Risk and Technology Review (RTR)
Risk Assessment Methodologies:
For Review by the EPA's Science Advisory Board

Case Studies –
MACT I Petroleum Refining Sources
Portland Cement Manufacturing

EPA-452/R-09-006
June 2009

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U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Health and Environmental Impacts Division
Research Triangle Park, NC

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ADD	Average daily dose
AEGL	Acute exposure guideline level
AERMOD	American Meteorological Society/EPA Regulatory Model
AIHA	American Industrial Hygiene Association
ANPRM	Advanced Notice of Proposed Rulemaking
ASTDR	US Agency for Toxic Substances and Disease Registry
CAA	Clean Air Act Amendments 0f 1990
CalEPA	California Environmental Protection Agency
CMAQ	Community Multiscale Air Quality (model)
CTE	Central tendency exposure
ERA	Environmental risk assessment
ERPG	Emergency Response Planning Guideline
FR	Federal Register
HAP	Hazardous air pollutant
HAPEM	Hazardous Air Pollutant Exposure Model
HEM	Human Exposure Model
HHRA	Human health risk assessment
HHRAP	Human Health Risk Assessment Protocol
HI	Hazard index
HQ	Hazard quotient
IARC	International Agency for Research on Cancer
IEUBK	Integrated Exposure Uptake Biokinetic (model)
IRIS	Integrated Risk Information System
ISH	Integrated Surface Hourly (database)
LOAEL	Lowest observed adverse effect level
MACT	Maximum Achievable Control Technology
MDL	Method detection limit
MIR	Maximum individual risk
MOA	Mode of action
MRL	Minimum Risk Level (ATSDR dose-response value)
NAC	National Advisory Committee
NATA	National Air Toxics Assessment
NCDC	National Climatic Data Center
NEI	National Emissions Inventory
NORM	Naturally occurring radioactive material
NPRM	Notice of Proposed Rulemaking
NY DEC	New York Department of Environmental Conservation
OAQPS	US EPA Office of Air Quality Planning and Standards
PAH	Polycyclic aromatic hydrocarbon
PB-HAP	Persistent and bioaccumulative HAP
POM	Polycyclic organic matter
REL	Reference exposure level
RfC	Reference concentration
RfD	Reference dose

RME	Reasonable maximum exposure
RTR	Risk and Technology Review
SF	Carcinogenic slope factor (usually for oral exposure)
TCDD	Tetrachlorodibenzo-*p*-dioxin, termed “dioxin” in this report
TOSHI	Target-organ-specific hazard index
TRIM	Total Risk Integrated Methodology
TRV	Toxicity reference value
URE	Unit risk estimate
USGS	US Geological Survey
WHO	World Health Organization
WOE	Weight-of-evidence for carcinogenicity in humans

1 Introduction

1.1 Purpose of this report

This report was developed to assist a panel of the EPA's Science Advisory Board (SAB) in reviewing the risk assessment approach and methods used by the EPA Office of Air Quality Planning and Standards (OAQPS) for its residual risk assessments in the Risk and Technology Review (RTR) program.

Although this document illustrates various components of our approach using two case studies from actual residual risk assessments (either previously performed or currently under development to support residual risk rulemaking), it is intended as a description of the approach itself. It is *not* intended to convey any definitive risk characterization. The case studies are drafts that may change as input data are revised as a result of public comment, or as methods are revised as a result of this review or our own improvement efforts. The case studies are included for the sole purpose of clarifying our approach for technical review, and assisting reviewers in understanding how EPA risk managers will use the information. They are not actual residual risk assessments that may be used to support regulatory decisions, and the results of the case studies are not the focus of this review. The final assessments for these source categories will be published in conjunction with their respective final rulemakings. It is important to note that each of these case study examples represents a snapshot of an analysis which is at a different stage of development – the petroleum refinery case study has proceeded through the ANPRM stage as well as a Notice of Proposed Rulemaking (NPRM) stage but has not yet been issued in support of any final rulemaking. The Portland cement case study has not yet been issued through an ANPRM, and therefore has not yet been subjected to any public scrutiny. The charge questions to the SAB panel are intended to elicit comment on whether the details of our approach constitute best science, and if not, how they could be improved.

In December of 2006 we obtained a consultation from a panel of the EPA Science Advisory Board (SAB) on our "RTR Assessment Plan." In June 2007 we received a letter [*1*] summarizing the key messages from that consultation on our risk assessment methods. We have attempted to respond to these key messages in developing this report.

1.2 Nature of RTR risk management decisions

The Clean Air Act establishes a two-stage regulatory process for addressing emissions of hazardous air pollutants (HAPs) from stationary sources. In the first stage, the Act requires the Environmental Protection Agency (EPA) to develop technology-based standards for categories of industrial sources (*e.g.*, petroleum refineries, pulp and paper mills, *etc.*)[*2*]. EPA has largely completed these standards. In the second stage, EPA is required to assess the health and environmental risks that remain after sources come into compliance with the technology-based standards, and to develop additional standards as necessary to protect public health with an ample margin of safety or to prevent adverse environmental effects. These risk-based standards must be completed within eight years of the technology-based standards. Several have already been completed.

In order to inform these risk-based decisions, EPA develops a risk assessment for each source category. In developing each assessment, EPA: (1) conducts a risk assessment using currently-available source and emissions data; (2) shares the source and emissions data and preliminary results of the assessment with the public through an Advance Notice of Proposed Rule Making (ANPRM) that asks for public comments on the methods and the source and emissions data; (3) receives comments; (4) reconciles comments and corrects the source and emissions data as appropriate, and; (5) reassesses the risks. The risk manager applies the results of the revised risk assessment, along with other information on cost, feasibility, and other non-risk-based information to support proposals and promulgations of technology- and risk-based regulatory decisions for each of the categories through the regular notice-and-comment rulemaking process.

1.2.1 Questions posed by risk managers

In order to determine if additional, risk-based, standards are needed, EPA needs to assess the "residual" risks to health and the environment that may remain after the technology-based standards are implemented. Residual risks are assessed separately for each source category. The Clean Air Act (CAA) requires that the EPA promulgate additional standards for a source category "if promulgation of such standards is required to provide an ample margin of safety to protect public health" or "to prevent, taking into consideration costs, energy, safety, and other relevant factors, an adverse environmental effect." A key factor in this risk management decision is the determination of the "lifetime excess cancer risk to the individual most exposed to emissions from a source in the category," or the maximum individual risk (MIR). The CAA specifically provides, for example, that a residual risk rulemaking is not required for a particular source category if EPA can show that the MIR for that category is less than 1 in a million.

EPA's risk management decision framework for residual risk rulemakings was first publicized in its finalization of the Benzene National Emission Standards for Hazardous Air Pollutants, or NESHAP, in 1989 (see 54 FR 38044). This framework implements the determination of an "ample margin of safety" in 2 steps. In the first step, the EPA determines "acceptable risk." Here, the goal is to limit the MIR for the entire source category to an acceptable level, with the proviso that the maximum limit on the acceptable MIR is ordinarily 100 in a million. EPA is allowed to adjust this limit (up or down) by considering other risk metrics (*e.g.*, the total estimated cancer incidence due to emissions from the source category) and other health factors, including the consideration of noncancer human health risks or environmental risks, as well as uncertainties in the risk estimates. The EPA is not allowed to consider costs of regulatory actions in this step. In the second step, the EPA determines the "ample margin of safety." Here, EPA is allowed to factor in the costs and feasibility of controlling emissions from the source category as it evaluates further risk reductions across the source category with the goal of maximizing the number of persons whose lifetime cancer risks due to emissions from the source category are less than 1 in a million.

For effects other than cancer, EPA estimates the ratios of chronic or acute exposure to appropriate health benchmarks and considers how each benchmark was developed when establishing a level of "acceptable risk" or determining if health is protected with an "ample margin of safety." For environmental effects, EPA compares exposures of nonhuman receptors to (1) human benchmarks for screening and (2) published benchmarks for similar species for refined assessments.

1.2.2 Answers provided by RTR risk assessment case studies

The final product of the risk assessment process is a set of overall conclusions about risk that are complete, informative, and useful for decision makers. In general, the assessment's ability to provide these things depends on the information available, the application of the risk information and the resources available.

In determining whether an ample margin of safety has been achieved, and if adverse environmental effects will not occur, EPA risk managers look to the residual risk assessment to provide estimates of:

1) maximum individual lifetime cancer risk
2) annualized lifetime cancer incidence and/or deaths
3) distribution of lifetime cancer risk in the exposed population
4) HAPs that contribute substantially to health or environmental risk
5) maximum individual hazard quotients (HQ[1]) for non-cancer chronic effects
6) target organ-specific hazard indices (TOSHI[2]) for chronic effects other than cancer
7) maximum individual hazard quotients (HQ) for non-cancer acute effects
8) distribution of hazard index in the exposed population;
9) ecological receptors for which exposures exceed benchmarks

HAP-specific cancer risks are added across chemicals because EPA does not yet recognize any combination of HAPs for which cancer risk is demonstrably not additive. Chemical specific HQs are added only for chemicals having the same mechanism of action, or (in the absence of such information) that affect the same target organ. Chronic risk estimates are based on annual average concentrations at individual census block centroids. Acute risks are estimated using additional protective assumptions explained in Section 2.2.5, based on maximum 1-hour concentrations at the worst location.

The residual risk assessment must also provide a risk characterization that transparently describes how these estimates were developed and the uncertainties associated with them. The risk manager uses this information, along with information on costs and feasibility of reducing emissions, legal requirements, public concern and comment, political considerations, and other factors in developing a residual risk rule.

The residual risk assessment, therefore, represents only part of the information used in making risk management decisions, but it is arguably the most critical element because it can determine that no rule is needed at all, or set an upper limit on how stringent any rule might need to be. EPA believes that it is possible to use a consistent, streamlined approach to these assessments that is scientifically sound and that also uses time and resources efficiently. This report describes the methods that EPA has developed to conduct these assessments through the use of two illustrative case studies – one for the petroleum refineries source category and one for Portland

[1] Hazard quotient – the ratio of an estimated exposure to an appropriate health benchmark (usually an exposure level associated with no adverse effects).
[2] Hazard index – the sum of hazard quotients for multiple chemicals. A TOSHI is a hazard index limited to chemicals that affect the same target organ or system.

cement manufacturers. Together, these two case studies cover the breadth of scientific issues often addressed in our residual risk assessments.

The quantitative results of the risk characterization for each case study have been used to address EPA risk managers' specific questions pertaining to the requirements of promulgating a residual risk rule under the Clean Air Act. Table 1-1 provides an example of summary information that the two example baseline risk assessments produce to support risk management decision-making. OAQPS staff also briefs risk managers on the context of the findings, and on the uncertainties surrounding the risk estimates. The remainder of this report provides the details of each of these two case study risk assessments, culminating with the presentation of a summary of baseline risk information and a characterization of the risk for each source category, including a discussion of uncertainties and the implications of the findings. Finally, we wrap up this report by presenting a number of sensitivity studies which address specific issues that have arisen during the process of developing and performing these risk assessments.

Table 1-1. Summary risk assessment results for petroleum refineries and Portland cement source category case studies.

Risk metric	Petroleum refineries source category	Portland cement source category
Facilities subject to MACT/modeled	156/156	118/104
Population within 50 km of modeled facility	90 million	54 million
Lifetime inhalation cancer risk		
Maximum individual cancer risk	30 in 1 million	800 in 1 million[3]
Population > 100 in 1 million	0	400
Population > 10 in 1 million	4000	15,000
Population > 1 in 1 million	460,000	470,000
Facilities w/ MIR > 1 in 1 million	77	29
HAP cancer risk drivers	Benzene, naphthalene, POM, 1,3-butadiene, TCE	Chromium (VI), arsenic, cadmium, beryllium, benzene
Chronic inhalation noncancer risk		
Target organs/systems	Respiratory	Neurological Respiratory Kidney
Maximum chronic hazard index	0.3	10 Neurological 6 Respiratory 3 Kidney
Facilities w/ HI > 1.0	0	2 Neurological 3 Respiratory 1 Kidney
Population > hazard index 1.0	0	~ 3000
HAP chronic risk drivers	Diethanolamine	Manganese, chlorine, HCl, and cadmium
Acute inhalation noncancer risk		
Screening:	50 – REL for Benzene	50 – AEGL-1 for HCl

[3] Does not include analysis of potential radionuclide risks in Section 3.6.1.3.

Table 1-1. Summary risk assessment results for petroleum refineries and Portland cement source category case studies.

Risk metric	Petroleum refineries source category	Portland cement source category
HAP/max. acute HQ/ benchmark[4]	20 – REL for Hydrogen fluoride 6 – AEGL-1 for Hydrogen fluoride	4 – AEGL-2 for HCl 7 – AEGL-1 for Chlorine 2 –AEGL-2 for Chlorine 3 – AEGL-1 for Formaldehyde
Refined: HAP/max. acute HQ/ benchmark	8 – REL for Benzene 5 – REL for Hydrogen fluoride 2 – AEGL-1 for Hydrogen fluoride	10 – AEGL-1 for Hydrogen chloride 2 – AEGL-1 for Chlorine 2 – AEGL-1 for Formaldehyde
Facilities w/ HQ > 1.0	20	8
Multipathway risk		
Maximum individual cancer risk	N/A[5]	200 in 1 million[6] 1-10 in 1 million[7]
HAP cancer risk drivers		Dioxin
Maximum individual hazard quotient		2[6] 0.08[7]
HAP noncancer risk drivers		Dioxin[8]
Ecological risk		
Direct contact screening: Max. concentration/RfC ratio	0.3 (Diethanolamine)	N/A
Direct contact refined: Max. concentration/RfC ratio	N/A	0.1 (HCl)
Multipathway screening: Max. concentration/benchmark ratio	1.0 (PAHs in soil)	N/A
Multipathway refined: Max. concentration/benchmark ratios	N/A	4 – Methylmercury, mink[6] 0.02 – Methylmercury, mink[9]

[4] Definitions of each benchmark appear in the glossary, with complete descriptions in Section 2.2.6.2.
[5] A multipathway assessment has been developed for petroleum refineries, but it has been omitted from the case study and from this table for brevity. Instead, we are using the Portland cement risk assessment to illustrate our approach to multipathway health risk assessment.
[6] Includes subsistence fishing in a nearby small pond at a harvest rate that is probably not sustainable.
[7] Omits small pond but includes subsistence farming at nearest farm and fishing from other water bodies.
[8] Methylmercury was also evaluated for noncancer effects via ingestion, but HQs did not exceed 1.0.
[9] Omits small pond but includes subsistence farming at nearest farm and fishing from other water bodies.

2 Petroleum refineries case study

2.1 Introduction

Section 2 contains the methods and the results of the baseline risk assessment performed for the petroleum refining source category. The methods discussion includes descriptions of the methods used to develop refined estimates of chronic inhalation exposures and human health risks for both cancer and noncancer endpoints, as well as descriptions of the methods used to screen for acute health risks, chronic non-inhalation health risks, and adverse environmental effects. Since the screening assessment did not indicate any significant potential for chronic non-inhalation health effects, or environmental impacts including effects to threatened and endangered species, no further refinement of this assessment was performed. A screening assessment did indicate a possible concern for acute health effects; thus, a more refined analysis for acute exposure impacts was performed and the results are presented.

2.2 Methods

2.2.1 Emissions and source data

The 2002 National Emissions Inventory (NEI) Final Version 1 (made publicly available February 2006) served as the starting point for this assessment. The 2002 NEI purportedly contains information on actual emissions during the entire 2002 base year. Using the process MACT code[10], we developed a subset of this inventory that contains emissions and facility data for the petroleum refining source category. Next, we performed an engineering review of these using EPA engineers who were directly involved in the development of the MACT standard for the source category, and/or who have extensive knowledge of the characteristics of this industry. NEI data were also updated with site-specific benzene emissions data for 22 refineries as provided by the American Petroleum Institute. The goal of the engineering review was to identify readily-apparent limitations and issues with the emissions data (particularly those that would greatly influence risk estimates) and to make changes to the dataset where possible to address these issues and decrease the uncertainties associated with the assessment.

Once the dataset for the entire source category was created, it was published through an Advanced Notice of Proposed Rulemaking (ANPRM), making it available for public comment. After a 60-day comment period, submitted comments and corrections were evaluated for quality and engineering consistency. Corrections we concluded were valid were incorporated into the inventory. In August 2007, a Notice of Proposed Rulemaking (NPRM) was published making the source category dataset available for a second 60-day comment period, which was subsequently re-opened for another 50 days. Again the comments and corrections were evaluated and incorporated into the inventory. The final petroleum refinery database for our case study contains information for 156 facilities, and this is thought to represent the source category

[10] The tagging of data with MACT codes allows EPA to determine reductions attributable to the MACT program. The NEI associates MACT codes corresponding to MACT source categories with stationary major and area source data. MACT codes may be assigned either at the process level or at the site level in the point source data (e.g., the MACT code for municipal waste combustors (MWCs) is assigned at the site level whereas the MACT code for petroleum refinery catalytic cracking is assigned at the process level).

in its entirety at this time. An analysis of the revisions to the emissions inventory resulting from this process and the associated revisions to the risk estimates is presented in Appendix A.

2.2.2 Dispersion modeling for inhalation exposure assessment

Both long- and short-term inhalation exposure concentrations and associated health risk from each facility of interest were estimated using the Human Exposure Model in combination with the American Meteorological Society/EPA Regulatory Model dispersion modeling system (HEM-AERMOD, or HEM3). HEM3 performs three main operations: atmospheric dispersion modeling, estimation of individual human exposures and health risks, and estimation of population risks. This section focuses on the dispersion modeling component. The exposure and risk characterization components are discussed in sections 2.2.3 and 2.2.7.

The dispersion model in the HEM3 system, AERMOD version 07026, is a state-of-the-science Gaussian plume dispersion model that is preferred by EPA for modeling point, area, and volume sources of continuous air emissions from facility applications [*3*]. Further details on AERMOD can be found in the AERMOD Users Guide [*4*]. The model is used to develop annual average ambient concentration through the simulation of hour-by-hour dispersion from the emission sources into the surrounding atmosphere. Hourly emission rates used for this simulation are generated by evenly dividing the total annual emission rate from the inventory into the 8,760 hours of the year.

The first step in the application of the HEM3 modeling system is to predict ambient concentrations at locations of interest. The AERMOD model options employed are summarized in Table 2-1 and are discussed further below.

Table 2-1. AERMOD version 07026 model options for RTR II modeling

Modeling Option	*Selected Parameter for chronic exposure*
Type of calculations	Hourly Ambient Concentration
Source type	Point and area sources
Receptor orientation	Polar (10 rings at 10-deg) Discrete (census block centroids)
Terrain characterization	Actual from USGS 1-degree DEM data
Building downwash	Not Included
Plume deposition/depletion	Not Included
Urban source option	No
Meteorology	1 year representative data

Meteorological data for HEM3 are selected from a list of 158 National Weather Service (NWS) surface observation stations across the continental United States, Alaska, Hawaii, and Puerto Rico. In most cases the nearest station is selected as representative of the conditions at the

subject facility. Two facilities[11] furnished representative meteorological datasets as part of the ANPRM process. For these two facilities, the facility-supplied meteorological data were utilized in place of the HEM "nearest selected" station. Ideally, when considering off-site meteorological data most site specific dispersion modeling efforts will employ up to five years of data to capture variability in weather patterns from year to year. However, because of the large number of facilities in the analysis and the extent of the dispersion modeling analysis (national scale), it was not practical to model five years of data and only the year 1991 was modeled. While the selection of a single year may result in under-prediction of long-term ambient levels at some locations, likewise it may result in over-prediction at others. For each facility identified by its characteristic latitude and longitude coordinates, the closest meteorological station was used in the dispersion modeling. The average distance between a modeled facility and the applicable meteorological station was 40 miles (72 km). A sensitivity analysis evaluating the potential change in risk if modeling was performed with a different meteorological station (not the nearest one) is presented in Section 4.5 of this document.

The HEM3 system estimates ambient concentrations at the geographic centroids of census blocks (using the 2000 Census), and at other receptor locations that can be specified by the user. In cases where the census block centroid was found to be located on facility property (as determined from satellite imagery) the receptor was moved to the nearest off-site location. The model accounts for the effects of multiple facilities when estimating concentration impacts at each block centroid. In this assessment, we combined only the impacts of facilities within the same source category, and assessed chronic exposure and risk only for census blocks with at least one resident (*i.e.*, locations where people may reasonably be assumed to reside rather than receptor points at the fenceline of a facility). Chronic ambient concentrations were calculated as the annual average of all estimated short-term (one-hour) concentrations at each block centroid. Possible future residential use of currently uninhabited areas was not considered. Census blocks, the finest resolution available in the census data, are typically comprised of approximately 40 people or about ten households.

In contrast to the development of ambient concentrations for evaluating long-term exposures, which was performed only for occupied census blocks, worst-case short-term (one-hour) concentrations were estimated both at the census block centroids and at points nearer the facility that represent locations where people may be present for short periods, but generally no nearer than 100 meters from the center of the facility (note that for large facilities, this 100-meter ring could still contain locations inside the facility property). Since short-term emission rates were needed to screen for the potential for hazard via acute exposures, and since the NEI contains only annual emission totals, we applied the general assumption to all source categories that the maximum one-hour emission rate from any source was ten times the average annual hourly emission rate for that source. Average hourly emissions rate is defined as the total emissions for a year divided by the total number of operating hours in the year. This choice of a factor of ten for screening was originally based on engineering judgment. To develop a more robust peak-to-mean emissions factor, and in response to one of the key messages from the SAB consultation on our RTR Assessment Plan, we recently performed an analysis using a short-term emissions dataset from a number of sources located in Texas (originally reported on by Allen *et al.*

[11] For NEI8406, data from the Fairbanks, Alaska met station from the year 2001 modeled and for NEI46556, data from St. Croix, Virgin Islands met station from the year 2005 was utilized.

2004)[5]. In that report, the Texas Environmental Research Consortium Project compared hourly and annual emissions data for volatile organic compounds for all facilities in a heavily-industrialized 4-county area (Harris, Galveston, Chambers, and Brazoria Counties, TX) over an eleven-month time period in 2001. We obtained the dataset and performed our own analysis, focusing that analysis on sources which reported emitting high quantities of HAP over short periods of time. Based on our analysis, ratios of short-term event release rate to long-term release rate varied from 0.00000004 to 74. The 99th percentile ratio was 9 (*i.e.*, an event release rate nine times the long-term average). Only 3 events were greater than 10 times the average, and of these, only one exceeded 11, and that single event was 74 times the average. While there are some documented emission excursions above this level, our analysis of the data from the Texas Environmental Research Consortium suggests that this factor should cover more than 99% of the short-term peak gaseous or volatile emissions from typical industrial sources. Details of this analysis are presented in Appendix B.

Census block elevations for HEM3 modeling were determined nationally from the US Geological Survey 1-degree digital elevation model (DEM) data files, which have a spatial resolution of about 90 meters. Polar grid elevations (used in estimating short- and long-term ambient concentrations) were evaluated at the highest elevation of any census block in that sector. If a sector does not contain any blocks, the model defaults to the elevation of the nearest block. If the elevation is not provided for the emission source, the model takes the average elevation of all sectors of the nearest model ring.

In addition to utilizing receptor elevation to determine plume height, AERMOD adjusts the plume's flow if nearby elevated hills are expected to influence the wind patterns.

2.2.3 Estimating human inhalation exposure

For this assessment, we used the annual average ambient air concentration of each HAP at each census block centroid as a surrogate for the lifetime inhalation exposure concentration of all the people who reside in the census block. That is, this risk analysis did not consider either the short-term or long-term behavior (mobility) of the exposed populations and its potential influence on their exposure.

We did not address short-term human activity in this assessment for two reasons. First, our experience with the 1996 and 1999 NATA assessments (which modeled daily activity using EPA's HAPEM model) suggests that, given our current understanding of microenvironment concentrations and daily activities, modeling short-term activity would, on average, reduce risk estimates about 25% for particulate HAPs; it will also reduce risk estimates for gaseous HAPs, but typically by much less. Second, basing exposure estimates on average ambient concentrations at census block centroids may underestimate or overestimate actual exposure concentrations at some residences. Further reducing exposure estimates for the most highly-exposed residents by modeling their short-term behavior could add a systematic low bias to these results.

We did not address long-term migration in this assessment nor population growth or decrease over 70 years, instead basing the assessment on the assumption that each person's predicted exposure is constant over the course of their lifetime, which is assumed to be 70 years. In

assessing cancer risk, 3 metrics are generally estimated, the maximum individual risk (MIR) which is defined as the risk associated with a lifetime of exposure at the highest concentration, the population risk distribution, and the cancer incidence. This assumption of not considering short or long-term population mobility does not bias the estimate of the theoretical MIR nor does it affect the estimate of cancer incidence since the total population number remains the same. It does, however, affect the shape of the distribution of individual risks across the affected population, shifting it toward higher estimated individual risks at the upper end and reducing the number of people estimated to be at lower risks, thereby biasing the risk estimates high. In section 4 of this report, we demonstrate a method for accounting for long-term population mobility, and show how it affects risk estimates for this source category.

When screening for potentially significant acute exposures, we used a modeled estimate of the highest hourly ambient concentration at any off-site location as the surrogate for the maximum potential acute exposure concentration for any individual.

2.2.4 Multipathway and environmental risk screening

The potential for significant human health risks due to exposures via routes other than inhalation (*i.e.*, multipathway exposures) was screened by first determining whether any sources emitted any hazardous air pollutants known to be persistent and bioaccumulative in the environment (PB-HAP). There are 14 PB-HAP compounds or compound classes identified for this screening in EPA's Air Toxics Risk Assessment Library [*6*]. They are cadmium compounds, chlordane, dioxins, DDE, heptachlor, hexachlorobenzene, hexachlorocyclohexane, lead compounds, mercury compounds, methoxychlor, polychlorinated biphenyls, polycyclic organic matter (POM), toxaphene, and trifluralin.

Emissions of one PB-HAP – polycyclic organic matter (POM) – were identified in the inventory for some petroleum refineries. These emissions were evaluated for potential non-inhalation risks and adverse environmental impacts using EPA's recently-developed screening scenario which was developed for use with the TRIM-FaTE[12] model. This screening scenario uses environmental media outputs from the peer-reviewed TRIM-Fate model to estimate the maximum potential ingestion risks for any specified emission scenario by using a generic farming/fishing exposure scenario that simulates a subsistence environment. The screening scenario retains many of the ingestion and scenario inputs developed for EPA's Human Health Risk Assessment Protocols (HHRAP) for hazardous waste combustion facilities [*7*]. In the development of the screening scenario a sensitivity analysis was conducted to ensure that its key design parameters were established, such that environmental media concentrations were not underestimated and to also minimize the occurrence of false positives for human health and ecological endpoints. See Appendix C for a complete discussion of the development and testing of the screening scenario, which we call TRIMScreen. For the purposes of multipathway risk screening, the levels of concern below which risks were considered insignificant were 1 in a million for lifetime cancer risk and a hazard quotient of 1.0 for noncancer impacts.

Additionally, we evaluated the potential for significant ecological exposures to non PB-HAP from exceedances of chronic human health inhalation thresholds in the ambient air near these facilities. Human health dose-response threshold values are generally derived from studies

[12] EPA's Total Risk Integrated Methodology (General Information) http://epa.gov/ttn/fera/trim_gen.html

conducted on laboratory animals (such as rodents) and developed with the inclusions of uncertainty factors that could be as high as 3000. Thus, these human threshold values are often significantly lower than the level expected to cause an adverse effect in an exposed rodent. It should be noted that there is a scarcity of data on the direct atmospheric impact of these HAPs on other receptors, such as plants, birds, and wildlife. Thus, if the maximum inhalation hazard in an ecosystem is below the level of concern for humans, we have generally concluded that mammalian receptors should be at no risk of adverse effects due to inhalation exposures from non PB-HAP, and have assured that other ecological receptors are also similarly not at any significant risk from direct atmospheric impact. In some isolated cases where we have data indicating potential adverse impacts on plants, birds, or other wildlife due to the direct atmospheric impacts of specific HAPs, we note that as an uncertainty and, where possible, refine our analysis by comparing our modeled impacts to available threshold values from the scientific literature. The case study for Portland cement manufacturing contains an example of such an analysis in Section 3.5.2.2 and Appendix K.

2.2.5 Acute Risk Screening and Refined Assessments

In establishing a scientifically-defensible approach for the assessment of potential health risks due to acute exposures to HAPs, we have followed the same general approach that has been used for developing chronic health risk assessments under the residual risk program. That is, we developed a tiered, iterative approach. This tiered, iterative approach to risk assessment has been endorsed by the National Academy of Sciences in its 1993 publication "Science and Judgment in Risk Assessment" and subsequently was endorsed in the EPA's "Residual Risk Report to Congress" in 1999.

The assessment methodology is designed to eliminate from further consideration those facilities for which we have confidence that no acute adverse health effects of concern will occur. To do so, we use what is called a tiered, iterative approach to the assessment. This means that we begin with a screening assessment, which relies on minimal data and uses conservative assumptions that in combination approximate a worst-case exposure. The result of this screening process is that either the facility being assessed poses no risk of acute health effects (*i.e.*, it "screens out"), or that it requires further, more refined, assessment. A refined assessment could utilize site-specific data on the temporal pattern of emissions, the layout of emission points at the facility, the boundaries of the facility, and the local meteorology. In some cases, all of these site-specific data would be needed to refine the assessment; in others, lesser amounts of site-specific data could be used to determine that acute exposures are not a concern, and significant additional data collection would not be necessary. The refinement process generally continues until the acute risk either proves to be an important part of the assessment, or it screens out.

Acute health risk screening was performed as the first step. We used conservative assumptions for emission rates, meteorology, and exposure location. We used the following worst-case assumptions in our screening approach:

- Peak 1-hour emissions were assumed to equal 10 times the average 1-hour emission rates.
- For facilities with multiple emission points, peak 1-hour emissions were assumed to occur at all emission points at the same time.

- For facilities with multiple emission points, 1-hour concentrations at each receptor were assumed to be the sum of the maximum concentrations due to each emission point, regardless of whether those maximum concentrations occurred during the same hour.
- Worst-case meteorology (from one year of local meteorology) was assumed to occur at the same time the peak emission rates occurred. The recommended EPA local-scale dispersion model, AERMOD, was used for simulating atmospheric dispersion.
- A person was located downwind at the point of maximum impact during this same 1-hour period, but no nearer to the source than 100 meters.
- The maximum impact was compared to multiple short-term health thresholds for the chemical being assessed to determine if a possible acute health risk might exist. These benchmarks are described in the next section of this report.

We performed more refined acute assessments for selected facilities for which the screening assessment showed exceedances of short-term health thresholds. In general, refined assessments proceed stepwise through the following activities:

- Examine aerial photographs of the site to determine if the impact area of concern is outside the facility property boundary.
- Adjust the peak one-hour emissions default (multiplier of 10) to a more source-specific value, where data are available and indicate that such an adjustment is appropriate.
- Perform refined modeling using site-specific information. Refined modeling can include running AERMOD (without HEM) to estimate 1-hour concentrations that reflect the maximum concentration due to each emission point simultaneously emitting at its maximum assumed short-term rate.

For facilities that still show off-site acute impacts above an HQ of 1 after refining the assessment, we present the maximum HQ values for the available acute thresholds and discuss the possible implications of these results in light of the available health effects information and knowledge regarding the actual facility configuration.

2.2.6 Dose-Response Assessment

2.2.6.1 Sources of chronic dose-response information

Dose-response assessment information (carcinogenic and non-carcinogenic) for chronic exposure (either by inhalation or ingestion) for the HAPs reported in the emissions inventory were based on the EPA Office of Air Quality Planning and Standards' existing recommendations for HAPs [*8*], also used for NATA 1999 [*9*]. This information has been obtained from various sources and prioritized according to (1) conceptual consistency with EPA risk assessment guidelines and (2) level of peer review received. The prioritization process was aimed at incorporating into our assessments the best available science with respect to dose-response information. The recommendations are based on the following sources, in order of priority:

1) US Environmental Protection Agency (EPA). EPA has developed dose-response assessments for chronic exposure for many of the pollutants in this risk assessment. These

assessments typically provide a qualitative statement regarding the strength of scientific data and specify a reference concentration (RfC, for inhalation) or reference dose (RfD, for ingestion) to protect against effects other than cancer and/or a unit risk estimate (URE, for inhalation) or slope factor (SF, for ingestion) to estimate the probability of developing cancer. The RfC is defined as "an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime." The RfD is "an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime." The URE is "the upper-bound excess cancer risk estimated to result from continuous lifetime exposure to an agent at a concentration of 1 $\mu g/m^3$ in air." The SF is "an upper bound, approximating a 95% confidence limit, on the increased cancer risk from a lifetime exposure to an agent. This estimate, [is] usually expressed in units of proportion (of a population) affected per mg/kg-day…" EPA disseminates dose-response assessment information in several forms, based on the level of review. The Integrated Risk Information System (IRIS) [*10*], is an EPA database that contains scientific health assessment information, including dose-response information, that has undergone interagency review. All IRIS assessments completed since 1996 have also undergone independent external peer review. The current IRIS process[13] includes review by EPA scientists, interagency reviewers from other federal agencies, and the public, and peer review by a panel of independent scientists external to EPA. Dose-response assessments for some substances were prepared by the EPA Office of Research and Development, but not submitted for EPA consensus. EPA has assembled the results of many such assessments in the Health Effects Assessment Summary Tables (HEAST) [*11*], which this assessment uses as a source of last resort for one HAP; 1,2,4-trichlorobenzene. EPA's science policy approach, under the current carcinogen guidelines, is to use linear low-dose extrapolation as a default option for carcinogens for which the mode of action (MOA) has not been identified. We expect future EPA dose-response assessments to identify nonlinear MOAs where appropriate, and we will use those analyses (once they are peer reviewed) in our risk assessments. At this time, however, there are no available carcinogen dose-response assessments for inhalation exposure that are based on a nonlinear MOA.

2) California Environmental Protection Agency (CalEPA). The CalEPA Office of Environmental Health Hazard Assessment has developed dose-response assessments for many substances, based both on carcinogenicity and health effects other than cancer. The process for developing these assessments is similar to that used by EPA to develop IRIS values and incorporates significant external scientific peer review. As cited in the CalEPA Technical Support Document for developing their chronic assessments [*12*]: "The guidelines for developing chronic inhalation exposure levels incorporate many recommendations of the U.S. EPA (1994) [*13*] and NAS (NRC, 1994) [*14*]." The non-cancer information includes available inhalation health risk guidance values expressed as chronic inhalation reference exposure levels (REL) [*15*]. CalEPA defines the REL as "the concentration level at or below which no adverse health effects are anticipated in the general human population". CalEPA's

[13] April 10, 2008 memorandum from EPA Deputy Administrator Marcus Peacock to Assistant Administrator George Gray, subject "Implementation of Revised IRIS Process"

quantitative dose-response information on carcinogenicity by inhalation exposure is expressed in terms of the URE [*16*], defined similarly to EPA's URE.

3) US Agency for Toxic Substances and Disease Registry (ATSDR). ATSDR, which is part of the US Department of Health and Human Services, develops and publishes Minimum Risk Levels (MRLs) [*17*] for inhalation and oral exposure to many toxic substances. As stated on the ATSDR web site: "Following discussions with scientists within the Department of Health and Human Services (HHS) and the EPA, ATSDR chose to adopt a practice similar to that of the EPA's Reference Dose (RfD) and Reference Concentration (RfC) for deriving substance specific health guidance levels for non neoplastic endpoints." The MRL is defined as "an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (other than cancer) over a specified duration of exposure". ATSDR describes MRLs as substance-specific estimates to be used by health assessors to select environmental contaminants for further evaluation. Exposures above an MRL do not necessarily represent a threat, and MRLs are therefore not intended for use as predictors of adverse health effects or for setting cleanup levels.

In developing chronic risk estimates, we adjusted dose-response values for some HAPs based on professional judgment, as follows:

1) In the case of HAP categories such as glycol ethers, the most conservative dose-response value of the chemical category was used as a surrogate for other compounds in the group for which dose-response values were not available. This was done in order to examine, under conservative assumptions, whether these HAPs that lack dose-response values may pose an unacceptable risk and require further examination, or screen from further assessment.

2) This assessment bases risk estimates for formaldehyde on a dose-response value published in 1999 by the CIIT Centers for Health Research. EPA is currently reviewing the existing IRIS assessment for formaldehyde.

3) A substantial proportion of POM reported to EPA's national emission inventory (NEI) were not speciated into individual compounds. As a result, it was necessary to apply the same simplifying assumptions to this assessment that were used for the 1999 NATA study [*18*]. This assessment divided POM emissions into eight categories. Categories 1 and 2 were assigned a URE equal to 5% of that for pure benzo[a]pyrene. Categories 3-7 were composed of emissions that were reported as individual compounds. These compounds were placed in the category with an appropriated URE. Category 8, composed of unspeciated carcinogenic polynuclear aromatic hydrocarbons (a subset of POM called 7-PAH), was assigned a URE equal to 18% of that for pure benzo[a]pyrene. Details of the development of the 5% and 18% URE estimated are available at http://www.epa.gov/ttn/atw/sab/appendix-h.pdf.

The emissions inventory for the petroleum refining source category includes emissions of 73 individual compounds comprising 54 HAP. Of the 54 HAP, 21 are classified as known, probable, or possible carcinogens, with quantitative cancer dose-response values available. The 21 HAP, their quantitative inhalation chronic cancer dose-response values, and the source of the value are listed below in Table 2-2. This source category emits several other HAPs (*i.e.*, cresols,

styrene, and vinyl acetate) for which some limited or inadequate evidence exists for determining carcinogenicity. Because these substances lack quantitative estimates of cancer potency, we did not estimate risks for them. The POM compounds with chronic oral cancer dose-response values available (for which a multipathway screening assessments was performed) are listed in Table 2-3.

The emissions inventory for the petroleum refining source category includes emissions of 45 HAP with quantitative chronic noncancer threshold values available. The 45 HAP, their threshold values, and the source of the value are listed in Table 2-4.

Table 2-2. Dose-Response Values for Chronic Inhalation Exposure to Carcinogens

URE (unit risk estimate for cancer)[14] = cancer risk per µg/m^3 of average lifetime exposure. Sources: IRIS = EPA Integrated Risk Information System, CAL = California EPA Office of Environmental Health Hazard Assessment, EPA/OAQPS = interim value recommended by the EPA Office of Air Quality Planning and Standards,

Pollutant	CAS Number[15]	URE (1/µg/m3)	Source
Acetaldehyde	75070	2.2E-06	IRIS
Acrylonitrile	107131	6.8E-05	IRIS
Aniline	62533	1.6E-06	CAL
Benzene[16]	71432	7.8E-06	IRIS
Bis(2-ethylhexyl)phthalate	117817	2.4E-06	CAL
1,3-Butadiene	106990	3.0E-05	IRIS
Carbon tetrachloride	56235	1.5E-05	IRIS
1,4-Dichlorobenzene	106467	1.1E-05	CAL
1,4-Dioxane	123911	7.7E-06	CAL
Ethylene dibromide	106934	6.0E-04	IRIS
Ethylene dichloride	107062	2.6E-05	IRIS
Formaldehyde	50000	5.5E-09	EPA/OAQPS
Methyl tert-butyl ether	1634044	2.6E-07	CAL
Methylene chloride	75092	4.7E-07	IRIS
Naphthalene	91203	3.4E-05	CAL
Pentachlorophenol	87865	5.1E-06	CAL
Polycyclic Organic Matter	246	[17]	EPA OAQPS[12]
- Benzo(a)anthracene	56553	1.1E-04	EPA OAQPS[12]
- Benzo(a)pyrene	50328	1.1E-03	EPA OAQPS[12]

[14] The URE is the upper-bound excess cancer risk estimated to result from continuous lifetime exposure to an agent at a concentration of 1 µg/m^3 in air. URE's are considered upper bound estimates meaning they represent a plausible upper limit to the true value.

[15] Chemical Abstract Services identification number. For groups of compounds that lack a CAS number we have used a surrogate 3-digit identifier corresponding to the group's position on the CAA list of HAPs.

[16] The EPA IRIS assessment for benzene provides a range of plausible UREs. This assessment used the highest value in that range, 7.8E-06 per ug/m3. The low end of the range is 2.2E-06 per ug/m^3.

[17] Assigned the URE associated with a mixture of POM compounds having a similar potency. Details of this method, also used in the 1999 National Air Toxics Assessment, are available at http://www.epa.gov/ttn/atw/nata1999/99pdfs/pomapproachjan.pdf

URE (unit risk estimate for cancer)[14] = cancer risk per µg/m^3 of average lifetime exposure. Sources: IRIS = EPA Integrated Risk Information System, CAL = California EPA Office of Environmental Health Hazard Assessment, EPA/OAQPS = interim value recommended by the EPA Office of Air Quality Planning and Standards,

Pollutant	CAS Number[15]	URE (1/µg/m3)	Source
- Benzo(b)fluoranthene	205992	1.1E-04	EPA OAQPS[12]
- Benzo(k)fluoranthene	207089	1.1E-04	EPA OAQPS[12]
- Chrysene	218019	1.1E-05	EPA OAQPS[12]
- Dibenzo(a,h)anthracene	53703	1.2E-03	EPA OAQPS[12]
- Indeno(1,2,3-cd)pyrene	193395	1.1E-04	EPA OAQPS[12]
1,1,2,2-Tetrachloroethane	79345	5.8E-05	IRIS
Tetrachloroethene	127184	5.9E-06	CAL
Trichloroethylene	79016	2.0E-06	CAL
Vinyl chloride	75014	8.8E-06	IRIS

Table 2-3. Dose-Response Values for Chronic Oral Exposure to Carcinogens

SF (oral slope factor for cancer) = cancer risk per mg/kg/d of average lifetime exposure. Sources: IRIS = EPA Integrated Risk Information System, CAL = California EPA Office of Environmental Health Hazard Assessment, EPA/OAQPS = interim value recommended by the EPA Office of Air Quality Planning and Standards.

Pollutant	CAS Number[10]	SF (1/mg/kg/d)	Source
Polycyclic organic matter (POM)	246	0.5	EPA OAQPS
- Benzo(a)anthracene	56553	1	EPA OAQPS
- Benzo(a)pyrene	50328	7	EPA OAQPS
- Benzo(b)fluoranthene	205992	1	EPA OAQPS
- Benzo(k)fluoranthene	207089	1	EPA OAQPS
- Chrysene	218019	0.1	EPA OAQPS
- Dibenz(a,h)anthracene	53703	4	EPA OAQPS
- Indeno(1,2,3-cd)pyrene	193395	1	EPA OAQPS

Table 2-4. Dose-Response Values for Chronic Inhalation Exposure to Noncarcinogens

RfC (or similar inhalation values) = an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Sources: IRIS = EPA Integrated Risk Information System, CAL = California EPA Office of Environmental Human Health Assessment, HEAST = EPA Health Effects Assessment Summary Table, ATSDR = US Agency for Toxic Substances and Disease Registry.

Pollutant	CAS Number[10]	RfC (mg/m3)	Source[18]
Acetaldehyde	75070	0.009	IRIS -- L
Acrylonitrile	107131	0.002	IRIS – M
Aniline	62533	0.001	IRIS – L
Benzene	71432	0.03	IRIS – M
Bis(2-ethylhexyl)phthalate	117817	0.01	CAL -- M
1,3-Butadiene	106990	0.002	IRIS – M
Carbon disulfide	75150	0.7	IRIS – M
Carbon tetrachloride	56235	0.19	ATSDR
Chlorobenzene	108907	1	CAL
Chloroform	67663	0.098	ATSDR
Cresols (mixed)	1319773	0.6	CAL
m-Cresol[19]	108394	0.6	CAL
Cumene	98828	0.4	IRIS – L
p-Dichlorobenzene	106467	0.8	IRIS – M
Diethanolamine	111422	0.003	CAL
1,4-Dioxane	123911	3.6	ATSDR
Ethyl benzene	100414	1	IRIS – L
Ethylene dibromide	106934	0.009	IRIS – M
Ethylene dichloride	107062	2.4	ATSDR
Ethylene glycol	107211	0.4	CAL
Formaldehyde	50000	0.0098	ATSDR
Glycol Ethers[20]	171	0.02	IRIS – M
- Ethylene glycol methyl ether	109864	0.02	IRIS – M
- Methoxytriglycol[15]	112356	0.02	IRIS – M
n-Hexane	110543	0.7	IRIS – M
Hydrochloric acid	7647010	0.02	IRIS – L
Hydrofluoric acid	7664393	0.014	CAL
Methanol	67561	4	CAL
Methyl chloride	74873	0.09	IRIS – M
Methyl isobutyl ketone	108101	3	IRIS – L/M
Methyl tert-butyl ether	1634044	3	IRIS – M
Methylene chloride	75092	1	ATSDR
Naphthalene	91203	0.003	IRIS – M

[18] The descriptors L (low), M (medium), and H (high) have been added for IRIS RfC values to indicate the overall level of confidence in the RfC value, as reported in the IRIS file.
[19] The value for cresols (mixed) was used as a surrogate.
[20] The value for ethylene glycol methyl ether was used as a surrogate for all glycol ethers.

RfC (or similar inhalation values) = an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Sources: IRIS = EPA Integrated Risk Information System, CAL = California EPA Office of Environmental Human Health Assessment, HEAST = EPA Health Effects Assessment Summary Table, ATSDR = US Agency for Toxic Substances and Disease Registry.

Pollutant	CAS Number[10]	RfC (mg/m3)	Source[18]
Pentachlorophenol	87865	0.1	CAL
Phenol	108952	0.2	CAL
Styrene	100425	1	IRIS – M
Tetrachloroethene	127184	0.27	ATSDR
Toluene	108883	5	IRIS – H
1,2,4-Trichlorobenzene	120821	0.2	HEAST
1,1,1-Trichloroethane	71556	1	CAL
Trichloroethylene	79016	0.6	CAL
Vinyl acetate	108054	0.2	IRIS – H
Vinyl chloride	75014	0.1	IRIS – M
Xylenes (mixed)	1330207	0.1	IRIS – M
m-Xylene[21]	108383	0.1	IRIS – M
o-Xylene[21]	95476	0.1	IRIS – M
p-Xylene[21]	106423	0.1	IRIS – M

2.2.6.2 Sources of acute dose-response information

Hazard identification and dose-response assessment information for acute exposure were based on OAQPS's existing recommendations for HAPs [*19*]. In contrast to the approach for chronic dose-response, no prioritization has been developed for acute noncancer reference values, in large part due to the lack of coverage across many chemicals by any one set of reference values specifically designed for this use. We looked to reference values developed for a variety of purposes, including Reference Exposure Levels (RELs), Acute Exposure Guideline Levels (AEGLs), and Emergency Response Planning Guideline (ERPGs) developed for 1-hour exposure durations.

The California Environmental Protection Agency (CalEPA) has developed acute dose-response assessments for many substances, expressing the results as acute inhalation reference exposure levels, or RELs.

> The acute REL (http://www.oehha.ca.gov/air/pdf/acuterel.pdf) is defined by CalEPA as "the concentration level at or below which no adverse health effects are anticipated for a specified exposure duration [*20*]. RELs are based on the most sensitive, relevant, adverse health effect reported in the medical and toxicological literature. RELs are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety. Since margins

[21] The RfC for mixed xylene was used as a surrogate.

> of safety are incorporated to address data gaps and uncertainties, exceeding the REL does not automatically indicate an adverse health impact."

The National Advisory Committee for Acute Exposure Guidelines (NAC-AEGL) is a Federal Advisory Committee Act committee consisting of representatives from multiple federal agencies, states, industry, non-governmental organizations, and several other nations that has been responsible for developing Acute Exposure Guideline Levels, or AEGLs. As described in their "Standing Operating Procedures (SOP) of the National Advisory Committee on Acute Exposure Guideline Levels for Hazardous Substances" (http://www.epa.gov/opptintr/aegl/pubs/sop.pdf), "the NRC's previous name for acute exposure levels — community emergency exposure levels (CEELs) — was replaced by the term AEGLs to reflect the broad application of these values to planning, response, and prevention in the community, the workplace, transportation, the military, and the remediation of Superfund sites." This document further states that AEGLs "represent threshold exposure limits for the general public and are applicable to emergency exposures ranging from 10 minute to 8 hours." The document lays out the purpose and objectives of AEGLs by stating that "the primary purpose of the AEGL program and the NAC/AEGL Committee is to develop guideline levels for once-in-a-lifetime, short-term exposures to airborne concentrations of acutely toxic, high-priority chemicals." In detailing the intended application of AEGL values, the document states that "It is anticipated that the AEGL values will be used for regulatory and nonregulatory purposes by U.S. Federal and State agencies, and possibly the international community in conjunction with chemical emergency response, planning, and prevention programs. More specifically, the AEGL values will be used for conducting various risk assessments to aid in the development of emergency preparedness and prevention plans, as well as real-time emergency response actions, for accidental chemical releases at fixed facilities and from transport carriers." The NAC-AEGL defines AEGL-1 and AEGL-2 as:

> "AEGL-1 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic nonsensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure."

> "AEGL-2 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape."

> "Airborne concentrations below AEGL-1 represent exposure levels that can produce mild and progressively increasing but transient and nondisabling odor, taste, and sensory irritation or certain asymptomatic, nonsensory effects. With increasing airborne concentrations above each AEGL, there is a progressive increase in the likelihood of occurrence and the severity of effects described for each corresponding AEGL. Although the AEGL values represent threshold levels for the general public, including susceptible subpopulations, such as infants, children, the elderly, persons with asthma, and those with other illnesses, it is recognized that individuals, subject to unique or idiosyncratic responses, could experience the effects described at concentrations below the corresponding AEGL."

The American Industrial Hygiene Association (AIHA) has developed emergency response planning guidelines (ERPGs) [*21*] for acute exposures at three different levels of severity. These guidelines represent concentrations for exposure of the general population for up to 1 hour associated with effects expected to be mild or transient (ERPG-1), irreversible or serious (ERPG-2), and potentially life-threatening (ERPG-3).

ERPG values (http://www.aiha.org/1documents/Committees/ERP-erpglevels.pdf) are described in their supporting documentation as follows: "Emergency Response Planning Guidelines (ERPGs) were developed for emergency planning and are intended as health based guideline concentrations for single exposures to chemicals. These guidelines (*i.e.*, the ERPG Documents and ERPG values) are intended for use as planning tools for assessing the adequacy of accident prevention and emergency response plans, including transportation emergency planning and for developing community emergency response plans. The emphasis is on ERPGs as planning values: When an actual chemical emergency occurs there is seldom time to measure airborne concentrations and then to take action." ERPG-1 and ERPG-2 values are defined by AIHA as follows:

> "ERPG-1 is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing other than mild transient adverse health effects or without perceiving a clearly defined, objectionable odor."
>
> "ERPG-2 is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action."

The emissions inventory for the petroleum refining source category includes emissions of 34 HAP with relevant and available quantitative acute dose-response threshold values. These HAPs, the acute threshold values, and the source of the value are listed below in Table 2-5.

Table 2-5. Dose-Response Values for Acute Exposure

Pollutant	CAS Number[10]	AEGL-1 (1-hr) (mg/m^3)	AEGL-2 (1-hr) (mg/m^3)	ERPG-1 (mg/m^3)	ERPG-2 (mg/m^3)	REL (mg/m^3)
Acetaldehyde	75070	81	490	81	490	0.47
Acrylonitrile	107131	10	130	22	77	
Aniline	62533	30	46			
Benzene	71432	170	2600	170	2600	1.3
Biphenyl	92524		61			
1,3-Butadiene	106990	1500	12000	1500	12000	
Carbon disulfide	75150	40	500	40	500	6.2
Carbon tetrachloride	56235	280	1200	280	1200	1.9
Chlorobenzene	108907	46	690			
Chloroform	67663		310		310	0.15
Cumene	98828	250	1500			
1,4-Dioxane	123911	61	1200			3
Ethylene dibromide	106934	130	180			
Ethylene dichloride	107062			200	810	
Formaldehyde	50000	1.1	17	1.1	17	0.094
Glycol Ether[22]	171					0.093
- Ethylene glycol methyl ether	109864					0.093
- Methoxytriglycol[16]	112356					0.093
n-Hexane	110543		12000			
Hydrochloric acid	7647010	2.7	33	2.7	33	2.1
Hydrofluoric acid	7664393	0.82	20	0.82	20	0.24
Methanol	67561	690	2700	690	2700	28
Methyl chloride	74873		1900		1900	
Methylene chloride	75092	690	1900	690	1900	14
Methyl tert-butyl ether	1634044	180	2100			
Phenol	108952	58	89	58	89	5.8
Styrene	100425	85	550	85	550	21
Tetrachloroethene	127184	240	1600	240	1600	20
Toluene	108883	750	4500	750	1900	37
1,1,1-Trichloroethane	71556	1300	3300	1300	3300	68
Trichloroethylene	79016	700	2400	700	2400	
Vinyl acetate	108054	24	630	18	260	
Vinyl chloride	75014	640	3100	640	3100	180
m-xylene	108383					22
p-xylene	106423					22
Xylenes (mixed)	1330207	560	4000			22

[22] The value for ethylene glycol methyl ether was used as a surrogate for all glycol ethers.

2.2.7 Risk characterization

2.2.7.1 General

The final product of the risk assessment is the risk characterization, in which the information from the previous steps is integrated and an overall conclusion about risk is synthesized that is complete, informative, and useful for decision makers. In general, the nature of this risk characterization depends on the information available, the application of the risk information and the resources available. In all cases, major issues associated with determining the nature and extent of the risk are identified and discussed. Further, the EPA Administrator's March 1995 *Policy for Risk Characterization* [*22*] specifies that a risk characterization "be prepared in a manner that is clear, transparent, reasonable, and consistent with other risk characterizations of similar scope prepared across programs in the Agency." These principles of transparency and consistency have been reinforced by the Agency's *Risk Characterization Handbook* [*23*], in 2002 by the Agency's information quality guidelines [*24*], and in the OMB/OSTP September 2007 Memorandum on Updated Principles for Risk Analysis[23], and are incorporated in these assessments.

Estimates of health risk are presented in the context of uncertainties and limitations in the data and methodology. Through our tiered, iterative analytical approach, we have attempted to reduce both uncertainty and bias to the greatest degree possible in this assessment. We have provided summaries of risk metrics for the source category (including maximum individual cancer risks and noncancer hazards, as well as cancer incidence estimates) along with a discussion of the major uncertainties associated with their derivation to provide decision makers with the fullest picture of the assessment and its limitations.

For each carcinogenic HAP included in this assessment that has a potency estimate available, individual and population cancer risks were calculated by multiplying the corresponding lifetime average exposure estimate by the appropriate URE. This calculated cancer risk is defined as the upper-bound probability of developing cancer over a 70-year period (*i.e.,* the assumed human lifespan) at that exposure. EPA's upper bound estimates represent a "plausible upper limit to the true value of a quantity" (although this is usually not a true statistical confidence limit).[24] In some circumstances, the true risk could be as low as zero; however, in other circumstances the risk could also be greater.

Because EPA has determined that two of the carcinogens listed in Table 2-2 (*i.e.,* POM and vinyl chloride) have a mutagenic mode of action, [*25*], EPA's *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* [*26*] was applied to this assessment. This guidance has the effect of increasing the mutagens' UREs by factors of 10 (for children aged 0-1), 3 (for children aged 2-15), or 1.6 (for 70 years of exposure beginning at birth), as appropriate for the exposed population. In this case, this has the effect of increasing the estimated life time risks for these pollutants by a factor of 1.6. In addition, although only a small

[23] Memorandum for the Heads of Executive Departments and Agencies - Updated Principles for Risk Analysis (September 19, 2007), From Susan E. Dudley, Administrator, Office of Information and Regulatory Affairs, Office of Management and Budget; and Sharon L. Hays, Associate Director and Deputy Director for Science, Office of Science and Technology Policy (http://georgewbush-whitehouse.archives.gov/omb/memoranda/fy2007/m07-24.pdf)

[24] IRIS glossary (www.epa.gov/NCEA/iris/help_gloss.htm).

fraction of the total POM emissions were reported as individual compounds, EPA expresses carcinogenic potency for compounds in this group in terms of benzo[a]pyrene equivalence, based on evidence that carcinogenic POM have the same mutagenic mechanism of action as does benzo[a]pyrene. For this reason EPA implementation policy [*27*] recommends applying the *Supplemental Guidance* to all carcinogenic PAHs for which risk estimates are based on relative potency. Accordingly, we have applied the Supplemental Guidance to all unspeciated POM mixtures.

Increased cancer incidence for the entire receptor population within the area of analysis was estimated by multiplying the estimated lifetime cancer risk for the average individual within each census block by the number of individuals residing in that block, then summing the results for all modeled census blocks. This lifetime population incidence estimate was divided by 70 years to obtain an estimate of the number of cancer cases per year for the entire modeling domain.

In the case of benzene, the high end of the reported cancer URE range was used in our assessment to provide a conservative estimate of potential cancer risks. Use of the high end of the range provides risk estimates that are approximately 3.5 times higher than use of the equally-plausible low end value. Use of the low end of the range and its impact on risk estimates is included as a sensitivity analysis in the discussion of uncertainties.

Unlike linear dose-response assessments for cancer, noncancer health hazards generally are not expressed as a probability of an adverse occurrence. Instead, "risk" for noncancer effects is expressed by comparing an exposure to a reference level as a ratio. The "hazard quotient" (HQ) is the estimated exposure divided by a reference level (*e.g.,* the RfC). For a given HAP, exposures at or below the reference level (HQ≤1) are not likely to cause adverse health effects. As exposures increase above the reference level (HQs increasingly greater than 1), the potential for adverse effects increases. For exposures predicted to be above the RfC, the risk characterization includes the degree of confidence ascribed to the RfC values for the compound(s) of concern (*i.e.,* high, medium, or low confidence) and discusses the impact of this on possible health interpretations.

The risk characterization for chronic effects other than cancer is expressed in terms of the HQ for inhalation, calculated for each HAP at each census block centroid. As discussed above, RfCs incorporate generally conservative uncertainty factors in the face of uncertain extrapolations, such that an HQ greater than one does not necessarily suggest the onset of adverse effects. The HQ cannot be translated to a probability that adverse effects will occur, and is unlikely to be proportional to adverse health effect outcomes in a population.

Screening for potentially significant acute inhalation exposures also followed the HQ approach. In this case, we divided the maximum estimated acute exposure by each available short-term threshold value to develop an array of HQ values relative to the various acute endpoints and thresholds. In general, when none of these HQ values are greater than one, there is no potential for acute risk. In those cases where HQ values above one are seen, additional information is used to determine if there is a potential for significant acute risks.

2.2.7.2 Mixtures

Since most or all receptors in these assessments receive exposures to multiple pollutants rather

than a single pollutant, we estimated the aggregate health risks associated with all the exposures from a particular source category combined.

To combine risks across multiple carcinogens, this assessment used the EPA mixtures guidelines' [*28, 29*] default assumption of additivity of effects, and combined risks by summing them using the independence formula in the mixtures guidelines.

In assessing noncancer hazard from chronic exposures, in cases where different pollutants cause adverse health effects via completely different modes of action, it may be inappropriate to aggregate HQs. In consideration of these mode-of-action differences, the mixtures guidelines support aggregating effects of different substances in specific and limited ways. To conform to these guidelines, we aggregated non-cancer HQs of HAPs that act by similar toxic modes of action, or (where this information is absent) that affect the same target organ. This process creates, for each target organ, a target-organ-specific hazard index (TOSHI), defined as the sum of hazard quotients for individual HAPs that affect the same organ or organ system. All TOSHI calculations presented here were based exclusively on effects occurring at the "critical dose" (*i.e.,* the lowest dose that produces adverse health effects). Although HQs associated with some pollutants have been aggregated into more than one TOSHI, this has been done only in cases where the critical dose affects more than one target organ. Because impacts on organs or systems that occur above the critical dose have not been included in the TOSHI calculations, some TOSHIs may have been underestimated. As with the HQ, the TOSHI should not be interpreted as a probability of adverse effects, or as strict delineation of "safe" and "unsafe" levels. Rather, the TOSHI is another measure of the potential for adverse health outcomes associated with pollutant exposure, and health scientists and risk managers should take care to clearly communicate its uncertainties and limitations when characterizing risks.

Because of the conservative nature of the acute inhalation screening approach and the transient nature of emissions fluctuations and potential exposures, acute impacts were screened on an individual pollutant basis, not using the TOSHI approach.

2.3 Results Summary and Risk Characterization

In this section, the results of the risk assessment for the petroleum refining MACT 1 source category are presented in terms of the following information:

1) A narrative description of the source category, including a discussion of the processes involved and the number of facilities EPA knows or expects are affected by the petroleum refinery MACT 1 standard;

2) A table of emissions for the entire category showing HAP emitted, total source category emission rates for each HAP, and numbers of facilities reporting emissions of each HAP;

3) A table summarizing the chronic inhalation risk results showing the number of facilities modeled, the number of people within 50 km, the MIR for the entire source category, the number of facilities for which the facility-specific MIR exceeds specific cancer and noncancer benchmarks, the number of people for whom the risks exceed the same benchmarks, the estimated total cancer incidence, and identifying the specific HAPs

contributing the most to those risks (HAPs identified as "drivers" include those contributing the most to the risk metric, up to 90% of its value). In addition, this table indicates the maximum HQ from the acute inhalation screening and an indication of how many facilities showed HQ values above 1;

4) In those cases where the acute inhalation screening showed an HQ value greater than 1 for any combination of source and pollutant, a table summarizing the acute screening results showing available acute dose-response values for each affected pollutant, for three effect levels (none, mild, and severe), if available, the maximum acute screening exposure estimated, and the associated HQ values;

5) A narrative summarizing the risk characterization for the entire source category.

Detailed facility-level results for both chronic and acute inhalation risk assessments can be found in Appendix D.

2.3.1 Source Category Description and Summary of Emissions

Petroleum Refineries are facilities engaged in refining and producing products made from crude oil or unfinished petroleum derivatives including gasoline, naphtha, kerosene, jet fuels, distillate fuel oils, residual fuel oils, and lubricants. In the list of MACT source categories (57 FR 31576, July 16, 1992), EPA listed two separate and distinct petroleum refinery source categories: (1) Petroleum Refineries - Catalytic Cracking (Fluid and Other) Units, Catalytic Reforming Units, and Sulfur Plant Units and (2) Petroleum Refineries - Other Sources Not Distinctly Listed. The MACT standard for the "Other Sources Not Distinctly Listed" source category (40 CR 63, subpart UU) was promulgated first, on August 18, 1995 in 60 FR 43244,. Therefore, it is commonly referred to as Petroleum Refineries MACT 1. MACT 2, which addresses the Petroleum Refineries - Catalytic Cracking (Fluid and Other) Units, Catalytic Reforming Units, and Sulfur Plant Units source category, was promulgated on April 11, 2002 (67 FR 17761).

Because MACT 1 and MACT 2 represent two separate and distinct source categories which were subjected to MACT standards at different times, EPA will assess the residual risk and make decisions on future regulations under section 112(f)(2) of the CAA independently. The data presented in this document are only for MACT 1, the "Petroleum Refineries, Other Sources Not Distinctly Listed" source category. Residual risk for MACT 2, Petroleum Refineries - Catalytic Cracking (Fluid and Other) Units, Catalytic Reforming Units, and Sulfur Plant Units, will be assessed by EPA at a later date in a later phase of RTR.

The petroleum refinery process units covered by MACT 1 include, but are not limited to, thermal cracking, vacuum distillation, crude distillation, hydroheating and hydrorefining, isomerization, polymerization, lube oil processing, and hydrogen production. Emissions originate from various process vents, storage vessels, wastewater streams, loading racks, marine tank vessel loading operations, and equipment leaks associated with refining facilities.

To create the ANPRM data set for Petroleum Refineries MACT 1, EPA started by retrieving all facilities identified by the Petroleum Refineries Other Sources Not Distinctly Listed MACT code (MACT Code 0503) in Version 1.0 of the 2002 NEI (February 2006). Next, we performed an

engineering review of these facilities and updated the dataset with site-specific benzene emissions data for 22 refineries as provided by the American Petroleum Institute. The goal of the engineering review was to identify readily-apparent limitations and issues with the emissions data and to make changes to the dataset where possible to address these issues and decrease the uncertainties associated with the assessment. EPA requested comments on the adjusted 2002 NEI data as part of the Risk and Technology Review (RTR) Phase II ANPRM in March 2007 (72FR14734), making it available for a 60-day public comment period. Comments and corrections were evaluated and incorporated into the inventory. A detailed discussion of the changes to the inventory as a result of the ANPRM process and the risk characterization effort are presented in the Draft Residual Risk Assessment for MACT I Petroleum Refining Sources and the Petroleum Refineries NPRM Data Input File available in the Risk and Technology Review Docket, ID No. EPA-HQ-OAR-2006-0895 at www.regulations.gov.

In August 2007, a NPRM was published making the source category dataset available for a second 60-day comment period, which was subsequently re-opened for another 50 days. Again the comments and corrections were evaluated and incorporated into the inventory. The final petroleum refinery database contained information for 156 facilities, and this is thought to represent the entire source category. Total HAP emissions did not change dramatically as a result of these comments, dropping by only about 2%. Notably, emissions of metal HAP were removed from the inventory since they cannot be emitted by the specific emission points covered by the petroleum refinery MACT 1. Instead, these emissions are thought to be emitted by the emission points covered by the petroleum refinery MACT 2. Details on the development of the emissions and source data for this source category are discussed in Section 2.2.1. The emissions data and modifications made to the NEI data are available in the Petroleum Refineries Baseline Data Input File available in the Risk and Technology Review Docket, ID No. EPA-HQ-OAR-2006-0895 at www.regulations.gov.

We also note that recent Canadian and European studies [*30*,*31*] indicate that emissions from some refineries are significantly higher than amounts estimated using standard techniques such as emission factors or AP-42 equations. This bias is apparently caused by omission (*e.g.*, process leaks into cooling towers) or mischaracterization of significant emission sources, and the same quantification issues appear to exist in the US. We have performed additional analyses (*i.e.*, model plant analysis and model-to-monitor comparison) in an attempt to characterize the possible magnitude of uncertainty in emissions estimates. The model-to-monitor analysis suggests that we may be underestimating emissions of benzene at two refineries in the Houston area by a factor of 2. The model plant analysis suggests that we may be underestimating risk by up to a factor of 3. Technical memoranda explaining these analyses can be found in the Docket under "Statistical Comparison of Monitored and Modeled Ambient Benzene Concentrations Near Two Petroleum Refineries in Texas City, TX" and "Model Plant Analysis of Residual Risk From Petroleum Refinery Emissions."

Organic chemicals account for the majority of the total mass of HAPs emitted by MACT 1 petroleum refinery sources, with toluene, benzene, xylene,, hexane, methanol, ethyl benzene, methyl isobutyl ketone, 2,2,4-trimethylpentane, methyl tert-butyl ether, hydrogen fluoride, naphthalene, diethanolamine, cumene, 1,3-butadiene, carbonyl sulfide, phenol, hydrochloric acid, hydrogen fluoride, cresols, tetrachloroethylene, ethylene glycol, chloroform,

trichloroethylene, 16- polycyclic aromatic hydrocarbons (PAH), and polycyclic organic matter accounting for 99 percent of the HAPs mass emitted across the source category. A range of persistent and bioaccumulative HAP (PB HAP) [*32*] emissions were included in the NPRM dataset, including various PAH.

The final petroleum refinery database contained information for 156 facilities, and this is thought to represent the source category in its entirety. The emissions data and modifications made to the NEI data are available in the Petroleum Refineries Baseline Data Input File available in the Risk and Technology Review Docket, ID No. EPA-HQ-OAR-2006-0895 at www.regulations.gov. Table 2-6 provides information summarizing emissions for this source category.

Comments received on the emission inventory used for the draft baseline risk assessment for petroleum refinery MACT 1 sources were evaluated and incorporated into the final inventory if deemed appropriate and reasonable from an engineering standpoint. The comments covered 101 facilities, and included data provided for three facilities not contained in the original dataset. After evaluating the comments, emissions data were corrected at 48 facilities, emission point identifiers were corrected at 3 facilities, stack parameters were revised at 4 facilities, and location data were corrected at 61 facilities. The final petroleum refinery emission inventory contains information for 156 facilities representing the entire source category.

Nationwide refinery HAP emission estimates did not change dramatically as a result of the revisions made pursuant to the public comments, dropping by only about 2 percent. In addition, metal HAP emissions were removed from the inventory because they are not emitted by the emission points covered by the petroleum refinery MACT 1. Metal HAPs are emitted by other source categories in refineries, most notably by the emission points covered by the petroleum refinery MACT 2. These metal HAP emissions will be included in the RTR assessment for that category. Appendix A provides a comparison of the risk estimates for this source category, before and after processing the NPRM revisions.

Table 2-6. Summary of Emissions from the MACT 1 Petroleum Refining Source Category

HAP[a]	Emissions (tpy)	Number of Facilities Reporting HAP (156 facilities in data set)	Prioritized Inhalation Dose-Response Value Identified by OAQPS[b]			PB-HAP?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	Health Benchmark Values for Acute Noncancer?	
Toluene	1,784	136		✓	✓	
Xylenes (Mixture of o, m, and p Isomers)	1,060	129		✓	✓	
Hexane	1,047	130		✓	✓	
Benzene	690	146	✓	✓	✓	
Methanol	569	61		✓	✓	
Methyl Tert-Butyl Ether	349	45	✓	✓	✓	
p-Xylene	337	13		✓	✓	
Ethyl Benzene	251	130		✓		
m-Xylene	138	17		✓	✓	

Table 2-6. Summary of Emissions from the MACT 1 Petroleum Refining Source Category

HAP[a]	Emissions (tpy)	Number of Facilities Reporting HAP (156 facilities in data set)	Prioritized Inhalation Dose-Response Value Identified by OAQPS[b]			PB-HAP?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	Health Benchmark Values for Acute Noncancer?	
2,2,4-Trimethylpentane	132	47				
Methyl Isobutyl Ketone	92	5		✓		
Naphthalene	82	104	✓	✓		
Hydrochloric Acid	73	19		✓	✓	
o-Xylene	72	21		✓		
Hydrogen Fluoride	53	34		✓	✓	
Cumene	53	81		✓	✓	
Diethanolamine	41	22		✓		
Phenol	32	42		✓	✓	
Ethylene Glycol	22	8		✓		
1,3-Butadiene	17	71	✓	✓	✓	
Cresol	16	27		✓		
Tetrachloroethylene	15	34	✓	✓	✓	
Formaldehyde	9	28	✓	✓	✓	
16-PAH	8	2				✓
Styrene	5	25		✓	✓	
Fluoranthene	5	10				✓
PAH, total	4	45				✓
Polycyclic Organic Matter	4	9	✓			✓
Carbon Disulfide	4	15		✓	✓	
Biphenyl	3	21			✓	
Carbon Tetrachloride	3	5	✓	✓	✓	
Glycol Ethers	3	4		✓	✓	
Carbonyl Sulfide	2	16				
Anthracene	1	8				✓
1,1,1-Trichloroethane	1	5		✓	✓	
Ethylene Dibromide	0.7	8	✓	✓	✓	
Ethylene Dichloride	0.7	11	✓	✓	✓	
Chloroform	0.6	7		✓	✓	
Phenanthrene	0.6	10				✓
Trichloroethylene	0.6	5	✓	✓	✓	
Vinyl Acetate	0.5	3		✓	✓	
Benzo[g,h,i,]Perylene	0.2	23				✓
Methylene Chloride	0.2	4	✓	✓	✓	
Acetaldehyde	0.2	14	✓	✓	✓	
Chlorobenzene	0.1	4		✓	✓	
Vinyl Chloride	0.1	1	✓	✓	✓	
Acetophenone	0.08	1				
Quinoline	0.04	1				

Table 2-6. Summary of Emissions from the MACT 1 Petroleum Refining Source Category

HAP[a]	Emissions (tpy)	Number of Facilities Reporting HAP (156 facilities in data set)	Prioritized Inhalation Dose-Response Value Identified by OAQPS[b]			PB-HAP?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	Health Benchmark Values for Acute Noncancer?	
p-Phenylenediamine	0.03	1				
Dibenzofuran	0.03	2				✓
Aniline	0.03	1	✓	✓	✓	
1,4-Dioxane	0.01	2	✓	✓	✓	
Methyl Chloride	0.01	2		✓	✓	
Ethylene Glycol Methyl Ether	0.007	2		✓	✓	
1,1,2,2-Tetrachloroethane	0.005	1	✓			
Pentachlorophenol	0.002	1	✓	✓		
Acrylonitrile	0.002	1	✓	✓	✓	
Bis(2-Ethylhexyl)Phthalate	0.001	3	✓	✓		
Methoxytriglycol	0.001	1		✓	✓	
Benzo[a]Pyrene	0.0006	4	✓			✓
m-Cresol	0.0005	1		✓		
1,4-Dichlorobenzene	0.0003	2	✓	✓		
1,2,4-Trichlorobenzene	0.0003	1		✓		
Benzo[k]Fluoranthene	0.00005	2	✓			✓
Chrysene	0.00003	3	✓			✓
Benz[a]Anthracene	0.00002	2	✓			✓
Dibenzo[a,h]Anthracene	0.000004	2	✓			✓
Benzo[b]Fluoranthene	0.000002	3	✓			✓
Fluorene	0.0000007	2				✓
Pyrene	0.0000002	2				✓
Acenaphthene	0.0000002	1				✓
Perylene	0.0000001	1				✓
Indeno[1,2,3-c,d]Pyrene	0.00000003	2	✓			✓

[a] Notes for how HAP were speciated for risk assessment:

- For emissions of any chemicals or chemical groups classified as polycyclic organic matter (POM), emissions were grouped into POM subgroups as found on the EPA's Technology Transfer Network website for the 1999 National-Scale Air Toxics Assessment at http://www.epa.gov/ttn/atw/nata1999/nsata99.html. Those that are grouped and do not have individual dose-response values are not checked in the table above.
- For emissions reported generically as "Glycol Ethers" or as specific glycol ethers not found on EPA's Technology Transfer Network website for air toxics (see footnote b), emissions will be treated as ethylene glycol methyl ether.

[b] Specific dose-response values for each chemical are identified in section 2.2.6 of this document and on EPA's Technology Transfer Network website for air toxics at http://www.epa.gov/ttn/atw/toxsource/summary.html. The acute benchmarks considered were the REL, AEGL-1 (1-hour), ERPG-1, AEGL-2 (1-hour), and ERPG-2.

2.3.2 Source Category Inhalation Risk Assessment Results

The petroleum refining source category consists of 156 facilities, all of which were included in this risk assessment. Refineries are located throughout the United States; we estimate that approximately 90 million people live within 50 kilometers of at least one petroleum refinery.

Table 2-7. Summary of Source Category Level Risks for Petroleum Refineries

Result		HAP "Drivers"
Facilities in Source Category		
Number of Facilities Estimated to be Subject to MACT in Source Category	156	n/a
Number of Facilities Identified in NEI and Modeled in Screening Risk Assessment	156	n/a
Cancer Risks		
Maximum Individual Lifetime Cancer Risk (in 1 million) from any Facility in the Category	30	naphthalene, POM
Number of Facilities with Maximum Individual Lifetime Cancer Risk:		
Greater than or equal to 100 in 1 million	0	n/a
Greater than or equal to 10 in 1 million	5	naphthalene, POM, benzene, ethylene dibromide, 1,3-butadiene, tetrachloroethylene, methyl tert-butyl ether, carbon tetrachloride
Greater than or equal to 1 in 1 million	77	naphthalene, POM, benzene, ethylene dibromide, 1,3-butadiene, tetrachloroethylene, methyl tert-butyl ether, carbon tetrachloride, ethylene dichloride, vinyl chloride
Chronic Noncancer Risks		
Maximum Respiratory Hazard Index	0.3	diethanolamine
Number of Facilities with Maximum Respiratory Hazard Index:		
Greater than 1	0	n/a
Acute Noncancer Screening Results		
Maximum Acute Hazard Quotient	50, 20, 6	Benzene (REL) hydrofluoric acid (REL, AEGL-1/ERPG-1)
Number of Facilities With Potential for Acute Effects	20	benzene, hydrofluoric acid,
Acute Noncancer Refined Results		
Maximum Acute Hazard Quotient	8, 0.06 5, 2, 0.06	benzene (REL, AEGL-1/ERPG-1) hydrofluoric acid (REL, AEGL-1/ERPG-1, AEGL-2/ERPG-2)
Number of Facilities With Potential for Acute Effects	8	benzene, hydrofluoric acid
Population Exposure		
Number of People Living Within 50 Kilometers of Facilities Modeled	90,000,000	n/a
Number of People Exposed to Cancer Risk:		
Greater than or equal to 100 in 1 million	0	n/a
Greater than or equal to 10 in 1 million	4,000	n/a
Greater than or equal to 1 in 1 million	460,000	n/a
Number of People Exposed to Noncancer Respiratory Hazard Index:		
Greater than 1	0	n/a
Estimated Cancer Incidence (excess cancer cases per year)	0.03 to 0.05	n/a

Table 2-7. Summary of Source Category Level Risks for Petroleum Refineries

Result		HAP "Drivers"
Facilities in Source Category		
Contribution of HAP to Cancer Incidence		
benzene	48%	n/a
naphthalene	21%	n/a
POM	15%	n/a
1,3-butadiene	5%	n/a
tetrachloroethylene	4%	n/a

Table 2-8. Summary of Acute Refined Results for Petroleum Refineries

Refined Results		MAXIMUM ACUTE HAZARD QUOTIENTS			ACUTE DOSE-RESPONSE VALUES				
HAP	Max. 1-hr. Air Conc. (mg/m^3)	Based on REL	Based on AEGL-1/ERPG-1	Based on AEGL-2/ERPG-2	REL (mg/m^3)	AEGL-1 (1-hr) (mg/m^3)	ERPG-1 (mg/m^3)	AEGL-2 (1-hr) (mg/m^3)	ERPG-2 (mg/m^3)
benzene	10	8	0.06	0.004	1.3	170	170	2600	2600
hydrofluoric acid	1.3	5	2	0.06	0.24	0.82	0.82	20	20

Notes on Process:

1) Acute screening was performed for all emitted HAP with available acute dose-response values. Where acute screening HQ values exceeded 1, refined analysis was performed. Only those pollutants whose refined HQs were equal to or greater than 1 for at least one acute threshold value are shown in the table.
2) HAP with available acute dose-response values which are not in the table do not carry any potential for posing acute health risks, based on an analysis of currently available emissions data.

Notes on Acute Dose-Response Values:

REL – California EPA reference exposure level for no adverse effects. Most, but not all RELs are for 1-hour exposures.

AEGL – Acute Exposure Guideline Levels represent exposure (1-hour) limits for the general public.

AEGL-1 is the exposure level above which it is predicted that the general population, including susceptible individuals, could experience effects that are notable discomfort, but which are transient and reversible upon cessation of exposure.

AEGL-2 is the exposure level above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.

ERPG – Emergency Response Program Guidelines represent emergency exposure (1-hour) limits for the general public.

ERPG-1 is the maximum level below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing other than mild, transient adverse health effects.

ERPG-2 is the maximum exposure below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action.

2.3.3 Risk Characterization

The maximum individual cancer risk for the petroleum refining source category is 30 in a million. The maximum individual cancer risk for the source category as a whole is dominated by the risks associated with emissions of naphthalene and polycyclic organic matter (POM); however, the maximum individual cancer risk level associated with each facility and the specific pollutants which contribute to most to that level vary significantly from facility to facility. The total cancer incidence for the source category was estimated to be between 0.03 and 0.05 cancer cases per year, or about 1 case in every 20 to 30 years (this range of cancer incidence depends on the range of the IRIS cancer potency factors for benzene, each end of which is considered equally plausible). The cancer incidence for the source category is dominated by risks associated

with benzene and naphthalene. The estimated maximum individual cancer risk exceeded 10 in a million at 5 facilities and exceeded 1 in a million at 77 facilities. Based on the assumption that all individuals are exposed for 70 years, approximately 4,000 people were estimated to have cancer risks above 10 in a million and approximately 460,000 people were estimated to have cancer risks above 1 in a million.

Chronic noncancer inhalation risks were not identified as significant, with the maximum chronic target organ specific hazard index associated with the cumulative impacts of all noncarcinogenic HAP emitted by these sources being less than 1. While there were reported emissions of one persistent HAP (polycyclic organic matter, or POM) from this source category, our multipathway screening indicated that neither significant ingestion health risks nor environmental risks would be anticipated to result from exposures to media concentrations associated with the deposition of these emissions. No other potential environmental risks, including those as a direct result of exposure of flora and fauna to ambient air concentrations, were identified.

As mentioned in the discussion of dose-response values, we calculated benzene risks throughout this assessment using the upper end of the range of cancer unit risk estimates, or URE, identified in IRIS. Specifically, IRIS recommends a range of URE for benzene, 2.2×10^{-6} to 7.8×10^{-6} per $\mu g/m^3$, explaining that each has equal scientific plausibility. Since benzene is an important risk driver for many petroleum refineries, we also estimated the risk using the lower end of this range to assess the impact of URE choice on the final results. However, we found that the maximum individual cancer risk (MIR) is driven by pollutants other benzene. Thus, the choice of benzene URE was seen to have little impact on the MIR for the source category. Additionally, without re-assessing the risks for each facility, we made a very rough projection of the impact of the benzene URE on the number of people whose individual risks are above 1 in a million, and estimated that use of the low end URE may reduce this population from 460,000 to about 275,000. Since benzene emissions are prevalent throughout the source category, however, total incidence estimates were seen to drop on average by about 35% (to 0.03 cases per year) when the low end URE was chosen.

While maximum individual cancer risks vary significantly from facility to facility (see Appendix D), they are typically dominated by risks from fugitive emissions which are responsible for about 52 percent the cancer risk. Leaks into process cooling water which are ultimately released to the atmosphere through cooling towers were not seen to contribute significantly to the emissions inventory or to cancer risks. However, recent studies [*30, 31*] suggest that these emissions, among others, may be underestimated and underreported in current emissions inventories, but to an unknown extent.

The initial acute screening risk calculations suggested that 20 petroleum refineries showed potential 1-hour exposures above an acute health benchmark, but the lack of readily available detailed property boundary information for many of the facilities evaluated made it difficult to determine whether the points of maximum concentration were on- or off-site. The facilities that exceeded an acute HQ of 1 were targeted for more refined evaluation. The refined evaluation included inspecting aerial maps of the sites to see if the locations for predicted potential exceedances occurred inside or outside the facility boundary. While exact facility boundaries

were not visible, the aerial photographs allowed us to assess locations likely to be accessible to the public. Results of these mapping efforts can be found in Appendix E.

Potential acute impacts of concern were identified in the acute inhalation screening assessment for facilities emitting benzene and hydrofluoric acid. Emissions of each of these pollutants showed the potential to create maximum offsite impacts corresponding to 50 and 20 times the acute REL, respectively (*i.e.*, for benzene, HQ_{REL}=50; for hydrofluoric acid, HQ_{REL}=20). One potential exceedance of an AEGL value was identified for hydrofluoric acid (HQ_{AEGL-1}=6). Subsequent refinement to the acute analysis discussed below, indicates the potential for acute concerns at 8 out of the 156 facilities, with maximum potential offsite impacts at 8 and 5 times the acute reference exposure level (REL) for benzene and hydrofluoric acid (*i.e.*, for benzene, maximum HQ_{REL}= 8, 5 facilities with potential HQ_{REL} greater than 1; and for hydrofluoric acid, maximum HQ_{REL}=5, 3 facilities with potential HQ_{AEGL-1} greater than 1), and a potential exceedance of the acute exposure guideline level (AEGL-1) and the emergency response planning guideline (ERPG-1) level for hydrofluoric acid (HQ_{AEGL-1} = HQ_{ERPG-1} = 2) at one facility. There were no potential exceedances of the AEGL-1 or the ERPG-1 levels for benzene (maximum HQ_{AEGL-1} = HQ_{ERPG-1} = 0.06). There were also no potential exceedances of the AEGL-2 level for hydrofluoric acid (maximum HQ_{AEGL-2} = 0.06). According to CalEPA, acute exposure to hydrofluoric acid can be associated with eye and respiratory irritation and acute exposure to benzene can be associated with reproductive/developmental effects (see http://www.oehha.ca.gov/air/pdf/acuterel.pdf). Maximum predicted acute HQ values for each of the facilities are presented in Appendix D and the refined acute analysis and results are presented in Appendix E. We note that the number of facilities with potential acute concerns (8) is small relative to the total number of facilities in the source category (156). The number of people living within a mile of the 5 sites with potential acute benzene impacts is about 3000; the number of people living within a mile of the 3 sites with potential acute hydrofluoric acid impacts is about 8000. Concerning potential acute benzene exposures, while the maximum benzene HQ_{REL} value is 8, the corresponding HQ_{AEGL-1} value is 0.06. This places estimated acute exposures in a "gray area" that is well below the level "above which the general population, including sensitive individuals, could experience notable discomfort, irritation, or certain asymptomatic, nonsensory effects" (*i.e.*, the AEGL-1), but still well above the level at which we can rule out the possibility of acute health impacts (*i.e.*, the REL). Regarding potential acute hydrofluoric acid exposures, we note that the source of the emissions is fugitive emissions, indicating that the reported emissions are estimates based on long-term consideration of leaking pipes, equipment, *etc.* In general, such emissions do not vary dramatically in time, and our use of the emissions multiplier of 10 in estimating acute exposures from long-term average emissions estimates is likely conservative. We note that our screening indicates no potential to exceed the AEGL-2 level for hydrofluoric acid, defined as an exposure level "above which the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape." We conclude that short-term exceedances of the AEGL-1/ERPG-1 level are possible, but unlikely for 1 facility and that HQ_{REL} values greater than 1 may still be possible for 3 facilities, indicating that we cannot completely rule out acute exposures of concern at these facilities.

It is important to note that acute risk estimates were based on the annual emission rate multiplied by a factor of 10. We were not able to refine our estimates of peak emission rates beyond the

default factor of 10 times the annual average hourly rate, nor were we able to simulate the typical distribution of peak emission events between emission points at a facility, making the final results of our acute assessments uncertain but probably conservative. Overall, these results neither prove that adverse acute health effects will occur, nor do they rule out the possibility, should all the assumed conditions of exposure (*i.e.*, simultaneous tenfold emission rate, worst-case meteorology, and presence of a human receptor) be met.

In addition to the inhalation risk results discussed above, human health multipathway risks were evaluated using screening techniques for POM emissions. These results indicated that the potential for significant cancer or noncancer human health risks due to the ingestion of these pollutants was low. Only a small fraction of the POM mass reported for the facilities evaluated was reported as PAH species that can be modeled individually. Consequently, the following modeling approach was used. POM species and groups reported in NEI, including emissions reported as mixtures of POM, were assigned to POM categories according to estimated cancer potency (*i.e.*, using the same methods employed to evaluate inhalation risk from POM). Multipathway fate and transport modeling of POM emissions was conducted using benzo[a]pyrene as a surrogate chemical for the behavior of other POM in the environment. Total emissions of POM (including unspeciated PAHs) were evaluated using a risk screening method based on a hypothetical ingestion exposure modeling scenario. Using this approach, we were able to confirm for 133 of the 156 facilities that risks via ingestion exposures were well below levels of concern. For the remaining 23 facilities, the results of the screen were less definitive, and incremental lifetime cancer risks modeled using the hypothetical scenario were estimated to be as high as 67 in a million at one facility. However, because we used a conservative speciation profile to estimate the risk contribution of individual POM compounds to the total POM risks, we believe that this screening result is highly conservative, and that actual PAH risks due to ingestion are much lower. As a result, we did not further refine our assessment of multipathway human health risks.

No ecological benchmarks were exceeded in our multipathway screening. Contaminant concentrations were evaluated against ecological benchmarks for sediment, soil, and water which were taken from the TRIM Ecological Toxicity Database [*33*]. For PAH, the lowest, and thus most conservative, ecological benchmark for soil (developed by the Canadian Council of Ministries of the Environment) [*34*] was approximately the same as the modeled soil concentration. This indicates little to no potential for adverse growth, reproductive effects, and mortality in the soil community, terrestrial plants, and earthworms. This result is associated with using a default speciation profile for assessing unspeciated PAH, as described in the previous paragraph, and is thought to be highly conservative. No further refinement of multipathway ecological risks was undertaken.

We also screened for potential adverse environmental impacts via direct atmospheric contact by comparing chronic atmospheric concentrations to RfC values at locations outside estimated facility boundaries, noting, as we have in previous residual risk assessments, that chronic human health inhalation thresholds are generally more stringent than direct contact environmental protection thresholds developed to date. None of the HAP emitted by petroleum refinery MACT 1 sources showed any potential for adverse environmental impacts based on this screening. We are aware that some concerns have been expressed regarding the adequacy of this screening

method for the pollutant hydrogen fluoride, since studies have been identified in the scientific literature showing adverse effects on some flora at levels below human health thresholds. Indeed, there is a significant lack of scientific understanding and assessment methodologies for such potential adverse environmental effects. Notwithstanding these concerns, we believe that the negative outcome of our assessment based on the chronic noncancer human health endpoint for hydrogen fluoride (the maximum HQ for this pollutant was 0.25) provides strong support for our conclusion that adverse environmental impacts are not expected for hydrogen fluoride emissions from this source category.

2.4 General Discussion of Uncertainties

Uncertainty and the potential for bias are inherent in all risk assessments, including the one performed for the petroleum refineries source category presented in this document. The primary uncertainties in this risk characterization focus around the site-specific emissions data set (as discussed in the previous sections and in [*30, 31*]) and the uncertainties in dose-response quantification. While other aspects of the assessment, including dispersion modeling, inhalation exposure estimates, and multi-pathway exposure modeling all bring some degree of uncertainty to the assessment, these uncertainties are secondary if emissions and site-specific characteristics are not represented correctly.

2.4.1 Exposure Modeling Uncertainties

Although the development of the RTR database involved quality assurance/quality control processes, the accuracy of emissions values will vary depending on the source of the data present, incomplete or missing data, errors in estimating emissions values, and other factors. Our review of the data indicates that there may be a low bias in reported emissions for many facilities, but the extent of potential underreporting is not known. It appears that data from several processes and operations are not included in the reported emissions from many facilities. These include exclusion of upset, malfunction, startup, and shutdown events as well as omission of emissions sources that are unexpected, not measured, or not considered in inventories, such as leaks in heat exchanger systems; emissions from process sewers and wastewater systems; fugitive emissions from delayed coking units; and emissions from tank roof landings. Further, the emissions values considered in this analysis are annual totals for a single calendar year (2002) and do not reflect actual fluctuations during the course of the year or variations from year to year, including plant closure or expansion. Finally, although we have performed a significant amount of quality control on the data set, for many facilities the physical characteristics (*i.e.*, stack height, physical location) of the reported sources may be inaccurate for detailed risk characterization purposes. The following general discussion of uncertainties applies to the remaining aspects of the risk assessment, which are thought to contribute less to overall uncertainties in the risk results, but are nonetheless included for completeness.

The chronic exposure modeling uncertainties are considered relatively small since we are using EPA's refined local dispersion model with site-specific parameters and reasonably representative meteorology. If anything, the population exposure estimates are biased high by not accounting for short- or long-term population mobility, and by neglecting processes like deposition, plume depletion, and atmospheric degradation. Additionally, estimates of the maximum individual risk (MIR) contain uncertainty, because they are derived at census block centroid locations rather than actual residences. This uncertainty is known to create potential underestimates and

overestimates of the actual MIR values for individual facilities, but, overall, it is not thought to have a significant impact on the estimated MIR for a source category. Finally, we did not factor in the possibility of a source closure occurring during the 70-year chronic exposure period, leading to a potential upward bias in both the MIR and population risk estimates; nor did we factor in the possibility of population growth or production expansion during the 70-year chronic exposure period, leading to a potential downward bias in both the MIR and population risk estimates.

As previously discussed in section 2.2.2, a sensitivity analysis performed for the 1999 NATA found that the selection of the meteorology dataset location could result in a range of chronic ambient concentrations which varied from as much as 17% below the predicted value to as much as 84% higher than the predicted value. This variability translates directly to the predicted exposures and risks in our assessment, indicating that the actual risks could vary from 17% lower to 84% higher than the predicted values.

We have purposely biased the acute screening results high, considering that they depend upon the joint occurrence of independent factors, such as hourly emissions rates, meteorology and human activity patterns. Furthermore, in cases where multiple acute threshold values are considered scientifically acceptable we have chosen the most conservative of these assessments, erring on the side of overestimating potential health risks from acute exposures. In the cases where these results indicated the potential for exceeding short-term health thresholds, we have refined our assessment by developing a better understanding of the geography of the facility relative to potential exposure locations. In each of these cases, we have determined that this refined information reduced the likelihood of acute health concerns. We were not able to refine these assessments to incorporate the true variability of short-term emission rates; such data are not currently available. Thus, by maintaining the peak-to-mean emission ratio of 10 even in our refined acute assessments, we believe the results generally overstate the potential for acute impacts. We base this conclusion on the fact that our analysis of short-term event emission data (Appendix B) indicates that the factor of 10 covers more than 99% of all actual peak emission events for volatile and gaseous HAPs.

2.4.2 Uncertainties in the Dose-Response Relationships

In the sections that follow, separate discussions are provided on uncertainty associated with cancer potency factors and for noncancer reference values. Cancer potency values are derived for chronic (lifetime) exposures. Noncancer reference values are generally derived for chronic exposures (up to a lifetime), but may also be derived for acute (<24 hours), short-term (>24 hours up to 30 days), and subchronic (>30 days up to 10% of lifetime) exposure durations, all of which are derived based on an assumption of continuous exposure throughout the duration specified. For the purposes of assessing all potential health risks associated with the emissions included in this assessment, we rely on both chronic (cancer and noncancer) and acute (noncancer) benchmarks, which are described in more detail below.

Although every effort is made to identify peer-reviewed dose-response values for all 75 HAPs emitted by the sources included in this assessment, some HAP have no peer-reviewed cancer potency values or reference values for chronic non-cancer or acute effects. Since exposures to

these pollutants cannot be included in a quantitative risk estimate, an understatement of risk for these pollutants at environmental exposure levels is possible.

Additionally, chronic dose-response values for 26 of the compounds included in this assessment are currently under EPA IRIS review and revised assessments may determine that these pollutants are more or less potent than currently thought. We will re-evaluate residual risks if, as a result of these reviews, a dose-response metric changes enough to indicate that the risk assessment supporting today's notice may significantly mischaracterize human health risk.

Cancer assessment

The discussion of dose-response uncertainties in the estimation of cancer risk below focuses on the uncertainties associated with the specific approach currently used by the EPA to develop cancer potency factors. In general, these same uncertainties attend the development of cancer potency factors by CalEPA, the source of peer-reviewed cancer potency factors used where EPA-developed values are not yet available. To place this discussion in context, we provide a quote from the EPA's *Guidelines for Carcinogen Risk Assessment*.[*35*] "The primary goal of EPA actions is protection of human health; accordingly, as an Agency policy, risk assessment procedures, including default options that are used in the absence of scientific data to the contrary, should be health protective." The approach adopted in this document is consistent with this approach as described in the *Cancer Guidelines*.

For cancer endpoints EPA usually derives an oral slope factor for ingestion and a unit risk value for inhalation exposures. These values allow estimation of a lifetime probability of developing cancer given long-term exposures to the pollutant. Depending on the pollutant being evaluated, EPA relies on both animal bioassay and epidemiological studies to characterize cancer risk. As a science policy approach, consistent with the *Cancer Guidelines*, EPA uses animal cancer bioassays as indicators of potential human health risk when other human cancer risk data are unavailable.

Extrapolation of study data to estimate potential risks to human populations is based upon EPA's assessment of the scientific database for a pollutant using EPA's guidance documents and other peer-reviewed methodologies. The EPA *Guidelines for Carcinogen Risk Assessment* describes the Agency's recommendations for methodologies for cancer risk assessment. EPA believes that cancer risk estimates developed following the procedures described in the *Cancer Guidelines* and outlined below generally provide an upper bound estimate of risk. That is, EPA's upper bound estimates represent a "plausible upper limit to the true value of a quantity" (although this is usually not a true statistical confidence limit).[25] In some circumstances, the true risk could be as low as zero; however, in other circumstances the risk could also be greater.[26] When developing an upper bound estimate of risk and to provide risk values that do not underestimate risk, EPA generally relies on conservative default approaches.[27] EPA also uses the upper bound (rather

[25] IRIS glossary (www.epa.gov/NCEA/iris/help_gloss.htm).

[26] The exception to this is the URE for benzene, which is considered to cover a range of values, each end of which is considered to be equally plausible, and which is based on maximum likelihood estimates.

[27] According to the NRC report *Science and Judgment in Risk Assessment* (NRC, 1994) "[Default] options are generic approaches, based on general scientific knowledge and policy judgment, that are applied to various elements

than lower bound or central) estimates in its assessments, although it is noted that this approach can have limitations for some uses (e.g. priority setting, expected benefits analysis).

Such health risk assessments have associated uncertainties, some of which may be considered quantitatively, and others which generally are expressed qualitatively. Uncertainties may vary substantially among cancer risk assessments associated with exposures to different pollutants, since the assessments employ different databases with different strengths and limitations and the procedures employed may differ in how well they represent actual biological processes for the assessed substance. EPA's *Risk Characterization Handbook* also recommends that risk characterizations present estimates demonstrating the impact on the assessment of alternative choices, data, models and assumptions (U.S. EPA, 2000). Some of the major sources of uncertainty and variability in deriving cancer risk values are described more fully below.

(1) The qualitative similarities or differences between tumor responses observed in experimental animal bioassays and those which would occur in humans are a source of uncertainty in cancer risk assessment. In general, EPA does not assume that tumor sites observed in an experimental animal bioassay are necessarily predictive of the sites at which tumors would occur in humans.[28] However, unless scientific support is available to show otherwise, EPA assumes that tumors in animals are relevant in humans, regardless of target organ concordance. For a specific pollutant, qualitative differences in species responses can lead to either under-estimation or over-estimation of human cancer risks.

(2) Uncertainties regarding the most appropriate dose metric for an assessment can also lead to differences in risk predictions. For example, the measure of dose is commonly expressed in units of mg/kg/d ingested or the inhaled concentration of the pollutant. However, data may support development of a pharmacokinetic model for the absorption, distribution, metabolism and excretion of an agent, which may result in improved dose metrics (*e.g.*, average blood concentration of the pollutant or the quantity of agent metabolized in the body). Quantitative uncertainties result when the appropriate choice of a dose metric is uncertain or when dose metric estimates are themselves uncertain (*e.g.*, as can occur when alternative pharmacokinetic models are available for a compound). Uncertainty in dose estimates may lead to either over or underestimation of risk.

(3) For the quantitative extrapolation of cancer risk estimates from experimental animals to humans, EPA uses scaling methodologies (relating expected response to differences in

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of the risk-assessment process when the correct scientific model is unknown or uncertain." The 1983 NRC report *Risk Assessment in the Federal Government: Managing the Process* defined *default option* as "the option chosen on the basis of risk assessment policy that appears to be the best choice in the absence of data to the contrary" (NRC, 1983a, p. 63). Therefore, default options are not rules that bind the agency; rather, the agency may depart from them in evaluating the risks posed by a specific substance when it believes this to be appropriate. In keeping with EPA's goal of protecting public health and the environment, default assumptions are used to ensure that risk to chemicals is not underestimated (although defaults are not intended to overtly overestimate risk). See EPA 2004 *An Examination of EPA Risk Assessment Principles and Practices*, EPA/100/B-04/001 available at: http://www.epa.gov/osa/pdfs/ratf-final.pdf.

[28] Per the EPA Cancer Guidelines: "The default option is that positive effects in animal cancer studies indicate that the agent under study can have carcinogenic potential in humans." and "Target organ concordance is not a prerequisite for evaluating the implications of animal study results for humans."

physical size of the species), which introduce another source of uncertainty. These methodologies are based on both biological data on differences in rates of process according to species size and empirical comparisons of toxicity between experimental animals and humans. For a particular pollutant, the quantitative difference in cancer potency between experimental animals and humans may be either greater than or less than that estimated by baseline scientific scaling predictions due to uncertainties associated with limitations in the test data and the correctness of scaled estimates.

(4) EPA cancer risk estimates, whether based on epidemiological or experimental animal data, are generally developed using a benchmark dose (BMD) analysis to estimate a dose at which there is a specified excess risk of cancer (called a "point of departure," or POD). Statistical uncertainty in developing a POD using a benchmark dose (BMD) approach is generally addressed through use of the 95% lower confidence limit on the dose at which the specified excess risk occurs (the BMDL), decreasing the likelihood of understating risk. EPA has generally utilized the multistage model for estimation of the BMDL using cancer bioassay data (see further discussion below).

(5) Extrapolation from high to low doses is an important, and potentially large, source of uncertainty in cancer risk assessment. EPA uses different approaches to low dose risk assessment (*i.e.*, developing estimates of risk for exposures to environmental doses of an agent from observations in experimental or epidemiological studies at higher dose) depending on the available data and understanding of a pollutant's mode of action (*i.e.*, the manner in which a pollutant causes cancer). EPA's cancer guidelines express a preference for the use of reliable, compound-specific, biologically-based risk models when feasible; however, such models are rarely available. The mode of action for a pollutant (*i.e.*, the manner in which a pollutant causes cancer) is a key consideration in determining how risks should be estimated for low-dose exposure. A reference value is calculated when the available mode of action data show the response to be nonlinear (*e.g.*, as in a threshold response). A linear low-dose (straight line from POD) approach is used when available mode of action data support a linear (*e.g.*, nonthreshold response) or as the most common default approach when a compound's mode of action is unknown. Linear extrapolation can be supported by both pollutant-specific data and broader scientific considerations. For example, EPA's *Cancer Guidelines* generally consider a linear dose-response to be appropriate for pollutants that interact with DNA and induce mutations. Pollutants whose effects are additive to background biological processes in cancer development can also be predicted to have low-dose linear responses, although the slope of this relationship may not be the same as the slope estimated by the straight line approach.

EPA most frequently utilizes a linear low-dose extrapolation approach as a baseline science-policy choice (a "default") when available data do not allow a compound-specific determination. This approach is designed to not underestimate risk in the face of uncertainty and variability. EPA believes that linear dose-response models, when appropriately applied as part of EPA's cancer risk assessment process, provide an upper bound estimate of risk and generally provide a health protective approach. Note that another source of uncertainty is the characterization of low-dose nonlinear, non-threshold relationships. The National Academy of Sciences has encouraged the exploration of sigmoidal type functions (*e.g.*, log-probit models) in representing dose response relationships due to the variability in response within human populations. A

recent National Research Council report (NRC, 2006) [*36*] suggests that models based on distributions of individual thresholds are likely to lead to sigmoidal-shaped dose-response functions for a population. This report notes sources of variability in the human population: "One might expect these individual tolerances to vary extensively in humans depending on genetics, coincident exposures, nutritional status, and various other susceptibility factors..." Thus, if a distribution of thresholds approach is considered for a carcinogen risk assessment, application would depend on ability of modeling to reflect the degree of variability in response in human populations (as opposed to responses in bioassays with genetically more uniform rodents). Note also that low dose linearity in risk can arise for reasons separate from population variability: due to the nature of a mode of action and additivity of a chemical's effect on top of background chemical exposures and biological processes.

As noted above, EPA's current approach to cancer risk assessment typically utilizes a straight line approach from the BMDL. This is equivalent to using an upper confidence limit on the slope of the straight line extrapolation. The impact of the choice of the BMDL on bottom line risk estimates can be quantified by comparing risk estimates using the BMDL value to central estimate BMD values, although these differences are generally not a large contributor to uncertainty in risk assessment [*37*]. It is important to note that earlier EPA assessments, including the majority of those for which risk values exist today, were generally developed using the multistage model to extrapolate down to environmental dose levels and did not involve the use of a POD. Comparisons indicating that slopes based on straight line extrapolation from a POD do not show large differences from those based on the upper confidence limit of the multistage model [*37*].

(6) Cancer risk estimates do not generally make specific adjustments to reflect the variability in response within the human population — resulting in another source of uncertainty in assessments. In the diverse human population, some individuals are likely to be more sensitive to the action of a carcinogen than the typical individual, although compound-specific data to evaluate this variability are generally not available. There may also be important life stage differences in the quantitative potency of carcinogens and, with the exception of the recommendations in EPA's *Supplemental Cancer Guidance* for carcinogens with a mutagenic mode of action, risk assessments do not generally quantitatively address life stage differences. However, one approach used commonly in EPA assessments that may help address variability in response is to extrapolate human response from results observed in the most sensitive species and sex tested, resulting typically in the highest URE which can be supported by reliable data, thus supporting estimates that are designed not to underestimate risk in the face of uncertainty and variability.

Chronic noncancer assessment

Chronic noncancer reference values represent chronic exposure levels that are intended to be health-protective. That is, EPA and other organizations which develop noncancer reference values (*e.g.*, the Agency for Toxic Substances and Disease Registry – ATSDR) utilize an approach that is intended not to underestimate risk in the face of uncertainty and variability. When there are gaps in the available information, uncertainty factors (UFs) are applied to derive reference values that are intended to be protective against appreciable risk of deleterious effects.

Uncertainty factors are commonly default values[29], *e.g.*, factors of 10 or 3 used in the absence of compound-specific data. Where data are available, uncertainty factors may also be developed using compound-specific information. When data are limited, more assumptions are needed and more default factors are used. Thus there may be a greater tendency to overestimate risk—in the sense that further study might support development of reference values that are higher (*i.e.*, less potent) because fewer default assumptions are needed. However, for some pollutants it is possible that risks may be underestimated.

For non-cancer endpoints related to chronic exposures, EPA derives a Reference Dose (RfD) for exposures via ingestion, and a Reference Concentration (RfC) for inhalation exposures. These values provide an estimate (with uncertainty spanning perhaps an order of magnitude) of daily oral exposure (RfD) or of a continuous inhalation exposure (RfC) to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.[30] To derive values that are intended to be "without appreciable risk," EPA's methodology relies upon an uncertainty factor (UF) approach (U.S. EPA, 1993, 1994) which includes consideration of both uncertainty and variability.

EPA begins by evaluating all of the available peer-reviewed literature to determine non-cancer endpoints of concern, evaluating the quality, strengths and limitations of the available studies. EPA typically chooses the relevant endpoint that occurs at the lowest dose, often using statistical modeling of the available data, and then determines the appropriate POD for derivation of the reference value. A POD is determined by (in order of preference): (1) a statistical estimation using the benchmark dose (BMD) approach; (2) use of the dose or concentration at which the toxic response was not significantly elevated (no observed adverse effect level— NOAEL); or (3) use of the lowest observed adverse effect level (LOAEL).

A series of downward adjustments using default UFs is then applied to the POD to estimate the reference value (U.S. EPA 1994, 2002). While collectively termed "UFs", these factors account for a number of different quantitative considerations when utilizing observed animal (usually rodent) or human toxicity data in a risk assessment. The UFs are intended to account for: (1) variation in susceptibility among the members of the human population (*i.e.*, inter-individual

[29] According to the NRC report *Science and Judgment in Risk Assessment* (NRC, 1994) "[Default] options are generic approaches, based on general scientific knowledge and policy judgment, that are applied to various elements of the risk-assessment process when the correct scientific model is unknown or uncertain." The 1983 NRC report *Risk Assessment in the Federal Government: Managing the Process* defined *default option* as "the option chosen on the basis of risk assessment policy that appears to be the best choice in the absence of data to the contrary" (NRC, 1983a, p. 63). Therefore, default options are not rules that bind the agency; rather, the agency may depart from them in evaluating the risks posed by a specific substance when it believes this to be appropriate. In keeping with EPA's goal of protecting public health and the environment, default assumptions are used to ensure that risk to chemicals is not underestimated (although defaults are not intended to overtly overestimate risk). See EPA 2004 *An examination of EPA Risk Assessment Principles and Practices,* EPA/100/B-04/001 available at: http://www.epa.gov/osa/pdfs/ratf-final.pdf

[30] See IRIS glossary

variability); (2) uncertainty in extrapolating from experimental animal data to humans (*i.e.*, interspecies differences); (3) uncertainty in extrapolating from data obtained in a study with less-than-lifetime exposure (*i.e.*, extrapolating from subchronic to chronic exposure); (4) uncertainty in extrapolating from a LOAEL in the absence of a NOAEL; and (5) uncertainty when the database is incomplete or there are problems with applicability of available studies. When scientifically sound, peer-reviewed assessment-specific data are not available, default adjustment values are selected for the individual UFs. For each type of uncertainty (when relevant to the assessment), EPA typically applies an UF value of 10 or 3 with the cumulative UF value leading to a downward adjustment of 10-3000 fold from the selected POD. An UF of 3 is used when the data do not support the use of a 10-fold factor. If an extrapolation step or adjustment is not relevant to an assessment (*e.g.*, if applying human toxicity data and an interspecies extrapolation is not required) the associated UF is not used. The major adjustment steps are described more fully below.

1) Heterogeneity among humans is a key source of variability as well as uncertainty. Uncertainty related to human variation is considered in extrapolating doses from a subset or smaller-sized population, often of one sex or of a narrow range of life stages (typical of occupational epidemiologic studies), to a larger, more diverse population. In the absence of pollutant-specific data on human variation, a 10-fold UF is used to account for uncertainty associated with human variation. Human variation may be larger or smaller; however, data to examine the potential magnitude of human variability are often unavailable. In some situations, a smaller UF of 3 may be applied to reflect a known lack of significant variability among humans.

2) Extrapolation from results of studies in experimental animals to humans is a necessary step for the majority of chemical risk assessments. When interpreting animal data, the concentration at the POD (e.g. NOAEL, BMDL) in an animal model (e.g. rodents) is extrapolated to estimate the human response. While there is long-standing scientific support for the use of animal studies as indicators of potential toxicity to humans, there are uncertainties in such extrapolations. In the absence of data to the contrary, the typical approach is to use the most relevant endpoint from the most sensitive species and the most sensitive sex in assessing risks to the average human. Typically, compound specific data to evaluate relative sensitivity in humans versus rodents are lacking, thus leading to uncertainty in this extrapolation. Size-related differences (allometric relationships) indicate that typically humans are more sensitive than rodents when compared on a mg/kg/day basis. The default choice of 10 for the interspecies UF is consistent with these differences. For a specific chemical, differences in species responses may be greater or less than this value.

Pharmacokinetic models are useful to examine species differences in pharmacokinetic processing and associated uncertainties; however, such dosimetric adjustments are not always possible. Information may not be available to quantitatively assess toxicokinetic or toxicodynamic differences between animals and humans, and in many cases a 10-fold UF (with separate factors of 3 for toxicokinetic and toxicodynamic components) is used to account for expected species differences and associated uncertainty in extrapolating from laboratory animals to humans in the derivation of a reference value. If information on one or the other of these

components is available and accounted for in the cross-species extrapolation, a UF of 3 may be used for the remaining component.

3) In the case of reference values for chronic exposures where only data from shorter durations are available (*e.g.*, 90-day subchronic studies in rodents) or when such data are judged more appropriate for development of an RfC, an additional UF of 3 or 10-fold is typically applied unless the available scientific information supports use of a different value.

4) Toxicity data are typically limited as to the dose or exposure levels that have been tested in individual studies; in an animal study, for example, treatment groups may differ in exposure by up to an order of magnitude. The preferred approach to arrive at a POD is to use BMD analysis; however, this approach requires adequate quantitative results for a meaningful analysis, which is not always possible. Use of a NOAEL is the next preferred approach after BMD analysis in determining a POD for deriving a health effect reference value. However, many studies lack a dose or exposure level at which an adverse effect is not observed (*i.e.*, a NOAEL is not identified). When using data limited to a LOAEL, a UF of 10 or 3-fold is often applied.

5) The database UF is intended to account for the potential for deriving an underprotective RfD/RfC due to a data gap preventing complete characterization of the chemical's toxicity. In the absence of studies for a known or suspected endpoint of concern, a UF of 10 or 3-fold is typically applied.

Acute noncancer assessment

Many of the UFs used to account for variability and uncertainty in the development of acute reference values are quite similar to those developed for chronic durations, but more often using individual UF values that may be less than 10. UFs are applied based on chemical-specific or health effect-specific information (*e.g.*, simple irritation effects do not vary appreciably between human individuals, hence a value of 3 is typically used), or based on the purpose for the reference value (see the following paragraph). The UFs applied in acute reference value derivation include: 1) heterogeneity among humans; 2) uncertainty in extrapolating from animals to humans; 3) uncertainty in LOAEL to NOAEL adjustments; and 4) uncertainty in accounting for an incomplete database on toxic effects of potential concern. Additional adjustments are often applied to account for uncertainty in extrapolation from observations at one exposure duration (*e.g.*, 4 hours) to arrive at a POD for derivation of an acute reference value at another exposure duration (*e.g.*, 1 hour).

Not all acute reference values are developed for the same purpose and care must be taken when interpreting the results of an acute assessment of human health effects relative to the reference value or values being exceeded. Where relevant to the estimated exposures, the lack of threshold values at different levels of severity should be factored into the risk characterization as potential uncertainties.

3 Portland cement case study

3.1 Introduction

This section provides documentation of our case study for the Portland Cement Manufacturing source category. Section 3.2 provides a description of the Portland Cement Manufacturing source category and a brief summary of the emissions data in the case study data set. Section 3.3 provides results of the inhalation risk assessment, including an assessment of the potential inhalation risks associated with radionuclide emissions from Portland cement facilities. Section 3.4 provides the details of the multipathway exposure and risk assessment results for Portland cement facilities, including a description of the methodologies used to refine this portion of the assessment. Section 3.5 presents the methodologies used to assess potential ecological risks associated with emissions from Portland cement facilities, and then provides a summary of results and interpretation.

3.2 Source category and emissions data

The Portland Cement Manufacturing source category includes facilities that produce Portland cement. Portland cement is a fine powder, usually gray in color, that consists of a mixture of the minerals dicalcium silicate, tricalcium silicate, tricalcium aluminate, and tetracalcium aluminoferrite, to which one or more forms of calcium sulfate have been added. The primary end use of Portland cement is as the key ingredient in Portland cement concrete, which is used in almost all construction applications.

The process of manufacturing Portland cement consists of four primary units of operation: (1) kiln feed preparation (*i.e.*, crushing and grinding the carefully proportioned raw materials to a high degree of fineness); (2) firing the raw mix in a rotary kiln to produce clinker (an intermediate product, before grinding), including fuel handling; (3) grinding the resulting clinker to a fine powder and mixing with gypsum to produce cement; and (4) raw and finished materials handling. As a whole, the manufacturing process is expected to result in the emission of the following HAP: acetaldehyde, arsenic, benzene, cadmium, chromium, chlorobenzene, dioxins, formaldehyde, hexane, hydrogen chloride, lead, manganese, mercury, naphthalene, nickel, phenol, polycyclic organic matter, selenium, styrene, toluene, and xylene. These HAP are associated with the emissions of specific production processes, including grinding and conveying operation dusts, exhaust gases from the raw material dryer; kiln exhaust gases; clinker cooler exhaust gases; and dusts from the finish grinding of clinker into cement. Emissions from the grinding and conveying operations are essentially particulate emissions (*e.g.*, dust from limestone, clay, and bauxite ore) that contain HAP metals. Raw material dryers are used as part of the feed preparation process (*i.e.*, drying, blending, and storage), and can produce emissions in two different ways. If the raw material dryer uses heat from a separate combustion source (fuel-fired raw material dryer), exhaust gases can contain trace quantities of products of incomplete combustion (PICs), HCl, and metals from the fuel. When feed materials contain organic matter, this material may volatilize in the raw material dryer (regardless of the source of the heat) adding organic HAPs to the dryer exhaust. Kiln exhaust emissions contain a wide variety of HAPs and other air pollutants that originate from the fuel combustion and from the feed material. These HAPs include gaseous organic HAPs, some of which are chlorinated, along with mercury (emitted as either a particulate or a gas), hydrogen chloride, dioxins, and the following metal

HAP emissions: chromium, lead, arsenic, mercury, antimony, and manganese. Because clinker coolers are not combustion devices, the only expected HAPs are metals associated with the clinker cooler particulate, *i.e.*, clinker dust. HAP metals that have been detected in clinker include chromium, lead, nickel, arsenic, beryllium, antimony, selenium, and mercury. The finish grinding of clinker into Portland cement produces dusts that can contain HAP metals associated with clinker, which are listed above.

From information gathered during the MACT development and from more recent contacts with the industry, EPA estimates that there are 104 facilities with processes belonging in the Portland cement manufacturing source category. EPA identified each of these facilities in Version 1.0 of the 2002 NEI (February 2006) and created a data set comprised of the HAP emissions and emissions release parameters for Portland cement production portions of these facilities. EPA reviewed the data set and identified processes, facilities, and chemicals that, based on SCC and other process identifications in the NEI, were erroneously included in the Portland cement source category, and made revisions to exclude these processes, facilities, or chemicals from the data set for this source category. There are several factors that make the emissions dataset for the Portland cement case study more provisional than that for the petroleum refineries case study. First, the data are still under development, and have already been revised since this case study was developed. Second, the data have not yet undergone public review, but will do so prior to any regulatory action. And third, the technology-based standards that the dataset reflects may yet be amended, with consequent reductions in emissions and risk. It is important to keep in mind that this case study will change substantially during the RTR rule development process, and that it is presented here only to illustrate a methodology.

Table 3-1 summarizes the emissions for the Portland Cement Manufacturing source category data set. Based on these data, the HAP emitted in the largest quantity is hydrochloric acid, which accounts for approximately 73 percent of the HAP mass emitted. Hydrochloric acid, along with benzene, formaldehyde, toluene, chlorine, 1,3-butadiene, naphthalene, xylenes (mixture of o, m, and p isomers), carbonyl sulfide, manganese, styrene, ethyl benzene, phenol, lead, manganese, ethylene glycol, chromium, methylene chloride, carbon disulfide, acetaldehyde, chromium, methyl chloride, lead & compounds, and hexane account for approximately 99 percent of the HAP mass emitted across the 104 facilities. Hydrochloric acid is the HAP reported most frequently across the source category, with reported emissions from 79 of the 104 facilities in the data set. The dataset includes emissions of substances representing 10 of the 14 PB-HAP[31] categories (mercury, lead, cadmium, POM, PCBs, hexachlorobenzene, trifluralin, methoxychlor, heptachlor, and chlordane).

[31] Persistent and bioaccumulative HAP are defined in the EPA's *Air Toxics Risk Assessment Library [6]*.

Table 3-1. Summary of Emissions from the Portland Cement Manufacturing Source Category

HAP[32]	Emissions (tpy)	Number of Facilities Reporting HAP (104 facilities in ANPRM data set)	Prioritized Inhalation Dose-Response Value Identified by OAQPS[33]			PB-HAP?
			Unit Risk Estimate for Cancer?	Reference Concentration for Chronic Noncancer?	Health Benchmark Values for Acute Noncancer?	
Hydrochloric Acid	3,162	79		✔	✔	
Benzene	330	56	✔	✔	✔	
Formaldehyde	155	49	✔	✔	✔	
Toluene	81	38		✔	✔	
Chlorine	77	14		✔	✔	
1,3-Butadiene	64	7	✔	✔	✔	
Naphthalene	55	46	✔	✔		
Xylenes (Mixture of o, m, and p Isomers)	50	35		✔	✔	
Carbonyl Sulfide	48	1				
Manganese	45	42		✔		
Styrene	30	28		✔	✔	
Ethyl Benzene	22	33		✔		
Phenol	20	32		✔	✔	
Lead	16	65		✔		✔
Manganese & Compounds	14	13		✔		
Ethylene Glycol	13	9		✔		
Chromium	11	36	✔	✔		
Methylene Chloride	11	30	✔	✔	✔	
Carbon Disulfide	10	23		✔	✔	

[32] Notes for how HAP were speciated for risk assessment:

- For most metals, emissions reported as the elemental metal are combined with metal compound emissions (*e.g.*, "cadmium" emissions modeled as "cadmium & compounds").
- For emissions reported generically as "chromium" or "chromium & compounds," emissions are speciated for this category as 92 percent "chromium (III) compounds" and 8 percent "chromium (VI) compounds." Chromium speciation profiles can be found on the EPA's Technology Transfer Network website for emissions inventories at http://www.epa.gov/ttn/chief/net/2002inventory.html.
- For emissions reported generically as "mercury" or "mercury & compounds," emissions are speciated for this category as 75 percent "mercury (elemental)" and 25 percent "mercuric chloride." Mercury speciation profiles can be found on the EPA's Technology Transfer Network website for emissions inventories at http://www.epa.gov/ttn/chief/net/2002inventory.
- For emissions of any chemicals or chemical groups classified as polycyclic organic matter (POM), emissions will be grouped into POM subgroups as found on EPA's Technology Transfer Network website for the 1999 National-Scale Air Toxics Assessment at http://www.epa.gov/ttn/atw/nata1999/nsata99.html.
- For emissions reported generically as "Glycol Ethers" or specific glycol ethers not found on EPA's Technology Transfer network for air toxics (see footnote b), emissions will be treated as ethylene glycol methyl ether.

[33] Specific dose-response values for each chemical are identified on EPA's Technology Transfer Network website for air toxics at http://www.epa.gov/ttn/atw/toxsource/summary.html.

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			Unit Risk Estimate for Cancer?	Reference Concentration for Chronic Noncancer?	Health Benchmark Values for Acute Noncancer?	
Acetaldehyde	9	10	✔	✔	✔	
Chromium & Compounds	9	27	✔	✔		
Methyl Chloride	7	20		✔	✔	
Lead & Compounds	6	28		✔		✔
Hexane	5	12		✔		
Mercury	5	53		✔	✔	✔
1,3-Propanesultone	4	1	✔			
Methanol	4	9		✔	✔	
Phenanthrene	4	19	✔			✔
Acrolein	3	3		✔	✔	
Dibenzofuran	3	8				✔
1-Chloro-2,3-Epoxypropane	2	1	✔	✔	✔	
Acetophenone	2	4				
Acrylonitrile	2	3	✔	✔	✔	
Bromoform	2	4	✔			
Hydrogen Fluoride	2	5		✔	✔	
Mercury & Compounds	2	35		✔		✔
Methyl Bromide	2	18		✔	✔	
Nickel & Compounds	2	12	✔	✔		
1,4-Dichlorobenzene	1	6	✔	✔		
Acenaphthylene	1	14	✔			✔
Beryllium	1	25	✔	✔		
Beryllium & Compounds	1	13	✔	✔	✔	
Biphenyl	1	20				
Chlorobenzene	1	22		✔		
Dibutyl Phthalate	1	19				
Diethanolamine	1	1		✔		
Fluorene	1	16	✔			✔
Methyl Chloroform	1	8		✔	✔	
Methyl Isobutyl Ketone	1	11		✔		
Nickel	1	30	✔	✔		
Polycyclic Organic Matter	1	2	✔			✔
Selenium	1	26		✔		
Selenium & Compounds	1	11		✔		
Tetrachloroethylene	1	11	✔	✔	✔	
Vinyl Chloride	1	7	✔	✔	✔	
Bis(2-Ethylhexyl)Phthalate	0.5	17	✔	✔		
Cadmium	0.4	27	✔	✔		✔
Cumene	0.4	6		✔		

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			Unit Risk Estimate for Cancer?	Reference Concentration for Chronic Noncancer?	Health Benchmark Values for Acute Noncancer?	
Glycol Ethers	0.4	4		✔		
N,N-Dimethyl formamide	0.4	5		✔	✔	
Acetonitrile	0.3	7		✔	✔	
Arsenic	0.3	25	✔	✔		
Methyl Tert-Butyl Ether	0.3	6	✔	✔		
m-Xylene	0.3	2				
Phosphorus	0.3	5		✔		
Vinyl Acetate	0.3	4		✔	✔	
Lead Compounds (Inorganic)	0.2	6		✔		✔
PAH, total	0.2	11	✔			✔
Allyl Chloride	0.1	2	✔	✔	✔	
Arsenic & Compounds (Inorganic Including Arsine)	0.1	12	✔	✔	✔	
Benzyl Chloride	0.1	2	✔		✔	
Cadmium & Compounds	0.1	14	✔	✔		✔
Carbon Tetrachloride	0.1	4	✔	✔	✔	
Chromium (VI)	0.1	10	✔	✔		
Cobalt	0.1	6		✔		
Dichloroethyl Ether	0.1	1	✔			
Ethylene Dibromide	0.1	4	✔	✔		
Ethylene Dichloride	0.1	9	✔	✔	✔	
Ethylene Glycol Methyl Ether	0.1	1		✔		
Fluoranthene	0.1	14	✔			✔
Methyl Iodide	0.1	1			✔	
o-Xylene	0.1	5				
Trichloroethylene	0.1	7	✔	✔	✔	
Cresol	0.05	3		✔		
Ethyl Chloride	0.04	3		✔		
Hexachlorobutadiene	0.04	3	✔	✔	✔	
Pyrene	0.04	15	✔			✔
1,3-Dichloropropene	0.03	3	✔	✔		
2,4-Dinitrophenol	0.03	2				
Antimony	0.03	5		✔		
Asbestos	0.03	1	✔			
Chloroform	0.03	7		✔	✔	
Vinylidene Chloride	0.03	3		✔		
1,1,2-Trichloroethane	0.02	2	✔	✔		
1,2-Epoxybutane	0.02	1		✔		

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HAP[32]	Emissions (tpy)	Number of Facilities Reporting HAP (104 facilities in ANPRM data set)	Prioritized Inhalation Dose-Response Value Identified by OAQPS[33]			PB-HAP?
			Unit Risk Estimate for Cancer?	Reference Concentration for Chronic Noncancer?	Health Benchmark Values for Acute Noncancer?	
2,4-Dinitrotoluene	0.02	2	✔	✔		
Antimony & Compounds	0.02	2		✔		
Cellosolve Solvent	0.02	1		✔		
Chromium III	0.02	7				
Methyl Methacrylate	0.02	6		✔	✔	
p-Cresol	0.02	3				
Pentachlorophenol	0.02	3	✔	✔		
Vinyl Bromide	0.02	1	✔	✔		
1,1,2,2-Tetrachloroethane	0.01	3	✔			
2,4,5-Trichlorophenol	0.01	2				
2,4,6-Trichlorophenol	0.01	2	✔			
3,3'-Dichlorobenzidene	0.01	2	✔			
Acrylamide	0.01	1	✔	✔		
Benzo[a]Pyrene	0.01	18	✔			✔
Chrysene	0.01	18	✔			✔
Dibenzo[a,h]Anthracene	0.01	18	✔			✔
Dimethyl Phthalate	0.01	3				
Ethylidene Dichloride (1,1-Dichloroethane)	0.01	3	✔	✔		
Hexachlorobenzene	0.01	3	✔	✔		✔
Hexachlorocyclopentadiene	0.01	2		✔		
Hexachloroethane	0.01	2	✔	✔		
Nitrobenzene	0.01	2		✔		
o-Cresol	0.01	1				
Propylene Dichloride	0.01	2	✔	✔		
Acenaphthene	0.005	2	✔			✔
Benz[a]Anthracene	0.005	18	✔			✔
Benzo[b]Fluoranthene	0.004	18	✔			✔
Ethyl Acrylate	0.004	1			✔	
4,6-Dinitro-o-Cresol	0.003	2				
4-Nitrophenol	0.003	1				
N-Nitrosodimethylamine	0.003	1	✔			
Pentachloronitrobenzene	0.003	1	✔			
Polychlorinated Biphenyls	0.003	5	✔			✔
4,4'-Methylenebis(2-Chloraniline)	0.002	1	✔			
Trifluralin	0.002	1	✔			✔
1,2,4-Trichlorobenzene	0.001	1		✔		
1,2-Dibromo-3-	0.001	1	✔	✔		

Table 3-1. Summary of Emissions from the Portland Cement Manufacturing Source Category

HAP[32]	Emissions (tpy)	Number of Facilities Reporting HAP (104 facilities in ANPRM data set)	Prioritized Inhalation Dose-Response Value Identified by OAQPS[33]			PB-HAP?
			Unit Risk Estimate for Cancer?	Reference Concentration for Chronic Noncancer?	Health Benchmark Values for Acute Noncancer?	
Chloropropane						
3,3'-Dimethoxybenzidine	0.001	1	✔			
3,3'-Dimethylbenzidine	0.001	1	✔			
4,4'-Methylenedianiline	0.001	1	✔	✔		
4-Dimethylaminoazobenzene	0.001	1	✔			
4-Nitrobiphenyl	0.001	1				
Aniline	0.001	1	✔	✔	✔	
Benzidine	0.001	1	✔	✔		
Benzo[k]Fluoranthene	0.001	18	✔			✔
Hydroquinone	0.001	1				
Indeno[1,2,3-c,d]Pyrene	0.001	18	✔			✔
Isophorone	0.001	2	✔	✔		
N-Nitrosomorpholine	0.001	1	✔			
o-Anisidine	0.001	1				
o-Toluidine	0.001	1	✔			
2-Chloroacetophenone	0.0005	1		✔		
4-Aminobiphenyl	0.0005	1				
N,N-Dimethylaniline	0.0004	2				
Benzo[g,h,i,]Perylene	0.0003	15	✔			✔
Anthracene	0.0001	3	✔			✔
m-Cresol	0.0001	1				
Methoxychlor	0.00004	1				✔
p-Dioxane	0.00004	1	✔	✔	✔	
Triethylamine	0.00003	1		✔		
Heptachlor	0.00002	1	✔			✔
2-Methylnaphthalene	0.00001	1	✔			✔
Phthalic Anhydride	0.00001	1		✔		
Chlordane	0.000004	1	✔	✔		✔
3-Methylcholanthrene	0.0000004	1	✔			✔
B[j]Fluoranthene	0.0000002	1	✔			✔

3.2.1 Dioxin emissions

In addition to the HAPs in Table 3-1 above, this assessment also considered emissions of chlorinated dibenzo-*p*-dioxins and -furans (CDD/Fs, or "dioxins"). In its dioxin inventory for 2000 [*38*], EPA derived a single emission factor of 0.27 ng/ kg[34] clinker (expressed in terms of

[34] TEQs are calculated values that allow us to combine different combinations of dioxins and dioxin-like compounds into a single value representing the equivalent amount of a single compound, 2,3,7,8-tetrachlorodibenzo-p-dioxin.

toxic equivalents of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, or 2378-$TCDD_{(TEQ)}$) for all non-hazardous waste combustion units for this source category, based upon stack tests from 13 sites. This factor was developed for all kilns regardless of type or operational parameters. For this assessment we statistically analyzed available dioxin emission data and developed separate emission factors for four different types of non-hazardous waste combustor kilns.

We obtained dioxin emission estimates and emission factors for 60 non-hazardous waste combustion cement plants from 2002-2006, and calculated a mean emission factor for each of the four facility types. An analysis of variance showed that the emission factors differed significantly among process type (Table 3-2).

Table 3-2. Mean and 95% upper confidence limit (UCL) 2378-$TCDD_{(TEQ)}$ emission factors for Portland cement facilities, by kiln type

Kiln type	Mean emission factor (ng/kg clinker capacity)	95% UCL emission factor (ng/kg clinker capacity)
Dry	0.110	0.229
Dry with preheater and precalciner	0.170	0.614
Dry with preheater	0.168	0.377
Wet	0.768	1.877

These emission factor estimates plausibly bracket the 0.27 ng/kg estimate. Given this plausibility, we characterized CDD/F emissions by kiln type for the Portland cement risk assessment, and calculated plant-specific risks separately using the mean and upper confidence limit (UCL) emissions factors. The complete analysis of dioxin emission data is described in Appendix F.

3.2.2 Radionuclide emissions

This assessment also evaluated risks associated with radionuclides, which are regulated as HAPs when emitted to the air. Emissions of radionuclides from industrial facilities are reported in the 2002 National Emissions Inventory (NEI) in mass-based units of US short tons per year. However, the known hazards from radionuclides are most closely associated with the type of and amount of radioactivity that each radioisotope releases rather than with its mass. Therefore the practice of reporting unspeciated emissions of radioactive substances from a single facility collectively in terms of mass, rather than individually by radioisotope in terms of radioactivity, prevents the accurate estimation of risks posed by radionuclides emitted from industrial facilities.

As a test of possible strategies to evaluate radionuclide hazards, we identified two Portland cement facilities in California that reported emissions in the 2002 NEI. On a mass basis, emissions reported for these facilities are very small but still potentially important because of the high carcinogenic potency of some radionuclides. The NEI entries did not specify which radionuclides were emitted and how much of each was emitted, nor is it clear that the facilities reported radionuclide emissions in a uniform manner.

We performed a more refined analysis (fully described in Appendix G) of radionuclide emissions and risks intended to (1) improve consistency and accuracy of these emissions estimates, (2) evaluate the utility of the NEI data for these HAPs, (3) consistently characterize actual

emissions, and (4) attempt to quantify potential incremental inhalation cancer risks associate with radionuclides.

We estimated radionuclide emissions for the two Portland cement sources using the NEI-reported emissions and scaling factors developed from a "typical" Portland cement facility. We derived the "typical" emission factors using the European Commission Radiation Protection 135 report [*39*], hereafter referred to as the "naturally occurring radioactive material (NORM) report." This approach resulted in three different emission estimates for each of the two facilities, one based on the NEI data and two based on the NORM report (one based on clinker production and the other based on PM emissions). Estimated emissions were modeled with HEM3 to estimate ambient concentrations, population exposures, and risks.

3.3 Risk assessment results – inhalation

This section summarizes the results of the inhalation risk assessment for the Portland Cement Manufacturing source category. The basic risk estimates presented are the maximum individual lifetime cancer risk, the maximum hazard index, and the cancer incidence. Also presented are the HAP "drivers," which are the HAP that collectively contribute 90 percent of the maximum cancer risk or maximum hazard at the highest receptor. Detailed facility-level results for both chronic and acute inhalation risk assessments can be found in Appendix H.

Table 3-3 and Table 3-4 summarize the inhalation risk results for this source category. Acute screening hazard quotients (HQs) were calculated for every HAP shown in Table 3-1 that has an acute benchmark. The highest acute HQ value (and its associated HAP) is shown in Table 3-3. Table 3-4 provides more information on the acute risk screening estimates for HAP that had an acute HQ of greater than 1 for any benchmark. Detailed results for each facility appear in Appendix H.

Table 3-3. Summary of Source Category Level Risks for Portland Cement Manufacturing

Result		HAP "Drivers"
Facilities in Source Category		
Number of Facilities Estimated to be Subject to MACT in Source Category in 1998, from the Proposal Preamble (63 FR 14181, March 24, 1998)	118	n/a
Number of Facilities Identified in NEI and Modeled in Screening Risk Assessment	104	n/a
Cancer Risks		
Maximum Individual Lifetime Cancer Risk (in 1 million) from any Facility in the Category	800	chromium (VI) compounds, arsenic compounds, cadmium compounds, beryllium compounds
Number of Facilities with Maximum Individual Lifetime Cancer Risk:		
Greater than or equal to 100 in 1 million	2	chromium (VI) compounds, arsenic compounds, cadmium compounds, beryllium compounds
Greater than or equal to 10 in 1 million	8	chromium (VI) compounds, cadmium compounds, arsenic compounds, nickel compounds, POM71002, benzene, naphthalene, acrylamide, POM72002,

Table 3-3. Summary of Source Category Level Risks for Portland Cement Manufacturing

Result		HAP "Drivers"
		beryllium compounds
Greater than or equal to 1 in 1 million	29	nickel compounds, chromium (VI) compounds, beryllium compounds, naphthalene, benzene, 1,3-butadiene, 1,3-propane sultone, arsenic compounds, cadmium compounds, POM71002, acrylamide, POM72002
Chronic Noncancer Risks		
Maximum Neurological Hazard Index	10	manganese compounds
Maximum Respiratory Hazard Index	6	chlorine, hydrochloric acid
Maximum Kidney Hazard Index	3	cadmium compounds
Number of Facilities with Maximum Neurological Hazard Index:		
Greater than 1	2	manganese compounds
Number of Facilities with Maximum Respiratory Hazard Index:		
Greater than 1	3	chlorine, hydrochloric acid, beryllium compounds, nickel compounds, chromium (VI) compounds, formaldehyde
Number of Facilities with Maximum Kidney Hazard Index:		
Greater than 1	1	cadmium compounds
Acute Noncancer Screening Results		
Maximum Acute Hazard Quotient	50	AEGL-1, hydrochloric acid
Number of Facilities With Potential for Acute Effects	8	chlorine, formaldehyde, hydrochloric acid
Population Exposure		
Number of People Living Within 50 Kilometers of Facilities Modeled	54,000,000	n/a
Number of People Exposed to Cancer Risk:		
Greater than or equal to 100 in 1 million	400	n/a
Greater than or equal to 10 in 1 million	15,000	n/a
Greater than or equal to 1 in 1 million	470,000	n/a
Number of People Exposed to Noncancer Neurological Hazard Index:		
Greater than 1	3,000	n/a
Number of People Exposed to Noncancer Respiratory Hazard Index:		
Greater than 1	200	n/a
Number of People Exposed to Noncancer Kidney Hazard Index:		
Greater than 1	170	n/a
Estimated Cancer Incidence (excess cancer cases per year)	0.05	n/a
Contribution of HAP to Cancer Incidence		
chromium (VI) compounds	61%	n/a
arsenic compounds	10%	n/a
cadmium compounds	9%	n/a
beryllium compounds	8%	n/a
benzene	5%	n/a

Table 3-4. Summary of Acute Screening Results for Portland Cement Manufacturing

		MAXIMUM ACUTE HAZARD QUOTIENTS		ACUTE DOSE-RESPONSE VALUES			
Screening Results		Max. 1-hr Conc. / Min. Acute Dose-Response Value		Mild Effects		Serious Effects	
HAP	Max. 1-hr. Air Conc. (mg/m^3)	Mild Effects	Serious Effects	AEGL-1 (1-hr) (mg/m^3)	ERPG-1 (mg/m^3)	AEGL-2 (1-hr) (mg/m^3)	ERPG-2 (mg/m^3)
Hydrochloric acid	138	50	5	2.7	4.5	33	30
Chlorine	10	7	2	1.5	2.9	5.8	8.7
Formaldehyde	4	3	0.3	1.1	1.2	17	12

Notes on Screening Process:

1) Screening process is based on a hypothetical worst-case combination of emission rates, meteorology, and exposure location and therefore likely represents an overestimate of actual health risk. The results are being provided only as a tool to aid in the fact-checking of the underlying emissions data and should not be interpreted as actual health risks. A more refined analysis is needed to determine actual risks.
2) The screening was performed for all emitted HAP with available acute dose-response values. Only those pollutants whose screening HQs greater than 1 for at least one acute threshold value are shown in the table.
3) HAP with available acute dose-response values which are not in the table do not carry any potential for posing acute health risks, based on an analysis of currently available emissions data.
4) The acute screening risk assessment results will not be used for decision making.

Notes on Acute Dose-Response Values:

AEGL – Acute exposure guideline levels represent emergency exposure (1-hour) limits for the general public.

AEGL-1 is the exposure level above which it is predicted that the general population, including susceptible individuals, could experience effects that are notable discomfort, but which are transient and reversible upon cessation of exposure.

AEGL-2 is the exposure level above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.

ERPG – US DOE Emergency Removal Program guidelines represent emergency exposure (1-hour) limits for the general public.

ERPG-1 is the maximum level below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing other than mild, transient adverse health effects.

ERPG-2 is the maximum exposure below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action.

3.4 Refined multipathway health risk assessment

3.4.1 Selection of HAPs for this analysis

As noted, facilities in the Portland cement manufacturing source category emit a variety of PB-HAPs, including metals (lead, cadmium, and mercury) and organic compounds (polycyclic organic matter and dioxins). For each facility in this source category, total emissions for each PB-HAP were compared to *de minimis* levels to initially screen for the potential for non-inhalation exposures and risks. The derivation of these *de minimis* emission rates is described in Appendix C.

Emissions of every PB-HAP on EPA's list are not reported for every facility in this source category. However, based on data from individual facilities and knowledge of the Portland cement manufacturing process, every facility is assumed to emit dioxins, with nearly all of these emission rates exceeding the *de minimis* level established for dioxin using the TRIM-based screening scenario. More than half of the facilities also report mercury emissions, and we consider it likely that all such facilities emit mercury. Although only one of these emission rates exceeds the mercury *de minimis* level, mercury is a relatively common PB-HAP reported as emissions from sources included in RTR. Given the potential for exposure via non-inhalation pathways to these two PB-HAPs for RTR facilities in general, and the relatively high emissions of dioxin (relative to the *de minimis* level) for Portland cement facilities in particular, mercury and dioxin were selected as the chemicals for the case study of non-inhalation human health risks.

3.4.2 Selection of facility for case study

To narrow the scope of the case study and enable a more in-depth evaluation, we focused on a single Portland cement facility. We first identified Portland cement facilities that had high emissions for both mercury and dioxins, assumed that higher emissions of the chemicals would generally lead to higher human exposures, and began with facilities having dioxin emission rates exceeding the *de minimis* levels described above, as well as facilities with relatively high mercury emission rates. Of these facilities, we looked for one that had geographic characteristics most similar to the two most significant basic multipathway exposure scenarios (consumption of produce and animals and consumption of fish). Minimum requirements included (a) close proximity to a freshwater lake of reasonable size, and (b) proximity to land used to support a range of agricultural activities (crops and animals).

The Ravena Lafarge Portland cement facility (hereafter referred to as the Ravena facility) in Ravena, NY, meets these criteria and was selected for evaluation in this case study. The Ravena facility is near populated areas, several fishable water bodies, and potential farmland. Although this facility may not necessarily represent the highest multipathway risk of all 91 Portland cement facilities, it is useful for demonstrating the methods of the refined multipathway human health risk assessment (HHRA), *i.e.*, what to do when the emissions from a source category exceed the *de minimis* levels. This is expected to be useful for soliciting feedback on a range of risk assessment-related issues pertaining to EPA's RTR program.

The facility is located approximately 12 miles south of Albany, NY, in the southeastern portion of Albany County (U.S. Census Bureau 2000; population 294,570). The population of Ravena,

NY, located just east of the facility, is 3,369. Nearby counties include Renesselaer, Greene, and Columbia, all in New York. In the 2002 U.S. Department of Agriculture (USDA) Census of Agriculture, these four counties reported that livestock were raised there and crops were grown for human and animal consumption (USDA 2002).

For the purpose of the Ravena HHRA, only dioxin and mercury emissions were evaluated. A scenario layout for the Ravena area was created to use in TRIM.FaTE so that all relevant ingestion pathways could be modeled.

3.4.3 Approach to exposure assessment

For this RTR case study, multipathway exposure estimates and risks were calculated for mercury and dioxin for two basic scenarios:

- A farmer scenario involving an individual living on a farm homestead in the vicinity of the source who (a) consumes produce grown on and meat and animal products raised on the farm, and (b) incidentally ingests surface soil at the location of the farm homestead; and
- A recreational angler scenario involving an individual who regularly consumes fish caught in freshwater lakes in the vicinity of the source of interest.

These two basic scenarios are expected to cover most of the highest possible exposures and risks. In addition to ingestion, non-inhalation exposure to PB-HAPs can also occur by way of the dermal pathway. However, the risk from dermal exposure is expected to be a small fraction of the risk from inhalation exposure or ingestion exposure. Therefore, the risk from dermal exposure was calculated as a special scenario as part of this site-specific refined analysis.

3.4.4 Fate and transport modeling (TRIM.FaTE)

Fate and transport modeling of PB-HAPs was completed using the Fate, Transport, and Ecological Exposure Module (TRIM.FaTE) of EPA's Total Risk Integrated Methodology (TRIM). TRIM.FaTE is a fully coupled multimedia model that estimates the flow of pollutants through time among environmental compartments including air, soil, water, and fish. For detailed information on TRIM.FaTE, refer to EPA's TRIM website (http://www.epa.gov/ttn/fera/trim_gen.html).

Ingestion exposures were calculated for the two exposure scenarios of interest using the TRIM.FaTE media concentrations and typical ingestion exposure algorithms similar to those found in the Human Health Risk Assessment Protocol [7]. Chemical concentrations in intermediate farm food types (*e.g.*, produce, animal products) were calculated using biotransfer factors to estimate the food chemical concentration based on the air and soil concentrations and deposition rates from TRIM.FaTE. The RTR Multipathway Screening TSD (Appendix C) provides details of the approach and methods used to calculate ingestion exposures. Individual lifetime cancer risks for dioxins and chronic non-cancer hazard quotients for dioxins, methylmercury, and divalent mercury were then calculated using oral cancer slope factors and ingestion reference doses (RfDs).

3.4.4.1 Source characterization

For this case study, we modeled dioxin emission rates based on mean and 95th percent upper confidence limit emission factors based on the clinker production of the facility. We present

details about the development of these emission factors in Appendix F. The divalent and elemental mercury emissions modeled were those reported in the 2002 NEI, and transformation of divalent mercury into methylmercury in the sediments was included in the model. Table 3-5 presents the estimated mercury and dioxin emissions to air from the Ravena facility.

Table 3-5. Emissions of Dioxins and Mercury from the Lafarge Facility in Ravena, NY, and Screening Results

PB-HAP		Emissions (tons per year)	Screening Results
Dioxins[a]	95 percent upper confidence limit of mean estimated emission factor	3.28E-06	Exceeds *de minimis* level
	Estimated mean emission factor	1.34E-06	Exceeds *de minimis* level
Mercury – Divalent [b] [soluble fraction, likely mercuric chloride]		5.63E-02	Screens out
Mercury – Elemental [b] [It is assumed that elemental mercury is transported beyond the modeled domain.]		1.69E-01	Screens out

[a] Emissions estimated based on tons of clinker produced using dioxin emission factors.
[b] Emissions reported in 2002 National Emissions Inventory (NEI) (EPA 2002).

The modeling scenario duration was 50 years (*i.e.*, sufficient time to achieve steady state concentrations in the environment), and emissions of both mercury and dioxin were assumed to be constant over the course of the simulation. TRIM.FaTE was used to estimate chemical concentrations in air, soil, and selected surface water bodies (and their corresponding benthic sediment layer), as well as components of a representative aquatic ecosystem in each water body of interest for the risk assessment.

3.4.4.2 Extent and dimensions of modeled environment

The TRIM.FaTE surface parcel layout is the two-dimensional configuration of soil and water regions included in the modeled domain; this is overlain by the air parcel layout. These layouts provide the spatial reference for three-dimensional compartments that hold the modeled chemical mass. The design of the modeling layout was developed based primarily on physical/geographic characteristics of the watersheds in the Ravena area and land-use data for the region. When designing the surface parcel layout, we sought to accurately capture the watersheds surrounding the water bodies selected for modeling (*i.e.*, those that contain fish that people are assumed to eat). In pursuing this goal, parcel shapes were kept as simple as possible to reduce complexity in the layout and the corresponding run time for the model.

The overall spatial extent of the air parcel layout is identical to that of the surface parcel layout, and the square surface source parcel where the Ravena facility is located is identical in size, shape and position to the air source parcel. For this assessment, the remaining air parcel layout was designed as a radial grid centered around the source parcel, consistent with information presented in the EPA's TRIM.FaTE Users' Guide [*40*]. This radial layout minimizes the TRIM.FaTE bias for over-accumulation of mass along the axes of the grid. Overall, 31 air parcels, including the source parcel, are included in the air parcel layout.

The overall spatial extent of the modeling scenario is a 770 km^2 rectangle that captures several significant water bodies in the area and their watersheds. Both divalent mercury and dioxins can accumulate in the farm food chain, so the scenario layout includes two farm homesteads, on the

east and west sides of the facility. The farm homesteads were located in areas where land use is classified as agricultural.

Methylmercury and dioxins bioaccumulate in fish, so four freshwater water bodies were also included in the Ravena layout to estimate exposure for the angler scenario. The Ravena area encompasses many other water bodies including the Hudson River, but for the purposes of TRIM.FaTE modeling, fish populations in three lakes and one pond were modeled. Alcove Reservoir is 7 miles west of the Ravena facility and supplies drinking water to the city of Albany. Kinderhook Lake (8 miles southeast of the facility) and Nassau Lake (11 miles northeast) allow recreational fishing. All three of these lakes are large enough to support large fish populations and were modeled in TRIM.FaTE. A small pond is located 2 miles southwest of the facility. The pond was also modeled, although there is significant uncertainty whether it is large enough to support a fishable aquatic ecosystem. The Ravena facility is within 2 miles of the Hudson River, which was also modeled as a water body in this case study. A fish population was not modeled in the river because of historically high pollutant levels in the river and the difficulty in accurately modeling pollutant movement through a river.

3.4.4.3 Abiotic environment

TRIM.FaTE requires various abiotic environmental properties for each compartment that is included in the scenario (*e.g.*, the depth of surface soil, soil porosity and water content, erosion and runoff rates from surface soil to water bodies, suspended sediment concentration, and others). Where site-specific data were readily available for this assessment they were used. For example, representative site-specific values based on available data were developed to estimate erosion rates for each surface parcel. Rainfall/erosivity values were used from Albany County for plots west of the Hudson River and Rensselaer County for regions east of the Hudson River [*41*]. Soils data were obtained from the Soil Survey Geographic (SSURGO) database for the counties of interest (obtained from the USDA Natural Resources Conservation Service) to calculate site-specific soil erodibility factors. Different cover management factors were used for farm parcels, natural forests, and grasses and herbs.

Regional or national defaults were used in numerous instances, especially for those parameters that are not expected to influence chemical concentration dramatically. For example, a regional pH value of 6.8 was used based on data compiled by McKone *et al*. [*42*] for use in multipathway modeling since variation in pH is not expected to dramatically impact fate and transport of the modeled chemicals. A complete list of TRIM.FaTE inputs for abiotic compartments is provided in Attachment 1 to Appendix I of this document. Surface water and sediment properties for all lakes and the river, along with the sources for these values are also listed in Attachment 1 of Appendix I.

For the modeled water bodies, a water balance was assumed in order to estimate annual flush rates by accounting for inputs to each water body (*i.e.*, runoff from the surrounding watershed and direct precipitation to the lake) and outputs from the water body (*i.e.*, flushing through the lake outlet and evaporation from the lake surface.) In addition, sediment inputs and outputs were assumed to balance. The sediment balance of each watershed/water body system modeled was estimated by accounting for sediment inputs to the lake based on the erosion calculations and the removal of sediment from the modeled system via benthic burial and outflow of suspended sediment in the water column.

TRIM.FaTE uses several meteorological inputs to determine chemical transfers among the air compartments in a scenario via advective transport (*i.e.*, wind-driven physical movement through the atmosphere) and from air to underlying soil or water surfaces via deposition transfers. These processes determine the long-term spatial patterns of chemical distribution within the scenario, and modeled concentrations are highly sensitive to the meteorological inputs used in TRIM.FaTE.

The meteorological inputs required by TRIM.FaTE include wind speed, wind direction, precipitation, ambient air temperature, and mixing height. For this assessment, hourly surface meteorological data from the National Oceanic and Atmospheric Administration's National Climatic Data Center (NCDC) Integrated Surface Hourly (ISH) Database [*43*] were obtained for the closest meteorological station, located in Albany, NY. Three consecutive years of data (for 2001–2003) were readily available and therefore used from this data set.

3.4.4.4 Aquatic ecosystem

To estimate risks to human health for the angler scenario, site-specific models of aquatic food webs were developed in TRIM.FaTE to represent the four modeled water bodies in the vicinity of Ravena, NY (*i.e.*, Nassau and Kinderhook Lakes, Alcove Reservoir, and the unnamed small pond near the facility. Characteristics of the TRIM.FaTE fish compartments used to represent fish in each water body were based on site-specific fish survey data, supplemented by information from the open literature.

The development of each food web consisted of three stages. First, for the three lakes, we collected local fish survey data for the water bodies from the New York State Department of Environmental Conservation (NY DEC), including data on the relative abundance and size/weight distribution of each species, to the extent available. Next, we formulated simplified food webs for each water body, including the Ravena Pond, based on the fish surveys and other biological and physical data for each water body. We used supplemental information on fish feeding habits, aquatic food webs, and biomass densities for different trophic levels from the open literature. Finally, we assigned values for the remaining parameters (*e.g.*, individual body weight, numeric density per unit area, lipid content) for each biotic compartment for each water body in TRIM.FaTE from the available data. Professional judgment was used where available data were incomplete. The process employed to configure TRIM.FaTE aquatic food webs and set model input properties is discussed in greater detail in Addendum C of Attachment 1 of Appendix I.

The following fish species were modeled in the TRIM.FaTE fish compartments:

- *Water Column Herbivore*: Black crappie, common carp, fantail darter, golden shiner, and young of the year;
- *Benthic Omnivore*: Bullhead and sunfish;
- *Water Column Omnivore*: Bluegill, pumpkinseed, redbreast sunfish, rock bass, smallmouth bass, white perch, white sucker, and yellow perch;
- *Benthic Carnivore*: American eel;
- *Water Column Carnivore*: Chain pickerel, largemouth bass, northern pike, tiger musky, and walleye.

3.4.5 Exposure assessment

3.4.5.1 Approach and exposure parameters

For the Ravena facility site-specific HHRA we evaluated a range of ingestion exposures for situations that could be encountered in the vicinity of the Ravena facility. The range of conditions considered when conceptualizing and building the scenario was chosen so that for any given individual, a long-term exposure condition would be reasonably likely to be captured. A summary of the sources of contaminated media for each of the three exposure scenarios evaluated is provided in Table 3-6.

Table 3-6. Ingestion Exposure Scenarios

Scenario	Source of Ingested Media
Consumption of locally-grown produce and animal products, and incidental ingestion of soil	Products and soil from two locations with agricultural land use: o East Farm parcel o West Farm parcel
Consumption of locally-caught fish by sport anglers	Fish from four water bodies: o Alcove Reservoir o Kinderhook Lake o Nassau Lake o Small pond to south
Ingestion of contaminated breast milk by infants	Breast milk; nursing mother would ingest farm and fish media from most exposed locations

For both the farmer and angler scenarios, we assumed that all media consumed were obtained from locations impacted by the Ravena facility. We estimated the central tendency exposure (CTE) using mean ingestion rates obtained primarily from EPA's Exposure Factors Handbooks data on home-produced food consumption for adults [*44*] and children [*45*]. The reasonable maximum exposure (RME) was estimated using the 90th percentile of the distribution of national ingestion rates from the Exposure Factors Handbook. This approach (consuming only contaminated media and ingesting at the 90^{th} percentile rates for all products) resulted in an overestimate of total exposure. However, these conservative assumptions ensure that exposure from any single food item is not underestimated. The CTE scenarios offer a less conservative estimate of exposure.

Other characteristics of exposed individuals were also obtained primarily from EPA's Exposure Factors Handbook. Table 3-7 summarizes the exposure parameters used in the CTE and RME estimates.

Table 3-7. Exposure Parameters Used to Derive Risk and Hazard Estimates.

		1–2 years old	3–5 years old	**6–11 years old**	**12–19 years old**	**20–69 years old**
Body Weight		12.6 kg	18.6 kg	31.8 kg	64.2 kg	71.4 kg
Exposure Frequency		365 days/year	365 days/year	365 days/year	365 days/year	365 days/year
Exposure Period	Non-Cancer Hazard Quotient	2 years	3 years	6 years	8 years	50 years
	Cancer Risk	Lifetime cancer risk calculated with sum of risks from 5 exposure periods above.				
Averaging Period	Non-Cancer Hazard Quotient	2 years	3 years	6 years	8 years	50 years
	Cancer Risk	Lifetime cancer risk calculated with sum of risks from 4 averaging periods above.				
90th Percentile Ingestion Rates	Beef (g/kg/day)	4.5	6.7	11.4	3.53	5.39
	Dairy(g/kg/day)	148	82	54.7	27.0	34.9
	Other (g/kg/day)	95.6	65.7	49.7	33.3	41.1
	Fish (g/day)	3.2	4.8	6.8	9.0	17
Mean Ingestion Rates	Beef (g/kg/day)	1.5	2.2	3.8	1.7	2.6
	Dairy(g/kg/day)	67	37	24.8	10.9	17.1
	Other (g/kg/day)	37.6	26.8	18.9	12.9	16.3
	Fish (g/day)	1.4	2.0	2.7	3.9	6.9

3.4.5.2 Exposure dose estimation

Ingestion exposures for the angler and farmer scenarios for all media were calculated using the Multimedia Ingestion Risk Calculator (MIRC) as average daily doses (ADDs), expressed in milligrams of PB-HAP per kilogram of receptor body weight per day (mg/kg-day). Inputs used to estimate exposure dose and risk included the following PB-HAP environmental media concentrations from TRIM.FaTE:

- Air concentrations (in $\mu g/m^3$);
- Air-to-surface deposition rates for both particle and vapor phases (in $\mu g/m^2$-yr);
- Fish tissue concentrations (in mg/kg wet weight); and
- Concentrations in surface soil and root zone soil (in μg/g dry weight).

These PB-HAP-specific values were then multiplied by empirical biotransfer factors (*e.g.*, soil-to-plant factors, which are the ratios of the concentrations in plants to concentrations in soil) to calculate chemical concentrations in farm food chain media and the receptor- and exposure scenario-specific ADDs. The equations used are presented in Appendix I, Attachment 4.
The calculated average daily doses and lifetime average daily doses were used with carcinogenic potency slope factors (SFs) for ingestion and non-cancer oral reference doses (RfDs) for chronic exposures to calculate individual lifetime cancer risks and hazard quotients, respectively.

This assessment is intended to estimate the maximum individual risk for the exposure scenarios evaluated, and the results are not intended to represent the actual exposure for a typical person living in the vicinity of the evaluated source. Rather, we estimated the exposure for a person who meets the criteria of the scenarios evaluated – that is, someone who consumes only produce grown and animals raised on local farms, and/or someone who regularly consumes self-caught fish from a local lake.

3.4.5.3 Risk Calculations

For this scenario-based risk assessment, we calculated lifetime individual cancer risks for dioxins and non-cancer hazard quotients for dioxins, divalent mercury, and methylmercury using the corresponding carcinogenic potency slope factors for ingestion and oral non-cancer reference doses shown in **Table 3-8**.

Table 3-8. Dose-response Values for PB-HAPs Addressed in this Assessment

PB-HAP	Oral Cancer Potency Slope Factor ([mg/kg-day]$^{-1}$)	Original Source	Ingestion Reference Dose (mg/kg-day)	Original Source
Mercury (elemental)	NA		NA	
Mercuric chloride	NA		3.0E-04	IRIS
Methylmercury	NA		1.0E-04	IRIS
2,3,7,8-TCDD	1.5E+05	EPA ORD [b]	1.0E-09	ATSDR

NA = not applicable. IRIS = EPA's Integrated Risk Information System; EPA ORD = EPA's Office of Research and Development; ATSDR = U.S. Agency for Toxic Substances and Disease Registry. Values presented here are recommended by OAQPS for evaluation of HAPs [8].

3.4.5.4 Breast milk pathway

The US EPA [*46, 47*] and the World Health Organization (WHO) [*48, 49*] have published reports documenting the presence of environmental chemicals and contaminants in human breast milk. These chemicals are ingested by the mother and partition into breast milk. A nursing infant may be exposed subsequently via the mother's breast milk. The nursing infant's exposure can be estimated from the levels of chemical concentrations in the breast milk, which in turn can be estimated from the mother's chemical intake. Exposures can occur for infants via this pathway for dioxins and mercury.

Exposure to dioxins and mercury via breast milk consumption during the first year of life is expected to have a small effect on the estimated lifetime ADD and on the individual's excess lifetime cancer risk for dioxins or the highest chronic non-cancer hazard for either chemical. Therefore, exposures to these chemicals via the breast milk pathway were not considered in estimating the lifetime cancer risk for dioxins or chronic non-cancer hazard quotients for mercury or dioxins for adults. The potential for non-cancer health effects (*e.g.*, when exposures are compared to the ATSDR MRL, which is based on developmental effects endpoints) is of greater concern for nursing infants exposed to either chemical during the first year of life.

The methodology and algorithms used to evaluate the breast milk consumption scenario for this case study are presented separately in Attachment C-2 of Appendix C.

3.4.5.5 Dermal pathway

Compared to both inhalation and ingestion pathways, dermal exposure to PB-HAPs is expected to be a minor exposure pathway. To assess the significance of the dermal exposure pathway, dermal hazard quotients were determined in soil and water for 2,3,7,8-TCDD, divalent mercury (Hg^{2+}), and methylmercury (MHg) as described in EPA's dermal risk assessment guidance for Superfund [*50*]. These quotients were then summed in order to determine an appropriate HQ for each chemical and age class. Site-specific soil and water concentrations from the Ravena modeling scenario were used.

3.5 Ecological risk assessment

3.5.1 Ecological risk screening

As mentioned above, PB-HAP emissions were screened for potential multipathway human health risks using the TRIM-based screening methodology. Emissions of any PB-HAP not passing the initial screen for human health endpoints were assumed to also create a potential for adverse multipathway environmental effects and subjected to more refined ecological assessment (in addition to the human health assessment).

In addition, for both petroleum refineries and Portland cement manufacturing, the potential for adverse ecological effects of non-PB-HAPs in air was generally screened by evaluating the potential for chronic ambient air concentration estimates to exceed chronic human health inhalation thresholds in the ambient air near these facilities. That is, if chronic ambient concentrations were not estimated to exceed their respective chronic reference concentrations, the potential for adverse environmental effects associated with direct contact with air was considered to be insignificant. The rationale behind this thinking is that, in general, chronic human health dose-response threshold values for HAPs are derived from studies conducted on laboratory animals and developed with the inclusions of uncertainty factors that in some cases aggregate as high as 3000. As a result, these human health benchmarks are often significantly lower than levels expected or observed to cause adverse effects observed in studies with other species. We note that there is a scarcity of data on direct atmospheric impacts of these HAPs on other receptors, such as plants, birds, and wildlife. In those cases where the maximum predicted inhalation hazard in an ecosystem is below the level of concern for humans, we have concluded that mammalian receptors are unlikely to be at risk of adverse effects due to inhalation exposures from non PB-HAPs, and have assumed that other ecological receptors are similarly not at any significant risk.

EPA has not yet developed general criteria to select candidate HAPs for direct-contact ecological assessments. However, the large masses of hydrogen chloride (HCl) emitted by Portland cement facilities, and the unusually reactive and acidic nature of these emissions, suggested that HCl should be an appropriate candidate to evaluate for potential adverse effects to ecological receptors by direct contact (*i.e.*, rather than by multipathway exposures). Accordingly, we included an assessment of the threshold ambient air concentration for HCl-induced damage to plant foliage and compared it with the threshold for chronic human health effects. As a result, we concluded that HCl emissions from Portland cement facilities did not pass the ecological screening and we included them in our refined ecological assessment. Our choice of HCl for this case study is not meant to suggest that other HAPs do not pose similar concerns via direct contact; rather, the case study is meant to demonstrate the refined ecological risk assessment methodology for the purposes of review by the SAB.

3.5.2 Refined ecological risk assessment

3.5.2.1 ERA for mercury and dioxin

A refined multipathway ecological risk assessment (ERA) was performed for the Portland cement source category. The portion of that ERA evaluating the potential impacts of dioxin and mercury utilized the same case study facility as in the refined human health multipathway assessment, building off of the estimated media impacts to develop estimates of exposures for four key ecological species (tree swallow, common merganser, bald eagle, and mink) living near each of four bodies of water in the vicinity of the facility. The rationale for the selection of these key species and a detailed description of the methods used to estimate their exposures (including the sources of dietary information) are described in Appendix J. Finally, to characterize the impacts of these exposures, they are compared to derived Toxicity Reference Values (TRV) obtained from the literature intended for those species (Table 3-9). The comparison takes the form of Hazard Quotient (HQ) values (Table 3-10 through Table 3-12), which are used to determine if these exposures might be expected to result in adverse effects. The development of the species-specific TRV values and HQs is explained in Appendix J.

Table 3-9. Summary of Wildlife TRVs (µg[chemical]/kg[BW]-day) for Ravena

Chemical	Avian Values			Mink Values		
	POD (µg/kg-day)	UF_{Tot}	TRV (µg/kg-day)	POD (µg/kg-day)	UF_{Tot}	TRV (µg/kg-day)
2,3,7,8-TCDD	14 E-03	10	1.4 E-03	1.0 E-03	10	0.10 E-3
Methylmercury	78	6	13	55	30	1.8
Divalent Mercury	N/A	N/A	Smaller birds: 26 Larger birds: 65	300	30/1.55	16

N/A = Not applicable.

Table 3-10. Hazard Quotients for Wildlife Exposure to Methylmercury for Ravena

Wildlife Species	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Tree Swallow	0.605	0.004	0.005	0.006
Common Merganser	**1.304**	0.004	0.006	0.005
Bald Eagle	0.634	0.002	0.003	0.003
Mink	**3.919**	0.014	0.021	0.020

[a] Hazard quotients highlighted in blue and **bold** indicate exceed the hazard quotient threshold of 1.

Table 3-11. Hazard Quotients for Wildlife Exposure to Divalent Mercury for Ravena

Wildlife Species	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Tree Swallow	**2.37**	<1	<1	<1
Common Merganser	0.40	<1	<1	<1
Bald Eagle	0.04	<1	<1	<1
Mink	0.98	<1	<1	<1

[a] Hazard quotients highlighted in blue and **bold** indicate exceed the hazard quotient threshold of 1.
[b] The HQs for Hg+2 are likely to be less than 1.0 at water-bodies other Ravena Pond given that exposure doses are more than two orders of magnitude lower for wildlife consuming prey from those water bodies.

Table 3-12. Hazard Quotients for Wildlife Exposure to 2,3,7,8-TCDD[a] for Ravena

Wildlife Species	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Tree Swallow	0.01	0.00004	0.0001	0.0001
Common Merganser	0.70	0.001	0.01	0.002
Bald Eagle	0.77	0.001	0.01	0.004
Mink	**4.27**	0.003	0.03	0.01

[a] Exposure doses are based on the estimated 95-percent UCL dioxin emission rates.
[b] Hazard quotients highlighted in blue and **bold** indicate exceed the hazard quotient threshold of 1.

The only water body for which any HQ exceeds 1 is a hypothetical worst-case farm pond with a surface area of only 0.02 km^2 and a shoreline of 0.8 km. Such a water body would probably support very few individuals of these species, and that any adverse impacts to these few individuals would be unlikely to adversely impact populations of those species. We conclude that populations of piscivorous and insectivorous wildlife should not be adversely affected by methylmercury, divalent mercury, and dioxin emissions from the facility.

3.5.2.2 ERA for hydrogen chloride

3.5.2.2.1 Local impacts

The portion of the refined ERA focusing on the potential impacts of HCl proceeded along two parallel paths, one aimed at evaluating the potential impact of HCl emissions from the source category directly on plant leaves in the vicinity of individual facilities and the other aimed at evaluating the potential for individual facilities to cause or contribute to soil or water acidification in their vicinities to the extent that adverse impacts on plants or animals might result. The first path focused on evaluating estimated maximum ambient impacts from the inhalation risk assessment and comparing them against derived benchmarks for foliar damage. The second path revolved around identifying specific facilities in the source category with the highest potential to cause acidification and then searching for soil and water pH data near those facilities to see if effects can actually be detected.

Following the first path, we conducted a literature search in the attempt to locate information that could be used in developing HCl ecological exposure thresholds for foliar damage. Over 50 scientific databases were accessed in the literature search (described in Appendix K). Available studies included information about gaseous HCl injury to plants from visual observations, photosynthetic and oxygen evolution rates, and electron microscopy of localized cellular damage following exposure. Investigators consistently concluded that foliar damage is caused by gaseous HCl condensing on the leaf surface, producing an aqueous acid solution that promotes cellular injury with degree of injury proportional to exposure to gaseous HCl. This injury is not specific to HCl, but would be expected with exposure to any strong acid.

Available studies were all designed to determine the impact of short-term, high-concentration exposures to gaseous HCl. While these data can provide strong support for the development of acute ecological exposure thresholds, more uncertainty is involved in extrapolating these data to develop chronic thresholds.

Given the limited number of available studies, however, we developed 20-minute threshold exposure estimates (described in Appendix K) based on the lowest reported LOEL (1.5 mg/m^3, at which traces of leaf discoloration and necrosis occurred) and LOAEL (4 mg/m^3, at which 25% of leaves were necrotic), rather than the multiple-effect-level approach recommended in EPA Guidelines for Ecological Risk Assessment. To be consistent with our dispersion model outputs, we selected acute and chronic exposure periods of one hour and one year, respectively. We extrapolated the LOEL and LOAEL exposures to 1-hour equivalent concentrations of 0.5 and 1 mg/m^3, respectively using the common application of Haber's law, as modified by ten Berge et al. [*51*]. Lacking long-term study data, we applied an additional uncertainty factor of 10 to extrapolate the lower of the two acute thresholds (0.5 mg/m^3) from a 1-hour to a 1-year exposure threshold of 0.05 mg/m^3. In our refined ecological assessment for HCl impacts on plants, these thresholds were used to evaluate maximum ambient HCl concentration estimates near Portland cement facilities for potential foliar damage. It is worth noting that while the 1-year threshold for foliar damage is greater than the RfC for health effects (0.05 vs. 0.02 mg/m^3, respectively), the 1-hour threshold for foliar damage is less than the 1-hour California REL (0.5 vs. 2.1 mg/m^3, respectively).

3.5.2.2.2 Regional impacts

Following the second path (evaluating the potential for HCl emissions to cause acidification), we developed a ranking procedure to determine indirect effects of HCl deposition on ecologically sensitive environments. Facilities were ranked according to emission rates, the pH of regional rainfall, surface water alkalinity, and proximity to sensitive environments. Following the identification of potential high-impact facilities, we searched for environmental measurement data near each of the top 4 sources to determine if such measurements might corroborate or refute the hypothesis that current emission levels are resulting in localized acidification impacts. This ranking procedure and the subsequent data search process are described in detail in Appendix J.

3.6 Risk characterization

3.6.1 Inhalation risks

3.6.1.1 Chronic inhalation risk assessment results

The maximum individual cancer risk (MIR) is 800 in a million, dominated by risks associated with emissions of hexavalent chromium compounds and cadmium compounds. Out of the 104 facilities included in the assessment, 8 are associated with an MIR greater than 10 in a million and 29 are associated with an MIR greater than 1 in a million. We estimated the total cancer incidence attributable to the source category to be 0.05 excess cancer cases per year, with about 93% of the total contributed by hexavalent chromium compounds, arsenic compounds, cadmium compounds, beryllium compounds, and benzene. We estimate that 15,000 people reside in areas where the lifetime cancer risk estimate exceeds 10 in a million, and 470,000 people reside in areas where lifetime cancer risk exceeds 1 in a million.

The maximum chronic noncancer hazard index for the Portland cement manufacturing source category is 10, associated with potential effects of manganese compounds on the central nervous system. Other potentially important effects include a respiratory hazard index of 6 (associated with chlorine and hydrogen chloride), and a kidney hazard index of 3 (associated with cadmium

compounds). We estimate that 3000 people reside in areas where the hazard index for neurological effects exceeds 1, 200 people where the respiratory hazard index exceeds 1, and 170 people where the kidney hazard index exceeds 1.

3.6.1.2 Acute inhalation risk screening and refined results

The maximum acute screening hazard quotients (HQs) for hydrogen chloride were 4 and 50, based on potential exceedance of the AEGL-2 and AEGL-1, respectively. Maximum HQs for chlorine and formaldehyde were 7 and 3, respectively, also based on potential AEGL-1 exceedances. All other acute HQs were less than 1. The 8 facilities that exceeded an acute HQ of 1 at the screening level were targeted for a more refined evaluation and are presented in Appendix E. The refined analysis looks at the proximity of maximum predicted impacts to plant property line. Following this refined assessment, maximum predicted acute HQ for hydrogen chloride is 10 based on potential exceedance of the AEGL-1, and less than 1 based on the AEGL-2. Maximum HQs for chlorine and formaldehyde are both 2, also based on potential AEGL-1 exceedances.

When considering acute risks it is important to understand that acute health benchmarks, like any dose-response values, are surrounded by uncertainty. For the Portland cement source category, every acute HQ that exceeded 1 was based on the AEGL-1, a one-time *mild-effect* acute value. These results suggest that (1) facilities that have one-time acute exposures above the AEGL-1 are likely to cause increases in mild, reversible, but nevertheless adverse health effects, and (2) those whose predicted exposures are below the AEGL-1 may or may not pose acute health risks.

3.6.1.3 Radionuclides results

As described in Section 3.2.2 and Appendix G, we tested possible strategies to evaluate radionuclide hazards by estimating emissions for two Portland cement facilities in California that reported emissions in the 2002 NEI. We developed one emissions estimate for each facility using NEI-reported data, and two estimates based on the NORM report [*39*].

The NORM emission estimates for the facilities (*i.e.*, those based on clinker production and PM, respectively, shown in Table 3-13) fell nearly within the same order of magnitude, but were many orders of magnitude less than the NEI-based emissions.

Table 3-13. Estimation of Radionuclide Emissions for the Two California Facilities Using Three Approaches

NTI Site ID	Emissions, Based on NEI Emissions and Speciation Assumptions		Emissions, Based on Clinker Production Scaling Factors		Emissions, Based on PM Emission Scaling Factors	
	^{210}Po (Ci/yr)	^{222}Rn (Ci/yr)	^{210}Po (Ci/yr)	^{222}Rn (Ci/yr)	^{210}Po (Ci/yr)	^{222}Rn (Ci/yr)
NEICA1505122	3.48E+07	7.01E+07	9.59E-01	1.93E+00	7.20E-02	1.45E-01
NEI2CA151186	6.02E+01	1.21E+02	8.13E-01	1.64E+00	1.03E-01	2.07E-01

Estimated cancer risks (Table 3-14) associated with the NEI emissions exceeded unity, but only reached 10 in one million for the NORM-based estimates.

Table 3-14. Risk Calculated for Two California Portland Cement Facilities Using AERMOD Modeling Results and Three Emission Estimation Approaches

NTI Site ID	Conc. ($\mu g/m^3$)	MIR, Based on NEI Emissions and Speciation Assumptions		MIR, Based on Clinker Production Scaling Factors		MIR, Based on PM Emission Scaling Factors	
		^{210}Po	^{222}Rn	^{210}Po	^{222}Rn	^{210}Po	^{222}Rn
NEICA1505122	1.53E-03	2.94E+02	9.86E-01	8.09E-06	2.71E-08	6.07E-07	2.04E-09
NEI2CA151186	2.53E-03	8.43E-04	2.83E-06	1.14E-05	3.82E-08	1.44E-06	4.82E-09

Using the NORM-based clinker production scaling factor, radionuclide emission rates were extrapolated to 91 facilities modeled for the source category. Where actual clinker production data were not available for a facility, clinker production was assumed to equal 95 percent of clinker production capacity, based on the median actual production relative to production capacity from all facilities having data. See Appendix G, page G-7, for details. Maximum incremental risks were estimated for each using the HEM3 model. Of these 91 domestic Portland cement facilities, 4 were estimated to have radionuclide-associated maximum cancer risk higher than 100 in a million. Approximately 35 percent of the facilities (32) were estimated to have maximum cancer risk higher than 10 in a million, and all but one facility had maximum cancer risks higher than 1 in a million. These risk estimates, which are more or less similar in magnitude to risks from all other HAPs combined, suggest that radionuclide emissions may be an important source of risk for this source category. However, the extremely poor quality of available radionuclide emissions data prompts caution in the interpretation of these risk values, especially when comparing to better characterized risks.

In summary, using NEI mass emission estimates for radionuclides appears to result in unrealistically high maximum incremental risk estimates, suggesting that these emissions were reported incorrectly. However, risk estimates based on NORM-based emission rates are still high enough to merit serious concern and to suggest that the lack of adequate radionuclide emission data is an important gap in RTR risk assessments.

3.6.2 Multipathway risks

The results of the human health multipathway risk assessment are presented in this section. Section 3.6.2.1 focuses on the results for 2,3,7,8-TCDD equivalence (a measure that includes all dioxins) and Section 3.6.2.2 focuses on the results for mercury.

For both chemicals, the concentrations and human health risks estimated in this assessment are also compared to analogous outputs estimated using the hypothetical multipathway screening scenario developed for RTR. To accomplish this comparison, the Ravena emission rates were modeled in the TRIM.FaTE screening scenario layout that is used in Step 1 of the multipathway HHRA to derive the *de minimis* levels for screening. In addition, the results from modeling the Ravena emissions in the screening scenario illustrate the level of conservatism associated with the screening scenario and provide additional context for the results estimated for this site-specific risk assessment. Throughout the multipathway HHRA discussion, the results of modeling the Ravena emissions in the screening scenario are labeled "Screening Scenario."

In general, the presentation of results here favors those calculated using RME ingestion rates that

are unlikely to occur but still within the bounds of what is possible. Exposures and risks calculated using more typical CTE ingestion rates for these scenarios are presented as well in some cases for comparison. More detailed discussion and tables of results are presented in Appendix I.

3.6.2.1 2,3,7,8-TCDD results

For 2,3,7,8-TCDD, media concentrations and risks were estimated for two emission rates, one based on a mean emission factor and a second rate based on the 95 percent upper confidence limit (UCL) of the dioxin emission factor (to provide an upper bound risk estimate that takes into account the uncertainty regarding the emissions estimate). A summary of results follows; a complete description of the multipathway risk assessment case study can be found in Appendix I.

For this case study, we estimated individual lifetime cancer risk and non-cancer HQs for 2,3,7,8-TCDD (assumed to be representative of risks from all emitted dioxins) for three scenarios (farmer, angler, and breastfeeding infant) and a range of combinations involving these three scenarios and the food source for the exposed individual. Risk estimates for two emission rates (mean and 95 percent UCL) and two sets of ingestion rate assumptions (central tendency exposure [CTE] and reasonable maximum exposure [RME]) were evaluated. In addition, it was assumed that emission and ingestion rates are constant over the exposure time period for each age group (2 to 50 years for the hazards for different ages, with the cancer risk calculated from the sum of exposures in each age bin).

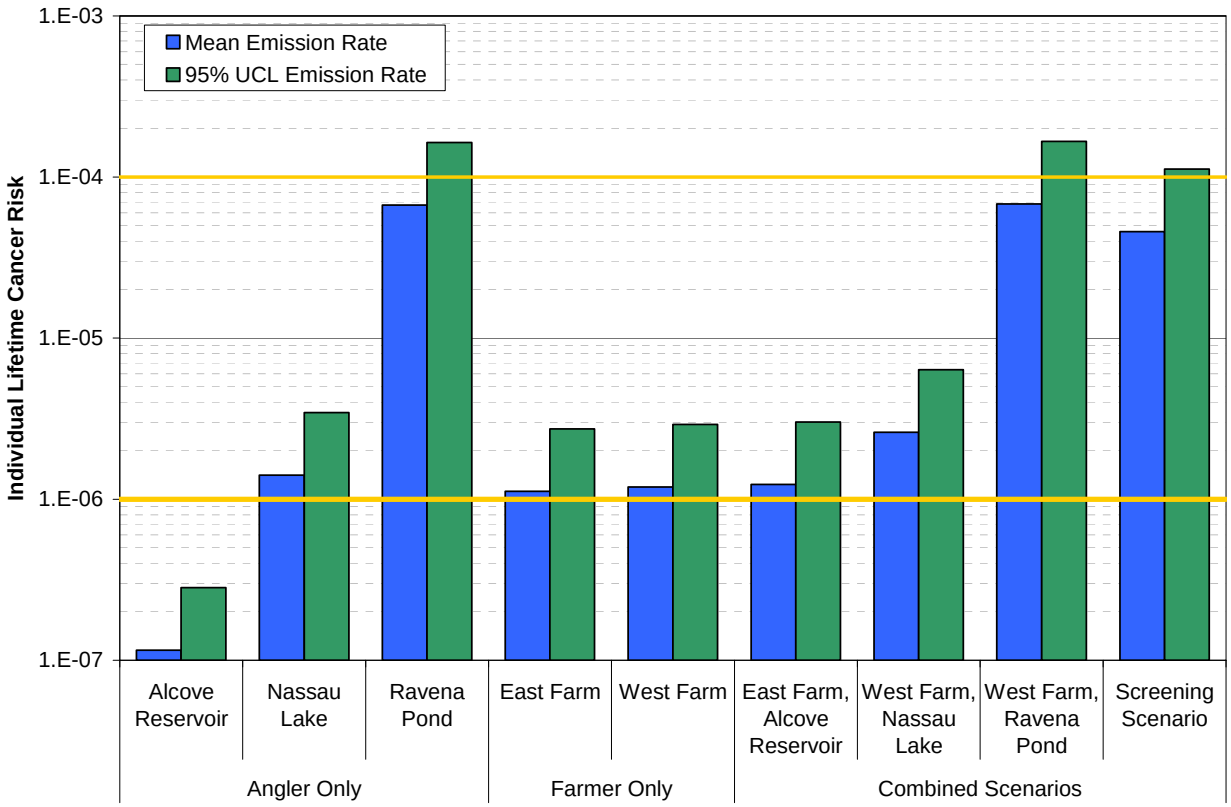
Estimated media concentrations

TRIM.FaTE results for the east and west farm parcels were similar, with air concentration and surface soil concentration higher at the east farm and dry deposition higher at the west farm. The concentrations in fish estimated by TRIM.FaTE were generally lower than total dioxin TEQ concentrations measured in fish in the Hudson River and associated bays, for all water bodies included at the Ravena site (including the pond), with a difference between the modeled and measured values of several orders of magnitude. This outcome seems reasonable given that the model includes a single source of chemical emissions to the air, while the reported values reflect all local and regional sources of dioxins, the contribution of existing background concentrations of dioxins from long-range sources, and any contributions from non-air sources (likely including historical PCB contamination introduced to the Hudson River).

Cancer risk

Cancer risk estimates for 2,3,7,8-TCDD are summarized in Figure 3-1.

Figure 3-1. 2,3,7,8-TCDD Individual Lifetime Cancer Risks for Ravena



Note: Presented results assume 90th percentile ingestion rates for all age groups (RME). For the reader's reference, the yellow lines mark a risk of 1 in 1 million (1e-6) and of 1 in 10,000 (1e-4).

In general, a lifetime individual cancer risk between 1 and 10 in a million was estimated for the combined farmer/angler and individual (farmer *or* angler) scenarios, assuming RME ingestion rates and using the 95 percent UCL dioxin emission factors, for all farm and water body locations evaluated with the exception of the ponds. Consumption of self-caught fish (for the angler scenario) and consumption of beef and dairy products (for the farmer scenario) are the exposure pathways driving cancer risk estimates, with the proportional contribution of these pathways varying by farm and lake location.

Introducing fish harvesting to the Ravena pond within the TRIM.FaTE model probably portrays more realistic fish concentrations and reduces the estimated lifetime cancer risk from 170 in a million to 120 in a million. However, the introduction of fish harvesting at this rate is unlikely to be ecologically sustainable, and at a minimum proves to significantly reduce the chemical concentrations in fish tissues in all fish types. We maintain that the water body with the second highest cancer risks, Nassau Lake, is a more realistic upper bound on potential Ravena area exposures for the angler scenario.

Impact of ingestion rates and dioxin emission factor

If the central tendency ingestion rates are used for produce/meat/animal products and fish or if mean dioxin emission rates are assumed, the estimated individual cancer risk is approximately 3 in a million or less (a decrease of approximately 40 to 60 percent for scenarios excluding

ingestion of fish from the Ravena pond). As expected, CTE ingestion rates are about 40 percent of RME ingestion rates for the food types influencing exposures for the combined farmer/angler scenario; the decrease in risk is proportional to the decrease in relevant ingestion rates. Using the mean dioxin emission factor assumption decreases the risk by roughly the same proportions (the difference between emission factors is similar to the difference in ingestion rates. In combination, if both CTE ingestion rates and mean emission factors are assumed, the estimated individual cancer risk for any given scenario is about 20 percent of the estimated risk when RME ingestion rates and the UCL of the dioxin emission factor are assumed.

Comparison to RTR screening scenario

When individual cancer risks are estimated for the same emission rates, but modeled using the generic RTR screening exposure scenario (*i.e.*, hypothetical modeling environment and high-end farmer and angler exposure scenarios), the risk results are 110 and 46 in a million for 95 percent UCL and mean dioxin emission factors, respectively. These risks are between one and two orders of magnitude higher than site-specific risk estimates for the Ravena scenarios (not including those assuming ingestion of fish from the pond). These results provide an indication of the degree of conservatism that the screening scenario holds, at least in comparison to the site-specific risk assessment conducted for the Ravena site.

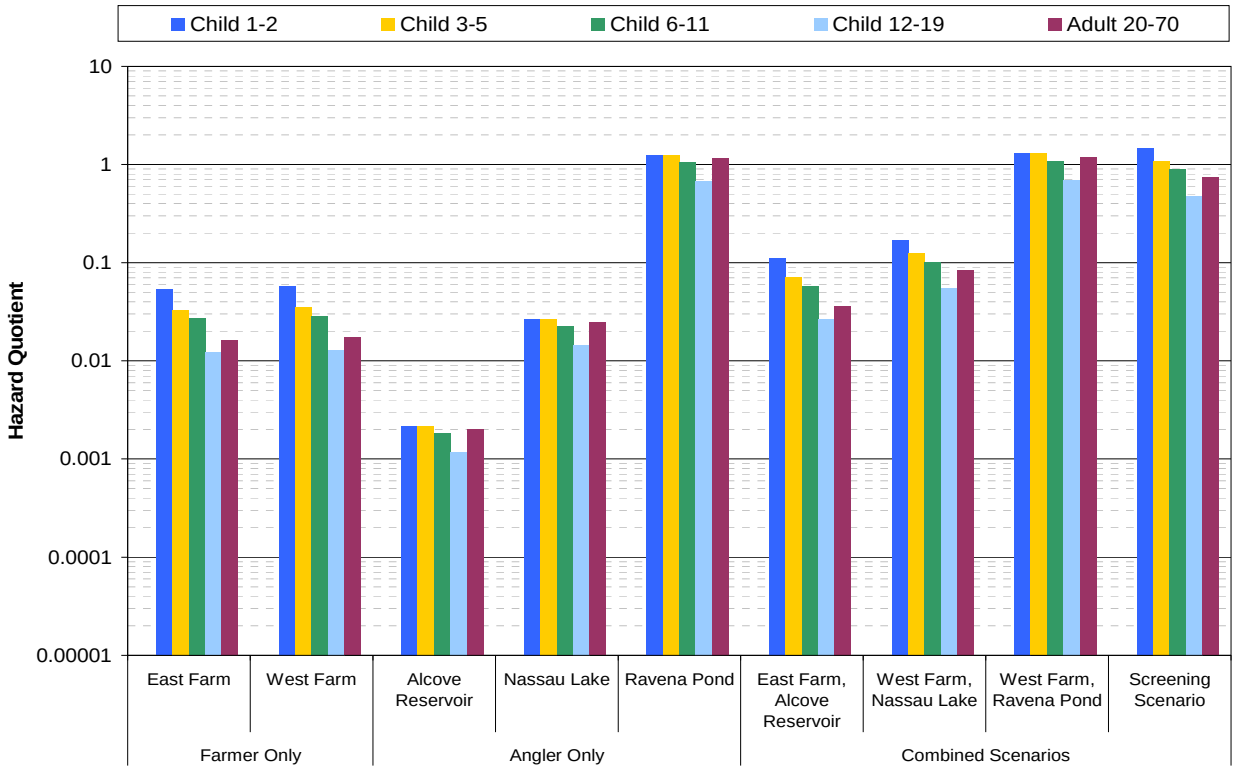
Dermal exposure cancer risks

Dermal exposures and associated lifetime cancer risks were estimated for soil and water exposures (as per [50]). Despite a conservative modeling approach, dermal cancer risk varied from 60 to 590 times less than ingestion risk under different exposure scenarios and locations. Because dermal exposure appears to add so little to ingestion risks, we did not evaluate it further.

Chronic non-cancer hazard quotient

Chronic non-cancer HQs are shown in Figure 3-2. HQs are below 0.1 for all farmer scenarios evaluated and all angler scenarios based on the higher dioxin emission factor, except when consumption of fish from the pond is assumed to occur. The calculated HQ (based on adverse liver, reproductive, developmental, endocrine, respiratory, and hematopoietic effects) for anglers consuming fish from the pond is about 0.7 in children ages 3 to 5, and between 1.1 and 1.3 in all other age groups, if RME ingestion rates are assumed. The estimated HQ for all age groups drops to 1 or below if central tendency fish ingestion rates are assumed, if mean dioxin emission factors are used, or if fish harvesting is introduced to the Ravena TRIM.FaTE modeling scenario. By comparison, when the Ravena dioxin emissions were modeled in the RTR screening scenario, the chronic HQs associated with the RME ingestion rates were estimated to be 0.5 to 1.5 if the higher dioxin emission factor is used, and approximately 0.2 to 0.6 if mean dioxin emissions are used.

Figure 3-2. 2,3,7,8-TCDD Chronic Non-cancer Hazard Quotients for Ravena
(95th Percentile UCL Emission Factor, RME Ingestion Rates)



Infant exposures via breast milk

The HQs calculated for nursing infants were generally higher than HQs for the mothers by about an order of magnitude when the same oral RfD was used to calculate HQ (as noted previously, this analysis did not evaluate whether the RfD used for dioxins is appropriate for evaluating chronic non-cancer hazards to nursing infants). Given the relatively low dioxin exposures assumed for the mother, the calculated HQs for a nursing infant are still below one for all scenario combinations evaluated except the angler-pond scenario. If the nursing mother is assumed to consume fish from the pond, the calculated HQs for a breast-feeding infant are very high given that the mother's HQ is calculated to be approximately 1.2, assuming RME ingestion rates. However, as discussed above, it is unlikely that the pond provides a suitable environment for sustained recreational fishing.

3.6.2.2 Mercury results

For this case study, we estimated individual non-cancer HQs for divalent and methylmercury for three basic scenarios (farmer, angler, and breastfeeding infant) and a range of combinations involving these three scenarios and the location of the exposed individual. It was assumed that emission and ingestion rates are constant over the exposure time period for each age group (2 to 50 years for the hazards for different ages).

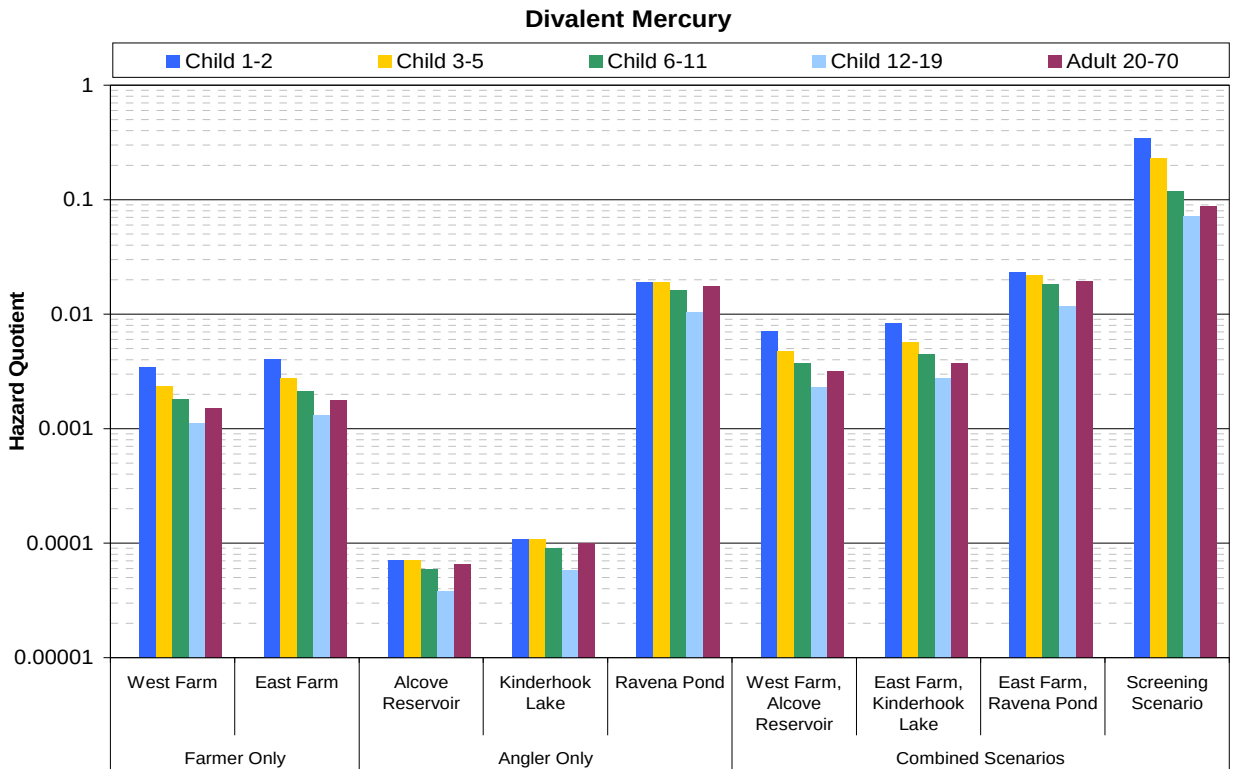
Estimated media concentrations

Model outputs for the east and west farm parcels were similar, with divalent mercury air concentration and dry deposition higher at the west farm and surface soil concentrations and wet deposition higher at the east farm. Mercury concentrations in fish estimated by TRIM.FaTE were generally lower by several orders of magnitude than the divalent, methyl, and total mercury concentrations measured in fish in the Hudson River and associated bays, for all water bodies included at the Ravena site, with the exception of the pond. This difference between modeled and measured concentrations seems reasonable given that the model includes a single source of chemical emissions to the air, while the reported values reflect all local and regional sources of mercury, the contribution of existing background deposition of mercury from long-range sources, and any contributions from non-air sources (including residual mercury resulting from historical deposition in the northeast United States).

Divalent mercury chronic non-cancer hazard quotient

For divalent mercury, chronic non-cancer HQs were below about 0.03 for all combined farmer/angler scenarios evaluated when RME ingestion rates are assumed. As discussed previously, the pond is not likely a plausible, viable source of fish for regular consumption by an angler. If consumption of fish from the pond is excluded, chronic non-cancer HQs were below 0.004 for divalent mercury for all farmer and angler scenarios. When central tendency ingestion rates were used for produce/meat/animal products and fish, the divalent mercury HQ decreased by 50 to 75 percent.

Figure 3-3. Mercury Chronic Non-Cancer Hazard Quotients for Ravena

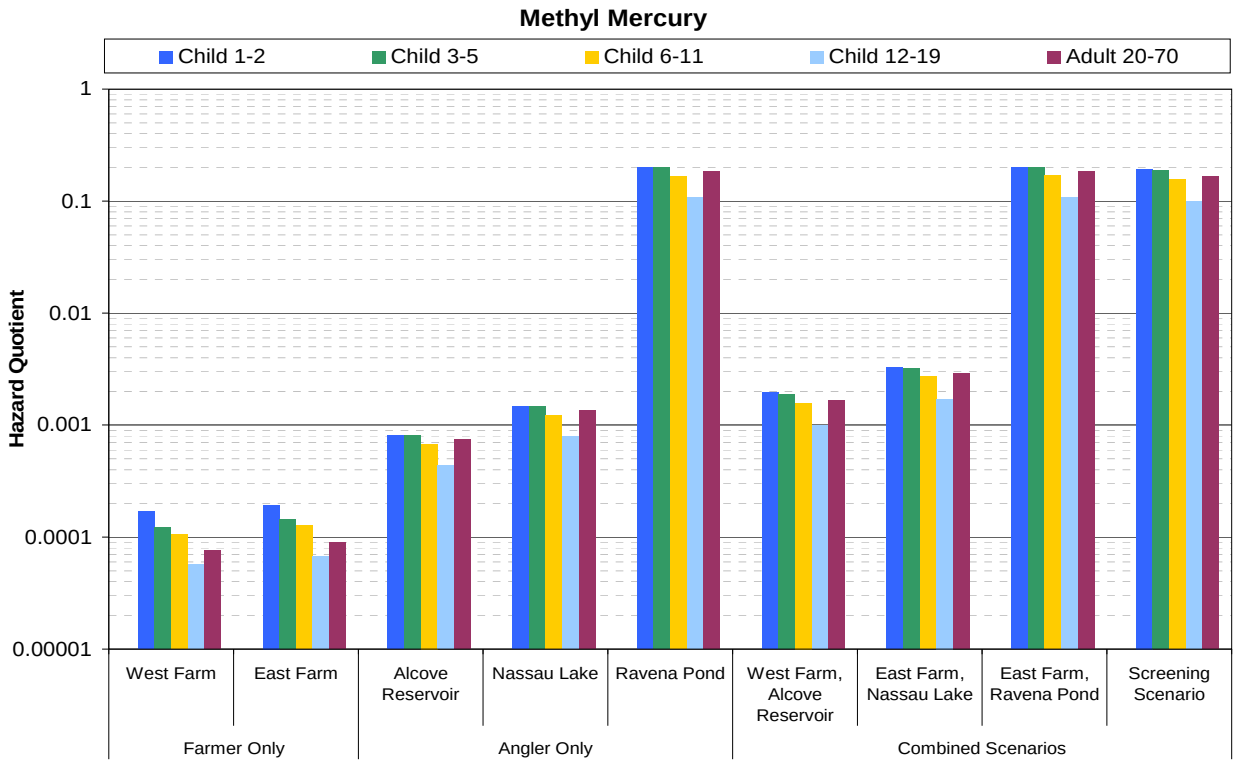


Note: Presented results were calculated using the 90th percentile ingestion rates (RME).

Methylmercury chronic non-cancer hazard quotient

For methylmercury, the estimated HQs ranged from very low values (less than 10^{-4}), associated with consumption of farm products from the west farm, up to a value of 0.2 calculated for consumption of fish from the pond. If the scenario concerning fish consumption from the pond was excluded, the highest estimated HQ for methylmercury was 0.002.

Figure 3-4. Mercury Chronic Non-Cancer Hazard Quotients for Ravena



Note: Presented results were calculated using the 90th percentile ingestion rates (RME).

Comparison to RTR screening scenario

Chronic non-cancer HQs were calculated for divalent mercury using the same emission rates but the general RTR screening exposure scenario (*i.e.*, hypothetical modeling environment and high-end farmer and angler exposure scenarios). These results were between 0.07 and 0.3 assuming RME ingestion rates. These HQs are at least two orders of magnitude higher than site-specific HQ estimates for the corresponding scenarios (with the exception of those involving consumption of fish from the pond).

The chronic non-cancer HQs calculated for methylmercury using the screening scenario were between 0.01 and 0.2 assuming RME ingestion rates. These results are about two orders of magnitude higher than site-specific HQs (with the exception of those involving consumption of fish from the pond). As noted, site-specific mercury results provide an indication of the degree of conservatism associated with the screening scenario, at least in comparison to the case-study

conducted for the Ravena site.

Exposures to infants via breast milk

As described in Attachment C-2 of Appendix C, there is significant uncertainty associated with the parameters used to estimate mercury HQs for breastfeeding infants. For methylmercury, data suggest that HQs for breastfeeding infants will be very similar to HQs for the nursing mother. Given the high level of uncertainty, mercury exposures via breast milk were not quantitatively modeled.

Dermal hazard quotients

Dermal hazard quotients were calculated for soil and water exposures as described in U.S. EPA (2004). Using a highly conservative modeling approach, dermal HQs for divalent mercury were on the same order as HQs calculated for ingestion exposures, suggesting that the methods used to estimate dermal exposures may be overly conservative. However, because the dermal HQs were well below levels of concern (i.e., less than 0.01 for the most conservative exposure scenario), no additional evaluation was conducted.

3.6.3 Combining risks from all facilities and exposure routes

The multipathway risk assessment covers only a single Portland cement facility, and cannot easily be applied to other similar facilities that may have different processes, emission characteristics, meteorology, and surrounding populations. However, a simple extrapolation of maximum individual risks can at least serve as a range-finding tool regarding the potential importance of multipathway risks relative to inhalation risks for the entire source category.

With this in mind, we calculated multipathway risk-to-emission ratios for dioxin and mercury emissions of the Ravena facility for three subsistence exposure scenarios: fishing in a nearby lake, subsistence farming at the most contaminated nearby farm, and both. These calculations assumed that all facilities emit mercury; where mercury emissions were not reported we estimated them using the average mercury-to-dioxin ratio for facilities that reported both. We then used the Ravena risk-to-emission ratios to estimate risks at the other 90 Portland cement facilities for which we estimated dioxin emissions data. This simple extrapolation omits site-specific variations in emission parameters (other than amount emitted), dispersion, and receptor location and behavior, and in effect assumes that every facility differs from Ravena only in the amounts of dioxin and mercury emitted. Since the Ravena facility was selected for multipathway analysis in part because of its proximity to farmland and fishable water bodies, this assumption is likely to create a high bias for risk estimates for other facilities. The results of this extrapolation are shown in Table 3-15.

Table 3-15. Averages of extrapolated risks for dioxins and divalent mercury emitted by the Portland cement source category, based on emissions-to-risk ratios estimated for the Ravena facility.

Exposure scenario	Dioxin cancer risk (avg/max)	Dioxin HQ (avg/max)	Divalent mercury HQ (avg/max)
Nassau Lake	2E-07 / 1E-06	9E-03 / 6E-02	
West Farm	2E-07 / 1E-06	5E-03 / 3E-02	
Kinderhook Lake			3E-05 / 4E-04

Table 3-15. Averages of extrapolated risks for dioxins and divalent mercury emitted by the Portland cement source category, based on emissions-to-risk ratios estimated for the Ravena facility.

Exposure scenario	Dioxin cancer risk (avg/max)	Dioxin HQ (avg/max)	Divalent mercury HQ (avg/max)
East Farm			1E-03 / 2E-02
Combined fishing/farming	4E-07 / 3E-06	3E-02 / 2E-01	2E-03 / 3E-02

The Ravena risk and HQ estimates for dioxin were the highest for any facility in the source category; Ravena HQ estimates for divalent mercury were about midway between the averages and the maxima for the category. The highest lifetime cancer risk estimate for dioxin, for the unlikely combined subsistence fishing and farming exposure scenario at the Ravena facility, was 3 in one million. The average risk for this combined scenario at all facilities was 4 in ten million. At the average risk level it would require about 9 million subsistence fisher-farmers living near Portland cement facilities to produce the inhalation-based incidence rate of 0.05 cancer cases per year. The highest HQs produced by any facility were 0.2 for dioxin and 0.03 for divalent mercury, suggesting no concern for noncancer hazards.

The individual risk estimates from the inhalation and multipathway assessments can be combined by assuming that a subsistence fisher/farmer is also the person with maximum inhalation exposure at each facility. For this source category, however, the multipathway risks are within rounding error of the inhalation risks, and combining them would have no effect. In summary, based on the preliminary emissions dataset used for this case study, health risks associated with the Portland cement source category appear to derive mainly from inhalation exposure rather than from indirect exposure.

3.6.4 General discussion of uncertainties

This risk assessment for the Portland cement source category currently exists only as a pre-ANPRM draft. When complete, it will include a discussion of uncertainties similar to that in the refineries case study (Section 2.4) that will appear in this section. In its current form it is subject to the limitations and uncertainties in the following discussion. We intend to solicit public comment about these parts of the assessment in the hope of reducing the uncertainties in the risk estimates.

- Chromium compounds were reported for about only about 30 percent of the sources in this category, yet they dominate the cancer risk from this source category. Other HAP drivers (*e.g.*, cadmium, benzene, naphthalene) were also reported for less than half the facilities. It is possible that the NEI does not have complete data for some sources that actually emit these HAPs, and that the associated risks may therefore be biased low.

- The reported speciation of chromium compounds into the most common oxidation states (III and VI) significantly impacts predicted risk estimates. In the absence of additional information, the default speciation profile applied to emissions reported as “chromium” or “chromium compounds” for this source category was 92 percent chromium (III) compounds and 8 percent chromium (VI) compounds. Because chromium (VI) compounds were a

dominant risk driver in the assessment, risk estimates for sources with substantially different speciation profiles may be either over- or underestimated.

- It is likely that most or all facilities in this source category emit quantities of naturally occurring radioactive materials, but only three facilities in California reported such emissions and these emissions were not included in the ANPRM data set. The emissions appear to have the potential to contribute substantially to total cancer risk, and risk estimates that omit them may be biased low.

- Emissions of dioxins as TCDD TEQ were estimated for every facility in this data set. Dioxins contributed substantially to total multipathway risk in our case study, and to the extent that our emission estimates were unrepresentative, the resulting risk estimates may be biased either high or low.

- As noted in Section 3.2, there is uncertainty in the identification of sources as major or area in the NEI, which may have affected the risk estimates for the entire category.

- Coordinates in the NEI are checked to ensure that they are generally correct (*e.g.*, in the correct county). However, there can still be errors in the coordinates that result in the emission sources not being properly located on plant property. These errors have the potential to bias the estimates of MIR either high or low.

- As discussed in section 3.6.1.2, the screening assessment for acute impacts suggests that no-effect levels for hydrogen chloride, chlorine, and formaldehyde could be exceeded under worst-case meteorological conditions if maximum hourly emissions of these HAP exceed their average hourly emission rate by a factor of 10. Given the generally conservative design of our acute screening scenario, the HQ values estimated are likely greater than those which could actually occur in the real world, but peak emissions of these compounds should be quantified to support a more refined assessment of potential acute impacts.

4 Supplemental analyses and discussion of uncertainty

4.1 Corrections to the emissions inventory – data analysis

In response to questions and concerns by both EPA scientists and the regulated community about the quality of the NEI emission data used in the initial ANPRM screening stage of RTR risk assessments, we compared the initial ANPRM assessment for petroleum refineries with the revised NPRM assessment to determine how much the emissions estimates (and thus the estimated cancer risks) changed as a result of public comment.[35] (See Appendix A for details of this analysis.)

The ANPRM data set for the petroleum refinery source category included 175 facilities. Through the ANPRM process, data changes or revisions were received for 113, or 65 percent, of the facilities. Changes to the data were supplied by EPA, State or local agencies, trade organizations, and/or facilities themselves. Types of changes to the data included data replacement, emissions changes, process changes, emission release point changes, and facility changes. In addition, 30 facilities were removed and 8 facilities were added by EPA after the screening risk assessment was conducted, resulting in 153 facilities in the NPRM data set.

The total HAP emissions included in the ANPRM data set equal 2,316 tons per year (tpy) and the total HAP emissions included in the NPRM data set equal 2,292 tpy, for an overall reduction of 24 tpy (or 1%). These changes were evaluated by comparing the change in cancer toxicity-weighted emissions (*i.e.*, the emitted mass for each HAP was multiplied by its respective URE). Overall, total toxicity-weighted emissions decreased by 12 percent from the ANPRM data set to the NPRM data set. Toxicity-weighted benzene emissions decreased by 20%, POM emissions by 26%, 1-3-butadiene emissions by 40%. On the other hand, toxicity-weighted naphthalene emissions increased by 19% and nickel emissions by 150%.

Five facilities had maximum individual risk (MIR) estimates that exceeded 100 in a million in the ANPRM assessment. During the comment period three of these facilities submitted revised emission and stack parameters and another submitted revised emission rates only. The fifth was determined not to be a refinery. EPA accepted these revisions, and each facility's MIR risk estimate declined below 100 in a million for the NPRM assessment. No other facilities exceeded this risk level in the NPRM assessment. Thirty-three facilities had MIR estimates between 10 and 100 in a million in the ANPRM assessment; 18 facilities were in this risk range in the NPRM assessment. The comparison showed that, on average, facilities had lower MIR estimates in the NPRM assessment. Facilities with higher MIR estimates in the ANPRM were more likely to provide data changes, and these changes resulted in larger-than-average reductions in MIR.

Estimated overall cancer incidence for the petroleum refining category was 0.08 in the ANPRM, and 0.05 in the NPRM. This reduction in estimated incidence was due almost entirely to data changes for the highest-risk facilities.

[35] The RTR process also allows for further refinements in the risk assessment between the NPRM and final rule, and such refinements were made to the petroleum refineries assessment.

4.2 Short-term emissions and exposures – data analysis

In contrast to the development of ambient concentrations for evaluating long-term exposures, which was performed only for occupied census blocks, worst-case short-term (one-hour) concentrations were estimated both at the census block centroids and at points nearer the facility that represent locations where people may be present for short periods, but generally no nearer than 100 meters from the center of the facility. For large facilities, this 100-meter ring could still contain locations inside the facility property, which could lead to unrealistically high exposure estimates in the acute screening. Since short-term emission rates were needed to screen for the potential for hazard via acute exposures, and since the NEI contains only annual emission totals, we applied the general screening-level assumption to all source categories that the maximum one-hour emission rate from any source was ten times the average annual hourly emission rate for that source. Average hourly emissions rate is defined as the total emissions for a year divided by the total number of operating hours in the year (assuming either continuous operations or more limited operating hours based on additional data). This choice of a factor of ten for screening was originally based on engineering judgment.

Public comments on other RTR assessments have suggested that assuming a maximum hourly emission rate equal to ten times the annualized rate may underestimate actual maximum short-term emissions for some facilities, and thereby also underestimate maximum acute risks. To test the conservatism of the tenfold emission rate assumption, we performed an analysis using a short-term emissions dataset from a number of sources, several of which are refineries, located in Texas (originally reported on by Allen *et al.* (2004)[*52*]). In that report, the Texas Environmental Research Consortium Project compared hourly and annual emissions data for volatile organic compounds for all facilities in a heavily-industrialized 4-county area (Harris, Galveston, Chambers, and Brazoria Counties, TX) over an eleven-month time period in 2001. We obtained the dataset and performed our own analysis, focusing that analysis on sources that reported emitting high quantities of volatile organic HAP over short periods of time (see Appendix B, *Analysis of data on short-term emission rates relative to long-term emission rates*).

To evaluate the potential for release events to cause acute toxicity, we examined low-probability events, *e.g.*, release rates that are exceeded only one hour per year (0.011 % of the time). Ratios of event release rate to long-term release rate varied from 0.00000004 to 74. The 99th percentile ratio was 9 (*i.e.*, an event release rate nine times the long-term average). Only 3 ratios exceeded our default assumption of 10, and of these only one exceeded 11. All three with ratios greater than 10 lasted less than one hour. The median ratio was less than two (*i.e.*, less than twice the annual average).

The factor of ten is intended to cover routinely variable emissions as well as startup, shutdown, and malfunction (SSM) emissions, and although there are some documented emission excursions above this level, our analysis suggests that this factor should cover more than 99% of the short-term peak gaseous or volatile emissions from source categories like petroleum refineries. Similar data were not available for particulate emissions from categories like Portland cement manufacturing, however, and are not likely to be available for individual RTR source categories.

In summary, the tenfold ratio assumption for short-term releases appears to be reasonably protective for the Texas VOC emitters for which data were available, but the analysis is limited by a lack of speciated long-term release data and by an absence of data from facilities that did

not experience a release event during the data collection period. It is also not clear whether, and how, it is appropriate to extrapolate these results to other source categories for which short-term release data do not exist.

4.3 Inventory under-reporting and gaps – data analysis

4.3.1 Ambient monitor-to-model comparison for two Texas refineries

As discussed in section 2.4.1 above, the development of the RTR emissions databases involved quality assurance and quality control processes, but the accuracy of emissions values will nevertheless vary depending on the original source of the data, the amount of incomplete or missing data, errors in estimating emissions values, and other factors.

In order to ground-truth our facility-specific risk assessment results, we compared ambient monitoring data for benzene from two monitoring sites to our dispersion modeling results for those facilities (Appendix L). Benzene monitoring data were obtained from the Texas Commission on Environmental Quality (TCEQ) for two benzene monitors in Texas City, TX. These monitors are each located near residential areas, and within 300 meters of major industrial sources including three large refineries (BP Refining, Marathon, and Valero Refining) and one chemical manufacturing facility (Sterling Chemicals).

A year of hourly monitoring data for each site was paired with hourly measurements of wind speed and wind direction. Raw hourly ambient data were evaluated and adjusted so that non-detected (ND) values were replaced with ½ the minimum detection limits (MDLs). Measurements that lacked matching hourly wind directions were omitted in order to support a statistical analysis of directional source contributions at each monitor. We estimated benzene contributions from other sources in the vicinity of each refinery using the background estimates for the 2002 National Air Toxics Assessment (NATA). We adjusted the monitored concentrations by subtracting these background estimates from each measurement to develop estimates of the refinery-specific benzene contributions at each monitor.

We used AERMOD to develop modeled ambient benzene concentrations due to petroleum refineries alone at both monitor locations using emissions data and meteorological data from the Galveston airport that represented the same time period as the monitor data. All modeling options were identical to those used in the baseline petroleum refinery assessment modeling.

We used analysis of variance to compare average modeled and monitored benzene concentrations, and also the average difference between the two, among 16 wind direction sectors. We used regression analysis to determine if a relationship exists between wind speed and the ratio of hourly monitored to modeled benzene concentrations, a measure of model error.

Annual averages for modeled estimates, monitor data, and the difference between them all varied significantly with wind direction at both monitors ($P<0.001$). Results for the monitor near the BP facility showed a reasonable resemblance between modeled and monitored benzene levels. The effects of the nearby refinery can be clearly seen in both sets of estimates and there appeared to be little overall bias in the annual modeled estimate. Results for the monitor near the Marathon facility showed that the model substantially underestimated the average measured benzene concentrations for every wind direction, and that the difference increased substantially

when the wind blew from the source. When winds moved from the facility toward the monitor, measured concentrations exceeded modeled estimates by more than 2-fold. These results suggest that the benzene emissions inventory for the BP refinery was representative of actual emissions, but that the inventory for the Marathon refinery may have been underestimated by more than twofold. There is no way to know which (if either) facility is representative of the whole sector.

One EPA staff reviewer of Appendix L disagreed with some of the methods used and conclusions reached by the authors. This reviewer's comments and suggestions are attached to the Appendix for consideration as an alternative viewpoint.

4.3.2 Comparison of RTR emissions inventory data and Refineries Emissions Model (REM) data

Throughout the development of the Risk and Technology Review (RTR) program, one potentially significant area of uncertainty has been the quality of emissions data from individual sources. While the general approach has been discussed elsewhere in this document, we note again that there are questions as to the emissions data quality due, in part, to inconsistencies in the values across pollutants and individual sources within a category. Emissions data are essentially estimates since few monitored data exist. Our confidence in emissions estimates varies depending on the original source of the data, the amount of apparent incomplete or missing data, questionable emissions values, and other factors.

To highlight some of these uncertainties in the emissions data and their associated estimated cancer inhalation risks, we compared two emissions datasets – the RTR inventory and an emissions dataset developed using the Refineries Emissions Model or "REM" [*53, 54*] – both of which are reasonable approaches to estimating emissions. After emissions estimates were developed, a dispersion/risk analysis was undertaken. Chronic inhalation exposure concentrations and associated health risks from each facility of interest were estimated using the Human Exposure Model in combination with the American Meteorological Society/EPA Regulatory Model dispersion modeling system (HEM-AERMOD, sometimes called HEM3). The cancer risks associated with each facility's estimated emissions were evaluated using the same dispersion models, exposure assumptions, and unit risk factors that were used to estimate risks based on the RTR data. It is important to note, however, that unlike the RTR database that sometimes includes (for less than half of emissions points) source-specific locations and release characteristics, emissions specifications (e.g., location and release characteristics) are not included in REM. Other limitations and uncertainties are described in Appendix P.

This analysis is not without significant uncertainties. While the purpose of the analysis is to compare risk results from two different approaches to estimating emissions, the REM approach did not account for specific controls at specific facilities. Except in the case of equipment leaks, which included some information on controls due to consent decrees and state/local requirements, the emission factor approach assumed facilities were only controlling at the MACT level or were uncontrolled in the case of cooling towers, which currently do not have a MACT requirement. While the RTR data can account for additional control measures, there is an unquantifiable amount of uncertainty in how emissions are estimated and if they are estimated correctly and completely. Secondly, differences in pollutant coverage may also contribute to these uncertainties. REM includes the 19 HAPs that make up the vast majority of the mass of

emissions included in the RTR database. Additionally, REM assumes these common HAPs (e.g., benzene, naphthalene) are present at all refineries whereas none of these pollutants are reported at all refineries in the RTR database. However, RTR includes 37 additional pollutants reported at up to 34 facilities. While some of these may be reported in error or inappropriately associated with this source category, others may be emitted by individual facilities in this source category and are just not included in the REM dataset because they are not universally emitted by all refineries. Finally, there are differences in the modeling due to different approaches to estimating emissions (e.g., specific number, location, and height of stacks or specific size and location of tank farms vs. default assumptions) that may influence the overall risk results. Without more detailed analyses, we cannot quantify the impact of these uncertainties. Given the uncertainties in this analysis, it is challenging to draw firm conclusions from these findings. Nonetheless, we summarize the main points from the analysis.

First, across all refineries and HAPs, emission estimates are 2.6 times higher using REM; at the facility level, differences between REM and RTR estimates can be an order of magnitude or more.

Second, using the high-end estimate of benzene potency, the highest facility MIR, 30 in 1 million, was the same using RTR and REM data, although the highest-risk facilities were different. The source category MIR for the RTR analysis was driven by naphthalene and POM. The source category MIR for the REM analysis was driven by benzene, naphthalene and POM. The MIR using the low-end estimate of benzene cancer potency is 20 in 1 million for REM and remains 30 in 1 million for the RTR analysis.

Third, assuming the high-end benzene potency value for both analyses, the distribution of facility MIR estimates shifted upwards using the REM data compared to the RTR data; 135 facilities in the REM analysis have MIR estimates greater than 1 in 1 million and 41 facilities have MIR estimates greater than 10 in 1 million, whereas 77 facilities using RTR emissions have MIR estimates greater than 1 in a 1 million and five facilities using RTR emissions have MIR estimates greater than 10 in 1 million. We do not know what the distribution of facility MIR estimates is using the equally probable lower estimate of benzene potency.

Fourth, we looked at the facilities with the highest MIRs from the REM and RTR analyses, using the higher estimate of benzene potency. The top 20 facilities with the highest MIRs based on RTR data have REM-based MIR estimates within an order of magnitude. For the top 20 REM-based MIR estimates, there was somewhat more variability in the magnitude of differences to RTR-based MIR estimates; 14 of these facilities showed differences in estimates of less than an order of magnitude, but the remainder of differences were at least a factor of 10 (and as high as 3,000-fold). Using the low-end benzene estimate may alter these differences, depending on the relative amounts of benzene estimated at each facility.

Fifth, we note that the facilities with the highest MIRs (using the high-end benzene cancer potency value) in either approach are generally different facilities, suggesting a more pronounced difference in the influence of the emissions estimation approach at the facility level than in aggregate. Additionally, the facilities with the highest MIRs in either case, with two exceptions, are not among the facilities with the most dramatic differences in emissions. These order of magnitude changes for facilities did not shift any individual facilities to have MIRs greater than

or equal to 100 in 1 million, but we cannot judge how alternative emissions estimation approaches might affect other source categories. We did not evaluate this issue using the low-end cancer potency value.

Sixth, depending on which benzene cancer potency estimate is used, the estimate for cancer incidence using the REM emissions estimates is three to four times higher than the incidence estimate using the RTR emissions estimates (using the high-end benzene potency estimate, REM incidence is 0.2 cases per year and RTR incidence is 0.05 cases per year; using the low-end benzene potency estimate, REM incidence is 0.1 cases per year and RTR incidence is 0.03 cases per year).

Finally, petroleum refinery emissions are thought to be relatively well-understood compared to those for some other source categories. Therefore, the result that the MIRs are similar in this case may be unique to this source category. It is difficult to generalize the results of this analysis to other source categories.

4.4 Time scale of meteorological data – sensitivity analysis

Ideally, site specific dispersion modeling efforts will employ up to five years of meteorological data to capture variability in weather patterns from year to year. However, because of the large number of facilities in the analysis and the extent of the dispersion modeling analysis (national scale), it was not practical to model five years of data and only the year 1991 was modeled. The selection of a single year may result in under-prediction of long-term ambient levels at some locations and over-prediction at others. To examine the sensitivity of ambient concentrations (and risk estimates) to the use of single-year versus 5-year meteorological data, we ran HEM3 using single-year and 5-year meteorological datasets from four different locations for the petroleum refinery with the highest estimated cancer risk (NEI12486). The four locations (Lancaster, CA; Charlotte, NC; Detroit, MI; and Houston, TX) selected represent several different climates. To determine the impact of meteorological data only we varied those data only, but did not relocate the facility or its surrounding census block and polar receptors.

As shown in Table 4-1, we compared single-year and 5-year averages of several risk metrics, including cancer MIR, incidence, and acute benzene concentration. For cancer MIR and incidence, the comparison was of single-year average to 5-year average. For the acute concentration, the comparison was of the highest hourly value in a single year to the highest hourly value in the 5-year period. Consequently, for this comparison, the highest acute concentration for any single year will always be less than or equal to the highest acute concentration for a 5-year period.

The single-year and 5-year estimates of cancer MIR and incidence differed by only 7 percent on average. The single-year average MIR and incidence differed from the 5-year average by as much as 18 percent below to 28 percent above, but differed by less than 10 percent in 15 out of the 20 comparisons. The highest acute concentration for a single year was, on average, 10 percent lower than the highest acute concentration for a 5-year period, with a maximum difference of 37 percent when examining the Charlotte NC meteorological data. A closer look at the Charlotte data found that one of the five years was significantly higher then the other 4 years. An examination of the meteorological data for year with the highest acute concentration at

Charlotte suggests that an unlikely confluence of factors led to that value, including a very low wind speed and a wind direction in line with the source and receptor.

In summary, the relatively small differences in risk metrics described above suggest that, in a majority of the cases considered, that the use of meteorological data for a single year does not introduce significant uncertainty into the risk assessment relative to other sources of uncertainty that limit reporting risk estimates to one significant figure.

Table 4-1. Comparison of Risk Assessment Results for 1-Year vs. 5-Year Meteorological Data for a Petroleum Refinery (NEI12486).

					Percent Difference Between 1 and 5-Year Data[36] [37]		
Location	**Data Year**	**Cancer MIR**	**Incidence**	**Acute Benzene Conc. (µg/m^3)**	**Cancer MIR**	**Incidence**	**Acute Benzene Conc. (µg/m^3)**
Lancaster, CA	90	1.43E-04	8.390E-04	3954	2	-2	-22
	91	1.39E-04	8.280E-04	5053	-1	-3	0
	92	1.40E-04	9.040E-04	4635	0	6	-8
	93	1.39E-04	8.280E-04	5025	-1	-3	-1
	94	1.39E-04	8.690E-04	4890	-1	2	-3
	90-94	1.40E-04	8.540E-04	5053	NA	NA	NA
Charlotte, NC	91	1.20E-04	4.380E-04	2395	6	5	-37
	92	1.12E-04	3.900E-04	2523	-1	-7	-34
	93	1.09E-04	3.820E-04	3811	-4	-9	0
	94	1.10E-04	3.960E-04	2546	-3	-5	-33
	95	1.15E-04	4.850E-04	2532	2	16	-34
	91-95	1.13E-04	4.180E-04	3811	NA	NA	NA
Detroit, MI	02	1.93E-04	9.897E-04	7301	28	11	-6
	03	1.48E-04	9.136E-04	7523	-2	2	-3
	04	1.43E-04	8.198E-04	7593	-6	-8	-2
	05	1.34E-04	8.661E-04	7081	-11	-3	-9
	06	1.38E-04	8.773E-04	7739	-9	-2	0
	02-06	1.51E-04	8.934E-04	7739	NA	NA	NA
Houston, TX	87	8.06E-05	4.950E-04	6781	-16	17	-3
	88	9.72E-05	4.740E-04	6889	2	12	-1
	89	9.69E-05	3.660E-04	6959	1	-14	0
	90	1.20E-04	4.390E-04	6643	26	4	-5
	91	8.29E-05	3.460E-04	6779	-13	-18	-3
	87-91	9.56E-05	4.241E-04	6959	NA	NA	NA
Mean of the Absolute Values of the Percent Differences					**7**	**7**	**10**

[36] For cancer MIR and incidence, the comparison is of single-year average to 5-year average. For acute concentration, the comparison is of the highest hourly value in a single year to the highest hourly value in the 5-year period.
[37] A negative value indicates that the 5-year value is higher than the single-year value.

4.5 Location of meteorological stations – sensitivity analysis

Meteorological data for HEM3 are selected from a list of 158 National Weather Service (NWS) surface observation stations across the continental United States, Alaska, Hawaii, and Puerto Rico. In most cases the nearest station is selected as representative of the conditions at the subject facility. Two petroleum refinery facilities[38] furnished representative meteorological datasets as part of the ANPRM process. For these two facilities, the facility-supplied meteorological data were utilized in place of the HEM "nearest selected" station.

For each facility, located by its characteristic latitude and longitude coordinates, the nearest meteorological station was used in the dispersion modeling. The average distance between a modeled facility and the nearest meteorological station was 72 km. Usually, the nearest meteorological station is the most appropriate to use because it best represents the conditions at the facility. However, there are situations where a more distant meteorological station may better represent facility conditions. For example, the nearest meteorological station for an inland facility may be on the coast, but the coastal effects on winds may make a more distant meteorological station more appropriate to use. We performed a sensitivity analysis to examine the variability attributable to the selection of meteorology station. We selected four petroleum refineries in different climates (Torrance, CA; Texas City, TX; Canton, OH; and Marcus Hook, PA) that had at least three surface meteorology stations within 200 km of the refinery. We then ran HEM3 for each refinery and for each meteorology dataset to estimate cancer MIR, incidence, and acute benzene concentration. The results are given in Table 4-2. Overall, cancer MIR, incidence, and acute benzene concentration differed from the values based on the nearest meteorological station by 26, 41, and 17 percent, respectively. Cancer MIR varied by a much as 63 percent below to 51 percent above the value based on the nearest meteorological station. Incidence varied by a much as 68 percent below to 120 percent above the value based on the nearest meteorological station. The acute benzene concentration varied by a much as 49 percent below to 21 percent above the value based on the nearest meteorological station. In summary, in three of four cases the meteorological station nearest the facility yielded risk estimates similar to most of the more distant stations. In the fourth case the more distant stations yielded risk estimates that were characteristically 20 to 40 percent lower, but it is not clear that the more distant stations would be more representative. Overall, the differences usually fall within rounding error for the 1-significant-figure characterization of risks, and therefore appear to be relatively less important than other sources of uncertainty, e.g., dose-response values or emission rates.

[38] For NEI8406, data from the Fairbanks, Alaska met station from the year 2001 modeled and for NEI46556, data from St. Croix, Virgin Islands met station from the year 2005 was utilized.

Table 4-2. Impact of Meteorological Station Selection on Risk Assessment.

							Percent Difference With Nearest Station [a]		
NEI ID	**Surface Meteorological Station**	**Upper Air Meteorological Station**	**Distance to Surface Station (km)**	**Cancer MIR**	**Incidence**	**Acute Benzene Conc. ($\mu g/m^3$)**	**Cancer MIR**	**Incidence**	**Acute Benzene Conc. ($\mu g/m^3$)**
NEI109	Philadelphia, PA	Atlantic City, NJ	17	2.0E-5	7.8E-4	1040	0	0	0
	Wilmington, DE	Atlantic City, NJ	22	2.1E-5	8.0E-4	1060	7	3	2
	Allentown, PA	Albany, NY	91	2.1E-5	9.1E-4	1080	4	17	4
	Baltimore, MD	Sterling, VA	127	2.0E-5	9.4E-4	1110	-1	21	7
	Sterling, VA	Sterling, VA	196	2.7E-5	8.9E-4	1100	36	14	6
NEI11574	Akron, OH	Pittsburgh, PA	16	5.1E-6	1.6E-4	397	0	0	0
	Cleveland, OH	Pittsburgh, PA	78	7.8E-6	3.5E-4	464	51	120	17
	Pittsburgh, PA	Pittsburgh, PA	102	4.8E-6	1.8E-4	412	-6	13	4
	Columbus, OH	Dayton, OH	149	5.3E-6	2.4E-4	431	4	50	9
	Erie, PA	Buffalo, NY	176	5.4E-6	2.0E-4	387	5	25	-3
NEI12044	Galveston, TX	Lake Charles, LA	12	1.6E-5	1.2E-3	17434	0	0	0
	Houston, TX	Lake Charles, LA	80	6.0E-6	3.9E-4	9160	-63	-68	-47
	Port Arthur, TX	Lake Charles, LA	105	8.9E-6	6.6E-4	12500	-44	-45	-28
	Lake Charles, LA	Lake Charles, LA	180	1.2E-5	6.9E-4	21100	-25	-43	21
	Victoria, TX	Corpus Christi, TX	201	7.6E-6	4.7E-4	8940	-53	-61	-49
NEI21034	Los Angeles, CA	Miramar, CA	10	8.7E-7	7.8E-4	171	0	0	0
	San Diego, CA	Miramar, CA	162	1.1E-6	4.7E-4	146	21	-40	-15
	Daggett, CA	Desert Rock, NV	180	4.4E-7	3.4E-4	129	-49	-56	-25
Mean of the Absolute Values of the Percent Differences							**26**	**41**	**17**

[a] A negative value indicates that the value for the station is lower than the value for the station nearest the source.

4.6 Atmospheric chemistry – sensitivity analysis

While the AERMOD model is not capable of simulating complex atmospheric chemical reactions, such as those simulated by the CMAQ model, it does contain the option to simulate an exponential atmospheric decay of the pollutant being modeled, a chemical transformation process which is common to many gaseous pollutants such as VOCs and SO_2. For the general RTR dispersion modeling and risk characterization, this feature was omitted from the analysis, under the assumption that such atmospheric decay would not occur prominently over the transport distances and time scales typically involved in estimating maximum risk impacts. To test this assumption, we conducted a separate modeling study to evaluate exactly how much including the atmospheric decay in our simulations would change our estimates of maximum individual risk (MIR), cancer incidence levels, and noncancer HQs.

For the petroleum refineries source category risk characterization, the primary risk drivers were seen to be benzene, ethylene dibromide, naphthalene, and polycyclic organic matter (POM). In general, none of these pollutants is expected to be highly reactive; with the exception of naphthalene and other small POM whose average half-lives are around 10 hours, their typical atmospheric half-lives are on the order of days or tens of days [*55*]. Only a few of the major pollutants emitted by petroleum refineries have atmospheric half-lives less than 12 hours. They are (in increasing order of estimated half-life): 1,3-butadiene (*ca.* 1 hour), aniline (2 hours), formaldehyde (4 hours), cresols (4-5 hours), phenol (9 hours), and acrolein (12 hours). Four other HAPs emitted by petroleum refineries have atmospheric half-lives between 12 and 24 hours; all other HAPs have estimated half-lives greater than a day.

To simulate the effects of the exponential decay on the MIR and incidence levels, we modeled one of the highest risk petroleum refineries (NEI7988) utilizing the AERMOD exponential decay option using the atmospheric half-life of the risk driver, benzene, which is reported as 14 days. Table 4-3 and Figure 4-1 depict the results of this model run, indicating that both MIR and incidence values showed no significant changes. We also performed simulations of this source utilizing smaller and smaller half-life values to determine how short the half-life needed to be to effect significant reductions in these estimated risk values. These results are also presented in Table 4-3 and Figure 4-1. For pollutants with half-lives greater than about 30 minutes, predicted MIR values are reduced by less than a few percent, suggesting that neglecting the influence of atmospheric decay for these pollutants is appropriate in predicting MIR estimates. For pollutants with half-lives of about 10 minutes or less, MIR impacts are reduced by at least 10%. A review of available literature on atmospheric half-lives for HAPs identified only one with an estimated atmospheric half-life less than 10 minutes (N-nitrosodimethylamine, with an estimated half-life of about 3 minutes). It is not known to be emitted by petroleum refineries. In addition, we identified two additional HAPs with half-lives less than 1 hour (methyl hydrazine and 1,1-dimethyl hydrazine, each with an estimated half-life of about 30 minutes). Accurately estimating MIR values for sources of these pollutants may require including the simulation of atmospheric decomposition.

Because cancer incidence can be contributed to by exposures significantly farther from the facility than the MIR location, the effect of atmospheric decay can be noticed at longer half-life values. As presented in Table 4-3 and Figure 4-1, a 5% reduction in incidence can be noted for

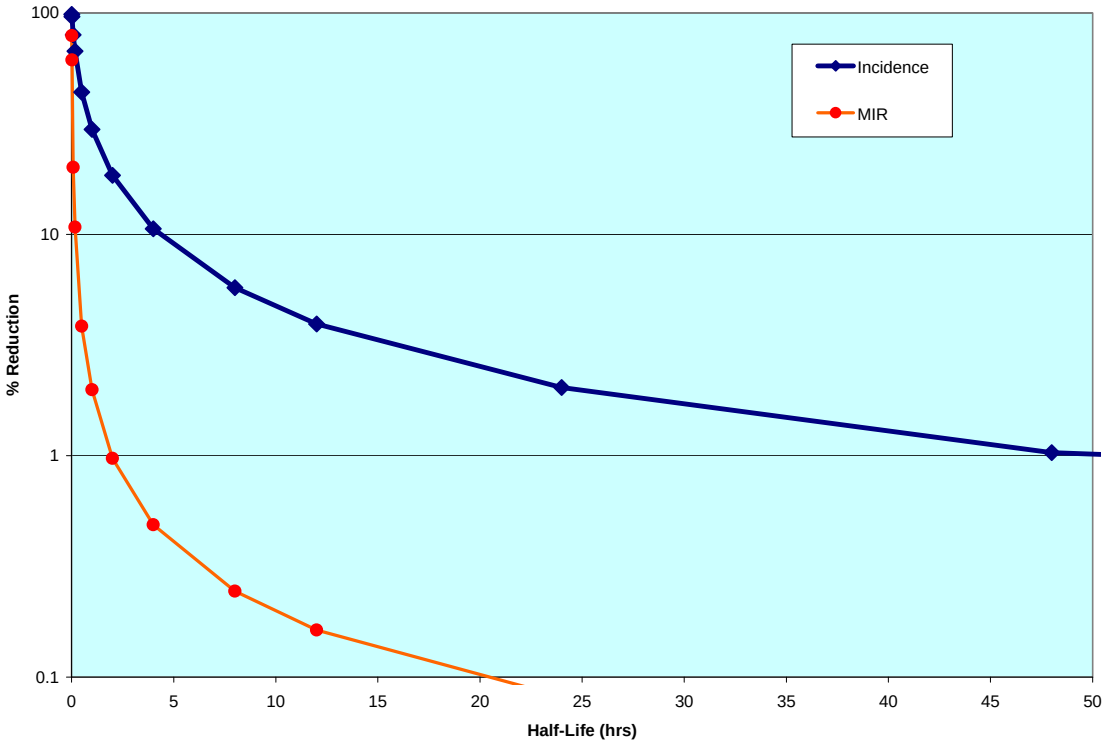
pollutants with a half-life of 8 hours and a 10% reduction for pollutants with a half-life of 4 hours.

Since we report our risk results to one significant figure (in order to avoid implying greater precision in the results than is warranted), these results suggest that including the simulation of atmospheric decay in this type of risk assessment is only necessary for pollutants whose half-lives are less than about 4 hours, and will not impact the estimation of MIR. For petroleum refineries, the only pollutants meeting this criterion are 1,3-butadiene, aniline, and formaldehyde. Since these pollutants were seen to contribute minimally to the cancer risks for petroleum refineries, omitting their reactivity will have little impact on the chronic risk results for this source category.

Table 4-3. Effects of Exponential Decay on MIR and Incidence levels.

Pollutant Half Life (pollutant)	MIR	% Reduced	Cancer Incidence	% Reduced
None (RTR Modeling)	29.3	-	0.000581	-
14 days (benzene)	29.3	0.0001	0.000580	0.1
48-hour	29.3	0.0004	0.000575	1.0
24-hour	29.3	0.0008	0.000569	2.0
12-hour	29.2	0.2	0.000558	3.9
8-hour	29.2	0.2	0.000548	5.7
4-hour	29.1	0.5	0.000520	10.6
2-hour	29.0	1.0	0.000474	18.5
1-hour	28.7	2.0	0.000408	29.8
30 minutes	28.2	3.9	0.000326	43.9
10 minutes	26.1	10.8	0.000190	67.2
5 minutes	23.4	20.1	0.000118	79.7
1 minute	11.3	61.4	0.000022	96.2
32 seconds (acrolein)	6.2	79.0	0.000008	98.6

Figure 4-1. MIR/Incidence Reduction as a function of Half-Life



As a final note, while it may be important to include the impact of atmospheric decay in order to accurately estimate MIR and incidence for the direct impacts of a relatively few fast-reacting HAP, this does not account for the potential for the by-products of such decomposition to cause any health risks. In fact, many decomposition products of rapidly-decaying HAPs are themselves HAPs. Formaldehyde, for example, is a byproduct of the atmospheric decomposition of acrolein, 1,3-butadiene, acetaldehyde, and propionaldehyde. While the creation of such byproducts depends on many interactions in the atmosphere that are beyond the scope of the typical RTR assessment, their potential formation should nonetheless be acknowledged when characterizing the ultimate risk results.

4.7 Deposition – sensitivity analysis

While AERMOD is capable of simulating the deposition of particulate pollution, we have generally not incorporated the simulation of particle deposition into the dispersion modeling being performed for RTR assessments, hypothesizing that its role is relatively minor in the calculation of risk metrics for these types of sources. Since much of the pollution arising from Portland cement manufacturing is emitted in particle form, we decided to test the hypothesis for this source category.

We chose the 5 highest risk facilities for the sensitivity analysis. We first simulated atmospheric dispersion and risks without including deposition and plume depletion in the calculation and then repeated the simulations using the deposition and depletion algorithms contained in AERMOD. We used parameters to characterize the deposition process which were indicative of a typical fine particle emission mixture after a particulate control device (baghouse or fabric filter, which are

required for Portland cement facilities). The results, shown in Table 4-4, provide a comparison of MIR values and total cancer incidence values with and without including the simulation of deposition. For all 5 facilities, deposition was seen to decrease the estimated MIR values by less than 3% (ranging from 0.05% to 2.27%). The estimated cancer incidence values for each facility decreased slightly more than this when deposition was included in the simulation, but all values decreased by less than 4% (ranging from 0.32% to 3.85%).

Table 4-4. Comparison of estimated cancer MIR and incidence with and without considering deposition and depletion at five Portland cement facilities.

Facility ID	Maximum Individual Cancer Risk (with dep and part and vap depletion)	Maximum Individual Cancer Risk (without deposition or depletion)	% Change	Incidence (with dep and part and vap depletion)	Incidence (without dep or depletion)	% Change
PTC_NEI22453	5.00E-05	5.11E-05	-2.12	8.33E-04	8.64E-04	-3.59
PTC_NEI22838	2.14E-06	2.16E-06	-1.21	7.34E-06	7.62E-06	-3.64
PTC_NEI2PRT14367	6.54E-06	6.57E-06	-0.44	4.32E-04	4.37E-04	-1.16
PTC_NEIAL1150002	4.19E-06	4.19E-06	-0.05	7.19E-04	7.22E-04	-0.32
PTC_NEIAZ0250421	1.68E-06	1.72E-06	-2.27	4.71E-05	4.90E-05	-3.85

The results of this sensitivity analysis bore out the hypothesis that ignoring deposition for these Portland cement sources will not significantly affect the risk results. It should be noted, however, that this result should not be extrapolated to the simulation of sources which contain either uncontrolled particle emissions or a significant fraction of coarse particles, as these particles are known to deposit from the atmosphere at significantly higher rates than fine particles.

4.8 Location of receptor populations – data analysis

The HEM3 system estimates ambient concentrations at the geographic centroids of census blocks and other receptor locations specified by the user. In cases where the census block centroid was found to be located on facility property (as determined from satellite imagery) the receptor is moved to the nearest off-site location. The model accounts for the effects of multiple facilities when estimating concentration impacts at each block centroid. In RTR risk assessments, we combine only the impacts of facilities within the same source category, and assess chronic exposure and risk only for census blocks with at least one resident (*i.e.*, locations where people may reasonably be assumed to reside rather than receptor points at the fenceline of a facility). Chronic ambient concentrations are calculated as the annual average of all estimated short-term (one-hour) concentrations at each block centroid. Possible future residential use of currently uninhabited areas is not considered. Census blocks, the finest resolution available in the census data, are typically comprised of approximately 40 people or about ten households.

Despite comments to the contrary on several residual risk rule proposals, we do not expect the use of census block centroids as receptors for chronic exposure to introduce a low bias into the risk assessment. However, we acknowledge that it does introduce uncertainty because the highest residential exposure (assumed to be the residence nearest the facility in this analysis)

may be either greater or less than the exposure at the block centroid. Large discrepancies are less likely in highly populated areas because census blocks are typically small in such areas. In less-populated areas census blocks are typically large, and it is possible that exposures at some residences could vary substantially from those at the census block centroid.

To test for possible systematic bias associated with the use of census block centroids as receptors for chronic exposure, we compared the estimated cancer MIR values at the census block centroid and at the nearest residence for the 21 petroleum refinery facilities with cancer MIR values greater than or equal to 10 in a million (Appendix M). We chose to refine the risk estimates for these facilities to ensure that we captured the MIR for the source category, and we contend that these facilities are representative of the entire source category with respect to the relative difference in risk estimates between the census block centroid and nearest residence. Because risk estimates are highly sensitive to the distance from source to receptor, we considered the possibility that by selecting the facilities with the highest estimated risk, we were biasing our sample with cases where the distance to the census block centroid is small. Larger distances from source to census block centroid typically mean that the census block is large and more likely to result in larger differences in estimated risk across the block. To determine if we were biasing our sample with cases of small distance to receptor, we calculated the median and 95th percentile values of distance from the source to the nearest census block centroid (at least one person in the block) for the sample of 21 facilities and the entire source category of 150 facilities. The median distance for the sample was 170 m compared to 190 m for the entire source category. The 95th percentile distance for the sample was 530 m compared to 550 m for the entire source category. These small differences indicate that the census blocks near facilities in the source category are not significantly larger than those in the sample. Therefore, we believe the sample is likely representative of the relative difference in risk estimates between census block centroid and the nearest residence.

In eleven cases, the census blocks were small, with a typical distance from the centroid to the block boundary less than 100 m. In these cases, we estimate that the MIR values at the census block centroid and nearest residence are identical. There were two cases where census blocks were relatively large, but for which the residences were located near the centroid. In these cases, we also estimate that the MIR values at the census block centroid and nearest residence are identical. In the remaining eight cases, the census blocks were relatively large, and the MIR values at the centroid were higher than the values estimated at the nearest residence, with the overestimates ranging from 40 to 2000 percent. In seven of these cases, the census blocks overlap both facility property and adjacent residential areas. In such situations, MIR estimates at the centroid are biased high because most of the area between the centroid and the boundary of the block nearest the facility is not residential.

In summary, in this analysis of facility-specific MIR values, the centroid-generated values overestimated the residence-generated values by 40 to 2000 percent in less than half the cases, were equivalent in over half the cases, and there were no cases where the value at a residence exceeded that at the centroid of the census block containing the residence. The MIR estimate for the source category as a whole was the same using either methodology. While it is possible that exposures at a residence in a large census block could be higher than at the centroid of the block, this analysis supports the use of the centroid as a reasonable representation of the MIR for the

nearest receptor, and it provides strong evidence that the use of the centroid is not creating a low bias in the overall risk results, indicating, in fact, the tendency for this approach to overestimate MIR values for the highest risk sources, and thus the MIR for the source category as a whole.

4.9 Population mobility – data analysis

The practice of omitting long-term population mobility does not bias the estimate of the theoretical MIR, nor does it affect the estimate of cancer incidence since the total population number remains the same. It does, however, affect the shape of the distribution of individual risks across the affected population, shifting it toward higher estimated individual risks at the upper end and reducing the number of people estimated to be at lower risks, thereby biasing the risk estimates high. Therefore, although our initial refined assessments did not address long-term activity (*i.e.*, migration or population growth trends over 70 years), we applied an example *ex post facto* adjustment for long-term population mobility to the estimates of lifetime cancer risk for both case studies, using residence time and emigration data from the Bureau of Census describing long-term migration patterns in the US.

As shown in Table 4-5 below, modeling long-term migration behavior can substantially reduce the numbers of people with lifetime cancer risks above specific levels. This is offset, however, by an increase in the number of people at lower levels of risk (*i.e.*, those who move into an area to replace those who leave). The estimate for total cancer incidence remains unchanged for carcinogens that have linear low-dose relationships. Details of this mobility analysis are provided in Appendix N.

Table 4-5. Results of adjustment of estimated inhalation cancer risk for long-term migration behavior for two source categories.

	Portland Cement		Petroleum Refineries	
Cancer Risk	Unadjusted	Adjusted	Unadjusted	Adjusted
> 100 in a million	0	0	0	0
> 10 in a million	125	43	4,378	2,556
> 1 in a million	5,066	2,955	430,800	292,003

4.10 Acute exposure – discussion of uncertainties

We have biased the acute screening results high, considering that they depend upon the joint occurrence of independent factors, such as peak hourly emissions rates, worst-case meteorology (*i.e.*, conditions that produce the highest 1-hour concentration at any modeled location), and human presence at the point of maximum impact. Furthermore, in cases where multiple acute threshold values are available we have chosen the most conservative of these values, thereby likely incorporating a high bias on estimates of potential acute health risks. In the cases where these results indicated the potential for exceeding short-term health thresholds we have refined our assessment by developing a better understanding of the geography of the facility relative to potential exposure locations and refining the acute multiplier based on input from industry. We were not able to refine these assessments to incorporate the true variability of short-term emission rates; such data for HAP emissions are seldom available. Thus, by maintaining the peak-to-mean emission ratio of 10, even in our refined acute assessments (absent better data), we believe the results generally overstate the potential for acute impacts.

4.11 Dose-response assessment – discussion of uncertainties

4.11.1 Chronic dose-response

This assessment used a single set of toxicological dose-response values, typically extrapolated from high-dose animal exposure or human occupational exposures, to estimate risk. These dose-response values include embedded default exposure assumptions such as inhalation rate and body mass (*e.g.,* 70 kg), and do not explicitly take into account inter-individual variability in health status or genetic makeup. Additional uncertainty arises from extrapolating from animals to humans, high-level shorter-term exposures to lower-level long-term exposures and from occupational exposures in healthy adult workers to environmental exposures to sensitive individuals or life stages. Some of these factors may result in overestimates of risk and others in underestimates, but in general the development of these dose-response values incorporates modeling approaches that are biased toward overestimating rather than underestimating risk.

Consistent with EPA guidance, RfCs are developed by using quantitative factors to account for uncertainties in developing values protective of sensitive subpopulations. The degree of aggregate uncertainty would depend on the individual HAP.

Most of the UREs in this assessment were developed using linear low-dose extrapolation. Risks would be overestimated if the true dose-response relationship (which is usually unknown) is sublinear and underestimated when the dose-response curve is actually supralinear. In addition, the extrapolation for most of the carcinogenic HAPs began with a statistical lower-bound (*i.e.*, protective) estimate of the lowest tumorigenic dose, rather than the central estimate. The exception to this is the URE for benzene, which is considered to cover a range of values considered to be equally plausible, and which is based on maximum likelihood estimates. The impact of selecting either end of the benzene URE range is discussed explicitly in section 2.3.3. Extrapolation from a lower statistical limit tends to overestimate risks for carcinogens with sparse health effects data, with the degree of overestimation decreasing as health effects data become more robust. In general, EPA considers most UREs to be upper-bound estimates based on the method of extrapolation, meaning they represent a plausible upper limit to the true value. (Note that this is usually not a true statistical confidence limit.) The true risk is generally likely to be less, could be as low as zero, but also could be greater. EPA's upper bound estimates represent a "plausible upper limit to the true value of a quantity" (although this is usually not a true statistical confidence limit).[39] In some circumstances, the true risk could be as low as zero; however, in other circumstances the risk could also be greater.[40] When developing an upper bound estimate of risk and to provide risk values that do not underestimate risk, EPA generally relies on conservative default approaches.[41]

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[39] IRIS glossary (www.epa.gov/NCEA/iris/help_gloss.htm).

[40] The exception to this is the URE for benzene, which is considered to cover a range of values, each end of which is considered to be equally plausible, and which is based on maximum likelihood estimates.

[41] According to the NRC report *Science and Judgment in Risk Assessment* (NRC, 1994) "[Default] options are generic approaches, based on general scientific knowledge and policy judgment, that are applied to various elements of the risk-assessment process when the correct scientific model is unknown or uncertain." The 1983 NRC report *Risk Assessment in the Federal Government: Managing the Process* defined *default option* as "the option chosen on the basis of risk assessment policy that appears to be the best choice in the absence of data to the contrary" (NRC, 1983a, p. 63). Therefore, default options are not rules that bind the agency; rather, the agency may depart from them in evaluating the risks posed by a specific substance when it believes this to be appropriate. In keeping with EPA's

The scientific understanding of dose-response relationships for these and other chemicals is continually evolving. For example, 28 of the HAPs in these case studies (counting PAH and glycol ether compounds each as a single HAP) are currently under assessments or revisions within EPA's IRIS program. In cases where IRIS currently lacks dose-response assessments, values were taken from other sources according to a predetermined hierarchy. In the case of benzene where a range of UREs is presented in EPA's IRIS database, we have chosen the most conservative of these values for these assessments, favoring to err on the side of health protection by estimating higher potential cancer risk. Since the resulting risk results were not negligible, we have also performed a sensitivity analysis to demonstrate the impact of choosing the lower end of the benzene URE range on MIR and estimated cancer incidence in Section 2.3.3.

4.11.2 Acute dose-response

Many of the UFs used to account for variability and uncertainty in the development of acute reference values are similar to those developed for chronic durations, but more often using individual UF values less than 10. UFs are applied based on chemical-specific or health effect-specific information (*e.g.*, simple irritation effects do not vary appreciably between human individuals, hence a value of 3 is typically used), or based on the purpose for the reference value (see the following paragraph). The UFs applied in acute reference value derivation include: 1) heterogeneity among humans; 2) uncertainty in extrapolating from animals to humans; 3) uncertainty in LOAEL to NOAEL adjustments; and 4) uncertainty in accounting for an incomplete database on toxic effects of potential concern. Additional adjustments are often applied to account for uncertainty in extrapolation from observations at one exposure duration (*e.g.*, 4 hours) to arrive at a POD for derivation of an acute reference value at another exposure duration (*e.g.*, 1 hour).

Not all acute reference values are developed for the same purpose and care must be taken when interpreting the results of an acute assessment of human health effects relative to the reference value or values being exceeded. Where relevant to the estimated exposures, the lack of threshold values at different levels of severity should be factored into the risk characterization as potential uncertainties. Further, when we compare our peak 1-hour exposures against MRL values (which are derived for 1- to 14-day exposure durations), we note that peak emission events are unlikely to last more than an hour. As such, these comparisons are a very conservative screen which is only useful in ruling out potential exposures of concern, limiting our ability to interpret situations where MRL values are exceeded.

4.12 Compounds without dose-response assessments – sensitivity analysis

Finally, many HAPs lack any dose-response values at all for cancer, chronic non-cancer and acute effects. In some cases this reflects a relative lack of concern for the pollutant/effect in question, but in others it may result from a lack of scientific data. This factor has the potential to

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goal of protecting public health and the environment, default assumptions are used to ensure that risk to chemicals is not underestimated (although defaults are not intended to overtly overestimate risk). See EPA 2004 *An Examination of EPA Risk Assessment Principles and Practices,* EPA/100/B-04/001 available at: http://www.epa.gov/osa/pdfs/ratf-final.pdf.

result in an understatement of risk if there are effects associated with these HAPs at environmental exposure levels.

In response to the SAB's previous concerns about our inability to estimate risks for HAPs that lack peer-reviewed dose-response assessments, we conducted a "what-if" analysis based on median and upper-bound estimates of toxic potency for these substances. Details of this analysis are presented in Appendix O. We included in this analysis the Portland cement and petroleum refinery source categories individually, and also all US sources combined. The analysis was based on toxicity-weighting of the 2002 NEI, a process that provides an estimate of relative potential cancer risk and noncancer respiratory hazard posed by each HAP. We weighted the pollutant emissions as follows: (1) for noncancer respiratory effects, the emitted amount for each chemical was divided by its RfC or similar chronic no-effect exposure level; (2) for cancer, the emitted amount of each chemical was multiplied by its inhalation URE for cancer.

For HAPs that lacked an RfC or URE, we selected as surrogates the following range of values selected from the universe of chronic RfCs and UREs in the OAQPS table of prioritized chronic dose-response values for inhalation exposure (http://www.epa.gov/ttn/atw/toxsource/table1.pdf):

Percentile of toxicity	RfC[42] (mg/m^3)	URE ($1/\mu g/m^3$)
5	2.28	1.0e-6
25	0.2	6.0e-6
50	0.0098	6.8e-5
75	0.00065	6.1e-4
95	0.000023	4.8e-2

All HAPs lacking an RfC were assigned this range of surrogate RfCs. Only HAPs lacking a URE but having an EPA or IARC WOE equivalent to "possible carcinogen" or greater were assigned the range of surrogate UREs. Toxicity-weighted emissions (TWEs) for cancer and noncancer effects were kept separate. TWE's were normalized by dividing each score by the maximum TWE from all chemicals that had a dose-response value.

The following compounds produced TWEs suggesting that, if their toxicity or carcinogenic potency were found to be at the high end of the surrogate ranges, they could contribute substantially[43] to total risk:

Source category	Noncarcinogens	Carcinogens
Petroleum refineries	2,2,4-trimethylpentane POMs Biphenyl carbonyl sulfide chromium III	quinoline

[42] Low RfCs connote high toxicity, so the RfC decreases as toxicity increases. UREs are directly proportional to carcinogenic potency, so the URE increases as potency increases.

[43] "Substantially" for this table is the 95th percentile TWE for the HAP exceeding 10% of the largest TWE for the sources.

Source category	Noncarcinogens	Carcinogens
Portland cement facilities	carbonyl sulfide POM 1,3-propane sultone chromium III bromoform	none
All NEI sources	2,2,4-trimethylpentane carbonyl sulfide POM propionaldehyde	ethyl acrylate

This toxicity-weighting analysis, while obviously simplistic, is nevertheless useful for determining whether particular assessments have overlooked any potentially important unassessed chemicals, and for informing decisions prioritizing pollutants for toxicity testing and dose-response assessment. Similar analyses can be conducted easily on other source categories, and with other inventory years, to identify new candidates.

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Appendix A: Comparison of initial risk estimates with risk estimates refined by public comment for petroleum refineries

Appendix A

This appendix presents the evolution of the data set used for the NPRM petroleum refinery source category risk assessment and the amount that the estimated cancer risks changed as a result of public comment[1]. This discussion includes comparisons of the maximum individual cancer risks (MIR), cancer incidence and population exposure, HAP emissions, and toxicity-weighted HAP emissions. In addition to a brief background, a summary of the types of changes received for the petroleum refinery data set through the ANPRM process is also presented.

A.1 Background

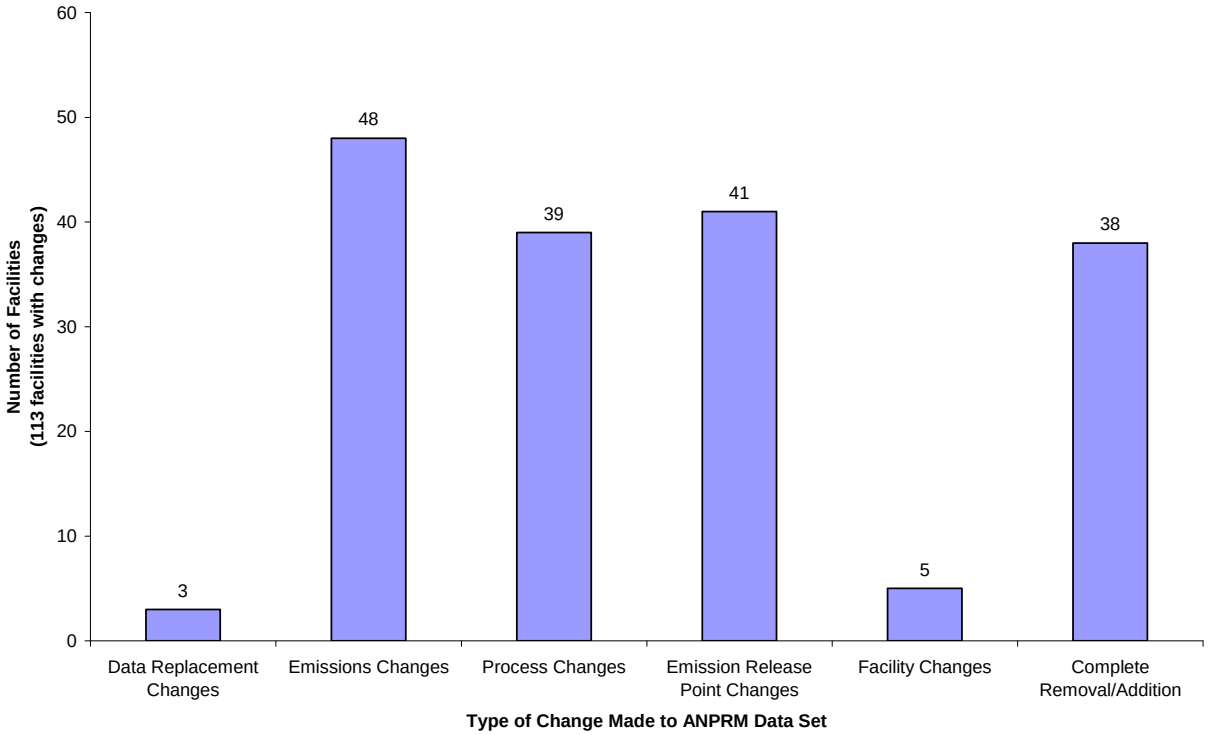
A screening risk assessment was conducted for the ANPRM in September 2006. After receipt of data revisions through the ANPRM process, a risk assessment was conducted for the NPRM in July 2007. The HEM3 model (with AERMOD) was used for both assessments; however, several updates to the model were made during the intervening time. Updates mainly involved the meteorological station data library and included: the library was expanded to include additional stations, the data was processed using a newer version of AERMET, and data was obtained for newer years than previously used. In addition, the HAP library of dose-response values was updated between the ANPRM and NPRM.

A.2 Summary of Data Revisions Received Through ANPRM Process

The ANPRM data set for the petroleum refinery source category included 175 facilities. Through the ANPRM process, data changes or revisions were received for 113, or 65 percent, of the facilities. Changes to the data were supplied by EPA, State or local agencies, trade organizations, and/or facilities themselves. Types of changes to the data included data replacement, emissions changes, process changes, emission release point changes, and facility changes. Data replacement changes were those where the commenter could not match the existing NEI data with new data they wished to provide and instead provided a complete replacement of the entire petroleum refinery NEI data set for that facility. Emissions changes were those related to the emissions estimates, such as an update to emissions estimates or the removal or addition of HAP from an existing emission point. Process changes were those changes that added or removed a process at the facility from the refinery source category, *e.g.*, by changing the MACT code. Emission release point changes included corrections to location coordinates and updated stack parameters. Finally, facility changes included changes in name, ownership, or status of the facility to major or area. A listing of these changes received for the petroleum refinery source category can be found in the RTR docket (EPA-HQ-OAR-2006-0859), item number 0261. In addition to those changes described above, 30 facilities were removed and 8 facilities were added by EPA after the screening risk assessment was conducted, resulting in 153 facilities in the NPRM data set. A summary of the frequency and types of changes made to the ANPRM data set are shown in Figure 1. As can be seen in Figure 1, the most frequent types of data changes were changes to emissions, followed by changes to emission release point information, process changes, and the complete removal or addition of facilities from the data set.

[1] In other words, this appendix compares two drafts of the Petroleum Refineries baseline risk assessment, before and after public comment. Subsequent changes made to the NPRM draft assessment to create the final baseline assessment are not discussed in this appendix.

Figure 1. Frequency and Type of Changes Made to ANPRM Data Set for Petroleum Refineries

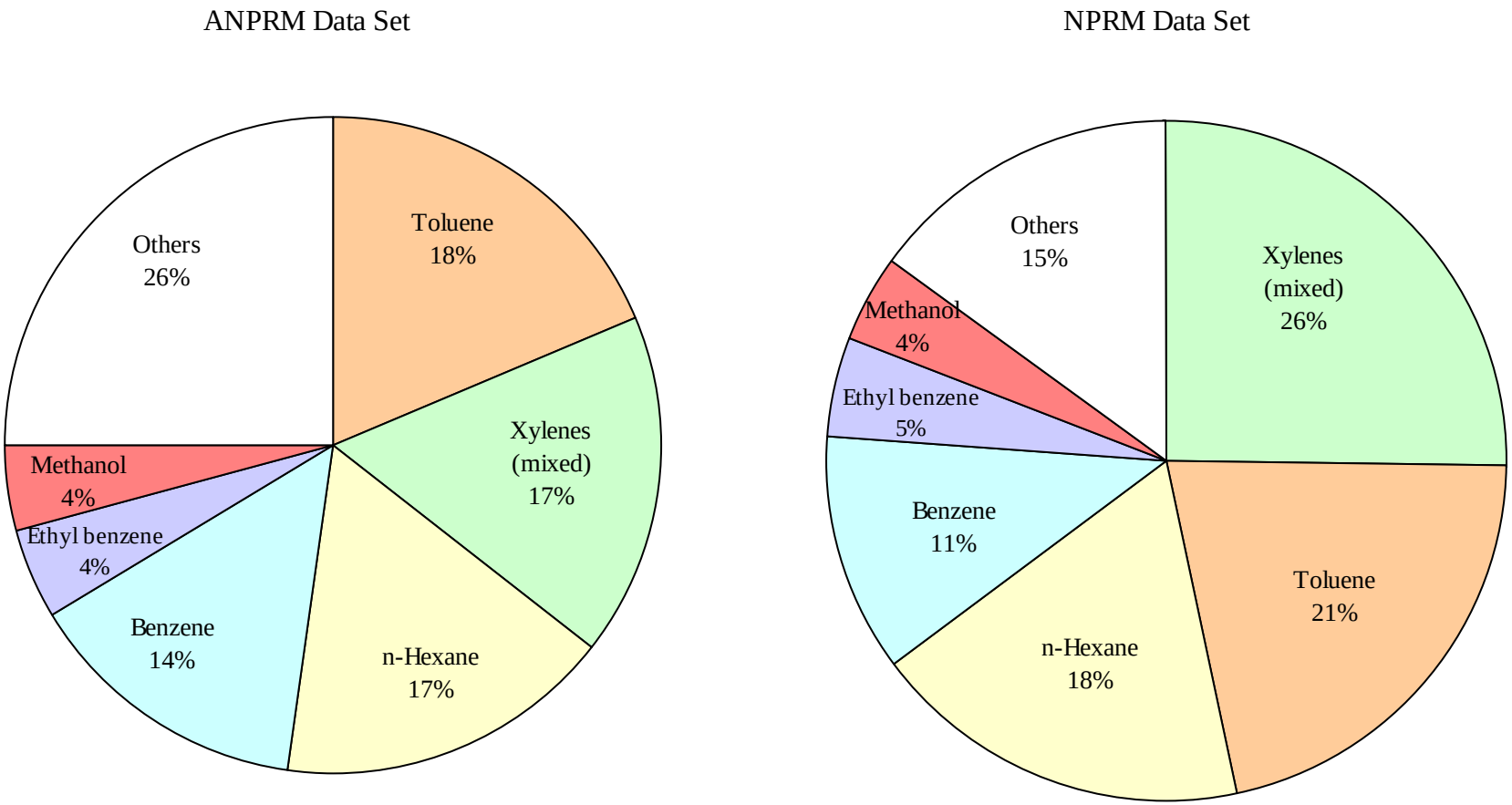


A.3 Discussion of Emissions Changes and Toxicity-Weighted Emissions Changes

As mentioned above, changes to the emissions estimates were the predominant type of change received through the ANPRM process for petroleum refineries. Emission changes included adding, deleting, or revising the emissions estimates for a specific point. Process changes could also have affected the emissions estimates for a facility. For example, if a process change removed an entire process from the data set, this would reduce the total emissions estimates for that facility. The total HAP emissions included in the ANPRM data set equal 2,316 tons per year (tpy), and the total HAP emissions included in the NPRM data set equal 2,292 tpy. Therefore, there was a reduction in overall HAP emissions of 24 tpy.

A summary of emissions by HAP for the ANPRM and NPRM data sets for petroleum refineries is presented in Figure 2. As can be seen in Figure 2, emissions of toluene (18 percent), xylenes (17 percent), hexane (17 percent), and benzene (14 percent) made up 66 percent of the emissions in the ANPRM data set. In the NPRM data set, the same four HAP make up 76 percent of the total emissions in the data set and have different individual percentages: xylenes (26 percent), toluene (21 percent), hexane (18 percent), and benzene (11 percent). However, while the overall percentages of these four HAP increased, the mass of emissions of each of these HAP decreased in the NPRM data set compared to the ANPRM data set.

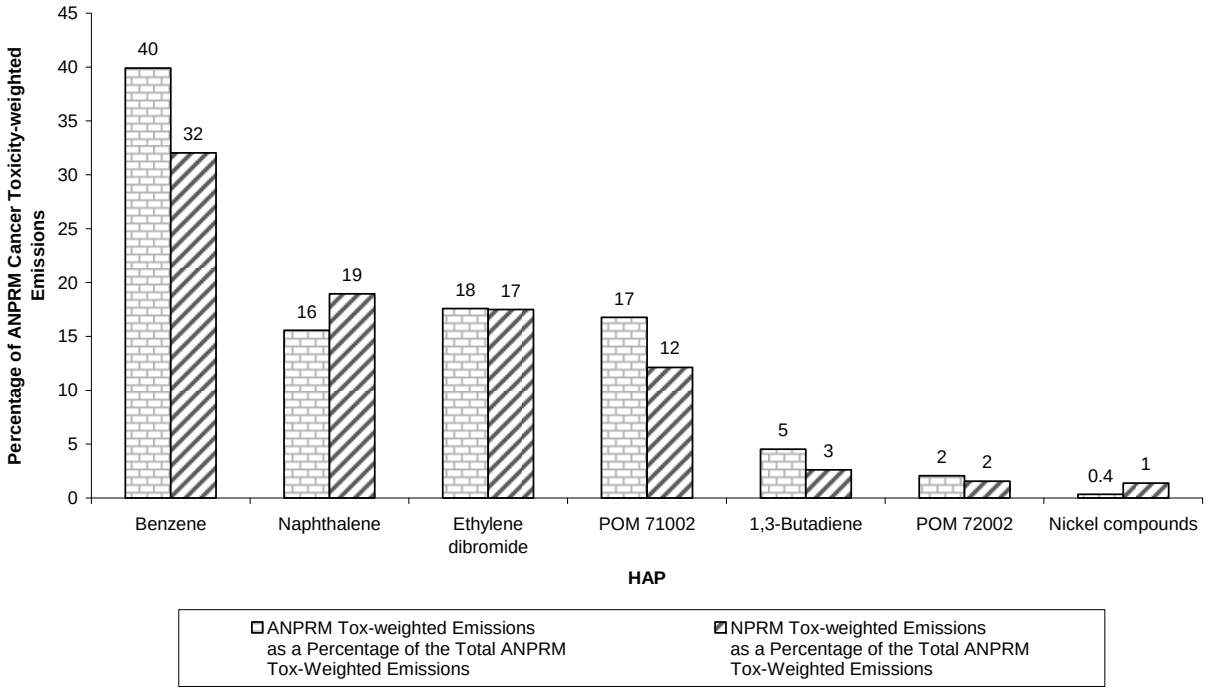
Figure 2. Percentage of Emissions by HAP for the Petroleum Refinery ANPRM and NPRM Data Sets



Emissions changes between the ANPRM and NPRM data sets were also evaluated by comparing the change in cancer toxicity-weighted emissions. Toxicity-weighted emissions take into account both the mass of the HAP emitted and its carcinogenic potency. The total emitted mass for each HAP was multiplied by its respective cancer unit risk estimate (URE) value in order to calculate cancer toxicity-weighted emissions. Overall, total toxicity-weighted emissions decreased by 12 percent from the ANPRM data set to the NPRM data set. The toxicity-weighted emissions of individual HAP generally decreased from the ANPRM data set to the NPRM data set, however they did increase for several HAP, including naphthalene, nickel compounds, and chromium compounds. In the ANPRM data set, 97 percent of the toxicity-weighted emissions can be accounted for by the following HAP: benzene, naphthalene, ethylene dibromide, POM71002, 1,3-butadiene, and POM72002. (As described in the approach for the 1999 NATA analysis, the name "POM71002" is used to represent the following compounds: 7-PAH, total PAH, polycyclic organic matter, 16-PAH, and 16 PAH-7 PAH. The name "POM72002" represents numerous compounds, including: anthracene, pyrene, benzo(g,h,i)perylene, perylene, fluoranthene, benzofluoranthenes, acenaphthene, phenanthrene, and fluorene.) These same six HAPs, plus nickel compounds and chromium compounds, account for almost 99 percent of the toxicity-weighted emissions in the NPRM data set. With the exception of benzene, none of these HAPs are those with the highest magnitude of emissions, as shown in Figure 2.

To illustrate the change in toxicity-weighted emissions between the ANPRM and NPRM data sets, we calculated the relative percentage that each HAP in the ANPRM and NPRM data sets contributed to the total ANPRM toxicity-weighted emissions. This comparison is presented in Figure 3, for those HAPs that contribute 1 percent or greater of the total toxicity-weighted ANPRM emissions. As shown in Figure 3, the toxicity-weighted ANPRM benzene emissions account for approximately 40 percent of the total ANPRM toxicity-weighted emissions, while the toxicity-weighted NPRM benzene emissions would relatively account for approximately 32 percent of the total ANPRM toxicity-weighted emissions. This is associated with a decrease of 64 tpy from the ANPRM to NPRM data set. Nickel compounds, on the other hand, saw a 0.4 tpy increase in emissions from the ANPRM to the NPRM data set. As shown in Figure 3, the toxicity-weighted ANPRM nickel compound emissions account for less than half a percent of the total ANPRM toxicity-weighted emissions, while the toxicity-weighted NPRM nickel compound emissions would relatively contribute slightly over 1 percent of the total ANPRM toxicity-weighted emissions.

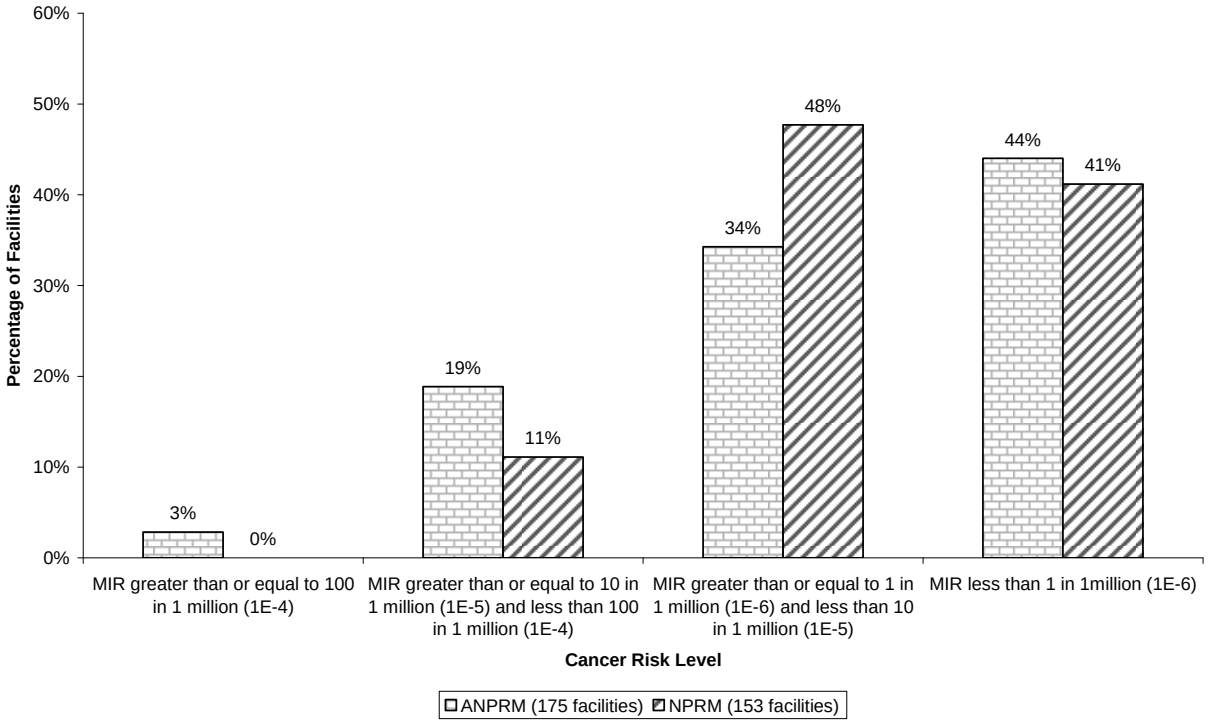
Figure 3. Cancer Toxicity-weighted Emissions for the Petroleum Refinery ANPRM and NPRM Source Category Data Sets



A.3.1 Comparison of ANPRM and NPRM Maximum Individual Cancer Risks

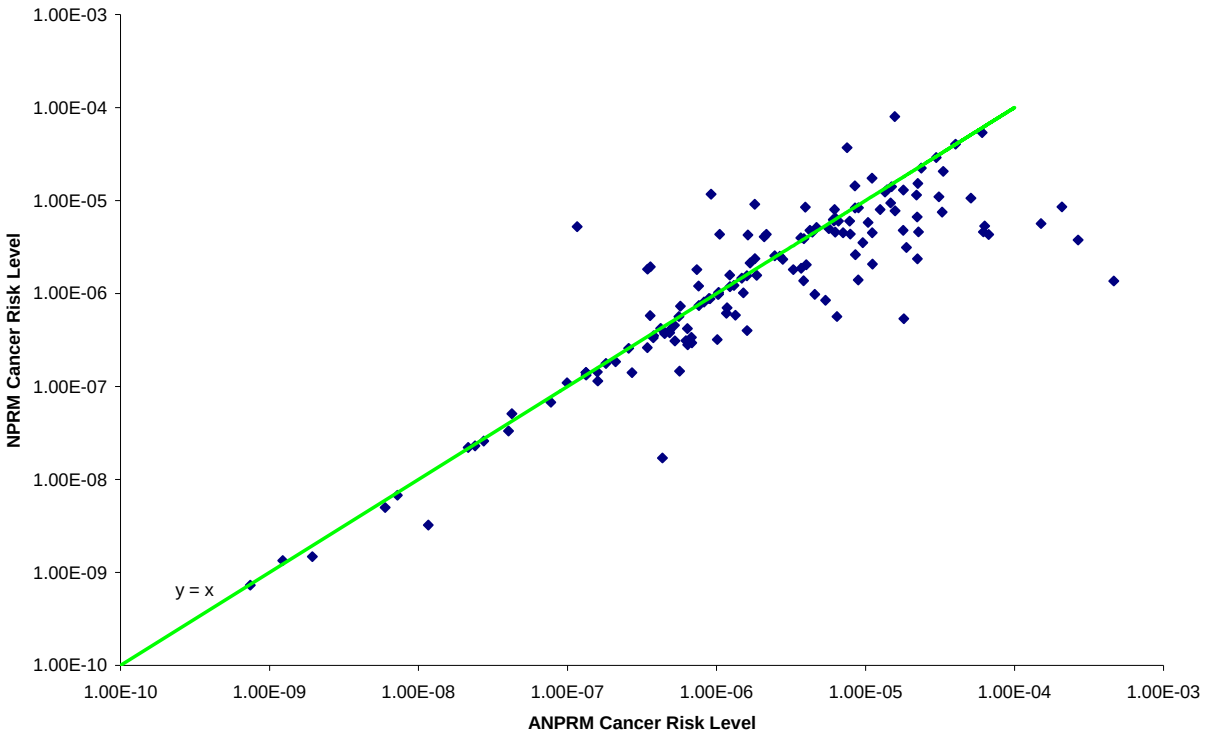
Figure 4 shows the comparison between the facility-level maximum individual cancer risk results for the ANPRM and NPRM by providing the percentage of facilities at each cancer risk level. As shown in Figure 4, there are no facilities in the NPRM data set with a cancer risk greater than or equal to 100 in 1 million but three percent (5 facilities) of the facilities in the ANPRM data set have a cancer risk greater than 100 in 1 million. There are also more facilities with a maximum individual cancer risk estimate greater than 10 in 1 million but less than 100 in 1 million in the ANPRM data set (19 percent or 33 facilities), compared to the NPRM data set (11 percent or 18 facilities).

Figure 4. Percentage of Facilities per Cancer Risk Level Petroleum Refinery ANPRM vs. NPRM Data Sets



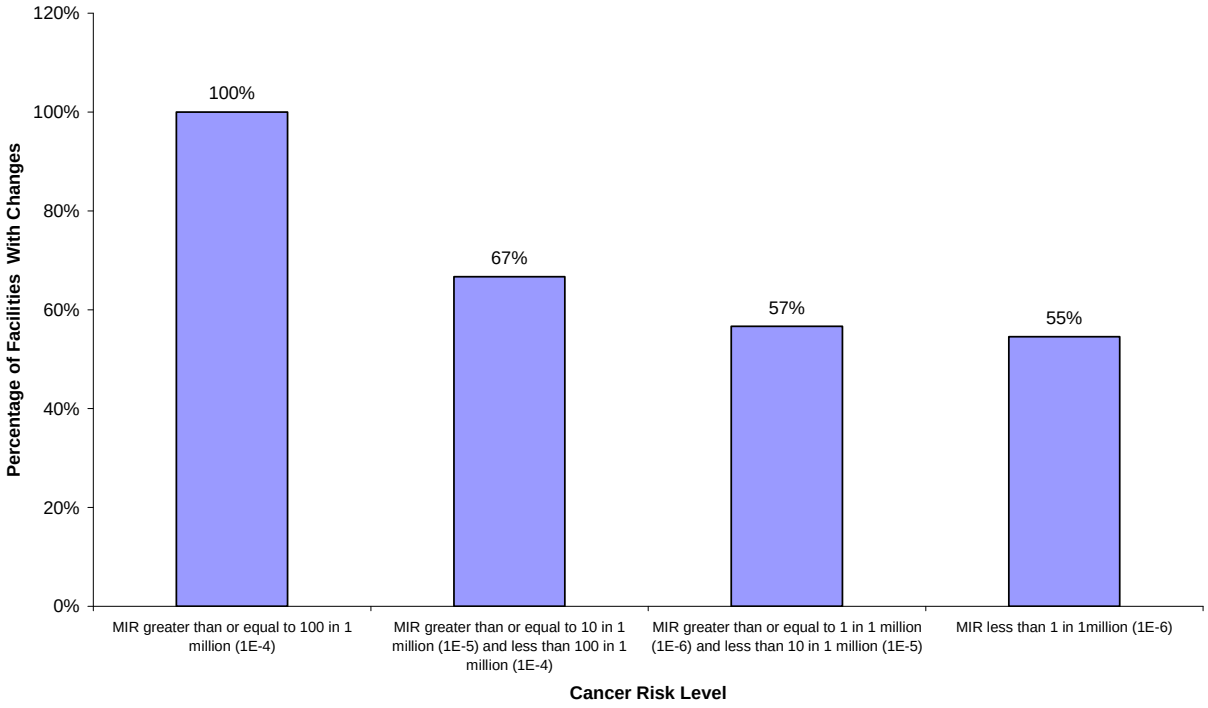
Another comparison between the risk results from the ANPRM and NPRM data sets is provided in Figure 5. In Figure 5, the ANPRM and NPRM maximum individual lifetime cancer risk for each facility is plotted as a point and shown compared to the y = x line. The points below the line indicate that the ANPRM risk was higher than the NPRM risk. The basic trend, shown in all the figures, is that facilities generally have lower cancer risks after the ANPRM than before.

Figure 5. NPRM Cancer Risk vs. ANPRM Cancer Risk for Petroleum Refinery Data Sets



In general, facilities with a higher maximum individual cancer risk in the ANPRM were more likely to provide data changes. This trend is illustrated in Figure 6. Data changes were provided for 100 percent of the 5 facilities with a maximum individual cancer risk greater than or equal to 100 in 1 million, and for 67 percent of the facilities with a cancer risk greater than or equal to 10 in 1 million but less than 100 in 1 million. In comparison, changes were received for only 55 percent of the facilities with a cancer risk less than 1 in 1 million.

Figure 6. Percentage of Facilities (per Cancer Risk Level) With Changes to Petroleum Refinery ANPRM Data Set



After changes were made for 100 percent of the facilities with a cancer risk level greater than or equal to 100 in 1 million, no facilities had risks at this level in the NPRM. However, while this trend does continue, it is less pronounced at lower risk levels. As shown in Figure 6, while 67 percent of the facilities with an ANPRM cancer risk greater than or equal to 10 in 1 million but less than 100 in 1 million provided data changes, there was only an 8 percent decrease in the facilities at this risk level. The magnitude of change between the ANPRM and NPRM cancer risk values are shown in Figure 7. (This figure only includes data for those facilities present in both the ANPRM and NPRM data sets.) As shown in Figure 7, the NPRM cancer risk decreased by over 50 percent from the ANPRM cancer risk for 41 percent of the facilities, while the NPRM cancer risk changed less than 1 percent for only 3 percent of the facilities. The same information is presented in Figure 8.

Figure 7. Risk Impacts for the Facilities with Changes to Petroleum Refinery ANPRM Data Set (not including those facilities that were completely added or removed)

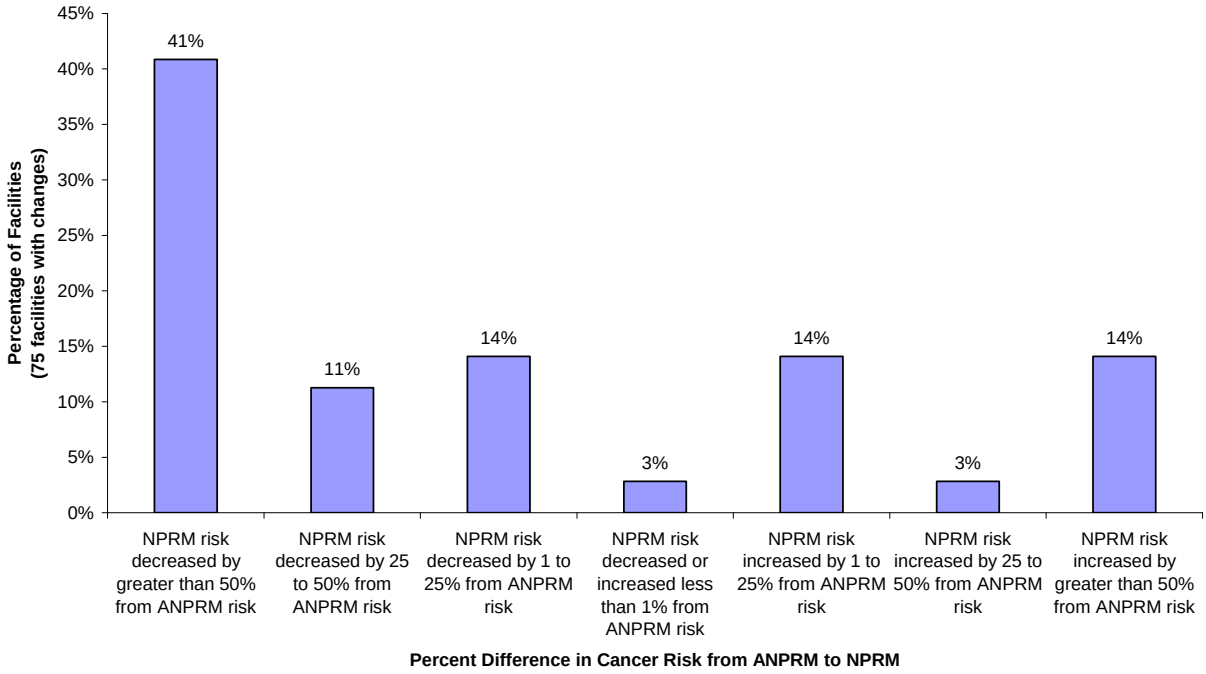
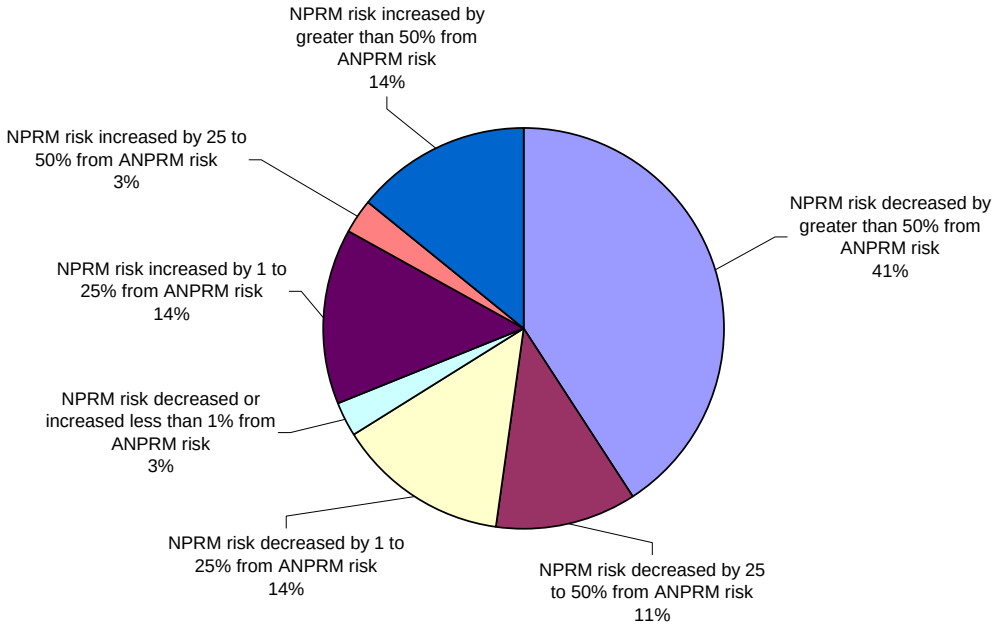


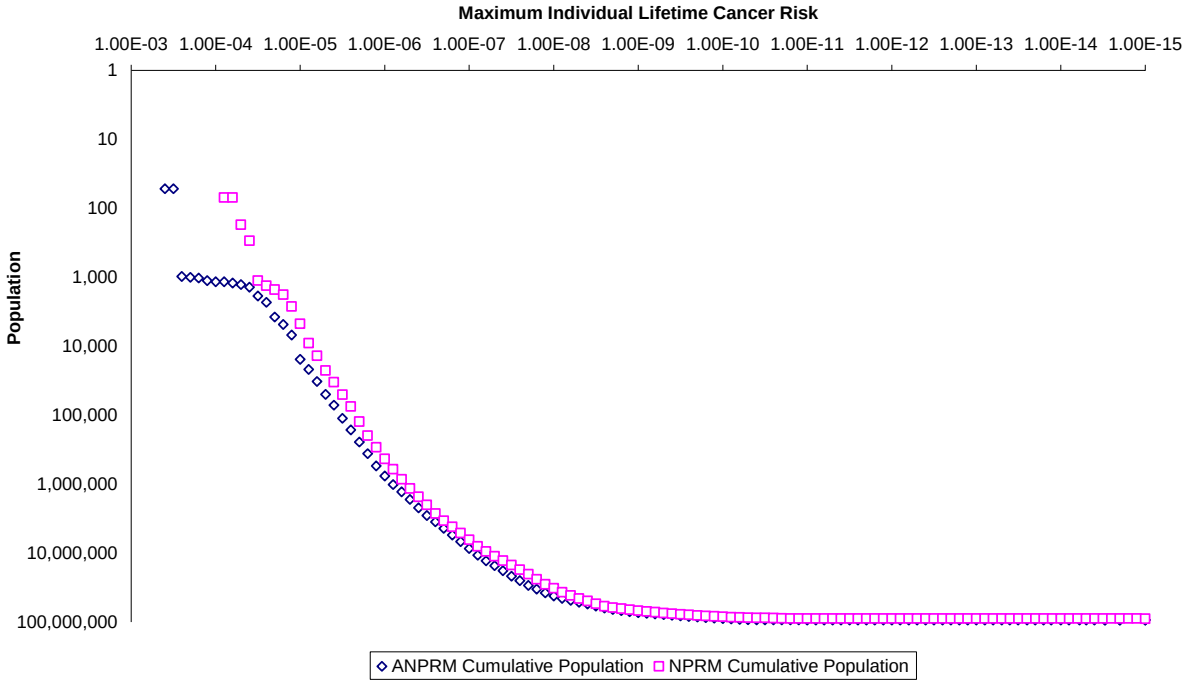
Figure 8. Risk Impacts for the Facilities with Changes to Petroleum Refinery ANPRM Data Set (not including those facilities that were completely added or removed)



A.3.2 Comparison of ANPRM and NPRM Cancer Incidence Values and Population Exposure

The estimated cancer incidence value resulting from modeling the ANPRM data set is 0.08. The estimated cancer incidence value resulting from modeling the NPRM data set is 0.05. The incidence value is calculated for each Census block by multiplying the estimated maximum individual lifetime cancer risk for that block by the number of people in that block and then dividing by the estimated lifespan value of 70 years. The values for each Census block are then summed to create a category-level incidence value. Where multiple facilities impact more than one Census block, this is taken into account before the summation so that people are not counted more than once. The change in the maximum individual risk estimate at each Census block, and therefore also the change in population affected between the ANPRM and NPRM, can be seen in Figure 9. As shown in Figure 9, there are fewer people exposed to all risk levels using the NPRM data set.

Figure 9. Comparison of Cumulative Populations per Cancer Risk Level for the Petroleum Refinery ANPRM and NPRM Data Sets



Appendix B
Analysis of Data on Short-Term Emission Rates Relative to Long-Term Emission Rates

Ted Palma
Roy Smith
EPA/OAQPS/SBAG

B1. Introduction

B1.1. The problem

The process of listing hazardous air pollutants (HAPs) provided by the Clean Air Act (CAA, section 112(b)(2)) explicitly includes acute toxicity as a listing criterion. For this reason, in addition to chronic exposures, EPA considers acute exposures in risk-based decision-making for the HAP regulatory program. Estimating acute exposures via dispersion modeling requires input data on hourly meteorological conditions (available for most areas of the US) and short-term emission rates of individual facilities (almost universally absent from the National Emissions Inventory (NEI), the Toxic Release Inventory (TRI), and state emission databases).

Lacking short-term emission rates, we must estimate peak short-term rates based on annual average rates, which are available. For Risk and Technology Review (RTR) rulemakings, we have assumed that the 1-hour emission rate for each facility could exceed the annual average hourly emission rate by as much as tenfold, and further assumed that this tenfold emission spike could coincide with worst-case meteorological conditions and the presence of a human receptor at the facility boundary, as a means of screening for potentially significant acute exposures.

In a consultation on the "RTR Assessment Plan", a panel of the EPA's Science Advisory Board (SAB), several reviewers questioned the appropriateness of the factor of ten; some even suggested that this tenfold assumption may underestimate actual maximum short-term emissions for some facilities, and thereby also underestimate maximum acute risks. The SAB recommended an analysis of available short-term emissions data for HAP to test this assumption. This analysis responds to that SAB recommendation and attempts to evaluate the protectiveness of the tenfold assumption using a database of "event emissions" collected from facilities in the Houston-Galveston area, to compare events representative of short-term HAP releases of specific events to long-term release rates for the entire facility. This evaluation is intended to estimate how many short-term events might have achieved a release rate that exceeded the routine emission rate for the entire facility during the ca. 2-year data collection period.

B2. Methods

B2.1. Texas Commission on Environmental Quality event emissions database

The Texas Commission on Environmental Quality (TCEQ) collects emissions data using online reporting required of any facility whenever it experienced a non-routine event that released 100 pounds or more of a listed chemical (primarily ozone-forming VOCs). The TCEQ data are intended to improve the state's knowledge of how short-term releases affect tropospheric ozone levels in that area. The database we utilized in our analysis was a subset of the TCEQ data covering emission events that occurred in an eight-county area in eastern Texas during a 756-day period between January 31, 2003 and February 25, 2005.

The complete emissions event data were obtained in April 2007 from Cynthia Folsom Murphy, a research scientist with the University of Texas at Austin (UTA) Center for Energy and

Environmental Resources. The data were provided in four Excel spreadsheets generated from an original MS Access file. We used these Excel files to reconstruct a MS Access database in order to facilitate selection of a representative subset of records for this analysis.

Although some of the released substances were HAPs, this was incidental to the database's primary purpose of enhancing the TCEQ's knowledge of photochemical activity. Thus, more than 80% of the released mass was ethene and propene, neither of which are HAPs. The database included release events caused by accidents, equipment failures, startup, shutdown, and malfunction. It also contained facility names, information on amounts of individual compounds released. To provide a basis for comparing the event releases with "typical" emissions, the UTA staff included total VOC emissions data for each facility for calendar year 2004, obtained from the EPA Toxic Release Inventory (TRI). The database did *not* contain any records for facilities that did not experience any reportable events during this period.

B2.2. Data filtering

Because the event release data were intended for modeling short-term releases of ozone-producing VOCs, the database includes releases from accidents (which are regulated under section 112(r) of the CAA and are therefore not considered in residual risk assessments) and releases of light hydrocarbon compounds that are not HAPs and are much more volatile than most HAPs. This intent of our analysis, on the other hand, was to evaluate short-term releases of HAPs due to normal process variability or scheduled startups, shutdowns, and malfunctions, relative to long-term release rates. Because the full emission events database was not representative of likely HAP emissions normally considered under the residual risk program, we filtered the release data as follows in an attempt to improve its representativeness:

1. Hydrocarbons of C5 or less were dropped, except that all HAPs (including non-VOCs) were retained regardless of volatility;

2. Releases labeled as accidental by the database were dropped, but all others (including those labeled startup, shutdown, and malfunction) were retained;

3. Only facilities whose long-term VOC releases exceeded 0.068 tons per day (25 tons per year) were retained, to approximate the population of facilities likely to be subject to residual risk standards (i.e., major facilities);

4. A few release records had to be dropped because their facility numbers did not link to any facility in the database;

5. A few facilities had to be dropped because the database did not include their 2004 TRI VOC release information.

B2.3. Analysis

Annual VOC emissions for each facility in its entirety and release data for each event were both converted to lb/hr. In order to conform to our atmospheric dispersion models, which estimate ambient concentrations for periods of 1 hour or more, amounts released during events shorter than 1 hour were assigned to the whole hour. For example, a release of 100 lb in ten minutes was converted to 100 lb/hr. Events longer than 1 hour were converted normally, e.g., a release of

100 lb in 120 minutes was converted to 50 lb/hr. The event release rates for individual compounds were summed, yielding a total release rate for each event. This total release rate for each event was divided by the annual VOC release rate for the entire facility to derive the ratio of peak-to-mean emission rate for the event.

B3. Results and Discussion

B3.1. Database filtering

The original database contained 505 individual contaminants, including multiple redundancies. These redundancies did not affect this analysis, so we did not resolve them. After filtering out light, non-HAP VOCs, 317 contaminants remained (Table 1).

The database contained release records for 150 unique facilities. Of these, 48 facilities (Table 2) were major VOC emitters that reported releases of at least one of the contaminants in Table 1.

The database contained 3641 individual release events reported by the original 150 facilities. Of these, 319 events involved a Table 1 contaminant released by a Table 2 facility during startup, shutdown, or malfunction. For evaluating short-term releases for residual risk assessments, these 319 events comprise the most representative subset of the full database.

B3.2. Descriptive statistics

For this subset of emission events, ratios of event release rate to long-term whole-facility release rate varied from 0.00000004 to 74. Distribution statistics appear in Tables 3 and 4. The 99th percentile ratio was 9 (i.e., an event release rate nine times the long-term average). Only 3 ratios exceeded our default assumption of 10, and of these only one exceeded 11. The full cumulative probability density of the ratios is shown in Figure 1.

Figure 2 shows the relationship between ratio and event duration. As expected, the ratio declined as duration increased. Only 18 events lasted less than 2 hours, but these events produced the three highest ratios. Figure 3 is a similar ratio vs. duration plot, but with duration as a percentage of total time. Only 35 events exceeded 1% of the total period covered by the database. Figure 4 shows the relationship between ratio and total amount released, and suggests that the highest ratios were produced by facilities whose routine VOC emissions were relatively small. Thus, the events themselves also tended to be relatively small in absolute terms.

B3.3. Discussion

These results suggest that the tenfold ratio assumption for short-term releases is protective, and that the facilities for which it may underestimate event releases may tend to be smaller emitters.

However, this analysis is limited in the following ways by the nature of the database and the filtering that we applied:

1. The only long-term release data available for VOCs from the database were total emissions for 2004, and the only short-term release data were emissions of the individual substances that triggered the data entry. Ideally, we would have preferred to have routine release rates for each individual compound, or at least event release rates for total VOCs.

However, retrieving these data from other sources and linking them to this database was not feasible.

2. Removing VOCs that are not representative of HAPs, and comparing the releases against all VOCs, would tend to underestimate the true ratios. This effect could be quantitatively large.

3. Retaining HAPs that are not VOCs and including them in the total to be compared against all VOCs, would tend to overestimate the true ratios. The size of this effect is not known, but seems likely to be less than for (2) above.

4. The database contains only facilities that had at least one release event during the reporting period. The number of facilities in the statistical population that did not experience an event is not known. The lack of data for these facilities (whose ratios in this analysis would have been zero) would cause the descriptive statistics to be skewed toward an overestimate. The size of this effect is unknown.

Table 1. Event emissions in the Houston-Galveston area. Representative contaminants included in the analysis, selected because they are either HAPs or VOCs with more than 5 carbon atoms. (These data were retrieved directly from the original database, which included multiple redundancies that did not affect the analysis and were left intact.)

Contaminant	*HAP*	*CAS*	*SAROAD*
2-Methyloctane	No	3221-61-2	90008
2-Methylpentane	No	107-83-5	43229
2-methylhexane	No	591-76-4	43263
2-Methylpentane	No	107-83-5	43229
2,2,3-Trimethylpentane	No	564-02-3	
2,2,4-Trimethylpentane	Yes	540-84-1	43250
dimethyl butane	No	75-83-2	43291
2,3-Dimethylbutane	No	79-29-8	43276
2,3,4-Trimethylpentane	No	565-75-3	43252
2,3-Dimethylbutane	No	79-29-8	43276
2,4-Dimethylpentane	No	108-08-7	43247
2-methylheptane	No	592-27-8	43296
2-methylhexane	No	591-76-4	43263
2-Methylpentane	No	107-83-5	43229
3-Methylhexane	No	589-34-4	43295
3-Methylpentane	No	96-14-0	43230
3-Methylhexane	No	589-34-4	43295
3-Methylpentane	No	96-14-0	43230
3-Methylheptane	No	589-81-1	43253
3-Methylhexane	No	589-34-4	43295
3-Methylpentane	No	96-14-0	43230
Acetaldehyde	Yes	75-07-0	43503
Acetic Acid	No	64-19-7	43404
Acetonitrile	Yes	75-05-8	70016

Table 1. Event emissions in the Houston-Galveston area. Representative contaminants included in the analysis, selected because they are either HAPs or VOCs with more than 5 carbon atoms. (These data were retrieved directly from the original database, which included multiple redundancies that did not affect the analysis and were left intact.)

Contaminant	*HAP*	*CAS*	*SAROAD*
Acetophenone	Yes	98-86-2	
Acrolein	Yes	107-02-8	43505
Acrylic acid	Yes	79-10-7	43407
Acrylonitrile	Yes	107-13-1	43704
alkylphenol	No	none	
Benzene	Yes	71-43-2	45201
Benzo[a]anthracene	Yes	56-55-3	46716
Benzo[a]pyrene	Yes	50-32-8	46719
Benzo[b]fluoranthene	Yes	205-99-2	46717
Biphenyl	Yes	92-52-4	45226
Butanol	No	35296-72-1	
Butyl Acrylate	No	141-32-2	43440
t-Butyl Alcohol	No	75-65-0	43309
butylcyclohexane	No	1678-93-9	90101
Butyraldehyde	No	123-72-8	43510
C9 Aromatics	No	none	
Naphthalene	Yes	91-20-3	46701
Nonane	No	111-84-2	43235
C9+	No	none	
Carbon tetrachloride	Yes	56-23-5	43804
Carbonyl Sulfide	Yes	463-58-1	43933
Chloral	No	75-87-6	
Trichloromethane	Yes	67-66-3	43803
Chlorothalonil	No	1897-45-6	
Petroleum	No	8002-05-9	
Petroleum	No	8002-05-9	
Cumene	Yes	98-82-8	45210
Cyclohexane	No	110-82-7	43248
Cyclohexanol	No	108-93-0	43317
Cyclohexanone	No	108-94-1	43561
Cyclohexanone	No	108-94-1	43561
Decane	No	124-18-5	43238
Decane	No	124-18-5	43238
1,2-Dichloroethane	No	107-06-2	43815
Diethylbenzene (mixture)	No	25340-17-4	45106
Methyl Ether	No	115-10-6	43350
Dimethylcyclohexane	No	27195-67-1	98059
Dimethylcyclopentane	No	28729-52-4	90064
Dimethylcyclopentane	No	28729-52-4	90064
Dimethyl formamide	Yes	68-12-2	43450
Dimethylhexane	No	28777-67-5	90067

Table 1. Event emissions in the Houston-Galveston area. Representative contaminants included in the analysis, selected because they are either HAPs or VOCs with more than 5 carbon atoms. (These data were retrieved directly from the original database, which included multiple redundancies that did not affect the analysis and were left intact.)

Contaminant	*HAP*	*CAS*	*SAROAD*
Dimethyl pentane	No	38815-29-1	90063
Epichlorohydrin	Yes	106-89-8	43863
Ethyl Alcohol	No	64-17-5	43302
Ethyl Acrylate	Yes	140-88-5	43438
Ethyl Alcohol	No	64-17-5	43302
Ethyl Benzene	Yes	100-41-4	45203
Ethyl Chloride	Yes	75-00-3	43812
Ethylcyclohexane	No	1678-91-7	43288
ethylacetylene	No	107-00-6	43281
Ethyl Benzene	Yes	100-41-4	45203
Ethylene Oxide	Yes	75-21-8	43601
ethylmethylbenzene	No	25550-14-5	45104
formaldehyde	Yes	50-00-0	43502
Furfural	No	98-01-1	45503
straight-run middle distillate	No	64741-44-2	
Gasoline	No	86290-81-5	
Gasoline	No	86290-81-5	
Heavy Olefins	No	none	
n-Heptane	No	142-82-5	43232
n-Heptane	No	142-82-5	43232
Heptylene	No	25339-56-4	
hexane	Yes	110-54-3	43231
hexane	Yes	110-54-3	43231
2-Methylpentane	No	107-83-5	43229
hexane	Yes	110-54-3	43231
Hexene	No	25264-93-1	43289
Indeno[1,2,3-cd]pyrene	Yes	193-39-5	46720
Isobutyraldehyde	No	78-84-2	43511
2-Methyl-1-propanol	No	78-83-1	43306
2-Methyl-1-propanol	No	78-83-1	43306
Isobutyraldehyde	No	78-84-2	43511
Isoheptanes (mixture)	No	31394-54-4	43106
2-Methylpentane	No	107-83-5	43229
2,2,4-Trimethylpentane	No	540-84-1	43250
2,2,4-Trimethylpentane	No	540-84-1	43250
Isopar E	No		
Isoprene	No	78-79-5	43243
2-Propanol	No	67-63-0	43304
2-Propanol	No	67-63-0	43304
Cumene	Yes	98-82-8	45210
Isopropylcyclohexane	No	696-29-7	90128

Table 1. Event emissions in the Houston-Galveston area. Representative contaminants included in the analysis, selected because they are either HAPs or VOCs with more than 5 carbon atoms. (These data were retrieved directly from the original database, which included multiple redundancies that did not affect the analysis and were left intact.)

Contaminant	*HAP*	*CAS*	*SAROAD*
Diisopropyl ether	No	108-20-3	85005
Kerosene	No	64742-81-0	
Methyl ethyl ketone	No	78-93-3	43552
Methyl isobutenyl ketone	Yes	141-79-7	
Methanol	Yes	67-56-1	43301
Methyl Acetylene	No	74-99-7	43209
Cresol	Yes	1319-77-3	45605
Methyl Chloride	Yes	74-87-3	43801
methyl cyclohexane	No	108-87-2	43261
Methyl ethyl ketone	No	78-93-3	43552
Iodomethane	No	74-88-4	86025
Methyl Mercaptan	No	74-93-1	43901
methyl cyclohexane	No	108-87-2	43261
Methylcyclopentane	No	96-37-7	43262
2-Methyldecane	No	6975-98-0	98155
Methylheptane	No	50985-84-7	90045
2-methylheptane	No	592-27-8	43296
2-Methyl nonane	No	871-83-0	90047
Tert-butyl methyl ether	No	1634-04-4	43376
meta-xylene	No	108-38-3	45205
Nonane	No	111-84-2	43235
Naphtha	No	8030-30-6	45101
Naphthalene	Yes	91-20-3	46701
Naphtha	No	8030-30-6	45101
Naphthalene	No	91-20-3	46701
Butyl acetate	No	123-86-4	43435
Butyraldehyde	No	123-72-8	43510
Nonane	No	111-84-2	43235
Nonane	No	111-84-2	43235
Octadecene	No	27070-58-2	
n-Octane	No	111-65-9	43233
Octene (mixed isomers)	No	25377-83-7	
ortho-xylene	No	95-47-6	45204
Parathion	Yes	56-38-2	
4-Aminohippuric Acid	No	61-78-9	
Phenol	Yes	108-95-2	45300
Silicone	No	63148-62-9	
Naphtha	No	8030-30-6	45101
Naphtha	No	8030-30-6	45101
Polyethylene	No	9002-88-4	
Poly(Isobutylene)	No	9003-27-4	

Table 1. Event emissions in the Houston-Galveston area. Representative contaminants included in the analysis, selected because they are either HAPs or VOCs with more than 5 carbon atoms. (These data were retrieved directly from the original database, which included multiple redundancies that did not affect the analysis and were left intact.)

Contaminant	*HAP*	*CAS*	*SAROAD*
Chloromethyl pivalate	No	18997-19-8	
Process fuel gas	No	none	
Propionic Acid	No	79-09-4	43405
Propylene oxide	No	75-56-9	43602
para-xylene	No	106-42-3	45206
Styrene	Yes	100-42-5	45220
Sulfolane	No	126-33-0	
t-Butyl Alcohol	No	75-65-0	43309
t-Butyl Alcohol	No	75-65-0	43309
tert-butyl hydroperoxide	No	75-91-2	
Toluene	Yes	108-88-3	45202
Aqualyte(TM), LSC cocktail	No	25551-13-7	45107
1,3,4-Trimethylbenzene	No	95-63-6	45208
trimethylcyclopentane	No	30498-64-7	98058
trimethylpentane	No	29222-48-8	90092
Undecane	No	1120-21-4	43241
Vinyl acetate	Yes	108-05-4	43453
Vinyl acetate	Yes	108-05-4	43453
Vinyl chloride	Yes	75-01-4	43860
vinyl resin	No	none	
Vinylcyclohexane	No	695-12-5	
xylenes	Yes	1330-20-7	45102
xylenes	Yes	1330-20-7	45102
meta-xylene	Yes	108-38-3	45205
ortho-xylene	Yes	95-47-6	45204
para-xylene	Yes	106-42-3	45206
Mineral spirits	No	64475-85-0	43118
Propylene glycol	No	57-55-6	43369
Vinyl chloride	Yes	75-01-4	43860
1-Decene	No	872-05-9	90014
2-Ethyl-1-hexanol	No	104-76-7	43318
2-Pyrrolidone	No	616-45-5	
Aromatic	No	none	
Decene	No	25339-53-1	90014
2-N,N-Dibutylaminoethanol	No	102-81-8	86007
Diisopropanolamine	No	110-97-4	86004
N,N-Dimethylethanolamine	No	108-01-0	84004
trifluoroethane	No	27987-06-0	
2,2'-Oxybisethanol	No	111-46-6	43367
Hydrocarbons	No	none	
Methyl Formate	No	107-31-3	43430

Table 1. Event emissions in the Houston-Galveston area. Representative contaminants included in the analysis, selected because they are either HAPs or VOCs with more than 5 carbon atoms. (These data were retrieved directly from the original database, which included multiple redundancies that did not affect the analysis and were left intact.)

Contaminant	*HAP*	*CAS*	*SAROAD*
Isopropylamine	No	75-31-0	86014
n-Butanol	No	71-36-3	43305
Polypropylene glycol ether	No		
N-Vinyl-2-Pyrrolidinone	No	88-12-0	
1,1-Di(t-Amylperoxy) Cyclohexane	No	15667-10-4	
1,2,3-Trimethyl-4-ethylbenzene	No	none	
2-Methyldecane	No	6975-98-0	98155
2-methylheptane	No	592-27-8	43296
2-Methyl nonane	No	871-83-0	90047
2,5-Dimethylhexane-2,5-dihydroperoxide	No	3025-88-5	
Butyl ether	No	142-96-1	43372
1,2-Dichloroethane	Yes	107-06-2	43815
Hydrindene	No	496-11-7	98044
Methylheptane	No	50985-84-7	90045
methyl methacrylate	No	80-62-6	43441
Naphtha	No	8030-30-6	45101
hexane	Yes	110-54-3	43231
tert-amyl hydroperoxide	No	3425-61-4	
1,3,4-Trimethylbenzene	No	95-63-6	45208
n-Butanol	No	71-36-3	43305
2-Butoxy ethanol	Yes	111-76-2	43308
hexane	Yes	110-54-3	43231
cycloheptane	No	291-64-5	43115
n-Heptane	No	142-82-5	43232
n-Octane	No	111-65-9	43233
Hexyl Carbitol	No	112-59-4	
Nonene	No	27215-95-8	
Silane, ethenyltrimethoxy	No	2768-02-7	
tetrahydrofuran	No	109-99-9	70014
Vinyl chloride	Yes	75-01-4	43860
Methyl Formate	No	107-31-3	43430
Phenyl ether	No	101-84-8	
phosgene	Yes	75-44-5	
1,2-Dichloroethane	No	107-06-2	43815
2-Butoxy ethanol	Yes	111-76-2	43308
Gasoline	No	86290-81-5	
1-Tridecanol	No	112-70-9	
1,2,4-Trichlorobenzene	Yes	120-82-1	45208
2-(2-Butoxyethoxy)ethanol	Yes	112-34-5	43312
2,3,4-trihydroxybenzophenone	No	1143-72-2	

Table 1. Event emissions in the Houston-Galveston area. Representative contaminants included in the analysis, selected because they are either HAPs or VOCs with more than 5 carbon atoms. (These data were retrieved directly from the original database, which included multiple redundancies that did not affect the analysis and were left intact.)

Contaminant	*HAP*	*CAS*	*SAROAD*
Ester			
Methyl n-amyl ketone	No	110-43-0	43562
4,4-Cyclohexylidenebis[phenol]	No	843-55-0	
Anisole	No	100-66-3	
2-Butoxy ethanol	Yes	111-76-2	43308
Cresol-Formaldehyde novolac Resin	No	proprietary	
Decane	No	124-18-5	43238
gamma-Butyrolactone	No	96-48-0	
Dimethyl pentane	No	38815-29-1	90063
Dodecyl Benzenesulfonic Acid	No	27176-87-0	
Ethanol Amine	No	141-43-5	43777
ethyl lactate	No	687-47-8	
Hexamethyldisilazane	No	999-97-3	
Methyl ethyl ketone	No	78-93-3	43552
Cresol	Yes	1319-77-3	45605
Naphthalene Sulfonic Acid Resin	No		
Naphthalene Sulfonic Acid Resin	No		
n-Butanol	No	71-36-3	43305
Decane	No	124-18-5	43238
1-Methyl-2-pyrrolidinone	No	872-50-4	70008
Pentyl Ester Acetic Acid	No		
Phenol Formaldehyde Resin, Novolac	No		
Phenol Formaldehyde Resin, Novolac	No		
Propylene Glycol Monomethyl Ether	No	107-98-2	70011
Pyrocatechol	No	120-80-9	
Carbon Disulfide	Yes	75-15-0	43934
Hexene	No	592-41-6	43245
VOC	No	none	
Methacrylic acid	No	79-41-4	84009
Methyl 3-hydroxybutyrate	No	1487-49-6	
t-Butyl Alcohol	No	75-65-0	43309
methyl valeraldehyde	No	123-15-9	
Butyl Methacrylate	No	97-88-1	85008
dipropyl ether	No	111-43-3	
n-Propanol	No	71-23-8	43303
Propyl propionate	No	106-36-5	86052
1,2-Epoxybutane	Yes	106-88-7	
Methylamine	No	74-89-5	

Table 1. Event emissions in the Houston-Galveston area. Representative contaminants included in the analysis, selected because they are either HAPs or VOCs with more than 5 carbon atoms. (These data were retrieved directly from the original database, which included multiple redundancies that did not affect the analysis and were left intact.)

Contaminant	*HAP*	*CAS*	*SAROAD*
1,1-Dimethylcyclohexane	No	590-66-9	
1,1-Dimethylcyclopentane	No	1638-26-2	
2-Methylpentane	No	107-83-5	43229
dimethyl butane	No	75-83-2	43291
2,3,3-Trimethylpentane	No	560-21-4	
2,3-Dimethylhexane	No	584-94-1	
2,3-Dimethylpentane	No	565-59-3	
2,4-Dimethylhexane	No	589-43-5	
2,5-Dimethyl-hexane	No	592-13-2	
2-Butoxy ethanol	Yes	111-76-2	43308
2-mercaptoethanol	No	60-24-2	
Bisphenol A	No	80-05-7	
straight-run middle distillate	No	64741-44-2	
4-Vinylcyclohexene	No	100-40-3	
straight-run middle distillate	No	64741-44-2	
Allyl alcohol	No	107-18-6	
xylenes	Yes	1330-20-7	45102
Naphthalene	Yes	91-20-3	46701
3-Methylethylcyclohexane	No		
VOC	No	none	
Gasoline	No	86290-81-5	
Butyl ether	No	142-96-1	
dimethyl butane	No	75-83-2	
Dodecene	No	25378-22-7	
Styrene	Yes	100-42-5	45220
tetrahydrofuran	No	109-99-9	70014
hexane	Yes	110-54-3	43231
2-Propanol	No	67-63-0	43304
liquified petroleum gas	No	68476-85-7	
Methyl acetylene propadiene	No		
methyl isobutyl ketone	Yes	108-10-1	
Methyl n-amyl ketone	No	110-43-0	43562
Methylpentane	No	43133-95-5	
Tert-butyl methyl ether	Yes	1634-04-4	43376
Toluene	Yes	108-88-3	45202
Mineral oil	No	8012-95-1	
Gasoline	No	86290-81-5	
2,2-Dimethylpropane	No	463-82-1	43222
n-propylbenzene	No	103-65-1	
propylcyclohexane	No	1678-92-8	
n-Octane	No	111-65-9	43233

Table 1. Event emissions in the Houston-Galveston area. Representative contaminants included in the analysis, selected because they are either HAPs or VOCs with more than 5 carbon atoms. (These data were retrieved directly from the original database, which included multiple redundancies that did not affect the analysis and were left intact.)

Contaminant	*HAP*	*CAS*	*SAROAD*
ortho-xylene	No	95-47-6	45204
Gasoline	No	86290-81-5	
propylenimine	No	75-55-8	
Gasoline	No	86290-81-5	
Technical White Oil	No		
Total Alkylate - non-speciated	No		
Trichloroethylene	Yes	79-01-6	
Di(2-ethylhexyl) peroxydicarbonate	No	16111-62-9	
trimethylcyclopentane	No	30498-64-7	98058
Ultraformate	No		
4-Vinylcyclohexene	No	100-40-3	

Table 2. Event emissions in the Houston-Galveston area. Major emitters reporting at least one release event of a representative substance.

Company Name	*2004 VOC Emission Rate (lb/h)*
ATOFINA PETROCHEMICALS LA PORTE PLANT	47.88
BALL METAL BEVERAGE CONTAINER CONROE FACILITY	24.18
BASF FREEPORT SITE	46.47
BELVIEU ENVIRONMENTAL FUELS	112.3
BOC GROUP CLEAR LAKE BOC GASES PLANT	9.52
BP AMOCO CHEMICAL CHOCOLATE BAYOU PLANT	130.4
BP AMOCO CHEMICAL PASADENA PLANT	36.92
BP AMOCO POLYMERS	57.18
BP PRODUCTS NORTH AMERICA TEXAS CITY	737.4
BP TEXAS CITY CHEMICAL PLANT B	112.2
CELANESE BAY CITY PLANT	17.12
CELANESE CLEAR LAKE PLANT	53.11
CELANESE PASADENA PLANT	5.934
CHEVRON PHILLIPS CEDAR BAYOU PLANT	105.3
CHEVRON PHILLIPS CHEMICAL SWEENY COMPLEX	106.7
CHEVRON PHILLIPS HOUSTON CHEMICAL COMPLEX	215.7
CROWN BEVERAGE PACKAGING	18.05
CROWN CENTRAL PETROLEUM PASADENA PLANT	114.3
CROWN CORK & SEAL	18.10
DEER PARK LIQUID STORAGE TERMINAL	124.8
DOW CHEMICAL LA PORTE SITE	5.902
DOW TEXAS OPERATIONS FREEPORT	203.2
E I DUPONT DE NEMOURS AND COMPANY - LA PORTE PLANT	51.30
EQUISTAR CHEMICALS CHANNELVIEW COMPLEX	275.4
EQUISTAR CHEMICALS CHOCOLATE BAYOU COMPLEX	84.87
EQUISTAR CHEMICALS LA PORTE COMPLEX	90.97
EXXON MOBIL CHEMICAL BAYTOWN OLEFINS PLANT	84.73
EXXONMOBIL CHEMICAL BAYTOWN CHEMICAL PLANT	313.7
EXXONMOBIL CHEMICAL MONT BELVIEU PLASTICS PLANT	40.64
GOODYEAR HOUSTON CHEMICAL PLANT	85.68
ISP TECHNOLOGIES TEXAS CITY PLANT	22.12
KANEKA TEXAS CORPORATION	20.55
KINDER MORGAN LIQUID TERMINALS PASADENA	913.9
KINDER MORGAN LIQUIDS TERMINALS	132.7
LBC HOUSTON BAYPORT TERMINAL	12.83
LYONDELL CHEMICAL BAYPORT PLANT	30.04
LYONDELL CHEMICAL CHANNELVIEW	74.15
MARATHON ASHLAND PETROLEUM TEXAS CITY REFINERY	111.8
MOBIL CHEMICAL HOUSTON OLEFINS PLANT	26.29
MORGANS POINT PLANT	31.03
PASADENA PLANT	13.40

Table 2. Event emissions in the Houston-Galveston area. Major emitters reporting at least one release event of a representative substance.

Company Name	*2004 VOC Emission Rate (lb/h)*
SHELL OIL DEER PARK	405.2
SOLUTIA CHOCOLATE BAYOU PLANT	53.09
STOLTHAVEN HOUSTON TERMINAL	7.347
SWEENY COMPLEX	157.1
UNION CARBIDE TEXAS CITY OPERATIONS	174.4
VALERO REFINING TEXAS CITY	260.1
WHARTON GAS PLANT	7.552

Table 3. Frequency distribution for ratio of event emission rate to long-term emission rate

Bin	*Frequency*	*Cumulative Frequency*
1.00E-08	0	0
3.16E-08	0	0
1.00E-07	2	2
3.16E-07	1	3
1.00E-06	0	3
3.16E-06	2	5
1.00E-05	1	6
3.16E-05	2	8
1.00E-04	5	13
3.16E-04	9	22
1.00E-03	15	37
3.16E-03	28	65
1.00E-02	33	98
3.16E-02	41	139
1.00E-01	59	198
3.16E-01	38	236
1.00E+00	33	269
3.16E+00	31	300
1.00E+01	16	316
3.16E+01	2	318
1.00E+02	1	319
3.16E+02	0	319

Table 4. Statistics for ratio of event emission rate to long-term emission rate	
Statistic for Ratio	*Value*
Median	0.043923
75th %ile	0.342655
90th %ile	2.204754
95th %ile	3.344422
96th %ile	3.400832
97th %ile	3.8126
98th %ile	4.790098
99th %ile	8.973897
Max	74.37138
Average	0.815352

Figure 1. Cumulative probability density for ratio of event to routine emission rates.

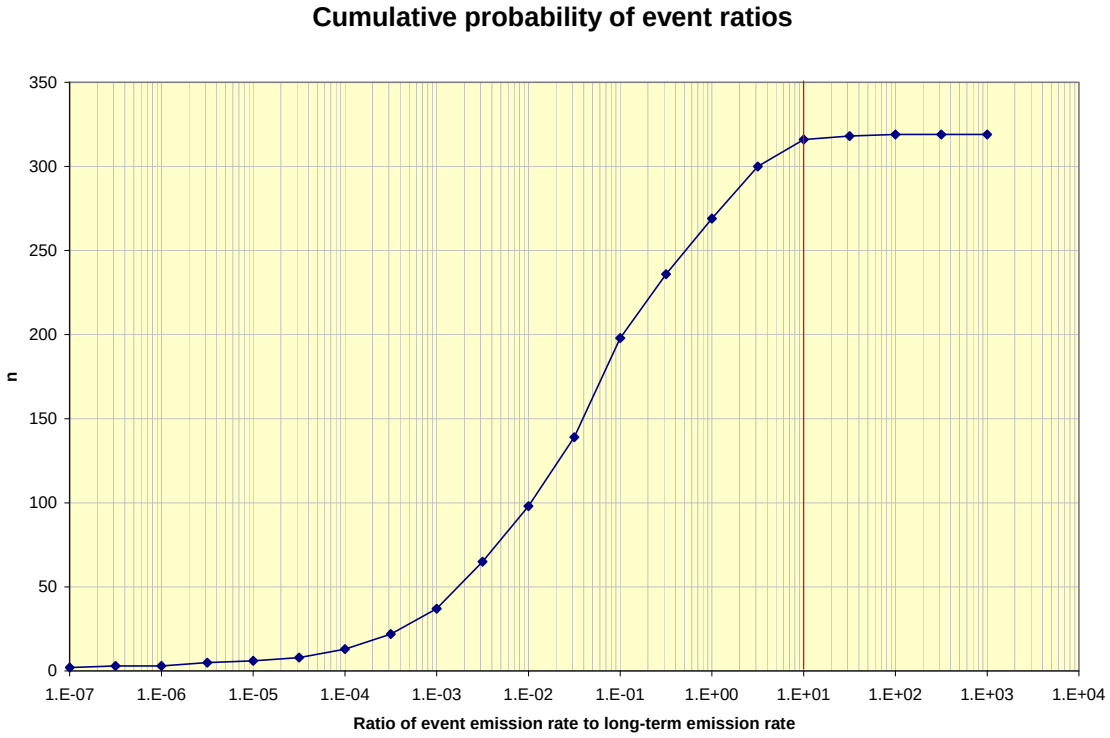


Figure 2. Relationship between ratio of event to duration emission rate and emission duration.

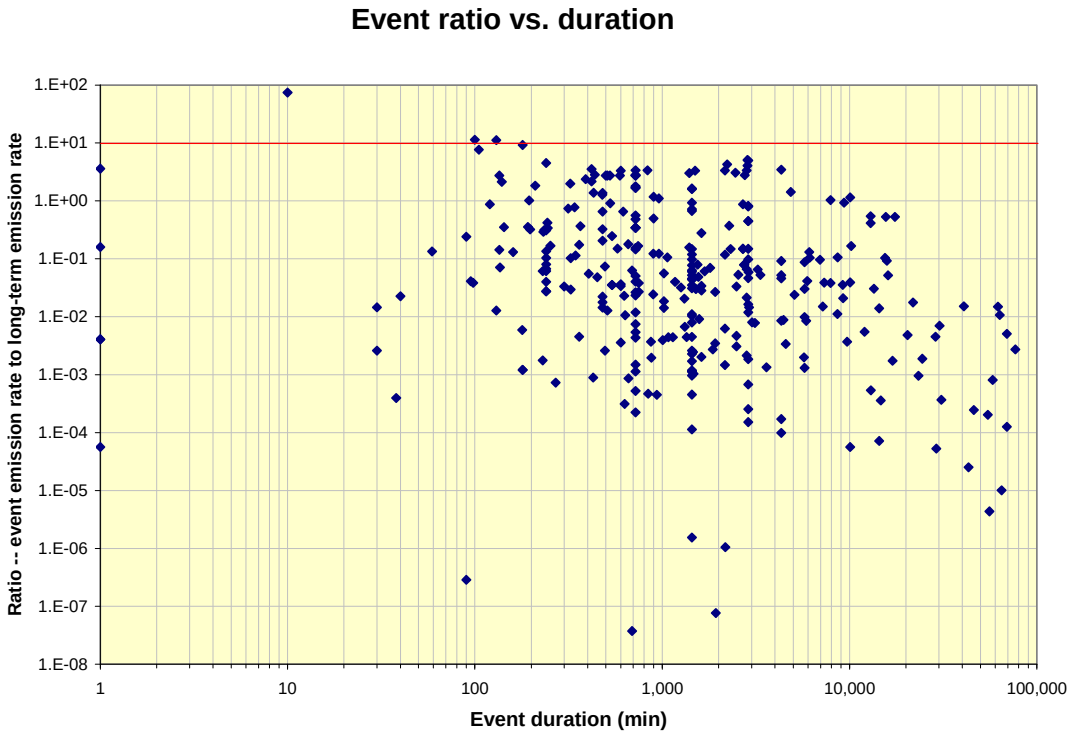


Figure 3. Relationship between ratio of event to duration emission rate and emission duration, as percentage of total time.

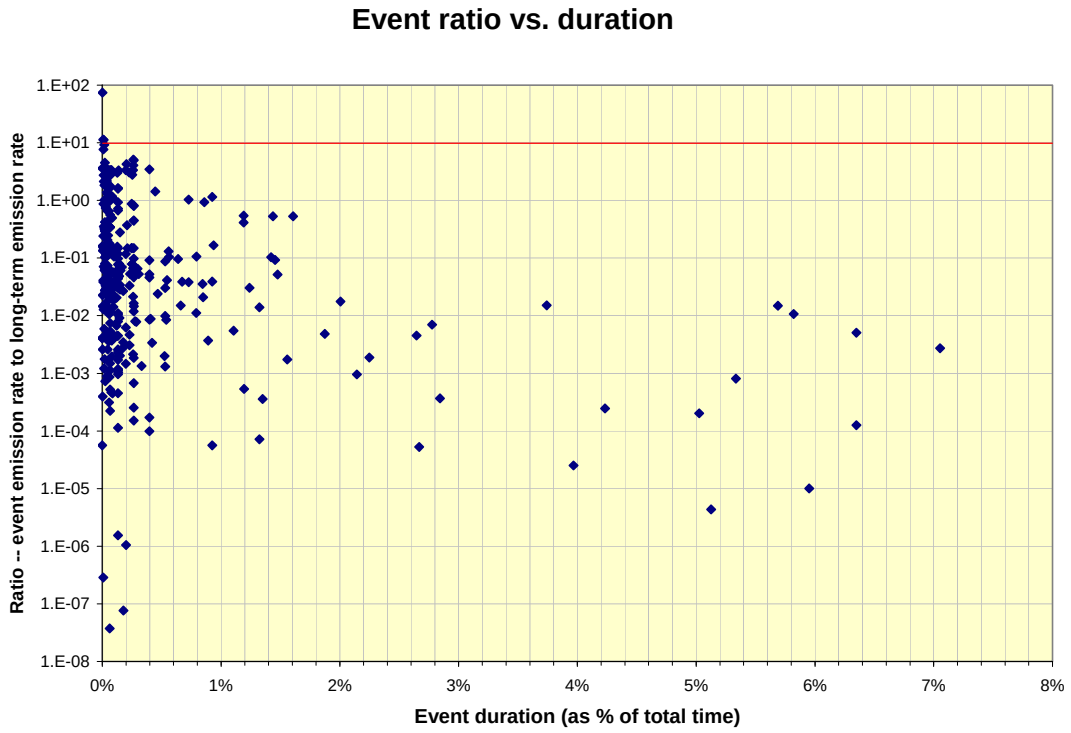
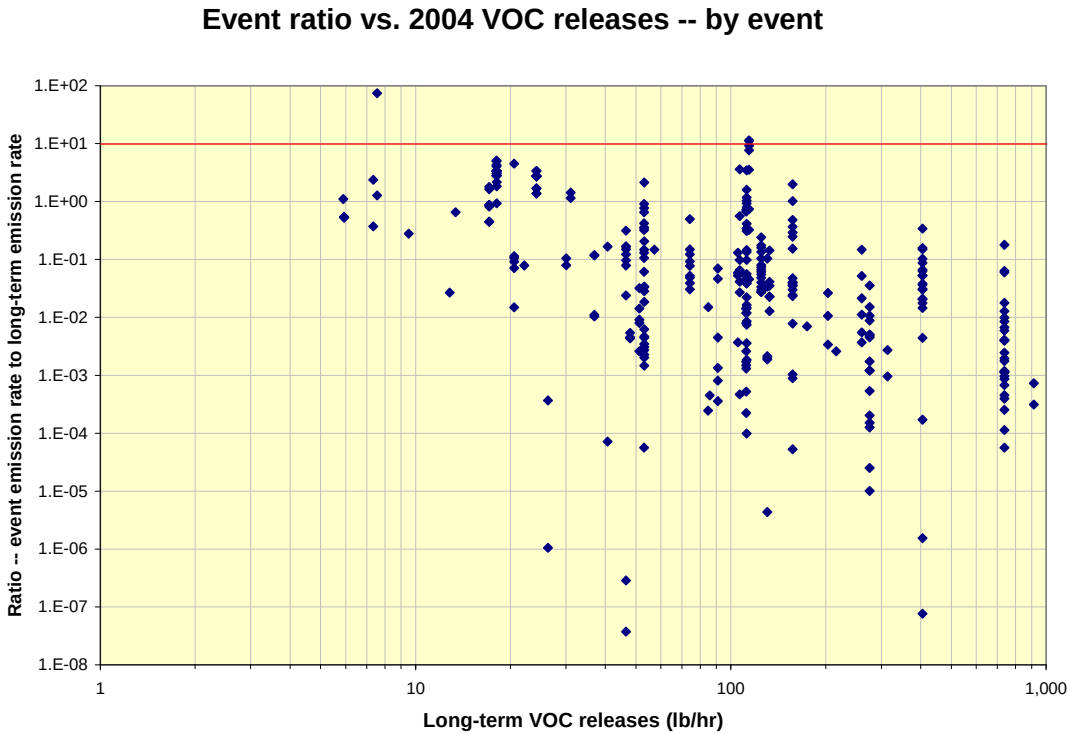


Figure 4. Relationship between ratio of event to duration emission rate and total amount emitted during the event.



APPENDIX C: Technical Support Document for TRIM-Based Multipathway Screening Scenario for RTR: Summary of Approach and Evaluation

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TABLE OF CONTENTS

LIST OF EXHIBITS

EXECUTIVE SUMMARY

This report describes the methods used by EPA to evaluate multipathway exposures to hazardous air pollutants and the associated human health risks. In particular, the report explains the methods, assumptions, and input data used to develop a screening scenario that is used in the first tier of Risk and Technology Review II (RTR II) assessments of such emissions. This report describes the development of the screening scenario and its application in generating qualitative, screening-level estimates of human exposure to PB-HAPs and consequent risk.

BACKGROUND

The Clean Air Act directs the U.S. Environmental Protection Agency (EPA) to assess the residual risk from hazardous air pollutants (HAPs) emitted by sources regulated by technology-based standards. To evaluate multipathway exposures and human health risks, a two-tiered approach was developed. In the first tier of the approach, a screening evaluation is conducted that uses the identity and magnitude of HAP emissions from a source to determine whether that source meets certain human health risk-based criteria with respect to multipathway exposures. The purpose of this first-tier screening is to eliminate facilities from further analysis that pose no unacceptable risk to human health, while identifying those facilities that warrant a second-tier, more refined, site-specific analysis of residual risk.

PURPOSE OF THE SCENARIO

The approach described here for evaluating human multipathway exposures and risks consists of an initial, screening-level tier that can be conducted quickly and efficiently to determine those facilities for which multipathway risks are expected to be below levels of concern. The key component of the first tier of this approach is a multipathway screening scenario based on EPA's Total Risk Integrated Methodology (TRIM). The TRIM-based modeling scenario provides a means for quickly and efficiently completing an initial non-inhalation exposure and risk screening analysis of a facility. The scenario is applied for use in RTR evaluations by calculating *de minimis* emission rates for selected PB-HAPs that correspond to a cancer risk of 1 in 1 million or a chronic non-cancer hazard quotient (HQ) of 1. These *de minimis* or threshold emission rates then can be used in the first risk screening step without requiring additional model runs. Sources whose emissions exceed the *de minimis* emission rate for any PB-HAP would be subjected to refined evaluation(s) in a second tier analysis.

The scenario has been used to calculate numerical exposure and risk values for 4 of the 14 HAPs that OAQPS has identified as candidates for multipathway risk assessments: cadmium, mercury, dioxins (i.e., chlorinated dibenzo-*p*-dioxins and -furans), and polycyclic organic matter. These compounds were selected because they are expected, based on current knowledge of relative emissions and toxicity, to pose a substantial share of the non-inhalation risks to humans from air emissions at sources subject to residual risk provisions of the Clean Air Act. The scenario is *not intended to be used to produce quantitative estimates* of actual or potential risk. Rather, it provides a basis for determining if residual human health risks are of potential concern. Such determinations can then be used to support decisions to proceed with or forego more definitive analyses of non-inhalation exposures to HAPs and the associated risks. The scenario does, however, provide a technically defensible starting point for additional fate and transport and exposure/risk analyses of facility emissions that are not "screened out" in the first tier evaluation.

OVERVIEW OF SCREENING SCENARIO

This screening scenario is intended to reduce the possibility that EPA will fail to identify unacceptable risks. Thus, the scenario's conservative approach most likely overestimates risk, which is appropriate for a screening assessment. Parameter values were defined carefully, and properties having more uncertainty were assigned greater conservative bias to prevent underestimating potential risks. The screening scenario is designed to estimate the upper end of the range of individual, long-term, non-inhalation exposures for situations likely to be encountered in the United States. The result of reviewing a broad range of conditions and selecting values representative of higher exposures in conceptualizing and building the screening tool is that the scenario is unlikely to occur at any one location but has a high likelihood of representing the upper end of any *potential* exposures.

The screening scenario addresses non-inhalation exposures, which can occur through both dermal and ingestion exposure pathways. Pathways examined include incidental ingestion of soil; ingestion of homegrown produce, beef, cows' milk, poultry and eggs, and pork; and ingestion of fish. Dermal absorption of chemicals that are originally airborne is generally relatively minor, and this pathway was not included in the scenario used to calculate *de minimis* emission thresholds. A highly conservative estimate of dermal exposures and risks was calculated for comparison to ingestion exposures and risks. In addition, exposure to nursing infants via consumption of contaminated breast milk was evaluated for dioxins as a separate scenario.

For this approach, chemicals were modeled separately to evaluate the potential for risks, with exposures for each PB-HAP summed across all ingestion exposure pathways. Exposures were modeled for a hypothetical farm homestead and fishable lake near an emissions source. For this setting, exposures were estimated for a hypothetical individual assuming subsistence consumption of all potentially contaminated foodstuffs from the farm or lake. The scenario was purposely designed to produce conservative (i.e., health-protective) results, and certain critical exposure/activity assumptions, such as food ingestion rates, were selected from the upper ends (e.g., the 90^{th} percentile) of representative exposure parameter distributions. The physical/chemical environment was parameterized with a mix of typical and health-protective values. The scenario's spatial/temporal aspects and the components that influence air concentrations were also chosen so that concentrations in environmental media would not be underestimated given the range of possible settings and meteorological conditions that might be encountered. Properties of the environmental media were parameterized with either typical or conservative values, with a more protective bias introduced for properties having greater uncertainty.

MODELING FRAMEWORK

The approach for risk evaluation of ingestion exposures and risk screening has four components:

1. fate and transport modeling of PB-HAPs emitted to air that partition into soil, water, and other environmental media (including fish);
2. modeling of transfer and uptake of PB-HAPs by farm food chain media from soil and air;
3. estimating ingestion exposures for the selected media contact scenarios and average daily ingestion doses for a hypothetical human receptor; and
4. calculating lifetime cancer risk estimates or chronic non-cancer HQs for each HAP.

TRIM.FATE SCENARIO CONFIGURATION AND PARAMETERIZATION

To model chemical fate and transport in the environment, EPA's Fate, Transport, and Ecological Exposure (TRIM.FaTE) module of the TRIM system was used. The physical configuration of the scenario was designed to be generally conservative and the environmental and chemical-specific properties were parameterized with either conservative or central-tendency values. Chemical/physical properties were obtained from peer-reviewed and standard reference sources. The spatial layout represents a farm homestead and a fishable lake near an emissions source. The predominant wind direction is toward the farm and lake watershed, and the downwind modeling area is symmetrical around a 10-kilometer east-west line and divided into five pairs of parcels. The aquatic food web in the scenario is meant to represent a generic aquatic ecosystem within a 47-hectare lake.

Fate and transport modeling outputs include average PB-HAP concentrations and deposition rates for various media (air, soil, surface water, and fish) for each year and for each parcel of the model scenario. TRIM.FaTE can output instantaneous chemical concentrations for a user-specified time step and also can be configured to calculate temporal averages. For the screening scenario, the model outputs results on a daily basis, and daily concentration results are averaged to obtain annual average concentrations. The source is assumed to emit for 50 years.

EXPOSURE AND RISK CALCULATIONS

The Multimedia Ingestion Risk Calculator (MIRC) was developed to carry out required farm food chain transfer, ingestion exposure, and risk calculations. Concentrations in farm food chain media are calculated using empirical biotransfer factors (e.g., soil-to-plant factors, which are the ratios of the concentrations in plants to concentrations in soil). Ingestion exposures based on exposure factors, including food-type-specific ingestion rates, are calculated for a hypothetical exposed individual. Lifetime cancer risks and the potential for chronic non-cancer effects are estimated using chemical-specific ingestion cancer slope factors and reference doses. Exposure pathways evaluated include incidental ingestion of soil and consumption of fish, produce, and farm animals and related products. Cancer risk estimates and HQs are calculated separately for each PB-HAP included in an analysis.

CALCULATION OF *DE MINIMIS* EMISSION THRESHOLDS

After the configuration of the TRIM.FaTE and MIRC modeling scenarios was completed, *de minimis* emission rate thresholds were calculated by conducting iterative model simulations to determine emission rates for cadmium, mercury, dioxins, and polycyclic organic matter that correspond to a cancer risk of 1 in 1 million or a chronic non-cancer hazard quotient (HQ) of 1. Given the generally conservative nature of the scenario inputs, these thresholds are assumed to be appropriate for screening sources emitting these HAPs.

EVALUATION OF SCREENING SCENARIO

Model evaluations serve as an important aspect of environmental risk assessments by illustrating the performance of the model under different conditions and assumptions and facilitating the comparison of model outputs to measurement data and other modeling results. Evaluations thereby provide an opportunity to gain confidence in model performance and identify and better characterize uncertainties associated with model construct and inputs. The screening scenario was analyzed through comparisons to the literature and sensitivity analyses.

Outputs from the screening scenario based on *de minimis* emission values were compared to literature values for cadmium, mercury, 2,3,7,8-TCDD, and benzo[a]pyrene. In general, taking into account the limitations of this type of comparison, the configuration of the models for screening appear to be reasonable. Chemical partitioning predicted by the model is generally consistent with information on environmental partitioning presented in the literature for media of concern. Where results are not consistent with literature values, a more detailed investigation of underlying assumptions may help to identify means of adjusting the scenario configuration.

Sensitivity analyses were performed to evaluate the influence of model parameters and to provide information on which parameters are likely to be most influential in dictating the uncertainty associated with the results. The sensitivity analyses conducted on the RTR screening modeling scenario encompassed the fate and transport modeling carried out using TRIM.FaTE and the farm food chain and ingestion exposure calculations performed using MIRC. A systematic sensitivity analysis was conducted by varying each input independently and calculating the resulting effect on the risk or hazard quotient estimates in order to rank the variables from most to least sensitive. The analysis suggests that several TRIM.FaTE variables (including wind speed, mixing height, emission rate and, for methyl mercury, sediment deposition rate) have the largest effect on the risk and hazard estimates. The estimates of hazard and risk are also highly sensitive to key parameters in the primary exposure pathway (i.e., ingestion of food types resulting in the highest exposures) for each PB-HAP. Other analyses performed indicated that accounting for temporal correlations in ingestion rates and body weights and varying the body weight and ingestion rate percentiles used in the model scenario have a limited effect on the risk and hazard estimates. The use of site-specific meteorological variable values (as opposed to the generic screening scenario values) resulted in a decrease in the risk and hazard estimates of approximately one order of magnitude.

In addition, media concentrations estimated using the screening scenario were compared to analogous outputs estimated using site-specific TRIM.FaTE model applications configured for two secondary lead smelting sources. The same emission rates for benzo[a]pyrene, 2,3,7,8-TCDD, elemental mercury, and divalent mercury were entered into the screening scenario and the site-specific model scenarios. Model results were compared for soil, water, and sediment compartment types. In all media, the screening scenario produced higher concentrations for all chemicals than the site-specific model applications, the expected result given that the screening scenario is conservative (and therefore tends to result in higher media concentrations).

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C-1 Introduction

C-1.1 Background

Section 112(f)(2)(A) of the Clean Air Act (CAA) directs the U.S. Environmental Protection Agency (EPA) to assess the risk remaining (residual risk) from hazardous air pollutants (HAPs) that continue to be emitted from sources after application of maximum achievable control technology (MACT) standards under section 112(d) of the CAA. Under these requirements, EPA will promulgate additional emission standards for a source category if the MACT standards do not provide an "ample margin of safety" for human health. One aspect of human health that EPA must consider is the potential for exposures to HAPs via non-inhalation pathways and the risks associated with such exposures.

As described in EPA's *Risk and Technology Review (RTR) Assessment Plan* (EPA 2006a), multipathway human health risks were preliminarily evaluated in 2006. The evaluation used draft National Emissions Inventory (NEI) data for RTR Phase II (RTR II) source categories and a simplified multipathway exposure modeling approach (see Appendix 5 in EPA 2006a).[1] However, as noted in the *RTR Assessment Plan*, EPA's intention was to develop an approach that would supersede the preliminary methods used in 2006 and involve the use of EPA's Total Risk Integrated Methodology (TRIM), a risk assessment modeling system for air toxics developed by OAQPS. The TRIM system can be used to predict the local impacts of persistent and bioaccumulative HAPs (PB-HAPs) from an emissions source to estimate associated human health risk.

EPA will implement a two-tiered approach to evaluate multipathway exposures and human health risks for RTR II. In the first tier, a screening evaluation is conducted that focuses on the identity and magnitude of HAP emissions from a given facility to determine whether a facility passes certain human health risk-based criteria. Sources that are "screened out" are assumed to pose no unacceptable risks to human health and are not considered in further analyses. For sources that do not pass the screen, more refined, site-specific multipathway assessments are conducted as appropriate. These human health risk results are considered, in combination with estimated inhalation human health risks, potential ecological risks, and other factors, to support decisions about residual risk for RTR II source categories.

This current document describes the technical basis for the first, screening-level tier of EPA's multipathway human health evaluation of RTR emission sources. Specifically, the models, configurations, and inputs used to derive *de minimis* emission thresholds in the first tier of the approach are described in detail here.[2] Analyses of the screening scenario conducted to evaluate the scenario's defensibility are also discussed. EPA expects that refined multipathway risk assessment methods (when required) will rely on the same TRIM-based modeling approach used to derive the *de minimis* thresholds used in the first screening tier. However, the details of refined assessments will vary depending on the facility location, source category, chemicals emitted, and other parameters, and the specific methods and processes involved in multipathway evaluations beyond the first tier are not explored in depth in this document.

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[1] The preliminary evaluation conducted in 2006 relied on a simpler modeling approach that did not utilize TRIM and involved a less rigorous analysis of parameter input values.

[2] *De minimis* is a Latin phrase that translates to "regarding minimal things." In the current context, the term *de minimis* is used in reference to human health risk that is below a level of concern (or, more specifically, a chemical emission rate that is not expected to result in unacceptable risks). See also the definition for "risk *de minimis*" in the "Glossary for Chemists of Terms Used in Toxicology" (IUPAC 1993).

C-1.2 Organization of this Document

This document presents the methods, assumptions, and inputs used to develop a method for evaluation of human multipathway exposures and health risks for RTR II risk assessments. Section 2 presents an overview of the methods for evaluating multipathway exposures and risks, a conceptual description of the TRIM-based scenario that is the basis for the screening-level *de minimis* emission thresholds, and a brief description of how refined analyses will be conducted for facility emissions that cannot be screened out in the first step. Section C-3 presents a technical description of the screening-level, TRIM-based modeling scenario and the configuration of the models used to estimate the *de minimis* levels. Section C-4 discusses evaluation activities conducted for this screening scenario and summarizes uncertainty. References cited in this report are listed in Section C-5.

C-2 Approach for Screening Evaluation of Multipathway Exposures

C-2.1 Overview

As described above, EPA's method for evaluating multipathway exposures for RTR risk assessments consists of a two-tiered process. Exhibit 2-1 diagrams the approach for evaluating non-inhalation, multipathway exposures to PB-HAPs. The first tier of this approach is the screening evaluation that relies on the TRIM-based "screening scenario" as the technical basis for decisions regarding whether a facility passes the screen. Air toxics emitted by a source under consideration are reviewed to determine first whether emissions of any PB-HAPs are reported. If such emissions are reported, the emission rates are compared to available *de minimis* threshold emission levels that have been derived using the TRIM-based screening scenario.[3] The list of chemicals that are PB-HAPs is discussed in Section C-2.2, and the use of *de minimis* emission thresholds is discussed in Section C-2.3.

The TRIM-based multipathway modeling configuration, referred to in this document as the "screening scenario," is a key component of the first tier of this approach, as this modeling application is the technical basis for determining the levels of PB-HAP *de minimis* emission thresholds. The term "screening scenario" is used in this document to refer collectively to the specific TRIM.FaTE and exposure modeling configuration described here, including the set of assumptions and input values associated with a hypothetical watershed and the exposure and risk scenarios evaluated for this watershed. The screening scenario is a static configuration, and its primary purpose is as a modeling tool to calculate the *de minimis* emission rates for PB-HAPs of concern. Descriptions of the components of the screening scenario are presented in Section C-2.4.

The two potential outcomes of the screening human health evaluation are:

- Non-inhalation exposures are unlikely to pose a human health problem; or
- The potential for unacceptable non-inhalation exposures cannot be ruled out and further assessment is required to determine the potential for unacceptable risk.

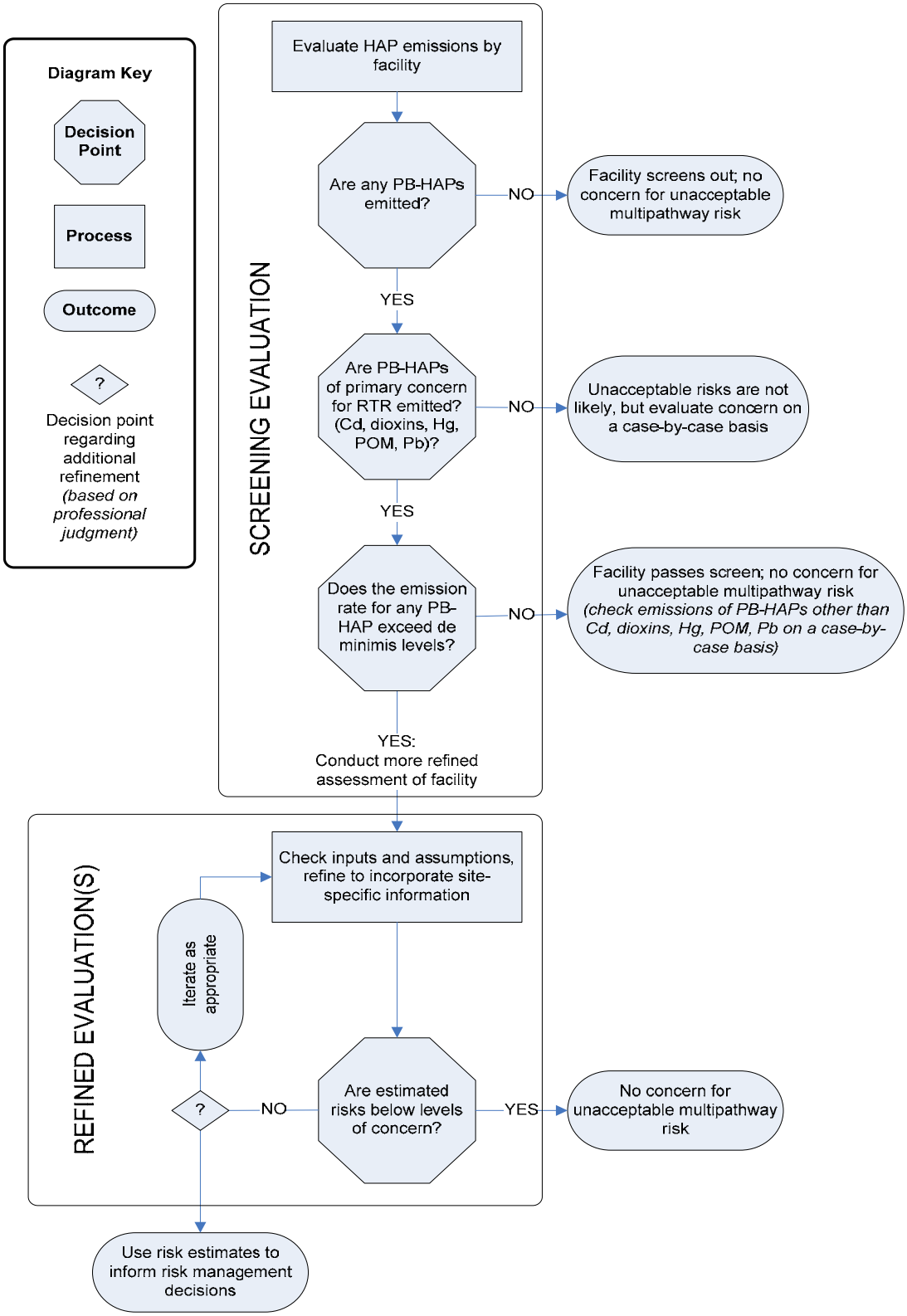
An ideal screening approach strikes a balance between being *conservative* – to ensure that unacceptable risks are identified, and being *accurate*, to minimize results suggesting that additional assessment is required when in fact the actual risk is low. Typically, gains in

[3] As described later in this report, to date, TRIM-based modeling has been used to calculate *de minimis* emission rates only for those PB-HAPs considered as those of highest concern.

accuracy in environmental modeling are accompanied by additional resource requirements. Stated another way, a suitable approach minimizes both false negatives and false positives. False negatives (i.e., results that suggest that the risk is acceptable when in fact the actual risk is high) can lead to inappropriate and non-protective health or environmental policy decisions. False positives (i.e., results that suggest more assessment is required when in fact the actual risk is low) can result in wasted resources by leading to additional, unnecessary analysis. For the evaluation of multipathway human health exposures to PB-HAPs, the methods for screening described in this document are intended to achieve this balance.

Facilities whose emissions exceed the established *de minimis* emission rate for any PB-HAP would be subjected to more refined evaluations. Because the initial screening evaluation enables EPA to confidently eliminate from consideration those facilities where risks from non-inhalation exposures are projected to be minimal, resources can be targeted toward those facilities that do not “pass” the screening test. An overview of the anticipated approach to refined evaluation is described in Section C-2.5.

Exhibit 2-1. Conceptual Decision Tree for Evaluation of Non-Inhalation Exposures of PB-HAPs



C-2.2 Chemicals of Potential Concern

EPA's assessment of multipathway human exposures for RTR focuses on PB-HAPs that OAQPS has identified as candidates for multipathway risk assessments. OAQPS developed a list of 14 chemicals and chemical groups that are PB-HAPs based on a two-step process taking into account the following:

- their presence on three existing EPA lists of persistent, bioaccumulative, and toxic substances, and
- a semi-quantitative ranking of toxicity and bioaccumulation potential of the entire list of HAPs.

The list's development and utility in hazard identification for multipathway risk assessment are further explained in Chapter 14 and Appendix D of Volume I of EPA's *Air Toxics Risk Assessment (ATRA) Reference Library* (EPA 2004a). Exhibit 2-2 presents the 14 chemicals and groups that are PB-HAPs.

The screening scenario described in this document is not configured for evaluating the risk potential for all 14 PB-HAPs on the list. Currently, the scenario can be used to quantitatively estimate exposures and risks for four PB-HAP compounds (indicated in bold in Exhibit 2-2). These compounds are the focus of the current scenario because, based on current emissions and toxicity considerations, they are expected to pose the vast majority of the non-inhalation risks to humans from air emissions at sources subject to residual risk provisions of the CAA.[4]

Exhibit 2-2. OAQPS PB-HAP Compounds [a]

PB-HAP Compound	Addressed by Screening Scenario?
Cadmium compounds	**Yes**
Chlordane	No
Chlorinated dibenzodioxins and furans	**Yes**
DDE	No
Heptachlor	No
Hexachlorobenzene	No
Hexachlorocyclohexane (all isomers)	No
Lead compounds	No
Mercury compounds	**Yes**
Methoxychlor	No
Polychlorinated biphenyls	No
Polycyclic organic matter (POM)	**Yes**
Toxaphene	No
Trifluralin	No

[a] Source of list: EPA 2004a. Compounds in bold text can be evaluated using the current version of the TRIM-based screening scenario.

[4] Potential impacts on human health from non-inhalation exposures to lead are evaluated for RTR using the National Ambient Air Quality Standard for lead, which takes into account multipathway risks. Non-inhalation exposures to the other nine PB-HAPs not addressed by the modeling scenario discussed in this report will be evaluated on an individual facility or source category basis as needed.

C-2.3 Use of *De Minimis* Emission Levels

The multipathway screening evaluation for RTR compares reported air emission rates of PB-HAPs (summed by PB-HAP for each facility) to *de minimis* emission rates derived using the screening scenario described here. A *de minimis* emission rate is the level that, when input to a risk model that uses emissions as a parameter, corresponds to a specified cancer risk or non-cancer HQ that, for the purposes of the evaluation being conducted, is assumed to be below a level of concern. *De minimis* rates were calculated for the screening scenario for a cancer risk of 1 in 1 million or an HQ of 1.0 (Exhibit 2-3).[5] Conceptually, a *de minimis* level for the RTR multipathway screening evaluation could be obtained by back-calculating the emission rate that results in the specified cancer risk or HQ level, taking into account the exposure and fate and transport calculations included in the model. Because the models used in this assessment are not designed to run "backwards," these rates were derived from regression equations established following a series of TRIM.FaTE and exposure/risk model runs spanning a wide range of emission rates for each chemical.

Exhibit 2-3. *De Minimis* Thresholds for Screening of Multipathway Exposures

Chemical	*De Minimis* Emission Rate (TPY)	Basis of Threshold (Type of Health Endpoint)
POM (as Benzo[a]pyrene toxic equivalents)	2.3E-03	Cancer
Dioxins (as 2,3,7,8-TCDD TEQ)	3.2E-08	Cancer
Divalent Mercury	1.6E-01	Non-cancer
Cadmium	6.5E-01	Non-cancer

The more probable risk for each emission rate would be lower than the level corresponding to the *de minimis* risk quantities in nearly all circumstances given the conservative and highly general nature of the screening scenario configuration. This conservatism ensures that a facility with cancer risk greater than 1 in 1 million or a chronic HQ greater than 1.0 is very unlikely to be omitted from refined evaluation.

Evaluation of Chemical Groups

In the screening evaluation, emissions of PB-HAPs are summed by chemical group for each facility. The summed emission rates for each group are then compared to the *de minimis* threshold corresponding to the appropriate chemical.

Emissions of polycyclic organic matter (POM, a HAP chemical group that includes polyaromatic hydrocarbons or PAHs) are often reported in NEI as unspeciated or partially speciated groups (such as "total PAHs" or "16-PAH") rather than as specific PAH compounds. In addition, quantitative data are lacking for some POM compounds and groups that are suspected carcinogens. To evaluate risks associated with exposure to emissions of the various POM species, EPA has grouped each POM species included in NEI into categories and then defined a cancer slope factor (CSF) for each group that can be used to estimate lifetime cancer risks.

[5] For chemicals that are known to cause both cancer and chronic non-cancer impacts, *and* for which acceptable quantitative dose-response values are available for both cancer and non-cancer endpoints, the endpoint that results in the lower *de minimis* level will be used for screening (i.e., the threshold will be based on the effect that occurs at the lower exposure level). For the set of PB-HAPs for which *de minimis* levels have been derived, only chlorinated dibenzo-dioxins and -furans meet both of these criteria. Because the cancer dose-response value is lower than that for non-cancer effects, the *de minimis* value is based on the cancer endpoint.

The CSFs used to evaluate risk for emissions of POM compounds and groups are listed in Attachment 2.

Similarly, polychlorinated dioxins and furans are reported in NEI as individual congeners, congener groups, or as toxic equivalents (TEQs) that are scaled relative to the toxicity of 2,3,7,8-TCDD. To evaluate cancer risks and non-cancer hazards from these compounds, EPA has developed CSFs and reference doses (RfDs) that apply to congener groups. These values are also presented in Attachment 2.

In the development of the RTR screening thresholds, we determined the *de minimis* levels for one compound from each of the POM and dioxin groups (i.e., benzo[a]pyrene and 2,3,7,8-TCDD). Then, to use this *de minimis* threshold in an evaluation of facility emissions of POMs or dioxins, a toxicity-weighted emissions sum is calculated for each group using the toxicity of each modeled chemical – benzo[a]pyrene and 2,3,7,8-TCDD – to scale the total POM or dioxin group emissions. This approach avoids the need to develop *de minimis* emission rates for every POM and dioxin congener (some of which are not included in the existing TRIM.FaTE algorithm library). A consequence of this approach is that benzo[a]pyrene and 2,3,7,8-TCDD serve as fate and transport surrogates. That is, the behavior of these two compounds is assumed to adequately represent the behavior of all other compounds included in the POM and dioxin PB-HAP groups, respectively.

De minimis emission thresholds were developed individually for elemental and divalent mercury. Both were based on the lower of the thresholds associated with multipathway exposures to divalent mercury and methyl mercury.[6] However, only speciated emissions of divalent mercury are screened because the sum of elemental mercury emissions across all NEI facilities is less than the elemental mercury *de minimis* level.

C-2.4 The TRIM-Based Screening Scenario: Basis for *De Minimis* Emission Rates

The TRIM-based modeling screening scenario described in this document was used to provide a means to *qualitatively estimate the potential for unacceptable non-inhalation risks for PB-HAPs emissions from facilities* in the context of residual risk assessments conducted as part of RTR II. The screening scenario used to derive *de minimis* emission rates is not intended to be representative of any particular situation. Rather, it was developed for the purpose of RTR to portray an exposure scenario at least as conservative as any situation that might plausibly be encountered in the United States. The range of conditions considered when conceptualizing and building the screening scenario was chosen so that any given individual, long-term exposure condition for a given geographic region would be reasonably likely to be captured. These criteria were met by constructing a hypothetical scenario that would be protective in key aspects, including spatial orientation, meteorology, types of exposures, and ingestion rates. The overall result is a scenario that is unlikely to occur at any one location but has a high likelihood of representing the upper end of all *potential* exposures. This latter aspect accomplishes the goal of striking a balance between conservatism and accuracy called for in the ideal screening approach.

For this approach, exposures were modeled for a hypothetical farm homestead and fishable lake near an emissions source. The hypothetical individual exposed to PB-HAPs in this

[6] Note that TRIM.FaTE models the transformation of mercury within the environment; thus, emissions of only divalent mercury will likewise result in multipathway exposures to both elemental and methyl mercury also. Emissions of only elemental mercury will result in multipathway exposures to both divalent and methyl mercury also.

scenario was assumed to derive all potentially contaminated foodstuffs from the farm or lake. Many of the exposure/activity assumptions were selected from the upper ends of representative exposure parameter distributions. The physical/chemical environment was parameterized with a mix of typical values (such as national averages) and health-protective values (i.e., values that would tend to overestimate concentrations in media). The spatial and temporal aspects of the scenario and the components of the scenario that influence air concentrations were chosen from the upper ends of their possible ranges so that concentrations in the environmental media would not be underestimated given the wide range of possible settings and meteorological conditions that might be encountered. Chemical-specific and non-chemical-specific properties of the environmental media were parameterized with either typical or conservative values (with a greater conservative bias introduced for properties having greater uncertainty).

The development and application of the screening scenario for residual risk evaluations considered EPA's technical and policy guidelines presented in the R*esidual Risk Report to Congress* (EPA 1999); Volumes I and II of the *Air Toxics Risk Assessment Reference Library* (EPA 2004a, 2005); and other EPA publications. The scenario described in this document is the culmination of analyses completed over the past 5 years; it provides the basis for an efficient and scientifically defensible method for screening multipathway human health risk and is a solid foundation for conducting more refined analyses when necessary. Nevertheless, this scenario should not be considered "final" but rather a product that can continue to evolve based on feedback from the scientific community and Agency reviewers, lessons learned as the scenario is further applied for RTR, variations in EPA's needs and requirements, and other factors.

C-2.4.1 Exposure Routes Evaluated

The screening scenario is intended to address non-inhalation exposures (inhalation exposures are being evaluated separately for RTR II using a dispersion modeling approach to estimate ambient air concentrations). The quantitative aspects of this non-inhalation screening evaluation for human exposures focus primarily on human exposures via the following ingestion pathways:

- Incidental ingestion of soil,
- Ingestion of homegrown produce,
- Ingestion of homegrown beef,
- Ingestion of milk from homegrown cows,
- Ingestion of homegrown poultry and eggs,
- Ingestion of homegrown pork, and
- Ingestion of fish.

Non-inhalation exposure to PB-HAPs also can occur by way of the dermal pathway (e.g., through incidental contact with PB-HAP-contaminated soil). However, dermal absorption of chemicals that are originally airborne is generally a relatively minor pathway of exposure compared to other exposure pathways (EPA 2006, CalEPA 2000). The risk from dermal exposure in the environmental setting from airborne toxicants is expected to be a fraction of the risk from inhalation exposure or exposure via ingestion of contaminated crops, soil, or breast milk, for example (CalEPA 2000). Preliminary calculations of estimated dermal exposures and risk of PB-HAPs, presented in Section C-3.2.4, showed that the dermal exposure route is not a significant risk pathway relative to ingestion exposures. Assessment of dermal exposure through incidental contact with soil could be conducted on facilities that require refined evaluation following the screening evaluation if deemed necessary. Procedures for estimating dermal absorption from soil would be based on EPA's dermal exposure assessment principles

and applications (EPA1992b) and EPA's *Superfund Human Health Evaluation Manual* (EPA 2004c).

Another ingestion pathway – through breast milk by nursing infants – can also be of concern for chlorinated dibenzo-dioxins and -furans (typically referred to collectively as "dioxins," nomenclature that is used elsewhere in this document when referencing the collective chemical category), and may also be of concern for mercury. Algorithms have been developed for calculating the exposure and risk associated with dioxin contamination of breast milk and are used to evaluate the likelihood of developmental effects resulting from exposure to dioxins via this pathway. Assessment of breast milk exposure for nursing infants will be assessed when refined evaluations are conducted; this exposure pathway is not incorporated in the calculation of the *de minimis* levels.

One other non-inhalation exposure route discussed in ATRA Volume I of possible concern for PB-HAPs is ingestion of drinking water from surface water sources. This exposure route, however, is not evaluated in the current assessment. The drinking water exposure pathway is not likely for the modeling scenario developed for this analysis because the likelihood that humans would use a lake as a drinking water source was assumed to be low.[7]

C-2.4.2 Approach to Configuration and Parameterization

This screening scenario is intended to *reduce* the possibility that EPA would ***not*** mis-identify unacceptable risks. Although the "conservative" approach likely overestimates risk, EPA determined that conservatism is appropriate for the purposes of screening assessments. As in the preliminary multipathway screening for RTR conducted in 2006 (EPA 2006a), exposures were modeled for a hypothetical farm homestead and fishable lake located adjacent to an emissions source. The hypothetical individual for which exposures were calculated was assumed to derive all potentially contaminated foodstuffs from these adjacent locations, and many of the exposure/activity assumptions (e.g., amount of food consumed per day) were selected from the upper ends of representative exposure parameter distributions.

The physical/chemical environment represented in the screening scenario was parameterized with two types of values. One type is typical values, such as national averages. The second type is health-protective, conservative values, or values that would tend to overestimate concentrations in media driving ingestion exposures for humans, based on knowledge of exposure patterns. In general, the spatial and temporal aspects of the scenario and the components of the scenario that influence air concentrations and deposition rates (which in turn affect all other exposures) were defined to be relatively conservative. That is, they were chosen from the upper ends of their respective possible ranges so that the wide range of possible physical settings and meteorological conditions would be captured. Chemical-specific and non-chemical-specific properties of the environmental media were parameterized with either typical or conservative values; properties having greater uncertainty were assigned greater conservative bias.

The spatial layout of the scenario and the meteorological data (or a combination of these two factors) are generally more influential than physical/chemical parameters in dictating the screening model outcomes, taking into account the potential range of variation in possible values. For example, where and how the layout is spatially oriented relative to the dominant

[7] An exception to this generality would be reservoirs used for drinking water supplies. This situation may be worthy of additional analysis, if warranted by the characteristics of a given assessment (e.g., to estimate PB-HAP concentrations in treated drinking water derived from reservoirs).

wind direction can dramatically affect the concentrations in air, thereby driving estimated concentrations of PB-HAPs in soil, water, and biota. In contrast, a relatively large change in soil characteristics within the range of possible values (e.g., organic carbon content, water content) might result in relatively small changes in outputs.

The mix of conservative and typical approaches and values is expected to result in a scenario configuration that, on average, is likely to over-predict environmental concentrations of PB-HAPs in media of interest for this evaluation. Given the intended application of this scenario as a screening tool, this conservative bias was deliberate, because of the desire to ensure that unacceptable risks are not overlooked (i.e., to minimize false negatives). Although the inclusion of typical values where warranted is intended to minimize the number of false positives, some false positives are to be expected from a screening scenario. These false positives would be addressed in iterations of the refined evaluation for a particular source.

C-2.4.3 Modeling Framework

The approach for risk screening (and ingestion exposure) evaluation described here can be divided into four steps:

1. Fate and transport modeling of PB-HAPs emitted to air by the source that partition into soil, water, and other environmental media (including fish);[8]

2. Modeling of transfer and uptake of PB-HAPs into farm food chain media (produce, livestock, dairy products) from soil and air;

3. Estimating ingestion exposures as a result of contact with the various selected media and estimating average daily ingestion doses for a hypothetical human receptor; and

4. Calculating lifetime cancer risk estimates or chronic non-cancer HQs, as appropriate, for each PB-HAP and comparing these metrics to selected risk management points of departure used in the RTR II.

The relationship among these four processes is shown in Exhibit 2-4.

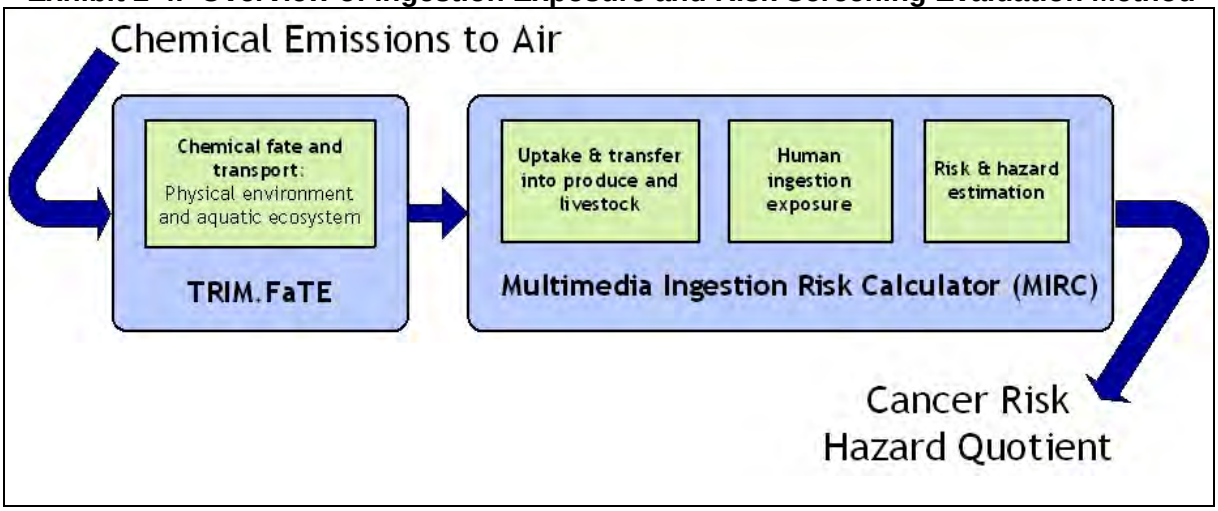
EPA's TRIM methodology was conceived as a comprehensive modeling framework for evaluating risks from air toxics, and the TRIM system was designed to address each of the four steps involved in screening ingestion risk.[9] Currently, however, only one component corresponding to the first step included in Exhibit 2-4 – the fate and transport module – is available for application in an ingestion risk assessment. EPA has completed some development activities for TRIM.Expo-Ingestion and TRIM.Risk-Human Health, two additional modules that cover the other three steps. Modeling software, however, is not currently available for these modules. For the RTR screening scenario, the Multimedia Ingestion Risk Calculator (MIRC), a Microsoft Access-based computer framework, was constructed to complete the calculations required for estimating PB-HAP concentrations in farm food chain media, average

[8] As discussed below, concentrations in fish calculated by the TRIM.FaTE model were used in the current approach to estimate ingestion exposures for humans consuming fish. Modeling of fish concentrations is therefore discussed in this document as part of the fate and transport modeling. Uptake of PB-HAPs into all other biotic media assumed to be ingested is modeled in the second step.

[9] Information regarding the current status of TRIM modules as well as comprehensive documentation of modules that have been developed thus far can be accessed on EPA's Technology Transfer Network (TTN) on the Fate, Exposure, and Risk Analysis web site (http://www.epa.gov/ttn/fera/).

daily ingestion doses, and cancer risks and chronic non-cancer HQs. This framework is conceptually identical to the ingestion exposure and risk analyses that TRIM is intended to cover.

Exhibit 2-4. Overview of Ingestion Exposure and Risk Screening Evaluation Method



C-2.4.3.1 Fate and Transport Modeling

The fate and transport modeling step depicted in the first box in Exhibit 2-4 is implemented for RTR using the Fate, Transport, and Ecological Exposure module of the TRIM modeling system (TRIM.FaTE).[10] In developing the screening scenario, Version 3.6.2 of TRIM.FaTE was used to model the fate and transport of emitted PB-HAPs and to estimate HAP concentrations in relevant media. Additional information about TRIM.FaTE, including support documentation, software, and the TRIM.FaTE public reference library, is available on EPA's TTN at http://www.epa.gov/ttn/fera/.

The algorithms used to model mercury species and PAHs are described in Volume II of the *TRIM.FaTE Technical Support Document* (EPA 2002a). A comprehensive evaluation of the performance of TRIM.FaTE for modeling mercury was documented in Volumes I and II of the T*RIM.FaTE Evaluation Report* (EPA 2002b, 2005b). Algorithms specific to the fate and transport of 14 chlorinated dibenzo-dioxin and -furan congeners were added following the addition of those for mercury and PAHs. Documentation of the application of TRIM.FaTE for dioxin emissions is contained in the third volume of the *TRIM.FaTE Evaluation Report* (EPA 2004b). Parameterization of the TRIM.FaTE library used for RTR analyses with regard to

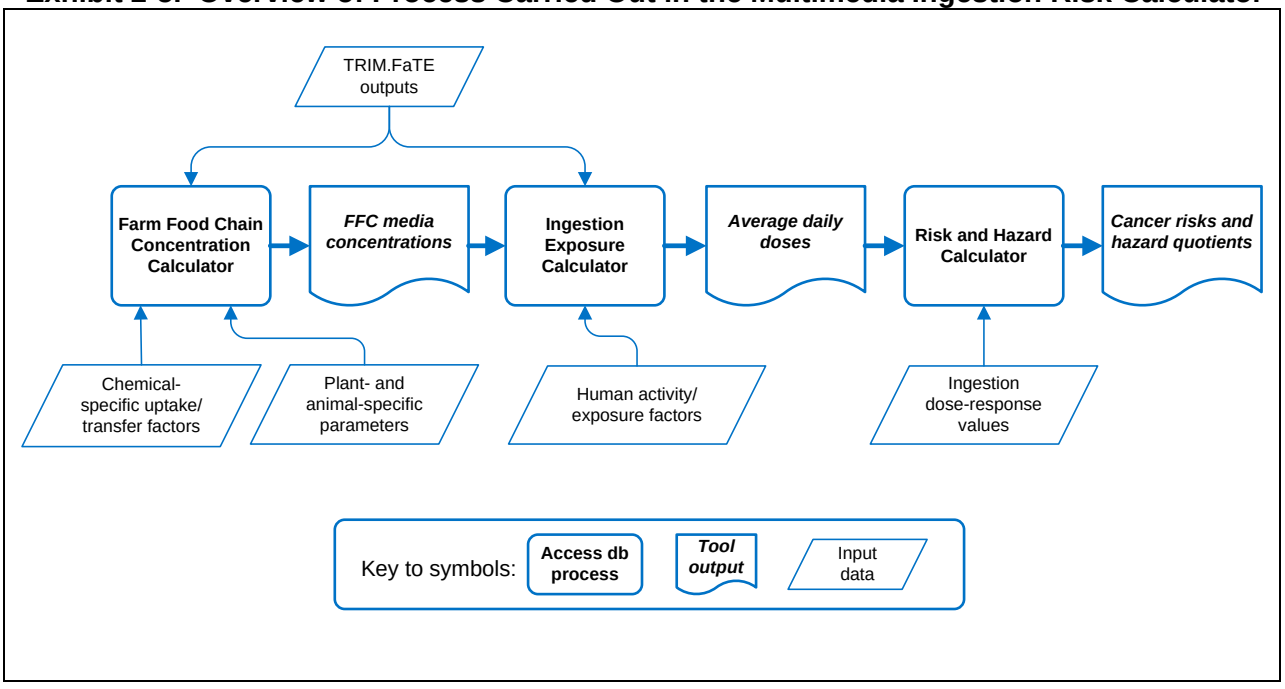
[10] TRIM.FaTE is a spatially explicit, compartmental mass balance model that describes the movement and transformation of pollutants over time, through a user-defined, bounded system that includes both biotic and abiotic compartments. Outputs include pollutant concentrations in multiple environmental media and biota, which provide exposure estimates for ecological receptors (i.e., plants and animals). The output concentrations from TRIM.FaTE are also intended to be used as inputs to a human ingestion exposure model to estimate human exposures. Significant features of TRIM.FaTE include: (1) a fully coupled multimedia model; (2) user flexibility in defining scenarios, in terms of the links among compartments, and number and types of compartments, as appropriate for the application spatial and temporal scale; (3) transparent, user-accessible algorithm and input library that allows the user to review and modify how environmental transfer and transformation processes are modeled; (4) a full accounting of all of the pollutant as it moves among environmental compartments during simulation; (5) an embedded procedure to characterize uncertainty and variability; and (6) the capability to provide exposure estimates for ecological receptors.

dioxins is identical to the configuration described in that third evaluation report. More recently (largely as part of this current project), the TRIM.FaTE public reference library has been updated to include information on modeling for cadmium. In general, many of the algorithms and properties included in the public reference library that are used to model mercury (except for the mercury transformation algorithms) are also applicable to cadmium. Comprehensive technical documentation of TRIM.FaTE algorithms specific to cadmium has not yet been compiled; however, all chemical-specific properties used by TRIM.FaTE to model cadmium (as well as PAHs, mercury, and dioxins) are documented in Attachment 1 to this document. Parameterization of the TRIM.FaTE scenario used for RTR screening is described in more detail in Section C-3.

C-2.4.3.2 Exposure Modeling and Risk Characterization

The algorithms included in MIRC that calculate chemical concentrations in farm food chain media and ingestion exposures for hypothetical individuals were generally obtained from EPA's *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities,* or HHRAP (EPA 2005a).[11] These algorithms, and the required exposure factors and other parameter values, were compiled into a database. An overview of the computational processes this tool carries out and the types of input data it requires is presented in Exhibit 2-5. This exhibit demonstrates the general relationships between the relevant TRIM.FaTE outputs (i.e., chemical concentrations in environmental media and fish) and the ingestion exposure and risk calculations carried out using MIRC. Additional discussion of exposure and risk calculations for this screening scenario is presented in Section C-3.2 and Attachment 2, and all inputs required by these calculations are documented in Attachment 2.

Exhibit 2-5. Overview of Process Carried Out in the Multimedia Ingestion Risk Calculator



[11] The farm food chain calculations and ingestion exposure equations to be included in the TRIM.Expo software are expected to be very similar to those included in HHRAP.

C-2.5 Refined Analyses

For facilities that are not screened out by comparison to the *de minimis* emission thresholds, additional analysis is required to evaluate the risks via multipathway exposures. EPA envisions that the screening scenario provides a technically defensible "starting point" for additional fate and transport and exposure/risk analyses, by substituting site-specific values for key parameters used in the modeling scenario. As appropriate, the scenario can be further refined in iterative steps until either the risks predicted are not of concern or sufficient information has been gathered to inform a risk management decision. Based on the model performance evaluations completed on the screening scenario, settings and parameters that might be appropriate for revision in a more refined analysis include meteorological inputs and spatial configuration assumed for TRIM.FaTE modeling, relevant ingestion exposure scenarios (based on surrounding land-use or other characteristics), and exposure factors such as ingestion rate for individual food types, among other inputs. The refined evaluation could eventually involve developing a site-specific TRIM.FaTE application that incorporates significant site-specific data (e.g., a model application that includes a site-specific spatial layout taking into account local geographic features and environmental parameter values selected from the best available data for that location).

C-3 Description of Modeling Scenario

C-3.1 TRIM.FaTE Scenario Configuration and Parameterization

As described in Section C-2.4.2, the physical configuration of the RTR Screening Scenario was designed to be generally conservative, and the environmental and chemical-specific properties were parameterized with either conservative or central-tendency values. Information regarding the scenario configuration and important aspects of the parameterization process, justifications for selecting particular property values, and certain uncertainties is presented in the sections that follow. Comprehensive documentation of TRIM.FaTE property values for this scenario is provided in Attachment 1.

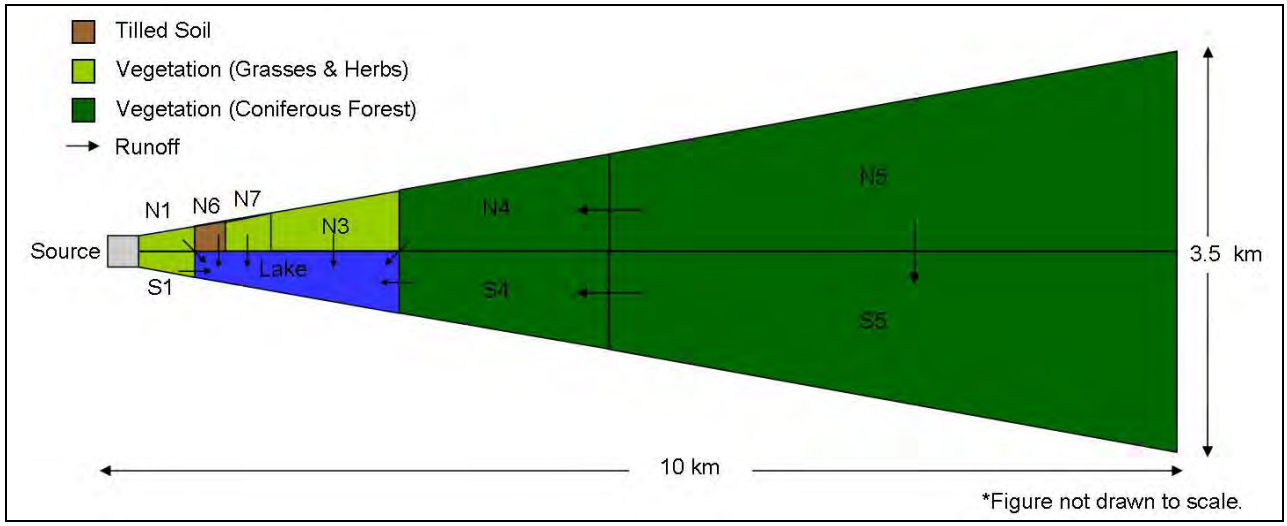
C-3.1.1 Chemical Properties

The chemical/physical properties that TRIM.FaTE requires, such as Henry's law constant, molecular weight, and other "general" parameters, were obtained from peer-reviewed and standard reference sources. Numerous other chemical-specific properties are related more specifically to a particular abiotic or biotic compartment type; these properties are discussed generally in the sections that follow and are documented in Attachment 1.

C-3.1.2 Spatial Layout

For the purpose of estimating media concentrations, the TRIM.FaTE scenario is intended to represent a farm homestead and a fishable lake (and its surrounding watershed) located near the emissions source of interest. A diagram of the surface parcel layout is presented in Exhibit 3-1. The source parcel is parameterized as a square with sides of 250 m, which is assumed to be a fair estimation for the size of a relatively small-to-medium facility at the fence line. With a predominant wind direction toward the east, the modeled layout is generally symmetric about an east-west line and is wedge-shaped to reflect Gaussian dispersion of the emission plume.

Exhibit 3-1. TRIM.FaTE Surface Parcel Layout



A lateral, downwind distance of 10 km was established for the watershed included in the scenario. Based on the results of dispersion modeling (results not presented here), the location of the maximum air concentration and deposition rate would be expected to occur relatively close to the facility (probably within a few hundred meters, with the exact location varying with stack height and other parameters) and well within a 10-km radius. Additionally, deposition rates for the PB-HAPs for which this screening scenario is applicable would be expected to decrease by about two orders of magnitude relative to the predicted maximum rate within a 10-km radius. Extending the modeling layout beyond a 10-km downwind distance would increase the amount of deposition "captured" by the modeled watershed, but the incremental chemical mass expected to accumulate in the watershed diminishes rapidly with distance. In addition, the impact of this additional deposited mass on ingestion exposures is expected to be negligible.[12] Given these conditions, a downwind length of 10 km was determined to be appropriate for the screening scenario.

The north-south width of the wedge-shaped watershed was set based on the observed behavior of chemicals emitted to the ambient air. If meteorological stability is known or can be assumed, the lateral spread of the plume (σ_y, measured from the centerline) at a certain distance from the source can be estimated using the Pasquill-Gifford curves. Turner (1970) derived the equations for these curves, which can be found in the ISC3 Dispersion Model Manual (among other sources).[13] For a relatively neutral atmosphere (stability class D), σ at 10 km is about 550 m using this estimation. In a Gaussian distribution, about 99.6 percent of the plume spread area is contained within 3σ of the median line. Therefore, the plume σ was set at 3 times 550 m, or approximately 1.75 km from the centerline at 10 km distance. The plume width for these conditions is expected to be about twice this distance, or 3.5 km. These dimensions were used to define the dimensions of the overall air and surface parcel layouts for the screening scenario.

[12] Mass deposited at the outer edge of the watershed is expected to result in only a very small increase in estimated exposure via fish consumption by increasing the chemical mass transported to the lake through erosion and runoff. The distance from these more distant locations to the lake would attenuate transport of chemical mass by erosion and runoff, dampening the effect of including additional deposition beyond 10 km. (Other exposure pathways would be largely unaffected; the soil concentrations used to calculate exposures for the farm food scenario are derived from soil parcels located close to the source and unaffected by deposition to the far reaches of the watershed.)

[13] http://www.epa.gov/scram001/userg/regmod/isc3v2.pdf

The surface (land and surface water) modeling area was initially divided into five pairs of parcels whose areas increase with distance from the source, which approximately corresponds to the spatial gradient that is expected in the downwind direction from the source. The second north parcel from the source was further divided into two parts, one of them tilled soil (Parcel N6) to represent agricultural conditions.

The depth of the surface soil compartments was set to 1 centimeter (cm), except for Parcel N6, for which the depth was set to 20 cm to simulate the effect of tillage. Characteristics of the soil layers (e.g., organic carbon content, air and water content, and sub-soil depth) were typically set to represent typical or national averages as summarized by McKone et al. (2001), for example.

Initial considerations when the layout was configured included the presence of a stream that ran along the bisecting east-west line from the southwestern corner of Parcel N3 through the eastern edge of the layout. In that configuration, the eastern extent of the pond was restricted by a parcel (S3) directly south of Parcel N3. The stream received chemical mass from Parcels S3, S4, and S5 and flowed directly into the pond. Preliminary modeling runs showed that the existence of a stream somewhat decreased the concentrations of 2,3,7,8-TCDD in the pond parcel and significantly decreased concentrations in Parcels S3 and S4. These results indicated that the pond was receiving more 2,3,7,8-TCDD mass through surface soil transfer than through the stream, perhaps due to a chemical sink into stream sediment. Given the goal of creating a conservative scenario, a stream was not included in the final layout.

The overall shape and boundaries of the air parcel layout mirror those of the surface parcel layout. A single air parcel (N2) overlies surface Parcels N6 and N7, and the air over the lake is divided into air Parcels S2 and S3 (mirroring the analogous parcels on the north side of the lake).

C-3.1.3 Watershed and Water Body Parameterization

Properties associated with the watershed soil and lake determine how pollutants in the system are transported through and accumulate in various compartments. These properties describe the physical characteristics of the environmental media included in the modeled region, as well as the assumed connections and relationships between media types and modeled spatial components that in turn affect chemical transport via water runoff, ground seepage, deposition of suspended sediments in the water column, and other processes. This section presents the justification for setting the key properties of the soil, water, and sediment compartments. Also discussed are some of the chemical properties related to watershed and waterbody processes (chemical-specific compartment properties in TRIM.FaTE) and the configuration of terrestrial plants included in the scenario.

C-3.1.3.1 Water Balance

Water-related properties of the lake and related watershed characteristics (e.g., runoff rates from each surface soil compartment) were set so that a simplified water balance is achieved. Although TRIM.FaTE maintains a chemical mass balance, the model does not calculate or maintain media mass balances (e.g., for water) except where specified in certain formulas. For the screening scenario, the parameters were set to satisfy two equations relating water volume. The first equation maintains a balance of water entering and leaving the terrestrial portion of the scenario:

[*total precipitation] = [evapotranspiration] + [total runoff]*

In this equation, total runoff is equal to the sum of overland runoff to the lake and seepage to the lake via groundwater.

The second equation describes the volumetric balance of transfers of water to and from the lake:

[total runoff] + [direct precipitation to the lake] = [evaporation from the lake surface] + [outflow from the lake]

Note that TRIM.FaTE actually uses only some of these properties (e.g., precipitation rate and surface runoff, but not evapotranspiration). The water characteristics assumed for the screening scenario are meant to represent a relatively wet and moderately warm location in the United States (USGS 1987). Following are the assumptions for this scenario:

- 35 percent of the total precipitation leaves the scenario through evapotranspiration.
- 65 percent of total precipitation remains in the modeled system and contributes to total runoff.
- Total runoff is divided between overland runoff and seepage to groundwater as follows:
    - 40 percent of total precipitation contributes to overland runoff.
    - 25 percent of total precipitation seeps into the groundwater and eventually flows into the lake.

For these calculations, the source parcel was considered to be outside the watershed and therefore was not included in the water balance. The evaporation rate from the lake was assumed to be 700 millimeters per year (mm/yr) based on data reported by Morton (1986) for various lakes. This estimate is probably more representative of cooler locations [by comparison, the overall average of evaporation rates from various reservoirs is reported by McKone et al. (2001) to be close to 1,200 mm/yr]. The runoff rate was defined to be spatially constant and temporally constant (i.e., it is not linked to precipitation events) throughout the modeled domain. Based on these assumptions, the outflow of water from the lake is about 18 million m^3/yr, which translates to a volumetric turnover rate of about 12.2 lake volumes per year.

Other quantitative water body and watershed characteristics TRIM.FaTE uses are listed in Attachment 1.

C-3.1.3.2 Sediment Balance

A simplified balance of sediment transfers between the watershed and the lake was also maintained for the screening scenario via the parameterization of sediment-related properties. As with water, the model does not internally balance sediment mass; these calculations were performed externally for the purposes of setting parameter values. The sediment balance maintained is described by the following equation, where terms represent mass of sediment:

[total surface soil transfers to the lake via erosion] =
[removal of sediment from the water column via outflow] + [sediment burial]

where the second term (removal of sediment from the water column via outflow) is accounted for in TRIM.FaTE by lake flushing rate and the third term (sediment burial) is the transfer of sediment from the unconsolidated benthic sediment compartment to the consolidated sediment layer.

To maintain the sediment balance, erosion rates were calculated for each surface soil compartment using the universal soil loss equation (Wischmeier and Smith 1978), assuming a relatively high rate of erosion. The total suspended sediment concentration is assumed to remain constant in TRIM.FaTE, and the flush rate of the lake (calculated via the water balance approach described above) was then used to estimate the removal of sediment from the modeling domain via lake water outflow. The difference between these sediment fluxes was taken to be the sediment burial rate. The sediment burial rate is the rate at which sediment particles in the unconsolidated benthic sediment layer are transported to the consolidated sediment, where the particles can no longer freely interact with the water column.

In TRIM.FaTE, the consolidated sediment layer is represented with a sediment sink; as with all sinks in TRIM.FaTE, chemical mass sorbed to buried sediment that is transported to the sink cannot be returned to the modeling domain. The burial rate is a formula property calculated by the model according to the difference between user-specified values for sediment deposition velocity (from the water column to the benthic sediment) and sediment resuspension velocity (back into the water column from the benthic sediments). These formula properties assume a constant volume of particles in the sediment layer (because the densities for benthic and suspended sediment particles were defined to the same value, the mass of particles in the sediment is also constant).

For the screening scenario described here, the average sediment delivery rate (i.e., transfer of sediment mass from watershed surface soil to the lake due to erosion) for the entire watershed was estimated to be about 0.0036 kilograms per square meter per day (kg/m^2-day), based on calculations using the universal soil loss equation (USLE). The HHRAP documentation notes that using the USLE to calculate sediment load to a lake from the surrounding watershed sometimes leads to overestimates (EPA 2005a). For this screening scenario, however, this approach was considered to be appropriate in that conservatism is a goal of the screening scenario.[14] Surface soil compartments adjacent to the lake are linked directly to the lake for the purposes of estimating erosion and runoff transfers (see layout in Exhibit 3-1). Erosion and runoff from the source parcel are linked directly to a sink and do not enter the screening scenario lake. The transport of sediment to the lake via overland flow (e.g., by streams) is thus assumed to be efficient. Note that erosion from parcels not directly adjacent to the lake is assumed to be somewhat attenuated, effected by using a lower sediment delivery ratio in the model.

Using the calculated surface soil erosion rates for the scenario, the total average daily sediment load to the lake from the watershed is about 16,600 kg/day. About 15 percent of this load is removed from the lake via outflow of suspended sediments (based on a calculated flush rate of 12.2 volume turnovers per year), with the remainder of the sediment input to the lake transferred to the sediment burial sink.

C-3.1.4 Meteorology

Meteorological properties used in TRIM.FaTE algorithms include air temperature, mixing height, wind speed and direction, and precipitation rate. These properties, which can vary significantly among geographic locations, as well as seasonally and hourly for a single location, greatly influence the chemical concentrations predicted in media of interest. Because the screening scenario is intended to be generally applicable for any U.S. location, and to minimize the

[14] Based on sensitivity analysis, a higher erosion rate will both increase surface water concentrations and decrease surface soil concentrations; however, the relative impact on resulting concentrations will be proportionally greater in the waterbody.

frequency of false negatives, a conservative configuration was used. The meteorology of the screening scenario was defined to ensure that (when used in combination with the selected spatial layout) the maximum exposures that might be encountered for the scenarios of interest would be encompassed (i.e., consumption of home-grown farm foodstuffs and self-caught fish, with all farm foods and fish obtained from locations impacted by chemicals emitted from the local source). However, ensuring that the meteorological parameters were not overly conservative, such as *always* having the wind blow toward the location of interest, was also important to avoid too many false positives.

The meteorological data for the screening scenario are intended to be representative of a location with a low wind speed, a wind direction that strongly favors the watershed, and a relatively high amount of total precipitation falling on the watershed. The values used were based on actual data trends for U.S. locations as specified in Exhibit 3-2; however, an artificial data set was compiled (for example, temporally variable meteorological parameters were made to vary only on a daily basis). This simplified approach allowed for greater control (relative to selecting a data set for an actual location) so that desired trends or outcomes could be specified. Also, using a meteorological data set with values varying on a daily basis rather than a shorter period (such as hourly, which is the typical temporal interval for meteorological measurements) reduced required model run time. Meteorological inputs are summarized in Exhibit 3-2.

The sensitivity of modeled 2,3,7,8-TCDD concentrations to changes in these meteorological variables was tested. Lower wind speeds and mixing heights affected concentrations the most. This sensitivity is not unexpected because lower wind speeds should increase pollutant deposition into the soil and lower mixing heights should reduce the volume through which pollutants disperse. The wind speed used for the screening scenario was 2.8 m/s, the 5^{th} percentile of annual average among 239 stations; by comparison, the mean annual average wind speed is 4.0 m/s in the contiguous United States). The mixing height (mean heights from four states) used was 710 m (the 5^{th} percentile of annual average among all 40 states in the SCRAM database).

Exhibit 3-2. Summary of Key Meteorological Inputs

Meteorological Property	Selected Value	Justification
Air temperature	Constant at 298 Kelvin	Recommended default value listed in HHRAP (EPA 2005a). Value is similar to the mean daily June temperature in the U.S. Deep South and to the mean daily July temperature in the U.S. Central Plains.[a]
Mixing height	Constant at 710 meters	Value is 5th percentile of annual average mixing heights for 463 U.S. locations, using data obtained from EPA's SCRAM Web site.[b] Value is the approximate U.S. median for periods without precipitation, based on data compiled by Holzworth (1972). Value is conservative compared to the 1- to 2-km typical mid-latitude daytime value (Stull 1988).
Wind direction	Blows from source parcel into scenario domain (west to east) 3 days per week; during other times does not blow into domain	A wind direction that favors the location of interest (for example, a watershed downwind of a source of concern) will tend to result in more emitted mass accumulating in the location of interest. For much of the U.S. mid-Atlantic and western regions, the wind tends to favor one direction (eastward).[d] For the hypothetical RTR scenario, a more extreme example of this pattern is represented by conditions in Yakima, Washington, where the wind blows eastward approximately 40 percent of the time based on a review of wind direction data compiled by the National Weather Service (NCDC 1995). This pattern was approximated in the RTR scenario with a configuration in which the modeled domain is downwind of the source three days out of seven.
Horizontal wind speed	Constant at 2.8 meters per second	Set to 5th percentile of annual average speed for 239 stations across the contiguous United States (about 50 years of data per station). Value is similar to the annual average wind speeds of the U.S. Deep South.[c]
Precipitation frequency	Precipitation occurs 3 days per week; wind direction blows into domain 2 of these days	This value was selected so that two-thirds of the total precipitation occurs when the domain is downwind of the modeled source. This pattern approximates that for rainy U.S. locations, where precipitation occurs 35 – 40% of the time (Holzworth 1972). These locations include parts of the U.S. Northeast and Northwest.[c]
Total Precipitation	1.5 meters per year	Assumed to represent rainy conditions for the United States. This annual precipitation amount is experienced in parts of the U.S. Deep South and parts of the U.S. Northwest Coast.[d] Conditional precipitation rate (rainfall rate when precipitation is occurring) is 9.59 mm per day, which is similar to conditions along the U.S. East Coast and Midwest.[c]

[a] National Climatic Data Center Historical Climate Series (NCDC-HCS) (2007). http://www5.ncdc.noaa.gov/climatenormals/hcs/HCS_MAP_7100.pdf
[b] Support Center for Regulatory Atmospheric Modeling; http://www.epa.gov/scram001/tt24.htm.
[c] National Climatic Data Center CliMaps (NCDC-CliMaps) (2007). http://cdo.ncdc.noaa.gov/cgi-bin/climaps/climaps.pl

C-3.1.5 Aquatic Food Web

The aquatic food web is an important part of the screening scenario because the chemical concentrations modeled in fish are used to calculate human ingestion exposure and risks associated with eating contaminated fish. A biokinetic approach to modeling bioaccumulation in fish is used in the RTR screening scenario. The primary producers (first trophic level) in the TRIM.FaTE aquatic ecosystems are algae and macrophytes. The scenario includes a benthic invertebrate compartment to represent the primary invertebrate consumers (second trophic level) in the benthic environment, and the fish compartments represent the higher tropic levels in the aquatic system. For TRIM.FaTE to provide reasonable predictions of the distribution of a chemical across biotic and abiotic compartments in aquatic systems, the biomass of the aquatic biotic compartments must represent all biota in the system and the distribution of biomass among the trophic levels and groups must be as realistic as possible.

To support the development of a relatively generic freshwater aquatic ecosystem in which to model bioaccumulation in fish, a literature search, review, and analysis was conducted in support of developing and parameterizing aquatic biotic compartments for TRIM.FaTE (ICF 2005). This research demonstrated that the diversity of species and food webs across U.S. aquatic ecosystems is substantial, reflecting the wide range of sizes, locations, and physical/chemical attributes of both flowing (rivers, streams) and low-flow (ponds, lakes, reservoirs) waterbodies. In general, lotic bodies of water (lakes and ponds) are at a higher risk of accumulating contaminants in both sediments and biota than are flowing systems (rivers, streams). Also, the previous research (ICF 2005) suggested that a lake of at least 60 hectares (ha) likely would be sufficient to support higher trophic level predatory fish, with some fraction of their diet comprising smaller fish.

The RTR screening scenario includes a generic aquatic ecosystem with a 47-ha lake. Although slightly smaller than the size suggested by the previous review (ICF 2005), a 47-ha lake is large enough to support higher trophic level fish given the appropriate conditions. Also, this size was compatible with the overall size of the defined watershed. In the lake ecosystem defined for the screening scenario, benthic invertebrates are an important food source for a large proportion of the total fish biomass. The fish types, biomass, diet fractions, and body weights recommended for fish compartments for the screening scenario of TRIM.FaTE are listed in Exhibit 3-3. Biomass is based on an assumption that the total fish biomass for the aquatic ecosystem is 5.4 grams per square meter expressed as a wet weight (g_w/m^2, ICF 2005).

A strict piscivore compartment was not selected for the screening scenario because such species are rare in lakes of small to moderate size. In general, the food web implemented in the screening scenario is intended to be generally applicable across the United States and is intended to be generally conservative (to simulate a food web that maximizes bioaccumulation).

C-3.1.6 Using TRIM.FaTE Media Concentrations

The screening scenario outputs include average PB-HAP concentrations and deposition rates for each year and for each parcel of the model scenario. In each surface parcel, soil concentrations are provided for the surface, root, and vadose zones and grass or leaf concentrations as appropriate for the plants. Groundwater concentrations and deposition rates to the soil are also provided. For each air parcel, air concentrations are provided. For the lake, surface water concentrations and concentrations in the various levels of the aquatic food chain are included. For the ingestion exposure calculations, some concentrations are used to calculate direct exposure, and some are used to perform the farm food chain concentration calculations in the various media that humans can ingest (see Exhibit 2-4).

Exhibit 3-3. Parameters for Aquatic Biota for the Screening Scenario of TRIM.FaTE

TRIM.FaTE Compartment Type	Organisms Represented by Compartment	Biomass		Diet	Average Body Weight (kg)
		Areal density (g_w/m^2)	Fraction of Total Fish Biomass		
Macrophyte	hydrilla, milfoil	500	--	--	--
Benthic invertebrate	aquatic insects, crustacean, mollusks[a]	20	--	Feeds in sediment	0.000255
Water column (WC) herbivore	young-of-the-year, minnows	1.0	18.5%	100% algae[b]	0.025
Water column omnivore	sucker, carp	1.0	18.5%	40% WC herbivore 30% benthic invert. 30% macrophyte	0.25
Water column carnivore	largemouth bass, walleye	0.4	7.5%	60% WC omnivore 20% WC herbivore 20% benthic omniv.	2.0
Benthic omnivore	small catfish, rock bass	2.0	37%	100% benthic invert.	0.25
Benthic carnivore	large catfish	1.0	18.5%	70% benthic invert. 30% benthic omniv.	2.0
Total Fish Biomass [c]		**5.4**			

[a] Benthic invertebrates include aquatic insects (e.g., nymphs of mayflies, caddisflies, dragonflies, and other species that emerge from the water when they become adults), crustacea (e.g., amphipods, crayfish), and mollusks (e.g., snails, mussels).
[b] Algae is modeled as a phase of surface water in TRIM.FaTE.
[c] Total fish biomass does not include macrophytes or benthic invertebrates.

Regardless of whether the concentration and deposition values are used to calculate ingestion directly or are used in farm food chain calculations, selecting the parcel that is the source of the values used as inputs to succeeding calculations is necessary. The locations that determine direct and indirect exposures were selected assuming generally conservative assumptions. In general, decisions regarding which TRIM.FaTE outputs to use in calculating exposures for the hypothetical scenario assume exposure at locations near the modeled source, thereby resulting in higher exposures to emitted chemicals. These assumptions are summarized in Exhibit 3-4.

TRIM.FaTE can output instantaneous chemical concentrations for a user-specified time step and also can be configured to calculate temporal averages (e.g., annual averages). For the screening scenario, the model is set up to output results on a daily basis, largely because daily is the smallest time step over which input data change (i.e., wind direction and precipitation rate). Daily concentration results were averaged to obtain annual average concentrations. The default assumption is annual average concentrations for media during the fiftieth year of emissions. For the chemicals modeled in this scenario, long-term concentrations in environmental media will be relatively constant at 50 years (most of the chemicals modeled for RTR approach steady state well before 50 years).

Exhibit 3-4. Spatial Considerations – TRIM.FaTE Results Selected for Calculating Farm Food Chain Media Concentrations and Receptor Exposures

TRIM.FaTE Output Used in Exposure Calculations	Representative Compartment
Concentration in air, for uptake by plants via vapor transfer	Air compartment in air Parcel N2 (air over tilled soil)
Deposition rates, for uptake by farm produce	Deposition to surface soil compartment in surface Parcel N6 (tilled soil)
Concentration in surface soil, for incidental ingestion by humans and farm animals	Surface soil compartment in surface Parcel N1 (untilled soil, closest to facility)
Concentration in soil, for uptake by farm produce and animal feed	Surface soil compartment in surface Parcel N6 (tilled soil)
Concentration in fish consumed by angler	Water column carnivore compartment in lake (50% of fish consumed) and benthic omnivore in lake (50% of fish consumed)

C-3.2 Exposure and Risk Calculations

This section describes the approach for modeling chemical concentrations in farm food chain (FFC) media (Section C-3.2.1); estimating human exposures associated with ingestion of FFC media, incidental ingestion of soil, consumption of fish, and infant consumption of breast milk (Section C-3.2.2); and calculating human health screening risk metrics associated with these exposure pathways (Section C-3.2.3). All of these calculations are conducted using the MIRC modeling software. For this multipathway screening evaluation, partitioning into FFC media is calculated with the same data set used to model exposure and risk, rather than as a component of the TRIM.FaTE modeling scenario. Consequently, processes and inputs related to estimating chemical levels in FFC media are summarized in this section and discussed in detail in Attachment 2.

C-3.2.1 Calculating Concentrations in Farm Food Chain Media

As was shown in Exhibit 2-5, MIRC was compiled to calculate concentrations of PB-HAPs in foodstuffs that are part of the farm food chain. The FFC media included in this screening scenario are as follows:

- exposed and protected fruit,
- exposed and protected vegetables,
- root vegetables,
- beef,
- total dairy products,
- pork, and
- poultry and eggs.

The algorithms used in MIRC were obtained from EPA's *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities* (HHRAP; EPA 2005a). These algorithms model the transfer of concentrations of PB-HAPs in FFC media using empirical biotransfer factors. As noted in Section C-1 this report, the algorithms involving ingestion exposure to be included eventually in TRIM are expected to be very similar to those presented in HHRAP. Environmental media concentrations (i.e., the chemical source terms in these algorithms) were

obtained from TRIM.FaTE. As noted in Section C-3.1.6, the TRIM.FaTE outputs included as inputs to MIRC are the following:

- PB-HAP concentrations in air;
- air-to-surface deposition rates for PB-HAPs in both particle and vapor phases;
- PB-HAP concentrations in groundwater (used as drinking water);
- PB-HAP concentrations in fish tissue for fish in trophic levels three and four [T3 and T4]); and
- PB-HAP concentrations in surface soil and root zone soil.

In general, plant- and animal-specific parameter values, including chemical-specific transfer factors for FFC media, were obtained from the Hazardous Waste Companion Database included in HHRAP (EPA 2005a). A list of variables and PB-HAP-specific input parameters, along with the input values used in this screening scenario, are provided in Attachment 2.

C-3.2.2 Ingestion Exposure Assessment

C-3.2.2.1 Ingestion Exposure Pathways and Routes of Uptake

MIRC was used to estimate ingestion rates as average daily doses (ADDs) normalized to body weight for a range of exposure pathways. Exposure pathways included are incidental ingestion of soil and consumption of fish, produce, and farm animals and related products. The ingestion exposure pathways included in the screening evaluation and the environmental media through which these exposures occur are summarized in Exhibit 3-5.

C-3.2.2.2 Exposure Scenarios and Corresponding Inputs

Specific exposure scenarios are developed by defining the ingestion activity patterns (i.e., estimating how much of each medium is consumed and the fraction of the consumed medium that is grown in or obtained from contaminated areas) and the characteristics of the hypothetical human exposed (e.g., age and body weight). MIRC computes exposure doses and risks for each ingestion pathway separately, enabling the pathway(s) of interest for each PB-HAP to be determined. Data related to exposure factors and receptor characteristics were obtained primarily from EPA's *Exposure Factors Handbook* (EPA 1997a).

For the screening scenario described here, exposure characteristics were selected that result in a highly conservative estimate of total exposure. The ingestion rate for each medium was set equal to the 90th percentile of the distribution of national data for that medium. All media were assumed to be obtained from locations impacted by the modeled source. Although this approach results in an *overestimate* of *total* chemical exposure for a hypothetical exposure scenario (for example, note that the total food ingestion rate that results is extremely high for a hypothetical consumer with ingestion rates in the 90th percentile for every farm food type), it was selected to *avoid underestimating* exposure for any *single farm food type*. The exposure characteristics selected for the ingestion screening scenario are summarized in Exhibit 3-6. Quantitative input values corresponding to these parameters are presented in Attachment 2.

Exhibit 3-5. Summary of Ingestion Exposure Pathways and Routes of Uptake

Ingestion Exposure Pathway	Medium Ingested	Intermediate Exposure Pathway – Farm Animals [a]	Environmental Uptake Route	
			Medium	Process [b]
Incidental ingestion of soil	Surface soil	N/A	Surface soil	Deposition; transfer through plants; transfer via erosion and runoff [c]
Consumption of fish	Fish from local water body	N/A	Fish tissue	Direct uptake from water and consumption of food compartments modeled in TRIM.FaTE [c]
Consumption of breast milk [d]	Breast milk	N/A	Breast milk	Ingested by mother and then partition to breast milk
Consumption of produce	Aboveground produce, exposed fruits and vegetables	N/A	Air Air Soil	Deposition to leaves/plants Vapor transfer Root uptake
	Aboveground produce, protected fruits and vegetables	N/A	Soil	Root uptake
	Belowground produce	N/A	Soil	Root uptake
Consumption of farm animals and related food products	Beef	Ingestion of forage	Air Air Soil	Direct deposition to plant Vapor transfer to plant Root uptake
		Ingestion of silage		
		Ingestion of grain	Soil	Root uptake
		Ingestion of soil	Soil	Ingestion from surface
	Dairy (milk)	Ingestion of forage	Air Air Soil	Direct deposition to plant Vapor transfer to plant Root uptake
		Ingestion of silage		
		Ingestion of grain	Soil	Root uptake
		Ingestion of soil	Soil	Ingestion from surface
	Pork	Ingestion of silage	Air Air Soil	Direct deposition to plant Vapor transfer to plant Root uptake
		Ingestion of grain	Soil	Root uptake
		Ingestion of soil	Soil	Ingestion from surface
	Poultry	Ingestion of grain	Soil	Root uptake
		Ingestion of soil	Soil	Ingestion from surface
	Eggs	Ingestion of grain	Soil	Root uptake
		Ingestion of soil	Soil	Ingestion from surface

[a] Calculation of intermediate exposure concentrations were required only for the farm animal/animal product ingestion pathways.
[b] Process by which HAP enters medium ingested by humans.
[c] Modeled in TRIM.FaTE.
[d] The consumption of breast milk exposure scenario is discussed in Section C-3.2.2.4.

Exhibit 3-6. Overview of Exposure Factors Used for RTR Multipathway Screening [a,b]

Exposure Factor	Selection for Screening Assessment
Age group evaluated	Infants under 1 year (breast milk only) Children 1 to 2 years of age Children 3 to 5 years of age Children 6 to 11 years of age Children 12 to 19 years of age Adult (20 to 70 years)
Body weight (BW; varies by age)	Weighted mean of national distribution
Intake rate and ingestion rate (IR) for farm produce and animal products (varies by age and media consumed)	90th percentile of distribution of consumers who produce own food
Ingestion rate for fish	17 g/day (approximately 90th percentile of general population; also equal to mean value for anglers); lower for children
Exposure frequency (EF)	365 days/year
Exposure duration	Lifetime, for estimating cancer risk; varies by chemical for chronic non-cancer evaluation
Fraction contaminated (FC) (varies by media consumed)[c]	1
Cooking loss [d]	Assumed to be "typical;" varies depending on food product (see Attachment 2)

[a] Values for exposure characteristics are presented in Attachment 2. Exposure parameter values were based on data obtained from the *Exposure Factors Handbook* (EPA 1997a). See Attachment 2 for details.
[b] Exposure factor inputs are used in calculating average daily dose (ADD) estimates for each exposure pathway. ADD equations for each pathway evaluated in this screening assessment are provided in Attachment 2.
[c] Fraction contaminated represents the fraction of food product that is derived from the environment included in the screening scenario (e.g., produce grown on soil impacted by PB-HAPs). This parameter is defined separately for each FFC medium.
[d] Cooking loss inputs were included to simulate the amount of a food product that is not ingested due to loss during preparation or cooking, or after cooking.

C-3.2.2.3 Calculating Average Daily Doses

MIRC calculates chemical-specific ADDs of chemicals normalized to body weight (mg PB-HAP per kg of body weight per day). Equations used to calculate ADDs were adapted from the algorithms provided in the technical documentation of EPA's *Multimedia, Multipathway, and Multireceptor Risk Assessment (3MRA) Modeling System* (EPA 2003), which derived much of its input data from the *Exposure Factors Handbook* (EPA 1997a). The ingestion exposure modeling approach embodied by 3MRA is conceptually similar to that presented in HHRAP. A discussion of exposure dose estimation is provided in Attachment 2. The equations to calculate ADDs for each ingestion pathway are provided in Attachment 2.

C-3.2.2.4 Infant Ingestion of Breast Milk

A nursing mother exposed to contaminants by any ingestion pathway described above can pass the contaminants on to her infant through breast milk (EPA 1998). The nursing infant's exposure can be estimated from the levels of chemical concentrations in the breast milk, which in turn can be estimated based on the mother's chemical intake. Exposures can occur for infants via this pathway for dioxins and possibly also for mercury.

Exposure to dioxin and mercury via breast milk consumption during the first year of life is expected to have a small effect on the estimated lifetime ADD and on the individual's excess lifetime cancer risk for dioxins or the highest chronic HQ for either chemical. Therefore,

exposures to these chemicals via the breast milk pathway were *not* considered in developing the *de minimis* emission thresholds for dioxins and mercury. The potential for non-cancer health effects (e.g., developmental effects) is of greater concern for nursing infants exposed to either chemical during the first year of life. These exposures will be considered in more detail for facilities and emissions that do not pass the initial screen.

C-3.2.3 Risk Characterization

MIRC was used to calculate excess lifetime cancer risk (ELCR) and non-cancer hazard (expressed as an HQ) using the calculated ADDs and ingestion dose-response values. Chemical dose-response data include CSFs for ingestion and non-cancer oral RfDs for chronic exposures. The CSFs and RfDs for the PB-HAPs included in this screening scenario are presented in Exhibit 3-7 and are discussed in more detail in Attachment 2. Equations used to estimate cancer risk and non-cancer hazard are provided in Attachment 2.

Estimated individual cancer risks for the PAHs included in the screening scenario were adjusted upward to account for the higher mutagenic cancer potency of these compounds during childhood, as specified by EPA in supplemental guidance for cancer risk assessment (EPA 2005c). Specifically, cancer potency is assumed to be tenfold greater for the first 2 years of life and threefold greater for the next 14 years for PAHs. These factors were incorporated into a time-weighted total increase in potency over a lifetime of 70 years. The cancer potency adjustment for chemicals with a mutagenic mode of action is discussed in detail in Attachment 2.

Exhibit 3-7. Dose-response Values for PB-HAPs Addressed by the Screening Scenario

PB-HAP	CSF ($[mg/kg\text{-}day]^{-1}$)	Source	RfD (mg/kg-day)	Source
Inorganics				
Cadmium Compounds	not available		5E-4	IRIS
Elemental Mercury	NA		not available	
Divalent Mercury	not available		3E-4	IRIS
Methyl Mercury	not available		1E-4	IRIS
Organics [a]				
Benzo[a]pyrene	10	IRIS	not available	
2,3,7,8-TCDD	1.5E+5	EPA ORD	1E-9	ATSDR

Source: EPA OAQPS 2007
CSF = cancer slope factor; RfD = reference dose; IRIS = Integrated Risk Information System; CalEPA = California Environmental Protection Agency; ASTDR = Agency for Toxic Substances and Disease Registry; EPA ORD = EPA's Office of Research and Development
NA = not applicable
[a] The CSF listed in IRIS for benzo[a]pyrene was adjusted for consistency with the overall approach for dose-response assessment of PAHs; see Attachment 2.

C-3.2.4 Dermal Risk Screening

Non-inhalation exposure to PB-HAPs can occur by way of the dermal pathway through contact with PB-HAP-contaminated soil and water. However, dermal absorption of chemicals that are originally airborne is generally a relatively minor pathway of exposure compared to other exposure pathways (EPA 2006, CalEPA 2000). This section demonstrates that for the

conservative screening scenario developed for RTR multipathway evaluation, the dermal exposure route is not a significant risk pathway when compared to the ingestion pathway. In general, the assessment followed the protocol for evaluating a reasonable maximum exposure as described in EPA's *Risk Assessment Guidance for Superfund (RAGS), Volume I: Human Health Evaluation Model, Part E, Supplemental Guidance for Dermal Risk Assessment* (EPA 2004c).

C-3.2.4.1 Hazard Identification and Dose Response Assessment

To assess the potential contribution of dermal exposure to non-inhalation exposure, we evaluated the potential for cancer and chronic non-cancer effects for the four PB-HAPs currently assessed in the multipathway screening evaluation for RTR: cadmium, divalent mercury, 2,3,7,8-TCDD, and benzo(a)pyrene. EPA has not developed carcinogenic potency slope factors (CSFs) and non-cancer reference doses (RfDs) specifically for evaluating potential human health concerns associated with dermal exposure to PB-HAPs. Instead, dermal toxicity values can be derived from oral toxicity values via route-to-route extrapolation by adjusting for gastrointestinal (GI) absorption. EPA recommends making this adjustment only when GI absorption of the chemical is significantly less than 100% (i.e., less than 50 percent). Otherwise, a default value of complete (100 percent) oral absorption is assumed, and no adjustment is made (EPA 2004c).

The absorbed cancer slope factor (CSF_{ABS}) is based on the oral cancer slope factor (CSF_O) and the fraction of the contaminant absorbed in the gastrointestinal track (ABS_{GI}), as follows:

$$CSF_{ABS} = \frac{CSF_o}{ABS_{GI}}$$

where:

CSF_{ABS} = Absorbed slope factor $(mg/kg\text{-}day)^{-1}$

CSF_o = Oral slope factor $(mg/kg\text{-}day)^{-1}$

ABS_{GI} = Fraction of chemical absorbed in gastrointestinal tract (unitless)

The absorbed reference dose (RfD_{ABS}) is based on the oral reference dose (RFD_O) and the fraction of the contaminant absorbed in the gastrointestinal track (ABS_{GI}), as shown below.

$$RfD_{ABS} = RfD_o \times ABS_{GI}$$

where:

RfD_{ABS} = Absorbed reference dose (mg/kg-day)

RfD_o = Oral reference dose (mg/kg-day)

$ABSGI$ = Fraction of chemical absorbed in gastrointestinal tract (unitless)

GI absorptions for 2,3,7,8-TCDD and all polycyclic aromatic hydrocarbons (PAHs) (which includes benzo[a]pyrene) were estimated to be greater than 50 percent. Therefore, as shown in Exhibit 3-8, no adjustments to the available oral CSFs were required. Similarly, no adjustment to the oral RfD for 2,3,7,8-TCDD was required. For cadmium and divalent mercury, adjustments

were made based on absorption data provided in RAGS Part E, Exhibit 4-1. The RfDs for dermal exposure to cadmium and divalent mercury are also shown in Exhibit 3-8.

Exhibit 3-8. Cancer Slope Factors and Reference Doses Based on Absorbed Dose

PB-HAP	ABS_{GI} (unitless)	CSF_{ABS} [a] $(mg/kg\text{-}day)^{-1}$	RfD_{ABS} [a] (mg/kg-day)
Cadmium Compounds	0.05	NA	2.5E-05
Divalent Mercury	0.07	NA	2.1E-05
2,3,7,8-TCDD	No adjustment required [b]	1.5E+05	1.0E-09
Benzo[a]pyrene	No adjustment required [b]	1.0E+01	NA

NA = Not applicable
[a] Oral dose response values are presented in Exhibit 3-7. Only the resulting adjusted dose response values are presented in this table.
[b] According to RAGS Part E, Exhibit 4-1, GI absorption is expected to be greater than 50%.

C-3.2.4.2 Dermal Exposure Estimation

Dermal exposures and risks resulting from absorption of the chemical through the skin from contact with contaminated water and soil were evaluated for the RTR screening scenario. Individuals were assumed to be exposed on a fraction of their bodies (i.e., their head, forearms, hands, lower legs, and feet) to contaminated soil from the TRIM.FaTE surface soil parcel with the highest concentration (N1) on a daily basis. For the water evaluation, individuals were assumed to be exposed to contaminated surface water with the same PB-HAP concentration as the TRIM.FaTE screening scenario lake over their entire bodies on a daily basis.

Equations for Estimating Dermal Exposure

The general equation used to estimate dermal absorbed dose (DAD) for water or soil is shown below, and is expressed in milligrams of PB-HAP per kilogram of receptor body weight per day (mg/kg-day). DAD is calculated separately for the water and soil pathways.

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

where:

DA_{event} = Absorbed dose per event; chemical-specific; equation for DA_{event} also differs depending on water or soil contact (mg/cm^2-event)

EV = Event frequency (events/day)

ED = Exposure duration (years)

EF = Exposure frequency (days/year)

SA = Skin surface area available for contact (cm^2)

BW = Body weight (kg)

AT = Averaging time; for non-cancer effects, equals ED x 365 days/year; for cancer effects, equals 70 years x 365 days/year (days)

DA_{event} is estimated to be the total dose absorbed through the skin at the end of exposure and the equation for calculation is different for organic and inorganic chemicals in water and for soil. The equations for calculating these chemical-specific DA_{event} values for water contact are provided in RAGS Part E, Chapter 3 (see Equations 3.2 – 3.4). For soil, the equation for calculating these chemical-specific DA_{event} values is provided in RAGS Part E, Chapter 3 (see Equation 3.12).

Water – Organic Chemicals: $$DA_{event} = C_w \times 2 \times FA \times K_p \sqrt{\frac{6 \times \tau_{event} \times t_{event}}{\pi}}$$

Water – Inorganic Chemicals: $$DA_{event} = C_w \times K_p \times t_{event}$$

Soil – All Chemicals: $$DA_{event} = C_s \times AF \times ABS \times CF$$

where:

DA_{event}	=	Absorbed dose per event (mg/cm^2-event)
C_w C_s	=	Chemical concentration in water (mg/cm^3) or soil (mg/kg)
K_p	=	Chemical-specific dermal permeability coefficient of compound in water (cm/hr)
FA	=	Chemical-specific fraction absorbed; accounts for loss due to the regular shedding of skin cells of some chemical originally dissolved into skin (unitless)
τ_{event}	=	Chemical-specific lag time per event (hr/event)
t_{event}	=	Receptor-specific event duration (hr/event)
AF	=	Receptor- and activity-specific adherence factor of soil to skin (mg/cm^2-event)
ABS	=	Chemical-specific dermal absorption fraction (unitless)
CF	=	Conversion factor (10^{-6} kg/mg)

Exposure Factors and Assumptions

The exposure parameters included in this assessment and their default and other value options are summarized in this subsection. Default values were selected to result in a highly conservative estimated of exposure (i.e., exposures are likely overestimated). Parameter values were primarily obtained or estimated from RAGS Part E (EPA 2004c) and the CSEFH (EPA 2008). Receptor-and scenario-specific exposure assumptions are discussed first, and a discussion of chemical-specific parameters values follows. Estimated water and soil exposure concentrations are presented at the end of this subsection.

Receptor-Specific Parameters

Dermal exposures and risks were estimated for the same age groups used in the ingestion exposure assessment: adults (ages 20 to 70 years) and five child age groups: <1 year; 1 to 2 years; 3 to 5 years; 6 to 11 years; and 12 to 19 years. The body weight values used in the ingestion exposure assessment were used in the dermal exposure assessment.

Body surface areas for water and soil exposures for adults were calculated using Appendix C, Exhibit C-1, of RAGS Part E. For children, SAs for water and soil exposures for the five children's age groups were estimated using Tables 7-1 and 7-2 of the CSEFH, respectively. For SA (water), individuals were assumed to shower or bathe in the water with 100 percent of their body exposed. For SA (soil), it was assumed that individuals were exposed on a fraction of their total body, specifically their head, forearms, hands, lower legs, and feet. Based on

information provided in RAGS Part E, the SA for forearms was calculated using the SA for arms and assuming a forearm-to-arm ratio of 0.45, and the SA for lower legs was estimated using the SA for legs and assuming a lower leg-to-leg ratio of 0.4.

Values for body SA by age group are summarized in Exhibit 3-9.

Exhibit 3-9. Receptor-Specific Body Surface Area Assumed to be Exposed to Chemicals

Age Group [a] (years)	Surface Area for Water Exposure (cm^2)	Surface Area for Soil Exposure (cm^2)
Adult 20-70	18,150 [g]	6,878 [h]
Child <1 [b]	3,992	1,772
Child 1-2 [c]	5,700	2,405
Child 3-5 [d]	7,600	3,354
Child 6-11 [e]	10,800	4,501
Child 12-19 [f]	17,150	6,906

[a] Sources for the child groups included Table 7-1 (total body surface area for SA-Water), and Table 7-2 (fraction of total body surface area for SA-Soil) of the 2008 CSEFH.
[b] Represents a time-weighted average for age groups birth to <1 month, 1 to <3 months, 3 to <6 months, and 6 to <12 months.
[c] Represents a time-weighted average for age groups 1 to <2 years and 2 to <3 years.
[d] Values for age group 3 to <6 years in the 2008 CSEFH.
[e] Values for age group 6 to <11 years in the 2008 CSEFH. Represents a conservative (i.e., slightly low) estimate for ages 6 through 11 years since 11-year olds are not included in this CSEFH age group.
[f] Represents a time-weighted average for age groups 11 to <16 years and 16 to <21 years. Note that estimated values include 11-year-olds and individuals through age 20, which contributes to uncertainty in the estimates for 12 to 19 years.
[g] Represents the average total surface area of adults from Table C-1 of RAGS Part E.
[h] Represents the average surface area of adults for head, forearms, hands, lower legs, and feet from Table C-1 of RAGS Part E.

Scenario-Specific Parameters

Exhibit 3-10 summarizes the exposure values related to frequency and duration of contact. In general, these are the recommended defaults for calculating a reasonable maximum exposure (RME) for a residential scenario as proposed by EPA in RAGS Part E, Chapter 3.

Exhibit 3-10. Scenario-Specific Exposure Values for Water and Soil Contact

Exposure Parameter	Receptor	Value	Source
Water Contact			
Event Duration (t_{event}) (hr/event)	Child	1	Reasonable maximum exposure scenario for showering/bathing from RAGS Part E, Exhibit 3-2
	Adult	0.58	
Soil Contact			
Soil Adherence Factor (AF) (mg/cm^2)	Child	0.2	For children, value is geometric mean value for children playing (wet soil) and for adults, value is geometric mean value for an adult farmer from RAGS Part E, Exhibit 3-3
	Adult	0.1	
Both Media			
Event Frequency (EV) (events/day)	All	1	Reasonable maximum exposure scenario from RAGS Part E, Exhibits 3-2 & 3-5.
Exposure Frequency (EF) (days/year)	All	350	
Exposure Duration (ED) (years)	Child <1	1	Represents the number of years included in the age group; also used in ingestion exposure calculations.
	Child 1-2	2	
	Child 3-5	3	
	Child 6-11	6	
	Child 12-19	8	
	Adult 20-70	50	
Averaging Time (AT) (days)	For cancer assessment, an AT equal to a lifetime (70 years) x 365 days/year is used. Same value used in ingestion exposure calculations. For non-cancer assessment, an AT equal to the exposure duration (ED) x 365 days/year is used, so AT will vary by receptor group. Same value used in ingestion exposure calculations.		

Chemical-Specific Parameters

The chemical-specific parameters required to quantitatively evaluate dermal pathway exposures are listed in Exhibit 3-11. For the water concentration in the dermal analysis, the modeled TRIM.FaTE chemical concentration in the screening scenario pond at the *de minimis* emission rate was used. For the soil concentration, the modeled TRIM.FaTE chemical concentration in surface soil in parcel N1 of the screening scenario at *de minimis* emission rate. This same soil concentration was also used in ingestion exposure calculations for soil ingestion.

Dermal absorption of chemicals in water is based on the use of a dermal permeability coefficient (K_p), which measures the rate that a chemical penetrates the skin. Dermal absorption of soil-bound chemicals is based on the use of a dermal absorption fraction (ABS), which is a measure of how much of a chemical the skin absorbs through contact with soil.

Exhibit 3-11. Chemical-Specific Dermal Exposure Values for Water and Soil Contact

PB-HAP	Cadmium	Divalent Mercury	2,3,7,8-TCDD	Benzo[a] pyrene	Source
Chemical concentration in Water (Cw) (mg/cm3)	4.77E-07	1.81E-07	6.20E-17	2.03E-12	TRIM.FaTE modeled concentration in screening scenario pond
Chemical concentration in Soil (Cs) (mg/kg)	1.37E+00	5.94E+00	5.36E-09	1.21E-03	TRIM.FaTE modeled concentration in surface soil in parcel N1 in screening scenario
Permeability coefficient in water (Kp) (cm/hour)	0.001	0.001	0.81	0.7	Values from RAGS Part E, Exhibits B-3 (organics) and B-4 (inorganics)
Fraction absorbed water (FA) (unitless)	NA	NA	0.5	1.00	Values from RAGS Part E, Exhibits B-3; only used for organic chemicals
Lag time per event (τevent) (hr/event)	NA	NA	6.82	2.69	Values from RAGS Part E, Exhibits B-3; only used for organic chemicals
Dermal absorption fraction (ABS) from soil (unitless)	0.001	0.045 [a]	0.03	0.13	Values from RAGS Part E, Exhibit 3-4, unless otherwise noted

[a] Value obtained from Bioavailability in Environmental Risk Assessment (Hrudey et al. 1996).

C-3.2.4.3 Screening-Level Cancer Risks and Non-Cancer Hazard Quotients

Toxicity values were used in conjunction with exposure information to evaluate the potential for cancer risks and non-cancer health hazards. Risk estimation methods are presented below.

Dermal Cancer Risk

Cancer risk for the dermal route was calculated as the product of the age-specific *DAD*s and the absorbed CSF for each chemical, as follows:

$$Dermal\ Cancer\ Risk = DAD \times CSF_{ABS}$$

where:

DAD = Dermal Absorbed Dose (mg/kg-day)

CSF_{ABS} = Absorbed cancer slope factor $(mg/kg\text{-}day)^{-1}$

Lifetime dermal cancer risks were calculated for 2,3,7,8-TCDD and benzo[a]pyrene. The total risk accounts for dermal exposures that an individual might receive from these PB-HAPs in water plus soil over his or her lifetime (70 years).

Dermal Hazard Quotient

Dermal hazard quotient (HQ) was estimated as the ratio of age-specific *DAD*s to the absorbed RfD for each chemical, as shown below:

$$Dermal\ HQ = DAD / RfD_{ABS}$$

where:

DAD = Dermal Absorbed Dose (mg/kg-day)

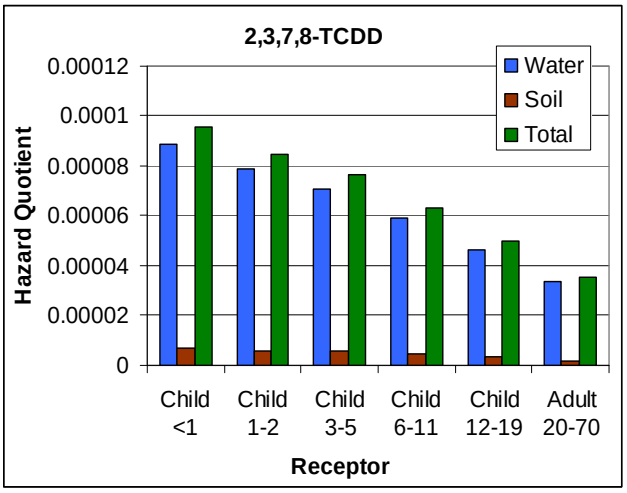
RfD_{ABS} = Absorbed reference dose (mg/kg-day)

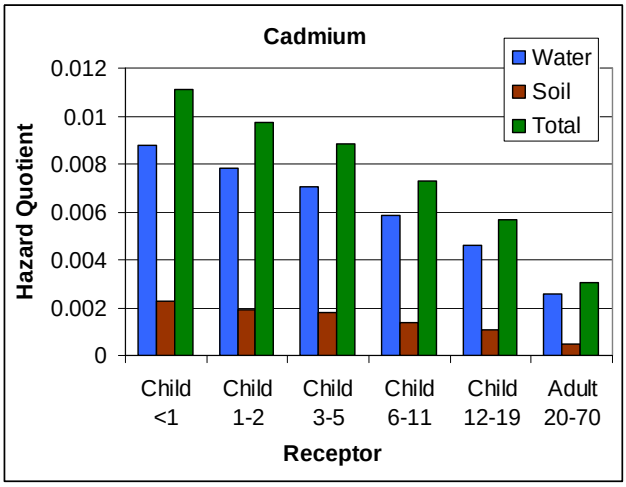
The aggregate HQ accounts for exposures that an individual in a receptor group may receive from the PB-HAP in water and soil over the exposure duration. Non-cancer hazard is not additive *across* the age groups evaluated here.

C-3.2.4.4 Dermal Screening Results

Exhibit 3-12 presents a summary of estimated dermal non-cancer hazards by age group. A summary of estimated lifetime dermal cancer risks is provided in Exhibit 3-13. All HQ values were 0.5 (representing divalent mercury exposure for children less than 1 year of age) or less, a factor of at least two smaller than the potential ingestion hazard quotients associated with the screening scenario. The highest estimated individual lifetime cancer risk associated with potential dermal exposures was 3.33E-8 for benzo[a]pyrene; this value is a factor of 30 times smaller than the ingestion risk estimated for the same *de minimis* emission rate.

Exhibit 3-12. Summary of Dermal Non-Cancer Hazards





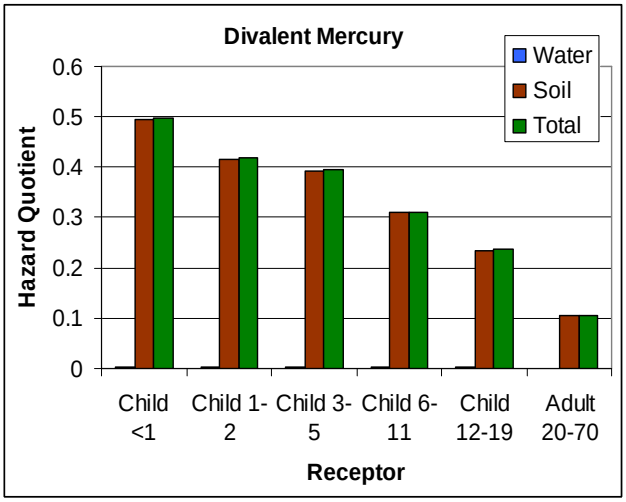


Exhibit 3-13. Summary of Dermal Cancer Risks

PB-HAP	Dermal Lifetime Cancer Risk	Magnitude of Difference
2,3,7,8-TCDD		
Water	6.14E-09	>160
Soil	3.48E-10	>2800
Total	6.49E-09	>150
Benzo[a]pyrene		
Water	1.06E-08	>90
Soil	2.27E-08	>40
Total	3.33E-08	>30

Based on these results and taking into consideration the extremely conservative nature of the dermal exposure calculations, it was assumed that it is not necessary to incorporate dermal exposures in calculating multipathway *de minimis* levels. Specifically, the daily exposure durations of 0.58 hour for adults and 1 hour for children used to calculate dermal exposure from water are highly conservative and assume that the individual is bathing in surface water taken directly from a contaminated lake or is swimming in the lake for 350 days of the year. The exposure frequency of 350 days and corresponding skin surface area available for contact with contaminated soils (i.e., head, hands, arms, legs, and feet) likely also grossly overestimates dermal exposure to soil.

C-3.3 Summary of Scenario Assumptions

As emphasized previously, the screening scenario created for evaluating PB-HAP emissions from RTR facilities is intended to be generally conservative to prevent underestimating risk. The overall degree of conservatism of the scenario is the sum total of the multiple assumptions that affect the outputs of the fate and transport, exposure, and risk modeling. Exhibit 3-14 summarizes important characteristics that influence exposure and risk estimates for this scenario and indicates the general degree of conservatism associated with the values for each assumption. Although this summary does not provide a quantitative estimate of the output uncertainty or the degree to which exposures and risks estimated using the scenario would be overestimated, it does demonstrate qualitatively that the scenario generally overestimates exposure and thus favors a health-protective risk output.

Exhibit 3-14. Summary of RTR Screening Scenario Assumptions and Associated Conservatism

Characteristic	Value	Conservatism	Comments on Assumptions
General Spatial Attributes			
Farm location	375 m from source; generally downwind	Conservative	Location dictates soil and air concentrations and deposition rates used to calculate chemical levels in farm produce.
Lake location	375 m from source; generally downwind	Conservative	Location dictates where impacted fish population is located.
Surface soil properties	Typical values or national averages	Neutral	Based on existing EPA documentation and other references.
Size of farm parcel	About 4 ha	Conservative	Relatively small parcel size results in higher chemical concentration.
Size of lake	46 ha; about 3 m average depth	Conservative	Lake is large enough to support an aquatic ecosystem with higher trophic level fish, but is relatively small and shallow (thus increasing surface area-to-volume ratio).
Meteorological Inputs			
Total precipitation	1.5 m/yr	Conservative	Intended to represent rainy U.S. location; set to highest state-wide average for the contiguous U.S.
Precipitation frequency (with respect to impacted farm/lake)	2/3 of total precipitation fall on farm/lake and watershed	Conservative	Most of total precipitation occurs when the farm/lake are downwind of the source.
Wind direction	Farm/lake are downwind 40% of the time	Conservative	Farm/lake located in the predominantly downwind direction. Temporal dominance of wind direction based on data from Yakima, WA, where wind is predominantly from the west.
Wind speed	2.8 m/sec	Conservative	Low wind speed (5^{th} percentile of long-term averages for contiguous U.S.); increases net deposition to lake/watershed.
Air temperature	298 K	Neutral	Typical for summer temperatures in central and southern U.S.
Mixing height	710 m	Conservative	Relatively low long-term average mixing height (5^{th} percentile of long-term averages for contiguous U.S.); increases estimated air concentration.
Watershed and Water Body Characteristics			
Evaporation of lake surface water	700 mm/yr	Neutral	Value is representative of cooler climates.
Surface runoff into lake	Equal to 40% of total precipitation	Conservative	Based on typical water flow in wetter U.S. locations; higher runoff results in greater transfer of chemical to lake.

Exhibit 3-14. Summary of RTR Screening Scenario Assumptions and Associated Conservatism

Characteristic	Value	Conservatism	Comments on Assumptions
Surface water turnover rate in lake	About 12 turnovers per year	Neutral	Consistent with calculated water balance; reasonable in light of published values for small lakes. May overestimate flush rate if water inputs are also overestimated. Note that after evapotranspiration, remaining water volume added via precipitation is assumed to flow into or through lake.
Soil erosion from surface soil into lake	Varies by parcel; ranges from 0.002 to 0.01 kg/m^2-day	Neutral	Erosion rates were calculated using the universal soil loss equation (USLE); inputs to USLE were selected to be generally conservative with regard to concentration in the pond (i.e. higher erosion rates were favored). May underestimate erosion for locations susceptible to high erosion rates. Note that higher erosion increases concentration in lake (and fish) but decreases levels in surface soil (and farm products).
Aquatic food web structure and components	Multi-level; includes large, upper trophic level fish	Conservative	Inclusion of upper-trophic level fish and absence of large-bodied herbivore/detritivore fish favor higher concentrations of bioaccumulative chemicals and result in higher concentrations in consumed fish.
Parameters for Estimating Concentrations in Farm Food Chain Media			
Fraction of plants and soil ingested by farm animals that is contaminated	1.0 (all food and soil from contaminated areas)	Conservative	Assumes livestock feed sources (including grains and silage) are all derived from most highly impacted locations.
Soil- and air-to-plant transfer factors for produce and related parameters	Typical (see Attachment 2 for details)	Neutral	Obtained from peer-reviewed and standard EPA reference sources.
Biotransfer factors for efficiency of uptake by animal of chemical in food/soil	Typical (see Attachment 2 for details)	Neutral	Obtained from peer-reviewed and standard EPA reference sources.
Bioavailability of chemicals in soil (for soil ingested by animals)	1.0 (relative to bioavailability of chemical in plant matter)	Conservative	Probably overestimates bioavailability in soil; many chemicals are less bioavailable in soil than in plants.
Ingestion Exposure Assumptions			
Ingestion rates for all farm produce/livestock types	Person obtains all food sources from local farm; ingestion rate is 90th percentile of rates for home-produced food items	Conservative	All food derived from impacted farm; total food ingestion rate would exceed expected body weight-normalized ingestion rates (prevents underestimating any individual food type).

Exhibit 3-14. Summary of RTR Screening Scenario Assumptions and Associated Conservatism

Characteristic	Value	Conservatism	Comments on Assumptions
Fish ingestion rate	0.24 g/kg-day for adult; between 0.14 and 0.26 g/kg-day for children aged 1-19	Conservative	Rates are based on EPA's analysis of freshwater and estuarine fish consumption from the USDA's Continuing Survey of Food Intake by Individuals (2002d). This likely overestimates long-term fish consumption rates for the general population. See Attachment C-2 for a detailed discussion.
Exposure frequency	Consumption of contaminated food items occurs 365 days/yr	Conservative	All meals from local farm products.
Body weight	Mean of national distribution	Neutral	Note that this does not affect the body weight-normalized rates for produce and animal products.
Other Chemical-Specific Characteristics			
General chemical properties used in fate and transport modeling (Henry's law, K_{ow}, etc.)	Varies	Neutral	Obtained from peer-reviewed sources; intended to be representative of typical behavior and characteristics.
"General" physical properties (plant matter density, aquatic life biomass, algal growth rate, etc.)	Varies	Neutral	Obtained from peer-reviewed sources; intended to be representative of typical behavior and characteristics.
Dose-response values	Varies	Neutral to conservative	Values used are those determined to be appropriate for risk assessment by OAQPS; some values may be health-protective.

C-4 Evaluation of Screening Scenario

C-4.1 Overview

To evidence our understanding of the models used, their configuration, and the total uncertainty associated with this model application, the screening scenario developed for RTR was subjected to a series of evaluations. These analyses were somewhat more focused on TRIM.FaTE, given the complexity and variability associated with the fate and transport modeling phase relative to other aspects of the screening calculations. These evaluations emphasized the application of this approach (and especially TRIM.FaTE) in the context of ingestion exposure and risk screening for RTR. The analyses were not intended to be general model performance evaluations of TRIM.FaTE. EPA has conducted such analyses, which are documented in detail in Volumes I and II of the *TRIM.FaTE Evaluation Report* (EPA 2002b, 2005b).

The purpose of the current evaluations, however, is similar to that of the previous model evaluations, in that both are intended to increase the confidence associated with model performance. Although the focus of the screening scenario evaluation (assessing the utility of the constructed modeling scenario) differs from the purpose of TRIM.FaTE model evaluations, the underlying objectives are comparable. In summary, the current evaluations are intended to achieve several objectives:

- enhance understanding of how the scenario operates;
- better characterize the uncertainty of model results;
- measure model sensitivity to changes in parameter values and scenario configuration; and
- strengthen the defensibility of the scenario's application as a component of air toxics residual risk assessment.

That these analyses do not attempt to validate or "prove" the accuracy of model results is important to note. As specified in the TRIM.FaTE mercury test case evaluation report (EPA 2005b):

> 'Validation' of such models, in the classic sense (e.g., proving the model produces accurate results across a range of input conditions), is not generally possible, in part because there are no comprehensive data sets of measured chemical concentrations (and associated contributing pollutant sources) for use in such comprehensive studies, nor are there other fully validated multimedia models against which TRIM.FaTE can be benchmarked. Thus, evaluation of TRIM.FaTE is not a yes/no exercise but a continuing accumulation of evidence leading to model refinement and eventually to increasing levels of confidence in the model results.

The screening scenario was evaluated through a series of analyses:

- a general evaluation of TRIM.FaTE fate and transport modeling outputs;
- comparisons of model outputs to the literature (e.g., measured concentrations of the chemicals evaluated, information on the expected distribution of chemical mass in the environment);
- sensitivity analyses of TRIM.FaTE and MIRC inputs and model configurations on endpoints of interest; and
- an evaluation of related information.

As noted previously, most of these evaluations focused on the TRIM.FaTE modeling scenario. Methods and results are discussed in the following sections.

C-4.2 Overall Chemical Mass Partitioning

A general evaluation of TRIM.FaTE outputs from simulations run using the screening scenario was conducted to inform the set-up and parameterization of the scenario and confirm that the model outputs were generally reasonable. One approach for evaluating the overall performance of the model is to review the distribution of chemical mass among the various compartment types and chemical sinks included in the scenario. In general, many persistent and bioaccumulative chemicals emitted to the air do not readily deposit within close range of a source but instead are transported in the air away from the source (sometimes great distances) before depositing on land or water surfaces or being transformed to other chemical species.

This is true of the PB-HAPs that can be evaluated using the RTR screening scenario, and thus much of the chemical mass emitted by the modeled sources, regardless of chemical, was not expected to accumulate in the soil, water, or sediment compartments. Instead, most of the chemical mass emitted by the source was expected to be removed through advective transport in the air and to end up in the air sinks (the compartments that capture chemical mass blown outside of the modeling domain). This outcome also would be expected, given the relatively small modeling domain. For chemical mass that remains in the domain (is deposited and does not degrade or leave the domain by other means, such as sediment burial), the overall distribution would be expected to approximate environmental observations, with much of the mass remaining in the soil or benthic sediment.

Exhibit 4-1 illustrates the distribution of chemical mass for the screening scenario for the PB-HAPs of primary concern (one dioxin, one PAH, cadmium, and mercury). Results show the distribution at the end of a 50-year simulation (or similar duration) for each chemical performed at the *de minimis* emission rate (the overall distribution for each chemical is not expected to be substantially different for other emission rates, given a relatively long simulation period). As anticipated, much of the chemical mass emitted to the screening scenario is largely removed from the scenario via air advection processes and transported to air sinks (blown out of the modeled domain of the scenario). The amount remaining in the scenario varies by chemical, with a larger fraction of mass deposited for mercury (which deposits relatively quickly as a divalent species) and much lower deposition for dioxin. Of the chemical mass deposited to plants, land, or water and remaining in the modeled domain, most accumulates in the soil. For the two metals evaluated (mercury and cadmium), the sediment compartment also contains a significant amount of mass.

The mass distribution for mercury presented in Exhibit 4-1 was calculated from a single simulation with emissions split evenly by mass between divalent mercury and elemental mercury. Recall that TRIM.FaTE models mercury transformation among elemental, divalent, and methyl mercury species. Consequently, the distribution of mercury mass shown in the middle and lower parts of the table accounts for inter-species transformation within media as well as movement between media (and all methyl mercury in the scenario was derived from emitted divalent or elemental mercury).

Exhibit 4-1. Distribution of Chemical Mass in Screening Scenario

	2,3,7,8-TCDD	Benzo[a] pyrene	Cadmium	Mercury (Divalent Mercury Emitted)			
				Divalent Mercury	Elemental Mercury	Methyl Mercury	Total Mercury
Distribution of Total Mass Added to Scenario from Modeled Source							
Emitted chemical mass removed from scenario and deposited into air sinks	99.5%	81.6%	97.3%	85.4%	–[b]	–[b]	–[b]
Emitted chemical mass remaining in scenario (not in air sinks)[a]	0.5%	18.4%	2.7%	14.6%	–[b]	–[b]	–[b]
Distribution of Mass Remaining in Scenario [c]							
Air	0.30%	0.2%	0.01%	0.001%	0.002%	0.0%	0.001%
Soil	89.2%	97.6%	96.5%	86.0%	91.1%	98.0%	86.3%
Plants	6.0%	1.6%	0.1%	0.7%	0.0%	0.0002%	0.7%
Surface Water	0.02%	0.01%	0.2%	0.0%	0.5%	0.03%	0.05%
Sediment	4.4%	0.6%	3.2%	13.2%	8.5%	2.0%	12.9%
Aquatic Biota	0.03%	0.001%	0.003%	0.0003%	0.0002%	0.005%	0.0004%
Groundwater	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Distribution of Mass in Soil							
Surface Soil	98.8%	98.9%	30.1%	98.9%	9.3%	98.9%	97.9%
Root Zone Soil	1.2%	1.1%	69.9%	1.1%	90.7%	1.1%	2.1%
Vadose Zone Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

[a] Fraction includes mass transferred from air to soil or other media, but subsequently removed from the modeling domain (e.g., accumulated in sediment burial or degradation/reaction sink).
[b] To calculate the *de minimis level* for mercury, neither elemental mercury or methyl mercury is emitted by the modeled source.
[c] Fractions represent amount of mass remaining in non-sink compartments.

C-4.3 Comparison to Measured Concentrations

C-4.3.1 Scope of the Evaluation

This section presents environmental measurements of the PB-HAPs evaluated for RTR and compares them to modeled concentrations estimated by TRIM.FaTE and the Multimedia Ingestion Risk Calculator (MIRC). Model outputs correspond to the estimates associated with emission rates at the *de minimis* thresholds for each representative PB-HAP calculated for RTR multipathway screening. We emphasize that this analysis is not intended to "validate" the accuracy of the models used for RTR or the specific model configurations used to calculate *de minimis* emission thresholds. In general, although the comparison of environmental data to model results is often a central component of model evaluation, deriving useful conclusions from such comparisons is complicated by a range factors, including: fundamental differences between the modeling scenario and the environmental system in which samples were collected; temporal and spatial issues (e.g., model results representative of long-term average

concentrations vs. measured point estimates); data quality issues; and other factors. In the current evaluation, it is difficult to derive conclusions from model-to-measurement comparisons given the use of a modeling scenario that is intended to be representative but hypothetical and conservative (i.e., possessing characteristics that lead to higher exposures while not representing any real location or specific site). In addition, the RTR modeling scenario is intended to calculate incremental environmental concentrations and exposures without accounting for any "background" concentrations from natural or anthropogenic sources.

In spite these complications, this comparative analysis is an appropriate—and necessary—aspect of the model evaluation process because environmental data provide a general frame of reference for the model outputs. Basic differences between the modeling scenario and the data sets collected preclude the verification or validation of model configuration, performance, or results, and the comparisons should be viewed in light of the complicating issues listed above. However, we believe that this comparative analysis, considered in conjunction with other aspects of the evaluation, is a useful tool in gauging the effectiveness of the screening scenario in informing regulatory decision-making.

C-4.3.2 Methods and Organization of this Section

The TRIM.FaTE and MIRC outputs presented in this section are those used to calculate the *de minimis* emission levels in the screening scenario (see Section C-2 for discussion of *de minimis* emission rates). Model outputs from the source parcel are not used for comparisons; instead, concentrations from the land parcel closest to the source (N1) are used in comparisons for soil results, while outputs from the pond parcel are used for comparing results for water, sediment, and fish (See Exhibit 3-1 for Surface Parcel Layout). Chemical concentrations in air are not evaluated in this section for two reasons. First, the TRIM.FaTE model was not designed to estimate air concentrations for the assessment of inhalation exposure, as noted in Section 2.4.1. Second, chemical concentrations in the air compartment affect exposure concentrations less directly than deposition of the chemical. Therefore, the comparison of chemical concentrations in air to observed values is not included in this evaluation.

The purpose of the 50-year duration of the scenario is to represent long-term input of source emissions over a hypothetical facility lifetime (many compartments also attain an approximate steady state by this time). For all environmental media compartments, the maximum annual average concentrations, taken from the final year of the simulation, are used in MIRC to determine the maximum average concentrations in ingestible products. These concentrations, along with ingestion rates for each food product, are used to calculate average daily doses (ADDs) for each chemical, from which the lifetime cancer risk or age-group-specific hazard quotient (HQ) is then calculated. The model results presented in this section are the environmental media concentrations used to calculate the *de minimis* emission rates (i.e., those concentrations calculated for the 50th year of the TRIM.FaTE simulation) and the ingestible media concentrations calculated in MIRC using those environmental media concentrations (see Exhibit 3-5 for TRIM.FaTE outputs used in exposure calculations).

The evaluations described in this section are presented separately for each chemical for which a screening threshold has been derived. Each chemical-specific section provides a review of the emission profiles used in the modeling process, and a summary of the expected chemical behavior in the environment. This is followed by comparisons between model results and ranges of observed concentrations in environmental media, partitioning behavior, and observed concentrations in ingestible products.

In the charts displaying the comparisons between model results and observed concentrations, the horizontal bars in the graph represent measured concentrations reported in the literature for various environmental media. The green bars show ranges of measured concentrations that were reported as less than a maximum concentration or detection limit, with the upper bound of the bar representing the limit of detection. Blue bars indicate a range of values reported by a particular source. The vertical red lines in the graph represent the modeled TRIM.FaTE concentrations obtained using the de minimis threshold emission rates. Observed data from areas exposed to varying ranges of chemical emissions were chosen for the comparison in order to represent a variety of scenarios. In general, the low end of the range represents concentrations from remote areas not located near a considerable pollution source, while the high end of the range represents concentrations observed near industrial sources (e.g., metal smelters, chlor-alkali plants, incineration/combustion facilities) and/or in urban areas.

The observed values provided as examples in this section include data collected from a range of sampling techniques, locations, emissions profiles, historical contributions, and other factors influencing concentration. They do not represent the entire range of possible measured values, and therefore should not be combined to create a contiguous range of concentrations representative of all patterns in the United States. Model outputs were derived using a generally conservative set of fate and transport inputs, and they reflect the estimated environmental levels assuming concentrations from only one source and no ambient background concentrations.

C-4.3.3 Chemical-Specific Comparisons

C-4.3.3.1 Cadmium

Behavior in the Environment

Based upon reviewed literature, some of the largest anthropogenic sources of cadmium to air are facilities that process, mine, or smelt cadmium-zinc or cadmium-zinc-lead ores; coal and oil-fired boilers; other urban and industrial facilities; phosphate fertilizer manufacturing facilities; road dust; and municipal sewage sludge incinerators (ATSDR 2008). These facilities can emit airborne cadmium particles that are capable of traveling long distances before depositing onto soil or water bodies via dust, rain, or snow. Cadmium adsorbs to soil particles in the surface layers of the soil profile, but to a lesser degree than many other heavy metals (HSDB 2005a). It may enter surface waters through atmospheric fallout, runoff, or wastewater streams, but most cadmium compounds will be removed from the surface water compartment through adsorption to organic matter in sediment or to other suspended compounds. Concentrations in bed sediment are expected to be roughly an order of magnitude higher than those in overlying surface water (HSDB 2005a).

Freshwater fish accumulate cadmium primarily through direct uptake of the dissolved form through the gills and secondarily through diet, which plays a variable role in total cadmium uptake (Reinfelder et al. 1998, Chen et al. 2000, Saiki et al. 1995). The degree to which cadmium bioaccumulates in fish is largely dependent upon water pH and humus content (ATSDR 2008). Reported bioaccumulation factors (BAFs), or bioconcentration factors (BCFs), of 3 to 4,190 (ATSDR 2008) and 907 (EPA 2005a) have been reported for freshwater organisms and fish. Although some biomagnification of cadmium has been reported for aquatic food chains involving fish in saltwater systems, biomagnification in freshwater systems appears to be present only at lower trophic levels (Chen et al. 2000) and in narrowly defined niches (e.g., plankton/macroinvertebrate food chains; Croteau et al. 2005). Biomagnification factors (BMFs)

of less than 1 have generally been reported for fish at higher trophic levels, indicating that cadmium concentrations generally biodiminish from lower to higher trophic levels (Chen et al. 2000, Mason et al. 2000).

Emission Profile

The cadmium *de minimis* emission rate derived from the RTR PB-HAP screening is **0.65 TPY** (based on an HQ of 1 in children aged 1-2). Although the annual concentration from year 50 is used for comparison, modeled concentrations of cadmium in most compartments generally leveled off after year 20. One notable exception was the soil compartment in parcel N6, where the soil in the screening scenario is tilled and no plants are modeled in the TRIM.FaTE scenario. Cadmium concentrations in this compartment continued to rise through year 50. The steady build-up of cadmium in this parcel is likely due to the thickness of the surface soil layer in the N6 parcel. Because this parcel is tilled, the surface soil layer is thicker than in the other parcels, and the rate of exchange between the surface soil and the root zone soil is correspondingly lower.

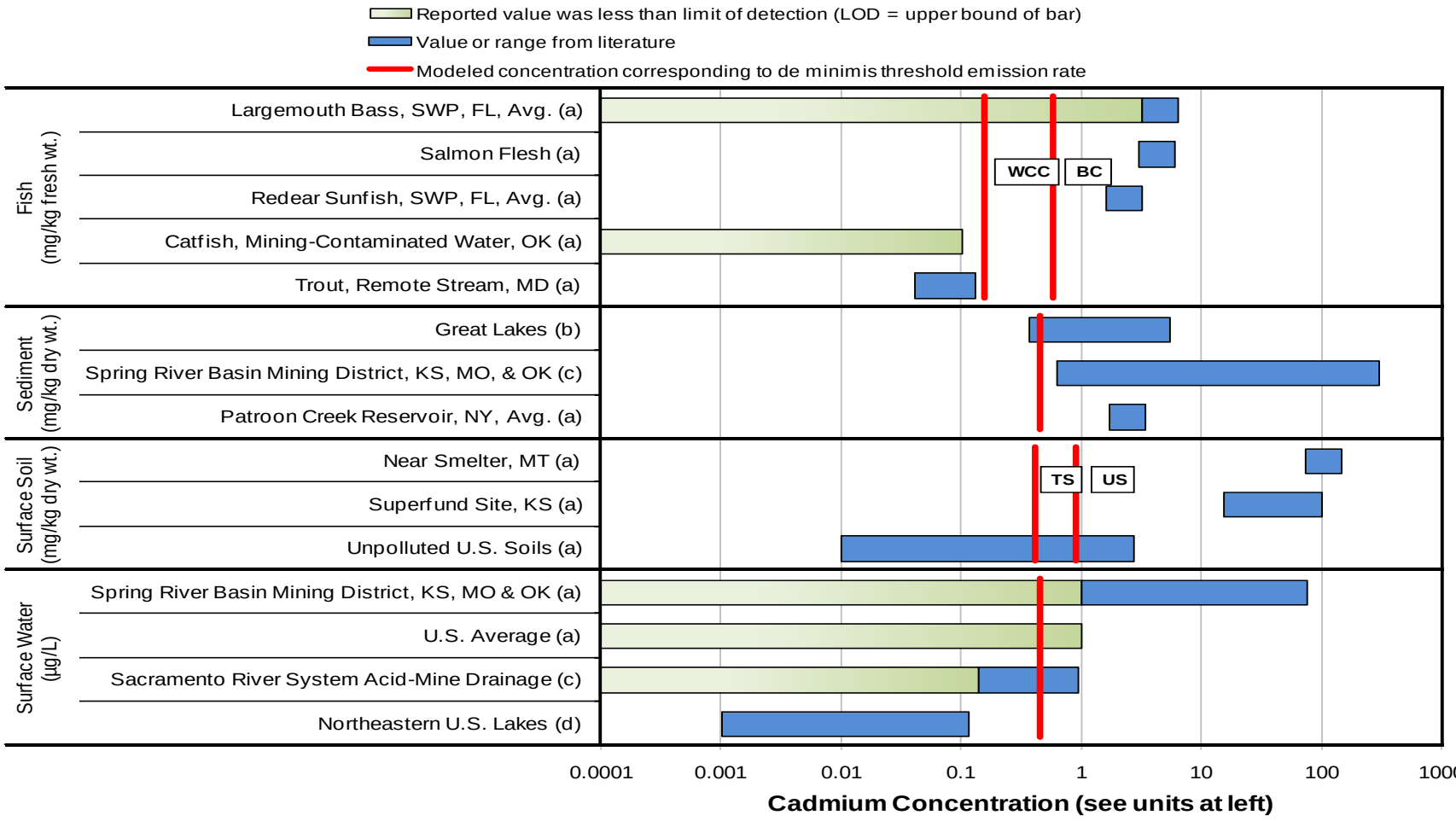
Concentrations in Environmental Media

Exhibit 4-2 displays measured concentrations in environmental media with TRIM.FaTE outputs for the screening scenario. Modeled concentrations and BAFs in fish compartments are shown in Exhibit 4-3. Because relationships between fish compartments in the screening scenario are established within a food web structure, there is no explicit delineation to specific trophic levels. However, for evaluation of trophic level patterns, the water column herbivore (WCH) can be considered to represent the lowest trophic level among fish, while the water column carnivore (WCC) represents the highest trophic level.

The cadmium concentrations output by the RTR screening scenario were consistent with reported values in all environmental media compartments. Consistent with trends noted in the literature, modeled cadmium concentrations were highest in fish at the lowest trophic level evaluated in the scenario (i.e., WCH). Though modeled concentrations did not uniformly decrease with an increase in trophic level, the concentrations for all but the benthic carnivore (BC) compartment were markedly lower than those in the WCH compartment. It should be noted that BAFs for the other chemicals evaluated in this section are several orders of magnitude greater—and span much wider ranges—than the BAFs reported for cadmium in fish. Thus, in the context of comparison between chemicals, the difference between cadmium BAFs in fish compartments is quite small.

The BAFs calculated using model outputs from the RTR screening scenario ranged from approximately 200 to 1,400, which is consistent with the range of values presented in the literature for freshwater systems. Overall, modeled concentrations and BAFs in all fish compartments were reasonably consistent with observed trends, with cadmium levels generally diminishing from lower to higher trophic levels, and consistent with the assumption that diet plays a variable role in the bioaccumulation of this substance in fish.

Exhibit 4-2. Summary of Modeled and Observed Concentrations of Cadmium in Environmental Media



WCC = Water Column Carnivore, BC = Benthic Carnivore, SWP = Stormwater Pond, TS = Tilled Soil, US = Untilled Soil

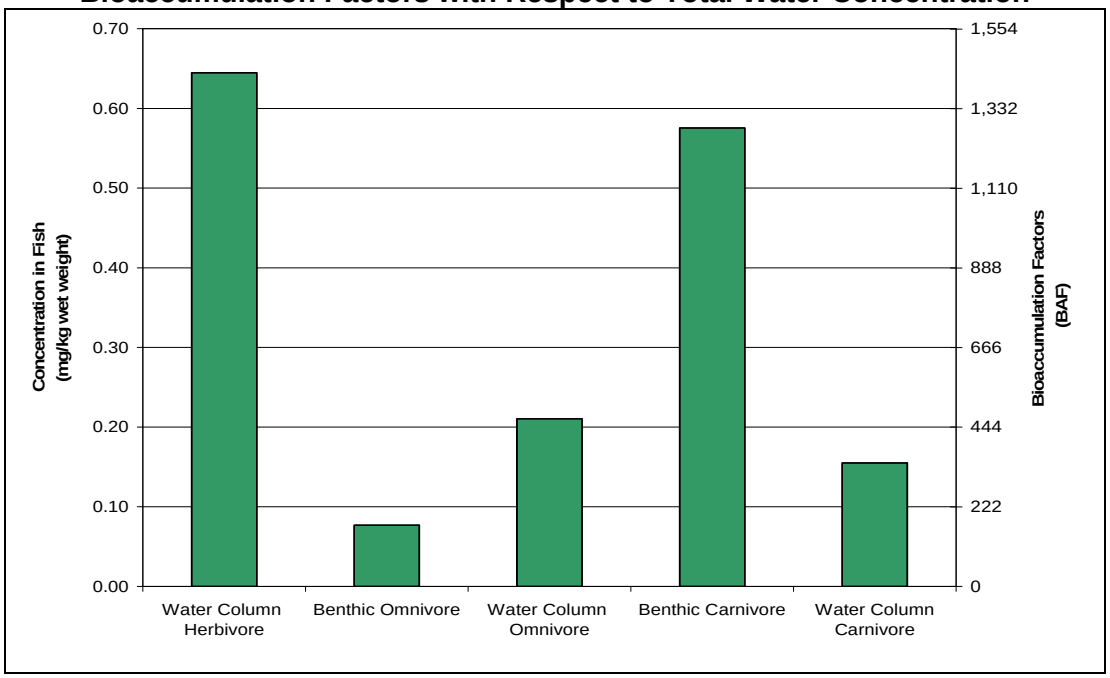
[a] Source: *ATSDR Toxicological Profile for Cadmium* (ATSDR 2008)

[b] Source: *Hazardous Substances Databank Record for Cadmium Compounds* (HSDB 2005a)

[c] Source: Copper, Cadmium, and Zinc Concentrations in Aquatic Food Chains from the Upper Sacramento River and Selected Tributaries (Saiki et al. 1995)

[d] Source: Accumulation of Heavy Metals in Food Web Components Across a Gradient of Lakes (Chen et al. 2000)

Exhibit 4-3. TRIM.FaTE Cadmium Concentrations in Fish and Calculated Bioaccumulation Factors with Respect to Total Water Concentration



Partitioning Behavior

Because most cadmium emitted to the air will eventually deposit onto soils, cadmium has been observed to partition primarily to soil when released to the environment (ATSDR 2008). Cadmium mobility in soil depends strongly on soil pH, clay content, and availability of organic matter—factors that determine whether the cadmium is dissolved or sorbed in surface soil. In general, cadmium binds strongly to organic matter, rendering the metal immobile; however, some plants are able to efficiently take up cadmium, thus providing an entry point for cadmium into the food chain (ATSDR 2008).

Most cadmium in a natural water column is dissolved, but some adsorption to humic substances and other organic complexing agents can occur. This behavior can be especially important in polluted or organic-rich waters, playing a dominant role in cadmium transport (ATSDR 2008). Concentrations in surface water tend to be lower than those in bed sediment because cadmium readily adsorbs to mineral surfaces, hydrous metal oxides, and organic materials present in sediment. Cadmium that has adsorbed to mineral surfaces in the sediment is not easily bioaccumulated in the aquatic food web unless the sediment is disturbed, and the metal is redissolved.

For the RTR screening scenario, 5.7 percent of cadmium in the pond compartment partitioned to surface water, while 94.3 percent partitioned to sediment. The percentages of cadmium in sorbed and dissolved states in surface water, sediment, and surface soil are presented in Exhibit 4-4. The partitioning behavior modeled in TRIM.FaTE is consistent with the behavior of cadmium in the natural environment. The presence of cadmium primarily in the dissolved state in surface water and sorbed to sediment and surface soil is consistent with trends noted in supporting literature.

Exhibit 4-4. Fraction of Cadmium Mass Sorbed vs. Dissolved in TRIM.FaTE Compartments

Compartment	Sorbed	Dissolved
Surface Soil	100.0%	0.0%
Surface Water	4.6%	95.4%
Sediment	99.9%	0.1%

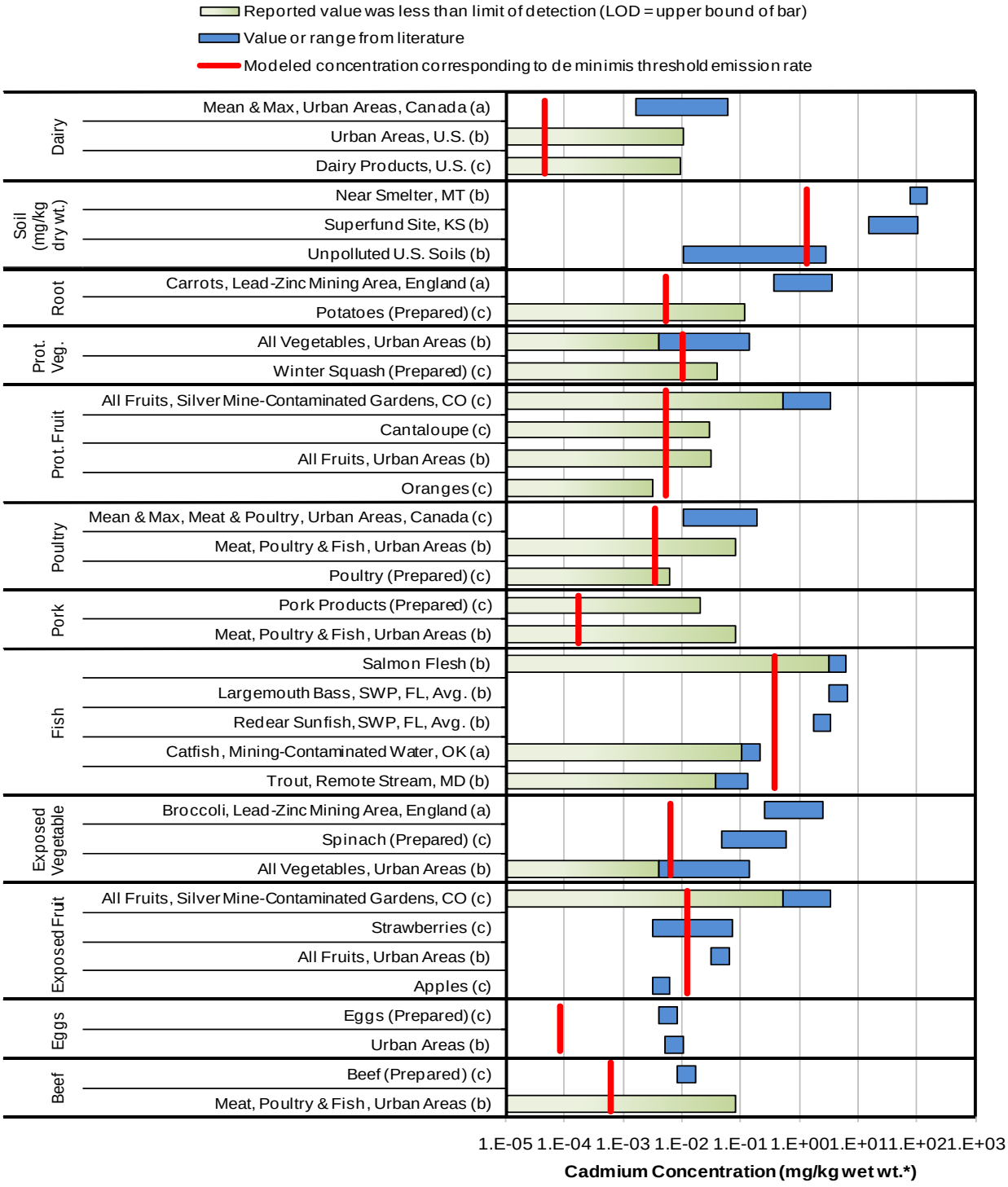
Concentrations in Ingestible Products

The major source of non-inhalation exposure to cadmium outside of occupational settings is through dietary intake. Available data indicate that cadmium accumulates in plants, aquatic organisms, and animals, offering multiple ingestion exposure pathways (ATSDR 2008). However, actual cadmium levels in ingestible products can vary based upon type of food, agricultural and cultivation practices, atmospheric deposition rates, conditions in environmental media, and presence of other anthropogenic pollutants. General trends indicate that high levels of cadmium can be found in green, leafy vegetables; peanuts; soybeans; and sunflower seeds. Meat and fish generally contain lower amounts of cadmium, overall, but cadmium can be found highly concentrated in certain organ meats, such as kidney and liver (ATSDR 2008). In a study of cadmium concentrations in 14 food groups (including prepared foods), meat, cheese, and fruits generally contained low levels of cadmium (ATSDR 2008).

Modeled cadmium concentrations in ingestible products are displayed in Exhibit 4-5 along with cadmium concentrations reported in literature. In the screening scenario we assume that individuals consume equal amounts of benthic carnivores and water column carnivores, so the concentration for fish is given as the average of these two fish compartments. The cadmium concentrations output by MIRC were consistent with reported values in all ingestible media products. The products with higher reported cadmium levels in the literature, including soil, plants, and fish, also contained the highest modeled concentrations.

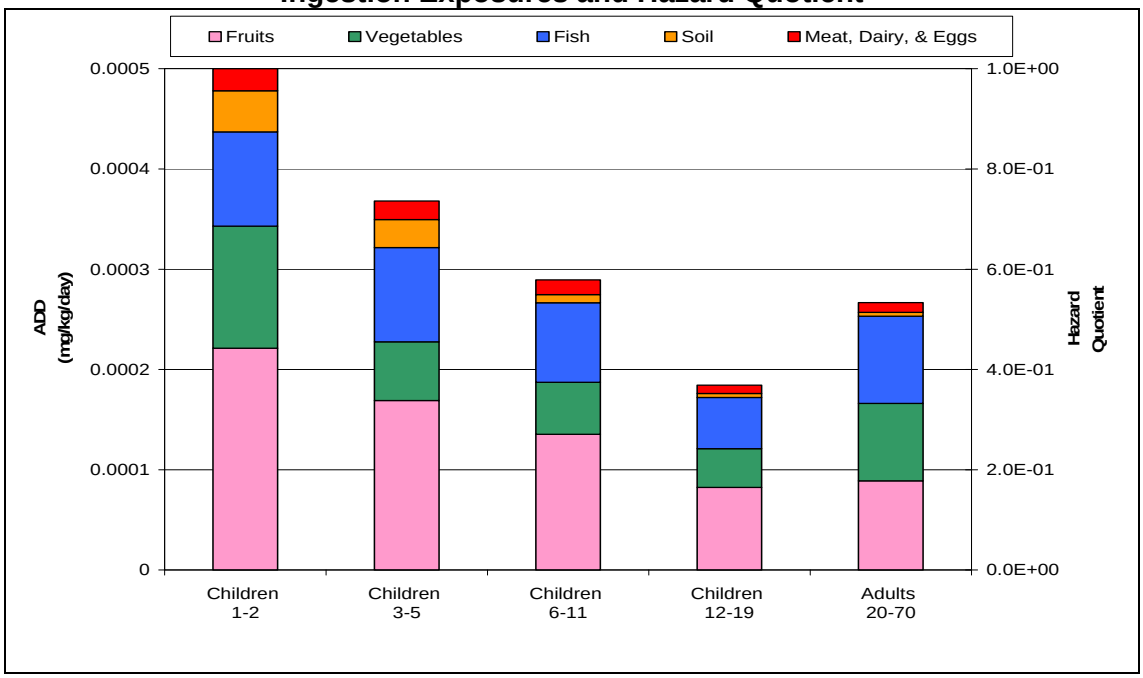
To determine media types most relevant to exposure and risk, the estimated media concentrations must be combined with ingestion exposure factors (i.e., higher concentrations do not necessarily equal higher risk. The contribution of ingestion exposure pathways to the ADD (and thus the HQ) for the different age categories are displayed in Exhibit 4-6. The HQ of 1 for children aged 1-2 was used to determine the *de minimis* level for cadmium in the screening scenario. The dominant exposure pathway for this age group was consumption of fruit (protected and exposed), which comprises almost half of the total ADD. Fruit was a dominant exposure pathway for all age groups, though less so for adults. The other dominant exposure pathways were fish, vegetables, and soil. This trend is consistent with observed trends and representative of the preferential bioaccumulative behavior of cadmium in the natural environment.

Exhibit 4-5. Summary of Modeled and Observed Concentrations of Cadmium in Ingestible Media



*except soil, which is expressed as mg/kg dry weight.
SWP = Stormwater Pond
[a] Source: *Hazardous Substances Databank for Cadmium Compounds* (HSDB 2005a)
[b] Source: *ATSDR Toxicological Profile for Cadmium* (ATSDR 2008)
[c] Source: *Total Diet Study, Market Baskets 1991-3 through 2005-4* (FDA 2007)

Exhibit 4-6. Estimated Contribution of Modeled Food Types to Cadmium Ingestion Exposures and Hazard Quotient



C-4.3.3.2 Mercury

Behavior in the Environment

Mercury emitted by anthropogenic sources undergoes changes in form and species as it moves through environmental media. The three most relevant forms of mercury in the environment are elemental mercury, divalent inorganic mercury, and methyl mercury. Based upon reviewed literature, some of the largest anthropogenic sources of mercury to air are facilities that process, mine, or smelt mercury ores; industrial/commercial boilers; fossil fuel combustion activities (primarily coal); cement production facilities; other urban and industrial facilities; and medical and municipal waste incinerators (ASTDR 1999). Stack emissions can include a mixture of elemental and divalent forms, mostly in the gaseous phase, with some divalent forms in particle-bound phases (EPA 1997b). Although elemental mercury is more prevalent in the atmosphere, divalent mercury can be found at higher concentrations near emissions sources. Elemental mercury has a very long residence time in the atmosphere and will thus be distributed relatively evenly on a global scale, resulting in negligible impacts from any single source on local and regional scales. Divalent mercury, however, is removed from the atmosphere at a faster rate than elemental mercury, and may be transferred to the surface near the emission source via wet or dry deposition, where it appears to adsorb tightly to soil particles (EPA 1997b). Some adsorbed divalent mercury can be reduced to elemental mercury and be revolatilized back into the atmosphere. Methylation to methyl mercury by microbes can also occur in the soil; however, divalent mercury is generally the dominant species in soil (EPA 1997b). Small amounts of divalent and methyl mercury in soil can be transported to surface water through runoff and leaching. Direct deposition to water can occur through atmospheric fallout. Once in the water body, divalent mercury may be methylated through microbial activity, and both divalent and methyl mercury may be further reduced to elemental mercury, which will reenter the atmosphere. But, as in soil, divalent mercury will generally be the dominant species in both

surface water and sediment. Methyl mercury is readily bioaccumulated and efficiently biomagnified in aquatic organisms and is typically the species of greatest concern for mercury exposure via the food chain.

Emission Profile

TRIM.FaTE assumes that only elemental mercury and divalent mercury are emitted from the source (see Section 2.3); but once in the environment, these species may change form, resulting in ingestion exposure to methyl mercury as well as divalent mercury. Relative to these two species, elemental mercury does not comprise a significant amount of total mercury in environmental media other than air. Because human health effects from exposure to methyl mercury and divalent mercury are not additive, two *de minimis* rates for emissions of divalent mercury were calculated for exposure to methyl mercury and divalent mercury, respectively, to determine the most health-protective level. The emission rate calculated for the HQ of divalent mercury was lower, and thus was used to define the *de minimis* emission threshold. At the *de minimis* rate, the HQ for divalent mercury in children aged 1-2 is approximately twice as high as that calculated for methyl mercury.

For the scale of this evaluation, we compared predicted concentrations of total mercury to measured concentrations of total mercury in environmental media compartments and ingestible products. In TRIM.FaTE, divalent mercury comprises from 83 to 100 percent of total mercury in all environmental and ingestible media compartments except fish. Because methyl mercury comprises approximately 90 percent of total mercury in fish, and this pathway is recognized as a significant contributor to mercury exposure in humans, we also examine methyl mercury bioaccumulation in fish and the contribution of this pathway to methyl mercury ingestion exposure and HQ.

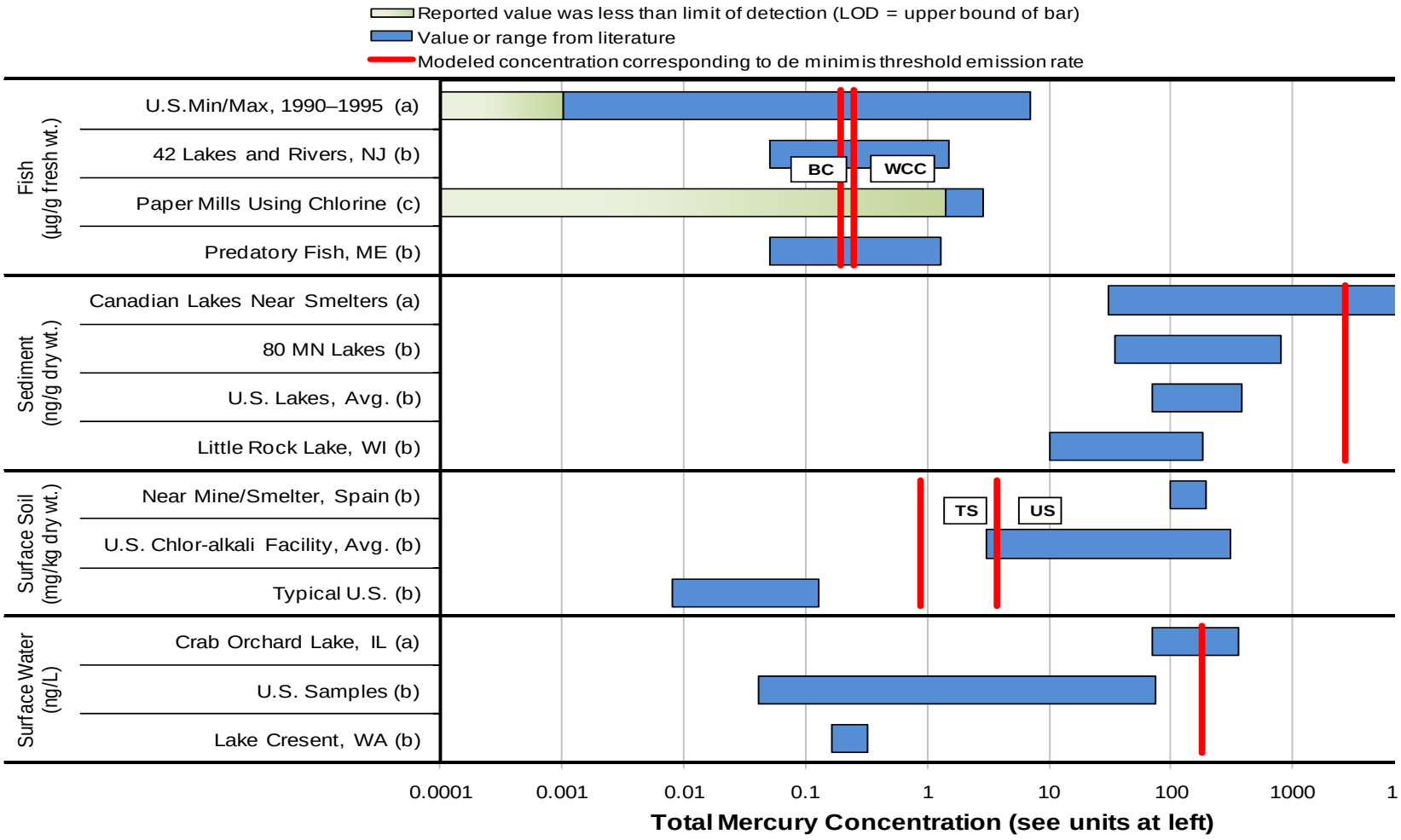
The *de minimis* emission rate used for mercury in the RTR screening scenario is **1.6E-01 TPY** of divalent mercury (based on an HQ of 1 for divalent mercury exposure for children aged 1-2). Annual concentrations from year 50 used to calculate the *de minimis* threshold are presented here, and these values are used for comparison. Modeled concentrations of mercury in all compartments but untilled soil continued to rise up to the end of the simulation, with the rate of increase steadily diminishing over time.

Concentrations in Environmental Media

Exhibit 4-7 displays measured ranges of total mercury concentrations in environmental media with TRIM.FaTE outputs for the screening scenario. The total mercury concentrations estimated by TRIM.FaTE were generally consistent with reported values in all environmental media compartments. Overall, modeled total mercury concentrations were generally within the range of values reported for areas near a significant pollution point source (e.g., smelter, chlor-alkali facility).

Methyl mercury is formed via microbial transformation of inorganic mercury in sediment, surface water, and soil and readily accumulates in the tissues of planktivorous and piscivorous fish. There seems to be a relationship between methyl mercury levels in fish and water pH, showing higher levels of methyl mercury in fish found in acidic lakes (EPA 1997b). Lake alkalinity and dissolved oxygen content may also influence the ability of fish to bioaccumulate methyl mercury (EPA 1997b). BAFs on the order of 10^5 to 10^7 have been reported for methyl mercury concentrations in fish (Alpers et al. 2008). Modeled concentrations of mercury species in fish compartments along with estimated methyl mercury BAFs are shown in Exhibit 4-8. The

Exhibit 4-7. Summary of Modeled and Observed Concentrations of Total Mercury in Environmental Media



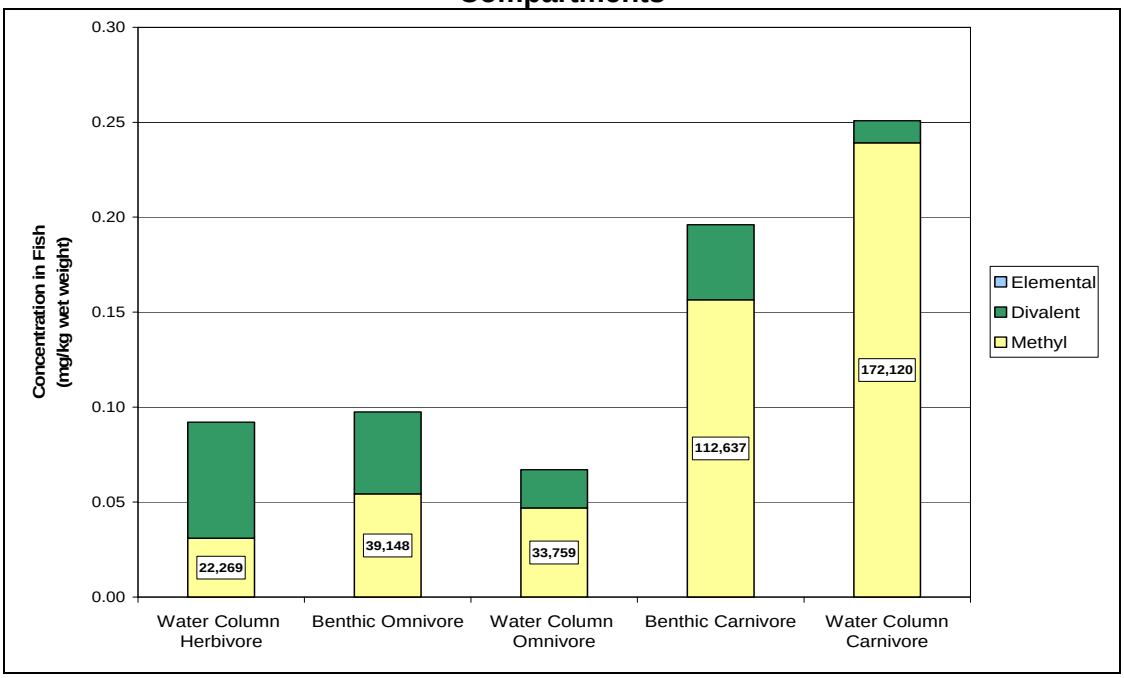
BC = Benthic Carnivore, WCC = Water Column Carnivore, TS = Tilled Soil, US = Untilled Soil

[a] Source: *ATSDR Toxicological Profile for Mercury* (ATSDR 1999)

[b] Source: *Mercury Study Report to Congress* (EPA 1997b)

[c] Source: *National Study of Chemical Residues in Fish* (EPA 1992a)

Exhibit 4-8. TRIM.FaTE Mercury Concentrations, Speciation, and Calculated Methyl Mercury Bioaccumulation Factors (in white boxes) in Fish Compartments



TRIM.FaTE outputs for the fish compartments mirror a trend noted in the literature in which divalent mercury is observed more prominently in lower trophic level organisms, while methyl mercury is the more prominent species in higher trophic level organisms (ATSDR 1999). Calculated BAFs are consistent with reported values, with the higher trophic levels exhibiting BAFs of approximately 10^5. The biomagnification of the methylated species in the aquatic ecosystem can be observed in Exhibit 4-8, with modeled methyl mercury concentrations increasing substantially from the lower to higher trophic levels.

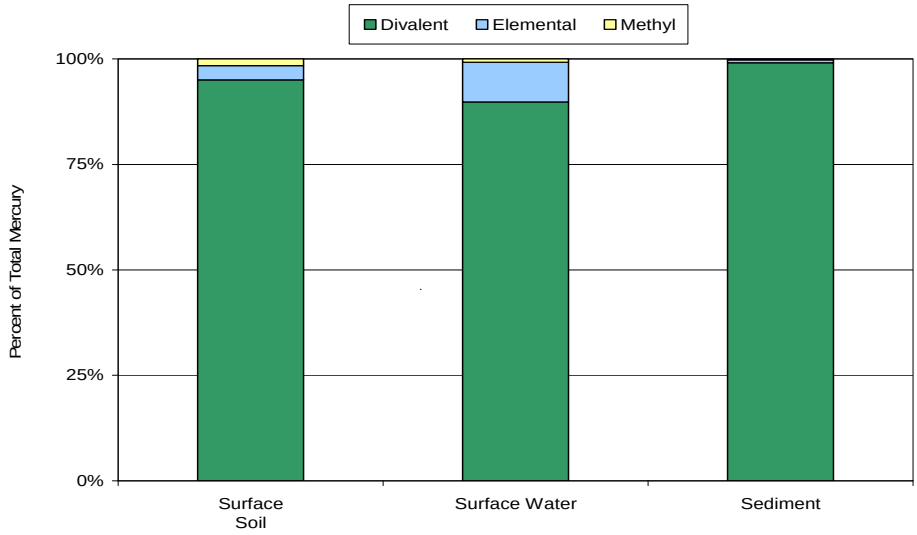
Partitioning Behavior

Soil acts as the primary reservoir for divalent mercury emitted from anthropogenic sources. In some cases, divalent mercury will be adsorbed onto forms of dissolved organic carbons (DOCs), which are susceptible to runoff. However, most divalent mercury from atmospheric deposition remains in the soil profile in the form of inorganic compounds bound to soil organic matter. The ability of mercury to form complexes with soil organic matter is highly dependent on soil conditions such as pH, temperature, and soil humic content. For example, mercury strongly adsorbs to humic materials and sesquioxides in soil at pH > 4 and in soils with high iron and aluminum content (ATSDR 1999). Although inorganic compounds containing divalent mercury are relatively soluble, this complexing behavior with organic matter significantly limits mercury transport. Only very small amounts of mercury present in soil are partitioned to runoff. Elemental mercury present in soil (e.g., as a result of the reduction of divalent mercury) will readily volatilize, especially in acidic soils, and this species therefore comprises very little of the total mercury content in soil. Typically, methyl mercury comprises 1 to 3 percent of total mercury in soil (EPA 1997b). For the TRIM.FaTE results in the RTR screening scenario, divalent mercury comprised approximately 95 percent of total mercury in the surface soil compartment; this result is consistent with environmental trends.

Solubility of mercury in water depends on the species and form of mercury, water pH, chloride ion concentrations, and other factors (ATSDR 1999). Solid forms of inorganic mercury compounds will partition to particulates in the water column and then partition to the sediment bed (EPA 1997b). Low pH favors the methylation of mercury in the water column, typically performed by sulfur-reducing bacteria in anaerobic conditions. Methyl mercury generally comprises 10 percent of total mercury observed in surface water (EPA 1997b). A considerable amount (25 to 60 percent) of both divalent mercury compounds and methyl mercury will be strongly particle-bound in the water column (EPA 1997b). The remaining mercury will be in a dissolved state. Most of the elemental mercury produced as a result of reduction of divalent mercury by humic acid will volatilize.

Screening scenario modeling results for mercury speciation in environmental media compartments are displayed in Exhibit 4-9 along with the percent of each species that was present in both adsorbed and dissolved states in surface soil, surface water, and sediment. Consistent with observed trends, divalent mercury was the most prevalent species in modeled surface soil, surface water, and sediment compartments, while methyl mercury was the dominant species in fish. For the RTR screening scenario, 99 percent of total mercury in the pond (i.e., surface water and sediment) was divalent mercury, while 0.7 percent was methyl mercury and 0.2 percent was elemental mercury. The TRIM.FaTE outputs of approximately 3 percent methyl mercury in soil and 9 percent methyl mercury in surface water are also consistent with trends noted in the literature (EPA 1997b).

Exhibit 4-9. TRIM.FaTE Mercury Speciation and Partitioning in Environmental Media Compartments



Chemical	Methyl Mercury		Elemental Mercury		Divalent Mercury	
	Sorbed	**Dissolved**	**Sorbed**	**Dissolved**	**Sorbed**	**Dissolved**
Surface soil	100.0%	0.0%	100.0%	0.0%	100.0%	0.0%
Surface Water	80.7%	19.3%	4.8%	80.7%	83.0%	17.0%
Sediment	100.0%	0.0%	100.0%	0.0%	100.0%	0.0%

Overall, the partitioning behavior in the RTR screening scenario is consistent with the behavior of mercury in the natural environment. Within the pond compartment of the model, 99.6 percent of divalent mercury, 98.6 percent methyl mercury, and 94.5 percent of elemental mercury partitioned to the sediment. Though all mercury in natural soils and sediment may not be in the

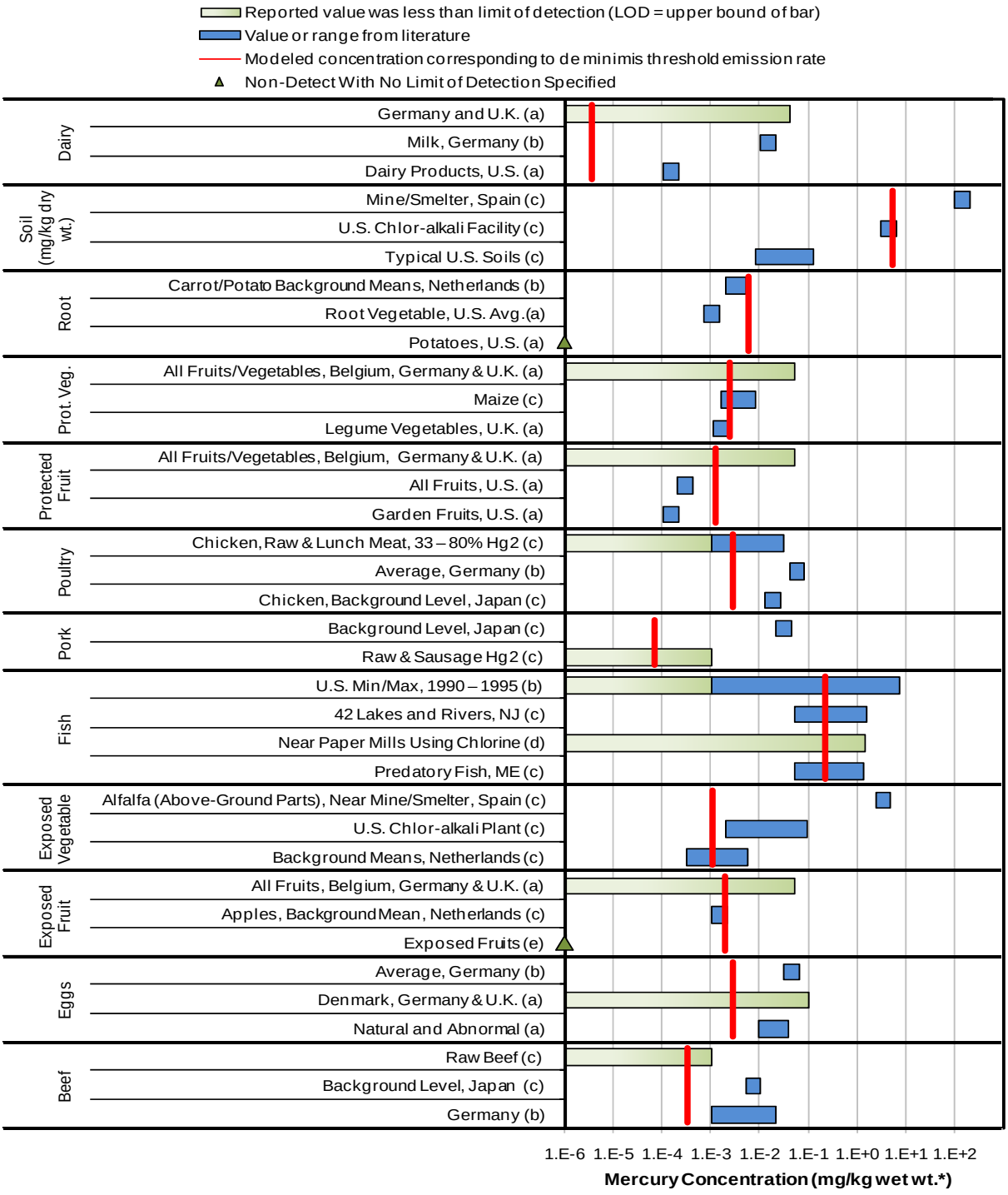
sorbed state, the percentage of mercury that does not complex with organic matter would likely be negligible. The behavior in the TRIM.FaTE surface water compartment is consistent with trends observed in natural waters in which divalent and methyl mercury species exhibit a strong preference for adsorption to suspended matter in the water column. These species may also form complexes with dissolved organic carbon and thus be considered as dissolved in the water column; however, this is not as prevalent as complexation with suspended solids. Conversely, elemental mercury does not adsorb readily to suspended particles, and is therefore found primarily in the dissolved form in natural waters, where it is highly susceptible to revolatilization into the atmosphere. The high percent dissolved elemental mercury estimated by TRIM.FaTE (when compared to percent dissolved divalent or methyl mercury) in surface water is consistent with this trend. It has been estimated that more than 97 percent of dissolved gaseous mercury in the water column is elemental mercury (EPA 1997b).

Concentrations in Ingestible Products

Available data indicate that mercury bioaccumulates in plants, aquatic organisms, and terrestrial animals, providing multiple ingestion exposure pathways (EPA 1997b, ASTDR 1999). Low levels of mercury are found in plants, with leafy vegetables containing higher concentrations than potatoes, grains, legumes, and other vegetables and fruits (ASTDR 1999, EPA 1997b). Cattle are capable of demethylating mercury in the rumen, and therefore store little of the small amount of mercury that is transferred to livestock via foraging or silage/grain consumption. Mercury content in meat and cow's milk is therefore low (ASTDR 1999). Concentrations of methyl mercury in fish are generally highest in larger, older specimens at the higher trophic levels (EPA 1997b).

Concentrations of total mercury in ingestible products are presented in Exhibit 4-9. Though data on mercury in foods other than fish are not abundant in the literature, total mercury concentrations output by MIRC were generally consistent with the reported values that were available. The exposure pathways that most influence the mercury HQs in the model can be seen in Exhibit 4-10, which displays the contributions of ingestion exposure pathways to the divalent mercury and methyl mercury ADDs for all age groups analyzed in the screening scenario. The dominant exposure pathway for divalent mercury in children aged 1-2 was ingestion of contaminated soil. Divalent mercury accumulates readily in the upper 20 cm of the soil profile, where it is accessible to children that may frequently ingest soil (EPA 1997b, EPA 2008). Exposures to children aged 1 to 2 are 4 to 5 times higher than exposures to children and adults aged 12 to 70 years. This is driven by a very high soil ingestion rate in children, and the assumption that the soil consumed is next to the emission source. In older children and adults who do not frequently ingest soil, fruits and vegetables provide the greatest exposure pathway for divalent mercury. Though divalent mercury is not considered to be readily taken up by plants from the soil, atmospheric deposition may figure strongly into elevated plant concentrations. In addition to the deposition of divalent mercury directly onto plant surfaces, divalent mercury may accumulate in edible portions of plants due to the transformation of elemental mercury following deposition. The relatively low contributions by meat and fish consumption are consistent with observed trends indicating that the divalent mercury biotransfer is not high between plants and animals, and that the most persistent mercury species in fish is methyl mercury, rather than divalent mercury (EPA 1997b, ATSDR 1999).

Exhibit 4-10. Summary of Modeled and Observed Concentrations of Total Mercury in Ingestible Media



*except soil, which is expressed as mg/kg dry weight

[a] Source: *Hazardous Substances Databank Report for Mercury Compounds* (HSDB 2005b)

[b] Source: *ATSDR Toxicological Profile for Mercury* (ATSDR 1999)

[c] Source: *Mercury Study Report to Congress* (EPA 1997b)

[d] Source: *National Study of Chemical Residues in Fish* (EPA 1992a)

[e] Source: *Total Diet Study, Market Baskets 1991-3 through 2005-4* (FDA 2007)

The dominant exposure pathway for methyl mercury in all age groups was ingestion of fish (Exhibit 4-11). Though methyl mercury concentrations in fish were very high (approximately 88 percent of total mercury) in Exhibit 4-11, and fish consumption can contribute substantially to methyl mercury exposure, the HQ for this species (Exhibit 4-12) is likely lower than that of divalent mercury in children aged 1-2 due to a relatively low fish consumption rate in combination with high exposure to divalent mercury from multiple pathways, but primarily through ingestion of soil. The divalent and methyl mercury age-group-specific HQs are displayed in Exhibit 4-12. In humans aged 6-70, the methyl mercury HQ is higher than that for divalent mercury, which is likely the result of lower soil consumption rates and higher fish consumption rates.

Exhibit 4-11. Estimated Contribution of Modeled Food Types to Divalent Mercury and Methyl Mercury Ingestion Exposures

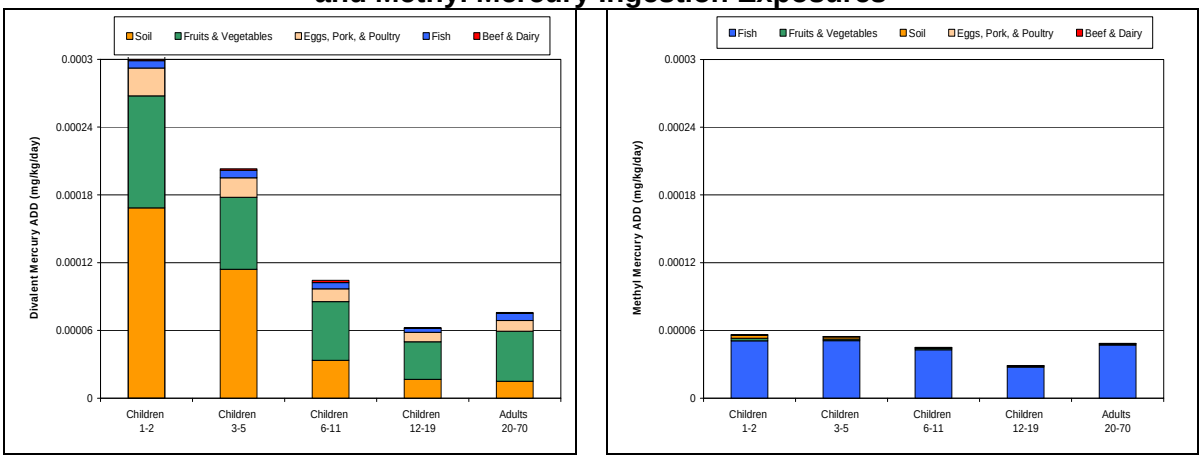
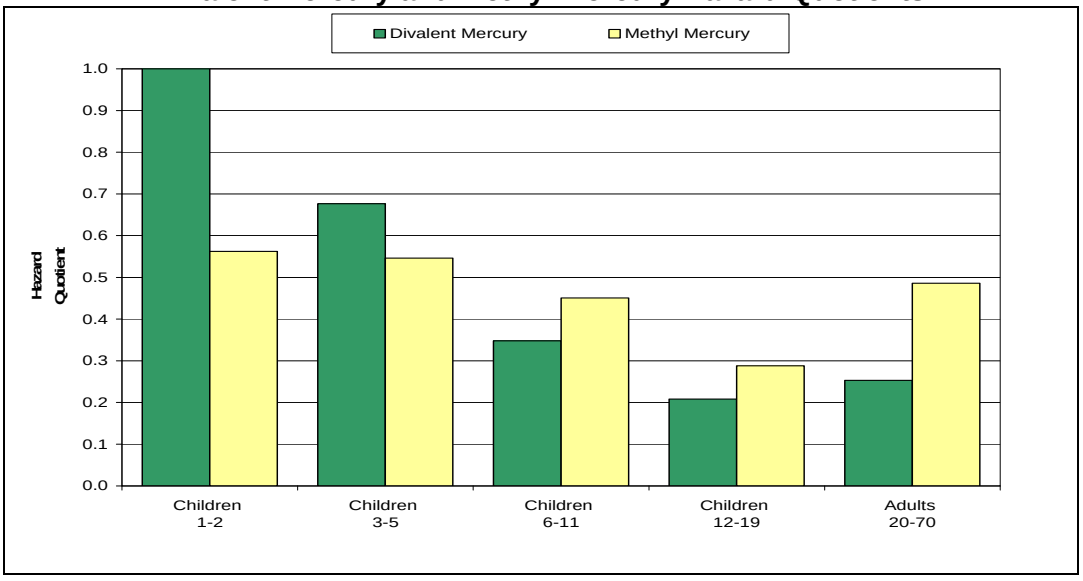


Exhibit 4-12. Estimated Contribution of Summed Modeled Food Types to Divalent Mercury and Methyl Mercury Hazard Quotients



C-4.3.3.3 Dioxins (2,3,7,8-TCDD)

Behavior in the Environment

Polychlorinated benzo-dioxins and -furans[15] are reported in the National Emission Inventory (NEI) as individual congeners, congener groups, or toxic equivalents (TEQs). The dioxin congener 2,3,7,8-TCDD was used as a surrogate to evaluate risks for chlorinated dibenzo-p-dioxins in the screening scenario. Of the chlorinated dioxin congeners, this compound has been the most widely studied, and is considered to be one of the two most toxic congeners to vertebrates (ASTDR 1998). As a result, the World Health Organization (WHO) assigned a toxic equivalency factor (TEF) of 1 to this congener, meaning that the concentrations of the other congeners are scaled relative to the toxicity of 2,3,7,8-TCDD (See Attachment C-2 for TEFs). We deemed this species an appropriate surrogate to represent the fate and transport behavior of the dioxin group.

Incineration/combustion processes are believed to be the primary emission sources for chlorinated dioxins (ASTDR 1998). The five stationary source categories that generate the vast majority of 2,3,7,8-TCDD emissions in the United States are municipal waste incineration, medical waste incineration, hazardous waste kilns from Portland cement manufacturing, secondary aluminum smelting, and biological incineration. Dioxins emitted to the atmosphere may be transported long-range as vapors or bound to particulates, depositing in soils and water bodies in otherwise pristine locations far from the source. Though airborne dioxins are susceptible to wet and dry deposition, most dioxins emitted to the atmosphere through incineration/combustion processes are not deposited close to the source (ASTDR 1998).

Dioxins strongly adsorb to organic matter in soil and show very little vertical movement, particularly in soils with a high organic carbon content (ASTDR 1998). Most surface water deposition occurs through dry deposition from the atmosphere and from wind-transported eroded soil particles contaminated with dioxins. Most dioxins entering the water column will partition to bed sediment, which appears to be the primary environmental sink for this chemical group (EPA 2004b). Although dioxins bound to aquatic sediment will primarily be buried in the sediment compartment, some resuspension and remobilization of congeners may occur as a result of disturbance of sediments by benthic organisms (ASTDR 1998).

Bioaccumulation factors in fish are high for 2,3,7,8-TCDD as a result of the lipophilic nature of chlorinated dioxins. Though the processes by which freshwater fish accumulate dioxins are not well understood, it has been shown that fish and invertebrates both bioaccumulate congeners when exposed to contaminated sediments, and bioconcentrate congeners dissolved in water (EPA 2004b). However, because most dioxins in the aquatic environment are adsorbed to suspended particles, it is unlikely that direct uptake from the water is the primary route of exposure for most aquatic organisms at higher trophic levels (ASTDR 1998). At the lower trophic levels, the primary route of exposure appears to be through uptake of water in contaminated sediment pores, whereas the primary route of exposure in the higher trophic levels appears to be through food chain transfer. Following ingestion, some fish are capable of slowly metabolizing certain congeners, such as 2,3,7,8-TCDD, and releasing the polar metabolites in bile. This may ultimately limit bioaccumulation at higher trophic levels (ASTDR 1998). Measured bioaccumulation factors for 2,3,7,8-TCDD are not widely reported due to the difficulty of detecting trace levels of congeners in ambient water. Reported bioconcentration factors, which can be measured in laboratory conditions, but do not account for exposure

[15] Group is commonly referred to as “dioxins;” this short hand is used in this section.

pathways other than direct uptake from water, range from 200-100,000, depending on the species and medium (ASTDR 1998).

Emission Profile

The 2,3,7,8-TCDD *de minimis* emission rate used for the RTR screening scenario is **3.2E-08 TPY** (based on lifetime cancer risk of 1-in-a-million). Although the annual concentration from year 50 is used for comparison, modeled concentrations of 2,3,7,8-TCDD either reached an approximate steady state or had begun to level off in all compartments by year 20.

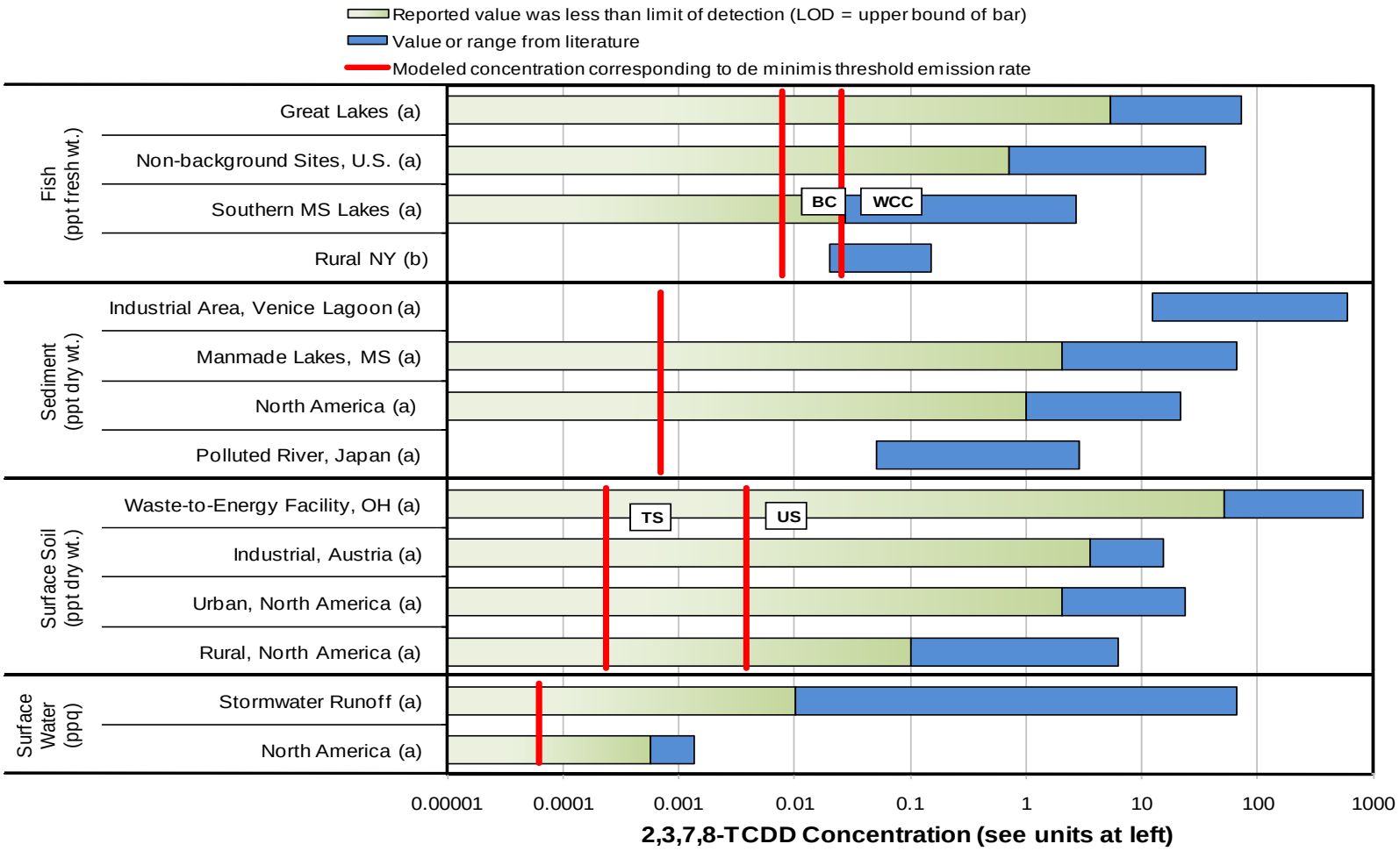
Concentrations in Environmental Media

Measured ranges of total dioxin TEQ concentrations[16] in environmental media are displayed with modeled TEQ concentrations of 2,3,7,8-TCDD in Exhibit 4-13. Modeled concentrations and BAFs in fish compartments are shown in Exhibit 4-14. The 2,3,7,8-TCDD concentrations output by the RTR screening scenario were consistent with reported dioxin TEQs in all environmental media compartments (Exhibit 4-13). Because dioxin congeners are present at trace levels, analytical instruments must be very sensitive if congeners are to be detected in environmental media. As a result, limits of detection in many instruments may not be sufficiently sensitive to produce quantifiable results for very low levels of dioxin congeners. Many of the concentrations output by TRIM.FaTE are low enough to be considered nondetects by most analytical instruments.

Though the 2,3,7,8-TCDD concentrations in environmental media estimated by TRIM.FaTE are generally low, they are not outside the range of plausible values when considered as a surrogate for the dioxin profile. In the screening scenario, the amount of chemical mass in the water column is small relative to the amount in the sediment. This is consistent with the trend that most congeners are removed from water bodies through adsorption to organic matter in soil and sediment. The screening scenario BAFs are also consistent with observed trends that indicate that 2,3,7,8-TCDD accumulates much less readily in herbivorous fish than in carnivorous fish that consume other contaminated organisms (ATSDR 1998).

[16] It was not possible to confirm that TEQ concentrations reported in the literature and summarized here were all estimated using the most recent TEF scheme adopted by the WHO in 1998. TEQ concentrations reported here may represent values determined using TEFs from EPA's 1989 scheme, WHO's 1994 scheme, or WHO's 1998 scheme (EPA 1997b).

Exhibit 4-13. Summary of Modeled 2,3,7,8-TCDD Concentrations and Observed Total Dioxin TEQ Concentrations in Environmental Media

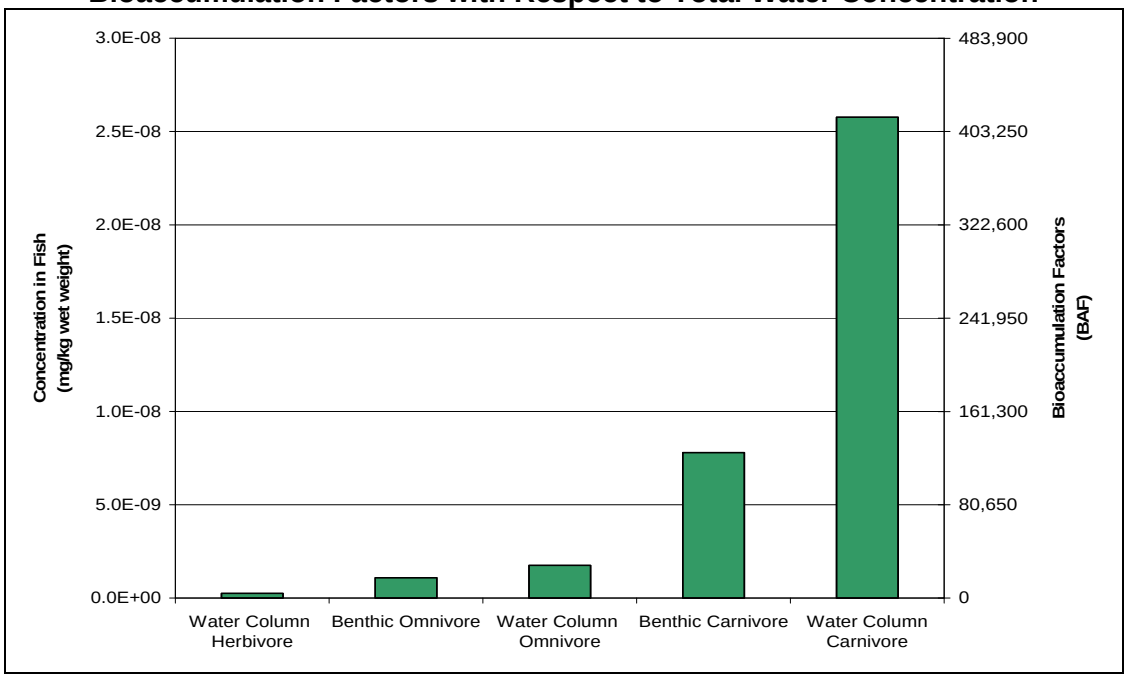


BC = Benthic Carnivore, WCC = Water Column Carnivore, TS = Tilled Soil, US = Untilled Soil

[a] Source: *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds* (EPA 2004b)

[b] Source: *Toxicological Profile for Chlorinated Dibenzo-p-dioxins* (ATSDR 1998)

Exhibit 4-14. TRIM.FaTE 2,3,7,8-TCDD Concentrations in Fish and Calculated Bioaccumulation Factors with Respect to Total Water Concentration



Partitioning Behavior

Chlorinated dioxins have been observed to partition mostly to soil when released to the environment, and sorbed congeners are unlikely to leach or volatilize out of the soil profile. In soils with a high organic carbon content, congeners below the top few millimeters of the soil are very strongly adsorbed to organic matter and exhibit very little migration. Most dioxins deposited onto soil are expected to remain buried in the soil profile, and erosion of contaminated soil particles is the only significant mechanism for transport to water bodies.

Because of the hydrophobic nature of the 2,3,7,8-TCDD congener, and its affinity for organic carbon, 70 to 80 percent of the congener is expected to bind to suspended organic particles in natural waters. The remainder in the water column is associated with dissolved organic substances (ASTDR 1998). Because most of the dioxins in water are in a sorbed state, the ultimate fate of most congener-laden particles is in the bed sediment. For the RTR screening scenario, more than 99.5 percent of 2,3,7,8-TCDD in the pond compartment was found in the sediment. The percentages of 2,3,7,8-TCDD in sorbed and dissolved states in surface water, sediment, and soil are presented in Exhibit 4-15.

Exhibit 4-15. Fraction of 2,3,7,8 - TCDD Mass Sorbed vs. Dissolved in TRIM.FaTE Compartments

Compartment	Sorbed	Dissolved
Surface Soil	100.0%	0.0%
Surface Water	75.1%	24.9%
Sediment	100.0%	0.0%

The partitioning behavior exhibited 2,3,7,8-TCDD in TRIM.FaTE is consistent with observed trends. In the surface water compartment, approximately 75 percent of 2,3,7,8-TCDD was present in the sorbed state, which is within the reported 70-80 percent range reported in the literature. In the sediment and soil compartments, 100 percent of the congener is in the sorbed state, which is representative of the strong preference of dioxins for adsorption to soil and sediment organic matter.

Concentrations in Ingestible Products

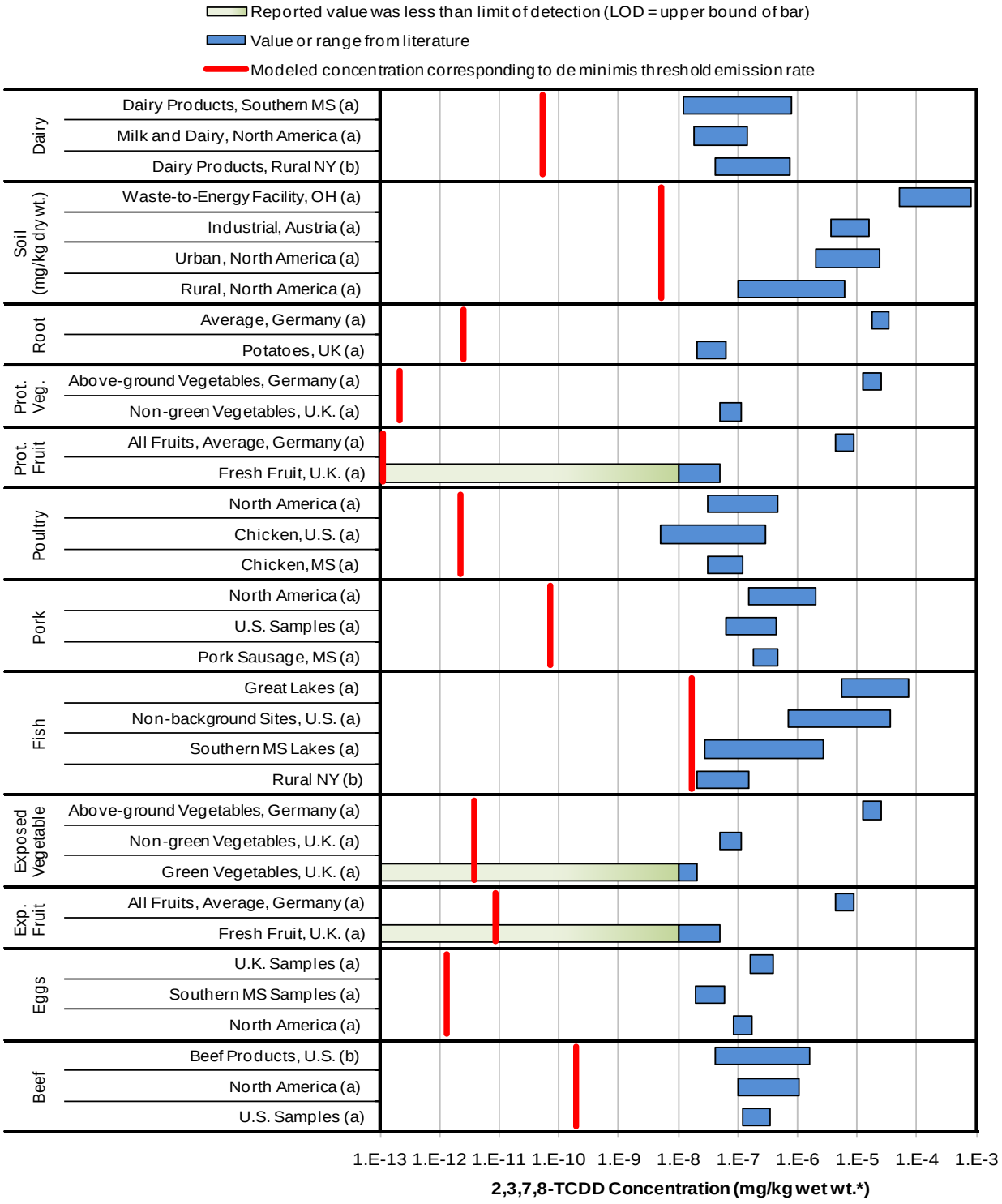
The primary source of non-inhalation exposure to dioxins outside of occupational settings is through dietary intake, which accounts for over 90 percent of daily dioxin intake (ATSDR 1998). Available data indicate that dioxins concentrate in plants, aquatic organisms, and animals, offering multiple ingestion exposure pathways. However, actual congener levels in ingestible products can vary based upon type of food, agricultural and cultivation practices, atmospheric deposition rates, conditions in environmental media, and presence of other anthropogenic pollutants. Dioxins appear to enter the terrestrial food chain primarily through vapor phase deposition onto plant surfaces, which are then consumed by larger animals. Another major source of exposure to dioxins is through ingestion of contaminated soil by animals.

Observed trends indicate that meat, dairy, and fish are the dominant exposure pathways, comprising 90 percent of dioxin dietary intake (ATSDR 1998). Though concentrations in vegetables are generally exceptionally low, root vegetables normally contain slightly higher concentrations of dioxins than vegetables that are affected primarily by atmospheric deposition, such as lettuce and peas (ATSDR 1998). The 2,3,7,8-TCDD concentrations in ingestible product compartments are displayed in Exhibit 4-16.

Data for concentrations of dioxin congeners in ingestible products are not abundant and sophisticated analytical instruments with sufficiently sensitive limits of detection were not widely available for older studies, which likely resulted in a greater number of nondetects in samples. As a result, the data available for comparison was limited, but concentrations of 2,3,7,8-TCDD in ingestible products were generally consistent with the available dioxin TEQ values (Exhibit 4-16). As noted in the literature, the concentration in the fish compartment for the screening scenario was at least one order of magnitude greater than those in the other compartments. Among the compartments with the lowest concentrations were fruits and vegetables, which do not readily accumulate 2,3,7,8-TCDD in the environment. The percent contributions of ingestion exposure pathways to the Lifetime ADD are displayed in Exhibit 4-17.

Consistent with trends reported in the literature, consumption of meat, fish, and dairy products contribute to over 90 percent of lifetime dioxin exposure in the screening scenario. Daily intakes of 2,3,7,8-TCDD from milk, produce, and fish have been reported in the literature to comprise 27 percent, 11 percent, and 10 percent, respectively, of the total daily intake in the general population. However, some studies note that specific subpopulations, such as subsistence farmers and fishers, may have very different exposure profiles in which fish, meat, and dairy drive congener exposure (ATSDR 1998). Given the subsistence diet modeled in the RTR screening scenario, the high exposure from consumption of fish, dairy, and beef are appropriate within the context of this analysis.

Exhibit 4-16. Summary of Modeled 2,3,7,8-TCDD Concentrations and Observed Total Dioxin TEQ Concentrations in Ingestible Media

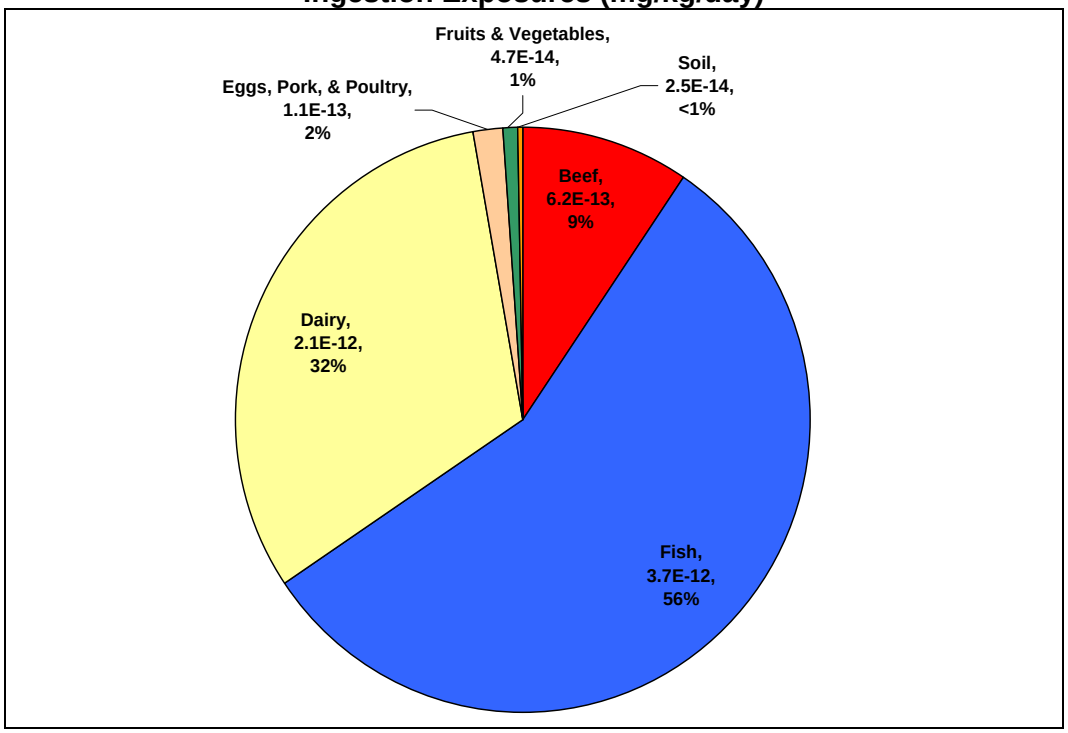


*except for soil, which is expressed as mg/kg dry weight.

[a] Source: *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds* (EPA 2004b)

[b] Source: *Toxicological Profile for Chlorinated Dibenzo-p-dioxins* (ATSDR 1998)

Exhibit 4-17. Contribution of Modeled Food Types to 2,3,7,8-TCDD Ingestion Exposures (mg/kg/day)



C-4.3.3.4 PAHs (Benzo[a]pyrene)

Behavior in the Environment

Emissions of POMs are often reported in terms of unspeciated or partially speciated groups, such as total PAHs, because they are often found in the environment as mixtures of two or more compounds exhibiting comparable behavior and toxicity. Because there are relatively few data concerning the behavior and toxicity of individual PAH compounds, EPA has proposed assigning a relative potency factor to PAHs based on the relative toxicity of these compounds to the most carcinogenic PAH compound(s) (EPA 1993). Like TEFs, this allows for risks from exposure to certain PAH compounds that are likely carcinogens to be determined relative to the toxicity of other PAH compounds that have been identified as probable carcinogens. Benzo[a]pyrene, a high molecular weight PAH identified by EPA as a probable carcinogenic compound (possibly the most potent carcinogen of the PAH group), was used to represent PAHs in the screening scenario. A relative potency factor of 1 has been proposed by EPA for this compound (EPA 1993). However, because the relative potency index has not been widely adopted by the scientific community, modeled concentrations of benzo[a]pyrene are mostly compared to reported concentrations of this species, rather than to total PAHs. Data for benzo[a]pyrene comprises much of the available exposure information on carcinogenic PAHs for the last few decades.

PAHs can enter the atmosphere as a result of a variety of combustion processes, both natural and anthropogenic. Based upon reviewed literature, stationary emission sources account for approximately 80 percent of total annual PAH emissions. Though the primary producer of stationary source PAH emissions is thought to be residential wood burning, other processes

such as power generation; incineration; coal tar, coke, and asphalt production; and petroleum catalytic cracking are also major contributors (ASTDR 1995). PAHs emitted to the atmosphere can travel long distances in vapor form or attached to particles, or they can deposit relatively close to an emission source via wet or dry deposition onto water, soil, and vegetation. In the atmosphere, PAHs are found primarily in the particle-bound phase, and atmospheric residence time and transport distances are highly influenced by climatic conditions and the size of the particles to which they are bound (ASTDR 1995).

As a result of sustained input from anthropogenic combustion sources and other sources, PAHs are ubiquitous in soil. Like other high molecular weight PAHs, benzo[a]pyrene strongly adsorbs to organic carbon in soil, indicating that adsorption to soil particles will limit the mobility of these compounds following deposition to soil. Most PAHs enter the water column directly through atmospheric fallout (ASTDR 1995). Following deposition onto surface waters, approximately two-thirds of PAHs adsorb strongly to sediment and suspended particles, while only small amounts revolatilize back to the atmosphere (ASTDR 1995). Aquatic organisms may accumulate PAHs via uptake of water, sediment, or food. Though fish and other organisms readily take up PAHs from contaminated food, biomagnification generally does not occur because many organisms are capable of rapidly metabolizing them (ASTDR 1995). As a result, concentrations of PAHs have generally been observed to decrease with increasing trophic levels (ASTDR 1995). Based upon observed data, bioaccumulation factors in fish are also not expected to be especially high because fish are able to readily metabolize the compound. BCFs in the range of 10-10,000 have been reported for fish and crustaceans, with the higher end of the range attributable to greater accumulation of the higher molecular weight compounds, such as benzo[a]pyrene (ASTDR 1995). Higher BCFs have also been observed in species at lower trophic levels, and BAFs will likely be higher in fish as a result of increased exposure from diet. Additionally, sediment-dwelling organisms may experience increased exposure to PAHs through association (e.g., direct uptake and/or consumption) with contaminated sediment (ASTDR 1995).

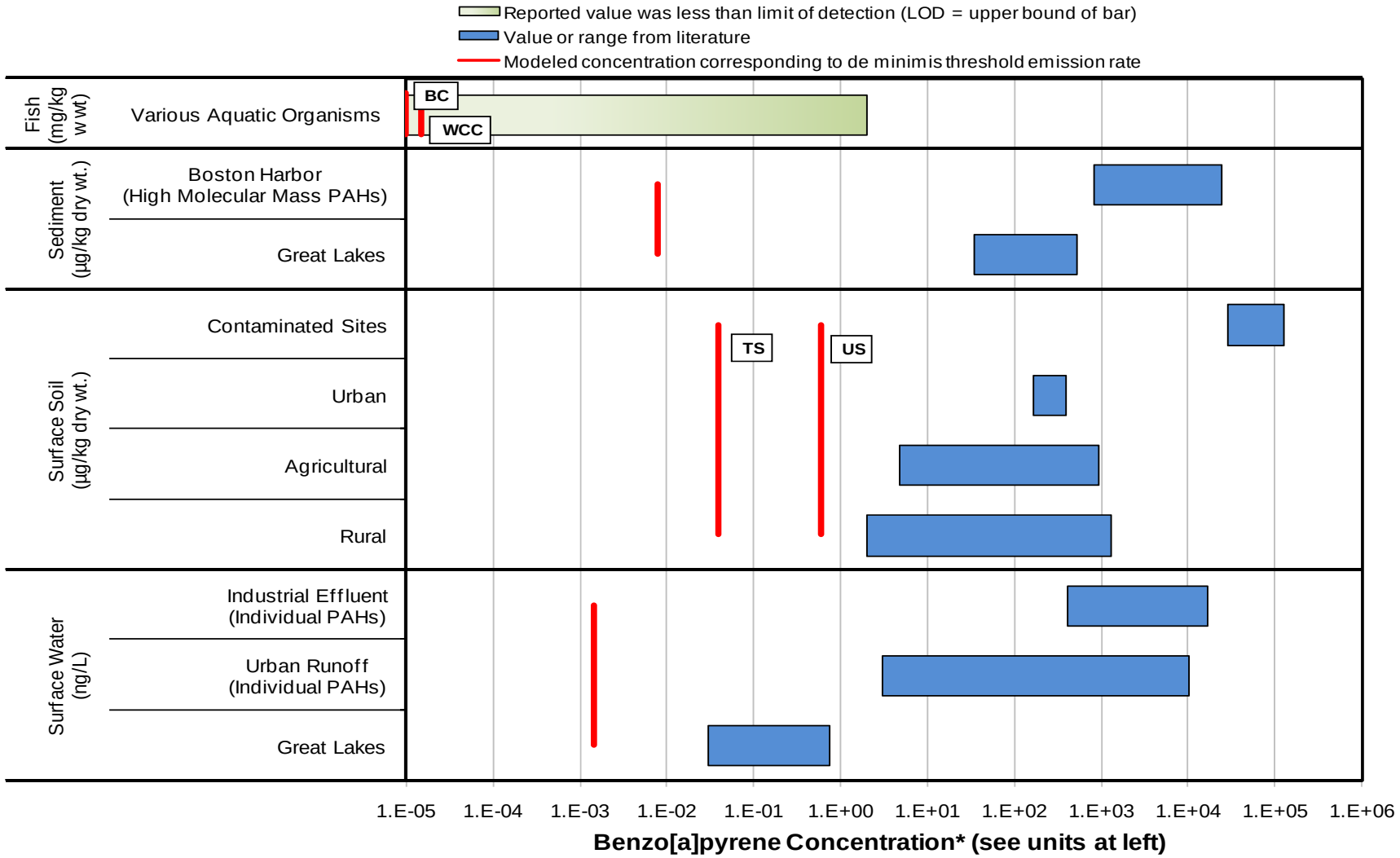
Emission Profile

The benzo[a]pyrene *de minimis* emission rate used for the RTR screening scenario is **2.3E-03 TPY** (based on lifetime cancer risk of 1-in-a-million). Although the annual concentration from year 50 is used for comparison, modeled concentrations benzo[a]pyrene either reached a steady state or had begun to level off in all compartments by year 10.

Concentrations in Environmental Media

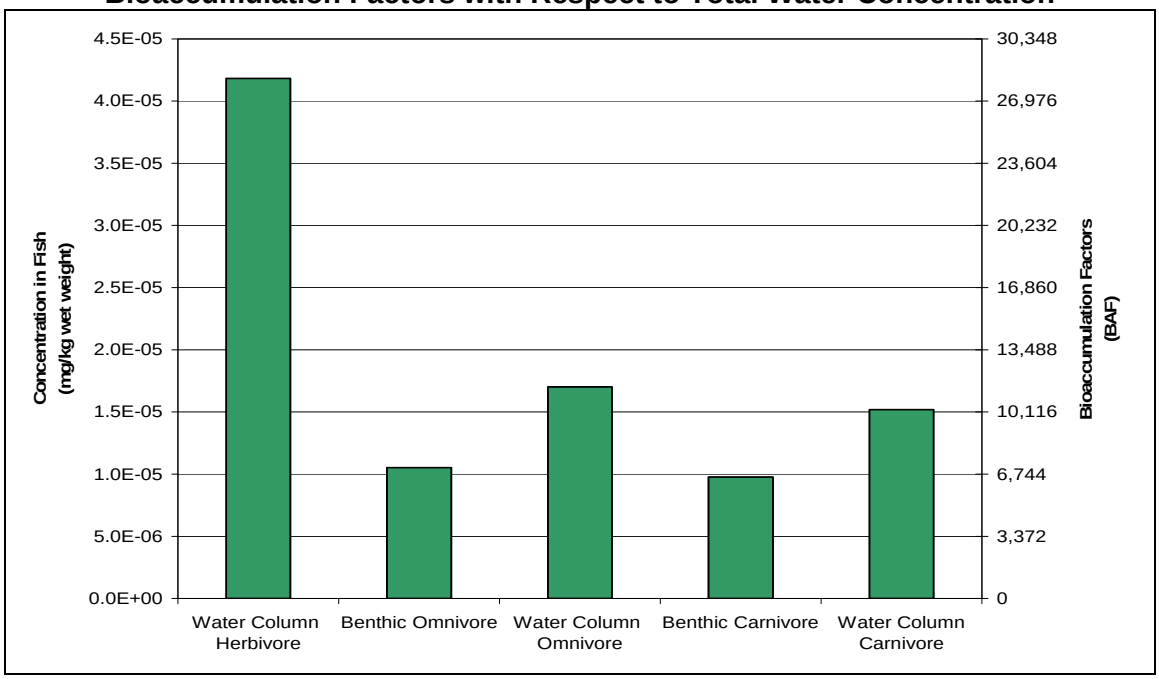
Measured ranges of benzo[a]pyrene (and occasionally PAH-group concentrations) in environmental media are presented in Exhibit 4-18 with concentrations from the screening scenario. Modeled concentrations and BAFs in fish compartments are shown in Exhibit 4-19. The benzo[a]pyrene concentrations output by the RTR screening scenario were consistently lower than values reported in the literature. Three main factors likely contributed to this trend. These are the high background values resulting from ubiquitous nature of PAHs in the environment, limited availability of benzo[a]pyrene-specific data, and use of conservative exposure factors to calculate the *de minimis* threshold. Firstly, due to the quantity of PAHs that are emitted from mobile sources (~20 percent), as well as stationary synthetic and natural sources, PAHs are typically present in the environment at relatively high background levels.

Exhibit 4-18. Summary of Modeled and Observed Concentrations of Benzo[a]pyrene in Environmental Media



* Source of literature data: *ATSDR Toxicological Profile for Polycyclic Aromatic Compounds (PAHs) (ATSDR 1995)*. Data are for benzo[a]pyrene (BaP) exclusively, unless otherwise noted.

Exhibit 4-19. TRIM.FaTE Benzo[a]Pyrene Concentrations in Fish and Calculated Bioaccumulation Factors with Respect to Total Water Concentration



The concentrations output from the screening scenario consider only emissions from a single facility. As a result, it is not unreasonable that some of the TRIM.FaTE outputs were several orders of magnitude smaller than those reported in the literature, as background exposure will be higher than incremental exposure. Secondly, though measured data for benzo[a]pyrene were used when available, concentrations in many media were available only for groups of PAHs or total PAHs. Thus, those ranges are not representative of benzo[a]pyrene alone and may contain values that are higher— and ranges that are wider— than those for a single chemical. Thirdly, it should be noted that in order to maintain a health-protective approach to screening emissions, a collection of moderately conservative exposure factors were used, which likely resulted in low, but not implausible values. For these reasons, we believe that the TRIM.FaTE outputs for concentrations of benzo[a]pyrene in environmental media are within the range of plausible values for this chemical. However, because of this discrepancy between the reported data and the screening scenario outputs, further investigation is necessary regarding site-appropriate biotransfer factors for facilities that do not pass the screen as a result of PAH emissions.

The screening scenario BAFs for benzo[a]pyrene in fish compartments were consistent with trends reported in the literature. BAFs for all but the WCH compartment were in the range of 5,500 to 11,500. No biomagnification of this chemical was exhibited in the fish compartments in the screening scenario, and the highest concentration of benzo[a]pyrene was in the WCH compartment, which represents the fish with the lowest trophic level evaluated in the scenario. The BAF for the WCH compartment in the screening scenario is approximately 28,000.

Partitioning Behavior

Data suggest that benzo[a]pyrene partitions mainly to soil (82 percent) and sediment (~17 percent) following deposition to a 1km^2 area adjacent to an emission source (ASTDR 1995). Once in soil, PAHs can volatilize, undergo abiotic or biotic degradation, accumulate in plants, or remain sorbed to soil organic matter. High molecular weight PAHs, such as benzo[a]pyrene, tend to adsorb more strongly to organic carbon than PAHs having lower molecular weights (ASTDR 1995). As a result of this affinity for organic carbon, volatilization is not an important loss mechanism for benzo[a]pyrene from soils (ASTDR 1995). Because of its low solubility and affinity for organic carbon, most benzo[a]pyrene is expected to be particle-bound in natural waters. Less than one-third of PAHs in aquatic systems are generally present in the dissolved phase (ASTDR 1995). The remainder may be associated with suspended particles in the water column or particles that have settled on the bed sediment. Because most of the benzo[a]pyrene found in natural water is in a sorbed state, the ultimate fate of most contaminant-laden particles is burial in the bed sediment. For the RTR screening scenario, >98.8 percent of benzo[a]pyrene in the pond compartment partitioned to the benthic sediment. The percentage of benzo[a]pyrene in sorbed and dissolved states in soil, surface water, and sediment are presented in Exhibit 4-19.

Exhibit 4-20. Fraction of Benzo[a]Pyrene Mass Sorbed vs. Dissolved in TRIM.FaTE Compartments

Compartment	Sorbed	Dissolved
Surface Soil	100.0%	0.0%
Surface Water	30.7%	69.3%
Sediment	100.0%	0.0%

The partitioning behavior of benzo[a]pyrene in the screening scenario is generally consistent with trends reported in the literature. The presence of this chemical in the sorbed state in the soil and sediment compartments is consistent with reported trends. A different trend was observed in the surface water compartment where more of the chemical was estimated by TRIM.FaTE to be dissolved. The dissolved concentration in TRIM.FaTE is affected by suspended sediment concentration, organic carbon content, and suspended sediment deposition and resuspension rates. Additional evaluation may be required to determine the specific factors affecting this behavior.

Concentrations in Ingestible Products

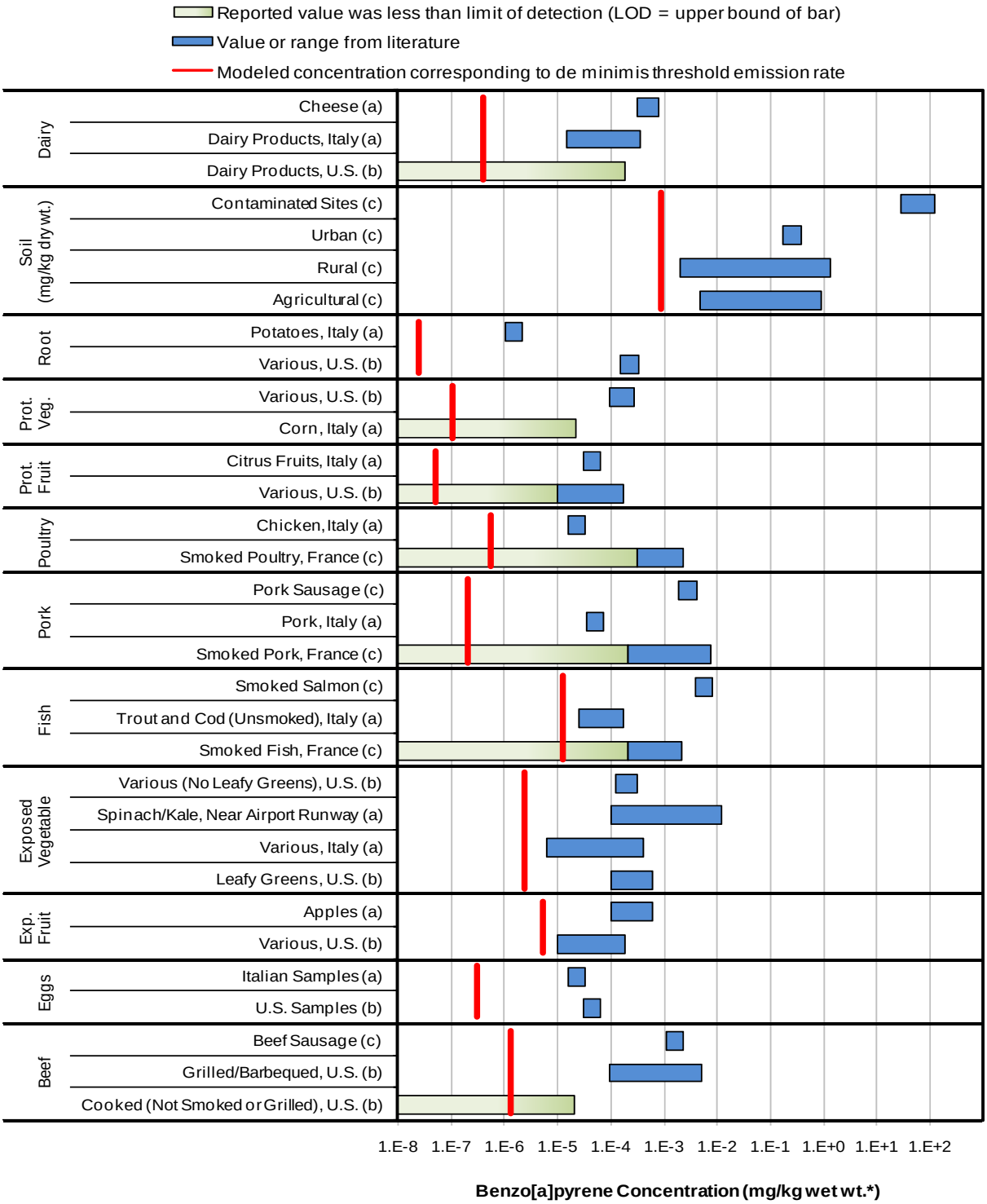
The primary source of non-inhalation exposure to benzo[a]pyrene outside of occupational settings is through dietary intake. Exposure may depend equally on the origin of the food (higher values often recorded at contaminated sites) and the method of food preparation (higher values reported for food that is smoked or grilled). PAHs have been observed to bioaccumulate in aquatic organisms and terrestrial animals through uptake of contaminated water, soil, and food. However, these compounds are readily metabolized by higher trophic level organisms, including humans, so biomagnification is not considered to be significant (ASTDR 1995). Plants accumulate PAHs primarily through atmospheric deposition, but chemical concentrations tend to be below detection levels. In general, grains and cereals may contain slightly higher concentrations of benzo[a]pyrene than fruits and vegetables. PAHs in meat have also been observed at concentrations below detection levels up to higher concentrations when the meat is smoked. Similar concentrations have been reported for fish, with smoked fish concentrations

sometimes quadruple those found in terrestrial animals. Because PAH concentrations are highest in products that are smoked or grilled, most of the available data for benzo[a]pyrene in food is for products that have been prepared using these processes. As a result, reported values may be significantly higher than those output by MIRC. Measured concentrations of benzo[a]pyrene in ingestible products are presented in Exhibit 4-21 along with RTR screening scenario concentrations.

The RTR screening scenario concentrations were generally lower than—or at the low end of—the reported ranges for benzo[a]pyrene in ingestible products. This trend is likely the result of background exposure in reported measurements and available data that is skewed toward concentrations in highly contaminated products. Considering these mitigating factors, the RTR screening scenario output concentrations are within the range of plausible values for benzo[a]pyrene in ingestible products. The percent contributions of ingestion exposure pathways to the lifetime ADD for benzo[a]pyrene are displayed in Exhibit 4-22.

No single exposure pathway in the RTR screening scenario appears to drive human exposure to benzo[a]pyrene, but dairy, vegetables, and fruits are the three most dominant pathways. This is consistent with observations indicating that only low concentrations of this chemical are present in most ingestible products due to the ability of most plants and animals to metabolizing it. It is also consistent with data suggesting that biotransfer factors between soil and plants/animals are relatively low.

Exhibit 4-21. Summary of Modeled and Observed Concentrations of Benzo[a]pyrene in Ingestible Media



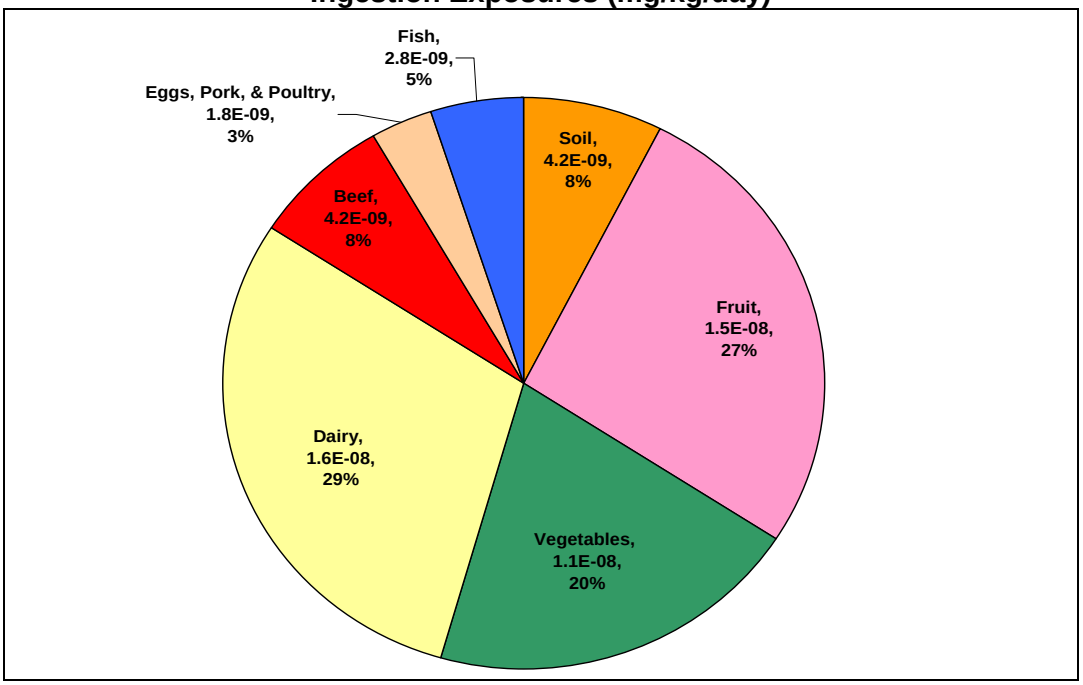
* except for soil, which is expressed as mg/kg dry weight

[a] Source: *Hazardous Substances Databank Record for Benzo[a]pyrene* (HSDB 2005c)

[b] Source: Analysis of 200 Food items for Benzo[a]pyrene and Estimation of its Intake in an Epidemiologic Study (Kazerouni et al. 2001)

[c] Source: *ATSDR Toxicological Profile for Polycyclic Aromatic Compounds (PAHs)* (ATSDR 1995)

Exhibit 4-22. Contribution of Modeled Food Types to Benzo[a]pyrene Ingestion Exposures (mg/kg/day)



C-4.3.4 Summary

This analysis compared outputs from the RTR screening scenario (using *de minimis* emission values) to observed data reported in the literature for cadmium, mercury, 2,3,7,8-TCDD, and benzo[a]pyrene. In general, most results from TRIM.FaTE do not appear to be unreasonable for a screening modeling approach based on this comparison to measured values. Briefly, the results of the evaluation are as follows:

- **Cadmium:** Modeled concentrations in environmental media and ingestible products and behavior with respect to partitioning and bioaccumulation appear to be reasonable.

- **Mercury:** Modeled concentrations in environmental media are comparable to levels for contaminated sites, and modeled concentrations in ingestible products are generally consistent with reported values. Speciation of mercury appears to be consistent with observed patterns in the environment.

- **2,3,7,8-TCDD**: Modeled concentrations in environmental media are consistent with observed TEQ values, and modeled concentrations in ingestible products are slightly lower than reported values, but still within a reasonable range. Both modeled values and measured values were primarily located in the noise around the limit of detection, which increases the uncertainty of the data.

- **Benzo[a]pyrene:** Modeled concentrations in both environmental media and ingestible products are generally lower than those reported in the literature, in some cases by more than two orders of magnitude. Because this chemical is found in the environment at background levels that would far exceed concentrations resulting from single facility-

emissions, a comparison to measured concentrations is not as informative for this chemical.

Note that the exposure factors driving farm food chain ingestion rates are also conservative, and that these parameters were not considered in this evaluation, meaning that the "actual" concentration associated with a given risk level (if such a value could be calculated) would be higher than the level associated with the modeled *de minimis* rate. Also, even "correct" model results may mask incorrect model assumptions that combine to produce compensating errors. Where results differ significantly from reported values, such as with benzo[a]pyrene, a more detailed investigation of underlying assumptions may be necessary to determine appropriate adjustments to the scenario configuration. As noted in the overview of this section, this type of evaluation cannot be used to verify model results.

C-4.4 Sensitivity Analyses

The hypothetical subsistence farmer/fisher exposure scenario used to screen emissions of PB-HAPs described in this document was parameterized using generally conservative inputs. The goal was to construct a modeling scenario that is sufficiently health-protective (i.e., conservative) such that it can be used with confidence to screen out emissions that do not pose unacceptable multipathway risks, while also avoiding overly conservative characteristics that diminish the functionality of the scenario (i.e., by allowing too many "false positives," or facilities that fail the screen for which the risks are actually acceptable). The level of conservatism of the scenario is dictated largely by the values selected by the user for model inputs. A sensitivity analysis is a useful method for evaluating the influence of model parameters and user selections for parameter values. By providing quantitative information on the importance of model parameters to a selected output, a sensitivity analysis thus also provides information on which parameters may be most influential in dictating the uncertainty associated with the results.

The sensitivity analyses conducted on the RTR screening modeling scenario encompassed the fate and transport modeling carried out using TRIM.FaTE and the farm food chain (FFC) and ingestion exposure modeling performed using MIRC.[17] A systematic sensitivity analysis was conducted on these parameters to obtain information regarding the relative importance of user inputs (Section C-4.4.1). In addition, several other analyses were performed to evaluate model performance with respect to parameterization of ingestion rates (Section C-4.4.2), body weight (Section 0), assumed relationships between ingestion and body weight over time (Section C-4.4.4), and meteorological conditions (Section C-4.4.5). Each section presents the methods employed to conduct the sensitivity analyses, evaluation results, and notes regarding interpretation of the results. Results of these sensitivity analyses are useful in informing the level of uncertainty in screening results, highlighting parameters and aspects of the modeling scenario worthy of additional research if refinement is appropriate (e.g., for evaluating sources of PB-HAPs that do not pass the screen), and suggesting variables that are likely to be worthy of more detailed examination if a more quantitative uncertainty analysis is desired (e.g., using probabilistic methods).

[17] PB-HAP dose-response values used for risk calculation (i.e., oral cancer slope factors and oral reference doses, plus the mutagenicity correction factor applied to certain age groups) were not included in the sensitivity analysis.

C-4.4.1 Systematic Sensitivity Analysis

C-4.4.1.1 Methods

As with all predictive models, the user-specified inputs for TRIM.FaTE and MIRC influence model outputs and thus estimation of *de minimis* screening thresholds. The degree to which model outputs diverge from a nominally "correct" answer (e.g., the actual incremental risks for an individual exposed to PB-HAP emissions) encompasses both uncertainty (because values are not known with perfect accuracy) and variability (because each value selected represents a discrete choice out of a range of possible parameter values within a distribution, such as variability in body weights among individuals in an exposed population). In this analysis, we did not attempt to separately evaluate uncertainty and variability, and variation in model outputs is assumed to encompass both uncertainty and variability. Representation of each parameter with a single fixed value without representing uncertainty or variability in model calculations is a limitation of deterministic model application.

As a first step to quantifying the combined uncertainty and variability in model outputs and the *de minimis* thresholds, an analysis was performed to identify the variables to which the risk or hazard quotient calculations are most sensitive. Model sensitivity was evaluated separately for each of the PB-HAPs. The model output to which sensitivity was measured varied by chemical depending on the health impacts of greatest concern. Parameter influence on cancer risk was measured for benzo[a]pyrene and 2,3,7,8-TCDD, and influence on non-cancer hazard quotient was measured for cadmium, divalent mercury, and methyl mercury. This part of the analysis was conducted systematically by "perturbing," or changing, the value of each selected parameter independently (i.e., one at a time, holding all other inputs at their base value) by a certain percentage and calculating the corresponding percent change in the risk or hazard quotient value. This metric is referred to as elasticity (i.e., ratio of the percent change of the model output to percent change in the input variable), with higher elasticities corresponding to greater influence.

This type of systematic sensitivity analysis has the advantage of focusing on a wide range of inputs at once so that the variables can be ranked in order of importance. However, by perturbing the variables by fixed percentages, the analysis does not necessarily focus on the most physically relevant values of each parameter. An alternate analysis could examine each variable independently (taking into account the plausible range of input values) and look at the effects of using different plausible variable values on the risk or hazard quotient estimates. We focus on a systematic sensitivity analysis here with the goal of prioritizing variables; additional examinations may be helpful in the future to better define the uncertainty.

In the systematic sensitivity analysis, we estimated both local sensitivity, quantified as the elasticity when a parameter value is perturbed by a small percentage of its base (or default) value, and range sensitivity, for which parameters were varied by a larger percentage of the base value. These two elasticities can be different if the relationship between input and output is nonlinear (e.g., if the perturbed parameter is in the denominator of an equation in the calculation). For the evaluation of risks associated with exposures to PB-HAPs, a large difference between local and range sensitivity could indicate that variables that are less important in the base scenario used to calculate *de minimis* thresholds scenario assumptions may be more important in a less conservative site-specific evaluation. In this case, following recommendations in the *Risk Assessment Guidance for Superfund* (EPA 2001; see Volume III, Part A, Appendix A), we perturbed parameter values by +/-5% of the base case default value in the local sensitivity analysis and +/-50% in the range sensitivity analysis. Thus, with some

exceptions noted below, this resulted in four model simulations and corresponding elasticities for each parameter included in the analysis for each of the PB-HAPs.

Typically, a range sensitivity analysis (also referred to as "nominal sensitivity analysis") measures the effect on model outputs across the entire expected or plausible range of values for a given input. This provides more comprehensive information on the behavior of the model with respect to the input being varied by demonstrating the maximum potential influence of that input on model outputs. In the case of the RTR modeling scenario, information on the expected or most likely input ranges is available on only a few of the parameters defined by the user, and there is a relatively large number of parameters to be evaluated. The selection of both 5 and 50 percent as perturbation increments was intended to be an efficient compromise between conducting an authentic range sensitivity analysis and evaluating only more localized sensitivity.

To obtain a comprehensive estimate of the relative sensitivity of risk results across the range of numerical user inputs, as many TRIM.FaTE and MIRC inputs as possible were included in the analysis. To that end, all the MIRC ingestion and exposure variables were included. For TRIM.FaTE, properties assumed *a priori* to have greater influence on model outputs were included. The large number of user-specified numerical inputs to TRIM.FaTE and computational limitations made inclusion of all TRIM.FaTE inputs in this sensitivity analysis impractical. TRIM.FaTE inputs for this sensitivity evaluation were selected based on results obtained from previous TRIM.FaTE evaluations (including the TRIM.FaTE mercury test case; EPA 2005b) and professional judgment/intuition drawing on experience with the model.

The full set of inputs included in the systematic analysis is shown in Attachment C-3, Exhibit 1. Parameters are grouped by the model in which they are used, with the MIRC variables further divided into farm food chain and ingestion/body weight categories. In some cases, inputs included in the analysis could not be increased by either 5% or 50% or both, since the variable has a physical upper bound which cannot be exceeded (e.g., the number of days of exposure per year was already set to 365, so it cannot be increased by 5% or 50%). The footnotes in Attachment C-3, Exhibit 1 indicate the variables that could not be increased by 5% and/or 50%. For some inputs, the ability to perturb the input depends on the PB-HAP being modeled, as indicated. In addition, some inputs were assigned a baseline value of zero for estimating *de minimis* threshold (e.g., the empirical correction factor for protected vegetables and the exposure variables related to the water pathway). These variables are not included in the systematic sensitivity analysis. In all, approximately 240 variables were examined.

Two types of inputs included in the systematic analysis, pH and the moisture adjustment factor (MAF), received special treatment.

- pH, which is a user input for the surface soil, root zone soil, surface water, and sediment compartments, is measured on a logarithmic scale, and this input is (in some cases) used in the exponent of TRIM.FaTE algorithms. To obtain sensitivity metrics for pH inputs that can be compared to results for other inputs with more linear relationships with risk, the values for pH were changed to the equivalent hydrogen ion concentration (that is, 10^{pH}) and that value was varied by 5 or 50%. Then, the log base ten of the varied hydrogen ion concentration was calculated to serve as the pH to use in TRIM.FaTE for the sensitivity case.

- The moisture adjustment factor indicates the percent of a produce item that is water; this factor is used to convert from wet weight to dry weight concentration in MIRC. This calculation is made by finding the percent of the produce that is not water (i.e., 100 minus MAF) and multiplying by the wet-weight basis concentration. Because the physical

variable of interest is really the fraction of produce that is NOT water, this value (100 minus MAF) was used in the sensitivity analysis as the base value (rather than MAF itself).

In addition, in two cases, known or assumed correlations between inputs were taken into account in the systematic sensitivity analysis.

- The fractions of T3 and T4 fish that are consumed must add to one in MIRC; thus, when one was changed by a given percentage, the other was also changed to ensure the sum was still one.

- In parameterizing several sediment and water inputs used in TRIM.FaTE, soil/sediment and runoff/surface water balance is assumed. These relationships are not explicitly accounted for by TRIM.FaTE but rather were calculated off-line prior to setting the input value in the model. TRIM.FaTE variables subject to soil or sediment balances include erosion rate, precipitation rate, runoff rate, sediment deposition velocity, suspended sediment concentration, sediment porosity, water retention time in the pond, and the suspended sediment concentration. For example, increasing the sediment deposition velocity results in an increase in the sediment resuspension velocity to ensure the sediment mass balance is preserved (the specific relationships assumed between inputs are described elsewhere). These correlations were respected in the systematic sensitivity analysis such that when an independent input was perturbed, any dependent input variables were altered correspondingly to preserve the water and sediment balances.

Other TRIM.FaTE and MIRC inputs are also related. Some of the relationships involving ingestion rates and body weight were evaluated in the context of the sensitivity analysis, as described in the sections that follow. In general, however, the current analysis did not endeavor to determine additional correlations or account for them in the sensitivity calculations conducted.

C-4.4.1.2 Results

Exhibit 4-23 through Exhibit 4-27 display the variables with the highest elasticities for each PB-HAP. The inputs were sorted according to the elasticities estimated for a decrease in input by 5%. This case (as opposed to the case where variables were increased by 5%) was chosen because all inputs, including those for which the base case value was set at the maximum, could be part of the sorting procedure. Parameters were sorted by absolute value of elasticity, and the bars in the charts are color coded to distinguish whether the input is used in TRIM.FaTE, MIRC farm food chain calculations, or MIRC ingestion exposure calculations. The top 25 inputs are shown for all PB-HAPs; for some chemicals, additional inputs are included if more than one input corresponded to the 25^{th} highest elasticity.

In general, mixing height, emission rate, and horizontal wind speed always appear near the top of the ranking for all PB-HAPs analyzed. The emission rate elasticity is always 1.00, indicating that a 5% reduction in the emission rate gives a 5% reduction in the risk or hazard quotient. The mixing height and horizontal wind speed elasticities are always negative and are either above or below 1.00. When the mixing height is decreased, the PB-HAP emissions spread over a smaller volume and more of the PB-HAP mass remains near the surface for deposition. When the horizontal velocity decreases, the PB-HAP remains within the model domain for a longer time and the concentrations in the model compartments increase accordingly.

A fourth input, sediment deposition/resuspension rate, has an elasticity greater than one in magnitude for methyl mercury. This sensitivity case represents a decrease in the user-specified

input for sediment deposition velocity by 5% and a concurrent 7% decrease in the resuspension velocity (calculated to maintain the sediment balance[18]). This value is particularly important for methyl mercury HQ because methylation of mercury occurs to a large degree in the sediment, and a decrease in deposition velocity results in a larger, proportionate decrease in the resuspension velocity, indicating a longer residence time in the sediment and a higher degree of methylation.

Only the above four inputs (mixing height, emission rate, horizontal wind speed, and sediment deposition) have absolute elasticities greater than or equal to one. Beyond these, the inputs with the highest elasticities are those that influence the primary exposure pathways for each PB-HAP. Primary ingestion exposure pathways (in this case defined as the food categories that account for 75% to 85% of the total risk or hazard) are indicated at the top of each of the charts.

- For benzo[a]pyrene (Exhibit 4-23), the primary pathways are ingestion of fruits, vegetables, and dairy products, and most of the inputs with the highest elasticities are used in farm food chain and ingestion exposure calculations. Because wet deposition is of particular importance, the exposed fruits and vegetables represent the more sensitive pathway compared with the protected fruits and vegetables. In addition, the analysis reveals that within the food chain diet of the dairy cows, risk is most sensitive to the inputs used to estimate chemical transfer via the cow's forage pathway (as opposed to the silage or grain pathways).

- Sensitivity results obtained for 2,3,7,8-TCDD (Exhibit 4-24) are consistent with the observation that the primary pathways are fish, beef, and dairy ingestion. Results also reinforce the conclusion that cancer risk is more sensitive to the forage pathway for dairy and beef than the grain or silage pathways.

- Sensitivity results for cadmium (Exhibit 4-25) reflect the primary pathways of fruits, vegetables, and fish, but in this case the hazard quotient is more sensitive to the protected fruits and vegetables (and thus also the soil variables) than the exposed fruits and vegetables. The HQ is also more sensitive to variables affecting the T3 fish than the T4 fish, consistent with higher cadmium concentrations estimated by TRIM.FaTE for that fish type.

- Elasticities for divalent mercury (Exhibit 4-26) reflect the primary pathways of soil, fruits, and vegetables through the importance of the rain and erosion variables and the protected fruit and vegetable variables. Finally, results for methyl mercury (Exhibit 4-27) are consistent with fish consumption as the primary pathway, with inputs specific to T4 fish more important than those specific to T3 fish due to the higher bioaccumulation potential (and therefore exposures) associated with higher trophic level fish.

Looking across the different PB-HAPs, several of the TRIM.FaTE variables with high elasticities are highly dependent on assumptions made in configuring the TRIM.FaTE modeling scenario, including mixing height, horizontal wind speed, rain rate, dry deposition velocity, surface water retention time (correlated directly with pond depth and other inputs given the water balance that is assumed), and surface water temperature. These inputs, which would likely be set differently

[18] The sediment balance maintains a zero net flux of sediment mass into the sediment at all times (i.e., a steady state) by balancing the deposition, resuspension, and burial fluxes. The burial flux is calculated by subtracting the amount of sediment flushed from the pond from the amount introduced into the pond via erosion at every time step. The deposition rate is specified. Then, the resuspension rate is calculated by adding the burial and deposition fluxes to ensure no net flux into or out of the sediment.

for a site-specific analysis, represent influential parameters worthy of additional evaluation to quantify the conservative nature of the scenario. The area of the parcels and the temporal pattern of the precipitation may also be influential; however, these were not evaluated quantitatively in the systematic sensitivity analysis given the complexity involved with adjusting these inputs.

Differences between the results of the local and range sensitivity analyses are presented in Attachment C-3, Exhibits 2 through 6. These tables show elasticities and elasticity rankings by PB-HAP for each input that appears in the top 25 elasticities for all four analyses (i.e., perturbation by -50%, -5%, 5%, and 50% of the base case value). The rankings for some inputs are somewhat different across the four cases, but no drastic differences are noted when comparing the range and local sensitivity analyses for any of the PB-HAPs. This suggests the local sensitivity analysis may be sufficient for drawing some conclusions about the relative influence of user-specified inputs on the risk and hazard quotient estimates.

The results of this sensitivity analysis indicate the 25-30 variables to which the risk and hazard quotient estimates are most sensitive. In proceeding with a probabilistic analysis which would quantify the uncertainty in the model estimates, these variables would be of primary importance and should be the focus when developing input variable distributions. Further research could also confirm that these variables are set in an appropriately conservative fashion for the purposes of developing a screening scenario.

Exhibit 4-23. The 26 Variables with the Highest Elasticities for Benzo[a]Pyrene Lifetime Risk (-5% Perturbation of Variable)
Primary Pathways: Fruits, Vegetables, and Dairy

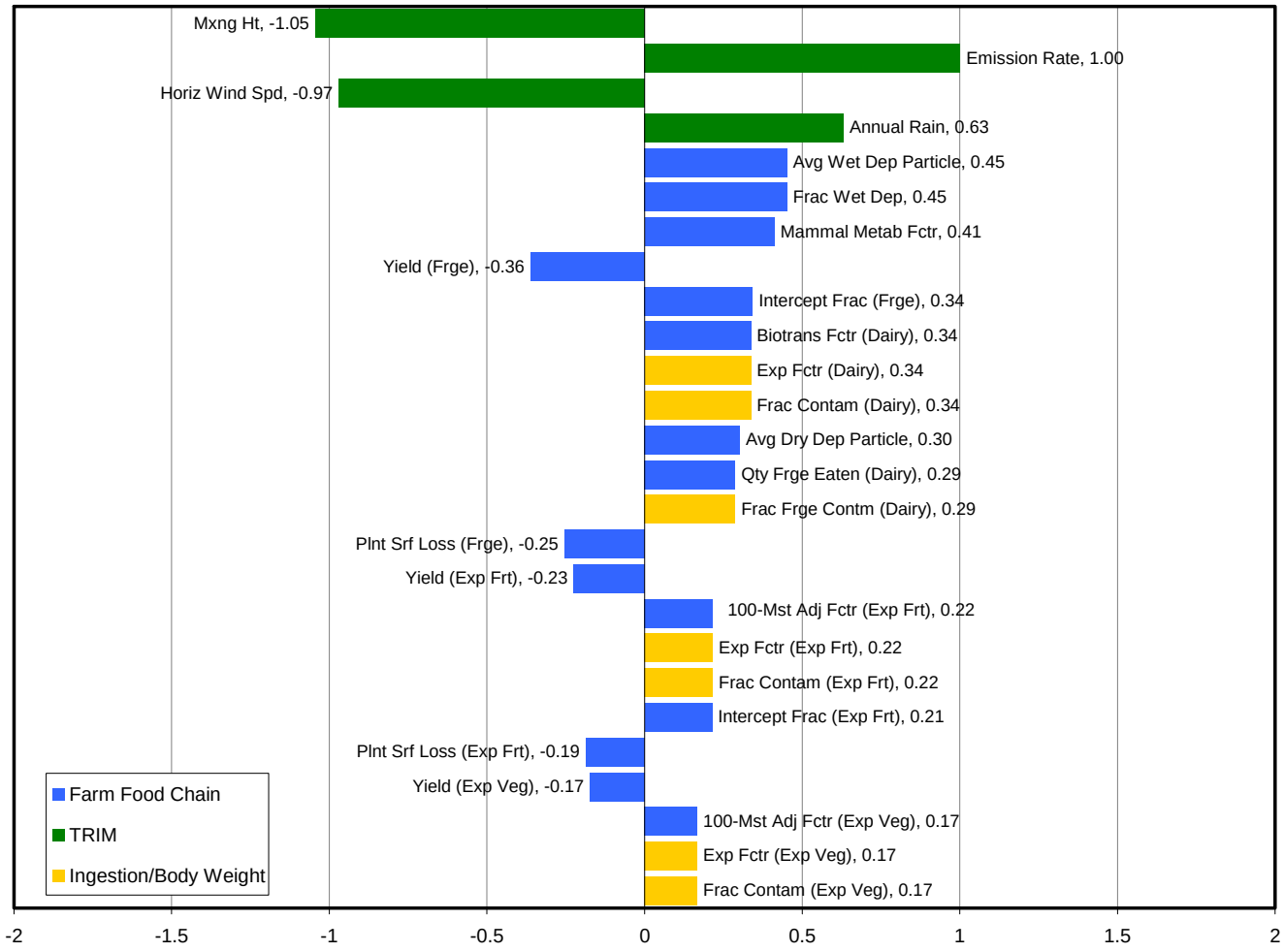


Exhibit 4-24. The 25 Variables with the Highest Elasticities for 2,3,7,8-TCDD Lifetime Risk (-5% Perturbation of Variable)
Primary Pathways: Fish, Beef, and Dairy

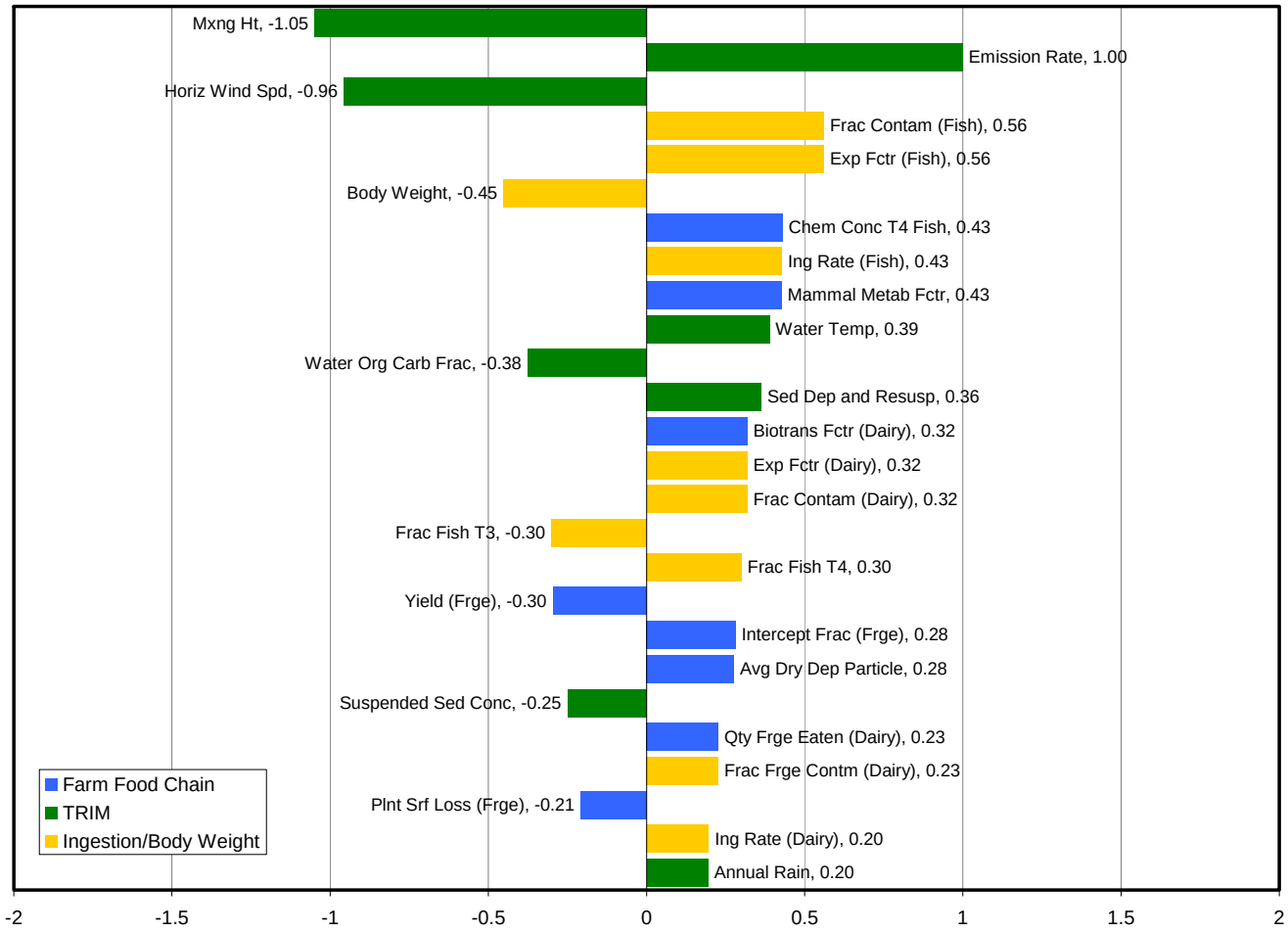


Exhibit 4-25. The 28 Variables with the Highest Elasticities for Cadmium Hazard Quotient for Child 1-2 (-5% Perturbation of Variable)

Primary Pathways: Fruits, Vegetables, and Fish

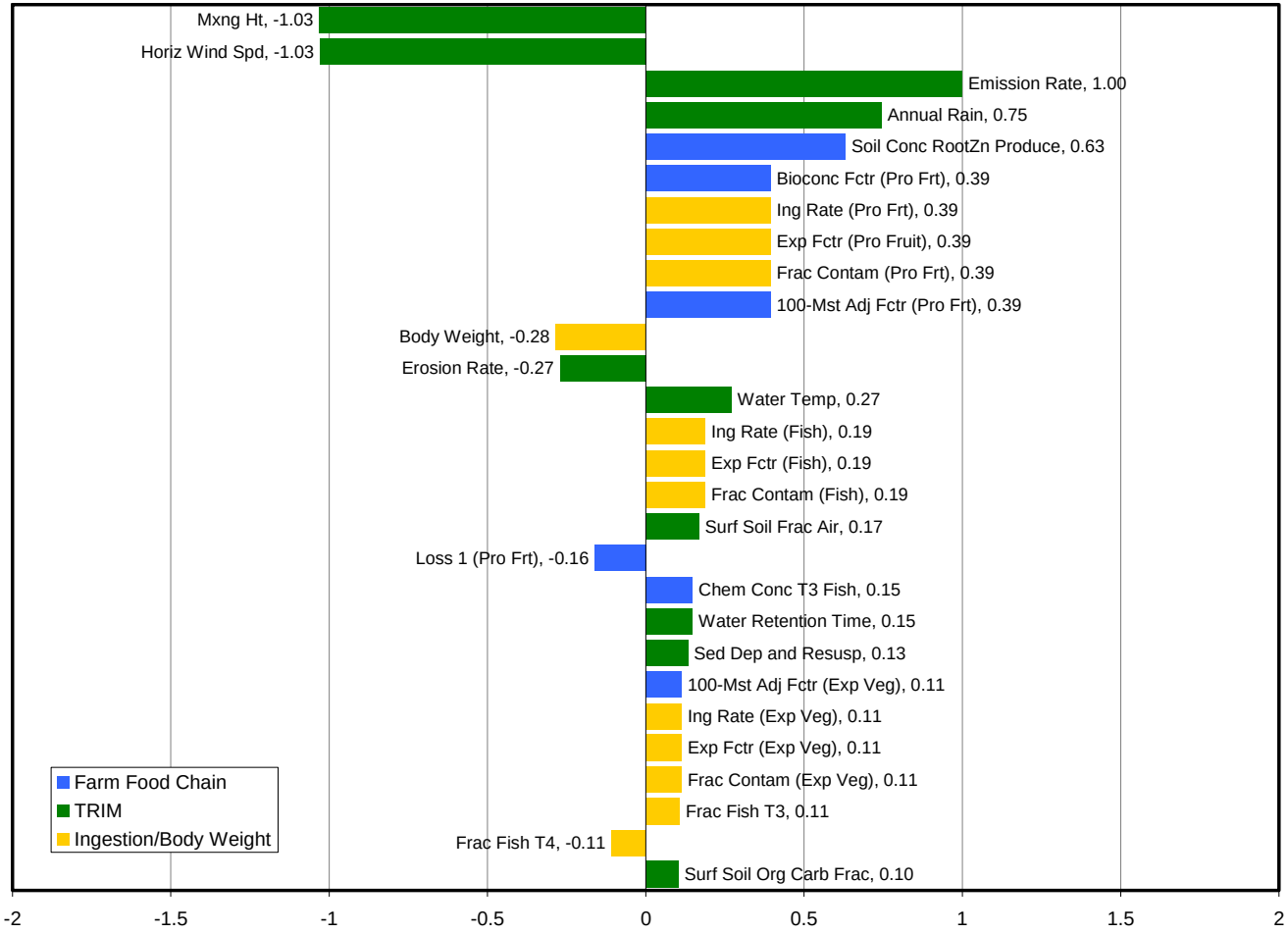


Exhibit 4-26. The 25 Variables with the Highest Elasticities for Divalent Mercury Hazard Quotient for Child 1-2 (-5% Perturbation of Variable)

Primary Pathways: Soil, Fruits and Vegetables

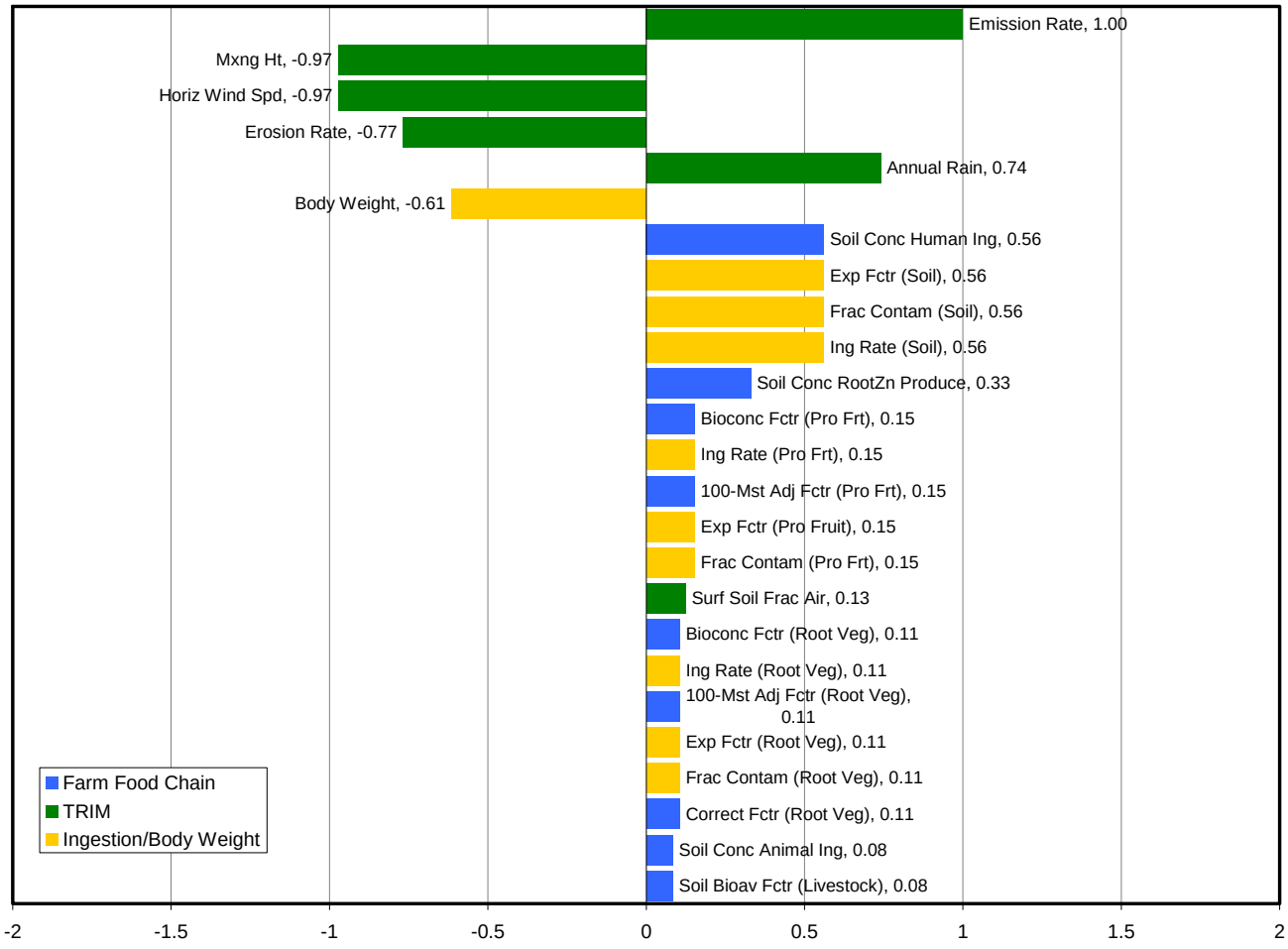
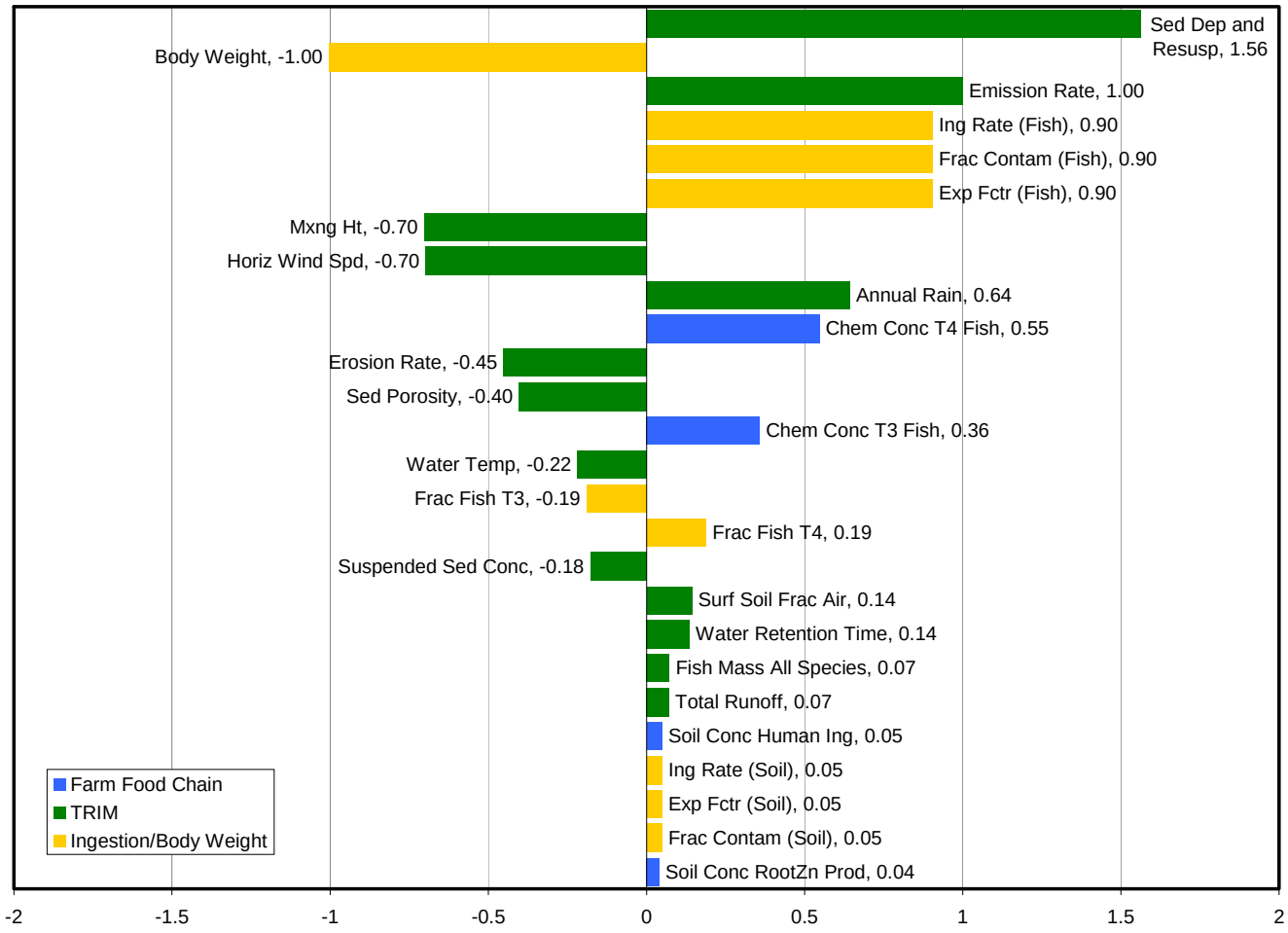


Exhibit 4-27. The 26 Variables with the Highest Elasticities for Methyl Mercury Hazard Quotient for Child 1-2 (-5% Perturbation of Variable)

Primary Pathway: Fish



C-4.4.2 Evaluation of Ingestion Rate Assumptions

To calculate the *de minimis* levels, the conservative assumption was made that a person ingests food at the 90th percentile level for each food type included in the analysis. These ingestion rates can then be added to get the assumed total ingestion rate.[19] USEPA 2008 and 2005d provide estimates of the 90th percentile total ingestion rate for different age groups (that is, percentiles for total amount of food ingested, rather than a sum of individual percentiles) based on CSFII data 1994-96 and 1998 (see Exhibit 6-12 of Attachment C-2). When comparing the two, the *de minimis* total ingestion rates tend to be a factor of two to three times higher than the 90th percentile total ingestion rate (see Exhibit 7-1 of Attachment C-2). An alternative approach that still captures the high-end would be to use the 90th percentile ingestion rates for food types associated with highest risks for a given PB-HAP (to account for the individual who may happen to eat higher-than-typical amounts for each of those food categories) while setting ingestion rates for other food types to a lower percentile in order to bring the total ingestion rate to a more realistic level (e.g., to something closer to the 90th percentile total ingestion rate).

In order to determine the effect of lowering the ingestion rates for the non-primary ingestion pathways (and provide information on whether this refinement would be worth the additional complexity in the model set-up), ingestion rates were kept at the 90th percentile for the primary (risk-driving) food types for each PB-HAP but were reduced to median ingestion rates for the same population (subsistence famers) for the other types of food. This scenario, referred to here as the "alternate ingestion case," was evaluated for each PB-HAP. Specifically, the total food ingestion rates was calculated for the alternate ingestion cases for each PB-HAP, and these values were compared with the USEPA 2008 and 2005d estimates of 90th percentile total ingestion (referred to as the "EPA total ingestion rate") via a ratio (Exhibit 4-28). Because the EPA total ingestion rate represents the total amount of *cooked* food consumed, preparation losses were applied to each of the food category-specific ingestion rates input into MIRC before the ratio was calculated. Soil consumption was not included in these calculations. In addition, the estimated *de minimis* cancer risk or HQ for each PB-HAP was compared to the recalculated risk/HQ for the alternate ingestion cases (Exhibit 4-29).

As expected, the ratios presented in Exhibit 4-28 demonstrate that using the alternate ingestion rate combination tends to result in lower estimated total ingestion rates, approaching the (more realistic) USEPA total ingestion rates for the child age groups. The degree to which the total ingestion rate is decreased depends on the number of pathways labeled "primary pathways" for risk (as well as the mass of food represented by a given pathway). Thus, it is reasonable that lower total ingestion rates (and lower ratios with respect to EPA total ingestion rate) are calculated for methyl mercury, for which only one pathway (fish) drives risk, and higher rates and ratios are calculated for benzo[a]pyrene, for which several food types are important.

Exhibit 4-29 compares the lifetime risk or child age 1-2 HQ for the alternate ingestion rate case to the corresponding risk/HQ for the *de minimis* case. Overall, the changes in risk and HQ were relatively modest, ranging between 7% and 20%. This result indicates that relaxing the conservative assumption of high ingestion rates for all the pathways does not significantly change the risk and hazard calculations but does drive the total ingestion rates closer to the USEPA 90th percentile total ingestion rates. However, implementing this multi-percentile technique requires prior knowledge of the dominant pathways for each chemical, which is determined primarily from the model estimates themselves. Assuming 90th percentile ingestion

[19] This calculated total ingestion rate will not include any other food consumed by the homestead family outside of the MIRC food categories. In making the comparison with the total ingestion rates, then, the assumption is made that the entire diet of the family is captured by the MIRC food categories.

rates for all food categories has the advantage of simplicity (using the same set of ingestion rates for all chemicals) and does not dramatically impact the risk and hazard quotient estimates.

Exhibit 4-28. Ratio of the Modeled Total Ingestion Rates and the USEPA Total Ingestion Rates

	Ratio of Ingestion Rates				
	Child (1-2)	**Child (3-5)**	**Child (6-11)**	**Child (12-19)**	**Adult (20-70)**
Benzo[a]Pyrene Alternate Ingestion	1.7	1.3	1.4	1.4	2.6
2,3,7,8 - TCDD Alternate Ingestion	1.3	1.0	1.0	1.0	1.8
Cadmium Alternate Ingestion	1.3	1.1	1.2	1.0	1.6
Divalent Mercury Alternate Ingestion	1.3	1.1	1.1	1.0	1.6
Methyl Mercury Alternate Ingestion	0.9	0.8	0.8	0.6	0.8
de minimis Ingestion	1.8	1.4	1.6	1.5	2.8

Exhibit 4-29. Comparison of the Risks and Hazard Quotients in the *de minimis* and Alternate Ingestion Cases

PB-HAP	Categories with 90th Percentile Ingestion in Alternate Case [a]	Categories with Median Ingestion Rates in Alternate Case [a]	Case	Value
Benzo[a]pyrene	Pro. fruit, pro. veg., exp. fruit, exp. veg., root veg., and dairy	Soil, fish, beef, pork poultry, and eggs	*de minimis* lifetime risk	1.00E-06
			Alternate ingestion case	7.98E-07
			Percent Change in Risk	**-20.2%**
2,3,7,8 - TCDD	Fish and dairy	Soil, pro. fruit, pro. veg., exp. fruit, exp. veg., root veg., beef, pork, poultry, and eggs	*de minimis* lifetime risk	1.00E-06
			Alternate ingestion case	9.14E-07
			Percent Change in Risk	**-8.6%**
Cadmium	Fish, pro. fruit, pro. veg., exp. fruit, exp. veg., and root veg.	Soil, beef, dairy, pork, poultry, and eggs	*de minimis* child (1-2) hazard quotient	1.00E+00
			Alternate ingestion case	9.05E-01
			Percent Change in Risk	**-9.5%**
Divalent Mercury	Soil, pro. fruit, pro. veg., exp. fruit, exp. veg., and root veg.	Fish, beef, dairy, pork, poultry, and eggs	*de minimis* child (1-2) hazard quotient	1.00E+00
			Alternate ingestion case	9.26E-01
			Percent Change in Risk	**-7.4%**
Methyl Mercury	Fish	Soil, pro. fruit, pro. veg., exp. fruit, exp. veg., root veg., beef, dairy, pork, poultry, and eggs	*de minimis* child (1-2) hazard quotient	5.62E-01
			Alternate ingestion case	5.19E-01
			Percent Change in Risk	**-7.8%**

[a] Pro. fruit is protected fruits, pro. veg. is protected vegetables, exp. fruit is exposed fruits, exp. veg. is exposed vegetables, and root veg. is root vegetables.

C-4.4.3 Evaluation of Body Weight Assumptions

As stated in Attachment C-2, Section 6.1.3, the *de minimis* rates were calculated using mean body weight following EPA's default screening recommendation. This assumption does not represent the most conservative assumption, however, since lower percentile body weights will give higher risk and hazard quotient estimates. In contrast, the ingestion rates were set to the more conservative 90th percentile level. To investigate the sensitivity of using alternate body weight percentiles, an alternate MIRC run was performed using the 5th, 50th, 90th, and 95th percentiles for body weight while keeping all other variable values as specified in the *de minimis* calculations. These alternate risk or hazard quotient model estimates and the percent change relative to the *de minimis* calculations are shown in Exhibit 4-30. Changing from the mean to the median body weights produces only very modest changes to the risks/hazard quotients. Changing to the 5th percentile body weights gives the most conservative model estimates, with percent changes of up to 22% for the PB-HAPs used to set the *de minimis* rates and a 26% change in methyl mercury. Using upper percentiles for body weights produces modest decreases in risk or hazard quotient of 3% to 16% for the PB-HAPs used to set the *de minimis* rates. Thus, changing the body weight to either a more conservative or less conservative percentile produces changes in the risk or hazard quotient which are appreciable but not

dramatic (i.e., typically less than 25%), suggesting the mean body weight assumption may be an appropriate choice for the screening scenario.

Exhibit 4-30. The Risk or Hazard Quotient Estimates Using Alternate Body Weight Percentiles

	de minimis	5th Percentile		Median		90th Percentile		95th Percentile	
		Risk or HQ [a]	% Ch [b]	Risk or HQ [a]	% Ch [b]	Risk or HQ [a]	% Ch [b]	Risk or HQ [a]	% Ch [b]
Benzo[a]pyrene [c]	1.0E-06	1.1E-06	6%	1.0E-06	0%	9.7E-07	-3%	9.6E-07	-4%
2,3,7,8-TCDD [c]	1.0E-06	1.2E-06	22%	1.0E-06	2%	8.8E-07	-12%	8.4E-07	-16%
Cadmium [d]	1.00	1.07	7%	1.00	0%	0.96	-4%	0.95	-5%
Divalent Mercury [d]	1.00	1.16	16%	1.00	0%	0.91	-9%	0.89	-11%
Methyl Mercury [d]	0.56	0.71	26%	0.57	1%	0.48	-15%	0.46	-18%

[a] HQ is the hazard quotient for a child aged 1-2.
[b] % Ch Is the percent change in the risk or hazard quotient relative to the *de minimis* calculations.
[c] Percent change in lifetime risk.
[d] Percent change in the hazard quotient for a child age 1 to 2.

C-4.4.4 Sensitivity When Accounting for Temporally Correlated Body Weight and Ingestion Rates

In calculating lifetime cancer risks, age-specific ingestion rates and body weights are used to estimate the lifetime average daily dose. In the systematic sensitivity analysis, the body weights and ingestion rates for each of the five age categories were changed independently to assess the sensitivity of lifetime risk to each input separately (for example, body weight was first perturbed only for a child 1-2 yrs old, leaving body weight unchanged for other age groups). However, in actuality, ingestion rates (and body weights) for an individual during different age groups will likely be correlated across that individual's lifetime. For example, a person who eats higher-than-average amounts of poultry when they are 11 can be reasonably expected to eat higher-than-average amounts when they are 50 due to lifetime dietary preferences. To estimate the effect this correlation has on sensitivity of cancer risk to ingestion rate, a perturbation of -5% for a given food type was applied to ingestion rate for all age categories and the resulting elasticity was compared to the previous result (obtained ignoring temporal/age-group correlations). This assumption was applied separately to each ingestion rate category, and separately to body weight, to estimate the effect of age group correlations for these inputs (Exhibit 4-31). Because only benzo[a]pyrene and 2,3,7,8-TCDD were evaluated for cancer risks, this analysis was only performed for these two PB-HAPs (the other PB-HAPs use HQs calculated separately for each age group, and the correlation analysis is not applicable).

Exhibit 4-31. Comparison in the Elasticities In Lifetime Risk in the Correlated and Uncorrelated Analyses Assuming a 5% Decrease in the Input Variables

		Elasticity in the Correlated Analysis	Maximum Elasticity in Uncorrelated Analysis	Age Group of Maximum Elasticity in Uncorrelated Analysis
Benzo[a]pyrene	Dairy Ingestion	0.34	0.11	Child 1-2
	Exp. Fruit Ingestion	0.22	0.10	Adult
	Body Weight	-0.17	-0.06	Child 1-2
	Exp. Veg. Ingestion	0.17	0.09	Adult
	Soil Ingestion	0.12	0.05	Child 1-2
	Beef Ingestion	0.07	0.03	Adult
	Fish Ingestion	0.04	0.02	Adult
	Poultry Ingestion	0.02	0.01	Adult
	Pro. Fruit Ingestion	0.01	< 0.01	Adult
	Egg Ingestion	0.01	< 0.01	Adult
	Pork Ingestion	< 0.01	< 0.01	Adult
	Prot. Veg. Ingestion	< 0.01	< 0.01	Adult
	Root Veg. Ingestion	< 0.01	< 0.01	Adult
2,3,7,8 - TCDD	Body Weight	-0.59	-0.45	Adult
	Fish Ingestion	0.56	0.43	Adult
	Dairy Ingestion	0.32	0.20	Child 1-2
	Beef Ingestion	0.09	0.06	Adult
	Pork Ingestion	0.01	0.01	Adult
	Soil Ingestion	< 0.01	< 0.01	Adult
	Exp. Fruit Ingestion	< 0.01	< 0.01	Adult
	Exp. Veg. Ingestion	< 0.01	< 0.01	Adult
	Root Veg. Ingestion	< 0.01	< 0.01	Adult
	Poultry Ingestion	< 0.01	< 0.01	Adult
	Egg Ingestion	< 0.01	< 0.01	Adult
	Pro. Fruit Ingestion	< 0.01	< 0.01	Adult
	Prot. Veg. Ingestion	< 0.01	< 0.01	Adult

The systematic sensitivity analysis suggested that cancer risks associated with benzo[a]pyrene are relatively insensitive to body weight and any individual food ingestion rate (where both inputs were varied for a single age group at a time). When body weight or ingestion rate for a given food type is decreased by 5% for all age groups in concert, the corresponding elasticity increases by a factor of two to three. This increase is significant enough to cause the dairy ingestion, exposed fruit ingestion, body weight, and exposed vegetable ingestion variables to be ranked among the most influential variables on the basis of absolute elasticity.

For 2,3,7,8-TCDD, the systematic sensitivity analysis illustrated that cancer risk is sensitive to body weight, fish ingestion rate, and dairy ingestion rate. Elasticities for these inputs increase by a factor of 1.3 to 1.6 when the inputs are kept constant across age groups. This difference results in rise in the systematic sensitivity ranking of these variables.

Overall, however, these differences represent a modest percent change in the lifetime risk values and indicate that accounting for the assumed correlation between the body weights and ingestion rates throughout an individual's lifetime does not significantly influence the model's predicted risks. For the other ingestion rates, the lifetime risks are not very sensitive to the parameters when the inputs are decreased by 5%, and accounting for the correlations between the variables does not affect this conclusion.

C-4.4.5 Comparison of Scenarios Using Site-Specific and de minimis Meteorological Data

For RTR, the *de minimis* thresholds were calculated using a conservative and hypothetical exposure scenario for a farm homestead. If the modeling scenario has been configured as intended, the substitution of site-specific data will nearly always result in a lower (or equal) exposure and risk estimate. To inform the degree of conservatism associated with the baseline meteorological inputs, three additional analyses were conducted in which observed (site-specific) meteorology data were used in place of the meteorological values set for the *de minimis* scenario. The three locations were selected to take advantage of readily-available TRIM.FaTE meteorological data. Exhibit 4-32 compares some of the summary statistics for the three meteorological data sets with those used for the *de minimis* screening scenario.[20]

Exhibit 4-32. Summary of Site-specific Meteorological Data Parameters

Parameter (units [a])	*de minimis* Scenario [b]	Site 1 [c]	Site 2 [c]	Site 3 [c]
Average air temperature (K)	298	291.9	284.7	283.5
Average horizontal wind speed (m/s)	2.8	4.4	4	3
Annual precipitation (m/yr)	1.5	1.1	1.1	1.1
Average urban mixing height (m)	710	1,087	1,225	861

[a] K = Kelvin, m/s = meters/second, m/yr = meters per year, m = meters

[b] Values listed for the baseline scenario indicate the fixed value used in the baseline screening scenario.

[c] Site-specific values are arithmetic averages of single or multiyear data sets; Site 1 = 1989–1993; Site 2 = 1994; Site 3 = 1990–1995.

The results of using these three site-specific meteorological data sets on the risk or hazard quotient for each PB-HAP are summarized in Exhibit 4-33. The results indicate that the wind direction – and specifically how often the wind blows from the source toward the hypothetical watershed, or toward due east – is an important influence on the estimated media concentrations and ingestion exposures. For the three data sets used in this analysis, the largest decrease in risk or hazard quotient was observed for Site 1. This finding is consistent with the underlying patterns in wind direction and their relationship to the locations where exposure is assumed to occur. Exhibit 4-34 shows that wind direction towards due east occurs less than two percent of the time for this location, reinforcing the conclusion that a limited wind flow directly from the source to the watershed will decrease contamination. These results suggest that using meteorological data more representative of a specific site will decrease the estimated risk or hazard quotient by as much as an order of magnitude for the hypothetical receptor represented.

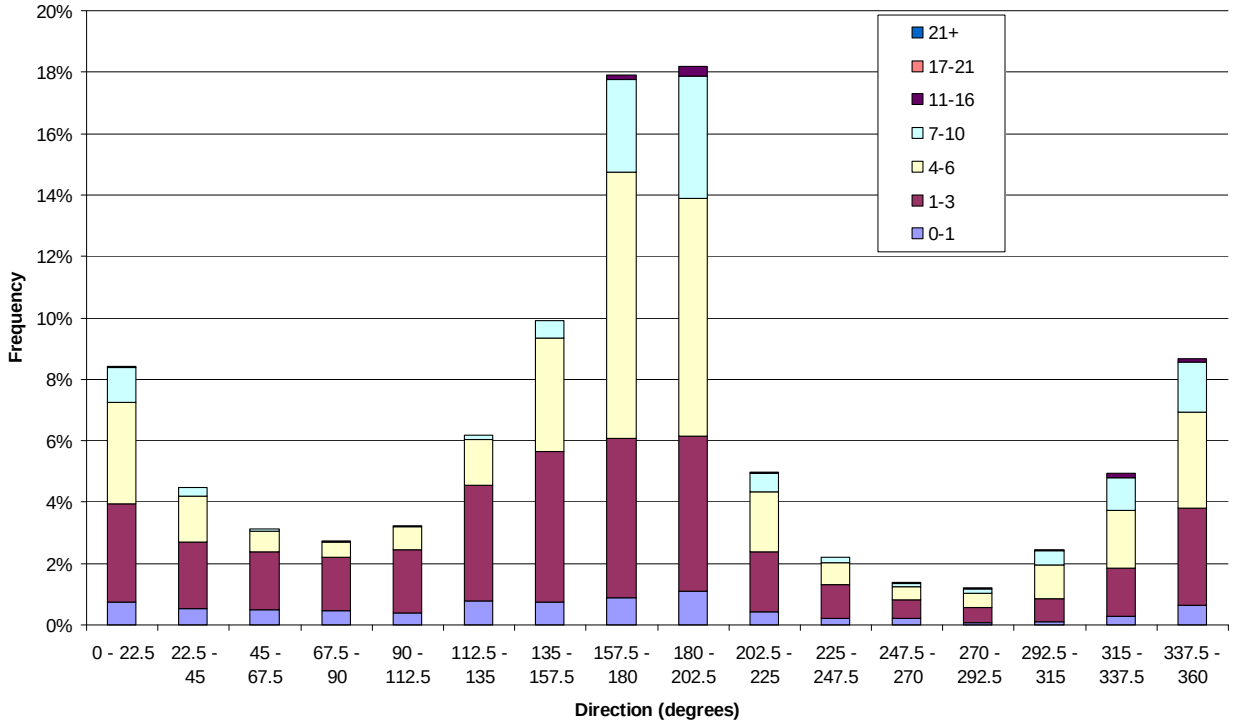
[20] Exhibit 4-33 does not include a measure of the frequency and average of wind directions, which can be illustrated with a wind rose but cannot be effectively characterized with a single value.

Exhibit 4-33. Percent Change in Risk or Hazard Quotient Using Site-specific Meteorological Data

	de minimis	Site 1		Site 2		Site 3	
		Risk or HQ [a]	% Ch [b]	Risk or HQ [a]	% Ch [b]	Risk or HQ [a]	% Ch [b]
Benzo[a]pyrene [c]	1.00E-06	1.05E-07	-89%	2.43E-07	-76%	2.19E-07	-78%
2,3,7,8-TCDD [c]	1.00E-06	8.39E-08	-92%	3.76E-07	-62%	2.56E-07	-74%
Cadmium [d]	1.00	0.07	-93%	0.14	-86%	0.15	-85%
Divalent Mercury [d]	1.00	0.08	-92%	0.19	-81%	0.18	-82%
Methyl Mercury [d]	0.56	0.02	-97%	0.06	-90%	0.05	-91%

[a] HQ is the hazard quotient for a child aged 1-2.
[b] % Ch Is the percent change in the risk or hazard quotient relative to the *de minimis* calculations.
[c] Percent change in lifetime risk.
[d] Percent change in the hazard quotient for a child age 1 to 2.

Exhibit 4-34. The Wind Speed and the Direction Toward Which the Wind is Blowing for All Conditions for Site 1



C-4.5 Comparison to Other Model Results

C-4.5.1 Comparison to Preliminary RTR Screening Runs (HHRAP Approach)

As another component of this evaluation, *de minimis* emission rates calculated using the screening scenario were compared to those calculated for the preliminary RTR screening analyses carried out by EPA in the fall of 2006 (EPA 2006a) that use fate and transport algorithms presented in the HHRAP documentation (EPA 2005a). Similar to the current TRIM-based scenario, the modeling scenario evaluated in the preliminary RTR analyses was based on a hypothetical working homestead. The homestead was adjacent to the modeled source, with a 16-acre lake and 100 acres of tillable farm and pasture land. The lake was placed at the location of highest total deposition and the total watershed area was just over 200 acres.

Exhibit 4-35 summarizes a comparison of the emission thresholds for each approach. Thresholds from the current analysis are presented to two significant figures for the purposes of comparison only. The emission threshold for benzo[a]pyrene is lower for the current scenario than for the previous evaluation (i.e., the current scenario is more conservative), while those of cadmium and divalent mercury are higher for the current runs. Because elemental mercury does not readily deposit and is largely blown out of the modeling domain into the air sinks in TRIM.FaTE, the threshold for divalent mercury is more relevant to this analysis. No threshold was available for dioxins for the preliminary HHRAP analysis. Additional refinements, analyses and comparisons described in this section support the current *de minimis* emission thresholds.

Exhibit 4-35. Emission Thresholds Derived in Preliminary HHRAP Screening Runs and in Current Analyses

Chemical	Basis of Threshold	Emission Thresholds (TPY)		Comparison
		Current Analysis	Preliminary RTR Analysis	
2,3,7,8-TCDD	Cancer	3.18E-08	NA	NA
Benzo[a]pyrene	Cancer	2.26E-03	2.2E-02	Preliminary threshold higher by ~10x
Cadmium	Non-cancer	6.54E-01	1.7E-01	Current threshold higher by ~4x
Divalent Mercury	Non-cancer	1.64E-01	5.4E-03	Current threshold higher by ~30x

NA = not available; threshold was not calculated for dioxins in 2006.

C-4.5.2 Comparison of Results for Screening Scenario and Previous TRIM.FaTE Applications

To obtain another estimate of the degree of conservatism associated with results from the screening scenario, a comparison run was performed using the TRIM.FaTE scenario developed for a secondary lead smelting facility previously evaluated for a TRIM to IEM[21] model comparison in the state of New York (ICF 2004). Results for the current comparison were obtained for benzo[a]pyrene; 2,3,7,8-tetrachlorodibenzo-p-dioxin; and elemental, divalent, and methyl mercury by running the screening scenario with the emission rates for these chemicals that were used in the previous secondary lead smelting application. The annually averaged results for the 30th year of the screening scenario were compared to annual average concentrations using results from years 28 through 32 from the secondary lead smelting

[21] IEM is the Indirect Exposure Methodology that is now referred to as the Multiple Pathways of Exposure Methodology (U.S. EPA 1999d)

application.[22] No adjustments to the screening or the New York (refined) scenario were implemented; thus, it should be recognized that the results of this comparison are particular to the inputs selected for the New York site TRIM.FaTE application.

Specifications of the New York site application are presented in the model comparison report and are not included here. In general, no comparisons of the values used for the screening and New York site TRIM.FaTE scenarios were conducted. However, because meteorological properties are highly influential on concentrations in all compartments, the characteristics of the New York meteorological inputs are summarized here. Five years of meteorological data collected at a station in Allentown, Pennsylvania were used for the New York application; average values for key properties are summarized in Exhibit 4-36.

Exhibit 4-36. Meteorological Data Parameters for TRIM.FaTE Secondary Lead Smelting Application

Meteorological Property	Average Value Used for New York Site Application
Air temperature	284.69 K
Horizontal wind speed	3.97 m/s
Precipitation rate	1.14 m/yr
Urban mixing height	1,224 m
Wind direction (overall)	Blows predominantly from the southeast
Wind direction (during rain events)	Blows predominantly from the southwest

Media concentrations for air, surface soil, lake surface water, lake benthic sediment, and water column fish from the two applications were compared collectively (i.e., using the mean of all compartments of a single type) and, for soil and air, according to approximate distance from the source. For the latter comparisons, air and soil parcels were grouped into “nearby” and “distant” subgroups. Nearby parcels were those situated within about 2 km of the source, and the remaining parcels are distant. Parcel groupings for air and surface soil output comparisons are presented in the figures that follow. Results for a lake near the source in the New York application were used for comparison to results for water, sediment, and fish in the screening scenario.

[22] Averaging results over 5 years of data minimizes bias introduced by any 1 year of meteorological conditions (5 years of repeating data were used for the New York site application).

Exhibit 4-37. Surface Soil Parcel Spatial Layouts for New York Site Lead Smelting TRIM.FaTE Application and Screening Scenario

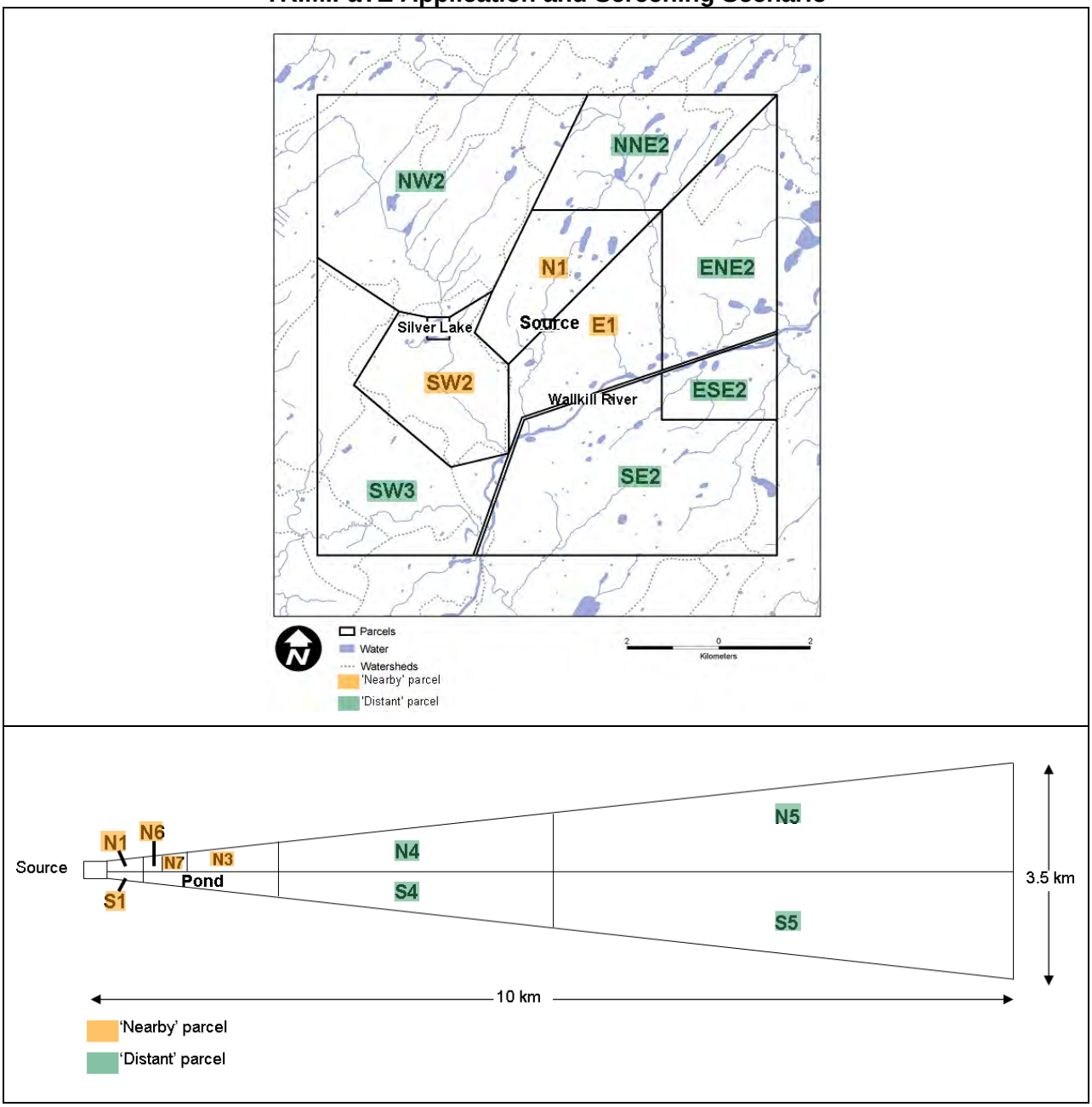
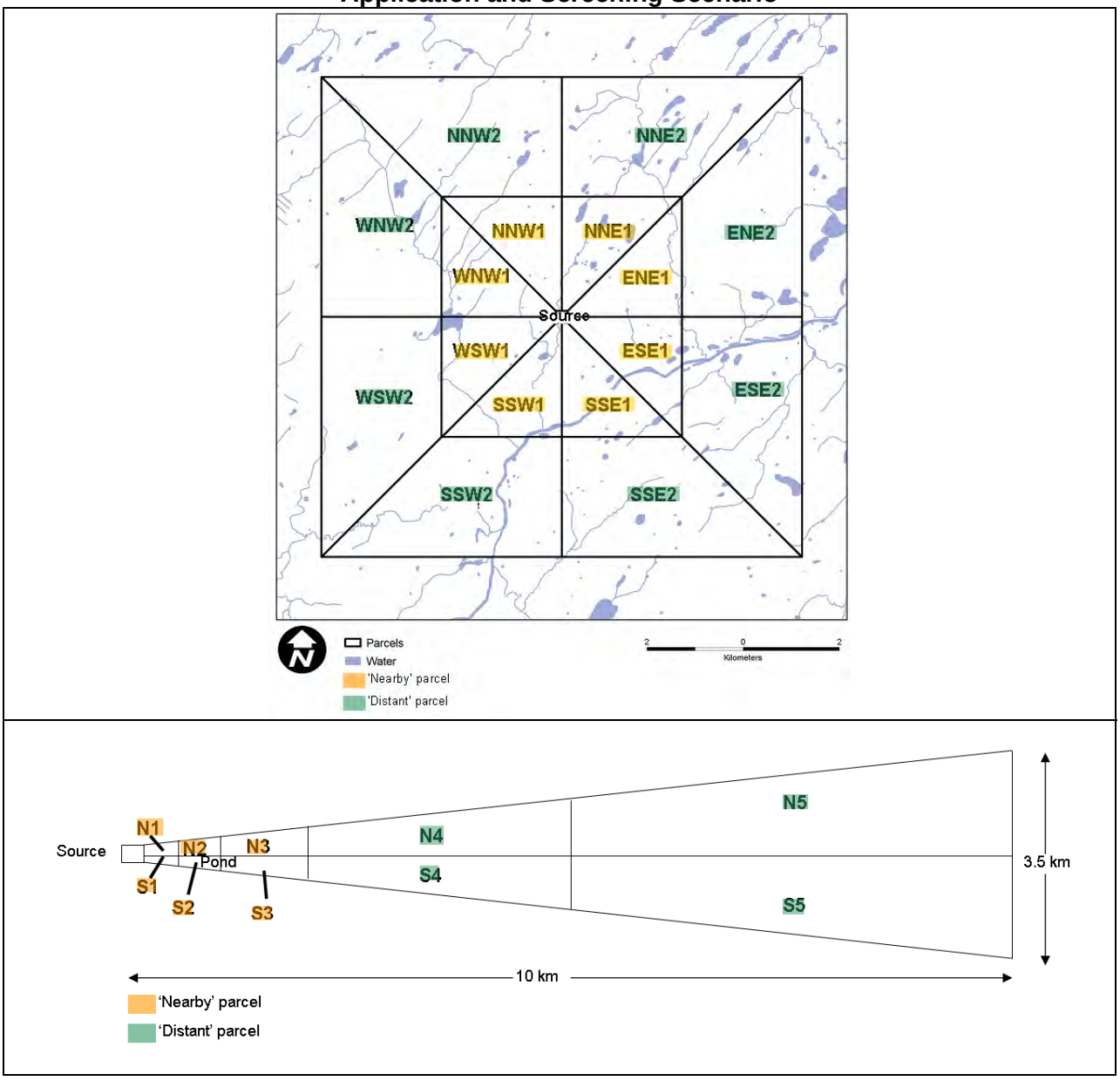


Exhibit 4-38. Air Parcel Spatial Layouts for New York Site Lead Smelting TRIM.FaTE Application and Screening Scenario



The screening and refined results are compared in Exhibit 4-39. This table shows the ratios of the concentrations for the screening scenario to the concentrations from the New York site application; thus, ratios greater than 1 indicate that screening scenario concentrations are higher than the lead smelting application concentrations. Exhibit 4-40 graphically illustrates selected concentration ratios.

Exhibit 4-39. Comparison of Concentration Outputs: NY Site Refined TRIM.FaTE Application vs. Screening Scenario

Output [a]	Concentration Ratio of Screening Scenario to Full Lead Smelting Run				
	Benzo[a] Pyrene	2,3,7,8-TCDD	Divalent Mercury	Elemental Mercury [b]	Methyl Mercury
Overall Air Mean	15.1	12.8	13.0	12.6	16.4
Nearby Air Mean	15.8	13.8	13.8	13.7	17.0
Distant Air Mean	6.6	6.2	5.7	6.1	8.2
Overall Surface Soil Mean	19.4	3.9	10.4	-	10.3
Nearby Surface Soil Mean	18.9	3.6	9.1	-	9.0
Distant Surface Soil Mean	8.6	2.7	7.6	-	7.6
Lake Surface Water	17.0	6.0	12.1	28.7	5.7
Lake Sediment	52.2	10.3	6.6	12.2	7.0
Lake WC Carnivore	19.4	12.6	2.4	-	1.9
Lake WC Herbivore	10.5	0.8	2.1	-	1.4
Lake WC Omnivore	18.0	2.1	2.3	-	1.6

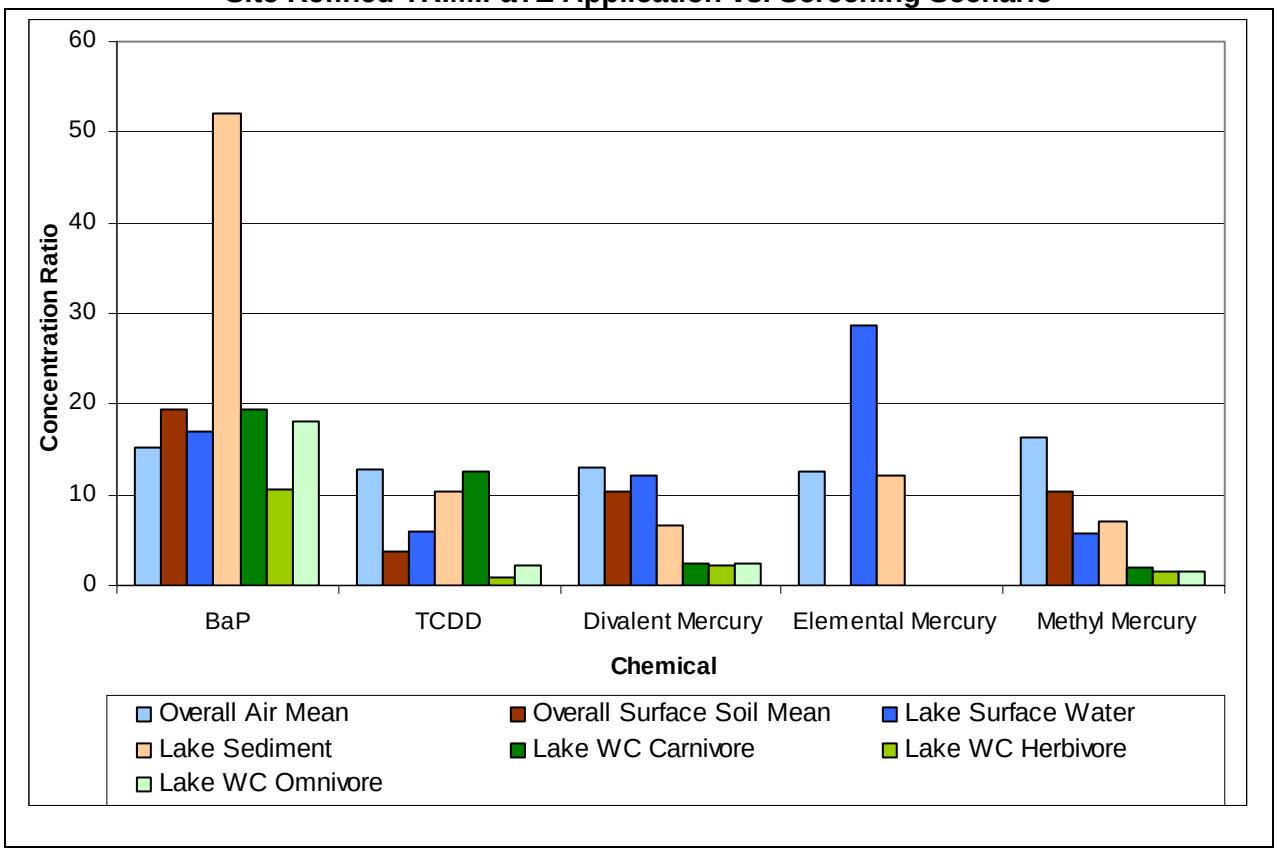
[a] "Overall Air" and "Overall Surface Soil" include all air and surface soil parcels, except for the source parcel. "Nearby" parcels include those within about 2 km of the source; "Distant" parcels include those greater than about 2 – 3 km from the source (i.e., all non-source parcels not classified as Nearby). Refer to Exhibit 7-14 and Exhibit 7-15 for details. The source parcel was excluded from all comparisons. "Lake" is the lake for the New York site application, and the pond for the screening scenario.

[b] Because elemental mercury is largely blown out of the modeling domain into the air sinks in TRIM.FaTE, concentrations of elemental mercury are not examined here in soil or in water column fish.

In all media categories, the screening scenario produces greater concentrations than does the lead smelting application. This result is expected because the screening scenario is designed to be health-protective and therefore tends more towards a high-end scenario with regard to media concentrations. The differences are generally larger for air results and smaller for concentrations in fish.

On the whole, the greatest difference between the concentrations in the screening scenario and those in the lead smelting application occurs with benzo[a]pyrene, especially with regard to concentrations in fish and lake sediment. The largest difference observed across all comparisons is the benzo[a]pyrene in lake sediment. Outputs were also substantially different for elemental mercury in the surface water and benzo[a]pyrene in water column fish.

Exhibit 4-40. Comparison of Concentration Outputs Grouped By Chemical: New York Site Refined TRIM.FaTE Application vs. Screening Scenario



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ATTACHMENT C-1: TRIM.FaTE Inputs

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TABLE OF CONTENTS

1 TRIM.FaTE Inputs

1.1 Introduction

This attachment provides the tables of the detailed modeling inputs for the TRIM.FaTE screening scenario. Exhibit 1 presents runtime settings for TRIM.FaTE. Exhibits 2 and 3 present air parameters entered into the model. Exhibits 3 through 8 present the terrestrial parameters. Exhibits 9 through 12 present the lake parameters, and 13 through 28 present the chemical specific parameters.

Exhibit 1. TRIM.FaTE Simulation Parameters for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value Used	Reference
Start of simulation	date/time	1/1/1990, midnight	Consistent with met data.
End of simulation	date/time	1/1/2040, midnight	Consistent with met data set; selected to provide a 50-year modeling period.
Simulation time step	hr	1	Selected value.
Output time step[a]	hr	4	Selected value.

[a] Output time step is set in TRIM.FaTE using the scenario properties "simulationStepsPerOutputStep" and "simulationTimeStep."

Exhibit 2. Meteorological Inputs for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value Used	Reference
Meteorological Inputs (all TRIM.FaTE scenario properties, except mixing height)			
Air temperature	degrees K	298	USEPA 2005.
Horizontal wind speed	m/sec	2.8	5th percentile annual average value for contiguous US, calculated from 30 yrs of annual normal temperature values.
Vertical wind speed	m/sec	0.0	Professional judgment; vertical wind speed not used by any of the algorithms in the version of the TRIM.FaTE library used for screening.
Wind direction	degrees clockwise from N (blowing from)	3-days-on 4-days-off	On is defined as time during which wind is blowing into the model domain. A conservative estimate of time during which wind should blow into the modeling domain was determined by evaluating HUSWO; it was concluded that a conservative estimate would be approximately 42% of the time.
Rainfall Rate	m^3[rain]/m^2[surface area]-day	varies daily	1.5 m/yr is the maximum statewide 30-year (1971-2000) average for the contiguous United States, excluding Rhode Island because of extreme weather conditions on Mt. Washington. Data obtained from the National Climatic Data Center at http://www.ncdc.noaa.gov/oa/climate/online/ccd/nrmpcp.txt. The precipitation frequency was 3-days-on:4-days-off based on data from Holzworth, 1972.
Mixing height (used to set air VE property named “top”)	m	710	5th percentile annual average mixing heights (calculated from daily morning and afternoon values), for all stations on SCRM (40 state, 70 stations).
isDay_SteadyState_forAir	unitless	--	Value not used in current dynamic runs (would need to be reevaluated if steady-state runs are needed).
isDay_SteadyState_forOther	unitless	--	

Exhibit 3. Air Parameters for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value Used	Reference
Atmospheric dust load	kg[dust]/m^3[air]	6.15E-08	Bidleman 1988
Density of air	g/cm^3	0.0012	U.S. EPA 1997
Dust density	kg[dust]/m^3[dust]	1,400	Bidleman 1988
Fraction organic matter on particulates	unitless	0.2	Harner and Bidleman 1998
Height	m	800	5th percentile for United States

Exhibit 4. Soil and Groundwater Parameters for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value Used	Reference
Surface Soil Compartment Type			
Air content	volume[air]/volume[compartment]	0.28	McKone et al. 2001.
Average vertical velocity of water (percolation)	m/day	8.22E-04	Assumed to be 0.2 times average precipitation for site.
Boundary layer thickness above surface soil	m	0.005	Thibodeaux 1996; McKone et al. 2001 (Table 3).
Density of soil solids (dry weight)	kg[soil]/m^3[soil]	2600	Default in McKone et al. 2001 (Table 3)
Thickness - untilled[a]	m	0.01	McKone et al. 2001 (p. 30).
Thickness - tilled[a]	m	0.20	USEPA 2005.
Erosion fraction	unitless	varies[b]	See Erosion and Runoff Fraction table.
Fraction of area available for erosion	m^2[area available]/m^2[total]	1	Professional judgment; area assumed rural.
Fraction of area available for runoff	m^2[area available]/m^2[total]	1	Professional judgment; area assumed rural.
Fraction of area availabe for vertical diffusion	m^2[area available]/m^2[total]	1	Professional judgment; area assumed rural.
Fraction Sand	unitless	0.25	Professional judgment.
Organic carbon fraction	unitless	0.008	U.S. average in McKone et al. 2001 (Table 16 and A-3).
pH	unitless	6.8	Professional judgment.
Runoff fraction	unitless	varies[b]	See Erosion and Runoff Fraction table.
Total erosion rate	kg [soil]/m^2/day	varies[b]	See Total Erosion Rates table.
Total runoff rate	m^3[water]/m^2/day	1.64E-03	Calculated using scenario-specific precipitation rate and assumptions associated with water balance.
Water content	volume[water]/volume[compartment]	0.19	McKone et al 2001 (Table 15).

Exhibit 4. Soil and Groundwater Parameters for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value Used	Reference
Root Zone Soil Compartment Type			
Air content	volume[air]/volume[compartment]	0.25	McKone et al 2001 (Table 16).
Average vertical velocity of water (percolation)	m/day	8.22E-04	Assumed as 0.2 times average precipitation for New England in McKone et al. 2001.
Density of soil solids (dry weight)	kg[soil]/m^3[soil]	2,600	McKone et al. 2001 (Table 3).
Fraction Sand	unitless	0.25	Professional judgment.
Thickness - untilled[a]	m	0.79	McKone et al. 2001 (Tabel 16 - U.S. Average).
Thickness - tilled[a]	m	0.6	Adjusted from McKone et al. 2001 (Table 16).
Organic carbon fraction	unitless	0.008	McKone et al. 2001 (Table 16 and A-3, U.S. Average).
pH	unitless	6.8	Professional judgment.
Water content	volume[water]/volume[compartment]	0.21	McKone et al. 2001 (Table 16).
Vadose Zone Soil Compartment Type			
Air content	volume[air]/volume[compartment]	0.22	McKone et al. 2001 (Table 17).
Average vertical velocity of water (percolation)	m/day	8.22E-04	Assumed as 0.2 times average precipitation for New England in McKone et al. 2001.
Density of soil solids (dry weight)	kg[soil]/m^3[soil]	2,600	Default in McKone et al. 2001 (Table 3).
Fraction Sand	unitless	0.35	Pofessional judgment.
Thickness[a]	m	1.4	McKone et al. 2001 (Table 17).
Organic carbon fraction	unitless	0.003	McKone et al. 2001 (Table 16 and A-3, U.S. Average).
pH	unitless	6.8	Professional judgment.
Water content	volume[water]/volume[compartment]	0.21	McKone et al. 2001 (Table 17 - national average).

Exhibit 4. Soil and Groundwater Parameters for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value Used	Reference
Ground Water Compartment Type			
Thickness[a]	m	3	McKone et al. 2001 (Table 3).
Fraction Sand	unitless	0.4	Professional judgment.
Organic carbon fraction	unitless	0.004	Professional judgment.
pH	unitless	6.8	Professional judgment.
Porosity	volume[total pore space]/volume[compartment]	0.2	Default in McKone et al. 2001 (Table 3).
Density of Solid material in aquifer	kg[soil]/m^3[soil]	2,600	Default in McKone et al. 2001 (Table 3).

[a]Set using the volume element properties file

[b]See separate tables for erosion/runoff fractions and total erosion rates.

Exhibit 5. Runoff Assumptions for the TRIM.FaTE Screening Scenario

Originating Compartment	Destination Compartment	Runoff/Erosion Fraction
SurfSoil_Source	SurfSoil_N1	0.0
	SurfSoil_S1	0.0
	sink	1.0
SurfSoil_N1	SW_Pond	1.0
	SurfSoil_Source	0.0
	SurfSoil_N6	0.0
	SurfSoil_S1	0.0
	sink	0.0
SurfSoil_S1	SW_Pond	1.0
	SurfSoil_Source	0.0
	SurfSoil_N1	0.0
	sink	0.0
SurfSoil_N6	SW_Pond	1.0
	SurfSoil_N1	0.0
	SurfSoil_N7	0.0
	sink	0.0
SurfSoil_N7	SW_Pond	1.0
	SurfSoil_N6	0.0
	SurfSoil_N3	0.0
	sink	0.0
SurfSoil_N3	SW_Pond	1.0
	SurfSoil_N7	0.0
	SurfSoil_N4	0.0
	sink	0.0
SurfSoil_N4	SW_Pond	1.0
	SurfSoil_N3	0.0
	SurfSoil_N5	0.0
	SurfSoil_S4	0.0
	sink	0.0
SurfSoil_S4	SW_Pond	1.0
	SurfSoil_N4	0.0
	SurfSoil_S5	0.0
	sink	0.0
SurfSoil_N5[a]	SW_Pond	0.0
	SurfSoil_N4	0.5
	SurfSoil_S5	0.5
	sink	0.0
SurfSoil_S5[a]	SW_Pond	0.0
	SurfSoil_N5	0.0
	SurfSoil_S4	1.0
	sink	0.0

[a] Assumes that N5 is higher ground that S5, and half of the runoff flows into N4, and the other half in S5. Assumes all runoff from S5 flows into S4.

Exhibit 6. USLE Erosion Parameters for the TRIM.FaTE Screening Scenario

Soil Parcel	Area	Rainfall/ Erosivity Index	Soil Erodibility Index	Length-Slope Factor	Land Use	Cover Mgmt Factor	Supporting Practices Factor	Unit Soil Loss		Sediment Delivery Ratio[a]	Calculated (Adjusted) Erosion Rate
	m^2	R (100 ft-ton/ac)	K (ton/ac/(100 ft-ton/acre))	LS (USCS)	type	C (USCS)	P	A (ton/ac/yr)	A ($kg/m^2/d$)	SDR[a]	calculated (adjusted) erosion rate ($kg/m^2/d$)
N1	5.8E+04	300	0.39	1.5	grass	0.1	1	17.55	0.010779	0.533	0.005740
N6	4.1E+04	300	0.39	1.5	crops	0.2	1	35.1	0.021557	0.557	0.012014
N7	7.3E+04	300	0.39	1.5	grass	0.1	1	17.55	0.010779	0.518	0.005580
N3	3.5E+05	300	0.39	1.5	grass	0.1	1	17.55	0.010779	0.385	0.004151
N4	2.0E+06	300	0.39	1.5	forest	0.1	1	17.55	0.010779	0.309	0.003331
N5	6.7E+06	300	0.39	1.5	forest	0.1	1	17.55	0.010779	0.196	0.002116
S1	5.8E+04	300	0.39	1.5	grass	0.1	1	17.55	0.010779	0.533	0.005740
S4	2.0E+06	300	0.39	1.5	forest	0.1	1	17.55	0.010779	0.309	0.003331
S5	6.7E+06	300	0.39	1.5	forest	0.1	1	17.55	0.010779	0.196	0.002116

[a]Calculated using $SD = a * (AL)^{-b}$; where a is the empirical intercept coefficient (based on the size of the watershed), AL is the total watershed area receiving deposition (m^2), and b is the empirical slope coefficient (always 0.125).

Exhibit 7. Terrestial Plant Placement for the TRIM.FaTE Screening Scenario

Surface Soil Volume Element	Surface Soil Depth (m)	Coniferous Forest	Grasses/ Herbs	None
Source	0.01			x
N1	0.01		x	
N6	0.20 (tilled)			
N7	0.01		x	
N3	0.01		x	
N4	0.01	x		
N5	0.01	x		
S1	0.01		x	
S4	0.01	x		
S5	0.01	x		

Exhibit 8. Terrestrial Plant Parameters for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Coniferous[a]		Grass/Herb[a]	
		Value Used	Reference	Value Used	Reference
Leaf Compartment Type					
Allow exchange	1=yes, 0=no	1	-	seasonal[b]	-
Average leaf area index	m^2[leaf]/ m^2[area]	5.0	Harvard Forest, dom. red oak and red maple, CDIAC website	5.0	Mid-range of 4-6 for old fields, R.J. Luxmoore, ORNL.
Calculate wet dep interception fraction (boolean)	1=yes, 0=no	0	Professional judgment.	0	Professional judgment.
Correction exponent, octanal to lipid	unitless	0.76	From roots, Trapp 1995.	0.76	From roots, Trapp 1995.
Degree stomatal opening	unitless	1	Set to 1 for daytime based on professional judgment (stomatal diffusion is turned off at night using a different property, IsDay).	1	Set to 1 for daytime based on professional judgment (stomatal diffusion is turned off at night using a different property, IsDay).
Density of wet leaf	kg/m^3	820	Paterson et al. 1991.	820	Paterson et al. 1991.
Leaf wetting factor	m	3.00E-04	1E-04 to 6E-04 for different crops and elements, Muller and Prohl 1993.	3.00E-04	1E-04 to 6E-04 for different crops and elements, Muller and Prohl 1993.
Length of leaf	m	0.01	Professional judgment.	0.05	Professional judgment.
Lipid content	kg/kg wet weight	0.00224	European beech, Riederer 1995.	0.00224	European beech, Riederer 1995.
Litter fall rate	1/day	0.0021	value assumes 1st-order relationship and that 99% of leaves fall over 6 years	seasonal[c]	-
Stomatal area normalized effective diffusion path length	1/m	200	Wilmer and Fricker 1996.	200	Wilmer and Fricker 1996.
Vegetation attenuation factor	m^2/kg	2.9	Grass/hay, Baes et al. 1984.	2.9	Grass/hay, Baes et al. 1984.
Water content	unitless	0.8	Paterson et al. 1991.	0.8	Paterson et al. 1991.
Wet dep interception fraction	unitless	0.2	Calculated based on 5 years of local met data, 1987-1991.	0.2	Calculated based on 5 years of local met data, 1987-1991.

Exhibit 8. Terrestrial Plant Parameters for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Coniferous[a]		Grass/Herb[a]	
		Value Used	Reference	Value Used	Reference
Wet mass of leaf per soil area	kg[fresh leaf]/m^2[area]	2.0	Calculated from leaf area index, leaf thickness (Simonich & Hites, 1994), density of wet foliage.	0.6	Calculated from leaf area index and Leith 1975.
Particle on Leaf Compartment Type					
Allow exchange	1=yes, 0=no	1	-	seasonal[b]	-
Volume particle per area leaf	m^3[leaf particles]/m^2[leaf]	1.00E-09	Based on particle density and size distribution for atmospheric particles measured on an adhesive surface, Coe and Lindberg 1987.	1.00E-09	Based on particle density and size distribution for atmospheric particles measured on an adhesive surface, Coe and Lindberg 1987.
Root Compartment Type - Nonwoody Only					
Allow exchange	1=yes, 0=no			seasonal[b]	-
Correction exponent, octanol to lipid	unitless			0.76	Trapp 1995.
Lipid content of root	kg/kg wet weight			0.011	Calculated.
Water content of root	kg/kg wet weight			0.8	Professional judgment.
Wet density of root	kg/m^3			820	Soybean, Paterson et al. 1991.
Wet mass per soil area	kg/m^2			1.4	Temperate grassland, Jackson et al. 1996.
Stem Compartment Type - Nonwoody Only					
Allow exchange	1=yes, 0=no			seasonal[b]	-
Correction exponent, octanol to lipid	unitless			0.76	Trapp 1995
Density of phloem fluid	kg/m^3			1,000	Professional judgment.
Density of xylem fluid	kg/cm^3			900	Professional judgment.
Flow rate of transpired water per leaf area	m^3[water]/m^2[leaf]			0.0048	Crank et al. 1981.
Fraction of transpiration flow rate that is phloem rate	unitless			0.05	Paterson et al. 1991.
Lipid content of stem	kg/kg wet weight			0.00224	Leaves of European beech, Riederer 1995.

Exhibit 8. Terrestrial Plant Parameters for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Coniferous[a]		Grass/Herb[a]	
		Value Used	Reference	Value Used	Reference
Water content of stem	unitless			0.8	Paterson et al. 1991
Wet density of stem	kg/m^3			830	Professional judgment.
Wet mass per soil area	kg/m^2			0.24	Calculated from leaf and root biomass density based on professional judgment.

[a]See separate table for assignment of plant types to surface soil compartments.

[b]Begins March 9 (set to 1), ends November 7 (set to 0). Nation-wide 80th percentile.

[c]Begins November 7, ends December 6; rate = 0.15/day during this time (value assumes 99 percent of leaves fall in 30 days).

Exhibit 9. Surface Water Parameters for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value Used	Reference
Algae carbon content (fraction)	unitless	0.465	APHA 1995.
Algae density in water column	g[algae]/L[water]	0.0025	Millard et al. 1996 as cited in ICF 2005.
Algae growth rate	1/day	0.7	Hudson et al. 1994 as cited in Mason et al. 1995b
Algae radius	um	2.5	Mason et al. 1995b.
Algae water content (fraction)	unitless	0.9	APHA 1995.
Average algae cell density (per vol cell, not water)	g[algae]/m^3[algae]	1,000,000	Mason et al. 1995b, Mason et al. 1996.
Boundary layer thickness above sediment	m	0.02	Cal EPA 1993.
Chloride concentration	mg/L	8.0	Kaushal et al. 2005.
Chlorophyll concentration	mg/L	0.0029	ICF 2005.
Depth[a]	m	3.18	WI DNR 2005 - calculation based on relationship between drainage basin and lake area size.
Dimensionless viscous sublayer thickness	unitless	4	Ambrose et al. 1995.
Drag coefficient for water body	unitless	0.0011	Ambrose et al. 1995.
Flush rate	1/year	12.17	Calculated based on pond dimensions and flow calculations.
Fraction Sand	unitless	0.25	Professional judgment.
Organic carbon fraction in suspended sediments	unitless	0.02	Professional judgment.
pH	unitless	7.3	Professional judgment.
Suspended sediment deposition velocity	m/day	2	USEPA 1997.
Total suspended sediment concentration	kg[sediment]/m3[water column]	0.05	USEPA 2005.
Water temperature	degrees K	298	USEPA 2005.

[a]Set using the volume element properties named "top" and "bottom."

Exhibit 10. Sediment Parameters for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value Used	Reference
Depth[a]	m	0.05	McKone et al. 2001 (Table 3).
Fraction Sand	unitless	0.25	Professional judgment.
Organic carbon fraction	unitless	0.02	McKone et al. 2001 (Table 3).
Porosity of the sediment zone	volume[total pore space]/volume[sediment compartment]	0.6	USEPA 1998.
Solid material density in sediment	kg[sediment]/m^3[sediment]	2,600	McKone et al. 2001 (Table 3).

[a] Set using the volume element properties named "top" and "bottom."

Exhibit 11. Aquatic Plants
for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value Used	Reference
Macrophyte biomass per water area	kg/m^2	0.5	Professional judgment.
Density of macrophytes	kg/L	1	Professional judgment.

Exhibit 12. Aquatic Animals Food Chain, Density, and Mass for the TRIM.FaTE Screening Scenario

Aquatic Biota (Consuming Organism)	Fraction Diet								Biomass (kg/m^2)	Body Weight	Reference
	Algae	Macrophyte	Bethic Invertebrate	Water Column Herbivore	Benthic Omnivore	Water Column Omnivore	Benthic Carnivore	Water Column Carnivore			
Benthic Invertebrate	0%	0%	0%	0%	0%	0%	0%	0%	0.020	2.55E-04	Professional judgment.
Water Column Herbivore	100%	0%	0%	0%	0%	0%	0%	0%	0.001	0.025	Professional judgment.
Benthic Omnivore	0%	0%	100%	0%	0%	0%	0%	0%	0.002	2.50E-01	Professional judgment.
Water Column Omnivore	0%	30%	30%	40%	0%	0%	0%	0%	0.001	0.25	Professional judgment.
Benthic Carnivore	0%	0%	70%	0%	30%	0%	0%	0%	0.001	2.0	Professional judgment.
Water Column Carnivore	0%	0%	0%	20%	20%	60%	0%	0%	0.0004	2.0	Professional judgment.

Exhibit 13. Cadmium Chemical-Specific Properties Documentation for the TRIM.FaTE Screening Scenario

Parameter Name[a]	Units	Value	Reference
CAS number[b]	unitless	7440-43-9	-
Diffusion coefficient in pure air	m^2[air]/day	0.71	USEPA 1999 (Table A-2-35).
Diffusion coefficient in pure water	m^2[water]/day	8.16E-05	USEPA 1999 (Table A-2-35).
Henry's Law Constant	Pa-m^3/mol	1.00E-37	USEPA 1999 (Table A-2-35; assumed to be zero).
Melting point	degrees K	594	ATSDR 1999.
Molecular weight	g/mol	112.41	ATSDR 1999.
Octanol-air partition coefficient (Koa)	m3[air]/m3[octanol]	-	-
Octanol-carbon partition coefficient (Koc)		-	-
Octanol-water partition coefficient (Kow)	L[water]/kg[octanol]	-	-

[a]All parameters in this table are TRIM.FaTE chemical properties.

[b]These CAS numbers apply to elemental Cd; however, the cations of cadmium are being modeled.

Exhibit 14. Mercury Chemical-Specific Properties
Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value			Reference
		Hg(0)[b]	Hg(2)[b]	MHg[b]	
CAS number	unitless	7439-97-6	14302-87-5	22967-92-6	-
Diffusion coefficient in pure air	m^2[air]/day	0.478	0.478	0.456	USEPA 1997.
Diffusion coefficient in pure water	m^2[water]/day	5.54E-05	5.54E-05	5.28E-05	USEPA 1997.
Henry's Law constant	Pa-m^3/mol	719	7.19E-05	0.0477	USEPA 1997.
Melting Point	degrees K	234	5.50E+02	443	CARB 1994.
Molecular weight	g/mol	201	201	216	USEPA 1997.
Octanol-water partition coefficient (Kow)	L[water]/kg[octanol]	4.15	3.33	1.7	Mason et al. 1996.
Vapor washout ratio	m^3[air]/m^3[rain]	1,200	1.6E+06	0	USEPA 1997, based on Petersen et al. 1995.

[a]All parameters in this table are TRIM.FaTE chemical properties.
[b]On this and all following tables, Hg(0) = elemental mercury, Hg(2) = divalent mercury, and MHg = methyl mercury.

Exhibit 15. PAH Chemical-Specific Properties
Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value							Reference
		BaP	BaA	BbF	BkF	Chr	DahA	IcdP	
CAS number	unitless	50-32-8	56-55-3	205-99-2	207-08-9	218-01-9	53-70-3	193-39-5	-
Diffusion coefficient in pure air	m^2/day	0.188	0.213	0.197	0.197	0.214	0.156	0.164	USEPA 1998.
Diffusion coefficient in pure water	m^2/day	5.05E-05	5.37E-05	4.74E-05	4.74E-05	5.37E-05	5.19E-05	4.89E-05	USEPA 1998.
Henry's Law constant	Pa-m^3/mol	8.50E-02	3.67E-01	6.26E-01	4.20E-02	1.23E-01	1.13E-03	4.92E-04	USEPA 1998.
Melting point	degrees K	452	433	441	490	531	539	437	Budavari 1996.
Molecular weight	g/mol	252.32	228.29	252.32	252.32	228.29	278.33	276.34	Budavari 1996.
Octanol-water partition coefficient (Kow)	L[water]/L[octanol]	9.33E+05	6.17E+05	3.98E+06	6.92E+06	5.37E+05	3.16E+06	4.57E+07	Hansch et al. 1995.

Exhibit 16. Dioxin Chemical-Specific Properties
Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value						
		1,2,3,4,6,7,8, 9-OCDD	1,2,3,4,6,7,8, 9-OCDF	1,2,3,4,6,7,8- HpCDD	1,2,3,4,6,7,8- HpCDF	1,2,3,4,7,8,9- HpCDF	1,2,3,4,7,8- HxCDD	1,2,3,4,7,8- HxCDF
CAS number	unitless	3268-87-9	39001-02-0	35822-46-9	67562-39-4	55673-89-7	39227-28-6	70648-26-9
Diffusion coefficient in pure air	m^2/day	0.0883	0.123	0.0925	0.129	0.129	0.0958	0.135
Diffusion coefficient in pure water	m^2/day	3.08E-06	3.15E-05	3.24E-05	3.33E-05	3.33E-05	3.43E-05	3.53E-05
Henry's Law constant	Pa-m^3/mol	0.68	0.19	1.28	1.43	1.43	1.08	1.45
Melting Point	degrees K	603	259	538	236.5	222	546	499.0
Molecular weight	g/mol	460.0	443.76	425.2	409.31	409.31	391.0	374.87
Octanol-water partition coefficient (Kow)	L[water]/L[octanol]	1.58E+08	1.00E+08	1.00E+08	2.51E+07	7.94E+06	6.31E+07	1.00E+07

Exhibit 16. Dioxin Chemical-Specific Properties
Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value						
		1,2,3,6,7,8-HxCDD	1,2,3,6,7,8-HxCDF	1,2,3,7,8,9-HxCDD	1,2,3,7,8,9-HxCDF	1,2,3,7,8-PeCDD	1,2,3,7,8-PeCDF	2,3,4,6,7,8-HxCDF
CAS number	unitless	57653-85-7	57117-44-9	19408-74-3	72918-21-9	40321-76-4	57117-41-6	60851-34-5
Diffusion coefficient in pure air	m^2/day	0.0958	0.135	0.0958	0.135	0.101	0.142	0.135
Diffusion coefficient in pure water	m^2/day	3.43E-05	3.53E-05	3.43E-05	3.53E-05	3.65E-05	3.76E-05	3.53E-05
Henry's Law constant	Pa-m^3/mol	1.08	0.74	1.08	0.74	3.33	0.5	0.74
Melting point	degrees K	558.0	506.0	517.0	509.0	513.0	499.0	512.5
Molecular weight	g/mol	390.84	374.9	390.8	374.9	356.4	340.4	374.9
Octanol-water partition coefficient (Kow)	L[water]/L[octanol]	1.62E+08	8.24E+07	1.62E+08	3.80E+07	4.37E+06	6.17E+06	8.31E+07

Exhibit 16. Dioxin Chemical-Specific Properties
Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value			Reference
		2,3,4,7,8-PeCDF	2,3,7,8-TCDD	2,3,7,8-TCDF	
CAS number	unitless	57117-31-4	1746-01-6	51207-31-9	-
Diffusion coefficient in pure air	m^2/day	0.142	0.106	0.149	USEPA 1999.
Diffusion coefficient in pure water	m^2/day	3.76E-05	5.68E-05	4.04E-05	USEPA 1999.
Henry's Law constant	Pa-m^3/mol	0.5	3.33	1.46	Mackay et al. 1992 as cited in USEPA 2000a. Exceptions include 1,2,3,4,6,7,8,9-OCDF; 1,2,3,4,7,8-HxCDF; and 1,2,3,6,7,8-HxCDF which are calculated by the VP/WS Ratio Technique (USEPA 2000a)
Melting point	degrees K	469.3	578.0	500.0	Mackay et al. 2000, exceptions include USEPA 2000b (1,2,3,6,7,8-HxCDD; 1,2,3,7,8,9-HxCDF; 1,2,3,7,8--PeCDD), ATSDR 1998(1,2,3,6,7,8-HxCDF; 1,2,3,7,8-PeCDF; 2,3,4,6,7,8-HxCDF) and NLM 2002 (1,2,3,7,8,9-HxCDD)
Molecular weight	g/mol	340.4	322.0	306.0	Mackay et al. 2000, exceptions include: ATSDR 1998(1,2,3,6,7,8-HxCDF; 1,2,3,7,8,9-HxCDF; 1,2,3,7,8-PeCDD; 1,2,3,7,8-PeCDF; 2,3,4,6,7,8-HxCDF) and NLM 2002 (1,2,3,6,7,8-HxCDD; 1,2,3,7,8,9-HxCDD)
Octanol-water partition coefficient (Kow)	L[water]/L[octanol]	3.16E+06	6.31E+06	1.26E+06	Mackay et al. 1992a as cited in USEPA 2000a, exceptions include: USEPA 2000b (1,2,3,6,7,8-HxCDD; 1,2,3,6,7,8-HxCDF; 1,2,3,7,8,9-HxCDD; 1,2,3,7,8,9-HxCDF; 2,3,4,6,7,8-HxCDF) and Sijm et al. 1989 (1,2,3,7,8-PeCDD)

Exhibit 17. Cadmium Chemical-Specific Properties for Abiotic Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value	Reference
Air Compartment Type			
Particle dry deposition velocity	m/day	260	Calculated from Mulbaier and Tisue 1980.
Washout Ratio	m3[air]/m3[rain]	200,000	MacKay et al. 1986.
Surface Soil Compartment Type			
Use input characteristic depth (boolean)	0 = no, Else = yes	0	Professional judgment.
Root Zone Soil Compartment Type			
Use input characteristic depth (boolean)	0 = no, Else = yes	0	Professional judgment.
Vadose Zone Soil Compartment Type			
Use input characteristic depth (boolean)	0 = no, Else = yes	0	Professional judgment.
Surface Water Compartment Type			
Ratio of concentration in water to concentration in algae to concentration dissolved in water	L[water]/g[algae wet wt]	1.87	McGeer et al. 2003.

Exhibit 18. Mercury Chemical-Specific Properties for Abiotic Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Air Compartment Type					
Particle dry deposition velocity	m/day	500	500	500	CalTOX value cited in McKone et al. 2001.
Demethylation rate	1/day	N/A	N/A	0	Professional judgment.
Methylation rate	1/day	0	0	0	Professional judgment.
Oxidation Rate	1/day	0.00385	0	0	Low end of half-life range (6 months to 2 years) in USEPA 1997.
Reduction rate	1/day	0	0	0	Professional judgment.
Washout Ratio	m3[air]/m3[rain]	200,000	200,000	200,000	Professional judgment.
Surface Soil Compartment Type					
Input characteristic depth (user supplied)	m	0.08	0.08	0.08	Not used (model set to calculate value).
Use input characteristic depth (boolean)	0 = no, Else = yes	0	0	0	Professional judgment.
Soil-water partition coefficient	L[water]/kg[soil wet wt]	1,000	58,000	7,000	USEPA 1997.
Vapor dry deposition velocity	m/day	50	2500	0	Hg(0) - from Lindberg et al. 1992 Hg(2) - estimate by USEPA using the Industrial Source Complex (ISC) Model - [See Vol. III, App. A of the Mercury Study Report (USEPA, 1997)].
Demethylation rate	1/day	N/A	N/A	0.06	Range reported in Porvari and Verta 1995 is 3E-2 to 6E-2 /day; value is average maximum potential demethylation rate constant under anaerobic conditions.
Methylation rate	1/day	0	0.001	0	Range reported in Porvari and Verta 1995 is 2E-4 to 1E-3 /day; value is average maximum potential methylation rate constant under anaerobic conditions.
Oxidation rate	1/day	0	0	0	Value assumed in USEPA 1997.
Reduction rate	1/day	0	1.25E-05	0	Value used for untilled surface soil (2cm), 10% moisture content, in USEPA 1997; general range is (0.0013/day)*moisture content to (0.0001/day)*moisture content for forested region (Lindberg 1996; Carpi and Lindberg 1997).

Exhibit 18. Mercury Chemical-Specific Properties for Abiotic Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Root Zone Soil Compartment Type					
Input characteristic depth (user supplied)	m	0.08	0.08	0.08	Not used (model set to calculate value).
Use input characteristic depth (boolean)	0 = no, Else = yes	0	0	0	Professional judgment.
Soil-water partition coefficient	L[water]/kg[soil wet wt]	1,000	58,000	7,000	USEPA 1997
Demethylation rate	1/day	N/A	N/A	0.06	Range reported in Porvari and Verta 1995 is 3E-2 to 6E-2 /day; value is average maximum potential demethylation rate constant under anaerobic conditions.
Methylation rate	1/day	0	0.001	0	Range reported in Porvari and Verta 1995 is 2E-4 to 1E-3 /day; value is average maximum potential methylation rate constant under anaerobic conditions.
Oxidation rate	1/day	0	0	0	Value assumed in USEPA 1997.
Reduction rate	1/day	0	3.25E-06	0	Value used for tilled surface soil (20cm), 10% moisture content, in USEPA 1997 (Lindberg 1996; Carpi and Lindberg, 1997).
Vadose Zone Soil Compartment Type					
Input characteristic depth (user supplied)	m	0.08	0.08	0.08	Not used (model set to calculate value),
Use input characteristic depth (boolean)	0 = no, Else = yes	0	0	0	Professional judgment.
Soil-water partition coefficient	L[water]/kg[soil wet wt]	1,000	58,000	7,000	USEPA 1997.
Demethylation rate	1/day	N/A	N/A	0.06	Range reported in Porvari and Verta 1995 is 3E-2 to 6E-2 /day; value is average maximum potential demethylation rate constant under anaerobic conditions.
Methylation rate	1/day	0	0.001	0	Range reported in Porvari and Verta 1995 is 2E-4 to 1E-3 /day; value is average maximum potential methylation rate constant under anaerobic conditions.
Oxidation rate	1/day	0	0	0	Value assumed in USEPA 1997.
Reduction rate	1/day	0	3.25E-06	0	Value used for tilled surface soil (20cm), 10% moisture content, in USEPA 1997 (Lindberg 1996; Carpi and Lindberg, 1997).

Exhibit 18. Mercury Chemical-Specific Properties for Abiotic Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Ground Water Compartment Type					
Soil-water partition coefficient	L[water]/kg[soil wet wt]	1,000	58,000	7,000	USEPA 1997.
Demethylation rate	1/day	N/A	N/A	0.06	Range reported in Porvari and Verta 1995 is 3E-2 to 6E-2 /day; value is average maximum potential demethylation rate constant under anaerobic conditions.
Methylation rate	1/day	0	0.001	0	Range reported in Porvari and Verta 1995 is 2E-4 to 1E-3 /day; value is average maximum potential methylation rate constant under anaerobic conditions.
Oxidation rate	1/day	1.00E-08	0	0	Small default nonzero value (0 assumed in USEPA 1997).
Reduction rate	1/day	0	3.25E-06	0	Value used for tilled surface soil (20cm), 10% moisture content, in USEPA 1997 (Lindberg 1996; Carpi and Lindberg 1997).
Surface Water Compartment Type					
Algal surface area-specific uptake rate constant	nmol/[μm^2-day-nmol]	0	2.04E-10	3.60E-10	Assumes radius = 2.5mm, Mason et al. 1995b, Mason et al. 1996; Hg(0) assumed same as Hg(2).
Dow ("overall Kow")	L[water]/kg[octanol]	0	-[a]	-[b]	Mason et al. 1996.
Solids-water partition coefficient	L[water]/kg[solids wet wt]	1,000	100,000	100,000	USEPA 1997.
Vapor dry deposition velocity	m/day	N/A	2500		USEPA 1997 (Vol. III, App. A).
Demethylation rate	1/day	N/A	N/A	0.013	Average of range of 1E-3 to 2.5E-2/day from Gilmour and Henry 1991.
Methylation rate	1/day	0	0.001	0	Value used in EPA 1997; range is from 1E-4 to 3E-4/day (Gilmour and Henry 1991).
Oxidation rate	1/day	0	0	0	Professional judgment.
Reduction rate	1/day	0	0.0075	0	Value used in USEPA 1997; reported values range from less than 5E-3/day for depths greater than 17m, up to 3.5/day (Xiao et al. 1995; Vandal et al. 1995; Mason et al. 1995a; Amyot et al. 1997).

Exhibit 18. Mercury Chemical-Specific Properties for Abiotic Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Sediment Compartment Type					
Solids-water partition coefficient	L[water]/kg[solids wet wt]	3,000	50,000	3,000	USEPA 1997.
Demethylation rate	1/day	N/A	N/A	0.0501	Average of range of 2E-4 to 1E-1/day from Gilmour and Henry 1991.
Methylation rate	1/day	0	1.00E-04	0	Value used in EPA 1997; range is from 1E-5 to 1E-3/day,Gilmour and Henry 1991.
Oxidation rate	1/day	0	0	0	Professional judgment.
Reduction rate	1/day	0	1.00E-06	0	Inferred value based on presence of Hg(0) in sediment porewater (USEPA 1997; Vandal et al. 1995).

[a]TRIM.FaTE Formula Property, which varies from 0.025 to 1.625 depending on pH and chloride concentration.

[b]TRIM.FaTE Formula Property, which varies from 0.075 to 1.7 depending on pH and chloride concentration.

Exhibit 19. PAH Chemical-Specific Properties for Abiotic Compartments
Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value							Reference
		BaP	BaA	BbF	BkF	Chr	DahA	IcdP	
Air Compartment Type									
Particle dry deposition velocity	m/day	500	500	500	500	500	500	500	McKone et al. 2001.
Half-life	day	0.046	0.125	0.596	0.458	0.334	0.178	0.262	Howard et al. 1991 / upper bound measured or estimated value.
Washout Ratio		200,000	200,000	200,000	200,000	200,000	200,000	200,000	Mackay et al. 1986.
Surface Soil Compartment Type									
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08	0.08	0.08	Not used (model set to calculate value).
Use input characteristic depth (boolean)	0 = No, Else = Yes	0	0	0	0	0	0	0	Professional judgment.
Halflife	day	530	680	610	2140	1000	940	730	MacKay et al. 2000.
Root Zone Soil Compartment Type									
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08	0.08	0.08	Not used (model set to calculate value).
Use input characteristic depth	0 = No, Else = Yes	0	0	0	0	0	0	0	Professional judgment.
Half-life	day	530	680	610	2140	1000	940	730	MacKay et al. 2000.
Vadose Zone Soil Compartment Type									
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08	0.08	0.08	Not used (model set to calculate value).
Use input characteristic depth (boolean)	0 = No, Else = Yes	0	0	0	0	0	0	0	Professional judgment.
Half-life	day	1060	1360	1220	4280	2000	1880	1460	Howard et al. 1991 / upper bound measured or estimated value for groundwater.
Groundwater Compartment Type									
Half-life	day	1060	1360	1220	4280	2000	1880	1460	Howard et al. 1991 / upper bound measured or estimated value for groundwater.

Exhibit 19. PAH Chemical-Specific Properties for Abiotic Compartments
Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value							Reference
		BaP	BaA	BbF	BkF	Chr	DahA	IcdP	
Surface Water Compartment Type									
RatioOfConcInAlgaeToConcDissolvedInWater	(g[chem]/kg[algae]) / (g[chem]/L[water])	3610	3610	3610	3610	3610	3610	3610	BCF data for green algae for BaP from Lu et al. 1977
Half-life	day	0.138	0.375	90	62.4	1.626	97.8	750	Howard et al. 1991 / upper bound measured or estimated value.
Sediment Compartment Type									
Half-life	day	2290	2290	2290	2290	2290	2290	2290	Mackay et al. 1992 / PAH values are the mean half-life of the log class that Mackay et al. assigned for sediment, except for BbF and IcdP, which were not on Table 2.3.

Exhibit 20. Dioxin Chemical-Specific Properties for Abiotic Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value					
		1,2,3,4,6,7,8,9-OCDD	1,2,3,4,6,7,8,9-OCDF	1,2,3,4,6,7,8-HpCDD	1,2,3,4,6,7,8-HpCDF	1,2,3,4,7,8,9-HpCDF	1,2,3,4,7,8-HxCDD
Air Compartment Type							
Deposition Velocity	m/day	500	500	500	500	500	500
Halflife	day	162	321	64	137	122	42
Washout Ratio	m^3[air]/m^3[rain]	91000	22000	64000	32000	32000	9000
Surface Soil Compartment Type							
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08	0.08
Use input characteristic depth (boolean)	0 = No, Else = Yes	0	0	0	0	0	0
Halflife	day	3650	3650	3650	3650	3650	3650
Root Zone Soil Compartment Type							
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08	0.08
Use input characteristic depth	0 = No, Else = Yes	0	0	0	0	0	0
Halflife	day	3650	3650	3650	3650	3650	3650
Vadose Zone Soil Compartment Type							
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08	0.08
Use input characteristic depth (boolean)	0 = No, Else = Yes	0	0	0	0	0	0
Halflife	day	1008	1008	1008	1008	1008	1008
Groundwater Compartment Type							
Half-life	day	1008	1008	1008	1008	1008	1008
Surface Water Compartment Type							
Ratio Of Conc In Algae To Conc Dissolved In Water	(g[chem]/g[algae])/ (g[chem]/L[water])	1.025	1.025	1.025	1.025	1.025	1.025
Half-life	day	0.67	0.58	47	0.58	0.58	6.3
Sediment Compartment Type							
Half-life	day	1095	1095	1095	1095	1095	1095

Exhibit 20. Dioxin Chemical-Specific Properties for Abiotic Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value					
		1,2,3,4,7,8-HxCDF	1,2,3,6,7,8-HxCDD	1,2,3,6,7,8-HxCDF	1,2,3,7,8,9-HxCDD	1,2,3,7,8,9-HxCDF	1,2,3,7,8-PeCDD
Air Compartment Type							
Deposition Velocity	m/day	500	500	500	500	500	500
Halflife	day	78	28	55	28	51	18
Washout Ratio	m^3[air]/m^3[rain]	10000	9000	10000	9000	10000	18000
Surface Soil Compartment Type							
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08	0.08
Use input characteristic depth (boolean)	0 = No, Else = Yes	0	0	0	0	0	0
Halflife	day	3650	3650	3650	3650	3650	3650
Root Zone Soil Compartment Type							
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08	0.08
Use input characteristic depth	0 = No, Else = Yes	0	0	0	0	0	0
Halflife	day	3650	3650	3650	3650	3650	3650
Vadose Zone Soil Compartment Type							
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08	0.08
Use input characteristic depth (boolean)	0 = No, Else = Yes	0	0	0	0	0	0
Halflife	day	1008	1008	1008	1008	1008	1008
Groundwater Compartment Type							
Half-life	day	1008	1008	1008	1008	1008	1008
Surface Water Compartment Type							
Ratio Of Conc In Algae To Conc Dissolved In Water	(g[chem]/g[algae])/ (g[chem]/L[water])	1.025	1.025	1.025	1.025	1.025	1.025
Half-life	day	0.58	6.3	0.58	6.3	0.58	2.7
Sediment Compartment Type							
Half-life	day	1095	1095	1095	1095	1095	1095

Exhibit 20. Dioxin Chemical-Specific Properties for Abiotic Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value				
		1,2,3,7,8-PeCDF	2,3,4,6,7,8-HxCDF	2,3,4,7,8-PeCDF	2,3,7,8-TCDD	2,3,7,8-TCDF
Air Compartment Type						
Deposition Velocity	m/day	500	500	500	500	500
Halflife	day	31	59	33	12	19
Washout Ratio	m^3[air]/m^3[rain]	13000	10000	14000	18000	19000
Surface Soil Compartment Type						
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08
Use input characteristic depth (boolean)	0 = No, Else = Yes	0	0	0	0	0
Halflife	day	3650	3650	3650	3650	3650
Root Zone Soil Compartment Type						
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08
Use input characteristic depth	0 = No, Else = Yes	0	0	0	0	0
Halflife	day	3650	3650	3650	3650	3650
Vadose Zone Soil Compartment Type						
Input characteristic depth	m	0.08	0.08	0.08	0.08	0.08
Use input characteristic depth (boolean)	0 = No, Else = Yes	0	0	0	0	0
Halflife	day	1008	1008	1008	1008	1008
Groundwater Compartment Type						
Half-life	day	1008	1008	1008	1008	1008
Surface Water Compartment Type						
Ratio Of Conc In Algae To Conc Dissolved In Water	(g[chem]/g[algae])/ (g[chem]/L[water])	1.025	1.025	1.025	1.025	1.025
Half-life	day	0.19	0.58	0.19	2.7	0.18
Sediment Compartment Type						
Half-life	day	1095	1095	1095	1095	1095

Exhibit 20. Dioxin Chemical-Specific Properties for Abiotic Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Reference
Air Compartment Type	
Deposition Velocity	McKone et al. 2001
Halflife	Atkinson 1996 as cited in USEPA 2000; vapor phase reaction with hydroxyl radical
Washout Ratio	Vulykh et al. 2001
Surface Soil Compartment Type	
Input characteristic depth	Not used (model set to calculate value)
Use input characteristic depth (boolean)	Professional judgment
Halflife	Mackay et al. 2000; the degradation rate was cited by multiple authors, value is for 2,3,7,8-TCDD
Root Zone Soil Compartment Type	
Input characteristic depth	not used (model set to calculate value)
Use input characteristic depth	professional judgment
Halflife	Mackay et al. 2000; the degradation rate was cited by multiple authors, value is for 2,3,7,8-TCDD
Vadose Zone Soil Compartment Type	
Input characteristic depth	Not used (model set to calculate value).
Use input characteristic depth (boolean)	Professional judgment.
Halflife	Average value of the range presented in Mackay et al. 2000; based on estimated unacclimated aerobic biodegradation half-life, value is for 2,3,7,8-TCDD.
Groundwater Compartment Type	
Half-life	Average value of the range presented in Mackay et al. 2000; based on estimated unacclimated aerobic biodegradation half-life, value is for 2,3,7,8-TCDD.
Surface Water Compartment Type	
Ratio Of Conc In Algae To Conc Dissolved In Water	BCF data for green algae for 2,3,7,8-TCDD from Isense 1978, at 32 days.
Half-life	Kim and O'Keefe. 1998, as cited in USEPA. 2000.
Sediment Compartment Type	
Half-life	Estimation based on Adriaens and Grbic-Galic 1992,1993 and Adriaens et al. 1995 as cited in USEPA 2000.

Exhibit 21. Cadmium Chemical-Specific Properties for Plant Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value	Reference
Leaf Compartment Type			
Transfer factor to leaf particle	1/day	0.002	Professional judgment.
Particle on Leaf Compartment Type			
Transfer factor to leaf	1/day	0.200	Professional judgment.
Root Compartment Type - Grasses and Herbs [a]			
Root to Root Soil Partition- Alpha of Steady State	unitless	0.95	Henning et al. 2001.
Root to Root Soil Partition- Partitioning Coefficient	m^3[bulk root soil]/m^3[root]	0.23	Nriagu 1980; based on average value calculated from various agricultural plant species.
Root to Root Soil Partition- Time to Reach Alpha	day	28	Henning et al. 2001.
Stem Compartment Type - Grasses and Herbs [a]			
Transpiration stream concentration factor (TSCF)	m^3[soil pore water]/m^3[xylem fluid]	0.45	Tsiros et al. 1999.
Aquatic Plants			
Macrophyte Compartment Type			
Water Column Dissolved Partition- Alpha of Equilibrium	unitless	0.95	Maine et al. 2001; based on assumption that equilibrium was nearly reached during 21 day experiment.
Water Column Dissolved Partition- Partition Coefficient	L[water]/kg[macrophyte wet wt]	100	Maine et al. 2001; based on calculations from an average of four macrophyte species.
Water Column Dissolved Partition- Time to Reach Equilibrium	day	21	Maine et al. 2001.

[a] Roots and stems are not modeled for deciduous or coniferous forest in the current version of TRIM.FaTE.

Exhibit 22. Mercury Chemical-Specific Properties for Plant Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Leaf Compartment Type					
Transfer factor to leaf particle	1/day	0.002	0.002	0.002	Professional judgment (assumed 1% of transfer factor from leaf particle to leaf).
Demethylation rate	1/day	N/A	N/A	0.03	Calculated from Bache et al. 1973.
Methylation rate	1/day	0	0	0	Assumed from Gay 1975, Bache et al. 1973.
Oxidation rate	1/day	1.0E+06	0	0	Professional judgment; assumed close to instantaneous
Reduction rate	1/day	0	0	0	Professional judgment.
Particle on Leaf Compartment Type					
Transfer factor to leaf	1/day	0.2	0.2	0.2	Professional judgment.
Demethylation rate	1/day	N/A	N/A	0	Professional judgment.
Methylation rate	1/day	0	0	0	Professional judgment.
Oxidation rate	1/day	0	0	0	Professional judgment.
Reduction rate	1/day	0	0	0	Professional judgment.
Root Compartment Type - Grasses and Herbs [a]					
Alpha for root-root zone bulk soil	unitless	0.95	0.95	0.95	Selected value.
Root/root-zone-soil-water partition coefficient	m^3[bulk root soil]/ m^3[root]	0	0.18	1.2	Hg2- geometric mean Leonard et al. 1998, John 1972, Hogg et al. 1978; MHg- assumed, based on Hogg et al. 1978.
t-alpha for root-root zone bulk soil	day	21	21	21	Professional judgment.
Demethylation rate	1/day	N/A	N/A	0	Professional judgment.
Methylation rate	1/day	0	0	0	Professional judgment.

Exhibit 22. Mercury Chemical-Specific Properties for Plant Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Oxidation rate	1/day	0	0	0	Professional judgment.
Reduction rate	1/day	0	0	0	Professional judgment.
Stem Compartment Type - Grasses and Herbs [a]					
Transpiration stream concentration factor (TSCF)	m^3[soil pore water]/ m^3[xylem fluid]	0	0.5	0.2	Calculation from Norway spruce, Scots pine, Bishop et al. 1998.
Demethylation rate	1/day	N/A	N/A	0.03	Calculated from Bache et al. 1973.
Methylation rate	1/day	0	0	0	Professional judgment.
Oxidation rate	1/day	0	0	0	Professional judgment.
Reduction rate	1/day	0	0	0	Professional judgment.
Aquatic Plants					
Macrophyte Compartment Type					
Water Column Dissolved Partition-Alpha of Equilibrium	unitless	0.95	0.95	0.95	Selected value.
Water Column Dissolved Partition-Partition Coefficient	L[water]/ kg[macrophyte wet wt]	0.883	0.883	4.4	*Elodea densa,* Ribeyre and Boudou 1994.
Water Column Dissolved Partition-Time to Reach Equilibrium	unitless	0.95	0.95	0.95	Selected value.
Oxidation rate	1/day	1.00E+09	0	0	Professional judgment.
t-alpha	day	18	18	18	Experiment duration from Ribeyre and Boudou 1994.

[a] Roots and stems are not modeled for deciduous or coniferous forest in the current version of TRIM.FaTE.

Exhibit 23. PAH Chemical-Specific Properties for Plant Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value							Reference
		BaP	BaA	BbF	BkF	Chr	DahA	IcdP	
Terrestrial Plants									
Leaf Compartment Type									
Transfer factor to leaf particle	1/day	1.00E-04	1.00E-04	1.00E-04	1.00E-04	1.00E-04	1.00E-04	1.00E-04	Professional judgment.
Half-life	day	3.5	3.5	3.5	3.5	3.5	3.5	3.5	Edwards 1988 (as cited in Efroymson 1997)/ calculated from metabolic rate constant.
Particle on Leaf Compartment Type									
Transfer factor to leaf	1/day	1.00E-04	1.00E-04	1.00E-04	1.00E-04	1.00E-04	1.00E-04	1.00E-04	Professional judgment.
Half-life	day	2.31	1.84	3.56	17.8	4.12	17.8	17.8	Edwards 1988 (as cited in Efroymson 1997)/ calculated from metabolic rate constant
Root Compartment Type - Grasses and Herbs [a]									
Half-life	day	34.6	34.6	34.6	34.6	34.6	34.6	34.6	Edwards 1988 (as cited in Efroymson 1997)/ calculated from metabolic rate constant.
Stem Compartment Type - Grasses and Herbs [a]									
Half-life	day	3.5	3.5	3.5	3.5	3.5	3.5	3.5	Edwards 1988 (as cited in Efroymson 1997)/ calculated from metabolic rate constant.
Aquatic Plants									
Macrophyte Compartment Type									
Half-life	days	3.5	3.5	3.5	3.5	3.5	3.5	3.5	Edwards 1988 (as cited in Efroymson 1997)/ calculated from metabolic rate constant.

[a] Roots and stems are not modeled for deciduous or coniferous forest in the current version of TRIM.FaTE.

Exhibit 24. Doixin Chemical-Specific Properties for Plant Compartments Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value	Reference
		All Dioxins	
Terrestrial Plants			
Leaf Compartment Type			
Transfer factor to leaf particle	1/day	0.003	Calculated as 1 percent of transfer factor to leaf; highly uncertain.
Half-life	day	70	Arjmand and Sandermann 1985, as cited in Komoba et al. 1995; soybean root cell culture metabolism test data for DDE.
Particle on Leaf Compartment Type			
Transfer factor to leaf	1/day	0.3	Professional judgment based on USEPA 2000c (an estimate for mercury) and Trapp 1995; highly uncertain.
Half-life	day	4.4	McCrady and Maggard 1993; photodegradation sorbed to grass foliage in sunlight; assumed 10 sunlight per day.
Root Compartment Type - Grasses and Herbs [a]			
Half-life	day	70	Arjmand and Sandermann 1985, as cited in Komoba, et al. 1995; soybean root cell culture metabolism test data for DDE.
Root Soil Water Interaction - Alpha	unitless	0.95	Professional judgment.
Stem Compartment Type - Grasses and Herbs [a]			
Half-life	day	70	Arjmand and Sandermann 1985, as cited in Komoba, et al. 1995; soybean root cell culture metabolism test data for DDE.
Aquatic Plants			
Macrophyte Compartment Type			
Half-life	days	70	Arjmand and Sandermann 1985, as cited in Komoba et al. 1995; soybean root cell culture metabolism test data for DDE.

[a] Roots and stems are not modeled for deciduous or coniferous forest in the current version of TRIM.FaTE.

Exhibit 25. Cadmium Chemical-Specific Properties for Aquatic Species Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value	Reference
Benthic Invertebrate Compartment Type			
Sediment Partitioning - Alpha of Equilibrium	unitless	0.95	Professional judgment
Sediment Partitioning - Partition Coefficient	kg[bulk sed/kg[invertebrate wet wt]	0.27	Professional judgment
Sediment Partitioning - Time to Reach Alpha of Equilibrium	day	21	Hare et al. 2001
Absorption Rate Constant	unitless	1.23	Calculated based on body weight from regression in Hendriks & Heikens 2000
Elimination Rate Constant	unitless	2.82E-03	Professional judgment
Benthic Omnivore Compartment Type			
Assimilation efficiency from food	unitless	0.1	Professional judgment based on Yan and Wang 2002.
Absorption Rate Constant	unitless	1.23E+00	Calculated based on body weight from regression in Hendriks & Heikens 2000
Elimination Rate Constant	unitless	2.82E-03	Professional judgment
Benthic Carnivore Compartment Type			
Assimilation efficiency from food	unitless	0.1	Professional judgment based on Yan and Wang 2002.
Absorption Rate Constant	unitless	6.60E-01	Calculated based on body weight from regression in Hendriks & Heikens 2000
Elimination Rate Constant	unitless	1.68E-03	Professional judgment
Water-column Herbivore Compartment Type			
Assimilation efficiency from food	unitless	0.1	Professional judgment based on Yan and Wang 2002.
Assimilation efficiency from plants		0.1	Professional judgment based on Yan and Wang 2002.
Absorption Rate Constant	unitless	2.46	calculated based on body weight from regression in Hendriks & Heikens 2000
Elimination Rate Constant	unitless	5.02E-03	Professional judgment
Water-column Omnivore Compartment Type			
Assimilation efficiency from food	unitless	0.1	Professional judgment based on Yan and Wang 2002.
Assimilation efficiency from plants		0.1	Professional judgment based on Yan and Wang 2002.
Absorption Rate Constant	unitless	1.232020679	Calculated based on body weight from regression in Hendriks & Heikens 2000
Elimination Rate Constant	unitless	2.82E-03	Professional judgment
Water-column Carnivore Compartment Type			
Assimilation efficiency from food	unitless	0.1	Professional judgment based on Yan and Wang 2002.
Absorption Rate Constant	unitless	0.660223535	Calculated based on body weight from regression in Hendriks & Heikens 2000
Elimination Rate Constant	unitless	1.68E-03	Professional judgment

Exhibit 26. Mercury Chemical-Specific Properties for Aquatic Species Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Benthic Invertebrate Compartment Type					
Alpha of equilibrium for sediment partitioning	unitless	0.95	0.95	0.95	Selected value.
Benthic invertebrate-bulk sediment partition coefficient	kg[bulk sediment]/kg[invertebrate wet wt]	0.0824	0.0824	5.04	Hg(0) - assumed based on Hg(2) value; Hg(2) and MHg - Saouter et al. 1991.
t-alpha for equilibrium for sediment partitioning	day	14	14	14	Experiment duration from Saouter et al. 1991.
All Fish Compartments Types [a]					
Elimination adjustment factor	unitless	3	3	1	Trudel and Rasmussen 1997.
Assimilation efficiency from food	unitless	0.04	0.04	0.2	Phillips and Gregory 1979.
Demethylation rate	1/day	N/A	N/A	0	Professional judgment.
Methylation rate	1/day	0	0	0	Professional judgment.
Oxidation rate	1/day	1.0E+06	0	0	Professional judgment.
Reduction rate	1/day	0	0	0	Professional judgment.
Water-column Herbivore Compartment Type					
Assimilation efficiency from plants	unitless	1	1	1	Phillips and Gregory 1979.
Water-column Omnivore Compartment Type					
Assimilation efficiency from food	unitless	1	1	1	Phillips and Gregory 1979.

[a] Screening scenario includes: Benthic Omnivore, Benthic Carnivore, Water-column Herbivore, Water-column Omnivore, and Water-column Carnivore.

Exhibit 27. PAH Chemical-Specific Properties for Aquatic Species Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value							Reference
		BaP	BaA	BbF	BkF	Chr	DahA	IcdP	
Benthic Invertebrate Compartment Type									
Clearance constant	unitless	157.6	157.6	157.6	157.6	157.6	157.6	157.6	Stehly et al. 1990 / estimated for mayfly, 120-day-old nymphs.
alpha of equilibrium for sediment partitioning	unitless	0.95	0.95	0.95	0.95	0.95	0.95	0.95	Professional judgment.
talpha for equilibrium for sediment partitioning	days	14	14	14	14	14	14	14	Professional judgment.
V_d (ratio of concentration in benthic invertebrates to concentration in water)	ml/g	7235.0	7235.0	7235.0	7235.0	7235	7235	7235	Stehly et al. 1990 / estimated for mayfly, 120-day-old nymphs.
Half-life	day	1.5	1.5	1.5	1.5	1.5	1.5	1.5	Stehly et al. 1990 / calculated from estimated elimination/depuration rate constant estimated for mayfly, 120-day-old nymphs.
All Fish Compartment Types[a]									
Gamma_fish	unitless	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Thomann 1989.
Assimilation efficiency from food[b]	unitless	1.0	1.0	1.0	1.0	1.0	1.0	1.0	Professional judgment.
Half-life	day	2.8	2.8	2.8	2.8	2.8	2.8	2.8	Spacie et al. 1983, as cited in MacKay et al. 1992 (bluegill sunfish) for benzo(a)pyrene.
Benthic Omnivore Compartment Type									
Assimilation efficiency from plants[b]	unitless	1.0	1.0	1.0	1.0	1.0	1.0	1.0	Professional judgment.
Water Column Omnivore Compartment Type									
Assimilation efficiency from plants[b]	unitless	1.0	1.0	1.0	1.0	1.0	1.0	1.0	Professional judgment.

[a] Screening scenario includes: Benthic Omnivore, Benthic Carnivore, Water-column Herbivore, Water-column Omnivore, and Water-column Carnivore.

[b] All ingestion assimilation efficiencies set to 1 to be consistent with excretion rate calculations.

Exhibit 28. Dioxin Chemical-Specific Properties for Aquatic Species
Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value								
		1,2,3,4,6,7,8, 9-OCDD	1,2,3,4,6,7,8, 9-OCDF	1,2,3,4,6,7,8- HpCDD	1,2,3,4,6,7,8- HpCDF	1,2,3,4,7,8,9- HpCDF	1,2,3,4,7,8- HxCDD	1,2,3,4,7,8- HxCDF	1,2,3,6,7,8- HxCDD	1,2,3,6,7,8- HxCDF
Benthic Invertebrate Compartment										
Clearance constant	unitless	0	0	0	0	0	0	0	0	0
Sediment Partitioning Partition Coefficient	kg/kg	0.0013	0.0017	0.0055	0.0012	0.042	0.033	0.0081	0.013	0.02
Sediment Partitioning Alpha of Equilibrium	unitless	0.95	0.95	0.95	0.95	0.95	0.95	0.95	0.95	0.95
Sediment Partitioning Time to Reach Alpha of Equilibrium	days	120	42	120	42	42	120	42	120	42
V_d (ratio of concentration in benthic invertebrates to concentration in water)	ml/g	0	0	0	0	0	0	0	0	0
Half-life	day	5776.2	5776.2	5776.2	5776.2	5776.2	5776.2	5776.2	5776.2	5776.2
All Fish Compartments [a]										
Assimilation efficiency from food	unitless	0.03	0.03	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Chemical Uptake Rate Via Gill	L[water]/kg[fish wet wt]-day	142	N/A [b]	14	N/A [b]	N/A [b]	127	N/A [b]	127	N/A [b]
Gamma_fish	unitless	N/A [b]	0.2	N/A [b]	0.2	0.2	N/A [b]	0.2	N/A [b]	0.2
Half-life	day	693.15	346.57	346.57	346.57	346.57	495.11	495.11	495.11	495.11
Water Column Herbivore Compartment										
Assimilation efficiency from plants	unitless	0.03	0.03	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Water Column Omnivore Compartment										
Assimilation efficiency from plants	unitless	0.03	0.03	0.5	0.5	0.5	0.5	0.5	0.5	0.5

[a] Screening scenario includes: Benthic Omnivore, Benthic Carnivore, Water-column Herbivore, Water-column Omnivore, and Water-column Carnivore.

[b] N/A = not applicable. This parameter is used in calculating the uptake when measured data are unavailable.

Exhibit 28. Dioxin Chemical-Specific Properties for Aquatic Species
Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Value							
		1,2,3,7,8,9-HxCDD	1,2,3,7,8,9-HxCDF	1,2,3,7,8-PeCDD	1,2,3,7,8-PeCDF	2,3,4,6,7,8-HxCDF	2,3,4,7,8-PeCDF	2,3,7,8-TCDD	2,3,7,8-TCDF
Benthic Invertebrate Compartment									
Clearance constant	unitless	0	0	0	0	0	0	0	0
Sediment Partitioning Partition Coefficient	kg/kg	0.015	0.067	0.098	0.024	0.072	0.17	0.205	0.056
Sediment Partitioning Alpha of Equilibrium	unitless	0.95	0.95	0.95	0.95	0.95	0.95	0.95	0.95
Sediment Partitioning Time to Reach Alpha of Equilibrium	days	120	42	120	42	42	42	120	42
V_d (ratio of concentration in benthic invertebrates to concentration in water)	ml/g	0	0	0	0	0	0	0	0
Half-life	day	5776.2	5776.2	5776.2	5776.2	5776.2	5776.2	5776.2	5776.2
All Fish Compartments [a]									
Assimilation efficiency from food	unitless	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Chemical Uptake Rate Via Gill	L[water]/kg[fish wet wt]-day	127	N/A [b]	700	N/A [b]	N/A [b]	N/A [b]	380	N/A [b]
Gamma_fish	unitless	N/A [b]	0.2	N/A [b]	0.2	0.2	0.2	N/A [b]	0.2
Half-life	day	495.11	495.11	420.09	420.09	495.11	420.09	5251.1	5251.1
Water Column Herbivore Compartment									
Assimilation efficiency from plants	unitless	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Water Column Omnivore Compartment									
Assimilation efficiency from plants	unitless	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5

[a] Screening scenario includes: Benthic Omnivore, Benthic Carnivore, Water-column Herbivore, Water-column Omnivore, and Water-column

[b] N/A = not applicable. This parameter is used in calculating the uptake when measured data are unavailable.

Exhibit 28. Dioxin Chemical-Specific Properties for Aquatic Species
Documentation for the TRIM.FaTE Screening Scenario

Parameter Name	Units	Reference
Benthic Invertebrate Compartment		
Clearance constant	unitless	Professional judgment.
Sediment Partitioning Partition Coefficient	kg/kg	TCDD data for sandworm in Rubenstein et al. 1990; dry weight sediment. PeCDF: multiplied TCDD partition coefficient for sandworm by congener-specific bioaccumulation equivalency factor in GLWQI from USEPA 1999.
Sediment Partitioning Alpha of Equilibrium	unitless	Professional judgment.
Sediment Partitioning Time to Reach Alpha of Equilibrium	days	TCDD: professional judgment; PeCDF: Rubinstein et al. 1990; data for TCDF in sandworm.
V_d (ratio of concentration in benthic invertebrates to concentration in water)	ml/g	Professional judgment.
Half-life	day	Change source to f-pass
All Fish Compartments [a]		
Assimilation efficiency from food	unitless	TCDD: calculated from data in Kleeman et al. 1986b trout data as cited in USEPA 1993; PeCDF: used assimilation efficiency for TCDD in trout.
Chemical Uptake Rate Via Gill	L[water]/kg[fish wet wt]-day	Muir et al. 1986.
Gamma_fish	unitless	Thomann 1989
Half-life	day	Change source to f-pass
Water Column Herbivore Compartment		
Assimilation efficiency from plants	unitless	TCDD: calculated from data in Kleeman et al. 1986b trout data as cited in USEPA 1993; PeCDF: used assimilation efficiency for TCDD in trout.
Water Column Omnivore Compartment		
Assimilation efficiency from plants	unitless	TCDD: calculated from data in Kleeman et al. 1986b trout data as cited in USEPA 1993; PeCDF: used assimilation efficiency for TCDD in trout.

[a] Screening scenario includes: Benthic Omnivore, Benthic Carnivore, Water-column Herbivore, Water-column Omnivore, and Water-column Carnivore.

[b] N/A = not applicable. This parameter is used in calculating the uptake when measured data are unavailable.

ATTACHMENT C-2: Description of Multimedia Ingestion Risk Calculator (MIRC) Used for RTR Exposure and Risk Estimates

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1 Introduction

1.1 Purpose and Overview

This document provides a detailed description of the *Multimedia Ingestion Risk Calculator* (MIRC), an Access-based tool and database designed to assist in estimating risks via multiple ingestion pathways, particularly for food products grown or raised at home or on a farm. MIRC was designed to estimate risks to humans from ingestion of produce or animal products, fish, and water in the vicinity of a source of chemical emissions to air. The user can evaluate either generalized (e.g., conservative default) or more site-specific scenarios using the same tool. MIRC includes a database of exposure parameter values, offering the user the option of selecting mean, median, and upper percentile values for many parameters, data permitting. Generally conservative default values were assigned to each parameter in the tool and the default configuration is used for initial risk screening efforts by EPA's Office of Air Quality Planning and Standards' (OAQPS) for Risk and Technology Review (RTR) multimedia risk assessments (the default inputs were used to calculate the *de minimis* screening thresholds). MIRC also allows the user to define the farm food chain (FFC) parameter values and receptor characteristics to better represent a site-specific scenario.

With user-input concentrations for one or more chemicals in air and soil and air-to-surface deposition rates, MIRC calculates the chemical's concentrations in home- or farm-grown produce and animal food products using FFC algorithms adapted from EPA's *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities* (hereafter referred to as HHRAP; EPA 2005a). MIRC uses these calculated concentrations, along with user-input chemical concentrations for fish and drinking water, to estimate chemical intake rates, as average daily doses (ADDs), for adults, children, and nursing infants. Users can obtain chemical input concentrations and deposition rates from measurements at an actual site or from a transport and fate model, such as TRIM.FaTE as is done for RTR risk assessment.

For a specified set of chemical concentrations and MIRC parameter options, MIRC calculates ADDs separately for adults, four age groups of children, and infants to reflect differences in food ingestion rates and diet at different lifestages. MIRC estimates age-specific hazard quotients (HQs) as the ratio of age-specific ADDs to the reference dose (RfD) for a chemical. The most appropriate HQ for a chemical depends on its toxic mode of action and the duration of exposure required to produce an effect. MIRC also estimates average lifetime ADDs and compares those to cancer slope factors (CSFs) to estimate cancer risks. A breast milk ingestion pathway is included to estimate exposure and risks to nursing infants.

MIRC was developed to be a flexible, transparent application using Microsoft Access software. The tool includes chemical transfer and ingestion exposure algorithms and a database of parameter values, many with several options, used by these equations. The MIRC database includes values for the relevant physiochemical properties and toxicity reference values for more than 500 chemicals, including approximately 60 inorganics taken primarily from a database developed for HHRAP (EPA 2005a). Although designed for OAQPS' RTR assessments for sources of hazardous air pollutants (HAPs), the tool is flexible in its design and can be used to assess risks in many other contexts where soil and air concentrations are predicted or measured.

1.2 Scope of MIRC

For persistent and bioaccumulative (PB) chemicals, risks from direct inhalation of the chemical can be much less than risks from ingestion of the chemical in water, fish, and food products

grown in an area of chemical deposition. Vegetables and fruits in such areas can become contaminated directly by deposition of the airborne chemical to foliage, fruits, and vegetables or indirectly by root uptake of the chemical deposited to soils. Livestock can be exposed to the PB chemicals via ingestion of contaminated forage and incidental ingestion of contaminated soils.

For PB chemicals, evaluation of the inhalation pathway for air pollutants may reveal only a portion of the risk to individuals in such populations. Households that consume high quantities of self-caught fish or locally grown produce and animal products may be particularly susceptible to ingestion of chemicals transferred from air in the vicinity of an air emissions source. For PB chemicals in particular, therefore, EPA developed methods of estimating risk from indirect exposure pathways associated with the deposition of airborne chemicals to gardens and farms, as described in HHRAP (EPA 2005a).

MIRC is an Access-based tool that facilitates calculation of risks associated with the indirect ingestion exposure pathways for persons consuming produce and animal products grown in an air depositional area of concern. The tool uses algorithms described in or adapted from HHRAP to calculate exposures from the produce and animal products. Included with MIRC is a fish ingestion pathway and drinking water ingestion pathway for scenarios in which those pathways may be important. MIRC also includes a breast milk ingestion pathway for nursing infants based primarily on EPA's *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions* (hereafter MPE; EPA 1998).

1.3 Use in EPA's Air Toxics Program

MIRC was designed to help predict human health risks from PB HAPs for EPA's RTR assessments. EPA evaluates the fate of HAP releases to air from source categories after implementation of technology-based Maximum Achievable Control Technology (MACT) standards. For volatile chemicals that do not partition to other environmental media and for non-persistent chemicals that degrade relatively quickly in the environment, evaluation of health risks from direct inhalation of the chemical in air can provide reasonable estimates of total risk.

For PB-HAPs, however, indirect exposure pathways, such as ingestion, might contribute more to total risk than the inhalation pathway. EPA therefore developed several computer software tools to assist in evaluating exposure and risk from non-inhalation pathways. EPA developed the *Total Risk Integrated Methodology* (TRIM) *Environmental Fate, Transport, and Ecological Exposure* (TRIM.FaTE) computer program to simulate the release, transport, and fate of HAPs from a specific source throughout the area in which local (non-source) chemical deposition is likely to be a concern. TRIM.FaTE models the transport of individual chemicals from the source through air by advection (wind) of particle- and vapor-phase chemical and deposition of the chemical from air to terrestrial and aquatic ecosystems by wet and dry deposition. Movement of the chemical through a watershed via erosion and runoff, uptake by plants, and other abiotic and biotic transfer processes also are simulated. For the chemical that reaches surface waters, TRIM.FaTE models uptake and bioaccumulation to trophic level (TL) 3 and 4 fish (i.e., pan fish and game fish, respectively).

MIRC was developed to process TRIM.FaTE results, in particular, air deposition rates and the concentrations of a chemical, after a specified duration of emissions, in several spatially explicit environmental compartments, including air, surface and root-zone soils, surface and ground waters, and fish. MIRC uses those results to calculate exposure to the chemical through ingestion of locally grown foods, including various types of fruits and vegetables, poultry, swine, and dairy (and beef) cattle. MIRC also calculates the associated risks for individuals who consume those foods. MIRC was designed to use specific TRIM.FaTE results to estimate FFC

concentrations, ingestion exposures, and human health risks for OAQPS' RTR assessments. It uses the same approach that OAQPS intends to implement directly in its TRIM system via three modules beyond TRIM.FaTE: TRIM Farm Food Chain, TRIM.Expo$_{Ingestion}$, and TRIM.Risk.[1]

1.4 MIRC Highlights

Although designed to assist EPA OAQPS in its RTR assessments, MIRC is a stand-alone software application that can be used in other contexts. A user can supply either measured or estimated chemical concentrations for soil, air, water, and fish and air deposition rates likely for the location(s) of interest based on local meteorology. The user can accept the default values for many exposure parameters and screen for small possibilities of risk, or the user can select other options or overwrite parameter values to tailor the estimates to a specific scenario or location.

MIRC complies with EPA's latest guidelines for exposure and risk assessment, including HHRAP; the Agency's 2005 *Guidelines for Carcinogen Risk Assessment* (Cancer Guidelines)*, Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (Supplemental Guidance), and *Guidance on Selecting Age Groups for Monitoring and Assessing Childhood Exposures to Environmental Contaminants* (EPA 2005b,c,d); and its 2008 *Child-Specific Exposure Factors Handbook* (EPA 2008a). In particular, MIRC provides several important capabilities:

- When provided air and soil concentrations, the MIRC software package allows rapid calculation of screening-level exposures and risks associated with household consumption of locally grown/raised foods.
- MIRC can calculate exposures and risks associated with incidental ingestion of surface soils, fish consumption, and drinking water.
- The tool calculates ADDs (i.e., chemical intake rates) for six "built-in" age groups to allow use of age-group-specific body weights, ingestion rates, food preferences, and susceptibility to toxic effects.
- Its database of chemical information covers plant- and animal-specific transfer factors and other inputs that determine concentrations in farm food stuffs.
- Value options for receptor characteristics in the database include the mean and 50th, 90th, 95th, and 99th percentile values where data permit.
- For carcinogens with a mutagenic mode of action, MIRC estimates a lifetime ADD using the three lifestages and potency adjustment factors recommended in EPA's 2005 Cancer Guidelines and Supplemental Guidance.
- The data for exposure parameters in the tool have been updated to include the latest recommended values for children issued September 30, 2008, in the Agency's Child-Specific Exposure Factors Handbook (CSEFH) (EPA 2008a).

1.5 Organization of This Document

Sections 2 through 5 of this document describe the exposure and risk models implemented in MIRC. Section 2 provides an overview of the FFC exposure scenario and indicates options

[1] General information about the TRIM system is available at http://www.epa.gov/ttn/fera/trim_gen.html.

available to a user to tailor the scenario to specific applications. Section 3 describes the exposure algorithms used in MIRC, including how ADDs are calculated. Section 4 presents the toxicity reference values included in MIRC to calculate risks. Section 5 describes the risk characterization algorithms in MIRC. Section 6 of this document describes data input options for the model. Section 7 describes the default parameterization of MIRC for application to conservative risk screening assessments, and Section 8 provides the references. Appendix A provides guidance to users on how to set up and run MIRC for their own applications.

Note that the default parameterization described in Section 7 was used to estimate *de minimis* releases of PB-HAPs from facilities assumed to pose negligible risk to subsistence communities in the vicinity of a facility emitting the HAPs to air. Users of MIRC can modify the default values for many of the parameters to better represent a specific exposure scenario.

2 MIRC Overview

The *Multimedia Ingestion Risk Calculator* (MIRC) software package is designed to allow rapid calculation of screening-level exposures and risks associated with a subsistence farmer/fisher population in the vicinity of a source of chemical emissions to air. The tool allows a user to assess human exposures via ingestion pathways, including drinking water consumption, incidental soil ingestion, fish ingestion, and ingestion of ten types of farm food chain (FFC) products: exposed fruits, protected fruits, exposed vegetables, protected vegetables, root vegetables, beef, total dairy, pork, poultry, and eggs. The tool also includes a breast milk ingestion and risk module for nursing infants. For fruits and vegetables, the terms "exposed" and "protected" refer to whether the edible portion of the plant is exposed to the atmosphere.

The remainder of this overview consists of three sections. The first (Section 2.1) provides an overview of the MIRC software package. The second and third sections summarize the ingestion exposure pathways included in the tool and the "built-in" receptor age categories, respectively (Sections 2.2 and 2.3).

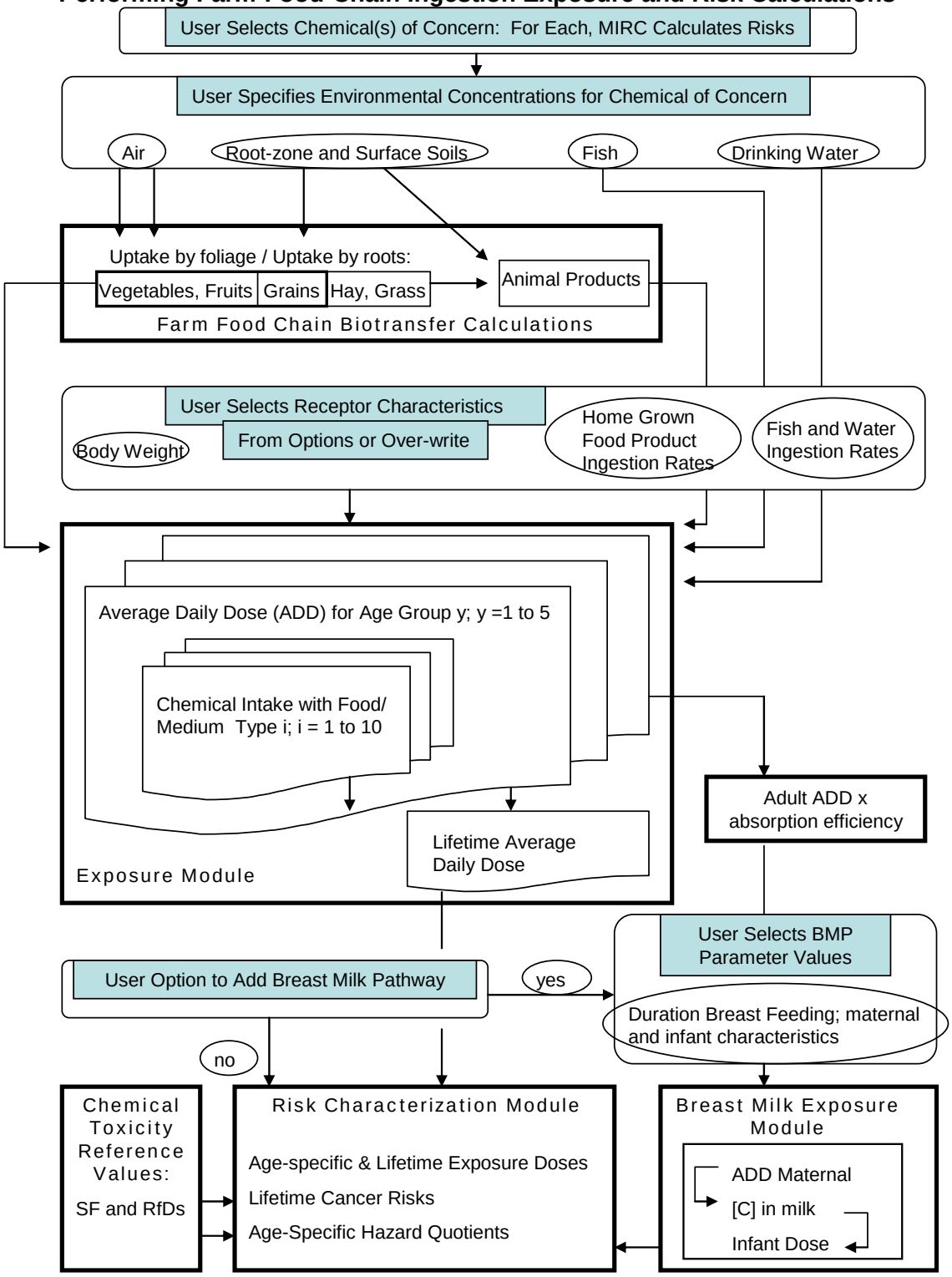
2.1 Software

The MIRC application includes the following components:

- A graphical user interface through which the user locates and accesses various input and output tables.
- Input tables in which the user can enter environmental concentrations of a chemical estimated for air, soil, drinking water, and fish tissue.
- Internal chemical transfer and exposure algorithms and database of options for FFC algorithm parameter value, chemical-specific inputs, and exposure factors.
- Tabulated outputs of calculated chemical concentrations in the various farm food products (e.g., fruits, vegetables, beef, eggs) and ADDs for those foods and for water and fish ingestion for each receptor category.
- Output tables with estimated cancer risks and non-cancer hazard estimates associated with total ingestion exposure to each chemical for each receptor category.

Exhibit 2-1 provides a flowchart displaying the types of inputs required or optional and general flow of calculations carried out by the tool.

Exhibit 2-1. Overview of Access-based MIRC Software Application for Performing Farm-Food-Chain Ingestion Exposure and Risk Calculations



An Access form within the graphical user interface enables the user to construct specific scenarios by choosing ingestion sources, receptor ages, and other input choices (e.g., diet composition, body weight percentiles). This feature facilitates the analysis of various exposure scenarios. To begin an analysis, the user must supply values for the following chemical-specific parameters for the scenario being evaluated:

- Air concentration of total chemical,
- Fraction chemical in air in vapor-phase,
- Wet and dry deposition rates for particle-phase chemical,
- Drinking water concentration,
- Chemical concentration in surface soils (two locations; can be tilled and untilled),
- Chemical concentration in root-zone soils (two locations; can be tilled and untilled), and
- Chemical concentrations in pan fish and in game fish.

Users can input measured values or values estimated by TRIM.FaTE or other models for these parameters.

The MIRC application uses the input data and a variety of empirical transfer factor values (included in its database) to estimate chemical concentrations in nine categories of FFC food types (Section 2.2). The FFC algorithms and transfer factor values included in MIRC are based on those presented in Chapter 5 of EPA's *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities,* hereafter referred to as HHRAP (EPA 2005a).

For outputs, MIRC is designed to calculate individual cancer risk and non-cancer hazard quotients for one chemical at a time. It is up to the risk assessor to determine if cancer risks or hazard quotients may be additive across two or more chemicals (i.e., if they cause toxic effects in the same target organ by the same mode of action, such as multiple PAHs that are carcinogenic by a mutagenic mode of action).

The tool assumes that the same individuals (farming family or household that gardens and raises animals) are exposed via all of the pathways specified (i.e., pathways with non-zero ingestion rates). The tool therefore is useful in estimating risk to the maximally exposed individuals (MEI) in a risk assessment. To evaluate multiple populations, the user must specify the full exposure scenario for each population separately.

2.2 Exposure Pathways

MIRC estimates the concentrations of chemicals in FFC food categories grown in an area of airborne chemical deposition using algorithms and parameter values provided in HHRAP (EPA 2005a). FFC foods are evaluated in ten categories: exposed fruit, protected fruit, exposed vegetables, protected vegetables, root vegetables, beef, total dairy, pork, poultry, and eggs. Exhibit 2-2 summarizes the pathways by which chemicals are transferred to these food media. Note that for a general screening-level assessment, all of the pathways can be modeled, as is the case for EPA's *Risk and Technology Review* (RTR) calculation of *de minimis* emission rates for persistent and bioaccumulative hazardous air pollutants (PB-HAPs) (EPA 2008b).

Exhibit 2-2. Transfer Pathways for Modeled Farm Food Chain (FFC) Media

Farm Food Media	Chemical Transfer Pathways
Exposed fruit and vegetables	• Direct deposition from air of particle-bound chemical • Air-to-plant transfer of vapor phase chemical • Root uptake from soil
Protected fruit and vegetables (including root vegetables)	• Root uptake from soil
Beef and total dairy (including milk)	• Ingestion of forage, silage, and grain [a] • Soil ingestion
Pork	• Ingestion of silage and grain [a] • Soil ingestion
Poultry and eggs	• Ingestion of grain [a] • Soil ingestion

[a] Chemical concentrations in forage, silage, and grain are estimated via intermediate calculations analogous to those used for aboveground produce.

Produce types included in the FFC can accumulate chemical directly from air and/or soil. For exposed produce, chemical mass is assumed to be transferred to plants from the air in two ways. First, particle-bound chemical can deposit directly on the plant surface. Second, the uptake of vapor-phase chemicals by plants through their foliage can occur. For both exposed and protected produce, the concentration in the plant derived from exposure to the chemical in soil is estimated using an empirical bioconcentration factor (BCF) that relates the concentration in the plant to the concentration present in the soil. For belowground root vegetables, a root concentration factor is applied. The algorithms used to estimate produce concentrations are presented in Section 3.1.1.

Chemical concentrations in animal products are estimated based on the amount of chemical consumed through the diet, including incidental ingestion of soil while grazing. The diet options for farm animals in MIRC include forage (plants grown on-site for animal grazing, such as grass), silage (wet forage grasses, fresh-cut hay, or other fresh plant material that has been stored and fermented), and feed grain products grown on the farm (e.g., corn, soybeans). All three animal feed products are assumed to accumulate chemical via root uptake from the soil. Forage and silage also can accumulate chemical via direct deposition of particle-bound chemical and vapor transfer.

The algorithms in MIRC are based on the assumptions that beef and dairy cattle consume all three feed products, while pigs consume only silage and grain and chickens consume only grain. The incidental ingestion of the chemical in soils during grazing or consumption of foods placed on the ground is estimated using empirical soil ingestion values. For secondary animal products (dairy products and eggs), chemical concentrations are estimated by applying a biotransfer factor to the estimated concentration in the "source" animal (cows and chickens, respectively). The algorithms used to estimate animal product concentrations are described in Section 3.1.2.

2.3 Receptor Groups

As noted in EPA risk assessment guidelines (EPA 2005b,c,d, 2008a), exposures of children are expected to differ from exposures of adults due to differences in body weights, ingestion rates, dietary preferences, and other factors. It is important, therefore, to evaluate the contribution of exposures during childhood to total lifetime risk using appropriate exposure factor values.

EPA's HHRAP (Chapter 4, EPA 2005a) recommends assessing exposures for children and adults separately, but considers all non-infant children in one category. Specifically, HHRAP recommends eight categories of receptor: farmer, child farmer, resident, child resident, fisher, child fisher, acute receptor, and nursing infant. Over time, different EPA programs have used different child age groupings to evaluate body weights, ingestion rates, and other parameter values needed to estimate chemical exposures and risks to children.

To improve the match between age groups used to estimate values across exposure parameters, in 2005, EPA recommended a standard set of child age categories for exposure and risk assessments (EPA 2005b). EPA recommended four age groups for infants: birth to < 1 month; 1 to < 3 months; 3 to < 6 months; and 6 to < 12 months. For young children, EPA recommended an additional four age groups: 1 to < 2 years; 2 to < 3 years; 3 to < 6 years; and 6 to < 11 years. Two age groupings were recommended for teenagers and young adults: 11 to < 16 years; and 16 to < 21 years. These age groupings correspond to different developmental stages and reflect different food ingestion rates per unit body weight, with the highest ingestion rates occurring for the youngest, most rapidly growing, age groups.

For assessment of cancer risks from early-life exposure, EPA recognizes that infants and children may be more sensitive to a carcinogenic chemical than adults, with cancers appearing earlier in life or with lower doses experienced during childhood (EPA 2005c,d). Thus, the "potency" of a carcinogen might be higher for infants and children than for adults. To date, however, data by which to evaluate the relative sensitivity of children and adults to the same daily dose of a carcinogen remain limited. Based on analyses of radioactive and other carcinogenic chemicals, EPA recommends evaluating two lifestages for children separately from adults for chemicals that cause cancer by a mutagenic mode of action (MOA): from birth to < 2 years and from 2 to < 16 years (EPA 2005c,d). EPA also suggests that, as data become available regarding carcinogens with a mutagenic MOA, further refinements of these age groupings may be considered.

For purposes of RTR assessment using MIRC, the selection of age categories is limited by the categories for which most of the FFC food ingestion rates have been calculated. In Chapter 13 of both its *Exposure Factors Handbook* (EFH; EPA 1997a) and its *Child-Specific Exposure Factors Handbook* (CSEFH; EPA 2008a), EPA summarized home-grown/raised food ingestion rates for four children's age groups: 1 to < 3 years; 3 to < 6 years; 6 to < 12 years; and 12 to < 20 years. Intake rates were not calculated for children younger than 1 year because infants are unlikely to consume those foods. They are more likely to be nursing or to be fed formula and other commercial baby-food products.

Although the age groupings used to estimate FFC ingestion rates do not match precisely the groupings that EPA recommended in 2005 for Agency exposure assessments (EPA 2005b), they are the only age-groupings for which such data are available. The U.S. Department of Agriculture's (USDA's) 1987-1988 *Nationwide Food Consumption Survey* (USDA 1992, 1993, 1994) remains the most recent survey of ingestion rates for home-grown foods, and EPA's analysis of those data, published in its 1997 EFH, remains the most recently published major analysis of those data. Because ingestion of home-grown produce and animal products are the primary exposure pathways for which MIRC was developed, those are the age groupings used for all child parameter values used to estimate exposure and risk in MIRC.

Thus, in MIRC, values for each exposure parameter were estimated for adults (20 to 70 years) and five children's age groups:

- infants under 1 year (i.e., 0 to < 1 year);
- children ages 1 through 2 years (i.e., 1 to < 3 years);
- children ages 3 through 5 years (i.e., 3 to < 6 years);
- children ages 6 through 11 years(i.e., 6 to < 12 years) and
- children ages 12 through 19 years (i.e., 12 to < 20 years).

Exposure and risks to infants under 1 year of age are estimated only for the breast-milk-ingestion pathway.

For assessing risks from exposures to carcinogenic chemicals that act via a mutagenic MOA, the two early lifestages recommended by EPA (EPA 2005c,d) also are included in MIRC:

- children under the age of 2 years (i.e., 0 to < 2 years); and
- children from 2 through 15 years (i.e., 2 to < 16 years).

Different age groupings are needed for the assessment of risks from carcinogenic chemicals with a mutagenic MOA and other carcinogens with other or unknown MOAs. Currently in MIRC, the only PB-HAPs with a mutagenic mode of carcinogenesis are some of the PAHs.

3 Exposure Algorithms

The exposure algorithms in MIRC are described below in four sections. Section 3.1 presents the algorithms used to estimate chemical concentrations in FFC foods from chemical concentrations in soil and air. Pathway-specific algorithms used to estimate chemical intakes by adults and non-infant children are described in Section 3.2, and total chemical intake calculations are described in Section 3.3. Finally, the sets of algorithms used to estimate chemical intake via consumption of breast milk by nursing infants are described in Section 3.4. As noted previously, the exposure algorithms used in MIRC are based on those presented in HHRAP (EPA 2005b). Any differences between MIRC and HHRAP are explained in this section.

3.1 Farm Food Chain Algorithms

The algorithms and parameters used to estimate chemical concentrations in produce and animal products are described in Sections 3.1.1 and 3.1.2, respectively. Discussions of the parameter value options and the values selected as defaults in MIRC for RTR risk assessment are provided in Section 6.2. The use of TRIM.FaTE to model chemical fate and transport in the environment prior to FFC calculations drives the most significant difference between the FFC algorithms included in HHRAP and the equations used for RTR. The approach in HHRAP uses estimated ambient air concentrations and deposition rates from dispersion model simulations that use unitized emission rates. Chemical-specific emission rates (adjusted for vapor and particle-bound fractions) are then incorporated into some of the HHRAP FFC algorithms to calculate concentrations in FFC media. Soil concentrations are calculated using a similar approach in HHRAP. For assessment of multipathway exposures for RTR, TRIM.FaTE is used to estimate air concentrations, air-to-surface deposition rates, and soil concentrations, and these outputs are used in the FFC algorithms.

3.1.1 Estimating Chemical Concentrations in Produce

Produce (vegetables and fruits) can become contaminated directly by deposition of airborne chemicals to foliage and fruits or indirectly by uptake of chemicals deposited to the soil. Given these two contamination processes, produce is divided into two main groups: aboveground and belowground produce. Aboveground produce is divided into fruits and vegetables. These groups are further subdivided into “exposed” and “protected” depending on whether the edible portion of the plant is exposed to the atmosphere or is protected by a husk, hull, or other outer covering.

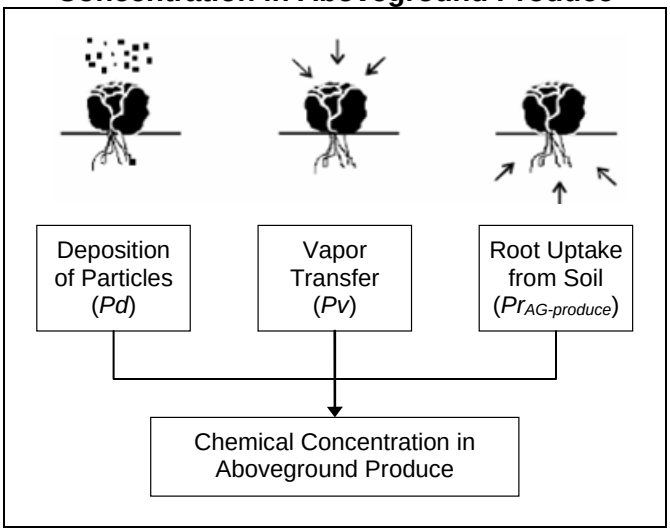
Exhibit 3-1 lists the pathways by which chemicals are transferred to the FFC produce categories. Note that for a general screening-level assessment, all of the pathways can be modeled, as was done for EPA’s calculation of *de minimis* emission rates for PB-HAPs in its RTR assessments (EPA 2008b), and as described in the “Technical Support Document for TRIM-Based Multipathway Screening Scenario for RTR”. Sections 3.1.1.1 and 3.1.1.1 describe the transfer pathways and algorithms for aboveground and belowground produce, respectively.

Exhibit 3-1. Chemical Transfer Pathways for Produce

Farm Food Media		Chemical Transfer Pathways
Aboveground Produce	Exposed fruits and vegetables	• Direct deposition from air of particle-bound chemical • Air-to-plant transfer of vapor phase chemical • Root uptake from soil
	Protected fruits and vegetables	• Root uptake from soil
Belowground Produce	Root vegetables	• Root uptake from soil

Aboveground Produce

Exhibit 3-2. Estimating Chemical Concentration in Aboveground Produce



For aboveground *exposed* produce, chemical mass is assumed to be transferred to plants from the air in three ways, as illustrated in Exhibit 3-2. First, particle-bound chemical can deposit directly on the plant surface via deposition (*Pd*). The amount of chemical accumulated is estimated based on the areal fraction of chemical deposition intercepted by the plant surface, minus a loss factor that is intended to account for removal of deposited chemical by wind and rain and changes in concentration due to growth dilution. Second, for chemical present in air in the vapor phase, the concentration of chemical accumulated by the plant's foliage is estimated using an empirical air-to-plant biotransfer factor (*Pv*). Third, the chemical concentration in the plant due to root uptake from the soil ($Pr_{AG\text{-}produce}$) is estimated using an empirical bioconcentration factor ($Br_{AG\text{-}produce}$) that relates the chemical concentration in the plant to the average chemical concentration in the soil at the root-zone depth in the produce-growing area ($Cs_{root\text{-}zone\_produce}$).

The edible portions of aboveground *protected* produce are not subject to contamination via particle deposition (*Pd*) or vapor transfer *(Pv*). Therefore, root uptake of chemicals is the primary mechanism through which aboveground protected produce becomes contaminated. The chemical concentration in the aboveground plant due to root uptake from soil ($Pr_{AG\text{-}produce\text{-}DW}$) is estimated using an empirical bioconcentration factor ($Br_{AG\text{-}produce\text{-}DW}$) that relates the chemical concentration in the plant to the average chemical concentration in the soil at the root-zone depth in the produce-growing area ($Cs_{root\text{-}zone\_produce}$).

Equation 3-1. Chemical Concentration in Aboveground Produce

$$C_{AG-produce-DW(i)} = Pr_{AG-produce-DW(i)} + Pd_{(i)} + Pv_{(i)}$$

where:

$C_{AG\text{-}produce\text{-}DW(i)}$ = Concentration of chemical in edible portion of aboveground produce type *i*, exposed or protected, on a dry-weight (DW) basis (mg/kg produce DW)

$Pd_{(i)}$ = Chemical concentration in edible portion of aboveground produce type *i* due to deposition of particles (mg/kg produce DW); for *protected* aboveground produce, *Pd* equals zero

$Pr_{AG\text{-}produce\text{-}DW(i)}$ = Chemical concentration in edible portion of aboveground produce type *i*, exposed or protected, due to root uptake from soil at the root-zone depth of the produce growing area (mg/kg produce DW)

$Pv_{(i)}$ = Chemical concentration in edible portion of aboveground produce type *i* due to air-to-plant transfer (µg/g [or mg/kg] produce DW); for *protected* aboveground produce, *Pv* equals zero

Equation 3-2. Chemical Concentration in Aboveground Produce Due to Root Uptake

$$Pr_{AG-produce-DW(i)} = Cs_{\text{root-zone\_produce}} \times Br_{AG-produce-DW(i)}$$

where:

$Pr_{AG\text{-}produce\text{-}DW(i)}$ = Concentration of chemical in edible portion of aboveground produce type *i*, *exposed* or *protected*, due to root uptake from soil at root-zone depth in the produce-growing area, on a dry-weight (DW) basis (mg/kg produce DW)

$Cs_{root\text{-}zone\_produce}$ = Average chemical concentration in soil at root-zone depth in produce-growing area (mg/kg soil DW)

$Br_{AG\text{-}produce\text{-}DW(i)}$ = Chemical-specific plant/soil chemical bioconcentration factor for edible portion of aboveground produce type *i*, *exposed* or *protected* (g soil DW / g produce DW)

Equation 3-3. Chemical Concentration in Aboveground Produce Due to Deposition of Particle-phase Chemical

$$Pd_{(i)} = \frac{1{,}000 \times (Drdp + (Fw \times Drwp)) \times Rp_{(i)} \times (1 - e^{(-kp(i) * Tp(i))})}{Yp_{(i)} \times kp_{(i)}}$$

where:

$Pd_{(i)}$	=	Chemical concentration in aboveground produce type *i* on a dry-weight (DW) basis due to particle deposition (mg/kg produce DW); set equal to zero for *protected* aboveground produce
Drdp	=	Average annual dry deposition of particle-phase chemical (g/m^2-yr)
Fw	=	Fraction of wet deposition that adheres to plant surfaces; 0.2 for anions, 0.6 for cations and most organics (unitless)
Drwp	=	Average annual wet deposition of particle-phase chemical (g/m^2-yr)
$Rp_{(i)}$	=	Interception fraction of the edible portion of plant type *i* (unitless)
$kp_{(i)}$	=	Plant surface loss coefficient for plant type *i* (yr^{-1})
$Tp_{(i)}$	=	Length of exposure to deposition in the field per harvest of the edible portion of plant type *i* (yr)
$Yp_{(i)}$	=	Yield or standing crop biomass of the edible portion of plant type *i* (kg produce DW/m^2)

Note that Equation 3-3 differs from Equation 5-14 in HHRAP, from which it is derived. In HHRAP, Equation 5-14 includes the term *Q* x (*1 – Fv*) to indicate the emissions rate, in g/sec, of chemical from the source and the proportion of the chemical that remains in, or partitions to, the particle-phase in the air. Also in HHRAP, the dry and wet particle phase deposition rates, *Dydp* and *Dywp,* respectively, are normalized to the emission rate and are expressed in units of sec/m^2-yr.

With MIRC, the user inputs both the dry and wet particle-phase deposition rates, *Drdp* and *Drwp*, respectively, in units of g/m^2-yr for a specific location relative to an emissions source. Those deposition rates might be values measured near that location or estimated using a fate and transport model, such as TRIM.FaTE, in conjunction with local meteorological information and emissions rate data. The chemical emissions term used in HHRAP, *Q*, therefore, is not used in MIRC's Equation 3-3. In addition, in MIRC, *Drdp* and *Drwp*, the average annual dry- and wet-particle-phase deposition rates, respectively, are in units of g/m^2-yr. Users of TRIM.FaTE should note that the dry- and wet-particle-deposition rates output from TRIM.FaTE are in units of g/m^2-day; therefore, users must adjust the TRIM.FaTE output values to units of g/m^2-yr (i.e., multiply by 365 days/yr) before inputting values for *Drdp* and *Drwp* into MIRC.

Equation 3-4. Chemical Concentration in Aboveground Produce Due to Air-to-Plant Transfer of Vapor-phase Chemical

$$Pv_{(i)} = \frac{Ca \times Fv \times Bv_{AG(i)} \times VG_{AG(i)}}{\rho_a}$$

where:

$Pv_{(i)}$	=	Concentration of chemical in edible portion of aboveground produce type *i* from air-to-plant transfer of vapor-phase chemical on a dry-weight (DW) basis (µg/g produce DW); set equal to zero for *protected* aboveground produce
Ca	=	Average annual *total* chemical concentration in air (g/m^3)
Fv	=	Fraction of airborne chemical in vapor phase (unitless)
$Bv_{AG(i)}$	=	Air-to-plant biotransfer factor for aboveground produce type *i* for vapor-phase chemical in air ([mg/g produce DW] / [mg/g air], i.e., g air/ g produce DW)
$VG_{AG(i)}$	=	Empirical correction factor for aboveground *exposed* produce type *i* to address possible overestimate of the diffusive transfer of chemical from the outside to the inside of bulky produce, such as fruit (unitless)
ρ_a	=	Density of air (g/m^3)

Note that Equation 3-4 differs from Equation 5-18 in HHRAP, from which it is derived. In HHRAP, Equation 5-18 includes the term *Q* x *Fv* to indicate the emissions rate, in g/sec, of chemical from the source and the fraction of the chemical in vapor phase in the air. HHRAP also includes the parameter *Cyv*, or the *unitized* yearly average air concentration of vapor-phase chemical in units of µg-sec/g-m^3. For MIRC, the user inputs the average annual total air concentration of the chemical, *Ca*, for a specific location relative to the source in units of g/m^3; MIRC includes a chemical-specific default value for *Fv* for chemicals included in its database. The air concentration might be a value measured near that location or a value estimated by a fate and transport model such as TRIM.FaTE. Users of TRIM.FaTE should note that the average annual concentration of the total chemical in air (i.e., total of both vapor and particulate phases), *Ca*, output from TRIM.FaTE is in units µg/m^3; therefore, the user must adjust the value to units of g/m^3 (i.e., divide by 1,000 µg/g) before entering it in MIRC.

The calculations of chemical concentration in aboveground produce, ($C_{AG\text{-}produce\text{-}DW}$), shown above, are on a dry-weight (DW) basis. The family FFC food ingestion rates, on the other hand, are on a fresh- or wet-weight (WW) basis. MIRC therefore calculates the concentration in aboveground produce on a wet-weight basis, $C_{AG\text{-}produce\text{-}WW}$, using Equation 3-5 and the moisture content (*MAF*) of the FFC food category.

Equation 3-5. Conversion of Aboveground Produce Chemical Concentration from Dry- to Wet-Weight Basis

$$C_{AG-produce-WW(i)} = C_{AG-produce-DW(i)} \times \left(\frac{(100 - MAF_{(i)})}{100} \right)$$

where:

$C_{AG\text{-}produce\text{-}WW(i)}$ = Chemical concentration in edible portion of aboveground produce type *i* on a wet-weight (WW) basis (mg/kg produce WW)

$C_{AG\text{-}produce\text{-}DW(i)}$ = Chemical concentration in edible portion of aboveground produce type *i* on a dry-weight (DW) basis (mg/kg produce DW)

$MAF_{(i)}$ = Moisture adjustment factor for aboveground produce type *i* to convert the chemical concentration estimated for dry-weight produce to the corresponding chemical concentration for full-weight fresh produce (percent water)

3.1.1.1 Belowground Produce

The equations by which chemical concentrations are estimated in belowground produce are different for nonionic organic chemicals than for inorganic chemicals and ionic organic chemicals.

Nonionic Organic Chemicals

For belowground produce and for nonionic organic chemicals, the concentration in the tuber or root vegetable derived from exposure to the chemical in soil is estimated using an empirical root concentration factor (*RCF*) and the average chemical concentration in the soil at the root-zone depth in the produce-growing area ($Cs_{root\text{-}zone\_produce}$), as shown in Equation 3-6. The RCF relates the chemical concentration in the plant on a wet-weight basis to the average chemical concentration in the root-zone soil ($Cs_{root\text{-}zone\_produce}$) on a dry-weight basis. Belowground produce (i.e., tubers or root vegetables) are protected from the deposition and vapor transfer by being covered by soil. Therefore, root uptake of chemicals is the primary mechanism through which belowground produce becomes contaminated.

Equation 3-6. Chemical Concentration in Belowground Produce: Nonionic Organic Chemicals

$$C_{\text{BG-produce-WW}} = \frac{Cs_{root-zone\_produce} \times RCF \times VG_{rootveg}}{Kds \times UCF}$$

where:

$C_{BG\text{-}produce\text{-}WW}$	=	Concentration of chemical in belowground (BG) produce (i.e., tuber or root vegetable) on a wet-weight (WW) basis (mg chemical/kg produce WW) *
$Cs_{root\text{-}zone\_produce}$	=	Average chemical concentration in soil at root-zone depth in produce-growing area, on a dry-weight (DW) basis (mg chemical/kg soil DW)
RCF	=	Chemical-specific root concentration factor for tubers and root produce (L soil pore water/kg root WW) *
$VG_{rootveg}$	=	Empirical correction factor for belowground produce (i.e., tuber or root vegetable) to account for possible overestimate of the diffusive transfer of chemicals from the outside to the inside of bulky tubers or roots (based on carrots and potatoes) (unitless) *
Kds	=	Chemical-specific soil/water partition coefficient (L soil pore water/kg soil DW)
UCF	=	Units conversion factor of 1 kg/L

* Note that there is only one type of BG produce; hence there are no plant-type-specific subscripts

The RCF, as developed by Briggs et al. (1982), is the ratio of the chemical concentration in the edible root on a wet-weight basis to its concentration in the soil pore water. RCFs are based on experiments with growth solutions (hydroponic) instead of soils; therefore, it is necessary to divide the soil concentration by the chemical-specific soil/water partition coefficient (*Kds*). There is no conversion of chemical concentrations in belowground produce from DW to WW because the values are already on a WW basis.

For nonionic organic chemicals, it is possible to predict RCF values and *Kds* values (for a specified soil organic carbon content) from an estimate of the chemical's Kow from empirically derived regression models. Those models are shown in HHRAP Appendix A-2, Equations A-2-14 and A-2-15 (*RCF*) and in Equations A-29 and A-2-10 (*Kds*). The RCF and Kds values so calculated for many of the chemicals in HHRAP are included in the MIRC database (including the values for PAHs and dioxins).

Inorganic Chemicals

For inorganic chemicals and ionized organic chemicals, it is not possible to predict *RCF* or *Kds* values from Kow. For inorganic chemicals, one must use empirical values for the root/soil bioconcentration factor measured for specific chemicals. The root/soil bioconcentration factor, now specified as $Br_{BG\text{-}produce\text{-}DW}$, must be obtained from the literature for each inorganic chemical on a DW basis. For inorganic chemicals, therefore, Equation 3-7 is used instead of Equation 3-6.

Equation 3-7. Chemical Concentration in Belowground Produce: Inorganic Chemicals

$$C_{BG\text{-}produce\text{-}DW} = \frac{Cs_{root-zone\_produce} \times Br_{BG-produce-DW} \times VG_{rootveg}}{1}$$

where:

$C_{BG\text{-}produce\text{-}DW}$ = Concentration of chemical in edible portion of aboveground produce, due to root uptake from soil at root-zone depth in the produce-growing area, on a dry-weight (DW) basis (mg/kg produce DW)

$Cs_{root\text{-}zone\_produce}$ = Average chemical concentration in soil at root-zone depth in produce-growing area (mg/kg soil DW)

$Br_{BG\text{-}produce\text{-}DW}$ = Chemical-specific root/soil chemical bioconcentration factor for edible portion of belowground produce (g soil DW / g produce DW)

$VG_{rootveg}$ = Empirical correction factor for belowground produce (as in Equation 3-6) (unitless)

As for the aboveground produce, the DW estimate of concentration of chemical in the root vegetables must be transformed to a WW estimate, as shown in Equation 3-8.

Equation 3-8. Conversion of Belowground Produce Chemical Concentration from Dry- to Wet-Weight Basis

$$C_{BG-produce-WW} = C_{BG-produce-DW} \times \left(\frac{(100 - MAF_{BG})}{100} \right)$$

where:

$C_{BG\text{-}produce\text{-}WW}$ = Chemical concentration in edible portion of belowground produce on a weight-weight (WW) basis (mg/kg produce WW)

$C_{BG\text{-}produce\text{-}DW}$ = Concentration of chemical in edible portion of aboveground produce, due to root uptake from soil at root-zone depth in the produce-growing area, on a dry-weight (DW) basis (mg/kg produce DW)

$MAF_{(BG)}$ = Moisture adjustment factor (as in Equation 3-5, but single value for below ground produce) (percent water)

3.1.2 Estimating Chemical Concentrations in Animal Products

Chemical concentrations in animal products are estimated based on the amount of chemical consumed by each animal group *m* through each plant feed type *i* ($Plant_{Ch\text{-}Intake(i,m)}$) and incidental ingestion of soil for ground-foraging animals ($Soil_{Ch\text{-}Intake(m)}$). Exhibit 3-3 summarizes the pathways by which chemicals are transferred to these home- or farm-raised animal food products. Note that for a general screening-level assessment, all of the pathways can be modeled, as is done for EPA's RTR calculation of *de minimis* emission rates for PB-HAPs (EPA 2008b).

The feed options for farm animals in MIRC include forage (plants grown on-site for animal grazing, such as grass), silage (wet forage grasses, fresh-cut hay, or other fresh plant material that has been stored and fermented), and grain products grown on the farm. As seen in Exhibit 3-3, the algorithms in MIRC for chemical intake with plant feeds ($Plant_{Ch\text{-}Intake(i,m)}$) are based on the assumptions that beef and dairy cattle consume all three plant feed products, while pigs consume only silage and grain, and chickens consume only grain.

Exhibit 3-3. Chemical Transfer Pathways for Animal Products

Farm Food Media		Chemical Transfer Pathways
Animal Products	Beef and total dairy (including milk)	• Ingestion of forage, silage, and grain [a] • Incidental soil ingestion
	Pork	• Ingestion of silage and grain [a] • Incidental soil ingestion
	Poultry and eggs	• Ingestion of grain [a] • Incidental soil ingestion

[a] Chemical concentrations in plant feed (i.e., forage, silage, and grain) are estimated via intermediate calculations (see Equations 3-13, 3-14, 3-3, and 3-4).

Forage and silage are exposed to the air and can accumulate chemical via direct deposition of particle-bound chemical and transfer of vapor-phase chemical, while all animal feed grains are assumed to be protected from the air by a husk or pod (e.g., corn, soybeans). All three animal feed products are assumed to accumulate chemical via root uptake.

Chemical concentrations are estimated for animal feeds using algorithms analogous to those for aboveground farm produce described above. MIRC uses Equation 3-9 to calculate the concentration of chemical in beef, pork, or total dairy and Equation 3-10 to calculate the concentration of chemical in poultry or eggs. The chemical concentration in mammalian farm animals (i.e., beef and pigs) is adjusted using a metabolism factor (*MF*) that accounts for endogenous degradation of the chemical (see Equation 3-9). *MF* is set to 1.0 for chemicals that are not metabolized and for chemicals for which the metabolic degradation rate is unknown. Although other vertebrates, including birds, are likely to have similar metabolic pathways for most chemicals, the conservative assumption is that birds do not metabolize any chemicals; therefore, the *MF* is omitted from Equation 3-10 for poultry and eggs.

Equation 3-9. Chemical Concentration in Beef, Pork, or Total Dairy

$$C_{mammal(m)} = Ba_{(m)} \times MF \times \left(Soil_{Ch-Intake(m)} + \sum_{i=1}^{n} Plant_{Ch-Intake(i,m)} \right)$$

where:

$C_{mammal(m)}$ = Concentration of chemical in mammalian animal product *m*, where *m* = beef, pork, or total dairy (mg chemical/kg animal product WW)

$Ba_{(m)}$ = Chemical-specific biotransfer factor for chemical in diet to chemical in animal food product *m*, where *m* = beef, pork, or total dairy ([mg chemical/kg animal product WW] / [mg chemical intake/day] or day/kg WW)

MF = Chemical-specific mammalian metabolism factor that accounts for endogenous degradation of the chemical (unitless)

$Soil_{Ch-Intake(m)}$ = Incidental ingestion of chemical in surface soils by livestock type *m* during grazing or consumption of foods placed on the ground (mg/day); see Equation 3-11 below

$Plant_{Ch-Intake(i,m)}$ = For livestock (animal product) type *m*, ingestion of chemical from plant feed type *i* (mg chemical/kg livestock WW); see Equation 3-12 below

(If *m* = beef or total dairy, then *n* = 3 and *i* = forage, silage, and grain;
m = pork, then *n* = 2 and *i* = silage and grain;
m = poultry, then *n* = 1 and *I* = grain.)

Equation 3-10. Chemical Concentration in Poultry or Eggs

$$C_{poultry(m)} = Ba_{(m)} \times \left(Soil_{Ch-Intake(m)} + Plant_{Ch-Intake(i,m)}\right)$$

where:

$C_{poultry(m)}$	=	Concentration of chemical in food product *m*, where *m* = poultry or eggs (mg chemical/kg animal product WW)
$Ba_{(m)}$	=	Chemical-specific biotransfer factor for food product *m*, where *m* = poultry or eggs (day/kg animal product WW)
$Soil_{Ch-Intake(m)}$	=	Incidental ingestion of chemical in surface soils by consumption of food on the ground (mg chemical/day) where *m* = poultry; see Equation 3-11
$Plant_{Ch-Intake(i,m)}$	=	For poultry (and eggs), animal *m*, ingestion of the chemical in plant feed type *i* (mg chemical/day), which for poultry is limited to grain; see Equation 3-12

In MIRC, the incidental ingestion of the chemical in soils by livestock during grazing or consumption of feed placed on the ground ($Soil_{Ch-Intake(m)}$) is estimated using empirical soil ingestion rates (*Qs*) and a soil bioavailability factor for livestock (*Bs*), as shown in Equation 3-11. At this time, the default value for *Bs* in MIRC for all chemicals is 1.0 (i.e., the chemical in soil is assumed to be 100 percent bioavailable to the animal). This assumption may be reasonably accurate for the soil surface to which airborne chemical is deposited. MIRC allows the user to enter a surface soil concentration for areas where livestock forage, $Cs_{S-livestock}$, that is distinct from the surface soil concentration input for areas where produce may be grown and where humans might incidentally ingest soils (see Section 6.1).

Equation 3-11. Incidental Ingestion of Chemical in Soil by Livestock

$$Soil_{Ch-Intake(m)} = Qs \times Cs_{s-livestock} \times Bs$$

where:

$Soil_{Ch-Intake(m)}$	=	Incidental ingestion of the chemical in surface soils by livestock type *m* during grazing or consumption of foods placed on the ground (mg chemical/day)
$Qs_{(m)}$	=	Quantity of soil eaten by animal type *m* each day (kg soil DW/day)
$Cs_{S-livestock}$	=	Chemical concentration in surface soil in contaminated area where livestock feed (mg chemical/kg soil DW)
Bs	=	Soil bioavailability factor for livestock (unitless) (assumed to be the same for birds and mammals)

Animal ingestion of the chemical in feed is calculated for each type of livestock based on their assumed diets. For *m* = beef and dairy cattle, chemical intake is estimated for all three feed types: *i* = forage, silage, and grain. For pork, chemical intake is estimated only for silage and grain. The chemical intake for poultry is based on grain consumption only. The intake of chemical with each feed type, *i*, $Plant_{Ch-Intake(i,m)}$, is calculated separately according to Equation 3-12. Note that the animal feed ingestion rates are on a dry-weight (DW) basis; hence, no DW to wet weight (WW) conversion is needed.

Equation 3-12. Ingestion of Chemical in Feed by Livestock

$$Plant_{Ch-Intake(i,m)} = F_{(i,m)} \times Qp_{(i,m)} \times C_{feed(i)}$$

where:

$Plant_{Ch\text{-}Intake(i,m)}$ = Ingestion of chemical in plant feed type *i* (mg chemical/day), where *i* = forage, silage, or grain, for livestock type *m*

$F_{(i,m)}$ = Fraction of plant feed type *i* obtained from contaminated area used to grow animal feed, where *I* = forage, silage, or grain (unitless) for lifestock type *m*

$Qp_{(i,m)}$ = Quantity of plant feed type *i* consumed per animal per day (kg plant feed DW/day), where *i* = forage, silage, or grain, for livestock type *m*

$C_{feed(i)}$ = Concentration of chemical in ingested plant feed type *i* (mg chemical/kg plant feed DW), where *i* = forage, silage, or grain

The concentrations of chemical in the three different types of plant feeds for livestock are calculated according to Equation 3-13. The equation is the same as that for aboveground produce in Equation 3-1, with the exception that the concentrations are for plants used as animal feeds (not produce consumed by humans) and all types of plant feed (i.e., forage, silage, and grain) are aboveground.

Equation 3-13. Chemical Concentration in Lifestock Feed (All Aboveground)

$$C_{feed(i)} = \mathrm{P}r_{feed(i)} + \mathrm{Pd}_{(i)} + \mathrm{Pv}_{(i)}$$

where:

$C_{feed(i)}$ = Concentration of chemical in plant feed type *i* on a dry-weight (DW) basis (mg chemical/kg plant feed DW), where *i* = forage, silage, or grain

$Pr_{feed(i)}$ = Concentration of chemical in plant feed type *i* due to root uptake from soil (mg/kg DW), where *i* = forage, silage, or grain; see Equation 3-14 below

$Pd_{(i)}$ = Concentration of chemical in plant feed type *i* due to wet and dry deposition of particle-phase chemical (mg/kg DW), where *i* = forage, silage, or grain; when *i* = grain, the *Pd* term equals zero

$Pv_{(i)}$ = Concentration of chemical in plant feed type *i* due to air-to-plant transfer of vapor-phase chemical (µg/g [or mg/kg] DW) where *i* = forage, silage, or grain; when *i* = grain, the *Pd* term equals zero

MIRC calculates the chemical concentration in animal feed due to root uptake from the soil using Equation 3-14. The equation is the same as Equation 3-2, except that a *Br* value appropriate to grasses is used and MIRC allows for different soil concentrations in the area used to grow animal feed than in the area used to grow produce for human consumption (see Section 6.1, user inputs). Note that for feed type *i* = grains, the *Pd* and *Pv* terms do not apply (are set to zero), because the feed products (i.e., corn kernels, soy beans) are protected from the air (i.e., by husks, pods).

Equation 3-14. Chemical Concentration in Livestock Feed Due to Root Uptake

$$Pr_{feed(i)} = Cs_{root-zone\_feed(i)} \times Br_{feed(i)}$$

where:

$Pr_{feed(i)}$ = Concentration of chemical in plant feed type *i* due to root uptake from soil on a dry-weight (DW) basis (mg chemical/kg plant feed DW), where *i* = forage, silage, or grain

$Cs_{root-zone\_feed(i)}$ = Average chemical concentration in soil at root-zone depth in area used to grow plant feed type *i* (mg chemical/kg soil DW), where *i* = forage, silage, or grain

$Br_{feed(i)}$ = Chemical-specific plant-soil bioconcentration factor for plant feed type *i* (kg soil DW/kg plant feed DW), where *i* = forage, silage, or grain

The algorithms used to calculate $Pd_{(i)}$ and $Pv_{(i)}$ when plant feed type *i* = forage and silage are identical to those used to calculate $Pd_{(i)}$ and $Pv_{(i)}$ for aboveground exposed produce (i.e., Equations 3-3 and 3-4, respectively).

There are no conversions of DW feed to WW feed, because all feed ingestion rates for livestock are based on DW feed.

3.2 Chemical Intake Calculations for Adults and Non-Infant Children

MIRC calculates human chemical intake rates from the ingestion of home-grown foods as average daily doses (ADDs) normalized to body weight for each age group, chemical, and food type separately. ADDs, calculated using Equation 3-15, are expressed in milligrams of chemical per kilogram of receptor body weight per day (mg/kg-day).

Equation 3-15. Average Daily Dose for Specified Age Group and Food Type

$$ADD_{(y,i)} = \left(\frac{C_{(i)} \times IR_{(y,i)} \times FC_{(i)} \times ED_{(y)}}{BW_{(y)} \times AT_{(y)}} \right) \left(\frac{EF_{(y)}}{365 \text{ days}} \right)$$

where:

$ADD_{(y,i)}$ = Average daily dose for age group *y* from food type or ingestion medium *i* (mg chemical/kg body weight-day)

$C_{(i)}$ = Concentration of chemical in food type *i* harvested from the contaminated area (mg chemical/kg food or mg food/L water)

$IR_{(y,i)}$ = Ingestion rate for age group *y* of food type *i* (kg/day or L/day)

$FC_{(i)}$ = Fraction of food type *i* that was harvested from contaminated area (unitless)

$ED_{(y)}$ = Exposure duration for age group *y* (years)

$BW_{(y)}$ = Body weight for age group *y* (kg)

$AT_{(y)}$ = Averaging time for calculation of daily dose (years) for age group *y*, set equal to ED in MIRC

$EF_{(y)}$ = Annual exposure frequency for age group *y* (days)

Equation 3-15 takes into account the chemical concentration in each food type *i* (or in water), the quantity of food brought into the home for consumption, the loss of some of the mass of the foods due to preparation and cooking, how much of the food is consumed per year, the amount

of the food obtained from contaminated areas, and the consumer's body weight (EPA 1997a, 2003a). In MIRC, ADDs are calculated separately for each chemical, home-grown food type, and consumer age group.

ADD values, expressed as intakes, not absorbed doses, are appropriate for comparison with RfDs and for use with cancer slope factors (CSFs) to estimate risk, as discussed in Section 5. An exception is for the breast-milk exposure pathway, where the dose absorbed by the mother is relevant to calculating the dose available to and absorbed by her nursing infant, as discussed in Section 3.4.

MIRC evaluates only one contaminated area (set of environmental concentrations), or exposure scenario, at a time. For screening level assessments, all components of this equation are assumed to remain constant for consumers in a given age group over time (e.g., seasonal and annual variations in diet are not explicitly taken into account). To calculate an $ADD_{(y,i)}$ from the contaminated area for food group *i* over an entire lifetime of exposure, age-group-specific ingestion rates and body weights are used for the age groups described in Section 2.3. In MIRC, the averaging time used to calculate the daily dose for an age group (AT_y) is equal to the exposure duration for that group (ED_y); therefore these variables drop out of Equation 3-15.

For each chemical included in a screening scenario, total average daily exposure for age group *y* ($ADD_{(y)}$) is estimated as the sum of chemical intake from all ingestion pathways combined:

- Incidental soil ingestion;
- Ingestion of fish;
- Ingestion of homegrown fruits (exposed and protected);
- Ingestion of homegrown vegetables (exposed, protected, and root);
- Ingestion of animal products from home-raised animals:
    - Milk and other dairy products from cows,
    - Beef products,
    - Pork products, and
    - Poultry and eggs;
- Ingestion of drinking water from specified source; and
- Ingestion of breast milk by infants.

Note that the last exposure pathway is limited to infants.

The algorithms for the first six exposure pathways listed above are described in Sections 3.2.1 through 3.2.6. The algorithms for the breast-milk ingestion pathway are described in Section 3.4.

3.2.1 Chemical Intake from Soil Ingestion

Equation 3-16 shows the equation used to estimate chemical intake through incidental ingestion of soil.

Equation 3-16. Chemical Intake from Soil Ingestion

$$ADD_{Soil\,(y)} = \left(\frac{C_{Soil} \times IR_{Soil(y)} \times FC_{Soil} \times 0.001 \ \frac{mg}{\mu g}}{BW_{(y)}} \right) \left(\frac{EF}{365 \text{ days}} \right)$$

where:

$ADD_{Soil(y)}$ = Average daily chemical intake from incidental ingestion of soil or ingestion by child in age group *y* (mg chemical/kg body weight-day)

C_{Soil} = Concentration of chemical in soil from contaminated area on a dry-weight (DW) basis (µg/g soil DW)

$IR_{Soil(y)}$ = Soil ingestion rate for age group *y* (g DW/day)

FC_{Soil} = Fraction of soil ingested that is from contaminated area (unitless)

$BW_{(y)}$ = Body weight for age group *y* (kg)

EF = Exposure frequency; number of days per year of exposure for family(ies) as specified for scenario (≤ 365 days)

Note: MIRC saves soil ingestion rates in units of mg/day (not g/day); therefore, there is an additional 0.001 g/mg conversion unit in the actual MIRC algorithm than shown here.

3.2.2 Chemical Intake from Fish Ingestion

Ingestion of locally caught fish is included as a possible exposure pathway in MIRC (Equation 3-17). Two types of fish are included in the exposure algorithm: trophic level 3 (T3) fish, equivalent to small “pan” fish such as bluegill, and trophic level 4 (T4) fish, equivalent to game fish such as trout and walleye. The chemical concentration in fish in Equation 3-17 is estimated as the consumption-weighted chemical concentration using Equation 3-18.

Equation 3-17. Chemical Intake from Fish Ingestion

$$ADD_{Fish(y)} = (1 - L1_{Fish}) \times (1 - L2_{Fish}) \times \frac{\left(C_{Fish} \times IR_{Fish(y)} \times 0.001 \frac{kg}{g} \times FC_{Fish} \right)}{BW_{(y)}} \times \left(\frac{EF}{365 \text{ days}} \right)$$

Equation 3-18. Consumption-weighted Chemical Concentration in Fish

$$C_{Fish} = (C_{FishT3} \times F_{T3}) + (C_{FishT4} \times F_{T4})$$

where:

$ADD_{Fish(y)}$	=	Average daily chemical intake from ingestion of local fish for age group *y* (mg/kg-day)
$L1_{Fish}$*	=	Weight of fish brought into home that is discarded during preparation (e.g., head, bones, liver, other viscera, belly fat, skin with fat) (unitless)
$L2_{Fish}$*	=	Loss of weight during cooking, such as evaporation and loss of fluids into pan (unitless)
C_{FishT3}	=	Chemical concentration in whole fish for trophic level 3 (T3) fish on a wet-weight (WW) basis (mg/kg WW)
C_{FishT4}	=	Chemical concentration in whole fish for trophic level 4 (T4) fish on a wet-weight (WW) basis (mg/kg WW)
F_{T3}	=	Fraction of fish intake that is from T3 (unitless)
F_{T4}	=	Fraction of fish intake that is from T4 (unitless)
C_{Fish}	=	Consumption-weighted mean chemical concentration in total fish (i.e., as specified by Equation 3-18) (mg/kg WW)
FC_{Fish}	=	Fraction of local fish consumed derived from contaminated area (unitless)
$BW_{(y)}$	=	Body weight for age *y* (kg)
$IR_{Fish(y)}$*	=	Local fish ingestion rate for age *y* (g WW/day)
EF	=	Exposure frequency; number of days per year of exposure for family(ies) as specified for scenario (≤ 365 days)

* Parameter values must be internally consistent. In contrast to the tables included in MIRC for ingestion rate options for homegrown food products, which are based on the products as brought into the home from the field (see Section 6.3.3), the tables of fish ingestion rate options included in MIRC are from CSFII data (see Section 6.3.4) and, therefore, are on an "as consumed" basis (i.e., after preparation and cooking losses), and L1 and L2 therefore are set equal to zero. If the user wishes to enter local fish ingestion rates on an "as harvested" basis, the user also should enter L1 and L2 values as specified in Section 6.4.3.

When whole fish are prepared for cooking, it is usual for the viscera, head, and fins to be removed, particularly for larger fish. Many persons also remove (or do not eat) the skin, bones, and belly fat. EPA has, therefore, estimated the proportion of the weight of whole fish that tends to be lost during preparation and cooking across a variety of fish species (Exposure Factors Handbook Table 13-5, EPA 1997a) and included those losses in its HHRAP algorithms for chemical intake from fish ($L1_{Fish}$ and $L2_{Fish}$ in Equation 3-17).

3.2.3 Chemical Intake from Fruit Ingestion

Average daily doses of a chemical from homegrown exposed fruits are calculated separately for exposed and protected fruits (Equations 3-19 and 3-20, respectively).

Equation 3-19. Chemical Intake from Consumption of Exposed Fruits

$$ADD_{ExpFruit(y)} = \left(1 - L1_{ExpFruit}\right) \times \left(1 - L2_{ExpFruit}\right) \times \left(C_{ExpFruit} \times IR_{ExpFruit(y)} \times 0.001 \frac{\text{kg}}{\text{g}} \times FC_{ExpFruit} \right) \times \left(\frac{EF}{365 \text{ days}} \right)$$

Equation 3-20. Chemical Intake from Consumption of Protected Fruits

$$ADD_{ProFruit(y)} = \left(1 - L1_{ProFruit}\right) \times \left(C_{ProFruit} \times IR_{ProFruit(y)} \times 0.001 \frac{\text{kg}}{\text{g}} \times FC_{ProFruit} \right) \times \left(\frac{EF}{365 \text{ days}} \right)$$

where:

$ADD_{ExpFruit(y)}$ $ADD_{ProFruit(y)}$	=	Average daily chemical intake from ingestion of exposed fruit or protected fruit (depending on subscript) (mg chemical/kg body weight-day)
$L1_{ExpFruit}$	=	Mean reduction in fruit weight resulting from removal of skin or peel, core or pit, stems or caps, seeds and defects, and from draining liquids from canned or frozen forms (unitless)
$L1_{ProFruit}$	=	Mean reduction in fruit weight that results from paring or other preparation techniques for protected fruits (unitless)
$L2_{ExpFruit}$	=	Mean reduction in fruit weight that results from draining liquids from cooked forms of the fruit (unitless)
$C_{ExpFruit}$ $C_{ProFruit}$	=	Chemical concentration in whole exposed fruits or whole protected fruits (depending on subscript) on a wet-weight (WW) basis (mg chemical/kg exposed fruit WW)
EF	=	Exposure frequency; number of days per year of exposure for family(ies) as specified for scenario (≤ 365 days)
$FC_{ExpFruit}$ $FC_{ProFruit}$	=	Fraction of exposed fruits or protected fruits (depending on subscript) obtained from contaminated area (unitless)
$IR_{ExpFruit(y)}$ $IR_{ProFruit(y)}$	=	Ingestion rate of home-grown exposed fruits or protected fruits (depending on subscript) for age *y* (g WW/kg body weight-day)

Fruit ingestion rates in the survey were based on weights of unprepared fruits (e.g., one apple; one pear) or the weight of a can of fruit (e.g., 8 oz can). The weight of the fruit ingested is less than the initial weight owing to common preparation actions ($L1_{ExpFruit}$ and $L1_{ProFruit}$; e.g., coring apples and pears; peeling apples; pitting cherries). Cooking of exposed fruit (e.g., berries, apples, peaches) often results in further weight loss that results from liquids lost during cooking and drained from the cooking vessel ($L2_{ExpFruit}$). EPA has assumed that cooking of protected fruit results in no loss of weight for the fruit.

3.2.4 Chemical Intake from Vegetable Ingestion

MIRC includes three separate algorithms for homegrown vegetables adapted from EPA's HHRAP Modeling System (EPA 2005a): one for exposed vegetables such as asparagus, broccoli, lettuce, and tomatoes (although they are actually a fruit); one for protected vegetables such as corn, cabbage, soybeans, and peas; and one for root vegetables such as carrots, beets, and potatoes (see Equations 3-21, 3-22, and 3-23, respectively).

Equation 3-21. Chemical Intake from Exposed Vegetables

$$ADD_{ExpVeg(y)} = \left(1 - L1_{ExpVeg}\right) \times \left(C_{ExpVeg} \times IR_{ExpVeg(y)} \times 0.001 \frac{\text{kg}}{\text{g}} \times FC_{ExpVeg} \right) \times \left(\frac{EF}{365 \text{ days}} \right)$$

Equation 3-22. Chemical Intake from Protected Vegetables

$$ADD_{ProVeg(y)} = \left(1 - L1_{ProVeg}\right) \times \left(C_{ProVeg} \times IR_{ProVeg(y)} \times 0.001 \frac{\text{kg}}{\text{g}} \times FC_{ProVeg} \right) \times \left(\frac{EF}{365 \text{ days}} \right)$$

Equation 3-23. Chemical Intake from Root Vegetables

$$ADD_{RootVeg(y)} = \left(1 - L1_{RootVeg}\right) \times \left(1 - L2_{RootVeg}\right) \times \left(C_{RootVeg} \times IR_{RootVeg(y)} \times 0.001 \frac{\text{kg}}{\text{g}} \times FC_{RootVeg} \right) \times \left(\frac{EF}{365 \text{ days}} \right)$$

where:

$ADD_{ExpVeg(y)}$ $ADD_{ProVeg(y)}$ $ADD_{RootVeg(y)}$	=	Average chemical intake from ingestion of exposed vegetables, protected vegetables, or root vegetables (depending on subscript) for age group *y* (mg chemical/kg body weight-day)
$L1_{ExpVeg}$	=	Mean net preparation and cooking weight loss for exposed vegetables (unitless); includes removing stalks, paring skins, discarding damaged leaves
$L1_{ProVeg}$	=	Mean net cooking weight loss for protected vegetables (unitless); includes removing husks, discarding pods of beans and peas, removal of outer leaves
$L1_{RootVeg}$	=	Mean net cooking weight loss for root vegetables (unitless); includes losses from removal of tops and paring skins
$L2_{RootVeg}$	=	Mean net post cooking weight loss for root vegetables from draining cooking liquids and removal of skin after cooking (unitless)
C_{ExpVeg} C_{ProVeg} $C_{RootVeg}$	=	Chemical concentration in exposed vegetables, protected vegetables, or root vegetables (depending on subscript) on a wet-weight (WW) basis (mg chemical/kg vegetable WW)
EF	=	Exposure frequency; number of days per year of exposure for family(ies) as specified for scenario ($\leq$ 365 days)
FC_{ExpVeg} FC_{ProVeg} $FC_{RootVeg}$	=	Fraction of exposed vegetables, protected vegetables, or root vegetables (depending on subscript) obtained from contaminated area (unitless)
$IR_{ExpVeg(y)}$ $IR_{ProVeg(y)}$ $IR_{RootVeg(y)}$	=	Ingestion rate of exposed vegetables, protected vegetables, or root vegetables (depending on subscript) for age group *y* (g vegetable WW/kg body weight-day)

3.2.5 Chemical Intake from Animal Product Ingestion

Calculations of chemical intake from the consumption of farm animals and related food products are provided below in Equations 3-24 through 3-28 for homegrown beef, dairy (milk), pork, poultry, and eggs, respectively.

Equation 3-24. Chemical Intake from Ingestion of Beef

$$ADD_{Beef(y)} = (1 - L1_{Beef}) \times (1 - L2_{Beef}) \times \left(C_{Beef} \times IR_{Beef(y)} \times 0.001 \frac{\text{kg}}{\text{g}} \times FC_{Beef} \right) \times \left(\frac{EF}{365 \text{ days}} \right)$$

where:

$ADD_{Beef(y)}$	=	Average daily chemical intake from ingestion of beef for age group y (mg/kg-day)
$L1_{Beef}$	=	Mean net cooking loss for beef (unitless)
$L2_{Beef}$	=	Mean net post cooking loss for beef (unitless)
C_{Beef}	=	Concentration of contaminant in beef (mg/kg WW))
EF	=	Exposure frequency; number of days per year of exposure for family(ies) as specified for scenario (≤ 365 days)
$IR_{Beef(y)}$	=	Ingestion rate of contaminated beef for age group y (g WW/kg-day)
FC_{Beef}	=	Fraction of beef consumed raised on contaminated area or fed contaminated silage and grains (unitless)

Equation 3-25. Chemical Intake from Dairy Ingestion

$$ADD_{Dairy(y)} = \left(C_{Dairy} \times IR_{Dairy(y)} \times 0.001 \frac{\text{kg}}{\text{g}} \times FC_{Dairy} \right) \times \left(\frac{EF}{365 \text{ days}} \right)$$

where:

$ADD_{Dairy(y)}$	=	Average daily chemical intake from ingestion of total dairy for age group *y* (mg/kg-day)
C_{Dairy}	=	Average concentration of contaminant in total dairy (mg/kg WW)
EF	=	Exposure frequency; number of days per year of exposure for family(ies) as specified for scenario (≤ 365 days)
$IR_{Dairy(y)}$	=	Ingestion rate of contaminated total dairy for age group *y* (g WW/kg-day)
FC_{Dairy}	=	Fraction of total dairy products from contaminated area (unitless)

Equation 3-26. Chemical Intake from Pork Ingestion

$$ADD_{Pork(y)} = (1 - L1_{Pork}) \times (1 - L2_{Pork}) \times \left(C_{Pork} \times IR_{Pork(y)} \times 0.001 \frac{\text{kg}}{\text{g}} \times FC_{Pork} \right) \times \left(\frac{EF}{365 \text{ days}} \right)$$

where:

$ADD_{Pork(y)}$	=	Average daily chemical intake from ingestion of pork for age group *y* (mg/kg-day)
$L1_{Pork}$	=	Mean net cooking loss for pork (unitless); includes dripping and volatile losses during cooking; averaged over various cuts and preparation methods
$L2_{Pork}$	=	Mean net post cooking loss for pork (unitless); includes losses from cutting, shrinkage, excess fat, bones, scraps, and juices; averaged over various cuts and preparation methods
C_{Pork}	=	Concentration of contaminant in pork (mg/kg WW)
EF	=	Exposure frequency; number of days per year of exposure for family(ies) as specified for scenario (≤ 365 days)
$IR_{Pork(y)}$	=	Ingestion rate of contaminated pork for age *y* (g WW/kg-day)
FC_{Pork}	=	Fraction of pork obtained from contaminated area (unitless)

The reduction in the weight of pork during and after cooking may correlate with an increase or decrease in the concentration of the chemical in the pork as consumed depending on the chemical and depending on the cooking method.

Equation 3-27. Chemical Intake from Poultry Ingestion

$$ADD_{Poultry(y)} = (1 - L1_{Poultry}) \times (1 - L2_{Poultry}) \times \left(C_{Poultry} \times IR_{Poultry(y)} \times 0.001 \frac{\text{kg}}{\text{g}} \times FC_{Poultry} \right) \times \left(\frac{EF}{365 \text{ days}} \right)$$

where:

$ADD_{Poultry(y)}$	=	Average daily dose (chemical intake) from ingestion of poultry (mg/kg-day)
$L1_{Poultry}$	=	Mean net cooking loss for poultry (unitless)
$L2_{Poultry}$	=	Mean net post cooking loss for poultry (unitless)
$C_{Poultry}$	=	Concentration of chemical in poultry (mg/kg WW)
EF	=	Exposure frequency; number of days per year of exposure for family(ies) as specified for scenario (≤ 365 days)
$IR_{Poultry(y)}$	=	Ingestion rate of poultry for age group *y* (g WW/kg-day)
$FC_{Poultry}$	=	Fraction of poultry from contaminated area or fed contaminated grains (unitless)

Equation 3-28. Chemical Intake from Egg Ingestion

$$ADD_{Egg(y)} = \left(C_{Egg} \times IR_{Egg(y)} \times 0.001 \frac{\text{kg}}{\text{g}} \times FC_{Egg} \right) \times \left(\frac{EF}{365 \text{ days}} \right)$$

where:

$ADD_{Egg(y)}$	=	Average daily chemical intake from ingestion of eggs for age group *y* (mg/kg-day)
C_{Egg}	=	Concentration of contaminant in eggs (mg/kg WW)
EF	=	Exposure frequency; number of days per year of exposure for family(ies) as specified for scenario (≤ 365 days)
$IR_{Egg(y)}$	=	Ingestion rate of contaminated eggs for age group *y* (g WW/kg-day)
FC_{Egg}	=	Fraction of eggs obtained from contaminated area (unitless)

3.2.6 Chemical Intake from Drinking Water Ingestion

If the user chooses to evaluate chemical ingestion via drinking water, the user specifies a chemical concentration in g/L (equivalent to mg/mL) based on their particular scenario. The chemical concentration could represent water from groundwater wells, community water, nearby surface waters, or other source. For this exposure pathway, ingestion rates are in units of milliliters of water per day (mL/day).

Equation 3-29. Chemical Intake from Drinking Water Ingestion

$$ADD_{DW(y)} = \left(\frac{C_{DW} \times IR_{DW(y)} \times FC_{DW}}{BW_{(y)}} \right) \times \left(\frac{EF}{365 \text{ days}} \right)$$

where:

$ADD_{DW(y)}$	=	Average daily chemical intake from ingestion of drinking water from local residential water source for age group *y* (mg/kg-day)
C_{DW}	=	Concentration of contaminant in drinking water (g/L)
$IR_{DW(y)}$	=	Drinking water ingestion rate for age group *y* (mL/day)
FC_{DW}	=	Fraction of drinking water obtained from contaminated area (unitless)
$BW_{(y)}$	=	Body weight of age group *y* (kg)
EF	=	Exposure frequency; number of days per year of exposure for family(ies) as specified for scenario (≤ 365 days)

3.3 Total Chemical Intake

To estimate the total ADD, or intake of a chemical from all of the exposure media that a single individual in each age group is expected to contact (e.g., soil, local fish, five types of home-grown produce, and five types of home-raised animals or animal products), the media-specific chemical intakes are summed for each age group. Total average daily exposure for a particular age group *y* ($ADD_{(y)}$) is estimated as the sum of chemical intake from all ingestion pathways combined, as illustrated in Equations 3-30 through 3-35 below.

Equations 3-30 to 3-35. Total Average Daily Dose of a Chemical for Different Age Groups

Equation 3-30. $$ADD_{(<1)} = ADD_{breastmilk}$$

Equation 3-31. $$ADD_{(1-2)} = \sum_{i=1}^{n} ADD_{(1-2,i)}$$

Equation 3-32. $$ADD_{(3-5)} = \sum_{i=1}^{n} ADD_{(3-5,i)}$$

Equation 3-33. $$ADD_{(6-11)} = \sum_{i=1}^{n} ADD_{(6-11,i)}$$

Equation 3-34. $$ADD_{(12-19)} = \sum_{i=1}^{n} ADD_{(12-19,i)}$$

Equation 3-35. $$ADD_{(adult)} = \sum_{i=1}^{n} ADD_{(adult,i)}$$

where *i* represents the i^{th} food type or ingestion medium and *n* equals the total number of food types or ingestion media, and *ADD* parameters are defined below:

$ADD_{(<1)}$	=	Total average daily dose of chemical for infants less than one year from ingestion of breast milk (mg/kg-day)
$ADD_{(1-2)}$	=	Total average daily dose of chemical from all ingestion sources for children ages 1 through 2 years (mg/kg-day)
$ADD_{(3-5)}$	=	Total average daily dose for children ages 3 through 5 years (mg/kg-day)
$ADD_{(6-11)}$	=	Total average daily dose for children ages 6 through 11 years (mg/kg-day)
$ADD_{(12-19)}$	=	Total average daily dose for children ages 12 through 19 years (mg/kg-day)
$ADD_{(adult)}$	=	Total average daily dose for adult age 20 up to 70 years (mg/kg-day)

The lifetime average daily dose (*LADD*) is calculated as the time-weight average of the *ADD* values for each age group (Equation 3-36).

Equation 3-36. Lifetime Average Daily Dose (LADD)

$$LADD = ADD_{(<1)}\left(\frac{1}{70}\right) + ADD_{(1-2)}\left(\frac{2}{70}\right) + ADD_{(3-5)}\left(\frac{3}{70}\right) + ADD_{(6-11)}\left(\frac{6}{70}\right) + ADD_{(12-19)}\left(\frac{8}{70}\right) + ADD_{(adult)}\left(\frac{50}{70}\right)$$

The time-weighting factors simply equal the duration of exposure for the specified age category in years divided by the total lifespan, assumed to be 70 years. For risk assessments for chemicals with a subchronic RfD or for developmental effects in children, $ADD_{(y)}$ values for the child age groups are compared with the RfD (see Section 5).

3.4 Chemical Intake Calculations for Nursing Infants

The scientific literature indicates that infants can be exposed to some chemicals via their mothers' breast milk. The magnitude of the exposure can be estimated from information on the mother's exposure, data on the partitioning of the chemical into various compartments of the mother's body and into breast milk, and information on the infant's consumption of milk and absorption of the chemical. To add this exposure pathway to the MIRC application, we adapted exposure algorithms and default assumptions from EPA's *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions* (EPA 1998), hereafter referred to as MPE, as explained below.

Note that this pathway generally is of most concern for *lipophilic* bioaccumulative chemicals (e.g., dioxins) that can cause developmental effects. The period of concern for the more *hydrophilic* chemicals that cause developmental effects generally is earlier, that is, from conception to birth. Hydrophilic chemicals generally exchange well between the maternal and fetal blood supplies at the placenta.

3.4.1 Infant Average Daily Absorbed Dose

The average daily dose of chemical *absorbed* by the infant (DAI_{inf}) is estimated in MIRC with Equation 3-37. This basic exposure equation relies on the concentration of the chemical in the breast milk, the infant's breast-milk ingestion rate (IR_{milk}), the absorption efficiency of the chemical by the oral route of exposure (AE_{inf}), the bodyweight of the infant (BW_{inf}), and the duration of breast feeding (ED). Equation 3-37 is EPA's (EPA 1998) modification of an average daily dose for the infant model first published by Smith (1987) and includes variables for both the concentration of the chemical in the breast milk fat ($C_{milkfat}$) and the concentration of the chemical in the aqueous phase of breast milk ($C_{aqueous}$). The remainder of the DAI_{inf}-associated equations assume that most chemicals of concern will partition *either* to the lipid phase *or* to the aqueous phase of breast milk, although some chemicals may partition significantly to both phases of milk. Thus, the remaining equations in MIRC assume that either $C_{milkfat}$ or $C_{aqueous}$ is equal to zero and hence drops out of the equation.

For the parameters in Equation 3-37 (and the equations that follow) that are not calculated from another equation, an EPA default value and options for other values available in MIRC for the infant breast-milk-exposure pathway are described in Section 6.4. The user also can overwrite those parameter values with a different value from the literature as appropriate.

Equation 3-37. Average Daily Dose of Chemical to the Nursing Infant

$$DAI_{inf} = \frac{\left[\left(C_{milkfat} \times f_{mbm}\right) + \left(C_{aqueous} \times \left(1 - f_{mbm}\right)\right)\right] \times IR_{milk} \times AE_{inf} \times ED}{BW_{inf} \times AT}$$

where:

DAI_{inf}	=	Average daily dose of chemical absorbed by infant (mg chemical/kg body weight-day)
$C_{milkfat}$	=	Concentration of chemical in lipid phase of maternal milk (mg chemical/kg milk lipid; calculated using Equation 3-38)
f_{mbm}	=	Fraction of fat in breast milk (unitless)
$C_{aqueous}$	=	Concentration of chemical in aqueous phase of maternal milk (mg chemical/kg aqueous phase milk; calculated using Equation 3-42)
IR_{milk}	=	Infant milk ingestion rate over the duration of nursing (kg milk/day)
AE_{inf}	=	Absorption efficiency of the chemical by the oral route of exposure (i.e., chemical-specific fraction of ingested chemical that is absorbed by the infant) (unitless)
ED	=	Exposure duration, i.e., duration of breast feeding (days)
BW_{inf}	=	Body weight of infant averaged over the duration of nursing (kg)
AT	=	Averaging time associated with exposure of interest; equal to ED (days)

As mentioned above, Equation 3-37 includes terms for the chemical in both the lipid- and non-lipid phases of milk. The remaining equations, however, assume that a chemical of concern will partition to the lipid or aqueous phase of breast milk. Different models are used to estimate $C_{milkfat}$ (described in Section 3.4.2) and $C_{aqueous}$ (described in Section 3.4.3).

3.4.2 Chemical Concentration in Breast Milk Fat

When developing MPE (EPA 1998), EPA reviewed three first-order kinetics models for estimating chemical concentration in breast milk fat. The model selected for use in MPE is the model used in MIRC. It is a changing-concentration model that EPA adapted from a model by Sullivan et al. (1991). The model, shown in Equation 3-38, estimates the average chemical concentration in the breast milk over the entire period of breast feeding by reference to a maximum theoretical steady-state concentration. Studies of lipophilic chemicals such as dioxins suggest that concentrations in the maternal milk are highest during the first few weeks of breast feeding and then decrease over time (ATSDR 1998). Equation 3-38 accounts for the changing concentration in breast milk fat, but estimates one average value to represent the concentration over the entire duration of breast feeding. The model is dependent on the maternal body burden of the chemical and assumes that the chemical concentration in breast milk fat is the same as the concentration in general maternal body fat. According to reviewers of the model, this assumption warrants further investigation because milk fat appears to be synthesized in the mammary glands and may have lower chemical concentrations than general body fat stores (EPA 2001a).

Equation 3-38. Chemical Concentration in Breast Milk Fat

$$C_{milkfat} = \frac{DAI_{mat} \times f_f}{k_{elim} \times f_{fm}} \times \left[\frac{k_{elim}}{k_{fat\_elac}} + \frac{1}{k_{fat\_elac} \times t_{bf}} \left(1 - e^{-k_{elim} t_{pn}} - \frac{k_{elim}}{k_{fat\_elac}} \right) \left(1 - e^{-k_{fat\_elac} t_{bf}} \right) \right]$$

where:

$C_{milkfat}$ = Concentration of chemical in lipid phase of maternal milk (mg chemical/kg lipid)

DAI_{mat} = Daily absorbed maternal chemical dose (mg chemical/kg maternal body weight-day; calculated using Equation 3-39)

f_f = Fraction of total maternal body burden of chemical that is stored in maternal fat (mg chemical in body fat / mg total chemical in whole body; value from literature or EPA default - see Section 6.5)

k_{elim} = Chemical-specific total elimination rate constant for elimination of the chemical by non-lactating women (per day; e.g., via urine, bile to feces, exhalation; value from literature or calculated using Equation 3-40)

f_{fm} = Fraction of maternal body weight that is fat stores (unitless)

k_{fat_elac} = Chemical-specific rate constant for total elimination of chemical in the lipid phase of milk during nursing (per day; value from literature or calculated using Equation 3-41)

t_{bf} = Duration of breast feeding (days)

t_{pn} = Duration of mother's exposure prior to parturition and initiation of breast feeding (days)

Equation 3-38 relies on the daily maternal absorbed intake (DAI_{mat}) to determine the concentration of the chemical in the breast milk fat. DAI_{mat} is multiplied by the fraction of the chemical that is stored in maternal fat (f_f) to determine the amount (i.e., mass) of chemical in the fat. This product, divided by the chemical-specific elimination rate constant (k_{elim}) for non-lactating adult women and the fraction of the mother's weight that is fat (f_{fm}), represents the maximum theoretical steady-state concentration of the chemical in an adult woman. If used alone to estimate the chemical concentration in breast milk fat, the equation as explained thus far is likely to overestimate the chemical concentration in milk fat because it does not account for losses due to breast feeding. Alone, this term ($DAI_{mat} f_f / k_{elim} f_{fm}$) also assumes that the biological half-life of the chemical in the mother's breast milk fat is small relative to the duration of the mother's exposure. However, for chemicals with half-lives that are longer than the exposure duration, which are the chemicals of concern in the applications of MIRC to date, an additional term is needed to determine the average concentration in the milk fat over the duration of her exposure.

To account for breast feeding losses and longer chemical half-lives in the mother than the exposure duration, an additional term is included in Equation 3-38. This term includes a fraction dependent on two rate constants, k_{elim} and the elimination constant for a lipophilic chemical in lactating women via the lipid phase of breast milk (k_{fat_elac}), the duration of the mother's chemical exposure prior to nursing (t_{pn}), and the duration of breast feeding (t_{bf}). The whole body concentration ($DAI_{mat} f_f / k_{elim} f_{fm}$), the maximum theoretical steady-state concentration, is multiplied by the rate of elimination averaged over the duration of the mother's exposure, including her exposure prior to and during lactation. To review the derivation of Equation 3-38, see Appendix B of MPE (EPA 1998).

To estimate an average daily dose *absorbed* by an infant's mother, or DAI_{mat}, the average daily dose (*ADD*) (in mg/kg-day) for the chemical from all sources that MIRC calculates for adults ($ADD_{(adult)}$, described in Section 3.3, Equation 3-35), is multiplied by an absorption efficiency (AE_{mat}) or fraction of the chemical absorbed by the oral route of exposure, as shown in Equation 3-39. The value for AE_{mat} can be estimated from absorption efficiencies for adults in general. Available data for some chemicals, in particular some inorganic compounds, indicate AE values for ingestion exposures of substantially less than 100 percent. For a few of these chemicals, data also indicate lower AEs for the chemical when ingested in food or in soil than when ingested in water (e.g., cadmium). For a screening level assessment, however, it is reasonable to either assume 100 percent for the AE_{mat} or to use the higher AE_{mat} of the food and water AE_{mat} values if available; hence, a single AE_{mat} parameter is included in Equation 3-39.

Equation 3-39. Daily Maternal Absorbed Intake

$$DAI_{mat} = ADD_{(adult)} \times AE_{mat}$$

where:

DAI_{mat} = Daily maternal dose of chemical absorbed from medium *i* (mg/kg-day)

$ADD_{(adult)}$ = Average daily dose to the mother (mg/kg-day) (calculated by MIRC – see Section 3.3, Equation 3-35)

AE_{mat} = Absorption efficiency of the chemical by the oral route of exposure (i.e., chemical-specific fraction of ingested chemical that is absorbed) by the mother (unitless) (value from literature or EPA default – see Section 6.4)

Equation 3-35, used to calculate $ADD_{(adult)}$, is based on many medium-specific ingestion rates that are normalized to body weight. The adult body weights to which the homegrown food ingestion rates are normalized are the body weights of the consumers in the original USDA survey (see Section 6.3.3), which included both males and females. An assumption in the breast-milk exposure pathway is that those ingestion rates also are applicable to nursing mothers. The original data for ingestion rates for soil, drinking water, and fish are on a per person basis for males and females combined. MIRC divides those chemical intakes by an adult body weight for males and females combined as specified by the user (e.g., 71.4 kg mean value) to estimate the ADD normalized to body weight from those sources. If the user finds that those exposure media contribute the majority of the chemical intake for the risk scenario under consideration, the user may use alternative ingestion rates for those media and alternative body weights for nursing women, as described in Section 6.5.

Elimination rates for chemicals often are reported as the half-life of the chemical in the body following a known dose of chemical. Many chemicals exhibit a two-phase elimination process, the first being more rapid than the second. For screening risks for persistent and bioaccumulative chemicals, the half-life of the slower phase of elimination, presumably from non-blood compartments of the body, is the more important of the two. Assuming first-order kinetics, Equation 3-40 is used to convert a measured half-life for elimination of a chemical for adults or non-lactating women to an elimination rate constant (EPA 1998). The equation can be used to estimate any kind of chemical loss rate constant from a measured chemical half-life.

Equation 3-40. Biological Elimination Rate Constant for Chemicals for Non-lactating Women

$$k_{elim} = \frac{\ln 2}{h}$$

where:

k_{elim} = Chemical-specific elimination rate constant for elimination of the chemical for non-lactating women (per day; e.g., via urine, bile to feces, exhalation)

$ln2$ = Natural log of 2 (unitless constant)

h = Chemical-specific biological half-life of chemical for non-lactating women (days)

For chemicals transferred from the body of lactating women to breast milk, the rate of chemical elimination is augmented by the rate of chemical loss via the milk. The total elimination rate for lactating women sometimes is measured directly and reported in the literature. Where direct measurements are not available, and for chemicals that partition predominantly to the lipid-phase of milk, EPA has used Equation 3-41 to estimate the total chemical elimination rate for lactating women, k_{fat_elac} (EPA 1998).

Equation 3-41. Biological Elimination Constant for Lipophilic Chemicals for Lactating Women

$$k_{fat\_elac} = k_{elim} + \frac{IR_{milk} \times f_f \times f_{mbm}}{f_{fm} \times BW_{mat}}$$

where:

k_{fat_elac} = Rate constant for total elimination of chemical during nursing (per day); accounts for both elimination by adults in general and the additional chemical elimination via the lipid phase of milk in nursing women

k_{elim} = Elimination rate constant for chemical from adults, including non-lactating women (per day; e.g., via urine, bile to feces, exhalation; chemical-specific; value from literature or calculated from half-life using Equation 3-40)

IR_{milk} = Infant milk ingestion rate over the duration of nursing (kg/d)

f_f = Fraction of total maternal body burden of chemical that is stored in maternal fat (mg chemical in body fat / mg chemical total in body; value from literature or EPA default)

f_{mbm} = Fraction of fat in breast milk (unitless)

f_{fm} = Fraction of maternal body weight that is fat stores (unitless)

BW_{mat} = Maternal body weight over the entire duration of the mother's exposure to the chemical including during pregnancy and lactation (kg)

Equation 3-41 is based on a model from Smith (1987) and accounts for the additional elimination pathway for lipophilic chemicals via the breast milk fat. The term K_{fat_elac} is estimated by adding an estimate of the first-order elimination constant for breast feeding losses to k_{elim}, which is the chemical-specific total elimination rate constant for non-lactating women. The breast feeding losses are estimated from the infant's intake rate of breast milk (IR_{milk}), the fraction of the total maternal body burden of the chemical that is stored in maternal body fat (f_f),

the fraction of the mother's breast milk that consists of fat (lipids) (f_{mbm}), the mother's body weight (BW_{mat}), and the fraction of the mother's weight that is body fat (f_{fm}). In Equation 3-41, the value for the mother's body weight should be specific to women of child-bearing age, as opposed to a body weight value for both males and females that is used to estimate an adult average daily dose and the mother's absorbed daily intake in Equation 3-39. Body weight values for the mother are described in Section 6.5. Smith's (1987) model assumes that the chemical partitions to the lipid-phase of breast milk to the same degree that it partitions into the mother's body fat. For highly lipophilic compounds, losses from breast feeding can be larger than losses by all other pathways (EPA 1998).

3.4.3 Chemical Concentration in Aqueous Phase of Breast Milk

When developing MPE (EPA 1998), EPA also considered models to estimate chemical concentrations in the aqueous phase of breast milk ($C_{aqueous}$). EPA adapted Smith's (1987) steady state concentration model for estimating $C_{milkfat}$ and developed the $C_{aqueous}$ model shown in Equation 3-42 (EPA 1998). Chemicals that would partition to the aqueous phase of human milk include water-soluble chemicals, such as salts of metals, and other hydrophilic chemicals that may be in equilibrium with bound forms of the chemical in different tissues. The $C_{aqueous}$ equation assumes that the chemical concentration in the aqueous phase of milk is directly proportional to the chemical concentration in the mother's blood plasma. The portion of chemical sequestered in red blood cells (e.g., bound to RBC proteins) is assumed to be unavailable for direct transfer to breast milk.

Equation 3-42. Chemical Concentration in Aqueous Phase of Breast Milk

$$C_{aqueous} = \frac{DAI_{mat} \times f_{pl} \times Pc_{bm}}{k_{aq\_elac} \times f_{pm}}$$

where:

$C_{aqueous}$	=	Concentration of chemical in aqueous phase of maternal milk (mg/kg)
DAI_{mat}	=	Daily absorbed maternal chemical dose (mg/kg-day; calculated by Equation 3-39)
f_{pl}	=	Fraction of chemical in the body (based on absorbed intake) that is in the blood plasma compartment (unitless; value from literature or calculated by Equation 3-43)
Pc_{bm}	=	Partition coefficient for chemical between the plasma and breast milk in the aqueous phase (unitless); assumed to equal 1.0
k_{aq_elac}	=	Chemical-specific rate constant for total elimination of chemical in the aqueous phase of milk during nursing (per day; value from literature or calculated in Equation 3-44)
f_{pm}	=	Fraction of maternal weight that is blood plasma (unitless)

Equation 3-42 is a steady-state concentration model that, like the Equation 3-38 for $C_{milkfat}$, is dependent on the maternal absorbed daily intake (DAI_{mat}). In Equation 3-42, DAI_{mat} is multiplied by the fraction of the absorbed chemical that is circulating in the blood plasma compartment (f_{pl}) and a partitioning coefficient for the chemical between plasma and the aqueous phase of breast milk (Pc_{bm}). For highly water-soluble chemicals that are not transported via special carrier molecules, the chemical is assumed to diffuse passively from the mother's blood serum to the aqueous phase of her milk, in which case Pc_{bm} would equal 1.0. The denominator includes the biological elimination constant for the chemical in the aqueous phase of breast milk in lactating

women (k_{aq_elac}) and the fraction of the mother's weight that is plasma (f_{pl}). Because the model assumes steady-state, it does not account for chemical species with long half-lives in the body or for body burden losses due to lactation. These factors are important for highly lipophilic chemicals and for non-lipophilic chemicals such as methyl mercury, lead, and cadmium that partition into body compartments such as red blood cells and bone. While these latter chemicals or forms of these chemicals are water-soluble when free, they have relatively long half-lives because they are in equilibrium with the chemical bound to macromolecules in some tissue compartments. Lead is of particular concern because it can be released from the bone into the blood during lactation, and thus into the breast milk (EPA 2001a). Due to this limitation, the model may over- or underestimate exposure to the infant.

Because Equation 3-42 is based on the relationship between the chemical concentrations in the aqueous phase of breast milk and the blood plasma, a value for the fraction of the chemical in the mother's blood plasma (f_{pl}) is required. Ideally, an empirical value for f_{pl} should be used. If empirical values are not available, f_{pl} can be estimated from Equation 3-43, provided that an empirical value can be found for the fraction of the chemical in the body that is in the mother's whole blood compartment (f_{bl}; EPA 1998).

Equation 3-43. Fraction of Total Chemical in Body in the Blood Plasma Compartment

$$f_{pl} = \frac{f_{bl} \times f_{bp}}{f_{bp} + Pc_{RBC}\left(1 - f_{bp}\right)}$$

where:

f_{pl} = Fraction of chemical in body (based on absorbed intake) that is in the blood plasma compartment (unitless); chemical-specific

f_{bl} = Fraction of chemical in body (based on absorbed intake) in the whole blood compartment (unitless); chemical-specific

f_{bp} = Fraction of whole blood that is plasma (unitless)

Pc_{RBC} = Partition coefficient for chemical between red blood cells and plasma (unitless); chemical-specific

If the fraction of the total chemical in the body that is in the whole blood compartment (f_{bl}) is known for a given chemical, then the fraction of that chemical that is in blood plasma depends only on the partition coefficient for the chemical between the red blood cells and the plasma (Pc_{RBC}) and the fraction of whole blood that is plasma (f_{bp}).

Another parameter for which a value is needed to solve Equation 3-42 is the total chemical elimination rate for lactating women for hydrophilic chemicals, k_{aq_elac}. As for k_{fat_elac} for lipophilic chemicals, k_{aq_elac} for hydrophilic chemicals would be equal to k_{elim} plus the loss rate for the chemical in the aqueous phase of breast-milk during lactation. In the case of hydrophilic chemicals, EPA has yet to propose a term for the additional elimination of a chemical in the aqueous phase of milk from breast feeding. Given basic physiological mechanisms, we assume that chemical loss rates via urine are likely to be significantly higher than loss rates from nursing, however. This is because the counter-current anatomy of kidney tubules allows substantial concentration of chemicals in the tubules for elimination in urine compared with the concentration in circulating blood and because of active secretion of some chemicals into urine. Therefore, the best estimation of elimination of hydrophilic chemicals by lactating women is simply k_{elim}, the elimination of the chemical from a non-lactating woman, as shown in Equation

3-40. The extent to which k_{elim} is an underestimate of k_{aq_elac} for a given chemical will determine the extent of conservative bias in k_{aq_elac}.

Equation 3-44. Biological Elimination Rate Constant for Hydrophilic Chemicals

$$k_{aq\_elac} = k_{elim}$$

where:

k_{aq_elac} = Chemical-specific rate constant for total elimination of chemical by lactating women for hydrophilic chemicals (per day)

k_{elim} = Chemical-specific rate constant for total elimination of chemical by non-lactating women (per day; e.g., via urine, bile to feces, exhalation; value from literature or calculated from half-life using Equation 3-40)

3.4.4 Alternative Model for Infant Intake of Methyl Mercury

In this version of MIRC, we were unable to fully parameterize the aqueous model for mercury. In particular, no empirical value could be found for the steady-state fraction of total hydrophilic chemical body burden in the mother that is in the blood plasma (f_{pl}, see Exhibit 6-20). This parameter could be estimated using Equation 3-43 if a suitable chemical-specific fraction of chemical in the body that is in the whole blood (f_{bl}) could be found. However, the value found for f_{bl} is based on a single-dose study and is not considered reliable for use in chronic exposure calculations.

We therefore conducted a literature search to identify existing physiologically based toxicokinetic (PBTK) models of lactational transfer of methylmercury (MeHg) in humans. Most PBTK models that we identified focused on gestational transfer of mercury between mother and fetus, including a PBTK dynamic compartmental model for gestational transfer of MeHg in humans developed by Gearhart et al. (1995, 1996), and reparameterized by Clewell et al. (1999).

We did find, however, that Byckowski and Lipscomb (2001) had added a lactational transfer module to the Clewell et al. (1999) model. Byckowski and Lipscomb compared their model's predictions to epidemiological data from mother-nursing-infant pairs obtained following an accidental high-dose poisoning in Iraq (Amin-Zaki et al. 1976) and from 34 mother-nursing-infant pairs examined in a low-dose, chronic exposure environment (Fujita and Takabatake 1977). Using data from the Iraq incident, Byckowski and Lipscomb (2001) found good agreement between their model's predictions and the clinical data relating MeHg concentrations in breast milk to MeHg concentrations in infant's blood with time following the poisoning. To compare their model's predictions to data from chronic exposure to low doses of MeHg, Byckowski and Lipscomb (2001) simulated MeHg intake for 500 days prior to conception, continued through gestation, and 6.5 months (200 days) of lactation. Their model's predictions were consistent with Fujita and Takabatak's (1977) study, although use of hair/blood partition coefficients based on the results of the 1977 study precluded use of this comparison as model validation. Both the model predictions and the mean values from the 1977 data indicated that the concentration of MeHg in the blood of nursing infants was close to the MeHg concentration in their mothers' blood (approximately 0.025 to 0.027 mg/L, Figure 4 of report). At those blood concentrations, the PBTK model estimated the average maternal intake of MeHg to be 0.68 ± 0.33 (SD) µg/kg-day and the average infant intake of MeHg to be 0.80 ± 0.38 µg/kg-day. Therefore, for purposes of MIRC, the DAI_{inf} of MeHg is estimated to be the same as the maternal intake per unit body weight (Equation 3-42).

Equation 3-45. Calculation of Infant Average Daily Absorbed Dose of Methyl Mercury

$$DAI_{inf\_MeHg} = DAI_{mat\_MeHg}$$

where:

DAI_{inf_MeHg} = Average daily dose ofMeHg absorbed by infant from breast milk (mg/kg-day)

DAI_{mat_MeHg} = Average daily dose of methyl mercury absorbed by the mother, predominantly from fish (mg/kg-day)

4 Dose-Response Values Used for Assessment

Chemical dose-response values included in MIRC include carcinogenic potency slope factors for ingestion and non-cancer oral reference doses (RfDs) for chronic exposures. The cancer slope factors (CSFs) and RfDs for chemicals used to calculate PB-HAP emission thresholds are provided in Exhibit 4-3. Dose-response values in MIRC that are used for EPA's Risk and Technology Review (RTR) evaluations are consistent with dose-response data that the Agency's Office of Air Quality Planning and Standards (OAQPS) uses for risk assessments of hazardous air pollutants (HAPs) (EPA 2007). In general, OAQPS chose these values based on the following hierarchy of sources: EPA's Integrated Risk Information System (IRIS); the Centers for Disease Control's Agency for Toxic Substances and Disease Registry (ATSDR); and the California Environmental Protection Agency (CalEPA).

Exhibit 4-1. Oral Dose-response Values for PB-HAP Chemicals Used to Calculate RTR De Minimis Thresholds

Chemical	CAS No.	Cancer Slope Factor		Reference Dose	
		$\left(\frac{mg}{kg-day}\right)^{-1}$	Source	$\left(\frac{mg}{kg-day}\right)$	Source
Inorganics					
Cadmium compounds in food	7440439	not available		1.0E-03	IRIS
Mercury (elemental)	7439976	NA		not available	
Mercuric chloride	7487947	not available		3.0E-04	IRIS
Methyl mercury (MeHg)	22967926	not available		1.0E-04	IRIS
Organics					
Benzo(a)pyrene	50328	1.0E+01	EPA OAQPS [a]	not available	
2,3,7,8-TCDD	1746016	1.5E+05	EPA ORD	1.0E-09	ATSDR

ATSDR = Agency for Toxic Substances and Disease Registry
EPA OAQPS = EPA's Office of Air Quality Planning and Standards
EPA ORD = EPA's Office of Research and Development
IRIS = Integrated Risk Information System
NA = not applicable

[a] The method to assign oral cancer slope factors for polycyclic organic matter (POM) is the same as was used in the 1999 National Air Toxics Assessment (EPA 1999b). A complete description of the methodology is available at: http://www.epa.gov/ttn/atw/nata1999/99pdfs/pomapproachjan.pdf.

Cadmium

EPA has developed two chronic RfDs for cadmium, one for food and one for water, based on data in IRIS indicating a lower absorption efficiency of cadmium from food than from water. The default RfD set in MIRC is the higher RfD for cadmium compounds in food (no drinking water is assumed to occur when calculating *de minimis* thresholds). Users of MIRC who assess exposures via drinking water may need to use the RfD for Cd compounds in water (i.e., 5.0E-4 mg/kg-day).

Mercury

EPA's RfD for MeHg of 1E-04 mg/kg-day is based on a Benchmark Dose Lower Limit (BMDL) to dose-response data from an epidemiological study of neurobehavioral effects in children for which mercury concentrations had been measured in cord blood at birth. The island populations included in the study had been exposed for many years to MeHg in their seafoods. The RfD applies to the pregnant mother as well as young children. EPA has not specified the

minimum exposure duration at the RfD level of exposure that is appropriate to use in characterizing risk; we assume ten years for women of child-bearing age and 1 year for infants.

We note that human exposures to MeHg are primarily through the consumption of fish and shellfish (EPA 2001b). EPA found that on average, approximately 76 percent of the exposure to MeHg for women of childbearing age could be attributed to ingestion of mercury in freshwater and estuarine fish and shellfish, with the remaining 24 percent derived from marine fish and shellfish. Other sources accounted for less than 0.06 percent of total exposures (EPA 2001b).

Dioxins (2,3,7,8-TCDD)

For chemicals for which the critical health effect is developmental, either *in utero* and/or during the first months or years of life, the exposure duration and timing of exposure for comparison with the RfD (or comparable value) require special consideration. The most sensitive health endpoints for both mercury and 2,3,7,8-TCDD are neurological effects during development that have long-lasting effects on learning and social behaviors. To ensure a protective risk characterization for these chemicals, it is important to use the shortest exposure duration appropriate, at the appropriate life stage, for comparison with the toxicity reference values. This approach avoids "dilution" of an estimated average ADD that would result from averaging the lower daily chemical intake rates normalized to body weight for older children and adults with the potentially higher daily intake rates of infants over a longer exposure averaging period.

For 2,3,7,8-TCDD, although exposures may start in utero, a period of special concern is the nursing stage, because the highly lipophilic chemical is effectively transferred to the infant in the lipid phase of its mother's milk. ATSDR has established a minimal risk level (MRL) of 1E-09 mg/kg-day for exposures of the mother or infant of 365 days or longer to 2,3,7,8-TCDD. The MRL is based on a behavioral study of offspring of female rhesus macaques that were exposed prior to conception, during gestation, and while nursing (ATSDR 1998). In the critical study, all mothers were exposed for seven months prior to opportunities for mating; however, dates of conception ranged over a five month period (some females did not conceive for several cycles). When the offspring were born, their mothers had been exposed for an average of 16.2 months. Exposure continued for the 4-month lactation period, after which the offspring were weaned and tested for non-social and social behavioral deficits (ATSDR 1998, Bowman et al. 1989, Schantz and Bowman 1989, Schantz et al. 1992). It is not known whether the behavior deficits resulted from pre- or post-natal exposures or both. In this case, it is appropriate to compare the 365-day MRL to the ADD for women of child-bearing age and to the ADD for a nursing infant less than 1 year of age during risk characterization (see Section 5).

The convention for assessing risk from mixtures of dioxins is by application of a toxic equivalency factor (TEF) to dioxin concentrations, which are then expressed as toxic equivalents (TEQs). Of the dioxin congeners, 2,3,7,8-TCDD is the most widely studied, and considered to be one of the two most toxic congeners. It is therefore assigned a TEF of one, with the other dioxin congener TEQ concentrations scaled relative to 2,3,7,8-TCDD concentrations on the basis of toxicity. The World Health Organization (WHO) 2005 TEFs presented in Exhibit 4-2 are used for risk assessment of dioxins for RTR.

Exhibit 4-2. WHO 2005 Toxic Equivalency Factors (TEFs) for Dioxins

Dioxin Congener	CAS No.	WHO 2005 Toxic Equivalency Factor
1,2,3,4,6,7,8-Heptachlorodibenzofuran	67562394	0.01
1,2,3,4,7,8,9-Heptachlorodibenzofuran	55673897	0.01
1,2,3,4,7,8-Hexachlorod-benzofuran	70648269	0.1
1,2,3,6,7,8-Hexachlorodibenzofuran	57117449	0.1
1,2,3,7,8,9-Hexachlorodibenzofuran	72918219	0.1
2,3,4,6,7,8-Hexachlorodibenzofuran	60851345	0.1
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	39227286	0.1
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	57653857	0.1
1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	19408743	0.1
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	35822469	0.01
1,2,3,4,6,7,8,9-Octachlorodibenzofuran	39001020	0.0003
1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	3268879	0.0003
1,2,3,7,8-Pentachlorodibenzofuran	57117416	0.03
2,3,4,7,8-Pentachlorodibenzofuran	57117314	0.3
1,2,3,7,8-Pentachlorodibenzo-p-dioxin	40321764	1
2,3,7,8-Tetrachlorodibenzofuran	51207319	0.1
2,3,7,8-Tetrachlorodibenzo-p-dioxin	1746016	1

Source: van den Berg et al. 2006

Polycyclic Organic Matter

Previously, for risk assessment of inhalation exposures to polycyclic organic matter (POM) for EPA's National Air Toxics Assessments (NATA) and for RTR, OAQPS developed an approach for characterizing risks associated with the individual POM species and POM groups reported in NEI. Individual PAHs were assigned to one of eight POM groups according to cancer potencies derived by EPA for IRIS and by CalEPA, and based on assumptions regarding relative carcinogenicity. OAQPS then estimated an inhalation CSF for each group. The same approach was used to derive oral CSFs for POMs for use in multipathway risk assessment for RTR. POM groups (with their member POM species reported in NEI) and the corresponding CSFs used for RTR risk assessment are presented in Exhibit 4-3. As noted in the main TSD, a *de minimis* threshold for non-inhalation risk was derived only for benzo[a]pyrene, and facility emissions were then screened by comparing the total POM emissions to this threshold (where a toxicity-weighted sum of POM emissions is calculated for each evaluated source).

Exhibit 4-3. Oral Dose-response Values for Polycyclic Organic Matter (POM) Groups

Individual POM or POM group	CAS No.	Cancer Slope Factor[1] $\left(\frac{mg}{kg-day}\right)^{-1}$
POM Group 71002		
Benz(a)Anthracene/Chrysene (7PAH)	103	0.5
Total PAH	234	
Polycyclic Organic Matter	246	
16-PAH	40	
16PAH-7PAH	75040	
POM Group 72002		
Anthracene	120127	0.5
Pyrene	129000	
Benzo[g,h,i,]perylene	191242	
Benzo[e]pyrene	192972	
Benzo(c)phenanthrene	195197	
Perylene	198550	
Benzo(g,h,i)Fluoranthene	203123	
Benzo(a)fluoranthene	203338	
Fluoranthene	206440	
Acenaphthylene	208968	
1-Methylpyrene	2381217	
12-Methylbenz(a)Anthracene	2422794	
Methylbenzopyrenes	247	
Methylchrysene	248	
Methylanthracene	26914181	
Benzofluoranthenes	56832736	
9-Methylbenz(a)Anthracene	779022	
1-Methylphenanthrene	832699	
Acenaphthene	83329	
Phenanthrene	85018	
Fluorene	86737	
2-Methylnaphthalene	91576	
2-Chloronaphthalene	91587	
POM Group 73002		
7,12-Dimethylbenz[a]anthracene	57976	1000
POM Group 74002		
Dibenzo[a,i]pyrene	189559	100
D[a,h]pyrene	189640	

Exhibit 4-3, continued. Oral Dose-response Values for Polycyclic Organic Matter (POM) Groups

Individual PAH or PAH group	CAS No.	Cancer Slope Factor [a] $\left(\frac{mg}{kg-day}\right)^{-1}$
POM Group 75002		
3-Methylcholanthrene	56495	10
D[a,e]pyrene	192654	
5-Methylchrysene	3697243	
Benzo[a]pyrene	**50328**	
Dibenzo[a,h]anthracene	53703	
POM Group 76002		
Benzo[b+k]fluoranthene	102	1
Indeno[1,2,3-c,d]pyrene	193395	
B[j]fluoranthen	205823	
Benzo[b]fluoranthene	205992	
Benzo[k]fluoranthene	207089	
D[a,j]acridine	224420	
Benz[a]anthracene	56553	
POM Group 77002		
Chrysene	218019	0.1
POM Group 77002		
7-PAH	75	0.5

[a] The method to assign oral cancer slope factors for polycyclic organic matter (POM) is the same as was used in the 1999 National Air Toxics Assessment (EPA 1999b). A complete description of the methodology is available at:
http://www.epa.gov/ttn/atw/nata1999/99pdfs/pomapproachjan.pdf.

5 Risk Characterization

For persistent and bioaccumulative hazardous air pollutants (BP-HAPs), risks from inhalation of a chemical directly from air generally will be negligible compared with risks from ingestion of the chemical with foodstuffs grown in an area subject to air deposition of the chemical. For other (non-PB) HAPs, inhalation risks can be estimated separately and compared with risks associated with ingestion exposure to determine the focus of subsequent tiers of the risk assessment. Risk characterization for carcinogens with a linear mode of action at low doses is described in Section 5.1. Risk characterization for chemicals likely to exhibit a threshold for response (e.g., non-cancer hazards) is described in Section 5.2.

5.1 Cancer Risks

The estimated risk of developing cancer from exposure to a chemical from a specified source is characterized as the excess lifetime cancer risk (ELCR). The ELCR represents the incremental probability of an individual developing cancer over a lifetime as a result of lifetime exposure to the chemical. For a known or suspected carcinogen with a low-dose linear mode of action, the estimated ELCR is calculated as the product of the lifetime average daily dose (LADD) and the cancer slope factor (SF):

Equation 5-1. Calculation of Excess Lifetime Cancer Risk

$$ELCR = LADD \times CSF$$

where:

ELCR	=	Estimated excess lifetime cancer risk from a chemical summed across all exposure pathways and media (unitless)
LADD	=	Lifetime average total daily dose from all exposure pathways and media (mg/kg-day)
CSF	=	Oral carcinogenic potency slope factor for chemical (per mg/kg-day)

As described in Section 3.3, the *LADD* (in mg/kg-day) for a chemical is calculated to reflect age-related differences in exposure rates that are experienced by a hypothetical individual throughout his or her lifetime of exposure. The total chemical intake is normalized to a lifetime, which for the purposes of this assessment is assumed to be 70 years.

EPA considers the possibility that children might be more sensitive than adults to toxic chemicals, including chemical carcinogens (EPA 2005b,c). Where data allow, EPA recommends development of lifestage-specific cancer potency CSFs. To date, EPA has developed a separate slope factor for early lifestage exposure for only one chemical (i.e., 1,1,1-trichloroethane; EPA 2007b), and current data availability for most chemicals preclude this approach. EPA has, therefore, examined options for default adjustments of the CSF to protect children. To date, the only mode of action (MOA) for carcinogenesis for which EPA has adequate data to develop a reasonable quantitative default approach is mutagenesis (EPA 2005b,c). For carcinogens with a mutagenic MOA for cancer, EPA concluded that the carcinogenic potency of a chemical may be approximately tenfold greater for the first 2 years of life (i.e., birth up to second birthday) and threefold greater for the next 14 years of life (i.e., ages 2 through 15) than for adults (EPA 2005c). These conclusions are represented by age-dependent adjustment factors (ADAFs) of 10, 3, and 1 for the first two lifestages and for adults, respectively.

These three lifestages do not match the age categories for the home-grown food ingestion rates, the age categories in MIRC. As a consequence, ADAFs for the age groups in MIRC are adapted as time-weighted average values as follows:

$ADAF_{(<1)} = 10$ $\qquad$ $ADAF_{(6\text{-}11)} = 3$

$ADAF_{(1\text{-}2)} = \frac{(10 \times 1\,\text{yr}) + (3 \times 1\,\text{yr})}{2} = 6.5$ $\qquad$ $ADAF_{(12\text{-}19)} = \frac{(3 \times 4\ \text{yrs}) + (1 \times 4\ \text{yrs})}{8} = 2$

$ADAF_{(3\text{-}5)} = 3$ $\qquad$ $ADAF_{(adult)} = 1$

To estimate total lifetime risk from a lifetime of exposure to such a chemical, EPA recommends estimating the cancer risk for each of the three lifestages separately and then adding the risks for i = 1 to 6 age groups.

Equations 5-2 to 5-8. Lifetime Cancer Risk: Chemicals with a Mutagenic MOA for Cancer

Equation 5-2. $Risk_{(<1)} = ADD_{(0-<1)} \times 10 \times CSF \times (1\text{ yr}/70\text{ yr})$

Equation 5-3. $Risk_{(1\text{-}2)} = ADD_{(1\text{-}2)} \times 6.5 \times CSF \times (2\text{ yr}/70\text{ yr})$

Equation 5-4. $Risk_{(3\text{-}5)} = ADD_{(3\text{-}5)} \times 3 \times CSF \times (3\text{ yr}/70\text{ yr})$

Equation 5-5. $Risk_{(6\text{-}11)} = ADD_{(6\text{-}11)} \times 3 \times CSF \times (6\text{ yr}/70\text{ yr})$

Equation 5-6. $Risk_{(12\text{-}19)} = ADD_{(12\text{-}19)} \times 2 \times CSF \times (8\text{ yr}/70\text{ yr})$

Equation 5-7. $Risk_{(adult)} = ADD_{(adult)} \times 1 \times CSF \times (50\text{ yr}/70\text{ yr})$

Equation 5-8. $ELCR = \sum_{i=1}^{n} Risk_{(i)}$

In other words, Equation 5-8 indicates that the total extra lifetime cancer risk (ELCR) equals the sum of the age-group-specific risks estimated by Equations 5-2 through 5-7, where:

$Risk_{(<1)}$ = Risk from chemical ingestion in first year of life
$Risk_{(1\text{-}2)}$ = Risk from chemical ingestion from first birthday through age 2 years
$Risk_{(3\text{-}5)}$ = Risk from chemical ingestion from age 3 through 5 years of age
$Risk_{(6\text{-}11)}$ = Risk from chemical ingestion from age 6 through 11 years of age
$Risk_{(12\text{-}19)}$ = Risk from chemical ingestion from age 12 through 19 years of age
$Risk_{(adult)}$ = Risk from chemical ingestion from age 20 to 70 years age
$ADD_{(<1)}$ = Average daily dose for infants under one year of age (mg/kg-day)
$ADD_{(1\text{-}2)}$ = Average daily dose from first birthday through age 2 years of age (mg/kg-day)
$ADD_{(3\text{-}5)}$ = Average daily dose from age 3 through 5 years of age (mg/kg-day)
$ADD_{(6\text{-}11)}$ = Average daily dose from age 6 through 11 years of age (mg/kg-day)
$ADD_{(12\text{-}19)}$ = Average daily dose from age 12 through 19 years of age (mg/kg-day)
$ADD_{(adult)}$ = Average daily dose for adults age 20 to 70 years of age (mg/kg-day)
CSF = Oral carcinogenic potency slope factor for chemical (per mg/kg-day)
$Risk_{(i)}$ = Risk from chemical ingestion for the i^{th} age group
$ELCR$ = Total extra lifetime cancer risk (incremental or extra risk)
n = Number of age groups (i.e., 6)

5.2 Non-cancer Hazard Quotients

Non-cancer risks are presented as hazard quotients (HQs), that is, the ratio of the estimated daily intake (i.e., ADD) to the reference dose (e.g., chronic RfD). If the HQ for a chemical is equal to or less than 1, EPA believes that there is no appreciable risk that non-cancer health effects will occur. If the HQ is greater than 1, however, EPA cautions that adverse health effects are possible, although an HQ above 1 does not indicate an effect will definitely occur. This is because of the margin of safety inherent in the derivation of all RfD values. The larger the HQ value, the more likely it is that an adverse effect may occur.

5.2.1 Hazard Quotients for Chemicals with a Chronic RfD

For chemicals with a chronic RfD, MIRC calculates an HQ for each age group separately using Equation 5-9 to indicate the potential for adverse health effects associated with chronic exposure via ingestion pathways. The HQ is the ratio of a long-term, daily average exposure normalized to the receptor's body weight (i.e., ADD) to the RfD for that chemical.

Equation 5-9. Hazard Quotient for Chemicals with a Chronic RfD

$$HQ = \frac{ADD}{RfD}$$

where:

HQ	=	Hazard quotient for chemical (unitless)
ADD	=	Average daily ingested dose of chemical (mg/kg-day) from all food types and ingested media for the age group
RfD	=	Chronic oral reference dose for chemical (mg/kg-day)

5.2.2 Hazard Quotients for Chemicals with RfD Based on Developmental Effects

For chemicals for which the toxicity reference value is an RfD based on developmental effects in infants, children, or young animals, a shorter exposure duration (ED) and averaging time (AT) may be required. For this type of chemical (e.g., methylmercury, 2,3,7,8-TCDD), the appropriate ED/AT and sensitive lifestage for exposure may need to be estimated from the information provided in the critical developmental study(ies) from which the RfD was derived (e.g., in consultation with the RfD documentation in EPA's IRIS or in a toxicological profile developed for the chemical). For screening-level risk assessments, however, a conservative approach is to compare the highest ADD from among the child age categories provided in MIRC to the RfD.

5.2.3 Hazard Index for Chemicals with RfDs

When conducting screening-level assessments for multiple chemicals, it can be informative to calculate a hazard index (HI) for toxicologically similar chemicals (EPA 2000). The HI is the sum of HQs across chemicals as shown in Equation 5-12. As with the HQ, if the HI value is less than 1, adverse health effects are not expected for that suite of chemicals. If the screening level HI exceeds 1, however, the risk assessor is advised to evaluate the assumptions of the screening-level assessment to determine if more realistic local values are available for parameters that drive risk. In addition, the risk assessor may need to examine the mode of action (MOA) and target organ(s) for the chemicals with the highest HQs to develop an appropriate approach to assessing their potential joint action.

Equation 5-10. Hazard Index Calculation

$$HI = HQ_1 + HQ_2 \ldots HQ_n$$

where:

HI	=	Hazard index (unitless)
HQ_1	=	Hazard Quotient for chemical 1 (unitless)
HQ_2	=	Hazard Quotient for chemical 2 (unitless)
HQ_n	=	Hazard Quotient for chemical *n* (unitless)

The HI approach can be appropriate for chemicals with the same MOA and same target organ; however, MOA often is difficult to determine. An HI usually is "developed for each exposure route of interest, and for a single toxic effect or for toxicity to a single target organ" (EPA 2000; p 79). If a receptor is exposed to multiple chemicals that affect different target organs or that operate by different MOAs, and if more than one HQ is close to 1, the risk assessor is advised to perform a follow-on evaluation of assumptions and to consider whether chemical interactions may play a role in chemical toxicity (EPA 2000). Exposures to more than one chemical can result in a greater or lesser toxic response than might be predicted on the basis of one or the other chemical acting alone (toxicologically independent) or acting in concert (toxicologically similar chemicals). Users are referred to EPA's *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures* for approaches to assessing the potential for adverse health effects from exposure to multiple chemicals (EPA 2000).

Note that users of MIRC are responsible for determining how to interpret HQs for multiple chemicals.

6 Model Input Options

This section describes the input options currently included in MIRC. Required user inputs for environmental media concentrations and air deposition rates, such as those predicted by (output of) TRIM.FaTE, are described in Section 6.1. Values for farm-food-chain (FFC) parameters for specific types of produce and animal products are discussed in Section 6.2. Options for parameterizing receptor characteristics are described in Section 6.3, including age-group-specific values for body weight, water ingestion, and food ingestion by food type. Options for other exposure parameter values in MIRC, such as exposure frequency and loss of chemical during food preparation and cooking, are provided in Section 6.4.

Where values for chemical-specific parameters are presented, values are presented only for PB-HAP chemicals currently evaluated using the TRIM-based RTR screening scenario. The database included with MIRC contains chemical-specific parameter values for a large number of chemicals, because all of the chemical-specific input data compiled by EPA for use in HHRAP were uploaded into MIRC. However, only chemicals that are PB-HAPs evaluated for RTR are discussed in this document, and the HHRAP inputs provided for other chemicals have not been evaluated. The data presented in this chapter were reviewed and used to develop the set of modeling defaults used to calculate *de minimis* screening thresholds for RTR. Note that the default values used to estimate RTR screening thresholds, and the justification for selecting a specific value from the data sets described in this chapter, are discussed in Chapter 7.

6.1 Environmental Concentrations

As noted in Section 2, MIRC is intended to estimate exposures and risks to self-sufficient farming families from airborne chemicals. The tool analyzes one exposure scenario at a time; therefore, it is best used to evaluate a maximally exposed individual (MEI) or family when MIRC is used to screen for possible risks.

The following values specific to the air pollutant of concern are required inputs to MIRC:

- a single air concentration (in g/m^3);
- the fraction of chemical in the air that is in the vapor phase;
- air-to-surface deposition rates for both vapor- and particle-phase chemical in the air (in g/m^2-yr);
- two fish tissue concentrations, one each for forage and game fish (i.e., fish in TL 3 and TL 4) (in mg/kg wet weight);
- concentrations in drinking water (in g/L); and
- four chemical concentrations in soil (in µg/g dry weight), one each for:
    1. surface soil in produce growing area,
    2. surface soil where livestock feed,
    3. root-zone soil in produce growing area, and
    4. root-zone soil in livestock feed growing area.

The MIRC software is configured to estimate ingestion exposures via drinking water for a specified chemical concentration in the drinking water source (e.g., groundwater well).

The user must provide the inputs listed above; no default values are included for these parameters in MIRC. Media concentrations output by TRIM.FaTE can be entered into the tool manually from model output files or can be imported. For RTR evaluations, a tool to facilitate this process was developed using a Microsoft Excel routine written in Visual Basic.

6.2 Farm-Food-Chain Parameter Values

Using the chemical information specified in Section 6.1 above as inputs, MIRC calculates chemical concentrations in foods that are commonly grown or raised on family farms: exposed and protected fruits; exposed and protected vegetables; root vegetables; beef; total dairy products; pork; and poultry and eggs.

6.2.1 List of Farm-Food-Chain (FFC) Parameters

MIRC estimates chemical concentrations in the produce identified above using algorithms from HHRAP (EPA 2005a) as described in Section 3.2. Parameter values required for these HHRAP algorithms, including chemical-specific media transfer factors (e.g., soil-to-plant transfer coefficients) and plant- and animal-specific properties (e.g., plant interception fraction, quantity of forage consumed by cattle), are included in tables in MIRC. As described in Section 7, the HHRAP-recommended parameter values are the default values in MIRC; however, these and other inputs in MIRC can be edited as needed. Exhibit 6-1 describes the parameters that are included in the algorithms used to estimate chemical concentrations in the farm food categories. The parameter names and symbols are referenced in this section for plants/produce and animal products.

Exhibit 6-1. MIRC Parameters Used to Estimate Chemical Concentrations in Farm Foods

Parameter	Description	Units
Plants/Produce		
$Br_{AG\text{-}produce\text{-}DW(i)}$	Chemical-specific plant/soil chemical bioconcentration factor for edible portion of aboveground produce type *i, exposed or protected*	Unitless (g soil DW / g produce DW)
$Bv_{AG(i)}$	Chemical-specific air-to-plant biotransfer factor for aboveground produce type *i* for vapor-phase chemical in air	Unitless ([mg chemical / g DW plant] / [mg chemical / g air])
Fw	Fraction of wet deposition that adheres to plant surfaces; 0.2 for anions, 0.6 for cations and most organics	Unitless
Kds	Chemical-specific soil/water partition coefficient	L soil pore water / kg soil DW
$kp_{(i)}$	Plant-specific surface loss coefficient for aboveground exposed produce and animal forage and silage	yr^{-1}
$MAF_{(i)}$	Moisture adjustment factor for aboveground produce type *i* to convert the chemical concentration estimated for dry-weight produce to the corresponding chemical concentration for full-weight fresh produce	Percent water
RCF	Chemical-specific root concentration factor for tubers and root produce on a wet-weight (WW) basis	L soil pore water/ kg root WW
$Rp_{(i)}$	Plant-specific interception fraction for the edible portion of aboveground exposed produce or animal forage and silage	Unitless
$Tp_{(i)}$	Length of plant exposure to deposition per harvest of the edible portion of aboveground exposed produce or animal forage and silage	Year
$VG_{AG(i)}$	Empirical correction factor for aboveground *exposed* produce type *i* to address possible overestimate of the diffusive transfer of chemical from the outside to the inside of bulky produce, such as fruit	Unitless

Exhibit 6-1, continued. MIRC Parameters Used to Estimate Chemical Concentrations in Farm Foods

Parameter	Description	Units
$VG_{rootveg}$	Empirical correction factor for belowground produce (i.e., tuber or root vegetable) to account for possible overestimate of the diffusive transfer of chemicals from the outside to the inside of bulky tubers or roots (based on carrots and potatoes)	Unitless
$Yp_{(i)}$	Plant-specific yield or standing crop biomass of the edible portion of produce or animal feed	kg produce DW/m^2
Animal Products		
Bs	Soil bioavailability factor for livestock	Unitless
MF	Chemical-specific mammalian metabolism factor that accounts for endogenous degradation of the chemical	Unitless
$Ba_{(beef)}$	Chemical-specific biotransfer factor for chemical in diet of cow to chemical in beef on a fresh-wet (FW; equivalent to WW) basis	mg chemical/kg FW tissue/mg chemical/day *or* day/kg FW tissue
$Ba_{(dairy)}$	Biotransfer factor in dairy	day/kg FW tissue
$Ba_{(pork)}$	Biotransfer factor in pork	day/kg FW tissue
$Ba_{(poultry)}$	Biotransfer factor in poultry	day/kg FW tissue
$Ba_{(eggs)}$	Biotransfer factor in eggs	day/kg FW tissue
$Qs_{(m)}$	Quantity of soil eaten by animal type *m* each day	kg/day
$Qp_{(i,m)}$	Quantity of plant feed type *i* consumed per animal type *m* each day	kg/day

Source: EPA Source: EPA 2005a
DW = dry weight; FW = fresh weight; WW = wet weight

6.2.2 Produce Parameter Values

Exhibit 6-2 and **Error! Reference source not found.** provide the chemical-specific input values that are the current defaults for produce FFC food types in MIRC. Exhibit 6-4 presents additional non-chemical-specific input values for parameters used in the algorithms that calculate chemical concentrations in produce. Unless otherwise noted, the default parameter values were obtained from HHRAP. Options for other parameter values are not included in MIRC at this time; however, the user can overwrite values if appropriate. Refer to HHRAP (EPA 2005a, Chapter 5 and associated appendices) for detailed descriptions of these parameters and documentation of input values.

Exhibit 6-2. Chemical-Specific Inputs for Produce Parameters for Chemicals Included in MIRC

Chemical	Fraction of Wet Deposition (*Fw*) (unitless)[a]	Root Concentration Factor (*RCF*) (belowground) (L/kg)[b]	Soil-Water Partition Coefficient (*Kds*) (L/kg)[c]	Chemical Air-to-Plant Biotransfer Factor ($Bv_{AG(i)}$) (unitless)[d]
Inorganics				
Cadmium compounds	0.6	NA	7.5E+01	NA [e]
Mercury (elemental)	0.6	NA	1.0E+03	0 [f]
Mercuric chloride	0.6	NA	5.8E+04	1.8E+03
Methyl mercury	0.6	NA	7.0E+03	0 [f]
Organics				
Benzo(a)anthracene	0.6	5.7E+03	6.0E+04	1.9E+04
Benzo(a)pyrene	0.6	9.7E+03	1.6E+05	1.2E+05
Benzo(b)fluoranthene	0.6	1.2E+04	1.0E+04	1.7E+03
Benzo(k)fluoranthene	0.6	1.2E+04	1.9E+05	2.1E+05
Chrysene	0.6	5.7E+03	6.0E+04	6.9E+02
Dibenz(a,h)anthracene	0.6	2.3E+04	5.8E+05	3.1E+07
Indeno(1,2,3-cd) pyrene	0.6	2.8E+04	5.3E+05	3.7E+05
2,3,7,8-TCDD	0.6	4.0E+04	3.9E+04	6.6E+04

Source: EPA 2005a. NA = not applicable.

[a] 6E-01 is the value for cations and most organic chemicals. As described in HHRAP (EPA 2005a), Appendix B (available at http://www.epa.gov/osw/hazard/tsd/td/combust/finalmact/ssra/05hhrapapb.pdf), EPA estimated this value (EPA 1994a, 1995a) from a study by Hoffman et al. (1992) in which soluble gamma-emitting radionuclides and insoluble particles tagged with gamma-emitting radionuclides were deposited onto pasture grass via simulated rain. Note that the values developed experimentally for pasture grass may not accurately represent all aboveground produce-specific values. Also note that values based on the behavior of insoluble particles tagged with radionuclides may not accurately represent the behavior of organic compounds under site-specific conditions.

[b] For nonionic organic chemicals, as described in HHRAP (EPA 2005a), Appendix A (available at http://www.epa.gov/osw/hazard/tsd/td/combust/finalmact/ssra/05hhrapapa.pdf), *RCF* is used to calculate the below-ground transfer of contaminants from soil to a root vegetable on a wet-weight basis as shown in Equation 3-6. EPA estimated chemical-specific values for *RCF* from empirical regression equations developed by Briggs et al. (1982) based on their experiments measuring uptake of compounds into barley roots from growth solution. Briggs' regression equations allow calculation of *RCF* values from log K_{ow}. For metals and mercuric compounds, empirical values for soil to root vegetable transfer on a dry-weight basis are available from EPA or other sources.

[c] As discussed in HHRAP (EPA 2005a), Appendix A, *Kds* describes the partitioning of a compound between soil pore-water and soil particles and strongly influences the release and movement of a compound into the subsurface soils and underlying aquifer. *Kds* values for mercuric compounds were obtained from EPA (1997b). For all PAHs except for benzo(b)fluoranthene, *Kds* values were obtained from EPA 2004a. For benzo(b)fluoranthene and 2,3,7,8-TCDD, *Kds* values were calculated using the correlation equation provided in EPA 1993.

[d] As discussed in HHRAP (EPA 2005a), Appendix A, the value for mercuric chloride was obtained from EPA 1997b. $Bv_{AG(i)}$ values for PAHs were calculated using the correlation equation derived for azalea leaves as cited in Bacci et al. (1992), then reducing this value by a factor of 100, as suggested by Lorber (1995), who concluded that the Bacci factor reduced by a factor of 100 was similar to his own observations in various studies. The 2,3,7,8-TCDD value was obtained from Lorber and Pinsky (2000).

[e] It is assumed that metals, with the exception of vapor-phase elemental mercury, do not transfer significantly from air into leaves.

[f] Speciation and fate and transport of mercury from emissions suggest that $Bv_{AG(i)}$ values for elemental and methyl mercury are likely to be zero (EPA 2005a).

Exhibit 6-3. Chemical-Specific Inputs by Plant Type for Chemicals in MIRC

Compound Name	Plant Part	Plant-Soil Bio-Concentration Factor ($Br_{AG\text{-}produce\text{-}DW(i)}$) (unitless) [a]	Empirical Correction Factor- Belowground Produce ($VG_{rootveg}$) (unitless) [b]	Empirical Correction Factor- Aboveground Produce ($VG_{AG(i)}$) (unitless) [c]
Inorganics				
Cadmium compounds	Exp. Fruit	1.3E-01	NA	1.0E+00
	Exp. Veg.	1.3E-01	NA	1.0E+00
	Forage	3.6E-01	NA	1.0E+00
	Grain	6.2E-02	NA	NA
	Prot. Fruit	1.3E-01	NA	NA
	Prot. Veg.	1.3E-01	NA	NA
	Root	6.4E-02	1.0E+00	NA
	Silage	3.6E-01	NA	5.0E-01
Mercury (elemental)	Exp. Fruit	NA	NA	1.0E+00
	Exp. Veg.	NA	NA	1.0E+00
	Forage	NA	NA	1.0E+00
	Grain	NA	NA	NA
	Prot. Fruit	NA	NA	NA
	Prot. Veg.	NA	NA	NA
	Root	NA	1.0E+00	NA
	Silage	NA	NA	5.0E-01
Mercuric chloride	Exp. Fruit	1.5E-02	NA	1.0E+00
	Exp. Veg.	1.5E-02	NA	1.0E+00
	Forage	0	NA	1.0E+00
	Grain	9.3E-03	NA	NA
	Prot. Fruit	1.5E-02	NA	NA
	Prot. Veg.	1.5E-02	NA	NA
	Root	3.6E-02	1.0E+00	NA
	Silage	0	NA	5.0E-01
Methyl mercury	Exp. Fruit	2.9E-02	NA	1.0E-02
	Exp. Veg.	2.9E-02	NA	1.0E-02
	Forage	0	NA	1.0E+00
	Grain	1.9E-02	NA	NA
	Prot. Fruit	2.9E-02	NA	NA
	Prot. Veg.	2.9E-02	NA	NA
	Root	9.9E-02	1.0E-02	NA
	Silage	0	NA	5.0E-01

Exhibit 6-3, continued. Chemical-Specific Inputs by Plant Type for Chemicals in MIRC

Compound Name	Plant Part	Plant-Soil Bio-Concentration Factor ($Br_{AG\text{-}produce\text{-}DW(i)}$) (unitless) [a]	Empirical Correction Factor- Belowground Produce ($VG_{rootveg}$) (unitless) [b]	Empirical Correction Factor- Aboveground Produce ($VG_{AG(i)}$) (unitless) [c]
Organics				
Benzo(a)anthracene	Exp. Fruit	2.0E-02	NA	1.0E-02
	Exp. Veg.	2.0E-02	NA	1.0E-02
	Forage	2.0E-02	NA	1.0E+00
	Grain	2.0E-02	NA	NA
	Prot. Fruit	2.0E-02	NA	NA
	Prot. Veg.	2.0E-02	NA	NA
	Root	9.5E-02	1.0E-02	NA
	Silage	2.0E-02	NA	5.0E-01
Benzo(a)pyrene	Exp. Fruit	1.3E-02	NA	1.0E-02
	Exp. Veg.	1.3E-02	NA	1.0E-02
	Forage	1.3E-02	NA	1.0E+00
	Grain	1.3E-02	NA	NA
	Prot. Fruit	1.3E-02	NA	NA
	Prot. Veg.	1.3E-02	NA	NA
	Root	6.1E-02	1.0E-02	NA
	Silage	1.3E-02	NA	5.0E-01
Benzo(b)fluoranthene	Exp. Fruit	1.1E-02	NA	1.0E-02
	Exp. Veg.	1.1E-02	NA	1.0E-02
	Forage	1.1E-02	NA	1.0E+00
	Grain	1.1E-02	NA	NA
	Prot. Fruit	1.1E-02	NA	NA
	Prot. Veg.	1.1E-02	NA	NA
	Root	1.2E+00	1.0E-02	NA
	Silage	1.1E-02	NA	5.0E-01
Benzo(k)fluoranthene	Exp. Fruit	1.2E-02	NA	1.0E-02
	Exp. Veg.	1.2E-02	NA	1.0E-02
	Forage	1.2E-02	NA	1.0E+00
	Grain	1.2E-02	NA	NA
	Prot. Fruit	1.2E-02	NA	NA
	Prot. Veg.	1.2E-02	NA	NA
	Root	6.1E-02	1.0E-02	NA
	Silage	1.2E-02	NA	5.0E-01
Chrysene	Exp. Fruit	2.0E-02	NA	1.0E-02
	Exp. Veg.	2.0E-02	NA	1.0E-02
	Forage	2.0E-02	NA	1.0E+00
	Grain	2.0E-02	NA	NA
	Prot. Fruit	2.0E-02	NA	NA
	Prot. Veg.	2.0E-02	NA	NA
	Root	9.5E-02	1.0E-02	NA
	Silage	2.0E-02	NA	5.0E-01

Exhibit 6-3, continued. Chemical-Specific Inputs by Plant Type for Chemicals in MIRC

Compound Name	Plant Part	Plant-Soil Bio-Concentration Factor ($Br_{AG\text{-}produce\text{-}DW(i)}$) (unitless) [a]	Empirical Correction Factor- Belowground Produce ($VG_{rootveg}$) (unitless) [b]	Empirical Correction Factor- Aboveground Produce ($VG_{AG(i)}$) (unitless) [c]
Dibenz(a,h)anthracene	Exp. Fruit	6.8E-03	NA	1.0E-02
	Exp. Veg.	6.8E-03	NA	1.0E-02
	Forage	6.8E-03	NA	1.0E+00
	Grain	6.8E-03	NA	NA
	Prot. Fruit	6.8E-03	NA	NA
	Prot. Veg.	6.8E-03	NA	NA
	Root	4.1E-02	1.0E-02	NA
	Silage	6.8E-03	NA	5.0E-01
Indeno(1,2,3-cd)pyrene	Exp. Fruit	5.9E-03	NA	1.0E-02
	Exp. Veg.	5.9E-03	NA	1.0E-02
	Forage	5.9E-03	NA	1.0E+00
	Grain	5.9E-03	NA	NA
	Prot. Fruit	5.9E-03	NA	NA
	Prot. Veg.	5.9E-03	NA	NA
	Root	5.3E-02	1.0E-02	NA
	Silage	5.9E-03	NA	5.0E-01
2,3,7,8-TCDD	Exp. Fruit	4.6E-03	NA	1.0E-02
	Exp. Veg.	4.6E-03	NA	1.0E-02
	Forage	4.6E-03	NA	1.0E+00
	Grain	4.6E-03	NA	NA
	Prot. Fruit	4.6E-03	NA	NA
	Prot. Veg.	4.6E-03	NA	NA
	Root	1.0E+00	1.0E-02	NA
	Silage	4.6E-03	NA	5.0E-01

Source: EPA 2005a. NA = not applicable.

[a] As discussed in HHRAP (EPA 2005a), the $Br_{AG\text{-}produce\text{-}DW(i)}$ for aboveground produce and forage accounts for the uptake from soil and the subsequent transport of contaminants through the roots to the aboveground plant parts. For organics, correlation equations to calculate values for *Br* on a dry weight basis were obtained from Travis and Arms (1988). For cadmium, *Br* values were derived from uptake slope factors provided in EPA 1992. Uptake slope is the ratio of contaminant concentration in dry weight plant tissue to the mass of contaminant applied per hectare soil. *Br* aboveground values for mercuric chloride and methyl mercury were calculated using methodology and data from Baes, et al. (1984). *Br* forage values for mercuric chloride and methyl mercury (on a dry weight basis) were obtained from EPA 1997b. The HHRAP methodology assumes that elemental mercury doesn't deposit onto soils. Therefore, it's assumed that there is no plant uptake through the soil.

[b] As discussed in HHRAP (EPA 2005a), Appendix B, $VG_{rootveg}$ represents an empirical correction factor that reduces produce concentration. Because of the protective outer skin, size, and shape of bulky produce, transfer of lipophilic chemicals (i.e., log Kow greater than 4) to the center of the produce is not likely. In addition, typical preparation techniques, such as washing, peeling, and cooking, further reduce the concentration of the chemical in the vegetable as consumed by removing the high concentration of chemical on and in the outer skin, leaving the flesh with a lower concentration than would be the case if the entire vegetable were pureed without washing. For belowground produce, HHRAP (EPA 2005a) recommends using a VGr_{ootveg} value of 0.01 for PB-HAP with a log Kow greater than 4 and a value of 1.0 for PB-HAP with a log Kow less than 4 based on information provided in EPA 1994b. In developing these values, EPA (1994b) assumed that the density of the skin and the whole vegetable are equal (potentially overestimating the concentration of PB-HAP in belowground produce due to root uptake).

Exhibit 6-3, continued. Chemical-Specific Inputs by Plant Type for Chemicals in MIRC

[c] As discussed in HHRAP (EPA 2005a), Appendix B, VG_{ag} represents an empirical correction factor that reduces aboveground produce concentration and was developed to estimate the transfer of PB-HAP into leafy vegetation versus bulkier aboveground produce (e.g., apples). Because of the protective outer skin, size, and shape of bulky produce, transfer of lipophilic PB-HAP (log Kow greater than 4) to the center of the produce is not likely. In addition, typical preparation techniques, such as washing, peeling, and cooking, further reduces residues. For aboveground produce, HHRAP (EPA 2005a) recommends using a VG_{ag} value of 0.01 for PB-HAP with a log Kow greater than 4 and a value of 1.0 for PB-HAP with a log Kow less than 4 based on information provided in EPA 1994b. In developing these values, EPA (1994b) assumed the following: (1) translocation of compounds deposited on the surface of aboveground vegetation to inner parts of aboveground produce would be insignificant (potentially underestimating the concentration of PB-HAP in aboveground produce due to air-to-plant transfer); (2) the density of the skin and the whole vegetable are equal (potentially overestimating the concentration of PB-HAP in aboveground produce due to air-to-plant transfer); and (3) the thickness of vegetable skin and broadleaf tree skin are equal (effects on the concentration of PB-HAP in aboveground produce due to air-toplant transfer unknown).

For forage, HHRAP recommends a VG_{ag} value of 1.0, also based on information provided in EPA 1994b.

A VG_{ag} value for silage is not provided in EPA 1994b; the VGag value for silage of 0.5 was obtained from NC DEHNR (1997); however, NC DEHNR does not present a specific rationale for this recommendation. Depending on the composition of the site-specific silage, this value may under- or overestimate the actual value.

Exhibit 6-4. Non-Chemical-Specific Produce Inputs

Plant Part	Interception Fraction ($Rp_{(i)}$) (unitless) [a]	Plant Surface Loss Coefficient ($kp_{(i)}$) (1/year) [b]	Length of Plant Exposure to Deposition ($Tp_{(i)}$) (year) [c]	Yield or Standing Crop Biomass ($Yp_{(i)}$) (kg/m^2) [d]	Plant Tissue-Specific Moisture Adjustment Factor ($MAF_{(i)}$) (percent) [e]
Exposed Vegetable	0.982	18	0.16	5.66	92
Protected Fruit	NA	NA	NA	NA	90
Protected Vegetable	NA	NA	NA	NA	80
Forage (animal feed)	0.5	18	0.12	0.24	92
Exposed Fruit	0.053	18	0.16	0.25	85
Root Vegetables	NA	NA	NA	NA	87
Silage (animal feed)	0.46	18	0.16	0.8	92
Grain (animal feed)	NA	NA	NA	NA	90

Source: EPA 2005a. NA = not applicable.

[a] Baes et al. (1984) used an empirical relationship developed by Chamberlain (1970) to identify a correlation between initial Rp values and pasture grass productivity (standing crop biomass [*Yp*]) to calculate *Rp* values for exposed vegetables, exposed fruits, forage, and silage. Two key uncertainties are associated with using these values for *Rp*: (1) Chamberlain's(1970) empirical relationship developed for pasture grass may not accurately represent aboveground produce. (2) The empirical constants developed by Baes et al. (1984) for use in the empirical relationship developed by Chamberlain (1970) may not accurately represent the site-specific mixes of aboveground produce consumed by humans or the site-specific mixes of forage or silage consumed by livestock.

[b] The term *kp* is a measure of the amount of chemical that is lost to natural physical processes (e.g., wind, water) over time. The HHRAP-recommended value of 18 yr^{-1} (also recommended by EPA 1994a and 1998) represents the midpoint of a range of values reported by Miller and Hoffman (1983). There are two key uncertainties associated with using these values for *kp*: (1) The recommended equation for calculating *kp* includes a conservative bias in that it does not consider chemical degradation processes. (2) Given the reported range of *kp* values from 7.44 to 90.36 yr^{-1}, plant concentrations could range from about 1.8 times higher to about 5 times lower than the plant concentrations estimated in FFC media using the midpoint *kp* value of 18.

[c] HHRAP (EPA 2005a) recommends using a *Tp* value of 0.16 years for aboveground produce and cattle silage. This is consistent with earlier reports by EPA (1994a, 1998) and NC DEHNR (1997), which recommended treating *Tp* as a constant based on the average period between successive hay harvests. Belcher and Travis (1989) estimated this period at 60 days. *Tp* is calculated as 60 days ÷ 365 days/year = 0.16 years. For forage, the average of the average period between successive hay harvests (60 days) and the average period between successive grazing (30 days) is used (that is, 45 days), and *Tp* is calculated as (60 days + 30 days)/ 2 ÷ 365 days/yr = 0.12 yr. Two key uncertainties are associated with use of these values for *Tp*: (1) The average period between successive hay harvests (60 days) may not reflect the length of the growing season or the length between successive harvests for site-specific aboveground produce crops. The concentration of chemical in aboveground produce due to direct (wet and dry) deposition (*Pd*) will be underestimated if the site-specific value of *Tp* is less than 60 days, or overestimated if the site-specific value of *Tp* is more than 60 days.

[d] *Yp* values for aboveground produce and forage were calculated using an equation presented in Baes et al. (1984) and Shor et al. (1982): $Yp = Y_{hi} / A_{hi}$, where Y_{hi} = Harvest yield of i^{th} crop (kg DW) and A_{hi} = Area planted to i^{th} crop (m2), and using values for Y_h and A_h from USDA (1994b and 1994c). A production-weighted U.S. average *Yp* of 0.8 kg DW/m2 for silage was obtained from Shor et al. 1982.

[e] *MAF* represents the plant tissue-specific moisture adjustment factor to convert dry-weight concentrations into wet-weight concentrations (which are lower owing to the dilution by water compared with dry-weight concentrations). Values obtained from Chapter 10 of EPA's 2003 SAB Review materials for 3MRA Modeling System, Volume II, "Farm Food Chain and Terrestrial Food Web Data" (EPA 2003a), which references EPA 1997c. Note that the value for grain used as animal feed is based on corn and soybeans, not seed grains such as barley, oats, or wheat.

6.2.3 Animal Product Parameter Values

MIRC also requires chemical-specific inputs for many of the animal product algorithms. The relevant values are shown in Exhibit 6-5 for the chemicals included in MIRC to date. The HHRAP algorithms require additional inputs for the animal products calculations that are not specific to PB-HAPs, but are specific to the animal and animal product type. The soil and plant ingestion rates recommended in HHRAP for beef cattle, dairy cattle, swine, and chicken are provided in Exhibit 6-6.

Exhibit 6-5. Animal Product Chemical-specific Inputs for Chemicals Included in MIRC

Compound Name	Soil Bio-Availability Factor (*Bs*) (unitless)	Biotransfer Factors (Ba_m) (day/kg FW tissue) [a] and Metabolism Factors (MF) (unitless) [b]						
		Mammal				Non-mammal		
		Beef (Ba_{beef})	Dairy (Ba_{dairy})	Pork (Ba_{pork})	*MF*	Eggs (Ba_{eggs})	Poultry ($Ba_{poultry}$)	*MF*
Cadmium compounds	1	1.2E-04	6.5E-06	1.9E-04	1	2.5E-03	1.1E-01	NA
Mercury (elemental)	1	0	0	0	1	0	0	NA
Mercuric chloride	1	1.1E-04	1.4E-06	3.4E-05	1	2.4E-02	2.4E-02	NA
Methyl mercury	1	1.2E-03	1.7E-05	5.1E-06	1	3.6E-03	3.6E-03	NA
Benzo(a)anthracene	1	4.0E-02	8.4E-03	4.8E-02	0.01	1.7E-02	2.9E-02	NA
Benzo(a)pyrene	1	3.8E-02	7.9E-03	4.5E-02	0.01	1.6E-02	2.8E-02	NA
Benzo(b)fluoranthene	1	3.6E-02	7.6E-03	4.4E-02	0.01	1.5E-02	2.7E-02	NA
Benzo(k)fluoranthene	1	3.6E-02	7.7E-03	4.4E-02	0.01	1.5E-02	2.7E-02	NA
Chrysene	1	4.0E-02	8.4E-03	4.8E-02	0.01	1.7E-02	2.9E-02	NA
Dibenz(a,h)anthracene	1	3.1E-02	6.5E-03	3.7E-02	0.01	1.3E-02	2.3E-02	NA
Indeno(1,2,3-cd) pyrene	1	2.9E-02	6.2E-03	3.6E-02	0.01	1.2E-02	2.2E-02	NA
2,3,7,8-TCDD	1	2.6E-02	5.5E-03	3.2E-02	1	1.1E-02	1.9E-02	NA

Source: EPA 2005a, unless otherwise indicated. NA = not applicable.

[a] As discussed in HHRAP (EPA 2005a), Appendix A, biotransfer factors for mercury compounds were obtained from EPA 1997b. Considering speciation, fate, and transport of mercury from emission sources, elemental mercury is assumed to be vapor-phase and hence is assumed not to deposit to soil or transfer into aboveground plant parts. As a consequence, there is no transfer of elemental mercury into animal tissues. Biotransfer factors for cadmium compounds were obtained from EPA 1995b. Biotransfer factors for 2,3,7,8-TCDD and PAHs were calculated from chemical octanol-water partitioning coefficients (Kow values) using the correlation equation from RTI (2005) and assuming the following fat contents: milk - 4%; beef - 19%; pork - 23%; poultry -14%; and eggs - 8%.

[b] As discussed in HHRAP (EPA 2005a), EPA (1995c) recommends using a metabolism factor (*MF*) to account for metabolism of PAHs by mammals to offset the amount of bioaccumulation suggested by biotransfer factors. EPA has recommended an *MF* of 0.01 for bis(2-ethylhexyl)phthalate (BEHP) and 1.0 for all other chemicals (EPA 1995d). For MIRC, an *MF* of 0.01 is also used to calculate concentrations of PAHs in food products from mammalian species based on the work of Hofelt et al. (2001). This factor takes into account the P450-mediated metabolism of PAHs in mammals; applying this factor in our approach reduced the concentrations of chemicals in beef, pork, and dairy by two orders of magnitude.

Exhibit 6-6. Soil and Plant Ingestion Rates for Animals

Animal	Soil Ingestion Rate – $Qs_{(m)}$ (kg/day) [a]	Plant Part Consumed by Animal	Plant Ingestion Rate – $Qp_{(l,m)}$ (kg/day)
Beef cattle [b]	0.5	Silage	2.5
		Forage	8.8
		Grain	0.47
Dairy cattle [c]	0.4	Silage	4.1
		Forage	13.2
		Grain	3.0
Swine [d]	0.37	Silage	1.4
		Grain	3.3
Chicken (eggs) [e]	0.022	Grain	0.2

Source: EPA 2005a HHRAP (Chapter 5).

[a] Beef cattle: NC DEHNR (1997) and EPA (1994b) recommended a soil ingestion rate for subsistence beef cattle of 0.5 kg/day based on Fries (1994) and NAS (1987). As discussed in HHRAP, Fries (1994) reported soil ingestion to be 4 percent of the total dry matter intake. NAS (1987) cited an average beef cattle weight of 590 kg, and a daily dry matter intake rate (non-lactating cows) of 2 percent of body weight. This results in a daily dry matter intake rate of 11.8 kg DW/day and a daily soil ingestion rate of about 0.5 kg/day.
Dairy cattle: NC DEHNR (1997) and EPA (1994b) recommended a soil ingestion rate for dairy cattle of 0.4 kg/day based on Fries (1994) and NAS (1987). As discussed in HHRAP, Fries (1994) reported soil ingestion to be 2 percent of the total dry matter intake. NAS (1987) cited an average beef cattle weight of 630 kg and a daily dry matter intake rate (non-lactating cows) of 3.2 percent of body weight. This resulted in a daily dry matter intake rate of 20 kg/day DW, and a daily soil ingestion rate of approximately 0.4 kg/day. Uncertainties associated with Qs include the lack of current empirical data to support soil ingestion rates for dairy cattle and the assumption of uniform contamination of soil ingested by cattle.
Swine: NC DEHNR (1997) recommended a soil ingestion rate for swine of 0.37, estimated by assuming a soil intake that is 8% of the plant ingestion rate of 4.3 kg DW/day. Uncertainties include the lack of current empirical data to support soil ingestion rates and the assumption of uniform contamination of the soil ingested by swine.
Chicken: HHRAP (EPA 2005a) assumes that chickens consume 10 percent of their total diet (which is approximately 0.2 kg/day grain) as soil, a percentage that is consistent with the study from Stephens et al. (1995). Uncertainties include the lack of current empirical data to support soil ingestion rates for chicken and the assumption of uniform contamination of soil ingested by chicken.

[b] The beef cattle ingestion rates of forage, silage, and grain are based on the total daily intake rate of about 12 kg DW/day (based on NAS [1987] reporting a daily dry matter intake that is 2 percent of an average beef cattle body weight of 590 kg) and are supported by NC DEHNR (1997), EPA (1994b and 1990), and Boone et al. (1981). The principal uncertainty associated with these *Qp* values is the variability between forage, silage, and grain ingestion rates for cattle.

[c] The dairy cattle ingestion rates of forage, silage, and grain are based on the total daily intake rate of about 20 kg DW/day (NAS 1987; EPA 1992) as recommended by NC DEHNR (1997). Uncertainties include the proportion of each food type in the diet, which varies from location to location. Assuming uniform contamination of plant materials consumed by cattle also introduces uncertainty.

[d] Swine are not grazing animals and are assumed not to eat forage (EPA 1998). EPA (1994b and 1998) and NC DEHNR (1997) recommended including only silage and grains in the diet of swine. EPA (1995c) recommended an ingestion rate of 4.7 kg DW/day for a swine, referencing NAS (1987). Assuming a diet of 70 percent grain and 30 percent silage (EPA 1990), HHRAP estimated ingestion rates of 3.3 kg DW/day (grain) and 1.4 kg DW/day (silage). Uncertainties associated with *Qp* include variability of the proportion of grain and silage in the diet, which varies from location to location.

[e] Chickens consume grain provided by the farmer. The daily quantity of grain feed consumed by chicken is assumed to be 0.2 kg/day (Ensminger [1980], Fries [1982], and NAS [1987]). Uncertainties associated with this variable include the variability of actual grain ingestion rates from site to site. In addition, assuming uniform contamination of plant materials consumed by chicken introduces some uncertainty.

6.3 Adult and Non-Infant Exposure Parameter Values

The exposure parameters included in MIRC and their default and other value options are summarized in the following subsections. The default values were selected to result in a highly conservative screening scenario. Parameter value options were primarily obtained or estimated from EPA's *Exposure Factors Handbook* (EFH; EPA 1997a) and *Child-Specific Exposure Factors Handbook* (CSEFH; EPA 2008a). Where values were reported for age groupings other than those used in MIRC (see Section 2.3 above for MIRC age groups), time-weighted average values were estimated for the MIRC age groups from the available data.

In MIRC, ingestion rates for home-produced farm food items are included for exposed fruit, protected fruit, exposed vegetables, protected vegetables, root vegetables, beef, total dairy, pork, poultry, and eggs. Those ingestion rates are already normalized to body weight (i.e., $g_{wet\ weight}$/kg-day), as presented in the original data analysis (EPA 1997a). The body weight parameter values presented in Exhibit 6-7, therefore, are not applied in the chemical intake (ADD) equations for these food types.

In MIRC, ingestion rates also are included for drinking water (mL/day), soil (mg/day), and fish (g/day). These ingestion rates, however, are on a per person basis (i.e., not normalized for body weight). The body weight parameter values presented in Exhibit 6-7, therefore, are applied in the chemical intake (ADD) equations for these media.

6.3.1 Body Weights

Body weight (BW) options included in MIRC include mean, 5^{th}, 10^{th}, 50^{th}, 90^{th}, and 95^{th} percentiles for adults and the five children's age groups. For its default screening assessment, EPA uses the mean BW for each age group. The BWs currently in the MIRC database are listed in Exhibit 6-7. For adults, BW represents the weighted average of male and female mean body weights for all races, ages 18-74 years, from EPA's 1997 EFH (EPA 1997a; Tables 7-4 and 7-5). In general, BW values for the five children's age groups were calculated from the summary data provided in Table 8-3 of EPA's 2008 CSEFH. For purposes of comparison, alternative BW values for children ages 12 through 19 years also were estimated using data from Portier et al. (2007). These values are listed in the last row of Exhibit 6-7, but are not included in MIRC. The means calculated using the two methods for children ages 12 through 19 years were essentially identical at 64 kg. The other percentile values differed by approximately 10 percent or less.

Exhibit 6-7. Mean and Percentile Body Weight Estimates for Adults and Children

Lifestage (years)	Duration (years)	Body Weight (kg)					
		Mean	5th	10th	50th	90th	95th
Adult [a] (20-70)	50	71.4	52.9	56.0	69.3	89.7	97.6
Child < 1 [b]	1	7.83	6.03	6.38	7.76	9.24	9.66
Child 1-2 [c]	2	12.6	9.9	10.4	12.5	14.9	15.6
Child 3-5 [d]	3	18.6	13.5	14.4	17.8	23.6	26.2
Child 6-11 [e]	6	31.8	19.7	21.3	29.3	45.6	52.5
Child 12-19 [f]	8	64.2	39.5	45.0	64.2	83.5	89.0
[Child 12-19 [g]	*8*	*64.3*	*41.1*	*44.6*	*60.9*	*88.5*	*98.4]*

[a] BW represents the sample-size weighted average of male and female mean body weights (all races, 18-74 years) from EPA's 1997 EFH (Tables 7-4 for males and 7-5 for females). Note that these weights include the weight of clothing, estimated to range from 0.09 to 0.28 kg. Although the 18 to 74 year age category in EPA's EFH does not match exactly the age 20 to 70 year categorization of adults in MIRC, the magnitude of error in the mean and percentile body weights is likely to be very small (i.e., less than 1%).

[b] Each BW represents a time-weighted average of body weights for age groups birth to <1 month, 1 to <3 months, 3 to <6 months, and 6 to <12 months from Table 8-3 of the 2008 CSEFH. Original sample sizes for each of these age groups can also be found in Table 8-3.

[c] Each BW represents a time-weighted average of body weights for age groups 1 to <2 years and 2 to <3 years from Table 8-3 of the 2008 CSEFH. Original sample sizes for each of these age groups can also be found in Table 8-3.

[d] BWs obtained directly from Table 8-3 of the 2008 CSEFH (age group 3 to <6 years).

[e] BWs obtained directly from Table 8-3 of the 2008 CSEFH (age group 6 to <11 years). This value represents a conservative (i.e., slightly low) estimate of BW for ages 6 through 11 years since 11-year olds are not included in this CSEFH age group.

[f] Mean BW estimated using Table 8-22 of the 2008 CSEFH, which is based on NHANES IV data as presented in Portier et al. (2007). This estimate was calculated as the average of the 8 single-year age groups from 12 to 13 years through 19 to 20 years. Values for the other percentiles were estimated using Portier et al., 2007.

[g] Each BW represents a time-weighted average of body weights for age groups 11 to <16 years and 16 to <21 years from Table 8-3 of the 2008 CSEFH. Note that estimated values include 11-year-olds and individuals through age 20, which contributes to uncertainty in the estimates for 12 to 19 years. Those values are provided for comparison purposes only and are not included in MIRC.

6.3.2 Water Ingestion Rates

MIRC also includes the option of calculating chemical ingestion via drinking water obtained from surface-water sources or from wells (i.e., from groundwater) in the contaminated area. Users have the option in MIRC to set drinking water ingestion rates to zero or to revise the drinking water ingestion rates in MIRC to better reflect site-specific water uses. The 2008 CSEFH recommends values for drinking water ingestion rates for children based on a study reported by Kahn and Stralka (2008). Table 3-4 of the CSEFH provides *per capita* estimates of community water ingestion rates by age categories. Community water ingestion includes both direct and indirect ingestion of water from the tap. *Direct ingestion* is defined as direct consumption of water as a beverage, while *indirect ingestion* includes water added during food or beverage preparation. The source of these data is the 1994-1996 and 1998 U.S. Department of Agriculture's (USDA's) *Continuing Survey of Food Intakes by Individuals* (CSFII) (USDA 2000). Exhibit 6-8 includes the drinking water ingestion rates for children that are included in MIRC.

Mean and percentile adult drinking water ingestion rates were obtained from EPA (2004b), which presents estimated *per capita* water ingestion rates for various age categories based on

data collected by the USDA's 1994–1996 and 1998 CSFII (USDA 2000). Adult ingestion rates, presented in Exhibit 6-8, represent community water ingestion, both direct and indirect as defined above, for males and females combined, ages 20 years and older.

Exhibit 6-8. Estimated Daily Per Capita Mean and Percentile Water Ingestion Rates for Children and Adults [a]

Lifestage (years)	Ingestion Rates, Community Water (mL/day)				
	Mean	50th	90th	95th	99th
Child <1 [b]	324	146	866 *	1,011 *	1,377 *
Child 1-2 [c]	294	217	654	857	1,290 *
Child 3-5 [d]	380	291	834	1,078	1,654
Child 6-11 [e]	447	350	980	1,235	1,870 *
Child 12-19 [f]	697	516	1,537	2,022 *	3,195 *
Adult [g]	1,098	920	2,224	2,801	4,488

Sources: EPA 2004b, 2008a

* The sample size does not meet minimum reporting requirements as described in EPA 2008a. For some of these MIRC age groupings, the values are based on the time-weighted average value for 2 or more age ranges from CSEFH Table 3-4. One or more age ranges within the group may not meet the minimum reporting requirements, but not necessarily all of them fall within this category.

[a] Source is Kahn and Stralka 2008, also presented in the CSEFH (EPA 2008a).

[b] Each IR represents a time-weighted average of ingestion rates for age groups birth to <1 month, 1 to <3 months, 3 to <6 months, and 6 to <12 months from Table 3-4 of the 2008 CSEFH.

[c] Each IR represents a time-weighted average of ingestion rates for age groups 1 to <2 years and 2 to <3 years from Table 3-4 of the 2008 CSEFH.

[d] Each IR represents the ingestion rate for age group 3 to <6 years from Table 3-4 of the 2008 CSEFH.

[e] Each IR represents the ingestion rate for age group 6 to <11 years from Table 3-4 of the 2008 CSEFH. This value represents a conservative (i.e., slightly low) estimate of IR for ages 6 through 11 years since 11-year olds are not included in this CSEFH age group.

[f] Each IR represents a time-weighted average of ingestion rates for age groups 11 to <16 years, 16 to <18, and 18 to <21 years from Table 3-4 of the 2008 CSEFH. Note that estimated values include 11-year-olds and individuals through age 20, which contributes to uncertainty in the estimates for 12 to 19 years.

[g] Adult drinking water ingestion rates were obtained from EPA (2004b), Appendix E, Part I, Table A1 for community water, both sexes (ages 20+), direct plus indirect water ingestion.

6.3.3 Local Food Ingestion Rates

MIRC includes mean, median, 90th, 95th, and 99th percentile food-specific ingestion rates (IRs) for consumers-only of farm food chain (FFC) media for adults and children. The mean and percentile values are from EPA's analysis of data from the USDA's 1987 to 1988 *Nationwide Food Consumption Survey* (NFCS) (USDA 1993), as presented in Chapter 13 of the Agency's *Exposure Factors Handbook* (i.e., Intake Rates for Various Home Produced Food Items) (EPA 1997a). Consumers-only means that individuals who did not report eating a specified type of food during the three-day period covered by the food ingestion part of the survey were not included in the analysis of ingestion rates for that food type. The questionnaire included the options for a household to self-identify in one or more of five categories: as a household that gardens, raises animals, hunts, fishes, or farms. As of September, 2008, that survey was the most recent NFCS available (EPA 2008a, CSEFH), and we are not aware of any that might be more recent.[2]

For the adult age group in MIRC, we compiled data on food-specific IRs separately for two types of households as indicated in the "Response to Questionnaire" (EPA 1997a, Chapter 13): (1) households that farm (F) and (2) households that garden or raise animals (HG for homegrown).

[2] *Note that EPA's 2008 CSEFH does not distinguish between exposed and protected fruits and vegetables when recommending food ingestion rates based on the same data set for the same age categories. EPA's 1997 analysis for its EFH therefore remains the most appropriate data source for use in MIRC.*

This division reflects EPA's data analysis. EPA tabulated IRs for fruits and vegetables for households that that farm and for households that garden. EPA tabulated IRs for animals and animal products for households that farm and for households that raise animals. Thus, the first type of household, F, represents farmers who may both grow crops and raise animals and who are likely to consume more home grown/raised foods than the second type of household. The second type of household, HG, represents the non-farming households that may consume lower amounts of home-grown or raised foods (i.e., HG encompasses both households that garden and households that raise animals).

The food-specific ingestion rates are based on the amount of each food type that households that farm (F) or households that garden and raise animals (HG) produced and brought into their homes for consumption and the number of persons consuming the food. EPA averaged the actual consumption rate for home-grown foods over the 1-week survey period.

The default food-specific ingestion rates in MIRC for adults are those for farming households (F) in Exhibit 6-9. The user can specify use of the generally less conservative, non-farming household (HG) ingestion rates if they are more appropriate for the user's exposure scenario (second column of IR values under Adults in Exhibit 6-9).

Exhibit 6-9. Summary of Age-Group-Specific Food Ingestion Rates for Farm Food Items

Product	Ingestion Rate by Age Group (g/kg-day)						
	Children (Farm and Homegrown) [e]					Adults	
	Child <1	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Farm [f]	Homegrown [g]
Mean							
Beef [a]	NA	1.49	2.21	3.77	1.72	2.63	2.66
Dairy [d]	NA	67	37	24.79	10.90	17.1	15.9
Eggs [d]	NA	2.5	1.4	0.86	0.61	0.90	0.75
Exposed Fruit [b]	NA	1.8	2.6	2.52	1.33	2.32	1.55
Exposed Vegetable	NA	3.5	1.7	1.39	1.07	2.17	1.57
Pork [d]	NA	2.2	2.1	1.49	1.17	1.30	1.34
Poultry [d]	NA	3.6	3.4	2.13	1.59	1.54	1.58
Protected Fruit [d]	NA	19	13	8.13	5.44	5.19 [c]	5.9
Protected Vegetable	NA	2.5	1.3	1.1	0.776	1.3	1.01
Root Vegetable	NA	2.5	1.3	1.32	0.937	1.39	1.15

Exhibit 6-9, continued. Summary of Age-Group-Specific Food Ingestion Rates for Farm Food Items

Product	Ingestion Rate by Age Group (g/kg-day)						
	Children (Farm and Homegrown) [e]					Adults	
	Child <1	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Farm [f]	Homegrown [g]
50th Percentile							
Beef [a]	NA	0.84	1.23	2.11	1.51	1.64	1.83
Dairy [d]	NA	102	60	39	14	12.1	10.8
Eggs [d]	NA	1.5	0.79	0.56	0.49	0.67	0.48
Exposed Fruit [b]	NA	1.2	1.82	1.11	0.609	1.3	0.88
Exposed Vegetable	NA	1.9	1.2	0.643	0.656	1.38	0.89
Pork [d]	NA	1.8	1.4	1.02	1.02	0.924	0.97
Poultry [d]	NA	2.9	2.7	1.4	1.4	1.06	1.37
Protected Fruit [d]	NA	10.2	7.6	4.2	2.3	2.08 [c]	2.42
Protected Vegetable	NA	1.94	1.04	0.791	0.583	0.599	0.64
Root Vegetable	NA	0.92	0.46	0.523	0.565	0.88	0.67
90th Percentile							
Beef [a]	NA	4.5	6.7	11.4	3.53	5.39	5.39
Dairy [d]	NA	148	82	54.67	26.98	34.9	34.9
Eggs [d]	NA	5.1	2.8	1.8	1.34	1.65	1.36
Exposed Fruit [b]	NA	3.7	5.4	6.98	3.41	5	3.41
Exposed Vegetable	NA	10.7	3.47	3.22	2.35	6.01	3.63
Pork [d]	NA	4.5	4.4	3.04	2.65	3.08	2.9
Poultry [d]	NA	7.4	6.8	4.58	3.28	3.47	2.93
Protected Fruit [d]	NA	53	36	24.14	16.19	15.14 [c]	16
Protected Vegetable	NA	3.9	2.5	2.14	1.85	3.55	2.32
Root Vegetable	NA	7.3	4.3	3.83	2.26	3.11	2.81
95th Percentile							
Beef [a]	NA	5.0	7.3	12.5	3.57	7.51	7.51
Dairy [d]	NA	139	75	52	27	44	44
Eggs [d]	NA	5.5	3.7	2.4	1.5	1.85	1.85
Exposed Fruit [b]	NA	4.1	6.1	12	4.8	6.12	5.0
Exposed Vegetable	NA	11.9	6.29	5.5	3.8	6.83	5.45
Pork [d]	NA	6.2	6.0	4.7	3.3	3.7	3.4
Poultry [d]	NA	8.2	7.2	5.3	3.7	4.8	3.3
Protected Fruit [d]	NA	59	42	28	20	19.16 [c]	19.1
Protected Vegetable	NA	9.4	5.1	3.12	2.2	5.4	3.05
Root Vegetable	NA	10.4	4.73	5.6	3.3	4.6	3.64

Exhibit 6-9, continued. Summary of Age-Group-Specific Food Ingestion Rates for Farm Food Items

Product	Ingestion Rate by Age Group (g/kg-day)						
	Children (Farm and Homegrown) [e]					Adults	
	Child <1	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Farm [f]	Homegrown [g]
99th Percentile							
Beef [a]	NA	5.3	7.8	13.3	4.3	11	12.5
Dairy [d]	NA	113	56	37	24	80	80
Eggs [d]	NA	16	12	8.6	5.0	6.6	6.6
Exposed Fruit [b]	NA	22	32.5	16	5.9	16	12.9
Exposed Vegetable	NA	12	7.4	13	5.7	10	10
Pork [d]	NA	9.1	9.9	6.3	4.2	4.9	4.3
Poultry [d]	NA	10	10	6.4	4.8	6.2	5.3
Protected Fruit [d]	NA	113	81	57	45	34.42 [c]	47.3
Protected Vegetable	NA	9.4	5.3	5.4	2.69	9.2	6.49
Root Vegetable	NA	10.4	4.7	7.5	5.1	7.5	7.5

Source: EPA 1997a (Chapter 13), unless otherwise noted.
NA = not applicable
[a] No data are available for Child 1-2 or Child 3-5. The value for Child 6-11 was used, scaled down by the ratio of the mean body weight for Child 1-2 or Child 3-5, as appropriate, to the mean body weight of Child 6-11.
[b] No data are available for Child 1-2. The value for this age group is the IR for Child 3-5, scaled down by the ratio of the mean body weight for Child 1-2 to the mean body weight for Child 3-5.
[c] These values represent a time-weighted average IR for two age groups, using exposure duration (ED) for the 20-39 (ED=20 years) and 40-69 year age groups (ED=30 years).
[d] In many cases, intake rates for children were not available in EPA's 1997 EFH. Intakes for these receptor groups were calculated using the methodology recommended in HHRAP (EPA 2005a), Section 6.2.2.2. Sources to develop these values included EPA 1997a and EPA 2003b.
[e] In Chapter 13 of the 1997 EPA EFH, age group-specific IRs are provided for home produced items as a whole; separate IRs are not presented for children from households that raise animals and households that farm.
[f] These values represent the IRs for "households who farm."
[g] These values represent the IRs for "households who raise animals."

For children, EPA estimated food-specific IRs for four age categories (EPA 1997a): 1 to 2 years, 3 to 5 years, 6 to 11 years, and 12 to 19 years. Sample sizes were insufficient to distinguish IRs for children in different types of households; hence, for children, a single IR value represents both F and HG households for a given food type and age category (Exhibit 6-9). For some food types and age categories, there were insufficient data for EPA to provide consumer-only intake rates (i.e., data set for the subpopulation consisted of fewer than 20 observations). The HHRAP methodology, Section 6.2.2.2, recommends a method by which to calculate the "missing" age-specific consumer-only ingestion rates, as explained below. Food-specific intake rates (IRs) for those child age groups and food items not included in Chapter 13 of the 1997 EFH, that is $IR_{age\_group\_x}$, were derived using the following information:

- Mean or percentile-specific *consumer-only* intake of the farm food item, as brought into the home, for the total NFCS survey population (from EFH Chapter 13) – IR_{CO_total};
- Mean or percentile-specific *per capita* intake of the food type from all sources, as consumed, for the specific child age group, from Chapter 3 of the *CSFII Analysis of Food Intake Distributions* (EPA 2003c) – $IR_{PC,\ age\_group\_x}$; and
- Mean or percentile-specific *per capita* intake of the farm food item for the total CSFII survey population (from Chapter 3 of EPA 2003c) – IR_{PC_total}.

The ratio of $IR_{PC,\ age\_group\_x}$ to IR_{PC_total} from the CSFII data shows the consumption rate of a particular food type by a specific age group relative to the consumption rate for that food type for the population as a whole. The ratio of $IR_{CO,\ age\_group\_x}$ to IR_{CO_total}, that is the consumption rate of a particular food type by a specific age group (consumers only) relative to the consumption rate for that food type for the NFCS survey population as a whole (consumers only), should be approximately the same. Given the assumption that the two ratios are equal, Equation 6-2 was used to calculate the "missing" age-specific consumer-only *IRs*:

Equation 6-1. Calculation of Age-Group-Specific and Food-Specific Ingestion Rates

$$IR_{CO,\ age\_group\_x} = \frac{IR_{CO\_total} \times IR_{PC,\ age\_group\_x}}{IR_{PC\_total}}$$

where:

$IR_{CO,\ age\_group\_x}$ = Mean or percentile-specific *consumer-only* intake of the food type from all sources, as consumed, for the specific child age group X

IR_{CO_total} = Mean or percentile-specific *consumer-only* intake of the farm food item, as brought into the home, for the total NFCS survey population

$IR_{PC,\ age\_group\_x}$ = Mean or percentile-specific *per capita* intake of the food type from all sources, as consumed, for the specific child age group X from the CSFII

IR_{PC_total} = Mean or percentile-specific *per capita* intake of the farm food item for the total CSFII survey population

In this discussion, *per capita* (as opposed to *consumer-only*) indicates the intake rates are based on the entire population rather than the subset of the population that ingests the particular food category (i.e., consumers). Here, the use of *per capita* ingestion rates are recommended by the HHRAP methodology because no consumer-only percentile-specific intakes are provided for the different age groups.

The above calculation implicitly assumes that the distribution of the consumption rate for a food type for a specific age group (consumers only) has the same shape as the distribution of the consumption rate for a food type for a specific age group in the general population (*per capita*). Otherwise, the separate calculation of each percentile might yield intake estimates that decrease as the percentile increases. This calculation artifact could occur if the shapes of the two distributions differ in the upper percentiles (or "tails") of the distributions.

In the instances where the above calculations were used to fill data gaps in the above exhibit, only the dairy child-specific age group intake estimates are not strictly increasing with increasing percentile. The distributions likely track better (and thus the above assumption of equal ratios is more reasonable) for lower percentiles, with deviations occurring due to outlier ingestion rates based on only a few respondents in the tails of the distributions. The MIRC defaults use the 90th percentile ingestion estimates, which are likely more reliable than the 95th or 99th percentile estimates in this particular calculation.

6.3.4 Local Fish Ingestion Rates

The USDA's 1987 to 1988 Nationwide Food Consumption Survey (NFCS) (USDA 1993, 1994a), as presented in Chapter 13 of the Agency's Exposure Factors Handbook (i.e., Intake Rates for Various Home Produced Food Items) (EPA 1997a), includes family-caught fish ingestion rates by age category. There are several disadvantages, however, to using that data source to

estimate fish ingestion rates. First, due to inadequate sample sizes, EPA did not report fish IRs for children less than 6 years of age. Second, the NFCS data were collected approximately two decades ago. Third, the reported fish IRs are for ages 6 to 11 and 12 to 19 and are based on 29 and 21 individuals in each age category, respectively (EPA 1997a, Table 13-23). Finally, the IRs from NFCS data are based on total weight of fish as brought into the home, and do not include losses from preparation of the fish (i.e., removal of inedible parts and, possibly, the skin). Estimates of preparation losses for fish intended to apply to the NFCS fish IR data are very uncertain and are based on a wide variety of freshwater, estuarine, and marine fish, and squid (EPA 1997a, Table 13-5). Therefore, a more recent survey was sought that included larger sample sizes, data for children younger than six years, and IRs for the parts of fish actually consumed.

EPA's (2002) analysis freshwater and estuarine fish consumption from the USDA's Continuing Survey of Food Intake by Individuals (CSFII) for 1994-96 and 1998 was chosen to provide *per capita* fish IR options by age category in MIRC. Although the fish consumption rates reported in the CSFII include all sources, commercial and self-caught, for purposes of screening level risk assessments, it was assumed that all freshwater and estuarine fish consumed are self-caught. The inclusion of commercially obtained and estuarine fish will overestimate locally caught freshwater fish IRs for many rural populations in the United States; however, it also may underestimate locally caught fish IRs for some populations (e.g., Native Americans, Asian and Pacific Island communities, rural African American communities). Because consumption of locally caught fish varies substantially from region to region in the United States and from one population or ethnic group to the next, users of MIRC are encouraged to use more locally relevant data whenever available.

For children ages 3 to 17 years and for adults, MIRC includes values for the mean and the 90th, 95th, and 99th percentile fish ingestion rates (freshwater and estuarine fish only) based on EPA's analysis of 1994-96 and 1998 CSFII data (EPA 2002, 2008a). As shown in EPA's 2008(a) CSEFH, Table 10-7, the 90th percentile *per capita* ingestion rates estimated from the two-day CSFII recall period are zero for some child age groups. Although not presented in CSEFH Table 10-7, median ingestion rates for all child age groups would be zero (considering the "consumer only" sample sizes [CSEFH Table 10-9] relative to the "*per capita*" sample sizes in Table 10-7).

The high percentile fish IRs that are zero result from the short duration of the CSFII survey (two days) compared with the averaging time of interest (a year) and the relatively infrequent consumption of fish (e.g., on the order of once a week to once a month or less) compared with the near daily ingestion of other types of food products (e.g., dairy, produce, meat). Use of zero for fish IRs, however, is not useful in MIRC. As a result, an alternative method was used to estimate fish ingestion rates for children and adults that could provide reasonable, non-zero values for all age groups and percentiles.

The alternative, age-group-specific fish ingestion rates were derived using values for each age group, *y*:

- Mean or other appropriate percentile *consumer-only* fish ingestion rates for age group *y*, $IR_{CO,y}$, from EPA's *Estimated Per Capita Fish Consumption in the United States* (EPA 2002), Section 5.2.1.1, Table 5, for freshwater/estuarine habitat.[3]

[3] *Most of these data also are provided in Table 10-9 of the CSEFH; the median values, however, are not presented in the CSEFH, and values for the mean and all other percentiles are slightly different due to rounding.*

- Fraction of the population consuming freshwater/estuarine fish, $F_{PC,y}$, calculated as consumer-only sample size / U.S. population sample for age group *y*. The data to calculate these fractions are available in the 2008 CSEFH and EPA 2002.

Equation 6-2 was used to calculate the alternative, *per capita* fish ingestion rates by age group ($IR_{PC,y}$):

Equation 6-2. Calculation of Alternative Age-Group-Specific Fish Ingestion Rates

$$IR_{PC,y} = IR_{CO,y} \times F_{PC,y}$$

where:

$IR_{PC,y}$	=	*Per capita* fish ingestion rate for age group *y* (g/day)
$IR_{CO,y}$	=	*Consumer-only* fish ingestion rates for age group *y* (g/day) (EPA 2002, Section 5.2.1.1, Table 5, for freshwater/estuarine habitat)
$F_{PC,y}$	=	Fraction of the population consuming freshwater/estuarine fish, calculated as consumer-only sample size / total U.S. population sample size for age group *y* (unitless) (2008 CSEFH, EPA 2002)

In the above, *per capita* (as opposed to *consumer-only*) indicates the intake rates are based on the entire population rather than the subset of the population that ingests the particular food category. Here, *per capita* ingestions are recommended by the HHRAP methodology because no consumer-only percentile-specific intakes are provided for the different age groups. However, over 90% of the respondents consumed milk products.

The mean and percentile consumer-only fish ingestion rates for children and adults and the fraction of the population consuming freshwater/estuarine fish used in calculating long-term *per capita* fish ingestion rates by age group are presented in **Error! Reference source not found.** and Exhibit 6-11. The mean and percentile *per capita* fish ingestion rates estimated using this methodology are summarized in Exhibit 6-12 and are available in MIRC.

The fish ingestion rates provided in Exhibit 6-12 and included in MIRC are intended to represent the harvest and consumption of fish in surface waters in a hypothetical depositional area. For site-specific application of this tool, users should consider using more localized survey data to estimate more appropriate fish ingestion rates. The fishing season varies substantially across the United States by latitude, and fish consumption patterns also vary by type of water body (e.g., ponds, lakes, rivers, streams, estuaries, coastal marine), cultural heritage, and general geographic area. Therefore, use of more localized information is encouraged.

As noted in Section 6.4.3, if the user overwrites the fish IRs shown in Exhibit 6-12 with fresh-weight as caught values (e.g., values obtained from a local creel survey), the user is advised to set non-zero values for the preparation and cooking loss factors L1 and L2 in Equation 3-15. Suggested values are presented in Section 6.4.3.

Exhibit 6-10. Daily Mean and Percentile *Consumer-Only* Fish Ingestion Rates for Children and Adults ($IR_{CO,y}$) [a]

Lifestage (years)	Ingestion Rates, All Fish (g/day)				
	Mean	50th	90th	95th	99th
Child <1	NA	NA	NA	NA	NA
Child 1-2 [b]	27.31	15.61	64.46	87.60	138.76 *
Child 3-5 [c]	40.31	23.04	95.16	129.31	204.84 *
Child 6-11 [d]	61.49	28.46	156.86 *	247.69 *	385.64 *
Child 12-19 [e]	79.07	43.18	181.40 *	211.15 *	423.38 *
Adult[f]	81.08	47.39	199.62 *	278.91	505.65 *

Sources: EPA 2002, 2008a

NA = not applicable; it is assumed that children < 1 year of age do not consume fish.

* Indicates that the sample size does not meet minimum reporting requirements as described in EPA 2002. Owing to the small sample sizes, these upper percentiles values are highly uncertain.

[a] *Per capita* fish ingestion (FI) rates for children by age group are available from Chapter 10 of the CSEFH (EPA 2008a); however, all 50th and some 90th percentile ingestion rates are zero. *Per capita* FI rates were therefore estimated as described in Equation 6-2 to provide reasonable, non-zero values for all age groups and percentiles.

[b] A fish IR for ages 1-2 years was not available. The value represents the consumer-only fish ingestion rate for ages 3 to 5 from EPA (2002) (Section 5.2.1.1 Table 5 [freshwater/estuarine habitat]), scaled down by the ratio of the mean Child 1-2 body weight to the mean Child 3-5 body weight.

[c] These values represent the consumer-only fish ingestion rate for ages 3 to 5 from EPA (2002), Section 5.2.1.1 Table 5 (freshwater/estuarine habitat). Sample size = 442.

[d] These values represent the consumer-only fish ingestion rate for ages 6 to 10 from EPA (2002), Section 5.2.1.1 Table 5 (freshwater/estuarine habitat). Sample size = 147.

[e] These values represent the time-weighted average *per capita* fish ingestion rate for ages 11 to 15 and 16 to 17 years from EPA (2002), Section 5.1.1.1 Table 5 (freshwater/estuarine habitat); the value may underestimate ingestion rate for ages 12 to 19 years. Sample size = 135.

[f] These values represent the consumer-only fish ingestion rate for individuals 18 years and older from EPA (2002), Section 5.2.1.1 Table 4 (freshwater/estuarine habitat). Sample size = 1,633.

Exhibit 6-11. Fraction of Population Consuming Freshwater/Estuarine Fish on a Single Day ($F_{PC,y}$)

Lifestage (years)	Fraction Consuming Fish
Child 3-5	0.0503 [a]
Child 6-11	0.0440 [b]
Child 12-19	0.0493 [c]
Adult	0.08509[d]

Sources: EPA 2002, 2008a

[a] This value was calculated using the ages 3 to 5 sample size for consumers only divided by the sample size for the U.S. population divided by 2 to represent the proportion consuming fish on a single day (the consumers-only group includes individuals who consumed fish on at least one of two survey days) to match the one-day ingestion rate.

[b] As in footnote a, the value was calculated using the ages 6 to 10 sample size for consumers only divided by the sample size for U.S. population divided by 2.

[c] The value was calculated by summing the ages 11 to 15 and 16 to 17 sample sizes for consumers only and dividing by both by the sum of the sample sizes for U.S. population and by a factor of 2.

[d] The value was calculated using the ages 18 and older sample size for consumers only divided by the sample size for U.S. population from Section 5.1.1.1 Table 4. The result was divided by 2 to represent a one-day sampling period in order to match the one-day ingestion rate.

Exhibit 6-12. Calculated Long-term Mean and Percentile *Per capita* Fish Ingestion Rates for Children and Adults ($IR_{PC,y}$)

Lifestage (years)	Ingestion Rates, All Fish (g/day)				
	Mean	50th	90th	95th	99th
Child <1	NA	NA	NA	NA	NA
Child 1-2 [a]	1.37	0.79	3.24	4.41	6.98
Child 3-5 [b]	2.03	1.16	4.79	6.51	10.3
Child 6-11[c]	2.71	1.25	6.90	10.9	17.0
Child 12-19 [d]	3.90	2.13	8.95	10.4	20.9
Adult [e]	6.90	4.03	16.99	23.73	43.02

Sources: EPA 2002, 2008a
NA = not applicable; it is assumed that children < 1 year of age do not consume fish.
[a] Values were calculated as (consumer-only IR for Child 1-2) x (fraction of population consuming fish for Child 3-5).
[b] Values were calculated as (consumer-only IR for Child 3-5) x (fraction of population consuming fish for Child 3-5).
[c] Values were calculated as (consumer-only IR for Child 6-11) x (fraction of population consuming fish for Child 6-11).
[d] Values were calculated as (consumer-only IR estimated for Child 12-19) x (fraction of population estimated to consume fish for Child 12-19).
[e] Values were calculated as (consumer-only IR for Adults) x (fraction of population consuming fish for Adults).

Applications to date of MIRC have used whole fish concentrations estimated by TRIM.FaTE. The proportion lipid in TL3 and TL4 fish in TRIM.FaTE is assumed to be 5.7 percent (by weight) for the whole fish, based on information provided by Thomann (1989). The lipid content of the part(s) of the fish normally consumed is likely to be less than 5.7 percent. For example, EPA estimated a consumption-weighted mean lipid value for fillets of fish from TL3 to be 2.6 percent and from TL4 to be 3.0 percent (Table 6-9 in EPA 2003b). If a user of MIRC wishes to account for reduced chemical concentration in fillet compared with whole fish for lipophilic chemicals, the user can specify a "preparation" loss of chemical (see Section 6.4).

For lipophilic chemicals (e.g., log Kow greater than 4), which partition primarily into the fatty tissues of fish, much of the higher concentration tissues might be stripped from the fish during preparation (e.g., belly fat, viscera which includes fat in liver, etc, fat under skin). The degree to which the concentration of chemical in a fillet is less than the average total concentration in the whole fish is chemical specific. Assuming that the chemical concentration in the fillet is the same as in the whole fish may result in a conservative bias for highly lipophilic chemicals. For persons who prefer to consume fillets with the skin on and do not discard belly fat, assuming the same concentration of chemical in the fish consumed as in the whole fish is protective.

6.3.5 Soil Ingestion Rates

Adult gardeners may incidentally ingest soils from gardening activities, and gardening and farming families might ingest soil particles that adhere to exposed fruits and exposed and belowground vegetables. Soils that are re-suspended in the air by wind can resettle on exposed fruits and vegetables. Children may incidentally ingest soils in those ways, but in addition, children playing outdoors may ingest soils directly or by hand-to-mouth activities during play. MIRC includes soil ingestion rate options by age group for these types of exposures. MIRC does not include options for children who may exhibit pica, or the recurrent ingestion of unusually high amounts of soil (i.e., on the order of 1,000 - 5,000 mg/day or more) (EPA 2008a).

Data on soil ingestion rates are sparse; the soil ingestion rates listed in Exhibit 6-13 and included in MIRC are based on very limited data, as is evident from the values listed. The studies evaluated by EPA for children generally focused on children between the ages of 1 and

3 to 6 years and were not specific to families that garden or farm. The default ingestion rates in MIRC are the 90th percentile values, as for other ingestion rate parameters.

Exhibit 6-13. Daily Mean and Percentile Soil Ingestion Rates for Children and Adults

Age Group (years)	Soil Ingestion Rate (mg/day)				
	Mean [a]	50th [a]	90th	95th	99th
Child < 1	NA				
Child 1-2	50	50	400 [b]	400 [b]	400 [b]
Child 3-5	50	50	400 [b]	400 [b]	400 [b]
Child 6-11	50	50	201 [c]	331 [d]	331 [d]
Child 12-19	50	50	201 [c]	331 [d]	331 [d]
Adult 20-70	50	50	201 [c]	331 [d]	331 [d]

Sources: EPA 1997a, 2008a

[a] For the mean and 50th percentile soil ingestion rates for children, value represents a "central tendency" estimate from EPA's 2008 CSEFH, Table 5-1. For adults, value is the recommended mean value for adults from EPA's 1997 EFH, Chapter 4, Table 4-23.

[b] These values are the recommended "upper percentile" value for children from EPA's 1997 EFH, Chapter 4, Table 4-23. The 2008 CSEFH included a high-end value associated with pica only.

[c] These values are 90th percentile adult ingestion rates calculated in Stanek et al. 1997, and they are used to represent older children and adults.

[d] These values are 95th percentile adult ingestion rates calculated in Stanek et al. 1997, and they are used to represent older children and adults.

6.3.6 Total Food Ingestion Rates

Although not included in MIRC for deterministic screening-level exposure and risk assessments, total food ingestion rates would be included in any probabilistic module developed for MIRC. The total food ingestion rates presented in Exhibit 6-14 will be used to normalize or to truncate the sum of food-specific ingestion rates to reasonable values. This procedure is particularly important when chemical intake from multiple upper-percentile food ingestion rates for different types of food are added together. Individuals representing the upper percentile ingestion rate for one food category might not be the same individuals who reported high percentile ingestion rates for one or any of the other food categories.

Exhibit 6-14. Daily Mean and Percentile *Per Capita* Total Food Intake for Children and Adults

Lifestage (years)	Percent of Group Consuming Food	Mean	50th	90th	95th	99th
Total Food Intake (g/day, as consumed)						
Child < 1 [a]	67.0% - 99.7% [h]	322	270	599	779	1152
Child 1-2 [b]	100%	1,032	996	1537	1703	2143
Child 3-5 [c]	100%	1,066	1,020	1,548	1,746	2,168
Child 6-11 [d]	100%	1,118	1,052	1,642	1,825	2,218
Child 12-19 [e]	100%	1,197	1,093	1,872	2,231	2,975
Adult [f]	100%	1,100	1,034	1,738	2,002	2,736
Total Food Intake (g/kg-day, as consumed)						
Child < 1 [a]	67.0% - 99.7% [h]	39	34	72	95	147
Child 1-2 [b]	100%	82	79	125	144	177
Child 3-5 [c]	100%	61	57	91	102	132
Child 6-11 [d]	100%	40	38	61	70	88
Child 12-19 [e]	100%	21	19	34	40	51
Adult [g]	100%	14.8	13.9	23.7	27.6	35.5

Sources: EPA 2005e, 2008a

[a] These values represent a time-weighted average for age groups birth to <1 month (N=88), 1 to <3 months (N=245), 3 to <6 months (N=411), and 6 to <12 months (N=678) from Table 14-3 of the 2008 CSEFH.

[b] These values represent a time-weighted average for age groups 1 to <2 years (N=1,002) and 2 to <3 years (N=994) from Table 14-3 of the 2008 CSEFH.

[c] These values were obtained from Table 14-3 of the 2008 CSEFH (age group 3 to <6 years, N=4,112).

[d] These values were obtained from Table 14-3 of the 2008 CSEFH (age group 6 to <11 years, N=1,553). These values represents a conservative (i.e., slightly low) estimate for ages 6 through 11 years since 11-year olds are not included in this CSEFH age group.

[e] These values represent a time-weighted average for age groups 11 to <16 years (N=975) and 16 to <21 (N=743) years from Table 14-3 of the 2008 CSEFH. Note that estimated values include 11-year-olds and individuals through age 20, which contributes to uncertainty in the estimates.

[f] These values represent a time-weighted average for age groups 20 to 39 years (N=2,950) and 40 to 69 years (N=4,818) from Table 5B of the 2005 EPA analysis of CSFII.

[g] These values represent a time-weighted average for age groups 20 to 39 years (N=2,950) and 40 to 69 years (N=4,818) from Table 5A of the 2005 EPA analysis of CSFII.

[h] Percents consuming foods from Table 14-3 of the 2008 CSEFH include: 67.0% (birth to <1 month); 74.7% (1 to <3 months); 93.7% (3 to <6 months); and 99.7% (6 to <12 months). Infants under the age of 1 that consume breast milk are classified as "non-consumers" of food.

6.4 Other Exposure Factor Values

The other exposure parameters included in the MIRC algorithms are exposure frequency (Section 6.4.1), fraction of the food type obtained from the contaminated area (Section 6.4.2), and reduction in the weight of the food types during preparation and cooking (Section 6.4.3). For the breast milk ingestion pathway, additional exposure parameters are included in the FFC algorithms (Section 6.5).

6.4.1 Exposure Frequency

The exposure frequency (EF) represents the number of days per year that an individual consumes home-produced food items that are contaminated with the chemical being evaluated. In MIRC, the default value for EF is 365 days/year for all exposure sources and all potential

receptors. This assumption is consistent with the food ingestion rates used in MIRC (i.e., daily intake rates equivalent to annual totals divided by 365 days) and does not imply that residents necessarily consume home-produced food products every day of the year.

If the user wishes to evaluate daily intake rates based on shorter averaging times, the user can overwrite both the food-specific ingestion rates and the EF for each home-grown food product. Users of MIRC might want to specify a lower EF values for various food types where residents obtain some of their diet from commercial sources and where consumption of home grown produce is seasonal.

6.4.2 Fraction Contaminated

The fraction contaminated (FC) represents the fraction of each food product consumed that is contaminated by the chemical at a level consistent with environmental concentrations in the area of concern (e.g., area with maximum deposition rates). Obviously, the most conservative assumption is that all food products consumed (i.e., 100 percent) are from the location represented by the chemical concentrations input into MIRC.

For non-infant children and the adult age cohorts, MIRC includes the default FC of 1, assuming that 100 percent of the food product consumed is produced by households that farm, garden, or raise animals. The user can vary this default FC value for individual food products to tailor the assessment to a particular exposure scenario.

6.4.3 Preparation and Cooking Losses

Food preparation and cooking losses are included in the FFC exposure calculations to account for the amount of a food product as brought into the home that is not ingested due to loss during preparation, cooking, or post-cooking. These losses need to be accounted for in the ADD equations because the food ingestion rates calculated from the USDA 1987 to 1988 NFCS are based on the weight of home grown produce and animal products brought from the field into the house prior to any type of preparation. Not all of the produce or products were eventually ingested. In general, some parts of the produce and products are discarded during preparation while other parts might not be consumed even after cooking (e.g., bones). Thus, the actual food ingested is generally less than the amount brought into the home.

Three distinct types of preparation and cooking losses are included in the ingestion exposure algorithms in MIRC: (1) loss of parts of the food type from paring (i.e., removing the skin from vegetables and fruits) or other types of preparation (e.g., removing pits, coring, deboning), (2) additional loss of weight for the food type during cooking (e.g., evaporation of water), and (3) post-cooking losses (e.g., non-consumption of bones, draining cooking liquid [e.g., spinach]). MIRC includes mean values for these three types of preparation and cooking losses for exposed fruit, protected fruit, exposed vegetables, protected vegetables, root vegetables, beef, pork, poultry, and fish. Different types of losses apply to different types of foods. Therefore, the losses can be represented by only two parameters, *L1* and *L2*, the definitions of which vary according to the food type as explained in the endnotes in Exhibit 6-15. All preparation and cooking loss parameter values were estimated as specified in the Exhibit's endnotes from data presented in Chapter 13 of the EPA's 1997 EFH.

Exhibit 6-15. Fraction Weight Losses from Preparation of Various Foods

Product	Mean Cooking, Paring, or Preparation Loss (Cooking Loss Type 1 [L1]) (unitless) [a]	Mean Net Post Cooking (Cooking Loss Type 2 [L2]) (unitless) [b]
Exposed Fruit [c]	0.244	0.305
Exposed Vegetable	0.162 [d]	NA
Protected Fruit	0.29 [e]	NA
Protected Vegetable	0.088 [f]	NA
Root Vegetable [g]	0.075	0.22
Beef	0.27	0.24
Pork	0.28	0.36
Poultry	0.32	0.295 [h]
Fish [i]	0.0	0.0

Source: EPA 1997a (Chapter 13; Tables 13-5 [meats], 13-6 [fruits], and 13-7 [vegetables])
NA = Not Available
[a] For *fruits*, includes losses from draining cooked forms. For *vegetables*, includes losses due to paring, trimming, flowering the stalk, thawing, draining, scraping, shelling, slicing, husking, chopping, and dicing and gains from the addition of water, fat, or other ingredients. For *meats*, includes dripping and volatile losses during cooking.
[b] For *fruits*, includes losses from removal of skin or peel, core or pit, stems or caps, seeds and defects; may also include losses from removal of drained liquids from canned or frozen forms. For *vegetables*, includes losses from draining or removal of skin. For *meats*, includes losses from cutting, shrinkage, excess fat, bones, scraps, and juices.
[c] These values represent averages of means for all fruits with available data (except oranges) (Table 13-6).
[d] This value represents an average of means for all exposed vegetables with available data (Table 13-7). Exposed vegetables include asparagus, broccoli, cabbage, cucumber, lettuce, okra, peppers, snap beans, and tomatoes.
[e] This value was set equal to the value for oranges (Table 13-6).
[f] This value represents an average of means for all protected vegetables with available data (Table 13-7). Protected vegetables include pumpkin, corn, peas, and lima beans.
[g] These values represent averages of means for all root vegetables with available data (Table 13-7). Root vegetables include beets, carrots, onions, and potatoes.
[h] This value represents an average of means for chicken and turkey (Table 13-5).
[i] If the user changes fish ingestion rates to match a survey of the whole weight of fish brought into the home from the field (divided by the consumers of the fish), an appropriate value for L1 would be 0.3 (EPA 1997a, Table 13-5). For volatile or water soluble chemicals, a non-zero value for L2 also may be appropriate. Although EPA (1997a) recommended 0.11 for L2, it varies substantially by chemical.

There are substantial uncertainties associated with the L1 and L2 parameters, including the wide variation in values across produce types that were averaged together to recommend a central tendency value for each. For example, the L2 factor does not distinguish between weight loss during cooking by water evaporation, which might leave the chemical in the fruit, and pouring the cooking liquid down the drain (chemical lost) or using the liquid to create a sauce (chemical not lost). In addition, the concentration of chemical might be highest in the skin, which often is discarded, and lower in the consumed portion of many bulky fruits and vegetables. Finally, the data EPA used to evaluate L1 included negative losses (i.e., weight gains) due to hydration of dried vegetables (e.g., peas and lima beans), which increases the range of L1 values across different vegetables.

Note that the default L1 and L2 values for fish are set to zero. That is because the data source for the fish ingestion rates is not the USDA's 1987 to 1988 NFCS (USDA 1993, 1994) as reported in EPA's EFH, which reported food as brought into the home, as is the case for the other food categories. Instead, the fish IR data included in MIRC are from a more recent and larger survey, EPA's (2002) analysis of freshwater and estuarine fish consumption from the USDA's 1994-96 and 1998 CSFII. That survey reports ingestion rates of fish parts actually

consumed, and so no loss processes for preparation are needed. The zero value for L2 assumes that no chemical is lost by volatilization and that pan juices are consumed. The user may reset that value where chemical-specific data are available.

If the user manually changes fish ingestion rates to match a local survey of the whole weight of fish brought into the home from the field (divided by number of persons consuming the fish), the user should also set the L1 and L2 parameter to non-zero values. An appropriate value for L1 would be 0.3 (EPA 1997a, Table 13-5). For volatile or water soluble chemicals, a non-zero value for L2 also may be appropriate. Although EPA recommended 0.11 for L2 (EPA 1997a, Table 13-5), it varies substantially by chemical.

6.5 Breast-Milk Infant Exposure Pathway Parameter Values

Values used for parameters in the breast-milk exposure pathway algorithms (Section 3.4) can be scenario-specific, receptor-specific, and/or chemical-specific and might be empirically derived or estimated by an appropriate model. For parameters that are scenario-specific or for which empirical values are required, the default values provided in MIRC are listed. For parameters for which MIRC calculates values, the appropriate equation is listed. Scenario- and receptor-specific parameters are discussed in Section 6.5.1 and chemical-specific parameters are discussed in Section 6.5.2.

6.5.1 Receptor-specific Parameters

Receptor-specific values are needed for parameters that describe the characteristics or activities of the exposed individual. In this context, there are two relevant receptors: the mother and the infant. Exhibit 6-16 lists the parameters and their default values. The text that follows describes the input value or value options for each exposure parameter required by MIRC to calculate the infant absorbed chemical intake rate, or DAI_{inf}. For parameter values that can be estimated when empirical values are not available, see the equation description in Section 3.4.

Exhibit 6-16. Scenario- and Receptor-Specific Input Parameter Values Used to Estimate Infant Exposures via Breast Milk

Parameter	Description	Default Value
AT	Averaging time for infant's exposure via breast milk, i.e., duration of nursing (days)	=ED
BW_{inf}	Body weight of infant (kg) averaged over duration of nursing exposure	7.8
BW_{mat}	Body weight of mother (kg) averaged over duration of mother's exposure	66
DAI_{mat}	Daily absorbed intake of chemical by mother (mg/kg-day)	Equation 3-36
ED	Exposure duration for infant, i.e., duration of breast feeding (days)	=AT
AT/ED	Averaging time divided by exposure duration	1.0
f_{bp}	Fraction of mother's whole blood that is plasma (unitless)	0.65
f_{fm}	Fraction of mother's body weight that is fat (unitless)	0.30
f_{mbm}	Fraction of fat in mother's breast milk (unitless)	0.04
f_{pm}	Fraction of mother's body weight that is plasma (unitless)	0.046
IR_{milk}	Mean infant milk ingestion rate over duration of nursing (kg/day)	0.709
t_{bf}	Duration of breast feeding (days)	365
t_{pn}	Duration of maternal chemical exposure prior to nursing (days)	3285

Averaging time (***AT***) and exposure duration (***ED***). AT refers to the time over which the infant's exposure to the chemical of concern is averaged. *ED* refers to the duration of the infant's exposure. For the exposure scenario considered for this age group, both *AT* and *ED* equal the duration of the nursing period, and they therefore cancel each other out in the infant average daily dose equation.

Infant body weight (***BW_{inf}***). The user selects a value for BW_{inf}, the time-weighted average body weight of the infant over the entire duration of breast feeding, based on the age at which the infant stops breast feeding. For example, if the infant breast feeds for one year, the user should select the body weight for an infant that is averaged from birth to the first birthday. Similarly, if an infant breast feeds for 6 months, the user should select the body weight for an infant that is averaged from birth to six months. Because the default breast feeding duration (t_{bf}) is one year (i.e., 365 days), the default infant body weight is 7.8 kg, which is the time-weighted average for the mean infant body weight between birth and its first birthday from EPA's (2008) *Child Specific Exposure Factors Handbook* (CSEFH; EPA 2008a). Exhibit 6-17 presents additional values for the infant body weight parameter that the user can select instead of the MIRC default.

Exhibit 6-17. Average Body Weight for Infants

Statistic	0 to < 6 months (kg)	0 to < 12 months (kg)	0 to < 18 months (kg)	0 to < 24 months (kg)
Mean	6.5	7.8[a]	9.0	9.6
5th percentile	5.0	6.0	7.0	7.5
10th percentile	5.3	6.4	7.4	7.8
15th percentile	5.5	6.7	7.7	8.2
25th percentile	5.8	7.0	8.1	8.7
50th percentile	6.4	7.8	8.9	9.5
75th percentile	7.1	8.6	9.9	10.5
85th percentile	7.4	9.0	10.3	11.0
90th percentile	7.7	9.2	10.6	11.3
95th percentile	8.0	9.7	11.1	11.8

Source: EPA 2008a; each value is the time-weighted average from the data summaries presented in the CSEFH, Table 8-3.
[a] MIRC default

Maternal body weight (***BW_{mat}***). This parameter represents the body weight of the mother averaged over the entire duration of the mother's exposure to the chemical of concern. The maternal body weight is needed to calculate the biological elimination constant for the lipophilic chemical in lactating women (k_{fat_elac}). MIRC assumes that the mother will be pregnant for 9 months (i.e., 0.75 year) and will be lactating for 1 year. The MIRC default maternal body weight also assumes that the mother has been exposed for 10 years total. For 8.25 years, she is not pregnant or lactating, for 0.75 year she is pregnant, and for 1 year she is lactating. The MIRC default BW_{mat} of 66 kg is based on CSFII data compiled by EPA for non-lactating and non-pregnant women between the ages of 15 and 44 (i.e., women of child-bearing age), lactating women, and pregnant women (EPA 2004b). Exhibit 6-18 presents additional values for the maternal body weight parameter which the user may choose to use instead of the MIRC default. The BW_{mat} value is *not* the value that MIRC uses to estimate the mother's absorbed daily intake (DAI_{mat}). The daily ingestion rates for home-grown/raised food products in MIRC are for men and women combined, with the rates normalized to body weight. The ingestion rates for soil,

water, and fish included in MIRC are not normalized to body weight but are based on both men and women. For those ingestion rates, MIRC uses an average body weight value for males and females to estimate the average daily dose (intake) of the chemical in mg/kg-day. These values are subject to the assumption that the body-weight normalized ingestion rates and resulting ADD values are applicable to nursing mothers.

Exhibit 6-18. Time-weighted Average Body Weight for Mothers

Statistic	Weight (kg)
Mean	66.0[a]
5th	47.1
10th	50.2
25th	54.3
50th	62.0
75th	72.0
90th	85.7
95th	97.0

Source: EPA 2004b
[a] MIRC default value

Exposure duration (*ED*). See discussion of *AT* and *ED* above.

Fraction of mother's whole blood that is plasma (f_{bp}). Steinbeck (1954) reported that plasma volume accounts for approximately 60 percent of the total blood volume in non-lactating human females (EPA 1998). Harrison (1967) and Ueland (1976) reported plasma volumes between 63 to 70 percent in postpartum women (EPA 1998). The default value in MIRC of 65 percent (0.65) is the value recommended by EPA in its *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions* (MPE, EPA 1998).

Fraction of mother's body weight that is fat (f_{fm}). A limitation of using a steady-state, instead of a dynamic partitioning, model for lactational transfer of chemicals is that several key parameters change over the course of exposure. For example, Equation 3-38, used to estimate the concentration of a lipophilic chemical in breast milk fat, assumes that the mother's body fat will remain constant over the entire duration of breast feeding (t_{bf}), which is unlikely to be true (EPA 2001a). Another limitation of the single analytic model is that chemical transfer rates from blood to milk are unlikely to be the same as the rate of mobilization of the chemical from fat stores to the blood (EPA 2001a). Studies cited in ATSDR's toxicological profile for chlorinated dibenzo-p-dioxins show a correlation between percent body fat and the elimination rate of dioxins, with longer half-lives for dioxins in individuals with a higher proportion of fat in their bodies (ATSDR 1998). In the context of a screening model, however, EPA recommends a default value for the fraction of a mother's body comprised of fat of 0.3 based on data and discussions presented by Smith (1987) and Sullivan et al. (1991) (EPA 1998). A fraction of 0.3 indicates that 30 percent of the mother's body weight is fat, which is a conservative value (EPA 2001a). To establish a conservative screening scenario, the MIRC default value for f_{fm} is 0.30**.**

Fraction of fat in mother's breast milk (f_{mbm}). The $C_{milkfat}$ model (Equation 3-38) assumes that a constant fraction of breast milk is fat, even though there is evidence that indicates variation in the fat content of breast milk throughout lactation (Sim and McNeil 1992). Different studies suggest a fat content of breast milk in humans of between 1 and 5 percent (Jensen 1987, Schecter et al. 1994, Hong et al. 1994, McLachlan 1993, Bates et al. 1994, NAS 1991, Butte et al. 1984, Maxwell and Burmaster 1993, EPA 1997a, Smith 1987, Sullivan et al. 1991). The

MIRC default value for f_{mbm} of 0.04 (i.e., 4 percent) is the value EPA recommended for MPE (EPA 1998).

Fraction of maternal weight that is plasma (f_{pm}). Altmann and Dittmer (1964) estimated that plasma volume for adult women ranged from 37 to 60 mL/kg of body weight and averaged about 45 mL/kg. Ueland (1976) observed that the average plasma volume of women 6 weeks postpartum was 45 mL/kg of body weight. Using a value of 1.026 for the specific gravity of plasma from Conley (1974), EPA estimated a value of 0.046 for the fraction of maternal weight that is plasma (EPA 1998). The MIRC default for f_{pm} therefore is 0.046.

Infant breast milk ingestion rate (IR_{milk}). Milk ingestion rates vary with several factors, including the age and size of the infant and use of other foods such as formula. Based on its review of a several studies, EPA recommended time-weighted average and upper percentile milk ingestion rates for infants that nurse for six and for twelve months (EPA 1997a, Chapter 14, Table 14-15). To estimate an "average" value, EPA first estimated study-sample-size weighted average values for 1, 3, 6, 9, and 12 months of age and then developed time-weighted average milk ingestion rates from those (EPA 1997a). EPA estimated an upper percentile (upper bound) value as the mean plus two standard deviations. MIRC converts the ingestion rates measured volumetrically (mL/day) to mass-based estimates (kg/day) assuming the density of human milk to be 1.03 g/mL (reported by NAS 1991 and recommended by EPA 1997a). The resulting values are shown in the first two rows of Exhibit 6-19. The MIRC screening-level default of 980 mL/day is an upper-bound estimate based on a one-year nursing period.

Exhibit 6-19 also includes the recommended values for four non-overlapping age categories from the CSEFH (EPA 2008, Table 15-1). The values demonstrate that although infants grow substantially from birth to one year of age, the "upper bound" estimates of their milk ingestion rates are very close to 1 liter per day at all stages of development in the first year.

Exhibit 6-19. Infant Breast Milk Intake Rates

Age Category	Average (mL/d)	Average (kg/d)	"Upper Bound" (mL/d)	"Upper Bound" (kg/d)	Reference
1 to 6 months	742	0.764	1,033	1.064	EPA 1997a[†]
0 to < 12 months	688	0.709	980[a]	1.01[a]	EPA 1997a[†]
0 to < 1 month	510	0.525	950	0.979	EPA 2008[††]
1 to < 3 months	690	0.711	980	1.01	EPA 2008[†]
3 to < 6 months	770	0.793	1,000	1.03	EPA 2008[†]
6 to < 12 months	620	0.639	1,000	1.03	EPA 2008[†]

[a] MIRC default; [†] Based on review of multiple studies; [††] Based on a single study

Duration of breast feeding (t_{bf}). This parameter is equal to the infant's exposure duration (*ED*) and the infant's averaging time (*AT*). In its MPE Methodology, EPA asserts a conservative value for the duration of breast feeding of 1 year (i.e., 365 days) and a central tendency estimate of 6 months (180 days) (EPA 1998). Reviewers of MPE noted that 365 days may be overly conservative, given than only 20 percent of infants are breast fed for 6 months, at which point alternative foods are introduced, at least in addition to breast milk (EPA 2001a). Nonetheless, to establish a conservative screening scenario, the MIRC default for t_{bf} is 365 days.

Duration of the mother's exposure to the chemical of concern prior to nursing (***t_{pn}***). The model shown as Equation 3-38 includes this parameter to reduce the over-estimate of chemical concentration in milk fat that occurs if the model is applied to a chemical with a long biological half-life (e.g., many years). The factor is needed for applications of the model to scenarios with a brief exposure duration (e.g., beginning a few months prior to the start of nursing) relative to the chemical half life. As the duration of an exposure scenario increases to meet and exceed the chemical half life, however, the overestimate that occurs without this parameter is reduced. For example, assume a chemical biological half-life of 8 years and a nursing period of 1 year. If exposure of the mother starts at the beginning of nursing, using Equation 3-38 without the t_{pn} term results in an over-estimate of the concentration of the chemical in breast milk by a factor of 28.1 compared with the prediction using Equation 3-38 with the t_{pn} term (EPA 1998, Table 9-6). However, at longer pre-natal exposures of the mother, the magnitude of the over-estimate is reduced: for a 10-year exposure, the magnitude of the overestimate without the t_{pn} term is 2.28, and for a 30-year exposure, the overestimate is reduced to 1.39.

For purposes of the screening-level of assessment for dioxins, we assume an exposure duration equal to the half-life of the chemical, or 10 years. Only 3285 days of that period are pre-natal (i.e., 3650 minus 365 days, assuming 1 year lactation period). Although longer exposure periods are possible for the screening scenario, there is sufficient uncertainty in the model to merit accepting a conservative bias for this parameter value.

6.5.2 Chemical-Specific Parameter Values

The chemical-specific parameters in the breast-milk pathway in MIRC are listed in Exhibit 6-20. Note that the parameters for which values are needed are different for the lipophilic chemicals (i.e., dioxins), for which lactational transfer is assumed to occur via milk fat, and inorganic chemicals, for which the transfer is assumed to occur via the aqueous phase of breast milk (i.e., mercury).

Absorption efficiency of the chemical by the oral route of exposure for the infant (***AE_{inf}***). The models included in MIRC assume that the AE_{inf} from the lipid phase of breast milk is equal to the AE_{inf} from the aqueous phase of the milk. Reviewers of the model stated that this assumption may not be valid and that ideally, the equation DAI_{inf} would include variables for the AE_{inf} from the breast milk fat and the AE_{inf} from the aqueous phase of breast milk (EPA 2001a). However, since the MIRC assumption is that chemicals will partition to either the lipid or aqueous phase of milk, it is not necessary at this time to have multiple AE_{inf} values for a given chemical. If data on the AE from the mother or an adult but not for the infant are available, data for the adult may be used for AE_{inf}. Reviewers also recommended that chemical-specific values come from studies that account for absorption of the chemical from milk, because absorption from other matrices (e.g., solid foods) may not be relevant (EPA 2001a). If chemical-specific data are not available for adults or infants, a conservative default value for AE_{inf} for a screening level assessment is 1.0, which assumes 100 percent absorption (EPA 1998).

The default value for AE_{inf} in MIRC for both MeHg and dioxin is 1.0. For ingested lipophilic chemicals, it is reasonable to assume that absorption will be high (EPA 2004c). ATSDR (1998) reported that dioxins are well absorbed by the oral route of exposure, with one human experiment indicating more than 86 percent absorption. It is EPA policy to assume 100 percent absorption for chemicals with reported *AEs* of 50 percent or higher (EPA 2004c). MeHg also is well absorbed, with measured values as high as 95 percent, and so a value of 100 percent is used in MIRC (EPA 2001b).

Exhibit 6-20. Chemical-specific Input Parameter Values for Breast Milk Exposure Pathway

Parameter and Description		2,3,7,8-TCDD	MeHg
AE_{inf}	Infant absorption efficiency of the chemical by the oral route of exposure (i.e., fraction of ingested chemical that is absorbed by the infant; unitless)	1.0 (default)	1.0 (default)
AE_{mat}	Maternal absorption efficiency of the chemical by the oral route of exposure (i.e., fraction of ingested chemical that is absorbed by the mother; unitless)	1.0 (default)	1.0 (default)
f_{bl}	Fraction of steady-state total body burden of hydrophilic chemical in mother that is in the mother's whole blood compartment (unitless)	NA	0.059 (Kershaw et al. 1980) [a]
f_f	Fraction of steady-state lipophilic chemical body burden in mother that is stored in body fat (unitless)	≥ 0.90 (ATSDR 1992)	NA
f_{pl}	Fraction of steady-state total hydrophilic chemical body burden in mother that is in the blood plasma compartment (unitless)	NA	Not yet identified [b]
h	Biological half-life for chemical in non-lactating women (days)	3650 (EPA 1994c)	50 (Sherlock et al. 1984)
k_{aq_elac}	Rate constant for total elimination of hydrophilic chemicals by lactating women (per day)	NA	= k_{elim}
k_{elim}	Rate constant for elimination of chemical for non-lactating women (per day; related to chemical half-life)	1.9E-04[b]	1.4E-02 [c]
k_{fat_elac}	Rate constant for total elimination of lipophilic chemicals by lactating women (per day)	Est. using Equation 3-41	NA
Pc_{bm}	Partition coefficient for hydrophilic chemical between maternal *blood plasma* and aqueous phase of breast milk (g milk/g plasma; model assumption)	NA	1.0 (model assumption)
Pc_{RBC}	Partition coefficient for hydrophilic or protein-bound chemical between *red blood cells* (RBC) and *plasma* in maternal blood (mL whole blood/mL RBC)	NA	40 (Hollins et al. 1975)

NA = not applicable. ND = not yet determined from literature.
[a] This value is based on a single-dose study and may not be appropriate for a chronic exposure model.
[b] An empirical value for this variable is currently missing for application of model.
[d] This value was calculated from biological half-life (*h*) using Equation 3-40.

Note that *AE* values for some inorganic compounds are substantially less than 1.0. For cadmium, for example, *AEs* for adults of 0.025 to 0.05 have been reported (EPA 2004c, Exhibit B-4).

Absorption efficiency of the chemical by the oral route of exposure for the mother (***AE_{mat}***). The default value for both dioxins and MeHg is 1.0, as described in the previous paragraph.

Fraction of total maternal chemical body burden that is in the whole blood (***f_{bl}***). The default value for MeHg in MIRC, 0.059, is from Kershaw et al. (1980), which reported kinetics of blood deposition and clearance of MeHg in humans. Individuals consumed one meal of fish that contained between 18 and 22 µg Hg/kg body weight. The fraction of the dose deposited in the blood volume after mercury was fully distributed in tissues was 5.9 percent or 0.059. This study used a single-dose and thus may not be appropriate for a chronic exposure analysis.

Fraction of total maternal chemical body burden that is in body fat (***f_f***). Based on ATSDR's *Toxicological Profile for Selected PCBs* (ATSDR 1992) and Sullivan et al. (1991), EPA concluded that the "fraction of ingested contaminant stored in fat may be >90%" for lipophilic chemicals such as PCBs and dioxins (EPA 1998). This statement was interpreted to mean that 90 percent of the maternal body burden of chemical at "steady state" is located in body fat for dioxins at steady state.

Fraction of total maternal chemical body burden that is in blood plasma (***f_{pl}***). For hydrophilic chemicals, this parameter represents the steady-state fraction of the total chemical in the body that is circulating in the blood plasma. Values for f_{pl} may be available for some chemicals in the scientific literature. No value for this parameter for methyl mercury has been identified from the literature at this time. A value can be calculated using Equation 3-43. However, this equation requires a reliable value for f_{bl}, and the value found for mercury may not be appropriate for a chronic exposure analysis (see above).

Chemical half-life in non-lactating women (***h***). In general, highly lipophilic chemicals tend to have relatively long biological half-lives. EPA estimates that the half-life for dioxins is between 7 and 10 years (EPA 1994a). ATSDR estimates that the half-life for 2,3,7,8-TCDD in particular may be as long as 12 years (ATSDR 1998). To establish a conservative screening scenario, the MIRC default half-life for dioxins is set to 10 years or 3650 days.

The half-life for methylmercury is on the order of weeks, not years. Greenwood et al. (1978) measured blood clearance rates for MeHg in lactating Iraqi women exposed accidentally to MeHg via bread prepared from wheat treated with a fungicide that contained MeHg. The data indicated a mean half-life for MeHg of approximately 42 days. Sherlock et al. (1984) reported an average measured half-life for MeHg of 50 days with a range of 42-70 days. The MIRC default for MeHg is set to the longer average half life of 50 days.

Chemical elimination rate constant for lactating women – aqueous (***k_{aq_elac}***). The parameter k_{aq_elac} is equal to k_{elim} plus the loss rate for the chemical in the aqueous phase of breast-milk during lactation. EPA has yet to propose a term for the additional elimination of a chemical in the aqueous phase of milk from breast feeding. In the absence of empirical values, a reasonable assumption for water soluble chemicals is that k_{aq_elac} is equal to k_{elim} as discussed for Equation 3-43. The extent to which k_{elim} is an underestimate of k_{aq_elac} for a given chemical will determine the extent of conservative bias in k_{aq_elac}.

Chemical elimination rate constant for non-lactating women (***k_{elim}***). Although values for this parameter often are reported directly in the literature, MIRC estimates k_{elim} from chemical half-life assuming first-order kinetics as shown in Equation 3-40. For example, for a biological half-life of 3,650 days for dioxins, k_{elim} is estimated to be 1.9E-04 per day. Assuming a biological half-life of 50 days for MeHg, the value for k_{elim} is estimated to be 0.014 per day.

Rate constant for total elimination of lipophilic chemicals by lactating women (***k_{fat_elac}***). Although values for this parameter might be found in the scientific literature for some chemicals, in MIRC, k_{fat_elac} for dioxins is calculated from Equation 3-41. When the parameters in that equation are set to the default values in MIRC for dioxins, MIRC estimates a value of 0.0015 per day for k_{fat_elac}.

Partition coefficient for chemical between maternal blood plasma and aqueous phase of breast milk (***Pc_{bm}***). The aqueous model, presented in Equation 3-42, assumes that the concentrations in the plasma and aqueous phase of breast milk are directly proportional (EPA 1998). Therefore, the default value for this parameter for MeHg in MIRC is 1.0.

Partition coefficient for chemical between red blood cells and plasma in maternal blood (Pc_{RBC}). Chemical-specific values for this parameter should be located in the scientific literature. If chemical-specific values are unavailable and it is assumed that there is equal distribution of the chemical in the plasma and red blood cells, EPA suggests a default value of 1.0 (EPA 1998). For MeHg, MIRC includes a value of 40 based on Hollins et al. (1975) study of cats exposed to MeHg, which reported a ratio of radio-labeled mercury in red blood cells to plasma of 97.7 to 2.3 (i.e., ratio of 42.5).

7 Summary of MIRC Default Exposure Parameter Settings

The default settings included in MIRC are intended to be characteristic of a conservative (but plausible) exposure scenario that results in a negligible or extremely low chance of underestimating risk to farming households in an area with chemical concentrations and air deposition rates as specified by the user. These default parameter values were used to derive the *de minimis* emission rates used for screening emissions of PB-HAPs from sources included in RTR risk assessments. These values are the default, or initial setting, for parameter values in MIRC as described in Section 6. This section summarizes the default parameter values used to calculate screening thresholds.

This chapter is organized to present the chemical- and scenario-specific inputs to MIRC by data type. The screening-level analysis uses 90th percentile ingestion rates, presented in Section 7.1, and population-specific characteristic assumptions, presented in Section 7.2, that are generally conservative in nature. *De minimis* thresholds were derived for five RTR chemical species: benzo(a)pyrene, cadmium, mercuric chloride, methyl mercury, and 2,3,7,8-TCDD; Section 7.3 presents chemical-specific parameter inputs for these five chemicals. Finally, Section 7.4 presents default parameter values for the nursing infant exposure scenario, which applied only to dioxin and methyl mercury.

7.1 Default Ingestion Rates

The screening-level (or default) values for ingestion rates for soil, fish, breast milk, and for each farm food type are equal to the 90th percentile of the distribution of national data for that ingestion medium (Exhibit 7-1). The default settings also assume that all food types are obtained from the area of chemical deposition specified by the user (i.e., fraction of food from contaminated area = 1.0).

For estimates of *de minimis* emission rates for PB-HAPS, environmental concentrations and air deposition rates were estimated using TRIM.FaTE for the area of maximal deposition in the vicinity of a hypothetical facility, and thus represent risks estimated for a maximally exposed individual/farm/family.

Exhibit 7-1 also includes a sum of the 90th percentile ingestion rates for homegrown food categories and fish ingestion (preceding rows) to show the implied total food ingestion rate associated with setting multiple food-type-specific ingestion rates at a 90th percentile. Because the 90th percentiles for each farm food category are likely to reflect different individuals, it is likely that addition of multiple 90th percentile intake values will exceed the total food ingestion rates likely for the general population.

The final row in Exhibit 7-1 lists the likely magnitude of the overestimates by age category. The preceding row includes the 90th percentile of the distribution of *individual total food ingestion rates* from the USDA's 1994-96 and 1998 *Continuing Survey of Food Intakes by Individuals* (CSFII) (USDA 2000) data sets, as analyzed by EPA (EPA 2005e). The total ingestion rate for the farming households takes into account the cooking losses typical of each food category to provide a better comparison with the 90th percentile individual total food ingestion rates (which are based on consumption of prepared foods).

Exhibit 7-1. Farm Food Category Ingestion Rates for Conservative Screening Scenario for Farming Households

Product	90th Percentile Consumer Ingestion Rate						Units [a]
	Infants < 1 yr	Child 1-2 yrs	Child 3-5 yrs	Child 6-11 yrs	Child 12-19 yrs	Adult	
Farm Food Item							
Beef	NA	4.5	6.7	11.4	3.53	5.39	g/kg-day
Dairy [c]	NA	148	82	54.7	27.0	34.9	g/kg-day
Eggs [c]	NA	5.1	2.8	1.80	1.34	1.65	g/kg-day
Exposed Fruit	NA	3.7	5.4	6.98	3.41	5	g/kg-day
Exposed Vegetable	NA	10.7	3.5	3.22	2.35	6.01	g/kg-day
Pork [c]	NA	4.5	4.4	3.04	2.65	3.08	g/kg-day
Poultry [c]	NA	7.4	6.8	4.58	3.28	3.47	g/kg-day
Protected Fruit [c]	NA	53	36	24.1	16.2	15.1 [b]	g/kg-day
Protected Vegetable	NA	3.9	2.5	2.14	1.85	3.55	g/kg-day
Root Vegetable	NA	7.3	4.3	3.83	2.26	3.11	g/kg-day
Other							
Breast milk[d]	1.01	NA	NA	NA	NA	NA	kg/day
Soil (dry)	NA	400[e]	400[e]	201 [f]	201 [f]	201 [f]	mg/day
Water	NA	654	834	980	1537	2224	mL/day
Fish (per individual) [g]	NA	3.24	4.79	6.9	8.95	17	g/day
Fish (per kg BW) [h]	NA	0.26	0.26	0.22	0.14	0.24	g/kg-day
Total Food Ingestion Rates for Comparison Only (not in MIRC; excludes soil and water)							
Total Food: Homegrown only [i]	NA	219	131	95	52	67	g/kg-day
Total Food: All Sources [j]	NA	125	91	61	34	23.7	g/kg-day
Overestimate (ratio of Homegrown/Total) [k]	NA	1.8	1.4	1.6	1.5	2.8	(unitless)

Sources: EPA 1997a (Chapter 13), unless otherwise noted.
NA = not applicable

[a] As indicated by the units, the *ingestion rates* for produce and animal products are already normalized to consumer body weight. *Ingestion rates* for soil (mg/day) and water (mL/day) are not normalized to body weight. Soil is reported as dry weight, water as volume, and the remaining values on a wet-weight basis.

[b] This value represents a weighted average for the 20-39 and 40-69 age groups.

[c] For several farm food categories, ingestion rates were not available in EPA's 1997 EFH or 2008 CSEFH (EPA 1997a, 2008a). Ingestion rates for these child age categories were calculated using the methodology recommended in HHRAP (EPA 2005a), Section 6.2.2.2, as described in Section 6.3.3 of this document. Sources to develop these values included EPA 1997a and EPA 2003c.

[d] Infants are assumed to consume only breast milk for one year.

[e] This value represents an estimated "upper percentile" for children (EPA 1997a).

[f] These values represent soil ingestion rates for individuals who consume homegrown food products from Stanek et al. 1997.

[g] 90th percentile adult fish ingestion rates are based on data from 1995-1996 and 1998 CSFII as summarized in EPA 2002; child fish ingestion rates are based on the same survey data, but estimated by multiplying average two-day consumption rate for children who consumed fish on one or both days of the survey by the frequency of fish consumption (i.e., proportion of children that reported consuming fish out of all children sampled).

[h] Fish ingestion rates, original data in g/day, have been normalized to body weight in this table to allow addition into total food estimate using the mean body weight for each age category.

[i] Sum of 90th percentile post-cooking food food ingestion rates. This estimate is calculated by multiplying the food ingestion rates on previous rows (excluding soil and water) by $(1-L_1)x(1-L_2)$, where L_1 and L_2 are the loss rates from Exhibit 6-15. The rows are then summed to get the total post-cooking ingestion rate.

[j] 90th percentile total food intake rates from EPA 2008a and 2005e based on CSFII data 1994-96 and 1998; see Exhibit 6-14 of this document.

The final row of Exhibit 7-1 is the ratio of the two preceding rows. The magnitude of the overestimate that results from adding 90th percentile values for 10 different categories of homegrown foods for children is a factor of 1.8. The overestimate of the 90th percentile total food ingestion rate for adults is larger, a factor of 2.8. This bias may be considered when evaluating the cancer risks and noncancer hazard quotients estimated by MIRC.

7.2 Default Screening-Level Population-Specific Parameter Values

The screening-level values for body weights (BWs) for the RTR *de minimis* analysis, which serve as the default values in MIRC, are mean values and are presented in Exhibit 7-2. As stated in Section 6, EPA recommends using the mean BW for each age group when using upper (90th) percentile values for medium ingestion rates. Use of the mean body weights introduces no bias toward over- or underestimating risk.

Exhibit 7-2. Mean Body Weight Estimates for Adults and Children [a]

Lifestage (years)	Duration (years)	Mean Body Weight (kg)
Adult [b] (20-70)	50	71.4
Child < 1 [c]	1	7.83
Child 1-2 [c]	2	12.6
Child 3-5 [d]	3	18.6
Child 6-11 [e]	6	31.8
Child 12-19 [f]	8	64.2

[a] Sources: EPA 1997, 2008a

[b] These values were calculated from data presented in EPA's (1997a) *Exposure Factors Handbook*.

[c] These values were calculated as time-weighted average body weight (BW) from data presented in Table 8-3 of EPA's (2008a) *Child-Specific Exposure Factors Handbook* (CSEFH).

[d] These values were obtained directly from Table 8-3 of the 2008 CSEFH.

[e] These values were obtained directly from Table 8-3 of the 2008 CSEFH for age group 6 to <11 years. The values represents a slight underestimate of BW for ages 6 through 11 years, since 11-year olds are not included in this CSEFH age group.

[f] These values were calculated as time-weighted average BW for age groups 11 to <16 years and 16 to <21 years from Table 8-3 of the 2008 CSEFH. The direction of the possible bias is unknown. The values match the estimate based on Table 8-22 of the NHANES IV data as presented by Portier et al. (2007).

7.3 Default Chemical-Specific Parameter Values for Screening Analysis

Exhibit 7-3 presents chemical-specific parameter values for input to MIRC for the screening-level analysis. Values for reference dose (RfD), cancer slope factor (CSF), bioavailability when ingested in soil (*Bs*), mammalian metabolism factors (*MF*), correction factors for belowground produce ($VG_{rootveg}$), wet deposition fractions (*Fw*), air to plant transfer factors (Bv_{AG}), root concentration factors (*RCF*), and soil-water partition coefficient (*Kds*) are presented in Exhibit 7-3.

Exhibit 7-3. Chemical-Specific Parameter Values for Input to MIRC [a]

Parameter	Description	Benzo(a)-pyrene	Cadmium	Mercuric chloride	Methyl mercury	2,3,7,8-TCDD	Units
RfD	Reference Dose for Chemical	0	0.0005	0.0003	0.0001	1E-09	mg/kg-day
CSF	Oral carcinogenic potency slope factor for chemical	10	0	0	0	150,000	mg/kg-day
Bs	Soil bioavailability factor for livestock	1	1	1	1	1	unitless
MF	Mammalian metabolism factor	0.01	1	1	1	1	unitless
$VG_{rootveg}$	Empirical correction factor for belowground produce, i.e., tuber or root vegetable, to account for possible overestimate of the transfer of chemicals from the outside to the inside of bulky tubers or roots (based on carrots and potatoes)	0.01	1	1	0.01	0.01	unitless
Fw	Fraction of wet deposition that adheres to plant surfaces; 0.2 for anions, 0.6 for cations and most organics	0.6	0.6	0.6	0.6	0.6	unitless
Bv_{AG}	Air-to-plant biotransfer factor for aboveground produce for vapor-phase chemical in air	124,742	0	1,800	0	65,500	[mg/g produce DW] / [mg/g air]
RCF	Chemical-specific root concentration factor for tubers and root produce	9,684	0	0	0	39,999	L soil pore water/kg root WW
Kds	Chemical-specific soil/water partition coefficient	160,000	75	58,000	7,000	38,904.51	L soil pore water/kg soil DW

[a] Values presented in this Exhibit are previously presented in Exhibit 4-1, Exhibit 6-2, **Error! Reference source not found.**, and Exhibit 6-5. However, exact values used in the analysis are presented here, rather than values restricted by significant figures.

Only single estimates were developed for each of these parameters for HHRAP (EPA 2005a), and the potential direction and magnitude of bias toward over- or underestimating risks were not investigated in this analysis. The inputs that are both chemical-specific and plant-type-specific, as presented in **Error! Reference source not found.**, are *not* repeated here. Again, only single estimates were developed for these parameters and the potential direction and magnitude of bias toward over- or underestimating risks were not investigated. Finally, Exhibit 7-4 presents biotransfer factors for each of the chemicals and animal types assessed in the screening level assessment.

Exhibit 7-4. Chemical and Animal-Type Specific Biotransfer Factor (Ba) values for Input to MIRC

([mg chemical/kg WW tissue or dairy] / [mg chemical intake/day] = day/kg WW tissue or dairy)

Chemical	Beef	Dairy	Pork	Poultry	Eggs
Benzo(a)pyrene	0.03756242	0.00790788	0.0454703	0.02767758	0.01581576
Cadmium	0.00012	0.0000065	0.00019149	0.10625	0.0025
Mercuric chloride	0.000105	0.00000143	0.00003393	0.023925	0.023925
Methyl mercury	0.00124	0.0000169	0.00000507	0.003575	0.003575
2,3,7,8-TCDD	0.02612123	0.00549921	0.03162044	0.01924722	0.01099841

Note: Exact values used in the analysis are presented here, rather than values restricted by significant figures.

Sensitivity analyses were conducted to identify which, if any, of these parameter values significantly influence risk estimates from MIRC; results are presented elsewhere. Further evaluation of the possible range and distribution of values can be conducted for any parameter that appears important to the model outputs.

7.4 Screening-Level Parameter Values for Nursing Infant Exposure

EPA also included an assessment of risk to nursing infants exposed to dioxins and to methylmercury (MeHg) in their mother's milk for a family farming and catching fish in the area of maximal air deposition of chemical. Input values were summarized in Section 6.5.

7.4.1 Dioxins

For dioxins, chemical intake via breast milk by nursing infants was estimated using the model presented in EPA's *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions* (MPE, EPA 1998a). The assumption that lactational transfer of dioxins to the infant occurs via the lipid-phase of milk appears reasonable. The following screening-level assumptions used in that model should bias the results toward some overestimate of risks.

- Duration of nursing is a full year and no other foods or liquids are consumed by the infant; a more "typical" value would be six months.
- Absorption efficiency of dioxin in food or milk by mother and infant are 100 percent; this assumption might overestimate absorption but probably by no more than 15 percent.
- The fat content of human milk is assumed to be 4 percent, a value toward the high end of the reported range of values (1 to 5 percent).
- The maternal chemical intake is estimated using 90th percentile ingestion rates for the different homegrown foods (see discussion for Exhibit 7-1); this assumption might overestimate total ingestion of homegrown foods by a factor of more than 3.

- If the fraction of the maternal body burden of dioxin that is in the body fat compartment is greater than 90 percent, as suggested by ATSDR (1998), then actual exposures of the infant may be less than estimated.

There also are parameter values and model assumptions for the lipid-phase breast-milk pathway for which possible bias is unknown.

- The accuracy of the model is unknown; it has not been verified or validated with empirical data.
- Using a half-life of 10 years for dioxins may over- or under-estimate risks.

Finally, there is one assumption that might possibly introduce some bias toward underestimating risks. The model results are sensitive to the biological half-life of the chemical in the mother relative to the length of her exposure prior to the lactation period. Using an exposure duration for the mother equal to the assumed half-life for dioxins, 10 years, may underestimate the duration of exposure of the mother.

7.4.2 Methyl Mercury

For MeHg, empirical data from a single human study (Fujita and Takabatake 1977) was used in conjunction with a physiologically based pharmacokinetic (PBPK) model of lactational transfer of MeHg developed and partially validated by Byckowski and Lipscomb (2001) to support a very simple predictive model. Both the human data and the PBPK model indicated that for relatively low MeHg exposures, the concentration of MeHg in the nursing infant's blood is similar to its concentration in the mother's blood. The PBPK model suggested in addition that the average daily dose of MeHg absorbed from milk by the nursing infant (DAI_{inf}) is indistinguishable from the dose of MeHg absorbed by its mother from her food (DAI_{mat}). The data are limited, and the model includes various assumptions; however, there is no known directional bias in the estimates.

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ATTACHMENT C-3: Systematic Sensitivity Analysis Variables and Results

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TABLE OF CONTENTS

1 Systematic Sensitivity Analysis Variables and Results

This attachment provides the tables of the variables included in the systematic sensitivity analysis in Exhibit 1. The variables are organized into three categories: TRIM.FaTE variables, MIRC farm food chain variables, and MIRC ingestion and bodyweight variables.

This attachment also provides detailed elasticities and rankings for the variables with the highest elasticities for Benzo[a]Pyrene (Exhibit 2), 2,3,7,8 – TCDD (Exhibit 3), cadmium (Exhibit 4), divalent mercury (Exhibit 5), and methyl mercury (Exhibit 6). In each case, the elasticities and rankings are provided for the local systematic sensitivity analysis (variables perturbed up and down by 5%) and the range systematic sensitivity analysis (variables perturbed up and down by 50%).

Exhibit 1-1. Variables Included in the Systematic Sensitivity Analysis

Variable Name	Variable Description
TRIM.FaTE Variables	
AirTemp	Ambient Air Temperature
AlgaeCarbonCont	Carbon Content of Algae in Pond
AlgaeDensity	Density of Algae in Pond
AlgaeGrowthRate	Growth Rate of Algae in Pond
AlgaeRadius	Radius of Single Algae in Pond
Biomass	Biomass in Pond for all Aquatic Species
ChlorideConc	Water- Chloride Concentration
ChlorophyllConc	Water- Chlorophyll Concentration
EmissionRate	Emission Rate of all PBHAPs
EroRate	Erosion Rate for all Parcels
FishMass	Fish Body Weight for all Aquatic Species
HorizWindSpeed	Horizontal Wind Speed
MixHeight	Mixing Height
Rain	Annual Rainfall
RootSoilAir	Root Soil- Fraction Air
RootSoilOCC	Root Soil- Organic Carbon Fraction
RootSoilpH	Root Soil- pH
RootSoilSand	Root Soil- Fraction Sand
RootSoilVertVel	Root Soil- Average vertical velocity of water (percolation)
RootSoilWat	Root Soil- Fraction Water
RunoffRate	Total Water Runoff Rate
SedDepVel, SedRusVel	Sediment Deposition Velocity and Resuspension Velocity
SedOCC	Sediment- Organic Carbon Fraction
SedpH	Sediment- pH
SedPorosity	Sediment Porosity
SedSand	Sediment- Fraction Sand
StackHeight	Stack Height
SurfSoilAir	Surface Soil- Fraction Air
SurfSoilOCC	Surface Soil- Organic Carbon Fraction
SurfSoilpH	Surface Soil- pH
SurfSoilSand	Surface Soil- Fraction Sand
SurfSoilVertVel	Surface Soil- Average vertical velocity of water (percolation)
SurfSoilWater	Surface Soil- Fraction Water
SurfWatTemp	Water Temperature
WatFractSand	Water- Fraction Sand
WatOCC	Water- Organic Carbon Fraction
WatpH	Water- pH
WatRetTime	Rentention Time in Pond
WatSuspSed	Water- Suspended Sediment Concentration

Exhibit 1-1. Variables Included in the Systematic Sensitivity Analysis

Variable Name	Variable Description
MIRC Farm Food Chain Variables	
100-MAF_ef	100 - Moisture adjustment factor, Exposed Fruit
100-MAF_ev	100 - Moisture adjustment factor, Exposed Vegetable
100-MAF_pf	100 - Moisture adjustment factor, Protected Fruit
100-MAF_pv	100 - Moisture adjustment factor, Protected Vegetable
100-MAF_rv	100 - Moisture adjustment factor, Root Vegetable
Ba_beef	PBHAP-specific biotransfer factor, Beef
Ba_dairy	PBHAP-specific biotransfer factor, Dairy
Ba_egg	PBHAP-specific biotransfer factor, Eggs
Ba_pork	PBHAP-specific biotransfer factor, Pork
Ba_poultry	PBHAP-specific biotransfer factor, Poultry
Br_AG_produce_ef	Plant-soil PBHAP bioconcentration factor, Exposed Fruit
Br_AG_produce_ev	Plant-soil PBHAP bioconcentration factor, Exposed Vegetables
Br_AG_produce_fo	Plant-soil PBHAP bioconcentration factor, Forage Feed
Br_AG_produce_gr	Plant-soil PBHAP bioconcentration factor, Grain Feed
Br_AG_produce_pf	Plant-soil PBHAP bioconcentration factor, Protected Fruit
Br_AG_produce_pv	Plant-soil PBHAP bioconcentration factor, Protected Vegetable
Br_AG_produce_si	Plant-soil PBHAP bioconcentration factor, Silage Feed
Br_AG_rootveg	Plant-soil PBHAP bioconcentration factor, Root Vegetables
Bs [a,b]	Soil bioavailability factor for livestock
Bv_AG	Air-to-plant biotransfer factor for aboveground produce for vapor-phase PBHAP in air
C_FishT3	Concentration of PBHAP in whole fish for T3 fish
C_FishT4	Concentration of PBHAP in whole fish for T4 fish
C_Soil	Concentration of PBHAP in soil from contaminated area
Ca	Average annual total PBHAP concentration in air
Cs_root_zone_feed	Average soil concentration in contaminated area used to grow animal feed
Cs_root_zone_ produce	Average PBHAP concentration in soil at root-zone depth in produce-growing area
Cs_S_animal_ ingest	PBHAP concentration in surface soil in contaminated area where livestock feed
Drdp	Average annual dry deposition of particle-phase PBHAP
Drwp	Average annual wet deposition of particle-phase PBHAP
Fv [b,c]	Fraction of airborne PBHAP in vapor phase
Fw	Fraction of wet deposition that adheres to plant surfaces
Kds	PBHAP-specific soil/water partition coefficient
Kp_ef	Plant surface loss coefficient, Exposed Fruit
Kp_ev	Plant surface loss coefficient, Exposed Vegetable
Kp_fo	Plant surface loss coefficient, Forage
Kp_si	Plant surface loss coefficient, Silage
L1_beef	Loss type 1, Beef
L1_ExpFruit	Loss type 1, Exposed Fruit
L1_ExpVeg	Loss type 1, Exposed Vegetable
L1_Fish	Loss type 1, Fish
L1_pork	Loss type 1, Pork

Exhibit 1-1. Variables Included in the Systematic Sensitivity Analysis

Variable Name	Variable Description
L1_poultry	Loss type 1, Poultry
L1_ProFruit	Loss type 1, Protected Fruit
L1_ProVeg	Loss type 1, Protected Vegetable
L1_RootVeg	Loss type 1, Root Vegetable
L2_beef	Loss type 2, Beef
L2_ExpFruit	Loss type 2, Exposed Fruit
L2_Fish	Loss type 2, Fish
L2_pork	Loss type 2, Pork
L2_poultry	Loss type 2, Poultry
L2_RootVeg	Loss type 2, Root Vegetable
MF [d,e]	Mammalian metabolism factor
ρ_a	Density of air
Qp_fo_beef	Quantity of forage plant type eaten per animal per day, Beef
Qp_fo_dairy	Quantity of forage plant type eaten per animal per day, Dairy
Qp_gr_beef	Quantity of grain plant type eaten per animal per day, Beef
Qp_gr_dairy	Quantity of grain plant type eaten per animal per day, Dairy
Qp_gr_egg	Quantity of grain plant type eaten per animal per day, Eggs
Qp_gr_pork	Quantity of grain plant type eaten per animal per day, Pork
Qp_gr_poultry	Quantity of grain plant type eaten per animal per day, Poultry
Qp_si_beef	Quantity of silage plant type eaten per animal per day, Beef
Qp_si_dairy	Quantity of silage plant type eaten per animal per day, Dairy
Qp_si_pork	Quantity of silage plant type eaten per animal per day, Pork
Qs_beef	Quantity of soil eaten by the animal each day, Beef
Qs_dairy	Quantity of soil eaten by the animal each day, Dairy
Qs_egg	Quantity of soil eaten by the animal each day, Eggs
Qs_pork	Quantity of soil eaten by the animal each day, Pork
Qs_poultry	Quantity of soil eaten by the animal each day, Poultry
RCF	PBHAP-specific root concentration factor for tubers and root produce
Rp_ef	Interception fraction of the edible portion of plant, Exposed Fruit
Rp_ev	Interception fraction of the edible portion of plant, Exposed Vegetable
Rp_fo	Interception fraction of the edible portion of plant, Forage
Rp_si	Interception fraction of the edible portion of plant, Silage
Tp_ef	Length of plant exposure to deposition per harvest, Exposed Fruit
Tp_ev	Length of plant exposure to deposition per harvest, Exposed Vegetable
Tp_fo	Length of plant exposure to deposition per harvest, Forage
Tp_si	Length of plant exposure to deposition per harvest, Silage
VG_AG_ef [f,g]	Empirical correction factor, Exposed Fruit
VG_AG_ev [f,g]	Empirical correction factor, Exposed Vegetable
VG_AG_fo [a,b]	Empirical correction factor, Forage
VG_AG_si	Empirical correction factor, Silage
VG_rootveg [f,g]	Empirical correction factor, Root Vegetable
Yp_ef	Yield or standing crop biomass, Exposed Fruit
Yp_ev	Yield or standing crop biomass, Exposed Vegetable
Yp_fo	Yield or standing crop biomass, Forage
Yp_si	Yield or standing crop biomass, Silage

Exhibit 1-1. Variables Included in the Systematic Sensitivity Analysis

Variable Name	Variable Description
MIRC Ingestion and Body Weight Variables	
BW_adlt	Body weight, Adult 20-70
BW_ch1	Body weight, Child 1-2
BW_ch2	Body weight, Child 3-5
BW_ch3	Body Weight, Child 6-11
BW_ch4	Body Weight, Child 12-19
EF_beef [a,b]	Exposure factor, Beef
EF_dairy [a,b]	Exposure factor, Dairy
EF_egg [a,b]	Exposure factor, Eggs
EF_ExpFruit [a,b]	Exposure factor, Exposed Fruit
EF_ExpVeg [a,b]	Exposure factor, Exposed Vegetables
EF_Fish [a,b]	Exposure factor, Fish
EF_pork [a,b]	Exposure factor, Pork
EF_poultry [a,b]	Exposure factor, Poultry
EF_ProFruit [a,b]	Exposure factor, Protected Fruit
EF_ProVeg [a,b]	Exposure factor, Protected Vegetables
EF_RootVeg [a,b]	Exposure factor, Root Vegetables
EF_Soil [a,b]	Exposure factor, Soil
EF_water [a,b]	Exposure factor, Water
F_fo_beef [a,b]	Fraction of forage plant type from contaminated area used to grow feed, Beef
F_fo_dairy [a,b]	Fraction of forage plant type from contaminated area used to grow feed, Dairy
F_gr_beef [a,b]	Fraction of grain plant type from contaminated area used to grow feed, Beef
F_gr_dairy [a,b]	Fraction of grain plant type from contaminated area used to grow feed, Dairy
F_gr_egg [a,b]	Fraction of grain plant type from contaminated area used to grow feed, Eggs
F_gr_pork [a,b]	Fraction of grain plant type from contaminated area used to grow feed, Pork
F_gr_poultry [a,b]	Fraction of grain plant type from contaminated area used to grow feed, Poultry
F_si_beef [a,b]	Fraction of silage plant type from contaminated area used to grow feed, Beef
F_si_dairy [a,b]	Fraction of silage plant type from contaminated area used to grow feed, Dairy
F_si_pork [a,b]	Fraction of silage plant type from contaminated area used to grow feed, Pork
F_T3	Fraction of fish intake that is from T3
F_T4	Fraction of fish intake that is from T4
FC_beef [a,b]	Fraction contaminated, Beef
FC_dairy [a,b]	Fraction contaminated, Dairy
FC_egg [a,b]	Fraction contaminated, Eggs
FC_ExpFruit [a,b]	Fraction contaminated, Exposed Fruit
FC_ExpVeg [a,b]	Fraction contaminated, Exposed Vegetable
FC_Fish [a,b]	Fraction contaminated, Fish
FC_pork [a,b]	Fraction contaminated, Pork
FC_poultry [a,b]	Fraction contaminated, Poultry
FC_ProFruit [a,b]	Fraction contaminated, Protected Fruit
FC_ProVeg [a,b]	Fraction contaminated, Protected Vegetable
FC_RootVeg [a,b]	Fraction contaminated, Root Vegetable
FC_Soil [a,b]	Fraction contaminated, Soil
IR_beef_adlt	Ingestion rate, Beef, Adult 20-70
IR_beef_ch1	Ingestion rate, Beef, Child 1-2

Exhibit 1-1. Variables Included in the Systematic Sensitivity Analysis

Variable Name	Variable Description
IR_beef_ch2	Ingestion rate, Beef, Child 3-5
IR_beef_ch3	Ingestion rate, Beef, Child 6-11
IR_beef_ch4	Ingestion rate, Beef, Child 12-19
IR_dairy_adlt	Ingestion rate, Dairy, Adult 20-70
IR_dairy_ch1	Ingestion rate, Dairy, Child 1-2
IR_dairy_ch2	Ingestion rate, Dairy, Child 3-5
IR_dairy_ch3	Ingestion rate, Dairy, Child 6-11
IR_dairy_ch4	Ingestion rate, Dairy, Child 12-19
IR_egg_adlt	Ingestion rate, Eggs, Adult 20-70
IR_egg_ch1	Ingestion rate, Eggs, Child 1-2
IR_egg_ch2	Ingestion rate, Eggs, Child 3-5
IR_egg_ch3	Ingestion rate, Eggs, Child 6-11
IR_egg_ch4	Ingestion rate, Eggs, Child 12-19
IR_ExpFruit_adlt	Ingestion rate, Exposed Fruit, Adult 20-70
IR_ExpFruit_ch1	Ingestion rate, Exposed Fruit, Child 1-2
IR_ExpFruit_ch2	Ingestion rate, Exposed Fruit, Child 3-5
IR_ExpFruit_ch3	Ingestion rate, Exposed Fruit, Child 6-11
IR_ExpFruit_ch4	Ingestion rate, Exposed Fruit, Child 12-19
IR_ExpVeg_adlt	Ingestion rate, Exposed Vegetable, Adult 20-70
IR_ExpVeg_ch1	Ingestion rate, Exposed Vegetable, Child 1-2
IR_ExpVeg_ch2	Ingestion rate, Exposed Vegetable, Child 3-5
IR_ExpVeg_ch3	Ingestion rate, Exposed Vegetable, Child 6-11
IR_ExpVeg_ch4	Ingestion rate, Exposed Vegetable, Child 12-19
IR_Fish_adlt	Ingestion rate, Fish, Adult 20-70
IR_Fish_ch1	Ingestion rate, Fish, Child 1-2
IR_Fish_ch2	Ingestion rate, Fish, Child 3-5
IR_Fish_ch3	Ingestion rate, Fish, Child 6-11
IR_Fish_ch4	Ingestion rate, Fish, Child 12-19
IR_pork_adlt	Ingestion rate, Pork, Adult 20-70
IR_pork_ch1	Ingestion rate, Pork, Child 1-2
IR_pork_ch2	Ingestion rate, Pork, Child 3-5
IR_pork_ch3	Ingestion rate, Pork, Child 6-11
IR_pork_ch4	Ingestion rate, Pork, Child 12-19
IR_poultry_adlt	Ingestion rate, Poultry, Adult 20-70
IR_poultry_ch1	Ingestion rate, Poultry, Child 1-2
IR_poultry_ch2	Ingestion rate, Poultry, Child 3-5
IR_poultry_ch3	Ingestion rate, Poultry, Child 6-11
IR_poultry_ch4	Ingestion rate, Poultry, Child 12-19
IR_ProFruit_adlt	Ingestion rate, Protected Fruit, Adult 20-70
IR_ProFruit_ch1	Ingestion rate, Protected Fruit, Child 1-2
IR_ProFruit_ch2	Ingestion rate, Protected Fruit, Child 3-5
IR_ProFruit_ch3	Ingestion rate, Protected Fruit, Child 6-11
IR_ProFruit_ch4	Ingestion rate, Protected Fruit, Child 12-19
IR_ProVeg_adlt	Ingestion rate, Protected Vegetable, Adult 20-70
IR_ProVeg_ch1	Ingestion rate, Protected Vegetable, Child 1-2

Exhibit 1-1. Variables Included in the Systematic Sensitivity Analysis

Variable Name	Variable Description
IR_ProVeg_ch2	Ingestion rate, Protected Vegetable, Child 3-5
IR_ProVeg_ch3	Ingestion rate, Protected Vegetable, Child 6-11
IR_ProVeg_ch4	Ingestion rate, Protected Vegetable, Child 12-19
IR_RootVeg_adlt	Ingestion rate, Root Vegetable, Adult 20-70
IR_RootVeg_ch1	Ingestion rate, Root Vegetable, Child 1-2
IR_RootVeg_ch2	Ingestion rate, Root Vegetable, Child 3-5
IR_RootVeg_ch3	Ingestion rate, Root Vegetable, Child 6-11
IR_RootVeg_ch4	Ingestion rate, Root Vegetable, Child 12-19
IR_Soil_adlt	Ingestion rate, Soil, Adult 20-70
IR_Soil_ch1	Ingestion rate, Soil, Child 1-2
IR_Soil_ch2	Ingestion rate, Soil, Child 3-5
IR_Soil_ch3	Ingestion rate, Soil, Child 6-11
IR_Soil_ch4	Ingestion rate, Soil, Child 12-19
IR_water_adlt	Ingestion rate, Water, Adult 20-70
IR_water_ch1	Ingestion rate, Water, Child 1-2
IR_water_ch2	Ingestion rate, Water, Child 3-5
IR_water_ch3	Ingestion rate, Water, Child 6-11
IR_water_ch4	Ingestion rate, Water, Child 12-19

a Values cannot be increased by 5% because the variable has an upper bound.

b Values cannot be increased by 50% because the variable has an upper bound.

c Values can only be increased by 5% for 2,3,7,8 – TCDD and Benzo[a]Pyrene because the variable has an upper bound.

d Values can only be increased by 5% for Benzo[a]Pyrene because the variable has an upper bound.

e Values can only be increased by 50% for Benzo[a]Pyrene because the variable has an upper bound.

f Values can only be increased by 5% for Benzo[a]Pyrene, 2,3,7,8-TCDD, and methyl mercury because the variable has an upper bound.

g Values can only be increased by 5% for Benzo[a]Pyrene, 2,3,7,8-TCDD, and methyl mercury because the variable has an upper bound.

Exhibit 1-2. Elasticities and Rankings for the Variables with the Highest Elasticities for Benzo[a]Pyrene

Name of Variable Changed	Variable Definition	Variable Category [a]	Elasticity of Risk for Input Variable Perturbation				Ranking of Elasticity for Input Variable Perturbation			
			-50%	-5%	5%	50%	-50%	-5%	5%	50%
MixHeight	Mixing Height	TRIM	-1.97	-1.05	-0.95	-0.66	1	1	2	2
EmissionRate	Emission Rate of all Chemicals	TRIM	1.00	1.00	1.00	1.00	3	2	1	1
HorizWindSpeed	Horizontal Wind Speed	TRIM	-1.74	-0.97	-0.88	-0.63	2	3	3	3
Rain	Annual Rainfall	TRIM	0.63	0.63	0.63	0.63	5	4	4	4
Drwp	Average annual wet deposition of particle-phase chemical	FFC	0.45	0.45	0.45	0.45	6	5	5	5
Fw	Fraction of wet deposition that adheres to plant surfaces	FFC	0.45	0.45	0.45	0.45	7	6	6	6
MF	Mammalian metabolism factor	FFC	0.41	0.41	0.41	0.41	9	7	7	7
Yp_fo	Yield or standing crop biomass, Forage	FFC	-0.69	-0.36	-0.33	-0.23	4	8	10	12
Rp_fo	Interception fraction of the edible portion of plant, Forage	FFC	0.34	0.34	0.34	0.34	10	9	8	8
Ba_dairy	Chemical-specific biotransfer factor, Dairy	FFC	0.34	0.34	0.34	0.34	12	10	9	9
EF_dairy	Exposure factor, Dairy	Ing./BW	0.34	0.34	N/A	N/A	13	11		
FC_dairy	Fraction contaminated, Dairy	Ing./BW	0.34	0.34	N/A	N/A	14	12		
Drdp	Average annual dry deposition of particle-phase chemical	FFC	0.30	0.30	0.30	0.30	16	13	11	10
Qp_fo_dairy	Quantity of forage plant type eaten per animal per day, Dairy	FFC	0.29	0.29	0.29	0.29	18	14	12	11
F_fo_dairy	Fraction of forage plant type obtained from contaminated area used to grow feed, Dairy	Ing./BW	0.29	0.29	N/A	N/A	17	15		
Kp_fo	Plant surface loss coefficient, Forage	FFC	-0.34	-0.25	-0.24	-0.19	11	16	13	15
Yp_ef	Yield or standing crop biomass, Exposed Fruit	FFC	-0.43	-0.23	-0.20	-0.14	8	17	16	17
100-MAF_ef	100 - Moisture adjustment factor, Exposed Fruit	FFC	0.22	0.22	0.22	0.22	22	18	14	13
EF_ExpFruit	Exposure factor, Exposed Fruit	Ing./BW	0.22	0.22	N/A	N/A	20	19		

Exhibit 1-2. Elasticities and Rankings for the Variables with the Highest Elasticities for Benzo[a]Pyrene

Name of Variable Changed	Variable Definition	Variable Category [a]	Elasticity of Risk for Input Variable Perturbation				Ranking of Elasticity for Input Variable Perturbation			
			-50%	-5%	5%	50%	-50%	-5%	5%	50%
FC_ExpFruit	Fraction contaminated, Exposed Fruit	Ing./BW	0.22	0.22	N/A	N/A	21	20		
Rp_ef	Interception fraction of the edible portion of plant, Exposed Fruit	FFC	0.21	0.21	0.21	0.21	23	21	15	14
Kp_ef	Plant surface loss coefficient, Exposed Fruit	FFC	-0.27	-0.19	-0.17	-0.13	19	22	17	18
Yp_ev	Yield or standing crop biomass, Exposed Vegetable	FFC	-0.33	-0.17	-0.16	-0.11	15	23	19	21
100-MAF_ev	100 - Moisture adjustment factor, Exposed Vegetable	FFC	0.17	0.17	0.17	0.17	26	24	18	16
EF_ExpVeg	Exposure factor, Exposed Vegetables	Ing./BW	0.17	0.17	N/A	N/A	27	25		
FC_ExpVeg	Fraction contaminated, Exposed Vegetable	Ing./BW	0.17	0.17	N/A	N/A	28	26		
Rp_ev	Interception fraction of the edible portion of plant, Exposed Vegetable	FFC	0.16	0.16	N/A	N/A	29	27		
Kp_ev	Plant surface loss coefficient, Exposed Vegetable	FFC	-0.21	-0.14	-0.13	-0.10	24	28	20	25
C_Soil	Concentration of chemical in soil from contaminated area	FFC	0.12	0.12	0.12	0.12	31	29	21	19
EF_Soil	Exposure factor, Soil	Ing./BW	0.12	0.12	N/A	N/A	32	30		
FC_Soil	Fraction contaminated, Soil	Ing./BW	0.12	0.12	N/A	N/A	33	31		
IR_dairy_ch1	Ingestion rate, Dairy, Child 1-2	Ing./BW	0.11	0.11	0.11	0.11	34	32	22	20
IR_ExpFruit_adlt	Ingestion rate, Exposed Fruit, Adult 20-70	Ing./BW	0.10	0.10	0.10	0.10	35	33	23	23
Tp_fo	Length of plant exposure to deposition per harvest, Forage	FFC	0.17	0.10	0.09	0.06	25	34	26	31
IR_dairy_adlt	Ingestion rate, Dairy, Adult 20-70	Ing./BW	0.10	0.10	0.10	0.10	36	35	24	24
L2_ExpFruit	Loss type 2, Exposed Fruit	FFC	-0.10	-0.10	-0.10	-0.10	37	36	25	26

[a] FFC refers to farm food chain variables and Ing./BW refers to ingestion and body weight variables.

Exhibit 1-3. Elasticities and Rankings for the Variables with the Highest Elasticities for 2,3,7,8 - TCDD

Name of Variable Changed	Variable Definition	Variable Category [a]	Elasticity of Risk for Input Variable Perturbation				Ranking of Elasticity for Input Variable Perturbation			
			-50%	-5%	5%	50%	-50%	-5%	5%	50%
MixHeight	Mixing Height	TRIM	-1.99	-1.05	-0.95	-0.67	1	1	2	2
EmissionRate	Emission Rate of all Chemicals	TRIM	1.00	1.00	1.00	1.00	3	2	1	1
HorizWindSpeed	Horizontal Wind Speed	TRIM	-1.80	-0.96	-0.87	-0.61	2	3	3	3
FC_Fish	Fraction contaminated, Fish	Ing./BW	0.56	0.56	N/A	N/A	8	4		
EF_Fish	Exposure factor, Fish	Ing./BW	0.56	0.56	N/A	N/A	7	5		
BW_adlt	Body weight, Adult 20-70	Ing./BW	-0.86	-0.45	-0.41	-0.29	4	6	6	11
C_FishT4	Concentration of chemical in whole fish for T4 fish	FFC	0.43	0.43	0.43	0.43	9	7	4	5
IR_Fish_adlt	Ingestion rate, Fish, Adult 20-70	Ing./BW	0.43	0.43	0.43	0.43	10	8	5	6
MF	Mammalian metabolism factor	FFC	0.43	0.43	N/A	N/A	11	9		
SurfWatTemp	Water Temperature	TRIM	0.32	0.39	0.41	0.53	16	10	7	4
WatOCC	Water- Organic Carbon Fraction	TRIM	-0.62	-0.38	-0.35	-0.25	5	11	9	14
SedDepVel	Sediment Deposition Velocity and Resuspension Velocity	TRIM	0.28	0.36	0.39	0.30	20	12	8	8
Ba_dairy	Chemical-specific biotransfer factor, Dairy	FFC	0.32	0.32	0.32	0.32	13	13	10	7
EF_dairy	Exposure factor, Dairy	Ing./BW	0.32	0.32	N/A	N/A	14	14		
FC_dairy	Fraction contaminated, Dairy	Ing./BW	0.32	0.32	N/A	N/A	15	15		
F_T3	Fraction of fish intake that is from T3	Ing./BW	-0.30	-0.30	-0.30	-0.30	17	16	12	10
F_T4	Fraction of fish intake that is from T4	Ing./BW	0.30	0.30	0.30	0.30	18	17	11	9
Yp_fo	Yield or standing crop biomass, Forage	FFC	-0.56	-0.30	-0.27	-0.19	6	18	15	19
Rp_fo	Interception fraction of the edible portion of plant, Forage	FFC	0.28	0.28	0.28	0.28	19	19	13	12
Drdp	Average annual dry deposition of particle-phase chemical	FFC	0.28	0.28	0.28	0.28	22	20	14	13
WatSuspSed	Water- Suspended Sediment Concentration	TRIM	-0.37	-0.25	-0.24	-0.19	12	21	16	18

Exhibit 1-3. Elasticities and Rankings for the Variables with the Highest Elasticities for 2,3,7,8 - TCDD

Name of Variable Changed	Variable Definition	Variable Category [a]	Elasticity of Risk for Input Variable Perturbation				Ranking of Elasticity for Input Variable Perturbation			
			-50%	-5%	5%	50%	-50%	-5%	5%	50%
Qp_fo_dairy	Quantity of forage plant type eaten per animal per day, Dairy	FFC	0.23	0.23	0.23	0.23	24	22	17	15
F_fo_dairy	Fraction of forage plant type obtained from contaminated area used to grow feed, Dairy	Ing./BW	0.23	0.23	N/A	N/A	23	23		
Kp_fo	Plant surface loss coefficient, Forage	FFC	-0.28	-0.21	-0.20	-0.16	21	24	19	21
IR_dairy_adlt	Ingestion rate, Dairy, Adult 20-70	Ing./BW	0.20	0.20	0.20	0.20	25	25	18	16
Rain	Annual Rainfall	TRIM	0.20	0.20	0.20	0.19	26	26	20	17
SurfSoilAir	Surface Soil- Fraction Air	TRIM	0.13	0.14	0.15	0.17	29	27	21	20
C_FishT3	Concentration of chemical in whole fish for T3 fish	FFC	0.13	0.13	0.13	0.13	28	28	22	22
Cs_S_animal_ingest	Chemical concentration in surface soil in contaminated area where livestock feed	FFC	0.12	0.12	0.12	0.12	31	29	23	23
Bs	Soil bioavailability factor for livestock	FFC	0.12	0.12	N/A	N/A	30	30		
Ba_beef	Chemical-specific biotransfer factor, Beef	FFC	0.09	0.09	0.09	0.09	33	31	24	24
EF_beef	Exposure factor, Beef	Ing./BW	0.09	0.09	N/A	N/A	34	32		
FC_beef	Fraction contaminated, Beef	Ing./BW	0.09	0.09	N/A	N/A	35	33		
Tp_fo	Length of plant exposure to deposition per harvest, Forage	FFC	0.14	0.08	0.08	0.05	27	34	25	29
Qs_dairy	Quantity of soil eaten by the animal each day, Dairy	FFC	0.07	0.07	0.07	0.07	38	35	26	25

[a] FFC refers to farm food chain variables and Ing./BW refers to ingestion and body weight variables.

Exhibit 1-4. Elasticities and Rankings for the Variables with the Highest Elasticities for Cadmium

Name of Variable Changed	Variable Definition	Variable Category [a]	Elasticity of Risk for Input Variable Perturbation				Ranking of Elasticity for Input Variable Perturbation			
			-50%	-5%	5%	50%	-50%	-5%	5%	50%
MixHeight	Mixing Height	TRIM	-1.92	-1.03	-0.93	-0.66	1	1	2	3
HorizWindSpeed	Horizontal Wind Speed	TRIM	-1.92	-1.03	-0.93	-0.66	2	2	3	4
EmissionRate	Emission Rate of all Chemicals	TRIM	1.00	1.00	1.00	1.00	3	3	1	1
Rain	Annual Rainfall	TRIM	0.78	0.75	0.74	0.71	4	4	4	2
Cs_root_zone_produce	Average chemical concentration in soil at root-zone depth in produce-growing area	FFC	0.63	0.63	0.63	0.63	5	5	5	5
Br_AG_produce_pf	Plant-soil chemical bioconcentration factor, Protected Fruit	FFC	0.39	0.39	0.39	0.39	7	6	6	8
IR_ProFruit_ch1	Ingestion rate, Protected Fruit, Child 1-2	Ing./BW	0.39	0.39	0.39	0.39	10	7	7	9
EF_ProFruit	Exposure factor, Protected Fruit	Ing./BW	0.39	0.39	N/A	N/A	8	8		
FC_ProFruit	Fraction contaminated, Protected Fruit	Ing./BW	0.39	0.39	N/A	N/A	9	9		
100-MAF_pf	100 - Moisture adjustment factor, Protected Fruit	FFC	0.39	0.39	0.39	0.39	11	10	8	7
BW_ch1	Body weight, Child 1-2	Ing./BW	-0.54	-0.28	-0.26	-0.18	6	11	11	13
EroRate	Erosion Rate for all Parcels	TRIM	-0.31	-0.27	-0.26	-0.24	12	12	10	10
SurfWatTemp	Water Temperature	TRIM	0.17	0.27	0.30	0.54	16	13	9	6
IR_Fish_ch1	Ingestion rate, Fish, Child 1-2	Ing./BW	0.19	0.19	0.19	0.19	15	14	12	12
EF_Fish	Exposure factor, Fish	Ing./BW	0.19	0.19	N/A	N/A	13	15		
FC_Fish	Fraction contaminated, Fish	Ing./BW	0.19	0.19	N/A	N/A	14	16		
SurfSoilAir	Surface Soil- Fraction Air	TRIM	0.15	0.17	0.17	0.19	19	17	13	11
L1_ProFruit	Loss type 1, Protected Fruit	FFC	-0.16	-0.16	-0.16	-0.16	18	18	14	14
C_FishT3	Concentration of chemical in whole fish for T3 fish	FFC	0.15	0.15	0.15	0.15	21	19	15	15
WatRetTime	Rentention Time in Pond	TRIM	0.16	0.15	0.14	0.13	17	20	17	16
SedDepVel	Sediment Deposition Velocity and Resuspension Velocity	TRIM	0.10	0.13	0.14	0.07	30	21	16	32

Exhibit 1-4. Elasticities and Rankings for the Variables with the Highest Elasticities for Cadmium

Name of Variable Changed	Variable Definition	Variable Category [a]	Elasticity of Risk for Input Variable Perturbation				Ranking of Elasticity for Input Variable Perturbation			
			-50%	-5%	5%	50%	-50%	-5%	5%	50%
100-MAF_ev	100 - Moisture adjustment factor, Exposed Vegetable	FFC	0.11	0.11	0.11	0.11	23	22	18	18
IR_ExpVeg_ch1	Ingestion rate, Exposed Vegetable, Child 1-2	Ing./BW	0.11	0.11	0.11	0.11	26	23	19	19
EF_ExpVeg	Exposure factor, Exposed Vegetables	Ing./BW	0.11	0.11	N/A	N/A	24	24		
FC_ExpVeg	Fraction contaminated, Exposed Vegetable	Ing./BW	0.11	0.11	N/A	N/A	25	25		
F_T3	Fraction of fish intake that is from T3	Ing./BW	0.11	0.11	0.11	0.11	28	26	20	20
F_T4	Fraction of fish intake that is from T4	Ing./BW	-0.11	-0.11	-0.11	-0.11	29	27	21	21
SurfSoilOCC	Surface Soil- Organic Carbon Fraction	TRIM	0.15	0.10	0.10	0.08	20	28	22	25
SurfSoilVertVel	Surface Soil- Average vertical velocity of water (percolation)	TRIM	-0.11	-0.10	-0.10	-0.09	27	29	23	22
SurfSoilpH	Surface Soil- pH	TRIM	0.13	0.09	0.09	0.07	22	30	25	31

[a] FFC refers to farm food chain variables and Ing./BW refers to ingestion and body weight variables.

Exhibit 1-5. Elasticities and Rankings for the Variables with the Highest Elasticities for Divalent Mercury

Name of Variable Changed	Variable Definition	Variable Category [a]	Elasticity of Risk for Input Variable Perturbation				Ranking of Elasticity for Input Variable Perturbation			
			-50%	-5%	5%	50%	-50%	-5%	5%	50%
EmissionRate	Emission Rate of all Chemicals	TRIM	1.00	1.00	1.00	1.00	5	1	1	1
MixHeight	Mixing Height	TRIM	-1.73	-0.97	-0.89	-0.63	1	2	2	3
HorizWindSpeed	Horizontal Wind Speed	TRIM	-1.73	-0.97	-0.89	-0.63	2	3	3	4
EroRate	Erosion Rate for all Parcels	TRIM	-1.27	-0.77	-0.71	-0.52	3	4	5	7
Rain	Annual Rainfall	TRIM	0.76	0.74	0.74	0.71	6	5	4	2
BW_ch1	Body weight, Child 1-2	Ing./BW	-1.17	-0.61	-0.56	-0.39	4	6	8	8
C_Soil	Concentration of chemical in soil from contaminated area	FFC	0.56	0.56	0.56	0.56	7	7	6	5
EF_Soil	Exposure factor, Soil	Ing./BW	0.56	0.56	N/A	N/A	8	8		
FC_Soil	Fraction contaminated, Soil	Ing./BW	0.56	0.56	N/A	N/A	9	9		
IR_Soil_ch1	Ingestion rate, Soil, Child 1-2	Ing./BW	0.56	0.56	0.56	0.56	10	10	7	6
Cs_root_zone_produce	Average chemical concentration in soil at root-zone depth in produce-growing area	FFC	0.33	0.33	0.33	0.33	11	11	9	9
Br_AG_produce_pf	Plant-soil chemical bioconcentration factor, Protected Fruit	FFC	0.15	0.15	0.15	0.15	12	12	10	10
IR_ProFruit_ch1	Ingestion rate, Protected Fruit, Child 1-2	Ing./BW	0.15	0.15	0.15	0.15	15	13	11	11
100-MAF_pf	100 - Moisture adjustment factor, Protected Fruit	FFC	0.15	0.15	0.15	0.15	16	14	12	12
EF_ProFruit	Exposure factor, Protected Fruit	Ing./BW	0.15	0.15	N/A	N/A	13	15		
FC_ProFruit	Fraction contaminated, Protected Fruit	Ing./BW	0.15	0.15	N/A	N/A	14	16		
SurfSoilAir	Surface Soil- Fraction Air	TRIM	0.11	0.13	0.13	0.15	17	17	13	13
Br_AG_rootveg	Plant-soil chemical bioconcentration factor, Root Vegetables	FFC	0.11	0.11	0.11	0.11	18	18	14	15
IR_RootVeg_ch1	Ingestion rate, Root Vegetable, Child 1-2	Ing./BW	0.11	0.11	0.11	0.11	21	19	15	16

Exhibit 1-5. Elasticities and Rankings for the Variables with the Highest Elasticities for Divalent Mercury

Name of Variable Changed	Variable Definition	Variable Category [a]	Elasticity of Risk for Input Variable Perturbation				Ranking of Elasticity for Input Variable Perturbation			
			-50%	-5%	5%	50%	-50%	-5%	5%	50%
100-MAF_rv	100 - Moisture adjustment factor, Root Vegetable	FFC	0.11	0.11	0.11	0.11	23	20	16	17
EF_RootVeg	Exposure factor, Root Vegetables	Ing./BW	0.11	0.11	N/A	N/A	19	21		
FC_RootVeg	Fraction contaminated, Root Vegetable	Ing./BW	0.11	0.11	N/A	N/A	20	22		
VG_rootveg	Empirical correction factor, Root Vegetable	FFC	0.11	0.11	N/A	N/A	22	23		
Cs_S_animal_ingest	Chemical concentration in surface soil in contaminated area where livestock feed	FFC	0.08	0.08	0.08	0.08	25	24	17	18
Bs	Soil bioavailability factor for livestock	FFC	0.08	0.08	N/A	N/A	24	25		
L1_ProFruit	Loss type 1, Protected Fruit	FFC	-0.06	-0.06	-0.06	-0.06	26	26	19	19
SedDepVel	Sediment Deposition Velocity and Resuspension Velocity	TRIM	0.02	0.05	0.06	0.11	48	27	18	14
Ba_egg	Chemical-specific biotransfer factor, Eggs	FFC	0.05	0.05	0.05	0.05	27	28	20	20
IR_egg_ch1	Ingestion rate, Eggs, Child 1-2	Ing./BW	0.05	0.05	0.05	0.05	30	29	21	21
EF_egg	Exposure factor, Eggs	Ing./BW	0.05	0.05	N/A	N/A	28	30		
FC_egg	Fraction contaminated, Eggs	Ing./BW	0.05	0.05	N/A	N/A	29	31		
Qs_egg	Quantity of soil eaten by the animal each day, Eggs	FFC	0.05	0.05	0.05	0.05	31	32	22	22
Ba_poultry	Chemical-specific biotransfer factor, Poultry	FFC	0.03	0.03	0.03	0.03	32	33	23	23
IR_poultry_ch1	Ingestion rate, Poultry, Child 1-2	Ing./BW	0.03	0.03	0.03	0.03	35	34	24	24
EF_poultry	Exposure factor, Poultry	Ing./BW	0.03	0.03	N/A	N/A	33	35		
FC_poultry	Fraction contaminated, Poultry	Ing./BW	0.03	0.03	N/A	N/A	34	36		
Qs_poultry	Quantity of soil eaten by the animal each day, Poultry	FFC	0.03	0.03	0.03	0.03	36	37	25	25

[a] FFC refers to farm food chain variables and Ing./BW refers to ingestion and body weight variables.

Exhibit 1-6. Elasticities and Rankings for the Variables with the Highest Elasticities for Methyl Mercury

Name of Variable Changed	Variable Definition	Variable Category [a]	Elasticity of Risk for Input Variable Perturbation				Ranking of Elasticity for Input Variable Perturbation			
			-50%	-5%	5%	50%	-50%	-5%	5%	50%
SedDepVel	Sediment Deposition Velocity and Resuspension Velocity	TRIM	0.78	1.56	2.01	3.67	9	1	1	1
BW_ch1	Body weight, Child 1-2	Ing./BW	-1.91	-1.00	-0.91	-0.64	1	2	3	4
EmissionRate	Emission Rate of all Chemicals	TRIM	1.00	1.00	1.00	1.00	5	3	2	2
IR_Fish_ch1	Ingestion rate, Fish, Child 1-2	Ing./BW	0.90	0.90	0.90	0.90	8	4	4	3
FC_Fish	Fraction contaminated, Fish	Ing./BW	0.90	0.90	N/A	N/A	7	5		
EF_Fish	Exposure factor, Fish	Ing./BW	0.90	0.90	N/A	N/A	6	6		
MixHeight	Mixing Height	TRIM	-1.02	-0.70	-0.66	-0.51	3	7	5	8
HorizWindSpeed	Horizontal Wind Speed	TRIM	-1.01	-0.70	-0.65	-0.51	4	8	6	9
Rain	Annual Rainfall	TRIM	0.70	0.64	0.63	0.60	10	9	7	6
C_FishT4	Concentration of chemical in whole fish for T4 fish	FFC	0.55	0.55	0.55	0.55	12	10	8	7
EroRate	Erosion Rate for all Parcels	TRIM	-0.57	-0.45	-0.43	-0.35	11	11	10	11
SedPorosity	Sediment Porosity	TRIM	-0.30	-0.40	-0.43	-0.60	15	12	9	5
C_FishT3	Concentration of chemical in whole fish for T3 fish	FFC	0.36	0.36	0.36	0.36	13	13	11	10
SurfWatTemp	Water Temperature	TRIM	-0.22	-0.22	-0.22	-0.21	18	14	12	12
F_T3	Fraction of fish intake that is from T3	Ing./BW	-0.19	-0.19	-0.19	-0.19	19	15	13	13
F_T4	Fraction of fish intake that is from T4	Ing./BW	0.19	0.19	0.19	0.19	20	16	14	14
WatSuspSed	Water- Suspended Sediment Concentration	TRIM	-0.32	-0.18	-0.16	-0.12	14	17	15	16
SurfSoilAir	Surface Soil- Fraction Air	TRIM	0.14	0.14	0.15	0.15	21	18	16	15
WatRetTime	Rentention Time in Pond	TRIM	0.23	0.14	0.13	0.09	17	19	17	17
FishMass	Fish Body Weight for all Aquatic Species	TRIM	0.10	0.07	0.07	0.06	22	20	19	19
Runoff	Total Water Runoff Rate	TRIM	0.07	0.07	0.07	0.07	23	21	18	18
C_Soil	Concentration of chemical in soil from contaminated area	FFC	0.05	0.05	0.05	0.05	24	22	20	20

Exhibit 1-6. Elasticities and Rankings for the Variables with the Highest Elasticities for Methyl Mercury

Name of Variable Changed	Variable Definition	Variable Category [a]	Elasticity of Risk for Input Variable Perturbation				Ranking of Elasticity for Input Variable Perturbation			
			-50%	-5%	5%	50%	-50%	-5%	5%	50%
IR_Soil_ch1	Ingestion rate, Soil, Child 1-2	Ing./BW	0.05	0.05	0.05	0.05	27	23	21	21
EF_Soil	Exposure factor, Soil	Ing./BW	0.05	0.05	N/A	N/A	25	24		
FC_Soil	Fraction contaminated, Soil	Ing./BW	0.05	0.05	N/A	N/A	26	25		
Cs_root_zone_produce	Average chemical concentration in soil at root-zone depth in produce-growing area	FFC	0.04	0.04	0.04	0.04	28	26	22	22
100-MAF_pf	100 - Moisture adjustment factor, Protected Fruit	FFC	0.03	0.03	0.03	0.03	33	27	25	23
Br_AG_produce_pf	Plant-soil chemical bioconcentration factor, Protected Fruit	FFC	0.03	0.03	0.03	0.03	29	28	23	24
IR_ProFruit_ch1	Ingestion rate, Protected Fruit, Child 1-2	Ing./BW	0.03	0.03	0.03	0.03	32	29	24	25

[a] FFC refers to farm food chain variables and Ing./BW refers to ingestion and body weight variables.

Appendix D: Detailed assessment inputs and results for petroleum refining facilities

Table 1 - Facility Identification Information

Facility NEI ID	Facility Name	Address	City	County	State
PET_NEI109	SUNOCO INC (R&M)/MARCUS HOOK REFINERY	100 GREEN ST PO BOX 426	MARCUS HOOK	Delaware County	PA
PET_NEI11119	Deer Park Refining Limited Partnership (Shell Oil Products US)	5900 HIGHWAY 225	Deer Park	Harris County	TX
PET_NEI11192	Western Refining Co. LP - North (prev. Chevron USA Inc.)	6501 TROWBRIDGE DR.	EL PASO	El Paso County	TX
PET_NEI11200	Valero Refining Co. - Port Arthur (prev. Premcor Refining Group Inc.)	10801 S. GULFWAY DR.	PORT ARTHUR	Jefferson County	TX
PET_NEI11232	HOUSTON REFINING LP	12000 LAWNDALE ST	HOUSTON	Harris County	TX
PET_NEI113	ConocoPhillips Co. (prev. Phillips 66 Co.)	4101 POST RD	TRAINER	Delaware County	PA
PET_NEI11449	BP OIL COMPANY TOLEDO REFINNERY	4001 CEDAR POINT ROAD	OREGON	Lucas County	OH
PET_NEI11450	Sunoco - Toledo	1819 Woodville Road	OREGON	Lucas County	OH
PET_NEI11574	Marathon Petroleum Company LLC	2408 Gambrinus Avenue SW	CANTON	Stark County	OH
PET_NEI11663	Valero Refining Co. (prev. Premcor Refining Group)	1150 S. METCALF ST.	LIMA	Allen County	OH
PET_NEI11715	BP PRODUCTS NORTH AMERICA INC, WHITING R	2815 INDIANAPOLIS BLVD.	WHITING	Lake County	IN
PET_NEI11885	Marathon Petroleum Company LLC	1300 S. FORT ST. HES DEPARTMENT	DETROIT	Wayne County	MI
PET_NEI12044	Marathon Petroleum Company LLC	1320 LOOP 197 S.	TEXAS CITY	Galveston County	TX
PET_NEI12084	Valero Refining Co. - Corpus Christi West	5900 UP RIVER ROAD	CORPUS CHRISTI	Nueces County	TX
PET_NEI12458	CENEX HARVEST STATES	802 US HWY 212 S, S OF LAUREL	LAUREL	Yellowstone County	MT
PET_NEI12459	ConocoPhillips Co. (prev. Conoco Inc.)	401 S 23RD ST	BILLINGS	Yellowstone County	MT
PET_NEI12460	EXXONMOBIL BILLINGS REFINERY	700 EXXONMOBIL RD	BILLINGS	Yellowstone County	MT
PET_NEI12464	MONTANA REFINING	1900 10TH STREET N.E.	GREAT FALLS	Cascade County	MT
PET_NEI12480	Pasadena Refining Systems Inc. (prev. Crown Central Petroleum Corp.)	111 RED BLUFF ROAD	PASADENA	Harris County	TX
PET_NEI12486	VALERO THREE RIVERS REFINERY	301 LEROY STREET	THREE RIVERS	Live Oak County	TX
PET_NEI12711	Valero Refining Co. - Houston	9701 MANCHESTER	HOUSTON	Harris County	TX
PET_NEI12790	Western Refining Co. LP - South (prev. Chevron USA Inc.)	6500 TROWBRIDGE ST.	EL PASO	El Paso County	TX
PET_NEI12790	Western Refining Co. LP - South (prev. Chevron USA Inc.)	6501 TROWBRIDGE DR.	EL PASO	El Paso County	TX
PET_NEI12791	Valero Refining Co. - Texas City	1301 LOOP 197 S.	TEXAS CITY	Galveston County	TX
PET_NEI12968	SINCLAIR OIL CORP	902 W 25TH ST	TULSA	Tulsa County	OK
PET_NEI12988	ConocoPhillips Co. (prev. Conoco Inc.)	1000 S PINE	PONCA CITY	Kay County	OK
PET_NEI13322	CHEVRON HAWAII REFINERY	91-480 MALAKOLE ST.	KAPOLEI	Honolulu County	HI
PET_NEI13371	TESORO ALASKA COMPANY - KENAI REFINERY	54741 TESORO ROAD	KENAI	Kenai Peninsula Boro	AK

Table 1 - Facility Identification Information

Facility NEI ID	Facility Name	Address	City	County	State
PET_NEI18372	Shell Chemical LP	LOCATION ADDRESS IS NEEDED	MOBILE	Mobile County	AL
PET_NEI18394	HUNT REFINING COMPANY	1855 FAIRLAWN RD	TUSCALOOSA	Tuscaloosa County	AL
PET_NEI18406	Flint Hills Resources (prev. Williams Alaska Petro Inc.)	1100 H&H Lane	North Pole	Fairbanks North Star	AK
PET_NEI18408	Petro Star Inc. - North Pole	1200 H & H LN.	NORTH POLE	Fairbanks North Star	AK
PET_NEI18415	PETRO STAR VALDEZ REFY.	2.5 MILE DAYVILLE RD.	VALDEZ	Valdez-Cordova Cen	AK
PET_NEI19587	Chevron USA Inc. - Richmond	841 CHEVRON WAY	RICHMOND	Contra Costa County	CA
PET_NEI19834	Shell Oil Products US - Martinez	3485 PACHECO BLVD	MARTINEZ	Contra Costa County	CA
PET_NEI19869	ConocoPhillips Co. - Santa Maria (prev. Phillips 66 Co.)	2555 WILLOW ROAD	ARROYO GRANDE	San Luis Obispo Cou	CA
PET_NEI19870	ConocoPhillips Co. - Rodeo (prev. Phillips 66 Co.)	1380 SAN PABLO AVE	RODEO	Contra Costa County	CA
PET_NEI20103	KERN OIL & REFINING COMPANY	PANAMA LN & WEEDPATCH HWY	BAKERSFIELD	Kern County	CA
PET_NEI20154	SAN JOAQUIN REFINING COMPANY	STANDARD AND SHELL ST	BAKERSFIELD	Kern County	CA
PET_NEI20174	Big West Oil LLC (prev. Shell Oil Products US)	6451 ROSEDALE HWY.	BAKERSFIELD	Kern County	CA
PET_NEI20467	Chevron USA Inc. - El Segundo	324 W EL SEGUNDO BLVD	EL SEGUNDO	Los Angeles County	CA
PET_NEI20616	EDGINGTON OIL COMPANY	2400 E ARTESIA BLVD	LONG BEACH	Los Angeles County	CA
PET_NEI20797	VALERO WILMINGTON ASPHALT PLANT	1651 ALAMEDA ST	WILMINGTON	Los Angeles County	CA
PET_NEI20966	LUNDAY-THAGARD OIL CO	9301 GARFIELD AVENUE	SOUTH GATE	Los Angeles County	CA
PET_NEI21034	ExxonMobil - Torrance	3700 W. 190TH ST.	TORRANCE	Los Angeles County	CA
PET_NEI21130	PARAMOUNT PETROLEUM CORP	14708 DOWNEY AV	PARAMOUNT	Los Angeles County	CA
PET_NEI21466	Valero Refining Co. - Wilmington (prev. Ultramar Inc.)	2402 E. ANAHEIM ST.	WILMINGTON	Los Angeles County	CA
PET_NEI25450	Valero Refining Co. - Benicia	3400 E 2ND STREET	BENICIA	Solano County	CA
PET_NEI25464	Valero Refining - Benicia Asphalt	3001 PARK ROAD	BENICIA	Solano County	CA
PET_NEI26101	TENBY INC.	3455 EAST FIFTH STREET	OXNARD	Ventura County	CA
PET_NEI26218	Valero Refining Co. (prev. Motiva Enterprises LLC)	2000 WRANGLE HILL RD	DELAWARE CITY	New Castle County	DE
PET_NEI26473	CITGO ASPHALT REFINING COMPANY	FOUNDATION DRIVE	SAVANNAH	Chatham County	GA
PET_NEI26489	YOUNG REFINING CORP.	7982 HUEY ROAD	DOUGLASVILLE	Douglas County	GA
PET_NEI26533	Tesoro Hawaii Corp.	91-325 KOMOHANA STREET	KAPOLEI	Honolulu County	HI
PET_NEI2CA131	BP West Coast Products	1801 E SEPULVEDA BLVD	CARSON	Los Angeles County	CA
PET_NEI2CA254	Greka Energy	1660 SINTON RD	SANTA MARIA	Santa Barbara Count	CA
PET_NEI2CA314	Tesoro (prev. Ultramar Inc.)	Avon Refinery	MARTINEZ	Contra Costa County	CA
PET_NEI2KS125	COFFEYVILLE RESOURCES REFINING & MARKETING	400 NORTH LINDEN	COFFEYVILLE	Montgomery County	KS
PET_NEI2TX141	Western Refining Co. LP - Marketing Terminal	6501 TROWBRIDGE DR.	EL PASO	El Paso County	TX
PET_NEI32353	Countrymark Cooperative Inc.	1200 REFINERY RD	MOUNT VERNON	Posey County	IN

Table 1 - Facility Identification Information

Facility NEI ID	Facility Name	Address	City	County	State
PET_NEI32762	FRONTIER EL DORADO REFINING COMPANY	1401 S. DOUGLAS ROAD	EL DORADO	Butler County	KS
PET_NEI32801	NATIONAL COOPERATIVE REFINERY ASSN	1391 IRON HORSE ROAD	MC PHERSON	McPherson County	KS
PET_NEI32864	Marathon Petroleum Company LLC	11631 US ROUTE 23	CATLETTSBURG	Boyd County	KY
PET_NEI32997	SOMERSET REFINERY INC	600 MONTICELLO RD	SOMERSET	Pulaski County	KY
PET_NEI33007	Calumet Lubricants Co. - Princeton Calumet Lubricants Co.	10234 HWY 157	PRINCETON	Bossier Parish	LA
PET_NEI33008	Calumet Shreveport LLC (prev. Calumet Lubricants Co.)	3333 MIDWAY	SHREVEPORT	Caddo Parish	LA
PET_NEI33010	CALCASIEU REFINING CO	4359 W TANK FARM RD	LAKE CHARLES	Calcasieu Parish	LA
PET_NEI33030	SHELL CHEMICAL LP/NORCO CHEM PLT EAST SITE	HWY 61 W	NORCO	St. Charles Parish	LA
PET_NEI33031	MOTIVA ENTERPRISES LLC/NORCO REFINERY	15536 River Road	NORCO	St. Charles Parish	LA
PET_NEI33039	Calumet Lubricants Co. - Cotton Valley	1756 OLD HWY 7	COTTON VALLEY	Webster Parish	LA
PET_NEI34022	FLINT HILLS RESOURCES LP - PINE BEND	JUNCTIONS 52 & 55	INVER GROVE HEI(	Dakota County	MN
PET_NEI34050	MARATHON PETROLEUM CO LLC SAINT PAUL PARK REFINER	300 3RD STREET	SAINT PAUL PARK	Washington County	MN
PET_NEI34057	CHEVRON TEXACO PRODUCTS COMPANY, PASCAGO	250 INDUSTRIAL ROAD	PASCAGOULA	Jackson County	MS
PET_NEI34061	HUNT SOUTHLAND REFINING COMPANY	HIGHWAY 11 NORTH	SANDERSVILLE	Jones County	MS
PET_NEI34062	Hunt Southland Refining (prev. Southland Oil Co.)	HIGHWAY 11 NORTH	LUMBERTON	Lamar County	MS
PET_NEI34069	ERGON REFINING INC	2611 HAINING ROAD	VICKSBURG	Warren County	MS
PET_NEI34862	Sunoco, Inc. (prev. Coastal Eagle Point Oil Co.)	US RT 130 AND 295	WESTVILLE	Gloucester County	NJ
PET_NEI34863	CITGO ASPHALT REFINING COMPANY	4 PARADISE RD	PAULSBORO	Gloucester County	NJ
PET_NEI34872	Hess Corporation (prev. Amerada-Hess Corp.)	750 CLIFF ROAD	PT. READING	Middlesex County	NJ
PET_NEI34873	CHEVRON PRODUCTS COMPANY	1200 STATE ST	PERTH AMBOY	Middlesex County	NJ
PET_NEI34898	Navajo Refining Co. - Artesia	501 E Main St	Artesia	Eddy County	NM
PET_NEI34907	Giant Refining Co. - Ciniza Refinery	I-40 EXIT 39	JAMESTOWN	McKinley County	NM
PET_NEI34912	Giant Industries Inc. - Bloomfield	#50 County Road 4990	Bloomfield	San Juan County	NM
PET_NEI363	FRONTIER REFINING INC	2700 EAST 5TH STREET	CHEYENNE	Laramie County	WY
PET_NEI371	Little America Refining Co. (Sinclair)	5700 E. HWY. 20/26	CASPER	Natrona County	WY
PET_NEI40371	Tesoro - Mandan	900 OLD RED TRAIL N.E.	MANDAN	Morton County	ND
PET_NEI404	WYOMING REFINING CO_NEWCASTLE REFINERY	740 W MAIN STREET	NEWCASTLE	Weston County	WY
PET_NEI40531	Wynnewood Refining Co. (prev. Gary-Williams Energy Corp.)	906 S POWELL	WYNNEWOOD	Garvin County	OK
PET_NEI40625	Paramount Petroleum Corp. (prev. Chevron USA)	5501 NW FRONT AVE	PORTLAND	Multnomah County	OR

Table 1 - Facility Identification Information

Facility NEI ID	Facility Name	Address	City	County	State
PET_NEI40723	Sunoco Inc. - Philadelphia	3144 PASSYUNK AVE.	Philadelphia	Philadelphia County	PA
PET_NEI40732	UNITED REFINING CO/WARREN PLT	15 BRADLEY ST	WARREN	Warren County	PA
PET_NEI415	Suncor Energy USA - Denver (prev. Colorado Refining Co.)	5800 BRIGHTON BLVD	COMMERCE CITY	Adams County	CO
PET_NEI41591	Valero Refining Co. (prev. Premcor Refining, prev. Williams Refining LLC)	543 West Mallory Avenue	Memphis	Shelby County	TN
PET_NEI41771	Total Petrochemicals Inc. (prev. Atofina Petrochemicals, Inc.)	32ND ST. & HWY. 366	PORT ARTHUR	Jefferson County	TX
PET_NEI41863	Valero Refining Co. - Corpus Christi East	1300 CANTWELL LN.	CORPUS CHRISTI	Nueces County	TX
PET_NEI41864	Flint Hills Resources LP - Corpus Christi West	2825 SUNTIDE RD.	CORPUS CHRISTI	Nueces County	TX
PET_NEI41865	Trigeant LTD	6600 UP RIVER ROAD	CORPUS CHRISTI	Nueces County	TX
PET_NEI42016	Big West Oil Co. (Flying J)	333 W CENTER ST	NORTH SALT LAKE	Davis County	UT
PET_NEI42020	Holly Corp. (prev. Phillips 66 Co.)	393 South 800 West	Woods Cross	Davis County	UT
PET_NEI42025	Silver Eagle Refining	2355 S. 1100 W.	WOODS CROSS	Davis County	UT
PET_NEI42040	Tesoro - Salt Lake City	474 W. 900 N.	SALT LAKE CITY	Salt Lake County	UT
PET_NEI42081	Chevron - Salt Lake City	2351 N 1100 W	SALT LAKE CITY	Salt Lake County	UT
PET_NEI42309	GIANT YORKTOWN REFINERY	2201 GOODWIN NECK RD	GRAFTON	York County	VA
PET_NEI42370	U S OIL & REFINING CO	3001 MARSHALL AVE	TACOMA	Pierce County	WA
PET_NEI42381	TESORO NORTHWEST COMPANY	1020 W MARCH POINT RD	ANACORTES	Skagit County	WA
PET_NEI42382	Shell - Anacortes	8505 SOUTH TEXAS ROAD	ANACORTES	Skagit County	WA
PET_NEI42413	BP West Coast Products - Cherry Point	4519 GRANDVIEW RD	BLAINE	Whatcom County	WA
PET_NEI42425	ConocoPhillips Co. (prev. Phillips 66 Co.)	3901 UNICK RD	FERNDALE	Whatcom County	WA
PET_NEI42583	MURPHY OIL USA	24TH AVE E AND 26TH ST	SUPERIOR	Douglas County	WI
PET_NEI43243	SINCLAIR OIL CORP-SINCLAIR REFINERY	BOX 277	SINCLAIR	Carbon County	WY
PET_NEI46556	HOVENSA L.L.C.	1 ESTATE HOPE	CHRISTIANSTED	St. Croix	VI
PET_NEI46752	ERGON - WEST VIRGINIA, INC.	ROUTE 2 SOUTH	NEWELL	Hancock County	WV
PET_NEI46764	American Refining Group Inc.	77 N KENDALL AVE	BRADFORD	McKean County	PA
PET_NEI49781	Marathon Petroleum Company LLC	100 Marathon Ave	Robinson	Crawford County	IL
PET_NEI53702	PDV Midwest Refining LLC (Citgo Petroleum)	135TH ST AND NEW AVE	LEMONT	Will County	IL
PET_NEI53718	EXXONMOBIL OIL CORP	INTERSTATE 55 & ARSENAL RD	JOLIET	Will County	IL
PET_NEI55835	ConocoPhillips Co. (prev. Phillips 66 Co.)	900 S Central Ave	Roxana	Madison County	IL
PET_NEI6018	SHELL CHEMICAL LP/ST. ROSE REFINERY	11842 RIVER RD	ST. ROSE	St. Charles Parish	LA
PET_NEI6022	ExxonMobil Corp. - Baton Rouge	4045 SCENIC HWY	BATON ROUGE	East Baton Rouge Pa	LA
PET_NEI6062	ConocoPhillips Co. - Westlake (prev. Conoco Inc.)	2200 OLD SPANISH TRAIL	WESTLAKE	Calcasieu Parish	LA
PET_NEI6084	MOTIVA ENTERPRISES,LLC/CONVENT	HWY 70 & HWY 44	CONVENT	St. James Parish	LA
PET_NEI6087	Marathon Petroleum Company LLC	E. BANK OF MS RIVER	GARYVILLE	St. John the Baptist P	LA
PET_NEI6095	Valero Refining - Norco (prev. Orion Refining Corp)	14902 RIVER RD.	NEW SARPY	St. Charles Parish	LA

Table 1 - Facility Identification Information

Facility NEI ID	Facility Name	Address	City	County	State
PET_NEI6116	ConocoPhillips Co. - Belle Chasse (prev. Phillips 66 Co.)	15551 HWY 23 S	BELLE CHASSE	Plaquemines Parish	LA
PET_NEI6123	Chalmette Refining LLC (ExxonMobil)	500 W. ST. BERNARD HWY	CHALMETTE	St. Bernard Parish	LA
PET_NEI6127	MURPHY OIL USA, INC./MERAUX REFINERY	2500 E ST. BERNARD HWY	MERAUX	St. Bernard Parish	LA
PET_NEI6130	PLACID REFINING CO LLC/PT ALLEN	1940 LA HWY 1, NORTH	PORT ALLEN	West Baton Rouge P	LA
PET_NEI6136	Valero Refining Co. - Krotz Springs	HIGHWAY 105 SOUTH	KROTZ SPRINGS	St. Landry Parish	LA
PET_NEI6166	Citgo Petroleum Corp. - Lake Charles	2 MI S	SULPHUR	Calcasieu Parish	LA
PET_NEI6375	ConocoPhillips Co. (prev. Phillips 66 Co.)	1400 Park Ave	Linden	Union County	NJ
PET_NEI6436	BP - Texas City	2401 5TH AVE. S.	TEXAS CITY	Galveston County	TX
PET_NEI6446	Alon USA Energy Inc. (prev. Alon USA LP)	I. 20 AT REFINERY ROAD	BIG SPRING	Howard County	TX
PET_NEI6475	Delek Refining Ltd (prev. LaGloria Oil & Gas Co.)	1702 E COMMERCE ST	TYLER	Smith County	TX
PET_NEI6519	ConocoPhillips Co. - Sweeny (prev. Phillips 66 Co.)	HWY 35 AND 524 AT OLD OCEAN	SWEENY	Brazoria County	TX
PET_NEI6617	Citgo Refining & Chemical Inc. - Corpus Christi West	7350 I. 37	CORPUS CHRISTI	Nueces County	TX
PET_NEI6963	ConocoPhillips Co. - Borger (prev. Phillips 66 Co.)	STATE HWY. SPUR 119 N.	BORGER	Hutchinson County	TX
PET_NEI7130	AGE Refining & Manufacturing	7811 S. PRESA ST.	SAN ANTONIO	Bexar County	TX
PET_NEI7134	Flint Hills Resources LP - Corpus Christi East	1700 NUECES BAY BOULEVARD	CORPUS CHRISTI	Nueces County	TX
PET_NEI7233	ExxonMobil Corp. - Beaumont	1795 Burt Street	BEAUMONT	Jefferson County	TX
PET_NEI7441	Motiva - Port Arthur	2100 HOUSTON AVE.	PORT ARTHUR	Jefferson County	TX
PET_NEI7781	ExxonMobil Corp. - Baytown	2800 DECKER DR	BAYTOWN	Harris County	TX
PET_NEI7973	South Hampton Resources Inc. (prev. South Hampton Refining Co.)	HWY. 418	SILSBEE	Hardin County	TX
PET_NEI7988	CITGO CORPUS CHRISTI REFINERY EAST PLANT	1801 NUECES BAY BLVD.	CORPUS CHRISTI	Nueces County	TX
PET_NEI8139	Valero Energy Corp. - McKee (prev. Diamond Shamrock Refining)	6701 FM 119	SUNRAY	Moore County	TX
PET_NEI8612	Gulf Atlantic Operations LLC (prev. Coastal Mobile Refining Co.)	200 VIADUCT RD.	CHICKASAW	Mobile County	AL
PET_NEI876	LION OIL COMPANY	1000 MCHENRY DRIVE	EL DORADO	Union County	AR
PET_NEI889	Suncor Energy USA - Commerce City (prev. Conoco Inc.)	5801 BRIGHTON BLVD	COMMERCE CITY	Adams County	CO
PET_NEICA0370	ConocoPhillips Co. - Wilmington (prev. Phillips 66 Co.)	1660 W ANAHEIM ST	WILMINGTON	Los Angeles County	CA
PET_NEICA0379	ConocoPhillips Co. - Carson (prev. Phillips 66 Co.)	1520 E SEPULVEDA BLVD	CARSON	Los Angeles County	CA

Table 1 - Facility Identification Information

Facility NEI ID	Facility Name	Address	City	County	State
PET_NEICA1057	Tricor Refining (prev. Golden Bear Oil Specialties)	1134 MANOR ST	OILDALE	Kern County	CA
PET_NEICA1910	Shell Oil Products US - Wilmington	2101 E PACIFIC COAST HWY	WILMINGTON	Los Angeles County	CA
PET_NEINJT$89	Valero Refining C0 - NJ	VALERO REFINING CO - NJ PAULSBORO REFINERY 800 BIL	PAULSBORO	Gloucester County	NJ
PET_NEINMT$12	Navajo Refining Co. - Lovington	5 MI SE OF LOVINGTION ON NM 18	LOVINGTON	Lea County	NM
PET_NEIOKT$11	Valero Refining Company - Oklahoma, Valero Ardmore Refinery	HWY. 142 & E. CAMERON RD.	ARDMORE	Carter County	OK
PET_NEIPRT$64	SHELL CHEMICAL YABUCOA INC.	RTE. 901 KM 2.7 CAMINO NUEVO WARD	YABUCOA	Yabucoa Municipio	PR
PET_NEIWYT$1	SILVER EAGLE REFINING-EVANSTON	2990 COUNTY RD. 180	EVANSTON	Uinta County	WY

Table 2 – Maximum Predicted HEM-3 Chronic Risks

Facility NEI ID	Chronic Risk [1]		
	Cancer MIR	Cancer Incidence	Noncancer Max HI
PET_NEI109	4.1E-06	8.1E-04	1.7E-02
PET_NEI11119	6.0E-07	4.1E-04	1.7E-03
PET_NEI11192	6.4E-07	9.2E-05	4.2E-03
PET_NEI11200	8.8E-07	2.6E-04	4.1E-02
PET_NEI11232	4.7E-06	3.0E-03	2.1E-02
PET_NEI113	5.3E-07	9.3E-05	2.3E-03
PET_NEI11449	9.1E-06	8.0E-04	1.9E-03
PET_NEI11450	1.7E-06	1.4E-04	6.5E-03
PET_NEI11574	5.2E-06	1.6E-04	2.0E-02
PET_NEI11663	4.4E-06	4.3E-04	1.4E-02
PET_NEI11715	0.0E+00	0.0E+00	0.0E+00
PET_NEI11885	1.8E-07	4.3E-05	2.9E-03
PET_NEI12044	6.0E-06	3.9E-04	2.3E-02
PET_NEI12084	4.3E-07	7.0E-05	9.9E-04
PET_NEI12458	9.8E-07	4.1E-05	2.1E-03
PET_NEI12459	1.0E-06	1.3E-05	4.3E-03
PET_NEI12460	8.6E-07	4.0E-05	2.9E-03
PET_NEI12464	1.0E-06	2.3E-05	5.1E-03
PET_NEI12480	6.0E-06	6.0E-04	2.5E-02
PET_NEI12486	4.3E-06	1.3E-05	1.0E-02
PET_NEI12711	2.84E-05	5.8E-03	1.0E-01
PET_NEI12790	3.8E-06	1.7E-04	1.6E-02
PET_NEI12791	9.8E-06	1.5E-03	1.7E-02
PET_NEI12968	1.9E-06	9.0E-05	1.5E-01
PET_NEI12969	1.2E-06	9.3E-05	5.0E-03
PET_NEI12988	1.90E-05	5.7E-04	3.7E-02
PET_NEI13322	1.3E-07	7.7E-06	1.3E-03
PET_NEI13371	5.2E-06	1.7E-05	1.8E-02
PET_NEI18372	3.9E-07	1.0E-04	1.5E-03
PET_NEI18394	1.1E-05	1.4E-04	6.6E-02
PET_NEI18406	8.6E-06	6.5E-05	2.6E-02
PET_NEI18408	1.4E-08	5.4E-08	6.1E-05
PET_NEI18415	2.4E-08	2.0E-07	1.0E-04
PET_NEI19587	4.8E-06	2.0E-03	3.6E-02
PET_NEI19834	1.9E-06	1.7E-04	2.6E-02
PET_NEI19869	1.4E-07	1.5E-06	6.0E-04
PET_NEI19870	5.6E-07	1.0E-04	3.6E-03
PET_NEI20103	5.7E-07	1.4E-05	3.6E-03
PET_NEI20154	6.5E-09	2.7E-07	1.2E-04
PET_NEI20174	1.2E-05	8.3E-04	4.9E-02
PET_NEI20467	1.4E-06	2.6E-04	1.1E-02
PET_NEI20616	3.4E-07	6.9E-05	1.8E-03
PET_NEI20797	3.2E-08	1.2E-05	1.4E-04
PET_NEI20966	7.2E-08	4.4E-05	2.6E-04
PET_NEI21034	7.6E-07	8.0E-04	2.6E-03
PET_NEI21130	2.4E-09	1.2E-06	5.2E-04
PET_NEI21466	1.5E-09	9.6E-07	3.2E-04
PET_NEI25450	9.9E-07	1.6E-04	2.7E-03
PET_NEI25464	4.6E-09	5.1E-07	2.0E-05
PET_NEI26101	4.1E-07	3.4E-05	7.2E-04
PET_NEI26218	2.9E-07	1.0E-04	1.7E-03
PET_NEI26473	9.3E-14	2.7E-11	3.6E-05
PET_NEI26489	4.3E-07	1.7E-05	1.8E-03

Table 2 – Maximum Predicted HEM-3 Chronic Risks

Facility NEI ID	Chronic Risk [1]		
	Cancer MIR	Cancer Incidence	Noncancer Max HI
PET_NEI26533	7.2E-08	9.5E-06	3.1E-04
PET_NEI2CA131003	3.7E-07	3.4E-04	1.2E-03
PET_NEI2CA254640	1.2E-07	1.4E-06	4.2E-04
PET_NEI2CA314628	**2.5E-06**	2.9E-04	4.0E-02
PET_NEI2KS125003	**2.7E-06**	4.9E-05	2.9E-02
PET_NEI2TX14199	4.3E-08	2.5E-06	1.9E-04
PET_NEI32353	2.3E-07	6.0E-06	1.1E-02
PET_NEI32762	**4.6E-06**	1.7E-04	9.2E-02
PET_NEI32801	**2.1E-06**	9.4E-05	1.5E-02
PET_NEI32864	**1.1E-05**	6.6E-04	3.4E-02
PET_NEI32997	3.8E-07	2.3E-06	4.4E-03
PET_NEI33007	1.3E-08	1.6E-06	2.0E-05
PET_NEI33008	**9.4E-06**	3.5E-04	4.0E-02
PET_NEI33010	1.6E-07	1.3E-05	5.5E-04
PET_NEI33030	**4.8E-06**	1.5E-04	2.0E-02
PET_NEI33031	**1.52E-05**	8.9E-04	1.2E-01
PET_NEI33039	**1.0E-05**	2.2E-05	7.9E-03
PET_NEI34022	5.4E-07	2.5E-04	1.5E-02
PET_NEI34050	**1.2E-05**	1.1E-03	3.4E-02
PET_NEI34057	**1.4E-05**	1.0E-03	2.2E-01
PET_NEI34061	2.9E-07	1.2E-06	4.3E-03
PET_NEI34062	**4.5E-06**	5.4E-06	2.1E-02
PET_NEI34069	2.0E-08	1.2E-06	6.3E-05
PET_NEI34862	**2.1E-06**	6.2E-04	8.8E-03
PET_NEI34863	3.3E-08	2.1E-05	1.8E-04
PET_NEI34872	6.9E-07	8.5E-05	2.6E-03
PET_NEI34873	**4.6E-06**	9.0E-04	2.0E-02
PET_NEI34898	**2.06E-05**	3.4E-04	1.8E-01
PET_NEI34907	4.5E-07	9.2E-06	1.7E-03
PET_NEI34912	1.8E-07	2.7E-06	7.9E-04
PET_NEI363	9.3E-07	3.6E-05	4.4E-03
PET_NEI371	4.9E-08	5.6E-07	2.7E-05
PET_NEI40371	**1.0E-05**	1.1E-04	2.7E-02
PET_NEI404	4.2E-08	3.7E-07	3.4E-03
PET_NEI40531	**8.3E-06**	4.2E-05	5.1E-02
PET_NEI40625	4.3E-07	8.3E-05	1.8E-03
PET_NEI40723	**2.4E-06**	8.1E-04	2.0E-02
PET_NEI40732	**5.0E-06**	1.7E-05	2.7E-02
PET_NEI415	**1.6E-06**	2.4E-04	6.6E-03
PET_NEI41591	**2.0E-06**	2.6E-04	1.1E-02
PET_NEI41771	**1.3E-05**	4.6E-04	4.6E-02
PET_NEI41863	**1.5E-06**	1.6E-04	1.7E-02
PET_NEI41864	3.8E-07	6.0E-05	1.1E-03
PET_NEI41865	1.9E-08	3.9E-06	8.0E-05
PET_NEI42016	**2.2E-06**	8.1E-05	2.3E-02
PET_NEI42020	**4.8E-06**	1.7E-04	1.9E-02
PET_NEI42025	**3.0E-06**	8.3E-05	1.1E-02
PET_NEI42040	**1.47E-05**	1.3E-04	2.8E-01
PET_NEI42081	1.4E-07	4.8E-05	4.3E-04
PET_NEI42309	**1.501E-05**	6.6E-05	6.8E-02
PET_NEI42370	5.1E-08	3.3E-05	3.2E-04
PET_NEI42381	**5.1E-06**	4.9E-05	4.1E-02
PET_NEI42382	1.9E-07	6.1E-06	7.2E-04

Table 2 – Maximum Predicted HEM-3 Chronic Risks

Facility NEI ID	Chronic Risk [1]		
	Cancer MIR	Cancer Incidence	Noncancer Max HI
PET_NEI42413	**1.6E-06**	1.3E-05	5.2E-03
PET_NEI42425	9.8E-07	2.2E-05	4.1E-03
PET_NEI42583	9.6E-08	3.3E-06	4.1E-04
PET_NEI43243	**1.6E-06**	6.2E-06	6.8E-03
PET_NEI46556	**6.0E-06**	4.0E-04	3.7E-02
PET_NEI46752	**1.8E-06**	3.2E-05	7.7E-03
PET_NEI46764	1.5E-07	2.5E-06	1.0E-03
PET_NEI49781	7.4E-07	2.3E-05	3.1E-03
PET_NEI53702	**2.1E-06**	4.8E-04	8.8E-03
PET_NEI53718	1.8E-07	6.1E-05	7.4E-04
PET_NEI55835	1.7E-08	1.9E-06	7.3E-05
PET_NEI6018	1.9E-07	1.2E-05	1.0E-03
PET_NEI6022	**9.7E-06**	1.6E-03	7.1E-02
PET_NEI6062	**4.6E-06**	2.2E-04	2.6E-02
PET_NEI6084	1.2E-06	2.3E-05	6.8E-03
PET_NEI6087	**1.2E-05**	5.1E-05	1.9E-01
PET_NEI6095	**1.3E-05**	4.5E-04	2.5E-02
PET_NEI6116	**7.5E-06**	1.2E-04	2.8E-02
PET_NEI6123	**5.0E-06**	1.5E-03	2.0E-02
PET_NEI6127	**2.5E-06**	8.2E-05	1.0E-02
PET_NEI6130	**5.8E-06**	2.5E-04	2.0E-02
PET_NEI6136	**5.8E-06**	1.2E-04	8.6E-03
PET_NEI6166	**7.3E-06**	9.7E-04	2.3E-02
PET_NEI6375	1.4E-06	1.7E-03	4.8E-03
PET_NEI6436	**1.2E-05**	1.7E-03	4.6E-02
PET_NEI6446	**2.4E-06**	1.8E-05	9.1E-03
PET_NEI6475	**1.3E-05**	2.2E-04	5.6E-02
PET_NEI6519	**5.3E-06**	4.0E-05	2.3E-02
PET_NEI6617	6.8E-08	1.1E-05	5.1E-03
PET_NEI6963	2.9E-07	1.9E-05	2.6E-03
PET_NEI7130	1.3E-07	1.4E-05	8.7E-04
PET_NEI7134	**2.8E-06**	1.4E-04	1.4E-02
PET_NEI7233	**5.3E-06**	7.9E-04	1.1E-01
PET_NEI7441	8.1E-07	8.8E-05	4.3E-03
PET_NEI7781	**6.0E-06**	1.0E-03	5.2E-02
PET_NEI7973	4.7E-09	2.0E-07	2.0E-05
PET_NEI7988	**6.3E-06**	4.7E-04	6.1E-02
PET_NEI8139	1.5E-06	1.6E-06	4.7E-03
PET_NEI8612	6.5E-07	1.6E-04	5.2E-03
PET_NEI876	**1.45E-05**	3.4E-04	3.5E-02
PET_NEI889	9.9E-07	9.9E-05	3.8E-03
PET_NEICA0370363	**2.0E-06**	4.1E-04	1.4E-02
PET_NEICA0379991	2.6E-07	2.1E-04	2.7E-03
PET_NEICA10578	3.1E-07	9.8E-06	1.8E-02
PET_NEICA1910268	**1.1E-05**	1.9E-03	1.4E-02
PET_NEINJT$891	1.2E-06	4.5E-04	6.2E-03
PET_NEINMT$12478	2.4E-07	1.4E-05	2.1E-03
PET_NEIOKT$11009	**9.2E-06**	6.0E-05	3.8E-02
PET_NEIPRT$64	**1.2E-05**	1.3E-03	1.1E-01
PET_NEIWYT$12156	1.5E-09	1.3E-07	6.4E-06

[1] **BOLD/Shaded RED** indicates a cancer risk great than 1 in a million or a noncancer HI greater than 1

Table 3 – Maximum Predicted HEM-AERMOD Acute Risks

Facility NEI ID	Maximum Hazard Quotient [1]				
	AEGL1	AEGL2	ERPG1	ERPG2	REL
PET_NEI109	3E-03	6E-04	3E-03	6E-04	4E-01
PET_NEI11119	1E-02	9E-04	1E-03	4E-04	1E-01
PET_NEI11192	3E-04	4E-05	2E-04	1E-04	3E-02
PET_NEI11200	3E-02	5E-03	7E-03	2E-03	9E-01
PET_NEI11232	5E-03	6E-04	5E-03	4E-04	7E-01
PET_NEI113	8E-03	7E-04	5E-03	1E-03	7E-01
PET_NEI11449	9E-03	4E-03	9E-03	2E-03	2E-01
PET_NEI11450	3E-04	4E-05	2E-04	7E-05	3E-02
PET_NEI11574	3E-02	1E-03	3E-02	1E-03	2E-01
PET_NEI11663	9E-03	6E-04	9E-03	1E-03	1E+00
PET_NEI11715	0E+00	0E+00	0E+00	0E+00	0E+00
PET_NEI11885	2E-04	2E-05	1E-05	1E-06	0E+00
PET_NEI12044	5E-02	3E-03	5E-02	3E-03	**7E+00**
PET_NEI12084	1E-01	6E-03	1E-01	6E-03	6E-01
PET_NEI12458	3E-01	1E-02	3E-01	1E-02	1E+00
PET_NEI12459	1E-03	1E-04	1E-03	2E-04	2E-01
PET_NEI12460	2E-01	1E-02	2E-01	1E-02	8E-01
PET_NEI12464	2E-03	3E-04	1E-03	5E-04	2E-01
PET_NEI12480	7E-03	5E-04	7E-03	6E-04	9E-01
PET_NEI12486	**4E+00**	2E-01	**4E+00**	2E-01	**1E+01**
PET_NEI12711	3E-03	2E-04	1E-03	5E-04	2E-01
PET_NEI12790	3E-03	2E-04	3E-03	2E-04	4E-01
PET_NEI12791	4E-01	3E-02	4E-01	3E-02	5E-01
PET_NEI12968	6E-03	4E-03	6E-03	4E-03	8E-02
PET_NEI12969	8E-03	5E-04	8E-03	5E-04	3E-01
PET_NEI12988	2E-01	9E-03	2E-01	9E-03	**2E+00**
PET_NEI13322	8E-04	5E-05	8E-04	5E-05	1E-01
PET_NEI13371	5E-03	4E-04	5E-03	8E-04	7E-01
PET_NEI18372	5E-03	3E-04	5E-03	6E-04	7E-01
PET_NEI18394	5E-03	6E-04	5E-03	1E-03	7E-01
PET_NEI18406	9E-04	6E-05	9E-04	9E-05	1E-01
PET_NEI18408	3E-06	2E-07	3E-06	2E-07	4E-04
PET_NEI18415	5E-05	3E-06	5E-05	3E-06	6E-03
PET_NEI19587	1E-02	2E-03	1E-02	4E-03	1E+00
PET_NEI19834	6E-04	9E-05	3E-04	6E-05	4E-02
PET_NEI19869	1E-04	3E-05	1E-04	3E-05	1E-02
PET_NEI19870	3E-03	2E-03	3E-03	2E-03	8E-02
PET_NEI20103	7E-04	1E-04	7E-04	3E-04	7E-02
PET_NEI20154	1E-05	2E-06	8E-06	3E-06	5E-04
PET_NEI20174	9E-03	6E-04	9E-03	6E-04	1E+00
PET_NEI20467	4E-04	1E-04	6E-04	1E-04	2E-02
PET_NEI20616	1E-04	2E-05	1E-04	4E-05	1E-02
PET_NEI20797	4E-05	2E-06	4E-05	2E-06	5E-03
PET_NEI20966	4E-04	7E-05	4E-04	2E-04	8E-03
PET_NEI21034	4E-03	1E-04	4E-03	1E-04	7E-02
PET_NEI21130	3E-03	2E-04	3E-03	2E-04	3E-02
PET_NEI21466	1E-02	5E-04	1E-02	5E-04	4E-02
PET_NEI25450	2E-02	2E-03	7E-03	3E-03	7E-01
PET_NEI25464	2E-05	1E-06	2E-05	1E-06	2E-03
PET_NEI26101	2E-04	1E-05	2E-04	1E-05	2E-02
PET_NEI26218	1E-02	1E-03	2E-03	3E-04	2E-01
PET_NEI26473	1E-06	2E-06	1E-06	5E-07	3E-05
PET_NEI26489	4E-04	2E-05	4E-04	2E-05	5E-02

Table 3 – Maximum Predicted HEM-AERMOD Acute Risks

Facility NEI ID	Maximum Hazard Quotient [1]				
	AEGL1	AEGL2	ERPG1	ERPG2	REL
PET_NEI26533	3E-03	2E-04	3E-03	2E-04	4E-01
PET_NEI2CA131003	2E-03	1E-04	2E-03	1E-04	1E-01
PET_NEI2CA254640	2E-04	1E-05	2E-04	2E-05	2E-02
PET_NEI2CA314628	8E-03	1E-03	1E-03	3E-04	2E-01
PET_NEI2KS125003	**6E+00**	3E-01	**6E+00**	3E-01	**2E+01**
PET_NEI2TX14199	6E-05	4E-06	6E-05	4E-06	8E-03
PET_NEI32353	4E-01	2E-02	4E-01	2E-02	1E+00
PET_NEI32762	1E-01	5E-03	1E-01	5E-03	6E-01
PET_NEI32801	3E-02	1E-03	3E-02	1E-03	4E-01
PET_NEI32864	3E-01	2E-02	3E-01	2E-02	**5E+01**
PET_NEI32997	7E-05	4E-06	7E-05	4E-06	9E-03
PET_NEI33007	2E-05	3E-06	3E-06	1E-06	5E-04
PET_NEI33008	2E-02	4E-03	2E-02	9E-03	5E-01
PET_NEI33010	1E-03	1E-04	1E-03	1E-04	2E-01
PET_NEI33030	3E-03	2E-04	3E-03	2E-04	4E-01
PET_NEI33031	3E-03	2E-04	3E-03	5E-04	2E-01
PET_NEI33039	3E-04	5E-05	3E-04	4E-05	4E-02
PET_NEI34022	1E-01	7E-03	1E-01	7E-03	1E+00
PET_NEI34050	2E-03	3E-04	1E-03	4E-04	2E-01
PET_NEI34057	3E-01	2E-02	3E-01	2E-02	**6E+00**
PET_NEI34061	4E-03	2E-04	4E-03	2E-04	4E-02
PET_NEI34062	7E-02	5E-03	7E-02	5E-03	9E-01
PET_NEI34069	1E-04	7E-06	1E-04	7E-06	1E-02
PET_NEI34862	2E-03	3E-04	2E-03	1E-04	3E-01
PET_NEI34863	2E-04	3E-05	2E-04	2E-05	3E-02
PET_NEI34872	9E-04	8E-05	2E-04	7E-05	1E-02
PET_NEI34873	4E-03	3E-04	4E-03	7E-04	6E-01
PET_NEI34898	1E-02	4E-04	1E-02	1E-03	2E-01
PET_NEI34907	9E-04	6E-05	9E-04	1E-04	1E-01
PET_NEI34912	2E-04	3E-05	2E-04	3E-05	3E-02
PET_NEI363	1E-03	1E-04	1E-03	1E-04	2E-01
PET_NEI371	0E+00	0E+00	0E+00	0E+00	0E+00
PET_NEI40371	1E-01	6E-03	1E-01	6E-03	**3E+00**
PET_NEI404	8E-02	3E-03	8E-02	3E-03	3E-01
PET_NEI40531	9E-02	6E-03	9E-02	6E-03	1E+00
PET_NEI40625	1E-03	7E-05	1E-03	1E-04	1E-01
PET_NEI40723	4E-04	5E-05	4E-04	1E-04	5E-02
PET_NEI40732	4E-04	3E-05	4E-04	7E-05	6E-02
PET_NEI415	5E-03	3E-04	5E-03	3E-04	2E-01
PET_NEI41591	1E-01	5E-03	1E-01	5E-03	4E-01
PET_NEI41771	2E-02	2E-03	2E-02	2E-03	**3E+00**
PET_NEI41863	4E-02	2E-03	4E-02	2E-03	3E-01
PET_NEI41864	4E-03	3E-04	2E-03	1E-04	3E-01
PET_NEI41865	2E-03	1E-04	2E-03	1E-04	2E-01
PET_NEI42016	9E-01	4E-02	9E-01	4E-02	**3E+00**
PET_NEI42020	2E-03	1E-04	2E-03	3E-04	3E-01
PET_NEI42025	2E-03	2E-04	2E-03	3E-04	3E-01
PET_NEI42040	2E-02	3E-03	5E-03	2E-03	6E-01
PET_NEI42081	2E-03	2E-04	2E-03	5E-04	2E-01
PET_NEI42309	2E-02	1E-03	5E-04	1E-04	6E-02
PET_NEI42370	2E-04	3E-05	2E-04	8E-05	2E-02
PET_NEI42381	1E-02	8E-04	1E-02	1E-03	6E-01
PET_NEI42382	6E-04	9E-05	6E-04	1E-04	8E-02

Table 3 – Maximum Predicted HEM-AERMOD Acute Risks

Facility NEI ID	Maximum Hazard Quotient [1]				
	AEGL1	AEGL2	ERPG1	ERPG2	REL
PET_NEI42413	3E-03	2E-04	3E-03	3E-04	3E-01
PET_NEI42425	1E-02	6E-04	1E-02	6E-04	8E-02
PET_NEI42583	4E-04	4E-05	4E-04	7E-05	5E-02
PET_NEI43243	3E-03	2E-04	3E-03	2E-04	4E-01
PET_NEI46556	3E-02	4E-03	2E-02	7E-03	1E+00
PET_NEI46752	1E-02	2E-03	1E-02	5E-03	4E-01
PET_NEI46764	3E-04	4E-05	2E-04	7E-05	2E-02
PET_NEI49781	1E-03	6E-05	1E-03	6E-05	1E-01
PET_NEI53702	2E-02	1E-03	2E-02	1E-03	**2E+00**
PET_NEI53718	8E-03	1E-03	8E-03	2E-03	1E+00
PET_NEI55835	2E-04	3E-05	2E-04	2E-05	2E-02
PET_NEI6018	2E-04	1E-05	2E-04	2E-05	2E-02
PET_NEI6022	9E-01	7E-02	9E-01	7E-02	1E+00
PET_NEI6062	3E-03	8E-04	3E-03	5E-04	4E-01
PET_NEI6084	4E-03	3E-04	4E-04	1E-04	3E-02
PET_NEI6087	8E-01	3E-02	8E-01	3E-02	**3E+00**
PET_NEI6095	2E-03	2E-04	2E-03	2E-04	2E-01
PET_NEI6116	6E-03	2E-03	6E-03	2E-03	8E-01
PET_NEI6123	6E-03	6E-04	6E-03	4E-04	8E-01
PET_NEI6127	7E-04	5E-05	7E-04	6E-05	1E-01
PET_NEI6130	2E-02	1E-03	2E-02	1E-03	7E-01
PET_NEI6136	1E-01	9E-03	1E-01	9E-03	**2E+00**
PET_NEI6166	3E-01	3E-02	3E-01	3E-02	**2E+00**
PET_NEI6375	1E-03	1E-04	8E-04	1E-04	1E-01
PET_NEI6436	2E-02	1E-03	2E-02	2E-03	**2E+00**
PET_NEI6446	1E-02	1E-03	1E-02	3E-03	**2E+00**
PET_NEI6475	5E-03	5E-04	5E-03	1E-03	6E-01
PET_NEI6519	4E-02	2E-03	4E-02	3E-03	**5E+00**
PET_NEI6617	3E-02	3E-03	3E-02	3E-03	3E-01
PET_NEI6963	3E-01	1E-02	3E-01	1E-02	1E+00
PET_NEI7130	2E-03	3E-04	1E-03	5E-04	2E-01
PET_NEI7134	2E-03	4E-04	2E-03	4E-04	3E-01
PET_NEI7233	4E-02	7E-03	4E-02	2E-02	9E-01
PET_NEI7441	1E-02	2E-03	4E-03	2E-03	**5E+00**
PET_NEI7781	8E-03	5E-04	8E-03	5E-04	1E+00
PET_NEI7973	3E-05	3E-06	3E-05	2E-06	4E-03
PET_NEI7988	6E-02	4E-03	6E-02	1E-02	**7E+00**
PET_NEI8139	2E-03	3E-04	2E-03	4E-04	3E-01
PET_NEI8612	1E-03	8E-05	1E-03	8E-05	2E-01
PET_NEI876	3E-04	2E-03	0E+00	0E+00	0E+00
PET_NEI889	1E-02	8E-04	1E-02	8E-04	1E-01
PET_NEICA0370363	5E-04	3E-05	5E-04	5E-05	5E-02
PET_NEICA0379991	7E-05	1E-05	4E-05	1E-05	2E-03
PET_NEICA10578	4E-03	5E-04	8E-04	3E-04	9E-02
PET_NEICA1910268	5E-03	7E-04	5E-03	7E-04	8E-02
PET_NEINJT$891	3E-02	1E-03	3E-02	1E-03	3E-01
PET_NEINMT$12478	8E-04	5E-05	8E-04	1E-04	1E-01
PET_NEIOKT$11009	4E-01	2E-02	4E-01	2E-02	**6E+00**
PET_NEIPRT$64	1E-02	9E-04	1E-02	1E-03	**2E+00**
PET_NEIWYT$12156	1E-04	7E-06	1E-04	7E-06	1E-02

[1] **BOLD RED** indicates a cancer risk great than 1 in a million or a noncancer risk greater than 1

Table 4 – Maximum Predicted Acute Risks Greater than 1 (Refined Approach)

Figure No.	Facility NEI ID	Pollutant	Criteria	HEM-3 (Screening)	Refined Results [1]	Refined Modeling Notes [2]
8-1	PET_NEI12044	Benzene	REL	7	1	NNW of facility
8-2	PET_NEI12486	Glycol Ethers	REL	5	1	W of facility
8-3	PET_NEI12486	Hydrofluoric acid	REL	15	4	W of facility
8-4	PET_NEI12486	Hydrofluoric acid	AEGL-1	4	1	W of facility
8-5	PET_NEI12988	Benzene	REL	2	1	NNW of facility
8-6	PET_NEI2KS125003	Hydrofluoric acid	REL	22	5	E of facility
8-7	PET_NEI2KS125003	Hydrofluoric acid	AEGL-1	6	2	E of facility
8-8	PET_NEI32864	Benzene	REL	45	8	E of facility
8-9	PET_NEI34057	Benzene	REL	6	<1	
8-10	PET_NEI34057	Formaldehyde	REL	4	<1	
8-11	PET_NEI34057	Methanol	REL	2	<1	
8-12	PET_NEI34057	p-Xylene	REL	3	<1	
8-13	PET_NEI40371	Glycol Ethers	REL	3	1	E of facility
8-14	PET_NEI41771	Benzene	REL	3	2	W of facility
8-15	PET_NEI42016	Hydrofluoric acid	REL	3	2	ESE of facility
8-16	PET_NEI53702	Benzene	REL	2	<1	
8-17	PET_NEI6087	Hydrofluoric acid	REL	3	<1	
8-18	PET_NEI6136	Formaldehyde	REL	2	1	WSW of facility
8-19	PET_NEI6166	Benzene	REL	2	1	SE of facility
NA	PET_NEI6436	Benzene	REL	2	2	no refinement; receptor is a census block
8-20	PET_NEI6446	Benzene	REL	2	<1	
8-21	PET_NEI6519	Benzene	REL	5	<1	
8-22	PET_NEI7441	Glycol Ethers	REL	5	<1	
8-23	PET_NEI7988	Benzene	REL	7	1	S of facility
8-24	PET_NEIOKT$11009	Benzene	REL	6	4	E of facility
8-25	PET_NEIPRT$64	Benzene	REL	2	2	W of facility

[1] Facilites with a HEM-3 screening acute value greater than 1 were remodeled with a more refined approach

[2] Indicates offsite impacts using aerial photographs of facility

Appendix E
Refinement of acute exposure estimates at petroleum refining facilities and Portland cement facilities

Appendix E1
Refined Acute Assessment for Petroleum Refineries

Initial acute screening risk calculations were performed with the HEM-3 model. HEM-3 estimates acute (1-hour) impacts at both polar and census block receptors. It is assumed for this short period of time that an exposed individual could be located at any offsite location. The lack of readily available detailed property boundary information for many of the facilities evaluated made it difficult to determine whether receptors were on- or offsite. In the absence of such information, the first ring of polar receptors was placed 100 meters from the plant center for many facilities. However, these polar rings often transected onsite locations, preventing public access to exposures at these levels and thereby overestimating exposures. The screening analysis indicated that 20 facilities had the potential to exceed a 1-hour reference value for one or more pollutants. To refine the analysis for these 20 facilities, the polar receptors for each facility were overlaid on an aerial photograph of the facility to determine the offsite receptor with the highest 1-hour exposure. Figures E1-1 through E1-25 depict the modeled acute hazard quotients for these facilities. Table E1-1 summarizes the results of this refinement by listing the modeled maximum screening and refined (offsite) hazard quotient values.

Table E1-1 – Maximum Modeled Acute Hazard Quotients (Refined Approach)

Figure No.	Facility NEI ID	Pollutant	Criteria	Screening HQ	Refined HQ [1]	Refined Modeling Notes
E1-1	PET_NEI12044	Benzene	REL	7	1	NNW of facility
E1-2	PET_NEI12486	Glycol Ethers	REL	5	1	W of facility
E1-3	PET_NEI12486	Hydrofluoric acid	REL	15	4	W of facility
E1-4	PET_NEI12486	Hydrofluoric acid	AEGL-1	4	1	W of facility
E1-5	PET_NEI12988	Benzene	REL	2	1	NNW of facility
E1-6	PET_NEI2KS125003	Hydrofluoric acid	REL	22	5	E of facility
E1-7	PET_NEI2KS125003	Hydrofluoric acid	AEGL-1	6	2	E of facility
E1-8	PET_NEI32864	Benzene	REL	45	8	E of facility
E1-9	PET_NEI34057	Benzene	REL	6	<1	
E1-10	PET_NEI34057	Formaldehyde	REL	4	<1	
E1-11	PET_NEI34057	Methanol	REL	2	<1	
E1-12	PET_NEI34057	p-Xylene	REL	3	<1	
E1-13	PET_NEI40371	Glycol Ethers	REL	3	1	E of facility
E1-14	PET_NEI41771	Benzene	REL	3	2	W of facility
E1-15	PET_NEI42016	Hydrofluoric acid	REL	3	2	ESE of facility
E1-16	PET_NEI53702	Benzene	REL	2	<1	
E1-17	PET_NEI6087	Hydrofluoric acid	REL	3	<1	
E1-18	PET_NEI6136	Formaldehyde	REL	2	1	WSW of facility
E1-19	PET_NEI6166	Benzene	REL	2	1	SE of facility
NA	PET_NEI6436	Benzene	REL	2	2	no refinement; receptor is a census block
E1-20	PET_NEI6446	Benzene	REL	2	<1	
E1-21	PET_NEI6519	Benzene	REL	5	<1	
E1-22	PET_NEI7441	Glycol Ethers	REL	5	<1	
E1-23	PET_NEI7988	Benzene	REL	7	1	S of facility
E1-24	PET_NEIOKT$11009	Benzene	REL	6	4	E of facility
E1-25	PET_NEIPRT$64	Benzene	REL	2	2	W of facility

[1] Where facilities had a HEM-3 screening acute HQ greater than 1, HQ values at polar receptors were overlaid on aerial photographs to determine the maximum offsite value.

Figure E1-1. NEI12044 Benzene Acute HQ values (based on REL)

Figure E1-2. NEI12486 Glycol Ethers Acute HQ values (based on REL)

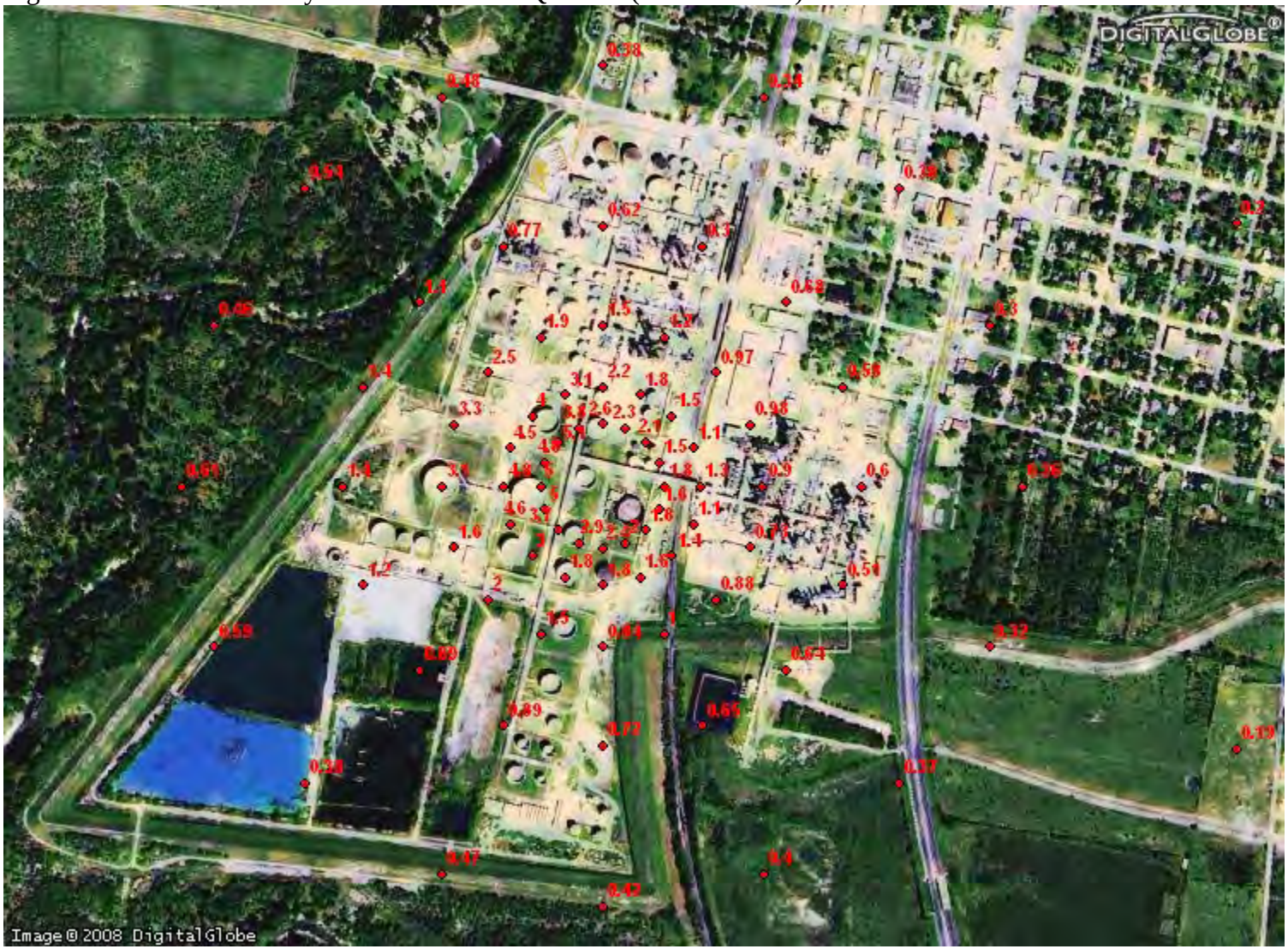

Figure E1-3. NEI12486 Hydrofluoric Acid Acute HQ values (based on REL)

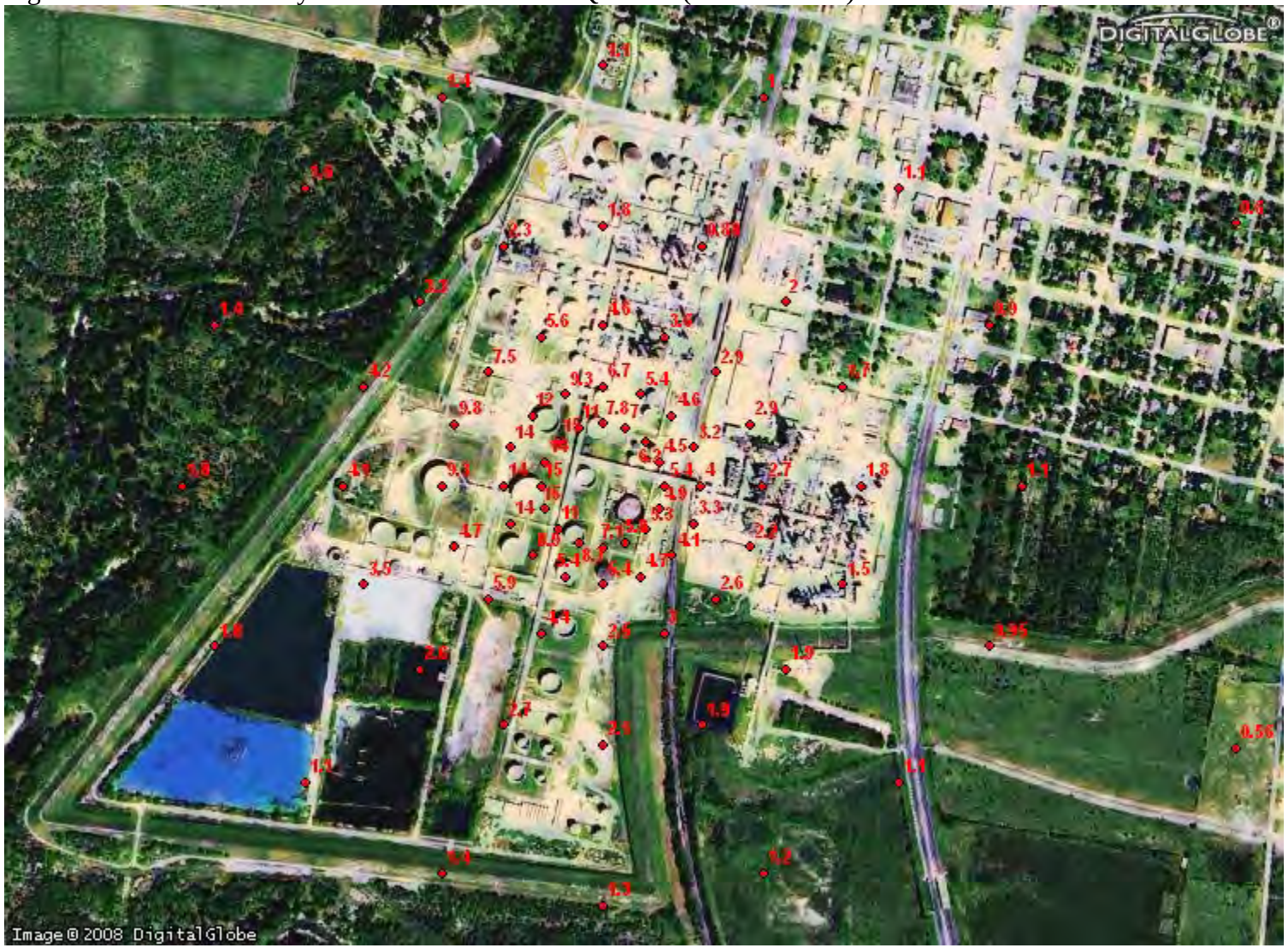

Figure E1-4. NEI12486 Hydrofluoric Acid Acute HQ values (based on AEGL-1)

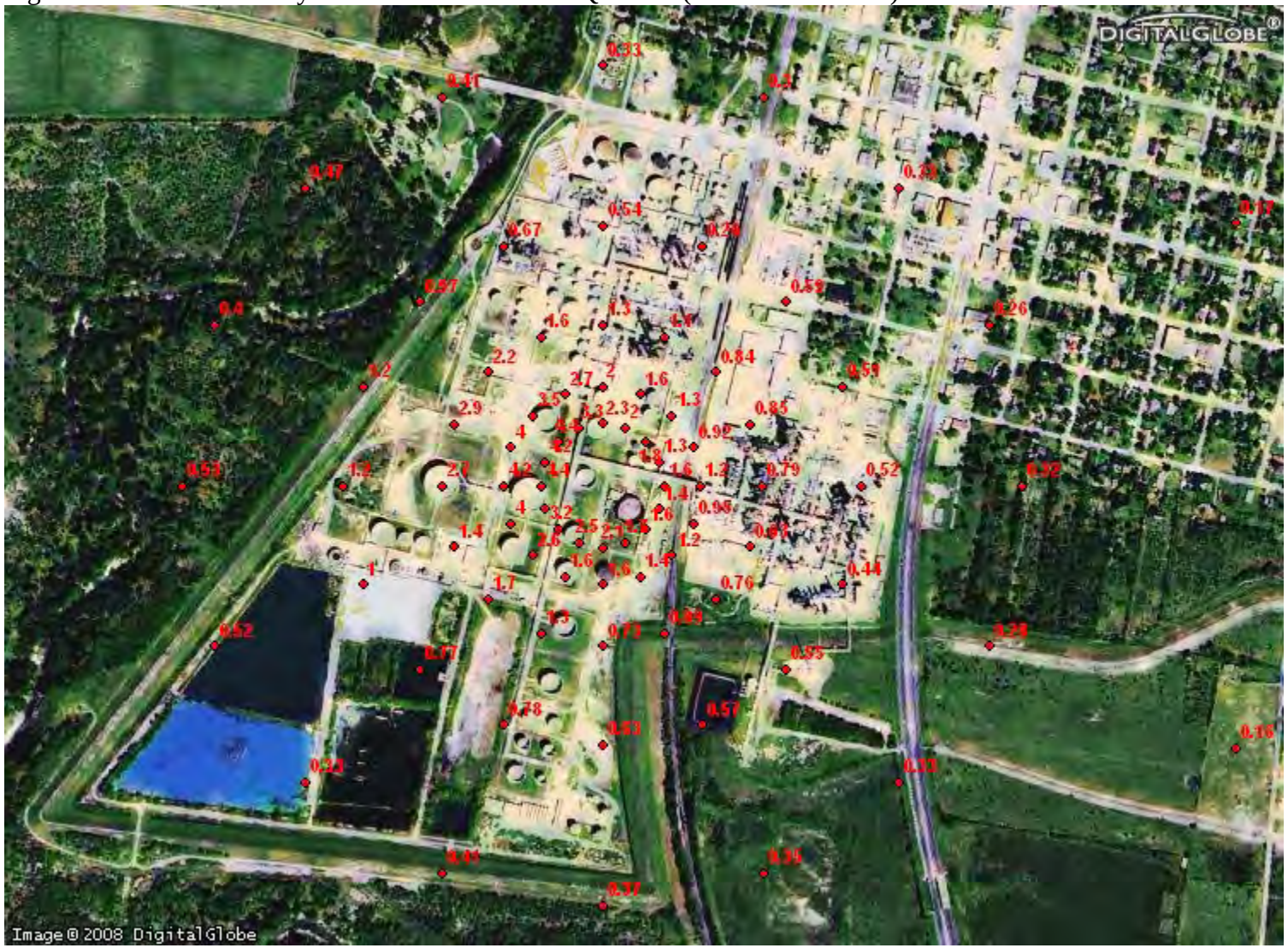

Figure E1-5. NEI12988 Benzene Acute HQ values (based on REL)

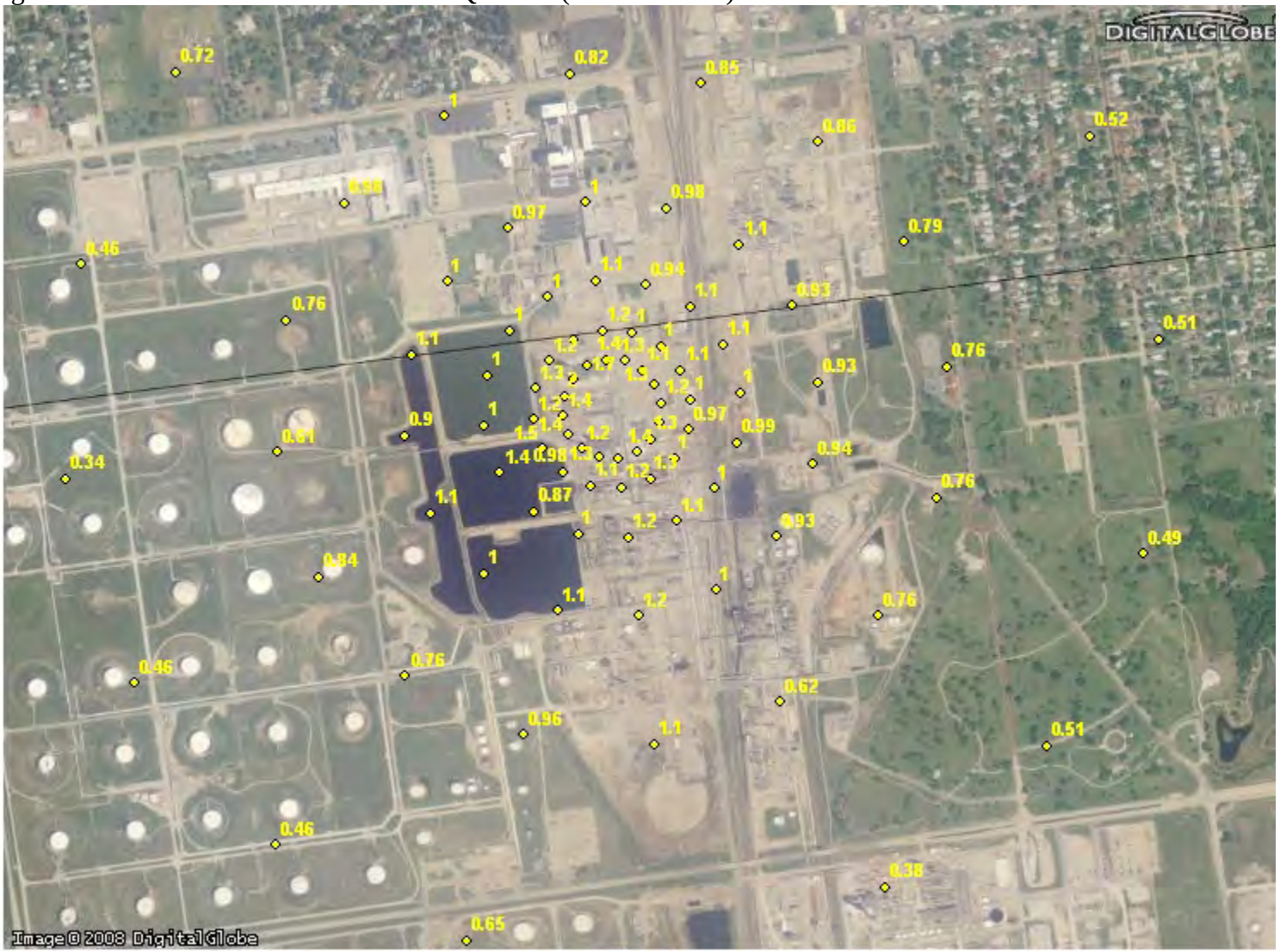

Figure E1-6. NEI2KS125003 Hydrofluoric Acid Acute HQ values (based on REL)

Figure E1-7. NEI2KS125003 Hydrofluoric Acid Acute HQ values (based on AEGL-1)

Figure E1-8. NEI32864 Benzene Acute HQ values (based on REL)

Figure E1-9. NEI34057 Benzene Acute HQ values (based on REL)

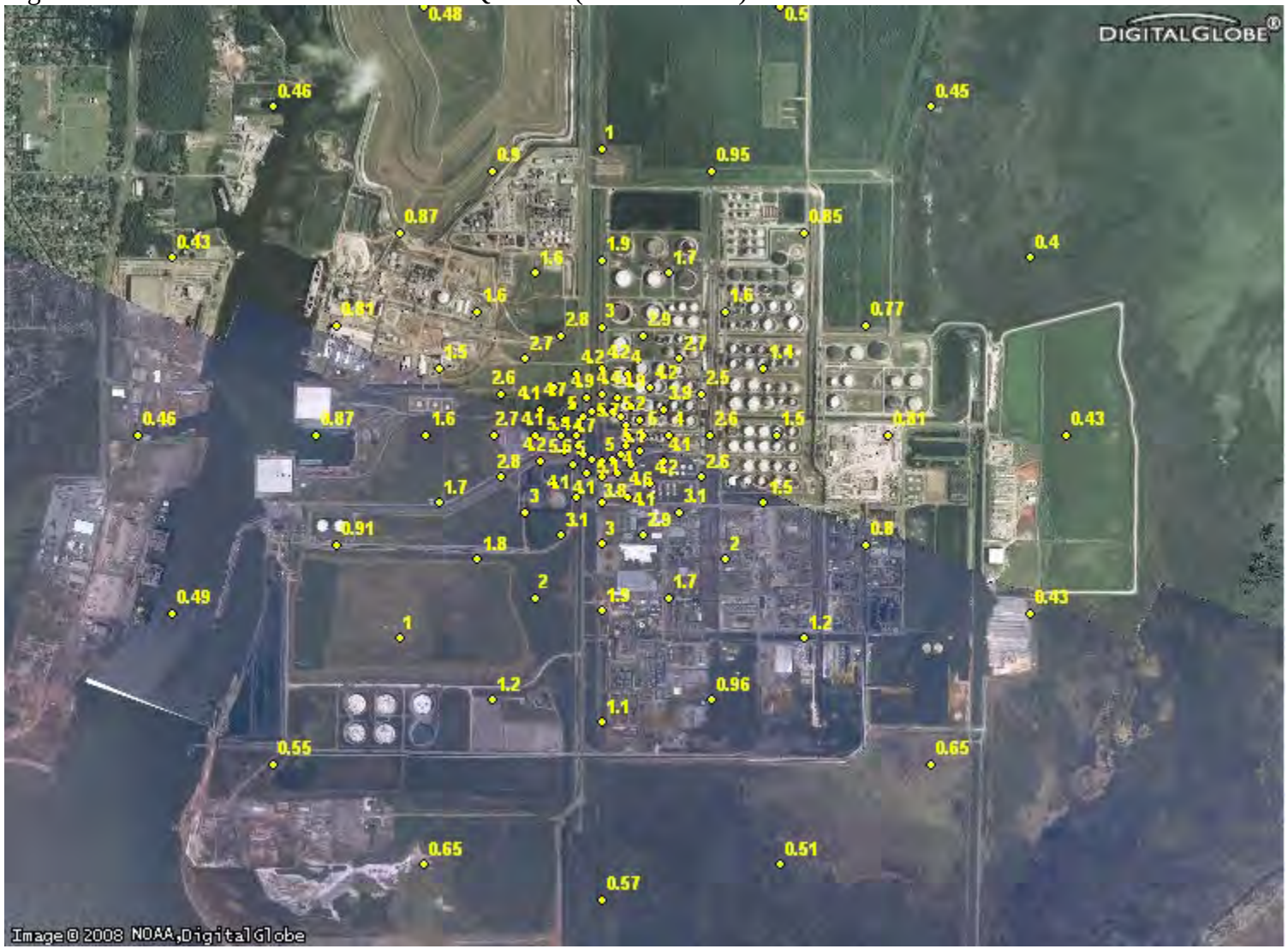

Figure E1-10. NEI34057 Formaldehyde Acute HQ values (based on REL)

Figure E1-11. NEI34057 Methanol Acute HQ values (based on REL)

Figure E1-12. NEI34057 p-Xylene Acute HQ values (based on REL)

Figure E1-13. NEI40371 Glycol Ethers Acute HQ values (based on REL)

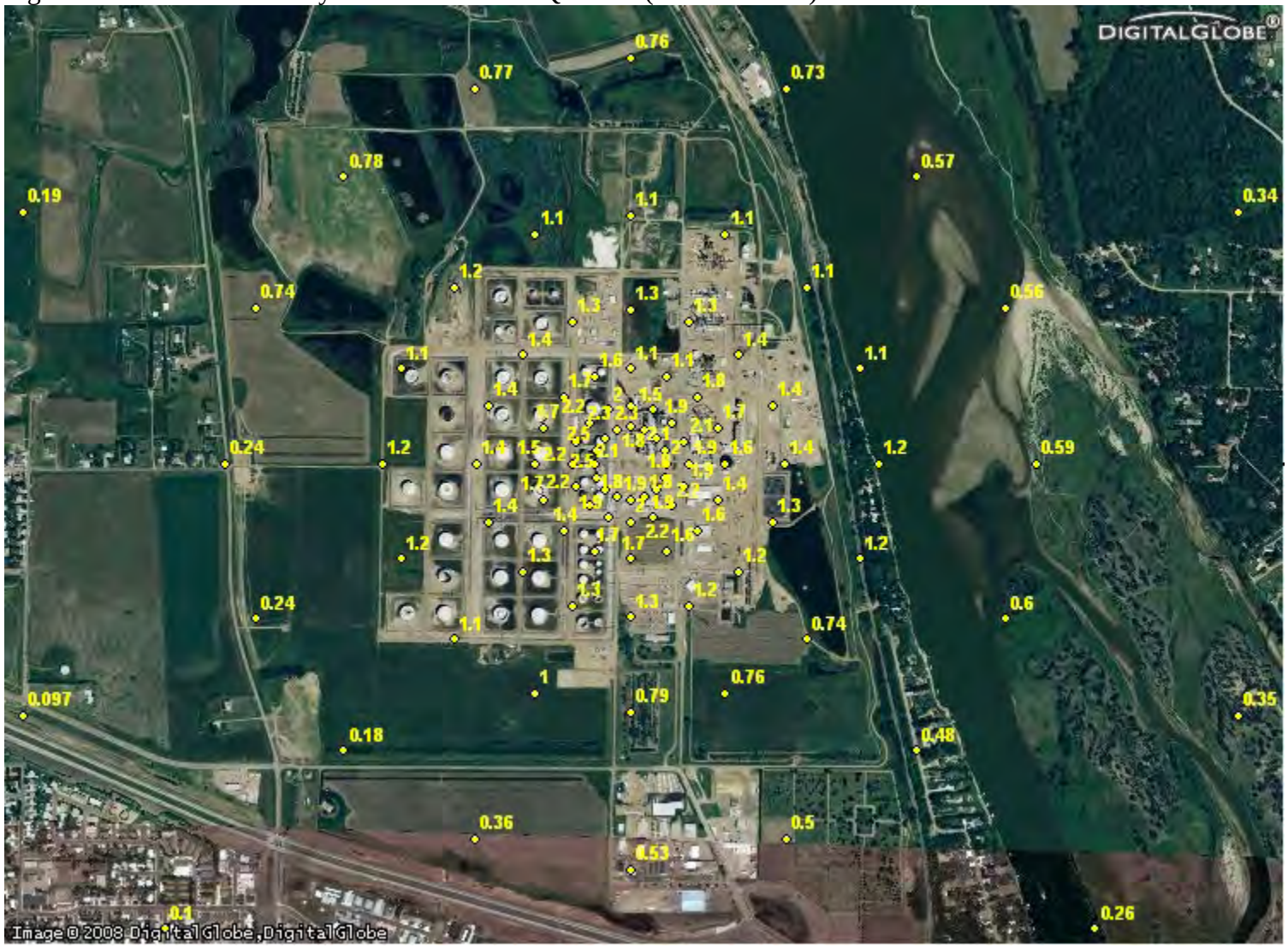

Figure E1-14. NEI41771 Benzene Acute HQ values (based on REL)

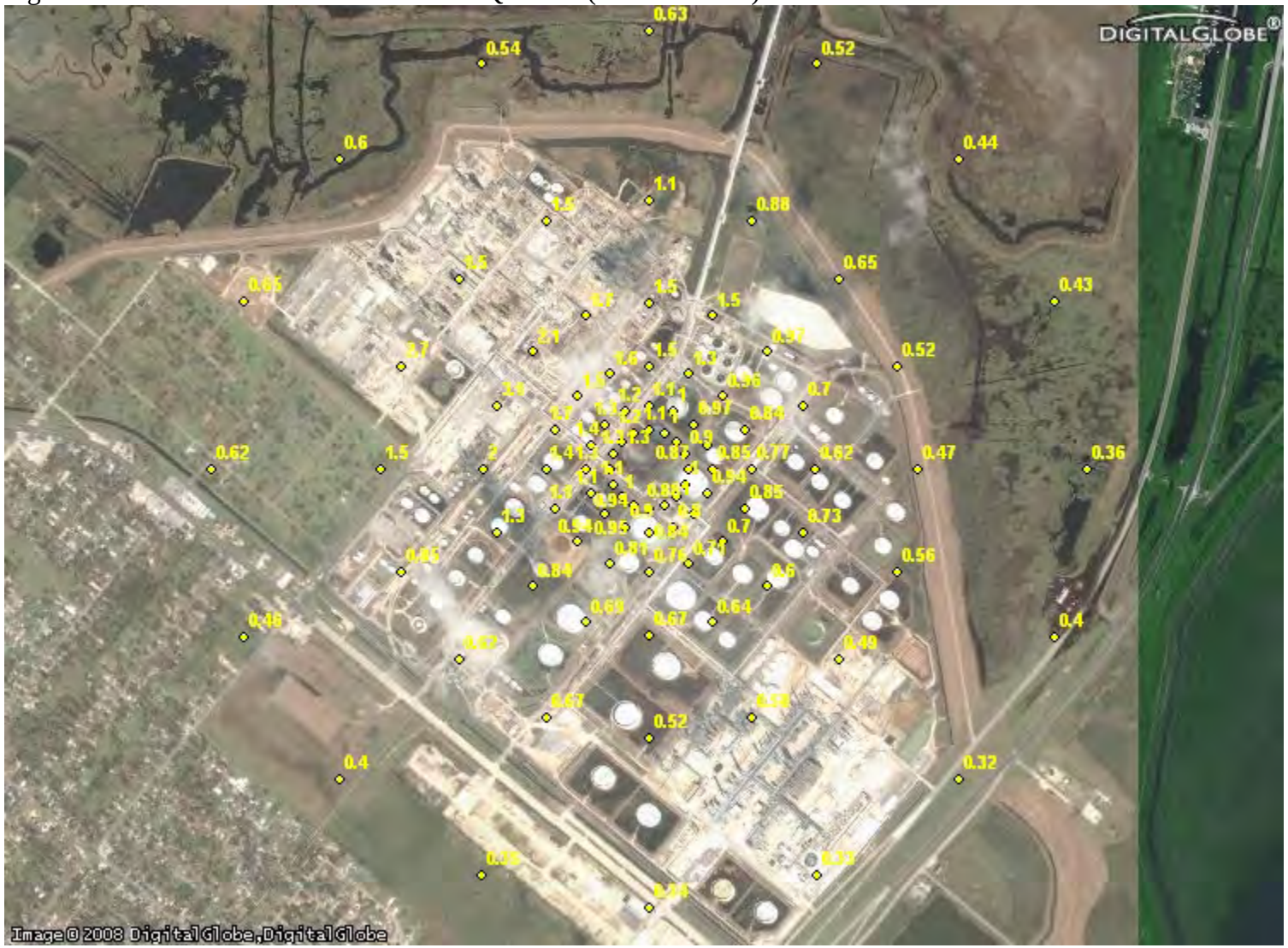

Figure E1-15. NEI42016 Hydrofluoric Acid Acute HQ values (based on REL)

Figure E1-16. NEI53702 Benzene Acute HQ values (based on REL)

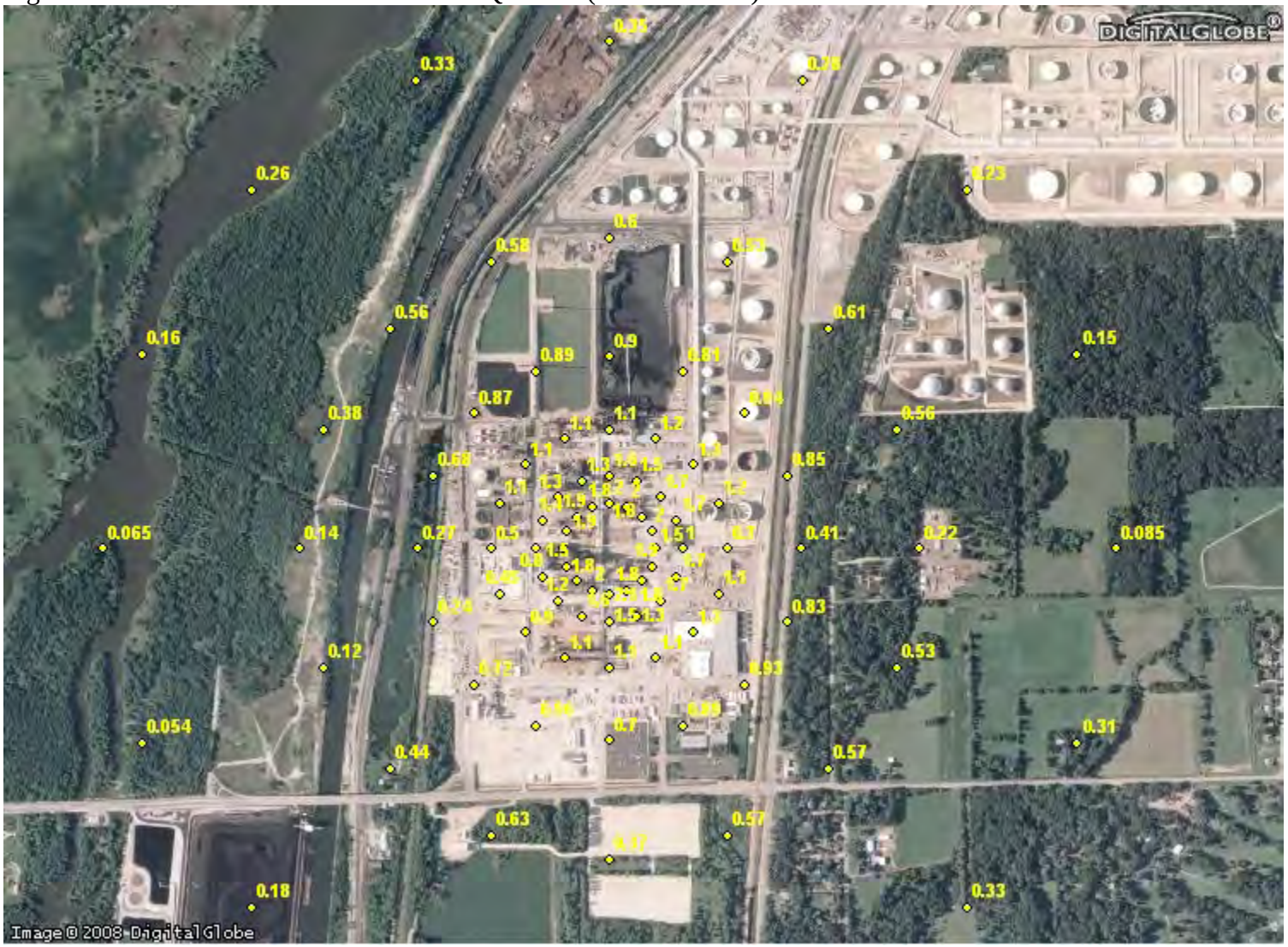

Figure E1-17. NEI6087 Hydrofluoric Acid Acute HQ values (based on REL)

Figure E1-18. NEI6136 Formaldehyde Acute HQ values (based on REL)

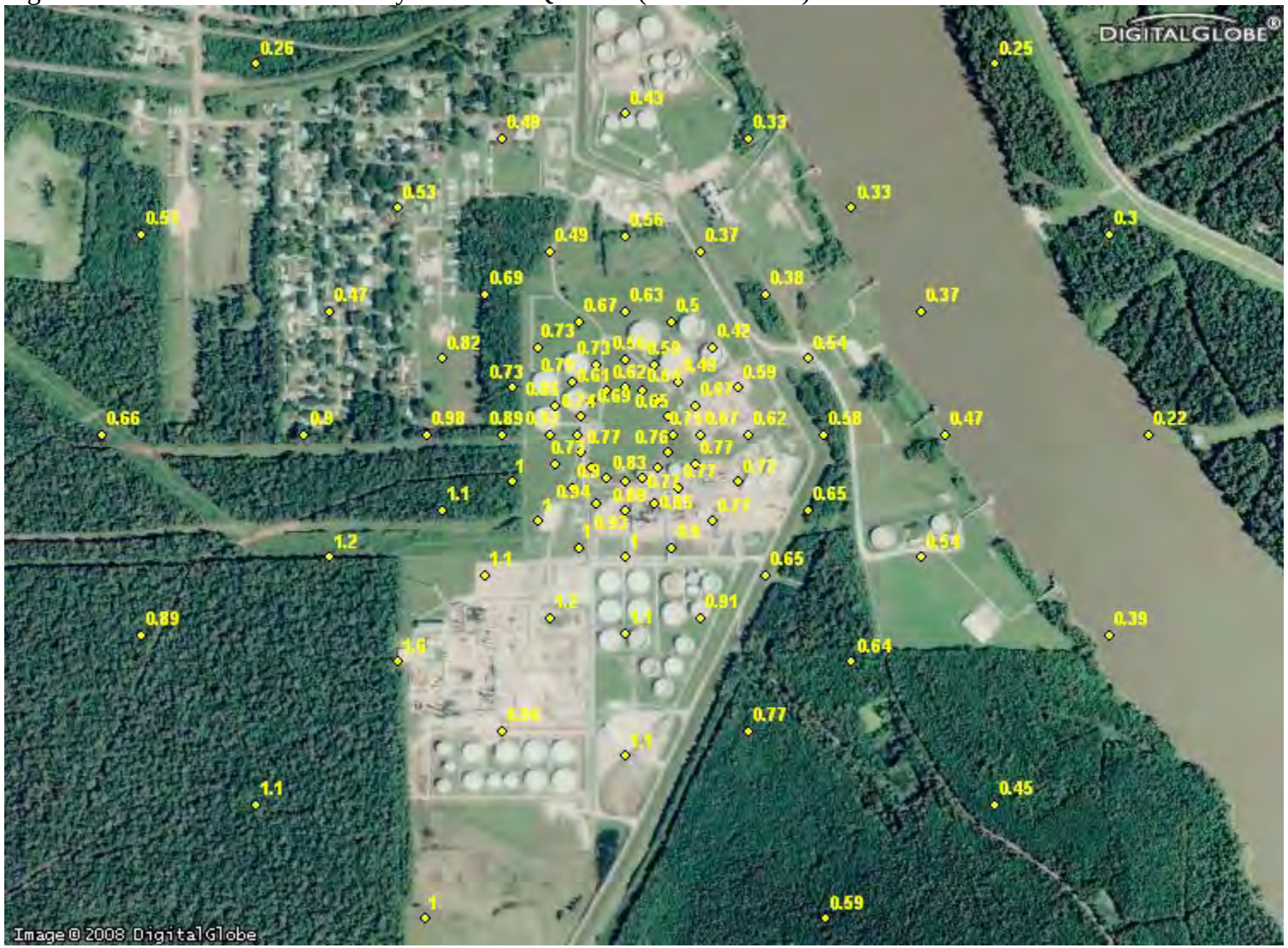

Figure E1-19. NEI6166 Benzene Acute HQ values (based on REL)

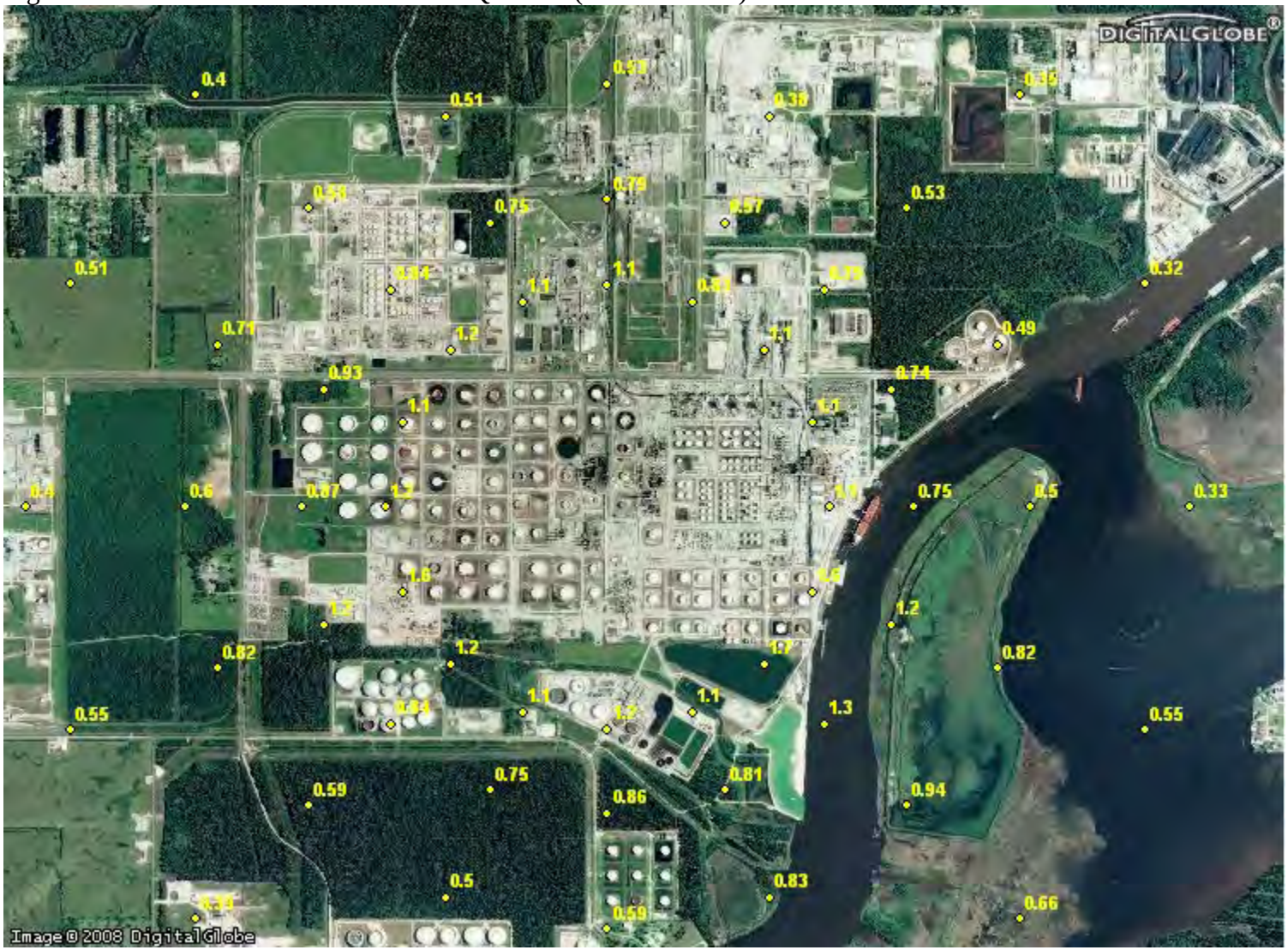

Figure E1-20. NEI6446 Benzene Acute HQ values (based on REL)

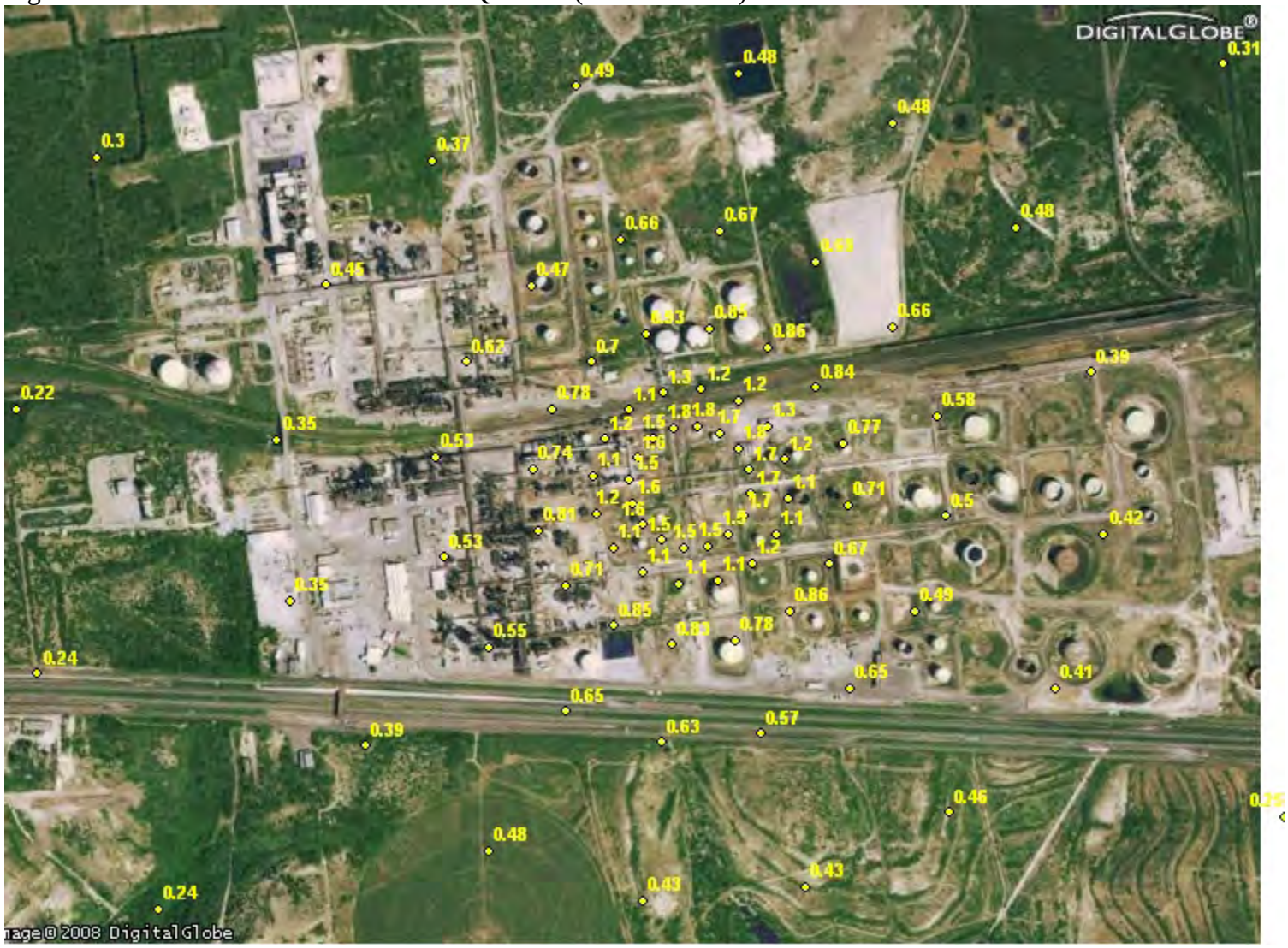

Figure E1-21. NEI6519 Benzene Acute HQ values (based on REL)

Figure E1-22. NEI7441 Glycol Ethers Acute HQ values (based on REL)

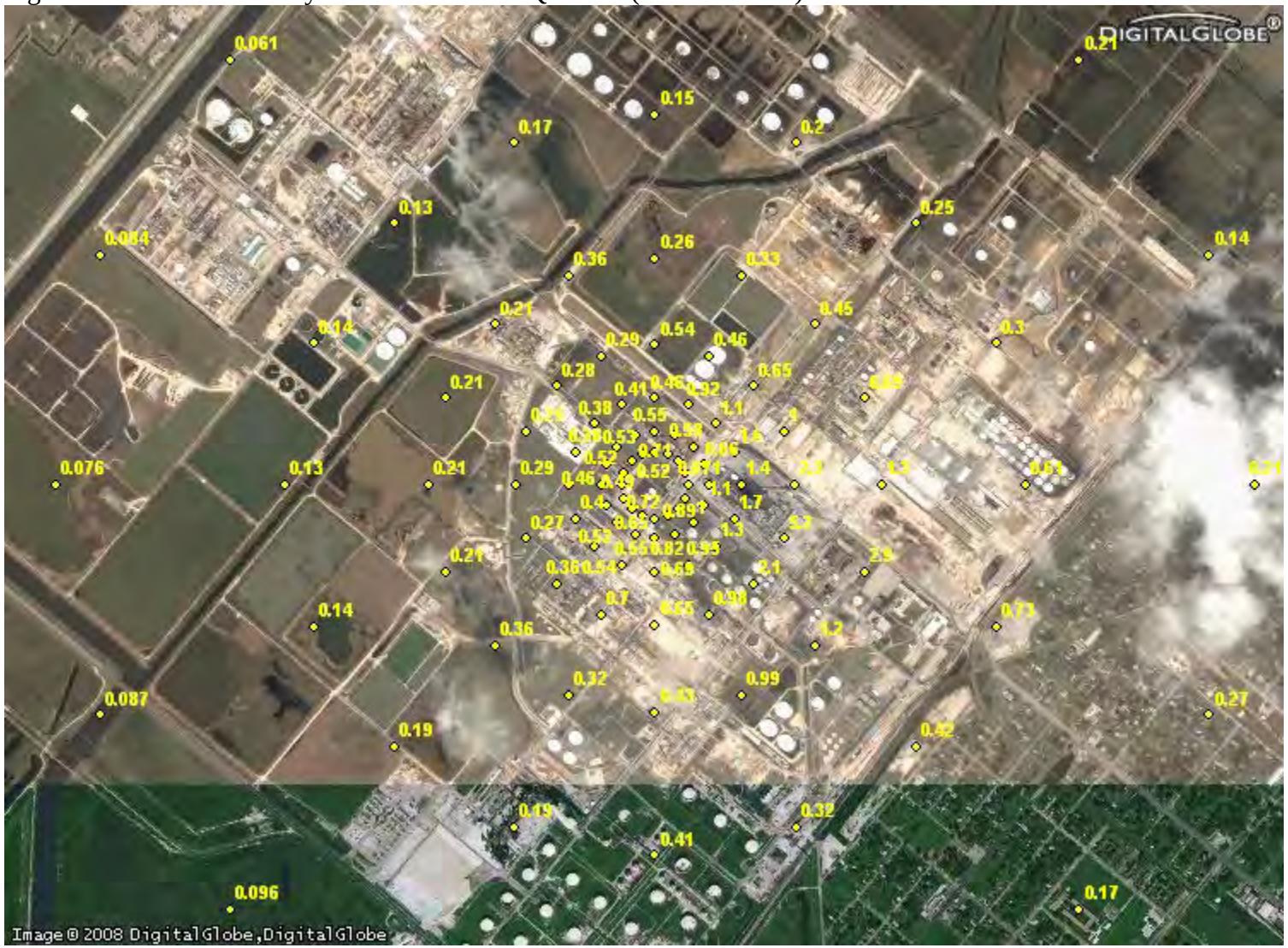

Figure E1-23. NEI7988 Benzene Acute HQ values (based on REL)

Figure E1-24. NEIOKT$11009 Benzene Acute HQ values (based on REL)

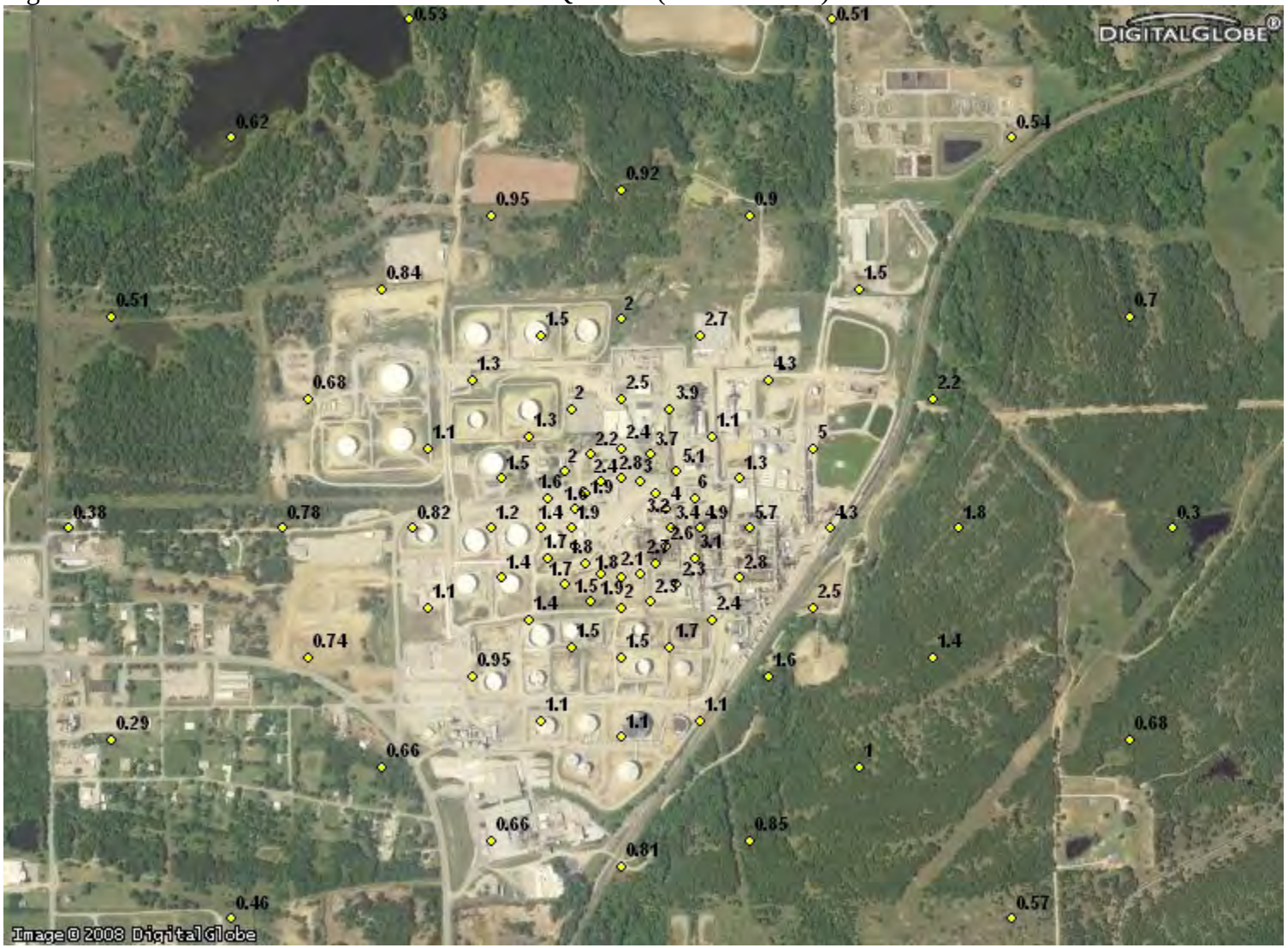

Figure E1-25. NEIPRT$64 Benzene Acute HQ values (based on REL)

Appendix E2
Refined Acute Assessment
For Portland Cement Facilities

Initial acute screening risk calculations were performed with the HEM-3 model. HEM-3 estimates acute (1-hour) impacts at both polar and census block receptors. It is assumed for this short period of time that an exposed individual could be located at any offsite location. The lack of readily available detailed property boundary information for many of the facilities evaluated made it difficult to determine whether receptors were on- or offsite. In the absence of such information, the first ring of polar receptors was placed 100 meters from the plant center for many facilities. However, these polar rings often transected onsite locations, preventing public access to exposures at these levels and thereby overestimating exposures. The screening analysis indicated that 8 facilities had the potential to exceed a 1-hour AEGL-1 reference value, and 4 facilities had the potential to exceed a 1-hour AEGL-2 reference value for one or more pollutants. To refine the analysis for these facilities, the polar receptors for each facility were overlaid on an aerial photograph of the facility to determine the offsite receptor with the highest 1-hour exposure. Figures E2-1 through E2-9 depict the modeled acute hazard quotients for these facilities. Table E2-1 summarizes the results of this refinement by listing the modeled maximum screening and refined (offsite) hazard quotient values.

Table E2-1 – Maximum Modeled Acute Hazard Quotients (Refined Approach)

Figure No.	Facility NEI ID	Pollutant	Criteria	Screening HQ	Refined HQ [1]	Refined Modeling Notes
E2-1	NEI11181	Hydrochloric acid	AEGL-1	40	6	SW of facility
	NEI11181	Hydrochloric acid	AEGL-2	3	<1	HQ reduced by same factor as AEGL-1 HQ
E2-2	NEI16783	Chlorine	AEGL-1	7	2	W of facility
	NEI16783	Chlorine	AEGL-2	2	<1	HQ reduced by same factor as AEGL-1 HQ
E2-3	NEI16783	Hydrochloric acid	AEGL-1	50	10	W of facility
	NEI16783	Hydrochloric acid	AEGL-2	4	<1	HQ reduced by same factor as AEGL-1 HQ
E2-4	NEI22743	Formaldehyde	AEGL-1	3	<1	HQ reduced by same factor as AEGL-1 HQ
E2-5	NEI22838	Hydrochloric acid	AEGL-1	20	3	N of facility
	NEI22838	Hydrochloric acid	AEGL-2	2	<1	HQ reduced by same factor as AEGL-1 HQ
E2-6	NEI25375	Hydrochloric acid	AEGL-1	20	3	Multiple Locations
	NEI25375	Hydrochloric acid	AEGL-2	2	<1	HQ reduced by same factor as AEGL-1 HQ
E2-7	NEI338	Formaldehyde	AEGL-1	3	2	E of facility
E2-8	NEI40539	Hydrochloric acid	AEGL-1	9	3	E of facility
E2-9	NEI51527	Hydrochloric acid	AEGL-1	10	2	Multiple Locations

[1] Where facilities had a HEM-3 screening acute HQ greater than 1, HQ values at polar receptors were overlaid on aerial photographs to determine the maximum offsite value.

Figure E2-1. NEI11181 Hydrochloric Acid Acute HQ values (based on AEGL-1)

Figure E2-2. NEI16783 Chlorine Acute HQ values (based on AEGL-1)

Figure E2-3. NEI16783 Hydrochloric Acid Acute HQ values (based on AEGL-1)

Figure E2-4. NEI22743 Formaldehyde Acute HQ values (based on AEGL-1)

Figure E2-5. NEI22838 Hydrochloric Acid Acute HQ values (based on AEGL-1)

Figure E2-6. NEI25375 Hydrochloric Acid Acute HQ values (based on AEGL-1)

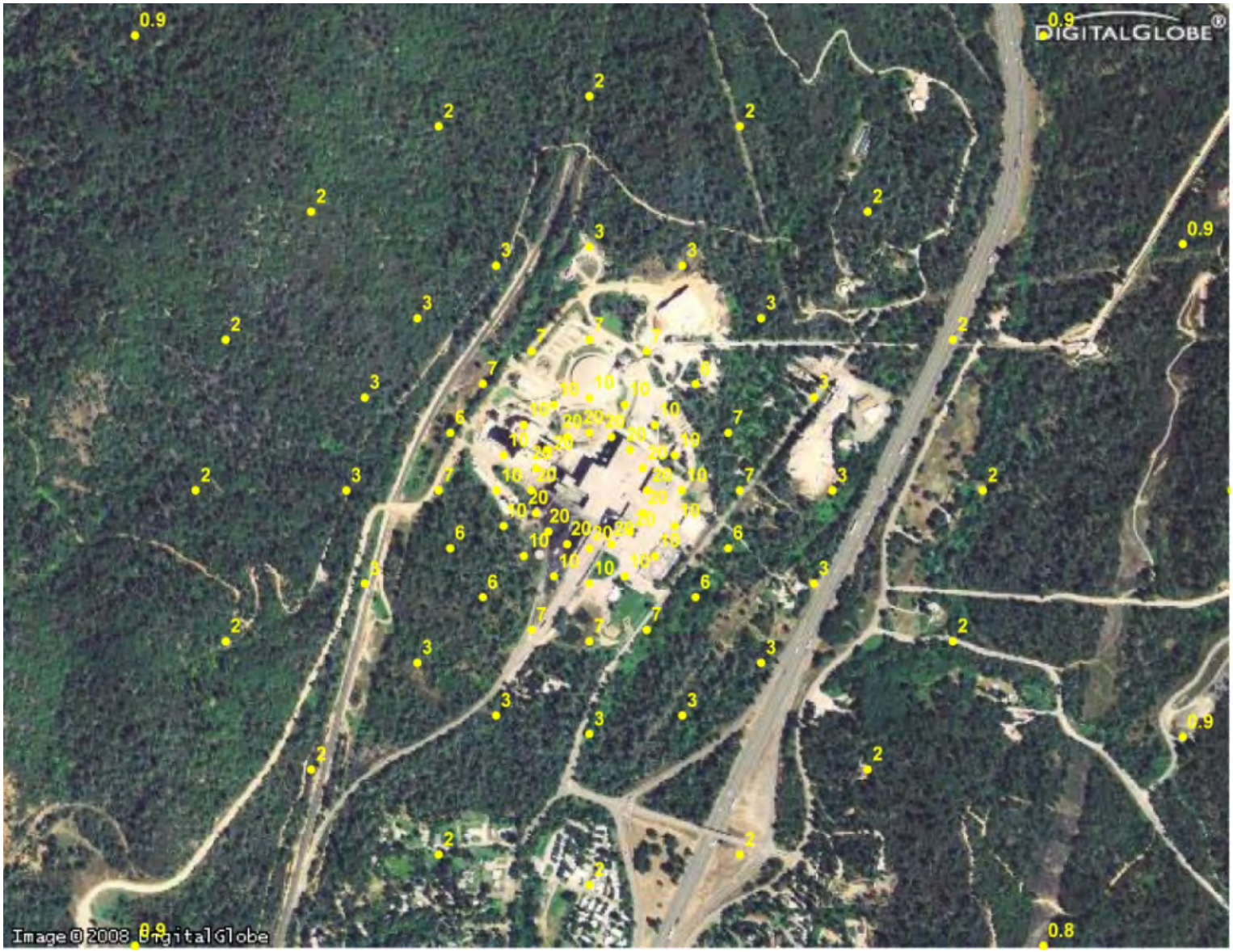

Figure E2-7. NEI338 Formaldehyde Acute HQ values (based on AEGL-1)

Figure E2-8. NEI40539 Hydrochloric Acid Acute HQ values (based on AEGL-1)

Figure E2-9. NEI51527 Hydrochloric Acid Acute HQ values (based on AEGL-1)

Appendix F
Statistical analysis of operational parameters to assess air emissions of chlorinated dibenzodioxins and furans (CDD/F) from portland cement facilities

Appendix F: Statistical analysis of operational parameters to assess air emissions of chlorinated dibenzodioxins and furans (CDD/F) from portland cement facilities

F.1 Introduction

Because very small amounts of CDD/F emitted from any source may become an important environmental and health stressor, data collection for these substances tends to be expensive and data scarce. Thus, estimates of risks associated with CDD/F exposure are often both important and uncertain.

In its 2002 dioxin report, EPA derived a single emission factor of 0.27 ng 2378-$TCDD_{(TEQ)}$ / kg clinker for all non-hazardous waste combustion units for this source category, based upon stack tests from 13 sites. This factor was developed for all kilns regardless of type or operational parameters.

This analysis attempts to refine EPA's 2002 effort by searching for statistical correlations between CDD/F emissions from non-hazardous waste combustors operated by the Portland cement industry and site-specific operational parameters.

F.2 Methods

We obtained the following operational parameters to use as independent variables:

1. Process parameters[1]: Temperature at the inlet to the air pollution control device, flow rate at the stack, kiln type, and estimated kiln clinker capacity.
2. Manufacturing process (type of kiln)[2] – wet, dry, dry with preheater, or dry with both preheater with precalciner.

The hierarchy of data resources for operational data was as follows: 1) stack compliance tests; 2) PCA survey of HCl stack emissions; and 3) 2002 NEI data for HCl. Process data for HCl were considered a plausible surrogate for dioxin because both are emitted from the same combustion stacks.

We obtained CDD/F emission estimates and emission factors reported to the Toxic Release Inventory (TRI)[3] by 60 non-hazardous waste combustion cement plants from 2002-2006. We then developed site-specific 2378-$TCDD_{(TEQ)}$ emission factors by dividing the 2378-$TCDD_{(TEQ)}$ emissions by the estimated clinker capacity data[4] for each facility. For emission data reported as CDD/F congeners, TEQ calculations were based on the 1998 World Health Organization (WHO) recommendations. The use of separate

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1) RTI Memo to EPA (August 31, 2007): Draft: Design Options and Data for Cement Emissions Trading Market Model: PCA kiln member companies compiled by RTI: Kiln_Data_11_05_07.xls
2) ENVIRON International Corporations (PCA kiln data from HCl assessment) and 2002 NEI data
3) Toxic Release Inventory (TRI): http://www.epa.gov/tri
4) RTI Memo to EPA (August 31, 2007): Draft: Design Options and Data for Cement Emissions Trading Market Model: PCA kiln member companies compiled by RTI: Kiln_Data_11_05_07.xls

emission factors for each kiln type helps to account for the variability in production for each year as well as the variability in the amount of dioxin that is generated by the combustion process from each type of kiln. This variability is evident when comparing emission factors between the wet and dry type kilns.

Based upon historic production rates for this source category, EPA assumed that the maximum allowable production rates were approximate to actual rates. Table F-1 contains a summary of TRI dioxin $_{(TEQ)}$ air emissions for this source category.

Table F-1: Dioxin$_{(TEQ)}$ emission inventory estimates for portland cement for non-hazardous waste combustion kilns

Source of Data	Air Emissions (grams/year)	Reporting Universe
2002-2006 TRI Data (Avg)	13.8	60
EPA's Dioxin Report (Calendar Year - 2000)	17.2	102*
EPA estimate for RTR with mean emission factor (Avg from 2002-06 TRI Data)	18	97

* - April 1999; Portland Cement NESHAP Final Rule Economic Analysis; http://epa.gov/ttn/atw/pcem/iv-a-004.pdf

Once these data were assembled we used SAS^{tm} software to calculate correlation coefficients for emissions vs. the two continuous variables (flow and inlet temperature), and an analysis of variance to test for differences among emission factors from different kiln types. Correlation analyses were run using a subset of the database that contained a complete set of temperature and flow measurements. The analysis of variance, for which only emission factor and kiln type were needed, used the full dataset.

F.3 Results and discussion

F.3.1 Correlation analysis

Results of the correlation analysis using the more limited complete data set are shown in Table F-2. Reported TCDD/F$_{(TEQ)}$ emissions did not correlate significantly with inlet temperature, stack flow rate, or an interaction between the two.

Table F-2: Results of SAS correlation analysis between TCDD/F$_{(TEQ)}$ emissions and continuous process variables for portland cement facilities.

Variable	DF	Type I SS	Mean Square	F Value	Pr>F
Temperature	1	0.00018237	0.00018237	1.82	0.1884
Flow	1	0.00010613	0.00010613	1.06	0.3123
Temp*Flow	1	0.00013916	0.00013916	1.39	0.2487

F.3.2 Analysis of variance

Based on the full TRI-derived database (212 observations spanning 5 years), the ANOVA found statistically significant differences in TCDD/F$_{(TEQ)}$ emission factors among the four

process types (F=17.7, P<0.001). Table F-3 shows the mean emission factors for each type, and the 95% upper confidence level for each.

Table F-3: Mean and 95% UCL TCDD/$F_{(TEQ)}$ emission factors for Portland cement facilities, by kiln type

Kiln type	Mean emission factor (ng/kg clinker capacity)	95% UCL emission factor (ng/kg clinker capacity)
Dry	0.110	0.229
Dry with preheater and precalciner	0.170	0.614
Dry with preheater	0.168	0.377
Wet	0.768	1.877

These emission factor estimates plausibly bracket the single estimate (0.27 ng/kg) from EPA's 2002 Dioxin Report. Given this plausibility, we characterized CDD/F emissions by kiln type for the Portland cement risk assessment, and calculated plant-specific risks separately using the mean and UCL emissions factors.

APPENDIX G:
Evaluation of Radionuclide Emissions Reported for Portland Cement Sources

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LIST OF EXHIBITS

G.1 Issue

Radionuclides, a class of atoms that spontaneously undergo radioactive decay, are regulated as hazardous air pollutants (HAPs) when they are emitted to the air. Emissions of radionuclides from industrial facilities are reported in the 2002 National Emissions Inventory (NEI) in mass-based units of tons (U.S., short) per year. However, the known hazards from radionuclides are most closely associated with the radioactivity and cancer potency of the material released, not with the mass of the material released. Radioactive isotopes vary by level of radioactivity (which can be expressed as picocuries [pCi] per metric ton of the material), cancer potency, and persistence in the environment. Furthermore, the products of radioactive decay are different depending on the isotope, and so different isotopes may produce different daughter products and subsequent radioactive decompositions. Therefore, reporting unspeciated emissions of radioactive substances from a single facility collectively by mass, rather than individually by radioactive isotope in units of radioactivity, prevents the accurate estimation of risks posed by radionuclides emitted from industrial facilities.

The Portland cement manufacturing sector is one source of radionuclide emissions. To evaluate the potential radionuclide hazards associated with Portland cement manufacturing facilities, we identified two facilities in California that reported emissions in the 2002 NEI (EPA 2008a).[1] Identifying details of these facilities, including NEI-reported radionuclide emissions, are presented in Exhibit G-1.[2] This exhibit also includes actual clinker production and particulate matter (PM) emissions because these values are used later in this appendix as an alternate means of estimating radionuclide emissions.

Exhibit G-1. Portland Cement Manufacturing Facilities in California Reporting Radionuclide Emissions in the 2007 NEI Database

Facility Name	NTI Site ID	NEI Reported Radionuclide Emissions (ton/yr)	Actual Clinker Production [a] (short ton/yr)	PM Emissions [b] (ton/yr)
Lehigh Southwest Cement Co.	NEICA1505122	8.21E-03	1.00E+06	2.73E+02
RMC Pacific Materials (CEMEX)	NEI2CA151186	1.42E-08	8.50E+05	3.90E+02

[a] EPA Dioxin Emission Inventory (2007a).
[b] CARB (2007).

On a mass basis, emissions reported for these facilities are very low (e.g., the highest emitter, Lehigh Southwest Cement, reports only 0.00821 ton of radionuclides). Nevertheless, significant risks may be associated with emissions of this magnitude because, at even minute concentrations, some radionuclides are associated with large risks. However, which radionuclides were emitted and how much of each was emitted is uncertain. Also, whether the facilities are reporting radionuclide emissions in a uniform manner is not certain. Some facilities may be reporting the total mass of materials that contain radionuclides, even in trace quantities, and some may be reporting what they believe to be the actual mass of radionuclides emitted.

[1] As part of the RTR data review process, EPA reviewed and updated the inventory, so the data analyzed in this appendix are the most recent data available. Although the most recent version does not include any radionuclide emissions for the Lehigh Southwest Cement Co. facility, radionuclide emission data from a previous version were included in this analysis to provide an additional point for analysis.

[2] Unless otherwise specified, tons are metric units throughout this appendix.

This appendix describes an analysis performed on the two facilities presented in Exhibit G-1 and on the Portland cement manufacturing sector. We performed this analysis to:

- Estimate and characterize the actual emissions of radionuclides from Portland cement manufacturing facilities that report radionuclide emissions and from all U.S. Portland cement manufacturing facility sources;
- Evaluate the utility of the values reported in NEI for estimating risks from radionuclides;
- Quantitatively estimate potential incremental inhalation cancer risks posed by radionuclides emitted by Portland cement manufacturing facilities; and
- Qualitatively evaluate background exposures and risks from inhalation and non-inhalation exposures to radionuclides.

The analysis has two parts: (1) estimating radionuclide emissions and (2) evaluating resultant potential incremental cancer risks associated with inhalation exposures to these emissions. We completed the first part by comparing NEI-reported emissions with modeled emissions, and we performed the second through a series of modeling exercises. This appendix chronicles the methods, results, and conclusions of the analysis, and also discusses the assumptions, uncertainties, and additional data needs.

G.2 Approach

This section presents the approaches used to model radionuclide emissions and to estimate potential incremental cancer risks from Portland cement manufacturing facilities. We estimated radionuclide emissions for the two Portland cement sources identified in Exhibit G-1 using the NEI-reported emissions and scaling factors developed from a "typical" Portland cement facility. We derived emission factors for a typical facility from the European Commission Radiation Protection 135 report (Chen et al. 2003), hereafter referred to as the "EU naturally occurring radioactive material (NORM) report."

The EU NORM report relied upon an analysis by Leenhouts et al. (1996) that examined one large Portland cement facility in the Netherlands using data from 1990. The heating of clinker in a cement kiln results in the volatilization of radioactive material, including polonium-210 (^{210}Po) and radon-222 (^{222}Rn), of which approximately 50 percent and 100 percent, respectively, of the radionuclide content in the material is assumed to escape (EU NORM report 2003 and Leenhouts et al. 1996). Uncertainties stemming from the assumption that the facility described by Leenhouts et al. (hereafter, "the Maastricht facility") is representative of U.S. Portland cement facilities are discussed in Sections G.2.1 and G.4.

Resultant ground-level radionuclide activity (in pCi/m^3) was modeled with HEM3 (Human Exposure Model-3, EPA 2007b) using the emissions described in Section G.2.1 below separately for (1) the two California facilities using linearly scalable mass-based emissions of other pollutants, and (2) all U.S. facilities, using emissions developed with the clinker production factors.

G.2.1 Estimating Radionuclide Emissions from Portland Cement Sources

To determine whether the emissions reported in NEI are plausible and useful for our purpose here, we compared NEI-reported emissions to our modeled emissions. The Maastricht facility was used to develop emission factors that were applied to U.S. Portland cement producers. Emission factors were developed by assuming that radionuclide emissions from Portland cement manufacturing facilities were proportional to (1) actual clinker production or (2) PM

emissions. The parameters of the Maastricht facility used to model U.S. Portland cement producers are presented Exhibit G-2. Factors derived from the data in Exhibit G-2 for emission rates of ^{210}Po and ^{222}Rn are presented in Exhibit G-3.

Exhibit G-2. Details of the Maastricht Facility for Modeling Portland Cement Facilities [a]

Cement production (kton) [b]	PM Emissions (kton)	^{210}Po (Ci/yr) [c]	^{222}Rn (Ci/yr)
2,000	8.0	2.108	4.243

[a] Leenhouts et al. (1996), EU NORM (2003).
[b] kton = kiloton; Leenhouts et al. report 2,107 kton total cement production, with 980 kton Portland cement, and the remaining amount as other types of output. They report only 365 kton Portland clinker production. The EU NORM document rounds to 2,000 kton cement production. Uncertainties related to these data are discussed below.
[c] Ci/yr = curies per year.

Exhibit G-3. Emission Scaling Factors Derived from Data from the Maastricht Facility

Method for Estimating Emissions of Radioactivity	Emission Scaling Factor	
	^{210}Po	^{222}Rn
Based on clinker production (Ci[emitted]/ton[clinker produced])	1.05E-06	2.12E-06
Based on PM emission rate (Ci[emitted]/ton[PM emitted])	2.64E-04	5.30E-04

Other than heating in the kiln, which was identified as the main emission source at the Maastricht facility, emissions were considered to result from three processes: extraction, transport, and mixing (Leenhouts et al. 1996). The facility is estimated to release 0.5 to 1 percent by mass of the total input material as dust (Leenhouts et al. 1996). Although the Maastricht facility provides a well-documented data set and serves as a good case study, the comparisons between it and U.S. facilities have limitations. First, the data from Maastricht (from 1990) are relatively outdated and may not reflect advances in pollution control technologies, especially for PM. The emission scaling factor approach assumes that the facilities in California in 2005 had industrial processes and PM emission control equipment that are identical to those at the Maastricht facility in 1990 and that radionuclide emission rates are directly proportional to clinker production volume or PM emissions. Similarly, the scaling factor approach assumes that radionuclide content in input materials is identical; this may not be true because raw materials are obtained from different regions of the globe and may contain different quantities of radiological materials.

The Maastricht facility data were used to estimate the relative contribution of ^{210}Po and ^{222}Rn (94 percent and 6 percent by mass, respectively) to the total radionuclide emissions reported in NEI. The emissions were then converted to radioactivity units by multiplying by the specific activity for each radionuclide: 4,493 curies per gram (Ci/g) for ^{210}Po and 153,800 Ci/g for ^{222}Rn (EPA 2001). We assumed that 100 percent of the radionuclide emissions reported in NEI were ^{210}Po or ^{222}Rn, rather than other radioactive material or non-radioactive material. If the materials reported as radionuclide emissions in NEI contain a large portion of non-radioactive material, this assumption would be highly conservative. For the current analysis, this conservative assumption was applied because the emissions are not speciated, and the proportion of the NEI-reported emissions that is actually radioactive material is unknown.

Based on knowledge of the source category, some of the ^{210}Po is likely to be physically bound to solid matter and, therefore, increased PM controls probably result in lower emissions of this

isotope. Because ^{222}Rn is released in gas phase, however, emissions of this isotope are less likely to be controlled. Therefore, the Maastricht facility model may overestimate the relative contribution to overall radionuclide risk from ^{210}Po and underestimate the relative contribution from ^{222}Rn. In both of these respects, the Maastricht model leads to conservative estimates of emissions and risk.

G.2.2 AERMOD Modeling Approach for Estimating Inhalation Risk at Two California Facilities

We applied a linear scaling approach for the California facilities to each emission value generated by the three approaches detailed in Section G.2.1 above. The linear scaling approach was developed for the two facilities by modeling emissions of 1.0 ton/yr of a pollutant with a unit risk estimate (URE) of 1.0 cubic meter per microgram ($m^3/\mu g$). (The model results were then converted into metric units for consistency.) AERMOD[3] (EPA 2008b) estimates ambient air concentrations using Gaussian dispersion equations, so the resulting model output (in concentration units of $\mu g/m^3$) can be multiplied by the actual emissions in ton/yr and the chemical-specific URE to derive the facility-specific ambient concentrations and risk estimates. Because AERMOD estimates ambient concentrations resulting from the modeled source(s) only, background exposures must be calculated independently. Therefore, the highest estimated concentration at a location where people reside (assumed to be represented by census block locations in this analysis) is the maximum incremental risk (MIR) estimate.

The MIR estimates were calculated by multiplying the AERMOD-scalable concentration result by the emission rate (in Ci/yr) and the inhalation URE (in pCi/m^3) for each radionuclide. The inhalation URE was calculated by multiplying the inhalation slope factor (EPA 2001), reported in risk/pCi, by an estimate of lifetime respiration by an exposed individual of 20 m^3/day for 70 years, consistent with the approach EPA used to calculate inhalation UREs for non-radioactive HAPs (EPA 2002). Therefore, the UREs presented here will lead to more conservative estimates of incremental risk. The inhalation UREs are presented in Exhibit G-4.

Exhibit G-4. Inhalation Unit Risk Estimation for ^{210}Po and ^{222}Rn

Unit	^{210}Po	^{222}Rn
Inhalation slope factor (risk/pCi) [a]	1.08E-08	1.8E-11
Inhalation URE $(pCi/m^3)^{-1}$ [b]	5.52E-03	9.20E-06

[a] EPA 2001.
[b] Assuming lifetime (70 years) respiration of 20 m^3/day.

G.2.3 CAP88 Approach for Estimating Inhalation Risk at Two California Facilities

We used the radioactivity model, Clean Air Assessment Package – 1988 or CAP88 (EPA 2007c), as an alternative means of estimating risk from the two California facilities. The CAP88 model is designed to estimate doses and risks from radionuclide emissions to the air, is comprised of databases and associated utility programs, and is EPA's preferred model for assessing radioactivity risk (EPA 2007b).

[3] AERMOD = AERMIC Model; AERMIC = AMS/EPA Regulatory Model Improvement Committee; AMS = American Meteorological Society

Populations exposed to emissions from each facility were determined by using modified versions of the default CAP88 population files for cities close to each location, rather than developing site-specific population files for each site.[4] Linear population-scaling factors were applied to each population to develop modified CAP88 population files. These population files are used by CAP88 to determine which individuals are exposed to radionuclide emissions. We developed the scaling factors applied to each location, shown in Exhibit G-5, by comparing the total populations within 70 kilometers (km) of each facility to the default population file in CAP88. We then applied the scaling factors uniformly to the CAP88 default population files. Therefore, the results of the modeling may be directionally inaccurate or may not reflect the risks as well as they might if accurate population files had been developed. The wind direction at the sites is predominantly west to east, and thus using the Los Angeles population file may lead to a more conservative measure of risk because the bulk of the population density in Los Angeles is to the east. The Berkeley population file has the bulk of the population to the south; therefore, using the Berkeley file may not lead to similarly conservative assumptions. However, the most important characteristic of the population file when estimating maximum individual risk is the assumed location of the most exposed individual (i.e., for this analysis, the census block centroid with the highest modeled concentration). Because the census block locations included in the population files were not modified, the use of the modified population files based on urban locations (e.g., Los Angeles) is likely to provide an overestimate of maximum individual risk when used in sparsely-populated locations.

Exhibit G-5. Population Scaling Factors Applied to the CAP88 Default Population Files Around Two California Portland Cement Facilities

NTI Site ID	City	CAP88 Default Population File	Scaling Factor
NEICA1505122	Monolith	Los Angeles	0.047
NEI2CA151186	Davenport	Berkeley	0.58

We assumed a mixing height of 643 m, based on the year-long average value of full-day mixing heights for the Oakland, California meteorological station (EPA 2008c).

CAP88 estimates incremental cancer risks from modeled radionuclide emissions and does not account for background radioactivity.

G.2.4 AERMOD Modeling Approach for Estimating Inhalation Risk at All U.S. Facilities

To obtain an approximation of total incremental cancer effects associated with radionuclide emissions from the entire source category, radionuclide emissions were modeled using AERMOD for all U.S. Portland cement facilities and the results were used to estimate MIR inhalation cancer risk. Radionuclide emissions for each facility were estimated using the clinker production scaling factor, detailed in Section G.2.1 above and presented in Exhibit G-3. Where actual clinker production data were not available for a facility, clinker production was assumed to equal 95 percent of clinker production capacity, based on the median actual production relative to production capacity from all facilities having data.

[4] CAP88 requires the use of a population file to estimate risk at the MIR location; however, the population data included with the model cover selected locations only (and none of the locations of the two facilities modeled in this analysis). Therefore, the existing CAP88 population files were modified to allow us to obtain an estimate of maximum individual risk for the facilities included in this analysis.

To facilitate AERMOD modeling of these facilities, stack parameters were compiled that were assumed to be representative of radionuclide emission release points. We assumed that lead is released along with radionuclides during the clinker heating, based on knowledge of the production process. An analysis by ICF of all these facilities revealed that, at 91 percent of the facilities, the largest release of lead compounds came from the tallest stack. Therefore, the radionuclide emission source at each of the 91 Portland cement manufacturing facilities in the United States was assumed to be the tallest stack at each site, usually the cement kiln.

Maximum incremental cancer risks from each facility were estimated by HEM3-AERMOD (EPA 2007a).

G.3 Results

G.3.1 Emissions

Exhibit G-6 presents the results of the two emission factor-based approaches and the NEI-reported emissions of radionuclides. Using the assumptions described above, the emissions reported in NEI are several orders of magnitude above those derived using data from the Maastricht facility, particularly for the Lehigh facility (NEICA1505122). At the Lehigh facility, NEI emissions are more than eight orders of magnitude higher than those predicted with the PM emission-scaling factor approach. The emissions calculated with the PM emission scaling factors are generally an order of magnitude lower than those produced using the clinker production factors; however, these sets of emission values are much more comparable to one another than either is to the NEI emissions. Although this difference does not necessarily mean that the NEI emissions are incorrect, it does compel caution in their use and suggests that they may not be correlated to actual emissions of radioactive isotopes in a useful way.

Exhibit G-6. Estimation of Radionuclide Emissions for the Two California Facilities Using Three Approaches

NTI Site ID	Emissions, Based on NEI Emissions and Speciation Assumptions		Emissions, Based on Clinker Production Scaling Factors		Emissions, Based on PM Emission Scaling Factors	
	^{210}Po (Ci/yr)	^{222}Rn (Ci/yr)	^{210}Po (Ci/yr)	^{222}Rn (Ci/yr)	^{210}Po (Ci/yr)	^{222}Rn (Ci/yr)
NEICA1505122	3.48E+07	7.01E+07	9.59E-01	1.93E+00	7.20E-02	1.45E-01
NEI2CA151186	6.02E+01	1.21E+02	8.13E-01	1.64E+00	1.03E-01	2.07E-01

The emission values derived for emissions from all 91 U.S. Portland cement manufacturing facilities using the clinker production-based emission factors are presented in Exhibit G-7, sorted by ^{210}Po emissions from highest to lowest. The two case study facilities are highlighted yellow.

Exhibit G-7. Estimation of Radionuclide Emissions for All U.S. Portland Cement Facilities, Based on the Clinker Production Scaling Factor

NTI Site ID	Emissions (short ton/yr)		Emissions (Ci/yr)	
	^{210}Po	^{222}Rn	^{210}Po	^{222}Rn
NEI22900	6.69945E-10	3.93935E-11	2.73068E+00	5.49638E+00
NEI12018	5.27644E-10	3.10260E-11	2.15066E+00	4.32890E+00

Exhibit G-7. Estimation of Radionuclide Emissions for All U.S. Portland Cement Facilities, Based on the Clinker Production Scaling Factor

NTI Site ID	Emissions (short ton/yr)		Emissions (Ci/yr)	
	^{210}Po	^{222}Rn	^{210}Po	^{222}Rn
NEITX139099J	4.97730E-10	2.92670E-11	2.02874E+00	4.08348E+00
NEI33394	4.21326E-10	2.47744E-11	1.71732E+00	3.45665E+00
NEI34931	4.10064E-10	2.41122E-11	1.67141E+00	3.36426E+00
NEI22838	3.79071E-10	2.22898E-11	1.54508E+00	3.10998E+00
NEI886	3.69776E-10	2.17432E-11	1.50720E+00	3.03372E+00
NEIAL1170004	3.68015E-10	2.16397E-11	1.50002E+00	3.01928E+00
NEI24859	3.67770E-10	2.16253E-11	1.49902E+00	3.01726E+00
NEIFL0860020	3.65493E-10	2.14914E-11	1.48974E+00	2.99859E+00
NEIAZ0250421	3.62856E-10	2.13363E-11	1.47900E+00	2.97695E+00
NEIAL8026	3.53766E-10	2.08018E-11	1.44195E+00	2.90238E+00
NEIKYR0060	3.35341E-10	1.97184E-11	1.36684E+00	2.75121E+00
NEI20046	3.34850E-10	1.96895E-11	1.36484E+00	2.74718E+00
NEI18621	3.27234E-10	1.92417E-11	1.33380E+00	2.68470E+00
NEI7255	3.14459E-10	1.84905E-11	1.28173E+00	2.57989E+00
NEIT$FNP1408	3.09791E-10	1.82161E-11	1.26270E+00	2.54160E+00
NEIMIB1559	2.95276E-10	1.73625E-11	1.20354E+00	2.42251E+00
NEIMO0990002	2.90875E-10	1.71037E-11	1.18560E+00	2.38640E+00
NEITXT$11924	2.76380E-10	1.62514E-11	1.12652E+00	2.26748E+00
NEI2PRT14359	2.75934E-10	1.62252E-11	1.12470E+00	2.26383E+00
NEI22877	2.66553E-10	1.56736E-11	1.08647E+00	2.18686E+00
NEI52351	2.59183E-10	1.52402E-11	1.05643E+00	2.12639E+00
NEI20130	2.53778E-10	1.49224E-11	1.03440E+00	2.08205E+00
NEIVA2553	2.50410E-10	1.47244E-11	1.02067E+00	2.05442E+00
NEIIA0330060	2.36757E-10	1.39216E-11	9.65016E-01	1.94240E+00
NEICA1505122	2.35353E-10	1.38390E-11	9.59294E-01	1.93089E+00
NEI12238	2.30661E-10	1.35631E-11	9.40171E-01	1.89239E+00
NEI34326	2.28474E-10	1.34345E-11	9.31256E-01	1.87445E+00
NEI26277	2.27331E-10	1.33673E-11	9.26596E-01	1.86507E+00
NEI2PA110039	2.24429E-10	1.31967E-11	9.14768E-01	1.84126E+00
NEIAL1150002	2.21104E-10	1.30012E-11	9.01216E-01	1.81399E+00
NEIPA94-2626	2.16789E-10	1.27474E-11	8.83627E-01	1.77858E+00
NEITXT$11872	2.13243E-10	1.25389E-11	8.69173E-01	1.74949E+00
NEIMIB1743	2.11643E-10	1.24448E-11	8.62651E-01	1.73636E+00
NEI7376	2.08575E-10	1.22644E-11	8.50147E-01	1.71119E+00
NEISDT$8989	2.07498E-10	1.22011E-11	8.45756E-01	1.70235E+00
NEI572	2.06035E-10	1.21150E-11	8.39793E-01	1.69035E+00
NEIAL321	2.01696E-10	1.18599E-11	8.22109E-01	1.65476E+00
NEI31319	2.01696E-10	1.18599E-11	8.22109E-01	1.65476E+00
NEITXRBG0259	2.01696E-10	1.18599E-11	8.22109E-01	1.65476E+00
NEI2CA151186	1.99485E-10	1.17299E-11	8.13097E-01	1.63662E+00
NEIUT10303	1.98994E-10	1.17010E-11	8.11094E-01	1.63259E+00
NEI40539	1.93844E-10	1.13982E-11	7.90105E-01	1.59034E+00

Exhibit G-7. Estimation of Radionuclide Emissions for All U.S. Portland Cement Facilities, Based on the Clinker Production Scaling Factor

NTI Site ID	Emissions (short ton/yr)		Emissions (Ci/yr)	
	^{210}Po	^{222}Rn	^{210}Po	^{222}Rn
NEI12739	1.93343E-10	1.13688E-11	7.88063E-01	1.58623E+00
NEI13290	1.91624E-10	1.12677E-11	7.81054E-01	1.57212E+00
NEI32033	1.90641E-10	1.12099E-11	7.77048E-01	1.56406E+00
NEITN0653070	1.89412E-10	1.11377E-11	7.72042E-01	1.55398E+00
NEIIA0330035	1.84090E-10	1.08247E-11	7.50348E-01	1.51031E+00
NEISC0351244	1.78790E-10	1.05130E-11	7.28745E-01	1.46683E+00
NEIFLR001008	1.77847E-10	1.04576E-11	7.24902E-01	1.45910E+00
NEIALT$4449	1.75901E-10	1.03431E-11	7.16967E-01	1.44313E+00
NEI42038	1.72953E-10	1.01698E-11	7.04951E-01	1.41894E+00
NEIOK4013107	1.69022E-10	9.93866E-12	6.88930E-01	1.38669E+00
NEIPA58-1290	1.67605E-10	9.85538E-12	6.83157E-01	1.37507E+00
NEI22743	1.67056E-10	9.82309E-12	6.80919E-01	1.37057E+00
NEITN0930008	1.63863E-10	9.63530E-12	6.67901E-01	1.34437E+00
NEIWA0331133	1.60349E-10	9.42869E-12	6.53579E-01	1.31554E+00
NEIOHT$6526	1.59662E-10	9.38831E-12	6.50780E-01	1.30990E+00
NEIWV0030006	1.58295E-10	9.30790E-12	6.45206E-01	1.29868E+00
NEIGA1530003	1.57624E-10	9.26846E-12	6.42473E-01	1.29318E+00
NEI46744	1.57100E-10	9.23766E-12	6.40338E-01	1.28888E+00
NEI12976	1.53544E-10	9.02858E-12	6.25844E-01	1.25971E+00
NEI2PRT14367	1.52119E-10	8.94478E-12	6.20036E-01	1.24802E+00
NEI26327	1.48206E-10	8.71467E-12	6.04085E-01	1.21591E+00
NEI51352	1.47894E-10	8.69633E-12	6.02813E-01	1.21336E+00
NEI25375	1.45437E-10	8.55187E-12	5.92800E-01	1.19320E+00
NEINY0394192	1.43963E-10	8.46520E-12	5.86792E-01	1.18111E+00
NEI51435	1.39787E-10	8.21962E-12	5.69769E-01	1.14684E+00
NEINYT$1163	1.39708E-10	8.21498E-12	5.69447E-01	1.14619E+00
NEINY4192600	1.38277E-10	8.13086E-12	5.63616E-01	1.13446E+00
NEIOK1826	1.36593E-10	8.03182E-12	5.56751E-01	1.12064E+00
NEI338	1.35350E-10	7.95873E-12	5.51685E-01	1.11044E+00
NEIPAT$1626	1.34657E-10	7.91800E-12	5.48861E-01	1.10476E+00
NEI51527	1.33645E-10	7.85847E-12	5.44735E-01	1.09645E+00
NEI33699	1.26029E-10	7.41066E-12	5.13693E-01	1.03397E+00
NEITXT$11980	1.14974E-10	6.76060E-12	4.68632E-01	9.43273E-01
NEI446	1.10185E-10	6.47897E-12	4.49110E-01	9.03978E-01
NEINMT$12442	1.06130E-10	6.24055E-12	4.32584E-01	8.70713E-01
NEIWA0331404	1.00960E-10	5.93658E-12	4.11513E-01	8.28301E-01
NEIME0130002	9.34565E-11	5.49534E-12	3.80927E-01	7.66738E-01
NEI16357	8.05801E-11	4.73820E-12	3.28443E-01	6.61097E-01
NEI33444	7.61581E-11	4.47817E-12	3.10419E-01	6.24817E-01
NEIMT0430001	7.19817E-11	4.23260E-12	2.93396E-01	5.90553E-01
NEITXT$12011	7.00163E-11	4.11703E-12	2.85385E-01	5.74429E-01
NEIMT0310005	6.87879E-11	4.04480E-12	2.80378E-01	5.64351E-01

Exhibit G-7. Estimation of Radionuclide Emissions for All U.S. Portland Cement Facilities, Based on the Clinker Production Scaling Factor

NTI Site ID	Emissions (short ton/yr)		Emissions (Ci/yr)	
	^{210}Po	^{222}Rn	^{210}Po	^{222}Rn
NEIPA01993-1	6.82837E-11	4.01515E-12	2.78323E-01	5.60214E-01
NEIID0050004	6.38745E-11	3.75589E-12	2.60351E-01	5.24040E-01
NEIPA23-0797	2.67405E-11	1.57237E-12	1.08994E-01	2.19385E-01
NEI22453	2.50585E-11	1.47346E-12	1.02138E-01	2.05585E-01
NEITX309123F	2.38301E-11	1.40124E-12	9.71311E-02	1.95507E-01

G.3.2 Risk

Estimated incremental cancer risks by radionuclide using the three approaches described above for the two California facilities are presented in Exhibit G-8 in table form. Note that the estimated cancer risks associated with mass emissions as reported in NEI are above or close to unity, indicating an extremely high probability that cancer could occur in the exposed individual.[5] Estimated maximum individual risks calculated using emissions generated with the clinker production- and PM emission-scaling factors range between approximately 2E-09 and 1E-05.

Exhibit G-8. Risk Calculated for Two California Portland Cement Facilities Using AERMOD Modeling Results and Three Emission Estimation Approaches

NTI Site ID	Conc. (µg/m^3)	MIR, Based on NEI Emissions and Speciation Assumptions		MIR, Based on Clinker Production Scaling Factors		MIR, Based on PM Emission Scaling Factors	
		^{210}Po	^{222}Rn	^{210}Po	^{222}Rn	^{210}Po	^{222}Rn
NEICA1505122	1.53E-03	2.94E+02	9.86E-01	8.09E-06	2.71E-08	6.07E-07	2.04E-09
NEI2CA151186	2.53E-03	8.43E-04	2.83E-06	1.14E-05	3.82E-08	1.44E-06	4.82E-09

Maximum incremental risk estimates from CAP88 for each radionuclide are presented in Exhibit G-9. In general, these estimates tend to be lower than those calculated using the AERMOD model, except (notably) for the Lehigh facility, where the calculated incremental risks are approximately an order of magnitude higher. A summary of total risks (taking into account both radioactive isotopes that are assumed to be emitted) estimated using each approach is presented in Exhibit G-10 in chart form and Exhibit G-11 in table form.

[5] Typically, if estimated cancer risks are calculated to be extremely high (e.g., > 0.01), alternate risk characterization equations are appropriate. This adjustment has not been made in this case because the estimated risks are being used primarily for evaluative purposes.

Exhibit G-9. Risk for Two California Portland Cement Facilities Modeled with CAP88 and Two Emission Estimation Approaches

NTI Site ID	MIR, Based on NEI Emissions and Speciation Assumptions		MIR, Based on Clinker Production Scaling Factors	
	^{210}Po	^{222}Rn	^{210}Po	^{222}Rn
NEICA1505122	3.95E+01	4.11E+00	1.09E-06	1.13E-07
NEI2CA151186	7.15E-05	8.06E-06	9.66E-07	1.09E-07

Exhibit G-10. Estimation of Maximum Incremental Risk from Estimated Radionuclide Emissions for the Two California Facilities Using Three Approaches and Two Modeling Systems

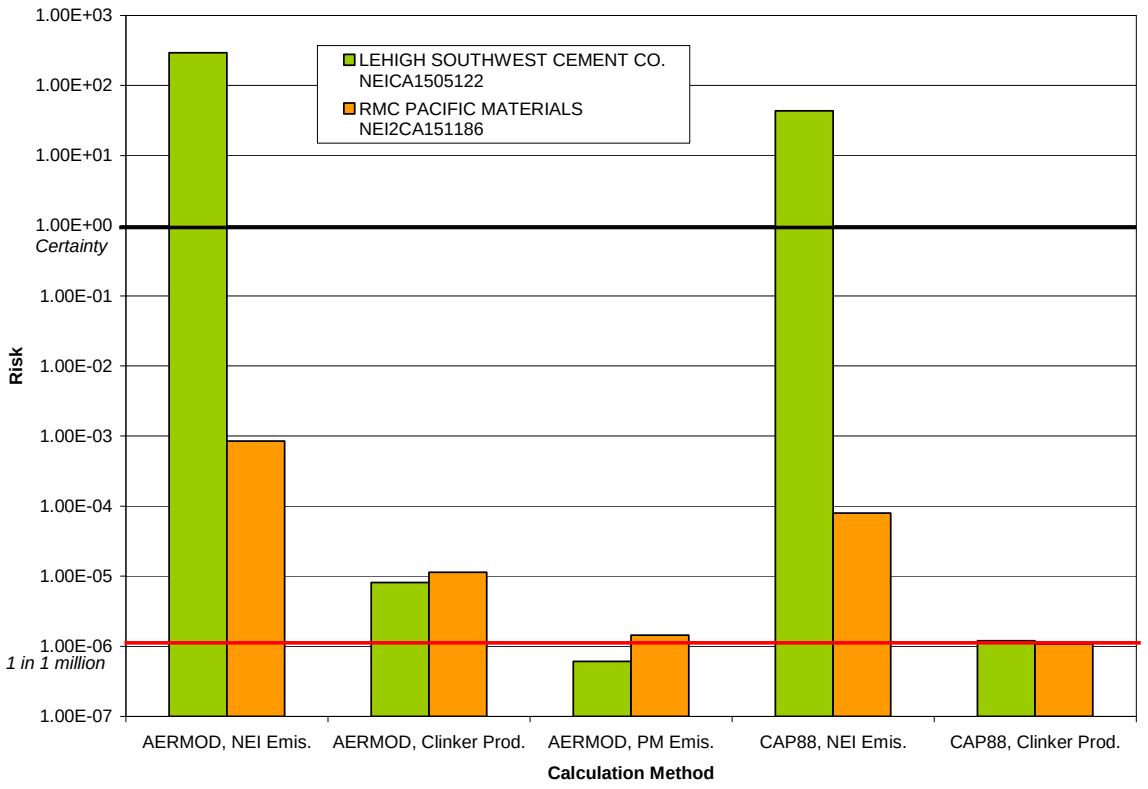


Exhibit G-11. Summary of Maximum Incremental Cancer Risk Modeled in AERMOD and CAP88, Using Different Radionuclide Emission Estimates

NTI Site ID	Total MIR, Based on AERMOD Model and:			Total MIR, Based on CAP88 Model and:	
	NEI Emissions and Speciation Assumptions	Clinker Production Scaling Factors	PM Emission Scaling Factors	NEI Emissions and Speciation Assumptions	Clinker Production Scaling Factors
NEICA1505122	2.95E+02	8.12E-06	6.09E-07	4.36E+01	1.20E-06
NEI2CA151186	8.46E-04	1.14E-05	1.44E-06	7.96E-05	1.08E-06

Examination of the results presented in Exhibit G-12 indicate that the maximum incremental risk estimates produced by both models, using non-NEI emission estimates, are near or above one in a million population. Using NEI mass emission estimates for radionuclides appears to result in unrealistically high maximum incremental risk estimates regardless of the dispersion model used. In particular, the NEI emission estimates for the Monolith facility are extremely high, indicating that the emissions reported in NEI may be incorrect, or that some of the assumptions used to analyze the emissions are overly conservative.

Both models attributed higher risks from ^{210}Po relative to risks from ^{222}Rn, although the CAP88 model attributed a much higher proportion (approximately 10 percent) of risk to ^{222}Rn compared to those attributed to ^{222}Rn by AERMOD (0.3 percent). This large differential between the attribution of risk from each radionuclide by each model reflects large uncertainties introduced by the assumptions detailed in the modeling approach and also reflects that CAP88 accounts for daughter products whereas AERMOD does not.

The MIRs from all other emissions from these two California Portland cement facilities are 1E-07 and 6E-08 for the Monolith and Davenport facilities, respectively. For each facility, the maximum inhalation individual cancer risk for radionuclides predicted by the AERMOD modeling of clinker-production scaled emissions are between 1 to 3 orders of magnitude higher than the inhalation MIRs predicted by AERMOD modeling for all other facility emissions, and the CAP88 predicted risks from clinker production-scaled emissions are between 1 to 2 orders of magnitude higher. This indicates that radionuclide emissions may be the primary driver for cancer risk from Portland cement facilities. However, the extremely poor quality of available radionuclide emissions data prompts caution in the interpretation of these risk values, especially when comparing to better characterized risks.

Maximum incremental risks were estimated for each of the 91 Portland cement facilities in the United States using the HEM-3 model as described in Section G.2.4 above. The results of the HEM-3 modeling are presented in Exhibit G-12, listed from highest to lowest estimated risk.

In general, maximum incremental risks from each facility were approximately 300 times higher for ^{210}Po than for ^{222}Rn. Of the 91 domestic Portland cement facilities, four are estimated to have maximum cancer risk higher than 1E-04, or 1 person per 10,000. Approximately 35 percent of the facilities (32) are estimated to have maximum cancer risk higher than 1E-05, or 1 person per 100,000, and all but one facility had maximum cancer risk higher than 1E-06, or 1 person per 1 million. Only for two facilities was the cancer risk greater than 1E-06 due to ^{222}Rn emissions.

Exhibit G-12. Estimation of Maximum Incremental Risk (MIR) for Cancer from Radionuclide Emissions for All U.S. Portland Cement Facilities, Based on the Clinker Production Scaling Factor

NTI Site ID	MIR	
	^{210}Po	^{222}Rn
NEI22453	3.48E-04	1.17E-06
NEI22838	3.16E-04	1.06E-06
NEIFL0860020	1.92E-04	6.45E-07
NEI22743	1.56E-04	5.24E-07
NEI338	6.28E-05	2.11E-07
NEI22900	5.08E-05	1.71E-07
NEI7376	4.42E-05	1.49E-07
NEIT$FNP1408	4.39E-05	1.48E-07
NEI2PRT14367	3.48E-05	1.17E-07
NEIAZ0250421	3.14E-05	1.06E-07
NEIID0050004	3.05E-05	1.03E-07
NEI24859	2.60E-05	8.75E-08
NEI33699	2.46E-05	8.29E-08
NEI18621	1.96E-05	6.59E-08
NEINYT$1163	1.86E-05	6.27E-08
NEI32033	1.86E-05	6.25E-08
NEISDT$8989	1.74E-05	5.85E-08
NEINY0394192	1.73E-05	5.82E-08
NEI20046	1.71E-05	5.74E-08
NEI51435	1.54E-05	5.17E-08
NEISC0351244	1.46E-05	4.91E-08
NEI12739	1.41E-05	4.76E-08
NEIIA0330060	1.40E-05	4.71E-08
NEI446	1.35E-05	4.55E-08
NEITN0653070	1.26E-05	4.23E-08
NEIOK4013107	1.23E-05	4.13E-08
NEI2PRT14359	1.21E-05	4.06E-08
NEI2CA151186	1.14E-05	3.83E-08
NEITN0930008	1.11E-05	3.74E-08
NEITX139099J	1.08E-05	3.62E-08
NEINY4192600	1.06E-05	3.56E-08
NEI31319	9.75E-06	3.28E-08
NEIPAT$1626	9.30E-06	3.13E-08
NEITXRBG0259	9.17E-06	3.09E-08
NEIMT0430001	8.96E-06	3.02E-08
NEIKYR0060	8.91E-06	3.00E-08
NEINMT$12442	8.90E-06	3.00E-08
NEI52351	8.89E-06	2.99E-08
NEIAL1150002	8.88E-06	2.99E-08
NEIOK1826	8.37E-06	2.82E-08
NEITXT$11924	8.29E-06	2.79E-08

Exhibit G-12. Estimation of Maximum Incremental Risk (MIR) for Cancer from Radionuclide Emissions for All U.S. Portland Cement Facilities, Based on the Clinker Production Scaling Factor

NTI Site ID	MIR	
	^{210}Po	^{222}Rn
NEIPA58-1290	8.28E-06	2.79E-08
NEICA1505122	8.09E-06	2.72E-08
NEIOHT$6526	8.03E-06	2.70E-08
NEIFLR001008	7.71E-06	2.60E-08
NEI7255	7.53E-06	2.53E-08
NEIAL321	7.46E-06	2.51E-08
NEI13290	7.15E-06	2.41E-08
NEI886	7.00E-06	2.36E-08
NEITXT$11872	6.42E-06	2.16E-08
NEI12976	6.34E-06	2.14E-08
NEI2PA110039	6.29E-06	2.12E-08
NEIPA94-2626	6.18E-06	2.08E-08
NEI22877	5.80E-06	1.95E-08
NEIAL8026	5.48E-06	1.85E-08
NEI12018	5.26E-06	1.77E-08
NEIWA0331133	5.24E-06	1.76E-08
NEIWV0030006	4.97E-06	1.67E-08
NEIALT$4449	4.96E-06	1.67E-08
NEI51352	4.72E-06	1.59E-08
NEI34931	4.47E-06	1.50E-08
NEI40539	4.27E-06	1.44E-08
NEIGA1530003	4.18E-06	1.41E-08
NEI12238	3.98E-06	1.34E-08
NEIAL1170004	3.91E-06	1.32E-08
NEI46744	3.86E-06	1.30E-08
NEIIA0330035	3.70E-06	1.25E-08
NEIMIB1743	3.69E-06	1.24E-08
NEITXT$11980	3.56E-06	1.20E-08
NEI572	3.53E-06	1.19E-08
NEIMIB1559	3.41E-06	1.15E-08
NEITXT$12011	3.39E-06	1.14E-08
NEIPA23-0797	3.34E-06	1.12E-08
NEITX309123F	3.29E-06	1.11E-08
NEI51527	3.22E-06	1.09E-08
NEIMO0990002	3.22E-06	1.08E-08
NEI33394	3.09E-06	1.04E-08
NEI16357	3.02E-06	1.02E-08
NEI42038	3.01E-06	1.01E-08
NEIWA0331404	2.96E-06	9.97E-09
NEI33444	2.79E-06	9.39E-09
NEIVA2553	2.76E-06	9.29E-09

Exhibit G-12. Estimation of Maximum Incremental Risk (MIR) for Cancer from Radionuclide Emissions for All U.S. Portland Cement Facilities, Based on the Clinker Production Scaling Factor

NTI Site ID	MIR	
	^{210}Po	^{222}Rn
NEI20130	2.48E-06	8.36E-09
NEI26327	2.28E-06	7.68E-09
NEI34326	2.00E-06	6.72E-09
NEIME0130002	1.77E-06	5.94E-09
NEI25375	1.53E-06	5.15E-09
NEI26277	1.52E-06	5.12E-09
NEIPA01993-1	1.27E-06	4.29E-09
NEIMT0310005	1.15E-06	3.89E-09
NEIUT10303	3.54E-07	1.19E-09

To provide context for the estimated incremental risks, background radiation risks can be estimated using reported values for natural radiological background concentrations. Exhibit G-13 shows the global background radiation dose by exposure route. Using the generic radiation risk estimate developed by the U.S. Department of Energy (2002) of 0.06 cancer mortality per sievert (Sv), the total cancer mortality risk from background radiation is calculated to be 1.4E-04. This estimate of background radiation cancer mortality risk is higher than or approximately equal to the MIR estimates from radionuclide emissions calculated with clinker production and PM emission-scaling factors for the two California facilities. Note that the background risks discussed here are from multiple pathways, including ingestion and dermal exposure, although inhalation exposure accounts for half of the total background dose.

Exhibit G-13. Annual Effective Radiation Dose from Background Sources [a]

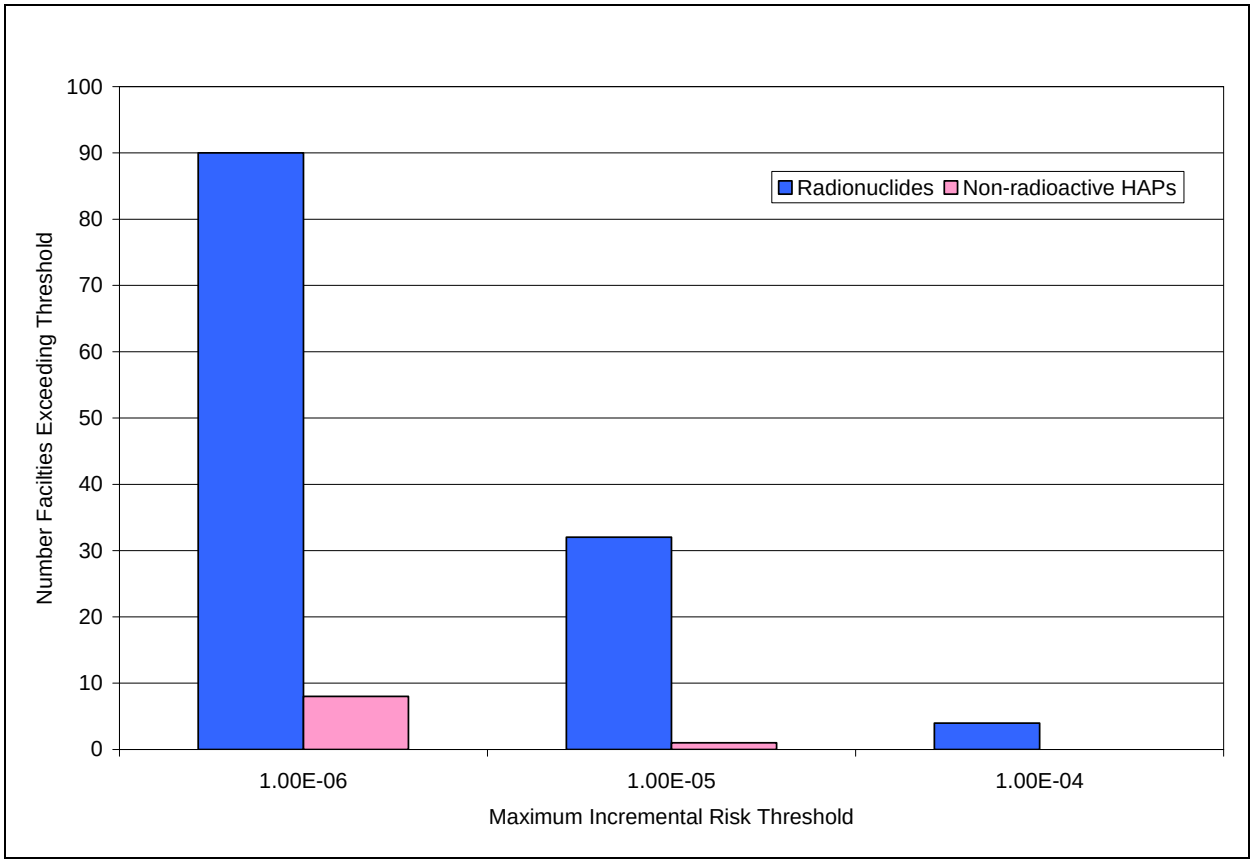
Exposure Route	Radiation Dose (mSv)[b]	Estimated MIR
Ingestion	0.3	2E-05
Inhalation	1.2	7.2E-05
Cosmic rays	0.4	2E-05
Terrestrial Gamma rays	0.5	3E-05
Total	2.4	1.4E-04

[a] United Nations 2000.
[b] mSv = millisievert

Radionuclide emissions from Portland cement facilities may represent relatively high incremental cancer risks, as shown by this analysis. Comparing radionuclide risks to high inhalation risks from emissions of other non-radioactive HAPs from these facilities may be informative. According to an analysis performed by EPA (2008d), maximum individual lifetime cancer risk from emissions of non-radioactive HAPs from domestic Portland cement facilities exceed 1E-6 for only 8 of 91 facilities, compared to 90 facilities potentially exceeding the same threshold due to radionuclide emissions. Similarly, the analysis showed that only 1 Portland cement facility exceeded the 1E-5 threshold (to a level of 5E-5, the highest reported for all of the facilities) due to non-radioactive HAP emissions, while 32 exceeded the same level due to radionuclide emissions. A summary of the comparison is presented graphically in Exhibit G-14.

No facilities were shown to exceed the 1E-4 level due to non-radioactive HAP emissions, while four exceeded it due to radionuclide emissions. Radionuclide emissions may therefore be the HAP emissions of greatest concern from Portland cement facilities.

Exhibit G-14. Comparison of Maximum Incremental Cancer Risk from Radionuclide Emissions and Non-Radioactive HAP Emissions from Portland Cement Facilities



G.4 Uncertainties

Several factors, mostly related to the poor quality of existing data on radionuclide emissions from Portland cement production facilities, contribute to uncertainties regarding the estimation of their incremental cancer risks. First, the lack of direct measurements of radionuclides at U.S. facilities available for this analysis makes evaluation of incremental cancer risks much more difficult. Additional data of this type would serve as substantial evidence to support (or refute) the claims and assumptions made in this analysis. Second, this analysis has relied heavily on emissions from the Maastricht facility measured in 1990. Use of these facility data implicitly assumes that U.S. and European Portland cement facilities have equivalent input materials, levels of emission control, and emission profiles. Third, the analysis relies upon many assumptions and model parameterizations. Although many of the assumptions are conservative, what their overall effect is on the final risk estimates is not entirely clear. Finally, the shortcomings in the model formulations for both CAP88 and AERMOD may lead to inaccuracies in risk estimation. For instance, CAP88 has a static mixing height and may therefore underestimate exposure during inversion events, because actual doses and resultant risks may be higher during periods when the mixing height is low (i.e., during an inversion event). The most important remedy to reduce these uncertainties would be to obtain actual

measured, speciated radionuclide emission data from U.S. Portland cement manufacturing facilities.

G.5 References

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Appendix H: Detailed assessment inputs and results for Portland cement manufacturing facilities

Table 1 - Facility Identification Information

Source Category	Facility NEI ID	Facility Name	Address	City	State
Portland Cement	PTC_NEI12018	LAFARGE NORTH AMERICA - ALPENA PLANT	1435 FORD AVE. P.O. BOX 396	ALPENA	MI
Portland Cement	PTC_NEI12238	LAFARGE NORTH AMERICA INC.	301 EAST FRONT STREET	BUFFALO	IA
Portland Cement	PTC_NEI12739	MONARCH CEMENT COMPANY (THE)	S10-T26S-R18E	HUMBOLDT	KS
Portland Cement	PTC_NEI12976	LONE STAR IND INC DBA BUZZI UNICEM USA-PRYOR	E 5 MI ON HWY 20 THEN S 2 MI	PRYOR	OK
Portland Cement	PTC_NEI13290	HUNTER PLANT	7781 F.M. 1102	NEW BRAUNFELS	TX
Portland Cement	PTC_NEI16357	HEARTLAND CEMENT COMPANY	1765 LIMESTONE LANE	INDEPENDENCE	KS
Portland Cement	PTC_NEI18621	ARIZONA PORTLAND CEMENT COMPANY	11115 N. CASA GRANDE HIGHWAY	RILLITO	AZ
Portland Cement	PTC_NEI20046	CALIFORNIA PORTLAND CEMENT CO.	9350 OAK CREEK ROAD	MOJAVE	CA
Portland Cement	PTC_NEI20130	NATIONAL CEMENT CO	1 MI. N OF HWY 138-1 MI E/I-5	LEBEC	CA
Portland Cement	PTC_NEI22453	RIVERSIDE CEMENT CO UNIT NO.04	1500 RUBIDOUX BLVD	RIVERSIDE	CA
Portland Cement	PTC_NEI22743	CALIFORNIA PORTLAND CEMENT CO	695 S RANCHO AV	COLTON	CA
Portland Cement	PTC_NEI22838	MITSUBISHI CEMENT 2000	5808 STATE HIGHWAY 18	LUCERNE VALLEY	CA
Portland Cement	PTC_NEI22877	TXI RIVERSIDE CEMENT COMPANY	19409 NATIONAL TRAILS HIGHWAY	ORO GRANDE	CA
Portland Cement	PTC_NEI22900	CEMEX - RIVER PLANT	16888 NORTH 'E' STREET	VICTORVILLE	CA
Portland Cement	PTC_NEI24859	HANSON PERMANENTE CEMENT	24001 STEVENS CREEK BLVD	CUPERTINO	CA
Portland Cement	PTC_NEI25375	LEHIGH SOUTHWEST CEMENT CO.	15390 WONDERLAND BLVD	REDDING	CA
Portland Cement	PTC_NEI26277	RINKER MATERIALS CORPORATION.	1200 NW 137TH AVE	MIAMI	FL
Portland Cement	PTC_NEI26327	FLORIDA CRUSHED STONE CO., INC.	10311 CEMENT PLANT ROAD	BROOKSVILLE	FL
Portland Cement	PTC_NEI2CA151186	RMC PACIFIC MATERIALS	HIGHWAY ONE	DAVENPORT	CA
Portland Cement	PTC_NEI2PA110039	LEHIGH CEMENT/EVANSVILLE CEMENT PLT & QUARRY	537 EVANSVILLE RD	FLEETWOOD	PA
Portland Cement	PTC_NEI2PRT14359	PUERTO RICAN CEMENT CO. INC.	STATE RD. 123 KM. 8.0	PONCE	PR
Portland Cement	PTC_NEI2PRT14367	ESSROC SAN JUAN INC.	PR HAIGHWAY #2 KM 26.7	DORADO	PR
Portland Cement	PTC_NEI31319	ESSROC CEMENT CORP.	HIGHWAY 31	SPEED	IN
Portland Cement	PTC_NEI32033	LEHIGH CEMENT COMPANY	121 NORTH FIRST STREET	MITCHELL	IN
Portland Cement	PTC_NEI33394	LEHIGH PORTLAND CEMENT	117 MAIN STREET, SOUTH	UNION BRIDGE	MD
Portland Cement	PTC_NEI33444	ESSROC CEMENT	4120 BUCKEYSTOWN PIKE	BUCKEYSTOWN	MD

Table 1 - Facility Identification Information

Source Category	Facility NEI ID	Facility Name	Address	City	State
Portland Cement	PTC_NEI33699	INDEPENDENT CEMENT/ST. LAWERENCE	1260 SECURITY ROAD, EXTENDED	HAGERSTOWN	MD
Portland Cement	PTC_NEI338	MOUNTAIN CEMENT CO	PO BOX 339	LARAMIE	WY
Portland Cement	PTC_NEI34326	LAFARGE NORTH AMERICA INC-INDEPENDENCE PLANT	2200 N COURTNEY ROAD	SUGAR CREEK	MO
Portland Cement	PTC_NEI34931	LAFARGE BUILDING MATERIALS INC	RT 9W	COEYMANS	NY
Portland Cement	PTC_NEI40539	ASH GROVE CEMENT COMPANY	33060 SHIRTTAIL CREEK RD	DURKEE	OR
Portland Cement	PTC_NEI42038	DEVIL'S SLIDE PLANT	6055 E. CROYDON RD.	MORGAN	UT
Portland Cement	PTC_NEI446	CEMEX, INC. - LYONS CEMENT PLANT	5134 UTE HWY	LYONS AREA	CO
Portland Cement	PTC_NEI46744	CEMEX INC/WAMPUM CEMENT PLT	2001 PORTLAND PARK	WAMPUM	PA
Portland Cement	PTC_NEI51352	ILLINOIS CEMENT CO	1601 ROCKWELL RD	LASALLE	IL
Portland Cement	PTC_NEI51435	LONE STAR INDUSTRIES INC	PORTLAND AVE	OGLESBY	IL
Portland Cement	PTC_NEI51527	DIXON-MARQUETTE CEMENT INC	1914 WHITE OAK LN	DIXON	IL
Portland Cement	PTC_NEI52351	LAFARGE MIDWEST INC	2500 PORTLAND RD	GRAND CHAIN	IL
Portland Cement	PTC_NEI572	ASH GROVE CEMENT CO	16215 HIGHWAY 50	LOUISVILLE	NE
Portland Cement	PTC_NEI7255	ESSROC/NAZARETH LOWER CEMENT PLT 1	ROUTE 248 AND EASTON RD	NAZARETH	PA
Portland Cement	PTC_NEI7376	NORTH TEXAS CEMENT CO.	2 MI. N.E. OF MIDLOTHIAN, TX.	MIDLOTHIAN	TX
Portland Cement	PTC_NEI886	HOLCIM (US) INC. PORTLAND PLANT	3500 HWY 120	FLORENCE, 3.8 MI E	CO
Portland Cement	PTC_NEIAL1150002	NATIONAL CEMENT CO OF ALABAMA	LOCATION ADDRESS IS NEEDED	RAGLAND	AL
Portland Cement	PTC_NEIAL1170004	LAFARGE BUILDING MATERIALS	8039 HWY 25	CALERA	AL
Portland Cement	PTC_NEIAL321	CEMEX, INC.	1617 ARCOLA ROAD	DEMOPOLIS	AL
Portland Cement	PTC_NEIAL8026	HOLCIM INC	3051 HAMILTON BLVD	THEODORE	AL
Portland Cement	PTC_NEIALT$4449	LEHIGH CEMENT COMPANY	8401 SECOND AVENUE	LEEDS, AL	AL
Portland Cement	PTC_NEIAZ0250421	PHOENIX CEMENT CO.	3000 W. CEMENT PLANT RD.	CLARKDALE	AZ
Portland Cement	PTC_NEICA1505122	LEHIGH SOUTHWEST CEMENT CO.	13573 TEHACHAPI BLVD.	MONOLITH	CA
Portland Cement	PTC_NEIFL0860020	TARMAC AMERICA LLC	11000 NW 121 WAY	MEDLEY	FL
Portland Cement	PTC_NEIFLR001008	FLORIDA ROCK INDUSTRIES, INC.	CR 235, 2.5 MILES NE OF CITY	NEWBERRY	FL
Portland Cement	PTC_NEIGA1530003	CEMEX, INC.	2720 HWY 341 SOUTH	CLINCHFIELD	GA
Portland Cement	PTC_NEIIA0330035	LEHIGH CEMENT COMPANY - MASON CITY	700 25TH STREET NW	MASON CITY	IA
Portland Cement	PTC_NEIIA0330060	HOLCIM (US) INC. - MASON CITY	1840 N. FEDERAL AVE	MASON CITY	IA
Portland Cement	PTC_NEIID0050004	ASH GROVE CEMENT	230 CEMENT ROAD	INKOM	ID
Portland Cement	PTC_NEIKYR0060	KOSMOS CEMENT CO	15301 DIXIE HIGHWAY	KOSMOSDALE	KY

Table 1 - Facility Identification Information

Source Category	Facility NEI ID	Facility Name	Address	City	State
Portland Cement	PTC_NEIME0130002	DRAGON PRODUCTS CO INC - THOMASTON	US RT 1	THOMASTON	ME
Portland Cement	PTC_NEIMIB1559	CEMEX, INC.	16000 BELLS BAY RD	CHARLEVOIX	MI
Portland Cement	PTC_NEIMIB1743	HOLCIM (US) INC.	15215 DAY RD	DUNDEE	MI
Portland Cement	PTC_NEIMO0990002	RC CEMENT COMPANY INC-RIVER CEMENT CO - SELMA PLAN	1000 RIVER CEMENT ROAD	FESTUS	MO
Portland Cement	PTC_NEIMT0310005	HOLCIM US INC - TRIDENT PLANT	4070 TRIDENT RD	THREE FORKS	MT
Portland Cement	PTC_NEIMT0430001	ASH GROVE CEMENT	100 HIGHWAY 518	CLANCY	MT
Portland Cement	PTC_NEINMT$12442	GCC RIO GRANDE, INC. TIJERAS PLANT	11783 STATE HIGHWAY 14 S	TIJERAS	NM
Portland Cement	PTC_NEINY0394192	GLENS FALLS LEHIGH CEMENT COMPANY	120 ALPHA ROAD, OFF ROUTE 9W	CATSKILL	NY
Portland Cement	PTC_NEINY4192600	ST LAWRENCE CEMENT CORP-CATSKILL QUARRY	RT 9W	CATSKILL	NY
Portland Cement	PTC_NEINYT$1163	GLENS FALLS LEHIGH CEMENT COMPANY	313 WARREN ST	GLENS FALLS	NY
Portland Cement	PTC_NEIOHT$6526	CEMEX, INC.	3250 LINEBAUGH ROAD	XENIA	OH
Portland Cement	PTC_NEIOK1826	HOLCIM US INC	1100 W 18TH ST	ADA	OK
Portland Cement	PTC_NEIOK4013107	LAFARGE BDLG MATERIALS	2609 N 145TH E AVE	TULSA	OK
Portland Cement	PTC_NEIPA01993_1	ARMSTRONG CEMENT & SUPPLY/WINFIELD	100 CLEARFIELD RD	CABOT	PA
Portland Cement	PTC_NEIPA23_0797	LEHIGH CEMENT CO/YORK OPERATIONS	HOKES MILL RD	YORK	PA
Portland Cement	PTC_NEIPA58_1290	LAFARGE CORP/WHITEHALL PLT	5160 MAIN ST	WHITEHALL	PA
Portland Cement	PTC_NEIPA94_2626	HERCULES CEMENT CO LP/STOCKERTOWN	501 CENTER ST	STOCKERTOWN	PA
Portland Cement	PTC_NEIPAT$1626	ESSROC/BESSEMER	SECOND ST	BESSEMER	PA
Portland Cement	PTC_NEISC0351244	LAFARGE BUILDING MATERIALS HARLEYVILLE	463 JUDGE ST	HARLEYVILLE	SC
Portland Cement	PTC_NEISDT$8989	GCC DACOTAH	501 N ST ONGE STREET	RAPID CITY	SD
Portland Cement	PTC_NEIT$FNP1408	BALCONES PLANT	AT THE INTERSECTION OF WALD & SOLMS ROADS	NEW BRAUNFELS	TX
Portland Cement	PTC_NEITN0653070	SIGNAL MOUNTAIN CEMENT CO.	1201 SUCK CREEK ROAD	CHATTANOOGA	TN
Portland Cement	PTC_NEITN0930008	CEMEX, INC.	6212 CEMENT PLANT ROAD	KNOXVILLE	TN
Portland Cement	PTC_NEITX139099J	HOLCIM (TEXAS) LP	1800 DOVE LN.	MIDLOTHIAN	TX

Table 1 - Facility Identification Information

Source Category	Facility NEI ID	Facility Name	Address	City	State
Portland Cement	PTC_NEITX309123F	LEHIGH PORTLAND CEMENT	100 S WICKSON	WACO	TX
Portland Cement	PTC_NEITXRBG0259	1604 PLANT	6055 W GREEN MOUNTAIN ROAD	SAN ANTONIO	TX
Portland Cement	PTC_NEITXT$11872	PORTLAND CEMENT	11551 NACOGDOCHES ROAD	SAN ANTONIO	TX
Portland Cement	PTC_NEITXT$11924	TEXAS LEHIGH CEMENT CO.	1000 JACK C. HAYS TRAIL	BUDA	TX
Portland Cement	PTC_NEITXT$11980	MARYNEAL CEMENT PLANT	0.5 MI. N.W. ON F.M. 608	MARYNEAL	TX
Portland Cement	PTC_NEITXT$12011	CEMEX CEMENT OF TEXAS L.P.	16501 W. MURPHY	ODESSA	TX
Portland Cement	PTC_NEIUT10303	LEAMINGTON CEMENT PLANT	HWY 132	LEAMINGTON	UT
Portland Cement	PTC_NEIVA2553	ROANOKE CEMENT COMPANY	6071 CATAWBA ROAD	TROUTVILLE	VA
Portland Cement	PTC_NEIWA0331133	ASH GROVE CEMENT CO, E MARGINAL	3801 E MARGINAL WAY S	SEATTLE	WA
Portland Cement	PTC_NEIWA0331404	LAFARGE NORTH AMERICA INC	5400 W MARGINAL WAY SW	SEATTLE	WA
Portland Cement	PTC_NEIWV0030006	CAPITOL CEMENT CORPORATION	1826 SOUTH QUEEN STREET	MARTINSBURG	WV

Table 2 – Maximum Predicted HEM-3 Chronic Risks

Source Category	Facility NEI ID	Chronic Risk [1]		
		Cancer MIR	Cancer Incidence	Noncancer Max HI
Portland Cement	PTC_NEI12018	6.52E-07	4.95E-05	1.57E-02
Portland Cement	PTC_NEI12238	1.63E-07	7.84E-05	7.03E-03
Portland Cement	PTC_NEI12739	4.50E-07	1.04E-05	1.93E-02
Portland Cement	PTC_NEI12976	8.24E-09	4.13E-07	3.53E-04
Portland Cement	PTC_NEI13290	1.51E-07	5.03E-05	1.04E-02
Portland Cement	PTC_NEI16357	7.04E-09	3.17E-07	4.85E-03
Portland Cement	PTC_NEI18621	7.07E-08	1.06E-05	3.66E-03
Portland Cement	PTC_NEI20046	3.65E-08	3.80E-06	2.01E-03
Portland Cement	PTC_NEI20130	2.21E-08	2.17E-06	3.11E-03
Portland Cement	PTC_NEI22453	**5.11E-05**	8.64E-04	3.89E-01
Portland Cement	PTC_NEI22743	3.37E-07	3.27E-05	9.93E-03
Portland Cement	PTC_NEI22838	**2.16E-06**	7.62E-06	7.16E-02
Portland Cement	PTC_NEI22877	6.43E-07	4.66E-06	1.44E-01
Portland Cement	PTC_NEI22900	1.09E-07	1.35E-05	6.75E-06
Portland Cement	PTC_NEI24859	5.56E-08	6.25E-05	1.33E-08
Portland Cement	PTC_NEI25375	5.76E-09	2.91E-06	4.59E-04
Portland Cement	PTC_NEI26277	1.85E-08	8.46E-05	8.53E-04
Portland Cement	PTC_NEI26327	4.58E-08	1.86E-05	1.53E-03
Portland Cement	PTC_NEI2CA151186	5.51E-08	1.60E-05	2.25E-03
Portland Cement	PTC_NEI2PA110039	2.91E-08	2.04E-05	8.16E-03
Portland Cement	PTC_NEI2PRT14359	1.11E-07	3.31E-05	1.36E-02
Portland Cement	PTC_NEI2PRT14367	**6.57E-06**	4.37E-04	1.36E-01
Portland Cement	PTC_NEI31319	1.96E-07	7.47E-05	1.91E-02
Portland Cement	PTC_NEI32033	3.69E-07	2.58E-05	2.86E-02
Portland Cement	PTC_NEI33394	4.62E-08	6.56E-05	2.69E-03
Portland Cement	PTC_NEI33444	6.80E-08	5.15E-05	5.35E-03
Portland Cement	PTC_NEI33699	3.20E-08	2.80E-06	1.50E-03
Portland Cement	PTC_NEI338	4.51E-07	4.30E-06	6.46E-02
Portland Cement	PTC_NEI34326	1.34E-08	3.17E-05	8.27E-04
Portland Cement	PTC_NEI34931	1.52E-07	1.62E-04	7.44E-04
Portland Cement	PTC_NEI40539	3.01E-07	1.10E-06	2.86E-03
Portland Cement	PTC_NEI42038	8.12E-09	1.11E-06	7.46E-05
Portland Cement	PTC_NEI446	2.89E-08	6.31E-06	2.49E-03
Portland Cement	PTC_NEI46744	6.98E-08	3.58E-05	8.09E-03
Portland Cement	PTC_NEI51352	5.28E-08	8.42E-06	2.14E-03
Portland Cement	PTC_NEI51435	2.50E-07	9.34E-06	9.22E-03
Portland Cement	PTC_NEI51527	7.06E-07	1.22E-04	1.86E-01

Table 2 – Maximum Predicted HEM-3 Chronic Risks

Source Category	Facility NEI ID	Chronic Risk [1]		
		Cancer MIR	Cancer Incidence	Noncancer Max HI
Portland Cement	PTC_NEI52351	8.43E-08	6.34E-06	2.40E-03
Portland Cement	PTC_NEI572	9.72E-09	7.26E-06	1.12E-03
Portland Cement	PTC_NEI7255	2.66E-08	2.34E-05	4.22E-03
Portland Cement	PTC_NEI7376	9.78E-07	3.01E-04	1.47E-02
Portland Cement	PTC_NEI886	1.20E-07	1.33E-05	4.58E-04
Portland Cement	PTC_NEIAL1150002	**4.19E-06**	7.22E-04	2.40E-03
Portland Cement	PTC_NEIAL1170004	8.37E-09	5.14E-06	1.02E-05
Portland Cement	PTC_NEIAL321	2.29E-07	7.29E-06	1.88E-01
Portland Cement	PTC_NEIAL8026	1.79E-07	9.11E-05	6.25E-04
Portland Cement	PTC_NEIALT$4449	1.06E-08	8.27E-06	1.67E-04
Portland Cement	PTC_NEIAZ0250421	**1.72E-06**	4.90E-05	2.95E-02
Portland Cement	PTC_NEICA1505122	1.19E-07	5.25E-06	6.08E-04
Portland Cement	PTC_NEIFL0860020	**1.95E-06**	7.53E-04	6.90E-02
Portland Cement	PTC_NEIFLR001008	2.34E-08	2.80E-06	7.64E-04
Portland Cement	PTC_NEIGA1530003	8.94E-09	1.31E-06	3.57E-04
Portland Cement	PTC_NEIIA0330035	1.59E-07	2.27E-05	4.14E-03
Portland Cement	PTC_NEIIA0330060	2.59E-07	2.64E-05	5.66E-04
Portland Cement	PTC_NEIID0050004	3.24E-07	3.79E-06	1.71E-03
Portland Cement	PTC_NEIKYR0060	1.90E-08	1.96E-05	4.39E-04
Portland Cement	PTC_NEIME0130002	6.85E-08	8.11E-06	4.14E-05
Portland Cement	PTC_NEIMIB1559	1.72E-07	2.43E-05	7.67E-03
Portland Cement	PTC_NEIMIB1743	**1.59E-06**	1.59E-03	8.94E-02
Portland Cement	PTC_NEIMO0990002	4.18E-09	3.67E-06	1.61E-03
Portland Cement	PTC_NEIMT0310005	1.46E-08	4.20E-07	2.37E-05
Portland Cement	PTC_NEIMT0430001	9.54E-08	6.39E-07	4.72E-04
Portland Cement	PTC_NEINMT$12442	2.79E-08	5.39E-06	9.47E-04
Portland Cement	PTC_NEINY0394192	1.88E-07	1.12E-05	2.78E-05
Portland Cement	PTC_NEINY4192600	2.41E-07	1.56E-05	3.67E-03
Portland Cement	PTC_NEINYT$1163	3.15E-07	1.38E-05	6.83E-02
Portland Cement	PTC_NEIOHT$6526	2.50E-08	2.01E-05	7.66E-04
Portland Cement	PTC_NEIOK1826	8.96E-08	2.89E-06	1.22E-02
Portland Cement	PTC_NEIOK4013107	3.71E-08	1.19E-05	8.31E-04
Portland Cement	PTC_NEIPA01993-1	1.50E-07	7.97E-05	1.25E-02
Portland Cement	PTC_NEIPA23-0797	1.06E-07	4.10E-05	6.46E-03
Portland Cement	PTC_NEIPA58-1290	8.81E-08	5.38E-05	8.79E-03
Portland Cement	PTC_NEIPA94-2626	1.57E-07	1.08E-04	1.27E-02
Portland Cement	PTC_NEIPAT$1626	2.99E-07	7.64E-05	3.79E-02

Table 2 – Maximum Predicted HEM-3 Chronic Risks

Source Category	Facility NEI ID	Chronic Risk [1]		
		Cancer MIR	Cancer Incidence	Noncancer Max HI
Portland Cement	PTC_NEISC0351244	9.11E-07	5.53E-05	1.95E-02
Portland Cement	PTC_NEISDT$8989	9.70E-08	1.02E-05	9.21E-03
Portland Cement	PTC_NEIT$FNP1408	1.76E-07	7.96E-05	5.71E-02
Portland Cement	PTC_NEITN0653070	6.22E-08	9.34E-06	1.73E-03
Portland Cement	PTC_NEITN0930008	3.87E-08	5.04E-06	7.51E-04
Portland Cement	PTC_NEITX139099J	3.00E-07	5.81E-04	1.63E-03
Portland Cement	PTC_NEITX309123F	3.50E-08	3.09E-06	2.14E-03
Portland Cement	PTC_NEITXRBG0259	5.85E-08	6.11E-05	3.77E-03
Portland Cement	PTC_NEITXT$11872	9.89E-08	1.75E-04	8.07E-04
Portland Cement	PTC_NEITXT$11924	1.77E-08	1.19E-05	4.53E-04
Portland Cement	PTC_NEITXT$11980	7.61E-09	1.46E-07	1.32E-02
Portland Cement	PTC_NEITXT$12011	7.24E-09	8.47E-07	3.17E-05
Portland Cement	PTC_NEIUT10303	7.58E-10	3.03E-08	1.97E-06
Portland Cement	PTC_NEIVA2553	9.61E-09	4.10E-06	1.90E-04
Portland Cement	PTC_NEIWA0331133	3.01E-08	1.05E-04	7.21E-05
Portland Cement	PTC_NEIWA0331404	3.17E-08	1.13E-04	1.01E-04
Portland Cement	PTC_NEIWV0030006	5.29E-08	1.49E-05	1.89E-04

[1] **BOLD** indicates a cancer risk great than 1 in a million or a noncancer risk greater than 1

Table 3 – Maximum Predicted Acute Risks (HEM-AERMOD)

Source Category	Facility NEI ID	Maximum Hazard Quotient[1]				
		REL	AEGL1	AEGL2	ERPG1	ERPG2
Portland Cement	PTC_NEI12018	6.86E-02	5.34E-02	4.37E-03	5.34E-02	4.37E-03
Portland Cement	PTC_NEI12238	8.82E-03	6.86E-03	5.61E-04	6.86E-03	5.61E-04
Portland Cement	PTC_NEI12739	1.69E-02	4.63E-03	3.78E-04	4.63E-03	3.78E-04
Portland Cement	PTC_NEI12976	1.72E-03	1.33E-03	1.09E-04	1.33E-03	1.09E-04
Portland Cement	PTC_NEI13290	1.05E-02	8.19E-03	6.70E-04	8.19E-03	6.70E-04
Portland Cement	PTC_NEI16357	5.47E-03	4.25E-03	3.48E-04	4.25E-03	3.48E-04
Portland Cement	PTC_NEI18621	1.67E-01	1.43E-02	1.12E-03	1.43E-02	1.12E-03
Portland Cement	PTC_NEI20046	5.11E-02	3.98E-02	3.25E-03	3.98E-02	3.25E-03
Portland Cement	PTC_NEI20130	5.09E-01	1.19E-02	7.71E-04	1.19E-02	7.71E-04
Portland Cement	PTC_NEI22453	**3.75E+00**	3.14E-01	2.03E-02	3.14E-01	3.16E-02
Portland Cement	PTC_NEI22743	1.14E-01	9.70E-03	6.28E-04	9.70E-03	6.28E-04
Portland Cement	PTC_NEI22838	3.74E-01	1.13E-01	9.26E-03	1.13E-01	9.26E-03
Portland Cement	PTC_NEI22877	1.22E+00	1.05E-01	6.76E-03	1.05E-01	6.76E-03
Portland Cement	PTC_NEI22900	5.31E-05	0.00E+00	8.03E-11	0.00E+00	3.64E-08
Portland Cement	PTC_NEI24859	2.56E-07	1.96E-09	2.51E-10	1.96E-09	5.95E-10
Portland Cement	PTC_NEI25375	1.50E-02	6.86E-03	5.62E-04	6.86E-03	5.62E-04
Portland Cement	PTC_NEI26277	6.12E-03	4.76E-03	3.89E-04	4.76E-03	3.89E-04
Portland Cement	PTC_NEI26327	3.94E-03	3.06E-03	2.51E-04	3.06E-03	2.51E-04
Portland Cement	PTC_NEI2CA151186	2.20E-01	6.73E-03	4.94E-04	6.73E-03	4.94E-04
Portland Cement	PTC_NEI2PA110039	5.83E-03	4.53E-03	3.71E-04	4.53E-03	3.71E-04
Portland Cement	PTC_NEI2PRT14359	6.33E-02	1.25E-02	1.02E-03	1.25E-02	1.02E-03
Portland Cement	PTC_NEI2PRT14367	1.02E+00	7.91E-01	6.47E-02	7.91E-01	6.47E-02
Portland Cement	PTC_NEI31319	4.01E-02	6.48E-03	5.30E-04	6.48E-03	5.30E-04
Portland Cement	PTC_NEI32033	3.54E-02	5.72E-03	4.68E-04	5.72E-03	4.68E-04
Portland Cement	PTC_NEI33394	1.06E-02	8.24E-03	6.74E-04	8.24E-03	6.74E-04
Portland Cement	PTC_NEI33444	5.93E-03	2.59E-05	6.27E-06	2.59E-05	5.33E-06
Portland Cement	PTC_NEI33699	9.80E-03	7.62E-03	6.24E-04	7.62E-03	6.24E-04
Portland Cement	PTC_NEI338	1.12E+00	9.58E-02	6.20E-03	9.58E-02	6.20E-03
Portland Cement	PTC_NEI34326	3.59E-02	3.07E-03	1.99E-04	3.07E-03	1.99E-04
Portland Cement	PTC_NEI34931	1.40E-01	0.00E+00	5.30E-05	0.00E+00	4.50E-05
Portland Cement	PTC_NEI40539	3.56E-01	6.94E-04	3.77E-04	6.94E-04	3.20E-04
Portland Cement	PTC_NEI42038	2.01E-02	0.00E+00	2.12E-05	0.00E+00	1.81E-05
Portland Cement	PTC_NEI446	7.76E-01	5.18E-03	8.22E-04	5.18E-03	6.98E-04
Portland Cement	PTC_NEI46744	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Portland Cement	PTC_NEI51352	6.79E-03	5.28E-03	4.32E-04	5.28E-03	4.32E-04
Portland Cement	PTC_NEI51435	5.68E-02	2.01E-02	1.65E-03	2.01E-02	1.65E-03
Portland Cement	PTC_NEI51527	5.39E-02	9.72E-04	7.95E-05	9.72E-04	7.95E-05
Portland Cement	PTC_NEI52351	6.85E-03	5.33E-03	4.36E-04	5.33E-03	4.36E-04
Portland Cement	PTC_NEI572	5.94E-02	5.07E-03	3.28E-04	5.07E-03	3.28E-04
Portland Cement	PTC_NEI7255	3.38E-02	3.62E-03	2.96E-04	3.62E-03	2.96E-04
Portland Cement	PTC_NEI7376	6.28E-02	5.37E-03	3.47E-04	5.37E-03	3.47E-04
Portland Cement	PTC_NEI886	2.47E-02	2.53E-03	2.07E-04	2.53E-03	2.07E-04
Portland Cement	PTC_NEIAL1150002	1.87E-02	1.45E-02	1.19E-03	1.45E-02	1.19E-03
Portland Cement	PTC_NEIAL1170004	5.71E-04	0.00E+00	6.05E-07	0.00E+00	5.14E-07
Portland Cement	PTC_NEIAL321	3.57E-01	1.20E-02	3.10E-03	1.20E-02	3.10E-03
Portland Cement	PTC_NEIAL8026	3.86E-03	3.12E-04	2.55E-05	3.12E-04	2.55E-05
Portland Cement	PTC_NEIALT$4449	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Portland Cement	PTC_NEIAZ0250421	**3.19E+01**	0.00E+00	9.91E-06	0.00E+00	8.42E-06
Portland Cement	PTC_NEICA1505122	8.63E-02	2.12E-03	1.73E-04	2.12E-03	1.73E-04
Portland Cement	PTC_NEIFL0860020	4.69E-02	5.94E-03	4.86E-04	5.94E-03	4.86E-04
Portland Cement	PTC_NEIFLR001008	1.08E-02	1.34E-03	1.10E-04	1.34E-03	1.10E-04
Portland Cement	PTC_NEIGA1530003	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Portland Cement	PTC_NEIIA0330035	6.12E-03	2.28E-03	1.86E-04	2.28E-03	1.86E-04

Table 3 – Maximum Predicted Acute Risks (HEM-AERMOD)

Source Category	Facility NEI ID	Maximum Hazard Quotient[1]				
		REL	AEGL1	AEGL2	ERPG1	ERPG2
Portland Cement	PTC_NEIIA0330060	2.24E-02	0.00E+00	3.77E-06	0.00E+00	9.39E-06
Portland Cement	PTC_NEIID0050004	3.55E-03	2.76E-03	2.26E-04	2.76E-03	2.26E-04
Portland Cement	PTC_NEIKYR0060	8.72E-03	6.78E-03	5.55E-04	6.78E-03	5.55E-04
Portland Cement	PTC_NEIME0130002	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Portland Cement	PTC_NEIMIB1559	5.93E-02	4.62E-02	3.78E-03	4.62E-02	3.78E-03
Portland Cement	PTC_NEIMIB1743	**2.41E+00**	6.64E-03	1.99E-03	6.64E-03	1.99E-03
Portland Cement	PTC_NEIMO0990002	8.90E-03	6.93E-03	5.67E-04	6.93E-03	5.67E-04
Portland Cement	PTC_NEIMT0310005	1.91E-02	0.00E+00	2.02E-05	0.00E+00	1.72E-05
Portland Cement	PTC_NEIMT0430001	5.16E-02	0.00E+00	5.46E-05	0.00E+00	4.64E-05
Portland Cement	PTC_NEINMT$12442	6.64E-03	5.16E-03	4.23E-04	5.16E-03	4.23E-04
Portland Cement	PTC_NEINY0394192	2.18E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Portland Cement	PTC_NEINY4192600	3.95E-02	0.00E+00	4.18E-05	0.00E+00	3.55E-05
Portland Cement	PTC_NEINYT$1163	1.95E-02	1.67E-03	1.08E-04	1.67E-03	1.08E-04
Portland Cement	PTC_NEIOHT$6526	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Portland Cement	PTC_NEIOK1826	1.50E-03	2.10E-04	5.44E-05	2.10E-04	5.44E-05
Portland Cement	PTC_NEIOK4013107	1.77E-01	1.51E-02	9.77E-04	1.51E-02	9.77E-04
Portland Cement	PTC_NEIPA01993-1	7.27E-05	5.65E-05	4.62E-06	5.65E-05	2.13E-04
Portland Cement	PTC_NEIPA23-0797	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.14E-04
Portland Cement	PTC_NEIPA58-1290	1.34E-03	1.04E-03	8.50E-05	1.04E-03	8.50E-05
Portland Cement	PTC_NEIPA94-2626	3.80E-03	7.21E-04	5.90E-05	7.21E-04	5.90E-05
Portland Cement	PTC_NEIPAT$1626	1.53E-02	2.26E-03	1.85E-04	2.26E-03	1.85E-04
Portland Cement	PTC_NEISC0351244	7.11E-02	5.34E-02	4.37E-03	5.34E-02	4.37E-03
Portland Cement	PTC_NEISDT$8989	1.88E-06	0.00E+00	1.99E-09	0.00E+00	1.69E-09
Portland Cement	PTC_NEIT$FNP1408	9.47E-02	1.33E-02	3.43E-03	1.33E-02	3.43E-03
Portland Cement	PTC_NEITN0653070	3.04E-01	2.60E-02	1.68E-03	2.60E-02	1.68E-03
Portland Cement	PTC_NEITN0930008	4.07E-02	0.00E+00	4.31E-05	0.00E+00	3.66E-05
Portland Cement	PTC_NEITX139099J	5.55E-03	2.39E-03	1.96E-04	2.39E-03	1.96E-04
Portland Cement	PTC_NEITX309123F	5.05E-03	3.93E-03	3.22E-04	3.93E-03	3.22E-04
Portland Cement	PTC_NEITXRBG0259	1.42E-03	1.99E-04	5.13E-05	1.99E-04	5.13E-05
Portland Cement	PTC_NEITXT$11872	5.38E-03	1.84E-03	1.51E-04	1.84E-03	1.51E-04
Portland Cement	PTC_NEITXT$11924	2.65E-03	2.06E-03	1.69E-04	2.06E-03	1.69E-04
Portland Cement	PTC_NEITXT$11980	1.02E-01	1.43E-02	3.70E-03	1.43E-02	3.70E-03
Portland Cement	PTC_NEITXT$12011	1.40E-02	0.00E+00	1.49E-05	0.00E+00	1.26E-05
Portland Cement	PTC_NEIUT10303	4.07E-03	0.00E+00	4.30E-06	0.00E+00	3.66E-06
Portland Cement	PTC_NEIVA2553	3.88E-03	3.01E-03	2.47E-04	3.01E-03	2.47E-04
Portland Cement	PTC_NEIWA0331133	2.97E-02	0.00E+00	3.15E-05	0.00E+00	2.68E-05
Portland Cement	PTC_NEIWA0331404	2.99E-02	0.00E+00	3.16E-05	0.00E+00	2.69E-05
Portland Cement	PTC_NEIWV0030006	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00

[1] Some maximum acute impacts may be at onsite locations.
Note: **BOLD** indicates acute risks greater than 1

Table 4 – Maximum Predicted Acute Risks Greater than 1 (Refined Approach)

Facility NEI ID	Pollutant	Criteria	HEM-3 (Screening)	HEM-3/ AERMOD (Refined) [1]	Refined Modeling Approach [2]
Portland Cement					
PTC_NEIAZ0250421	Nickel compounds	REL	3.19E+01		To be completedas part of RTR anlaysis
PTC_NEI22453	Mercury (elemental	REL	3.75E+00		To be completedas part of RTR anlaysis
PTC_NEI22453	Formaldehyde	REL	3.68E+00		To be completedas part of RTR anlaysis
PTC_NEIMIB1743	Acrolein	REL	2.41E+00		To be completedas part of RTR anlaysis

[1] Facilites with a HEM-3 screening acute value greater than 1 were remodeled with a more refined approach

[2] Indicates modeling technique used to refined estimates

APPENDIX I: Ravena Human Health Risk Assessment

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LIST OF EXHIBITS

I-1 Introduction

Under section 112(f)(2)(A) of the Clean Air Act (CAA), the U.S. Environmental Protection Agency (EPA) is directed to assess the residual risk from continued emissions of hazardous air pollutants (HAPs) from source categories regulated under Section 112(d) of the CAA. If existing maximum achievable control technology (MACT) standards do not provide an "ample margin of safety" for human health, EPA will promulgate additional emission standards for a source category. Among other aspects of human health that EPA must consider is the potential for exposures to HAPs via non-inhalation pathways and the risks associated with such exposures.

In Appendix C to EPA's report to SAB, a screening methodology is described that uses the Total Risk Integrated Methodology (TRIM), an overall risk assessment modeling system developed by OAQPS. The results are then used to support residual risk decisions for RTR II categories. The TRIM-based methodology includes a screening evaluation that determines whether a source warrants further consideration and then proceeds, as warranted, to more refined, site-specific assessment involving TRIM. To illustrate the application of the TRIM-based methodology for refined, site-specific risk assessment, ICF has conducted a case study of emissions of PB-HAPs from one source in the portland cement source category.

This appendix presents the approach and the results of this case study. The case study evaluates maximum individual ingestion exposures to mercury and dioxins and estimated resultant human cancer risks and chronic non-cancer hazards. We targeted a facility that had geographic characteristics amenable to two basic exposure scenarios (farmer and recreational angler) chosen to illustrate the application of the methodology. The Ravena LaFarge Portland Cement Facility in Ravena, New York (NY) was selected for the case study evaluation. The Ravena facility is close to populated areas, several fishable water bodies, and potential farmland. Although this facility may not necessarily represent the highest multipathway risk of all facilities in the source category, it is useful as a demonstration of the intended approach to be taken when the emissions from a source of interest for RTR *de minimis* levels and require refined risk assessment. In turn, this demonstration is expected to be useful for soliciting feedback on a range of risk assessment-related issues pertaining to EPA's RTR II program.

This document is divided several sections that describe the problem formulation, methodology, and results for this case study evaluation. Section I-2 describes the conceptual model we developed for examining potential exposure and risk. Sections I-3, I-4, and I-5 describes the methods and inputs for the TRIM.FaTE fate and transport modeling, ingestion exposure dose estimation, and dose-response values and risk characterization calculations conducted for this assessment. The results of the case study and a limited discussion of results are presented in Section I-6. References cited in this appendix are listed in Section I-7.

I-2 Conceptual Model for Potential Exposures/Risks

I-2.1 Selection of HAPs for this Analysis

To evaluate non-inhalation exposures and risks for RTR, the EPA has developed a list of 14 persistent, bioaccumulative hazardous air pollutants (PB-HAPs) for which the risks from non-inhalation exposure pathways may be relevant. OAQPS developed the list based on a two-step process taking into account the following:

- their presence on three existing EPA lists of persistent, bioaccumulative, and toxic substances, and

- a semi-quantitative ranking of toxicity and bioaccumulation potential of the entire list of HAPs.

The list's development and utility in hazard identification for multipathway risk assessment are further explained in Chapter 14 and Appendix D of Volume I of EPA's Air Toxics Risk Assessment (ATRA) Reference Library (EPA 2004a). As described in the RTR Multipathway Screening TSD (see Appendix G of this report), the first step in evaluating non-inhalation exposures and risks is to compare HAPs emitted by a facility of interest to the chemicals on this PB-HAP list. An initial screen is then conducted by comparing the facility-specific total emissions (in ton per year or TPY) for a given PB-HAP to the *de minimis* emission rate calculated using the RTR screening scenario. At each facility, PB-HAPs for which the total emissions exceed the *de minimis* emission rate for that chemical (or chemical group) are not screened out and may be subjected to further analysis.

Facilities in the portland cement manufacturing source category emit a variety of PB-HAPs, including metals (lead, cadmium, and mercury) and organic compounds (particulate organic matter and chlorinated dibenzo-p-dioxins and -furans, or "dioxins"). For each facility in this source category, total emissions for each PB-HAP were compared to *de minimis* levels to initially screen for the potential for non-inhalation exposures and risks. The results of this screening are described in EPA's main report (see Section 3.2 of that report).

Although emissions of every PB-HAP on EPA's list are not reported for every facility in this source category, more than half of the facilities report mercury emissions. Also, based on data from individual facilities and knowledge of the portland cement manufacturing process, every facility is assumed to emit dioxins. Both mercury and dioxins are presumed to be emitted in relatively large quantities from facilities in this source category. Given the potential for exposure via non-inhalation pathways to these two PB-HAPs and their relatively high emissions reported for portland cement facilities, both mercury and dioxins are expected to be chemicals of concern for the non-inhalation human health risk assessment of this source category for RTR. Consequently, mercury and dioxin were selected as the chemicals for this case study.

I-2.2 Selection of Relevant Exposure Pathways and Approach to Exposure Assessment

A multipathway exposure assessment of air toxics typically focuses on two categories of ingestion pathways: (1) incidental ingestion of contaminated environmental media and (2) consumption of contaminated food chain constituents. The range of exposure pathways included in multipathway air toxics assessments is described in Chapters 14 and 15 of EPA's ATRA Reference Library, Volume 1 (EPA 2004 a,b). For mercury and dioxins, exposures via the consumption of farm produce, farm animals and animal products, and fish are the primary concerns.

Mercury compounds that industrial processes emit to the air are typically a mixture of elemental and divalent mercury species and are not particularly bioaccumulative. However, once deposited to soil and surface waters, divalent mercury can be converted to methylmercury and other organic mercury forms that are highly bioaccumulative. Methylation of mercury can occur in the aquatic environment in particular, where it can enter the aquatic food chain. Elevated levels of methylmercury have been measured in freshwater and saltwater fish, especially fish species at higher trophic levels of aquatic food chains, which can accumulate mercury by consuming small fish (i.e., "biomagnification"). As a result, the consumption of fish that contain methylmercury represents the primary human exposure pathway of concern for mercury.

People can also be exposed to divalent mercury through FFC exposures and incidental ingestion of surface soil, although in general these exposures receive less attention and are typically of less concern relative to the fish-methyl mercury pathway due to high levels of methyl mercury present in the environment in some locations as a result of historical contamination.

Dioxins do not readily degrade in biotic or abiotic environmental media, and their chemical characteristics can promote bioaccumulation in aquatic and terrestrial food chains. In addition to accumulating in fish, dioxins emitted to air can enter the human food chain via the deposition to soil, surface water, and plant surfaces. Then, these chemicals accumulate in plants (including some produce) and animals and animal products (e.g., dairy products, eggs, and fish) that people consume. The consumption of fish and other food thus represents a non-inhalation exposure pathway of concern for dioxins as well. People can also be exposed to dioxins through the incidental ingestion of surface soil subject to deposition of dioxins, although this pathway is generally less significant than pathways involving the food chain.

In a residual risk assessment, a key risk metric of interest with respect to informing policy decisions is the risk to the individual most exposed (i.e., the "maximum individual risk," or MIR). For an inhalation risk assessment, the MIR can be approximated (taking into account a range of assumptions) using modeled long-term average air concentrations associated with a source and information on where people reside. For an evaluation of non-inhalation exposures, however, estimating the risk to the "most exposed" individual can be more difficult because chemical concentrations in environmental media to which people are exposed *and* individual exposure patterns associated with ingestion can vary greatly depending on location, timing, and other factors. For example, people can be exposed to chemicals that accumulate in the FFC by consuming a variety of fruits and vegetables, each of which may or may not be grown in the vicinity of the source. The amount of each type of produce consumed can vary widely among the individuals in a population, as can the fraction of each type of produce that is actually impacted by emissions from a source.

To simplify the exposure and risk analysis of a multipathway air toxics risk assessment a scenario approach can be employed. This approach, described in more detail in Chapter 15 of ATRA (EPA 2004b), entails evaluating a combination of exposure pathways by which an individual might be exposed to PB-HAPs (i.e., an "exposure scenario"). The scenario approach provides a systematic method for evaluating the relative importance of exposure pathways (e.g., consumption of farm food products vs. consumption of fish) that are of potential concern for different chemicals and locations. Only scenarios that are plausible for the situation of interest are typically evaluated, and the assessment usually focuses on those scenarios that are assumed *a priori* to lead to the highest individual exposure and risks. Risk metrics such as incremental lifetime cancer risk and chronic non-cancer hazard quotient are calculated as appropriate for each scenario, and information regarding the likelihood of a specific exposure scenario actually occurring can be used to develop estimates of uncertainty for each scenario and the variations thereof.

For this RTR case study, exposure estimates and risks were calculated for two basic scenarios:

- A **farmer scenario**, involving an individual living on a farm homestead in the vicinity of the source who (a) consumes produce grown on and meat and animal products raised on the farm, and (b) incidentally ingests surface soil at the location of the farm homestead; and

- A **recreational angler scenario**, involving an individual who regularly consumes fish caught in freshwater lakes in the vicinity of the source of interest.

Variations of these two scenarios were evaluated using different assumptions about location of the farm homestead or the water body, whether an individual is exposed via both scenarios, the age of the individual exposed (for non-cancer hazards), the assumed ingestion rate of each food type, and other factors. In addition, exposure estimates and risks to **infants via breastfeeding** were evaluated, with the assumption that the nursing mother was exposed to chemicals via one or both of the two basic scenarios listed above.

Exhibit I.2-1 presents the conceptual exposure model for the farmer scenario. The arrows represent the movement of chemical of concern through the environment and farm food chain. In this exposure scenario, the hypothetical receptors consume produce, meat, and animal products, and incidentally ingest soil. Exhibit I.2-2 presents the conceptual exposure model for the angler scenario. The hypothetical receptor, a recreational angler, consumes fish from a contaminated water body.

Exhibit I.2-1. Conceptual Exposure Model for Farmer Scenario

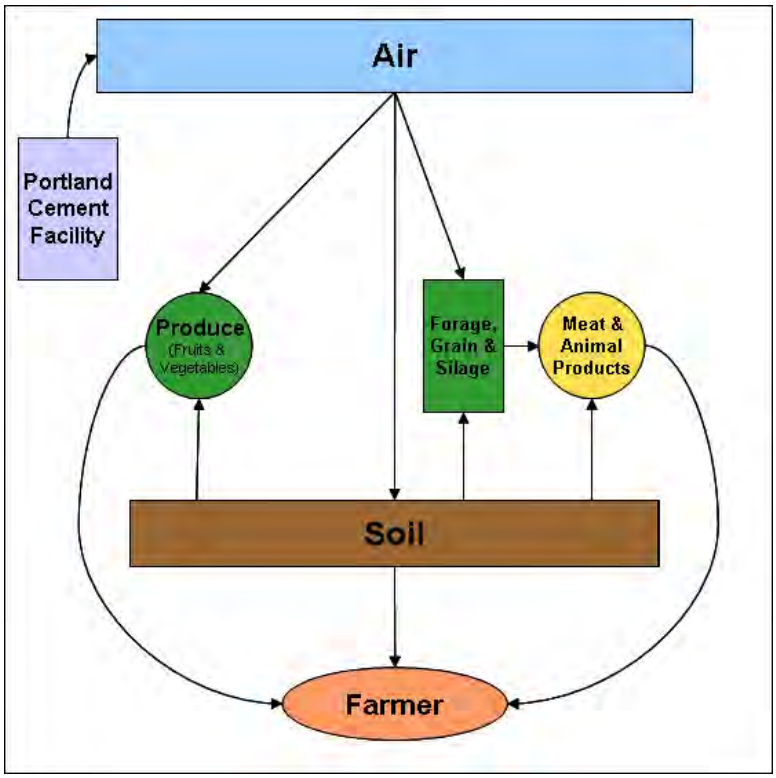
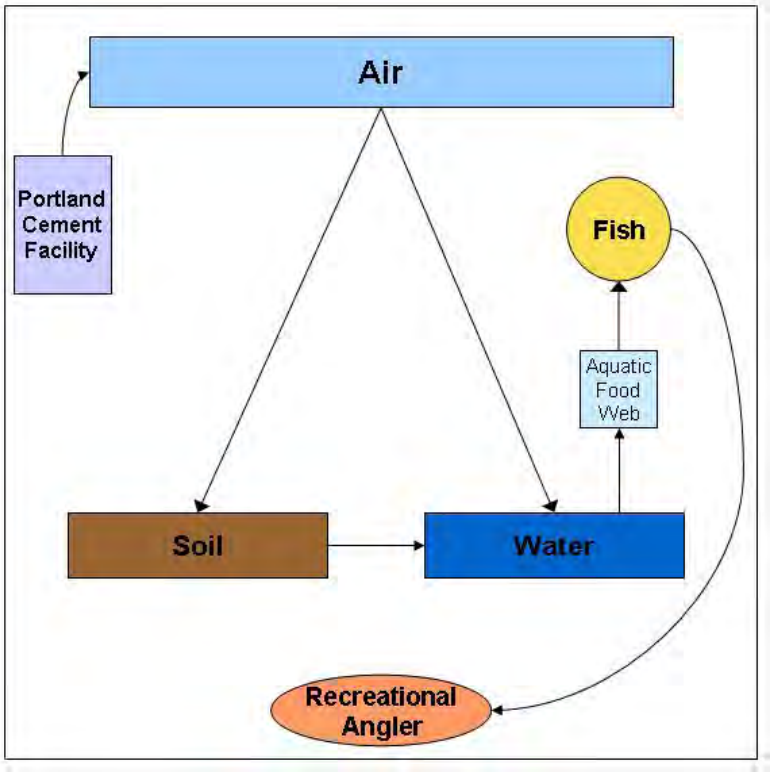


Exhibit I.2-2. Fish Consumption Exposure Pathway



I-2.3 Overview of Modeling Approach

Fate and transport modeling of PB-HAPs was completed using the Fate, Transport, and Ecological Exposure Module (TRIM.FaTE) of EPA's Total Risk Integrated Methodology. TRIM.FaTE is a fully coupled multimedia model that estimates the flow of pollutants through time among environmental compartments including air, soil, water, fish, and animals. The results of TRIM.FaTE modeling are chemical concentrations in abiotic environmental media (air, soil, surface water, and sediment) and in fish. TRIM.FaTE is essentially a spatially discrete, multi-compartment box model that partitions chemical mass among phases and between environmental compartments expressed (in part) using fugacity principles. For detailed information on TRIM.FaTE, refer to EPA's TRIM website (http://www.epa.gov/ttn/fera/trim_gen.html).

Ingestion exposures were calculated for the two exposure scenarios of interest using Multimedia Ingestion Risk Calculator (MIRC), and exposure and risk model that uses ingestion exposure algorithms similar to those found in the Human Health Risk Assessment Protocol (HHRAP) (EPA 2005). Chemical concentrations in intermediate farm food types (e.g., produce, animal products) were calculated using biotransfer factors to estimate the food chemical concentration based on the air and soil concentrations and deposition rates from TRIM.FaTE. Attachment C-2 of the main report provides details of the approach and methods used to calculate ingestion exposures. Individual lifetime cancer risks for dioxins and chronic non-cancer hazard quotients for dioxins, methylmercury, and divalent mercury were then calculated using oral cancer slope factors and ingestion reference doses (RfDs).

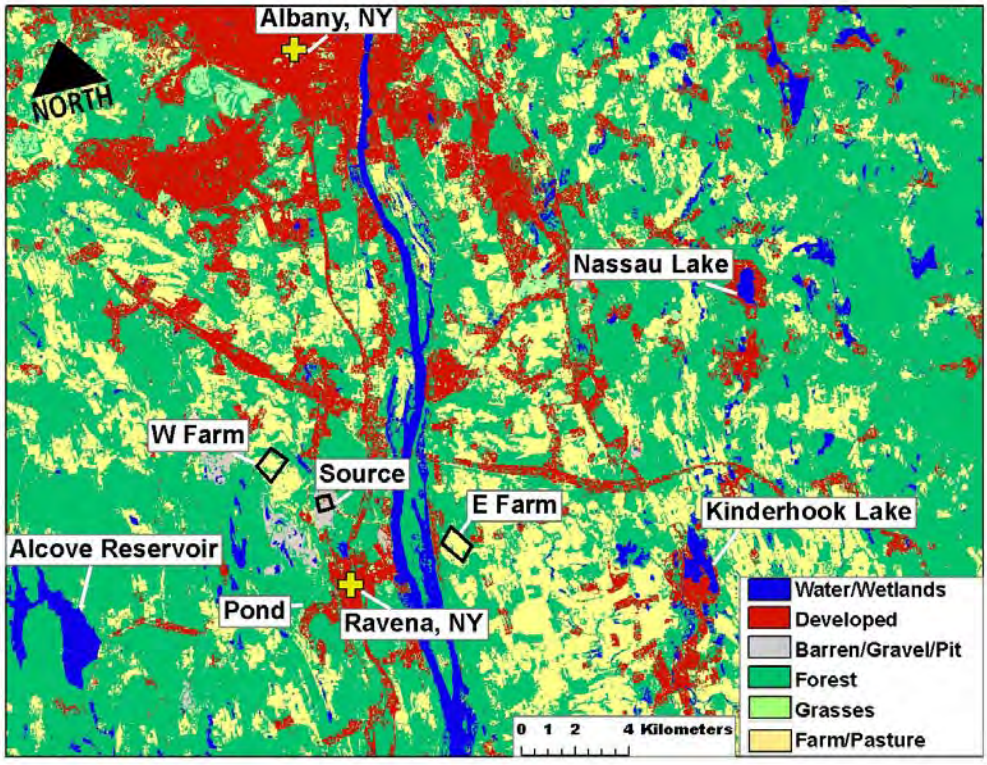
I-2.4 Selection of Facility for Analysis

To narrow the scope of this SAB case study and enable a more in-depth evaluation, we focused on one portland cement facility. We first identified portland cement facilities that had high emissions for both mercury and dioxins, assuming that higher emissions of the chemicals would lead to higher human exposures. Of these facilities, we looked for one with geographic characteristics amenable to the two basic exposure scenarios (farmer and recreational angler). Minimum requirements included:

- Close proximity to a freshwater lake of reasonable size,[1] and
- Proximity to land used to support a range of agricultural activities (crops and animals).

The Ravena Lafarge portland cement facility (hereafter referred to as the Ravena facility) in Ravena, New York (NY) meets these criteria and was selected for evaluation in this case study (see Exhibit I.2-3). The Ravena facility is close to populated areas, several fishable water bodies, and potential farmland. Although this facility may not necessarily represent the highest multipathway risk of all 91 portland cement facilities, it is useful for demonstrating the methods of the refined multipathway HHRA (i.e., what to do when the emissions from a source category exceed the *de minimis* levels), and this is expected to be useful for soliciting feedback on a range of risk assessment-related issues pertaining to EPA's RTR II program.

Exhibit I.2-3. Location of the Ravena Facility



[1] The goal of the case study was to examine incremental exposure from facility emissions. A very large lake would dilute the chemical. However, a very small pond would not sustain a fish population large enough to support regular consumption of fish by a local angler.

The facility is located approximately 12 miles south of Albany, NY, in the southeastern portion of Albany County (U.S. Census Bureau 2000; population 294,570). The population of Ravena, NY, the nearest town (located just east of the facility), is 3,369. Nearby counties include Renesselar, Greene, and Columbia, all in New York. According to the 2002 U.S. Department of Agriculture (USDA) Census of Agriculture, these four counties support livestock and crops grown for human and animal consumption (USDA 2002).

For the purpose of the Ravena HHRA, only dioxin and mercury emissions were evaluated. A scenario layout for the Ravena area was created to use in TRIM.FaTE so that all relevant ingestion pathways could be modeled. Both divalent mercury and dioxins can accumulate in the farm food chain, so the scenario layout includes two farm homesteads, on the east and west sides of the facility. The farm homesteads were located in areas where actual land use is agricultural.

Methylmercury and dioxins bioaccumulate in fish, so four freshwater water bodies were included in the Ravena layout to estimate exposure for the angler scenario. The Ravena area encompasses many other water bodies including the Hudson River, but for the purposes of TRIM.FaTE modeling, fish populations in only three lakes and one pond were modeled. The lakes and pond represent a range of sizes and locations that the Ravena facility emissions could impact. Alcove Reservoir is 7 miles west of the Ravena facility and supplies drinking water to the city of Albany. Kinderhook Lake (8 miles southeast of the facility) and Nassau Lake (11 miles northeast) allow recreational fishing. All three of these lakes are large enough to support large fish populations and were modeled in TRIM.FaTE. A small pond is located 2 miles southwest of the facility. The pond was also modeled, although there is significant uncertainty whether it is large enough to support a fishable aquatic ecosystem (discussed in more detail in Section I-6.4). The Ravena facility is within 2 miles of the Hudson River, which was modeled as a water body in this case study. A fish population was not modeled in the river.[2]

For this case study, we modeled dioxin emission rates based on mean and 95th percentile upper confidence limit emission factors based on the clinker production of the facility. (See Appendix F to EPA's report to SAB.) The divalent and elemental mercury emissions modeled were those reported in the 2002 NEI, and transformation of mercury (e.g., divalent mercury into methylmercury) was included in the model (EPA 2002).

Exposure factors used in this case study are described in Section I-4 of this appendix.

I-3 Fate and Transport Modeling (TRIM.FaTE)

This section describes the TRIM.FaTE modeling conducted for this case study risk assessment. Most of the material presented here describes the assumptions and data sources used to set TRIM.FaTE inputs and settings related to meteorological inputs used by the model (Section I-3.2), the spatial aspects of the modeled region (Section I-3.3), characteristics of abiotic environmental compartments (Section I-3.5), and plants (Section I-3.6) included in the scenario, and the aquatic ecosystems set up in each water body of interest (Section I-3.7). In Section I-3.8, a summary of the distribution of mass among the modeled compartments in the scenario at the end of the simulation period is presented to provide an overview of the model results (more

[2] Incremental concentrations of dioxins and mercury in the river (i.e., those resulting from Ravena facility emissions) are expected to be significantly lower than incremental concentrations in nearby lakes; the flow of the river will lead to greater dilution. In addition, the emissions from the Ravena facility are only a small part of the total emissions that affect the chemical concentrations in the river.

detailed estimated concentrations are presented with other assessment results later in this appendix).

I-3.1 Source Characterization

For this case study, TRIM.FaTE was used to model emissions from the Ravena facility of total dioxins (modeled using 2,3,7,8-TCDD as a surrogate for total emissions calculated as dioxin TEQ) and mercury (modeled as the appropriate mix of divalent and elemental). Transformation of mercury to methylmercury was also modeled (thus, three separate mercury species were modeled in TRIM.FaTE). The modeling scenario duration was 50 years, and emissions of both mercury and dioxin were assumed to be constant over the course of the simulation.[3] TRIM.FaTE was used to estimate chemical concentrations in air, soil, and selected surface water bodies (and their corresponding benthic sediment layer), as well as components of a representative aquatic ecosystem in each water body of interest for the risk assessment. Chemical concentrations were estimated by the model on a bihourly basis for the scenario duration and used to calculate annual average concentrations.

Estimated mercury and dioxin emissions to air from the Ravena facility are presented in Exhibit I.3-1. All mercury and dioxin emissions were modeled as coming from the main stack, at a height of 350 feet to match the reported stack height in NEI. Details regarding emission estimates are presented in a separate appendix to this report. Despite the fact that divalent and elemental mercury emissions for the Ravena facility did not exceed the *de minimis* levels, they were still modeled for the case study to demonstrate the effect of applying site-specific parameters The modeled divalent mercury emission rate for Ravena is approximately 70% of the *de minimis* level.

Exhibit I.3-1. Emissions of Dioxins and Mercury from the Lafarge Facility in Ravena, NY, and Screening Results

PB-HAP		Emissions (tons per year)	Screening Results
Dioxins/Furans [a]	95th percentile upper confidence limit of estimated emission factor	3.28E-06	Exceeds *de minimis* Level [c]
	Estimated mean emission factor	1.34E-06	Exceeds *de minimis* Level [c]
Mercury – Divalent [b]		0.05625	Screens out [c]
Mercury – Elemental [b]		0.016875	Screens out [c]

[a] Emissions estimated based on tons of clinker produced using dioxin emission factors. Details about this estimation are recorded in Appendix F.
[b] Emissions reported in 2002 National Emissions Inventory (NEI) (EPA 2002).
[c] *De minimis* levels for 2,3,7,8-TCDD TEQ and divalent mercury are 3.18E-08 and 1.64E-01, respectively. The *de minimis* level for elemental mercury is larger than and elemental mercury emission rate found in NEI; calculations of *de minimis* levels are further described in Appendix C.

I-3.2 Relevant Meteorological Data

TRIM.FaTE uses several meteorological inputs to determine chemical transfers among the air compartments in a scenario via advective transport (i.e., wind-driven physical movement through the atmosphere) and from air to underlying soil or water surfaces via deposition

[3] Although actual emissions from portland cement facilities may fluctuate with time due to process characteristics, start-up/shut-down operations, and other factors, modeling the emissions as constant was assumed to be appropriate for estimating long-term chemical concentrations.

transfers. These processes determine the long-term spatial patterns of chemical distribution within the scenario, and modeled concentrations are highly sensitive to the meteorological inputs used in TRIM.FaTE. Consequently, an initial step in developing the TRIM.FaTE application for this case study was to collect meteorological data for the expected modeling region (i.e., the area near Ravena, NY). The long-term trends in these data were then used to inform the development of the modeling layout that is the basis of this scenario. To maintain consistency with the development of the TRIM.FaTE application, we present in this section of the appendix the specifications of the meteorological data and a summary of the long-term temporal trends.

The meteorological inputs TRIM.FaTE requires include wind speed, wind direction, precipitation, ambient air temperature, and mixing height. A suitable data set was selected based on how closely the data are assumed to represent typical long-term conditions near the modeled source, data availability, and completeness of the data set. For this assessment, hourly surface meteorological data from the National Oceanic and Atmospheric Administration's National Climatic Data Center (NCDC) Integrated Surface Hourly (ISH) Database were obtained (NOAA 2001). The ISH Database contains more than 20,000 stations and is quality controlled, and so was judged to be a reliable source of meteorological data. Using this database, the closest meteorological station to the Ravena facility is approximately 30 km north at the international airport in Albany, NY. This surface meteorological station also hosts a radiosonde site, which results in collocated surface and upper-air meteorological data (the upper-air data set contains information used to determine mixing height) (NOAA 2007).

Three consecutive years of data (for 2001–2003) were readily available from this data set. To facilitate the use of these data for a longer application, one of the years (2002) was repeated (to create a 4-year dataset with the appropriate number of days to account for leap years) over the duration of the simulation to create a dataset from 2001–2003. Exhibit I.3-2 shows the 30-year climate normals for annual total precipitation and annual average daily temperature compared with the statistics for the 2001-2004 data used in this study and the overall average of the data set used for modeling. The three years of observed meteorological data used for this study are warmer and drier than the 1971-2000 NCDC 30-year climate normals for the Albany meteorological station (NOAA 2003). The parenthetical numbers indicate percent deviation of the 2001-2003 values from the 30-year normal values.

Exhibit I.3-2. Comparison of Historical and Modeled Temperature and Precipitation

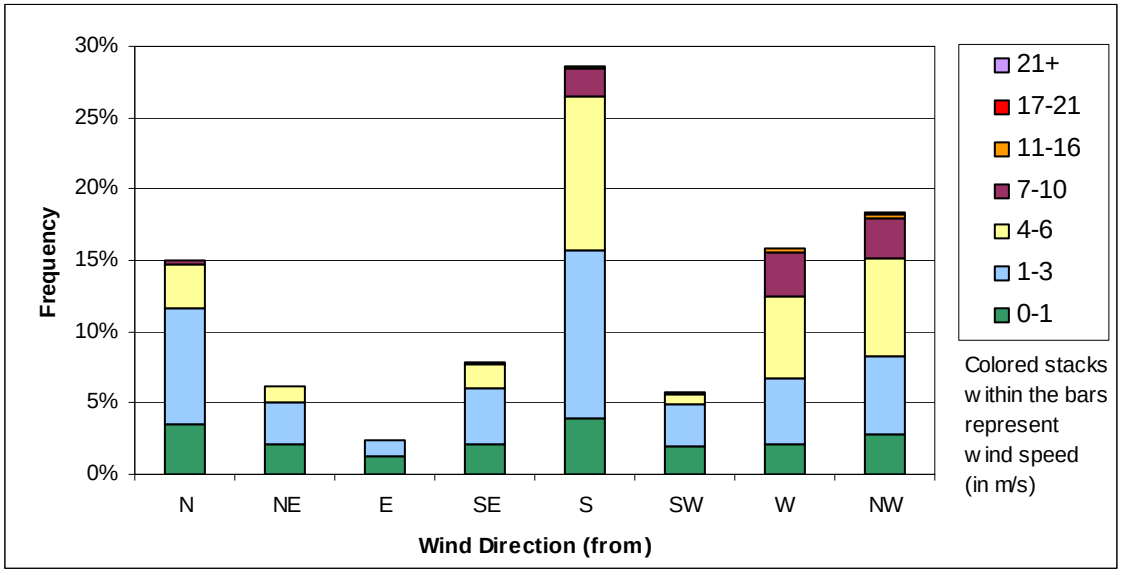
Statistic	Albany, NY: Historical 30-year Normal (1971-2000) [a]	Data Used for This Assessment			
		2001	2002	2003	Overall [b]
Annual average of daily average temperature (°C)	8.6	10	9.9	8.7	9.6
Deviation from normal temperature (°C)		+1.4 (+16%)	+1.3 (+15%)	+0.1 (+1%)	+1.0 (+12%)
Annual precipitation amount (mm)	980	570	862	919	803
Deviation from normal annual precipitation (mm)		-410 (-42%)	-118 (-18%)	-61 (-6%)	177 (-18%)

[a] Historical temperature and precipitation data from NOAA's NCDC (2003).
[b] Overall includes the 2002 year data weighted twice as much as other years because they are repeated to create a 4-year meteorological time series. This 4-year series was repeated to create the full 50-year meteorological data set.

In the natural environment, rainfall amounts that are less than climatologically typical quantities in the Albany area can have the effect of decreasing modeled wet deposition. Decreased rainfall may also increase chemical concentrations in water bodies by reducing their volumes and flush rates, although the water bodies would also collect reduced amounts of chemicals from their tributaries. The relatively small deviations from normal (warmer and drier) intrinsic to the data used for modeling were assumed to be acceptable for this assessment.

Hourly average wind speed was reported as calm for about 20 percent of the time for the repeated 4 year period. When not calm, wind speeds across the repeated 4-year period are typically less than about 3 meter/second (m/s) 41 percent of the time. The wind direction is most often from the south (29 percent of the time), with 49 percent of observed wind direction split fairly evenly from among the north, northwest, and west. These wind direction preferences indicate that areas south, east, and especially north of the Ravena facility should experience the greatest dry deposition from facility's emissions. Exhibit I.3-3 shows the frequency distribution of wind directions and coincidental wind speeds for the repeated 4-year dataset. In general, the observed trends in wind direction and speed in the modeling data set are expected to represent the overall trends for the Ravena area (patterns were similar for all three years and correspond to general conditions for the mid-Atlantic region).

Exhibit I.3-3. Frequency Distribution of Wind Direction and Coincidental Wind Speed (2001-2003 Albany dataset, all time periods)



When precipitation occurs, wind speeds less than 3 m/s and wind direction from the south, north, and west still dominate the wind pattern. However, winds from the north occur slightly more often than from other directions during precipitation (27 percent of the time). During precipitation, these wind direction preferences indicate that areas north, east, and especially south of the Ravena facility should experience the greatest wet deposition from the facility's emissions (see Exhibit I.3-4).

Mixing height is used in calculating air concentrations and related processes. In addition, for time periods when the mixing height is less than the stack height modeled in TRIM.FaTE (i.e., 350 feet), chemical emissions from the source are transferred to an upper air layer and are not available for deposition to modeled soil and water surfaces (this situation occurs less than 2 percent of the time for the modeling data set used). About 70 percent of morning mixing height

values are less than 1,500 meters (m), and the morning frequency distribution decreases logarithmically with increasing height values (see Exhibit I.3-5). The afternoon mixing height values follow a Gaussian distribution with a median value of 1,646 m.

Exhibit I.3-4. Frequency Distribution of Wind Direction and Speed During Rain Events (2001-2003 Albany dataset, hours with precipitation only)

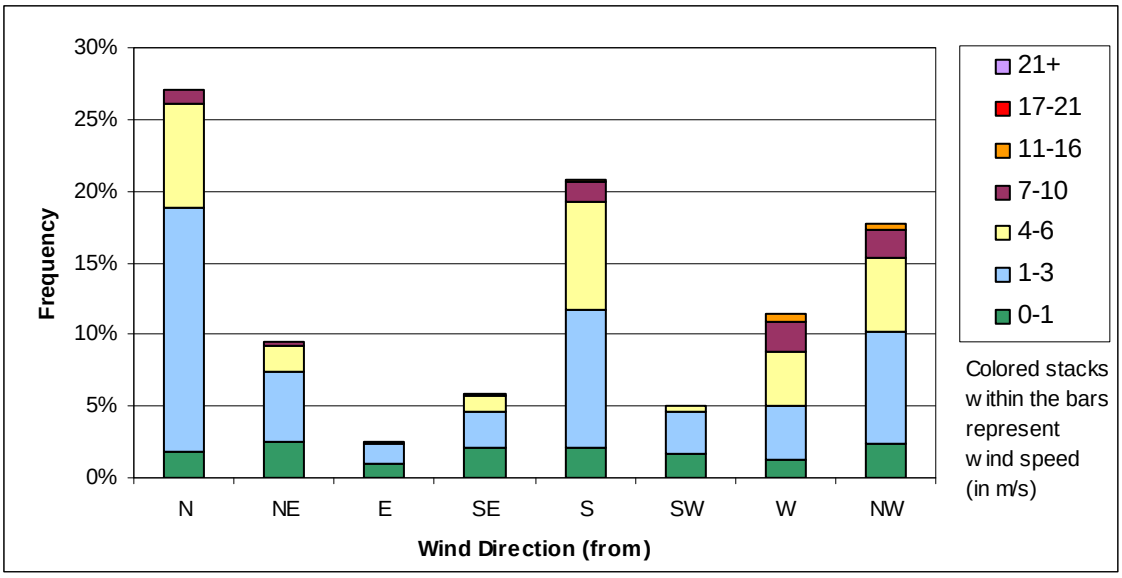
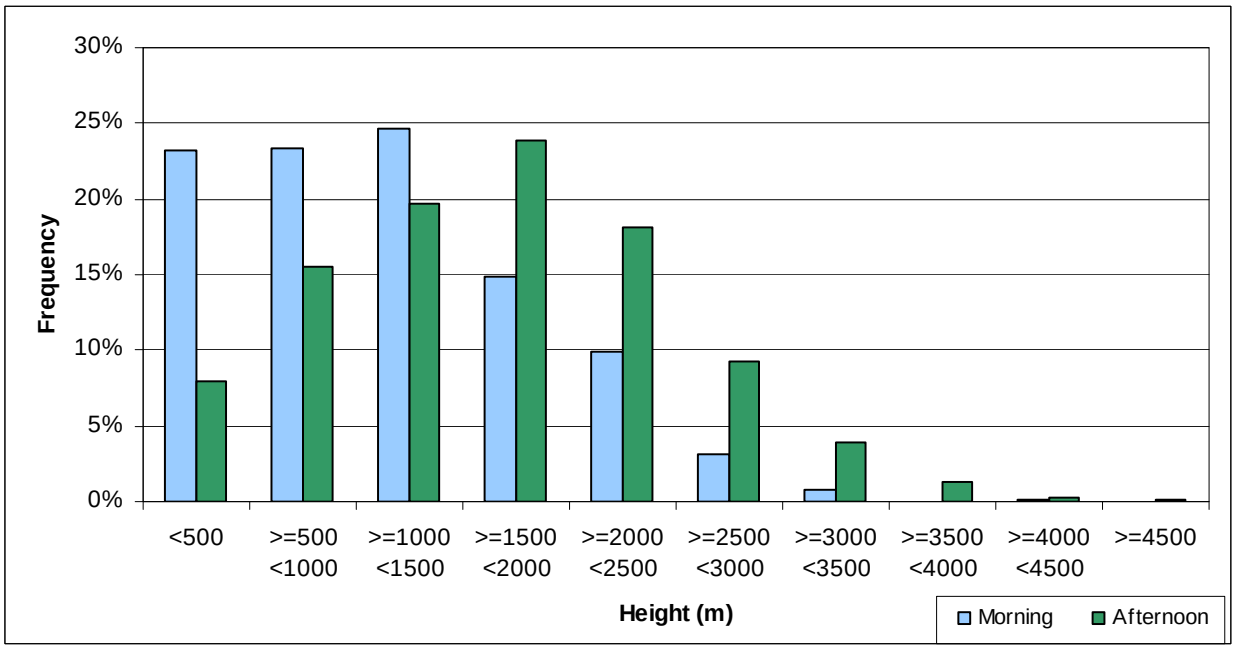


Exhibit I.3-5. Frequency Distribution of Calculated Morning/Afternoon Mixing Height (m)



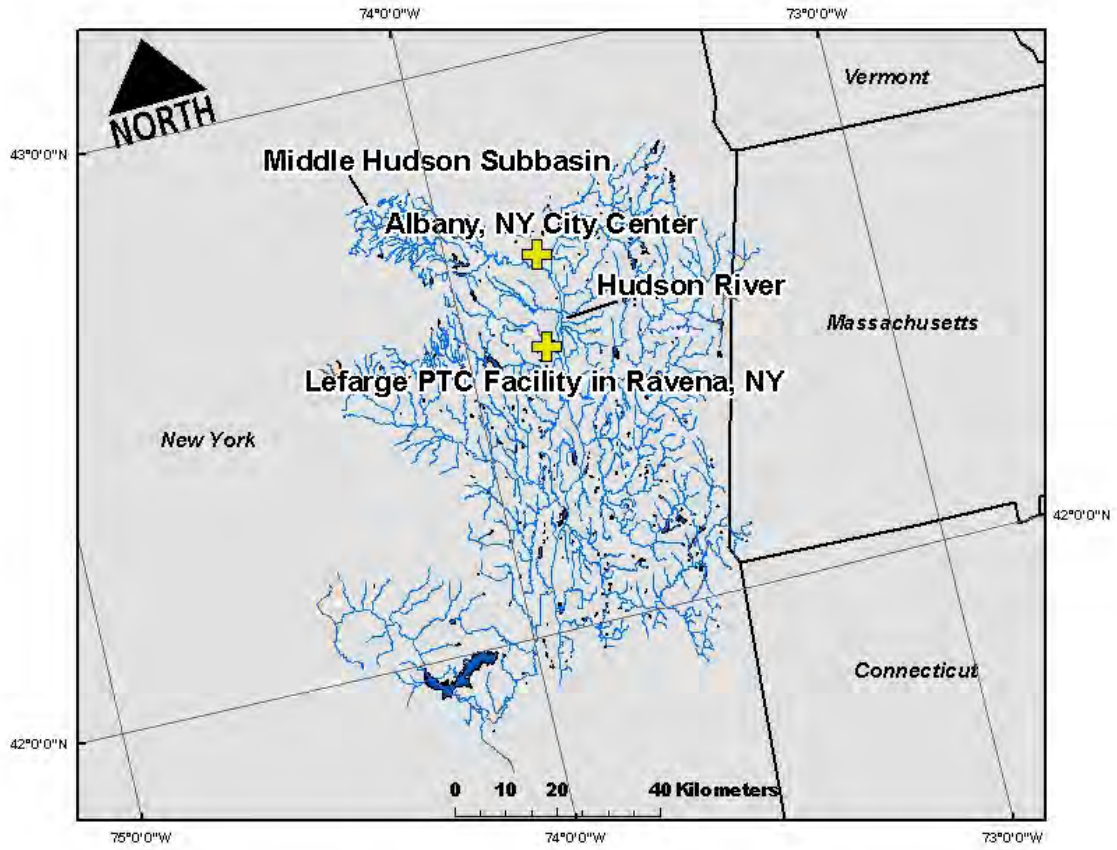
I-3.3 Extent and Dimensions of Modeled Environment

This section describes the environment for which media concentrations were estimated using TRIM.FaTE and the geographic characteristics of the modeled environment (e.g., layout of the modeled domain and geometry of the constituents included).

The design of the modeling layout was developed based primarily on physical/geographic characteristics of the watersheds in the area and land-use data for the region. This section provides a brief overview of the features present in the vicinity of the modeled facility.

As illustrated in Exhibit I.3-6, the Ravena facility lies within the Middle Hudson Subbasin (HUC-8 Code 02020006).[4] Rivers and streams in this subbasin drain to the Hudson River that flows from north to south through this basin. No major lakes dominate this region, although numerous reservoirs and lakes are located throughout the subbasin. Based on data from the U.S. Geological Survey (USGS), land use in the vicinity of the Ravena facility is classified as a mixture of forested land (with both deciduous and evergreen forests), land in agricultural use (for pasture and cropland), and commercial and residential uses (see Exhibit I.3-7). Land use becomes more urban proceeding northward from the facility toward the city of Albany, NY.

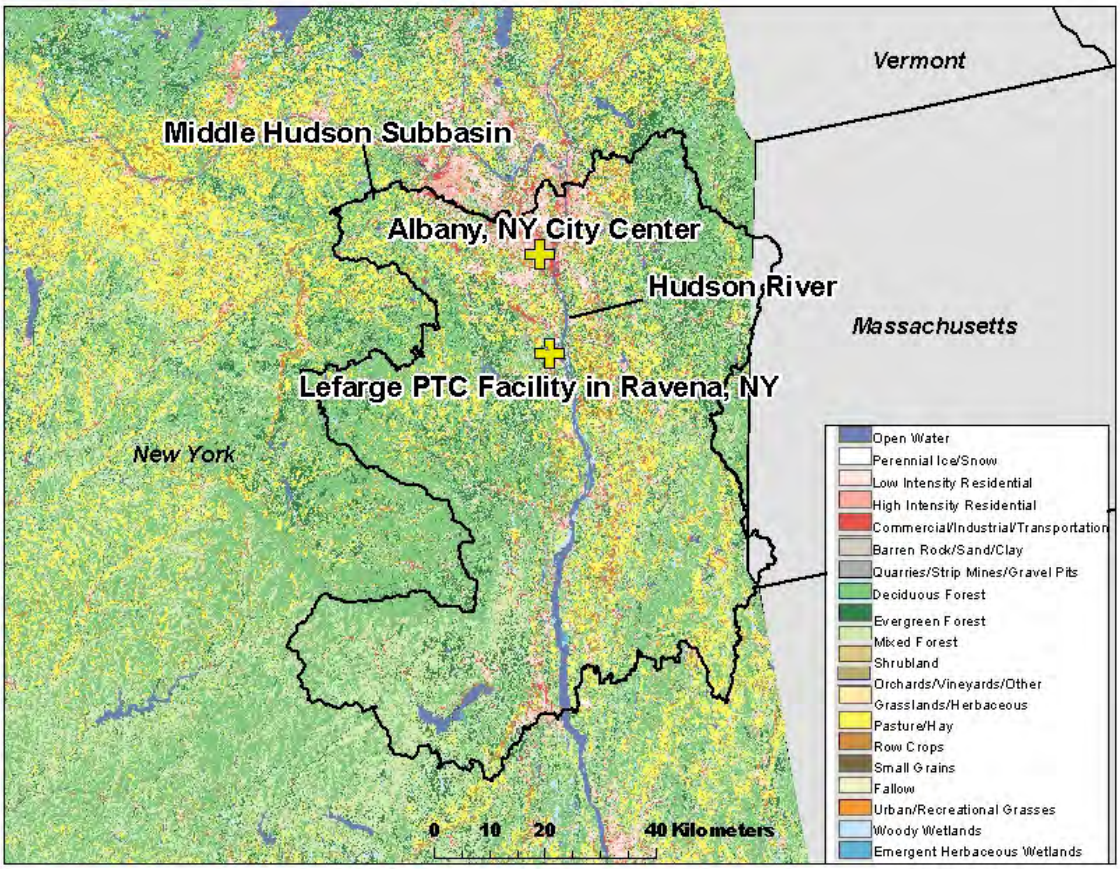
Exhibit I.3-6. Streams, Rivers, and Water Bodies of the Middle Hudson Sub-basin



[a] Data obtained from the US Geological Survey (USGS) National Hydrography Dataset (NHD) for the Middle Hudson Subbasin (USGS 2002b). These data are based on the content of USGS 1:100,000-scale data.

[4] This and the following maps describing the modeling spatial layout are in a projected coordinate system, which means that north is toward the upper-left of the page rather than the top of the page.

Exhibit I.3-7. Land Use in Region of the Ravena Facility



[a] Land-use data were obtained from the USGS Multi-Resolution Land Characteristics Consortium (MRLC) National Land Cover Dataset 1992 (USGS 1992). Data were derived from the early- to mid-1990s Landsat Thematic Mapper scans, have a spatial resolution of 30 m, and contain 21 landcover classifications (e.g., deciduous, evergreen, and mixed forest; urban/recreational grasses; pasture/hay; row crops; low- and high-intensity residential; and commercial/industrial/transportation).

I-3.4 TRIM.FaTE Parcel Design

The TRIM.FaTE surface parcel layout is the two-dimensional configuration of soil and water regions included in the modeled domain; this is overlain by the air parcel layout. These layouts provide the spatial reference for three-dimensional compartments that hold the modeled chemical mass.

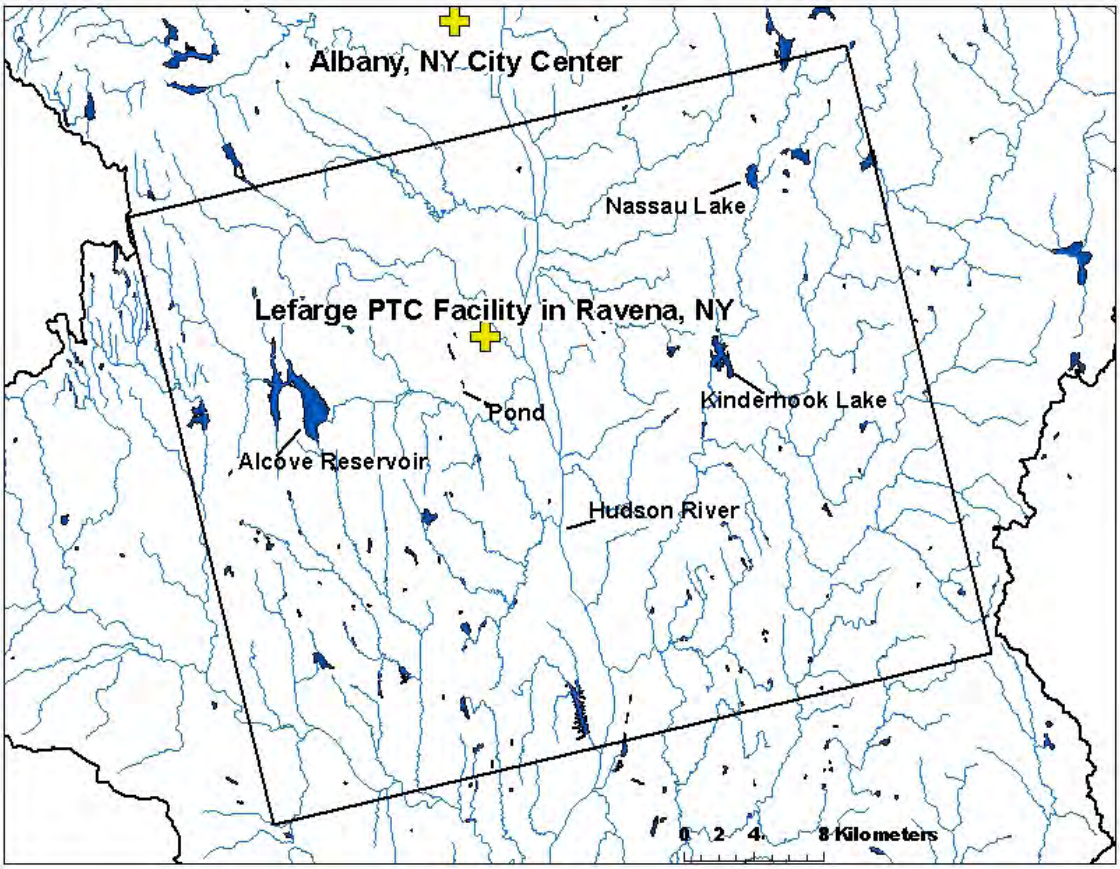
I-3.4.1 *Surface Parcel Layout*

The chief goal in designing the surface parcel layout was to accurately capture the watersheds surrounding the water bodies selected for modeling (i.e., those that contain fish people are assumed to eat) and the watersheds unique to the tributaries of the Hudson River that are in the vicinity of the facility. In pursuing this goal, parcel shapes were kept as simple as possible to reduce complexity in the layout and corresponding run time. As required by TRIM.FaTE, no parcel is fully contained within any other parcel; all parcels share at least one side or corner with another parcel.

The overall spatial extent of the modeling scenario is a 770 km^2 rectangle that captures several significant water bodies in the area and their watersheds (see Exhibit I.3-8). The area

approaches the metropolitan area of Albany to the north but generally stops short of the residential areas.

Exhibit I.3-8. Overall Modeling Region



The Ravena facility is represented within this layout as a source parcel with a surface area of 0.25 square kilometers (km^2), which is intended to roughly correspond to the size of the facility and source locations of mercury and dioxin emissions.[5] The location of the source parcel (centered at 42.501 °N and 73.815 °W) is about 1 km northwest of the facility coordinates included in NEI (i.e., 42.49 °N 73.81 °W). This modeled location was selected based on an examination of land-use imagery (see Exhibit I.3-9).

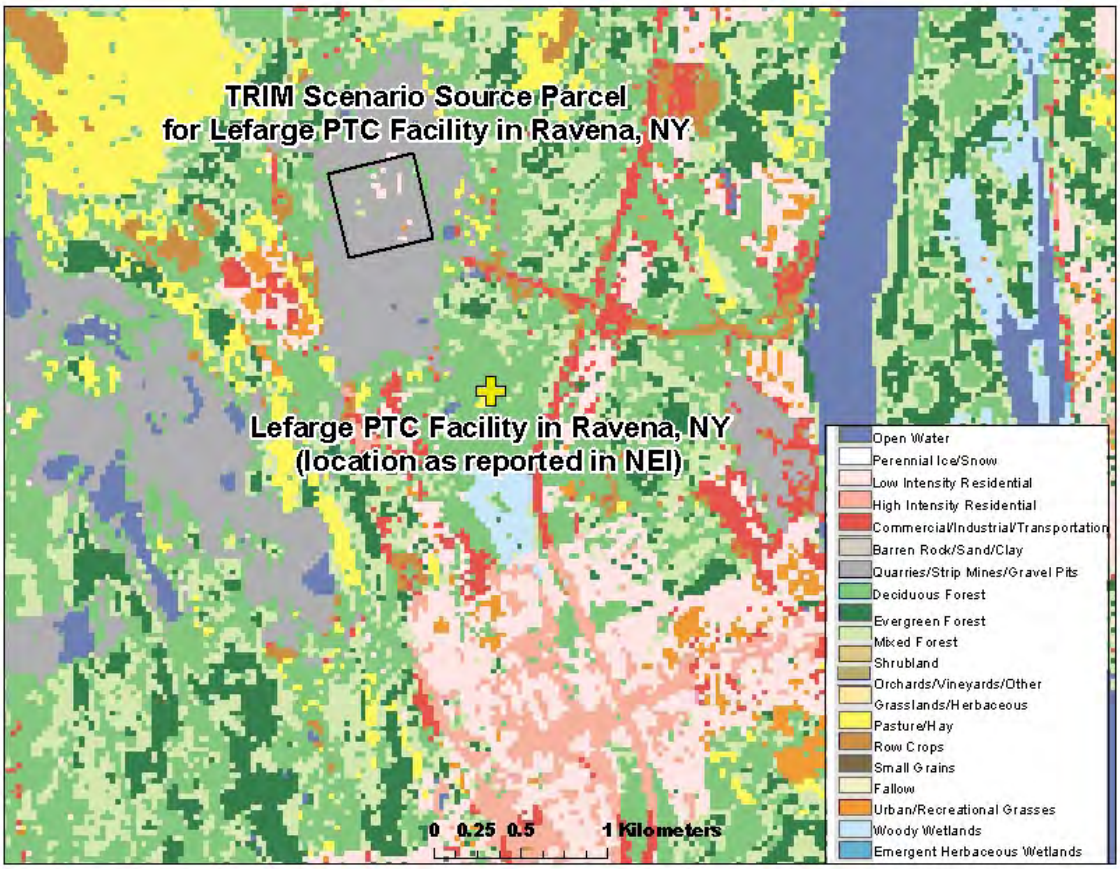
I-3.4.2 *Modeled Water Bodies*

Four water bodies were included in the modeled area for the purposes of modeling fish concentrations for the risk assessment: Alcove Reservoir to the west, the Kinderhook and Nassau Lake system to the east (these two lakes are connected), and an unnamed pond to the southwest of the facility. Exhibit I.3-8 indicates the locations and names of these water bodies within the scenario. The first three water bodies are large enough to support large populations of fish. The pond to the southwest is substantially smaller than these three, but it was judged to be large enough to support a fishable aquatic population if it were stocked regularly. Whether

[5] Although the actual facility size may vary from these dimensions, it is not critical that the size of the source parcel in a TRIM.FaTE scenario exactly represent the actual source size.

this small pond actually is fishable is not clear from the information collected for this assessment; this uncertainty is discussed in more detail in the results (Section I-6).

Exhibit I.3-9. Modeled and Reported Location of the Ravena Facility



[a] Land-use data from USGS 1992.

The Hudson River flows through the middle of the modeled domain; this water body was also included as a modeled constituent in the TRIM.FaTE scenario. However, no biota were included in the river compartment, and it was modeled primarily to "collect" water and sediment flowing from other regions (i.e., to simplify the set-up of the modeling layout).

The surface parcels created to represent these five water bodies in TRIM.FaTE were developed as simplified shapes with the goal of representing the actual water body surface area as accurately as possible (the water body surface area determines the amount of chemical mass deposited from air compartments in TRIM.FaTE). Exhibit I.3-10 indicates the actual surface areas of these water bodies compared to the surface areas of the parcels representing them. The surface areas of all water parcels are within 1 percent of water body surface areas reported in the USGS National Hydrography Dataset (NHD) (USGS 2002b).

Exhibit I.3-10. Water Bodies Included in the Modeled Region

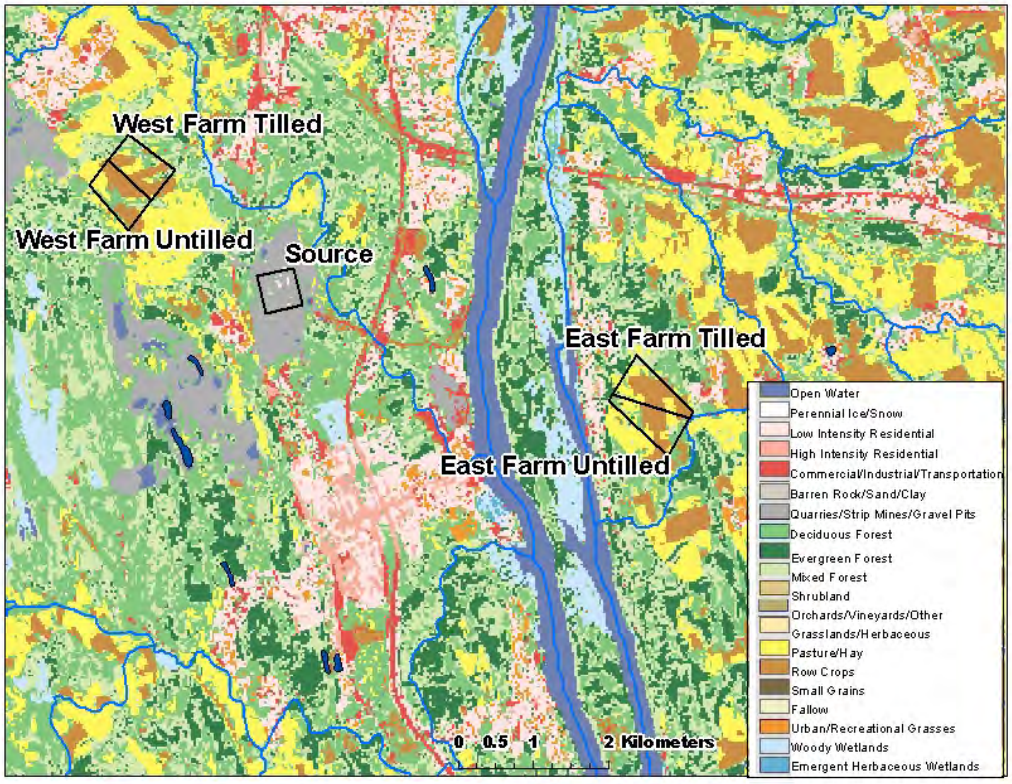
Water Body Name	Actual Surface Area (km^2, from NHD [a])	Modeled Surface Area of TRIM.FaTE Parcel (km^2)
Nassau Lake	0.654	0.654
Alcove Reservoir	5.511	5.514
Pond (unnamed)	0.020	0.020
Kinderhook Lake	1.341	1.342
Hudson River	22.318	22.329

[a] NHD = National Hydrography Dataset

I-3.4.3 *Modeled Agricultural Parcels*

Agricultural use regions also were included in the modeled domain, to estimate soil concentrations and other TRIM.FaTE outputs for use in calculating FFC exposures. Two farm regions were created: one 2.5 km northwest of the facility and the other 5 km south-southeast (see Exhibit I.3-11). The locations of these two regions were selected based on land-use patterns. The northwest location is the closest to the facility with a large area of predominantly row crops land-use designation. However, the wind pattern in this area, as measured from the Albany airport, is somewhat evenly split among westerly (i.e., blowing from the west), northerly, and southerly, and thus this location is generally upwind relative to the facility. A second farm parcel to the east was also included that is the closest large row crops land-use area that is approximately downwind from the facility. Each of these farm regions was roughly bisected to create two parcels (to accommodate modeling of tilled and untilled surface soil for use in estimating various farm food media concentrations).

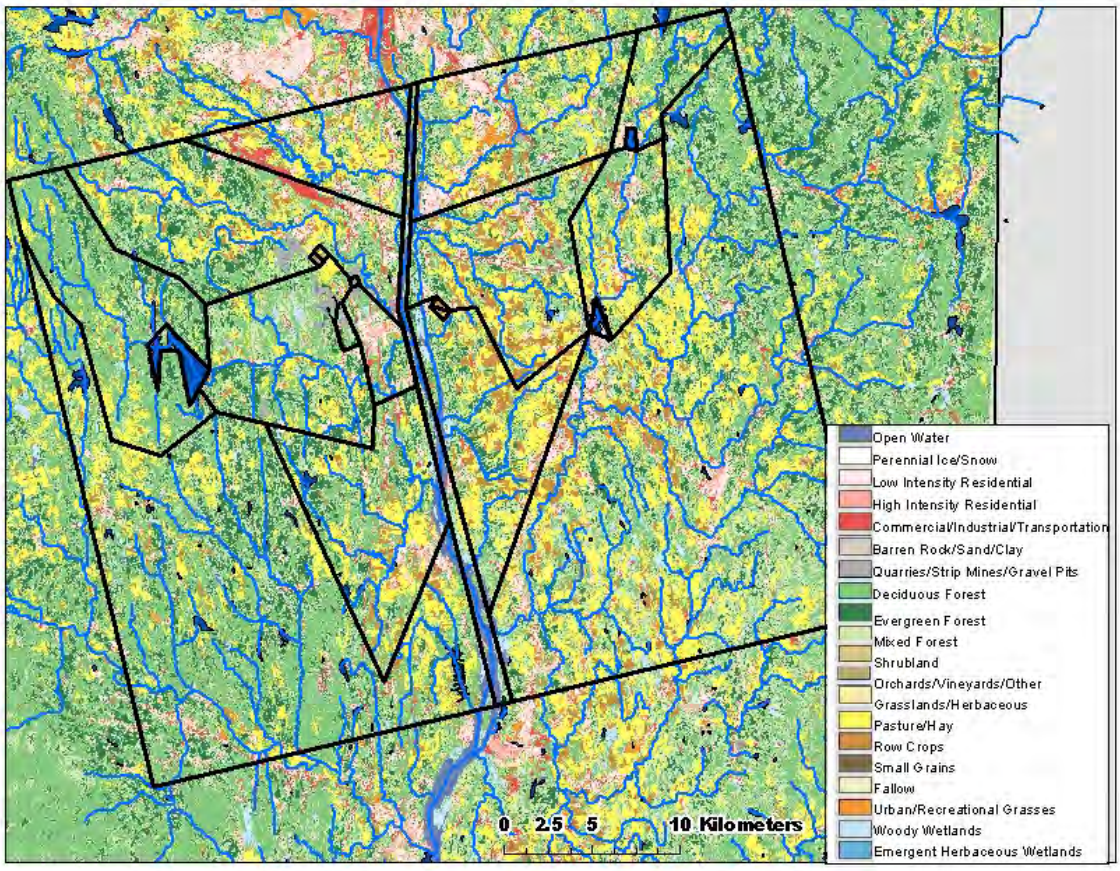
Exhibit I.3-11. Agricultural Parcels Included in the TRIM.FaTE Scenario



Watershed Parcels

The remaining area within the modeled domain was divided according to watershed boundaries, based on stream and topographic data from NHD. Exhibit I.3-12 presents the final surface parcel layout for the TRIM.FaTE scenario. For clarity, the parcel names are omitted; see Exhibit I.3-11 for parcel names.

Exhibit I.3-12. Surface Parcel Layout with Water Bodies and Land Use



I-3.4.4 *Air Parcel Layout*

Design of the air parcel layout for the TRIM.FaTE scenario was simpler than for the surface parcel layout because watersheds, terrain, and land use were not taken into account in the design. The overall spatial extent of the air parcel layout is identical to that of the surface parcel layout, and the surface source parcel is reproduced in the air parcel layout. For this assessment, the air parcel layout was designed as a radial grid (see Exhibit I.3-13), consistent with information presented in the EPA's TRIM.FaTE Users' Guide (EPA 2003). The layout consists of concentric squares around the source overlaid onto lines emanating radially in a regular 45-degree pattern. The distance from the side of a given square to the side of the next outward square increases with increasing distance from the source (i.e., 1 km, 5 km, and 13 km from the edge of the source parcel). Radial lines divide each concentric square such that eight parcels of equal area can be formed, although if that square intersects the boundaries of the layout then many of its parcels will be reduced in size. This radial layout minimizes the TRIM.FaTE bias for over-accumulation of mass along the axes of the grid (refer to the

TRIM.FaTE Users' Guide for additional discussion of this design (EPA 2003). Overall, 31 air parcels, including the source parcel, are included in the air parcel layout.
Exhibit I.3-14 overlays the air parcel and surface parcel layouts.

Exhibit I.3-13. Air Parcel Layout

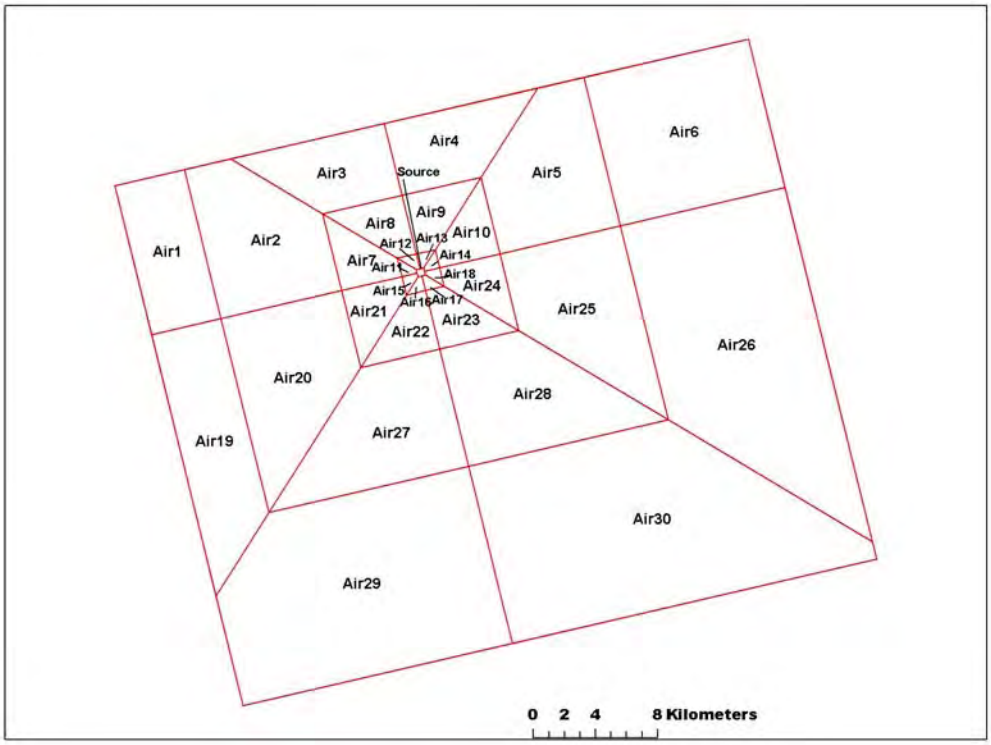
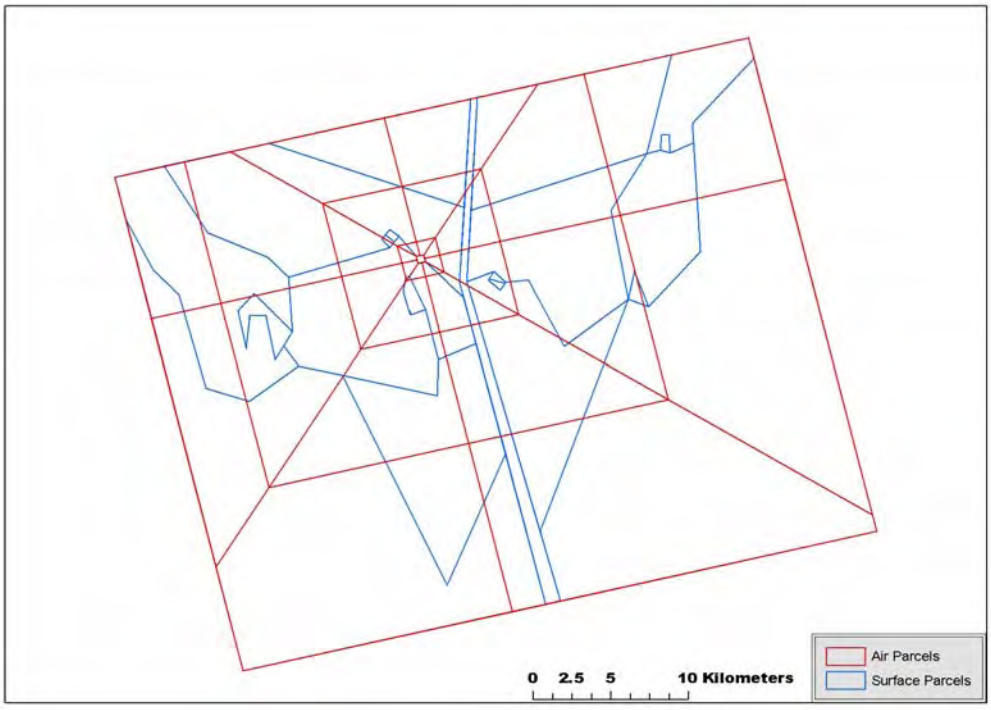


Exhibit I.3-14. Air and Surface Parcel Layouts (Overlay)



I-3.5 Abiotic Environment

TRIM.FaTE requires various abiotic environmental properties for each compartment that is included in the scenario (e.g., the depth of surface soil, soil porosity and water content, erosion and runoff rates from surface soil to water bodies, suspended sediment concentration, and others). Where site-specific data were readily available for this assessment they were used. Regional or national defaults were used in numerous instances, especially for those parameters that are not expected to influence chemical concentration dramatically. This section lists some of the important values used for this application. A complete list of TRIM.FaTE inputs for abiotic compartments is provided in Attachment I-1 to this appendix.

I-3.5.1 *Soil and Watershed Characteristics*

I-3.5.1.1 Soil Properties

For this assessment, soils were modeled as three stacked soil layers (surface, root zone, and vadose zone soil) over ground water. For soils not specifically modeled as land in agricultural use, the surface soil layer was assumed to be 1 centimeter (cm) deep. Agricultural soils were assumed to be tilled, and so a depth of 20 cm for the homogeneously mixed "surface soil" layer was assumed. The tilled soil compartments were used to estimate concentrations in farmed soil where produce is grown. Depths for surface and subsurface soil layers are presented in Exhibit I.3-15. Where soils were assumed to be tilled, the thickness of the root zone soil was reduced accordingly. Depths to and thicknesses of the vadose zone soil and groundwater layers were identical regardless of whether surface layer was tilled.

Exhibit I.3-15. Soil Compartment Depths

	Untilled Soil (m)	Tilled Soil (m)
Surface soil	0.00 – 0.01	0.00 – 0.20
Root soil	0.01 – 0.70	0.20 – 0.70
Vadose soil	0.70 – 2.10	0.70 – 2.10
Groundwater	2.10 – 5.10	2.10 – 5.10

For most of the basic surface soil properties, values were defined using typical regional or state values compiled by McKone et al. for use in multipathway modeling (2001). A list of selected soil properties is shown below in Exhibit I.3-16.

Exhibit I.3-16. Selected Properties of Soil and Groundwater

Property	Surface Soil	Root Zone Soil	Vadose Soil	Groundwater
pH	6.8	6.8	6.8	6.8
Organic carbon content	0.008	0.008	0.003	0.004
Volume fraction, vapor (air content)	0.28	0.25	0.22	--
Volume fraction, liquid (water content)	0.19	0.18	0.17	--
Average downwind vertical velocity of water infiltrating the soil (m/day)	8.22E-4	8.22E-4	8.22E-4	--

I-3.5.1.2 Erosion

Erosion rates for each surface parcel were estimated using the Universal Soil Loss Equation (USLE), with a sediment delivery (SD) ratio adjustment. The USLE is intended to predict the long-term average soil losses from individual field areas (Wischmeier and Smith 1978) and represents the sheet and rill erosion from a small plot or agricultural field. Application of the

USLE to an entire watershed requires modification of the equation result to account for subsequent re-deposition of eroded soil before it reaches the water body. The SD ratio was developed for this purpose: it estimates the fraction of sediment that reaches a water body based on the size of the watershed.

The USLE and SD equations use only a few inputs; representative site-specific values were developed to estimate erosion for this application with readily available data. Rainfall/erosivity values were used from Albany County for plots west of the Hudson River and Rensselaer County for regions east of the Hudson River (NRCS 2007). Soils data were obtained from the Soil Survey Geographic (SSURGO) database for the counties of interest (obtained from the USDA Natural Resources Conservation Service) to calculate site-specific soil erodibility factors. Different cover management factors were used for farm parcels and natural forests and grasses and herbs. For more information on the equations that were used and the derivation of values, see Attachment I-1. Calculated erosion rates for each surface soil parcel ranged from 9.5E-5 kilograms per square meter per day ($kg/m^2/day$) to 2.1E-3 $kg/m^2/day$.

The USLE is an empirical model, and therefore modeled conditions must be similar to conditions for which the model has been calibrated to output useful results. In particular, the USLE was designed for application to a single slope or field, rather than to an entire watershed. Using average values across a watershed parcel would likely introduce uncertainties in the prediction; predictions are improved when individual analyses of the slopes within the watershed are conducted. We note that the EPA's HHRAP documentation states that using the USLE to calculate sediment load to a lake from the surrounding watershed can sometimes lead to overestimates (EPA 2005). The use of area-weighted averages for some of the USLE variables helps to avoid under- or over-estimating by assuming uniformity across the watershed. The area-weighted soil erodibility factor (K) and cover management factor (C) are not expected to contribute significantly to inaccurate soil erosion estimates.

Estimating the length-slope (LS) factor is more challenging than any other factor for the USLE (Moore and Wilson 1992), especially for complex watersheds. In actual watersheds, the entire watershed has neither uniform slope length nor uniform slope steepness. Also, due to nonlinearities in the equation to calculate the LS factor, the assumption of uniformity can result in underestimates or overestimates of the LS factor. The use of average slope likely would underpredict the LS factor. An average slope-length of 200 m may be accurate or slightly greater than average, and thus may slightly overpredict the LS factor by some unknown amount. Finally, uncertainty is introduced when using the SD ratio to account for the re-deposition of soil before it reaches the water body. The degree by which the SD ratio underpredicts or overpredicts actual sediment delivery is unknown. Additional discussion of the assumptions made in estimating erosion rates for this modeling application and the associated uncertainties is included in Attachment I-1.

I-3.5.1.3 Runoff

Runoff from surface parcels into water bodies was calculated by subtracting the annual evaporation (0.508 m/year, USGS 2004) from the annual precipitation (0.980 m/year, NCDC 2003). This total runoff value includes interflow and ground water recharge; to estimate surface runoff only, total runoff was reduced by 50 percent per the recommendation included in HHRAP (EPA 2005). Total runoff rate for all surface parcels except the source parcel was estimated to equal 4.04E-4 $m^3/m^2/day$. The source parcel was not included in runoff because the Ravena facility is assumed to have different containment configurations than the rest of the area.

I-3.5.2 *Water Body Characteristics*

I-3.5.2.1 Surface Water and Sediment Properties

Important surface water and sediment properties for all lakes and the river are shown in Exhibit I.3-17; sources for these properties are values for all other inputs are listed in Attachment I-1.

Exhibit I.3-17. Selected Surface Water and Sediment Properties

Property	Value
Temperature (K)	287
Suspended sediment concentration (kg sediment/ kg water)	0.01
Water column and sediment organic carbon content (kg organic carbon/ kg solid wet weight)	0.02
Water column and sediment pH	7.3
Chlorophyll concentration (mg/L)	0.0029
Chloride concentration (mg/L)	8.0
Algae density in water column (g/L)	0.0025

I-3.5.2.2 Water Transfers

A water balance was assumed in order to estimate annual flush rates for each modeled water body. Inputs to each water body included runoff from the surrounding watershed and direct precipitation to the lake. Outputs from the water body included flushing through the lake outlet and evaporation from the lake surface.

Long-term average precipitation used to calculate the water balance was obtained from the Albany airport cooperative observation station. For the water body, this value was added as a water input, based on surface area of the lake. Runoff from the watershed was calculated by subtracting annual average evapotranspiration from annual average precipitation and multiplying the difference by the total watershed area. Evapotranspiration data were obtained from USGS (2004); a value of 20 inches was assumed to apply across the entire scenario. Reported runoff values closely matched the value we calculated by this method. For Kinderhook Lake, the calculated outflow from Nassau Lake was also included as a water input.

Evaporation from the lake surface was subtracted from the water inputs to estimate the volumetric flow of water leaving the water body. Using surface area and mean depth to calculate lake volume, a turnover rate in flushes per year was calculated. The values of these turnover rates are presented in Exhibit I.3-18.

Exhibit I.3-18. Turnover Rates for Ravena Water Bodies

Water Body	Turnover Rate
Kinderhook Lake	3.35
Nassau Lake	4.17
Alcove Reservoir	0.51
Pond	10.30

For water transfer calculations for the river, water velocity is required. The river velocity was calculated by dividing the average discharge rate of the Hudson (USGS 2008a) by the cross-sectional area of the Hudson River near Ravena (Oak Ridge National Laboratories 1977). The estimated river velocity calculated in this way is 0.88 meters per second (m/sec).

I-3.5.2.3 Sediment

The sediment balance of each watershed/water body system modeled was estimated by accounting for sediment inputs to the lake based on the erosion calculations and the removal of sediment from the modeled system via benthic burial and outflow of suspended sediment in the water column. In this scenario, assumptions about the physical environment were used to calculate sediment input through erosion and sediment removal through suspended sediment flushing. All sediment inputs to the watershed are derived from the erosion calculations.

For this modeled system, all sediment that is not flushed out as suspended sediment is assumed to be buried (i.e., removed from the modeled system by transfer to the consolidated benthic sediment layer, where it is assumed to no longer interact with the overlying water column). Suspended sediment depositional velocity is used to calculate total deposition to the lake bottom, and the difference between deposition and burial is then used to calculate the sediment that is resuspended. In TRIM.FaTE, resuspension rate is used to internally calculate burial rather than using the burial rate directly. Resuspension rates were calculated to match the calculated burial rates.

Based on these calculation methods, burial rates for the three Ravena lakes ranged from 0.0052 kg/(m^2-day) to 0.0129 kg/(m^2-day), with a value of 0.2066 kg/(m^2-day) calculated for the pond. The burial rate for the pond was set higher than the values for other watebodies in order to maintain the sediment input/output balance and offset the high erosion rates estimated for the pond watershed based on the presence of the land use category "Quarries/Strip Mines/Gravel Pits" (likely associated with the facility) covering about 20 percent of the watershed. In a survey of 56 lakes across the United States, median burial rates were range from 0.0027 to 0.0137 kg/(m^2-day) (USGS 2004), which is comparable to the values calculated for the lake scenarios (but substantially lower than the burial rate used for the pond). Exhibit I.3-19 presents estimated suspended sediment concentrations and calculated burial rates.

Exhibit I.3-19. Sediment Total Suspended Solids and Burial Rates for Ravena Water Bodies

Water Body	Suspend Sediment Concentration (kg sediment/ m^3 water)	Burial Rate	
		$\frac{m^3}{m^2 - day}$	$\frac{kg}{m^2 - day}$
Kinderhook Lake	0.010	5.0E-06	5.2E-03
Nassau Lake	0.010	1.2E-05	1.3E-02
Alcove Reservoir	0.010	6.6E-06	6.9E-03
Pond	0.110[a]	2.0E-04	2.1E-01

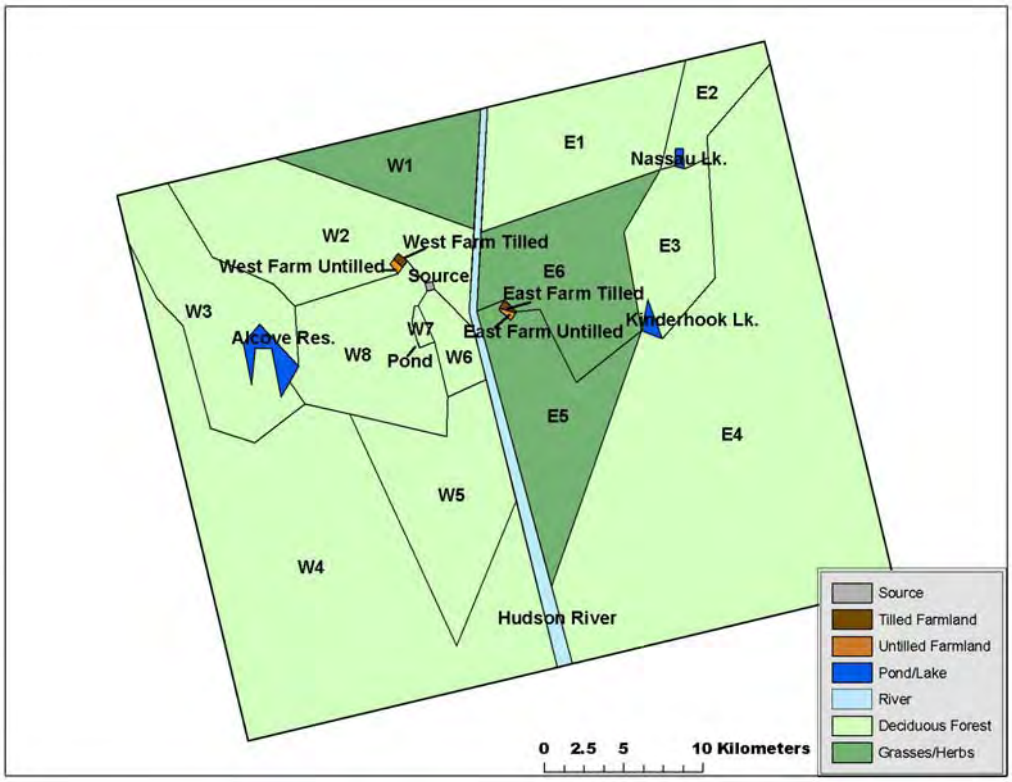
[a] Pond suspended sediment concentration was assumed to be higher than those for other water bodies because of higher erosion rates and small water body size.

I-3.6 Terrestrial Plants

Calculations of the areal coverage of each land-use type within each parcel were used to set each modeling surface parcel's dominant vegetation type (using the National Land Cover Dataset 1992 (USGS 1992) classifications grouped to match the TRIM.FaTE vegetation types as described in Section I-3.6). This strategy results in some simplification because most parcels are at least several square kilometers in area and contain a variety of land-use. However, key parcels such as the farms are drawn smaller in order to more accurately represent actual land

use. The TRIM.FaTE vegetation designations are presented in Exhibit I.3-20, along with the names of each parcel.

Exhibit I.3-20. Surface Parcel Layout with Plant Types and Relevant Land Use



Modeling plants in TRIM.FaTE requires two additional properties: (1) an "allow exchange" property that is used in TRIM.FaTE algorithms to determine whether plants are actively growing (and thus able to exchange chemical mass to and from the ambient air and take up chemical mass from soil); and (2) a litterfall rate property that dictates when and how fast chemical mass accumulated by a leaf is transferred to underlying surface soil (to account for chemical transfers to soil from leaves dropped by deciduous trees and plants). For this assessment, the dates at which these seasonal events occur were based on the dates of the first and last frosts reported for Albany, NY (NOAA 1988). The average last day of frost in the spring is April 23, and the first date of frost in the fall is October 15, assuming a 50-percent probability of a temperature threshold of 28 °F. Litterfall is assumed to begin on the first day of frost and to end 30 days after this date, with a litterfall rate of 15 percent of the remaining detritus falling per day.

I-3.7 Aquatic Ecosystem

To estimate risks to human health for the angler scenario, site-specific models of aquatic food webs were developed in TRIM.FaTE to represent four water bodies in the vicinity of Ravena, NY: Nassau and Kinderhook Lakes, Alcove Reservoir, and an unnamed small pond near the facility. Characteristics of the TRIM.FaTE fish compartments used to represent fish in each water body were based on site-specific fish survey data, supplemented by information from the open literature.

The development of each food web consisted of three stages:

1. Collection of local fish survey data for the water bodies from the New York State Department of Environmental Conservation (NY DEC), including data on the relative abundance and size/weight distribution of each species, to the extent available;

2. Formulation of simplified food webs for each water body, based on the fish surveys and other biological and physical data for each water body, with supplemental information on fish feeding habits, aquatic food webs, and biomass densities for different trophic levels from the open literature; and

3. Assignment of values for the remaining parameters (e.g., individual body weight, numeric density per unit area, lipid content) for each biotic compartment for each water body in TRIM.FaTE from the available data.

Professional judgment was used where available data were incomplete. The process employed to configure TRIM.FaTE aquatic food webs and set model input properties is summarized here and discussed in greater detail in Attachment I-1.

I-3.7.1 *Collection of Information on Species Present in Water Bodies*

To support the development of the aquatic food webs, ICF contacted fishery biologists at the NY DEC Region 4 Bureau of Fisheries. The NY DEC conducted surveys of fish in Nassau and Kinderhook Lakes at various times between 1988 and 2006 (NY DEC 2008). Due to the presence of polychlorinated biphenyls (PCBs) at Nassau and Kinderhook Lakes, there are fish consumption advisories at these water bodies (NY DOH 2007), and aquatic sampling is performed to assess current contaminant levels. The New York State Fish and Wildlife Department published the results of fish surveys conducted from 1963 to 1970 for Alcove Reservoir (NY FWD 1971). This 1971 survey report presented data on average fish weights, which were used, where applicable, to estimate the average weight per individual fish for each species in all of the modeled water bodies for this assessment. Because data on fish length or weight were not available for the other water bodies, average fish weights for each species from the Alcove report were used as the average fish weights for the same species in the other water bodies.

No survey or other site-specific data were identified for the small pond. Professional judgment and published data were used to develop a model food web for the small pond. The food web for the small pond was developed from an analysis of data presented by Demers et al. (2001) for two small lakes in Canada. As a conservative position, the small pond was assumed to sustain a viable fish community from year to year. In each water body, young of the year were assumed to comprise 15 percent of the total fish biomass on an annual basis biomass.

I-3.7.2 *Creation of Food Webs*

Food webs for each of the four water bodies were constructed from the information sources identified above. Several steps were required to construct each food web and to assign parameter values for all aquatic biotic compartments for TRIM.FaTE:

1. Estimate total standing fish stock (i.e., total fish biomass per unit area) for each water body based on total biomass estimates reported for similar water bodies in the literature;

2. List for each water body all fish species found in the surveys of the water body;

3. Identify for each species an average body weight per individual based on the Alcove Reservoir data;

4. Estimate total biomass caught for each species in the surveys by multiplying the number of individuals of each species caught over the survey years for the water body by the average body weight per individual for each species;

5. Estimate the relative total biomass for each species (percentage of total biomass represented in surveys);

6. Estimate the absolute biomass of each species by multiplying its percent relative biomass by the estimated total standing fish stock (Step 1);

7. Estimate the numeric density of each fish species (number per unit area) based on biomass density and average individual weight for each species; and

8. Evaluate the feeding habits of each fish species, as determined from a variety of sources, relative to the food/prey categories supported by TRIM.FaTE:

    - plankton (called algae in TRIM.FaTE; however, it represents both phytoplankton and zooplankton);
    - macrophytes;
    - benthic invertebrates (e.g., aquatic insects, crustaceans, mollusks);
    - small planktivorous fish (e.g., young of the year, minnows; feed on algae and zooplankton in the water column);
    - larger omnivorous fish that feed on smaller fish in the water column and benthic invertebrates and/or macrophytes (e.g., sunfish, yellow perch)
    - small-to-medium sized benthivores/omnivores that feed primarily on benthic invertebrates, detritus, and possibly macrophytes (e.g., small carp, white sucker).

Additionally, the lipid content of each species was estimated based on values reported in national surveys.

The initial estimates of relative abundance for each fish species were based on the fish survey data. These data are presented for reference in Attachment I-1. Only the species identified by fish surveys were assumed to be present in the four modeled water bodies. The body weight of each individual was assumed to be equal to the average fish weight estimated from the Alcove surveys. When species were present in the other lakes, but not in the Alcove Reservoir, professional judgment and readily available data for other locations (e.g., Minnesota fish surveys) were used to estimate an average individual body weight for the species.

At the small pond, only three species/groups were assumed to be present: largemouth bass, sunfish (e.g., bluegill or pumpkinseed), and shiners. The mass of the individuals was estimated based on professional judgment and the Demers et al. (2001) study of two small lakes.

Total relative biomass for each species within a water body was estimated differently for the four water bodies. At Alcove Reservoir, each species' biomass representation was determined by taking the observed biomass of the species caught across all survey years and dividing that by the total fish biomass reported in the Alcove report across all survey years. At both Nassau and Kinderhook Lakes, the survey data seemed biased towards several species, specifically yellow and white perch, perhaps due to sampling techniques. We therefore adjusted biomass representation to reflect a more balanced abundance across different species for these two

water bodies. For the small pond, the distribution of biomass among the several species was estimated based on the study published by Demers and co-workers (2001).

I-3.7.3 *Parameterization of Fish Compartments to be Included in Application*

All fish species were assigned to one of the following five fish compartments established in TRIM.FaTE:

- Water column carnivore (large predominantly piscivorous species, e.g., walleye and largemouth bass);
- Water column omnivore (medium-sized fish that feed primarily in the water column, e.g., sunfish, yellow perch);
- Water column herbivore (more appropriately termed planktivore);
- Benthic carnivore (large carnivorous species, e.g., large bullhead, eel); and
- Benthic omnivore (medium-sized fish that feed primarily on benthic invertebrates).

The compartment to which each species was assigned was determined by its general foraging habitat (i.e., benthic or water column) and its primary food sources (e.g., invertebrates, smaller fish, plant material). The total biomass for each of the five fish TRIM.FaTE compartments was set equal to the sum of the biomass of the species assigned to each compartment.

The diet composition for each of the five fish compartments was calculated as being proportional to the biomass representation of each species assigned to that compartment. For example, if largemouth bass comprised 75 percent and smallmouth bass comprised 25 percent of the biomass of the WCC compartment, then the diet composition of the largemouth bass multiplied by 0.75 would be added to the diet composition of the smallmouth bass multiplied by 0.25 to estimate the diet composition for the WCC compartment. The four aquatic food webs developed for the Ravena case study are summarized in Exhibit I.3-21. Similarly, the lipid content for each of the five fish compartments in TRIM.FaTE was estimated from the biomass-weighted lipid content of the individual species assigned to the compartment. Thus, using the same example, the largemouth bass lipid content, multiplied by 0.75, would be added to the smallmouth bass lipid content, multiplied by 0.25, to estimate the lipid content of the WCC compartment.

Exhibit I.3-21. Aquatic Food Webs for Modeled Water Bodies

TRIM.FaTE Compartment	Representative Fish Species	Diet Fractions					
		Algae/ Zooplankton	Macrophytes	Benthic Invertebrates	Benthic Omnivores	Water Column Herbivores	Water Column Omnivores
Alcove Reservoir							
Water Column Carnivore	Chain pickerel, largemouth bass, northern pike, walleye			41.1%	25.0%	4.5%	29.5%
Water Column Omnivore	Bluegill, pumpkinseed, redbreast sunfish, smallmouth bass, white perch, yellow perch	7.8%		53.5%		38.8%	
Water Column Herbivore	Black crappie, young of the year	96.3%		3.7%			
Benthic Carnivore	American eel			50.0%	50.0%		
Benthic Omnivore	Bullhead			100%			
Kinderhook Lake							
Water Column Carnivore	Largemouth bass, tiger musky, walleye			33.0%	25.7%	7.8%	33.5%
Water Column Omnivore	Bluegill, pumpkinseed, redbreast sunfish, rock bass, smallmouth bass, white perch, white sucker, yellow perch	8.1%		57.9%		34.0%	
Water Column Herbivore	Black crappie, common carp, fantail darter, golden shiner, young of the year	81.8%	13.5%	4.7%			
Benthic Carnivore	American eel			50.0%	50.0%		
Benthic Omnivore	Bullhead			100%			
Nassau Lake							
Water Column Carnivore	Chain pickerel, largemouth bass				25.0%	25.0%	50.0%
Water Column Omnivore	Bluegill, pumpkinseed, redbreast sunfish, smallmouth bass, white perch white sucker, yellow perch	8.7%		61.0%		30.3%	
Water Column Herbivore	Black crappie, common carp, golden shiner, young of the year	92.4%	2.7%	4.9%			
Benthic Carnivore	American eel			50.0%	50.0%		
Benthic Omnivore	Bullhead			100%			

Exhibit I.3-21, continued. Aquatic Food Webs for Modeled Water Bodies

TRIM.FaTE Compartment	Representative Fish Species	Diet Fractions					
		Algae/ Zooplankton	Macrophytes	Benthic Invertebrates	Benthic Omnivores	Water Column Herbivores	Water Column Omnivores
Small Pond							
Water Column Carnivore	Largemouth bass			50.0%		50.0%	
Water Column Herbivore	Golden shiner, young of the year	100%					
Benthic Omnivore	Sunfish			100%			

Note: For the purpose of the ecological risk assessment, mallards were included in the four water bodies at the Ravena site. The mallard diet consists of 67 percent macrophytes and 33 percent benthic invertebrates. See Appendix J for further discussion.

I-3.8 Mass Balance Results

One summary generated by TRIM.FaTE for each model run is a report of the chemical mass at the conclusion of the simulation in each compartment type included in the modeled environment. Exhibit I.3-22 presents the distribution of modeled chemical mass in the modeled environment for TRIM.FaTE 2,3,7,8-TCDD and mercury modeling for the Ravena site. The first section of this table presents the proportion of chemical mass emitted over the 50 year period in Ravena that was removed from the scenario by transfer to air advection sinks (i.e., the fraction of mass that was blown out of the modeled environment by wind). The remaining fraction is the amount emitted and deposited from air to soil, water, and plant surfaces comprising the overall surface layout via wet and dry deposition processes (including vapor diffusion where applicable).[6] In the second part of this table, the *final* distribution of the *deposited* chemical mass at the ending time step is summarized by media and modeling sink type and within soil layers.

For dioxins, most of the mass emitted by the modeled source is blown out of the modeled domain into air sinks, and less than 2 percent of total dioxin emitted is deposited within the scenario. Of the amount present in the scenario at the end of the simulation (minus emitted mass in the air advection sinks), 85 percent had degraded; most of the remaining chemical mass was found in the surface soil. Only trace amounts of the deposited are estimated to remain present in surface water, and aquatic biota. No abnormal resuspension events are assumed; taking into account these events would result in higher concentrations and exposure. Mass distributions between emission factors; the only the total amount in the system changed.

[6] Note that the fraction "emitted and deposited" in this table represents the proportion of emitted chemical that is immediately deposited from air, not the fraction of emitted chemical mass in the soil, water, and plant compartments at the conclusion of the simulation. Once deposited, chemical mass in TRIM.FaTE can be re-emitted to the air (e.g., via volatilization of vapor-phase chemical or dust resuspension), transported to another environmental compartment via advective or other processes, accumulated by biotic compartment types included in the scenario, metabolized or broken down by abiotic degradation processes, or (in the case of mercury) transformed to another modeled chemical.

Five percent of the total emitted chemical mercury mass remained within the bounds of the Ravena screening scenario; the remaining was found in air sinks.

Of the mass remaining in the scenario, most was found in the soil and sediment, very little had been lost due to reaction/degradation, and most of the remaining about had been lost in sediment sinks and removal via the Hudson River outside of the system.

Exhibit I.3-22. Distribution of Chemical Mass in Ravena, NY Scenario

Compartment	2,3,7,8-TCDD (95th)	2,3,7,8-TCDD (Mean)	Divalent Mercury (Hg2)	Elemental Mercury (Hg0)	Methyl Mercury (MeHg)	Total Mercury
Distribution of Total Mass Added to Scenario from Modeled Source						
Emitted chemical mass removed from scenario and transferred to air sinks	98.1%	98.1%	79.0%	99.9%	- [a]	94.7%
Emitted chemical mass that is deposited to soil, water, and plants	1.9%	1.9%	21.0%	0.1%	- [a]	5.3%
Distribution of Mass Remaining in Scenario						
Air	0.03%	0.03%	0.002%	0.5%	0.00%	0.01%
Soil	11.2%	11.2%	83.7%	39.3%	88.6%	83.1%
Plants	0.1%	0.1%	0.03%	0.00%	0.00%	0.03%
Surface Water	0.001%	0.001%	0.01%	0.1%	0.01%	0.01%
Sediment	0.1%	0.1%	2.0%	0.8%	0.3%	2.0%
Aquatic Biota [b]	0.0002%	0.0002%	0.00%	0.00%	0.0003%	0.0000%
Groundwater	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
Reaction Sink	85.6%	85.6%	0.00%	0.00%	0.00%	0.00%
All Other Sinks [c]	2.9%	2.9%	14.2%	59.4%	11.1%	14.8%
Distribution of Mass in Soil						
Surface Soil	99.2%	99.2%	99.4%	2.3%	99.4%	98.7%
Root Zone Soil	0.8%	0.8%	0.6%	97.5%	0.6%	1.3%
Vadose Zone Soil	0.00%	0.00%	0.00%	0.2%	0.00%	0.001%

[a] No methyl mercury was emitted directly from the point source; it was created via transformation within the TRIM.FaTE system, therefore the percentage as a fraction of total emissions cannot be calculated
[b] Compartment includes mallard, but does not include mink.
[c] Other sinks include soil sinks, sediment sinks for water bodies and the river, and a flush sink for chemical removed by being carried via the Hudson river.

I-4 Exposure Assessment

I-4.1 Approach

The Ravena facility site-specific HHRA is intended to address non-inhalation (ingestion) exposures to potential human receptors. Consistent with the scenario assessment approach described in Section I-2.2, exposures for specific scenarios were estimated using assumed ingestion activity patterns (i.e., estimating how much of each medium is consumed and the fraction of the consumed medium that is grown in or obtained from contaminated areas) and

characteristics of the individual potentially exposed (e.g., age and body weight). For the human health assessment, three ingestion exposure scenarios were evaluated:

1. Consumption of farm-grown fruits, vegetables, and animal products, and incidental ingestion of soil;
2. Consumption of self-caught fish from local water bodies; and
3. Ingestion of contaminated breast milk by infant.

Exposure doses (and subsequent risks) associated with dioxins and mercury for each ingestion pathway were computed separately using the MIRC model so that the pathway(s) of interest for each PB-HAP could be evaluated separately. Data related to exposure factors and characteristics of exposed individuals were obtained primarily from EPA's Exposure Factors Handbook (EPA 1997a, b).

As described in previous sections, exposures were modeled for two hypothetical farm homesteads (referred to here as West Farm parcel and East Farm parcel) and four potentially fishable water bodies near the Ravena facility. West Farm parcel is located approximately 2.5 kilometers (km) northwest of the Ravena facility; East Farm parcel is approximately 5 km south-southeast of the facility. Each parcel is approximately 0.72 km^2 in size, and each is roughly bisected into tilled and untilled parts. The size and location of the four water bodies are provided below. The unnamed pond is the closest body of water to the source that could theoretically support a fish population.

- Alcove Reservoir (5.5 km^2) – west of source
- Nassau Lake (0.65 km^2) – east of the source
- Kinderhook Lake (1.3 km^2) – east of the source
- Small (unnamed) pond (0.020 km^2) – south of source

A summary of the sources of contaminated media for each of the three exposure scenarios evaluated is provided in Exhibit I.4-1. See Section I-3.3 for site maps and a detailed description of the spatial layout of the site, including the areas and locations of the farm and watershed parcels relative to the Ravena facility, as well as land use patterns in the area surrounding the facility.

Exhibit I.4-1. Ingestion Exposure Scenarios

Scenario	Source of Ingested Media
Consumption of locally-grown produce and animal products, and incidental ingestion of soil	Products and soil from two locations with agricultural land use: o East Farm parcel o West Farm parcel
Consumption of locally-caught fish by sport anglers	Fish from four water bodies: o Alcove Reservoir o Kinderhook Lake o Nassau Lake o Small pond to south
Ingestion of contaminated breast milk by infants	Breast milk; nursing mother would ingest farm and fish media from most exposed locations

Estimated individual contact rates (i.e., exposure) for each exposure medium were evaluated using two point estimates – one to represent average or central tendency exposure (CTE) and another to represent upper-bound or reasonable maximum exposure (RME). The CTE calculation is used to estimate exposure for individuals with average or typical intake of

environmental media, taking into account the basic assumptions of the scenario (for example, for the angler scenario, the exposed individual was assumed to regularly consume self-caught fish). The RME is used to estimate exposures for individuals exposed via the evaluated scenarios who are at the high end of the exposure distribution. The intent of the RME is to estimate a conservative exposure case (i.e., well above the average case) that is still within the range of possible exposure patterns. For the CTE scenario, consumer-only intakes/ingestion rates for soil, fish, breast milk, and every FFC medium were set equal to the mean of the distribution of national or other representative data for that food type. For the RME scenario, estimates of the 90th percentile of consumer-only intakes/ingestion rates were used. For both the CTE and RME ingestion exposure scenarios, all food types specific to a certain exposure scenario (i.e., farmer, angler, or breast-feeding infant) were assumed to be obtained from locations evaluated in this assessment (i.e., a farm parcel and/or one of the two water bodies, or from a nursing mother consuming media from one or both of these routes). The approach to estimating RME ingestion exposure is analogous to EPA's recommended approach for conducting risk assessments at Superfund sites – that is, we are estimating the "highest exposure that is reasonably expected to occur at a site," taking into account current and future (potential) land-use conditions (EPA 1989). The RME is assumed in this instance to approximate the maximum individual risk (i.e., the upper bound of individual risk based on conservative assumptions that is unlikely to be exceeded). We have also estimated risk for the CTE scenario to provide additional information that may be helpful in evaluating the level of conservatism associated with this MIR estimate.

The conditions defined when conceptualizing and building the scenario were selected so that for any given individual, a long-term exposure condition would be reasonably likely to be captured, thereby ensuring that this estimate encompasses the MIR. However, we emphasize again that because this assessment is designed to estimate the maximum individual risk ***for the exposure scenarios evaluated***, the results are not intended to represent the actual exposure for a typical person living in the vicinity of the evaluated source, but rather the estimated exposure for a person who meets the criteria of the scenarios evaluated (that is, someone who consumes only produce grown and animals raised on a local farms, and/or someone who regularly consumes self-caught fish from a local lake). The CTE scenario, therefore, represents an average, "central tendency" exposure estimate within the relatively strict specifications of the exposure scenarios developed for this assessment.

The remainder of this section describes the approach for estimating human exposures associated with the incidental ingestion of soil, ingestion of FFC media, consumption of fish, and infant consumption of breast milk. A discussion of exposure pathways for potential human receptors is presented in Section I-4.2. Section I-4.3 describes the approach used to estimate exposure-related dose for each relevant ingestion source and pathway, and includes a summary of exposure parameters and assumptions.

I-4.2 Exposure Pathways

A summary of the ingestion exposure pathways evaluated in this assessment is provided in Exhibit I.4-2. The quantitative aspects of this non-inhalation evaluation focused primarily on human exposures via the following ingestion pathways: incidental ingestion of soil; ingestion of farm-food chain (FFC) media; ingestion of fish; and infant ingestion of breast milk. Each pathway is discussed in the following subsections.

Exhibit I.4-2. Summary of Ingestion Exposure Pathways and Routes of Uptake

Ingestion Exposure Pathway	Medium Ingested	Intermediate Exposure Pathway – Farm Animals [a]	Environmental Uptake Route	
			Medium	Process [b]
Farm-Food Chain				
Consumption of produce	Aboveground produce, exposed fruits and vegetables [c]	NA	Air Air Soil	Deposition to leaves/plants Vapor transfer Root uptake
	Aboveground produce, protected fruits and vegetables [c]	NA	Soil	Root uptake
	Belowground produce	NA	Soil	Root uptake
Consumption of farm animals and related food products	Beef	Ingestion of forage	Air Air Soil	Direct deposition to plant Vapor transfer to plant Root uptake
		Ingestion of silage		
		Ingestion of grain	Soil	Root uptake
		Ingestion of soil	Soil	Ingestion from surface
	Dairy (milk)	Ingestion of forage	Air Air Soil	Direct deposition to plant Vapor transfer to plant Root uptake
		Ingestion of silage		
		Ingestion of grain	Soil	Root uptake
		Ingestion of soil	Soil	Ingestion from surface
	Pork	Ingestion of silage	Air Air Soil	Direct deposition to plant Vapor transfer to plant Root uptake
		Ingestion of grain	Soil	Root uptake
		Ingestion of soil	Soil	Ingestion from surface
	Poultry	Ingestion of grain	Soil	Root uptake
		Ingestion of soil	Soil	Ingestion from surface
	Eggs	Ingestion of grain	Soil	Root uptake
		Ingestion of soil	Soil	Ingestion from surface
Incidental ingestion of soil	Surface soil	NA	Surface soil	Deposition; transfer through plants; transfer via erosion and runoff [c]
Fish				
Consumption of fish	Locally-caught fish (see Exhibit I.4-3)	NA	Fish tissue	Direct uptake from water and consumption of other contaminated media modeled in TRIM.FaTE [d]

Exhibit I.4-2, continued. Summary of Ingestion Exposure Pathways and Routes of Uptake

Ingestion Exposure Pathway	Medium Ingested	Intermediate Exposure Pathway – Farm Animals [a]	Environmental Uptake Route	
			Medium	Process [b]
Drinking Water				
Ingestion of drinking water	Surface Water	NA	Surface Water	Deposition to surface water and transfer via erosion and runoff
Breast Milk				
Consumption of breast milk	Breast milk	NA	Breast milk	Ingested by mother and then partition to breast milk

[a] NA = not applicable; calculation of intermediate exposure concentrations was required only for the farm animal/animal product ingestion pathways.
[b] Process by which HAP enters medium ingested by humans.
[c] For fruits and vegetables, the terms "exposed" and "protected" refer to whether the edible portion of the plant is exposed to or protected from the atmosphere.
[d] Modeled in TRIM.FaTE.

Alcove Reservoir is a drinking water reservoir for eastern New York, therefore the exposure calculations assume that Alcove Reservoir is the only source of drinking water for the exposed individual. The surface water concentrations of PB-HAPs in Alcove Reservoir modeled by TRIM.FaTE were used in exposure scenarios.

In addition to ingestion, non-inhalation exposure to PB-HAPs also can occur by way of the dermal pathway (e.g., through incidental contact with PB-HAP-contaminated soil). However, dermal absorption of chemicals that are originally airborne is generally a minor pathway of exposure relative to other exposure pathways such as inhalation exposure or exposure via ingestion of contaminated crops, soil, or breast milk (EPA 2008, CalEPA 2000). In general, the assessment followed the protocol for evaluating a reasonable maximum exposure as described in EPA's Risk Assessment Guidance for Superfund (RAGS), Volume I: Human Health Evaluation Model, Part E, Supplemental Guidance for Dermal Risk Assessment (EPA 2004c).

I-4.2.1 *Farm Food Chain Media Pathway*

Data from the 2002 Census of Agriculture (USDA 2002) for Albany, Columbia, Greene, and Rensselaer Counties of New York were examined to determine the relevant FFC exposure pathways for this analysis. The census recorded the presence of cattle (for beef and milk), hogs and pigs, chickens (for eggs and meat), corn, wheat, oats, beans, potatoes, forage, vegetables and orchards in the four counties included in the Ravena, NY, spatial layout. Based on this information, the following FFC pathways were evaluated in this assessment:

- Ingestion of homegrown produce (fruits and vegetables),
- Ingestion of homegrown beef,
- Ingestion of milk from homegrown cows,
- Ingestion of homegrown pork, and
- Ingestion of homegrown poultry and eggs.

Exposures to dioxin and mercury (as divalent mercury and methyl mercury) via these FFC pathways were evaluated.

I-4.2.2 *Fish Consumption Pathway*

This site-specific evaluation assessed human exposures via aquatic food chain contamination, considering both game fish, which are generally top predators in general within aquatic ecosystems, and bottom-feeding fish that might also be consumed by humans. As described in Section I-3.7, data from several fish surveys conducted by the New York State Department of Environmental Conservation (NY DEC) between 1988 and 2006 were used to estimate the relative abundance of different fish species in each lake for Kinderhook and Nassau Lakes, and data for Alcove Reservoir were obtained from NY DEC fish surveys conducted between 1963 and 1970. The food web for the small pond was derived from an analysis of data presented by Demers et al. (2001) for two small lakes in Ontario. We were not able to confirm what type of fish population (if any) is present in the pond; the assumptions made for this risk assessment are intended to err on the conservative side (i.e., by assuming that regular consumption of fish caught in the pond could occur). The proportion of total fish biomass for each water body contributed by each species was assigned to one of the following five fish compartments on the basis of professional judgment and descriptions of their feeding habits available from online fishing communities and from NYS DEC online documents: benthic omnivore (BO); benthic carnivore (BC); water column herbivore (WCH); water column omnivore (WCO); or water column carnivore (WCC). A summary of fish species present in water bodies around the Ravena facility that are considered in the human health assessment is provided in Exhibit I.4-3. See Section I-3.7.3for a discussion of these compartments and the aquatic food web modeled by the TRIM.FaTE for this simulation. Uncertainties associated with the assumptions regarding fish populations and consumption rates are discussed in more detail in Section I-6.

Exhibit I.4-3. Fish Species Assumed to be Consumed in this Assessment

Water Body	TRIM.FaTE Compartment Type [a]	Representative Species	Fraction of Total Angler Consumption (by mass)
Alcove Reservoir	WCC	Chain pickerel, largemouth bass, northern pike, walleye	33%
	WCH	Black crappie, young of the year	--
	WCO	Bluegill, pumpkinseed, redbreast sunfish, smallmouth bass, white perch, yellow perch	67%
	BC	American eel	--
	BO	Bullhead	--
Kinderhook Lake	WCC	Largemouth bass, tiger musky, walleye	33%
	WCH	Black crappie, common carp, fantail darter, golden shiner, young of the year	--
	WCO	Bluegill, pumpkinseed, redbreast sunfish, rock bass, smallmouth bass, white perch, white sucker, yellow perch	67%
	BC	American eel	--
	BO	Bullhead	--

Exhibit I.4-3, continued. Fish Species Assumed to be Consumed in this Assessment

Water Body	TRIM.FaTE Compartment Type [a]	Representative Species	Fraction of Total Angler Consumption (by mass)
Nassau Lake	WCC	Chain pickerel, largemouth bass	33%
	WCH	Black crappie, common carp, golden shiner, young of the year	--
	WCO	Bluegill, pumpkinseed, redbreast sunfish, smallmouth bass, white perch, white sucker, yellow perch	67%
	BC	American eel	--
	BO	Bullhead	--
Small (unnamed) pond	WCC	Largemouth bass	33%
	WCH	Golden shiner, young of the year	--
	BO	Sunfish	67%

[a] BO = benthic omnivore; BC = benthic carnivore; WCC = water column carnivore; WCH = water column herbivore; WCO = water column omnivore

I-4.2.3 *Breast Milk Pathway*

The U.S. EPA (EPA 1980,1983) and the World Health Organization (WHO1985,1989) have published multiple reports documenting the presence of environmental chemicals and contaminants in human breast milk. The magnitude of the nursing infant's exposure via ingestion of contaminated breast milk can be estimated from information on the mother's exposure, data on the partitioning of the chemical into various compartments of the mother's body and into breast milk, and information on the infant's consumption of milk and absorption of the chemical. This pathway is generally of most concern for *lipophilic* bioaccumulative chemicals (e.g., dioxins) that can cause developmental effects. The methodology and algorithms used for the breast milk consumption scenario for this case study are presented separately in Attachment C-2. Only the results of the analyses are presented in this appendix.

I-4.3 Exposure Dose Estimation

Ingestion exposures for the angler and farmer scenarios for all media were calculated as average daily doses (ADDs), expressed in milligrams of PB-HAP per kilogram of receptor body weight per day (mg/kg-day). The equations in MIRC that were used to calculate ADDs for each of the ingestion pathways are presented in Attachment C-2. Inputs to MIRC used for the exposure dose estimates (as ADDs) and risk estimates for this assessment included TRIM.FaTE PB-HAP concentrations, FFC algorithm parameters dictating the chemical quantity accumulated in produce and animals/animal products, and exposure factors. Each of these inputs is discussed below.

I-4.3.1 *Media Concentrations*

For the human health assessment, TRIM.FaTE was used to estimate human exposures to 2,3,7,8-TCDD and mercury via ingestion of locally grown produce and animal products and ingestion of self-caught fish in several bodies of water in the vicinity of the Ravena facility. As mentioned above, the water bodies include a farm pond near the facility, the Alcove Reservoir, Kinderhook Lake, and Nassau Lake. Because it is largely a flow-through system, concentrations of 2,3,7,8-TCDD and mercury attributable to the Ravena facility were not estimated for the Hudson River.

From TRIM.FaTE, the following environmental media concentrations specific to the PB-HAP of concern were obtained:

- air concentrations (in g/m^3);
- air-to-surface deposition rates for both particle and vapor phases (in g/m^2-yr);
- ground water concentrations (in g/L);
- fish tissue concentrations for fish in trophic levels three and four (T3 and T4) (in mg/kg wet weight); and
- concentrations in surface soil and root zone soil (in µg/g dry weight).

These PB-HAP-specific values were then used to calculate receptor- and exposure scenario-specific ADDs, using the pathway-specific equations provided in Attachment C-2.

For FFC exposure calculations, concentrations in FFC media were calculated using empirical biotransfer factors (e.g., soil-to-plant factors, which are the ratios of the concentrations in plants to concentrations in soil). In general, plant- and animal-specific parameter values, including chemical-specific transfer factors for FFC media, were obtained from the Hazardous Waste Companion Database included in HHRAP (EPA 2005). A list of variables and PB-HAP-specific input parameters, along with the input values used in the evaluation of the FFC pathway, are provided in Attachment I-4.

I-4.3.2 *Exposure Factors*

Specific exposure factors used to estimate ADDs for the evaluated scenarios are summarized in the following subsections. For this evaluation, exposure characteristics were selected to calculate average (CTE) and upper-bound (RME) estimates of exposure for the scenarios of interest. These two estimates were derived by varying only the assumed intake and ingestion rates for an individual; the values remained the same for both CTE and RME estimates for other exposure factors (i.e., body weight, exposure frequency, fraction of food from contaminated sources, and cooking loss). Exposure factors were obtained primarily from EPA's Exposure Factors Handbook (EPA 1997a, b).

Average ingestion rates used to calculate the CTE estimate were based on mean values reported for relevant individuals. For the RME estimate, 90^{th} percentile ingestion rates were used for all food types assumed to be eaten. We realize that such an assumption can lead to a ***total*** food ingestion rate that is extreme; for example, the total amount of food consumed per day is nearly 6 kg for the farmer exposure scenario if 90^{th} percentile ingestion rates from the selected data set are assumed for all produce and meat/animal products. One approach to developing a more realistic estimate of RME (that still evaluates high-end exposures) is to assume that the exposed individual consumes food at the upper percentile ingestion rate only for the one or two food types that dictate the total exposure to each chemical based on the assumptions included in the current assessment, and ingestion rates for other FFC media or fish are equal to mean reported values. For dioxins, consumption of fish, beef, and dairy products are the food types that drive the long-term exposures. For mercury, the food types that dictate exposures vary by chemical species, with beef/dairy consumption driving exposures to Hg2, ingestion of soil and exposed vegetables and fruits driving exposures to Hg0, and fish consumption driving exposure to MeHg. However, if this approach is taken, the total exposure (via all ingestion pathways) to each chemical evaluated was found to be roughly the same regardless of whether the upper percentile ingestion rates are assumed for only the risk-driving food types or for all food types. Therefore, to simplify the presentation of methods and results,

we chose to assume ingestion at the 90th percentile for ***all*** consumed media (farm products, soil, and/or fish) included in a scenario for the RME estimate.

Age of Exposed Individual

Exposures (i.e., ADDs) were calculated separately for four children's age groups: 1 to 2 years (Child Group 1), 3 to 6 years (Child Group 2), 6 to 11 years (Child Group 3), and 12 to 19 years (Child Group 4)) and for adults. Exposures were also calculated for infants less than 1 year of age but only for the breast milk ingestion pathway. As described in Attachment C-2, these age groupings were selected based on the availability of ingestion rate data for adults and children.

Body Weight

Body weights for each age group were estimated from data included in EPA's Exposure Factors Handbook (EPA 1997a); body weights used were weighted means of the national distribution. The body weights assumed for this assessment are presented in Exhibit I.4-4. A single body weight was used for each age group for all scenarios (i.e., separate RME and CTE estimates of body weight were not evaluated).

Exhibit I.4-4. Body Weight Estimates Used in This Assessment

Age of Exposed Individual	Mean Body Weight (kg)
Less than 1 year old	7.8 [a]
1 to 2 years	12.6 [b]
3 to 5 years	18.6 [c]
6 to 11 years	31.8 [d]
12 to 19 years	64.2 [e]
Adult	71.4 [f]
Nursing Mother	66.0 [g]

[a] Derived from time-weighted averages of body weights for age groups birth to <1 month, 1 to <3 months, 3 to <6 months, and 6 to <12 months from Table 8-3 of EPA 2008.
[b] Derived from time-weighted averages of body weights for age groups 1 to <2 years and 2 to <3 years from Table 8-3 of EPA 2008.
[c] Obtained directly from Table 8-3 of EPA 2008 (age group 3 to <6 years)
[d] Obtained directly from Table 8-3 of EPA 2008 (age group 6 to <11 years). Value represents a conservative (i.e., slightly low) estimate of BW for ages 6 through 11 years.
[e] Estimated using Table 8-22 of EPA 2008, based on NHANES IV data as presented in Portier et al. (2007). This estimate was calculated as the average of the 8 single-year age groups from 12 to 13 years through 19 to 20 years.
[f] Derived from the sample-size weighted average of male and female mean body weights (all races, 18-74 years) from EPA's 1997 EFH (Tables 7-4 for males and 7-5 for females).
[g] Used as the maternal body weight only in calculations for the breast milk exposure analysis. This value is from EPA 2004d.

Intake and Ingestion Rates for Farmer Scenario

Mean and 90th percentile intake and ingestion rate inputs were used for adults and children for the RME and CTE estimates, respectively. The rates used in this assessment are provided in Exhibit I.4-5, Exhibit I.4-6, and Exhibit I.4-7.

Values used for intake rates (in $g_{wet\ weight}/kg_{body\ weight}$-day) in exposure calculations for the farmer scenario are presented in Exhibit I.4-5 for exposed fruit, protected fruit, exposed vegetables, protected vegetables, root vegetables, beef, total dairy, pork, poultry, and eggs. As the units suggest, the intake rates for produce and animal products are normalized for body weight.

Exhibit I.4-5. Age-Specific Ingestion Rates for the FFC Pathway

Product	Age Group						Units [a]
	Child < 1 yr	Child 1-2 yrs	Child 3-5 yrs	Child 6-11 yrs	Child 12-19 yrs	Adult	
RME – 90th Percentile Consumer Ingestion Rate							
Farm Food Item (wet weight)							
Beef	NA	4.5	6.7	11.4	3.53	5.39	g/kg-day
Dairy [c]	NA	148	82	54.7	27.0	34.9	g/kg-day
Eggs [c]	NA	5.1	2.8	1.80	1.34	1.65	g/kg-day
Exposed Fruit	NA	3.7	5.4	6.98	3.41	5	g/kg-day
Exposed Vegetable	NA	10.7	3.5	3.22	2.35	6.01	g/kg-day
Pork [c]	NA	4.5	4.4	3.04	2.65	3.08	g/kg-day
Poultry [c]	NA	7.4	6.8	4.58	3.28	3.47	g/kg-day
Protected Fruit [c]	NA	53	36	24.1	16.2	15.1 [b]	g/kg-day
Protected Vegetable	NA	3.9	2.5	2.14	1.85	3.55	g/kg-day
Root Vegetable	NA	7.3	4.3	3.83	2.26	3.11	g/kg-day
Other							
Soil (dry weight)	NA	400[d]	400[d]	201 [e]	201 [e]	201 [e]	mg/day
Water (volume)	NA	654	834	980	1537	2224	mL/day
Fish (per individual) [f]	NA	3.24	4.79	6.9	8.95	17	g/day
Fish (per kg BW)	NA	0.26	0.26	0.22	0.14	0.24	g/kg-day
CTE – Mean Consumer Ingestion Rate							
Farm Food Item (wet weight)							
Beef	NA	1.49	2.21	3.77	1.72	2.63	g/kg-day
Dairy [c]	NA	67	37	24.8	10.9	17.1	g/kg-day
Eggs [c]	NA	2.5	1.4	0.86	0.61	0.90	g/kg-day
Exposed Fruit	NA	1.8	2.6	2.52	1.33	2.32	g/kg-day
Exposed Vegetable	NA	3.5	1.7	1.39	1.07	2.17	g/kg-day
Pork [c]	NA	2.2	2.1	1.49	1.17	1.30	g/kg-day
Poultry [c]	NA	3.6	3.4	2.13	1.59	1.54	g/kg-day
Protected Fruit [c]	NA	19	13	8.13	5.44	5.19 [b]	g/kg-day
Protected Vegetable	NA	2.5	1.3	1.1	0.78	1.3	g/kg-day
Root Vegetable	NA	2.5	1.3	1.32	0.94	1.39	g/kg-day
Other							
Soil (dry weight)[g]	NA	50	50	50	50	50	mg/day
Water (volume)	NA	294	380	447	697	1,098	mL/day
Fish (per individual) [f]	NA	1.37	2.03	2.71	3.90	6.90	g/day
Fish (per kg BW)	NA	0.11	0.11	0.09	0.06	0.10	g/kg-day

Source: EPA 1997a (Chapter 13), unless otherwise noted in table notes. See Attachment C-2 for additional information on sources. NA = not applicable.

[a] Ingestion rates for produce and animal products are normalized to consumer body weight. Ingestion rates for soil (mg/day) and water (mL/day) are not normalized to body weight.

[b] This value represents a weighted average for the 20-39 and 40-69 age groups.

[c] In many cases, ingestion rates for certain child age groups were not available in EPA 1997a. Intakes for these receptor groups were calculated using the methodology recommended in HHRAP (EPA 2005) (See Attachment C-2).

[d] This value represents an estimated "upper percentile" for children (EPA 1997a).

[e] Values represent soil ingestion rates for individuals consuming homegrown food products from Stanek et al. 1997.

[f] Adult fish ingestion rates are based on data from 1995-1996 and 1998 CSFII as summarized in EPA 2002b; child fish ingestion rates are based on the same survey data, but estimated by multiplying average two-day consumption rate for children who consumed fish on one or both days of the survey by the frequency of fish consumption (i.e., proportion of children that reported consuming fish out of all children sampled).

[g] Represents CTE from EPA 2008, Table 5-1 for children and recommended mean value for adults from EPA 1997a, Chapter 4, Table 4-23.

Therefore, BW values presented in Exhibit I.4-4 are not used in the farmer's ADD estimates in this assessment. For estimating adult exposures for the farmer scenario, intake rates provided in EPA's Exposure Factors Handbook associated with responses to the 1987-88 National Food Consumption Survey (NFCS; USDA 1980) questionnaire (households who garden, households who farm, or households who raise animals) were used instead of using the intake data associated with ages 20 to 39, 40 to 69, and 70 plus years. This is a more conservative estimate of intake, given that households that farm, garden, or raise animals will typically have a higher level of intake than those that do not participate in those activities (EPA 1998). Incidental soil ingestion rates are also presented in this table. For the RME exposure estimate, elevated soil ingestion rates based on data for individuals who consume only home-grown produce was used.

Fish Ingestion Rates for Angler Scenario

The daily fish consumption rates (in g_{fish}/day) used to estimate exposures for the angler scenario are presented in Exhibit 4-6. No site-specific fish consumption data were identified for the four water bodies included in this risk assessment or the Ravena, NY, region. Instead, the 90th percentile adult fish ingestion rates are based on data from 1995-1996 and 1998 CSFII as summarized in EPA 2002b; child fish ingestion rates are based on the same survey data, but estimated by multiplying average two-day consumption rate for children who consumed fish on one or both days of the survey by the frequency of fish consumption (i.e., proportion of children that reported consuming fish out of all children sampled). These values are discussed in detail in Attachment 2 of Appendix C.

The adult mean consumption rate for is 6.9 g/day; the 90th percentile consumption rate of fish is 17 g/day.

Exhibit I.4-6. Fish Ingestion Rates for all Scenarios

Product	Age Group						
	Units [g]	Child < 1 Year	Child 1-2 Years	Child 3-5 Years	Child 6-11 Years	Child 12-19 Years	Adult 20 - 70 years
Ingestion of Fish							
Mean	g/kg/day	NA [a]	1.37	2.03	2.71	3.9	6.9
90th Percentile	g/kg/day	NA [a]	3.24	4.79	6.9	8.95	17

Source: EPA 2002b

[a] Infants are assumed to consume only breast milk for one year.

Additionally, Alcove Reservoir was closed to public use as a fishing destination in 1970; therefore, it is unlikely that the conditions of the angler scenario (i.e., catching and consuming fish on a regular, long-term basis) would be met. However, exposure estimates were calculated for this lake as a check. Fish consumption advisories have been published by the State of New York for Kinderhook and Nassau Lakes (NY DOH 2007). Although the efficacy of fish advisories has been questioned by some investigators, especially for certain ethnic groups that are more likely to regularly consume self-caught fish,[7] it is possible that the specific advisories for these lakes may reduce the likelihood of regular, long-term consumption from these water bodies. Finally, the pond included in this risk assessment is probably too small to support a

[7] Studies have concluded that fish consumption advisories are often ineffective because anglers are not aware of advisories (Burger 2000), anglers do not have knowledge of the contaminants, health effects, exposure, or risks and therefore ignore the advisory (Jardine 2003; Burger et al. 1998; Beehler et al. 2003), and/or question the credibility of agencies posting the advisories (May and Burger 1996).

regularly-fished aquatic food web. These uncertainties are discussed in additional detail in the discussion of results in Section I-6.

Breast Milk Ingestion Rates for Nursing Infant Scenario

The breast milk ingestion rates (in kg/day) assumed for nursing infants are presented in Exhibit I.4-7. These values were calculated using the EPA-recommended upper percentile breast milk ingestion rate for infants nursing up to one year of 980 mL/day (EPA 1997b) and the density of human milk (1.03 g/mL).

Exhibit I.4-7. Breast Milk Ingestion Rates for Infants Less Than 1 Year of Age

Percentile	Breast Milk Ingestion Rate (kg/day)
Mean	0.70864
90th Percentile	1.0094

The mean ingestion rate was calculated by multiplying the EPA-recommended mean breast milk ingestion rate for infants nursing up to one year of 688 mL/day (EPA 1997b, Table 14-16) by the density of human milk (1.03 g/mL) and converting to kg/day, for a final value of 0.70864 kg/day. The 90th percentile ingestion rate was calculated by multiplying the EPA-recommended upper percentile breast milk ingestion rate for infants nursing up to one year of 980 mL/day (EPA 1997b, Table 14-16) by the density of human milk (1.03 g/mL) and converting to kg/day, for a final value of 1.0094 kg/day. As the units suggest, the ingestion rates for breast milk (kg/day) are not normalized for body weight.

Exposure Frequency

The exposure frequency (EF) represents the number of days per year that an individual consumes farm food items that are contaminated with PB-HAPs. For the CTE and RME evaluations, the exposure frequency was set to 365 days per year for all scenarios; a conservative estimate for constant exposure throughout the year.

Fraction Contaminated

The fraction contaminated (FC) represents the fraction of each food product that is homegrown (i.e., derived from the environment evaluated in this assessment). Individuals potentially exposed to PB-HAPs in this evaluation were assumed to derive all potentially contaminated foodstuffs from the modeled farm and watershed parcels. This means that for the CTE and RME evaluations, the FC default for all food products was set to 1 (including the value for nursing mothers used to calculate concentrations in breast milk).

Cooking Loss

Cooking loss (CL) inputs were included to simulate the amount of a food product that is not ingested due to loss during preparation, cooking, or post-cooking. These inputs are detailed in Appendix C-2 of the main report.

I-5 Dose-Response Assessment and Estimation of Human Health Risks

Estimates of exposure (as ADDs, discussed in Section I-4) for each PB-HAP and dose-response data were used to calculate excess lifetime cancer risk and non-cancer hazard for chronic exposures. Exhibit I.5-1 provides chemical dose-response data for the PB-HAPs evaluated in this assessment.

The carcinogenic potency slope factors (CSFs) for ingestion and non-cancer oral reference doses (RfDs) for chronic exposures for the PB-HAP included in this assessment are provided in Exhibit I.5-1. The dose-response values were obtained from tabulated dose-response data that OAQPS uses for risk assessments of hazardous air pollutants (EPA 2007), and a detailed discussion of these values is provided in Appendix C. In general, OAQPS chose these values based on the following hierarchy of sources: EPA's Integrated Risk Information System (IRIS); the Centers for Disease Control's Agency for Toxic Substances and Disease Registry (ATSDR); and the California Environmental Protection Agency (CalEPA).

Exhibit I.5-1. Dose-response Values for PB-HAPs Addressed in this Assessment

Chemical	CAS No.	Cancer Slope Factor $\left(\frac{mg}{kg-day}\right)^{-1}$	Source	Reference Dose $\left(\frac{mg}{kg-day}\right)$	Source
Inorganics					
Cadmium compounds in food	7440439	not available		1.0E-03	IRIS
Cadmium compounds in water	7440439	not available		5.0E-04	IRIS
Mercury (elemental)	7439976	NA		not available	
Mercuric chloride	7487947	not available		3.0E-04	IRIS
Methyl mercury (MeHg)	22967926	not available		1.0E-04	IRIS
Organics					
Benzo(a)pyrene	50328	1.0E+01	EPA OAQPS[1]	not available	
2,3,7,8-TCDD	1746016	1.5E+05	EPA ORD	1.0E-09	ATSDR

ATSDR = Agency for Toxic Substances and Disease Registry
EPA OAQPS = EPA's Office of Air Quality Planning and Standards
EPA ORD = EPA's Office of Research and Development
IRIS = Integrated Risk Information System
NA = not applicable

[1] The method to assign oral cancer slope factors for polycyclic organic matter (POM) is the same as was used in the 1999 National Air Toxics Assessment (EPA 1999b). A complete description of the methodology is available at: http://www.epa.gov/ttn/atw/nata1999/99pdfs/pomapproachjan.pdf.

Sources: Values presented here are consistent with those defined by OAQPS for evaluation of HAPs (EPA 2007). Sources listed in this table are the original references cited by EPA. For more information and the original references that provide the derivation of these dose-response values, refer to EPA 2007.

I-6 Results and Discussion

I-6.1 Ravena Human Health Multipathway Risk Assessment Results

The results of the human health risk assessment are presented in this section. Section I-6.2 focuses on the results for 2,3,7,8-TCDD (as a representative of total dioxins), Section I-6.3

focuses on the results for mercury, and Section I-6.4 present the results associated with an alternative modeling scenario in which a fish harvester is introduced into Ravena Pond.

For both dioxin and mercury, the concentrations and human health risks estimated in this assessment are compared to analogous outputs estimated using the hypothetical multipathway screening scenario developed for RTR. To accomplish this comparison, the Ravena emission rates were modeled in the TRIM.FaTE screening scenario layout that is used to derive the RTR *de minimis* levels for screening. This comparison helps to illustrate the level of conservatism associated with the screening scenario and provides additional context for the results estimated for this site-specific risk assessment. Throughout the multipathway HHRA discussion, the results of modeling the Ravena emissions in the screening scenario are labeled "Screening Scenario."

In general, the presentation of results here favors those calculated using reasonable maximum exposure (RME) ingestion rates that are unlikely to occur but are still within the bounds of what is possible. Exposures and risks calculated using more typical, central tendency exposure (CTE) ingestion rates for these scenarios are also presented for comparison in some cases. Note that most graphs display results plotted on a logarithmic scale.

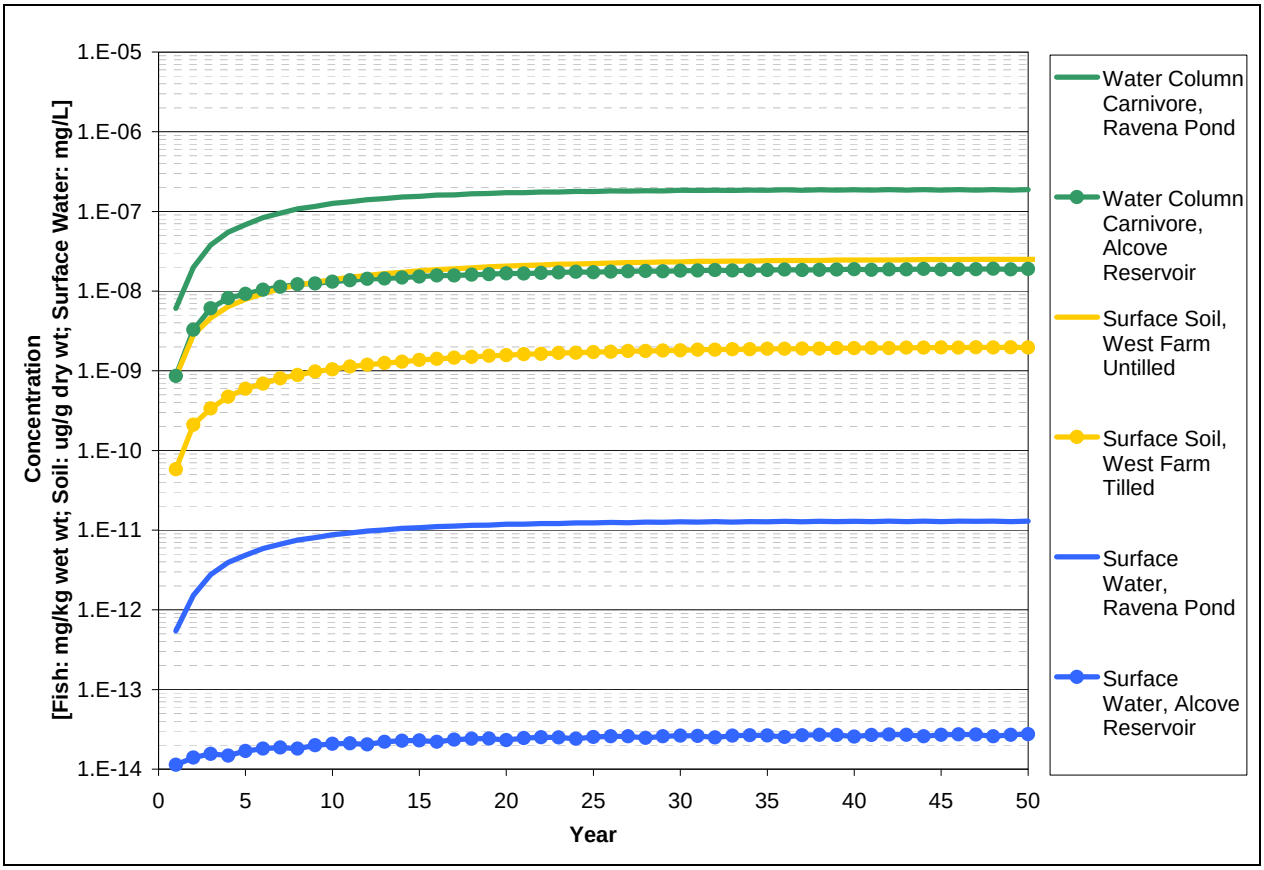
I-6.2 2,3,7,8-TCDD

The Ravena site-specific TRIM.FaTE scenario was run for 50 years using two emission rates for 2,3,7,8-TCDD and assuming constant emissions for the duration of the simulation. The first emission rate was calculated using a mean emission factor, and the second rate was calculated based on the 95-percent upper confidence limit (UCL) of the dioxin emission factor, to provide an upper bound risk estimate that takes into account the uncertainty regarding the emissions estimate. A discussion of the derivation of the emission factors is presented in Appendix F. Media concentrations and risks were estimated for both emission rates; however, results presented in this section are primarily calculated using the 95-percent UCL emission rate.

I-6.2.1 *Estimated Media Concentrations*

Exhibit I.6-2 presents a time series of dioxin concentrations in selected compartments modeled by TRIM.FaTE using the 95-percent UCL emission rate. Included here are annual average results for water column carnivores in the Ravena Pond and Alcove Reservoir compartments, for surface water in the Ravena Pond and the Alcove Reservoir compartments, and surface soil in the West Farm compartment (the layout of the Ravena TRIM.FaTE scenario is previously described in Section I-3.4).

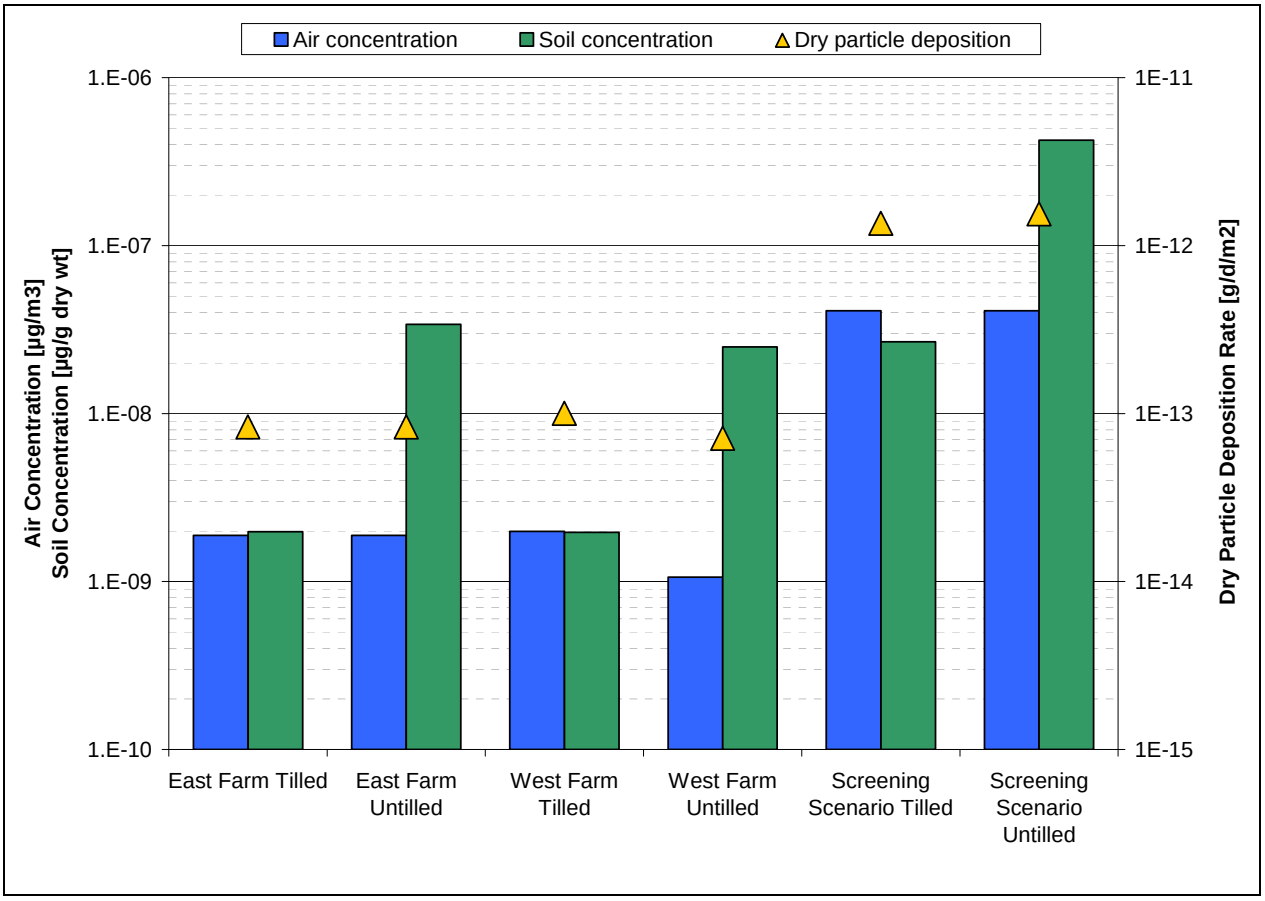
Exhibit I.6-2. 2,3,7,8-TCDD Media Concentration Time Series Using 95% UCL Dioxin Emission Rate



As can be seen in Exhibit I.6-2, concentrations typically increase rapidly over the first fifteen years and then level off, increasing more slowly over the remainder of the model duration as the concentration approached a steady-state. For the current analysis, risks were calculated based on modeled environmental concentrations after fifty years of continuous emissions. This fifty year period accounts for future impacts from long-term emissions at the assumed rate for the modeled source.

Annually-averaged air and surface soil concentrations from the 50th model year for each farming parcel in the Ravena scenario are shown in Exhibit I.6-3 along with the dry particle deposition rates to these locations from the final year. Concentrations presented here were modeled using the 95-percent UCL emission rate, and these concentrations were later used to estimate concentrations in farm media assumed to be ingested in this scenario. For comparison, annually-averaged concentrations and deposition rates during the final year of a 50 year run of the generic RTR screening scenario (run with the same Ravena dioxin emissions rate as the case study evaluation) are also shown in this chart.

Exhibit I.6-3. 2,3,7,8-TCDD Air and Surface Soil Concentrations and Dry Particle Deposition Rates During the 50th Model Year Using 95-Percent UCL Emission Rate

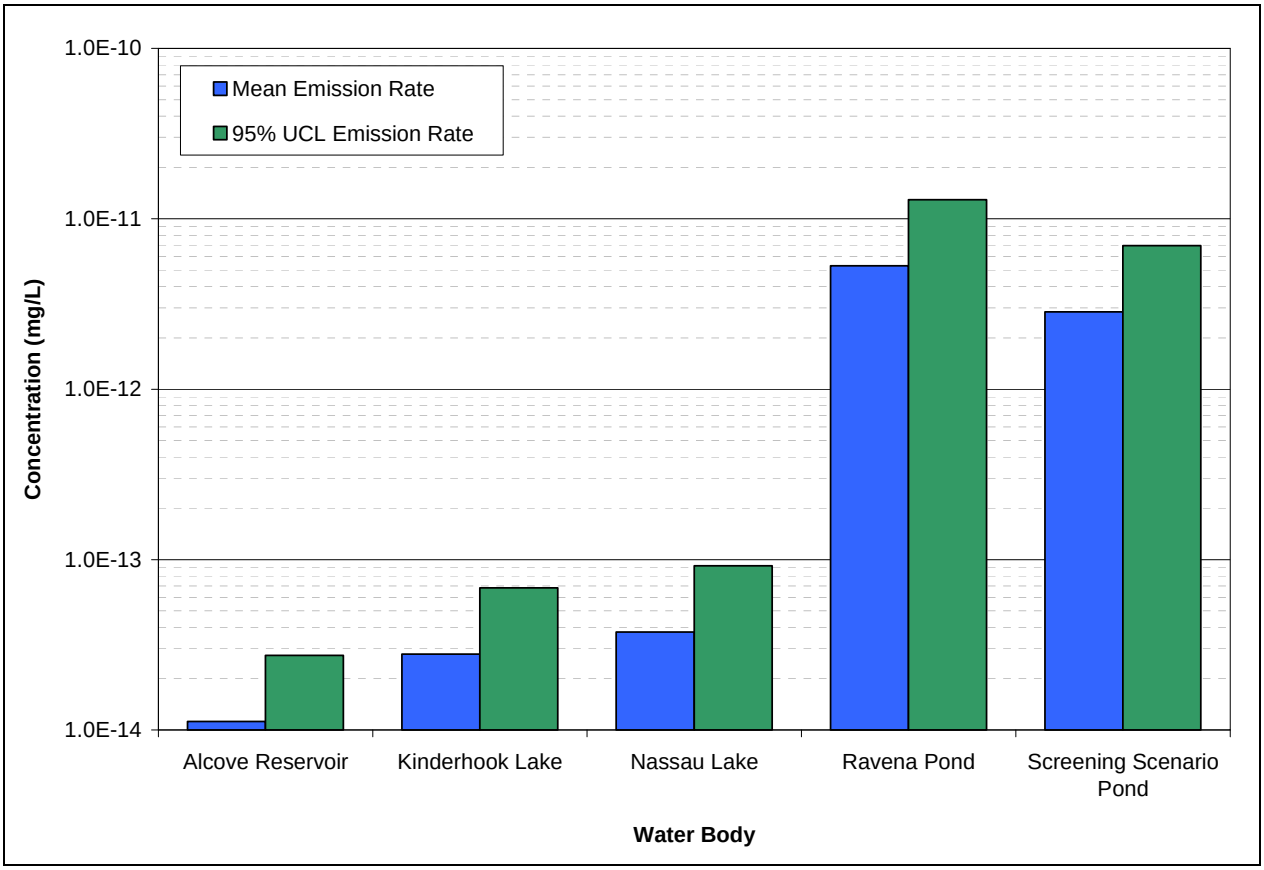


In all cases, 2,3,7,8-TCDD surface soil concentrations are higher in the untilled plots than the tilled plots, with the eastern untilled compartment having the higher concentration of the two Ravena farm compartments. The chemical mass in the tilled and untilled compartments at a given location is roughly comparable, but the volume in which the tilled chemical mass is mixed is larger, resulting in a lower overall concentration. Comparison of the Ravena and screening scenario soil concentrations for one type of soil compartment (i.e., either tilled or untilled) illustrates a decrease of about an order of magnitude when site-specific characteristics (including meteorology and proximity of the farm parcel) are included in the model.

Air concentrations are similar over the Ravena farm compartments. Unlike surface soil concentrations, dry deposition values tend to be slightly higher in the western tilled soil than in the eastern tilled soil. This difference likely reflects patterns of wind direction and precipitation in the data set used for this model scenario.

Annually-averaged surface water concentrations estimated for the 50th year of the simulation for each water body in the Ravena scenario are shown in Exhibit I.6-4. The results of modeling the Ravena dioxin emission rates in the RTR screening scenario are also included for comparison.

Exhibit I.6-4. 2,3,7,8-TCDD Surface Water Concentrations During the 50th Model Year Using Mean and 95-Percent UCL Emission Rates



Dioxin concentrations in surface water were estimated to be highest in the Ravena Pond, where concentrations were slightly higher in the screening scenario pond for a given emission rate. The lowest concentrations were estimated in Alcove Reservoir. This result is reasonable given that Alcove Reservoir is the farthest from the source of the four water bodies modeled in the Ravena scenario.

Fish Species Modeled by the TRIM.FaTE Fish Compartments

Water Column Herbivore: Black crappie, common carp, fantail darter, golden shiner, and young of the year

Benthic Omnivore: Bullhead and sunfish

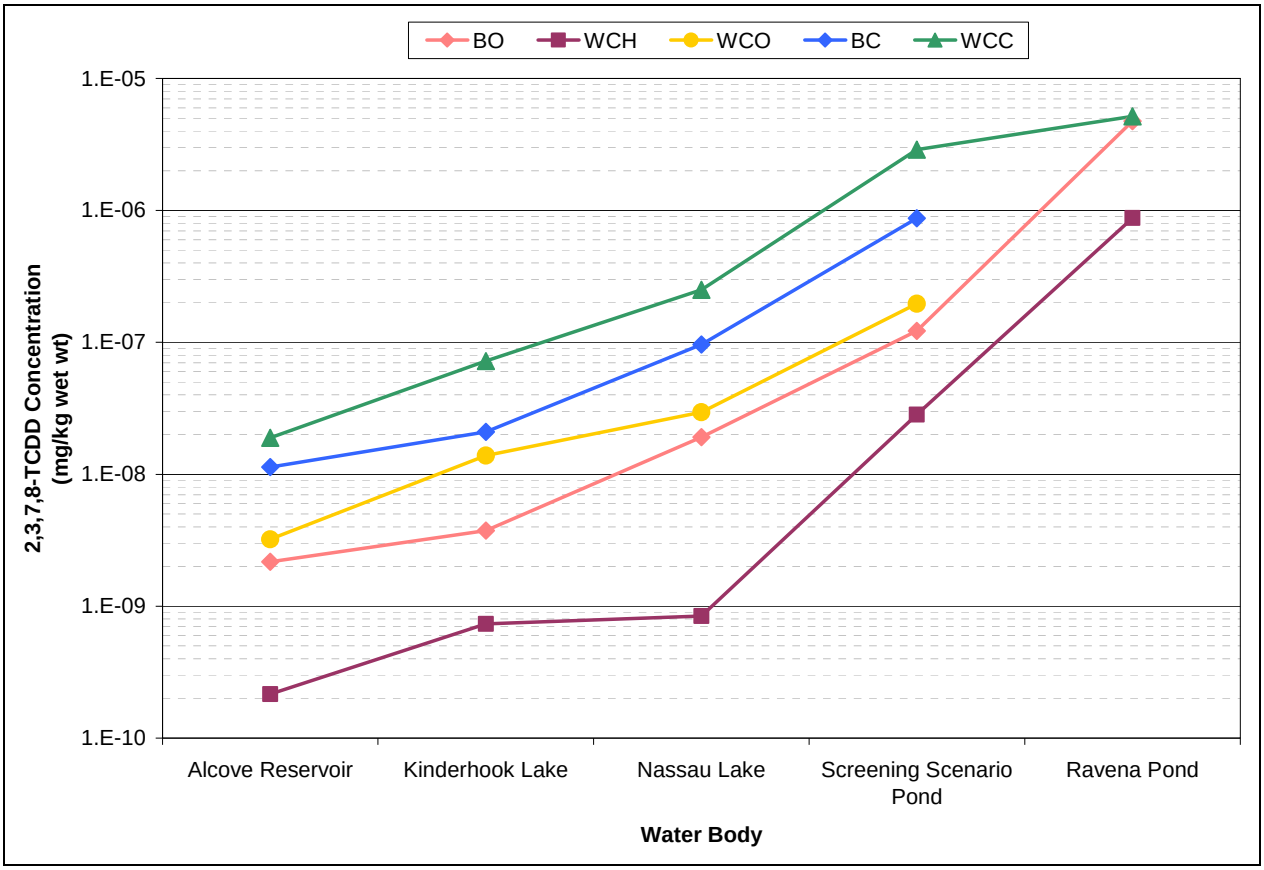
Water Column Omnivore: Bluegill, pumpkinseed, redbreast sunfish, rock bass, smallmouth bass, white perch, white sucker, and yellow perch

Benthic Carnivore: American eel

Water Column Carnivore: Chain pickerel, largemouth bass, northern pike, tiger musky, and walleye

Concentrations of 2,3,7,8-TCDD for each fish compartment for all water bodies containing fish in the Ravena and RTR screening scenarios are presented in Exhibit I.6-5. TRIM.FaTE includes compartments for benthic invertebrates, benthic omnivores, benthic carnivores, water column herbivores, water column omnivores, and water column carnivores. The accompanying text box indicates fish species assumed to be present in the Ravena area water bodies that are represented by TRIM.FaTE fish compartment types. A complete description of the development of the aquatic food webs for the Ravena scenario is included in Attachment I-1.

Exhibit I.6-5. 2,3,7,8-TCDD Concentration in Fish Species During the 50th Model Year Using the 95-Percent UCL Emission Rate



Results by location correspond generally to surface water concentration trends. Water column carnivores and benthic carnivores were estimated to have the highest concentrations of 2,3,7,8-TCDD in all water bodies except the Ravena Pond. The Ravena Pond does not contain benthic carnivores, and so water column carnivores and benthic omnivores, the two largest types of fish in this water body, had the highest concentrations.

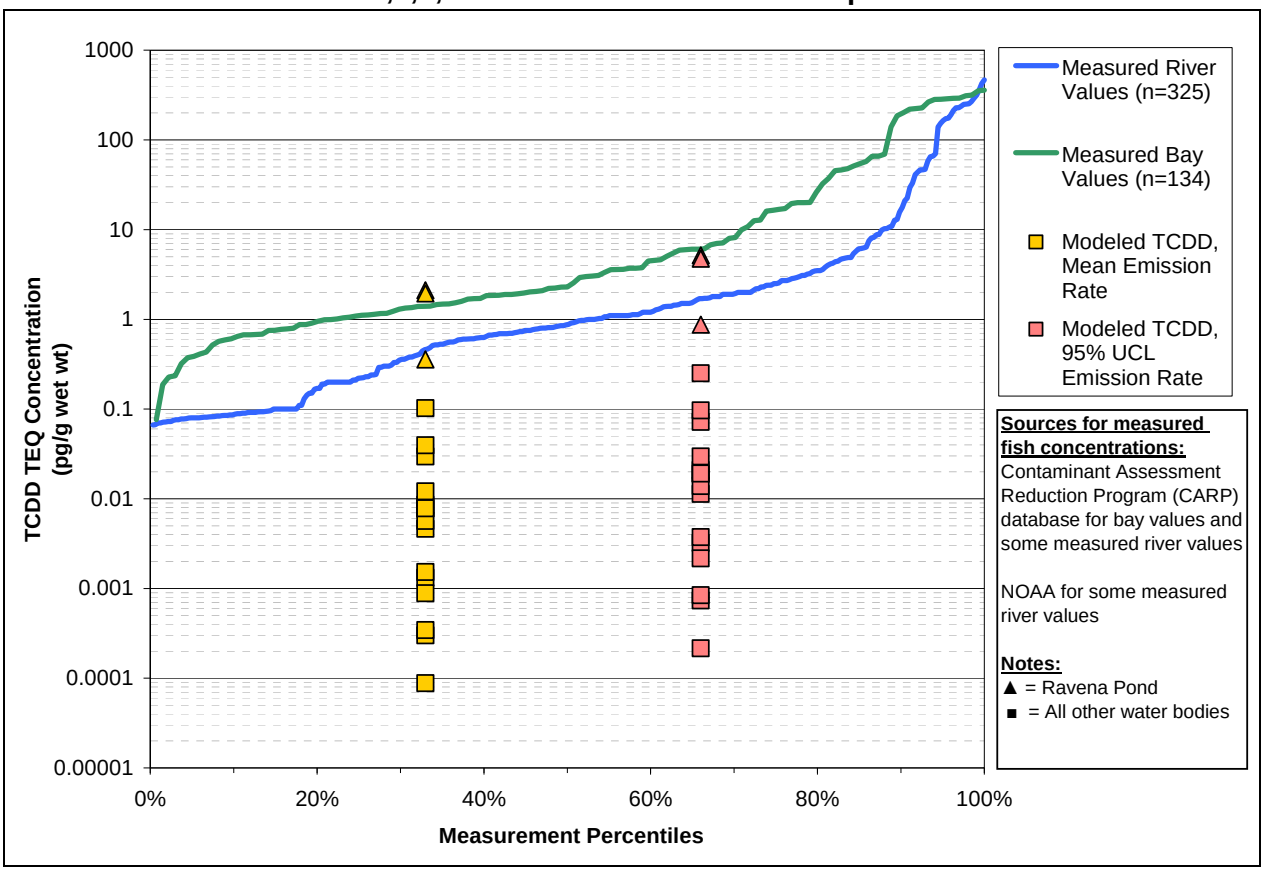
Fish that are higher on the food chain (carnivores and omnivores, compared to herbivores and invertebrates) tend to have higher concentrations, reflecting the biomagnification that occurs for dioxins in an aquatic food chain. The carnivores and omnivores in the Ravena Pond and the Screening Scenario Pond have the highest concentrations, consistent with the surface water concentration trends.

I-6.2.2 *Comparison of Modeled Surface Water Concentrations to Measured Values*

In Exhibit I.6-6 estimated fish concentrations for the water bodies modeled in the Ravena screening scenario are compared to measured values for the Hudson River and nearby bays (HSF 2007) . Note that the concentrations are plotted on a logarithmic scale. For the measured values, the environmental data were ranked (separately for the river and for bays) to create a distribution. The modeled concentrations for the Ravena case study corresponding to both the mean and 95 percent UCL emission factors are presented as two separate series and do not correspond to the percentiles on the x-axis.

Fish concentrations measured in the Hudson River and Bay ranged from 0.08 to 600 pg/g wet weight. By comparison, in the modeled Ravena water bodies, estimated concentrations in fish ranged from 9E-05 to 6 pg/g wet weight. This outcome seems reasonable because the model includes only a single source of chemical emissions to the air, while the reported values reflect all local and regional sources of dioxins, existing background concentrations of dioxins from historical air sources, and any contributions from non-air sources (likely including historical PCB contamination introduced to the Hudson River).

Exhibit I.6-6. Modeled 2,3,7,8-TCDD Concentrations Compared to Measured Values



I-6.2.3 *2,3,7,8-TCDD Risk Assessment Results*

The annually-averaged concentrations for the 50th year estimated by TRIM.FaTE were used to calculate lifetime individual cancer risks and chronic non-cancer hazard quotients (HQs) based on assumptions of ingestion rates for fish, dairy, beef, and other foods. Ingestion rates were assumed to vary by age, and exposures were calculated for five age groups. Because dioxin exposure can occur via consumption breast milk by nursing infants, non-cancer hazards in this subpopulation were also evaluated.

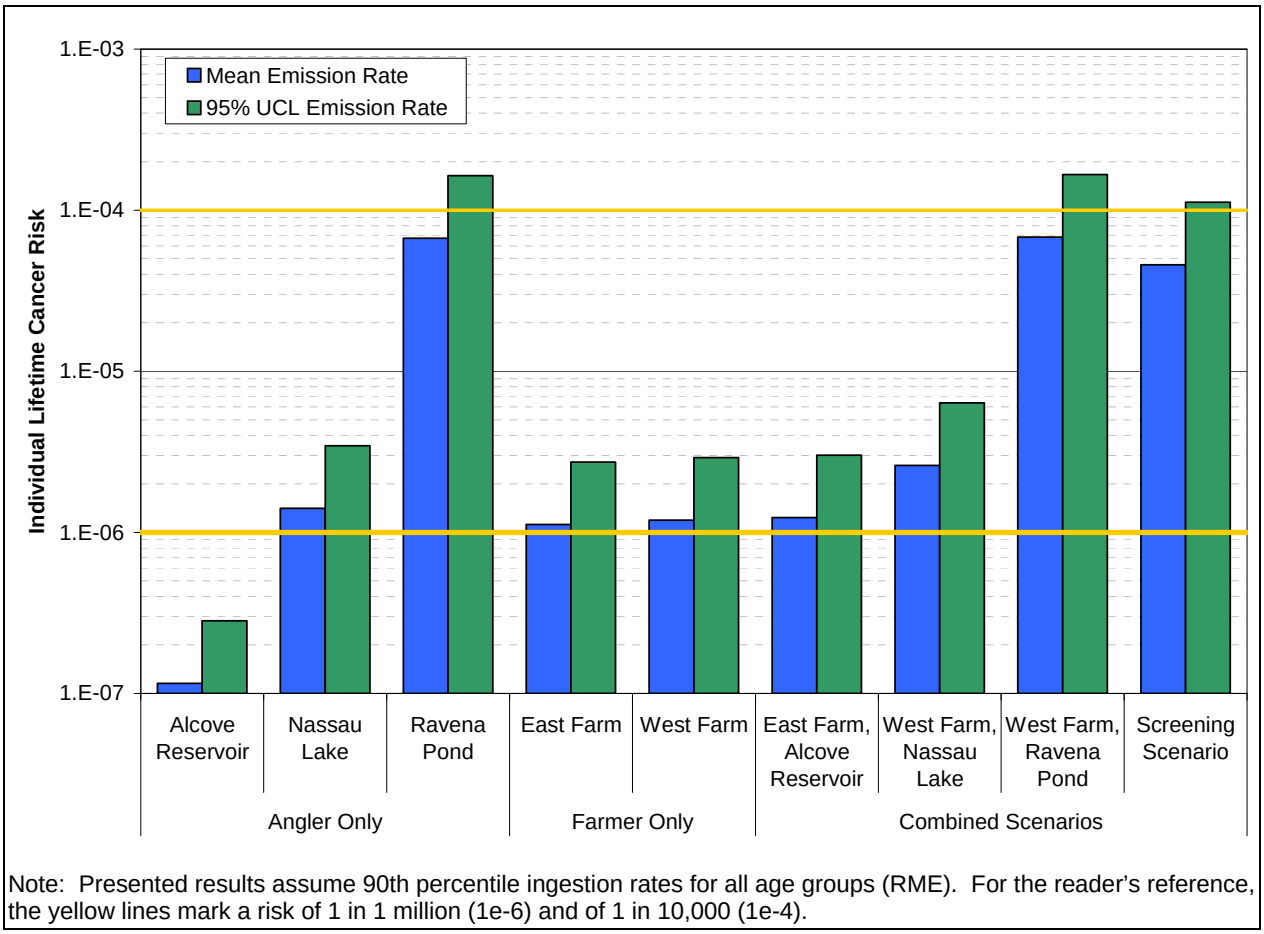
Different combinations and variations of results for the two main exposure scenarios are presented, including angler-only (ingestion only of fish), farmer-only (ingestion only of dairy, beef, and other), and combined scenarios created for each unique combination of farm parcel and water body. For the combined scenarios, the scenario location combination with the lowest risks (the East Farm and the Alcove Reservoir), the highest risks (the West Farm and the Ravena Pond), and the second highest risks (the West Farm and Nassau Lake) are typically

presented. The West Farm/Nassau Lake scenario is expected to provide a more realistic high-end estimate than the West farm/Ravena Pond combination.

I-6.2.3.1 2,3,7,8-TCDD Estimated Lifetime Cancer Risks

Exhibit I.6-7 below presents the individual lifetime cancer risks for modeled scenarios. The one-in-a-million and 100-in-a-million cancer risk thresholds are highlighted for reference. The results were calculated using the TRIM.FaTE results from both the mean and 95-percent UCL emission rates, and use the 90th percentile RME ingestion rates for all age groups. Only the angler exposure scenario calculated with fish consumption from Alcove Reservoir yields a lifetime risk of less than one in a million.

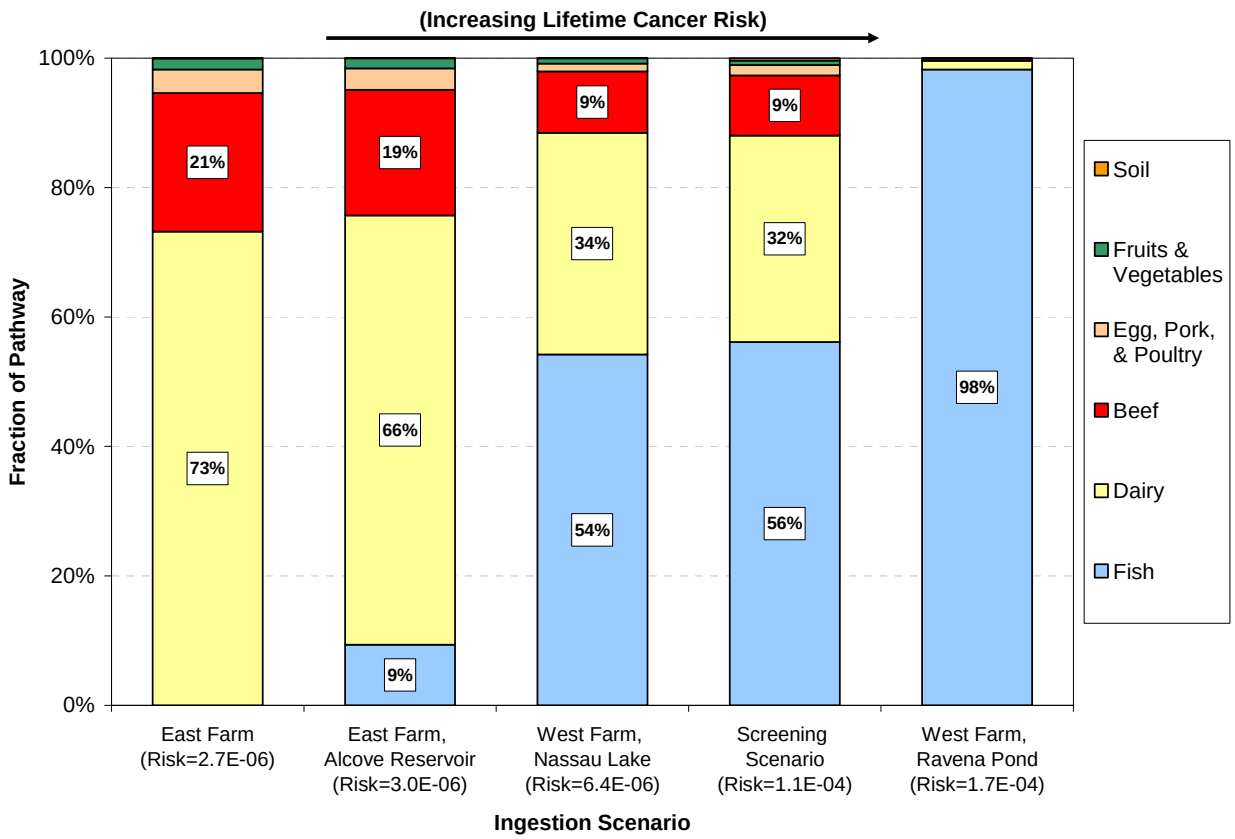
Exhibit I.6-7. 2,3,7,8-TCDD Individual Lifetime Cancer Risks



Note: Presented results assume 90th percentile ingestion rates for all age groups (RME). For the reader's reference, the yellow lines mark a risk of 1 in 1 million (1e-6) and of 1 in 10,000 (1e-4).

Exhibit I.6-8 presents the pathway contribution to dioxin exposures for all combined farmer/angler scenarios, including results from the screening scenario. In this chart, exposure scenarios are presented from left to right in order of increasing cancer risk. For the scenario combination at the Ravena site with the second highest exposures (i.e., consumption of farm products from the West Farm location and consumption of fish caught in Nassau Lake), consumption of fish is the dominant exposure pathway contributing to overall risk. Conversely, for the lowest exposure Ravena scenario (i.e., consumption of foodstuffs from East Farm and Alcove Reservoir), consumption of dairy contributes more to overall risk than exposure to dioxin via consumption of fish.

Exhibit I.6-8. Pathway Contributions to 2,3,7,8-TCDD Individual Lifetime Cancer Risks



Note: Presented results assume fish harvesting from the Ravena Pond, 95-percent UCL emission rate, and 90th percentile ingestion rates for all age groups (RME).

Exhibit I.6-9 presents lifetime cancer risks for each combination of emission rate, exposure scenario, and ingestion rate (some of these results are included in the two preceding charts). Estimated individual lifetime cancer risks that exceed one in a million are highlighted in blue. Only the CTE ingestion rate combined with the mean emission rate for the combined scenarios using Kinderhook Lake or Alcove Reservoir and either farm parcel produced estimated cancer risks below the one-in-a-million threshold.

Exhibit I.6-9. 2,3,7,8-TCDD Individual Lifetime Cancer Risks

Scenario Type	Water Body	Farm Parcel	95% UCL Emission Rate		Mean Emission Rate		Ratios	
			RME Ingestion Rate	CTE Ingestion Rate	RME Ingestion Rate	CTE Ingestion Rate	Ingestion RME : CTE	Emissions 95% : Mean
Screening	Screening	Screening	**1.1E-04**	**4.8E-05**	**4.6E-05**	**2.0E-05**	2.3	2.4
Combined Farmer and Angler	Ravena Pond	West	**1.7E-04**	**6.8E-05**	**6.8E-05**	**2.8E-05**	2.4	2.4
		East	**1.7E-04**	**6.8E-05**	**6.8E-05**	**2.8E-05**	2.4	2.4
	Nassau Lake	West	**6.4E-06**	**2.8E-06**	**2.6E-06**	**1.1E-06**	2.3	2.4
		East	**6.2E-06**	**2.7E-06**	**2.5E-06**	**1.1E-06**	2.4	2.4
	Kinderhook Lake	West	**4.0E-06**	**1.8E-06**	**1.6E-06**	7.4E-07	2.2	2.4
		East	**3.8E-06**	**1.7E-06**	**1.6E-06**	7.1E-07	2.2	2.4
	Alcove Reservoir	West	**3.2E-06**	**1.5E-06**	**1.3E-06**	6.0E-07	2.3	2.4
		East	**3.0E-06**	**1.4E-06**	**1.2E-06**	5.7E-07	2.4	2.4
Farmer Only	None	West	**2.9E-06**	**1.4E-06**	**1.2E-06**	5.5E-07	2.2	2.4
		East	**2.7E-06**	**1.3E-06**	**1.1E-06**	5.2E-07	2.2	2.4
Angler Only	Ravena Pond	None	**1.6E-04**	**6.7E-05**	**6.7E-05**	**2.7E-05**	2.4	2.4
	Nassau Lake	None	**3.4E-06**	**1.4E-06**	**1.4E-06**	5.8E-07	2.4	2.4
	Kinderhook Lake	None	**1.1E-06**	4.5E-07	4.6E-07	1.9E-07	2.4	2.4
	Alcove Reservoir	None	2.8E-07	1.2E-07	1.2E-07	4.7E-08	2.4	2.4
Water Ingestion Only	Alcove Reservoir	None	1.3E-13	6.2E-14	5.2E-14	2.5E-14	2.1	2.4

Cancer risk is between 2- and 2.5-times greater when assuming RME ingestion instead of CTE ingestion rates. In all cases, using the 95-percent UCL emission rate resulted in cancer risks approximately 2.4 times greater than using the mean emission rate. Because the emission rates are run through an identical TRIM.FaTE screening scenario and farm-food chain, it is reasonable that risks are exactly proportional to the emission rate used. The 95th emission factor for the modeled facility is 2.4 times greater than the mean emission factor (3.23 g/yr vs. 1.32 g/yr, respectively), and this relationship is maintained throughout the scenario.

In all cases estimated cancer risks are slightly higher when products from the western farm parcel are consumed rather than the eastern farm parcel. In the special cases where only consumption of farm food products is considered, risks are approximately 7 percent higher using the western farm than the eastern farm. When only fish consumption is considered, risks are more than 400 times greater when fish are consumed from the Ravena Pond than when fish are consumed from Alcove Reservoir. Risks are approximately 12 times greater when fish are consumed only from Nassau Lake compared to estimated risks associated with fish consumption from only Alcove Reservoir.

I-6.2.3.2 2,3,7,8-TCDD Chronic Non-Cancer Hazard Quotients

Chronic non-cancer hazard quotients (HQs) for dioxins were also estimated using modeled TRIM.FaTE environmental concentrations and exposure estimates. Results are presented for all age groups in Exhibit I.6-10 corresponding to 95-percent UCL emission rates, RME ingestion rates, individual and combined farmer and angler scenarios, both farm locations, and three of the water bodies. The highest HQs were estimated for children aged 1 to 2, and the lowest HQs were estimated for adolescents aged 12 to 19, with differences between age groups dictated by age-specific ingestion rates of farm food products and fish.

2,3,7,8-TCDD HQs for Ravena scenarios not including the pond are generally at least an order of magnitude lower than 1. When the Ravena emission rates were modeled in the screening scenario layout the calculated HQs for Child 1-2 and Child 3-5 were both greater than 1. The application of site-specific parameters reduced these HQs and illustrates the conservative nature of the screening scenario. If fish from the Ravena Pond are assumed to be consumed, the HQs were always estimated to be above 1.

The difference in HQs between the CTE and RME (i.e., mean vs. 90^{th} percentile ingestion rates; results not shown) is, on average, a factor of 2. The spread between the lowest exposure scenario (Alcove Reservoir with the East Farm) and the second highest exposure scenario (Nassau Lake and West Farm) is higher when the 90th percentile ingestion rates are used, with the difference being approximately a factor of 2. When mean ingestion rates are used, there is a factor of 2 difference in the HQs for these two scenarios. As in the case with cancer risks, there is a factor of 2.4 difference in HQs when the RME ingestion rates are used instead of the CTE rates.

Tables of all results for the calculated HQs for 2,3,7,8-TCDD are located in Attachment I-2.

Exhibit I.6-10. 2,3,7,8-TCDD Chronic Non-cancer Hazard Quotients (95th Percentile UCL Emission Factor, RME Ingestion Rates)

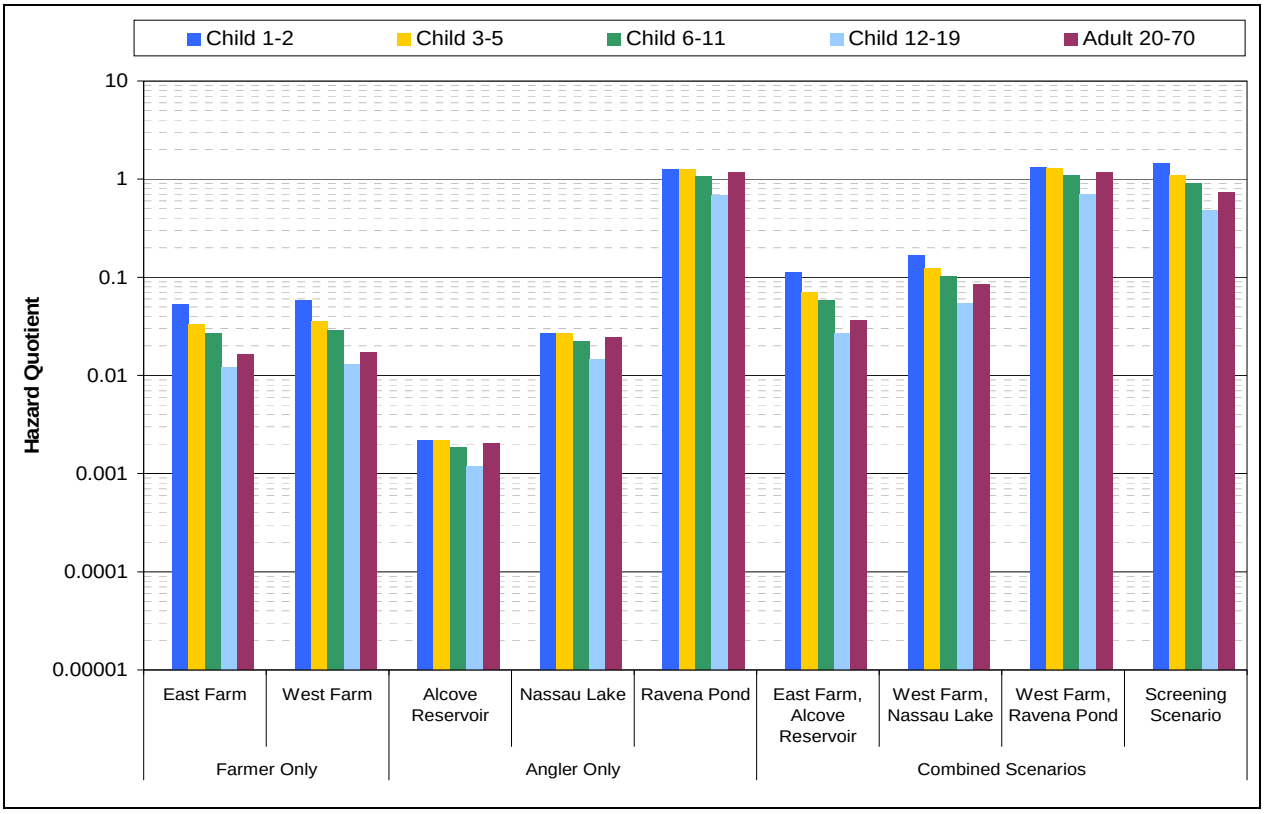
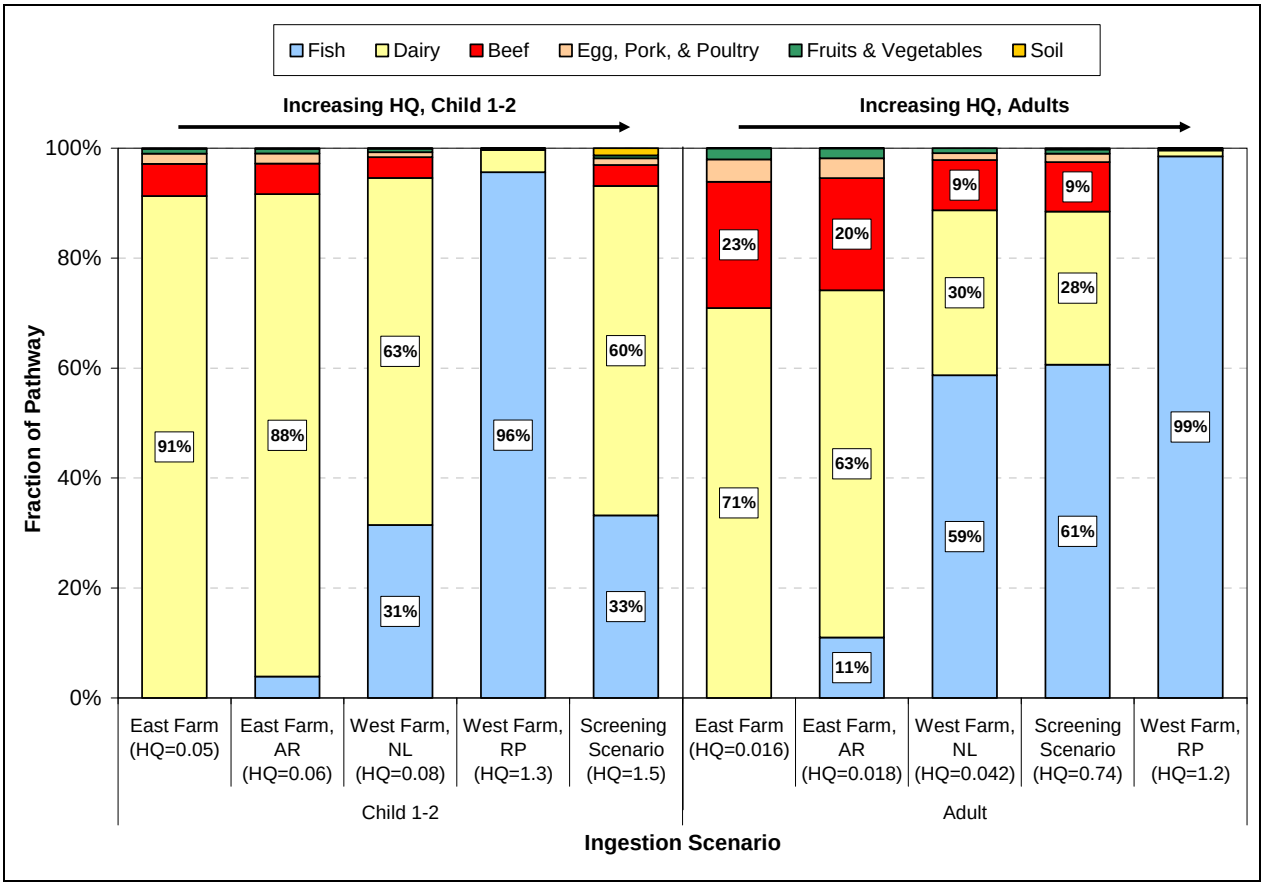


Exhibit I.6-11 presents the contribution of each ingestion exposure pathway to the overall HQ for children aged 1-2 and adults. The scenarios examined in the Ravena case study are displayed in order of increasing HQ, with the location resulting in the lowest HQ displayed at the far left and the location combination resulting in the highest HQ at the far right.

Modeled fish concentrations in Nassau Lake are approximately an order of magnitude greater than those in Alcove Reservoir, and, for the West Farm/Nassau Lake scenario, fish consumption is the risk-driving pathway for all age groups. The influence of the fish consumption pathway on the HQ increases with age while that of dairy consumption decreases with age, reflecting the relative magnitude of the ingestion rate for each food type for different age groups (*i.e.*, children are assumed to consume more dairy products per kg body weight, while adults are assumed to consume more fish per kg body weight). For the East Farm/Alcove Reservoir scenario, dairy consumption is the primary risk driver. This pathway has the most influence on exposures in the youngest age group, with the influence of the dairy pathway decreasing with age.

Exhibit I.6-11. Pathway Contributions to Divalent Mercury Chronic Non-Cancer Hazard Quotients (95th Percentile UCL Emission Factor, RME Ingestion Rates)



Ar=Alcove Reservoir; NL=Nassau Lake; RP=Ravena Pond

I-6.2.3.3 2,3,7,8-TCDD Risks and Hazard Quotients Resulting from Dermal Exposure

Non-inhalation exposure to PB-HAPs can occur by way of the dermal pathway through contact with PB-HAP-contaminated soil and water. However, as discussed in Appendix C of the main report, dermal absorption of chemicals that are originally airborne is expected to be a relatively minor pathway of exposure compared to other exposure pathways. The dermal cancer risks and non-cancer HQs for 2,3,7,8-TCDD were calculated using the methodology discussed in Appendix C using soil from each of the farm areas and water from Alcove Reservoir (assumed to be the source of bathing water). The highest combined cancer risks occurred when the calculations used soil concentrations from the untilled East Farm compartment. Exhibit I.6-12 below summarizes these risks assuming both the mean and 95th percentile emission factors. Lifetime risks using the emission rate in the screening scenario are also shown for comparison. A table providing risks and HQs for all age groups and farm compartments considered is provided in Attachment I-2.

The maximum lifetime risk using the site-specific scenario with Alcove Reservoir is 4.9E-9, and the ingestion risks are at least 200 times the dermal risks. The dermal calculations are highly conservative and they are compared to the least conservative ingestion scenario (that is, the mean ingestion rather than the 90th percentile ingestion). Because the dermal lifetime risks remain over two orders of magnitude lower than the ingestion lifetime risks, dermal exposure is

not a primary exposure pathway and does not strongly contribute to 2,3,7,8-TCDD lifetime risk at this site.

Exhibit I.6-12. Estimated Lifetime Cancer Risks Associated with Modeled Dermal Exposure to 2,3,7,8-TCDD

Emission Rate	Farm	Water body	Lifetime Risk			
			Soil	Water	Soil and Water	Ratio of Ingestion to Dermal [a]
95% UCL Emission Rate	East Farm - Untilled	Alcove Reservoir	2.2E-09	2.7E-09	4.9E-09	>280
	Screening Scenario	Screening Scenario	3.9E-08	6.9E-07	7.3E-07	>60
Mean Emission Rate	East Farm - Untilled	Alcove Reservoir	9.0E-10	1.1E-09	2.0E-09	>590
	Screening Scenario	Screening Scenario	1.6E-08	2.8E-07	3.0E-07	>150

[a] Ratio compares total dermal HQ calculated based on RME parameters to total ingestion HQ based on CTE ingestion rates (i.e., ratio of highest dermal HQ to lowest ingestion HQ).

Ingestion hazard quotients calculated using the mean emission rate and the mean ingestion rate (*i.e.*, the lowest estimated ingestion hazard quotients) are between approximately 50 and 150 times greater for than the highest estimated dermal hazard quotients (i.e., the HQs calculated based on the 95-percent UCL emission rate). These results are included in Attachment 2.

I-6.2.3.4 Chronic Non-Cancer Hazard Quotients in Nursing Infants

Dioxin compounds ingested by a lactating mother can partition into breast milk and be passed to a nursing infant. To evaluate the potential for hazard to a nursing infant at this facility, HQs were estimated for an infant assumed to ingest breast milk from a woman exposed via consumption of local farm products and fish during the duration of breast feeding and for nine years prior to the birth of the child (see Attachment C-2). The infant's average daily ingestion dose was calculated using the mother's average daily dose, partition factors and other parameters that estimate the transfer of dioxins from the mother to breast milk, and exposure factors for the nursing infant (See Attachment C-2, Section 3.4). This dose was then compared to the same reference dose as the adult (rather than an infant-specific one). Estimated HQs for the mother and child for the different emission factors, ingestion rates, farms, and water bodies are shown below in Exhibit I.6-13. Hazard quotients greater than one are shown in boldface type.

The infant's hazard quotients tend to be higher than the mother's hazard quotients by a factor of 22 for all cases examined. The infant hazard quotients exceed one when consumption of fish by the mother from Ravena Pond and the Screening Pond is assumed for all emission factors, ingestion rates, and farm combinations considered. The hazard quotients shown do not include the modeled fish harvesting from the Ravena Pond (discussed in Section I-6.4). If fish harvesting is modeled and exposures are calculated based on those concentrations, the hazard quotients for the infant and mother decrease by 70-73%, but the infant hazard quotients remain above one. For the more probable consumptions of fish from Alcove, Kinderhook, or Nassau Lakes, the infant hazard quotient is lower than one for the highest emission factors and ingestion rates.

Exhibit I.6-13. Mother and Infant non-cancer Hazard Quotients for 2,3,7,8-TCDD

TCDD Emission Factor	Ingestion Rate	Farm	Water body	Mother HQ	Infant HQ
95th Percentile UCL	90th Percentile	East	Alcove Reservoir	0.02	0.45
			Kinderhook Lake	0.03	0.58
			Nassau Lake	0.04	0.95
			Ravena Pond	**1.18**	**26.32**
		West	Alcove Reservoir	0.02	0.48
			Kinderhook Lake	0.03	0.61
			Nassau Lake	0.04	0.98
			Ravena Pond	**1.18**	**26.34**
		Screening	Screening	0.77	**17.32**
	Mean	East	Alcove Reservoir	0.01	0.21
			Kinderhook Lake	0.01	0.26
			Nassau Lake	0.02	0.41
			Ravena Pond	0.48	**10.77**
		West	Alcove Reservoir	0.01	0.22
			Kinderhook Lake	0.01	0.27
			Nassau Lake	0.02	0.43
			Ravena Pond	0.48	**10.78**
		Screening	Screening	0.33	**7.47**
Mean	90th Percentile	East	Alcove Reservoir	0.01	0.19
			Kinderhook Lake	0.01	0.24
			Nassau Lake	0.02	0.39
			Ravena Pond	0.48	**10.77**
		West	Alcove Reservoir	0.01	0.20
			Kinderhook Lake	0.01	0.25
			Nassau Lake	0.02	0.40
			Ravena Pond	0.48	**10.78**
		Screening	Screening	0.32	**7.08**
	Mean	East	Alcove Reservoir	0.00	0.09
			Kinderhook Lake	0.00	0.11
			Nassau Lake	0.01	0.17
			Ravena Pond	0.20	**4.41**
		West	Alcove Reservoir	0.00	0.09
			Kinderhook Lake	0.01	0.11
			Nassau Lake	0.01	0.17
			Ravena Pond	0.20	**4.41**
		Screening	Screening	0.14	**3.05**

I-6.3 Mercury

Elemental and divalent mercury emitted by the Ravena portland cement facility were modeled as individual species in TRIM.FaTE. Mercury emissions reported in NEI for the Ravena facility were not specified with regard to the proportion of elemental and divalent species; consequently, emissions were modeled as 75% elemental and 25% divalent (the default mercury speciation for the portland cement source category).

Methyl mercury is created via transformation reactions in the environment, and TRIM.FaTE includes transformation algorithms to model key transformation reactions. In the charts that follow, results are presented for individual mercury species and total mercury (*i.e.*, the sum of mass or concentrations of the three modeled species). TRIM.FaTE mass and concentration outputs for all modeled mercury species are reported by the model and expressed here in terms of mass of mercury. For example, methyl mercury concentrations are expressed as "mercury as methyl mercury" (where the reported value excludes the mass of the carbon and hydrogen elements present in the compound). This convention is consistent with the way EPA has defined the oral RfD for methyl mercury.

In most cases, mercury results are plotted on graphs with logarithmic scales.

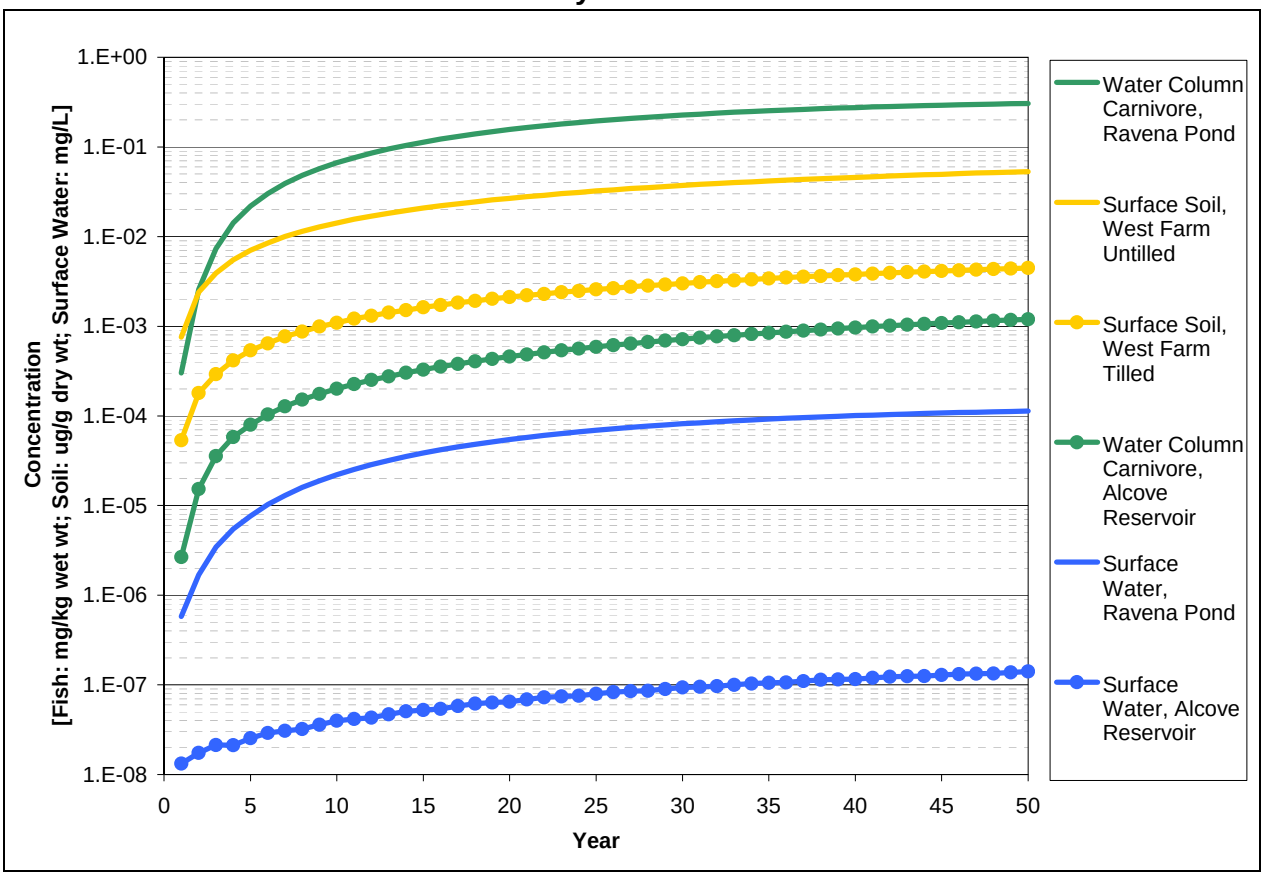
I-6.3.1 *Mercury Media Concentrations*

Exhibit I.6-14 presents a time series of total mercury concentrations for:

- Fish species (water column carnivores) in the Ravena Pond and the Alcove Reservoir compartments,
- Surface water in the pond and the Alcove Reservoir, and
- Tilled and untilled surface soil in the western farm compartment.

Similar to the TRIM.FaTE results observed for 2,3,7,8-TCDD, concentrations increase rapidly over the first fifteen years and then increase less rapidly for the remainder of the model run. The long-term rate at which modeled mercury concentrations increase appears to be somewhat higher than dioxin (in other words, in our modeling scenario, mercury concentrations do not approach steady-state as quickly as dioxins). This is likely due to the fact that dioxin is assumed to degrade in the environment (with a half-life on the order of years), while mercury does not degrade. Processes that remove mercury from the modeled system include volatilization of elemental mercury from soil and water, outflow of dissolved and suspended sediment-borne mercury from lakes and the river, and other transfers, which collectively appear to remove mercury at a slower overall rate than processes (including degradation) that affect dioxin concentrations. Despite this difference, the rate of change of modeled mercury concentrations is relatively low at the end of the 50-year model run.

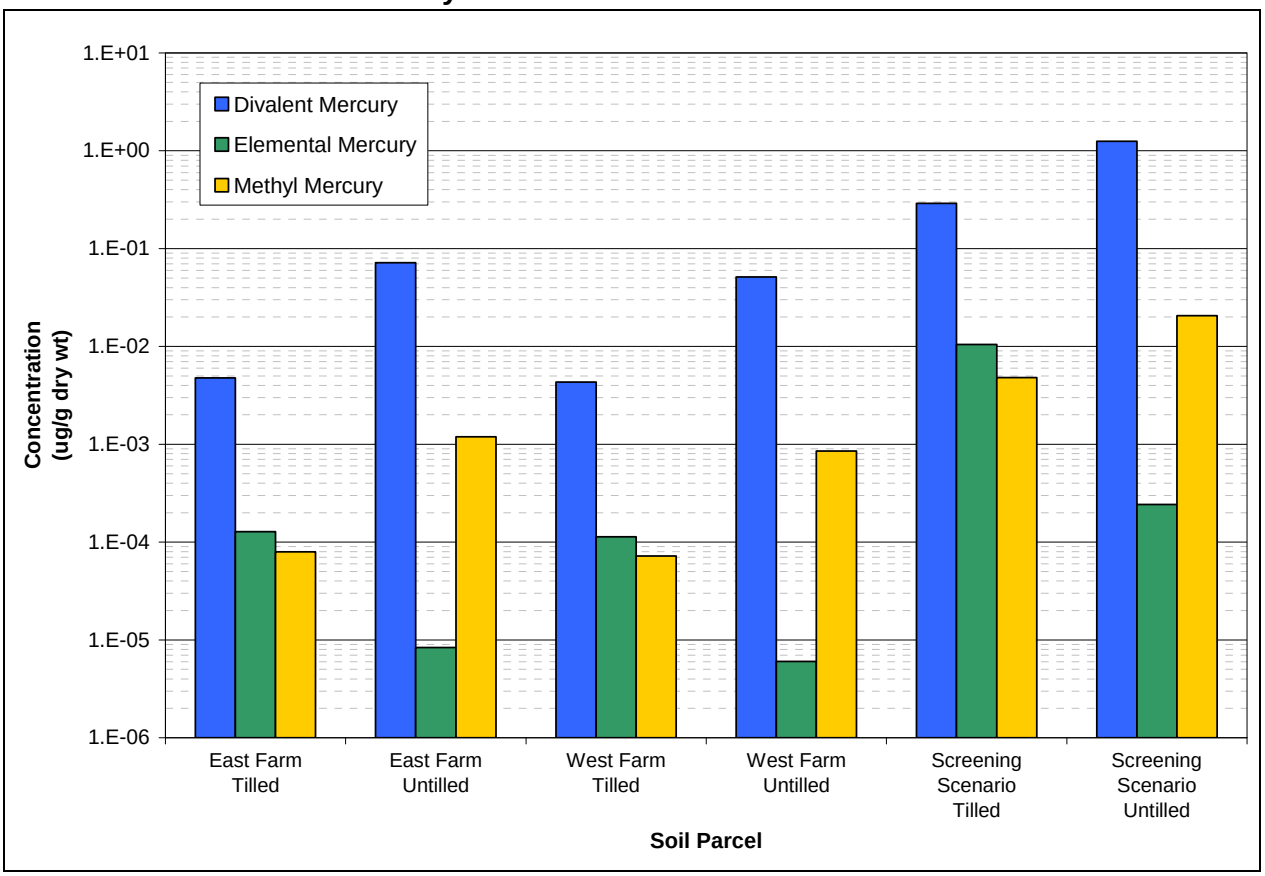
Exhibit I.6-14. Total Mercury Media Concentration Time Series



Elemental, divalent, and methyl mercury surface soil concentrations are presented in Exhibit I.6-15. Divalent and methyl mercury concentrations (shown in blue and yellow, respectively) are higher in untilled parcels than in tilled parcels, while elemental mercury concentrations (shown in blue) are higher in tilled parcels. This trend occurs because elemental mercury is volatile, and the TRIM.FaTE-estimated volatilization rate is dependent on the estimated vertical concentration gradient of mercury in the soil. Though roughly the same mass of mercury is mixed in the tilled and untilled compartments, the soil profile in the tilled parcels is deeper. As a result, volatilization occurs more slowly and concentrations of elemental mercury are higher (even though total mercury concentrations are lower).

Divalent mercury concentrations in soil are typically higher than elemental mercury at all locations because this species deposits much more readily than elemental mercury (as reflected by the mass distribution summary). Furthermore, the volatilization of elemental mercury ensures that its residence time in the soil is short compared to divalent mercury. As with 2,3,7,8-TCDD, estimated soil concentrations are higher when the Ravena mercury emissions are modeled in the RTR screening scenario than when the same emissions are modeled in the site-specific Ravena layout. This is true for both the tilled and untilled compartments.

Exhibit I.6-15. Mercury Surface Soil Concentrations at 50th Model Year



Total mercury air concentrations (shown in blue), surface soil concentrations (in green), and dry particle deposition rates (yellow triangles with values corresponding to the right y-axis) are presented in Exhibit I.6-16. Soil concentrations were estimated to be higher at the east farm location while air concentrations and dry deposition rates were slightly higher at the west farm location. Dry deposition rates are highest for the tilled west farm compartment. These air, soil, and deposition values were used to calculate chemical concentrations in farm food chain media.

Exhibit I.6-16. Total Mercury Air and Surface Soil Concentrations and Dry Particle Deposition Rates at 50th Model Year

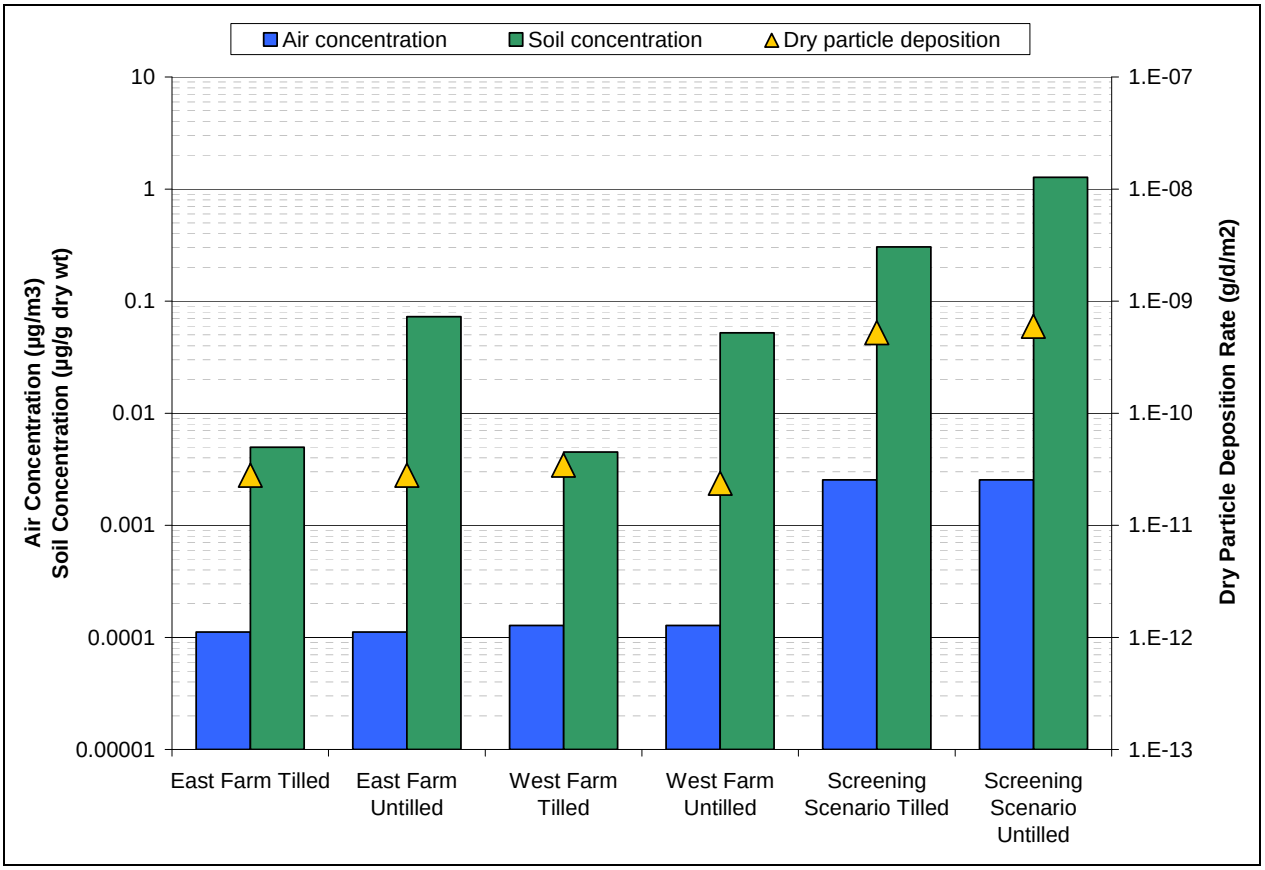


Exhibit I.6-17 shows the surface water concentrations in the Ravena water bodies and in the Screening Scenario Pond for all mercury species. Concentrations are generally similar across all three of the large Ravena water bodies. The concentrations in Ravena Pond and the Screening Scenario Pond are approximately equivalent, with mercury levels far exceeding those in the other bodies of water. This result is discussed further in Section I-6.4.

Exhibit I.6-17. Mercury Surface Water Concentrations During the 50th Model Year

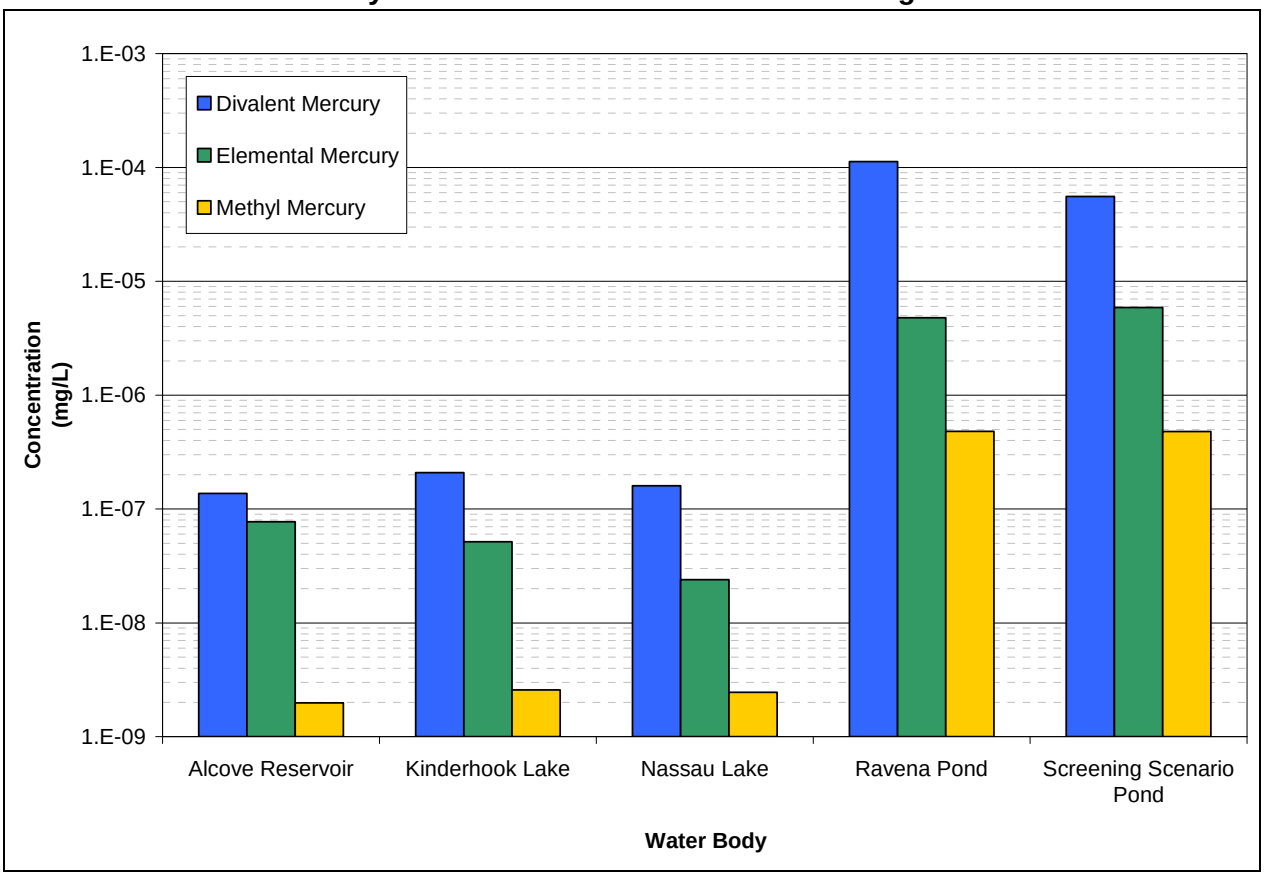


Exhibit I.6-18 illustrates the total mercury concentration in different aquatic species for all water bodies. Note that the lines in Exhibit I.6-18 do not indicate trends but rather are included to assist the reader in making comparisons between concentrations in the water bodies and fish compartments. Fish in the Ravena Pond and Screening Scenario Pond have the highest total mercury concentrations, with some results for the pond almost 100 times higher than the larger lakes and reservoir. Note that the dimensions of the Ravena Pond do not enable it to support water column omnivores and benthic carnivores (see Section I-6.4).

Exhibit I.6-18. Total Mercury Concentration in Fish Species During the 50th Model Year

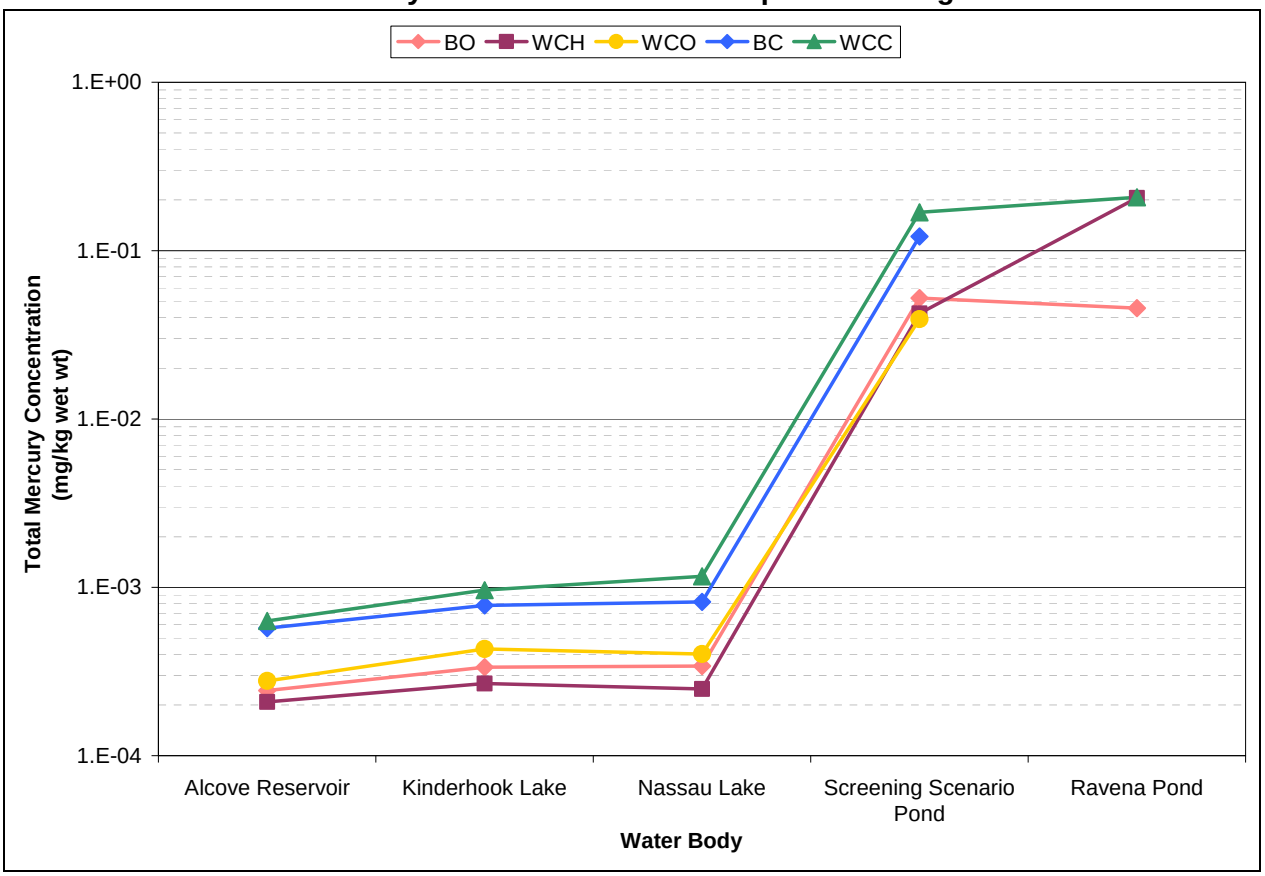


Exhibit I.6-19 summarizes the mercury speciation in modeled environmental media. Elemental mercury (shown in green) is the most predominant form of mercury in the air (roughly reflecting the emission profile of the modeled source). Most of the mercury in soil and sediment is present as divalent mercury (shown in blue). Methyl mercury (in yellow) is present in the modeled aquatic biota compartments, and the fraction of methyl mercury in the aquatic biota compartments (benthic invertebrates, water column herbivores, and water column carnivores) increases with higher trophic levels. The water column carnivores have the highest fraction of methyl mercury while the benthic invertebrates have the lowest.

Exhibit I.6-19. Mercury Speciation Across Different Model Compartments

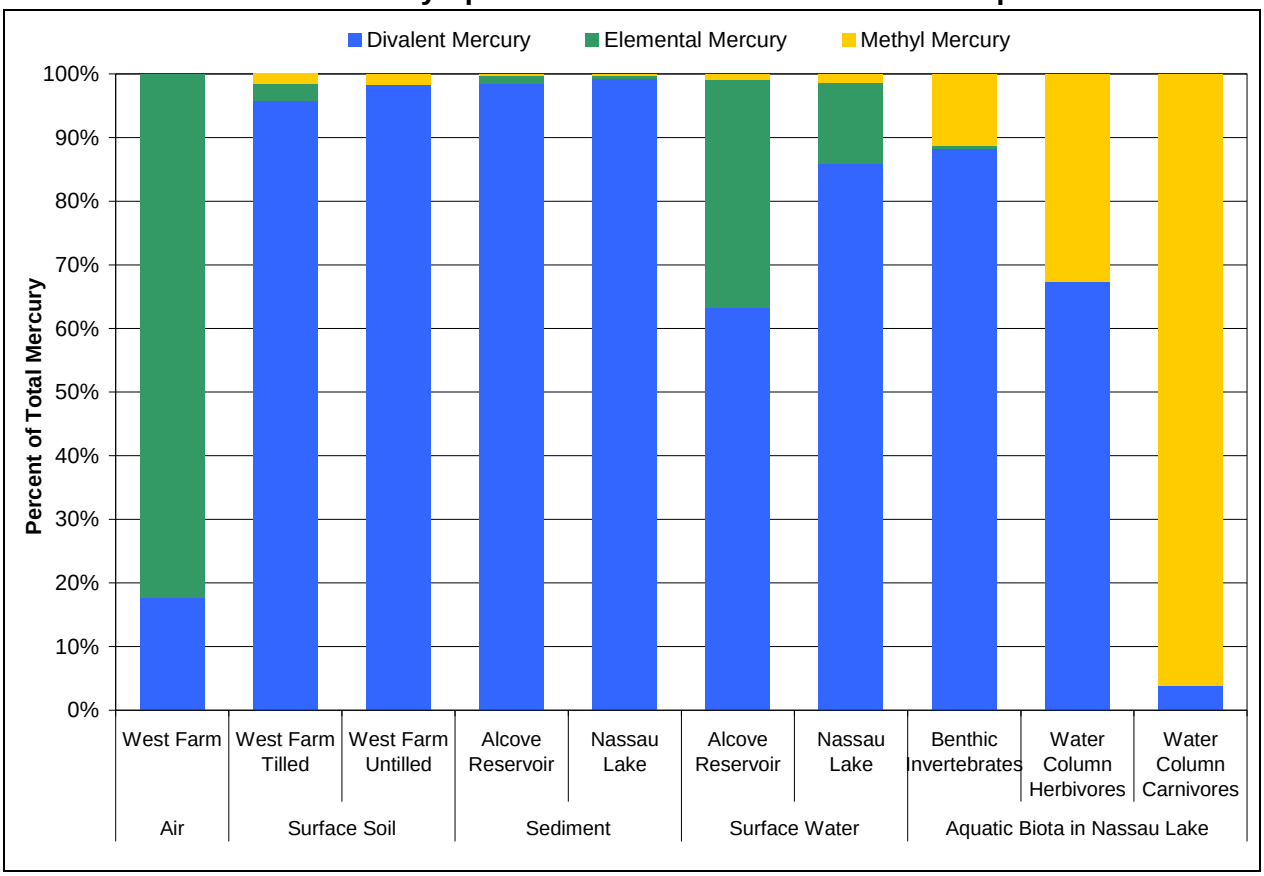
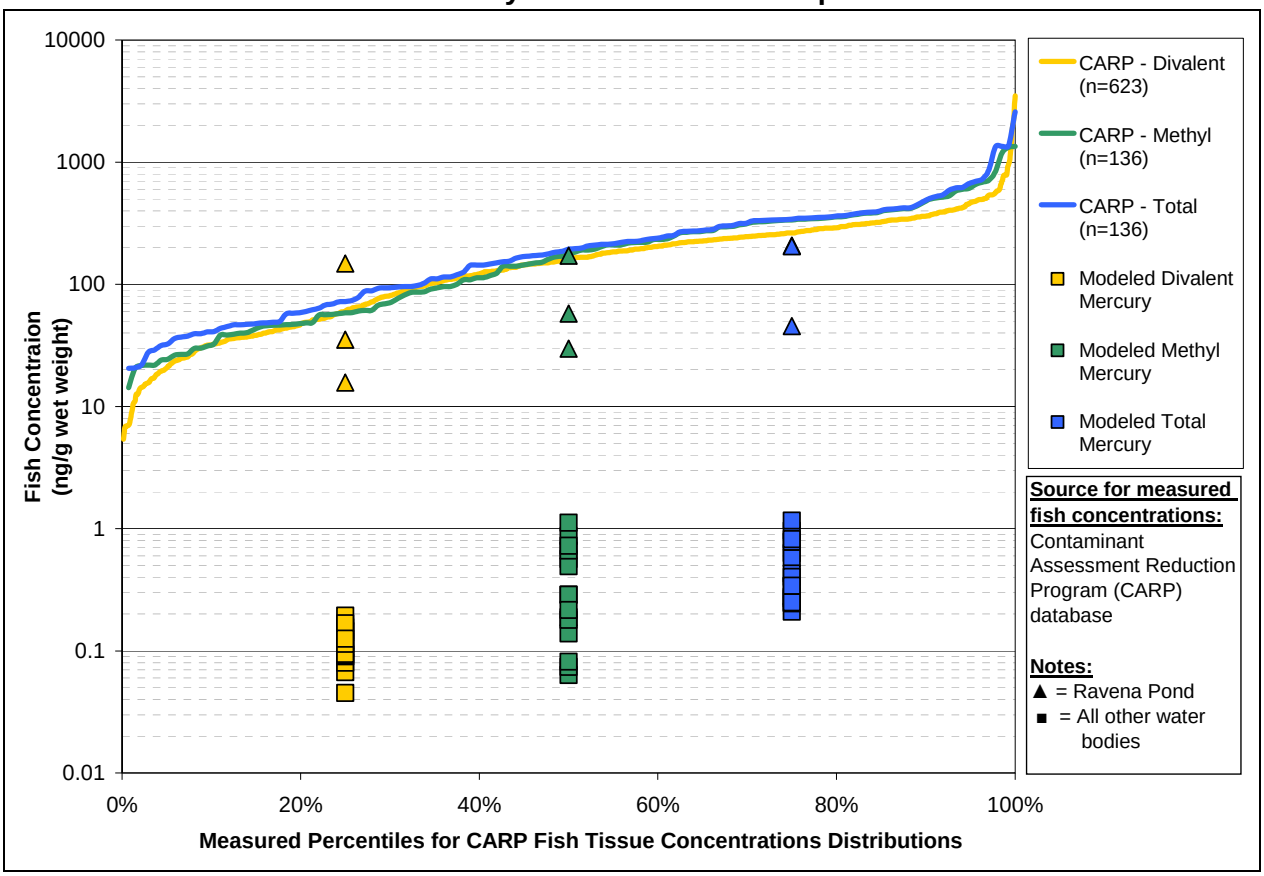


Exhibit I.6-20 compares divalent, methyl, and total mercury concentrations for fish in all modeled water bodies in the Ravena scenario to measured values for the Hudson River and surrounding bays (HRF 2007). Note that the concentrations are plotted on a logarithmic scale. For the measured values, the environmental data were ranked to show the distribution of values.

The modeled concentrations for the different mercury species are presented as three separate series and do not correspond to the percentiles on the x-axis. The modeled concentrations from the pond are shown with triangles and are the only values that overlap the measured concentrations. All other modeled concentrations are shown with squares, and these concentrations are lower than any of the measured concentrations. This was the expected result since the measured values in the Hudson Bay include contributions from many sources, whereas the Ravena water bodies reflect only the incremental impact of mercury emissions from the Ravena Portland cement facility.

Exhibit I.6-20. Modeled Mercury Concentrations Compared to Measured Values



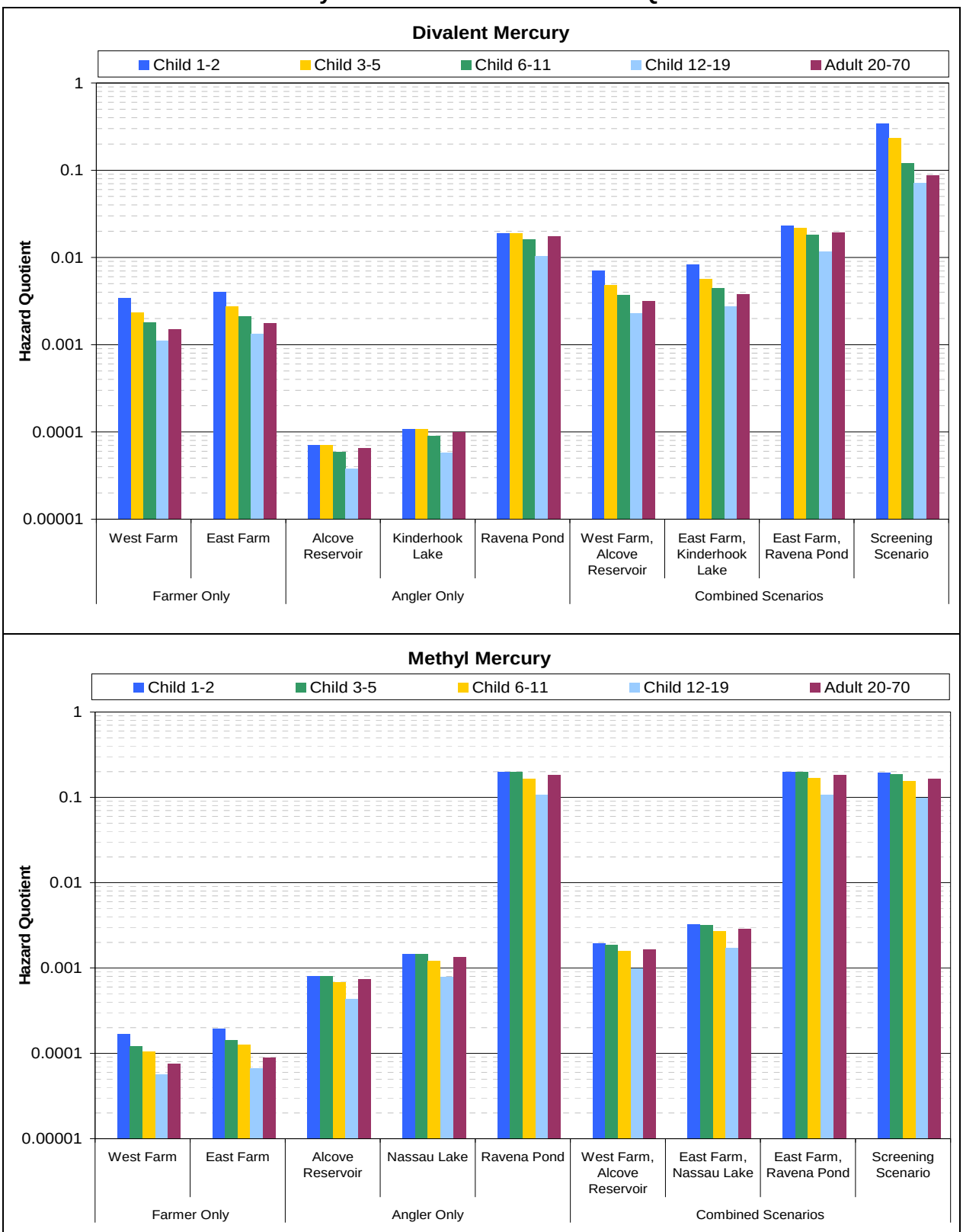
I-6.3.2 *Mercury Risk Assessment Results*

As in the 2,3,7,8-TCDD analysis, the annually-averaged mercury concentrations from the 50th year were used to derive chronic non-cancer HQs based on assumptions of ingestion rates for fish, dairy, beef, and all other foods. Different combinations and variations of the two main exposure scenarios were investigated, and the results presented here largely assume RME (reasonable maximum exposure) ingestion rates. For the combined scenarios, the case associated with the lowest HQ (the West Farm and the Alcove Reservoir) and the case associated with the highest HQ (the East Farm and the Ravena Pond) are both presented, along with the second highest HQ case for comparison.

I-6.3.2.1 Mercury Chronic Non-cancer Hazard Quotients

HQs were calculated separately for methyl and divalent mercury. The top graph in Exhibit I.6-21 presents the estimated divalent mercury HQs using exposure scenarios for farmer ingestion, angler ingestion, and combinations of these two pathways using RME ingestion rates. Methyl mercury HQs are shown in the bottom graph in Exhibit I.6-21. In all cases evaluated, estimated HQs were less than 1.

Exhibit I.6-21. Mercury Chronic Non-Cancer Hazard Quotients for Ravena



Note: Presented results were calculated using the 90th percentile ingestion rates (RME).

In both figures, hazard quotients are highest with children aged 1-2 and they decrease with age as children grow older. Estimated hazard quotients for adults are greater than for all child age groups except the child aged 1-2 group. HQs in the site-specific analysis are approximately an order of magnitude lower than those for the Screening Scenario analysis. The HQs associated with fish consumption from the Ravena Pond are an exception to this and are discussed in further detail in Section I-6.4.

In all scenarios for all chemicals, ingestion of products from the East Farm results in higher HQs than ingestion of products from the West Farm. The difference in HQs related to consumption of products from the two farms is greater for divalent mercury than for methyl mercury; this result makes sense because divalent mercury accumulates more in farm-food chain media than in fish. Hazard quotients calculated based on consumption of fish from either Kinderhook or Nassau Lakes are slightly larger than those based on fish consumption from Alcove Reservoir.

The percent contributions to HQ for each exposure pathway were also calculated for divalent mercury and methyl mercury and are presented in Exhibit I.6-22 and Exhibit I.6-23, respectively. The scenarios are displayed in order of increasing HQ, with the scenario resulting in the lowest HQ displayed at the far left and the scenario resulting in the highest HQ at the far right.

Exhibit I.6-22. Pathway Contributions to Divalent Mercury Chronic Non-Cancer Hazard Quotients

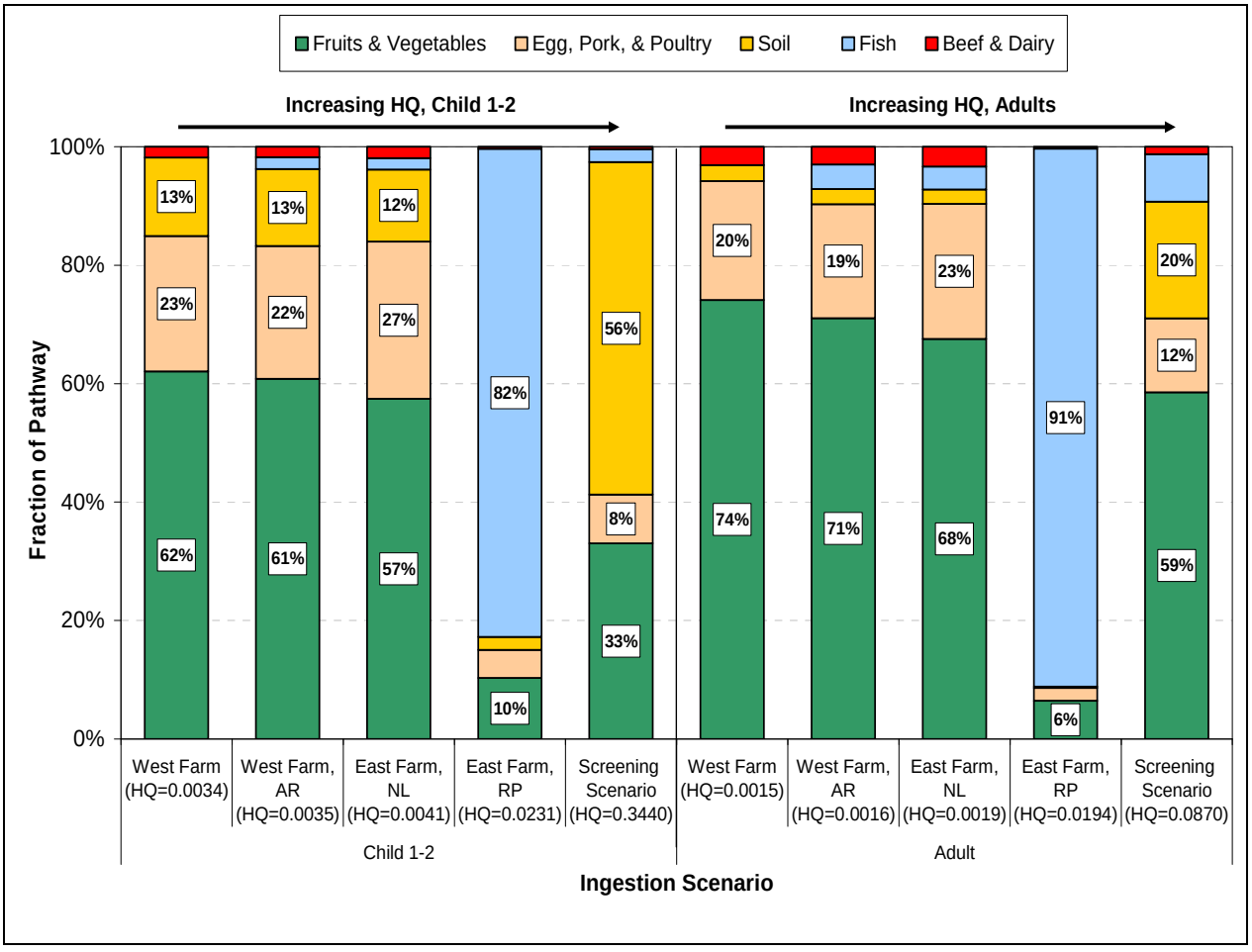
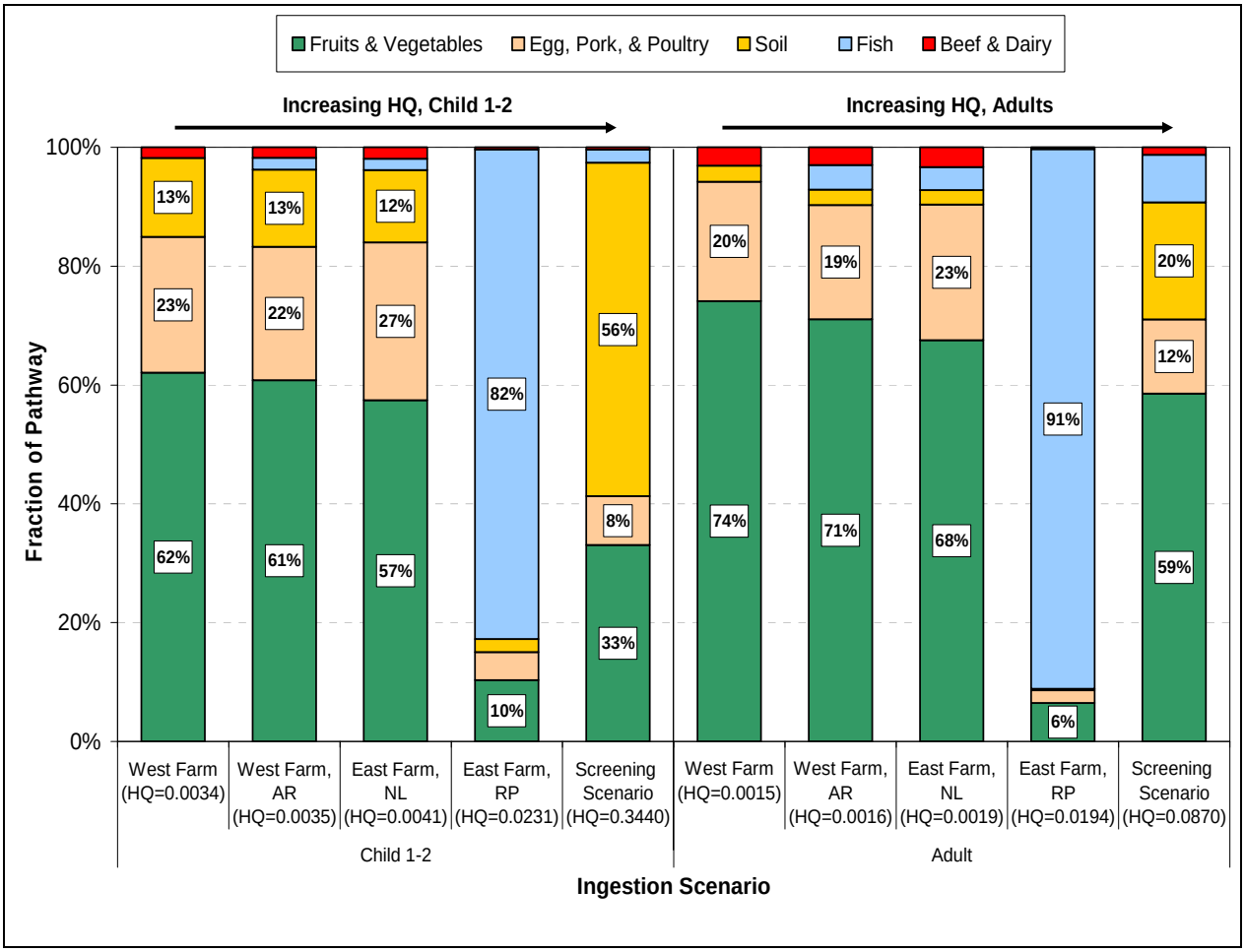


Exhibit I.6-23. Pathway Contributions to Methyl Mercury Chronic Non-Cancer Hazard Quotients



In general, consumption of soil is associated with the largest contribution to the hazard quotient for divalent mercury in children aged 1-2, while consumption of fruits and vegetables is the dominant exposure pathway for divalent mercury in adults. As expected, the ingestion of fish is the dominant exposure pathway for methyl mercury in all age groups.

CTE (central tendency exposure) and 90th percentile RME ingestion rates were also evaluated to better understand the differences in hazard quotients when using different ingestion rates. Exhibit I.6-24 presents the hazard quotient based on the 90th percentile ingestion rate and hazard quotient based on the mean ingestion rate for various scenarios for children aged 1-2. A ratio of hazard quotients using the 90th percentile ingestion rate and mean ingestion rate was also calculated. In general, when using the 90th percentile ingestion rates for all ages, hazard quotients are generally 2- to 3-times greater than the hazard quotients associated with the mean ingestion rates. In many—but not all—cases, the ratio of the HQs decreases slightly as children age.

Exhibit I.6-24. Comparison of Hazard Quotients for Ravena Scenario Using Mean and 90th Percentile Ingestion Rates

Scenario	Farm Parcel	Water body	90th Percentile Ingestion Rate, HQ Child 1-2	Mean Ingestion Rate, HQ Child 1-2	Ratio 90th : Mean HQ
Divalent Mercury					
Screening	Screening	Screening Pond	0.344	0.085	4.1
Combined	West	Alcove Reservoir	0.007	0.003	2.7
	East	Kinderhook Lake	0.008	0.003	2.6
		Ravena Pond	0.023	0.010	2.4
Farm Only	West	-	0.003	0.001	2.7
	East	-	0.004	0.002	2.6
Fisherman Only	-	Alcove Reservoir	0.0001	0.00003	2.4
		Kinderhook Lake	0.0001	0.00005	2.4
		Ravena Pond	0.019	0.008	2.4
Water Ingestion Only	-	-	2.4E-08	1.1E-08	2.2
Methyl Mercury					
Screening	Screening	Screening Pond	0.193	0.079	2.5
Combined	West	Alcove Reservoir	0.002	0.001	2.4
	East	Nassau Lake	0.003	0.001	2.4
		Ravena Pond	0.199	0.084	2.4
Farm Only	West	-	0.0002	0.0001	2.8
	East	-	0.0002	0.0001	2.8
Fisherman Only	-	Alcove Reservoir	0.001	0.0003	2.4
		Nassau Lake	0.001	0.001	2.4
		Ravena Pond	0.198	0.084	2.4
Water Ingestion Only	-	-	1.0E-09	4.6E-10	2.2

Note: Values presented have been rounded

I-6.3.2.2 Mercury Chronic Non-Cancer Hazard Quotients from Dermal Exposure

Dermal exposures were assessed for divalent mercury using the methodology described in Appendix C. The highest combined HQs occurred when the calculations used soil concentrations from the untilled East farm compartment for a child age 1-2. These HQs are presented in Exhibit I.6-25 along with HQs derived using the Ravena mercury emission rate in the Screening Scenario. A table providing hazard quotients for all age groups and farm compartments considered is provided in Attachment I-2 of this document.

The HQ derived from the site-specific scenario with Alcove Reservoir is 6.7E-3 for children aged 1-2. In this case, the dermal HQs are the same order of magnitude (and somewhat higher than) the ingestion HQs. Thus, the dermal exposure pathway seems to be of equal importance as the ingestion pathway for divalent mercury exposures at this site. However, all HQs are well below 1. It should be noted that the dermal calculations are based on highly conservative assumptions including a high surface area in children that is exposed to soil.

Exhibit I.6-25. Divalent Mercury Dermal Hazard Quotients for a Child Aged 1-2

Farm	Water body	Hazard Quotient			
		Soil	Water	Soil and Water	Ratio of Ingestion to Dermal [a]
East Farm - Untilled	Alcove Reservoir	6.7E-03	3.2E-06	6.7E-03	0.30
Screening	Screening	1.7E-01	1.3E-03	1.7E-01	0.50

[a] Ratio compares total dermal HQ calculated based on RME parameters to total ingestion HQ based on CTE ingestion rates (i.e., ratio of highest dermal HQ to lowest ingestion HQ).

I-6.4 Alternate Modeling Scenario - Incorporation of Fish Harvesting from Ravena Pond

During the development of the conceptual exposure model for the risk assessment of the Ravena facility, we determined that the possibility of an angler exposure scenario existing for the Ravena pond was low; however, the Ravena Pond was retained in this case study evaluation to provide an additional "what-if" analysis. Juxtaposition of the exposure (i.e., fish ingestion rates) and environmental assumptions associated with the angler-pond scenario illustrates the implausibility of this scenario. Specifically, it is assumed that the angler fishes at the pond regularly for a lifetime and consumes his or her catch. Due to the small size of the pond (20,000 m^2 in surface area and 1 m deep), it is unlikely that this water body could sustain fishable populations at the assumed ingestion rates without regular, substantial restocking of fish.

The total standing fish biomass in the pond was assumed to be 80 kg on average over the course of a year. We assumed approximately 5 percent of this total (4 kg) was present as adult bass, represented in the water column carnivore compartment, and 75 percent of this total (60 kg) was present as benthic omnivores such as catfish or sunfish. The angler is assumed to consume these two types of fish (bass and benthic omnivores) at a ratio of 1:2, with preferential harvesting of bass. The 90th percentile, reasonable maximum exposure (RME) fish ingestion rate for an adult angler was assumed to be 17 g/day.

Based on these exposure assumptions and using an exposure duration of 365 days/year, the amount of bass harvested in one year would need to be about 2 kg (i.e., 17 g/day x 365 days/yr x 0.33 of total consumption).[8] This represents 50% of the standing biomass in the Ravena Pond. Using the same exposure assumptions, the amount of benthic omnivores harvested would be nearly 4 kg/yr, which is about 7% of the standing biomass. Note that these calculations assume that only one single angler is fishing in the pond, and the associated risk calculations assume that one single consumer eats all of the fish that are caught.

This situation appears not to be ecologically sustainable, and at a minimum is likely to significantly reduce the chemical concentrations in fish tissues in all fish types. Thus, to obtain a more realistic estimate of concentrations in fish in the Ravena Pond, we modified the TRIM.FaTE scenario and incorporated a fish harvesting rate from the pond of 17 g/day to represent consumption and restocking of the pond within the TRIM.FaTE model. This harvesting rate corresponds to the 90th percentile fish ingestion rate for humans used to

[8] We recognize that these calculations do not take into account other details (for example, the biomass and mass of fish consumed are directly compared, even though the entire mass of a fish is not consumed), but the intent is a subjective examination of the underlying assumptions.

calculate hazard quotients and lifetime cancer risks associated with consumption of contaminated fish.

Exhibit I.6-26 compares concentrations of 2,3,7,8–TCDD and total mercury in various aquatic compartments when TRIM.FaTE was run with and without modeled fish harvesting from the Ravena Pond compartment. By incorporating fish harvesting within the TRIM.FaTE scenario, estimated dioxin concentrations in fish consumed by humans decreased by 22% and 38%,and estimated total mercury concentrations decreased by 5% and 39% in water column herbivores and carnivores, respectively.

Exhibit I.6-26. Effect of Fish Harvesting on Annually Averaged PB-HAP Concentrations During the 50th Model Year in Ravena Pond Using 95-Percent UCL Emission Rate

Compartment	Units	Concentration		Percent Reduction in Concentration With Addition of Harvesting
		With Fish Harvesting	No Fish Harvesting	
2,3,7,8-TCDD				
Surface Water	mg/L	1.3E-11	1.3E-11	0%
Sediment concentration	ug/g dry wt.	6.3E-08	6.3E-08	0%
Macrophyte	mg/kg wet wt.	1.9E-07	1.9E-07	0%
Benthic Invertebrate	mg/kg wet wt.	6.2E-09	6.2E-09	0%
Benthic Omnivore	mg/kg wet wt.	8.8E-07	8.8E-07	0%
Water Column Herbivore	mg/kg wet wt.	3.7E-06	4.7E-06	22%
Water Column Carnivore	mg/kg wet wt.	3.2E-06	5.2E-06	38%
Mallard	mg/kg wet wt.	3.1E-05	3.1E-05	0%
Mink [a]	mg/kg wet wt.	4.9E-09	-	-
Total Mercury				
Surface Water	mg/L	1.2E-04	1.2E-04	0%
Sediment concentration	ug/g dry wt.	9.1E-01	9.1E-01	0%
Macrophyte	mg/kg wet wt.	5.4E-05	5.4E-05	0%
Benthic Invertebrate	mg/kg wet wt.	5.2E-02	5.2E-02	0%
Benthic Omnivore	mg/kg wet wt.	2.1E-01	2.1E-01	0%
Water Column Herbivore	mg/kg wet wt.	4.3E-02	4.5E-02	5%
Water Column Carnivore	mg/kg wet wt.	1.3E-01	2.1E-01	39%
Mallard	mg/kg wet wt.	3.7E-02	3.7E-02	0%
Mink [a]	mg/kg wet wt.	6.7E-05	-	-

[a] The harvester was modeled in the mammalian mink compartment because no human compartment exists in the TRIM.FaTE modeling system.

Exhibit I.6-27 presents the corresponding changes in 1) the lifetime cancer risk for 2,3,7,8–TCDD and 2) the hazard quotient for a child age 1-2 for divalent and methyl mercury when fish harvesting is included in the model scenario. The comparison reveals that in all cases, the addition of fish harvesting from the Ravena Pond results in approximately a 27% decrease in the individual lifetime cancer risk for 2,3,7,8 - TCDD, no matter which ingestion rates or emission rates are used. This relatively modest change in magnitude is not enough to change any conclusions with regard to typical risk thresholds (e.g., one-in-a-million or 100-in-a-million). For mercury, risks are reduced more for the methyl mercury (34%) than divalent mercury (10-12%), reflecting the fact that the fish pathway is more important for methyl mercury. However, the hazard quotients are less than one both with and without the modeled fish harvesting.

Exhibit I.6-27. Risks and Hazard Quotients in Ravena Pond with and without Fish Harvesting

Chemical	Ingestion Rate	Scenario Type	Farm Parcel	Risk/HQ Without Fish Harvesting from Pond [a]	Risk/HQ With Fish Harvesting from Pond [a]	Percent Reduction in Risk/HQ with Addition of Harvesting [a]
2,3,7,8 – TCDD, 95th Emission Factor	90th Ingestion Rates	Combined	West	1.7E-04	1.2E-04	27%
			East	1.7E-04	1.2E-04	27%
		Fisherman Only	N/A	1.6E-04	1.2E-04	27%
	Mean Ingestion Rates	Combined	West	6.8E-05	5.0E-05	27%
			East	6.8E-05	5.0E-05	27%
		Fisherman Only	N/A	6.7E-05	4.9E-05	27%
2,3,7,8 – TCDD, Mean Emission Factor	90th Ingestion Rates	Combined	West	6.8E-05	5.0E-05	27%
			East	6.8E-05	5.0E-05	27%
		Fisherman Only	N/A	6.7E-05	4.9E-05	27%
	Mean Ingestion Rates	Combined	West	2.8E-05	2.0E-05	27%
			East	2.8E-05	2.0E-05	27%
		Fisherman Only	N/A	2.7E-05	2.0E-05	27%
Divalent Mercury	90th Ingestion Rates	Combined	West	2.0E-02	2.2E-02	10% [b]
			East	2.1E-02	2.3E-02	10% [b]
		Fisherman Only	N/A	1.7E-02	1.9E-02	12% [b]
	Mean Ingestion Rates	Combined	West	8.4E-03	9.3E-03	10% [b]
			East	8.6E-03	9.6E-03	10% [b]
		Fisherman Only	N/A	7.1E-03	8.0E-03	12% [b]
Methyl Mercury	90th Ingestion Rates	Combined	West	1.3E-01	2.0E-01	34% [b]
			East	1.3E-01	2.0E-01	34% [b]
		Fisherman Only	N/A	1.3E-01	2.0E-01	34% [b]
	Mean Ingestion Rates	Combined	West	5.6E-02	8.4E-02	34% [b]
			East	5.6E-02	8.4E-02	34% [b]
		Fisherman Only	N/A	5.6E-02	8.4E-02	34% [b]

[a] Represents the lifetime cancer risk for 2,3,7,8 – TCDD and the hazard quotient for a child age 1-2 for divalent mercury and methyl mercury.

[b] The change in hazard quotients with and without the harvester was also compared for children of other age groups and adults, and the percent reduction in risk was found to be broadly consistent across these age groups.

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I-7 References

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ATTACHMENT I-1: TRIM.FaTE Inputs for the Ravena Case Study

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TABLE OF CONTENTS

LIST OF EXHIBITS

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I-1-1TRIM.FaTE Modeling Inputs

This section provides the tables of the detailed modeling inputs for the TRIM.FaTE screening scenario. Exhibits 1-1 and 1-2 present the air parameters entered into the model. In Section 2 of this attachment, supplemental information is provided for Exhibit 1-2, which is a summary of meteorological inputs for the TRIM.FaTE analysis.

Exhibits 1-3 through 1-8 present the terrestrial parameters. In Section 3 of this attachment, supplemental information is provided for Exhibit 1-6, which presents the inputs for the Universal Soil Loss Equation.

Exhibits 1-9 through 1-12 present the lake parameters, and 1-13 through 1-20 present the chemical specific parameters. In Section 4 of this attachment, supplemental information is provided for Exhibit 1-12, which is a summary of non-chemical-dependent parameter inputs for aquatic animals.

Exhibit 1-1. Non-Chemical-Dependent Air Parameters

Parameter Name	Units	Value Used	Reference
Atmospheric dust load	kg[dust]/m^3[air]	6.15E-08	Bidleman 1988
Air density	g/cm^3	0.0012	U.S. EPA 1997
Dust density	kg[dust]/m^3[dust]	1,400	Bidleman 1988
Fraction organic matter on particulates	unitless	0.2	Harner and Bidleman 1998
Height [VE property]	m	varies	Meteorological data used

Exhibit 1-2. Meteorological and Other Settings [a]

Parameter Name	Units	Value Used	Reference
Meteorological Inputs (all TRIM.FaTE scenario properties, except mixing height)			
Air temperature	degrees K	varies daily	MET data assembled from actual 2001-2003 records
Horizontal wind speed	m/sec	varies daily	MET data assembled from actual 2001-2003 records
Vertical wind speed	m/sec	0.0	Professional judgment; vertical wind speed not used by any of the algorithms in the version of the TRIM.FaTE library used for screening
Wind direction	degrees clockwise from N (blowing from)	varies daily	MET data assembled from actual 2001-2003 records
Rainfall rate	m^3[rain]/ m^2[surface area]-day	varies daily	MET data assembled from actual 2001-2003 records
Mixing height (used to set air VE property named “top”)	m	varies daily	MET data assembled from actual 2001-2003 records
isDay_SteadyState_forAir	unitless	--	Value not used in current dynamic runs (would need to be reevaluated if steady-state runs are needed)
isDay_SteadyState_forOther	unitless	--	
Other Settings (all TRIM.FaTE scenario properties)			
Start of simulation	date/time	1/1/1990, midnight	Consistent with met data
End of simulation	date/time	1/1/2020, midnight	Consistent with met data set; selected to provide a 30-year modeling period
Simulation time step	hr	1	Selected value
Output time step [b]	hr	2	Selected value

[a] For more information, see Section 2 of this attachment.

[b] Output time step is set in TRIM.FaTE using the scenario properties "simulationStepsPerOutputStep" and "simulationTimeStep."

Exhibit 1-3. Non-Chemical-Dependent Soil Parameters

Parameter Name	Units	Value Used	Reference
Surface Soil Compartment Type			
Air content	volume[air]/ volume[compartment]	0.28	McKone et al 2001 (Table 15)
Average vertical velocity of water (percolation)	m/day	4.40E-04	Assumed to be 0.2 times average precipitation for site
Boundary layer thickness above surface soil	m	0.005	Thibodeaux 1996; McKone et al. 2001 (Table 3)
Density of soil solids (dry weight)	kg[soil]/m^3[soil]	2600	Default in McKone et al. 2001 (Table 3)
Thickness -untilled [VE property] [a]	m	0.01	McKone et al. 2001 (p. 30)
Thickness -tilled [VE property] [a]	m	0.20	USEPA 2005
Erosion fraction [Link property]	unitless	varies [b]	See Erosion and Runoff Fraction table.
Fraction of area available for erosion	m^2[area available]/m^2[total]	1	Professional judgment; area assumed rural
Fraction of area available for runoff	m^2[area available]/m^2[total]	1	Professional judgment; area assumed rural
Fraction of area availabe for vertical diffusion	m^2[area available]/m^2[total]	1	Professional judgment; area assumed rural
Fraction Sand	unitless	0.25	Professional judgment
Organic carbon fraction	unitless	0.008	U.S. average in McKone et al. 2001 (Table 16 and A-3)
pH	unitless	6.8	Professional judgment
Runoff fraction [Link property]	unitless	varies [b]	See Erosion and Runoff Fraction table.
Total erosion rate	kg [soil]/m^2/day	varies [b]	See Total Erosion Rates table.
Total runoff rate	m^3[water]/m^2/day	4.04E-04	Calculated using scenario-specific precipitation rate and assumptions associated with water balance.
Water content	volume[water]/ volume[compartment]	0.19	McKone et al 2001 (Table 15)

Exhibit 1-3. Non-Chemical-Dependent Soil Parameters

Parameter Name	Units	Value Used	Reference
Root Zone Soil Compartment Type			
Air content	volume[air]/ volume[compartment]	0.25	McKone et al 2001 (Table 16)
Average vertical velocity of water (percolation)	m/day	4.40E-04	Assumed as 0.2 times average precipitation for New England in McKone et al. 2001
Density of soil solids (dry weight)	kg[soil]/m^3[soil]	2,600	McKone et al. 2001 (Table 3)
Fraction Sand	unitless	0.25	Professional judgment
Thickness - untilled [VE property] [a]	m	0.69	McKone et al. 2001 (Table 16 - Middle Atlantic value)
Thickness - tilled [VE property] [a]	m	0.6	Adjusted from McKone et al. 2001 (Table 16)
Organic carbon fraction	unitless	0.008	McKone et al. 2001 (Table 16 and A-3, U.S. Average)
pH	unitless	6.8	Professional judgment
Water content	volume[water]/ volume[compartment]	0.21	McKone et al 2001 (Table 16)
Vadose Zone Soil Compartment Type			
Air content	volume[air]/ volume[compartment]	0.22	McKone et al 2001 (Table 17)
Average vertical velocity of water (percolation)	m/day	4.40E-04	Assumed as 0.2 times average precipitation for New England in McKone et al. 2001
Density of soil solids (dry weight)	kg[soil]/m^3[soil]	2,600	Default in McKone et al. 2001 (Table 3)
Fraction Sand	unitless	0.35	Professional judgment
Thickness [VE property] [a]	m	1.4	McKone et al. 2001 (Table 17)
Organic carbon fraction	unitless	0.003	McKone et al. 2001 (Table 17 and A-3, U.S. Average)
pH	unitless	6.8	Professional judgment
Water content	volume[water]/ volume[compartment]	0.21	McKone et al 2001 (Table 17 - national average)

Exhibit 1-3. Non-Chemical-Dependent Soil Parameters

Parameter Name	Units	Value Used	Reference
Ground Water Compartment Type			
Thickness [VE property] [a]	m	3	McKone et al. 2001 (Table 3)
Fraction Sand	unitless	0.4	Professional judgment
Organic carbon fraction	unitless	0.004	Professional judgment
pH	unitless	6.8	Professional judgment
Porosity	volume[total pore space]/ volume[compartment]	0.2	Default in McKone et al. 2001 (Table 3)
Solid material density in aquifer	kg[soil]/m^3[soil]	2,600	Default in McKone et al. 2001 (Table 3)

[a] Set using the volume element properties file.

[b] See separate tables for erosion/runoff fractions and total erosion rates.

Exhibit 1-4. Erosion and Runoff Fractions

Originating Compartment	Destination Compartment	Runoff/Erosion Fraction
SurfSoil_E1	Hudson River	1
SurfSoil_E2	Nassau Lake	1
SurfSoil_E3	Kinderhook Lake	1
SurfSoil_E4	Hudson River	1
SurfSoil_E5	Hudson River	1
SurfSoil_E6	Hudson River	1
SurfSoil_Tilled East Farm	SurfSoil_E5	1
SurfSoil_Untilled East Farm	SurfSoil_E5	1
Nassau Lake	Kinderhook Lake	1
SurfSoil_W1	Hudson River	1
SurfSoil_W2	Hudson River	1
SurfSoil_W3	Alcove Reservoir	1
SurfSoil_W4	Hudson River	1
SurfSoil_W5	Hudson River	1
SurfSoil_W6	Hudson River	1
SurfSoil_W7	Pond	1
SurfSoil_W8	Hudson River	1
SurfSoil_Tilled West Farm	SurfSoil_W2	1
SurfSoil_Untilled West Farm	SurfSoil_W2	1

Exhibit 1-5. Total Erosion Rates from Surface Soil Volume Elements [a]

Soil Component	Total Erosion Rate (kg/m^2-day)
E1	0.000190
E2	0.000323
E3	0.000220
E4	0.000117
E5	0.000148
E6	0.000153
Efarm_tilled	0.001270
Efarm_untilled	0.000500
W1	0.000082
W2	0.000310
W3	0.000337
W4	0.000095
W5	0.000231
W6	0.000370
W7	0.005916
W8	0.000410
Wfarm_tilled	0.006116
Wfarm_untilled	0.000469

[a] Calculated using the Universal Soil Loss Equation in combination with precipitation rate and other assumptions.

Exhibit 1-6. Universal Soil Loss Equation Inputs [a]

Soil Parcel	Area	Rainfall/Erosivity Index	Soil Erodibility Index	Slope Length	Slope Steepness	Topographical (LS) Factor	Land Use
	km^2	100 ft-ton/acre	ton/ac/(100 ft-ton/acre)	meters	%	unitless	
E1	85.224	114	0.3145	200	7.23%	2.206	Grasses
E2	26.719	114	0.3113	200	8.00%	2.539	Deciduous Forest
E3	44.517	114	0.3102	200	7.86%	2.478	Deciduous forest
E4	420.32	114	0.3145	200	9.49%	3.248	Grasses
E5	77.709	114	0.3145	200	6.53%	1.924	Grasses
E6	81.925	114	0.3145	200	6.43%	1.886	Grasses
Efarm tilled	0.358	114	0.3145	200	2.88%	0.484	Tilled Soil/crops
Efarm untilled	0.358	114	0.3145	200	3.98%	0.844	Untilled corn 50 bu/acre
W1	48.836	105	0.3145	200	4.78%	1.041	Grasses
W2	109.84	105	0.3145	200	9.25%	3.125	Deciduous
W3	86.819	105	0.3222	200	9.45%	3.228	Deciduous Forest
W4	306.58	105	0.3145	200	7.16%	2.178	Deciduous Forest
W5	84.025	105	0.3145	200	8.71%	2.864	Deciduous Forest
W6	15.011	105	0.3145	200	9.42%	3.211	Grasses
W7	2.028	105	0.3145	200	8.65%	2.837	Deciduous Forest
W8	72.532	105	0.3145	200	10.77%	3.919	Deciduous Forest
Wfarm tilled	0.359	105	0.3145	200	7.99%	2.534	Tilled Soil/crops
Wfarm untilled	0.358	105	0.3145	200	4.05%	0.860	Untilled corn 50 bu/acre

Exhibit 1-6. Universal Soil Loss Equation Inputs [a]

Soil Parcel	Cover/Mgt Factor	Supporting Practices Factor	Unit Soil Loss		Sediment Delivery Ratio [b]	Calculated (Adjusted) Erosion Rate
	unitless	unitless	ton/ac/yr	kg/m^2/day		kg/m^2/day
E1	0.032	1	2.530	0.0016	0.122	0.000190
E2	0.041	1	3.717	0.0023	0.142	0.000323
E3	0.031	1	2.693	0.0017	0.133	0.000220
E4	0.033	1	3.808	0.0023	0.050	0.000117
E5	0.028	1	1.951	0.0012	0.124	0.000148
E6	0.030	1	2.026	0.0012	0.123	0.000153
Efarm tilled	0.310	1	5.384	0.0033	0.384	0.001270
Efarm untilled	0.070	1	2.119	0.0013	0.384	0.000500
W1	0.029	1	1.012	0.0006	0.131	0.000082
W2	0.041	1	4.263	0.0026	0.119	0.000310
W3	0.041	1	4.497	0.0028	0.122	0.000337
W4	0.041	1	2.963	0.0018	0.052	0.000095
W5	0.032	1	3.072	0.0019	0.123	0.000231
W6	0.032	1	3.395	0.0021	0.177	0.000370
W7	0.333	1	31.156	0.0191	0.309	0.005916
W8	0.041	1	5.348	0.0033	0.125	0.000410
Wfarm tilled	0.310	1	25.940	0.0159	0.384	0.006116
Wfarm untilled	0.070	1	1.989	0.0012	0.384	0.000469

[a] For more information, see Section 3 of this attachment.

[b] Calculated using SD = a * (AL)-b; where a is the empirical intercept coefficient (based on the size of the watershed), AL is the total watershed area receiving deposition (m2), and b is the empirical slope coefficient (always 0.125).

Exhibit 1-7. Surface Soil Terrestrial Plant Types

Surface Soil Volume Element	Surface Soil Depth (m)	Deciduous Forest	Grasses/ Herbs	Crops
E1	0.01	x		
E2	0.01	x		
E3	0.01	x		
E4	0.01	x		
E5	0.01		x	
E6	0.01		x	
Efarm_tilled	0.20 (tilled)			x
Efarm_untilled	0.01			x
W1	0.01		x	
W2	0.01	x		
W3	0.01	x		
W4	0.01	x		
W5	0.01	x		
W6	0.01	x		
W7	0.01	x		
W8	0.01	x		
Wfarm_tilled	0.20 (tilled)			x
Wfarm_untilled	0.01			x

Exhibit 1-8. Non-Chemical-Dependent Terrestrial Plant Parameters

Parameter Name	Units	Deciduous [a]	
		Value Used	Reference
Leaf Compartment Type			
Allow exchange	1=yes, 0=no	seasonal [b]	-
Average leaf area index	m^2[leaf]/ m^2[area]	3.4	Harvard Forest, dom. red oak and red maple, CDIAC website
Calculate wet dep interception fraction (boolean)	1=yes, 0=no	0	Professional judgment
Correction exponent, octanol to lipid	unitless	0.76	From roots; Trapp, S. 1995. Model for uptake of xenobiotics into plants.
Degree stomatal opening	unitless	1	Set to 1 for daytime based on professional judgment (stomatal diffusion is turned off at night using a different property, IsDay)
Density of wet leaf	kg/m^3	820	Paterson et al. 1991
Leaf wetting factor	m	3.00E-04	1E-04 to 6E-04 for different crops and elements, Muller and Prohl 1993
Length of leaf	m	0.01	Professional judgment
Lipid content	kg/ kg wet weight	0.00224	European beech, Riederer 1995
Litter fall rate	1/day	seasonal [c]	-
Stomatal area normalized effective diffusion path length	1/m	200	Wilmer and Fricker 1996
Vegetation attenuation factor	m^2/kg	2.9	Grass/hay, Baes et al. 1984
Water content	unitless	0.8	Paterson et al. 1991
Wet dep interception fraction	unitless	0.2	Calculated based on 5 years of local met data, 1987-1991
Wet mass of leaf per soil area	kg[fresh leaf]/ m^2[area]	0.6	Calculated from leaf area index, leaf thickness (Simonich & Hites, 1994), density of wet foliage
Particle on Leaf Compartment Type			
Allow exchange	1=yes, 0=no	-	-
Volume particle per area leaf	m^3[leaf particles]/m^2[leaf]	1.00E-09	Based on particle density and size distribution for atmospheric particles measured on an adhesive surface, Coe and Lindberg 1987

Exhibit 1-8. Non-Chemical-Dependent Terrestrial Plant Parameters

Parameter Name	Units	Deciduous [a]	
		Value Used	Reference
Root Compartment Type - Nonwoody Only			
Allow exchange	1=yes, 0=no	n/a	n/a
Correction exponent, octanol to lipid	unitless	n/a	n/a
Lipid content of root	kg/kg wet weight	n/a	n/a
Water content of root	kg/kg wet weight	n/a	n/a
Wet density of root	kg/m^3	n/a	n/a
Wet mass per soil area	kg/m^2	n/a	n/a
Stem Compartment Type - Nonwoody Only			
Allow exchange	1=yes, 0=no	n/a	n/a
Correction exponent, octanol to lipid	unitless	n/a	n/a
Density of phloem fluid	kg/m^3	n/a	n/a
Density of xylem fluid	kg/cm^3	n/a	n/a
Flow rate of transpired water per leaf area	m^3[water]/m^2[leaf]	n/a	n/a
Fraction of transpiration flow rate that is phloem rate	unitless	n/a	n/a
Lipid content of stem	kg/kg wet weight	n/a	n/a
Water content of stem	unitless	n/a	n/a
Wet density of stem	kg/m^3	n/a	n/a
Wet mass per soil area	kg/m^2	n/a	n/a

Exhibit 1-8. Non-Chemical-Dependent Terrestrial Plant Parameters

Parameter Name	Units	Grass/Herb [a]	
		Value Used	Reference
Leaf Compartment Type			
Allow exchange	1=yes, 0=no	seasonal [b]	-
Average leaf area index	m^2[leaf]/ m^2[area]	5.0	Mid-range of 4-6 for old fields, R.J. Luxmoore, ORNL
Calculate wet dep interception fraction (boolean)	1=yes, 0=no	0	Professional judgment
Correction exponent, octanol to lipid	unitless	0.76	From roots, Trapp 1995
Degree stomatal opening	unitless	1	Set to 1 for daytime based on professional judgment (stomatal diffusion is turned off at night using a different property, IsDay)
Density of wet leaf	kg/m^3	820	Paterson et al. 1991
Leaf wetting factor	m	3.00E-04	1E-04 to 6E-04 for different crops and elements, Muller and Prohl 1993
Length of leaf	m	0.05	Professional judgment
Lipid content	kg/ kg wet weight	0.00224	European beech, Riederer 1995
Litter fall rate	1/day	seasonal [c]	-
Stomatal area normalized effective diffusion path length	1/m	200	Wilmer and Fricker 1996
Vegetation attenuation factor	m^2/kg	2.9	Grass/hay, Baes et al. 1984
Water content	unitless	0.8	Paterson et al. 1991
Wet dep interception fraction	unitless	0.2	Calculated based on 5 years of local met data, 1987-1991
Wet mass of leaf per soil area	kg[fresh leaf]/ m^2[area]	0.6	Calculated from leaf area index and Leith 1975
Particle on Leaf Compartment Type			
Allow exchange	1=yes, 0=no	seasonal [b]	-
Volume particle per area leaf	m^3[leaf particles]/m^2[leaf]	1.00E-09	Based on particle density and size distribution for atmospheric particles measured on an adhesive surface, Coe and Lindberg 1987

Exhibit 1-8. Non-Chemical-Dependent Terrestrial Plant Parameters

Parameter Name	Units	Grass/Herb [a]	
		Value Used	Reference
Root Compartment Type - Nonwoody Only			
Allow exchange	1=yes, 0=no	seasonal [b]	-
Correction exponent, octanol to lipid	unitless	0.76	Trapp 1995
Lipid content of root	kg/kg wet weight	0.011	Calculated
Water content of root	kg/kg wet weight	0.8	Professional judgment
Wet density of root	kg/m^3	820	Soybean, Paterson et al. 1991
Wet mass per soil area	kg/m^2	1.4	Temperate grassland, Jackson et al. 1996
Stem Compartment Type - Nonwoody Only			
Allow exchange	1=yes, 0=no	seasonal [b]	-
Correction exponent, octanol to lipid	unitless	0.76	Trapp 1995
Density of phloem fluid	kg/m^3	1,000	Professional judgment
Density of xylem fluid	kg/cm^3	900	Professional judgment
Flow rate of transpired water per leaf area	m^3[water]/m^2[leaf]	0.0048	Crank et al. 1981
Fraction of transpiration flow rate that is phloem rate	unitless	0.05	Paterson et al. 1991
Lipid content of stem	kg/kg wet weight	0.00224	Leaves of European beech, Riederer 1995
Water content of stem	unitless	0.8	Paterson et al. 1991
Wet density of stem	kg/m^3	830	Professional judgment
Wet mass per soil area	kg/m^2	0.24	Calculated from leaf and root biomass density based on professional judgment

Exhibit 1-8. Non-Chemical-Dependent Terrestrial Plant Parameters

Parameter Name	Units	Agriculture [a]	
		Value Used	Reference
Leaf Compartment Type			
Allow exchange	1=yes, 0=no	seasonal[b]	-
Average leaf area index	m^2[leaf]/ m^2[area]	2.0	Mid-range of 4-6 for old fields, R.J. Luxmoore, ORNL
Calculate wet dep interception fraction (boolean)	1=yes, 0=no	0	Professional judgment
Correction exponent, octanol to lipid	unitless	0.76	From roots, Trapp 1995
Degree stomatal opening	unitless	1	Set to 1 for daytime based on professional judgment (stomatal diffusion is turned off at night using a different property, IsDay)
Density of wet leaf	kg/m^3	820	Paterson et al. 1991
Leaf wetting factor	m	3.00E-04	1E-04 to 6E-04 for different crops and elements, Muller and Prohl 1993
Length of leaf	m	0.1	Professional judgment
Lipid content	kg/ kg wet weight	0.00224	European beech, Riederer 1995
Litter fall rate	1/day	seasonal [c]	-
Stomatal area normalized effective diffusion path length	1/m	200	Wilmer and Fricker 1996
Vegetation attenuation factor	m^2/kg	2.9	Grass/hay, Baes et al. 1984
Water content	unitless	0.8	Paterson et al. 1991
Wet dep interception fraction	unitless	0.2	Calculated based on 5 years of local met data, 1987-1991
Wet mass of leaf per soil area	kg[fresh leaf]/ m^2[area]	0.4	Calculated from leaf area index and Leith 1975
Particle on Leaf Compartment Type			
Allow exchange	1=yes, 0=no	seasonal [b]	-
Volume particle per area leaf	m^3[leaf particles]/m^2[leaf]	1.00E-09	Based on particle density and size distribution for atmospheric particles measured on an adhesive surface, Coe and Lindberg 1987

Exhibit 1-8. Non-Chemical-Dependent Terrestrial Plant Parameters

Parameter Name	Units	Agriculture [a]	
		Value Used	Reference
Root Compartment Type - Nonwoody Only			
Allow exchange	1=yes, 0=no	seasonal [b]	-
Correction exponent, octanol to lipid	unitless	0.76	Trapp 1995
Lipid content of root	kg/kg wet weight	0.011	Calculated
Water content of root	kg/kg wet weight	0.8	Professional judgment
Wet density of root	kg/m^3	820	Soybean, Paterson et al. 1991
Wet mass per soil area	kg/m^2	0.16	Temperate grassland, Jackson et al. 1996
Stem Compartment Type - Nonwoody Only			
Allow exchange	1=yes, 0=no	seasonal [b]	-
Correction exponent, octanol to lipid	unitless	0.76	Trapp 1995
Density of phloem fluid	kg/m^3	1,000	Professional judgment
Density of xylem fluid	kg/cm^3	900	Professional judgment
Flow rate of transpired water per leaf area	m^3[water]/m^2[leaf]	0.0048	Crank et al. 1981
Fraction of transpiration flow rate that is phloem rate	unitless	0.05	Paterson et al. 1991
Lipid content of stem	kg/kg wet weight	0.00224	Leaves of European beech, Riederer 1995
Water content of stem	unitless	0.8	Paterson et al. 1991
Wet density of stem	kg/m^3	830	Professional judgment
Wet mass per soil area	kg/m^2	0.15	Calculated from leaf and root biomass density based on professional judgment

[a] See separate table for assignment of plant types to surface soil compartments.

[b] Begins March 9 (set to 1), ends November 7 (set to 0). Nation-wide 80th percentile.

[c] Begins November 7, ends December 6; rate = 0.15/day during this time (value assumes 99 percent of leaves fall in 30 days).

Exhibit 1-9. Surface Water Non-Chemical-Dependent Properties

Parameter Name	Units	Value Used	Reference
Constant Across All Water Bodies			
Algae carbon content (fraction)	unitless	0.465	APHA 1995
Algae density in water column	g[algae]/L[water]	0.0025	Millard et al. 1996 as cited in ICF 2005
Algae growth rate	1/day	0.7	Hudson et al. 1994 as cited in Mason et al. 1995b
Algae radius	um	2.5	Mason et al. 1995b
Algae water content (fraction)	unitless	0.9	APHA 1995
Average algae cell density (per vol cell, not water)	g[algae]/m^3[algae]	1,000,000	Mason et al. 1995b, Mason et al. 1996
Boundary layer thickness above sediment	m	0.02	Cal EPA 1993
Chloride concentration	mg/L	8.0	Kaushal et al. 2005
Chlorophyll concentration	mg/L	0.0029	ICF 2005
Dimensionless viscous sublayer thickness	unitless	4	Ambrose et al. 1995
Drag coefficient for water body	unitless	0.0011	Ambrose et al. 1995
Fraction Sand	unitless	0.25	Professional judgment
Organic carbon fraction in suspended sediments	unitless	0.02	Professional judgment
pH	unitless	7.3	Professional judgment
Suspended sediment deposition velocity	m/day	2	US EPA 1997
Total suspended sediment concentration	kg[sediment]/m3[water column]	0.01	US EPA 2005
Water temperature	degrees K	287	US EPA 2005

Exhibit 1-9. Surface Water Non-Chemical-Dependent Properties

Parameter Name	Units	Value Used	Reference
Water Body-specific Inputs			
Alcove Reservoir			
Flush rate	1/year	0.51	Calculated based on pond dimensions and flow calculations.
Depth [VE property] [a]	m	9.25	WI DNR 2005 - calculation based on relationship between drainage basin and lake area size.
Nassau Lake			
Flush rate	1/year	4.17	Calculated based on pond dimensions and flow calculations.
Depth [VE property] [a]	m	2.90	WI DNR 2005 - calculation based on relationship between drainage basin and lake area size.
Kinderhook Lake			
Flush rate	1/year	3.35	Calculated based on pond dimensions and flow calculations.
Depth [VE property] [a]	m	4.70	WI DNR 2005 - calculation based on relationship between drainage basin and lake area size.
Pond			
Flush rate	1/year	10.30	Calculated based on pond dimensions and flow calculations.
Depth [VE property] [a]	m	2.90	WI DNR 2005 - calculation based on relationship between drainage basin and lake area size.
Hudson River			
Flush rate	1/year	87.04	Calculated based on pond dimensions and flow calculations.
Depth [VE property] [a]	m	6.00	WI DNR 2005 - calculation based on relationship between drainage basin and lake area size.
Current Velocity	m/s	0.088	Professional judgment

[a] Set using the volume element properties named "top" and "bottom."

Exhibit 1-10. Sediment Non-Chemical-Dependent Parameters

Parameter Name	Units	Value Used	Reference
Depth [VE property] [a]	m	0.05	McKone et al. 2001 (Table 3)
Fraction Sand	unitless	0.25	Professional judgment
Organic carbon fraction	unitless	0.02	McKone et al. 2001 (Table 3)
Porosity of the sediment zone	volume[total pore space]/ volume[sediment compartment]	0.6	US EPA 1998
Solid material density in sediment	kg[sediment]/m^3[sediment]	2,600	McKone et al. 2001 (Table 3)

[a] Set using the volume element properties named "top" and "bottom."

Exhibit 1-11. Aquatic Plant Non-Chemical-Dependent Parameters

Parameter Name	Units	Value Used	Reference
Biomass per water area	kg/m^2	0.6	Bonar et al. 1993
Density of macrophytes	kg/L	1	professional judgment

Exhibit 1-12. Aquatic Animal Non-Chemical-Dependent Parameters [a]

Aquatic Biota (Consuming Organism)	Fraction Diet								Biomass (kg/m^2)	Body Weight	Reference
	Algae	Bethic Invertebrate	Macrophyte	Water Column Herbivore	Water Column Omnivore	Water Column Carnivore	Benthic Omnivore	Benthic Carnivore			
Alcove Reservoir											
Water column herbivore	96%	4%	-	-	-	-	-	-	1.30E-03	0.025	Professional judgment
Water column omnivore	8%	53%	-	39%	-	-	-	-	3.34E-03	0.25	Professional judgment
Water column carnivore	-	41%		4%	29%	-	25%	-	9.44E-04	2.0	Professional judgment
Benthic omnivore	-	100%	-	-	-	-	-	-	2.40E-03	2.0	Professional judgment
Benthic carnivore	-	50%	-	-	-	-	50%	-	1.60E-05	2.0	Professional judgment
Benthic invertebrate	-	-	-	-	-	-	-	-	2.00E-02	2.55E-04	Professional judgment
Kinderhook Lake											
Water column herbivore	82%	5%	14%	-	-	-	-	-	1.06E-03	0.025	Professional judgment
Water column omnivore	8%	58%	-	34%	-	-	-	-	2.95E-03	0.25	Professional judgment
Water column carnivore	-	33%	-	8%	34%	-	26%	-	3.60E-04	2.0	Professional judgment
Benthic omnivore	-	100%	-	-	-	-	-	-	6.25E-04	2.0	Professional judgment
Benthic carnivore	-	50%	-	-	-	-	50%	-	1.50E-05	2.0	Professional judgment
Benthic invertebrate	-	-	-	-	-	-	-	-	2.00E-02	2.55E-04	Professional judgment
Nassau Lake											
Water column herbivore	92%	5%	3%	-	-	-	-	-	9.15E-04	0.025	Professional judgment
Water column omnivore	9%	61%	-	30%	-	-	-	-	2.73E-03	0.25	Professional judgment
Water column carnivore	-	-	-	25%	50%	-	25%	-	8.00E-05	2.0	Professional judgment
Benthic omnivore	-	100%	-	-	-	-	-	-	1.25E-03	2.0	Professional judgment
Benthic carnivore	-	50%	-	-	-	-	50%	-	2.50E-05	2.0	Professional judgment
Benthic invertebrate	-	-	-	-	-	-	-	-	2.00E-02	2.55E-04	Professional judgment
Pond											
Fish harvester [b]	-	-	-	-	-	33%	67%	-	3.57E-03	71.4	Professional judgment
Water column herbivore	100%	-	-	-	-	-	-	-	8.00E-04	0.025	Professional judgment
Water column carnivore	-	50%	-	50%	-	-	-	-	2.00E-04	2.0	Professional judgment
Benthic omnivore	-	100%	-	-	-	-	-	-	3.00E-03	2.0	Professional judgment
Benthic invertebrate	-	-	-	-	-	-	-	-	2.00E-02	2.55E-04	Professional judgment

[a] For more information, see Section 4 of this attachment.

[b] Fish harvester is only used in some model runs and is parameterized as one human fisherman. In was modeled as a human-sized mink in TRIM.FaTE, as a compartment for humans does not exist.

Exhibit 1-13. Mercury Chemical-Specific Properties [a]

Parameter Name	Units	Value [b]			Reference
		Hg(0)	Hg(2)	MHg	
CAS number	unitless	7439-97-6	14302-87-5	2296-92-6	-
Diffusion coefficient in pure air	m^2[air]/day	0.478	0.478	0.456	US EPA 1997
Diffusion coefficient in pure water	m^2[water]/day	5.54E-05	5.54E-05	5.28E-05	US EPA 1997
Henry's Law constant	Pa-m^3/mol	719	7.19E-05	0.0477	US EPA 1997
Melting point	degrees K	234	550	443	CARB 1994
Molecular weight	g/mol	201	201	216	US EPA 1997
Octanol-water partition coefficient (Kow)	L[water]/kg[octanol]	4.15	3.33	1.7	Mason, et al. 1996
Vapor washout ratio	m^3[air]/m^3[rain]	1,200	1.6E+06	0	US EPA 1997, based on Petersen et al. 1995

[a] All parameters in this table are TRIM.FaTE chemical properties.

[b] On this and all following tables, Hg(0) = elemental mercury, Hg(2) = divalent mercury, and MHg = methyl mercury.

Exhibit 1-14. Chemical-Specific Properties for 2,3,7,8-TCDD

Parameter Name	Units	Value	Reference
CAS number	unitless	1746-01-6	-
Diffusion coefficient in pure air	m^2/day	0.106	US EPA 1999
Diffusion coefficient in pure water	m^2/day	5.68E-05	US EPA 1999
Henry's Law constant	Pa-m^3/mol	3.33	Mackay et al. 1992 as cited in U.S. EPA 2000a
Melting point	degrees K	578.0	Mackay et al. 2000
Molecular weight	g/mol	322.0	Mackay et al. 2000
Octanol-water partition coefficient (Kow)	L[water]/L[octanol]	6.31E+06	Mackay et al. 1992a as cited in U.S. EPA 2000a

Exhibit 1-15. Mercury Chemical-Specific Properties for Abiotic Compartments

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Air Compartment Type					
Particle dry deposition velocity	m/day	500	500	500	CalTOX value cited in McKone et al. 2001
Demethylation rate	1/day	N/A	N/A	0	Professional judgment
Methylation rate	1/day	0	0	0	Professional judgment
Oxidation rate	1/day	0.00385	0	0	Low end of half-life range (6 months to 2 years) in EPA 1997
Reduction rate	1/day	0	0	0	Professional judgment
Washout Ratio	m3[air]/m3[rain]	200,000	200,000	200,000	Professional judgment
Surface Soil Compartment Type					
Input characteristic depth (user supplied)	m	0.08	0.08	0.08	Not used (model set to calculate value)
Soil-water partition coefficient	L[water]/ kg[soil wet wt]	1,000	58,000	7,000	U.S. EPA 1997
Use input characteristic depth (boolean)	0 = no, Else = yes	0	0	0	Professional judgment
Vapor dry deposition velocity	m/day	50	2500	0	Hg(0) - from Lindberg et al. 1992 Hg(2) - estimate by U.S.EPA using the Industrial Source Complex (ISC) Model - [See Vol. III, App. A of the Mercury Study Report (USEPA, 1997)].
Demethylation rate	1/day	N/A	N/A	0.06	Range reported in Porvari and Verta 1995 is 3E-2 to 6E-2 /day; value is average maximum potential demethylation rate constant under anaerobic conditions
Methylation rate	1/day	0	0.001	0	Range reported in Porvari and Verta 1995 is 2E-4 to 1E-3 /day; value is average maximum potential methylation rate constant under anaerobic conditions
Oxidation rate	1/day	0	0	0	Value assumed in EPA 1997
Reduction rate	1/day	0	1.25E-05	0	Value used for untilled surface soil (2cm), 10% moisture content, in U.S. EPA 1997; general range is (0.0013/day)*moisture content to (0.0001/day)*moisture content for forested region (Lindberg 1996; Carpi and Lindberg 1997)

Exhibit 1-15. Mercury Chemical-Specific Properties for Abiotic Compartments

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Root Zone Soil Compartment Type					
Input characteristic depth (user supplied)	m	0.08	0.08	0.08	Not used (model set to calculate value)
Soil-water partition coefficient	L[water]/kg[soil wet wt]	1,000	58,000	7,000	U.S. EPA 1997
Use input characteristic depth (boolean)	0 = no, Else = yes	0	0	0	Professional judgment
Demethylation rate	1/day	N/A	N/A	0.06	Range reported in Porvari and Verta 1995 is 3E-2 to 6E-2 /day; value is average maximum potential demethylation rate constant under anaerobic conditions
Methylation rate	1/day	0	0.001	0	Range reported in Porvari and Verta 1995 is 2E-4 to 1E-3 /day; value is average maximum potential methylation rate constant under anaerobic conditions
Oxidation rate	1/day	0	0	0	Value assumed in U.S. EPA 1997
Reduction rate	1/day	0	3.25E-06	0	Value used for tilled surface soil (20cm), 10% moisture content, in U.S. EPA 1997 (Lindberg 1996; Carpi and Lindberg, 1997)
Vadose Zone Soil Compartment Type					
Input characteristic depth (user supplied)	m	0.08	0.08	0.08	Not used (model set to calculate value)
Soil-water partition coefficient	L[water]/kg[soil wet wt]	1,000	58,000	7,000	U.S. EPA 1997
Use input characteristic depth (boolean)	0 = no, Else = yes	0	0	0	Professional judgment
Demethylation rate	1/day	N/A	N/A	0.06	Range reported in Porvari and Verta 1995 is 3E-2 to 6E-2 /day; value is average maximum potential demethylation rate constant under anaerobic conditions
Methylation rate	1/day	0	0.001	0	Range reported in Porvari and Verta 1995 is 2E-4 to 1E-3 /day; value is average maximum potential methylation rate constant under anaerobic conditions
Oxidation rate	1/day	0	0	0	Value assumed in U.S. EPA 1997
Reduction rate	1/day	0	3.25E-06	0	Value used for tilled surface soil (20cm), 10% moisture content, in U.S. EPA 1997 (Lindberg 1996; Carpi and Lindberg, 1997)

Exhibit 1-15. Mercury Chemical-Specific Properties for Abiotic Compartments

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Ground Water Compartment Type					
Soil-water partition coefficient	L[water]/kg[soil wet wt]	1,000	58,000	7,000	U.S. EPA 1997
Demethylation rate	1/day	N/A	N/A	0.06	Range reported in Porvari and Verta 1995 is 3E-2 to 6E-2 /day; value is average maximum potential demethylation rate constant under anaerobic conditions
Methylation rate	1/day	0	0.001	0	Range reported in Porvari and Verta 1995 is 2E-4 to 1E-3 /day; value is average maximum potential methylation rate constant under anaerobic conditions
Oxidation rate	1/day	1.00E-08	0	0	Small default nonzero value (0 assumed in U.S. EPA 1997)
Reduction rate	1/day	0	3.25E-06	0	Value used for tilled surface soil (20cm), 10% moisture content, in U.S. EPA 1997 (Lindberg 1996; Carpi and Lindberg, 1997)
Surface Water Compartment Type					
Algal surface area-specific uptake rate constant	nmol/[μm^2-day-nmol]	0	2.04E-10	3.60E-10	Assumes radius = 2.5mm, Mason et al. 1995b, Mason et al. 1996; Hg(0) assumed same as Hg(2)
Dow ("overall Kow")	L[water]/ kg[octanol]	0	-[a]	-[b]	Mason et al. 1996
Solids-water partition coefficient	L[water]/ kg[solids wet wt]	1,000	100,000	100,000	U.S. EPA 1997
Vapor dry deposition velocity	m/day	N/A	2500		U.S. EPA 1997 (Vol. III, App. A)
Demethylation rate	1/day	N/A	N/A	0.013	Average of range of 1E-3 to 2.5E-2/day from Gilmour and Henry 1991
Methylation rate	1/day	0	0.001	0	Value used in EPA 1997; range is from 1E-4 to 3E-4/day (Gilmour and Henry 1991)
Oxidation rate	1/day	0	0	0	Professional judgment
Reduction rate	1/day	0	0.0075	0	Value used in EPA 1997; reported values range from less than 5E-3/day for depths greater than 17m, up to 3.5/day (Xiao et al. 1995; Vandal et al. 1995; Mason et al. 1995a; Amyot et al. 1997)

Exhibit 1-15. Mercury Chemical-Specific Properties for Abiotic Compartments

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Sediment Compartment Type					
Solids-water partition coefficient	L[water]/ kg[solids wet wt]	3,000	50,000	3,000	U.S. EPA 1997
Demethylation rate	1/day	N/A	N/A	0.0501	Average of range of 2E-4 to 1E-1/day from Gilmour and Henry 1991
Methylation rate	1/day	0	1.00E-04	0	Value used in EPA 1997; range is from 1E-5 to 1E-3/day,Gilmour and Henry 1991
Oxidation rate	1/day	0	0	0	Professional judgment
Reduction rate	1/day	0	1.00E-06	0	Inferred value based on presence of Hg(0) in sediment porewater (U.S. EPA 1997; Vandal et al. 1995)

[a] TRIM.FaTE Formula Property, which varies from 0.025 to 1.625 depending on pH and chloride concentration.

[b] TRIM.FaTE Formula Property, which varies from 0.075 to 1.7 depending on pH and chloride concentration.

Exhibit 1-16. Chemical-Specific Properties of 2,3,7,8-TCDD for Abiotic Compartments

Parameter Name	Units	Value	Reference
Air Compartment Type			
Deposition Velocity	m/day	500	McKone et al. 2001
Halflife	day	12	Atkinson 1996 as cited in USEPA 2000; vapor phase reaction with hydroxyl radical
Washout Ratio	m^3[air]/m^3[rain]	18000	Vulykh et al. 2001
Surface Soil Compartment Type			
Input characteristic depth	m	0.08	Not used (model set to calculate value)
Use input characteristic depth (boolean)	0 = No, Else = Yes	0	Professional judgment
Halflife	day	3650	Mackay et al. 2000; the degradation rate was cited by multiple authors, value is for 2,3,7,8-TCDD
Root Zone Soil Compartment Type			
Input characteristic depth	m	0.08	Not used (model set to calculate value)
Use input characteristic depth	0 = No, Else = Yes	0	Professional judgment
Halflife	day	3650	Mackay et al. 2000; the degradation rate was cited by multiple authors, value is for 2,3,7,8-TCDD
Vadose Zone Soil Compartment Type			
Input characteristic depth	m	0.08	Not used (model set to calculate value)
Use input characteristic depth (boolean)	0 = No, Else = Yes	0	Professional judgment
Halflife	day	1008	Average value of the range presented in Mackay et al. 2000; based on estimated unacclimated aerobic biodegradation half-life, value is for 2,3,7,8-TCDD
Groundwater Compartment Type			
Half-life	day	1008	Average value of the range presented in Mackay et al. 2000; based on estimated unacclimated aerobic biodegradation half-life, value is for 2,3,7,8-TCDD
Surface Water Compartment Type			
RatioOfConcInAlgaeToConcDissolvedInWater	(g[chem]/g[algae]) / (g[chem]/L[water])	1.025	BCF data for green algae for 2,3,7,8-TCDD from Isense 1978, at 32 days
Half-life	day	2.7	Kim, M., and P. O'Keefe. 1998. as cited in U.S. EPA. 2000.
Sediment Compartment Type			
Half-life	day	1095	Estimation based on Adriaens and Grbic-Galic 1992,1993 and Adriaens et al. 1995 as cited in U.S. EPA 2000.

Exhibit 1-17. Mercury Chemical-Specific Properties for Plants [a]

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Terrestrial Plants					
Leaf Compartment Type					
Transfer factor to leaf particle	1/day	0.002	0.002	0.002	Professional judgment (assumed 1% of transfer factor from leaf particle to leaf)
Demethylation rate	1/day	N/A	N/A	0.03	Calculated from Bache et al. 1973
Methylation rate	1/day	0	0	0	Assumed from Gay 1975, Bache et al. 1973
Oxidation rate	1/day	1.0E+06	0	0	Professional judgment; Assumed close to instantaneous
Reduction rate	1/day	0	0	0	Professional judgment
Particle on Leaf Compartment Type					
Transfer factor to leaf	1/day	0.2	0.2	0.2	Professional judgment
Demethylation rate	1/day	N/A	N/A	0	Professional judgment
Methylation rate	1/day	0	0	0	Professional judgment
Oxidation rate	1/day	0	0	0	Professional judgment
Reduction rate	1/day	0	0	0	Professional judgment
Root Compartment Type - Nonwoody Plants Only [b]					
Alpha for root-root zone bulk soil	unitless	0.95	0.95	0.95	Selected value
Root/root-zone-soil-water partition coefficient	m^3[bulk root soil]/m^3[root]	0	0.18	1.2	Hg2- geometric mean Leonard et al. 1998, John 1972, Hogg et al. 1978; MHg- assumed, based on Hogg et al. 1978
t-alpha for root-root zone bulk soil	day	21	21	21	Professional judgment
Demethylation rate	1/day	N/A	N/A	0	Professional judgment
Methylation rate	1/day	0	0	0	Professional judgment
Oxidation rate	1/day	0	0	0	Professional judgment
Reduction rate	1/day	0	0	0	Professional judgment

Exhibit 1-17. Mercury Chemical-Specific Properties for Plants [a]

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Stem Compartment Type - Nonwoody Plants Only					
Transpiration stream concentration factor (TSCF)	m^3[soil pore water]/m^3[xylem fluid]	0	0.5	0.2	Calculation from Norway spruce, Scots pine, Bishop et al. 1998
Demethylation rate	1/day	N/A	N/A	0.03	Calculated from Bache et al. 1973
Methylation rate	1/day	0	0	0	Professional judgment
Oxidation rate	1/day	0	0	0	Professional judgment
Reduction rate	1/day	0	0	0	Professional judgment
Aquatic Plants					
Macrophyte Compartment Type					
Water Column Dissolve Partitioning Alpha of Equilibrium	unitless	0.95	0.95	0.95	Selected value
Macrophyte/water partition coefficient	L[water]/kg[macrophyte wet wt]	0.883	q	4.4	*Elodea densa,* Ribeyre and Boudou 1994
Oxidation rate	1/day	1.00E+09	0	0	Professional judgment
t-alpha	day	18	18	18	Experiment duration from Ribeyre and Boudou 1994

[a] TRIM.FaTE currently includes four kinds of terrestrial plants: deciduous forest (not used in screening scenario),

[b] Roots and stems are not modeled for deciduous or coniferous forest in the current version of TRIM.FaTE.

Exhibit 1-18. Chemical-Specific Properties of 2,3,7,8-TCDD for Plants

Parameter Name	Units	Value	Reference
		All Dioxins	
Terrestrial Plants			
Leaf Compartment Type			
Transfer factor to leaf particle	1/day	0.003	Calculated as 1 percent of transfer factor to leaf; highly uncertain
Half-life	day	70	Arjmand and Sandermann 1985, as cited in Komoba, et al. 1995; soybean root cell culture metabolism test data for DDE
Particle on Leaf Compartment Type			
Transfer factor to leaf	1/day	0.3	Professional judgment based on U.S. EPA 2000c (an estimate for mercury) and Trapp 1995; highly uncertain
Half-life	day	4.4	McCrady and Maggard 1993; photodegradation sorbed to grass foliage in sunlight; assumed 10 sunlight per day
Root Compartment Type			
Half-life	day	70	Arjmand and Sandermann 1985, as cited in Komoba, et al. 1995; soybean root cell culture metabolism test data for DDE
Root Soil Water Interaction_Alpha	unitless	0.95	Professional judgment
Stem Compartment Type			
Half-life	day	70	Arjmand and Sandermann 1985, as cited in Komoba, et al. 1995; soybean root cell culture metabolism test data for DDE
Aquatic Plants			
Macrophyte Compartment Type			
Half-life	days	70	Arjmand and Sandermann 1985, as cited in Komoba, et al. 1995; soybean root cell culture metabolism test data for DDE

Exhibit 1-19. Mercury Chemical-Specific Properties for Aquatic Species [a]

Parameter Name	Units	Value			Reference
		Hg(0)	Hg(2)	MHg	
Benthic Invertebrate Compartment Type					
Alpha of equilibrium for sediment partitioning	unitless	0.95	0.95	0.95	Selected value
Benthic invertebrate-bulk sediment partition coefficient	kg[bulk sediment]/ kg[invertebrate wet wt]	0.0824	0.0824	5.04	Hg(0) - assumed based on Hg(2) value; Hg(2) and MHg - Saouter et al. 1991
t-alpha for equilibrium for sediment partitioning	day	14	14	14	Experiment duration from Saouter et al. 1991
All Fish Compartment Types					
Demethylation rate	1/day	N/A	N/A	0	Professional judgment
Methylation rate	1/day	0	0	0	Professional judgment
Oxidation rate	1/day	1.0E+06	0	0	Professional judgment
Reduction rate	1/day	0	0	0	Professional judgment
Water-column Carnivore Compartment Type					
Assimilation efficiency from food	unitless	0.04	0.04	0.2	Phillips and Gregory 1979
Elimination adjustment factor	unitless	3	3	1	Trudel and Rasmussen 1997
Water-column Herbivore Compartment Type					
Assimilation efficiency from food	unitless	0.04	0.04	0.2	Phillips and Gregory 1979
Elimination adjustment factor	unitless	3	3	1	Trudel and Rasmussen 1997
Water-column Omnivore Compartment Type					
Assimilation efficiency from food	unitless	0.04	0.04	0.2	Phillips and Gregory 1979
Elimination adjustment factor	unitless	3	3	1	Trudel and Rasmussen 1997
Benthic Omnivore Compartment Type					
Assimilation efficiency from food	unitless	0.04	0.04	0.2	Phillips and Gregory 1979
Elimination adjustment factor	unitless	3	3	1	Trudel and Rasmussen 1997

[a] Screening scenario includes: Benthic Omnivore, Water-column Carnivore, Water-column Herbivore, Water-column Omnivore.

Exhibit 1-20. Chemical-Specific Properties of 2,3,7,8-TCDD for Aquatic Species

Parameter Name	Units	Value	Reference
Benthic Invertebrate Compartment Type			
Clearance constant	unitless	0	Professional judgment
Sediment Partitioning Partition Coefficient	kg/kg	0.107	TCDD data for sandworm in Rubenstein et al. 1990; dry weight sediment. PeCDF: multiplied TCDD partition coefficient for sandworm by congener-specific bioaccumulation equivalency factor in GLWQI from U.S. EPA 1999.
Sediment Partitioning Alpha of Equilibrium	unitless	0.95	Professional judgment
Sediment Partitioning Time to Reach Alpha of Equilibrium	days	120	TCDD: professional judgment; PeCDF: Rubinstein et al. 1990; data for TCDF in sandworm.
V_d (ratio of concentration in benthic invertebrates to concentration in water)	ml/g	0	Professional judgment
Half-life	day	140	TCDD: estimated based on data for yellow perch in Keeman et al. 1986b; PeCDF: Sijm et al. 1990 quoted elimination rate for carp, metabolic rate calculated assuming 9% metabolites like hepta and hexa isomers as cited in Muir et al. 1986a
All Fish Compartment Types [a]			
Assimilation efficiency from food	unitless	0.5	TCDD: calculated from data in Kleeman et al. 1986b trout data as cited in U.S. EPA 1993; PeCDF: used assimilation efficiency for TCDD in trout
Gamma_fish	unitless	N/A [b]	Thomann 1989
Water Column Carnivore Compartment Type			
Chemical Uptake Rate Via Gill	L[water]/ kg[fish wet wt]-day	104	Muir et al. 1986
Half-life	day	160	TCDD: estimated based on data for rainbow trout in Kleeman et al. 1986a; PeCDF: Sijm et al. 1990 quoted elimination rate for rainbow trout, metabolic rate calculated assuming 9% metabolites like hepta and hexa isomers cited in Muir et al. 1986a
Water Column Herbivore Compartment Type			
Assimilation efficiency from plants	unitless	0.5	TCDD: calculated from data in Kleeman et al. 1986b trout data as cited in U.S. EPA 1993; PeCDF: used assimilation efficiency for TCDD in trout
Chemical Uptake Rate Via Gill	L[water]/ kg[fish wet wt]-day	380	Muir et al. 1986
Half-life	day	140	TCDD: estimated based on data for rainbow trout in Kleeman et al. 1986a; PeCDF: Sijm et al. 1990 quoted elimination rate for rainbow trout, metabolic rate calculated assuming 9% metabolites like hepta and hexa isomers cited in Muir et al. 1986a

Exhibit 1-20. Chemical-Specific Properties of 2,3,7,8-TCDD for Aquatic Species

Parameter Name	Units	Value	Reference
Water Column Omnivore Compartment Type			
Assimilation efficiency from plants	unitless	0.5	TCDD: calculated from data in Kleeman et al. 1986b trout data as cited in U.S. EPA 1993; PeCDF: used assimilation efficiency for TCDD in trout
Chemical Uptake Rate Via Gill	L[water]/ kg[fish wet wt]-day	380	Muir et al. 1986
Half-life	day	140	TCDD: estimated based on data for rainbow trout in Kleeman et al. 1986a; PeCDF: Sijm et al. 1990 quoted elimination rate for rainbow trout, metabolic rate calculated assuming 9% metabolites like hepta and hexa isomers cited in Muir et al. 1986a
Benthic Omnivore Compartment Type			
Chemical Uptake Rate Via Gill	L[water]/k g[fish wet wt]-day	380	Muir et al. 1986
Half-life	day	140	TCDD: estimated based on data for rainbow trout in Kleeman et al. 1986a; PeCDF: Sijm et al. 1990 quoted elimination rate for rainbow trout, metabolic rate calculated assuming 9% metabolites like hepta and hexa isomers cited in Muir et al. 1986a

[a]Screening scenario includes: Benthic Omnivore, Water-column Carnivore, Water-column Herbivore, Water-column Omnivore.

[b]N/A = not applicable. This parameter is used in calculating the uptake when measured data are unavailable.

I-1-2Supplemental Information for Exhibit 1-2 – Meteorological and Other Settings

I-1-2.1 PCRAMMET

The EPA's PCRAMMET meteorological data processor[1] is used to combine the surface meteorological data with the twice-daily mixing height data into the format necessary for many EPA air quality models. PCRAMMET requires that surface data be in either Solar and Meteorological Surface Observational Network (SAMSON) format, Hourly United States Weather Observations (HUSWO) format, or CD-144 format. For this study, the delimited format downloaded from the ISH database is converted into SAMSON format using the converter available from RF Lee Consulting.[2] This converter does not accurately process the precipitation information, so hourly precipitation is manually inserted into the SAMSON file that the converter produces.

PCRAMMET also requires daily morning and afternoon mixing height data, which is not an explicit field in the upper-air data from NOAA. The EPA's mixing height calculator[3] is used to generate morning and afternoon mixing heights from the FSL-formatted upper-air data. This calculator uses the Holzworth methodology,[4] which requires the morning (07 EST) upper-air sounding and the daily observed minimum and maximum hourly surface temperatures in order to perform the calculations.

PCRAMMET converts surface wind directions into vectors (i.e., converts wind directions to 'blowing to' rather than 'blowing from'). PCRAMMET also randomly applies to the wind vector a variation of -4° to +5° in order to remove the directional bias from the hourly surface reports, which record wind directions in increments of 10°. Wind directions of 0°, which indicate calm winds, are set to the wind direction of the previous non-calm hour and then randomly varied by -4° to +5°.

In PCRAMMET, the cloud layer with the greatest cloud coverage is used to represent the cloud coverage for that hour. If the ceiling height observation is missing, then PCRAMMET sets the ceiling height as the height of the lowest cloud layer with cloud coverage that is at least 'broken' (at least 6/10 cloud coverage).

PCRAMMET interpolates twice-daily mixing heights into hourly values using the maximum mixing height value from the previous, current, and upcoming day as well as the minimum height value from the current and upcoming day. Then, two different methodologies are used to derive the urban and rural mixing height values, respectively.[5]

For this study, PCRAMMET is set to calculate wet deposition fields. These fields include friction velocity, Monin-Obukhov length, and roughness length.[5]

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[1] http://www.epa.gov/scram001/metobsdata_procaccprogs.htm#pcrammet

[2] http://www.rflee.com/RFL_Pages/Meteor.html

[3] http://www.epa.gov/scram001/metobsdata_procaccprogs.htm#mixing

[4] Holzworth, G., 1967. Mixing Depths, Wind Speeds and Air Pollution Potential for Selected Locations in the United States. *J. Appl. Meteor.*, 6, 1039-1044.

[5] http://www.epa.gov/scram001/userg/relat/pcramtd.pdf

I-1-2.2 TRIM.FaTE Processing

Before the PCRAMMET-processed meteorological files can be processed by the TRIM.FaTE meteorological processor, some fields must be filtered to replace missing values. The fields of opaque sky cover, ceiling height, surface temperature, wind speed, wind direction, and mixing heights cannot have any missing values. The EPA has provided procedures for objectively replacing missing data in these fields.[6] To generally summarize the missing value procedures, missing opaque sky cover values are replaced by values that depend on the availability and values of total sky cover and ceiling height; missing ceiling height values are replaced by values that depend on the availability and values of total sky cover and opaque sky cover; and, missing surface temperature, wind direction, wind speed, and mixing height values are interpolated from the values of surrounding times. After these objective measures are used to replace missing data in these fields, any remaining missing values are subjectively and manually replaced with values based on observations from surrounding times. Exhibit 2-1 shows the completeness of the various meteorological datasets and data fields in this study.

Exhibit 2-1. Completeness of Meteorological Data Types [a]

Year	Data Type	Statistic	Before Missing Values Objectively Replaced	After Missing Values Objectively Replaced
2001	Surface Data	Hours Not Reporting	0.03%	0.03%
		Hours Missing Opaque Sky Cover	2%	0.02%
		Hours Missing Ceiling Height	0.07%	0.07%
		Hours Missing Temperature	0.1%	0.02%
		Hours Missing Wind Speed	3%	0.7%
		Hours Missing Wind Direction	6%	2%
	Upper-Air Data	Soundings Not Reporting	5%	5%
		Missing Calculated Morning Mixing Heights	7%	2%
		Missing Calculated Evening Mixing Heights	3%	0%
2002	Surface Data	Hours Not Reporting	0.06%	0.06%
		Hours Missing Opaque Sky Cover	3%	0.07%
		Hours Missing Ceiling Height	0.1%	0.1%
		Hours Missing Temperature	0.3%	0.07%
		Hours Missing Wind Speed	0.5%	0.09%
		Hours Missing Wind Direction	3%	1%
	Upper-Air Data	Soundings Not Reporting	5%	5%
		Missing Calculated Morning Mixing Heights	8%	5%
		Missing Calculated Evening Mixing Heights	6%	3%
2003	Surface	Hours Not Reporting	0.09%	0.09%

[6] See http://www.epa.gov/scram001/surface/missdata.txt.

Exhibit 2-1. Completeness of Meteorological Data Types [a]

Year	Data Type	Statistic	Before Missing Values Objectively Replaced	After Missing Values Objectively Replaced
	Data	Hours Missing Opaque Sky Cover	4%	0.05%
		Hours Missing Ceiling Height	0.2%	0.2%
		Hours Missing Temperature	2%	1%
		Hours Missing Wind Speed	0.2%	0.07%
		Hours Missing Wind Direction	3%	1%
	Upper-Air Data	Soundings Not Reporting	5%	5%
		Missing Calculated Morning Mixing Heights	6%	2%
		Missing Calculated Evening Mixing Heights	5%	0.8%

[a] The percentage of the 2001-2003 surface and upper-air reports that are completely missing ('Hours Not Reporting', 'Soundings Not Reporting'), the percentage of non-missing hourly surface reports where specific surface variables were missing, and the percentage of non-missing upper-air soundings where the morning or afternoon mixing heights could not be calculated. These percentages of missing data are also shown after the EPA's objective measures are employed to replace missing values.

Because the Ravena Lefarge Portland Cement scenario is modeled for 1990-2039, the 2001-2003 meteorological data are duplicated. First, the data from 2002 are duplicated for 2004 (with a leap day added, which was comprised of the data from 28 February) to create a complete four-year cycle of data. Then, this four-year cycle of meteorological data are duplicated to fill the modeling time period.

Finally, this 50-year set of meteorological data are processed by the TRIM.FaTE meteorological processor. The TRIM.FaTE meteorological processor reverts the PCRAMMET-process wind directions back into 'blowing from' designation and it converts hourly precipitation amounts (previously in mm) to a daily precipitation rate in meters ($m\ day^{-1}$). Calm wind speeds are set to 0.75 ms^{-1} so that chemical advection is always occurring. Mixing heights are set to a minimum of 20 m. Daytime and nighttime hours are identified by inputting the latitude, longitude, and US time zone of the meteorology station.

I-1-3Supplemental Information for Exhibit 1-6 – Universal Soil Loss Equations

I-1-3.1 Universal Soil Loss Equation

Sediment delivery for the parcels in this scenario were determined by using the Universal Soil Loss Equation (USLE). The USLE is the most widely used empirical soil erosion model, which estimates soil erosion from the product of empirically derived coefficients (Amore et al. 2004). The values for these coefficients, and the equation itself, have been derived from over 10,000 plot-years of runoff and soil loss data (Pilotti et al. 1977).

The formula for the USLE is the product of five factors, as shown in the equation below:

$$A = R * K * LS * C * P$$

where:

A = Total
R = Rainfall/erosivity factor
K = Soil erodibility factor
LS = Combined length-slope factor
C = Cover management factor
P = Supporting practice factor

The USLE is intended to predict the long-term average soil losses from individual field areas (Wischmeier and Smith, 1978), and represent the sheet and rill erosion from a small plot or agricultural field. Application of the USLE to an entire watershed requires modification of the result of the equation to account for subsequent re-deposition of eroded soil before reaching the water body. The sediment delivery ratio, further described in Section 3.9., was developed for this purpose, and is an additional factor to determine the amount of sediment that reaches a water body based on watershed size (Vanoni 1975 in EPA 2005a).

Representative values were determined for each parcel use in the USLE, as outlined below.

I-1-3.2 Rainfall/erosivity Factor (R)

The rainfall/erosivity factor represents the erosive potential of the typical rainfall over a given period (Wischmeier and Smith, 1978), and due to the typical cyclic nature of rainfall in a given area can be considered constant for a given location. R values for this scenario were looked up from county specific data in the Revised Universal Soil Loss Equation 2 (RUSLE2) software (RUSLE2 was not used to calculate erosion predictions directly due to the intensive site analysis required for this process, as discussed below). Data was available for both Albany county and Rensselaer county; therefore, values for parcels located west of the Hudson river were assumed to have R values the same as Albany county, and values for parcels located east of the Hudson were assumed to have R values the same as Rensselaer county. These values were consistent with regional maps of R values.

I-1-3.3 Soil Erodibility Factor (K)

Specific soil types have different natural susceptibilities to erosion, depending on the specific makeup of their components (Wischmeier and Smith, 1978). To determine the site-specific K values of the soils around the location in Ravena, NY, soil data was obtained from the Soil Survey Geographic (SSURGO) database for the counties of interest, in the form of GIS

shapefiles (obtained from Natural Resources Conservation Service with the USDA). The percentage of each soil type present in a parcel was determined by including all polygons with their centroid within the parcel. An area weighted average was determined for the three parcels most driving risk with respect to sediment delivery – the three lake watershed parcels. Given the very close similarity of these values, especially in comparison to the greater influence of assumptions related to other USLE variables, the rest of the non-watershed parcels were assigned the average value of these three parcels. Values for all parcels are given in Exhibit 3-1 below.

Exhibit 3-1. Soil Erodibility Factor for Watershed Parcels and All Other Parcels

Watershed Parcel	Water Body	Soil Erodibility Factor (ton/acre/(100 ft-ton/acre))
E2	Nassau Lake	0.3113
E3	Kinderhook Lake	0.3102
W3	Alcove Reservoir	0.3222
All other parcels	N/A	0.3145

I-1-3.4 Length Slope (LS) Factor

The amount of soil eroded from a given field increases as the slope increases, and as the length of the field increases. For this assessment, the length slope factor was calculated from the equation provided in Wischmeier and Smith (1978).

This equation for the length-slope factor is:

$$LS = (\frac{\lambda}{72.6})^{m}(65.41\sin^{2}\theta + 4.56\sin\theta + 0.65)$$

where:

- λ = the slope length in feet
- θ = angle of slope
- m = 0.5 if the slope is 5% or more, 0.4 for slopes of 3.5-4.5%, 0.3 for slopes of 1-3%, and 0.2 on slopes of less than 1 percent.

The field length is measured from the start of erosion to either a well defined channel or decrease in slope sufficient enough for deposition to occur (Wischmeier and Smith, 1978). This value would be different for each specific slope within a watershed, and exact evaluation of the slope length requires detailed analysis of the watershed topography and evaluation of the USLE for each slope. As an approximation for evaluation purposes, and consistent with the Dioxin Reassessment (EPA 2004), an average field size was assumed to be 4 hectares. A square field of 4 hectares translates to a side length of 200 meters. The length of a slope was assumed, therefore, to be 200 meters in length, or 656 feet.

Average slope for each parcel was determined from GIS data of the topography of the four counties included. This average slope was used in calculating the LS factor for each parcel.

I-1-3.5 Cover Management Factor (C)

The type of ground cover present on a field plays a major factor in determining the amount of soil eroded from a slope. Values of the cover management factor can range from less than 0.001 for dense grasses and undisturbed forestland to 1 for bare construction sites. C values were determined from guidance in Wischmeier and Smith, RUSLE2 values for specific crop cycles, and GIS data on land use for the area of interest.

For the four farm parcels, representative C values were looked up for specific crop types from the RUSLE2 software. For untilled parcels, C values for various grain crops ranged from 0.015 to 0.07, from which a conservative value of 0.07 was assumed (representative of no till corn 50 bushels/acre). For the tilled parcels, an area weighted C value was determined for the top three vegetable crops reported in the agricultural census. This average covered 85% of all vegetable crops reported. This method was seen to best represent the C value of farmland in the region, as the most grown crop (sweet corn) also had the lowest C value, and as such using the C value for only this crop would likely under-predict sediment delivery. Values for watermelons/cucumbers were used as a surrogate for pumpkins, and green beans were used as a surrogate for tomatoes, due to similar plant and growing styles.

For the non-farm parcels, area weighted cover management factors were determined from the top three reported land uses in that parcel. Values were determined from tables provided by Wischmeier and Smith (1978). Deciduous forest and Evergreen forest land uses were to have 75% tree cover, with weeds below at 80% ground cover. Mixed forests were assumed to 50% tree cover and weeds for ground cover, also at 80%. Pasture/hay was assumed to have 80% ground cover. Finally, quarries/strip mines/gravel pits were assumed to have a C value of 1, consistent with no ground cover on construction sites.

I-1-3.6 Supporting Practice Factor (P)

Supporting practices include contour tillage, strip-cropping on the contour, and terracing. For this assessment, no supporting practices were assumed, and therefore a value of 1 was assigned for all parcels.

I-1-3.7 Total Erosion Losses Per Parcel

By using the above described approach, erosion rates were estimated for each parcel. The values of these erosion losses were developed on a per-area basis; however, differences in cover type, soil, and slope in the parcels yielded different per-area erosion rates. The calculated rates are presented in Section 3.9.

I-1-3.8 Limitations to This Approach

The USLE is an empirical model, and therefore modeled conditions must be similar to conditions for which the USLE is calibrated. In particular, the USLE is designed for application to a single slope or field, rather than a whole watershed. Using average values across a watershed parcel will likely introduce uncertainties in the prediction that would be better predicted by individual analyses of the slopes within the watershed. It is noted in the HHRAP documentation that using the universal soil loss equation to calculate sediment load to a lake from the surrounding watershed can sometimes lead to overestimates (EPA 2005a).

The use of area weighted averages for some of the USLE variables does help to avoid this problem in not under- or over-estimating by assuming uniformity across the watershed. The

area weighted K (soil erodibility factor) and C (cover management factor) are not expected to contribute significantly to an inaccurate estimate of soil erosion.

Estimation of the LS factor poses more problems than any other factor of the USLE (Moore & Wilson, 1992), especially in complex watersheds. In the real watershed, an entire watershed has neither uniform slope length nor uniform slope steepness. Additionally, due to nonlinearities in the equation to calculate the LS factor, this assumption can introduce uncertainties into the degree by which the LS factor is under- or over-estimated. The use of average slope likely will under-predict the LS factor by some small degree. The use of an average slope-length of 200 meters may be accurate or slightly longer than average, and therefore may slightly over-predict the LS factor by an unknown amount.

Finally, there is uncertainty in the use of the sediment delivery ratio (SD) to account for the re-deposition of soil before it reaches the water body. It is not known the degree by which the SD ratio will under- or over- predict actual sediment delivery.

I-1-3.9 Sediment Balance Calculations

The sediment balance of the watershed is determined by accounting for both the inputs of sediment from the erosion calculations and the outputs of sediment through removal and burial. In this scenario, assumptions about the physical environment were used in calculation of the sediment input through erosion and removal through suspended sediment flushing. All sediment inputs to the watershed come from the erosion calculations. The sediment delivery ratio accounts for how much of that is re-deposited within the watershed.

The sediment delivery ratio is calculated using the following equation:

$$SD = a(A\_L)^{-b}$$

where:

SD = sediment delivery ratio
a = empirical intercept coefficient
A_L = total watershed area receiving deposition
b = empirical slope coefficient

The value of the empirical intercept coefficient is determined based on watershed area (see Exhibit 3-2). The empirical slope coefficient is a unitless constant set to 0.125.

Exhibit 3-2. USLE Empirical Intercept Coefficient

Area of Watershed (sq. miles)	*a*
Area ≤ 0.1	2.1
0.1 < Area ≤ 1	1.9
1 < Area ≤ 10	1.4
10 < Area ≤ 100	1.2
Area > 100	0.6

Each parcel's sediment delivery ratio was calculated based on its size, and results are presented below in Exhibit 3-3. Finally, the adjusted erosion rate was calculated by multiplying

the USLE erosion rate by the sediment delivery ratio. The adjusted erosion rates are the final erosion values used in the TRIM scenario.

Exhibit 3-3. Calculated USLE Soil Erosion Rates, Sediment Delivery Ratios, and Adjusted Erosion Rates for Each Soil Parcel

Parcel	Erosion Rate (USLE) ($kg/m^2/day$)	Sediment Delivery Ratio (unitless)	Adjusted Erosion Rate ($kg/m^2/day$)
E1	1.6E-03	0.122	1.9E-04
E2	2.3E-03	0.142	3.2E-04
E3	1.7E-03	0.133	2.2E-04
E4	2.3E-03	0.050	1.2E-04
E5	1.2E-03	0.124	1.5E-04
E6	1.2E-03	0.123	1.5E-04
E Farm Tilled	3.3E-03	0.384	1.3E-03
E Farm Untilled	1.3E-03	0.384	5.0E-04
W1	6.2E-04	0.131	8.2E-05
W2	2.6E-03	0.119	3.1E-04
W3	2.8E-03	0.122	3.4E-04
W4	1.8E-03	0.052	9.5E-05
W5	1.9E-03	0.123	2.3E-04
W6	2.1E-03	0.177	3.7E-04
W7	6.9E-03	0.309	2.1E-03
W8	3.3E-03	0.125	4.1E-04
W Farm Tilled	1.6E-02	0.384	6.1E-03
W Farm Untilled	1.2E-03	0.384	4.7E-04

I-1-3.10 References

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I-1-4Supplemental Information for Exhibit 1-12 – Aquatic Animals

I-1-4.1 Introduction

To estimate risks to human health and the environment, site-specific models of aquatic food webs were developed in TRIM.FaTE to represent the four water bodies in the vicinity of Ravena, NY: Nassau and Kinderhook Lakes, Alcove Reservoir, and an unnamed small pond near the facility. Characteristics of the fish compartments used to represent each water body were based on site-specific fish survey data and some additional information from the open literature. The development of each food web consisted of three stages:

1. Collection of local fish survey data for the water bodies from the New York State Department of Environmental Conservation (NY DEC), including data on the relative abundance and size/weight distribution of each species, to the extent available;

2. Formulation of simplified food webs for each water body, based on the fish surveys and other biological and physical data for each water body, with supplemental information on fish feeding habits, aquatic food webs, and biomass densities for different trophic levels from the open literature; and

3. Assignment of values for the remaining parameters (e.g., individual body weight, numeric density per unit area, lipid content) for each biotic compartment for each water body in TRIM.FaTE from the available data.

These stages are discussed in greater detail in the sections below. Professional judgment was used where available data were incomplete.

I-1-4.2 Collection of Information on Species Present in Water Bodies

To support the development of the aquatic food webs, fishery biologists at the NY DEC Region 4 Bureau of Fisheries were contacted. The NY DEC conducted surveys of fish in Nassau and Kinderhook Lakes at various times between 1988 and 2006. Due to contamination at Nassau and Kinderhook Lakes, there are fishing restrictions at these water bodies, and aquatic sampling is performed to assess current contaminant levels. The New York State Fish and Wildlife Department published the results of fish surveys conducted from 1963 to 1970 for Alcove Reservoir (NY FWD 1971). No surveys were available for the small pond. Professional judgment and published data from two small lakes in Canada were used to develop a model food web for the small pond.

The 1971 Alcove Reservoir fish survey report also presented data on average fish weights, which were used, where applicable, to estimate the average weight per individual fish for each species in all water bodies. These data are summarized in Exhibit 4-1. Surveys of the Alcove Reservoir have not been conducted since 1970 because the reservoir, which serves as a public

Exhibit 4-1. Fish Survey Data for Alcove Reservoir [a]

Species	Year	Count	Total Weight (lb)	Average Weight (lb)
American eel (*Anguilla rostrata*)	1970	1	4.4	4.40
Black crappie (*Pomoxis nigromaculatus*)	1966	6	4	0.67
	1965	70	50	0.71
Bluegill (*Lepomis macrochirus*)	1970	50	20	0.40
	1969	36	15	0.42
	1968	72	24	0.33
	1967	101	42	0.42
	1966	81	27	0.33
	1965	180	60	0.33
	1964	125	55	0.44
	1963	825	165	0.20
Bullhead (*Ameiurus* sp.)	1970	250	150	0.60
	1969	40	45	1.13
	1968	67	70	1.04
	1967	65	65	1.00
	1965	840	820	0.98
	1964	34	30	0.88
	1963	243	146	0.60
Chain pickerel (*Esox niger*)	1964	5	5	1.00
Largemouth bass (*Micropterus salmoides*)	1969	10	12	1.20
	1964	49	39	0.80
Northern pike (*Esox lucius*)	1969	6	8	1.33
	1968	6	11	1.83
	1967	7	6	0.86
	1966	2	3	1.50
	1964	3	2	0.67
Pumpkinseed (*Lepomis gibbosus*)	1966	50	17	0.34
	1965	170	65	0.38
	1964	75	30	0.40
	1963	421	84	0.20
Redbreast sunfish (*Lepomis* sp.)	1964	75	25	0.33
Smallmouth bass (*Micropterus dolomieu*)	1970	50	38	0.76
	1969	60	64	1.07
	1968	98	122	1.24
	1967	176	244	1.39
	1966	115	130	1.13
	1965	30	35	1.17
	1964	174	170	0.98
	1963	89	100	1.12
Walleye (*Stizostedion vitreum*)	1970	12	26	2.17

Exhibit 4-1. Fish Survey Data for Alcove Reservoir [a]

Species	Year	Count	Total Weight (lb)	Average Weight (lb)
	1969	10	14	1.40
	1968	21	33	1.57
	1967	167	204	1.22
	1966	33	49	1.48
	1965	31	42	1.35
	1963	7	10	1.43
White perch (*Morone americana*)	1964	9	8	0.89
White sucker (*Catostomus* sp.)	1970	3	6	2.00
Yellow perch (*Perca flavescens*)	1970	10	4	0.40
	1968	108	49	0.45
	1967	104	48	0.46
	1966	30	19	0.63
	1965	140	110	0.79
	1964	16	14	0.88
	1963	324	96	0.30

[a] NY FWD, 1971.

drinking water supply, has been closed to public fishing. Because data on fish length or weight were not available for the other water bodies, average fish weights for each species from the Alcove report were used as the average fish weights for the same species in the other water bodies. The relevant survey data provided by NY DEC for Nassau and Kinderhook Lakes are summarized in Exhibit 4-2 and Exhibit 4-3 (NY DEC 2008b).

While these surveys provide some indication of the relative abundance of different fish species in each water body over the periods of time represented, they do not indicate the absolute abundance of each species. No estimates were available for total fish standing stock (e.g., total biomass in the water body or biomass per unit area of the water body). In addition, potential biases introduced by selection of sampling times and locations and fish capture techniques have not been evaluated; information on the sampling methods (i.e., gill netting, electro-fishing) for Nassau and Kinderhook Lakes was not available at the time of this analysis. Personal communication with the Daniel Zielinski of NY DEC indicated that the fish surveys occurred at or after dusk, and that the timing of the sampling could have a large effect on the number and type of fish collected (NY DEC 2008a). Nonetheless, the best available estimates of the relative abundance of each species in the lakes and reservoir are the relative abundance of each species as reported in the fish surveys.

The food web for the small pond was developed from an analysis of data presented by Demers et al. (2001) for two small lakes in Canada. As a conservative position, the small pond is assumed to sustain a viable fish community from year to year. In each water body, young of the year were assumed to comprise 15 percent of the total fish biomass on an annual basis biomass.

Exhibit 4-2. Fish Survey Data for Nassau Lake [a]

Species	Year	Count
American eel (*Anguilla rostrata*)	1989	36
	2001	3
Black crappie (*Pomoxis nigromaculatus*)	1989	3
	2001	2
Bluegill (*Lepomis macrochirus*)	1989	100
	1997	70
	2001	7
Brown bullhead (*Ameiurus nebulosus*)	1989	2
	1997	15
	2001	18
Chain pickerel (*Esox niger*)	1988	4
	1989	1
	1997	2
Common carp (*Cyprinus carpio*)	1997	2
	2001	1
Golden shiner (*Notemigonus crysoleucas*)	1989	12
	1997	2
Largemouth bass (*Micropterus salmoides*)	1988	58
	1989	16
	1997	146
	2001	17
Pumpkinseed (*Lepomis gibbosus*)	1989	100
	1997	75
	2001	11
Redbreast sunfish (*Lepomis auritus*)	1997	1
Smallmouth bass (*Micropterus dolomieu*)	1997	5
	2001	3
White perch (*Morone americana*)	1988	20
	1989	10
	2001	5
White sucker (*Catostomus commersonii*)	1989	20
	2001	2
Yellow bullhead (*Ameiurus natalis*)	2001	3
Yellow perch (*Perca flavescens*)	1989	310
	1997	321
	2001	21

[a] NY DEC, 2008b.

Exhibit 4-3. Fish Survey Data for Kinderhook Lake [a]

Species	Year	Count
American eel (*Anguilla rostrata*)	1993	1
	1998	4
	2006	10
Black crappie (*Pomoxis nigromaculatus*)	1998	10
	2001	24
	2006	5
Bluegill (*Lepomis macrochirus*)	1998	27
	2001	30
	2006	12
Brown bullhead (*Ameiurus nebulosus*)	1998	2
	2001	10
	2006	1
Common carp (*Cyprinus carpio*)	1998	100
	2001	53
	2006	28
Fantail darter (*Etheostoma flabellare*)	2001	1
Golden shiner (*Notemigonus crysoleucas*)	1998	1
	2001	21
	2006	2
Largemouth bass (*Micropterus salmoides*)	1988	50
	1993	20
	1998	66
	2001	64
	2006	49
Pumpkinseed (*Lepomis gibbosus*)	1998	13
	2001	21
	2006	38
Redbreast sunfish (*Lepomis auritus*)	1998	10
	2001	1
Rock bass (*Ambloplites* sp.)	2006	1
Smallmouth bass (*Micropterus dolomieu*)	1998	7
	2001	51
	2006	90
Sunfish family (*Centrarchidae* sp.)	2006	1
Tiger musky (*Masquinongy* sp.)	2006	1
Walleye (*Stizostedion vitreum*)	2006	1

Exhibit 4-3. Fish Survey Data for Kinderhook Lake [a]

Species	Year	Count
White perch (*Morone americana*)	1988	18
	1993	20
	1998	19
	2001	618
	2006	504
White sucker (*Catostomus commersonii*)	1998	2
	2001	12
	2006	9
Yellow bullhead (*Ameiurus natalis*)	2001	1
	2006	1
Yellow perch (*Perca flavescens*)	1998	171
	2001	163
	2006	97

[a] NY DEC, 2008b.

I-1-4.3 Creation of Food Webs

Food webs for each of the four water bodies were constructed from the information sources identified above. Several steps were required to construct each food web and to assign parameter values for all aquatic biotic compartments for TRIM.FaTE:

1. Estimate total standing fish stock (i.e., total fish biomass per unit area) for each water body based on total biomass estimates reported for similar water bodies in the literature;

2. List for each water body all fish species found in the surveys of the water body;

3. Identify for each species an average body weight per individual based on the Alcove Reservoir data;

4. Estimate total biomass caught for each species in the surveys by multiplying the number of individuals of each species caught over the survey years for the water body by the average body weight per individual for each species;

5. Estimate the relative total biomass for each species (percentage of total biomass represented in surveys);

6. Estimate the absolute biomass of each species by multiplying its percent relative biomass by the estimated total standing fish stock (Step 1);

7. Estimate the numeric density of each fish species (number per unit area) based on biomass density and average individual weight for each species; and

8. Evaluate the feeding habits of each fish species, as determined from a variety of sources, relative to the food/prey categories supported by TRIM.FaTE:

    - plankton (called algae; however, it represents both phytoplankton and zooplankton);

- macrophytes;
- benthic invertebrates (e.g., aquatic insects, crustaceans, mollusks);
- small planktivorous fish (e.g., young of the year, minnows; feed on algae and zooplankton in the water column);
- larger omnivorous fish that feed on smaller fish in the water column and benthic invertebrates and/or macrophytes (e.g., sunfish, yellow perch)
- small-to-medium sized benthivores/omnivores that feed primarily on benthic invertebrates, detritus, and possibly macrophytes (e.g., small carp, white sucker).

Additionally, the lipid content of each species was estimated based on values reported in national surveys.

The total fish standing stock (biomass density of all fish species expressed as kilograms [kg] of fish [wet weight] per hectare [ha] of lake surface area) was determined from literature sources for similar water bodies in other locations. For Alcove Reservoir, standing stock estimates from comparably sized reservoirs from the literature were reviewed. Lynn and Tygart Reservoirs in West Virginia both have surface areas of approximately 1,700 acres (Yurk and Ney 1989), slightly larger than Alcove's estimated 1,360 acres. In the late 1980s, Yurk and Ney (1989) estimated the standing stock in these reservoirs, which were regularly fished, to be 77 and 104 kg/ha, respectively. We note, however, that these water bodies are, on average, 50 to 100 percent deeper than Alcove Reservoir. Because Alcove Reservoir is not fished, we considered it appropriate to assume a standing stock of 80 kg/ha for Alcove Reservoir despite its more shallow depth relative to Lynn and Tygart Reservoirs.

The smallest lake discussed by Yurk and Ney (1989) had the smallest standing stock (34 kg/ha), so we assumed that a value in this range would be appropriate for Kinderhook and Nassau Lakes, which are 134.1 and 64.9 hectares in surface area, respectively. Scaling standing stock proportionally to lake surface area suggests that the small unnamed pond near the facility might be too small to sustain game or panfish over the long term. We therefore used the lowest standing stock reported in Yurk and Ney (1989) as a floor for estimating fish standing stock for the pond. Exhibit shows the standing stock values selected for all four water bodies.

Exhibit 4-4. Estimated Total Fish Standing Stocks for Water Bodies Near Lafarge Facility

Water body	Stock (kg/ha)	Stock (g/m^2)
Alcove Reservoir	80	8
Nassau Lake	50	5
Kinderhook Lake	50	5
Small "farm" pond	40	4

The initial estimates of relative abundance for each fish species were based on the fish survey data. Only the species identified by fish surveys were assumed to be present in the four modeled water bodies. The body weight of each individual was assumed to be equal to the average fish weight estimated from the Alcove surveys. When species were present in the other lakes, but not in the Alcove Reservoir, professional judgment and data from other locations (e.g., Minnesota fish surveys) were used to estimate an average individual body weight for the species.

At the small pond, only three species/groups were assumed to be present: largemouth bass, sunfish (e.g., bluegill or pumpkinseed), and shiners. The mass of the individuals was estimated based on professional judgment and the Demers et al. (2001) study of two small lakes.

Total relative biomass for each species within a water body was estimated differently for Alcove Reservoir, Nassau and Kinderhook Lakes, and the small pond. At Alcove, each species' biomass representation was determined by taking the observed biomass of the species caught across all survey years and dividing that by the total fish biomass reported in the Alcove report across all survey years. At both Nassau and Kinderhook Lakes, the survey data seemed biased towards several species, specifically yellow and white perch, perhaps due to sampling techniques. The biomass representation was therefore adjusted to reflect a more balanced abundance across different species for these two water bodies. At the small pond, the distribution of biomass among the several species was estimated based on the Demers et al. study (2001).

Fish lipid content was estimated from data collected for the 1978 – 1981 National Contaminant Biomonitoring Program (compiled from Lowe et al. 1985). Because Lowe et al. (1985) did not report the lipid content of American eels, we used measurements of eel lipid content from a U.S. Army study of Lake Cochituate in Natick, MA (ICF Consulting 2001). The fish lipid estimates are presented in Exhibit 4-5.

Exhibit 4-5. Lipid Content for Fish Species Included in Model Food Webs

Fish species	Lipid content	Source
American eel	16.9%	ICF, 2001
Black crappie	5.7%	Lowe et al., 1985
Bluegill	3.1%	Lowe et al., 1985
Bullhead	2.8%	Lowe et al., 1985
Chain pickerel	1.8%	Mierzykowski and Carr, 2004
Common carp	3.6%	Lowe et al., 1985
Fantail darter	3.5%	Professional judgment
Golden shiner	3.5%	Lowe et al., 1985
Largemouth bass	3.3%	Lowe et al., 1985
Northern pike	2.9%	Lowe et al., 1985
Pumpkinseed	1.5%	Lowe et al., 1985
Redbreast sunfish	5.9%	Lowe et al., 1985
Rock bass	6.2%	Lowe et al., 1985
Smallmouth bass	4.4%	Lowe et al., 1985
Sunfish	5.9%	Lowe et al., 1985
Tiger musky	4.0%	Professional judgment
Walleye	7.9%	Lowe et al., 1985
Young of the year	3.5%	Professional judgment
White perch	17.1%	EPA, 1990b
White sucker	5.1%	Lowe et al., 1985
Yellow perch	4.3%	Lowe et al., 1985

Exhibit 4-6 presents the diets that we assumed for each fish species when determining which TRIM.FaTE fish compartment in which to include the biomass for that species. These assumptions are based on abundance of prey species in each water body and on abundance of species that compete for similar food sources.

Finally, each species was assigned to one of the TRIM.FaTE fish compartment categories as discussed above.

Exhibit 4-6. Aquatic Species Diets by TRIM.FaTE Model Compartments

Fish Species	Algae/ zooplankton	Macrophytes	Benthic invertebrates	Benthic omnivores	Water column herbivore	Water column omnivore
American eel			50%	50%		
Black crappie	50%		50%			
Bluegill (Kinderhook)[a]			100%			
Bluegill (Nassau and Alcove)			50%		50%	
Bullhead			100%			
Chain pickerel				25%	25%	50%
Common carp		100%				
Fantail darter	100%					
Golden shiner	100%					
Largemouth bass				25%	25%	50%
Largemouth bass (pond)[b]			50%		50%	
Northern Pike				25%	25%	50%
Pumpkinseed	25%		75%			
Redbreast sunfish			100%			
Rock bass			50%		50%	
Smallmouth bass			50%		50%	
Tiger musky				50%		50%
White perch					100%	
White sucker	25%		75%			
Yellow perch	25%		50%		25%	
Walleye			50%	25%		25%
Young of the year	100%					

[a] Bluegills in Kinderhook were assumed to feed primarily on invertebrates rather than on water column herbivores, as they are assumed to do in Nassau and Alcove, because Kinderhook contains a large population of white perch who also feed exclusively on the relatively sparse herbivorous population.

[b] Bass in the small pond are assumed to feed in part on benthic invertebrates because most of the omnivorous fish are assumed to be too large for the bass to swallow.

I-1-4.4 Parameterization of Fish Compartments to be Included in Application

Exhibit 4-7 through Exhibit 4-10 summarize the fish compartments constructed using the methods discussed in Section I-1-4.3. All fish species were assigned to one of the following five fish compartments established in TRIM.FaTE:

- Water column carnivore (WCC – large predominantly piscivorous species, e.g., walleye and largemouth bass);
- Water column omnivore (WCO – medium-sized fish that feed primarily in the water column, e.g., sunfish, yellow perch; see Section I-1-4.3, Step 8 bullets);
- Water column herbivore (WCH – more appropriately termed planktivore, e.g., black crappie);
- Benthic carnivore (BC – large carnivorous species, e.g., large bullhead, eel); and
- Benthic omnivore (BO – medium-sized fish that feed primarily on benthic invertebrates; see Section I-1-4.3).

The compartment to which each species was assigned was determined by its general foraging habitat (i.e., benthic or water column) and its primary food sources (e.g., invertebrates, smaller fish, plant material). The total biomass for each of the five fish TRIM.FaTE compartments was set equal to the sum of the biomass of the species assigned to each compartment.

Exhibit 4-7. Small Pond Parameters: Fish Mass, Abundance, and Model Representation

Fish Species	Individual Mass (g) [a]	Count (ha^{-1}) [b]	Total Count [c]	Biomass [d] (g ww/m^2)	Percentage Biomass [e]	Model Compartment [f]
Largemouth bass	1000	2	4	0.2	5.0%	WCC
Sunfish	250	120	240	3	75.0%	BO
Golden shiner	25	80	160	0.2	5.0%	WCH
Young of the year	50	120	240	0.6	15.0%	WCH
Total		**322**	**644**	**4**	**100%**	

[a] Average individual fish body weights based on data from Alcove Reservoir .
[b] Abundance or numerical fish density per hectare based on total fish abundance, survey data from Alcove Reservoir, and individual fish body weights.
[c] Total abundance or total numerical fish count based on total fish abundance, survey data from Alcove Reservoir, and individual fish body weights.
[d] Biomass density per square meter; calculated by multiplying average individual body weight by numeric density per square meter.
[e] Percentage biomass; calculated by dividing biomass per unit area by total biomass per unit area.
[f] TRIM.FaTE model compartment to which this species is assigned.

Exhibit 4-8. Alcove Reservoir Parameters: Fish Mass, Abundance, and Model Representation

Fish Species	Individual Mass [a] (g)	Count [b] (ha^{-1})	Total Count [c]	Biomass [d] (g ww/m^2)	Percentage Biomass [e]	Model Compartment [f]
American eel	2100	0.08	42	0.016	0.2%	BC
Black crappie	350	2.74	1512	0.096	1.2%	WCH
Bluegill	110	58.18	32064	0.64	8.0%	WCO
Bullhead	220	109.1	60120	2.4	30.0%	BO
Chain pickerel	280	0.29	157	0.008	0.1%	WCC
Largemouth bass	410	2.34	1290	0.096	1.2%	WCC
Northern pike	540	1.19	653	0.064	0.8%	WCC
Pumpkinseed	120	33.33	18370	0.4	5.0%	WCO
Redbreast sunfish	120	4.00	2204	0.048	0.6%	WCO
Smallmouth bass	260	61.54	33914	1.6	20.0%	WCO
Walleye	490	15.84	8728	0.776	9.7%	WCC
White perch	900	0.18	98	0.016	0.2%	WCO
Yellow perch	250	25.60	14108	0.64	8.0%	WCO
Young of the year	50	0.24	132	0.0012	15.0%	WCH
Total		**315**	**173597**	**8**	**100%**	

[a] Average individual fish body weights based on data from Alcove Reservoir.
[b] Abundance or numerical fish density per hectare based on total fish abundance, survey data from Alcove Reservoir, and individual fish body weights.
[c] Total abundance or total numerical fish count based on total fish abundance, survey data from Alcove Reservoir, and individual fish body weights.
[d] Biomass density per square meter; calculated by multiplying average individual body weight by numeric density per square meter.
[e] Percentage biomass; calculated by dividing biomass per unit area by total biomass per unit area.
[f] TRIM.FaTE model compartment to which this species was assigned.

Exhibit 4-9. Nassau Lake Parameters: Fish Mass, Abundance, and Model Representation

Fish Species	Individual Mass [a] (g)	Count [b] (ha^{-1})	Total Count [c]	Biomass [d] (g ww/m^2)	Percentage Biomass [e]	Model Compartment [f]
American eel	2100	0.12	8	0.025	0.5%	BC
Black crappie	350	2.57	167	0.09	1.8%	WCH
Bluegill	110	34.09	2213	0.375	7.5%	WCO
Bullhead	220	56.82	3688	1.25	25.0%	BO
Chain pickerel	280	0.18	12	0.005	0.1%	WCC
Common carp	300	0.83	54	0.025	0.5%	WCH
Golden shiner	10	50.00	3245	0.05	1.0%	WCH
Largemouth bass	410	1.83	119	0.075	1.5%	WCC
Pumpkinseed	120	26.67	1731	0.32	6.4%	WCO
Redbreast sunfish	120	27.08	1758	0.325	6.5%	WCO
Smallmouth bass	260	40.96	2658	1.065	21.3%	WCO
White perch	900	0.11	7	0.01	0.2%	WCO
White sucker	400	6.25	406	0.25	5.0%	WCO
Yellow perch	250	15.40	999	0.385	7.7%	CO
Young of the year	50	0.15	10	0.00075	15.0%	WCH
Total		**263**	**17069**	**5**	**100%**	

[a] Average individual fish body weights based on data from Alcove Reservoir
[b] Abundance or numerical fish density per hectare based on total fish abundance, survey data from Nassau Lake, and individual fish body weights.
[c] Total abundance or total numerical fish count based on total fish abundance, survey data from Nassau Lake, and individual fish body weights.
[d] Biomass density per square meter; calculated by multiplying average individual body weight by numeric density per square meter.
[e] Percentage biomass; calculated by dividing biomass per unit area by total biomass per unit area.
[f] TRIM.FaTE model compartment to which this species was assigned.

Exhibit 4-10. Kinderhook Lake Parameters: Fish Mass, Abundance, and Model Representation

Fish Species	Individual Mass [a] (g)	Count [b] (ha^{-1})	Total Count [c]	Biomass [d] (g ww/m^2)	Percentage Biomass [e]	Model Compartment [f]
American eel	2000	0.08	10	0.015	0.3%	BC
Black crappie	350	2.86	383	0.1	2.0%	WCH
Bluegill	110	36.36	4876	0.4	8.0%	WCO
Bullhead	220	28.41	3810	0.625	12.5%	BO
Common carp	300	4.75	637	0.1425	2.85%	WCH
Fantail darter	5	50.00	6705	0.025	0.5%	WCH
Golden shiner	10	37.50	5029	0.0375	0.75%	WCH
Largemouth bass	410	2.74	368	0.1125	2.25%	WCC
Pumpkinseed	120	18.75	2514	0.225	4.5%	WCO
Redbreast sunfish	120	20.00	2682	0.24	4.8%	WCO
Smallmouth bass	260	36.54	4900	0.95	19.0%	WCO
White perch	900	4.44	596	0.4	8.0%	WCO
White sucker	400	5.63	754	0.225	4.5%	WCO
Yellow perch	250	20.00	2682	0.5	10.0%	WCO
Rock bass	225	0.22	30	0.005	0.1%	WCO
Walleye	490	4.85	650	0.2375	4.75%	WCC
Tiger musky	500	0.20	27	0.01	0.2%	WCC
Young of the year	50	4.28	573	0.021375	15.0%	WCH
Total		**278**	**37280**	**5**	**100%**	

[a] Average individual fish body weights based on data from Alcove Reservoir.
[b] Abundance or numerical fish density per hectare based on total fish abundance, survey data from Kinderhook Lake, and individual fish body weights.
[c] Total abundance or total numerical fish count based on total fish abundance, survey data from Kinderhook Lake, and individual fish body weights.
[d] Biomass density per square meter; calculated by multiplying average individual body weight by numeric density per square meter.
[e] Percentage biomass; calculated by dividing biomass per unit area by total biomass per unit area.
[f] TRIM.FaTE model compartment to which this species was assigned.

The diet composition for each of the five fish compartments was calculated as being proportional to the biomass representation of each species assigned to that compartment. For example, if largemouth bass comprised 75 percent and smallmouth bass comprised 25 percent of the biomass of the WCC compartment, then the diet composition of the largemouth bass multiplied by 0.75 would be added to the diet composition of the smallmouth bass multiplied by 0.25 to estimate the diet composition for the WCC compartment.

Similarly, the lipid content for each of the five fish compartments in TRIM.FaTE was estimated from the biomass-weighted lipid content of the individual species assigned to the compartment. Thus, using the same example, the largemouth bass lipid content, multiplied by 0.75, would be added to the smallmouth bass lipid content, multiplied by 0.25, to estimate the lipid content of the WCC compartment.

Exhibit 4-11 through Exhibit 4-14 present the numeric and biomass densities for each model compartment as well as the lipid content used to represent the compartment. Exhibit 4-15 through Exhibit 4-18 present the diet composition used for each fish compartment derived as described above.

Exhibit 4-11. Small Pond Model Parameters: Fish Mass, Abundance (Number per Hectare), and Lipid Content

Model Compartment [a]	Individual Mass [b] (g)	Count [c] (ha^{-1})	Total Count [d]	Biomass [e] (g ww/m^2)	Percentage Biomass [f]	Lipid Content
Water column carnivore	1000	2	4	0.2	5.0%	3.3%
Water column herbivore	44	182.9	366	0.8	20.0%	3.5%
Benthic omnivore	250	120	240	3	75.0%	5.9%
Total		**305**	**610**	**4**	**100%**	

[a] TRIM.FaTE model compartment.
[b] Average individual fish body weights for each compartment based on biomass-weighted species-specific individual fish body weights.
[c] Abundance or numerical fish density per hectare
[d] Total abundance or total numerical fish count.
[e] Biomass density per square meter; calculated by multiplying average individual body weight by numeric density per square meter.
[f] Percentage biomass; calculated by dividing biomass per unit area by total biomass per unit area.

Exhibit 4-12. Alcove Reservoir Model Parameters: Fish Mass, Abundance, and Lipid Content

Model Compartment [a]	Individual Mass [b] (g)	Count [c] (ha^{-1})	Total Count [d]	Biomass [e] (g ww/m^2)	Percentage Biomass [f]	Lipid Content
Water column carnivore	483	19.53	10760	0.944	11.8%	7.0%
Water column omnivore	214	156.49	86243	3.344	41.8%	3.9%
Water column herbivore	72	179.45	98893	1.296	16.2%	3.7%
Benthic carnivore	2100	0.08	42	0.016	0.2%	16.9%
Benthic omnivore	220	109.09	60120	2.4	30.0%	2.8%
Total		**465**	**256058**	**8**	**100%**	

[a] TRIM.FaTE model compartment.
[b] Average individual fish body weights for each compartment based on biomass-weighted species-specific individual fish body weights.
[c] Abundance or numerical fish density per hectare
[d] Total abundance or total numerical fish count.
[e] Biomass density per square meter; calculated by multiplying average individual body weight by numeric density per square meter.
[f] Percentage biomass; calculated by dividing biomass per unit area by total biomass per unit area.

Exhibit 4-13. Nassau Lake Model Parameters: Fish Mass, Abundance, and Lipid Content

Model Compartment [a]	Individual Mass [b] (g)	Count [c] (ha^{-1})	Total Count [d]	Biomass [e] (g ww/m^2)	Percentage Biomass [f]	Lipid Content
Water column carnivore	402	1.99	129	0.08	1.6%	3.2%
Water column omnivore	220	124.05	8051	2.73	54.6%	4.2%
Water column herbivore	84	108.73	7057	0.915	18.3%	3.7%
Benthic carnivore	2100	0.12	8	0.025	0.5%	16.9%
Benthic omnivore	220	56.82	3688	1.25	25.0%	2.8%
Total		**291.7**	**18932**	**5**	**100%**	

[a] TRIM.FaTE model compartment.
[b] Average individual fish body weights for each compartment based on biomass-weighted species-specific individual fish body weights.
[c] Abundance or numerical fish density per hectare
[d] Total abundance or total numerical fish count.
[e] Biomass density per square meter; calculated by multiplying average individual body weight by numeric density per square meter.
[f] Percentage biomass; calculated by dividing biomass per unit area by total biomass per unit area.

Exhibit 4-14. Kinderhook Lake Model Parameters: Fish Mass, Abundance, and Lipid Content

Model Compartment [a]	Individual Mass [b] (g)	Count [c] (ha^{-1})	Total Count [d]	Biomass [e] (g ww/m^2)	Percentage Biomass [f]	Lipid Content
Water column carnivore	465	7.74	1038	0.36	7.2%	6.4%
Water column omnivore	313	93.97	12602	2.945	58.9%	5.9%
Water column herbivore	110	96.16	12895	1.055	21.1%	3.7%
Benthic carnivore	2000	0.08	10	0.015	0.3%	16.9%
Benthic omnivore	220	28.41	3810	0.625	12.5%	2.8%
Total		**226.4**	**30354**	**5**	**100%**	

[a] TRIM.FaTE model compartment.
[b] Average individual fish body weights for each compartment based on biomass-weighted species-specific individual fish body weights.
[c] Abundance or numerical fish density per hectare
[d] Total abundance or total numerical fish count.
[e] Biomass density per square meter; calculated by multiplying average individual body weight by numeric density per square meter.
[f] Percentage biomass; calculated by dividing biomass per unit area by total biomass per unit area.

Exhibit 4-15. Small Pond Aquatic Food Web

TRIM.FaTE Model Compartment	Algae/ zooplankton	Macrophytes	Benthic invertebrates	Benthic omnivores	Water column herbivore	Water column omnivore
Water column carnivore			50.0%		50.0%	
Water column herbivore	100.0%					
Benthic omnivore			100.0%			

Exhibit 4-16. Alcove Reservoir Aquatic Food Web

TRIM.FaTE Model Compartment	Algae/ zooplankton	Macrophytes	Benthic invertebrates	Benthic omnivores	Water column herbivore	Water column omnivore
Water column carnivore			41.0%	25.0%	4.4%	29.4%
Water column omnivore	7.8%		53.5%		38.8%	
Water column herbivore	96.3%		3.7%			
Benthic carnivore			50.0%	50.0%		
Benthic omnivore			100.0%			

Exhibit 4-17. Nassau Lake Aquatic Food Web

TRIM.FaTE Model Compartment	Algae/ zooplankton	Macrophytes	Benthic invertebrates	Benthic omnivores	Water column herbivore	Water column omnivore
Water column carnivore				25.0%	25.0%	50.0%
Water column omnivore	8.7%		61.0%		30.3%	
Water column herbivore	92.3%	2.7%	4.9%			
Benthic carnivore			50.0%	50.0%		
Benthic omnivore			100.0%			

Exhibit 4-18. Kinderhook Lake Aquatic Food Web

TRIM.FaTE Model Compartment	Algae/ zooplankton	Macrophytes	Benthic invertebrates	Benthic omnivores	Water column herbivore	Water column omnivore
Water column carnivore			33.0%	25.7%	7.8%	33.5%
Water column omnivore	8.1%		57.9%		34.0%	
Water column herbivore	81.8%	13.5%	4.7%			
Benthic carnivore			50.0%	50.0%		
Benthic omnivore			100.0%			

I-1-4.5 Fish Harvesting from Ravena Pond

During the development of the conceptual exposure model for the risk assessment of the Ravena facility, we judged that the possibility of an angler exposure scenario existing for the Ravena pond was low. It is assumed that the angler fishes at the pond regularly for a lifetime and consumes his or her catch. Due to the small size of the pond, however, it is unlikely that this water body could sustain fishable populations at the assumed ingestion rates without regular, substantial restocking of fish.

In order to obtain a realistic estimate of concentrations in fish in the Ravena Pond, we modified the TRIM.FaTE scenario and incorporated a fish harvest rate from the pond of 17 g/day to represent consumption and restocking of the pond within the TRIM.FaTE model. This harvest rate corresponds to the 90th percentile fish ingestion rate for an adult angler used to calculate hazard quotients and lifetime cancer risks from consumption of contaminated fish. For the purposes of this assessment, we also assumed that the angler consumes two types of fish — largemouth bass (33% of total consumption) and benthic omnivores (67% of total consumption).

I-1-4.6 References

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ATTACHMENT I-2: Detailed Ravena Human Health Assessment Exposure, Risk, and Hazard Quotient Estimates

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LIST OF EXHIBITS

I-2.1 Detailed Ravena Exposure Concentrations

Exhibit 1-1 presents annually averaged media concentrations at the 50th year for all compartments in the Ravena site-specific TRIM.FaTE scenario for 2,3,7,8-TCDD and mercury, including modeled fish harvesting from the Ravena Pond. The 2,3,7,8 – TCDD emissions include both a 95th percentile upper confidence limit (UCL) emission and a mean emission. The reported total mercury emissions from the Ravena facility were separated into elemental and divalent species based on a speciation factor of 75% elemental and 25% divalent. Methyl mercury is created via transformation reactions in the environment. Total mercury concentrations (i.e., the sum of mass or concentrations of these three species) are presented as well.

Exhibit 1-2 presents annually averaged media concentrations at the 50th year for all compartments in the TRIM.FaTE screening scenario, using the Ravena emission rates.

Exhibit 1-1. Annually Averaged Concentrations at Year 50 for All Compartments in the Ravena Site-Specific TRIM.FaTE Scenario, Including Modeled Fish Harvesting from the Ravena Pond

Compartment	Volume Element	Units	Mean TCDD	UCL TCDD	Divalent Mercury	Elemental Mercury	Methyl Mercury	Total Mercury
Air	Air_source	$\mu g/m^3$	4.E-08	9.E-08	1.E-03	4.E-03	9.E-11	6.E-03
Air	Air_1	$\mu g/m^3$	1.E-10	3.E-10	2.E-06	1.E-05	1.E-11	2.E-05
Air	Air_2	$\mu g/m^3$	2.E-10	4.E-10	3.E-06	2.E-05	3.E-11	3.E-05
Air	Air_3	$\mu g/m^3$	5.E-10	1.E-09	1.E-05	6.E-05	4.E-11	7.E-05
Air	Air_4	$\mu g/m^3$	6.E-10	1.E-09	1.E-05	7.E-05	9.E-11	9.E-05
Air	Air_5	$\mu g/m^3$	3.E-10	7.E-10	5.E-06	4.E-05	6.E-11	4.E-05
Air	Air_6	$\mu g/m^3$	1.E-10	4.E-10	2.E-06	2.E-05	4.E-11	2.E-05
Air	Air_7	$\mu g/m^3$	4.E-10	1.E-09	1.E-05	5.E-05	5.E-11	6.E-05
Air	Air_8	$\mu g/m^3$	8.E-10	2.E-09	2.E-05	1.E-04	5.E-11	1.E-04
Air	Air_9	$\mu g/m^3$	1.E-09	3.E-09	3.E-05	1.E-04	1.E-10	2.E-04
Air	Air_10	$\mu g/m^3$	6.E-10	2.E-09	2.E-05	8.E-05	1.E-10	9.E-05
Air	Air_11	$\mu g/m^3$	2.E-09	5.E-09	7.E-05	2.E-04	5.E-11	3.E-04
Air	Air_12	$\mu g/m^3$	3.E-09	8.E-09	1.E-04	4.E-04	6.E-11	5.E-04
Air	Air_13	$\mu g/m^3$	4.E-09	9.E-09	1.E-04	4.E-04	8.E-11	6.E-04
Air	Air_14	$\mu g/m^3$	3.E-09	7.E-09	9.E-05	3.E-04	8.E-11	4.E-04
Air	Air_15	$\mu g/m^3$	3.E-09	6.E-09	9.E-05	3.E-04	6.E-11	4.E-04
Air	Air_16	$\mu g/m^3$	4.E-09	1.E-08	1.E-04	5.E-04	7.E-11	6.E-04
Air	Air_17	$\mu g/m^3$	4.E-09	1.E-08	1.E-04	5.E-04	9.E-11	6.E-04
Air	Air_18	$\mu g/m^3$	3.E-09	7.E-09	1.E-04	4.E-04	8.E-11	5.E-04
Air	Air_19	$\mu g/m^3$	2.E-10	4.E-10	3.E-06	2.E-05	2.E-11	2.E-05
Air	Air_20	$\mu g/m^3$	3.E-10	7.E-10	5.E-06	3.E-05	6.E-11	4.E-05
Air	Air_21	$\mu g/m^3$	6.E-10	2.E-09	2.E-05	8.E-05	6.E-11	9.E-05
Air	Air_22	$\mu g/m^3$	1.E-09	3.E-09	3.E-05	1.E-04	1.E-10	2.E-04
Air	Air_23	$\mu g/m^3$	1.E-09	3.E-09	3.E-05	1.E-04	1.E-10	1.E-04
Air	Air_24	$\mu g/m^3$	8.E-10	2.E-09	2.E-05	9.E-05	1.E-10	1.E-04
Air	Air_25	$\mu g/m^3$	3.E-10	8.E-10	7.E-06	4.E-05	8.E-11	5.E-05
Air	Air_26	$\mu g/m^3$	2.E-10	4.E-10	3.E-06	2.E-05	5.E-11	2.E-05
Air	Air_27	$\mu g/m^3$	5.E-10	1.E-09	9.E-06	6.E-05	5.E-11	7.E-05
Air	Air_28	$\mu g/m^3$	4.E-10	1.E-09	8.E-06	5.E-05	1.E-10	6.E-05
Air	Air_29	$\mu g/m^3$	3.E-10	7.E-10	4.E-06	3.E-05	3.E-11	4.E-05
Air	Air_30	$\mu g/m^3$	2.E-10	5.E-10	3.E-06	3.E-05	9.E-11	3.E-05
Air	Air_Layer2	$\mu g/m^3$	1.E-12	3.E-12	5.E-08	2.E-07	0.E+00	2.E-07
Surface water	SW_AR	mg/L	1.E-14	3.E-14	1.E-07	8.E-08	2.E-09	2.E-07
Water Column Carnivore	SW_AR	mg/kg wet weight	8.E-09	2.E-08	7.E-05	2.E-12	6.E-04	6.E-04
Water Column Herbivore	SW_AR	mg/kg wet weight	9.E-11	2.E-10	1.E-04	4.E-13	6.E-05	2.E-04
Water Column Omnivore	SW_AR	mg/kg wet weight	1.E-09	3.E-09	9.E-05	2.E-12	2.E-04	3.E-04

Exhibit 1-1. Annually Averaged Concentrations at Year 50 for All Compartments in the Ravena Site-Specific TRIM.FaTE Scenario, Including Modeled Fish Harvesting from the Ravena Pond

Compartment	Volume Element	Units	Mean TCDD	UCL TCDD	Divalent Mercury	Elemental Mercury	Methyl Mercury	Total Mercury
Macrophyte	SW_AR	mg/kg wet weight	2.E-10	4.E-10	1.E-07	4.E-17	4.E-09	1.E-07
Mallard	SW_AR	μg/g wet weight	3.E-08	7.E-08	2.E-04	7.E-07	4.E-05	3.E-04
Sediment	Sed_AR	μg/g dry weight	2.E-10	4.E-10	6.E-03	9.E-05	1.E-05	6.E-03
Benthic Invertebrate	Sed_AR	mg/kg wet weight	2.E-11	4.E-11	3.E-04	5.E-06	4.E-05	4.E-04
Benthic Omnivore	Sed_AR	mg/kg wet weight	9.E-10	2.E-09	1.E-04	3.E-12	1.E-04	2.E-04
Benthic Carnivore	Sed_AR	mg/kg wet weight	5.E-09	1.E-08	8.E-05	2.E-12	5.E-04	6.E-04
Surface water	SW_KL	mg/L	3.E-14	7.E-14	2.E-07	5.E-08	3.E-09	3.E-07
Water Column Carnivore	SW_KL	mg/kg wet weight	3.E-08	7.E-08	9.E-05	2.E-12	9.E-04	1.E-03
Water Column Herbivore	SW_KL	mg/kg wet weight	3.E-10	7.E-10	2.E-04	2.E-13	7.E-05	3.E-04
Water Column Omnivore	SW_KL	mg/kg wet weight	6.E-09	1.E-08	1.E-04	2.E-12	3.E-04	4.E-04
Macrophyte	SW_KL	mg/kg wet weight	4.E-10	1.E-09	1.E-07	2.E-17	5.E-09	1.E-07
Mallard	SW_KL	μg/g wet weight	7.E-08	2.E-07	3.E-04	6.E-07	7.E-05	4.E-04
Sediment	Sed_KL	μg/g dry weight	4.E-10	1.E-09	9.E-03	7.E-05	2.E-05	9.E-03
Benthic Invertebrate	Sed_KL	mg/kg wet weight	4.E-11	1.E-10	5.E-04	4.E-06	6.E-05	5.E-04
Benthic Omnivore	Sed_KL	mg/kg wet weight	2.E-09	4.E-09	2.E-04	5.E-12	2.E-04	3.E-04
Benthic Carnivore	Sed_KL	mg/kg wet weight	9.E-09	2.E-08	1.E-04	2.E-12	7.E-04	8.E-04
Surface water	SW_NL	mg/L	4.E-14	9.E-14	2.E-07	2.E-08	2.E-09	2.E-07
Water Column Carnivore	SW_NL	mg/kg wet weight	1.E-07	3.E-07	5.E-05	4.E-19	1.E-03	1.E-03
Water Column Herbivore	SW_NL	mg/kg wet weight	3.E-10	8.E-10	2.E-04	1.E-13	8.E-05	2.E-04
Water Column Omnivore	SW_NL	mg/kg wet weight	1.E-08	3.E-08	1.E-04	2.E-12	3.E-04	4.E-04
Macrophyte	SW_NL	mg/kg wet weight	6.E-10	1.E-09	9.E-08	2.E-17	5.E-09	1.E-07
Mallard	SW_NL	μg/g wet weight	9.E-08	2.E-07	2.E-04	3.E-07	5.E-05	3.E-04
Sediment	Sed_NL	μg/g dry weight	5.E-10	1.E-09	7.E-03	4.E-05	2.E-05	7.E-03
Benthic Invertebrate	Sed_NL	mg/kg wet weight	6.E-11	1.E-10	4.E-04	2.E-06	5.E-05	4.E-04
Benthic Omnivore	Sed_NL	mg/kg wet weight	8.E-09	2.E-08	1.E-04	3.E-12	2.E-04	3.E-04
Benthic Carnivore	Sed_NL	mg/kg wet weight	4.E-08	1.E-07	9.E-05	1.E-12	7.E-04	8.E-04
Surface water	SW_Pond	mg/L	5.E-12	1.E-11	1.E-04	5.E-06	5.E-07	1.E-04
Mink	SW_Pond	μg/g wet weight	2.E-09	5.E-09	3.E-05	4.E-07	3.E-05	7.E-05
Mallard	SW_Pond	μg/g wet weight	1.E-05	3.E-05	3.E-02	9.E-06	6.E-03	4.E-02
Water Column Carnivore	SW_Pond	mg/kg wet weight	1.E-06	3.E-06	3.E-02	4.E-11	1.E-01	1.E-01
Water Column Herbivore	SW_Pond	mg/kg wet weight	4.E-07	9.E-07	1.E-01	0.E+00	6.E-02	2.E-01
Macrophyte	SW_Pond	mg/kg wet weight	8.E-08	2.E-07	5.E-05	7.E-15	1.E-06	5.E-05
Sediment	Sed_Pond	μg/g dry weight	3.E-08	6.E-08	9.E-01	1.E-03	2.E-03	9.E-01
Benthic Invertebrate	Sed_Pond	mg/kg wet weight	3.E-09	6.E-09	5.E-02	6.E-05	6.E-03	5.E-02
Benthic Omnivore	Sed_Pond	mg/kg wet weight	2.E-06	4.E-06	2.E-02	8.E-11	3.E-02	4.E-02
Surface water	SW_River	mg/L	6.E-14	1.E-13	1.E-07	9.E-09	3.E-09	1.E-07
Sediment	Sed_River	μg/g dry weight	8.E-10	2.E-09	6.E-03	2.E-05	1.E-05	6.E-03

Exhibit 1-1. Annually Averaged Concentrations at Year 50 for All Compartments in the Ravena Site-Specific TRIM.FaTE Scenario, Including Modeled Fish Harvesting from the Ravena Pond

Compartment	Volume Element	Units	Mean TCDD	UCL TCDD	Divalent Mercury	Elemental Mercury	Methyl Mercury	Total Mercury
Soil - Surface	SurfSoil_source	μg/g dry weight	6.E-07	1.E-06	9.E+00	1.E-03	1.E-01	9.E+00
Soil - Surface	SurfSoil_W1	μg/g dry weight	2.E-08	5.E-08	5.E-02	6.E-06	9.E-04	5.E-02
Leaf - Grasses/Herbs [a]	SurfSoil_W1	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Grasses/Herbs	SurfSoil_W1	mg/kg wet weight	1.E-06	3.E-06	5.E-02	9.E-08	3.E-09	5.E-02
Root - Grasses/Herbs	SurfSoil_W1	mg/kg wet weight	1.E-13	3.E-13	1.E-06	0.E+00	1.E-07	1.E-06
Stem - Grasses/Herbs	SurfSoil_W1	mg/kg wet weight	9.E-10	2.E-09	8.E-05	4.E-11	5.E-12	8.E-05
Soil - Surface	SurfSoil_W2	μg/g dry weight	2.E-08	4.E-08	4.E-02	5.E-06	7.E-04	4.E-02
Leaf - Deciduous Forest [a]	SurfSoil_W2	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Deciduous Forest	SurfSoil_W2	mg/kg wet weight	1.E-06	3.E-06	2.E-02	9.E-08	3.E-10	2.E-02
Soil - Surface	SurfSoil_W3	μg/g dry weight	3.E-09	8.E-09	8.E-03	1.E-06	1.E-04	8.E-03
Leaf - Deciduous Forest [a]	SurfSoil_W3	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Deciduous Forest	SurfSoil_W3	mg/kg wet weight	1.E-07	3.E-07	5.E-03	8.E-09	9.E-11	5.E-03
Soil - Surface	SurfSoil_W4	μg/g dry weight	6.E-09	1.E-08	1.E-02	2.E-06	2.E-04	1.E-02
Leaf - Deciduous Forest [a]	SurfSoil_W4	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Deciduous Forest	SurfSoil_W4	mg/kg wet weight	2.E-07	6.E-07	1.E-02	2.E-08	2.E-10	1.E-02
Soil - Surface	SurfSoil_W5	μg/g dry weight	9.E-09	2.E-08	2.E-02	3.E-06	4.E-04	2.E-02
Leaf - Deciduous Forest [a]	SurfSoil_W5	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Deciduous Forest	SurfSoil_W5	mg/kg wet weight	4.E-07	1.E-06	2.E-02	3.E-08	3.E-10	2.E-02
Soil - Surface	SurfSoil_W6	μg/g dry weight	2.E-08	5.E-08	1.E-01	1.E-05	2.E-03	1.E-01
Leaf - Deciduous Forest [a]	SurfSoil_W6	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Deciduous Forest	SurfSoil_W6	mg/kg wet weight	1.E-06	2.E-06	8.E-02	7.E-08	9.E-10	8.E-02
Soil - Surface	SurfSoil_W7	μg/g dry weight	2.E-08	4.E-08	5.E-02	6.E-06	8.E-04	5.E-02
Leaf - Deciduous Forest [a]	SurfSoil_W7	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Deciduous Forest	SurfSoil_W7	mg/kg wet weight	1.E-06	3.E-06	2.E-01	7.E-08	8.E-10	2.E-01
Soil - Surface	SurfSoil_W8	μg/g dry weight	1.E-08	3.E-08	4.E-02	5.E-06	7.E-04	4.E-02
Leaf - Deciduous Forest [a]	SurfSoil_W8	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Deciduous Forest	SurfSoil_W8	mg/kg wet weight	5.E-07	1.E-06	2.E-02	4.E-08	4.E-10	2.E-02
Soil - Surface	SurfSoil_E1	μg/g dry weight	7.E-09	2.E-08	2.E-02	2.E-06	3.E-04	2.E-02
Leaf - Deciduous Forest [a]	SurfSoil_E1	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Deciduous Forest	SurfSoil_E1	mg/kg wet weight	5.E-07	1.E-06	2.E-02	3.E-08	5.E-10	2.E-02
Soil - Surface	SurfSoil_E2	μg/g dry weight	3.E-09	8.E-09	8.E-03	1.E-06	1.E-04	8.E-03
Leaf - Deciduous Forest [a]	SurfSoil_E2	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Deciduous Forest	SurfSoil_E2	mg/kg wet weight	3.E-07	7.E-07	2.E-02	2.E-08	3.E-10	2.E-02
Soil - Surface	SurfSoil_E3	μg/g dry weight	4.E-09	9.E-09	1.E-02	1.E-06	2.E-04	1.E-02
Leaf - Deciduous Forest [a]	SurfSoil_E3	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Deciduous Forest	SurfSoil_E3	mg/kg wet weight	2.E-07	5.E-07	1.E-02	2.E-08	4.E-10	1.E-02
Soil - Surface	SurfSoil_E4	μg/g dry weight	4.E-09	1.E-08	1.E-02	1.E-06	2.E-04	1.E-02

Exhibit 1-1. Annually Averaged Concentrations at Year 50 for All Compartments in the Ravena Site-Specific TRIM.FaTE Scenario, Including Modeled Fish Harvesting from the Ravena Pond

Compartment	Volume Element	Units	Mean TCDD	UCL TCDD	Divalent Mercury	Elemental Mercury	Methyl Mercury	Total Mercury
Leaf - Deciduous Forest [a]	SurfSoil_E4	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Deciduous Forest	SurfSoil_E4	mg/kg wet weight	2.E-07	5.E-07	9.E-03	2.E-08	4.E-10	9.E-03
Soil - Surface	SurfSoil_E5	μg/g dry weight	1.E-08	3.E-08	3.E-02	4.E-06	5.E-04	3.E-02
Leaf - Grasses/Herbs [a]	SurfSoil_E5	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Grasses/Herbs	SurfSoil_E5	mg/kg wet weight	5.E-07	1.E-06	2.E-02	4.E-08	2.E-09	2.E-02
Root - Grasses/Herbs	SurfSoil_E5	mg/kg wet weight	6.E-14	2.E-13	8.E-07	0.E+00	9.E-08	9.E-07
Stem - Grasses/Herbs	SurfSoil_E5	mg/kg wet weight	4.E-10	9.E-10	4.E-05	2.E-11	4.E-12	4.E-05
Soil - Surface	SurfSoil_E6	μg/g dry weight	9.E-09	2.E-08	3.E-02	3.E-06	5.E-04	3.E-02
Leaf - Grasses/Herbs [a]	SurfSoil_E6	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Grasses/Herbs	SurfSoil_E6	mg/kg wet weight	5.E-07	1.E-06	3.E-02	4.E-08	2.E-09	3.E-02
Root - Grasses/Herbs	SurfSoil_E6	mg/kg wet weight	6.E-14	1.E-13	7.E-07	0.E+00	8.E-08	8.E-07
Stem - Grasses/Herbs	SurfSoil_E6	mg/kg wet weight	4.E-10	9.E-10	4.E-05	2.E-11	3.E-12	4.E-05
Soil - Surface	SurfSoil_WFT	μg/g dry weight	8.E-10	2.E-09	4.E-03	1.E-04	7.E-05	5.E-03
Leaf - Agriculture - General [a]	SurfSoil_WFT	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Agriculture - General	SurfSoil_WFT	mg/kg wet weight	1.E-06	3.E-06	2.E-01	5.E-08	2.E-10	2.E-01
Root - Agriculture - General	SurfSoil_WFT	mg/kg wet weight	2.E-15	4.E-15	4.E-08	0.E+00	4.E-09	4.E-08
Stem - Agriculture - General	SurfSoil_WFT	mg/kg wet weight	7.E-10	2.E-09	3.E-04	4.E-11	3.E-13	3.E-04
Soil - Surface	SurfSoil_WFU	μg/g dry weight	1.E-08	2.E-08	5.E-02	6.E-06	9.E-04	5.E-02
Leaf - Agriculture - General [a]	SurfSoil_WFU	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Agriculture - General	SurfSoil_WFU	mg/kg wet weight	5.E-07	1.E-06	6.E-02	2.E-08	1.E-09	6.E-02
Root - Agriculture - General	SurfSoil_WFU	mg/kg wet weight	6.E-14	2.E-13	1.E-06	0.E+00	2.E-07	2.E-06
Stem - Agriculture - General	SurfSoil_WFU	mg/kg wet weight	3.E-10	9.E-10	9.E-05	4.E-11	3.E-12	9.E-05
Soil - Surface	SurfSoil_EFT	μg/g dry weight	8.E-10	2.E-09	5.E-03	1.E-04	8.E-05	5.E-03
Leaf - Agriculture - General [a]	SurfSoil_EFT	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Agriculture - General	SurfSoil_EFT	mg/kg wet weight	1.E-06	3.E-06	2.E-01	4.E-08	5.E-10	2.E-01
Root - Agriculture - General	SurfSoil_EFT	mg/kg wet weight	2.E-15	4.E-15	4.E-08	0.E+00	4.E-09	4.E-08
Stem - Agriculture - General	SurfSoil_EFT	mg/kg wet weight	5.E-10	1.E-09	3.E-04	2.E-11	6.E-13	3.E-04
Soil - Surface	SurfSoil_EFU	μg/g dry weight	1.E-08	3.E-08	7.E-02	8.E-06	1.E-03	7.E-02
Leaf - Agriculture - General [a]	SurfSoil_EFU	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Agriculture - General	SurfSoil_EFU	mg/kg wet weight	1.E-06	3.E-06	2.E-01	4.E-08	2.E-09	2.E-01
Root - Agriculture - General	SurfSoil_EFU	mg/kg wet weight	9.E-14	2.E-13	2.E-06	0.E+00	2.E-07	2.E-06
Stem - Agriculture - General	SurfSoil_EFU	mg/kg wet weight	5.E-10	1.E-09	3.E-04	2.E-11	5.E-12	3.E-04
Soil - Root Zone	RootSoil_source	μg/g dry weight	6.E-11	1.E-10	7.E-04	7.E-04	1.E-05	1.E-03
Soil - Root Zone	RootSoil_W1	μg/g dry weight	2.E-12	5.E-12	4.E-06	5.E-06	7.E-08	9.E-06
Soil - Root Zone	RootSoil_W2	μg/g dry weight	2.E-12	4.E-12	4.E-06	4.E-06	6.E-08	8.E-06
Soil - Root Zone	RootSoil_W3	μg/g dry weight	3.E-13	8.E-13	7.E-07	8.E-07	1.E-08	1.E-06
Soil - Root Zone	RootSoil_W4	μg/g dry weight	6.E-13	1.E-12	1.E-06	1.E-06	2.E-08	2.E-06

Exhibit 1-1. Annually Averaged Concentrations at Year 50 for All Compartments in the Ravena Site-Specific TRIM.FaTE Scenario, Including Modeled Fish Harvesting from the Ravena Pond

Compartment	Volume Element	Units	Mean TCDD	UCL TCDD	Divalent Mercury	Elemental Mercury	Methyl Mercury	Total Mercury
Soil - Root Zone	RootSoil_W5	μg/g dry weight	9.E-13	2.E-12	2.E-06	2.E-06	3.E-08	4.E-06
Soil - Root Zone	RootSoil_W6	μg/g dry weight	2.E-12	6.E-12	1.E-05	1.E-05	2.E-07	2.E-05
Soil - Root Zone	RootSoil_W7	μg/g dry weight	2.E-12	4.E-12	5.E-06	6.E-06	9.E-08	1.E-05
Soil - Root Zone	RootSoil_W8	μg/g dry weight	1.E-12	3.E-12	3.E-06	4.E-06	6.E-08	7.E-06
Soil - Root Zone	RootSoil_E1	μg/g dry weight	8.E-13	2.E-12	2.E-06	2.E-06	3.E-08	3.E-06
Soil - Root Zone	RootSoil_E2	μg/g dry weight	4.E-13	9.E-13	7.E-07	8.E-07	1.E-08	1.E-06
Soil - Root Zone	RootSoil_E3	μg/g dry weight	4.E-13	1.E-12	8.E-07	1.E-06	1.E-08	2.E-06
Soil - Root Zone	RootSoil_E4	μg/g dry weight	4.E-13	1.E-12	9.E-07	1.E-06	2.E-08	2.E-06
Soil - Root Zone	RootSoil_E5	μg/g dry weight	1.E-12	3.E-12	3.E-06	3.E-06	4.E-08	5.E-06
Soil - Root Zone	RootSoil_E6	μg/g dry weight	1.E-12	2.E-12	2.E-06	3.E-06	4.E-08	5.E-06
Soil - Root Zone	RootSoil_WFT	μg/g dry weight	3.E-14	7.E-14	1.E-07	4.E-05	2.E-09	4.E-05
Soil - Root Zone	RootSoil_WFU	μg/g dry weight	1.E-12	3.E-12	4.E-06	5.E-06	7.E-08	9.E-06
Soil - Root Zone	RootSoil_EFT	μg/g dry weight	3.E-14	7.E-14	1.E-07	5.E-05	2.E-09	5.E-05
Soil - Root Zone	RootSoil_EFU	μg/g dry weight	2.E-12	4.E-12	6.E-06	7.E-06	1.E-07	1.E-05
Soil - Vadose Zone	VadoseSoil_source	g/g dry weight	1.E-24	3.E-24	3.E-17	5.E-13	4.E-19	5.E-13
Soil - Vadose Zone	VadoseSoil_W1	g/g dry weight	4.E-26	1.E-25	2.E-19	3.E-15	3.E-21	3.E-15
Soil - Vadose Zone	VadoseSoil_W2	g/g dry weight	3.E-26	8.E-26	1.E-19	3.E-15	2.E-21	3.E-15
Soil - Vadose Zone	VadoseSoil_W3	g/g dry weight	7.E-27	2.E-26	3.E-20	5.E-16	4.E-22	5.E-16
Soil - Vadose Zone	VadoseSoil_W4	g/g dry weight	1.E-26	3.E-26	4.E-20	9.E-16	7.E-22	9.E-16
Soil - Vadose Zone	VadoseSoil_W5	g/g dry weight	2.E-26	5.E-26	7.E-20	1.E-15	1.E-21	1.E-15
Soil - Vadose Zone	VadoseSoil_W6	g/g dry weight	5.E-26	1.E-25	4.E-19	7.E-15	6.E-21	7.E-15
Soil - Vadose Zone	VadoseSoil_W7	g/g dry weight	4.E-26	9.E-26	2.E-19	5.E-15	4.E-21	5.E-15
Soil - Vadose Zone	VadoseSoil_W8	g/g dry weight	2.E-26	6.E-26	1.E-19	3.E-15	2.E-21	3.E-15
Soil - Vadose Zone	VadoseSoil_E1	g/g dry weight	2.E-26	4.E-26	6.E-20	1.E-15	1.E-21	1.E-15
Soil - Vadose Zone	VadoseSoil_E2	g/g dry weight	8.E-27	2.E-26	3.E-20	5.E-16	4.E-22	5.E-16
Soil - Vadose Zone	VadoseSoil_E3	g/g dry weight	8.E-27	2.E-26	3.E-20	7.E-16	5.E-22	7.E-16
Soil - Vadose Zone	VadoseSoil_E4	g/g dry weight	9.E-27	2.E-26	3.E-20	7.E-16	6.E-22	7.E-16
Soil - Vadose Zone	VadoseSoil_E5	g/g dry weight	2.E-26	5.E-26	9.E-20	2.E-15	2.E-21	2.E-15
Soil - Vadose Zone	VadoseSoil_E6	g/g dry weight	2.E-26	5.E-26	8.E-20	2.E-15	1.E-21	2.E-15
Soil - Vadose Zone	VadoseSoil_WFT	g/g dry weight	4.E-27	1.E-26	3.E-20	2.E-13	6.E-22	2.E-13
Soil - Vadose Zone	VadoseSoil_WFU	g/g dry weight	2.E-26	6.E-26	2.E-19	3.E-15	3.E-21	3.E-15
Soil - Vadose Zone	VadoseSoil_EFT	g/g dry weight	4.E-27	1.E-26	3.E-20	2.E-13	6.E-22	2.E-13
Soil - Vadose Zone	VadoseSoil_EFU	g/g dry weight	3.E-26	8.E-26	2.E-19	5.E-15	4.E-21	5.E-15
Groundwater	GW_source	g/L	1.E-32	3.E-32	6.E-24	2.E-19	1.E-25	2.E-19
Groundwater	GW_W1	g/L	4.E-34	1.E-33	4.E-26	1.E-21	7.E-28	1.E-21
Groundwater	GW_W2	g/L	3.E-34	8.E-34	4.E-26	1.E-21	7.E-28	1.E-21
Groundwater	GW_W3	g/L	7.E-35	2.E-34	8.E-27	2.E-22	1.E-28	2.E-22

Exhibit 1-1. Annually Averaged Concentrations at Year 50 for All Compartments in the Ravena Site-Specific TRIM.FaTE Scenario, Including Modeled Fish Harvesting from the Ravena Pond

Compartment	Volume Element	Units	Mean TCDD	UCL TCDD	Divalent Mercury	Elemental Mercury	Methyl Mercury	Total Mercury
Groundwater	GW_W4	g/L	1.E-34	3.E-34	1.E-26	3.E-22	2.E-28	3.E-22
Groundwater	GW_W5	g/L	2.E-34	5.E-34	2.E-26	5.E-22	3.E-28	5.E-22
Groundwater	GW_W6	g/L	5.E-34	1.E-33	1.E-25	3.E-21	2.E-27	3.E-21
Groundwater	GW_W7	g/L	4.E-34	9.E-34	8.E-26	2.E-21	1.E-27	2.E-21
Groundwater	GW_W8	g/L	2.E-34	6.E-34	4.E-26	9.E-22	7.E-28	9.E-22
Groundwater	GW_E1	g/L	2.E-34	4.E-34	2.E-26	4.E-22	3.E-28	4.E-22
Groundwater	GW_E2	g/L	8.E-35	2.E-34	8.E-27	2.E-22	1.E-28	2.E-22
Groundwater	GW_E3	g/L	8.E-35	2.E-34	1.E-26	2.E-22	2.E-28	2.E-22
Groundwater	GW_E4	g/L	9.E-35	2.E-34	1.E-26	3.E-22	2.E-28	3.E-22
Groundwater	GW_E5	g/L	2.E-34	5.E-34	3.E-26	7.E-22	5.E-28	7.E-22
Groundwater	GW_E6	g/L	2.E-34	5.E-34	3.E-26	6.E-22	4.E-28	6.E-22
Groundwater	GW_WFT	g/L	4.E-35	1.E-34	2.E-24	5.E-20	3.E-26	5.E-20
Groundwater	GW_WFU	g/L	2.E-34	6.E-34	5.E-26	1.E-21	8.E-28	1.E-21
Groundwater	GW_EFT	g/L	4.E-35	1.E-34	2.E-24	5.E-20	3.E-26	5.E-20
Groundwater	GW_EFU	g/L	3.E-34	8.E-34	7.E-26	2.E-21	1.E-27	2.E-21

[a] Annually averaged leaf concentrations are unavailable because of the seasonally changing leaf compartments.

Exhibit 1-2. Annually Averaged Concentrations at Year 50 for All Compartments in the TRIM.FaTE Screening Scenario using Ravena Emission Rates [a]

Compartment	Volume Element	Units	Mean TCDD	UCL TCDD	Divalent Mercury	Elemental Mercury	Methyl Mercury	Total Mercury
Air	Air_source	μg/m^3	8.E-08	2.E-07	3.E-03	1.E-02	2.E-09	1.E-02
Air	Air_N1	μg/m^3	2.E-08	6.E-08	9.E-04	3.E-03	2.E-11	4.E-03
Air	Air_N2	μg/m^3	2.E-08	4.E-08	6.E-04	2.E-03	2.E-11	3.E-03
Air	Air_N3	μg/m^3	1.E-08	3.E-08	4.E-04	1.E-03	2.E-11	2.E-03
Air	Air_N4	μg/m^3	5.E-09	1.E-08	1.E-04	6.E-04	7.E-12	7.E-04
Air	Air_N5	μg/m^3	3.E-09	6.E-09	6.E-05	3.E-04	4.E-12	4.E-04
Air	Air_S1	μg/m^3	2.E-08	6.E-08	9.E-04	3.E-03	5.E-09	4.E-03
Air	Air_S2	μg/m^3	2.E-08	4.E-08	6.E-04	2.E-03	6.E-09	3.E-03
Air	Air_S3	μg/m^3	1.E-08	3.E-08	4.E-04	1.E-03	6.E-09	2.E-03
Air	Air_S4	μg/m^3	5.E-09	1.E-08	1.E-04	6.E-04	1.E-09	7.E-04
Air	Air_S5	μg/m^3	3.E-09	6.E-09	6.E-05	3.E-04	7.E-10	4.E-04
Surface water	SW_pond	mg/L	3.E-12	7.E-12	6.E-05	6.E-06	5.E-07	6.E-05
Water Column Carnivore	SW_pond	mg/kg wet weight	1.E-06	3.E-06	4.E-03	2.E-21	8.E-02	9.E-02
Water Column Herbivore	SW_pond	mg/kg wet weight	1.E-08	3.E-08	2.E-02	0.E+00	1.E-02	3.E-02
Water Column Omnivore	SW_pond	mg/kg wet weight	8.E-08	2.E-07	7.E-03	5.E-13	2.E-02	2.E-02
Macrophyte	SW_pond	mg/kg wet weight	2.E-08	4.E-08	1.E-05	8.E-16	3.E-07	1.E-05
Sediment	Sed_pond	μg/g dry weight	3.E-08	8.E-08	9.E-01	6.E-03	2.E-03	9.E-01
Benthic Invertebrate	Sed_pond	mg/kg wet weight	3.E-09	8.E-09	5.E-02	3.E-04	7.E-03	6.E-02
Benthic Omnivore	Sed_pond	mg/kg wet weight	5.E-08	1.E-07	1.E-02	2.E-12	2.E-02	3.E-02
Benthic Carnivore	Sed_pond	mg/kg wet weight	4.E-07	9.E-07	1.E-02	8.E-13	5.E-02	7.E-02
Soil - Surface	SurfSoil_source	μg/g dry weight	1.E-06	3.E-06	3.E+01	5.E-03	5.E-01	3.E+01
Soil - Surface	SurfSoil_N1	μg/g dry weight	2.E-07	6.E-07	2.E+00	4.E-04	3.E-02	2.E+00
Leaf - Grasses/Herbs [b]	SurfSoil_N1	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle – Grasses/Herbs	SurfSoil_N1	mg/kg wet weight	1.E-04	3.E-04	3.E+01	3.E-06	2.E-07	3.E+01
Root - Grasses/Herbs	SurfSoil_N1	mg/kg wet weight	2.E-12	6.E-12	1.E-04	0.E+00	1.E-05	1.E-04
Stem - Grasses/Herbs	SurfSoil_N1	mg/kg wet weight	3.E-08	7.E-08	3.E-02	2.E-09	4.E-10	3.E-02
Soil - Surface	SurfSoil_N6	μg/g dry weight	1.E-08	3.E-08	3.E-01	1.E-02	5.E-03	3.E-01
Soil - Surface	SurfSoil_N7	μg/g dry weight	2.E-07	4.E-07	1.E+00	2.E-04	2.E-02	1.E+00
Leaf - Grasses/Herbs [b]	SurfSoil_N7	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle – Grasses/Herbs	SurfSoil_N7	mg/kg wet weight	8.E-05	2.E-04	2.E+01	2.E-06	2.E-07	2.E+01
Root - Grasses/Herbs	SurfSoil_N7	mg/kg wet weight	2.E-12	4.E-12	8.E-05	0.E+00	9.E-06	9.E-05

Exhibit 1-2. Annually Averaged Concentrations at Year 50 for All Compartments in the TRIM.FaTE Screening Scenario using Ravena Emission Rates [a]

Compartment	Volume Element	Units	Mean TCDD	UCL TCDD	Divalent Mercury	Elemental Mercury	Methyl Mercury	Total Mercury
Stem - Grasses/Herbs	SurfSoil_N7	mg/kg wet weight	2.E-08	5.E-08	2.E-02	1.E-09	3.E-10	2.E-02
Soil - Surface	SurfSoil_N3	μg/g dry weight	1.E-07	3.E-07	9.E-01	2.E-04	2.E-02	1.E+00
Leaf - Grasses/Herbs [b]	SurfSoil_N3	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Grasses/Herbs	SurfSoil_N3	mg/kg wet weight	5.E-05	1.E-04	1.E+01	1.E-06	1.E-07	1.E+01
Root - Grasses/Herbs	SurfSoil_N3	mg/kg wet weight	1.E-12	3.E-12	6.E-05	0.E+00	6.E-06	6.E-05
Stem - Grasses/Herbs	SurfSoil_N3	mg/kg wet weight	1.E-08	3.E-08	1.E-02	8.E-10	2.E-10	1.E-02
Soil - Surface	SurfSoil_N4	μg/g dry weight	7.E-08	2.E-07	6.E-01	1.E-04	1.E-02	6.E-01
Leaf - Coniferous Forest	SurfSoil_N4	mg/kg wet weight	4.E-08	1.E-07	6.E-02	1.E-08	2.E-12	6.E-02
Leaf Particle - Coniferous Forest	SurfSoil_N4	mg/kg wet weight	4.E-05	9.E-05	1.E+01	2.E-06	3.E-10	1.E+01
Soil - Surface	SurfSoil_N5	μg/g dry weight	3.E-08	9.E-08	2.E-01	4.E-05	4.E-03	2.E-01
Leaf - Coniferous Forest	SurfSoil_N5	mg/kg wet weight	2.E-08	6.E-08	3.E-02	5.E-09	9.E-13	3.E-02
Leaf Particle - Coniferous Forest	SurfSoil_N5	mg/kg wet weight	2.E-05	5.E-05	4.E+00	9.E-07	1.E-10	4.E+00
Soil - Surface	SurfSoil_S1	μg/g dry weight	2.E-07	6.E-07	2.E+00	4.E-04	3.E-02	2.E+00
Leaf - Grasses/Herbs	SurfSoil_S1	mg/kg wet weight	-	-	-	-	-	-
Leaf Particle - Grasses/Herbs	SurfSoil_S1	mg/kg wet weight	1.E-04	3.E-04	3.E+01	3.E-06	3.E-07	3.E+01
Root - Grasses/Herbs	SurfSoil_S1	mg/kg wet weight	2.E-12	6.E-12	1.E-04	0.E+00	1.E-05	1.E-04
Stem - Grasses/Herbs	SurfSoil_S1	mg/kg wet weight	3.E-08	7.E-08	3.E-02	2.E-09	4.E-10	3.E-02
Soil - Surface	SurfSoil_S4	μg/g dry weight	1.E-07	2.E-07	1.E+00	2.E-04	2.E-02	1.E+00
Leaf - Coniferous Forest	SurfSoil_S4	mg/kg wet weight	4.E-08	1.E-07	6.E-02	1.E-08	3.E-10	6.E-02
Leaf Particle - Coniferous Forest	SurfSoil_S4	mg/kg wet weight	4.E-05	9.E-05	1.E+01	2.E-06	5.E-08	1.E+01
Soil - Surface	SurfSoil_S5	μg/g dry weight	4.E-08	1.E-07	3.E-01	6.E-05	5.E-03	3.E-01
Leaf - Coniferous Forest	SurfSoil_S5	mg/kg wet weight	2.E-08	6.E-08	3.E-02	5.E-09	2.E-10	3.E-02
Leaf Particle - Coniferous Forest	SurfSoil_S5	mg/kg wet weight	2.E-05	5.E-05	4.E+00	9.E-07	3.E-08	4.E+00
Soil - Root Zone	RootSoil_source	μg/g dry weight	2.E-10	4.E-10	3.E-03	3.E-03	6.E-05	7.E-03
Soil - Root Zone	RootSoil_N1	μg/g dry weight	4.E-11	9.E-11	3.E-04	3.E-04	6.E-06	6.E-04
Soil - Root Zone	RootSoil_N6	μg/g dry weight	5.E-13	1.E-12	1.E-05	2.E-03	2.E-07	2.E-03
Soil - Root Zone	RootSoil_N7	μg/g dry weight	3.E-11	6.E-11	2.E-04	2.E-04	4.E-06	4.E-04
Soil - Root Zone	RootSoil_N3	μg/g dry weight	2.E-11	5.E-11	2.E-04	1.E-04	3.E-06	3.E-04

Exhibit 1-2. Annually Averaged Concentrations at Year 50 for All Compartments in the TRIM.FaTE Screening Scenario using Ravena Emission Rates [a]

Compartment	Volume Element	Units	Mean TCDD	UCL TCDD	Divalent Mercury	Elemental Mercury	Methyl Mercury	Total Mercury
Soil - Root Zone	RootSoil_N4	μg/g dry weight	1.E-11	3.E-11	9.E-05	8.E-05	2.E-06	2.E-04
Soil - Root Zone	RootSoil_N5	μg/g dry weight	5.E-12	1.E-11	4.E-05	3.E-05	6.E-07	7.E-05
Soil - Root Zone	RootSoil_S1	μg/g dry weight	4.E-11	9.E-11	3.E-04	3.E-04	6.E-06	6.E-04
Soil - Root Zone	RootSoil_S4	μg/g dry weight	1.E-11	4.E-11	1.E-04	1.E-04	2.E-06	2.E-04
Soil - Root Zone	RootSoil_S5	μg/g dry weight	6.E-12	2.E-11	5.E-05	4.E-05	8.E-07	9.E-05
Soil - Vadose Zone	VadoseSoil_source	g/g dry weight	2.E-24	5.E-24	8.E-17	4.E-13	1.E-18	4.E-13
Soil - Vadose Zone	VadoseSoil_N1	g/g dry weight	5.E-25	1.E-24	1.E-17	6.E-14	2.E-19	6.E-14
Soil - Vadose Zone	VadoseSoil_N6	g/g dry weight	5.E-26	1.E-25	2.E-18	2.E-12	4.E-20	2.E-12
Soil - Vadose Zone	VadoseSoil_N7	g/g dry weight	4.E-25	9.E-25	7.E-18	4.E-14	1.E-19	4.E-14
Soil - Vadose Zone	VadoseSoil_N3	g/g dry weight	3.E-25	6.E-25	5.E-18	3.E-14	9.E-20	3.E-14
Soil - Vadose Zone	VadoseSoil_N4	g/g dry weight	1.E-25	4.E-25	3.E-18	1.E-14	4.E-20	1.E-14
Soil - Vadose Zone	VadoseSoil_N5	g/g dry weight	7.E-26	2.E-25	1.E-18	6.E-15	2.E-20	6.E-15
Soil - Vadose Zone	VadoseSoil_S1	g/g dry weight	5.E-25	1.E-24	1.E-17	6.E-14	2.E-19	6.E-14
Soil - Vadose Zone	VadoseSoil_S4	g/g dry weight	2.E-25	5.E-25	3.E-18	2.E-14	6.E-20	2.E-14
Soil - Vadose Zone	VadoseSoil_S5	g/g dry weight	8.E-26	2.E-25	1.E-18	7.E-15	2.E-20	7.E-15
Groundwater	GW_source	g/L	4.E-32	1.E-31	1.E-23	3.E-19	2.E-25	3.E-19
Groundwater	GW_N1	g/L	9.E-33	2.E-32	2.E-24	4.E-20	3.E-26	4.E-20
Groundwater	GW_N6	g/L	1.E-33	2.E-33	4.E-23	1.E-18	7.E-25	1.E-18
Groundwater	GW_N7	g/L	7.E-33	2.E-32	1.E-24	3.E-20	2.E-26	3.E-20
Groundwater	GW_N3	g/L	5.E-33	1.E-32	9.E-25	2.E-20	1.E-26	2.E-20
Groundwater	GW_N4	g/L	3.E-33	7.E-33	4.E-25	9.E-21	7.E-27	9.E-21
Groundwater	GW_N5	g/L	1.E-33	3.E-33	2.E-25	4.E-21	3.E-27	4.E-21
Groundwater	GW_S1	g/L	9.E-33	2.E-32	2.E-24	4.E-20	3.E-26	4.E-20
Groundwater	GW_S4	g/L	4.E-33	9.E-33	5.E-25	1.E-20	8.E-27	1.E-20
Groundwater	GW_S5	g/L	2.E-33	4.E-33	2.E-25	4.E-21	3.E-27	4.E-21

[a] For more information of the TRIM.FaTE screening scenario, refer to Appendix C.
[b] Annually averaged leaf concentrations are unavailable because of the seasonally changing leaf compartments.

I-2.2 Detailed 2,3,7,8 – TCDD Cancer Risk and Hazard Quotient Results

This section provides tables showing detailed cancer risk and hazard quotient modeling estimates for 2,3,7,8 – TCDD for all the different ingestion scenarios (combinations of the selected soil compartment, water body compartment, ingestion rate, and emission rate) considered. Exhibit 2-1 through Exhibit 2-4 provide the estimated hazard quotients and individual lifetime cancer risk estimates using the combinations of 95th percentile (upper confidence limit, or UCL) and mean emission rates as well as 90th percentile (reasonable maximum exposure, or RME) and mean (central tendency exposure, or CTE) ingestion rates. Exhibit 2-5 gives detailed individual lifetime cancer risk and age-specific hazard quotient estimates broken down by different ingestion pathways. Exhibit 2-6 and Exhibit 2-7 provide comparisons and percent changes in individual lifetime cancer risk when using either UCL or mean emission rates and using either RME or CTE ingestion rates, respectively. Finally, Exhibit 2-8 provides dermal hazard quotients and risk estimates due to exposure to water in Alcove Reservoir for all emission rates and age groups.

Exhibit 2-1. Estimated Hazard Quotients and Individual Lifetime Cancer Risks for 2,3,7,8-TCDD Using UCL Emission Rate and RME Ingestion Rates

Note: HQs greater than 1 and risks greater than 1E-06 are in boldface type.

Scenario Type	Water body	Farm Parcel	Harvesting from Ravena Pond	Chronic Non-Cancer Hazard Quotient					Individual Lifetime Cancer Risk
				Child (1-2)	Child (3-5)	Child (6-11)	Child (12-19)	Adult (20-70)	
Screening	Screening	Screening	n/a	**1.456**	**1.086**	0.892	0.480	0.739	**1.1E-04**
Combined	Ravena Pond	West	Harvesting	0.971	0.950	0.800	0.508	0.863	**1.2E-04**
			No Harvesting	**1.314**	**1.293**	**1.089**	0.694	**1.180**	**1.7E-04**
		East	Harvesting	0.967	0.948	0.798	0.507	0.862	**1.2E-04**
			No Harvesting	**1.310**	**1.291**	**1.087**	0.693	**1.179**	**1.7E-04**
	Nassau Lake	West	n/a	0.084	0.062	0.051	0.027	0.042	**6.4E-06**
		East	n/a	0.080	0.060	0.049	0.026	0.041	**6.2E-06**
	Kinderhook Lake	West	n/a	0.066	0.044	0.036	0.017	0.025	**4.0E-06**
		East	n/a	0.062	0.042	0.034	0.017	0.024	**3.8E-06**
	Alcove Reservoir	West	n/a	0.060	0.038	0.030	0.014	0.019	**3.2E-06**
		East	n/a	0.056	0.035	0.029	0.013	0.018	**3.0E-06**
Farmer Only	-	West	n/a	0.058	0.035	0.029	0.013	0.017	**2.9E-06**
	-	East	n/a	0.053	0.033	0.027	0.012	0.016	**2.7E-06**
Angler Only	Pond	-	Harvesting	0.914	0.915	0.771	0.495	0.846	**1.2E-04**
		-	No Harvesting	**1.256**	0.681	**1.258**	**1.060**	**1.163**	**1.6E-04**
	Nassau Lake	-	n/a	0.026	0.027	0.022	0.014	0.025	**3.4E-06**
	Kinderhook Lake	-	n/a	0.009	0.009	0.007	0.005	0.008	**1.1E-06**
	Alcove Reservoir	-	n/a	0.002	0.002	0.002	0.001	0.002	2.8E-07
Water Ingestion Only	-	-	n/a	0.000	0.000	0.000	0.000	0.000	1.3E-13

Exhibit 2-2. Estimated Hazard Quotients and Individual Lifetime Cancer Risks for 2,3,7,8-TCDD Using UCL Emission Rate and CTE Ingestion Rates

Note: HQs greater than 1 and risks greater than 1E-06 are in boldface type.

Scenario Type	Water body	Farm Parcel	Harvesting from Ravena Pond	Chronic Non-Cancer Hazard Quotient					Individual Lifetime Cancer Risk
				Child (1-2)	Child (3-5)	Child (6-11)	Child (12-19)	Adult (20-70)	
Screening	Screening	Screening	n/a	0.632	0.463	0.362	0.206	0.323	**4.8E-05**
Combined	Ravena Pond	West	Harvesting	0.412	0.403	0.315	0.221	0.352	**5.0E-05**
			No Harvesting	0.557	0.549	0.428	0.302	0.480	**6.8E-05**
		East	Harvesting	0.410	0.402	0.314	0.221	0.351	**5.0E-05**
			No Harvesting	0.555	0.548	0.428	0.302	0.480	**6.8E-05**
	Nassau Lake	West	n/a	0.037	0.027	0.021	0.012	0.018	**2.8E-06**
		East	n/a	0.035	0.026	0.020	0.011	0.018	**2.7E-06**
	Kinderhook Lake	West	n/a	0.029	0.019	0.015	0.007	0.012	**1.8E-06**
		East	n/a	0.027	0.018	0.014	0.007	0.011	**1.7E-06**
	Alcove Reservoir	West	n/a	0.027	0.016	0.013	0.006	0.009	**1.5E-06**
		East	n/a	0.025	0.015	0.012	0.006	0.009	**1.4E-06**
Farmer Only	-	West	n/a	0.026	0.015	0.012	0.005	0.008	**1.4E-06**
	-	East	n/a	0.024	0.014	0.011	0.005	0.008	**1.3E-06**
Angler Only	Pond	-	Harvesting	0.386	0.388	0.303	0.216	0.343	**4.9E-05**
		-	No Harvesting	0.531	0.297	0.533	0.416	0.472	**6.7E-05**
	Nassau Lake	-	n/a	0.011	0.011	0.009	0.006	0.010	**1.4E-06**
	Kinderhook Lake	-	n/a	0.004	0.004	0.003	0.002	0.003	4.5E-07
	Alcove Reservoir	-	n/a	0.001	0.001	0.001	0.001	0.001	1.2E-07
Water Ingestion Only	-	-	n/a	0.000	0.000	0.000	0.000	0.000	6.2E-14

Exhibit 2-3. Estimated Hazard Quotients and Individual Lifetime Cancer Risks for 2,3,7,8-TCDD Using Mean Emission Rate and RME Ingestion Rates

Note: HQs greater than 1 and risks greater than 1E-06 are highlighted in boldface type.

Scenario Type	Water body	Farm Parcel	Harvesting from Ravena Pond	Chronic Non-Cancer Hazard Quotient					Individual Lifetime Cancer Risk
				Child (1-2)	Child (3-5)	Child (6-11)	Child (12-19)	Adult (20-70)	
Screening	Screening	Screening	n/a	0.596	0.444	0.365	0.196	0.302	**4.6E-05**
Combined	Ravena Pond	West	Harvesting	0.398	0.389	0.327	0.208	0.353	**5.0E-05**
			No Harvesting	0.538	0.529	0.445	0.284	0.483	**6.8E-05**
		East	Harvesting	0.396	0.388	0.327	0.208	0.353	**5.0E-05**
			No Harvesting	0.536	0.528	0.445	0.284	0.483	**6.8E-05**
	Nassau Lake	West	n/a	0.034	0.025	0.021	0.011	0.017	**2.6E-06**
		East	n/a	0.033	0.024	0.020	0.011	0.017	**2.5E-06**
	Kinderhook Lake	West	n/a	0.027	0.018	0.015	0.007	0.010	**1.6E-06**
		East	n/a	0.025	0.017	0.014	0.007	0.010	**1.6E-06**
	Alcove Reservoir	West	n/a	0.024	0.015	0.012	0.006	0.008	**1.3E-06**
		East	n/a	0.023	0.014	0.012	0.005	0.007	**1.2E-06**
Farmer Only	-	West	n/a	0.024	0.014	0.012	0.005	0.007	**1.2E-06**
	-	East	n/a	0.022	0.013	0.011	0.005	0.007	**1.1E-06**
Angler Only	Pond	-	Harvesting	0.374	0.374	0.316	0.203	0.346	**4.9E-05**
		-	No Harvesting	0.514	0.279	0.515	0.434	0.476	**6.7E-05**
	Nassau Lake	-	n/a	0.011	0.011	0.009	0.006	0.010	**1.4E-06**
	Kinderhook Lake	-	n/a	0.003	0.004	0.003	0.002	0.003	4.6E-07
	Alcove Reservoir	-	n/a	0.001	0.001	0.001	0.000	0.001	1.2E-07
Water Ingestion Only	-	-	n/a	0.000	0.000	0.000	0.000	0.000	5.2E-14

Exhibit 2-4. Estimated Hazard Quotients and Individual Lifetime Cancer Risks for 2,3,7,8-TCDD Using Mean Emission Rate and CTE Ingestion Rates

Note: HQs greater than 1 and risks greater than 1E-06 are highlighted in boldface type.

Scenario Type	Water body	Farm Parcel	Harvesting from Ravena Pond	Chronic Non-Cancer Hazard Quotient					Individual Lifetime Cancer Risk
				Child (1-2)	Child (3-5)	Child (6-11)	Child (12-19)	Adult (20-70)	
Screening	Screening	Screening	n/a	0.258	0.189	0.148	0.084	0.132	**2.0E-05**
Combined	Ravena Pond	West	Harvesting	0.169	0.165	0.129	0.091	0.144	**2.0E-05**
			No Harvesting	0.228	0.224	0.175	0.124	0.197	**2.8E-05**
		East	Harvesting	0.168	0.165	0.129	0.090	0.144	**2.0E-05**
			No Harvesting	0.227	0.224	0.175	0.124	0.196	**2.8E-05**
	Nassau Lake	West	n/a	0.015	0.011	0.008	0.005	0.008	**1.1E-06**
		East	n/a	0.014	0.010	0.008	0.005	0.007	**1.1E-06**
	Kinderhook Lake	West	n/a	0.012	0.008	0.006	0.003	0.005	7.4E-07
		East	n/a	0.011	0.007	0.006	0.003	0.005	7.1E-07
	Alcove Reservoir	West	n/a	0.011	0.007	0.005	0.002	0.004	6.0E-07
		East	n/a	0.010	0.006	0.005	0.002	0.004	5.7E-07
Farmer Only	-	West	n/a	0.011	0.006	0.005	0.002	0.003	5.5E-07
	-	East	n/a	0.010	0.006	0.005	0.002	0.003	5.2E-07
Angler Only	Pond	-	Harvesting	0.158	0.159	0.124	0.088	0.141	**2.0E-05**
		-	No Harvesting	0.217	0.121	0.218	0.170	0.193	**2.7E-05**
	Nassau Lake	-	n/a	0.005	0.005	0.004	0.003	0.004	5.8E-07
	Kinderhook Lake	-	n/a	0.001	0.001	0.001	0.001	0.001	1.9E-07
	Alcove Reservoir	-	n/a	0.000	0.000	0.000	0.000	0.000	4.7E-08
Water Ingestion Only	-	-	n/a	0.000	0.000	0.000	0.000	0.000	2.5E-14

Exhibit 2-5. Fractional Pathway of Cancer Risks and Age-Specific Hazard Quotients for 2,3,7,8-TCDD for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult 20-70	Cancer
UCL Emission Rate, CTE Ingestion Rate, Alcove Reservoir, East Farm						
Fruits & Vegetables	0.7%	0.9%	1.1%	1.4%	1.6%	1.4%
Egg, Pork, & Poultry	2.0%	3.0%	2.7%	4.5%	3.2%	3.2%
Beef & Dairy	93.6%	90.0%	90.2%	85.0%	85.8%	87.1%
Fish	3.7%	6.0%	6.0%	9.1%	9.4%	8.3%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, CTE Ingestion Rate, Alcove Reservoir, West Farm						
Fruits & Vegetables	0.7%	1.0%	1.1%	1.5%	1.7%	1.5%
Egg, Pork, & Poultry	1.4%	2.2%	2.0%	3.3%	2.4%	2.3%
Beef & Dairy	94.3%	91.2%	91.2%	86.5%	87.0%	88.3%
Fish	3.4%	5.7%	5.7%	8.6%	8.9%	7.8%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, CTE ngestion Rate, Kinderhook Lake, East Farm						
Fruits & Vegetables	0.6%	0.8%	0.9%	1.1%	1.2%	1.1%
Egg, Pork, & Poultry	1.8%	2.6%	2.3%	3.6%	2.5%	2.5%
Beef & Dairy	84.3%	76.4%	76.6%	67.0%	67.2%	70.0%
Fish	13.2%	20.2%	20.2%	28.3%	29.0%	26.4%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, CTE Ingestion Rate, Kinderhook Lake, West Farm						
Fruits & Vegetables	0.7%	0.8%	1.0%	1.2%	1.3%	1.2%
Egg, Pork, & Poultry	1.3%	1.9%	1.7%	2.6%	1.9%	1.9%
Beef & Dairy	85.7%	78.2%	78.2%	69.0%	69.0%	71.8%
Fish	12.3%	19.1%	19.1%	27.1%	27.7%	25.1%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, CTE Ingestion Rate, Nassau Lake, East Farm						
Fruits & Vegetables	0.5%	0.5%	0.6%	0.7%	0.8%	0.7%
Egg, Pork, & Poultry	1.4%	1.8%	1.6%	2.2%	1.6%	1.6%
Beef & Dairy	66.1%	53.7%	53.9%	42.0%	41.8%	45.1%
Fish	32.0%	44.0%	43.9%	55.0%	55.8%	52.6%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, CTE Ingestion Rate, Nassau Lake, West Farm						
Fruits & Vegetables	0.5%	0.6%	0.7%	0.8%	0.9%	0.8%
Egg, Pork, & Poultry	1.0%	1.3%	1.2%	1.7%	1.2%	1.2%
Beef & Dairy	68.0%	55.8%	55.8%	44.0%	43.7%	47.0%
Fish	30.4%	42.2%	42.3%	53.6%	54.3%	51.0%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 2-5. Fractional Pathway of Cancer Risks and Age-Specific Hazard Quotients for 2,3,7,8-TCDD for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult 20-70	Cancer
UCL Emission Rate, CTE Ingestion Rate, Ravena Pond, East Farm						
Fruits & Vegetables	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Egg, Pork, & Poultry	0.1%	0.1%	0.1%	0.1%	0.1%	0.1%
Beef & Dairy	5.6%	3.4%	3.4%	2.2%	2.1%	2.4%
Fish	94.2%	96.4%	96.4%	97.7%	97.8%	97.5%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, CTE Ingestion Rate, Ravena Pond, West Farm						
Fruits & Vegetables	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Egg, Pork, & Poultry	0.1%	0.1%	0.1%	0.1%	0.1%	0.1%
Beef & Dairy	6.1%	3.7%	3.7%	2.3%	2.3%	2.6%
Fish	93.8%	96.2%	96.2%	97.5%	97.6%	97.3%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, CTE Ingestion Rate, Alcove Reservoir, East Farm						
Fruits & Vegetables	0.8%	0.9%	1.2%	1.4%	1.8%	1.5%
Egg, Pork, & Poultry	1.8%	2.7%	2.3%	4.3%	3.6%	3.3%
Beef & Dairy	93.3%	90.1%	90.1%	85.4%	83.5%	85.7%
Fish	3.9%	6.2%	6.4%	8.9%	11.0%	9.4%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, RME Ingestion Rate, Alcove Reservoir, West Farm						
Fruits & Vegetables	0.9%	0.9%	1.3%	1.5%	1.9%	1.7%
Egg, Pork, & Poultry	1.3%	2.0%	1.7%	3.2%	2.7%	2.4%
Beef & Dairy	94.1%	91.2%	91.0%	86.9%	84.9%	87.0%
Fish	3.6%	5.8%	6.0%	8.4%	10.4%	8.8%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, RME Ingestion Rate, Kinderhook Lake, East Farm						
Fruits & Vegetables	0.8%	0.8%	1.0%	1.1%	1.3%	1.2%
Egg, Pork, & Poultry	1.6%	2.3%	1.9%	3.4%	2.7%	2.6%
Beef & Dairy	83.7%	76.2%	75.9%	67.7%	63.1%	67.2%
Fish	13.8%	20.6%	21.1%	27.7%	32.8%	28.9%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, RME Ingestion Rate, Kinderhook Lake, West Farm						
Fruits & Vegetables	0.8%	0.8%	1.1%	1.2%	1.5%	1.3%
Egg, Pork, & Poultry	1.2%	1.7%	1.4%	2.5%	2.0%	1.9%
Beef & Dairy	85.0%	77.9%	77.3%	69.7%	65.0%	69.1%
Fish	12.9%	19.5%	20.1%	26.5%	31.5%	27.6%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 2-5. Fractional Pathway of Cancer Risks and Age-Specific Hazard Quotients for 2,3,7,8-TCDD for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult 20-70	Cancer
UCL Emission Rate, RME Ingestion Rate, Nassau Lake, East Farm						
Fruits & Vegetables	0.6%	0.5%	0.7%	0.7%	0.8%	0.8%
Egg, Pork, & Poultry	1.3%	1.6%	1.3%	2.2%	1.6%	1.6%
Beef & Dairy	64.9%	53.2%	52.6%	42.8%	37.4%	41.8%
Fish	33.1%	44.6%	45.4%	54.3%	60.2%	55.8%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, RME Ingestion Rate, Nassau Lake, West Farm						
Fruits & Vegetables	0.6%	0.6%	0.8%	0.8%	0.9%	0.8%
Egg, Pork, & Poultry	0.9%	1.2%	1.0%	1.6%	1.2%	1.2%
Beef & Dairy	66.9%	55.3%	54.4%	44.8%	39.1%	43.7%
Fish	31.5%	42.9%	43.8%	52.8%	58.7%	54.2%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, RME Ingestion Rate, Ravena Pond, East Farm						
Fruits & Vegetables	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Egg, Pork, & Poultry	0.1%	0.1%	0.1%	0.1%	0.1%	0.1%
Beef & Dairy	5.4%	3.3%	3.2%	2.2%	1.8%	2.1%
Fish	94.5%	96.5%	96.6%	97.6%	98.1%	97.8%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
UCL Emission Rate, RME Ingestion Rate, Ravena Pond, West Farm						
Fruits & Vegetables	0.1%	0.0%	0.0%	0.0%	0.0%	0.0%
Egg, Pork, & Poultry	0.1%	0.1%	0.1%	0.1%	0.1%	0.1%
Beef & Dairy	5.8%	3.6%	3.5%	2.4%	1.9%	2.3%
Fish	94.1%	96.3%	96.4%	97.5%	98.0%	97.6%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, CTE Ingestion Rate, Alcove Reservoir, East Farm						
Fruits & Vegetables	0.7%	0.9%	1.1%	1.4%	1.6%	1.4%
Egg, Pork, & Poultry	2.0%	3.0%	2.7%	4.5%	3.2%	3.2%
Beef & Dairy	93.6%	90.0%	90.2%	85.0%	85.8%	87.1%
Fish	3.7%	6.0%	6.0%	9.1%	9.4%	8.3%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, CTE Ingestion Rate, Alcove Reservoir, West Farm						
Fruits & Vegetables	0.7%	1.0%	1.1%	1.5%	1.7%	1.5%
Egg, Pork, & Poultry	1.4%	2.2%	2.0%	3.3%	2.4%	2.3%
Beef & Dairy	94.3%	91.2%	91.2%	86.5%	87.0%	88.3%
Fish	3.4%	5.7%	5.7%	8.6%	8.9%	7.8%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 2-5. Fractional Pathway of Cancer Risks and Age-Specific Hazard Quotients for 2,3,7,8-TCDD for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult 20-70	Cancer
Mean Emission Rate, CTE Ingestion Rate, Kinderhook Lake, East Farm						
Fruits & Vegetables	0.6%	0.8%	0.9%	1.1%	1.2%	1.1%
Egg, Pork, & Poultry	1.8%	2.6%	2.3%	3.6%	2.5%	2.5%
Beef & Dairy	84.3%	76.4%	76.6%	67.0%	67.2%	70.0%
Fish	13.2%	20.2%	20.2%	28.3%	29.0%	26.4%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, CTE Ingestion Rate, Kinderhook Lake, West Farm						
Fruits & Vegetables	0.7%	0.8%	1.0%	1.2%	1.3%	1.2%
Egg, Pork, & Poultry	1.3%	1.9%	1.7%	2.6%	1.9%	1.9%
Beef & Dairy	85.7%	78.2%	78.2%	69.0%	69.0%	71.8%
Fish	12.3%	19.1%	19.1%	27.1%	27.7%	25.1%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, CTE Ingestion Rate, Nassau Lake, East Farm						
Fruits & Vegetables	0.5%	0.5%	0.6%	0.7%	0.8%	0.7%
Egg, Pork, & Poultry	1.4%	1.8%	1.6%	2.2%	1.6%	1.6%
Beef & Dairy	66.1%	53.7%	53.9%	42.0%	41.8%	45.1%
Fish	32.0%	44.0%	43.9%	55.0%	55.8%	52.6%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, CTE Ingestion Rate, Nassau Lake, West Farm						
Fruits & Vegetables	0.5%	0.6%	0.7%	0.8%	0.9%	0.8%
Egg, Pork, & Poultry	1.0%	1.3%	1.2%	1.7%	1.2%	1.2%
Beef & Dairy	68.0%	55.8%	55.8%	44.0%	43.7%	47.0%
Fish	30.4%	42.2%	42.3%	53.6%	54.3%	51.0%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, CTE Ingestion Rate, Ravena Pond, East Farm						
Fruits & Vegetables	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Egg, Pork, & Poultry	0.1%	0.1%	0.1%	0.1%	0.1%	0.1%
Beef & Dairy	5.6%	3.4%	3.4%	2.2%	2.1%	2.4%
Fish	94.2%	96.4%	96.4%	97.7%	97.8%	97.5%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, CTE Ingestion Rate, Ravena Pond, West Farm						
Fruits & Vegetables	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Egg, Pork, & Poultry	0.1%	0.1%	0.1%	0.1%	0.1%	0.1%
Beef & Dairy	6.1%	3.7%	3.7%	2.3%	2.3%	2.6%
Fish	93.8%	96.2%	96.2%	97.5%	97.6%	97.3%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 2-5. Fractional Pathway of Cancer Risks and Age-Specific Hazard Quotients for 2,3,7,8-TCDD for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult 20-70	Cancer
Mean Emission Rate, RME Ingestion Rate, Alcove Reservoir, East Farm						
Fruits & Vegetables	0.8%	0.9%	1.2%	1.4%	1.8%	1.5%
Egg, Pork, & Poultry	1.8%	2.7%	2.3%	4.3%	3.6%	3.3%
Beef & Dairy	93.3%	90.1%	90.1%	85.4%	83.5%	85.7%
Fish	3.9%	6.2%	6.4%	8.9%	11.0%	9.4%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	***0.0%***
Mean Emission Rate, RME Ingestion Rate, Alcove Reservoir, West Farm						
Fruits & Vegetables	0.9%	0.9%	1.3%	1.5%	1.9%	1.7%
Egg, Pork, & Poultry	1.3%	2.0%	1.7%	3.2%	2.7%	2.4%
Beef & Dairy	94.1%	91.2%	91.0%	86.9%	84.9%	87.0%
Fish	3.6%	5.8%	6.0%	8.4%	10.4%	8.8%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, RME Ingestion Rate, Kinderhook Lake, East Farm						
Fruits & Vegetables	0.8%	0.8%	1.0%	1.1%	1.3%	1.2%
Egg, Pork, & Poultry	1.6%	2.3%	1.9%	3.4%	2.7%	2.6%
Beef & Dairy	83.7%	76.2%	75.9%	67.7%	63.1%	67.2%
Fish	13.8%	20.6%	21.1%	27.7%	32.8%	28.9%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, RME Ingestion Rate, Kinderhook Lake, West Farm						
Fruits & Vegetables	0.8%	0.8%	1.1%	1.2%	1.5%	1.3%
Egg, Pork, & Poultry	1.2%	1.7%	1.4%	2.5%	2.0%	1.9%
Beef & Dairy	85.0%	77.9%	77.3%	69.7%	65.0%	69.1%
Fish	12.9%	19.5%	20.1%	26.5%	31.5%	27.6%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, RME Ingestion Rate, Nassau Lake, East Farm						
Fruits & Vegetables	0.6%	0.5%	0.7%	0.7%	0.8%	0.8%
Egg, Pork, & Poultry	1.3%	1.6%	1.3%	2.2%	1.6%	1.6%
Beef & Dairy	64.9%	53.2%	52.6%	42.8%	37.4%	41.8%
Fish	33.1%	44.6%	45.4%	54.3%	60.2%	55.8%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, RME Ingestion Rate, Nassau Lake, West Farm						
Fruits & Vegetables	0.6%	0.6%	0.8%	0.8%	0.9%	0.8%
Egg, Pork, & Poultry	0.9%	1.2%	1.0%	1.6%	1.2%	1.2%
Beef & Dairy	66.9%	55.3%	54.4%	44.8%	39.1%	43.7%
Fish	31.5%	42.9%	43.8%	52.8%	58.7%	54.2%
Soil	0.1%	0.1%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 2-5. Fractional Pathway of Cancer Risks and Age-Specific Hazard Quotients for 2,3,7,8-TCDD for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult 20-70	Cancer
Mean Emission Rate, RME Ingestion Rate, Ravena Pond, East Farm						
Fruits & Vegetables	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Egg, Pork, & Poultry	0.1%	0.1%	0.1%	0.1%	0.1%	0.1%
Beef & Dairy	5.4%	3.3%	3.2%	2.2%	1.8%	2.1%
Fish	94.5%	96.5%	96.6%	97.6%	98.1%	97.8%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Mean Emission Rate, RME Ingestion Rate, Ravena Pond, West Farm						
Fruits & Vegetables	0.1%	0.0%	0.0%	0.0%	0.0%	0.0%
Egg, Pork, & Poultry	0.1%	0.1%	0.1%	0.1%	0.1%	0.1%
Beef & Dairy	5.8%	3.6%	3.5%	2.4%	1.9%	2.3%
Fish	94.1%	96.3%	96.4%	97.5%	98.0%	97.6%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 2-6. Comparison of 2,3,7,8-TCDD Ravena Cancer Risks Using UCL Versus Mean Emission Rates [a]

Scenario Type	Farm Parcel	Water body	Mean Emission Factor	UCL Emission Factor	Ratio of UCL : Mean EF
Screening	Screening	Screening	4.6E-05	1.1E-04	2.4
Combined	West	Alcove	1.3E-06	3.2E-06	2.4
		Kinderhook	1.6E-06	4.0E-06	2.4
		Nassau	2.6E-06	6.4E-06	2.4
		Pond	5.0E-05	1.2E-04	2.4
	East	Alcove	1.2E-06	3.0E-06	2.4
		Kinderhook	1.6E-06	3.8E-06	2.4
		Nassau	2.5E-06	6.2E-06	2.4
		Pond	5.0E-05	1.2E-04	2.4
Farm Only	West	-	1.2E-06	2.9E-06	2.4
	East	-	1.1E-06	2.7E-06	2.4
Fisherman Only	-	Alcove	1.2E-07	2.8E-07	2.4
		Kinderhook	4.6E-07	1.1E-06	2.4
		Nassau	1.4E-06	3.4E-06	2.4
		Pond	4.9E-05	1.2E-04	2.4
Water Ingestion Only	-	-	5.2E-14	1.3E-13	2.4

[a] Selected scenarios use RME percentile ingestion rates and contain a fish harvester in Ravena Pond

Exhibit 2-7. Comparison of 2,3,7,8-TCDD Ravena Cancer Risks Using CTE Versus RME Ingestion Rates [a]

Emission Rate	Scenario Type	Farm Parcel	Water body	CTE Ingestion Rates	RME Ingestion Rates	Ratio of RME : CTE
UCL	Screening	Screening	Screening	4.8E-05	1.1E-04	2.3
	Combined	West	Alcove	1.5E-06	3.2E-06	2.2
			Kinderhook	1.8E-06	4.0E-06	2.2
			Nassau	2.8E-06	6.4E-06	2.3
			Pond	5.0E-05	1.2E-04	2.4
		East	Alcove	1.4E-06	3.0E-06	2.2
			Kinderhook	1.7E-06	3.8E-06	2.2
			Nassau	2.7E-06	6.2E-06	2.3
			Pond	5.0E-05	1.2E-04	2.4
	Farm Only	West	-	1.4E-06	2.9E-06	2.2
		East	-	1.3E-06	2.7E-06	2.2
	Fisherman Only	-	Alcove	1.2E-07	2.8E-07	2.4
			Kinderhook	4.5E-07	1.1E-06	2.4
			Nassau	1.4E-06	3.4E-06	2.4
			Pond	4.9E-05	1.2E-04	2.4
	Water Ingestion Only	-	-	6.2E-14	1.3E-13	2.1
Mean	Screening	Screening	Screening	2.0E-05	4.6E-05	2.3
	Combined	West	Alcove	6.0E-07	1.3E-06	2.2
			Kinderhook	7.4E-07	1.6E-06	2.2
			Nassau	1.1E-06	2.6E-06	2.3
			Pond	2.0E-05	5.0E-05	2.4
		East	Alcove	5.7E-07	1.2E-06	2.2
			Kinderhook	7.1E-07	1.6E-06	2.2
			Nassau	1.1E-06	2.5E-06	2.3
			Pond	2.0E-05	5.0E-05	2.4
	Farm Only	West	-	5.5E-07	1.2E-06	2.2
		East	-	5.2E-07	1.1E-06	2.2
	Fisherman Only	-	Alcove	4.7E-08	1.2E-07	2.4
			Kinderhook	1.9E-07	4.6E-07	2.4
			Nassau	5.8E-07	1.4E-06	2.4
			Pond	2.0E-05	4.9E-05	2.4
	Water Ingestion Only	-	-	2.5E-14	5.2E-14	2.1

[a] Selected scenarios use UCL emission rates and contain a fish harvester in Ravena Pond

Exhibit 2-8. 2,3,7,8 TCDD Dermal Hazard Quotients and Lifetime Risks for Water and Soil Contact

Age-Specific Hazard Quotient or Lifetime Risk	Exposure Media	UCL Emission Factor					Mean Emission Factor				
		East Farm – Tilled, Alcove Reservoir	East Farm – Untilled, Alcove Reservoir	West Farm – Tilled, Alcove Reservoir	West Farm – Untilled, Alcove Reservoir	Screen	East Farm – Tilled, Alcove Reservoir	East Farm – Untilled, Alcove Reservoir	West Farm – Tilled, Alcove Reservoir	West Farm – Untilled, Alcove Reservoir	Screen
Child < 1 HQ	Soil	2.58E-06	4.42E-05	2.56E-06	3.25E-05	7.82E-04	1.06E-06	1.81E-05	1.05E-06	1.33E-05	3.20E-04
	Water	3.92E-05	3.92E-05	3.92E-05	3.92E-05	9.93E-03	1.60E-05	1.60E-05	1.60E-05	1.60E-05	4.06E-03
	Soil and Water	4.18E-05	8.34E-05	4.17E-05	7.16E-05	1.07E-02	1.71E-05	3.41E-05	1.71E-05	2.93E-05	4.38E-03
Child 1-2 HQ	Soil	2.18E-06	3.73E-05	2.16E-06	2.74E-05	6.60E-04	8.90E-07	1.52E-05	8.82E-07	1.12E-05	2.70E-04
	Water	3.48E-05	3.48E-05	3.48E-05	3.48E-05	8.81E-03	1.42E-05	1.42E-05	1.42E-05	1.42E-05	3.60E-03
	Soil and Water	3.69E-05	7.20E-05	3.69E-05	6.21E-05	9.47E-03	1.51E-05	2.95E-05	1.51E-05	2.54E-05	3.87E-03
Child 3-5 HQ	Soil	2.06E-06	3.52E-05	2.04E-06	2.59E-05	6.23E-04	8.41E-07	1.44E-05	8.34E-07	1.06E-05	2.55E-04
	Water	3.14E-05	3.14E-05	3.14E-05	3.14E-05	7.96E-03	1.28E-05	1.28E-05	1.28E-05	1.28E-05	3.26E-03
	Soil and Water	3.35E-05	6.66E-05	3.34E-05	5.73E-05	8.58E-03	1.37E-05	2.73E-05	1.37E-05	2.34E-05	3.51E-03
Child 6-11 HQ	Soil	1.61E-06	2.76E-05	1.60E-06	2.03E-05	4.89E-04	6.60E-07	1.13E-05	6.54E-07	8.31E-06	2.00E-04
	Water	2.61E-05	2.61E-05	2.61E-05	2.61E-05	6.62E-03	1.07E-05	1.07E-05	1.07E-05	1.07E-05	2.71E-03
	Soil and Water	2.77E-05	5.37E-05	2.77E-05	4.64E-05	7.11E-03	1.13E-05	2.20E-05	1.13E-05	1.90E-05	2.91E-03
Child 12-19 HQ	Soil	1.23E-06	2.10E-05	1.22E-06	1.54E-05	3.72E-04	5.02E-07	8.59E-06	4.97E-07	6.31E-06	1.52E-04
	Water	2.05E-05	2.05E-05	2.05E-05	2.05E-05	5.20E-03	8.40E-06	8.40E-06	8.40E-06	8.40E-06	2.13E-03
	Soil and Water	2.18E-05	4.15E-05	2.17E-05	3.60E-05	5.58E-03	8.90E-06	1.70E-05	8.90E-06	1.47E-05	2.28E-03
Adult HQ	Soil	5.49E-07	9.40E-06	5.44E-07	6.91E-06	1.66E-04	2.25E-07	3.85E-06	2.23E-07	2.83E-06	6.81E-05
	Water	1.49E-05	1.49E-05	1.49E-05	1.49E-05	3.77E-03	6.09E-06	6.09E-06	6.09E-06	6.09E-06	1.54E-03
	Soil and Water	1.54E-05	2.43E-05	1.54E-05	2.18E-05	3.94E-03	6.31E-06	9.94E-06	6.31E-06	8.91E-06	1.61E-03
Lifetime Risk	Soil	1.29E-10	2.20E-09	1.28E-10	1.62E-09	3.90E-08	5.26E-11	9.02E-10	5.22E-11	6.62E-10	1.60E-08
	Water	2.72E-09	2.72E-09	2.72E-09	2.72E-09	6.89E-07	1.11E-09	1.11E-09	1.11E-09	1.11E-09	2.82E-07
	Soil and Water	2.85E-09	4.92E-09	2.84E-09	4.34E-09	7.28E-07	1.16E-09	2.01E-09	1.16E-09	1.77E-09	2.98E-07

I-2.3 Detailed Mercury Hazard Quotient Results

This section provides tables showing detailed hazard quotient modeling estimates for divalent mercury and methyl mercury for all the different ingestion scenarios (combinations of selected soil compartment, water body compartment, and ingestion rate) considered. Exhibit 3-1 and Exhibit 3-2 provide hazard quotient estimates using the 90th percentile (reasonable maximum exposure, or RME) and mean (central tendency exposure, or CTE) ingestion rates for divalent mercury, respectively. Exhibit 3-3 and Exhibit 3-4 provide hazard quotient estimates using the RME and CTE ingestion rates for methyl mercury, respectively. Exhibit 3-5 and Exhibit 3-6 gives detailed age-specific hazard quotient estimates broken down by different ingestion pathways for divalent and methyl mercury, respectively. Exhibit 3-7 highlights the differences in hazard quotient estimates when using the Alcove Reservoir compared to all other water bodies. Exhibit 3-8 compares the hazard quotient estimates generated using the east farm versus the west farm TRIM.FaTE compartments, and Exhibit 3-9 provide comparisons and percent changes in hazard quotient that arise from using either RME or CTE ingestion rates. Finally, Exhibit 3-10 provides dermal hazard quotients due to exposure to water in Alcove reservoir for all age groups.

Exhibit 3-1. Summary Results - Hazard Quotients for Divalent Mercury Scenarios using RME Ingestion Rates

Ingestion Rates	Scenario Type	Water body	Farm Parcel	Harvester in Ravena Pond?	HQ Child (1-2)	HQ Child (3-5)	HQ Child (6-11)	HQ Child (12-19)	HQ Adult (20-70)
RME Ingestion Rate	Screening	Screening	Screening	Harvester	0.344	0.233	0.120	0.072	0.087
	Combined	Ravena Pond	West	Harvester	0.020	0.019	0.016	0.010	0.017
				No Harvester	0.022	0.021	0.018	0.011	0.019
			East	Harvester	0.021	0.020	0.016	0.010	0.017
				No Harvester	0.023	0.022	0.018	0.012	0.019
		Nassau Lake	West	Harvester	0.004	0.002	0.002	0.001	0.002
			East	Harvester	0.004	0.003	0.002	0.001	0.002
		Kinderhook Lake	West	Harvester	0.004	0.002	0.002	0.001	0.002
			East	Harvester	0.004	0.003	0.002	0.001	0.002
		Alcove Reservoir	West	Harvester	0.004	0.002	0.002	0.001	0.002
			East	Harvester	0.004	0.003	0.002	0.001	0.002
	Farm Only	-	West	Harvester	0.003	0.002	0.002	0.001	0.002
		-	East	Harvester	0.004	0.003	0.002	0.001	0.002
	Fisherman Only	Pond	-	Harvester	0.017	0.017	0.014	0.009	0.016
			-	No Harvester	0.019	0.010	0.019	0.016	0.018
		Nassau Lake	-	Harvester	0.000	0.000	0.000	0.000	0.000
		Kinderhook Lake	-	Harvester	0.000	0.000	0.000	0.000	0.000
		Alcove Reservoir	-	Harvester	0.000	0.000	0.000	0.000	0.000
	Water Ingestion Only	-	-	Harvester	0.000	0.000	0.000	0.000	0.000

Exhibit 3-2. Summary Results - Hazard Quotients for Divalent Mercury Scenarios using CTE Ingestion Rates

Ingestion Rates	Scenario Type	Water body	Farm Parcel	Harvester in Ravena Pond?	HQ Child (1-2)	HQ Child (3-5)	HQ Child (6-11)	HQ Child (12-19)	HQ Adult (20-70)
CTE Ingestion Rate	Screening	Screening	Screening	Harvester	0.085	0.057	0.041	0.026	0.033
	Combined	Ravena Pond	West	Harvester	0.008	0.008	0.006	0.004	0.007
				No Harvester	0.009	0.009	0.007	0.005	0.008
			East	Harvester	0.009	0.008	0.006	0.005	0.007
				No Harvester	0.010	0.009	0.007	0.005	0.008
		Nassau Lake	West	Harvester	0.001	0.001	0.001	0.000	0.001
			East	Harvester	0.002	0.001	0.001	0.001	0.001
		Kinderhook Lake	West	Harvester	0.001	0.001	0.001	0.000	0.001
			East	Harvester	0.002	0.001	0.001	0.001	0.001
		Alcove Reservoir	West	Harvester	0.001	0.001	0.001	0.000	0.001
			East	Harvester	0.002	0.001	0.001	0.001	0.001
	Farm Only	-	West	Harvester	0.001	0.001	0.001	0.000	0.001
		-	East	Harvester	0.002	0.001	0.001	0.001	0.001
	Fisherman Only	Pond	-	Harvester	0.007	0.007	0.006	0.004	0.006
			-	No Harvester	0.008	0.004	0.008	0.006	0.007
		Nassau Lake	-	Harvester	0.000	0.000	0.000	0.000	0.000
		Kinderhook Lake	-	Harvester	0.000	0.000	0.000	0.000	0.000
		Alcove Reservoir	-	Harvester	0.000	0.000	0.000	0.000	0.000
	Water Ingestion Only	-	-	Harvester	0.000	0.000	0.000	0.000	0.000

Exhibit 3-3. Summary Results - Hazard Quotients for Methyl Mercury Scenarios using RME Ingestion Rates

Ingestion Rates	Scenario Type	Water body	Farm Parcel	Harvester in Ravena Pond?	HQ Child (1-2)	HQ Child (3-5)	HQ Child (6-11)	HQ Child (12-19)	HQ Adult (20-70)
RME Ingestion Rate	Screening	Screening	Screening	Harvester	0.193	0.188	0.155	0.099	0.167
	Combined	Ravena Pond	West	Harvester	0.132	0.132	0.111	0.071	0.122
				No Harvester	0.199	0.199	0.168	0.108	0.184
			East	Harvester	0.132	0.132	0.111	0.071	0.122
				No Harvester	0.199	0.199	0.168	0.108	0.184
		Nassau Lake	West	Harvester	0.002	0.002	0.001	0.001	0.001
			East	Harvester	0.002	0.002	0.001	0.001	0.001
		Kinderhook Lake	West	Harvester	0.001	0.001	0.001	0.001	0.001
			East	Harvester	0.001	0.001	0.001	0.001	0.001
		Alcove Reservoir	West	Harvester	0.001	0.001	0.001	0.000	0.001
			East	Harvester	0.001	0.001	0.001	0.001	0.001
	Farm Only	-	West	Harvester	0.000	0.000	0.000	0.000	0.000
		-	East	Harvester	0.000	0.000	0.000	0.000	0.000
	Fisherman Only	Pond	-	Harvester	0.132	0.132	0.111	0.071	0.122
			-	No Harvester	0.198	0.108	0.199	0.167	0.184
		Nassau Lake	-	Harvester	0.001	0.001	0.001	0.001	0.001
		Kinderhook Lake	-	Harvester	0.001	0.001	0.001	0.001	0.001
		Alcove Reservoir	-	Harvester	0.001	0.001	0.001	0.000	0.001
	Water Ingestion Only	-	-	Harvester	0.000	0.000	0.000	0.000	0.000

Exhibit 3-4. Summary Results - Hazard Quotients for Methyl Mercury Scenarios using CTE Ingestion Rates

Ingestion Rates	Scenario Type	Water body	Farm Parcel	Harvester in Ravena Pond?	HQ Child (1-2)	HQ Child (3-5)	HQ Child (6-11)	HQ Child (12-19)	HQ Adult (20-70)
CTE Ingestion Rate	Screening	Screening	Screening	Harvester	0.079	0.078	0.060	0.043	0.068
	Combined	Ravena Pond	West	Harvester	0.056	0.056	0.044	0.031	0.049
				No Harvester	0.084	0.084	0.066	0.047	0.075
			East	Harvester	0.056	0.056	0.044	0.031	0.049
				No Harvester	0.084	0.084	0.066	0.047	0.075
		Nassau Lake	West	Harvester	0.001	0.001	0.001	0.000	0.001
			East	Harvester	0.001	0.001	0.001	0.000	0.001
		Kinderhook Lake	West	Harvester	0.001	0.001	0.000	0.000	0.000
			East	Harvester	0.001	0.001	0.000	0.000	0.001
		Alcove Reservoir	West	Harvester	0.000	0.000	0.000	0.000	0.000
			East	Harvester	0.000	0.000	0.000	0.000	0.000
	Farm Only	-	West	Harvester	0.000	0.000	0.000	0.000	0.000
		-	East	Harvester	0.000	0.000	0.000	0.000	0.000
	Fisherman Only	Pond	-	Harvester	0.056	0.056	0.044	0.031	0.049
			-	No Harvester	0.084	0.047	0.084	0.066	0.075
		Nassau Lake	-	Harvester	0.001	0.001	0.000	0.000	0.001
		Kinderhook Lake	-	Harvester	0.001	0.001	0.000	0.000	0.000
		Alcove Reservoir	-	Harvester	0.000	0.000	0.000	0.000	0.000
	Water Ingestion Only	-	-	Harvester	0.000	0.000	0.000	0.000	0.000

Exhibit 3-5. Fractional Pathway of Age-Specific Hazard Quotients for Divalent Mercury for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult
CTE Ingestion Rate, Alcove Reservoir, East Farm					
Fruits & Vegetables	57.7%	55.8%	60.7%	59.8%	64.5%
Egg, Pork, & Poultry	34.4%	34.8%	28.4%	31.4%	26.8%
Beef & Dairy	2.0%	2.8%	5.1%	3.5%	3.9%
Fish	1.9%	2.7%	2.8%	3.0%	3.4%
Soil	4.0%	3.9%	3.0%	2.2%	1.4%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Alcove Reservoir, West Farm					
Fruits & Vegetables	62.0%	60.1%	64.6%	64.0%	68.2%
Egg, Pork, & Poultry	29.5%	30.0%	24.2%	26.9%	22.7%
Beef & Dairy	1.8%	2.6%	4.6%	3.2%	3.5%
Fish	2.3%	3.2%	3.3%	3.6%	4.0%
Soil	4.4%	4.2%	3.2%	2.4%	1.5%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Kinderhook Lake, East Farm					
Fruits & Vegetables	57.1%	55.1%	59.8%	58.9%	63.4%
Egg, Pork, & Poultry	34.0%	34.3%	28.0%	31.0%	26.3%
Beef & Dairy	2.0%	2.7%	5.0%	3.5%	3.8%
Fish	2.9%	4.1%	4.2%	4.5%	5.1%
Soil	4.0%	3.8%	2.9%	2.2%	1.4%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Kinderhook Lake, West Farm					
Fruits & Vegetables	61.3%	59.1%	63.5%	62.8%	66.8%
Egg, Pork, & Poultry	29.1%	29.5%	23.8%	26.4%	22.2%
Beef & Dairy	1.8%	2.5%	4.6%	3.1%	3.4%
Fish	3.4%	4.9%	4.9%	5.3%	6.0%
Soil	4.3%	4.1%	3.1%	2.4%	1.5%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Nassau Lake, East Farm					
Fruits & Vegetables	57.6%	55.7%	60.5%	59.6%	64.3%
Egg, Pork, & Poultry	34.3%	34.7%	28.4%	31.3%	26.7%
Beef & Dairy	2.0%	2.8%	5.1%	3.5%	3.8%
Fish	2.1%	3.0%	3.1%	3.3%	3.8%
Soil	4.0%	3.8%	3.0%	2.2%	1.4%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Nassau Lake, West Farm					
Fruits & Vegetables	61.9%	59.9%	64.4%	63.7%	68.0%
Egg, Pork, & Poultry	29.4%	29.9%	24.1%	26.8%	22.6%
Beef & Dairy	1.8%	2.5%	4.6%	3.2%	3.5%
Fish	2.5%	3.6%	3.6%	3.9%	4.4%
Soil	4.4%	4.2%	3.2%	2.4%	1.5%
Water	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 3-5. Fractional Pathway of Age-Specific Hazard Quotients for Divalent Mercury for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult
CTE Ingestion Rate, Ravena Pond, East Farm					
Fruits & Vegetables	10.5%	7.5%	8.0%	7.4%	7.1%
Egg, Pork, & Poultry	6.2%	4.7%	3.8%	3.9%	3.0%
Beef & Dairy	0.4%	0.4%	0.7%	0.4%	0.4%
Fish	82.2%	86.9%	87.2%	88.0%	89.3%
Soil	0.7%	0.5%	0.4%	0.3%	0.2%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Ravena Pond, West Farm					
Fruits & Vegetables	9.7%	6.9%	7.3%	6.8%	6.5%
Egg, Pork, & Poultry	4.6%	3.5%	2.8%	2.8%	2.2%
Beef & Dairy	0.3%	0.3%	0.5%	0.3%	0.3%
Fish	84.7%	88.8%	89.0%	89.8%	90.9%
Soil	0.7%	0.5%	0.4%	0.3%	0.1%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Alcove Reservoir, East Farm					
Fruits & Vegetables	57.5%	55.0%	63.7%	63.0%	67.8%
Egg, Pork, & Poultry	26.6%	27.3%	23.3%	27.4%	22.9%
Beef & Dairy	1.9%	3.0%	5.7%	3.0%	3.3%
Fish	1.7%	2.5%	2.7%	2.8%	3.5%
Soil	12.2%	12.1%	4.6%	3.7%	2.4%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Alcove Reservoir, West Farm					
Fruits & Vegetables	60.8%	58.3%	67.1%	66.8%	71.1%
Egg, Pork, & Poultry	22.4%	23.1%	19.6%	23.2%	19.2%
Beef & Dairy	1.8%	2.7%	5.2%	2.7%	3.0%
Fish	2.0%	2.9%	3.2%	3.3%	4.1%
Soil	13.0%	12.9%	4.9%	3.9%	2.6%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Kinderhook Lake, East Farm					
Fruits & Vegetables	57.0%	54.3%	62.8%	62.1%	66.6%
Egg, Pork, & Poultry	26.4%	27.0%	23.0%	27.0%	22.5%
Beef & Dairy	1.9%	3.0%	5.6%	3.0%	3.3%
Fish	2.6%	3.8%	4.1%	4.2%	5.3%
Soil	12.1%	12.0%	4.5%	3.6%	2.4%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Kinderhook Lake, West Farm					
Fruits & Vegetables	60.2%	57.4%	66.0%	65.6%	69.6%
Egg, Pork, & Poultry	22.2%	22.8%	19.3%	22.8%	18.8%
Beef & Dairy	1.7%	2.7%	5.1%	2.7%	2.9%
Fish	3.0%	4.4%	4.8%	5.0%	6.2%
Soil	12.9%	12.7%	4.8%	3.9%	2.5%
Water	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 3-5. Fractional Pathway of Age-Specific Hazard Quotients for Divalent Mercury for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult
RME Ingestion Rate, Nassau Lake, East Farm					
Fruits & Vegetables	57.4%	54.9%	63.5%	62.9%	67.5%
Egg, Pork, & Poultry	26.6%	27.3%	23.2%	27.3%	22.8%
Beef & Dairy	1.9%	3.0%	5.7%	3.0%	3.3%
Fish	1.9%	2.8%	3.0%	3.1%	3.9%
Soil	12.2%	12.1%	4.6%	3.7%	2.4%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Nassau Lake, West Farm					
Fruits & Vegetables	60.7%	58.1%	66.9%	66.5%	70.8%
Egg, Pork, & Poultry	22.4%	23.0%	19.6%	23.1%	19.2%
Beef & Dairy	1.8%	2.7%	5.1%	2.7%	3.0%
Fish	2.2%	3.3%	3.5%	3.7%	4.6%
Soil	13.0%	12.9%	4.9%	3.9%	2.6%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Ravena Pond, East Farm					
Fruits & Vegetables	11.4%	7.9%	8.5%	8.2%	7.2%
Egg, Pork, & Poultry	5.3%	3.9%	3.1%	3.6%	2.4%
Beef & Dairy	0.4%	0.4%	0.8%	0.4%	0.4%
Fish	80.5%	85.9%	86.9%	87.4%	89.7%
Soil	2.4%	1.7%	0.6%	0.5%	0.3%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Ravena Pond, West Farm					
Fruits & Vegetables	10.6%	7.3%	7.8%	7.5%	6.6%
Egg, Pork, & Poultry	3.9%	2.9%	2.3%	2.6%	1.8%
Beef & Dairy	0.3%	0.3%	0.6%	0.3%	0.3%
Fish	83.0%	87.8%	88.7%	89.1%	91.1%
Soil	2.3%	1.6%	0.6%	0.4%	0.2%
Water	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 3-6. Fractional Pathway of Age-Specific Hazard Quotients for Methyl Mercury for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult 20-70
CTE Ingestion Rate, Alcove Reservoir, East Farm					
Fruits & Vegetables	12.5%	8.8%	8.1%	7.7%	6.5%
Egg, Pork, & Poultry	1.0%	0.7%	0.6%	0.6%	0.5%
Beef & Dairy	2.8%	3.1%	5.6%	3.7%	3.6%
Fish	0.829217	0.868195	0.853748	0.877842	0.892817
Soil	0.8%	0.5%	0.4%	0.3%	0.2%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Alcove Reservoir, West Farm					
Fruits & Vegetables	11.6%	8.2%	7.5%	7.1%	6.0%
Egg, Pork, & Poultry	0.7%	0.5%	0.4%	0.4%	0.3%
Beef & Dairy	2.1%	2.2%	4.1%	2.7%	2.6%
Fish	84.9%	88.6%	87.6%	89.5%	90.9%
Soil	0.7%	0.5%	0.4%	0.3%	0.2%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Kinderhook Lake, East Farm					
Fruits & Vegetables	8.7%	6.0%	5.5%	5.2%	4.4%
Egg, Pork, & Poultry	0.7%	0.5%	0.4%	0.4%	0.3%
Beef & Dairy	1.9%	2.1%	3.8%	2.5%	2.4%
Fish	88.2%	91.0%	90.0%	91.7%	92.8%
Soil	0.5%	0.4%	0.3%	0.2%	0.1%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Kinderhook Lake, West Farm					
Fruits & Vegetables	8.0%	5.5%	5.1%	4.8%	4.0%
Egg, Pork, & Poultry	0.5%	0.4%	0.3%	0.3%	0.2%
Beef & Dairy	1.4%	1.5%	2.8%	1.8%	1.8%
Fish	89.6%	92.2%	91.6%	92.9%	93.9%
Soil	0.5%	0.3%	0.3%	0.2%	0.1%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Nassau Lake, East Farm					
Fruits & Vegetables	7.5%	5.2%	4.8%	4.5%	3.8%
Egg, Pork, & Poultry	0.6%	0.4%	0.3%	0.4%	0.3%
Beef & Dairy	1.7%	1.8%	3.3%	2.2%	2.1%
Fish	89.7%	92.2%	91.3%	92.8%	93.7%
Soil	0.5%	0.3%	0.2%	0.2%	0.1%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Nassau Lake, West Farm					
Fruits & Vegetables	6.9%	4.8%	4.4%	4.1%	3.5%
Egg, Pork, & Poultry	0.4%	0.3%	0.2%	0.3%	0.2%
Beef & Dairy	1.2%	1.3%	2.4%	1.6%	1.5%
Fish	91.0%	93.3%	92.7%	93.9%	94.7%
Soil	0.4%	0.3%	0.2%	0.2%	0.1%
Water	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 3-6. Fractional Pathway of Age-Specific Hazard Quotients for Methyl Mercury for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult 20-70
CTE Ingestion Rate, Ravena Pond, East Farm					
Fruits & Vegetables	0.1%	0.1%	0.1%	0.1%	0.0%
Egg, Pork, & Poultry	0.0%	0.0%	0.0%	0.0%	0.0%
Beef & Dairy	0.0%	0.0%	0.0%	0.0%	0.0%
Fish	99.9%	99.9%	99.9%	99.9%	99.9%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
CTE Ingestion Rate, Ravena Pond, West Farm					
Fruits & Vegetables	0.1%	0.1%	0.1%	0.0%	0.0%
Egg, Pork, & Poultry	0.0%	0.0%	0.0%	0.0%	0.0%
Beef & Dairy	0.0%	0.0%	0.0%	0.0%	0.0%
Fish	99.9%	99.9%	99.9%	99.9%	99.9%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Alcove Reservoir, East Farm					
Fruits & Vegetables	13.0%	9.1%	8.4%	8.9%	7.1%
Egg, Pork, & Poultry	0.8%	0.6%	0.5%	0.6%	0.4%
Beef & Dairy	3.1%	3.6%	6.3%	3.3%	3.0%
Fish	80.6%	84.9%	84.2%	86.7%	89.3%
Soil	2.5%	1.8%	0.6%	0.5%	0.3%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Alcove Reservoir, West Farm					
Fruits & Vegetables	12.1%	8.4%	7.8%	8.3%	6.5%
Egg, Pork, & Poultry	0.6%	0.4%	0.3%	0.4%	0.3%
Beef & Dairy	2.2%	2.6%	4.7%	2.4%	2.2%
Fish	82.7%	86.8%	86.6%	88.5%	90.8%
Soil	2.3%	1.7%	0.6%	0.5%	0.2%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Kinderhook Lake, East Farm					
Fruits & Vegetables	9.1%	6.3%	5.8%	6.1%	4.8%
Egg, Pork, & Poultry	0.6%	0.4%	0.3%	0.4%	0.3%
Beef & Dairy	2.1%	2.5%	4.4%	2.3%	2.0%
Fish	86.5%	89.6%	89.1%	90.9%	92.7%
Soil	1.8%	1.2%	0.4%	0.3%	0.2%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Kinderhook Lake, West Farm					
Fruits & Vegetables	8.3%	5.7%	5.3%	5.6%	4.4%
Egg, Pork, & Poultry	0.4%	0.3%	0.2%	0.3%	0.2%
Beef & Dairy	1.6%	1.8%	3.2%	1.6%	1.5%
Fish	88.1%	91.0%	90.8%	92.2%	93.8%
Soil	1.6%	1.1%	0.4%	0.3%	0.2%
Water	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 3-6. Fractional Pathway of Age-Specific Hazard Quotients for Methyl Mercury for all Ravena Scenarios, with Harvester in Pond

Pathway	Child 1-2	Child 3-5	Child 6-11	Child 12-19	Adult 20-70
RME Ingestion Rate, Nassau Lake, East Farm					
Fruits & Vegetables	7.9%	5.4%	5.0%	5.3%	4.1%
Egg, Pork, & Poultry	0.5%	0.4%	0.3%	0.3%	0.2%
Beef & Dairy	1.9%	2.1%	3.8%	2.0%	1.7%
Fish	88.2%	91.0%	90.5%	92.1%	93.7%
Soil	1.5%	1.1%	0.4%	0.3%	0.2%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Nassau Lake, West Farm					
Fruits & Vegetables	7.3%	5.0%	4.6%	4.8%	3.8%
Egg, Pork, & Poultry	0.4%	0.3%	0.2%	0.2%	0.2%
Beef & Dairy	1.4%	1.6%	2.8%	1.4%	1.3%
Fish	89.6%	92.2%	92.1%	93.2%	94.6%
Soil	1.4%	1.0%	0.3%	0.3%	0.1%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Ravena Pond, East Farm					
Fruits & Vegetables	0.1%	0.1%	0.1%	0.1%	0.0%
Egg, Pork, & Poultry	0.0%	0.0%	0.0%	0.0%	0.0%
Beef & Dairy	0.0%	0.0%	0.0%	0.0%	0.0%
Fish	99.9%	99.9%	99.9%	99.9%	99.9%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%
RME Ingestion Rate, Ravena Pond, West Farm					
Fruits & Vegetables	0.1%	0.1%	0.1%	0.1%	0.0%
Egg, Pork, & Poultry	0.0%	0.0%	0.0%	0.0%	0.0%
Beef & Dairy	0.0%	0.0%	0.0%	0.0%	0.0%
Fish	99.9%	99.9%	99.9%	99.9%	99.9%
Soil	0.0%	0.0%	0.0%	0.0%	0.0%
Water	0.0%	0.0%	0.0%	0.0%	0.0%

Exhibit 3-7. Mercury Hazard Quotients in All Water Bodies Compared to Alcove Reservoir [a]

Chemical	Farm Parcel	Water Body	HQ Child 1-2	Child 1-2 Ratio of Water Body : Alcove Reservoir	HQ Adult 20-70	Adult 20-70 Ratio of Water Body : Alcove Reservoir
Divalent Mercury	West	Alcove Reservoir	0.004	-	0.002	-
		Kinderhook Lake	0.004	1.0	0.002	1.0
		Nassau Lake	0.004	1.0	0.002	1.0
		Ravena Pond	0.020	5.7	0.017	10.8
	East	Alcove Reservoir	0.004	-	0.002	-
		Kinderhook Lake	0.004	1.0	0.002	1.0
		Nassau Lake	0.004	1.0	0.002	1.0
		Ravena Pond	0.021	5.0	0.017	9.4
	None	Alcove Reservoir	0.000	-	0.000	-
		Kinderhook Lake	0.000	1.5	0.000	1.5
		Nassau Lake	0.000	0.7	0.000	1.1
		Ravena Pond	0.017	215.0	0.016	237.9
Methyl Mercury	West	Alcove Reservoir	0.001	-	0.001	-
		Kinderhook Lake	0.001	1.4	0.001	1.5
		Nassau Lake	0.002	1.2	0.001	1.7
		Ravena Pond	0.132	81.4	0.122	148.3
	East	Alcove Reservoir	0.001	-	0.001	-
		Kinderhook Lake	0.001	1.4	0.001	1.5
		Nassau Lake	0.002	1.1	0.001	1.7
		Ravena Pond	0.132	80.1	0.122	145.9
	None	Alcove Reservoir	0.001	-	0.001	-
		Kinderhook Lake	0.001	1.5	0.001	1.5
		Nassau Lake	0.001	1.2	0.001	1.8
		Ravena Pond	0.132	90.7	0.122	163.3

[a] Results include a harvester in the Ravena Pond. Results were also consistent when using the RME ingestion rate and the CTE ingestion rates.

Exhibit 3-8. Comparison of Hazard Quotients Using East Farm Parcels Versus West Farm Parcels in the Ravena Scenario

Chemical	Ingestion Rate	Scenario Type	Water body	HQ Child (1-2)
Divalent Mercury	RME	Combined	Alcove Reservoir	18%
			Kinderhook Lake	17%
			Nassau Lake	17%
			Ravena Pond	3%
		Farm Only	-	18%
	CTE	Combined	Alcove Reservoir	20%
			Kinderhook Lake	19%
			Nassau Lake	20%
			Ravena Pond	3%
		Farm Only	-	20%
Methyl Mercury	RME	Combined	Alcove Reservoir	3%
			Kinderhook Lake	2%
			Nassau Lake	2%
			Ravena Pond	0%
		Farm Only	-	15%
	CTE	Combined	Alcove Reservoir	2%
			Kinderhook Lake	2%
			Nassau Lake	1%
			Ravena Pond	0%
		Farm Only	-	16%

[a] The change in hazard quotients using each farm parcel was also compared for children of other age groups and adults, and the percent reduction in risk was found to be fairly consistent across these age groups. Results include a harvester in the Ravena Pond.

Exhibit 3-9. Comparison of Hazard Quotients for Ravena Scenario Using CTE and RME Ingestion Rates						
Chemical	**Scenario**	**Farm Parcel**	**Water body**	**RME Ingestion Rate, HQ Child 1-2**	**CTE Ingestion Rate, HQ Child 1-2**	**Ratio RME : CTE HQ**
Divalent Mercury	Screening	Screening	Screening Pond	0.344	0.085	4.1
	Combined	West	Alcove Reservoir	0.004	0.001	2.7
			Kinderhook Lake	0.004	0.001	2.7
			Nassau Lake	0.004	0.001	2.7
			Ravena Pond	0.022	0.009	2.4
		East	Alcove Reservoir	0.004	0.002	2.6
			Kinderhook Lake	0.004	0.002	2.6
			Nassau Lake	0.004	0.002	2.6
			Ravena Pond	0.023	0.010	2.4
	Farm Only	West	-	0.003	0.001	2.7
		East	-	0.004	0.002	2.7
	Fisherman Only	-	Alcove Reservoir	0.000	0.000	2.4
			Kinderhook Lake	0.000	0.000	2.4
			Nassau Lake	0.000	0.000	2.4
			Ravena Pond	0.019	0.008	2.4
	Water Ingestion Only	-	-	0.000	0.000	0.0
Methyl Mercury	Screening	Screening	Screening Pond	0.193	0.079	2.5
	Combined	West	Alcove Reservoir	0.001	0.000	2.4
			Kinderhook Lake	0.001	0.001	2.4
			Nassau Lake	0.002	0.001	2.4
			Ravena Pond	0.199	0.084	2.4
		East	Alcove Reservoir	0.001	0.000	2.4
			Kinderhook Lake	0.001	0.001	2.4
			Nassau Lake	0.002	0.001	2.4
			Ravena Pond	0.199	0.084	2.4
	Farm Only	West	-	0.000	0.000	2.8
		East	-	0.000	0.000	2.8
	Fisherman Only	-	Alcove Reservoir	0.001	0.000	2.4
			Kinderhook Lake	0.001	0.001	2.4
			Nassau Lake	0.001	0.001	2.4
			Ravena Pond	0.198	0.084	2.4
	Water Ingestion Only	-	-	0.000	0.000	2.2

Exhibit 3-10. Mercury Dermal Hazard Quotients for Water and Soil Contact

Age-Specific Hazard Quotient	Exposure Media	East Farm – Tilled, Alcove Reservoir	East Farm – Untilled, Alcove Reservoir	West Farm – Tilled, Alcove Reservoir	West Farm – Untilled, Alcove Reservoir	Screen
Child < 1 HQ	Soil	4.44E-04	6.66E-03	4.02E-04	4.77E-03	1.70E-01
	Water	3.19E-06	3.19E-06	3.19E-06	3.19E-06	1.30E-03
	Soil and Water	4.47E-04	6.67E-03	4.05E-04	4.77E-03	1.71E-01
Child 1-2 HQ	Soil	3.74E-04	5.62E-03	3.39E-04	4.03E-03	1.43E-01
	Water	2.83E-06	2.83E-06	2.83E-06	2.83E-06	1.15E-03
	Soil and Water	3.77E-04	5.62E-03	3.42E-04	4.03E-03	1.44E-01
Child 3-5 HQ	Soil	3.54E-04	5.31E-03	3.20E-04	3.80E-03	1.35E-01
	Water	2.55E-06	2.55E-06	2.55E-06	2.55E-06	1.04E-03
	Soil and Water	3.56E-04	5.31E-03	3.23E-04	3.80E-03	1.36E-01
Child 6-11	Soil	2.78E-04	4.17E-03	2.51E-04	2.98E-03	1.06E-01
	Water	2.12E-06	2.12E-06	2.12E-06	2.12E-06	8.64E-04
	Soil and Water	2.80E-04	4.17E-03	2.53E-04	2.99E-03	1.07E-01
Child 12-19 HQ	Soil	2.11E-04	3.17E-03	1.91E-04	2.27E-03	8.07E-02
	Water	1.67E-06	1.67E-06	1.67E-06	1.67E-06	6.79E-04
	Soil and Water	2.13E-04	3.17E-03	1.93E-04	2.27E-03	8.14E-02
Adult HQ	Soil	9.45E-05	1.42E-03	8.55E-05	1.02E-03	3.61E-02
	Water	9.22E-07	9.22E-07	9.22E-07	9.22E-07	3.75E-04
	Soil and Water	9.54E-05	1.42E-03	8.65E-05	1.02E-03	3.65E-02

APPENDIX J: Ecological Risk Assessment Case Study – Lafarge Ravena Portland Cement Facility

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LIST OF EXHIBITS

J-1 Introduction

Section 112 of the Clean Air Act (CAA) establishes a two-stage regulatory process for addressing emissions of hazardous air pollutants (HAPs) from stationary sources. In the first stage, section 112(d)(2) requires the U.S. Environmental Protection Agency (EPA) to develop technology-based standards for categories of industrial sources (e.g., Portland Cement manufacturing, pulp and paper mills). EPA has largely completed the initial Maximum Achievable Control Technology (MACT) standards as required under this provision. Under section 112(d)(6), EPA must review each of these technology-based standards at least every eight years and revise a standard, as necessary, "taking into account developments in practices, processes and control technologies." In the second stage, EPA is required under section 112(f)(2) to assess the health and environmental risks that remain after sources come into compliance with MACT. If additional risk reductions are necessary to protect public health with an ample margin of safety or to prevent adverse environmental effects, EPA must develop standards to address these remaining risks. This second stage of the regulatory process is known as the residual risk stage, and EPA is implementing this stage in accordance with its Risk and Technology Review (RTR) Assessment Plan (EPA 2006).

This appendix presents an ecological risk assessment (ERA) in support of the RTR analysis for the Portland Cement manufacturing industry. In particular, ERAs are presented for releases of hydrogen chloride (HCl), mercury (Hg), and dioxins (specifically, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin or 2,3,7,8-TCDD) from Portland Cement facilities in the United States. Potential ecological effects from HCl emissions include both direct damage to the foliage of plants and indirect adverse effects from the acidification of soils and surface waters. Ecological effects of concern for Hg and dioxins are similar to those for humans, because these chemicals tend to bioaccumulate in food chains and because they are highly toxic to mammals and other vertebrate wildlife.

J-1.1 Scope of this ERA

For HCl, this Appendix evaluates possible indirect ecological effects that might result if pH were reduced in soils and surface waters in the vicinity of one or more Portland Cement facilities. It describes a screening-level ranking assessment to identify which facilities are expected to pose the highest risk, if any, of indirect effects of HCl deposition on nearby sensitive ecosystems. Available data are examined for top-ranked facilities to determine if there is any evidence to date of such effects. A separate document evaluates possible direct effects of HCl in air to the foliage of plants in the vicinity of these facilities (Appendix K).

For Hg and dioxin, this Appendix presents a site-specific, refined ERA for the Portland Cement manufacturing facility with the highest releases of these chemicals, the Ravena Lefarge Portland Cement Facility near Ravena, New York. Although the selection of chemicals and the Ravena facility initially was based on a screen of Portland Cement facilities in relation to *de minimis* emission rates derived to protect human health, the same chemicals and site also are appropriate for evaluating potential ecological risks from persistent and bioaccumulative (PB) hazardous air pollutants (HAPs) emitted by this source category. The Ravena ERA evaluates whether post-MACT emissions from the facility could result in concentrations of Hg and dioxins in nearby aquatic food webs sufficient to cause adverse effects in local populations of birds and mammals that feed from these food webs.

For future RTR risk assessments, EPA is developing a systematic tiered ecological screening methodology to identify chemicals and facilities of most ecological concern.

J-1.2 Modeling of the Ravena Facility

The Ravena ERA and human health risk assessment (HHRA) (Appendix I) for Hg and dioxin were designed to use common data sources, methods, and assumptions when possible to ensure consistency and efficiency. The Ravena ERA and HHRA use the same emissions data and environmental fate and transport modeling for Hg and dioxin. Aquatic food webs that could be used to assess both human and wildlife exposures were developed for surface waters in the vicinity of the Ravena facility. Those aspects of the Ravena ERA problem formulation and methods that are shared with the Ravena HHRA are summarized briefly in this Appendix and presented in detail in the HHRA documentation (Appendix I).

J-1.3 Organization of this Appendix

The problem formulation for and methods used in the ERA are described in Section J-2 and Section J-3, respectively, of this Appendix. Section J-4 presents the results of the ERA, and Section J-5 identifies the references cited.

J-2 Problem Formulation

"Problem formulation" in an ecological risk assessment (ERA) defines one or more hypotheses regarding the potential ecological effects to be evaluated and establishes the scope and methods for analyzing risks (EPA 1998). Problem formulation for evaluation of residual risks requires several decisions, including identification of chemicals of potential concern (COPC) emitted by source category facilities. For source categories with a large number of facilities across the United States, screening or ranking facilities based on emissions and other factors, exposure pathways, and surrogate measures of risk can narrow the focus of an assessment to the facilities most likely to pose risks. These considerations hold for both human health risk assessments (HHRAs) and ERAs, even though problem formulation is a phrase first used in the context of ERAs (EPA 1998). This initial phase of a risk assessment includes identification of appropriate assessment endpoints for the COPC and the measures or models that can be used to evaluate or predict adverse changes in those endpoints. Problem formulation generally concludes with development of an analysis plan for the risk assessment.

Available resources can be put to the best use when an HHRA and an ERA are planned together, with fate and transport analyses serving double-duty where possible. Problem formulation activities for the HHRA and the ERA for the Ravena facility were conducted jointly. The HHRA for Portland Cement is described in an earlier Appendix (Appendix I). Part of the ERA for the Ravena facility was planned side-by-side with the HHRA and uses the same fate and transport modeling setup as used for the HHRA. Therefore, many aspects of ERA problem formulation, including selection of the Ravena facility and its spatial modeling layout, are described in detail in the documentation of the Ravena HHRA (Appendix I). The documentation of the Ravena ERA in this Appendix refers to relevant sections of the HHRA Appendix, but focuses on aspects of the problem formulation (e.g., ecological endpoints) and analyses that are unique to the ERA.

Problem formulation for the ERA is described below in four parts. Selection of hazardous air pollutants (HAPs) of potential ecological concern is described in Section J-2.1. Site selection for a refined ecological risk assessment for those HAPs is described in Section J-2.2. Selection of "assessment endpoints" is documented in Section J-2.3. Remaining aspects of problem formulation are described in Section J-2.4. Because the starting information, assessment endpoints, and approach to exposure and effects analysis, as well as to risk characterization,

differ for the ecological chemicals of concern, Sections J-2.1 through J-2.4 are each divided into two subsections based on COPC.

J-2.1 Selection of Ecological HAPs of Concern

The Portland Cement source category encompasses 91 facilities in the United States identified from the National Emissions Inventory (NEI), as described in the initial emissions screening analysis presented in Section 3.2 of EPA's report to SAB. An initial screen of persistent and bioaccumulative (PB-HAPs) was conducted for those facilities by comparing the facility-specific total emissions for a given PB-HAP to *de minimis* emission quantities derived for human health risk endpoints. For persistent and bioaccumulative (PB) HAPs, the *de minimis* emission quantities were estimated using a conservatively constructed screening scenario within TRIM.FaTE to estimate chemical fate and transport, including transfer through both terrestrial and aquatic food chains. At each facility, PB-HAPs for which the total emissions exceed the *de minimis* emissions quantity for that chemical (or chemical group) were selected to further analysis. The *de minimis* screening analysis is documented further in Appendix C.

Although emissions of every PB-HAP on EPA's list are not reported for every facility in this source category, over half of the facilities report emissions of mercury (Hg). In addition, based on measurements at individual facilities and knowledge of the Portland cement manufacturing process, it was assumed that every facility emits dioxins. Both Hg and dioxins are presumed to be emitted in relatively large quantities from at least some facilities in this source category. Given the potential for human exposure via non-inhalation pathways to these two PB-HAPs and the relatively high emissions of these chemicals reported for Portland cement facilities, both Hg and dioxins were expected to be chemicals of concern for the non-inhalation human health risk assessment (see Appendix I).

Overall, for the ERA we considered two categories of HAPs separately: (1) those that are sufficiently persistent and bioaccumulative to reach levels in aquatic food chains that are toxic to piscivorous wildlife at environmental concentrations (e.g., in air, soil, and water) unlikely to cause direct toxicity to any other group of organisms and (2) those that might cause direct adverse ecological effects at lower air concentrations than are of concern for human health. From the first category of chemicals, which roughly corresponds to the PB-HAPs of concern for human health, Hg and dioxins were selected as described in Section J-2.1.1 below. From the second category of chemicals, which includes hydrogen fluoride (toxic to plants), we selected HCl as described in Section J-2.1.2. Data from NEI indicated that hydrogen fluoride (HF) is emitted from only 3 of the 91 facilities; HF was therefore not included in the ERA for the Ravena facility.

J-2.1.1 Mercury and Dioxins

PB-HAPs might pose threats to ecological receptors at lower environmental concentrations than those that pose human health risks because several wildlife species feed almost exclusively on aquatic prey, while human diets generally are more diversified and, therefore, humans generally consume less fish per unit body weight than piscivorous wildlife. Based on the prevalence of HAP emission from Portland Cement facilities and the toxicity of those HAPs to wildlife, we concluded that dioxins and Hg are the PB-HAPs from this source category most likely to pose a risk to wildlife predators of aquatic organisms.

The same characteristics of dioxins and Hg that resulted in their selection for the HHRA indicate their potential for adverse effects in piscivorous wildlife. Dioxins and Hg in both its methylated (MeHg) and divalent (Hg+2) forms, are toxic to non-human mammals and other classes of

vertebrates, including birds. Wildlife that feed from aquatic food chains, particularly those that consume primarily larger fish, tend to be the components of ecosystems that are most highly exposed to bioaccumulative chemicals.

For both the HHRA and the ERA, ICF used 2,3,7,8-TCDD, the most toxic dioxin congener, to represent total dioxins. Available data also suggest that 2,3,7,8-TCDD is the most bioaccumulative of the dioxins in part because it is the least well metabolized by vertebrates and invertebrates alike and in part because it is more readily taken up than other dioxins and furans.

For Hg, in addition to total Hg, ICF considered three species of Hg – divalent (Hg+2), elemental (Hg0), and methyl mercury (MeHg) – for purposes of modeling fate and transport. The proportion of total Hg present as MeHg in top predatory fish generally is high, more than 90 percent in most studies (EPA 2001, 2009).

J-2.1.2 Hydrogen Chloride

Hydrogen chloride is the chemical released in the highest quantities each year by many Portland Cement facilities. Hydrogen chloride also is one of the few HAPs that might produce *direct* toxic effects on vegetation or other direct adverse ecological effects near facilities at concentrations lower than a reference concentration (RfC) for the protection of human health. At sufficiently high or prolonged air exposures, HCl can directly impact the structure and function of plant leaves at several levels. The derivation of short-term (1 hr) and long-term (e.g., days to weeks) air concentration benchmarks for the protection of plant communities from direct effects of HCl on leaves is documented in a separate Appendix (Appendix K). Comparison of the long-term benchmark to the RfC for HCl for humans indicates that air concentrations protective of humans also will protect plant communities from adverse effects due to chronic exposure to airborne HCl (Appendix K). The 1-hr HCl air concentration benchmark for foliar damage, on the other hand, is lower than the most conservative reference exposure concentration for the protection of human health. See Appendix K for the assessment of risk of foliar damage from direct exposure of plants to airborne HCl.

Local emissions of HCl also, however, might cause adverse ecosystem effects *indirectly* through a gradual decrease in the pH of receiving ecosystems. Lower pH in soils and surface waters can increase the bioavailability of inorganic contaminants (e.g., aluminum, mercury, selenium) and cause several types of deleterious effects at multiple levels of biological organization (Brezonik et al. 1991; Sparling 1995). A significant issue in assessing these effects is that they are mediated in large part by changes in pH in the receiving surface water and in soils. Whether pH will change in rain, soils, and surface waters near a facility in response to local emissions of HCl to air depends on several characteristics of the environment, particularly current regional levels of acid deposition from all sources, including oxides of nitrogen (NO_X) and sulfur (SO_X), and the acid buffering capacity of the receiving ecosystem. In addition, for soils, the type, depth, and slope also may influence the extent to which pH changes in response to acid deposition. Whether adverse effects are likely to occur in response to a given change in pH (range or central tendency) also depends on many environmental factors, including the pH in the absence of local HCl emissions, the occurrence of other possibly toxic chemicals in soils and surface waters that may become more bioavailable at lower pH, and the sensitivity of local organisms to acid conditions (e.g., acid-tolerant plants).

Rainfall in remote areas (away from anthropogenic sources of air pollution) generally has a slightly acidic pH of approximately 5.6, because carbon dioxide and water in the air react together to form carbonic acid, a weak acid. Anthropogenic contributions, particularly NO_X and

SO_X, to lowering pH (increasing acidity) of rainfall varies regionally, with highest acidities in the northeastern United States.

In aquatic ecosystems, different plant and animal species have different tolerances for pH of the water. Loss of sensitive species due to acidification can, depending on location, change aquatic community structure in ways deleterious to wildlife and to human welfare. In addition, changes in surface water pH can affect the toxicity of other pollutants to aquatic organisms in the system, particularly ionic inorganic chemicals and weak organic acids or bases.

In terrestrial ecosystems, soil pH affects the solubility and bioavailability of inorganic nutrient and pollutant chemicals to plants and can affect microbial community processes. Strongly acid soils can result in sufficient aluminum^{+3} activity to be toxic to plants.

This appendix documents a screening-level ERA to evaluate the likelihood that any Portland Cement facilities might cause any of the adverse indirect effects listed above through releases of HCl.

J-2.2 Site Selection

In this section, site selection for a refined ecological analysis is first described for Hg and dioxin (Section J-2.2.1) and then for HCl (Section J-2.2.2).

J-2.2.1 Site Selection for Mercury and Dioxins

The approach used to select the Ravena facility for the HHRA and ERA is documented in Appendix I. Briefly, we first identified Portland cement facilities that had high emissions for both Hg and dioxins, assuming that higher emissions of the chemicals would lead to higher human exposures. For these facilities, we looked for one that had suitable geographic characteristics for the two basic human health exposure scenarios (i.e., fisher and farmer) and for an ERA. The Ravena facility was considered appropriate for the ERA because of its proximity to pond, lake, reservoir, forest, and field habitats for wildlife. With those habitats, several piscivorous wildlife species can reasonably be expected to be present.

J-2.2.2 Site Selection for Hydrogen Chloride

As a consequence of the influence of environmental characteristics on pH change and ecological effects of pH change in response to localized HCl emissions, ICF concluded that it could not estimate a *de minimis* emission rate for HCl applicable to all facilities. A different approach to identifying facilities of concern from among the 91 facilities under consideration was needed than that used for PB-HAPs.

Using the emission data for HCl from Portland facilities across the United States compiled for use in the HHRA, ICF conducted a proximity and vulnerability screening assessment for ecologically sensitive environments, as described in Section J-3.3.

J-2.3 Selection of Assessment Endpoints

Assessment endpoints are “explicit expressions of the actual environmental value that is to be protected, operationally defined by an ecological entity and its attributes” (EPA 1988). Different assessment endpoints are appropriate for Hg and dioxins (Section J-2.3.1) and HCl (Section J-2.3.2).

J-2.3.1 For Mercury and Dioxins at Ravena Facility

The available literature indicates that piscivorous wildlife tend to be early warning indicators in ecosystems known to be contaminated with Hg and polychlorinated biphenyls (PCBs). Adverse effects, particularly reproductive and developmental impairments, associated with these chemical groups have been observed in piscivorous wildlife where other adverse ecological effects are not evident (Colborn 1991; Giesy et al. 1994; Gilbertson et al. 1991; Keith 1996; Rice and O'Keefe 1995). In addition, measurements of these chemicals in animal tissues tend to be higher for piscivorous wildlife than for other wildlife species (Sheffy and St. Amant 1982; Wren et al. 1986, Wolfe et al. 2006). The persistence, toxicity, and bioaccumulative potential for 2,3,7,8-TCDD are similar to those of many PCBs. The focus of the ERA for dioxins and Hg, therefore, was narrowed to a risk assessment primarily for piscivorous wildlife in the area surrounding the Ravena facility.

From an ecological perspective, adverse effects on piscivorous avian and mammalian populations are likely to be the most sensitive endpoint for TCDD and Hg, largely because they will be the most highly exposed organisms. Terrestrial food "chains" (e.g., plants → herbivores [such as voles, mice, and rabbits] → predators [such as hawks, owls, and canines]) tend to be "shorter" than aquatic food chains (e.g., algae or detritus → zooplankton or benthic invertebrates → small fish → larger fish → piscivorous wildlife). Moreover, the wildlife species that have been documented with the highest tissue concentrations of, and in some cases substantial observed impacts from, bioaccumulative chemicals from the environment tend to be the piscivores (e.g., mink, otter, osprey, gulls, terns, cormorants, mergansers, bald eagles) (Colborn 1991; Environment Canada 1991; Eisler 1987; Giesy et al. 1994; Gilbertson et al. 1991; Sheffy and St. Amant 1982; Rice and O'Keefe 1995; Wolfe et al. 2006). Thus, adverse effects from bioaccumulative chemicals can occur in piscivores at lower environmental concentrations than are likely to cause adverse effects on other ecological receptors.

Three piscivorous wildlife species were selected for the Ravena ERA based on their likely presence in the area, their dietary habits, and their overall body size, which affects metabolic rates and might affect effective dose relative to body weight. In addition, tree swallows were included to represent consumers of benthic invertebrates (insects) from potentially contaminiated aquatic environments around the Ravena facility.

- **Tree swallows** (*Tachycineta bicolor*) are aerial insectivores. These relatively small passerine birds (20 grams) have an energetically intensive method of foraging (catching insects on the wing), a relatively high metabolic rate and food ingestion rate relative to their body weight, and females can consume up to 100% of their body weight daily when forming eggs (clutches of 4 to 6 eggs common). For this ERA, it is assumed that 100% of the tree swallow diet consists of insects emerging from aquatic environments, and 100% of those insects lived in the benthos as nymphs. In short, tree swallows consume a diet that is equivalent to 100% benthic invertebrates.
- **Common mergansers** (Mergus merganser) are included in the ERA primarily for two reasons: (1) they can and do capture and consume larger (e.g., up to 25 to 30 cm), higher-trophic-level fish in general than do other birds that might be present near Ravena (e.g., belted kingfishers are limited to fish of 10 cm or less; great blue herons forage in shallow areas with smaller fish than those available to mergansers), and (2) their diet consists entirely of fish, unlike some other semi-piscivorous birds that might consume both terrestrial and aquatic organisms (e.g., great blue herons).
- **Bald eagles** (*Haliaeetus leucocephalus*) are large birds (approximately 4.5 kg) with a relatively low metabolic rate compared to smaller birds. Therefore, a toxicity

reference dose in mg/kg-day scaled to bald eagles from smaller experimental species on the basis of metabolic rate would be lower than for smaller bird species. Bald eagles are long-lived and can scavenge relatively large fish (and other dead animals) from surface waters as well as catch and carry large live prey under some circumstances. Finally, although no longer classified as endangered, they continue to be rare in much of their historic range.

- **American minks** (*Neovison vison*) are piscivorous and are likely to occur in the area around the Ravena facility. EPA has quantified exposure factors for mink in its1993 Wildlife Exposure Factors Handbook, although some more recent studies also are available. Mink reproductive success is sensitive to environmental contaminants, most notably PCBs and Hg. Other potentially exposed mammalian species in the area of the Ravena facility (e.g., raccoons) may take some or all of their diet from aquatic ecosystems, but are more omnivorous and variable in their diet than mink, and so are likely to be less exposed than mink.

J-2.3.2 For Hydrogen Chloride

The types of ecological effects that might be expected from indirect effects of HCl deposition have been described in Section J-2.1.2. Because those effects cannot yet readily be predicted on the basis of modeling HCl fate and transport and soil and water chemistry at a local level, evaluation of assessment endpoints will require observed evidence that adverse ecological effects consistent with increased acidification are occurring near a facility. Attribution to the facility will only be possible if there is a clear gradient of effects with increasing distance from the facility.

J-2.4 Modeling Fate and Transport

J-2.4.1 For Mercury and Dioxins at Ravena Facility

For both the ERA and the HHRA, TRIM.FaTE was used to simulate air dispersion, deposition, and transport of Hg and TCDD emissions from the Ravena facility and to predict concentrations of Hg and dioxin in fish for four water bodies in the vicinity of the facility. For Hg, three forms (Hg0, Hg+2, and MeHg) were modeled with transformations among forms simulated as appropriate in various environmental media. For the HHRA, TRIM.FaTE also was used to calculate chemical concentrations in additional exposure media (e.g., locally grown produce and animal products). The water bodies include a small pond (Ravena Pond) near the facility (located south of the facility), the Alcove Reservoir (located west of the source), and Kinderhook Lake and Nassau Lake (both located east of the source). See Appendix I for site maps and a detailed description of the spatial layout of the site, including the areas and locations of the farm and watershed parcels relative to the Ravena facility, as well as land-use patterns in the area surrounding the facility.

The ERA evaluates risks to piscivorous wildlife species that obtain prey from the four water bodies and an insectivorous bird that is assumed to obtain its prey from aquatic environments around the Ravena facility. Concentrations of TCDD and Hg attributable to the Ravena facility were not estimated for the nearby Hudson River because it is largely a flow-through system.

J-2.4.2 For Hydrogen Chloride

EC/R modeled fate and transport in air of HCl from Portland Cement facilities across the United States. See Appendix K for a description of that overall approach.

J-3 Methods

This section describes the method used for the Ravena ERA. Section J-3.1 identifies the source of HAP emissions data for the ERA. Section J-3.2 presents the ERA methods used for mercury (Hg) and dioxins (TCDD). The methods for hydrogen chloride (HCl) are presented in Section J-3.3.

J-3.1 HAP Emissions Data

Mercury and HCl emissions data for the Ravena, NY, facility were obtained or derived from the inventory compiled for the Portland cement source category for this case study (based on the 2002 National Emissions Inventory [NEI]). As discussed in the documentation of the initial emissions screening analysis (Appendix C), the Hg data were examined to confirm that default speciation was applied to emissions reported as "total Hg" or similar designations. Items entered in the NEI as "mercury" or "mercury and compounds" were divided into estimated divalent and elemental Hg emissions. This speciation was achieved using speciation factors by source category provided by EPA. For Portland Cement, 25 percent of the emissions were assumed to be divalent mercury (Hg+2) and 75 percent were assumed to be elemental mercury (Hg0).

The NEI does not include dioxin/furan emissions, so a separate analysis was conducted to estimate the dioxins/furans emissions for Portland Cement facilities (Appendix F). Clinker production data (in tons per year) were obtained for each facility. Emission factors then were applied to the clinker production data to calculate a mean and 95th percent upper confidence limit (UCL) emission rate for 2,3,7,8-TCDD equivalents (TEQs). Both the mean and 95th percent UCL emission estimates were used in the Ravena ERA.

J-3.2 Mercury and Dioxins

J-3.2.1 TRIM.FaTE Aquatic Ecosystem Modeling

Part of the site-specific HHRA assesses human exposures via aquatic food chain contamination, considering both bottom-feeding fish that might be consumed by humans and game fish, in general the top predators in aquatic ecosystems. The same aquatic food webs developed in TRIM.FaTE for the HHRA are appropriate for use in estimating dose to the wildlife species chosen as assessment endpoints.

Aquatic food webs in TRIM.FaTE were developed to predict bioaccumulation of chemicals in a small "farm" pond near the Ravena facility (hereafter called Ravena Pond), Alcove Reservoir, Kinderhook Lake, and Nassau Lake. There are nine groups of aquatic organisms included in TRIM.FaTE for one or more of these water bodies:

1. **Plankton** includes both algae and zooplankton modeled as a "phase" of the water column;
2. **Macrophytes** which can accumulate and "sequester" some chemicals (modeled as a separate compartment);
3. **Benthic invertebrates** such as mollusks, crustacea, and aquatic insect nymphs that consume periphyton and detritus (modeled as a compartment in chemical equilibrium with bottom sediments);
4. **Benthivorous fish** which are bottom-feeding fish (e.g., bullhead catfish) that consume primarily benthic invertebrates;

5. **Bottom-feeding carnivores** (i.e., eels) that consume both benthic invertebrates and fish;
6. **Water column planktivores,** such as young-of-the-year for many species and other small fish (e.g., shiners) that consume primarily planktonic organisms;
7. **Water column omnivores,** which are larger fish that consume invertebrates and smaller fish from both the benthic and pelagic environments (e.g., "panfish" like bluegill, yellow perch, and young age classes of the game species);
8. **Water column piscivores,** which are larger size game fish species that primarily consume smaller fish in pelagic and/or benthic environments (e.g., walleye, largemouth bass); and
9. **Mallard ducks,** which consume aquatic insects, invertebrates, and vegetation (ducks included as prey for bald eagles as discussed in Section J.4.1.1.1).

The parameterization of these compartments (with the exception of mallards) is described in Appendix I, Attachment I-1. Briefly, for Kinderhook and Nassau Lakes, data from several fish surveys conducted by the New York State Department of Environmental Conservation (NYS DEC) between 1988 and 2006 were used to estimate the relative abundance of different fish species in each lake. Data on fish species presence and fish weights in the Alcove Reservoir were obtained from the NYS DEC fish surveys conducted between 1963 and 1970, after which surveys ceased and the Reservoir was closed to public uses. The same fish weight data were used for the other water bodies. The proportion of total fish biomass for each water body contributed by each species was assigned to one of five fish compartments (numbers 4 through 8 above) on the basis of descriptions of their feeding habits available from online fishing communities and from NYS DEC online documents. The food web for the small "farm" pond was derived from an analysis of data presented by Demers et al. (2001) for two small lakes in Ontario.[1]

J-3.2.2 Exposure Assessment

Assumptions about the composition of each species' diet were developed based on published field observations and methods outlined in EPA's Wildlife Exposure Factors Handbook (EPA 1993 WEFH). These assumptions are expressed as percentages of the total diet obtained from the TRIM.FaTE food web compartments described earlier.

A weight-of-evidence approach was used to develop appropriate ingestion rates for each food type. Measurements of fish ingestion by captive animals, if available, were compared to estimates of ingestion rates of free-living animals based on measured or allometric predictions of average metabolic rates for free-living animals, a central tendency estimate of the gross energy content of the food type, and the assimilation efficiency of the food type. Where ingestion estimates differed from measurements, the methods used in primary studies were reviewed and an ingestion value was selected to best represent an annual average daily ingestion rate for free-living adult animals.

Mercury and dioxin intakes (exposure doses) for the five potential wildlife receptors were calculated from concentrations of Hg and TCDD in the TRIM.FaTE aquatic food web

[1] To provide the most accurate predictions possible with TRIM.FaTE, it is important to account for all of the plant and fish biomass that might accumulate chemicals in each water body. The latter requires assigning measured fish biomass densities for all fish species to the smaller number of fish compartments that are used in TRIM.FaTE to represent different "trophic" groups of fish (e.g., species/sizes of fish that feed on the same general type/size of foods in the same general environments – benthic and pelagic). For Ravena Pond, the fish harvest rate by humans and wildlife would reduce concentrations in fish, as discussed in Section J-4.1.4.

compartments (i.e., wildlife food types) described above. For each wildlife species, the chemical concentration in each food type was multiplied by the average daily ingestion rate for that food type. The resulting daily chemical intake rates for each food type were summed across food types, and the total chemical intake was normalized to adult body weight to estimate exposure dose (i.e., micrograms chemical ingested per gram of body weight per day, µg/g-day, equivalent to milligrams chemical per kilogram body weight per day, mg/kg-day).

J-3.2.3 Ecological Effects Assessment

Several sources were reviewed for wildlife toxicity reference values (TRVs) for Hg (expressed either as total Hg or MeHg) and 2,3,7,8-TCDD; however, only one source proved to be useful for the Ravena ERA:

- EPA's 1995 Great Lakes Water Quality Initiative (GLWQI) Criteria Documents for the Protection of Wildlife: DDT; Mercury; 2,3,7,8-TCDD; PCBs (adequate documentation of TRVs expressed as chemical dose);
- EPA's 2005 TRIM Ecotox Database (inadequate documentation – did not describe quality of full database for each chemical; original toxicity values not included; dosimetric scaling between experimental and wildlife species performed using body weights that were not reported) (http://www.epa.gov/ttn/fera/data/trim/ecotoxdatabaseDoc-Nov152005);
- EPA's 2005 Ecological Soil Screening Levels (http://www.epa.gov/ecotox/ecossl/) (no values for Hg or TCDD);
- EPA's Region 9 Biological Technical Assistance Group (BTAG) Recommended Toxicity Reference Values for Mammals/ for Birds (last revised 11/21/2002) (no values for TCDD; Hg values based on EPA 1995 above);
- California Office of Environmental Health Hazard Assessment Ecotox Database, developed in collaboration with the University of California at Davis (http://www.oehha.org/cal_ecotox/) (no values for TCDD or MeHg);
- The Risk Assessment Information System (RAIS) sponsored by the US Department of Energy (DOE) Office of Environmental Management, Oak Ridge Operations (ORO) Office, Ecological Benchmarks (http://rais.ornl.gov/homepage/benchmark.shtml), provides access to a large number of ecological benchmarks (expressed as concentrations in soil, water, sediments, or biota), developed by numerous state and federal agencies (no values expressed as doses or chemical intake rates for wildlife); and
- US DOD's Wildlife Toxicity Assessment Program (http://chppm-www.apgea.army.mil/erawg/tox/) (did not include Hg or TCDD).

We concluded that only the EPA 1995 GLWQI documents for Hg and 2,3,7,8-TCDD, with modifications specified in EPA's 1997 Mercury Study Report to Congress (MeRTC), were adequate for establishing and documenting TRVs, expressed as doses (chemical intake in mg/kg-day) for wildlife in the Ravena ERA. The GLWQI documents represent the only source that documented the available toxicity data at the time, why a study was selected as the critical study upon which to base a reference dose, and which uncertainty factors (UFs) were needed and what their values should be given specific limitations of the available database and the critical study.

Concerns have been expressed that EPA's 1995 wildlife criteria may be overly conservative. These concerns, however, center on the food chain model for the Great Lakes (e.g., Wolfe and Norman 1998) from which the criteria expressed as water concentrations were back-calculated.

Food chains modeled on data form the Great Lakes might predict more bioaccumulation than is likely in smaller ecosystems with shorter food chains overall. The original critical toxicity studies and uncertainty factors applied to those studies have been used widely by other state and federal agencies. For the Ravena ERA, ICF is not using the Great Lakes food chain model; instead it is using food webs developed specifically for the water bodies near the Ravena facility.

Given the date of publication of the GLWQI wildlife criteria, ICF conducted a literature search using keywords in online biobliographic databases for more recent information on the toxicity of Hg and TCDD to wildlife. We found that a large proportion of recent wildlife ecotoxicity studies have focused on Hg (few on TCDD) and on correlating wildlife tissue concentrations (including chemical concentrations in mammalian fur and in bird feathers and eggs, as well as chemical concentrations in dead or sacrificed animal liver, kidney, brain, muscle, and fat) with adverse reproductive outcomes in field situations (e.g., Barr 1998, de Sorbo and Evers 2005, Evers et al. 2004, Evers and Reaman 1998, Heath and Frederick 2005, Hoffman et al. 1996, Mierle et al. 2000, Thompson 1996, Wolfe and Norman 1998, Wolfe et al. 1998). These studies are intended to determine the utility of various wildlife species as monitors of environmental pollution or to generate exposure-response relationships at the population level in the field. The measures of exposure, tissue-specific chemical concentrations, in general were only weakly associated with measures of chemical concentrations in potential prey species, limiting the utility of the studies for relating chemical intake (dose) to effect levels.

ICF considered the option of using toxicity reference values for mammals and birds expressed as tissue concentrations to compare to TRIM.FaTE-estimated tissue concentrations in wildlife. We decided against that approach for several reasons.

- There are as yet no consensus TRVs based on wildlife tissue concentrations at a federal level for Hg or 2,3,7,8-TCDD.
- One would need to determine which tissue concentrations would be most appropriate for establishing a TRV; some effects data are related to kidney and liver concentrations, which tend to be high, while other effects data are related to target tissue concentrations (e.g., Hg in brain tissues), while still other effects data are related to blood concentrations, which can be collected from wildlife without their sacrifice. For birds, chemical concentrations in eggs often are related with egg shell thinning, breakage, or hatching success.
- For a risk assessment that starts with emissions of chemicals to air, it would be necessary to predict not only the uptake of chemicals from the environment, but also the distribution of chemical to different organs in the bodies of birds and mammals. That would require the addition of PBPK models for Hg and 2,3,7,8-TCDD in birds and mammals.

Finally, for Hg and 2,3,7,8-TCDD, ICF used EPA's approach of using an inter-species uncertainty factor of only 3 to account for unknown toxicokinetic and toxicodynamic differences among different species of bird and mammal. The most often used alternative is to scale dose between species on the basis of relative metabolic rate (body weight raised to approximately the 0.75 power). For bioaccumulative chemicals that accumulate over time in specific tissues, however, metabolic rate may have only a minor influence on accumulated chemical residues in tissues.

J-3.2.4 Risk Characterization

A two-stage approach is used to characterize ecological risks from Hg and dioxin emissions from the Ravena facility. In the first phase, hazard quotients (HQs) (exposure doses divided by TRVs) are calculated by chemical for each of the five wildlife species and four water bodies. HQs exceeding 1.0 indicate a potential for individual animals to be adversely affected by their exposure.

For those scenarios where HQ values exceed 1.0, a preliminary evaluation of the potential for population-level effects is conducted. Specifically, the maximum number of individuals of a species for which the HQ exceeds 1.0 is estimated based on available data on population densities or territory size as reported in available literature. If the maximum number of individuals with an HQ greater than 1.0 is one or two, as could be the case for the small Ravena Pond, then the threat of population-level effects would be considered to be negligible. However, if the estimated HQ exceeds 1.0 for multiple individuals, we could not exclude the possibility of population-level risks without further analysis..

J-3.3 Hydrogen Chloride

ICF conducted a proximity and vulnerability screening assessment for possible indirect effects of HCl deposition on ecologically sensitive environments. Portland Cement facilities were ranked according to emission rates, the pH of regional rainfall, surface water alkalinity, and proximity to sensitive environments, as described below.

J-3.3.1 Facility Ranking

ICF conducted an initial ranking of all Portland Cement Facilities emitting HCl according to three indicators of ecological risk: (1) “background” acid deposition (regional pH of rainfall) in the area surrounding each facility, (2) surface water alkalinity (an indicator of acid buffering capacity or resistance to changing pH), and (3) annual HCl emissions reported by each facility. The background acid deposition and the surface water alkalinity were used as indicators of ecosystem susceptibility to additional acid deposition. Annual HCl emissions were used as an indicator of potential additional acid deposition due to Portland Cement facilities.

Regional pH of rainfall (i.e., background acid deposition) is one indicator of ecosystem “susceptibility”. The pH of rainfall shows regional patterns across the United States that have resulted from multiple point and non-point sources of chemical precursors of acid rain that change slowly over time. Areas subject to rainfall of relatively low pH already may be under stress from acid deposition. At a minimum, the buffering capacity of ecosystems in areas of highly acidic rainfall is likely to have been lowered from “natural” levels for the area.

ATTACHMENT J-1 Exhibit 1 provides a map of the pH of rainfall across the United States as measured by the National Atmospheric Deposition Program, National Trends Network.

A second indicator of ecosystem susceptibility is its ability to buffer acid deposition as indicated by measurements of surface water alkalinity in an area. Water alkalinity, which can be expressed as mg of calcium carbonate and magnesium bicarbonate per liter (mg/L or ppm by

weight) or simply milliequivalents of carbonate per liter (meq/L), is an indicator of the water's ability to absorb hydrogen ions (H+) without changing pH. The carbonate and hydrogen ions react to produce carbonic acid and then water and carbon dioxide. The higher the alkalinity, the more acid must be added for a noticeable reduction in pH to occur. The alkalinity of surface waters is due in large part to geological characteristics of the area, including the type of parent materials that weather to soils and sediments. Thus, alkalinity can be considered an indicator of the ecosystem sensitivity to acid deposition.

ATTACHMENT J-1 Exhibit 2 provides a copy of EPA's map of surface water alkalinity across the United States. We use this map as an indicator of both surface water and soil alkalinity as influenced by parent geological materials. Other factors can influence alkalinity as well (e.g., soil type and grain); however, for a national-scale screen, we consider this map adequate to identify areas of likely low acid buffering capacity in surface waters and in soils.

To quantify a facility-specific indicator of exposure, ICF used NEI data on emission rates (in tons per year) of HCl for all Portland Cement facilities.

The Portland Cement facilities were ranked according to the *product* of scores assigned to the susceptibility and emission factors as described below. Each factor was scored on a scale of 1 to 5.

- *Potential ecosystem susceptibility – background acid deposition*. Background acid deposition was assigned a score based on the measured pH of rainfall (log scale maintained) using the map in
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- **ATTACHMENT** J-1 Exhibit 1. Considering the range of pH of rainfall across the United States, we used a scoring range of 1 to 5, with a score of 1 representing a pH of ≤ 4.5, a score of 2 representing a pH of > 4.5 and ≤ 4.7, a score of 3 representing a pH of > 4.7 and ≤ 4.9, a score of 4 representing a pH of > 4.9 and ≤ 5.1, and a score of 5 representing a pH of > 5.1.
- *Potential ecosystem susceptibility – acid buffering capacity*. EPA maps of surface water total alkalinity (meq/L) are based on five alkalinity categories. We assigned scores based on those categories: a score of 1 for areas with total alkalinity less than 50 meq/L; 2 for alkalinity of 50 to 100 meq/L; 3 for alkalinity of 100 to 200; 4 for alkalinity of 200 to 400; and 5 for total alkalinity of surface waters greater than 400 meq/L.
- *Facility emissions rate*. Facility HCl emissions were ranked from 1 to 5 based on the estimated HCl emission rates, with facilities in the top 20th percentile receiving a score of 1 and those in the lowest 20th percentile receiving a score of 5.

Equation J-1 shows the calculation of a preliminary hazard ranking score for each facility. Based on the individual factor scoring system above, the lower the hazard score, the higher the possible ecological risks of adverse indirect effects of HCl deposition.

Equation J-1:

$$\textit{Preliminary Hazard Score}_{\textit{facility n}} = \textit{Rainfall pH}_{\textit{facility n}} \times \textit{Surface Water Alkalinity}_{\textit{facility n}} \times \textit{Emissions}_{\textit{facility n}}$$

J-3.3.2 Refined Facility Ranking

The next step to refine the ecological risk ranking for the facilities was to identify nearby sensitive environments on maps of the area surrounding each facility and to score facilities on the basis of distance to the sensitive environment. This step not only identified ecosystems of potential concern, but it also allowed quantification of a proximity factor, another indicator of the potential for exposure because of dispersal of contaminants in air with increasing distance from an emission source.

Sensitive environments, as defined in EPA's Hazard Ranking System for potential Superfund sites, may include areas such as wildlife refuges, national parks, waterfowl staging areas, water bodies, and wetlands larger than 5 acres (EPA 1992). Because this step requires substantially more effort than quantifying and scoring the susceptibility and emission factors, we intended to conduct this step only for the ten facilities with the lowest preliminary hazard score (i.e., highest preliminary risk ranking). Because several facilities received the same score, we used a criterion of a preliminary hazard score of 20 or less to identify facilities for the sensitive environment proximity assessment (total of thirteen facilities).

The distances between the thirteen facilities with a hazard score of 20 or less and sensitive environments were estimated using GIS maps with data layers for sensitive environments provided by Environmental Systems Research Institute, Inc. (ESRI) (ESRI 2006). Sensitive environments included in the data layers are water bodies (e.g., canals, glaciers, lakes, reservoirs, streams, swamps, and marshes); national, state, and local forests; and national, state, and local parks. For this analysis, we first identified the nearest sensitive environment of any type. Finding that about half of the environments identified in this way were extremely large bodies of water (e.g., the Mississippi River, Lake Michigan) for which localized emissions of HCl from Portland Cement facilities are not likely to affect pH, we reviewed those environments again to find smaller bodies of water on which localized acid deposition might have an effect. For consistency, we identified the nearest "small" water bodies for all thirteen facilities.

Using a measuring tool in MapWindow for GIS maps, ICF measured the shortest distance between each facility and the shore of the closest water body (excluding the Great Lakes and large rivers). For three facilities, the distance to a nearby terrestrial sensitive environment (state and national forests and a state park) also was measured. Finally, we assigned a proximity score to each facility. The proximity score was equal to the square root of the distance between the facility and the sensitive environment. Although we have not seen a precedent for this, we selected the square root function to quantify this indicator on the basis of a simple conceptual model of primarily horizontal chemical dispersion in all compass point directions with increasing distance from a source. The score was rounded to one significant digit, and stopped at a top score of 5. For example, the proximity factor for a separation of 4 km would be assigned a score of 2; a separation of 10 km (square root = 3.2) would correspond to a score of 3; and a separation of 23 km (square root of 4.8) would receive a score of 5, as would any separation greater than 25 km.

ICF calculated a final composite hazard score for each of the thirteen facilities by multiplying the susceptibility, emissions, and proximity scores (see Equation J-2). The final scores were ranked to determine the facilities most likely to pose ecological risks, if any.

Equation J-2:

$$\textit{Final Hazard Score}_{\textit{facility n}} = \textit{Rainfall pH}_{\textit{facility n}} \times \textit{Surface Water Alkalinity}_{\textit{facility n}} \times \textit{Emissions}_{\textit{facility n}} \times \textit{Proximity}_{\textit{facility n}}$$

J-3.3.3 Exposure Assessment - Site-specific Data

For the HCl exposure assessment, the indirect effects of concern are mediated through changes in soil or surface water pH. Predictions of the rate of HCl deposition required to produce pH changes would require substantial site-specific information and model development. ICF therefore considered existing measurements of soil and surface water pH values as an indicator of the possibility of indirect adverse ecological effects resulting from acidification. Note, however, that such measurements serve only as another screening tier. They *do not* indicate the relative contribution of a Portland Cement facility and "background" regional acid deposition to the measured pH values. An analysis of relative source contributions would be warranted only if the screening criteria suggested non-negligible effects consistent with acidification in the vicinity of a Portland Cement facility.

Originally, ICF intended to focus on only one or two facilities with the lowest composite hazard score (highest likelihood of ecological effects) to search for local measurements of pH in soils or surface waters to compare with pH ecotoxicity benchmarks or for reports of acidification or adverse ecological effects in the vicinity of the facility. The types of nearby sensitive environments, however, vary substantially for the thirteen facilities with the lowest composite score (highest hazard). We therefore looked for localized data for all thirteen locations.

J-3.3.4 Terrestrial Environments

For analysis of terrestrial environments, we defined "areas of interest" as the nearest boundary of the sensitive environment to the respective Portland Cement facility. Each area of interest was less than 60 acres. Measurements of soil pH and other parameters for the upper soil layers in ecologically valued and protected areas (e.g., state parks) close to three Portland Cement facilities were obtained from the U.S. Department of Agriculture's Web Soil Survey (USDA 2008). The Web Soil Survey provides data from the National Cooperative Soil Survey, which is a partnership of federal, regional, state, local and private agencies that provides information about soils across the United States. Most data were collected over the past 40 years, and approximately 75 percent of the data are less than twenty years old. Soil data are available for more than 95 percent of the counties in the United States. We found, however, that the range of pH values reported for a single soil layer and soil type was sufficiently high (at least 1 or 1.5 pH unit, and often more) that it is unlikely to be predictive of plant community responses. This limitation is not surprising given that pH measurements are sensitive to humidity, temperature, and other parameters that can vary seasonally and daily. We therefore also considered the cation-exchange capacity measured for the surface soil layers in the area of interest as a more precise indicator of local soil conditions. The higher the cation-exchange capacity of a soil, the higher the soil buffering capacity, and the more resistant the soil is to changes in pH with acid deposition.

A final line of evidence for the possibility of indirect ecological effects of HCl emissions from a Portland Cement facility would be reports of adverse ecological effects in the vicinity of a facility that are consistent with effects of soil or surface water acidification.[2] For example, evidence of

[2] The existing facilities have been emitting HCl against a background rate of acid deposition for many years. Confirmation of ecological risks by reported observations of existing ecological impacts might, therefore, be

a gradient of aluminum toxicity to plants with distance from a facility would be consistent with excessive acidification of soils possibly due to HCl emitted by the facility. We searched state departments of natural resources and other Internet sites for possible reports of adverse ecological effects that might be related to acid deposition. In the absence of site-specific data, we examined aerial photographs of the area surrounding a facility to determine if there was any evidence visible in the photographs of adverse effects on vegetation in the vicinity of a facility.

For nearby surface waters, we searched for pH measurements in EPA's STORET Database (EPA 2008). STORET is an operational data repository that is updated continuously with water data for all states, territories, and jurisdictions in the United States. STORET contains water quality data in addition to biological and physical data. When water quality data were not available in STORET, ICF sought alternative data sources that are described in Section J-4.2.

J-3.3.5 Ecological Effects Assessment

To evaluate potential aquatic ecological effects associated with measured surface water pH, we relied on EPA's documentation of its criteria for pH for freshwaters of the United States (EPA 1986).

To evaluate potential terrestrial ecological effects associated with measured soil pH values, we considered recommendations for soil pH for maximizing plant growth, including consideration of the moderately acid-tolerant native and agricultural plant species. Use of this type of benchmark for soil pH assumes that the concentrations of other potentially toxic heavy metals (e.g., aluminum) are not above the range of "background" levels that characterize most of the United States.

To evaluate potential terrestrial community effects of measured cation-exchange capacity, we used the U.S. Department of Agriculture's (USDA) classification of cation-exchange capacity.

J-3.3.6 Ecological Risk Characterization

Where local measurements of surface water or soil pH are available, they are compared to EPA's ambient water quality criteria (AWQC) for pH for the protection of aquatic life (Section J-4.2.1.1) or the soil pH benchmark (Section J-4.2.1.2). Where the local surface water or soil pH measurements are below the ecotoxicity benchmarks or where there are observations of adverse ecological effects in the vicinity of a facility, further investigation might be warranted.

We consider surface waters with measured pH values below the AWQC criterion of 6.5 to be at risk of reduced biodiversity due to the loss of acid-sensitive species. We consider soils with pH levels below 5.5 to possibly be at risk of reduced plant biodiversity (i.e., species restricted to the more acid-tolerant groups), which might affect plant community structure. As described in Section J-4.2.1.2, we consider soils with pH values above 6.0 at negligible risk of indirect adverse effects from existing acid deposition. In between pH 5.5 and 6.0, additional lines of evidence are needed before one would conclude that some level of adverse effect is possible.

As noted in Section J-3.3.3 above, pH values below (more acidic than) the pH benchmarks or observations of adverse effects consistent with acidification in the vicinity of a Portland Cement facility do not indicate whether or to what extent the effects are attributable to releases of HCl from the facility compared to regional acid deposition. Attribution of effects to a facility would

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possible. In particular, any adverse effects that decrease with increasing distance from the facility and that are consistent with effects associated with acidification might be due to HCl emissions from the facility.

require additional lines of evidence, such as a gradient of decreasing adverse effects on plants with increasing distance from the facility. Exposure-response evidence of this type would not necessarily identify HCl alone as the chemical causing affects (it could be HCl in combination with some heavy metals released in lower quantities), but it would strongly suggest the facility as the source. A relative-source-attribution analysis is warranted only if the screening data indicate that adverse ecological effects might occur or be occurring in the vicinity of a facility.

J-4 Results

Detailed results are presented separately for mercury (Hg) and dioxins (2,3,7,8-TCDD) (Section J-4.1) and hydrogen chloride (HCl) (Section J-4.2).

J-4.1 Results for Mercury and Dioxins

This section includes the results of the exposure assessment (Section J-4.1.1), the dose-response analysis for Hg and 2,3,7,8-TCDD (Section J-4.1.2), and risk characterization (Section J-4.1.3). Key data and model limitations and uncertainties of the ecological risk assessment (ERA) for Hg and TCDD are discussed in Section J-4.1.4.

J-4.1.1 Exposure Assessment

An exposure assessment was performed for "individuals" of each of the four wildlife species of concern to calculate daily doses of Hg and TCDD from ingestion of prey obtained from surface water bodies near the Ravena facility. Exposure doses were calculated for each wildlife species and each water body modeled for the Ravena facility assuming that an individual animal obtained all of its food from the single water body. This required a characterization of each species' mean body weight, an assumed diet for the TRIM.FaTE modeled food web compartments, and food ingestion rates.

TRIM.FaTE estimates of concentrations of Hg and 2,3,7,8-TCDD in each prey type were multiplied by daily prey ingestion rates to estimate the daily intake for each chemical for each wildlife species and water body (i.e., Ravena Pond near the facility, Alcove Reservoir, Kinderhook Lake, and Nassau Lake). The chemical intakes (doses) were normalized to body weight (i.e., mg chemical ingested per kg animal body weight per day). Exposure estimates for individuals of the same wildlife species varied by location because of differences in several factors across the water bodies (described in Appendix I), including:

- water and sediment chemical concentrations estimated by TRIM.FaTE;
- the food webs constructed for each water body (food chains were shorter and fewer fish compartments were included in the small pond relative to the three large water bodies based on our experience in parameterizing aquatic food webs for use in TRIM.FaTE for case studies in Maine, New York, and the RTR screening scenario);
- total biomass of fish assumed for each water body relative to the volume of water in the system; and
- the distribution of fish biomass across the fish compartments as estimated from local fish surveys of all water bodies except the small pond.

Construction of the aquatic food webs was described briefly in Section J-3.2.1 and in detail in Appendix I. The exposure assumptions used for each of the avian and mammalian wildlife species are described below.

J-4.1.1.1 Exposure Assumptions

For each wildlife species, we identified values for three exposure factors: (a) adult body weight, (b) the percentage of total diet obtained from the food types included in the TRIM.FaTE model, and (c) an estimated daily ingestion rate for each consumed food type. For (a) adult body weights, we used *mean* values reported in the literature for populations closest to New York.

For (b), we used reviews of the dietary habits of wildlife species recently prepared by ICF for EPA's Office of Water, but not yet published. For these data, we cite original sources here rather than the secondary Office of Water documents. We emphasize that prey selection and dietary habits of different wildlife species vary with location, time of year, habitat, relative abundance of different prey species, breeding status of both predators and prey, and other factors. Nonetheless, some attributes of diet composition are fairly common for some of the more specialized predators, including swallows and piscivorous wildlife, as discussed for each species below.

One of the most important considerations in modeling bioaccumulation of chemicals through food chains is the size of fish consumed, which loosely corresponds to trophic level depending on the species of fish and their feeding habits. There generally are limits to the size of fish that can be captured and swallowed by avian wildlife that swallow their prey whole (e.g., merganser, swallows). For wildlife that can consume larger fish by tearing pieces off while standing on land (e.g., mink, eagle), the distribution of fish sizes consumed depends on fish availability and population age/size class structure, the size of fish in habitats fished by the wildlife, and in the agility of the fish in escaping capture compared with the abilities of the wildlife species. We therefore evaluated available data on the size of fish consumed. For species that consume both aquatic and terrestrial prey (e.g., mink, bald eagle) in many locations, we conservatively assumed 100 percent consumption of aquatic prey.

For (c) we intended the food ingestion rates to represent an *annual average* ingestion rate for a free-living animal rather than a breeding-season-only ingestion rate, even though the bird species are likely to migrate away from the site during the winter (particularly the swallow). We selected the measurement or estimate of food ingestion rates that we judged most likely to represent a free-living metabolic rate (FMR) averaged across all seasons.

The exposure assumptions for the four wildlife species evaluated for the Ravena facility site-specific ecological risk assessment are described below.

Tree Swallow

Body weight. The mean weight of 82 birds of both sexes captured at the Powdermill Nature Center in Pennsylvania during spring migration was 20.1 ± 1.58 grams (g), with a range of 15.6 to 25.4 g (Dunning 1984, 1993, citing unpublished data by the PNC). The mean body weight for 12 birds was 21.6 ± 1.9 g (Williams 1988). The first mean value (i.e., 20.1 g) is used to represent tree swallows throughout the year.

Diet composition. As aerial insectivores, tree swallows consume virtually 100 percent small flying insects, including adult midges, mosquitoes, mayflies, and other groups with aquatic larval forms (Quinney and Ankney 1985). For this ERA, we assumed that 100 percent of the insects consumed by swallows had been aquatic nymphs in the water body under consideration.

Food ingestion rate. Using doubly labeled water to study free-living (field) metabolic rates (FMRs) in tree swallows in New Brunswick Canada, Williams (1988) found that incubating

females required 118.9 ± 9.3 kiloJoules per day (kJ/d) (mean ± SD; n=9; average body weight [BW] 22.6 g), or 1.3 kcal/g BW-d. Females feeding young exhibited higher energy requirements and lower body weights: 128.3 ± 21.3 kJ/d for females with three young (n=5; average adult female body weight 18.8 ± 2.0 g) and 136.4 ± 15.6 kJ/d for females with five young (n=11; average adult female body weight 19.4 ± 1.2 g). Those daily energy expenditures equal 1.6 and 1.7 kcal/g BW-d, respectively. Williams (1988) noted that the FMR for aerial-feeding insectivorous passerines, such as swallows, is higher than the FMR for ground- or tree-feeding insectivorous passerine birds, such as sparrows, of similar size. We estimate from their data and discussion that the FMR for 20 g swallows is perhaps as much as 33 percent higher than for non-aerial feeding passerines of similar size.

To estimate an FMR more in keeping with a year-round food ingestion rate for chronic exposures, we used Nagy et al.'s (1999) allometric equation for passerine birds [FMR (kJ/d) = 10.4 * $BW(g)^{0.64}$ = 79.8 kJ/d for tree swallows weighing 20 g]. Note that measured FMRs (using doubly labeled water) for barn swallows (95.8 kJ/d for 20.4 g bird) and house martins (79.8 kJ/day for 19 g bird) cited by Nagy et al. (1999) are similar to the allometric estimate for tree swallows. An FMR of 79.8 kJ/d equals 19.1 kcal/d, or 0.96 kcal/g BW-d.

The FMR estimated using the allometric equation for passerine birds from Nagy et al. (1999) is about 26 percent less than the mean value of 1.3 kcal/g BW-d measured for incubating females (males take over incubation for short periods to allow the females to feed) and 44 percent less than the mean value of 1.7 kcal/g BW-d measured for females feeding five nestlings (Williams 1988). Those observations are consistent with Williams estimate that swallows require approximately a third more calories per day to forage for food than do ground- or tree-foraging insectivorous passerines. The weather during the field study in New Brunswick was cool and moist, possibly requiring more energy for thermoregulation that would be required in New York. Nonetheless, to be conservative, we use the measured FMRs instead of the allometric-model estimate of FMR. We judge that the FMR during incubation is likely to be somewhat lower than an annual average and that the FMR when feeding a clutch of 5 young is probably substantially higher than an annual average energy requirement (not considering migration). We therefore use an FMR of 1.4 kcal/g BW-d as the energetic requirement for tree swallows in this ERA.

To estimate an insect ingestion rate on a wet-weight basis, ICF used the procedure recommended in EPA's (1993) *Wildlife Exposure Factors Handbook*. Our estimate of a wet-weight insect ingestion rate for tree swallows of 1.33 g/g BW-day is based on the following assumptions:

- tree swallow body weight - 20.1 g/bird (Dunning 1984, 1993);
- energetic requirement is 1.4 kcal/g BW-day (see discussion above);
- gross energy (GE) content of insects - 22.09 kJ/g dry weight (5.28 kcal/g dry weight) (Bell 1990);
- water content of insects - 72.5 percent (midpoint of range of 70 to 75 percent) (Bell 1990); and
- energy assimilation efficiency (AE) for birds consuming insects - 72 percent (USEPA 1993a, Table 4-3).

Common Merganser

Body weight. Adult male common mergansers typically are heavier than adult females; however, not all investigators report weights separately for the sexes. In addition, Feltham

(1995) noted that although female mergansers were smaller than males, and tended to have higher FMRs, those differences were not significant.

There is some seasonal variation in body weights. Anderson and Timken (1972) found that as winter temperatures in South Dakota, Minnesota, and Oklahoma became colder, the average body weights of mergansers increased. The body weight assumed for the Ravena ERA is 1.27 kg, which is the mean body weight of 124 adults and juveniles of both sexes measured in winter in Michigan (Salyer and Lagler 1940).

Diet composition. Common mergansers typically forage in the shallower parts of large water bodies (e.g., lower reaches or mouths of rivers), moving to the middle reaches as the slower moving waters freeze over in winter (Salyer and Lagler 1940). They typically locate their prey by swimming on the surface and half-submerging their heads to look underwater (White 1937). They then pursue and capture their prey during short (10 to 20 second) dives (Salyer and Lagler 1940). In very shallow water, they sometimes feed by probing under rocks and sticks while partially submerged (Salyer and Lagler 1940).

The diet of common mergansers varies with local abundance of prey (Timken and Anderson 1969, White 1937). Several studies comparing fish availability with the composition of common merganser diets suggest that the birds consume the most abundant of the suitably sized available prey (White 1957, Huntington and Roberts 1959, Latta and Sharkey 1966, Sjöberg 1988, and McCaw et al. 1996 as cited in Mallory and Metz 1999).

For the exposure assessment, ICF used a review of the available literature to develop assumptions concerning the diet composition of common mergansers for this ERA. We used data, summarized in Exhibit 4-1, on the length distribution of fish reported caught in Michigan by Alexander (1977), with some consideration of studies from other locations (e.g., White 1936, 1967 and Huntington and Roberts 1959) and experimental choice studies (Latta and Sharkey 1966).

Exhibit 4-1. Distribution of Length of Fish Consumed by Common Mergansers in Michigan (Alexander 1977)

Measure	Length of Fish (inches)									
	1	2	3	4	5	6	7	8	9	10-13
Number of Fish Consumed	77	65	50	45	27	16	23	19	21	6
Percentage (n = 349)	22%	19%	14%	13%	8%	5%	7%	5%	6%	<2%

Latta and Sharkey (1966) reported that the largest captive merganser (1.7 kg) could consume a trout with a girth of 15.8 cm, while the smallest merganser (0.94 kg) could swallow trout with girth of up to only 12.5 cm. Offering six mergansers a total of 25 trout, 5 in each of five trout-size categories between 9.9 and 21 cm in length (that is 20 percent of the total trout in each size category), Latta and Sharkey (1966) found that the larger prey were captured less often than expected on the basis of their relative abundance: 28 percent of all trout consumed (N = 130) were from 9.9 to 11 cm (approximately 4 inches); 28 percent were from 12 to 13 cm (approximately 5 inches); 24 percent were from 15 to 16 cm (approximately 6 inches); 15 percent were from 17.5 to 18.3 cm (7 inches), and only 5 percent were from 20 to 21 cm (approximately 8 inches), although 20 percent of the trout offered were in that size range.

White (1936, 1937) found that common mergansers in Nova Scotia consumed tomcod and rainbow smelt that averaged 21 (8.3 inches) and 18 cm (7.1 inches), respectively, although some tomcod up to 27 cm (10.6 inches) were consumed. Huntington and Roberts (1959) found that 67 percent of 344 fish consumed by common mergansers in New Mexico were less than 10 cm (4 inches) in length; 84 percent were less than 20 cm (8 inches) in length, 94 percent were less than 24 cm (10 inches) in length. Less than 1 percent were greater than 30.5 cm (12 inches). The majority of the fish consumed (86 percent) were gizzard shad.

Given the fish compartments modeled in TRIM.FaTE, we assumed that medium-sized benthivorous fish and water column planktivorous fish (e.g., shiners 4 inches or less) were each 35 percent of the mergansers' total diet (on a wet-weight basis). Medium-sized "panfish" (water column omnivores 5 to 10 inches) were assumed to comprise 25 percent of the diet. Finally, we allowed 5 percent of the diet to be water column piscivorous fish (e.g., largemouth bass) greater than 10 inches to account for the larger fish consumed by common mergansers.

Food ingestion rate. Based on analyses of stomach contents and observed feeding rates, Salyer and Lagler (1940) estimated that American mergansers consume fish at a rate of between one third and one half of their body weight daily during winter in Michigan (0.33 to 0.50 g/g BW-day). Alexander (1977) also estimated a food ingestion rate of 0.33 g/g BW-day for mergansers consuming fish in Michigan. Gooders and Boyer (1986) estimated that mergansers consume an average of 445 g/d, or more than 0.33 g/g BW-day. Feltham (1995) used the doubly labeled water technique to demonstrate that males and females of *M.m. merganser* released on Scottish Rivers required 522 g and 480 g of food, equivalent to 0.32 and 0.40 g/g-day, respectively. Latta and Sharkey (1966) found that 8 captive common mergansers consumed between 0.183 and 0.257 g/g-day (mean ± SD of 0.208 ± 0.035 g/g-day). Based on these studies, we assumed a fish consumption rate of 0.33 g/g-day for the Ravena ERA.

Bald Eagle

Body weight. As for most raptors, female bald eagles typically weigh more than males. Snyder and Wiley (1976) reported a mean weight of 5.35 kg for 37 female and 4.13 kg for 35 male bald eagles. The adult body weight assumed for the Ravena ERA was 4.5 kg, which the average body weight for males and females combined used by Stalmaster and Gessaman (1984) and Craig et al. (1988) in their studies of bald eagle FMR.

Diet composition. Our assumption for the composition of the diet of bald eagles in the Ravena area is based on a review primary data sources (e.g., Dunstan and Harper 1975, Bowerman 1993, Grubb and Hensel 1978, Kozie and Anderson 1991, Todd et al. 1982) summarized in USEPA's draft Trophic Level and Exposure Analyses for Selected Piscivorous Birds and Mammals, Volumes 2 and 3 (USEPA 2005b). Overall, we assumed the diet to consist of 80 percent fish and 20 percent ducks. Further assumptions about the composition of the fish diet were based on length of fish documented by Bowerman (1993) for eagles in Michigan and Watson et al. (1991) for eagles in the Columbia River estuary. As reported in these studies, fish species consumed tend to include slow-moving benthivores, particularly suckers and catfish, as well as gizzard shad and carp, which generally are herbivores/detritivores. The high end of the proportion of piscivorous fish included in the bald eagle diet among the studies reviewed is 30 percent. Often, gizzard shad or other slow-swimming, lower trophic level species predominate. For the Ravena water bodies, we did not include the latter two species. The pelagic species caught by eagles can include salmonids, pike, and bass. For the Ravena ERA, we assumed the following breakdown of fish for the diet in addition to 20 percent ducks: 28 percent benthivores, 28 percent water column omnivores, and 24 percent water column carnivores

Food ingestion rate. Food ingestion rates were estimated separately for free-living adult bald eagles with diets consisting of 100 percent fish or 100 percent birds (i.e., mallards). Because birds have more metabolizable energy per unit wet weight than do fish, the eagle's caloric needs may be met with a smaller ingestion rate of birds than of fish (USEPA 2005b). The separate ingestion rates for fish and birds were then used to calculate a fresh-weight ingestion rate for bald eagles for a combined diet of 80 percent fish and 20 percent mallard on a wet-weight basis.

The ingestion rate for a diet consisting entirely of fish is based on similar results obtained by two separate research teams. Stalmaster and Gessaman (1984) observed captures of pre-weighed salmon provided at artificial feeding stations in Washington State. Although the eagles may have fed elsewhere on occasion, Stalmaster and Gessaman (1984) believed that the feeding stations provided most of the eagles' intake. They estimated the adult (including both sexes) ingestion rate to be 0.12 g/g-day. Craig et al. (1988) obtained the same estimate for bald eagles in Connecticut. Both research teams assumed the eagles weighed approximately 4.5 kg.

No feeding studies were available for bald eagles consuming waterfowl. Therefore, we estimated a mallard ingestion rate using the procedure and assumptions recommended in EPA's (1993a) Wildlife Exposure Factors Handbook. The assumptions included a GE content of 1.2 kcal/g fish and 2.0 kcal/g bird wet weight (EPA 1993a, Table 4-1) and a general energy AE (EPA 1993a, Table 4-3) of 79 percent for birds consuming fish and 78 percent for birds eating other birds. We then could estimate the average metabolizable energy (ME) for fish and birds in Equation J- below.

Equation J-3:

$$ME = GE \times AE$$

ME_{fish} = 1.2 kcal/g fish wet wt x 0.79 = 0.95 kcal ME/g fish wet wt
$ME_{mallard}$ = 2.0 kcal/g bird wet wt x 0.78 = 1.56 kcal ME/g bird wet wt

Next, the average ME was calculated for a diet of 80 percent fish and 20 percent mallard:

$ME_{average}$ = (0.8 x 0.95 kcal/g) + (0.2 x 1.56 kcal/g)
= 1.07 kcal ME/g combined diet wet wt

A total food ingestion rate (FIR) then could be estimated from the $ME_{average}$ and FMR estimated with data from Stalmaster and Gessaman (1984), normalized to body weight (4.5 kg), using EquationJ-4 below.

Equation J-4:

$$FIR_{total} = FMR / ME_{average}$$

FIR_{total} = (0.114 kcal/g BW-day) / (1.07 kcal ME/g food wet wt)
FIR_{total} = 0.1065 g/g-BW-day wet weight

Assuming a body weight of 4.5 kg, an adult eagle is estimated to consume a total of 480 g/d fresh food (FIR_{total} = 1.065 g/g-day x 4.5 kg x 1000 g/kg = 480 g/d). Still assuming that the combined diet includes 80 percent fish and 20 percent mallard, the daily ingestion rates for each food type are calculated as follows:

FIR_{fish} = 480 g/d x 0.8
= 384 g/d

$FIR_{mallard}$ = 480 g/d x 0.2
= 96 g/day

Mink

Body weights. Mink body size varies greatly throughout the species' range (adult males reaching no more than 1.4 kg in the east but up to 2.3 kg in the west of the United States according to Harding 1934 as cited in Linscombe et al. 1982). Males weigh markedly more than females (in some populations, almost twice as much). Mitchell (1961) reported mean body weights for wild mink captured in summer in Montana as 0.55 kg for females (n = 25) and 1.04 kg for males (n = 5). We averaged these mean male and female body weights to estimate a mink body weight of 0.8 kg for use in the Ravena ERA.

Diet composition. We reviewed the diet composition of mink reported on a wet weight basis by Alexander (1977) for southern Michigan year-round and by Sealander (1943) for the same area, but in the winter only. The other studies of mink diets summarized by EPA (1993b, 2005b) were based on measurements of remains in scats, which provide a poor indication of the proportion of diet on a wet-weight basis. In addition, we considered southern Michigan to be an adequate surrogate location for New York. Based on the two Michigan mink studies and the assumption of 100 percent aquatic prey, we specified the following diet for the Ravena ERA:

- 24 percent benthic invertebrates (e.g., crayfish),
- 25 percent medium-sized, benthivorous fish (e.g., small bullheads),
- 1 percent benthic carnivores (e.g., eels – captured in proportion to their relative biomass density in the Ravena water bodies),
- 25 percent small water column planktivorous fish, and
- 25 percent medium-sized “panfish” (water column omnivores).

Mink generally are not fast enough to capture the larger game fish. In reality, mink generally consume some of their diet from terrestrial sources; the diet specified above will introduce a conservative bias that could be reexamined later. In addition, mink are likely to include amphibians in their diet.

Food ingestion rates. Studies of captive mink indicate that mink eat at least 12 to 16 percent of their body weight in food daily. Assuming a body weight 1.4 kg (e.g., for a male mink) and Cowan et al.'s (1957) food-ingestion model derived from measures of prey consumed by captive mink, Arnold and Fritzell (1987) estimated that mink require 180 g/day fresh prey. Normalized to body weight, that food ingestion estimate is equivalent to 0.13 g/g-day. Bleavins and Aulerich (1981) measured food ingestion rates of farm-raised mink provided a diet of whole chicken (20 percent), commercial mink cereal (17 percent), ocean fish scraps (13 percent), beef parts, cooked eggs, and powdered milk. The moisture content of the feed overall was 66.2 percent. On this diet, the food consumption rate of female mink was 0.16 ± 0.0075 SE g/g-day, and that of male mink was 0.12 ± 0.0048 SE g/g-day (Bleavins and Aulerich 1981). Farrell and Wood (1968) documented how food requirements of mink depended on their activity level. Mink maintained in small cages required 20 kcal/100 g of body weight compared with 26 kcal/100 g body weight when the same animals were housed in larger ranch-type cages (Farrell and Wood 1968).

Nagy's (1987) allometric equation for estimating an FMR for non-herbivorous mammals predicts that a mink weighing 0.8 kg would require 196 kcal/d. Assuming that mink prey entirely on fish, that fish provide 1.2 kcal/g GE wet weight (EPA 1993a, Table 4-1), and that mink consuming fish exhibit an AE of 91 percent (EPA 1993a, Table 4-3), the fish ingestion rate of a male mink weighing 1.04 kg would be 0.22 g/g-day and of a female mink weighing 0.55 kg would be 0.24 g/g-day. For an average adult of 0.8 kg, the fish ingestion rate would be 0.23 g/g-day. Assuming that mink prey entirely on benthic invertebrates, that crayfish provide the same GE as shrimp (1.1 kcal/g wet weight; EPA 1993, Table 4-1), and that the AE is similar to mammals consuming insects (87 percent; EPA 1993, Table 4-3), the invertebrate ingestion rate would be very similar rounded to two significant digits, or 0.24 g/g-day. With a diet of 75 percent fish and 25 percent benthic invertebrates, we set a food ingestion rate for mink in the Ravena ERA of 0.23 g/g-day.

Assuming that wild mink are likely to be more active than captive mink, the higher food ingestion rates estimated from Nagy's (1987) allometric equation appear to be more appropriate for wildlife exposure analyses than the food ingestion rates measured for captive animals.

Summary of Exposure Assumptions

Exhibit 4-2 through Exhibit 4-6 summarize the exposure assumptions developed above for the four wildlife species included in the Ravena ERA. These assumptions were used to estimate daily intake rates of Hg and TCDD by each species.

Average adult body weight and food ingestion rates are summarized in Exhibit 4-2. Because the averages are for adults of both sexes, exposure estimates calculated with these assumptions may be over- or under-estimated by gender for sexually dimorphic species. For example, adult male mink are markedly larger than females, and adult female bald eagles are larger than males. Regional variation in body weight among different populations also may mean that the body weights assumed for the Ravena ERA are over- or under-estimated.

Exhibit 4-2. Annual Mean Adult Body Weights and Food Ingestion Rtes Assumed for Wildlife Species

Species	Mean Adult Body Weight (kg)	Food Ingestion Rate (g/day)
Tree Swallow	0.0201	26.9
Common Merganser	1.27	419.1
Bald Eagle	4.5	179.6
Mink	0.8	478.3

Exhibit 4-3 summarizes the percentages of each species' diet assumed for each of the nine TRIM.FaTE aquatic food web compartments in Alcove Reservoir, Nassau Lake, and Kinderhook Lake. As discussed above, these assumptions are based on published reports of feeding behavior of free-living animals, and do not account for regional and seasonal variations (e.g., due to local variations in prey abundance).

Exhibit 4-3. Fraction Diet Assumptions for Wildlife Feeding from Alcove Reservoir, Nassau Lake, and Kinderhook Lake

Food Type	Tree Swallow	Common Merganser	Bald Eagle	Mink
Algae	0%	0%	0%	0%

Macrophytes	0%	0%	0%	0%
Benthic Invertebrates	100%	0%	0%	24%
Benthivorous Fish	0%	35%	28%	25%
Bottom-feeding Carnivores	0%	0%	0%	1%
Water Column Planktivores	0%	35%	0%	25%
Water Column Omnivores	0%	25%	28%	25%
Water Column Carnivores	0%	5%	24%	0%
Mallard Duck	0%	0%	20%	0%
All Food Types	**100%**	**100%**	**100%**	**100%**

Because Ravena Pond is smaller than the other water bodies, its food web was shortened to be consistent with available literature. Specifically, bottom-feeding carnivores and water column omnivores were not included in the Ravena Pond food web. Because these food types are not available to wildlife species that feed at Ravena Pond, the percentages of available food types in each species' diet were scaled upward so that the percentages would sum to 100 percent. Exhibit 4-4 displays the diet compositions for species for feeding at Ravena Pond.

Exhibit 4-4. Fraction Diet Assumptions for Wildlife Feeding from Ravena Pond as Modeled in TRIM.FaTE

Food Type	Tree Swallow	Common Merganser	Bald Eagle	Mink
Algae	0%	0%	0%	0%
Macrophytes	0%	0%	0%	0%
Benthic Invertebrates	100%	0%	0%	32%
Benthivorous Fish	0%	47%	43%	34%
Bottom-feeding Carnivores	--	--	--	--
Water Column Planktivores	0%	47%	0%	34%
Water Column Omnivores	--	--	--	--
Water Column Carnivores	0%	6%	37%	0%
Mallard Duck	0%	0%	20%	0%
All Food Types	**100%**	**100%**	**100%**	**100%**

The diet composition percentages in Exhibit 4-3 and Exhibit 4-4 were multiplied by total daily food ingestion rates in Exhibit 4-2, to estimate daily ingestion rates for each of the nine food types. These ingestion rate estimates are presented in Exhibit 4-5 for Alcove Reservoir, Kinderhook Lake, and Nassau Lake, and in Exhibit 4-6 for Ravena Pond.

Exhibit 4-5. Estimated Average Daily Ingestion Rate of Each Food Type in the Diets of Wildlife Species from Alcove Reservoir, Nassau Lake, and Kinderhook Lake (g/day)

Food Type	Tree Swallow	Common Merganser	Bald Eagle	Mink
Algae	--	--	--	--
Macrophytes	--	--	--	--
Benthic Invertebrates	26.9	--	--	43.1
Benthivorous Fish	--	146.7	133.9	44.9

Bottom-feeding Carnivores	--	--	--	1.8
Water Column Planktivores	--	146.7	--	44.9
Water Column Omnivores	--	104.8	133.9	44.9
Water Column Carnivores	--	21.0	114.8	--
Mallard Duck	--	--	95.7	--
All Food Types	**26.9**	**419.1**	**478.3**	**179.6**

Exhibit 4-6. Estimated Average Daily Ingestion Rate of Each Food Type in the Diets of Wildlife Species from Ravena Pond (g/day)

Food Type	Tree Swallow	Common Merganser	Bald Eagle	Mink
Algae	--	--	--	--
Macrophytes	--	--	--	--
Benthic Invertebrates	26.9	--	--	58.2
Benthivorous Fish	--	197.0	206.0	60.7
Bottom-feeding Carnivores	--	--	--	--
Water Column Planktivores	--	197.0	--	60.7
Water Column Omnivores	--	--	--	--
Water Column Carnivores	--	25.1	176.6	--
Mallard Duck	--	--	95.7	--
All Food Types	**26.9**	**419.1**	**478.3**	**179.6**

J-4.1.1.2 Exposure Concentrations

Exhibit 4-7 through Exhibit 4-10 present estimated exposure concentrations for Hg and TCDD in each of the nine biotic and two abiotic compartments of the TRIM.FaTE aquatic food web in year 50 of the Ravena TRIM.FaTE simulation. For Hg, results are presented separately for methyl mercury (MeHg) and divalent mercury (Hg+2) because it is the methylated form that bioaccumulates (i.e., is not readily eliminated from animals). The MeHg fish tissue concentrations associated with the mean emissions rate of Hg from the Ravena facility are presented in Exhibit 4-7, and the analogous data for Hg+2 are presented in Exhibit 4-8.

Exhibit 4-7. Concentrations (µg/g) of Methyl Mercury in Compartments of the TRIM.FaTE Aquatic Food Web at Year 50 – Based on Mean Measured Annual Hg Emission Rate [a]

Compartment	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Surface Water	4.8E-07	2.0E-09	2.5E-09	2.6E-09
Sediment	1.9E-03	1.3E-05	1.5E-05	1.9E-05
Algae	1.7E-02	7.0E-05	8.6E-05	9.1E-05
Macrophytes	9.6E-07	4.0E-09	4.9E-09	5.2E-09
Benthic Invertebrates	5.9E-03	4.0E-05	4.8E-05	6.1E-05
Benthivorous Fish	3.0E-02	1.4E-04	2.2E-04	1.8E-04
Bottom-feeding Carnivores	N/A	4.9E-04	7.3E-04	6.6E-04
Water Column Planktivores	5.8E-02	6.3E-05	8.1E-05	7.4E-05

Water Column Omnivores	N/A	1.9E-04	2.9E-04	2.9E-04
Water Column Carnivores	1.7E-01	5.6E-04	1.1E-03	8.7E-04
Mallard Duck	6.5E-03	4.4E-05	5.3E-05	6.8E-05

N/A = Not applicable. Bottom-feeding carnivores and water column omnivores are not included in the Ravena Pond food web.
[a] Concentrations in surface water are in mg [MeHg]/L [water], which is equivalent to µg [MeHg]/g [water]. Concentrations in bulk sediment are in µg [MeHg]/g [sediment] dry weight. Concentrations in biota are µg [MeHg]/g [biotal] wet weight.

Exhibit 4-8. Concentrations (µg/g) of Divalent Mercury in Compartments of the TRIM.FaTE Aquatic Food Web at Year 50 – Based on Mean Measured Annual Hg Emission Rate [a]

Compartment	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Surface Water	1.1E-04	1.4E-07	1.6E-07	2.1E-07
Sediment	9.1E-01	6.1E-03	7.2E-03	9.3E-03
Algae	4.9E-01	5.9E-04	6.9E-04	9.0E-04
Macrophytes	5.3E-05	1.3E-07	9.1E-08	1.4E-07
Benthic Invertebrates	4.6E-02	3.2E-04	3.7E-04	4.8E-04
Benthivorous Fish	1.6E-02	1.0E-04	1.3E-04	1.6E-04
Bottom-feeding Carnivores	N/A	8.0E-05	9.4E-05	1.2E-04
Water Column Planktivores	1.5E-01	1.5E-04	1.7E-04	1.9E-04
Water Column Omnivores	N/A	9.0E-05	1.1E-04	1.4E-04
Water Column Carnivores	3.5E-02	6.7E-05	4.5E-05	9.2E-05
Mallard Duck	3.1E-02	2.1E-04	2.5E-04	3.2E-04

N/A = Not applicable. Bottom-feeding carnivores and water column omnivores are not included in the Ravena Pond food web.
[a] Concentrations in surface water are in mg [Hg2+]/L [water], which is equivalent to µg [Hg2+]/g [water]. Concentrations in bulk sediment are in µg [Hg2+]/g [sediment] dry weight. Concentrations in biota are µg [Hg2+]/g [biotal] wet weight.

MeHg and Hg+2 exposure concentrations (Exhibit 4-7 and Exhibit 4-8, respectively) are presented in micrograms of MeHg per gram of the food type in wet weight (µg/g ww). For example, the concentration of MeHg estimated for mallard ducks feeding in Ravena Pond of 0.0065 µg/g wet weight represents the average whole-body concentration of MeHg in an individual duck.

As described in Section J-3.1, dioxin emissions monitoring data were not available for the Ravena facility. Therefore, mean and 95-percent UCL emission rates were using emissions factors based on clinker capacity and process type. Whole-body fish tissue and abiotic media concentrations estimated for the mean and 95-percent UCL emissions estimates from year 50 of the Ravena TRIM.FaTE simulation are presented in Exhibit 4-9 and Exhibit 4-10, respectively.

Exhibit 4-9. Concentrations (µg/g) of 2,3,7,8-TCDD in Compartments of the TRIM.FaTE Aquatic Food Web at Year 50 with Mean Emission Rate [a]

Compartment	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Surface Water	5.3E-12	1.1E-14	3.8E-14	2.8E-14
Sediment	2.6E-08	1.6E-10	5.4E-10	4.0E-10
Algae	3.4E-09	7.1E-12	2.4E-11	1.8E-11
Macrophytes	7.7E-08	1.7E-10	5.6E-10	4.1E-10
Benthic Invertebrates	2.5E-09	1.6E-11	5.8E-11	4.5E-11
Benthivorous Fish	1.9E-06	8.9E-10	7.8E-09	1.5E-09
Bottom-feeding Carnivores	N/A	4.6E-09	3.9E-08	8.6E-09
Water Column Planktivores	3.6E-07	8.8E-11	3.4E-10	3.0E-10
Water Column Omnivores	N/A	1.3E-09	1.2E-08	5.7E-09
Water Column Carnivores	2.1E-06	7.7E-09	1.0E-07	2.9E-08
Mallard Duck	1.3E-05	2.8E-08	9.4E-08	7.0E-08

N/A = Not applicable. Bottom-feeding carnivores and water column omnivores are not included in the Ravena Pond food web.

[a] Concentrations in surface water are in mg [2,3,7,8-TCDD]/L [water], which is equivalent to µg/g. Concentrations in bulk sediment are in µg [2,3,7,8-TCDD]/g [sediment] dry weight. Concentrations in biota are µg[2,3,7,8-TCDD]/g [biota] wet weight.

Exhibit 4-10. Concentrations (µg/g) of 2,3,7,8-TCDD in Compartments of the TRIM.FaTE Aquatic Food Web at Year 50 with 95-percent UCL Emission Rate [a]

Compartment	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Surface Water	1.3E-11	2.7E-14	9.2E-14	6.8E-14
Sediment	6.3E-08	3.9E-10	1.3E-09	9.7E-10
Algae	8.3E-09	1.7E-11	5.9E-11	4.3E-11
Macrophytes	1.9E-07	4.1E-10	1.4E-09	1.0E-09
Benthic Invertebrates	6.2E-09	3.8E-11	1.4E-10	1.1E-10
Benthivorous Fish	4.7E-06	2.2E-09	1.9E-08	3.7E-09
Bottom-feeding Carnivores	N/A	1.1E-08	9.6E-08	2.1E-08
Water Column Planktivores	8.8E-07	2.2E-10	8.4E-10	7.3E-10
Water Column Omnivores	N/A	3.2E-09	3.0E-08	1.4E-08
Water Column Carnivores	5.2E-06	1.9E-08	2.5E-07	7.2E-08
Mallard Duck	3.1E-05	6.8E-08	2.3E-07	1.7E-07

N/A = Not applicable. Bottom-feeding carnivores and water column omnivores are not included in the Ravena Pond food web.

[a] Concentrations in surface water are in mg [2,3,7,8-TCDD]/L [which = mg/kg or µg/g]. Concentrations in bulk sediment are in µg [2,3,7,8-TCDD]/g [sediment] dry weight. Concentrations in biota are µg [2,3,7,8-TCDD]/g [biota] wet weight.

2,3,7,8-TCDD exposure concentrations based on the 95-percent UCL emission rate are generally less than an order of magnitude greater than exposure concentrations based on the mean emission rate.

Chemical concentrations in the fish compartments are highest in Ravena pond which has the highest ratio of catchment area to water volume of the four water bodies evaluated. Previous case studies using TRIM.FaTE had indicated that harvest rate of fish from a water body influenced chemical concentrations in the fish compartments by removing chemical from the system. The influence of a given harvest rate on chemical concentrations in fish decreased with increasing total fish biomass in the water body. We therefore decided to investigate the consequences of simulating a single angler harvesting fish from Ravena Pond (as assumed in the Ravena HHRA, Appendix I) on the chemical concentrations in fish compartments.

A single mammalian TRIM.FaTE compartment was added to the terrestrial parcel adjacent to Ravena Pond. The body weight of the angler, who was assumed to live in the house near Ravena Pond, was set to 71.4 kg, the average weight of an adult human as used in the Ravena HHRA (Appendix I). We assumed that the angler would harvest fish at a rate of 17 g/day (90^{th} percentile fish ingestion rate, see Appendix C, Attachment 2) from Ravena Pond, and of the fish harvested 67 percent were fish from the benthic omnivore (BO) fish compartment and 33 percent were fish from the water column carnivore (WCC) compartment to reflect the assumptions used in the Ravena HHRA (Appendix I). We assumed that the small forage fish (water column herbivore) were too small to be keepers. Note that the actual fish *harvest* rate expressed as *total biomass of fish removed* from Ravena Pond that would correspond to the human fish ingestion rate listed here is likely to be 2 to 3 times higher because humans do not consume an entire fish. The fillet generally constitutes a third of the wet weight of a fish, and edible muscle with skin generally is no more than half of the wet weight of a fish. Although wildlife also may feed on fish from Ravena Pond, we did not include consumption of fish by wildlife.

Exhibit 4-11 compares the annual angler fish harvest to the standing biomass for each fish compartment in Ravena Pond, which has a surface area of 20,000 m^2. Note that as the angler removes fish (with chemical) from the pond, TRIM.FaTE maintains the same biomass for the fish compartment. That model feature is consistent with recruitment of younger, less contaminated fish, into the pond adult fish population at a rate equal to the removal rate of the adults.

Exhibit 4-11. Biomass of Fish Harvested by a Single Angler Fishing in Ravena Pond Relative to Standing Biomass of Fish in Each Compartment [a]

Compartment-specific Properties	Water Column Carnivore	Benthic Omnivore	Total Fish Biomass
Fish Biomass Density (kg ww/m2)	0.00020	0.0030	0.0040
Total Fish Biomass in Pond (kg)	4.01	60.2	80.2
Fish Biomass (kg) Harvested Annually by Single Angler at Mean Fish Ingestion Rate (17 g/day)	4.14	2.07	6.21

[a] Total surface area of Ravena Pond is approximately 20,000 m^2.

Exhibit 4-11 illustrates several points. First, an angler harvesting fish at a rate of 17.0 g/day would need to catch fish at other water bodies in addition to Ravena Pond. The water column

carnivore harvest rate of 4.14 kg/year is not possible with a standing stock of only 4.01 kg total in Ravena Pond. The benthic omnivore harvest rate of 2.07 kg/year from a standing biomass of 60.2 kg is more reasonable and likely to be sustainable.

Exhibit 4-12 summarizes the chemical concentrations in each of the aquatic compartments for Ravena Pond as presented in the previous exhibits. Exhibit 4-13 summarizes the chemical concentrations in the two fish compartments for which concentration changed with the addition of the single angler harvester.

Exhibit 4-12. Concentrations (µg/g) of Mercury and 2,3,7,8-TCDD in the Ravena Pond Aquatic Compartments at Year 50 Without Fish Harvesting by Humans or Wildlife [a]

Compartment	Ravena Pond			
	MeHg	Hg+2	Mean TCDD	95th UCL TCDD
Surface Water	4.8E-07	1.1E-04	5.3E-12	1.3E-11
Sediment	1.9E-03	9.1E-01	2.6E-08	6.3E-08
Algae	1.7E-02	4.9E-01	3.4E-09	8.3E-09
Macrophytes	9.6E-07	5.3E-05	7.7E-08	1.9E-07
Benthic Invertebrates	5.9E-03	4.6E-02	2.5E-09	6.2E-09
Benthivorous Fish	3.0E-02	1.6E-02	1.9E-06	4.7E-06
Bottom-feeding Carnivores	N/A	N/A	N/A	N/A
Water Column Planktivores	5.8E-02	1.5E-01	3.6E-07	8.8E-07
Water Column Omnivores	N/A	N/A	N/A	N/A
Water Column Carnivores	1.7E-01	3.5E-02	2.1E-06	5.2E-06
Mallard Duck	6.5E-03	3.1E-02	1.3E-05	3.1E-05

N/A= Not applicable. Bottom-feeding carnivores and water column omnivores are not included in the Ravena Pond food web.

[a] Concentrations in surface water are in mg/L. Concentrations in sediment are in mg/kg [sediment] dry wt. Concentrations in biota are mg/kg wet weight.

Exhibit 4-13. Concentrations (µg/g) of Mercury and 2,3,7,8-TCDD in the Ravena Pond Aquatic Compartments at Year 50 With 17 Grams Fish Harvested per Day by One Angler from Two Fish Compartments [a]

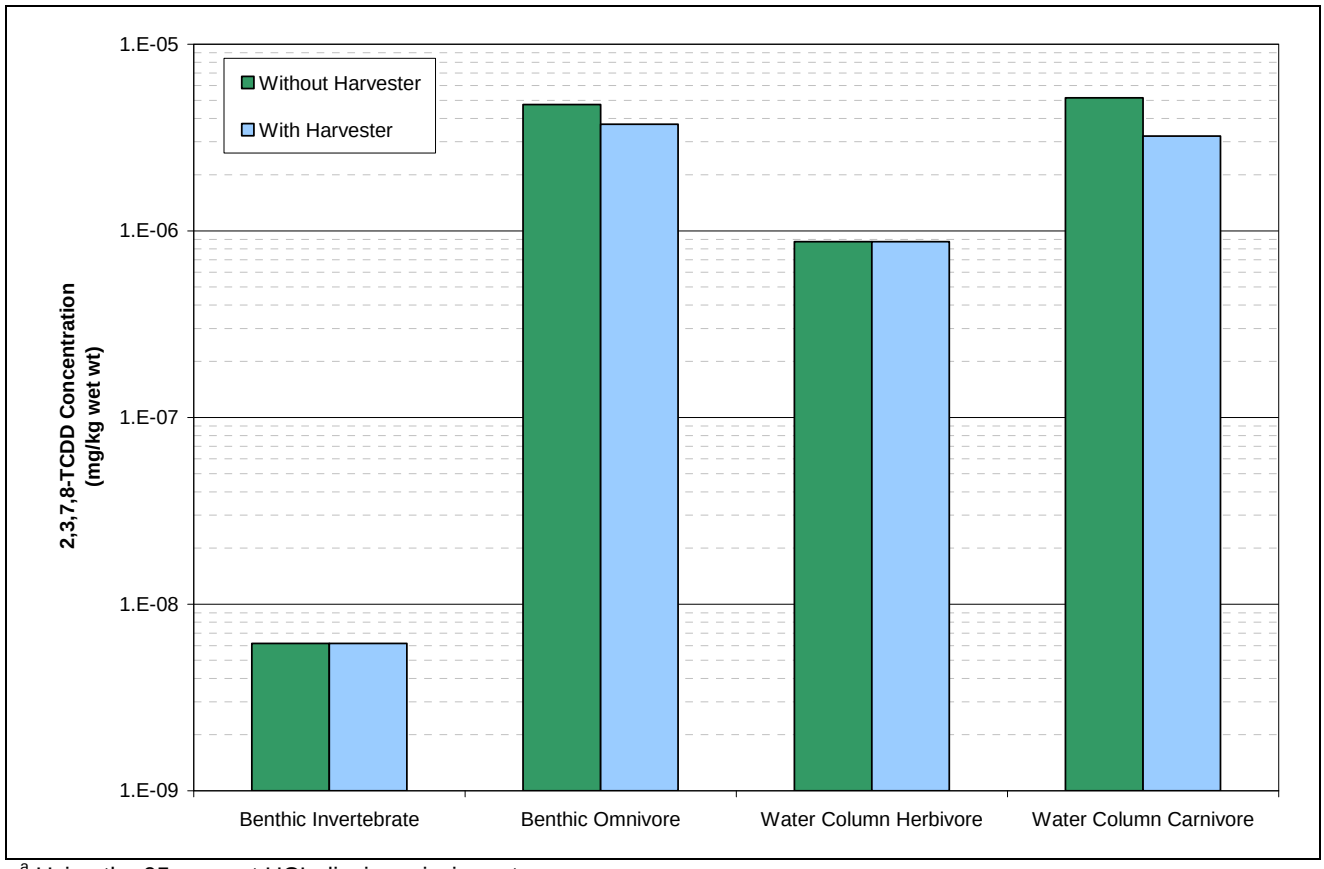
Compartment	Ravena Pond			
	MeHg	Hg+2	Mean TCDD	95th Percentile TCDD
Benthivorous Fish	3.0E-02	1.6E-02	1.9E-06	4.7E-06
Water Column Carnivores	1.7E-01	3.5E-02	2.1E-06	5.2E-06

[a] Concentrations in biota are µg [2,3,7,8-TCDD]/g [biota] wet weight. Concentrations for all other aquatic compartments are the same as in Exhibit 4-12; they were unaffected by harvesting 17 grams of fish daily (33 percent from water column carnivore and 67 percent from benthic omnivore (benthivorous) fish compartments).

With the addition of an angler harvesting 17.0 g/day or 6.21 kg/year of fish from Ravena Pond, chemical concentrations in the fish compartments which the angler harvests decrease substantially. In other fish compartments, chemical concentrations do not change with the addition of the angler. Exhibit 4-14 below presents the 2,3,7,8-TCDD concentrations with and without the angler in Ravena Pond. When the angler is present, fish concentrations in both the water column carnivore and benthic omnivore fish compartments are reduced by 21 percent for the benthic omnivore, and 38 percent for the water column carnivore. The proportional reduction in chemical concentration in the water column carnivore represents an unrealistically high harvest rate. The concentrations in the water column herbivore and the benthic

invertebrates included for comparison are the same with and without the angler harvesting fish. The concentrations in these compartments remain unchanged because there are no changes to the rate at which their predators consume biomass (and chemical) from those compartments.

Exhibit 4-14. 2,3,7,8-TCDD Concentrations in Aquatic Foodweb Compartments With and Without Angler Harvesting of 17 Grams of Fish Daily in Ravena Pond



[a] Using the 95-percent UCL dioxin emission rate.

Exhibit 4-15 presents the mercury concentrations with and without an angler harvesting fish from Ravena Pond. With harvesting, fish concentrations are reduced substantially for the water column carnivore for both divalent and methyl mercury (20 percent and 42 percent, respectively). Fish concentrations are reduced to a lesser amount with the angler present for the benthic omnivore fish compartment (2 percent Hg+2 reduction, 7 percent MeHg reduction).

Exhibit 4-15. Concentrations of Divalent and Methyl Mercury in Aquatic Foodweb Compartments, With and Without Angler Harvesting of 17.0 grams of Fish Daily in Ravena Pond

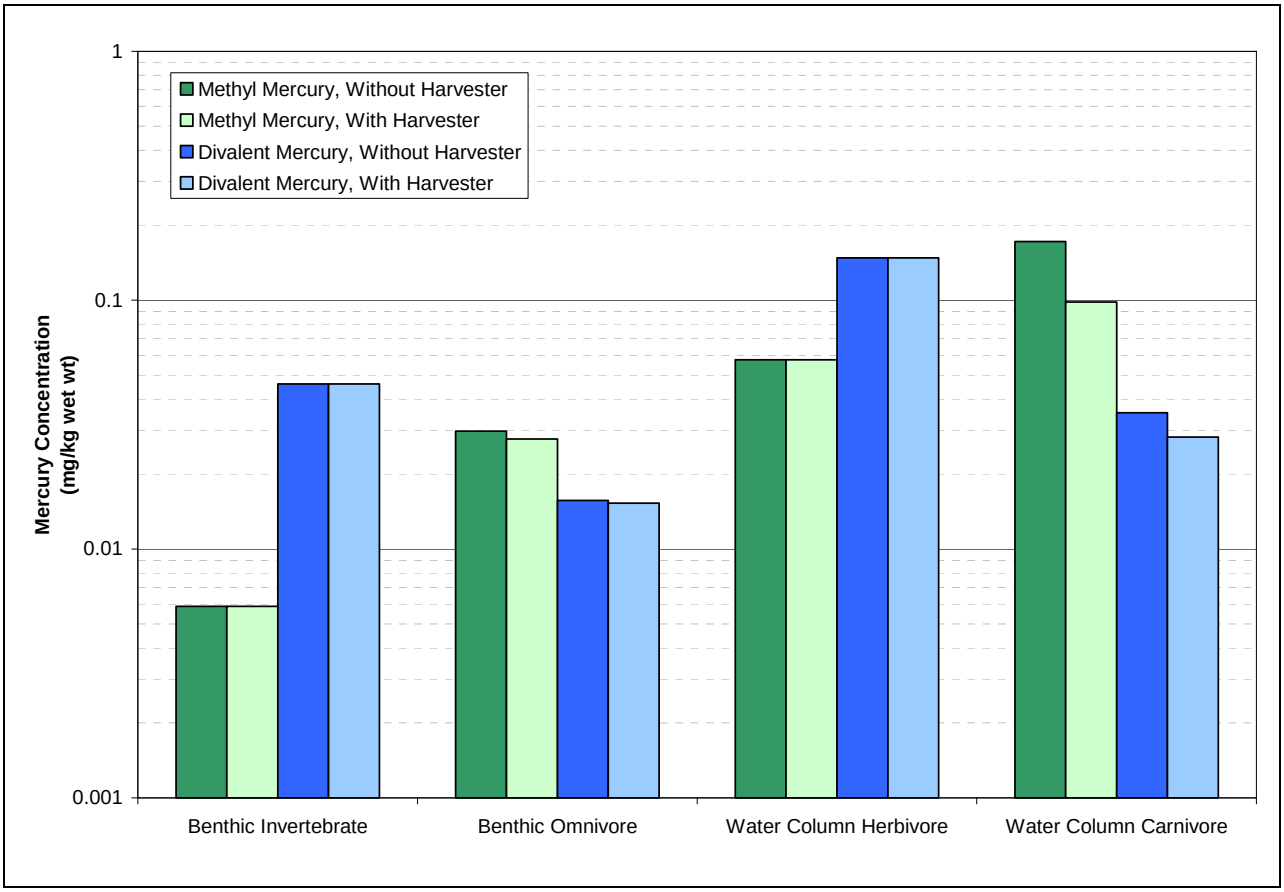


Exhibit 4-15 also illustrates that the proportion of total mercury present as MeHg is higher for the water column carnivore than for the water column herbivore (higher for the higher fish trophic level), which is consistent with reports from the literature (EPA 2005). While humans harvest primarily the larger fish, wildlife that swallow their prey whole (e.g., mergansers) generally harvest smaller fish, and so may be exposed to less MeHg than indicated by measurements or estimates of total mercury.

Exhibit 4-16 shows the TRIM.FaTE predicted speciation of mercury in the aquatic ecosystem compartments for Nassau Lake. Two of the fish compartments, the benthic carnivore and water column omnivore, were not modeled in Ravena Pond; therefore, Nassau Lake is used to illustrate Hg speciation among aquatic compartments. The majority of the total Hg in algae/zooplankton, macrophytes, and benthic invertebrates (89, 95, and 88 percent, respectively) is in the inorganic, Hg+2, form. At the next *benthic* trophic level (benthivorous fish), TRIM.FaTE estimated 37 percent Hg+2 and 63 percent MeHg. At the next trophic level in the *water column* (water column planktivores), however, TRIM.FaTE estimated 67 percent Hg+2 and 33 percent MeHg. Given that MeHg comprises 11 percent of the total Hg in the diets of both the benthivorous fish (100% benthic invertebrates) and the water column planktivorous fish (97% plankton), this difference in Hg speciation at the next higher trophic level was not expected. Possible reasons are still under examination. As expected for top predators, most of the total Hg in the bottom-feeding carnivorous fish, 89 percent, was estimated to be MeHg, while 96 percent was estimated to be MeHg in the water column carnivores.

Exhibit 4-16. Speciated Mercury Concentrations for Surface Water, Sediment, and Biota in Nassau Lake (ppm [SW: mg/L; sediment: µg/g dry weight; algae, BI, fish: µg/g wet weight])

Foodweb Compartment	Total Hg Conc.	Divalent Hg		Elemental Hg		Methyl Hg	
		Conc.	% Total Hg	Conc.	% Total Hg	Conc.	% Total Hg
Surface Water	1.9E-07	1.6E-07	86%	2.4E-08	13%	2.5E-09	1%
Sediment	7.2E-03	7.2E-03	99%	3.9E-05	1%	1.5E-05	0%
Algae/zooplankton	7.8E-01	6.9E-01	89%	0.0E+00	0%	8.6E-02	11%
Macrophytes	9.6E-08	9.1E-08	95%	1.6E-17	0%	4.9E-09	5%
Benthic Invertebrates	4.2E-04	3.7E-04	88%	2.0E-06	0%	4.8E-05	11%
Benthivorous Fish	3.4E-04	1.3E-04	37%	2.9E-12	0%	2.2E-04	63%
Bottom-feeding Carnivores	8.2E-04	9.4E-05	11%	1.0E-12	0%	7.3E-04	89%
Water Column Planktivores	2.5E-04	1.7E-04	67%	1.2E-13	0%	8.1E-05	33%
Water Column Omnivores	4.0E-04	1.1E-04	28%	2.3E-12	0%	2.9E-04	72%
Water Column Carnivores	1.2E-03	4.5E-05	4%	3.8E-19	0%	1.1E-03	96%
Mallards	3.0E-04	2.5E-04	82%	3.1E-07	0%	5.3E-05	17%

J-4.1.1.3 Exposure Doses

MeHg and 2,3,7,8-TCDD exposure doses for the four wildlife species included in the Ravena ERA were estimated by first multiplying the estimated chemical concentrations in each food type (Exhibit 4-7 through Exhibit 4-10) by the daily ingestion rates of each food type (Exhibit 4-5 and Exhibit 4-6) to yield average daily intake rates for each chemical for each surface water body. Then, the intake rates were divided by body weights (Exhibit 4-2) to calculate the body-weight normalized chemical intake rate or dose (µg/g-day). Intakes of MeHg and 2,3,7,8-TCDD are calculated for each wildlife species, food type, and water body. Because of the different health endpoints for MeHg and Hg+2, the exposures are estimated separately for each. For Ravena Pond, we used the most conservative scenario to calculate the fish compartment concentrations – no fish harvesting by anglers or wildlife.

MeHg intake rates are presented in Exhibit 4-17 through Exhibit 4-20. Each exhibit includes the MeHg intake rates for one wildlife species by water body and by prey type. Total MeHg intake rates for each water body are shown in the bottom row of each exhibit. These total MeHg intake rates are compared to reference toxicity values to calculate hazard indices in Section J-4.1.2.

Exhibit 4-17. Tree Swallow Intake of MeHg (µg/g-day)

Food Type	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Benthic Invertebrates	1.6E-04	1.1E-06	1.3E-06	1.6E-06
Total	**1.6E-04**	**1.1E-06**	**1.3E-06**	**1.6E-06**

Exhibit 4-18. Common Merganser Intake of MeHg (µg/g-day)

Food Type	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Benthivorous Fish	5.9E-03	2.0E-05	3.2E-05	2.6E-05
Water Column Planktivores	1.1E-02	9.3E-06	1.2E-05	1.1E-05
Water Column Omnivores	N/A	2.0E-05	3.0E-05	3.0E-05
Water Column Carnivores	4.3E-03	1.2E-05	2.3E-05	1.8E-05
Total	**2.2E-02**	**6.1E-05**	**9.7E-05**	**8.6E-05**

N/A = Not applicable. Water column omnivores are not included in the Ravena Pond food web.

Exhibit 4-19. Bald Eagle Intake of MeHg (µg/g-day)

Food Type	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Benthivorous Fish	6.1E-03	1.9E-05	2.9E-05	2.4E-05
Water Column Omnivores	N/A	2.5E-05	3.9E-05	3.9E-05
Water Column Carnivores	3.0E-02	6.5E-05	1.3E-04	1.0E-04
Mallard Duck	6.2E-04	4.3E-06	5.0E-06	6.5E-06
Total	**3.7E-02**	**1.1E-04**	**2.0E-04**	**1.7E-04**

N/A = Not applicable. Water column omnivores are not included in the Ravena Pond food web.

Exhibit 4-20. Mink Intake of MeHg (µg/g-day)

Food Type	Water Body			
	Ravena Pond	Alcove Res	Nassau Lake	Kinderhook Lake
Benthic Invertebrates	3.4E-04	1.7E-06	2.1E-06	2.6E-06
Benthivorous Fish	1.8E-03	6.2E-06	9.7E-06	8.1E-06
Bottom-feeding Carnivores	N/A	8.8E-07	1.3E-06	1.2E-06
Water Column Planktivores	3.5E-03	2.9E-06	3.7E-06	3.3E-06
Water Column Omnivores	N/A	8.4E-06	1.3E-05	1.3E-05
Total	**5.6E-03**	**2.0E-05**	**3.0E-05**	**2.8E-05**

N/A = Not applicable. Bottom-feeding carnivores and water column omnivores are not included in the Ravena Pond food web.

Given the relative magnitude of MeHg intakes from Ravena Pond compared with the three other water bodies (more than two orders of magnitude higher), we estimated intakes of Hg+2 (divalent mercury) for wildlife at Ravena Pond only as shown in Exhibit 4-21.

Exhibit 4-21. Wildlife Intakes of Hg+2 (µg/g-day) at Ravena Pond

Food Type	Species			
	Swallow	Merganser	Bald Eagle	Mink
Benthic Invertebrates	1.2E-03	--	--	2.7E-03
Benthivorous Fish	--	3.1E-03	3.2E-03	9.5E-04
Bottom-feeding Carnivores	--	--	--	--
Water Column Planktivores	--	2.9E-02	--	9.0E-03
Water Column Omnivores	--	--	--	--
Water Column Carnivores	--	8.9E-04	6.2E-03	--
Mallard Duck	--	--	2.9E-03	--
Total	**1.2E-03**	**3.3E-02**	**1.2E-02**	**1.3E-02**

-- = No ingestion of this compartment.

As discussed in the previous section, mean and 95-percent UCL emission rates for 2,3,7,8-TCDD were used to estimate exposure concentrations. Because the exposure concentrations based on the 95-percent UCL emission rates were generally within an order of magnitude of the exposure concentrations based on mean emissions rate, exposure doses for 2,3,7,8-TCDD were calculated using only the 95-percent UCL emission rates (Exhibit 4-10). The resulting 2,3,7,8-TCDD exposure doses are presented in Exhibit 4-22 through Exhibit 4-25.

Exhibit 4-22. Tree Swallow Intake of 2,3,7,8-TCDD (µg/g-day)[a]

Food Type	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Benthic Invertebrates	1.7E-10	1.0E-12	3.8E-12	2.9E-12
Total	**1.7E-10**	**1.0E-12**	**3.8E-12**	**2.9E-12**

[a] Exposure doses are based on the estimated 95-percent UCL dioxin emission rates.

Exhibit 4-23. Common Merganser Intake of 2,3,7,8-TCDD (µg/g-day)[a]

Food Type	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Benthivorous Fish	9.3E-07	3.2E-10	2.8E-09	5.5E-10
Water Column Planktivores	1.7E-07	3.2E-11	1.2E-10	1.1E-10
Water Column Omnivores	N/A	3.4E-10	3.1E-09	1.5E-09
Water Column Carnivores	1.3E-07	4.0E-10	5.2E-09	1.5E-09
Total	**1.2E-06**	**1.1E-09**	**1.1E-08**	**3.6E-09**

[a] Exposure doses are based on the estimated 95-percent UCL dioxin emission rates.
N/A = Not applicable. Water column omnivores are not included in the Ravena Pond food web.

Exhibit 4-24. Bald Eagle Intake of 2,3,7,8-TCDD (µg/g-day)[a]

Food Type	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Benthivorous Fish	9.8E-07	2.9E-10	2.6E-09	5.0E-10
Water Column Omnivores	N/A	4.3E-10	4.0E-09	1.9E-09
Water Column Carnivores	9.1E-07	2.2E-09	2.9E-08	8.3E-09
Mallard Duck	3.0E-06	6.5E-09	2.2E-08	1.6E-08
Total	**4.9E-06**	**9.4E-09**	**5.7E-08**	**2.7E-08**

[a] Exposure doses are based on the estimated 95-percent UCL dioxin emission rates.
na = Not applicable. Bottom-feeding carnivores and water column omnivores are not included in the Ravena Pond food web.

Exhibit 4-25. Mink Intake of 2,3,7,8-TCDD (µg/g-day)[a]

Food Type	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Benthic Invertebrates	3.6E-10	1.7E-12	6.2E-12	4.7E-12
Benthivorous Fish	2.9E-07	9.7E-11	8.6E-10	1.7E-10
Bottom-feeding Carnivores	N/A	2.0E-11	1.7E-10	3.8E-11
Water Column Planktivores	5.3E-08	9.7E-12	3.8E-11	3.3E-11
Water Column Omnivores	N/A	1.4E-10	1.3E-09	6.2E-10
Total	**3.4E-07**	**2.7E-10**	**2.4E-09**	**8.6E-10**

[a] Exposure doses are based on the estimated 95-percent UCL dioxin emission rates.
N/A = Not applicable. Bottom-feeding carnivores and water column omnivores are not included in the Ravena Pond food web.

J-4.1.2 Ecological Effects Assessment

As described in Section J-2, protection of local populations of three species of piscivorous and one species of insectivorous wildlife were selected as the ecological assessment endpoints. To evaluate the risks associated with exposure to 2,3,7,8-TCDD and Hg in fish or insects, a benchmark dose below which population-level effects are considered unlikely is needed for each combination of chemical and receptor species. This section describes the derivation of these benchmarks, termed toxicity reference values (TRVs), for the local wildlife of concern near the Ravena facility.

A TRV for a mammalian or avian species is calculated from a "critical" study, reporting the highest dose at which no adverse effects on reproduction, development, or survival are observed. This test dose (TD) or point of departure (POD) might be reduced by one or more uncertainty factors (UF) that reflect the limitations of the database from which the critical study was selected. The resultant POD/UF value can be referred to as a toxicity benchmark or TRV. During risk characterization, TRVs are compared with the estimated exposure (dose) to assess a hazard quotient (HQ) for adverse effects.

ICF reviewed TRVs that have been developed for avian and mammalian wildlife over the past several decades as described in Section J-3.2.3. The best documented of those values were published by EPA in its 1995 Great Lakes Water Quality Initiative Criteria Documents for the

Protection of Wildlife: DDT, Mercury, 2,3,7,8-TCDD, PCBs (EPA 1995a). EPA performed some additional review of the mercury criteria for wildlife in its 1997 Mercury Study Report to Congress (EPA 1997 Volume 5). We therefore began with those values and conducted a literature search for more recent studies that might indicate the need for revision of one or more of those values to lower doses. The TRVs are expressed in units of milligrams[chemical]/kilogram[fresh body weight (BW)]-day (mg/kg-day), micrograms[chemical] /kilogram[BW]-day (µg/kg-day), or µg/g-day (equivalent to mg/kg-day). Where use of dietary concentrations (e.g., ppm Hg in the diet expressed as mg[Hg]/kg diet) might be confused with dose to an animal, BW is specified in the units of dose.

J-4.1.2.1 Calculation of Wildlife TRVs for 2,3,7,8-TCDD

In the Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife, EPA (1995a) calculated wildlife toxicity values for the effects of 2,3,7,8-TCDD on avian and mammalian species. EPA conducted a computer-based and manual search for published studies on the effects of 2,3,7,8-TCDD available in the literature through approximately 1994. As a result of this search, 26 adequately documented reports of dose-response data were identified and summarized in EPA's 1995 GLWQI wildlife criteria report. The derivations of the avian and mammalian TRVs for 2,3,7,8-TCDD are described below.

Avian 2,3,7,8-TCDD TRV

As of 1995, EPA had identified only one comprehensive avian dose-response study on the effects of 2,3,7,8-TCDD that was adequate for the calculation of an avian TRV. Three publications by Nosek et al. (1992a,b, 1993) outline the effects of 10 weekly 2,3,7,8-TCDD i.p. injections on ring-necked pheasants at levels equivalent to 0.0014 µg[TCDD]/kg[BW]-day, 0.014 µg/kg-day, and 0.14 µg/kg-day. Pheasants in the 0.14 µg[TCDD]/kg[BW]-day group showed a significant decrease in egg production and increase in the mortality of embryos from fertilized eggs. These effects were not seen in the other two dose groups. Based on these results, EPA (1995a) concluded that the lowest-observed-adverse-effect level (LOAEL) for fertility and embryo mortality in pheasants was 0.14 µg[TCDD]/kg[BW]-day, and the no-observed-adverse-effect level (NOAEL) was 0.014 µg[TCDD]/kg[BW]-day. EPA selected this study for use in developing wildlife criteria for the GLWQI because it showed meaningful endpoints for long-term (70 days) i.p. administration of 2,3,7,8-TCDD.

For wildlife TRVs in general, EPA considers three uncertainty factors that might need to be applied to a dose from a critical study. Interspecies uncertainty factors (UF_A) are used to develop TRVs for species other than the test species to account for toxicokinetic and toxicodynamic differences between the species. Because gallinaceous birds are thought to be among the most sensitive avian species, EPA set the UF_A for a TRV for belted kingfisher, herring gull, and bald eagle equal to 1. The Nosek et al. (1992a,b, 1993) studies are subchronic; in order to extrapolate the results to chronic effects, a sub-chronic-to-chronic uncertainty factor (UF_S) was set equal to 10. This factor accounts for the rate of steady-state accumulation and whole-body elimination of 2,3,7,8-TCDD. Lastly, the NOAEL was identified by the investigators, indicating that the LOAEL-to-NOAEL uncertainty factor (UF_L) can be set equal to 1.

The NOAEL of 0.014 µg[TCDD]/kg[BW]-day for adverse reproductive effects in pheasants divided by a compound UF of 10 results in an avian TRV of 0.0014 µg/kg[BW]-day for 2,3,7,8-TCDD.

EPA (1995a) conducted a brief sensitivity analysis to illustrate the significance of the assumptions made on the value calculated for the TRV. The first assumption evaluated was that gallinaceous birds are the most sensitive of the avian species. If the UF_A for all representative species were set equal to 3, rather than 1, the avian TRV would equal 0.47 picograms/kg[BW]-day (i.e., 0.00047 µg/kg-day).

No additional data have been identified to indicate the need for a lower avian TRV for 2,3,7,8-TCDD.

Mammalian 2,3,7,8-TCDD TRV

As of 1994, chronic or subchronic studies of the effects of 2,3,7,8-TCDD on mammalian wildlife species were not available. EPA therefore reviewed studies of TCDD toxicity to laboratory mammals. The Agency identified five adequate chronic and subchronic studies of the effects of dietary exposure to 2,3,7,8-TCDD, as described in the GLWQI wildlife criteria document (EPA 1995a). After considering the relevance and adequacy of each study, EPA selected the three-generation rat study using three dietary doses of 2,3,7,8-TCDD conducted by Murray et al. (1979) as the TD (POD) for three reasons: it covered a wide range of reproductive effects; both a NOAEL and a LOAEL were identified by the investigators; and the test species was exposed to the 2,3,7,8-TCDD over three generations.

Sprague-Dawley rats of the f0, f1, and f2 generations were exposed to dietary doses of 0.001, 0.01, or 0.1 µg[TCDD]/kg[BW]-day for 90 days prior to and throughout gestation (Murray et al. 1979). In the f0 generation of the 0.1 µg[TCDD]/kg[BW]-day group, the fertility, litter size, and neonatal survival of pups was significantly lower while the incidence of stillbirths was significantly higher than the control group. In the 0.01 µg/kg-day group, no effect on fertility was evident in the f0 generation, but the f1 and f2 generations exhibited significantly lower fertility, litter sizes, and postnatal body weights and a significantly higher incidence of still-births. At the lowest dose, there was no significant difference between the fertility of experimental and control animals. Murray et al. (1979) concluded that the LOAEL and NOAEL in this study of reproductive endpoints in Sprague-Dawley rats were 0.01 and 0.001 µg[TCDD]/kg[BW]-day, respectively.

The UFs considered in the mammalian analysis are the same factors as considered in the avian analysis. EPA set the UF_L to 1 because the critical study identified a NOAEL. EPA set the UF_S to 1 because the study covered three generations. Given the limited number of mammalian species for which chronic data were available, and considering the high sensitivity of mink to PCBs and other chemicals, EPA determined that the UF_A to extrapolate from rats to mink should be 10.

The NOAEL of 0.001 µg/kg-day for adverse effects on reproductive endpoints in rats divided by a composite UF of 10 results in a mammalian TRV for 2,3,7,8-TCDD of 0.0001 µg/kg[BW]-day.

EPA conducted a brief analysis to assess how sensitive the calculated TRV was to assumptions included in its derivation. Although Murray et al. (1979) concluded that the 0.001 µg[TCDD]/kg[BW]-day dose was a NOEAL for rats, others have reinterpreted the same data and concluded that 0.001 µg/kg-day is actually the LOAEL. Using a LOAEL of 0.001 µg/kg-day and a UFL of 3, the resulting TRV (mammalian) would be 0.00033 µg[TCDD]/kg[BW]-day, a less conservative value. Another assumption EPA evaluated is whether mink are the most sensitive mammalian species. Bowman et al. (1989a,b) had identified NOAEL and LOAEL values for survival to weaning of young Rhesus monkeys of 0.00012 and 0.0059 µg[TCDD]/kg[BW]-day, respectively. If this NOAEL were used as the POD instead, setting the UF_A values for mink and

otter to 1, the resulting mammalian TRV for 2,3,7,8-TCDD would be 0.000012 µg/kg-day, a lower value. In general, however, EPA does not use toxicity results for primates, which often are very sensitive to chemical exposures, as a POD for North American mammalian wildlife species.

No additional data have been identified to indicate the need for a lower mammalian TRV for 2,3,7,8-TCDD.

J-4.1.2.2 Calculation of Wildlife TRVs for Mercury

Wildlife that consume aquatic prey may ingest mercury in its divalent state (Hg+2) and as methyl mercury (MeHg). As illustrated in Exhibit 4-16 above, although TRIM.FaTE predicts that most mercury in the top predators, or "game" fish, is in the form of MeHg, that might not hold true for the smaller forage fish that can comprise a large proportion of the fish diet consumed by wildlife that swallow their prey whole (e.g., kingfishers, mergansers, cormorants). For example, for Nassau Lake near Ravena, TRIM.FaTE predicted that a majority of the Hg in small water column planktivorous fish is present as Hg+2 (Exhibit 4-16). TRIM.FaTE predicts that most (88 percent) of the total Hg present in benthic invertebrates, which are consumed after emergence by swallows, is present as Hg+2.

For humans, EPA has derived separate reference doses for Hg+2 and MeHg. For wildlife, we also consider these two forms of mercury separately.

Avian Methyl Mercury TRV

EPA summarized subchronic and chronic toxicity test results for birds exposed to Hg in its GLWQI wildlife criteria document for mercury (EPA 1995a). The most robust data identifying both LOAEL and NOAEL values from the data examined were the mallard studies by Heinz (1974, 1975, 1976b, and 1979). These studies covered three generations, quantified several different measures of reproductive success, and provided dose-response information even though a NOAEL was not identified.

Heinz (1974, 1975, 1976a, 1976b, 1979) assessed the effects of dietary MeHg in mallards in two sets of experiments. In the first set, Heinz (1974, 1975, 1976a) exposed adult mallards to commercial feed treated with MeHg dicyandiamide at concentrations of 0, 0.5, or 3.0 ppm from 18 months of age through two consecutive breeding seasons. Egg production stopped earlier in the 3 ppm group compared with the 0.5 ppm and control groups (Heinz 1974). The number of normal hatchlings and survival of hatchlings through one week were significantly reduced in the 3.0 ppm group but not in the 0.5 ppm group, compared with the control group. During the second breeding season, most measures of reproduction for hens exposed to 3.0 ppm had improved from the first breeding season and matched control levels, with the exception of normal hatchlings surviving through one week, which remained significantly lower (Heinz 1976a). The LOAEL and NOAEL determined from these studies for the reproductive performance of adult mallards exposed to MeHg in their diet is 3.0 ppm Hg and 0.5 ppm Hg, respectively.

The second series of experiments considered the effects of dietary MeHg on reproduction and behavior in three consecutive generations of mallards. The second season offspring from adult mallards exposed to MeHg dicyandiamide at 0.5 ppm dietary Hg were themselves exposed to 0.5 ppm dietary Hg from 9 days of age through their third reproductive season (Heinz 1976b). The offspring of these birds then were exposed to 0.5 ppm dietary Hg beginning at 9 days of age (Heinz 1979). Both a statistically significant increase in eggs laid outside of the nest box

and decrease in the number of one-week-old ducklings produced were observed in the second generation exposed to dietary concentrations of 0.5 ppm Hg (Heinz 1976b). These trends were observed in the third generation, but were not significant (Heinz 1979); however, these data combined with the results from the second generation were significantly different from controls on both measures (Heinz 1979). These results suggest that MeHg at 0.5 ppm Hg in the diet may be associated with reproductive effects in multigenerational exposure; therefore, a LOAEL of 0.5 ppm Hg for MeHg was inferred. Multiplying the LOAEL by the average food ingestion rate for treated mallards in the second and third generation (i.e., 0.156 kg/kg-day) results in a LOAEL for MeHg of 0.078 mg[Hg]/kg[BW]-day (or µg[Hg]/g[BW]-day), the value used as the POD (EPA 1995a, 1997).

EPA evaluated the three standard UFs, although the Agency revised its estimate of the species-to-species UF_A in its Report to Congress (EPA 1997).

- To extrapolate the results from the mallard to other species of birds in other orders or families, for the GLWQI, EPA concluded that a UF_A greater than 1 would be required (EPA 1995a). Of the avian species for which data were presented in the GLWQI criteria document (EPA 1995a), the mallard and pheasant appear to be the most sensitive. The pheasant study used an exposure duration of only 12 weeks, and the LOAEL was determined to be 0.093 mg[Hg]/kg[BW]-day (Fimreite 1971). With such a short duration study, EPA concluded that the pheasant might be even more sensitive than the mallard. EPA therefore assigned an intermediate value of 3 for the UF_A to extrapolate to other species of birds (EPA 1995a).
- In its Report to Congress, however, EPA (1997) decided to set the UF_A to a value of 1.0 instead of 3. The decision was based on a review of the literature that indicated piscivorous birds are better able to detoxify MeHg than non-piscivorous birds (Dietz et al. 1990), apparently including mallards which consume benthic invertebrates in the former category.
- A UF_S greater than 1 was not necessary because Heinz's studies covered three generations.
- For the GLWQI, EPA set the UF_L to 2 because the LOAEL appeared to EPA to be very near the threshold for effects of Hg on mallards (EPA 1995a). For the Report to Congress, EPA set the UF_L to 3, citing the GLWQI methodology (EPA 1995b).

The resultant GLWQI TRV for mallards, obtained by dividing the NOAEL of 78 µg[Hg]/kg[BW]-day by UF_L of 2 would be 39 µg[Hg]/kg[BW]-day. For the GLWQI, the TRV for other species of birds, for which the UF_A is 3 is 13 µg[Hg]/kg[BW]-day. For its Mercury Report to Congress, the NOAEL of 78 µg/kg-day divided by a total UF of 3 established an avian TRV of 26 µg/kg-day for all avian species (EPA 1997).

Of the two EPA avian TRVs for MeHg, we prefer the GLWQI TRV for two reasons. First, we are not confident that mallards, which consume benthic invertebrates that might have relatively low MeHg in relation to Hg+2 content, have the same higher ability as piscivorous birds to detoxify MeHg. In addition, birds larger than mallards (e.g., merganser, bald eagle) might have, on average, longer lives lives than mallards, which might result in higher tissue concentrations of Hg, particularly in older birds, for the same daily exposure dose per unit body weight. We agree that the identified LOAEL represents a low level of adverse effects, and that a UF_L of 2 to estimate a NOAEL is likely to be adequate. We therefore use the value of 0.13 µg[Hg]/kg[BW]-day as the avian TRV for the Ravena ERA.

ICF conducted a literature review to identify more recent publications with exposure-response information for the effects of MeHg on avian wildlife. Recent studies of common loons in Maine indicate that MeHg concentrations in fish of 0.05 µg[Hg]/g[diet wet weight] or less pose "low risks" and of 0.05 to 0.15 µg[Hg]/g[diet] pose "moderate risks" of adverse effects on reproduction (Evers et al. 2004). Evers et al. (2004) provided some evidence that fish concentrations of 0.15 µg[total Hg]/g[fish wet weight (ww)] were roughly associated with loon blood total Hg concentrations of 3 µg/g, which was a LOAEL associated with lowered reproductive success in the field, although the criteria for a "LOAEL" were unclear. For adult female loons weighing 4.7 kg, and a fish ingestion rate of 15 percent of the adult body weight estimated from Nagy's (1987) allometric equation for non-passerine birds, a value which is slightly less than the fish ingestion rate of 20 percent of body weight measured for growing 35-day-old loon chicks by Fournier et al. (2002) using doubly labeled water, 0.15 µg[Hg]/g[fish ww] would correspond to an exposure dose of 0.11 µg[Hg]/g[BW]-day. Using a food ingestion rate of 20 percent of body weight daily, the exposure dose would be 0.14 µg[Hg]/g[BW]-day (Evers et al. 2004). Using a UF_L of 3 to extrapolate from a possible LOAEL to a NOAEL, a UF_A of 3 for inter-species variation in sensitivity, and UF_S of 1 (field exposures are of the duration of interest), a final TRV for birds based on field data from Evers et al. (2004) would be 0.012 to 0.016 µg[total Hg]/g[BW]-day. Therefore, the MeHg TRV of 0.013 µg[Hg]/g[BW]-day established by EPA in 1995 is consistent with the more recent data.

Mammalian Methyl Mercury TRV

From its review of available subchronic and chronic toxicity studies on the effect of MeHg on mammalian species for its GLWQI, EPA selected a NOAEL for MeHg of 0.16 mg [Hg]/kg[BW]-day as the POD (EPA 1995a). The NOAEL is from a 93-day study of MeHg chloride administered in the diet to mink (Wobeser et al. 1976b).

Wobester et al. (1976b) exposed adult female mink to dietary concentrations of MeHg chloride of 1.1, 1.8, 4.8, 8.3, and 15.0 ppm Hg for up to 93 days. Clinical signs of Hg intoxication (anorexia and ataxia) were observed in all mink exposed to concentrations of 1.8 ppm Hg and greater. All five of the mink exposed to 1.8 ppm Hg developed ataxia: two of the mink died, and the remaining three were killed following onset of symptoms for examination. The investigators determined that dietary Hg concentration was directly related to the time of the onset of toxic effects and death. Pathological alterations in the nervous system were observed at the 1.1 ppm Hg in the diet, but additional clinical symptoms were absent; therefore, EPA initially concluded that this dietary concentration would not have clear implications for population-level effects on mink (EPA 1995a). Using the captive mink body weight of 1.0 kg and food ingestion rate of 0.15 kg/day, the dietary concentration of 1.1 ppm Hg was converted to a NOAEL of 0.16 mg[Hg]/kg[BW]-day (or µg[Hg]/g[BW]-day).

After obtaining the doctoral thesis of Wobeser (Wobeser 1973), EPA concluded that the effects observed at the 1.1 ppm concentration in the diet, lesions of the central nervous system and axonal degeneration, were sufficiently adverse to consider that exposure to be a LOAEL; EPA used data from the first part of the study to identify a NOAEL of 0.33 ppm (EPA 1997). In addition, EPA recalculated the doses using data on the weights of female mink and kits used in the experiments, for a NOAEL for MeHg for mink of 55 µg[Hg]/kg[BW]-day.

In order to extrapolate the results from this study to a chronic TRV for mink, EPA evaluated two UFs: a subchronic-to-chronic factor and an interspecies uncertainty factor. A third UF, to estimate a NOAEL from a LOAEL, was not needed.

- Wobester et al. (1976b) had concluded that the pathological alterations observed at the 1.1 ppm dietary concentration after 93 days would have resulted in distinct clinical signs of toxicity had the exposure period been longer. In a prior study, Wobester et al. (1976a) determined that the NOAEL for MeHg for adult mink was 0.05 mg[Hg]/kg[BW]-day over a 145-day dietary exposure period. This NOAEL is approximately a factor of 3 less than the 93-day NOAEL of 0.16 mg[Hg]/kg[BW]-day for MeHg discussed above. Considering a mink's lifetime of 6 or 7 years, 145 days represents a relatively short subchronic exposure. Therefore, for the GLWQI, EPA set the UF_S to 10 (EPA1995a). When using a lower dose for the NOAEL, however, EPA set the UF_S to 3 for the Report to Congress (EPA 1997).
- The UF_A was set to 1 for mink because mink was the test species (EPA 1995a, 1997).

The resultant MeHg TRV for mink, obtained by dividing the NOAEL for mink by the product of the UFs discussed above was 0.016 and 0.018 mg[Hg]/kg[BW]-day for the GLWQI (EPA 1995a) and the Report to Congress (EPA 1997), respectively. Because we are concerned that the 93-day exposure is short compared to the lifetime of a mink, and because MeHg is only slowly eliminated and therefore tends to increase in concentration in older animals (EPA 1997), we prefer to retain the more conservative UF_S to 10 from the GLWQI to use with the more conservative NOAEL of 55 µg[Hg]/kg[BW]-day to estimate a TRV for mink of 1.8 µg[Hg]/kg[BW]-day.

ICF conducted a literature review to identify more recent publications with exposure-response information for the effects of MeHg on mammalian wildlife. While many recent studies focus on the relationship between environmental Hg contamination and mammalian wildlife total Hg tissue concentrations in North America (e.g., Halbrook et al. 1994, Mierle et al. 2000, Thompson 1996, Yates et al. 2004, Wolfe et al. 1998), we did not identify any new data linking exposure doses to adverse effects at lower levels than the mink study used by EPA in 1995 and 1997.

Avian and Mammalian TRVs for Divalent Mercury

We did not identify any TRVs developed for Hg+2 for avian or mammalian wildlife; concern and research have largely focused on MeHg or, in some cases, total Hg assuming most of it is methylated.

Mammalian TRV for Divalent Mercury

To determine whether we should expend the effort to derive a TRV for Hg+2, we first compared the human reference dose (RfD) for Hg+2 (i.e., 3 µg/kg-day) to the human RfD for MeHg, (i.e., 1 µg/kg-day). The two RfDs were derived from different health endpoints using substantially different (UFs) as described briefly below.

RfD for Mercuric Chloride. The human RfD for chronic oral exposure to mercuric chloride (essentially Hg+2) is 3E-4 mg[Hg+2]/kg[BW]-day based on autoimmune effects (i.e., "...formation of mercuric-mercury-induced autoimmune glomerulonephritis") (EPA IRIS). Dose conversions for three studies of the Brown Norway Rat (Druet et al. 1978; Bernaudin et al. 1981; Andres 1984) were used to derive the RfD. The conversions were a factor of 0.739 to convert the weight of HgCl2 to Hg+2, a factor of 1 for the different routes of exposure (i.e., an assumption of 100 percent absorption efficiency by both the subcutaneous (s.c.) and oral routes of exposure), and a factor to estimate an average daily exposure from the days per week injections were administered. The three identified LOAELs, as converted, were 0.226, 0.317,

and 0.633 mg[Hg+2]/kg[BW]-day. A composite uncertainty factor (UF) of 1000 was applied to the LOAEL determine the RfD. The UF included a factor of 10 for conversion from LOAEL to NOAEL, a factor of 10 for use of subchronic studies, and a factor of 10 for both animal-to-human extrapolation (interspecies variation) and sensitive human populations (intraspecies variation).

RfD for Methyl Mercury. The human RfD for chronic oral exposure to MeHg is 1E-4 mg [MeHg]/kg[BW]-day based on developmental neuropsychological impairment (EPA IRIS). (We note that the molecular weights of MeHg and Hg are similar.) Human epidemiological studies were used to derive the RfD (Grandjean et al. 1997). Surrogate data on maternal daily dietary intake were used for the observed developmental effects in children exposed *in utero*. Maternal daily dietary intake rates were calculated primarily from concentrations in cord blood. Almost all of the Hg in cord blood was MeHg. A benchmark-dose (BMD), not NOAEL/LOAEL, approach was used to identify a POD within the observed range of response in the critical study of neurological impairment in children. The lower 95 percent confidence limit of the BMD in maternal blood corresponding to a 5 percent response above the control ($BMDL_{05}$) ranged from 46 ppb to 79 ppb. This blood concentration corresponded to a range of maternal daily intakes of 0.858 to 1.472 µg [MeHg]/kg[BW]-day. A composite uncertainty factor of 10 was applied to the estimated maternal daily intake. The UF included a factor of 3 to account for pharmacokinetic variability within humans and uncertainty in estimating an ingested mercury dose from cord-blood mercury concentration and a factor of 3 for pharmacodynamic variability among humans.

A comparison of the derivation of the human RfDs for Hg+2 and MeHg indicates that although similar in magnitude, they are based on substantially different studies, health endpoints, and types of uncertainty. The chronic oral RfD for Hg+2 includes a composite UF of 1000, whereas the UF is only 10 in the RfD derivation for MeHg. The Hg+2 RfD is based on a LOAEL (as opposed to a NOAEL), animal (as opposed to human) data, and subchronic exposures. We conclude that comparing the human RfD for Hg+2 to that for MeHg does not provide an adequate basis by which to compare the chronic toxicity of Hg+2 to MeHg in wildlife.

Comparison of Chronic Organic and Inorganic Hg Toxicity in Rats. We identified one animal study that provides sufficiently similar experiments by which to compare the relative chronic toxicity of organic and inorganic mercury. Fitzhugh et al. (1950) compared the toxicity of dietary organic (phenyl mercuric acetate) and inorganic (mercuric acetate) Hg to rats exposed for up to two years. For both experiments, groups of rats were exposed at 0 (control), 0.1, 0.5, 2.5, 10, 40, or 160 ppm Hg in the diet. The organic form reduced growth in males at 10 ppm Hg in males (40 ppm reduced growth in both males and females); survival was reduced only in the 160 ppm group. The inorganic form reduced growth in males exposed at 160 ppm, but no other adverse effects were observed. The LOAEL for reduced growth in males, therefore, was 160 ppm Hg in the diet as inorganic mercury and 10 ppm Hg as organic mercury, suggesting that the organic form was more toxic than the inorganic form for chronic exposures.

Fitzhugh et al. (1950) calculated the doses associated with 10, 40, and 160 ppm diets to be 0.15, 0.6, and 2.4 mg[Hg]/rat-day, but did not report body weights for the different groups, did not distinguish males from females, and did not consider the reduced body weight of rats in groups that exhibited reduced body weight. Assuming a body weight of 0.175 kg, and using data presented by the investigators where possible, EPA (1995a) calculated the doses as 0.56, 2.2, and 14 mg[Hg]/kg-day for these studies (EPA 1995a). Thus, the LOAEL and NOAEL for growth in rats (male) for organic Hg are 2.2 and 0.56 µg/g-day, respectively; and a LOAEL for growth in male rats and a NOAEL for reproduction and development for inorganic Hg is14 µg/g-

day for this study. Thus, the LOAEL for inorganic Hg is between 4 and 6 times higher than the LOAEL for the same effect (reduced growth in males) for organic mercury.

Limitations of this study include its age, that the sensitive endpoints of reproduction and neurodevelopment (for organic Hg) and nephrotoxicity (for Hg+2) were not assessed, and that the chemical form of organic mercury may or may not be absorbed and distributed to the body as is MeHg. In addition, data on the final tissue concentrations of Hg were not reported; whether final tissue concentrations of total Hg were similar or lower for the Hg+2 group than for the MeHg groups is not known. Given the higher clearance rate of Hg+2 than MeHg, it is possible that the lower LOAEL for the organic Hg than for Hg+2 results in part from its accumulation in tissues over time.

Derivation of Mammalian TRV for Hg+2 for Autoimmune Glemerulonephritis. Using the rat Hg+2 toxicity studies cited in the derivation of the human RfD, we identify a POD for mammalian wildlife as the geometric mean of the three LOAELs identified for autoimmune glomerulonephritis in rats of 0.3 mg[Hg+2]/kg[BW]-day or 300 µg[Hg+2]/kg[BW]-day. We propose a UF_L of 10 to estimate a NOAEL for a population-level effect from a LOAEL for a sublethal individual effect that might affect reproductive success or survival. In addition, we applied UF_A of 3 is applied to account for toxicodynamic differences among mammals. Toxicokinetic differences among mammalian wildlife species for Hg+2 should be estimated on the basis of metabolic rate (body weight to the ¾ power) relative to the metabolic rate of the Brown Norway rat, because Hg+2 is readily eliminated by animals (in contrast to MeHg). To extrapolate from a rat weighing 0.175 kg to a 1 kg mink, the POD is multiplied by a factor of 1.55 (i.e., $0.175^{-0.25}$ / $1^{-0.25}$) providing a final mink TRV of 16 µg[Hg+2]/kg[BW]-day. The mink TRV derived as described above for Hg+2 is about 9 times higher than the mink TRV for MeHg of 16 µg[Hg+2]/kg[BW]-day.

Avian TRV for Divalent Mercury

We did not identify any chronic toxicity values for dietary Hg+2 exposure for birds or appropriate data by which to estimate the chronic toxicity of Hg+2 by comparison with MeHg, although acute toxicity tests indicate similar acute toxicities (EPA 1995a, Table 2-5). Given that it is unlikely that chronic exposure to Hg+2 is as toxic as chronic exposure to MeHg in birds owing in part to the more rapid elimination of Hg+2 than MeHg, we judge that the TRV for Hg+2 can be at least 2 times the MeHg TRV for birds smaller than mallards and at least 5 times the MeHg TRV for birds larger than mallards, such as bald eagles and common mergansers.

Summary

A summary of the wildlife TRVs used in this risk assessment is provided in Exhibit 4-26.

Exhibit 4-26. Summary of Wildlife TRVs (µg[chemical]/kg[BW]-day)

Chemical	Avian Values			Mink Values		
	POD (µg/kg-day)	UF_{Tot}	TRV (µg/kg-day)	POD (µg/kg-day)	UF_{Tot}	TRV (µg/kg-day)
2,3,7,8-TCDD	14 E-03	10	1.4 E-03	1.0 E-03	10	0.10 E-3
Methyl Mercury	78	6	13	55	30	1.8
Divalent Mercury	N/A	N/A	Smaller birds: 26 Larger birds: 65	300	30/1.55 = 19	16

N/A = Not applicable.

J-4.1.3 Risk Characterization

In Section J.3.2.4, a two-stage approach was proposed to characterize ecological risk of MeHg and dioxin exposure. In the first step, HQs are calculated as an indicator of potential adverse effects at the level of the individual organism. For those species or locations where HQ values exceeded 1.0, a second stage analysis is conducted to provide a preliminary evaluation of potential population-level effects.

For the first stage of the risk characterization, hazard quotients were calculated by dividing the total exposure doses in Exhibit 4-17 through Exhibit 4-25 by the applicable avian or mammalian TRVs in Exhibit 4-26. The resulting HQs for MeHg, Hg+2, and TCDD are presented in Exhibit 4-27, Exhibit 4-28, and Exhibit 4-29, respectively.

Exhibit 4-27. Hazard Quotients for Wildlife Exposure to Methyl Mercury [a]

Wildlife Species	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Tree Swallow	0.605	0.004	0.005	0.006
Common Merganser	**1.304**	0.004	0.006	0.005
Bald Eagle	0.634	0.002	0.003	0.003
Mink	**3.919**	0.014	0.021	0.020

[a] Hazard quotients highlighted in blue and **bold** indicate exceed the hazard quotient threshold of 1.

Exhibit 4-28. Hazard Quotients for Wildlife Exposure to Divalent Mercury [a,b]

Wildlife Species	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Tree Swallow	**2.37**	<1	<1	<1
Common Merganser	0.40	<1	<1	<1
Bald Eagle	0.04	<1	<1	<1
Mink	0.98	<1	<1	<1

[a] Hazard quotients highlighted in blue and **bold** indicate exceed the hazard quotient threshold of 1.
[b] The HQs for Hg+2 are likely to be less than 1.0 at water-bodies other Ravena Pond given that exposure doses are more than two orders of magnitude lower for wildlife consuming prey from those water bodies.

Exhibit 4-29. Hazard Quotients for Wildlife Exposure to 2,3,7,8-TCDD [a,b]

Wildlife Species	Water Body			
	Ravena Pond	Alcove Reservoir	Nassau Lake	Kinderhook Lake
Tree Swallow	0.01	0.00004	0.0001	0.0001
Common Merganser	0.70	0.001	0.01	0.002
Bald Eagle	0.77	0.001	0.01	0.004
Mink	**4.27**	0.003	0.03	0.01

[a] Exposure doses are based on the estimated 95-percent UCL dioxin emission rates.
[b] Hazard quotients highlighted in blue and **bold** indicate exceed the hazard quotient threshold of 1.

All HQs for all species are below 1.0 for all chemicals with a few exceptions. For MeHg, an HQ of 1.3 was calculated for the common merganser in Ravena Pond. For Hg+2, an HQ of 2.4 was calculated for the tree swallow in Ravena Pond. Finally, for 2,3,7,8-TCDD, an HQ of 4.3 was calculated for the mink in Ravena Pond. No hazard quotients were found to be greater than at water bodies other than Ravena Pond.

Ravena Pond has a surface area of only 0.02 km^2 and a shoreline of approximately 0.8 km. At that size, at most a few pairs of swallows, one pair of mergansers, and one female mink might forage there each season. De Graaf et al. (1981) reported tree swallows breeding at a density of about 0.0007 pairs/ha or 0.7 pair/km^2. Typical breeding densities for common mergansers are 0.07 to 0.11 breeding pairs/ km^2, or about 1 pair per 10 km^2 of habitat containing many suitable bodies of water (Cadman et al. 1987, Erskine 1987, and Ross 1987 as cited in Mallory and Metz 1999). The density of female mink in the vicinity of a Michigan river was reported to be 0.006 per hectare or 0.6 per km^2 (Marshall 1936). Along a Montana river, Mitchell (1961) reported densities of mink of between 0.03 and 0.085 individuals per hectare of area near shore or 3 to 8.5 individuals per km^2. Estimating the mink density per unit river shoreline instead of per unit area, Marshall (1936) reported the mink density along the Michigan river to be 0.6 mink/km.

Populations of piscivorous and insectivorous wildlife are not expected to be adversely affected by MeHg, Hg+2, and dioxin attributable to emissions from the facility. Adverse effects on the reproductive success of a single pair or female of a non-endangered avian species should not result in any population-level effects. These results indicate that emissions of neither Hg or 2,3,7,8-TCDD from the Ravena facility are expected to result in adverse toxic effects on the selected wildlife species. As discussed in Section J-2, the wildlife species chosen for the Ravena ERA are expected to be the most highly exposed species likely to be found near the Ravena facility.

J-4.1.4 Uncertainties in Ravena ERA Related to Mercury and Dioxin

This section identifies uncertainties and limitations of the data and approaches used for the Ravena ERA for MeHg, Hg+2, and 2,3,7,8-TCDD. Where possible, we qualitatively identify the likely direction in which these limitations may affect the relevant results.

- As discussed in problem formulation (Section J-2), some aspects of the analysis scope were determined based on screening analyses or other decisions associated with the HHRA. For example, the contaminants of concern for both the HHRA and Ravena ERA were selected based on a *de minimis* emissions screen that identified mercury and dioxins as the persistent and bioaccumulative chemicals of highest potential concern from a human health perspective. Although human health criteria were used to select the HAPs, we determined, based on release quantities, chemical characteristics, and toxicity to ecological receptors, that these pollutants, as well as HCl, are the pollutants most likely to pose ecological risks. However, it is possible that a systematic ecological screening analysis may have identified additional HAPs to include in the ERA.
- The Ravena ERA does not address or include background concentrations of total Hg, MeHg, or 2,3,7,8-TCDD. It also does not consider the impact of other environmental pollutants and sources (e.g., PCB contamination in the nearby Hudson River) on the baseline condition of the wildlife receptors.
- The assessment endpoints for the ERA include piscivores and insectivores that were chosen because they are likely to be the most highly exposed receptors given their

feeding habits and the high bioaccumulation potential for MeHg and 2,3,7,8-TCDD. However, it is possible that other locally present species that were not considered (e.g., because their local presence has not been documented, such as river otter, or because relevant data to estimate values for their body weights, diets, or food ingestion rates is not readily available) might be more highly exposed.

- The Ravena ERA used fate and transport modeling results performed for the HHRA, although the aquatic food webs were constructed with both the HHRA and ERA in mind. Details of the TRIM.FaTE fate and transport modeling methodology and its potential limitations are discussed in Appendix I.
- The TRIM.FaTE aquatic food web models for the four water bodies are based on information obtained from NYS DEC and other sources. Like any model, the aquatic foodweb models are fairly gross simplifications that may not accurately estimate exposure concentrations.
- Although the TRIM.FaTE aquatic foodweb models were conceived using field data on the composition and relative abundance of fish species in local aquatic communities, the original model does not consider the influence of harvesting fish on removal of chemicals from the aquatic ecosystem. For the two lakes and reservoir, it is unlikely that this limitation is of significance. For Ravena Pond, however, the mean and 90th percentile human harvest rates alone are not sustainable or not possible, and smaller harvest rates plus predation by any wildlife species are likely to remove sufficient chemical from the system to significantly lower the chemical concentration in the benthic omnivores and water column carnivores.
- Mercury speciation as predicted by TRIM.FaTE indicates a higher proportion of Hg+2 than MeHg in water column planktivores. If this is an overestimate of Hg+2 and an underestimate of MeHg in that compartment, the exposures to MeHg may be somewhat underestimated for mink and mergansers. This bias is too small to affect our conclusions, however.
- Emission rates for mercury are based on data from the 2002 NEI (taking into account any recent changes made as a part of the SAB analyses). These data are not necessarily consistent with current emissions rates. The NEI does not include TCDD emissions data for the Ravena facility. Therefore, emissions factors were used to estimate mean and 95-percent UCL 2,3,7,8-TCDD emission rates. The 95-percent UCL emission rates were used in the ERA. Ecological exposures and risks estimated based on the 95-percent UCL emission rates may be overly conservative.
- Exposure factors for which values were estimated or assumed for the wildlife species include body weight, diet composition, and feeding rate. These data were obtained from the literature. Published values, particularly for diet composition, can vary substantially depending on location, time of year, sex, or other factors. In choosing assumptions using the available literature, we considered several factors, including the sample size, distance or latitude difference from the Ravena facility, and study methods (e.g., using captive vs. free-living animals) with the general goal of using the most robust and representative data. Where temporal variations were evident, assumptions were based on average annual values if possible. Adult body weights were averages for adult males and females. Because some species display significant sexual dimorphism, the body weights and, to a lesser extent, the food ingestion rates are under- or over-estimates for each sex.
- ICF used diet composition information from the literature to make assumptions about the percentage of each species' diet obtained from each of the nine food types included in the TRIM.FaTE aquatic food web model. In making these assumptions, ICF judged the closest match between the reported prey species/types and the biotic

compartments included in the model based on the diet and size of the prey species/types. This limitation may cause over- or under-estimation of actual ecological exposure levels. In some cases, we made conservative assumptions to facilitate modeling exposures, such as assuming that mink and bald eagles take 100 percent of their prey from aquatic ecosystems, assuming year-round residency for the migratory birds (see next bullet), assuming all prey come from a single water body near the Ravena facility, and using a high-end estimate of consumption of top predatory fish by bald eagles.

- Because chronic exposures are of concern, with both 2,3,7,8-TCDD and MeHg accumulating in tissues over time, ingestion rates for free-living adult animals are used and no attempt has been made to define ingestion rates for nestling birds fed by their parents, by adult female birds laying eggs, or by pregnant or lactating mink. Note that while mink are resident year-round, the mergansers and bald eagles migrate further southward to follow the open (not ice-covered) water, while the swallows migrate to South America for the winter. Thus, the bird species would not be exposed year-round to dioxins and methyl mercury that originated with the Ravena facility. Given the global nature of contamination of aquatic ecosystems with dioxins and Hg, however, it is likely that the birds will be exposed year-round to these chemicals.

The only issue that might affect our conclusions of negligible ecological risks from Hg and TCDD emissions from the Ravena facility is omission of existing background concentrations of these chemicals, particularly, Hg.

J-4.2 Results for HCl

This section discusses the facility-ranking analysis (Section J-4.2.1) and the indirect ecological effects assessment for HCl (Section J-4.2.2).

J-4.2.1 Results for Facility-Ranking Analysis

The preliminary facility ranking according to ecological hazards was based on scores for three factors (Section J-4.2.1.1). For the facilities with the lowest scores (i.e., highest potential for ecological hazards), proximity to specially valued ecosystems was assessed (Section J-4.2.1.2) to determine the facilities for which to examine readily available lines of evidence for indirect ecological effects that might result from ongoing emissions of HCl from the facility.

J-4.2.1.1 Preliminary Facility Ranking

ICF conducted an initial ranking of all Portland Cement facilities that emit HCl based on three indicators of ecological risk: (1) background acid deposition (regional pH of rainfall;

ATTACHMENT J-1 Exhibit 1), (2) surface water alkalinity (

ATTACHMENT J-1 Exhibit 2) as an indicator of both surface water and soil alkalinity of a geographic area, and (3) annual HCl emissions reported for the facility. Please refer to Section J-3.3.1 for a detailed discussion of these indicators. We assigned background acid deposition (an indicator of potential ecosystem susceptibility), surface water alkalinity (an indicator of inherent ecosystem susceptibility [sensitivity] to acid deposition), and annual HCl emissions (an indicator of potential exposure) scores from 1 to 5, with 1 indicating greatest potential for ecological effects and 5 indicating lowest potential for ecological effects. These three scores were multiplied for each facility to generate preliminary facility-specific ecological hazard scores.

Ecosystem susceptibility scores (both rainfall pH and surface water alkalinity), facility emission scores, and preliminary ecological hazard scores are presented in

ATTACHMENT J-1 Exhibit 3. Background acid deposition and annual HCl emission scores for the facilities were relatively evenly distributed from 1 through 5, allowing a reasonable ranking of facilities based on the product of those two scores. The vast majority of facilities, however, were located in areas of high surface water alkalinity (i.e., > 400 meq/L) indicating a high buffering capacity. Few of the facilities that reported HCl emissions were located in areas identified by EPA as having lower surface water alkalinity, as shown in

ATTACHMENT J-1 Exhibit 4. Only three facilities were located in areas with alkalinity less than 50 meq/L, and two of those are in Puerto Rico and not included in this analysis of the conterminous United States. Thus, the surface water alkalinity score, which we used as an indicator of both aquatic and local terrestiral ecosystems' abilities to resist changes in pH with acid deposition, was a poor discriminator among Portland Cement facilities.

Four Portland Cement facilities shared the lowest preliminary ecological hazard score of ten, indicating that these facilities may have the greatest potential to cause adverse ecological effects. The four highest hazard (lowest score) facilities are located in Albany County, New York; Hernando County, Florida; Carroll County, Maryland; and Dorchester County, South Carolina. Each of these facilities had background acid deposition scores of 1 (with regional pH of rainfall in the range of 4.5 to 4.7), alkalinity scores of 5 (with surface water alkalinity measurements exceeding 400 meq/L), and facility emission scores of 2 (with total emissions between approximately 50 and 180 tons per year).

Nine additional Portland Cement facilities had a preliminary hazard score of 20 or less. A facility in Santa Cruz County, California, had the only alkalinity score of 1 (less than 50 meq/L). The ecosystems in the vicinity of this facility are likely to have very limited acid buffering capacity,

and soils and surface waters might show reduced pH with added HCl deposition from the facility. This facility, however, is in a region of low background acid deposition (score of 5) and is in the second lowest quintile of annual HCl emissions (emissions score of 4).

J-4.2.1.2 Refined Facility Ranking

To further refine the facility ranking to focus assessment of evidence of ecological effects, facilities were selected with preliminary ecological risk scores of 20 or less (the top thirteen facilities) to conduct a proximity analysis. Nearby ecologically valued areas were identified for the top thirteen facilities. Ecologically valued environments were described in Section J-3 and included reservoirs, rivers, lakes, and parks and preserves. Very large water bodies (e.g., the Great Lakes and major rivers), which are not expected to show changes in pH from localized HCl emissions, were excluded from the analysis. A proximity score was assigned to each facility based on the square root of the distance between the facility and the valued environment. The proximity score was rounded to one significant digit and stopped at a top score of 5 (any separation greater than 25 km was assigned a proximity factor of 5).

A final hazard score was determined by calculating the product of the background rainfall pH score, the alkalinity score, the facility emission score, and the proximity score for each of the top thirteen facilities. Final hazard scores for each facility are reported in Exhibit 4-30.

Exhibit 4-30. Final Hazard Scores for Top Thirteen Portland Cement Facilities Emitting HCl

Facility	Facility Location	Rainfall pH Score	Alkalinity Score	Emissions Score	Preliminary Hazard Score	Closest Sensitive Environment	Distance to Sensitive Environment (km)	Proximity Score	Final Hazard Score
PTC_NEI34931	Albany County, NY	2	5	1	10	Alcove Reservoir	11.30	3	30
PTC_NEI26327	Hernando County, FL	2	5	1	10	Withlacoochee State Forest	11.15	3	30
PTC_NEIMIB1559	Charlevoix County, MI	3	5	1	15	Lake Charlevoix	4.42	2	30
PTC_NEI51435	La Salle County, IL	3	5	1	15	Illinois River	5.71	2	30
PTC_NEI12018	Alpena County, MI	3	5	1	15	Elbow Lake	11.37	3	45
PTC_NEIPAT$1626	Lawrence County, PA	1	5	3	15	Evans Lake	9.76	3	45
PTC_NEI33394	Carroll County, MD	2	5	1	10	Patuxent River State Park	24.83	5	50
PTC_NEISC0351244	Dorchester County, SC	2	5	1	10	Lake Moultrie	28.75	5	50
PTC_NEIMO0990002	Jefferson County, MO	3	5	1	15	Moredock Lake	17.49	4	60
PTC_NEI51352	La Salle County, IL	4	5	1	20	Illinois River	6.41	3	60
PTC_NEI2CA151186	Santa Cruz County, CA	5	1	4	20	Big Basin Redwoods State Park	10.97	3	60
PTC_NEI31319	Clark County, IN	2	5	2	20	Quick Creek Reservoir	38.52	5	100
PTC_NEI7255	Northhampton County, PA	2	5	2	20	Beltzville Lake	30.03	5	100

Four Portland Cement facilities shared the lowest final hazard score of 30 (with proximity scores of 2 or 3), indicating that these facilities may have the greatest potential to cause harm to local valued ecosystems due to indirect effects of HCl deposition to soils or surface waters. These four facilities are located in Albany, New York (the Ravena facility); Hernando County, Florida; Charlevoix County, Michigan; and La Salle County, Illinois.

Calculation of the final hazard score suggests a different ranking of the thirteen facilities than the preliminary hazard scores indicated. The facilities in New York and Florida remained in the top four, but the inclusion of the proximity score removed facilities in Maryland and South Carolina from the top four and replaced them with facilities in Michigan and Illinois.

J-4.2.2 Indirect Ecological Effects Assessment

The localized indirect ecological effects of HCl released from Portland Cement facilities, if any, would be mediated through changes in the pH of surface waters or the top layers of soil (plant root zone). We therefore attempted to identify pH benchmarks associated with ecological effects for surface waters (Section J-4.2.2.1) and soils (Section J-4.2.2.2).

J-4.2.2.1 Benchmarks for Surface Waters

Several water characteristics are related to the potential for acid loading to cause adverse effects: pH, alkalinity, and water hardness. We discuss each below to clarify how they might be used in assessing risks to aquatic communities.

pH

For freshwaters, EPA (1976, still current as of 2008) has recommended that pH be no lower than 6.5. "pH" equals the negative of the log (base 10) of hydrogen ion (H^+) activity in the water. The materials in natural waters that most influence pH include carbon dioxide (CO_2), carbonic acid (H_2CO_3), bicarbonate ion (HCO_3^-), and carbonate ions (CO_3^-). The pH of surface waters affects the toxicity of many chemical compounds to aquatic life by changing the degree of dissociation for weak acids or bases; the undissociated compounds generally are more bioavailable than are the hydrophilic dissociated ions. In general, one cannot identify a "threshold" pH for adverse effects on aquatic life because of the influence of pH on the toxicity of other chemicals that may or may not be present. Typically, water is not directly lethal to fish at pH values as low as 5; however, several common water pollutants are more toxic at lower pH.

The European Inland Fisheries Advisory Commission (EIFAC 1969) recommended that pH in the range of 5.0 to 6.0 was unlikely to cause adverse effects to freshwater fish unless the concentration of free CO_2 in water was higher than 20 mg/L (or if excess iron salts were available). The Commission observed that fish in waters with pH in a range from 6.0 to 6.5 were unlikely to be harmed unless the concentration of free CO_2 was higher than 100 mg/L, which it often can be. The Commission concluded that pH values between 6.5 and 9.0 should not harm fish, although the toxicity of other chemical contaminants might be enhanced within this range (EIFAC 1969, EPA 1976).

In establishing its lower criterion for pH in freshwaters of 6.5, EPA (1976) summarized additional studies of freshwater organisms exposed to different pH levels. In a 13-month (1-generation) exposure study using fathead minnows, Mount (1973) found fish deformities at pH values of 4.5 and 5.2 and reduced egg production and hatchability at a pH of 6.6 compared with the control fish at pH 7.5. Bell (1971) examined the responses of two species of caddisfly, four species of

stonefly, two species of dragonfly, and one species of mayfly nymphs to water at different pH values. He found 50 percent mortality in mayfly nymphs in water at a pH of 5.4 for 30 days. He also found the "50-percent-emergence" effect level to be as high as pH 6.6 for some species. A pH of 6.5 appears, therefore, to be a reasonable screening value for adverse effects on freshwater aquatic animals. ICF did not attempt to identify additional literature on the effects of pH on aquatic organisms.

Alkalinity

Another water quality parameter relevant to interpreting acid loading to surface waters is alkalinity as described earlier. Water alkalinity is the sum of the components in water that tend to elevate water pH above a value of 4.5. Such materials include carbonates, bicarbonates, phosphates, and hydroxides (EPA 1986). Some of the materials that contribute to alkalinity (e.g., carbonates) reduce the toxicity of metals in surface waters by complexing with the metals so that they are no longer bioavailable. Alkalinity is measured by titration with a standardized acid to a pH value of 4.5, and it generally is expressed as mg/L equivalents of calcium carbonate ($CaCO_3$). Photosynthesis by aquatic plants generates dissolved carbon dioxide, which can acidify waters with limited buffering capacity (low alkalinity and high acidity).

The National Academy of Sciences' National Academy of Engineering (NAS/NAE 1974) recommended that natural alkalinity not be reduced more than 25 percent, and for areas with naturally low alkalinity (e.g., below 20 mg/L as calcium carbonate), alkalinity should not be reduced further. In 1976, EPA established a lower bound criterion for alkalinity in freshwaters of 20 mg/L as calcium carbonate (EPA 1976); however, that criterion was replaced with a narrative statement in 1986 (EPA 1986).

Water hardness

Water hardness also is expressed in units of mg/L as calcium carbonate. However, water hardness reflects polyvalent metallic ions dissolved in water, primarily calcium and magnesium in fresh waters, but also iron, strontium, and manganese. Based on human water uses, classification of water hardness into soft (0 to 75 mg/L $CaCO_3$), moderately hard (75 to 150 mg/L), hard (150 to 300 mg/L), and very hard (300 mg/L or higher) (Sawyer 1960; EPA 1986). Limestone is a natural source of water hardness.

Freshwater hardness can be divided into the carbonate and non-carbonate fractions. The carbonate fraction is chemically equivalent to the bicarbonates present, and so carbonate water hardness is considered equal to alkalinity. In general, the toxicity of metals to aquatic organisms is reduced at higher levels of carbonate hardness/alkalinity (EPA 1986).

J-4.2.2.2 pH Benchmark for Soils

The pH of soils across the United States varies both regionally and locally due to a wide variety of contributing factors. EPA does not have a soil criterion for pH of which we are aware, nor have we identified a "threshold" for "adverse effects" established by any other agency. Because of long-term adaptation of plants and soil communities to more acidic conditions in some regions of the country, in some types of habitats, and with some soil types, there is no single soil pH value that could serve as an ecotoxicity benchmark in all areas or all regions. Nonetheless, we examined literature on soil pH associated with agricultural and horticultural practices to identify a pH benchmark to assist in screening Portland Cement facilities for the potential to cause indirect ecological effects of HCl deposition.

There are many different methods of measuring soil pH, some of which provide different results. Compared with shaking a soil sample with water to measure pH, the calcium chloride ($CaCl_2$) extraction method tends to result in pH estimates of 0.5 to 0.8 pH units lower.[3] The $CaCl_2$ method does not provide an actual soil solution pH, but rather a result that depends on soil solution pH and hydrogen ions that are readily available through cation exchange. (Cation exchange capacity is discussed in greater detail at the end of this section.) Most measurements of pH include a temperature correction to a standard temperature of 25 ºC.

Soil pH can change seasonally, daily, and hourly depending on temperature and moisture content of the soil. Soil pH generally is reported as a range of pH values for a specified soil depth (USDA 1998).

For agricultural areas, intensive growing of crops can cause a reduction in soil pH; however, farmers can amend the soil to bring its pH back to more neutral levels. Several factors affect soil pH in agricultural lands in particular:

- Addition of organic matter to soil to improve soil aeration and nutrients can result in acidification as the organic materials decompose. Liming can restore pH.
- Addition of ammonium fertilizers to soils results in the production of nitrates, which can hydrolyze in soils to nitric and nitrous acids.
- Harvesting crops can remove some of the alkaline elements (cations) originally in the soil, reducing the soils buffering capacity. (See below for a discussion of soil buffering capacity.)

Phosphorus, one key nutrient for plants, generally is most soluble at soil pH of 6.5 (6.0 to 7.0). Some nutrients are more soluble at lower pH values and some at higher pH values. A pH range of 6.0 to 7.0 generally is considered most favorable for growth for most species of plants because it provides the highest availability of plant nutrients overall. Soil micro-organisms that contribute to the availability of nitrogen, sulfur, and phosphorus in soils perform well in a pH range of 6.6 to 7.3. Soils with a pH less than 5.5 generally have a low availability not only of phosphorus, but also magnesium, calcium , and molybdenum (USDA 1998). Extremely acidic soils (pH 4.0 to 5.0) often result in sufficiently high concentrations of soluble aluminum (and sometimes iron and manganese) to be toxic to many species of plants (SUNY ESF; http://www.esf.edu/pubprog/brochure/soilph/soilph.htm).

Acid-tolerant plants, such as rhododendrons, blueberries, azaleas, and certain pines and other coniferous trees, can grow in soils of pH 4.0 to 5.0, depending on the species (and the source of information), although pH of 5.0 to 5.5 may result in more vigorous growth for these species because of increased nutrient availability.

The ability of a soil to resist changes in pH depends on its buffering capacity. In general, soils with high clay and organic matter content and high cation exchange capacities tend to have higher buffering capacities. Cation exchange capacity (CEC) is the ability of a soil to hold, retain, and exchange cations (i.e., positively charged ions) such as calcium, magnesium, potassium, sodium, ammonium, aluminum, and hydrogen (Daniels and Haering 2006). Soils are generally characterized by a negative surface charge. Negative charges attract cations and prevent their leaching. The higher the CEC, the more cations it can retain. A soil's CEC is calculated by adding the charge equivalents of potassium, ammonium, calcium, magnesium, aluminum, sodium, and hydrogen that are extracted from the exchangeable fraction of the soil.

[3] http://www.bettersoils.com.au/module2/2_3.htm

Low CEC values are in the range of 1 to 10 ten milliequivalents per 100 grams (meq/100g) and high CEC values are in the range of 11 to 50 meq/100g. Soils with low CEC are often characterized by high sand content and low clay content, low organic matter content, and low soil pH. A low CEC indicates that the soil is not resistant to changes in pH or other chemical changes and that the soil is more prone to cation leaching. Soils with high CEC often have low sand and higher silt content, and moderate to high organic matter content. A high CEC indicates that the soil is resistant to changes in pH and is less prone to cation leaching. Thus, soils with a high CEC have a greater buffering capacity than do soils with a low CEC (Daniels and Haering 2006).

Based on this information, we conclude that soils with pH lower than 4.0 are likely to produce adverse effects in most species of plants, including those adapted to acid soil conditions. At a pH less than 4.5, many plants would exhibit reduced growth owing to reduced availability of key nutrients, but necrosis and death are possible where metal ions are mobilized. In areas for which acid-tolerant plants are not native, pH values less than 5.5 are likely to cause adverse effects on plant growth and survival. Soils with CEC values less than 11 meq/100g have low buffering capacity and are less resistant to changes in soil pH than soils with CEC values greater that 11 meq/100g.

J-4.2.3 Indirect Ecological Risk Characterization

We identified local measurements of surface water pH and local measurements of soil pH for the top four facilities according to the final hazard ranking.

J-4.2.3.1 Catskill State Park Ecological Risk Characterization

Surface Waters

The Ravena facility (Facility ID NEI34931), located in Albany County, New York, is close to Alcove Reservoir and Kinderhook Lake. EPA's STORET database did not have local measurements of water pH for Alcove Reservoir or Kinderhook Lake when this analysis was conducted. However, data for these water bodies were available from other sources. The City of Albany Department of Water provided water pH data for Alcove Reservoir (NYS FWD). Annual averages for 2007 and 2008 are presented in Exhibit 4-31. pH measurements were taken at the surface, 5 feet, 20 feet, 34 feet, 48 feet, and at the bottom (65 feet) in 2007 and 2008. Annual pH averages of measurements taken at all depths ranged from 7.0 to 7.8. The lowest annual average of water pH, 7.0, is above the EPA pH benchmark of 6.5. Therefore, adverse ecological effects on fish and other aquatic wildlife associated with low water pH are not anticipated for the Alcove Reservoir near the Ravena Facility.

Exhibit 4-31. Measurements of Water pH for Alcove Reservoir in Albany County, NY

	Depth in Alcove Reservoir (ft)					
	Surface	**5**	**20**	**34**	**48**	**65 (Bottom)**
2007						
Sample Size	9	7	9	9	9	9
Average (SD)	7.7 (0.3)	7.8 (0.2)	7.7 (0.2)	7.2 (0.4)	7.1 (0.4)	7.0 (0.4)
Exceeds pH Benchmark (6.5)	No	No	No	No	No	No
2008						
Sample Size	4	4	4	4	4	4
Average (SD)	7.5 (0.2)	7.7 (0.2)	7.7 (0.2)	7.5 (0.2)	7.4 (0.2)	7.2 (0.2)
Exceeds pH Benchmark (6.5)	No	No	No	No	No	No

Data collected from Kinderhook Lake were provided by a private citizen to whom ICF was referred by the New York Department of Environmental Conservation (NYS DEC). Data are presented in Exhibit 4-32. From 2001 to 2008, Kinderhook Late was treated with alum to bind phosphate and to reduce blue-green algae growth in the summer. In 2001, only the surface water was treated and only surface water pH values were collected. Since 2001, measurements were taken at the surface and at 20 feet. Since alum is acidic, application to the deep regions may have lowered the pH by several tenths, but the pH returned to the pre-treatment values presented in Exhibit 4-32 within two days. Measurements taken before 2004 were obtained using a pH meter that tested up to pH 10. Measurements taken after 2004 were obtained using a color test with a limit of pH 8.2. It is not anticipated that this test underestimated pH levels significantly because surface water values obtained with the original meter did not register values above 8.3. Surface water (1 foot) and deep water (20 feet) pH values are all above the EPA pH benchmark for surface water of 6.5. Therefore, adverse effects on aquatic communities from the ongoing HCl deposition near the Ravena facility are not anticipated.

Exhibit 4-32. Measurements of Water pH for Kinderhook Lake in Albany County, NY

Year	Sample Size	Surface Water pH Annual Average (SD)	Deep Water pH Annual Average (SD)	pH Benchmark	Exceeds pH Benchmark
2001	2	8.1 (0.1)	NA (NA)	6.5	No
2002	3	8.0 (0.4)	7.3 (0.2)		No
2003	3	8.1 (0.1)	7.1 (0.1)		No
2004	3	8.2 (0)	7.5 (0.3)		No
2005	1	8.2 (NA)	7.2 (NA)		No
2006	1	8.2 (NA)	7.2 (NA)		No
2007	1	8.2 (NA)	7.2 (NA)		No

Soils

ICF used EPA's surface water alkalinity map (

ATTACHMENT J-1 Exhibit 2) to determine if the Ravena facility is located in an area of low surface water alkalinity (as an indicator of soil alkalinity). As illustrated in

ATTACHMENT J-1 Exhibit 5, the Ravena facility modeling domain appears to lie almost entirely in an area with high surface water buffering capacity.

ICF also used the USDA's Web Soil Survey to obtain local measurements of soil pH for Catskill State Park, an ecologically valued terrestrial environment, which is approximately 30 km from the Ravena facility (

ATTACHMENT J-1 Exhibit 6). An area of interest (AOI) of 11.2 acres was defined at the nearest boundary of the park to the Ravena facility. Soil data for this AOI are presented in Exhibit 4-33.

Exhibit 4-33. Measurements of Soil pH and Effective CEC for Sensitive Terrestrial Environments Near Portland Cement Facilities Emitting HCl

Facility ID	Facility Location	Nearest Terrestrial Sensitive Environment	Soil Type (% of AOI)	Soil Depth (inches)	Soil pH Measurement	Effective CEC (meq/100g)	pH and ECEC Benchmarks for Soil [a,b]	Relationships to pH and CEC Benchmarks
PTC_NEI34931	Albany County, NY	Catskill State Park	Lewbeach and Willowemoc channery silt loams, moderately steep, very bouldery (≈ 60%)	0 to 6	3.6 to 5.5	0.0 to 2.9	pH: < 4.0 = high risk 4.0 to 5.5 = moderate risk ≥ 5.5 = low risk CEC: 1 to 10 meq/100g = low buffering capacity 11 to 50 meq/100g = high buffering capacity	High, Low
				6 to 21	3.6 to 5.5	0.0 to 4.0		High, Low
			Vly-Halcott complex, strongly sloping, very rocky (≈40%)	0 to 2	3.6 to 6.0	0.1 to 82		High, High
				2 to 28	3.6 to 5.5	0.1 to 15		High, High
PTC_NEI26327	Hernando County, FL	Withlacoochee State Forest	Arrendo Fine Sand (≈ 30%)	0 to 8	4.5-6.0	0.2 to 1.6		Medium, Low
				8 to 62	4.5-6.0	0.0 to 3.2		Medium, Low
			Candler Fine Sand (≈ 70%)	0 to 4	4.5 to 5.5	0.1 to 1.8		Medium, Low
				4 to 48	4.5 to 5.5	0.0 to 1.6		Medium, Low

[a] pH <4.0 = high risk adverse effects in most species of plants, including those adapted to acid soil conditions; pH 4.0 to 5.5 = medium risk to plant species; ph ≥ 5.5 = low risk to all plant species

[b] CEC 1 to 10 meq/100g = low buffering capacity and low resistance to changes in soil pH; CEC 11 to 50 meq/100g = high buffering capacity and resistant to changes in soil pH

This AOI was characterized by two types of soil. Lewbeach and Willowmoc soils comprised approximately 60 percent of the AOI and were characterized by USDA (2008) as “silt loams” and “moderately steep.” The top soil (0 to 6 inches) and deeper soil (6 to 21 inches) pH ranged from 3.6 to 5.5. Effective cation-exchange capacity (ECEC) for the top soil layer ranged from 0.0 to 2.9 meq/100g, and for the deeper soil ranged from 0.0 to 4.0. Of note is the large range in soil pH that characterizes the same soil layer and type, limiting the value of the soil pH benchmarks indicated in Section J-4.2.2.2.

The lower boundary of the pH range for the Lewbeach and Willowmoc soils is below the benchmark of 4.0, indicating a possibly high risk of adverse effect on many plant species, including acid tolerant plants. The CEC values for the Lewbeach and Willowmoc soils also are low, suggesting that significant parts of the Catskill State Park’s soil has a low acid buffering capacity and is not resistant to changes in soil pH. Note that the Catskill State Park *is* in an area of the EPA surface water alkalinity map associated with alkalinity measurements of less than 100 mg/L.

ATTACHMENT J-1 Exhibit 5).

The other soil type, Vly-Halcott soil, comprised approximately 40 percent of the AOI and was described by USDA (2008) as “complex, strongly sloping” and “very rocky.” The top two inches of soil had soil pH values in the range of 3.6 to 6.0, indicating a low pH below the soil benchmark of 4.0. However, this soil had CEC values ranging up to 82 meq/100g, suggesting that at least patches of this soil have high acid buffering capacity. Deeper soil (2 to 28 inches) of the same type had similarly low pH values (3.6 to 5.5) and relatively high CEC values (up to 15 meq/100g).

Given the possibly high sensitivity of the Catskill State Park area to further acid deposition, GIS was used to evaluate the overlap between the HCl air concentrations estimated around the Ravena facility and nearby parks.

ATTACHMENT J-1 Exhibit 6 illustrates that deposition of HCl emitted by the facility is unlikely to reach the Catskill State Park.

ATTACHMENT J-1 Exhibit 7 shows that deposition of HCl emitted by the facility might reach into the John Boyd Thacher and Hudson River Islands State Parks, but only in areas with surface water alkalinity greater than 400 mg/L (as $CaCO_3$) and therefore soils with a relatively high acid buffering capacity. HCl emissions from the Ravena facility, therefore, are not expected to produce indirect adverse ecological effects associated with acidification of either surface waters or valued terrestrial ecosystems near the facility. This conclusion is supported by aerial photography in the vicinity of the facility which indicates no discernable adverse effects (e.g., die-back, chlorosis) on coniferous vegetation and no indication of adverse effects (e.g., excess fallen trunks) in the deciduous portions of the forests.[4]

J-4.2.3.3.2 Withlacoochee State Forest Ecological Risk Characterization

The Florida facility in Hernando County (NEI126327) is closest to Withlacochee State Forest, an ecologically valued terrestrial environment located approximately 11 km from the Portland Cement facility. We defined an AOI of 57.4 acres at the boundary of the forest closest to the Hernando County facility using USDA's Web Soil Survey. Soils in this AOI were characterized as either Arrendo fine sand, which comprised approximately 30 percent of the AOI, or Candler fine sand, which comprised approximately 70 percent of the AOI (USDA 2008). EPA's surface water alkalinity map suggests that the acid buffering capacity of surface waters in the area surrounding the facility is not unusually low (i.e., greater than 400 mg/L).

The range of soil pH values listed for Candler top soil (0 to 4 inches) and deeper soils (4 to 48 inches) were the same: 4.5 to 5.5. CEC was slightly higher in the top soil (0.1 to 1.8 meq/100g) than in deeper soils (0.0 to 1.6 meq/100g). The soil pH range is consistent with an acid-tolerant plant community, but is unlikely to support acid-intolerant species. A low CEC suggests that the soil does not have a high acid buffering capacity, suggesting a low tolerance for further acidification. Soil pH in the other soil type, Arrendo fine sand, also did not differ for surface soil (0 to 8 inches) and deeper soil (8 to 62 inches): pH range of 4.5 to 6.0. The lower boundary of this range indicates some risk to acid-intolerant plant species; however, the vegetation of the area may have evolved to be acid-tolerant given the sandy nature of both types of soils. Effective CEC in Arrendo top soil ranged from 0.2 to 1.6 meq/100g and in deeper soil ranged from 0.0 to 3.2 meq/100g. The range of soil pH measurements in Arrendo soil suggests moderate risk to acid-intolerant plant species, and low CEC suggests that the soil does not have significant buffering capacity, and might not be resistant to changes in soil pH. The sandy soils in this area, however, may have contributed to the development of acid-tolerant plant communities.

Inspection of aerial photographs of this facility was inconclusive owing to patchy distribution of land uses near the facility and areas of grass and shrubs predominating in some directions and with trees only apparent in other directions from the facility. Without additional data for the environment in the vicinity of this facility, we cannot draw any conclusions regarding the potential for indirect ecological effects of HCl emitted from the facility. HCl emissions rates and background acid deposition rates are lower than for Ravena, however.

J-4.2.3.3.3 Lake Charlevoix Ecological Risk Characterization

The nearest sensitive environment to the Michigan facility (NEIMIB1559) is Lake Charlevoix, which is approximately 4 km from the Portland Cement facility. EPA's STORET database had local measurements of surface water pH for Lake Charlevoix that ranged from 7.7 to 8.3 (Exhibit 4-34). This range is above the EPA pH benchmark for surface water of 6.5. Therefore, adverse

[4] EnviroMapper for Envirofacts located at: http://www.epa.gov/enviro/emef/.

ecological effects on fish and other aquatic wildlife associated with low surface water pH are not anticipated.

Exhibit 4-34. Measurements of Surface Water pH for Sensitive Aquatic Environments Near Portland Cement Facilities Emitting HCl

Facility ID	Facility Location	Nearest Aquatic Sensitive Environment	Surface Water pH Measurement	pH Benchmark	Exceeds pH Benchmark
PTC_NEIMIB1559	Charlevoix County, MI	Lake Charlevoix	7.7 to 8.3	6.5	No
PTC_NEI51435	La Salle County, IL	Depue Lake	7.5 to 9.0	6.5	No

J-4.2.3.4 Lake Depue Ecological Risk Characterization

The Illinois River is the closest sensitive environment to the facility in La Salle County, Illinois (NEI151435). However, EPA's STORET database did not have local surface water data for the Illinois River when this analysis was conducted. Local surface water measurements were, however, available for Depue Lake, which is approximately 20 km from the Portland Cement facility (see Exhibit 4-34). The reported surface water pH ranged from 7.5 to 9.0. This range is above EPA's 6.5 pH benchmark for surface water, thus adverse ecological effects associated with low surface water pH are not anticipated.

J-4.2.3.5 Summary of Risk Characterization for Indirect Effects of HCl

ICF used four factors to identify the most likely of the 91 Portland Cement facilities to pose risks of indirect ecological effects associated with HCl deposition surrounding a facility. Four facilities tied for the low hazard score (highest potential ecological risks). For all four facilities, pH values in all nearby bodies of water were above the EPA pH criterion for freshwater of 6.5. For the terrestrial environments, several lines of evidence indicate that the Ravena facility is not likely to cause indirect adverse ecological effects associated with soil acidification in nearby valued or unprotected terrestrial environments. Data from the Florida facility are inconclusive, but are consistent with sustainable acid-tolerant plant communities which often occur in sandy soils.

J-5 References

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ATTACHMENT J-1: Ecological Risk Assessment Case Study Supporting Documents

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Exhibit 1. Background Acid Precipitation Monitoring Data from USGS

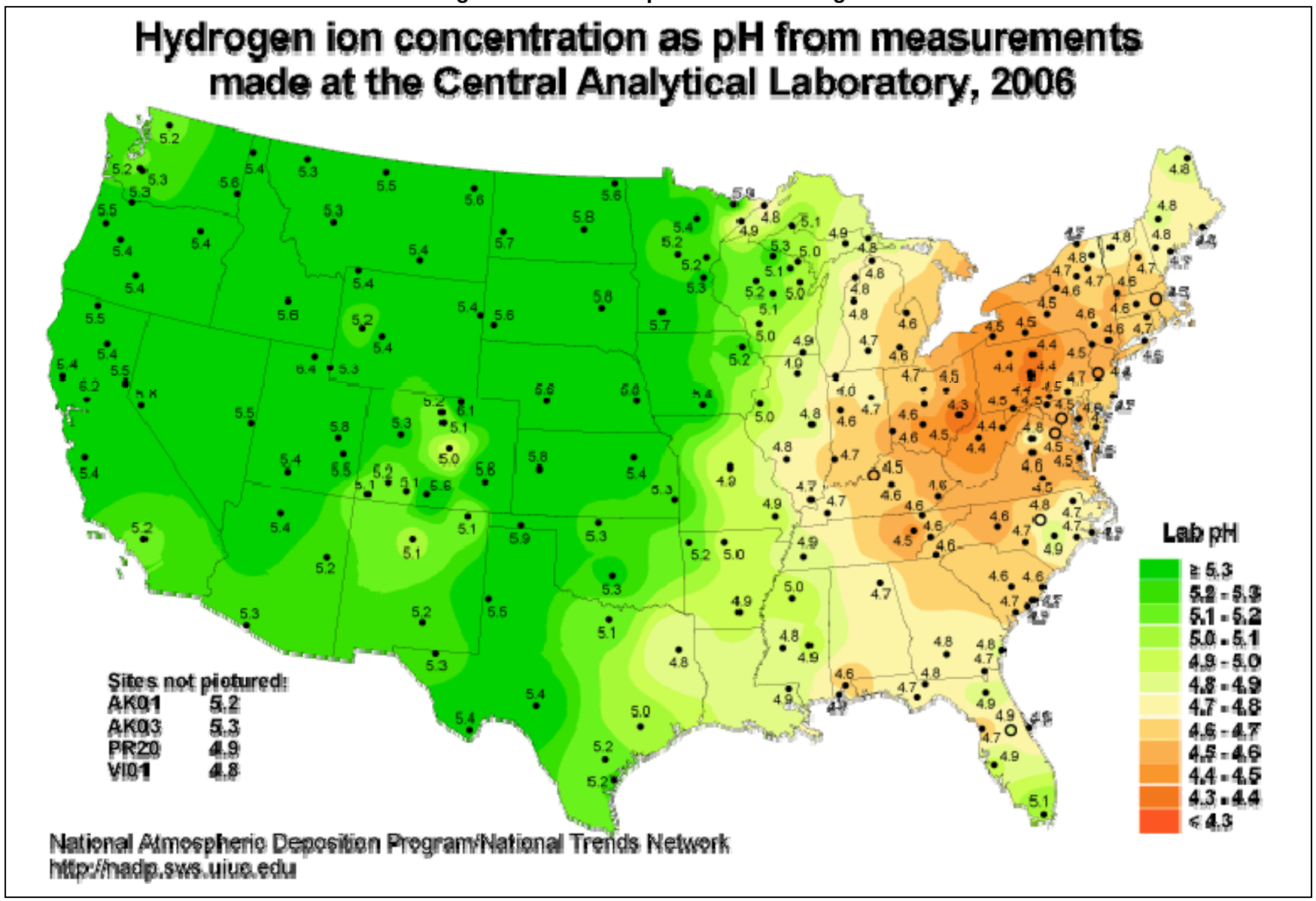
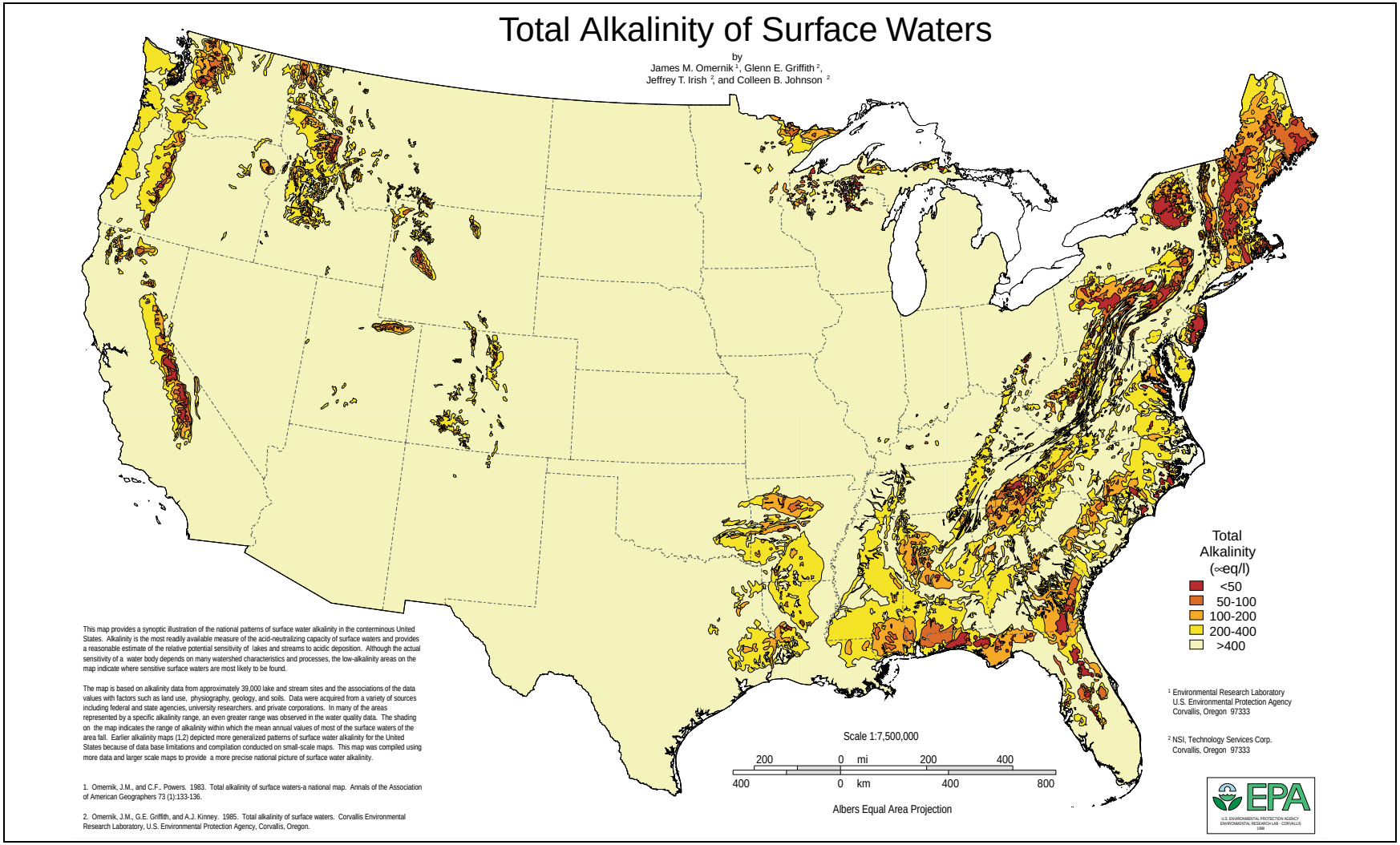


Exhibit 2. Total Alkalinity of Surface Waters of the Conterminous United States



Available from http://geodata.epa.gov/WAF/Total%20Alkalinity%20of%20Surface%20Waters%20of%20the%20US.xml

Exhibit 3. Ecosystem Background Deposition, Alkalinity, Facility Emission, and Preliminary Ecological Hazard Scores for Portland Cement Facilities Emitting HCl

Facility	Facility Location	Acid Deposition (pH of Rainfall)	Background Exposure Score	Surface Water Alkalinity (meq/L)	Alkalinity Score	Emissions (TPY)	Emissions Score	Preliminary Hazard Score
PTC_NEI34931	Albany County, NY	4.5-4.6	2	>400	5	71.08	1	10
PTC_NEI26327	Hernando County, FL	4.6-4.7	2	>400	5	48.48	1	10
PTC_NEI33394	Carroll County, MD	4.5-4.6	2	>400	5	180.00	1	10
PTC_NEISC0351244	Dorchester County, SC	4.6-4.7	2	>400	5	97.53	1	10
PTC_NEIMIB1559	Charlevoix County, MI	4.7-4.8	3	>400	5	323.47	1	15
PTC_NEI51435	La Salle County, IL	4.8-4.9	3	>400	5	39.70	1	15
PTC_NEI12018	Alpena County, MI	4.7-4.8	3	>400	5	474.56	1	15
PTC_NEIPAT$1626	Lawrence County, PA	4.4-4.5	1	>400	5	10.90	3	15
PTC_NEIMO0990002	Jefferson County, MO	4.8-4.9	3	>400	5	72.15	1	15
PTC_NEI51352	La Salle County, IL	5.0-5.1	4	>400	5	32.69	1	20
PTC_NEI31319	Clark County, IN	4.6-4.7	2	>400	5	18.86	2	20
PTC_NEI7255	Northampton County, PA	4.5-4.6	2	>400	5	16.85	2	20

Exhibit 3. Ecosystem Background Deposition, Alkalinity, Facility Emission, and Preliminary Ecological Hazard Scores for Portland Cement Facilities Emitting HCl

Facility	Facility Location	Acid Deposition (pH of Rainfall)	Background Exposure Score	Surface Water Alkalinity (meq/L)	Alkalinity Score	Emissions (TPY)	Emissions Score	Preliminary Hazard Score
PTC_NEI2CA151186	Santa Cruz County, CA	>5.3	5	<50	1	4.79	4	20
PTC_NEI12238	Scott County, IA	5.2-5.3	5	>400	5	76.60	1	25
PTC_NEI26277	Miami-Dade County, FL	5.1-5.2	5	>400	5	63.17	1	25
PTC_NEI22838	San Bernardino County, CA	5.1-5.2	5	>400	5	38.89	1	25
PTC_NEIPA01993-1	Butler County, PA	4.4-4.5	1	>400	5	0.43	5	25
PTC_NEI52351	Massac County, IL	4.7-4.8	3	>400	5	32.49	2	30
PTC_NEIAL1150002	St. Clair County, AL	4.7-4.8	3	>400	5	28.11	2	30
PTC_NEI32033	Lawrence County, IN	4.6-4.7	2	>400	5	13.57	3	30
PTC_NEI2PA110039	Berks County, PA	4.5-4.6	2	>400	5	11.74	3	30
PTC_NEIVA2553	Botetourt County, VA	4.5-4.6	2	>400	5	8.54	3	30
PTC_NEIKYR0060	Jefferson County, KY	4.5-4.6	2	>400	5	8.20	3	30
PTC_NEIAL8026	Mobile County, AL	4.6-4.7	2	200-400	4	4.04	4	32
PTC_NEITX139099J	Ellis County, TX	4.9-5.0	4	>400	5	18.53	2	40
PTC_NEIPA94-2626	Northampton County, PA	4.5-4.6	2	>400	5	4.27	4	40

Exhibit 3. Ecosystem Background Deposition, Alkalinity, Facility Emission, and Preliminary Ecological Hazard Scores for Portland Cement Facilities Emitting HCl

Facility	Facility Location	Acid Deposition (pH of Rainfall)	Background Exposure Score	Surface Water Alkalinity (meq/L)	Alkalinity Score	Emissions (TPY)	Emissions Score	Preliminary Hazard Score
PTC_NEI33699	Washington County, MD	4.5-4.6	2	>400	5	3.80	4	40
PTC_NEIFLR001008	Alachua County, FL	4.8-4.9	3	>400	5	8.60	3	45
PTC_NEI51527	Lee County, IL	4.8-4.9	3	>400	5	8.02	3	45
PTC_NEI22877	San Bernardino County, CA	5.1-5.2	5	>400	5	32.14	2	50
PTC_NEI16357	Montgomery County, KS	>5.3	5	>400	5	31.01	2	50
PTC_NEIIA0330035	Cerro Gordo County, IA	5.1-5.2	5	>400	5	22.49	2	50
PTC_NEI25375	Shasta County, CA	>5.3	5	>400	5	20.00	2	50
PTC_NEI20046	Kern County, CA	>5.3	5	>400	5	19.53	2	50
PTC_NEI12739	Allen County, KS	>5.3	5	>400	5	19.50	2	50
PTC_NEIPA58-1290	Lehigh County, PA	4.5-4.6	2	>400	5	2.15	5	50
PTC_NEITN0653070	Hamilton County, TN	4.6-4.7	2	>400	5	1.98	5	50
PTC_NEITX309123F	McLennan County, TX	5.0-5.1	4	>400	5	7.68	3	60
PTC_NEIMIB1743	Monroe County, MI	4.6-4.7	3	>400	5	3.83	4	60
PTC_NEIAL321	Marengo County, AL	4.8-4.9	3	>400	5	2.52	4	60

Exhibit 3. Ecosystem Background Deposition, Alkalinity, Facility Emission, and Preliminary Ecological Hazard Scores for Portland Cement Facilities Emitting HCl

Facility	Facility Location	Acid Deposition (pH of Rainfall)	Background Exposure Score	Surface Water Alkalinity (meq/L)	Alkalinity Score	Emissions (TPY)	Emissions Score	Preliminary Hazard Score
PTC_NEI18621	Pima County, AZ	5.2-5.3	5	>400	5	16.55	3	75
PTC_NEI13290	Comal County, TX	5.1-5.2	5	>400	5	14.22	3	75
PTC_NEI34326	Jackson County, MO	5.2-5.3	5	>400	5	14.16	3	75
PTC_NEITXT$11924	Hays County, TX	5.1-5.2	5	>400	5	7.40	3	75
PTC_NEI886	Fremont County, CO	5.0-5.1	4	>400	5	6.43	4	80
PTC_NEI7376	Ellis County, TX	4.9-5.0	4	>400	5	3.25	4	80
PTC_NEITXT$11872	Bexar County, TX	5.1-5.2	5	>400	5	6.31	4	100
PTC_NEI12976	Mayes County, OK	5.2-5.3	5	>400	5	3.56	4	100
PTC_NEIFL0860020	Miami-Dade County, FL	5.1-5.2	5	>400	5	3.07	4	100
PTC_NEICA1505122	Kern County, CA	>5.3	5	>400	5	2.97	4	100
PTC_NEI338	Albany County, WA	>5.3	5	>400	5	2.23	5	125
PTC_NEI40539	Baker County, OR	>5.3	5	>400	5	2.00	5	125
PTC_NEI20130	Kern County, CA	>5.3	5	>400	5	1.96	5	125
PTC_NEINMT$12442	Bernalillo County, NM	5.1-5.2	5	>400	5	1.95	5	125
PTC_NEIID0050004	Bannock County, ID	>5.3	5	>400	5	1.78	5	125

Exhibit 3. Ecosystem Background Deposition, Alkalinity, Facility Emission, and Preliminary Ecological Hazard Scores for Portland Cement Facilities Emitting HCl

Facility	Facility Location	Acid Deposition (pH of Rainfall)	Background Exposure Score	Surface Water Alkalinity (meq/L)	Alkalinity Score	Emissions (TPY)	Emissions Score	Preliminary Hazard Score
PTC_NEI446	Boulder County, CO	5.1-5.2	5	>400	5	1.51	5	125
PTC_NEITXT$11980	Nolan County, TX	>5.3	5	>400	5	0.80	5	125
PTC_NEI22743	San Bernardino County, CA	5.1-5.2	5	>400	5	0.33	5	125
PTC_NEI22453	Riverside County, CA	5.1-5.2	5	>400	5	0.17	5	125

Exhibit 4. Location of Portland Cement Facilities Relative to Surface Water Alkalinity in the Conterminous United States

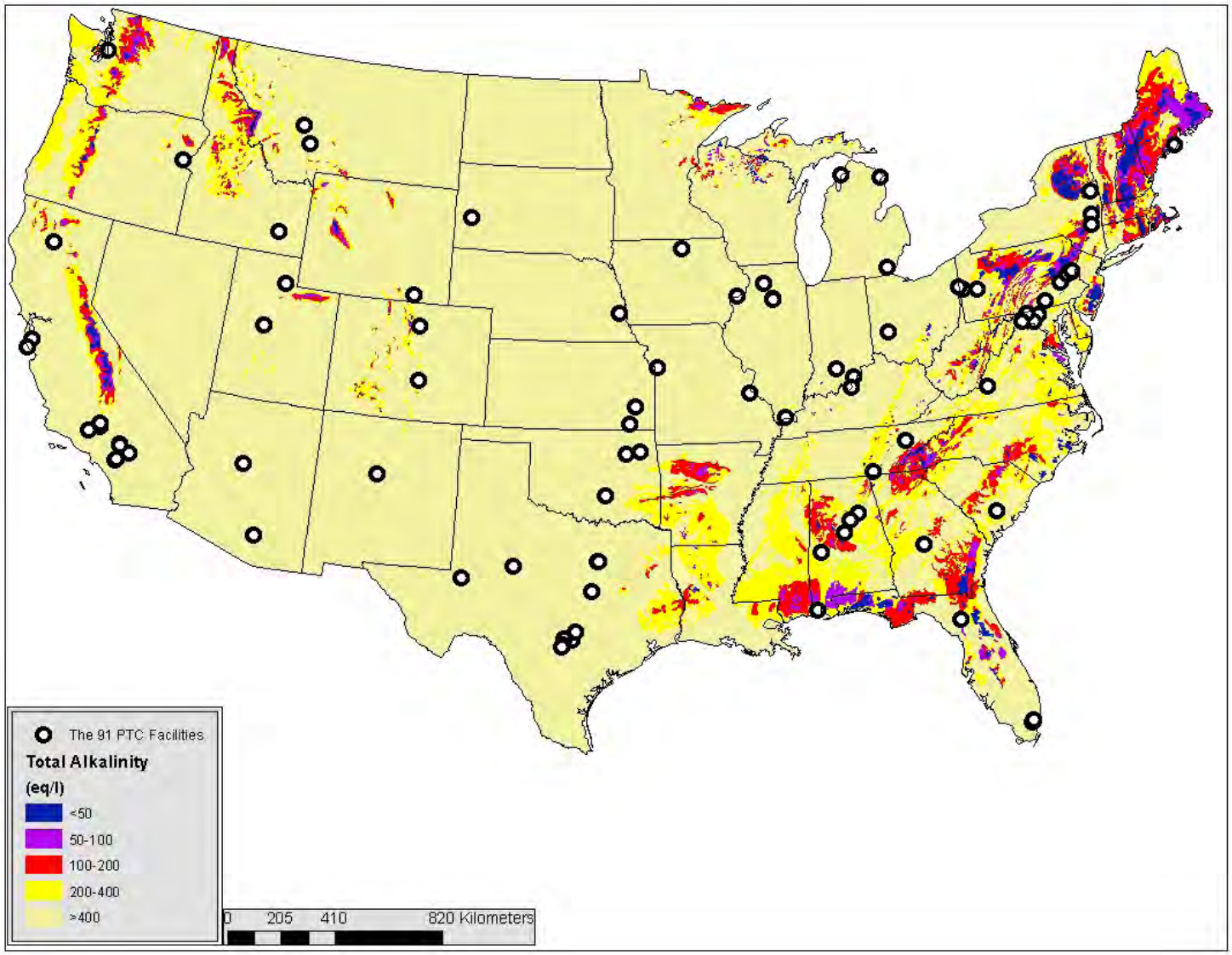


Exhibit 5. Location of Ravena Facility
Modeling Domain Relative to Surface Water Alkalinity

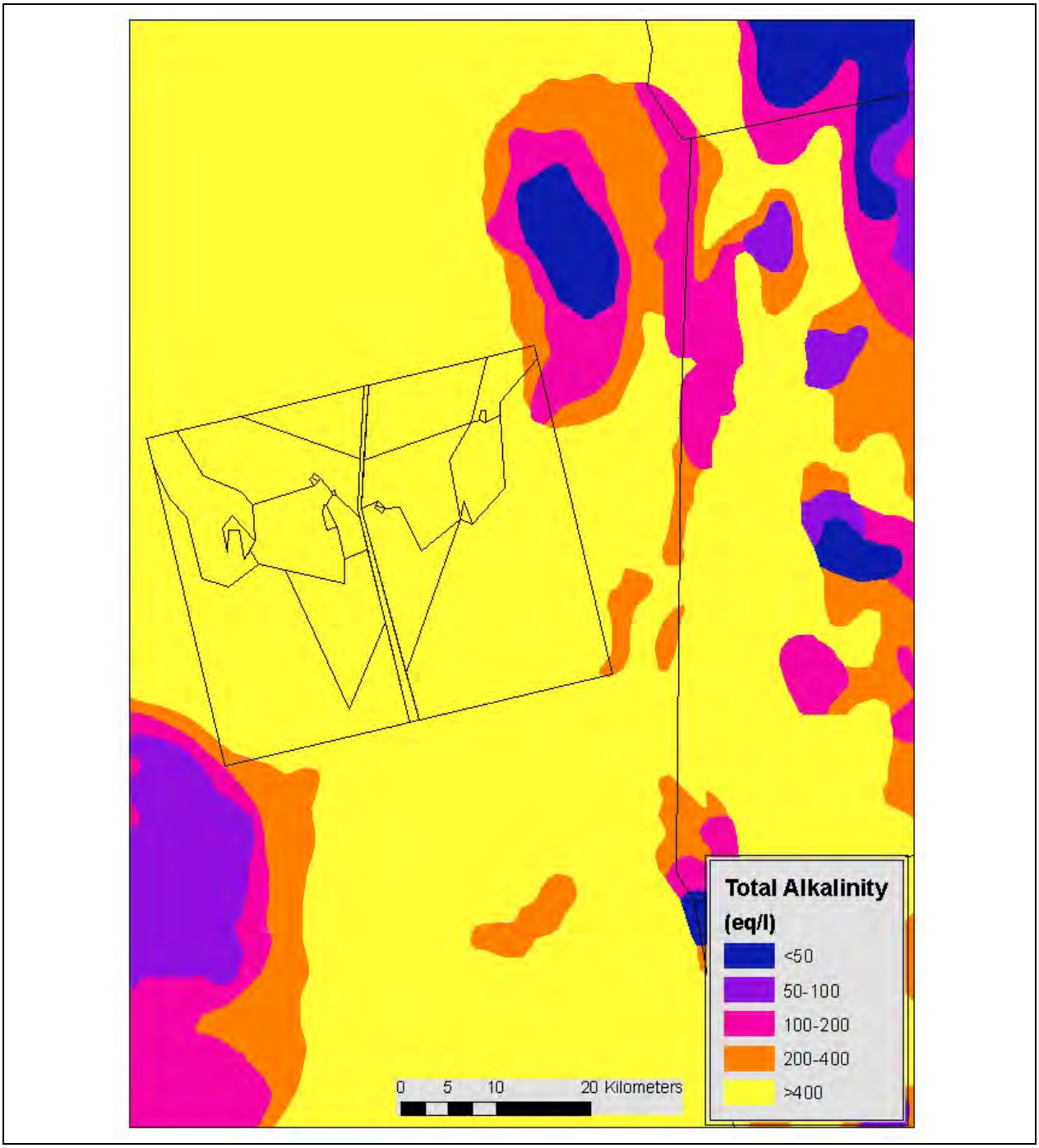


Exhibit 6. HCl Concentrations in Air Estimated from HCl Emissions from Ravena Facility Relative to Nearest Large Parks and Preserves

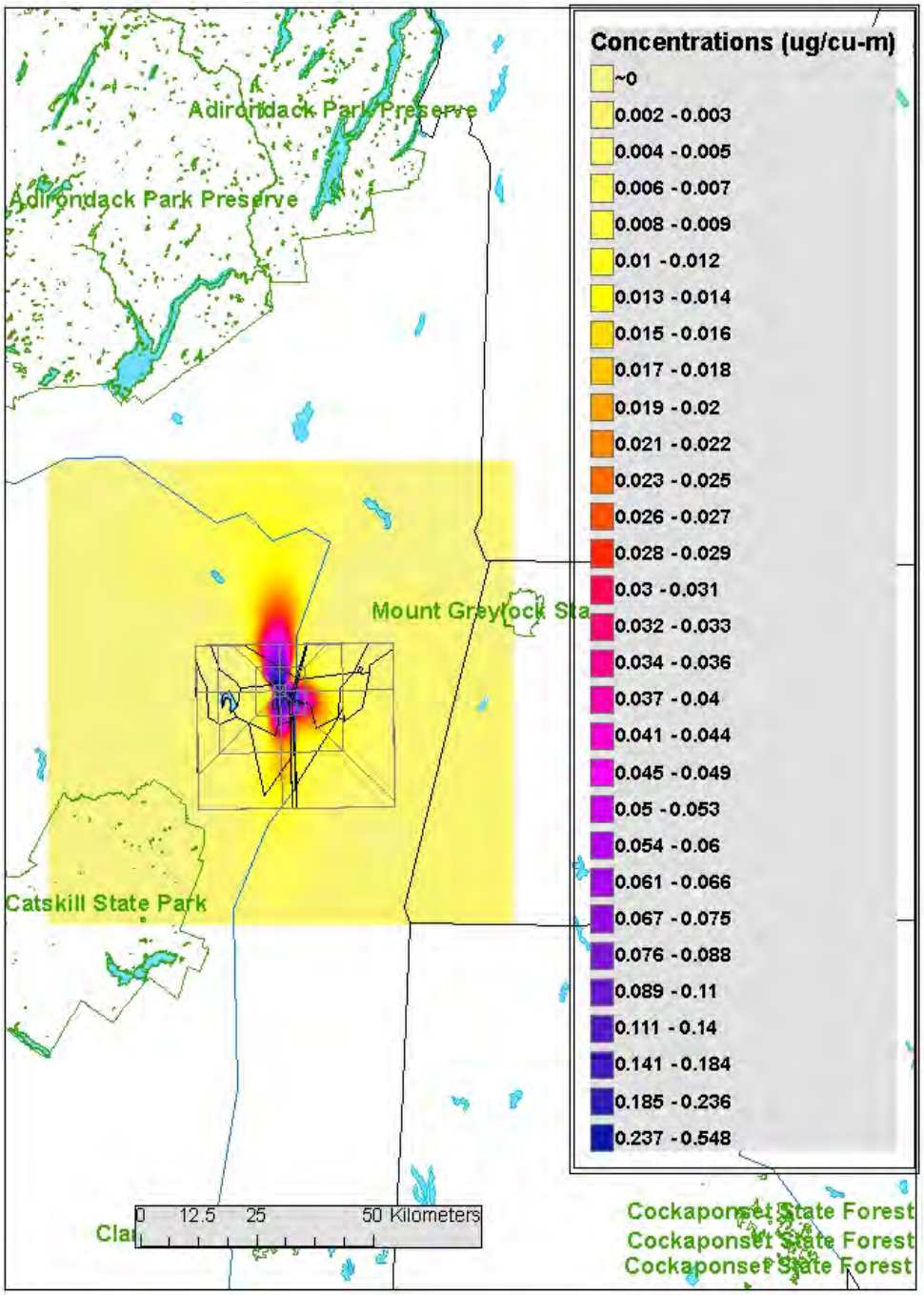
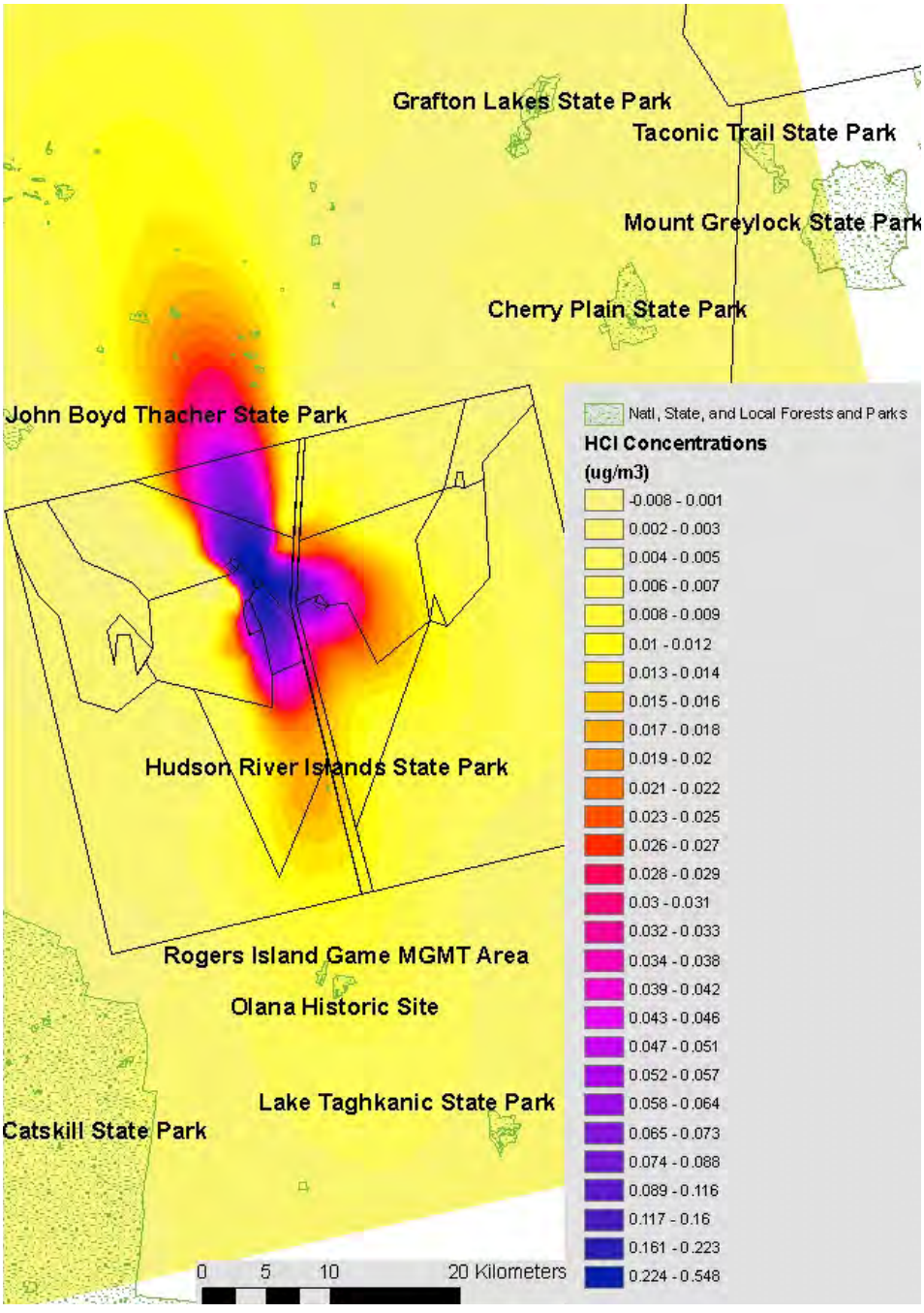


Exhibit 7. HCl Concentrations in Air Estimated from HCl Emissions from the Ravena Facility Relative to Nearby Smaller Valued Parks and Other Areas



Appendix K

Development of a threshold concentration for foliar damage caused by ambient hydrogen chloride concentrations

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K.1 Introduction

Exposure thresholds have been developed for many chemicals for exposure of plants and animals to media such as water, soil and sediments. Screening level ecological exposure thresholds are frequently compared to measured or modeled media concentrations to determine whether a full ecological risk assessment is required. Unfortunately, few exposure thresholds have been developed for direct air exposures, and risk assessments done under the air toxics program do not routinely assess these exposures. As a result, in order to develop a case study for direct plant exposure to a hazardous air pollutant we were required to develop an exposure threshold for use in this assessment. A literature search was conducted through several university libraries in the attempt to locate information that could be used in developing screening-level hydrogen chloride (HCl) ecological exposure thresholds for foliar damage. Over 50 scientific databases were accessed in the literature search, yielding the studies described below.

In the late 1970's and early 1980's, a series of studies were conducted by the Statewide Air Pollution Research Center of the University of California at Riverside to determine the impact of gaseous hydrogen chloride (HCl) on plants. These experiments, designed to examine the effects of massive, nearly instantaneous, releases of HCl on vegetation, and the subsequent journal articles, were supported by grants from the U.S. Air Force Office of Scientific Research to assess the potential damage to plants, as compared to controls, from short-term exposures to high concentrations of gaseous HCl in the exhaust from some types of solid-fuel rockets.

In addition to the studies at the University of California at Riverside, the Air Force summarized supported studies done at the University of California at Irvine in the document *The Phytotoxicity of Designated Pollutants on Plant Species* (USAF, 1983). The designated pollutants are HCl and aluminum oxide emitted from the solid rocket fuel used in the rockets that launch the space shuttle.

These exposure conditions could also be characteristic of mass releases from spills or equipment failures. EPA is interested in determining how the results of these studies could be used to develop gaseous HCl ecological exposure thresholds to compare to short-term average (1 to 24 hour) and long-term average (annual) air concentrations of gaseous HCl from routine industrial releases to estimate the potential for them to cause foliar damage. The studies are summarized below.

K.2 Summary of Studies

K.2.1 Phytotoxicity of Hydrogen Chloride Gas with a Short-Term Exposure

In the introduction to this journal article, Lerman, et. al. (1976) provided a synopsis of research on damage from gaseous HCl plant exposures from the early 1900's on. This information is presented in table form in Table K-1.

The purpose of this study was to determine the concentration of HCl required to induce morphological injury to eight types of ornamental plants: aster, calendula, cornflower, cosmos, American marigold, French marigold, nasturtium, and zinnia. Several ages of plants were

exposed for 20 minutes to gaseous HCl concentrations ranging from 1 to 35 mg/m^3.[1] The degree of injury to the plants was evaluated 24 and 48 hours after exposure by external examination using an arbitrary 1 to 10 scale. The evaluation was based on number of injured leaves per plant, estimated percentage of foliar surface affected, and the overall appearance of the plant.

Table K-1. Synopsis of Early Research on Damage from Gaseous HCl Plant Exposures

Exposure	Results	Reference
Single exposure, 2 day duration at 5-20 ppm (8-30 mg/m^3)	Seedlings of viburnum and larch killed in less than 2 days	Haselhoff and Lindau, 1903
Single exposure, 1 hour duration at 1,000 ppm (1,500 mg/m^3)	Bleached lesions on leaves of fir, birch and oak	
80 exposures, duration 1 hour/day at 2,000 ppm (3,000 mg/m^3)	Necrosis on margins of maple, birch and pear tree leaves	
Single exposure, 2 hour duration at 5 ppm (8 mg/m^3)	28 day old tomato plants developed interveinal bronzing followed by necrosis within 72 hours	Shriner and Lacasse, 1969
Single exposure, 4 hour duration at 3-43 ppm (5-65 mg/m^3)	2-5 year old seedlings, 12 types of coniferous and broadleaf trees: - Most sensitive *Liriodendron tulipfera* (tulip tree) visible injury at the lowest concentration of 3 ppm (5 mg/m^3) - Most sensitive conifer *Pinus strobes* (white pine) visible injury at 8 ppm (12 mg/m^3) - No injury to *Thuja occidentalis* (white cedar) at 43 ppm (65 mg/m^3)	Means and Lacosse, 1969
Single exposure, 5 min. duration at 95, 300, and 2,071 ppm (140, 460, and 3,150 mg/m^3)	Mature, flowering marigold plants: 95 ppm – little or no visible injury 300 ppm – temporary wilting and leaf spots 2,071 ppm – severe wilting, marginal and interveinal leaf necrosis, stem collapse, and death	Lind and London, 1971
208 hours of exposure within 2 weeks at 1.6 mg/m^3 (1 ppm)	Slight necrosis and chlorosis on *Spinacia oleracea* L. (spinach) leaves	Mausch *et.al*., 1973
Single exposure, 29 hour duration, 0.5 mg/m^3 (0.3 ppm)	Carrots: - Exposed 45 days after germination showed 32.2 to 49.7% decrease in crop yield - Exposed 96 days after germination showed only 5.3% crop yield decrease	Hulensberg, 1974

[1] mg HCl/m^3 = ppm x 1.52

Exposure	Results	Reference
	- Winter grape only leaf discoloration, radishes no damage - Tomato, cucumber and bush bean plants showed leaf damage and increase in leaf chlorides - Reduction in yield severe in tomato and slight in cucumber plants	

Source: Lerman *et. al.*, 1976

Table K-2 presents a qualitative summary of injury symptoms as a function of HCl concentration. Table K-3 presents a comparison of the relative sensitivity of the eight plant species. The relative sensitivity is expressed as the concentration of gaseous HCl required to cause 10% relative injury calculated using first and second order, polynomial type regression equations.

Table K-2. Injury symptoms on Eight Plant Species 23 Hours After Exposure to HCl Gas

HCl Concentration	Aster	Calendula	Centaurea	Cosmos
21-35 mg/m^3	Temporary wilting, extensive interveinal bronzing on lower leaf surface, necrosis of young tissue.	Temporary wilting, lower surface, discoloration, necrosis. Younger the leaf the more distal the damage.	Extensive necrosis, rolling, speckling, temporary wilting, discoloration.	Extensive necrosis, extensive rolling, flower discoloration, tipburn of sepals.
10-20 mg/m^3	Interveinal bronzing on lower surface, trace of necrosis.	Bronzing of lower leaf surface, interveinal necrosis, marginal discoloration.	Discoloration along the leaf margin, rolling.	Tipburn, tip rolling.
1.5-9 mg/m^3	Trace of necrotic spots on young leaves.	Trace of lower surface bronzing.		Tipburn.
HCl Concentration	**Marigold, Fr. Dwarf**	**Marigold, Am. Sen. Dirksen**	**Nasturtium**	**Zinnia**
21-35 mg/m^3	Severe necrosis of almost all leaves, rolling.	Severe necrosis, extensive rolling, tipburn of sepals on flowers.	Interveinal bleached lesions, on younger leaves, in addition, marginal bleaching and	Bronzing on basal leaf portions, extensive necrosis and rolling on rest of leaf. Occasional petal necrotic spots.

			rolling.	
10-20 mg/m^3	Discoloration, necrosis of mid-aged leaves, some rolling.	Interveinal discoloration of mid-aged leaves, some rolling.	Discoloration, necrotic speckling, rolling.	Speckling, interveinal bronzing.
1.5-9 mg/m^3	Traces of necrosis or discoloration.	Traces of necrosis or discoloration.	Traces of discoloration.	Traces of lower surface bronzing.

Source: Lerman *et. al.*, 1976

Table K-3. Relative Sensitivity of Eight Species of Ornamental Plants to HCl Gas

Species	Concentration of HCl Gas (mg/m^3) in a 20 Minute Exposure Required to Cause 10% Relative Injury
Cosmos	6.5
Marigold (French)	8.8
Marigold (American)	9.5
Zinnia	15.3
Nasturtium	15.7
Calendula	16.1
Centaurea	18.3
Aster	29.9

Source: Lerman *et. al.*, 1976

K.2.2 Foliar and Microscopic Observations of Bean Leaves Exposed to Hydrogen Chloride Gas

In this Endress et.al. (1978) experiment, pinto beans 8 days and 12 days from seeding were exposed for 20 minutes to gaseous HCl concentrations ranging from 6 to 54.2 mg/m^3. The plants were evaluated immediately after exposure, and at 30 minutes and 1, 2, 3, and 24 hours after exposure. The leaves were evaluated for visible effects and for cellular level changes using microscopy.

The first visible symptom of injury to primary leaves was glazing of the lower leaf surface followed by injury to the upper leaf surface. Interveinal necrosis and/or rolling of the leaf occurred as the HCl concentration increased. These symptoms were similar to those observed for other species. Table K-4 presents the extent of necrotic lesions observed from the multiple exposure concentrations.

In sectioned leaf tissue, the glazing appeared to result from collapse of epidermal cells that seem to result from deformation of both the inner and outer cell walls. A frequently observed

symptom related to cell collapse was plasmolysis[2] of the protoplast. The cytoplasm left its normal position by retracting from the cell wall. In cases of more severe HCl stress, irreversible plasmolysis occurred as well as cell wall collapse. Mesophyll tissue was usually affected to a lesser extent than the epidermis, with the most common symptom of injury being plasmolysis. Other changes noted included the formation of vesicles or small vacuoles and the formation of crystals in mesophyll cells. Table K-5 presents the microscopic cellular injury observed and the concentrations of gaseous HCl necessary to invoke the injuries.

Table K-4. Extent of Necrotic Lesions on Primary Leaves of Pinto Bean 24 Hours After Exposure for 20 Minutes to Varying Concentrations of HCl Gas

Treatment Concenration mg HCl/m^3	Average Leaf Area cm^2	Range of Average Necrotic Area* cm^2	Range of Average % Necrotic Area*
0	16.21 ± 4.31**	0	0
6.0	16.63 ± 5.31	0	0
11.3	18.00 ± 4.34	0	0
17.9	19.49 ± 5.95	0.40 ± 0.52** (0.1-1.0)	1.6 ± 2.1** (0.3-4.0)
25	17.99 ± 5.90	0.1	0.1
32	16.67 ± 6.51	0.57 ± 1.00 (0.4-4.30)	4.9 ± 10.9 (0.1-45.8)
41.3	13.83 ± 6.71	1.20 ± 1.66 (0.08-7.80)	11.0 ± 16.8 (0.4-78.9)
54.2	11.46 ± 6.52	5.69 ± 4.25 (0.10-16.80)	55.6 ± 34.2 (0.6-96.7)

*Calculations of average necrotic area and average percent necrotic area excluded leaves devoid of necrotic lesions.
**± Standard deviation. No attempt was made to determine statistical significance between treatments because of large variabilities within treatments.

Source: Endress et.al., 1978

Table K-5. Microscopic Cellular Injury Symptoms Observed in Sectioned Primary Leaves of Pinto Bean Following Exposure to Several Concentrations of Gaseous HCl*

Symptom and Location	Immediate Post-Fumigation mg HCl/m^3	24 Hour Post-Fumigation mg HCl/m^3
Plasmolysis in		
adazial epidermis	≥6.0	≥6.0
palisade parenchyma	≥17.9	≥17.9
spongy parenchyma	≥17.9	≥17.9
abaxial epidermis	≥17.9	11.3, 17.9

[2] Plasmolysis is the contraction of cells within plants due to the loss of water through osmosis. It is the cell membrane peeling off of the cell wall and the vacuole collapsing. plasmolysis occurs when a plant cell's membrane shrinks away from its cell wall. This phenomenon occurs when water is drawn out of the cell and into the extracellular (outside cell) fluid. The movement of water occurs across the membrane moving from an area of high water concentration to an area of lower water concentration outside the cell. It is unlikely to occur in nature, except in severe conditions. http://www.bio-medicine.org/biology-definition/Plasmolysis/

Symptom and Location	Immediate Post-Fumigation mg HCl/m^3	24 Hour Post-Fumigation mg HCl/m^3
Vacuolar particulates in		
adazial epidermis	≥6.0	≥6.0
palisade parenchyma	6.0-21.1	54.2
spongy parenchyma	6.0, 11.3	54.2
abaxial epidermis	≥11.3	41.3, 54.2
Vesiculation in		
adazial epidermis	≥6.0	≥6.0
palisade parenchyma	≥6.0	≥6.0
spongy parenchyma	≥6.0	≥6.0
abaxial epidermis	≥11.3	≥6.0
Chloroplast crystals in		
palisade parenchyma	≥11.3	54.2
spongy parenchyma	≥11.3	54.2
Collapse of		
palisade parenchyma	≥17.9	≥17.9
spongy parenchyma	≥17.9	≥17.9
Glazing of		
adazial epidermis	≥21.9	≥17.9
abaxial epidermis	≥17.9	≥6.0
*When primary leaf tissue was sampled immediately after the 20 minute exposure to HCl, particulates were present in the vacuoles of abaxial epidermal cells from leaves treated with 11.3 or greater mg HCl/m^3, but only leaves exposed to 41.3 or 54.2 mg HCl/m^3 had abaxial epidermal cells with vacuolar particulates when the tissue was sampled 24 hours after the HCl treatment.		

. Source: Endress et.al., 1978

K.2.3 Reversible Fine Structural Alterations of Pinto Bean Chloroplasts Following Treatment with Hydrogen Chloride Gas

In this 1979 study, Endress et. al. examined the development of injury to cells following treatment with HCl gas and looked for reversible chloroplast alterations. Pinto bean plants that were 8, 12, and 16 days from seeding were exposed for 20 minutes to 6 to 54.2 mg/m^3 concentrations of gaseous HCl and examined at multiple intervals up to 24 hours. Tissue samples from the two primary leaves were prepared for electron microscopy.

Chloroplast structure was distinctly modified in all tissue samples. But not in all cells of each sample. A distinctive feature of the chloroplast appearance was the presence of crystalline structures. Crystals were not observed in cells treated with 6 mg/m^3 nor 54.2 mg/m^3 gaseous HCl. One percent of chloroplasts contained crystals in cells treated with 11.3 mg/m^3 with the frequency of crystals increasing rapidly above that concentration to all chloroplasts containing crystals after treatment at 41.3 mg/m^3 HCl. Recovery of chloroplasts was found when samples were observed at 0.5, 1, 2, 3 and 4 hours after treatment with 21.1 mg/m^3 HCl. The frequency of crystals declined from 65 percent of chloroplasts containing crystals immediately after treatment, to 15 percent at 0.5 hours after treatment, and no remaining crystals after 4 hours. Other authors have hypothesized that crystal formation reflects a generalized stress response. In the sample

cells characterized by severe plasmolysis, dehydration of chloroplasts and associated crystal formation irreparable cell membrane damage occurred. It is not clear whether the normal repair mechanism was inhibited by HCl exposure or if the magnitude of membrane injury was greater than the capacity of the repair process.

The authors compared their results from 20 minute exposures to work reported by Masuch et. al. (1973) on chronic exposures. They reported that chronic exposures of spinach to HCl gas (0.13 and 0.25 mg/m^3 for 43 hours within 5 days and 1.6 mg/m^3 for 208 hours within 14 days) increased the average frequency of changes in the chloroplasts, although they did not report the presence of crystals.

Altered mitochondrial appearance was one of the most consistent indicators of HCl exposure in this study. In tissues sampled at longer periods after exposure, the mitochondria retained their altered morphology. The authors were able to find qualitative structural differences between 8, 12, and 16 day old plants exposed to similar HCl concentrations with the greatest number in the 12 day old plants.

K.2.4 Peroxidase Activity in Plant Leaves Exposed to Gaseous HCl or Ozone

This Endress et.al. study (1980) was designed to determine if peroxidase activity was elevated in bean and tomato leaf tissues that did not show macroscopic injury after exposure to HCl and ozone gases. Perioxidase is an enzyme found in almost all higher plants and animals that is associated with cellular growth and development. Pinto bean plants 12 days from seeding and tomato plants 88 days from seeding were exposed for 20 minutes to multiple concentrations of gaseous HCl and ozone. The HCl concentrations used were 0, 4.08, and 12.52 mg/m^3. The treated plants were sampled immediately for peroxidase and at 24 and 48 hours for both peroxidase and macroscopic injury. Preparations for determining peroxidase levels included preparation of enzyme assay solutions and for polyacrylamide slab gel electrophoresis.

Of the four independent experiments (a) bean and HCl, (b) tomato and HCl, (c) bean and ozone, and (d) tomato and ozone, only the tomato plants exposed to ozone showed a concentration related significantly different level of perioxodase activity than the controls. Leaves were also scored at 24 and 48 hours after exposure for macroscopic injury symptoms. The visible injury was statistically related to the pollutant treatment in all case except HCl and tomato. Greater than 10 percent of bean leaves showed injury at an HCl exposure level of 4.08 mg/m^3, while approximately 75 percent showed injury and 20 percent showed necrosis at 12.52 mg/m^3.

Several previous studies of plants exposed to stressors including air pollutants have shown that increased total peroxidase activity or altered isozyme patterns are frequently induced. Others indicate that crop yields may be reduced by exposure to air pollutants, even though no discernable macroscopic injury symptoms were present. Total peroxidase activity appears unsuitable as a biomarker of latent injury.

K.2.5 Photosynthesis and Respiratory Consequences of Hydrogen Chloride Gas Exposures of Phaseolus Vulgaris L. and Spinacea Oleracea L.

The purpose of the Endress, et. al. 1982 study was to measure the photosynthetic and respiratory activities of plant leaf tissue following exposures of pinto bean plants at 8 to 16 days from seeding for 20 minutes to gaseous HCl concentrations of 3.3 and 45.4 mg/m^3. Locally

purchased spinach leaves were treated in the same manner. Visible foliar damage was estimated at 24 hours after exposure. Immediate sampling was completed on isolated chloroplasts in an assay solution to determine both light and dark oxygen evolution. Additionally, leaf discs were used to estimate photosynthesis and respiration rates.

Chloropyll levels sampled immediately following exposure were slightly higher in exposed plants than in controls. However, of the 8, 10, 12, 14, and 16 day old plants only the 14 day old sample showed a statistically significant increase. When plants were sampled 24 hours after treatment, chlorophyll levels were slightly less in the exposed plants than in the controls. Chloroplasts were sensitive to HCl gas exposure, but as the time between exposure and sampling increased many chloroplasts gradually recovered their normal appearance while a few others became totally disrupted.

Exposure to HCl that resulted in <15% necrotic leaf injury appeared to stimulate both photosynthesis and respiration. These rates decreased linearly with increased injury severity. Leaf discs sampled 24 hours after treatment generally exhibited greater rates of photosynthesis and respiration than those sampled immediately following exposure. Table K-6 presents the rates observed. No significant difference was found between control and exposed plants in oxygen evolution or consumption among the spinach plants except for the variety Bloomsdale. Significantly higher respiration rates at both sample times were exhibited by variety Bloomsdale.

Treated pinto bean chloroplasts exposed to HCl concentrations ranging from 9.5 to 21.8 mg/m^3 evolved less oxygen than controls. Significant reductions in oxygen evolution occurred following treatment with 14.9 or 18.5 mg HCl/m^3. Chloroplasts of spinach also exhibited reduced oxygen evolution. The 24 hour samples showed no recovery by from the initial depressed rates of oxygen evolution. Table K-7 presents the rates of oxygen evolution.

Foliar injury and photosynthetic rates for discs that were dipped in various concentrations of dilute liquid hydrochloric acid was comparable to with that from the gaseous HCl treatment. Oxygen evolution from isolated spinach chloroplasts was examined with regard to pH. Increased acidification of the reaction solution caused a linear inhibition of the oxygen evolving capability regardless of the acid used: HCl, H_2SO_4, or HNO_3. Unlike the leaf disc experiments, no recovery was observed in the 24 hour sample.

Table K-6. Comparison of Photosynthesis and Respiration Rates Exhibited by *S. oleracea*

***S. oleracea* (Spinach) Variety**	**% of Control**	
	0 hour	**24 hour**
	Photosynthetic Rate	
Melody	109	105
Bloomsdale	84	112
Avon	87	109
	Respiration Rate	
Melody	115	93
Bloomsdale	123*	142*
Avon	101	99
Leaf discs were taken from plants exposed either to 22.1±5.01		

S. oleracea (Spinach) Variety	% of Control	
	0 hour	24 hour
	Photosynthetic Rate	
mg anhydrous HCl /m^3 or carbon filtered air (control) for 20 minutes. Data are mean values of four experiments. Significance: * $P<0.01$ determined by Student's *t* test.		

Source: Endress, et. al. 1982

Table K-7. Oxygen Evolution of Chloroplasts Isolated from *P. vulgasis* and *S. oleracea*

Species	mg HCl /m^3	µl O_2 / hr-mg Chlorophyll		% of Control
		Control	HCl	
P. vulgaris	9.5	180.5	170.9	94.7
(pinto bean)	14.9	199.1	83.1	41.7*
	18.5	110.4	75.8	68.7*
	20.7	64.5	58.5	90.7
	21.8	81.1	63.3	78.4
S. oleracea	27.7	163.6	106.5	65.1***
(spinach var.	29.2	143.3	87.2	60.9**
Melody)	31.7	212.8	157.0	73.8*
S. oleracea	28.9	223.2	211.6	94.8
(spinach var.	29.0	199.5	174.0	87.2
Boomsdale)	30.4	261.3	209.5	80.2
Data presented are average of a minimum of five samples per treatment with carbon filtered air or anhydrous HCl at concentrations indicated. Significance: * $P<0.05$, ** $P<0.01$, *** $P<0.001$.				

Source: Endress, et. al. 1982

Photosynthetic CO_2 fixation in HCl treated pinto beans was followed by examining the activity of RubPCase. After exposure to HCl gas, experiments showed an initial sharp decrease in RubPCase activity followed by a continued but more gradual decrease. Low concentrations of HCl stimulated RubPCase and with minimal or no necrotic injury observed, but with either increasing severity of injury or HCl concentration, RubPCase activity decreased. Sampling 24 hours after treatment showed enzyme activity was not as depressed and recovery in samples exposed to 20 mg HCl/m^3 or lower.

K.2.6 Histological Effects of Aqueous Acids and Gaseous Hydrogen Chloride on Bean Leaves

This study (Swiecki et. al., 1982) was conducted to look for the possible mechanism of action for gaseous HCl phytotoxicity. From previous work, it was hypothesized that gaseous HCl condensed as aqueous acid on leaf surfaces due to HCl's high water solubility and the high humidity at the leaf boundary layer. This experiment compared injury symptoms following treatment with aqueous HCl or HCl gas and assessed whether the injury was attributable to H+, Cl-, or a combination of the two. Using 20 minute exposures to 12 day old pinto bean plants, aqueous acids' and chloride salts' effects were compared to the effect of gaseous HCl exposures of 14.5 to 19 mg/m^3.

Injury was observed in all leaves when observed 1 hour after exposure to dilute aqueous HCl concentrations and other dilute acids. For treatments exhibiting injury, the 24 hour observations differed from the 1 hour observations only by minor increases in visible injury. Equivalent concentrations of aqueous HCl, H_2SO_4, and HNO_3 produced essentially identical injury symptoms. Equivalent levels of injury were produced in leaves exposed to 0.06N (pH = 1.45) aqueous HCl or 15-30 mg gaseous HCl/m^3 for 20 minutes.

Injury from 12 day old pinto bean leaf aqueous HCl exposure was similar to injury the author had found in 8 day old leaves exposed to gaseous HCl. The dependency of injury susceptibility on tissue age was a generally agreed upon hypothesis and using microscopic features, this study found the only difference in age appears to be in the numbers of affected cells and not in the type of injury. At the levels tested, the authors found that effect of the chloride anion was inconsequential relative to the hydrogen ion concentration. Further, they found the generalized injury response to acid and particularly aqueous acid corresponds closely to injury caused by sulfate acid precipitation.

K.2.7 The Phytotoxicity of Designated Pollutants on Plant Species

Additional Air Force funded research on plant exposures to HCl and aluminum oxide was conducted at the University of California at Irvine (Granett, 1984). In this research aluminum oxide particulate matter was found to be nontoxic. The exposure of plants to HCl was conducted using acidic mist solutions These HCl solutions ranged from 0 to 5,000 ppm by volume, with measured pH ranging from 4.91 to 0.75. The researchers either sprayed the mist on the plant or soaked the plant in the solution, rinsed, and then checked for effects. Since the effects were compared to doses in terms of the pH of the solutions rather than air concentrations, we decided not to include these results in our development of ecological exposure thresholds.

K.3 Development of Gaseous HCl Ecological Exposure Thresholds

The series of studies described provide increasingly detailed information about gaseous HCl injury to plants from visual observations, photosynthetic and oxygen evolution rates, and electron microscopy of localized cellular damage following exposure. The hypothesis of the process for the damage remains consistent – gaseous HCl condenses on the leaf surface producing an aqueous acid solution that promotes cellular injury. Degree of injury is proportional to exposure to gaseous HCl and this injury is a response to exposure to an acid rather than being specific only to HCl. Unfortunately, the data was developed to determine the impact of exposure to gaseous HCl from short-term high concentration exposures. While this data can be extrapolated for use in developing acute ecological exposure thresholds, more uncertainty is involved in extrapolating the data to develop chronic ecological exposure thresholds. As a result, one might look to the literature on acid rain injury to increase the data available for use in developing screening-level chronic ecological exposure thresholds. The results of the studies, emphasizing the more conservative results are summarized in Table K-8.

Table K-8. Summary of Studies from Literature Review of Gaseous HCl and Foliar Damage

Study	Summary

Study	Summary
Phytotoxicity of Hydrogen Chloride Gas with a Short-Term Exposure (Lerman et. al., 1976)	After 20 minute exposure, 10% damage to most sensitive 1 of 8 plant types at 6.5 mg/m^3. Changes in 7 of 8 types at lowest concentration - 1 mg/m^3. (Plant types: aster, calendula, centaurea, cosmos, dwarf marigold, marigold, nasturtium, and zinnia.)
Foliar and Microscopic Observations of Bean Leaves Exposed to Hydrogen Chloride Gas (Endress et.al., 1978)	After 20 minute exposure to pinto bean plants, damage found at lowest exposure concentration of 6 mg/m^3. At 25 mg/m^3 10% showed necrotic lesions.
Reversible Fine Structural Alterations of Pinto Bean Chloroplasts Following Treatment with Hydrogen Chloride Gas (Endress et.al., 1979)	After 20 minute exposure to pinto bean plants, no observed adverse effects at lowest exposure concentration of 6 mg/m^3. 1% damaged after 20 minutes exposure to 11.3 mg/m^3.
Peroxidase Activity in Plant Leaves Exposed to Gaseous HCl or Ozone (Endress et.al., 1980)	After 20 minute exposure to bean and tomato plants, increase in peroxidase activity found at lowest exposure concentration of 4 mg/m^3. 25% necrotic or injured bean leaves at 4 mg/m^3. 10% tomato necrotic or injured at 12.5 mg/m^3.
Photosynthesis and Respiratory Consequences of Hydrogen Chloride Gas Exposures of Phaseolus Vulgaris L. and Spinacea Oleracea L. (Endress et.al., 1982)	After 20 minutes exposure to gaseous HCl concentrations of 3.3 and 45.4 mg/m^3, measured photosynthetic and respiratory activities of spinach and pinto bean plants at 8 to 16 days from seeding. Exposure to HCl that resulted in <15% necrotic leaf injury appeared to stimulate both photosynthesis and respiration. These rates then decreased linearly with increased injury severity. Bean plants exposed to HCl concentrations ranging from 9.5 to 21.8 mg/m3 evolved less oxygen than controls with significant reductions following treatment with 14.9 or 18.5 mg HCl/m3. Similar reduction for spinach. No recovery in 24 hour samples.
Histological Effects of Aqueous Acids and Gaseous Hydrogen Chloride on Bean Leaves (Swiecki et. al., 1982)	After 20 minute exposure to pinto bean plants, damage found at lowest exposure concentration of 15 mg/m^3.
Cited in (Lerman et.al., 1976)	Haselhoff and Lindau, 1903: Single exposure, 2 day duration at 5-20 ppm (8-30 mg/m^3). Seedlings of viburnum and larch killed in less than 2 days
Cited in (Lerman et.al., 1976)	Shriner and Lacasse, 1969: Single exposure, 2 hour duration at 5 ppm (8 mg/m^3)28 day old tomato plants developed interveinal bronzing followed by necrosis within 72 hours
Cited in (Lerman et.al., 1976)	Means and Lacosse, 1969: Single exposure, 4 hour duration at 3-43 ppm (5-65 mg/m^3) to 12 types of 2-5 year old coniferous and broadleaf seedlings: most sensitive broadleaf (tulip tree) visible injury at 3 ppm (5 mg/m3); most sensitive conifer (white pine) visible injury at 8 ppm (12 mg/m3) No injury to least sensitive Thuja occidentalis (white cedar) at 43 ppm (65 mg/m3).

Study	Summary
Cited in (Lerman et.al., 1976)	Hulensberg, 1974: Single exposure, 29 hour duration, 0.5 mg/m3 (0.3 ppm) Carrots exposed 45 days after germination showed 32.2 to 49.7% decrease in crop yield; exposed 96 days showed only 5.3% crop yield decrease. Winter grape only leaf discoloration, radishes no damage. Tomato,cucumber and bush bean plants showed leaf damage and increase in leaf chlorides. Reduction in yield severe in tomato and slight in cucumber plants.
Cited in (Lerman et.al., 1976)	Masuch et. al., 1973: Exposure of spinach to HCl gas for 43 hours within 5 days at 0.13 to 0.25 mg/m^3 and for 208 hours within 14 days at 1.6 mg/m^3 resulted in changes in chloroplasts. Authors attributed differences between Masuch's study and theirs could be attributed to differences between acute and chronic exposures, differences in species characteristics, or both.

K.3.1 Recommended Methods for Developing Ecological Exposure Thresholds

The *EPA Guidelines for Ecological Risk Assessment* (EPA, 1998) discusses the use of a stressor-response analysis in characterization of ecological effects. Point estimates are frequently adequate for simple assessments or comparative studies of risk with a median effect level frequently used because the level of uncertainty is minimized at the midpoint of the regression curve. The guidance points out that a 50% effect level for an endpoint such as survival may not be appropriately protective for an assessment endpoint. Median effect levels can be used for preliminary assessment or comparative purposes especially when used in combination with uncertainty factors. Selection of a different effect level (10%, 20%, etc.) can be arbitrary unless there is some clearly defined benchmark for the assessment endpoint, making it preferable to carry several levels of effect or the entire dose response curve forward to risk estimation.

At the conservative end of the spectrum, EPA's Region 5 Superfund Office (EPA, 2007a) recommends the use of No-Observed-Adverse-Effect-Levels (NOAELs) for screening level ecological effects evaluations. [3] They suggest the NOAEL be from scientific studies that exposed the plants or animals to the chemical for a long time (chronic). Short- or medium-time exposure studies are less desirable because it may take a long time of exposure to a chemical in order for there to be an adverse effect. However, they point out that time should be measured relative to the life span of the plant or animal being studied. For a plant or animal with a short life span, it may only be necessary to have a relatively short study. They further guide the risk assessor to be consistently conservative in selecting literature values, to describe the limitations of using the data for the assessment, and to discuss the uncertainty before moving onto the risk calculation. Region 5 also has available a setoff ecological screening levels that include exposures to chemicals through air (EPA, 2003). Unfortunately, they have not developed values for HCl for any exposure medium.

[3] IRIS defines the No-Observed-Adverse-Effect Level (NOAEL) as the highest exposure level at which there are no biologically significant increases in the frequency or severity of adverse effect between the exposed population and its appropriate control; some effects may be produced at this level, but they are not considered adverse or precursors of adverse effects.

In the Department of Energy document providing benchmarks for soil to plant chemical exposures, *Toxicological Benchmarks for Screening Contaminants of Potential Concern for Effects on Terrestrial Plants: 1997 Revision* (Efroymson, et.al., 1997), growth and yield are selected as the two significant parameters because 1) they are the most common class of response parameters reported from phytotoxicity studies and 2) they are ecologically significant responses both in terms of the plant population and the ability of the vegetation to support higher trophic levels.

They recommend 20% reduction in growth or yield as the threshold for significant effects to be consistent with other screening benchmarks and with current regulatory practice. They justify the 20% level because most regulatory criteria are based on concentrations in toxicity tests that cause effects that are statistically significantly different than the controls. On average, those concentrations correspond to greater than a 20% difference effects. Additionally, in programs such as Superfund, regulatory actions may be based on comparisons of biological parameters measured on a contaminated site to those from reference sites. Differences between those parameters must be greater than 20% to be reliably detected in such studies. Therefore, the 20% effects level is treated as a conservative approximation of the threshold for regulatory concern.

Using the method for deriving soil benchmarks based on the National Oceanic and Atmospheric Administration's (NOAA) method for deriving the Effects Range Low (ER-L). This method has been recommended as a sediment screening benchmark by EPA Region 9. The ER-L is the 10^{th} percentile of the distribution of various toxic effects thresholds for various organisms in sediments. Justifications include that the phytotoxicity of a chemical in soil is a random variable, the toxicity of the soil at a given site is drawn from the same distribution, and the assessor should be 90% certain of plants growing in that soil. Analogously, site-specific atmospheric conditions, including the concentrations of other pollutants, would affect the phytotoxicity of a chemical in air in the same manner.

The 10^{th} percentile phytotoxicity benchmarks are derived by rank ordering the Lowest-Observed-Effects-Level (LOEL)[4] values and selecting a number that approximates the10th percentile. If ten or fewer values are available for a chemical, the one with the lowest LOEC is used. Though the derivation of a benchmark through this method implies a significant impact on approximately 10% of the species, the authors defend their level of conservatism because: 1) the benchmarks are for the community level and a loss of 10% of the community species is likely acceptable and 2) the benchmarks derived by this method have proved to be conservative in practice.

Finally, the authors attempt to assign levels of confidence to the benchmarks:

1. Low Confidence – Benchmarks based on 10 or fewer literature values.
2. Moderate Confidence – Benchmarks based on 10 to 20 literature values.
3. High Confidence – Benchmarks based on over 20 literature values.

[4] IRIS defines the Lowest-Observed-Effect Level (LOEL or LEL) as the lowest dose or exposure level at which a statistically or biologically significant effect is observed in the exposed population compared with an appropriate unexposed control group.

Based on professional judgment, the authors confidence in a benchmark were lowered a level if the range of plant species is narrow or if the 10^{th} percentile is the lowest value tested and caused a greater than 30% reduction in the measured growth parameters.

K.3.2 Development of Screening-Level HCl Ecological Thresholds

As recommended in *EPA Guidelines for Ecological Risk Assessment*, we attempted to develop and carry several levels of effect forward to risk estimation in terms of multiple screening-level ecological exposure thresholds. However, with fewer than 10 studies to use in the development of a threshold, a more conservative approach of basing the threshold on the lowest LOEL was selected (Efroymson, et.al., 1997).

A first step in developing thresholds is to adjust from the exposure duration of the experiments to the desired acute and chronic exposure durations. Because of the time periods available from the modeling exercise, the acute or short-term exposure duration we are interested in is one-hour. The chronic or long-term exposure duration is one-year. To extrapolate from the many experiments using 20 minute exposure durations to the desired one-hour duration, and from the few longer term studies to an one-year duration, we will follow the recommendations of EPA's Office of Research and Development (EPA, 2007b) for human health exposure duration adjustments as described below.

Haber's Law (i.e., C x t = k, where C = concentration, t = time, and k = a constant) traditionally has been used to relate exposure concentration and duration to a toxic effect (Rinehart and Hatch, 1964). Specifically, the equation implies that exposure concentration or duration may be adjusted to attain a cumulative exposure constant (k) which relates to a toxic response of specific magnitude. Work by ten Berge et al. (1986), affirmed that chemical-specific relationships between exposure concentration and exposure time may be exponential rather than linear; i.e., the expression now becomes $C^n \times t = k$, where n represents a chemical-specific exponent. Upon examining the concentration and time relationship of the lethal response to approximately 20 chemicals, ten Berge et al. (1986) reported that the empirically derived value of n varied from 0.8 to 3.5. The magnitude of the exponent (n) provides insight into the relationship between exposure concentration and exposure duration such that if n = 1, the toxic response to the chemical is dependent solely upon total dose (i.e., a linear relationship, or Haber's Law). Generally, if n < 1, the exposure duration is the determinant of the toxic response and if n > 1, the exposure concentration is the primary determinant of the toxic response.

Ten Berge developed an exponent value of one for HCl. Thus for HCl a linear relationship exists and 20 minute exposure concentrations can be extrapolated to one-hour exposure concentrations by multiplying by 1/3 (20 minutes / 60 minutes). Because of the lack of data from long term exposures, we will use an uncertainty factor of 10 to extrapolate from acute to chronic exposure thresholds. This is a value EPA frequently uses in developing human health dose response values when long term studies are not available. Table K-9 presents the impacts at the lowest levels noted for potential use in establishing the screening-level thresholds. While potentially overestimating impacts to more resistant species, the level is more likely to be protective of all species, including those not studied.

Table K-9. Results of Gaseous HCl Studies for Use in Development of Screening-Level Thresholds

Selected Significant Impacts from 20 Minute Exposures		
1.5 mg/m^3 Lowest concentration in study Changes in 7 of 8 plant types **6.5 mg/m^3** 10% leaves damaged in 1 plant type (Lerman et.al., 1976)	**4 mg/m^3** Lowest concentration in study Increase in perioxidase activity 25% necrotic or injured bean leaves **11. 5 mg/m^3** 25% necrotic or injured tomato leaves (Endress et.al., 1980)	**9.5 to 21.8 mg/m^3** Lowest concentration in study Evolved less oxygen than controls **14.9 or 18.5 mg HCl/m^3** Significant reductions in oxygen evolution (Endress et.al., 1982)
Selected Significant Impacts from 2 to 4 Hour Exposures		
3 mg/m^3 Lowest concentration in study Visible injury to most sensitive broadleaf of 12 tree species after 4 hour exposure (Means and Lacasse, 1969; as cited in Lerman et.al., 1976)	**8 mg/m^3** Interveinal bronzing followed by necrosis after 2 hour exposure (Shriner and Lacasse, 1969; as cited in Lerman et.al., 1976)	**8-30 mg/m^3** Death to viburnum and birch seedlings after 2 hour exposure (Haselhoff and Lindau, 1903; as cited in Lerman et.al., 1976)
Selected Significant Impacts from Longer Exposure Durations		
0.13 to 0.25 mg/m^3 Changes in spinach chloroplasts after 43 hours exposure within 5 days (Masuch et.al., 1973; as cited in Endress et.al., 1979)	**0.5 mg/m^3** 32.2 to 49. 7% crop yield decrease 45 day old carrots exposed for 29 hours (Hulensberg, 1974; as cited in Lerman et.al., 1976)	**1.6 mg/m^3** Necrosis and chlorosis in spinach after 208 hours exposure within 2 weeks (Masuch et.al., 1973; as cited in Lerman et.al., 1976)

Two studies were selected to be used in establishing the LOEL and LOAEL for establishing screening-level phytotoxicological thresholds for HCl. The Lerman, et.al. study (1976) was determined to be most appropriate for estimating an LOEL. The 20 minute plant exposure to the lowest concentration in the study, 1.5 mg/m^3, resulted in changes in 7 of the 8 plant types in the study. Adjusted to an one-hour exposure duration, the LOEL is 0.5 mg/m^3 or 500 $\mu g/m^3$. The Endress et. al. 1980 study was determined to be most appropriate for estimating an LOAEL. The 20 minute plant exposure to the lowest concentration in the study, 4 mg/m^3, resulted in 25% necrotic or injured pinto bean leaves. Adjusted to an one-hour exposure duration, the LOAEL is 1 mg/m^3 or 1,000 $\mu g/m^3$.

Because of the lack of ample data to statistically establish a ecotoxicological threshold, and the critical exposure concentrations were the lowest in the studies, we recommend using the more conservative LOEL value of 0.5 mg/m^3 for the short-term screening-level phytotoxicological threshold for HCl. Applying the factor of 10 extrapolation for using a short-term study to establish a chronic dose response value, our recommended long-term screening-level phytotoxicological threshold for HCl is 0.05 mg/m^3 or 50 $\mu g/m^3$.

Screening-level phytotoxicological thresholds for HCl based on Lerman, et.al. (1976):

Critical Effect	Point of Departure	Acute Threshold	Chronic UF	Chronic Threshold for
Leaf changes	LOEL (Adj): 0.5 mg/m^3	$5x10^{-1}$ mg/m^3	10	$5x10^{-2}$ mg/m^3

K.3.3 Comparison of Modeled HCl Air Concentration Estimates to Screening Level Ecological Thresholds

The highest one-hour average HCl air concentration of HCl modeled was compared to the ecological threshold developed. This maximum one-hour air concentration estimate is 2 mg/m^3. To calculate the hazard quotient (HQ) for foliar damage, the air concentration estimate is divided by the screening level ecological threshold. In this case, a short term screening level ecological HQ of 4 is calculated for potential foliar damage (2 mg/m^3 / 0.5 mg/m^3 = 4).

An HQ > 1 indicates that there is the potential for foliar damage to plants from the estimated air concentration. However, due to the lack of data, this relates to potential individual leaf damage rather than the 20% reduction in growth or yield recommended as the threshold for significant effects. Thus, the HQ resulting from the screening level ecological threshold is a conservative value and a value so near to 1 cannot be construed to mean that significant ecological damage would be anticipated. Rather, because in addition to the use of a conservative threshold due to lack of data coupled with the highest one-hour average air concentration modeled, it is more likely that HCl would not cause significant effects to exposed plants.

The comparison of long term average modeled air concentrations is discussed in another section. Because the long term screening level ecological threshold is greater than the reference concentration (RfC) used to assess noncancer adverse health effects, the RfC is protective of both human health and of potential foliar damage and estimating a long term HQ for ecological effects is not necessary.

K.4 References

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Appendix L: Statistical comparison of monitored and modeled ambient benzene concentrations near petroleum refineries in Texas City, TX

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L-1. Introduction

The risk assessment performed by the EPA for the petroleum refinery source category to support its proposed rulemaking regarding residual risks has been criticized by many, primarily those who suggest that the emissions estimates used as the basis for the risk assessment are too low by a factor of 10 to 100. EPA has countered this criticism by utilizing an extensive review process, including multiple elements of public and expert review, to develop the inventory of emissions and source information used in its risk assessments.

This analysis compares ambient monitoring data for benzene from two monitoring sites near two petroleum refineries in Texas City, TX to dispersion modeling results for those facilities. The monitors were selected for this exercise because of their proximity to large refineries and relatively complete datasets of hourly benzene measurements. We did this to assess the general magnitude of uncertainty, and the possibility of bias, in our facility-specific emissions estimates for benzene, recognizing that benzene exposures tend to drive total cancer risk estimates for refinery emissions and also that benzene emissions originate from many common sources (primarily mobile) besides refineries.

This case study illustrates both the utility and limitations of conducting such an assessment, and provides a general indication of whether our benzene emissions estimates for the two facilities in question are reasonable representations of actual benzene emissions during the monitoring year. It attempts to answer the question, "Are our benzene emission estimates truly low by a factor of 10 to 100 (at least for these 2 facilities), or are they close enough to be useful in residual risk decision-making?" We attempt to answer this last part keeping in mind the 2 order of magnitude range of MIR values embodied in the residual risk decision framework.

L-2 Methods

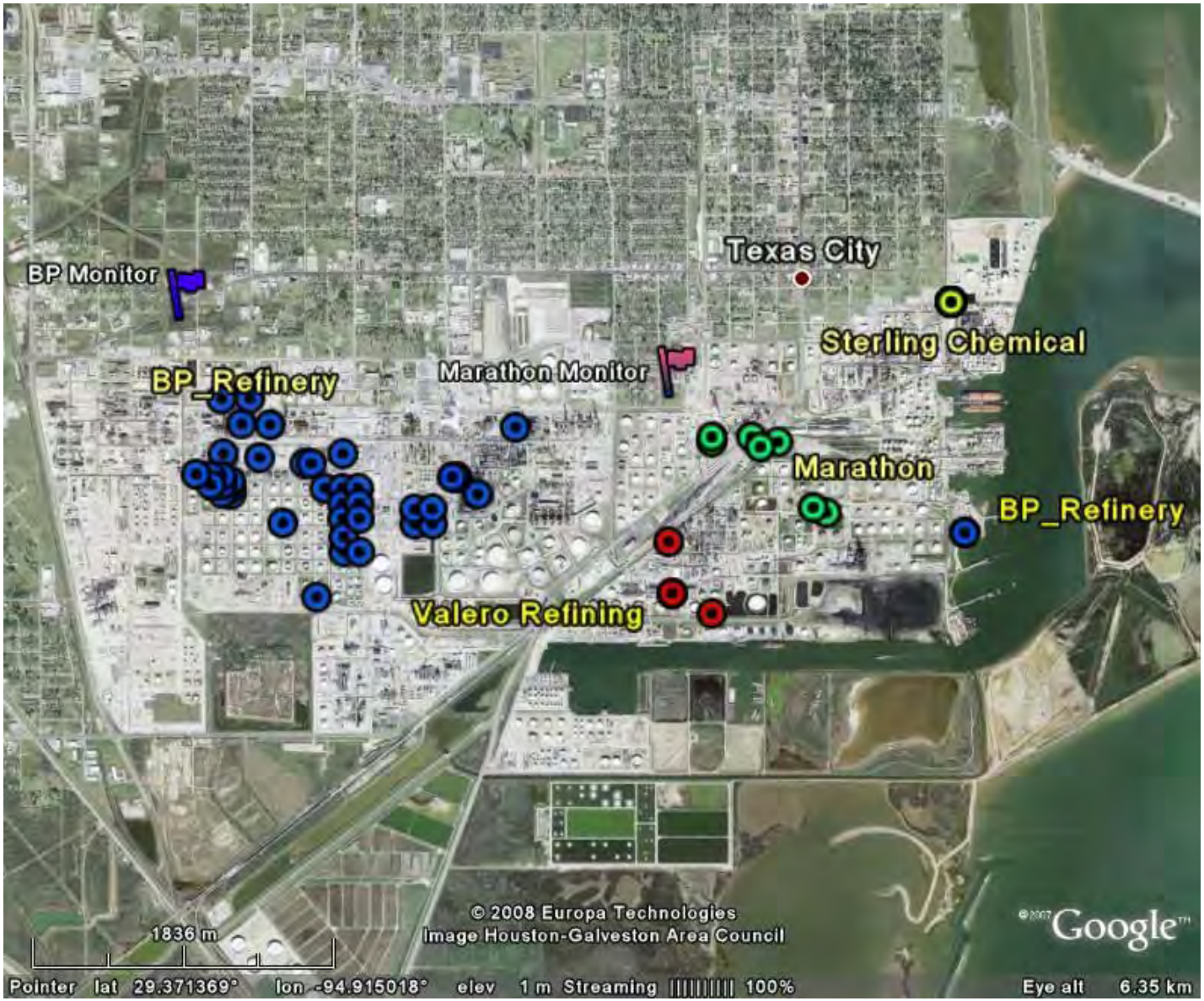
L-2.1 Monitoring sites

Benzene monitoring data were obtained from the Texas Commission on Environmental Quality (TCEQ) for two continuous flame ionization detection (FID) monitors located in Texas City, TX. The latitude, longitude, and dates for the monitors are provided in Table L-1. The FID monitors are the most common gas chromatograph monitors with reliable detection limits for volatile organic compounds (VOCs) such as benzene, toluene, and xylene (BTX). These two benzene monitors are each located within 300 meters of major industrial sources that emit benzene, including three large refineries (BP Refining, Marathon, and Valero Refining) and one chemical manufacturing facility (Sterling Chemical). Figure L-1 shows an aerial view of the study area.

Table L-1: Texas City, TX benzene monitors

Monitoring Site	Latitude	Longitude	Census Tract	Monitoring Period
BP-31st	29.381361	-94.940806	48167721900	June 1, 2003 to September 30, 2007
Marathon-Ashland	29.377	-94.9104	48167722400	October 1, 2004 to September 30, 2007

Figure L-1. Aerial photo of Texas City, TX.



L-2.2 Monitoring data

The BP monitor at 31st Street began collecting continuous monitoring data for benzene on June 1, 2003. The monitoring data were averaged to 1-hour values, with analytical results from 06/01/2003 to 09/30/2007, coupled with hourly measurements of wind speed and wind direction. The Marathon monitor at 11th Street began operation on October 1, 2004. The hourly monitoring data, also including wind speed and direction, included the period from 10/01/2004 to 09/30/2007. Table L-2 contains a summary of the annual average concentrations for these sites.

Table L-2: Monitored benzene concentrations – annual average

Monitor	Year	Avg. Conc (ug/m^3)	# Hours
BP-31st	2004	5.41	7716
BP-31st	2005	8.74	7454
BP-31st	2006	5.67	7634
BP-31st	All Hours	5.74	33205
Marathon-Ashland	2005	7.47	5377
Marathon-Ashland	2006	6.81	5817
Marathon-Ashland	All Hours	7.17	16860

The raw hourly ambient data were evaluated and adjusted so that non-detected (ND) values were replaced with ½ the minimum detection limits (MDLs). In addition, measurements that lacked matching hourly wind directions were omitted, in order to support a statistical analysis of directional source contributions at the monitor. These adjustments had little effect on the annual averages. For example, the adjustments caused the annual average benzene concentration at the Marathon monitor for 2006 to decrease from 6.81 to 6.72 μg/m^3, or about 1.3%.

L-2.3 Modeling data

Modeling results developed for the petroleum refinery source category at the census tracts where the monitors are located indicate that benzene is responsible for over 90% of the estimated cancer risk associated with these three petroleum refineries. The monitors are located within 200 meters of residential areas and are relatively close to the locations where the MIR for the BP refinery and the Marathon refinery were identified based on the RTR modeling. In addition, the RTR modeling results for each monitor indicated that the modeled benzene concentrations at the BP monitoring site were overwhelmingly (greater than 99%) influenced by emissions from the BP facility, and that modeled benzene concentrations at the Marathon monitoring site were predominantly (greater than 85%) influenced by emissions from the Marathon facility, with the remaining influence coming largely from Valero.

L-2.4 Model to monitor comparison

To compare modeled refineries emissions data from the RTR database (referred to as "modeling data") to the ambient monitor concentrations (referred to as "monitoring data"), we had to prepare the emissions data from the three refineries and one chemical

plant in the vicinity of the two monitors. We assumed constant hourly emissions and developed estimates of hourly ambient concentrations using dispersion modeling that was largely based on the RTR modeling, with adjustments to emission rates from each individual emission point made by multiplying them by the ratio of the total TRI emissions from that facility in the monitor year to the TRI emissions from that facility in the base RTR modeling year. We focused the comparison on 2004 data at the BP monitor and 2006 data at the Marathon monitor. We chose 2004 for the BP monitor because the initial RTR modeling run for the BP refinery was based on emissions data from the year 2004, affording us the opportunity to perform a direct comparison. For the Marathon monitor, we chose not to use data from 2005 because a large explosion and fire occurred at one of the refineries in 2005, altering annual benzene emission levels for 2006 in an unknowable way. Although the original RTR modeling run for the Marathon refinery was based on emissions data from 2002, we revised that modeling run for this analysis by scaling benzene emissions using TRI information for 2006 (*i.e.*, adjusting by the ratio of 2006 benzene emissions to 2002 benzene emissions) and using meteorological data from 2006.

Meteorological data were available from the Texas City Ball Park (just north of the refineries) and the Galveston airport (about 14 km SSE of the refineries). Both stations exhibited southerly winds. However, given that the emission sources were south of the BP and Marathon monitors, the Galveston winds were considered more representative of the area because of the more open exposure of the Galveston instrument tower, especially from the south. Galveston was less affected by obstacles around the tower. Also, when comparing 2004 and 2006, the Galveston winds are more consistent in direction, a general southeast direction, while the ball park shifts from predominantly south in 2004 to south and southeast for 2006. The variations in wind roses for the Ball Park site between 2004 and 2006, given the consistency of the patterns at Galveston for those two years, may be indicative of an exposure problem for the Ball Park site resulting in very localized influences. For these reasons the modeling was conducted using data (including high-altitude data) from the Galveston airport.

We conducted one AERMOD run to develop hour-by-hour estimated benzene concentrations at the BP monitor site for the year 2004 using 2004 emissions data for the BP refinery, 2002 emissions data from Marathon, Valero, and Sterling (adjusted to 2004 using TRI activity indices), and meteorological data from the Galveston airport for 2004. We conducted a second AERMOD run to simulate hourly benzene concentrations at the Marathon monitor site for the year 2006 using emissions data from Marathon, Valero, and Sterling from 2002 (scaled to 2006 using TRI activity data), and hourly meteorological data from the Galveston airport for 2006. The BP refinery emissions were omitted from the Marathon monitor site comparison due to the 2005 explosion, which disrupted activities at the BP refinery in 2006. All modeling options were identical to those used in the RTR baseline petroleum refinery assessment modeling.

In addition to preparing the emissions data, we also adjusted the monitoring data to focus our analysis as specifically as possible on the benzene contribution from petroleum refineries. To help characterize the impact of benzene emissions from the petroleum

refineries on each of the monitors, we first estimated the contribution at each monitor that could be attributed to unmodeled sources such as mobile and area sources. To do this, we extracted estimates of the ambient benzene concentration contributions for all other sources besides the major industrial sources (this included area sources, mobile sources, and long-range transport) from the 2002 National Air Toxics Assessment (NATA)[1] at each of the census tracts within 20 km of the monitors. We created an isopleth map of these contributions to develop estimates at each of the monitors (Figure L-2), giving us an annual background benzene concentration estimate of 1.0 $\mu g/m^3$ at the BP monitor and 1.4 $\mu g/m^3$ at the Marathon monitor. These background estimates were subtracted from the individual monitor data, thereby limiting the comparison between modeled estimates and measurements to contributions from the refineries. We recognize that the contribution from background sources can vary on an hourly basis, and that this simplistic approach cannot be valid for any time scale less than annual. Thus, a great deal of variation in monitored data may be caused by changes in background contribution.

Since our initial modeling determined that the BP monitor is overwhelmingly influenced by benzene emissions from the BP facility (greater than 99%), we use the results of our comparisons to derive inferences about the BP emissions inventory. Since our initial modeling determined that the Marathon monitor was predominantly influenced by benzene emissions from the Marathon facility (greater than 85%), we use the results of our comparisons to derive inferences about the Marathon emissions inventory. We recognize that this can lead to greater uncertainties regarding the interpretation at the Marathon monitor relative to the BP monitor.

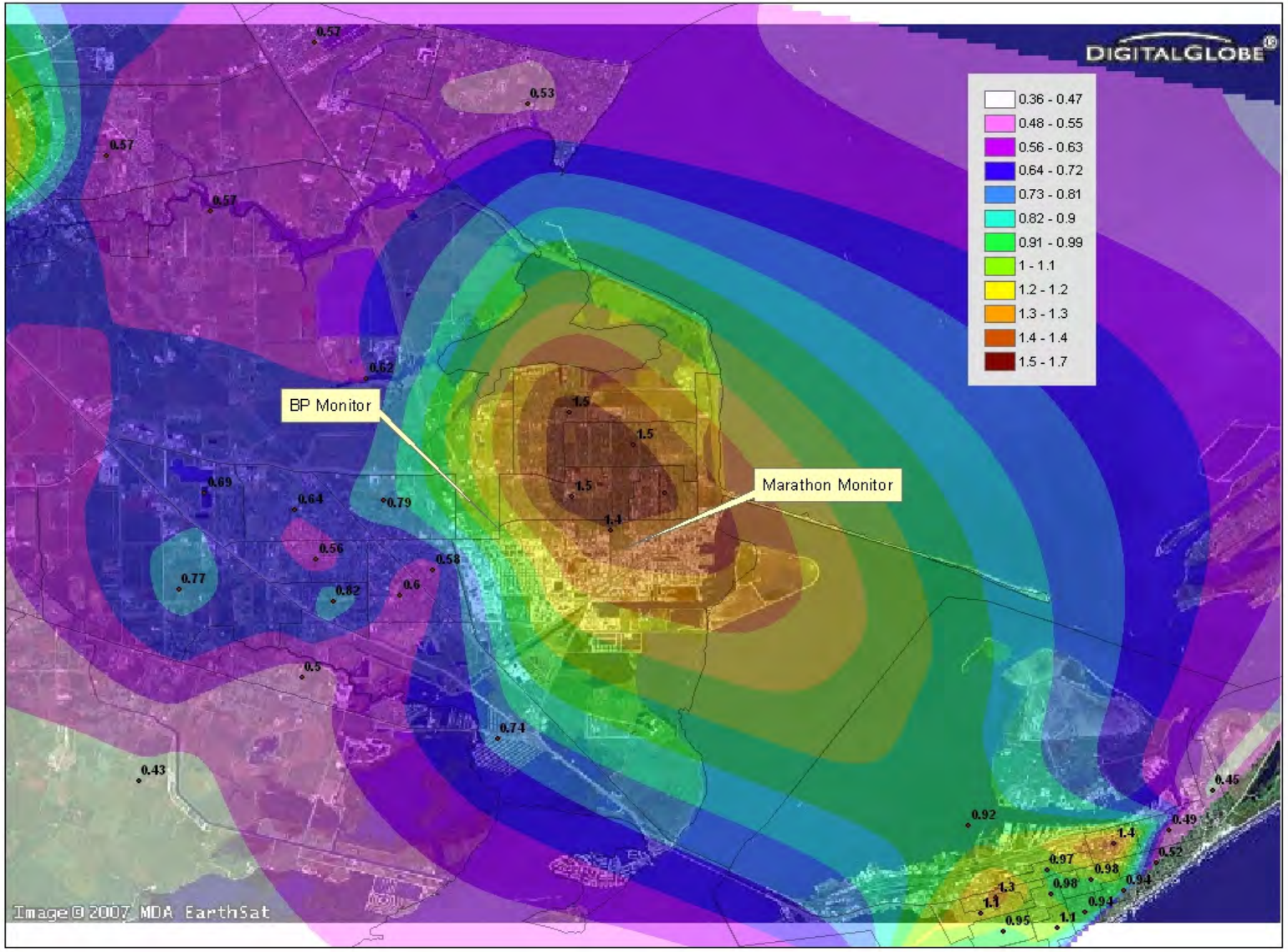
L-2.5 Statistical analyses

We used SAS software to perform an analysis of variance comparing mean modeled and monitored benzene concentrations among 16 equal wind direction sectors of 22.5 degrees each, numbered clockwise with sector #1 centered on zero degrees. (H_o: No difference exists in benzene concentration with wind sector; H_a: Benzene concentration varies with wind sector.)

We used Excel software regression analysis to assess the effects of wind sector by plotting average monitored and modeled concentrations and hourly monitor data by wind sector. We also examined if a regression existed between wind speed and the ratio of hourly monitored to modeled benzene concentrations, a measure of model error (H_o: Model error does not vary with wind speed; H_a: Model error varies with wind speed.) Separate regressions were run for each wind sector, and for the combination of wind sectors coming from the sources to the monitors. Finally, we developed frequency distributions of monitor-to-model ratios to examine possible short-term, high-emission events.

[1] 1999 NATA Tables – Pollutant Specific Database: http://www.epa.gov/ttn/atw/nata1999/tables.html

Figure L-2. Background benzene concentration isopleths (μg/m3), Texas City, TX.



L-3. Results and discussion

L-3.1 Comparison of annual average concentrations

A simple comparison of the annual average modeled concentrations at each monitor with their measured values (minus the estimated contribution of background sources) shows that, for the BP facility, the modeled estimate (4.0 $\mu g/m^3$) is lower than the measured value (4.5 $\mu g/m^3$) by only about 11%. This suggests that our annual benzene emission estimates for this facility are close to the actual values, potentially being underestimates by this same amount, 11%. This difference may also be due to other uncertainties, in our estimate of background source contributions, for example.

A similar comparison for the Marathon facility shows a greater difference, the modeled estimate of 2.1 $\mu g/m^3$ being lower than the corresponding monitored value of 5.5 $\mu g/m^3$ by about 72%. This suggests that our annual benzene emission estimates for this facility may be underestimated by 72%, *i.e*, low by a factor of 2.6.

Annual means for modeled estimates, monitor data, and the difference between them (Δ), are shown in Tables L-3 and L-4. Data were stratified by wind sector in the expectation that the monitors would be most strongly influenced by the BP and Marathon refinery emissions when the respective sources were directly upwind and less strongly influenced at other times. All three quantities varied significantly with wind sector at both monitors ($P<0.001$). Shaded cells in Tables L-3 and L-4 indicate that the monitor was downwind of the source. An analysis of variance comparing the means of hourly modeled and monitored concentrations (with results shown in the "P<" column) indicate that 28 out of 32 pairs of modeled and monitored annual means were significantly different at the $P<0.05$ level or less). The same results are shown graphically on Figures L-3 and L-4, below.

Table L-3. BP Monitor, 2004. Comparison of monitored and modeled means of hourly benzene concentrations, by wind sector (north=1).

Wind Sector	hours	Δ	Monitor μg/m3	Model μg/m3	P<
1	485	1.75679397	1.8424446	0.0856506	0.001
2	523	1.05785342	1.233489	0.1756355	0.001
3	349	1.2798939	1.506188	0.2262941	0.001
4	359	1.01127838	1.1558281	0.1445497	0.001
5	586	1.15972321	2.1760263	1.0163031	0.001
6	757	2.44873468	4.2204341	1.7716994	0.001
7	992	3.15899482	9.2269185	6.0679237	0.001
8	1091	-4.76722805	10.7390541	15.5062822	0.001
9	589	-0.15653462	4.3366263	4.4931609	0.754
10	189	1.19336501	1.5591725	0.3658075	0.023
11	136	0.17954026	0.7641936	0.5846534	0.205
12	132	0.60588579	1.0385461	0.4326603	0.001
13	160	0.8036979	0.9544022	0.1507042	0.005
14	233	0.72722058	0.8481373	0.1209167	0.001

Wind Sector	hours	Δ	Monitor μg/m3	Model μg/m3	P<
15	197	0.93016269	1.0969735	0.1668108	0.001
16	274	1.22653114	1.3211776	0.0946465	0.001

Table L-4. Marathon monitor, 2006. Comparison of monitored and modeled means of hourly benzene concentrations, by wind sector (north=1).

Wind Sector	hours	Δ	Monitor μg/m3	Model μg/m3	P<
1	549	1.93530075	1.9589727	0.02367193	0.001
2	387	0.50857501	0.5664341	0.0578591	0.001
3	213	1.00833775	1.0939155	0.08557775	0.001
4	220	0.84957914	5.1024727	4.25289359	0.578
5	389	5.93874823	6.6997172	0.760969	0.001
6	525	8.12713267	13.2994095	5.17227686	0.001
7	852	6.09892079	11.4159108	5.31699001	0.001
8	973	2.2703778	4.2261274	1.95574964	0.001
9	570	3.1886853	5.125814	1.93712874	0.001
10	166	4.48408789	4.9027831	0.41869524	0.001
11	117	3.25752179	3.5228034	0.26528162	0.001
12	74	1.67502297	1.8754324	0.20040946	0.001
13	124	2.10250782	2.1906774	0.0881696	0.094
14	156	0.34009731	0.410141	0.07004372	0.001
15	138	1.61167471	1.7061449	0.09447022	0.023
16	199	0.6831391	0.7269548	0.04381568	0.001

The effects of both refineries can be clearly seen as elevated benzene levels in both the measured and modeled concentrations. Although the differences between modeled and monitored results were statistically significant, the magnitude of the differences may not be important from a policy perspective, namely the residual risk decision framework which allows for some amount of uncertainty in the assessments by its design.

For the BP monitor, the absolute difference between average measured and modeled concentrations for 13 of 16 wind sectors was less than 2 $\mu g/m^3$. Wind sectors 7 and 8, where the BP monitor was downwind of the BP facility and concentrations were highest, had the largest model-to-monitor variation. While modeling of sector 7 resulted in an underestimate relative to monitored data, sector 8 showed an overestimate. In contrast to the results for all wind sectors combined, for wind sectors 7 through 9 the BP monitor concentrations averaged 0.81 $\mu g/m^3$ (9.1 %) *less* than the modeled estimates; one explanation for this finding may be that the 2004 emissions inventory for the BP facility was overstated.

For the Marathon monitor, mean differences between modeled and measured concentrations were less than 2 $\mu g/m^3$ for eight of the 16 wind sectors. For wind sectors 5 to 7, where the Marathon monitor was downwind of the Marathon facility and concentrations were highest, modeled estimates appear to underestimate monitored concentrations by an average of 6.7 $\mu g/m^3$ (a factor of 2.6, agreeing closely with the results for all wind sectors combined). One explanation for these results may be that the

2006 emissions inventory for the Marathon facility may have understated actual emissions by 2.6-fold.

Figure L-3.

BP Monitor, 2004:
Mean Modeled and Monitored Benzene Concentrations
by Wind Sector

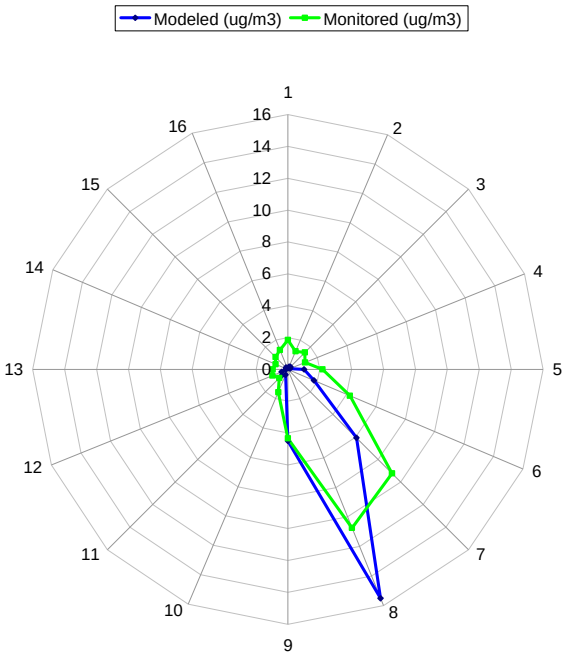
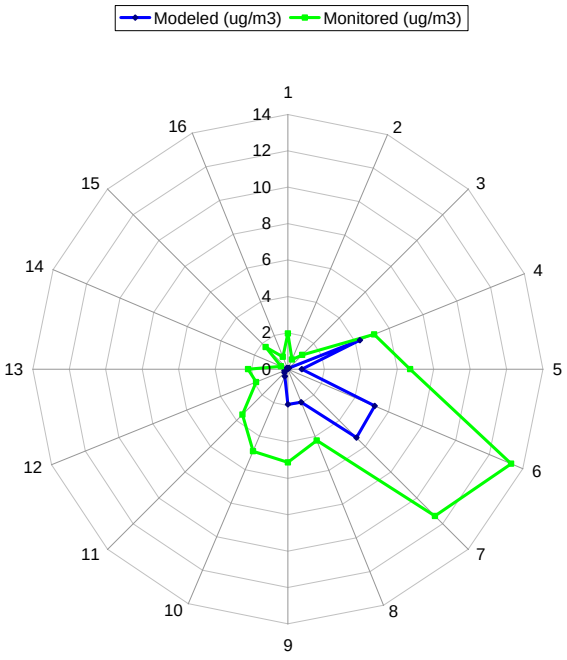


Figure L-4.

Marathon Monitor, 2006:
Mean Modeled and Monitored Benzene Concentrations
by Wind Sector



L-3.2 Comparison of hourly concentrations

Even if the inventories for both facilities and the dispersion modeling were perfectly accurate, we could not expect the hourly monitor data to match the hourly modeled estimates. This is because the emissions inventory is composed only of annual emission data, and contains no information on hourly variations in emissions. Furthermore, the monitored data were adjusted only for average background, not hourly background. Thus, the modeled estimates capture only variation in meteorological effects, whereas the monitored data capture variation in meteorology, emission rate, and hourly background variation for which we could not compensate.

For this reason, Figures L-5 and L-6 compare hourly monitor data with annual average modeled estimates for each wind sector. Figure L-5 (for the BP site) shows approximately the same number of points on either side of the 1:1 correlation line, suggesting little overall bias. However, the 1:1 line does not appear to fit the data well, suggesting that the model tends on average to underestimate lower monitor concentrations (*e.g.*, 1 $\mu g/m^3$) and overestimate higher ones (*e.g.*, 5 $\mu g/m^3$). This may be a result of the simplistic background adjustment of the monitor data or contributions from other nearby refineries. Short-term variations in background may have caused many of the high monitor readings at the lower right of Figure L-5.

The same trend appears in Figure L-6 (for the Marathon site). However, this figure also shows that most monitor data points are below the 1:1 correlation line, suggesting an overall low bias for the model consistent with Figure L-4 above. This low bias appears most prominently where the model predicted low concentrations, *e.g.*, less than 1 $\mu g/m^3$, but the monitor measured levels above 10 $\mu g/m^3$, presumably when the monitor was not downwind of the Marathon refinery. As with Figure L-5, the average background adjustment method or contributions from nearby refineries may have contributed to this effect, but Figure L-4 suggests that the model was also biased low.

L-3.3 Regression analysis of hourly concentrations and wind speed

Regression analysis of hourly monitor-to-model ratios (Figures L-7 and L-8, below) shows that the relationship between measured and modeled concentrations at both monitors was significantly correlated with wind speed. As wind speed increased at each location, the tendency for the model to underestimate the measured concentration increased, reaching more than tenfold at the highest wind speeds. Both the regression slope and R^2 values increased when source was directly upwind of the monitor, vs. the regressions for winds from all sectors, showing that this effect was somewhat stronger when the monitor was directly affected by the source.

L-3.4 Hourly monitor-to-model ratios and short-term events

Hourly monitor data were divided by corresponding hourly modeled estimates to develop hourly monitor-to-model ratios. Figures L-9 and L-10 show frequency distributions of these ratios for each monitor, at times when the monitor was downwind of the source. Both distributions are approximately log-normal, but with somewhat exaggerated tails on the low side. Figure L-9 shows that the mode of the distribution for the BP site occurs at a 1:1 ratio (mean = 1.2, median = 0.73), further supporting the suggestion that the modeled estimates were reasonably unbiased. The mode of the distribution for the Marathon site (Figure L-10) occurred at a monitor-to-model ratio of 2.5 (mean = 2.7,

median = 1.9), consistent with a 2.6-fold underestimate by the model suggested by the ANOVA results above.

Possible short-term, high-emission events at either facility, representing periods when the emission rate substantially exceeded the annual average, should appear on Figures L-9 and L-10 as the highest monitor-to-model ratios. In screening for potential acute risks, OAQPS uses a default assumption that the maximum hourly emission rate may exceed the annual average rate by tenfold, so that the location with the greatest modeled 1-hour concentration (based on average emissions but hourly meteorology data) may experience a concentration ten times higher than modeled. Therefore, the number of monitor-to-model ratios above ten is of interest. For the BP site, 0.8% of the ratios (16 of 1984) exceeded ten, and the highest was 20. For the Marathon site, 2.7% of the ratios (35 of 1277) exceeded ten, and the highest was 15. The possible contribution to this result by short-term variations in both background and contributions from other nearby refineries is not known, but could be important.

Narrowing this comparison to the highest concentrations at the monitor, application of the OAQPS default acute screening method (*i.e.*, using the peak-to-mean emission factor of 10, in combination with hourly meteorology data) at the BP facility results in an estimate of a peak hourly benzene concentration at the monitor of 2140 $\mu g/m^3$, whereas the maximum measured hourly concentration at the monitor was only 130 $\mu g/m^3$, more than 16 times lower than our screening estimate. This suggests that our screening approach for this facility is very conservative, and that peak emission rates for this facility may not vary much from their average values. Application of the default screening method at the Marathon facility results in an estimate of a peak hourly benzene concentration at the monitor of 960 $\mu g/m^3$, whereas the maximum measured hourly concentration at the monitor was only 275 $\mu g/m^3$, lower by a factor of about 3.5. This suggests that our screening approach is also conservative for the Marathon facility, but less so than for the BP facility. If, however, we had adjusted our annual emission estimates for the Marathon facility to remove bias (*i.e.,* based on the analysis shown above), the conservatism of our screening methodology goes back up, with our estimate of peak hourly benzene concentration being 7 times greater than the peak value actually measured at the monitor.

This result suggests that the tenfold default assumption captured a very high percentage (though not all) of short-term emissions events at these facilities, but was more than sufficiently conservative in screening the highest hourly concentrations.

L-3.5 Summary

Modeled concentrations averaged about 11% less than measured concentrations at the BP facility, but about 72% less at the Marathon facility. When this comparison was stratified by wind sector (16 sectors of 22.5 degrees each), 28 of 32 model-to-monitor pairs were significantly different (at the $P<0.05$ level or less). For 26 of the 28 significant results the monitored concentration exceeded the modeled estimate. Limiting the comparison to periods when the monitors were downwind of their respective sources, monitor concentrations averaged 0.81 $\mu g/m^3$ (9.1 %) *less* than the modeled estimates at the BP site, but 6.7 $\mu g/m^3$ (260%) more than modeled estimates at the Marathon site. Given that both monitors were modeled with similar input data and uncertainties, except for the emissions data and the calendar year, these results suggest that the inventory for the BP refinery may be reasonably accurate and possibly slightly overestimated, but the

inventory for the Marathon site may be somewhat underestimated. However, these discrepancies are within the range of those expected for such model-to-monitor comparisons, and may be the result of model error and inaccuracies in other model inputs.

Comparing hourly monitor data with annual average modeled estimates by wind sector, the model tended on average to underestimate lower monitor concentrations (*e.g.*, 1 $\mu g/m^3$) and overestimate higher ones (*e.g.*, 5 $\mu g/m^3$) at both sites. For the BP site (Figure L-5) the modeled results appeared unbiased, but for the Marathon site (Figure L-6) the modeled estimated appeared to be biased low, consistent with the comparison of means for all wind directions.

As wind speed increased at each location, the tendency for the model to underestimate the measured concentration increased, reaching more than tenfold at the highest wind speeds. This effect was somewhat stronger when the monitor was directly affected by the source. This result suggests that the model performs better at lower wind speeds, and is biased low at higher wind speeds.

Frequency distributions of monitor-to-model ratios (Figures L-9 and L-10) provide further support for the suggestion that the modeled estimates were reasonably unbiased at the BP site, but biased low for the Marathon site.

Only 0.8% of monitor-to-model ratios at the BP site exceeded ten, but 2.7% exceeded 10 at the Marathon site. The maximum ratios were 20 and 15, respectively. However, none of these ratios exceeded 10 when monitor concentrations were highest, suggesting that the OAQPS approach for screening short-term emissions and exposures was very conservative at both facilities, and that refinery emissions do not vary dramatically in time. This conservatism would be further increased if the Marathon emissions inventory did indeed prove to be underestimated, and was corrected.

L-3.6 Uncertainty

1. Inventory data. The RTR inventory may contain errors in amounts, locations, or release parameters that would affect the dispersion modeling results. In particular, activity at the BP refinery was disrupted during 2006, but nevertheless emitted an unknown amount of benzene that may have influenced monitored concentrations at the Marathon monitor site. Also, benzene emissions from shipping activities, roadways, and more distant industrial sources may have influenced both monitors, although the background adjustment was applied with the intent of removing this effect from the analysis.

The inventory is limited to annual emission rates, and lacks any information on short-term variations. Furthermore, it does not include emissions from upset conditions or emergency releases that the monitors may have measured. Finally, the inventory data for one of the two facilities had to be adjusted because an explosion altered its emissions.

2. Background adjustment. We estimated the average contribution of unmodeled sources and subtracted it from the monitor data to improve the comparison with modeled estimates. A model-to-monitor comparison done as part of NATA (http://www.epa.gov/nata/mtom_pre.html) found that the median model-to-monitor ratio at 87 sites was 0.93, that 89% of ratios were within a factor of 2 and 59% were within

30%. Although the comparison results for benzene were better than for any other HAP, this amount of uncertainty must still be considered substantial, and it is likely that the background adjustment contributes to it.

In addition, adjustment for short-term background variations was not possible, adding further uncertainty to short-term model-to-monitor comparisons.

3. Monitor data. As with any study involving sampling and analysis, the monitor data are subject to both sampling and measurement error. The monitors were also affected by unmodeled (background) benzene sources, including vehicles and industrial facilities outside the modeled domain. Some adjustment of the monitor data was also necessary: hours that lacked wind data were dropped, and non-detect results were entered as one-half the method detection limit.

4. Meteorological data. While the meteorological data station at the Galveston airport was considered to be more representative for this application of AERMOD given the focus on southerly winds, the assessment for a routine application of AERMOD for the same facilities including receptors for all directions around the emission sources may lead to a different conclusion. We recognize that selecting the most appropriate meteorological data for coastal locations is challenging.

Meteorological data are also subject to sampling and measurement errors, and they may fail to accurately represent conditions for every hour monitored.

5. Other modeling inputs. (a) We relied on information supplied by the American Petroleum Institute for stack and other release parameters, but we did not independently verify these data. (b) For BP Chemical, BP Refinery, and Marathon Refinery, the TRI indicates that a large fraction of benzene releases are fugitive emissions, which may drive much of the concentration picked up at the two nearby monitoring sites. (c) We believe that the ways we used to adjust emissions across years were reasonable, but they are still uncertain. (d) The modeling did not consider topography or building downwash. Each of these sources of uncertainty will be reflected in the results, but the aggregate error and direction of potential bias, if any, are unknown.

Of these uncertainties, the most important are probably those associated with the inventory and the adjustment for background. In particular, the lack of short-term variability in both these databases effectively limits the input variation available to the model, and prevents it from fully reproducing the monitor results. This effect limits our ability to draw conclusions.

Nevertheless, several trends seem clear. It appears that the modeling effort represented one facility reasonably accurately but underestimated the other by more than twofold. There is no way to know which (if either) facility is representative of the whole sector. The model tended to overestimate low monitored concentrations and underestimate high ones, perhaps not surprising since the model captured only some of the sources of variability in the monitor data. The model's tendency to underestimate high monitor levels increased with wind speed, and the increase was more pronounced when the source was directly upwind. Despite this tendency, however, we found that the OAQPS acute exposure screen (which assumes ten times the annual emission rate, worst-case meteorology, and a receptor at the monitor) was protective for these facilities by a substantial margin.

L.4 An Alternative Viewpoint

One EPA staff reviewer of this document, Fred Talcott, disagreed with some of the methods used in this model-to-monitor analysis and some of the conclusions reached by the authors of this Appendix. Since we did not have time to reach a consensus view on this issue within EPA or conduct some of the additional analyses suggested by Fred, in Attachment L-1 we present his comments and suggestions as an alternative viewpoint for the SAB panel to consider as they develop their comments on this analysis and its interpretations.

Figure L-5.

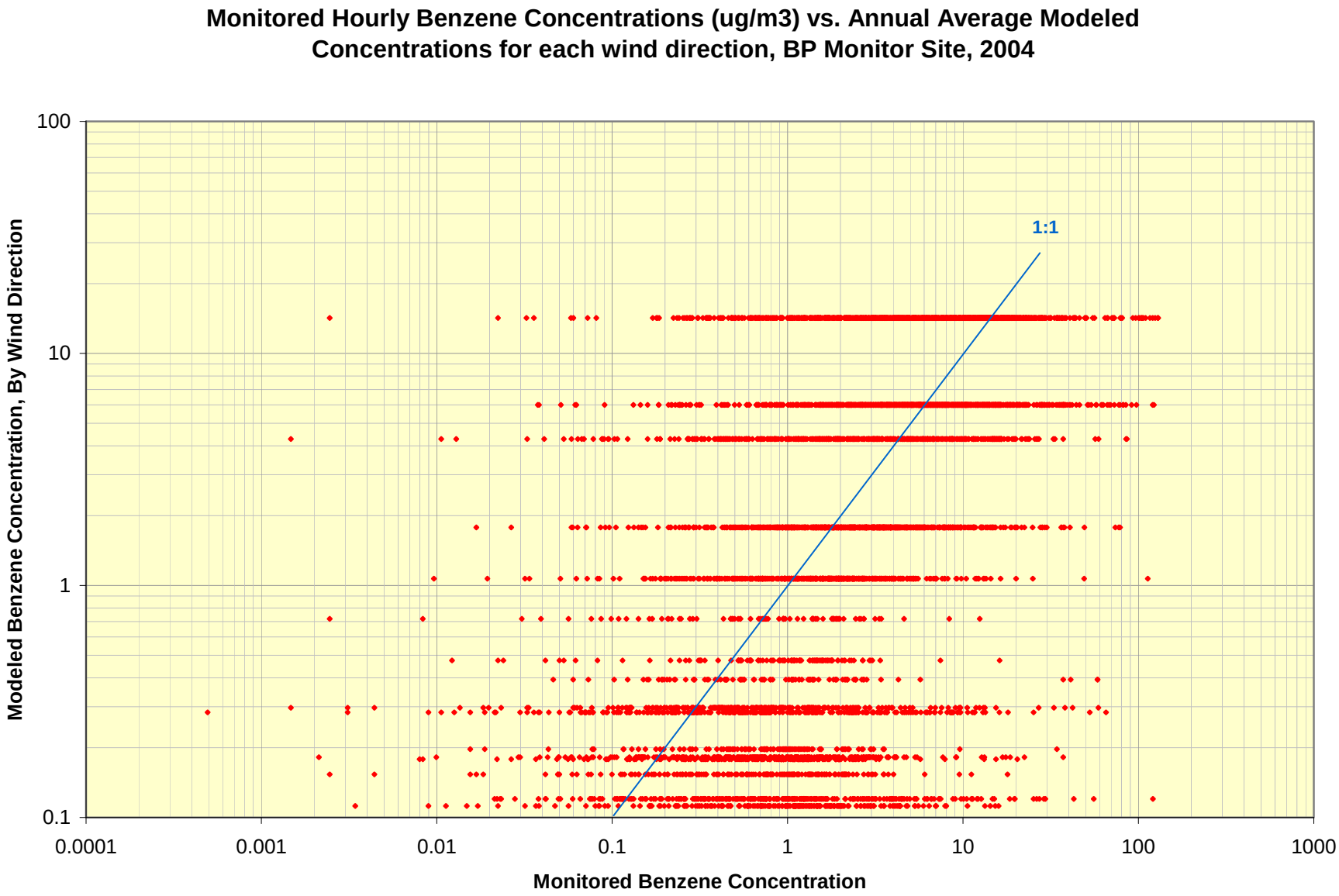

Monitored Hourly Benzene Concentrations (ug/m3) vs. Annual Average Modeled Concentrations for each wind direction, BP Monitor Site, 2004
Modeled Benzene Concentration, By Wind Direction
100
10
1
0.1
1:1
0.0001
0.001
0.01
0.1
1
10
100
1000
Monitored Benzene Concentration

Figure L-6.

Monitored Hourly Benzene Concentrations (ug/m3) vs. Annual Average Modeled Concentrations for each wind direction, Marathon Monitor Site, 2006

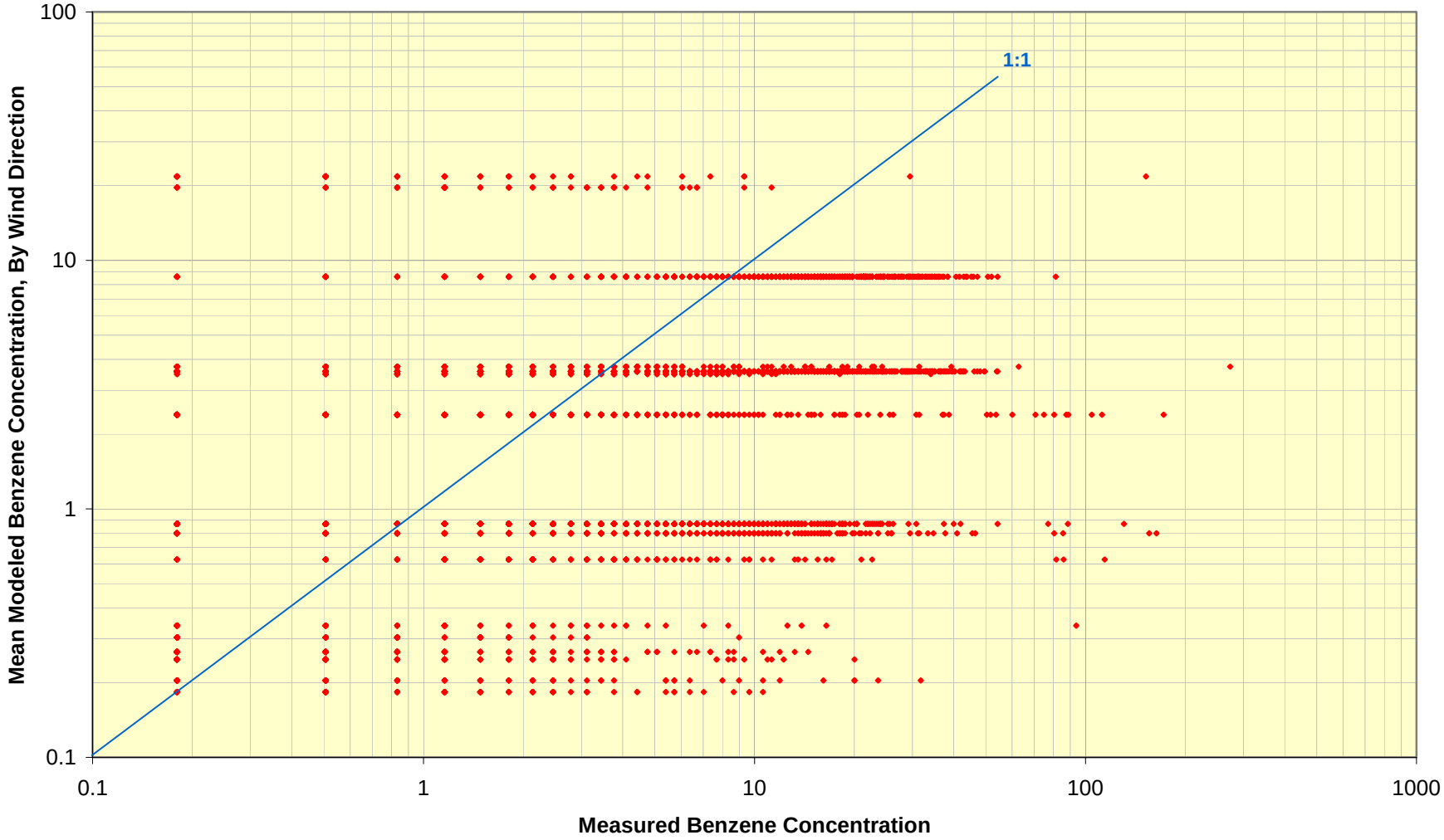


Figure L-7.

Wind Speed vs. Ratio of Hourly Monitored and Modeled Benzene Concentrations, BP Monitor Site, 2004

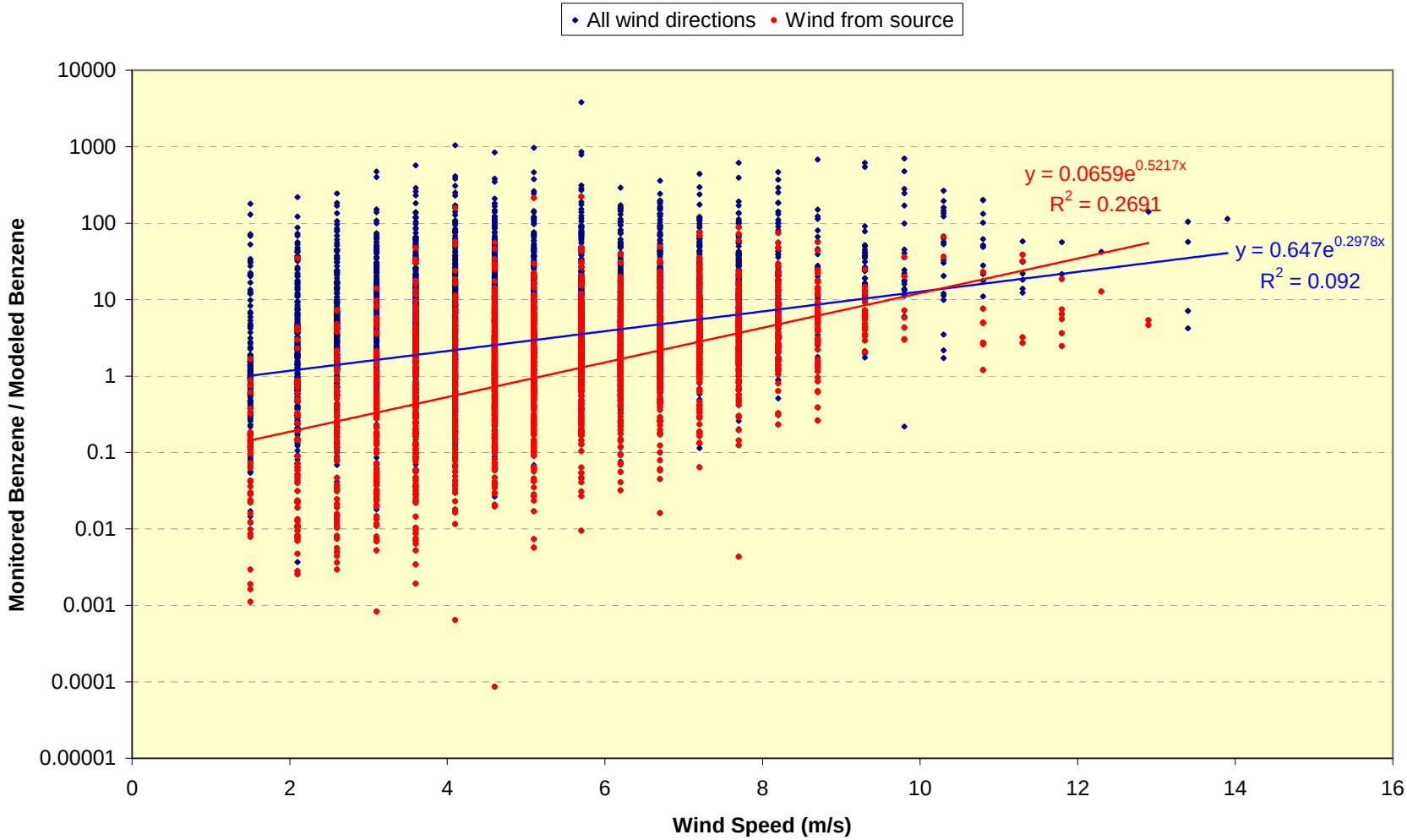


Figure L-8.

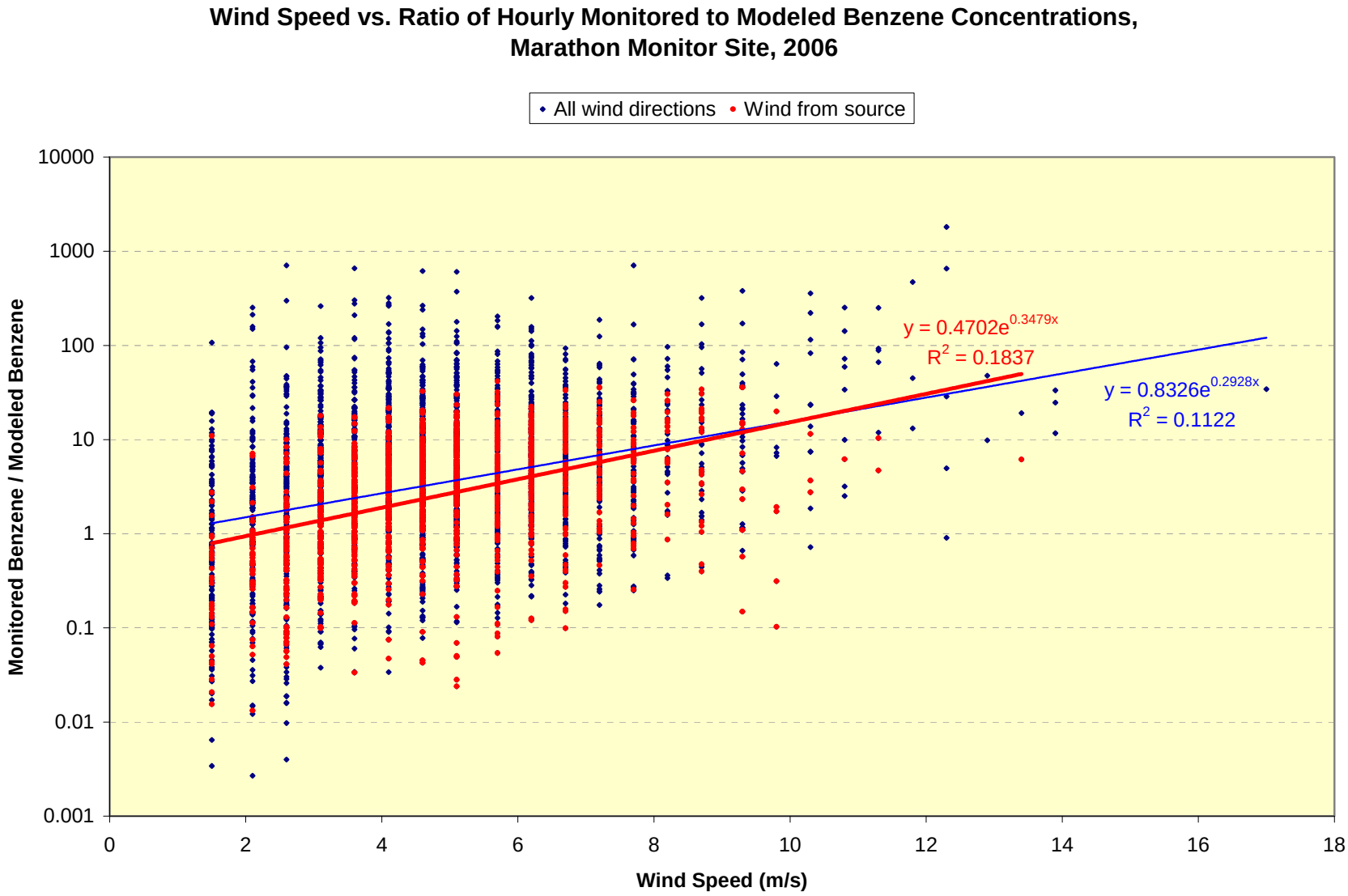

Wind Speed vs. Ratio of Hourly Monitored to Modeled Benzene Concentrations, Marathon Monitor Site, 2006
All wind directions
Wind from source
y = 0.4702e^0.3479x
R^2 = 0.1837
y = 0.8326e^0.2928x
R^2 = 0.1122
Monitored Benzene / Modeled Benzene
10000
1000
100
10
1
0.1
0.01
0.001
0
2
4
6
8
10
12
14
16
18
Wind Speed (m/s)

Figure L-9.

Frequency Distribution of Ratio of Hourly Monitored Benzene Concentration to Mean Modeled Concentration, BP Monitor Site, 2004, Monitor Downwind of Source

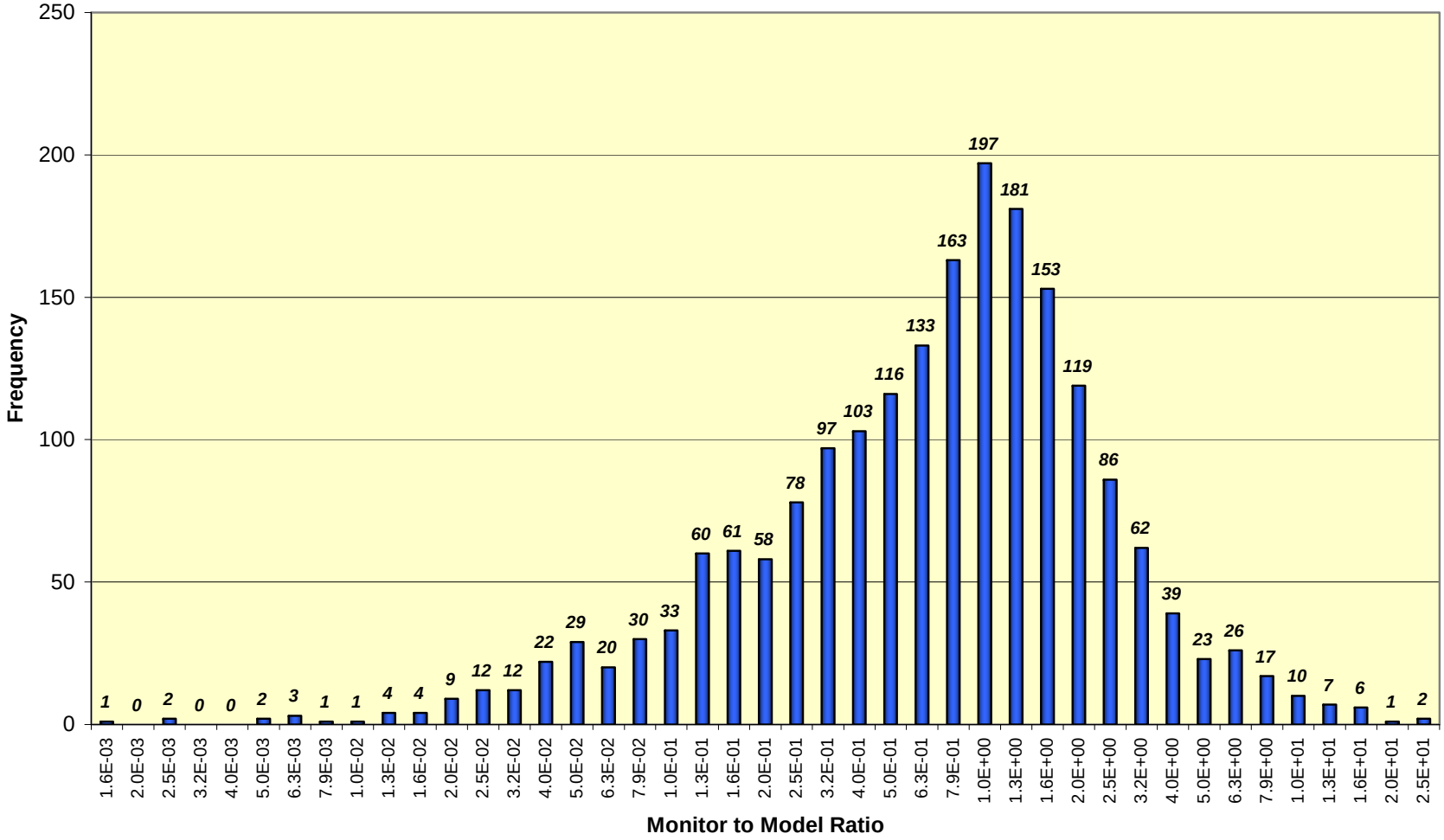
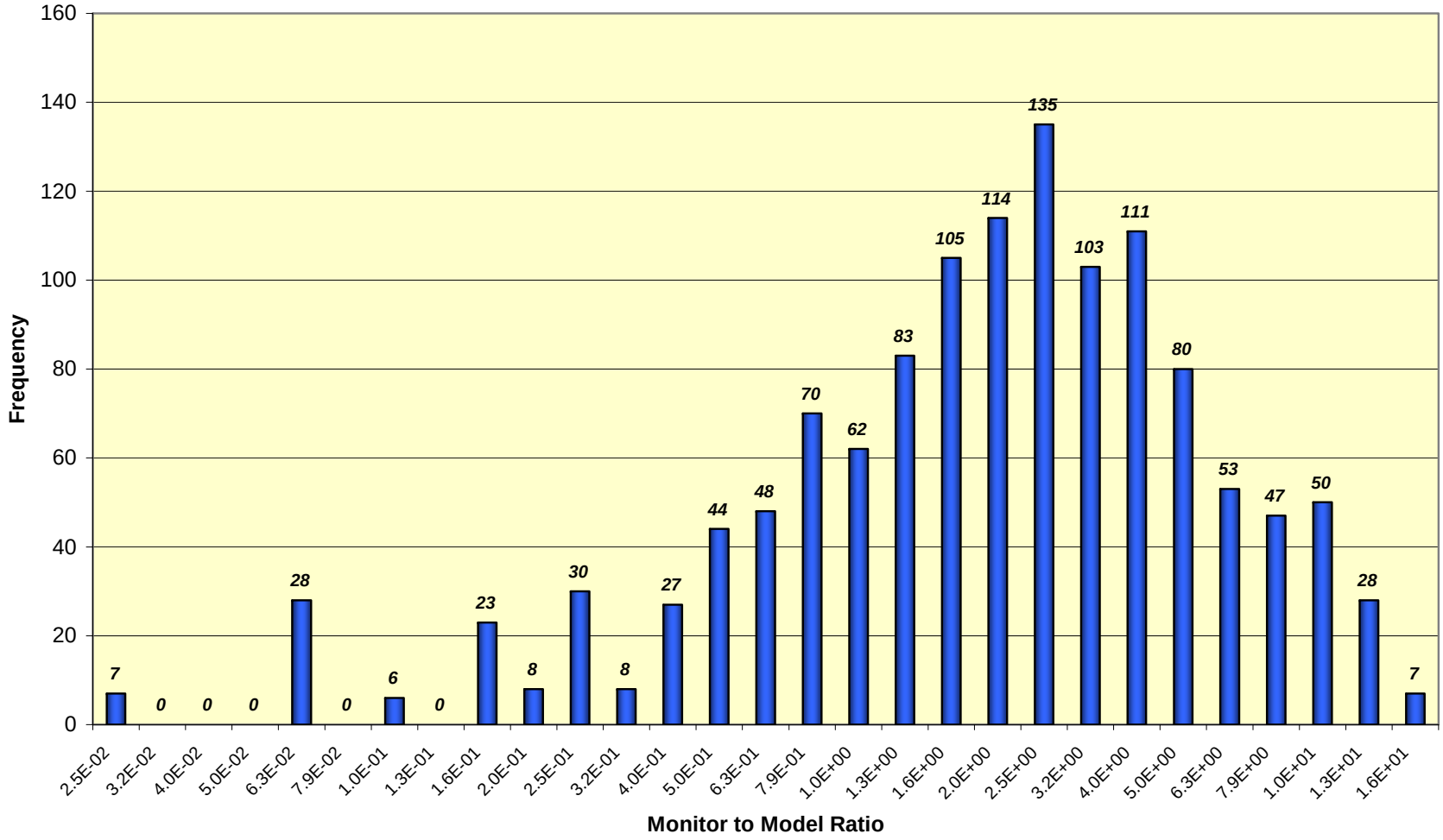


Figure L-10.

Frequency Distribution of Ratio of Hourly Monitored Benzene Concentration to Mean Modeled Concentration, Marathon Monitor Site, 2006, Monitor Downwind of Source



Attachment L-1: Alternative View: A Summary of One EPA Reviewer Comments on the Model- to-Monitor Comparison

The following is a summary of comments from Fred Talcott, EPA's Office of Policy, Economics, and Innovation (OPEI) on the current draft of the Model- to-Monitor comparison conducted in conjunction with the Petroleum Refinery case study. He felt that the current draft does not adequately address these comments, and they are provided for SAB reviewers as a plausible critique of and alternative to the current draft of Appendix L.

Conclusion: The approach makes sense and much of it is well-presented. Nevertheless, **I urge you to qualify your conclusion that the inventory for the Marathon may be an underestimate, and re-run the analysis considering the Specific Comments, provided below.** The difference in the modeled concentration may be explained primarily or in part by an underestimate in emissions, but the analysis provided does not make a case for that conclusion.

Specific Comments:

The focus of these comments is on the "Marathon" monitor (the model compares quite well for the BP monitor).

First: **Missing sources**.

The analysis uses 2002 emissions for the Marathon, Valero, and Sterling facilities, scaled to assumed 2006 emissions using TRI activity data. Benzene concentrations based on these emissions are compared to 2006 measurements at the "Marathon" monitor. Emissions from the BP refinery were included for the "BP" monitor comparison for 2004, but not for the "Marathon" comparison for 2006. The reason is the explosion and fire at the BP refinery in 2005; emissions from that site would be hard to estimate for the 2006 comparison year. While it is likely that fugitives would have been emitted from the BP site from clean-up and any residual storage and production activities in 2006, the analysis assumes 2006 emissions from BP are zero.

Further, the analysis does not estimate benzene emissions from shipping and barge traffic in the channel surrounding the Marathon facility on two sides, nor from shipping activities a little further afield in the channel between Galveston and Texas City on the mainland. This omission affects modeling at the "Marathon" site much more than at the "BP" site, which is about 5 km further inland.

Omitting these sources of benzene (emissions from the BP facility and nearby shipping and barge activities) probably contributes to the underestimate of benzene concentrations at the "Marathon" monitor site.

Second: **Meteorological data**.

The analysis considered, but rejected, the use of the data from the "Ball Park" met station, and opted to use met data from the Galveston Airport.

The Ball Park met station is located in an urban area, 1 to 2 km from the "BP" and "Marathon" monitors, and 2 to 4 km from the modeled facilities. The Galveston Airport met station is located across the bay on the field at the airport, about 14 km from the refineries. The Galveston Airport met station is located in an area where there are few building obstructions, about 1 km from a bay on one side and about 2 km from the Gulf of Mexico on the other.

Although both met stations have roughly similar distributions of wind directions (though Ball Park is more from the SE, and Galveston from the south), there is more variation in the wind roses for 2004 and 2006 for the Ball Park station than for the Galveston station. While this variation is given as a reason for preferring the Galveston station over the Ball Park station, this logic does not make sense. The meteorology is more variable at the Ball Park met station that is almost on top of the monitors and the sources OAQPS is modeling. The modeling should reflect the actual variability, and not the steadier, and less representative, conditions 14 km away at the Galveston station.

OAQPS has provided this reviewer a document titled "Galveston, TX and Texas City Ball Park, TX meteorological towers." What is not pointed out in the document, but is quite apparent in comparing the wind roses, is how much greater the wind speed is at the Galveston station in comparison to the Ball Park station. Reading off the wind roses (Figure 3 for the Ball Park station and Figure 5 for the Galveston station), about 54% of measurements are below 7 knots for the Ball Park station, but only 19% are below 7 knots for the Galveston station. Fewer than 6% of the measurements for the Ball Park stations are greater than 11 knots, but about 32% of the measurements at the Galveston station are above 11 knots, with some above 17 knots. That is, it's a great deal "blowier" on the field on a narrow island facing the Gulf of Mexico than it is inland in a developed area. This is no surprise.

The effect of using an artificially high wind speed is to push plumes from both stacks and fugitive emissions further down-wind, and artificially to dilute the estimated concentrations at points close to the sources, such as the two monitoring sites.

Use of the Galveston Airport met station probably contributes a sizable amount to the underestimate of benzene concentrations at both the "Marathon" and the "BP" monitor sites.

I suggest that you (a) include the information in the document titled "Galveston, TX and Texas City Ball Park, TX meteorological towers" as part of appendix L, and (b) re-run the analysis, using the met data from the Ball Park met station.

Third: **Adjusting for "background" benzene levels is highly uncertain**.

It is interesting and reasonable to try to adjust the monitored levels downward to account for the contributions from sources other than the industrial facilities you are modeling. The study took the 2002 NATA results for area and mobile sources at Census tracts in the Texas City area, and created an isopleth map (Figure 2), interpolating for values at the two monitoring sites. These estimated annual concentrations were then subtracted from the monitored values for each of the 8,700 hours for the year-long comparisons.

It is not easy to guess whether this might result in an upward or a downward bias in the resulting model to monitor comparison, but you do need to address this as a possible important source of uncertainty. Looking at the model to monitor comparisons for the 1996, 1999, and 2002 NATA, the study finds substantial uncertainties for benzene and for the other HAPs that were analyzed. These are not, regrettably, broken down by type of site (those heavily influenced by industrial emissions versus those with mostly area and mobile sources), but differences of a factor of over 2.0 are common in the comparisons. Thus, the adjustment factor should be viewed as having a substantial uncertainty, and this uncertainty then translates into uncertainty in the Texas City comparisons for the modeled facilities versus the background-adjusted monitors. A part of that is also the compounding factor of subtracting the estimated annual background from each of the hourly observations.

Thus, this background adjustment inserts a non-trivial uncertainty into the analysis process.

Fourth: **Other sources of uncertainty need to be summarized and discussed**.

Information provided to this reviewer said that you relied on the API effort to get stack and other release parameters, as well as any updated stack parameters provided through the FR process. I don't know what we can say about the completeness and the QC behind these release parameters. For BP Chemical, BP Refinery, and Marathon, TRI indicates that a large fraction of benzene releases are from fugitives. Fugitives would seem to drive much of the concentration picked up at the two nearby monitoring sites. I suggest that OAQPS take a careful look at the release parameters for the facilities and make a judgment about how accurate or how approximate they may be.

The study uses reasonable, but still uncertain, ways of adjusting across years. It is not clear, but it appears that for some years you scaled emissions by the ratio of TRI emissions, and used the TRI "activity ratios" for others. Acknowledge that there is uncertainty in these ratios, and that adds to the uncertainty in the whole.

Surface roughness can be an important determinant of concentrations, especially from fugitives and in the near field. You should document the values that you used (different for different wind directions?) and the rationale. Then, make some qualitative statement about how any uncertainty in surface roughness may translate into uncertainties in modeled concentrations at the monitoring sites.

With regard to stability class, please indicate which met site was the source for upper-atmospheric met data. What was the distance from Texas City? Qualitatively discuss how the different location may affect the validity of the hourly stability class used in the modeling, and the resulting uncertainty in the modeling results.

Make a clear, concise, and coherent statement of the presence and importance of each of these sources of uncertainty, and say something semi-quantitative or qualitative about their combined impact on the modeling results.

Fifth: **Put these results into the context of wider model to monitor comparisons**.

OAQPS has conducted and made available model to monitor comparisons for a few dozen HAPs for the 1996, 1999, and 2002 NATA analyses. For 1996, the median model-to-monitor ratio for benzene was 0.92; it was 1.47 for 2002. The modeled benzene concentration was within a factor of 2 of the monitored value in 89% of cases in 1996, and 69% in 2002. Modeling results were generally not as good for the other HAPs. If it is true that 31% of NATA predictions for benzene were off by two-fold in the most recent version of NATA, that helps to put the 2.6-fold difference found for Marathon in a different light.

Many factors contribute to the model's predictions being different from the monitors -- emission rates, location errors and approximations (especially for area and mobile sources), meteorological data, release parameters, surface roughness, etc.

What are we to make of the fact that the computations found for modeling at the "Marathon" site were 2.6-fold lower than the monitored values?

I make the case, above, that missing sources and use of the wrong met data may explain a significant part of the under-prediction. But I think that we have an obligation to acknowledge that there is sizable uncertainty in this kind of modeling, and that the 2.6-fold difference may well be within the noise of this noisy enterprise.

Specific recommendations:

1. Modify the Summary section to say that the differences between model and monitor values at the "Marathon" site may be explained principally by the omitted sources and the choice of the met station. Indicate that discrepancies such as these may be within the range of what can be expected in modeling of this type.

2. Include a discussion of the use of the Ball Park versus the Galveston Airport met stations.

3. If at all possible, re-run the analyses using the Ball Park met data. Use the full two years of met data from the Ball Park station, since there seems to be sizable variation from year-to-year. If computational resources are an issue, OAQPS might take a random selection of hours, or perhaps six-hour or daily segments from the 2004 and the 2006 Ball Park years, as a short-cut.

4. Add a summary section about all the sources of uncertainties in this modeling. See the first four points, above.

5. Include a section of ASPEN/NATA model to monitor comparisons. I suggest showing the benzene comparisons for each of the 1996, 1999, and 2002 comparisons, and perhaps just the 2002 data for the other HAPs. Can you answer the question: What fraction of model to monitor comparisons are within a factor of 1.5; 2; 2.5; 3; 10 (separately for high and for low)? FYI, for the 35 HAPs in Table 1 of the "Comparison of 2002 Model-Predicted Concentrations to Monitored Data," more than half of the HAPs missed the monitored value by more than a factor of 2-fold in at least 50% of the observations, i.e., more than a two-fold error is more than just common-place. Greater than a two-fold error is the norm.

Appendix M: Sensitivity analysis of uncertainty in risk estimates resulting from estimating exposures at Census block centroids near petroleum refineries

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
RESEARCH TRIANGLE PARK, NC 27711

OFFICE OF
AIR QUALITY PLANNING
AND STANDARDS

MEMORANDUM

DATE: December 23, 2008

SUBJECT: Uncertainties Associated with the Use of Census Block Centroids as Receptors for Chronic Exposure

FROM: Mark Morris
Sector Based Assessment Group (C539-02)

TO: David Guinnup
Sector Based Assessment Group (C539-02)

The HEM-AERMOD model estimates ambient concentrations at the geographic centroids of census blocks (using the 2000 Census), and at other receptor locations that can be specified by the user. In cases where the census block centroids are found to be located on facility property (as determined from aerial imagery), receptors are moved to the nearest off-site location. The model accounts for the effects of multiple facilities when estimating concentration impacts at each block centroid, and assesses chronic exposure and risk only for census blocks with at least one resident (i.e., locations where people may reasonably be assumed to reside rather than receptor points at the fenceline of a facility). Chronic ambient concentrations are calculated as the annual average of all estimated short-term (one-hour) concentrations at each block centroid. Possible future residential use of currently uninhabited areas is not considered. Census blocks, the finest resolution available in the census data, are typically comprised of approximately 40 people or about ten households.

The use of census block centroids as receptors for chronic exposure instead of actual residence locations introduces uncertainty into the risk assessment because residences within a census block may not be located near the centroid. This is minimized in highly populated areas because census blocks are typically small in such areas. In less populated areas, census blocks are typically large, and residences may be nearer to or farther from the source than the centroid, resulting in higher or lower actual exposures at those locations than at the centroid. However, this would not seem to introduce bias

because residences seem equally likely to be located on either side of the centroid relative to the source.

To test for possible systematic bias associated with the use of census block centroids as receptors for chronic exposure, we analyzed a sample of 21 petroleum refineries. We overlaid census block boundaries and cancer risks at census block centroids on aerial photographs of refineries with estimated cancer MIR values greater than or equal to 10 in a million. In some cases, we also overlaid cancer risk contours to allow estimates of cancer risk at residences that may not be reflected by the census block or polar receptors. These photographs are given in Figures 1 through 21. The cancer risk contours were created with geographic information system software using "natural neighbor" interpolation, which finds (for every point in the modeling domain) the closest polar receptor risk values and applies weights to them based on proportionate areas in order to interpolate a value. For several facilities, the density of polar receptors was not adequate to create cancer risk contours, and estimates of cancer risk at residences were made using the nearest census block and polar receptors. Because census block centroids sometimes fall within facility boundaries, HEM-AERMOD assigns zero risk to census block centroids that are within a user-specified distance (default is 30 m) of any emission source. This does not ensure that HEM-AERMOD will not assign a cancer risk to a census block centroid within facility boundaries, but it is the method that we use in the absence of specific facility boundary information. As can be seen, several of the figures show census block centroids within facility boundaries.

Table 1 gives the estimated facility-specific cancer MIR values at the census block centroid and at the nearest residence for 21 petroleum refineries with cancer MIR values greater than or equal to 10 in a million. In eleven cases, the census blocks were small, with a typical distance from the centroid to the block boundary less than 100 m. In these cases, we estimate that the MIR values at the census block centroid and nearest residence are identical. There were two cases where census blocks were relatively large, but for which the residences were located near the centroid. In these cases, we also estimate that the MIR values at the census block centroid and nearest residence are identical. In the remaining eight cases, the census blocks were relatively large, and the MIR values at the centroid were higher than the values estimated at the nearest residence, with the overestimates ranging from 40 to 2000 percent. In seven of these cases, the census blocks overlap both facility property and adjacent residential areas. In such situations, MIR estimates at the centroid are biased high because most of the area between the centroid and the boundary of the block nearest the facility is not residential.

In summary, in this analysis of facility-specific MIR values, the centroid-generated values overestimated the residence-generated values by 40 to 2000 percent in less than half the cases, were equivalent in over half the cases, and there were no cases where the value at a residence exceeded that at the centroid of the census block containing the residence. The MIR estimate for the source category as a whole was the same using either methodology. While it is possible that exposures at a residence in a large census block could be higher than at the centroid of the block, this analysis supports the use of the centroid as a reasonable representation of the MIR for the nearest receptor,

and it provides strong evidence that the use of the centroid is not creating a low bias in the overall risk results, indicating, in fact, the tendency for this approach to overestimate MIR values for the highest risk sources, and thus the MIR for the source category as a whole.

Table 1. Comparison of Risks at Census Block Centroid Versus Nearest Residence

Facility NEI ID	Maximum Individual Cancer Risk (in a Million)		Census Block Percent Overestimate
	Census Block	Nearest Residence	
NEI876	10	10	0
NEI6022	10	10	0
NEI6087	10	1	1000
NEI6436	10	10	0
NEI6475	10	10	0
NEI12711	30	30	0
NEI12791	10	10	0
NEI12988	20	20	0
NEI20174	10	7	40
NEI32864	10	10	0
NEI33031	20	10	100
NEI33039	10	3	300
NEI34050	10	10	0
NEI34057	10	10	0
NEI34898	20	20	0
NEI40371	10	5	100
NEI41771	10	10	0
NEI42040	10	3	300
NEI42309	20	1	2000
NEI CA1910268	10	10	0
NEIPRT$64	10	5	100

Figure 1. Cancer Risk for NEI876

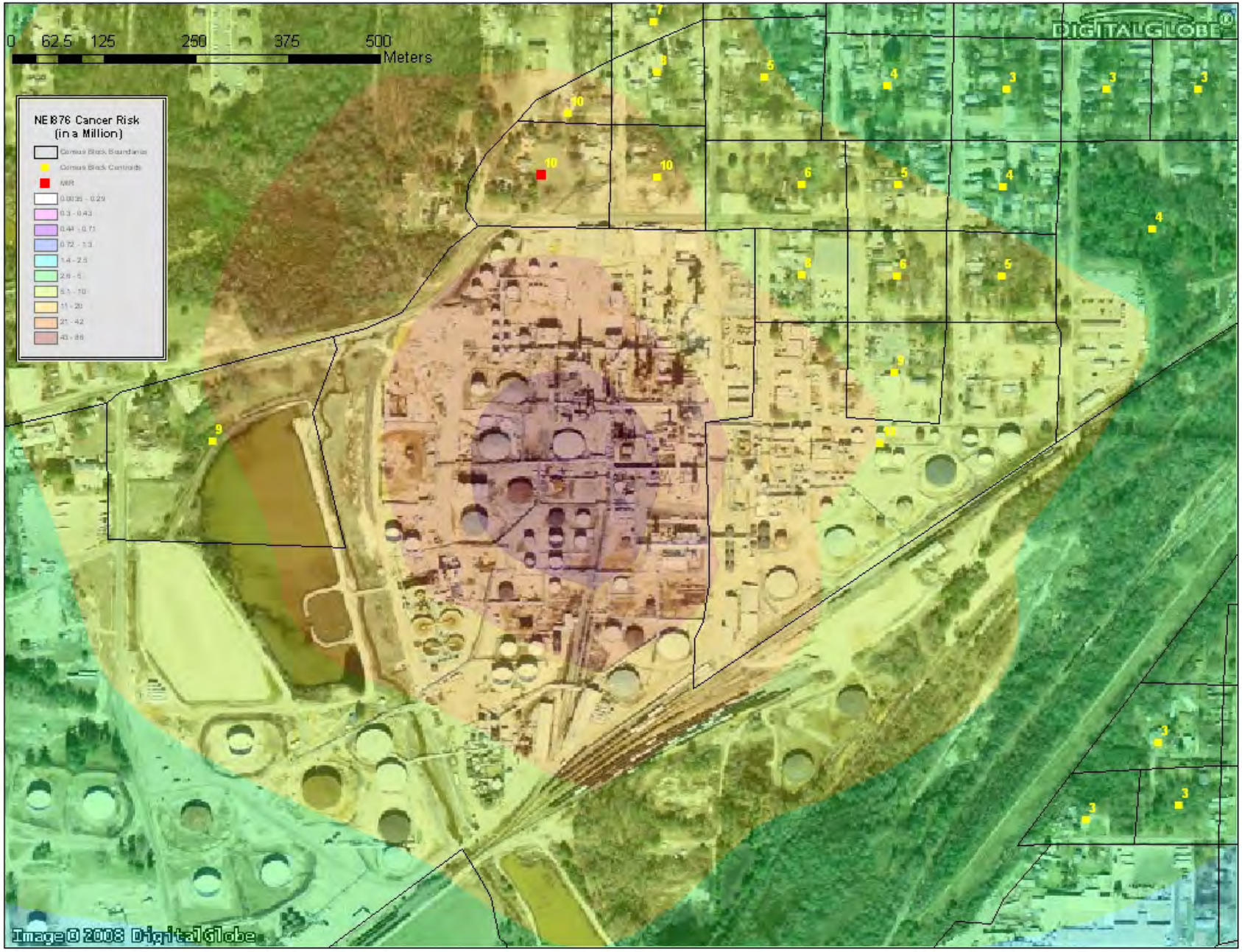


Figure 2. Cancer Risk for NEI6022

Figure 3. Cancer Risk for NEI6087

Figure 4. Cancer Risk for NEI6436

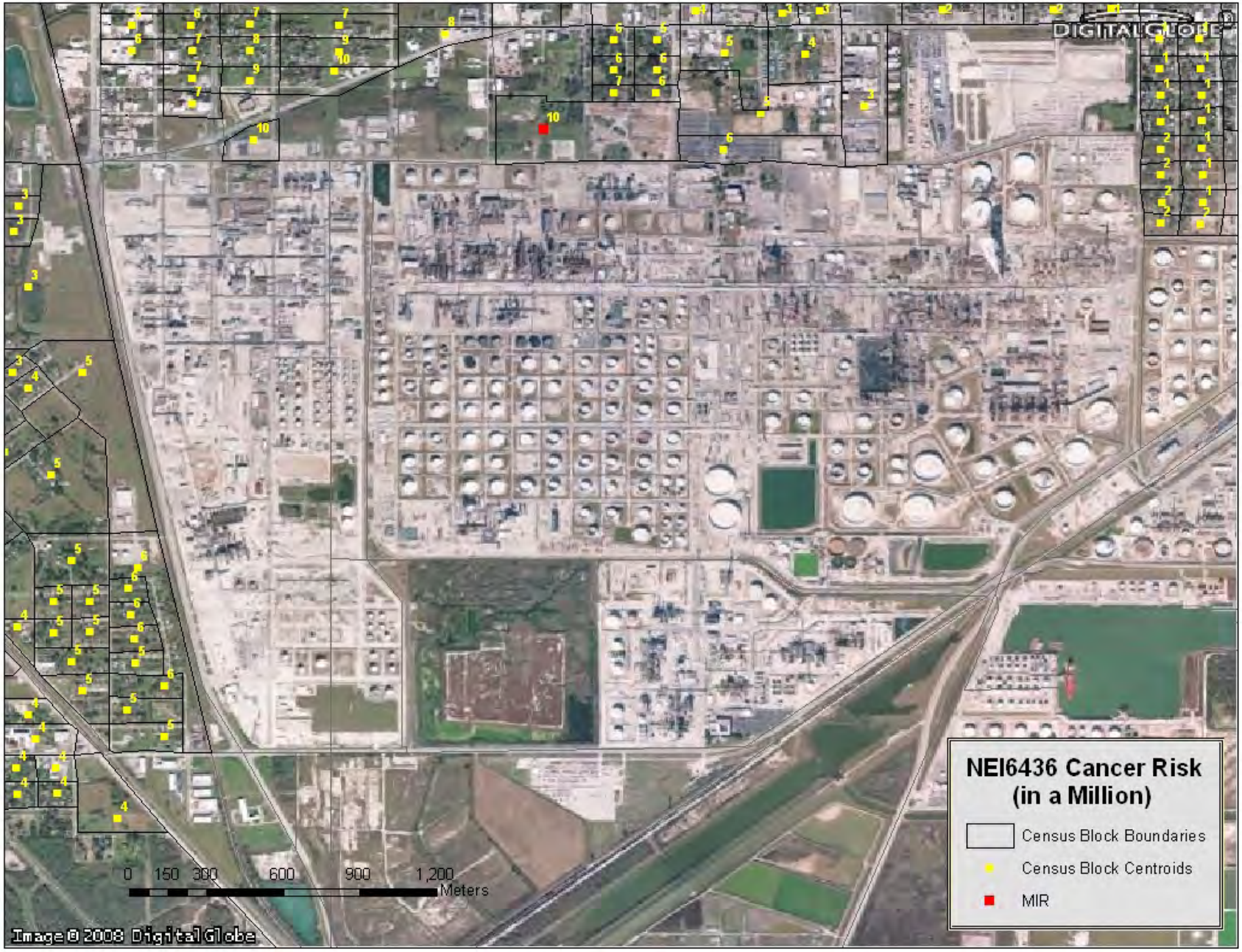


Figure 5. Cancer Risk for NEI6475

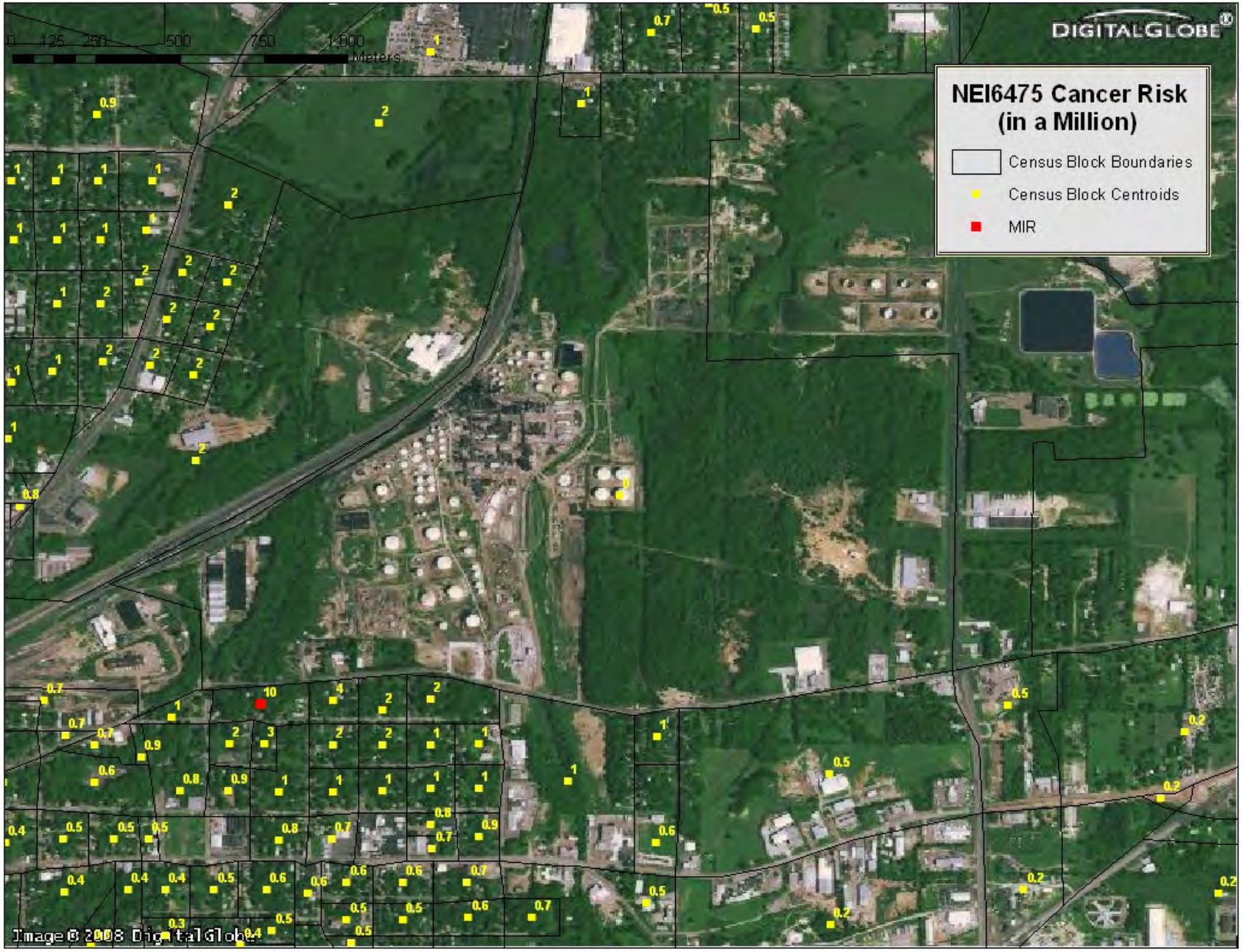


Figure 6. Cancer Risk for NEI12711

Figure 7. Cancer Risk for NEI12791

Figure 8. Cancer Risk for NEI12988

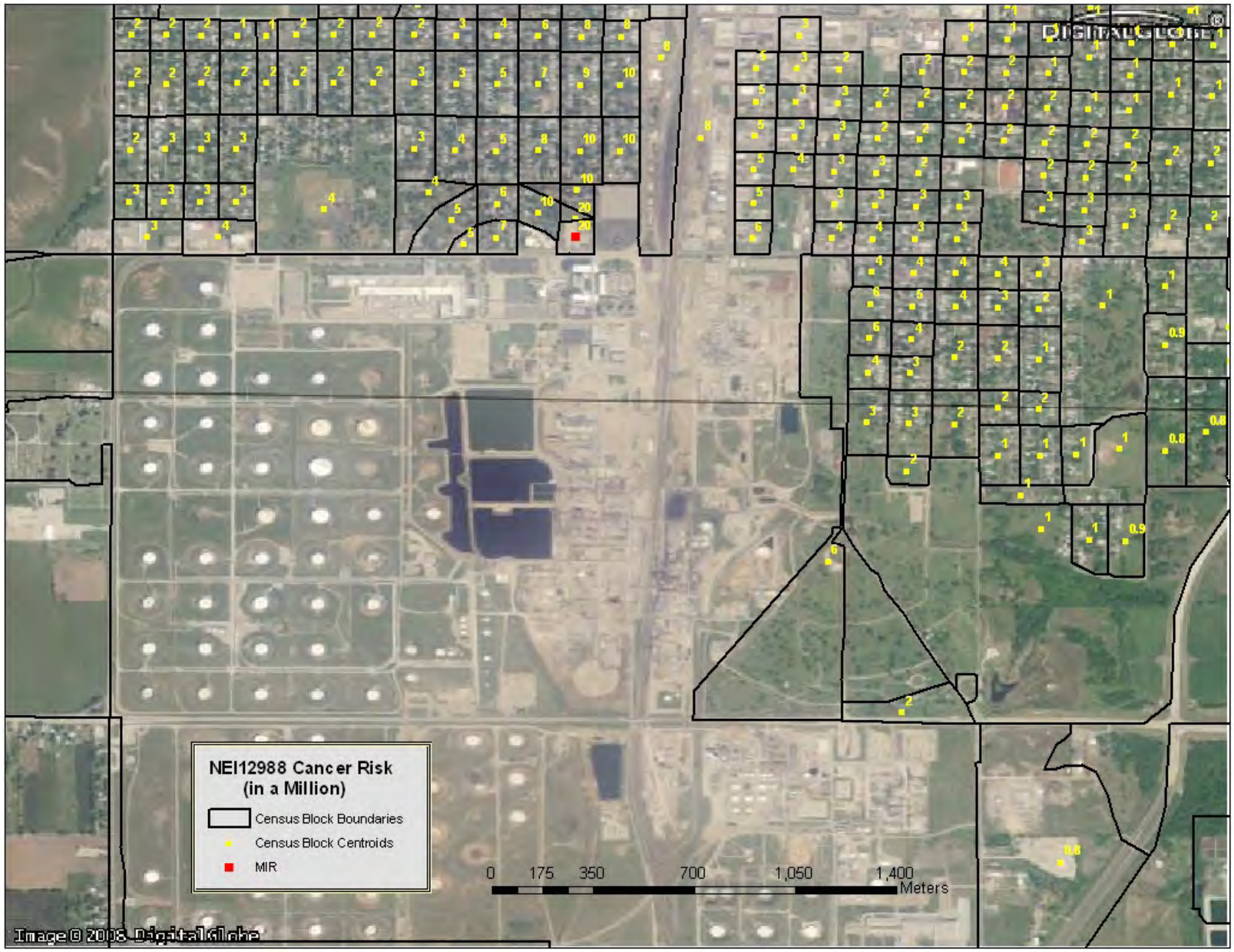


Figure 9. Cancer Risk for NEI20174

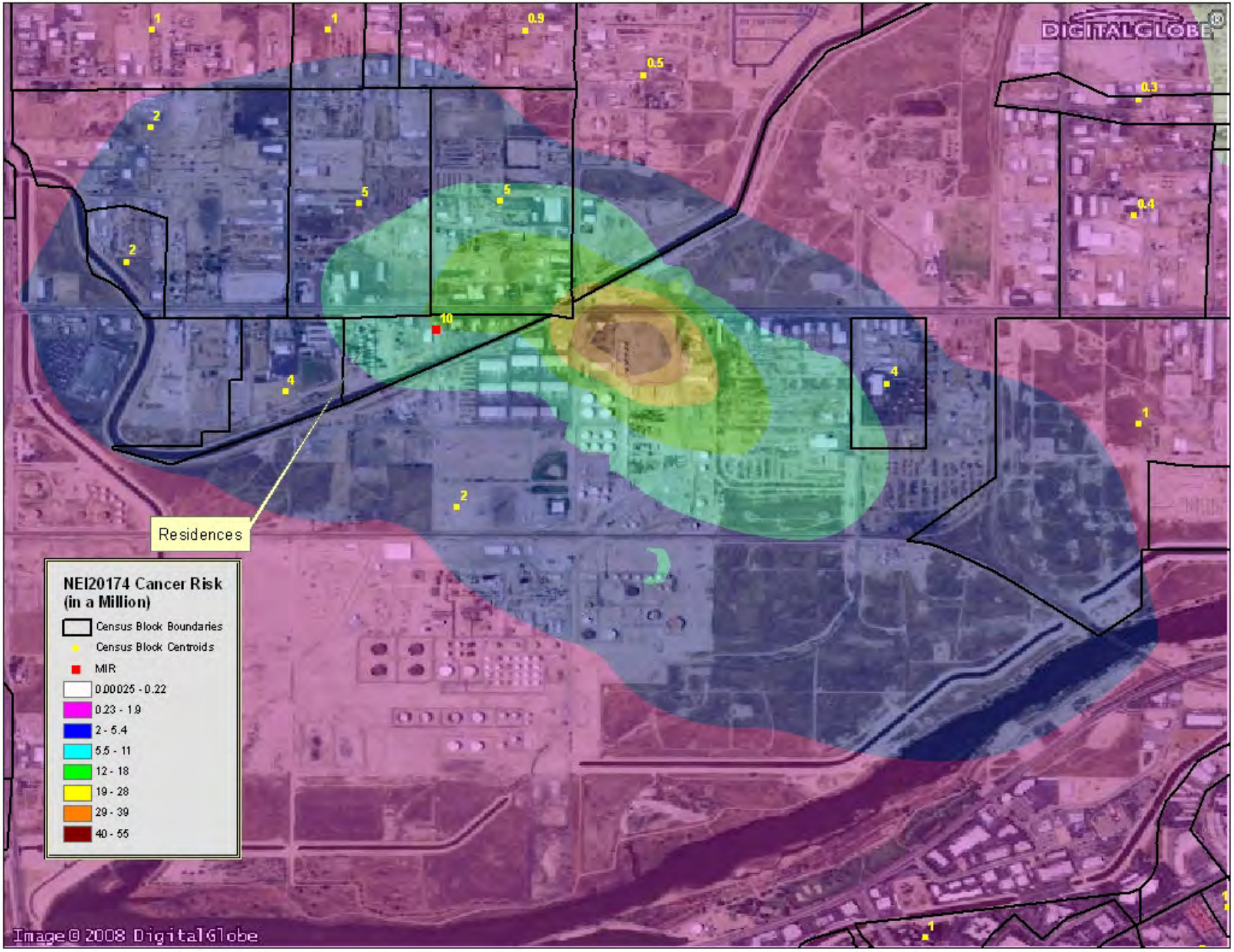


Figure 10. Cancer Risk for NEI32864

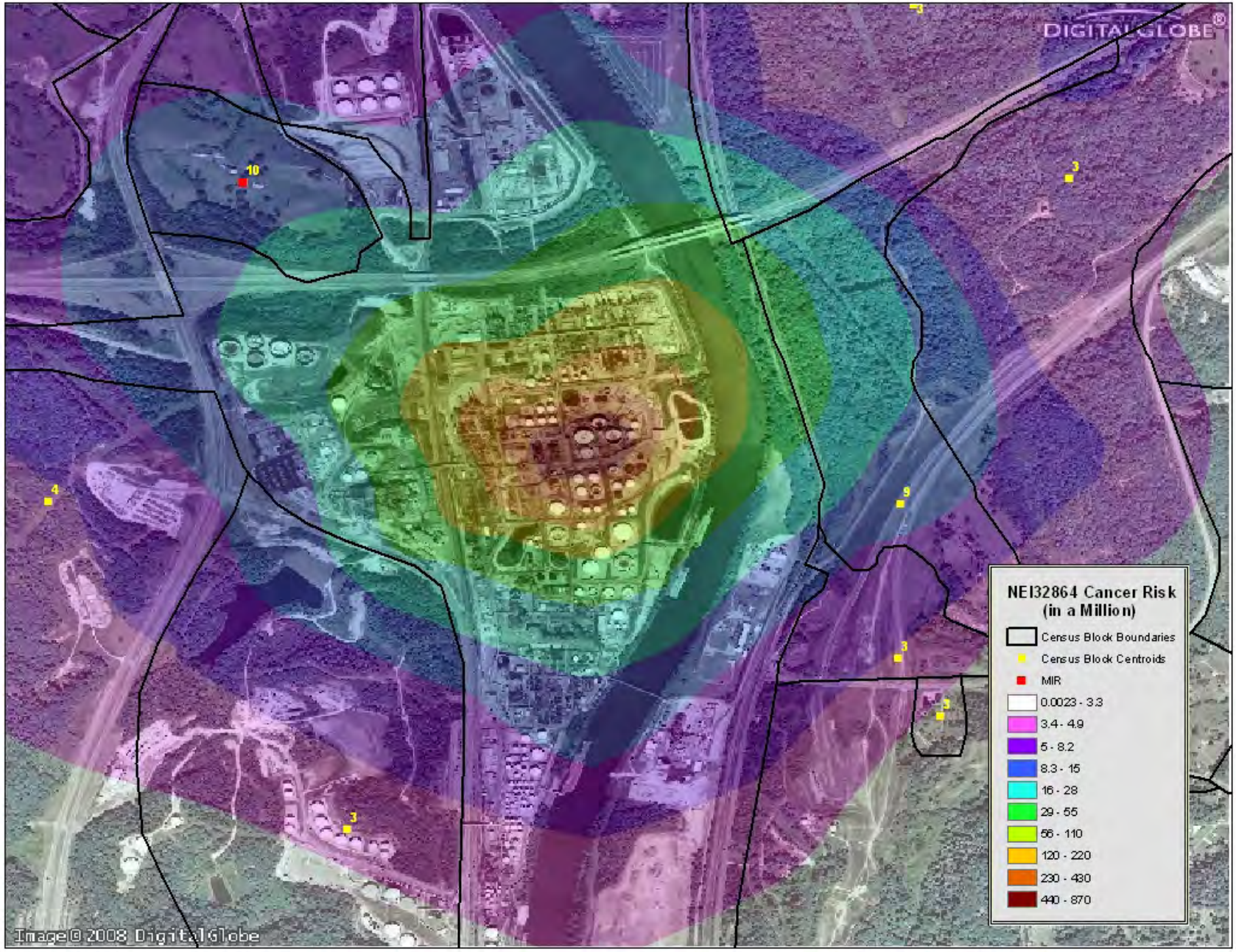


Figure 11. Cancer Risk for NEI33031

Figure 12. Cancer Risk for NEI33039

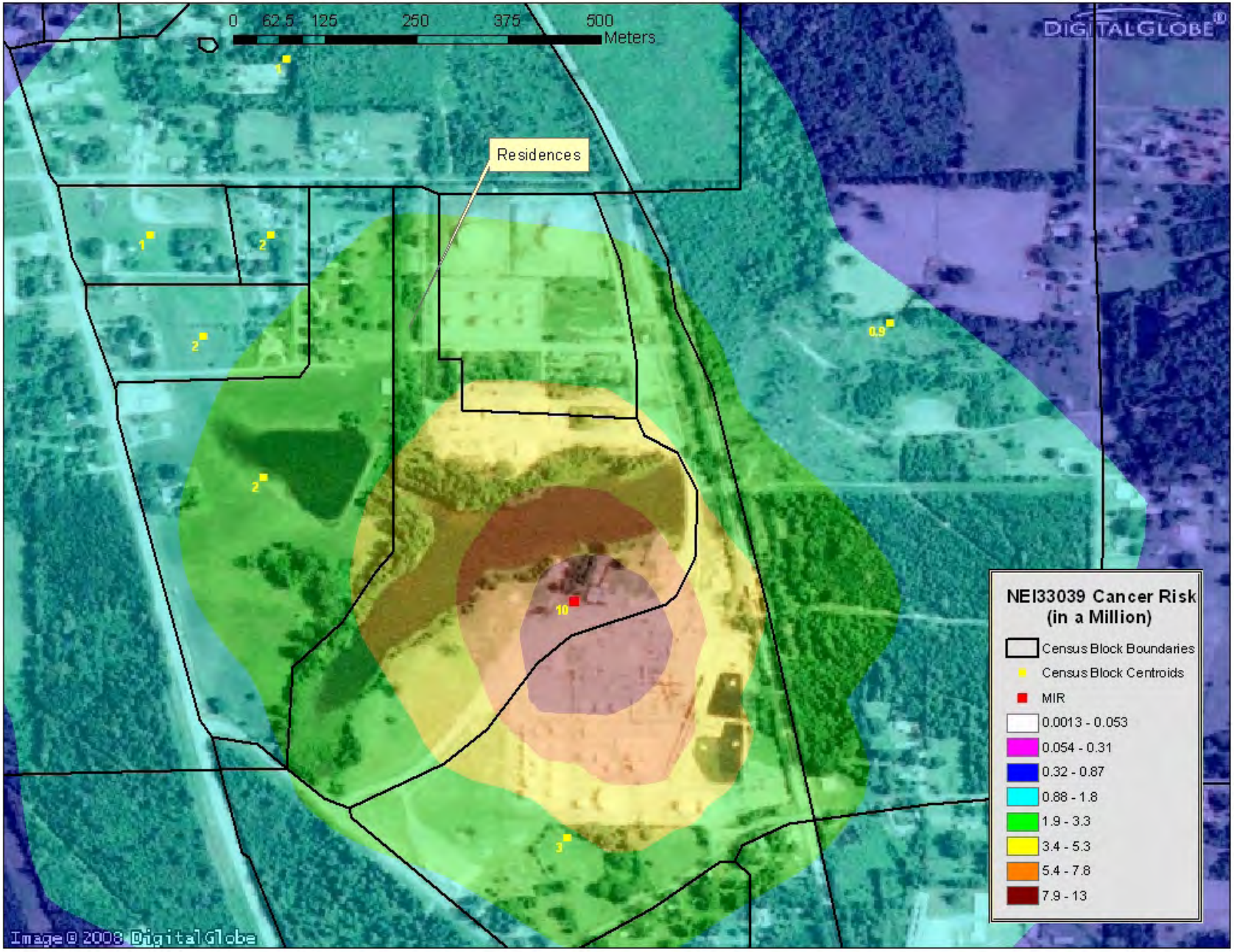


Figure 13. Cancer Risk for NEI34050

Figure 14. Cancer Risk for NEI34057

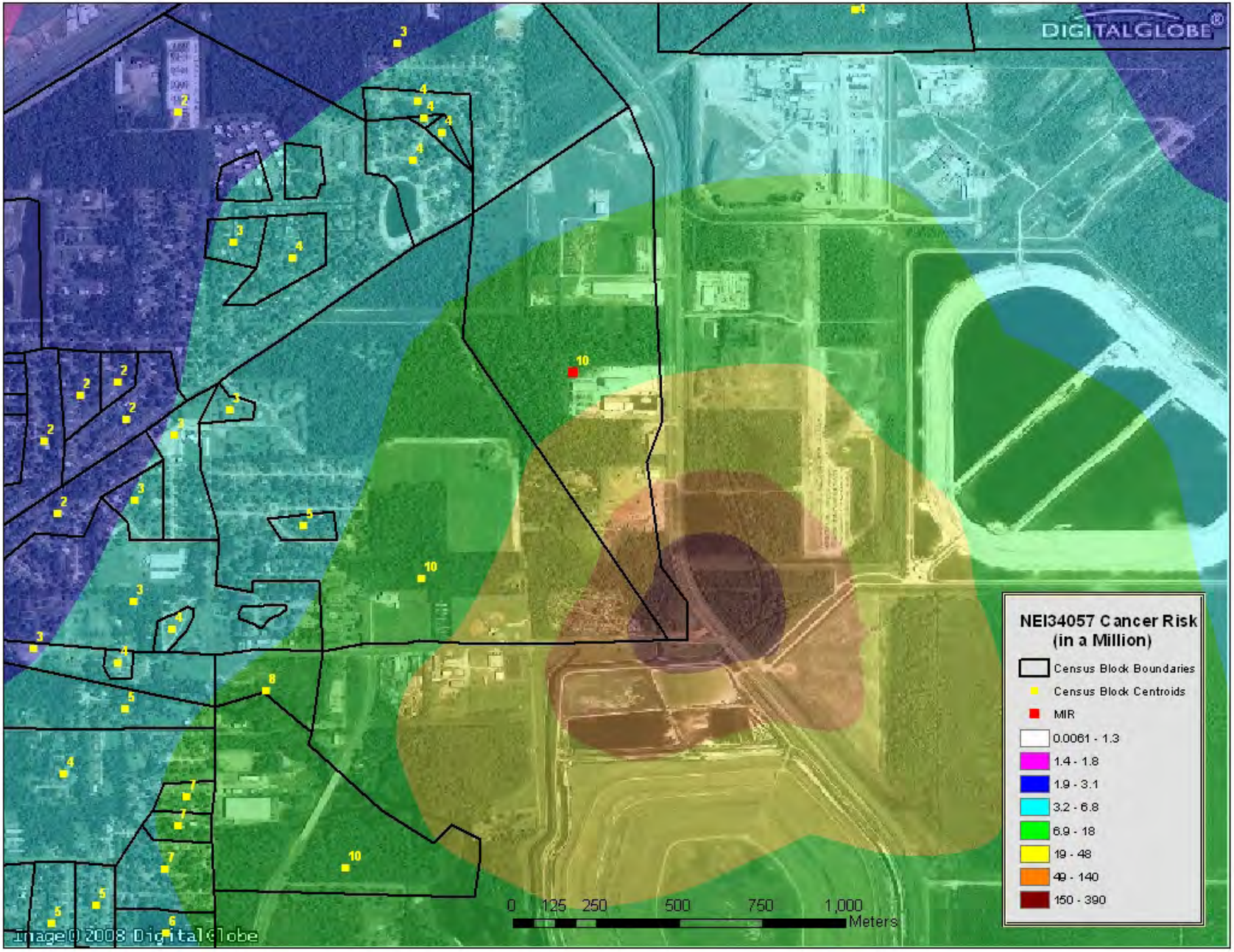


Figure 15. Cancer Risk for NEI34898

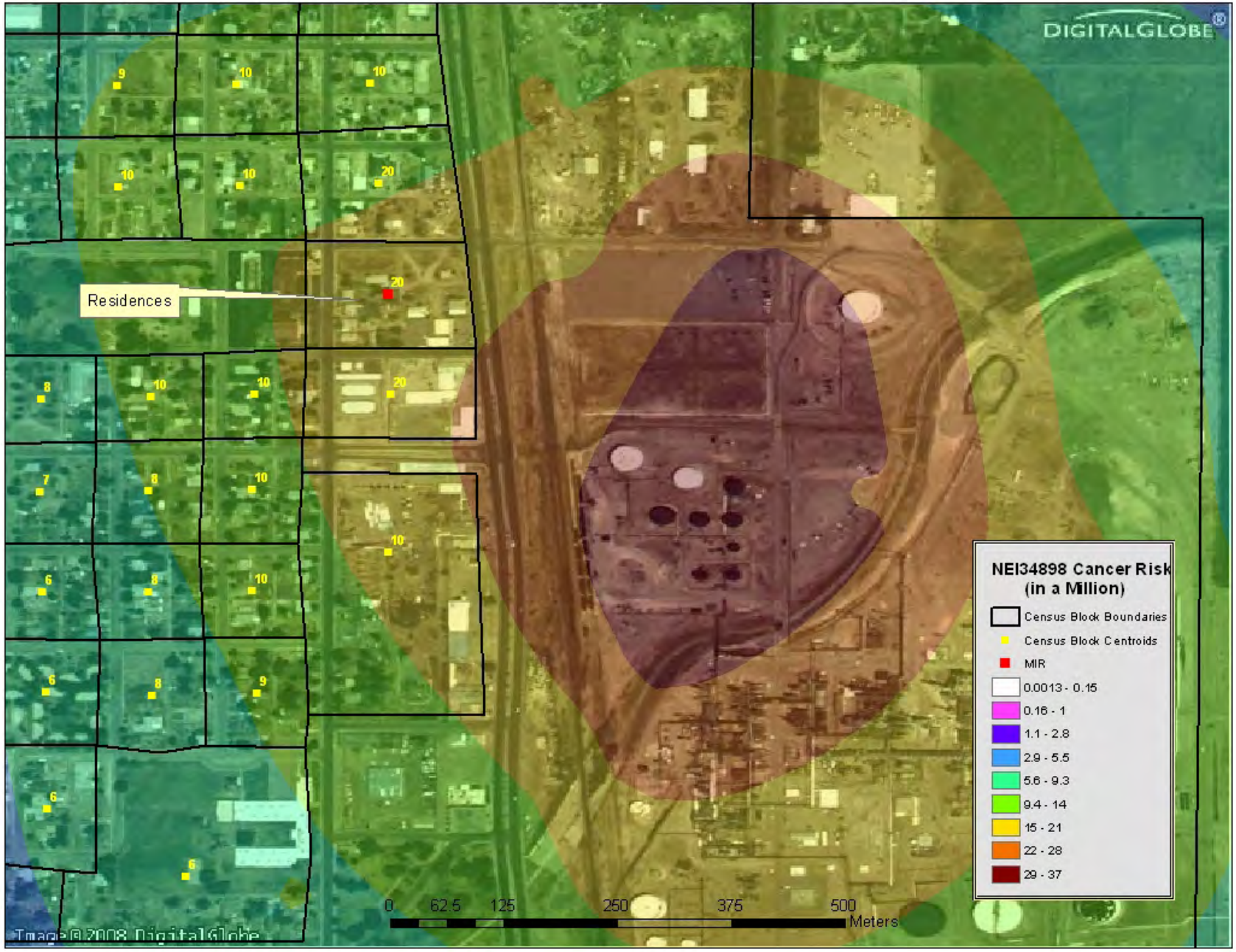


Figure 16. Cancer Risk for NEI40371

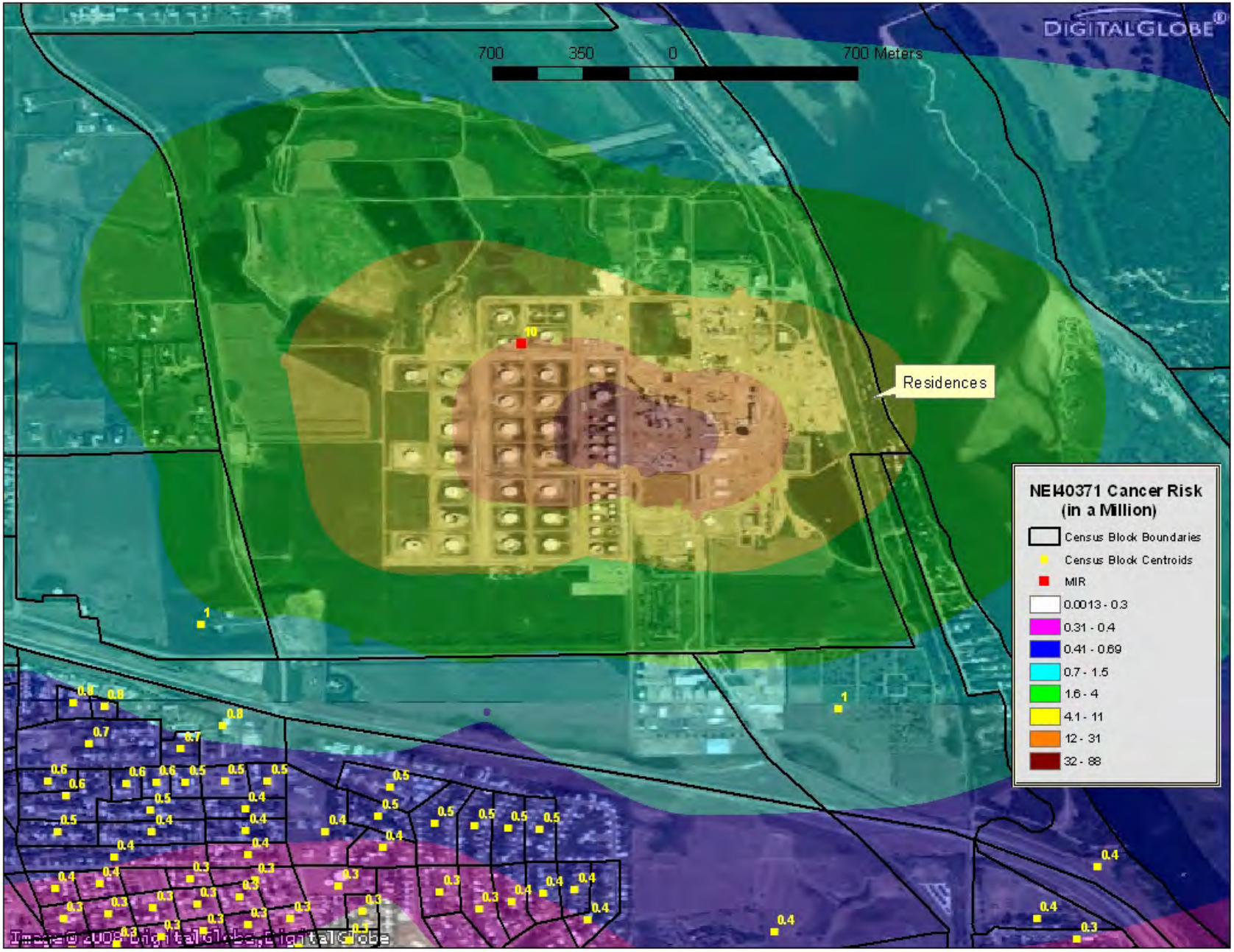


Figure 17. Cancer Risk for NEI41771

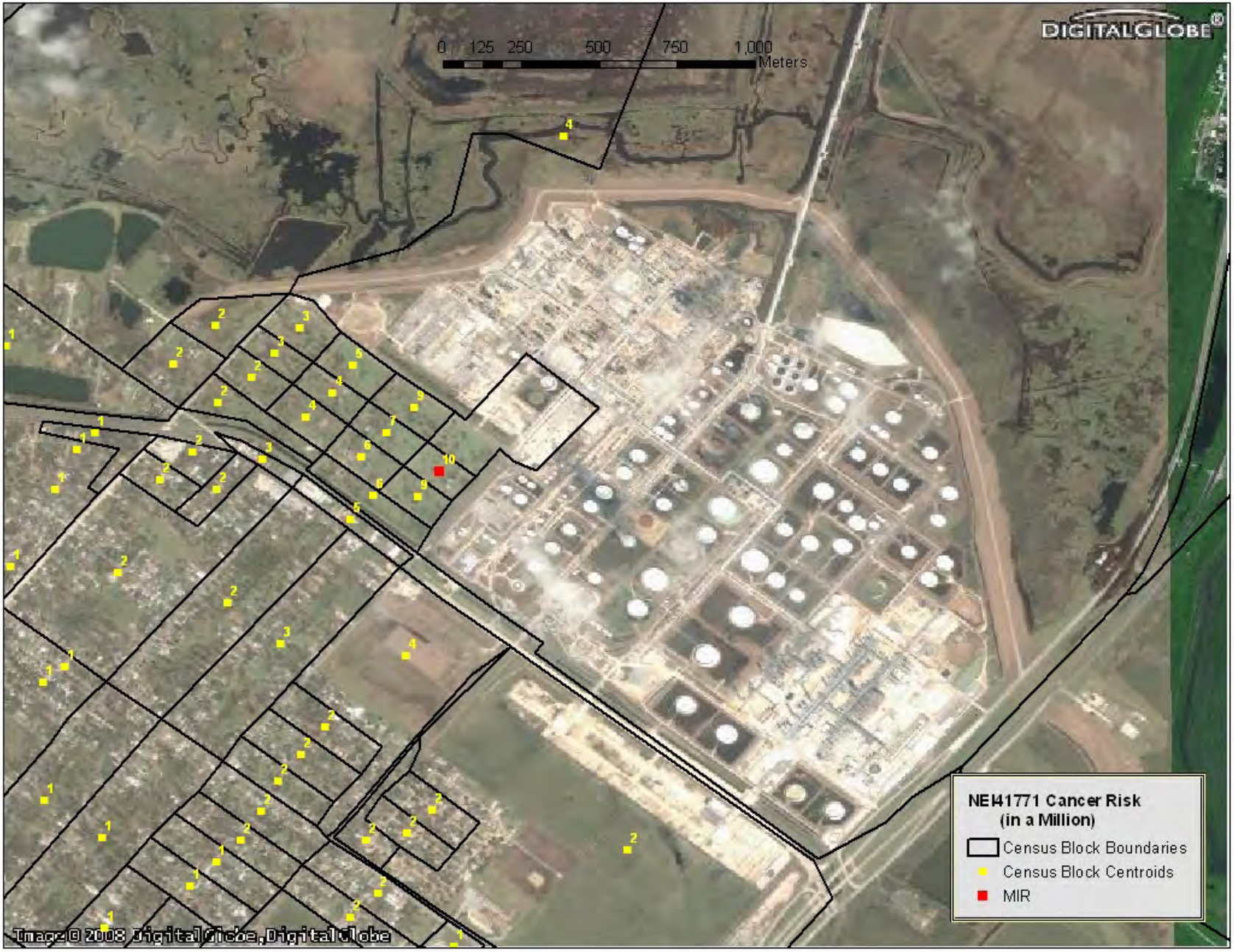


Figure 18. Cancer Risk for NEI42040

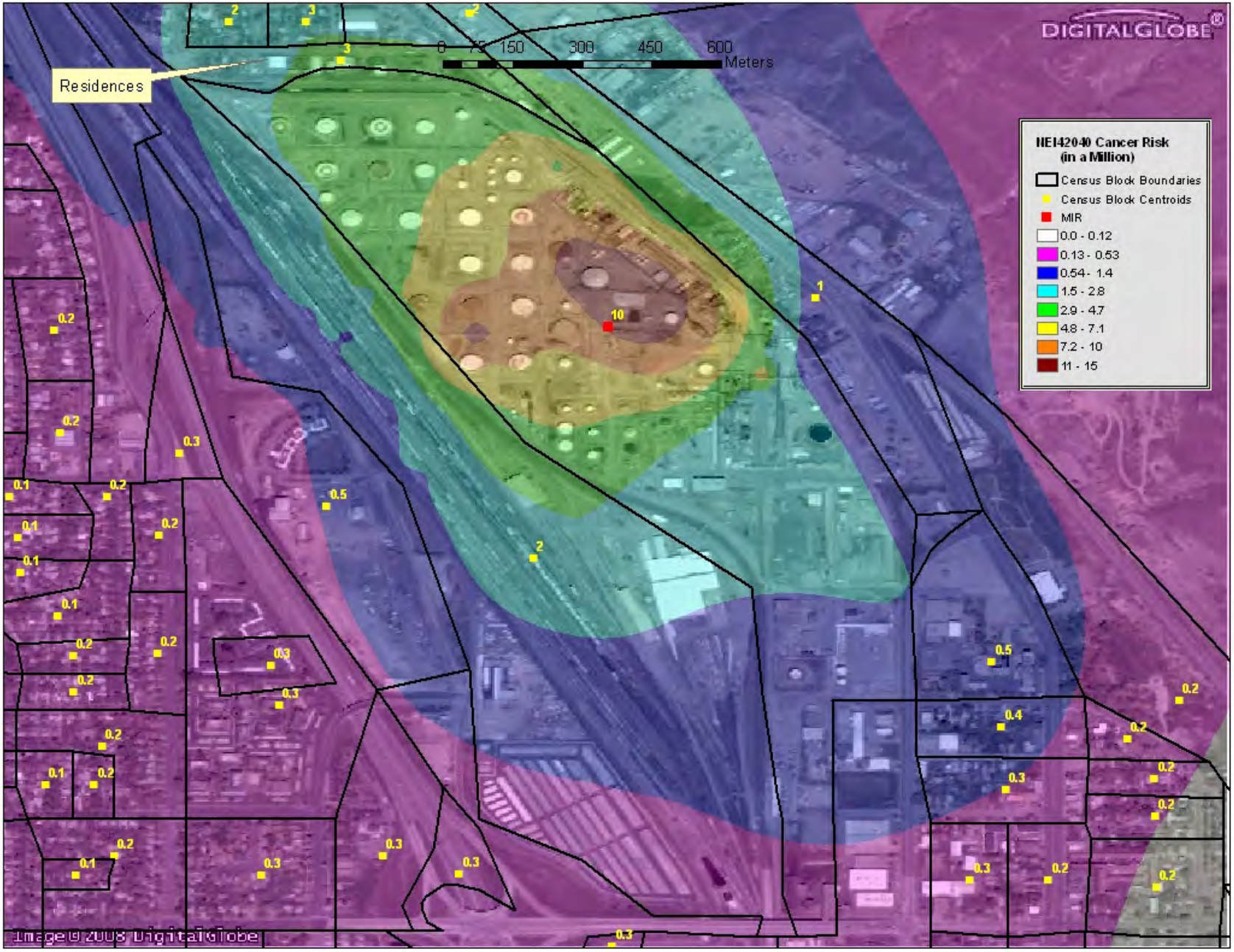


Figure 19. Cancer Risk for NEI 42309

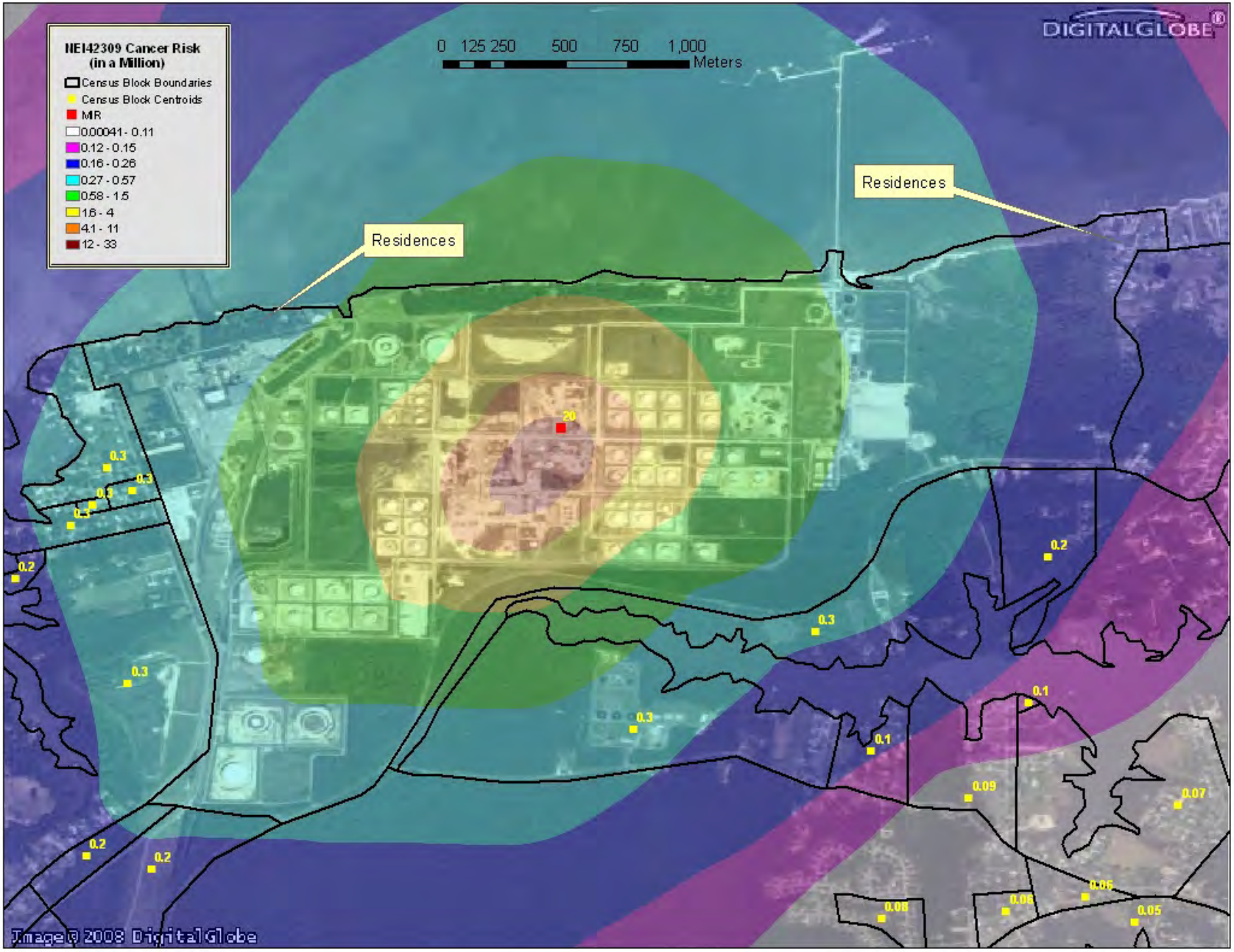


Figure 20. Cancer Risk for NEICA1910268

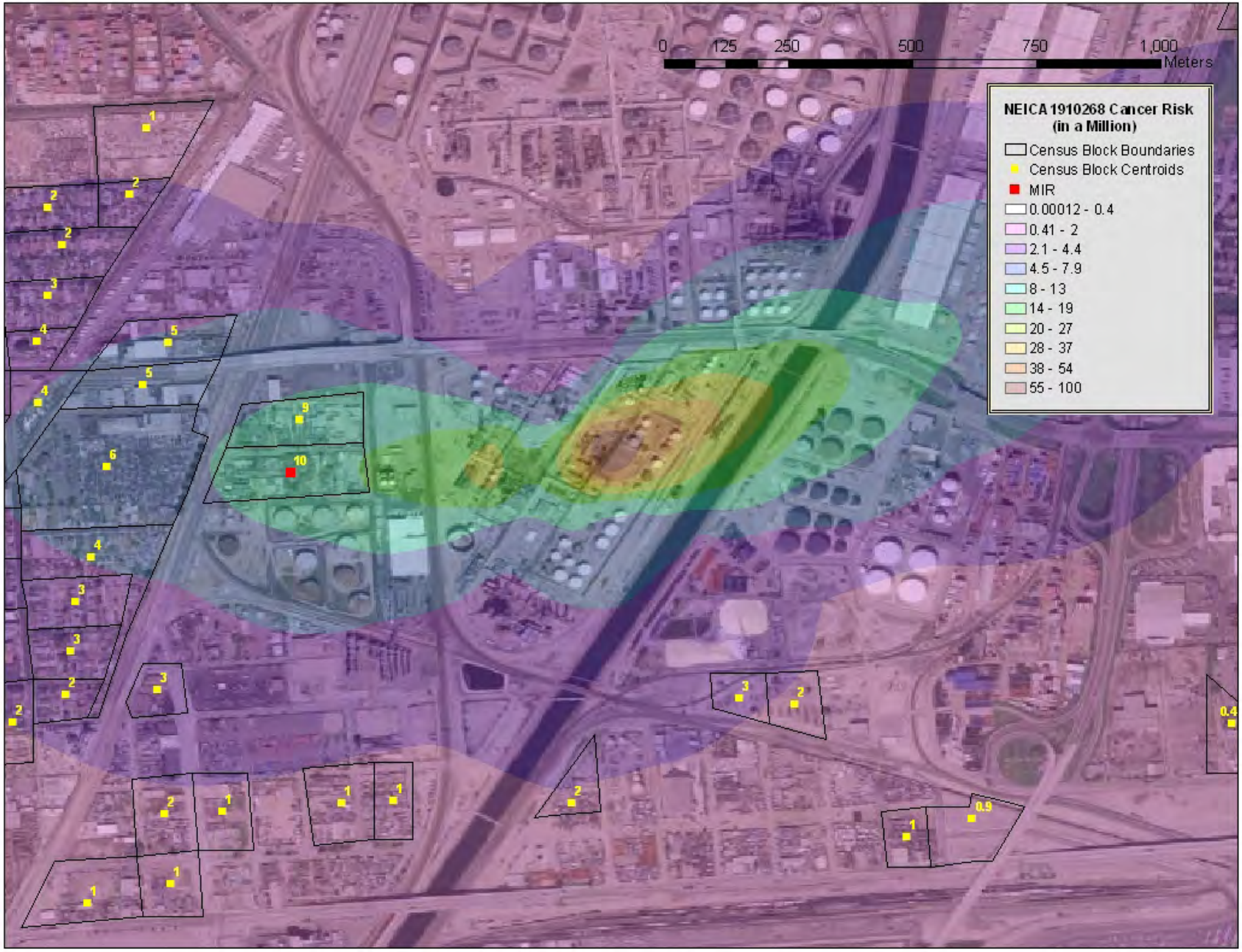
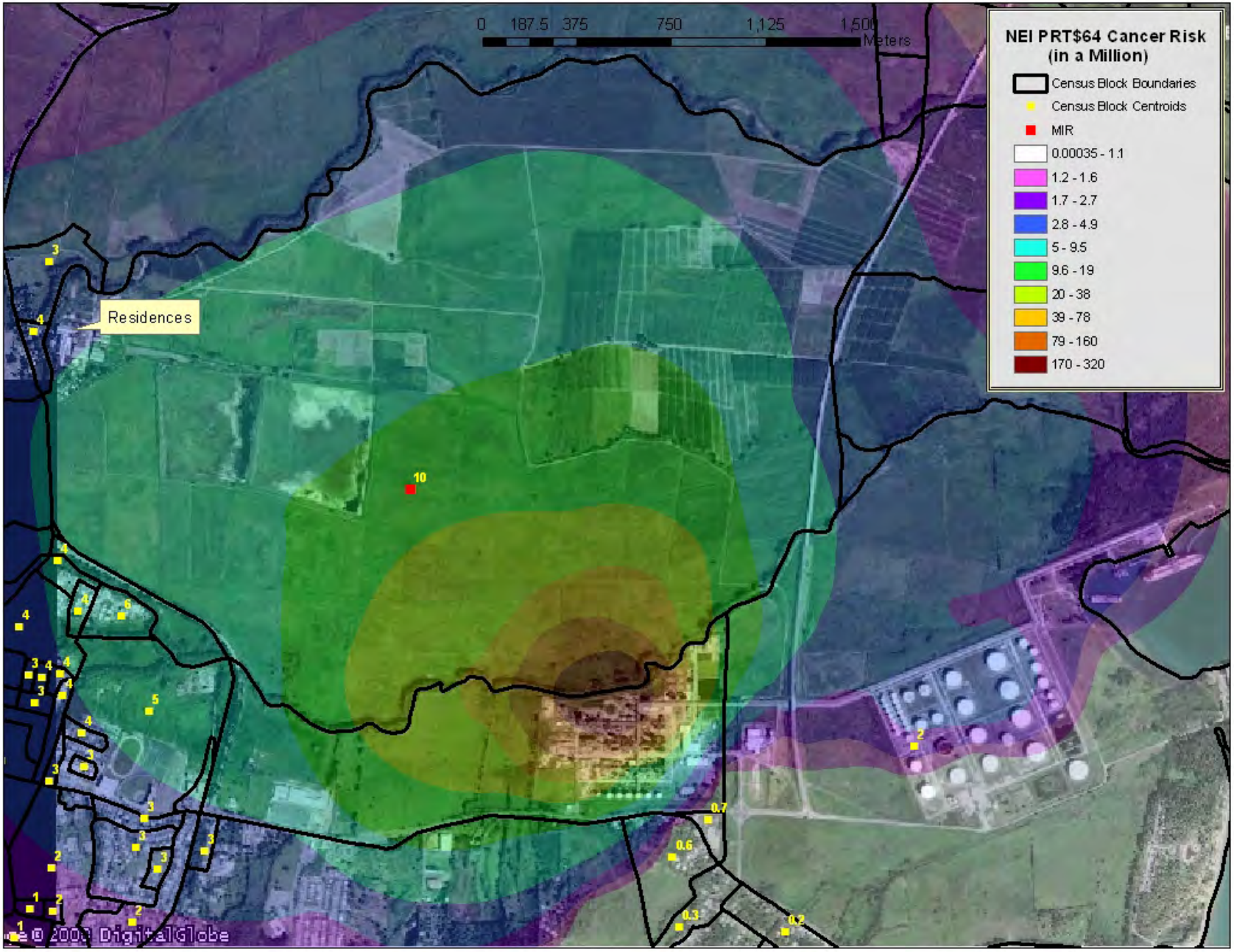


Figure 21. Cancer Risk for NEIPRT$64



Appendix N: Analysis of the effect of considering long-term mobility of receptor populations on estimates of lifetime cancer risk

N.1 Methods

This appendix describes probabilistic calculations performed on the estimated 70-year inhalation cancer risk output from the HEM-SCREEN model for the petroleum refining[1] and Portland cement source categories. The goal of these calculations was to adjust the HEM-SCREEN estimates to account for the receptor population's periodic relocation to new residences, either within or outside the exposure area. These calculations do *not* account for the effects of short-term behavior patterns (e.g., daily commuting or time spent outdoors) on exposure, nor do they consider sources of variation and uncertainty other than relocation and emigration.

N.1.1 Residence Time Input

In previous air toxics assessments EPA used a residence time frequency distribution from the EPA Exposure Factors Handbook (1996), based on an analysis by Johnson and Capel (1992). This analysis has two shortcomings that currently limit its usefulness for residual risk assessments. First, the underlying data were from 1987. Second, the approach was based on one-year move rates that appear to have underestimated residence time for long-term residents, as reported by the US Census Bureau in its Surveys of Income and Program Participation (SIPP, 1996 and 2001).

At EPA's request, Ted Johnson (one of the original authors) updated the Johnson and Capel (1992) analysis to reflect the more recent SIPP data and a newer, more complete modeling approach. Johnson's model randomly selected subjects from the US Census Bureau's American Community Survey database and estimated (1) time already spent in the residence, (2) future time to be spent in the residence, and (3) future length of life. These estimates were combined to predict the total time, past and future, that the subject would occupy the current residence. Johnson then compared the modeling results with SIPP residence time data and adjusted the results to compensate for "residential inertia" (*i.e.*, a tendency in the SIPP data for long-term residents to have lower-than-expected move rates). EPA is in the process of updating the Exposure Factors Handbook, and expects to replace its current residence time recommendations (Table N-1, below) with Johnson's new estimates (Table N-2). However, the entire Handbook must undergo scientific review, and we are not certain when that process will be complete.

Table N-1. Residence time estimates (in years) from Johnson and Capel (1992).

From	To	Probability
0	1.5	0.05
1.5	2.5	0.05
2.5	3.5	0.15
3.5	9	0.25
9	16	0.25

[1] For the petroleum refineries source category, the modeling exercise was conducted using the NPRM draft baseline assessment. Thus, the "before" results may differ somewhat from the final version of the assessment presented in the main report.

From	To	Probability
16	26	0.15
26	33	0.05
33	41	0.03
41	47	0.01
47	51	0.005
51	55	0.003
55	59	0.001
59	85	0.001

Table N-2. Revised residence time estimates (in years) described above.

From	To	Probability
0	1	0.05
1	2	0.05
2	5	0.15
5	12.6	0.25
12.6	27.2	0.25
27.2	45.6	0.15
45.6	56.3	0.05
56.3	74.9	0.04
74.9	81	0.005
81	91	0.004
91	100	0.001

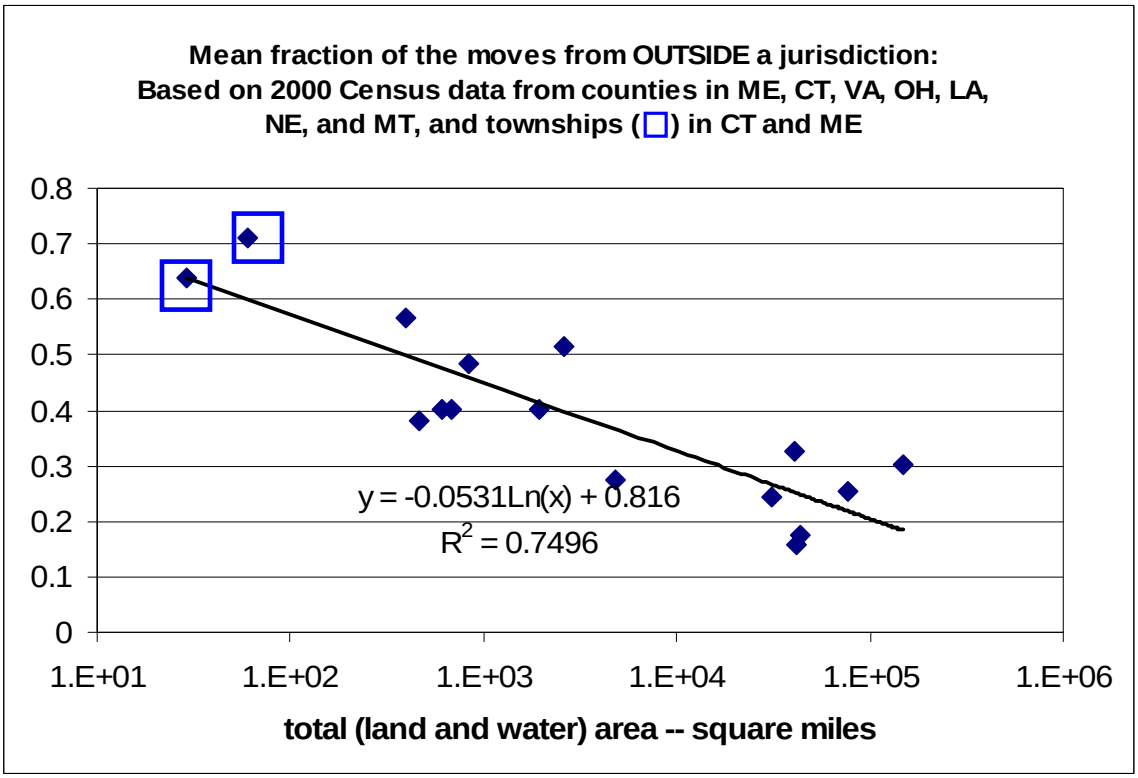
N.1.2 Emigration Input

The second distribution describes the likelihood that each relocation will remove the individual from the exposure area. This distribution is based on a regression analysis of 5-year population migration data (US Census Bureau, 2003) from seven states: Maine, Connecticut, Virginia, Ohio, Louisiana, Nebraska, and Montana. These states were non-randomly selected to provide a range of areas with different sizes and population characteristics that included large/small, rural/urban, east/west, dense/sparse population, and counties varying widely in size. Finally, two states (CT and ME) also included Census data broken down by townships (169 for CT and 523 for ME), supporting an extension of the regression into areas smaller than counties. Land areas were calculated using population density data from the late 1990's (Wright, 2003).

The regression indicates a highly significant ($P<0.00005$, $R^2 = 0.75$) inverse relationship between the fraction of moves from outside a jurisdiction (e.g., a state, county, or township) and the area of that jurisdiction (Figure N-1). This regression confirms common sense – as the target area becomes smaller, it becomes less likely that a random movers will "hit" it. The modeling domain for the source category was considered in the aggregate (rather than separately for each facility) to allow for the possibility that a person who moves away from one facility could relocate near another facility in the same source category. For the sake of simplicity in this analysis, the total population size was assumed to be constant, and the rate of emigration from the area was therefore assumed equal to the rate of immigration. That is, each person coming in replaced a person who

left. This is unlikely to be true in the real world, but including a population growth variable would require site-specific information for each facility.

Figure N-1. Association between immigration rate and total area.



The HEM-SCREEN assessment included 153 refineries and 91 Portland cement facilities, all for which we had input data at the time this analysis was developed. Each facility had a circular modeled domain with a 20-km radius, for a total modeled area of about 120,000 and 70,000 square miles, respectively. These areas correspond to those at the far right of Figure N-1, suggesting an emigration rate of about 20%. Therefore, the calculations for all receptors assumed that 20% of those who changed residences left the exposure area and 80% relocated to another residence within it. The estimates of total modeled area did not consider overlap of neighboring facilities, which could cause an underestimate of the emigration rate. On the other hand, some facilities in each source category could not be modeled, which could overestimate emigration. No allowance was made for individuals returning to the exposure area after once leaving it.

There are several important sources of uncertainty associated with this approach:

1. The regression data were selected non-randomly in order in maximize the diversity of the included populations *(e.g.*, with respect to density, land area, etc.) These data may not be fully representative of the populations affected by the emissions from these source categories.

2. The population size was assumed to be constant. To the extent that this is not true for the receptor populations affected by these source categories, this important simplifying assumption will underestimate the number of people exposed where populations are growing and overestimate it where populations are contracting. The uncertainty associated with constant population size is probably minor compared to other uncertainties.

3. The Census data represented a comparison of respondents' residence with their residence five years earlier. People may have moved more than once during this period, potentially biasing the immigration rate estimates either low or high. The Census data themselves are a subset of the population who filled out longer data forms, who may not fully represent the entire population.

4. This analysis has not specifically considered several demographic variables that are known to strongly influence move rates, e.g., age, income, marital status, and owner/renter status. These factors may vary substantially among Census tracts, meaning that move rates may also vary substantially. Applying central tendency move rates to the entire modeled domain means represents an important source of uncertainty.

5. Individuals who once emigrated from the modeled area were assumed never to return to it. Because the size of the unmodeled area is so much larger than that of the modeled area, this assumption probably did not have a strong effect on the results.

6. The total modeled areas of these assessments represent the upper limit of the regression, and the estimated emigration rate therefore is subject to greater error than an area in the regression's center (e.g., 1000 to 10,000 square miles). The regression itself is subject to statistical error, meaning the true relocation rates should be viewed as falling within a range of approximately 10-30%, rather than fixed at exactly 20%. Using a different relocation rate within this range might have produced significantly different results.

N.1.3 Lifetime Inhalation Cancer Risk Input

The initial lifetime (70-year) inhalation cancer risk estimates from the HEM-SCREEN model included risk estimates for approximately 53 million and 89 million individuals who live within 20 km of one or more of the modeled Portland cement and petroleum refining facilities, respectively. However, in order to focus on the most-exposed subpopulation of potential regulatory interest, mobility adjustments were calculated only for individuals whose 70-year risk estimate was 1 in a million or greater.

2. Probabilistic Calculations. Calculations were performed using Crystal Ball™ and Microsoft Excel™ software. Each probabilistic simulation included 100,000 trials based on the Monte Carlo sampling method, using the same random number seed to ensure repeatability.

The probabilistic calculations for all individuals began by randomly selecting one individual from the HEM-SCREEN distribution of lifetime risks. This individual was assigned a residence time (selected randomly from the residence time distribution), and the risk associated with the exposure in that residence was calculated as follows:

Equation 1

$$Risk_R = \frac{Risk_L}{70y} \times RT$$

Where: $Risk_R$ = Estimated cancer risk from years in residence R
$Risk_L$ = Estimated total lifetime cancer risk (from HEM-SCREEN)
RT= Residence time (y)

This individual was also assigned a random binary emigration value that determined if he/she moved to another home within the exposure area at the end of the residence time, or left the assessment either by emigration or death. If the binary emigration value for the individual was 1, the person was deemed not to have emigrated and was randomly assigned another residence within the exposure area. If the emigration value was zero, the person was deemed to have emigrated. Individuals were tracked through seven residences until they either emigrated or reached 70 years of total exposure (*i.e.*, "died"). The procedure was limited to seven residences to optimize calculation times, because test runs showed that virtually all individuals either emigrated or died before reaching an eighth residence.

Individuals who emigrated were assumed not to return to the exposure area in a subsequent move, which could potentially underestimate the lifetime exposure of some individuals. Total lifetime risk was the aggregate of risks associated with all residences occupied, as follows:

Equation 2

$$Risk_T = \sum_{R=1}^{n} Risk_R$$

Where: $Risk_T$ = Estimated total cancer risk associated with multiple residences
$Risk_R$ = Estimated cancer risk from years in residence R
n = Total number of residences occupied by the individual, up to 7

The simulation results were extrapolated into the full population. Because individuals who "died" or moved away were replaced by new individuals, the size of the true receptor population was greater than was considered by the 70-year analysis. The size of this full receptor population was determined by the ratio of the average aggregate residence time to the total assumed 70-y lifetime.

N.2 Results

Outputs of the probabilistic residence time adjustment are shown below in Table N-3. Figures N-3 and N-4 compare estimated 70-y cancer risk distribution with the residence-time adjusted risks for petroleum refineries and portland cement facilities, respectively.

Table N-3. Comparison of populations exceeding three lifetime inhalation cancer risk benchmarks, with and without adjustment for long-term mobility, for two source categories.

	Portland Cement		Petroleum Refineries	
Cancer Risk	Unadjusted	Adjusted	Unadjusted	Adjusted
> 1e-4	0	0	0	0
> 1e-5	125	43	4,378	2,556
> 1e-6	5,066	2,955	430,800	292,003

Figure N-3.

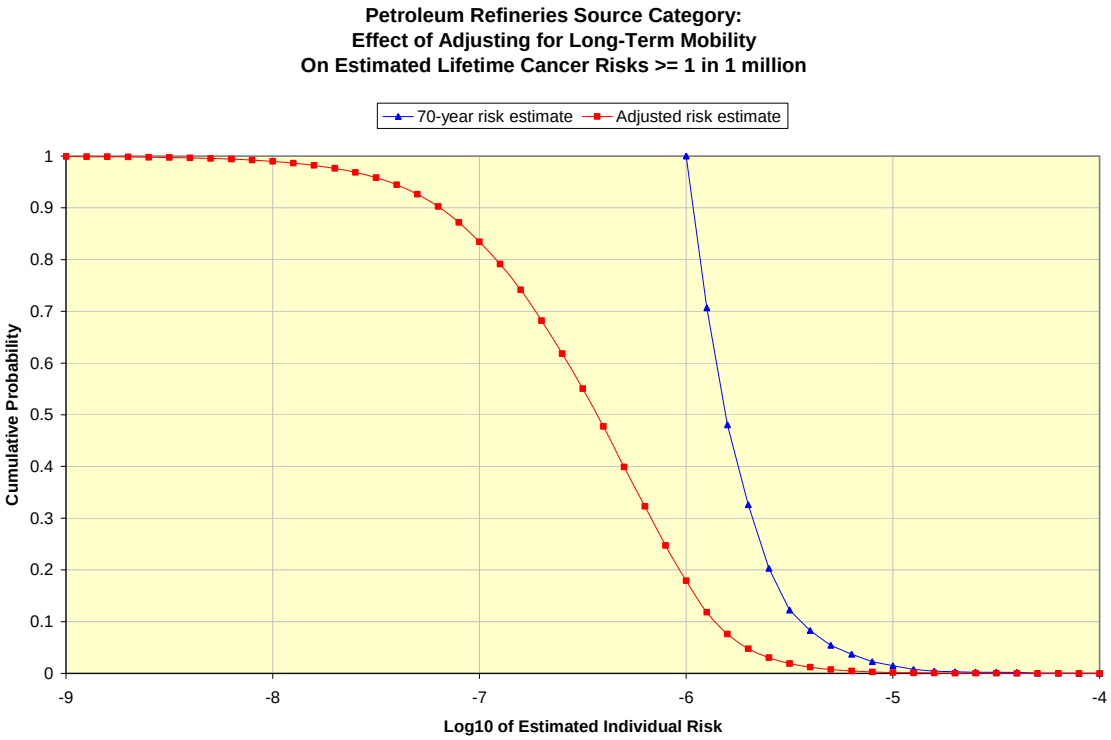
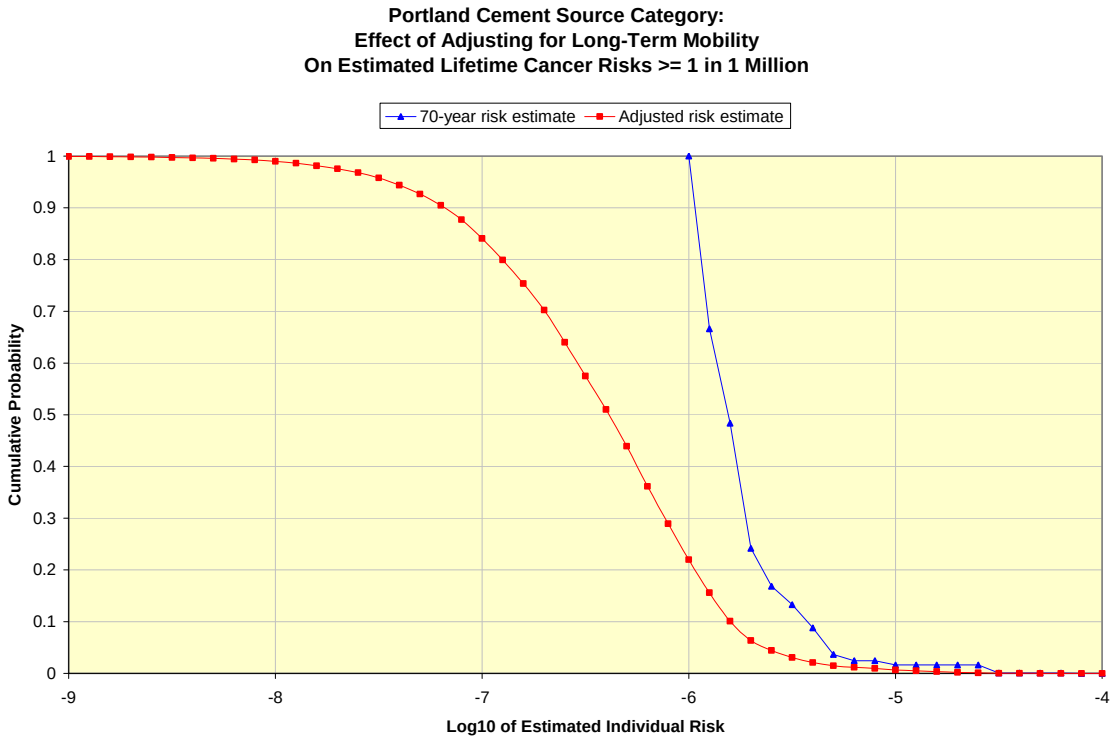


Figure N-4.



N.3 References

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Appendix O: Potential importance of hazardous air pollutants lacking dose-response values

O.1 Introduction

In their comments following the SAB consultation on the first RTR risk assessment plan in December 2006, the panel expressed concern about that EPA's quantitative assessment methods generally omit risks from HAPs that lack peer-reviewed dose-response assessments. The panel requested a sensitivity analysis to test how important the effects of unassessed HAPs might be to the total risk. In response to this comment, we conducted a simple "what-if" analysis based on median and upper-bound estimates of toxic potency for these substances. We included in this analysis the Portland cement and petroleum refinery[1] source categories individually, and also all US sources combined. Calculations were done separately for cancer and effects other than cancer.

This study is intended as a rough range-finding exercise to examine the potential magnitude of risks posed by HAPs that lack dose-response assessments, and to prioritize HAPs for toxicity-testing and dose-response assessment. The results are not intended to propose dose-response ranges for unassessed compounds in refined risk assessments done in support of regulatory decisionmaking.

O.2 Methods

The analysis was based on toxicity-weighting of the 2002 NEI, a process that provides an estimate of relative potential cancer risk and noncancer respiratory hazard posed by each HAP. Health risks associated with exposure to environmental chemicals are a function of (1) the amount of chemical released, (2) the toxicity of the chemical, (3) the dispersion of the chemical in the environment (as influenced by release conditions and meteorology), and (4) receptor exposure (as influenced by receptor location and behavior). Toxicity-weighting represents a partial analysis of health risks, using information covering only areas (1) and (2). Toxicity-weighting is useful as a screening tool because the data are readily available, the analysis can be conducted quickly, and the inputs account for a large part of the variation in risks obtained from a complete assessment. However, toxicity weighting is useful only for relative estimates of risk, and the omission of information in areas (3) and (4) means that toxicity-weighted results are more uncertain than results from a complete assessment. Toxicity-weighting is most appropriately used as a screening and prioritization tool.

We weighted the pollutant emissions as follows: (1) for noncancer respiratory effects, the emitted amount for each chemical was divided by its RfC or similar chronic no-effect exposure level; (2) for cancer, the emitted amount of each chemical was multiplied by its inhalation URE for cancer.

For HAPs that lacked an RfC or URE, we selected as surrogates the following range of values selected from the universe of chronic RfCs and UREs in the OAQPS table of prioritized chronic dose-response values for inhalation exposure (http://www.epa.gov/ttn/atw/toxsource/table1.pdf):

[1] For the petroleum refineries source category, the analysis was conducted using the NPRM draft baseline inventory, which differs slightly from the final version of the inventory described in the main report.

Percentile of toxicity	RfC[2] (mg/m^3)	URE ($1/\mu g/m^3$)
5	2.28	1.0e-6
25	0.2	6.0e-6
50	0.0098	6.8e-5
75	0.00065	6.1e-4
95	0.000023	4.8e-2

All HAPs lacking an RfC were assigned this range of surrogate RfCs. Only HAPs lacking a URE but having an EPA or IARC WOE equivalent to "possible carcinogen" or greater were assigned the range of surrogate UREs. Toxicity-weighted emissions (TWEs) for cancer and noncancer effects were kept separate. TWE's were normalized by dividing each score by the maximum TWE from all chemicals that had a dose-response value.

We did not attempt to reduce these toxicity ranges (e.g., by grouping HAPs by chemical class or structure-activity characteristics) because there is no universally accepted grouping system. Developing and defending such a system would require a major effort that would be beyond the scope of a range-finding exercise.

O.3 Results and Discussion

Results of the analysis are shown in Figures O-1 to O-6. TWEs appear as points for chemicals that have dose-response values and ranges for those that do not. TWE ranges for both carcinogens and noncarginogens spanned about five orders of magnitude (as did the surrogate RfC and URE ranges in the table above). Chemicals on each figure are shown in order of decreasing TWE, with the median TWE value used for sorting ranges. The graphs, with one exception, were limited to the 40 chemicals with the highest TWEs.

For petroleum refineries, Figure O-1 shows that four unassessed noncarcinogens (2,2,4-trimethylpentane, POMs, biphenyl, and carbonyl sulfide) are emitted in amounts that could produce a relative TWE of 0.1 or higher if they had 75th percentile toxicity or worse. Figure O-2 shows only one unassessed carcinogen, quinoline, that could produce a relative TWE of 0.1 or higher if it had 95th percentile carcinogenic potency.

For Portland cement facilities, Figure O-3 shows five chemicals (carbonyl sulfide, POM, 1,3-propane sultone, chromium III, and bromoform) that could produce a relative TWE of 0.1 or higher at 95th percentile toxicity. Of these, only carbonyl sulfide would have a TWE of 0.1 or higher at 75th percentile toxicity. Figure O-4 shows that no unassessed carcinogens would be likely to contribute a TWE greater than 0.1, even at 95th percentile potency.

Considering HAPs emitted from all sources nationally, Figures O-5 shows shows five chemicals (2,2,4-trimethylpentane, carbonyl sulfide, POM, and propionaldehyde) that could produce a relative TWE of 0.1 or higher at 95th percentile toxicity. Of these, only 2,2,4-trimethylpentane would have a TWE of 0.1 or higher at 75th percentile toxicity. Figures O-6a and O-6b show one unassessed carcinogen, ethyl acrylate, with the potential for a TWE greater than 0.1, if it had 95th percentile potency.

[2] Low RfCs connote high toxicity, so the RfC decreases as toxicity increases. UREs are directly proportional to carcinogenic potency, so the URE increases as potency increases.

This toxicity-weighting analysis, while obviously simplistic, is nevertheless useful for determining whether particular assessments have overlooked any potentially important unassessed chemicals, and for informing decisions prioritizing pollutants for toxicity testing and dose-response assessment. Obvious candidates for study or dose-response assessment that emerge from the analysis include 2,2,4-trimethylpentane, carbonyl sulfide, POM, biphenyl, propionaldehyde, and ethyl acrylate. Similar analyses can be conducted easily on other source categories, and with other inventory years, to identify new candidates.

In addition to the limitations discussed in the introduction above, it's important to reiterate that TWE scoring of carcinogens was limited to substances that lacked a URE but had a WOE determination of "possible carcinogen" or worse. This assumes, in effect, that all chemicals that lack a WOE, or that have a WOE of "no data," are not carcinogens. This is unlikely to be true, and for this reason this analysis may underestimate the potential TWE contributions of unassessed carcinogens.

Figure O-1. Petroleum Refinineries: Noncancer Tox-Weighted Emissions for HAPs 1-40

TWE ranges for HAPs lacking RfCs compared with TWEs HAPs with RfCs

(Ranges are 5th, 25th, 50th, 75th, and 95th percentile TWEs)

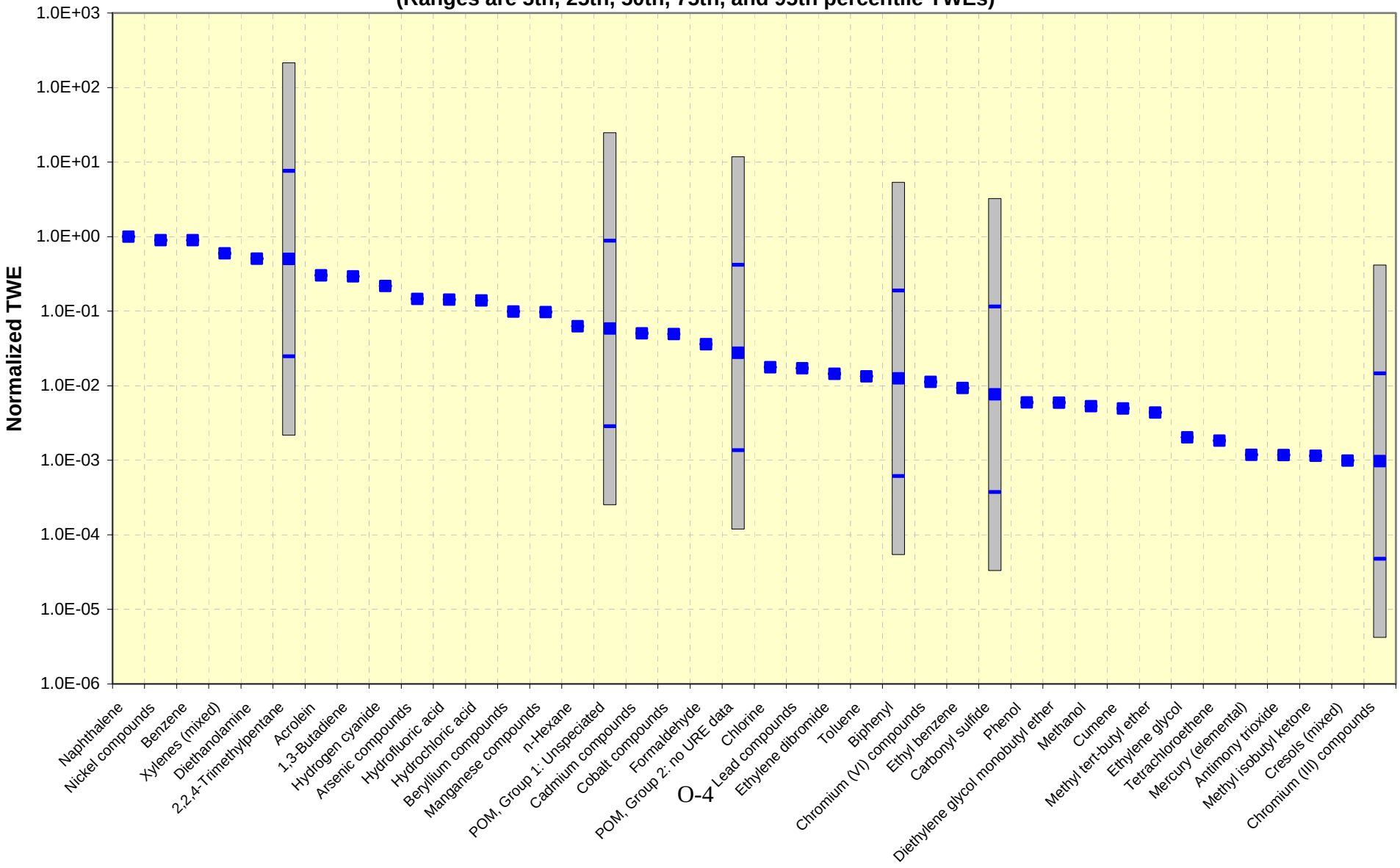


Figure O-2. Petroleum Refineries: Cancer Tox-Weighted Emissions for HAPs 1-40

TWE ranges for HAPs with WOE of "possible" or higher but lacking UREs, compared with TWEs HAPs with UREs

(Ranges are 5th, 25th, 50th, 75th, and 95th percentile TWEs)

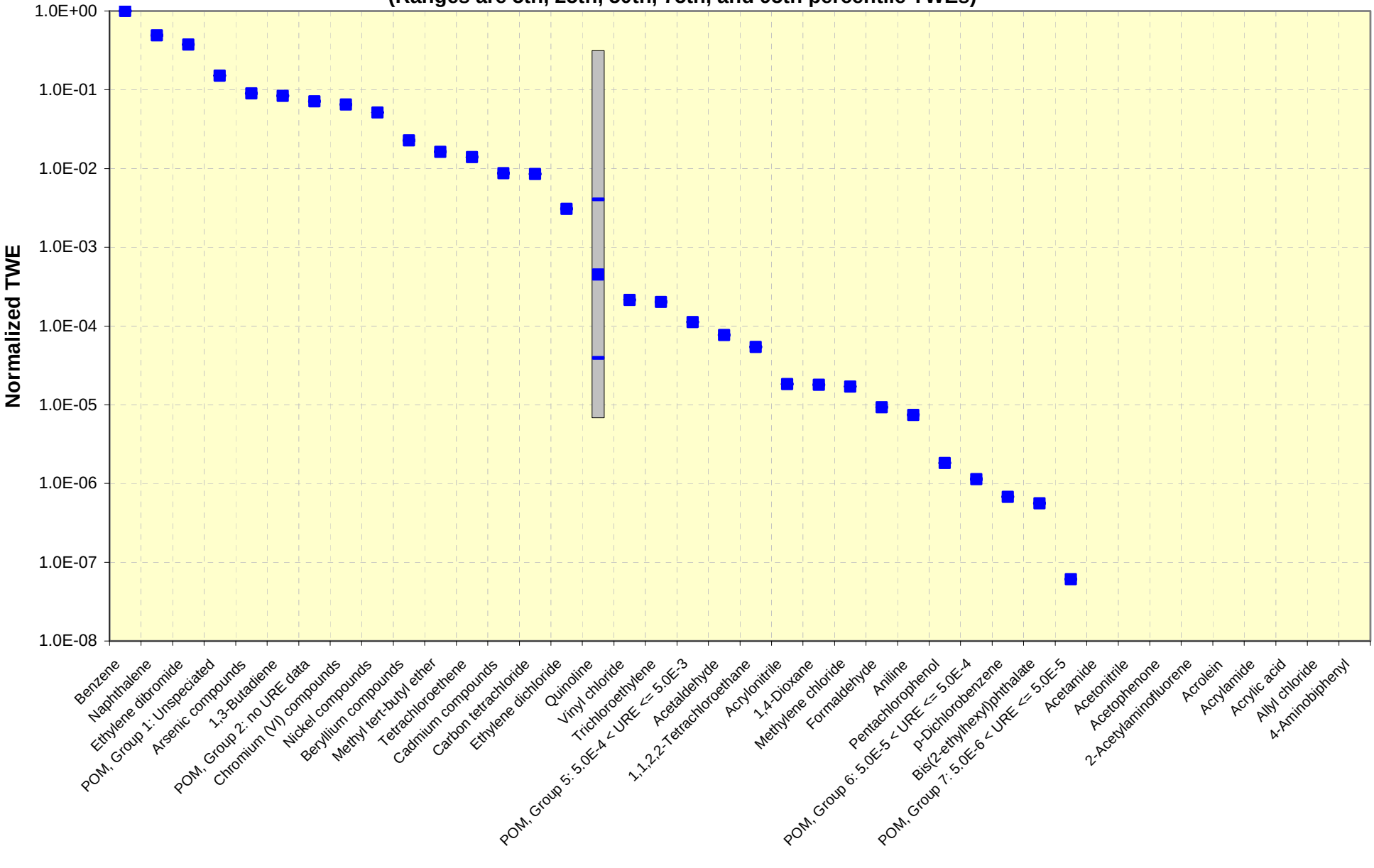


Figure O-3. Portland Cement Facilities: Noncancer Tox-Weighted Emissions for HAPs 1-40

TWE ranges for HAPs lacking RfCs compared with TWEs HAPs with RfCs

(Ranges are 5th, 25th, 50th, 75th, and 95th percentile TWEs)

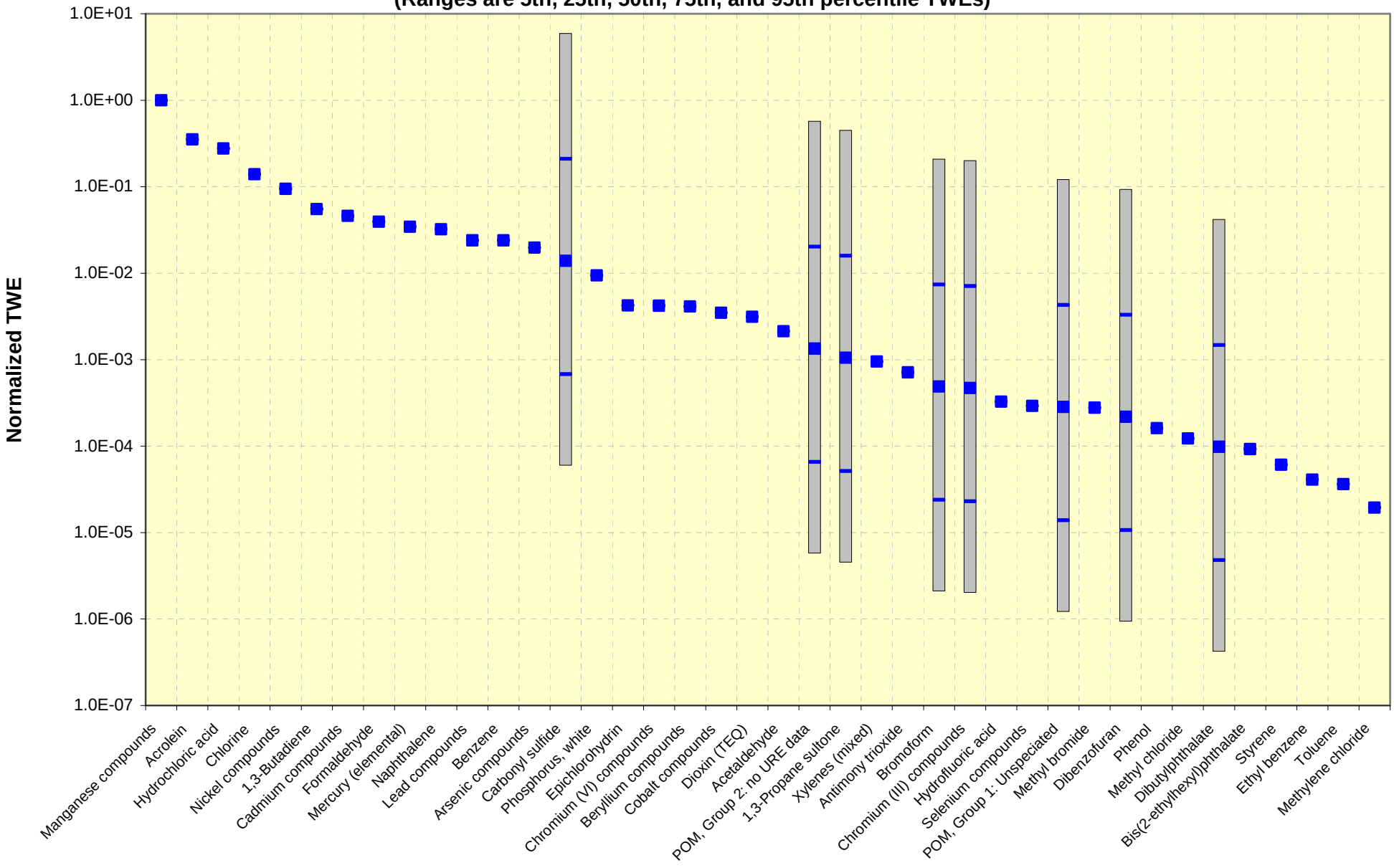


Figure O-4. Portland Cement Facilities: Cancer Tox-Weighted Emissions for HAPs 1-40

TWE ranges for HAPs with WOE of "possible" or higher but lacking UREs, compared with TWEs HAPs with UREs

(Ranges are 5th, 25th, 50th, 75th, and 95th percentile TWEs)

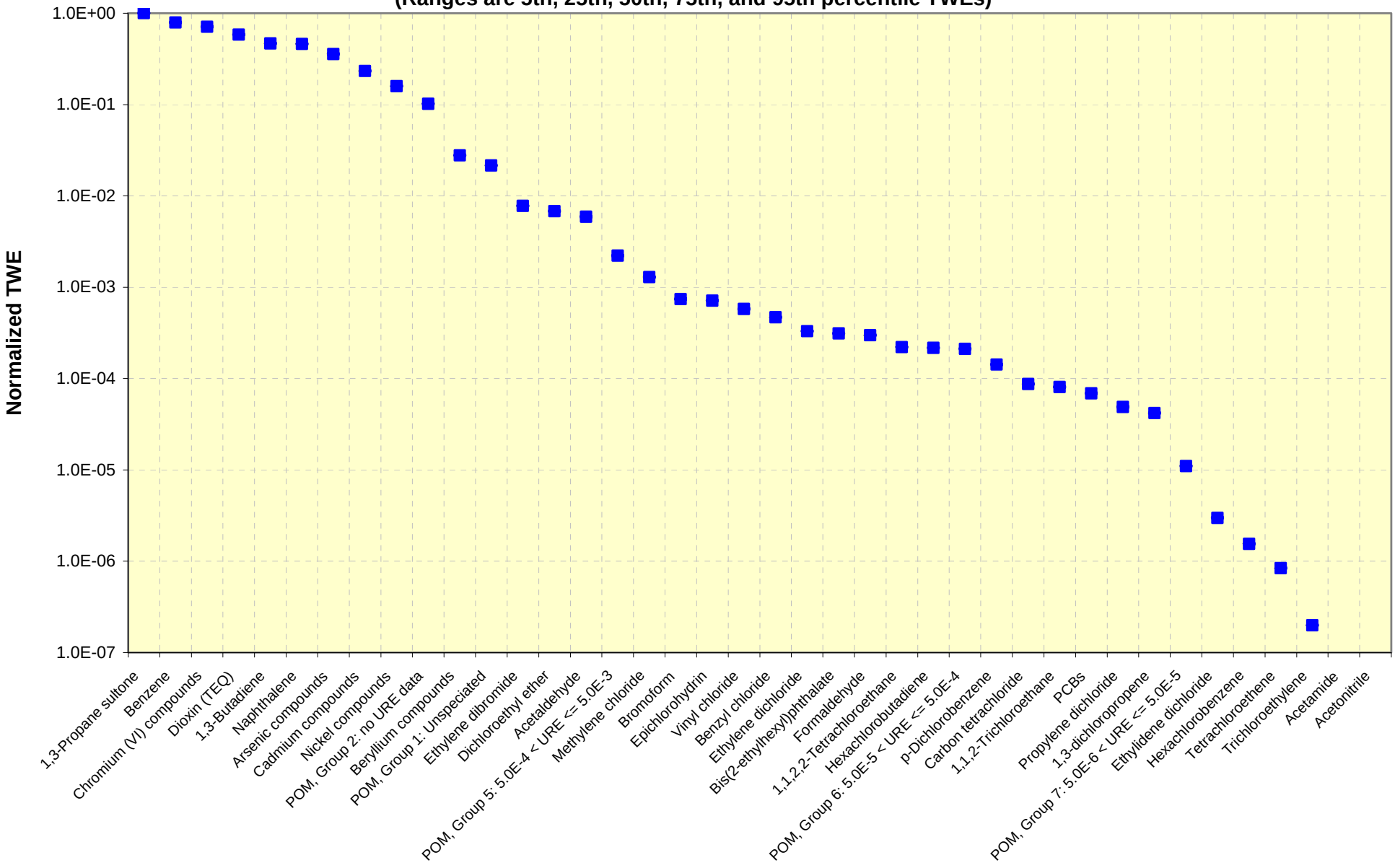


Figure O-5. All NEI sources: Noncancer Tox-Weighted Emissions for HAPs 1-40

TWE ranges for HAPs lacking RfCs compared with TWEs HAPs with RfCs

(Ranges are 5th, 25th, 50th, 75th, and 95th percentile TWEs)

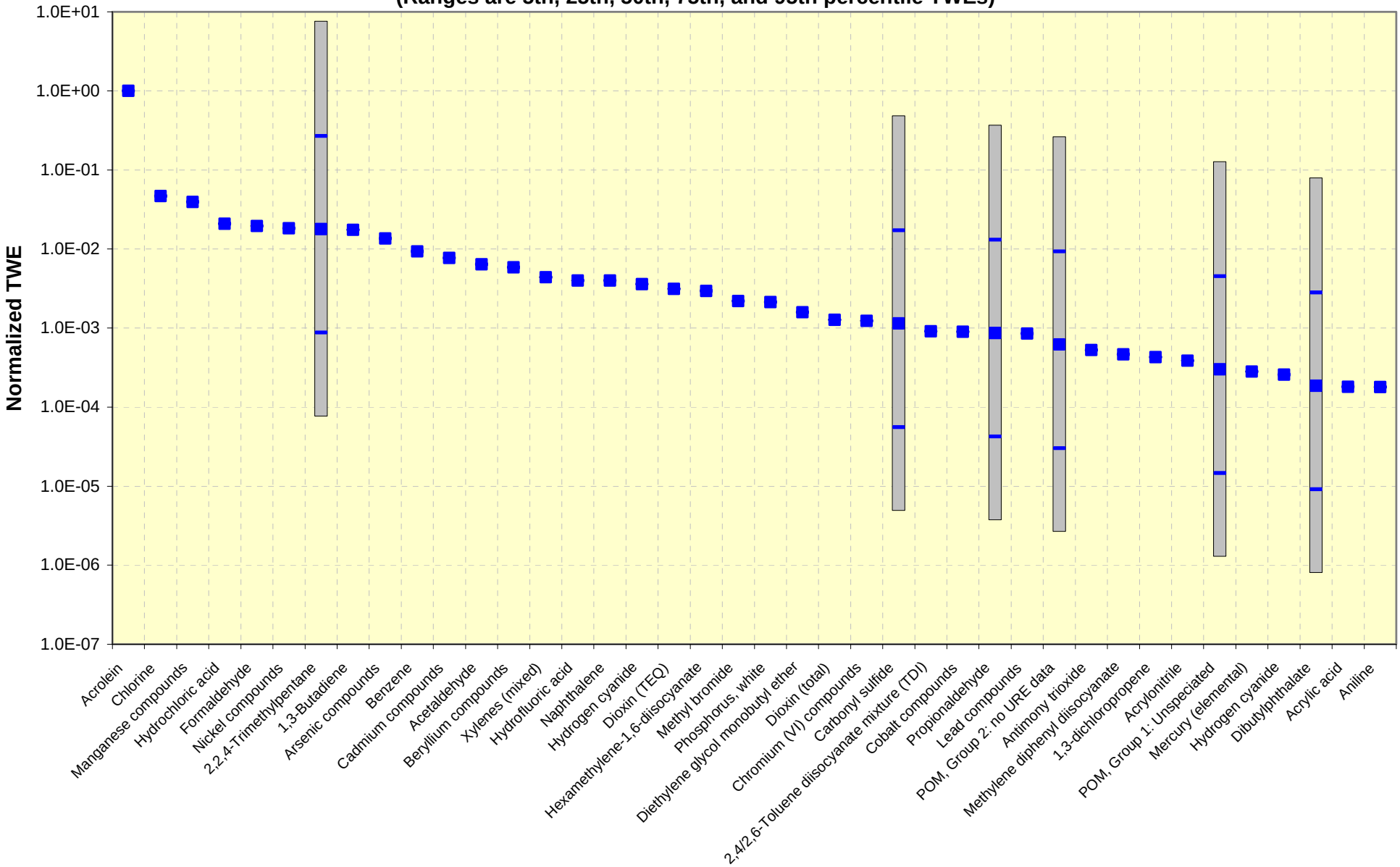


Figure O-6a. All NEI Sources: Cancer Tox-Weighted Emissions for HAPs 1-40

TWE ranges for HAPs with WOE of "possible" or higher but lacking UREs, compared with TWEs HAPs with UREs

(Ranges are 5th, 25th, 50th, 75th, and 95th percentile TWEs)

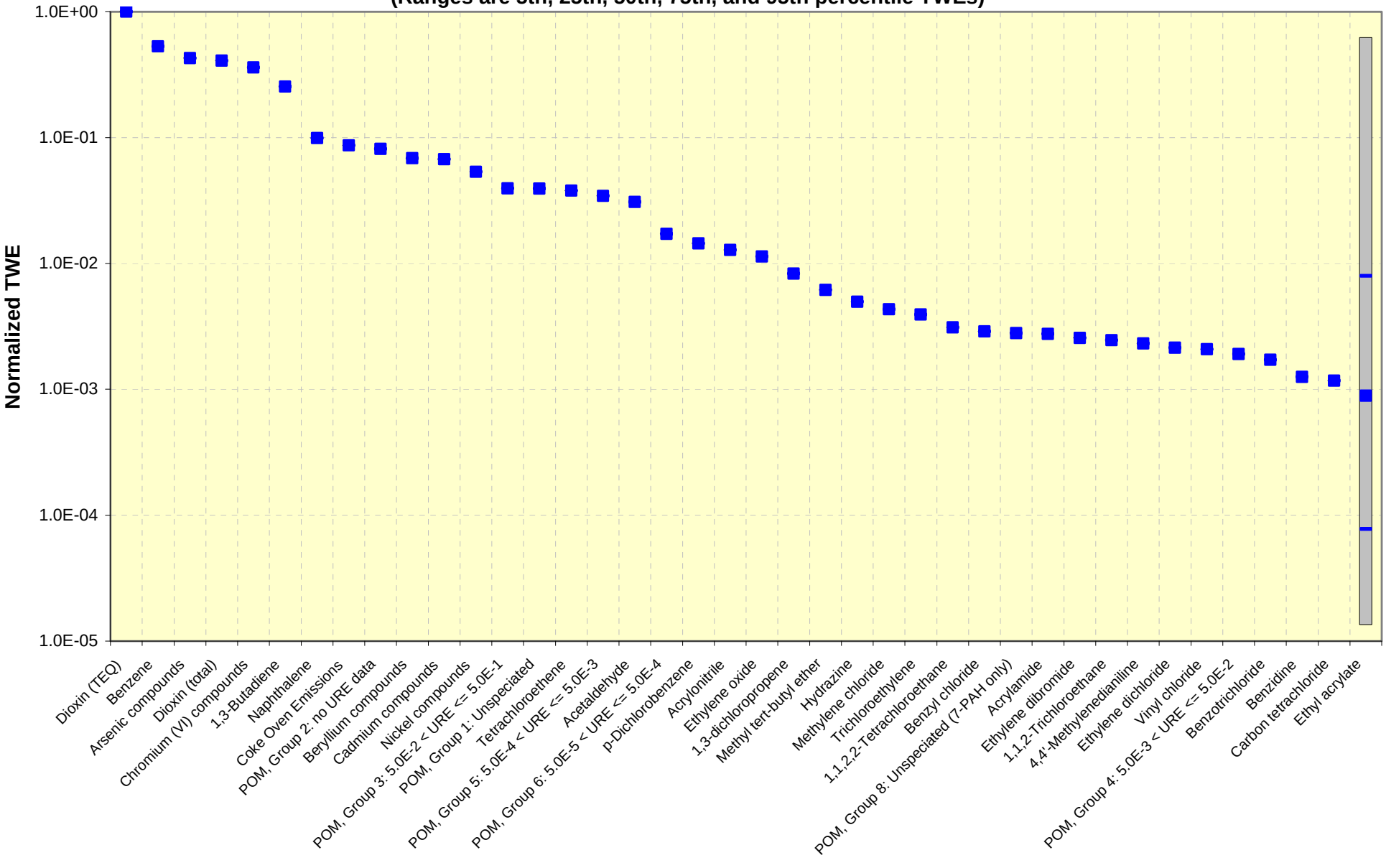
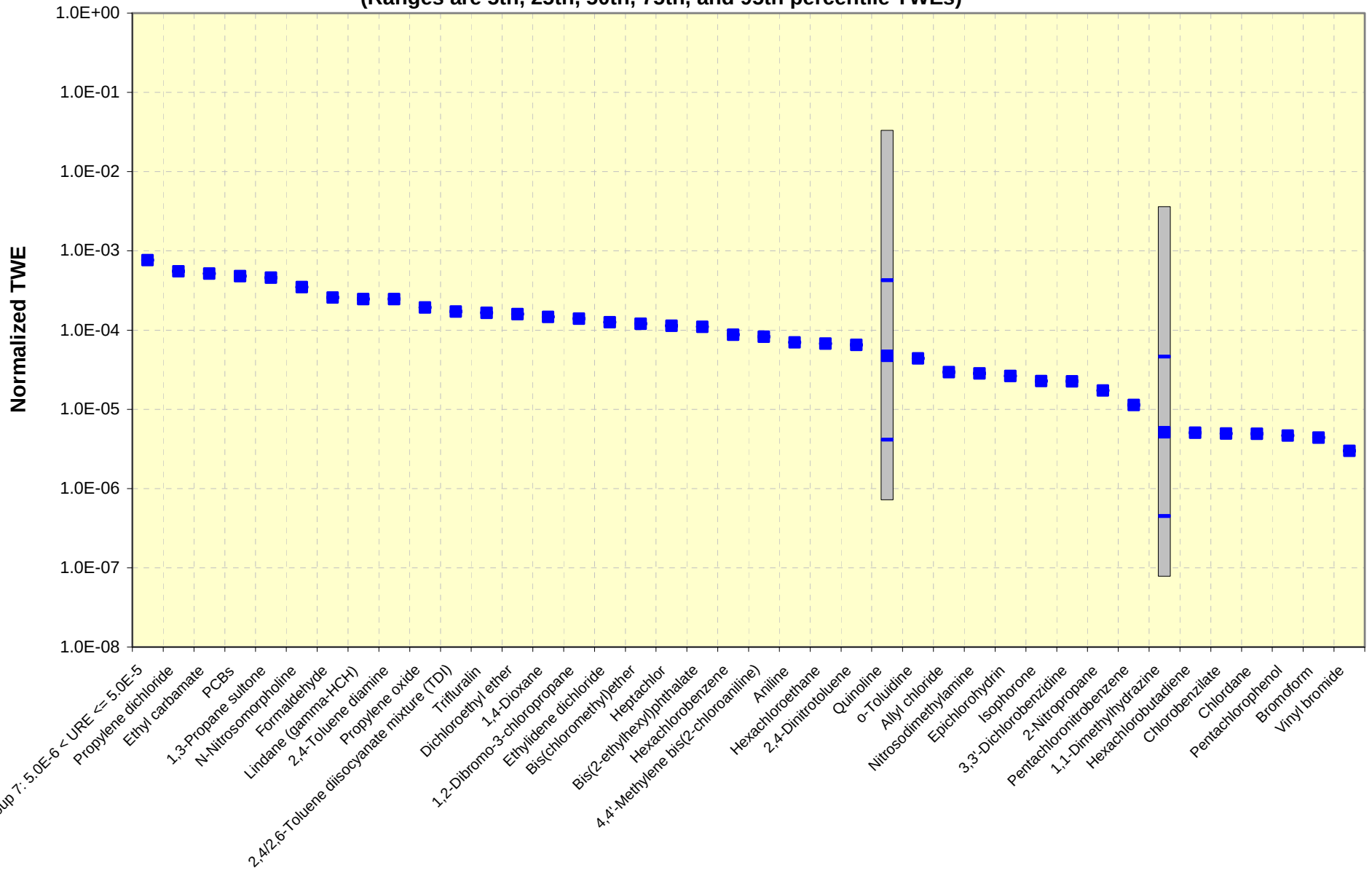


Figure O-6b. All NEI Sources: Cancer Tox-Weighted Emissions for HAPs 41-80

TWE ranges for HAPs with WOE of "possible" or higher but lacking UREs, compared with TWEs HAPs with UREs

(Ranges are 5th, 25th, 50th, 75th, and 95th percentile TWEs)



Appendix P: Comparison of RTR Emissions Inventory Data and Refineries Emissions Model (REM) Data

P.1 Background

Throughout the development of the Risk and Technology Review (RTR) program, one potentially significant area of uncertainty has been the quality of emissions data from individual sources. The general approach has been to model these sources based on data contained in the National Emissions Inventory (NEI), provide the results of this modeling to the public in an Advanced Notice of Proposed Rulemaking, soliciting further data from individual sources or state/local air pollution agencies. While this approach has proved somewhat successful, questions often remain as to the variable quality of much of the data. Given the requirement to examine the potential risks from all hazardous air pollutants listed in the Clean Air Act, inconsistencies often remain across both pollutants and individual sources within a category.

EPA's Science Advisory Board (SAB) and EPA's Office of Inspector General (OIG) have commented on the emissions uncertainties associated with the RTR rulemaking. In addition to expressing concern over emissions uncertainties, both groups have suggested that EPA conduct sensitivity analyses regarding the potential uncertainties in emissions data. Independent of these reviews, EPA has considered anecdotal data on petroleum refineries emissions and has expressed concern that refinery emissions and risk estimates may be understated in the NEI.[1]

For these reasons, we have modeled risk from petroleum refineries using a different set of emissions data, generated using the Refineries Emissions Model (REM). Our aim is to compare two different, but reasonable, sets of emissions data to examine the potential scope of uncertainty in the emissions data and the implications of these differences for estimated cancer inhalation risks. This appendix documents this alternative approach used to assess the baseline emissions and risks from the petroleum refineries MACT I source category. This analysis is based on an emission factor approach, using emission factors along with facility-specific production and throughput data to estimate emissions. In addition to its relevance for this particular source category, this analysis may serve as an example for the RTR program more broadly. Other source categories may have even less certain emissions data in the NEI, perhaps making them candidates for this type of analysis.

In the present analysis, we employed a HAP emissions model developed specifically for petroleum refineries known as the Refinery Emissions Model, or REM (RTI, 2002; Lucas, 2007b). This model was used to generate an "REM" emissions database, including emissions estimates for each refinery in the source category. These emission estimates are compared, by individual pollutant, with those generated using the RTR method (see section 2.2.1 of RTR Methodologies Report). We then used these emissions data to develop alternative risk estimates that are compared to the risk estimated using the RTR emissions data.

P.2 Methods

Emissions and excess cancer risk associated with Refinery MACT 1 emission sources have been estimated from data reported in the NEI; these emission and associated risk estimates are described in the main body of this report. The detailed emission factor analysis described in this

[1] See EPA Docket No. EPA-HQ-OAR-2003-0146, "Potential Low Bias of Reported VOC Emissions from the Petroleum Refining Industry."

appendix is being used to provide an alternative baseline HAP emission estimate for all Refinery MACT 1 emission sources. The modeling approach and assumptions used to estimate the emissions and the source release characteristics have been described elsewhere (RTI, 2002). In essence, the emission factors used in this model are based on MACT compliance, but for the most part do not take into account the impact of any state/local regulations, or any "overcontrol" on the part of facilities beyond MACT requirements. For example, because cooling towers do not currently have a MACT standard, we assume they are uncontrolled, whereas it is likely that some portion of cooling towers is controlled for state regulations or other reasons. We also know that some portion of external floating roof storage vessels have some controls.

Although this analysis uses process-specific production capacities, it provides emission estimates by source type for the facility. For example, the analysis models emissions from classes of storage vessels (*e.g.*, crude oil tanks, gasoline tanks), not the emissions from individual storage vessels. It also accumulates and assigns the emissions to one large area source representing the tank farm rather than attempting to estimate the number and characteristics of individual storage vessels. Since the analysis by RTI (2002), some enhancements to the emission estimates and source characteristic assumptions were made, partly as a result of the "22 Refinery Study" (Lucas, 2007b). These enhancements are described in the Addendum of this appendix.

The REM uses facility-specific data on the types of processes and their capacities to estimate emissions for each refinery in the United States (US) and its territories. The original model includes algorithms for estimating emissions from various petroleum refinery MACT 1 sources, i.e., storage vessels, equipment leaks, wastewater treatment systems, cooling towers, flares, product loading, as well as from various MACT 2 sources, i.e., process heaters, boilers, catalytic cracking units, catalytic reforming units, and sulfur recovery plants (RTI, 2002). While the overall framework of the model is the same, some revisions to the model have been made, as mentioned above. For this analysis, emissions output were only estimated for the MACT 1 sources. The product loading estimate assumes all light and middle distillates are loaded in tanker trucks. Marine vessel loading operations, when co-located at a refinery (and therefore subject to Refinery MACT 1), are typically controlled; these emissions were included in the emissions estimates for flares. Similarly, nearly all miscellaneous vents at a refinery are controlled and the emissions from these vents are also included in the flare emission estimates. Table K in the Addendum is a complete table of facility-specific assumptions used for the REM analysis. Table L shows all of the emissions results by facility. For comparison, Table M provides the RTR emissions by facility.

After emissions estimates have been developed, a dispersion/risk analysis was undertaken. Chronic inhalation exposure concentrations and associated health risk from each facility of interest were estimated using the Human Exposure Model in combination with the American Meteorological Society/EPA Regulatory Model dispersion modeling system (HEM-AERMOD, sometimes called HEM3). More details on the HEM modeling system and the approach used to estimate health risks is outlined in Section 2.2.2 of the RTR Methodologies Report. The REM analysis consisted of modeling 151[2] refineries nationwide. The RTR (NEI-based) risk modeling

[2] While we have emissions data for 153 facilities, 151 facilities included in the REM dataset were modeled in this analysis. One facility that was not modeled is thought to be a duplicate and the other has minor emissions and does not have a corresponding RTR facility with an NEI_ID.

included 156 refineries. However, there are some adjacent refineries that have come under single ownership. At times, the permits for these facilities are merged and the State reports the emissions as a single refinery. In other cases, the permits are kept separate and the State reports emissions for these facilities separately (although under the definition of facility within the CAA, the plants are contiguous and under common ownership/control, so they should be a single facility for the purposes of the CAA). Thus, while there is a small discrepancy in the number of refineries modeled, the two analyses effectively cover identical refining operations.

The risk associated with each facility's estimated emissions was evaluated using the same dispersion models, exposure assumptions, and unit risk factors that were used to estimate risk based on the RTR data. It is important to note, however, that unlike the RTR database for which it is possible to report source-specific locations and release characteristics (18-42% of emission points include unique data, depending on the parameter), these details are not included in REM. Instead we made assumptions about location and other specifications that are described in the Addendum. For example, the REM risk analysis is based on all emissions being released at or near the centroid of the facility and uses default emission source release parameters. As such, differences in the risk results between RTR and REM may be a function of emissions magnitude, but they may also be caused by differences in release characteristics (e.g., individual storage vessels vs. tank farms), and/or emission source locations.

P.3 Comparison of Emission Estimates – REM vs. RTR

The total nationwide HAP emissions estimate at baseline projected by the REM emissions estimates is about 17,800 tons/yr; the total nationwide HAP emissions estimate in the RTR dataset for refineries is about 6,820 tons/yr. Thus, the REM analysis projects approximately 2.6 times higher emissions than the RTR data. As indicated in Table 1, benzene emissions are estimated to be about 1,990 tons/yr nationwide in REM, whereas the RTR dataset includes a total of 693 tons/yr of benzene emissions. REM includes 135 tons/yr of 1,3-butadiene, which is about 8.3 times higher than the 16.2 tons/yr reported in RTR. For naphthalene, REM estimates emissions of about 113 tons/yr, and the RTR data set contains about 77.0 tons/yr. Thus, while the REM data indicate higher emissions of benzene and 1,3-butadiene than the NEI by a factor of 8.3, naphthalene emissions are only about 47 percent higher.

Table 1 shows the HAP emissions estimates for those pollutants included both in the RTR and REM datasets. Overall emissions are higher in the REM dataset for 17 of the 19 HAPs that appear in both estimates. However, there are 37 pollutants that are shown as emissions from at least one facility in RTR that are not included in REM. In addition, REM assumes that most pollutants would be expected from essentially all refineries; only six pollutants are reported to be emitted from more than 100 RTR facilities, and nine pollutants are reported by 50 or more facilities. The pollutants reported by the most facilities in RTR are benzene, toluene, ethyl benzene, xylenes, hexane, naphthalene, cumene, 1,3 butadiene, and methanol.

As shown in Table 1, overall REM emissions are higher by a factor of 2.6, but this factor varies significantly among pollutants. Part of this difference stems from the fact that the REM analysis applies the emission factors at all petroleum refineries, but RTR reports emissions of these pollutants only for a subset of facilities. For some pollutants, the fraction of sources where emissions are reported in the RTR database represents a majority of facilities (e.g., for benzene,

144 of 156 refineries report emissions), but for others this percentage represents significantly less than half of refineries (see Tables 1 and 2).

As we have noted, REM only covers a subset of HAPs, though we believe this includes most of the major hydrocarbons thought to be common to virtually all petroleum refineries. However, the RTR database includes emission estimates for 37 pollutants not covered by REM, reported to be emitted from anywhere from 1 to 34 facilities nationwide. Table 2 lists these pollutants, the amount or RTR-reported emissions, and the number of refineries that reported these emissions. Several of these (e.g., vinyl chloride) are considered to be highly toxic. It is not clear whether these are erroneously reported, a function of specific products of a given refinery, or whether they represent systematic under-reporting for the other refineries in the source category.

At a facility level, there is great variability in the magnitude of difference in emissions. About two-thirds of the facilities have emissions estimates from REM and RTR within the same order of magnitude. However, many REM emission estimates are over one order of magnitude higher, and some are over 1000 times higher. It is unclear from this analysis what factors are driving these differences (e.g., lack of reporting of certain pollutants, difference in quantity of certain pollutants, or incorrect assumptions about emissions in REM).

Table 1: Comparison of HAP Emission Estimates Between RTR and REM Datasets

Pollutant	RTR Emissions (tpy)	# Facilities w/ RTR Emissions	REM Emissions (tpy)	# Facilities w/ REM Emissions
1,3-Butadiene	16.2	71	135	153
2,2,4-Trimethylpentane	137	48	1170	153
Benzene	693	144	1990	153
Biphenyl	3.28	21	11.0	153
Cresols	8.64	27 or 28	112	153
Cumene	52.1	1	162	153
Ethyl Benzene	244	129	506	153
Formaldehyde	7.74	28	23.0	153
Hexane	1180	127	4770	153
Methanol	549	61	10.9	152
Methyl Isobutyl Ketone	92.0	5	925	152
Methyl Tert-Butyl Ether	347	45	2220	153
Naphthalene	77.0	104	113	153
Phenol	17.1	42	88.3	153
POM 71002[A]	16.0	44 to 61	5.15	151
POM 72002[B]	5.28	23 to 58	7.18	151
Styrene	5.46	25	372	153
Toluene	1650	135	3010	153
Xylenes (Mixture of o, m, and p Isomers)	1570	128 to 156	2200	153
TOTAL	**6670**	**156**	**17800**	**153**

[A] POM 71002 is a modeling category that contains the following pollutant descriptions from RTR and/or REM: chrysene, polycyclic organic matter, PAH total, benz[a]Anthracene, 16-PAH, and PNA/PAH.
[B] POM 72002 is a modeling category that contains the following pollutant descriptions from RTR and/or REM: anthracene, fluorine, phenanthrene, pyrene, benzo[g,h,i]Perylene, fluoranthene, acenaphthene, and perylene.
More information on the POM modeling categories can be found at **http://www.epa.gov/ttn/atw/nata1999/99pdfs/pomapproachjan.pdf**.

Table 2. Pollutants in the RTR but not included in the REM database ranked in decreasing order by # facilities reporting.

HAP Category	Emissions (tpy)	# Facilities Reporting [A]
Tetrachloroethylene (Perchloroethylene)	15.3	34
Hydrogen Fluoride (Hydrofluoric Acid)	51.3	32
Diethanolamine	36.7	22
Carbon Disulfide	3.52	15
Carbonyl Sulfide	2.01	15
Acetaldehyde	0.195	14
Ethylene Dichloride (1,2-Dichloroethane)	0.609	11
Hydrochloric Acid (Hydrogen Chloride [Gas Only])	10.7	10
Ethylene Dibromide (Dibromoethane)	0.695	8
Ethylene Glycol	21.8	8
Glycol Ethers	3.16	4 to 8
POM 76002[B]	0.0000482	2 to 7
POM 75002[C]	0.000625	2 to 6
Carbon Tetrachloride	1.66	5
Methyl Chloroform (1,1,1-Trichloroethane)	0.979	5
Trichloroethylene	0.567	5
Dioxins/Furans	0.000105	3 to 5
Chlorobenzene	0.144	4
Methylene Chloride (Dichloromethane)	0.202	4
Bis(2-Ethylhexyl)Phthalate (Dehp)	0.0013	3
Vinyl Acetate	0.0825	3
Dibenzofuran	0.0254	2
Methyl Chloride (Chloromethane)	0.012	2
p-Dioxane	0.013	2
1,1,2,2-Tetrachloroethane	0.0052	1
1,2,4-Trichlorobenzene	0.0003	1
1,4-Dichlorobenzene	0.000245	1
Acetophenone	0.0840	1
Acrylonitrile	0.0015	1
Aniline	0.026	1
Ethyl Chloride	0	1
Methyl Bromide (Bromomethane)	0	1
Pentachlorophenol	0.002	1
p-Phenylenediamine	0.031	1
Propylene Oxide	0	1
Quinoline	0.037	1
Vinyl Chloride	0.137	1
TOTAL	**150**	

[A] A range of numbers may be presented because of potential facility overlap resulting from the aggregation of multiple RTR pollutants into a single HAP category. We did not go back and determine the actual number of overlapping facilities within those categories.

[B] POM 76002 contains individual POM species for which the UREs are between: 5e-5<URE<5e-4. See http://www.epa.gov/ttn/atw/nata1999/99pdfs/pomapproachjan.pdf for more details. For the RTR data for this source category, POM 76002 includes Benzo[b]Fluoranthene, Indeno[1,2,3-c,d]Pyrene, and Benzo[k]Fluoranthene.

[C] POM 75002 contains individual POM species for which the UREs are between: 5e-4<URE<5e-3. For this source category, RTR POM 75002 includes Benzo[a]Pyrene and Dibenzo[a,h]Anthracene.

P.4 Comparison of Risk Estimates

In general, we see a modest increase in risk estimates for REM compared to RTR modeling. Table 3 indicates that the highest maximum individual risk (MIR) for an individual facility (i.e., the source category MIR) for the REM and RTR analyses. The highest MIR using REM data is 30 in one million (3×10^{-5}) using the high-end benzene potency and 20 in 1 million using the low-end benzene potency.[3] The source category MIR for the REM analysis was driven by benzene, naphthalene, and POM. The highest MIR (the source category MIR) is also 30 in 1 million (3×10^{-5}) based on RTR data, but it occurs at a different facility. The source category MIR for the RTR analysis was driven by naphthalene and POM. Because benzene is not a driver at this facility, the MIR using RTR data is also 30 in 1 million using the low-end benzene potency estimate.

Additionally, the distribution of individual facility MIRs for the entire source category is shifted upward using REM data as compared to RTR, assuming the high-end cancer potency value. Using the REM emissions estimates 135 facilities have an MIR greater than 1 in 1 million and 45 facilities have a MIR greater than 10 in 1 million. Using the RTR emissions data, 77 facilities had MIRs greater than 1 in 1 million and 5 facilities had MIRs greater than 10 in 1 million. We do not know what the distribution of REM or RTR facility MIR estimates would be using the equally probable lower estimate of benzene potency.

The estimate for cancer incidence using the REM emissions estimates is three to four times higher than the incidence estimate using the RTR emissions estimates. Using the low-end cancer potency value, the REM incidence is 0.1 excess cases per year and the RTR incidence is 0.03 excess cases per year. Using the high-end benzene cancer potency value, the REM incidence is 0.2 excess cancer cases per year and the RTR incidence is 0.05 excess cancer cases per year. These results are also displayed in Table 3 below. Looking across facilities, about two-thirds of the facilities, as analyzed, were within the same order of magnitude, most of the rest were one order of magnitude different, and a handful of outliers were two or more orders of magnitude different. Table J in the Addendum shows the full set of cancer incidence estimates.

The EPA IRIS assessment for benzene provides a range of plausible unit risk estimates. This comparative analysis used the highest value in that range, 7.8E-06 per ug/m^3, and provides a conservative estimate of potential benzene cancer risks. The low end of the range is 2.2E-06 per ug/m^3. We applied this low-end value to estimate the potential range in cancer incidence, shown in Table 3, but did not use it in any other aspect of the REM analyses. In the RTR analysis, we were able to report the source category MIR because benzene was not a driver at that facility. The distribution of facility MIRs in both REM and RTR is based on the high-end benzene cancer potency value. Therefore, the distribution of facility MIRs from the REM and RTR analyses could be lower (and not necessarily proportionately so) when the lower estimate for benzene is applied.

[3] The EPA IRIS assessment for benzene provides a range of plausible unit risk estimates between 2.2E-06 per ug/m^3 and 7.8E-06 per ug/m^3. While we originally did this analysis using the high-end of that range, we have since tried to add low-end calculations where possible without completely remodeling.

Table 3. Summary of Risk Estimates Projected from the RTR and REM Analyses

Parameter	REM	RTR
Number of facilities modeled	151	156
Annual HAP emissions (tons/yr)	17,800	6,820
Highest Maximum Individual Lifetime Cancer Risk (MIR, in 1 million) from any one Refinery	20 to 30 (benzene, naphthalene, POM)	30 (naphthalene, POM)
No. Facilities with MIR ≥ 100 in 1 million	0	0
No. Facilities with MIR ≥ 10 in 1 million	41	5
No. Facilities with MIR ≥ 1 in 1 million	135	77
Estimated Cancer Incidence (excess cancer cases per year)	0.1 to 0.2	0.03 to 0.05
Contribution of HAP to Cancer Incidence[A]		
benzene	63%	48%
naphthalene	17%	21%
1,3-butadiene	11%	5%
POM[B]	6%	15%

[A] These percentage contributions are based on the high-end benzene cancer potency value. They likely will be different assuming the low-end benzene cancer potency value.
[B] POM refers to groups 71002 and 72002 in the REM dataset because no other groups are represented in REM.

P.4.1 Facility Risks

Looking across facilities, the relative ranking of facility-specific MIRs varied between the RTR and REM approaches. Table 4 shows the 20 highest facility MIRs using REM data, the corresponding RTR MIR estimates, and the magnitude of difference in the emissions estimates. Table 5 similarly shows the 20 highest facility-specific MIRs based on the RTR data and the corresponding MIRs using REM data.

Only two facilities are ranked among the top 20 facilities in both analyses. Interestingly, all but one MIR estimates based on RTR data (Table 5) are higher than the corresponding REM MIR estimates at those same facilities; however, these differences are less than 10-fold and almost half (9) are roughly the same, i.e., have ratios of 1. Similarly, the highest MIRs using REM data are almost all higher than corresponding RTR MIRs (Table 4), but there is more variability in the magnitude of difference. About half of the MIRs for these facilities are less than 10-fold higher than the corresponding RTR MIR estimates. Also, two of these facilities have a three-order magnitude of difference. A full comparison of MIR estimates is included as Table I in the Addendum.

As mentioned previously, this section is based on cancer MIR values assuming the high-end benzene cancer potency value. The comparisons would likely be different assuming the low-end benzene potency value because of the difference in benzene emissions estimates between REM and RTR datasets; however, without specifically calculating those values, we cannot say how different they would be.

Table 4. 20 Highest Maximum Individual Risk at REM facilities vs. RTR estimates[A]

Facility ID	REM Cancer MIR (in 1 million)	RTR Cancer MIR (in 1 million)	Ratio (REM/RTR)
PET_NEI34872	30	1	40
PET_NEI109	30	4	7
PET_NEI46556	30	6	5
PET_NEI40732	30	5	6
PET_NEI20467	20	1	20
PET_NEICA1910268	20	10	2
PET_NEI6022	20	10	2
PET_NEI7781	20	6	3
PET_NEI11450	20	2	10
PET_NEI11192	20	1	30
PET_NEI20154	20	0.007	3000
PET_NEI18406	20	9	2
PET_NEI6130	20	6	3
PET_NEI11574	20	5	4
PET_NEI42309	20	20	1
PET_NEI13371	20	5	4
PET_NEICA0370363	20	2	10
PET_NEI6519	10	5	2
PET_NEI33039	10	10	1
PET_NEI876	10	20	0.7

[A] Numbers in this table are rounded to one significant digit. Facilities were determined by sorting first by descending REM Cancer MIR then by descending REM Cancer Incidence, and the list was capped at 20.

Table 5. 20 Highest Maximum Individual Risk at RTR facilities vs. REM estimates [A]

Facility ID	RTR Cancer MIR (in 1 million)	REM Cancer MIR (in 1 million)	Ratio (RTR:REM)
PET_NEI12711	30	9	3
PET_NEI34898	20	7	3
PET_NEI12988	20	10	2
PET_NEI33031	20	10	2
PET_NEI42309	20	20	0.8
PET_NEI42040	20	10	1
PET_NEI876	20	10	1
PET_NEI34057	10	2	9
PET_NEI41771	10	9	1
PET_NEI6475	10	5	3
PET_NEI6095	10	1	9
PET_NEI6087	10	8	1
PET_NEI6436	10	10	1
PET_NEIPRT$64	10	2	5

Facility ID	RTR Cancer MIR (in 1 million)	REM Cancer MIR (in 1 million)	Ratio (RTR:REM)
PET_NEI34050	10	5	2
PET_NEI20174	10	5	2
PET_NEI18394	10	8	1
PET_NEICA1910268	10	20	0.5
PET_NEI32864	10	10	1
PET_NEI40371	10	4	2

[A] Numbers in this table are rounded to one significant digit. Facilities were determined by sorting first by descending REM Cancer MIR then by descending REM Cancer Incidence, and the list was capped at 20.

P.4.2 Pollutant Risks

For the highest REM facility MIR, benzene, naphthalene, and POM were the risk drivers, assuming the high-end benzene potency value. Naphthalene and POM were the risk drivers for the RTR MIR. Benzene, naphthalene, 1,3-butadiene, and POM were the risk drivers for the REM cancer incidence. These were also drivers for the RTR cancer incidence, but benzene and 1,3-butadiene contribute more overall using REM data. We did not assess how these HAP contributions to cancer incidence using REM or RTR would change assuming the lower estimate of benzene cancer potency.

Whereas we determined the emissions of benzene are about three times more in REM than RTR, they make up about 15% more of the relative cancer incidence risk. 1,3-butadiene emissions are about eight times greater in REM than RTR, and their relative contribution to overall cancer incidence is about double using REM than using RTR data. The relative influence of naphthalene on cancer incidence is roughly the same, and the influence of POM is greater using RTR than using REM data. RTR contains two more toxic groups of POM that are not included in the REM data. In addition to total quantity of these pollutants emitted, the number of facilities reporting these pollutants (only two-thirds of RTR facilities report naphthalene and one-half report 1,3-butadiene emissions) along with the relative contributions of pollutants that are not included in REM may also influence these contributions. The relative contributions of individual pollutants to the overall REM and RTR cancer incidence would likely change when calculated using the low-end benzene potency value.

P.5 Limitations and Uncertainty

While this analysis provides a general comparison of the standard inventory approach to gathering emissions data to the emission factor approach, using REM in this case, it is not without significant uncertainties. Some of the major differences are described in detail, and Table 5 includes a list of specific differences in the two approaches.

P.5.1 Emissions Estimates

Both RTR and REM emissions data are modeled estimates, based on few, if any, actual site-specific measured data. RTR emissions estimates typically do not include record of calculation method and are based on the 2002 NEI with some information updated through 2005. They are rarely measured and there may be some similarities between the method used for REM and the

methods used at some facilities and/or states to compile RTR data. REM-based emissions estimates are calculated using emissions factors and are generally a function of production and process charge capacities based on the Energy Information Administration's (EIA) *Petroleum Supply Annual 2004* (EIA, 2005). As with any generic approach, the REM analysis cannot account for differences due to site-specific modifications at individual facilities, and actual emissions may be greater or less than estimated for the purposes of this analysis. The assumptions made about particular emission point (e.g., storage tanks) specifications are explained in the Addendum to this Appendix. Also, as all these data are limited to annual emission rates; this analysis does not attempt to estimate short-term releases or health risks associated with such releases.

For REM, we have attempted to use emission factors that are consistent with the requirements associated with the existing MACT regulations. For those emission points not controlled by the existing MACT standards (i.e., cooling towers), no controls are assumed. If facilities control emissions beyond the level of MACT, whether to meet state/local regulations, to provide a "buffer" below those allowed under MACT, or for any other reasons (e.g., occupational exposure reduction), those controls are not reflected in this analysis other than some state control considered for equipment leaks. The fact that additional control beyond what is allowed (either uncontrolled or to meet MACT), is not considered in REM may account for differences between RTR and REM emission estimates. The extent of the difference they account for is unknown because we do not have facility-specific control data.

P.5.2 Pollutant Coverage

The REM covers 19 organic pollutants and pollutant categories that represent the majority of HAP emissions by mass. This does not represent the full range of possible pollutants emitted from at least some facilities in this source category; the RTR database reports emissions of 37 additional HAP categories for MACT I petroleum refineries. While much less important in terms of gross emissions, several of these pollutants are relatively potent in terms of their potential health effects. For example, RTR indicates that some facilities emit from MACT I processes tetrachloroethylene (i.e., perchloroethylene), some of the more toxic POM species, dioxins/furans, and vinyl chloride, some of which have relatively high cancer potency values, and the REM analysis does not address these pollutants.

We are uncertain to the extent to which these missing pollutants should be considered for more facilities within this source category, and we have not evaluated the impact they would have on overall MIR and cancer incidence if we did include them more broadly.

P.5.3 Facility Risk Modeling

Whereas RTR sometimes contains detailed emission point specifications (18-42% of the time, depending on the parameter, for petroleum refineries), REM estimates emissions more broadly, using default stack parameters and not accounting for specific number of emission points or their locations within the facility. Therefore, we have had to make assumptions about the size and location of these sources within the facility, as described elsewhere in this appendix (for example, see Addendum). Placing area sources in the center of facilities tends to dampen the extreme risk estimates from those sources, assuming that risk is independent of where these sources are actually located. Therefore, this may result in an understatement of high MIRs.

While these differences influence the risk results, we are uncertain of the magnitude of their influence without doing more targeted and detailed analysis of this question.

Table 5. Differences between the REM-based and RTR-based emissions and risk estimates

	RTR	REM
Emissions estimates methodology	Methods unreported. For some emission points, such as equipment leaks and cooling towers, refineries may estimate using monitoring data and equipment leak correlation equations. There is no national requirement to produce emissions estimates using a standard protocol or identifying what emissions points must be reported.	Emissions factors from AP-42 or RTI (2002).
Pollutant coverage	There are no national requirements for what pollutants must be reported. For petroleum refineries, RTR happens to contain 56 HAP reported at between one and 144 facilities of 156. Additionally, there are no standards for speciating data. For example, sometimes VOCs are reported but not speciated by HAP and they are not included in RTR.	REM was designed to include 19 pollutants that were thought to cover the common pollutants from all refineries. As such, emissions of these pollutants are estimated for each facility.
Level of control assumed	If controls are on, emissions estimates account for them, but RTR does not have facility-specific control information.	REM generally assumes facilities are controlled at the MACT level. For cooling towers, which are not currently controlled by MACT are assumed to be uncontrolled. Estimates for equipment leaks account for control requirements from states and consent decrees.
Modeling parameters	Depends on what information is provided in RTR. While about 40% of emission points include facility-specific stack height, only about 20% include facility-specific temperature, diameter, flow rate, and velocity. If facility-specific data are not known, national-, source classification code (SCC)-, or standard industrial classification (SIC)-defaults are applied.	Assume emission points are located in the center of the facility. Apply tiered size categories based on refinery crude capacity. REM assigns stack parameters based on the generalized SCC.

P.6 Summary

Emissions estimation and risk modeling are complex processes and given the uncertainties discussed above (e.g., differences in modeling of area sources), it is challenging to draw firm conclusions as to the reasons for these findings. The following are the salient points we believe one can take from this analysis:

1. Across all petroleum refineries and HAPs, total HAP emissions estimated with REM are 2.6 times higher than those in the RTR database. At the individual facility level, the differences can span an order of magnitude or more.

2. On an aggregate level, the MIR results of the REM analysis are similar to the RTR results. The source category MIR for both the REM and RTR analyses was 30 in a million (though not at the same facilities) using the high-end benzene cancer potency value. Using the low-end benzene value, the MIR in the REM analysis dropped to 20 in 1 million while the MIR in the RTR analysis remained 30 in 1 million because the source category MIR for the RTR analysis was driven by POM and naphthalene, and not benzene. The source category MIR for the REM analysis was driven by benzene, naphthalene, and POM.

3. Assuming the high-end benzene potency, we found a shift toward higher facility MIR estimates. 135 facilities in the REM analysis have MIR estimates greater than 1 in 1 million and 41 facilities have MIR estimates greater than 10 in 1 million, whereas in the RTR analysis, 77 facilities have risks greater than 1 in 1 million and five facilities have MIR estimates greater than 10 in 1 million. We do not know what the distribution of facility MIR estimates for REM or RTR is using the equally-probable low-end estimate of benzene potency.

4. The top 20 facilities with the highest MIRs based on RTR data have REM-based MIR estimates within the same order of magnitude. For the top 20 REM-based MIR estimates, there was somewhat more variability in the magnitude of differences compared to RTR-based MIR estimates; 14 of these facilities showed differences in estimates of less than an order of magnitude, but the remainder of differences were at least a factor of 10 (and as high as 3,000-fold). Using the low-end benzene estimate may alter these differences, depending on the relative amounts of benzene estimated at each facility.

5. The facilities with the highest MIRs (using the high-end benzene cancer potency value) in either approach are generally different facilities. This suggests a more pronounced difference in the influence of the emissions estimation approach at the facility level than in aggregate. Additionally, the facilities with the highest MIRs in either case, with two exceptions, are not among the facilities with the most dramatic differences in emissions. These order of magnitude changes for facilities did not shift any individual facilities to have MIRs greater than or equal to 100 in 1 million, but we cannot judge how alternative emissions estimation approaches might affect other source categories. We did not evaluate this issue using the low-end cancer potency value.

6. Depending on which benzene cancer potency estimate is used, the estimate for cancer incidence using the REM emissions estimates is three to four times higher than the

incidence estimate using the RTR emissions estimates (using the high-end benzene potency estimate, REM incidence is 0.2 cases per year and RTR incidence is 0.05 cases per year; using the low-end benzene potency estimate, REM incidence is 0.1 cases per year and RTR incidence is 0.03 cases per year).

7. Petroleum refineries are highly regulated facilities for which emissions are thought to be relatively well understood compared to many other source categories. The relative similarity in MIRs may be unique in this case. It is difficult to generalize the results of this analysis to other source categories.

P.7 References

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Addendum

Details of Modeling Approach

The basis for this emission factors modeling approach can be found in *Petroleum Refinery Source Characterization and Emission Model for Residual Risk Assessment* (RTI, 2002), with more recent adjustments as described here. REM emissions estimates are generally a function of production and process charge capacities.

Revised Emission Factors for Equipment Leaks

For equipment leaks, based on the 22 Benzene Study (Lucas, 2007b), revised emission factors were developed to account for different stringencies of leak detection and repair (LDAR) programs. Three tiers of LDAR programs were defined based on leak definitions and inclusion of connectors as follows:

1) Leak definition of 500 or 1,000 ppmv including connector monitoring
2) Leak definition of 500 or 1,000 ppmv; no connector monitoring
3) Leak definition of 10,000 ppmv

The emission factors for benzene were projected using the small and large model plant equipment component counts and average benzene concentrations for various refinery process units from *Locating and Estimating Emissions of Benzene* (USEPA, 1998). The equipment leak emission factors are summarized in Table A.

Table A. Emission Factors for Benzene from Fugitive Equipment Leaks

Process Unit	Large Refinery Cut-off	Emissions of Benzene (tons/yr per process unit)					
		Tier 1		Tier 2		Tier 3	
		Small	Large	Small	Large	Small	Large
Crude Distillation	50,000	0.0146	0.0296	0.0326	0.0631	0.0628	0.1247
Vacuum Distillation	25,000	0.0018	0.0067	0.0038	0.0125	0.0074	0.0265
Catalytic Cracking	17,500	0.0108	0.0111	0.0218	0.0255	0.0456	0.0475
Catalytic Reforming	10,000	0.0409	0.0530	0.0858	0.1131	0.1688	0.2253
Hydrocracking	5,000	0.0180	0.0292	0.0382	0.0816	0.0741	0.1347
Thermal Cracking (coking)	10,000	0.0063	0.0110	0.0139	0.0278	0.0277	0.0481
Thermal Cracking (visbreaking)	10,000	0.0103	0.0184	0.0192	0.0415	0.0432	0.0769
Hydrotreating/Hydrorefining	35,000	0.0130	0.0185	0.0283	0.0415	0.0545	0.0790
Alkylation (sulfuric acid)	5,000	0.0044	0.0044	0.0097	0.0092	0.0180	0.0187
Isomerization	2,500	0.0377	0.0298	0.0768	0.0653	0.1584	0.1295
Polymerization/Dimerization	1,000	0.0014	0.0015	0.0023	0.0033	0.0064	0.0062
Full-Range Distillation	5,000	0.0145	0.0254	0.0282	0.0557	0.0640	0.1069
Aromatics (as CRU)	5,000	0.0409	0.0530	0.0858	0.1131	0.1688	0.2253
Product Blending	5,000	0.0233	0.0282	0.0523	0.0573	0.1003	0.1195

Table A. Emission Factors for Benzene from Fugitive Equipment Leaks

Process Unit	Large Refinery Cut-off	Emissions of Benzene (tons/yr per process unit)					
		Tier 1		Tier 2		Tier 3	
		Small	Large	Small	Large	Small	Large
Hydrogen Plant (MMcfd)	10	0.0001	0.0004	0.0001	0.0007	0.0002	0.0021
Other Lube Oil Processes	5,000	0.0112	0.0094	0.0250	0.0204	0.0509	0.0412
MEK Dewaxing	5,000	0.0020	0.0056	0.0044	0.0128	0.0078	0.0206
Asphalt Plant	5,000	0.0005	0.0001	0.0011	0.0004	0.0022	0.0006
Sulfur Plant	75	0.0001	0.0001	0.0004	0.0002	0.0006	0.0004

Each refinery was assigned an equipment leak code based on its consent decree requirements or State requirements so that appropriate equipment leak benzene emission factors were assigned to each refinery. For refineries where this information was not available or applicable, the default values for leak definition of 10,000 ppmv were used (Equipment Leak Code = 3). The emission factors for benzene for each of the process units that are present at the refinery were summed to calculate the facility's total benzene emissions from equipment leaks. The total benzene emissions were subsequently multiplied by refinery-wide average process stream individual HAP to benzene concentration ratios to calculate the emissions of other HAP at the refinery. These concentration ratios were revised based on the relative volume of each processing or product stream to crude input. The average concentration ratios used to estimate the fugitive equipment leaks emissions for HAP other than benzene are summarized in Table B. These concentration ratios were multiplied by the total mass fugitive equipment leak emissions calculated for benzene to estimate the fugitive equipment leak mass emissions of the other HAP compounds.

Table B. Concentration Ratios Used for Equipment Leak Emission Estimates

CASRN	HAP	Average Refinery Stream Liquid Concentration[a] (wt%)	Ratio of HAP to Benzene Concentration[b]
106-99-0	1,3-Butadiene	0.0007	0.0006
540-84-1	2,2,4-Trimethylpentane	2.27	1.97
71-43-2	Benzene	1.15	1
92-52-4	Biphenyl	0.040	0.034
1319-77-3	Cresols	0.29	0.25
98-82-8	Cumene	0.43	0.37
100-41-4	Ethylbenzene	1.02	0.88
110-54-3	Hexane	4.05	3.50
1634-04-4	Methyl tertiary butyl ether	0.67	0.58
91-20-3	Naphthalene	0.33	0.29
108-93-0	Phenol	0.21	0.18
100-42-5	Styrene	0.67	0.58
108-88-3	Toluene	3.86	3.34

Table B. Concentration Ratios Used for Equipment Leak Emission Estimates

CASRN	HAP	Average Refinery Stream Liquid Concentration[a] (wt%)	Ratio of HAP to Benzene Concentration[b]
1330-20-7	Xylene	4.13	3.57

[a] Weighted average composition of all liquid process streams (Lucas, 2007a).
[b] Ratio of weighted average liquid concentration of selected HAP to weighted average liquid concentration for benzene.

Finally, the source characteristics for the process equipment area were revised to reduce the chance of the emission source area exceeding the dimensions of the refinery. The revised release areas associated with the process equipment leaks are summarized in Table C.

Table C. Areas Assigned for Fugitive Equipment Leaks

Refinery Crude Capacity (bbl/day)	Assigned Size Category	Assigned Equipment Leak Process Area (MM ft^2)
0 to <125,000	Small	0.3
125,000 to <225,000	Medium	1.7
≥225,000	Large	4

Emission Factors for Cooling Towers

For cooling towers, the emission estimates were developed for each refinery based on the uncontrolled AP-42 emission factor of 6 lbs total hydrocarbon (THC)/million gallons (MMgal). Cooling water flow rates were assumed to be 40 times the crude capacity. The HAP contents of the organics in the cooling water were estimated based on the weighted average refinery stream composition considering both liquid and gaseous streams as summarized by Lucas (2007a). The resulting HAP emission factors normalized by crude throughput are summarized in Table D.

Table D. Emission Factors for Cooling Towers

CASRN	HAP	Uncontrolled AP-42 Emission Factor (tpy/bbl/d)[a]
106-99-0	1,3-Butadiene	1.3E-08
540-84-1	2,2,4-Trimethylpentane	2.7E-05
71-43-2	Benzene	2.0E-05
92-52-4	Biphenyl	3.6E-07
1319-77-3	Cresols	2.7E-06
98-82-8	Cumene	4.1E-06
100-41-4	Ethylbenzene	1.0E-05
110-54-3	Hexane	9.7E-05
1634-04-4	Methyl tertiary butyl ether	1.2E-05
91-20-3	Naphthalene	3.1E-06
108-93-0	Phenol	1.9E-06
100-42-5	Styrene	6.4E-06
108-88-3	Toluene	4.4E-05
1330-20-7	Xylene	4.1E-05

[a] tpy/bbl/d = tons per year HAP emissions per barrel per day crude throughput.

Revised Emission Methodology and Source Areas for Storage Vessels

Emissions from storage vessels were originally developed based on emission estimates reported in permit applications. While there are separate emission factors for crude oil, light distillates, heavy distillates, and aromatics, only crude oil and light distillates are thought to be associated with the MACT I NESHAP. The "light distillates" originally included gasoline, naphtha, jet fuel, and diesel fuel (i.e., No. 2 fuel oil). This category was divided into two categories: "light distillates," which includes gasoline and jet naphtha, and "middle distillates," which includes other jet fuels, kerosene, and diesel fuel.

Revised emission factors were developed to model external floating roof (EFR) crude oil storage tanks, EFR light distillate (based on gasoline) storage tanks, and EFR middle distillate (based on jet fuel) storage tanks based on TANKS v4.09 model estimates. For the REM analysis, slotted guide poles and other openings or hatches with no fitting controls were assumed to be the level of control at all facilities.

The results of the TANKS model runs are summarized elsewhere (Lucas, 2008). The specific HAP composition of the volatile organic compound (VOC) emissions were estimated based on the average liquid and vapor phase composition for crude oil, gasoline, and jet naphtha. It was assumed that most of the VOC losses would be via gaseous losses, but that 20 percent of the losses would be via liquid losses (e.g., liquid clinging to the sides of the wall or guide pole). The average HAP concentrations of the VOC losses used in the analysis are presented in Table E. These concentrations combined with the tank emission losses and throughputs yield the emission factors presented in Table F.

In addition to revising these emission factors, the area associated with the tank farm were revised, again to limit the chance that the modeled emission source area would exceed the boundaries of the facility. The revised tank farm release areas are presented in Table G.

Table E. HAP Concentration of VOC Storage Vessel Emissions

CASRN	HAP	Crude Oil (wt%)	Gasoline (wt%)	Jet Naphtha (wt%)
106-99-0	1,3-Butadiene	0%	0.022%	0%
540-84-1	2,2,4-Trimethylpentane	0.25%	1.71%	0.44%
71-43-2	Benzene	0.73%	0.82%	1.10%
92-52-4	Biphenyl	0.012%	0.002%	0%
1319-77-3	Cresols	0.044%	0.16%	0.004%
98-82-8	Cumene	0.034%	0.18%	0.21%
100-41-4	Ethylbenzene	0.12%	0.37%	0.37%
110-54-3	Hexane	6.18%	4.97%	9.72%
1634-04-4	Methyl tertiary butyl ether	0%	3.60%	0%
91-20-3	Naphthalene	0.045%	0.089%	0.081%
108-93-0	Phenol	0.067%	0.011%	0.013%
100-42-5	Styrene	0%	0.776%	0%
108-88-3	Toluene	0.56%	2.12%	2.05%
1330-20-7	Xylene	0.46%	1.62%	1.41%

Table F. Storage Vessel HAP Emissions Factors

CASRN	HAP	Crude Oil (lbs/MMbbl)	Gasoline (lbs/MMbbl)	Jet Naphtha (lbs/MMbbl)
106-99-0	1,3-Butadiene	0.00	1.83	0.00
540-84-1	2,2,4-Trimethylpentane	3.39	144.62	22.00
71-43-2	Benzene	9.95	69.73	54.38
92-52-4	Biphenyl	0.17	0.17	0.00
1319-77-3	Cresols	0.60	13.42	0.19
98-82-8	Cumene	0.46	15.45	10.48
100-41-4	Ethylbenzene	1.62	31.48	18.23
110-54-3	Hexane	83.72	421.21	482.02
1634-04-4	Methyl tertiary butyl ether	0.00	305.44	0.00
91-20-3	Naphthalene	0.61	7.57	4.00
108-93-0	Phenol	0.91	0.94	0.67
100-42-5	Styrene	0.00	65.78	0.00
108-88-3	Toluene	7.55	179.30	101.44
1330-20-7	Xylene	6.19	137.47	70.11

Table G. Assumed Areas for Storage Vessel Tank Farms

Refinery Crude Capacity (bbls/day)	Assigned Size Category	Storage Vessel Tank Farm Area (MM ft^2)
0 to <125,000	Small	0.5
125,000 to <225,000	Medium	4
≥225,000	Large	7

Revised Emission Methodology and Source Areas for Wastewater Treatment Systems

A simple correlation was previously used to estimate benzene emissions for wastewater systems subject to the Benzene Waste Operations NESHAP (BWON; 40 CFR part 61, subpart FF) given the total mass benzene loading rate to wastewater. The methodology used to estimate the "controlled" BWON emissions were revised to better estimate the relative emissions from wastewater collection systems and wastewater treatment systems and to evaluate different levels of control.

The benzene loading rates to wastewater are estimated using the methodology from *Locating and Estimating Air Emissions from Sources of Benzene* (US EPA, 1998) as was done previously. For facilities that have benzene wastewater loadings (assumed to be the total annual benzene, or

TAB, quantity) exceeding 10 Mg/yr, then the facility is assumed to be subject to BWON requirements. For BWON facilities, the wastewater collection system is assumed to be 98 percent efficient, so that 2 percent of the TAB is released from the wastewater collection system. It is assumed that approximately 50 percent of the remaining benzene is recovered in the oil water separator and that 50 percent of the original TAB enters the enhanced biological unit (EBU). Eighty percent control efficiency was assumed for the EBU. For wastewater systems not subject to BWON, 85 percent of the benzene load is assumed to be emitted across the refinery; 50 percent of these emissions were attributed to the wastewater collection area and 50 percent were assigned to the EBU. Emissions of other HAP were estimated from the calculated benzene emissions using an adjustment factor based on the relative concentration of the HAP in wastewater streams, its octanol-water partition coefficient, and WATER9 emission estimates as was done previously (RTI, 2002).

As with the fugitive and storage tank farm release area parameters, the release areas for wastewater treatment sources were reduced to reduce the likelihood that the wastewater sources would exceed the boundaries of the facility. The revised release areas for wastewater treatment sources are provided in Table H.

Table H. Assumed Areas for Wastewater Collection and Treatment

Refinery Crude Capacity (bbls/day)	Assigned Size Category	Wastewater Collection Area (MM ft^2)	Wastewater Treatment Area (MM ft^2)
0 to <125,000	Small	0.10	0.10
125,000 to <225,000	Medium	0.43	0.43
≥225,000	Large	1.7	1.7

Additional Data

Table I. Comparison of REM and RTR Modeled Maximum Individual Risks (MIR), By Facility

	Facility ID	MIR Ratio (REM:RTR)	REM Cancer MIR (in 1 million)	RTR Cancer MIR (in 1 million)
1	PET_NEI34057	0.1	2	14
2	PET_NEI6095	0.1	1	13
3	PET_NEIPRT$64	0.2	2	12
4	PET_NEI12711	0.3	9	28
5	PET_NEI34898	0.3	7	21
6	PET_NEI34050	0.4	5	12
7	PET_NEI40371	0.4	4	10
8	PET_NEI6475	0.4	5	13
9	PET_NEI12480	0.5	3	6
10	PET_NEI12791	0.5	4	10
11	PET_NEI12988	0.5	10	19
12	PET_NEI20174	0.5	5	12
13	PET_NEI40531	0.5	4	8
14	PET_NEI46752	0.6	1	2
15	PET_NEIOKT$11009	0.6	6	9
16	PET_NEI33031	0.7	10	15
17	PET_NEI876	0.7	10	15
18	PET_NEI11449	1	5	9
19	PET_NEI12044	1	6	6
20	PET_NEI12458	1	1	1
21	PET_NEI12968	1	3	2
22	PET_NEI18394	1	8	11
23	PET_NEI19587	1	4	5
24	PET_NEI32864	1	10	11
25	PET_NEI33008	1	10	9
26	PET_NEI33039	1	10	10
27	PET_NEI34907	1	0.5	0
28	PET_NEI41771	1	9	13
29	PET_NEI42040	1	10	15
30	PET_NEI42309	1	20	15
31	PET_NEI42413	1	2	2
32	PET_NEI6087	1	8	12
33	PET_NEI6116	1	10	7
34	PET_NEI6136	1	5	6
35	PET_NEI6166	1	5	7
36	PET_NEI6436	1	10	12
37	PET_NEI6446	1	2	2
38	PET_NEI6963	1	0.3	0
39	PET_NEI11200	2	2	1
40	PET_NEI11232	2	9	5
41	PET_NEI11663	2	8	4

	Facility ID	MIR Ratio (REM:RTR)	REM Cancer MIR (in 1 million)	RTR Cancer MIR (in 1 million)
42	PET_NEI12460	2	1	1
43	PET_NEI12486	2	9	4
44	PET_NEI12969	2	2	1
45	PET_NEI18372	2	0.8	0
46	PET_NEI2CA314628	2	5	3
47	PET_NEI2KS125003	2	5	3
48	PET_NEI32762	2	7	5
49	PET_NEI32801	2	4	2
50	PET_NEI33010	2	0.3	0
51	PET_NEI34062	2	10	5
52	PET_NEI34862	2	4	2
53	PET_NEI34873	2	10	5
54	PET_NEI42020	2	8	5
55	PET_NEI42025	2	6	3
56	PET_NEI42381	2	9	5
57	PET_NEI42425	2	2	1
58	PET_NEI43243	2	4	2
59	PET_NEI53702	2	5	2
60	PET_NEI6022	2	20	10
61	PET_NEI6062	2	10	5
62	PET_NEI6123	2	10	5
63	PET_NEI6519	2	10	5
64	PET_NEI7233	2	10	5
65	PET_NEICA1910268	2	20	11
66	PET_NEI12464	3	4	1
67	PET_NEI19834	3	6	2
68	PET_NEI26533	3	0.2	0
69	PET_NEI41591	3	6	2
70	PET_NEI6130	3	20	6
71	PET_NEI7781	3	20	6
72	PET_NEI11574	4	20	5
73	PET_NEI13322	4	0.5	0
74	PET_NEI13371	4	20	5
75	PET_NEI363	4	3	1
76	PET_NEI40723	4	10	2
77	PET_NEI415	4	6	2
78	PET_NEI42016	4	9	2
79	PET_NEI6127	4	10	2
80	PET_NEI8139	4	6	1
81	PET_NEI12084	5	2	0
82	PET_NEI19870	5	3	1
83	PET_NEI26218	5	2	0
84	PET_NEI32997	5	2	0
85	PET_NEI42081	5	0.7	0
86	PET_NEI46556	5	30	6
87	PET_NEIWYT$12156	5	0.007	0.002
88	PET_NEI18406	6	20	9

	Facility ID	MIR Ratio (REM:RTR)	REM Cancer MIR (in 1 million)	RTR Cancer MIR (in 1 million)
89	PET_NEI26101	6	3	0
90	PET_NEI34022	6	3	1
91	PET_NEI40732	6	30	5
92	PET_NEI6375	6	8	1
93	PET_NEI7441	6	5	1
94	PET_NEI8612	6	4	1
95	PET_NEI109	7	30	4
96	PET_NEI12459	7	7	1
97	PET_NEI41863	7	10	2
98	PET_NEI53718	7	1	0
99	PET_NEINJT$891	7	8	1
100	PET_NEI40625	8	3	0
101	PET_NEI42370	8	0.4	0
102	PET_NEI6084	8	10	1
103	PET_NEI889	8	8	1
104	PET_NEI11119(B)	9	5	1
105	PET_NEI41864	9	3	0
106	PET_NEICA0379991	9	2	0
107	PET_NEI11450	10	20	2
108	PET_NEI21034	10	9	1
109	PET_NEI34061	10	4	0
110	PET_NEI49781	10	10	1
111	PET_NEICA0370363	10	20	2
112	PET_NEI41865	11	0.2	0
113	PET_NEI113	20	9	1
114	PET_NEI20103	20	10	1
115	PET_NEI20467	20	20	1
116	PET_NEI2CA254640	20	3	0
117	PET_NEI32353	20	5	0
118	PET_NEI34912	20	3	0
119	PET_NEI42382	20	5	0
120	PET_NEI6018	20	5	0
121	PET_NEI7130	20	2	0
122	PET_NEI20616	22	8	0
123	PET_NEI11192	30	20	1
124	PET_NEI11885	30	5	0
125	PET_NEI19869	30	5	0
126	PET_NEI20966	30	2	0
127	PET_NEI2CA131003	30	10	0
128	PET_NEI34069	30	0.6	0
129	PET_NEI46764	30	4	0
130	PET_NEI34863	40	1	0
131	PET_NEI34872	40	30	1
132	PET_NEI371	40	2	0
133	PET_NEI42583	40	4	0
134	PET_NEI18415	50	1	0
135	PET_NEI6617	60	4	0

	Facility ID	MIR Ratio (REM:RTR)	REM Cancer MIR (in 1 million)	RTR Cancer MIR (in 1 million)
136	PET_NEI33007	70	0.9	0
137	PET_NEI404	100	4	0.04
138	PET_NEI25464	300	1	0
139	PET_NEI55835	400	7	0
140	PET_NEI18408	500	7	0.01
141	PET_NEI20154	3000	20	0.007
142	PET_NEI21130	4000	10	0
143	PET_NEI21466	5000	7	0
144	PET_NEI26473	5000000	0.5	0.00000009
145	PET_NEI11715		10	
146	PET_NEI18673		5	
147	PET_NEI2AK530001		0.02	
148	PET_NEI2AK560004		0.08	
149	PET_NEI2CA312611		2	
150	PET_NEI2NV110905		0.7	
151	PET_NEI33009		4	
152				6
153				5
154				4
155				3
156				1
157				0.005

Table J. Comparison of REM and RTR Modeled Annual Cancer Incidence, By Facility

	Facility ID	MIR Ratio (REM:RTR)	REM Cancer Incidence (excess cancer cases per year)	RTR Cancer Incidence (excess cancer cases per year)
1	PET_NEIPRT$64	0.1	0.0002	0.001
2	PET_NEI12711	0.2	0.001	0.006
3	PET_NEI34057	0.3	0.0003	0.001
4	PET_NEI34898	0.4	0.0001	0.0003
5	PET_NEI12791	0.4	0.0007	0.002
6	PET_NEI34050	0.5	0.0006	0.001
7	PET_NEI40371	0.5	0.00006	0.0001
8	PET_NEI6166	0.6	0.0006	0.001
9	PET_NEI33039	0.6	0.00001	0.00002
10	PET_NEI12044	0.7	0.0003	0.0004
11	PET_NEI876	0.7	0.0002	0.0003
12	PET_NEI11449	0.8	0.0006	0.0008
13	PET_NEI20174	0.8	0.0007	0.0008
14	PET_NEI12988	0.9	0.0005	0.0006
15	PET_NEI6095	0.9	0.0004	0.0004
16	PET_NEI18394	0.9	0.0001	0.0001
17	PET_NEI40531	0.9	0.00004	0.00004
18	PET_NEI33031	0.9	0.0008	0.0009
19	PET_NEI19587	0.9	0.002	0.002
20	PET_NEI43243	0.9	0.000006	0.000006
21	PET_NEI34907	1	0.000009	0.000009
22	PET_NEI6963	1	0.00002	0.00002
23	PET_NEI6136	1	0.0001	0.0001
24	PET_NEI32801	1	0.0001	0.00009
25	PET_NEI8612	1	0.0002	0.0002
26	PET_NEI32864	1	0.0008	0.0007
27	PET_NEI6436	1	0.002	0.002
28	PET_NEI32762	1	0.0002	0.0002
29	PET_NEI6446	1	0.00002	0.00002
30	PET_NEI42381	1	0.00007	0.00005
31	PET_NEIOKT$11009	1	0.00009	0.00006
32	PET_NEI12458	2	0.00006	0.00004
33	PET_NEI11232	2	0.005	0.003
34	PET_NEI42309	2	0.0001	0.00007
35	PET_NEI12486	2	0.00002	0.00001
36	PET_NEI33010	2	0.00002	0.00001
37	PET_NEI6475	2	0.0004	0.0002
38	PET_NEI12460	2	0.00007	0.00004
39	PET_NEI11663	2	0.0008	0.0004
40	PET_NEI11200	2	0.0005	0.0003
41	PET_NEI6123	2	0.003	0.001
42	PET_NEI42020	2	0.0003	0.0002
43	PET_NEI12969	2	0.0002	0.0001
44	PET_NEI2KS125003	2	0.0001	0.00005
45	PET_NEI33008	2	0.0007	0.0004

	Facility ID	MIR Ratio (REM:RTR)	REM Cancer Incidence (excess cancer cases per year)	RTR Cancer Incidence (excess cancer cases per year)
46	PET_NEI6022	2	0.003	0.002
47	PET_NEI12480	2	0.001	0.0006
48	PET_NEI46752	2	0.00007	0.00003
49	PET_NEI6130	2	0.0006	0.0003
50	PET_NEI42025	2	0.0002	0.00008
51	PET_NEI34062	2	0.00001	0.000005
52	PET_NEI42425	2	0.00005	0.00002
53	PET_NEI7233	2	0.002	0.0008
54	PET_NEI26533	3	0.00002	0.00001
55	PET_NEI41771	3	0.001	0.0005
56	PET_NEI34873	3	0.002	0.0009
57	PET_NEI12968	3	0.0002	0.00009
58	PET_NEI18372	3	0.0003	0.0001
59	PET_NEI13371	3	0.00005	0.00002
60	PET_NEI6062	3	0.0006	0.0002
61	PET_NEI11574	3	0.0004	0.0002
62	PET_NEI6519	3	0.0001	0.00004
63	PET_NEI2CA314628	3	0.0008	0.0003
64	PET_NEI53702	3	0.001	0.0005
65	PET_NEI7781	3	0.003	0.001
66	PET_NEICA1910268	3	0.005	0.002
67	PET_NEI41863	3	0.0005	0.0002
68	PET_NEI46556	3	0.001	0.0004
69	PET_NEI6116	3	0.0004	0.0001
70	PET_NEI13322	3	0.00003	0.000008
71	PET_NEI109	4	0.003	0.0008
72	PET_NEI19870	4	0.0004	0.0001
73	PET_NEI42081	4	0.0002	0.00005
74	PET_NEI363	4	0.0001	0.00004
75	PET_NEI415	4	0.001	0.0002
76	PET_NEIWYT$12156	4	0.0000005	0.0000001
77	PET_NEI41591	4	0.001	0.0003
78	PET_NEI12464	4	0.0001	0.00002
79	PET_NEI42040	5	0.0006	0.0001
80	PET_NEI42016	5	0.0004	0.00008
81	PET_NEI8139	5	0.000008	0.000002
82	PET_NEI6375	5	0.009	0.002
83	PET_NEINJT$891	5	0.002	0.0005
84	PET_NEI26218	5	0.0006	0.0001
85	PET_NEI12084	6	0.0004	0.00007
86	PET_NEI34862	6	0.003	0.0006
87	PET_NEI19834	6	0.001	0.0002
88	PET_NEI26101	6	0.0002	0.00003
89	PET_NEI6087	7	0.0003	0.00005
90	PET_NEI11119B	7	0.003	0.0004
91	PET_NEI34022	7	0.002	0.0002

	Facility ID	MIR Ratio (REM:RTR)	REM Cancer Incidence (excess cancer cases per year)	RTR Cancer Incidence (excess cancer cases per year)
92	PET_NEI42413	7	0.00009	0.00001
93	PET_NEI40625	8	0.0006	0.00008
94	PET_NEI53718	8	0.0005	0.00006
95	PET_NEI889	8	0.0008	0.0001
96	PET_NEI42370	9	0.0003	0.00003
97	PET_NEI12459	9	0.0001	0.00001
98	PET_NEI40732	9	0.0002	0.00002
99	PET_NEI18406	9	0.0006	0.00007
100	PET_NEI41864	9	0.0006	0.00006
101	PET_NEI7441	10	0.0008	0.00009
102	PET_NEI21034	10	0.008	0.0008
103	PET_NEICA0370363	10	0.005	0.0004
104	PET_NEI11192	10	0.001	0.00009
105	PET_NEI49781	10	0.0003	0.00002
106	PET_NEICA0379991	10	0.002	0.0002
107	PET_NEI11450	10	0.002	0.0001
108	PET_NEI41865	10	0.00005	0.000004
109	PET_NEI7988	10	0.045	0.0005
110	PET_NEI40723	10	0.01	0.0008
111	PET_NEI6084	10	0.0003	0.00002
112	PET_NEI20103	20	0.0002	0.00001
113	PET_NEI6127	20	0.001	0.00008
114	PET_NEI42382	20	0.0001	0.000006
115	PET_NEI33007	20	0.00003	0.000002
116	PET_NEI34912	20	0.00006	0.000003
117	PET_NEI20616	20	0.001	0.00007
118	PET_NEI32997	20	0.00005	0.000002
119	PET_NEI34061	20	0.00003	0.000001
120	PET_NEI32353	20	0.0001	0.000006
121	PET_NEI20966	20	0.001	0.00004
122	PET_NEI113	20	0.002	0.00009
123	PET_NEI7130	30	0.0004	0.00001
124	PET_NEI42583	30	0.00009	0.000003
125	PET_NEI46764	30	0.00007	0.000003
126	PET_NEI6018	30	0.0004	0.00001
127	PET_NEI2CA254640	30	0.00004	0.000001
128	PET_NEI11885	30	0.002	0.00004
129	PET_NEI2CA131003	40	0.01	0.0003
130	PET_NEI34069	40	0.00005	0.000001
131	PET_NEI19869	40	0.00006	0.000002
132	PET_NEI34872	40	0.003	0.00009
133	PET_NEI6617	40	0.0004	0.00001
134	PET_NEI371	40	0.00002	0.000001
135	PET_NEI20467	50	0.01	0.0003
136	PET_NEI34863	50	0.001	0.00002
137	PET_NEI18415	60	0.00001	0.0000002

	Facility ID	MIR Ratio (REM:RTR)	REM Cancer Incidence (excess cancer cases per year)	RTR Cancer Incidence (excess cancer cases per year)
138	PET_NEI404	90	0.00003	0.0000004
139	PET_NEI25464	300	0.0001	0.000001
140	PET_NEI55835	500	0.001	0.000002
141	PET_NEI18408	2000	0.00010	0.00000005
142	PET_NEI20154	3000	0.0009	0.0000003
143	PET_NEI21130	4000	0.004	0.000001
144	PET_NEI21466	4000	0.004	0.000001
145	PET_NEI26473	4000000	0.0001	0.00000000003
146	PET_NEI11715		0.003	
147	PET_NEI18673		0.00002	
148	PET_NEI33009		0.00005	
149	PET_NEI2CA312611		0.0006	
150	PET_NEI2NV110905		0.0000001	
151	PET_NEI2AK560004		0.0000004	
152	PET_NEI2AK530001		0.000000002	
153	PET_NEI33030			0.0002
154	PET_NEI12790			0.0002
155	PET_NEI7134			0.0001
156	PET_NEI25450			0.0002
157	PET_NEI26489			0.00002
158	PET_NEICA10578			0.00001
159	PET_NEINMT$12478			0.00001
160	PET_NEI2TX14199			0.000003
161	PET_NEI20797			0.00001
162	PET_NEI7973			0.0000002

Table K. REM Input Assumptions for Each Facility

This table is in-progress and will be available upon request.

Table L. REM Emissions Estimates for Each Facility

This table is in-progress and will be available upon request.

Table M. RTR Emissions Estimates for Each Facility

This table is in-progress and will be available upon request.

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Office of Air Quality Planning and Standards
Health and Environmental Impacts Division
Research Triangle Park, NC

Publication No. EPA-452/R-09-006
June, 2009