E-07	*(1E-07, 5E-07)*	1E-06	*(1E-06, 2E-06)*	b	b	b	b
Area Source Cement Kiln								
Baseline	3E-08	*(1E-08, 2E-07)*	b	b	b	b	b	b
Final Standards	3E-08	*(1E-08, 2E-07)*	b	b	b	b	b	b
Lightweight Aggregate Kilns								
Baseline	3E-07		2E-06		b		b	
Final Standards	8E-08		5E-07		b		b	
All Incinerators								
Baseline	5E-09	*(3E-09, 7E-09)*	5E-07	*(3E-07, 8E-07)*	9E-07	*(8E-07, 1E-06)*	2E-06	*(2E-06, 3E-06)*
Final Standards	4E-09	*(2E-09, 6E-09)*	1E-07	*(9E-08, 2E-07)*	4E-07	*(2E-07, 6E-07)*	1E-06	b
Area Source Incinerators								
Baseline	6E-09	*(5E-09, 2E-08)*	2E-07	*(3E-08, 4E-07)*	4E-07	*(5E-08, 7E-07)*	b	b
Final Standards	6E-09	*(5E-09, 1E-08)*	5E-08	*(3E-08, 7E-08)*	8E-08	*(3E-08, 1E-07)*	b	b
Commercial Incinerators								
Baseline	8E-08	*(3E-08, 1E-07)*	1E-06	*(6E-07, 2E-06)*	b	b	b	b
Final Standards	2E-08	*(1E-08, 3E-08)*	1E-07	*(8E-08, 2E-07)*	2E-07	*(1E-07, 3E-07)*	4E-07	b
Large On-site Incinerators								
Baseline	4E-08	*(7E-09, 9E-08)*	9E-07	*(6E-07, 1E-06)*	1E-06	*(9E-07, 2E-06)*	b	b
Final Standards	1E-08	*(7E-09, 9E-08)*	6E-07	*(3E-07, 9E-07)*	1E-06	*(7E-07, 2E-06)*	b	b
Small On-site Incinerators								
Baseline	1E-09	*(8E-10, 2E-09)*	1E-07	*(2E-08, 3E-07)*	5E-07	*(9E-08, 8E-07)*	2E-06	*(7E-07, 3E-06)*
Final Standards	1E-09	*(9E-10, 2E-09)*	2E-08	*(1E-08, 5E-08)*	7E-08	*(2E-08, 1E-07)*	2E-07	*(9E-08, 2E-07)*
Waste Heat Boilers								
Baseline	2E-07	*(1E-07, 3E-07)*	1E-06	*(9E-07, 2E-06)*	2E-06	*(1E-06, 3E-06)*	b	b
Final Standards	2E-08	*(1E-08, 3E-08)*	1E-07	*(9E-08, 1E-07)*	2E-07	*(1E-07, 2E-07)*	b	b

[a] Includes cancer risk from incremental exposures to 2,3,7,8-chlorine-substituted dibenzo(*p*)dioxins and dibenzofurans, expressed as TCDD-TEQs.

[b] Percentile and/or confidence level could not be estimated due to small sample size or an insufficient spread of modeled risk values.

Under the final MACT standards, lifetime excess cancer risks from dioxin exposures associated with subsistence fishing (child 0 to 5 years of age) are projected to be below 1E-06 for area source cement kilns, area source incinerators, commercial incinerators, small on-site incinerators, waste heat boilers, and lightweight aggregate kilns. For cement kilns and large on-site incinerators, cancer risks under MACT standard are approximately 1E-06 at a frequency of 0.1 and 0.05, respectively.

Risk estimates generated for both methylmercury and dioxin-TEQ (for the subsistence fisher) are subject to a high degree of uncertainty resulting primarily from the inability to (1) characterize subsistence fishing activity at specific waterbodies and (2) estimate the number of subsistence fishers associated with a given study area (see Section 2.1.1.4). Unlike the recreational fisher receptor that is modeled assuming fishing activity distributed between modeled waterbodies, risks for the subsistence fisher are modeled assuming that fishing activity for each individual is restricted to a single waterbody (i.e., one of the modeled waterbodies). The assumption of single-waterbody activity results in a risk distribution for the subsistence fisher that exhibits greater variance than that generated for the recreational fisher since risks for the subsistence fisher are not diluted through activity distributed across modeled waterbodies (the subsistence fisher also has higher ingestion rates than the recreational fisher). Although single-waterbody activity is considered plausible for the subsistence fisher, if specific individuals do distribute their activity among waterbodies, the single-waterbody assumption would, in some cases, overestimate the exposure and risk that they experience and could introduce uncertainty into risk characterization for this receptor. Uncertainty is also associated with the exposure data used in modeling exposure through fish ingestion. It is not known how representative the data on fish ingestion by Native Americans tribes who are high fish consumers are of other individuals who may engage in subsistence fishing, such as low-income individuals and racial minorities. Uncertainty also stems from the use of the RfD for mercury and the CSF for dioxins/furans (see Section 2.1.1.5) and from modeling the fate and transport of mercury and dioxins/furans (see Section 2.1.1.3).

2.1.4 Population-Level Risks for Human Receptors

The HWC risk analysis assessed population-level risk for a number of cancer and noncancer effects impacting individuals residing within study areas (i.e., local populations). Because these estimates require sector-level population data, they can only be completed for enumerated receptors. Population-level risk estimates have been generated for the following effects: (1) annual cancer incidence resulting from exposure to modeled carcinogens, (2) elevated blood lead (e.g., PbB > 10 µg/dL), (3) inhalation effects resulting from exposure to $PM_{2.5}$ and PM_{10}, and (4) noncancer effects resulting from recreational fisher exposure through fish ingestion. In addition to characterizing population-level risk for individuals residing within study areas, this analysis also assessed the annual cancer incidence in the national population due to dioxin/furan exposures from the consumption of locally produced agricultural commodities. Results generated in this analysis for each of these categories of population risk are summarized below.

2.1.4.1 <u>Annual Cancer Incidence in Local Population Due to Direct and Indirect Exposures to Modeled Carcinogens</u>. Individuals who live and work in the vicinity of hazardous waste combustors are exposed to a number of compounds that are carcinogenic via oral or

inhalation routes of exposure or both. These include dioxins/furans, arsenic, beryllium, cadmium, chromium, and nickel. This category of risk results provides an estimate of the total number of statistical cancer cases projected to occur during a model year within the enumerated receptor populations modeled in this analysis as a result of exposure to these carcinogens. These estimates are generated assuming response additivity across receptors, exposure pathways, and carcinogenic constituents. It is important to recognize the potential sources of risk that are not reflected in these risk results, which include: (1) risks to local receptors that were screened out at proposal (e.g., commercial poultry farmers), and (2) risks to local residents resulting from the consumption of locally produced agricultural commodities (this pathway is assessed only for the commercial farmers and home gardeners). The methodology used to generate annual cancer incidence estimates for local receptors involves combining sector-level individual cancer risk estimates with sector-level population data for the corresponding receptor populations (see Section 8.3.1.1).

Table 2-15 presents summary data for this category of risk results.

Annual cancer incidence estimates for all combustor categories and receptor populations are relatively low. The risk reduction associated with MACT standard implementation, which occurs almost entirely among nonfarm residents, stems mainly from reductions in emissions of metals (primarily arsenic, chromium, and cadmium).

The annual cancer incidence estimates for local populations are subject to many of the same uncertainties associated with characterizing individual cancer risk for commercial farmer receptors. These uncertainties, which are discussed in Section 2.1.1, include: (1) uncertainties associated with air dispersion/deposition modeling; (2) uncertainties associated with farm food-chain modeling; (3) difficulties in projecting human and livestock populations at the sector level; (4) utilization of national-level exposure parameters and assumptions regarding farming practices (rather than regional-differentiated parameters/assumptions); and (5) uncertainties associated with the chemical-specific cancer slope factors used in this portion of the analysis. This category of risk results also assumes additivity across carcinogens, an assumption that introduces uncertainty. As discussed in Section 2.1.1.4, mixtures of carcinogens may display either synergistic or antagonistic behavior with regard to tumor initiation and promotion. For example, different carcinogens may use the same metabolic pathway resulting in competitive inhibition and an effective reduction in the overall incidence of tumors relative to the incidence rate if each chemical were acting alone. It is not currently possible to reflect the synergistic and antagonistic effects that mixtures of carcinogens can display in characterizing cancer risk. This technical limitation introduces uncertainty into the risk estimates that are generated assuming additivity.

2.1.4.2 Annual Excess Incidence of Childhood Blood Lead Levels Exceeding 10 µg/dL. This category of risk results presents an estimate of the annual rate of increase in the number of children with elevated blood lead beyond background (i.e., due to incremental exposure). As with the characterization of individual risk for lead exposure, this analysis focuses on enumerated children (i.e., 0- to 5-yr-olds from each of the enumerated receptors). Elevated

Table 2-15. Annual Cancer Incidence in the Local Population due to Direct and Indirect Exposures (with 90% Confidence Intervals)[a]

Emissions	Incidence
Cement Kilns	
Baseline	7E-04 *(6E-04, 9E-04)*
Final Standards	4E-04 *(4E-04, 5E-04)*
Area Source Cement Kilns	
Baseline	2E-04 *(2E-04, 2E-04)*
Final Standards	9E-05 *(9E-05, 1E-04)*
Lightweight Aggregate Kilns	
Baseline	1E-03
Floor	1E-03
Final Standards	5E-04
All Incinerators	
Baseline	1E-01 *(8E-02, 2E-01)*
Final Standards	2E-02 *(1E-02, 3E-02)*
Area Source Incinerators	
Baseline	4E-03 *(3E-03, 4E-03)*
Final Standards	2E-03 *(2E-03, 2E-03)*
Commercial Incinerators	
Baseline	5E-03 *(4E-03, 6E-03)*
Final Standards	2E-03 *(2E-03, 3E-03)*
Large On-site Incinerators	
Baseline	1E-01 *(7E-02, 2E-01)*
Final Standards	2E-02 *(9E-03, 3E-02)*
Small On-site Incinerators	
Baseline	5E-03 *(3E-03, 9E-03)*
Final Standards	4E-03 *(2E-03, 8E-03)*
Waste Heat Boilers	
Baseline	5E-03 *(3E-03, 6E-03)*
Floor	3E-03 *(2E-03, 5E-03)*
Final Standards	1E-03 *(8E-04, 2E-03)*

[a] Includes excess cancer risk in all receptor populations and age groups from incremental exposures to arsenic, beryllium, cadmium, chromium (VI), nickel, and TCDD-TEQs, assuming additivity.

PbB levels are defined as PbB levels that exceed the action level of 10 µg/dL established for lead.[21]

Population-level risk results for lead are generated by determining the number of children within each sector that exceed the action level for lead and then summing those values across the sectors constituting a given combustor category (for a detailed description of the methodology used to generate these results, see Section 8.3.3).

Table 2-16 presents summary data for this category of risk results.

Annualized projections of the number of children with PbB levels equal to or greater than the action level for lead (i.e., 10 µg/dL) due to incremental exposure at baseline are less than 1 case per year for all combustor categories except OINC-L. OINC-L facilities are projected to have 6 children with PbB levels greater than or equal to 10 µg/dL as a result of incremental exposure to lead assuming baseline conditions. These estimated incremental exceedances are evenly divided between children of residents and children of home gardeners. Implementation of MACT standards will reduce projected incremental exceedances for both combustor categories to less than 1 case per year.

There is significant uncertainty associated with the population-level risk results generated for lead exposure. Many of the same sources of uncertainty that impact the individual-level risk results for lead also impact the population-level results including: (1) uncertainty associated with the characterization of background lead exposure, (2) model uncertainty associated with PbB modeling (i.e., the IEUBK model), and (3) use of the action level for lead to characterize risk (each of these factors is discussed in greater detail in Section 2.1.2.3). In addition, there are socioeconomic factors that are not considered. Specifically, because background exposures are higher among children of minority and low-income households, these children are more likely to have their blood levels raised above 10 µg/dL than children from other demographic groups. The importance of such socioeconomic factors was not considered in the analysis. Therefore, the reductions in excess incidence of elevated blood lead levels may have been underestimated and potential reductions attributable to cement kilns and lightweight aggregate kilns may also have been underestimated.

2.1.4.3 <u>Avoided Incidence Associated with Reductions in Particulate Matter Emissions</u>. This category of population-level risk results characterizes reductions in the annual incidence of specific health endpoints related to PM exposure that are projected to result from reductions in ambient PM concentrations associated with different MACT options). The PM analysis is based on air modeling conducted for HWC study areas and, therefore, applies to enumerated individuals residing within a distance of 20 km from HWC facilities; these estimates do not reflect PM impacts beyond HWC study areas.

The PM analysis uses concentration response functions, which are based on epidemiological studies and relate changes in ambient levels of PM_{10} and $PM_{2.5}$ to changes in the

[21] See Section 2.1.2.3 for a discussion of both PbB modeling using the IEUBK model and the use of the action level for lead in characterizing lead risk.

Table 2-16. Annual Incremental Incidence of Childhood Blood Lead Levels Exceeding 10 µg/dL[a, b]

Source	Annual Excess Incremental Incidence
Cement Kilns	
Baseline	< 1
Final Standards	< 1
Area Source Cement Kiln	
Baseline	< 1
Final Standards	< 1
Lightweight Aggregate Kilns	
Baseline	< 1
Floor	< 1
Final Standards	< 1
All Incinerators	
Baseline	6
Final Standards	< 1
Area Source Incinerators	
Baseline	< 1
Final Standards	< 1
Commercial Incinerators	
Baseline	< 1
Final Standards	< 1
Large On-site Incinerators	
Baseline	6
Final Standards	< 1
Small On-site Incinerators	
Baseline	< 1
Final Standards	< 1

[a] A blood level of 10 ug Pb/dL is the level at which community-wide lead poisoning prevention activities are indicated.
[b] Includes children in all receptor populations ages 0 to 5 years.

incidence of specific health endpoints. PM_{10} includes all air particles that are less than 10 µm in diameter and smaller; $PM_{2.5}$ includes all particles that are 2.5 µm in diameter and smaller. The epidemiological studies underlying the concentration response functions examined the potential effects associated with specific temporal patterns of PM levels. These epidemiological studies also focused on specific types of individuals (i.e., specific age groups including both younger individuals and the elderly). Consequently, the concentration response functions that are used in

the analysis require different mixes of PM modeling data and sector-level population data in order to match the criteria of the underlying epidemiological studies. The concentration-response functions used in this analysis are based on epidemiological studies that covered a variety of health effects and, consequently, provide risk estimates for a range of acute and chronic health endpoints.

Several of the categories of PM-related health effects that are assessed are based on concentration-response functions that contain thresholds of effect (i.e., PM levels below which no effects are projected to occur). If background PM levels together with incremental PM levels (i.e., PM levels associated with modeled facility emissions) do not exceed these thresholds, then the concentration-response functions will project no incidence for these health effects. Ideally, site-specific background PM levels would have been obtained for all modeled HWC facilities but these were not available. To account for the lack of background PM data, avoided incidence for all health endpoints was modeled without threshold, allowing an estimate to be calculated based on the PM reduction associated with the MACT standard. Although this represents a conservative assumption, many of the HWC facilities are located in urban/industrialized areas where background PM levels are likely to be elevated.

Table 2-17 presents summary data for this category of risk results.

The results of the PM analysis indicate that, under MACT standard implementation, there will be 1.5 fewer premature mortalities per year across all combustor categories (for 30+ year-old individuals associated with long-term exposure). In addition, there will be 6 fewer hospitalizations, 25 fewer cases of chronic bronchitis, and 180 fewer cases of lower respiratory disease per year across all combustor categories.

The mortality estimates are subject to some uncertainty due to the fact that the estimate that is derived from long-term studies assumes no threshold for effects. The no threshold assumption used may be appropriate, however, considering that the reduction in mortality is projected to occur entirely from incinerators, especially on-site incinerators. Such incinerators are located at manufacturing facilities that are likely to have other PM emissions, and are typically located in industrial areas where there may be many other sources of PM emissions, resulting in ambient PM levels well above the threshold.

Estimates derived from short-term studies (not shown in Table 2-17) indicated 4.1 fewer premature mortalities per year across all combustor categories (for all ages associated with short-term exposure). This higher estimate may include mortalities that are premature by as little as a few days. Because Pope et al. (1995) is a long-term study, it may be expected that the results of applying the Pope et al. (1995) mortality study to the full population would result in higher estimates of incidence than applying the results of Schwartz et al. (1996), a short-term study. It is possible, however, that the change in air quality is greater using the data required by the Pope function because the studies use different measures of air quality data.

The PM analysis is impacted by a number of uncertainties associated with: (1) limited data on the size distribution of PM emissions from HWC sources, (2) air modeling conducted to generate sector-level PM concentrations (including averaging of air modeling results to generate sector-level estimates), and (3) assumptions/techniques used in establishing sector-level age-

Table 2-17. Avoided Incidence Associated with Reductions in Particulate Matter (PM) Emissions

	Cement Kilns	Area Source Cement Kilns	Lightweight Aggregate Kilns	All Incinerators	Area Source Incinerators	Commercial Incinerators	Large On-site Incinerators	Small On-site Incinerators
Mortality								
Long-term Exposure, 30+ Age Group	0.0	0.0	0.0	1.49	0.01	0.01	1.42	0.06
Hospital Admissions								
All Respiratory	0.02	0.0	0.01	3.96	0.06	0.07	3.48	0.42
Congestive Heart Failure	0.01	0.0	0.00	0.89	0.01	0.02	0.79	0.08
Ischemic Heart Disease	0.01	0.0	0.00	0.98	0.01	0.02	0.88	0.09
Respiratory Symptoms								
Chronic Bronchitis	0.15	0.0	0.07	25.15	0.45	0.51	22.05	2.59
Acute Bronchitis	0.13	0.0	0.05	20.40	0.40	0.46	17.86	2.08
Lower Respiratory Symptoms	1.18	0.0	0.44	181.00	3.61	4.09	158.42	18.49
Upper Respiratory Symptoms	0.14	0.0	0.05	20.99	0.42	0.47	18.38	2.13

delineated population estimates (see Sections 2.1.1.2 and 2.1.1.3). In addition to these sources, there is uncertainty associated with the use of the concentration response functions. Because these functions are based on epidemiological data, a close match must be achieved between the modeled PM levels and population data to which the concentration response functions are being applied and the original sampled population considered in the epidemiological study. While care has been taken in matching these two sets of data (i.e., the modeled to the sampled), uncertainty is associated with the application of the concentration response functions to the modeled data developed for the HWC risk analysis and, consequently, with the incidence estimates that are generated. An expanded discussion of uncertainties in concentration-response functions and in the PM analysis in general is presented in Appendix E.

2.1.4.4 Projection of Number of Potentially "At Risk" Recreational Fishers as a Result of Ingestion of Fish Containing Methylmercury. Because it was not possible to characterize the level of recreational fishing activity at specific modeled waterbodies, quantitative population-level risk estimates could not be generated for the recreational fisher. Instead, the approach was to examine the number of recreational fishers associated with "at risk" facilities (i.e., those modeled facilities with 95th percentile methylmercury individual risk HQ values, including exposure parameter variability, greater than or equal to 1.0).[22] Based on this definition, recreational fishers associated with potentially at risk facilities would represent those individuals who could engage in fishing activity at waterbodies with methylmercury exposure at or above levels of concern due to fish ingestion. The number of recreational fishers located within each study area was estimated by combining U.S. Census block-group-level data with data obtained from the National Survey of Hunting, Fishing, and Wildlife (U.S. DOI, 1993). Facilities identified as urban, based on U.S. Census criteria, are not considered because the characterization of recreational fishing activity in urban areas is especially difficult.

A further refinement to the qualitative statement of potentially exposed recreational fishers has also been included in the recreational fisher assessment. The proportion of the potentially at-risk population that, by virtue of interindividual variation in fish ingestion rates, could be exposed at levels above the RfD, assuming that recreational fishing occurs exclusively at the modeled waterbodies, would be identified. This approach would determine, for each of the facilities identified as at-risk, the proportion of the modeled recreational fisher population that fishes exclusively at modeled waterbodies having an HQ greater than or equal to 1.0.

It is important that the limitations in the recreational fisher population values shown in Table 2-18 be clearly stated—the population numbers do not represent quantitative estimates of the numbers of individuals who are associated with the recreational fishing scenarios modeled for the HWC facilities (i.e., engaged in fishing activity exclusively at the modeled waterbodies). These population numbers represent the number of recreational fishers whose recreational fishing activity might include some activity at modeled waterbodies. It is likely that the large majority of these individuals engage in fishing activity that involves nonmodeled waterbodies.

[22] Because the National Survey data provide recreational fishing activity data for the adult age group, the at-risk designation used in making the qualitative population risk statements was based on individual risk estimates generated for the adult recreational fisher receptor.

Table 2-18. Number of Recreational Fishers Associated with Rural Sites Having Modeled Waterbody Methylmercury HQs of Potential Concern [a]

MACT Options	Facilities		Recreational Fisher Population		
	Total Universe	Potential at Risk[b]	Total for Combustor Category [c]	Associated with at-Risk Facilities	Percentage of Population at at-Risk Facilities above an HQ of 1.0[d]
Cement Kilns					
Baseline	18	0	88,816	0	0
Final Standards	18	0	88,816	0	0
Commercial Incinerators					
Baseline	20	0	633,248	0	0
Final Standards	20	0	633,248	0	0
Lightweight Aggregate Kilns					
Baseline	5	0	123,244	0	0
Final Standards	5	0	123,244	0	0
Large On-Site Incinerators					
Baseline	43	0	1,744,765	0	0
Final Standards	43	0	1,744,765	0	0
Small On-Site Incinerators					
Baseline	79	0	1,712,284	0	0
Final Standards	79	0	1,712,284	0	0
Area Source Cement Kilns					
Baseline	2	0	8,839	0	0
Final Standards	2	0	8,839	0	0
Area Source Incinerators					
Baseline	28	0	603,554	0	0
Final Standards	28	0	603,554	0	0
All Incinerators					
Baseline	142	0	4,090,297	0	0
Final Standards	142	0	4,090,297	0	0

[a] The recreational fisher totals presented in this table are qualitative estimates since it is not possible to state definitively that fishing activity will occur exclusively at the modeled waterbodies evaluated for each site.
[b] "At-risk" facilities are identified as those sites having 95th percentile methylmercury HQs (reflective of exposure parameter variability) greater than or equal to 1.0 for modeled waterbodies.
[c] Excluding urban facilities.
[d] Reflects proportion of recreational fisher population above an HQ of 1.0 at at-risk sites.

Table 2-18 presents summary data for this category of risk results.

Under final MACT standards, no combustor categories are identified as having at-risk facilities as defined for the semiquantitative population-level recreational fisher analysis.

This category of risk results is impacted by significant uncertainty resulting primarily from the fact that the level of recreational fishing activity at specific waterbodies could not be projected (see Section 2.1.1.4). In addition to this factor, additional sources of uncertainty include fate/transport modeling for methylmercury,the methyl mercury RfD, and the fact that modeled waterbodies were not selected using a random sampling techniques (i.e., waterbodies were selected using a systematic approach that may have favored waterbodies more heavily impacted by mercury - see Sections 2.1.1.2 and 2.1.1.3).

2.1.4.5 Annual Cancer Incidence in General Population Due to Dioxin Exposure from Consumption of Locally Produced Agricultural Commodities. Although the HWC risk analysis assessed risk resulting from the consumption of locally produced agricultural commodities by the farmers producing those commodities, this pathway represents only a fraction of the population-level risk associated with these agricultural commodities since the majority of those commodities are distributed and consumed by the general population. In order to assess population-level risk for this potentially significant pathway, the HWC risk analysis also generated annual cancer incidence estimates for the general population resulting from the consumption of key locally produced agricultural commodities.[23]

These estimates are generated by projecting the amount of dioxin/furan contained in the agricultural commodities (specifically beef, pork, and milk) produced within the study areas comprising a given combustor category and using those data togther with EPA's population risk equation to project the annual number of statistical cancer cases resulting from the consumption of that diet-accessible dioxin-TEQ. This calculation is made using the ingestion cancer slope factor for 2,3,7,8-TCDD.

It is important in interpreting this category of risk results to realize that they represent annual incidence rates over the entire national population (although it is possible that these statistical cancer cases may be concentrated in specific areas of the country, reflecting both the location of HWC facilities and the pattern of agricultural commodity processing and distribution at the national level). In addition, these risk results do not include consideration of dioxins/furans that are transported beyond study areas.

Table 2-19 presents summary data for this category of risk results.

The cement kiln combustor category has projected annual statistical cancer incidence estimates at baseline of 7E-02 (for dioxins/furans in beef, pork, and milk combined). With MACT standard implementation, this estimate decreases to 6E-02. The LWAK category has a projected annual statistical cancer incidence estimate of 1E-01 for baseline, which is estimated to

[23] This analysis assessed risk for dioxins/furans contained in beef, pork, and milk since previous risk assessment work completed at proposal showed dioxin-TEQ in these key commodities to be the risk-driving pathway for food ingestion risk.

Table 2-19. Annual Cancer Incidence in General Population due to Dioxin Exposures from Consumption of Locally Produced Agricultural Commodities (with 90% Confidence Intervals)[a, b]

Source	Beef	Pork	Milk	Total
Cement Kilns				
Baseline	6E-03 *(5E-03, 7E-03)*	5E-03 *(4E-03, 5E-03)*	6E-02 *(5E-02, 8E-02)*	7E-02 *(6E-02, 9E-02)*
Final Standards	5E-03 *(4E-03, 5E-03)*	3E-03 *(3E-03, 4E-03)*	5E-02 *(4E-02, 6E-02)*	6E-02 *(5E-02, 7E-02)*
Area Source Cement Kiln				
Baseline	1E-03 *(1E-03, 1E-03)*	1E-03 *(1E-03, 1E-03)*	1E-02 *(1E-02, 1E-02)*	2E-02 *(2E-02, 2E-02)*
Final Standards	1E-03 *(1E-03, 1E-03)*	1E-03 *(1E-03, 1E-03)*	1E-02 *(1E-02, 1E-02)*	2E-02 *(2E-02, 2E-02)*
Lightweight Aggregate Kilns				
Baseline	5E-03 n/a	2E-03 n/a	1E-01 n/a	1E-01 n/a
Floor	5E-03 n/a	2E-03 n/a	1E-01 n/a	1E-01 n/a
Final Standards	7E-04 n/a	2E-04 n/a	1E-02 n/a	1E-02 n/a
All Incinerators				
Baseline	3E-02	1E-02	3E-01	3E-01
Final Standards	5E-03	2E-03	4E-02	5E-02
Area Source Incinerators				
Baseline	1E-02 *(6E-03, 2E-02)*	6E-03 *(3E-03, 1E-02)*	1E-01 *(5E-02, 2E-01)*	1E-01 *(6E-02, 3E-01)*
Final Standards	2E-03 *(9E-04, 3E-03)*	9E-04 *(4E-04,. 2E-03)*	1E-02 *(8E-03, 8E-02)*	2E-02 *(9E-03, 3E-02)*
Waste Heat Boilers				
Baseline	2E-02 *(1E-02, 3E-02)*	6E-03 *(3E-03, 1E-02)*	2E-01 *(1E-01, 3E-01)*	2E-01 *(1E-01, 4E-01)*
Final Standards	2E-03 *(1E-03, 3E-03)*	5E-04 *(3E-04, 1E-03)*	2E-02 *(9E-03, 3E-02)*	2E-02 *(1E-02, 3E-02)*

[a] Includes excess cancer risk from incremental exposures to 2,3,7,8-chlorine-substituted dibenzo(*p*)dioxins and dibenzofurans, expressed as TCDD-TEQs.
[b] Risk results of annual cancer in the general population were not generated for commercial incinerators, large on-site incinerators, or small on-site incinerators.

drop to 1E-02 with MACT standard implementation. The “all incinerators” category has annual statistical cancer incidence estimates at baseline of 3E-01 (the majority of this value is contributed by the WHB category). With MACT standard implementation, this estimate decreases to 5E-02.

These risk results are impacted by a number of uncertainties associated with: (1) assumptions/techniques used in producing sector-level population estimates for the agricultural commodities considered in the analysis (beef cattle, dairy cattle, and hogs), (2) fate/transport modeling completed to derive final concentrations in agricultural commodities (e.g., the modeling of dioxin/furan concentration in plants and the resulting concentration in milk

following grazing), and (3) the cancer slope factor for 2,3,7,8-TCDD (specifically, the assumption that the dose-response curve for dioxins follows a linear, no-threshold model in the low-dose region where these exposures are likely to occur). These sources of uncertainty are discussed in Sections 2.1.1.2, 2.1.1.3, and 2.1.1.5.

2.2 Ecological Risk Characterization

This section summarizes the key findings of the hazardous waste combustors screening ecological risk analysis (HWC-SERA). Ecological risk results were generated for the following combustor categories: cement kilns (CK), area source cement kilns (ASCK), lightweight aggregate kilns (LWAK), commercial incinerator (CINC), large on-site incinerator (OINC-L), small on-site incinerator (OINC-S), area source incinerators (ASINC), all incinerators (INC), and waste heat boilers (WHB). Although the HWC risk analysis was conducted on multiple constituents released from HWC facilities, only those constituents with HQs above the target quotient of 1 (i.e., mercury, dioxins/furans, lead and selenium) are discussed in this section. All other constituents have HQs below the target quotient of 1, indicating low risk to the selected ecological receptors.

Following a brief overview of the methodology used to conduct the HWC-SERA (expanded in Section 9.0), risk results generated for the four constituents of concern are discussed. This discussion is presented in two parts: risk estimation and risk description. The risk estimation section tabulates the results of the HQ calculations for modeled combustor emissions, noting the frequency, magnitude, and rate of exceedances within each facility category. The risk description section describes the ecological significance of the HQ exceedances and the potential for adverse ecological effects associated with HWC emissions. The approach and terminology used in describing the ecological risk results (i.e., inclusion of the risk estimation and risk description sections) reflects guidance presented in EPA's *Guidelines for Ecological Risk Analysis* (U.S. EPA, 1998a). A discussion of the uncertainty and level of confidence associated with the HWC-SERA results follows the presentation of the risk results. This section outlines the limitations and uncertainties associated with ecological exposure assessment and criteria development. It is important that the HWC-SERA results be viewed in light of the key uncertainties outlined. Concluding remarks about the confidence in the analysis results are provided by constituent.

2.2.1 Methodology Overview

The general methodology of the ecological risk assessment was similar across all constituents and exposure pathways, although there are methodological differences in the risk estimations for dioxin and furans compared to the other metal constituents (these are outlined in detail in Section 9.0). The overall methodology implemented to determine ecological risk of HWC emissions is comprised of four basic components:

- Selection of receptors of concern
- Estimation of protective media concentrations (i.e., criteria) for receptors of concern

- Fate and transport modeling of constituents in environmental media

- Calculation of the HQ as the ratio of the modeled concentrations in media to the derived receptor criteria.

Ecosystems were broadly represented as either terrestrial-based or freshwater-based. Representative ecological receptors (e.g., plants, birds, and aquatic biota) were selected to assess impacts for each ecosystem type. Ecological effects data were gathered on receptors of concern to generate protective benchmarks and criteria. Fate and transport modeling was used to simulate the release and deposition of constituents of concern for the 76 modeled HWC facilities and to project the resulting constituent concentrations in media of interest (e.g., soil, surface water) within the modeled study areas. The ecological hazard quotient, which is generated by dividing the modeled exposure concentrations by the ecotoxicological criterion, is used as the metric for assessing ecological risk. At a basic level, the HQ result has a binary outcome: either the constituent concentration is above the screening criteria (HQ > 1) or the concentration is below the criteria (HQ < 1). Because the ecotoxicological criteria are based on de minimis ecological effects, hazard quotients below 1 are presumed to indicate a low potential for adverse ecological effects for those receptor/pathway combinations for which data are available. Hazard quotients above 1 suggest that the potential for adverse ecological effects exists; however, further investigation is needed to improve the resolution of the risk estimates.

To facilitate interpretation of the ecological risk results, three different analyses of the HQ results were conducted. Sector-based HQ values have been normalized for surface areas in conducting each of these analyses to allow area results across sectors to be compared. The three analyses conducted produced the following results:

- **Cumulative Frequency Distributions of Ecotoxicological HQs:** This set of results provides information on the distribution of HQ values across a combustor category. Specifically, HQs associated with specific cumulative frequencies (or percentiles) of the aggregated surface area modeled for a given combustor category are identified. In presenting these risk results, emphasis is placed on characterizing central tendency and high-end HQ levels (i.e., cumulative frequencies presented include: >0.50, >0.10, >0.05, and >0.01).

- **Area (km^2) Within Ecotoxicological HQ Ranges:** This set of results provides combined information on both the magnitude and spatial extent of the HQ exceedances. Specifically, the number of square kilometers associated with specific HQ exceedance ranges is presented.

- **Frequency Bins for the Number of Facilities with Areas That Exceed Ecotoxicological HQ of 1:** This set of results provides additional information on the spatial pattern of HQ exceedances. This information is useful in determining whether the potential for adverse ecotoxicological impacts, as identified by the HWC-SERA, are likely to be restricted to a single facility within a combustor category or distributed across multiple facilities.

2.2.2 Mercury

2.2.2.1 <u>Risk Estimation</u>. In this HWC-SERA assessment, risks were assessed for terrestrial and freshwater ecosystems. Terrestrial ecosystems were evaluated using total mercury concentrations in soil. Freshwater ecosystems were evaluated using dissolved methylmercury concentrations in the surface water and total mercury concentrations in sediment. As discussed in Section 5.3, freshwater systems were modeled using the same techniques reported in the *Mercury Study Report to Congress* (U.S. EPA, 1997) because the MRTC contains the state-of-science modeling techniques and appropriately conservative methylmercury benchmark derivation methods for a screening analysis.

Although total dissolved mercury was evaluated in freshwater ecosystems, the risks associated with methylmercury in surface waters reflect a more significant exposure pathway and higher potential for adverse effects in piscivorous receptors. Because fish accumulate and store methylmercury more readily in their tissues, methylmercury is the predominant chemical species to which upper-level predators are exposed. Further, higher confidence is placed in the modeled results for methylmercury based on the robust technical modeling approach used to estimate concentrations in waterbodies surrounding hazardous waste combustors. Given the greater confidence in the methylmercury media concentrations and criteria, the methylmercury results should be viewed as more representative of the risk in freshwater ecosystems. The risk estimates for freshwater ecosystems suggested no potential for adverse effects based on concentrations of methylmercury in surface water, and minimal risks were indicated in sediments.

Results Overview. The baseline results for total mercury in soil suggest that the soil community (i.e., earthworms) may be at risk from releases associated with hazardous waste combustion. Facility types, in descending order of soil areas exceeding, were INC, OINC-L, CK, ASCK, and LWAK. Across all combustor categories, mercury is the constituent exceeding at the highest frequency. However, mercury HQs are less than 10 across all facility types. The frequency of facilities exceeding as a percentage of the total facilities within a combustor category is provided in Table 2-20. With the exception of ASCKs, where only two facilities were represented, mercury exceeds at up to 20 percent of facility sites within a combustor category. Soil areas exceeding are always less than 1 percent of the total soil area in each facility category.

The results generated for the MACT standard indicate a decrease in both the number of facilities with HQ exceedances and the overall spatial extent of HQ exceedances for terrestrial ecosystems. Specifically, the results presented in Table 2-20 indicate that the areas with HQ exceedances in soil dropped for LWAK, INC, and OINC-L. In several cases, soils surrounding facilities continued to indicate HQ exceedances following MACT standard implementation (i.e., CK, ASCK).

Baseline and MACT standard results for freshwater ecosystems are also reported in Table 2-20. No HQ exceedances for methylmercury were indicated for either baseline or the MACT standard modeled results in surface water. Exceedances in sediment were less than 1 percent of total sediment area assessed.

The exceedances generated for soils and sediments were minimal in that in most combustor categories' exceedances represented less than 1 percent of all HQs generated. For

sediments and soils surrounding cement kilns (CKs), the cumulative frequency distribution (CFD) of HQs indicated that less than 1 percent of HQs exceeded 0.5. The MACT standard results for soils indicated that less than 1 percent of HQs exceeded 0.3, and sediments showed no exceedances. The CFD of HQs for ASCK indicated that less than 1 percent of HQs exceeded 0.7 across soil areas. The corresponding ASCK MACT standard results indicated that less than 1 percent of HQs were greater than 0.6 across soil areas. In soils modeled for the facility categories LWAK, INC, and OINC-L, the HQ CFD results indicated that less than 1 percent of HQs were greater than 0.3. Under the corresponding MACT standard results, exceedances were no longer indicated for these same three facilities (i.e., LWAKs, OINC-L and INC).

Freshwater Ecosystems. Both mammals and birds were evaluated for exposure to methylmercury via ingestion of contaminated fish. Receptors evaluated include the kingfisher, river otter, mink, osprey, and bald eagle—wildlife species identified in the MRTC as being highly exposed to mercury.[24] No exceedance was indicated based on modeling the food web methylmercury exposures to mammals and birds that forage on fish. The measures of effect for both mammals and birds associated with freshwater ecosystems included endpoints on reproductive fitness and survival that are considered relevant to population sustainability. Baseline and MACT standard risks to fish, aquatic invertebrates, and algae are below levels of concern (HQ < 1). Consequently, the results suggest that populations of kingfishers, river otters, mink, osprey, and bald eagles living in aquatic ecosystems near HWC units are unlikely to be at risk.

As presented in Table 2-20, exceedances generated for the benthic community indicate the potential for altered community structure and a decrease in the abundance of benthic organisms. However, because exceedances were indicated over a limited spatial scale (i.e., 1 km^2 exceeding), effects are likely to be localized. No significant impacts to freshwater ecosystems at the national scale are indicated in these results.

Terrestrial Ecosystems. Exceedances of the target HQ suggest that one species, earthworms commonly used to indicate soil productivity, may be exposed to mercury at levels of concern. The relevance to the assessment endpoint—soil community structure and function— is unclear because the ecotoxicological criteria only represents earthworm species (i.e., adequate data were not identified to develop a criteria based on multiple species in the soil community). Earthworms have been shown to be sensitive to a variety of contaminants and are known to serve crucial functions for the soil community (Efroymson et al., 1997). HQ values for terrestrial mammals are below 1 for representative species considered in this analysis. Only the lowest criterion identified (i.e., earthworms, in this case) was used for risk estimates; hence other receptors with similar sensitivity to exposure were not evaluated. In the case of mercury, the soil benchmark for avian receptors was only 1.5 times higher than the earthworm benchmark, so

[24] The loon and Florida panther were included as representative receptors in the *Mercury Study Report to Congress* (U.S. EPA, 1997); however, these receptors were not evaluated in the HWC analysis. Because the HWIR methodology was adopted as the basis for the HWC SERA and to remain consistent in evaluating the same receptors for all constituents, only those receptors overlapping between the HWIR approach and the MRTC were evaluated. However, the loon is likely to be protect at the more conservative criterion used for the kingfisher. The Florida panther is not as representative at the national scale as other receptors chosen for this analysis.

Table 2-20. Summary of Exceedance Results for Mercury

Combustor Category	Soil		Sediment		Surface Water	
	Area (km^2) Exceeding (% of total area)	No. of Facilities Exceeding (% of total facilities)	Area (km^2) Exceeding (% of total area)	No. of Facilities Exceeding (% of total facilities)	Area (km^2) Exceeding (% of total area)	No. of Facilities Exceeding (% of total facilities)
Cement Kilns (Total number of facilities = 15)						
Baseline	39 (<1)	2 (13)	1 (<1)	1 (7)	0	0
Final Standard	14(<1)	1(7)	0	0	0	0
Area Source Cement Kilns (Total number of facilities = 2)						
Baseline	14 (<1)	1 (50)	0	0	0	0
Final Standard	14 (<1)	1 (50)	0	0	0	0
Lightweight Aggregate Kilns (Total number of facilities = 5)						
Baseline	3(<1)	1(20)	0	0	0	0
Final Standard	0	0	0	0	0	0
All Incinerators (Total number of facilities = 56)						
Baseline	87 (<1)	2 (4)	0	0	0	0
Final Standard	0	0	0	0	0	0
Large On-site Incinerators (Total number of facilities = 18)						
Baseline	87 (<1)	2 (11)	0	0	0	0
Final Standard	0	0	0	0	0	0
Small On-site Incinerator						
Baseline	0	0	0	0	0	0
Final Standard	0	0	0	0	0	0

there is a possibility that these receptors may be adversely impacted by mercury exposures as well.

The area exceedance results in Table 2-20 provide a relative national context for the spatial extent of exceedances within a facility type; however, they do not provide the spatial resolution to delineate the ecological significance of predicted effects across the United States. Further information, such as habitat suitability, distribution of contaminants within a habitat, and wildlife foraging patterns and predator-prey interactions, would be required to evaluate the ecological impacts of exceedances.

The MACT standard results indicated no changes in exceedance for ASCK, whereas the frequency of facilities exceeding was reduced but not eliminated for CK. Exceedances were eliminated for the facility types LWAK, INC, and OINC-L upon modeling the MACT standard. The changes identified by modeling the MACT standard are incremental. Facilities minimally exceeding a criterion at baseline fell below the criterion under the MACT standard, but those

with higher HQs under baseline modeling were still exceeding after modeling the MACT standard. Because of the lack in resolution inherent in a screening-level analysis, these reductions from a hazard quotient value greater than 1 to a lower value that is still greater than 1 do not necessarily indicate a decrease in risk.

2.2.2.2 Risk Description.

Ecological Significance of Target HQ Exceedances for Mercury

- Exceedances are indicated for soil and benthic communities surrounding facilities.
- The potential for adverse effects impacts at the national scale is minimal for the following reasons:
  - HQs are less than 10
  - These communities show high functional redundancy
  - Potential impacts are very localized (i.e., small areas indicating exceedance).

Freshwater Ecosystem. As indicated in the risk estimation, no HQ exceedances occurred for freshwater receptors: kingfisher, river otter, mink, osprey, and bald eagle. The criteria used in calculating HQ exceedances were based on reproductive and developmental endpoints for mammalian and avian species; hence the lack of exceedances indicates that it is unlikely for these receptors to experience inhibited reproductive potential due to methylmercury exposure resulting from HWC units. There is some concern associated with anthropogenic background concentrations of methylmercury in surface water. In some areas, anthropogenic background concentrations alone can exceed the wildlife criterion (U.S. EPA, 1997). If the HWC incremental increases of methylmercury to surface waters were added to these background concentrations, then there might be exceedances of the HQ for freshwater receptors. For a more detailed review of this uncertainty, see Section 2.2.6.2.

The sediment exceedances for the benthic biota were isolated to one facility type over a limited spatial scale, indicating a minimum potential for ecological impacts. The sediment exceedances are not likely to be significant at the national scale.

Terrestrial Ecosystem. Target HQ exceedances indicate the potential for adverse effects to the reproductive capacity of earthworms, which was used as a surrogate for the soil community assessment endpoint. Although it is desirable to base the soil criteria on toxicity data for soil receptors that perform a variety of functions, the paucity of data did not support the development of a community-based criterion. Hence, the criterion used to generate the soil HQs for mercury may be inappropriately conservative with respect to the overall structure and function of the soil community. Because the mercury soil criterion is based on two earthworm studies, confidence in this value to characterize the ecological responses that could occur in the field is low. In effect, the conclusion that exceedances will result in adverse impacts to the soil community and, indirectly, to other trophic levels cannot be supported in the screening analysis.

2.2.3 Dioxin/Furans

2.2.3.1 Risk Estimation.

Results Overview. The baseline results for dioxin suggest that terrestrial mammals are potentially at risk from dioxin releases associated with hazardous waste combustion, but no risks

were indicated for terrestrial avian receptors considered in this analysis. None of the HQ exceedances generated for these receptors were greater than 10 across all combustor categories. Study data suitable to develop ecotoxicological criteria for various communities (e.g., fish and plant communities) were not identified. Although target HQ exceedances were noted across several combustor categories for soils, exceedances were observed in less than 1 percent of total areas (km^2) for each combustor category. The frequency of facilities exceeding and the area in square kilometers as a percentage of total facilities and area, respectively, within a combustor category are provided in Table 2-21. The facility category INC had the highest percentage of total area exceeding the target HQ of 1 followed by OINC-S, with other combustor categories indicating minimal exceedances. WHB incinerators were noted to be the primary contributors to risk exceedance in soil for dioxin/furans.

The dioxin/furan congener exceedances generated for soils were minimal, in that, in most combustor categories, exceedances represented less than 1 percent of all HQs generated. Dioxin HQ CFD results in soil indicated that less than 1 percent of HQs were greater than 0.2 for CKs, LWAKs, and CINC, while less than 1 percent of HQs exceeded 0.008 for OINC-S, ASINC, and INC facility categories. Waste heat boiler CFD results indicated that less than 1 percent of HQs were greater than 0.2 across soil areas.

MACT standard results overall indicated that exceedances seen for dioxin were reduced to HQs less than 1 by the MACT standard for all facility types.

Freshwater Ecosystem. The dioxin HQs are based on the toxicity equivalency concentration (TEqC) dose approach and, consequently, do not reflect a direct comparison with either the surface water or sediment concentrations predicted by the fate and transport model (See section 9.0). However, because the HQs represent risks to piscivorous mammals and birds in a general aquatic ecosystem, it is more appropriate to present the results under the surface water column. The presentation of the freshwater results as surface water HQs takes into account the fact that dioxin/furan congeners move from sediments to the water column in dynamic equilibrium, the assumption being that the exposure dose remains consistent whether the exposures occur via the sediment or the surface water in the freshwater ecosystem.

Food web modeling of uptake through fish to representative mammals did not indicate the potential for adverse effects. Risks to aquatic (including algae) and benthic communities were not evaluated due to a lack of suitable data identified, but, as indicated in the ecotoxicity profile (see Appendix J), risks to invertebrates are minimal compared to vertebrate responses. Of the species not evaluated due to a lack of data, fish are probably the most likely vertebrate receptors to elicit adverse effects from dioxin/furan exposures.

Terrestrial Ecosystems. In terrestrial ecosystems, the exposure concentrations in soil suggested that the white-tailed deer may receive doses through food chain exposures that could potentially result in adverse effects. Other mammals that indicate the potential for adverse effects are the eastern cottontail, red fox, and the raccoon. The criteria used in calculating HQ exceedances were based on reproductive and developmental endpoints for mammalian species; hence exceedances indicate the potential for inhibited reproductive potential. The endpoint suggests that the potential impacts on these species could result in a decrease in populations over time. However, the approach used for estimating food chain bioaccumulation is very

Table 2-21. Summary of Exceedance Results for Dioxin in Soil

Combustor Category	Soil	
	Area (km^2) Exceeding (% of total area)	**No. of Facilities Exceeding (% of total facilities)**
Cement Kilns (Total number of facilities = 15)		
Baseline	4 (<1)	1 (7)
Final Standard	0	0
Lightweight Aggregate Kilns (Total number of facilities = 5)		
Baseline	3 (<1)	1 (20)
Final Standard	0	0
All Incinerators (Total number of facilities = 56)		
Baseline	19 (<1)	4 (7)
Final Standard	0	0
Area Source Incinerators (Total number of facilities = 9)		
Baseline	7 (<1)	2 (22)
Final Standard	0	0
Commercial Incinerators (Total number of facilities = 13)		
Baseline	7 (<1)	2 (15)
Final Standard	0	0
Small On-site Incinerators (Total number of facilities = 25)		
Baseline	12 (<1)	1 (4)
Final Standard	0	0

Note: No exceedances were indicated for dioxin for the following facilities: ASCK, OINC-L.

conservative since all congeners were assumed to bioaccumulate in mammals to the same extent as TCDD. Data on bioaccumulation in domestic grazing animals show that the more highly chlorinated congeners exhibit much lower bioaccumulation. Soil exceedances were restricted to relatively small surface areas (less than 1 percent of total area within a combustor category) across combustor categories.

Exceedance results generated for the MACT standard indicated a reduction in risk (HQ<1) to the white-tailed deer, eastern

Ecological Significance of Target HQ Exceedances for Dioxin/Furans

- Exceedance area are noted for mammalian receptors surrounding facilities.
- Criteria species indicating risk include: white-tailed deer, Eastern cottontail, red fox, and raccoon.
- Exceedances indicate the potential for a reduction in the reproductive capacity of receptors.
- Changes in food web structure may result from a loss of predators.
- Exceedances observed in the terrestrial ecosystems were eliminated upon modeling the MACT standard for facilities.

cottontail, red fox, and the raccoon from food chain exposure for all combustor categories. Potential impacts to terrestrial plant communities and soil communities could not be evaluated due to the lack of ecotoxicity data, but, given that vertebrates are more sensitive to dioxin exposure, effects to nonvertebrate receptors are not expected.

2.2.3.2 Risk Description.

Freshwater Ecosystem. The HQ results indicate that risk is not expected for receptors of the aquatic community.

Terrestrial Ecosystems. For the terrestrial ecosystem, target HQ exceedances were observed for the white-tailed deer, eastern cottontail, red fox, and raccoon. Because the assessment endpoints of the analysis were based on reproductive and developmental endpoints, population declines via diminished reproductive capacity are possible as a result of higher exposure to dioxin/furan congeners. The impact estimated for a decline in deer populations may limit other secondary predators that depend on deer as prey. As a result, these predators may have to choose different prey, which may lead to alterations in community dynamics. The overall impacts at baseline to the white-tailed deer may be minimal since exceedances are projected for relatively small areas. Under the MACT standard, terrestrial HQ exceedances are reduced to levels below which adverse effects are eliminated for dioxin/furan congeners.

2.2.4 Lead

2.2.4.1 Risk Estimation.

Results Overview. Target HQ exceedances were indicated for lead upon assessing soil and benthic communities and the reproductive capacity of the river otter. Exceedances were indicated in all media at INC and OINC-L facility types. Only surface water exceedances were indicated in ASINC, CINC, and CK facilities. No exceedances were seen at facility types ASCK, LWAK, and OINC-S. Lead HQ exceedances fell in the range of 1 to 10. Exceedances estimated for baseline were eliminated by modeling the MACT standard for all facility types. WHBs contributed to approximately half of the risk exceedance estimated for surface water. The frequency of facilities exceeding and the area in square kilometers as a percentage of total facilities and area, respectively, within combustor category are provided in Table 2-22.

The lead exceedances generated for soil, surface water, and sediment were minimal in most combustor categories where exceedances represented less than 1 percent of all HQs generated. In waterbodies, the CFD of HQs for lead in CKs indicated that less than 1 percent of HQs exceeded 0.5 across waterbody areas. For INC and OINC-L categories, CFDs indicated that less than 1 percent of HQs exceeded 1 across waterbodies while less than 5 percent of INC HQs exceeded 0.2 across waterbodies. Waste heat boiler CFD results indicated that less than 1 percent of HQs were greater than 0.2 across waterbodies. In sediments, HQ CFDs for the same facility categories indicated that less than 1 percent of HQs were greater than 0.2. In soils, the CFD of HQs for INC and OINC-L combustor categories indicated that less than 1 percent of exceedances were greater than 0.004. Modeling of the MACT option for lead indicated no exceedances across all combustor categories.

Table 2-22. Summary of Exceedance Results for Lead in Soil, Sediment, and Surface Water

Combustor Category	Soil		Surface Water		Sediment	
	Area (km^2) Exceeding (% of total area)	No. of Facilities Exceeding (% of total facilities)	Area (km^2) Exceeding (% of total area)	No. of Facilities Exceeding (% of total facilities)	Area (km^2) Exceeding (% of total area)	No. of Facilities Exceeding (% of total facilities)
Cement Kilns (Total number of facilities = 15)						
Baseline	0	0	1 (<1)	1 (7)	0	0
Final Standard	0	0	0	0	0	0
All Incinerators (Total number of facilities = 56)						
Baseline	6 (<1)	2 (4)	37(1)	5(9)	2 (<1)	2 (4)
Final Standard	0	0	5(<1)	0	0	0

Note: No exceedances were indicated for lead in the following facilities: ASCK, LWAK, OINC-S.

Freshwater Ecosystems. The modeled surface water concentrations exceeded the surface water criterion for the river otter in five combustor categories. The largest area and number of facilities indicating exceedance were generated for the INC category. Modeled results indicate that fish and algae are not likely to be adversely impacted by modeled exposures to lead. Sediment exceedances were noted for the benthic community in INC and OINC-L facility categories. MACT standard results indicated that all exceedances were reduced to an HQ < 1 (i.e., risk not anticipated).

Terrestrial Ecosystems. In terrestrial ecosystems, target HQ exceedances were estimated for the soil community as well as for birds and mammals. However, the lead criteria for birds and mammals were below the national mean background concentration range for lead and, therefore, were considered inappropriate for the SERA. The HQ exceedances for soil fauna suggest the potential for adverse effects to the soil community. Soil exceedances were indicated only at the facility types INC and OINC-L. The soil concentrations estimated for the MACT standard were below levels of concern (HQ < 1) for the soil community.

Ecological Significance of Target HQ Exceedances for Lead

- Exceedance sectors are noted only for mammals (i.e., river otter), soil community, and the benthic community.
- Criteria species demonstrating risk include the river otter, soil invertebrates, and benthic invertebrates.
- Exceedances indicate the potential for a change in soil and sediment community structure that may have an impact on community function.
- Further review of the risk to mammalian and avian wildlife exposed to lead in the terrestrial ecosystem is indicated.

2.2.4.2 Risk Description.

Freshwater Ecosystem. Exceedances were indicated for mammals (i.e., predators characteristic of aquatic ecosystems) and the benthic community. The criteria used in calculating HQ exceedances for the river otter were based on reproductive and

developmental endpoints. Exceedances for the river otter indicate the potential for inhibited reproductive potential to this receptor. The endpoint suggests that the potential impacts on these species could result in a decrease in population numbers over time. Because this mammal, associated with aquatic habitats, feeds on trophic level 3 and 4 fish, the absence of this predator may disturb the dynamics of the community freshwater fish. Exceedances for the sediment community indicate the potential for changes in the benthic community structure, function, and species abundance. Because these receptors provide a food base for the aquatic community, indirect impacts caused by a loss of available prey to benthic predators may alter community dynamics. Because the exceedances occur over a limited amount of watershed area and only a few facilities indicate exceedance, the impacts are not likely to be significant.

Terrestrial Ecosystem. Exceedances were shown for species important to the structure and function of the soil community. HQ exceedances were estimated in combustor categories of OINC-L and INC. Exceedances for lead in the terrestrial ecosystem were associated with relatively small spatial areas (i.e., HQ exceedances represent less than 1 percent of the total soil surface area across categories), and potential impacts are not likely to result in significant impacts at the national scale. The soil community criterion was selected for HQ determinations because terrestrial mammalian and avian criteria were below national mean background concentrations. Criteria that fall below background concentrations are considered to be of marginal ecological relevance and require further investigation of the benchmarks and exposure inputs (e.g., bioconcentration factors in food items). Although these criteria prevented avian and mammalian species from being evaluated, the discrepancy between background concentrations and the proposed criteria (i.e., the fact that the criteria are below background) should not be interpreted as suggesting that these receptors are not at risk. Better characterization of the sensitivity of these receptors to lead exposure is required to evaluate the true nature of effects.

2.2.5 Selenium

2.2.5.1 Risk Estimation.

Results Overview. HQ exceedances observed for river otters in freshwater ecosystems near cement kilns were all less than 10; however, no exceedances were estimated for any other receptors included in the HWC SERA. Exceedances were noted in only one facility in an area of 1 km^2. The results for selenium indicated exceedance only in surface waters surrounding CKs. The CFD of HQs for selenium indicated that less than 1 percent of HQs were greater than 0.2 for both baseline and MACT standard modeled results.

The MACT standard did not change the risk results for selenium. However, the number of sector exceedances and the relatively low HQ values (below 10) suggest that the potential for adverse effects applies to a small spatial scale.

Freshwater Ecosystems. The results suggest that surface water exposures to river otters through the aquatic food web may inhibit the reproductive potential of this species. The modeled selenium concentrations were below levels of concern (i.e., HQ < 1) for other receptors associated with the freshwater ecosystem: birds, algae, fish, and aquatic invertebrates (data were insufficient to evaluate the benthic community). No changes in surface water exceedances were noted for the river otter receptor upon modeling the MACT standard.

Terrestrial Ecosystems. Target HQ exceedances were not indicated for ecological receptors representing the terrestrial ecosystem.

2.2.5.2 Risk Description.

Freshwater Ecosystem. Target HQ exceedances for selenium were shown for the river otter. The relatively small spatial extent of sector exceedances (projected exceedances involved one cement kiln facility site) indicates that, at a national level, the ecological risks to river otters from selenium may be highly localized. Moreover, selenium is required for optimal growth and homeostatic regulation and, therefore, many organisms have mechanisms to regulate selenium uptake and retention. Although field studies documenting selenium exposures through the food web suggest that this exposure pathway presents risks to wildlife species (e.g., Ohlendorf et al., 1989), most studies indicate effects only to birds and aquatic organisms (e.g., fish). Therefore, the potential for population-level effects to the river otter or similar species is highly uncertain.

Terrestrial Ecosystem. No exceedances were noted for receptors in the terrestrial ecosystem across combustor categories for selenium.

Ecological Significance of Target HQ Exceedances for Selenium

- Small exceedances were indicated for the river otter at cement kiln facilities.
- Exceedances suggest the potential for adverse effects to river otter populations; however, impacts are likely to be localized.
- Exceedances were indicated over a 1-km^2 area at one CK site suggesting a limited spatial distribution of potential impacts.

2.2.6 Limitations and Uncertainty

Ecological risk characterization is impacted by uncertainties associated with the characterization of a number of factors including facility emission profiles, site-specific physical features, receptor location and behavior (linked to exposure), and dose-response profiles for constituents. Of special note for ecological risk characterization is the critical role played by both spatial and temporal factors in determining the significance of ecological impacts. While the methodology used in this analysis does not characterize ecological exposure in terms of temporal aspects, some evaluation of spatial aspects is provided. The key findings summarized in previous sections must be interpreted within the context of the limitations and uncertainties inherent in the SERA, which can be grouped as exposure issues and criteria development issues. While certain limitations are intrinsic to **any** ecological risk assessment (e.g., extrapolation of laboratory data to field exposures), this section is focused on the uncertainty, limitations, and confidence specific to the HWC screening analysis.

2.2.6.1 Exposure Issues. The issues of uncertainty associated with exposure can influence the risk estimation results by changing the relative magnitude, frequency, and duration of exposures. Because this is a screening analysis, most of the assumptions made about ecological exposures were conservative. The issues of uncertainty related to exposure that should be considered in interpreting the significance of the HWC SERA results are discussed below.

Co-occurrence of Receptor and Constituent. The co-occurrence of the stressor and the assessment endpoint was assumed for each HWC facility. This simplification is adopted for screening-level analyses in which site-specific ecological data are not within the scope of the assessment. Consequently, the analysis does not assess the probability that (1) a receptor will be found in a contaminated sector, (2) a receptor will forage for food in contaminated sectors, or (3) a habitat will support the type of habitat needs of the receptor. This implicit assumption adds to the conservative nature of the screening assessment since not all HWC facilities may be located in areas that are capable of sustaining receptors included in this analysis. However, the ecological receptors that were included in the analysis are commonly occurring species and, taken as whole, are expected (or can be presumed) to be more or less representative of other ecological receptors (barring evidence to the contrary).

Assumptions on Dietary Exposure. Screening-level assessments typically assume maximum intake of contaminated prey in the diets of primary and secondary consumers (i.e., 100 percent of the diet originates from the contaminated area). Obviously, under field conditions, many receptors are opportunistic feeders with substantial variability in both the type of food items consumed as well as the seasonal patterns of feeding and foraging. The home range of the ecological receptor is an issue here as well. If an animal forages or hunts for prey over an area larger than a sector, then the exposure could be under- or overestimated. Consequently, the exclusive diet of contaminated food items tends to provide a very conservative estimate of potential risks.

Spatial and Temporal Scales of Exposure. Consideration of the spatial extent and pattern of projected HQ exceedances is important in assessing the potential impact to ecological receptors. For example, defining the intersection between projected HQ exceedance areas and ecological receptor habitats at the site-specific level would allow more refined statements regarding potential impacts to those receptors to be made. Although the HWC risk analysis used a 16-sector template in modeling media concentrations within specific study areas, which does provide significant refinement in evaluating the areal extent of HQ exceedances, the identification of specific habitat areas at the site-specific level was beyond the scope of this analysis. Consequently, it was not possible to quantitatively assess the relationship between projected HQ exceedances and ecological receptor habitats.

The timing of exposure will also influence the impact to a population. If peak exposures occur during sensitive life stages (e.g., juvenile) or during the breeding season, impacts on population dynamics (e.g., percent survival) may be significant. Hence, averaging exposure concentrations over longer periods of time may underpredict population risks. Long-term, low-level releases may have cumulative impacts on populations and communities that are not evident from the available laboratory data (i.e., multigenerational studies are not frequently available). Alternatively, such chronic exposures may not ever exceed threshold concentrations at which adverse effects may be observed. The HWC screening analysis was based on a maximum annual exposure concentration and, assuming that peak exposures would not be significantly different from the annual average, the risk estimates tend to be conservative. The magnitude of this conservatism depends on the overall exposure profile (i.e., how variable are the annual exposure concentrations from the maximum).

Bioavailability of Constituents of Concern. For the purposes of this screening-level analysis, all forms of a constituent are assumed to be equally bioavailable. This assumption tends to overestimate the actual exposures that may occur in the field. This assumption is appropriate given the screening nature of this analysis; however, both the chemical form and the environmental conditions influence bioavailability and, ultimately, the expression of adverse effects. In particular, the form of selenium has been shown to influence toxicity.

Characterizing Bioaccumulation. Characterizing the uptake of constituents of concern through the food chain was estimated by selecting accumulation factors in preferred prey of mammalian and avian receptors of concern. Deriving an appropriate bioaccumulation metric that properly characterizes the magnitude, rate of uptake, and elimination of constituents in ecological receptors is a point of uncertainty in this analysis. The rationale and selection of these values are detailed in Appendix J (which contains ecotoxicity profiles for each constituent). A brief review of the uncertainty in these values is presented here. In the case of metals, measured values found in the literature were used to generate high-end estimates of bioaccumulation. The uncertainties related to bioavailability, duration of exposure, and lifestage exposed can highly influence the actual versus predicted accumulation. Because only the high-end value was used, this one value may not represent the range and variability this parameter presents at the national scale. A brief discussion of uncertainty in the BAFs for metals is reviewed here while the key uncertainties associated with the dioxin/furan BSAFs are reviewed in Section 2.2.6.3.

- *Lead*—In the freshwater ecosystem, the database for lead uptake factors in fish was the most limited compared to other constituents indicating exceedance. One BAF value was identified to characterize the uptake of lead. Applying this value introduces some uncertainty into the analysis in that high-end conservatism could not be confirmed without a distribution of values. In terrestrial ecosystems, the uptake factors in earthworms were characterized by 20 studies, which provided better resolution to assess the uptake factor variability. From these 20 studies, the 90th percentile value was selected as the BAF. Terrestrial plant uptake values were derived from a database of 204 values that represented differences across the variables such as soil chemistry, plant species, and soil depth.

- *Selenium*—In freshwater ecosystems, the uptake factors for fish were also limited by data availability. The BAFs selected for fish were pulled from one study reporting six different BAFs across trophic level 3 and 4 fish. Although differences were seen across trophic levels of fish, the lack of comparable studies increased the uncertainty in these uptake values. In terrestrial ecosystems, a similar database limitation was evident in characterizing the uptake of selenium in earthworms. One study reporting 14 observations was used to derive earthworm BAFs. Terrestrial plant uptake values were derived from a database of 237 values that represented differences across the variables such as soil chemistry, plant species, and soil depth. High-end values were selected in all cases; however, the lack of data did not allow the variability of this parameter to be assessed on a national scale.

- *Mercury*—In freshwater ecosystems, uptake factors for methylmercury were adopted directly from the *Mercury Study Report to Congress* (U.S. EPA, 1997). Relative to other constituents indicating exceedance, the variability in mercury BAFs was well represented in both freshwater and terrestrial ecosystems. In the freshwater ecosystem, the MRTC conducted a Monte Carlo analysis to characterize the variability in BAFs in both trophic level 3 and 4 fish. The data used were derived from field studies measuring uptake values. A large source of variability identified in the uptake values was correlated with fish size and fish age. To remain consistent with the methods and recommendations of the MRTC, the geometric mean of the methylmercury BAFs was used instead of the high-end values.

In terrestrial ecosystems, uptake factors for worms were characterized by five studies reporting 30 observations. The uptake factors for the terrestrial ecosystem were based on total mercury concentrations. High-end (i.e., 90th percentile values) were applied to determine exposures to terrestrial receptors preying on invertebrates. Uptake data for total mercury in plants were not identified to characterize this prey item.

Multiple Constituent Exposures. The risk of each constituent is considered separately in this analysis. However, stack emissions data suggest that exposure to multiple constituents simultaneously is highly probable. The synergism or antagonism between different constituent combinations may elicit unexpected adverse impacts to ecosystems. Hence, the screening analysis may underestimate risks associated with multiple chemical stressors. This concern should be mitigated to some extent by the fact that for most constituents assessed, exposures were estimated to be low relative to ecotoxicological criteria.

Waterbody Characterization. Waterbodies were selected based on their utility as a drinking water source, their recreational importance, or their location directly downwind from the source. Although the selection process is appropriate for evaluating human health risks, it may not adequately represent the aquatic habitats at risk from HWC emissions. Waterbodies and wetlands with high ecological significance may not have been represented in the HWC ecological risk analysis. In addition, a single waterbody exhibiting a target HQ exceedance may be assumed to have local (and somewhat limited) ecological significance. However, if several waterbodies in the proximity of the facility are shown to have modeled concentrations that exceed the ecotoxicological criterion, the adverse impacts on aquatic life may be more significant. This issue was looked at indirectly by estimating the total waterbody area in exceedance and the corresponding number of facilities.

2.2.6.2 Criteria Development.

Mercury Background Concentrations. Mercury and other constituents can be transported to remote areas through long- range transport, and this process increases the potential accumulation of these constituents to overall background levels (i.e., natural background and

anthropogenic background)[25]. The cumulative effect of both HWC emissions and other background sources may elevate the potential for risk to ecological receptors. EPA has indicated that mercury release from different industries over time has resulted in elevated anthropogenic background concentrations. Comparing these concentrations to wildlife criteria occasionally results in exceedance of protective levels (U.S. EPA, 1997). This indicates that anthropogenic background concentrations of mercury can contribute to the potential risks to ecological receptors. The HWC SERA represented only incremental risks associated with the release of methylmercury from HWC facilities, and it does not assess the cumulative risk of anthropogenic background that may contribute to the risk to ecological species. The HWC SERA did not consider the contribution of constituents transport by long-range mechanisms to overall media concentrations. Because this has the potential to result in underestimation of risk, a level of uncertainty is introduced into the HWC results.

For comparison, mercury concentrations (total dissolved methylmercury) generated in HWC for the 90th percentile risk ranged from 0.0003 to 7 pg/L. These correspond to the incremental increase in methylmercury concentrations in surface water resulting from the release of mercury across all combustor categories. Estimated and measured background concentrations reported by the MRTC indicated a range of dissolved methylmercury concentrations from 2.7 to 70 pg/L (U.S. EPA, 1997). These background levels reflect both natural and anthropogenic background concentrations. The ecotoxicological criteria used to generate methylmercury risk estimates in HWC and MRTC ranged from 42 to 57 pg/L for mammals and 33 to 100 pg/L for birds (U.S. EPA, 1997). When HWC incremental increases in concentrations are compared to the benchmarks, no apparent risks are indicated; however, when background concentrations are added to the incremental risk, the sum of these concentrations falls within the range of the criterion. This creates some uncertainty with the risk results generated for this analysis that indicate no risk for freshwater receptors.

Data Gaps. Ecotoxicological criteria were developed for constituents when sufficient data were available. In many cases, sufficient data were unavailable for a receptor/constituent combination and, therefore, the potential risk to this receptor could not be assessed. For instance, there were insufficient data to develop a dioxin criterion for the freshwater community. Because the risk results can only be interpreted within the context of available data, the absence of data should not be construed to indicate that adverse ecological effects will not occur.

Conservatism of Criteria Development. The conservatism of criteria development was appropriate for a screening analysis. However, because the approach is generally based on “no effects” data, these criteria tend to be fairly conservative. In site-based approaches, an approach is often used to allow for a level of effect that is predicted to be below a level of concern for reproducing populations (e.g., a low-effects approach). Since no-effects benchmarks are frequently an order of magnitude below a low-effects benchmark, the level of conservatism built into the ecological benchmarks (in mg/kg-day) is approximately an order of magnitude.

[25] Natural background is defined as low levels of constituents found in environmental media resulting from natural processes (e.g., mineral weathering). Anthropogenic background is defined as level of constituent found in environmental media resulting from the releases of compounds from industrial processes over time.

2.2.6.3 Criteria Development Issues Specific to Dioxin. In determining the potential risk to mammals and birds exposed to dioxin and furan congeners in the freshwater aquatic ecosystem, a tissue-based TEqCs method was used. The uncertainties associated with this approach are examined here; the details of methods and results of the approach are presented in Section 9.0. There are three primary issues of uncertainty in this approach: database uncertainty, BSAF uncertainty, and TEF uncertainty. Each of these sources is summarized below.

Database Uncertainty.

Regional Representation of Data: The database used to develop the BSAFs was adopted from work done by the Connecticut Department of Environmental Protection (CT DEP). Uncertainty is introduced by using these data because they were collected from one regional area. There is uncertainty associated with applying these data to represent the uptake of dioxin congeners in fish at the national level. Variables such as lipid content and organic carbon will vary across different regions and waterbodies. However, since BSAFs are purposely normalized for lipids and organic carbon, this should not be a limitation of using the data.

Pooled Data: The documents identified that reported the cumulative data from the CT DEP study pooled site media concentration data for congeners (with the exception of three congeners) in the soil, sediment, and fish tissues. This limited the ability to truly characterize the nature of contaminant uptake in fish using site-specific lipid contents, sediment organic carbon, and fish tissue concentrations. Data pooling generated uncertainty by prohibiting the characterization of the variability associated with the uptake of congeners into fish tissues on a site-specific basis.

Measurement Results: Two specific areas of uncertainty were indicated in the results: outliers and nondetection estimates. The CT DEP database generated some values that were inconsistent with trends seen for most congeners in the database (i.e., mean fish concentrations were significantly higher in preoperational conditions than in those reported during operational conditions) (see Section 5.4.1.6). Because there is no reasonable explanation for this observation, the preoperational data were not included in the development of BSAFs for two congeners (i.e., 1,2,3,4,7,8- HxCDF and 1,2,3,7,8,9-HxCDF). For these congeners, only mean fish tissue concentrations collected during operational conditions were used. By not using preoperational values in calculating the BSAFs, some uncertainty in BSAF development was generated. By eliminating these values from the data set, potential high-end exposures may not be characterized fully in the results. Second, measurements of dioxin concentrations in the ecological media (i.e., soil, sediment, and fish tissue) sometimes fell below the level of detection. In these cases, the concentration was reported at one-half of the detection level. This assumption may underpredict or overpredict actual concentrations in the media depending on the overall distribution. Further, because the data set had many nondetection measurements, it artificially creates a skewed concentration data distribution for some congeners, which introduces

uncertainty into the estimation of mean and median values used in the HWC analysis.

BSAF Uncertainty.

Equilibrium Partitioning: In calculating BSAFs, equilibrium between sediment concentrations and fish tissue concentrations is assumed. Considering the duration of the study (i.e., 4 years), these concentrations were probably closer to equilibrium than other studies conducted over shorter durations that were considered for BSAF derivation. However, since continued loading was occurring to the waterbodies over the 4 years of sampling, equilibrium conditions in these waterbodies cannot be confirmed. The disequilibrium conditions introduce a level of uncertainty into the calculated BSAFs.

Trophic Level: BSAFs vary depending on the trophic level of the fish. The pooling of fish data did not distinguish between fish trophic levels; therefore, only one generalized fish BSAF could be derived. The lack of characterization by trophic level introduces a level of uncertainty into BSAF metrics.

TEF Uncertainty.

Toxicity Equivalency Factors: TEFs are derived by comparing the toxicity response of like species upon exposure to different dioxin congeners relative to 2,3,7,8-TCDD. Most dioxin and furan congeners are equally or less toxic than 2,3,7,8-TCDD, and, therefore, the TEF for 2,3,7,8- TCDD is 1. TEFs have been derived for mammals and birds; however, there are several issues of uncertainty in applying these TEFs. Two major uncertainties have been identified: (1) TEFs are based on the assumption that the effects of dioxin and furan congeners are additive, and they do not consider possible synergistic or antagonistic relationships between various congeners; (2) TEFs do not account for pharmokinetics within the organism, which can influence the dose (i.e., the change in mixture composition related to elimination and in vivo transformation of congeners) . In other words, it is assumed that there is no change in the mixture composition from uptake to the receptor. The observation that metabolism plays a large part in the dose-response relationship makes this intrinsic assumption to applying TEFs an uncertainty in this analysis that may underestimate or overestimate the potential for adverse effects.

Taxa-specific TEFs: As mentioned previously, TEFs have been developed for only the broad categories of mammals and birds; however, even within these categories, interspecies variability in responses to exposure can differ by up to 3 orders of magnitude. For example, the toxicity responses of guinea pigs and hamsters induced by exposure to dioxin mixtures can differ by 1,000 (Kociba and Cabey, 1985). Further, TEFs are not specific to the lifestage of the receptor. Toxic responses are highly influenced by the age of the organism being exposed. The data available do not yet support the development of TEFs at this level of

resolution; however, the uncertainty associated with assuming that one TEF represents all mammals generates some uncertainty in the exposure estimates.

2.2.7 Confidence in Findings and Conclusions

The HWC-SERA represents a screening-level analysis designed to identify the potential for adverse effects in the suite of ecological receptors considered in the analysis (i.e., the analysis does not provide coverage for those ecological receptors not included in the analysis, such as endangered species). Consequently, although the HQ exceedances identified in the analysis should be interpreted as indicating the potential for adverse effects in representative receptors, they provide limited insight into the ecological significance of these effects.

To support the use of the HWC-SERA results as a tool in the decision making process, an evaluation of confidence in the findings was conducted. In determining the confidence in the findings of the HWC-SERA, two levels were evaluated: (1) the confidence in established criteria to adequately predict the potential for adverse effects and (2) the confidence with which potential ecological impacts, as determined by HQ exceedances, may be asserted. The first of these tasks is addressed in Section 9.2 for the benchmarks and criteria developed for the HWC-SERA. In Section 9.2, each criterion is assigned a confidence rank as a function of the quality and quantity of data used to derive the criterion. The focus of this section—confidence in the assertions supported by HQ exceedances—is directly related to the previous two sections that described the limitations of the analysis and the potential ecological significance of predicted effects. A review of factors relevant to the confidence in the HQ exceedances **to indicate the potential for adverse ecological effects** is provided below for mercury, dioxin, lead, and selenium.

2.2.7.1 <u>Mercury</u>.

- Exceedances occur across various combustor types; therefore, the spatial potential for adverse effects includes HWC units and habitats across the United States.

- The criteria used to generate HQs for waterbodies are derived from EPA reports that have undergone extensive peer review and are considered an authoritative source of wildlife criteria for mercury (U.S. EPA, 1997).

- Exceedances occur in more than one receptor taxa and in more than one media type (i.e., water, soil, and sediment); hence, the potential for impacts at multiple levels of the food web may contribute to the degree of potential adverse effects.

- Surface water assessments based on methylmercury concentrations showed no HQ exceedances. The same evaluation based on total dissolved mercury resulted in HQs greater than 1 (see Section 9.0). The EPA has derived total dissolved mercury benchmarks (U.S. EPA, 1997) and these are commonly used to evaluate water quality. This approach is appropriate when methylmercury values are not available. If fate and transport models can simulate mercury species that reasonably reflect waterbody characteristics, there is no need to use the total dissolved mercury benchmark. Because the surface water modeling conducted in

this risk assessment explicitly estimated methylmercury concentrations (see Section 5.3), methylmercury benchmarks were used to calculate HQs.

2.2.7.2 <u>Dioxin</u>.

- The exceedances seen for dioxin in terrestrial ecosystems reflect a lower level of confidence than the assessment of the freshwater ecosystem. Because bioaccumulation equivalents factors (BEFs) were not available to scale congener uptake into terrestrial prey items, all uptake factors were presumed to accumulate to the same extent as 2,3,7,8-TCDD congener. BEFs were available in the freshwater ecosystem to evaluate fish, and, for these prey items, the relative bioaccumulation potential across different dioxin and furan congeners could vary up to 2 orders of magnitude. Because BEFs were not used in the development of risk estimates for upper-level terrestrial consumers, a higher level of conservatism is included in the risk results.

- Exceedances are based on environmental concentrations that are only slightly (typically less than a factor of 10) above levels associated with a reduction in reproductive fitness in laboratory experiments. Hence, confidence in the exceedances as an indicator of potential impacts at the population level is relatively low. In addition, the use of a lowest observed adverse effect level (LOAEL) to support the ecological benchmarks (versus the no effects approach) would result in no target HQ exceedances for the baseline SERA.

- Although target HQ exceedances occur across combustor types, modeled exposure concentrations were above levels of concern for less than 1 percent of total areas for soil. The relatively low level of area exceedances may indicate a lower potential for ecological effects on a national basis.

- The criteria used to generate HQs for waterbodies and soils are derived from EPA reports that have undergone extensive peer review and are considered authoritative sources of wildlife criteria for dioxin.

- Exceedances occurred for only one receptor taxa (i.e., mammals). Data deficiencies have limited the number of criteria that could be developed for other receptors, particularly for aquatic life. The lack of data on lower trophic levels does not necessarily reduce our confidence in the risk results because: (1) dioxin biomagnifies significantly in both aquatic and terrestrial food webs and, therefore, higher trophic level predators may receive elevated exposures and (2) aquatic organisms (e.g., fish) have been shown to metabolize dioxin.

2.2.7.3 <u>Lead</u>.

- The target HQ exceedances are based on environmental concentrations that are slightly above a very conservative criterion (i.e., no effects approach) for soil community structure and function. Confidence that this exceedance represents a potentially serious ecological impact is low because: (1) the HQ exceedances

were less than 10, (2) the functional redundancy of soil species suggests that a no effects approach may be overprotective in most settings, and (3) the criterion is only slightly above the mean background concentration for lead.

- The soil criterion has not undergone extensive peer review and, based on the comparison with other similar activities, it is expected that the criterion will be revised to reflect a low-effects approach.

- Exceedances were indicated for only one assessment endpoint for one medium of concern. As a result, the potential for impacts at multiple trophic levels is considered low for the terrestrial ecosystem. Although damage to the soil community has been observed following lead contamination, impacts are typically seen at much higher exposure levels (>200 ppm).

2.2.7.4 Selenium.

- The target HQ exceedances are based on environmental concentrations that are slightly above the surface water criterion for the river otter (based on ingestion of contaminated fish and surface water). Because selenium is an essential element for many biological systems, and based on the low level of exceedance shown for a conservative exposure scenario (e.g., otter eats 100 percent contaminated fish), the confidence in the HQ exceedance to represent the potential for adverse effects is low. As with dioxin, the use of a LOAEL would result in HQ values below 1 for all waterbodies.

- Exceedances occur for only one combustor type (cement kilns). HQ exceedances are restricted to relatively small surface areas.

- The selenium criterion has not undergone extensive peer review.

- Exceedances occur for only one assessment endpoint for one medium of concern. As a result, the potential for impacts at multiple trophic levels is considered low for the aquatic ecosystem. Although damages to aquatic systems contaminated with selenium have been documented (Ohlendorf et al., 1986; Hothem and Ohlendorf, 1989; Saiki et al., 1993), receptors at risk typically include aquatic biota and birds. Because the exposure concentrations are below the criteria for these receptors, it is expected that the potential for population-level effects is low for selenium exposure in mammals living in aquatic ecosystems near HWC units.

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3.0 Risk Assessment Framework

The HWC risk analysis completed for the final rule characterizes both human health and ecological risk for the universe of HWC facilities located within the continental United States for the following combustor categories:

- Cement kilns
- Lightweight aggregate kilns
- Commercial incinerators
- On-site incinerators (large and small)
- Waste heat boilers
- Area sources.

Section 3.1 discusses the key components of the analytical approach used for this risk assessment and Section 3.2 describes the modeling process used.

The analytical approach described in this section differs in important ways from the approach used for the risk analysis for the proposed rule. Specifically, there are seven major differences:

- For the final rule risk analysis, 76 facilities were modeled, which is a substantial increase over the 11 facilities modeled for the proposed rule risk analysis. Moreover, the facilities modeled were selected in a statistically meaningful manner so that inferences could be made about the universe of facilities. That is, the 76 facilities modeled are representative of the larger universe.

- For the final rule risk analysis, all human receptor populations were enumerated except for the subsistence scenarios. The human populations for a given receptor were further divided into four age groups to allow risk characterization for children.

- The proposed rule risk analysis located specific residences and farms in the proximity of the modeled facility. In the risk analysis for the final rule, risk to the entire population was evaluated. Results of the modeling are presented as a distribution of exposure and of risk weighted by the affected populations.

- The basic risk results are based on central tendency values for all exposure parameters. The resulting distribution of risk captures most but not all of the variability in exposure and risk. Therefore, the risk analysis for the final rule also contains an assessment of the variability in selected exposure parameters and

models their influence on exposure and risk values. This exposure variability assessment was conducted for the important risk-driving exposure pathways.

- The final rule risk analysis includes a multipathway risk analysis for three species of mercury.

- The final rule risk analysis includes a lead analysis. Blood lead levels were modeled for the 0- to 5-yr-old age group. This allowed lead risk levels to be characterized in terms of the number of individuals in the 0- to 5-yr-old age group who exceeded a blood lead level of concern.

- Finally, the risk assessment for the final rule includes a comprehensive screening-level analysis of ecotoxicological risks.

3.1 Analytical Overview

This section provides an overview of the analytical approach used to evaluate both human health and ecological risk for the final rule. Emphasis is placed on introducing those techniques and approaches related to exposure assessment and risk characterization that were developed specifically for the HWC risk analysis.

3.1.1 Facility Selection

A critical requirement in developing the HWC risk analysis methodology was that it allow clear statistical statements to be made concerning the representativeness of the risk results for the universe of HWC facilities (those within the continental United States). The methodology developed for this analysis specifically addressed this representativeness goal by incorporating a facility-specific modeling approach and using stratified random sampling to select the facilities to be modeled.

3.1.1.1 Facility-Specific Modeling Approach. The facility-specific modeling approach combined the site-specific analyses of facility emissions, fate and transport, and exposed receptor populations with national data on exposure factors to generate estimates of exposure and risk.

3.1.1.2 Stratified Random Sampling Approach. The stratified random sampling approach was used to select specific facilities from the HWC universe, which forms the basis of the risk analysis. The HWC universe was stratified according to the combustor categories of interest (e.g., cement kilns and waste heat boilers), and facilities to be modeled were randomly sampled from those strata. The use of random sampling allowed clear statistical statements to be made concerning the representativeness of risk results generated for the modeled facilities (i.e., how representative those results are of the universe of HWC facilities). Sampling error, which results from not having sampled all of the facilities in the universe, could be quantified by placing confidence intervals (reflecting sampling error) around specific risk estimates.

Stratified random sampling was conducted separately for each combustor category and was continued within each category until a sufficient number of facilities had been sampled to provide a 90 percent probability that at least one selected facility was a high-risk facility. With

random sampling, a quantitative statistical criterion (i.e., a 90 percent probability of selecting a high-risk facility) could be identified and reflected directly in the selection of facilities.

3.1.2 Exposure Assessment

The exposure assessment examined the exposure of human receptor populations to those constituents released to the atmosphere by HWC facilities that can be quantified. Constituents assessed were

 # Seven congeners of chlorinated dioxin

2,4,7,8 - Tetrachlorodibenzo(*p*)dioxin
1,2,3,7,8- Pentachlorodibenzo(*p*)dioxin
1,2,3,7,8,9 - Hexachlorodibenzo(*p*)dioxin
1,2,3,4,7,8, - Hexachlorodibenzo(*p*)dioxin
1,2,3,6,7,8 - Hexachlorodibenzo(*p*)dioxin
1,2,3,4,6,7,8 - Heptachlorodibenzo(*p*)dioxin
1,2,3,4,5,7,8,9 - Octachlorodibenzo(*p*)dioxin

 # Ten congeners of chlorinated furan

2,3,7,8 - Tetrachlorodibenzo(*p*)furan
1,2,3,7,8- Pentachlorodibenzo(*p*)furan
2,3,4,7,8- Pentachlorodibenzo(*p*)furan
1,2,3,6,7,8- Hexachlorodibenzo(*p*)furan
2,3,4,6,7,8- Hexachlorodibenzo(*p*)furan
1,2,3,4,7,8- Hexachlorodibenzo(*p*)furan
1,2,3,7,8,9- Hexachlorodibenzo(*p*)furan
1,2,3,4,6,7,8- Heptachlorodibenzo(*p*)furan
1,2,3,4,7,8,9- Heptachlorodibenzo(*p*)furan
1,2,3,4,6,7,8,9- Octachlorodibenzo(*p*)furan

 # Three species of mercury

Elemental mercury
Divalent mercury
Methylmercury

 # Eleven metals that were modeled for the proposed rule

Antimony
Chromium III, VI
Arsenic
Lead
Barium
Nickel
Beryllium
Selenium
Cadmium
Silver
Thallium

 # Three additional metals modeled for the final rule

Cobalt
Copper
Manganese

- **Particulate matter**
  PM_{10}
  $PM_{2.5}$

- **Hydrochloric acid**

- **Chlorine gas**

The HWC risk analysis assessed human health risks for various receptor populations. A critical component of the analysis was the location and density of receptor populations relative to the modeled facilities. Air modeling results for a given facility define a pattern of air concentration and deposition values for constituents of concern within the study area. For this final rule analysis, these detailed air model results were linked to spatially refined population estimates and land use characteristics. Specifically, each modeled study area (comprising the modeled facility and the surrounding 20-km radius area) was divided into 16 sectors using four concentric rings combined with a north-south and east-west transect (see Section 4.3).

A geographic information system (GIS) platform was used to enhance 16-sector spatial resolution since key site attributes linked to exposure could be defined at the sector level. These attributes were: air model results, density of receptor populations, topography, waterbodies, watersheds, soils, and land use type. The ability to define these attributes at the sector level provided the level of resolution required to generate sector-level projections of both individual and population risk for the human health component of the analysis as well as sector-level characterization of potential ecological impacts.

To further enhance exposure assessment with regard to human health for the final rule, four separate age groups were used to characterize risk. The use of four age groups (0-5, 6-11, 12-19, and >19 years) allowed age-dependent differences in exposure parameters to be reflected in both exposure assessment and risk characterization. The U.S. Census contains data with sufficient age-group resolution to allow the generation of population estimates at the sector level for these age groups. Also included in the analysis for selected constituents (e.g., dioxins and furans) is an assessment of nursing infants exposed via maternal milk.

3.1.3 Human Health Risk Characterization

The risk assessment methodology implemented for the final rule characterized risks to both human and ecological receptors located within 20 km of facilities within the HWC universe. There was no consideration of risks resulting from atmospheric constituents transported beyond the 20-km study areas. Inferences about risks posed by the universe of HWC facilities were made based on risk estimates generated for the subset of modeled facilities. The statistical analysis that applied facility sample weights and population weights to the sector-level risk results based on a stratified random sample of facilities was conducted using SUDAAN, a statistical analysis software package developed by RTI. All risk estimates generated for the final rule are presented according to the key combustor categories.

Because risks were generated at the sector level through the use of the 16-sector template, sector-level risk estimates form the basis for projecting both individual and population

risk estimates for the human receptor as well as ecological risk estimates. The HWC analysis was designed to characterize two broad categories of human health risk: individual and population. For individual risk, emphasis was placed on characterizing distribution of individual risk within the receptor population (e.g., risk to the 50th percentile individual within the population and risk for the 90th, 95th, and 99th percentile individual). Population risk was evaluated both for local populations (those individuals residing within 20 km of an HWC facility) and the national population (those individuals who consume agricultural commodities produced within 20 km of an HWC facility but who reside outside the 20-km study area).

A significant enhancement in individual risk characterization implemented for the final rule was the use of population-weighted individual risk distributions for the identification of specific individual risk percentiles. For the final rule, population-weighted individual risk estimates were used as the basis for a cumulative individual risk distribution rather than unweighted sector estimates. Each sector-level individual risk estimate was first weighted to reflect the number of individuals from the receptor population of interest located within that sector. This approach allowed the distribution of individuals across a study area to be reflected in the cumulative risk distributions used to identify specific individual risk percentiles.

The population-weighted individual risk approach can be applied only to enumerated receptor populations. For those populations that could not be enumerated using Census data (e.g., subsistence scenarios), unweighted sector-level individual risk estimates were used to form the cumulative risk distributions from which individual risk percentiles were selected.

Individual risk estimates were generated for those constituents with carcinogenic effects using standard risk assessment techniques. For noncancer effects, exposures were compared to a reference dose and expressed as a ratio or hazard quotient. In addition, for lead, individual exposures in children were generated as body burden levels in blood. Furthermore, an incremental margin of exposure was used to assess the potential for noncancer effects for dioxin. This was done for infants exposed to dioxin through breast milk as well as for the full set of receptor populations and age groups considered in this risk analysis.

Individual risk estimates were generated for those constituents identified as having carcinogenic effects based on the lifetime average daily dose combined with a cancer slope factor. The CSF is an upper bound estimate of the probability of an individual developing cancer over a lifetime per unit intake of a contaminant. Overall cancer risk was estimated assuming additivity.

Individual risk estimates were generated for those constituents identified as having non-cancer effects based on the ratio of the average daily dose (ADD) to a reference dose or the ratio of annual average air concentrations to a reference concentration. The ratio representing individual risk estimates is the hazard quotient. The reference dose is an estimate of the average daily dose that is without appreciable risk of deleterious effects during a lifetime. An overall hazard index was generated as the sum of the constituent-specific hazard quotients.

The HWC risk analysis completed for the final rule characterizes population risk resulting from human exposure to constituents deposited within HWC study areas. The selection of population risk categories for the final rule focused on those health effects that could be

quantified. With regard to carcinogenic risk, two types of statistical cancer incidence estimates are presented:

- Agricultural commodity statistical cancer incidence analysis estimates the number of statistical cancer incidence cases occurring nationally as a result of the public's consumption of beef, milk, and pork raised within HWC study areas. These agriculture commodities have been impacted by dioxin released from their local HWC facility.

- Local statistical cancer incidence analysis estimates the number of statistical cancer cases occurring strictly within the HWC study areas as a result of local (i.e., individuals living within study areas) exposure to all modeled carcinogens. This analysis considers all modeled exposure pathways including the ingestion of home-produced agricultural commodities.

Besides these cancer population risk analyses, the HWC risk analysis also included population risk analyses, including the number of children exposed to lead above health-based levels and adverse health effects resulting from inhalation of PM_{10} and $PM_{2.5}$.

In addition to the above quantitative population risk categories, semiquantitative population risk statements are also provided for exposure of recreational fishers to mercury through fish ingestion. This population risk category estimates the number of recreational fishers potentially engaging in fishing activity in at-risk waterbodies (i.e., modeled waterbodies with individual risk levels for fish ingestion above the health benchmark level [HBL] for methylmercury).

3.1.4 Ecological Risk Characterization

The ecological risk component of the HWC analysis assessed the potential for adverse impacts to both aquatic and terrestrial receptors as a result of exposure to modeled constituents released from HWC facilities. The ecological risk analysis considered impacts only to ecological receptors located primarily within study areas. This analysis was based on the development of criteria (e.g., protective media concentrations) that, in turn, were based on ecological benchmarks (e.g., no observed adverse effects levels or NOAELs). Modeled media concentrations (including soil, surface water, and sediment) were compared to these ecological criteria at the sector level to determine whether the potential for ecological impacts existed within a given study area (i.e., do HQs exceed unity).

For dioxin, a different approach was taken to address ecological risks in aquatic systems. Instead of comparing modeled water concentrations to media-specific ecotoxicological criteria, the dietary intake of dioxins (expressed as toxicity equivalents or TEQs) for receptor organisms was compared directly to the ecotoxicological benchmarks for 2,3,7,8,-TCDD. This approach allowed the assessment of ecological exposures for all 2,3,7,8-chlorine-substituted congeners, taking into consideration the differential toxicity and bioaccumulation of different congeners in the aquatic food chain.

A critical factor in determining the significance of HQ exceedances is the spatial pattern of those exceedances. The use of the 16-sector template allowed spatial patterns to be identified and evaluated for their potential ecological significance.

Although this ecological analysis was based on a comprehensive set of ecological criteria, it is a screening-level analysis designed to identify the **potential** for adverse impacts to ecological receptors and does not provide quantitative results as does the human health evaluation.

As with the human health analysis, ecological risk results generated for modeled HWC facilities are facility-sample-weighted to represent the universe of HWC facilities (see discussion in Section 3.1.3).

3.2 Overview of Modeling Process

The modeling process used in this human health and ecological risk assessment of HWC facilities involves a series of steps beginning with selection of HWC facilities to be modeled and ending with characterization of human and ecological risks. The purpose of this section is twofold: (1) to provide an overview of the steps involved in the modeling process and (2) to provide a map to the discussion of modeling methodologies presented in subsequent sections of this report.

Figure 3-1 shows the steps involved in the modeling process used and groups those steps into six broad categories:

- Characterizing modeled facilities
- Determining environmental media concentrations
- Determining food chain concentrations
- Calculating human intake and dose
- Characterizing human health risks
- Characterizing ecological risks.

These six categories define the main components of the modeling process. Figure 3-1 also cross references each of these components to the appropriate section of this document containing greater detail.

3.2.1 Characterizing Modeled Facilities

The HWC risk assessment methodology is based on a facility-specific modeling approach; therefore, the first step in the modeling process is to define the universe of all HWC facilities and then select the facilities to be modeled from this universe. Stratified random sampling was used to select facilities for the final rule, which resulted in 66 facilities being selected. These 66 were combined with 10 of the 11 facilities modeled for the proposed rule, resulting in 76 facilities modeled for this risk analysis. These 76 facilities represent the universe of incinerator, cement kiln, and lightweight aggregate kiln source categories (see Section 4.1.1).

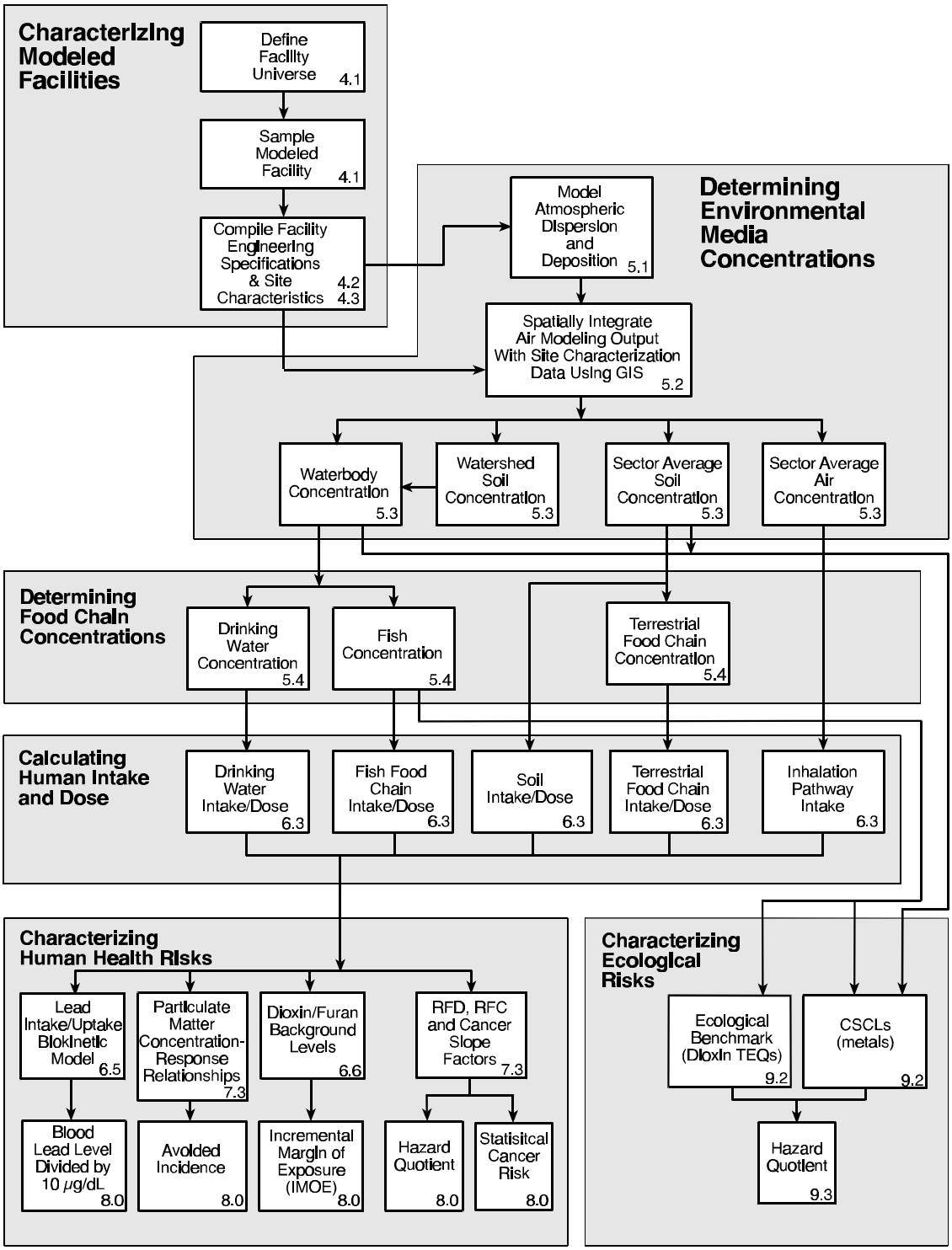

Figure 3-1. Overview of risk assessment framework.

3.2.2 Determining Environmental Media Concentrations

Air dispersion and deposition modeling was conducted using EPA's Industrial Source Complex Model - Short Term Version 3 (ISCST3) to arrive at normalized air concentrations and deposition fluxes (see Section 5.1). Modeling was based on a 1-g/s emission rate (a normalized emission rate). The air modeling grid data were then converted using a GIS into average normalized values for geographic features in the study area: sectors, watersheds, and waterbodies (Section 5.2). These normalized values were then combined with facility-specific emissions data to calculate waterbody concentrations, watershed soil concentrations, sector air concentrations, and sector soil concentrations (Section 5.3). Sector soils, watershed soils, and waterbody concentrations were modeled using the 1993 Addendum to the Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions for all constituents except mercury (U.S. EPA, 1993). Mercury species in soils and waterbodies were modeled in two different ways. Mercury modeling for the aquatic food chain pathway (watershed-waterbody-fish tissue) was done using IEM-2M based on the 1997 *Mercury Study Report to Congress* (U.S. EPA, 1997) (see Section 5.3.3.2). The IEM-2M methodology was not used to model mercury in sector soils and the drinking water pathway; they were modeled using a version of the IEM-2 methodology that was modified specifically for this risk assessment (see Appendix F).

3.2.3 Determining Food Chain Concentrations

The media concentrations obtained in the previous step were used to calculate food chain concentrations as follows (Section 5.4):

- Terrestrial food chain concentrations were based on air and soil concentrations for each sector.

- Drinking water concentrations were based on waterbody concentrations. The majority of modeled facilities had at least one waterbody identified as the drinking water source for a community.

- Fish tissue concentrations were based on modeled waterbody concentrations for recreational and subsistence fishers and on farm pond concentrations for subsistence farmer populations.

Media and food chain concentrations calculated in the previous step were combined with intake rates, which were generated for each of the modeled pathways to produce constituent-specific exposure estimates for those pathways. Intake rates refer to the modeled rates of ingestion or inhalation that were generated for specific types of media or food commodities (e.g., incidental ingestion rates for soil generated for the adult commercial beef farmer). Exposure estimates, which were calculated separately for each constituent/pathway combination, represent the rate of exposure to a specific constituent that results from the ingestion or inhalation of a specific type of media or food commodity.

3.2.4 Modeling Human Exposure

The HWC risk analysis assessed exposure for a number of receptors, each of which was modeled using a suite of exposure pathways designed to capture the receptor's activity/behavior pattern. Receptors modeled in the analysis and their pathways are listed in Table 3-1. Receptors are defined as follows:

- Residents: individuals residing within HWC study areas
- Home gardeners: individuals residing within HWC study areas who engage in home gardening activity
- Recreational fishers: individuals residing within HWC study areas who engage in recreational fishing activity
- Commercial beef farmers: individuals who operate commercial beef farms within HWC study areas
- Commercial pork farmers: individuals who operate commercial hog farms within HWC study areas
- Commercial dairy farmers: individuals who operate commercial dairy farms within HWC study areas
- Commercial produce farmers: individuals who operate commercial produce farms within HWC study areas
- Subsistence fishers: individuals who reside within HWC study areas and obtain all of their dietary fish intake from home-caught fish
- Subsistence farmers: individuals who reside within HWC study areas and obtain all of their dietary intake from home-produced food items.

To gain greater resolution in assessing exposure for the receptors listed above, each receptor was further differentiated into four age groups (i.e., 0-5, 6-11, 12-19, and >19 yr), and separate exposure estimates were generated for each age group. In addition, for dioxins, exposure to human infants from maternal milk was modeled.

Exposure was calculated based on intake values for each of the pathways presented in Table 3-1. Two different types of exposure estimates were generated depending on the type of health effect being characterized. Carcinogenic health effects are characterized using exposure estimates that are averaged over the lifetime of the individual (LADDs). Noncancer effects are characterized using exposure estimates that are averaged over the relevant averaging period (nominally 1 year) during which the exposure occurs (ADDs). All exposure estimates are expressed as daily doses for a specific constituent normalized for the body weight of the receptor (i.e., mg *constituent*/kg *body weight* per day or mg/kg-d).

Table 3-1. Receptors Modeled by Pathways

Receptors	Inhalation of ambient air	Incidental soil ingestion	Ingestion of drinking water	Ingestion of home-produced fruits and vegetables	Ingestion of home-caught fish	Ingestion of home-produced beef	Ingestion of home-produced pork	Ingestion of home-produced milk	Ingestion of home-produced chicken
Residents	✓	✓	✓						
Home gardeners	✓	✓	✓	✓					
Recreational fishers	✓	✓	✓		✓				
Commercial beef farmers	✓	✓	✓			✓			
Commercial hog farmers	✓	✓	✓				✓		
Commercial dairy farmers	✓	✓	✓					✓	
Commercial produce farmers	✓	✓	✓	✓					
Subsistence fishers	✓	✓	✓		✓				
Subsistence farmers	✓	✓	✓	✓	✓	✓	✓	✓	✓

3.2.5 Characterizing Human Health Risks

The HWC risk analysis assessed risks for a number of different human health effects, including cancer, noncancer effects, health effects from lead, health effects from PM, and noncancer effects associated with dioxin/furan exposure. A combination of both individual and population-level risk descriptors were used in characterizing risks for these health effects.

3.2.5.1 Cancer. Individual cancer risk was evaluated by multiplying the LADD estimates generated for each receptor/pathway by the appropriate cancer slope factor. Cancer slope factors were derived from either human or animal data and relate the level of exposure to a particular constituent to the lifetime excess cancer risk that results from that exposure. In developing cancer slope factors, the relationship between exposure and risk is generally assumed to be linear with the slope factor representing the upper bound on the slope of the dose-response curve in the low-dose region where modeled human exposure typically occurs. Total individual cancer risk was determined for each receptor, assuming additivity across constituents.

In the HWC risk analysis, population-level cancer risk is characterized using annual lifetime cancer incidence estimates. These estimates represent the excess number of cancer cases predicted to occur due to emissions released from the facility under evaluation during a single model year. Accordingly, annual incidence is estimated by dividing the total lifetime cancer incidence by the exposure duration.

3.2.5.2 Noncancer Effects. Individual noncancer risk for ingestion pathways was evaluated by dividing the ADD estimates generated for each receptor/pathway by the appropriate RfD to produce a hazard quotient. Inhalation pathways were evaluated for noncancer effects by dividing modeled ambient air concentrations for specific constituents by the corresponding RfC to produce inhalation hazard quotients. RfDs and RfCs, both of which can be based either on human or animal data, represent estimates of daily exposure to the human population, including sensitive subgroups, that are likely to be without an appreciable risk of deleterious effects during a lifetime. Ingestion and inhalation hazard indices were generated for each receptor by adding constituent-specific hazard quotients by route of exposure.

3.2.5.3 Health Effects from Lead. Risk resulting from exposure to lead was assessed for the child age group (i.e., 0 to 5 years old) of every receptor population evaluated in the analysis. Risk for this age group was assessed by modeling body burdens (as blood lead levels) and comparing these levels to the level at which efforts aimed at prevention are indicated (i.e., 10 μg lead/dL blood). In addition to characterizing individual risk levels for lead exposure in the modeled receptor populations, this analysis included population risk estimates expressed as the annual excess incidence of elevated blood lead (i.e., above 10 µg/dL).

3.2.5.4 Health Effects from PM. Risk associated with inhalation exposure to particulate matter was evaluated in the elderly and the general population through the use of concentration-response functions derived from human epidemiological studies that describe the incidence of mortality and morbidity avoided annually due to an incremental reduction in PM. The PM analysis generates only population-level risk estimates.

3.2.5.5 <u>Noncancer Effects from Dioxin/Furan Exposure</u>. Potential noncancer risk associated with dioxin/furan exposure is evaluated using an incremental margin of exposure (incremental MOE) approach. With this approach, modeled exposure levels for specific receptors, expressed as 2,3,7,8-TCDD toxicity equivalents (TEQs), were compared to background TEQ exposure levels in the general population and expressed as a ratio. In addition to generating incremental MOE estimates for each of the four age groups within each receptor, this analysis generated incremental MOE estimates for infant receptors who are exposed to dioxin/furan through the ingestion of breast milk. As a measure of hazard, the incremental MOE presumes that background exposures pose only a de minimis level of risk.

3.2.6 Characterizing Ecological Risks

The ecological risk assessment is a screening-level analysis designed to identify the potential for adverse ecological effects. The process is based on current EPA guidelines for ecological risk assessment and begins with the selection of assessment endpoints (i.e., the actual environmental values to be protected).

The assessment endpoints are defined by two key elements: (1) a valued ecological entity such as a wildlife species, and (2) an attribute of that entity that is important to protect (e.g., reproductive fitness). Once the assessment endpoints are defined, ecological receptors that may be susceptible to the chemical constituents released from HWC facilities are selected. These receptors include assemblages of species typical of soil, sediment, and surface water communities as well as representative species of mammals and birds found in most parts of the contiguous United States.

For each constituent, ecotoxicological data were reviewed to derive benchmarks (in units of dose) and ecotoxicological criteria below which adverse ecological effects are presumed to be negligible. Ecological benchmarks derived for representative species of birds and mammals (generally no observed adverse effect levels, or NOAELs) were used to calculate ecotoxicological criteria using the assumption that all food items originate from the same contaminated area. For species associated with aquatic habitats (e.g., riverine), the ecotoxicological criteria are given in units of surface water concentration and include ingestion of contaminated water and biota (e.g., fish and aquatic invertebrates). For species associated with terrestrial habitats, the ecotoxicological criteria are given in units of soil concentration and include ingestion of contaminated soil and terrestrial biota (e.g., vascular plants, earthworms). The ecotoxicological criteria for assemblages of species typical of soil, sediment, and surface water communities were derived using statistical inference on ecotoxicological data on individual species attributed to the community. For all metal constituents evaluated in this analysis, the media-specific ecotoxicological criteria were compared to the media concentrations predicted using the environmental fate and transport model with an HQ approach. The HQ approach is similar to the approach used in noncancer health risk assessment (i.e., HQ > 1 indicates the potential for adverse ecological effects).

For dioxin/furan congeners in aquatic systems, a toxicity equivalency concentration approach was used so that congener-specific differences in toxicity and bioaccumulation could be considered. Consequently, the HQ approach for dioxin compared the predicted TEC dose (as an administered dose) to the ecological benchmarks for the representative species evaluated in

this screening analysis. For the terrestrial system, a soil TEQ concentration, which reflects only the application of TEFs, was compared to a soil ecotoxicological criterion for 2,3,7,8-TCDD, similar to the approach taken for metals. This approach does not consider the differential bioaccumulation of different congeners and, as such, is likely to be exceedingly conservative.

3.3 References

U.S. EPA (Environmental Protection Agency). 1993. *Addendum to Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions* (External Review Draft). EPA/600/AP-93/003. Exposure Assessment Group, Office of Health and Environmental Assessment, Washington, DC.

U.S. EPA (Environmental Protection Agency). 1997. *Mercury Study Report to Congress. Volume III - Fate and Transport of Mercury in the Environment*. EPA 452/R-97/005. Office of Air Quality Planning and Standards and Office of Research and Development, Washington, DC.

4.0 Characterization of Modeled Facilities

The risk assessment for the final rule is based on a facility-specific modeling approach. This section presents the methodology used to select modeled facilities and obtain the site data required to characterize those facilities. Section 4.1 describes the approach used for selecting modeled facilities including the definition of the HWC facility universe. Section 4.2 describes the facility-specific engineering and annual emissions data used in conducting air modeling for each of the modeled facilities. Section 4.3 presents the methodologies used to obtain site data for the study area surrounding each of the modeled HWC facilities including delineation of key topographical features and estimation of human and livestock populations. Figure 4-1 diagrams the relationships between specific analytical tasks related to facility characterization.

4.1 Selection of Modeled Facilities

This section presents the methodology used to define the HWC facility universe and randomly select facilities modeled for risk analysis.

4.1.1 Facility Universe

A critical step in developing the HWC risk analysis involved defining the facility universe that the risk analysis would represent. This universe was developed initially as part of the proposed rule-making effort for the HWC risk analysis. After the initial HWC facility universe had been defined, it was updated to reflect new information on facility closures and entrants to the market. In addition, in the fall of 1997, site visits were made to state environmental and EPA Regional offices to identify additional information that could be used to update the facility universe (e.g., changes in the operational status of existing facilities or identification of new facilities). The HWC facility universe used for the final rule reflects both the public comments and the information gathered during this data collection effort. It includes all HWC facilities located within the continental United States that were operational in 1997. For a more detailed discussion of the facility universe, the reader is referred to *Assessment of the Potential Costs, Benefits, and Other Impacts of the Hazardous Waste Combustion MACT Standards* (U.S. EPA, 1999a).

Facilities outside the continental United States were not included in the facility universe because critical data used in site characterization (e.g., U.S. Census data, Census of Agriculture data, and GIS land use coverage data) were not readily available for them. Therefore, the risk

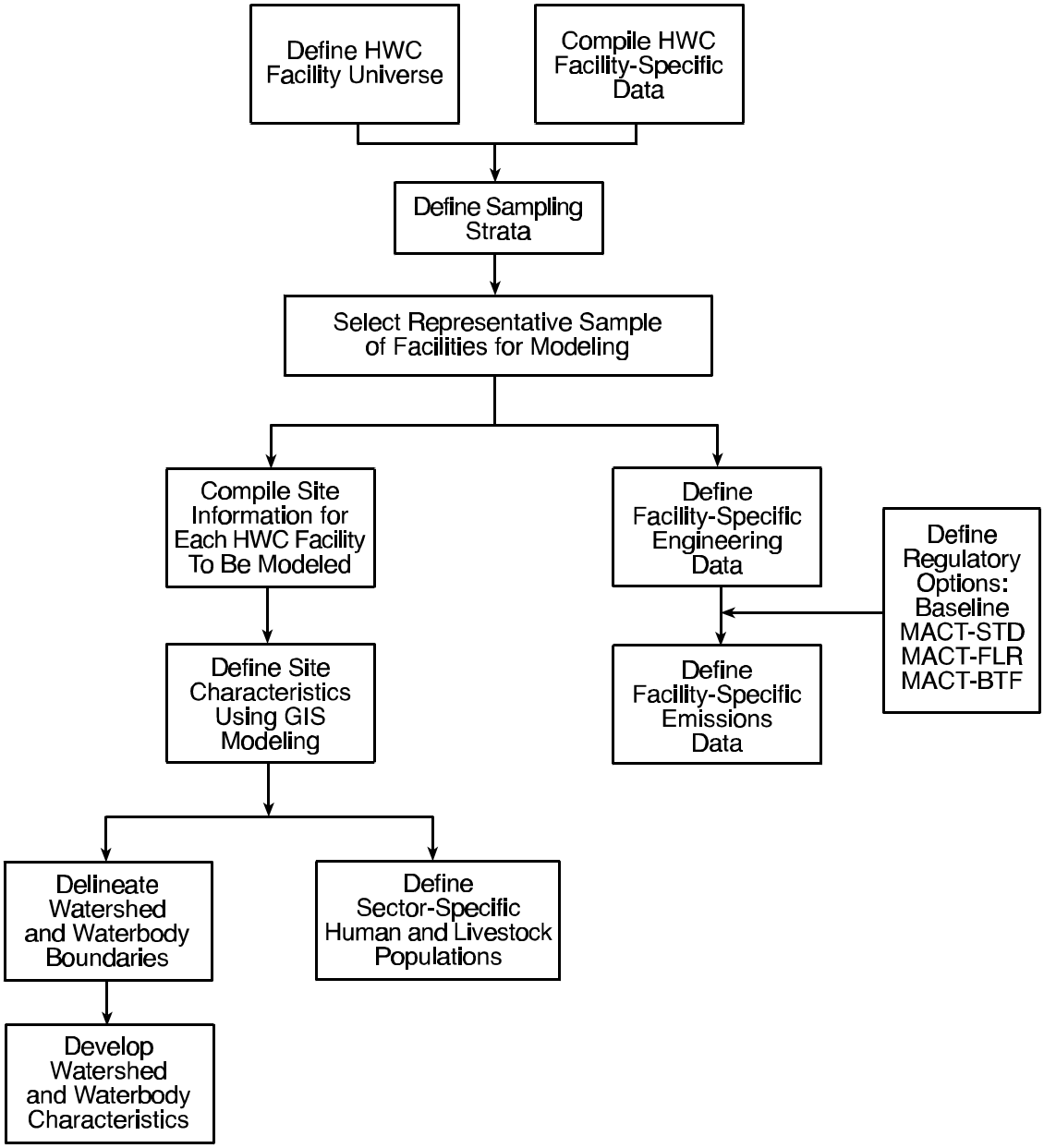

Figure 4-1. Overview of analytical tasks completed for facility characterization.

assessment applies only to those facilities located in the contiguous United States and not to facilities located outside the contiguous United States such as Puerto Rico and Johnson Altoll[1].

4.1.2 Facility Categories

Combustor facilities contained in the HWC universe fall into one of three source categories:

- Cement kilns
- Lightweight aggregate kilns
- Incinerators.

Because the facilities in each of these source categories are linked to specific commercial activities, they tend to share more operational attributes with other facilities in their particular category than with facilities in other categories. Therefore, in evaluating the potential benefits associated with establishing emissions control standards for HWC facilities, EPA initially stratified the facility universe into categories based on these three source categories and separately evaluated the benefits for each. The proposed rule presented risk results for these three source categories.

Combustor Categories added for the Final Rule

- Commercial incinerators
- On-site incinerators
- Waste heat boilers
- Area sources

EPA retained these three source categories as the basis for the final rule analysis. To provide greater resolution in identifying those facility attributes that are correlated with specific categories of risk, however, EPA further stratified the HWC universe by adding several combustor categories to the analysis for the final rule. Some of these new combustor categories are mutually exclusive (e.g., on-site and commercial incinerators), while others extend across several different categories to group facilities that share a particular operational attribute (e.g., waste heat recovery boilers). The following combustor categories have been added for this analysis:

- **Commercial incinerators:** Function specifically as commercial facilities that earn revenue by burning hazardous waste. As such, the incinerators in this combustor category are often larger (i.e., higher throughput) and burn a greater variety of wastes than those in the on-site category. General differences between the commercial and on-site incinerators with regard to facility attributes (e.g., emissions rates and stack parameters) raised interest in stratifying the incinerator category to determine whether the different incinerator categories could be linked to specific patterns of risk.

[1] One small on-site incinerator facility located in Alaska (AK0000094888) was included in the facility universe, despite the fact that it is not located within the contiguous United States. Inclusion of this facility in the sample frame for small on-site incineration does not introduce significant error due to size of the small on-site incineration facility universe.

- **On-site incinerators:** As part of a larger commercial manufacturing operation, handle hazardous wastes generated specifically by that operation (these facilities do not burn wastes from other companies for profit). Because on-site facilities play a support role and are not dependent on earning profit through hazardous waste combustion, they are often smaller than commercial facilities (their size is dependent on the type of operation they support) and burn a limited variety of wastes. To gain additional resolution in identifying facility attributes linked to risk, the on-site incinerator combustor category was further stratified for the final rule into large on-site incinerators (those with stack gas exhaust volumes greater than 20,000 acfm) and small on-site incinerators (those with stack gas exhaust volumes less than 20,000 acfm).

- **Waste heat boilers:** Recover excess heat generated in the incineration process as a thermal source for industrial applications rather than releasing it directly to the environment. Only a subset of incinerators have WHBs—cement kilns and LWAK facilities do not. Concerns have surfaced that the operating parameters associated with waste heat boilers may result in greater dioxin/furan formation. Therefore, the WHB category was selected for inclusion in the final rule. Because dioxin/furan formation is the focus for this combustor category, all those risk results involving dioxin-TEQ have WHBs broken out as a separate category.

- **Area sources:** Facilities with relatively low emission rates of HAPs (facilities with relatively high HAP emission rates are major sources). The Clean Air Act definition of an area source was used in the HWC risk analysis to identify area sources: those facilities having an emission rate for a single HAP of less than 10 tons per year or an emissions rate for combined HAPs of less than 25 tons per year. The area source stratification was included in the HWC risk analysis because area sources are not always subject to MACT standards. To gain greater resolution in evaluating area sources, these facilities were further stratified for purposes of the HWC risk analysis into area source cement kilns and area source incinerators (no area source LWAKs were identified). Because the statutory definition of an area source is based on total facility (industrial complex) emissions, it was not possible to distinguish on-site incinerators located at small industrial complexes that are classified as area sources from on-site incinerators located at large industrial complexes that are classified as major sources. Therefore, most on-site incinerators were excluded from the area source incinerator category.

4.1.3 Facility Definition

For the purpose of this risk analysis, a facility is defined as an industrial complex consisting of one or more hazardous waste combustion units (e.g., incinerators, cement kilns) vented through one or more stacks. For facilities with more than one combustion unit and more

than one stack, each stack was modeled separately[2]. Subsequently, the air concentrations and deposition resulting from the emissions from these combustion units/stacks were summed to provide air concentration and deposition values for the total facility. Exposure and risk values were attributed to this combined facility impact. Therefore, for this analysis, the hazardous waste combustion units are described in terms of facilities, and risk results are reported accordingly.

4.1.4 Facility Sample Size

The proposed rule included risk characterization for a purposive sample of 11 HWC facilities. These 11 facilities were selected to provide coverage for the following factors: (1) HWC combustor categories being considered, (2) location of the HWC facilities (land use, topography, meteorological conditions), and (3) facility attributes (e.g., stack gas exhaust volume). Comments to the proposed rule identified the need for a modeled facility selection strategy that was more representative of the universe of HWC facilities than the original 11 HWC facilities (10 of which were retained in the final rule risk analysis)[3]. This requirement resulted in an approach for the final rule that utilized stratified random sampling for the selection of additional modeled facilities. This approach allowed statistical statements to be made regarding representativeness of the risk analysis.

The sample design chosen for the final rule was a stratified, one-stage cluster sample, for which the facilities were selected without replacement. The facilities were considered clusters since the final sampling units were the 16 sectors within each facility study area (see Section 4.3). The facility sampling strata correspond to the six combustor categories of interest:

- Cement kilns
- Lightweight aggregate kilns
- Commercial incinerators
- Large on-site incinerators
- Small on-site incinerators
- Waste heat boilers (a subset of incinerators).

Area sources were not treated as separate strata for the purpose of sampling due to difficulties in defining area source universe.

Sample sizes for each combustor category were based on the goal of having a 90 percent probability of selecting a facility from the top 10 percent of facilities within a given combustor category with regard to risk (i.e., a 90 percent probability of having included a “high-risk” facility in the sample). Table 4-1 presents the sample sizes established for each combustor category and the resulting probabilities for selecting at least one high-risk facility from that combustor category. Because waste heat boilers are a subset of the incinerators (but were

[2] There are a few cases in which an industrial complex has more than one combustion unit. If these combustion units do not all belong to the same source category, the emissions were apportioned to the different source categories and the industrial complex was treated as separate facilities, one for each of the source categories coexisting at the industrial complex.

[3] The 11th facility is undergoing RCRA closure and is no longer burning hazardous waste.

Table 4-1. Hazardous Waste Combustion Facility Stratum Sizes and Sample Sizes

Combustion Facility Category	Stratum Size	Random Sample Size	Original Sample Size	Total Sample Size	High-End Sampling Probability[a]
Cement Kilns	18	10	5	15	98
Lightweight Aggregate Kilns	5	3	2	5	100
Commercial Incinerators					
Including Waste Heat Boilers	20	11	2	13	97
Excluding Waste Heat Boilers	12	7	2	9	95
Large On-Site Incinerators					
Including Waste Heat Boilers	43	17	1	18	94
Excluding Waste Heat Boilers	36	15	0	15	90
Small On-Site Incinerators					
Including Waste Heat Boilers	79	25	0	25	96
Excluding Waste Heat Boilers	65	16	0	16	88
Incinerators with Waste Heat Boilers					

[a]Probability that a facility that lies in the upper 10% of the distribution of risk will be sampled.

sampled as an independent category), information for incinerators is presented in Table 4-1 for each incinerator category as a whole (with waste heat boilers included), each incinerator category without waste heat boilers included, and waste heat boilers as a whole (aggregated across the three incinerator categories). Sampling was conducted separately to provide coverage for each of these different incinerator/waste heat boiler combinations, and risks were generated as separate results for each of these categories.

Because of difficulties in defining the area source universe, area sources were not specifically targeted for sampling, and no specific sample size was considered. The reason for this is that the statutory definition of major sources versus area sources under Section 112 of the CAA is based on total facility-wide emissions of hazardous air pollutants. Specifically, those industrial complexes emitting greater than 10 tons of any one hazardous air pollutant or greater than 25 tons of multiple hazardous air pollutants per year are considered major sources. To define an area source under this definition, information about the industrial complex in which an on-site incinerator is located is needed. Such information was not readily available, making it impossible to adequately characterize the area source universe and, therefore, to define the sampling frame. Because area sources are of interest, however, inferences were made regarding exposure and risk based on those incinerators that could be identified and had otherwise been

sampled[4]. For cement kilns, all area sources had been sampled and, therefore, all were used for making such inferences.

In determining the sample size and allocation, a large enough number of sites from each stratum (combustor category) were selected so that at least one of the sites posing the greatest risk was included in the sample. To define what is meant by "the greatest risk," some number of sites in each stratum were specified. For example, if the N_h sites in the *h*-th stratum were to be ordered from lowest to highest risk, then some number $N_h^* < N_h$ of sites at the top of the list could be identified as posing the greatest risk. Given N_h^*, the problem becomes one of determining the smallest stratum-level sample size, n_h, that will provide a specified probability of including at least one of these sites. The probabilities are given by

$$\text{Prob}\left\{N_h^* \geq 1 \in S\right\} = 1 - \frac{\binom{N_h - N_h^*}{n_h}}{\binom{N_h}{n_h}} \tag{4-1}$$

where $\text{Prob}\left\{N_h^* \geq 1 \in S\right\}$ means the probability associated with having at least one high-risk facility, N_h, in the sample. What remains is a numerical exercise to determine the smallest value n_h that will provide the specified probability.

The sample size solutions shown in Table 4-1 are obtained by defining $N_h^* = 0.10\, N_h$ and requiring $\text{Prob}\left\{N_h^* \geq 1 \in S\right\} \geq 0.90$. That is, a large enough stratum-level sample size was required to provide a 90 percent chance of including at least one facility from the top 10 percent of facilities with respect to risk.

4.1.5 Facility Sampling

The 11 modeled facilities from the proposed rule (10 of which were retained for the final rule) had been selected purposively, which complicated their inclusion in the risk characterization for the final rule. From a statistical standpoint, however, these 10 facilities were considered along with facility selection conducted for the final rule. Therefore, the 10 facilities evaluated for the proposed rule were defined as certainty samples (had a 100 percent chance of being selected), and the remaining HWC facilities (minus the 10) were used to construct the sampling frame for the stratified random sample.

The sample of facilities for the final rule were randomly selected within each stratum. During facility sampling, two unanticipated circumstances arose that complicated the sample design and sample selection:

[4]Area source incinerators that could be identified included commercial incinerators and on-site incinerators at U.S. Department of Defense installations.

- Information obtained from state/EPA Regional offices and reviewed after sample selection had started indicated the need to make changes in facility status (e.g., combustor category classification and operational status).

- After sample selection had been initiated, the decision was made to include waste heat boilers as an analysis domain.

One development that impacted sample selection (the change in facility classification and operational status) meant that the original sampling frame used for sample selection was not representative. Specifically, the sampling frame had some facility type misclassifications, contained some ineligible facilities, and was missing several eligible facilities that were identified during the review of information obtained from states/EPA Regions. After cleaning the sampling frame and recalculating the coverage probabilities, two more supplemental strata were created to increase the coverage for waste heat boilers and large on-site incinerators to the target goal of having a 90 percent probability of selecting a high-risk facility. The decision to include waste heat boilers as a separate analysis domain resulted in the construction of an additional supplemental stratum, since the number of waste heat boilers selected during initial sample selection (i.e., before waste heat boilers were identified as a separate stratum) did not provide an adequate coverage probability.

Table 4-2 presents the frame sizes and sample sizes by sampling strata. The frame and sample sizes exclude facilities that were later determined ineligible. Strata 1 through 6 are associated with the initial sample of 68 facilities from the total of 159 facilities within the original frame. As referred to earlier, the supplemental sample of two facilities was selected in stratum 7 to increase the sample of waste heat boilers. The frame for stratum 7 included all the facilities classified as waste heat boilers at that time that were not previously selected in strata 1 through 6.

Cleaning up the sampling frame involved

- Correcting previously misclassified combustor category classifications

- Removing ineligible facilities

- Adding six new facilities not listed on the original frame (bringing the universe total to 165).

After the sampling frame was corrected, additional waste heat boilers and large on-site incinerators were sampled to provide sufficient coverage for these combustor categories. Specifically, additional waste heat boilers were sampled from stratum 8, which contained all the waste heat boilers not selected in strata 1 through 7, and additional large on-site incinerators were sampled from stratum 9, which contained all the large on-site incinerators not selected in strata 1 through 8.

Table 4-2. Frame and Sample Sizes

Facility Stratum	Number Facilities in Frame	Facility Sample Size	Actual Waste Heat Boilers
1. Facilities Evaluated for Proposed Rule (certainty sample)	10	10	1
2. Cement Kilns	13	10	0
3. Lightweight Aggregate Kilns	3	3	0
4. Commercial Incinerators	16	11	4
5. Large On-site Incinerators	36	13	2
6. Small On-site Incinerators	81	21	6
Total	159	68	13
7. Additional Waste Heat Boilers, First Time	19	2	0 (classification error)
8. Additional Waste Heat Boilers, Second Time	16	3	3
9. Additional Large On-site Incinerators	30	3	0
Total	--	76	16

Because three of the six new facilities identified through review of the state/EPA Regional information were small on-site incinerators that were not waste heat boilers, they did not have a chance to be selected during original sample selection, resulting in undercoverage for the small on-site incinerator category. As described in the weighting section, facility poststratification adjustment was used to compensate for inefficiencies in the original sampling frame, including such factors as undercoverage due to not having included viable facilities in the original sampling frame.

The supplemental sampling strata complicated the selection probabilities. Although the task to account for these complications was not trivial, the large sampling rates for the replacement sampling ameliorate the variance-inflating effects of the inefficient sampling. (Note: Both the initial and supplemental sample have relatively high sampling rates.) That is, because the large sampling rates yield very small variances, the variance inflation effects from the inefficient sampling are negligible in comparison. For additional discussion on the effect of sample/population size and inefficient sampling on variance, see Appendix A.

Table 4-3 presents the final set of sampled facilities used in the risk assessment for the final rule.

Table 4-3. Sample Facilities, Classification, and Sampling Weights

Site Type	Site IDs	Company Name	Location	Area	WHB	Adjusted Facility Sampling Weight[a]
CINC	331	Ross Incineration Serv	Grafton, OH	x		1.959
CINC[b]	221	Rollins Environmental Services	Deer Park, TX	x		1.347
CINC	324	Allied Corp.	Birmingham, AL		x	1.521
CINC	325	Aptus	Coffeyville, KS	x		1.959
CINC	333, 612	Trade Waste Incineration	Sauget, IL			0.857
CINC[b]	214	Rollins Environmental Services	Baton Rouge, LA	x		1.347
CINC	601	Laidlaw Environmental Services INC	Clive, UT	x	x	2.479
CINC	486, 487	Ensco, Inc	El Dorado, AR			0.857
CINC	359	Atochem	Carrollton, KY	x	x	2.479
CINC	210, 211, 212	LWD, Inc.	Calvert City, KY			0.857
CINC	A15	BDT Inc.	Clarence, NY	x		1.959
CINC	209	Laidlaw Environmental Services	Roebuck, SC		x	1.521
CINC	A18	Chemical Waste Mgmt	Port Arthur, TX			0.857
CK[b]	401, 402	Ash Grove Cement Company	Chanute, KS			1.019
CK[b]	320	Lafarge	Alpena, MI			1.325
CK	321	Medusa Cement Company	Demopolis, AL	x		1.130
CK	403, 404, 228	Ash Grove Cement Company	Foreman, AR			1.325

(continued)

Table 4-3. (continued)

Site Type	Site IDs	Company Name	Location	Area	WHB	Adjusted Facility Sampling Weight[a]
CK[b]	304	Lone Star Industries	Greencastle, IN	x		0.870
CK[b]	207, 208	Keystone Cement Company	Bath, PA			1.019
CK	305, 335	Medusa Cement	Wampum, PA			1.325
CK	318, 473	Texas Industries	Midlothian, TX			1.325
CK	322, 323	Lafarge	Fredonia, KS			1.325
CK	302	Lafarge	Paulding, OH			1.019
CK	202	Heartland Cement	Independence, KS			1.325
CK[b]	205, 206	Holnam, Inc.	Holly Hill, SC			1.325
CK	204	Holnam, Inc.	Clarksville, MO			1.325
CK	203	Holnam, Inc.	Artesia, MS			1.019
CK	200, 201, 680, 681	Giant Cement Company	Harleyville, SC			1.325
LWAK[b]	311, 312, 336	Solite	Cascade, VA			1.000
LWAK	310, 475	Solite	Brooks, KY			1.000
LWAK[b]	307, 479	Thermalkem (Norlite)	Cohoes, NY			1.000
LWAK	225	Solite	Norwood, NC			1.000
LWAK	313, 314	Solite	Arvonia, VA			1.000
OINC-Large	A62	Texaco Chemical Co.	Conroe, TX			2.328

(continued)

Table 4-3. (continued)

Site Type	Site IDs	Company Name	Location	Area	WHB	Adjusted Facility Sampling Weight[a]
OINC-Large	504	Chevron Chemical	Philadelphia, PA			2.328
OINC-Large	464	BP Chemicals	Lima, OH			2.328
OINC-Large	A43	Occidental Chemical Corp	Niagara Falls, NY			3.243
OINC-Large	463	Miles	Kansas City, MO			1.978
OINC-Large	480, 706	Ciba-Geigy	St. Gabriel, LA			1.978
OINC-Large	915	Eastman Kodak	Rochester, NY			2.328
OINC-Large	809, 810	Tennessee Eastman	Kingsport, TN			2.328
OINC-Large	711	Chevron Chemical Co.	Belle Chasse, LA		x	3.314
OINC-Large	705, 490	Ciba-Geigy Corporation	McIntosh, AL			2.328
OINC-Large	353, 354	Dow Chemical Co.,	Midland, MI			2.328
OINC-Large[b]	334	3M	Cottage Grove, MN		x	1.197
OINC-Large	600	Dow Chemical	Freeport, TX		x	2.489
OINC-Large	B20	GSX Chemical Services	Cleveland, OH			2.083
OINC-Large	806	Amoco Oil, Co.	Whiting, IN			2.328
OINC-Large	483	Hoechst Celanese	Seabrook, TX			2.522
OINC-Large	A50	Quantum Chemical Company	La Porte, TX			3.243
OINC-Large	477, 478, 805	American Cyanamid	Hannibal, MO			2.328

(continued)

Table 4-3. (continued)

Site Type	Site IDs	Company Name	Location	Area	WHB	Adjusted Facility Sampling Weight[a]
OINC-Small	A31	Hercules, Inc	Franklin, VA		x	0.981
OINC-Small	A26	Eastman Chemical Co,	Magness, AR		x	2.026
OINC-Small	B32	Miles Corp.	Baytown, TX			3.965
OINC-Small	A14	Basf Corporation	Geismar, LA			3.049
OINC-Small	A46	OSI Specialties Inc	Sisterville, WV			3.965
OINC-Small	824	Penwalt Corp.	Thorofare, NJ			3.965
OINC-Small	A47	Phillips Research Center	Bartlesville, OK			3.965
OINC-Small	B37	Pine Bluff Arsenal	Pine Bluff, AR	x		7.236
OINC-Small	340	Miles Inc.	New Martinsville, WV		x	1.319
OINC-Small	704	Ashland Chemical Company	Los Angeles, CA		x	1.138
OINC-Small	701	Eli Lilly and Company	Clinton, IN			3.965
OINC-Small	708	Burroughs Welcome	Greenville, NC			3.965
OINC-Small	A55	Schenectady International, Inc.	Rotterdam Jct., NY		x	2.026
OINC-Small	B44	Shell Chemical Co.	Deer Park, TX			2.847
OINC-Small	453	Cargill Chemical Products	Forest Park, GA		x	2.026
OINC-Small	906	Monsanto Agricultural Company	Muscatine, IA			3.965
OINC-Small	904	First Chemical Co.	Pascagoula, MS		x	1.319

(continued)

Table 4-3. (continued)

Site Type	Site IDs	Company Name	Location	Area	WHB	Adjusted Facility Sampling Weight[a]
OINC-Small	468	Lonza Chemical	Conshohocken, PA			3.965
OINC-Small	A45	Occidental Chemical Vcm	Deer Park, TX			2.847
OINC-Small	B23	Huntsman Corp.	Port Neches, TX			3.049
OINC-Small	B18	Georgia Gulf Corp	Plaquemine, LA		x	2.026
OINC-Small	B31	Merck and Co.	West Point, PA			3.049
OINC-Small	342	Upjohn Company	Kalamazoo, MI		x	1.138
OINC-Small	725	Zeneca	Bayonne, NJ			3.965
OINC-Small	493, 494	U.S. Army Tooele Depot North	Tooele, UT	x		7.236

CINC = Commercial incinerator.
CK = Cement kiln.
LWAK = Lightweight aggregate kiln.
OINC = On-site incinerator.
WHB = Waste heat boiler.

[a]These facility weights do not include the sector-level population component.

[b]Facilities modeled for proposed rule.

4.1.6 Analysis Weights

This section discusses how the analysis weights and their components were calculated. The analysis weights were used to make inferences about individual and population risk estimates from the modeled facilities to all HWC facilities. Analysis weights were derived separately for each of the modeled facilities. These weights were then applied to each of the sector-specific risk estimates to create weighted estimates, which could then be used to create cumulative risk distributions for a given combustor category. The overall analysis weight was calculated as the product of two weight components: (1) facility sampling weight, including facility poststratification adjustments, and (2) sector-specific population weight. Each of these weight components is described below.

4.1.6.1 <u>Facility Sampling Weight</u>. The facility sampling weight (WT1) for each sampled facility was the reciprocal of the probability of selection. In most cases, the probability of selection was simply the stratum sample size divided by the stratum frame size. However, the inclusion of a supplemental strata (i.e., the waste heat boilers) complicated the probability structure and resulted in some facilities having multiple chances of selection. Hence, the facility probability of selection was not uniform within a given combustor category and is defined as:

$$\pi_h(i) = \begin{cases} 1 & \text{for certainty facilities, else} \\ \dfrac{n_h}{N_h} & \text{for facilities with one selection chance, else} \\ P_1 + (1-P_1)\,P_2 & \text{for facilities with two selection chances, else} \\ P_1 + (1-P_1)\,P_2 + (1-P_1)\,(1-P_2)\,P_3 & \text{for facilities with three selection chances,} \end{cases} \qquad (4\text{-}2)$$

where

h	=	sampling stratum
P_1	=	probability selected in first possible stratum
P_2	=	probability selected in second possible stratum
P_3	=	probability selected in third possible stratum.

Therefore, the facility sampling weight was assigned as follows:

$$WT1 = 1 / \pi_h(i) \ . \qquad (4\text{-}3)$$

Table 4-4 lists the possible selection strata for the facilities with multiple chances of selection and indicates how classification changes affected the possible selection strata.

Table 4-4. Facilities with Multiple Chances of Selection

Facility IDs	Actual Selection Stratum	Possible Selection Strata	Classification Change
209, 324, 359, 601	4	4, 7, 8	None
a31	5	5, 7, 8	OINC-L ⇒ OINC-S
342, 704	6	6, 7, 8	none
b20	6	6, 7, 9	OINC-S, WHB ⇒ OINC-L, non-WHB
b31	6	6, 7	WHB ⇒ non-WHB
a14, b23	7	6, 7	WHB ⇒ non-WHB
600	8	5, 8	None
340, 904	8	6, 8	None
463, stg	9	5, 9	None
a32	9	6, 9	OINC-S ⇒ OINC-L

OINC-L = On-site incinerators - large.
OINC-S = On-site incinerators - small.
WHB = Waste heat boilers.

Facility Poststratification Adjustment. The cumulative design modifications (described in Section 4.1.5) have the effect of reducing the efficiency of the sample. To improve the quality of the sample estimates, the facility sampling weights (WT1) were adjusted to force sample estimates of the total number of facilities in the categories listed in Table 4-5 to equal the known totals for these categories. The categories were established by cross-classifying combustor type with waste heat boiler status and combustor type again with area source status.

The individual facility adjustment factors are the quantities λ_i in the equation

$$\sum_{i \in S} w_i \lambda_i \underline{x}_i' = \underline{T}', \tag{4-4}$$

where the range of summation is taken over all facilities in the sample and

w_i = facility sampling weight (i.e., WT1 defined above)

$\underline{x}_i'$ = transpose of a vector of indicator (0,1) variables identifying the categories of facilities listed in Table 4-5

$\underline{T}'$ = transpose of the vector of known category totals.

Table 4-5. Average Weight Adjustment Factors from Exponential Model for Poststratifying to Facility Population Totals

Exponential Model Variable	Population Control Total	Average Facility Sampling Weight Adjustment Factor
Combustor Type / Waste Heat Boiler Status		
Cement Kiln	18	1.00
Lightweight Aggregate Kiln	5	1.00
Commercial Incinerator, Waste Heat Boiler	8	1.55
Commercial Incinerator, not Waste Heat Boiler	12	1.01
Large On-site Incinerator, Waste Heat Boiler	7	1.20
Large On-site Incinerator, not Waste Heat Boiler	36	0.84
Small On-site Incinerator, Waste Heat Boiler	14	0.53
Small On-site Incinerator, not Waste Heat Boiler	65	1.13
Combustor Type / Area Source Status		
Cement Kiln, Area Source	2	0.87
Cement Kiln, not Area Source	16	1.02
Lightweight Aggregate Kiln	5	1.00
Incinerator, Area Source	28	1.59
Incinerator, Not Area Source	114	0.85

The adjustment factors were computed as the solutions to the exponential regression relation

$$\lambda_i = \exp\left(\alpha + \underline{x}_i \underline{\beta}\right), \tag{4-5}$$

where

α = value of the relation at $\underline{x}_i = \underline{0}$ (i.e., the intercept)

$\underline{\beta}$ = vector of regression coefficients relating the weighted sample observations to the facility categories.

The α- and $\underline{\beta}$-values were determined numerically to satisfy Equation 4-5. The solutions were constrained so that $0.5 \le \lambda_i \le 2.0$. The imposition of these constraints ensured that sampling

variances were not excessively inflated because of unequal weighting effects associated with making the (poststratification) adjustments.

The adjusted facility sampling weights are the product of the initial facility sampling weights (WT1) and the adjustment factors (λ_i). The adjusted facility sampling weights are presented in Table 4-3. The average weight adjustment factors and the known population counts (control totals) are shown in Table 4-5 for each of the defined combustor categories.

4.1.6.2 <u>Sector-Population Weight (WT2)</u>. Since all 16 sectors for every sampled facility were selected (i.e., included in the risk characterization), the sector sampling weight is 1.0. However, because the analysis is at the sector level and estimates on the human population are being made, the sector weight needs to be multiplied by the human population in each sector. Consequently, the sector population weight is

$$WT2 = 1 \bullet pop_{ij} \tag{4-6}$$

where

i = facility
j = sector.

For recreational fishers, subsistence farmers, and subsistence fishers, the human population was set to 1 because information was not obtained to approximate sector-level populations for those groups (i.e., these receptor populations were not weighted).

4.1.7 Variance Estimation (Confidence Intervals)

Most statistical software packages assume simple random sampling from an infinite population and are not appropriate for variance estimation of sample survey estimates. That is, they do not compensate for survey design features such as stratification, clustering, and sampling from a finite population. Hence, they would produce biased variance estimates for sample survey data. To account for these survey design features, all of which are components of the HWC risk analysis, the majority of risk estimates (and associated confidence intervals) for the HWC risk analysis were computed using RTI's statistical software package, SUDAAN®. SUDAAN is a multiprocedure package that takes into account survey design features (i.e., sample design parameters can be specified and correct standard errors can be computed).

In addition, for probability-based sample surveys, most estimates are nonlinear statistics. Hence, the variances of the estimates cannot be expressed in closed form. For example, a mean or proportion, which is expressed as $\Sigma wy / \Sigma w$, is nonlinear because the denominator is a survey estimate of the (unknown) population total. SUDAAN offers both the Taylor series linearization and replication methods (BRR and Jackknife) for robust variance estimation of nonlinear statistics. For this analysis, the Taylor series linearization method was used. This method computes the Taylor series approximation of the nonlinear statistic and then substitutes the linear representation into the appropriate sample design variance formula.

There were four basic types of estimates computed by SUDAAN for the HWC risk analysis:

- Cumulative distributions for risks (or hazard quotients) not weighted by population
- Population-weighted individual risk (or hazard quotient) percentiles
- Population estimates of cancer incidence (both local and national)
- Proportion of population with risk (or hazard quotient) greater than the health benchmark level.

The uncertainty of all the estimates was measured by 90 percent confidence intervals. The confidence intervals for the percentiles were computed internally by SUDAAN. To obtain confidence intervals of a given percentile, SUDAAN first computes the confidence intervals for the cumulative distribution based on the sampling error of the cumulative distribution. Then, the confidence bounds for a given percentile are determined from the confidence bound formulas of the cumulative distribution. This method was used to compute confidence intervals for cumulative distributions for risks (or hazard quotients) not weighted by population and population-weighted individual risk (or hazard quotient) percentiles.

The 90 percent confidence intervals for the population estimates of cancer incidence (both local and national) were computed from a log transformation. Because the cancer incidence estimates are small and the sample sizes are small for some domains, the underlying distribution was assumed to be asymmetric and the log transformation was used to compute asymmetric confidence intervals. These asymmetric intervals are more balanced with respect to the probability that the interval covers the true population value than do standard symmetric confidence intervals. For this analysis, only 90 percent confidence intervals were calculated. To illustrate the method, let

T = estimated population total ($\Sigma w_i x_i$)
L = natural log of T
SE(L) = standard error of L.

The 90 percent confidence intervals for L were then calculated as

$$A = L - t_{.05}SE\{L\}$$
$$B = L + t_{.05}SE\{L\}. \qquad (4\text{-}7)$$

The Student's t-distribution with 70 degrees of freedom was used instead of assuming a normal distribution. However, with 70 degrees of freedom, the normal and Student's distributions are essentially equal. The degrees of freedom are equal to the number of selected facilities (76) minus the number of analysis strata (6), which are the first six strata listed in Table 4-2.

By taking the exponential values of A and B, the confidence intervals for the population total, T, are

$$\begin{aligned} T_{lower} &= \exp(A) \\ T_{upper} &= \exp(B) \ . \end{aligned} \tag{4-8}$$

For the proportion of population with risk (or hazard quotient) greater than the health benchmark level, the logit transformation, ln[p/(1-p)], was used to compute the confidence intervals. The confidence intervals using the logit transformation were computed in a manner similar to that used for the log transformation for the population totals. The logit transformation prevents estimates of prevalence rates from being either less than zero or greater than unity. The transformation itself is given by

$$X_d = \text{Logit}\left\{\hat{P}_d\right\} = \ln\left\{\frac{\hat{P}_d}{1 - \hat{P}_d}\right\} \tag{4-9}$$

where $\hat{P}_d$ is the estimated prevalence rate for the *d*-th reporting domain (e.g., type of chemical by receptor population). The interval estimate can be written as

$$\text{Prob}\left\{P_{d,\ell} \le P_d \le P_{d,u}\right\} = 1 - \alpha . \tag{4-10}$$

On the transformed scale, the interval estimate becomes

$$\hat{X}_d \pm t_{\alpha/2}\,\text{SE}\left\{\hat{X}_d\right\} = \hat{X}_d \pm t_{\alpha/2}\left(\frac{\sqrt{\text{Var}\left\{\hat{P}_d\right\}}}{\hat{P}_d\left(1 - \hat{P}_d\right)}\right), \tag{4-11}$$

and the inverse transformations

$$\begin{aligned} P_{d,\ell} &= \frac{1}{1 + \exp\left\{\hat{X}_d - t_\alpha\,\text{SE}\left\{\hat{X}_d\right\}\right\}}, \\ P_{d,u} &= \frac{1}{1 + \exp\left\{\hat{X}_d + t_\alpha\,\text{SE}\left\{\hat{X}_d\right\}\right\}} \end{aligned} \tag{4-12}$$

provide the upper and lower bounds of the intervals on the arithmetic scale. The intervals in this case are not necessarily symmetric.

Confidence intervals generated for the HWC risk analysis are symmetric around each of the quantiles being considered and are calculated as follows. Denote the distribution function of interest by

$$F(x) = \frac{1}{N}\sum_{g=1}^{N} I(y_g \le x) \tag{4-13}$$

where the subscript $g = 1, 2, \ldots, N$ identifies units in the population, in this case sectors, and

y_g = value returned by the risk model for the g-th sector,

$I(y_g \le x) = 1$, if $y_g \le x$,
$= 0$, otherwise.

The quantiles of the distribution are defined by the values k such that

$$F(x_k) \le Q_p \le F(x_{k+1}). \tag{4-14}$$

SUDAAN estimates the distribution function by

$$\hat{F}(x) = \frac{\sum_{g \in S} w_g I(y_g \le x)}{\sum_{g \in S} w_g}, \tag{4-15}$$

where w_g are the sampling weights, and finds the values $k = 1, 2, \ldots, p$ such that $\hat{F}(x_k) \le Q_p \le \hat{F}(x_{k+1})$. The confidence interval is computed using the standard error

$$SE\{Q_p\} = \frac{\hat{U}_p - \hat{L}_p}{2t_{\alpha/2}} \tag{4-16}$$

where $\hat{U}_p$ and $\hat{L}_p$ are the limits implied by $\hat{F}(x_k) \pm t_{\alpha/2} SE\{\hat{F}(x_k)\}$, and $t_{\alpha/2}$ = value of Student's t-distribution at the significance level $\alpha/2$.

Hence, the variance of interest, that is the quantity $(SE\{Q_p\})^2$, involves the point on the estimated distribution function $\hat{F}(x_k)$ and the standard error associated with the estimate at that point. The intervals are seen to be symmetric about the quantile. For some of the risk (and HQ) percentiles of cumulative distributions, confidence intervals could not be generated because of an insufficient sample size or insufficient spread of modeled risk values.

4.2 Facility Operating Characteristics and Emissions Estimates

This section describes the facility-specific engineering and annual emissions data used to conduct air modeling for purposes of generating sector-level air concentration and deposition estimates for modeled HWC facilities[5]. Assumptions concerning operational, facility-specific engineering and annual emissions estimates are presented.

Section 4.2.1 describes the database used to characterize modeled HWC facilities and Section 4.2.2 describes operating scenarios, engineering data, and annual emission estimates.

4.2.1 Facility Database

In conducting the HWC risk analysis, information on the universe of facilities as well as HWC facility-specific engineering data were required. A brief overview of the data sources and methodologies used to develop these data is presented here. A more comprehensive discussion is provided in U.S. EPA (1999b).

The database used in this analysis contains the following facility-specific data:

- Facility equipment and operational data (e.g., engineering data including stack heights, combustors, air pollutant control device [APCD], temperatures, exit velocities)

- Emission rates for constituents discharged to the atmosphere (e.g., metals, chlorine, PM, PCDD/PCDF, CO, and HCl) from the facility's main stack.

The HWC facilities included in the database are all facilities known to be operational in 1997.

These data were revised since proposal in an effort to incorporate additional facility-specific information as it became available and to address data issues raised in public comments. Specifically, the database was augmented with facility-specific information obtained during an initial comment period (at proposal), a subsequent Notice of Data Availability (NODA) comment period, and further data-gathering efforts involving visits to Regional EPA and state environmental offices, which were conducted in the fall of 1997.

EPA published a notice in the *Federal Register* covering the database that was used to set the floor levels via a NODA on January 7, 1997. The database contained all the information available from trial burn and certificate of compliance reports that was used in the analysis, including emissions data and engineering information on APCD and operating parameters as well as stack information. This information was used to characterize stack emissions where measurements were available and for imputing exhaust gas concentrations where they were not.

[5] The term "engineering data," as used here, refers to data used to characterize the physical release of emissions (e.g., stack height, stack diameter, and exit velocity), including all parameters necessary for conducting air modeling.

Those facilities that were in the universe of facilities covered by the rule but for which EPA did not have test reports were not in the database.

This additional information, obtained chiefly from newly identified trial burn and compliance test reports, resulted in adjustments to facility-specific engineering parameters and emissions estimates. In some cases, the new information resulted in a facility being removed from the database (e.g., closure) or changing its classification from one source category to another (e.g., OINC-L with WHBs to OINC-L without WHBs).

4.2.2 Facility-Specific Engineering and Emissions Data

The HWC facilities modeled for this risk analysis may contain one or more combustion units with associated stacks. Although risks were assessed at the facility level, air modeling was conducted separately for each stack. Emissions that result from materials handling, fugitive releases, emergency safety valve releases, disruptions in the normal combustion operation, startup, and shutdown (none of which are subject to the MACT standards) were not modeled. Therefore, emission rates may not be representative of all operating scenarios experienced at an actual facility.

These combustion units were assumed to be operating continuously for 24 h/d, 365 d/yr. Annual stack emissions were calculated assuming continuous operation for an entire year. Therefore, annual emissions rates may not be representative of actual facility operation with regard to temporal fluctuation in emissions and actual times of emission release. Short-term emissions derived from annual emission rates and used in air dispersion and deposition modeling were based on 8,760 hours of operation per year (see Section 5.1 for more information on air modeling inputs).

Emissions scenarios were developed for a base case and for three regulatory alternatives:

- **Baseline** —The baseline scenario assumes emissions rates associated with normal operation as they currently exist without application of additional air pollution controls.

- **MACT-Standard**—The MACT standard scenario assumes emissions rates based on a set of air pollution controls that would be required to satisfy the final rule. These controls are a mixture of MACT floor and MACT beyond-the-floor requirements.

- **MACT-Floor**—The MACT floor scenario assumes emission rates based on a set of air pollution controls required to satisfy the minimum control requirements for HWC facilities under Section 112(d)(3) of the CAA.

- **MACT-Beyond-the-Floor**—The MACT beyond-the-floor scenario assumes emission rates based on a set of controls necessary to achieve a greater degree of emissions reduction than is required for the MACT floor scenario. These more effective controls are applied to dioxins, mercury, lead, hydrogen chloride, and chlorine gases for certain combustor categories.

No increase in emissions was assumed for those facilities that were operating below the design level needed to satisfy the MACT standard. That is, emission rates used in this risk assessment do not reflect that a facility would make changes to their operations and increase to an emissions level higher than they were emitting before the standards. For a more detailed discussion of the regulatory scenarios evaluated for the HWC MACT rulemaking, see U.S. EPA (1999b).

Engineering data were required to estimate emissions and as input to air modeling. The following categories of facility-specific engineering data were used for air modeling: stack location (latitude and longitude), stack height (m), stack inside diameter (m), exit velocity (m/s), stack gas temperature (K), and building height and width (m). Facility-specific engineering data used for air dispersion modeling are presented in Appendix B.

The final list of constituents selected for evaluation in the HWC risk analysis consisted of

- 17 dioxin/furan congeners
- 14 metals
- Chlorine and hydrogen chloride
- $PM_{2.5}$ and PM_{10}.

Emissions estimates were made for all chemical constituents covered by the rule for which sufficient data were available. These included chlorine-substituted dibenzo(*p*) dioxins and dibenzofurans, elemental mercury (Hg^0), divalent mercury (Hg^{+2}), lead, cadmium, arsenic, beryllium, trivalent chromium (Cr^{+3}), hexavalent chromium (Cr^{+6}), chlorine, and hydrogen chloride. In addition, emissions estimates were made for particulate matter (PM_{10} and $PM_{2.5}$) and nine other metals, three of which (cobalt, copper, and manganese) were not assessed at proposal but were included in the risk assessment for the final rule. Chemical-specific emissions estimates were not made for organic constituents other than dioxins and furans (e.g., various products of incomplete combustion) due to insufficient emissions measurement data. Risks from all constituents for which chemical-specific emissions estimates could be made as well as from PM were evaluated in this risk assessment.

The original facility-specific emissions concentration and flow rate data were obtained primarily from trial burn and certificate of compliance test reports. When more than one source of emissions data was available, data were obtained from the report based on the most recent sampling. An imputation scheme was used to fill in missing emissions data for HWC facilities. In conducting imputation, efforts were made to match the missing data to the group of facilities from which values were being imputed based on similarities in equipment and operations (i.e., data would be imputed for a given facility from a set of facilities with characteristics similar to those of that facility). Facilities were matched for purposes of imputation to improve the representativeness of the imputed data. An in-depth discussion of the imputation procedure as well as the overall approach used in developing the database is provided in U.S. EPA (1999b).

4.3 Site Characterization for Modeled Facilities

This section describes the methodologies and data sources used in site characterization specifically with regard to

- Selecting and characterizing waterbody/watersheds for inclusion in risk modeling
- Establishing site-specific human and livestock populations.

Site characteristics associated with air dispersion modeling (e.g., terrain, meteorology) are discussed in Section 5.1.

4.3.1 Study Area

The HWC risk analysis conducted for the final rule generates spatially refined human health and ecological risk results based on a 16-sector study area template. To achieve the desired degree of spatial resolution for this risk assessment, a 20-km radius polar grid was used (see Figure 4-2). This polar grid, which is centered on the geographic coordinate for the HWC facility, was divided into 16 sectors and numbered as indicated in Figure 4-2. An individual polar grid, together with its HWC facility, is termed a "study area." The term "sector" refers to the 16-sector grid that defines the study area. The sector polygons were created by dividing four concentric circles around the site location (2, 5, 10, and 20 km radius) by the north-south and east-west axes.

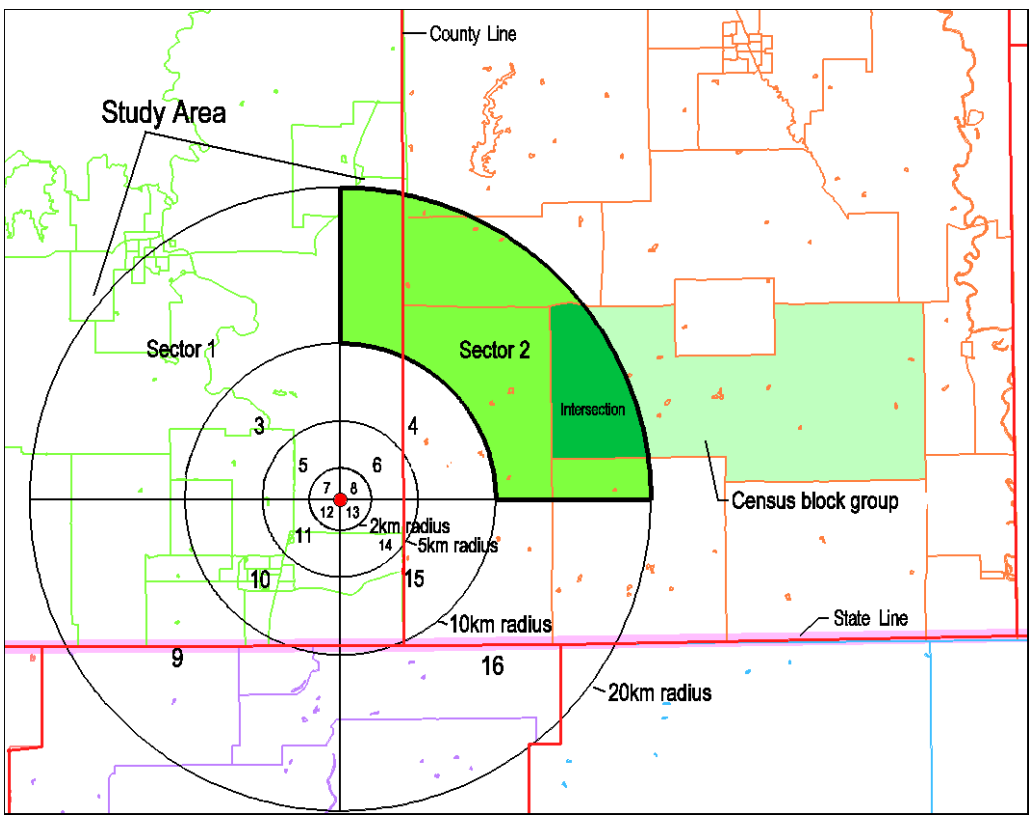

Figure 4-2. Example of study area, sectors, and area-weighted population apportionment.

Population counts for each receptor population at the sector level were combined with individual sector-level risk estimates to determine human health risks within study areas. Similarly, livestock population counts at the sector level were combined with sector-level dioxin concentration estimates to estimate national population risks resulting from exposure to dioxin contained in livestock raised within study areas.

Because of the volume of data required for the analysis, semiautomated techniques were used to access existing nationwide databases to provide the site-specific data used in site characterization. A geographic information system provided the platform for projecting the impact of HWC emissions on individual study areas and watersheds/waterbodies and for characterizing land use for estimation of human and ecological risk (e.g., location, shape, and size of watersheds and waterbodies and densities of human and livestock populations). The human health component of the HWC risk assessment includes risk estimates for receptor populations located within the modeled study areas (e.g., beef cattle farmers and recreational fishers). The HWC risk assessment also includes risk estimates for those human populations located outside of modeled study areas that may be impacted by ingestion of dioxin contained in food commodities produced within these study areas (e.g., individuals eating beef from beef cattle that were raised within a given study area and thereby exposed to dioxin from the associated HWC facility).

A GIS was selected as the platform for conducting the site characterization component of the HWC risk analysis because it can be easily automated and can perform spatial overlay of georegistered data. Most of the GIS processing was conducted using ARC/INFO for UNIX workstations; some took place in the PC environment with ARC/VIEW. The term “program” is used throughout this section to refer to Arc Macro Language (AML) scripts, a batch-process scripting language used with the ARC/INFO GIS software. The term “coverage” refers to a GIS map layer (e.g., geographically referenced digital points, lines, or polygons with attached data).

The GIS modeling results provided three sets of data inputs for the risk analysis:

- Waterbody characteristics and average air concentration and deposition values by watershed and waterbody within the study area
- Average air concentration and deposition values by sector within the study area
- Spatially averaged human and livestock populations by sector.

The remainder of this section discusses the various methodologies used to derive these data inputs for the risk analysis.

4.3.2 Waterbody/Watershed Selection, Delineation, and Characterization

With the exception of one site, from one to four waterbodies were selected for inclusion in the HWC risk analysis from each study area. For one site and region, there were no waterbodies modeled. Selected waterbodies were delineated and characterized. These waterbodies, termed “modeled waterbodies,” were used to provide site-specific data used in the

risk analysis. In characterizing modeled waterbodies, the following attributes were compiled for each waterbody and associated watershed:

- Watershed area
- Length of stream reach
- Waterbody area
- Universal soil loss equation (USLE) parameter
- Flow velocity and discharge
- Stream width/depth
- Total suspended solids concentrations.

Each of these attributes was defined for that portion of the watershed/waterbody located within the 20-km radius study area under consideration.

A combination of desktop evaluations using available maps/databases and GIS techniques was used to obtain site-specific values for each of these attributes. This section describes the approach used to select and delineate modeled waterbodies. In addition, the data sources and methodologies used in site-specific characterization of those modeled waterbodies are described.

4.3.2.1 Compilation of Study Area Data. Existing data layers were compiled to create a single comprehensive map for each study area. These maps, which were generated with GIS tools, are called "compilation maps." They were used to select waterbodies for inclusion in the study and delineate their associated watersheds. These 17 x 17 inch color maps were generated using an automated batch script that started with the point coverage of the site's location and then added the following map layers:

- **Sector boundaries:** Generated previously with an automated batch script

- **RF3 data:** EPA stream reach files (U.S. EPA, 1994) generated from 1:100,000 scale U.S. Geological Survey (USGS) digital line graphs (DLGs)

- **Drinking Water Supply Sites:** Supplied from the BASINS CD-ROM database (Laveck and Coombs, 1996)

- **Stream Gaging Stations:** Obtained from the BASINS CD-ROM and WATSTORE databases (USGS, 1994)

- **Pseudo drainage basin lines:** Generated from 1:250,000 Digital Elevation Model (DEM) coverages obtained from USGS. (A DEM consists of an array of elevation values for ground positions that are usually at regularly spaced intervals.)

4.3.2.2 Waterbody Selection. The following criteria were used to select modeled waterbodies/watersheds for inclusion in the HWC risk analysis:

- **Probable impact from facility emissions:** Those waterbodies located in the direction of prevalent winds and relatively close to the HWC facility were favored in selecting waterbodies for modeling to ensure that risks generated for study areas included more heavily impacted waterbodies.

- **Probable recreational use (including fishing):** Although it is difficult to determine patterns of recreational use at waterbodies from the maps used in selecting modeled waterbodies, characteristics suggestive of recreational use (e.g., larger waterbody size, location in favorable land-use areas, and good public access as determined from road and parking lot patterns) were considered in selecting waterbodies for inclusion in the HWC risk analysis.

- **Drinking water source:** Priority was given to waterbodies identified as drinking water sources. If several drinking water sources were identified for a given study area, priority was given to the one likely to be impacted to a greater extent by HWC emissions due to its location.

In general, the waterbodies selected for modeling favor those located in areas more heavily impacted by HWC emissions and do not represent a random sample that can be considered representative of all waterbodies located across the study areas. There is, however, an important caveat to this general statement. In selecting waterbodies for a given study area, often a different waterbody was selected to match each of the three criteria listed above (e.g., waterbody A may be selected from a more impacted location within the study area, waterbody B may be selected because it looked like a probable recreational location, and waterbody C may be selected because it was the drinking water source closest to the facility). Because all three criteria were considered in selecting waterbodies for inclusion in the HWC risk analysis, the waterbodies that were selected do not always represent those waterbodies most impacted by HWC emissions. In certain instances, the goal of including a drinking water source or a waterbody that appeared to be a likely location of recreational activity resulted in the exclusion of a more heavily impacted waterbody.

4.3.2.3 <u>Watershed Delineation</u>. The compilation maps and USGS 7.5 minute 1:24,000 scale quadrangle maps were used to delineate the watersheds for the selected waterbodies. The following delineation protocol was applied to each selected waterbody:

- Watershed boundaries were delineated by starting at the farthest downstream point of the selected stream (or outlet of the selected lake) that was still within the 20-km radius. A line was then drawn perpendicular to the topographic contour lines upgradient from that point. This line was extended until it reached the point at which the elevation ceased to increase or until it intersected with the boundary of the study area. Then, starting again from the farthest downstream point, a line was drawn (in the opposite direction) perpendicular to the contour lines until the elevation ceased to increase. The endpoints of these two lines were connected through the peaks and ridges on the map or along the boundary of the study area, whichever covered less area.

- Only the watershed area that drains into the selected waterbody **before** the waterbody flows out of the 20-km study area was included. If a tributary to the selected waterbody merged with the waterbody downstream of the study area, the area that drained to this tributary was not included in the watershed area.

- No watershed area that lies outside of the radius was included. Because only the 20-km study area was of interest, the radius acts as a cutoff distance for watershed delineation. Cutting off the watershed at 20 km is consistent with the definition of the study area used for this risk assessment. The result is that contaminant loading to waterbodies is based only on that portion of total emissions that deposit on watershed areas located in the study area. As noted below, however, waterbody flow is based on total flow (including tributaries located outside the study area), which best characterizes the waterbody's properties. The resulting uncertainty generally underestimates that portion of waterbody constituent concentrations that results from watershed loadings.

- Only the main stem for the selected streams (i.e., no tributaries) and waterbody surface areas for lakes and reservoirs were included.

- Arcs (lines) and polygons were digitized manually with a standard digitizing tablet and ARC/INFO workstation. Lakes and watershed polygons were labeled with name and site identification. Stream/river lines were labeled with name, width, and site identification. Stream coverages were processed in a program that changed the line coverage into a polygon coverage based on each stream's width.

Watershed delineation and digitization allowed for collection of the necessary model input parameters. Watershed area, stream length, and waterbody area values were extracted from the data tables associated with the digitized coverages.

Quality control measures were taken on each major step of the delineation process. A quality control check was completed after manual delineation to ensure correct watershed delineation and on the completed GIS coverages to ensure correct translation of watershed area and other parameters.

4.3.2.4 <u>Watershed Universal Soil Loss Equation Parameters</u>. The Indirect Exposure Emissions Model used for this risk analysis uses the USLE to estimate soil erosion losses (X_e) from watersheds that drain into modeled waterbodies surrounding each hazardous waste combustor site (Section 5.3.2). USLE is an empirically derived equation originally developed by the Soil Conservation Service (SCS) of the U.S. Department of Agriculture (USDA) to estimate soil erosion losses from agricultural fields during soil conservation planning. In the IEM methodology, USLE is applied in the context of the Gross Erosion Sediment-Delivery Ratio method outlined in USDA (1978) and described in greater detail in the SCS *National Engineering Handbook* (USDA, 1971). Gross erosion is defined as the summation of erosion from all sources within a watershed, as estimated for sheet and rill erosion by USLE. The sediment delivery ratio adjusts gross erosion rates to account for eroded soil that does not reach the waterbody in question. USLE requires inputs to estimate soil erosion losses, including rainfall and runoff factor (R), soil erodibility factor (K), topographic factor (LS), cover and

management factor (C), and supporting practice factor (P). In this context, USDA (1978) suggests the use of watershed-averaged values for K, LS, C, and P to simplify computational and data collection requirements. With the exception of K, this approach was adopted for developing site-specific USLE gross erosion loss estimates for the combustor sites. Each of these inputs is discussed below.

Rainfall and Runoff Factor. This factor quantifies the rainfall impact effect and provides relative information on the amount and rate of runoff associated with rain. The rainfall and runoff factor is the number of rainfall erosion index units, plus a factor for snowmelt or applied water where such runoff is significant. The rainfall erosion index for a given rainfall event is equal to the total storm energy times the maximum 30-min intensity. Local values of the erosion index were taken directly from the isoerodant maps provided in USDA (1978) by locating each site based on its location in the United States and selecting the closest value, interpolated as necessary. The rainfall erosion index, however, does not account for runoff associated with surface thaws and snowmelt. Soil erosion by thaw runoff is most pronounced in the northwest United States, but it may be significant in other northern states. In this analysis, surface thaw and snowmelt were not considered, which for some facilities could underpredict the amount of runoff, but the overall influence on not including this information was not expected to be substantial because only a subset of facilities would be affected (none are located in the Pacific Northwest for instance).

Soil Erodibility Factor. This factor accounts for variability in different soils' tendencies to erode. A national value for K for silt loam, which is a predominant soil type both nationally and for the combustor sites, was obtained for consistency with the national parameterization of other soil properties required for the model. STATSGO national soils data, compiled by the STATSGO map unit in the USSOILS database, were used to estimate various central tendency statistics for the more than 1,400 STATSGO map units across the country with silt loam soils and nonzero K values. Results are shown in Table 4-6. All central tendency statistics (mean, median, mode, area-weighted mean) were 0.34, which was the K value used in the analysis.

Table 4-6. National Central-Tendency Statistics: USLE Erodibility Factor (K) for Silt Loam Soils

Statistic	USLE K
Median	0.3400
Area-weighted average	0.3436
Mode	0.3400
Mean	0.3420

Data source: STATSGO/USSOILS national soils database.

Use of a single national value of K does not create any additional uncertainties beyond those associated with assuming a national soil type for all of the other soil properties (e.g., bulk density, porosity). Because soil erodibility is correlated with soil type, there would be a disconnect in using a site-based K and national parameterization for all the other soil properties (i.e., assuming different soils in different model components). The length-slope, cover and management, supporting practice, and rainfall and runoff factors are not strongly correlated with soil type, enabling use of site-specific values for those parameters.

Topographic Factor. The topographic factor quantifies the effects of slope (S) and slope length (L) on soil erosion loss. Both average slope (S) and average slope length (L) are required to determine an average watershed length-slope factor (LS). The STATSGO and USSOILS databases, which are maintained by the USGS, provide spatial information on soil series by map units, spatially contiguous areas with similar soil properties. For each watershed, S was queried from the USSOILS version of the STATSGO database. However, the STATSGO/USSOILS database does not contain data characterizing the slope length component (L) of the LS parameter. Although options were identified for estimating site-specific length values based on watershed area and total stream length, adequate site-specific data on stream length could not be obtained (existing sources such as Reach File Version 3 [RF3] do not contain true first-order streams and, therefore, underestimate total stream length).

Because no consistent national data sources were available for this parameter (other than direct field measurements), national default L values, dependent on slope length, were obtained from personnel at the USDA Natural Resources Conservation Service experienced in erosion prediction, agronomy, and soil services. These L and corresponding S values were estimated for national use in pesticide and construction erosion studies (Weesies, 1998) as shown in Table 4-7.

The site-specific average watershed S values were used to determine the corresponding L values, per Table 4-7. The L values thus determined were used to calculate an average LS for each watershed using the following equation (USDA, 1978):

$$LS = \left(\frac{L}{72.6}\right)^{m} \bullet (65.41\sin^2\theta + 4.56\sin\theta + 0.065) \qquad (4\text{-}17)$$

where

L	=	slope length (feet)
m	=	exponent dependent on slope (0.5 if S is 5% or more, 0.4 at 3.5 to 4.5%, 0.3 at 1 to 3%, 0.2 at less than 1%).
θ	=	angle of slope

This application of a site-specific value, a lookup table, and the equation to derive LS results in the use of an LS value that reflects site characteristics yet is not purely site-specific.

Table 4-7. Default Slope Length (L) used in Combustor Risk Analysis

Slope (%)	Default length (ft)	Slope (%)	Default length (ft)
< 0.05	100	9	125
1	200	10	120
2	300	11	110
3	200	12	100
4	180	13	90
5	160	14	80
6	150	15	70
7	140	17	60
8	130	>17	70

Source: Weesies (1998).

Cover and Management and Supporting Practice Factors. The cover and management factor measures the effect of land cover (e.g., crops, forests) on soil erosion losses; the supporting practice factor accounts for erosion control measures that may be applied (such as contour plowing for cropland). The cover management and supporting practice factors were derived from site-specific land use data obtained from GIRAS (Geographic Information Retrieval and Analysis System) data, which are coded using the Anderson land use II classification scheme
(Anderson et al., 1976)[6]. Using the crosswalk shown in Table 4-8, a database was created to convert Anderson codes to broader land use categories for which typical C and P values are available (Wanielista and Yousef, 1993). These values were then spatially averaged in the database to give an average C and P for each watershed.

4.3.2.5 <u>Waterbody Characterization</u>. Parameters that were required for the model but were not supplied by watershed delineation were stream/river velocity, discharge, width, and depth. BASINS Reach Files Version 1 (RF1) was queried by region for the selected waterbodies. Most of the selected waterbodies were listed, with all of the necessary parameter values. Many of the waterbodies had multiple data sets associated with different locations along the waterbody. If there was more than one valid datapoint, the most inclusive data were selected; thus, dilution effects from all tributaries were included.

[6] It should be noted that HWC air modeling runs were also completed with roughness height obtained from GIRAS coverage interpretation (see Section 5.1).

Table 4-8. Cover Factor (C) and Supporting Practice Factor (P) by Land Use Code

Wanielista and Yousef (1993)			
Land Use Type	**C**	**P**	**Anderson Land Use Codes (Anderson et al., 1976)**
Forestland	0.005	1	4 Forest (41-43)
Cropland	0.08	0.5	21 Cropland & Pasture; 25 Other agricultural land
Pastureland	0.01	1	3 Rangeland (31-33); 81-82, 83-84 Tundra
Urban	0.01	1	1 Urban or built-up land (11-17)
Water	0	1	5 Water (51-54); 6 Wetland (61, 62)
No erosion	0	1	74 Bare rock; 91 Snowfields; 92 Glaciers
No cover	1	1	23 Confined feeding operations; 7 Barren land (71, 73, 76); 83 Bare ground

If the waterbody did not appear in the RF1 tables, the BASINS Stream Gaging File was queried. This file provides values only for waterbodies' discharges. The discharge value was then entered into three equations derived from Keup (1985): velocity (ft/s) = $1.0662x^{0.127}$, width (ft) = $5.1867x^{0.4559}$, and depth (ft) = $0.1808x^{0.4171}$ where x (ft^3/s) is the discharge value.

If a discharge value for a particular waterbody was not available in BASINS Stream Gaging File, the USGS WATSTORE database was queried. If a value was available, it was used to estimate the velocity, width, and depth of the waterbody using the three equations from Keup (1985).

If no discharge data were available, the parameter estimations were derived from stream order using RF3 maps or preferably 1:24,000 USGS quad maps. Strahler's stream order classification system was used to order the selected streams. The stream order (1-10) was used in Keup's table (Keup, 1985) to estimate values for discharge, velocity, width, and depth.

Water column concentrations are intrinsically dependent upon the concentration of total suspended solids (TSS). TSS concentration was included in the surface water model as a parameter because there was insufficient information about other parameters (e.g., benthic burial rate for sediments) for both lakes and flowing waterbodies to allow the model to calculate TSS concentrations. Modeling TSS without sufficient information would produce an unacceptable level of uncertainty in the TSS values to which the modeled constituent concentration values are sensitive. Therefore, it was decided to set the parameter value for TSS and treat the benthic burial rate as a variable (Section 5.3.3.1).

Values for TSS were developed from STORET TSS data using a regional approach (http://www.epa.gov/OWOW/STORET/). A regional approach was selected because site-specific TSS estimates could not be calculated reliably with the existing data due to limitations in the STORET data. TSS data collection involved assigning the combustor sites to the regions and establishing typical (or central tendency) TSS values for each region.

The USGS Hydrologic Regions (Seaber et al., 1987) were selected as appropriate regions for the analysis because they are large enough to have an adequate number of STORET TSS values, adequately diverse to show some variability in TSS, and generally accepted as a way of organizing hydrologic and water quality data. Of the 18 continental U.S. Hydrologic Regions, 12 (2-8, 10-12, 16, 18) have modeled HWC facilities.

Ambient water quality monitoring data for TSS were extracted from EPA's mainframe computer located in Research Triangle Park, NC, by hydrologic region for all years of record (1960 to 1997). Data were extracted separately for flowing waterbodies (streams and rivers) and still waterbodies (lakes and reservoirs) because flowing water was anticipated to have significantly different TSS levels.

The STORET data (U.S. EPA, n.d.) (in flat file format) were read into SAS™ for statistical analysis. The SAS PROCUNIVARIATE procedure was used to calculate median TSS values for each region of interest over the entire period of record. The median was selected as the best central tendency statistic because (1) it does not require distributional assumptions and (2) it is relatively stable and not as sensitive as the mean to extreme values that may result from natural variability or errors in the STORET data.

Similarly, a large number of TSS values was needed so that extreme values or data of questionable representativeness would not bias the calculated median. For rivers and streams, thousands and often tens of thousands of TSS values were available in each hydrologic region and robust regional medians could be calculated. For lakes and reservoirs, fewer TSS measurements were available and it was necessary to combine like regions where possible to compile adequate data for calculation of a median.

Regional river and stream TSS medians and professional judgment (considering climatic and topographic characteristics) were used to identify and group regions. Six combined regions were used to develop TSS means for lakes and reservoirs. Combined regions included the East (Mid-Atlantic, South Atlantic-Gulf, Great Lakes, Ohio, and Tennessee), the Mississippi (Upper and Lower Mississippi), and the Midwest (Missouri, Arkansas-White-Red, and Texas-Gulf). Although they had considerably fewer TSS measurements than the combined regions, the Great Basin and California regions were analyzed separately because their waterbodies and climate are too different in character to combine with other regions.

Table 4-9 shows for each region and waterbody type the calculated median values and the years of record and number of measurements used to calculate the median. Note that lakes show significantly lower TSS values than rivers and that patterns in inter-regional variability are similar for the river and lake datasets, supporting the rationale used to aggregate and analyze the STORET TSS data.

4.4 Generating Spatially Averaged Sector-Level Human and Livestock Populations

Human population estimates were used to generate estimates for both cancer and noncancer effects for local populations (i.e., those human populations living within study areas). Livestock population estimates were used to project statistical cancer incidence within the

Table 4-9. Default TSS Values Used in Combustor Risk Analysis

	Hydrologic Region		Years of Record	No. of Measurements	Median TSS (mg/L)
STORET Median TSS - Rivers and Streams					
	2	Mid-Atlantic	60 - 97	47,076	27
	3	South Atlantic-Gulf	60 - 64, 67 - 97	43,013	24
	4	Great Lakes	60 - 96	29,538	21
	5	Ohio	60 - 97	39,899	27
	6	Tennessee	60 - 61, 65, 71, 73 - 96	4,136	15
	7	Upper Mississippi	60 - 96	34,382	68
	8	Lower Mississippi	60 - 97	44,649	163
	10	Missouri	60 - 97	62,767	120
	11	Arkansas-White-Red	60 - 97	46,863	206
	12	Texas-Gulf	60 - 61, 64 - 96	7,268	72
	16	Great Basin	64, 66 - 97	19,930	13
	18	California	60 - 96	41,999	57
STORET Median TSS - Lakes and Reservoirs					
Group					
East	2	Mid-Atlantic	63, 66 - 69, 73 - 93	549	6
	3	South Atlantic-Gulf			
	4	Great Lakes			
	5	Ohio			
	6	Tennessee			
Mississippi	7	Upper Mississippi	60 - 63, 67, 72 - 75, 77, 83, 93, 95 - 96	1,694	38
	8	Lower Mississippi			
Midwest	10	Missouri	60, 62 - 79, 81 - 96	2,142	70
	11	Arkansas-White-Red			
	12	Texas-Gulf			
Great Basin	16	Great Basin	80 - 82, 85	35	1
California	18	California	74 - 79, 88, 90	23	9

general population (i.e., the human population located across the United States) resulting from the ingestion of agricultural commodities that are produced within study areas and impacted by dioxin released from HWC facilities but distributed nationally for consumption. This section describes the data sources and methodologies used to generate the sector-level human and livestock population estimates that were used in the HWC risk analysis.

4.4.1 Human Receptor Populations

Sector-level population projections for human receptors could be generated only for "enumerated receptors" (i.e., those receptor populations for which U.S. Census and Census of Agriculture data could be used to generate sector-level population estimates). The enumerated receptor populations considered in the HWC risk analysis were: residents; home gardeners; and commercial beef, dairy, pork, and produce farmers.

The recreational fishers are an enumerated receptor population but with some important differences from the other receptor populations. The recreational fisher is discussed separately in Section 4.4.1.2.

4.4.1.1 <u>Enumerated Receptor Populations</u>. Estimation of sector-level population totals for enumerated receptor populations involves the use of both 1990 U.S. Census (U.S. EPA, 1995) block-group-level data (U.S. Census data) and 1987/1992 Census of Agriculture (U.S. Department of Commerce, 1993) county-level data (Census of Agriculture data)[7]. The U.S. Census provides detailed population density data, which are broken down into the number of *total persons* and the number of *persons in rural area on farm*. The HWC risk analysis estimates risks for four separate age groups for each receptor population (0-5, 6-11, 12-19, and >19 years). Therefore, U.S. Census data, for both total persons and persons in rural areas on farms, were obtained for each of these four age groups. However, the U.S. Census does not provide a detailed breakdown of the type of agricultural activity for individuals or families (e.g., how many beef cattle farm families or dairy farm families are present in a given census block). Therefore, county-level Census of Agriculture data, which do contain detailed agricultural activity data at the farm level, were used in conjunction with the U.S. census data.

Because individual U.S. Census block groups often do not correspond exactly to the shape of individual sectors within a given study area (e.g., some Census blocks may overlap several sectors while others are contained completely within a given sector), it is often necessary to apportion a given Census block group's population between several sectors. The assumption was made in the HWC risk analysis that the U.S. Census block group populations are evenly distributed within each Census block. Therefore, the proportion of a Census block group that lies within a given sector was used to determine the proportion of that Census block group's population that was apportioned to that sector (see Figure 4-3).

[7] The Census of Agriculture county-level data are collected on a 5-year cycle. The most recent collection efforts (1987 and 1992) did not include 1990, the year when the most recent U.S. Census data were published. Therefore, to match the U.S. Census data with the Census of Agriculture data with regard to year of coverage, Census of Agriculture data from 1987 were averaged together with Census of Agriculture data from 1992 in order to represent 1990.

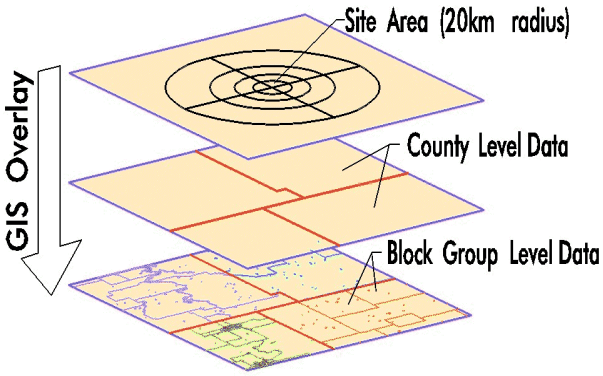

Figure 4-3. Example of inputs to sector/population averaging program.

County-level Census of Agriculture data were then used to further differentiate sector population estimates for total farmers (differentiated into four age groups) into estimates for specific farmer receptor populations (e.g., to convert the number of farmers >19 years of age in a given sector into the number of beef cattle farmers >19 years of age or the number of dairy cattle farmers >19 years of age). Because Census of Agriculture data are available only at the county level and not at the smaller scale block level, the assumption was made that the ratios of *specific farm type* to *total farms* at the county level applied uniformly across the entire county. This assumption allowed the trends in specific farm family ratios (e.g., the percentage of farm families that are dairy farm families) to be applied to all sectors that fall within a given county. When a given sector extended across more than one county, the specific farm family ratios from the different counties were apportioned based on the area proportion of the sector that each county overlapped.

The enumeration of population was conducted for each study area for each facility independent of other HWC facilities regardless of the proximity of these other facilities. Therefore, there are situations in which the 20-km study areas of two or more facilities overlap but the effect of this overlap was not considered. Overall, approximately 15 percent of the individuals residing within HWC study areas are impacted by more than one HWC facilities.

The effect of overlapping study areas on risk results is not known. The aggregate impact of chemical constituents emitted from multiple facilities on individuals residing within overlap areas was not evaluated. Failure to model aggregate impacts for human receptors residing in the area of overlap would underestimate chemical constituent concentrations. On the other hand, the overlap of study areas results in “double counting” of exposed individuals, since the same overlap population is assessed separately for each facility. Because some areas are impacted by more than two facilities, the amount of double counting varies. Those facilities located at industrial facilities in proximity to one another, such as on-site incinerators, overlap with greatest frequency. In aggregate, the percent of double counting is approximately 24 percent.

The following U.S. Census and Census of Agriculture data categories were used to differentiate specific receptor populations for each sector using the methods described above:

- **Residents:** U.S. Census data were used to estimate the number of residents in each of the four age groups of interest. Residents were further differentiated into "residents" and "residents who are home gardeners." The percentage of residents who are home gardeners was set at 38 percent (U.S. EPA, 1997, *Exposure Factors Handbook* Table 13-1, 1986 Vegetable Gardening by Demographic Factors). This percentage applied to all age groups; that is, children of home gardeners were included in the home gardener population. Nonfarm resident household population estimates were adjusted to exclude nonfarm residents engaged in home gardening so that these two receptor populations were mutually exclusive.

- **Beef cattle farmers:** The total number of individuals on farms, obtained from the U.S. Census data, was adjusted by the ratio of beef farms to total farms obtained from county-level Census of Agriculture data. The specific Census of Agriculture data category used to represent beef farmers was "beef cows (farms)" obtained from Table 1, County Summary Highlights.

- **Dairy cattle farmers:** The total number of individuals on farms, obtained from the U.S. Census data, was adjusted by the ratio of dairy farms to total farms obtained from county-level Census of Agriculture data. The specific Census of Agriculture data category used to represent dairy farmers was "milk cows (farms)" obtained from Table 1, County Summary Highlights.

- **Pork farmers:** The total number of individuals on farms, obtained from the U.S. Census data, was adjusted by the ratio of pork farms to total farms obtained from county-level Census of Agriculture data. The specific Census of Agriculture data category used to represent pork farmers was "hog and pig inventory (farms)" obtained from Table 1, County Summary Highlights.

- **Produce farmers:** The total number of individuals on farms, obtained from the U.S. Census data, was adjusted by the ratio of produce farms to total farms obtained from county-level Census of Agriculture data. The produce receptor is intended to include all individuals engaged in raising exposed fruits/vegetables and root vegetables. Therefore, the following Census of Agriculture data categories were summed to obtain an estimate of the total number of farms raising these crops: "Irish potatoes (farms)," "Veg hv for sale (farms)," "Land in orchards (farms)," and "Dry edible beans, exc dry limas (farms)." Each of these data categories is found in Table 1 of the Census of Agriculture Data, County Summary Highlights.

Individual commercial farmer receptor populations (i.e., beef, dairy, pork, and produce farmers) were determined by multiplying total farm population within a sector by the percentage of total farm population that represents each type of commercial farmer. These percentages were obtained from the U.S. Census of Agriculture, which lists farm types and the percentage of total farmers within the farm type. These U.S. Census of Agriculture percentages total to more than

100 percent of total farmers, indicating that an individual farmer who is engaged in both beef and dairy production is counted in both groups. For the purposes of this risk analysis, counting a farmer in more than one group was consistent with the intent of the commercial farmer receptor populations, which was to determine exposures associated with a particular type of farming activity. In the case of multipurpose farms, the farmer is exposed by more than one route (e.g., beef and dairy) and is counted in both receptor populations. The fact that this farmer was counted separately means that the peak exposure that may result from multipurpose farming activity was not considered. It should also be noted that not all farm categories were considered in this risk analysis and, therefore, commercial farm receptor populations do not total to the number of total farms. Only farm types that represent the most important exposure pathways for the constituents modeled in this risk analysis were considered.

Table 4-10 presents the enumerated population counts for each human receptor population. These receptor population totals are shown for each source category. The population counts in Table 4-10 are facility-weighted values, meaning that they are the total estimated population counts for individuals residing within 20 km of all HWC combustor facilities nationwide.

The sector-level estimates for each receptor population obtained using the methodologies detailed above are combined with sector-specific individual risk estimates for each receptor population to project population risks for a given study area.

4.4.1.2 <u>Recreational Fisher</u>. A key factor in generating sector-level risk estimates for the recreational fisher was the ability to characterize the magnitude of recreational fishing activity at specific modeled waterbodies. Unlike the other enumerated receptor populations, risk for recreational fishers is not primarily dependent on their sector location but rather on which waterbodies they frequent for fishing. Multiple factors influence the level of fishing activity at a specific waterbody including: (1) population density within the study area in which the waterbody is located, (2) accessibility to the waterbody, and (3) the presence of competing waterbodies (with regard to fishing activity) in the vicinity of the waterbody under evaluation. If a relatively larger number of waterbodies favored for recreational fishing activity were located outside of a given study area (but still within a reasonable fishing trip travel distance), then recreational fishers residing within that study area may regularly travel outside of the study area to fish at waterbodies that are less impacted by HWC emissions because those waterbodies are farther from the HWC facility.

Local population risk was characterized for the recreational fisher using semiquantitative risk statements (see Section 6.2.2). These semiquantitative population risk statements required the generation of study-area-level recreational fisher population projections (instead of sector-level projections). This site-characterization task was conducted using 1991 National Survey of Fishing, Hunting, and Wildlife data (U.S. DOI, 1993). Specifically, National Survey data provide county-level values for the fraction of the rural and urban population that engages in recreational fishing activity and is 16 years old or older. These county-level values were applied to each of the U.S. Census block groups within a given study area (block groups are differentiated into urban versus rural categories) to project the number of recreational fishers per block group. The block-group-specific estimates for recreational fishers were then spatially apportioned to the sectors within a given study area in the same manner used for other receptor populations to

Table 4-10. Population Summary by Source Category and Receptor (V2)1

	CK	CINC	LWAK	OINCS	OINCL	Area Sources: CK	Area Sources: INC	All INC
Resident								
0-1 yr	7,942	43,152	6,948	365,994	157,524	261	32,698	566,670
0-5 yr	54,736	291,622	47,753	2,639,476	1,008,948	2,016	213,370	3,940,045
6-11 yr	56,158	290,335	46,083	2,665,349	1,019,878	2,222	223,839	3,975,561
12-19 yr	74,096	372,948	64,333	3,519,595	1,280,145	3,338	292,876	5,172,688
20 yr +	477,459	2,353,478	424,962	23,799,666	8,303,097	16,972	1,702,740	34,456,241
Total	662,450	3,308,383	583,130	32,624,085	11,612,067	24,548	2,432,825	47,544,535
Home Gardner								
0-1 yr	4,868	26,448	4,259	224,319	96,547	160	20,041	347,314
0-5 yr	33,548	178,736	29,268	1,617,743	618,387	1,236	130,775	2,414,866
6-11 yr	34,419	177,947	28,244	1,633,601	625,086	1,362	137,192	2,436,634
12-19 yr	45,414	228,581	39,430	2,157,171	784,605	2,046	179,504	3,170,357
20 yr +	292,636	1,442,454	260,461	14,586,892	5,088,995	10,402	1,043,615	21,118,341
Total	406,018	2,027,718	357,403	19,995,407	7,117,073	15,045	1,491,086	29,140,199
Beef Farmer								
0-1 yr	80	83	23	182	118	9	79	383
0-5 yr	631	661	160	1,256	768	77	621	2,684
6-11 yr	721	806	166	1,481	908	82	786	3,195
12-19 yr	947	920	246	1,857	1,167	119	896	3,945
20 yr +	5,291	5,513	1,466	10,509	6,504	632	5,072	22,526
Total	7,590	7,900	2,038	15,104	9,347	910	7,375	32,350
Dairy Farmer								
0-1 yr	16	15	4	24	28	1	15	67
0-5 yr	122	123	27	171	191	6	122	485
6-11 yr	149	147	28	194	217	6	147	558
12-19 yr	180	171	39	255	268	9	173	694
20 yr +	1,029	1,018	231	1,555	1,511	45	994	4,084
Total	1,480	1,460	325	2,174	2,187	65	1,436	5,821

(continued)

Table 4-10. (continued)

	CK	CINC	LWAK	OINCS	OINCL	Area Sources: CK	Area Sources: INC	All INC
Produce Farmer								
0-1 yr	6	9	2	24	42	0	8	76
0-5 yr	43	80	12	192	311	2	67	584
6-11 yr	51	90	12	198	337	2	77	625
12-19 yr	63	110	17	256	435	3	91	801
20 yr +	363	665	98	1,604	2,471	16	544	4,740
Total	521	946	137	2,249	3,554	23	778	6,749
Pork Farmer								
0-1 yr	33	12	4	78	46	4	11	136
0-5 yr	244	101	25	526	311	31	91	938
6-11 yr	279	121	26	608	362	34	114	1,092
12-19 yr	359	146	38	780	443	49	137	1,370
20 yr +	1,986	861	225	4,536	2,441	270	750	7,838
Total	2,868	1,228	314	6,451	3,558	384	1,092	11,237

generate an estimate of the number of recreational fishers per sector. The estimates for each of the 16 sectors within a given study area were then summed to generate an overall estimate for the total recreational fishers within a given study area. Although these study-area-level population estimates were generated using sector-level calculations, the underlying data do not have sufficient resolution to allow sector-level population inferences to be drawn and used in risk characterization.

4.4.1.3 Enumeration of Human Populations for PM Analysis. To evaluate functions that relate particulate matter to specific health effects for a specific subpopulation (e.g., ages 65 and over), estimates are needed of the number of people in a particular population subgroup who are exposed to a given change in air quality. For this risk assessment, in addition to the receptor populations described in Section 4.4.1.2, it was necessary to develop sector-level population estimates for the variety of population subgroups that were examined by the PM concentration-response functions.

For the PM analysis, sector-level population estimates developed from the U.S. Census were available for two age categories that are commonly examined in the concentration-response functions used in this analysis: ages 18 to 65 and ages 65 and over. For other age groups needed for the PM analysis, the percentage of persons in the subpopulation at the county level was applied to the sector level. For example, Census data are available that estimate that, in Autauga

County, AL, 5 percent of the population is between the ages of 8 and 12 (the population examined by Schwartz et al. (1994) in a study of lower respiratory symptoms). This percentage was then multiplied by the total sector population to estimate the total number of children ages 8 to 12 in a sector that lies completely within Autauga County.

The above-described method is straightforward when a sector lies completely within one county; however, many sectors lie in multiple counties. Sectors lying in more than one county were assigned a spatially weighted average of the county-level subpopulation percentage breakdowns. This spatially weighted average was determined by multiplying the proportion of each sector (in terms of area) located in a given county by that county's subpopulation percentage. The resulting proportion-adjusted county-specific data were then summed for all the counties in which a sector lies, giving an estimate of sector-level subpopulation percentages. This spatially weighted averaging method assumes that county populations are uniformly distributed throughout the county. See Appendix E to obtain a more complete explanation of how the population data were incorporated into the PM analysis.

4.4.2 Livestock Populations

The projection of livestock populations at the sector level also involves integrating U.S. Census block-group-level data with Census of Agriculture county-level data. The U.S. Census data provide detailed estimates of the number of farms located within each sector. These sector-level estimates were modified using adjustment factors derived from county-level Census of Agriculture data to estimate the total number of animals, for livestock animals of interest, located within each sector[8]. The adjustment factors used were

- **Proportion of total farms that are within each specific farm category:** This adjustment factor converts the sector-level total farm numbers into totals for each of the farm categories (e.g., beef farms and dairy farms).

- **Average number of animals located on a single farm:** This adjustment factor allows the number of farms within a given sector to be converted to number of animals for livestock categories of interest within a given sector.

The use of Census of Agriculture data in this manner assumes that trends in the county-level data hold across the entire county and therefore can be applied to all sectors falling within that county.

The assumption was also made that, when a given U.S. Census block group falls within several sectors, the total number of farms within that block group can be apportioned to the different sectors based on the relative portion of that Census block that falls within each sector. This assumption is the same as that used in projecting human receptor populations.

[8] The HWC risk analysis evaluated dioxin cancer risks to the general public from the ingestion of the following food commodities: pork, beef, and milk. Therefore, sector-level livestock population projections were completed for beef cattle, dairy cattle, and hogs.

It is important to note that the Census of Agriculture includes all farms, irrespective of whether there is a house located on the farm, while the U.S. Census data include only those farms containing houses. Because livestock on all farms were of interest, not just farms with houses, the ratio of *total farms*, obtained from the county-level Census of Agriculture data, to *housing units rural (farm)*, obtained from the county-level U.S. Census data, was used to adjust the sector-specific estimates of total farm numbers. This ratio corrects for the fact that the U.S. Census data, which form the basis of the sector-level projections of total farms, do not include farms without houses.

The sector-level estimates for each livestock category obtained were used in assessing national population cancer risk (see Section 8.3.1.2).

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5.0 Fate and Transport Modeling

This section describes the methodology used to estimate the fate and transport of chemical constituents through the environment. The end result of the fate and transport analysis is media concentrations to which humans and ecological receptors are exposed. Section 5.1 discusses the air dispersion and deposition modeling conducted using the Industrial Source Complex Model -Short Term Version 3. Section 5.2 presents the methods used to spatially integrate air concentrations and deposition fluxes to determine average normalized values for sectors, watersheds, and waterbodies. Section 5.3 discusses the methodologies used to estimate chemical- and facility-specific air, soil, and water concentrations used in the human health and ecological risk analyses. The methodology for calculating food chain concentrations based on air, soil and water concentrations is discussed in Section 5.4.

Figure 5-1 shows an overview of the modeling steps involved in the fate and transport analysis. Other than data on meteorological conditions, the primary inputs to this modeling, facility engineering information (e.g., emissions data, stack parameters) and site characteristics (e.g., study area dimensions, land use, terrain), are discussed in Section 4.0. Air modeling produces an array of air concentrations and deposition fluxes that are normalized to a unit emission rate (1 g/s). These concentration and deposition values are then input to a geographical information system, which integrates the air modeling data over the spatial extent of geographic features in the study area. This spatial integration produces normalized average air concentrations and deposition fluxes for sectors, watershed areas, and waterbodies.

Normalized air concentrations and deposition fluxes for sectors, watersheds, and waterbodies are then combined with facility-specific air emissions data to calculate sector, watershed, and waterbody environmental media concentrations. Waterbody concentrations are also a function of the constituent levels in the soil of the waterbody's watershed. Sector air concentrations are used directly to determine inhalation pathway intakes. Sector air and soil concentrations are used to calculate terrestrial food chain concentrations (e.g., vegetables, beef, pork). Waterbody concentrations are used directly to determine drinking water pathway exposure and to calculate aquatic food chain concentrations (e.g., fish). Sector average soil concentrations and waterbody concentrations (as well as fish tissue concentrations) are also used in the screening-level ecotoxicological assessment (see Section 9.0).

5.1 Dispersion and Deposition Modeling

5.1.1 ISCST3 Dispersion Modeling

The HWC risk analysis required an air dispersion model that could

- Model continuous releases from multiple point sources (stacks) at a single facility for site-specific analyses

- Compute annual average air concentrations and depositions in either simple or complex terrain, depending on location

- Model the effects of building downwash on the plume when site-specific building information was available.

ISCST3, a steady-state Gaussian plume dispersion model, was chosen for this analysis because it can be used to model a variety of sources (U.S. EPA, 1995a). It can estimate concentration, dry deposition rates (particles only), and wet deposition rates and is approved by EPA for the following regulatory applications:

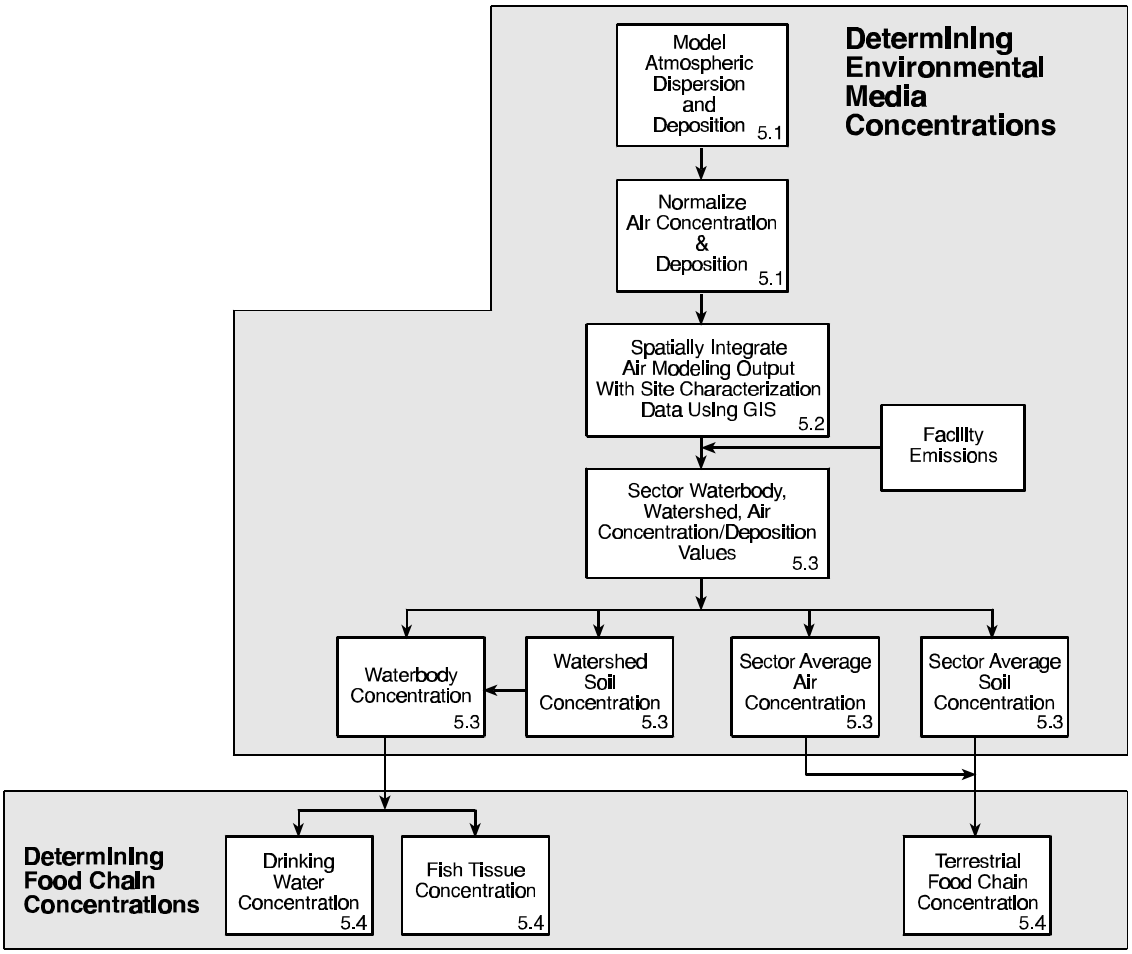


Figure 5-1. Overview of emission and modeling steps followed to arrive at media concentrations in the final rule analysis.

- Industrial source complexes
- Rural or urban areas
- Simple or complex terrain
- Transport distances of less than 50 km
- Averaging times from hourly to annual
- Continuous releases.

The input file for ISCST3 is composed of six pathways: control pathway (CO), source pathway (SO), receptor pathway (RE), meteorological pathway (ME), terrain grid pathway (TG), and output pathway (OU) (U.S. EPA, 1995a). The following sections outline the construction of model runs by pathway. Figure B-1, Appendix B, contains examples of runstream files used in this analysis.

5.1.1.1 Control Pathway. Rural dispersion was selected for all runs. Of the 76 facilities modeled, 57 are located in rural areas. Nineteen facilities are located in areas that are 50 percent or greater urban in nature. Therefore, the predominance of facilities are in rural settings, which supports the selection of rural for an overall classification. The distinction between urban and rural was not based on the procedures outlined in the EPA *Supplement C to the Guidelines on Air Quality Models* (U.S. EPA, 1995c). Modeling some facilities with rural dispersion instead of urban dispersion introduces greater uncertainty because of differences in the way certain dispersion parameters are determined within ISCST. These differences include the following:

- Dispersion coefficients used in urban areas differ from those used in rural areas. Urban coefficients account for greater effects of mechanical turbulence, which are caused by the interaction between the wind and man-made structures.

- Wind profile exponents used in urban areas differ from those used in rural areas. Greater variation in winds with height is seen in rural areas during stable conditions due to the lack of mechanical turbulence, which causes more mixing in urban settings.

- Different mixing heights are used by ISCST3 depending on whether rural or urban parameters are selected. Rural and urban mixing heights are both calculated by the PCRAMMET preprocessor and are found in different columns. Urban nighttime mixing heights are slightly higher than rural ones due to urban heat island effects.

At distances close to the source, urban dispersion conditions will normally increase ground-level concentrations. Hence, by selecting the rural dispersion option for all facilities, this analysis underestimated ground-level concentration data close to the source for those facilities actually located in urban areas. This potential underestimation of ground-level concentrations near the source occurred at the 21 facilities that are in urban settings. The magnitude of this discrepancy varies depending on the meteorological data being used for each facility and the stack parameters at the facility.

The influence of differences between urban and rural dispersion, which are most pronounced during nighttime hours, is somewhat mitigated by the fact that, except for the

analysis of particulate matter health effects, averages (annual averages) were calculated over the long term and then spatially averaged over sectors and waterbodies for use in this risk analysis.

Annual averaging time was selected because human and ecological risk benchmarks are based on long-term exposure. For the particulate matter health effects evaluation, 24-h averages were also calculated (Section 5.3.1.2). Five years of meteorological data were evaluated. Within ISCST3, air concentration and deposition values were determined for each of the 5 years. The annual average values, which were used in the risk assessment, were determined within ISCST3 by averaging the five annual averages (i.e., by taking a "period" average over the 5-year modeling period).

The following regulatory default settings were selected:

- Stack tip downwash
- Buoyancy-induced dispersion
- Final plume rise
- Calm wind processing routine
- Default values for vertical wind profile exponents and vertical potential temperature gradients
- Upper-bound estimation for super squat buildings that have an effect on lateral plume dispersion.

ISCST3 was run 10 times for each facility. These runs resulted in

- Air concentration of particles
- Air concentration of vapors
- Dry deposition of particles
- Wet deposition of particles
- Combined wet and dry deposition of particles
- Wet deposition of vapors
- Air concentration of elemental mercury vapors
- Air concentration of divalent mercury vapors
- Wet deposition of elemental mercury vapors
- Wet deposition of divalent mercury vapors.

Wet and dry depletion of the plume concentration was selected for all runs.

5.1.1.2 <u>Source Pathway</u>. Source characteristics required for ISCST3 include physical location of the stack(s), emission rates, other stack parameters, and particle size information. The number of stacks varied from facility to facility in this analysis. Coordinates of a single stack were set at (0,0). Coordinates of multiple stacks were assumed to be (10,10), (-10,-10), (10,-10), (-10,10) if actual coordinates were not known. Source groups were set to one stack per group so

that each stack's contribution to the total air concentration or deposition flux resulting from an individual facility could be examined.

A unit emission rate of 1 g/s was used for ISCST modeling. Chemical- and facility-specific emission rates were factored in at a later stage in the analysis. Other source parameters, such as exit velocity, stack gas temperature, stack height, and stack diameter, were entered as appropriate. The effect of buildings near the stack (building downwash) on constituent dispersion was considered for those facilities for which building dimensions were available.

Complete building information (e.g., drawings of locations and dimensions of the buildings relative to the stack) was available from EPA Regional office files for only seven facilities. Building heights and some building dimensions were available from EPA permit applications. The available information was evaluated using the Building Profile Input Program (BPIP) (U.S. EPA, 1993b). The BPIP determines whether buildings have an effect on the plume trajectory and creates the necessary inputs to the ISCST3 runstream file. Only four of the facilities for which sufficient data were available were determined to have an effect, and these four were modeled using building downwash within ISCST3. If buildings were located near the stack and insufficient information was available to permit modeling of building downwash, the air concentration and deposition values downwind of the stack could be underestimated. There is uncertainty as to the number of facilities affected by building downwash and the magnitude of the underestimation of concentration and deposition. It is known, however, that the most significant differences in concentration and deposition values will occur very close to the facility. The uncertainty introduced by not having complete information to characterize building downwash is somewhat mitigated by the temporal and spatial averaging of the air modeling results that is done prior to their use in this risk analysis.

Tables B-1 through B-5 in Appendix B list the facility parameters used for modeling each facility.

Particle size distributions and scavenging coefficients specific to the combustion unit type were used in the modeling effort. Data on combustor unit-type-specific particle size distributions were gathered by EER (Springsteen and Rizeq, 1997). These data were in the form of particle size distribution by mass. To properly represent the surface area available for sorption of chemicals onto the surface of particles, particle size distribution by mass was transformed into particle size distribution by surface area following EPA guidelines (U.S. EPA, 1998). Whether or not this is appropriate is a point of uncertainty. There are two primary uncertainties associated with this method. First, particle size distributions were generalized by combustion unit type. In reality, the particle size distribution may vary by specific unit due to the type of air pollution control device used and the specific waste being burned. This method also assumes that the particles being emitted are perfectly spherical, which is not likely the case. Second, use of particle size distribution by surface area is intended to represent the dispersion and deposition of constituents that are sorbed onto particles, such as volatile organics. The method does not accurately account for metals in the stack effluent, which are in the particulate phase. These would be better represented by a particle size distribution by mass.

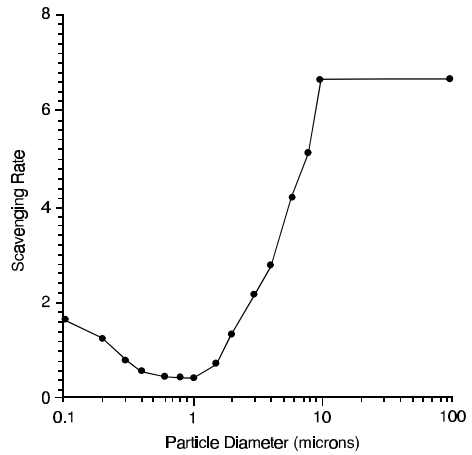
Table 5-1 shows the particle size distribution and wet scavenging coefficients for each combustion unit type. A value of 1.7E-04 was used for the gas-scavenging coefficients. This value was estimated by representing vapor as a 0.1-μm particle and using Figure 5-2 to estimate the scavenging coefficient for both vapor and particle. Species-specific gas scavenging rates were used for elemental and divalent mercury (see Section 5.1.2 for a discussion of deposition modeling).

5.1.1.3 Receptor Pathway. Receptors were laid out in a polar grid with 23 receptor rings and 32 radials, 11.25 degrees apart. Twenty-three receptor distances were modeled. The distances (in meters) were 100, 150, 200, 300, 400, 500, 700, 1,000, 1,500, 2,000, 3,000, 4,000, 5,000, 6,000, 7,000, 8,000, 9,000, 10,000,12,000, 14,000, 16,000, 18,000, and 20,000. The farthest receptors from the source were at 20 km. When elevations were included in the receptor pathway, the polar coordinates were converted to Cartesian coordinates.

Table 5-1. Particle Size and Wet Scavenging Coefficients Used in Dispersion Modeling

Representative Particle Diameter (μm)	Fraction of Surface Area [a]	Wet Scavenging Rate Coefficient (h/mm-s)[b]
Cement Kilns and LWAKs		
1.6	0.83	7.0E-05
7.0	0.16	4.6E-04
24.0	0.01	6.7E-04
Solid Waste Incinerators		
0.7	0.84	4.0E-05
1.8	0.12	8.0E-05
7.4	0.04	4.7E-04
Liquid Waste Incinerators		
0.2	0.80	1.3E-04
0.4	0.13	6.0E-05
0.8	0.07	4.0E-05

LWAK = Lightweight aggregate kiln.
[a] Springsteen and Rizeq, 1997.
[b] U.S. EPA, 1995d.



Source: Jindal and Heinold, 1991, as cited in U.S. EPA, 1995d.

Figure 5-2. Wet scavenging rate coefficient as a function of particle size.

The decision to use simple or complex terrain was made by screening sites for elevated terrain in the immediate proximity of the stack. If terrain elevation was greater than or equal to 100 feet above the stack within 2 km of the facility stack, the facility was modeled for complex terrain. Otherwise, the facility was modeled for simple terrain. The *Supplement C to the Guideline on Air Quality Models* (U.S. EPA, 1995c) indicate that any terrain above stack height should be modeled as complex terrain.

The ISCST3 model classifies terrain three ways:

- Simple (below stack height)
- Intermediate (above stack height, but below plume)
- Complex (above plume height).

When ISCST3 is executed, it invokes the complex1 algorithm if intermediate or complex terrain exists, as determined by examining the elevation of receptors. In the complex terrain case, it uses only complex1 results. In the intermediate terrain case, it uses both models and selects the higher concentration and deposition values that result from ISC or complex1. In the simple terrain case, only ISC is executed. In any event, the ISC model cuts off terrain above stack height.

By screening sites for complex terrain outside the ISCST3 model using the method described above, it is possible that some sites that ISCST3 would have treated as intermediate or complex terrain were modeled as simple terrain resulting in lower ground-level concentrations and deposition rates. Thirteen facilities were modeled for complex terrain. The number of facilities that were modeled as simple terrain instead of complex terrain is not known. The magnitude and extent of uncertainty introduced by screening facilities outside the ISCST3 model is also not known.

For those facilities that were modeled as complex terrain cases, a Cartesian array of receptor elevations was input to the ISCST3 model and terrain was modeled as indicated above in accordance with U.S. EPA (1995a). Receptor elevations were determined using 1:250000 scale USGS Digital Elevation Model (DEM) files. A DEM consists of a sampled array of elevations for ground positions that are normally at regularly spaced intervals. The 1-Degree DEM (3- by 3-arc-second data spacing) provides coverage in 1- by 1-degree blocks for all of the contiguous United States, Hawaii, and limited portions of Alaska. The basic elevation model is produced by or for the Defense Mapping Agency (DMA) but is distributed by the USGS EROS Data Center in the DEM data record format. In reformatting the product, the USGS does not change the basic elevation information. These 1-degree DEMs are also referred to as "3-arc second" or "1:250,000 scale" DEM data.

For those facilities that were modeled as simple terrain, a polar array of receptors was input to the ISCST3 model and no elevations were used. Not using receptor elevations for the simple terrain case introduces uncertainty into the model analysis by not calculating air concentrations and deposition values at actual receptor elevations, thus underpredicting these values. The magnitude and extent of uncertainty introduced by the simple terrain assumptions is not known.

5.1.1.4 <u>Meteorological Pathway</u>. Five years of data were assembled using the PCRAMMET Meteorological Preprocessor (U.S. EPA, 1995b). The raw surface data were obtained from the SAMSON CD-ROM (U.S. DOC, U.S. DOE, 1993). Precipitation data were included so that wet deposition could be calculated. Upper air data were retrieved from EPA's SCRAM bulletin board (U.S. EPA, n.d.). Land use was estimated using Anderson land use codes, which were created using a GIS (Anderson et al., 1976). Roughness length, Bowen ratio, noontime albedo, and minimum Monin-Obukhov length were estimated based on land use for input into PCRAMMET (U.S. EPA, 1995b). See Section 5.1.2 for further discussion of these parameters. Table B-6 in Appendix B shows the inputs used in PCRAMMET for all cases. Meteorological stations used to model facilities evaluated in their risk analysis are presented in Table B-6 of Appendix B.

Specific surface and upper air stations were assigned to each combustion facility based on multiple factors: location relative to one another, terrain effects on climate and wind patterns, and proximity to major waterbodies. In some locations, especially data-sparse areas such as the Rocky Mountains, efforts were made to select the most representative stations, but representativeness is an area of uncertainty.

Missing meteorological data were supplied using the guidance set forth by Atkinson and Lee (1992). If missing data exceeded 90 percent of the data file, that year of data was discarded and an alternate year was chosen. Most often, only an isolated record of data was missing. Generally, these cases were filled in objectively, by interpolation, using the preceding and subsequent records. When multiple consecutive records were missing, the values were filled in using professional judgment. Appendix B, Table B-6, lists the meteorological stations used for each facility.

5.1.1.5 <u>Terrain Grid Pathway</u>. This pathway was not used in this analysis. The terrain grid pathway can be used with ISCST3 in complex terrain situations and involves a separate

input file. Use of this pathway increases the accuracy of the estimated amount of dry plume depletion caused by the effects of intervening terrain. Concentration and dry deposition rates were probably overestimated for receptors on the far side of intervening terrain and may be underestimated for the near side. Because there are a number of uncertainties introduced via air dispersion modeling that act in different directions, the impact on risk estimates is not known.

5.1.1.6 Output Pathway. The ISCST3 model output was formatted to fit the needs of subsequent modeling procedures: indirect exposure modeling and particulate matter human risk modeling. Plotter files, which contain receptor location and associated air quality data, best fit these subsequent modeling procedures.

Output files for all constituents except PM contained air quality data for annual averages. Air quality data for PM included 24-hour averages in addition to annual averages.

5.1.2 Deposition Modeling

ISCST3 computes wet deposition rates of particles and vapor and computes dry deposition rates of particles. Dry deposition of vapors is not computed by ISCST3 but, for this analysis, dry deposition of vapor was computed outside of the ISCST3 model. Wet deposition is the deposition of material on a surface from a plume as a result of precipitation. The amount of material removed by wet deposition from the plume is a function of the scavenging rate coefficient, which is based on particle size (U.S. EPA, 1995d). Table 5-1 shows the particle size distribution and scavenging rate coefficients used in this analysis. Dry deposition refers to the deposition of material on a surface from a plume of material as a result of processes such as gravitational settling, turbulent diffusion, and molecular diffusion. Dry deposition is calculated as the product of air concentration and dry deposition velocity.

5.1.2.1 Wet Deposition of Particles and Vapor. Wet deposition was modeled for both particles and vapor using ISCST3. To perform these calculations, wet deposition, wet depletion, and dry depletion were all selected in the input runstream file. Precipitation data from the SAMSON CD-ROM were required to process the meteorological inputs for this analysis. Cement kilns and lightweight aggregate kilns emit significantly larger particles than do incinerators. Because these larger particles are removed by precipitation at different rates, it was necessary to base scavenging rate coefficients for particulate matter on particle size distributions specific to the combustion type. The same particulate matter scavenging rate coefficient was used for both liquid and frozen precipitation for each particle size category, as shown in Table 5-1. These particulate matter scavenging rate coefficients were obtained from Figure 5-2 (from U.S. EPA, 1995d). A vapor scavenging rate coefficient of 1.7E-04 was used for all combustor categories. This was obtained by using a 0.1-μm particle as a surrogate for vapor and using the value shown in Figure 5-2.

> Mercury particulate matter was modeled using the same wet scavenging coefficients as the other constituents. Mercury vapor, on the other hand, was modeled using site-specific scavenging rate coefficients for both elemental and divalent mercury. These coefficients were dependent upon the annual average mixing height at the location.

Site-specific wet scavenging rate coefficients for elemental and ionic mercury vapor were input into ISCST3, which multiplied these by the precipitation rate to calculate a wet scavenging coefficient. The wet scavenging rate coefficients were calculated by the equation:

$$\lambda = \frac{W}{H_L}\left(\frac{hours}{3{,}600\ s}\right)\left(\frac{meters}{1{,}000\ mm}\right) \qquad (5\text{-}1)$$

where

λ	=	wet scavenging rate coefficient (h/mm-s)
W	=	washout ratio (unitless)
H_L	=	mixing height (m).

The mixing height was used as an approximation of the height from which precipitation fell. The annual average mixing height for each location was used. This was calculated by averaging the annual average morning and afternoon mixing heights, which were taken from Holzworth (1972). Table B-7, in Appendix B, shows the mixing heights and scavenging rate coefficients at all modeled locations.

The washout ratio is defined as the concentration of a contaminant in surface level precipitation divided by the concentration in surface level air. The values used in this analysis were 1.6E+06 for divalent mercury vapor and 1.6E+04 for elemental mercury vapor. These were taken from the *Mercury Study Report to Congress* (U.S. EPA, 1996). The 1997 *Mercury Study Report to Congress* (U.S. EPA, 1997) gives the values of 1.6E+06 and 1,200 for divalent and elemental mercury, respectively. Uncertainty exists concerning the actual values of the washout ratio because there were few empirical data regarding mercury specifically upon which to base these values. The consequences of these uncertainties were to overpredict (or underpredict) wet deposition with corresponding underprediction (or overprediction) of air concentrations.

Wet deposition of dioxin congener vapors was modeled with a scavenging rate coefficient of 1.7E-04. Values of scavenging rate coefficients calculated based on the log of washout ratios for Indianapolis and Bloomington given in Koester and Hites (1992) span about 2 orders of magnitude when using Equation 5-1 to calculate the scavenging rate coefficients. The values of the results range from 2.52E-06 to 9.89E-04. The value of 1.7E-04 falls in the upper end of this range. Further uncertainty may be introduced due to differences in meteorological regime.

5.1.2.2 <u>Dry Deposition of Particles Using ISCST3</u>. Dry deposition of particles was modeled for all constituents with both dry and wet depletion selected. In order to calculate dry deposition, ISCST3 requires mass mean diameter, particle density, and mass fraction to be input into the source pathway for deposition calculations (U.S. EPA, 1995a). Dry deposition calculations also require the meteorological input file to contain surface friction velocity, minimum Monin-Obukhov length, and surface roughness length (U.S. EPA, 1995b).

Surface friction velocity and minimum Monin-Obukhov length are calculated in the PCRAMMET preprocessor (U.S. EPA, 1995b). Surface friction velocity is calculated in the PCRAMMET preprocessor by the following equation:

$$u_* = \frac{kU}{\ln(\frac{z_{ref}}{z_0}) - \Psi + \Psi_0} \tag{5-2}$$

where

u_*	=	surface friction velocity (m/s)
k	=	von Karman's constant
U	=	windspeed (m/s)
z_{ref}	=	anemometer height (m)
z_0	=	surface roughness height at the measurement site (m)

and Ψ and Ψ_0 are calculated based on an iterative process described in the PCRAMMET User's Guide (U.S. EPA, 1995b).

The Monin-Obukhov length is calculated in the PCRAMMET preprocessor by the equation:

$$L = -\frac{\rho C_P T u_*^3}{kgH} \tag{5-3}$$

where

L	=	Monin-Obukhov length
ρ	=	density of air (kg/m^3)
C_P	=	specific heat capacity of air (J/kg-deg)
T	=	temperature (K)
u_*	=	surface friction velocity (m/s)
k	=	von Karman's constant
g	=	acceleration of gravity (m/s^2)
H	=	sensible heat flux at the surface (W/m^2).

Surface roughness length was calculated from Table B-1 in the PCRAMMET User's Guide (U.S. EPA, 1995b). These values for roughness length were weighted by land use percentage, which was taken from GIRAS spatial data, using a GIS. Land use was based on Anderson land use codes (Anderson et al., 1976).

5.1.2.3 <u>Dry Deposition of Vapors</u>. Dry deposition of vapors cannot be calculated using the current version of ISCST3 (U.S. EPA, 1995a). Dry deposition of vapors must be calculated using a separate step. Dry deposition of vapor is the product of vapor air concentration and a chemical-specific dry deposition velocity. The value used in this study for dry deposition velocity was 0.2 cm/s for all dioxin congeners. There is considerable uncertainty associated with the use of this value. This value represents the average value for all dioxin congeners and is more accurate for particulate deposition. Koester and Hites (1992) indicate that the actual value for vapors is lower. This would tend to lower the amount of dioxin vapor deposited on the surface.

Because dry deposition of vapor phase constituents is calculated external to ISCST3, the plume is not depleted within the model. Because mass balance is not maintained with this approach, there is a tendency to overpredict the deposition of constituent vapors to the ground. Wet deposition of vapors and particles and dry deposition of particles were calculated within ISCST3 and mass balance was maintained for these deposition processes.

Site-specific dry deposition velocity for divalent mercury vapor was weighted based on land use and stability class. Values for dry deposition velocity for each land use category and stability class were found in the *Mercury Study Report to Congress* and averaged to annualized values (U.S. EPA, 1997). These values are meant to be used in the RELMAP model and were calculated based on an assumed similarity between the deposition properties of divalent mercury vapor and nitric acid. Generally, nighttime dry deposition velocity values can be treated as constant across all stability classes and land use types (U.S. EPA, 1997). In this analysis, it was assumed that the nighttime values for dry deposition velocity were simply equal to the daytime values. These assumptions introduced uncertainties into the calculation of dry deposition of vapors.

To calculate the weighted dry deposition velocity, land use was obtained from 1:250,000 scale quadrangles of land use and GIRAS spatial data obtained from the EPA website and placed in an ARC-INFO format (U.S. EPA, 1994b). Land use was based on data from the mid-1970s to the early 1980s. The fraction of time in each stability class was based on 5-year hourly meteorological files used in ISCST3 modeling. Table B-8, in Appendix B, shows the weighted dry deposition velocity for divalent mercury vapor at the modeled facilities. Dry deposition of elemental mercury was not included in this analysis, which is consistent with the 1997 *Mercury Study Report to Congress*.

5.1.3 Air Concentration and Deposition Model Outputs

The air dispersion and deposition modeling, described in Sections 5.1.1 and 5.1.2, respectively, produced output files of data that were used to calculate environmental media concentrations (see Section 5.3) and food chain concentrations (see Section 5.4).

All modeled air concentration and deposition values were annual averages that were averaged together, so they really represent “period” averages over 5 years, with the exception of particulate matter. Particulate matter air concentrations were calculated for the 24-h averaging time in addition to annual averages.

All air concentrations and deposition values were unit values based on modeling a default emission rate of 1 g/s. Later in the modeling process these unit values were scaled with site-specific emission rates for each of the subject constituents to produce values used to calculate environmental media concentrations. This step is discussed in Section 5.3.

The air concentration and deposition values were specific to each of the 76 modeled facilities. For each facility, values were produced for each receptor in a polar array consisting of 32 radials spaced every 11.25 degrees and 23 rings spaced at 100, 150, 200, 400, 500, 700, 1,000, 1,500, 2,000, 3,000, 4,000, 5,000, 6,000, 7,000, 8,000, 9,000, 10,000, 12,000, 14,000, 16,000, 18,000, and 20,000 meters.

For each facility/receptor combination there are 10 values, one for each of the following categories:

- Air concentration of particles (AP)
- Air concentration of vapors (AV)
- Dry deposition of particles (DDP)
- Wet deposition of particles (WDP)
- Combined wet and dry deposition of particles (CDP)
- Wet deposition of vapors (WDV)
- Air concentration of elemental mercury vapors (VHG)
- Air concentration of divalent mercury vapors (VHG2)
- Wet deposition of elemental mercury vapors (WVHG)
- Wet deposition of divalent mercury vapors (WVHG2).

These output files are in plotter file format—files that contain the location of the receptor and the specific value modeled in the ISCST model run (e.g., air concentration of particles). These plotter files were postprocessed (as discussed in Section 5.3) to produce environmental media values used in the human health and ecological risk assessments.

5.2 GIS Processing

A geographical information system model was used to calculate average air concentration and deposition rates for each sector, watershed, and waterbody. This crucial step, as shown in Figure 5-3, combines the spatial characterization of sectors, watersheds, and waterbodies in the study area with 10 air modeling outputs (see Section 5.1.3) for each facility. After the waterbodies and watersheds selected for inclusion in the HWC risk analysis were integrated into the GIS platform (see Section 4.3), the results of air modeling runs were used to generate spatial averages for air concentration and deposition rates for those waterbodies and watersheds. Similarly, the results of the air modeling runs were used to generate normalized spatial averages for normalized air concentration and deposition rates for the 16 sectors located within each of the study areas. The results from this GIS model were then used in equations that will calculate air, watershed, and waterbody values.

This section describes the methodology used to establish air concentration/deposition values for the modeled waterbodies, watersheds, and sectors.

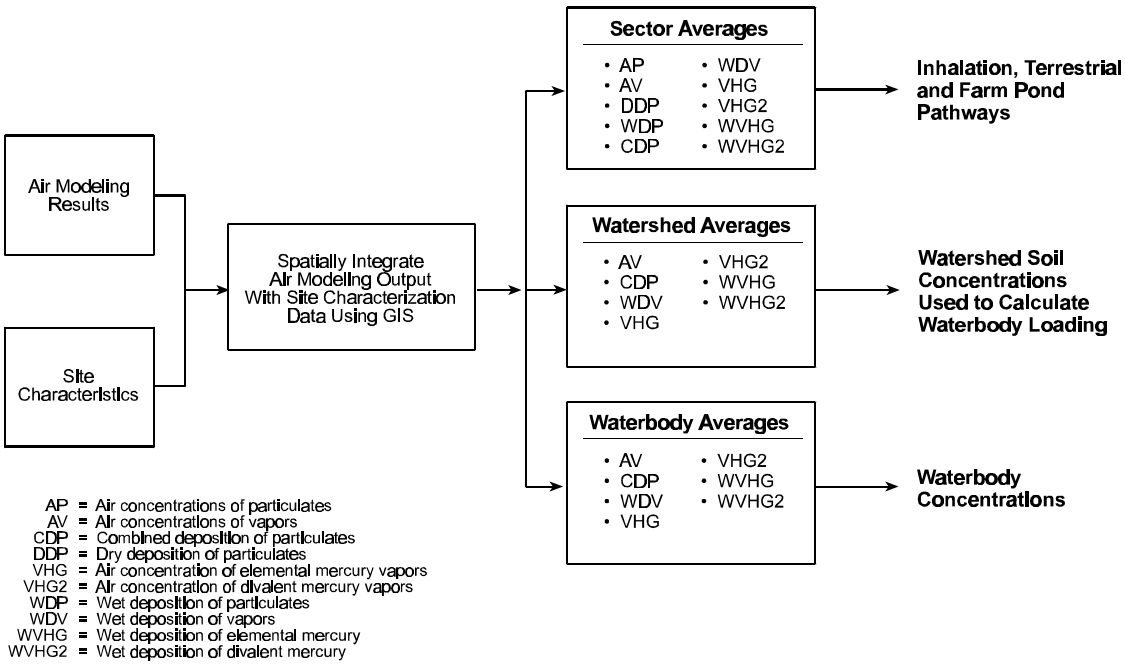


Figure 5-3. GIS modeling of sector, watershed, and waterbody averages.

5.2.1 Waterbodies and Watersheds

Air concentration and deposition values and coordinates were provided by the ISCST3 model in the form of ASCII files labeled individually by site and concentration type. For modeling waterbodies and watersheds, the following values were used:

AV	=	Air concentration of vapors
CDP	=	Combined deposition of particles
WDV	=	Wet deposition of vapors
VHG	=	Air concentration of elemental mercury vapor
VHG2	=	Air concentration of divalent mercury vapor
WVHG	=	Wet deposition of elemental mercury vapor
WVHG2	=	Wet deposition of divalent mercury vapor.

In an automated batch program, the ASCII files produced by ISCST3 were converted from a polar or Cartesian array of values into an evenly spaced grid of concentration values distributed around the center of the study area grid in the form of a GIS point coverage. The program then individually overlaid the watershed and waterbody polygons with this point coverage and averaged the overlapping points (see Figure 5-4). These mean concentration values and their associated watershed or waterbody names are the output of the program and represent the average air concentrations and deposition values falling within that particular waterbody or watershed.

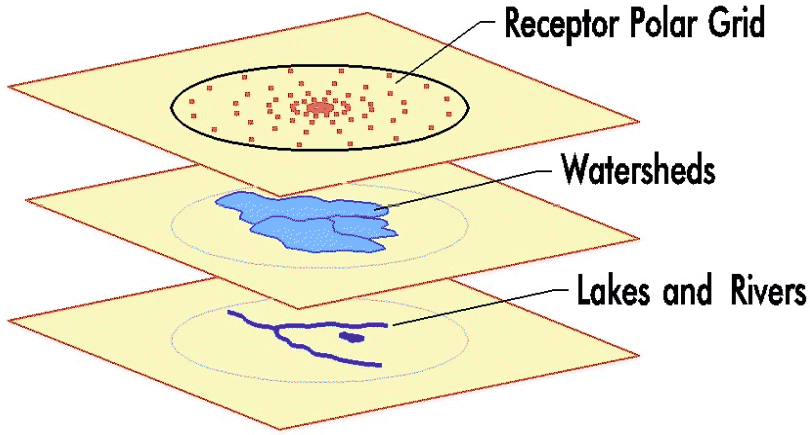


Figure 5-4. Example of inputs to waterbody/watershed averaging program.

5.2.2 Sectors within Study Areas

Air concentration averages and average deposition values by sector were generated in much the same way as the watershed and waterbody averages. Air concentrations and deposition values of the following types were provided by the ISCST3 model in the form of ASCII files:

AP	=	Air concentration of particles
AV	=	Air concentration of vapors
CDP	=	Combined deposition of particles
DDP	=	Dry deposition of particles
WDP	=	Wet deposition of particles
WDV	=	Wet deposition of vapors
VHG	=	Air concentration of elemental mercury vapor
VHG2	=	Air concentration of divalent mercury vapor
WVHG	=	Wet deposition of elemental mercury vapor
WVHG2	=	Wet deposition of divalent mercury vapor.

The polar array of concentration values from the ISCST3 model (which are in polar and Cartesian format) were converted from an ASCII list into a GIS point coverage. One of the main values of the GIS program was its ability to convert the ISCST3 model output into an evenly spaced grid of points for more accurate spatial analysis. This point coverage was then overlaid with the sector coverage (see Figure 5-4). Sector coverages for each site had been created previously with another GIS program and were in the form of polygon coverages. Air concentration and deposition point values within each sector were averaged to determine mean air concentration and deposition values for each sector. Output from this program was an ASCII file with a list of site identification numbers, sector numbers, air concentration averages, and

average deposition values. These average concentration values represent the average air concentration and deposition values falling within that particular sector.

5.3 Chemical Modeling for Environmental Media Concentrations

This section describes the chemical fate and transport modeling used to arrive at concentrations in air, soil, and water. The methodology used to estimate air, soil, and water concentrations is described in Sections 5.3.1, 5.3.2, and 5.3.3, respectively. Figure 5-1 illustrates the steps undertaken to calculate media concentration.

This section describes the methods used to calculate values for seven media concentration values:

- Air concentration for each sector (vapors and particles)
- Soil concentration for each sector (for tilled and untilled soils)
- Soil concentration for each watershed (for untilled soils only)
- Concentration for each waterbody (water column and bed sediment).

Discussions in this section are general in nature, with only the major equations presented. Two appendixes support the discussions with more detailed information:

- Appendix C contains the full set of equations used to calculate media concentrations.

- Appendix D lists the physical/chemical properties used; the parameter values selected for fate, transport, and exposure modeling; and citations for the parameter values selected.

5.3.1 Ambient Air Concentrations for Sectors

This section presents the methods used to calculate ambient air concentrations of both vapors and particles resulting from the operation of HWC units.

5.3.1.1 <u>Calculation of Ambient Air Concentrations</u>. Figure 5-5 shows the method used to calculate values for average vapor and particle concentrations for each sector. Two inputs to this process are facility-specific emission rates and average unit air concentration for each sector, which are derived using dispersion model outputs and GIS as described in Section 5.2. Except for mercury, emission rates were reported at total concentrations without regard to fraction in vapor and particle phases. As discussed below, values from the literature were used to divide total emissions for each of the constituents (except mercury) into vapor and particle phase emission rates.

These inputs (sector average air concentrations and facility-specific emission rates) were then used to calculate average ambient air concentrations for each sector for each facility using the following calculation:

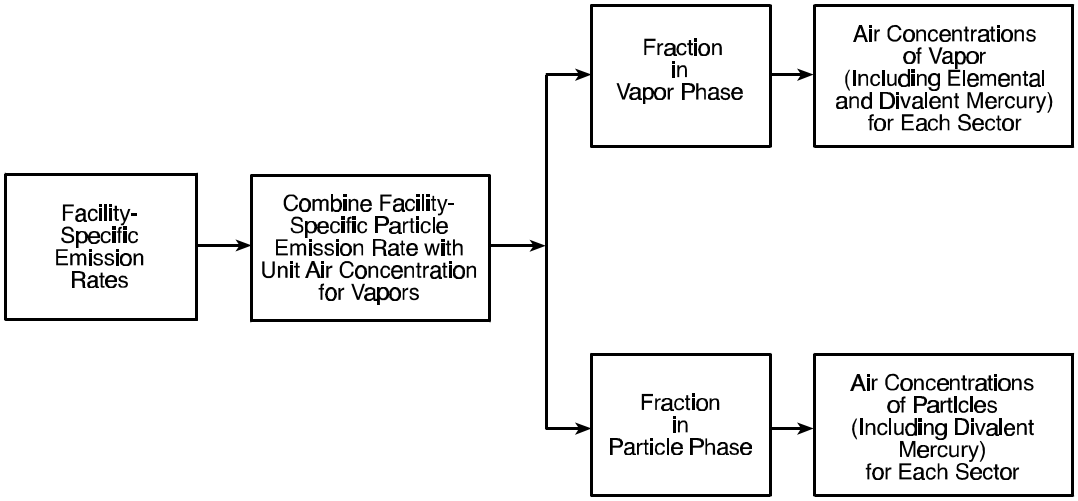


Figure 5-5. Steps involved in calculating sector average air concentrations of constituent vapors and particles.

$$\begin{matrix} \textit{Facility-specific} \\ \textit{air concentration} \end{matrix} = \begin{matrix} \textit{Facility-specific} \\ \textit{emission rate} \end{matrix} \quad \begin{matrix} \textit{Modeled unit} \\ \textit{air concentration} \end{matrix} . \qquad (5\text{-}4)$$

Vapor concentrations are determined by multiplying the vapor-specific unit air concentration by the vapor fraction (Fv)and emission rate. Similarly, particle concentrations are determined by multiplying the particle-specific unit air concentrations by the particle fraction (1-Fv) and emission rate. The total air concentration is the sum of the vapor phase concentration and the particle phase concentration. The fraction of semivolatile compounds that was found in the vapor phase (Fv) was estimated using the Junge-Pankow equation, as cited in Bidleman (1988). The fraction in vapor phase for the individual congeners of dioxins and furans used in this analysis was the same as the values calculated for the Dioxin Exposure Reassessment (U.S. EPA, 1994a). For the majority of metals, Fv was zero; for chlorine and hydrogen chloride, Fv was 1.0. Appendix D contains the values used in this assessment.

Exposure estimates are based on ambient conditions, since the conditions at the points of exposure are more similar to ambient conditions than conditions at point of release. Some chemicals, such as chlorine, are found to be completely in the vapor phase under ambient conditions. The majority of metals (excluding mercury) are particle-bound under ambient atmospheric conditions. The more complex, semivolatile compounds, such as divalent mercury and the dioxin and furan congeners, partition to vapor and particle-bound fractions depending on the ambient conditions of temperature and concentrations of particle surface area available for sorption.

The Junge-Pankow equation was not used to partition mercury between the vapor and particle-bound phases in the atmosphere. The mercury species phase in the atmosphere was determined from facility-specific emission tests. In these tests, mercury was measured as fraction of divalent mercury in the vapor phase, fraction of divalent mercury that was particle-bound, and fraction of mercury emitted that was in the elemental form (vapor phase only). Thus, the vapor/particle partitioning for mercury was estimated at the release point. For mercury, the result of estimating vapor/particles partitioning at the point of release (under stack instead of ambient conditions) was an overestimation of the vapor phase divalent mercury and an underestimation of the particle-bound divalent mercury because, as the gas stream cools to ambient conditions, more of the divalent mercury is expected to condense on particles.

> Stack emissions of total elemental and divalent mercury (g/yr) were received from EER. Also, the site-specific percents of total mercury that are divalent mercury, divalent vapor, and elemental vapor were received. Using this information in conjunction with the assumption that all elemental mercury emissions are vapor, the vapor fraction of divalent mercury was calculated for each site. That is, the percent of divalent Hg that is vapor was calculated from the percent of total mercury that is divalent vapor .

5.3.1.2 <u>Twenty-four Hour Particulate Matter Concentrations</u>. The PM modeling was conducted using ISCST3 in the same manner as described above for other constituents. Although no deposition values were calculated for PM, ISCST3 was run to account for plume depletion due to deposition processes. The results of the ISCST3 analysis were unit air concentrations of particulates. These unit concentrations were multiplied by facility-specific emission rates for $PM_{2.5}$ and PM_{10} to yield $PM_{2.5}$ and PM_{10} concentrations used in the PM health effects analysis.

Particle size distributions specific to each combustor category were used for all model runs. Applying an overall particle size distribution to the modeling of $PM_{2.5}$ and PM_{10} introduces uncertainty into the analysis because particles in the smaller size range deposit at a lower rate than larger size particles. The effect would be to overestimate the depletion of smaller particles and underestimate ambient concentrations. For this analysis, the particle size distributions presented in Table 5-1 were used. These data show that all particles are assumed to be below 10 µm except for 1 percent of the cement kiln and LWAK PM emissions. For incinerators, 100 percent of the liquid incinerators and 96 percent of the solid waste incinerators are below 2.5 µm. Therefore, the effect of not using particle size distributions specific to $PM_{2.5}$ and PM_{10} is minimal.

Air quality data for all constituents except particulate matter were calculated only for annual averages. Air concentration values for PM were calculated for 24-h averages. Twenty-four-hour average air concentration values for PM_{10} and $PM_{2.5}$ were calculated for each sector for each facility using 5 years of meteorological data. For each sector, the set of approximately 1,825 values was used to develop a statistical profile of PM air concentrations. From these 24-h values, mean and median values for the entire 5-year period were calculated. In addition, a discrete frequency distribution of 24-h concentrations was developed using 20 frequency bins where each bin contained values representing $1/20^{th}$ of the year. This frequency distribution for each of the 16 sectors in the facility study area was used to describe the PM_{10} and $PM_{2.5}$ air concentrations for each facility. The frequency distribution subsequently were used to estimate the impact on human health associated with reductions in PM emissions (see Section 8.3.4). Additional

information on the processing of PM air concentrations for modeling PM health effects is provided in Appendix E.

5.3.2 Soil Concentration

Figure 5-6 generally illustrates the method used to calculate values for average constituent concentrations for watershed and sector soils. As with the calculation for air concentrations and deposition rates, this calculation begins by applying facility-specific emission rates (see Section 4.2 for details), which were modified to determine vapor and particle phase concentrations for each constituent to normalized air concentrations and deposition rates for each watershed and sector. The result was a total constituent load to the soil with the exception of vapor deposition contributions. Vapor deposition was calculated by using facility-specific vapor phase emission rates, normalized air concentration of vapors, and a dry vapor deposition rate. The constituent-specific deposition rates were calculated by combining the constituent-specific emission rates obtained from EER with the ISCST3 air model output (i.e., unitized deposition rate).

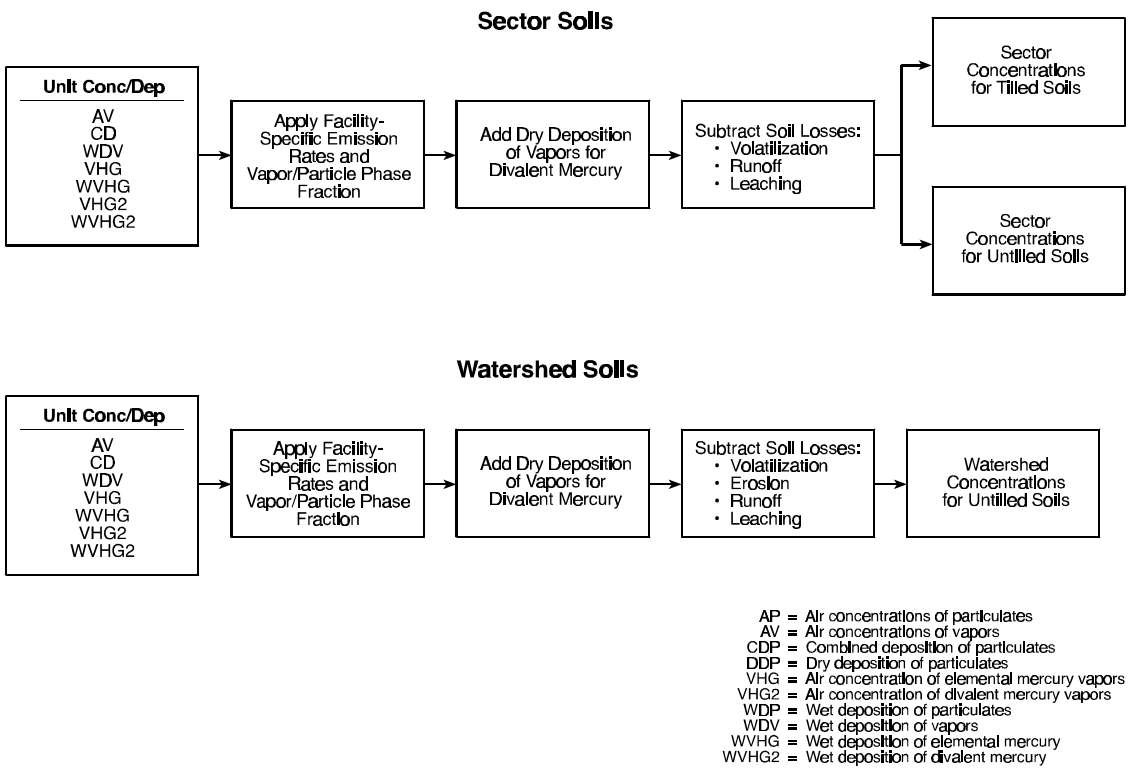


Figure 5-6. Steps involved in calculating sector and watershed soil constituent concentrations.

The resulting vapor deposition rates were added to the other deposition values to determine the total soil load. Because dry deposition of vapor phase materials was evaluated external to the air dispersion model, the plume was not depleted, and, therefore, mass balance was not maintained. The effect of this was to overestimate the deposition to the ground; however, the magnitude of this overestimation is not known. Mass balance was maintained for other forms of deposition (i.e., wet deposition and particle phase dry deposition). Soil concentrations were determined after accounting for soil losses due to volatilization, erosion (watershed only), runoff, and leaching. Soil erosion was treated differently for watershed soils and sector soils. For sector soils, it was assumed that there was no net loss of soil (i.e., no erosion). However, net erosion loss was assumed for watersheds because soil erosion is a major source of sediment and constituent loadings to surface waters. Sector soil concentrations were determined separately for tilled and untilled soils. Watershed soil concentrations were determined for untilled soils only.

5.3.2.1 General Soil Calculations. As described above, soil constituent concentrations result from the summation of particle-bound and vapor phase deposition of contaminants onto the soil, less soil losses due to volatilization, leaching, surface runoff, and erosion.

5.3.2.1.1 Loading to Site Soils. Existence of, and additions to, constituent loading in site soils results from atmospheric deposition. Equation 5-5 (also Table C.1-1 in Appendix C) contains the factors used to calculate atmospheric deposition loads.

Factors used to calculate atmospheric deposition loads include the following air modeling outputs:

- Cyv, air concentration of vapors (used with a deposition velocity to estimate dry vapor deposition rates)
- Dywv, wet deposition rates of vapors
- Dydp, dry deposition rates of particles
- Dywp, wet deposition rates of particles.

For particle-bound contaminants, the wet and dry deposition from the atmosphere follows the physical processes of washout and gravitational settling and/or diffusion/impaction, respectively. These processes were estimated using the ISCST3 air dispersion and deposition model. Vapor phase wet deposition to soil was also calculated as washout within the ISCST3 model. Because the dry deposition of vapors is more highly dependent upon the chemical type and the properties of the surface type encountered by the vapor, for this analysis the estimation of dry vapor deposition to soils was calculated outside the ISCST3 model. Section 5.1.2.3 has a more detailed discussion of dry vapor deposition for both dioxins and mercury. A dry deposition velocity based on empirical data (Koester and Hites, 1992) was applied to the air concentration of vapors to arrive at chemical-specific dry deposition rates for vapors (see Section 5.1).

Atmospheric Deposition to Soils

$$Ds = \frac{100 \quad Q}{z \quad BD} \left[F_v (0.31536 \quad Vdv \quad Cyv + Dywv) + (Dydp + Dywp) \quad (1 - F_v) \right] \qquad (5\text{-}5)$$

Parameter	Definition	Values
Ds	Deposition term (mg/kg-yr)	
100	Units conversion factor ([mg-m^2]/[kg-cm^2])	
Q	Stack emissions (g/s)	Calculated
z	Soil mixing depth, tilled and untilled (cm)	20 (tilled) or 1 (untilled)
BD	Soil bulk density (g/cm^3)	1.5
F_v	Fraction of air concentration in vapor phase (dimensionless)	Chemical-specific
0.31536	Units conversion factor (m-g-s/cm-µg-yr)	
Vdv	Dry deposition velocity (cm/s)	Chemical-specific
Cyv	Normalized vapor phase air concentration ([µg/m^3]/[g/s])	Modeled
Dywv	Normalized yearly wet deposition from vapor phase ([g/m^2-yr]/[g/s])	Modeled
Dydp	Normalized yearly dry deposition from particle phase ([g/m^2-yr]/[g/s])	Modeled
Dywp	Normalized yearly wet deposition from particle phase ([g/m^2-yr]/[g/s])	Modeled

The factor F_v, the fraction in the vapor phase, apportions the air concentration and deposition of the chemical (Q) between the vapor and particle-bound phases. The mixing depth in the soil (z) depends on whether the soil is disturbed by agricultural tilling. In this analysis, 20 cm was used as the mixing depth for tilled soils and 1 cm was used for untilled soils (U.S. EPA, 1993a). The soil bulk density (BD) converts a deposition rate from grams of contaminant per unit area to grams of contaminant per kilogram of soil.

5.3.2.1.2 Losses from Site Soils. Constituent losses from the soil were calculated using Equation 5-6 (also presented in Tables C.1-2 and C.3-2 in Appendix C). The total loss term (ks) is the summation of the following loss processes:

- ksl, leaching of the chemical into the groundwater due to precipitation
- kse, erosion of the chemical laterally along with the soil due to wind and water
- ksr, runoff of the dissolved chemical with the lateral flow of water
- ksg, degradation of the chemical in situ
- ksv, volatilization losses of the chemical.

Leaching (ksl). The loss constant due to leaching was used to adjust for contaminant losses due to leaching from soil. The leaching loss constant ksl is calculated as shown in Table C.3-3 in Appendix C. It is a chemical-specific value that is a function of site-specific precipitation, irrigation, runoff, and evapotranspiration. Site-specific meteorological factors (precipitation, irrigation, runoff, and evapotranspiration) were taken from several sources. Precipitation values were derived from over 40 years of annual average data from the International Station Climate Summaries (on CD ROM). Study areas were assumed to have precipitation equal to that of the closest meteorological stations(s). Irrigation was assumed to equal zero for all sites. Runoff data were taken from the *Water Atlas* (Geraghty et al., 1973); because values in the *Water Atlas* refer to runoff from both groundwater and surface water, they were halved to yield the values used. Evapotranspiration was calculated as 70 percent of site-specific average annual precipitation.

Erosion (kse). The loss constant due to erosion was assumed to be zero for sector soils (used in terrestrial exposure pathways). This is because the small size of the exposure areas would result in nearly equal chemical erosion onto the area of exposure as that off the exposure area. For watershed soils there was a net loss of soil due to erosion. Thus, for the watershed soil concentrations, the loss due to erosion was considered in the analysis.

The IEM model used for this risk analysis uses the Universal Soil Loss Equation to estimate soil erosion losses (X_e) from watersheds that drain into modeled waterbodies surrounding each hazardous waste combustor site. USLE is an empirically derived equation originally developed by the Soil Conservation Service of the U.S. Department of Agriculture to estimate soil erosion losses from agricultural fields during soil conservation planning. In the IEM methodology, USLE was applied in the context of the Gross Erosion Sediment-Delivery Ratio method outlined in USDA (1978) and described in greater detail in the SCS *National Engineering Handbook* (USDA, 1971). Gross erosion is defined as the summation of erosion from all sources within a watershed, as estimated for sheet and rill erosion by USLE. The sediment delivery ratio adjusts gross erosion rates to account for eroded soil that does not reach the waterbody in question. USLE requires inputs to estimate soil erosion losses; including rainfall and runoff factor (R), soil erodibility factor (K), topographic factor (LS), cover and management factor (C), and supporting practice factor (P). In this context, USDA (1978) suggested the use of watershed-averaged values for K, LS, C, and P to simplify computational and data collection requirements. With the exception of K, this approach was adopted for developing site-specific USLE gross erosion loss estimates for the combustor sites. Each of these inputs is discussed in Section 4.3.2.4.

Runoff (ksr). The loss constant due to surface runoff (used in Equation 5-6) was calculated as a function of chemical-specific values and site-specific average annual runoff, as shown in Table C.3-4 in Appendix C. As discussed previously, site-specific values of runoff were taken from the *Water Atlas* (Geraghty et al., 1973).

Degradation (ksg). Degradation refers to chemical or biological degradation processes, not to those physical processes separately accounted for (i.e., leaching, runoff, erosion, and volatilization). Degradation is not the same as half-life in soil since the latter includes losses due to all processes. In this analysis, the degradation losses were assumed to be zero for all chemicals due to limited availability of data on the rate constants for degradation and the site-

Soil Loss Constant

$$ks = ksl + kse + ksr + ksg + ksv \quad (5\text{-}6)$$

Parameter	Definition	Values
ks	Soil loss constant due to all processes (yr^{-1})	
ksl	Loss constant due to leaching (yr^{-1})	Calculated (see Table C.3-3)
kse	Loss constant due to soil erosion (yr^{-1})	0 for terrestrial paths; calculated for watershed soils (see Table C.3-6)
ksr	Loss constant due to surface runoff (yr^{-1})	Calculated (see Table C.3-4)
ksg	Loss constant due to degradation (yr^{-1})	0
ksv	Loss constant due to volatilization (yr^{-1})	Calculated (see Table C.3-5)

specificity of most rate constants. For organic compounds, the uncertainty associated with not considering degradation could result in an overestimation of the soil concentration of a specific chemical. Although the soil concentration of constituents could be lower, the possibility remains that a chemical could degrade into a more toxic form. However, this analysis included dioxins and furans as the only organics; for dioxins and furans sorbed to soil, the predominant environmental fate is burial in-place, resuspension back into the air, or erosion of soil to waterbodies (U.S. EPA, 1994a). Congener-specific data are lacking for basic chemical properties such as degradation rates; however, available data indicate very slow rates of degradation and only photolysis as a possible degradation mechanism (U.S. EPA, 1994a). This process would not impact residues below the surface.

Although the chemical loss due to degradation was assumed to equal zero for most chemicals, mercury transformation between species in the soil was modeled implicitly. Mercury modeling is discussed in Section 5.3.2.2.

***Volatilization (ksv)*.** The loss constant due to volatilization calculates the contaminant loss due to volatilization from soil. The calculation of the volatilization loss factor ksv is shown in Table C.3-5 in Appendix C. Volatilization kinetics are driven by specified chemical properties and environmental conditions, including site-specific properties. Chemical properties that influence volatilization include Henry's law constant, the soil-water partitioning coefficient, and the diffusivity in air. Volatilization is further influenced by environmental conditions including

soil mixing depth, soil bulk density, ambient air temperature, average annual windspeed, and impacted surface area. In the HWC analysis, site-specific values were used for the ambient air temperature and the average annual windspeed.

5.3.2.1.3 Calculation of Site Soil Concentrations. Following calculation of the chemical deposition rates and the rate loss constants, soil concentrations can be estimated. Equations 5-7 and 5-8 (also presented in Tables C.1-1 and C.3-1 in Appendix C) were used to estimate soil concentrations using the chemical deposition and losses derived from Equations 5-5 and 5-6, respectively.

Soil constituent concentration changes with each year of operation of a facility. During the 30-year period in which an HWC facility is assumed to operate, soil concentrations build up. To account for this phenomenon, the equation to calculate average soil concentration over the time period of deposition (facility operation) explicitly considers the time period of exposure for the receptor of interest. This is accomplished by integrating the instantaneous soil concentration equation over the time period of exposure.

Soil Concentration Due to Deposition with Soil Losses

For Carcinogens

$$Sc_1 = \frac{Ds}{ks\ (Tc - T_1)} \left[\left(Tc + \frac{\exp(-ks\ Tc)}{ks} \right) - \left(T_1 + \frac{\exp(-ks\ T_1)}{ks} \right) \right] for\ 0 < T_1 < Tc \quad (5\text{-}7)$$

For Noncarcinogens

$$Sc_{Tc} = \frac{Ds\ (1 - \exp(-ks\ Tc))}{ks} \quad (5\text{-}8)$$

Sc_1	Average soil concentration over exposure duration (mg/kg)	
Ds	Deposition term (mg/kg-yr)	
Tc	Time period over which deposition occurs (yr)	30
Sc_{Tc}	Soil concentration at time Tc (mg/kg)	
ks	Soil loss constant (yr^{-1})	Calculated (see Equation 5-6)
T_1	Related to the exposure duration as follows: $T_1 = T_C$ - ED where ED = exposure duration	Scenario-specific

Cancer Risks. For evaluating cancer risks from chemicals that are carcinogens, soil concentrations were explicitly averaged over the exposure period for the specific human receptor (Equation 5-7). This was accomplished using three time measures: T_c (the time period over which deposition occurs or the operation period of the facility), T_1 (time at the beginning of exposure period for a given receptor), and T_2 (time at the end of exposure period for a given receptor).

Values of T_c, ED, T_1, and T_2 are presented in Table 5-2 for the four age groups considered in the HWC analysis. Since soil concentrations are used to evaluate both terrestrial and aquatic food chain pathways, the exposure duration influences all dietary pathways.

Noncancer Effects. To evaluate noncancer effects from chemicals (inclusive of both carcinogens and noncarcinogens), soil concentrations were used for the time corresponding to the end of the exposure period, i.e., year 30. This is because less-than-lifetime average exposures are of interest for evaluating the potential for noncancer effects, such as developmental toxicity. Many chemicals will have reached steady-state well before year 30. However, other chemicals (such as dioxin and mercury) will tend to build up in soils for much longer periods. Limiting the soil concentrations to year 30 represents a balance between the period of facility operation (which is uncertain and could extend over a much longer period) and a shorter time period (which could underestimate exposure from soil). It is important to note that the deposition loads to soils represent annual averages averaged over a period of 5 years, as discussed in Section 5.1, and not the maximum year deposition value. Therefore, the estimated soil concentrations implicitly included some averaging over time. Ecotoxicological risks also were evaluated based on the year 30 soil concentrations (see Section 9.0).

5.3.2.2 <u>Mercury Soil Calculation: Sector Soils</u>. Modeling fate and transport of mercury through soils generally and substantially follows the methods described in the peer-reviewed 1996 *Mercury Study Report to Congress* (U.S. EPA, 1996). The peer review conducted

Table 5-2. Time Periods Used To Calculate Soil Concentrations

Receptor	T_c (years)	ED (years)	T_1 (years)	T_2 (years)
Adult farmer	30	17.3	12.7	30
Adult nonfarmer	30	13.5	16.5	30
Child 12-19 years	30	9.1	20.9	30
Child 6-11 years	30	8.9	21.1	30
Child 0-5 years	30	6.5	23.5	30

ED = Exposure duration.
T_c = Time period over which deposition occurs.
T_1 = Time at beginning of exposure period.
T_2 = Time at end of exposure period.

on the 1996 MRTC indicated that quantified mercury risk estimates associated with specific emission levels were highly uncertain and difficult to determine. Several significant deviations from the methodology were made in the HWC risk assessment. As a result of the modification made to the methodology, the quantified mercury risk analysis prepared for this rule was not externally peer reviewed. The 1997 *Mercury Study Report to Congress* (U.S. EPA, 1997) was used to update parameter values where new information was available and appropriate. These values are cited and referenced in Appendix D of this report. In a few situations, the 1997 methodology was used to modify the 1996 methodology. In the following text, these situations are clearly identified and referenced to the 1997 report.

As discussed in Section 5.1, two mercury species are released from the stack of HWC units: elemental and divalent. Atmospheric deposition causes mercury in the atmosphere to enter the soil environment. Deposition modeling for this analysis results in deposition of the divalent mercury species via wet and dry deposition of its particle phase fraction and via dry and wet deposition of its vapor phase fraction. Wet deposition of elemental vapor was modeled to deposit on soils; however, dry vapor deposition for elemental mercury was assumed to be zero.

Once in the soil environment, the loads of individual mercury species were totaled and the equilibrium concentrations of mercury species in the soil were then determined. The equations used to model mercury concentrations in soil are not the same as discussed above (i.e., Equations 5-7 and 5-8) because they incorporate mercury speciation. Mercury-specific soil concentration due to deposition onto watershed soil is shown in Equation 5-9 (also C.3-28 in Appendix C).

The annual total load of mercury to watershed soils, L_w, was calculated as the sum across the mercury species of the total mercury deposition to the watershed (as modeled) and the diffusion flux of divalent and elemental mercury to site soils.

The soil concentration due to deposition was depleted by losses. Potential losses include degradation, volatilization, leaching, runoff, and erosion. These loss mechanisms are represented by the overall soil loss constant (ks), as calculated in Equation 5-10.

In addition to these loss mechanisms, mercury in the environment was presumed to undergo transformation. This transformation represents a departure from the 1996 draft *Mercury Study Report to Congress*, which does not incorporate transformation explicitly. For this analysis, divalent mercury in soil was assumed to be transformed to elemental mercury via chemical reduction. Elemental mercury was assumed to be at steady state so that the rate of reduction of divalent mercury is equal to the rate of volatilization of elemental mercury.

Leaching (ksl), Erosion (kse), and Runoff (ksr). Each of these loss constants was calculated in a manner similar to, and using the same types of chemical-specific and site-specific data as, the loss constants discussed in Section 5.3.2.1.2. In the case of mercury, the loss constants due to leaching , erosion, and runoff were calculated for each species, as presented in Tables C.3-31, C.3-34, and C.3-32 in Appendix C, respectively. Variability in the rate loss constants between the three mercury species was introduced as a result of differing soil-water partition coefficients.

Soil Concentration Due to Deposition onto Watershed Soil

$$S_c = \frac{L_w}{ks \quad Z \quad BD} (1 - e^{ks\,Tc})\,100 + C_{sb} \qquad (5\text{-}9)$$

$$L_w = \sum_i (D_{TDWi} + D_{WVWi} + L_{ISi} + L_{DIFi})$$

$$Sc_i = Sc \quad f_{si}$$

Parameter	Definition	Values
S_c	Average soil concentration of total mercury in watershed soil (µg/g)	
L_w	Load of total mercury to watershed soil on an areal basis (g/m^2-yr)	
ks	Soil loss constant for total mercury (yr^{-1})	Calculated (see Equation 5-10)
Z	Mixing depth of soil (cm)	1 cm tilled; 20 cm untilled
BD	Bulk density of soil (g/cm^3)	1.5
T_c	Time period of combustion	30
C_{sb}	Background "natural" soil concentration (µg/g)	0
D_{TDWi}	Total (wet and dry) deposition of particles to the watershed soil for divalent mercury (g/m^2-yr)	Modeled
D_{WVWi}	Wet deposition of divalent and elemental mercury to watershed soil (g/m^2-yr)	Modeled
L_{ISi}	Internal transformation load of mercury species (g/m^2-yr)	0
L_{DIFi}	Diffusion flux of divalent and elemental mercury to watershed soil (g/m^2-yr)	Calculated (see Table C.3-29)
Sc_i	Soil concentration of mercury species "i" in watershed soil (µg/g)	
f_{si}	Equilibrium fraction of mercury species "i" in watershed soil	Elemental & divalent = 0.98 Methyl = 0.02

Soil Loss Constant

$$ks = \sum ks_i \; fs_i \qquad (5\text{-}10)$$

$$ks_i = ksl_i + kse_i + ksr_i + ksg_i + ksv_i$$

Parameter	Definition	Values
ks	Soil loss constant for total mercury (yr^{-1})	
ks_i	Soil loss constant due to all processes for mercury species "i" (yr^{-1})	
fs_i	Equilibrium fraction of mercury species "i" in watershed soils	Elemental & divalent = 0.98 Methyl = 0.02
ksl_i	Loss constant due to leaching for mercury species "i" (yr^{-1})	Calculated (see Table C.3-31)
kse_i	Loss constant due to soil erosion for mercury species "i" (yr^{-1})	Calculated (see Table C.3-34)
ksr_i	Loss constant due to surface runoff for mercury species "i" (yr^{-1})	Calculated (see Table C.3-32)
ksg_i	Loss constant due to degradation for mercury species "i" (yr^{-1})	0
ksv_i	Loss constant due to volatilization for mercury species "i" (yr^{-1})	Calculated (see Table C.3-33)

Degradation (ksg). Although the degradation term is chemical-specific, degradation losses were assumed to equal zero for all mercury species. Intra-species transformation (reduction of divalent mercury to elemental mercury) is treated separately, as described in Section 5.3.3.2.

Volatilization (ksv). Volatilization losses were calculated for all species (see Table C.3-5 in Appendix C). Elemental mercury in untilled soils (1 cm depth) was subject to the most extreme volatilization losses. The equation for the loss constant due to volatilization is presented in Table C.3-33 in Appendix C. The calculation is performed using the same types of data as described in Section 5.3.2.1 for other constituents; however, for mercury it is performed for each species. Rate loss constants differ between the species as a result of the chemical-specific values input for Henry's law constant, diffusivity in air, and soil-water partitioning coefficient used to calculate ksv.

Loss mechanisms are species specific due to the differential species-specific partitioning that occurs. Following the calculation of the rate loss constants (degradation, volatilization, leaching, runoff, and erosion) for each mercury species, the rate loss constants were summed to produce a species-specific soil loss constant due to all processes. This resulted in three values of ks for mercury, one for each species. The three ks values were weighted and summed to produce a single soil loss constant for total mercury. The constants were combined using the speciation ratio (fs_i) as shown in Equation 5-10 to weight the contributions from each species. The speciation ratio represents the equilibrium fractions of the three mercury species in the watershed soils.

The equilibrium state for the elemental and divalent species can be represented by the following equation:

$$Hg^{II} \quad ks_r = Hg^0 \quad ks_v \qquad (5\text{-}11)$$

where

Hg^{II}	=	concentration of divalent mercury in soil
ks_r	=	Hg^{II} to Hg^0 reduction constant
Hg^0	=	concentration of elemental mercury in soil
ks_v	=	loss constant due to volatilization.

This presumes that, in an equilibrium state, the divalent mercury that reduces to elemental mercury then volatilizes. When this relationship was used in the model, the amount of elemental mercury in a particular period of time was $Hg^0 = (Hg^{II})(k_r)/(kv)$.

> The mercury speciation in soil assumes a steady state equilibrium with 2 percent of methylmercury and variable percentages of elemental and divalent mercury totaling to 98 percent. Elemental mercury is assumed to be at steady state so that the rate of reduction of divalent mercury is equal to the rate of volatilization of elemental mercury.

Methylmercury was assumed to equal 2 percent of the total mercury concentration ([mHg] = 0.02 * [Hg]), with the remaining 98 percent comprised of elemental and divalent mercury. The exact proportion of the latter two species was determined using Equation 5-11 and the assumption that the sum of the elemental and divalent mercury species was equal to 98 percent of the total mercury concentration ($[Hg^0] + [Hg^{II}] = 0.98[Hg]$). Because divalent mercury compounds form complexes with soil organic matter, the majority of total soil mercury can be considered largely divalent mercury complexes, although a small fraction of mercury in typical soil will be elemental mercury (U.S. EPA, 1997). The speciation ratio fs_i was calculated as the equilibrium concentration of species “i” divided by the total mercury concentration.

The total mercury soil concentration due to deposition was calculated based on Equation 5-9 and was used to evaluate noncancer effects as discussed in Section 5.3.2.1.

5.3.2.3 <u>Mercury Soil Calculation: Watershed Soils</u>. Mercury concentrations in watershed soils are used to determine corresponding mercury concentrations in waterbodies. The 1997 MRTC used the IEM-2M model, which is composed of two integrated modules that simulate mercury fate using mass balance equations describing watershed soils and waterbodies. Due to the integrated nature of the soil and water modules in IEM-2M, and as a result of the application of IEM-2M for aquatic pathway modeling (see Section 5.3.3.2 and Appendix F), mercury soil concentrations in watershed soils were determined separately from sector soil concentrations for the evaluation of the aquatic pathway. However, watershed soil concentrations used to evaluate the drinking water pathway were determined in the same manner as sector soil concentrations (see Section 5.3.2.2). The IEM-2M methodology is described in detail in the 1997 MRTC. Application of the model is presented in Lyon et al. (1998).

5.3.3 Water Concentrations

The modified IEM-2 model was used to model surface water concentrations of all contaminants except mercury. In the case of mercury, IEM-2M was used. Figure 5-7 illustrates the method used to calculate values for average concentrations for each waterbody (also see Equation 5-12). As with the calculation for air concentrations and deposition rates and watershed soil concentrations, the calculation of average concentrations for each waterbody begins with applying facility-specific emission rates (see Section 4.2 for details) to normalized air concentrations (which are modified to determine vapor and particle phase concentrations for each constituent) and deposition rates for each waterbody and its associated watershed. The result is total constituent load to the waterbody. Five pathways cause constituent loading of the waterbody: (1) direct air deposition, (2) runoff from impervious surfaces within the watershed, (3) runoff from pervious surfaces within the watershed, (4) soil erosion from the watershed, and (5) direct diffusion of vapor phase contaminant into the surface water.

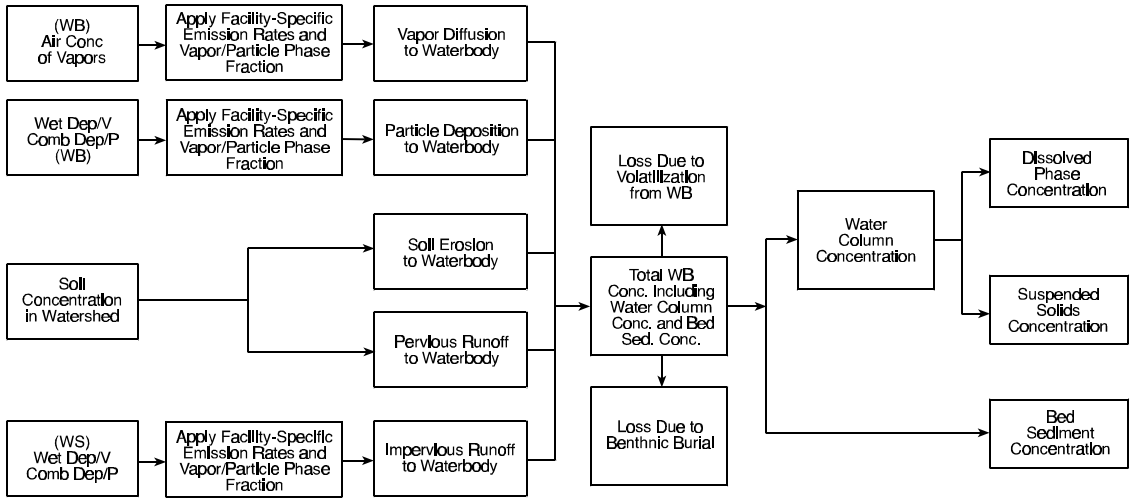


Figure 5-7. Steps involved in calculating waterbody constituent concentrations.

Total Waterbody Concentration

$$C_{wtot} = \frac{L_T}{Vf_x \cdot f_{water} \cdot \left[\frac{d_w + d_b}{d_w}\right] + k_{wt} \cdot WA_w \cdot (d_w + d_b)} \qquad (5\text{-}12)$$

Parameter	Definition	Values
C_{wtot}	Total waterbody concentration, including water column and bed sediment (mg/L)	
L_T	Total chemical load into waterbody (g/yr)	Calculated
Vf_x	Average volumetric flow rate through waterbody (m^3/yr)	Site-specific
f_{water}	Fraction of total waterbody contaminant concentration that occurs in the water column (unitless)	Calculated (see Equation 5-13)
k_{wt}	Overall total waterbody dissipation rate constant (unitless)	Calculated
WA_w	Waterbody surface area (m^2)	Site-specific
d_w	Depth of water column (m)	Site-specific
d_b	Depth of upper benthic layer (m)	0.03

5.3.3.1 General Waterbody Calculations. For all chemicals except mercury, general waterbody calculations were performed using the modified IEM-2 model. The algorithms for the modified IEM-2 are described in this section. The total waterbody concentration was calculated as shown in Equation 5-12. The total waterbody concentration was the sum of the contaminant concentrations in the water column (sorbed to TSS and dissolved phase) and in the benthic layer (sorbed to sediments and dissolved in pore water). Equation 5-12 represents mass balance within the waterbody.

> The calculation of the total chemical concentration in the waterbody is presented in Equation 5-12 for all chemicals except mercury; because mercury speciation is incorporated into the mercury modeling, the calculation is significantly more complex, as discussed in Section 5.3.3.2 and as derived in Appendix F. Equation 5-12 represents the modified IEM-2 model, whereas mercury modeling was completed using IEM-2M.

The fraction of the total waterbody contaminant concentration that occurs in the water column (f_{water}) was calculated as shown in Equation 5-13. The fraction of the total waterbody contaminant concentration that occurs in the water column plus the total waterbody contaminant concentration that occurs in the benthic layer equals 1 (equals the total contaminant concentration in the waterbody). The f_{water} term is dependent upon site-specific TSS

Fraction of Contaminant in Water Column

$$f_{water} = \frac{(1 + Kd_{sw} \cdot TSS \cdot 10^{-6}) \cdot d_w/d_z}{(1 + Kd_{sw} \cdot TSS \cdot 10^{-6}) \cdot d_w/d_z + (\theta_{bs} + Kd_{bs} \cdot BS) \cdot d_b/d_z} \qquad (5\text{-}13)$$

$$f_{benth} = 1 - f_{water}$$

$$Kd_{bs} = OC_{sed} \quad K_{oc}$$

$$Kd_{sw} = OC_{ss} \quad K_{oc}$$

Parameter	Definition	Values
f_{water}	Fraction of total waterbody contaminant concentration that occurs in the water column (unitless)	
Kd_{sw}	Suspended sediment/surface water partition coefficient (L/kg)	Metals - Appendix D Dioxins - calculated
TSS	Total suspended solids (mg/L)	Site-specific
10^{-6}	Conversion factor (kg/mg)	
d_w	Depth of the water column (m)	Site-specific
d_z	Total waterbody depth (m)	Calculated (d_w+d_b)
θ_{bs}	Bed sediment porosity (L_{water}/L)	0.6
Kd_{bs}	Bed sediment/sediment pore water partition coefficient (L/kg)	Metals - Appendix D Dioxins - calculated
BS	Bed sediment concentration (g/cm^3)	1.0
d_b	Depth of the upper benthic layer (m)	0.03
f_{benth}	Fraction of total waterbody contaminant concentration that occurs in the benthic sediment (unitless)	
K_{oc}	Organic carbon portion coefficient (ml/g)	Chemical-specific (see Appendix D)
OC_{sed}	Fraction of organic carbon in bed sediment	0.014
OC_{ss}	Fraction of organic carbon in suspended sediment	0.075

concentration and water column depth and chemical-specific properties including solids-water partitioning coefficients.

Water column concentrations are intrinsically dependent upon TSS concentration, which was included in the surface water model as a parameter. Default regional TSS values were developed as presented in Section 4.3.2.5. Contaminants are removed from the water column via "burial" in the surficial bed sediment layer. The burial rate constant is a function of sediment deposition from the water column to the bed; it accounts for the fact that a significant amount of soils eroded into a waterbody become bottom, rather than suspended, sediment. The suspended solids deposition rate (W_{dep}) reflects benthic burial losses that are innately related to TSS in a waterbody and are based on mass balance considerations.

Either TSS or W_{dep} can be set to default values; the other parameter is then calculated. Default TSS values were determined for waterbodies in hydrologic regions because site-specific TSS estimates could not be calculated reliably with existing data. Calculated TSS values demonstrated a consistently low bias from highest and lowest annual medians from a 30-year period. Given the apparent low bias of the modeled TSS results and the sensitivity of dissolved water concentrations to TSS, use of a simple surface water model to model TSS was deemed inappropriate and TSS values were set to regional defaults.

Suspended solids deposition rates for waterbodies were calculated as follows:

$$W_{dep} = \frac{X_e \quad WA_L \quad SD \quad 1000 - Vf_x \quad TSS}{WA_w \quad TSS} \tag{5-14}$$

where

X_e	=	unit soil loss ($kg/m^2/yr$)
WA_L	=	watershed area (m^2)
SD	=	watershed delivery ratio
Vf_x	=	flow rate (m^3/yr)
Wa_w	=	waterbody area (m^2).

The rate of "burial" was approximated as a function of the deposition rate of sediments from the water column to the surficial sediment layer. Application of this calculated value of W_{dep} preserves mass balance within the model. In the case of a calculated value of W_{dep} that was less than zero (i.e., the mass balance requires resuspension), the benthic burial loss rate was truncated to a value of zero (i.e., no sediments are resuspended). "Buried" sediments were treated as a permanent sink (net depositional loss) for sorbed contaminants. In the case of a waterbody with a high TSS, less sorbed contaminant will be lost through deposition; conversely, in those waterbodies displaying a lower TSS concentration, more sorbed contaminants will be lost given the same sediment load. When the suspended solids deposition loss is truncated to zero, the solids mass balance is violated and uncertainty is introduced by the prohibition of resuspension of sediments to the waterbody. Because sediment deposition losses are handled as a permanent sink, computationally setting them to zero increases the total waterbody concentration

and, therefore, the water column and surficial sediment concentrations relative to what they would be if settling was occurring. The likely cause of negative sediment deposition rates derived from mass balance considerations is either underestimation of the loading of sediments to the waterbody or overestimation of the default TSS level (or both).

Because watersheds were not mapped to their natural extent but instead were truncated at 20 km, it can be assumed that sediment loadings were systematically underestimated in the surface water analysis. This must, necessarily, have resulted in a systematic underestimation of chemical loadings to the waterbodies so affected. While setting the TSS level to an average default value may have improved the water column partitioning in those cases where TSS levels would otherwise appear to have been underpredicted, it cannot account for the loss of contaminant loading to the waterbody. However, this loss is partly offset by zeroing out losses due to sediment deposition (i.e., burial losses) as indicated above. The net effect on the model predictions is unclear. Calculated values for the benthic burial losses are presented by combustor category in Appendix B, Tables B-9 through B-13, for all waterbodies.

The total waterbody concentration is a function of load to the waterbody; as shown in Equation 5-15, total constituent load to the waterbody is the sum of five parameters.

Air deposition load to the waterbody (L_{Dep}) uses the facility-specific emission rate, the vapor/particle partitioning, the waterbody area, and the air modeling output of wet deposition of vapors and combined (wet and dry) deposition of particles.

The diffusion load from the air to the waterbody (L_{Dif}) is also a function of the facility-specific emission rate, the vapor/particle partitioning, the waterbody area, and the air modeling output (e.g., air concentration of vapors). Also influencing the amount of vapor transfer are the temperature, the Henry's law constant, and the liquid and gas phase transfer coefficients.

Total Waterbody Load

$$L_T = L_{Dep} + L_{Dif} + L_{RI} + L_R + L_E \qquad (5\text{-}15)$$

Parameter	Definition	Values
L_T	Total contaminant load to the waterbody (g/yr)	
L_{Dep}	Total (wet and dry) particle phase and wet vapor phase contaminant direct deposition load to waterbody (g/yr)	Calculated
L_{Dif}	Vapor phase contaminant diffusion (dry deposition) load to waterbody (g/yr)	Calculated
L_{RI}	Runoff load from impervious surfaces (g/yr)	Calculated
L_R	Runoff load from pervious surfaces (g/yr)	Calculated
L_E	Soil erosion load (g/yr)	Calculated

The impervious runoff load (L_{RI}) is directly related to the amount of paved or other hard surfaces in the watershed from which runoff is conveyed directly to the waterbody. The pervious runoff load to the waterbody (L_R) is dominated by those chemicals that tend to dissolve in water rather than sorb to soil particles, while the erosion load (L_E) is dominated by sorbed chemicals. Partitioning between runoff and erosion is a function of the runoff amount, the soil characteristics (erodibility and fraction organic carbon), and the properties of the chemical.

Farm pond water concentrations were calculated in a manner similar to that described for waterbodies. Default USLE and TSS parameter values unique to farm ponds were developed (see Table D-38 in Appendix D) and used to model constituent water concentrations for all farm ponds for all facilities. Air concentration and deposition values were based on sector averages. Soil concentrations were based on average sector soil concentrations.

Total waterbody concentration was determined by summing the five loading pathways and subtracting losses due to volatilization and benthic burial. Final water concentrations were then determined for dissolved phase constituent concentration, water column constituent concentrations, and bed sediment constituent concentration. As presented in Equation 5-12 (also Table C.3-15 in Appendix C), the total waterbody concentration (C_{wtot}) was estimated by dispersing the constituent load to the waterbody throughout the entire volume of the waterbody (in the water column and sediments). Chemical dissipation from within the waterbody was also considered, specifically the dissipation due to volatilization and burial in benthic sediment. Volatilization and burial losses affect the total waterbody concentration, which is then apportioned to the water column and bed sediments irrespective of the loss mechanism. Therefore, at equilibrium, both loss mechanisms affect both compartments to the same extent.

As shown in Figure 5-7, the total waterbody contaminant concentration was apportioned between the water column concentration (C_{wt}) as calculated in Equation 5-16 (also Table C.3-23 in Appendix C) and the bed sediment concentration. This apportionment involves the fraction of the contaminant in the water column, as calculated in Equation 5-13.

Total Water Column Concentration

$$C_{wt} = f_{water} \quad C_{wtot} \quad \frac{d_w + d_b}{d_w} \tag{5-16}$$

Parameter	Definition	Values
C_{wt}	Total concentration in water column (mg/L)	
f_{water}	Fraction of total waterbody contaminant concentration that occurs in the water column (unitless)	Calculated (see Equation 5-13)
C_{wtot}	Total waterbody concentration, including water column and bed sediment (mg/L)	Calculated (see Equation 5-12)
d_w	Depth of the water column (m)	Site-specific
d_b	Depth of upper benthic layer (m)	0.03

The two divisions of the total waterbody concentration (into the water column concentration and the bed sediment concentration) were further divided chemically between the fraction that is sorbed to sediments and suspended solids and the fraction that is dissolved. Equation 5-17 (also Table C.3-25 in Appendix C) was used to calculate the chemical concentration sorbed to the bed sediment. This concentration was used to estimate fish tissue concentration for dioxins; metals concentrations in fish tissue were based on dissolved water concentrations (see Equation 5-18). Note that Equation 5-17 is used to calculate only the sorbed portion of the bed sediment concentration and does not include the chemical concentration found in the sediment pore water.

Concentration Sorbed to Bed Sediment

$$C_{sb} = f_{benth} \quad C_{wtot} \quad \frac{Kd_{bs}}{\theta_{bs} + Kd_{bs} \quad BS} \quad \frac{d_w + d_b}{d_b} \qquad (5\text{-}17)$$

$$Kd_{bs} = OC_{sed} \quad K_{oc}$$

Parameter	Definition	Values
C_{sb}	Concentration sorbed to bed sediments (mg/kg)	
f_{benth}	Fraction of total waterbody contaminant concentration that occurs in the bed sediment (unitless)	Calculated ($1 - f_{water}$)
C_{wtot}	Total waterbody concentration, including water column and bed sediment (mg/L)	Calculated (see Equation 5-12)
Kd_{bs}	Bed sediment/sediment pore water partition coefficient (L/kg)	Metals, see Appendix D; Dioxins, calculated as above
d_w	Depth of water column (m)	Site-specific
d_b	Depth of upper benthic layer (m)	0.03
θ_{bs}	Bed sediment porosity (unitless)	0.6
BS	Bed sediment concentration (kg/L)	1.0
OC_{sed}	Fraction of organic carbon in bed sediment	0.04
K_{oc}	Organic carbon partition coefficient (mL/g)	Chemical-specific

Dissolved Water Concentration

$$C_{dw} = \frac{C_{wt}}{1 + Kd_{sw} \cdot TSS \cdot 10^{-6}} \qquad (5\text{-}18)$$

$$Kd_{sw} = OC_{ss} \quad K_{oc}$$

Parameter	Definition	Values
C_{dw}	Dissolved phase water concentration (mg/L)	
C_{wt}	Total concentration in water column (mg/L)	Calculated (see Table C.3-23)
Kd_{sw}	Suspended sediment/surface water partition coefficient (L/kg)	Metals, see Appendix D; Dioxins, calculated as above
TSS	Total suspended solids (mg/L)	Site-specific
OC_{ss}	Fraction of organic carbon in suspended sediment	0.075
K_{oc}	Organic carbon partition coefficient (mL/g)	Chemical-specific (see Appendix D)

The total water column concentration C_{wt} is the sum of the constituent dissolved in the water and the constituent associated with suspended solids. Because drinking water is filtered to remove suspended solids, risks from drinking water ingestion were calculated only from the concentrations of constituents dissolved in the water column for each waterbody identified as a drinking water source. Equation 5-18 calculates the concentration of contaminant dissolved in the water column (C_{dw}) which is used to determine human health risks associated with drinking water. For metals, the dissolved water concentration is also used for determining bioaccumulation in fish because it represents the most bioavailable form.

Concentrations in fish tissue are estimated using compound-specific bioaccumulation factors (BAFs) or biota-sediment bioaccumulation factors (BSAFs). Due to the limited availability of BSAFs, these factors were applied only for dioxins in this analysis. The BSAF was used to calculate dioxin concentrations in fish from the bed sediment concentration, and the BAF was used to calculate metal concentrations in fish from the dissolved water concentration. The BAF is preferred to the bioconcentration factor (BCF) because it accounts for uptake from dissolved water and the food supply. In contrast, the BCF accounts for uptake from dissolved water only. In cases where a BAF was not available, a BCF was substituted. The BAFs, BCFs,

and BSAFs used in this analysis are presented in Appendix D. These calculations are discussed in detail in Section 5.4.1.6.

5.3.3.2 Mercury Waterbody Calculations. The following discussion is a summary of the discussion and results presented in Lyon et al. (1998), unless otherwise referenced. Modeling fate and transport of mercury through waterbodies follows the refined methods contained in the *Mercury Study Report to Congress* (U.S. EPA, 1997) for recreational and subsistence fishers and the ecological receptors. Drinking water concentrations of mercury for the remaining human receptors were modeled using the HWC-modified IEM-2 (see Appendix F for details).

The two models, IEM-2 and IEM-2M, were compared (see Appendix F for further detail on the comparison performed by Lyon et al. (1998)) and it was determined that the IEM-2M model predicts substantially lower mercury concentrations than the modified IEM-2 model. This is because the IEM-2 overestimates both the loading of mercury to the waterbody and the conversion of inorganic mercury to methylmercury. These overestimations occur because IEM-2 is a fully steady-state model that does not properly account for the gradual buildup and methylation of mercury over time. The IEM-2M mercury watershed/waterbody model is an extension of the IEM-2 model, which itself is an extension of the Indirect Exposure Methodology (IEM) developed by EPA (U.S. EPA, 1993a). The IEM-2M model was used in both the 1997 MRTC and the Utility Report to Congress (URTC) to simulate the fate of mercury deposited onto a watershed/waterbody system.

The IEM-2M model is composed of two integrated modules that simulate mercury fate using mass balance equations describing watershed soils and waterbodies. The mass balances are performed for each mercury component, with internal transformation rates linking elemental mercury, divalent mercury, and methylmercury. Sources include wet and dry deposition loadings of each component to watershed soils and to the waterbody. An additional source is diffusion of atmospheric elemental mercury vapor to watershed soils and the waterbody. Sinks include leaching of each component from watershed soils, burial of each component from lake sediments, volatilization of elemental mercury and methylmercury from the soil and water column, and advection of each component out of the waterbody. At the core of IEM-2M are nine differential equations describing the mass balance of each mercury component in the surficial soil layer, in the water column, and in the surficial benthic sediments. The equations are solved for a specified interval of time, and predicted concentrations are output at fixed intervals. For each calculational time step, IEM-2M first performs a terrestrial mass balance to obtain mercury concentrations in watershed soils. IEM-2M next performs an aquatic mass balance driven by direct atmospheric deposition along with runoff and erosion loads from watershed soils. Methylmercury concentrations in fish are derived from dissolved methylmercury water concentrations using bioaccumulation factors.

IEM-2M was developed to include specific kinetic transformation rates (oxidation, reduction, methylation, and demethylation) affecting mercury components in soil, water, and sediments. These transformation rates are driven by specified rate constants. Volatilization kinetics are included as a transfer reaction driven by specified chemical properties and environmental conditions.

While IEM-2M tracks the buildup of soil and water concentrations over the years given a steady depositional load and long-term average hydrological behavior, it does not respond to unsteady loading or meteorological events. Like its predecessors, IEM-2M is a spatially homogeneous model. There are, thus, limitations on the analysis and interpretations imposed by these simplifications. The model's calculations of average waterbody concentrations are less reliable for unsteady heterogeneous environments, such as streams, than for more steady homogeneous environments, such as lakes (see Section 4.4.1 of the 1997 MRTC).

Per the 1997 MRTC, key features and assumptions in the IEM-2M surface waterbody module include the following:

- The partitioning of mercury components between the water column and suspended biotic and abiotic solids and between pore water and sediment particles is in local equilibrium as described by a set of partition coefficients.

- Atmospheric mercury wet and dry deposition loads are handled as a constant average flux.

- Surface runoff mercury loadings are estimated as a function of the dissolved concentration of mercury in the surficial soil water (calculated by the soil module as a function of time) and the specified annual water runoff.

- Soil erosion mercury loadings are calculated as a function of the sorbed concentration of mercury in the surficial soil layer (calculated by the soil module as a function of time), together with the calculated annual soil erosion, a sediment delivery ratio, and an enrichment ratio.

- Diffusive mercury loadings from the atmosphere are calculated as a function of a specified atmospheric vapor concentration, the calculated dissolved water column concentration, and the calculated transfer velocity. The dissolved concentration in a waterbody is driven toward equilibrium with the vapor phase concentration above the waterbody. At equilibrium, gaseous diffusion into the waterbody is matched by volatilization out of the waterbody. This specified air concentration is an output of the atmospheric transport model (ISCST3).

- The rate of contaminant burial in bed sediments is estimated as a function of the rate at which biotic and abiotic solids deposit from the water column onto the surficial sediment layer minus the rate at which they resuspend to the water column. Burial represents a permanent sink of eroded soil and mercury concentrations scavenged from the water column.

- Separate transformation rate constants allow for the calculation of mercury components in the water column and benthic sediments.

Model inputs and intermediates are provided in Appendix H. Media concentrations produced by the IEM-2M modeling were used to generate risk results for recreational fishers and

subsistence fishers, as well as for ecological receptors. The IEM-2M methodology was modified or adapted for use in the HWC risk assessment, as described below.

Modifications to IEM-2M Methodology. Several modifications to IEM-2M were necessary in order to evaluate the impacts of mercury sources on rivers and streams. These modifications were necessary because only shallow lakes were considered in the 1997 MRTC, which used IEM-2M. These additions/modifications are described below.

Volatilization from Rivers/Flowing Waterbodies. In IEM (U.S. EPA, 1993a), for both lakes and flowing waterbodies, volatilization from waterbodies is addressed in the same general manner as a first-order volatilization loss constant (units of yr^{-1}) is calculated (O'Conner 1983, as cited in Lyon et al. (1998)). However, there are differences in the calculation of this term for flowing waterbodies, where the O'Conner and Dobbins (1958), as cited in Lyon et al. (1998) formula is used for the liquid film transfer coefficient, and the gas phase transfer coefficient is assumed constant at 36,500 m/yr (U.S. EPA, 1993a). Implementation of these methods was not necessary for the 1997 MRTC because only lakes were considered. For the HWC risk assessment, the methods used in IEM (U.S. EPA, 1993a) for estimating volatilization from flowing waterbodies were implemented in IEM-2M. Implementing the volatilization algorithms used in IEM for flowing waterbodies introduces uncertainty into the modeling.

Mercury Transformation in Rivers/Flowing Waterbodies. In general, methylation of divalent mercury has been observed to be reduced in aerobic conditions. Since flowing waterbodies will tend to be better aerated than nonflowing waterbodies, it follows that methylation should be lower in flowing waterbodies. However, the proper magnitude of this difference is uncertain. In this analysis, it was assumed that the methylation rates in flowing waterbodies were 10 percent of the values assumed for lakes (in both the water column and surficial sediments), which themselves are uncertain. The precise nature of the dependence of the methylmercury concentration in fish on the methylation rates in the waterbody is not trivial, although clearly it is monotonic. This, therefore, represents a potentially significant uncertainty in the results, with the primary uncertainty being the uncertainty about the precise magnitude of the effect of aeration on the methylation rates and the secondary uncertainty being the uncertainty about the methylation rates for lakes in general.

Modeling of Suspended Solids in Waterbodies. The site-specific nature of the analysis prompted the collection of TSS data for the hydrologic region of each waterbody to address the variability inherent in the sediment dynamics of the individual waterbodies. This effort utilized the STORET database and is described in Section 4.3.2.5.

The hydrologic regions generally included more than one of the waterbodies considered, and it was not deemed appropriate to use the TSS values themselves as inputs in this analysis. Instead, these values were used in conjunction with an input settling velocity v_s to estimate a resuspension velocity. The resuspension velocity was calculated based on the assumption that the volumetric resuspension is equal to the volumetric deposition of solids implied by the TSS data. In particular, an abiotic solids settling velocity of 730 m/yr (U.S. EPA, 1997) was assumed for all waterbodies, and the abiotic solids resuspension velocity v_{rs} was calculated for each waterbody:

$$v_{rs} = v_s \frac{TSS_{data}}{S_b} \tag{5-19}$$

where

v_{rs}	=	abiotic solids resuspension velocity, m/yr
v_s	=	abiotic solids settling velocity, m/yr
TSS_{data}	=	median solids concentration for hydrologic region, g/m^3
S_b	=	benthic solids concentration, g/m^3.

Subsequent calculation of the abiotic solids concentration S_w used the MRTC (U.S. EPA, 1997) method; namely, that S_w is given by

$$S_w = \frac{F_{soil} + v_{rs}A_wS_b}{Vf_x + v_sA_w} \tag{5-20}$$

where

S_w	=	abiotic solids concentration in waterbody, g/m^3
F_{soil}	=	loading of soil from the watershed to the waterbody, g/yr
v_{rs}	=	abiotic solids resuspension velocity, m/yr
A_w	=	waterbody area, m^2
S_b	=	benthic solids concentration, g/m^3
Vf_x	=	dilution flow through waterbody, m^3/yr
v_s	=	abiotic solids settling velocity, m/yr.

Modeling abiotic solids dynamics in waterbodies introduces uncertainty. The abiotic solids dynamics affect the exchange of mercury between the benthic sediment and the water column. This in turn impacts the predicted dissolved methylmercury concentration in the water column, which is used to estimate the methylmercury fish concentration. The main terms used in the modeling of the abiotic solids dynamics are the total suspended solids in the water column, the settling and resuspension rates to and from the benthic sediment, the burial rate in the sediment, and the loading of soil from the watershed.

In this study, waterbody-specific data were not available for these terms. However, TSS data for the hydrologic region of each waterbody were used in the abiotic solids modeling. As explained above, these TSS data were used to estimate the resuspension rate, based on an assumed constant settling velocity. The calculated resuspension rate and input settling velocity were then used with the estimated soil loading from the watershed and the water dilution flow through the waterbody to calculate a waterbody-specific abiotic solids suspended sediment concentration. One can show that the method used to calculate the abiotic solids suspended sediment concentration is equivalent to:

$$S_w = TSS_{data} + \frac{F_{soil} - Vf_x\, TSS_{data}}{Vf_x + v_s A_w} \tag{5-21}$$

where

S_w	=	abiotic solids concentration in waterbody, g/m^3
TSS_{data}	=	median solids concentration for hydrologic region, g/m^3
F_{soil}	=	loading of soil from the watershed to the waterbody, g/yr
Vf_x	=	dilution flow through waterbody, m^3/yr
v_s	=	abiotic solids settling velocity, m/yr
A_w	=	waterbody area, m^2.

In this form, we can see that S_w will be less than TSS_{data} whenever F_{soil} - $Vf_x\, TSS_{data} < 0$, and vice versa. Further, the choice of settling velocity v_s has no impact on whether or not this occurs. One can also show that the burial rate (calculated using Equation 5-22) will almost always be negative if F_{soil} - $Vf_x\, TSS_{data} < 0$[1]. This condition means that either the soil loading to the waterbody is too low, or the dilution flow too large, to sustain the median TSS_{data} estimated for the hydrologic region.

In the results generated by Lyon et al. (1998), this was seen to occur for 123 out of 263 waterbodies analyzed, all of which were rivers or streams. This is not surprising, given that solids loading from upstream was not considered; i.e., the soil loading for rivers may have been underestimated. As discussed below, in these cases the burial rate was set to zero.

Calculation of Burial Rate in Benthic Sediment. The same general formula used in the MRTC was used to calculate the benthic burial rate:

$$v_b = \frac{v_s\, S_w + v_{sbio}\, S_{Bio} - (v_{rs} + v_{\min})\, S_b}{S_b} \tag{5-22}$$

where

v_b	=	benthic burial velocity (m/yr)
v_s	=	settling velocity for abiotic solids (m/yr)
S_w	=	abiotic solids concentration in the water column, g/cm^3
v_{sbio}	=	settling velocity for biotic solids (m/yr)
S_{Bio}	=	biotic solids concentration in the water column, g/cm^3
v_{rs}	=	resuspension velocity of abiotic solids (m/yr)

[1] Depending on the value of the mineralization rate, it may happen that F_{soil} - $Vf_x\, TSS_{data} < 0$, and yet the calculated burial rate is slightly greater than 0. However, this was not seen to occur in the current analysis.

v_{min} = mineralization rate for upper benthic solids (m/year)
S_b = concentration of solids in the benthic sediment, g/cm^3.

In some cases the calculated value of v_b is negative. In these cases, the burial velocity is set to zero (this was not required in the MRTC). At first glance, this may appear to violate mass balance. It also ignores situations where contaminated sediments are actually being mobilized and therefore contributing to both the solids and mercury loadings to the waterbody. The practical effect of resetting v_b to zero in this way is equivalent to assuming only clean sediment is being mobilized. This was deemed appropriate for the purpose of the current study, in which incremental impacts from a source that is assumed to operate for only 30 years were being evaluated. It is unlikely that the facility being modeled would be the source of any mercury contained in the deeper sediments, and it was considered unrealistic to allow the sediments to be mobilized continuously over the 30-year modeling period.

Treatment of Background Environmental Mercury. In the MRTC, estimates of background values of mercury concentrations and deposition rates were made and included in the analysis. However, in the current analysis, no background mercury concentrations or deposition rates are assumed. This approach is motivated by the comparative nature of the analyses. Further, the system of differential equations used in IEM-2M is such that the incremental impact of a facility is independent of whatever background values are assumed, as long as the background values are constant in time.

Input Parameters. A complete list of the parameter values assumed for each waterbody is provided in Appendix F. In this section, we discuss only the input parameters for IEM-2M that are in addition to the input parameters used by IEM-2. These parameters consist of terms used in estimating waterbody sediment dynamics and general chemical transformation between the different mercury species considered. The values assumed are summarized in Tables 5-3 and 5-4. A more detailed discussion is provided in Lyon et al. (1998).

Chemical transformation rate constants must be specified or calculated for the soil, water column, and benthic sediment equations. The transformations include oxidation of Hg^0, reduction of HgII, methylation of HgII, demethylation of MHg to HgII, and *mer* cleavage demethylation of MHg to Hg^0. The values assumed (in day^{-1}) are summarized in Table 5-4.

Mercury transformation in the environment is not a well understood aspect of the mercury cycle and is an area requiring substantial research. Consequently, it is also an area of uncertainty. For the HWC risk assessment, perhaps the greatest uncertainty is the dependence of mercury transformation on particular characteristics of individual waterbodies. Examples of such properties relevant to mercury transformation are: types of bacteria that may accelerate methylation or reduction of divalent mercury (Watras et al., 1995; Mason et al., 1995, as cited in Lyon et al., 1998); dissolved organic carbon, which can reduce the reduction rate of divalent mercury (Amyot et al., 1997, as cited in Lyon et al., 1998); sulfate concentrations, which can increase methylation in the water column (Watras et al., 1995, as cited in Lyon et al., 1998); and sunlight, which can increase demethylation of methylmercury and reduction of divalent mercury within the water column (Gilmour and Henry, 1991, as cited in Lyon et al., 1998). Any such dependencies are not considered in the current analysis, as the same transformation rates were

Table 5-3. Additional Input Parameters Required for IEM-2M

Parameter	Value	Reference/Comment
Abiotic solids deposition velocity, m/yr	730	MRTC
Biotic solids deposition velocity, m/yr	73	MRTC
Biotic solids production rate, g/yr	1,000	Value for mesotrophic waterbodies (value used in MRTC was 100, based on oligotrophic waterbody)
Hg^0 biotic solids partition coefficient (L/kg)	1,000	MRTC
Hg(II) biotic solids partition coefficient (L/kg)	200,000	MRTC
MHg biotic solids partition coefficient (L/kg)	500,000	MRTC
Biotic mortality rate (day^{-1})	0.03	MRTC
Pore water diffusion coefficient (m^2/yr)	0.1575	MRTC
Abiotic solids mineralization rate (m/yr)	0.001	MRTC
Sediment concentration (g/cm^3)	75,000	MRTC
Trophic level 3 fish bioaccumulation factor (L/kg) [a]	1,600,000	MRTC
Trophic level 4 fish bioaccumulation factor (L/kg) [a]	6,800,00	MRTC

[a] This factor is multiplied by the calculated dissolved methylmercury concentration in the water column to estimate the methylmercury concentration in fish.

Table 5-4. Mercury Transformation Rate Constants (unless otherwise noted, values are from 1997 MRTC)

Rate Constants, day^{-1}	Watershed Soil	Water Column		Benthic Sediments	
		Rivers/Creeks	Lakes	Rivers/Creeks	Lakes
Oxidation	0	0	0	0	0
Reduction	0.000055[a]	0.0075	0.0075	0.000001	0.000001
Methylation	0.00005	0.0001[b]	0.001	0.00001[b]	0.0001
Demethylation to Hg^{II}	0.0025	0.015	0.015	0.002	0.002
Mer demethylation to Hg^0	0	0	0	0	0

[a] Calculated using the method described in Appendix B of MRTC (U.S. EPA, 1997), based on a reference reduction rate of 0.0005 L/L_w-day in the surficial 5-mm layer of soil, a soil depth of 2 cm, and a water content of 0.22 L_w/L.

[b] Set to 10 percent of value used for lakes.

used for all lakes and for all rivers/streams. This may over- or underestimate methylmercury, depending on how the different transformation rates are affected by the specific properties of the different waterbodies.

Bioaccumulation Factor. The following discussion of uncertainties inherent in the use of the fish BAF is from Appendix D of Volume 3 of the MRTC (U.S. EPA, 1997). Note that the median of the distribution derived there was used in the current analysis (and in the waterbody modeling in the MRTC).

The BAF distributions were designed to estimate an average concentration of methylmercury in fish of a given trophic level from an average concentration of dissolved methylmercury in the epilimnion for a (single) randomly selected lake in the continental United States. In the overall mercury fate and exposure model, the input (water concentrations) to this distribution represented an annual average, aggregating variability in methylmercury concentrations in the epilimnion over an entire year, and the output (fish concentrations) represented the average methylmercury concentration in the diet of a specific receptor. Available data were inadequate to satisfy these representations fully. In most cases, water methylmercury concentrations incorporated limited or no cross-seasonal variability. Also, fish diets for specific receptors have not been determined. For HWC risk assessment, a generic receptor was assumed and was approximated by including a large range of fish age or size classes whenever possible. Also, because of the general paucity of appropriate data, many studies on lakes in other countries were included in the analysis; biotic and abiotic processes in these lakes were assumed to be similar to lakes in the continental United States. These limitations introduced additional uncertainty in the BAF output that was not quantified in HWC risk assessment.

Except as is discussed in MRTC, there were no distinctions in the BAF distributions as to size of fish, lake trophic status, lake pH, or relative methylmercury concentrations in the water column. The data, however, are heavily biased toward northern oligotrophic lakes and somewhat toward smaller (younger) fish.

Perhaps the greatest source of uncertainty is that of model uncertainty, that is, uncertainty introduced by failure of the model to account for significant real-world processes. The simple linear BAF model relating methylmercury in fish to methylmercury in water masks a number of nonlinear processes leading to the formation of bioavailable methylmercury in the water column and bioaccumulation in the aquatic food chain. Much of the variability in field data applicable to the estimation of mercury BAFs can be attributed to differences between aquatic systems. As an example, in lake surveys conducted within a relatively restricted geographic region, large differences can exist between lakes with respect to mercury concentrations in a given species of fish (Cope et al., 1990; Grieb et al., 1990; Jackson, 1991; Lange et al., 1993, as cited in Lyon et al., 1998). These observations have led to the suggestion that much of this variability is due to differences in within-lake processes that determine the percentage of total mercury that exists as the methylated form. Limited data also indicate that, within a given waterbody, concentrations of methylmercury are likely to vary with depth and season. Unfortunately, while the concentration of methylmercury in fish flesh is presumably a function of these varying concentrations, published BAFs are generally estimated from a small number of measured water values, the representativeness of which is poorly known.

5.4 Calculation of Food Chain Concentrations

This section presents the methodology used to calculate contaminant concentrations for each of the food chain pathways considered in this risk analysis. Pathways considered are

- Aboveground produce (fruits and vegetables)
- Belowground produce
- Milk
- Beef
- Poultry and eggs
- Pork
- Fish.

Direct inhalation and direct soil ingestion are based on human intake of contaminated air and soil. Section 5.3 presents the methodology for calculating air and soil concentrations. Section 6.0 discusses the methods used to estimate human exposure (dose levels) resulting from direct intake of air and soil.

The terrestrial food chain includes aboveground fruits and vegetables, belowground produce, milk, eggs, and beef, poultry, and pork. Aboveground produce may be contaminated by combustion emissions through several mechanisms, including direct deposition of particulate phase contaminants onto the plant, direct uptake of vapor phase contaminants, and root uptake of contaminants deposited on the soil. Soil concentrations are sector averages for tilled soil. Root vegetables may be contaminated via uptake of contaminants through the roots. The contamination of plant matter consumed by livestock differs depending on the type of plant. Forage, which includes pasture grass and hay, and silage may be contaminated by combustion emissions through direct deposition of particulate phase contaminants onto the plant, direct uptake of vapor phase contaminants, and root uptake of contaminants deposited on the soil. Tilled soil concentrations (mixing depth of 20 cm) are used for silage, and untilled soil concentrations (mixing depth of 1 cm) are used for forage. Appendix C contains the equations used to estimate air-to-plant and soil-to-plant transfers, and chemical-specific transfer factors are provided in Appendix D.

Animal tissue (beef, pork, poultry, eggs, and milk) may be contaminated through ingestion of contaminated forage, silage, and soil by livestock. Beef and dairy cattle ingest grain, silage, forage, and soil. Hogs ingest grain, silage, and soil. In all instances, the grain is assumed not to be contaminated. This assumption is made based on the fact that grains are protected from direct deposition. Also, feed grains are typically acquired commercially from the marketplace. The same assumption (that grain is a protected crop and, as such, is not contaminated) was made for subsistence farmers. Chickens raised by subsistence farmers are assumed to consume contaminated soil as 10 percent of their diet. Appendix C contains the equations used to estimate soil or plant to animal transfers; chemical-specific transfer factors are provided in Appendix D.

The aquatic food chain consists of fish consumption. The fish concentration was calculated from the total water column concentration, the dissolved water concentration, or the bed sediment concentration using a bioconcentration factor (BCF), a bioaccumulation factor (BAF), or a sediment bioaccumulation factor (BSAF), as appropriate/available. BCFs include

only uptake from dissolved water concentrations and do not include uptake through the food chain. Therefore, bioaccumulation (BAF) factors (which do include food sources) are much preferred over bioconcentration factors, the latter only being used where data on bioaccumulation factors are not available.

This analysis follows the methodology set out in the *Mercury Study Report to Congress* (U.S. EPA, 1997). Methylmercury concentrations in fish were derived from dissolved water concentrations using bioaccumulation factors. The BAF of methylmercury is defined as the ratio of the methyl-mercury concentration in fish tissue divided by the concentration of methylmercury in the water column.

5.4.1 Food Chain Pathways and Parameters

Individual food chain pathways are presented in this section. Greater detail is provided in Appendixes C and D. Appendix C presents all of the algorithms used to model food chain concentrations. Appendix D presents parameter values used in this analysis.

5.4.1.1 <u>Aboveground Vegetation (Produce, Forage, and Silage)</u>. Indirect exposure due to the human ingestion of aboveground produce (fruits and vegetables) and the animal ingestion of silage and forage depends on the total concentration of contaminants of concern in the leaf and fruit portions of the plant. In this analysis, aboveground produce was classified as protected and unprotected. Protected produce has a protective covering over its edible portion (e.g., citrus fruits); unprotected produce (e.g., an apple) does not have a protective covering.

As shown in Figure 5-8, vegetation can be contaminated by three mechanisms:

- Root uptake: Transfer of contaminants available from the soil to the aboveground portions of the plant through the roots.
- Deposition of particles: Deposition of wet and dry particle-bound contaminants on the leaves and fruits of plants
- Vapor transfer: Transfer of contaminants to the plants through their foliage.

Contamination of unprotected produce was assumed to occur through all three of the above mechanisms. Because the outer covering on protected produce acts as a barrier, contamination of this type of produce through deposition of particles and vapor transfer is assumed to be negligible.

The total contaminant concentration in aboveground produce is calculated as a sum of contamination occurring through all three of these mechanisms. Although it is not the predominant mechanism for plant chemical concentration increase in produce that is exposed to the other mechanisms, root uptake is a mechanism that is present in all types of produce as well as forage and silage. Equation 5-23 presents the relationship between the aboveground produce and forage and silage contamination through root uptake and the soil concentration in which the plant is growing. The root uptake is estimated through the use of an empirically derived plant-soil bioconcentration factor, Br. Br represents the ratio of the contaminant concentration in

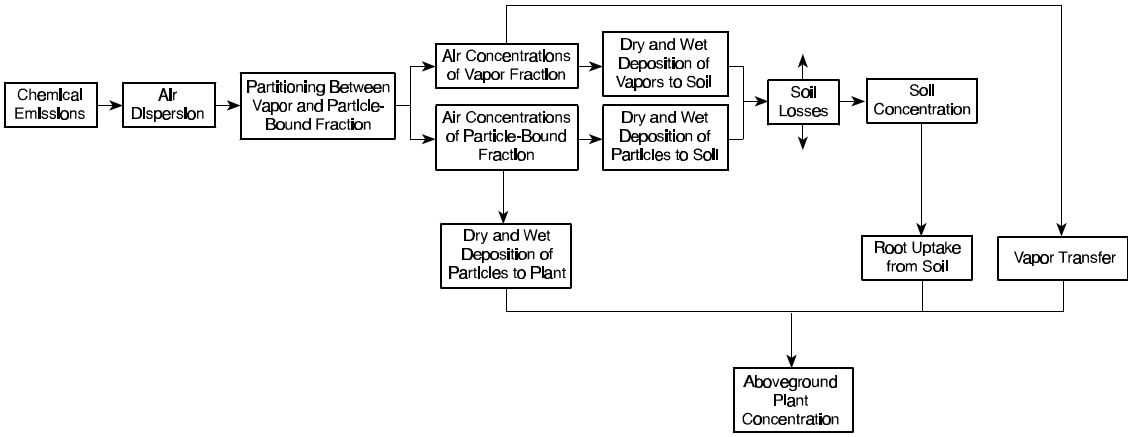


Figure 5-8. Mechanisms used to arrive at aboveground produce concentration.

Aboveground Vegetation Concentration Due to Root Uptake

$$Pr = Sc \ Br \qquad (5\text{-}23)$$

Parameter	Definition	Values
Pr	Concentration of pollutant in the plant due to direct uptake from soil (mg/kg Dw)	
Sc	Average soil concentration over exposure duration (mg/kg)	Calculated (see Table C-1.1)
Br	Plant-soil bioconcentration factor for aboveground vegetation [µg/g DW]/[µg/g soil]	Chemical-specific (see Appendix D)

plants (based on dry weight) to that in the soil. For a discussion of how the soil concentration of contaminant is estimated, see Section 5.3.2.

The Br for dioxins was calculated from an equation in Travis and Arms (1988). This equation for the bioconcentration of these organics is related to the octanol-water partitioning coefficient, as shown in Equation 5-24:

$$\log(Br) = 1.588 - 0.578 \log K_{ow} \qquad (5\text{-}24)$$

where

Br = soil to plant biotransfer factor ([µg/g DW plant]/[µg/g soil])

K_{ow} = octanol water partition coefficient (unitless) - (see Appendix D, Tables D-21 to D-37).

Because the K_{ow}s for dioxins are so high, root uptake is not a significant contaminant pathway (especially in comparison with air deposition).

The Br values for metals were taken from several sources, including Baes et al. (1984), U.S. EPA (1992), and U.S. EPA (1997). Baes et al. (1984) determined average Br_i values for numerous elements of the periodic table, based on experimentally derived literature values or otherwise estimated. Br values for the following metals were taken from Baes et al. (1984):

- Antimony
- Barium
- Beryllium
- Chromium (III and VI)
- Cobalt
- Copper
- Manganese
- Lead
- Silver
- Thallium.

Br values for the three mercury species were taken from U.S. EPA (1997). Values of Br for the following metals were taken from U.S. EPA (1992):

- Arsenic
- Cadmium
- Nickel
- Selenium.

Although Br may be strongly influenced by chemical and physical soil properties (i.e., pH and organic matter content) as well as the plant species, a default chemical-specific Br was applied to all HWC sites instead of a site-specific Br. Consequently, there is considerable

uncertainty associated with use of default estimates for Br. It is important to point out that root uptake is not a significant contaminant pathway for metals in comparison with air deposition.

Equation 5-25 calculates the contaminant concentration in aboveground vegetation due to wet and dry deposition of contaminant on the plant surface. The vegetative concentration due to direct deposition is estimated similarly to the soil concentration due to atmospheric deposition. The emission rate of the chemical (Q) and the fraction in the vapor (f_v) and particle phases are used together with the sector-averaged air modeling output to estimate the atmospheric deposition to the plant surface. Dry deposition of particles is considered directly; however, an adjustment is made to the wet deposition rate of particles to account for the portion of the particles washed off the plant during the precipitation event. For most chemicals, the fraction of wet particle deposition that adheres to the plant is 0.6. The other factors that influence the contaminant concentration in the vegetation are related to the length of time and amount of exposure of the edible portion of the plant to the deposition process. Wet deposition is not considered for vapors because of the high level of uncertainties in the process (e.g., revolatilization). Vapor uptake, an analog to dry vapor deposition, is discussed below.

Equation 5-26 calculates the contaminant concentration in aboveground vegetation, forage, and silage due to direct uptake of vapor phase contaminants into the plant leaves. The methodology used to estimate contamination through vapor transfer incorporates a semi-empirical correction factor to account for the reduction of lipophilic contaminant (i.e., dioxin) concentrations resulting from mechanisms responsible for inhibiting the transfer of the contaminant (i.e., the shape of the produce) and the removal of the contaminants from the edible portion of the produce (e.g., washing, peeling, and cooking).

Bv, the air to plant biotransfer factor, is a chemical-specific value likely to be important for leafy plants and exposed produce, but not protected produce or root crops. With the exception of mercury, which can exist as a vapor, Bv values for metals are set equal to 0 since it is assumed they do not exist in the vapor phase. Bv values for dioxins were taken from Lorber and Rice (1995). Atmospheric deposition is the primary mechanism by which dioxins enter the terrestrial food chain (U.S. EPA, 1994a); therefore, the air-to-plant transfer of dioxins drives the food chain transport of dioxins. A default chemical-specific Bv was used for all types of aboveground vegetation. Application of a chemical-specific default air-to-plant biotransfer factor introduces uncertainty because it varies with the type of vegetation. Also, use of the Bv factor assumes equilibrium partitioning between the plant and the atmosphere, a condition that does not occur under natural conditions. This assumption may, therefore, overstate the magnitude of the vapor transfer.

The algorithm used to estimate contamination through vapor transfer was developed to estimate the transfer of contaminants into leafy vegetation rather than into bulky aboveground vegetation, such as apples. Because of the shape of bulky produce, transfer of contaminant to the interior of the produce is unlikely to occur and, as a result, the inner portions will be largely unimpacted. Additionally, typical removal mechanisms, such as washing, peeling, and cooking, will further reduce residues. Therefore, applying this algorithm to bulk produce would result in overestimating contaminant concentrations. An adjustment factor (VG_{ag}) has been incorporated into the algorithm to address this overestimation for lipophilic compounds (i.e., compounds with a log K_{ow} value greater than 4). In this analysis, VG_{ag} was assigned a value of 0.01 for dioxins

Vegetative Concentration Due to Direct Particle Deposition

$$Pd = \frac{1000 \cdot Q \cdot (1 - F_v) \cdot [Dydp + (Fw \cdot Dywp)] \cdot Rp \cdot [(1.0 - exp(-kp \cdot Tp)]}{Yp \cdot kp} \quad (5\text{-}25)$$

Parameter	Definition	Values
Pd	Concentration in plant due to direct deposition (mg/kg DW)	
1000	Units conversion factor (mg/g)	
Q	Stack emissions (g/s)	Calculated
F_v	Fraction of air concentration in vapor phase (dimensionless)	Chemical-specific (see Appendix D)
Dydp	Normalized yearly dry deposition from particle phase (s/m^2-yr)	Modeled
Fw	Fraction of wet deposition that adheres to plant (dimensionless)	Chemical-specific (see Appendix D)
Dywp	Normalized yearly wet deposition from particle phase (s/m^2-yr)	Modeled
Rp	Interception fraction of edible portion of plant (dimensionless)	Varies (see Appendix D)
kp	Plant surface loss coefficient (yr^{-1})	18
Tp	Length of plant exposure to deposition of edible portion of plant, per harvest (yr)	Varies (see Appendix D)
Yp	Yield or standing crop biomass of the edible portion of the plant (kg DW/m^2)	Varies (see Appendix D)

for all aboveground fruits and vegetables intended for human consumption (U.S. EPA, 1994a). This is because many of the most commonly consumed "unprotected" aboveground fruits and vegetables (e.g., tomatoes, peppers, cucumbers, snap beans, apples, pears, strawberries) are bulky, not leafy.

As discussed in the descriptions of the animal ingestion pathways, these same algorithms were applied to forage and silage crops used for animal feed. The compound-specific transfer factors for soil and vapor to aboveground produce are provided in Appendix D.

5.4.1.2 <u>Belowground Produce</u>. As shown in Figure 5-9, the contaminant concentrations in belowground vegetables were estimated from the contaminant concentration in the soil in which they were cultivated.

Vegetative Concentration Due to Air-to-Plant Vapor Transfer

$$Pv = Q \cdot F_v \cdot \frac{Cyv \cdot Bv \cdot VG_{ag}}{\rho_a} \qquad (5\text{-}26)$$

Parameter	Definition	Values
Pv	Concentration of pollutant in the plant due to air-to-plant transfer (mg/kg Dw)	
Q	Stack emissions (g/s)	Calculated
F_v	Fraction of air concentration in vapor phase (dimensionless)	Chemical-specific (see Appendix D)
Cyv	Normalized vapor phase air concentration (µg-s/g-m^3)	Modeled
Bv	Air-to-plant biotransfer factor ([mg pollutant/kg plant tissue DW]/[µg pollutant/g air])	Chemical-specific (see Appendix D)
VG_{ag}	Empirical correction factor for aboveground produce (dimensionless)	Varies according to produce and chemical (see Appendix D)
ρ_a	Density of air (g/m^3)	1.2 x 10^3

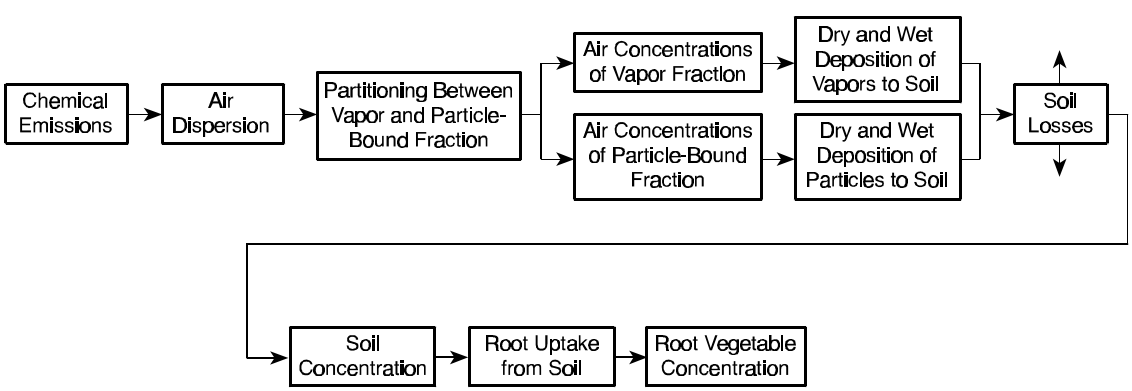


Figure 5-9. Mechanisms used to arrive at belowground produce concentration.

Equation 5-27 presents the method for estimating the belowground vegetable concentration due to root uptake. The soil-to-root vegetable transfer factors varied for each constituent; Appendix D contains the constituent-specific transfer factors.

The methodology used to estimate contamination through root uptake takes into consideration the reduction of lipophilic contaminants (i.e., dioxins) resulting from mechanisms responsible for inhibiting the transfer of the contaminant (i.e., the shape of the produce) and the removal of the contaminants from the edible portion of the produce (e.g., washing, peeling, and cooking). Specifically, the algorithm used to estimate contamination through root uptake was developed to estimate the transfer of contaminants into barley roots rather than into bulky root vegetation, such as carrots. Because of the shape of bulky produce, transfer of the contaminant to the interior of the produce is unlikely to occur and the inner portions will be largely unimpacted. Additionally, typical removal mechanisms, such as washing, peeling, and cooking, further reduce residues. Therefore, applying this algorithm to bulk produce would likely overestimate contaminant concentrations. An adjustment factor (VG_{bg}) has been incorporated into the algorithm to address this overestimation for lipophilic compounds (i.e., compounds with a log K_{ow} value greater than 4). In this analysis, VG_{bg} was assigned a value of 0.01 for dioxins for all belowground vegetables intended for human consumption (U.S. EPA, 1994a).

Root Vegetable Concentration Due to Root Uptake

$$Pr_{bg} = \frac{Sc \quad RCF \quad VG_{bg}}{Kd_s} \quad (Dioxins) \qquad (5\text{-}27)$$

$$Pr_{bg} = Sc \quad Br \quad (Metals)$$

Parameter	Definition	Values
Pr_{bg}	Concentration of pollutant in belowground plant parts due to root uptake (for dioxins (mg/kg Fw); for metals (mg/kg Dw))	
Sc	Average soil concentration over exposure duration (mg/kg)	Calculated (see Table C.1-1)
RCF	Ratio of concentration in roots to concentration in soil pore water ([mg pollutant/kg plant tissue FW] / [µg pollutant/mL pore water])	Chemical-specific (see Appendix D)
VG_{bg}	Empirical correction factor for root vegetables (unitless)	0.01
Kd_s	Soil-water partition coefficient (mL/g)	Calculated (see Table C-1.3)
Br	Plant-soil bioconcentration factor for root vegetables [µg/g Dw] / [µg/g soil]	Chemical-specific (see Appendix D)

5.4.1.3 <u>Beef and Milk</u>. The contaminant concentrations in beef tissue and milk were estimated based on the amount of contaminant that the cattle were assumed to have consumed through their diet. The cattle's diet was assumed to consist of forage (i.e., pasture grass and hay) and silage. Additional contamination of the cattle occurred through the ingestion of soil. In this analysis, it was assumed that each contaminated item consumed originated from the site.

Equation 5-28 calculates the concentration of contaminant in an animal from ingestion of forage, silage, and soil. The amount of grain (assumed to be uncontaminated), silage, forage, and soil consumed was assumed to vary between dairy and beef cattle. Consumption of these items was also assumed to vary between cattle raised by subsistence and those raised by commercial farmers. The diet of the subsistence beef cattle comprises mainly pasture grasses, hay, and silage. Soil consumption is relatively high, resulting from the time at pasture. The diet of commercial beef cattle is supplemented with an increased amount of grain because these cattle are fattened on grain at feed lots prior to slaughter. This also limits the commercial beef cattle's exposure to contaminated soil. Total consumption rates for commercial beef cattle are lower because they are slaughtered younger and lighter. Unlike beef cattle, the subsistence and commercial dairy cattle were assumed to be the same weight. However, dairy cattle raised by commercial farmers were assumed to be fed a high grain diet. As a result, the diet of these dairy cattle from consumption of pasture grass was limited. The limited grazing for the commercial dairy cattle also limited their exposure to contaminated soil (Rice, 1994). Table 5-5 presents the varying consumption rates for the different types of cattle.

Equation 5-28 is used to calculate the concentration of contaminant in milk and beef from animal ingestion of forage, silage, and soil. The animal concentration is dependent on the biotransfer factor, Ba, the ratio of the contaminant concentration in animal tissue to the daily intake of contaminant by the animal. The Ba is used to calculate contaminant concentration in animal tissues as a result of consumption of contaminated vegetation; it is both chemical- and animal-tissue-specific.

The Ba_{milk} for dioxin was taken from Lorber and Rice (1995). The Ba_{beef} for dioxin congeners was calculated from the Ba_{milk} as the ratio of percent beef fat (19 percent) to percent milk fat (3.5 percent). These values are from the dioxin document as cited in Lorber and Rice (1995). Therefore, the biotransfer factor for beef is 5.4 times higher (19/3.5) than for milk.

Although this assumption was used for the purposes of the HWC analysis, a strong argument can be made that it is not appropriate to ratio the dioxin Ba_{milk} values by the fat contents of beef and milk to derive dioxin Ba values for beef. The biotransfer factor represents the ratio of the contaminant concentration in the animal tissue to the contaminant concentration in the feed normalized by the overall feed consumption rate. Because of this dependence on the feed consumption rate, the biotransfer factors will be different for different animals whether or not the factors are adjusted for fat content. This fact was not accounted for in the analysis.

Therefore, because beef cattle have lower feed consumption rates than dairy cattle (by a factor of 20.3/8.6 = 2.4), it is likely that beef tissue concentrations have been underestimated by a similar factor. An alternative approach would be to use a simple bioconcentration factor that implicitly accounts for different feeding, fat production, and carryover rates in different animals. The argument can be made that, on a fat basis, the BCF values should be similar in different

Animal Concentration Due to Plant and Soil Ingestion

$$A = \left(F_i \, Qp_i \quad P_i + Qs \quad Sc \right) \quad Ba \qquad (5\text{-}28)$$

$$P_i = Pd_i + Pv_i + Pr_i$$

Parameter	Definition	Values
A	Concentration of pollutant in beef, milk, or pork (mg/kg Fw)[a]	
F_i	Fraction of plant grown on contaminated soil and eaten by the animal (dimensionless) for each plant type.	1
Qp_i	Quantity of plant matter eaten by the animal each day (kg plant tissue DW/d) for each plant type	Varies for each plant type and between subsistence and commercial farmers (see Appendix D)
Qs	Quantity of soil eaten by the animal (kg soil/d)	Varies between subsistence and commercial farmers (see Appendix D)
Sc	Average soil concentration (mg/kg)	Calculated (see Table C.1-1)
Ba	Biotransfer factor for beef, milk, or pork (d/kg)	Chemical-specific (see Appendix D)
Pd, Pv, Pr	Total concentration of pollutant in the each plant type eaten by the animal (mg/kg Dw)	Calculated (see Tables C.2-1, C.2-2, C.2-3)

[a] For the chemicals selenium, cadmium, divalent mercury, and methylmercury, the concentration in beef is in mg/kg Dw.

animals and should depend mainly on the effective "carryover rate" (which would reflect the effect of lactation and other factors on the disposition of dioxins) rather than the animal's feeding habits. The same argument holds for the use of Ba_{beef} values for pork, although the effect is further magnified by the lower feeding rates for hogs (by a factor of 8.6/4.3 x 20.3/8.6 = 4.7 overall).

Ba factors for milk and beef for metals were from Baes et al. (1984), Lorber and Rice (1995), or U.S. EPA (1997).

Table 5-5. Consumption Rates for Commercial and Subsistence Beef and Dairy Cattle

Commodity	Commercial Beef Cattle	Subsistence Beef Cattle	Commercial Dairy Cattle	Subsistence Dairy Cattle
Forage (kg DW/d)	3.8	8.8	6.2	13.2
Silage (kg DW/d)	1	2.5	1.9	4.1
Grain (kg DW/d)	3.8	0.47	12.2	3
Total in Feed	8.6	11.8	20.3	20.3
Soil (kg/d)	0.25	0.5	0.2	0.4

The total contaminant concentration in the feed items (i.e., forage and silage) is calculated as a sum of contamination occurring through root uptake, particle deposition, and vapor transfer. Contamination of forage and silage, unprotected vegetation, was assumed to occur through all three of the above mechanisms.

The methodology used to estimate contamination through vapor transfer takes into consideration the reduction of lipophilic contaminant (i.e., dioxin) concentrations resulting from mechanisms responsible for inhibiting the transfer of the contaminant. Specifically, the algorithm used to estimate contamination through vapor transfer was developed to estimate the transfer of contaminants into leafy vegetation rather than into bulky aboveground vegetation, such as silage. Transfer of contaminant to parts of the interior of silage is likely to be limited. Therefore, applying this algorithm to bulk silage would result in overestimating contaminant concentrations. An adjustment factor (VG_{ag}) has been incorporated into the algorithm to address this overestimation for lipophilic compounds (i.e., compounds with a log K_{ow} value greater than 4). In this analysis, VG_{ag} was assigned a value of 0.5 for dioxins for all silage (U.S. EPA, 1994a).

5.4.1.4 <u>Poultry and Eggs</u>. The poultry and egg ingestion pathways were considered only for exposures to dioxins and furans. The chickens considered in the subsistence poultry farm scenario were assumed to be free-range animals exposed to combustion emissions through the incidental ingestion of soil with their diet. Ten percent of their ingestion rate was assumed to be contaminated soil. Ten percent was selected for use in the analysis to be consistent with the study from which the biotransfer factors were obtained. The grain that the subsistence farmers' free-range chickens consumed was assumed to be free of contamination. The soil concentrations were estimated using the soil equations described in Section 5.3.2. The poultry and egg concentrations of contaminant are estimated through empirical bioconcentration factors taken from Stephens et al. (1992). For those isomers lacking BCFs in Stephens et al., the BCF value was assumed to be equal to the most structurally similar isomer listed in the reference. This creates a degree of uncertainty that cannot be determined due to a lack of data. Equation 5-29 calculates the concentration in poultry or eggs due to ingestion of contaminated soil by the chickens raised by the subsistence farmer.

Concentration in Poultry and Eggs due to Soil Uptake by Free-Range Chickens - Subsistence Farmer

$$A = Sc \quad Fd \quad BCF \tag{5-29}$$

Parameter	Definition	Values
A	Concentration of pollutant in poultry or eggs (mg/kg Fw)	
Sc	Average soil concentration over exposure duration (mg/kg)	Calculated (see Table C.1-1)
Fd	Fraction of diet that is soil (dimensionless)	0.1
BCF	Bioconcentration factor for congener in poultry or eggs (unitless)	Chemical-specific (see Appendix D)

5.4.1.5 Pork. The hogs in this analysis were assumed to be animals raised in outdoor feedlots. Grain is the primary component of the dietary intake of hogs; however, grain consumption was assumed not to be a contaminant pathway for hogs. Therefore, the contaminated diet of hogs was assumed to consist of silage and associated soil. Table 5-6 presents the consumption rates for hogs.

Equation 5-28 calculates the concentration of contaminant in an animal (in this case, pork) from ingestion of silage and soil. Forage ingestion was not used because hogs are not grazing animals. Because the silage and soil were assumed to have been obtained from the site under evaluation, the fraction contaminant assigned to each was assumed to be 1. The silage contaminant concentrations were estimated using the aboveground vegetation algorithm

Table 5-6. Consumption Rates for Hogs

Commodity	Hogs
Forage (kg DW/d)	0.0
Silage (kg DW/d)	1.3
Grain (kg DW/d)	3.0
Total in Feed	4.3
Soil (kg/d)	0.37

(Equation 5-23) presented in Section 5.4.1.1. Through the use of this algorithm, the total contaminant concentration in the aboveground vegetation was calculated as a sum of contamination occurring through root uptake, deposition of particles, and vapor transfer.

The methodology used to estimate contamination through vapor transfer takes into consideration the reduction of lipophilic contaminant (i.e., dioxin) concentrations resulting from mechanisms responsible for inhibiting the transfer of the contaminant. Specifically, the algorithm used to estimate contamination through vapor transfer was developed to estimate the transfer of contaminants into leafy vegetation rather than into bulky aboveground vegetation, such as silage. Transfer of contaminant to parts of the interior of silage is likely to be limited. Therefore, applying this algorithm to bulk silage would result in overestimating contaminant concentrations. An adjustment factor (VG_{ag}) has been incorporated into the algorithm to address this overestimation for lipophilic compounds (i.e., compounds with a log K_{ow} value greater than 4). In this analysis, VG_{ag} was assigned a value of 0.5 for dioxins for all silage (U.S. EPA, 1994a).

Biotransfer factors for pork were readily available only for cadmium, mercury, and selenium. Due to a lack of reported biotransfer factors for pork for dioxins and most metals, beef biotransfer factors were applied in the absence of chemical-specific data (i.e., $Ba_{pork} = Ba_{beef}$). The Ba_{beef} was derived from the Ba_{milk} (see Section 5.4.1.3 and the discussion of the inherent uncertainty therein), and the Ba_{pork} was assumed to be equal to the Ba_{beef}. The uncertainty associated with estimating the pork biotransfer factor from the beef biotransfer factor cannot be determined due to a lack of data on the pork biotransfer factor. Consequently, there is considerable uncertainty associated with the use of default estimates for Ba. The equations for the pork ingestion pathway are provided in Appendix C.2-7.

5.4.1.6 <u>Fish</u>. Fish were assumed to be exposed to combustion emissions through the water column and bed sediment in the waterbodies near combustors. The contaminants in the water column consist of dissolved constituents and constituents associated with suspended solids. For metals, the dissolved fraction is more significant and is the most bioavailable form. The equations used to estimate surface water concentrations are presented in Section 5.3.3. The results of these equations are used to estimate the concentration of contaminants in fish; the concentrations in fish tissue are estimated using compound-specific bioaccumulation factors or biota-sediment bioaccumulation factors (BSAFs). Due to the limited availability of BSAFs, these factors were applied only for dioxins in this analysis. The BAFs and BSAFs used in this analysis are presented in Appendix D. Equation 5-30 presents the method for estimating tissue concentrations of metals in freshwater fish.

BAF values for metals used in Equation 5-30 were taken from the literature:

- Stephan (1993): antimony, arsenic, chromium III, chromium VI, copper, lead, nickel, and silver

- Barrows et al. (1980): beryllium

Fish Concentration from Dissolved Water Concentration

$$C_{fish} = C_{dw} \cdot BAF \qquad (5\text{-}30)$$

Parameter	Definition	Values
C_{fish}	Fish concentration (mg/kg)	
C_{dw}	Dissolved water concentration (mg/L)	Calculated (see Equation 5-18)
BAF	Bioaccumulation factor (L/kg)	Chemical-specific (see Appendix D)

- Kumada et al. (1972): cadmium
- Lemly (1985): selenium.

Mercury concentrations in fish were based on the IEM-2M model used in the 1997 MRTC, modified to include rivers and streams. Methylmercury concentrations in fish are derived from dissolved methylmercury water concentrations using bioaccumulation factors. The BAF for mercury is defined in Appendix D of Volume III of the MRTC (U.S. EPA, 1997) as the concentration of the methylmercury in fish divided by the concentration of total dissolved methylmercury in water. The MRTC has BAFs for mercury for trophic level 3 and 4 fish. These values were used, in conjunction with the IEM-2M model in the MRTC to project fish tissue concentrations. Although the mercury BAFs presented in the MRTC are the best available, they still possess a substantial degree of uncertainty. The following discussion is from Section D.3.9, Discussion of Uncertainty and Variability in the BAF, in Appendix D of Volume III of the MRTC (U.S. EPA, 1997).

BAF distributions were designed to estimate an average methylmercury concentration in fish of a given trophic level from an average concentration of dissolved methylmercury in a waterbody. In the overall mercury fate and exposure model, the input (water concentrations) to this distribution represented an annual average that aggregates variability in methylmercury concentrations in the waterbody over an entire year and the output (fish concentrations) represented the average methylmercury concentration in the diet of a specific receptor.

Available data were inadequate to satisfy these representations fully. In most cases, water methylmercury concentrations incorporated limited or no cross-seasonal variability. Also, fish diets for specific receptors have not been determined. For the MRTC, analysis of a generic receptor was assumed and was approximated by including a large range of fish age or size classes whenever possible. Also, due to the general meagerness of appropriate data, many studies on lakes in other countries were included in the MRTC analysis under the assumption that biotic and abiotic processes in these lakes were similar to lakes in the continental United States. These

limitations introduced additional uncertainty in the BAF output that was not quantified in the MRTC analysis. Further discussions and details can be found in Appendix D of Volume III.

The transfer of dioxin-like compounds from fish to piscivorous organisms is key to determining the potential for adverse effects to organisms foraging in an aquatic environment. To characterize the accumulation of dioxin into fish species, two metrics were considered that quantify the uptake of dioxin and furan congeners from environmental media into fish tissues: BAFs and BSAFs. By definition, a BAF is a measure of chemical accumulation in fish tissue from both direct uptake from the water column and uptake from contaminated prey. A BSAF is a similar measure of uptake, but it is calculated based on concentrations of the constituent in sediment rather than the water column. A BSAF assumes equilibrium between sediment, pore water, and the water column. When partitioning of constituents between sediments, particles, pore water, and surface water are accounted for, good correlation between BSAFs and surface-water-derived BAFs are noted (U.S. EPA, 1993a).

In the freshwater ecosystem, TCDD can bioaccumulate in fish even though concentrations of TCDD in the water column are below detection. Hence, calculating BAFs based on surface water concentrations introduces greater uncertainty. Given these limitations (the accuracy of TCDD measurement and BAF estimation), use of surface water concentrations may misrepresent actual bioaccumulation. However, extremely hydrophobic constituents, such as dioxin congeners, can be measured more easily in sediments and aquatic life because these dioxin and furan congeners tend to partition into organic carbon (OC) in the sediment and into fish lipids once taken into the organism. For these reasons, biological uptake factors that reflect the relationship between sediment concentrations and organism concentrations, such as a BSAF, may be more appropriate to characterize food chain transfer of these constituents. Consequently, the BSAF is the preferred metric for estimating accumulation for dioxin congeners. BSAFs in [mg congener/kg LP]/[mg congener/kg sediment OC] were calculated from measured data as described below.

The State of Connecticut Department of Environmental Protection (CT DEP) performed an analysis of dioxin and furan concentrations in environmental media associated with the freshwater and terrestrial ecosystems. A statistical analysis of the data conducted by the Midwest Research Institute (Bauer, 1992) reports measurements of dioxin and furan congeners in waterbodies in the vicinity of five resource recovery facilities (RRFs). Samples were collected over a 4-year period between sites that were grouped as control, preoperational, and operational. Residues of polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) were measured in fish tissue, sediment, and soil. The results were assessed by the CT DEP to determine whether RRF emissions would influence PCDD and PCDF levels in environments surrounding facilities. Although the issue of equilibrium between sediment and surface water and dioxin/furan congeners is in question in the CT DEP study due to the continued loading of congeners to the waterbodies from facilities operating during the study, it is not highly relevant. The study design for the CT DEP data is similar to the way in which the data are being used here, i.e., to assess fish exposures to dioxins in the vicinity of operating hazardous waste incinerators. Furthermore, the data were used in lieu of the data from the Great Lakes because those data are now recognized as being unduly influenced by past contamination and, therefore, are not appropriate for the purpose of this analysis.

BSAFs were determined using a hierarchy for data identification and selection. When available, site-specific data (i.e., fish and sediment concentrations from the same site) were used preferentially. Although these data were available for only three congeners, they were deemed the best data available. For the remaining congeners, either the operational data or the pre-operational and operational data were used. In general terms, the BSAF was calculated as shown in Equation 5-31.

The three methods used to select and apply data to determine congener concentrations in lipids and in OC sediment are discussed below. Following a sample calculation of the BSAF for TCDD in Table 5-7, calculated BSAF values are provided in Table 5-8; these values were applied as shown in Equation 5-32.

Site-Specific Data. Site-specific data from five sites in the CT DEP study (Bristol, Hartford, Sterling, Windham, and Wallingford) were used for three congeners (2,3,7,8-TCDD, 2,3,7,8-TCDF, and 2,3,4,7,8-PeCDF). The congener concentrations in the whole fish were adjusted to represent the concentration in the fish lipid. The congener concentrations in fish lipid at each site then were weighted by the number of fish sampled at the site. Finally, the mean of the weighted fish lipid concentrations was calculated and divided by the total number of fish samples from the five sites. The end result was a mean fish lipid concentration weighted across sites for the three congeners. The sediment data from the sites were treated in the same manner (adjusted by site-specific organic carbon) to derive a weighted mean sediment concentration. The adjustment involved dividing the whole sediment concentrations by the sediment fraction of

Biota-Sediment Accumulation Factor

$$BSAF = \frac{\dfrac{C_{fish}}{f_{lipid}}}{\dfrac{C_{sb}}{OC_{sediment}}} \qquad (5\text{-}31)$$

Parameter	Definition	Values
BSAF	Biota-sediment accumulation factor [(mg congener/kg lipid)]/[mg congener/kg OC sediment)]	
C_{fish}	Congener concentration in fish tissue (mg congener/kg fish)	Calculated (see text)
f_{lipid}	Fish lipid content (unitless)	Bauer, 1992
C_{sb}	Concentration of contaminant sorbed to bed sediment (mg/kg)	Bauer, 1992
OC Sediment	Fraction organic carbon in bottom sediment (unitless)	Bauer, 1992

Table 5-7. Calculation of BSAF for 2,3,7,8-TCDD Using Site-Specific Data

	Bristol	Hartford	Sterling	Windham	Wallingford
TCDD concentration in fish tissue (mg/kg)	0.26	2.4	0.11	0.25	1.4
Lipid fraction	0.038	0.071	0.053	0.044	0.071
TCDD concentration in fish lipid (mg/kg lipid)	6.88	33.9	2.13	5.74	19.2
Number of samples	140.0	159	40.00	59	75
TCDD concentration in fish lipid (mg/kg lipid) number of samples	964	5,392	85.0	339	1,444
Weighted mean TCDD concentration					17.4
TCDD concentration in sediment	1.7	2.0	0.90	0.97	0.54
Fraction organic carbon	0.19	0.056	0.067	0.129	0.019
TCDD concentration in sediment normalized for OC	8.8	34.9	13.5	7.5	28.7
Number of samples	59.0	90	20	20	40
TCDD concentration in sediment normalized for OC number of samples	519	3,143	270	150	1,146
Weighted mean TCDD concentrations in OC-corrected sediment					22.8
$BSAF_{TCDD}$ = 17.4 / 22.8 = 0.76					

organic carbon for the site; the resulting organic carbon-corrected dioxin/furan concentrations were multiplied by the total number of sediment samples at that site. Finally, the mean of the organic carbon-corrected concentrations was calculated and divided by the total number of sediment samples from the five sites. The end result was a mean sediment concentration weighted across sites for the three congeners.

The BSAFs were calculated as the ratio of congener concentration in the fish on a lipid basis to the congener concentration in the sediment normalized for organic carbon. As an example of method calculations, the data used and the BSAF calculated are shown for 2,3,7,8-TCDD in Table 5-7. Although this calculation involves the use of site-specific data, the same principle is applied when using the operational data only or the preoperational and operational data as discussed in the following text.

Table 5-8. Biota-Sediment Accumulation Factors Values Derived from the State of Connecticut DEP Report (Bauer, 1992)

Congener	BSAF	Comments
TCDF, 2,3,7,8-	0.226	Calculated using site-specific data
TCDD, 2,3,7,8-	0.762	Calculated using site-specific data
PeCDF, 1,2,3,7,8-	0.255	Calculated using preoperational and operational facility data
PeCDF, 2,3,4,7,8-	0.389	Calculated using site-specific data
PeCDD, 1,2,3,7,8-	0.567	Calculated using preoperational and operational facility data
HxCDF, 1,2,3,4,7,8-	0.056	Calculated using operational facility data
HxCDF, 1,2,3,6,7,8-	0.093	Calculated using preoperational and operational facility data
HxCDF, 2,3,4,6,7,8-	0.175	Calculated using preoperational and operational facility data
HxCDF, 1,2,3,7,8,9-	0.152	Calculated using operational facility data
HxCDD, 1,2,3,4,7,8-	0.155	Calculated using preoperational and operational facility data
HxCDD, 1,2,3,6,7,8-	0.172	Calculated using preoperational and operational facility data
HxCDD, 1,2,3,7,8,9-	0.045	Calculated using preoperational and operational facility data
HpCDF,1,2,3,4,6,7,8-	0.011	Calculated using preoperational and operational facility data
HpCDF,1,2,3,4,7,8,9-	0.027	Calculated using preoperational and operational facility data
HpCDD, 1,2,3,4,6,7,8,-	0.033	Calculated using preoperational and operational facility data
OCDF, 1,2,3,4,6,7,8,9-	0.003	Calculated using preoperational and operational facility data
OCDD, 1,2,3,4,5,7,8,9-	0.034	Calculated using preoperational and operational facility data

***Operational Facility Data*.** For two congeners (1,2,3,4,7,8-HxCDF and 1,2,3,7,8,9-HxCDF), preoperational data were considered suspect because fish concentrations were higher than for the postoperational data. Therefore, for two congeners, only the operational data were selected based on the fact that dioxins and furans are exceptionally persistent, and usually these compounds are not expected to show drastic reductions from preoperational to operational conditions. For these two congeners, the mean fish concentrations were significantly higher in the preoperational conditions than in the operational conditions. Because there is no definitive explanation for this occurrence, the preoperational data were not included in the development of the BSAFs for these two congeners, and only the mean fish tissue concentrations from the operational conditions were used. However, natural variability in fish concentrations associated with sampling different species of fish, age, and foraging behavior could account for the seemingly counterintuitive outcome. For the rest of the congeners, data from the preoperational and operational conditions were combined as a weighted mean.

Dioxin/Furan Concentrations in Fish Tissue (via Sediment Pathway)

$$C_{fish} = \frac{C_{sb} \cdot f_{lipid} \cdot BSAF}{OC_{sed}} \tag{5-32}$$

Parameter	Definition	Values
C_{fish}	Fish concentration (mg/kg)	
C_{sb}	Concentration of contaminant sorbed to bed sediment (mg/kg)	Calculated (see Table C-3.25)
f_{lipid}	Fish lipid content (fraction)	0.03
BSAF	Biota to sediment accumulation factor (kg OC/kg lipid)	Chemical-specific (see Table 5-8)
$OC_{sediment}$	Fraction organic carbon in bottom sediment (unitless)	0.014

Preoperational and Operational Facility Data. The following discussion applies to the remaining 12 dioxin and furan congers. Because there was no intuitive discongruity in the preoperational and operational facility data, they were combined. The preoperational data are unlikely to be any less representative of partitioning between sediment organic carbon and fish lipids than the postoperational data and, therefore, aggregating the two data sets should provide a more robust estimate. Dioxin congener concentrations in fish tissue presented in Bauer (1992) were normalized by the mean fish lipid percent (5.57 percent). This resulted in a congener concentration in the fish lipid (mg congener/kg lipid). The congener concentrations in the fish lipids from the preoperational and operational facilities were weighted by the respective sample sizes and divided by the total number of samples (preoperational plus operational) to yield a weighted mean congener concentration in fish lipids. Dioxin congener concentrations in OC sediments were similarly addressed to derive a weighted mean congener concentration in OC sediment. The congener-specific BSAFs were calculated as the ratio of the weighted mean concentration in lipid to the weighted mean concentration in OC sediments.

Calculated BSAFs are presented in Table 5-8.

In reviewing the BASFs given in Table 5-8, it should be noted that fugacity theory would predict that if fish lipid and organic carbon have similar fugacity capacities, then the BSAF values should approach 1 at equilibrium. The results in Table 5-8 indicate this is indeed approached for the tetra and penta congeners (with BSAF values ranging from 0.2 to 0.8) but not for the more highly chlorinated congeners. This is not unexpected given how strongly the more highly chlorinated congeners are bound to sediments and the slow rate of uptake of these compounds in fish.

The BSAF values were needed to estimate exposures to humans and terrestrial mammals and birds consuming fish. The same BSAF values were used for both human and ecological receptor populations to estimate food chain transfer of dioxin and furan congeners. BSAFs were used as shown in Equation 5-32 to calculate congener concentrations in fish for human consumption. BSAFs were also used to determine food chain exposures to receptors that forage in the freshwater ecosystem (see Section 9).

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6.0 Human Exposure and Risk Methodology

The human health risk analysis completed for the final rule assessed risks resulting from HWC emissions deposited within (or air concentrations modeled for) a 20-km radius area surrounding each facility within the HWC universe. Emissions transported beyond the 20-km radius study areas were not considered. This focus resulted in an analysis that primarily targeted risk to individuals residing within these study areas. However, the analysis also included a population risk component that estimated statistical cancer incidence associated with the national consumption of food commodities raised within HWC study areas (these commodities contain varying levels of dioxin as a result of their proximity to HWC facilities). To provide greater spatial resolution in exposure assessment (for both human and livestock populations), the 20-km radius study area surrounding each modeled facility was subdivided using a 16-sector GIS-based template. The use of the template allows greater resolution in characterizing both the spatial distribution of modeled media concentrations and the spatial distribution of human receptors, both key factors in exposure assessment (see Section 4.3).

Key Attributes of Exposure Assessment for the Final Rule

Study area focus: Individuals residing within 20 km of HWC facilities (with exception of dioxin contained in locally grown food commodities that are distributed nationally).

Sector-level analysis: 16-sector template used in site characterization.

Pathways considered: All receptors evaluated for soil ingestion, inhalation, and tap water ingestion (if local waterbody identified as drinking water source). Food chain pathways evaluated for certain receptors.

Exposure parameter variability analysis: Factored into intake/dose calculation for key risk-driving receptor populations including commercial dairy and beef farmers (for dioxin) and recreational fishers (for mercury).

Section 6.1 describes intake and dose calculations, Section 6.2 describes human receptors and exposure pathways, Section 6.3 presents exposure factors, Section 6.4 presents exposure estimates, Section 6.5 discusses body burden estimates, and Section 6.6 summarizes background exposures for dioxin, lead, and mercury.

6.1 Overview of Approach

Exposure assessment for the final rule has two fundamental components: (1) assessing intake for a representative individual from each of the modeled receptor populations within a given sector, and (2) projecting the number of individuals experiencing that level of exposure (i.e., number of individuals from a given receptor population within each sector). When combined, these two components of exposure assessment allow both population-weighted

individual risk distributions[1] and population risk statements to be generated. This section presents the methodology and data sources used to generate the sector-level intake rates for each of the receptor populations. Section 4.4 presents the methodology and data sources used to generate the sector-level population projections for the receptor population evaluated in the HWC risk analysis.

The risk analysis completed for the proposed rule included three exposure categories based on three different assumptions regarding exposure:

- Central tendency–aimed at approximating near 50th percentile exposure and risk
- High-end–designed to represent exposure above the 90th percentile of the distribution of individual exposures, but not higher than the individual with the highest exposure
- Bounding–a hypothetical scenario involving placement of receptors at the highest modeled concentration locations; designed to account for the relatively low sample size for modeled facilities included in the proposed rule.

> The number of modeled HWC facilities increased from 11 for the proposed rule to 76 for the final rule.

For the final rule, central-tendency exposure estimates were used to represent each sector within the modeled study areas. Characterization of exposure for high-end receptors was accomplished by identifying upper percentiles (e.g., 90th, 95th, or 99th) from the population-weighted distributions that were generated for each receptor population. This strategy allows the spatial distribution of individuals within a given receptor population around modeled HWC facilities to form the basis for differentiating central-tendency from high-end exposure (i.e., the 99th percentile estimate would represent a specific sector that, due to a combination of its exposure level and population density relative to other modeled sectors, would potentially contain the 99th percentile individuals). In addition, for key risk-driving receptor populations (i.e., the commercial beef and dairy farmers and the recreational fisher), the impact of exposure parameter variability (i.e., intrasector variability) on exposure was assessed using a variability analysis designed specifically for the HWC risk analysis (Section 6.3.2). This variability analysis allows the aggregate impact of interfacility, intersector, and intrasector variability on exposure to be evaluated. The risk analysis completed for the final rule included a significant increase in the number of modeled HWC facilities relative to the proposed rule (76 versus 11). This increase removed the need for the bounding estimates included in the proposed rule since the increased sample size reduces the probability that the modeled facilities selected for a given combustor category will not include a “high-risk” facility (see Section 4.1).

[1] Population-weighted individual risk distributions are developed by first weighting sector-level individual risk values by the number of individuals from that receptor population located within that sector and then pooling all of those population-weighted individual risk values (for a given combustor category). For a detailed discussion of population-weighted individual risk distributions, see Section 8.

Two specialized analyses conducted for the HWC risk analysis (i.e., breast milk incremental margin of exposure (MOE) and the blood lead level analysis) involve specialized exposure modeling that is described in Sections 6.3 and 6.4. The incremental margin of exposure analysis takes aggregate intake rates for dioxin-TEQ for adult receptors and projects resulting breast milk concentrations for dioxin-TEQ. These breast milk estimates are then used in the incremental margin of exposure analysis for infant exposure. In the case of lead, aggregated intake rates for lead for a given receptor are used to project resulting blood lead levels, which are used in characterizing risk associated with lead exposure.

6.2 Human Receptors and Exposure Pathways

The receptor populations evaluated for the final rule represent those individuals identified as having the highest potential exposures as a result of both direct and indirect exposure to HWC emissions. Some receptor populations evaluated under the proposed rule had relatively low potential exposures and thus were dropped from the analysis for the final rule (e.g., commercial poultry farmers because commercial poultry farming practices do not result in significant exposure to dioxins). An individual's exposure through inhalation was assumed to occur exclusively in the sector in which the individual was located based on Census data (i.e., mobility between sectors was assumed to be minimal). Receptors were evaluated for four age groups: 0-5 years, 6-11 years, 12-19 years, and >19 years[2].

Age Groups Evaluated for Final Rule

Four age groups were considered for every receptor population and exposure scenario:

- 0-5 years
- 6-11 years
- 12-19 years
- >19 years.

Receptor populations evaluated for the final rule were: commercial beef, dairy, pork, and produce farmers, residents, home gardeners, and recreational fishers. These populations are referred to as enumerated receptor populations because it is possible to generate population projections for these receptors. Section 4.4 presents the methodology and Census data used to generate the sector-level population projections for the enumerated receptor populations.

Receptor Populations Evaluated for Final Rule

- Commercial beef, dairy, pork, and produce farmers
- Residents
- Home gardeners
- Recreational fishers

Subsistence Scenarios Evaluated for Final Rule

- Subsistence farmers
- Subsistence fishers

[2] Although the HWC risk analysis does generate age-group-specific exposure estimates, age-group-differentiated toxicity factors were not used in the analysis - these values are not currently available for the majority of the chemicals evaluated. Consequently, the majority of risk estimates that were generated, although reflecting age-dependent differences in exposure, cannot be considered truly age-group-specific because they do not reflect age-dependent differences in susceptibility to toxic effects.

Although recreational fishers are an enumerated population, there are no ready means by which to determine at which waterbodies they fish. An assumption of uniform population distribution across sectors was used in assessing exposure for the recreational fisher (i.e., a single individual from each receptor population was assumed to reside in each of the 16 sectors within a given study area). Because sector-level population totals were not available for the recreational fisher populations and population risk estimates could not be completed for them, semiquantitative population estimates at the study-area level were produced. The recreational fisher receptor population represents a special case in the HWC risk analysis and is discussed in detail in Section 4.4.1.2.

In addition to these receptor populations, exposures that could occur as a consequence of subsistence activities (i.e., subsistence scenarios) were also assessed, including subsistence farming and subsistence fishing. To characterize the range of exposures that could result from subsistence farming, it was assumed that a subsistence farm could be located in each sector, irrespective of whether other farming activity occurred there. Similarly, it was assumed that subsistence fishing would take place at each waterbody modeled. Human receptors and their associated exposures and population assumptions are presented in Table 6-1.

Table 6-1. Human Receptors

Receptor Population or Subsistence Scenario	Exposure Scenario	Population Enumeration Assumptions
Enumerated Population		
Commercial farmers Beef Dairy Pork Produce	Home-produced food linked to type of farming activity	Determined from U.S. Census and Census of Agriculture data (see Section 4.4)
Residents	Nondietary exposure	Determined from U.S. Census data
Home gardeners	Portion of dietary intake of fruits and vegetables from home production	Data on proportion of residents who engage in home gardening
Recreational fishers	Portion of dietary fish intake from recreationally caught fish	Equal distribution across all modeled sectors; data from U.S. Fish and Wildlife Service
Non-enumerated Population		
Subsistence farmers	Dietary intake exclusively from home-produced foods	Equal distribution across all modeled sectors
Subsistence fishers	Dietary intake from self-caught fish	Equal distribution across all modeled sectors

The remainder of this section describes each of the receptor populations and subsistence scenarios evaluated for the final rule. Key assumptions used to define each receptor population (specifically, assumptions related to behavior) are provided along with exposure pathways considered in assessing overall exposure and risk for each receptor population. In addition to modeling adult exposure scenarios (i.e., ages >19 years), three younger age groups (i.e., 0-5, 6-11, and 12-19 years) were modeled for each receptor population so that age-dependent differences in exposure could be reflected in the risk estimates generated for the final rule. All receptor populations were evaluated for inhalation and incidental soil ingestion. Only those individuals residing within study areas identified as having surface waterbodies that were sources of drinking water were evaluated for exposure from tap water ingestion. Table 6-2 presents human receptors and their associated exposure pathways for both the receptor populations and the subsistence scenarios.

6.2.1 Enumerated Populations

6.2.1.1 <u>Farm Households</u>. All of the commercial farm households evaluated for the final rule were assumed to ingest home-produced food commodities linked to the type of farming activity in which they engage (e.g., the commercial beef farm households were assumed to ingest home-produced beef). Furthermore, the home-produced food items that they ingest were assumed to originate exclusively from their own farms (i.e., bartering of home-produced food items between individuals was not considered). The commercial farm households were considered enumerated receptor populations since sector-level age-specific population estimates could be generated (see Section 4.4). The pathways evaluated for each of the commercial farm households were

- Commercial Beef Farm–ingestion of home-produced beef
- Commercial Pork Farm–ingestion of home-produced pork
- Commercial Dairy Farm–ingestion of home-produced milk
- Commercial Produce Farm–ingestion of home-produced produce (including a combination of exposed fruits, root vegetables, and exposed vegetables).

In addition, commercial farm households were assumed to be exposed via inhalation and incidental soil ingestion, as well as tap water ingestion in those study areas for which a waterbody was used as a drinking water source. As discussed in Section 6.3, rates of consumption of home-produced foods were based on data specific to foods that are home-produced rather than as a percentage of the total dietary intake of these food commodities.

6.2.1.2 <u>Nonfarm Households (Residents and Home Gardeners)</u>. Nonfarm resident households were assumed to be exposed through soil ingestion, inhalation, and tap water ingestion (as noted earlier, tap water ingestion was evaluated only for those study areas containing verified drinking water sources).

Table 6-2. Human Receptors and Their Exposure Pathways

Receptor	Household Description	Exposure Pathways[a]
Beef farmers	Raise commercial beef cattle	Ingestion of beef from home-produced cattle
Pork farmers	Raise commercial hogs	Ingestion of pork from home-produced hogs
Dairy farmers	Raise commercial dairy cattle	Ingestion of milk from home-produced dairy cattle
Produce farmers	Raise crops for commercial market	Ingestion of home-grown fruits and vegetables
Home gardeners	Engage in home gardening	Ingestion of home-grown fruits and vegetables
Recreational fishers	Engage in recreational fishing	Ingestion of a portion of dietary fish from recreational fishing
Residents	Not engaged in farming or home gardening	Incidental ingestion of soil, ingestion of drinking water, inhalation of ambient air
Subsistence farmers	Obtain nearly all their dietary intake from home-produced foods	Ingestion of home-produced beef, pork, chicken, eggs, milk, root vegetables, exposed fruit, exposed vegetables, and fish caught on farm ponds
Subsistence fisher	Obtain a significant portion of their dietary intake from self-caught fish	Ingestion of fish caught during subsistence fishing activity

[a] All human receptors were evaluated for inhalation and soil ingestion. Those residing within study areas identified as having surface waterbodies that were sources of drinking water were also evaluated for exposures from tap water ingestion.

Sector-level population totals for the home gardener were subtracted from the sector-level resident population to avoid double counting.

Home gardener households were assumed to obtain a portion of their dietary intake of fruits and vegetables from home production. As with the commercial produce farmer, home gardener households were assumed to ingest a combination of exposed fruits, root vegetables, and exposed vegetables. These population estimates were generated using data on the proportion of residents who engage in home gardening. The percentage of total households in the United States that have vegetables gardens was taken as 38 percent, from

Table 13-1, 1986 Vegetable Gardening by Demographic Factors, in the 1997 *Exposure Factors Handbook* (EFH) (U.S. EPA, 1997a). Sector-level population totals for the home gardener were subtracted from the sector-level resident population to avoid double counting the two populations.

6.2.2 Recreational Fishers

Recreational fisher households were assumed to obtain a portion of their dietary fish intake from recreationally caught fish. The recreational fisher household receptor populations represent a special case with regard to enumeration. Although sector-level population totals were not generated for these receptors, study-area-level population estimates of the numbers of fishers were used to make semiquantitative statements regarding the number of individuals potentially fishing in "at-risk" waterbodies (see Section 8.2.2). Fishing activity modeled for the recreational fisher was assumed to be distributed between the one to four modeled waterbodies identified for each study area. Specifically, the level of recreational fishing activity at a given modeled waterbody was assumed to be correlated to the relative surface area of each waterbody—the larger the waterbody, the greater the recreational fishing activity. In addition to fish ingestion, recreational fisher households were evaluated for exposures via inhalation and incidental soil ingestion, as well as tap water ingestion in those study areas for which a waterbody was used as a drinking water source.

6.2.3 Subsistence Receptors (Farmers and Fishers)

Subsistence farm households obtain nearly all of their dietary intake of food commodities from home production. For this scenario, it was assumed that one household was located in each sector within a given study area. This scenario was evaluated for exposure through ingestion of home-produced beef, pork, chicken, eggs, milk, root vegetables, exposed fruit, exposed vegetables, and fish caught on farm ponds. For purposes of evaluating the fish ingestion pathway, a farm pond was modeled for each sector from which fish were obtained. Subsistence activity for this receptor is reflected in the wide variety of food commodities that are obtained from home production. In addition, subsistence farm households were evaluated for exposures via inhalation and incidental soil ingestion, as well as tap water ingestion in those study areas for which a waterbody was used as a drinking water source.

Subsistence fisher households obtain a significant portion of their dietary intake from self-caught fish. Fishing activity by a given subsistence fisher household was assumed to be restricted to a single modeled waterbody (specifically one of the one to four modeled waterbodies selected for each study area). Therefore, one to four subsistence fisher households were evaluated for each modeled sector—one household for each of the modeled waterbodies identified for a given study area. This receptor scenario was evaluated for exposure through fish ingestion in addition to exposures via inhalation and incidental soil ingestion, as well as tap water ingestion in those study areas for which a waterbody was used as a drinking water source. Subsistence activity for this scenario is reflected in the relatively high ingestion rate for self-caught fish (see Section 6.3.2.4).

6.3 Exposure Factors

This section presents the methodologies and data sources used to generate intake rates and dose estimates for each of the modeled exposure pathways. For a given pathway, separate intake rates were generated for each of the nine human receptor households and for each of the four age groups within a household, reflecting the differences in behavior between the different receptors and age groups. Consequently, the discussion of each pathway includes an explanation of the exposure factors developed specifically for each of the receptors and age groups. (Note: For several of the pathways, the same parameter value was used for several receptors and/or age groups reflecting either a lack of data or an inability to differentiate the receptors with regard to that pathway.) The general equations for calculating the average daily dose (ADD) and lifetime average daily dose (LADD) are discussed in Section 6.4. The actual equations used to calculate receptor-specific intake rates for a given exposure pathway are presented in Appendix C. The derivation of media and food chain concentrations (i.e., exposure point concentrations), which are a critical input in the calculation of chemical intake, is discussed in Sections 5.3 and 5.4.

Mean values were used for each of the exposure factors for all of the receptors evaluated. The mean is a statistical descriptor that has highly desirable numerical properties (e.g., the product of the means of two or more variables is the mean of the product of the variables). This property is important for estimating population risk and is, therefore, used throughout the exposure analysis. However, because the mean is influenced by the upper tail of the underlying distribution, it will always be greater than the median, or 50th percentile, value. Although the use of mean exposure factors is necessary for estimating population risk (number of cases), the aggregate impact of multiple mean exposure factors on a single risk estimate can result in individual risk estimates that are more accurately classified as upper percentile than central tendency.

6.3.1 Central Tendency Exposure Factors

This section first presents the methodology and data sets used to derive key parameters (i.e., ingestion rates, body weight, and exposure duration) that are applicable to each receptor. Methodologies and data sets used to calculate unadjusted ingestion rates for each food commodity within a specific food category follow. Ingestion rate derivations are grouped by food category because similar methodologies were used to derive ingestion rates for each commodity within a category. Adjustment factors used to account for food losses, such as cooking or preparation losses, are also presented in this section and the final receptor-specific ingestion rates derived. Table 6-3 summarizes the central tendency exposure parameters and values for all the exposure factors used in the HWC analysis; specific methodologies and data used are presented in the following sections. All exposure factors were obtained or derived from information contained in the *Exposure Factors Handbook* (U.S. EPA, 1997a), referred to as the "EFH" in the following discussion.

6.3.1.1 <u>Body Weight</u>. No distinction was made between different receptors (e.g., farmer versus fisher) with respect to body weight. However, different body weights were identified for each of the four age groups evaluated for the final rule.

Table 6-3. Exposure Factor Point Estimates Used in HWC Risk Analysis

Receptor Population, Age Group (yr)	Body Weight (kg)	Exposure Duration (yr)	Inhalation (m^3/d)	Incidental Soil Ingestion (mg/d)	Tap Water Ingestion (L/d)	Breast Milk Ingestion (L/d)	Meat Ingestion (beef, pork, chicken) (g/kg-d)			Fish Ingestion (g/kg-d)	Dairy (eggs, milk) (g/kg-d)		Root and Exposed Vegetables; Exposed Fruit (g/kg-d)		
							B	P	C		E	M	EV	RV	EF
Subsistence Receptor Populations															
Subsistence Farmer															
0-5	14.3	6.5	6.5	179	0.653	0.742	1.8	1.5	1.7	0.37	1.69	70.0	0.15	0.41	0.28
6-11	30.7	8.9	11.8	100	0.787	-	2.1	0.91	1.3	0.28	1.10	35.3	0.082	0.29	0.27
12-19	58.3	9.1	14.0	100	0.963	-	0.95	0.59	0.64	0.15	0.66	16.2	0.064	0.20	0.14
>19	71.8	17.3	13.3	50	1.38	-	1.1	0.51	0.63	0.16	0.61	7.1	0.085	0.23	0.13
Subsistence Fisher															
0-5	14.3	6.5	6.5	179	0.653	0.742	-	-	-	1.37	-	-	-	-	-
6-11	30.7	8.9	11.8	100	0.787	-	-	-	-	1.37	-	-	-	-	-
12-19	58.3	9.1	14.0	100	0.963	-	-	-	-	0.97	-	-	-	-	-
>19	71.8	17.3	13.3	50	1.38	-	-	-	-	0.97	-	-	-	-	-
Nonsubsistence (Commercial) Receptor Populations															
Beef Farmer															
0-5	14.3	6.5	6.5	179	0.653	0.742	1.8	-	-	-	-	-	-	-	-
6-11	30.7	8.9	11.8	100	0.787	-	2.1	-	-	-	-	-	-	-	-
12-19	58.3	9.1	14.0	100	0.963	-	0.95	-	-	-	-	-	-	-	-
>19	71.8	17.3	13.3	50	1.38	-	1.1	-	-	-	-	-	-	-	-

(continued)

Table 6-3. (continued)

Receptor Population, Age Group (yr)	Body Weight (kg)	Exposure Duration (yr)	Inhalation (m^3/d)	Incidental Soil Ingestion (mg/d)	Tap Water Ingestion (L/d)	Breast Milk Ingestion (L/d)	Meat Ingestion (beef, pork, chicken) (g/kg-d)			Fish Ingestion (g/kg-d)	Dairy (eggs, milk) (g/kg-d)		Root and Exposed Vegetables; Exposed Fruit (g/kg-d)		
							B	P	C		E	M	EV	RV	EF
Pork Farmer															
0-5	14.3	6.5	6.5	179	0.653	0.742	-	1.5	-	-	-	-	-	-	-
6-11	30.7	8.9	11.8	100	0.787	-	-	0.91	-	-	-	-	-	-	-
12-19s	58.3	9.1	14.0	100	0.963	-	-	0.59	-	-	-	-	-	-	-
>19	71.8	17.3	13.3	50	1.38	-	-	0.51	-	-	-	-	-	-	-
Dairy Farmer															
0-5	14.3	6.5	6.5	179	0.653	0.742	-	-	-	-	-	70.0	-	-	-
6-11	30.7	8.9	11.8	100	0.787	-	-	-	-	-	-	35.3	-	-	-
12-19	58.3	9.1	14.0	100	0.963	-	-	-	-	-	-	16.2	-	-	-
>19	71.8	17.3	13.3	50	1.38	-	-	-	-	-	-	7.1	-	-	-
Produce Farmer															
0-5	14.3	6.5	6.5	179	0.653	0.742	-	-	-	-	-	-	0.15	0.41	0.28
6-11	30.7	8.9	11.8	100	0.787	-	-	-	-	-	-	-	0.082	0.29	0.27
12-19	58.3	9.1	14.0	100	0.963	-	-	-	-	-	-	-	0.064	0.20	0.14
>19	71.8	17.3	13.3	50	1.38	-	-	-	-	-	-	-	0.085	0.23	0.13
Home Gardener															
0-5	14.3	6.5	6.5	179	0.653	0.742	-	-	-	-	-	-	0.15	0.41	0.28
6-11	30.7	8.9	11.8	100	0.787	-	-	-	-	-	-	-	0.082	0.29	0.27
12-19	58.3	9.1	14.0	100	0.963	-	-	-	-	-	-	-	0.064	0.20	0.14
>19	71.8	13.5	13.3	50	1.38	-	-	-	-	-	-	-	0.085	0.23	0.13

(continued)

Table 6-3. (continued)

Receptor Population, Age Group (yr)	Body Weight (kg)	Exposure Duration (yr)	Inhalation (m^3/d)	Incidental Soil Ingestion (mg/d)	Tap Water Ingestion (L/d)	Breast Milk Ingestion (L/d)	Meat Ingestion (beef, pork, chicken) (g/kg-d)			Fish Ingestion (g/kg-d)	Dairy (eggs, milk) (g/kg-d)		Root and Exposed Vegetables; Exposed Fruit (g/kg-d)		
							B	P	C		E	M	EV	RV	EF
Recreational Fisher															
0-5	14.3	6.5	6.5	179	0.653	0.742	-	-	-	0.37	-	-	-	-	-
6-11	30.7	8.9	11.8	100	0.787	-	-	-	-	0.28	-	-	-	-	-
12-19	58.3	9.1	14.0	100	0.963	-	-	-	-	0.15	-	-	-	-	-
>19	71.8	13.5	13.3	50	1.38	-	-	-	-	0.16	-	-	-	-	-
Resident															
0-5	14.3	6.5	6.5	179	0.653	0.742	-	-	-	-	-	-	-	-	-
6-11	30.7	8.9	11.8	100	0.787	-	-	-	-	-	-	-	-	-	-
12-19	58.3	9.1	14.0	100	0.963	-	-	-	-	-	-	-	-	-	-
>19	71.8	13.5	13.3	50	1.38	-	-	-	-	-	-	-	-	-	-

Note that extrapolated dairy ingestion rates for the younger age groups appear high in comparison to the values provided in EFH Table 13-28 for the total population. Although these values appear high, note that younger age groups are anticipated to consume more milk than are adults, and therefore this result is not unexpected.

Data presented in the EFH (U.S. EPA, 1997a) were used to derive body weight estimates for each of the four age groups. Mean-based estimates were used for all receptors. Data for body weights of children include mean body weight values (boys and girls aggregated) for each year of age up to 19 years. These data were used to derive body weight values for the first three age groups (i.e., 0-5, 6-11, and 12-19 years) by averaging those years together that fall within each age group. For example, body weights for ages 6, 7, 8, 9, 10, and 11 were averaged to approximate a body weight for the 6- to 11-yr-old age group. The mean value for "Men and Women" 18 to 75 years old was used for all >19-yr-old (adult) age groups. The resulting body weights for the four age groups are given in Table 6-4.

6.3.1.2 Exposure Durations. Data presented in the 1997 EFH were used to select mean exposure durations for each of the four age groups. Separate exposure durations were used for farm and nonfarm adults (the latter including recreational fishers, residents, and home gardeners). Adult subsistence fishers were assumed to have an exposure duration equal to that of the subsistence farmer. A single set of exposure durations was established for all three of the younger age groups (i.e., 0-5, 6-11, and 12-19 years) since there were no age-specific data characterizing exposure durations for different categories of receptors (i.e., farmer versus resident). The exposure durations and supporting information for each of the four age groups are given in Table 6-5.

6.3.1.3 Inhalation. No distinction was made between different receptors with respect to inhalation rates. However, different inhalation rates were identified for each of the four age groups evaluated for the final rule. The recommended inhalation value for adults from the 1997 EFH was used to represent the >19-yr-old age group. However, EFH-recommended rates for other age groups corresponded poorly to the three lower age groups of interest in the HWC risk analysis (i.e., 0-5, 6-11, and 12- to 19-yr-old age groups); therefore, inhalation rates based on metabolic rates as cited in the 1997 EFH were used to derive these inhalation rates. Age groups were selected to represent the three lower age groups evaluated in the HWC analysis (see Table 6-6).

6.3.1.4 Incidental Soil Ingestion. The 1997 EFH provides a recommended incidental soil ingestion value for children of 100 mg/d; however, as noted, this value may not reflect the contribution of intermittent pica behavior since it is based on studies of relatively short duration. Therefore, the mean value presented for the "overall" child study population (179 mg/d) was selected to represent the 0- to 5-yr-old age group. Because this value is larger than the 100-mg/d value, it may be considered reflective of intermittent pica behavior. The 1997 EFH-recommended incidental soil ingestion value for children (i.e., 100 mg/d) was selected to represent the next two age groups (6- to 11- and 12- to 19-yr-old age groups). This value was selected based on the assumption that pica behavior is uncommon among older children and adolescents. The 1997 EFH-recommended mean value for adults (50 mg/d) presented in the "Results" section of EFH, Chapter 4, was used for all adult receptors (i.e., >19-yr-old age group). The incidental soil ingestion rates and supporting information for each of the four age groups are presented in Table 6-7.

6.3.1.5 Tap Water Ingestion. Age-group-specific tap water ingestion rates for receptors were derived using data tabulated in the 1997 EFH. Tap water ingestion rates for all

Table 6-4. Representative Body Weights and Sources

Age Group (yr)	Body Weight (kg)	Body Weight Values Averaged	Original Reference	1997 *Exposure Factors Handbook* Table Name
0-5	14.3	6-11 mo, 1, 2, 3, 4, 5 yr	NCHS 1987	Body Weights of Children (kg) (Table 7-3)
6-11	30.7	6, 7, 8, 9, 10, 11 yr	NCHS 1987	Body Weights of Children (kg) (Table 7-3)
12-19	58.3	12, 13, 14, 15, 16, 17, 18, 19 yr	NCHS 1987	Body Weights of Children (kg) (Table 7-3)
>19	71.8	18-<75 yr	NCHS 1987	Body Weights of Adults (kg) (Table 7-2)

EFH = *Exposure Factors Handbook* (U.S. EPA, 1997a).
NCHS = National Center for Health Statistics.

Table 6-5. Representative Exposure Durations and Sources

Age Group (yr)	Exposure Duration (yr)	Receptors	Mean Residential Occupancy Period Age Group Used (yr)	Original Reference	1997 *Exposure Factors Handbook* Table Name
0-5	6.5	All	3	Johnson and Capel, 1992	Descriptive Statistics for Both Genders by Current Age (Table 15-168)
6-11	8.9	All	5	Johnson and Capel, 1992	Descriptive Statistics for Both Genders by Current Age (Table 15-168)
12-19	9.1	All	15	Johnson and Capel, 1992	Descriptive Statistics for Both Genders by Current Age (Table 15-168)
>19	13.5	All but farmers and subsistence fishers	42	Johnson and Capel, 1992	Descriptive Statistics for Both Genders by Current Age (Table 15-168)
>19	17.3	Farmers and subsistence fishers	Overall	Israeli and Nelson, 1992	Values and Their Standard Errors for Average Total Residence Time, T, for Each Group in Survey (Table 15-163)

Table 6-6. Representative Inhalation Rates and Sources

Age Group (yr)	Inhalation rate (m^3/d)	Receptors	Age Groups of Mean Inhalation Rates Used	Original Reference	1997 *Exposure Factors Handbook* Table Name
0-5	6.5	All	<1, 1-2, 3-5	Layton, 1993	Daily Inhalation Rates Calculated from Food-Energy Intakes (Table 5-11)
6-11	11.8	All	6-8, 9-11	Layton, 1993	Daily Inhalation Rates Calculated from Food-Energy Intakes (Table 5-11)
12-19	14.0	All	12-14, 15-18	Layton, 1993	Daily Inhalation Rates Calculated from Food-Energy Intakes (Table 5-11)
> 19	13.3	All	NA	NA	EFH-recommended value

NA = Not applicable.

Table 6-7. Soil Ingestion Rates and Sources

Age Group (yr)	Soil Ingestion Rate (mg/d)	Soil Ingestion Rate Used	Original Reference	1997 *Exposure Factors Handbook* Table
0-5	179	Overall child	Stanek & Calabrese, 1995	Distribution of Average (Mean) Daily Soil Ingestion Estimates Per Child for 64 Children (mg/day) (Table 4-9)
6-11	100	Child	EFH Chapter 4 Recommendation	Not applicable
12-19	100	Child	EFH Chapter 4 Recommendation	Not applicable
>19	50	Adult	EFH Chapter 4 Recommendation	Not applicable

receptors were characterized using mean intake values; when the age groups presented did not correspond to those used in the HWC risk analysis, various age-group-specific data were combined by first weighting individual values to reflect their respective sample sizes and then summing the weighted values. The tap water ingestion rates for the four age groups and supporting information are presented in Table 6-8.

6.3.1.6 <u>Breast Milk Ingestion</u>. The breast milk ingestion pathway was evaluated exclusively for dioxin TEQ because of its lipophilic characteristics and, consequently, its propensity for bioaccumulation in breast milk. The “recommended” average value for 0- to 6-month-old children presented in the 1997 EFH (0.742 L/d) was adopted for use in the HWC risk analysis. The same value was used for all receptors (e.g., farmers, fishers).

Table 6-8. Representative Tap Water Ingestion Rates and Sources

Age Group (yr)	Ingestion Rate (L/d)	Mean Tap Water Ingestion Rates Used (yr)	Original Reference	1997 *Exposure Factors Handbook* Table Name
0-5	0.653	<0.5, 0.5-0.9, 1-3, 4-6	Ershow and Cantor, 1989	Total Tapwater Intake (mL/day) for Both Sexes Combined (Table 3-6)
6-11	0.787	7-10	Ershow and Cantor, 1989	Total Tapwater Intake (mL/day) for Both Sexes Combined (Table 3-6)
12-19	0.963	11-14, 15-19	Ershow and Cantor, 1989	Total Tapwater Intake (mL/day) for Both Sexes Combined (Table 3-6)
>19	1.38	20-44, 45-64, 65-74	Ershow and Cantor, 1989	Total Tapwater Intake (mL/day) for Both Sexes Combined (Table 3-6)

6.3.1.7 Dietary Ingestion Rates for Meats (Excluding Fish) and Fruits/Vegetables. In characterizing risks to local receptor populations (i.e., those receptors located within study areas) resulting from food ingestion, the HWC risk analysis focused on the ingestion of home-produced dietary items. The same basic methodology was used to identify dietary ingestion rates for beef, pork, poultry, fruits/vegetables, milk, and eggs because dietary ingestion rates for these food items are based on data obtained from the 1997 EFH 87/88 NFCS data tables summarizing the mean intake of **home-produced** food items. The methodology uses the USDA 87/88 data for food consumption because the HWC analysis is based on consumption of local commodities. The Nationwide Food Consumption Survey (NFCS) data were used to generate intake rates for home-produced foods (U.S. EPA, 1997a). The USDA conducts the NFCS every 10 years to analyze the food consumption behavior of Americans; the most recent NFCS was 1987/1988 (U.S. EPA, 1997a).

Specific tables used are presented in Table 6-9. The derivation of ingestion rates for fish, which is based on alternate data sources, is discussed in Section 6.3.1.9. The same ingestion rates were used for the commercial farm households as for the subsistence family households. Also, the same ingestion rates were used for the home gardener households as for the produce farmer households.

Limitations of the 87/88 NFCS data (e.g., missing ingestion rates for certain age groups) required that two types of derivations be completed to generate a complete set of unadjusted ingestion rates for all of the age group/receptor population combinations evaluated in the HWC risk analysis. These derivations[3] included:

[3]The HWC risk analysis assumed that all individuals consume the food commodity being evaluated (i.e., that all individuals are "consumers"). Thus, data for "consumers" were preferred when extrapolating dietary ingestion rates for age groups not covered in the home-produced data tables.

Table 6-9. Summary of References Used To Determine Unadjusted Intake Rates

Commodity	Original Reference	1997 *Exposure Factors Handbook* Table Name
Beef	87/88 NFCS	Consumer-Only Intake of Home-Produced Beef (Table 13-36)
Pork	87/88 NFCS	Consumer-Only Intake of Home-Produced Pork (Table 13-54)
Chicken	87/88 NFCS	Consumer-Only Intake of Home-Produced Poultry (Table 13-55)
Meat (beef, pork, chicken)	USDA, 1992	Mean Meat Intakes per Individual in a Day by Sex and Age (g/day as consumed) for 1987-1988 (Table 11-11)
Exposed fruit	87/88 NFCS	Consumer-Only Intake of Homegrown Exposed Fruit (g/kg-day) (Table 13-61)
Exposed vegetables	87/88 NFCS	Consumer-Only Intake of Homegrown Exposed Vegetables (g/kg-day) (Table 13-63)
Root vegetables	87/88 NFCS	Consumer-Only Intake of Homegrown Exposed Root Vegetables (g/kg-day) (Table 13-65)
Milk	87/88 NFCS	Consumer-Only Intake of Home-Produced Dairy (g/kg-day) (Table 13-28)
Eggs	87/88 NFCS	Consumer-Only Intake of Home-Produced Eggs (g/kg-day) (Table 13-43)
Milk and eggs	USDA, 1992	Mean Dairy Product Intakes Per Individual in a Day (g/day as consumed) for 1987-1988 (Table 11-13)
Fish (rec. fisher, sub. farmer)	West et al., 1989	Mean Fish Intake Among Individuals Who Eat Fish and Reside in Households with Recreational Fish Consumption (Table 10-61)
Fish (sub. fisher)	EFH Ch. 12	Not applicable

NFCS = Nationwide Food Consumption Survey.
USDA = U.S. Department of Agriculture.

Extrapolating the home-produced ingestion rate(s) for missing age groups: When ingestion rate data for home-produced food items were not available for a given age group, age-group-specific data from 87/88 NFCS tables summarizing ingestion patterns for the national population (i.e., data that are not specific to the ingestion of home-produced food items) were used to extrapolate the home-produced ingestion rate for the age group of interest. Specifically, the mean home-produced ingestion rate for **the total sampled population** (in units of grams/day) was multiplied by **the ratio of the mean ingestion rate for the age group of interest in the general population to the mean ingestion rate for all ages in the general population**. This extrapolation assumes that age differences in food consumption in the general population are representative of age differences in households that consume home-produced foods. It is necessary to have all of the ingestion rates used in the extrapolation in units of grams/day if the age-group-specific ingestion rates are to fully reflect differences in intake across age groups.

Although the ultimate output is converted to units of grams/kilogram-day, the extrapolation is performed in units of grams/day for consistency in the calculation. Once the extrapolated ingestion rate was generated using the approach described here, that value was divided by the body weight for the age group of interest to convert the ingestion rate into units of gram/kilogram per day. This process is referred to as the "general population extrapolation" in subsequent text and tables.

Deriving average ingestion rates for the 0- to 5-yr-old and >19-yr-old age groups: The 87/88 NFCS tables present age-specific ingestion rates for the 1- to 2-yr-old, 3- to 5-yr-old, 20- to 39-yr-old, 40- to 69-yr-old, and (in some cases) 70+-yr-old age groups. These ingestion rates must be combined to generate a single ingestion rate for the 0- to 5-yr-old and >19-yr-old age groups. To generate these single ingestion rates, the ingestion rates for each age group (i.e., 1-2, 3-5, 20-39, 40-69, and 70+) were first weighted by their relative sample size (i.e., each ingestion rate multiplied by **the ratio of sample size for the age group of interest to sample size for all age groups combined**) and then summed. This process is referred to as "age group averaging" in subsequent text and tables.

The values obtained from the 87/88 NFCS data tables for meats, fruits/vegetables, and dairy products are **unadjusted** ingestion rates (UIRs) that had to be adjusted to reflect the following factors before they were used in the HWC risk analysis: (1) preparation/cooking and postcooking losses (meats and fruits/vegetables), (2) wet weight to dry weight conversion (fruits/vegetables only), and (3) fraction of overall dairy consumption that is milk (dairy products only). The 1997 EFH provides data sources that allow the characterization of each of these factors. Table 6-10 presents the unadjusted ingestion rates, adjustment factors, and final adjusted intake rates for the commodities considered in the HWC analysis.

Ingestion rates for certain age groups that required derivation of a UIR are indicated; these derivations are explained in the text specific to the commodities consumed. Similarly, loss adjustment factors are discussed in the commodity-specific sections that follow. Adjustment factors are presented in Table 6-10; references for the sources of the loss factors are summarized in Table 6-11.

Meats (Excluding Fish). To obtain a complete set of unadjusted ingestion rates for all age-group/receptor combinations (for beef, pork, and poultry), the derivations presented in Table 6-12 had to be completed.

Figure 6-1 presents a sample calculation (general population extrapolation) of the unadjusted ingestion rate for beef for the 0- to 5-yr-old age group.

Once the unadjusted ingestion rate was established for each age group, either directly from the EFH (U.S. EPA, 1997a) or derived from data therein, the values were adjusted to account for losses (i.e., preparation, cooking, and postcooking losses). This step resulted in the derivation of the adjusted ingestion rate (AIR). Equation 6-1 was used to generate mean adjusted ingestion rates for home-produced meats:

Table 6-10. Adjustment Factors and Unadjusted Age-Group-Specific Intake Rates Used in Deriving Ingestion Rates for Food Commodities

Food Type, Receptor	Unadjusted Ingestion Rates (g/kg-d)				Adjustment Factors for Losses	Final Age-Group-Specific Ingestion Rates (g/kg-d)			
	Child (0-5 yr)	**Child (6-11 yr)**	**Adolescent (12-19 yr)**	**Adult (>19 yr)**		**Child (0-5 yr)**	**Child (6-11 yr)**	**Adolescent (12-19 yr)**	**Adult (>19 yr)**
Meats (beef, pork, poultry)									
Beef farm household, Subsistence farm household	3.2[a]	3.8	1.7	2.0[b]	0.27 cooking; 0.24 postcooking losses	1.8	2.1	0.95	1.1
Pork farm household, Subsistence farm household	3.3[a]	2.0[a]	1.3	1.1[b]	0.28 cooking; 0.36 postcooking losses	1.5	0.91	0.59	0.51
Subsistence farm household (poultry)	3.5[a]	2.8[a]	1.4[a]	1.3[b]	0.32 cooking; 0.31 postcooking losses	1.7	1.3	0.64	0.63
Vegetables (exposed/root) and Fruits (exposed) - dry weight intake calculated									
Subsistence farm household, Home gardener household (exposed vegetables)	2.5	1.4	1.1	1.4[b]	0.16 cooking losses; 0.93 wet weight fraction	0.15	0.082	0.064	0.085
Subsistence farm household, Home gardener household (root vegetables)	1.9	1.3	0.94	1.1[b]	-0.042 cooking losses; 0.79 wet weight fraction	0.41	0.29	0.20	0.23
Subsistence farm household, Home gardener household (fruit)	2.6	2.5	1.3	1.2[b]	0.21 preparation losses; 0.86 wet weight fraction	0.28	0.27	0.14	0.13
Dairy (milk, eggs)									
Dairy farm household, Subsistence farm household (milk)	90.7[a]	45.7[a]	21.0[a]	9.1[a]	0.77 fraction of total dairy intake that is milk	70.0	35.3	16.2	7.1
Subsistence farm household (eggs)	1.7[a]	1.1[a]	0.66[a]	0.61[b]	NA[c]	1.7	1.1	0.66	0.61
Freshwater Fish									
Recreational fisher	0.37	0.28	0.15[d]	0.16[a]	NA[e]	0.37	0.28	0.15	0.16
Subsistence fisher	1.37	1.37[c]	0.97[c]	0.97	NA[e]	1.37	1.37	0.97	0.97
Subsistence farmer	0.37	0.28	0.15[d]	0.16[a]	NA[e]	0.37	0.28	0.15	0.16

[a] General population extrapolation.
[b] Age group averaging.
[c] EFH does not provide subsistence fish ingestion rates for the 6- to 11- and 12- to 19-yr-old age groups. Therefore, the rates were assumed to equal rates for the 0- to 5- and >19-yr-old groups, respectively.
[d] Backcalculated using 1- to 5-, 6- to 10-, and 1- to 20-year sample sizes.
[e] Data used to establish ingestion rates account for losses.

Table 6-11. Summary of References Used To Determine Loss Adjustment Factor

Commodity	Adjustment Factor	Original Reference	1997 *Exposure Factors Handbook* Table Name
Meat	Cooking loss and postcooking loss	USDA, 1975	Percent Weight Losses from Preparation of Various Meats (Table 13-5)
Fruits	Cooking or preparation	USDA, 1975	Percent Weight Losses from Preparation of Various Fruits (Table 13-6)
Vegetables	Cooking or preparation	USDA, 1975	Percent Weight Losses from Preparation of Various Vegetables (Table 13-7)
Fruits & vegetables	Fraction of wet weight that is water	USDA, 1979-1986	Mean Moisture Content of Selected Fruits, Vegetables and Grains Expressed as Percentages of Edible Portions (Table 9-27)
Fruits & vegetables	Consumption ratios [a]	87/88 NFCS	Consumer-Only Intake of Homegrown [*Individual Fruit/Vegetables*] (g/kg-day) (Tables 13-34, -35, -37, -38, -39, -40, -42, -45, -47, -48, -50, -51, -53, -57, -58, -59, and -60)
Milk	Fraction of dairy ingested that is milk	USDA 1980, 1992, 1996	Main Daily Intake of Meat and Dairy Products Per Individual in a Day for USDA 1977-78, 87-88, 89-91, 94, and 95 Surveys (Table 11-8)

[a] These consumption ratios were used with individual fruit and vegetable loss factors to derive aggregated loss factors for exposed fruits and vegetables and root vegetables. This process is described in the "Fruits and Vegetables" section of the text, and values used are shown in Table 6-14.

EFH = *Exposure Factors Handbook* (U.S. EPA, 1997a).
USDA = U.S. Department of Agriculture.

Table 6-12. Required Derivations for Age Group/Receptor Population Unadjusted Ingestion Rates for Meats

Age Group (yr)	Meat	Derivation Performed
0-5	Beef	General population extrapolation
>19	Beef	Age group averaging
0-5, 6-11	Pork	General population extrapolation
>19	Pork	Age group averaging
0-5, 6-11, 12-19	Poultry	General population extrapolation
>19	Poultry	Age group averaging

Step 1. Extrapolate unadjusted ingestion rate (UIR) for 0- to 5-yr-old age group (87/88 NFCS data tables do not have home-produced beef ingestion rate for this age group):

$$UIR = (2.45\ g/kg\text{-}d)^{a}(60\ kg)^{b}[(10\ g/d)/32\ g/d)]^{c} = 45.9\ g/d$$

[a] Mean home-produced ingestion rate for all ages combined (EFH Table 13-16, Consumer-Only Intake of Home-Produced Beef).

[b] The body weight value that should be used in converting ingestion rates for the overall sampled population (i.e., all ages combined) into units of g/d.

[c] Ratio of beef ingestion rate (not home-produced) for 0- to 5-yr-old age group to that of entire sampled population (EFH Table 11-11, Mean Meat Intakes per Individual in a Day by Sex and Age (g/day as consumed) for 1987-1988).

Step 2. Convert the UIR for the 0- to 5-yr-old age group into units of g/kg-d:

$$UIR = (45.9\ g/d)/(14.3\ kg)^{d} = 3.2\ g/kg\text{-}d$$

[d] Body weight for 0- to 5-yr-old age group (see Table 6-4).

Figure 6-1. Sample calculation for beef unadjusted ingestion rate (0- to 5-yr-old age group, beef farmer).

Dietary Ingestion Rates for Meats

$$AIR_{meat} = (UIR)\ (1\text{-}CL)\ (1\text{-}PCL) \qquad (6\text{-}1)$$

Parameter	Definition
AIR_{meat}	Adjusted ingestion rate for meat (value used in HWC risk analysis)
UIR[a]	Unadjusted age-group-specific ingestion rates
CL	Cooking losses — dripping and volatile losses during cooking.
PCL	Postcooking losses — cutting, shrinking, excess fat, scraps, and juices, all of which occur following cooking.

[a]UIR values had to be extrapolated for several age group/meat type combinations.

Figure 6-2 presents a sample calculation of a beef-adjusted ingestion rate. Table 6-10 presents the specific values used for each of the adjustment factors described as well as the final central tendency ingestion rates for home-produced meats that were used in the HWC risk analysis.

Use unadjusted ingestion rate (UIR) for 0- to 5-yr-old age group to generate adjusted ingestion rate (AIR) for beef that will be used in risk equations (this AIR accounts for cooking and postcooking losses):

$$AIR_{meat} = (3.2\ g/kg\text{-}d)^{a}(1\text{-}0.27)^{b}\ (1\text{-}0.24)^{c} = 1.8\ g/kg\text{-}d$$

[a] UIR for beef for 0- to 5-yr-old age group (derived in Figure 6-1).

[b] Adjustment factor for cooking losses (EFH Table 13-5, Percent Weight Losses from Preparation of Various Meats).

[c] Adjustment factor for postcooking losses (EFH Table 13-5, Percent Weight Losses from Preparation of Various Meats).

Figure 6-2. Sample calculation for adjusted beef ingestion rate (0- to 5-yr-old age group).

Fruits and Vegetables. Only "exposed" fruits and vegetables and root vegetables were included in the exposure assessment. To obtain a complete set of unadjusted ingestion rates for all age group/receptor population combinations (for exposed fruit, exposed vegetables, and root vegetables), the specific derivations presented in Table 6-13 were completed.

Table 6-13. Required Derivations for Age Group/Receptor Population Unadjusted Ingestion Rates for Fruits and Vegetables

Age Group (yr)	Commodity	Derivation
0-5	Root and exposed vegetables	Age group averaging
0-5	Exposed fruit	Use of 3- to 5-yr-old age group ingestion rate to represent population in absence of 1- to 2-yr-old ingestion rate
>19	Root vegetables, exposed fruit, and exposed vegetables	Age group averaging

Once the UIR was established for each age group, the values were adjusted to account for losses (i.e., preparation, cooking, and postcooking losses). Based on the data available in the 1997 EFH for deriving adjusted ingestion rates, the following specific fruit/vegetable types were used to represent each of the broader fruit/vegetable categories when calculating the adjustment factors:

- Exposed fruits–apples, pears, peaches, and strawberries
- Root vegetables–beets, carrots, onions, and potatoes
- Exposed vegetables–asparagus, broccoli, cabbage, cucumber, lettuce, okra, peppers, snap beans, and tomatoes.

Because vegetable- and fruit-specific data (e.g., specific values for cabbage and apples) were available for characterizing cooking losses and preparation losses and for the conversion of wet weight to dry weight, these data had to be aggregated to represent the larger groupings used in this analysis (i.e., exposed fruit, root vegetables, and exposed vegetables). To derive factors for the larger groupings, fruit- and vegetable-specific values were combined with weighting factors reflecting the consumption rates for each fruit and vegetable type by the survey population of interest (i.e., mean values for the "total" population obtained from 1997 EFH 87/88 NFCS data tables: Intake of Homegrown *[specific fruit/vegetable]*). Table 6-14 presents the data used to derive the values for cooking losses and preparation losses and for the conversion of wet weight to dry weight for exposed fruit, root vegetables, and exposed vegetables. Loss factors were then applied as shown in Equation 6-2 to generate mean adjusted ingestion rates for home-produced exposed vegetables, root vegetables, and exposed fruits:

Dietary Ingestion Rates for Exposed Vegetables, Root Vegetables, and Exposed Fruits

$$AIR_{\text{exposed fruit; root vegetable; exposed vegetable}} = (UIR)\ (1\text{-}CLPL)\ (1\text{-}WW) \qquad (6\text{-}2)$$

Parameter	Definition
$AIR_{\text{exposed fruit, root vegetable, exposed vegetable}}$	Adjusted ingestion rate for exposed fruit, root vegetable, or exposed vegetable (value used in HWC risk analysis)
UIR[a]	Unadjusted ingestion rate (see Table 6-10)
CLPL	Cooking or preparation losses
WW	Fraction of wet weight that is water

[a]UIR values had to be extrapolated for several age group/fruit or vegetable type combinations.

Table 6-14. Derivation of Consumption-Adjusted Loss Values and Dry Weight Values for Fruits and Vegetables

Exposed Fruits Paring/Preparation Loss										
	Apples	Pears	Peaches	Strawberries						TOTAL
Mean consumption (g/kg-d)	1.19	0.937	1.67	0.652						4.4
Crop-specific consumption/total consumption	0.267475837	0.210609126	0.375365251	0.146549786						
Paring and preparation loss (%)	22	22	24	10						
Adjusted paring and preparation loss (%)	5.88446842	4.63	9.01	1.465497865						21.0
Root Vegetables Mean Net Cooking Loss										
	Beets	Carrots	Onions	Potatoes						TOTAL
Mean consumption (g/kg-d)	0.512	0.438	0.296	1.66						2.9
Crop-specific consumption/total consumption	0.176187199	0.150722643	0.101858224	0.571231934						
Paring and preparation loss (%)	27.71	19.13	4.54	-21.83						
Adjusted paring and preparation loss (%)	4.882147281	2.88	0.46	-12.46999312						-4.2
Exposed Vegetables Mean Net Cooking Loss										
	Asparagus	Broccoli	Cabbage	Cucumber	Lettuce	Okra	Peppers	Snap Beans	Tomatoes	TOTAL
Mean consumption (g/kg-d)	0.559	0.42	1.03	1.02	0.387	0.391	0.239311152	0.8	1.18	6.0
Crop-specific consumption/total consumption	0.092759897	0.069694377	0.170917162	0.169257772	0.06421839	0.064882146	0.039711051	0.132751194	0.195808011	
Paring and preparation loss (%)	22.830	13.830	11.250	17.500	21.630	11.830	13.400	18.000	15.130	
Adjusted paring and preparation loss (%)	2.118	0.964	1.923	2.962	1.389	0.768	0.532	2.390	2.963	16.0
Exposed Fruit Mean Moisture Content										
	Apples	Pears	Peaches	Strawberries						TOTAL
Mean consumption (g/kg-d)	1.19	0.937	1.67	0.652						4.4
Crop-specific consumption/total consumption	0.267475837	0.210609126	0.375365251	0.146549786						
Mean moisture content (%)	83.930	83.810	87.660	91.570						
Adjusted moisture content (%)	22.449	17.651	32.905	13.420						86.4
Root Vegetables Mean Moisture Content										
	Beets	Carrots	Onions	Potatoes						TOTAL
Mean consumption (g/kg-d)	0.512	0.438	0.296	1.66						2.9
Crop-specific consumption/total consumption	0.176187199	0.150722643	0.101858224	0.571231934						
Mean moisture content (%)	90.900	87.585	91.530	71.200						
Adjusted moisture content (%)	16.015	13.201	9.323	40.672						79.2
Exposed Vegetables Mean Moisture Content										
	Asparagus	Broccoli	Cabbage	Cucumber	Lettuce	Okra	Peppers	Snap Beans	Tomatoes	TOTAL
Mean consumption (g/kg-d)	0.559	0.42	1.03	1.02	0.387	0.391	0.239311152	0.8	1.18	6.0
Crop-specific consumption/total consumption	0.092759897	0.069694377	0.170917162	0.169257772	0.06421839	0.064882146	0.039711051	0.132751194	0.195808011	
Mean moisture content (%)	92.040	90.445	92.575	96.050	95.890	89.745	93.735	89.745	93.950	
Adjusted moisture content (%)	8.538	6.304	15.823	16.257	6.158	5.823	3.722	11.914	18.396	92.9

Note: Mean moisture content was selected as the most likely form (i.e., raw vs. cooked) for consumption. If both forms were equally likely to be consumed, the raw and cooked moisture contents were averaged.

Figure 6-3 presents a sample calculation for the exposed vegetable adjusted ingestion rate for the 6- to 11-yr-old age group, produce farmer. Final adjusted ingestion rates are presented in Table 6-11.

Use unadjusted ingestion rate (UIR) for 6- to 11-yr-old age group to generate adjusted ingestion rate (AIR) for exposed vegetables that will be used in risk equations (this air accounts for cooking and postcooking losses):

$$AIR_{exposed\ vegetable} = (1.4\ g/kg\text{-}d)^{a}(1\text{-}0.16)^{b}(1\text{-}0.93)^{c} = 0.082\ g/kg\text{-}d$$

[a]UIR for exposed vegetables for 6- to 11-yr-old age group (EFH Table 13-63, Consumer-Only Intake of Homegrown Exposed Vegetables (g/kg-d)).

[b]Adjustment factor for cooking losses (see Table 6-14).

[c]Adjustment factor for wet weight fraction (see Table 6-14) .

Figure 6-3. Sample calculation for exposed vegetable adjusted ingestion rate (6- to 11-yr-old age-group, produce farmer).

6.3.1.8 <u>Dairy (Milk and Eggs)</u>. For both milk and eggs, mean unadjusted ingestion rates were obtained from 1997 EFH 87/88 NFCS tables for home-produced dairy and eggs, respectively. To obtain a complete set of unadjusted ingestion rates for all age group/receptor population combinations (for milk and eggs), the specific derivations indicated in Table 6-15 were completed.

Table 6-15. Required Derivations for Age Group/Receptor Population Unadjusted Ingestion Rates for Dairy Products

Age Group (yr)	Product	Derivation
0-5, 6-11, 12-19, >19	Milk	General population extrapolation
0-5, 6-11, 12-15	Eggs	General population extrapolation
>19	Eggs	Age group averaging

Figure 6-4 presents a sample calculation for unadjusted milk ingestion rate for the 0- to 5-yr-old age group commercial beef farmer using the general population extrapolation.

Step 1. Extrapolate unadjusted ingestion rate (UIR) for 0- to 5-yr-old age group (87/88 NFCS data tables do not have home-produced dairy ingestion rate for this age group):

$$UIR = (14.0\ g/kg\text{-}d)^{a}(60\ kg)^{b}[(347\ g/d)/(224\ g/d)]^{c} = 1301.3\ g/d$$

[a] Mean home-produced ingestion rate for all ages combined (EFH Table 13-28, Consumer-Only Intake of Home-Produced Dairy (g/kg-d)).

[b] The body weight value that should be used in converting ingestion rates for the overall sampled population (i.e., all ages combined) into units of g/d.

[c] Ratio of milk ingestion rate (not home-produced) for 0- to 5-yr-old age group to that of entire sampled population (EFH Table 11-13, Mean Daily Intake of Meat and Dairy Product Per Individual in a Day for USDA 1977-78, 87-88, 89-91, 94, and 95 Surveys).

Step 2. Convert the UIR for the 0- to 5-yr-old age group into units of g/kg-d:

$$UIR = 1301.3\ g/d/14.3\ kg^{d} = 90.9\ g/kg$$

[d] Body weight for 0- to 5-yr-old age group (see Table 6-4).

Figure 6-4. Sample calculation for unadjusted milk ingestion rate (0- to 5-yr-old age group, farmer).

Once the UIR was established for each age group, the values were adjusted to account for the fraction of total dairy consumed that is milk only. Ingestion rates for home-produced dairy obtained from the 87/88 NFCS table are in terms of total dairy products. Therefore, these values had to be converted to equivalent ingestion rates for milk. The 87/88 NFCS provides ingestion rates for the general population for both milk and total dairy products. These values were used to derive a **fraction of total dairy ingestion that is milk** adjustment factor (i.e., the “FM” factor presented in Equation 6-3). In deriving the ingestion rates for milk, it was assumed that the ratio of milk ingestion (EFH Table 11-13, Mean Dairy Product Intakes Per Individual in a Day, by Sex and Age (g/day as consumed) for 1987-1988) to total dairy product ingestion (EFH Table 11-8, Mean Dairy Product Intakes Per Individual in a Day, by Sex and Age for 1987-1988) for the overall population would be representative of the ratio of milk ingestion to total dairy ingestion for those individuals who consume home-produced dairy. The extrapolated dairy ingestion rates for the younger age groups appear high in comparison to the values for the total population provided in EFH Table 13-28. Although these values appear to be high, younger age groups are anticipated to consume significantly more milk than are adults (on a g/kg-d basis), and therefore this result is not unexpected. Equation 6-3 was used to generate mean adjusted ingestion rates for home-produced milk. This step resulted in the derivation of the age-specific AIR presented in Table 6-10.

Dietary Ingestion Rates for Milk

$$AIR_{milk\ or\ egg} = (UIR)(FM)_{dairy\ only} \quad (6\text{-}3)$$

Parameter	Definition
AIR_{milk}	Adjusted ingestion rate for milk (value used in HWC risk analysis)
UIR[a]	Unadjusted age-group-specific ingestion rates
FM	Fraction of total dairy ingestion that is milk

[a]UIR values had to be extrapolated for several age-group/fruit or vegetable combinations.

Figure 6-5 presents a sample calculation of an adjusted ingestion rate for milk. Final ingestion rates for home-produced milk and eggs that were used in the HWC risk analysis are presented in Table 6-10.

Use unadjusted ingestion rate (UIR) for 0- to 5-yr-old age group to generate adjusted ingestion rate (AIR) for milk that will be used in risk equations (this AIR accounts for the fraction of total dairy ingestion that is milk):

$$AIR_{milk} = (90.9\ g/kg\text{-}d)^{a}(0.77)^{b} = 70.0\ g/kg\text{-}d$$

[a] UIR for milk for 0- to 5-yr age group (see Figure 6-4).
[b] Adjustment factor for the fraction of total dairy ingestion that is milk (EFH Tables 11-8 and 11-13).

Figure 6-5. Sample calculation for milk adjusted ingestion rate.

6.3.1.9 <u>Fish</u>. Fish ingestion rates were derived for three different receptors: (1) subsistence fishers who obtain a significant fraction of their total dietary intake from self-caught freshwater fish, (2) recreational fishers who obtain a portion of their dietary fish intake from recreationally caught freshwater fish, and (3) subsistence farmers who obtain a portion of their dietary intake from freshwater fish from farm ponds.

To use the fish ingestion values described in this section to estimate human health impacts, the ingestion rates had to be apportioned between trophic level 3 and 4 fish. Based on a review of all the study data available for characterizing fish ingestion, the Chemrisk 1991 study, summarized in the 1997 EFH in Table 10-66, Total Consumption of Freshwater Fish Caught by All Survey Respondents During 1990 Season, was identified as the only study that presented sufficient data for determining the fraction of fish ingestion that was trophic 3 versus trophic 4. The ratios of **trophic 3 fish ingestion to total fish ingestion** and **trophic level 4 fish ingestion**

to total fish ingestion (both ratios reflecting grams of fish consumed) were calculated using data from this table. These ratios (0.64 for trophic level 4 fish and 0.36 for trophic level 3 fish) were used to apportion the recreational and subsistence fish ingestion rates presented above into trophic level 3-specific and trophic level 4-specific ingestion rates (trophic-level-specific fish ingestion rates are not presented in Table 6-10 but were integrated into the HWC risk analysis equation structure).

The sources of the data and the rationale behind the fish ingestion rates identified for each of the three receptors assessed for exposure to HWC (including all four age groups for each receptor) are described below. Final ingestion rates for fish are presented for each of the three receptors in Table 6-3.

No adjustments for losses were made to the fish UIRs. Cooking and postcooking losses were not factored into the derivation of final fish ingestion rates because the data used to establish ingestion rates for these receptor populations already accounted for these losses (i.e., the data used for the recreational fisher and the subsistence farm household were based on measuring an "edible" portion, while the data used for the subsistence fisher were based on measuring the "serving size").

Subsistence Fishers. Chapter 10 of the 1997 EFH recommends a mean ingestion value for the adult subsistence fisher of 70 g/d. In addition, the Columbia River Inter-Tribal Fish Commission (CRITFC, 1994), as cited in 1997 EFH, provides a subsistence value for the 0- to 5-yr-old age group of 19.6 g/d. However, the 1997 EFH does not provide subsistence ingestion rates for either the 6- to 11-yr-old age group or the 12- to 19-yr-old age group. The EFH-recommended ingestion rates of 70 and 19.6 g/d were divided by the age-group-specific body weights for the adult and 0- to 5-yr-old receptors used in the HWC to produce ingestion rates in grams/kilogram per day. These resulting ingestion rates were applied to the 12- to 19-yr-old and 6- to 11-yr-old age groups, respectively.

Recreational Fishers. To obtain a complete set of unadjusted ingestion rates for all age group/receptor combinations, the derivations presented in Table 6-16 were performed using the West et al. (1989) data referenced in Table 6-9.

Table 6-16. Required Derivations for Recreational Fisher Unadjusted Ingestion Rates for Fish

Age Group (yr)	Ingestion rate (g/kg-d)	Ingestion rate (g/d)	Derivation
0-5	0.37	5.3	Use of 1- to 5-yr-old age group ingestion rate
6-11	0.28	8.6	Use of 6- to 10-yr-old age group ingestion rate
12-19	0.15	8.7	Backcalculation using 1- to 5-, 6- to 10-, and 1- to 20-yr-old sample sizes and rates
>19	0.16	11.5	Age group averaging

An ingestion rate for the 12- to 19-yr-old age group was backcalculated using known population counts and ingestion rates for the 1- to 5-yr-old, 6- to 10-yr-old, and 1- to 20-yr-old age groups. The steps involved in this backcalculation were as follows:

1. Assume that the 11- to 20-yr-old age group is representative of the 12- to 19-yr-old age group.

2. Subtract the sample size for the 1- to 5-yr-old and 6- to 10-yr-old age groups from the total (1- to 20-yr-old) population to determine the sample size for the 11- to 20-yr-old age group.

3. Sum the sample size times the intake rate (g/d) for the three age groups (1- to 5-, 6- to 10-, and 11- to 20-yr-old) and set it equal to the sample size times the intake rate for the entire 1- to 20-yr-old population. In this equation, the intake rate for the 11- to 20-yr-old age group is unknown.

4. Solve for the 11- to 20-yr-old age group intake rate (g/d).

5. Divide by the 12- to 19-yr-old age-group-specific body weight to produce a recreational fish intake rate in g/kg-d for the 12- to 19-yr-old age group.

The mean value recommended in the EFH for freshwater recreational fish consumption is 8 g/d. This recommended value is in line with the derived fish consumption rates for the various age groups of the recreational fisher, as shown in Table 6-16. Although the ingestion rate (11.5 g/d or 0.16 g/kg-d) for the >19-yr-old age group is greater than the recommended recreational ingestion rate provided in the 1997 EFH (8 g/d, or 0.11 g/kg-d), it was selected for use in the HWC risk analysis because it is based on the same data used to derive recreational ingestion rates identified for the other three age groups (i.e., the West et al., 1989, data) and it is generally in line with the other three values (meaning it follows the trend in magnitude seen with the values for the other three age groups).

Subsistence Farmers. Subsistence farmers were considered to be engaging in recreational fishing in farm ponds. Therefore, the unadjusted ingestion rates calculated for the recreational fisher population were applied to the subsistence farmer population as well. The 87/88 NFCS data for self-caught fish were not used for subsistence farmers (even though those data were used for every other food type) because the consumption rates reported in the EFH are heavily weighted toward marine fish and, therefore, are much higher than consumption rates would be for freshwater fish alone.

6.3.2 Distribution of Variability for Selected Parameters

6.3.2.1 General Issues. The calculations of risk performed for this analysis involve several parameters and modeling results characterized by both geographic and intersubject variability. These are:

- Interfacility variability of the source terms for facilities within a category

- Interfacility and intersector variability in the dispersion factors
- Variability in the exposure factors between different receptor populations
- Interindividual variability in the exposure factors for a given receptor population
- Interindividual variability in the sensitivity to carcinogens and noncarcinogens in the different receptor populations.

There is no variability of the source term within the sectors surrounding a given facility, although there is variability of the source term between facilities. This component of variability was accounted for in the HWC analysis through use of facility-specific source terms. There is variability of the dispersion factor across the sectors surrounding a given facility (e.g., the dispersion coefficient or relationship between emission rate and concentration). This component of variability was accounted for in the HWC analysis through use of sector-specific dispersion factors that are a function of distance from the source and direction. In addition, there is geographic variability of the dispersion factor within a sector, although the HWC analysis eliminated that variability by averaging air concentration over the geographic region of a sector. With respect to both source terms and dispersion factors, therefore, variability was accounted for by the use of parameter values that are specific to both the facility (source terms) and sector (dispersion factors). Variability in exposure factors between receptor populations was incorporated into the analysis through the development of separate exposure estimates for different receptor populations (beef farmer, dairy farmer, and fisher).

The exposure factors and sensitivities, however, are variable across individuals within a given receptor population in a given sector. This intersubject variability was reflected through use of probability density functions for each of these parameters. The variability of sensitivity of individuals to carcinogens and noncarcinogens is not readily characterized quantitatively at present and, therefore, was not represented as a probability density function in this analysis. Instead, this intersubject variability of sensitivity is reflected in the use of cancer slope factors and hazard indices (e.g., the ratio of dose for an individual over the reference dose) that are derived using a methodology designed to reflect characteristics of the more sensitive segments of an exposed population (e.g., through the use of confidence limits that tend to apply to individuals at the greatest risk following a unit exposure).

Variability of exposure factors within a receptor population was, however, incorporated formally into the HWC analysis. The exposure factor, EF, converts the concentration in an environmental medium to the average daily rate of intake (ADRI). If C is the concentration in the environmental medium, the ADRI may be found by:

$$ADRI = C \bullet IR \bullet ED / (AT \bullet BW) \qquad (6\text{-}4)$$

where

IR = intake rate of the environmental medium (e.g., water or food)

ED = exposure duration

AT = averaging time (taken to be 70 years for carcinogens and equal to ED for noncarcinogens)

BW = body weight or mass.

The exposure factor, EF, may be found by dividing the right-hand side of the above equation by C, or

$$EF = (IR/BW) \bullet (ED/AT) \qquad (6\text{-}5)$$

The intersubject variability of exposure factors used in the HWC analysis incorporates three primary components:

- Variability in the ingestion rate per unit body mass for beef, milk, and fish for both dioxin and mercury exposures. This is the ratio of IR over BW. Separate distributions were developed for the three receptor populations and for age categories within those receptor populations.

- Variability in exposure duration, ED, for the beef and milk ingestion (i.e., for dioxin where cancer risks are dominant) but not for fish ingestion (i.e., not for mercury where noncancer risks are dominant and for which exposures are not time-averaged - ED and AT cancel). Separate distributions were developed for the two relevant receptor populations and for age categories within those receptor populations.

- Variability in a correction factor for crossing age groups. This factor accounts for the fact that the central tendency risk estimates presuppose that values for EF are constant during the period of exposure for an individual beginning exposure in a given age group, whereas the aging of an individual may cause movement between age groups and their associated exposure factors during the period of exposure. For example, if an individual began exposure in the 0- to-5-yr-old age group and the exposure continued for 7 years, the exposure parameters used in the central tendency calculations were those of the 0- to 5-yr-old age group for all 7 years of exposure, despite the fact that two of those years were spent in the 6- to 11-yr-old age group.

Note that there is no variability in the averaging time, AT, since this is a matter of the definition of the ADRI.

These three sources of variability in the exposure factors (IR/BW, ED, and the age correction factor or ACF) were analyzed separately using available data sets. They then were combined analytically for a composite variability distribution for EF specific to each receptor population and age category. Separate discussions of the method for developing probability density functions (PDFs) and their associated cumulative distribution functions (CDFs) are provided in Section 6.3.2.4 for each of the exposure parameters.

In generating the PDFs for the parameters, a choice was made to use an a priori parametric form rather than an empirical (nonparametric) equation. In selecting the appropriate form for the PDF, several criteria were examined:

- Distributional form must provide a reasonable visual fit to the available data over the relevant range
- Distributional form must provide reasonable quantitative agreement with the measured values at the upper ends of the CDF (i.e., the 95th percentile)
- Distributional form must compare well to alternative forms with respect to quantitative goodness-of-fit measures (e.g., p values)
- Distributional form must have a precedent for reliable use in human health risk analysis
- Distributional form must be mathematically tractable for repeated use in the overall analysis.

Any choice between marginally different distributions (i.e., distributions with marginally different goodness-of-fit measures) should reflect the sensitivity of final risk estimates to the inability to select reasonably between these distributions.

A comparison was made of the best-fitting curves from each of several distributions used commonly in risk analysis: the lognormal, the gamma, the Weibull, and the generalized gamma. As a formal goodness-of-fit test, the data sets discussed below were examined under Chi square and p-value tests using the gamma, lognormal, Weibull, and generalized gamma models. In addition, a simpler square of the residuals examination was performed for the lognormal, beta, and normal distributions. For the residuals test, the lognormal distribution provided the smallest residual for all of the parameters fit; this test, however, is the least sensitive.

The more germane comparison of the different distributional fits is the p-value shown in Table 6-17. In this table, the p-value represents the probability that the deviations of the data from the parametric form of the distribution can be accounted for by random variations in those data. Therefore, a higher p-value indicates a better agreement between the data and selected PDF (using the best-fitting parameter values for each distribution). In considering this more rigorous p-value criterion, the lognormal distribution provided either the best, or an approximately equivalent, p value for the majority of the data sets considered, as shown in Table 6-17. This was particularly true for the IR/BW parameter values for beef ingestion (three age groups considered) and fish ingestion and for the ACF factor.

Lognormal distributions are used most commonly in human health risk analysis and have been found historically to provide a good fit to data on environmental and biological parameters (Morgan and Henrion, 1990). As shown in Figures 6-6 through 6-10 and discussed later with respect to each separate exposure parameter, the lognormal distribution provided a good visual fit to the relevant data contained in the 1997 *Exposure Factors Handbook* (U.S. EPA, 1997a) and provided reasonable predictions with respect to the upper percentiles of the data distribution.

Table 6-17. p Values for a Priori PDFs Considered in This Analysis

Parameter	p Value			
	Gamma	**Lognormal**	**Weibull**	**General Gamma**
Beef, 6-11	0.17	0.53	0.091	0.314
Beef, 12-19	0.57	0.45	0.46	0.42
Beef, adult	0.001	0.001	0.001	0.001
ACF	0.20	0.26	0.043	0.13
ED, farmers[a]	<0.1	<0.1	<0.1	<0.1
Milk IR/BW[a]	0.4	0.1	0.4	0.56
Fish, all ages	<0.1	<0.1	<0.1	<0.1

[a] These are small data sets. Use of p values is significantly less reliable for these small sets.

It also is a mathematically tractable form for use in repeated analyses, being characterized by a median (50th percentile or central tendency value) and a geometric standard deviation (GSD), and is particularly well suited to models in which parameter values are multiplied. The associated probability density function for a median of M and a geometric standard deviation of S is given as:

$$PDF(x) = \exp\{-(\ln x-\ln M)^2/(2\ln^2 S)\}/\{2p(x\ln S)\} \qquad (6\text{-}5)$$

In the lognormal distribution, 68 percent of the values are contained in the area under the PDF defined by a lower and upper limit. The lower limit is obtained by dividing the median by the GSD, and the upper limit is obtained by multiplying the median times the GSD. (Note: In the normal distribution, this 68 percent interval is defined by the mean minus the standard deviation and the mean plus the standard deviation). For the lognormal distribution, 95 percent of the values are contained in the area defined at the lower end by the median divided by the square of the GSD and at the upper end by the median times the square of the GSD. (Note: In the normal distribution, this same interval is defined by the mean minus twice the standard deviation, and the mean plus twice the standard deviation; equivalent additive operations in the Normal distribution become multiplicative operations in the lognormal distribution.) The GSD of the lognormal distribution, in turn, equals the ratio of the 84th percentile of the distribution divided by the 50th percentile (see Crawford-Brown, 1997) or the 50th percentile divided by the 16th percentile (note that the 16th to 84th percentile contains 68 percent of the values of the distribution).

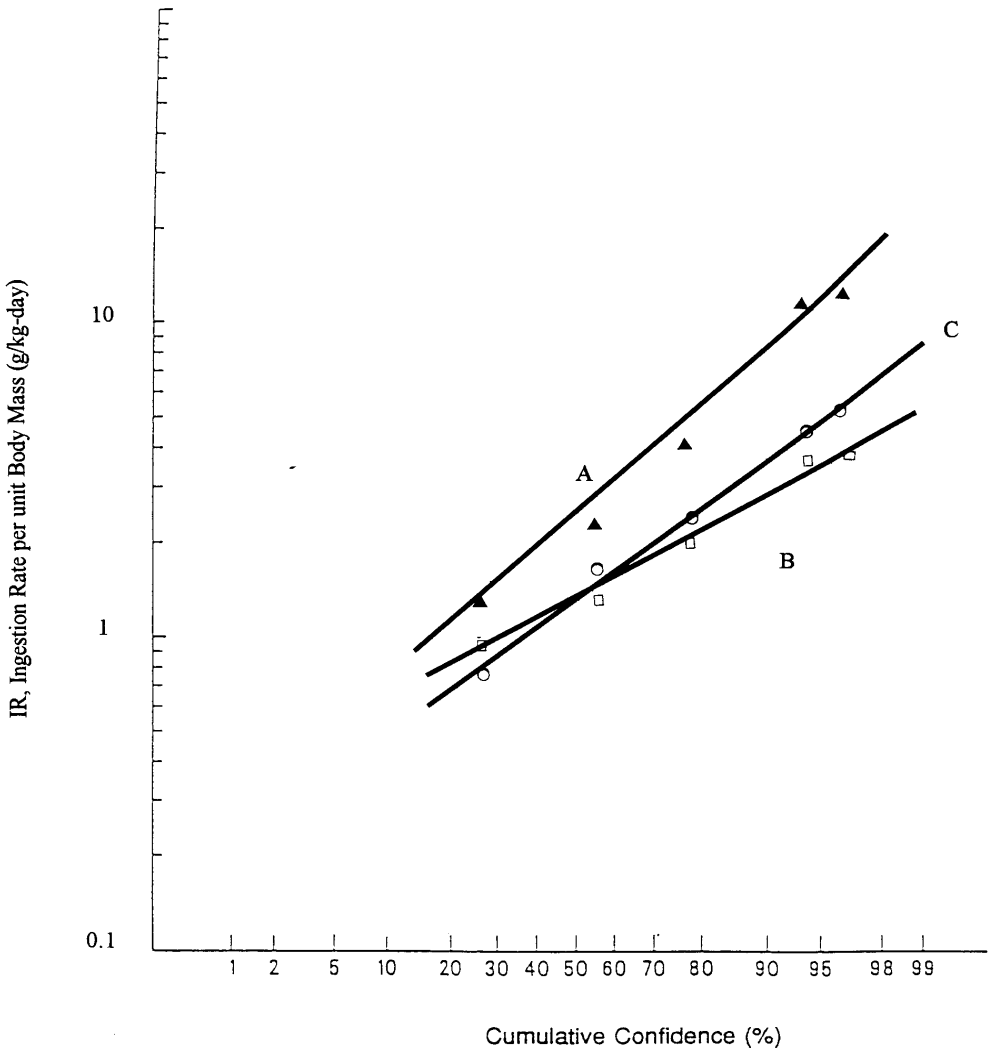

Figure 6-6. Variability of the ingestion rate per unit body mass for home-produced beef. Curve A is for the 0- to 5-yr-old and 6- to 11-yr-old age groups; curve B is for the 12- to 19-yr-old age group; curve C is for adults. The cumulative confidence refers to the fraction of results with a value less than or equal to that shown on the y-axis. The GSDs (equal to the ratio of the 84th to the 50th percentile) are used in this analysis.

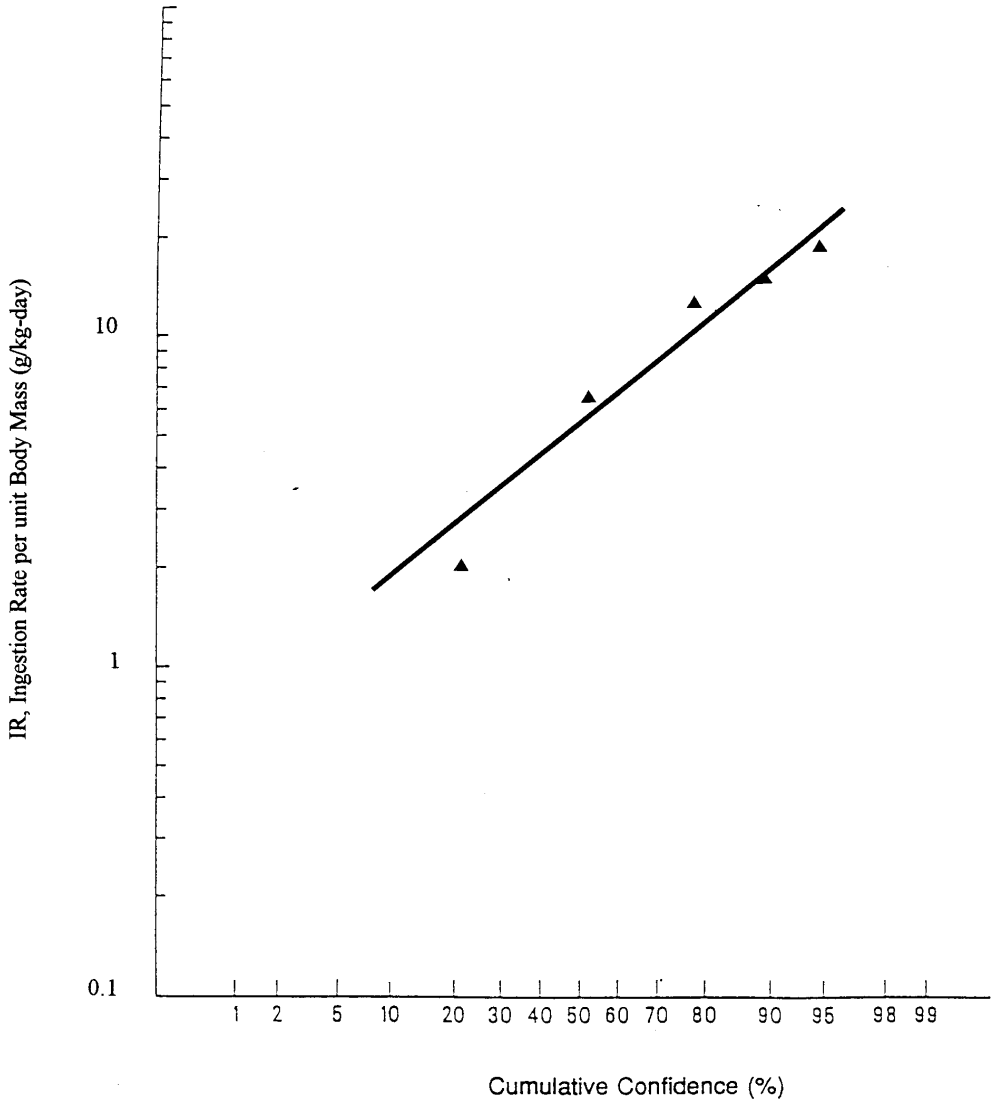

Figure 6-7. Variability of the ingestion rate per unit body mass for home-produced milk. The curve is for all age groups. The cumulative confidence refers to the fraction of results with a value less than or equal to that shown on the y-axis. The GSDs (equal the ratio of the 84th percentile to the 50th percentile) are used in this analysis.

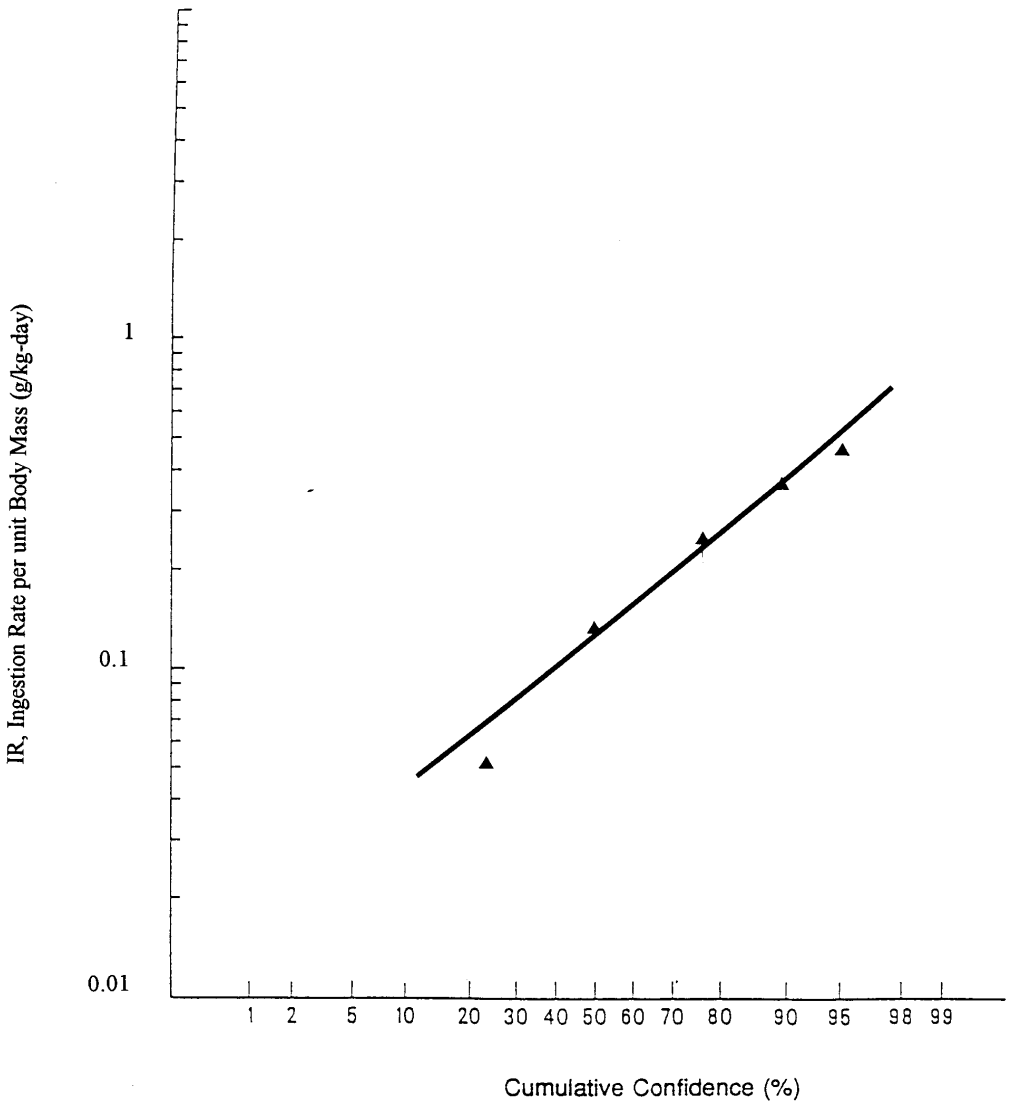

Figure 6-8. Variability of the ingestion rate per unit body mass for the recreational fisher. The curve is for all age groups. The cumulative confidence refers to the fraction of results with a value less than or equal to that shown on the y-axis. The GSDs (equal the ratio of the 84th percentile to the 50th percentile) are used in this analysis.

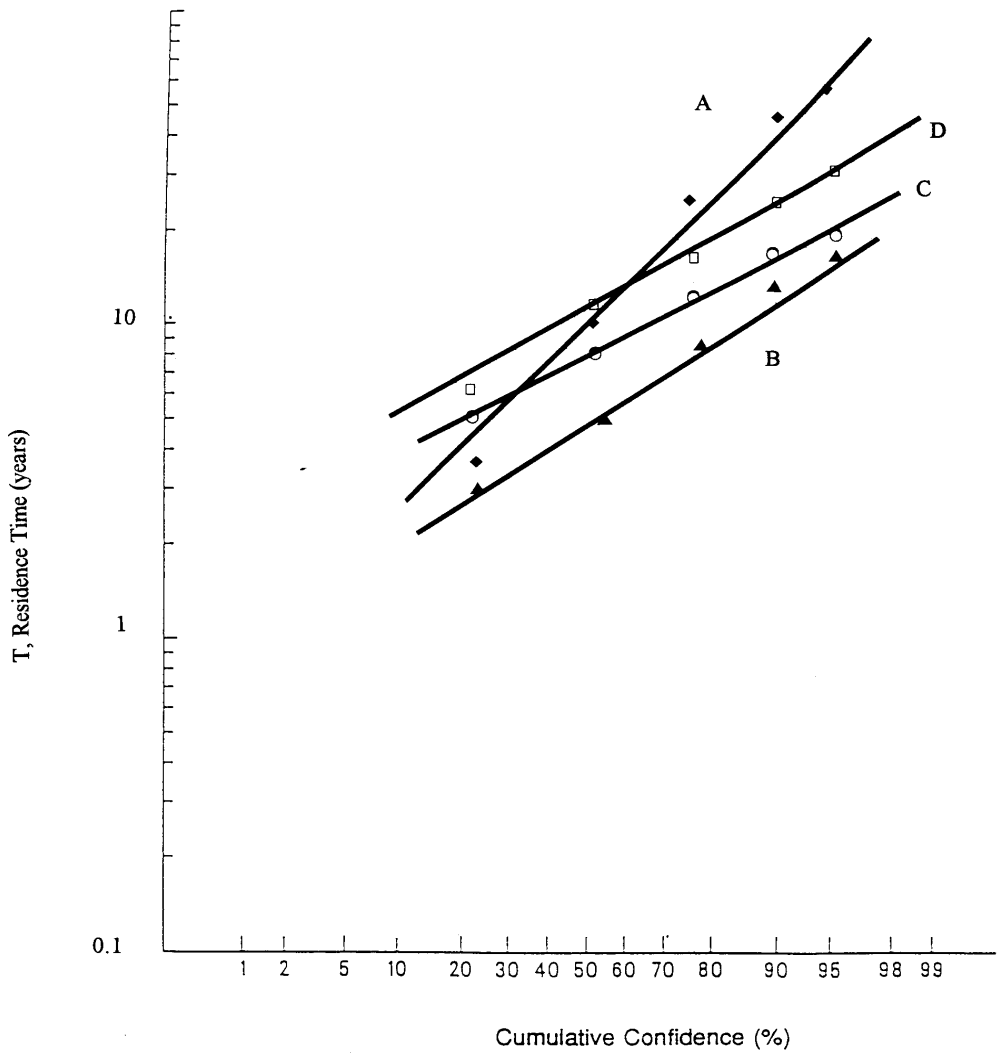

Figure 6-9. Variability of the residence times (occupancy periods) for the farming and nonfarming populations. Curve A is for the farming population, all ages; curve B is for the nonfarming population 0 to 5 years. Curve C is for the nonfarming population, 6 to 11 and 12 to 19 years, and curve D is for the nonfarming population (adults). The cumulative confidence refers to the fraction of results with a value less than or equal to that shown on the y-axis. The GSDs (equal the ratio of the 84th percentile to the 50th percentile) are used in this analysis.

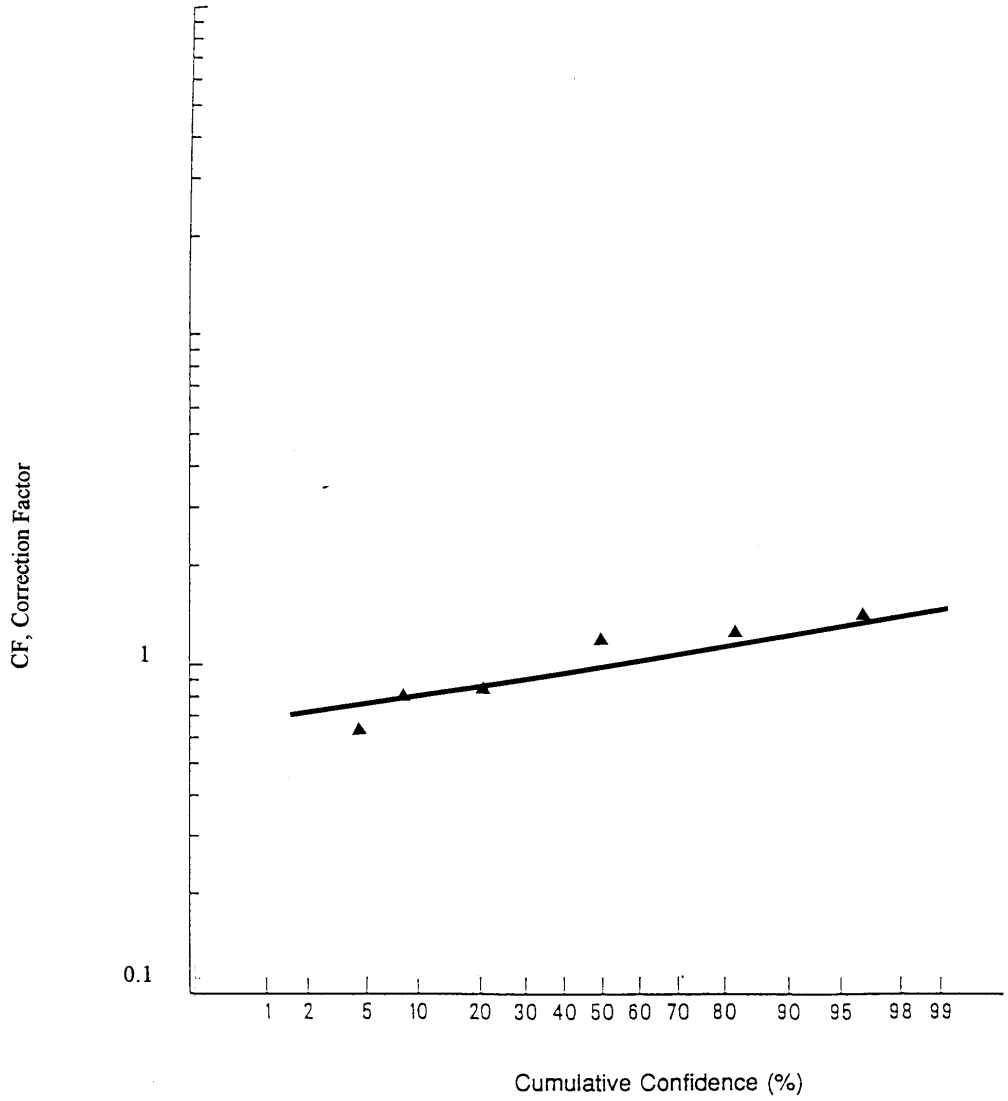

Figure 6-10. Variability of the correction factor for crossing age groups. There is no significant difference for the three exposure pathways, so the same distribution is used. The curve applies to the 0- to 5-, 6- to 11- and 12- to 19-yr-old age groups only. The cumulative confidence refers to the fraction of results with a value less than or equal to that shown on the y-axis.

To consider the issue of whether the marginally better p values provided by the gamma and generalized gamma indicated a need to switch to these for at least the milk ingestion rate, a sensitivity analysis was performed considering the first three distributions (the gamma, lognormal, and Weibull; the generalized gamma can be simulated in the Monte Carlo software used here but only with significant effort that was not justified by its marginal improvement over the gamma distribution). The sensitivity analysis was conducted for the product of the milk ingestion rate and farm occupancy factor, the two parameters for which there was even an issue about switching from the lognormal. (For the other parameters, there either was little difference in the p-values, or none of the p-values were reliable due to small sample size; an example of the latter is the exposure duration.) The analysis simulated a four-sector geographic region. The distributions for the milk ingestion rate and occupancy period were taken from the best fits to the three distributions above. The medians for the milk ingestion rate then were adjusted for the four sectors to reflect intersector variability of concentrations in milk. One sector was multiplied by 0.1, one by 0.5, one by 1.0, and one by 5.0. Equal populations were placed into each of the four sectors. A random sample of 5,000 individuals then was drawn using Monte Carlo (CrystalBall)® methods. The 95th, 97th, and 99th percentiles of the resulting population distribution were obtained, and the procedure repeated over the three candidate distributions. The ratio of the percentile value obtained from a given distributional form over the percentile value from the lognormal form was calculated. This produces a ratio of 1.0 for the lognormal distribution itself. The resulting ratios are reported in Table 6-18.

It may be noted from the sensitivity analysis that the sensitivity of the upper percentiles in an aggregated population (aggregated across sectors) is not large when at least four sectors are present. There are significantly more than four sectors in the analyses developed for this study, so it is unlikely that the selection of a distribution other than the lognormal for the factors where lognormal did not provide the best p value will be significant. Given the results of this sensitivity analysis, and that the lognormal distribution (an equally valid method of selecting fits for data of this quality) is a good visual fit; that it provided the best, or an equally good, p value in five of the seven parameters considered; and that it can retain lognormal properties when one takes the product of lognormally distributed parameters, it was clear that the lognormal distribution was the best choice for use throughout this analysis.

Table 6-18. Ratio of the 95th, 97th, and 99th Percentile Values as Predicted by the Best-Fitting Gamma, Lognormal, and Weibull Distributions, Relative to the Lognormal Predictions

Percentile	Ratio to Lognormal Prediction		
	Gamma	**Lognormal**	**Weibull**
95th	0.85	1.00	1.16
97th	0.90	1.00	1.22
99th	0.83	1.00	1.25

6.3.2.2 <u>Truncation of Distributions</u>. It is common in fitting variability data to find that the distributions are partially truncated at the lower and upper ends of the distribution, with truncation usually at between 2 and 3 GSDs (Crawford-Brown, 1997). This is due to physical and biological limitations on the range of values that can occur. This general result was found to hold in the HWC analysis, as the data from the 1997 EFH could be fitted appropriately by a lognormal distribution out to approximately 2 to 3 GSDs around the median. Beyond that range, the lognormal distribution was inaccurate (as are all analytic, a priori, distributional forms) because the probability density for the data outside this region was significantly less than that predicted by the distribution. In the HWC analysis, truncation for sampling was at 3 GSDs; values beyond these limits were rejected and resampled. This truncation is not shown in Figures 6-6 through 6-10. To reflect truncation, the reader can follow the displayed curves to approximately the 1 percent and 99 percent values at the two tails and then draw lines horizontal to the X-axis from these two points.

Note: Truncation does not introduce inaccuracies into the composite risk or HQ variability distribution for the population. As discussed, truncation is a feature of the underlying data on which the parameter variability distributions are developed and is not introduced a priori into the analysis. Failure to truncate the lognormal distributions would introduce inaccuracies by artificially increasing the likelihood of parameters being selected at values more than 3 GSDs from the median.

6.3.2.3 <u>Correlated Parameters</u>. In developing the aggregate variability distribution resulting from the product of the ingestion rate per unit body mass, the exposure duration, and the ACF, correlations between parameters must be considered. The three factors considered in this variability analysis are statistically independent (i.e., there is no correlation). Although ingestion rate (IR) and body mass (BW) are correlated, this analysis used a data set in which IR and BW were determined for each individual in the population, and the ratio (IR/BW) calculated based on values of IR and BW specific to that individual. As a result, there was no need to select randomly from a distribution of values of IR, and then from a distribution of values of BW, and obtain the ratio IR/BW (in which case the issue of correlation between IR and BW would have arisen). The approach used here fully incorporates any correlation between IR and BW. The exposure duration (ED) is correlated with the ratio IR/BW, since both quantities are functions of age (in other words, both ED and IR/BW depend upon age and, therefore, should be correlated; knowing an individual's age should provide information on the appropriate value of IR/BW). Once a range of ages is selected for analysis, however, as was done in this study by focusing all exposure calculations on specific age groups rather than on the entire population, there is no reason to suspect correlation between ED and the ratio IR/BW. Although the available data do not allow a test of this assumption (that ED and IR/BW are uncorrelated) since there has been no study in which ED and IR/BW are measured for the same individual, it appears reasonable to assume they are not correlated. A correlation would require that an individual's exposure duration (length of residence) be in some way related to the ratio of their intake rate over body mass. This would be the case only if, for example, durations between changes of address correlate with IR/BW. Such a correlation might exist if there were systematic differences in IR/BW and in ED for different subpopulations such as farmers, fishers, and the general population, but that possibility was dealt with in the current analysis by separating these subpopulations into different exposure groups. Still, the possibility remains that there is some

residual correlation between ED and IR/BW not captured in the present analysis and on which future data collection might focus.

With respect to ACF, it has been assumed here that it is not correlated with IR, BW, or ED. The same argument provided above for treating correlation of ED and IR/BW was used here, both with respect to the lack of data against which alternative assumptions might be tested and with respect to the treatment of subpopulations. The value of ACF should, however, depend on the value of ED selected. For example, if individuals move to a geographic sector where exposures occur at an age near the upper end of an age category, they will generally have a higher value of ACF (since a greater proportion of their period ED will be spent in the next age category). Fortunately, as shown in Table 6-24, the variability introduced by ACF is significantly smaller than that introduced by other factors and will contribute negligibly to the composite intersubject variability.

6.3.2.4 <u>The Data, PDFs and CDFs</u>. This report does not discuss the particular data sets used to obtain these individual variability distributions. The justification for using these data sets as a basis for estimating exposure to the receptor populations examined here is summarized in the 1997 EFH, and the reader is referred to that reference for a full discussion of the validity of those data sets. What is noted here is that the data sets described below are specific to the receptor populations to which they have been applied in this analysis, with difficulty in applying the recreational fisher data as described later.

Figures 6-6 through 6-10 provide the cumulative distribution function for each of the parameters displaying variability. The data and distribution fits are compared in Tables 6-19 through 6-23, and distributional characteristics are summarized in Table 6-24. Note that the distributions are given as the ratio of a percentile value (e.g., the 95th percentile of the intersubject variability distribution for IR/BW) over the median. The reason for this choice is that the equation defining exposure (see Equation 6-5) is multiplicative with respect to the factors in Tables 6-6 through 6-10. If lognormal PDFs are multiplied, the median of the product is equal to the product of the medians; the mean of the product is not equal to the product of the means. This, therefore, required the following procedure:

- The intersubject variability distribution for each factor was divided by the median for that factor. This produced an intersubject variability distribution for the ratio of the exposure factor in an individual over the median exposure factor for the population.

- The risk calculations without intersubject variability employed mean values for each of these exposure factors. If the variability distribution noted in the bullet above were multiplied by this mean value, the result would be an intersubject variability distribution with a mean larger than that used in the (original) risk calculations that did not include variability (these two means should be the same). To correct for this, the mean value used in the original risk calculation was converted to a median (for lognormal distributions, the median equals the mean times $\exp\{-\ln^2 S/2\}$ where S is the geometric standard deviation).

Table 6-19. Cumulative Frequencies for Exposure Factor Parameters—Variability of Ingestion Rate per Unit Body Mass for Home-Produced Beef

	Cumulative Percentile								
Age Group (yr)	**1**	**5**	**10**	**25**	**50**	**75**	**90**	**95**	**99**
	0.17	0.31	0.36	0.62	1.00	2.10	5.43	5.95	6.33
6-11	0.10	0.12	0.22	0.44	1.00	2.20	4.50	6.60	6.80
	0.25	0.32	0.34	0.60	1.00	1.62	2.34	2.36	2.83
12-19	0.25	0.30	0.40	0.60	1.00	1.60	2.50	3.10	3.20
	0.17	0.22	0.25	0.43	1.00	1.72	3.07	4.09	5.19
Adult	0.15	0.30	0.40	0.60	1.00	1.70	2.80	4.00	5.50

Note: The values are EFH/log, where EFH (in the upper row) is the numerical value in the 1997 EFH and log (in the lower row) is the numerical value from the best-fitting lognormal distribution. All values in a row are the ratio of the percentile value over the median value in the same row, as described in the text (see Figure 6-6).

Table 6-20. Cumulative Frequencies for Exposure Factor Parameters—Variability of Ingestion Rate per Unit Body Mass for Home-Produced Milk

	Cumulative Percentile								
Age Group (yr)	**1**	**5**	**10**	**25**	**50**	**75**	**90**	**95**	**99**
	0.03	0.06	0.07	0.29	1.00	1.87	2.38	3.01	3.56
All	0.07	0.11	0.20	0.35	0.70	1.50	2.70	3.79	3.85

Note: The values are EFH/log, where EFH (in the upper row) is the numerical value in the 1997 EFH and log (in the lower row) is the numerical value from the best-fitting lognormal distribution. All values in a row are the ratio of the percentile value over the median value for the measurements, as described in the text (see Figure 6-7).

Table 6-21. Cumulative Frequencies for Exposure Factor Parameters—Variability of Ingestion Rate per Unit Body Mass for Recreational Fishing

	Cumulative Percentile								
Age Group (yr)	**1**	**5**	**10**	**25**	**50**	**75**	**90**	**95**	**99**
	NA*	NA	0.11	0.36	1.00	1.93	2.38	3.00	NA
All	NA	NA	0.25	0.42	0.84	1.77	3.19	4.53	NA

NA = Measurements not available at these percentiles.

Note: The values are EFH/log, where EFH (in the upper row) is the numerical value in the 1997 EFH and log (in the lower row) is the numerical value from the best-fitting lognormal distribution. All values in a row are the ratio of the percentile value over the median value for the measurements, as described in the text (see Figure 6-8).

Table 6-22. Cumulative Frequencies for Exposure Factor Parameters—Variability of Occupancy Periods for Farming Populations

	Cumulative Percentile								
Age Group (yr)	**1**	**5**	**10**	**25**	**50**	**75**	**90**	**95**	**99**
	NA	NA	NA	0.25	1.00	2.67	4.83	5.8	NA
All	NA	NA	NA	0.50	1.00	2.20	4.60	7.00	NA

NA = Measurements not available at these percentiles.

Note: The values are EFH/log, where EFH (in the upper row) is the numerical value in the 1997 EFH and log (in the lower row) is the numerical value from the best-fitting lognormal distribution. All values in a row are the ratio of the percentile value over the median value in the same row, as described in the text (see Figure 6-9).

Table 6-23. Cumulative Frequencies for Exposure Factor Parameters—Variability of Age-Crossing Correction Factor

	Cumulative Percentile								
Age Group (yr)	**1**	**5**	**10**	**25**	**50**	**75**	**90**	**95**	**99**
	NA	NA	0.83	0.92	1.00	1.20	1.30	1.45	NA
All	NA	NA	0.80	0.90	1.00	1.13	1.25	1.30	NA

NA = Simulations not available at these percentiles.

Note: The values are the ratio of the percentile value over the median value for the simulations, as described in the text (see Figure 6-10).

Table 6-24. Summary of Variability Expressed as GSDs of Exposure Parameters for Three Pathways and Four Age Groups

Receptor Population, Age Group (yr)	IR	OP	ACF	COMP
Commercial Beef Farmer				
0-5	3.3(1.17)	3.2(1.19)	1.2	5.3
6-11	3.3(1.17)	3.2(1.19)	1.2	5.3
12-19	2.0(0.92)	3.2(1.19)	1.2	3.9
>19	2.3(0.92)	3.2(1.19)	1.0	4.2
Commercial Dairy Farmer				
0-5	2.8(1.12)	3.2(1.19)	1.2	4.8
6-11	2.8(1.12)	3.2(1.19)	1.2	4.8
12-19	2.8(1.12)	3.2(1.19)	1.2	4.8
>19	2.8(1.12)	3.2(1.19)	1.0	4.7
Recreational Fisher				
0-5	2.8(0.94)	NA	NA	2.8
6-11	2.8(0.94)	NA	NA	2.8
12-19	2.8(0.94)	NA	NA	2.8
>20	2.8(0.94)	NA	NA	2.8

ACF = Age correction factor.
COMP = Composite geometric standard deviation.
IR = Ingestion rate per unit body mass.
NA = Not applicable due to the factor being irrelevant for noncancer endpoints.
OP = Occupancy period or exposure duration.

Notes:

1. Values in parentheses are the ratio of the mean as calculated from the lognormal distribution over the mean as calculated from the data (no ratio is shown for ACF since there are no data).

2. All measures of variability are the geometric standard deviations associated with a lognormal distribution. All medians are 1.0 (including the median of the composite distribution for EF given in the last column). The mean for any of these distributions may be obtained from the formula (remembering that the median is 1.0): Mean = $\exp((\ln^2(GSD))/2)$.

- The median value for the second bullet was multiplied by the intersubject variability from the first bullet. This new distribution then has the property that its median equals the median from the second bullet and its mean equals the mean value used in the original risk calculations that did not include intersubject variability.

The net effect of the process outlined in the bullets above is that the new intersubject variability distribution for risk in a population has a mean equal to the original risk calculation obtained when intersubject variability was not considered. The medians and means for each distribution are provided in Table 6-24, which contains an equation for estimating the mean of any of the distributions from the median value and the GSD reported in that table. Also shown in the table is the ratio of the mean as calculated from the lognormal fit over the mean as calculated from the data. It may be noted that this ratio is close to 1 in all cases.

Ingestion Rate per Unit Body Mass. This exposure factor applies to both dioxin and mercury exposures. Variability distributions were developed for ingestion of beef, milk, and fish, and for the age groups 0 to 5 years, 6 to 11 years, 12 to 19 years, and >19 (adult) years.

Beef Ingestion. The variability of the ingestion rate per unit body mass for home-produced beef was determined from the summary of data in Table 13-36 of the 1997 EFH, Intake of Home Produced Beef (g/kg-day). The numerical value associated with each percentile (for a given age group) was first divided by the median (50th percentile) value to obtain $(IR/BW)_{ratio}$. The distribution of this ratio then was plotted on log-probit paper using the maximum likelihood estimate (see Figure 6-6); on such paper a lognormal distribution appears as a straight line. The GSD was determined from the best-fitting line by dividing the 84th percentile by the 50th percentile.

This procedure was followed for the 6- to 11-, 12- to 19-, and >19-yr-old age groups since data were available in the table for those groups. No such data had been reported for the 0- to 5-yr-old age group. As a result, the data for the 6- to 11-yr-old age group were used as surrogate data for the 0-to 5-yr-old age group in estimating the GSD for the latter group (although the mean for the 0- to 5-yr-old age group was derived from data on that age group and not from the data on the 6- to 11-yr-old age group, as discussed in Section 6.3.1). The decision to use the 6- to 11-yr-old age group as the surrogate for the 0- to 5-yr-old age group in estimating the measure of variance (GSD) was based on the findings in Figure 6-6, which indicate that the GSD varies as a function of age. This implies that the best surrogate data are those of the population closest in age. The resulting distributional characteristics for all age groups are shown in Table 6-19.

Milk Ingestion. The variability of the ingestion rate per unit body mass for home-produced milk was determined from the summary of data in Table 13-28 of the 1997 EFH, Intake of Home Produced Dairy (g/kg-day). This table summarizes data on intake of all home-produced dairy products as averaged over all regions of the country. It is the most complete data set on variability for this exposure pathway, although it requires the assumption that the GSD is the same across all age groups (since only results aggregated across age groups were reported). It

also requires the assumption that the variability in fluid milk consumption is the same as the variability in total dairy consumption. Some data suggest this assumption may be more appropriate for some age groups than others. For example, data in the 1997 EFH indicate that the variability in fluid milk consumption is less than that for total dairy consumption for the youngest age groups. If this is the case, intersubject variability in exposure may have been overestimated here.

The numerical value associated with each percentile (for the aggregate population) was first divided by the median (50th percentile) value to obtain $(IR/BW)_{ratio}$. The distribution of this ratio then was plotted on log-probit paper using the maximum likelihood estimate (see Figure 6-7); on such paper a lognormal distribution appears as a straight line. The GSD was determined from the best-fitting line by dividing the 84th percentile by the 50th percentile. The same variability distribution was applied to all age groups (with the GSD values the same for all ages, but the means taken from the age-specific values, as discussed in Section 6.3.1). The resulting distributional characteristics for all age groups are shown in Table 6-20.

***Fish Ingestion*.** The variability of the ingestion rate per unit body mass for recreational fishing was determined from the summary of data in the final column of Table 10-63 of the 1997 EFH, Distribution of Usual Fish Intake Among Survey Main Respondents who Fished and Consumed Recreationally Caught Fish." This table summarizes data on intake of fish caught through recreational activities as aggregated over all ages. It is the most complete data set on variability for this exposure pathway, although it requires the assumption that the GSD is the same across all age groups (since only results aggregated across age groups were reported).

The numerical value associated with each percentile (for the aggregate population) was first divided by the median (50th percentile) value to obtain $(IR/BW)_{ratio}$. The distribution of this ratio then was plotted on log-probit paper using the maximum likelihood estimate (see Figure 6-8); on such paper a lognormal distribution appears as a straight line. The GSD was determined from the best-fitting line by dividing the 84th percentile by the 50th percentile. The same variability distribution is applied to all age groups with the GSD values the same for all ages but the means taken from the age-specific values as discussed in Section 6.3.1. The resulting distributional characteristics for all age groups are shown in Table 6-21.

Treatment of Loss Factors. The data described above for beef, milk, and fish reflect the amount of each food category used. They do not reflect the fact that some of the food (particularly in the case of beef) is lost during preparation for eating. The data in the 1997 EFH on loss during preparation were examined using Table 13-5, Percent Weight Losses from Preparation of Various Meats. The data in this table reflect variability across separate instances of food preparation and are based on a random sample from a population similar to that examined in the HWC analysis (so the issue of surrogate data is not significant here). An individual exposed over many years will prepare the food many times, with intrasubject variability between instances of preparation. This will tend to cause an individual's lifetime average food loss to converge onto the mean food loss for the population (the issue of convergence to the mean in sampling). As a result, intersubject variability of the loss factor must reflect the variability of the mean loss factor for the exposed population, not the variability of individual instances of food preparation (the latter variability being much larger than the former for the reasons given above).

To determine the intersubject variability in the mean loss factor, the distribution of loss factors from Table 13-5 was used as the basis of a Monte Carlo sampling. A sample size of 100 (a reasonable estimate of the number of food preparation events over which the loss factor might be averaged in a person's exposure duration) was selected at random from this empirical distribution (no distributional shape was assigned a priori) and the mean determined for this sample. A second random sample of 100 then was taken and the mean estimated. This process was repeated 2,000 times, yielding a sample of the variability of this mean. The GSD associated with this distribution was less than 1.1, indicating that the loss factor is not a significant source of intersubject variability relative to the factors described above**.** It should be noted, however, that the intersubject variability in loss factor may have been underestimated here since it was assumed an individual had a loss factor for each instance of food preparation that was drawn randomly from the distribution of loss factors. In actuality, an individual might draw consistently from one or the other tail of this distribution, which would lower the tendency of loss factors to average out over time. The available data do not, however, permit further exploration of this issue**. No further consideration of the loss factor was given in the analysis due to the results of this analysis of the relative contribution of different sources of variability, which indicates that this source of variability is insignificant compared to the other sources**.

Exposure Duration in Farming Populations. Exposure duration was taken to be equal to the occupancy period in a home. For the farming populations, data in Table 15-164 of the 1997 EFH, Total Residence Time, t (years), Corresponding to Selected Values of R(t) by Housing Category, were used to determine variability in exposure duration for this population. This table contains a statistical summary of such data aggregated over all age groups. It is the most complete data set on variability for exposure duration in the farming population, although it requires the assumption that the GSD is the same across all age groups (since only results aggregated across age groups were reported. To determine the age-specific median values of ED for the farming population, the data on the nonfarming population (in which the data were obtained on different age groups) were used in conjunction with the data on the farming population (in which the data were averages over all age groups). For any specific population (farming or nonfarming), and for any specific age group (one for farming and three for nonfarming), the numerical value associated with each percentile was first divided by the median (50th percentile) value for that population/age group to obtain ED_{ratio}. The distribution of this ratio then was plotted on log-probit paper using the maximum likelihood estimate (see Figure 6-9); on such paper a lognormal distribution appears as a straight line. The GSD was determined from the best-fitting line by dividing the 84th percentile by the 50th percentile.

To obtain intersubject variability distributions of exposure duration (or occupancy period) for the different age groups in the case of the farming population, it first was noted that the GSD for the intersubject variability distributions in the three age groups in the nonfarming population were similar (ranging between 2 and 2.3). The sole difference in the distributions for these three age groups was in the median (and, hence, mean). It was assumed that a similar pattern would apply to the farming population if data on the different age groups were available; i.e., that the age groups for the farming population would have the same GSD between them, but different medians/means. Further, the age-adjusted median occupancy period for the nonfarming and farming populations was approximately the same (10 years). This resulted in the following procedure:

- The ratio of the median occupancy period for an age group in the nonfarming population (e.g., 0 to 5 years) was divided by the median for the age-adjusted nonfarming distribution.

- This same ratio was assumed to apply for that age group (e.g., 0 to 5 years) in the farming population. The median of the age-adjusted distribution for the farming population was multiplied by this ratio obtained from the same age group in the nonfarming population.

- For this same age group in the farming population, the GSD of the distribution was assumed to be the GSD of the adjusted distribution for the farming population (the same GSD used for all age groups in the farming population), following the pattern observed in the nonfarming population).

The characteristics for the age-adjusted distribution for the farming population are shown in Table 6-22. The GSDs for all age groups are shown in Table 6-24. Because there are only age-adjusted data for the farming population, the ratio of the predicted over measured mean (in parentheses within that table) refer only to the age-adjusted values; these ratios cannot be estimated for the age-group-specific means since data are not available for those groups. Using the procedure described above, the median values for the three age groups in the farming population are 4.5 years (0- to 5-yr-old age group), 7 years (6- to 11- and 12- to 19-yr-old age groups), 10 years (adults).

Correction Factors for Crossing Age Groups. The risk calculations without intersubject variability in exposure factors employed the assumption that an individual beginning exposure at age X continued exposure under parameter values identical to those of the age group in which the age X falls. This means the ingestion rate per unit body mass was assumed constant during the exposure interval even if, in reality, that individual would have crossed into a higher age group at some point during the exposure interval. Since the ingestion rate per unit body mass generally decreases with age, particularly in the first several age groups, the central tendency estimates will tend to overestimate slightly the mean values of exposure for an age group if individuals in that age group cross age boundaries during the exposure duration.

To explore the effect of this assumption used in the central tendency estimates, the calculations of lifetime risk were repeated using a lifetable approach in which individuals were followed year by year throughout the period of exposure. During each year of life, age-specific intake rates per unit body mass were used, with these changing as the individual aged. The specific values of IR/BW used for an age group were the central tendency values described previously. In other words, an individual retained the central tendency value of IR/BW for an age group while that individual remained in the age group, and then took on the central tendency value for the next higher age group once they had crossed into that age group. A Monte Carlo procedure was developed in which

- An age at beginning of exposure was selected at random (uniform distribution) from within an age interval (e.g., using the 0- to 5-yr-old age interval, a starting age between 0 and 5 was selected randomly using a uniform PDF between 0 and 5).

- An exposure duration was selected at random based on the distribution described previously. From the combination of the starting age and exposure duration, the fraction of time spent in the initial (e.g. 0-5 year) and next higher (e.g. 6-11) age group was determined.

- The time-weighted average ingestion per unit body mass (g/kg) over the selected exposure period was calculated by multiplying the value of IR/BW for the first age interval by the fraction of the exposure duration spent in that interval, multiplying the value of IR/BW for the second age interval by the fraction of time spent in that interval, and summing these two contributions.

- The ratio of the time-weighted average ingestion per unit body mass from above over the value of IR/BW for the starting time interval was obtained.

- This process was repeated over a sample size of 1,000 for the same starting age interval (e.g., 0- to 5-yr-old age interval).

The result of this process is a sample of 1,000 calculations of the ratio of the intake rate per unit body mass when crossing of age intervals is accounted for and when they are not. This, in turn, is equivalent to a sample of 1,000 ratios of the "true" intake per unit body mass over the value obtained when it is assumed that the exposure factors remain the same during the exposure duration as those applicable at the initial ages of a cohort. The median for this population of samples is 1.0 (so the original assumption used in the central tendency estimates did not produce a biased result). The GSD was obtained by plotting this distribution on log-probit paper (see Figure 6-10), determining the maximum likelihood fit, and determining the ratio of the 84th percentile over the 50th percentile. There were no significant differences in either median, mean, or GSD for the age groups 0- to 5-, 6- to 11-, and 12- to 19-yr-olds. Therefore, these groups were combined into a single intersubject variability distribution summarized in Table 6-23 (and Figure 6-10). The age-crossing factor was not significant for adults due to the assumption that exposure values of IR/BW had stabilized by age 20 years. This correction factor was applied only for estimation of cancer risks (i.e., TCDD calculations for beef and milk ingestion) and not for fish ingestion (i.e., methylmercury calculations), since it is not relevant in the case of noncancer effects (such as developmental effects for which averaging of exposures is inappropriate).

Aggregate Variability. For dioxin exposures, there are three parameters displaying variability; for methylmercury, there is one. In both cases, the risk for an individual is the product of the separate terms shown in the equations at the beginning of this section. The product of lognormally distributed terms is itself a lognormal distribution, with median equal to the product of the medians for the composite terms. Assuming independence of the contributions to variability (as discussed in Section 6.3.1 with noted caveats), and that each contribution is described by a lognormal PDF (as discussed in Section 6.3.1), the variability of risk or HQ within a sector is described by a lognormal distribution with a median equal to the central tendency value, since the medians for the distributions of $(IR/BW)_{ratio}$, ED_{ratio} and CF_{ratio} all are 1.0. The GSD of this distribution of risk or HQ within the sector may be calculated from a formula relating the separate GSDs to the GSD of the product of lognormally distributed quantities:

$$GSD_{product} = \exp(\ln^2(GSD_1) + \ln^2(GSD_2) \ldots + \ln^2(GSD_n))^{0.5} \quad (6\text{-}6)$$

where there are n terms multiplied in the equation for exposure. Again, for exposure to carcinogens, there are three terms with variability; these are $(IR/BW)_{ratio}$, ED_{ratio}, and CF_{ratio}. For noncarcinogens, there is one term with variability: $(IR/BW)_{ratio}$. The aggregate variability also is displayed in Table 6-23.

6.3.2.5 <u>Incorporating Variability</u>. The HWC methodology for incorporating intersubject variability used a postprocessing approach wherein the cumulative risk distribution first was generated using only central tendency values for each exposure factor (means), and then inter-subject exposure parameter variability was incorporated into this cumulative risk distribution (hence the term "postprocessing"). The HWC methodology evaluated the aggregate impact of exposure parameter variability associated with three factors as described previously in this section: (1) ingestion rate per unit body mass, (2) occupancy period, and (3) age correction factor. (Note: The latter two factors do not apply to the recreational fisher for which noncarcinogenic HQs are generated.) The methodology described below is valid for cases in which a single constituent and pathway dominates for a given age group and receptor population, an assumption first confirmed to be valid for this analysis by ensuring that this constituent/pathway contributed at least 90 percent of the total risk or HQ.

The methodology of Monte Carlo analysis employed here to assess variability was structured to follow the guidelines set out in EPA's *Guiding Principles for Monte Carlo Analysis* (U.S. EPA, 1997b). To incorporate variability of exposure into the distributions of risk R and hazard quotient HQ, the postprocessing assessment returned to the stage in the original (preprocessing) calculations at which the sector-constituent-pathway-specific exposures were calculated. Variability of the exposure factor in a specific sector from a specific pathway and constituent then was characterized using the distributions described previously in this section (bearing in mind that the intersubject variability used in all cases was selected to yield the correct mean value of risk in the exposed population). Monte Carlo analysis then was performed using the defining equation for either risk or HQ, with the probability of sampling an individual from a given sector being equal to the fraction of the total exposed population in that sector. Once an individual was selected from a sector, random values for EF were selected using the intersubject variability distributions described previously (with the same distribution used in all sectors, although different distributions were used for different ages and exposure pathways where available). This process was repeated over all sectors, resulting in a composite variability distribution for either risk or HQ that was specific to an age group, receptor population, and constituent.

Consider this postprocessing approach in more detail. The HWC risk analysis performed without intersubject variability in exposure factors (hereafter called the "original" analysis) generated one risk estimate for each:

- Facility (the number of facilities depends on the facility category)
- Sector surrounding a facility (16 per facility)
- Compound or constituent (dioxin or mercury)

- Age group (0-5, 6-11, 12-19, adult)

- Relevant receptor subpopulation (beef cattle farmer, dairy cattle farmer, or recreational fisher).

For example, if there were 10 facilities, there would be 160 sectors and for each of these 160 sectors there would be a single risk estimate for dioxin exposures to dairy cattle farmers in the 11- to 19-yr-old age group. This example will be used in the following discussion; exactly the same methodology was applied to all facility category/constituent/age group/receptor analyses.

In the example above, there are 160 central tendency estimates of the risk or HQ value generated in the original analysis (one for each sector). For each of these 160 central tendency estimates, a lognormal variability distribution for EF with a median of 1.0 and an associated geometric standard deviation was assigned based on some combination of the factors described previously in this section (variability of ingestion rate per unit body mass, exposure duration, and correction factor for crossing age groups in the case of dioxin exposures; variability of ingestion rate per unit body mass in the case of mercury exposures). Note that while the median of this intersubject variability distribution is 1.0, the mean is larger. It was necessary, therefore, to first convert the estimate of risk or HQ from the original analysis into a median value so the intersubject variability distribution could be applied. This was accomplished through multiplying the original risk or HQ value by $\exp(-(\ln^2 GSD)/2)$, which is the ratio of the median over the mean for a lognormal distribution (GSD is the geometric standard deviation, specific values applied are shown in Table 6-25). The intersubject variability distribution of EF was then scaled through multiplication by the median risk value in a sector to yield the distribution of risk or HQ in that sector. This resulted in 160 separate variability distributions for the example used here, each describing inter-subject variability of risk or HQ within a sector. Note again that the means of these distributions were equal to the risk or HQ estimate from the original analysis, since the latter also represented means.

The task then was to combine these 160 separate variability distributions into a single, composite, variability distribution across the entire exposed population. This composite distribution must weight in the 160 separate distributions according to their relative contribution to the total population; i.e., the contribution of a given sector's variability distribution to the composite variability distribution must equal the fraction of the total exposed population contained in that sector. While the separate distributions are lognormal, the weighted sum of lognormal distributions is not itself lognormal. As a result, there is no analytic solution to the statistical characteristics of this composite distribution. A Monte Carlo sampling procedure based in the software CrystalBall® was used, therefore, to construct the composite distribution. The steps of sampling were as follows:

- The population size in each separate sector was determined from GIS analysis.

- The total size of the exposed population was determined by summing populations across all sectors in the assessment.

- The fraction of the total exposed population contained in each sector was calculated by dividing the population in a sector by the total exposed population.

- A random number was generated using a uniform probability density on the interval [0,1]; the algorithm for this generation was the RAND function in EXCEL (CrystalBall® resides on top of EXCEL).

- A sector was selected at random from the total population of 160 sectors using this random number. The probability of a particular sector being selected was equal to the fraction of the total exposed population in that sector (see Crawford-Brown, 1997).

- Once a sector was selected at random, the variability of EF_{ratio} in that sector was assigned as a lognormal probability density function with median of 1.0 and GSD specific to that constituent, pathway, and receptor population (see Table 6-25).

- One sample of the value of EF was selected from the variability distribution using Monte Carlo sampling with a seed value of 0.0. This value of EF was multiplied by the median value for risk or HQ in that sector to obtain the value of risk or HQ for that sampled individual; this was stored in a file (a "forecast" file within CrystalBall®).

- This process was repeated for the number of trials necessary to meet criteria of stability for the resulting composite variability distribution.

- Risks and values of HQ associated with prescribed percentiles of the composite variability distribution (e.g., 50th, 75th and 97th percentiles) then were determined.

A trade-off was necessary in selecting the sample size for the Monte Carlo analysis. A larger sample size improves the estimates of risk associated with each percentile in the variability distribution. This larger sample size, however, requires greater computation time, with the potential for computation times that are too long to provide timely answers for decisions. The number of samples employed in the Monte Carlo analysis performed here was based on criteria related to the stability of the median (50th percentile) risk value and of the 97th percentile, following guidelines in EPA's *Guiding Principles for Monte Carlo Analysis* (U.S. EPA, 1997b). Sample size was selected initially to be 1,000 runs of the model (i.e., 1,000 randomly selected individuals from the exposed population). Sample size then was increased in increments of 500 on the same model, and estimates at the 50th and 97th percentiles of the intersubject variability distributions for the entire exposed population compared (e.g., the median estimate for a sample size of 1,500 compared against that for a sample size of 1000, and the 97th percentile estimate also compared at these two sample sizes). Sample size was increased until the change in the estimate (for both the 50th and 97th percentiles) was not larger than 5 percent. This criterion ensures the stability of the first decimal place of the percentile estimates, which is consistent with the number of significant digits available through the underlying data sets and is in keeping with current best practice in the field of Monte Carlo analysis.

For example, if the 97th percentile value of the risk was 1×10^{-6} with a sample size of 3000, this sample size was considered adequate if and only if the 97th percentile value of the risk for a sample size of 2,500 was between 0.995×10^{-6} and 1.05×10^{-6}. If it was not, the sample size would be increased to 3,500 and the run performed again. This test was run on several of the

facility categories with the largest number of facilities (where meeting the criterion would be most difficult). In particular, it was run for the facility categories with more than 200 facilities. From this analysis, it was determined that a sample size of 3,000 runs provided the necessary stability of the variability distribution at both of these percentiles.

6.3.2.6 Sensitivity and Uncertainty. Prior to the development of intersubject variability distributions, a sensitivity analysis was performed to identify the most significant factors for which variability and uncertainty should be assessed. The original analysis (without intersubject variability) was used to identify the most significant receptors, constituents, and factors. Three receptor populations showed the largest risks and values of HQ for the facility categories examined here and were determined to be the "driving" receptor categories for regulatory decisions:

- Beef cattle farmers
- Dairy cattle farmers
- Recreational fishers.

When the separate contributions of the pathways and constituents were examined for these receptor populations, two constituents contributed more than 95 percent of the total risk when their risks or HQs were summed; these were determined to be the "driving" constituents:

- Dioxin
- Methylmercury.

Finally, exposure pathways were considered for these different combinations of receptor population and constituent. In all facility categories considered in this analysis, the following exposure pathways contributed more than 95 percent of the risk for all age groups:

- Ingestion of dioxin in beef for the beef farmer
- Ingestion of dioxin in milk for the dairy farmer
- Ingestion of methylmercury in fish for the recreational fisher.

These three exposure scenarios, for all four age groups, were examined in the present analysis and variability distributions developed for each as described previously in this section.

Quantitative uncertainty analyses also were performed of the predictions of the intersubject variability distributions for risk and HQ, based on the uncertainties in the various factors going into the calculations. The goal of this aspect of the analysis was to construct confidence intervals around the point estimates of specific percentiles in the intersubject variability distribution for the aggregated, exposed population. The procedure for estimating these confidence intervals was the SUDAAN software. SUDAAN allows the calculation of variances associated with the selection of a subset of facilities from a facility category. SUDAAN does not, however, explicitly handle intersubject variability distributions within a sector such as those that arise from intersubject variability of exposure parameters. Without this latter source of variability incorporated into the SUDAAN analysis, the estimates of variance do not include variance introduced by random sampling of a finite subset of individuals from the variability distribution in a sector.

It is possible, however, to reflect intersubject variability within a geographic sector using SUDAAN (and, hence, to estimate uncertainty that reflects intersubject variability) through the use of a discrete approximation to the intersubject variability distribution within that sector. In the approach used here, the continuous intersubject variability distribution for a sector generated by the method described in Section 6.3.2.5 was replaced by a discrete distribution with the same median, mean, and GSD through division of the distribution into 20 equal probability intervals (5 percent of the population of the sector contained within each interval). Let PDF(x) be the probability density function (lognormal) for the risk (or HI) in a sector; PDF(x) reflects the intersubject variability of risk (or HQ) in that sector. PDF(x) must then be divided into 20 subsectors of equal probability (i.e., 0.05). For the first subsector, the condition is that the integral of PDF(x) from 0 to UL (the upper limit of the first subsector) must equal 0.05. The lower limit (LL) of the second subsector is the value of the risk (or HI) for which the integral from 0 to LL is 0.05; this corresponds to the value of UL for the first subsector. This process is continued through all 20 subsectors. Each interval for a sector's distribution of risk or HQ was treated as a subsector within the sector. SUDAAN then was used to determine the uncertainty in estimates of specific percentiles of the intersubject variability distribution for risk and/or HQ as described in Appendix I.

This use of a discrete approximation introduces a potential error into the calculation of both the best estimate values and confidence intervals for the risk (or HI) associated with specific percentiles in the aggregated variability distribution. This leads to the question: **Does the use of a discrete distribution, with the characteristics described above, cause significant inaccuracies in the best estimates of the percentile values in the variability distribution developed for the aggregate exposed population (i.e., aggregated over all geographical sectors)?** To address this question, a Monte Carlo procedure for sampling from variability distributions associated with populations (one population from each of two sectors) was developed. Two populations were assumed: one with a mean of 10 and a GSD of 4 (lognormal PDF) and a second with a mean of 1 and a GSD of 4 (lognormal PDF). The discrete approximation to the variability distributions should introduce the largest inaccuracies into the estimates of the 95th, 97th, and 99th percentiles of the aggregated distribution when 100 percent of the exposed population is in only one of the two distributions described above. As the two distributions become more equal in size, the errors in the 95th, 97th, and 99th percentiles should be reduced.

The procedure for this analysis was to first select a fraction of the aggregate population in each of the two distributions. The parameter f is taken to be the fraction in the first population (the population with mean of 10). The fraction in the second population then is 1-f. A Monte Carlo procedure then was developed to sample at random from each of the two distributions, with the probability of sampling from the first distribution being f and the probability of sampling from the second being 1-f. A total of 10,000 samples were obtained to construct the variability distribution for the aggregated population (this being the sample size necessary to ensure that the 97th percentile can be estimated to within 5 percent, the criterion selected for stability of that estimate). Both the 95th, 97th, and 99th percentiles for this distribution were obtained and recorded. This process then was repeated for values of f between 0 and 1.0, in increments of 0.1.

To simulate the SUDAAN analysis, the two distributions (one for each of the two subpopulations) then were developed in discrete form using the method described previously. The mean in each "slice" of this discrete distribution was assigned to each individual from that slice. This resulted in N numerical values for each of the two distributions, where N is the number of slices in the discretized distribution. The fraction of people assigned to each slice equals the fraction of people in that population (there are two populations) divided by N (1/N being the fraction of a given population contained in a slice). Monte Carlo analysis then was used (with a sample size of 10,000) to determine the variability distribution for the aggregate population and to specify the 95th, 97th, and 99th percentiles of the aggregated distribution.

The 95th percentile from the discretized (SUDAAN-like) result minus the 95th percentile from the nondiscretized (exact) result then was calculated and divided by the 95th percentile from the nondiscretized result for each value of f. The same was done for the 97th and 99th percentiles. The magnitude of this ratio (with the absolute value of the differences) indicates the fractional degree of inaccuracy introduced by the discretization of the variability distributions for the two populations. This inaccuracy will become smaller as the number of "slices" is increased (going to 0.0 as N approaches infinity). Values of N equal to 5, 10, 20, 30, and 40 were examined. The results are summarized in Appendix I. The use of 20 slices for each variability distribution satisfied a criterion that the upper percentiles (95th and 97th) of the aggregated distributions should be estimated to within an accuracy of 25 percent. The actual error introduced into the estimates of these percentiles by the used of a discretized distribution within SUDAAN decreases as the number of populations sampled to create the aggregated population increases; in the actual SUDAAN analysis performed for this project, the number of sampled populations was significantly larger than 2, so the estimates of the confidence limits will be accurate to within less than 25 percent.

6.4 Exposure Estimate

Estimates of exposure are based on the potential dose (e.g., the dose ingested or inhaled) rather than the applied dose (e.g., the dose delivered to the gastrointestinal tract) or the internal dose (e.g., the dose delivered to the target organ). This is generally consistent with the exposure metric used in most epidemiologic and toxicologic studies that serve as the basis for establishing the toxicological benchmarks used for assessing risk (see Section 7.0).

6.4.1 Average Daily Dose

For the purposes of the HWC analysis, the average daily dose is defined as

$$\mathrm{ADD} = \sum_{\mathrm{Pathways}} \frac{\mathrm{C} \cdot \mathrm{IR}}{\mathrm{BW}} \qquad (6\text{-}7)$$

where

C	=	concentration, mass/volume or mass/mass
IR	=	intake rate, mass/time or volume/time
BW	=	body weight, mass.

Equation 6-7 does not have an exposure duration or an averaging time included in the estimation of the ADD. Although this appears inconsistent with the 1992 Exposure Guidelines (U.S. EPA, 1992), it is not appropriate to include these terms in the HWC analysis, as neither the exposure duration nor the averaging time can be included in a meaningful way. The ADD is nominally calculated for a 1-year period (i.e., year 30), and the fate and transport modeling is based on long-term values reflecting multiyear averaging of data for certain model inputs (see Section 5.3).

Contaminant concentration represents the concentration of a chemical in a medium that contacts the body. Intake rate depends upon the route of exposure; for example, it might be an inhalation rate or an ingestion rate.

The ADD is used for assessing risks for noncancer effects by averaging exposures or doses over the period of time during which exposure occurred. Note that while there is no explicit time averaging in Equation 6-7, the media concentrations used in the HWC analysis for the ADD represent longer-term, multiyear averaging times. Although model-estimated peak concentrations are used for assessing the ADD (as discussed in Section 5.0), the peak concentrations are reflective of a number of important parameters that represent longer-term averages. These include averaging of air modeling results over 5 years of meteorological data, the use of long-term average hydrological data for surface water modeling, and the use of quasi-steady-state bioaccumulation factors, among others. Therefore, the ADD may be considered a peak chronic exposure that represents a balance between a lifetime average exposure and a short-term acute or subchronic exposure.

6.4.2 Lifetime Average Daily Dose

The lifetime average daily dose, used for assessing risks for carcinogenic effects, is defined as

$$\mathrm{LADD} = \frac{\overline{C} \cdot \mathrm{IR}}{\mathrm{BW}} \cdot \frac{\mathrm{ED}}{\mathrm{LT}} \tag{6-8}$$

where

$\overline{C}$	=	average concentration, mass/mass or mass/volume
ED	=	exposure duration, time
BW	=	body weight, mass
LT	=	lifetime, time.

For cancer effects, biological responses are described in terms of lifetime probabilities, even though exposure may not be lifelong. Here, the exposure duration (the length of time of contact with a contaminant) is used to average the ADD over a lifetime (70 years). Note that the media concentrations used in the HWC analysis for assessing the LADD (e.g., soil concentration) have generally been averaged explicitly over the duration of exposure. This provides a more exact estimate of the lifetime average daily dose.

6.4.3 Infant Average Daily Dose

The ADD for an infant reflects contaminant intake as a result of breast milk ingestion. The infant ADD is calculated as:

$$ADD_{infant} = \frac{C_{milkfat} \cdot f_3 \cdot f_4 \cdot IR_{milk} \cdot ED}{BW_{infant} \cdot AT} \qquad (6\text{-}9)$$

where

$C_{milkfat}$	=	concentration in maternal milk
f_3	=	fraction of fat in breast milk
f_4	=	fraction of ingested contaminant absorbed
IR_{milk}	=	ingestion rate of breast milk
ED	=	exposure duration
BW_{infant}	=	infant body weight
AT	=	averaging time.

The infant ADD was applied in the assessment of a breast milk exposure scenario for TCDD-TEQ for the infants of mothers (both 12- to 19- and >19-yr-olds) from each receptor population considered in the HWC analysis. The exposure of the infant through the consumption of contaminated breast milk was estimated based on the mother's exposure (assumed to be at steady state over her period of exposure). The concentration in maternal milk fat was calculated from the mother’s ADD, as described in Section 5.4.

TCDD-TEQ exposure through the consumption of breast milk was estimated for infants nursed by the adult receptors in this analysis. The concentration in maternal milk was calculated as shown in Equation 6-10. The equation used to calculate concentration in maternal milkfat does not account for the loss of contaminant from the mother’s body that occurs as a consequence of breast feeding; therefore, it may tend to overestimate concentrations in breast milk.

Values for the model parameters used to characterize breast milk exposure are provided in Table 6-25.

6.5 Body Burden Estimates

A body burden is a specific measure of exposure, such as a blood lead level, that evaluates the potential accumulation of a contaminant over time in an individual’s body. Incremental exposures (i.e., those not attributable to background levels) can be evaluated in terms of increased individual body burdens.

Concentration in Maternal Milk

$$C_{(milkfat)} = \frac{ADD_m \bullet 10^9 \bullet h \bullet f_1}{0.693 \bullet f_2} \qquad (6\text{-}10)$$

Parameter	Description	Values
$C_{(milkfat)}$	Concentration in maternal milk (pg/kg of milkfat)	
ADD_m	Maternal average daily dose (mg/kg-d)	Calculated (see Table C-5.8)
10^9	Conversion constant (pg/mg)	
h	Half-life of dioxin in adults (d)	2,555
f_1	Proportion of ingested dioxin that is stored in fat (unitless)	0.9
f_2	Proportion of mother's weight that is fat (unitless)	0.3

Table 6-25. Model Parameter Values Used To Characterize Breast Milk Exposure for TCDD-TEQ

Parameter	Parameter Values	References
Body weight of infant	10.2 kg	U.S. EPA, 1997a: Body Weights of Children (Table 7-3) (mean values for 6-11 mos and 1-yr age groups were averaged together)
Exposure duration for infant	1 yr	U.S. EPA, 1994a
Ingestion rate of breast milk	0.742 L/d	U.S. EPA, 1997a (EPA-recommended average value)
Body weight of mother	65.4 kg	U.S. EPA 1997a: Body Weights of Adults (Table 7-2) (mean for women)
Fraction of mother's weight that is fat	0.3	U.S. EPA, 1994a
Fraction of dioxin that is absorbed	0.90	U.S. EPA, 1994a
Fraction of absorbed dioxin that is stored in fat	0.90	U.S. EPA, 1994a
Fraction of fat in breast milk	0.04	U.S. EPA, 1994a

6.5.1 Blood Lead Modeling

Human health risk characterization for lead is based on a comparison between modeled blood lead levels (i.e., PbB levels) and the health benchmark level (HBL) established for lead: 10 µg/dL. Because of heightened sensitivity to lead in children, the lead analysis focuses on the 0- to 5-yr-old age group with a separate set of risk results being generated for each modeled receptor population (and each modeled combustor category). To fully characterize the potential for adverse health effects linked to lead exposure, the HWC analysis generates two types of risk results for lead: (1) individual risk results presenting the PbB levels for the 50th, 75th, 90th, 95th, 97th, and 99th percentiles of the individual risk distribution for a given receptor population/combustor category combination; and (2) population risk results including the number of children with PbB levels at or above the HBL for lead. Both categories of risk incorporate interindividual and intrasector variability in PbB levels. Because the objective of the analysis is to characterize the incremental risk posed by the HWC facilities over and above background (i.e., the "excess risk"), the population risk results include estimates based on: background exposure, incremental exposure, and total exposure (background and incremental aggregated). Similarly, the individual risk results (i.e., the percentile results) are presented as background, incremental, and total (background and incremental aggregated). Additional detail on the risk characterization for lead exposure can be found in Section 8.3.

Modeled PbB levels are generated for the analysis using a combination of site-specific media concentrations (i.e., soil, drinking water, and ambient air) and dietary intake rate data obtained from the Indirect Exposure Model. These data are processed using the Integrated Exposure Uptake Biokinetic (IEUBK) model to generate sector-level PbB estimates for each modeled 0- to 5-yr-old age group.

The IEUBK model (U.S. EPA, 1994b), developed by EPA to predict PbB levels for an individual child or population of children, was specifically designed to evaluate lead exposure in young children (birth to 7 years of age) because this age group is known to be highly sensitive to lead exposure. The IEUBK is a versatile assessment tool that allows the user to make rapid calculations from a complex array of intake, absorption, distribution, and elimination equations by building site-specific and age-dependent exposure scenarios. The IEUBK model allows the user to input different media concentrations and dietary intake rates for lead for the set of consecutive years being modeled (i.e., different concentrations/ingestion rates can be entered for different years to reflect changing site conditions; the model does not allow a temporal resolution finer than a year). The user can also either elect to use the exposure parameters contained within the IEUBK model, which are age-differentiated, or enter their own exposure parameters. The IEUBK model then uses the inputted data to generate a yearly average PbB level for the population being modeled. The IEUBK model is comprised of four distinct components that work together in series:

- **Exposure component:** Determines how much lead enters the child's body over the exposure period. This component combines media-specific (e.g., air, soil, food, water) lead concentrations and age-dependent media intake rates to calculate age- and media-specific lead intake rates.

- **Uptake component:** Calculates how much of the lead that enters the body through the exposure routes is actually absorbed into the blood.
- **Biokinetic component:** Models the distribution of the lead from the blood to other body tissues and/or elimination from the body.
- **Probability distribution component:** Calculates a probability distribution of PbB for a hypothetical child or population of children. The geometric mean PbB is calculated and is combined with a prescribed GSD representing interindividual variability in lead uptake to generate a PbB distribution from which the probability of (e.g., the estimated proportion of) the target population exceeding a PbB level of 10 µg/dL can be estimated. As discussed below, interindividual variability in lead uptake is evaluated outside of IEUBK for the HWC risk analysis.

The IEUBK model is typically used to characterize lead exposure for scenarios where the ingestion of soil, dust, or drinking water and/or the inhalation of ambient air are the pathways of primary concern (e.g., lead exposure at contaminated waste sites or older houses containing lead paint). Often with these scenarios, dietary exposure to lead is considered a non-site-specific parameter and default dietary intake rates for lead contained within IEUBK are used. However, because the HWC risk analysis focuses on site-specific emissions that can impact home-produced dietary items (via food chain impacts), dietary exposure is considered site-specific and may represent a critical pathway for lead exposure. Therefore, rather than using the default dietary exposure values for lead provided in the IEUBK model, sector-level values (in the form of µg lead/day dose estimates generated by IEM) were used in modeling lead exposure[4]. Nondietary exposures, including incidental soil ingestion, drinking water ingestion, and inhalation, were evaluated by inputting IEM-derived media concentrations into IEUBK and using the default exposure factors contained within IEUBK[5]. For the HWC risk analysis, it was also assumed that indoor dust had the same lead concentration as modeled sector soils for purposes of modeling lead exposure[6].

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[4] All dietary exposure was modeled within IEUBK using a bioavailability factor of 0.5. Failure to differentiate this factor for different food types does introduce parameter uncertainty into the analysis.

[5] Exposure factors used within IEUBK to model air, soil/dust, and drinking water ingestion are: 4 m^3/d, 0.118 g/d, and 0.46 L/d, respectively. IEUBK actually uses separate exposure factors for each year of life; these values are the averages of exposure factors for years 1 through 5. The IEUBK exposure factors are somewhat lower than the exposure factors used to model similar pathways in the HWC risk analysis, reflecting the fact that the HWC uses mean values, while the IEUBK model uses median values (corresponding HWC exposure factors for the 0- to 5-yr-old age group are: 6.5 m^3/d, 0.179 g/d, and 0.653 L/d for air, soil, and drinking water, respectively) .

[6] In the absence of measured data, the IEUBK guidance manual provides two options for estimating lead concentrations in indoor dust: (1) setting dust concentrations equal to soil concentrations and (2) predicting dust concentrations using contributions from both modeled soil and ambient air concentrations. To decide which approach to use in generating lead concentration in indoor dust, a sensitivity analysis was conducted to compare the dust concentration values that would be generated using both options. Given the specific source type and media impacts associated with the HWC risk analysis (i.e., lead released into ambient air from HWC facilities with resulting loading to sector-level soils), the two options were shown to generate essentially identical dust concentrations. Therefore, the first option of setting dust equal to modeled soil concentrations was used because it is simpler to implement within the analysis.

An age of 60 months was used in conducting the IEUBK modeling for the HWC risk analysis (i.e., the IEUBK model was configured to generate lead exposure estimates for a 5-yr-old child). Although this approach is reasonable because the lead analysis is focusing on the 0- to 5-yr-old age group, some uncertainty is introduced into the PbB estimates by using a single age in conducting lead modeling. In reality, the 0- to 5-yr-old age group within any given sector is comprised of a mix of children ranging in age from newborn status to 5 years of age. Consequently, these children will display a range of intake rates and exposure durations reflecting their varying ages. Intake rates for most media and dietary items (on a mg media per kg body weight basis) are higher for the first few years of life than for the 5th year of life and, consequently, the assumption of 5 years of age for all children in this age group may actually underestimate exposure levels for some of the younger children. The overall impact on modeled PbB levels resulting from the use of a single age for the 0- to 5-yr-old age group will depend on a number of factors including specific differences in exposure levels for different ages and key factors related to pharmacokinetic modeling for lead including clearance rates and half-lives. A quantitative analysis of uncertainty associated with using a single age (i.e., 5 years) to characterize the child cohort evaluated in the lead analysis has not been conducted.

The IEUBK modeling described above produces geometric mean PbB estimates at the sector level for each receptor population and combustor category combination. These basic modeling results represent incremental PbB levels resulting exclusively from exposure to emissions from the HWC facility associated with a given study area. They do not include consideration either of background exposure or interindividual variability in lead uptake. The IEUBK model does have the potential to account for these two factors. However, because the model was not designed to evaluate risk across sites or to account for population density in generating either individual or population-level risk results, the decision was made to model both background exposure to lead and interindividual variability in lead uptake outside of IEUBK.

Background exposure was modeled by adding 3.6 µg/dL (the geometric mean background blood lead level for 0- to 5-year-olds, Pirkle et al., 1994) to each sector-level modeled incremental median PbB level. The resulting aggregated PbB value represents the total median PbB level for a given sector (Section 6.6.3 provides an expanded discussion of the approach used to characterize background exposure for lead). Interindividual variability in lead uptake was modeled by applying a GSD of 1.6 (U.S. EPA, 1994b) to the sector-level median incremental and total PbB values. This GSD is the value used in the IEUBK model to reflect interindividual variability. When applied to the sector-level median incremental PbB value, this GSD produces a variability distribution that reflects the range of PbB values for individuals in that sector resulting from incremental lead exposure.

The GSD of 1.6 obtained from the IEUBK documentation is designed to represent interindividual variability among individuals located within the same neighborhood or block, which reflects the intended use of IEUBK for evaluating lead exposure for individual(s) at the site level. Consequently, this GSD does not reflect intersite variability in background exposure (e.g., differences in background lead levels in soil between urban and rural locations). Consequently, uncertainty is introduced into the analysis through the use of the IEUBK-based GSD because it does not fully account for intersite variability in background lead levels.

The sector-level modeled PbB levels (including both incremental and total) are used together with the GSD of 1.6 to generate both individual- and population-level lead PbB estimates. Individual-level estimates are generated by conducting population-weighted sampling from the sector-level PbB distributions to produce an overall cumulative PbB distribution for a given combustor category. PbB estimates for specific percentiles (e.g., 50th or 95th) can be obtained from this distribution. Because the distribution is population weighted, these percentiles actually represent the PbB level for a modeled individual located at that specific point on the distribution.

Population-level lead risk results are produced by determining the number of children that exceed the HBL for lead within each sector and then summing those exceedance estimates across all sectors within a given combustor category to generate an overall exceedance estimate. The sector-level exceedance estimates are produced by querying the PbB distribution generated for a given sector (either background or total) to determine the percentage of children exceeding the lead HBL and then multiplying that percentage by the number of children in that sector. Total exceedance estimates for a given combustor category/receptor population combination are generated separately for background and total lead exposure and then these two values are subtracted to generate the total exceedance estimate for incremental exposure.

As discussed later in Section 6.6.3, subsequent to completing PbB modeling for the HWC risk analysis, the CDC released the NHANES III report containing updated national-level data on lead exposure in children (CDC, 1997). These data allow more complete characterization of background lead exposure in children including the derivation of an interindividual variability distribution that accounts for both variability in lead uptake and site-to-site variation in background media concentrations (this GSD would be preferable to the 1.6 used in the current analysis for characterizing background variability). However, because these data were identified subsequent to developing and implementing the lead component of the HWC risk analysis, it was not possible to incorporate them into the analysis.

Identification of the newly released CDC data also resulted in identification of an improved methodology for evaluating background and total lead exposure for the ultimate purpose of characterizing incremental exposure. The alternative approach involves conducting a Monte Carlo simulation in which the two underlying distributions (i.e., background based on the NHANES III data and the modeled incremental distribution) would be sampled individually to obtain the incremental and background components of exposure for a sampled individual. These two values would then be summed to generate an aggregated total lead exposure level for that individual. This process would be repeated a sufficient number of times (i.e., number of Monte Carlo iterations) to generate a stable distribution reflecting total exposure for that population. The advantage of this approach is that it does not require any assumptions about the form of the distributions (e.g., they need not have the same GSD), only that they be independent. Although it may be a preferred approach, this approach was not carried out as part of the HWC risk analysis because it was not identified as an option in time to allow its application within the analysis.

6.5.2 Dioxin Incremental MOE (Margin of Exposure)

In the absence of a verified RfD value for 2,3,7,8-TCDD, the potential for noncarcinogenic effects resulting from exposure to dioxin/furan congeners modeled for the HWC risk analysis was assessed using incremental margin of exposure estimates based on benchmarks derived from background body burden data The incremental margin of exposure is the degree of exposure above that which is experienced as a result of background concentrations. The incremental margin of exposure analysis is used when an RfD is not available to assess the potential for noncarcinogenic effects. The incremental margin of exposure analysis was conducted for all age groups for all receptors evaluated in the HWC risk analysis. Modeled intake rates for TCDD-TEQ for a given receptor population or subsistence scenario, aggregated across all pathways, were compared to the background body-burden-based intake rate. The body burden is simply the total amount of a substance that has accumulated in the body at a given time. The background body-burden-based intake rate is the intake rate of the substance that is extrapolated given the known body-burden of that substance.

Pharmacokinetic modeling was used to derive the daily intake rate based on a background body burden value for TCDD-TEQ in human adipose tissue of 30 ppt. Specifically, the 30-ppt value was combined with a half-life for TCDD-TEQ in humans of 7 years to generate a central tendency intake rate of 110 pg/d that is reflective of background. When applied to a 70-kg adult, the 110-pg/d value translates into a daily intake rate of 1.5 pg/kg-d.

Incremental MOE results were generated by dividing the modeled intake rate of TCDD-TEQ (aggregated across all modeled pathways) by a TCDD-TEQ intake rate reflective of background body burden data. The modeled intake rates were generated by aggregating TCDD-TEQ intake rates for all modeled exposure pathways (generated using the noncarcinogenic risk paradigm where the actual intake rate for TCDD-TEQ experienced during the period of exposure is modeled rather than an intake rate standardized to a lifetime equivalent daily intake rate) into a single intake rate. This paradigm uses the average daily dose, as defined previously. The number and type of exposure pathways modeled for each receptor population reflect the specific exposure assumptions used to characterize a particular receptor population. Central tendency exposure parameters were used in generating incremental MOE estimates for all receptor populations. An exposure parameter variability analysis was **not** conducted for the TCDD-TEQ incremental MOE (margin of exposure) risk category because the estimated incremental MOEs were relatively low (at or below 0.1 at the 95th percentile) for the most exposed of the enumerated receptor populations (i.e., children of dairy farmers).

6.6 Background Exposures

6.6.1 Dioxins

An incremental margin of exposure analysis was conducted for all four age groups of each receptor population evaluated in the HWC risk analysis. This analysis compared modeled intake rates for TCDD-TEQ aggregated across all pathways for a given receptor population to the background body-burden-based intake rate. In addition, a breast milk exposure scenario was assessed using incremental margin of exposure for the infants of mothers from each receptor population considered in the HWC analysis. For the breast milk scenario, both 12- to 19- and

>19-yr-old age groups were considered to have the potential to give birth; therefore, the breast milk scenario was evaluated for both age groups.

6.6.1.1 Infants. The modeled daily intake rate for TCDD-TEQ for infants that breastfeed was compared to the background-based intake rate for TCDD-TEQ in breast milk to generate the incremental margin of exposure. The 50-pg/kg-d background exposure is based on typical breast milk ingestion rates (as well as background concentrations in breast milk). The background-based value of 50 pg/kg-d is based on a measured U.S. background level of 16 ppt in the lipid portion of maternal breast milk (U.S. EPA, 1994a).

6.6.1.2 Non-Infants. Background exposure estimates intended to be representative of the general population are presented in U.S. EPA (1994a). The diet-based estimated range of TEQ background exposures in the United States for adults is 1 to 3 pg/kg-d. Background exposures can also be estimated on the basis of body burdens through the use of pharmacokinetic models using adipose tissue or blood lipid concentrations. Using this approach, exposure levels to 2,3,7,8-TCDD are estimated to be consistent with the estimates derived using diet-based approaches.

The same background body-burden-based intake rate for TCDD-TEQ (1.5 pg/kg-d) was used to derive incremental margin of exposure estimates for all non-infant receptor populations evaluated in the HWC risk analysis. This daily intake rate was derived using a pharmacokinetic (PK) model and steady-state assumptions based on a background body burden value for TCDD-TEQ in human adipose tissue of 30 ppt (personal communication, David Cleverly, EPA-ORD-NCEA, 9/19/97) and a half-life for TCDD-TEQ in humans of 7 years (U.S. EPA, 1994a). This results in a central tendency intake rate reflectivity background of 110 pg/d, which is similar to the intake rate reflectivity background of 119 pg/d, which was presented in the draft dioxin reassessment (U.S. EPA, 1994a). When the 110 pg/d ingestion rate is applied to a 70-kg adult, a daily intake rate of 1.5 pg/kg-d is generated. Although it would be preferable to derive separate daily intake rates for TCDD-TEQ background for each of the four age groups considered in the HWC risk analysis, no data were identified for developing age-group-specific values for the three younger age groups. Therefore, the 1.5-pg/kg-d value (based on a 70-kg adult) was used for all four age groups.

Questions arise as to whether current exposures are appropriately estimated using backward steady-state PK modeling. The steady-state assumption in this approach implies that dose has been constant in the past. Much evidence has been collected suggesting that, in fact, current adult body burdens are not the result of a constant past dose. For example, sampling of historical meat samples from past decades of this century showed a rise and fall in dioxin meat concentrations (Winters et al., 1998). This suggests that current body burdens are better explained by a temporally varying dose and not a steady dose in the past, and, therefore, calculating current dose using the steady-state PK model combined with current body burdens will overestimate current dose by a significant amount.

6.6.2 Mercury

The *Mercury Study Report to Congress* (U.S. EPA, 1997c) estimated national exposure distributions using National Health and Nutritional Examination Survey (NHANES III) data on

fish and shellfish consumption frequency over a 1-month interval in conjunction with recall data for fish and shellfish consumption, body weight, and mean mercury concentrations in fish and shellfish. Mercury concentrations in marine, estuarine, and freshwater fish were obtained from databases maintained for marine and estuarine fish and shellfish (National Marine Fisheries Service, 1978, as cited in U.S. EPA, 1997c) and freshwater fish (Lowe et al., 1985, and Bahnick et al., 1994, as cited in U.S. EPA, 1997c).

Every variable used was assumed to be independent and to have a lognormal distribution; because data available were in the form of arithmetic means and standard deviations, analytic formulas were used to estimate the geometric mean and geometric standard deviation.

The distribution of mercury exposure from fish consumption was based on 24-h fish and shellfish recall data (fish species consumed), average mercury concentrations reported for each fish species consumed, and the number of fish meals per month. The distribution of the number of fish meals per month was based on consumption frequency data and was treated as a continuous variable to estimate long-term fish consumption rates. Monthly average mercury exposure distributions are presented in Table 6-26.

Table 6-26. Month-Long Estimates of Mercury Exposure ($\mu g/kg_{BW}/d$)
Population by Ethnic/Racial Group
National Estimates Based on NHANES III Data

	White		Black		Hispanic		Other		Women	
Percentile	**Adults**	**Children**	**Adults**	**Children**	**Adults**	**Children**	**Adults**	**Children**	**Adults**	**All Children**
50th	0.015	0.029	0.020	0.031	0.015	0.023	0.021	0.041	0.011	0.029
75th	0.039	0.072	0.053	0.082	0.047	0.060	0.064	0.11	0.30	0.075
90th	0.092	0.17	0.13	0.19	0.11	0.14	0.17	0.25	0.74	0.18
95th	0.15	0.28	0.21	0.33	0.18	0.24	0.31	0.42	0.13	0.29

Because these data represent a national average, the values in Table 6-26 can be interpreted as representing the recreational fisher population. The values in Table 6-27 represent modeled dose estimates for the recreational fisher receptor generated for the final rule. National background exposure levels for methylmercury (for recreational fishers) can be compared to projected exposure levels for recreational fishers in the vicinity of HWC facilities by comparing the values presented in Tables 6-26 and 6-27. The values in Table 6-27 were derived by converting the hazard quotients reflecting exposure parameter variability in Tables V-A9 through V-A16 of the detailed risk results to ingestion rates, using the following correlation:

$$IR = HQ \cdot RfD \cdot 10^3 \qquad (6\text{-}11)$$

where

IR	=	ingestion rate (µg/kg-d)
HQ	=	hazard quotient (unitless)
RfD	=	reference dose (mg/kg-d)
10^3	=	unit conversion factor (10^3 µg/mg).

Modeled results are presented by age group for four age groups considered in the HWC analysis, according to the same percentiles presented in the 1997 *Mercury Study Report to Congress* (see Table 6-26). The HWC analysis does not differentiate between populations of different ethnicities and genders. In addition to accounting for interindividual variability and variability across modeled waterbodies, the HWC methodology used a postprocessing approach wherein exposure parameter variability was incorporated into the cumulative risk distribution after that distribution (based on central tendency exposure parameters) had been generated (see Section 6.3.2). Note that exposure factor postprocessing will miss annual spikes, which are of importance for risk of developmental effects from methylmercury exposure. The 1-year resolution scale of the basic model is a limiting factor that is a generic issue because short-term maxima are of primary concern for any developmental toxicant. For recreational fish ingestion, the HWC methodology evaluated the aggregate impact of exposure parameter variability associated with ingestion rate per unit body mass in generating average daily doses.

Comparison of the exposure levels presented in Tables 6-26 and 6-27 suggests that incremental exposure to methylmercury released from HWC facilities through recreational fish ingestion is significantly lower than background exposure levels for methylmercury experienced by the general population (including exposure to both naturally occurring and anthropogenic methylmercury). It is important to note in comparing the incremental and background methylmercury exposure data that the incremental data reflect methylmercury impacts only within the immediate study area surrounding a given facility. The HWC risk analysis did not consider long-range transport of mercury beyond individual study areas, which does introduce uncertainty into the analysis. With regard to the incremental-to-background methylmercury comparison, failure to consider long-range transport means that possible transport of mercury from one HWC study area to another (with resulting aggregated impacts on modeled waterbodies) was not considered, which could result in an underprediction of incremental methylmercury risk resulting from the ingestion of recreationally caught fish.

6.6.3 Lead

A study by Pirkle et al. (1994) on the trends in PbB levels in the U.S. population was used in the HWC analysis to characterize background lead concentrations. The study showed that children 0 to 5 years of age have a median PbB concentration of 3.6 µg/dL. This concentration, along with a GSD of 1.6 (as specified in the IEUBK documentation), was used in the HWC analysis, since the general population is not immediately exposed to HWC facility emissions.

The CDC has conducted an ongoing series of national studies of the health of the civilian noninstitutionalized population. The NHANES has been the primary source for monitoring blood lead levels in the U.S. population. Phase 2 of NHANES III, conducted from 1991 to 1994,

Table 6-27. Distribution of Modeled Average Daily Dose of Methylmercury Reflecting Exposure Parameter Variability for Recreational Fisher Population, µg/kg$_{BW}$-d

Percentile	Population Age Group			
	Child 0-5 years	Child 6-11 years	Child 12-19 years	Adult >19 years
Cement Kilns				
50th	0.001	0.0008	0.0005	0.0005
75th	0.006	0.004	0.002	0.003
90th	0.02	0.01	0.007	0.009
95th	0.03	0.02	0.01	0.02
Lightweight Aggregate Kilns				
50th	0.0002	0.0001	0.00006	0.00008
75th	0.0009	0.0005	0.0003	0.0004
90th	0.002	0.001	0.0008	0.001
95th	0.004	0.002	0.001	0.002
Commercial Incinerators				
50th	0.000008	0.000006	0.000003	0.000004
75th	0.0001	0.00008	0.00004	0.00005
90th	0.0004	0.0003	0.0002	0.0002
95th	0.0008	0.0005	0.0003	0.0003
Large On-site Incinerators				
50th	0.00000008	0.00000002	0.00000001	0.000000004
75th	0.000007	0.000004	0.000002	0.000003
90th	0.00007	0.00005	0.00003	0.00003
95th	0.0002	0.0002	0.0001	0.0001
Small On-site Incinerators				
50th	0.0000006	0.0000004	0.0000002	0.0000003
75th	0.000009	0.000006	0.000003	0.000004
90th	0.0001	0.00008	0.00005	0.00006
95th	0.0004	0.0003	0.0002	0.0002

indicated approximately 4.4 percent of the population in the 1- to 5-yr-old age group has an elevated (i.e., >10 µg/dL) blood lead level. This percentage ranged from 11.2 percent for blacks to 2.3 percent for whites and from 8 percent for low-income families to 1 percent for high-income families. For the 1- to 5-yr-old age group, the weighted geometric mean blood lead level was found to be 2.7 µg/dL. This central tendency background PbB level is lower than the value used in the HWC risk analysis (3.6 ug/dL); as a result, the central tendency value used in the HWC analysis may overestimate the effects of background exposures.

The HWC PbB analysis is also affected by model uncertainty associated with the approach used to represent variability in background exposures for modeled receptors. The IEUBK model specifies that a GSD of 1.6 should be used to reflect interindividual variability in blood lead levels when evaluating lead exposure. This GSD was used in the HWC PbB analysis. However, this GSD was developed for use in representing interindividual differences in pharmacokinetics and behavioral factors related to lead exposure and not specifically as a means of reflecting variability in background exposure. Application of a GSD reflective of inter-individual variability only and not spatial variability (i.e., on a national scale) introduces an element of uncertainty into the characterization of background exposure.

A preliminary analysis of the data contained in the CDC report suggests that a GSD for background lead exposure in children could be higher than the value of 1.6 used in this analysis. A lower GSD constricts the tail of a lognormal distribution; a higher GSD expands the tail. Therefore, the background PbB levels generated in this analysis could be underestimated, especially for individuals falling in the upper end of the PbB distribution, as a result of underestimating the variability of the background exposures.

These two sources of uncertainty counteract each other: the use of a larger median value results in higher background exposures while the use of a lower GSD results in lower modeled exposures, especially in the upper end of a distribution. The cumulative effect of these two sources of uncertainty is projected by comparing the background distribution generated in the HWC analysis to the CDC data. The latter indicates that 4.4 percent of 0- to 5-yr-olds have elevated blood lead levels (above 10 µg/dL), whereas the HWC analysis (using a GM of 3.6 and a GSD of 1.6) projects fewer than 2 percent.

6.7 References

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7.0 Human Health Effects

This section presents the human health benchmarks used to evaluate human health effects that may result from exposure to constituents modeled for this risk assessment. This includes presentation of the benchmark values used to characterize human health risks and the scientific basis for these values. A summary of the human health benchmarks is presented in Section 7.1. Section 7.2 summarizes epidemiological studies that have addressed possible human health effects associated with the operation of HWC facilities. Section 7.3 contains summaries of the scientific information supporting development of the human health benchmarks by EPA and other agencies. Particulate matter is discussed in Section 7.4.

7.1 Summary of Human Health Benchmarks

Table 7-1 summarizes the benchmark values used for each of the constituents evaluated in this risk assessment. The benchmarks fall into four categories:

- Oral cancer slope factor $(mg/kg\text{-}d)^{-1}$
- Inhalation cancer slope factor $(mg/kg\text{-}d)^{-1}$
- Reference dose (mg/kg-d)
- Reference concentration (mg/m^3).

The oral cancer slope factors, reference doses, and reference concentrations were obtained from IRIS (U.S. EPA, 1998), except for those values footnoted in the table. The inhalation cancer slope factors are not available on IRIS but were calculated for use in this risk assessment based on inhalation unit risk factors (Inhal URFs), which are available from IRIS. Summaries of the health effects data that form the basis for the benchmark values shown in Table 7-1, including unit risk factors that form the basis for the inhalation cancer slope factors, are provided in Section 7.3.

The inhalation cancer slope factors were developed to characterize cancer risks associated with inhalation exposures by adults and children. Because of the assumptions used in the development of the Inhal URFs, the Inhal URFs themselves could not be used directly to evaluate child exposure to human carcinogens. For this risk assessment, the Inhal URFs were converted to Inhal CSFs using the following equation:

$$\text{Inhal CSF } (mg/kg\text{-}d)^{-1} = \text{Inhal (URF) } (\mu g/m^3)^{-1} \bullet 70 \text{ kg} \div 20 \text{ } m^3/d \bullet 1{,}000 \text{ } \mu g/mg \qquad (7\text{-}1)$$

Table 7-1. Health Benchmark Values Used in Modeling

Chemical	Oral Cancer Slope Factor $(mg/kg\text{-}d)^{-1}$	Inhalation Cancer Slope Factor $(mg/kg\text{-}d)^{-1}$	Reference Dose (mg/kg-d)	Reference Concentration (mg/m^3)
Carcinogenic Effects				
Arsenic	1.5E+00	1.5E+01	3.0E-04	NA
Beryllium	NA	8.4E+00	2.0E-03	2.0E-05
Cadmium	NA	6.3E+00	1.0E-03	NA
Chromium VI	NA	4.2E+01	5.0E-03	NA
Nickel	NA	8.4E-01	2.0E-02	NA
2,3,7,8-TCDD	1.56E+05[a]	1.56E+05[a]	NA	NA
Noncancer Effects				
Antimony	NA	NA	4.0E-04	NA
Barium	NA	NA	7.0E-02	5.0E-04[b]
Chlorine	NA	NA	NA	1.0E-03[c]
Chromium III	NA	NA	1.0E+00	NA
Cobalt	NA	NA	6.0E-02	NA
Hydrogen chloride	NA	NA	NA	2.0E-02
Manganese	NA	NA	1.4E-01	5.0E-05
Lead	NA	NA	NA[d]	NA
Elemental mercury	NA	NA	NA	3.0E-04
Inorganic mercury	NA	NA	3.0E-04	NA
Methylmercury	NA	NA	1.0E-04	NA
Selenium	NA	NA	5.0E-03	NA
Silver	NA	NA	5.0E-03	NA
Thallium	NA	NA	8.0E-05	NA

NA = Not available.

[a] Provisional value from EPA's Health Effects Assessment for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (U.S. EPA, 1984).
[b] Provisional RfC from EPA's Health Effects Assessment Summary Tables (U.S. EPA, 1997b).
[c] Interim RfC developed for use in this HWC risk assessment.
[d] A lead blood level of 10 μg/dL was used as a benchmark for characterization of human health risks associated with lead exposure.

where

70 kg	=	default adult human body weight
20 m^3/d	=	default adult human daily rate of inhalation
1,000 μg	=	1 mg.

Particulate matter health effects were evaluated using concentration-response relationships that relate reductions in ambient concentrations of PM_{10} and $PM_{2.5}$ to avoided incidence of adverse health effects. The health effects data that form the basis for the PM evaluation are presented in Section 7.4.

7.2 Epidemiology Studies

This section summarizes the results of key epidemiologic studies that have been conducted on the adverse health effects from hazardous waste incinerator emissions. Most of these studies were not designed to examine individual pollutants; instead they investigated incinerator emissions in general.

Often epidemiologic studies of hazardous waste incinerators suffer from a variety of weaknesses. For example, because these studies generally lack exposure estimates for individuals, substantial exposure misclassification can occur due to exposure heterogeneity within the exposed population. Exposure misclassification is especially problematic for studies that use an ecologic study design, because persons who experience the disease outcome of interest may have been completely unexposed (e.g., they may have recently moved to the "exposed" community). Moreover, information on potential confounders (e.g., socioeconomic differences or occupational exposure differences between exposed and unexposed groups) is typically unavailable. For comparisons of chronic disease rates by exposure status, such as the cancer studies by Elliott et al. (1992, 1996), past exposures are more pertinent than are current exposures, but historical measurements are often lacking. Sometimes the facilities have closed so that exposure measurements are not possible. In addition, the small sample sizes associated with many of these studies yield imprecise results, especially for rare outcomes.

Various epidemiologic studies have investigated possible adverse health effects associated with incinerator emissions and environmental pollutants that can be found in incinerator emissions. Because of the problems associated with the interpretation of these epidemiologic studies, firm conclusions about exposure-disease relationships are difficult and often impossible. However, there is some evidence that cancer (especially stomach cancer), respiratory diseases, and reproductive effects may be associated with environmental pollutants from incinerator emissions. Although all of the epidemiologic studies reviewed suffered from some problems and often did not yield definitive results about health risks, these studies do target human populations exposed to environmental pollutants under real-world conditions.

7.2.1 Cancer

Elliot et al. (1992) found no evidence of increased laryngeal and lung cancer in communities in Great Britain exposed to emissions from incinerators of waste solvents and oils. However, the lag periods of 5 and 10 years used for defining the at-risk period may have been

too short to allow for a cancer excess to appear, and the exposure categorization did not take into account stack height, wind patterns, or emissions abatement equipment, so exposure misclassification appears likely. A subsequent study by Elliot et al. (1996) did find evidence of modest cancer excesses (stomach, colorectal, liver, and lung) that reached nominal statistical significance among persons living within 7.5 km of waste incinerators in Great Britain. The authors felt that residual confounding from socioeconomic differences may explain most of the cancer excess. As with the 1992 study, the 1996 study did not take into account stack height, wind patterns, or emissions abatement equipment. The study by Rapiti et al. (1997) also suggested a possible excess of stomach cancer among workers in a municipal waste incinerating plant in Rome who had worked at least 10 years at the facility. The authors noted that the study had low statistical power and that the workers were fairly young, but excess stomach cancer was found nonetheless. They indicated that exposure to bacterial toxins, low socioeconomic status, and nutritional factors may have contributed to the gastric cancer excess.

7.2.2 Respiratory Disease

Feigley et al. (1994) found evidence of increased respiratory symptoms among persons living near a hazardous waste incinerator, including a symptom (morning cough) reported in a similar study by Shy et al. (1995). Persons who expressed concern about health effects from hazardous waste incineration were more likely to report symptoms than were persons with less concern, so a reporting bias may have contributed to the difference between communities. Shy et al. (1995) found a small excess of some respiratory symptoms, but most symptoms showed no excess in the exposed population. Marth et al. (1995) presented fairly persuasive evidence that municipal and hospital waste incineration in Cairo may compromise certain types of immune system function that could increase the risk of bacterial and viral infections; there was also an increase in pulmonary allergic reactions among exposed children. However, that paper included no information about how the children were selected, and there is no information about the participation rate among children/families who were asked to participate, so possible selection bias is a concern.

7.2.3 Reproductive Effects

Using retrospective estimates of air pollution levels from two incineration plants, Williams et al. (1992) found that the only district with a statistically significant different sex ratio compared to the Scottish average was the district identified a priori as having the highest exposure level. Computer mapping of the sex ratios showed aberrations where pollution levels were expected to be relatively high. Lloyd et al. (1988) found evidence that community exposure to municipal waste and/or chemical waste incinerator emissions may increase the proportion of twin births in humans and cattle. However, there were no exposure measurements available for any members of the study population, and thus there is no assurance that persons living in the high-exposure areas actually were the ones who experienced the highest exposures or that mothers of twins on average had relatively high exposures.

In addition to the above problems, studies of incinerator emissions usually cannot ascertain possible health effects associated with the individual pollutants emitted from facilities because the study population is usually exposed to several pollutants simultaneously. This aspect of incinerator studies is not necessarily a weakness in that exposure-disease associations can be

estimated for the pollutant mixture, but attempts to identify safe levels for any given pollutant are difficult at best with these studies. On the other hand, multiple pollutants could have synergistic effects with regard to adverse health effects, and studies of individual pollutants at similar exposure levels could fail to detect hazards that are present in populations exposed to a pollutant mixture.

7.3 Constituent Health Effects

This section presents (in alphabetical order) health benchmarks and supporting information on 20 of the constituents evaluated in this risk assessment. Particulate matter is discussed separately in Section 7.4.

7.3.1 Antimony

7.3.1.1 <u>Introduction</u>. Antimony is found at very low levels throughout the environment. Soil usually contains very low concentrations of antimony (less than 1 ppm). However, higher concentrations have been detected at hazardous waste sites and at antimony processing sites. Food contains small amounts of antimony: the average concentration of antimony in meats, vegetables, and seafood is 0.2 to 1.1 ppb. There are many different antimony compounds that occur naturally or are manufactured chemicals. Antimony trioxide is one example; it is found naturally in the environment and may also be produced by oxidizing antimony sulfide ore or antimony metal in air at 600° to 800° C. The most common industrial use of antimony compounds is to produce antimony trioxide for fire retardation. Persons who work in industries that process antimony ore and metal or manufacture antimony trioxide may be exposed to antimony by breathing dust or by skin contact (ATSDR, 1992a).

7.3.1.2 <u>Cancer Effects</u>. Limited data are available on the carcinogenic effects of antimony. One study in humans did not report an increased incidence of cancer in workers exposed to antimony oxide in the workplace for 9 to 31 years. Animal studies have shown conflicting results. Several studies have reported an increase in lung tumors in rats exposed by inhalation to antimony trioxide and antimony trisulfide, while other studies did not report an increase in these tumors (ATSDR, 1992a).

EPA has not classified antimony or antimony trioxide for carcinogenicity and has not calculated a unit risk estimate for antimony (U.S. EPA, 1998).

7.3.1.3 <u>Noncancer Effects</u>. The primary effects from chronic (long-term) inhalation exposure to antimony in humans are respiratory effects that include antimony pneumoconiosis (inflammation of the lungs due to irritation caused by the inhalation of dust), alterations in pulmonary function, chronic bronchitis, chronic emphysema, inactive tuberculosis, pleural adhesions, and irritation. Other effects noted in humans chronically exposed to antimony by inhalation are cardiovascular effects (increased blood pressure, altered EKG readings, and heart muscle damage) and gastrointestinal disorders (ATSDR, 1992a).

Animal studies have reported lung, cardiovascular, liver, and kidney damage from exposure to high levels of antimony by inhalation. Exposure to lower levels has resulted in eye irritation, hair loss, lung damage, and cardiovascular effects (changes in EKGs). Reproductive

effects, including failure to conceive, were reported in rats exposed to antimony trioxide by inhalation (ATSDR, 1992a).

Reference Dose. The RfD for antimony is 4.0E-04 mg/kg-d, based on a LOAEL of 0.35 mg/kg-d, an uncertainty factor of 1,000, and a modifying factor of 1. The RfD was based on a study in which 50 male and 50 female rats were administered 5 ppm potassium antimony tartrate in water (Schroeder et al., 1970, as cited in U.S. EPA, 1998). Over the period of the study, growth rates of treated animals were not affected, but male rats survived 106 and females survived 107 fewer days than did controls at median lifespans. Nonfasting blood glucose levels were decreased in treated males, and cholesterol levels were altered in both sexes. A decrease in mean heart weight for the males was noted and no increase in tumors was seen as a result of treatment. Since there was only one level of antimony administered, a NOAEL could not be established in the study. The concentration of 5 ppm antimony was expressed as an exposure of 0.35 mg/kg-d by the authors.

An uncertainty factor of 1000 was applied based on a tenfold factor for interspecies conversion, a tenfold factor to protect sensitive individuals, and an additional tenfold factor because the effect level was a LOAEL, and a NOAEL was not established (U.S. EPA, 1998).

EPA has low confidence in the study on which the RfD was based because only one species and one dose level were used, a NOAEL was not determined, and gross pathology and histopathology were not well described; low confidence in the database due to lack of adequate oral exposure investigations; and, consequently, low confidence in the RfD (U.S. EPA, 1998).

Reference Concentration. EPA has not established an RfC for antimony (U.S. EPA 1998). However, EPA has established an RfC for antimony trioxide of 2.0E-04 mg/m^3 based on a benchmark concentration (adjusted) of 0.074 mg/m^3, an uncertainty factor of 300, and a modifying factor of 1. This RfC was based on a study in which groups of 65 rats /sex/group were exposed to actual concentrations of 0, 0.06, 0.51, or 4.50 mg/m^3 antimony trioxide for 6 h/d, 5 d/wk for 1 year (Newton et al., 1994, as cited in U.S. EPA, 1998). No significant changes in hematological parameters were observed that were concentration related. An increase in cataracts was noted but a dose-response relationship was not observed. Microscopic lesions of the lungs revealed interstitial inflammation in control and exposure groups at the end of 6, 12, 18, and 24 months. This incidence was analyzed to determine a benchmark concentration. The concentrations associated with 1, 5, and 10 percent relative increases in the probability of response were estimated using both the Weibull and linear models. The lower 95 percent confidence limit for the 10 percent relative increase in probability of response was determined to be 0.87 mg/m^3 and a human equivalent concentration of 0.074 mg/m^3 was calculated (U.S. EPA, 1998).

An uncertainty factor of 300 was applied based on a tenfold factor for the protection of sensitive human subpopulations, a threefold factor for interspecies extrapolation because the dosimetric adjustments account for part of this area of uncertainty, a threefold uncertainty factor for lack of reproductive and developmental bioassays, and an additional threefold uncertainty factor to account for less-than-lifetime exposure duration, since there is no evidence that, at the lowest exposure level tested in the Newton et al. (1994) study, the levels of antimony in the rat reached a steady-state concentration (U.S. EPA, 1998).

EPA has medium confidence in the study on which the RfC was based because it was not a chronic, lifetime study; medium confidence in the database because no adequate developmental or reproductive studies are available, and consequently, medium confidence in the RfC (U.S. EPA, 1998).

Note: Risks from antimony trioxide were not evaluated in the model because HWCs are not expected to emit significant quantities of antimony trioxide based on thermodynamic considerations.

7.3.2 Arsenic

7.3.2.1 Introduction. Arsenic is a naturally occurring element in the earth's crust that is usually found combined with other elements. Arsenic combined with elements such as oxygen, chlorine, and sulfur is referred to as inorganic arsenic; arsenic combined with carbon and hydrogen is referred to as organic arsenic. In this health effects summary, arsenic refers to inorganic arsenic and its associated compounds. Organic arsenic compounds, such as arsine gas, are not discussed.

7.3.2.2 Cancer Effects. There is clear evidence that chronic exposure to inorganic arsenic in humans increases the risk of cancer. Studies have reported that inhalation of arsenic results in an increased risk of lung cancer. In addition, ingestion of arsenic has been associated with an increased risk of nonmelanoma skin cancer and bladder, liver, kidney, and lung cancer. No information is available on the risk of cancer in humans from dermal exposure to arsenic (U.S. EPA, 1998).

Animal studies have not clearly associated arsenic exposure, via ingestion exposure, with cancer. No studies have investigated the risk of cancer in animals as a result of inhalation or dermal exposure (U.S. EPA, 1998).

EPA has classified inorganic arsenic in Group A - Known Human Carcinogen. For arsenic, the Group A classification was based on the increased incidence in humans of lung cancer through inhalation exposure and the increased risk of skin, bladder, liver, kidney, and lung cancer through drinking water exposure (U.S. EPA, 1998).

An expert panel on arsenic carcinogenicity was convened by EPA in May 1997. They concluded that, “it is clear from epidemiological studies that arsenic is a human carcinogen via the oral and inhalation routes.” They also concluded that “one important mode of action is unlikely to be operative for arsenic” and that “the dose-response for arsenic at very low doses would likely be truly nonlinear, i.e., with a decreasing slope as the dose decreased. However, at very low doses such a curve might be linear with a very shallow slope, probably indistinguishable from a threshold” (U.S. EPA, 1998).

Inhalation Cancer Risk. EPA used the absolute-risk linear extrapolation model to estimate the inhalation unit risk for inorganic arsenic. Five studies on arsenic-exposed copper smelter workers were modeled for excess cancer risk (Brown and Chu, 1982, 1983a, 1983b; Enterline and Marsh, 1982; Higgins et al., 1982; Lee-Feldstein 1983; Welch et al., 1982). All five studies showed excess risks of lung cancer that were related to the intensity and duration of

exposure and the duration of the latency period. The estimates of unit risk obtained from the five studies were in reasonably good agreement, ranging from 1.25×10^{-3} to 7.6×10^{-3} $(\mu g/m^3)^{-1}$. Using the geometric mean of these data, EPA calculated an inhalation unit risk estimate of 4.3E-03 $(\mu g/m^3)^{-1}$ (U.S. EPA, 1998).

EPA did not rank their confidence in the arsenic cancer risk estimate for inhalation exposure. However, EPA stated that the studies examined a large number of people, the exposure assessments included air measurements and urinary arsenic measurements, lung cancer incidence was significantly increased over expected values, and the range of the estimates from two different exposure areas was within a factor of 6 (U.S. EPA, 1998).

Oral Cancer Risk. To estimate the risks posed by ingestion of arsenic, EPA used the data that Tseng (1977) obtained in Taiwan concerning skin cancer incidence, age, and level of exposure via drinking water. In 37 villages that had obtained drinking water for 45 years from artesian wells with various elevated levels of arsenic, 40,421 individuals were examined for hyperpigmentation, keratosis, skin cancer, and blackfoot disease (gangrene of the extremities caused by injury to the peripheral vasculature). The local well waters were analyzed for arsenic, and the age-specific cancer prevalence rates were found to be correlated with both local arsenic concentrations and age (duration of exposure). EPA used these data to calculate a unit risk estimate for arsenic. It was assumed that Taiwanese persons had a constant exposure from birth and that males consumed 3.5 liters of drinking water per day and females consumed 2.0 liters per day. Doses were converted to equivalent doses for U.S. males and females based on differences in body weights and differences in water consumption, and it was assumed that skin cancer risk in the U.S. population would be similar to that in the Taiwanese population. The multistage model with time was used to predict dose-specific and age-specific skin cancer prevalence rates associated with ingestion of inorganic arsenic. EPA calculated an oral cancer slope factor of 1.5 E+00 $(mg/kg\text{-}d)^{-1}$ with a corresponding unit risk estimate of 5.0E-05 $(\mu g/L)^{-1}$ from oral exposure to arsenic in drinking water (U.S. EPA, 1998).

The Tseng (1977) cancer data have the following limitations: (1) total arsenic exposure was uncertain because of intake from the diet and other sources, (2) there was uncertainty as to the amount of water consumed per day by Taiwanese males, (3) temporal variability of arsenic concentrations in specific wells was not known, (4) there was uncertainty concerning exposure durations, and (5) fluorescent substances were found in the water that are possible confounders or could cause synergistic effects (U.S. EPA, 1998).

7.3.2.3 Noncancer Effects. The primary effect noted in humans from chronic exposure to arsenic, through both inhalation and oral exposure, are effects on the skin. The inhalation route has resulted primarily in irritation of the skin and mucous membranes (dermatitis, conjunctivitis, pharyngitis, and rhinitis), while chronic oral exposure has resulted in a pattern of skin changes that includes the formation of warts or corns on the palms and soles, along with areas of darkened skin on the face, neck, and back. Other effects noted from chronic oral exposure include peripheral neuropathy, cardiovascular disorders, liver and kidney disorders, and blackfoot disease. No information is available on effects in humans from chronic low-level dermal exposure to arsenic (ATSDR, 1993a).

No studies are available on the chronic noncancer effects of arsenic in animals, from inhalation or dermal exposure. Oral animal studies have noted effects on the kidney and liver (ATSDR, 1993a).

Reference Dose. EPA has established an RfD for inorganic arsenic of 3.0E-04 mg/kg-d, based on a NOAEL (adjusted to include arsenic exposure from food) of 0.0008 mg/kg-d, an uncertainty factor of 3, and a modifying factor of 1 (U.S. EPA, 1998). This was based on two studies (Tseng et al., 1968, and Tseng, 1977, as cited in U.S. EPA, 1998) that showed that the prevalence of blackfoot disease increased with both age and dose for individuals exposed to high levels of arsenic in drinking water. This same population also displayed a greater incidence of hyperpigmentation and skin lesions. Other human studies support these findings, with several studies noting an increase in skin lesions from chronic exposure to arsenic through the drinking water (Cebrian et al., 1983; Hindmarsh et al., 1977; Southwick et al., 1983, as cited in U.S. EPA, 1998).

An uncertainty factor of 3 was applied to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals (U.S. EPA, 1998).

EPA has medium confidence in the studies on which the RfD was based, in the database, and in the RfD. The key studies were extensive epidemiologic reports that examined effects in a large number of people. However, doses were not well-characterized, other contaminants were present, and potential exposure from food or other sources was not examined. The supporting studies suffer from other limitations, primarily the small populations studied. However, the general database on arsenic does support the findings in the key studies; this was the basis for EPA's "medium confidence" ranking of the RfD (U.S. EPA, 1998).

Reference Concentration. EPA has not established an RfC for inorganic arsenic (U.S. EPA, 1998).

7.3.3 Barium

7.3.3.1 Introduction. Barium is a naturally occurring element that is found in the earth's crust. Barium enters the environment primarily through the weathering of rocks and minerals. The general population is exposed to barium usually at low levels, through consumption of drinking water and foods. Barium and its compounds are used in automotive paints, stabilizers for plastics, and jet fuel (ATSDR, 1990a).

7.3.3.2 Cancer Effects. Limited human data are available on the carcinogenicity of barium. The only available studies involve a single topical application of barium chloride to the cervix of one woman. These studies reported a number of cell transformations in the cervix; however, 1 to 2 weeks after the application, these cellular alterations were no longer observed (U.S. EPA, 1998).

Two chronic oral animal studies evaluated the carcinogenicity of barium in rats and mice. No statistically significant increases in the incidences of tumors were observed in the barium-exposed rats (U.S. EPA, 1998).

EPA has classified barium in Group D - Not Classifiable as to Human Carcinogenicity. This was based on the availability of adequate chronic oral studies in rats and mice that have not demonstrated carcinogenic effects but a lack of adequate inhalation studies (U.S. EPA, 1998).

EPA has not calculated a unit risk estimate for barium (U.S. EPA, 1998).

7.3.3.3 Noncancer Effects. Hypertension has been noted in humans who ingested high doses of barium and workers who inhaled dusts of barium ores and barium carbonate (U.S. EPA, 1998). Other effects noted in humans from chronic exposure include musculoskeletal effects, such as progressive muscle weakness, and neurological effects, including numbness and tingling around the mouth and neck (ATSDR, 1990a).

Chronic, oral exposure to barium in experimental animals has resulted in increases in blood pressure and kidney effects (ATSDR, 1990a; U.S. EPA, 1998).

Reference Dose. EPA has calculated an RfD for barium of 7.0E-02 mg/kg-d based on a NOAEL (adjusted) of 0.21 mg/kg-d, an uncertainty factor of 3, and a modifying factor of 1. This was based on several epidemiological studies that investigated the effects of elevated levels of barium in drinking water (Brenniman and Levy, 1984; Wones et al., 1990, as cited in U.S. EPA, 1998). Wones et al. (1990) found no increases in systolic or diastolic blood pressure in subjects who consumed drinking water containing barium at levels ranging from 0 to 10 mg/L for 10 weeks. Brenniman and Levy (1984) conducted a retrospective epidemiology study that compared mortality and morbidity rates in populations ingesting elevated barium levels (2 to 10 mg/L) in their drinking water to populations ingesting very little or no barium (less than or equal to 0.2 mg/L). Differences in mortality rates from all cardiovascular diseases were significantly higher in the communities with elevated barium. However, these differences were largely in the 65 and over age group and did not account for confounding variables such as population mobility or use of water softeners or medication. In addition, several rat studies that reported increased kidney weights in rats exposed to barium in drinking water for 13 weeks or 2 years were considered (NTP, 1994, as cited in U.S. EPA, 1998). NOAELs of 45 and 65 mg/kg-d were selected from these studies (U.S. EPA, 1998).

An uncertainty factor of 3 was applied to account for potential differences between adults and children and the existence of adequate developmental toxicity studies (U.S. EPA, 1998).

EPA has medium confidence in the principal studies used as the basis for the RfD because LOAELs for cardiovascular and kidney disease were not identified in the human studies. However, the animal studies provided information regarding NOAELs and LOAELs for kidney effects of barium, but cardiovascular effects did not occur in these studies. EPA has medium confidence in the database because of the existence of subchronic and chronic human studies, suchronic and chronic animal studies in more than one species, and a reproductive/developmental study in rats and mice. EPA has medium confidence in the RfD as well (U.S. EPA, 1998).

Reference Concentration. EPA has not calculated an RfC for barium (U.S. EPA, 1998). However, EPA has calculated a provisional RfC of 5.0E-04 mg/m^3 for barium (U.S. EPA, 1997b). This was based on a 4-month reproductive study in rats in which a NOAEL of 0.8 mg/m^3 was selected (Tarasenko et al. 1977, as cited in U.S. EPA, 1997b).

7.3.4 Beryllium

7.3.4.1 Introduction. Pure beryllium is a hard gray metal that does not occur naturally but does occur as a chemical component of certain kinds of rocks, coal and oil, soil, and volcanic dust. Two kinds of mineral rocks, bertrandite and beryl, are mined commercially for the recovery of beryllium. Beryllium is also found combined with other elements such as fluoride, chlorine, sulfur, oxygen, and phosphorus (ATSDR, 1993b).

7.3.4.2 Cancer Effects. Several human epidemiological studies have shown increases in lung cancer in beryllium-processing workers (U.S. EPA, 1998).

Beryllium compounds have been shown to cause lung cancer in rats and monkeys from inhalation exposure and lung tumors in rats exposed by intratracheal instillation. Osteosarcomas have been produced in rabbits and in mice by intravenous and intramedullary injection. Oral exposure to beryllium in animals has not resulted in a statistically significant increased incidence of tumors (U.S. EPA, 1998).

EPA has classified beryllium in Group B1 - Probable Human Carcinogen. This classification was based on limited evidence of lung cancer in humans exposed to airborne beryllium and sufficient evidence of carcinogenicity in animals (lung cancer in rats and monkeys inhaling beryllium, lung tumors in rats exposed via intratracheal instillation, and osteosarcomas in rabbits and possibly mice receiving intravenous injection) (U.S. EPA, 1998).

Inhalation Cancer Risk. EPA used the relative risk extrapolation model, based on an epidemiologic study (Wagoner et al., 1980, as cited in U.S. EPA, 1998) to estimate the inhalation unit cancer risk for beryllium. EPA calculated an inhalation unit risk estimate of 2.4 E-03 $(\mu g/m^3)^{-1}$ (U.S. EPA, 1998).

This cancer risk estimate was based on an epidemiologic study having several confounding factors, including the lack of individual exposure monitoring or job history data. Newer studies are currently under peer review and may be used in the future by EPA to derive a revised unit risk estimate (U.S. EPA, 1998).

Oral Cancer Risk. EPA has not calculated an oral unit risk estimate for beryllium because the oral database is considered inadequate for the assessment of carcinogenicity (U.S. EPA, 1998).

7.3.4.3 Noncancer Effects. The major effect from chronic inhalation exposure to beryllium in humans is chronic beryllium disease (berylliosis), in which granulomatous lesions (noncancerous) develop in the lung. The onset of these effects may be delayed by 3 months to more than 20 years. Symptoms of chronic beryllium disease include irritation of the mucous membranes, reduced lung capacity, shortness of breath, fatigue, anorexia, dyspnea, malaise, and weight loss. Chronic beryllium disease may cause death in severe cases. No information is available on the effects of beryllium in humans from chronic oral exposure; a skin allergy may result from chronic dermal exposure to beryllium (ATSDR, 1993b).

Animal studies have also reported effects on the lung, such as chronic pneumonitis, from chronic inhalation exposure to beryllium. Effects on the adrenal gland and immune system have been noted in animals chronically exposed by inhalation. No effects were observed in the lung, heart, blood, liver, or kidney from chronic oral exposure to beryllium in animals. Chronic dermal exposure to beryllium in animals has resulted in effects on the immune system (ATSDR, 1993b).

Reference Dose. EPA has established an RfD for beryllium of 2.0E-03 mg/kg-d, based on a benchmark dose of 0.46 mg/kg-d, an uncertainty factor of 300, and a modifying factor of 1 (U.S. EPA, 1998). This was based on a study (Morgareidge et al., 1976, as cited in U.S. EPA, 1998) in which groups of five male and five female beagle dogs were fed diets containing 0, 5, 50, or 500 ppm beryllium for 172 weeks. Lesions in the small intestine and hypoplasia of the bone marrow were observed. Dose-response modeling of the data for small intestinal lesions in dogs was used to determine a benchmark dose.

An uncertainty factor of 300 was applied based on a tenfold factor for extrapolation for interspecies differences, a tenfold factor for intraspecies variation, and a threefold factor for database deficiencies (U.S. EPA, 1998).

EPA has medium confidence in the study on which the RfD was based, because the study was administered by a relevant route (oral), at multiple dose levels, and for a chronic duration. However, there were a small number of animals, early mortality at the high-dose level, and no measure of immune response or function. EPA has low to medium confidence in the database because there is only one chronic study in dogs showing adverse effect levels; other chronic studies in rodents demonstrated NOAELs at the highest doses tested; consequently, EPA has low-to-medium confidence in the RfD (U.S. EPA, 1998).

EPA also stated that the major areas of scientific uncertainty in the RfD assessment are the lack of chronic oral studies establishing LOAELs, the lack of a chronic oral study examining immunologic endpoints, the lack of critical effects in humans by inhalation as identified in dogs and the lack of sensitive indicators for rickets, the lack of reproductive and developmental studies, and the lack of human toxicity information (U.S. EPA, 1998).

Reference Concentration. EPA has established an RfC for beryllium of 2.0E-05 mg/m^3 based upon two human studies that examined beryllium sensitization and progression to chronic beryllium disease (Kreiss et al., 1996; Eisenbud et al., 1949, as cited in U.S. EPA, 1998). A LOAEL (human equivalent concentration) of 0.20 µg/m^3 was identified from the Kreiss et al. (1996) study and a NOAEL (human equivalent concentration) of 0.01 to 0.1 µg/m^3 was identified from the Eisenbud et al. (1949) study. An uncertainty factor of 10 and a modifying factor of 1 were applied (U.S. EPA, 1998).

Kreiss et al. (1996) examined beryllium workers in a plant that made beryllia ceramics from beryllium oxide powder. The study found an increased beryllium sensitization rate among machinists exposed to an average concentration of beryllium of 0.55 µg/m^3. Eisenbud et al. (1949) evaluated beryllium exposure for 11 cases of chronic beryllium disease in a community located near a beryllium production plant.

An uncertainty factor of 10 was applied based on a threefold uncertainty factor to account for the poor quality of exposure monitoring in the co-principal studies and other epidemiology studies that assessed the incidence of beryllium sensitization and chronic beryllium disease among exposed workers and community residents, and an additional threefold uncertainty factor was applied to account for the sensitive nature of the endpoint (beryllium sensitization) (U.S. EPA, 1998).

EPA has medium confidence in the study on which the RfC was based because it is an occupational study performed on a moderate-to-large-sized group in which sensitive measures were used to identify the affected population. However, there was poor quality monitoring in the co-principal studies. EPA also has medium confidence in the database due to a lack of adequate exposure monitoring in the epidemiology studies, and some uncertainty regarding the mechanisms associated with the progression to chronic beryllium disease in beryllium-sensitized individuals. Confidence in the RfD was also medium, reflecting the other classifications (U.S. EPA, 1998).

7.3.5 Cadmium

7.3.5.1 Introduction. Cadmium is a soft, silver-white metal that occurs naturally in the earth's crust and is usually found in combination with other elements such as oxygen, chlorine, or sulfur. The major uses of cadmium are in the manufacture of pigments and batteries and in the metal-plating and plastics industries. Most of the cadmium used in this country is obtained as a byproduct from the smelting of zinc, lead, or copper ores (ATSDR, 1997a).

7.3.5.2 Cancer Effects. Several occupational studies have reported an excess risk of lung cancer from exposure to inhaled cadmium. However, the evidence is limited rather than conclusive due to confounding factors such as the presence of other carcinogens and smoking. Studies of human ingestion to cadmium are inadequate to assess its carcinogenicity (U.S. EPA, 1998).

Animal studies have reported lung cancer resulting from inhalation exposure to several forms of cadmium, while animal ingestion studies have not reported cancer from exposure to cadmium compounds (U.S. EPA, 1998).

EPA has classified cadmium in Group B1 - Probable Human Carcinogen based on human studies showing a possible association between cadmium exposure and lung cancer, and animal studies showing an increased incidence of lung cancer (U.S. EPA, 1998).

Inhalation Cancer Risk. EPA used the two-stage extrapolation model based on data from an occupational study of workers exposed to cadmium (Thun et al., 1985, as cited in U.S. EPA, 1998) to estimate the inhalation unit risk estimate for cadmium. EPA calculated an inhalation unit risk estimate of 1.8E-03 $(\mu g/m^3)^{-1}$ (U.S. EPA, 1998).

EPA used human data to develop the risk estimate for cadmium because the data were derived from a relatively large cohort, and the effects of arsenic and smoking were accounted for in the quantitative analysis of cadmium's effects. EPA also calculated an inhalation unit risk of 9.2×10^{-2} $(\mu g/m^3)^{-1}$ for cadmium based on animal data (Takenda et al., 1983, as cited in U.S. EPA, 1998). This estimate was higher than that derived from human data and thus more conservative. However, EPA felt that the use of the available human data was more reliable because of species variations in response and the type of exposure (U.S. EPA, 1998).

Oral Cancer Risk. EPA has not calculated an oral unit risk estimate for cadmium (U.S. EPA, 1998).

7.3.5.3 Noncancer Effects. The kidney appears to be the main target organ in humans following chronic inhalation exposure to cadmium. Abnormal kidney function, indicated by proteinuria and a decrease in glomerular filtration rate, and an increased frequency of kidney stone formation are some of the effects noted. Respiratory effects, such as bronchitis and emphysema, have also been noted in humans chronically exposed to cadmium through inhalation. Oral exposure to cadmium in humans also results in effects on the kidney, with effects similar to those seen following inhalation exposure. In humans, dermal exposure to cadmium does not appear to cause allergic reactions (ATSDR, 1997a).

Animal studies have reported effects on the kidney, liver, lung, and blood from chronic inhalation exposure to cadmium. Chronic oral exposure to cadmium in animals results in effects on the kidney, bone, immune system, blood, and nervous system. No information is available on chronic dermal exposure to cadmium in animals (ATSDR, 1997a).

Reference Dose. EPA has established two RfDs for cadmium: one for cadmium ingested in drinking water and one for cadmium ingested in food. The RfD for cadmium in drinking water is 5.0E-04 mg/kg-d and the RfD for dietary exposure to cadmium is 1.0E-03 mg/kg-d. These RfDs were based on a number of human studies that showed kidney effects (significant proteinuria) from chronic exposure to cadmium. Both RfDs were calculated based on the highest level of cadmium in the human renal cortex (200 µg/g) that was not associated with the critical effect, i.e., significant proteinuria (U.S. EPA, 1985, as cited in U.S. EPA, 1998). A toxicokinetic model was then used to determine the NOAEL. This model took into account the difference in absorption between drinking water and food. The NOAELs for water and food were calculated to be 0.005 mg/kg-d and 0.01 mg/kg-d, respectively. The RfDs were calculated by applying an uncertainty factor of 10 and a modifying factor of 1 to each NOAEL (U.S. EPA, 1998).

An uncertainty factor of 10 was applied to account for intrahuman variability to the toxicity of cadmium in the absence of data on sensitive individuals (U.S. EPA, 1998).

EPA has high confidence in the studies and the database on which the RfDs were based. The RfDs were not based on a single study, but rather on data obtained from many studies on the toxicity of cadmium in humans and animals. These data permit calculation of pharmacokinetic parameters of cadmium absorption, distribution, metabolism, and elimination (U.S. EPA, 1998).

Reference Concentration. EPA has not established an RfC for cadmium.

7.3.6 Chlorine

7.3.6.1 Introduction. Chlorine is a greenish-yellow gas that has a suffocating odor. In water, chlorine reacts to form hypochlorous acid and hypochlorite ion. Chlorine is added to drinking water for disinfection purposes and is also used as an intermediate in the manufacture and preparation of a number of products, such as antifreeze, cleaning agents, and pharmaceuticals (U.S. EPA, 1994a).

7.3.6.2 Cancer Effects. No information is available on the carcinogenic effects of chlorine in humans from inhalation exposure. Several human studies have investigated the relationship between exposure to chlorinated drinking water and cancer. These studies were not designed to assess whether chlorine itself causes cancer, but whether trihalomethanes or other organic compounds occurring in drinking water are associated with an increased risk of cancer. These studies show an association between bladder and rectal cancer and chlorinated byproducts in drinking water (U.S. EPA, 1994a).

Chlorine has not been found to be carcinogenic in animals. No tumors were found in rats exposed to chlorine in their drinking water over their lifetime (U.S. EPA, 1994a).

EPA has not classified chlorine for carcinogenicity or calculated a unit risk estimate for chlorine (U.S. EPA, 1998).

7.3.6.3 Noncancer Effects. Chlorine is a potent irritant in humans to the eyes, upper respiratory tract, and the lung. It is also extremely irritating to the skin and can cause severe burns (U.S. EPA, 1994a).

Animal studies have reported decreased body weight gain, eye and nose irritation, and effects on the respiratory tract, liver, and kidney from inhalation exposure to chlorine. No significant effects have been observed in animal studies from oral exposure to chlorine (U.S. EPA, 1994a).

Acute Toxicity Value. EPA proposed an acute toxicity value of 0.5 ppm for chlorine. This value was derived based on data in human volunteers in which no significant sensory irritation or pulmonary effects were associated with 4- or 8-h exposures to 0.5 ppm chlorine (Talmage, 1996; Rotman et al., 1983). The dose-response relationship for irritant gases follows the following equation:

$$C^n \times t = k,$$

where C = concentration, time is time, and k is a constant. For chlorine, n = 2 (ten Berge et al., 1986). Uncertainty factors were not applied since a no-effect-level was identified in humans.

EPA has high confidence in the acute toxicity values for chlorine because the study in human volunteers was well conducted and well documented. In addition, both sensory irritation and pulmonary function parameters were measured in both males and females. Also, the exposure concentrations were measured by several different methods and all of them gave similar results.

Reference Dose. EPA has not established an RfD for chlorine.

Reference Concentration. EPA has not calculated an RfC for chlorine. However, an interim chronic RfC for chlorine of 0.001 mg/m^3 has been calculated based on a lifetime inhalation study in rats and mice (Wolf et al., 1995). In this study, groups of male and female rats and mice were exposed to 0, 0.4, 1.0, or 2.5 ppm chlorine gas for 6 hours per day, 5 days per week (mice and male rats) or 3 days per week (female rats) for 2 years. The study reported several exposure-dependent lesions of the nasal passages in all sex and species groups, including respiratory and olfactory epithelial degeneration, septal fenestration, mucosal inflammation, respiratory epithelial hyperplasia, squamous metaplasia, and other effects. No effects were observed in the larynx or lower respiratory tract (Wolf et al., 1995). Although several statistically significant effects were reported at the lowest exposure concentration, the severity of the lesions were generally judged to be slight to minimal. The changes seen at the lowest exposure concentration are of questionable clinical significance; therefore, 0.4 ppm (1.2 mg/m^3) was considered a NOAEL in mice.

The NOAEL was adjusted for duration of exposure ($NOAEL_{ADJ}$). The $NOAEL_{ADJ}$ was converted to a human equivalent concentration NOAEL ($NOAEL_{HEC}$) based on effects in the extrathoracic region by a category 1 gas in accordance with EPA (1994d) guidelines (equation 4-18 in U.S. EPA, 1994d). A $NOAEL_{HEC}$ of 0.04 mg/m^3 was calculated. An uncertainty factor (UF) of 30 was applied based on a factor of 3 for interspecies extrapolation and a factor of 10 to account for sensitive individuals, resulting in an interim RfC of 0.001 mg/m^3. These calculations were performed as shown below:

$$NOAEL_{ADJ} = 1.2\ mg/m^3 \times 6\ h/24\ h \times 5\ d/7\ d = 0.21\ mg/m^3.$$

$$\begin{aligned} NOAEL_{HEC} &= NOAEL_{ADJ} \times RGDR \\ &= NOAEL_{ADJ} \times [V_E/SA_{ET}]_A/[V_E/SA_{ET}]_H \\ &= 0.21\ mg/m^3 \times [0.06\ /3]/[20/200] = 0.04\ mg/m^3 \end{aligned}$$

where RGDR = regional gas dose ratio, V_E = minute volume, and SA_{ET} = surface area of extrathoracic region (ET) for the mouse (A) and human (H). $(V_E)_A$ = 0.06 m^3/d, $(V_E)_H$ = 20 m^3/d, $(SA_{ET})_A$ = 3 cm^2, $(SA_{ET})_H$ = 200 cm^2 (U.S. EPA 1994d).

$$\text{interim RfC} = NOAEL_{HEC} \div UF = 0.04\ mg/m^3 \div 30 = 0.001\ mg/m^3$$

EPA has low confidence in the interim chronic RfC for chlorine because tissue dosimetry and susceptibility differences in rodents complicate extrapolation from animal results to humans. There are also some questions regarding the significance of the effects reported and whether or not the low dose should be considered a NOAEL or LOAEL.

7.3.7 Chromium

7.3.7.1 Introduction. Chromium is a metallic element that occurs in the environment in two major valence states: trivalent chromium (chromium III) and hexavalent chromium (chromium VI). Chromium (VI) compounds are much more toxic than chromium (III) compounds; chromium (III) is an essential element in humans, with a daily intake of 50 to

200 µg/d recommended for an adult. Chromium (VI) is quite toxic; however, the human body can detoxify some amount of chromium (VI) to chromium (III) (ATSDR, 1993c).

7.3.7.2 Cancer Effects. Epidemiological studies of workers have clearly established that inhaled chromium is a human carcinogen, resulting in an increased risk of lung cancer. These studies were not able to differentiate between exposure to chromium (III) and chromium (VI) compounds. No information is available on cancer in humans from oral or dermal exposure to chromium (ATSDR, 1993c; U.S. EPA 1998).

Animal studies have shown chromium (VI) to cause lung tumors via inhalation exposure. No studies are available that investigated cancer in animals from oral or dermal exposure to chromium (VI). Chromium (III) has been tested in mice and rats by the oral route, with several studies reporting no increase in tumor incidence. No studies are available on cancer in animals from inhalation or dermal exposure to chromium (III) (ATSDR, 1993c; U.S. EPA, 1998).

EPA has classified chromium (VI) in Group A -Known Human Carcinogen, because results of occupational epidemiologic studies show a dose-response relationship for chromium exposure and lung cancer. Since the human studies could not differentiate between chromium (III) and chromium (VI) exposure and only chromium (VI) was found to be carcinogenic in animal studies, EPA concluded that only chromium (VI) should be classified as a human carcinogen (U.S. EPA, 1998). EPA has not classified chromium (III) for carcinogenicity (U.S. EPA, 1998).

Inhalation Cancer Risk. EPA used the multistage extrapolation model, based on data from an occupational study of chromate production workers (Mancuso, 1975, as cited in U.S. EPA, 1998) to estimate the unit cancer risk for chromium (VI). EPA calculated an inhalation unit risk estimate of 1.2E-02 $(\mu g/m^3)^{-1}$ (U.S. EPA, 1998). EPA has not calculated a risk estimate from inhalation exposure to chromium (III) (U.S. EPA, 1998).

EPA has confidence in the risk estimate for chromium (VI) because results of studies of chromium exposure are consistent across investigators and countries, and a dose-response for lung tumors has been established. However, an overestimation of risk may be due to the implicit assumption that the smoking habits of chromate workers were similar to those of the general white male population, since it is generally accepted that the proportion of smokers is higher for industrial workers than for the general population (U.S. EPA, 1998).

Oral Cancer Risk. EPA has not calculated a risk estimate from oral exposure to chromium (VI) or chromium (III) (U.S. EPA, 1998).

7.3.7.3 Noncancer Effects. Chronic inhalation exposure to chromium (VI) in humans results in effects on the respiratory tract, with perforations and ulcerations of the septum, bronchitis, decreased pulmonary function, pneumonia, asthma, and nasal itching and soreness reported. Chronic exposure to high levels of chromium (VI) by inhalation or oral exposure may also produce effects on the liver, kidney, gastrointestinal and immune systems, and possibly the blood. Dermal exposure to chromium (VI) may cause contact dermatitis, sensitivity, and ulceration of the skin (ATSDR, 1993c).

Limited information is available on the chronic effects of chromium in animals. The available data indicate that, following inhalation exposure, the lung and kidney have the highest tissue levels of chromium. No effects were noted in several oral animal studies with chromium (VI) and chromium (III) (ATSDR, 1993c).

Reference Dose. EPA has established an RfD for chromium (VI) of 5.0E-03 mg/kg-d, based upon a NOAEL (adjusted) of 2.4 mg/kg-d, an uncertainty factor of 500, and a modifying factor of 1 (U.S. EPA, 1998). This was based on a study in rats (MacKenzie et al., 1958, as cited in U.S. EPA, 1998) that reported no adverse effects after exposure to chromium (VI) in the drinking water for 1 year. Other studies support these findings; one study reported no significant effects in female dogs given chromium (VI) in the drinking water for 4 years and a case study in humans reported no adverse health effects in a family of four who drank water for 3 years from a private well containing chromium (VI) at 1 mg/L (U.S. EPA, 1998).

An uncertainty factor of 500 was applied based on two tenfold factors to account for both the expected interhuman and interspecies variability in the toxicity of the chemical in lieu of specific data and an additional fivefold factor to compensate for the less-than-lifetime exposure duration of the principal study (U.S. EPA, 1998).

EPA has low confidence in the study on which the RfD for chromium (VI) was based, in the database, and in the RfD. Confidence in the key study was ranked low due to the small number of animals tested, the small number of parameters measured, and the lack of toxic effects at the highest dose tested. Confidence in the database was also ranked low because the supporting studies are of equally low quality and teratogenic and reproductive endpoints are not well studied, thus a low confidence in the RfD follows (U.S. EPA, 1998).

The RfD for chromium (III) is 1.0E+00 mg/kg-d, based on a NOAEL (adjusted) of 1,468 mg/kg-d, an uncertainty factor of 1,000, and a modifying factor of 1 (U.S. EPA, 1998). This was based on no effects observed in rats fed chromium (III) in the diet for 2 years (Ivankovic and Preussman, 1975, as cited in U.S. EPA, 1998). In this study, groups of 60 male and female rats were fed chromic oxide in the diet for 600 feedings. All major organs were examined histologically, and no effects due to chromium treatment were observed at any dose level. This study also included a 90-day study, where the only effects observed were reductions in the absolute weights of the livers and spleens in animals in the high-dose group.

An uncertainty factor of 1,000 was applied based on two tenfold factors to account for both the expected interhuman and interspecies variability in the toxicity of the chemical in lieu of specific data, and an additional tenfold factor was applied to reflect uncertainty in the NOAEL because the effects observed in the 90-day study were not explicitly addressed in the 2-year study, the absorption of chromium is low, the animals were allowed to die naturally after feeding stopped (2 years), and only then was histology performed (U.S. EPA, 1998).

EPA has low confidence in the study on which the RfD was based, in the database, and in the RfD. The low ranking of the key study was due to the lack of explicit detail on study protocol and results, the low ranking of the database was due to the lack of supporting data, and the low ranking of the RfD was due to the lack of an observed effect level in the key study (U.S. EPA, 1998).

Reference Concentration. EPA has not established an RfC for chromium (III) or chromium (VI) (U.S. EPA, 1998).

7.3.8 Cobalt

7.3.8.1 Introduction. Cobalt occurs naturally in the environment in most rocks, soil, water, plants, and animals. Cobalt is used in superalloys, magnetic alloys, and cutting- and water-resistant alloys, as a drier in paint, a catalyst, for porcelain enameling of steel bathroom fixtures and appliances, in pigment manufacture, and as a feed and nutritional additive. Cobalt is an essential element in humans and animals as a constituent of vitamin B_{12}. Cobalt has also been used as a treatment for anemia, because it stimulates red blood cell production (ATSDR, 1992b; NLM, 1999).

7.3.8.2 Cancer Effects. Limited data are available on the carcinogenic effects of cobalt. In one study on workers who refined and processed cobalt and sodium, an increase in deaths due to lung cancer was found for workers exposed only to cobalt. However, when this study was controlled for date of birth, age at death, and smoking habits, the difference in deaths due to lung cancer was found not to be statistically significant. In another study assessing the correlation between cancer deaths and trace metals in water supplies in the United States, no correlation was found between cancer mortality and the level of cobalt in the water (ATSDR, 1992b).

In an animal study, inhalation of cobalt over a lifetime did not increase the incidence of tumors in hamsters. Cobalt, via direct injection (intramuscular and subcutaneous under the muscles or skin) has been reported to cause tumors at the injection site in animals (ATSDR, 1992b; NLM, 1999).

EPA has not classified cobalt for carcinogenicity or calculated a unit risk estimate for cobalt.

7.3.8.3 Noncancer Effects. Acute exposure to cobalt in humans has been reported to result in cough, dyspnea, decreased pulmonary function, weight loss, diffuse nodular fibrosis, and respiratory hypersensitivity. Contact with cobalt in humans has resulted in dermatitis, with eruptions of the erythematous papular type on the ankles, elbows, and neck (NLM, 1998).

Chronic exposure to cobalt by inhalation in humans also results in effects on the respiratory system, such as respiratory irritation, wheezing, asthma, pneumonia, and fibrosis. Other effects noted from inhalation exposure to cobalt in humans include cardiac effects, such as functional effects on the ventricles and enlargement of the heart; congestion of the liver, kidneys, and conjunctiva; and immunological effects that include cobalt sensitization, which can precipitate an asthmatic attack in sensitized individuals (ATSDR, 1992b).

Cardiovascular effects (cardiomyopathy) were observed in people who consumed large amounts of beer over several years containing cobalt sulfate as a foam stabilizer. The effects were characterized by cardiogenic shock, sinus tachycardia, left ventricular failure, and enlarged hearts. Gastrointestinal effects (nausea, vomiting, and diarrhea), effects on the blood, liver injury, and allergic dermatitis have also been reported in humans from oral exposure to cobalt (ATSDR, 1992b).

Animal studies have reported decreased body weight, necrosis of the thymus, and effects on the blood, liver, kidneys, and respiratory, cardiovascular, and central nervous system from inhalation exposure to cobalt (ATSDR, 1992b). Acute oral cobalt toxicity has been demonstrated in some animals; at doses higher than 5 mg/kg of diet/day in chickens and sheep, loss of appetite, loss of weight, and debilitation were observed (NLM, 1999).

Reference Dose. EPA has established a provisional RfD for cobalt of 6.0E-2 mg/kg/d based on the upper range of average intake in children, which is below the levels of cobalt necessary to induce polycythemia in either renally compromised patients or normal patients (U.S. EPA, nd).

Reference Concentration. EPA has not established an RfC for cobalt.

7.3.9 Copper

7.3.9.1 Introduction. Copper occurs naturally in rock, soil, water, sediment, and air and is an essential element for humans. It is extensively mined and processed in the United States and is primarily used as the metal or alloy in the manufacture of wire and sheet metal, in agriculture to treat plant diseases, and as a preservative for wood, leather, and fabrics (ATSDR, 1989).

7.3.9.2 Cancer Effects. An increased incidence of cancer has not been observed in humans or animals exposed to copper via inhalation, oral, or dermal routes (ATSDR, 1989). In laboratory animal studies, two strains of mice administered copper for 53 weeks failed to show any evidence of statistically significant increases in tumor incidence (U.S. EPA, 1998).

EPA has classified copper in Group D - Not Classifiable as to Human Carcinogenicity, based on no human data, inadequate animal data, and equivocal mutagenicity data (U.S. EPA, 1998).

7.3.9.3 Noncancer Effects. The majority of information on copper toxicity in humans involves the consumption of water contaminated with high levels of copper or suicide attempts using copper sulfate. Effects observed in humans include gastrointestinal, hepatic, and immunological (from dermal exposure) and respiratory effects (from inhalation exposure). An example of significant (but rare) copper toxicity in humans is Wilson's Disease, an autosomal recessive disorder that affects normal copper homeostasis. The disease is characterized by excessive retention of hepatic copper, decreased concentration of plasma ceruloplasmin, and impaired biliary excretion (ATSDR, 1989).

Longer-term or chronic human exposure to copper has been associated with a number of effects including metal fume fever and enlarged livers and spleens. Metal fume fever is characterized by chills, fever, aching muscles, dryness in the mouth and throat, and headaches that last for 1 or 2 days. Anorexia, nausea, and occasional diarrhea in factory workers exposed to high concentrations of airborne copper have also been reported (ATSDR, 1989).

The effects observed in animals from exposure to high levels of copper include gastrointestinal, hepatic, hematologic, immunologic, and developmental effects (ATSDR, 1989).

Copper is an essential dietary nutrient for which a recommended daily allowance (RDA) has been developed. Copper is needed for human hemoglobin formation, carbohydrate metabolism, catecholamine biosynthesis, and cross-linking of collagen, elastin, and hair keratin. Copper is also essential for incorporation into copper-dependent enzymes. An RDA of 2 to 3 mg copper/d is recommended by the National Academy of Sciences (ATSDR, 1989).

EPA has not established an RfC or RfD for copper (U.S. EPA, 1998).

7.3.10 Hydrogen Chloride

7.3.10.1 Introduction. Hydrogen chloride (liquid) is an aqueous solution of hydrogen chloride gas and is commercially available in several concentrations and purities. Because of impurities, commercial varieties of hydrogen chloride are generally yellow. Hydrogen chloride is used in the refining of metal ore, as a lab reagent, and in the removal of scale from boilers (Budavari, 1989).

7.3.10.2 Cancer Effects. No information is available on the carcinogenic effects of hydrogen chloride in humans or animals. EPA has not classified hydrogen chloride for carcinogenicity (U.S. EPA, 1998).

7.3.10.3 Noncancer Effects. The acute effects on humans exposed by inhalation to hydrogen chloride include coughing, choking, and inflammation and ulceration of the respiratory tract, chest pain, and pulmonary edema. Oral exposure may result in corrosion of the mucous membranes, esophagus, and stomach, with nausea, vomiting, intense thirst, and diarrhea. Dermal contact with hydrogen chloride can cause burns, ulcerations, and scarring. Cases of gastritis, chronic bronchitis, dermatitis, and photosensitization have been reported among individuals exposed occupationally to hydrogen chloride (NLM, 1998).

In animals, the only study of the effects of long-term inhalation of hydrogen chloride reported epithelial or squamous hyperplasia of the nasal mucosa, larynx, and trachea. In a 90-day inhalation study, decreased body weight gains, minimum-to-mild rhinitis, nasal cavity lesions, and eosinophilic globules in the epithelial lining of the nasal tissues were reported in test animals (U.S. EPA, 1998).

Acute Toxicity Value. EPA proposed an acute toxicity value of 1.4 ppm for hydrogen chloride. This value was derived based on data in human volunteers exposed to 0, 0.8, or 1.8 ppm hydrogen chloride for 45 minutes. The volunteers rated the following symptoms: sore throat, nasal discharge, cough, chest pain, wheezing, fatigue, headache, dizziness, and unusual taste or smell. Respiratory parameters such as total respiratory resistance and forced vital capacity were also measured. No adverse exposure-related effects were observed (Stevens et al., 1992). The dose-response relationship for irritant gases follows the following equation: $C^n \times t = k$, where C = concentration, time is time, and k is a constant. For hydrogen chloride, n =1 (ten Berge et al., 1986). Uncertainty factors were not applied since a no-effect-level was identified in humans.

EPA has low confidence in the acute toxicity value for hydrogen chloride because the study group was limited to 10 subjects with a narrow age distribution. In addition, it is unclear

whether the pulmonary function test used in the study was a sensitive measure of the effects of hydrogen chloride.

Reference Dose. EPA has not established an RfD for hydrogen chloride (U.S. EPA, 1998).

Reference Concentration. EPA has established an RfC for hydrogen chloride of 2.0E-02 mg/m^3 based on a LOAEL (human equivalent concentration) of 6.1 mg/m^3, an uncertainty factor of 300, and a modifying factor of 1 (U.S. EPA, 1998). The RfC was based on a chronic rat inhalation study that reported an increased incidence of hyperplasia of the nasal mucosa as well as the laryngeal-tracheal segments in the group exposed to hydrochloric acid (Sellakumar et al., 1985, as cited in U.S. EPA, 1998).

An uncertainty factor of 300 was applied based on a tenfold factor for intraspecies extrapolation, a tenfold factor to extrapolate from a LOAEL to a NOAEL, and a threefold factor for interspecies differences (U.S. EPA, 1998).

EPA has low confidence in the chronic study on which the RfC was based because it used only one dose and limited toxicological measurements. Confidence in the database is also low because the supporting data consisted of two subchronic bioassays and the database does not provide any additional chronic or reproductive studies. Therefore, EPA's confidence in the RfC is also low (U.S. EPA, 1998).

7.3.11 Lead

7.3.11.1 Introduction. Lead is a naturally occurring, bluish-gray metal that is found in small quantities in the earth's crust. It is present in a variety of compounds such as lead acetate, lead chloride, lead chromate, lead nitrate, and lead oxide (ATSDR, 1997b).

Exposure to lead can occur through the air, drinking water, food, and soil. Most lead exposure occurs through a combination of the inhalation and oral routes, with inhalation generally contributing a greater proportion of the dose for occupationally exposed groups, and the oral route generally contributing a greater proportion for the general population. The effects of lead are the same regardless of the route of exposure (inhalation or oral) and are correlated with internal exposure as blood lead levels. For this reason, the discussion in this summary will not discuss lead exposure in terms of route, but will present it in terms of blood lead levels (ATSDR, 1997b).

Children are at particular risk to lead exposure since they commonly put hands, toys, and other items, that may come in contact with lead-containing dust and dirt in their mouths. In addition, lead-based paints were commonly used for many years and flaking paint, paint chips, and weathered paint powder may be a major source of lead exposure, particularly for children (ATSDR, 1997b).

7.3.11.2 Cancer Effects. Human studies are inconclusive regarding lead and an increased cancer risk. Four major human studies of workers exposed to lead have been carried out; two studies did not find an association between lead exposure and cancer, one study found

an increased incidence of respiratory tract and kidney cancers, and the fourth study found excesses for lung and stomach cancers. However, all of these studies are limited in usefulness because the route(s) of exposure and levels of lead to which the workers were exposed were not reported. In addition, exposure to other chemicals probably occurred (U.S. EPA, 1998).

Animal studies have reported kidney cancer in rats and mice exposed to lead via the oral route. No studies are available on cancer in animals exposed to lead via the inhalation or dermal routes (U.S. EPA, 1998).

EPA has classified lead in Group B2 - Probable Human Carcinogen. This classification was based on animal studies showing an increased risk of kidney tumors and inadequate human evidence (U.S. EPA, 1998).

EPA has not calculated a cancer risk estimate for lead due to the number of uncertainties that are unique to lead. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, EPA believes that "the current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk" (U.S. EPA, 1998).

7.3.11.3 <u>Noncancer Effects</u>. The primary effects in humans from chronic exposure to lead are to the nervous system. Neurological symptoms have been reported in workers with blood lead levels of 40 to 60 µg/dL, and slowed nerve conduction in peripheral nerves in adults occurs at blood lead levels of 30 to 40 µg/dL. Children are particularly sensitive to the neurotoxic effects of lead. There is evidence that blood lead levels of 10 to 30 µg/dL, or lower, may affect the hearing threshold and growth in children. Chronic exposure to lead in humans can also affect the blood. Anemia has been reported in adults at blood lead levels of 50 to 80 µg/dL and in children at blood lead levels of 40 to 70 µg/dL. Other effects from chronic lead exposure in humans include effects on blood pressure and kidney function and interference with vitamin D metabolism (ATSDR, 1997b).

Animal studies have reported effects similar to those found in humans, with effects on the blood, kidneys, and nervous, immune, and cardiovascular systems noted (ATSDR, 1997b).

EPA has not established an RfD or RfC for lead. EPA believes that it is inappropriate to develop an RfD for lead because, by comparison to most other environmental toxicants, there is a low degree of uncertainty about the health effects of lead. In addition, "it appears that some of these effects, particularly children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold" (U.S. EPA, 1998).

The Centers for Disease Control and Prevention (CDC) has set an "intervention level" for childhood lead poisoning of 10 μg/dL. This level was reduced in 1991 from the previous threshold level of 25 μg/dL and was based on scientific evidence that adverse health effects can occur at levels as low as 10 μg/dL (CDC, 1991). However, the CDC does not recommend environmental or medical intervention at 10 μg/dL. They recommend medical evaluation at or above 20 μg/dL or if blood lead levels of 15-19 μg/dL persist. Various counseling, montioring, and communitywide prevention activities were recommended at levels between 10-19 μg/dL (CDC, 1991).

7.3.12 Manganese

7.3.12.1 <u>Introduction</u>. Manganese is a naturally occurring substance found in many types of rock in combination with other chemicals such as oxygen, sulfur, and chlorine. Manganese is an essential element for humans. Manganese metal is produced from rocks containing high levels of manganese and the metal is mixed with iron to make various types of steel. Some manganese compounds are used in the production of batteries, as a component of some ceramics, pesticides, and fertilizers, and in nutritional supplements (ATSDR, 1997c).

7.3.12.2 <u>Cancer Effects</u>. No data are available on the carcinogenic effects in humans following inhalation, oral, or dermal exposure to manganese (ATSDR, 1997c).

No studies were found regarding the carcinogenic effects in animals as a result of inhalation or dermal exposure. Oral animal studies on manganese have produced mixed results, with one study reporting an increased incidence of pancreatic tumors (ATSDR, 1997c).

EPA has classified manganese as a Group D - Not Classifiable as to Carcinogenicity in Humans. EPA has not calculated a unit risk estimate for manganese (U.S. EPA, 1998).

7.3.12.3 <u>Noncancer Effects</u>. Chronic exposure to high levels of manganese by inhalation in humans results in a disease called manganism, characterized by feelings of weakness and lethargy and progressing to other symptoms such as speech disturbances, a mask-like face, tremors, and psychological disturbances. Other chronic effects from inhalation include respiratory effects such as an increased incidence of cough and bronchitis and an increased susceptibility to infectious lung disease (ATSDR, 1997c).

Neurological effects in animals have been detected following inhalation exposure to high manganese levels. No adverse effects have been reported as a result of oral or dermal exposure in animals (ATSDR, 1997c).

<u>Reference Dose</u>. EPA has established an RfD for manganese of 1.4E-01 mg/kg-d based on a NOAEL of 0.14 mg/kg-d, an uncertainty factor of 1, and a modifying factor of l. The RfD is based on data from several sources, including the National Research Council, which has determined that an “estimated safe and adequate daily dietary intake” for manganese is 2 to 5 mg/d for adults (NRC, 1989, as cited in U.S. EPA, 1998).

EPA applied an uncertainty factor of 1 because the information used to determine the RfD was taken from many large populations consuming normal diets over an extended period of time with no adverse health effects (U.S. EPA, 1998).

EPA has medium confidence in the studies on which the RfD was based, in the database, and in the RfD because many studies have reported similar findings with regard to the normal dietary intake of manganese in humans (U.S. EPA, 1998).

<u>Reference Concentration</u>. EPA has established an RfC for manganese of 5.0E-05 mg/m^3 based on a LOAEL (human equivalent concentration) of 0.05 mg/m^3, an uncertainty factor of 1,000, and a modifying factor of 1 (U.S. EPA, 1998). The RfC is based on two studies of

occupational exposure to manganese dioxide that reported increases in the impairment of neurobehavioral function (Roels et al., 1992, 1987, as cited in U.S. EPA, 1998).

EPA applied an uncertainty factor of 1,000, based on a tenfold factor to protect sensitive individuals, a tenfold factor for use of a LOAEL, and a tenfold factor for database limitations reflecting less-than-chronic periods of exposure and lack of developmental data (U.S. EPA, 1998).

EPA has medium confidence in the study on which the RfC is based, because neither of the principal studies identified a NOAEL for neurobehavioral effects, nor did either study provide information on particle size. EPA also has medium confidence in the database and RfC because the duration of exposure was limited in all the studies and insufficient information is available on the developmental and reproductive effects of manganese (U.S. EPA, 1998).

7.3.13 Elemental Mercury

7.3.13.1 Introduction. Elemental mercury is a shiny, silver-white, odorless liquid. Elemental mercury is released to the air by natural and industrial processes. A major route of exposure to elemental mercury is inhalation in occupational settings, such as chlorine-alkaline manufacturing facilities. Exposure may also occur from dental and medical treatments; dental amalgams may contain between 43 and 54 percent elemental liquid mercury (ATSDR, 1997d).

7.3.13.2 Cancer Effects. There are a number of epidemiological studies that have examined cancer mortality and morbidity among workers occupationally exposed to elemental mercury. All of these studies have limitations, including small sample sizes, probable exposure to other lung carcinogens, failure to consider confounding factors such as smoking, and failure to observe correlations between estimated exposure and cancer incidence (U.S. EPA, 1997c).

One available animal study identified cancer incidence in animals exposed to elemental mercury by injection. Tumors were found at the contact sites; however, the study was incompletely reported as to controls and statistics (U.S. EPA, 1997c).

EPA has classified elemental mercury in Group D - Not Classifiable as to Human Carcinogenicity, based on inadequate human and animal data. EPA has not calculated a unit risk estimate for elemental mercury (U.S. EPA, 1998).

7.3.13.3 Noncancer Effects. Nervous system effects are the most sensitive toxicologic endpoint observed following exposure to elemental mercury. Symptoms associated with elemental mercury neurological toxicity include tremors, irritability, excessive shyness, nervousness, insomnia, headaches, polyneuropathy, and memory loss. At higher concentrations, kidney and respiratory effects have been observed (U.S. EPA, 1997c).

Reference Dose. EPA has not calculated an RfD for elemental mercury.

Reference Concentration. EPA has calculated an RfC for elemental mercury of 3.0E-04 mg/m^3, based on a LOAEL (adjusted) of 0.09 mg/m^3, an uncertainty factor of 30, and a modifying factor of 1. A human occupational study was used as the basis for the RfC and the

LOAEL (Fawer et al., 1983, as cited in U.S. EPA, 1998) and several other human occupational studies were used to corroborate this LOAEL. These studies investigated neurological effects in humans exposed to elemental mercury in the workplace; hand tremors, increases in memory disturbances, and evidence of autonomic dysfunction were observed and were the basis for the LOAEL (U.S. EPA, 1998).

An uncertainty factor of 30 was applied based on a tenfold factor for the protection of sensitive human subpopulations and an additional threefold factor for database deficiencies, particularly developmental and reproductive studies (U.S. EPA, 1998).

EPA has medium confidence in the studies on which the RfC was based because there were a sufficient number of human subjects, an appropriate control group, and the exposure levels in a number of studies had to be extrapolated from blood mercury levels. EPA also has medium confidence in the database due to lack of human or multispecies reproductive/ developmental studies and medium confidence in the RfC (U.S. EPA, 1998).

7.3.14 Inorganic Mercury (Mercuric Chloride; Divalent Mercury)

7.3.14.1 Introduction. Inorganic mercury compounds are usually white powders of crystals. Until 30 years ago, inorganic mercury compounds were used extensively as pharmaceuticals, such as components of antiseptics, diuretics, skin lightening creams, and laxatives. Since then, more effective and less harmful alternatives have replaced most pharmaceutical uses of mercury. Today, most exposure to inorganic mercury compounds occurs through dental treatments (ATSDR, 1997d).

7.3.14.2 Cancer Effects. There are no data concerning the carcinogenic effects of mercuric chloride in humans (U.S. EPA, 1997c).

Limited animal data are available on the carcinogenic effects of inorganic mercury. Cancer of the forestomach and thyroid were seen in rats exposed to mercuric chloride by gavage, and evidence of cancer of the forestomach and kidneys was considered equivocal in mice (U.S. EPA, 1997c).

EPA has classified mercuric chloride in Group C - Possible Human Carcinogen, based on the absence of data in humans and limited evidence in rats and mice. EPA has not calculated a unit risk estimate for mercuric chloride (U.S. EPA, 1998).

7.3.14.3 Noncancer Effects. The primary effect from chronic exposure to inorganic mercury is kidney damage, primarily due to mercury-induced autoimmune glomerulonephritis (induction of an immune response to the body's kidney tissue). In addition, several animal studies have reported developmental effects from exposure to inorganic mercury (U.S. EPA, 1997c).

Reference Dose. EPA has established an RfD of 3.0E-04 mg/kg-d for inorganic mercury. This was based on a consensus decision of a panel of mercury experts who used several LOAELs ranging from 0.23 to 0.63 mg/kg-d (Shultz, 1988, as cited in U.S. EPA, 1998), an uncertainty factor of 1,000, and a modifying factor of 1. The LOAELs were derived from several rat feeding

and subcutaneous studies in which autoimmune glomerulonephritis was observed (U.S. EPA, 1998).

An uncertainty factor of 1,000 was applied based a tenfold factor for an animal study with a LOAEL, a tenfold factor for use of a subchronic study, and an additional tenfold factor for sensitive human subpopulations (U.S. EPA, 1998).

The studies on which the RfD was based were not given a confidence ranking; the RfD and database were given a high confidence ranking based on the weight of evidence from several studies using Brown Norway rats (U.S. EPA, 1998).

Reference Concentration. EPA has not established an RfC for inorganic mercury.

7.3.15 Organic Mercury (Methylmercury)

7.3.15.1 Introduction. Organic mercury compounds are white crystalline solids. Most exposure to organic mercury occurs through the diet, with fish and fish products as the dominant source. Sources of past exposure to organic mercury include fungicide-treated grains and meat from animals fed such grain. However, fungicides containing mercury are banned in the United States today and this source of exposure is now negligible (ATSDR, 1997d).

7.3.15.2 Cancer Effects. Three human studies have examined the relationship between methylmercury and cancer incidence. However, these studies were considered extremely limited because of study design or incomplete data reporting (U.S. EPA, 1997c).

Several animal studies have shown an increased incidence of kidney tumors in mice exposed orally to methylmercury. However, these tumors were observed only at a single site (kidney), in a single species (mice), and a single sex (males) (U.S. EPA, 1997c).

EPA has classified methylmercury in Group C - Possible Human Carcinogen, based on the absence of data in humans and limited evidence in animals. EPA has not calculated a unit risk estimate for methylmercury (U.S. EPA, 1998).

7.3.15.3 Noncancer Effects. A large number of human studies are available on the systemic effects of methylmercury. This database is the result of two large-scale poisoning episodes in Japan and Iraq, as well as several epidemiologic studies investigating populations that consume large quantities of fish. Methylmercury mainly affects the central nervous system. Early symptoms from chronic exposure to low levels of methylmercury are prickling on the skin, blurred vision, and malaise. At higher doses, deafness, speech difficulties, and constriction of the visual field are seen. The fetus is at particular risk from methylmercury exposure. Offspring born to women exposed to methylmercury during pregnancy have exhibited a number of developmental abnormalities including delayed onset of walking and talking, cerebral palsy, altered muscle tone, and reduced neurological test scores (U.S. EPA, 1997c).

Reference Dose. EPA has established an RfD of 1.0E-04 mg/kg-d for methylmercury, based on a benchmark dose of 0.0011 mg/kg-d, an uncertainty factor of 10, and a modifying factor of 1 (U.S. EPA, 1998). This was based on developmental abnormalities in infants born to

mothers exposed to methylmercury in contaminated grain in Iraq (Marsh et al., 1987, and Ahmed, 1991, as cited in U.S. EPA, 1998). EPA used a benchmark dose, the lower 95 percent confidence level for a 10 percent incidence rate of neurologic changes, based on modeling of all effects in children. This lower bound was 11 ppm methylmercury in maternal hair. A dose conversion was used to estimate a daily intake of 1.1 µg methylmercury/kg body weight/d that, when ingested by a 60-kg individual, will maintain a concentration of approximately 44 µg/L of blood or a hair concentration of 11 µg mercury/g hair (11 ppm) (U.S. EPA, 1997c, 1998).

EPA applied an uncertainty factor of 10, based on a threefold factor for variability in the human population and an additional threefold factor for the lack of a two-generation reproductive study and lack of data for the effect of exposure duration on developmental neurotoxicity effects and on adult paresthesia (U.S. EPA, 1998).

EPA has medium confidence in the studies on which the RfD was based, in the database, and in the RfD. These rankings are based on the fact that the benchmark dose approach allowed use of the entire dose-response assessment with a resulting value that is consistent with the traditional NOAEL/LOAEL approach. However, EPA has some concerns related to the applicability of a dose-response estimate based on a grain-consuming population when the actual application is likely to help characterize risk for fish-consuming segments of the population (U.S. EPA, 1998).

It is also important to consider the fact that the RfD represents a “no-effect” level that is presumed to be without appreciable risk. As discussed above, EPA used an uncertainty factor of 10 to derive the RfD for methylmercury. An uncertainty factor of 100 to 1,000 is usually applied when the RfD is based on animal data; however, since this RfD was based on human data, an uncertainty factor of 10 was deemed appropriate. In addition, the RfD was based on a benchmark dose that itself was derived as the lower 95 percent confidence level for the 10 percent incidence rate of neurologic abnormalities in children. Therefore, there is a margin of safety between the RfD and the level corresponding to the threshold for adverse effects, as indicated by the human data.

Considerable new data on the health effects of methylmercury are becoming available. Large studies of fish- and marine-mammal-consuming populations in the Seychelles and Faroe Islands have been carried out. Smaller-scale studies also describe effects in populations around the U.S. Great Lakes. However, EPA has decided “that it is premature to make a change in the methylmercury RfD at this time (U.S. EPA, 1997c). In November 1998, EPA and other federal Agencies participated in an interagency review of available human neurodevelopmental data on methylmercury, including the most recent studies from the Seychelles and Faroe Islands. Preliminary review of the Seychellois and Faroese data supports the current RfD as scientifically valid and protective of human health. The National Academy of Sciences (NAS) is currently independently assessing the EPA’s RfD for methylmercury. Pending the completion of the NAS study, EPA will reevaluate the RfD for methylmercury following careful review of the results of the NAS study.

Reference Concentration. EPA has not established an RfC for methylmercury.

7.3.16 Nickel

7.3.16.1 <u>Introduction</u>. Nickel is a silvery-white metal that is usually found in nature as a component of silicate, sulfide, or arsenide ores. The predominant forms of nickel in the atmosphere are nickel sulfate, nickel oxides, and the complex oxides of nickel. Each form of nickel exhibits different physical properties. Most nickel is used to make stainless steel; other uses include the manufacture of batteries, electroplating baths, textile dyes, coins, sparkplugs, and machinery parts (ATSDR, 1997e).

7.3.16.2 <u>Cancer Effects</u>. Human studies have reported an increased risk of lung and nasal cancers among nickel refinery workers exposed to nickel refinery dust. Nickel refinery dust is defined as the "dust from pyro-metallurgical sulfide nickel matte" refineries and is a mixture of many nickel compounds, including nickel subsulfide. It is not certain which compound is carcinogenic in the nickel refinery dust (U.S. EPA, 1998). No information is available on the carcinogenic effects of nickel in humans from oral or dermal exposure (ATSDR, 1997e; U.S. EPA, 1998).

Animal studies have reported lung tumors from inhalation exposure to the following nickel compounds and mixtures: nickel refinery dusts, nickel subsulfide, and nickel carbonyl. Oral animal studies have not reported tumors from exposure to nickel acetate in the drinking water. No information is available on the carcinogenic effects of nickel in animals from dermal exposure (ATSDR, 1997e; U.S. EPA, 1998).

EPA has classified nickel refinery dust in Group A - Known Human Carcinogen. The Group A classification was based on an increased risk of lung and nasal cancer in humans through inhalation exposure and increased lung tumor incidences in animals by inhalation and injection (U.S. EPA, 1998).

<u>Inhalation Cancer Risk</u>. EPA used the additive and multiplicative extrapolation method, based on human data, to estimate the unit cancer risk for nickel refinery dust. EPA calculated an inhalation unit risk estimate of 2.4E-04 $(\mu g/m^3)^{-1}$. EPA used four data sets, all from human exposure, to calculate the unit risk estimates for nickel refinery dusts. A range of incremental unit risk estimates were calculated from these data sets that were consistent with each other (U.S. EPA, 1998).

<u>Oral Cancer Risk</u>. EPA has not calculated an oral cancer risk estimate for any nickel compound.

7.3.16.3 <u>Noncancer Effects</u>. Contact dermatitis is the most common effect in humans from exposure to nickel via inhalation, oral, or dermal exposure. Cases of nickel-contact dermatitis have been reported following occupational and nonoccupational exposure, with symptoms of itching of the fingers, wrists, and forearms. Chronic inhalation exposure to nickel in humans also results in respiratory effects. These effects include direct respiratory effects such as asthma due to primary irritation or an allergic response and an increased risk of chronic respiratory tract infections (ATSDR, 1997e).

Animal studies have reported effect on the lungs, kidneys, and immune system from inhalation exposure to nickel, and effects on the respiratory and gastrointestinal systems, heart, blood, liver, kidney, and decreased body weight from oral exposure to nickel. Dermal animal studies have reported effects on the skin (ATSDR, 1997e).

Reference Dose. EPA has established an RfD for nickel (soluble salts) of 2.0E-02 mg/kg-d, based upon a NOAEL (adjusted) of 5 mg/kg-d, an uncertainty factor of 300, and a modifying factor of 1. This was based on a study in rats (Ambrose et al., 1976, as cited in U.S. EPA, 1998) that showed decreased body and organ weights from chronic (2-year) exposure to nickel in the diet. Several other studies showed similar results, with decreased body and organ weights after exposure to nickel chloride via gavage and through the drinking water (U.S. EPA, 1998).

An uncertainty factor of 300 was applied, based on a tenfold factor of interspecies extrapolation, a tenfold factor to protect sensitive subpopulations, and a threefold factor for inadequacies in the reproductive studies (U.S. EPA, 1998).

EPA has low confidence in the study on which the RfD was based because, although it was properly designed and provided adequate toxicological endpoints, high mortality occurred in the controls. EPA has medium confidence in the database because it provided adequate supporting subchronic studies and consequently medium confidence level in the RfD (U.S. EPA, 1998).

Reference Concentration. EPA has not established an RfC for any nickel compound.

7.3.17 Selenium

7.3.17.1 Introduction. Selenium is a naturally occurring substance in the earth's crust and is commonly found in sedimentary rock combined with other substances, such as sulfide minerals, or with silver, copper, lead, and nickel minerals. Selenium is an essential element for humans and animals and exposure occurs daily through food intake. lt is used in the electronics industry; the glass industry; in pigments used in plastics, paints, enamels, inks, and rubber; in pharmaceuticals manufacturing; and as a constituent of fungicides (ATSDR, 1996).

7.3.17.2 Cancer Effects. Several epidemiological studies have examined the relationship between cancer death rates in humans and selenium levels in forage crops. These studies have reported an increased incidence of colon, breast, and other forms of cancer in areas where selenium is deficient and a lowered cancer incidence with higher selenium concentrations. Other studies have reported that blood serum levels in patients with cancer had significantly lower selenium levels than healthy patients (U.S. EPA, 1998).

Several animal studies have investigated the carcinogenicity of selenium. However, the data are conflicting and difficult to interpret because of apparent anticarcinogenicity and high toxicity of some selenium compounds (U.S. EPA, 1998).

EPA has classified selenium in Group D - Not Classifiable as to Carcinogenicity in Humans because of inadequate human data and inadequate evidence of carcinogenicity in animals (U.S. EPA, 1998).

7.3.17.3 Noncancer Effects. No information is available on the chronic effects of selenium in humans from inhalation exposure. Ingestion of high levels of selenium in food and water has led to “selenosis,” characterized by discoloration of the skin, deformation and loss of nails, hair loss, excessive tooth decay and discoloration, lack of mental alertness, and listlessness. Dermal exposure has resulted in skin rashes and contact dermatitis (ATSDR, 1996).

No data are available on the chronic effects in animals from inhalation exposure. Livestock exposed through consumption of high levels of selenium develop "alkali disease." (ATSDR, 1996).

Reference Dose. EPA has established an RfD for selenium of 5.0E-03 mg/kg-d based on an adjusted NOAEL of 0.015 mg/kg-d, an uncertainty factor of 3, and a modifying factor of 1. The RfD is based on an epidemiological study (Yang et al., 1989, as cited in U.S. EPA, 1998), which reported selenosis in a population in China. Clinical signs observed included “garlic odor” of the breath and urine, thickened and brittle nails, hair and nail loss, lowered hemoglobin levels, mottled teeth, skin lesions, and central nervous system abnormalities (U.S. EPA, 1998).

EPA applied an uncertainty factor of 3 to account for sensitive individuals. A full factor of 10 was not deemed necessary since similar NOAELs were identified in two moderate-sized populations exposed to selenium in excess of the recommended daily allowance without apparent signs of selenosis (U.S. EPA, 1998).

EPA has medium confidence in the study on which the RfD was based, because even though this was a study in which a sizable population with sensitive subpopulations was studied, there were still several possible interactions that were not fully accounted for. EPA has high confidence in the database because many animal studies and epidemiologic studies support the principal study and consequently high confidence in the RfD (U.S. EPA, 1998).

Reference Concentration. EPA has not established an RfC for selenium (U.S. EPA, 1998).

7.3.18 Silver

7.3.18.1 Introduction. Silver is a naturally occurring element that is often found deposited as a mineral ore in association with other elements. It is acquired as a by-product during the retrieval of copper, lead, zinc, and gold ores. It is used in photographic materials, electrical products, silver paints, batteries, sterling ware, and jewelry (ATSDR, 1990b).

7.3.18.2 Cancer Effects. No evidence of cancer in humans has been reported despite frequent therapeutic use of silver compounds over the years. Animal studies have shown local sarcomas after the implantation of foils and discs of silver (U.S. EPA, 1998).

EPA has classified silver in Group D - Not Classifiable as to Human Carcinogenicity, based on questionable interpretation of the local sarcomas seen in animal studies. Even insoluble solids such as plastics have been shown to result in local sarcomas (U.S. EPA, 1998).

7.3.18.3 Noncancer Effects. The only clinical condition that is known in humans to be associated with long-term exposure to silver is argyria, a gray or blue-gray discoloring of the skin. Argyria was common around the turn of the century when many pharmacological preparations contained silver. It is much less common now. Today, case reports in humans have reported that repeated dermal contact with silver may in some cases lead to contact dermatitis and a generalized allergic reaction to silver (ATSDR, 1990b).

EPA has established an RfD for silver of 5.0E-03 mg/kg-d based on a LOAEL (adjusted) of 0.014 mg/kg-d, an uncertainty factor of 3, and a modifying factor of 1 (U.S. EPA, 1998). The RfD is based on a report summarizing 70 cases of argyria following use of silver medication in humans (Gaul and Staud, 1935, as cited in U.S. EPA, 1998).

An uncertainty factor of 3 was applied to account for minimal effects in a subpopulation that has exhibited an increased propensity for the development of argyria. The critical effect is cosmetic, with no associated adverse health effects (U.S. EPA, 1998).

EPA has medium confidence in the critical study used as the basis for the RfD because it is an old study and only describes patients who developed argyria; no information is presented on patients who received injections of silver and did not develop argyria. EPA has low confidence in the database because the studies used to support the RfD were not controlled studies, and low-to-medium confidence in the RfD because the RfD is based on a study using intravenous administration, which necessitated a dose conversion with inherent uncertainties (U.S. EPA, 1998).

Reference Concentration. EPA has not established an RfC for silver (U.S. EPA 1998).

7.3.19 2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin

7.3.19.1 Introduction. 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) belongs to the class of compounds, chlorinated dibenzo-*p*-dioxins, that are referred to as dioxins. 2,3,7,8-TCDD is a colorless solid with no known odor. It does not occur naturally nor is it intentionally manufactured by any industry, although it can be produced inadvertently in small amounts as an impurity during the manufacture of certain herbicides and germicides and has been detected in products of incineration of municipal and industrial wastes. The only current use for 2,3,7,8-TCDD is in chemical research (ATSDR, 1998).

EPA issued a draft *Health Assessment Document for 2,3,7,8-TCDD and Related Compounds* in 1994. This document is a three-volume series consisting of a complete reassessment of the toxic effects of 2,3,7,8-TCDD (U.S. EPA, 1994b, c). The document was reviewed by EPA's Science Advisory Board (SAB) but has not yet been issued in final form. Most of the information in this summary is from this draft document and is subject to change, pending the release of the final document.

7.3.19.2 Cancer Effects. An increase in lung cancer risks was observed among Japanese males exposed to 2,3,7,8-TCDD as a result of an oil poisoning accident. Human studies have also found an association between 2,3,7,8-TCDD and soft-tissue sarcomas, lymphomas, and stomach carcinomas, although for malignant lymphomas, the increase in risk is not consistent.

The increase in risk is of borderline significance for highly exposed groups and is less among groups exposed to lower levels of 2,3,7,8-TCDD (U.S. EPA, 1994c).

An increased incidence of soft tissue sarcoma was found to be elevated in several recent studies. EPA stated that (U.S. EPA, 1994c)

> . . . the fact that similar results were obtained in independent studies of differing design and evaluating populations exposed to dioxin-like compounds under varying conditions, along with the rarity of this tumor type, weighs in favor of a consistent and real association. On the other hand, arguments regarding selection bias, differential exposure misclassification, confounding, and chance in each individual study have been presented in the scientific literature which increase uncertainty around this association. In addition excess respiratory cancer was noted in other studies. These results are also supported by significantly increased mortality from lung and liver cancers subsequent to the Japanese rice oil poisoning accident where exposure to PCDFs and PCBs occurred. Again, while smoking as a confounder cannot be totally eliminated as a potential explanation of these results, analyses conducted to date suggest that smoking is not likely to explain the entire increase in lung cancer. The question of confounding exposures, such as asbestos and other chemicals, in addition to smoking, has not been entirely ruled out and must be considered as potentially adding to the observed increases. Although increases of cancer at other sites (e.g., non-Hodgkin's lymphoma, stomach cancer) have been reported, the data for an association with exposure to dioxin-like compounds are less compelling.

Information on the carcinogenicity of 2,3,7,8-TCDD following inhalation exposure of animals is not available. In animal studies of oral exposure to 2,3,7,8-TCDD, multisite tumors in rats and mice, including the tongue, lung, nasal turbinates, liver, and thyroid, have been reported from long-term bioassays. It has also been shown to be carcinogenic in hamsters (U.S. EPA, 1994c).

EPA has classified 2,3,7,8-TCDD as a Group B2 - Probable Human Carcinogen (U.S. EPA, 1984, 1997b).

Toxicity Equivalency Factors. EPA has assigned the dioxin compounds individual toxicity equivalency factors (TEFs). TEFs are estimates of the toxicity of dioxin-like compounds relative to the toxicity of TCDD, which is assigned a TEF of 1.0. Table 7-2 lists the TEFs for dioxin compounds (Van den Berg et al., 1998).

Cancer Risk. EPA examined the available carcinogenicity data for 2,3,7,8-TCDD and stated (U.S. EPA, 1994c):

> Epidemiology studies suggest that the lung in the human male is a much more sensitive target organ for TCDD than is the liver and that the human is a sensitive species for cancer response, probably more sensitive than the rat. Although smoking may be a modifier for the lung cancer response, the studies also show increases for all cancers combined. Estimates derived from the human data

Table 7-2. Toxicity Equivalency Factors (TEFs) for Dioxin Compounds

Compound	TEF
2,3,7,8-TCDD	1
1,2,3,4,5,7,8,9-OCDD	0.0001
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
1,2,3,4,6,7,8,9-OCDF	0.0001
1,2,3,4,7,8-HxCDD,	0.1
1,2,3,7,8-PeCDD,	1
2,3,7,8-TCDF	0.1
1,2,3,4,7,8,9-HpCDF	0.01
2,3,4,7,8-PeCDF	0.5
1,2,3,7,8-PeCDF	0.05
1,2,3,6,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDD	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1

suggest a unit risk for lung cancer of 3 to $5x10^{-4}$ $(pg/kg\text{-}day)^{-1}$, for all cancers combined the unit risk estimate is 2 to $3x10^{-3}$ $(pg/kg\text{-}day)^{-1}$. While unit risk estimates based on rat tumors are somewhat less, they are within the range of uncertainty of those based on human data. Both animal and human responses are consistent with low-dose linearity.

EPA then concluded:

With regard to carcinogenicity, a weight of evidence evaluation suggests that dioxin and related compounds (CDDs, CDFs, and dioxin-like PCBs) are likely to present a cancer hazard to humans. While major uncertainties remain, efforts of this reassessment to bring more data into the evaluation of cancer potency have resulted in a risk specific dose estimate (1×10^{-6} or one additional cancer in one million exposed) of approximately 0.01 pg TEQ/kg body weight/day. This risk

> specific dose estimate represents a plausible upper bound on risk based on the evaluation of animal and human data. "True" risks are not likely to exceed this value, may be less, and may even be zero for some members of the population.

Dose-Response Modeling. EPA recently completed a draft assessment of the scientific foundation for dose-response modeling for 2,3,7,8-TCDD. Different models were reviewed for use in risk assessment. An empirical analysis was done for a broad range of experimental data on 2,3,7,8-TCDD. For each data set with enough data for a dose-response analysis, benchmark doses were calculated at levels of 1, 5, and 10 percent (animal data) and 0.1, 0.5, and 1 percent (epidemiological data). In addition, for the experimental data, the shape of the overall dose-response curve was examined (U.S. EPA, 1997a).

EPA stated that it was not possible to make any firm conclusions about the shape of the dose-response curve for 2,3,7,8-TCDD beyond the experimental range. In addition, EPA felt that there were a sufficient number of dose-response curves consistent with linearity to warrant concern about nonlinear extrapolations, but there is no way to disprove scientifically the existence of nonlinearity in the area below the experimental region (U.S. EPA, 1997a).

In summary, EPA (U.S. EPA, 1997a) stated,

> It is clear from this analysis that dioxin causes a variety of toxicities in test animals following chronic and bolus exposures. The human data is less clear, but qualitatively and quantitatively consistent with the animal findings when expressed on the basis of steady-state body burden rather than a daily dose or area-under-the-curve basis. There are sufficient data suggesting response proportionate to dose to warrant concern that this compound will induce toxic effects in humans in the range of the experimental animal data. Also, based on a lack of data to argue for an immediate and steep change in slope for many of the responses analyzed there is the possibility of response 1 to 2 orders of magnitude below this range.

Inhalation Cancer Risk. EPA has calculated an inhalation cancer slope factor for 2,3,7,8-TCDD of 1.56E+05 $(mg/kg\text{-}d)^{-1}$ and an inhalation unit risk estimate of 3.3E-05 $(pg/m^3)^{-1}$. These values are under review and are subject to change; they are based on an oral study in which rats were exposed to 2,3,7,8-TCDD in the diet for 720 days with resulting tumors of the respiratory system and liver (Kociba et al., 1978, as cited in U.S. EPA, 1984). This cancer slope factor is identical with the oral cancer slope factor; the inhalation unit risk estimate was based on route-to-route extrapolation from the oral cancer slope factor, assuming 75 percent absorption (U.S. EPA, 1984, 1997b).

Oral Cancer Risk. EPA has derived an oral cancer slope factor of 1.56E+05 $(mg/kg\text{-}d)^{-1}$ for 2,3,7,8,-TCDD, based on the Kociba et al. (1978) study as discussed above (U.S. EPA, 1984).

7.3.19.3 Noncancer Effects. The major noncarcinogenic effect from exposure to 2,3,7,8-TCDD is chloracne, a severe acne-like condition that develops within months of first exposure to high levels of 2,3,7,8-TCDD. For many individuals, the condition disappears after

discontinuation of exposure, for others it may remain for years. There are limited human data to suggest the doses at which chloracne is likely to occur. Occupational studies suggest that persistent chloracne is more often associated with high-intensity exposures, for long periods of time, and starting at an early age (U.S. EPA, 1994b, c). Acute exposures or chronic exposures to 2,3,7,8-TCDD at low levels have usually resulted in chloracne lasting for no longer than a few months to a few years (U.S. EPA 1994b, c).

Epidemiological studies have reported conflicting evidence on the immunotoxicity of 2,3,7,8-TCDD in humans. Some studies have suggested evidence of immunotoxicity, such as alterations in lymphocyte populations, cell surface markers, or lymphocyte proliferative response (ATSDR, 1998). However, studies have not reported changes in the immune system directly related to 2,3,7,8-TCDD exposure (U.S. EPA, 1994b, c).

An association has been reported between levels of male reproductive hormones and 2,3,7,8-TCDD exposure. Decreased testosterone levels were detected in several human studies, and animal data are available to support these findings. Other effects noted in human studies include an association between 2,3,7,8-TCDD exposure and

- An increased risk of diabetes and an elevated prevalence of abnormal fasting serum glucose levels
- The induction of cytochrome P-450 1A1, an enzyme involved in biotransformation reactions
- Elevation of gamma glutamyl transferase, a liver enzyme
- A possible increased risk of endometriosis, a disease of the female reproductive system (U.S. EPA, 1994b, c).

Animal studies have reported reproductive and developmental effects from exposure to 2,3,7,8-TCDD. These studies have suggested that altered development may be among the most sensitive endpoints of 2,3,7,8-TCDD exposure. Several animal species have reported developmental toxicity occurring at lower levels than male and female reproductive toxicity effects. 2,3,7,8-TCDD appears to affect a large number of critical developmental effects at specific developmental stages. These changes can lead to increases in fetal mortality, disruption of organ system structure, and irreversible impairment of organ function. Developmental toxicity from 2,3,7,8-TCDD has been seen in fish, birds, and mammals. Thus, it is likely to occur at some level in humans. However, it is not possible to state what sort of effects will occur or at what levels (U.S. EPA, 1994b, c).

Animal studies have reported changes in the skin resembling chloracne from 2,3,7,8-TCDD exposure. Distinctive changes in animals include swelling and inflamed eyelids, nail loss, and facial hair loss (ATSDR, 1998).

The immune system also appears to be a target from 2,3,7,8-TCDD exposure in animal studies. Alterations in specific immune effector functions and increased susceptibility to infectious diseases have been observed in animals exposed to 2,3,7,8-TCDD. Both cell-mediated

and humoral immune responses were suppressed following 2,3,7,8-TCDD exposure (U.S. EPA, 1994b, c).

EPA has not calculated an RfD or an RfC for 2,3,7,8-TCDD.

7.3.20 Thallium

7.3.20.1 Introduction. Thallium is a metallic element that exists in the environment combined with other elements, such as oxygen, sulfur, and the halogens. Thallium is quite stable in the environment, since it is neither transformed nor biodegraded. It is released to the environment from coal burning and smelting, and its major use is in the semiconductor industry where it is used in the production of switches and closures (ATSDR, 1990c).

7.3.20.2 Cancer Effects. Limited human studies are available on the carcinogenic effects of thallium. One epidemiologic study did not report an increase in tumors in workers exposed to thallium. No animal studies are available (U.S. EPA, 1998).

EPA has classified thallium in Group D - Not Classifiable as to Human Carcinogenicity, based on the lack of carcinogenicity data in animals and humans (U.S. EPA, 1998).

7.3.20.3 Noncancer Effects. Thallium compounds can affect the respiratory, cardiovascular, and gastrointestinal systems, liver, kidneys, and the male reproductive systems in humans. Temporary hair loss has also been associated with ingestion of thallium in humans. Developmental effects were not noted in children born to mothers who had been exposed to thallium during pregancy (ATSDR, 1990c).

Reference Dose. EPA has established an RfD for thallium (thallium sulfate, thallium chloride, and thallium carbonate) of 8.0E-05 mg/kg-d based on an adjusted NOAEL of 0.25 mg/kg-d, an uncertainty factor of 3,000, and a modifying factor of 1. The RfD is based on a subchronic toxicity study of thallium sulfate in rats in which no adverse effects were reported (U.S. EPA, 1986, as reported in U.S. EPA, 1998).

An uncertainty factor of 3,000 was applied, based on a tenfold factor to extrapolate from subchronic to chronic data, a tenfold factor for intraspecies extrapolation, a tenfold factor for interspecies variability, and a threefold factor to account for lack of reproductive and chronic toxicity data (U.S. EPA, 1998).

EPA has low confidence in the critical study used as the basis for the RfD due to uncertainties in the results and because supporting studies show adverse health effects at doses slightly higher than the NOAEL; low confidence in the database because there is only one subchronic study and some anecdotal human data, and consequently low confidence in the RfD (U.S. EPA, 1998).

Reference Concentration. EPA has not established an RfC for thallium (U.S. EPA, 1998).

7.4 Particulate Matter (PM_{10} and $PM_{2.5}$)

Epidemiological studies that have estimated relationships between ambient PM concentrations and health effects are available for several health effects and several different population groups. The broad categories of health endpoints for which concentration-response functions have been estimated based on measures of PM are

- Mortality
- Hospital admissions
- Respiratory symptoms and restricted activity days (not requiring hospitalization).

The health endpoints included in each of these categories and the possible overlap among health effects and populations studied are described in Table 7-3. Descriptions of the populations investigated in the relevant studies are important because, in most cases, the concentration-response functions from these studies are applied only to the subpopulation (e.g., asthmatic children) investigated in the epidemiologic study. A detailed discussion of modeling analysis conducted to evaluate PM health effects for this risk assessment, including uncertainties in the data and modeling methods, is provided in Appendix E.

7.4.1 Mortality Studies

The studies that associate PM exposures with premature mortality presented in this analysis differ primarily in the type of PM exposure used as input to the concentration response functions (i.e., whether $PM_{2.5}$ or PM_{10} is used and whether short-term or long-term exposure is used). The mortality studies also differ slightly in the populations studied. Brief descriptions of the mortality studies used in this analysis and the issues related to the overlap in the incidence predicted from these studies are discussed here.

One long-term exposure study is presented here. Pope et al. (1995) is a prospective cohort study that investigated the association between long-term exposure to ambient $PM_{2.5}$ concentrations (measured in the study as the median of all daily concentrations measured over a 4-year period) and mortality in a cohort of adults age 30 and older.[1]

Two estimates of the relationship between mortality and short-term exposure to PM are presented. One estimate is from a pooled analysis of 10 individual studies in which PM_{10} concentrations are averaged over a period of 1 to 5 days. The second estimate is taken from Schwartz et al. (1996) and uses a 2-day average $PM_{2.5}$ measure. In both cases, short-term exposure is related to daily mortality for the full population.

Long-term studies may be preferable to “short-term” (daily average) studies for estimating health effects for a couple of reasons. First, by their basic design, daily studies detect acute effects but cannot detect the effects of long-term exposures. A chronic exposure study

[1]Dockery et al., 1993, is another study relating long-term exposures to PM to premature mortality. However, the study by Pope et al. considered a much larger population and included many more locations (52 cities versus 6 in the Dockery study). The Pope study is therefore considered to be preferable.

Table 7-3. Concentration-Response Functions Used To Estimate Health Effects Associated with Exposure to Particulate Matter

Endpoint	Concentration-Response Function		PM Averaging Time		Population[a]	Annual Baseline Incidence (per 100,000 population)[b]	Pollutant Coefficient[c]
	Source	Functional Form	Studied	Applied			
Mortality							
Mortality (long-term exposure), using $PM_{2.5}$ indicator	Pope et al., 1995	Loglinear	Median of 4 years of data	Annual median[d]	Ages 30+	759 (number of nonaccidental deaths in the population ages 30 + divided by 100,000 individuals of all ages)	0.006408
Mortality (short-term exposure), using $PM_{2.5}$ indicator	Schwartz et al., 1996a (Boston, Knoxville, St. Louis, Steubenville, Portage & Topeka)	Loglinear	2-day average	1-day average[e]	All	803 (nonaccidental deaths in general population)	0.001433
Mortality (short-term exposure), using PM_{10} indicator[e]	Ito & Thurston, 1996 (Chicago)	Loglinear	2-day average	1-day average[f]	All	803 (nonaccidental deaths in general population)	0.000782
	Kinney et al., 1995 (Los Angeles)	Loglinear	1-day average		All		
	Pope et al., 1992 (Utah)	Loglinear	5-day average		All		

(continued)

Table 7-3. (continued)

Endpoint	Concentration-Response Function		PM Averaging Time		Population[a]	Annual Baseline Incidence (per 100,000 population)[b]	Pollutant Coefficient[c]
	Source	Functional Form	Studied	Applied			
	Schwartz, 1993a (Birmingham)	Loglinear	3-day average		All		
	Schwartz et al., 1996 (Boston)	Loglinear	2-day average		All		
	Schwartz et al., 1996 (Knoxville)	Loglinear	2-day average		All		
	Schwartz et al., 1996 (St. Louis)	Loglinear	2-day average		All		
	Schwartz et al., 1996 (Steubenville)	Loglinear	2-day average		All		
	Schwartz et al., 1996 (Portage)	Loglinear	2-day average		All		
	Schwartz et al., 1996 (Topeka)	Loglinear	2-day average		All		
Hospital Admissions							
All respiratory illnesses, using $PM_{2.5}$ indicator	Thurston et al., 1994 (Toronto)	Linear	1-day average	1-day average	All	n/a	3.45×10^{-8} [f]
All respiratory illnesses, using PM_{10} indicator	Schwartz, 1995 (Tacoma)	Loglinear	1-day average	1-day average	Age 65+	504 (general population)	0.00170
	Schwartz, 1995 (New Haven)	Loglinear	1-day average		Age 65+		
	Schwartz, 1996 (Spokane)	Loglinear	1-day average		Age 65+		
COPD, using PM_{10} indicator	Schwartz, 1994a (Birmingham)	Loglinear	1-day average	1-day average	Age 65+	103	0.002533

(continued)

Table 7-3. (continued)

Endpoint	Concentration-Response Function		PM Averaging Time		Population[a]	Annual Baseline Incidence (per 100,000 population)[b]	Pollutant Coefficient[c]
	Source	Functional Form	Studied	Applied			
	Schwartz, 1994b (Detroit)	Loglinear	1-day average		Age 65+	(general population)	
	Schwartz, 1996 (Spokane)	Loglinear	1-day average		Age 65+		
Pneumonia, using PM_{10} indicator	Schwartz, 1994a (Birmingham)	Loglinear	1-day average	1-day average	Age 65+	229 (general population)	0.0013345
	Schwartz, 1994b (Detroit)	Loglinear	1-day average		Age 65+		
	Schwartz, 1994c (Minneapolis)	Loglinear	1-day average		Age 65+		
	Schwartz, 1996 (Spokane)	Loglinear	1-day average		Age 65+		
Congestive heart failure, using PM_{10} indicator	Schwartz & Morris, 1995 (Detroit)	Loglinear	2-day average	1-day average	Age 65+	231 (general population)	0.00098
Ischemic heart disease, using PM_{10} indicator	Schwartz & Morris, 1995 (Detroit)	Loglinear	1-day average	1-day average	Age 65+	450 (general population)	0.00056
Respiratory Symptoms/Illnesses Not Requiring Hospitalization							
Chronic bronchitis, using PM_{10} indicator	Schwartz, 1993b		Annual mean	Annual mean	All	N/A	0.012
Acute bronchitis, using $PM_{2.5}$ indicator	Dockery et al., 1989	Logistic	Annual mean	Annual mean[d]	Ages 10-12	N/A	0.0298

(continued)

Table 7-3. (continued)

Endpoint	Concentration-Response Function		PM Averaging Time		Population[a]	Annual Baseline Incidence (per 100,000 population)[b]	Pollutant Coefficient[c]
	Source	Functional Form	Studied	Applied			
Upper respiratory symptoms (URS), using PM_{10} indicator	Pope et al., 1991	Loglinear	1-day average	1-day average	Asthmatics, ages 9-11	38,187 (applied population)	0.0036
Lower respiratory symptoms (LRS), using $PM_{2.5}$ indicator	Schwartz et al., 1994	Logistic	1-day average	1-day average	Ages 8-12	N/A	0.01823
MRADs, using $PM_{2.5}$ indicator	Ostro and Rothschild, 1989	Loglinear	2-week average	1-day average	Ages 18-65	780,000 d/yr (applied population)	0.00741
RADs, using $PM_{2.5}$ indicator	Ostro, 1987	Loglinear	2-week average	1-day average	Ages 18-65	400,531 d/yr (applied population)	0.00475
Acute respiratory symptoms (any of 19), using PM_{10} indicator	Krupnick et al., 1990	Logistic	1-day average COH	1-day average	Ages 18-65 (study examined "Adults")	N/A	0.00046
Shortness of breath (days), using PM_{10} indicator	Ostro et al., 1995	Logistic	1-day average	1-day average[d]	African-American asthmatics, ages 7-12	N/A	0.00841

(continued)

Table 7-3. (continued)

Endpoint	Concentration-Response Function		PM Averaging Time		Population[a]	Annual Baseline Incidence (per 100,000 population)[b]	Pollutant Coefficient[c]
	Source	Functional Form	Studied	Applied			
Work loss days (WLDs), using $PM_{2.5}$ indicator	Ostro, 1987	Loglinear	2-week average	1-day average	Ages 18-65	150,750 d/yr (applied population)	0.0046

NOTES:

[a] The population examined in the study and to which this analysis applies the reported concentration-response relationship. In general, epidemiological studies analyzed the concentration-response relationship for a specific age group (e.g., ages 65+) in a specific geographical area. This analysis applies the reported pollutant coefficient to all individuals in the age group nationwide.

[b] Annual baseline incidence in the applied population per 100,000 individuals in the indicated population. For hospital admissions and mortality, the national baseline incidence rates are meant to provide the reader with a general perspective of the potential magnitude of the baseline incidence; for other endpoints, the annual baseline incidence estimates were taken directly from the epidemiological literature and were applied to all sectors in the analysis.

[c] A single pollutant coefficient reported for several studies indicates a pooled analysis; see text for discussion of pooling concentration-response relationships across studies.

[d] The following studies report a lowest observed pollution level:

Pope et al., 1995	Mortality (long-term exposure)	9 $\mu g/m^3$ $PM_{2.5}$
Dockery et al., 1995	Acute bronchitis	11.8 $\mu g/m^3$ $PM_{2.5}$ (20.1 $\mu g/m^3$ PM_{10})
Ostro et al., 1995	Shortness of breath, days	19.63 $\mu g/m^3$ PM_{10}

The remaining studies did not report lowest observed concentrations.

[e] Pooling of the ten studies used for this endpoint is described in EPA (1996).

[f] All 1-day averages are 24-hour averages, 2-day averages are 48-hour averages, etc.

design (a prospective cohort study) is best able to identify the long-term exposure effects and will likely detect some of the short-term exposure effects as well.

The second reason that long-term studies may be preferable to short-term studies is that long-term study results may be less likely to be affected by deaths that are premature by only a very short amount of time. Critics of the use of short-term studies for policy analysis purposes correctly point out that an added risk factor that results in terminally ill individuals dying a few days or weeks earlier than they otherwise would have (a phenomenon referred to as "harvesting") is potentially included in the measured PM mortality "signal" detected in such a study. Because the short-term study design does not examine individual people (but instead uses daily mortality rates in large, typically city, populations), it is impossible to know anything about the overall health status of the people who die on any given day. Although some of the excess deaths associated with peak PM exposures may have resulted in a substantial loss of life (measuring loss of life in terms of lost years of remaining life), others may have resulted in a relatively short amount of lifespan lost. Although it is not clear that the results of prospective cohort (long-term) studies are completely unaffected by "harvesting," because they follow individuals, such studies are better able to examine the health status of individuals who die during the course of the study.

Although long-term exposure studies may be preferable, only one is presented in this analysis. Therefore, results of studies that use short-term PM exposures are also presented in this analysis for comparison. However, because a long-term exposure study may detect some of the same short-term exposure effects detected by short-term studies, including both types of study in a benefit analysis would likely result in some degree of double counting of benefits.

7.4.2 Hospital Admissions Studies

Several studies have investigated the association between ambient PM concentrations and increased hospital admissions for a variety of ailments and among different population groups. These studies and the issues of overlap among the endpoints and populations investigated are described below. All of these studies compare PM concentrations averaged over 1 to 2 days with daily hospital admissions.

7.4.2.1 Hospital Admissions for Respiratory Illnesses. Several studies have investigated hospital admissions specifically for respiratory ailments. Two estimates are available for hospital admissions for "all respiratory illnesses." The first study, Thurston et al. (1994), investigated respiratory admissions for individuals of all ages. The pooled analysis using information from Schwartz (1995, 1996) estimates all respiratory hospital admissions for individuals aged 65 years and older. Studies of hospital admissions for chronic obstructive pulmonary disease (COPD) and pneumonia, which are both subsets of hospital admissions for all respiratory diseases, are also presented.

Because Thurston et al. (1994) include hospital admissions for a large group of respiratory illnesses and all age groups, this study is the most comprehensive and is therefore considered to be the most appropriate study for predicting changes in hospital admissions for respiratory illnesses related to PM exposure. Because Schwartz (1994a,b,c, 1996) estimates incidence for a subset of hospital admissions counted by Thurston et al. (1994), the incidence

predicted by the Schwartz studies should not be added to the incidence predicted by Thurston et al. (1994).

7.4.2.2 Hospital Admissions for Cardiac Disease. Hospital admissions for ischemic heart disease and congestive heart failure related to PM exposure have been investigated by Schwartz and Morris (1995). These admissions are not included in the group of respiratory illness hospital admissions. In addition, there is no overlap between hospital admissions for ischemic heart disease and admissions for congestive heart failure. Therefore, they can both be counted as benefits associated with reducing exposure to PM.

7.4.3 Respiratory Symptoms and Restricted Activity Days

Several studies have investigated changes in a variety of respiratory symptoms not requiring admission to the hospital. These studies have investigated illnesses in both the general population and in asthmatic individuals; many of the studies have used children as the study population. The types of symptoms investigated and issues related to potential overlap among the symptoms examined in these studies are described here. Because some of these symptoms may vary only slightly among the studies, there is considerable overlap among the health effects investigated in these studies. Table 7-4 defines the symptoms and the populations investigated for each of the studies presented in this analysis.

7.4.3.1 Respiratory Illnesses Measured in the General Population. There may be some overlap between bronchitis studied by Dockery et al. (1989) and chronic bronchitis defined by Schwartz (1993b). In particular, Dockery et al. (1989) considered the effects of PM exposure on bronchitis that was diagnosed by a doctor within the previous year, which may include some of the same types of cases investigated by Schwartz (1993b). Although the bronchitis measured in Dockery et al. (1989) is likely to include more cases of acute bronchitis than the bronchitis cases measured by Schwartz (1993b), the measure in Dockery et al. (1989) may also include some cases of chronic bronchitis if the cases diagnosed in the year prior to the study continue into future years. For this reason, and because the populations studied overlap each other, the estimates of avoided incidence based on these studies are not necessarily mutually exclusive. However, both studies give valuable information regarding the incidence of bronchitis avoided in two different population groups.

Lower respiratory symptoms (LRS), as described in Schwartz et al. (1994), are distinct from doctor-diagnosed bronchitis and therefore do not overlap with the avoided cases of bronchitis.

There are several aggregation issues related to the set of endpoints that are studied in adults. Acute respiratory symptoms (any of 19 symptoms) studied by Krupnick et al. (1990) may overlap with minor restricted activity days (MRADs) studied by Ostro and Rothschild (1989) because the age ranges of the populations studied are the same, and it is possible that an acute respiratory symptom could result in a minor respiratory restricted activity day. The degree of overlap, however, is not known, and it is possible that some of the benefit associated with each endpoint is not included within the benefit associated with the other endpoint.

Table 7-4. Descriptions of Studies of Respiratory Symptoms Not Requiring Hospitalization

Health Endpoint, PM Indicator	Definition of Health Endpoint	Population Studied	Reference
Chronic bronchitis, using PM_{10} indicator	Chronic bronchitis was defined as positive responses to the following questions: (1) whether a doctor had ever told the subject that he or she had chronic bronchitis and (2) whether he or she still had bronchitis at the time of the study.	All	Schwartz, 1993b
Acute bronchitis, using $PM_{2.5}$ indicator	Bronchitis was defined as a doctor's diagnosis of bronchitis reported within the year prior to the study. Occurrence of bronchitis diagnosed during the year was compared with the annual mean PM concentration reported during the year.	Ages 10-12	Dockery et al., 1989
Upper respiratory symptoms (URS), using PM_{10} indicator	URS includes runny or stuffy nose; wet cough; and burning, aching, or red eyes. Presence of symptoms on a given day were compared with the PM concentration on the same day.	Asthmatics, ages 9-11	Pope et al., 1991
Lower respiratory symptoms (LRS), using $PM_{2.5}$ indicator	LRS is the presence of at least two of the following symptoms: cough, chest pain, phlegm, or wheeze. Presence of symptoms on a given day was compared with PM concentrations measured on the previous day; symptoms were counted only if they were not present on the previous day.	Ages 8-12	Schwartz et al., 1994
Minor Restricted Activity Days (MRADs), using $PM_{2.5}$ indicator	An MRAD is a day in which an individual restricts his or her activity due to either respiratory or nonrespiratory symptoms; an MRAD does not result in either work loss or bed disability Occurrence of MRADs was compared with PM concentrations averaged over a 2-week period.	Ages 18-65	Ostro and Rothschild, 1989
Restricted Activity Days (RADs), using $PM_{2.5}$ indicator	A RAD is a day in which an individual restricts his activity; RADs include both days of work loss or bed disability as well as minor restrictions. Occurrence of RADs was compared with 2-week average PM concentrations.	Ages 18-65	Ostro, 1987

(continued)

Health Endpoint, PM Indicator	Definition of Health Endpoint	Population Studied	Reference
Acute respiratory symptoms (any of 19), using PM_{10} indicator	The study measured daily presence of any of 19 symptoms, including chest discomfort, coughing, wheezing, sore throat, cold, doctor-diagnosed flu, asthma, hay fever (all symptoms considered were not reported in the study)	Adults	Krupnick et al., 1990
Shortness of breath, using PM_{10} indicator	The study measured daily presence of shortness of breath.	African-American asthmatics, ages 7-12	Ostro et al., 1995
Work loss days (WLDs), using $PM_{2.5}$ indicator	Days of work loss were compared with 2-week average PM concentrations.	Ages 18-65	Ostro, 1987

MRADs and Work Loss Days (WLDs) are defined specifically as mutually exclusive endpoints (Ostro and Rothschild, 1989). Both of these estimates (MRADs and WLDs) are subsets of Restricted Activity Days (RADs). However, because the concentration-response functions for RADs and MRADs were estimated by different studies, there is no guarantee that the predicted incidence of MRADs will be less than the predicted incidence of RADs.

7.4.3.2 Respiratory Illnesses Measured in the Asthmatic Population. Three studies in Table 7-4 measured respiratory illnesses exclusively in asthmatic individuals. Pope et al. (1991) studied upper respiratory symptoms (URS) in children ages 9 to 11. Ostro et al. (1995) measured shortness of breath among African-American asthmatics ages 7 to 12[2].

Estimates using Pope et al. (1991) do not appear to overlap with estimates predicted using Ostro et al. (1995).

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[2]Another study, Ostro et al. (1991), measured days of moderate or worse asthma status in adults. Although this study investigated health effects in a population (asthmatics) that is important to consider, the concentration-response function from the study was not used in the current analysis because the incidence estimated using the study is very sensitive to the actual baseline and control scenario air quality data. Because this analysis uses only the air quality contributed by hazardous waste combustors without adding other ambient anthropogenic and natural air concentrations, the actual incidence could not be estimated.

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8.0 Human Health Risk Characterization Methodology

This section describes the risk characterization methodology used to evaluate the potential human health benefits associated with the proposed emission control strategies for HWC facilities. The HWC risk analysis completed for the final rule characterized risk for those human receptors who may experience significant exposure to constituents released from HWC facilities due to their proximity to these facilities and/or their behavior[1]. In addition to characterizing risks to human receptors residing within the vicinity of HWC facilities (termed "local" receptors), the analysis also assessed annual cancer incidence in the general population resulting from the ingestion of agricultural commodities that are produced within the vicinity of HWC facilities but distributed nationally for consumption.

The HWC risk analysis evaluated specific categories of risk—cancer effects and noncancer effects. Each of the chemicals evaluated within the HWC risk analysis can be placed into one or both of these categories of risk depending on the health effect being considered (e.g., dioxin was evaluated for cancer and noncancer effects). A risk descriptor is a specific type of risk estimate (e.g., individual cancer risk estimates or statistical cancer incidence estimates for local populations) that is used as the metric for a given risk category. Each of the risk categories is characterized using a suite of risk descriptors (e.g., cancer risk is characterized using both individual and population-level risk descriptors in the form of lifetime excess cancer risk estimates and annual excess cancer incidence, respectively).

Section 8.1 provides an overview of the risk descriptors used in the HWC risk analysis. Section 8.2 discusses the methodologies used to characterize individual risk (e.g., individual cancer risk and individual blood lead level analysis), and Section 8.3 describes the analysis methodologies used to characterize population-level risk (e.g., local cancer incidence, avoided incidence estimates for PM exposure).

8.1 Risk Descriptors

A variety of risk descriptors were used in the HWC risk analysis to provide coverage for the range of health effects potentially associated with human exposure to the constituents modeled in the analysis. These risk descriptors can be broadly characterized as either describing

[1] The HWC risk analysis assessed risks for a variety of different receptors located in the vicinity of HWC facilities. Although these receptors display a wide range of exposures and risks, the term "significant exposure" is considered appropriate here since certain receptors were screened out at proposal due to low projected risks (e.g., the commercial poultry farmer).

Table 8-1. Risk Descriptors Used for Risk Categories

Risk Category	Risk Descriptor	
	Individual Risk	**Population Risk**
Carcinogens	Lifetime excess cancer risk	Annual excess cancer incidence (local and national population)
Noncarcinogens	Ingestion hazard quotient	Number of exceedances (non-cancer effects)
	Inhalation hazard quotient	
	Ingestion hazard index	
	Inhalation hazard index	
Lead	Body burden (blood lead levels)	Excess exceedances (blood lead)
Dioxin (non-cancer)	Incremental margin of exposure	Not applicable
Particulate Matter	Not applicable	Avoided incidence

the magnitude of health impacts to the modeled individual (i.e., individual risk estimates) or the magnitude of health impacts to specific receptor populations (i.e., population risk estimates). Risk descriptors are listed in Table 8-1 and discussed in the following sections.

8.1.1 Individual Risk

Individual risk estimates characterize the risk experienced by individuals from a specific receptor residing within HWC study areas. These risk estimates were typically generated at the sector level by combining modeled dose estimates for specific constituents with corresponding toxicity factors (e.g., CSFs or RfDs). Each of these risk descriptors is briefly described in the following subsections.

8.1.1.1 <u>Lifetime Excess Cancer Risk</u>. Cancer risk was characterized using lifetime excess cancer risk estimates to represent the excess probability of developing cancer over a lifetime as a result of exposure to the constituent of interest. Lifetime excess cancer risk estimates, which were generated at the sector level, are the product of the lifetime average daily dose for a specific receptor and the corresponding cancer slope factor, as shown in Equation 8-1:

$$\textit{Lifetime excess cancer risk} = LADD \bullet CSF \qquad (8\text{-}1)$$

Parameter	Definition (units)
LADD	Lifetime average daily dose (mg/kg BW/d)
CSF	Cancer slope factor $(mg/kg\ BW/d)^{-1}$

The cancer slope factor is derived from either human or animal data and is taken as the upper bound on the slope of the dose-response curve in the low-dose region, generally assumed to be linear, expressed as a lifetime excess cancer risk per unit exposure. The same slope factor was used for estimating cancer risks in both adults and children. However, individuals exposed to carcinogens in the first few years of life may be at increased risk of developing cancer. Therefore, significant uncertainties and unknowns exist regarding the estimation of lifetime cancer risks in children.

For inhalation carcinogens, a cancer slope factor was derived from the unit risk factor. The unit risk factor presumes an adult exposure for a lifetime. However, the HWC risk analysis is intended to assess risks from less than lifetime exposures to both adults and children. Therefore, a slope factor was derived from the unit risk factor, as shown in Equation 8-2. The slope factor was then used in conjunction with a receptor-specific LADD to estimate the lifetime excess cancer risk as shown in Equation 8-1.

$$CSF = \frac{URF \cdot BW}{IR} \cdot 1000\ \mu g/mg \qquad (8\text{-}2)$$

Parameter	Definition (units)
CSF	Cancer slope factor $(mg/kg\ BW/d)^{-1}$
URF	Unit risk factor $(\mu g/m^3)^{-1}$
BW	Body weight used to derive the URF (70 kg)
IR	Inhalation rate used to derive the URF (20 m^3/d)
1,000 µg/mg	Units conversion factor

8.1.1.2 <u>Total Lifetime Excess Cancer Risk</u>. Constituent-based individual lifetime excess cancer risks were generated for each age group of each receptor for each combustor category. These constituent-based lifetime excess cancer risks were then summed to generate additive total lifetime excess ingestion and inhalation risks for each age group of each receptor for each combustor category. Although exposures to multiple carcinogens may not result in additive risks, assuming additivity is considered an appropriate, if sometimes conservative, assumption in the absence of specific evidence of synergism or antagonism.

8.1.1.3 <u>Ingestion Hazard Quotient</u>. Noncancer risk is characterized through the use of hazard quotients, which are generated by dividing an average daily dose by the corresponding reference dose (RfD). The ingestion hazard quotient uses the average daily dose as the exposure metric (see Section 6.4). An HQ establishes whether a particular individual has experienced exposure that places him or her either above or below a threshold of concern for a specific health effect. Therefore, unlike cancer risk estimates, HQs are not probability statements. The reference dose represents a "no-effects" level that is presumed to be without appreciable risk from chronic exposures over a lifetime. The RfD may be derived from human or animal studies and may include uncertainty factors to account for deficiencies in the available studies. Equation 8-3 shows the derivation of the ingestion hazard quotient:

$$HQ_{ing} = \frac{ADD}{RfD} \qquad (8\text{-}3)$$

Parameter	Definition (units)
ADD	Average daily dose (mg/kg-d)
RfD	Reference dose (mg/kg-d)

8.1.1.4 <u>Ingestion Hazard Index</u>. Constituent-based individual ingestion hazard quotients were generated for each age group of each receptor for each combustor category. Through the assumption of additivity, HQs can be summed across constituents to generate a hazard index (HI) for a specific receptor/pathway combination. In the HWC analysis, these constituent-based hazard quotients were summed to generate additive ingestion HIs for each age group of each receptor for each combustor category. The hazard index is a measure of the potential risk of adverse health effects from a mixture of chemical constituents. Whether or not a particular chemical mixture poses an additive risk depends on the targets (tissue, organ, or organ system) and the mechanisms of action of the individual chemicals.

8.1.1.5 <u>Inhalation Hazard Quotient</u>. The inhalation hazard quotient is similar to the ingestion hazard quotient in that it represents a ratio of an exposure to a reference value. However, unlike the ingestion hazard quotient, which uses the average daily dose as the exposure metric, the inhalation hazard quotient uses an air concentration as the exposure metric. This concentration is compared to a reference concentration (or RfC). Like the reference dose, the

reference concentration represents a "no-effects" level that is presumed to be without appreciable risk of adverse effects from chronic exposures over a lifetime. The RfC may be derived from human or animal studies and may include uncertainty factors to account for deficiencies in the available studies. Equation 8-4 shows the derivation of the inhalation hazard quotient.

$$HQ_{inh} = \frac{C_{air}}{RfC} \qquad (8\text{-}4)$$

Parameter	Definition (units)
C_{air}	Ambient air concentration (µg/m^3)
RfC	Reference concentration (µg/m^3)

8.1.1.6 <u>Inhalation Hazard Index</u>. As was the case with ingestion hazard quotients, constituent-based inhalation hazard quotients were summed to generate additive inhalation HIs for each age group of each receptor for each combustor category. Like the ingestion hazard index, the inhalation hazard index is a measure of the potential risk of adverse health effects from a mixture of chemical constituents. Whether or not a particular chemical mixture poses an additive risk depends on the targets (tissue, organ, or organ system) and the mechanisms of action of the individual chemicals.

8.1.1.7 <u>Body Burden (Blood Lead Levels)</u>. Because an RfD has not been developed for lead, the potential for adverse effects resulting from exposure of children (0- to 5-yr-old age group) to this metal was evaluated by comparing modeled blood lead levels (PbB levels) to the health-based level established for lead of 10 µg/dL (see Section 7.3.11)[2]. As with noncancer HQs, an exceedance of the health-based level should not be interpreted as a probability statement concerning the potential for adverse health effects. Instead, it identifies the modeled individual as having the potential for experiencing an adverse effect.

Incremental, background, and total (incremental plus background) individual blood lead level results were generated for a single age group (0 to 5 years) for each combustor category.

8.1.1.8 <u>Incremental Margin of Exposure (Dioxin-TEQ)</u>. An RfD has not been developed for use in characterizing noncancer health effects resulting from exposure to dioxins/furans. Instead, this category of risk was characterized using an incremental margin of exposure (MOE) as the risk descriptor. The average daily dose in terms of 2,3,7,8-TCDD toxicity equivalents (dioxin-TEQ) was compared to background dioxin-TEQ exposures. The incremental MOE analysis establishes whether modeled ADDs for a given receptor exceed typical

[2] A PbB level of 10 µg/dL is the blood lead level at which community-wide lead poisoning prevention activities are indicated (CDC, 1991).

background exposure experienced by the general population; if so, they must be carefully interpreted when drawing conclusions concerning risk. The HWC risk analysis included an incremental MOE analysis for all modeled receptors as well as for infant exposure to dioxin-TEQ through breast milk ingestion.

Individual incremental MOE results were generated for each age group of each receptor for each combustor category. Additionally, incremental MOE results were generated for infants exposed to dioxin-TEQ through ingestion of breast milk; these infants are assumed to be children of women in the 12- to 19- and >19-yr-old age groups.

8.1.2 Population Risk

The HWC risk analysis assessed population-level risk for a number of cancer and noncancer effects impacting individuals residing within study areas (i.e., local populations). Because these estimates require sector-level population data, they could be completed only for enumerated receptors (see Section 4.4). Population-level risk estimates have been generated for the following effects: (1) annual excess cancer incidence resulting from exposure to modeled carcinogens through all exposure pathways including both direct and indirect; (2) potential exceedances of the methylmercury RfD resulting from mercury exposure through fish ingestion; (3) excess incidence of elevated blood lead associated with lead exposure; and (4) avoided incidence of inhalation effects resulting from exposure to $PM_{2.5}$ and PM_{10}.[3] In addition to characterizing population-level risk for individuals residing within study areas (i.e., local population risk), this analysis also assessed the annual cancer incidence in the national population due to dioxin/furan exposures from the consumption of locally produced agricultural commodities (i.e., annual cancer incidence due to agricultural commodity risk). Each of the risk descriptors used in characterizing population risk is described briefly below.

8.1.2.1 <u>Annual Excess Cancer Incidence (Local Population)</u>. Cancer population risk for local receptor populations was characterized using annual excess cancer incidence estimates, which represent the number of excess cancer cases projected to occur within a given receptor population due to exposure to carcinogens released from HWC facilities. These estimates were derived by multiplying mean sector-level lifetime excess cancer risk estimates (i.e., LADD • CSF) for a given constituent by the number of individuals from that receptor population located in that sector. The resulting value (which represents the lifetime excess cancer incidence estimate in a given population cohort) was then divided by the exposure duration for that receptor population to produce an annual incidence estimate. This estimate represents the rate of incidence of cancer in the exposed population per year of exposure (regardless of when those cancers may occur) and

[3] Local population risk characterization for noncancer effects also included an analysis of inhalation risk for Cl_2 and HCl. Specifically, each of the sectors making up study areas within a given combustor categhor were queried to determine whether those sectors had inhalation HQs for Cl_2 and HCl that exceeded a health benchmark of 1.0 for any of the enumerated receptor populations. If any of the sectors had been identified as having HQ exceedances, the population counts for the receptor populations with the HQ exceedances would have been totaled to generate the population counts for the receptor populations with the HQ exceedances would have been totaled to generate the population risk estimate for that combustor category. However, no exceedances of the health benchmark for either Cl_2 or HCl were identified; consequently results for this population risk category are not reported.

applies to any population cohorts exposed over time. The annual excess cancer incidence is the excess cancer incidence in a given receptor population per year of exposure and, as such, includes all cancers regardless of when they may occur over individuals' lifetimes. The annual excess cancer incidence was calculated as shown in Equation 8-5.

$$\textit{Annual excess cancer incidence} = \frac{LADD \bullet CSF \bullet Population}{ED} \qquad (8\text{-}5)$$

Parameter	Definition (units)
LADD	Lifetime average daily dose (mg/kg-d)
CSF	Cancer slope factor (per mg/kg-d)
Population	Exposure population (individuals)
ED	Exposure duration (years)

Constituent-based annual excess cancer risks were generated for each receptor (all age groups combined, as well as by age group) for each combustor category. These constituent-based annual excess cancer risks were then summed to generate additive (ingestion and inhalation) risks for each receptor for each combustor category.

8.1.2.2 <u>Annual Excess Cancer Incidence (Agricultural Commodity Risk)</u>. Cancer population risk for the national population resulting from the ingestion of locally produced agricultural commodities containing dioxins/furans was characterized using annual excess cancer incidence estimates. These estimates were generated by:

- Projecting the level of dioxin/furans in key agricultural commodities (i.e., milk, beef and pork) at the sector level
- Generating sector-level projections of amounts of these key commodities that are produced
- Combining these two factors to make sector-level projections concerning the amount of "diet-accessible" dioxin/furans
- Using these estimates (expressed as dioxin-TEQs) together with EPA's population risk equation to project the annual number of statistical cancer cases resulting from the consumption of the diet-accessible dioxin-TEQ.

This calculation (see Equation 8-6) uses the ingestion cancer slope factor for 2,3,7,8-TCDD. These population-level cancer risk estimates represent the number of excess cancer cases

projected to occur within the national population per year of exposure due to the ingestion of dioxins/furans contained in locally produced agricultural commodities.

The annual statistical cancer incidence results for the general population were generated by combustor category for beef, pork, and dairy ingestion.

$$\textit{Agricultural Commodity Annual Excess Cancer Incidence} = \frac{CSF \cdot ED}{BW \cdot LT} \sum_{i=1}^{n} (FPi * Cfi) \qquad (8\text{-}6)$$

Parameter	Definition
Agricultural Commodity Annual Excess Cancer Incidence	National annual statistical cancer risk estimate for dioxin (excess number of annual statistical cancer cases) due to consumption of locally produced agricultural commodities
CSF	Ingestion cancer risk factor for dioxin $(mg/kg\text{-}d)^{-1}$
ED	Duration of exposure (1 year)
BW	Body weight (70 kg)
LT	Lifetime (70 yr)
n	Number of study area sectors within a given combustor category
FPi	Average annual food production (beef, pork, milk) within HWC study area sectors “1” through “n” (kg-d).
Cfi	Contaminant concentration in food from modeled HWC study area sectors “1” through”n” (mg/kg) (Note: Cfi * FPi produces the “diet-accessible dioxin” estimate for each sector)

8.1.2.3 <u>Excess Exceedances (Mercury)</u>. Excess exceedances were calculated for the adult recreational fisher receptor population. It is not possible at present to characterize the level of recreational fishing activity at specific waterbodies (i.e., number of recreational fishers engaging in fishing activity and the number of fishing trips made at a specific waterbody over a year). Therefore, for the final rule, qualitative population-level risk statements were generated for the recreational fisher. These qualitative statements identified the number of recreational fishers associated with “at-risk” HWC facilities. An at-risk facility is defined as a facility whose 95th percentile individual risk level for methylmercury exposure exceeds a health benchmark of 1.0. That is, recreational fishing activity could place a portion of that facility’s recreational fishers at unacceptable risk for adverse health impacts from methylmercury exposure. The 95th percentile methylmercury ingestion HQ levels used to classify facilities as to at-risk status were obtained from a cumulative risk distribution generated separately for each modeled study area based on sector-level individual risk estimates for the recreational fisher (i.e., methylmercury ingestion HQ estimates). The cumulative risk distributions were generated for each study area using a

Monte Carlo simulation approach that integrated exposure parameter variability (in fish ingestion rates) into the cumulative risk distributions. It is important to note that the qualitative population-level risk analysis for the recreational fisher completed for the final rule identified no at-risk facilities for any of the combustor categories considered in the analysis.

Significant uncertainty is associated with the qualitative population-level risk analysis completed for the recreational fisher resulting primarily from an inability to characterize recreational fishing activity at specific waterbodies. Methylmercury fish tissue concentrations can vary greatly between waterbodies that are proximate to one another, which can translate into widely varying individual-level and population-level risk results depending on the pattern of recreational fishing activity that is modeled. Consequently, not being able to accurately characterize recreational fishing activity at the waterbodies modeled for a given study area introduced significant uncertainty into both the individual- and population-level risk estimates.

8.1.2.4 Excess Exceedances (Blood Lead). Population-level risk resulting from exposure to lead is characterized by identifying the number of additional (excess) children (0 to 5 years of age) from a particular receptor population who have modeled PbB levels above the HBL for lead (i.e., the action level of 10 µg/dL) resulting from incremental exposure to HWC emissions. These population-level risk estimates were generated by:

- Estimating the number of children within a given sector who exceed the HBL for lead because of background exposure

- Estimating the number of children in that same sector who exceed the HBL because of total lead exposure (background plus incremental)

- Subtracting the background value from the total value to estimate the number of children in that sector exceeding the HBL because of incremental exposure alone (i.e., incremental exceedances)

- Repeating this procedure for all sectors within a given combustor category and adding the resulting sector-level incremental exceedance estimates to produce a total estimate for a given combustor category.

The incremental, background, and total (incremental plus background) population blood lead level results were generated for a single age group (0 to 5 years) for each combustor category and are presented as annualized projections.

8.1.2.5 Avoided Incidence. This category of population-level risk results characterizes reductions in the annual incidence of specific health endpoints related to particulate matter (PM) exposures that are projected to result from MACT standards (i.e., from reductions in ambient PM concentrations that would result from the implementation of the MACT standards). The PM analysis used concentration-response functions, which are based on epidemiological studies, to relate changes in ambient PM levels that would result from implementation of the MACT standard to changes in the incidence of specific health endpoints.

The concentration-response functions used in this analysis were based on epidemiological studies that covered a variety of different conditions and, consequently, provide risk estimates for a range of acute and chronic health endpoints. Avoided incidence results are presented for these health endpoints by combustor category and reflect the change in ambient PM concentrations that would be achieved by implementing the MACT standards.

8.2 Individual Risk Analysis

Individual risk estimates were derived to characterize the range of risk experienced by individuals from a specific receptor population residing within HWC study areas. Sector-level individual risk estimates for a specific receptor population (and health effect) were pooled to form a cumulative risk distribution that represents the range of individual risk experienced by the individuals in that population.[4] Specific individual risk percentiles (i.e., 50th, 90th, 95th, and 99th) could then be identified using the cumulative risk distribution. Each of these percentiles represents the risk level experienced by the individual located at that specific point on the risk distribution. Although the majority of the individual risk estimates generated for the HWC risk analysis were based on central tendency exposure parameters, a refined set of individual risk estimates reflecting variability in exposure factors (i.e., interindividual variability in exposure) was generated for key risk-driving receptors (see Section 8.2.3). Risk descriptors for individual risk are shown in Table 8-2. These risk descriptors are discussed in detail in the following sections.

8.2.1 Cancer Risk

Individual cancer risk is expressed as the lifetime excess individual cancer risk experienced by specific percentiles of a given receptor population (i.e., 50th, 90th, 95th, and 99th). Characterization of individual risk for carcinogenic effects is based on sector-level individual risk estimates. The sector-level estimates were generated by combining modeled lifetime average daily dose estimates for a given chemical/pathway combination (Section 6.4) with the cancer slope factor specific to that chemical/pathway (Section 7.1) using Equation 8-1 to produce an individual lifetime excess cancer risk.

Sector-level individual cancer risk estimates for all of the sectors within the combustor category being evaluated (i.e., for all of the sectors within the HWC study areas comprising that category) were pooled to form route-specific and chemical-specific cumulative risk distributions that provide the basis for identifying individual risk percentiles. The following steps were required to generate individual risk percentiles.

- **Generate sector-level individual risk estimates.** Sector-level excess cancer risk estimates were generated for each receptor/constituent combination using mean exposure parameters. Consequently, each individual risk estimate that results

[4] For enumerated receptor populations, sector-level individual risk estimates were first population-weighted prior to formation of a cumulative risk distribution to allow the distribution of receptors across the modeled study areas to be reflected in the cumulative risk distribution that was formed. All sector-level individual risk estimates (for both enumerated and non-enumerated receptors) were also facility-weighted to allow the individual risk percentiles that were generated to be extrapolated to the universe of HWC facilities.

Table 8-2. Risk Descriptors Used in Individual Risk Categories

Risk Descriptor	Individual Risk (central tendency assumption)	Individual Risk (exposure parameter variability analysis)
Lifetime excess cancer risk	Cumulative distribution of excess individual risk across modeled sectors. Specific individual risk percentiles are identified.	Excess individual risk levels for dioxins/furans for specific percentiles of the population (including consideration of interindividual variability)
Hazard quotient (inhalation and ingestion)	Cumulative distribution of individual noncancer risk across modeled sectors. Specific individual HQ percentiles are identified. For recreational fisher, "minimum," "median," and "maximum" HQ values characterizing the range of methylmercury risks experienced by recreational fishers in a given combustor category.	For recreational fisher only, HQ values for methylmercury for specific percentiles of the population (including consideration of interindividual variability)
Body burden (blood lead levels)	Not applicable	Cumulative distribution of individual blood lead levels across sectors. Specific individual blood lead level percentiles are identified.
Incremental margin of exposure (MOE): TCDD-TEQ	Cumulative distribution of individual incremental MOE results across modeled sectors. Specific individual percentiles are identified	Not applicable

represents the mean risk for the portion of the receptor population located in a specific sector. (Note: In actuality, there is a range of individual risks for the group of individuals located within each sector, but this is considered in the exposure parameter variability analysis—see Section 8.2.3.) Once sector-level individual risk estimates were generated for all chemical/pathway combinations, these results were aggregated across pathways **at the sector level** to produce chemical-specific risk estimates for both the ingestion and inhalation exposure routes.

Generate the cumulative risk distribution incorporating facility sampling weights and population weights. The sector-level individual cancer risk estimates generated in Step 1 were pooled and then ranked from lowest risk to highest risk to form a cumulative risk distribution. This procedure was completed separately for each of the chemical/exposure route combinations. For those receptor populations that could be enumerated (see Section 4.4), each of the sector-level risk values was population-weighted prior to generate the cumulative risk distribution. Each sector-level risk value was assigned a weight equal to the number of individuals from the receptor population being considered that are

located in that sector (i.e., the individual risk value for a particular sector was represented a specified number of times in the cumulative risk distribution corresponding to that sector's population). All sector-level risk estimates (i.e., for both enumerated and non-enumerated receptors) were also facility-sample-weighted prior to the formation of the cumulative risk distribution. This was done by using the facility sampling weight for a specific HWC facility to adjust all of the sector-level individual risk estimates associated with that facility in a manner identical to that described above for population weighting (i.e., each sector-level individual risk estimate was represented within the cumulative risk distribution with a frequency equal to the facility sampling weight). In the case of enumerated receptor populations, the population weight for a specific sector was multiplied by the facility sampling weight for that sector to generate a hybrid weighting factor reflecting both attributes. This hybrid factor was then used to represent that sector's individual risk estimate within the cumulative risk distribution. A cumulative risk distribution generated using this approach characterizes the distribution of individual risk for a specific constituent/receptor combination across the sectors comprising a given combustor category.

Identify specific individual risk percentiles. Specific individual risk percentiles were obtained from the cumulative risk distributions generated in Step 2. These individual risk percentiles represent the mean risk to individuals in a sector located at a given percentile of the risk distribution (e.g., a 90th percentile individual risk estimate represents the mean risk experienced by the individual located at the 90th percentile of the risk distribution). This interpretation is applicable only with enumerated receptor populations because the distribution of individuals across modeled study areas has been incorporated into the construction of the cumulative risk distributions. Emphasis was placed in the HWC risk analysis on characterizing the distribution of risk in the receptor population. Therefore, with regard to the characterization of individual risk, both central-tendency (50th percentile) and high-end (90th, 95th, and 99th percentile) individual risk percentiles were identified for all receptor population/constituent combinations.

The individual lifetime excess ingestion and inhalation cancer risks of individual constituents were combined to generate an exposure-route-specific (i.e., ingestion or inhalation) total lifetime excess cancer risk. Carcinogenic effects of single constituents evaluated for individuals on a sector-level basis were summed under the assumption of additivity of effects. This assumption introduced an element of uncertainty because it fails to incorporate the potential for synergistic and/or antagonistic effects of exposure to multiple constituents.

An exposure parameter variability analysis generates refined individual percentile risk estimates that reflect interindividual variability in exposure factors. This is discussed in Section 8.2.3.

8.2.2 Noncancer Risk

Individual risk characterization for noncancer risk resembles the methodology used for carcinogens in that sector-level HQ values were used to generate a cumulative risk distribution that could, in turn, be used to identify specific individual risk percentiles. Individual noncancer risk was expressed as the hazard quotient experienced by selected percentiles of the receptor population under consideration (percentiles used are identical to those used for carcinogens, i.e., 50th, 90th, 95th, and 99th). As with individual cancer risk, individual HQ values were generated for each receptor/constituent combination using mean exposure parameters. Consequently, these noncancer risk estimates represent the mean HQ values for the portion of the receptor population located in a specific sector. Similarly, the individual noncancer risk percentiles represent the mean HQ values for individuals in a sector located at a given percentile of the risk distribution. Therefore, the procedure outlined in Section 8.2.1 for generating individual risk percentiles for carcinogens can also be applied to hazard quotients for noncarcinogens.[5]

8.2.3 Exposure Factor Variability

This section provides an overview of the exposure parameter variability analysis developed and implemented for the final rule. (Appendix I provides a more detailed discussion of specific topics presented in this section.) The HWC risk analysis completed for the final rule includes an exposure parameter variability analysis that was designed to incorporate interindividual variability in exposure factors into the characterization of individual risk. Specifically, the HWC exposure parameter variability analysis generated cumulative risk distributions that reflect exposure parameter variability. As explained in Section 8.2.1, cumulative risk distributions were used to identify specific individual risk percentiles (i.e., to characterize individual risk).

Although the HWC risk characterization methodology, excluding the exposure variability analysis component, includes consideration of both interfacility variability (with regard to facility source terms) and intersector variability (with regard to both modeled media concentrations and receptor population density), it does not incorporate interindividual variation in exposure factors.

[5] Because it is not possible to characterize recreational fishing activity at specific waterbodies, it is assumed that recreational fishing activity is distributed among modeled waterbodies in a given study area (see Section 6.2.2). Consequently, a single weighted average methylmercury fish tissue concentration is used to generate individual-level risk estimates for each of the 16 sectors at a given study area, resulting in essentially the same methylmercury fish ingestion risk for recreational fishers in each sector. As a result, the effective sample size for risk values that can be used to generate a cumulative risk distribution and ultimately to identify individual risk percentiles for the recreational fisher is significantly reduced relative to other receptor populations (other receptors will usually have a different risk value for each sector within a given study area). The low sample size results in an inability to generate risk percentiles and confidence intervals for this receptor population/constituent combination using SUDAAN. Therefore, rather then presenting individual risk percentiles for the recreational fisher, the following summary risk values were used to characterize individual risk: (1) the risk associated with the lowest-risk modeled facility (“minimum”), (2) the risk associated with the median-risk modeled facility (“median”), and (3) the risk associated with the maximum-risk modeled facility (“maximum”). It is important to note that this discussion applies only to mean exposure-factor-based risk estimates; when exposure parameter variability is incorporated into the characterization of recreational fisher risk, the effective sample size increases and it is possible to generate specific risk percentiles (see Section 8.2.3).

Instead, this methodology generates sector-level risk estimates using central tendency (here, mean) exposure parameter values. With this approach, each mean risk estimate was used to represent all of the individuals from a particular receptor group who were located in the corresponding sector. The HWC exposure parameter variability analysis characterized the range of risks experienced by individuals located in a given sector by incorporating interindividual variability in exposure factors into the characterization of sector-level risks. This was done by generating interindividual variability distributions with the same mean values as in the core HWC risk characterization. This additional data layer was then used to build the cumulative risk distributions for a given receptor population and, ultimately, to characterize individual risk. The resulting distributions then had the same mean risk for the exposed population as in the core methodology, but also reflected interindividual variability in this risk.

Interindividual variability is only one component among many that contribute to variability in exposure. Other contributors are facility parameters, such as emissions and the conditions of release, and fate and transport parameters, such as meteorological conditions, topography, soils, and hydrology, all of which were allowed to vary in the HWC analysis through use (to the degree possible) of site-specific values. These components of variability are generally much larger than interindividual variability in exposure factors (such as food consumption and residence time) and, therefore, are the main determining factors that influence exposures at the high end of the distribution. For this reason, the exposure parameter variability analysis represents a refinement of the core analysis's use of only central tendency (mean) exposure factors. The effect of that refinement on the distribution of risks is determined by interindividual variability in relation to the variability implicit in the core analysis. Therefore, although the inclusion of interindividual variability has the effect of increasing risks at the upper tail of the distribution, the magnitude of that effect may be relatively modest or even minimal depending on the relative magnitude of interindividual variability versus other components of variability.

The HWC exposure parameter variability analysis was used to characterize individual risk for key risk-driving exposure pathways. A sensitivity analysis was performed in which exposure pathways were considered for different combinations of receptor population, constituent, and age group. In all facility categories considered in this analysis, the following exposure pathways contributed more than 95 percent of the risk for all age groups:

- Ingestion of dioxin in beef for the beef farmer
- Ingestion of dioxin in milk for the dairy farmer
- Ingestion of methylmercury in fish for the recreational fisher.

As a result of this sensitivity analysis, the interindividual variability of exposure focused on: (1) ingestion of fish containing methylmercury by recreational fishers, (2) ingestion of beef containing dioxin by commercial beef farmers, and (3) ingestion of milk containing dioxin by commercial dairy farmers. Each of these three pathways was evaluated for each of the four age groups considered in the HWC risk analysis. Because each represents the risk-driving pathway for the receptor population involved, the individual risk estimates generated using the HWC exposure parameter variability analysis can be viewed as being representative of total risks for the receptor population. Any additional variability due to exposure pathways not considered in the analysis of

interindividual variability would produce insignificant changes in the overall variability of risk within the exposed population.

The HWC exposure parameter variability analysis evaluated the aggregate impact of exposure parameter variability associated with three factors: (1) ingestion rate per unit body mass (i.e., for fish, beef, and milk), (2) occupancy period (or residence time), and (3) age correction factor. (Note: The latter two factors are used only in characterizing carcinogenic risk and consequently are applied only to dioxin.)

The exposure parameter variability distributions developed for the HWC exposure parameter variability analysis are based on data contained in the 1997 *Exposure Factors Handbook* (U.S. EPA, 1997). The data sets used to develop these distributions parallel, to the extent possible, the data sets used to generate the central tendency (mean) exposure parameter values that form the basis of the original sector-level risk estimates generated in the core methodology (i.e., the methodology used prior to incorporating interindividual variability of exposure).

The HWC exposure parameter variability analysis was implemented as a postprocessing procedure in the sense that interindividual variability in exposure was integrated into the characterization of risk in each sector after sector-level central tendency risk estimates had been generated (but before these sector-level estimates had been combined to produce the composite risk assessment for the entire exposed population). This adjustment was completed at the sector level by adjusting the central tendency risk estimates generated for each sector to reflect the variance in exposure factors experienced by individuals within that sector (using an interindividual variability distribution with the same mean risk value as in the risk assigned to individuals in that sector during application of the core methodology). Population weighting of sector-level risk estimates was retained in the HWC exposure parameter variability analysis.

In risk assessment, variability analyses are typically implemented by incorporating exposure parameter variability distributions directly into the risk calculation framework and completing multiple iterations of the risk calculation process to generate a pool of risk values that reflects exposure parameter variability. However, the complex nature of the risk calculation framework developed for the HWC risk analysis (i.e., the IEM framework) makes this type of approach infeasible because of the run time required to complete a sufficient number of probabilistic simulation modeling runs using IEM. By using the postprocessing approach, significant run-time reductions were achieved over what would be required to conduct the variability analysis within IEM.

The variability analysis completed for the HWC risk analysis is comprised of two distinct components:

- **Monte Carlo simulation.** A simulation designed specifically to integrate interindividual exposure parameter variability into the relevant cumulative risk distribution. This analysis was completed without the use of SUDAAN and does not allow for the development of confidence intervals reflecting sampling error (see Section 4.1).

- **Discrete approximation approach.** A postprocessing approach that defines intrasector variance using discrete approximations (i.e., histograms) and exports those data to SUDAAN to generate cumulative risk distributions that not only reflect exposure parameter variability but also sampling error (the sole source of uncertainty evaluated quantitatively in the HWC risk analysis using SUDAAN).

Each of these two components of the HWC exposure parameter variability analysis is briefly summarized below.

8.2.3.1 <u>Monte Carlo Simulation Approach</u>. Implementation of the Monte Carlo simulation component of the HWC exposure parameter variability analysis involved the following computational steps (this overview uses the adult commercial beef farmer for purposes of illustration):

1. **Generate a cumulative risk distribution based on central tendency exposure parameters.** Generate a cumulative risk distribution for the adult commercial beef farmer based on central tendency exposure parameters (this step is conducted as part of the core individual risk characterization methodology completed for all receptor populations, not only those evaluated in the HWC exposure parameter variability analysis). This distribution was weighted by population and facility sampling frequency to represent the national distribution of risk in the total exposed population.

2. **Develop a hybrid exposure parameter variability distribution.** Develop the exposure parameter variability distributions that will be used in the variability analysis. Three distributions were required for the commercial beef farmer: beef ingestion rate, occupancy period, and age correction factor. Each of these distributions, which were developed using data from the 1997 *Exposure Factors Handbook* (U.S. EPA, 1997), were presented as lognormal distributions that have been normalized so that their medians equal 1.0. The normalization was conducted to simplify the variability analysis process (see below). Once the normalized distributions were generated, they were aggregated to form a single hybrid distribution that has a median of 1.0 and a GSD that reflects variance from all three contributing distributions. The development of the distributions is discussed in Section 6.3.2 and presented in greater detail in Appendix I.

3. **Use Monte Carlo simulation to generate the new cumulative risk incorporating exposure parameter variability.** Monte Carlo simulation was used to randomly select a sector-level risk value from the cumulative risk distribution (this random selection is biased to reflect the population weighting of sectors). Concurrently, Monte Carlo simulation was used to randomly select a value from the hybrid exposure parameter variability distribution described in Step 2 above. Note that, because (1) both cancer and noncancer risk calculations are linear with regard to dose estimation and (2) the hybrid distribution has a median of 1.0, the value that was randomly selected from the hybrid distribution can be used directly to adjust the sector-level risk value. The newly adjusted sector-level

risk value now reflects exposure parameter variability and can be viewed as representing a single individual within that sector rather than a central tendency value for that sector. When repeated many times, the resulting distribution of risk in a sector has the same mean value as in the core methodology (prior to consideration of interindividual variability). This adjusted sector-level risk value was placed in a pool along with other similarly generated values, which ultimately formed the new cumulative risk distribution for the adult commercial beef farmer, and the procedure was repeated. A total of 3,000 iterations were completed for this analysis to generate a cumulative risk distribution for a given pathway. This number of iterations was selected through a sensitivity analysis in which the Monte Carlo procedure was repeated for a representative sector using 1,000, 2,000, 3,000, 5,000, and 100,000 iterations (taken to represent an "ideal" sampling). Three thousand iterations was the minimum number of iterations needed to produce an estimate of the 95th percentile that was accurate to within 10 percent.

4. **Identify specific individual risk percentiles of interest from the newly generated cumulative risk distribution.** Individual risk percentiles of interest were obtained from the newly generated cumulative risk distribution. These individual risk percentiles now reflect not only interfacility and intersector variability for such factors as modeled media concentrations and population density, but also intrasector variability in exposure factors.

8.2.3.2 <u>Discrete Approximation Approach</u>. Implementation of the discrete approximation component of the HWC exposure parameter variability analysis involved the following computational steps (as with the description of the Monte Carlo simulation approach, the adult commercial beef farmer is presented for purposes of illustration):

1. **Generate a cumulative risk distribution based on central tendency exposure parameters.** This is the same initial distribution (i.e., central tendency exposure parameter-based) used for the Monte Carlo simulation approach.

2. **Develop a hybrid exposure parameter variability distribution.** This is the same step described under Step 2 of the Monte Carlo simulation approach (see Section 8.2.3.1).

3. **Develop discrete approximations of the risk distribution contained in each sector.** The central-tendency-based risk value generated for each sector, when combined with the hybrid exposure parameter variability distributions, could be used to generate a distribution of risk for each sector that reflects exposure parameter variability. A numerical approach was then used to generate 20 “subsector” risk values for each sector that would represent discrete approximations of the risk distribution generated for that sector. Each of the 20 subsector risk values generated for a given sector represents the mean risk for 5 percent of the adult commercial beef farmer population located in that sector. The net result is a correct estimate of the mean for the entire population in a sector.

4. **Export subsector data to SUDAAN for processing.** Subsector data for all of the sectors in a given combustor category (i.e., that developed in Step 3 above) were exported to SUDAAN where they were processed to generate specific individual risk percentiles. SUDAAN allows individual risk percentiles to be generated that include confidence intervals reflecting sampling error. The Monte Carlo simulation approach does not allow the quantification of sampling error.

From a decision-making standpoint, the results produced using the discrete approximation approach are preferred to those generated using Monte Carlo simulation because they not only reflect exposure parameter variability, but also include confidence intervals reflecting sampling error. Because the discrete approximation approach involving SUDAAN has not been used as frequently in regulatory risk analysis as have the Monte Carlo-based methods, it was decided that the variability analysis results obtained using the discrete approximation approach should be benchmarked against the Monte Carlo simulation-based results. The results produced using the discrete approximation approach and those generated using Monte Carlo simulation are in agreement to the first decimal place, which is an amount attributable to differences in rounding procedures used by the two software programs.

8.2.4 Lead (Blood Lead Level Analysis)

Human health risk characterization for lead uses an approach different from that used for other noncancer effects. Rather than comparing average daily doses to RfDs, lead risk characterization is based on a comparison between modeled PbB levels and the HBL established for lead, 10 µg/dL. This analysis was completed for the 0- to 5-yr-old age group for all modeled receptor populations. Site-specific media concentrations obtained from the Indirect Exposure Model were processed using the Integrated Exposure Uptake Biokinetic model to generate the PbB levels used in lead risk characterization. These PbB levels were generated at the sector level and were used to construct a cumulative risk distribution for use in characterizing individual risk.

Specifically, the sector-specific outputs from the IEUBK model were used to generate individual risk results presenting the PbB levels for the 50th, 75th, 90th, 95th , 97th, and 99th percentiles of the cumulative risk distribution for a given receptor population/combustor category combination. These percentiles reflect the emphasis placed on characterizing the upper tails of the risk distributions. With the exception of the subsistence receptors and the recreational fisher receptor, for which population data were not obtained at the sector level (recreational fishing population estimates were generated at the study area level), the individual lead risk estimates for all receptor populations reflect population weighting (i.e., the contribution of a given sector's individual risk estimate to the overall distribution of individual risk for a given combustor category is directly proportional to the number of individuals residing within that sector relative to the number of individuals residing within other sectors). This category of risk incorporates interindividual (i.e., differences in pharmacokinetics between individuals with regard to the lead metabolism) and intersector variability in modeled PbB levels.

The sector-level modeled PbB levels were aggregated across sectors to form a cumulative risk distribution for a given combustor category/receptor combination (following the methodology described below). Specific percentiles of interest were obtained from the cumulative risk

distribution and compared to the HBL established for lead. Three separate types of individual-level risk estimates were generated for lead: (1) incremental (reflecting only exposure to lead released from HWC facilities), (2) background (reflecting exposure to background lead levels), and (3) total (reflecting aggregate exposure to both background and facility-related lead).

The PbB levels at specific percentiles due to background exposure can be obtained by sampling the background variability distribution (median 3.6 µg/dL and GSD 1.6) to produce a sampling distribution from which the desired percentiles are identified. A total of 10,000 runs were conducted in producing the sampling distribution to ensure stability in the percentile estimates that were generated. This number of runs provides a 95 percent confidence level that the percentile obtained from the distribution generated using this procedure will lie between its two neighboring percentiles (e.g., that $x_{.49}$ and $x_{.51}$ represent a 95 percent confidence interval for $x_{.50}$). However, despite the level of stability achieved in identifying percentiles from this distribution, as discussed below, there is significant uncertainty associated with using this lognormal distribution to characterize background lead exposures for sites located across the nation.

The derivation of an aggregated PbB distribution for a given combustor category reflecting total lead exposure (i.e., background plus incremental) is less straightforward than deriving the distribution for background exposure. For total lead exposure, a different lognormal distribution was generated for each sector reflecting the range of incremental and background lead exposures experienced by individuals in that sector. The lognormal distribution for each sector was comprised of a mean background PbB level (3.6 µg/dL, see Section 6.6.3) added to a modeled incremental mean PbB level generated using IEUBK modeling (see Section 6.5.1). A GSD of 1.6, reflecting interindividual variability in PbB levels, was then applied to each aggregate mean to produce a lognormal distribution reflecting total lead exposure for each sector. These sector-level lognormal distributions characterizing total lead exposure were then aggregated across all study areas comprising a given combustor category to produce a single aggregated distribution reflecting total lead exposure for that combustor category. Specific percentiles of interest could then be identified using this aggregated PbB distribution. A numerical solution was used for aggregating the lognormal sector-level PbB distributions. Specifically, Monte Carlo simulation was used to sample PbB levels from the sector level total PbB distributions associated with a given combustor category. The Monte Carlo simulation was population-weighted at the sector-level. The steps involved in using Monte Carlo simulation to generate an aggregated PbB distribution reflecting total lead exposure are as follows (note: a separate simulation would be conducted for each receptor population/combustor category combination that is modeled—the example below uses commercial beef farmer children associated with cement kilns for purposes of illustration):

- **Generate sector-level mean PbB levels reflecting incremental lead exposure:** use the IEUBK model combined with modeled media concentrations (for ambient air, soil, and drinking water) and modeled dose estimates (for dietary items) to generate sector-level geometric mean PbB levels reflecting incremental lead exposure for commercial beef farmer children associated with cement kilns (see Section 6.5.1).

Rank the sectors according to PbB levels: Order the sectors associated with all modeled cement kilns according to the geometric mean incremental PbB levels generated for the commercial beef farmer children.

Determine the cumulative fraction of the total commercial beef farmer child population that is within each sector: First, integrate sampling weights into the analysis by multiplying the sector-level population value for the commercial beef farmer child by the appropriate facility sampling weight. This produces adjusted sector-level population values that now reflect facility sampling weights. Second, establish the fraction of the total population that is located within each sector. The fraction of the total population located in each sector is determined by dividing the commercial beef farmer child population in the sector by the total commercial beef farmer child population across all sectors within the cement kiln combustor category. Finally, determine the cumulative fraction of the total population within each sector by summing the population fractions for all the sectors that have blood level concentrations lower than the sector under consideration.

Randomly select a sector and associated incremental mean PbB level for processing: Now that the sectors for the cement kiln combustor category have been ranked according to mean incremental PbB level and the cumulative fraction of the total population residing within each sector has been determined, a specific sector can be selected for processing by randomly selecting a number between 0 and 1 and using that value to identify a specific cumulative fraction of total population value; that value will belong to a specific sector. The random value is generated using a uniform distribution with an interval of [0,1]. This approach to selecting sectors for processing ensures that selection is population-weighted to reflect the distribution of commercial beef farmer children across sectors constituting the cement kiln combustor category (e.g., a sector with twice the population of another sector, assuming facility sampling weights are identical, will have twice the probability of being selected for processing). The mean incremental PbB level for the selected sector is then processed as described below to reflect both background lead exposure and interindividual variability in PbB levels.

Adjust the sampled sector-level mean incremental PbB value to reflect both background lead exposure and interindividual variability in individual PbB levels: The incremental mean PbB level selected in the last step is now adjusted to reflect both background lead exposure as well as an element of interindividual PbB variability (i.e., the incremental mean value selected in the last step is adjusted to represent the total PbB level of a hypothetical individual from that sector). This is accomplished first by adding a mean background PbB level of 3.6 (see Section 6.6.3) to the modeled incremental mean PbB level for the randomly selected sector. This step produces a mean **total** PbB level for the sampled sector. Next, an interindividual variability adjustment factor is randomly selected from a lognormal distribution with a mean of 1.0 and a GSD of 1.6 (the GSD reflects interindividual variability in PbB levels). The sector-level total mean PbB level is then multiplied by this randomly selected interindividual variability adjustment factor to produce

an adjusted total PbB level. This new processed PbB value now represents the total PbB level for a randomly selected individual from that sector (i.e., the sector-level incremental mean PbB value selected in the last step has now been adjusted to represent a total PbB value selected from a lognormal distribution representing total PbB levels for individuals in that sector).

Conduct multiple iterations to generate the aggregated total PbB distribution for child commercial beef farmers in the vicinity of cement kilns: The steps described above are repeated 10,000 times to generate a stable aggregated individual risk distribution of total PbB levels for commercial beef farmer children in the vicinity of cement kilns. The following stability criteria were used in selecting this number of iterations: (1) repeated simulations of this total PbB analysis for the same receptor population/combustor category combination would generate identical 95th percentile total PbB levels out to the second decimal place (i.e., 0.00 µg/dL); and (2) repeated simulations for the same receptor population/combustor category combination would produce 99th percentile total PbB levels that are within 0.01 µg/dL of each other. These two stability criteria are considered sufficient given that the IEUBK model used to generate incremental PbB levels has model accuracy to the 0.00 µg/dL level.

Extract individual risk percentiles of interest from the aggregated total cumulative PbB distribution: 50th , 75th , 90th , 95th , 97th , and 99th percentile total PbB levels for the child of the commercial beef farmer receptor population are extracted from the cumulative total PbB distribution.

Generate incremental PbB levels for specific percentiles of the modeled receptor population: Incremental PbB levels for these same percentiles of the population (i.e., 50th , 75th , 90th , 95th , 97th , and 99th percentiles) are obtained by subtracting the total PbB level identified for a specific percentile by the background PbB level identified for that same percentile (the derivation of background PbB levels is discussed earlier in this section). This approach for generating incremental PbB levels for specific percentiles of the population does assume that background lead exposure and total lead exposure are perfectly correlated. In reality, it is probable that the two categories of lead exposure are not perfectly correlated (e.g., those individuals experiencing elevated background lead exposure levels may not be the same individuals residing in study areas with elevated incremental lead exposure levels). Consequently, the approach used in the HWC risk analysis for identifying incremental PbB levels for specific percentiles introduces uncertainty into those estimates, although the magnitude of that uncertainty has not been quantified.

The stepwise approach detailed above for generating background, incremental, and total PbB estimates for specific percentiles of a given receptor population was repeated for all modeled receptor populations, combustor categories, and MACT options (including baseline emissions levels) considered for the final rule.

It is important to note that the individual-level lead analysis detailed in this section is subject to a number of sources of uncertainty. The IEUBK model used to generate sector-level geometric mean incremental PbB levels is impacted by model uncertainty stemming from the approach used to model indoor dust exposure in addition to uncertainty related to pharmacokinetic modeling of PbB levels (see Section 6.5.1). Uncertainty is also associated with the approach used to characterize interindividual variability in background lead exposure. As discussed in Section 6.6.3, subsequent to completing PbB modeling for the HWC risk analysis, the CDC released the NHANES III report containing updated national level data on lead exposure in children (CDC, 1997). These data allow more complete characterization of background lead exposure in children, including the derivation of an interindividual variability distribution that accounts for both variability in lead uptake and site-to-site variation in background media concentrations. This GSD would be preferable to the 1.6 used in the current analysis for characterizing background variability; however, because these data were identified subsequent to developing and implementing the lead component of the HWC risk analysis, it was not possible to incorporate them.

8.2.5 Incremental Margin of Exposure for TCDD-TEQ

The HWC analysis includes incremental margin of exposure (MOE) results only for individual risk to assess the potential for noncancer health impacts resulting from exposure to modeled dioxin/furan congeners released from HWC facilities. This analysis used background exposures as the benchmark for comparison. As with the calculation of hazard quotients, incremental MOE estimates were generated by comparing modeled average daily dose for specific receptor populations at the sector level to exposures based on background body burden data. Sector-level incremental MOE estimates pooled across the sectors making up a combustor category form cumulative incremental MOE distributions from which specific percentiles of interest can be identified. These cumulative incremental MOE distributions are generated in essentially the same manner as cumulative risk distributions for either cancer or noncancer health effects in the core HWC risk analysis. The steps involved are as follows:

1. Rank sector-level incremental MOE estimates for a given receptor population/combustor category combination from lowest to highest estimate

2. Weight these sector-level estimates by facility sampling weight (i.e., the sector-level incremental MOE estimates are represented within the cumulative distribution with a frequency equaling the facility sampling weight)

3. If the receptor population is enumerated, then weight the sector-level incremental MOE estimates by the sector-level population counts identified for the receptor population (an aggregated weight is developed for each sector by multiplying the facility sampling weight for that sector by the population count for that sector)

4. Identify incremental MOE values for percentile of interest from the cumulative incremental MOE distribution that has been generated.

The potential for noncancer effects resulting from exposure to dioxin/furan congeners was assessed using incremental MOE estimates based on benchmarks derived from background

body burden data. Although this approach is essentially identical to the standard HQ approach used in assessing noncancer risk, because the incremental MOE approach compares modeled intake rates to background-based levels and not to an RfD derived from health effects studies, care must be taken in interpreting the results. The HWC analysis includes incremental MOE results only for **individuals**. All incremental MOE estimates generated for the HWC risk analysis were based on central tendency exposure factors—an exposure parameter variability analysis was not conducted for this risk descriptor. In addition to evaluating incremental MOE for all receptor populations and age groups considered in the HWC risk analysis, incremental MOE results were also provided for infants based on exposure to TCDD-TEQ though breast milk ingestion. The breast milk exposure scenario was assessed using incremental MOE for the infants of mothers from each receptor population considered in the HWC analysis (i.e., infants of 12- to 19-yr-old commercial beef farmers). For the breast milk scenario, both 12- to 19-yr-old and >19-yr-old age groups were considered to have the potential to nurse; therefore, the breast milk scenario was evaluated for both age groups.

8.2.5.1 <u>Incremental MOE Analysis for Non-Infant Receptors</u>. Incremental MOE results for noninfant receptor populations were generated by dividing the modeled average daily dose of TCDD-TEQ by a level of exposure reflective of background body burden data. Central tendency exposure parameters were used to generate incremental MOE estimates for all receptor populations (for a detailed description of exposure pathways considered for each receptor population and the exposure parameter values used in modeling each pathway, refer to Section 6.0). An exposure parameter variability analysis was not conducted for the TCDD-TEQ incremental MOE risk category.

The same background body-burden-based average daily dose for TCDD-TEQ (1.5 pg/kg-d) was used to derive incremental MOE estimates for all non-infant receptor populations evaluated in the HWC risk analysis. Pharmacokinetic modeling was used to derive this daily intake rate based on a background body burden value for TCDD-TEQ in human adipose tissue of 30 ppt. Specifically, the 30-ppt value was combined with a half-life for TCDD-TEQ in humans of 7 years to generate a central tendency intake rate of 110 pg/d that is reflective of background (see Section 6.6.1). When applied to a 70-kg adult, the 110-pg/d value translates into a daily intake rate of 1.5 pg/kg-d.

Although it would be preferable to derive separate daily intake rates for TCDD-TEQ background for each of the four age groups considered in the HWC risk analysis, no data were identified for developing age-group-specific values for the three younger age groups. Therefore, the 1.5-pg/kg-d value, which is based on a 70-kg adult, was used for all age groups. As discussed in Sections 6.6.1.1 and 6.6.1.2, the 1.5-pg/kg-d value may be somewhat high for adults but is probably fairly representative for children.

8.2.5.2 <u>Incremental MOE Analysis for Infants (Breast Milk Exposure)</u> Incremental. MOE estimates for infants exposed to TCDD-TEQ through breast milk ingestion were generated by: (1) estimating the modeled TCDD-TEQ concentration in maternal breast milk, (2) using the breast milk value to generate a daily dose for the infant reflecting breast milk intake, and (3) comparing this modeled average daily dose to a level that reflects background TCDD-TEQ exposures. Separate incremental MOE estimates were generated for infants of 12- to 19-yr-old

mothers and infants of >19-yr-old mothers (i.e., for the 12- to 19-yr-old age group and the >19-yr-old age group for each modeled receptor population). In generating these incremental MOE results, the sector-level incremental MOE estimates that form the basis for the cumulative distributions were weighted both by the facility sampling weight and by the appropriate population count. Specifically, for enumerated receptor populations, the number of 12- to 19-yr-olds or the number of >19-yr-olds from the receptor population of interest were used as the basis for weighting the sector-level incremental MOE values. Note that these population counts were not further adjusted on a study area level to reflect either (1) the actual number of mothers within each sector, or (2) the actual number of infants of breast-feeding age. The fact that population weighting for this risk results category uses total counts for the 12- to 19-yr-olds or >19-yr-olds for a specific receptor population and does not further adjust those values to reflect either the number of mothers or number of breast-feeding infants does introduce some uncertainty into the incremental MOE results generated for enumerated receptor populations. This uncertainty results from the fact that this approach does not account for geographical differences in birth rates, but instead assumes that all modeled study areas have the same birth rates for both 12- to 19-yr-olds and >19-yr-olds.

The modeled average daily dose for TCDD-TEQ generated for a given receptor population (i.e., infants of commercial beef farmers) was compared to a background level of 50 pg/kg-d for breast milk exposures. The background value of 50 pg/kg-d is based on a measured U.S. body burden level of 16 ppt in the lipid portion of maternal breast milk (U.S. EPA, 1994).

8.3 Population Risk Analyses

Local population risk estimates were derived to characterize the incidence of specific health effects for receptor populations residing in HWC study areas. These estimates were generated by combining sector-level individual risk estimates (i.e., the same individual risk estimates used in creating the cumulative risk distributions) with sector-level population estimates. For cancer, the sector-level individual risk estimates were combined with corresponding sector-level population totals for a given receptor population to generate sector-level statistical excess cancer incidence estimates.

Local population risk estimates generated for the recreational fisher (specifically for the ingestion of fish containing methylmercury) were based on fishing activity distributed between modeled waterbodies and not on sector-level risk estimates. Only population estimates of the number of recreational fishers **located within study areas** could be generated (compared with the sector-level estimates generated for other receptor populations). Consequently, in characterizing local population risk for the recreational fisher, a new approach was developed that (1) identifies those “at-risk” facilities within a given combustor category that could potentially pose risk to recreational fishers at or above levels of concern (i.e., an individual risk HQ of 1.0), and (2) provides an estimate of the number of recreational fishers who live within the study areas associated with those at-risk facilities. This scenario introduces uncertainty into the risk estimates because recreational fishing activity could also involve activity at nonmodeled waterbodies that could be impacted by HWC emissions to a greater or lesser extent than the modeled waterbodies.

The risk descriptors presented in Table 8-3 are discussed in detail in the following sections.

Table 8-3. Risk Descriptors Used in Population Risk Category

Risk Descriptor	Local Population Risk	Agricultural Commodity Risk
Annual excess cancer incidence	Statistical excess cancer incidence for specific receptor populations	Statistical excess cancer incidence for the national population from consumption of agricultural commodities raised within the HWC study areas
Number of exceedances (noncancer effects)	Number of individuals above health benchmarks (i.e., HQ = 1.0)	Not applicable
Excess exceedances (lead)	Excess number of children with blood lead levels above 10 µg/dL	Not applicable
Avoided incidence (particulate matter)	Avoided incidence rates for specific respiratory and cardiovascular effects	Not applicable

8.3.1 Carcinogenic Risk

Local population cancer risk was characterized using excess cancer incidence estimates (i.e., statistical excess cancer cases) for a specific receptor population. *National population cancer risk* was characterized by generating estimates of the excess cancer incidence for the national population resulting from the ingestion of agricultural commodities that are grown within HWC study areas and therefore contain dioxin released from HWC facilities. Local statistical excess cancer incidence estimates were generated for those receptor populations located within study areas that were exposed to modeled carcinogens through contact with environmental media or home-produced food commodities that contain carcinogens released from the specific HWC facility located within a given study area. Given the need for sector-level population estimates in generating local population risk estimates, these estimates are generated only for enumerated receptor populations: residents, home gardeners, commercial beef farmers, commercial dairy farmers, commercial pork farmers, and commercial produce farmers.

8.3.1.1 <u>Local Cancer Incidence</u>. Sector-level individual cancer risk estimates were combined with sector-level population estimates to generate sector-level excess cancer incidence estimates. The specific steps associated with the generation of local population cancer risk estimates are outlined below:

1. **Generate sector-level individual risk estimates.** See Step 1 in Section 8.2.1. These sector level risk estimates were derived using mean exposure parameters and, as such, represent mean risks within a sector.

2. **Obtain sector-level population estimates for each of the receptor populations evaluated.** See Section 4.4.

3. **Generate sector-level local population risk estimates with sample facility weighting.** Sector-level individual risk estimates generated in Step 1 are

multiplied by the corresponding sector-level population estimates generated in Step 2 to produce sector-level local population risk estimates for the receptor population/constituent combination being considered. Each of the sector-level local population cancer risk estimates is also facility-sample-weighted by multiplying each sector-level risk estimate by the facility sampling weight for the corresponding HWC facility.

4. **Generate an aggregate local population risk estimate for the combustor category being considered.** Each of the local population cancer risk estimates for the sectors making up a given combustor category are aggregated to generate a local population cancer risk estimate for that combustor category (each of these aggregated estimates is specific to a constituent/receptor population combination).

5. **Generate annualized local population risk estimates.** The lifetime estimates generated in steps 1-4 above reflect the modeling of risk resulting from the entire period of exposure to HWC emissions (i.e., the period of time that a receptor resides within the HWC study area). Annual estimates, which reflect the risk resulting from a single year of exposure to HWC emissions (i.e., a year of residence time within the HWC study area), were generated by dividing the lifetime estimate by the duration of exposure.

In addition, the annualized local population excess cancer risk estimates were summed across constituents (assuming additivity of carcinogenic response) and receptor populations within a combustor category to generate a **total** excess cancer incidence for each category of combustors.

8.3.1.2 <u>Agricultural Commodity Risk</u>. National population risk was evaluated for a specific category of cancer risk: excess incidence estimates for the national population resulting from the ingestion of dioxin contained in agricultural food commodities that were raised within HWC study areas but distributed nationally. These estimates (termed agricultural commodity risk estimates) were derived for key risk-driving commodities including commercially raised beef, pork, and milk. Agricultural commodity risk estimates were based on sector-level projections of (1) the amount of dioxin contained in agricultural commodities and (2) the number/amount of the specific agricultural commodity produced during a model year. These two estimates were combined at the sector level to generate sector-level estimates of the amount of dioxin contained in agricultural commodities raised during a model year (i.e., the amount of "diet-accessible dioxin" produced within each sector). These sector values were then facility-sample-weighted and aggregated across a combustor category to derive an aggregate diet-accessible dioxin estimate for a particular combustor category. That value can then be used in a national agricultural commodity risk equation (Section 8.3.1.2) to derive excess cancer incidence estimate for that combustor category/agricultural commodity combination. These agricultural commodity excess cancer incidence estimates reflect an incidence rate for the national population and not just for those individuals residing within the vicinity of HWC facilities, as is the case with local population statistical excess cancer incidence estimates. There is potential overlap between the local population statistical excess cancer incidence estimates and the national population statistical cancer incidence estimates with respect to dioxin-related risks. Therefore, care must be taken in interpreting these two sets of results.

Risks to the general population were assessed by estimating excess cancer incidence that results within the general population from the ingestion of agricultural commodities raised within HWC study areas that contain dioxin-TEQ released from HWC facilities.

The methodology used to project population risk resulting from ingestion of each of the agricultural commodities of concern in the HWC analysis involves the same basic steps (beef cattle are used as an example of an agricultural commodity):

1. **Model the dioxin concentration in beef cattle.** Project dioxin concentrations in commercially raised beef cattle for each sector. Dioxin may bioaccumulate in beef through direct contact with media containing dioxin (ingestion of soil) and through ingestion of plants containing dioxin (see Section 5.4.1.3).

2. **Determine the number of beef cattle slaughtered within each of the 16 sectors of a given study area.** County-level data are used to determine the number of beef cattle slaughtered in each of the 16 sectors within each study area. This estimation involves two steps:

   # **Determine the number of beef cattle farms in each sector.** The number of beef cattle farms relative to the number of total farms is determined at the county level. The fraction of total farms that are beef cattle farms (based on county-level data) can be used to adjust U.S. Census block level data on the number of total farms to represent the number of beef cattle farms in each sector (see Section 4.4).

   # **Determine the number of beef cattle slaughtered in each sector.** The number of slaughtered beef cattle per beef cattle farm is determined at the county level. The county-level data on the number of beef cattle slaughtered per beef cattle farm can then be used (along with the number of projected beef cattle farms per sector) to project the number of beef cattle slaughtered in each sector per year.

3. **Adjust the sector-level agricultural commodity estimates using facility sampling weights.** Facility sampling weights are incorporated into the agricultural commodity risk methodology by using them to adjust the sector-level agricultural commodity estimates. Specifically, in this example, the sector-level beef cattle estimates of steer slaughtered associated with a given study area were multiplied by the sampling weight developed for the HWC facility located in that study area.

4. **Determine the annual amount of diet-accessible dioxin (in beef) produced per year.** Data on the average weight of a beef cow/steer (obtained from the Census of Agriculture summary tables) are combined with data on the percentage of a given beef cow/steer that is available for human consumption to determine a representative value for the amount of diet-accessible beef resulting from each slaughtered beef cow/steer. These data are combined with the estimated number of beef cattle slaughtered per sector to project that amount of diet-accessible beef

produced per sector. Modeled dioxin concentrations in beef cattle can then be combined with the projected amount of diet-accessible beef produced per sector to determine the amount of diet-accessible dioxin (contained within beef) per sector. Projected amounts of diet-accessible dioxin at the sector level can be summed across a given study area to determine the amount of diet-accessible dioxin associated with a given study area. Table 8-4 presents the data required to adjust sector-level agriculture production estimates into diet-accessible dioxin estimates.

Table 8-4. Data Used to Adjust Sector-Level Agricultural Production Estimates into Diet-Accessible Dioxin Estimates

Parameter	Value (units)	Reference
Average steer weight	1,256 lb	USDA, 1996
Fraction of total beef cattle weight available for human consumption	53%	Personal communication - Matthew Claeys, Extension Livestock Specialist, North Carolina State University (June 1997)
Typical amount of milk produced per dairy cow	15,704 lb/cow-yr	USDA, 1996
Average hog weight	255 lb	USDA, 1996
Fraction of total hog weight available for human consumption	56%	NPPC, 1999

5. **Repeat the above calculations for each study area and use these data to generate an aggregate diet-accessible dioxin estimate for a given combustor category.** Repeat the series of calculations described above for each of the study areas within a given combustor category. Once diet-accessible dioxin values have been estimated for each of the study areas within a given combustor category, sum these values to obtain an aggregate diet-accessible dioxin value for that combustor category. This is shown in Equation 8-7.[6]

6. **Generate an agricultural commodity annual excess cancer incidence estimate for the combustor category of interest.** Using Equation 8-6 together with the aggregated diet-accessible dioxin estimate from Step 8 above, generate an agricultural commodity annual excess cancer incidence estimate for the combustor category of interest.

The approach outlined above for projecting national population risk resulting from the ingestion of beef containing dioxin can, with minor modification, be applied to pork and milk.

[6] Estimates of the sector-level number of steer slaughtered were adjusted in SUDAAN to reflect facility weights.

$$Total\ Diet-Accessable\ Dioxin-TEQ = \sum_{i=1}^{n} Ns_i \cdot B \cdot Cd_i \qquad (8\text{-}7)$$

Parameter	Definition
Ns_i	Number of steer slaughtered in i^{th} sector (unitless)
B	Beef consumed (average weight of beef cattle * percent edible) (kg)
Cd_i	Dioxin concentration in i^{th} sector (mg TCDD-TEQ/kg)

8.3.2 Noncancer Risk

Local population noncancer risk was characterized for three categories of health effects including (1) neurological effects in adults resulting from the consumption of recreationally caught fish containing methylmercury; (2) developmental effects in children 0-5 years of age following exposure to lead released from HWC facilities (with consideration for simultaneous background lead exposure); and (3) adverse respiratory effects resulting from exposure to $PM_{2.5}$ and PM_{10}. The local population noncancer risk estimates for PM and lead are based on sector-level population totals for the receptor population and cohort of interest. Analysis of recreational fisher risk is based on facility-level recreational fisher totals. Facility sampling weights were applied in generating all three types of local population noncancer risk estimates. As with the population cancer risk estimates, all three of the population noncancer risk estimates are presented as annualized estimates.

Recreational Fisher. Risk for the recreational fisher resulting from the ingestion of self-caught fish containing methylmercury is closely related to the level of fishing activity at specific waterbodies. Consequently, in order to generate quantitative population-level risk estimates for this receptor population, it would be necessary to characterize the level of recreational fishing activity at specific waterbodies including the number of recreational fishers frequenting each waterbody and the number of fishing trips made by each fisher over a typical year. It was, however, not possible to characterize recreational fisher activity at specific waterbodies with this level of detail. Consequently, qualitative statements concerning the number of recreational fishers associated with “at-risk” facilities were generated in place of quantitative local population risk estimates for the methylmercury fish ingestion pathway.

At-risk facilities are defined as those facilities where 5 percent or more of the adult recreational fisher population is estimated to have a methylmercury fish ingestion risk level above the HBL for methylmercury (i.e., HG $\geq$1.0). This criterion is evaluated by examining the 95th percentile of the cumulative HQ distribution generated for each study area for methylmercury ingestion. If the 95th percentile value is equal to or exceeds 1.0, then the facility is classified as at-risk. It is important to note that recreational fisher risk is modeled for the final rule assuming that all recreational fishers within a given study area distribute their fishing activity among all modeled waterbodies in the study and in proportion to the waterbody surface area. The only source of

variability in modeled HQ values for the recreational fisher is exposure parameter variability in fish ingestion rates.[7] Therefore, the 95th percentile risk values used to determine at-risk status for HWC facilities, assume that fishing takes place exclusively at modeled waterbodies and fish consumption rates are at the 95th percentile of recreationally caught freshwater fish ingestion rates (see Section 6.3).

The qualitative population risk statements generated using this approach identify the total number of recreational fishers residing within the study areas associated with the at-risk facilities. The number of facility sampling weighted recreational fishers associated with at-risk facilities represents the number of recreational fishers who may spend some fraction of their fishing activity at waterbodies with a potential hazard for methylmercury.

A second, more refined set of qualitative population-level risk statements is also generated for the recreational fisher. Specifically, the percentage of recreational fishers exceeding the HBL for methylmercury exposure at at-risk facilities is estimated. This second set of qualitative population-level risk statements is produced by determining the number of recreational fishers at each at-risk facility with modeled HQs above the methylmercury HBL (i.e., HG $\geq$1.0). These exceedance estimates are then summed across all at-risk facilities for a given combustor category and divided by the total number of recreational fishers associated with those at-risk facilities to generate the desired percentage. As with the initial estimate of the number of recreational fishers associated with at-risk facilities, this percentage estimate also assumes that recreational fishers fish exclusively at modeled waterbodies and that all recreational fishers within a given study area display identical behavior regarding fishing activity, with the exception of fish ingestion rates, which are assumed to vary.

Recreational fisher population estimates used in this calculation are generated using 1991 National Survey of Fishing, Hunting, and Wildlife data (U.S. DOI, 1993). The National Survey data provides county-level estimates on the percentage of the adult population that engages in recreational fishing activity. These percentages are used to generate study-level population estimates for the adult recreational fisher (see Section 4.4.1.2).[8] These estimates form the basis for the qualitative population risk statements generated for the recreational fisher.

Because it is difficult to predict the level of recreational fishing activity at waterbodies located within heavily urbanized areas, those study areas classified as predominantly urban (i.e., over 50 percent of the census block group surface area for the study area has an urban density

[7] A single surface-area averaged methylmercury fish tissue concentration is generated for each study area, reflecting the assumption of identical fishing activity for each recreational fisher (i.e., activity distributed between modeled waterbodies based on surface area). This averaged fish tissue concentration is used to generate a mean recreational fisher HQ estimate for a specific study area and consequently, all recreational fishers within a given study area have the same mean methylmercury fish ingestion risk level. Exposure parameter variability in fish ingestion rates is used to generate a range of risk levels based on this mean fish ingestion risk for each study area. However, it is important to note that this range of risk levels reflects interindividual differences in ingestion rates and not recreational fishing behavior, which is assumed to be the same for all individuals within a given study area (see Section 8.2.3).

[8] Because the National Survey data provides percentages for adults, the population-level risk analysis completed for the recreational fisher focuses exclusively on the adult recreational fisher.

using U.S. Census criteria for urban land-use)[9] were excluded from the recreational fisher population-level risk analysis.[10]

The analytical steps completed to generate both types of qualitative population risk statements for the recreational fisher (i.e., number of recreational fishers associated with at-risk facilities and the percentage of recreational fishers projected to have risk levels exceeding the health benchmark of 1.0) are outlined below:

Generate a mean exposure parameter-based recreational fisher methylmercury HQ for each study area. A single surface-area averaged methylmercury fish tissue concentration is generated for each study area, reflecting the assumption of identical fishing activity for each recreational fisher (i.e., activity distributed between modeled waterbodies based on surface area). This averaged fish tissue concentration is combined with mean exposure parameters to generate a mean recreational fisher HQ estimate for each study area.

Generate a cumulative risk distribution for the recreational fisher for each study area. The mean methylmercury fish ingestion HQ value generated for each study area is combined with an exposure parameter variability distribution for recreational fish ingestion to produce a cumulative risk distribution for this pathway. These cumulative risk distributions are generated using a post-processing approach similar to that used in conducting the exposure parameter variability analysis for the recreational fisher (see Section 8.2.3.1). Specifically, the exposure parameter variability distribution for fish ingestion is sampled to produce a set of ingestion rates representing a group of hypothetical individuals from that study area. Each of these sampled ingestion rates is then converted to an individual methylmercury HQ estimate (i.e., each ingestion rate is divided by the mean ingestion rate for the adult recreational fisher and then multiplied by the mean methylmercury HQ value for that study area). This produces a set of risk levels for the group of hypothetical recreational fishers that reflects variance in fish ingestion rates among those individuals. This set of hypothetical risk levels forms the cumulative risk distribution for that study area. This procedure is completed for each modeled study area.

[9] The U.S. Census identifies those areas with a population density of 1,000 individuals/mile^2 or more as urban. Conversely, those areas with less than 1,000 individuals/mile^2 are classified as rural.

[10] Significant uncertainty is associated with projecting recreational fishing activity for individuals residing in heavily urbanized areas (i.e., cities). Applying county-level recreational fishing percentages obtained from National Survey data to urbanized areas often produces a large recreational fisher population estimate. If it is assumed that all of the recreational fishers fish at modeled waterbodies located near them, then fishing pressures on these waterbodies can be extremely high and potentially unrealistic (it is reasonable, therefore, to assume that many urban recreational fishers travel some distance away from the city to fish). Because there is significant uncertainty associated with characterizing recreational fishing activity in heavily urbanized areas, these areas are excluded from the qualitative population-level risk analysis completed for the recreational fisher (these areas are, however, included in the individual-risk analysis completed for the recreational fisher).

Identify at-risk facilities. Query the cumulative risk distributions generated for each study area to identify the 95th percentile HQ. If this HQ is equal to or greater than the health benchmark of 1.0, then the facility associated with that study area is classified as at-risk. This procedure is repeated for all modeled facilities in a given combustor category.

Identify the number of recreational fishers associated with at-risk facilities. National Survey data are used to generate recreational fisher population estimates for each modeled study area (see Section 4.4.1.2). Facility sampling weights are incorporated into these estimates of recreational fishers associated with modeled study areas—the facility sampling weights are multiplied by the study area-level population estimates that are generated. The recreational fisher population estimates for all study areas associated with at-risk facilities within the combustor category of concern are summed to produce an estimate of the number of recreational fishers associated with at-risk facilities. This procedure is repeated for all combustor categories considered in the analysis.

For at-risk facilities, identify the percentage of recreational fishers with HQs greater than 1.0. For all at-risk facilities, the cumulative risk distributions generated in the first step described above are queried again to identify the percentage of the recreational fishers with HQs greater than or equal to the health benchmark of 1.0 (given the criterion for identifying at-risk facilities, this value will be at least 5 percent of the recreational fisher population for each study area). For each at-risk facility, this percentage is then multiplied by the number of recreational fishers associated with that facility to produce an estimate of the number of recreational fishers with modeled HQs above the health benchmark. This procedure is completed for all at-risk facilities within a given combustor category, and the resulting values are summed to produce a single estimate of the number of recreational fishers with HQs greater than or equal to the health benchmark. This value is then divided by the total number of recreational fishers identified for those at-risk facilities to produce an estimate of the percentage of recreational fishers associated with at-risk facilities (for that combustor category) with modeled fish ingestion HQs greater than or equal to 1.0.

It is important that the limitations of these semiquantitative population risk projections be clearly stated—the population numbers that are generated do not represent quantitative estimates of the numbers of individuals who are either above a given threshold of concern or associated with the recreational fishing scenarios modeled for the HWC facilities (i.e., engaged in fishing activity exclusively at the modeled waterbodies). These population numbers represent the number of recreational fishers whose recreational fishing activity might include some activity at modeled waterbodies with fish ingestion risks at or above levels of concern. It is possible that these individuals engage in fishing activity that involves nonmodeled waterbodies, thereby resulting in risk values that are different from those generated assuming activity only at modeled waterbodies.

8.3.3 Blood Lead

The objective of the population-level PbB analysis is to estimate the "excess" incidence of elevated blood lead levels (above 10 µg/dL) above the background incidence rate for children (0 to 5 yrs old) for each of the modeled receptor populations. These incremental exceedance estimates are generated by: (a) estimating the number of children that exceed the lead HBL because of background lead exposure alone; (b) estimating the number of children that exceed the HBL because of total lead exposure; and (c) subtracting the background exceedance estimate from the total exceedance estimate to produce an estimate of the number exceeding that occur because of incremental exposure. Facility sampling weights are used in deriving population risk results for lead. The analytical steps involved in generating this category of quantitative population-level risk results are outlined below:

- **Generate cumulative PbB distributions reflecting total lead exposure at the sector level.** The sector-level PbB distributions reflecting total lead exposure that form the basis for the individual-level lead risk analysis also form the basis for the population-level lead risk analysis. The approach used to generate these total lead PbB distributions, which is described in detail in Section 8.2.4, is briefly summarized here. Site-specific media concentrations and dietary dose estimates obtained from the Indirect Exposure Model were processed using the IEUBK model to generate sector-specific modeled geometric mean incremental PbB estimates (i.e., PbB levels resulting from exposure to lead released from the local HWC facility). A background mean exposure level of 3.6 µg/dL was then added to each sector-specific incremental mean PbB estimate to produce a mean total PbB level (i.e., an estimate of the mean PbB level reflecting both incremental and background lead exposure). Each sector-level total mean PbB estimate was then combined with a GSD of 1.6, which reflects interindividual variability in PbB levels, to produce a lognormal distribution of total PbB levels for each sector.

- **Establish cumulative PbB distribution reflecting background lead exposure.** Background lead exposure is characterized using a lognormal distribution reflecting interindividual variability in background lead exposure levels. This lognormal distribution has a mean of 3.6 µg/dL and a GSD of 1.6 (see Section 6.6.3 for an expanded discussion of the background PbB distribution). Because background exposure is assumed to be the same across all modeled study areas, the lognormal distribution described here is used to characterize background exposure for all receptors and locations, although incremental lead exposure is modeled separately for each receptor/location combination. Failure to reflect differences in background lead exposure related to location and socioeconomic status (which often are related) introduces uncertainty into the lead analysis.

- **Estimate the number of individuals within each sector exceeding the lead HBL because of background and total lead exposure.** The cumulative PbB distributions characterizing total lead exposure for each sector and modeled receptor population are queried to identify the percentages of the population within that sector that exceed the lead HBL because of total lead exposure. This percentage is then multiplied by the sector-level population for that receptor to

generate an estimate of the number of children exceeding the lead HBL because of total lead exposure for that sector (i.e., the number of total exceedances). This procedure is then repeated for background exposure. Specifically, the cumulative PbB distribution for background lead exposure is queried to identify the percentage of individuals exceeding the HBL for lead because of background exposure (note that, because background exposure is assumed to be the same across all modeled study areas, this percentage will be the same for all sectors). The estimate of the number of children exceeding the lead HBL because of background lead exposure is then multiplied by the number of children (from that receptor population) located in that sector to produce an estimate of the number of children exceeding the lead HBL because of background exposure (i.e., the number of background exceedances). In order to identifying the percentage of a sector's population that exceeds the lead HBL because of background and total lead exposure, sector-level background and total PbB distributions (which are lognormal distributions) are transformed into standardized normal distributions of Z, as shown in Equation 8-8:

$$Z = \frac{\ln\left(\dfrac{\textit{blood lead level of concern}}{\textit{model predicted blood lead} + \textit{background}}\right)}{\ln(GSD)} . \qquad (8\text{-}8)$$

Once a Z value is calculated for the HBL, a standard normal distribution table can be used to obtain the associate probability, which represents the risk of exceeding the HBL.

Subtract background exceedances from total exceedances to estimate incremental exceedances of the lead HBL. Sector-level background exceedances (step 2) are subtracted from sector level total exceedances (step 1) to produce sector-level estimates of incremental exceedances. These calculations are completed for each sector, and then the resulting values are summed across sectors making up a given combustor category to produce the overall incremental exceedance estimate for that combustor category.

To estimate the excess **annual** incremental exceedances for a given combustor category, the total incremental exceedance estimate is multiplied by the rate of turnover (yr^{-1}) in the exposed population. The turnover rate is estimated for a given receptor population as the reciprocal of the number of years in an age cohort (e.g., 5 years for the 0- to 5-yr-old cohort) since this represents the maximum exposure duration for that receptor population (the PbB analysis assumes 60 months of exposure in calculating modeled PbB levels—see Section 6.5.1).

The population-level PbB analysis detailed here was completed for all combustor categories and receptor scenarios except subsistence and recreational fishers. The reason for this is that sector-level population data are not available for the subsistence receptors and the recreational fisher receptor population risk estimates for lead were not generated for these receptors.

As with the individual-level PbB analysis, the population-level analysis is impacted by a number of sources of uncertainty. The IEUBK model used to generate sector-level geometric mean incremental PbB levels is impacted by model uncertainty stemming from the approach used to model indoor dust exposure in addition to uncertainty related to pharmacokinetic modeling of PbB levels (see Section 6.5.1). Uncertainty is also associated with the approach used to characterize interindividual variability in background lead exposure. As discussed in Section 6.6.3, after completing PbB modeling for the HWC risk analysis, CDC released the NHANES III report containing updated national-level data on lead exposure in children (CDC, 1997). This data allows more complete characterization of background lead exposure in children including the derivation of an interindividual variability distribution that accounts for both variability in lead uptake and site-to-site variation in background media concentrations (this GSD would be preferable to the 1.6 used in the current analysis for characterizing background variability). However, because this data was identified after developing and implementing the lead component of the HWC risk analysis, it was not possible to incorporate these data.

8.3.4 Particulate Matter

Only local population risk estimates were generated for PM. Transport of PM (primary or secondary) beyond modeled study areas was not assessed. The methodology used to evaluate risk resulting from inhalation exposure to PM combines sector-level modeling results for PM with concentration response functions derived from epidemiological studies to project incidence rates for specific health endpoints related to PM exposure. Avoided incidence rates were generated based on the difference in PM levels between baseline levels and levels projected to occur with the MACT standards. Facility sampling weights were incorporated into the analysis to adjust sector-level population data used in the PM analysis.

This section provides an overview of the PM risk analysis conducted for the final rule to determine the potential human health benefits associated with reductions in PM concentrations as a result of MACT emissions control standards for HWC facilities. For a more detailed treatment of the topics discussed in this section, please refer to Appendix E.

The HWC risk analysis completed for the final rule used a methodology for evaluating human health benefits resulting from reductions in ambient PM concentrations (both PM_{10} and $PM_{2.5}$) that is based on concentration response functions derived from epidemiological data. These concentration response functions relate sector-level reductions in modeled PM air concentrations to sector-level reductions in the incidence of specific health endpoints (e.g., mortality and hospital admissions for specific respiratory ailments). Although the concentration response functions used in HWC PM analysis all use air concentrations as the exposure metric, the specific exposure metric (e.g., PM_{10} vs. $PM_{2.5}$ and daily average vs. annual average concentrations) is different for different concentration response functions derived from different epidemiological studies.

The HWC PM analysis was conducted using the Criteria Air Pollutant Modeling System (CAPMS), which has been the primary analytical tool used for evaluating the health benefits attributable to both the Clean Air Act and the proposed alternatives to the current PM and ozone National Ambient Air Quality Standards (NAAQS) (see Appendix E).

8.3.4.1 Concentration Response Functions. Two primary functional forms are used in the concentration response functions that form the basis for the HWC PM analysis: log-linear and linear. The specific functional form used reflects the underlying response data contained in the epidemiological study used in deriving the particular concentration response function.

In several instances, more than one epidemiological study was identified for characterizing a particular category of PM-related health effects (i.e., several studies had examined a specific category of health effects involving similar subpopulations, PM exposure patterns, and health endpoints). When this occurred, the data from the different studies were pooled and used to develop a central tendency concentration response function that reflected findings from all of the studies.

8.3.4.2 Sector-Level Modeled PM Concentration Data. Each of the concentration response functions used in the HWC PM analysis generates health effects incidence estimates for a specific temporal pattern of PM exposure (e.g., short-term exposure represented by 2- to 3-day median concentrations for $PM_{2.5}$ and chronic exposure characterized by 1-yr median concentrations for PM_{10}). The air modeling completed for the HWC risk analysis generated 24-h average PM concentrations ($PM_{2.5}$ and PM_{10}) for 5 model years at the sector level. This 24-h average air modeling data set allows a wide variety of different temporal PM concentration estimates to be generated to match the requirements of specific concentration response functions.

The health endpoints evaluated in the PM analysis vary by time period of exposure. Section 7 and Appendix E present greater details on health endpoints. For example, mortality was evaluated using three different endpoints. One (Pope et al., 1995) was a long-term study using $PM_{2.5}$ exposures. Another mortality study used in the PM analysis (Schwartz et al., 1996) was a short-term study using $PM_{2.5}$. A third was a pooled analysis using 10 different studies. Estimates of annual avoided mortality incidence are calculated by the CAPSM computer model for the Pope study based on change in annual median $PM_{2.5}$ concentration. In contrast, CAPMS calculates mortality incidence estimates for the Schwartz study based on the change in 20 separate daily average $PM_{2.5}$ concentration over a year. In a similar manner, CAPMS calculates mortality incidence estimates for the pooled analysis based on the change in 20 separate daily average PM_{10} concentrations over the year. Because the studies will yield different estimates of avoided incidence, three separate estimates are generated and reported.

8.3.4.3 PM Receptor Populations. Because the health endpoints evaluated in the PM analysis vary by receptor population, use of the concentration-response functions in the PM analysis requires an estimate of the size of specific subpopulations. For example, the Schwartz (1995, 1996) studies of hospital admissions for all respiratory symptoms examined respiratory hospital admissions for people age 65 and older. Therefore, in order to estimate the change in incidence of respiratory hospital admissions predicted by the Schwartz (1995,1996) studies for a given change in air quality, it is necessary to have an estimate of the number of persons age 65 and older that are exposed to that air quality change. Details on the method used to generate baseline and subpopulation estimates are provided in Appendix E.

8.3.4.4 Baseline Incidence Data. The log-linear concentration response functions used in the HWC PM analysis required sector-level baseline incidence estimates for the health endpoints being evaluated (i.e., number of cases occurring in the absence of MACT standard

implementation). These baseline incidence values were used to project benefits to human health associated with MACT standard implementation. A variety of different sources, generally providing incidence information at the county level, were used to generate the sector-level baseline incidence values for a given location (e.g., county level mortality rates were obtained from the National Center for Health Statistics).

8.3.4.5 Background PM Concentrations. PM concentrations modeled for HWC facilities represent only a portion of total ambient PM concentrations in a given sector. The remainder is contributed by naturally occurring background levels and other anthropogenic sources. In evaluating avoided incidence resulting from reductions in HWC PM emissions, it is necessary to first establish the total aggregate baseline PM levels in each sector including all three sources (i.e., HWC facility, natural background, and other anthropogenic sources). However, the task of characterizing anthropogenic sources of PM for each modeled study area was beyond the scope of this analysis.

Failure to include anthropogenic PM source contributions specific to each modeled study area (other than the HWC facility being modeled) does introduce uncertainty into the PM risk assessment. HWC facilities can be located in relatively urbanized/industrialized areas where PM concentrations contributed by other sources could be significant, resulting in baseline PM levels above threshold levels established for certain PM health effects.

Because baseline PM levels were generated without consideration for the contribution of anthropogenic sources other then HWC facilities, these levels could significantly underestimate actual PM levels especially in heavily urbanized/industrialized study areas. Because of the potential to underestimate PM risks in areas that have ambient PM above threshold levels, the avoided incidence estimates were made assuming no threshold. Three health endpoints were effected by the no-threshold assumption because these, in fact, do report a lowest observed pollution level. The studies reporting a lowest observed pollution level are Pope et al., 1995 (mortality, long-term), Dockery et al., 1989 (acute bronchitis), and Ostro et al., 1995 (shortness of breath, days). By excluding consideration of the threshold effects levels (TELs), the avoidance incidence estimates remove the potential for underestimating risk through failure to consider anthropogenic sources of PM other than the HWC facilities (although there is the potential that these avoided incidence estimates could be conservative because they include no consideration of TELs).

8.3.4.6 Aggregated PM Risk Reductions. The incidence reductions projected for specific categories of health effects as a result of reductions in PM concentrations can overlap, resulting in the double counting of health benefits. Therefore, efforts were made to avoid double counting of benefits by identifying the specific types of health effects covered by each concentration response function and looking for areas of overlap. For example, potential overlap was identified in the results generated by PM benefits calculations based on short-term and long-term mortality studies. The short-term studies characterize mortality rates resulting from short-term temporal fluctuations in PM levels, while long-term studies characterize mortality rates associated with longer-term trends in PM levels. Calculations based on long-term study data are believed to capture more of the mortality incidence linked to PM concentrations than are calculations based on short-term studies, although there can be overlap between the two types of

mortality estimates. Therefore, it was decided to report a range of estimates using overlapping concentration-response functions because these reflect actual differences in the underlying studies.

8.4 References

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9.0 Ecological Risk Assessment Methodology

This section describes the methodology used to conduct a screening ecological risk assessment (SERA) for constituents released from hazardous waste combustors for the final rule risk assessment. The underlying technical framework for the SERA was developed by RTI in support of OSW for the Hazardous Waste Identification Rule proposed in 1995 (HWIR95). Since 1993, OSW has been actively involved in developing a methodology to evaluate ecological risks posed by chemical stressors found in solid wastes. The HWIR95 methodology was the first step in that endeavor and has continued to evolve in its application to the HWC risk assessment and other OSW risk assessments, notably, the ongoing development of an integrated risk modeling system to support the HWIR. The HWIR95 framework provided a screening-level approach to evaluate ecological risks to species that are representative of general terrestrial and freshwater aquatic ecosystems. Consequently, much of the data and many of the model constructs developed for HWIR95 were considered appropriate for the SERA of hazardous waste combustors. In addition, the HWIR95 technical framework is consistent with the management goals for the HWC risk analysis as well as ongoing EPA initiatives such as the development of a protocol for ecological risk assessment of HWCs.

Although the HWC SERA retains the basic technical framework and many of the data sources employed under HWIR95, the methodology has been revised for application to HWCs. In particular, the SERA methodology relies on current data and methods developed by EPA for assessing ecological risks from dioxin and mercury exposures. However, it is important to recognize that the SERA is used to evaluate the potential for adverse ecological effects attributable to HWC emissions; the SERA is not designed nor is it intended to quantify the ecological significance and scale of adverse effects predicted in this model. The determination of the ecological significance of HWC emissions at a national scale was beyond the scope of this analysis (see, for example, the *Mercury Study Report to Congress* (U.S. EPA, 1997) for additional discussion of this topic). It is important to recognize that the HWC methodology represents a screening-level approach that is designed to identify the **potential** for adverse ecological effects. The screening nature of the analysis calls to attention three important caveats in interpreting the results:

1. Because the screening methodology is based on the exceedance of a target hazard quotient of 1, the outcome of the screen is binary: HQ > 1 or HQ $\leq$ 1. Although large exceedances suggest a greater **potential** for ecological damage, an HQ of 50 at one site is not necessarily five times worse than an HQ of 10 at another site.

2. The potential for adverse ecological effects (as indicated by an HQ exceedance) should not be confused with the ecological significance of those effects. Regardless of the magnitude of an HQ exceedance, screening results can only

suggest ecological damage; they do not demonstrate actual ecological effects nor do they indicate whether those effects will have significant implications for ecosystems and their components.

3. Ecological receptors for the screening methodology were chosen to represent relatively common species populations and communities of wildlife. Threatened and endangered species and/or habitats were not included in the analysis because a different type of spatial resolution would have been required (i.e., co-occurrence of threatened and endangered species/habitats with HWCs). Consequently, the screening results do not indicate whether endangered species/habitats are at risk.

With these caveats in mind, the screening methodology may be thought of as progressing in three basic phases as described in EPA's *Guidelines for Ecological Risk Assessment* (U.S. EPA, 1998a) and outlined below. The remainder of this section presents the methodologies implemented in each of the SERA phases.

Problem formulation phase–Defines the problem by answering these questions:

– What are the constituents of concern?

– Once released, what is the environmental behavior of the constituents (e.g., persistence, bioaccumulation, speciation)?

– Given the source characterization, what ecosystems and ecological receptors are potentially at risk?

– What adverse ecological effects are possible following exposure?

Analysis phase–Provides estimates of the constituent concentrations in the environment to which ecological receptors are exposed (i.e., exposure profile) and develops ecotoxicological criteria (i.e., acceptable concentrations in environmental media) from data on adverse ecological effects on various receptors.

Risk characterization phase–Compares the modeled exposure concentrations to ecotoxicological criteria developed in the analysis phase to estimate the potential ecological risk (i.e., hazard quotients used in screening-level analysis). Includes a risk description that describes the limitations of the assessment and discusses the ecological significance of HQ exceedances.

9.1 Problem Formulation

A successful problem formulation depends upon the quality of three products: "(1) assessment endpoints that adequately reflect management goals and the ecosystem they represent, (2) conceptual models that describe key relationships between a stressor and assessment endpoint or among several stressors and assessment endpoints, and (3) an analysis plan." (U.S. EPA, 1998a)

9.1.1 Assessment Endpoint Selection

Perhaps the most important step in the problem formulation phase is the selection of assessment endpoints, defined as "explicit expressions of the actual environmental value that is to be protected" (U.S. EPA, 1998a). The assessment endpoints serve as critical links between the ecological risk assessment and the management goal, which, for the HWC risk assessment, is to evaluate the potential ecological benefits associated with various MACT options. Candidates for assessment endpoints often include threatened/endangered species, commercially or recreationally important species, functional attributes that support food sources or flood control, or aesthetic values; e.g., the existence of charismatic species such as eagles (U.S. EPA, 1998a). The assessment endpoints selected for this analysis are outlined in Table 9-1. Assessment endpoints that represent both wildlife populations as well as ecosystem structure and function (e.g., the multiple receptor approach) were identified by their

- Significance to the ecosystem
- Position along a continuum of trophic levels
- Susceptibility to constituents based on exposure and/or toxicological sensitivity.

This approach assumes that, if assessment endpoints are protected from stress caused by exposure to constituents, protection at a higher level of organization (i.e., the ecosystem) may be inferred.

9.1.2 Development of Conceptual Model

Conceptual model development requires the integration of information related to the constituents to be modeled (e.g., environmental behavior such as speciation), ecotoxicological effects data for constituents of concern, receptors and ecosystems potentially at risk, and relevant pathways of exposure. Because combustors are found throughout the United States, virtually any type of ecosystem and ecological receptor may be exposed to HWC constituents. For screening purposes, the conceptual model included ecological receptors that are representative of either freshwater aquatic ecosystems (e.g., streams, lakes, wetlands) or terrestrial ecosystems (e.g., forests, grasslands). Particular emphasis during conceptual model development was placed on constituents demonstrating the potential for adverse effects to receptors (i.e., dioxin and furan congeners, mercury, lead, and selenium).

Because the release of constituents from various combustion units impacts a study area of 20-km radius around the site and because the constituents of concern include metals as well as persistent, bioaccumulative organics, the conceptual model includes both direct and indirect (i.e., food chain) exposures for ecological receptors. Constituents released from HWC stacks may be deposited directly onto plants, soils, and surface waterbodies by wet and/or dry deposition mechanisms. The deposition of constituents within a watershed results in constituent movement by overland transport into waterbodies and, frequently, burial in the bed sediment. Soils and sediments have been shown to be sinks for environmental releases of metals and, therefore, direct contact with these contaminated media may pose potential risks to ecological receptors (e.g., benthic dwellers). In addition, persistent, bioaccumulative constituents such as mercury and dioxin have been shown to bioaccumulate in the food chain and, as a result, upper-trophic-level receptors are particularly at risk through food chain exposures. Inhalation exposures were not

Table 9-1. Assessment Endpoints for Hazardous Waste Combustors Ecological Risk Assessment

Ecological Significance	Assessment Endpoint	Receptors	Characteristic(s)	Measure of Effect
# Upper-trophic-level consumers # Socially valued (e.g., endangered species) # Top recipients of bioaccumulative chemicals # Represent species with large foraging ranges # Represent species with longer life spans	Viable mammalian wildlife populations	e.g., deer mouse, meadow vole, red fox	Reproductive and developmental success	Chronic or subchronic NOAEL(s) or LOAEL(s) for developmental and reproductive effects
	Viable avian wildlife populations	e.g., red-tailed hawk, northern bobwhite	Reproductive and developmental success	Chronic or subchronic NOAEL(s) or LOAEL(s) for developmental and reproductive effects
# Represent species with unique habitat niches (e.g., partially aquatic and terrestrial) # Some species are sensitive to contaminant exposure	Viable amphibian and reptile wildlife populations (“herps”)	e.g., frog, newt, snake, turtle	Reproductive and developmental success	Chronic or subchronic NOAEL(s) or LOAEL(s) for developmental and reproductive effects
# Represent base food web in terrestrial systems # Habitat vital to decomposers and soil aerators # Proper soil community function related to nutrient cycling	Sustainable soil community structure and function	e.g., nematodes, soil mites, springtails, annelids, arthropods	Growth, survival, reproductive success	Point estimates protective of 95% of representative soil species derived from LOEC/NOEC data distributions.
# Primary producers of energy in ecosystems # Act as food base for herbivores # Able to sequester some contaminants # Can act as vectors to bioaccumulation # Constitute a large fraction of the earth’s biomass	Maintain primary terrestrial producers (plant community)	e.g., soy beans, alfalfa, rye grass	Growth, yield, germination	10th percentile from LOEC data distribution
# Highly exposed receptors from constant contact with contaminated media # Act as vectors to transfer contaminants to terrestrial species	Sustainable aquatic community structure and function	e.g., fish (salmonids), aquatic invertebrates (daphnids)	Growth, survival, reproductive success	NAWQC for aquatic life (95% species protection)
# Represent food base for fish # Provide habitat for reproductive lifestages (e.g., eggs, larval forms) # Habitat for key invertebrate species # Act to process nutrients and decompose organic matter	Sustainable benthic community structure and function	e.g., protozoa, flat worms, ostracods	Growth, survival, reproductive success	10th percentile from LOEC data distribution
# Primary producers of energy in the aquatic system # Base food source in the aquatic system # Can act to sequester contaminants from the water column # Act as substrate for other organisms in the water column (e.g., periphyton)	Maintain primary aquatic producers (algal and plant community)	e.g., algae and vascular aquatic plants	Growth, mortality, biomass, root length	EC_{20} for algae; lowest LOEC for aquatic plants

EC_{20} = Effects concentration to 20%.
LOAEL = Lowest observed adverse effects level.
LOEC = Lowest observed effects concentration
NAWQC = National Ambient Water Quality Criteria.

considered in this screening analysis because (1) in most cases aboveground inhalation exposures modeled for humans will result in higher risks than for mammalian wildlife, and (2) inhalation data are generally scant for birds and other wildlife orders. The conceptual model is developed through analysis of (1) environmental behavior of constituents, (2) potential ecological effects, and (3) identification of ecosystems, receptors, and pathways of concern. Figure 9-1 presents a graphic representation of the conceptual model.

In developing the conceptual model, there is some uncertainty associated with characterizing receptor exposures. Uncertainties are specifically related to (1) whether the constituent and receptor will co-occur, (2) to what degree the predator's diet is contaminated, and (3) what role spatial and temporal variables play in the potential for impacts to freshwater and terrestrial receptors. Some of the key issues of uncertainties associated with the development of the conceptual model are outlined briefly below.

Co-occurrence of Receptor and Constituent

The co-occurrence of the constituent and the assessment endpoint was assumed for each HWC facility. This simplification is adopted for screening-level analyses in which site-specific data are not within the scope of the assessment. The HWC SERA does not assess the probability that (1) a receptor will be found in a contaminated sector, (2) a receptor will forage for food in contaminated sectors, or (3) an ecosystem will support the habitat needs of the receptor. This implicit assumption adds to the conservative nature of the screening assessment because not all HWC facilities may be located in areas that are capable of sustaining receptors of concern. However, the ecological receptors that were included in the analysis are commonly occurring species and are presumed to be representative of species that may occur in habitats surrounding facilities.

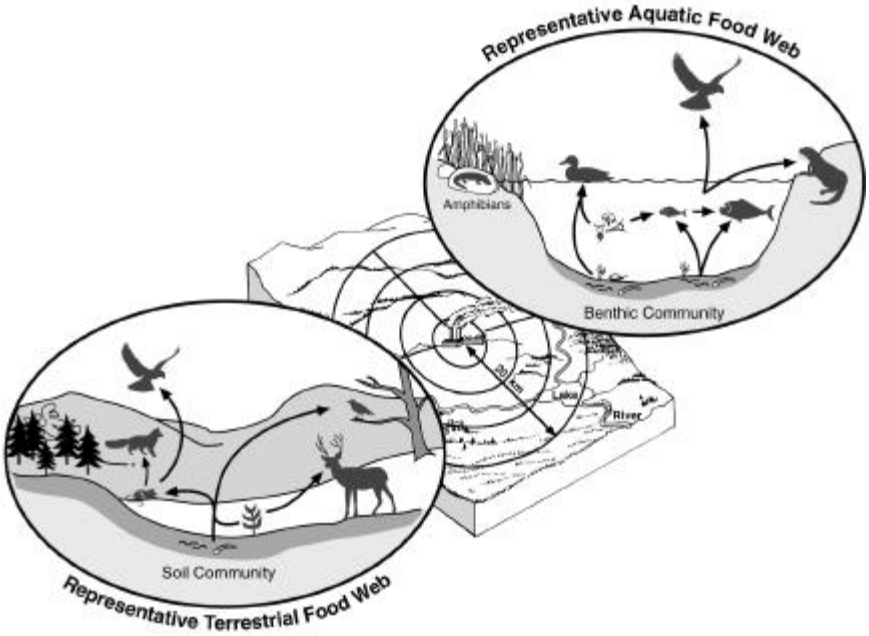

Figure 9-1. Conceptual model for generalized ecosystem approach for HWC screening level assessment.

Assumptions of Dietary Exposure

Screening-level assessments typically assume maximum intake of contaminated prey in the diets of primary and secondary consumers (i.e., 100 percent of the diet originates from the contaminated area). Obviously, under field conditions, many receptors are opportunistic feeders with substantial variability in both the type of food items consumed as well as the seasonal patterns of feeding and foraging. The home range of the ecological receptor is an issue here as well. If an animal forages or hunts for prey over an area larger than a sector, then the exposure could be under- or overestimated. Consequently, the assumption of an exclusive diet of contaminated food items tends to provide a conservative estimate of potential exposures.

Spatial and Temporal Scales of Exposure

Consideration of the spatial extent and pattern of projected HQ exceedances is important in assessing the potential impact to ecological receptors. For example, defining the intersection between projected HQ exceedance areas and ecological receptor habitats at the site-specific level would allow a more refined statement of potential impacts to receptors. Although the HWC risk analysis used a 16-sector template in modeling media concentrations within specific study areas, which does provide significant refinement in evaluating the areal extent of HQ exceedances, the identification of specific habitat areas at the site-specific level was beyond the scope of this analysis. Consequently, it was not possible to quantitatively assess the relationship between projected HQ exceedances and ecological receptor habitats.

The timing of exposure will also influence the impact to a population. If peak exposures occur during sensitive life stages (e.g., juvenile) or during the breeding season, impacts on population dynamics (e.g., percent survival) may be significant. Hence, averaging exposure concentrations over longer periods of time may underpredict risks. Long-term, low-level releases may have cumulative impacts on populations and communities that are not evident from the available laboratory data (i.e., multigenerational studies are not frequently available). Alternatively, such chronic exposures may not ever exceed threshold concentrations at which adverse effects may be observed. The HWC screening analysis was based on a maximum annual exposure concentration and, assuming that peak exposures would not be significantly different from the annual average, the risk estimates tend to be conservative. The magnitude of this conservatism depends on the overall exposure profile (i.e., how variable are the annual exposure concentrations from the maximum).

9.1.2.1 Characterization of Environmental Behavior. The relationship between the chemical properties of each constituent and the chemical conditions in the surrounding media determines, to a large degree, the behavior (e.g., mobility and chemical speciation) of the chemical in ecological systems. Chemical descriptors such as reactivity, solubility, and valency influence how a constituent moves through the environment. For example, a methylated form of a metal adsorbs to organic matter and moves with soil and sediment particles. In contrast, a free ionic form of a metal may react with other anions to form insoluble salts that remain relatively immobile. This discussion of the environmental behavior of chemicals of concern (COCs) considers only the risk drivers identified in the analysis—dioxin and metals (i.e., mercury, lead, and selenium). Environmental behavior issues are discussed further in the geochemistry section of the ecotoxicological profiles provided in Appendix J.

Dioxins and Furans. Dioxin and furan congeners, like other PCDDs and PCDFs, are persistent, bioaccumulative, and hydrophobic. These characteristics influence the environmental behavior of congeners in ecological systems. Overall, the movement of sediments, particulates, and soil erosion mimics the mobility and fate of dioxin. For example, in surface water, dioxins and furans are associated primarily with suspended organic matter, which eventually settles into sediments that commonly act as a sink for dioxin. In addition to the movement of dioxin in soil, sediment, and surface water by abiotic means, dioxin also is mobile biotically. Dioxin is highly bioaccumulative and biomagnifies in food chains. Typically, dioxin is stored in the fat tissues of organisms and undergoes minimal metabolism over time. The secondary sink of dioxin in the tissues of organisms is minimal compared to abiotic sinks such as sediments. However, when stored in biota, dioxin can severely impact particularly sensitive receptors, such as vertebrates, that consume contaminated prey.

Metals. Metals considered in this analysis are influenced by similar chemical reactions and equilibria but can behave very differently depending on environmental conditions. Metals commonly have unique speciation issues related to chemical conditions in surrounding media (e.g., pH, redox potential), which influence not only the ionic form of the metal present but also the complexes and compounds likely to be formed. The metal risk drivers in the combustion analysis are mercury, lead, and selenium. The likely chemical-specific speciation in environmental media is outlined in Table 9-2.

Changes in chemical speciation can alter chemical bioavailability and ultimately the degree of exposure. For the purposes of this screening-level analysis, all forms of a constituent are assumed to be equally bioavailable. This conservative assumption generates some uncertainty in the risk estimates. In many cases, only a fraction of total constituent concentrations are bioavailable and taken up into the tissues of ecological receptors. This assumption provides added conservatism to the analysis. The assumption of complete bioavailability of constituents is appropriate given the screening nature of the analysis; however, both the chemical form and the environmental conditions influence bioavailability and, ultimately, the expression of adverse effects.

9.1.2.2 <u>Identification of Ecological Effects Data</u>. Effects data are evaluated during the development of the conceptual model to identify receptors that may be particularly sensitive to exposure. For example, in the case of silver, significant adverse effects for mammals are not noted in the literature; however, fish appear to be highly sensitive to silver exposure. In this example, a more detailed analysis of fish and the aquatic community is indicated.

Extensive review of the ecotoxicity of risk-driving constituents is provided in the ecotoxicological profile in Appendix J. Ranges of acute and chronic ecotoxicity data, bioaccumulation potential, and criteria development are provided in Appendix J for dioxins/furans, mercury, lead, and selenium.

9.1.2.3 <u>Identification of Ecosystems, Receptors, and Pathways of Concern</u>.

Constituent Selection. The 16 constituents of concern identified by EPA were selected because they have been measured in stack emissions at HWC facilities (see box). The adverse effects suggested by exposure to these constituents are related to: (1) their bioaccumulative

Table 9-2. Characterization of Environmental Behavior of Risk-Driving Metals in HWC ERA

	Mercury	Selenium	Lead
Speciated forms	Hg^0 (elemental) Hg^+ (mercurous) Hg^{2+} (mercuric) Methylmercury	Se^0 (elemental, colloidal forms) Se^{6+} (selenate, SeO_4^{2-}) Se^{4+} (selenite, SeO_3^{2-}, $HSeO_3^-$) Se^{2-} (selenide, organic and inorganic)	Pb^0 (elemental) Pb^{2+} (ionic) Alkylated Pb
Unique behaviors	# Hg^0 readily vaporizes # Microbially mediated biotransformation of mercury compounds forms methylmercury	# Complex speciation scheme determined primarily by pH and Eh	# Speciation dependent on adsorption, precipitation, and complexation
Behavior in soil	# Strongly sorbed to soil substrates (e.g., humic substances) # Leaching is relatively insignificant # Remobilization occurs by pH, chloride ion content, and microbial biotransformation	# Se^0, a stable and insoluble form, occurs under anaerobic conditions # Selenides predominate soils with low pH and high organic content # Soluble selenites, occurring in alkaline to mildly acidic environments, have limited mobility # Selenate predominates at pH>6.5 under oxidizing conditions and is bioavailable to plants	# Primarily sorbed to organic matter # Minimally transported to surface and groundwater # Forms insoluble organocomplexes
Behavior in sediment	# Concentrations correlated with particle size and fraction of organic matter # Strongly sorbs to sediment particulates # Sediments act as a sink # Under anaerobic conditions, methyl-mercury is released from sediments into the water column	# Sediments act as a sink for Se # Associated with the organic material, iron and manganese oxides, carbonates, or other mineral phases # Dissolved ions are scavenged by abiotic and biotic means	# Sediments act as a sink for Pb # Anaerobic conditions produce relatively volatile organo-tetramethyl lead through biological alkylation
Behavior in surface water	# Principal forms include Hg^0, Hg^{2+}, dissolved organic complexes, and particulate-bound methylmercury and Hg^{2+} # Reactions with particulates dominate the fate of Hg	# Found in dissolved and particulate forms # Both selenite and selenate are common in surface waters # Elemental selenium and selenide should dominate under anoxic conditions	# Speciation of lead controlled by balance between complexed and dissolved organic matter and suspended solids

nature, resulting in high exposures to receptors further up the food chain; or (2) their highly persistent nature, resulting in chronic long-term exposures to some receptors.

Constituents Screened for Ecological Risks

Antimony
Arsenic
Barium
Beryllium
Cadmium
Chromium VI
Chromium III
Cobalt
Copper
Lead
Manganese
Mercury (elemental)
Mercury (divalent)
Mercury (methyl)
Nickel
Selenium
Silver
2,3,7,8-TCDD (as TEQs)
Thallium

Ecosystem Selection. The selection of generalized freshwater and terrestrial ecosystems provides a screening-level context for the HWC risk analysis. Generalized representative ecosystems are a simplification of true ecosystems, but they capture the basic elements characteristic of most freshwater and terrestrial ecosystems. Generalized freshwater ecosystems include a variety of waterbodies such as lakes and rivers. Variables that will influence the wildlife communities able to inhabit the waterbodies include water flow rate, bed sediment composition, periodicity of flood events, and the presence of aquatic vegetation. Since these variables were not explicitly used to characterize freshwater ecosystems, a level of uncertainty is introduced into the assignment of appropriate food webs to waterbodies. There is added uncertainty associated with how the waterbodies were selected in the HWC assessment. Waterbodies evaluated for potential impacts within the 20-km radius surrounding facilities were selected based on their utility as a drinking water source, their recreational importance, or their proximity to facilities. Although the selection process is appropriate for evaluating human health risks, it may not adequately represent the aquatic habitats at risk from HWC emissions. Waterbodies and wetlands with high ecological significance may not have been represented in the analysis. In addition, a single waterbody exhibiting a target HQ exceedance may be assumed to have local (and somewhat limited) ecological significance. However, if several waterbodies in the proximity of the facility are shown to have modeled concentrations that exceed the ecotoxicological criterion, the adverse impacts on aquatic life may be more significant. This issue was examined indirectly by estimating the total waterbody area in exceedance and the corresponding number of facilities. However, since not all waterbodies surrounding facilities were represented in the total area, there is some uncertainty in characterizing the potential impacts to freshwater ecosystems.

Generalized terrestrial ecosystems are soil-based ecosystems such as forests and grasslands. The composition of receptors within a terrestrial ecosystem is highly dependent on the physical structures (i.e., geology, soil composition, and vegetation) of the habitat. Since the variability in vegetation cover and soil types was not considered in this analysis, a level of uncertainty was introduced into assigning food webs that are appropriate to most generalized terrestrial habitats. Because modeled combustion facilities are selected to represent the universe of facilities, release of COCs could occur virtually anywhere within the United States; therefore, the selection of more generalized ecosystem habitats (i.e., freshwater, terrestrial) is likely to cover the broadest range of potentially exposed habitats.

Receptor Selection. Lacking a precedent for the selection of ecological receptors in this regulatory context, criteria [1] have been developed that reflect the assessment endpoints and goals for this risk analysis. Given the national scale of this analysis, it is appropriate to select a suite of ecological receptors that represent major trophic elements of ecosystems into which constituents may be released. The ecological receptors should encompass a wide range of dietary preferences and body sizes and, by virtue of their ecological niche, should have the potential to be highly exposed to constituents released to the environment. By protecting producers (i.e., plants) and consumers (i.e., predators) at different trophic levels, as well as certain structural components (e.g., benthic community), a degree of protection from constituents may be inferred to the ecosystem as a whole.

For the HWC analysis, a fundamental approach has been developed to select ecological receptors representing different levels of biological organization based on: (1) the spatial distribution of chemical stressors in the environment with respect to receptor characteristics (e.g., home ranges), and (2) the availability of data with which to assess the risks to that receptor (e.g., toxicity, accumulation potential). The ecological receptor groups include representative species populations as well as generalized communities (e.g., soil community). The representative receptors given as examples in the bullets are often chosen for screening ecological analyses because data are available to characterize exposures in the *Wildlife Exposure Factors Handbook* (U.S. EPA, 1993b).

- **Mammals**–Mammals include upper-trophic-level predators (e.g., red fox), and lower-trophic-level consumers such as ruminants (e.g., deer) and insectivores (e.g., shrew, bat). Representative species cover a variety of body sizes, habitats, and dietary habits for which life history data (e.g., body weight, food intake) are available.

- **Birds**–Birds also include upper-trophic-level predators (e.g., great blue heron) and lower-trophic-level consumers that eat small vertebrates (e.g., hawk), earthworms or large insects (e.g., kestrel), or vegetation (e.g., bobwhite quail). As with mammals, representative species encompass a variety of body sizes, habitats, and dietary preferences for which data are available.

- **Amphibians**–Amphibians are currently under significant stress worldwide. Moreover, these organisms appear to be highly sensitive to a number of toxicants during the developmental stages of their life cycle (e.g., trace metals). They are essential parts of a number of food webs (particularly wetlands areas) and are likely to provide a fairly sensitive indicator for chemical stressors relevant to higher levels of biological organization (e.g., ecosystem level). Though amphibians are a significant ecological receptor, ecotoxicity data characterizing the low effects dose-response relationship are limited. After a review of several compendia presenting amphibian ecotoxicity data (e.g., U.S. EPA, 1996; Power et

[1] For the purposes of this report, ecological screening criteria refer to pollutant concentrations (e.g., mg/kg soil) in environmental media that are presumed to cause no adverse effects to ecological receptors. Benchmark studies, reported as doses mg/kg-d, provide the ecotoxicity data to derive the criterion. Criteria can be based on one benchmark study or on a series of study results.

al., 1989) as well as primary literature sources, it was determined that the chronic data available were insufficient to develop a chronic criteria for amphibians. Given the lack of data, this receptor will not be assessed further in the HWC SERA.

Plants–As primary producers, vascular plants are crucial components of virtually any type of terrestrial ecosystem. Representative species for plant communities are problematic for this application due to the general paucity of toxicity data on plants not grown as food crops. Species of plants used to represent plants within terrestrial ecosystems are frequently limited to forage grasses and food crops.

Soil Community–Invertebrate species (e.g., earthworms, nematodes, insects) and microflora are crucial to the structure and function of a "healthy" soil community (i.e., the community performs all of the essential functions such as mineralization, decomposition, etc). Organisms living in or on the soil are exposed through direct contact with contaminated soil and through the ingestion of contaminated soil and other soil biota such as centipedes (i.e., indirect food web exposure).

Freshwater Community–Fish and aquatic invertebrates are important organisms in the aquatic ecosystem. Both are subject to continuous exposure to contaminated water through gill exchange and may be highly exposed to bioaccumulative chemicals through the food chain. They occupy niches as both predator and prey, and the aquatic invertebrates include a diverse community of organisms (e.g., arthropods, molluscs, annelids). The extensive database on aquatic invertebrates suggests that arthropods are among the most sensitive aquatic species (Suter, 1993).

Algae and Aquatic Plants–Vascular aquatic plants and algae typical of freshwater aquatic ecosystems help oxygenate the water and are important food sources. Algal species primarily include green, blue-green, and diatoms; data on vascular plants are generally found only for duckweed (e.g., *Lemma minor*, *Spriodela polyrhize*).

Benthic Community–The benthic community is composed of a variety of organisms that are indigenous to most freshwater ecosystems, including organisms that break down decaying materials (e.g., detritivores) and others that filter organic materials from the water (e.g., filter feeders). Because these organisms spend most (if not all) of their lives in the sediment, they are exposed through direct contact and ingestion of contaminated sediments.

9.1.3 Analysis Plan

The analysis plan is the third critical product of the problem formulation phase. In essence, the analysis plan provides a blueprint for implementing the conceptual model to identify which receptors may elicit adverse effects from exposure to constituents. The analysis plan can be broken down into two sections: an exposure analysis and an ecological response analysis. For the exposure analysis, fate and transport algorithms from atmospheric and overland transport

were used to determine concentrations of constituents in the environmental media surrounding HWC facilities. For the ecological response analysis, an extensive review of ecotoxicological data was conducted to determine levels of constituents that are anticipated to cause no adverse effects to representative receptors surrounding HWC facilities. These two components (i.e., exposure analysis and ecological response analysis) are used in risk estimation to determine whether modeled media concentrations exceed concentrations determined to be protective of receptors. When exceedances are observed, the potential for adverse effects to receptors is indicated. It may be necessary, however, to identify the specific nature of these potential effects to interpret their ecological significance. The implementation of the analysis plan is conducted in the analysis phase of the ecological risk assessment process.

9.2 Analysis Phase

The analysis phase supports the development of two critical products: (1) the exposure profile, which, for this analysis, provides modeled constituent concentrations in various environmental media for sector-specific areas surrounding HWC facilities; and (2) the stressor-response profile, which is used to support the development of ecotoxicological benchmarks and criteria for the representative aquatic and terrestrial receptors based on de minimus risk to receptors. For the population of representative combustion facilities, environmental concentrations of constituents were modeled in 16 sectors for surficial soil, surface water (i.e., dissolved and total concentrations), and bed sediment to produce sector-specific exposure profiles. For the 16 constituents modeled for this assessment, ecological effects data, including information on speciation and bioaccumulation potential, were identified and ecotoxicological criteria were developed within the limits of the available data. Methodologies used to estimate the exposure of ecological receptors to metal constituents was consistent throughout the assessment; however, assessing the exposure of dioxin and furan congeners required an expanded approach. Variations between methods are detailed in different subsections where necessary in the analysis phase. The exposure profile is outlined in Section 9.2.1; however, the implementation of the fate and transport model used to generate media concentrations is fully discussed in Section 5.0. The ecotoxicological criteria (i.e., methods and proposed criteria) for sediment, soil, and surface water receptors are explained fully in Section 9.2.2.

9.2.1 Development of Exposure Profile

For each of the representative sites, constituent concentrations in the soil, surface waterbody, and sediments were estimated. For soils, area-averaged concentrations were estimated for the 16 sectors in the modeling grid. A conservative approach was used at the screening level and, therefore, it was presumed that each sector included terrestrial and/or freshwater habitats suitable for the ecological receptors chosen for this analysis. Presentation and discussion of input parameters and modeling methodologies for both air and overland transport are expanded in Section 5. Issues related specifically to modeling exposure concentrations in ecological systems include:

- The primary products generated by the model include concentrations of the constituents in the environmental media of soil, bed sediment, and surface water.

- Constituent releases are modeled assuming that (1) emissions from facilities were constant for 30 years, (2) the resulting soil concentrations were calculated at year 30, and (3) surface water concentrations were modeled at steady state assuming year 30 soil concentrations.

- Media concentrations for metals in soil are generated as total metal concentrations (i.e., not distinguishing between chemical forms) while both total and dissolved metal concentrations are generated for surface water.

- With respect to fate and transport pathways, constituent concentrations in soil resulted from both particulate and vapor deposition in conjunction with loss terms for leaching, erosion, runoff, and volatilization.

- Constituent concentrations in surface water, and ultimately sediment, result from five fate and transport pathways including: (1) direct deposition, (2) runoff from impervious surfaces within the watershed, (3) runoff from pervious surfaces (i.e., watershed soils) within the watershed, (4) soil erosion from the watershed, and (5) direct diffusion of the dry-vapor-phase contaminant into the surface water.

9.2.1.1 Metals. The exposure profile was generated using the fate and transport model discussed in Section 5. It should be noted that these modeling techniques do not reflect the complex speciation dynamics of many metals (with the exception of mercury). The model is designed to partition cationic metals between suspended particulates and the “dissolved” phase, but it does not distinguish between free ionic metals and other “dissolved” forms of metals (e.g., complexes) that are less toxic. The implication of applying this model is that the exposure concentrations estimated for sediment and surface water tend to be conservative. Although it is clear that the vast majority of free ionic forms of many metals released into the environment are rapidly sorbed, precipitated, or complexed into relatively nontoxic forms, the fraction of metals that remain soluble and bioavailable is not well characterized. For mercury, a more robust model was adopted. This model characterized the natural speciation of mercury (i.e., total and dissolved) and methylmercury in soil and surface water with more confidence than previous methods. Since the toxicity between divalent mercury and methylmercury can vary by orders of magnitude, the importance of adequately representing this relationship warranted use of this more refined modeling methodology. The intricacies of the model are outlined in detail in Sections 5.3.2.3 for soils and 5.3.3.2 for surface water.

9.2.1.2 Dioxin. The exposure profile for dioxin and furan congeners was generated for specific dioxin congeners. An expanded list of congeners is provided in the text box. The specific methodology and assumptions used to model the fate and transport of dioxin-like substances is outlined in Section 5. Soil and sediment media concentrations developed in this task are used to derive exposure concentrations for mammals and birds of terrestrial and freshwater ecosystems, respectively.

9.2.2 Development of Stressor-Response Profiles

The stressor-response profile supports the derivation of benchmarks and criteria that are relevant to the assessment endpoints. Ecotoxicological data are collected and evaluated on acute

endpoints (e.g., lethality), chronic endpoints (e.g., growth, reproduction), and bioaccumulation potential to develop criteria that reflect the nature of exposure (i.e., direct or food web). For clarity, a distinction is made between the definitions of benchmarks and criteria. Benchmarks are dose values (mg constituent/kg body weight-d) derived from ecotoxicological studies on mammals and birds. Converting a benchmark to a criterion requires background information on consumption rates, dietary preferences, and constituent concentrations in prey. Criteria are the media-specific environmental concentrations in either surface water (mg/L), sediment (mg/kg), or soil (mg/kg) that are estimated to be protective of receptors of concern.

List of Dioxin and Furan Congeners of Ecological Concern

Furans*

1, 2 ,3, 4, 6, 7, 8-HpCDF
1,2 ,3 ,4, 7, 8, 9-HpCDF
1, 2, 3, 7, 8, 9-HxCDF
1,2, 3, 4, 7, 8-HxCDF
1, 2, 3, 6, 7, 8-HxCDF
2, 3, 4, 6, 7, 8-HxCDF
1, 2, 3, 7, 8-PeCDF
2, 3, 4, 7, 8-PeCDF
2, 3, 7, 8-TCDF
OCDF

Dioxins*

1, 2, 3, 4, 6, 7,8- HpCDD
1, 2, 3, 4, 7, 8-HxCDD
1, 2, 3, 6, 7, 8-HxCDD
1, 2, 3, 7, 8, 9-HxCDD
1, 2, 3, 7, 8-PeCDD
2, 3, 7, 8-TCDD
OCDD

* Hp = Hepta; Hx = Hexa; Pe = Penta; T = Tetra; O = Octo; CDF = chlorodibenzofuran; CDD = chlorodibenzodioxin

It is useful to think about the criteria developed from stressor-response profiles in terms of either population-inference criteria or community-type criteria. Population inference concentration limits are established as described in Section 9.2.2.1 and generally reflect exposures through ingestion of contaminated media and food items (e.g., plants, prey). For wildlife populations of mammals and birds, ecological effects data are gathered on endpoints presumed to be relevant to population dynamics, such as reproductive effects (e.g., decreased sperm count), developmental anomalies that reduce the number of viable offspring, or behavioral changes that could impair the ability of an animal to survive. Effects data are reviewed and summarized in the ecotoxicological profiles for risk-driving constituents (Appendix J) (also called the stressor-response profile). After the benchmark study is identified, this value is converted using life history data, food consumption rates, and dietary preferences about the receptor to determine a media-specific criterion. On the other hand, community-type concentration limits (e.g., ambient water quality criteria) are established as described in Section 9.2.2.2 and generally reflect direct exposures to contaminated media. It should be noted that the criteria for receptor communities are not truly community-level concentration limits because predator-prey interactions are not considered. Rather, only the direct effect caused by exposure to constituents was considered (see, for example, Stephan et al., 1985, for additional detail).

For metals and dioxin congeners, different methods were used to assess food chain exposures. In the presentation of this methodology (Section 9.2.2.1), dioxin and metals are considered separately in the case of mammals and birds. The basic steps to estimate exposure to these receptor taxa (i.e., scaling of benchmark, identification of bioaccumulation data, and

calculation of criteria/benchmark) remained consistent across the constituents; however, the specific equations and some data requirements changed between constituents. NOTE: Because of differences in methodologies, metals are reported as criteria (i.e., protective media concentrations) and dioxin is reported as a benchmark (i.e., protective dose).

9.2.2.1 Criteria Development for Wildlife Populations. For populations of **mammals and birds**, the overall approach used to establish ecotoxicological benchmarks is similar to the methods used to establish reference doses for humans as described in IRIS (U.S. EPA, 1998b). Each method uses a hierarchy for the selection of toxicity data and extrapolates from a test species to the species of interest. However, there are fundamental differences in the goals of noncancer risk assessments for humans and ecological receptors. Risk assessments of humans seek to protect the individual, while risk assessments of ecological receptors typically seek to protect populations or communities of important species (U.S. EPA, 1992b). Consequently, benchmarks for mammals and birds were established using three key guidelines:

1. Because population viability in mammals and birds was selected as the assessment endpoint, the benchmarks were developed from measures of reproductive/developmental success or, if unavailable, other effects that could conceivably impair population dynamics.

2. The population-inference benchmark based on a NOAEL for individual organisms on reproductive endpoints was the measure of effect used for mammals and birds). Relatively few "true" population-level benchmarks have been developed to date; these benchmarks are considered to be more rigorous than the point estimates developed from toxicity studies.

3. Uncertainty factors (UFs) were only applied to extrapolate a NOAEL from a LOAEL (i.e., division by 10).

Once the benchmark study is identified, the criterion is calculated for each medium of interest using a three-step progression of data collection and derivation calculations. The remainder of this section outlines the basic technical approach applied to estimate benchmarks and criteria protective of ecological receptors for metals and dioxin congeners, respectively.

***Estimating Criteria for Metals*.**

Step 1. Scale benchmark: The benchmarks derived for various taxa (e.g., mammals) can be extrapolated to other species within a taxa by the cross-species scaling equation (Sample et al., 1996). For population-inference benchmarks utilized in the HWC analysis, the extrapolation is performed using Equation 9-1.

$$\text{Benchmark}_w = \text{NOAEL}_t \times \left(\frac{bw_t}{bw_w}\right)^{1/4} \qquad (9\text{-}1)$$

where

$Benchmark_w$	=	scaled benchmark for wildlife species (mg/kg-d) (Table 9-3)
$NOAEL_t$	=	no-observed-adverse-effects level for test species (mg/kg-d) (Table 9-4)
bw_t	=	body weight of test species (kg) (Table 9-4)
bw_w	=	body weight of aquatic wildlife species (kg) (Table 9-5) or of terrestrial wildlife species (kg) (Table 9-6).

This is the default methodology EPA proposed for carcinogenicity assessments and reportable quantity documents for adjusting animal data to an equivalent human dose (U.S. EPA, 1992a). It should be noted that recent research suggests that cross-species scaling may be problematic for avian species (Mineau et al., 1996). Mineau et al. (1996) used a database that characterized acute toxicity of pesticides to avian receptors of various body weights. The results of the regression analysis revealed that applying mammalian scaling equations may not sufficiently predict protective doses for avian species. Small-bodied avian species were especially at risk from dose estimates generated by cross-species scaling that are not protective enough. It is also unclear whether protective levels for constituents with different toxic mechanisms (such as metals) would be underpredicted when cross-species scaling equations are used. Applying the scaling equation to birds generates some uncertainty in the development of criteria for avian receptors.

To remain consistent with the *Mercury Study Report to Congress* (U.S. EPA, 1997), the same benchmark dose studies were used to evaluate freshwater and terrestrial receptors. However, for terrestrial receptors, scaling methods were used to extrapolate to representative wildlife, and, for freshwater receptors, uncertainty factors were used to extrapolate to representative wildlife.

Step 2. Identify BCFs/BAFs: Movement of contaminants through the food web is the primary vector of exposure for mammals and birds. To derive a media- specific criteria, estimates of chemical accumulation in the tissues of prey items are necessary. Uptake factors (e.g., bioaccumulation factors) of various prey items were used to estimate ingestion exposures to mammals and birds. The prey items for which uptake factors were required included fish and aquatic invertebrates for the freshwater ecosystem and plants, soil invertebrates (e.g., earthworms), and small vertebrates for the terrestrial ecosystem. For metals, measured uptake factors were identified in the primary literature, EPA databases (e.g., AQUIRE), and other compendia and review sources (e.g., National Ambient Water Quality Criteria; Fish and Wildlife Service Hazard Reviews). The results of data collection efforts to identify measured uptake factors are presented in Table 9-7 for freshwater prey items and Table 9-8 for terrestrial prey items. In many cases, data were not sufficient to quantify uptake into all prey items.

There is some uncertainty associated with selecting uptake factors for prey items in freshwater and terrestrial ecosystems. Deriving an appropriate bioaccumulation metric that properly characterizes the magnitude, rate of uptake, and elimination of constituents in ecological receptors is a point of uncertainty in this analysis. The rationale and selection of these values for

Table 9-3. Scaled Benchmark Doses for Aquatic and Terrestrial Receptors (mg/kg-d)[a]

Species	Antimony	Arsenic	Barium	Cadmium	Chromium^{6+}	Chromium^{3+}	Copper	Lead	Methyl-mercury[b]	Nickel	Selenium	2,3,7,8-TCDD
Mammals												
Short-tailed shrew	0.28	10.43	ID	2.09	3.51	ID	15.93	0.0096	0.77	92.17	0.42	2.1E-06
Deer mouse	0.28	10.19	ID	2.02	3.38	ID	15.57	0.0093	0.75	88.98	0.41	2.1E-06
Meadow vole	0.23	8.49	ID	1.77	2.79	ID	12.97	0.0077	0.63	77.95	0.34	1.7E-06
Eastern cottontail	0.10	3.58	ID	0.72	1.23	ID	5.47	0.0034	0.26	31.58	0.14	7.3E-07
Red fox	0.07	2.66	ID	0.52	0.85	ID	4.06	0.0023	0.20	22.74	0.11	5.4E-07
Raccoon	0.07	2.56	ID	0.49	0.80	ID	3.91	0.0022	0.19	21.56	0.10	5.2E-07
White-tailed deer	0.03	1.28	ID	0.25	0.39	ID	1.95	0.0011	0.0939	10.9308	0.0515	2.6E-07
Mink	0.11	4.11	ID	0.75	1.18	ID	6.28	0.0032	0.018	33.03	0.17	8.4E-07
River otter	0.06	2.29	ID	0.45	0.74	ID	3.50	0.00203	0.018	19.74	0.09	4.7E-07
Birds												
Red-tailed hawk	ID	0.03	12.00	1.55	ID	1.02	38.94	0.0123	0.0064	70.54	0.49	1.4E-05
American kestrel	ID	0.06	21.08	1.59	ID	0.98	43.87	0.0120	0.0073	72.43	0.55	2.4E-05
Northern bobwhite	ID	0.05	19.19	2.48	ID	1.64	62.24	0.0201	0.0103	112.76	0.79	2.2E-05

(continued)

Table 9-3. (continued)

Species	Antimony	Arsenic	Barium	Cadmium	Chromium^{6+}	Chromium^{3+}	Copper	Lead	Methyl-mercury[b]	Nickel	Selenium	2,3,7,8-TCDD
American robin	ID	0.06	23.21	3.00	ID	1.98	75.31	0.0240	0.0125	136.43	0.95	2.7E-05
American woodcock	ID	0.05	19.33	2.50	ID	1.65	62.70	0.0191	0.0104	113.59	0.79	2.1E-05
Bald eagle	ID	0.02	8.90	1.15	ID	0.76	28.87	0.009	0.026	52.30	0.36	9.8E-03
Osprey	ID	0.03	10.96	1.42	ID	0.94	35.56	0.011	0.026	64.43	0.45	1.2E-02
Great blue heron	ID	0.03	10.02	1.29	ID	0.86	32.50	0.011	ID	58.87	0.41	1.2E-02
Mallard	ID	0.03	11.93	1.54	ID	1.02	38.70	0.013	ID	70.11	0.49	1.4E-02
Lesser scaup	ID	0.03	13.31	1.72	ID	1.14	43.17	0.014	ID	78.21	0.54	1.5E-02
Kingfisher	ID	0.05	19.99	2.58	ID	1.71	64.85	0.021	0.026	117.48	0.82	2.3E-02
Spotted sandpiper	ID	0.07	27.28	3.52	ID	2.33	88.49	0.028	ID	160.30	1.12	3.1E-02
Herring gull	ID	0.03	12.12	1.56	ID	1.03	39.31	0.013	ID	71.21	0.50	1.4E-02

ID = Insufficient data.
[a]For the following constituents, ecotoxicity data were unavailable for mammals and birds: beryllium, cobalt, manganese, elemental mercury, and silver.
[b]Scaling factors used for terrestrial mammals and birds; uncertainty factor of 3 applied to freshwater mammals and birds (U.S. EPA, 1997).

Table 9-4. Values Used to Calculate Scaled Benchmark for Aquatic and Terrestrial Wildlife

Constituent	bw_t (Kg)	$NOAEL_t$ (mg/kg-d)	Source
Antimony	0.255 (rat)	0.143	Rossi et al., 1987
Arsenic	1.043 (mallard) 0.439 (rat)	0.006 4.627	Stanley et al., 1994 Byron et al., 1967
Barium	0.121 (chick)	21.0	Johnson et al., 1960 (cited in Sample et al., 1996)
Beryllium	ID	ID	
Cadmium	1.53 (mallard) 0.321 (rat)	1.438 1.000	White and Finley, 1978 Sutou et al., 1980
$Chromium^{6+}$	0.023 (mouse)	3.3	Zahid et al., 1990
$Chromium^{3+}$	1.25 (duck)	1.0	Sample et al., 1996
Cobalt	ID	ID	
Copper	0.534 (chick) 0.745 (mink)	47 6.2	Sample et al., 1996 Aulerich et al., 1982
Lead	0.15 (quail) 0.235 (rat)	0.0207 0.005	Edens and Garlich, 1983 Krasovskii et al., 1979
Manganese	ID	ID	
Mercury (elemental)	ID	ID	
Mercury (inorganic)[a]	1.162 (mallard) 0.80 (mink)	0.078 (LOAEL) 0.055	Heinz, 1975; 1976a,b; 1979 Wobeser, 1973, 1976a,b
Mercury (methyl)	1.162 (mallard) 0.80 (mink)	0.078 (LOAEL) 0.055	Heinz, 1975; 1976a,b; 1979 Wobeser, 1976a,b
Nickel	0.782 (mallard) 0.148 (rat)	77.4 53.511	Sample et al., 1996 Ambrose et al., 1976
Selenium	1.055 (mallard) 0.320 (rat)	0.500 0.202	Heinz et al., 1987 Rosenfeld and Beath, 1954
Silver	ID	ID	
Thallium	ID	ID	
2,3,7,8-TCDD	0.255 (rat) 1.1 (pheasant)	1.0E-06 1.4E-05	U.S. EPA, 1995b

ID = Insufficient data.

[a] Same benchmark studies used to evaluate mammals and birds; however, a final adjustment factor (0.078) factor applied to methylmercury criteria to derive total dissolved mercury criteria in surface water (U.S. EPA, 1997).

Note: For the aquatic bw_w value, see Table 9-5.
For the terrestrial bw_w value, see Table 9-6.
For the $benchmark_w$ value, see Table 9-3.

Table 9-5. Life History Parameters on Representative Piscivorous Species in the Freshwater Ecosystem

	Representative Species	Body Weight (kg)	Water Intake (L/d)	Food Intake (kg-d)	Dietary Consumption (% volume)
Mammals	**Mink**				
	female	0.70	0.05	0.11	100% fish (trophic level 3)
	male	1.34	0.13	0.21	
	both	1.02	0.081	0.16	
	River otter				
	female	7.32	0.60	1.18	100% fish (0.5 trophic level 3) (0.5 trophic level 4)
	male	8.67	0.69	1.35	
	both	7.99	0.65	1.26	
Birds	**Bald eagle**				
	female	4.50	0.16	0.54	100% fish (trophic level 4)
	male	3.00	0.11	0.36	
	both	3.75	0.14	0.45	
	Osprey				
	female	1.77	0.09	0.37	100% fish (trophic level 3)
	male	1.43	0.08	0.30	
	both	1.63	0.08	0.34	
	Great blue heron				
	female	2.20	0.10	0.40	100% fish (trophic level 4)
	male	2.58	0.12	0.46	
	both	2.34	0.11	0.42	
	Mallard				
	female	1.11	0.06	0.31	100% aquatic invertebrates (trophic level 2)
	male	1.24	0.07	0.33	
	both	1.16	0.07	0.32	
	Lesser scaup				
	female	0.73	0.05	0.24	100% aquatic invertebrates (trophic level 2)
	male	0.86	0.05	0.26	
	both	0.75	0.05	0.24	
	Kingfisher				
	female	0.15	0.02	0.07	100% fish (trophic level 3)
	male	0.15	0.02	0.07	
	both	0.15	0.02	0.07	
	Spotted sandpiper				
	female	0.05	0.01	0.03	100% aquatic invertebrates (trophic level 2)
	male	0.04	0.01	0.03	
	both	0.04	0.01	0.03	
	Herring gull				
	female	0.98	0.06	0.19	100% fish (trophic level 3)
	male	1.21	0.07	0.24	
	both	1.09	0.06	0.21	

Note: Unless otherwise indicated, all values are taken from U.S. EPA, 1993b.

Table 9-6. Life History Parameters on Representative Terrestrial Species

	Representative Species	Body Weight (kg)	Soil Intake		Food Intake (kg-d)	Dietary Consumption (% volume)
			% of diet	kg-d		
Mammals	**Short-tailed shrew**					
	female	0.017	1	9.4E-05	0.0094	13% plants 31% earthworms 39% invertebrates
	male	0.017	1	9.5E-05	0.0095	
	both	0.017	1	9.2E-05	0.0092	
	Deer mouse					
	female	0.019	2	7.1E-05	0.0035	44% plants 43% invertebrates
	male	0.020	2	8.8E-05	0.0044	
	both	0.019	2	7.4E-05	0.0037	
	Meadow vole					
	female	0.039	2.4	3.0E-04	0.013	98% plants 2% invertebrates
	male	0.043	2.4	3.3E-04	0.014	
	both	0.033	2.4	2.6E-04	0.011	
	Eastern cottontail					
	female	1.22	6.3	6.4E-03	0.10	100% plants
	male	1.13	6.3	6.0E-03	0.10	
	both	1.22	6.3	6.4E-03	0.10	
	Red fox					
	female	4.04	2.8	8.1E-03	0.29	4% plants 96% vertebrates
	male	5.04	2.8	1.0E-02	0.36	
	both	4.54	2.8	1.2E-02	0.43	
	Raccoon					
	female	4.71	9.4	2.3E-02	0.25	29% plants 52% invertebrates 10% vertebrates
	male	6.22	9.4	2.9E-02	0.31	
	both	5.62	9.4	2.7E-02	0.28	
	White-tailed deer					
	female	76.00	2	4.1E-02	2.04	100% plants
	male	110.00	2	5.3E-02	2.67	
	both	85.00	2	4.4E-02	2.21	
Birds	**Red-tailed hawk**					
	female	1.20	1	1.3E-03	0.13	100% vertebrates
	male	1.06	1	1.1E-03	0.11	
	both	1.13	1	1.1E-03	0.11	
	American kestrel					
	female	0.13	1	3.7E-04	0.037	49% invertebrates 51% vertebrates
	male	0.11	1	3.4E-04	0.034	
	both	0.12	1	3.6E-04	0.036	
	Northern bobwhite					
	female	0.17	9.3	1.2E-03	0.013	87% plants 13% invertebrates
	male	0.16	9.3	1.2E-03	0.013	
	both	0.17	9.3	1.3E-03	0.014	
	American robin					
	female	0.082	1	9.9E-04	0.10	11% plants 89% invertebrates
	male	0.082	1	9.9E-04	0.10	
	both	0.081	1	9.8E-04	0.10	
	American woodcock					
	female	0.20	10.4	1.6E-02	0.16	(summer diet) 68% earthworms 11% plants 20% invertebrates
	male	0.15	10.4	1.2E-02	0.12	
	both	0.17	10.4	1.3E-02	0.13	

Table 9-7. Bioconcentration Factors for the Generalized Terrestrial Ecosystem

Constituent	Worms	Reference	Invertebrates	Reference	Vertebrates	Reference	Plants	Reference
Arsenic	ID		ID		ID		2.0E-03	U.S. EPA, 1992c
Cadmium	2.3E+00	Taylor, 1983 Eisler, 1985 Canton and Slooff, 1982 Kumada et al., 1980 Kumada et al., 1972 U.S. EPA, 1992c Williams and Geisey, 1978 Geisey et al., 1977	ID		ID		3.4E+00	U.S. EPA, 1992c
Copper	ID		ID		ID		1.5E+00	U.S. EPA, 1992c
Lead	3.5E-02	Hartenstein et al., 1980	ID		ID		2.0E-03	U.S. EPA, 1992c
Mercury	2.7E+01		ID		ID		ID	
Nickel	3.2E-02	Hartenstein et al., 1980	ID		ID		8.5E-01	U.S. EPA, 1992c
2,3,7,8-TCDD	9.1E+00	Martinucci et al., 1983 Abt and Associates, 1993 Reinecke and Nash, 1984	1.3E+00	Abt, 1993	7.2E+00	Kociba et al., 1978 Jensen et al., 1981 Weerasinghe & Gross, 1985 Garten & Trabalka, 1983 Abt and Associates, 1993	ID	

ID = Insufficient data.

Sufficient data not available for the following chemicals: antimony, barium, beryllium, chromium, cobalt, manganese, selenium, silver, and thallium.

Table 9-8. Bioaccumulation Factors and Bioconcentration Factors for Generalized Freshwater Ecosystem

Constituent	BCF or BAF	Dissolved or Total	Fish Tissue	Trophic Level 3 Fish	Trophic Level 4 Fish	Reference
Antimony	BCF	t	whole	0	0	Stephan, 1993
Arsenic	BCF	t	whole	3.46	3.46	Stephan, 1993
Beryllium	BCF	t	whole	19	19	Barrows et al., 1980
Cadmium	BCF	t	whole	187	187	Taylor, 1983 Eisler, 1985 Canton and Slooff, 1982 Kumada et al., 1980 Kumada et al., 1972 U.S. EPA, 1992c Williams and Geisey, 1978 Geisey et al., 1977
Chromium	BCF	t	whole	0.6	0.6	Stephan, 1993
Copper	BCF	t	muscle	0	0	Stephan, 1993
Lead	BAF	t	whole	45.7	45.7	Stephan, 1993
Manganese				ID	ID	
Methylmercury	BAF	d	whole	1.6E+06	6.8E+06	U.S. EPA, 1997
Nickel	BCF	t	whole	0.80	0.80	Stephan, 1993
Selenium	BAF	t	muscle	485	1,692	Lemly, 1985
Silver	BAF	t	whole	0	0	Stephan, 1993
2,3,7,8- TCDD and Congeners	BSAF	t	lipid	See Table 5-7	See Table 5-7	Bauer, 1992

ID = Insufficient data.

Note: Insufficient data for trophic level 2 organisms. Sufficient data not available for the following constituents: barium, cobalt, manganese, and thallium.

risk-driving constituents are described in Appendix J (which contains ecotoxicity profiles for each constituent). However, only a brief review of the uncertainty in uptake values is presented here. In the case of metals, measured values found in the literature were used to generate high-end estimates of bioaccumulation. The uncertainties related to bioavailability, duration of exposure, and life stage exposed can highly influence the actual versus predicted accumulation. Because only the high-end value was used, this one value does not represent the range and variability this parameter presents at the national scale. A brief discussion of uncertainty in the BAFs for metals is reviewed below.

Lead - In the freshwater ecosystem, the database for lead uptake factors in fish was the most limited compared to other constituents indicating exceedance. One BAF value was identified to characterize the uptake of lead. Applying this value introduces some uncertainty into the analysis in that high-end conservatism could not be confirmed without a distribution of values. In terrestrial ecosystems, the uptake factors in earthworms were characterized by 20 studies, which provided better resolution to assess the uptake factor variability. From these 20 studies, the 90th percentile value was selected as the BAF. Terrestrial plant uptake values were derived from a database of 204 values that represented differences across the variables such as soil chemistry, plant species, and soil depth. There is higher confidence in the terrestrial bioaccumulation factors because the distribution and variability in the data were more represented.

Selenium - In freshwater ecosystems, the uptake factors for fish were also limited by data availability. The BAFs selected for fish were pulled from one study reporting six different BAFs across trophic level 3 and 4 fish. Although differences were seen across trophic levels of fish, the lack of comparable studies increased the uncertainty in these uptake values. In terrestrial ecosystems, a similar database limitation was evident in characterizing the uptake of selenium in earthworms. One study reporting 14 observations was used to derive earthworm BAFs. Terrestrial plant uptake values were derived from a database of 237 values that represented differences across the variables such as soil chemistry, plant species, and soil depth. High-end values were selected in all cases; however, the lack of data did not allow the variability of this parameter to be assessed on a national scale.

Mercury - In freshwater ecosystems, uptake factors for methylmercury were adopted directly from the *Mercury Study Report to Congress* (U.S. EPA, 1997). Relative to other constituents indicating exceedance, the variability in mercury BAFs was well represented in both freshwater and terrestrial ecosystems. In the freshwater ecosystem, the MRTC conducted a Monte Carlo analysis to characterize the variability in BAFs in both trophic level 3 and 4 fish. The uptake factors were derived from field studies. A large source of variability identified in the uptake values was correlated with fish size and fish age. To remain consistent with the methods and recommendations of the MRTC, the geometric means of the methylmercury BAFs were used instead of the high-end values.

In terrestrial ecosystems, uptake factors for worms were characterized by five studies reporting 30 observations. The uptake factors for the terrestrial ecosystem were based on total mercury concentrations. High-end (i.e., 90th percentile values) were applied to determine exposures to terrestrial receptors preying on invertebrates. Uptake data for total mercury in plants were not identified.

Steps 3a and 3b Deriving Criteria from Benchmark Doses for Metals: The criteria were derived using Equations 9-2 and 9-3 for freshwater and terrestrial ecosystems, respectively.

$$\text{Criteria}_w = \frac{\text{benchmark}_w \times \text{bw}_w}{I_w + I_f \sum(F_j \times \text{BAF} \times \text{AB}_j)} \qquad (9\text{-}2)$$

where

Criteria_w	=	protective freshwater criteria (surface water concentration) for mammals and birds (mg/L) (Table 9-9)
benchmark_w	=	calculated benchmark for wildlife species (mg/kg-d) (Table 9-3)
bw_w	=	body weight of wildlife species (kg) (Table 9-5)
I_w	=	intake rate of water (L/d) (Table 9-5)
I_f	=	intake rate of food (kg-d) (Table 9-5)
F_j	=	dietary fraction of prey species j (unitless) (Table 9-5)
BAF	=	bioaccumulation factor in prey species j (unitless) (Table 9-7)
AB_j	=	fraction of constituent absorbed in gut of predator (assumed to be 1).

$$\text{Criteria}_w = \frac{\text{benchmark}_w \times \text{bw}_w}{I_f \sum(F_j \times \text{BAF} \times \text{AB}_j)} \qquad (9\text{-}3)$$

where

Criteria_w	=	protective terrestrial (soil concentration) for mammals and birds (mg/kg soil) (Table 9-10)
benchmark_w	=	calculated benchmark for wildlife species (mg/kg-d) (Table 9-3)
bw_w	=	body weight of wildlife species (kg) (Table 9-6)
I_f	=	intake rate of food (kg-d) (Table 9-6)
Fj	=	dietary fraction of prey species j (unitless) (Table 9-6)

Table 9-9. Total Surface Water Concentrations (mg/L) Corresponding to NOAELs for Representative Receptors of Freshwater Ecosystems

(Sediment Concentrations in mg/kg and Total Surface Water Concentrations in mg/L)

	Mink		Bald Eagle		Great Blue Heron		Lesser Scaup		Spotted Sandpiper	
Constituent	NOAEL	NOAEL	NOAEL	NOAEL	NOAEL	NOAEL	NOAEL	NOAEL	NOAEL	NOAEL
Antimony		7.0E-01		no benmrk		no benmrk		no benmrk		no benmrk
	6.6E+00		5.2E-02		3.9E-02		5.6E-01		4.3E-01	
Barium		no benmrk		2.1E+02		2.1E+02		1.8E+02		2.1E+02
	no benmrk		no benmrk		no benmrk		no benmrk		no benmrk	
Cadmium		1.1E-02		2.5E-02		2.7E+01		1.9E-02		3.0E-02
	6.0E+00		7.0E+00		5.6E+00		1.8E+01		1.4E+01	
Cobalt		no benmrk		no benmrk		no benmrk		no benmrk		no benmrk
	9.1E+01		8.0E+02		7.2E+02		6.9E+02		5.4E+02	
Lead		3.0E-04		1.2E-03		2.2E-01		9.0E-04		1.4E-03
	no benmrk		no benmrk		no benmrk		no benmrk		no benmrk	
Mercury[a]	7.3E-07		1.3E-06		no benmrk		no benmrk		no benmrk	
Methylmercury		4.2E-08		8.2E-08		no benmrk		3.3E-08		no benmrk
	1.6E+02		4.0E+02		3.1E+02		1.2E+03		9.7E+02	
Selenium		2.6E-04		4.4E-03		8.6E+00		3.4E-03		5.2E-03
	no benmrk		no benmrk		no benmrk		no benmrk		no benmrk	
Thallium		no benmrk		no benmrk		no benmrk		no benmrk		no benmrk

Mercury based on total dissolved water concentrations. Derived from dividing methylmercury criteria by 0.078.

2,3,78-TCDD not included in table because methodology differed for this constituent. Dose TEQs were compared to benchmark

Table 9-10. Soil Concentrations Corresponding to NOAELs for Representative Receptors of Terrestrial Ecosystems (mg/kg)

Constituent	Meadow vole	Eastern cottontail	White-Tailed deer	Northern Bobwhite	Short-Tailed Shrew	Deer Mouse	Red Fox	Raccoon	Red-Tailed Hawk	American Kestrel	American Robin	American Woodcock
Antimony	2.9E+01	1.9E+01	no benmrk	no benmrk	5.1E+01	7.2E+01	3.6E+01	1.4E+01	no benmrk	no benmrk	no benmrk	no benmrk
Arsenic	1.1E+03	6.9E+02	2.4E+03	6.8E+00	1.9E+03	2.7E+03	1.3E+03	5.2E+02	3.3E+01	1.9E+01	5.1E+00	7.3E-01
Barium	no benmrk/BCF	no benmrk/BCF	no benmrk/BCF	2.6E+03	no benmrk/BCF	no benmrk/BCF	no benmrk/BCF	no benmrk/BCF	1.2E+04	7.0E+03	1.9E+03	2.4E+02
Beryllium	no benmrk/BCF	no benmrk/BCF	no benmrk/BCF	no benmrk	no benmrk/BCF	no benmrk/BCF	no benmrk/BCF	no benmrk/BCF	no benmrk/BCF	no benmrk/BCF	no benmrk/BCF	no benmrk/BCF
Cadmium	1.4E+00	2.5E+00	2.8E+00	1.0E+01	3.3E+00	7.0E+00	3.3E+01	8.9E+00	1.6E+03	5.3E+02	6.5E+00	1.6E+00
Chromium	3.6E+02	2.3E+02	8.1E+02	2.2E+02	6.4E+02	7.7E+02	4.2E+02	1.7E+02	9.9E+02	3.1E+02	1.7E+02	1.9E+01
Cobalt	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk
Copper	1.7E+03	1.0E+03	3.6E+03	8.4E+03	2.9E+03	4.1E+03	2.0E+03	8.0E+02	4.1E+04	1.6E+04	6.3E+03	9.1E+02
Lead	9.1E-01	6.1E-01	2.0E+00	2.7E+00	8.4E-01	2.0E+00	1.1E+00	4.7E-01	1.0E+01	3.6E+00	1.9E+00	1.6E-01
Manganese	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk
Mercury	8.0E+01	5.0E+01	1.7E+02	1.4E+00	1.4E+02	2.0E+02	9.7E+01	3.8E+01	6.8E+00	2.6E+00	1.0E+00	1.5E-01
Methylmercury	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk
Nickel	2.8E+02	4.2E+02	4.8E+02	1.7E+03	1.3E+03	1.2E+03	3.9E+03	1.3E+03	7.1E+04	2.4E+04	1.1E+03	6.7E+02
Selenium	4.4E+01	2.8E+01	9.6E+01	1.1E+02	7.7E+01	1.1E+02	5.3E+01	2.1E+01	5.2E+02	2.0E+02	8.0E+01	1.1E+01
Silver	4.4E+01	2.8E+01	9.6E+01	1.1E+02	7.7E+01	1.1E+02	5.3E+01	2.1E+01	5.2E+02	2.0E+02	8.0E+01	1.1E+01
Thallium	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk	no benmrk

BAF = bioaccumulation factor in prey species j (unitless) (Table 9-8)

AB_j = absorption of chemical from food j (assumed to be 1).

The scaled benchmark values and BCFs derived in Steps 1 and 2 (Tables 9-3, 9-7, and 9-8) as well as the life history data (Tables 9-5 and 9-6) were used to estimate typical wildlife exposures. Models proposed by EPA (U.S. EPA, 1993b) were used to estimate oral exposures for this analysis. The models sum exposures via contaminated food, water, and soil ingestion; however, ingestion of contaminated prey is the driving exposure route in oral ingestion. Since criteria are derived for multiple representative receptors, the lowest criteria for each taxa group was selected for risk estimation. The media concentrations derived from these calculations are compared to other freshwater and terrestrial criteria (e.g., freshwater community criteria, algae and aquatic plant criteria) identified in Section 9.2.2.2, and the lowest criterion among each media type (i.e., surface water, soil, and sediment) is selected to calculate the potential for risk.

Estimating Benchmark Doses for Dioxin.

Step 1. Scale benchmark: This step was conducted using the same methods and equations outlined for metal constituents (see Equation 9-1). The same issues related to avian cross-species scaling arise when extrapolating test species doses to predict protective wildlife doses.

Step 2. Identify BAFs/BSAFs: To estimate exposure through ingestion of contaminated media, bioaccumulation in prey is a necessary parameter. Bioaccumulation potential in freshwater and terrestrial systems was assessed using different methods. In the terrestrial ecosystem, BAFs were identified directly from measured tissue data for worms, invertebrates, vertebrates, and plants. In the freshwater ecosystem, problems in calculating a bioconcentration factor (BCF)/BAF occur because TCDD can bioaccumulate in fish even though concentrations of TCDD in the water column fall below detection. Given these limitations, the accuracy of TCDD measurement and BAF estimation, using surface water concentrations, may not adequately represent actual bioaccumulation. However, extremely hydrophobic constituents, such as dioxin congeners, can be measured in sediments and aquatic life. Because these chemicals tend to partition to lipids in the organism and organic carbon in the sediment, a biological uptake factor that reflects the relationship between sediment concentrations and organism concentrations is more appropriate. Consequently, biota-sediment accumulation factors are the preferred metric for estimating the accumulation of dioxin congeners (see Section 5.4.1.6). Concentrations in sediment are more readily measured at detectable levels and can be used to determine BSAFs in freshwater species. When partitioning of constituents between sediment particles, pore water, and surface water is accounted for, good correlation between BSAFs and surface-water-derived BAFs is noted (U.S. EPA, 1993a). Several sources were identified to derive BSAF values representative of fish across the nation. BSAFs in [mg congener/kg LP]/[mg congener/kg sediment OC] were calculated from measured data collected by the State of Connecticut Department of Environmental Protection (CT DEP). The specific methods used to calculate BSAFs and the rationale for using the CT DEP BSAFs are discussed in Section 5.4.1.6.

Steps 3a and 3b. Deriving Protective Benchmark Doses for Dioxin and Furan Congeners: To determine a dioxin benchmark protective of food chain exposures to mammals and birds, a

tissue-based approach using TEqCs was implemented. The rationale for implementing a tissue-based TEqC approach is that:

- The tissue-based approach is more scientifically defensible than a water quality approach.
- The development of tissue-based TEqCs is supported by U.S. EPA (1993a, 1995c).
- It is consistent with the TEqC approach used to evaluate human health risks from fish ingestion.

The approach assessing risk based on a mixture of dioxin and furan congeners is supported by the following observations: (1) dioxins and furans predominantly occur in the environment as mixtures; therefore, it is more likely that a receptor will be exposed to multiple congeners, and (2) dioxin-like substance act via similar modes of action. Therefore, varying degrees of similar effects are noted upon exposure to mixtures.

In step 3a, conger-specific TEqCs, measured as fish tissue concentrations, were calculated using Equation 9-4.

$$TEqC_{Tl} = \sum[(C_{sed})_i \cdot (BSAF_l)_{i,T} \cdot (TEF)_{i,j}] \qquad (9\text{-}4)$$

where

$TEqC_{Tl}$ = lipid-based toxicity equivalent concentration in trophic level T fish (mg/kg)

$C_{sed\ i}$ = concentration of congener i normalized for organic carbon in sediment (mg/kg)

$BSAF_{l\ i,T}$ = lipid-based biota-sediment bioaccumulation factor for congener i in trophic level T fish (kg/kg_l)

$TEF_{i,j}$ = toxicity equivalency factor for congener i, biota group j (unitless) Table 9-11 (personal communication, L. Birnbaum, U.S. EPA, Washington, DC)

The TEqCs generated by Equation 9-4 were then used to calculate concentration doses ($Dose_{TEqCs}$) for different receptors using Equation 9-5 in step 3b.

$$Dose_{TEqC} = \frac{I_{receptor}\ [(TEqC_l \cdot flip_{T3fish} \cdot f_{T3fish}) + (TEqC_l \cdot flip_{T4fish} \cdot f_{T4fish})]}{BW_{receptor}} \qquad (9\text{-}5)$$

Table 9-11. Toxicity Equivalency Factors for Ecological Receptors

Congener	Mammals	Birds
HpCDD 1,2, 3, 4, 6, 7,8-	0.01	<0.001
HpCDF 1, 2, 3, 4, 7, 8, 9-	0.01	0.01
HpCDF 1, 2, 3, 4, 6, 7, 8-	0.01	0.01
HxCDD 1, 2, 3, 6, 7, 8-	0.1	0.01
HxCDD 1, 2, 3, 7, 8, 9-	0.1	0.1
HxCDD 1, 2, 3, 4, 7, 8-	0.1	0.05
HxCDF 1, 2, 3, 6, 7, 8-	0.1	0.1
HxCDF 1,2, 3, 4, 7, 8-	0.1	0.1
HxCDF 1, 2, 3, 7, 8, 9-	0.1	0.1
HxCDF 2, 3, 4, 6, 7, 8-	0.1	0.1
OCDD	0.0001	0.0001
OCDF	0.0001	0.0001
PeCDD 1, 2, 3, 7, 8-	1	1
PeCDF 2, 3, 4, 7, 8-	0.5	1
PeCDF 1, 2, 3, 7, 8-	0.05	0.1
TCDD 2, 3, 7, 8-	1	1
TCDF 2, 3, 7, 8-	0.1	1

where

$Dose_{TEqC}$ = average daily TEqC dose from ingestion of fish (mg/kg-d)
$I_{receptor}$ = daily fish intake for the receptor (kg fish/d)
$TEqC_{T3l}$ = lipid-based concentration in fish (mg/kg_l) (Equation 9-4)
$flip_{T3fish}$ = fraction of lipid in T3 fish (kg_l/kg fish)
$flip_{T4fish}$ = fraction of lipid in T4 fish (kg_l/kg fish)
f_{T3fish} = fraction of T3 fish consumed by receptor (unitless)
f_{T4fish} = fraction of T4 fish consumed by receptor (unitless)
BW_{eagle} = body weight of the receptor (kg).

Trophic level specificity was not provided in the BSAF data, but, when considering the exposure to representative receptors, generalized fish BSAFs were normalized to the appropriate trophic level lipid content in Equation 9-5. Lipid fractions (i.e., flip $_{T3fish}$ and flip $_{T4fish}$) in fish were

assumed to approximate predictions provided in the GLWQI of 6.5 percent and 10.3 percent lipid for T3 and T4 fish, respectively (U.S. EPA, 1995a). For this analysis, trophic levels 3 and 4 fish are assumed to accumulate dioxin compounds at similar rates because data did not differentiate between fish trophic levels. However, typically under field conditions, fish usually accumulate dioxin compounds at different rates and to different degrees. This simplification introduces uncertainty in predicting the potential exposure of aquatic wildlife via the food chain.

The $Dose_{TEqC}$, calculated using Equation 9-5, was then compared directly to the receptor-specific ecotoxicological benchmark (mg/kg-d) for 2,3,7,8-TCDD to calculate a hazard quotient for dioxin equivalents.

Mammals and birds characteristic of the terrestrial ecosystems were assessed using analogous methods as those implemented in the freshwater ecosystem, except that (1) representative receptors and prey differed, (2) BAFs were not developed on a lipid basis, and (3) bioaccumulation equivalency factors (BEFs) were unavailable for terrestrial prey items. Equations 9-4 and 9-5 were modified to Equations 9-6 and 9-7 for terrestrial systems.

$$TEqC = \sum[(C_{soil})_i \cdot (BAF_l)_i \cdot (TEF)_{i,j}] \qquad (9\text{-}6)$$

where

TeqC	=	toxicity equivalent concentration in terrestrial prey item (mg/kg)
$C_{soil\ i}$	=	concentration of congener i normalized for organic carbon in soil (mg/kg)
BAF_i	=	biota-sediment bioaccumulation factor for terrestrial prey item congener i (kg/kg_l)
$TEF_{i,j}$	=	toxicity equivalency factor for congener i, biota group j (unitless) Table 9-3

$$Dose_{TEqC} = \frac{I_{receptor} \sum[TEqC_i \cdot f_{prey}]}{BW_{receptor}} \qquad (9\text{-}7)$$

where

$Dose_{TEqC}$	=	average daily TEqC dose from ingestion of contaminated prey (mg/kg-d)
$I_{receptor}$	=	daily prey intake for the receptor (kg-d)
TEqC	=	toxicity equivalent concentration in prey (mg/kg)
f_{prey}	=	fraction of prey consumed by receptor as a fraction of whole diet (unitless)
$BW_{receptor}$	=	body weight of the receptor (kg).

There were several issues of uncertainty in deriving the dioxin criteria for mammals and birds. The key sources of uncertainty discussed here were specifically related to database uncertainty, BSAF uncertainty, and TEF uncertainty.

Database Uncertainty

Regional Specific Data: The database used to develop the BSAFs was adopted from work done by the CT DEP. Uncertainty is introduced by using these data because they were collected from one regional area. There is uncertainty associated with applying these data to represent the uptake of dioxin congeners in fish at the national level. Variables such as lipid content and organic carbon will vary across different regions and waterbodies. However, since BSAFs are purposely normalized for lipids and organic carbon, this should not be a limitation of using the data.

Pooled Data: The documents identified that reported the cumulative data from the CT DEP study pooled site media concentration data for congeners (with the exception of three congeners) in the soil, sediment, and fish tissues. This limited the ability to truly characterize the nature of contaminant uptake in fish using site-specific lipid contents, sediment organic carbon, and fish tissue concentrations. Data pooling generated uncertainty by prohibiting the characterization of the variability associated with the uptake of congeners into fish tissues on a site-specific basis.

Measurement Results: Two specific areas of uncertainty were indicated in the results: outliers and nondetection estimates. The CT DEP database generated some values that were inconsistent with trends seen for most congeners in the database (i.e., mean fish concentrations were significantly higher in preoperational conditions than in those reported during operational conditions) (see Section 5.4.1.6). Because there is no reasonable explanation for this observation, the pre-operational data were not included in the development of BSAFs for two congeners (i.e., 1,2,3,4,7,8- HxCDF and 1,2,3,7,8,9-HxCDF). For these congeners, only mean fish tissue concentrations collected during operational conditions were used. By not using preoperational values in calculating the BSAFs, some uncertainty in BSAF development is generated. By eliminating these values from the data set, potential high-end exposures may not be characterized fully in the results. Second, measurements of dioxin concentrations in the ecological media (i.e., soil, sediment, and fish tissue) sometimes fell below the level of detection. In these cases, the concentration was reported at one-half of the detection level. This assumption may underpredict or overpredict actual concentrations in the media depending on the overall distribution. Further, because the data set had many nondetection measurements, it artificially creates a skewed concentration data distribution for some congeners, which introduces uncertainty into the estimation of mean and median values used in the HWC analysis.

BSAF Uncertainty

Equilibrium partitioning: In calculating BSAFs, equilibrium between sediment concentrations and fish tissue concentrations is assumed. Considering the duration of the study (i.e., 4 years), these concentrations were probably closer to equilibrium than other studies conducted over shorter durations that were considered for BSAF derivation. However, since

continued loading was occurring to the waterbodies over the 4 years of sampling, equilibrium conditions in these waterbodies cannot be confirmed. The disequilibrium conditions introduce a level of uncertainty into the calculated BSAFs.

Trophic level: BSAFs vary depending on the trophic level of the fish. The pooling of fish data did not distinguish between fish trophic levels; therefore, only one generalized fish BSAF could be derived. The lack of characterization by trophic level introduces a level of uncertainty into BSAF metrics.

TEF Uncertainty

Toxicity equivalency factors: TEFs are derived by comparing the toxicity response of like species upon exposure to different dioxin congeners relative to 2,3,7,8-TCDD. Most dioxin and furan congeners are equally or less toxic than 2,3,7,8-TCDD, and, therefore, the TEF for 2,3,7,8-TCDD is 1. TEFs have been derived for mammals and birds; however, there are several issues of uncertainty in applying these TEFs. Two major uncertainties have been identified: (1) TEFs are based on the assumption that the effects of dioxin and furan congeners are additive, and they do not consider possible synergistic or antagonistic relationships between various congeners; and (2) TEFs do not account for pharmokinetics within the organism, which can influence the dose (i.e., the change in mixture composition related to elimination and in vivo transformation of congeners) . In other words, it is assumed that there is no change in the mixture composition from initial uptake to the site-of-action. The observation that metabolism plays a large part in the dose-response relationship makes this intrinsic assumption to applying TEFs an uncertainty in this analysis that may underestimate or overestimate the potential for adverse effects.

Taxa-specific TEFs: As mentioned previously, TEFs have been developed for only the broad categories of mammals and birds; however, even within these categories, interspecies variability in responses to exposure can differ by up to 3 orders of magnitude. For example, the toxicity responses of guinea pigs and hamsters induced by exposure to dioxin mixtures can differ by 1,000 (Kociba and Cabey, 1985). Further, TEFs are not specific to the lifestage of the receptor. Toxic responses are highly influenced by the age of the organism being exposed. The data available do not yet support the development of TEFs at this level of resolution; however, the uncertainty associated with assuming that one TEF represents all mammals generates some uncertainty in the exposure estimates.

9.2.2.2 Criteria Development for Communities. The final step in the development of the stressor-response profile was to derive criteria protective of ecological receptors exposed directly to constituents rather than through the food web. In this section, the methods used to develop surface water, sediment, and soil criteria for aquatic and terrestrial communities are outlined. For populations of the **freshwater** community (e.g., fish, aquatic invertebrates), the final chronic value (FCV) developed for the National Ambient Water Quality Criteria (NAWQC) was preferred as the toxicological benchmark. If an FCV was unavailable and could not be calculated from available data, a secondary chronic value (SCV) was estimated using methods developed for wildlife criteria estimated for the Great Lakes Initiative (e.g., 58 FR 20802). The SCV methodology is based on the original species data set established for the NAWQC; however, it requires fewer data points and includes statistically derived UFs.

Adverse effects levels for fish and invertebrates were identified for endpoints ranging from mortality to growth and reproductive effects. The minimum data set established for fish and aquatic invertebrates was based on the Tier II guidelines proposed in the *Final Water Quality Guidance for the Great Lakes System* (60 FR 15366-15425). The Tier II guidelines establish a procedure to calculate an SCV when data are insufficient to estimate an FCV, as described in the *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses* (Stephan et al., 1985). These guidelines require acute toxicity data representing eight taxonomic families (e.g., a fish from the family salmonids) (see text box) and chronic toxicity data for at least three of the eight families, including an acutely sensitive freshwater species. In contrast, the Tier II methods require data on only one of the eight genera and are based on a statistical analysis of NAWQC data conducted by Host et al. (1991). The authors developed adjustment factors (AFs) (Table 9-12) for varying degrees of data availability (i.e., one to seven data requirements) and a default acute-to-chronic ratio (ACR) to ensure that the SCVs would be below the FCVs within a specified confidence limit. The difference between calculating an FCV and an SCV is summarized as follows.

Data Requirements for FCV Calculation

- The family Salmonidae in the class Osteichthyes
- One other family (preferably a commercially or recreationally important warmwater species) in the class Osteichthyes (e.g., bluegill, channel catfish)
- A third family in the phylum Chordata (e.g., fish, amphibian)
- A planktonic crustacean (e.g., a cladoceran, copepod)
- A benthic crustacean (e.g., ostracod, isopod, amphipod)
- An insect (e.g., mayfly, dragonfly, damselfly, stonefly, midge)
- A family in a phylum other than Arthropoda or Chordata (e.g., Rotifera, Annelida, Mollusca)
- A family in any order of insect or any phylum not already presented

An FCV is calculated in one of two ways. If acceptable chronic toxicity data are available on at least one species representing each of the eight different requirements, the FCV is essentially the concentration corresponding to a cumulative probability of 0.05 for the appropriate species. If the chronic toxicity data do not meet the eight genera requirements, the FCV is calculated by: (1) calculating a final acute value (FAV) in the same manner described for chronic toxicity data, (2) estimating an ACR as the ratio of at least three comparable (e.g., same species) acute and chronic toxicity studies, (3) dividing the FAV by two, and (4) dividing that value by the ACR. It is important to note that this description is a simplification of the actual methods and does not address many of the nuances of study selection and data interpretation.

Table 9-12. Adjustment Factors (Daphnid Data Required)

Sample Size (number of FCV data requirements fulfilled)						
1	2	3	4	5	6	7
21.9	13.0	8.0	7.0	6.1	5.2	4.3

For example, if multiple chronic studies are available on the same species, the geometric mean (i.e., the species mean chronic value, or SMCV) is calculated because the distribution of sensitivities of individual species within a genus are more likely to be lognormal than normal (Stephan et al., 1985).

An SCV is calculated in essentially the same way. However, because the minimum data set only requires data from one to seven genera, the SCV is always calculated from a secondary acute value (SAV). The SAV is calculated in the same way as the FAV and divided by the adjustment factor appropriate to the data set. For example, if the SAV was based on four data points as required in the box on page 9-31 (e.g., fish, salmonid, planktonic crustacean, and insect toxicity data), the SAV value is divided by 7.0, as shown in Table 9-13. The adjustment factors are statistically derived to ensure that the SAV is always lower than the FAV. This value is then divided by an ACR or the default ACR of 18 to estimate the SCV. The Tier II methodology was designed to generate SCVs that are below FCVs (for a complete data set) with a 95 percent confidence limit. For this analysis, the minimum data set required at least one data point for daphnids.

For the **sediment community**, the approach used to establish toxicological benchmarks was based on a complete assessment of several sources proposing sediment benchmark values and outlined in the text box. The premier sources of field sediment data are the National Oceanic and Atmospheric Administration (NOAA) and the Florida Department of Environmental Protection (FDEP) sediment criteria documents. NOAA annually collects and analyzes sediment samples from sites located in coastal marine and estuarine environments throughout the United States as part of the National Status and Trends Program (NSTP). Data measured in the NOAA studies include measures of toxicity of in situ species such as amphipods, arthropods, and bivalves on a variety of community-based endpoints (e.g., abundance, mortality, species composition, and species richness). These data are used by NOAA to estimate the 10th percentile effects concentration (ER-L) and a median effects concentration (ER-M) for adverse effects in the sediment community. These values are not NOAA standards; rather, they are used to rank sites based on the potential for adverse ecological effects. In contrast, the FDEP sediment criteria are developed from the ER-L and ER-M data to approximate a probable effects level (PEL) (estimated from ER-M data) and a threshold effects level (TEL) (estimated from ER-L data). PELs and TELs correspond to the upper limit of contaminated sediment concentrations that demonstrate probable effects and no effects to the benthic community, respectively. Generally, FDEP values are more conservative than NOAA values. From the data evaluated, the lowest of

Sources Evaluated for Developing Benthic Community Criteria

- *Approach to the Assessment of Sediment Quality in Florida Coastal Waters, Volume 1: Development and Evaluation of Sediment Quality Assessment Guidelines*. Florida Department of Environmental Protection. (MacDonald, 1994).
- Potential for Biological Effects of Sediment-Sorbed Contaminants Tested in the National Status and Trends Program Technical Memorandum NOS OMA 52
- National Oceanic and Atmospheric Administration (Long and Morgan, 1991).
- Oak Ridge National Laboratory. *Screening Benchmarks for Ecological Risk Assessment*. U.S. Department of Energy (DOE). (ORNL, 1996).

Table 9-13. Ecotoxicological Criteria Benchmarks for Ecological Receptors in General Aquatic and Terrestrial Ecosystems[a]

						Representative Species in Freshwater Ecosystems (mg/L)		Representative Species in Terrestrial Ecosystems (mg/kg soil)	
	Aquatic Community (mg/L)	**Algae (mg/L)**	**Soil Community (mg/kg soil)**	**Terrestrial Plants (mg/kg soil)**	**Benthic Community (mg/kg sediment)**	**Mammals**	**Birds**	**Mammals**	**Birds**
Antimony	3.0E-02(*p*)	6.1E-01(*i*)	ID	ID	2.0E+00(*i*)	7.0E-01(*a*)	ID	1.4E+01(*a*)	ID
Arsenic^{5+}	8.1E-03(*i*)	4.8E-02(*i*)	6.0E+01(*i*)	1.0E+01(*p*)	7.2E+00(*a*)	3.3E+00(*a*)	2.9E-02(*a*)	5.2E+02(*a*)	7.3E-01 (BB)(*a*)
Barium	3.9E-03(*i*)	ID	3.0E+03(*i*)	5.0E+02(*i*)	ID	ID	1.8E+02(*a*)	ID	2.4E+02(*a*)
Beryllium	6.6E-04(*i*)	1.0E+02(*i*)	ID	ID	ID	ID	ID	ID	ID
Cadmium	1.4E-03(*a*)	2.0E-03(*i*)	1.0E+00(*p*)	3.0E+00(*p*)	6.8E-01(*a*)	1.1E-02(*a*)	1.9E-02(*a*)	1.4E+00(*a*)	1.6E+00(*a*)
Chromium^{6+}	1.1E-02(*a*)	2.0E-03(*i*)	6.4E+01(*i*)	1.0E+00(BB)(*i*)	5.2E+01(*a*)	4.5E+00(*p*)	4.1E+00(*a*)	1.7E+02(*p*)	1.9E+01(BB)(*a*)
Chromium^{3+}	4.9E-02(*a*)	ID	6.4E+01(*i*)	1.0E+00(BB)(*i*)	5.2E+01(*a*)	ID	ID	ID	ID
Cobalt	2.3E-02(*i*)	ID	ID	ID	ID	ID	ID	ID	ID
Copper	5.1E-03(*a*)	1.0E-03(*i*)	2.1E+01(*p*)	1.0E+02(*i*)	1.9E+01(*a*)	4.0E+01(*a*)	5.9E+02(*a*)	8.0E+02(*a*)	9.1E+02(*a*)
Lead	3.2E-03(*a*)	5.0E-01(*i*)	2.8E+01(*p*)	5.0E+01(*p*)	3.0E+01(*a*)	3.0E-04(*a*)	9.0E-04(*a*)	4.7E-01(BB)(*a*)	1.6E-01(BB)(*a*)
Manganese	1.2E-01(*i*)	ID	ID	5.0E+02(*i*)	ID	ID	ID	ID	ID
Mercury[b]	9.1E-04(*a*)	5.0E+00(*i*)	1.0E-01(*i*)	ID	1.3E-01(*a*)	5.4E-07(*a*)[c]	4.2E-07(*a*)[c]	3.8E+01(*a*)	1.5E-01(*p*)
Methylmercury[b]	2.8E-06(*i*)	ID	ID	ID	ID	4.2E-08(*a*)[c]	3.3E-08(*a*)[c]	ID	ID
Nickel	2.9E-02(*a*)	2.0E-03(*i*)	9.0E+01(*i*)	3.0E+01(*i*)	1.6E+01(*a*)	9.5E+01(*a*)	2.3E+02(*a*)	2.8E+02(*a*)	6.7E+02(*a*)
Selenium	5.0E-03(*a*)	1.0E-01(*i*)	7.0E+01(*i*)	1.0E+00(*i*)	ID	2.6E-04(*a*)	3.4E-03(*a*)	2.1E+01(*a*)	1.1E+01(*a*)
Silver	3.6E-04(*i*)	3.0E-02(*i*)	ID	ID	7.3E-01(*a*)	ID	ID	ID	ID
Thallium	ID	ID	ID	ID	ID	ID	ID	ID	ID
2,3,7,8-TCDD (mg/kg-d)	ID	ID	ID	ID	ID	4.7E-07(*a*) [d]	9.8E-03(*a*) [d]	2.6E-07(*a*) [d]	1.4E-05(*a*)[d]

BB = Below U.S. average background concentrations (see Table 9-15). ID = Insufficient data identified.
[a] Shaded cells represent the selected criterion for risk estimation.
[b] Total dissolved for freshwater criteria for mammals and birds.
[c] Adopted from *Mercury Study Report to Congress* (U.S. EPA, 1997).
[d] Criteria for 2,3,7,8 TCDD-TEQs are in units of dose mg/kg-d.
Note: (*a*) = adequate; (*p*) = provisional; (*i*) = interim. The relative confidence represented by adequate, provisional, and interim categories is expanded in Table 9-14.

all criteria are used for the HWC analysis. Even though these criteria were developed specifically for a marine community, researchers have demonstrated that marine TELs have good correlation with no-effects levels found for freshwater systems (Smith et al., 1996). Below this range, sediment biota are not expected to demonstrate adverse effects. In both criteria documents, the protective level was generated using the ER-L data and the TELs, which provide levels at which low or no adverse effects are expected, offering a conservative level of protection.

For **algae and aquatic plants**, toxicological benchmarks were identified in the open literature or from a data compilation presented in *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision* (Suter and Tsao, 1996). For most contaminants, studies were not available for aquatic vascular plants and lowest effects concentrations were identified for algae. The criteria for algae and aquatic plants were based on (1) a lowest-observed-effects concentration (LOEC) for vascular aquatic plants or (2) an effective concentration (EC_{xx}) for a species of freshwater algae, frequently a species of green algae (e.g., *Selenastrum capricornutum*). Because of the lack of data in this receptor group and the differences between vascular aquatic plants and algae sensitivity, usually the lowest value of those identified was used.

For the **terrestrial plant community**, toxicological benchmarks were identified from a summary document prepared at the Oak Ridge National Laboratory (ORNL): *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1997 Revision* (Efroymson et al., 1997a). The measurement endpoints were generally limited to growth and yield parameters because: (1) they are the most common class of response reported in phytotoxicity studies and, therefore, allow for criterion calculations for a large number of constituents; and (2) they are ecologically significant responses both in terms of plant populations and, by extension, the ability of producers to support higher trophic levels. As presented in Efroymson et al. (1997a), criteria for phytotoxicity were selected by rank ordering the LOEC values and then approximating the 10th percentile. If there were 10 or fewer values for a chemical, the lowest LOEC was used. If there were more than 10 values, the 10th percentile LOEC was used. Such LOECs applied to toxicity endpoints measuring plant growth, yield reduction, or other effects are reasonably assumed to impair the ability of a plant population to sustain itself.

For the **soil community**, criteria were developed using methods analogous to those used in deriving the NAWQC. In brief, the criteria values for soil fauna were estimated to protect 95 percent of the species found in a typical soil community, including earthworms, insects, and other various soil fauna. Microflora were not included in the soil community primarily because of the difficulty in assigning ecological significance to effects levels for soil microorganisms. This introduces some uncertainty in the soil criteria because: (1) microflora make up approximately 80 to 90 percent of the biomass in soil and (2) microflora are responsible for the majority of the biological activity in soil (e.g., N mineralization). However, when data were insufficient for criterion development, criteria studies identifying effects to earthworms and other soil biota proposed by ORNL (Efroymson et al., 1997a) or criteria developed by the Canadian Council of Ministers of the Environment (CCME, 1997) were used to estimate protective soil concentrations.

In developing criteria, eight taxa of soil fauna were identified to capture the key structural (e.g., trophic elements) and functional (e.g., decomposers) components of the soil ecosystem. The methodology presumes that protecting 95 percent of the soil species with a 50th percentile level of confidence ensures long-term sustainability of a functioning soil community. The toxicity data on soil fauna were compiled from several major compendia and supplemented with additional studies identified in the open literature. Generally, the studies were not evaluated in terms of quality because there is currently no consensus on standard methods and species for soil testing (although earthworms are frequently used as indicator species). However, acceptable toxicity data were limited to soil studies (versus aqueous studies) on measurement endpoints believed to be relevant to population survival (e.g., growth, reproduction). In general, insufficient data were identified to delineate the relationship between toxicity and the metal species applied to the soil. Although the process of developing criteria for the generalized soil community is iterative in nature, the approach may be divided into three basic components:

1. **Selection of representative soil species–**Two important assumptions were made in developing the approach to select representative soil species. First, species using resources in a similar way (e.g., similar diet) should receive similar exposures (i.e., guild theory). Second, taxonomically related soil invertebrates tend to have similar toxicological sensitivity to chemicals (Neuhauser et al., 1986).

2. **Collection of toxicological data on soil species–**Guidelines were established to collect data on LOECs for representative species in the soil community. The toxicological data included studies on a variety of relevant physiological and process-based endpoints. Assumed routes of exposure were direct contact and ingestion.

3. **Calculation of criteria for the soil community–**The statistical approach adopted consisted of two steps: (1) fitting the LOEC data on representative species of soil biota to a lognormal distribution, (2) extrapolating to a criterion based on the mean and standard deviation of the toxicity dataset. The key assumptions were that: (1) LOEC data are distributed lognormally, (2) the selection of LOEC data (rather than no observed effects concentration [NOEC] data) is appropriate for this methodology, and (3) the 95 percent level of protection is ecologically significant.

Selection of Representative Soil Species. Soil communities are made up of numerous groups of species performing one or more functions for the community. Thus, the set of "representative" species was designed to reflect the breadth and variety of taxonomic and structural/functional groups. Five metrics were identified to serve as a practical guide in the selection of appropriate soil species. Figure 9-2 illustrates the generalized soil community that is reflected in these metrics.

Five metrics were used to select representative soil species:

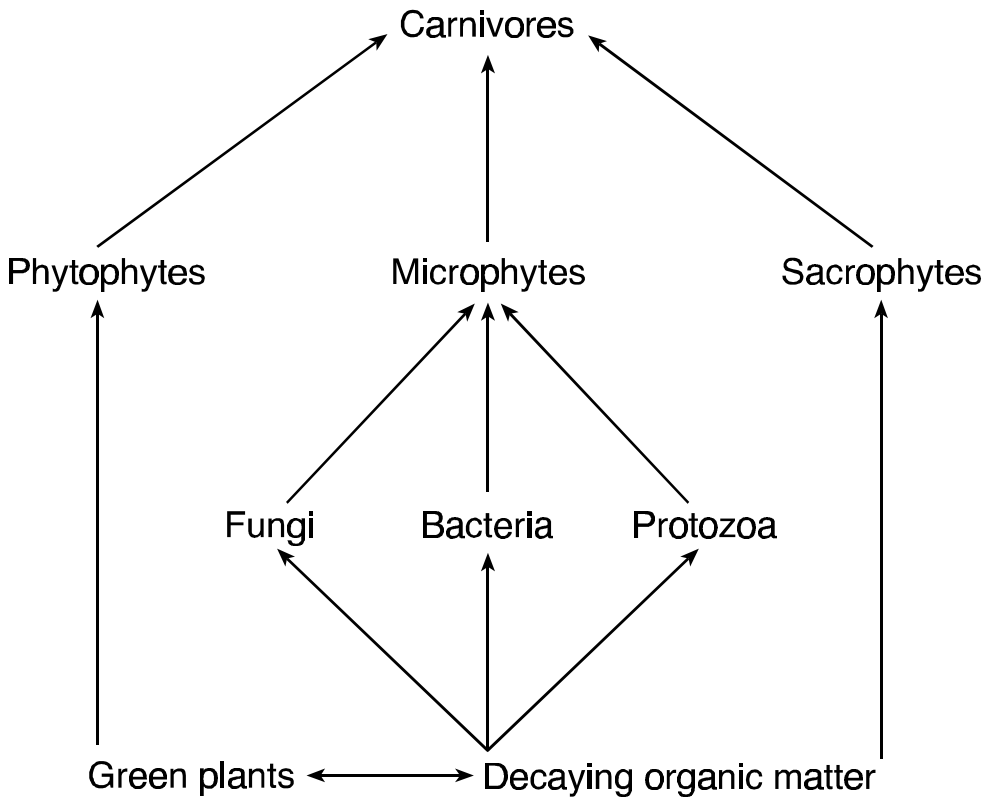

Figure 9-2. Simplified trophic structure of a generalized soil community.

1. *Organism size*–classified into three groups: microfauna (<0.15 mm; e.g., Protozoa, Nematoda), mesofauna (0.16 to 10 mm; e.g., Enchytraeidae, Acari), and macrofauna (>10 mm; i.e., larger invertebrates). This convenient, albeit somewhat arbitrary, classification was useful in considering the interactions between soil species and their habitat.

2. *Distribution in soil horizon*–divided into three layers: deep mineral, shallow organic, and soil litter. Exposures to soil contaminants are presumed to occur for organisms at any horizon. However, the top two horizons tend to receive higher exposures to persistent and relatively immobile contaminants (such as some metals).

3. *Abundance*–number of individuals present in a typical habitat. Caution must be implemented in using this criteria because abundance species are not always the most ecologically significant. For example, nematodes and annelids both contribute equally to the flux of CO_2, yet nematodes outnumber annelids more than 100 to 1 (Reichle, 1977).

4. *Energy metabolism*–relative importance of a species to the overall community can be based on the contribution of energy that species provides (Curry, 1994). Increasingly, energy budgets are being viewed as a useful tool in assessing ecological significance.

5. *Function in community*–feeding preferences of different organisms largely define their role in the trophic structure (see Figure 9-2), shaping the dynamics of the soil community. The selection of species should adequately represent different functional roles within the trophic structure. To ensure a balanced representation of a generalized soil community, organisms were classified into four functional categories (Brown, 1978):

   - Microphytic–organisms that feed on fungal spores, hyphae, lichens, and bacteria (e.g., ants, fungus gnats, nematodes, and protozoa)

   - Saprophytic–organisms that feed on dead or decaying organic matter (e.g., earthworms, acari, and collembola)

   - Phytophagous–organisms that feed on living plant material including plant stems, leaves, roots, or woody parts (e.g., mollusks, symphylids, termites, insect larvae)

   - Carnivorous–organisms that are true predators (e.g., carabids, mites, spiders).

Implementation of metrics to select soil species:

Group 1–one species from the phylum Nematoda. Nematodes are the most abundant organisms in the soil and provide the third largest amount of biomass. In addition, they represent the only microfauna evaluated.

Group 2–one species of soil mite (Acarina) from one of the following suborders: Cryptostigmata, Prostigmata, Mesostigmata, or Metastigmata. Soil mites are important as decomposers, predators, and plant eaters. Mites provide the largest amount of CO_2 flux among these groups.

Group 3–one insect from the order Collembola. Springtails were selected because they are saprophytic and the second most abundant invertebrates in the soil. Their high abundance also results in moderately high biomass.

Groups 4 & 5–two annelids from the orders Plesiopora or Opisthopora (families Enchytraeidea and Lumbricidae preferred). The oligochaeta represent some of the largest soil organisms and, as subterranean animals, are important saprophytic feeders. Members of Opisthopora are the largest contributors to soil fauna biomass.

Groups 6 & 7–two additional species of arthropods selected from one of the following taxonomic groups: Diptera, Coleoptara, Isopoda, Chilopoda, and Diplopoda. Arthropods play a variety of critical roles in the soil community and rank high in terms of all five metrics.

Group 8–a species of mollusc from the order Stylommatophora. Although the majority of molluscs are marine organisms, they represent surface decomposers in the trophic structure that are not duplicated by the other organisms in the representative set.

***Calculation of Criteria for the Soil Community*.** The approach to calculating benchmarks for the soil community was based on efforts by Dutch scientists (the RIVM methodology) to develop hazardous concentrations (HCs) at specified levels of protection (primarily 95 percent) at both a 95th percentile and a 50th percentile level of confidence (Slooff, 1992). For the soil fauna benchmarks, the 50th percentile level of confidence was selected because the 95th percentile appeared to be overly conservative for a no-effects approach. The RIVM methodology follows two steps: (1) fitting a distribution to the log of the selected endpoints and (2) extrapolating to a benchmark concentration based on the mean and standard deviation of a set of endpoints. The key assumptions in the Dutch methodology are that: (1) LOEC data are lognormally distributed, and (2) the 95 percent level of protection is ecologically significant. Equation 9-4 is used to calculate soil fauna benchmarks:

$$HC_{5\%} = [x_m - k_l \; s_m] \qquad (9\text{-}4)$$

where

$HC_{5\%}$ = soil concentration protecting 95 percent of the soil species

x_m = sample mean of the log LOEC data

k_l = extrapolation constant for calculating the one-sided leftmost confidence limit for a 95 percent protection level

s_m = sample standard deviation of the log LOEC data.

It is important to note that only one value for k_l is calculated for the 50th and 95th percentile confidence limits, respectively, for each sample size (*m*). Consequently, it is assumed that: (1) there is just one extrapolation constant with the required confidence property for each species sample size, and (2) extrapolation factors may be determined through Monte Carlo simulation by generating random sample averages and deviations for the **standard** logistic distribution and adjusting for a specified confidence level (i.e., 50th or 95th).

9.2.2.3 <u>Limitations of Data Availability</u>. Criteria could not be calculated using the methods proposed in the previous sections when ecotoxicity data were lacking to evaluate the potential for adverse effects in receptors of concern. The data gaps evident in Table 9-13 generate some uncertainty as to the extent of protection that is provided to the entire ecosystem if only a

few categories of receptors are represented in risk estimates. Ecotoxicological criteria were developed for constituents when sufficient data were available. Because the risk results can be interpreted only within the context of available data, the absence of data should not be construed to indicate that adverse ecological effects will not occur.

Further, the conservatism in the development of criteria also generated uncertainty in the risk estimates. However, the conservatism of criteria development was appropriate for a screening analysis. Because the approach is generally based on "no effects" data, criteria tend to be fairly conservative. In site-based analysis, an approach is often used to allow for a level of effect that is predicted to be below a level of concern for reproducing populations (e.g., a low-effects approach). Since no-effects benchmarks are frequently an order of magnitude below a low-effects benchmark, the level of conservatism built into the ecological benchmarks (in mg/kg-d) is approximately an order of magnitude. The criteria are assessed in Section 9.2.4 to apply a relative confidence ranking based on the quality and quantity of data that were identified.

9.2.3 Ecotoxicological Criteria Selection

This section presents the criteria values used in the HWC analysis, reviews the process of selecting criteria for risk determinations, and assesses the respective confidence in the criteria. The final criteria developed for receptors of concern are presented in Table 9-13. All criteria were examined closely to select the most appropriate criteria for each media (i.e., soil, surface water, and sediment) to use in risk determinations. This process was somewhat simple for the sediment community because only one criterion was developed for this receptor category. However, for surface water and soil, several media concentrations for various receptors are presented when sufficient data were available for criteria development. For soil, criteria (mg/kg soil) for mammals, birds, terrestrial plants, and the soil community are presented; for surface water, criteria (mg/L) for mammals, birds, aquatic plants, and the freshwater community were available. Three steps were taken to select an appropriate criteria for surface water and soil:

1. Confidence ranks were assigned to each criteria. Confidence ranks, as outlined in Table 9-14, indicate the relative confidence between the criteria and were used to assess the subsequent confidence and the uncertainty in the risk estimates. In order of high to low confidence, the criteria are adequate (*a*), provisional (*p*), and interim (*i*). Generally, the ranks differ for each ecological receptor category because varying degrees of data quality and quantity are found across different receptor types. For instance, the soil community criterion assigned a confidence rank based on the quantity of data available for soil organisms as well as on the diversity of species that the data set represents. Confidence ranks are intended to evaluate the pool of available ecotoxicity data for selecting the study that fulfilled the data standards (e.g., dose-response information, adequate quality assurance/quality control , and appropriate endpoints). In a general sense, in a given receptor group, adequate benchmarks are selected over provisional and interim, respectively.

Table 9-14. Confidence Ranks for Criteria by Receptor Taxa

Mammal and Bird Criteria

Adequate—The study value selected was a no-effects level (NEL) based on a reproductive, developmental, growth, or survival endpoint that was lower than any other NEL or low effects level (LEL) for these endpoints. Studies were conducted over chronic or subchronic durations or during a sensitive life stage for the three types of endpoints relevant to the population viability.

Provisional—The study value selected was an LEL/10 (LEL divided by an LOAEL-to-NOAEL safety factor of 10) on a reproductive, developmental, or growth/survival endpoint that was lower than any other NEL or LEL for these endpoints. In addition, the data set contained studies conducted over chronic or subchronic durations or during a sensitive life stage.

Interim—The study value selected was the lowest NEL or LEL/10 on a reproductive, developmental, or growth/survival endpoint. This category did not require studies of the entire suite of endpoints for population sustainability.

Plant Community Criteria

Adequate—No benchmarks were assigned to this category. At present, the phytotoxicity database is very limited, and EPA has not proposed standard protocols to develop toxicological benchmarks for plants. At a minimum, further research is needed on: (1) quantifying the impact of soil characteristics on phytotoxicity, (2) identifying endpoints with high biological significance to plant physiology and toxic response, and (3) selecting species and testing methods (e.g., duration of exposure) to form a core requirement for phytotoxicity benchmarks.

Provisional—The benchmark selected was the 10th percentile of study LOECs that met the criteria described above.

Interim—The benchmark selected was the lowest LOEC presented in Efroymson (1997b) or identified in the open literature.

Algae and Aquatic Plants Criteria

Adequate—No benchmarks were assigned to this category. Test endpoints for effects on algae have been less well standardized and their relevance to the field are less clear than for animals (Lewis, 1990). Relatively few tests of effects on vascular aquatic plants have been conducted, and development of culture techniques, test methods, and sensitive endpoints has been limited (Klaine and Lewis, 1995). Further research is needed to develop more realistic test designs to evaluate the effects on natural algal assemblages and vascular aquatic plant communities.

Provisional—No benchmarks were assigned to this category. A benchmark would have been designated as provisional if the following conditions had been met: (1) the benchmark study provided an LOEC for a vascular aquatic plant estimated from at least two data points ***or*** the lowest EC_{20} value from representative algal species; (2) phytotoxicity studies were available on at least one species of floating macrophytes, one species of submerged aquatic vegetation, and one species of emergent aquatic vegetation; and (3) EC_{20} values were available for at least three of the six algal classes proposed by Swanson et al. (1991), including green and blue-green algae, diatoms, and dinoflagellates.

Interim—All of the benchmarks were assigned to this category. The benchmark selected was the lowest LOEC identified for vascular aquatic plants or the lowest effective concentration (EC_{xx}) identified for a species of freshwater algae.

(continued)

Table 9-14. (continued)

Freshwater Community Criteria

Adequate—The criteria selected was an FCV, in order of preference, from the following sources: (1) an FCV derived for the GLWQI or (2) an FCV from an AWQC document.

Provisional—The criteria selected was a draft FCV, in order of preference, from the following sources: (1) an FCV calculated by the U.S. EPA Environmental Research Laboratory in Duluth, MN, or Narragansett, RI, or (2) an FCV estimated from data extracted from Aquatic Toxicity Information Retrieval Database (AQUIRE) (or literature) meeting the general 1985 guidelines for study selection.

Interim—The criteria selected was an SCV estimated using Tier II methods on data extracted from AQUIRE (or literature) meeting the general 1985 guidelines for study selection. The data set contained at least one usable data point on a daphnid species.

Benthic Community Criteria

Adequate—Criteria were developed from data sets containing at least 100 toxicity values for sediments biota. This level of data was presumed to adequately reflect an array of toxic responses on a variety of benthic species.

Provisional—Criteria were based on data sets containing at least 20 data points. None of the benthic criteria derived fell into this category. Twenty studies should not be considered an absolute threshold, rather, the quality of the data and the toxicity endpoints (e.g., abundance, growth, lethality) of these studies should also be considered.

Interim—Criteria were based on data sets containing less than 20 data points. Twenty studies should not be considered an absolute threshold, rather, the quality of the data and the toxicity endpoints (e.g., abundance, growth, lethality) of these studies should also be considered.

Soil Community Criteria

Adequate—All of the benchmarks assigned to this category fulfilled the eight taxonomic data requirements. For each species, an NOEC or LOEC/10 was identified with sufficient information on soil characteristics to calculate a normalized effects level. Appropriate studies were limited to exposure routes that matched the spatial location of the soil organism.

Provisional—The study data for this category were of equal quality as the adequate category. However, the minimum data set was reduced to five of the eight representative soil species.

Interim—For benchmarks assigned to this category, NOEC and/or LOEC/10 data existed for four representative soil species (Slooff, 1992; Okkerman et al., 1993). More flexibility was assigned to the provisional category and studies were included for a wider range of exposure routes (e.g., dermal application). In addition, when toxicity data on earthworms were available, a criterion was proposed using the lowest toxicity value.

2. Criteria for soil were compared to background concentration averages and ranges reported for the conterminous United States (see Table 9-15). Several of the derived terrestrial criteria fell below background concentrations measured in soils. Values falling below background concentrations were presented but are not considered as appropriate screening values.

3. From the remaining criteria in each media type (i.e., soil and surface water), the lowest criterion that met the greatest number of data standards was selected. If several studies were of adequate rigor to meet data standards, the lower of the values was selected to maintain an appropriate level of conservatism for a screening level analysis. By selecting the lowest value, a level of protection to other receptors that are apparently more tolerant of exposures was inferred.

Criteria for all representative species exposed via the food web were calculated using the methods outlined in Section 9.2.21. However, only the lowest criterion for aquatic mammals, aquatic birds, terrestrial mammals, and terrestrial birds was presented in Table 9-13. For example, the criteria developed for the two mammals characteristic of aquatic habitats, the river otter and the mink, were compared, and the lowest value was selected for inclusion in Table 9-13. The following list provides supplemental information to Table 9-13 to identify where varying methods and receptors were indicated in criteria development:

Surface Water Criteria

- Aquatic community criteria were based on FCVs or draft FCVs derived using NAWQC methods. When insufficient data were available to develop FCVs, SCVs using Tier II methods were adopted for criteria development. Other groups and government programs have proposed FCV and SCV criteria based on NAWQC and Tier II methods, respectively. The criteria developed through these other groups were adopted when methods were consistent with NAWQC and Tier II (e.g., GLWQ1; Oak Ridge National Laboratories).

- Metals criteria and dioxin dose TEQs for mammals in the freshwater ecosystem were based on the river otter as the representative receptor.

- Metals criteria and dioxin dose TEQs for birds in the freshwater ecosystem were based on the kingfisher and bald eagle as the representative receptors, respectively.

Sediment Criteria

- Sediment criteria protective of benthic species were all derived from FDEP work, with the exception of antimony, which was developed by NOAA (Long and Morgan, 1991).

Table 9-15. Background Concentrations of Metals Found in the United States (All soil concentrations in mg/kg soil)

	Background Concentration by Region								
	Conterminous U.S.			Eastern U.S.			Western U.S.		
Constituent	Geo. Mean	Range	Sample Size	Geo. Mean	Range	Sample Size	Geo. Mean	Range	Sample Size
Antimony	0.48	<1.0 - 8.8	354	0.52	<1.0 - 8.8	131	0.47	<1.0 - 2.6	223
Arsenic	5.2	<1.0 - 97	1257	4.8	<1.0 - 73	527	5.5	<1.0 - 97	730
Barium	440	10 - 5000	1319	290	10 - 1500	541	580	70 - 5000	778
Beryllium	0.63	<1.0 - 15	1303	0.55	<1.0 - 70	525	0.68	<1.0 - 15	778
Cadmium	--	--	--	--	--	--	4.3[a]	1.0 - 10	12
Chromium	37	1.0 - 2000	1319	33	1.0 - 1000	541	41	3.0 - 2000	778
Copper	17	<1.0 - 700	1311	13	<1.0 - 700	533	21	2.0 - 300	778
Lead	16	<10 - 700	1319	14	<10 - 300	541	17	<10 - 700	778
Mercury	0.058	<0.01 - 4.6	1267	0.081	<0.01 - 3.4	534	0.046	<0.01 - 4.6	733
Molybdenum	0.59	<3.0 - 15	1298	0.32	<3.0 - 15	524	0.85	<3.0 - 7.0	774
Nickel	13	<5.0 - 700	1318	11	<5.0 - 700	540	15	<5.0 - 700	778
Selenium	0.26	<0.1 - 4.3	1267	0.3	<0.1 - 3.9	534	0.23	<0.1 - 4.3	733
Silver	--	--	--	0.14[b]	<0.22 - 0.49	136	<0.5	0.5 - 1.5	168
Vanadium	58	7.0 - 500	1319	43	<7.0 - 300	541	70	7.0 - 500	778
Zinc	48	<5.0 - 2900	1248	40	50 - 2900	482	55	10 - 2100	1248

Source: Dragun and Chiasson, 1991.

[a]Data from southeastern United States.
[b]Data from Northern Great Plains.

Soil Criteria

- Soil community criteria were derived using the methods presented in this section and Appendix J for cadmium, copper, and lead; soil criteria proposed by ORNL were used for the following constituents: arsenic^{5+}, barium, mercury, nickel, and selenium; for chromium, criteria proposed for earthworms by the CCME were adopted.

- Criteria for mammals in the terrestrial ecosystem were based on the raccoon as the representative receptor with the exception of the meadow vole for cadmium and nickel. Dioxin dose TEQs (mg/kg-d) were based on the white-tailed deer.

- In the terrestrial ecosystem, criteria for birds were based on the American woodcock as the representative receptor. Dioxin dose TEQs were based on the red-tailed hawk.

9.3 Risk Characterization Methods

Ecological risk was estimated according to the methods outlined in EPA's *Guidelines for Ecological Risk Assessment* (U.S. EPA 1998a). Accordingly, risk characterization in this risk assessment is defined in terms of: (1) **risk estimation**, which compares modeled exposure concentrations to the ecotoxicological criteria to identify HQ exceedances[2]; and (2) **risk description**, which synthesizes and interprets the results of that comparison, evaluates the limitations of the screening assessment, and presents the overall conclusions of the assessment. This section focuses on the *methods* used to characterize risk; the final presentation and interpretation of results are discussed in Section 2.

9.3.1 Risk Estimation

In performing the risk estimation, the modeled concentrations generated in the exposure profile in various media were compared with the ecological criteria or benchmark developed in the analysis phase. This process is accomplished by generating an HQ from the ratio of the expected media concentrations in soil, sediment, and water to the criterion developed for the receptors of concern. Media concentrations selected for comparison to receptor criteria take into account the most likely habitat for the receptors. For example, sediment concentrations are used to determine risk for the benthic community, whereas soil concentrations are used to estimate the risk to plants. The risk estimation approach used for dioxin was unique from that used for metals in that the HQ was the ratio of the dose TEQ (mg/kg-d) and the 2,3,7,8-TCDD benchmark dose (mg/kg-d) (see section 9.2 for an expanded discussion). In brief, the dose TEQ is an estimated dose for mammals or birds derived from modeled media concentrations that quantify the additive exposure of all dioxin and furan congeners. The dose TEQ is normalized into 2,3,7,8-TCDD equivalents so that it can be directly compared to a 2,3,7,8-TCDD benchmark.

[2] For discussions on the HQ method, refer to any of the following sources: *Ecological Risk Assessment* (Suter, 1993); *Mercury Study Report to Congress* (U.S. EPA, 1997); and *Guidelines for Ecological Risk Assessment* (U.S. EPA, 1998).

The results of the risk estimation have a binary outcome: either the constituent concentration is above the screening criteria (HQ > 1) or the concentration is below the criteria (HQ ≤ 1). Because the ecotoxicological criteria are based on de minimis ecological effects, it is presumed that an HQ below 1 indicates a low potential for adverse ecological effects for those receptors included in the analysis. The HQ results should not be used to assess the potential for adverse ecological effects on sensitive species and habitats (e.g., endangered species) nor should they be used to explicate the likelihood of effects on receptors not included in the analysis (e.g., insects, bats, reptiles). Therefore, a limitation of a screening analysis of this nature is the exclusion of threatened and endangered species, particularly from the standpoint of evaluating recognized ecological benefits. These comments notwithstanding, HQ results above 1 suggest that the potential for adverse ecological effects exists and that the constituent requires further evaluation. Given the simplicity of the risk screen quotient, several issues of uncertainty arise from assumptions intrinsic to the analysis. The risk estimation results, as presented in Section 2, emphasize uncertainty related to issues of exposure and issues of criteria/benchmark development.

9.3.2 Risk Description

This section provides interpretations of the risk results. Interpreting the impact of a risk exceedance in this analysis requires an interpretation of the biological significance the impact may have on ecosystem function and structure. In some cases, risk exceedance values may have low ecological significance. For example, the spatial distribution of constituents in contaminated media may be a localized phenomenon; therefore, receptors with broad spatial foraging ranges may have minimal exposure if no suitable habitat is located within the area of constituent contamination. Further, the resistence or resilience of a particular receptor to compensate for the loss of an individual species may result in relatively few population impacts. For example, the toxicity endpoint selected for plants, an LOEL, may result in a reduced yield, growth, or lethality in some plant species, but these effects are not likely to result in population losses that will impact the plant community as a whole because of their high resistance and resilience (i.e., high reproductive rate to recover population).

For constituents that fail the ecological screen, the risk characterization investigates the following issues to interpret whether HQ exceedances represent potentially serious ecological effects:

- **Types of receptors**–Ecological receptors have varying levels of intrinsic value to ecosystems. Consequently, it is important to identify the types of ecological receptors for which HQ exceedances are observed. For example, HQ exceedances for raptors may be considered potentially serious because they feed at the top of the food chain and are responsible for maintaining a check on local vermin populations. In contrast, an HQ exceedance for an omnivorous animal such as a raccoon may not have the same level of impact on the structure and function of the ecosystem because most habitats contain a variety of omnivorous species.

- **Number of receptors**–Although the HQ exceedances for each constituent are calculated for only one ecotoxicological criterion per medium of concern, constituent concentrations may exceed de minimis concentrations for more than

one receptor. Because adverse effects to multiple receptors within an ecosystem would be expected to create a higher potential for adverse effects to a community, it is important to identify HQ exceedances for all receptors presumed to occupy a given habitat.

Frequency of exceedances–An HQ exceedance in a small area relative to the total area assessed (given a nationwide sample) may be interpreted as having limited ecological significance because so few habitats would be impacted. Therefore, the frequency with which environmental concentrations of HWC constituent releases result in exposures that are above de minimis levels should be estimated by generating cumulative frequency distributions (CFDs) of HQs across combustor categories for all three media types. For each constituent, a CFD can be generated and evaluated to assess the extent of the potential ecological risks (e.g., HQ > 1 for category-specific sectors at the 0.50, 0.10, 0.05, and 0.01 percentiles).

Magnitude of exceedances–As stated above, HQ values above 1 do not provide estimates of the ecological significance of effects in an absolute sense. However, the level of concern and the importance of recognizing limitations increases directly with the relative magnitude of the HQ exceedances. In short, the magnitudes are used in performing relative comparisons of MACT options and for source categories. To ascertain the magnitude of HQ exceedances, frequency bins may be created (e.g., "areas (km^2) with HQs between 1 and 10" for a given combustor category). Given the low level of resolution of the screening analysis, frequency bins are designated for the following four groups: HQ $\leq$ 1, 1 < HQ$\leq$10, 10 < HQ $\leq$ 100, and 100 < HQ. As with the cumulative frequency distribution tables, separate tables containing data for all constituents are generated for all three media types (soil, surface water, and sediment) for each of the combustor categories.

Spatial character of exceedances–Having characterized the frequency and magnitude of HQ exceedances, the spatial extent of potential ecological risks may be evaluated by generating the number of facilities with predetermined HQ exceedance areas. For example, it is possible that one or two facilities within a facility category may be associated with virtually all of the HQ exceedances. Conversely, the data may indicate that there are exceedances across several facilities within a facility category, indicating that the potential for adverse ecological effects may be at a broad scale. The spatial data do not identify specific facilities–only the total number of facilities with an HQ exceedance rate greater than the value presented is given. The area exceedance value bins included in the tables are: 0-314, >314-628,> 628-942, and > 942-1258 km^2. As with the previous two categories of data analyses, tables are generated for all three media for each of the combustor categories.

Background concentrations–The metal constituents modeled in this analysis are found as naturally occurring elements throughout the contiguous United States. Consequently, it is crucial that ecotoxicological criteria be compared to

background concentrations to determine whether the criteria may reasonably be applied to evaluate ecological risks. Background concentrations, defined as low levels of constituents found in environmental media resulting from natural processes (e.g., mineral weathering), measured across the United States were identified in Dragun and Chiasson (1991). (Note: This comparison to background concentrations should not be confused with other background comparisons used in this analysis to assess the benefits of the MACT options.) Interpreting the significance of HQ exceedances for ecotoxicological criteria that fall within background concentrations is problematic because background concentrations per se are presumed not to be appropriate target levels to protect ecological receptors. Areas where background concentrations are high can commonly contain thriving communities because species dwelling there have developed a tolerance to higher metal concentrations. For organic constituents such as dioxin and furan congeners, the mobility and persistence of these compounds have resulted in anthropogenic "background" concentrations in most areas of the country. For these constituents, there is no presumption that levels below background concentrations are, de facto, not of ecological concern. In the case of dioxin, no background concentrations were identified for comparison to benchmark values. This is a source of uncertainty in the HWC analysis.

Mitigating factors–The fate and transport model used to estimate exposure concentrations typically does not account for the speciation of most metal constituents (with the exception of mercury). However, the geochemistry of metals behavior is often quite complex and the species of metal to which an ecological receptor is exposed, as well as the biological behavior of the metal species (e.g., bioaccumulation potential), may have a profound impact on whether adverse ecological effects occur. For example, it is well known that chromium exists in two valence states (III and VI) that have very different environmental behaviors and are associated with very different ecotoxicological responses. Although chromium is perhaps the best studied of the metal constituents with regard to speciation, it is not unique in terms of differences in behavior and toxicology related to its valence state. Therefore, in characterizing potential ecological risks, it is important to understand the relationship between the valence state of metal species in the environment and the valence state of metals administered in ecotoxicological studies.

Along with interpreting the results, key issues of uncertainty are presented in Section 2. In some cases, the assumptions of the analysis cannot adequately reflect the behavior of contaminants in the field. For example, in the case of metals, the HWC analysis assumed that all concentrations are bioavailable to receptors of concern; however, in natural systems, metals can bind to soils and sediment (e.g., adsorption), undergo chemical transformation to reduce or enhance toxicity (i.e., speciation), or remain freely available for uptake. Usually all of these conditions exist, but they may not be fully represented in the modeling effort because different environmental conditions (e.g., pH, carbon) influence the extent to which each of these variables dominates constituent bioavailability. Discussions of uncertainty and confidence in the results are specifically addressed in the presentation of results in Section 2.2.

Given the simplicity of the risk quotient screen, several issues of uncertainty arise from assumptions intrinsic to the analysis. Many of the uncertainties associated with the design of the analysis have been examined within the various sections of the methodology. As a final note, the HWC SERA assessed the incremental risks associated with HWC releases into generalized freshwater and terrestrial ecosystems. It did not consider the contribution of other anthropogenic sources deposited to soils and surface waters through long-range transport. Mercury and other constituents can be transported to remote areas through long-range transport, and this process increases the potential accumulation of these constituents to overall background levels (i.e., natural background and anthropogenic background).[3] The cumulative effect of both HWC emissions and other background sources may elevate the potential for risk to ecological receptors. EPA has indicated that mercury release from different industries over time has resulted in elevated anthropogenic background concentrations. Comparing these concentrations to wildlife criteria occasionally results in exceedance of protective levels (U.S. EPA, 1997). This indicates that anthropogenic background concentrations of mercury can contribute to the potential risks to ecological receptors. The HWC SERA did not consider the contribution of constituents transport by long-range mechanisms to overall media concentrations. Because this has the potential to result in underestimation of risk, a level of uncertainty is introduced into the HWC results.

In addition, some facilities were located proximate to one another, which may result in added loading of constituents to ecosystems. These cumulative impacts were not considered because each facility was modeled independently of the others. Finally, this analysis did not evaluate the potential adverse impacts associated with multiple constituent exposure. Each constituent was evaluated separately without accounting for potential cumulative impacts to ecological receptors resulting from simultaneous exposure to multiple constituents. Despite these simplifications, because of the overall conservative nature of the HWC SERA, these limitations are not likely to play a significant role in driving risk.

9.4 References

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[3] Natural background is defined as low levels of constituents found in environmental media resulting from natural processes (e.g., mineral weathering). Anthropogenic background is defined as level of constituent found in environmental media resulting from the releases of compounds from industrial processes over time.

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